

UltraClear Plus and AdvaClear for Balanced Detoxification:

How Does the Body Remove Toxic Substances?

In order to remove the toxins, the body has a complex system that converts them into non-toxic molecules for removal. This complex system occurs in two phases: Phase I and Phase II, that together convert a toxic molecule into a non-toxic molecule that can be easily excreted. The majority of detoxification occurs in the liver; however, all tissues have some ability to detoxify, including the intestines, skin, and lungs.

Nutritional Support for Detoxification

Detoxification is an energy-requiring process that puts a metabolic burden on the body. Therefore, water or juice fasts are not beneficial because they deplete the body of the essential nutrients required for healthy detoxification. These fasts can have many adverse health effects, including decreased energy production, breakdown of lean tissue instead of fat, increased oxidative stress, and unbalanced detoxification.^{15,16}

UltraClear Plus supports Phase 1 and 2 Detox Pathways:

In Phase I, a functional group is added to the toxic molecule producing an intermediate that needs to be further transformed. Phase II detoxification involves a process called conjugation, in which various enzymes in the liver attach protective compounds to the intermediate, making it less harmful and more readily excretable. Instead of decreasing nutrient support, a focused, high-impact, low-allergy-potential source of macronutrients should be provided.

- High-quality protein provides methionine and cysteine, which are beneficial to Phase II and may help with toxic metal burdens.¹⁷
- Medium chain triglycerides (MCTs) support energy production,¹⁸ and olive oil may protect against chemically-induced liver damage.¹⁹
- Fibre supports fecal excretion of toxins and the integrity of the intestinal barrier, which decreases toxic burden. In particular, rice bran can directly bind some toxins, thereby removing them before they can enter the body and cause damage.²⁰
- Antioxidant nutrients protect from oxidative stress include vitamins C and E, zinc, selenium, and copper.^{12,13}
- Nutrients that support energy production include vitamin B1 (thiamin), vitamin B2 (riboflavin), niacin, vitamin B5 (pantothenic acid), and magnesium.

AdvaClear is a bifunctional modulator:

Because the products of Phase I can be highly reactive and more harmful than the original compound, achieving and maintaining a balance between the Phase I and Phase II processes is critical. Furthermore, a significant side effect of all this metabolic activity is the production of free radicals as the toxins are transformed, resulting in oxidative stress. Optimal detoxification requires that both Phase I and Phase II pathways function optimally and in balance with each other.

Bifunctional modulators are phytonutrients that support balanced detoxification by modulating Phase I and promoting Phase II. This minimizes damage by reactive intermediates and free radicals. Fruits and vegetables contain many bifunctional modulators, which is one reason these foods are associated with reduced susceptibilities to cancer and degenerative diseases.¹⁴

In addition, the following nutrients and phytonutrients provide targeted support for optimal detoxification:

- N-Acetylcysteine and Sodium Sulfate promote generation of glutathione, which is used in Phase II and is a major route for detoxification of heavy metals, and supports Phase II sulfation.^{16,21}
- Vitamin B12, Folate, Methionine, and Choline promote balanced detoxification by supporting Phase II methylation and healthy homocysteine recycling. Choline deficiency is causative for liver disease, and is a newly-designated essential nutrient.²²⁻²⁴ The biologically-active, natural form of folate is 5-methyltetrahydrofolate.²⁵
- Ellagic Acid from pomegranate significantly reduces tumors in animals with chemically induced cancers, protects from toxin liver damage, enhances glutathione production, decreases lipid peroxidation, and binds some metals, thus promoting their excretion.²⁶⁻²⁹ It is a bifunctional modulator that can bind some toxins directly, rendering them non-toxic, and can directly bind and protect DNA.^{30,31}
- Catechins from green tea are bifunctional modulators that are strong antioxidants possessing anticarcinogenic and antimutagenic potential.^{32,33} Catechins are associated with lower incidence of Parkinson's disease.^{33,34} The National Cancer Institute is currently investigating the chemotherapeutic potential of green tea catechins.³⁵ Catechins also promote healthy gastrointestinal function.³⁶
- Watercress (*Nasturtium officinale*) contains high levels of glucosinolates, which are precursors to several bioactives that can inhibit chemically-induced cancers in animals, and promote excretion of carcinogens in humans.³⁷⁻⁴¹ The bifunctional activity of watercress is one of the proposed mechanisms for its chemoprotective effect.^{37,42-44}
- Silymarin from milk thistle is a well-known liver-protectant that may improve liver function in patients with liver disease and toxicity.⁴⁵⁻⁴⁷ Silymarin increases glutathione and is a strong antioxidant.⁴⁶⁻⁴⁹
- Artichoke (*Cynara scolymus*) is also a liver-protectant with a long history of traditional use that provides strong antioxidant protection and may decrease the loss of glutathione after toxic exposure.⁵⁰⁻⁵³

References

1. Bachurin SO, Tkachenki SE, Lermontova NN. Pyridin derivatives: structure-activity relationships causing parkinsonism-like symptoms. *Rev Environ Contam Toxicol* 1991;122:1-36.
2. Buckley JD, Meadows AT, Kadin ME, et al. Pesticide exposures in children with non-Hodgkin lymphoma. *Cancer* 2000;89(11): 2315-21.
3. Meinert R, Schuz J, Kaletsch U, et al. Leukemia and non-Hodgkin's lymphoma in childhood and exposure to pesticides: results of a register-based case-control study in Germany. *Am J Epidemiol* 2000;151(7):639-46, 647-50.
4. Rothman N, Cantor KP, Blair A, et al. A nested case-control study of non-Hodgkin lymphoma and serum organochlorine residues. *Lancet* 1997;350:240-44.
5. Dunstan RH, Donohoe M, Taylor W, et al. A preliminary investigation of chlorinated hydrocarbons and chronic fatigue syndrome. *Med J Aust* 1995;163(6):294-97.
6. Bell IR, Baldwin CM, Schwartz GE. Illness from low levels of environmental chemicals: relevance to chronic fatigue syndrome and fibromyalgia. *Am J Med* 1998;105(3A):745-825.
7. Salonen JT, Seppanen K, Lakka TA, et al. Mercury accumulation and accelerated progression of carotid atherosclerosis: a population-based prospective 4-year follow-up study in men in eastern Finland. *Atherosclerosis* 2000;148:265-73.
8. Racciatti D, Vecchiet J, Ceccomancini A, et al. Chronic fatigue syndrome following a toxic exposure. *Sci Total Environ* 2001;270(1-3):27-31.
9. Wang C-H, Jeng J-S, Yip P-K, et al. Biological gradient between long-term arsenic exposure and carotid atherosclerosis. *Circulation* 2002;105:1804-09.
10. Kirkhorn SR, Schenker MB. Current health effects of agricultural work: respiratory disease, cancer, reproductive effects, musculoskeletal injuries, and pesticide-related illnesses. *J Agric Saf Health* 2002;8(2):199-214.
11. Liska DJ. The detoxification enzyme systems. *Altern Med Rev* 1998;3(3):187-98.
12. Aw TY, Jones DP. Nutrient supply and mitochondrial function. *Annu Rec Nutr* 1989;9:229-51.
13. Bland JS, Bralley JA. Nutritional upregulation of hepatic detoxification enzymes. *J Appl Nutr* 1992;44:2-15.
14. Nestle M. Broccoli sprouts in cancer prevention. *Nutr Rev* 1998;56(4 Pt 1):127-30.
15. Lall SB, Singh B, Gulati K, et al. Role of nutrition in toxic injury. *Indian J Exp Biol* 1999;37(2):109-16.
16. Bland JS, Barrager E, Reedy G, et al. A medical food-supplemented detoxification program in the management of chronic health problems. *Altern Ther Health Med* 1995;1(5):62-71.
17. Quig D. Cysteine metabolism and metal toxicity. *Altern Med Rev* 1998;3(4):262-70.
18. DeGaetano A, Castagneto M, Mingrone G, et al. Kinetics of the medium-chain triglycerides and free fatty acids in healthy volunteers and surgically stressed patients. *J Parenteral Enteral Nutr* 1994;18:134-40.
19. McDanell RE, Henderson LA, Russell K, et al. The effect of Brassica vegetable consumption on caffeine metabolism in humans. *Human Exp Toxicol* 1992;11:167-72.
20. Harris PJ, Sasidharan VK, Robertson AM, et al. Adsorption of a hydrophobic mutagen to cereal brans and cereal bran dietary fibres. *Mutation Res* 1998;412:323-31.
21. Olmstead MJ. Heavy metal sources, effects, and detoxification. *Altern Ther Complement Med* 2000;Dec:347-54.
22. Buchman AL, Ament ME, Sohler M, et al. Choline deficiency causes reversible hepatic abnormalities in patients receiving parenteral nutrition: Proof of a human choline requirement: A placebo-controlled trial. *J Parenteral Enteral Nutr* 2001;25:260-68.
23. Zeisel SH. Choline: an essential nutrient for humans. *Nutrition* 2000;16:669-71.
24. Miller DL. Health benefits of lecithin and choline. *Cereal Foods World* 2002;47:178-84.
25. Scott J. Methyltetrahydrofolate: the superior alternative to folic acid. In: Krhamer K, Hoppel P-P, eds. *Nutraceuticals in Health and Disease Prevention*. New York: Marvel Dekker, 2001;6:75-90.
26. Khanduja KL, Gandhi RK, Pathania V, et al. Prevention of N-nitrosodiethylamine-induced lung tumorigenesis by ellagic acid and quercetin in mice. *Food Chem Toxicol* 1999;37(4):313-18.
27. Anderson KE, Kappas A. Dietary regulation of cytochrome P450. *Annu Rev Nutr* 1991;11:141-67.
28. Ahn D, Putt D, Kresty L, et al. The effects of dietary ellagic acid on rat hepatic and esophageal mucosal cytochromes P450 and phase II enzymes. *Carcinogenesis* 1996;17(4):821-28.
29. Ahmed S, Rahman A, Saleem M, et al. Ellagic acid ameliorates nickel induced biochemical alterations: diminution of oxidative stress. *Human Exp Toxicol* 1999;18:691-98.
30. Barch DH, Rundhaugen LM, Stoner GD, et al. Structure-function relationships of the dietary anticarcinogen ellagic acid. *Carcinogenesis* 1996;17(2):265-69.
31. Barch DH, Rundhaugen LM, Pillay NS. Ellagic acid induces transcription of the rat glutathione S-transferase-Ya gene. *Carcinogenesis* 1995;16(3):665-68.
32. Ahmad N, Muktar H. Green tea polyphenols and cancer: biological mechanisms and practical implications. *Nutr Rev* 1999;57(3):78-83.
33. McKay DL, Blumberg JB. The role of tea in human health: an update. *J Am Coll Nutr* 2002;21(1):1-13.
34. Ross GW, Abbott RD, Petrovitch H, et al. Association of coffee and caffeine intake with the risk of Parkinson disease. *JAMA* 2000;283:2674-79.
35. Steele VE, Kelloff GJ, Balentine D, et al. Comparative chemopreventive mechanisms of green tea, black tea, and selected polyphenol extracts measured by in vitro bioassays. *Carcinogenesis* 2000;21(1):63-67.
36. Goto K, Kanaya S, Ishigami T, et al. The effects of tea catechins on fecal conditions of elderly residents in a long-term care facility. *J Nutr Sci Vitaminol* 1999;45:135-41.
37. Getahun SM, Chung F-L. Conversion of glucosinolates to isothiocyanates in humans after ingestion of cooked watercress. *Cancer Epidemiol Biomarkers Prev* 1999;8:447-51.
38. Krul C, Humblot C, Phillippe C, et al. Metabolism of sinigrin (2-propenyl glucosinolate) by the human colonic microflora in a dynamic in vitro large-intestine model. *Carcinogenesis* 2002;23:1009-16.
39. Hecht SS. Chemoprevention of cancer by isothiocyanates, modifiers of carcinogen metabolism. *J Nutr* 1999;129:768S-74S.
40. Chung FL, Conaway CC, Rao CV, et al. Chemoprevention of colonic aberrant crypt foci in Fischer rats by sulforaphane and phenethyl isothiocyanate. *Carcinogenesis* 2000;21(12):2287-91.
41. Hecht SS, Carmella SG, Murphy SE. Effects of watercress consumption on urinary metabolites of nicotine in smokers. *Cancer Epidemiol Biomarkers Prev* 1999;8:907-13.
42. Rose P, Faulkner K, Williamson G, et al. 7-Methylsulfinylheptyl and 8-methylsulfinyloctyl isothiocyanates from watercress are potent inducers of phase II enzymes. *Carcinogenesis* 2000;21(11):1983-88.
43. Leclercq J, Desager JP, Horsmans Y. Inhibition of chlorzoxazone metabolism, a clinical probe for CYP2E1, by a single ingestion of watercress. *Clin Pharmacol Ther* 1998;64(2):144-49.
44. Hecht SS, Chung F-L, Richie JP Jr, et al. Effects of watercress consumption on metabolism of a tobacco-specific lung carcinogen in smokers. *Cancer Epidemiol Biomarkers Prev* 1995;4:877-84.
45. Saller R, Meier R, Brignoli R. The use of silymarin in the treatment of liver diseases. *Drugs* 2001;61(14):2035-63.
46. Wellington K, Jarvis B. Silymarin: a review of its clinical properties in the management of hepatic disorders. *BioDrugs* 2001;15(7):465-89.
47. DeLeve LD, Kaplowitz N. Glutathione metabolism and its role in hepatotoxicity. *Pharmacol Ther* 1991;52(3):287-305.
48. Yanaida Y, Kohno H, Yoshida K, et al. Dietary silymarin suppresses 4-nitroquinoline 1-oxide-induced tongue carcinogenesis in male F344 rats. *Carcinogenesis* 2002;23(5):787-94.
49. Kosina P, Kren V, Gebhardt R, et al. Antioxidant properties of silybin glycosides. *Phytotherapy Res* 2002;16:533-539.
50. Perez-Garcia F, Adzet T, Canigueral S. Activity of artichoke leaf extract on reactive oxygen species in human leukocytes. *Free Rad Res* 2000;33:661-65.
51. Llorach R, Espin JC, Thomas-Barberan FA, et al. Artichoke (*Cynara scolymus* L.) byproducts as a potential source of healthpromoting antioxidant phenolics. *J Agric Food Chem* 2002;50:3458-64.
52. Rechner AR, Pannala AS, Rice-Evans CA. Caffeic acid derivatives in artichoke extract are metabolised to phenolic acids in vivo. *Free Rad Res* 2001;35:195-202.
53. Gebhardt R. Antioxidant and protective properties of extracts from leaves of the artichoke (*Cynara scolymus* L.) against hydroperoxide-induced oxidative stress in cultured rat hepatocytes. *Toxicol Appl Pharmacol* 1997;144:279-86.