Detox Antiox™ synergistically combines many nutrients that have a positive effect on the immune system. This formula contains multiple nutrients known to raise glutathione levels making it helpful for supporting phase II liver detoxification. It also combats free radicals and helps detoxify harmful chemicals including heavy metals. L-Leucine when taken with NAC prevents mercury from being reabsorbed into the central nervous system. Detox Antiox™ is also designed to aid the production of metallothionein. The vitamin E is 60% gamma, mixed tocopherols. Lipoic acid regenerates vitamins E and C and supplies sulfur for detoxification. This powerful formula also provides the well-researched antioxidants green tea, grape seed extract and curcumin.

**Did You Know?**

1. Green tea EGCg (epigallactocatechin gallate) is effective against H. Pylori (known to cause ulcers). Research shows that antibiotics such as amoxicillin worked BETTER in the presence of EGCg.²

2. "It is concluded that pathways activated by GTTPPs or EGCg in normal epithelial versus tumor cells create different oxidative environments, favoring either normal cell survival or tumor cell destruction. This finding may lead to applications of naturally occurring polyphenols to enhance the effectiveness of chemo/radiation therapy to promote cancer cell death while protecting normal cells."³

3. EGCg is more effective when taken along with curcumin. Curcumin increases its cellular absorption.⁴

**Unique Features of Detox Antiox™:**

- Lipoic Acid regenerates the Vitamin E and C in this formula so they can have long acting antioxidant activity. It also provides SH (sulphydryl groups) that protect against metal toxicity including iron and copper.

- Vitamin E protects cell membranes from oxidative destruction and retards breakdown of cell membranes. Literature shows that vitamin E can reduce the toxic effects of mercury. Vitamin E reduces chromosomal breakage and has sulphydryl-protective abilities.

- Selenium helps to make glutathione enzymes needed for liver detoxification of harmful chemicals. This is the primary nutrient for binding mercury to allow for its excretion. Selenium also enhances the antioxidant abilities of vitamin E.

- The zinc and selenium are bound to methionine which aids the synthesis of metallothioneine, the important zinc binding protein, known to aid the removal of heavy metals such as cadmium. Methionine is a sulfur containing amino acid involved in Phase II detoxification.

- Polyphenols from grape seed extract and green tea have been shown to protect against iron and copper toxicity by chelating them.

- Mice given NAC while being exposed to mercury excreted about 50% into the urine while control animals excreted only 4-10% over 48 hours.
Toxic metals and antioxidants: Part II. The role of antioxidants in arsenic and cadmium toxicity.


Exposure to toxic metals has become an increasingly recognized source of illness worldwide. Both cadmium and arsenic are ubiquitous in the environment, and exposure through food and water as well as occupational sources can contribute to a well-defined spectrum of disease. The symptom picture of arsenic toxicity is characterized by dermal lesions, anemia, and an increased risk for cardiovascular disease, diabetes, and liver damage. Cadmium has a significant effect on renal function, and as a result alters bone metabolism, leading to osteoporosis and osteomalacia. Cadmium-induced genotoxicity also increases risk for several cancers. The mechanisms of arsenic- and cadmium-induced damage include the production of free radicals that alter mitochondrial activity and genetic information. The metabolism and excretion of these heavy metals depend on the presence of antioxidants and thiols that aid arsenic methylation and both arsenic and cadmium metallothionein-binding. S-adenosylmethionine, lipoic acid, glutathione, selenium, zinc, N-acetylcysteine (NAC), methionine, cysteine, alphatocopherol, and ascorbic acid have specific roles in the mitigation of heavy metal toxicity. Several antioxidants including NAC, zinc, methionine, and cysteine, when used in conjunction with standard chelating agents, can improve the mobilization and excretion of arsenic and cadmium.

**Study of the effect of the administration of Cd(II), cysteine, methionine, and Cd(II) together with cysteine or methionine on the conversion of xanthine dehydrogenase into xanthine oxidase.**


Cadmium is known to be a potent pulmonary carcinogen to human beings and to induce prostate tumor. The sequestration of cadmium, an extremely toxic element to living cells, which is performed by biological ligands such as amino acids, peptides, proteins or enzymes is important to minimize its participation in such deleterious processes. The synthesis of metallothionein is induced by a wide range of metals, in which cadmium is a particularly potent inducer. This protein is usually associated with cadmium exposure in man. Because metallothioneins may act as a detoxification agent for cadmium and chelation involves sulfur donor atoms, we administered only cadmium, cysteine, or methionine to rats and also each of these S-amino acids together with cadmium and measured the production of superoxide radicals derived from the conversion of xanthine dehydrogenase to xanthine oxidase. It could be seen in this work that the presence of cadmium enhances this conversion. However, its inoculation with cysteine or methionine almost completely diminishes this effect and this can be the result of the fact that these amino acids complex Cd(II). Thus, these compounds can be a model of the action of metallothionein, removing cadmium from circulation and preventing its deleterious effect.

**Influence of dietary methionine level on the liver metallothionein mRNA level in rats.**


The effects of some methyl-containing compounds added to a choline-deficient diet on the metallothionein mRNA level in the rat liver were studied. The addition of choline or carnitine to the choline-deficient diet did not induce a gain in body weight, while the addition of either betaine or methionine to the choline-deficient diet, or of methionine to the choline-deficient diet with choline significantly increased the body weight. The metallothionein mRNA level in the liver of rats fed on the choline-deficient diet was similar to that of rats fed on the choline-deficient diet with choline, betaine or carnitine. However, the addition of methionine to the choline-deficient diet with or without choline caused a marked suppression in the metallothionein mRNA level in the liver. It is thus surmised that the metallothionein mRNA level in the liver might be regulated by the dietary content of methionine.

**References:**