GENEVIRONMENT

SENSITIVITY TO CHEMICALS AND POLLUTANTS

Genetic Analysis

CONFIDENTIAL INFORMATION

GENEVIRONMENT

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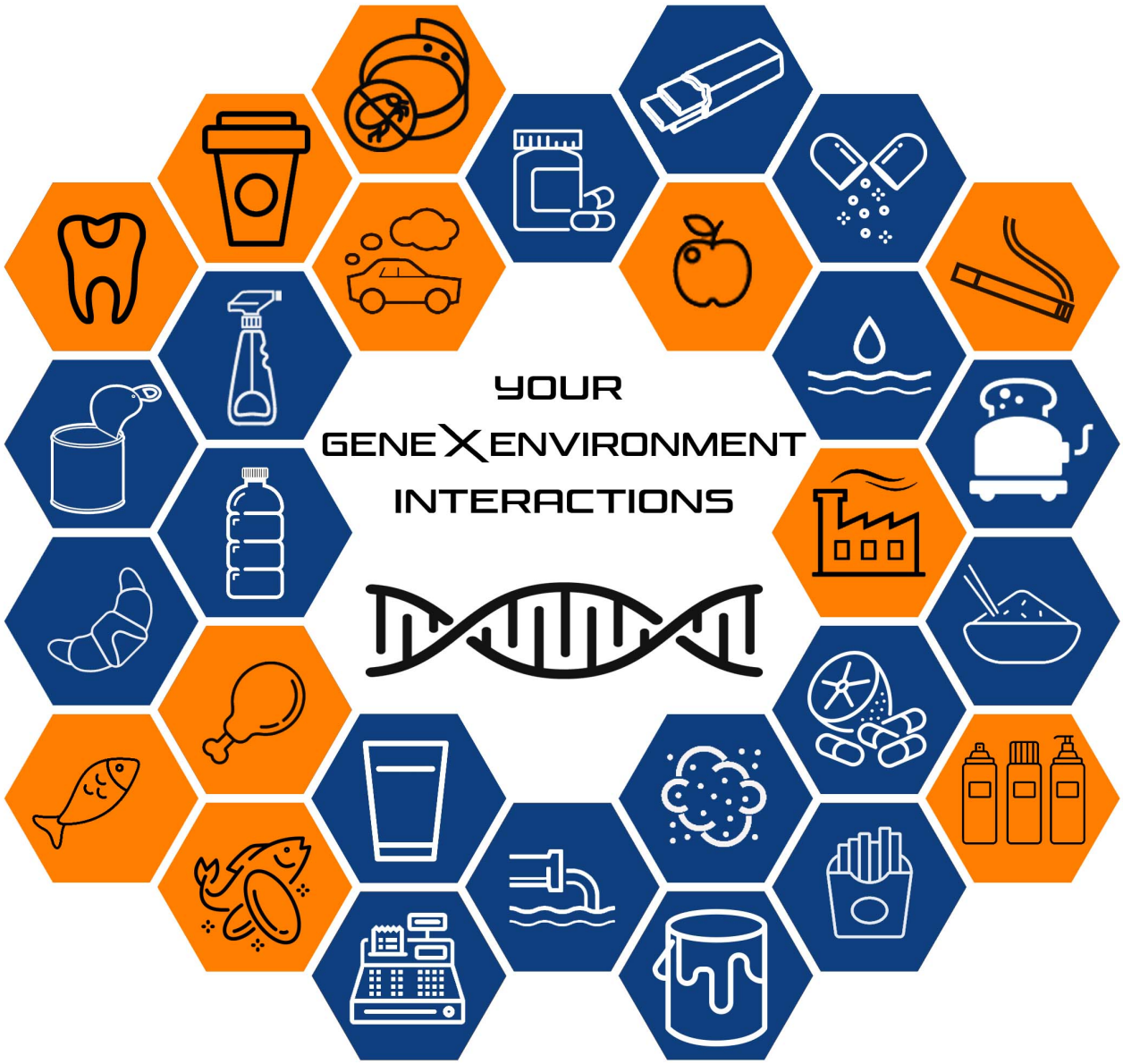
GENEVIRONMENT

Graphic Summary: Personalized Map of Hazardous Exposures

An overview of your inheritted responses to toxic chemicals and pollutants.



Attention! genetic sensitivity diagnosed

No genetice sensitivity found

GENEVIRONMENT™

Analysis Summary List

Your body's ability to detoxify the following chemicals is different than the average person:

MERCURY

BENZENE

BPA

INSECTICIDE (pyrethroid)

MEAT CARCINOGENS

HIGH GENETIC RISK

High Genetic Sensitivity means that your ability to cope with these chemicals is greatly reduced. Exposure puts you at high risk for adverse health outcomes.

ADDED GENETIC RISK

Added Genetic Sensitivity means that your ability to cope with these chemicals is somewhat degraded. Exposure elevates your risk for related adverse outcomes in comparison with most people.

PROTECTIVE GENETIC MAKE-UP

Protective Genetic Make-up means that your ability to cope with these chemicals is better than the average person.

Personalized Advice and Detailed Analysis

Learn how you are exposed to these chemicals and understand the health risks involved. Read the detailed section and discover how to reduce exposure. Take simple actions to boost your detoxification ability and deal better with these chemicals.

CONSULT YOUR PHYSICIAN before making any lifestyle changes following this report. Your doctor is able to consider additional factor such as current medical status and pharmaceuticals in use.

Genetic Sensitivity Analysis: Toxic Chemicals and Pollutants

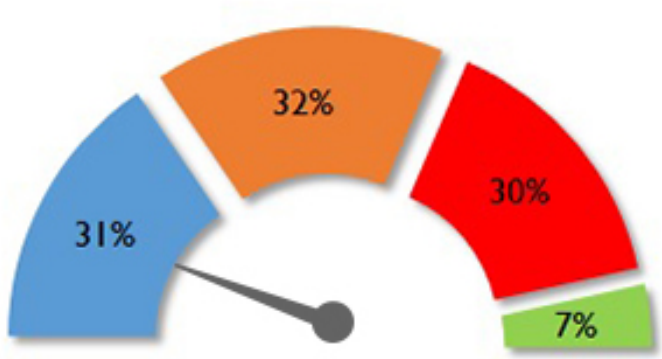
Detailed analysis for each test, tips and recommendations

CHEMICAL ARSENIC

GENETIC FINDINGS SUMMARY

No susceptibility variants found. Your body's response to arsenic is normal and puts you at an average risk for the negative health effects caused by arsenic.

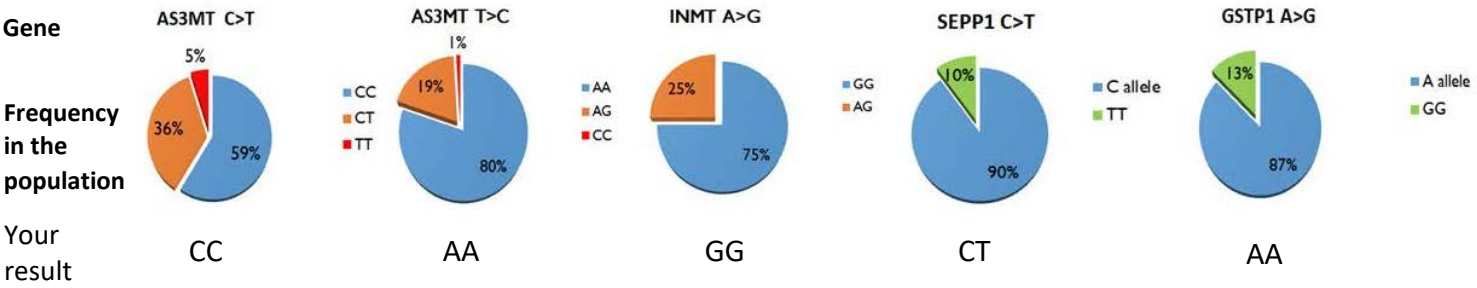
Although genetic sensitivity was not identified, you may wish to take the general precautions to further reduce your risk. For more information, visit *www.geneinformed.com/arsenic*



Your Total Genetic Risk score

AVERAGE RISK

Your genetic makeup Arsenic metabolizing enzymes



CHEMICAL

ARSENIC

About Arsenic

Arsenic is considered toxic and carcinogenic by the major health agencies. In some populations that have been exposed to arsenic for many generations, genetic variation in metabolizing genes have enabled these people to thrive despite the toxic load in their environment.

Routes of exposure

Arsenic (As) is found in the air, soil and water. We breathe it and come into contact with it through our skin, but the major route of exposure is through drinking water. Approximately 13 million in the United States are exposed to arsenic-contaminated drinking water. The second most common source for arsenic is rice.

Health effects

Arsenic is a class I human carcinogen, and chronic exposure to high levels of arsenic (>300 µg/L) is associated with substantial increased risk for a wide array of diseases including cancers of the lung, bladder, liver, skin, and kidney, as well as neurological and cardiovascular diseases, diabetes and an earlier onset of menopause. Emerging evidence suggests that arsenic may have adverse effects on health even at concentrations as low as 10–50 µg/L. Once individuals are chronically exposed to arsenic, risk for arsenic-related diseases and mortality remains high for several decades even after cessation of exposure.

On top of genetic makeup, additional factors such as age, sex and low-protein intake have shown associations with cancer risk in conjunction with arsenic exposures. Smokers show greater levels of arsenic. Pregnant women show lower and lower arsenic levels, as week of gestation progresses.

CHEMICAL

ARSENIC

Genetic sensitivity to arsenic

Certain genetic makeups are associated with metabolism, secretion and levels of arsenic in body tissues, making some people more sensitive to its deleterious effects.
Methylation of arsenic is catalyzed by the arsenic methyltransferase (AS3MT) enzyme. Genetic variants in the gene encoding for the enzyme Arsenic (+3 oxidation state) Methyltransferase (AS3MT) play a key role in arsenic methylation capacity.

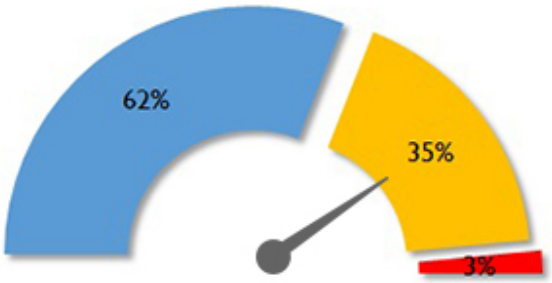
Genetic association studies have found that a certain variant in the INMT gene, involved in selenium metabolism, affects both selenium and arsenic levels. This variant promotes urinary excretion of arsenic, while slowing loss of selenium. SEPP1 is another gene involved in selenium metabolism. This gene might be responsible for some of the extracellular antioxidant defense properties of selenium or might be involved in the transport of selenium to tissues such as brain and testis. Selenium (Se) can decrease arsenic levels by binding to it and easing its excretion.

The GSTP1 gene encodes for the Glutathione S-transferase P1 enzyme. It plays a significant role in antioxidant defense mechanisms. Its variants were associated with levels of trace elements such as mercury and arsenic.

CHEMICAL BENZENE

GENETIC FINDINGS SUMMARY

Susceptibility variants found. Less efficient metabolism of benzene is expected, which means that you are more sensitive to benzene's deletirious effects.



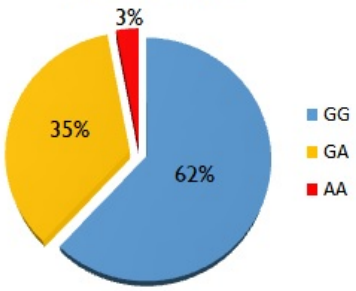
Your Total Genetic Risk score

ADDED RISK

Your genetic makeup Benzene metabolizing enzymes

Gene **NOQ1 G>A**

Frequency in the population



Your result GA

CHEMICAL BENZENE

Personalized Recommendations

Reduce your risk from benzene exposure by taking the following steps:

1. Quit smoking. Average smokers take in about 10 times more benzene than nonsmokers each day. Passive second hand smokers are also at risk.
2. Benzene is a major component of gasoline. Try to limit gasoline fumes by pumping gas carefully and using gas stations with vapor recovery systems that capture the fumes. Avoid skin contact with gasoline.
3. When possible, limiting the time you spend near idling car engines can help lower your exposure to exhaust fumes, which contain benzene (as well as other potentially harmful chemicals).
4. Benzene is common in air emissions from manufacturing plants and hazardous waste sites, so try to limit your time around such sites.
5. Limit or avoid exposure to fumes from solvents, paints, and art supplies, especially in unventilated spaces.
6. If you are exposed at your workplace, talk to your employer about limiting your exposure through process changes (such as replacing the benzene with another solvent or enclosing the benzene source) or by using personal protective equipment. If needed, the Occupational Safety & Health Administration (OSHA) can provide more information or make an inspection.
7. Adopt lifestyle and dietary patterns that promote your detoxification capacities (appendix A).

CHEMICAL BENZENE

About Benzene

Benzene is a human carcinogen widely used in the United States, ranked in the top 20 chemicals for production volume. It is the primary starting material for chemicals used to make plastics, resins, synthetic fibers, dyes, detergents, drugs, and pesticides. Benzene is also a component of crude oil, gasoline, and cigarette smoke.

Benzene is strongly associated with blood and immune system disorders, including anemia, leukemia and lymphoma.

The general population is exposed to benzene mainly through breathing of fuel emission exhaust, tobacco smoking and second-hand smoking, and through breathing vapors of household items like paints, adhesives, marking pens, rubber products and tapes. Common genetic variations in metabolic genes can alter individual susceptibility to benzene toxicity.

Routes of exposure

The main environmental sources of benzene exposure are related to fuel emission exhaust from motor vehicles and evaporation losses from handling, distribution and storage of fuels. Another major source of environmental benzene exposure is tobacco smoking for both mainstream and second-hand smokers. It has been found that smokers receive 90% of their total benzene exposure from smoking and that benzene concentration in the breathing air of smokers can be 10–20-times higher than in nonsmokers. Other sources may also contribute to the cumulative benzene indoor concentration. Household items like paints, adhesives, marking pens, rubber products and tapes emit benzene vapors.

CHEMICAL BENZENE

Health effects of benzene

In general, metabolism of benzene creates highly reactive oxygen elements, leading oxidative stress. This stress promotes cell damage, DNA strand breaks, mutagenesis and chromosomal aberrations. Benzene uptake by bone marrow affects stem cell differentiation and proliferation, leading to anemia, abnormal blood count and leukemia. B-cell and T-cell proliferation is also reduced by benzene, altering the immune system response.

Occupational exposure to benzene was found to have a causal role in inducing leukemia, lymphoma and myeloma. However, both toxic and carcinogenic effects have been demonstrated also for minor exposures of the several parts per billion(ppb), which is much lower than the 8 h occupational exposure limit (1 part in a million) recommended by the U.S. Occupational Safety and Health Administration (OSHA). A supralinear ("more than expected") relationships between low-dose exposure and metabolism of benzene has been discovered. Low dose exposure may be as bad as being exposed to a dose 1000 times larger.

In the United States, workers are exposed to potentially high levels of benzene in the chemical industry, in petroleum refineries, in oil pipelines, on ships and tankers, in auto repair shops, and in bus garages. Occupational exposures in the developing world are sometimes very high because of the continuing presence of benzene in industrial solvents and glues.

The general population is exposed to benzene mainly through breathing of fuel emission exhaust from motor vehicles, tobacco smoking and second-hand smoking, and through breathing vapors of household items like paints, adhesives, marking pens, rubber products and tapes.

Metabolism

Inhaled benzene is readily absorbed into the blood, taking 40 to 70% of airborne dose by passive diffusion through the lung capillary membranes. Benzene undergoes biotransformation within the liver but can be metabolized also in the lung and bone marrow.

Biotransformation of benzene generates electrophilic metabolites as well as reactive oxygen species (ROS) as biotransformation byproducts. Both these categories of electrophilic species may modify, upon covalent binding to nucleophilic moieties, cell macromolecules (DNA, RNA, proteins, lipids).

Genetic sensitivity to benzene

The NQO1 enzyme acts by decreasing the formation of free radicals (semiquinones), hence protecting cells against the adverse effects of quinones and their derivatives. The mutant NQO1*2 protein is very unstable and is rapidly degraded, making it virtually undetectable in humans carrying two copies of this variant (NQO1*2/*2 genotype). A higher risk of chronic benzene poisoning was observed among heavily exposed workers carrying the homozygous NQO1 variant genotype. Deleterious effects were also found in workers exposed to low levels of benzene, such as taxi drivers and gasoline station attendants. In addition, the NQO1*2 polymorphism prevalence was found higher in families whose children are affected by acute lymphocytic leukemia. For both the general population and exposed workers, it seems that inefficient metabolism of benzene by the NQO1 enzyme leads to high genetic sensitivity to benzene and its deleterious effects.

CHEMICAL BISPHENOL-A

GENETIC FINDINGS SUMMARY

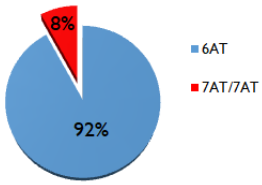
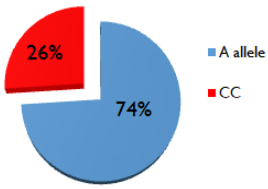
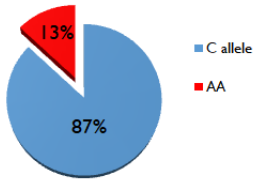
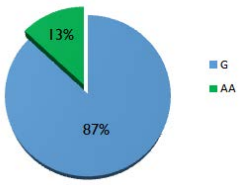
Your genetic makeup grants better-than-average metabolism of BPA. Your body's response to BPA puts you at relatively lower risk for the negative health effects caused by BPA.

You may wish to further reduce your risk from BPA by taking general precautions. Read more at http://www.geneinformed.com/bpa

Your Total Genetic Risk score

REDUCED RISK

Your genetic makeup BPA metabolizing enzymes

Genes	UGT1A1 6AT>7AT	UGT2B15 A>C	UGT2B15 C>A	GSTP1 A>G
Frequency in the population				
Your results	6AT/6AT	AA	CC	AA

CHEMICAL BISPHENOL-A

About Bisphenol-A

Bisphenol-A (BPA) is widely spread in the plastic we use every day. It may be toxic even at low doses, and has been associated with various human diseases. Differences in the susceptibility of people to the adverse effects of BPA depend on the different amount of BPA consumed, and on personal ability of an individual to effectively excrete BPA.

Routes of exposure

BPA is mainly found in toys, drinking containers, dental sealants, water pipes, and food containers (both plastic and cans). The primary route of exposure to BPA is through oral intake, but exposure can also occur via dermal contact or through inhalation.

Health effects of BPA

BPA is an environmental endocrine disruptor, mimicking the action of human estrogen. It is considered a health threat due to its widespread exposure and the potential for toxicity at low doses. BPA has been associated with various human diseases including breast cancer, prostate cancer, polycystic ovary disease, cardiovascular disease, diabetes, obesity, asthma and thyroid maladies.

CHEMICAL

BISPHENOL-A

Genetic sensitivity to BPA

Common genetic variants of the UGT2B15 gene (UGT2B15*2 and UGT2B15*5) markedly reduce BPA clearance. Individuals carrying two copies of these forms of the gene are 10 to 25 times less efficient in excreting BPA out of the body.

Carriers of two copies of the UGT1A1*28 allele show a 10-fold reduction in BPA detoxification in breast tissue, in comparison to non-carriers.

Among children exposed to higher BPA levels, those with the GSTP1 AA genotypes had a 4.84-fold higher risk for asthma.

Metabolism

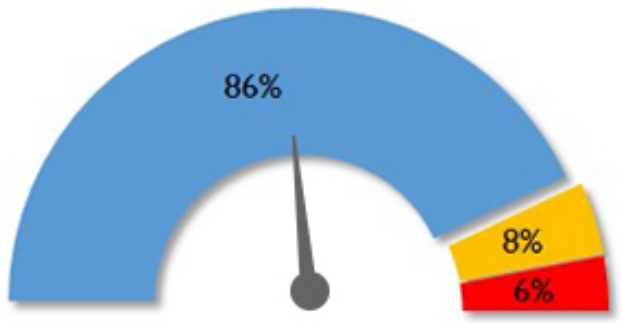
After ingestion, BPA is taken up by the body and must be metabolized to enable efficient excretion. The main route of metabolism of BPA in humans is glucuronidation, which occurs primarily in the liver and is mediated by the UDP-glucuronosyltransferase (UGT) enzymes. The UGT2B15 enzyme acts mainly in the liver, and is responsible for 50-80% of total BPA metabolism. The UGT1A1 enzyme contributes to BPA metabolism mainly in breast tissue.

The GSTP1 gene plays a significant role in antioxidant defense mechanisms. It may be responsible for conjugation of BPA metabolites with glutathione, which could increase BPA excretion.

CHEMICAL PHTHALATES

GENETIC FINDINGS SUMMARY

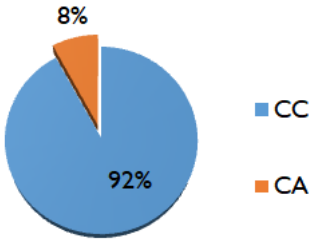
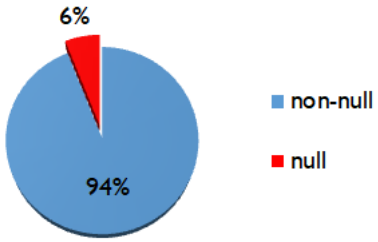
No susceptibility variants found. No genetic sensitivity to phthalates is expected. Your body's response to phthalates is normal and puts you at an average risk for the negative health effects caused by phthalates.



Your Total Genetic Risk score

NORMAL RISK

Your genetic makeup Phthalate metabolizing enzymes

Genes	**TNFα C>A**	**FLG mutations**
Frequency in the population		
Your results	CC	non-null

CHEMICAL PHTHALATES

About phthalates

Phthalates are man-made plasticizers invented about a hundred years ago, and are now ubiquitous environmental contaminants. This means that we are exposed to phthalates pretty much constantly.

Exposure to phthalates is a threat to public health and is associated with allergies, obesity, cancer, among other disease.

Routes of exposure

Phthalates are used in a wide range of industrial applications such as plasticizers in flexible vinyl products (flooring, wall coverings, toys, food containers and medical supplies), in personal care products (nail polish, deodorants, shampoos, conditioners and perfumes) and in solvents.

Phthalates are used in the food industry as well. Manufacturers had replaced expensive natural emulsifiers with di-(2-ethylhexyl) phthalate (DEHP) in numerous food and drinks.

Health effects

Phthalates are considered estrogenic endocrine disruptors, and recent research has suggested that they may act as a risk factor for estrogen-dependent diseases, as well as cancer.

Breast cancer risk has been differentially associated with urinary levels of some phthalate metabolites. Animal studies show phthalates cause liver injury and reproductive and developmental toxicity.

Other studies have also shown that phthalates are related to several health problems in women, such as endometriosis, uterine leiomyomata, a decrease in thyroid hormone levels, changes in body mass index and waist circumference, and adverse pregnancy outcomes. Other adverse effects of phthalates include irritation and allergic reaction such as asthma and contact dermatitis.

CHEMICAL PHTHALATES

Genetic sensitivity to phthalates

Filaggrin is an epidermal protein that is crucial for skin barrier function. Reduced expression of filaggrin is associated with facilitated transfer of allergens across the epidermis and into our body.

Individuals carrying an inactive FLG genetic variant (FLG-null carriers) have an increased uptake of phthalates through their skin. A significantly higher concentration of phthalate metabolites were found in their urine, indicating greater exposure to phthalates and increased risk of related disease.

Up to 10% of Europeans and 5% of Asians carry at least one null allele in the filaggrin gene (FLG).

Phthalates, epigenetics and asthma

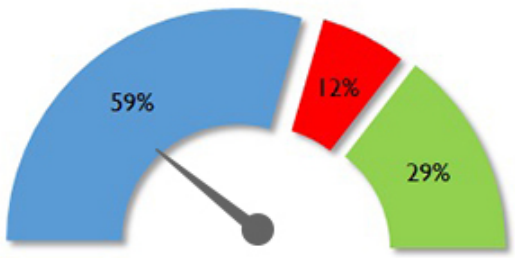
Epidemiological studies suggested an association between exposure to phthalates and increased prevalence of asthma or wheezing. Pulmonary physiological data also show that phthalates may promote and aggravate allergic asthma.

A certain kind of phthalate, DEHP, promotes a deviation of the Th2 immune response, which results in allergy. It does so by modulating (IL-4) and immunoglobulin E (IgE) production, as well as suppressing expression of immune system genes IFN-α and IFN-β. It seems that DEHP, as other endocrine disruptors, modifies epigenetic marks on these genes. The resulting lower methylation levels of the TNFα gene doubles the risk of asthma in children.

CHEMICAL DIOXIN

GENETIC FINDINGS SUMMARY

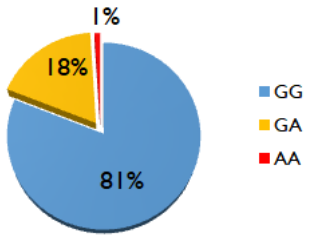
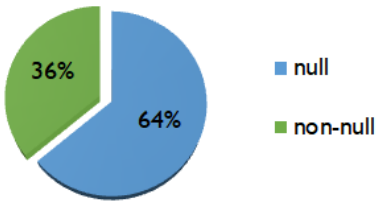
Your total genetic risk score is average. Your body's response to dioxin is expected to be normal and puts you at the average risk for the negative health effects caused by dioxins.



Your Total Genetic Risk score

NORMAL RISK

Your genetic makeup Dioxin metabolizing enzymes

Genes	AHR G>A	GSTM1
Frequency in the population		
Your genetic makeup	GG	null

CHEMICAL DIOXIN

About Dioxins

Dioxins are widely common persistent organic pollutants (POPs). Dioxins are byproducts in the manufacturing process of certain chlorinated intermediates and products, like chlorine bleaching processes and manufacture of herbicides and bactericides.

Due to its persistency and its multiple adverse health effects, dioxins are a serious world-wide public health concern.

Routes of exposure

Dioxins bio-accumulate in the food chain, mainly in fatty tissue of animals. More than 90% of human exposure is through meat and dairy products, fish and shellfish. Once dioxins enter the body, they last a long time because of their chemical stability and their ability to be absorbed by fat tissue, where they are then stored in the body. Their half-life in the body is estimated to be 7 to 11 years.

Health effects

Dioxins are highly toxic and can cause reproductive and developmental problems, damage the immune system, interfere with hormones and also cause cancer. Long-term exposure is linked with impairment of the immune system, the developing nervous system, the endocrine system and reproductive functions.

CHEMICAL DIOXIN

Dioxins promote oxidative damage and DNA fractures, initiating and supporting cancerous processes. On top of that, dioxins alter the metabolism of estradiol and estrogen in breast cells tissue, which may further increase the risk for breast cancer.

Dioxin alters thyroid-stimulating hormone production. In pregnant women with poor dioxin-metabolizing enzyme activity, chances for impaired fetal brain development and hypothyroidism in the fetus increase.

Genetic sensitivity to dioxin

Dioxin affects our health by changing the expression of several important enzymes. The CYP1A2 enzyme plays an important role in bio-transformation of various compounds, including drugs and carcinogens. Dioxin-like chemicals bind to a receptor known as AhR, which controls the expression of the CYP1A2 gene. Studies found that exposure to dioxin alter CYP1A2 activity via the AhR receptor, resulting in adverse human health effects.

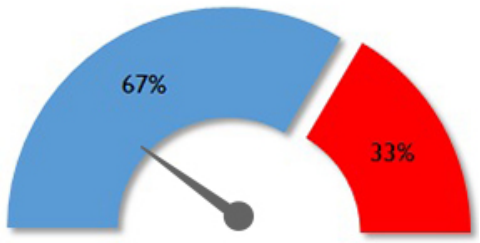
In some people, dioxin's affinity to the AhR receptor is greater, which causes more interference with the normal activity of the CYP1A2 enzyme. Carriers of the AhR gene variant are more sensitive to dioxin exposure and its deleterious health effects.

CHEMICAL PERCHLORATE

GENETIC FINDINGS SUMMARY

No added genetic sensitivity to perchlorate was found. Your body's response to perchlorate is normal and puts you at an average risk for the negative health effects caused by perchlorate .

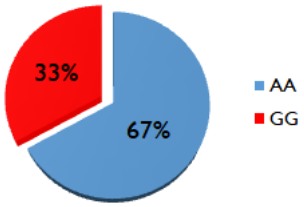
You may further reduce your risk from perchlorate exposure by following the general recommendations. Visit www.geneinformed.com/perchlorate



Your Total Genetic Risk score

NORMAL RISK

Your genetic makeup Perchlorate metabolizing enzymes

Gene	**FOXE1 A>G**
Frequency in the population	
Your result	AA

CHEMICAL PERCHLORATE

About perchlorate

Perchlorate is commonly used in explosives and batteries. It is found in herbicides and fertilizers and used as agricultural soil sterilant. Perchlorate is a contaminant in our drinking water and our food.

Perchlorate inhibits iodine uptake by the thyroid gland, which may result in various adverse health effects.

Routes of exposure

The major source of perchlorate is in contaminated drinking water, however it may be found in food. Perchlorate enters foods in the course of their production and/or processing, through herbicides or when in contact with water which has been treated with chlorinated biocidal products for disinfection purposes. Another source of contamination may be food packages, where perchlorate is sometimes used as an anti-static or a sealant agent.

Perchlorate found in drinking water and swimming pools may originate from using sodium hypochlorite bleach.

CHEMICAL PERCHLORATE

Health effects

Perchlorate can interfere with the human body's ability to absorb iodine into the thyroid gland, which is a critical element in the production of thyroid hormones. In adults, the thyroid plays an important role in metabolism, making and storing hormones that help regulate the heart rate, blood pressure, body temperature, and the rate at which food is converted into energy. In fetuses and infants, thyroid hormones are critical for normal growth and development of the central nervous system.

Genetic sensitivity to perchlorate

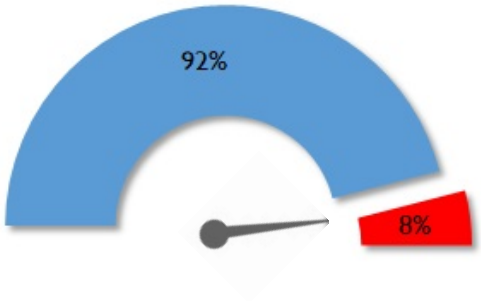
People with thyroid disorders or an iodine deficiency can be particularly affected by perchlorate's undesired effects. A common variant in the FOXE1/PTCSC2 gene is associated with primary hypothyroidism, making its carriers more susceptible to perchlorate exposure.

Primary hypothyroidism is the most common thyroid disorder, affecting 1-5% of the population, and is characterized by deficiencies of thyroid hormones T3 (triiodothyronine) and T4 (thyroxine).

CHEMICAL MEAT CARCINOGENS

GENETIC FINDINGS SUMMARY

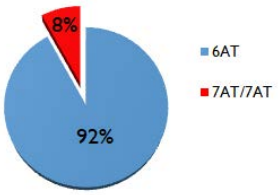
Genetic sensitivity to meat carcinogens is expected. Your body's response to PAH's and HCA's is less favorable than most people and puts you at greater risk to be impacted by the negative health outcomes of these pollutants.
To reduce your risk, follow the personalized advice section below.



Your Total Genetic Risk

HIGH RISK

Your genetic makeup Meat carcinogens metabolizing enzymes

Genes	UGT1A1 6AT>7AT
Frequency in the population	
Your results	6AT/6AT

Visit www.geneinformed.com/meat-carcinogens for more information.

CHEMICAL MEAT CARCINOGENS

Personalized Recommendations

Reduce your risk from meat carcinogen exposure by taking the following steps:

1. Go vegetarian or consider eating less meat- its especially healthy for people like you. Two to three servings a week should maintain your dietary needs and keep your risk levels low.

2. The amount of HCAs produced depends on how long the meat is exposed to high temperatures. Cooking in a temperature lower than 300°F is recommended. Grilled, barbecued, smoked meat and sausages should be avoided.

3. Roasted coffee and black tea contain high levels of PAH's. Choose high-quality, fresh coffee for reduced exposure.

4. Quit smoking. Average smokers take in about 10 times more benzene than nonsmokers each day. Passive second hand smokers are also at risk.

5. Avoid as much as you can breathing gasoline and diesel exhaust, open fires and incinerators.

6. You can help your body produce more of the enzyme you lack!
   By eating cruciferous vegetables, you increase the production of the UGT1A1 enzyme and help your body deal with the chemicals .
   Eat more cauliflower, cabbage, kale, garden cress, bok choy, broccoli and Brussels sprouts, especially when eating meat.

CHEMICAL MEAT CARCINOGENS

About PAH's and HCA's

Hetrocyclic Amins (HCAs) and Polycyclic Aromatic Hydrocarbons (PAHs) are environmental pollutants associated with adverse health outcomes including cancer, asthma and reduced fertility. These chemicals are commonly found in polluted air, as well as in our diet.

Eating cooked meat, and moreover grilled meat, exposes your body to HCAs and PAHs, formed when cooking meat at a high temperatures (typically above 300°F).

Routes of exposure

Exposure to PAHs and HCA's is common both by breathing and by diet. PAH and HCA originate from burning wood and fuel for homes and in gasoline and diesel exhaust, cigar and cigarette smoke.

PAH and HCA are also commonly found in smoked, barbecued, or charcoal-broiled foods, roasted coffees, and sausages. These chemicals are created as a result of cooking components of meat -amino acids, sugar, and creatine, at high temperatures (typically above 300°F). The amount of HCAs produced may depend on how long the meat is exposed to those high temperatures. Grill over an open flame and smoking meat yields higher levels of the carcinogens.

Metabolism

The UGT1A1 is an enzyme responsible for the detoxification and elimination of PAH and HCA. UGT1A1 enzymes have been seen to metabolize additional carcinogens, like benzo-pyrenes, PhIP and estradiol. The enzyme is also responsible for the metabolism of bilirubin, a normal break-down product of old blood cells.

CHEMICAL MEAT CARCINOGENS

Genetic sensitivity to meat carcinogens

Genetic variant forms of UGT1A1 are quite common. The UGT1A1*28 polymorphism affects about 8%-10% of Caucasian and African-American populations, while the UGT1A1*6 affects 2-5% of East-Asians. Both variants reduce UGT1A1 expression by about 30%, compared to the activity of the enzyme in an average healthy average individual.

Health effects of meat carcinogens

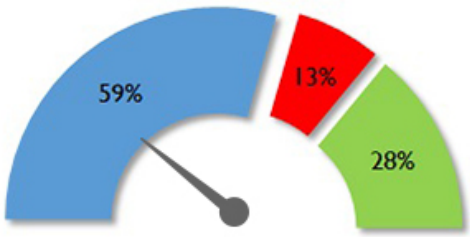
Reduced UGT1A1 activity has been shown to increase the risk of developing colorectal and breast cancer across multiple studies in both Chinese and White populations.

Individuals with low UGT1A1 activity are the majority of Gilbert's syndrome patients. They typically have fluctuating bilirubin levels, which are often within the standard range. Illness, stress, or fasting can precipitate a rise in bilirubin levels, leading to hyperbilirubinemia (high levels of bilirubin) and symptoms such as temporary jaundice or abdominal discomfort. However, these symptoms will typically resolve themselves, and the syndrome is considered harmless in adults.

CHEMICAL ACRYLAMIDE

GENETIC FINDINGS SUMMARY

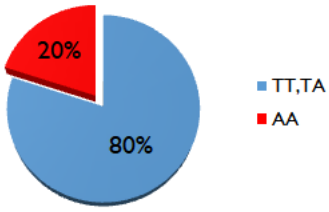
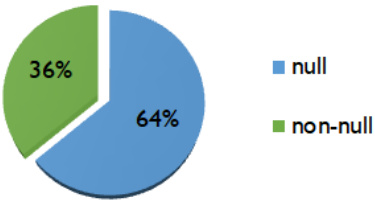
Genetic sensitivity to acrylamid is not expected. Your body's response to acrylamide is normal and puts you at an average risk for the negative health effects caused by this pollutant. Although genetic sensitivity was not identified, you may wish to take the general precautions to reduce your risk.
Visit www.geneinformed.com/acrylamide for more information.



Your Total Genetic Risk score

NORMAL RISK

Your genetic makeup Acrylamide metabolizing enzymes

Genes	**CASP10 T>A**	**GSTM1**
Frequency in the population		
Your results	TA	null

CHEMICAL ACRYLAMIDE

About acrylamide

Acrylamide is a neurotoxin and a human carcinogen, found in very common food items. It is generated in heat-processed foods high in carbohydrate, where it is formed by an interaction between amino acids, primarily asparagine (the major amino acid in potatoes and cereals) and sugars such as fructose, glucose and maltose.

Routes of exposure

Carbohydrate-rich foods that had been fried, roasted or baked above 120°C (248°F) show high acrylamide levels. The crust holds the majority of the acrylamide, being exposed to the highest temperatures and low moisture content. High levels of acrylamide are common in snack foods, cookies, cakes, potato crisps, French fries, breads, cereal products, and coffee.

Acrylamide is also a component of tobacco smoke, and is also used in cosmetic additives, including creams, body lotions, shampoos and some sealants. The main routs of exposure though are through smoking and ingesting acrylamide from food.

Health effects

Acrylamide has been classified as a probable human carcinogen by International Agency for Research on Cancer (IARC). Exposures to acrylamide in workplaces and daily ingestion of acrylamide from consumption of high-temperature processed foods have been of great concerns.

CHEMICAL ACRYLAMIDE

Genetic sensitivity to acrylamide

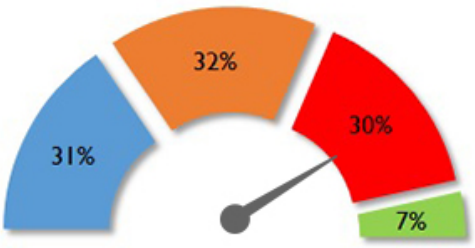
Damage to the DNA triggers apoptosis (planned cell death) in order to prevent abnormal growth of cancer cells. Variations in key genes that regulate this mechanism may hamper the efficiency of the apoptosis pathway. The caspase genes are key regulators in the initiation and execution of apoptosis, and are vital in fighting cancer cells. Carriers of the I522L variant of the CASP10 gene are at a higher risk for cancer susceptibility following the exposure to acrylamide and GA.

The GSTM1 gene encodes for the glutathione S-transferase M1, a member of the GST family of enzymes. These enzymes have broad detoxifying abilities against carcinogens, drugs, or other toxins. The GSTM1 genotypes significantly modify the excretion of urinary acrylamide and the GA. Carriers of the active enzyme variant enjoy some protection against acrylamide genotoxic effects.

CHEMICAL MERCURY

GENETIC FINDINGS SUMMARY

Your genetic makeup is associated with high genetic sensitivity to mercury.



Your Total Genetic Risk score

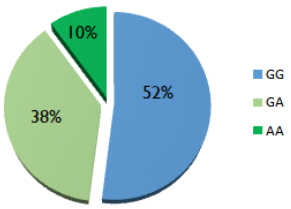
HIGH RISK

Your genetic makeup Mercury metabolizing enzymes

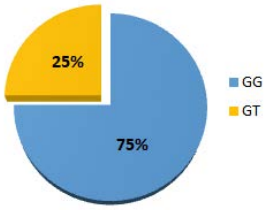
Genes

Frequency in the population

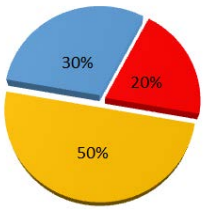
SEPP1 G>A



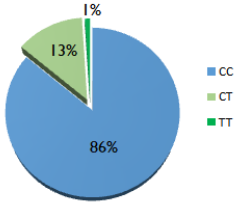
INMT G>T



COMT G>A



APOE C>T

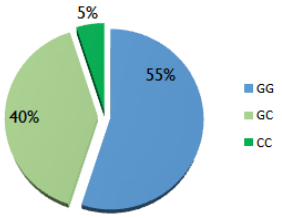


Your results GA GG GG CC

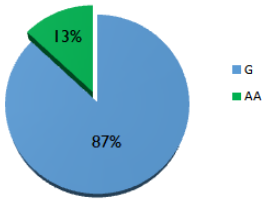
Genes

Frequency in the population

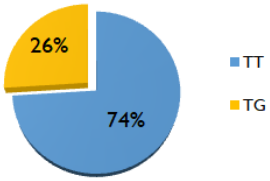
MT2A G>C



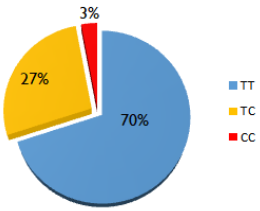
GSTP1 A>G



CPOX4 T>G



APOE T>C



Your results GG AA TT TC

CHEMICAL MERCURY

Take the following precautions to reduce your additional genetic sensitivity to mercury:

1. Limit fish consumption to 2-3 servings per week (adult serving portion is about 4 ounces). Avoid fish with high-mercury content:

Best Choices EAT 2 TO 3 SERVINGS A WEEK

Anchovy	Herring	Scallop
Atlantic croaker	Lobster, American and spiny	Shad
Atlantic mackerel	Mullet	Shrimp
Black sea bass	Oyster	Skate
Butterfish	Pacific chub mackerel	Smelt
Catfish	Perch, freshwater and ocean	Sole
Clam	Pickerel	Squid
Cod	Plaice	Tilapia
Crab	Pollock	Trout, freshwater
Crawfish	Salmon	Tuna, canned light (includes skipjack)
Flounder	Sardine	Whitefish
Haddock		Whiting
Hake		

OR

Good Choices EAT 1 SERVING A WEEK

Bluefish	Monkfish	Tilefish (Atlantic Ocean)
Buffalofish	Rockfish	Tuna, albacore/ white tuna, canned and fresh/frozen
Carp	Sablefish	Tuna, yellowfin
Chilean sea bass/ Patagonian toothfish	Sheepshead	Weakfish/seatrout
Grouper	Snapper	White croaker/ Pacific croaker
Halibut	Spanish mackerel	
Mahi mahi/ dolphinfish	Striped bass (ocean)	

Choices to Avoid HIGHEST MERCURY LEVELS

King mackerel	Shark	Tilefish (Gulf of Mexico)
Marlin	Swordfish	Tuna, bigeye
Orange roughy		

2. Choose safe dental fillings. Next time you are in for a tooth filling choose amalgam-alternatives like composite resin, glass and porcelain, if possible. For an existing amalgam filling, removal will reduce exposure to mercury in the long run but may expose you to high mercury levels in the process.
3. Make sure that you are getting your vaccines thimerosal-free. Look for vaccines in single-use presentations, as thimerosal is mainly used in multi-dose vials of vaccines. In general, many vaccines are also available in formulations that do not contain thimerosal.
4. Beware of skin lightening or freckle creams, as some are highly contaminated with mercury.
5. Mercury thermometers, some batteries and different fluorescent light bulbs contain mercury. In case one of these breaks, stay away and allow 5-10 minutes of air flow by opening a window or door to the outdoor

environment. Shut off the central forced air heating/air-conditioning system and don't use a vacuum cleaner before collecting all remains. Avoid skin contact when cleaning up and dispose into a a sealable container.

6. Eat 1-3 Brazil nuts a day. Brazil nuts are the number one source of selenium in the human diet. Selenium-based enzymes detoxify mercury, so dietary intake of selenium directly helps fight against mercury toxicity. One Brazil nut contains 45-95 micrograms of selenium, which is just about the recommended dietary allowance (RDA) for selenium in adults. Avoid eating more than 4-5 Brazil nuts a day, since the safe upper limit for selenium intake is 400 micrograms a day. Anything above that is considered an overdose. Other sources for selenium include brown rice, barley, baked beans, sunflower seeds, mushrooms and oatmeal. Some fish are good source for selenium as well.
7. Other food stuff associated with detoxification of mercury include pumpkin seeds, cilantro, garlic, chlorella, turmeric, berries, pomegranates, tomatoes, prunes, and pink/red grapefruit, broccoli, cauliflower, mustard. Find more about promoting your detoxification capacities on appendix A.

CHEMICAL MERCURY

About mercury

Mercury (Hg) is a heavy metal, considered a global pollutant and is a well-known neurotoxin. Exposure to mercury has a variety of significant and documented adverse effects on human health.

Chronic human exposure may occur through a variety of pathways, including industrial processes, occupational and household uses, dental amalgams, Hg-containing vaccines, and consumption of contaminated fish and seafood.

Routes of exposure

Methyl Hg (organic Hg) is one of the best-known toxic biohazards. It forms in marine ecosystems and enters the aquatic food chain, where it undergoes a remarkable bioaccumulation process in muscle tissues of fish, particularly of long-lived predatory species. These include shark, swordfish, fresh tuna, marlin, king mackerel, tilefish from the Gulf of Mexico, northern pike and more.

Some cosmetic products contain mercury, which is easily absorbed through the skin. About 6% of skin lightening products purchased in the United States were found to contain mercury in significant excess. The contaminated creams are usually imported to the US as skin lightening or freckle creams.

CHEMICAL MERCURY

Many other articles contain mercury, but for the most part are not a major route for chronic exposure. These include batteries, different fluorescent light bulbs, jewels with mercury encased in glass, switches, LCD screens and monitors, preservative or antibacterial agent in pharmaceuticals.

Occupational exposure to mercury is of great concern. It is mostly relevant to workers exposed to vapors of elemental mercury, which readily vaporizes at room temperature and quickly absorbed via the lungs. Doctors and lab technicians preparing dental amalgams fillings (“silver filling”) are at high risk. As for the patients themselves, amalgam tooth fillings are considered safe by the FDA and EPA, but some studies found that amalgam is a source of life-long exposure to mercury.

There are concerns over the use of thimerosal in vaccines, a mercury-containing preservative. Robust studies support the safety of thimerosal-containing vaccines, and this is the current view of the FDA. With that, informed individuals can avoid thimerosal in their vaccines. The use of this preservative in vaccines is less common these days, and mainly used in multi-dose vials of vaccines, so look for vaccines in single-use presentations. All vaccines routinely recommended for children 6 years of age and younger in the U.S. are also available in formulations that do not contain thimerosal. Vaccines that do not contain thimerosal as a preservative are also available for adolescents and adults.

CHEMICAL MERCURY

Health effects

Exposure to mercury can be differently associated to over 250 symptoms, involving the neurological, renal, respiratory, gastrointestinal, cardiovascular, hepatic, reproductive, and immune systems.

Hg exposure represents a significant concern during the course of pregnancy, because of the risk to the fetus. Methyl mercury readily passes across the placental barrier to the fetus.

As for occupational exposure, inhalation of Hg vapor was found associated with neurological and behavioral signs and symptoms, such as depression, paranoia, extreme irritability, hallucinations, inability to concentrate, memory loss, hand tremors, weight loss, perpetually low body temperature, drowsiness, headaches, insomnia, and fatigue, may also occur.

Genetic sensitivity to mercury

A certain variation in CPOX4, a gene encoding for a red blood cell heme pathway enzyme, is known to modify performance on multiple neurobehavioral tests. Among boys carrying this CPOX4 variant, chronic mercury exposure reduced scores in attention, learning & memory, executive function, visual spatial acuity and motor function.

The Metallothioneins (MT) are a multigene family of proteins involved in Hg distribution and excretion. Numerous significant adverse effects found among boys carrying either MT1M or MT2A variants and chronically exposed to mercury. These effects were principally within the domains of visual spatial acuity and learning & memory, but also on tests of attention and motor function. Girls carrying these variants were found much less susceptible.

CHEMICAL MERCURY

Catechol-O-methyltransferase (COMT) is an enzyme that maintains neurologic functions by regulating the availability of key neurotransmitters such as dopamine. Polymorphisms in the COMT gene that modify the activity of the COMT enzyme are known to underlie a number of neuropsychiatric disorders as well as process-specific functions that accompany neurobehavioral tasks. Exposure to mercury was associated with risk of neurobehavioral deficits among elementary school students who carry COMT variants. Carriers working as dentists and technicians show altered cognitive flexibility, working and visual memory, as well as bad mood.

The APOE gene provides instructions for making a protein called apolipoprotein E. This protein combines with fats (lipids) to form molecules called lipoproteins, responsible for packaging and carrying cholesterol and other fats. This gene may also detoxify methyl mercury when it crosses the Blood Brain Barrier. However, individuals carrying one variant form of the gene perform a less efficient elimination process.

Selenium (Se) plays an important role in the storage of mercury and its tissue distribution. Seleno-enzymes metabolize this essential mineral, and also have anti-oxidative properties that help eliminate the reactive oxygen species induced by mercury. Adequate intake of selenium can protect against the effects of mercury. This is especially true for individuals carrying a variant form of the SEPP1 gene. The SEPP1 gene products interact with dietary selenium, allowing certain antioxidant defense properties in tissues such as brain and testis. The SEPP1 variant form reduces protective binding of seleno-proteins to mercury.

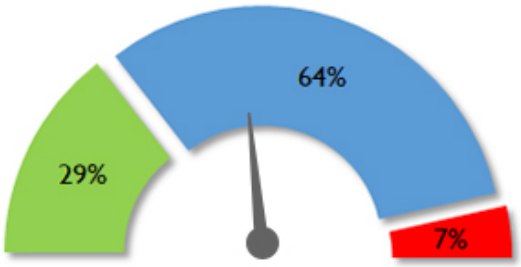
A variation in the INMT gene is a major predictor for the rate of selenium excretion out of the body. The variant form of the gene lowers selenium levels and allow higher levels of mercury to accumulate.

CHEMICAL LEAD

GENETIC FINDINGS SUMMARY

Your total Genetic Risk Score indicates that your body's response to Lead is normal. Genetic sensitivity to lead is not expected.

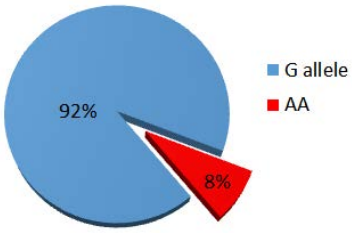
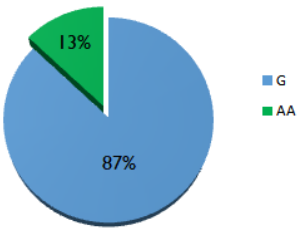
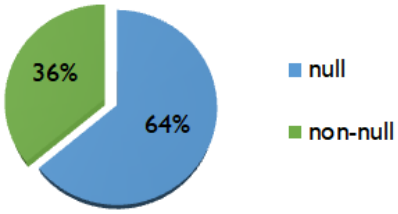
You may wish to take the general precautions to further reduce your risk. Visit www.geneinformed.com/lead for more information.



Your Total Genetic Risk score

NORMAL RISK

Your genetic makeup Lead metabolizing enzymes

Genes	**ALAD G>A**	**GSTP1 A>G**	**GSTM1**
Frequency in the population			
Your results	GA	GG	null

CHEMICAL LEAD

About lead

Lead (Pb) poses an enormous risk to human health due to its wide distribution in the environment. Although the risk of exposure to acute, high levels of lead has been recently reduced in developed countries, it is still a major risk in developing countries. However, continuous exposure to small quantities of lead is a serious risk all over the world. Chronic exposure to lead has irreversible toxicity in several human organs and systems, such as the nervous, blood and bones, reproductive systems, kidney and more.

Routes of exposure

The use of lead was phased out in the USA. However, lead is still present everywhere. It is especially found in older houses, since house paint made before 1978 contains lead.

Lead-contaminated soil is still a major problem around highways and in some urban settings. Some soil close to walls of older houses contains lead.

Lead particles may also be released into our tap water by pipes, brass plumbing fixtures and copper pipes soldered with lead. Lead release follows corrosion, most likely to happen when water has a high acid or low mineral content and sits inside pipes for several hours. While homes built before 1986 are the most likely to have lead plumbing, it can be found in newer homes as well. Until two years ago, the legal limit for "lead-free" pipes was up to 8% lead.

CHEMICAL LEAD

As of January 1, 2014, all newly installed water faucets, fixtures, pipes and fittings must meet new lead-free requirements, which reduce the amount of lead allowed to 0.25%. The Environmental Protection Agency says between 10% and 20% of exposure to lead comes from contaminated water. Babies can get between 40% and 60% of their exposure to lead by drinking formula mixed with contaminated water.

Foods stored in pewter, lead crystal glassware or pottery containing lead-based glazing may become contaminated with lead. Imported cans from specialty stores with irregularly soldered side seams may contain high levels of lead.

Most lead poisoning in children results from eating chips of deteriorating lead-based paint. Toys, household items and playground facilities painted before 1978 are major routes of exposure to lead, as well as toys made and painted outside the United States.

Workers can be exposed to lead and can bring it home on their clothes when they work in auto repair, mining, pipe fitting, battery manufacturing, painting, construction and certain other fields.

Lead is a component of tobacco and tobacco smoke. Smokers and second-hand smokers have higher blood lead levels than do people not exposed to cigarettes smoke.

CHEMICAL LEAD

Other sources for lead exposure include:

- bullets, curtain weights, old coins, medals and fishing sinkers made of lead
- jewelry, pottery, and lead figures
- storage batteries
- some traditional ethnic or alternative medicines - some cosmetics manufactured abroad
- illicit opium

Health effects

Lead alters the ability of various cell signaling pathways that use calcium. It also promotes the formation of free radicals, and therefor lead is defined as a potential human carcinogen (group 2A) by the International Agency for Research on Cancer (IARC).

Other than promoting cancer, chronic lead exposure can cause many systematic effects such as hypertension, anemia, cognitive deficits, infertility, immune imbalances, delayed development, vitamin D deficiency, and gastrointestinal effects.

Successive rounds of the US National Health and Nutrition Examination Survey (NHANES), both before and after lead reduction measures, have shown associations between blood lead, cardiovascular and cancer deaths, and all-cause mortality.

Young children are most vulnerable to lead, which may cause severe mental and physical impairment. Any damage caused by lead poisoning cannot be reversed.

CHEMICAL LEAD

The Institute for Health Metrics and Evaluation (IHME) estimated that in 2016 lead exposure accounted for 540,000 deaths and 13.9 million years of healthy life lost (disability-adjusted life years (DALYs)) worldwide due to long-term effects on health. The highest burden was in low- and middle-income countries. IHME also estimated that in 2016, lead exposure accounted for 63.8% of the global burden of idiopathic developmental intellectual disability, 3% of the global burden of ischaemic heart disease and 3.1% of the global burden of stroke.

Genetic sensitivity to lead

The enzyme aminolevulinate dehydratase (ALAD) is essential for the synthesis of some blood cell particles. Lead inhibits ALAD activity, and measurement of the enzyme levels has been used as a biomarker of lead exposure. Genetic polymorphism in this gene is associated with susceptibility to lead poisoning, as well as various neurological or haematological aspects of lead toxicity.

The GSTP1 gene encodes for a glutathione S-transferase P1 enzyme. The GSTM1 gene encodes for the glutathione S-transferase M1 enzyme. Both play a significant role in antioxidant defense mechanisms. Genetic variants of these genes were associated with lead toxicity.

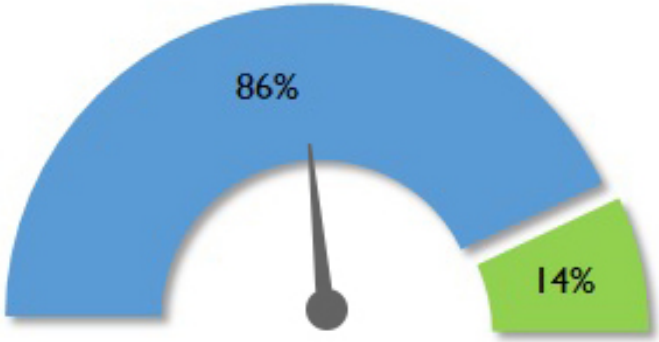
CHEMICAL PESTICIDES

CHLORPYRIFOS *ORGANOPHOSPHATE INSECTICIDE*

GENETIC FINDINGS SUMMARY

Average metabolism of chlorpyrifos is expected.

Your sensitivity to pesticides containing this chemical is similar to the average person.



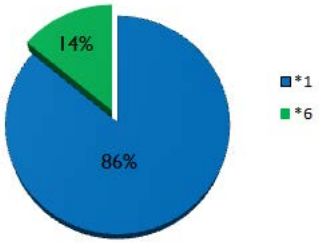
Your Total Genetic Risk score

AVERAGE RISK

Your genetic makeup Chlorpyrifos metabolizing enzymes

Genes **CYP2B6**

Frequency in the population



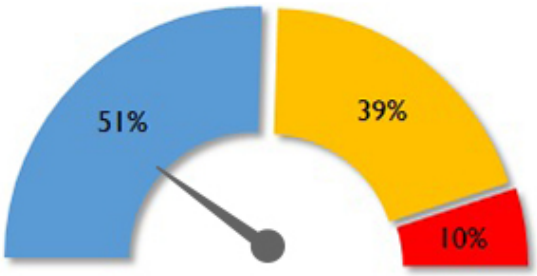
Your result *1

CHEMICAL PESTICIDES

***GENERAL** ORGANOPHOSPHATE INSECTICIDES*

GENETIC FINDINGS SUMMARY

You carry the common genetic setup for the NOS1 gene. This gene version response to organophosphate pesticides is normal and puts you at an average risk for the negative health outcomes of these pesticides. You may follow the advice for the general public. See more at www.geneinformed.com/pesticides



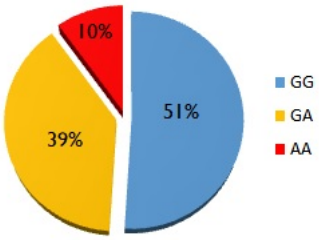
Your Total Genetic Risk score

NORMAL RISK

Your genetic makeup Organophsphate metabolizing enzymes

Genes **NOS1 G>A**

Frequency in the population



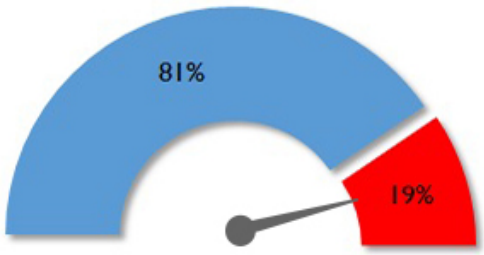
Your result GG

CHEMICAL PESTICIDES

PYRETHROID INSECTICIDES

GENETIC FINDINGS SUMMARY

Your genetic setup is associated with high genetic sensitivity to pyrethroid insecticides. You are more vulnerable to chronic inflammation over exposure to pyrethroids. Your risk for Parkinson's disease is greater than average if exposed to pyrethroids in the ambient air.



Your Total Genetic Risk score

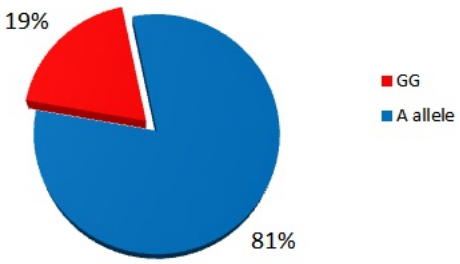
HIGH RISK

Your genetic makeup Pyrethroid metabolizing enzymes

Genes

HLA-DRA A>G

Frequency in the population



Your result GG

CHEMICAL PESTICIDES

Personalized Recommendations

Its healthy to eat fresh fruits and vegetables, so don't stop doing so. It is possible to reduce exposure to pesticides by taking these steps:

1. Choose cleaner. Some fruits and vegetables are more contaminated than others. High levels of pesticide residues in produce sampled in the US were found in leafy greens such as spinach, lettuce, celery, cabbage, cilantro and parsley. Such crops are frequently treated and tend to absorb a lot of the chemical. Same goes with strawberries, raspberries
   and wolfberries. Grapes, pome fruit like apple and pear, and stone fruits like apricots, peaches, nectarines, cherries and plum tend to contain more pesticide residues. Tomatoes and bell peppers lead the list for vegetables.

2. Consider buying organic produce, especially for fruits and vegetables that were listed here as more contaminated. Controlled trials have shown that consumption of organic food significantly decreased organophosphate pesticides levels in the blood serum of study participants.

3. Peeling can remove some pesticides, but many of these chemicals penetrate deep. Systemic pesticides are applied via the roots and reach most of the plant's tissues, including the fruit.
   Another down side for peeling is missing out on the nutrient-rich skin. Did you know that potato skin is particularly rich in vitamin C?

CHEMICAL PESTICIDES

4. To reduce the load of pesticides on your body, make sure you clean the produce thoroughly. Rinse fruits and vegetables under running water for at least 30 seconds, rubbing the skin with your fingers if the skin is delicate or with a brush if the skin is tougher. For produce that's difficult to scrub thoroughly with a brush because of its shape or the number of cracks and crevasses it has, like cauliflower, broccoli or lettuce, soak for one to two minutes in cold water. Wash fragile fruit like berries in a colander under running water using a kitchen faucet spray attachment. Don't use soap, detergents or bleach to wash produce to remove pesticides as they can contain substances that aren't safe to eat. Special vegetable rinses were not found any more effective than soaking in clean tap water. A 15-minute baking soda rinse was most effective for getting rid of some pesticides, but not all. A concentration of about one teaspoon of baking soda per two cups of water should do the job. Thoroughly dry the produce after washing and before eating to remove more pesticide residue.

5. Pesticide residues can be found in commodities as well, such as oil and rice, and in other processed foods. That's why it is important to support our body with varied and rich plant-based diet. Check appendix A. for a list of food, beverages, and bioactive compounds with demonstrated, or potential, clinical impact on detoxification systems.

CHEMICAL PESTICIDES

6. Try to avoid or reduce the use of pesticides in your house, including your garden and on your pets. Use natural pest control methods when possible. Make pesticides your last resort when other options have failed. Make sure to use pesticide applications with precaution, as instructed by the label.

7. Pesticide residues can be found in house dust or on surfaces like garden furniture and playgrounds. People living close to fields or where pesticides are used in the outdoors can reduce your exposure by cleaning such surfaces and vacuuming more often.

Most contaminated fruit and vegetables

High levels of pesticides residues found in lab tests

Strawberries
Apiaceae vegetables- carrot, celery, fennel, parsley, parsnip

Cruciferous vegetables- cauliflower, cabbage, garden cress, bok choy, broccoli,

Brussels sprouts

Soybean, black soybean

Purple sweet potato

Cleanest produce

Lowest levels of pesticides residues found in lab tests

Pomegranate

Black raspberry

Blueberry

Citrus

CHEMICAL PESTICIDES

About pesticides

The use of pesticides is so integral in modern agriculture that residues are in everything we eat. Our body is constantly exposed to various pesticides, herbicides and fungicides that it has to deal with.

Solid and large-scale studies indicate that low-dose, long-term exposure is a root cause for health related disorders such as cancer and neurodegenerative, respiratory, reproductive and developmental disorders.

Routes of exposure

We are all inevitably exposed to pesticides in our diet. Some restrictions are set on use of pesticides in agriculture, but there are major doubts whether these are enough to protect our health. Even if current standards for each pesticide are kept, public health experts and scientists are worried over the combined effect of overall pesticide exposure. We ingest a cocktail of many pesticides, and exposed to various other carcinogens and toxicants. Since different chemicals are metabolized by the same detoxification enzymes, the overall burden is thought to overwhelm our capacities and lead to many chronic diseases that are common today.

Other than in our food, residues of pesticides and fertilizers find their way into our drinking water as well. Pesticides may contaminate the ambient air of houses that's in proximity to fields or when household insecticides are used.

CHEMICAL PESTICIDES

Health effects

One of the most prominent effects of pesticides is induction of oxidative stress, resulting in an increase of free radicals that damage biological molecules and promoting inflammation, immune response, chronic diseases and aging.

Different pesticides are associated with various maladies.
Organophosphate insecticides (OP's) are very common in the last decades, due to its relatively short half-life, relatively fast degradation rates, lower price, lower susceptibility to pest resistance and lastly, due to the ban on persistent organochlorine pesticides in the 1970s. However, as evidence accumulates, more and more health concerns are raised. The organophosphate insecticides are potent inhibitors that alter receptors' activity in the nervous system. Associated effects on human health include disruption of the endocrine system, neuropsychological disorders, developmental anomaly, hypersensitivity, non-Hodgkin’s lymphoma, lung and prostate cancer and Parkinson's disease.

The class of insecticide called pyrethroid promotes Parkinson’s
disease. Pyrethroids constitute the majority of commercial household insecticides and insect repellents.

Genetic sensitivity to pesticides

Various enzymes are your front line against pesticides. These enzymes, mainly found in the liver, metabolize and detoxify various toxic chemicals.

Some of us are born more apt to deal with pesticides, while others are especially sensitive to its effects on health. Individuals with unfavourable combinations of gene variants are more prone to pesticides' adverse health outcomes, having less efficient enzyme activity. Others enjoy gene versions that cope better with pesticides, coding for enzymes that allow faster secretion or better detoxification rates.

CHEMICAL PESTICIDES

The HLA-DRA marker is found on the surface of various cells and serve as markers needed to initiate immune responses. Genetic variation in the gene is associated with increased risk for Parkinson's disease, synergized with exposure to a commonly used class of insecticide called pyrethroid. This gene–environment interaction promotes risk via alterations in the immune responses, however in Caucasians only.

Organophosphate pesticides are strongly associated with Parkinson's disease among participants with variant genotypes in the NOS1 gene. The Nitric Oxide Synthase gene is involved in producing nitric oxide (NO), a pro-oxidant that can damage neurons. The variant T allele of NOS1 increases risk for PD with pesticide use in household, and ambient agricultural pesticide applications exposure.

Chlorpyrifos (CPS) is a broad-spectrum organophosphate insecticide that is neurotoxic in humans, and its use has been restricted in many countries. Production of the toxic metabolite of chlorpyrifos varies wildly between individuals. CYP2B6 is the main enzyme metabolizing chlorpyrifos in the human brain, the critical site for chlorpyrifos poisoning. Individual genetic background in this gene influences the rate of metabolic activation of chlorpyrifos and its toxicity. Individuals carrying two copies of a variant known as CYP2B6*6 produce significantly less of the toxic derivative of chlorpyrifos. These homozygous CYP2B6*6 are more resistant to the adverse effects of chlorpyrifos and similar pesticides.

APPENDIX A. Support your detoxification systems

Individuals carrying a genetic sensitivity to a chemical are encouraged to follow the chemical-specific recommendations presented in their report. In addition, it is advised to take some general measures that will boost your general detoxification abilities, and promote health and wellness.

The basic actions to reduce the burden of toxicity on your body are to stop smoking, perform physical activity regularly and reduce emotional stress.

As for nutritional advice, follow a balanced diet favoring whole, unprocessed, plant-based foods. In addition, enrich your diet with the foodstuffs listed below. These were found to support human detoxification capabilities and help us cope with different carcinogens and toxicants.

Food, beverages, and bioactive compounds with demonstrated or potential clinical impact on detoxification systems.

Vegetables	Fruits
Allium vegetables- garlic, onion, shallots, chives, leeks	Pomegranate
Apiaceae vegetables- carrot, celery, fennel, parsley, parsnip	Black raspberry
Cruciferous vegetables- cauliflower, cabbage, garden cress, bok choy, broccoli, Brussels sprouts	Blueberry
Soybean, black soybean	Citrus
Purple sweet potato	

Beverages	Herbs and spices
Coffee	Chicory root
Chicory coffee substitute	Chives
Black tea, green tea, peppermint, rooibos, chamomile, dandelion, honeybush tea	Rosemary
	Turmeric
	Curcumin
	Ginger

APPENDIX A. Support your detoxification systems

Bioactive compounds marketed as supplements*	Naturally found in
Catechins, EGCG	Highest content is in cocoa. Also high in tea, especially green tea. Also found in apple, pear, prune juice, Açaí oil, argan oil, vinegar
Daidzein	Soy
Genistein	Soy, fava bean, and in coffee
Chrysin	Passionflower, silver linden, geranium species, honey and bee propolis
Ellagic acid	Raspberries, strawberries, blackberries, cranberries, grapes, pomegranate, guava, pecans, walnuts
Ferulic acid	Wheat bran, brown rice, barley, popcorn, navy beans
Luteolin	Carrots, artichoke, peppers, celery, olive oil, peppermint, thyme, rosemary, oregano
Lycopene	Tomatoes and ketchup, watermelon, pink grapefruit, pink guava, papaya, seabuckthorn, goji berry, rosehip
MCTs- Medium Chain Triglycerides	Coconut oil, butter, whole milk and cheddar cheese
Myricetin/ myricetol	Oranges, peppers, garlic, cashew, guava, French beans, tomato, apple, green and black tea
N-acetyl cysteine (NAC)	Various animal proteins, plant sources
Naringenin	Grapes, oranges, tomato skins
Quercetin	Capers, red onion, watercress, kale
Resveratrol	Red wine, red grapes, chocolate
Omega-3	Fish oil

Adopted from Hodges R. and Minich D. Modulation of Metabolic Detoxification Pathways Using Foods and Food-Derived Components: A Scientific Review with Clinical Application. Journal of Nutrition and Metabolism. 2015 vol: 2015 pp: 1-23

**CONSULT YOUR DOCTOR before making any lifestyle changes following this report. Your doctor is able to consider additional factor such as current medical status and pharmaceuticals in use.*