Detoxification Health Report

Welcome to the future of health and human potential

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TEST METHODOLOGY AND LIMITATIONS

Recommendations in this report apply to all ages, however for any patient under 18 years, a guardian must purchase the test and be present for the report recommendations. The information in this report is not intended to treat, diagnose or cure any medical condition or disease.

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Phase I Detoxication

SNPs in phase I detoxication are the cytochrome P450 enzymes located mainly in the liver but also in enterocytes, kidneys, lungs, and even the brain. The function of the CYP450 enzymes is to add a reactive group such as a hydroxyl, carboxyl, or an amino group through oxidation, reduction, and hydrolysis. Phase I can create oxidative stress depending on the type of toxin, the amount, and the ability of the body to detoxify it.

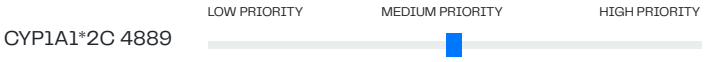
Phase I CYP450 liver enzymes are the rst defense employed by the body to biotransform xenobiotics, steroid hormones, and pharmaceuticals. Drugs can inhibit or induce CYP450 activity by directly interacting with the enzyme or altering its expression. The ability of an individual to metabolize 90% of currently used drugs will largely depend on the genetic

Phase II Detoxication

Phase II detoxication enzymes are known as the conjugation pathway, a process of adding a water-soluble group to this now reactive site where toxins are made less harmful and excreted by the body. Unlike phase I enzymes serving for activation metabolism, phase II enzymes deactivate and detoxify foreign compounds and are referred to as detoxication enzymes.

DETOXIFICATION

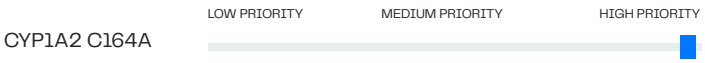
Phase I Detoxification



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.

- You have the heterozygous genotype for CYP1A1 that may affect the beginning phase of estrogen metabolism and polycyclic aromatic hydrocarbon detoxification
- For estrogen metabolism and detoxification, iodine, resveratrol, green tea, and decreasing exposure to polycyclic aromatic hydrocarbons have been shown to assist the pathway
- Epidemiological studies have demonstrated that a large proportion of cancer cases may be associated with dietary factors, including dietary exposure to PAHs
- Over 70% of polycyclic aromatic hydrocarbon exposure of non-smokers is associated with food consumption and is highly influenced by the growing environment and cooking techniques
- The highest sources of PAHs come from cigarette smoke, burning coal, vegetable oils, grains, vegetable oils, smoked meat, fried meat, and meat cooked over charcoal
- Marinating meat with citrus, tomatoes, grapes, dark beer, onion, garlic, and numerous herbs and spices with high antioxidant capacity has been shown to reduce PAHs by up to 80%
- There is evidence that intestinal microbiota modulates the activity of PAHs, with Lactobacillus bulgaricus EMCC 1102 showing the highest effectivity in PAHs bioremediation

Phase I Detoxification



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 ultra-rapid 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

Phase I Detoxification

CYP1B1*6 L432V

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.

- Your CYP1B1 genotype is not associated with increased CYP1B1 mRNA expression, improving 4-hydroxy estradiol and polycyclic aromatic hydrocarbon detoxification

Phase I Detoxification

CYP2A6 C164A

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

Phase I Detoxification

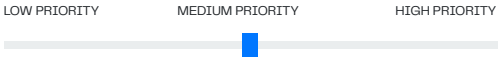
CYP2C9*2 C430T

CYP2C9 *2 metabolizes Warfarin, sulfonylurea, hypoglycemic drugs, NSAIDS, and vegetable oils.

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

Phase I Detoxification

CYP2C9*3 A1075C



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

- You have the heterozygous genotype for CYP2C9*3 associated with being a poor metabolizer of THC
- While the heterozygous genotype does not affect clearance as much as the homozygous genotype, this could create a higher sensitivity to THC, especially in the edible form
- For respiratory viruses, slow metabolizers of THC could be susceptible to viral activity if using THC

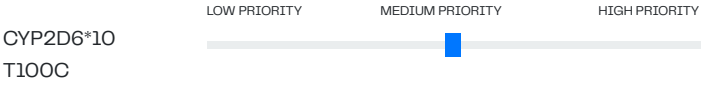
Phase I Detoxification

CYP2C19*17

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

Phase I Detoxification



CYP2D6*10
T100C

CYP2D6 metabolizes approximately 50% of drugs in clinical use.

- You have the heterozygous CYP2D6*10 genotype associated with being a poor metabolizer
- The polymorphism of CYP2D6 significantly affects the pharmacokinetics of about 50% of the drugs in clinical use, which are CYP2D6 substrates
- Please talk with your doctor about running a full CYP2D6 pharmacogenetic panel for a more complete assessment

Phase I Detoxification

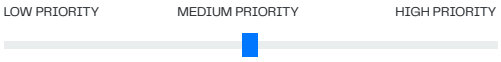
CYP3A4*1B

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

Phase II Detoxification

COMT V158M

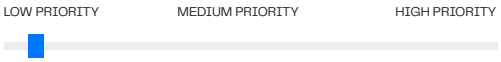


The major oxidative routes of estrone and estradiol are 2- and 4-hydroxylation by cytochrome P450 CYP1A1, CYP1B1, CYP1A2 and CYP3A, while hydroxylated metabolites go through COMT to form 2 methyloxy-estradiol and 4 methyloxy-estradiol.

- 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

Phase II Detoxification

NAT1 R187Q



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 do not have the slow acetylator genotype for NAT1 R187Q, improving arylamine carcinogen detoxification for the bladder and lungs

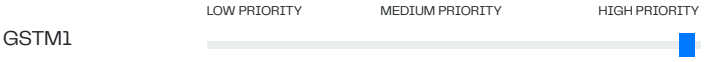
Phase II Detoxification

NAT2

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 have the homozygous, slow acetylator genotype for the NAT2 gene
- The slow acetylator is associated with a reduced detoxification of aromatic amines found in tobacco smoke, commercial hair dyes, industrial and manufacturing plants, charred meat, and diesel exhaust for bladder, prostate, and breast health
- Cruciferous vegetables, carotenoids, and vitamin C all assist NAT2 detoxification
- Marinades, cruciferous vegetables, unfiltered fermented drinks, blueberries, blackberries, red grapes, kiwi, watermelon, rosemary, and parsley all help reduce the carcinogenic risk posed by heterocyclic amines in meat cooked at high temperatures

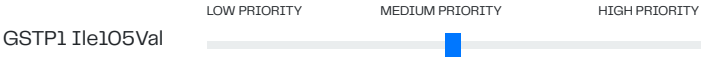
Phase II Detoxification



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.

- You have the wild-type genotype, known as a deletion or null genotype, that confers the absence of the GSTM1 protein
- The frequency of the null genotype varies from 20% to 80%, depending on the ethnic group studied
- The null genotype is less frequent in western and southern African populations, less frequent in South American populations, intermediate in Japanese, but is higher in Egyptian, European, American, and Asian populations
- While the GSTM1 null genotype has been associated with a greater sensitivity to benzo(a)pyrene, this genotype also retains a higher level of isothiocyanates, the anti-cancer compounds found in cruciferous vegetables
- Cruciferous vegetables, vitamin C, vitamin E, vitamin A, milk thistle, resveratrol, curcumin, green tea, and white tea all assist with the null genotype

Phase II Detoxification



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 heterozygous genotype for GSTP1 rs1695 that is associated with a higher sensitivity to mercury, cadmium, arsenic, pesticides, and air pollution for breast, prostate, urinary, esophagus, and skin health
- Selenium, vitamin C, vitamin E, milk thistle, and cruciferous vegetables all assist GSTP1 gene function
- While the heterozygous genotype for GSTP1 rs1695 is associated with a higher sensitivity to heavy metals, one advantage may be an increased VO2 max response from endurance training compared to the wild-type genotype

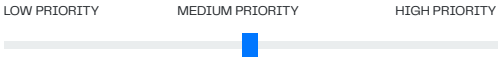
Phase II Detoxification

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

Phase II Detoxification

SOD2 V16A

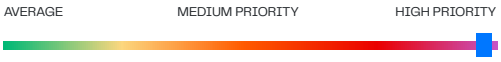


The SOD2 provides instructions for making superoxide dismutase in the mitochondria. SOD2 is manganese dependent and breaks down toxic, superoxide radicals.

- 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

Detoxification Summary

Glutathione Protection

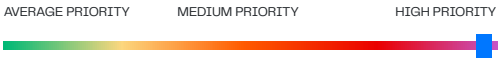


Glutathione is the master antioxidant system involved in oxidative stress, detoxification, and immunity. Glutathione status parallels telomerase activity, an important indicator of lifespan.

- Your genotype combinations are associated with decreased baseline glutathione levels
- Glutathione decreases with age, and low levels of glutathione are associated with chronic exposure to chemical toxins, heavy metals and excess alcohol, immunocompromised conditions, and neurodegenerative disorders
- Glutathione has been found to increase by 20% with deep breathing practices like Tai Chi or yoga
- For exercise, a combination of aerobic exercise and circuit weight training produced the highest glutathione effect
- Selenium, glycine, cysteine, vitamin C, and cruciferous vegetables all improve glutathione levels
- Chicken or bone broth, herbs, and spices are some of the best dietary ways to maintain higher levels of glutathione
- Some of the all-stars include cinnamon, anise, sage, and thyme due to also containing the antiviral compound caffeic acid

Detoxification Summary

Mycotoxins



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 lower glutathione levels which may cause glutathione depletion to occur at a faster rate and decrease mycotoxin detoxification
- The highest exposure to mycotoxins can be in foods grown or stored in damp conditions
- This may include grains, nuts, corn, coffee, wine, beer, grape juice, sorghum, rice, dried beans, apples, pulses, cacao products, and spices
- Boosting glutathione can be accomplished with selenium, glycine, cysteine, alpha lipoic acid, vitamin C, and cruciferous vegetables

Detoxification Summary

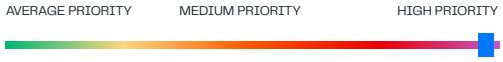
Xenoestrogens

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

Benzo(a)pyrene

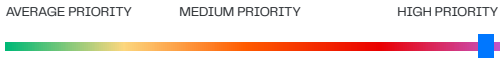


Benzo(a)pyrene is a carcinogenic compound produced from the burning of wood or trash, tobacco smoke, asphalt, coal, diesel exhaust, charred meat, and gas cooking.

- Your genotype combinations are associated with decreased detoxification of benzo(a)pyrene
- It is recommended to increase your intake of cruciferous vegetables, vitamin C, vitamin E, vitamin A, resveratrol, curcumin, green tea, and white tea to protect and detoxify benzo(a)pyrene

Detoxification Summary

Aromatic Amines



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 poor 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

The Detoxification panel was designed to address genetic susceptibilities from Phase I and Phase II detoxification SNPs of environmental procarcinogens, estrogen and xenoestrogens, THC metabolism, and baseline levels of endogenous antioxidants.

Industrial chemicals, including known carcinogens and their residues, have been detected in the blood and tissues of all populations. Over the past sixty years, evidence supports pollutants as a cause of damage to the central nervous, cardiovascular, renal, dermal, and reproductive systems, as well as a cause of cancer. Protecting and detoxifying these chemicals are highly dependent on genetics, metabolism, nutrition, the current environment, and chemical toxicity load.

Humans have synthesized more than 350,000 chemicals and mixtures of chemicals, most of which were non-existent before. The USA alone introduces an average of 1,500 new substances a year. These chemicals have been detected in the upper atmosphere, on the highest mountains, in the deepest oceans, from pole to pole, and in the most remote, uninhabited regions, in soil, water, air, and in the human food chain.Many of these chemicals are known to be toxic, even in small doses.

Chemical pollution has the potential to pose one of the largest environmental threats to humanity. The number of “silent” deaths caused by environmental pollution exceeds any other widely recognized risk factor.[1] More than nine million humans die prematurely each year—one in six deaths—due to contamination of their air, water, food, homes, workplaces, or consumer goods.

Phase I Detoxification

SNPs in phase I detoxification are the cytochrome P450 enzymes located mainly in the liver but also in enterocytes, kidneys, lungs, and the brain. The function of the CYP450 enzymes is to add a reactive group such as a hydroxyl, carboxyl, or an amino group through oxidation, reduction, and hydrolysis. Phase I can create oxidative stress depending on the type of toxin, the amount, and the ability of the body to detoxify it.

Phase I CYP450 liver enzymes are the first defense employed by the body to biotransform xenobiotics, steroid hormones, and pharmaceuticals. Drugs can inhibit or induce CYP450 activity by directly interacting with the enzyme or altering its expression. The ability of an individual to metabolize 90% of currently used drugs will largely depend on the genetic expression of these enzymes.

Phase II Detoxification

Phase II detoxification enzymes are known as the conjugation pathway, a process of adding a water-soluble group to this now reactive site where toxins are made less harmful and excreted by the body.

Unlike phase I enzymes serving for activation metabolism, phase II enzymes deactivate and detoxify foreign compounds and are referred to as detoxification enzymes.

Detoxification Panel Modules and SNPs

Phase I Detoxification

- CYP1A1*2C 4889
- CYP1A2 C164A
- CYP1B1*6 L432V
- CYP2A6
- CYP2C9*2
- CYP2C9*3
- CYP2C19*17
- CYP2D6 T100C
- CYP3A4*1B

Phase II Detoxification

- COMT V158M
- NAT1 R187Q
- NAT2
- GSTM1
- GSTP1 I105V
- GSTP1 C341T
- SOD1 A4V
- SOD1 G93A
- SOD2

Detoxification Summary

- Glutathione Protection
- Mycotoxins
- Xenoestrogens
- Benzo(a)pyrene
- Aromatic Amines
- GSTP1 C341T
- SOD1 A4V
- SOD1 G93A
- SOD2

Phase I Gene Highlight: CYP1A1*2C 4889

CYP1A1 is a liver enzyme that catalyzes many reactions in drug metabolism and estrogen and is induced by polycyclic aromatic hydrocarbons (PAH). The highest sources of PAHs come from cigarette smoke, burning coal, vegetable oils, smoked meat, and charred meat. Benzopyrene is the most common PAH discussed in the literature regarding individual Phase I and Phase II detoxification.

Variants in CYP1A1 cause an increased activity of 17β-estradiol and estrone.[2] Heterozygous and homozygous variants are associated with breast and prostate cancer risk in Caucasians and lung cancer risk in Chinese. A combination of the GSTM1 null genotype and variants in CYP1A1 were found in head and neck cancer cases. Researchers also found that a high heterocyclic aromatic amine intake was significantly associated with an increased risk of prostate cancer among individuals with the NAT2 slow acetylator phenotype, CYP1A1 rs1048943 CT and CC genotype, and CYP1A2 AC and AA genotype.[3]

In addition to inducing the occurrence of cancers, the polymorphisms of CYP1A1 may also lead to other diseases, such as ulcerative colitis, colorectal adenoma, atherosclerosis, and myocardial infarction.[4]

Along with assessing environmental and dietary exposure for patients with variants in CYP1A1, there are also cooking strategies. Braising or baking meat and marinating meat with citrus, tomatoes, grapes, dark beer, onion, garlic, and numerous herbs and spices with high antioxidant capacity reduce PAHs by up to 90%.[5]

Other strategies include a higher need to support estrogen metabolism and detoxification with iodine, resveratrol, and green tea. Optimal levels of iodine can help modulate the estrogen pathway and help prevent cancerous growth by targeting CYP1A1 and CYP1B1. Iodine deficient breast tissue exhibits early markers of breast cancer.[6] Resveratrol protects against dioxins and β-naphthoflavone, known inducers of CYP1A1.[7]

When reviewing the COMT genotype, the dosage of green tea can be optimized. Multiple studies[8,9] in men and women have found that green tea polyphenols were retained the highest in the homozygous AA COMT genotype, to a lesser extent in the heterozygous genotype (in men but remained high in women), and the lowest in the wild-type GG genotype. Due to the lowest retention for the GG genotype, the dosage of green tea would need to be higher and more frequent to obtain the same benefit for cancer protection.

Phase I Gene Highlight: CYP1A2 C164A

CYP1A2 is a critical enzyme in caffeine metabolism and the 2-hydroxylation of the main estrogens, estrone, and estradiol. 2-hydroxylation and 16a-hydroxylation are two mutually exclusive pathways in estrogen metabolism. 2-hydroxyestrone acts as a weak estrogen or anti-estrogen. 16α-OHE1 acts as a procarcinogen.

Coffee may protect against breast cancer by altering estrogen metabolism. Women with higher coffee intake and the CYP1A2 homozygous AA fast metabolizer genotype have a ratio of high 2-

hydroxyestrone to low 16α-OHE1.[10] Researchers found that higher coffee intake was more protective against ER-positive breast cancer

In men, one study found that low to moderate coffee intake and the AA fast caffeine metabolizer genotype were less likely to experience prostate-grade cancer progression than non-consumers. In a large, pooled cohort of men with prostate cancer, coffee intake of more than 2.5 cups per day was associated with longer survival with the AA fast metabolizer genotype.[11]

Phase II Gene Highlight: NAT1 R187Q

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

The slow acetylators are sensitive to arylamine carcinogens such as 4-aminobiphenyl (rubber antioxidants, food dyes, commercial hair dyes, smoke from vegetable oils, fungicides on apples, and cigarette smoke).

A higher risk of lung cancer in smokers was found in those with variants in NAT1 R187Q.[12]

The frequency of variants varies with ethnic origin but has been identified in almost half of the Lebanese population. Bladder cancer is the second most common cancer among Lebanese males and is seven times higher in those with variants in NAT1 R187Q.[13]

Patients are recommended cruciferous vegetables, carotenoids, and vitamin C to assist NAT1 detoxification.

Phase II Gene Highlight: GSTM1

GSTM1 rs366631 is a pseudo-SNP that is used as a GSTM1 deletion marker. The deletion is also known as the null genotype and confers the absence of the GSTM1 protein. The frequency of the null genotype varies from 20% to 80%, depending on the ethnic group studied.

High frequencies of the GSTM1 null genotype have been found in patients with lung cancer (East Asians), breast cancer (over 50 age group and in Asians), bladder cancer (with NAT2 slow acetylator), colorectal cancer, skin cancer, gastric cancer (among Asians with H. Pylori), chronic bronchitis, kidney disease progression, acute myeloid leukemia, acute lymphoblastic leukemia, head and neck cancer (combined with CYP1A1 variant), endometriosis, type 2 diabetes retinopathy, and recurrent pregnancy loss. [13,14,15,16,17,18,19,20,21,22]

Isothiocyanates from cruciferous vegetables are known for their anti-cancer activity. They are stored as glucosinolates in cruciferous vegetables and are hydrolyzed by myrosinase (an enzyme found in plants and intestinal microflora) to form isothiocyanates. Isothiocyanates from cruciferous vegetables are substrates and inducers of GSTM1.

The GSTM1 null genotype may alter isothiocyanates clearance, with this genotype retaining higher levels of isothiocyanates and therefore the benefits. In numerous studies, the GSTM1 null genotype was the most responsive to cruciferous vegetables for anti-cancer effects against lung cancer, colon cancer, breast cancer, and kidney disease. [23,24,25]

The levels of isothiocyanate in cruciferous vegetables will range based on growing conditions including sulfur and nitrogen levels, time after harvest and storage (cold transportation and storage of broccoli also cause a loss of glucosinolates up to 70-80%), plant genetics, and cooking preparation. Broccoli sprouts will yield the highest isothiocyanate levels.

Breast Cancer

The GSTP1 Ile105Val gene polymorphism, a significant factor in the context of breast cancer risk, involves an A-G substitution that reduces enzymatic activity. Carriers of these mutations, due to their reduced ability to detoxify carcinogens, show varying susceptibility to different chemical carcinogens, as indicated by epidemiological studies.

A meta-analysis of 10,067 cancer cases and 12,276 controls in 41 independent case–control studies from 19 articles found a significant increase in risk in breast cancer in Caucasians with variants in GSTP1 rs1695.[26] A second meta-analysis found the same results with Asians that had the GG genotype. A 2020 study found that the rs1695 homozygous GG genotype was associated with an increased risk of breast cancer, but not the AG genotype.[27] Other research has shown the risk to be higher in premenopausal women vs. postmenopausal women.

An analysis that included 3,035 breast cancer cases and 3,037 population controls in a Chinese population found that cruciferous vegetable intake helped offset the risk of the GG genotype, with a lower risk associated with a higher cruciferous vegetable intake.[28]

Other research has shown that glycine, cysteine, selenium, vitamin C, B1, B6, zinc, magnesium, optimal iron levels, alpha lipoic acid, milk thistle, holy basil and vitamin E supplementation (homozygous GG genotype only), all target GSTP1.

Male Fertility

Studies have also shown that men exposed to higher levels of air pollution were more likely to experience abnormal sperm morphology, decreased motility, and an increased chance of DNA fragmentation.[29]

Pesticides, cadmium, mercury, and arsenic have all been shown to lower GSTP1 expression, increasing the elevation and toxicity of these chemicals and heavy metals. The exposure and sensitivity to these chemicals and heavy metals are suspected reasons for the increased risk of certain cancers and male infertility related to GSTP1 variants.

Men with the heterozygous or homozygous genotype for GSTP1 rs1138272 were three times more likely to experience a disturbance in sperm motility and 2.5 times higher for abnormalities in the

morphology of spermatozoa.[30]

Selenium, vitamin C, B1, B6, folate, zinc, magnesium, healthy iron levels, milk thistle, holy basil, and cruciferous vegetables all assist GSTP1.

Discussion

The Detoxification Panel, through essential genetic lab work, provides a powerful tool to understand individual metabolism of environmental and dietary toxic risk factors. This understanding is crucial for disease prevention. By assessing exposure, individual metabolism, and implementing strategies, we can help patients with targeted protocols, offering them a promising path to improved health.

Sources

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