DetoxiGenomic™ Profile

Physician's Copy

Genova Diagnostics® Europe

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PHASE I Detoxification: The First Line of Defense

In Phase I detoxification, enzymes, known collectively as the cytochrome P-450 system, use oxygen to modify toxic compounds, drugs, or steroid hormones. Many toxins must undergo Phase II detoxification after a reactive site has been formed. Because there are many different toxic compounds the body might encounter, there are many variants of Phase I enzymes.

(CYP1A1) detoxifies polycyclic aromatic hydrocarbons (PAHs) produced from the combustion of organic materials (exhaust fumes, charbroiled meats, etc.).

(CYP1B1) is involved in the 4-hydroxylation of estrogen.

(CYP2A6) detoxifies nitrosamines and nicotine

(CYP2C9) detoxifies coumadin® and sulfonylureas.

(CYP2C19) detoxifies proton-pump inhibitors (e.g., prilosec®) and many anticonvulsants (e.g., valium®).

(CYP2D6) detoxifies ~20% of all prescription drugs including tricyclics, MAOIs, SSRIs, opiates, anti-arrhythmics, beta-blockers, Cimetidine, etc.

(CYP2E1) detoxifies nitrosamines and ethanol (acetaldehyde).

(CYP3A4) detoxifies over 50% of all prescription medications and most steroid hormones.

Cytochrome P-450		
Result	Gene	internet information
●	**CYP1A1** *	www.genovations.com/gdgen01
●	**CYP1B1** *	www.genovations.com/gdgen02
✔	**CYP2A6**	www.genovations.com/gdgen10
●	**CYP2C9** *	www.genovations.com/gdgen05
✔	**CYP2C19** *	www.genovations.com/gdgen06
✔	**CYP2D6**	www.genovations.com/gdgen03
✔	**CYP2E1**	www.genovations.com/gdgen04
✔	**CYP3A4** *	www.genovations.com/gdgen07

Use of H2 blockers (e.g. Cimetidine) should be avoided as these bind to the heme-containing reactive site of all CYPs inhibiting binding to toxins.

Your Results: Polymorphisms (SNPs) in the genes coding for a particular enzyme can increase or, more commonly, decrease the activity of that enzyme. Both increased and decreased activity may be harmful. Increased phase I clearance without increased clearance in Phase II can lead to the formation of toxic intermediates that may be more toxic than the original toxin. Decreased Phase I clearance will cause toxic accumulation in the body. Adverse reactions to drugs are often due to a decreased capacity for clearing them from the system.

General Therapies to Improve Detoxification:

Foods that generally improve Phase I detoxification and as well improve the efficiency of Phase II conjugation are generally recommended for individuals with CYP SNPs. These include most vegetables and fruits, but especially cruciferous vegetables (broccoli, Brussels sprouts, cauliflower, watercress, and cabbage), garlic, onions, soy, grapes, berries, green and black tea, and many herbs and spices like rosemary, basil, turmeric, cumin, poppy seeds, and black pepper. Indeed, improving Phase I and Phase II detoxification helps explain why vegetables and fruits protect against many cancers.

Key

✔	Optimal genomic potential - no polymorphism detected
●	Polymorphism detected in this enzyme, increasing your susceptibility to toxins, if exposed
*	Multiple SNP locations were evaluated for these genes
NR	See commentary if applicable.

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Innovative Testing for Optimal Health

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PHASE II Detoxification: Conjugation of Toxins and Elimination

In Phase II detoxification, large water-soluble molecules are added to toxins, usually at the reactive site formed by Phase I reactions. After Phase II modifications, the body is able to eliminate the transformed toxins in the urine or the feces (through the bile).

(COMT SNP) higher risk for depression, bipolar disorder, ADHD and alcoholism.

Methylation

Result	Gene	SNP Location	Internet Information	Affects
++	COMT	V158M	www.genovations.com/gdv158m	Liver/Gut

(NAT SNP) both slow and rapid acetylators are at increased risk for developing lung, colon, bladder, or head & neck cancer.

(GST SNP) The GST isoforms (M1, P1, and T1) are more or less prevalent in various tissues; all catalyze the conjugation of electrophilic compounds with glutathione. Defects in GST activity can contribute to fatigue syndromes, and to various cancers throughout the body.

Acetylation (N-acetyl transferase)

SLOW METABOLIZER POLYMORPHISM

Result	Gene	SNP Location	Internet Information	Affects
- -	NAT1	R64W	www.genovations.com/gdr64w	All Cells
- -	NAT1	R187Q	www.genovations.com/gdr187q	Liver/Gut
- -	NAT2	I114T	www.genovations.com/gdi114t	Liver/Gut
++	NAT2	R197Q	www.genovations.com/gdr197q	Liver/Gut
- -	NAT2	G286E	www.genovations.com/gdg286e	Liver/Gut
- -	NAT2	R64Q	www.genovations.com/gdr64q	Liver/Gut

FAST METABOLIZER POLYMORPHISM

- -	NAT2	K268R	www.genovations.com/gdk268r	Liver/Gut

Glutathione Conjugation (Glutathione s-transferase)

Result	Gene	Location	Internet Information	Affects
PRESENT	GSTM1	1p13.3	www.genovations.com/gdgstm1	Liver/Kidney
+-	GSTP1	I105V	www.genovations.com/gdgstp1	Brain/Skin
- -	GSTP1	A114V	www.genovations.com/gda114v	Brain/Skin

(SOD SNP) SOD1 is present in the cytosol; SOD2 is present in the mitochondria. Changes in the SOD enzyme are associated with changes in risk for neurodegenerative disorders like ALS.

Oxidative Protection

Result	Gene	SNP Location	Internet Information	Affects
- -	SOD1	G93A	www.genovations.com/gdg93a	Cytosol
- -	SOD1	A4V	www.genovations.com/gda4v	Cytosol
- -	SOD2	A16V	www.genovations.com/gda16v	Mitochondria

Your Results: Catechol-O-methyl transferase is the enzyme primarily responsible for breaking down the neurotransmitters dopamine, epinephrine, and norepinephrine.

Your Results: N-acetyl Transferase detoxifies many environmental toxins, including tobacco smoke and exhaust fumes. Polymorphisms can result in slower than normal or faster than normal addition of an acetyl group to these toxins. Slow acetylators have a build up of toxins in the system and rapid acetylators add acetyl groups so rapidly that they make mistakes in the process. Both slow and rapid acetylators are at increased risk for toxic overload if they are exposed to environmental toxins. If the toxin exposure is reduced, the risk is reduced.

Your Results:
Glutathione-S-transferase detoxifies many water-soluble environmental toxins, including many solvents, herbicides, fungicides, lipid peroxides, and heavy metals (e.g., mercury, cadmium, and lead). The various forms of GST work together to eliminate toxins. Decreased glutathione conjugation capacity may increase toxic burden and increase oxidative stress.

Your Results:
Superoxide Dismutase is an enzyme that protects cells from increased oxidative stress and free radical damage to cell structures like membranes, mitochondria, DNA, and proteins.

Key			
	- -	Neither chromosome carries the genetic variation.	Homozygous negative or wild type
	+ -	One chromosome (of two) carries the genetic variation.	Heterozygous positive
	++	Both chromosomes carry the genetic variation.	Homozygous positive
	(You inherit one chromosome from each parent)		

This test has been developed and its performance characteristics determined by Genova Diagnostics, Inc. It has not been cleared or approved by the U.S. Food and Drug Administration.

Commentary is provided to the practicioner for educational purposes, and should not be interpreted as diagnostic or treatment recommendations. Diagnosis and treatment decisions are the responsibility of the practicioner.

The accuracy of genetic testing is not 100%. Results of genetic tests should be taken in the context of clinical representation and familial risk. The prevalence and significance of some allelic variations may be population specific.

Any positive findings in your patient's test indicate genetic predisposition that could affect physiologic function and risk of disease. We do not measure every possible genetic variation. Your patient may have additional risk that is not measured by this test. Negative findings do not imply that your patient is risk-free.

The Third Wave ™ Invader DNA assay is used to detect polymorphisms in the patient's DNA sample. In this assay, a solution hybridization method is used in which two oligonucleotides hybridize in tandem with the specific DNA sequences. Subsequent Cleavase® and hybridization reactions result in generation of fluorescent signal. The biplex format of the assay enables simultaneous detection of all variants in a single reaction tube. The sensitivity and specificity of this assay is 99.7%.

Dr. Amy Peace-Brewer
Laboratory Director

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Phase I Detoxification (Commentary for polymorphisms may not appear in this section unless the polymorphism has been indicated to have impaired activity.)

Note: In the following charts, substrates, inhibitors, and inducers are listed for each cytochrome P450 enzyme (Phase I) included in the DetoxiGenomic Profile.
Substrates are compounds that are metabolized by that enzyme. The metabolism of some of these compounds is shared by other P450 enzymes (refer to chart).
Inhibitors may or may not be substrates of that enzyme, but will reliably reduce that enzyme's activity if present.
Inducers also may or may not be substrates, but will tend to increase the enzyme's activity.

Drug Interaction Resources
http://medicine.iupui.edu/flockhart/table.htm

CYP1A1 www.genovations.com\gdgen01

There are 2 SNPs measured in this gene that predict risk. In this patient, the specific variants are MspI positive and I462V negative. The commentary below reflects these results. Please refer to the drug pathway chart on the following page.

Health Implications: Cytochrome P450 1A1 is responsible for the metabolism of estrogen and certain medications, as well as the activation of numerous environmental toxins such as polycyclic aromatic hydrocarbons (e.g., cigarette smoke, car exhaust, charbroiled meats) and chlorinated benzenes (solvents). Polymorphisms convey a higher capacity for induction with toxin exposure, thus greater activation and potential toxicity of these compounds.
The 2-hydroxyestrogens produced by CYP1A1 are protective against breast cancer when further methylated, but may be carcinogenic when not. Hyperinduction of CYP1A1 also generates mutagenic metabolites, increasing the risk of cancers of the lung, ovary (in smokers), prostate, and colon (in smokers). This SNP has been associated with both decreased and increased risk (in smokers) of breast cancer. Female smokers with the SNP show higher levels of DNA damage than either non-smokers or women without the SNP. The CYP1A1 SNP is also associated with moderately increased risk of systemic lupus erythematosus and endometriosis (although studies are inconsistent).

Minimizing Risk: Do not smoke. Minimize exposure to charbroiled and well-done meats, tobacco smoke, car and diesel exhaust, industrial solvents, dioxin-contaminated meats and dairy, incineration, and PVC plastics. Excess exposure to these compounds can generate free radicals and reactive compounds that can increase your long-term risk of developing some cancers. Emphasize a diet rich in anti-oxidants (colorful fruits and vegetables). Extra protection may be afforded by cruciferous vegetables (e.g., broccoli and cauliflower) and green tea, especially in smokers.
DNA damage from reactive intermediates may also be minimized by rosemary, epigallocatechin gallate (EGCG), curcumin, resveratrol, genistein, hops, vitamin E, and DHEA. Support glutathione conjugation with precursors and cofactors.

Substrates		Inhibitors	Inducers
Chlorinated Benzenes	Ondansetron	Alpha-naphthoflavone	***Polycyclic Aromatic Hydrocarbons:***
Heterocyclic amines	Phenacetin	Amiodarone	E.g., cigarette smoke, charbroiled foods
Polycyclic aromatic hydrocarbons, (e.g., benzo(a)pyrene)	Propanolol	Cimetidine	***Heterocyclic amines:***
	Riluzole	Fluoroquinolones	E.g., fried meat
	Ropivacaine	Fluvoxamine	3-methylcholanthrene (carcinogen)
Acetaminophen	Sparteine (mostly 2D6)	Furafylline	
Acetanilide	Tacrine	Interferon	Atorvastatin
Antipyrine	Tamoxifen	Mexiletine	Beta-naphthoflavone
Bufuralol	Testosterone	Methoxsalen	Flutamide
Caffeine	Theophylline	Mibefradil	Leflunomide
Chlorzoxazone	Verlukast	Propofol	Methyl cholanthrene
Coumarin activation (parts of)	Warfarin	Quinidine	Minodipine
Cyclobenzaprine (Flexeril)	Zoxazolamine	Safrole (e.g., root beer)	Omeprazole
7-ethoxyresorufin		Tacrine	
Dextromethorphan		THC	Cruciferous vegetables (including I3C and DIM)
Diethylstilbestrol		Apigenin	
Erlotinib (minor)		Benzoflavone	
Estradiol		Quercetin	
Gefitinib		Grapefruit	
Granisetron		Turmeric/Curcumin (animal & in-vitro studies)	
Haloperidol			
Lidocaine			

Physician Recommendations:

CYP1B1 www.genovations.com\gdgen02

There are 2 SNPs measured for this gene that predict risk. In this patient, the specific variants are L432V +/- and N453S negative. The commentary below reflects these results.

Health Implications: Cytochrome P450 1B1 is responsible for the 4-hydroxylation of estrogen as well as the activation of common environmental toxins such as polycyclic aromatic hydrocarbons (e.g., products from cigarette smoke, car exhaust, and charbroiled foods), polychlorinated biphenyls (e.g., PCBs), and aflatoxin B1. Polymorphisms convey a higher capacity for induction with toxin exposure, thus greater activation and potential toxicity of these compounds and greater production of 4-hydroxyestrogens.
Hyperinduction can generate oxidative stress and the 4-hydroxyestrogens may convert to quinone compounds that can cause DNA damage in breast tissue. Polymorphisms have been associated with lower 2:16α-hydroxyestrone ratios and increased risk of breast cancer, especially if xenobiotic exposure, high body mass index, long-term HRT, or concomitant CYP1A1 polymorphism. Risk is also increased for cancers of the ovary, prostate, lung and head & neck, especially in smokers.

Minimizing Risk: Do not smoke. Minimize exposure to xenobiotics (e.g., polycyclic aromatic hydrocarbons), also xenoestrogens (e.g., organochlorines), which tend to increase CYP1B1 activity. Eat a diet rich in antioxidants; consider supplementation. Redirect estrogen metabolism away from 4-hydroxylation with cruciferous vegetables and/or agents such as indole 3-carbinol (I3C), diindolylmethane (DIM), fish oils or rosemary.
Use caution with long-term HRT, especially conjugated equine estrogens which are preferentially 4-hydroxylated.

Substrates	Inhibitors	Inducers
Polycyclic aromatic hydrocarbons, (e.g., benzo(a)pyrene) **Antidepressants:** Amitryiptyline (Elavil) Clomipramine (Anafranil) Imipramine (Tofranil) Acetaminophen (NAPQI) Caffeine Clozapine (Clazaril) Coumarin activation Estradiol, Estrone (4-hydroxylation) Heterocyclic amines Naproxen Propranolol (Inderal) Resveratrol Tacrine (Cognex) Testosterone Theophylline	Cimetidine Ciprofloxacin (Cipro) Erythromycin Fluvoxamine (Luvox) Pyrene Ticlopidine Grapefruit juice (naringenin) Ginseng (possible)	Omeprazole (Prilosec) Phenytoin (Dilantin) Phenobarbital Rifampin ***Polycyclic Aromatic Hydrocarbons:*** Cigarette smoke Charbroiled foods
CYP1B1: Up regulator - is involved in the 4-hydroxylation of estrogen.		

Physician Recommendations:

CYP2C9 www.genovations.com\gdgen05

Health Implications : Cytochrome P450 2C9 is involved in the metabolism of many drugs including blood thinners like Coumadin ®. Polymorphisms may prevent the normal metabolism of these drugs and side effects are possible. Please refer to the drug pathway chart on the following page.

Minimizing Risks: Your health care provider has a list of drugs cleared through CYP2C9. Consult your physician. You may still need these drugs, but your physician may opt to prescribe a smaller therapeutic dose. Should you need to be placed on a blood thinning agent in the future, make sure your physician knows you have a genetic polymorphism that impairs your ability to break down Coumadin ®. If you are taking aspirin to reduce the risk of colon cancer, switch to a non-aspirin alternative.

Substrates		Inhibitors		Inducers
NSAIDs	**Miscellaneous Continued**	**Anti-depressants**	**Miscellaneous Continued**	Aminoglutethimide
Diclofenac	Febuxostat	Fluvoxamine (Luvox)	Imatinib	Aprepitant
Ibuprofen	Fluoxetine	Paroxetine (Paxil)	Isoniazid	Barbiturates
Lomoxicam	Flurbiprofen	Sertraline (Zoloft)	Leflunomide	Bosentan
Meloxicam	Fluvastatin	Fluoxetine (Prozac)	Lovastatin	Carbamazepine
S-Naproxen	Formoterol		Metronidazole (Flagyl)	Ethanol
Piroxicam	Glyburide	**Azole Antifungals**	Omeprazole	Griseovulfin
Suprofen	Hexobarbital	Itraconazole (Sporonox)	Phenylbutazone	Phenobarbital
	Hyzaar	Ketoconazole (Nizoral)	Phenytoin (Dilantin)	Phenytoin
Oral Hypoglycemic Agents	Ibuprofen	Fluconazol (Diflucan)	Probenicid	Primidone
Tolbutamide	Imipramine (Tofranil)	Miconazole (Nystatin)	Retonavir (Norvir)	Rifabutin
Glipizide	Indomethacin	Voriconazole (Vfend)	Sulfa-methoxazole-Trimethoprim (Bactrim)	Rifampin
	Isoniazid		Sulfaphenazole	Rifapentine
Angiotensin II Blockers	Nateglinide	**Miscellaneous**	Sulfinpyrazone	Secobarbital
Losartan	Phenobarbital	Amiodarone	Teniposide	
Irbesartan	Phenytoin (Dilantin)	Cimetidine (Tagamet)	Ticlopidine	
	Piroxicam	Chloram-phenicol	Valproic acid (Depakote)	
Sulfonylureas	Retinoids	Clopidogrel (Plavix)	Zafirlukast	
Glyburide/glibenclamide	Rosiglitazone	Delavirdine		
Glipizide	Rosuvastatin (Crestor)	Disulfram	Echinacea	
Glimepiride	Sildenafil (Viagra)	Efavirenz	Garlic (possible)	
Tolbutamide	Sulfa Drugs	Etravirine	Kava kava	
	Sulfaphenazole	Fenofibrate	Milk thistle (in-vitro/ probably insignificant in-vivo)	
Miscellaneous	Suprofen	Fluorouracil	Saw palmetto (in-vitro)	
Alosetron (Lotronex)	Tamoxifen	Fluvastatin	St. John's wort (in-vitro studies)	
Amitriptyline (Elavil) (demethylation)	THC (marijuana)	Gemfibrozil		
Angiotensin	Torsemide (Demadex)			
Carvedilol	Valdecoxib			
Celecoxib	S-warfarin (active)			
Chloramphenicol	Zolpidem (Ambien, Edluar)			
Clomipramine	(mostly CYP3A4)			
Coumadin (Warfarin)				
Desogestrel				
Diazepam				
Diclofenac				
Dronabinol				
Etravirine				

Continued...

CYP2C9

Continued...

CYP2C9: Down regulator - detoxifies coumarin and suflonylureas.
Note: Individuals with deficient CYP2C9 activity may be anti-coagulated on 0.5mg of coumadin/day, as they cannot efficiently clear S-coumadin. ARBs in these people may be ineffective because a pro-drug like losartan may be poorly activated.

Physician Recommendations:

Phase II Detoxification commentary is provided only for polymorphisms with known health implications.

+ + COMT V158M www.genovations.com/gdv158m

Clinical Implications: Catechol-O-methyltransferase (COMT) inactivates catecholamines, catechol estrogens, and catechol drugs such as L-DOPA. A polymorphism in COMT results in reduced COMT activity, thus decreased degradation of these compounds. Risk may be increased for some neuropsychiatric disorders, impaired estrogen metabolism, cardiovascular problems, and increased sensitivity to pain.
Individuals with the (+/+) genotype have a 3-4-fold reduced clearance of catecholamines from neural synapses. As a result, risk is increased for anxiety, panic disorder, and ultra rapid cycling in bipolar disorder. Risk is also increased for fibromyalgia, breast cancer (esp. when coupled with cumulative estrogen exposure), hypertension (at least in men), and acute coronary events if also high homocysteine or heavy coffee consumption.

Minimizing Risks : Minimize sustained mental and environmental stress, as adrenaline levels may already be high. Stress hormones also require COMT for degradation, thus can decrease the methylation of estrogen compounds. Ensure adequate intake of B vitamins, magnesium, and protein.
Avoid high homocysteine (S-adenosylhomocysteine inhibits COMT). Consider betaine (TMG) along with the B vitamins, for remethylation of homocysteine. Ensure adequate antioxidants to prevent oxidation of pro-carcinogenic 4-hydroxyestrogens. Use caution with amphetamine-based medications and catechol drugs, also with conjugated equine estrogens (e.g., Premarin®), as 4-hydroxyequilenin is more likely to inhibit COMT in carriers of the polymorphism. In bipolar patients, use caution with MAO inhibitors, tricyclics, or stimulants including Ritalin. The anti-depressants mirtazapine (Remeron®) or paroxetine (Paxil®) may be less effective with this genotype. Parkinson's patients may response to lower doses of levodopa and benefit the most from vitamin B6.

Physician Recommendations:

+ + NAT2 R197Q www.genovations.com/gdr197q

Health Implications: N-acetyltransferase 1 is found in extra-hepatic tissues, while NAT2 is found predominantly in the liver and the gut. Both are used in the Phase II acetylation of numerous environmental toxins, including heterocyclic aromatic amines. Slow acetylators do not clear toxins well and the resulting increased total toxic burden can increase the risk of lung, colon, breast, bladder, and head and neck cancers, though results have not been consistent in all studies. Urinary bladder cancer appears to have the most consistent association with slow acetylation.

Minimizing Risk: If you smoke, stop. Your risk of lung cancer is substantially higher than someone with normal NAT activity. Even occasional smoking or exposure to second hand smoke is harmful. Liberal consumption of most vegetables and fruits but especially cruciferous vegetables (broccoli, Brussels sprouts, cauliflower, watercress, and cabbage), garlic, onions, soy, grapes and berries will increase Phase II efficiency, including acetylation.

Physician Recommendations:

PRESENT	**GSTM1**	1p13.3	www.genovations.com/gdgstm1
+ -	**GSTP1**	I105V	www.genovations.com/gdgstp1

Health Implications: Glutathione S-transferases (GST) are responsible for detoxifying certain products of oxidative stress and a variety of electrophilic xenobiotics and carcinogens such as solvents, herbicides, pesticides, polycyclic aromatic hydrocarbons, steroids, and heavy metals. GSTM1 is located primarily in the liver, whereas GSTP1 is located primarily in the brain and lungs.

The test indicates that the GSTM1 gene is present, although it is not clear whether the gene is present on one or both chromosomes. This suggests normal GSTM1 enzyme activity and hepatic detoxification of xenobiotics and toxic metals.

GSTP1 polymorphisms are associated with either higher or lower enzyme activity, depending on the exposure. This GSTP1 polymorphism is associated with increased risk of various cancers, risk that is compounded by exposure to cigarette smoke.

Minimizing Risk: Minimize exposure to cigarette smoke, charred food, herbicides, fungicides, insect sprays, industrial solvents, and toxic metals. Ensure availability of glutathione (GSH) precursors and cofactors, e.g., methionine, N-acetylcysteine, glutamine, glycine, magnesium, and pyridoxal-5-phosphate (B6). GSH depletion may be offset by alpha lipoic acid, milk thistle, and taurine. Allium vegetables (e.g., onions, leeks, garlic) and crucifers (e.g., broccoli, cauliflower, cabbage, kale, Brussels sprouts, radish sprouts) can increase GST activity and reduce cancer risk. Consume an antioxidant-rich diet to prevent oxidative stress.

Physician Recommendations:

- -	**SOD2**	A16V	www.genovations.com/gda16v

Health Implications: Superoxide dismutase (SOD) is the primary antioxidant enzyme within the mitochondria of cells (where most of our energy is made). SOD2 converts reactive oxygen species into less reactive hydrogen peroxide. The wild-type genotype (-/-) is associated with higher SOD2 activity and a greater sensitivity to antioxidant status compared to the other genotypes. The combination of higher SOD2 activity and low antioxidant intake and/or excessive oxidative stress (e.g., smoking) may result in an accumulation of hydrogen peroxide and increased risk of cancers of the breast or prostate. This genotype has also been associated with a higher risk of motor neuron disease. Risk of cancer may be *reduced* in individuals taking anti-oxidants.

Minimizing Risk: Because the (-/-) genotype is particularly sensitive to antioxidant status, liberal consumption of dietary antioxidants in colorful vegetables and fruits is recommended. Broad-spectrum anti-oxidant supplements may also be helpful, including agents that help to raise glutathione levels (e.g., vitamin C, N-acetylcysteine, milk thistle) and support glutathione peroxidase (selenium). Consult your health care provider to find the supplement regimen that best fits your overall health anti-oxidant needs.

Physician Recommendations: