COMPLETE GLUTATHIONE

Ingredients: Each tablet supplies: Vitamin B-1 10 mg, Vitamin B-2 10 mg, Vitamin B-6 5 mg, Vitamin B-12 50 mcg, Vitamin C 75 mg, Folic Acid 50 mcg, Vitamin E (as succinate) (CONTAINS WHEAT) 10 i.u., Magnesium (as chelate) 500 mcg, Zinc (as chelate) 500 mcg, Selenium (as chelate) 10 mcg, N-Acetyl Cysteine 60 mg, Alpha Lipoic Acid 10 mg, L-Glutamine 15 mg, Super Oxide Dismutase Type-G (glutathione) 25 mg, Catalase 20 mg, Glutathione (reduced) 5 mg, Milk Thistle 50 mg, Silybin 10 mg, Rosemary 75 mg, Turmeric 10 mg.

Supportive Function: Glutathione is a powerful antioxidant, immune nutrient, and detoxifier. Complete glutathione is the nutritional answer for raising glutathione in the cells without a whey source. In convenient tablet form.

When is glutathione support helpful? Helpful for general health, stress, athletic performance, skin disorders, detoxification, fatigue, digestion, pregnancy/lactation, sleep, psychoneurobiology, trauma and burns, seizures, stomach/bowel conditions, kidney issues, arthritis, eyesight, hearing loss, sinusitis, lung problems, MS, lung disease, hepatitis, diabetes, heart disease, ear infections, stroke, cholesterol, BPH, Parkinson's, Alzheimer's, PSA levels, balding/hair loss, infertility...and many other conditions where decreased levels of glutathione are suspected of being a problem.

Clinical Applications/Research: N-acetyl cysteine raises glutathione levels in the body by increasing cellular cysteine, the rate-limiting step to glutathione synthesis: Glutathione (GSH) is a potent intracellular antioxidant that is backed by strong research describing its dramatic health benefits. The biggest roadblock in glutathione supplementation is the breakdown of glutathione in the digestive tract (it is quickly broken down and eliminated), lowering the efficiency of glutathione supplementation. However, many nutrients, such as the glutathione precursor N-acetyl cysteine (NAC), are able to bypass the normal roadblocks to successful cellular glutathione production by increasing glutathione production once it reaches the cell. NAC is a readily absorbable building block for glutathione, and since cysteine is the rate-limiting amino acid in glutathione, NAC supplementation increases the glutathione concentration.
Supplementation of the glutathione precursor cysteine in several clinical trials improved skeletal muscle functions, decreased the body fat/lean, body mass ratio, decreased inflammatory cytokines, and improved immune functions. "As all these parameters degenerate with age, these findings suggest: (i) that **loss of youth, health and quality of life may be partly explained by a deficit in cysteine** and (ii) that the dietary consumption of cysteine is generally suboptimal and **everybody is likely to have a cysteine deficiency sooner or later**" Droge W. Oxidative stress and ageing: is ageing a cysteine deficiency syndrome? Philos Trans R Soc Lond B Biol Sci. 2005 Dec 29; 360(1464): 2355-72**. Supplementation with N-acetyl cysteine is more efficient and absorbable** than cysteine supplementation, and some people are sensitive to straight cysteine supplementation – it can be toxic and not easily transported into cells. When patients suffer from Tylenol poisoning, they are given N-acetyl cysteine, the best known

and fastest way to raise cellular glutathione levels; the glutathione in turn binds to the toxic by-products of the excess Tylenol and prevents tissue damage.
N-acetyl cysteine is well known as a life-saving nutrient when it comes to acetaminophen (Tylenol) poisoning. If someone takes excessive acetaminophen and they are rushed to the hospital, they are immediately administered n-acetyl cysteine, because n-acetyl cysteine is the fastest, most effective way to raise intracellular levels of glutathione. It is a membrane-permeable precursor to cysteine and glutathione. Being membrane-permeable, N-acetyl cysteine is more readily absorbed than either cysteine by itself, or glutathione. As the rate-limiting amino acid in glutathione synthesis, it readily provides the necessary building block to the cells, and glutathione concentrations are increased.

Excessive acetaminophen burdens the liver's ability to remove the toxic by-products, and extensive cell damage or even death may occur. As long as there is enough glutathione in the liver to take care of this damaging intermediate product of acetaminophen, the glutathione will hook onto it and draw it out of the system. This happens in what is called phase II detoxification in the liver. Glutathione binds many toxic substances in this way and targets them for removal from the body. In phase II, where the body is preparing to make the toxin water-soluble, a highly toxic, reactive substance is formed as an intermediary by-product, and it is much more toxic than the original form of acetaminophen. As the quantity of acetaminophen increases, the amount that is detoxified in this pathway increases, and the highly toxic, reactive by-products are left unbound, depleting the body's stores of glutathione and causing extensive major cell damage and liver injury.

NAC has been reported to have many benefits, from **antioxidant properties** to **restoration of nitric oxide bioavailability** in the circulation (Xia Z, Antioxidant N-acetylcysteine restores systemic nitric oxide availability and corrects depressions in arterial blood pressure and heart rate in diabetic rats. Free Radic Res. 2006 Feb; 40(2): 175-84.) NAC has a very **strong protective effect on cell health**. Brain neurons with reduced glutathione content were found to have increased oxidant levels and increased susceptibility to injury, however this situation was reversed with NAC supplementation (Aoyama K, et al. Neuronal glutathione deficiency and age-dependent neurodegeneration in the EAAC1 deficient mouse. Nat Neurosci. 2006 Jan; 9(1): 119-26.)

NAC has a reputation as being a nutrient that prevents the toxic assault from many strong chemicals. The scientific literature is full of documentation for this protection (Olanders K. et al. Protective effects of N-acetyl-L-cysteine and platelet activating factor inhibition are not linked to intercellular adhesion molecule-1 expression after intestinal ischemia and reperfusion injury in rats. Scand J Gastroenterol. 2003 Jun; 38(6): 618-25; Isoniemi H, Poyhia R. N-Acetylcysteine--a new possibility in the therapy of hepatic failure. Duodecim. 1999; 115(4): 361-2; Muldoon LL, et al. Rescue from enhanced alkylator-induced cell death with low molecular weight sulfur-containing chemoprotectants. J Pharmacol Exp Ther. 2001 Mar; 296(3): 797-805; setting if chemotherapy and chemoprotectant can be physically and/or temporally separated; Karg E, et al. Glutathione in human melanoma cells. Effects of cysteine, cysteine esters and glutathione isopropyl ester. : J Dermatol Sci. 1990 Jan; 1(1): 39-45.)

NAC and neuronal health:

In an interesting study conducted by brain researchers, they noted "Oxidative stress caused by various stimuli lead to oxidation of glutathione (GSH), the major redox (antioxidant) power of the cell. Amyloid beta is one of the key components of senile plaques surrounding nerves, and is involved in the initiation and triggers of Alzheimer's

disease (AD). Lower GSH levels correlated with the activation of protein kinases (enzymes) have been demonstrated in AD, Parkinson's disease (PD) and other neurodegenerative disorders and have been proposed to play a central role in the deterioration of the aging and neurodegenerative brain." The same study concluded that N-acetyl cysteine replenished GSH levels, and the cell death induced by amyloid beta in primary neuronal cells was reversed by N-acetyl cysteine. "Likewise, protein oxidation, loss of mitochondrial function and DNA fragmentation all returned to control levels by pretreatment" (Bartov O, et al. Low molecular weight thiol amides attenuate MAPK activity... Brain Res. 2005 Dec 28.)

Selenium is an integral nutrient for the production of glutathione peroxidase, an important antioxidant enzyme that also contains **glutathione**. Acting as a component in this enzyme is the main biological function of selenium in our bodies. Many of the health characteristics of selenium and glutathione are actually attributed to the glutathione peroxidase enzyme. Supplementation of selenium is reported to have many benefits, including an ability to promote cell-mediated immunity in humans (Yu B, Wang M, Li D. Zhonghua Wai Ke Za Zhi. 1996 Jan; 34(1): 50-3. (The relationship between selenium and immunity in large bowel cancer).

Alpha lipoic acid, like many of the antioxidants, works synergistically with glutathione to protect the body from oxidative stress, elevates the GSH-peroxidase enzyme, and specifically prevents the depletion of GSH (Shila s. et al. Arsenic intoxication-induced reduction of glutathione level and of the activity of related enzymes in rat brain regions: reversal by DL-alpha-lipoic acid. Arch Toxicol. 2005 Mar; 79(3): 140-6.) Alpha lipoic acid and N-acetyl cysteine have been shown to reverse oxidative damage caused by zinc deficiency (Mackenzie GG et al. alpha-Lipoic acid and N-acetyl cysteine... Free Radic Res. 2006 Jan; 40(1): 75-84.) Additionally, alpha lipoic acid contains precursors for cysteine and glutathione.

Kwashiorkor is a severe form of malnutrition and has been reported to be associated with oxidative stress. Even though the therapy of kwashiorkor is still ineffective, a pilot study tested the hypothesis that raising glutathione status would be beneficial for the clinical recovery of kwashiorkor patients. "Both **glutathione** and **alpha-lipoic acid** supplementation had positive effects on survival… The data strongly suggest that a **therapy restoring the antioxidative capacity** by applying cysteine equivalents in the form of **glutathione and/or alpha-lipoic acid** is beneficial for biochemical and clinical recovery of kwashiorkor patients" (Becker K, Redox Rep. 2005;10(4):215-26.)

Glutamine is an amino acid that can balance pH, protect the intestinal lining, and exhibit strong antioxidant properties. Research has shown that glutamine can entirely prevent the decrease in glutathione, which normally happens after an oxidative stress challenge (Gonzales S, et al. World J Gastroenterol. 2005 Jun 21; 11(23): 3533-8.)

Milk Thistle is an herb historically known for cleansing and supporting the liver. One of its main components, **silybin, is a highly active ingredient** that has been shown to **increase** patient serum levels of **glutathione and glutathione peroxidase, while protecting against glutathione depletion** (Wellington K, Jarvis B. Silymarin…BioDrugs. 2001; 15(7): 465-89.) and protect against GSH depletion; Campos R, et al. Silybin dihemisuccinate protects against glutathione depletion and lipid peroxidation induced by acetaminophen on rat liver. Planta Med. 1989 Oct; 55(5): 417-9.)

Vitamin C, vitamin E, zinc, and the B vitamins all work together in the synthesis and maintenance of sufficient amounts of antioxidants, including active, reduced glutathione (Sprietsma JE. Med Hypotheses. 1999 Jul; 53(1): 6-16. Modern diets and diseases). **Magnesium deficiency**

is known to affect the glutathione peroxidase enzyme and the ability to respond to oxidative stress (Vernet P, et al. Biochim Biophys Acta. 2004 Nov 18; 1675(1-3): 32-45).

Superoxide dismutase (Type G glutathione) is a natural free-radical scavenging enzyme. It mops ups the superoxide free radical, and works synergistically with many of the antioxidants in the oxidative defense system of the body, particularly glutathione. The superoxide radical produces oxidative stress associated with tissue damage and dysfunction of physiological signals. Molecular studies reveal that insulin receptor activity (associated with aging) is increased by this oxidative stress, and decreased by certain antioxidants such as glutathione (Droge W. Oxidative stress and ageing: is aging a cysteine deficiency syndrome? Philos Trans R Soc Lond B Biol Sci. 2005 Dec 29; 360(1464): 2355-72.) **Catalase** is a radical fighting enzyme that turns potentially dangerous hydrogen peroxide into harmless water and oxygen.

Anti-inflammatory, natural cox-2 inhibitors: The Cox 2 enzyme causes inflammatory substances to be produced in the body and is also the enzyme targeted by the cox-2 inhibitor drugs such as Vioxx and Celebrex. **Raising glutathione has the ability to modulate cox-2 gene expression and prostaglandin (inflammatory substance) synthesis** (Chen JX et al. Glutathione mediates LPS-stimulated COX-2 expression. J Cell Physiol. 2003 Oct; 197(1): 86-93.)

Glutathione (GSH) functions as a natural cox-2 inhibitor because adequate levels of glutathione affect this gene expression of the cox-2 enzyme. Reduced Glutathione is the preferred form of glutathione, because it is the active form. When glutathione is oxidized, it needs to be recycled into its reduced form. As a supplement, GSH breaks down in digestion and is not the most effective way to raise glutathione in the cells, HOWEVER, oral glutathione supplementation has been shown to have some effect on GSH concentration in the liver because of the efficient extraction by the liver of the cysteine that originated from the breakdown of GSH in the gut (Grattagliano I, et al. Effect of oral glutathione monoethyl ester and glutathione on circulating and hepatic sulfhydrils in the rat. Pharmacol Toxicol. 1994 Dec; 75(6): 343-7.) Glutathione protects not only individual cells but also the tissues of the arteries, brain, heart, immune cells, kidneys, lenses of the eyes, liver, lungs, and skin.

In addition to glutathione, the following nutrients ALSO inhibit inflammatory cox-2 naturally, and they have been added to the **Nutri-West Complete Glutathione** formula because their high antioxidant activity protects glutathione levels, plus they boost the anti-inflammatory support of the formula:

Turmeric is a spice with high antioxidant activity, and possesses the ability to preserve glutathione levels and to recycle oxidized glutathione. The **curcumin** in turmeric plays a particularly important regulatory role in this process (Dickinson DA Biol Chem. 2003 Apr; 384(4): 527-37. Cytoprotection against oxidative stress and the regulation of glutathione synthesis). **Curcumin** also **possesses powerful cox-2 inhibiting activity**. "Curcumin has been described as a potent antioxidant and anti-inflammatory agent. Evidence has also been presented to suggest that curcumin can suppress tumor initiation, promotion and metastasis… All of these studies suggest that curcumin has enormous potential in the prevention and therapy of cancer" (Aggarwal BB, Kumar A, Bharti AC. Anticancer potential of curcumin: pre-clinical and clinical studies. Anticancer Res. 2003 Jan-Feb; 23(1A): 363-98.) It has also been suggested that curcumin's "anti-inflammatory and anti-oxidant actions may be useful in the prevention-treatment of neurodegenerative diseases, e.g. Alzheimer's and Parkinson's Diseases" (Ambegaokar SS, et al. Neuroendocrinol Lett. 2003 Dec; 24(6): 469-73).

Rosemary contains carnosol, rosmanol, and epirosmanol, which all demonstrate high antioxidant activity (Zeng HH, et al. 2001), and additionally, carnasol is a potent **inhibitor of cox 2** (Subbaramaiah K. et al. 2002). **Rosemary** reduces pathogenic substances such as bacteria, raises detoxification enzymes in the liver, and decreases inflammation and liver cell injury (Ahn J. et al. 2004; Sotelo-Felix JI, et al. 2002.) **Rosemary can specifically raise glutathione levels.**

Suggested Dosage: 1 tablet daily or as directed

Size: 60 tablets

Vegetarian: No

Contraindications: None known.