## Detox Comprehensive Health Report





Welcome to the future of health and human potential

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CYP1A1 is a liver enzyme that catalyzes many reactions in drug metabolism and estrogen, and is induced by polycyclic aromatic hydrocarbons (PAH).

Benzopyrene is the most common PAH discussed in the literature regarding individual Phase I and Phase II detoxification alternations.

- You have the wild-type genotype for CYP1A1, improving the beginning phase of estrogen metabolism and assisting PAH detoxification
- The highest sources of PAHs come from cigarette smoke, burning coal, vegetable oils, smoked meat, and charred meat

## Phase I Detoxification LOW PRIORITY MEDIUM PRIORITY HIGH PRIORITY CYP1A2 C164A

CYP1A2 is a critical enzyme in caffeine metabolism, the 2-hydroxylation of the main estrogens, estrone, and estradiol, and the metabolic activity of heterocyclic amines, nitrosamines, aflatoxin B1, polycyclic aromatic hydrocarbons, dioxins, and beta-naphthoflavone.

- You have the homozygous genotype for CYP1A2, known as the ultrarapid metabolizer
- Excessive CYP1A2 activity without adequate phase II support may enhance the destructive effects of environmental procarcinogens
- Researchers have found that the inhibition activity of this enzyme may represent a logical strategy for preventing the development of human cancers induced by the aromatic and heterocyclic amines
- Heterocyclic amines are created by high heat reacting with proteins
- Blueberries, blackberries, red grapes, kiwi, watermelon, parsley, spinach, and hops have all been shown to inhibit the mutagenic activity of certain heterocyclic amines
- For men and women with the CYP1A2 ultra-rapid metabolizer genotype, coffee intake was found to be more protective against estrogen receptor-positive breast cancer and prostate cancer



The CYP1B1 gene metabolizes pro-carcinogens such as polycyclic aromatic hydrocarbons and 17 beta-estradiol. Due to the carcinogenic activation of polycyclic aromatic hydrocarbons and estrogens to genotoxic catechol estrogens – both of which cause DNA mutations – variants in the CYP1B1 gene are essential for breast, ovarian, colon, lung, and prostate health.

- You the wild-type genotype for CYP1B1 that is associated with increased CYP1B1 mRNA expression
- The wild-type genotype is associated with a subsequent elevation in 4hydroxy estradiol formation and reduced detoxification of polycyclic aromatic hydrocarbons, however, this has not been proven in human studies
- One study found that high-dose biotin supplementation increased CYP1B1 expression and was associated with an increase in the occurrence of single-stranded DNA breaks compared with biotindeficient cells
- Inhibition of CYP1B1 activity was observed with seaweed, celery, berries, rooibos tea, grapes, and dark roast coffee



CYP2A6 is expressed in the liver, lung, trachea, nasal mucosa, and sex organs such as the breast. This enzyme metabolizes oral contraceptives, dexamethasone, phenobarbital, and nicotine.

 You have the wild-type genotype for CYP2A6, associated with improved metabolism



 ${\tt CYP2C9*2}\ {\tt metabolizes}\ {\tt Warfarin, sulfonylurea, hypoglycemic\ drugs, NSAIDS, and\ vegetable\ oils.}$ 

 You have the wild-type genotype for CYP2C9\*2 C430T that is associated with improved metabolism



Variants in CYP2C9 rs1057910 may alter the metabolism of THC, the psychoactive compound found in cannabis.

 You have the wild-type genotype for CYP2C9\*3 that is associated with improved metabolism of THC



The CYP2C19 gene influences drug metabolism and catabolism of estrogens.

- You have the ultra-rapid metabolizer genotype for CYP2C19\*17
- Research suggests that the ultra-rapid metabolizers have increased catabolism of estrogens by CYP2C19 and may lead to decreased estrogen levels and, therefore, reduce breast cancer risk
- Women with CYP2C19\*17 T allele were associated with a decreased risk
  of breast cancer due to the increased metabolism of estrogen, thereby
  reducing the level of harmful estrogen metabolites
- The CYP2C19\*17 T allele decreased the risk of breast cancer in patients using hormone therapy



CYP2D6 metabolizes approximately 50% of drugs in clinical use.

 You have the wild-type CYP2D6\*10 T100C genotype associated with normal enzymatic activity



 ${\tt CYP3A4}\ converts\ estrogen\ into\ 16a-OHE1.\ Variants\ may\ increase\ 16a-OHE1\ levels\ and\ carcinogenic\ activity.$ 

• You have the wild-type genotype for CYP3A4\*1B, associated with improved 16a-OHE1 levels and reduced carcinogenic activity





The major oxidative routes of estrone and estradiol are 2– and 4–hydroxylation by cytochrome P450 CYP1A1, CYP1B1, CYP1A2 and CYP3A, while hydroxylated metabolites go through COMT to form 2 methyloxy–estradiol and 4 methyloxy–estradiol.

- You have the heterozygous genotype for COMT
- The heterozygous genotypes may have slightly higher estradiol levels and reduced estrogen clearance than the wild-type carriers
- A focus should be on reducing exposure to xenoestrogens with this genotype
- One benefit is that heterozygous genotypes retain polyphenols longer, obtaining a higher benefit with a lower intake
- Slowing this pathway down further with a high catecholamine intake combined with low fiber and magnesium intake negatively affects this pathway



NAT1 is a phase II cytosolic enzyme responsible for the activation or deactivation of many arylamine compounds, including pharmaceuticals and environmental carcinogens. NAT1 R187Q is the most common "slow acetylator" arylamine NAT1 genetic variant.

You have the wild-type genotype for NAT1\*14B R1870, improving arylamine carcinogen detoxification for the bladder and lungs



The NAT2 gene encodes an enzyme that activates and deactivates arylamine, hydrazine drugs, and carcinogens. The NAT2 phenotype can be classified as a slow, intermediate, or rapid acetylator. The slow acetylator genotype is associated with reduced detoxification.

- You do not have the slow acetylator genotype for NAT2
- Your genotype is associated with a reduced risk of bladder cancer in smokers and may improve the detoxification of aromatic amines found in commercial hair dyes, industrial and manufacturing plants, meat cooked at high temperatures, and diesel exhaust

## Phase II Detoxification LOW PRIORITY MEDIUM PRIORITY HIGH PRIORITY GSTM1

GSTM1 catalyzes the detoxification of alkyl and polycyclic aromatic hydrocarbons (PAHs), intermediate forms of many carcinogens, specifically metabolically generated epoxide intermediates of benzo(a)pyrene.

- Your genotype for GSTM1 is associated with improved detoxification of benzo(a)pyrene from the burning of wood or trash, tobacco smoke, asphalt, coal, diesel exhaust, and charred meat
- Your NAT1 and NAT2 genotype may increase or decrease this ability

## Phase II Detoxification LOW PRIORITY MEDIUM PRIORITY HIGH PRIORITY GSTP1 Ile105Val

Glutathione S-Transferase (GSTP1) encodes for the metabolism of mutagens, carcinogens, and other poisonous chemicals. It plays a crucial role in detoxification, thereby protecting cells from these compounds. GSTP1 rs1695 is connected to breast, prostate, urinary, esophagus, and skin health.

- You have the wild-type genotype for GSTP1 rs1695 that is associated with improved glutathione antioxidant protection for breast, lung, or prostate health
- Supplemental vitamin E as alpha-tocopherol in higher doses may be inflammatory for this genotype
- Your GSTP1 rs1138272 genotype may increase or decrease total glutathione protection



Glutathione S-Transferase (GSTP1) encodes for the metabolism of mutagens, carcinogens, and other poisonous chemicals. It plays a crucial role in detoxification, thereby protecting cells from these compounds. GSTP1 rs1138272 is connected to the colon, prostate, lung, throat, and fertility.

- You have the wild-type genotype for GSTP1 rs1138272 that is associated with improved glutathione antioxidant protection against heavy metals, pesticides, and air pollution for colon, prostate, lung, throat, and fertility health
- Your GSTP1 rs1695 genotype may increase or decrease this effect



The SOD1 gene provides instructions for making superoxide dismutase in the cytosol. SOD1 is zinc and copper dependent, and breaks down toxic, superoxide radicals.

 You have the wild-type genotype for SOD1, improving superoxide dismutation function in the cytosol of the cell



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The SOD2 provides instructions for making superoxide dismutase in the mitochondria. SOD2 is manganese dependent and breaks down toxic, superoxide radicals.

- You have the heterozygous genotype for SOD2
- Your mitochondria may have a higher sensitivity to glyphosate, fluoridated water, chronic stress, poor sleep, and shallow breathing
- Manganese, lycopene, vitamin C, milk thistle, reishi, cordyceps, and moderate exercise that encourages deep breathing all assist SOD2 function
- Flavonoids and black cumin seed oil have been shown to assist SOD by reducing superoxide





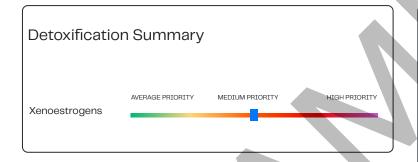
Glutathione is the master antioxidant system involved in oxidative stress, detoxification, and immunity. Glutathione status parallels telomerase activity, an important indicator of lifespan.

- Your genotype combinations are associated with increased baseline glutathione levels
- Research shows that people who live the longest have the bestpreserved antioxidant system and highest glutathione levels



Mycotoxins are toxic compounds that are naturally produced by certain types of fungi. Research suggests that mycotoxins can decrease the formation of glutathione due to decreased gene expression of the enzymes needed to form glutathione.

 Your genotype is associated with improved GSTM1 gene expression and may improve detoxification of mycotoxins unless other epigenetic factors are causing glutathione depletion



Xenoestrogens are synthetic hormone disruptors found in plastics and pesticides.

- Your genotype is associated with a slower metabolism of xenoestrogens, and therefore the damage may be greater from xenoestrogen exposure
- Increasing magnesium targets the enzyme responsible for assisting xenoestrogen detoxification

Detoxification Summary

AVERAGE PRIORITY MEDIUM PRIORITY HIGH PRIORITY

Benzo(a)pyrene

Benzo(a)pyrene is a carcinogenic compound produced from the burning of wood or trash, tobacco smoke, asphalt, coal, diesel exhaust, charred meat, and gas cooking.

 Your genotype combinations are associated with an improvement in the detoxification of benzo(a)pyrene



Aromatic amines are found in cigarettes, rubber factories, hair dyes that contain 4-aminobiphenyl, and meat cooked at high temperatures.

- Your genotype combinations are associated with a slightly below average detoxification ability of aromatic amines
- If your exposure is higher to aromatic amines, increase cruciferous vegetable intake, carotenoids, vitamin C, and use marinades for meat when barbecuing