GAYLE MERCHANT
— Nutrition & Functional Medicine —

GPL-TOX

Screens for 173 different environmental pollutants using 18 different metabolites, all from a single urine sample.

The Great Plains Laboratory, Inc.

GPL-TOX

The GPL-TOX analyses chemicals can accumulate in our bodies causing disruption to our physiological functioning and illness such as chronic fatigue syndrome, cancer, Alzheimer's disease, Parkinson's disease, sensitivities, skin conditions and more are on the increase. Many of these toxins affect reproductive health, the immune system, the central nervous system causing persistent headaches, nausea, concentration problems, fatigue, weakness, brain fog and dizziness. Some toxins have been linked to the development of autism and complications in pregnancy. Others can cause problems with liver and kidney function, cardiovascular health and cancer.

Price: £266.25

DESCRIPTION

The test is for people who:

- Have neurological concerns
- Issues with mood and anxiety
- Autism
- ADHD (attention deficit hyperactivity disorder)
- Cancer
- Chronic Fatigue
- Fibromyalgia
- Autoimmune conditions
- Have jobs that may have exposed them to such chemicals

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GAYLE MERCHANT
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Sample Test results

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MEMBER BANT

CNM COLLEGE OF NATUROPATHIC MEDICINE
QUALIFIED NUTRITIONAL THERAPIST

Clinical Education IFM
AFMCP-UK GRADUATE
LONDON 2018

CNHC Complementary & Natural Healthcare Council
professional standards authority
accredited register

THE BRAIN HEALTH PROGRAMME

Toxic Non-Metal Chemical Profile

Requisition #:
Patient Name:
Patient Age:
Sex:

Physician Name:
Date of Collection:
Time of Collection:
Print Date:

Toxic Compounds

Metabolite	Result µg/g creatinine	Percentile
Industrial Toxicants		
1) 2-Hydroxyisobutyric Acid (2HIB)	2,445	LLOQ 200 — 75th 5,530 — 95th 7,000

Parent: MTBE/ETBE

MTBE and ETBE are gasoline additives used to improve octane ratings. Exposure to these compounds is most likely due to groundwater contamination, inhalation or skin exposure to gasoline or its vapors, and exhaust fumes. MTBE has been demonstrated to cause hepatic, kidney, and central nervous system toxicity, peripheral neurotoxicity, and cancer in animals. Very high values have been reported in genetic disorders. Because the metabolites of these compounds are the same, ETBE may be similarly toxic.

2) Monoethylphthalate (MEP)	4.4	LLOQ 5.0 — 75th 150 — 95th 850

Parent: Diethylphthalates

Phthalates may be the most widespread group of toxins in our environment, commonly found in many bath and beauty products, cosmetics, perfumes, oral pharmaceuticals, insect repellants, adhesives, inks, and varnishes. Phthalates have been implicated in reproductive damage, depressed leukocyte function, and cancer. Phthalates have also been found to impede blood coagulation, lower testosterone, and alter sexual development in children. Low levels of phthalates can feminize the male brain of the fetus, while high levels can hyper-masculinize the developing male brain.

3) 2-3-4 Methylhippuric Acid (2,-3-,4-MHA)	93	LLOQ 10 — 75th 388 — 95th 1,220

Parent: Xylene

Xylenes (dimethylbenzenes) are found not only in common products such as paints, lacquers, pesticides, cleaning fluids, fuel and exhaust fumes, but also in perfumes and insect repellents. Xylenes are oxidized in the liver and bound to glycine before eliminated in urine. High exposures to xylene create an increase in oxidative stress, causing symptoms such as nausea, vomiting, dizziness, central nervous system depression, and death. Occupational exposure is often found in pathology laboratories where xylene is used for tissue processing.

4) Phenylglyoxylic Acid (PGO)	259	LLOQ 5.0 — 75th 255 — 95th 394

Parent: Styrene/Ethylbenzene

Styrene is used in the manufacturing of plastics, in building materials, and is found in car exhaust fumes. Polystyrene and its copolymers are widely used as food-packaging materials. The ability of styrene monomer to leach from polystyrene packaging to food has been reported. Occupational exposure due to inhalation of large amounts of styrene adversely impacts the central nervous system, causes concentration problems, muscle weakness, fatigue, and nausea, and irritates the mucous membranes of the eyes, nose, and throat.

**LLOQ - Lower Limit of Quantitation*

***N.D. - Not Detected*

Testing performed by The Great Plains Laboratory, Inc., Lenexa, Kansas. The Great Plains Laboratory has developed and determined the performance characteristics of this test. This test has not been evaluated by the U.S. FDA; the FDA does not currently regulate such testing.

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The Great Plains Laboratory, Inc.

Requisition #:
Patient Name:
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Sex:

Physician Name:
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Toxic Compounds

Metabolite	Result µg/g creatinine	Percentile
5) N-acetyl phenyl cysteine (NAP)	0.20	LLOQ 0.20 · 75th 1.3 · 95th 3.3

Parent: Benzene

Benzene is an organic solvent that is widespread in the environment. Benzene is a by-product of all types of industrial processes and combustion, including motor vehicle exhaust and cigarette smoke, and is released by outgassing from synthetic materials. Benzene is an extremely toxic chemical that is mutagenic and carcinogenic. High exposures to benzene cause symptoms of nausea, vomiting, dizziness, lack of coordination, central nervous system depression, and death. It can also cause hematological abnormalities.

6) N-acetyl(2-cyanoethyl)cysteine (NACE)	1.2	LLOQ 1.0 · 95th 256

Parent: Acrylonitrile

Acrylonitrile is a colorless liquid with a pungent odor. It is used in the production of acrylic fibers, resins, and rubber. Use of any of these products could lead to exposure to acrylonitrile. Smoking tobacco and cigarettes is another potential exposure. Exposure to acrylonitrile can lead to headaches, nausea, dizziness, fatigue, and chest pains. The European Union has classified acrylonitrile as a carcinogen.

7) Perchlorate (PERC)	1.8	LLOQ 2.0 · 75th 5.5 · 95th 15

Parent: Perchlorate

This chemical is used in the production of rocket fuel, missiles, fireworks, flares, explosives, fertilizers, and bleach. Studies show that perchlorate is often found in water supplies. Many food sources are also contaminated with percholate. Percholate can disrupt the thyroid's ability to produce hormones. The EPA has also labeled perchlorate a likely human carcinogen. Patients that are high in perchlorate can use a reverse osmosis water treatment system.

8) Diphenyl phosphate (DPP)	0.75	LLOQ 1.0 · 75th 1.8 · 95th 5.6

Parent: Diphenyl Phosphate

This is a metabolite of the organophosphate flame retardant triphenyl phosphate (TPHP), which is used in plastics, electronic equipment, nail polish, and resins. TPHP can cause endocrine disruption. Studies have also linked TPHP to reproductive and developmental problems.

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Toxic Compounds

Metabolite	Result µg/g creatinine	Percentile
9) 2-hydroxyethyl mercapturic (HEMA)	3.9	LLOQ 0.80 · 75th 1.7 · 95th 4.8

Parent: Ethylene oxide, Vinyl chloride, Halopropane

High HEMA may be due to exposure to ethylene oxide, which is used in many different industries including agrochemicals detergents, pharmaceuticals, and personal care products. Ethylene oxide is also used as a sterilant on rubber, plastics, and electronics. Chronic exposure to ethylene oxide has been determined to be mutagenic to humans. Multiple agencies have reported it as a carcinogen. Studies of people exposed to ethylene oxide show an increased incidence of breast cancer and leukemia. Ethylene oxide may be difficult to detect since it is odorless at toxic levels.

High HEMA may also due to exposure to vinyl chloride, an intermediate in the synthesis of several major commercial chemicals, including polyvinyl chloride, and used in the past as an aerosol propellant. Exposure to vinyl chloride has been associated with increased incidence of autism. High concentrations of vinyl chloride may cause central nervous system depression, nausea, headache, dizziness, liver damage and liver cancer, degenerative bone changes, thrombocytopenia, enlargement of the spleen and even death. To reduce exposure to vinyl chloride, eliminate use of plastic containers for cooking, reheating, eating or drinking (especially warm or hot) food or beverages. Replace these containers with glass, paper, or stainless steel whenever possible. Elimination of vinyl chloride can also be accelerated by sauna treatment, the Hubbard detoxification protocol employing niacin supplementation, vitamin B-12 therapy, by glutathione (reduced) supplementation (oral, intravenous, transdermal, or precursors such as N-acetyl cysteine [NAC]).

10) N-acetyl(propyl)cysteine (NAPR)	2.9	LLOQ 4.0 · 75th 11 · 95th 46

Parent: 1-bromopropane

1-bromopropane is an organic solvent used for metal cleaning, foam gluing, and dry cleaning. Studies have shown that 1-BP is a neurotoxin as well as a reproductive toxin. Research indicates that exposure to 1-BP can cause sensory and motor deficits. Chronic exposure can lead to decreased cognitive function and impairment of the central nervous system. Acute exposure can lead to headaches.

11) N-acetyl(2-hydroxypropyl)cysteine (NAHP)	25	LLOQ 4.0 · 75th 101 · 95th 403

Parent: Propylene oxide

This chemical is used in the production of plastics and is used as a fumigant. Propylene oxide is used to make polyester resins for textile and construction industries. It is also used in the preparation of lubricants, surfactants, and oil demulsifiers. It has also been used as a food additive, an herbicide, a microbicide, an insecticide, a fungicide, and a miticide. Propylene oxide is a probable human carcinogen.

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Toxic Compounds

Metabolite	Result µg/g creatinine	Percentile
12) N-acetyl-S-(2-carbamoylethyl)cysteine (NAE)	47	LLOQ 4.0 · 75th 82 · 95th 199

Parent: Acrylamide

Acrylamide can polymerize to form polyacrylamide. These chemicals are used in many industrial processes such as plastics, food packaging, cosmetics, dyes, and treatment of drinking water. Food and cigarette smoke are also two major sources of exposure. Acrylamide has been found in foods like potato chips and French fries. This is because asparagine, an important amino acid for central nervous system function, can produce acrylamide when cooked at high temperature in the presence of sugars. Foods rich in asparagine include asparagus, potatoes, legumes, nuts, seeds, beef, eggs, and fish, and are potential sources of exposure to acrylamide. High levels of acrylamide can elevate a patient's risk of cancer. In addition, acrylamide is known to cause neurological damage.

13) N-acetyl(3,4-dihydroxybutyl)cysteine (NADB)	823	LLOQ 4.0 · 75th 374 · 95th 583

Parent: 1,3 butadiene

This is a chemical made from the processing of petroleum. It is often a colorless gas with a mild gasoline-like odor. Most of this chemical is used in the production of synthetic rubber. 1,3 butadiene is a known carcinogen and has been linked to increased risk of cardiovascular disease. Individuals that come into contact with rubber, such as car tires, could absorb 1,3 butadiene through the skin. The increased use of old tires in the production of crumb rubber playgrounds and athletic fields is quite concerning since soccer players on such fields have increased cancer rates.

Organophosphate Insecticide Metabolites

14) Dimethylphosphate (DMP)	11	LLOQ 4.0 · 75th 9.1 · 95th 34

Parent: Organophosphates

Organophosphates are one of the most toxic groups of substances in the world, primarily found in pesticide formulations. They are inhibitors of cholinesterase enzymes, leading to overstimulation of nerve cells, causing sweating, salivation, diarrhea, abnormal behavior, including aggression and depression. Children exposed to organophosphates have more than twice the risk of developing pervasive developmental disorder (PDD), an autism spectrum disorder. Maternal organophosphate exposure has been associated with various adverse outcomes including having shorter pregnancies and children with impaired reflexes.

15) Diethylphosphate (DEP)	5.3	LLOQ 0.60 · 75th 2.7 · 95th 12

Parent: Organophosphates

Organophosphates are one of the most toxic groups of substances in the world, primarily found in pesticide formulations. They are inhibitors of cholinesterase enzymes, leading to overstimulation of nerve cells, causing sweating, salivation, diarrhea, abnormal behavior, including aggression and depression. Children exposed to organophosphates have more than twice the risk of developing pervasive developmental disorder (PDD), an autism spectrum disorder. Maternal organophosphate exposure has been associated with various adverse outcomes including having shorter pregnancies and children with impaired reflexes.

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Toxic Compounds

Metabolite	Result µg/g creatinine	Percentile
Herbicide		
16) 2,4-Dichlorophenoxyacetic Acid (2-,4-D)	3.8	LLOQ 0.20 · 75th 0.50 · 95th 1.9

2,4-Dichlorophenoxyacetic Acid (2,4-D) is a very common herbicide that was a part of Agent Orange, which was used by the U.S. in the Vietnam War. It is most commonly used in agriculture on genetically modified foods, and as a weed killer for lawns. Exposure to 2, 4-D via skin or oral ingestion is associated with neuritis, weakness, nausea, abdominal pain, headache, dizziness, peripheral neuropathy, stupor, seizures, brain damage, and impaired reflexes. 2, 4-D is a known endocrine disruptor, and can block hormone distribution and cause glandular breakdown.

Pyrethroid Insecticide		
17) 3-Phenoxybenzoic Acid (3PBA)	4.3	LLOQ 0.30 · 75th 1.0 · 95th 5.8

Parent: Pyrethroids - Including Permethrin, Cypermethrin, Cyhalothrins, Fenpropathrin, Deltamethrin, Trihalomethrin

Pyrethrins are widely used as insecticides. Exposure during pregnancy doubles the likelihood of autism. Pyrethrins may affect neurological development, disrupt hormones, induce cancer, and suppress the immune system.

Metabolite	Result Creat mmol/mol	Percentile
Marker for Mitochondria Function		
18) Tiglylglycine (TG)	0.75	LLOQ 0.04 · 75th 4.7 · 95th 11

Tiglylglycine (TG) is a marker for mitochondrial disorders resulting from mutations of mitochondrial DNA, which can manifest from exposure to toxic chemicals, infections, inflammation, and nutritional deficiencies. TG indicates mitochondrial dysfunction by monitoring a metabolite that is elevated in mitochondrial deficiency of cofactors such as NAD+, flavin-containing coenzymes, and Coenzyme Q10. Disorders associated with mitochondrial dysfunction include autism, Parkinson's disease, and cancer.

Note: The reference ranges for Tiglylglycine have been updated based on an analysis of almost 1000 total patients. The previous values were based on a more limited patient population.

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Metabolite	Result µg/g creatinine	Percentile

List of Organophosphate Insecticides that are converted to DMP

14) Dimethylphosphate (DMP)	11

-Amidithion
-Anilofos
-Azamethiphos
-Azinphos
-Azinphos-methyl
-Azinphos-methyl oxygen analog
-Azothoate
-Bomyl
-Bromophos
-Chlorpyrifos-methyl
-Chlorthion
-cis-Azodrin
-cis-Methocrotophos
-Crotoxyphos
-Cyanophos
-Cythioate
-DDVP
-Demephion-O
-Demephion-S
-Demeton-O-methyl
-Demeton-S-methyl
-Dicrotophos
-Dimethoate
-Dimethoate-ethyl
-DMCP
-Endothion
-Etrimfos
-Famphur
-Famphur O-analog
-Fenitrothion
-Fenthion
-Fenthion oxon
-Formothion
-Fosmethilan
-Fospirate
-Heptenophos
-Iodofenfos
-Isazophos-methyl
-Isochlorthion
-Isothioate
-Lythidathion
-Malaoxon
-Malathion
-Menazon
-Methacrifos
-Methidathion OA
-Methyl paraoxon
-Methyl phenkapton
-Methyl trithion
-Mevinphos
-(E)-Mevinphos
-(Z)-Mevinphos
-Monocrotophos
-Morphothion
-Naled
-OOS-Trimethyl phosphorodithiate
-Omethoate
-Oxydemeton-methyl
-Phenthoate
-Phosmet
-Phosmetoxon
-Phosnichlor
-Phosphamidon
-Phoxim-methyl
-Pirimiphos-methyl
-Quinalphos-methyl
-Ronnel
-Sophamide
-Temephos
-Temephos sulfoxide
-Tetrachlorvinphos
-Thiometon
-Tolclofos-methyl
-Vamidothion

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Sex:

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Print Date:

Metabolite	Result µg/g creatinine	Percentile

List of Organophosphate Insecticides that are converted to DEP

		LLOQ	75th	95th
15) Diethylphosphate (DEP)	5.3	0.60	2.7	12

-Acethion
-Acetoxon
-Akton
-Amiton
-Amiton oxalate
-Athidathion
-Azethion
-Azinphos-ethyl
-Bromophos-ethyl
-Butathiofos
-Carbophenothion
-Chlorethoxyphos
-Chlorfenvinphos
-Chlorphoxim
-Chlorprazophos
-Chlorpyrifos
-Chlorpyrifos oxygen analog
-Chlorthiophos
-Chlorthiophos II
-Chlorthiophos III
-Coumaphos
-Coumithioate
-Cyanthoate
-Demeton
-Demeton-O
-Demeton-S
-Dialifor
-Diazinon
-Diazoxon
-Dichlofenthion
-5-Dichloro-alpha-(chloro-methylene) benzyl diethyl phosphate
-Diethyldithio phosphate
-Diethylthio phosphate
-Dioxathion
-Disulfoton
-Disulfoton sulfone
-Disulfoton sulfoxide
-Ethion
-Ethion O-analog
-Fensulfothion
-Isazophos
-Isoxathion
-Mecarbam
-Miral
-Naphthalophos
-OO-diethyl O-naphthaloximido phosphorothioate
-OO-diethyl phosphoro chloridothionate
-OO-Diethyl S-(46-dimethyl-2-pyrimidinyl) phosphorodithioate
-OO-diethyl-O-phenyl phosphoro thioate
-Paraoxon
-Parathion
-Phenkapton
-Phorate
-Phosalone
-Phoxim
-Pirimiphos ethyl
-Primidophos
-Propoxon
-Prothidathion
-Prothion
-Prothoate
-Pyrazophos
-Pyridiphenthion
-Quinalphos
-Quinothion
-Sulfotep
-TEPP
-Terbufos
-Terbufos sulfone
-Terbufos sulfoxide
-Thionazin
-Thionazin O-analog
-Triazophos

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SAMPLE REPORT

11813 West 77th Street Lenexa, KS 66214 | (913) 341-8949 | Fax: (913) 341-6207 | www.GPL4U.com

The Great Plains Laboratory, Inc.

Requisition #:	*Physician Name:*
Patient Name:	*Date of Collection:*
Patient Age:	*Time of Collection:*
Sex:	*Print Date:*

Interpretation

2-hydroxyisobutyric acid (2HIB) (Marker 1) is most often the result of exposure to methyl tertiary-butyl ether (MTBE) or ethyl tertiary butyl ether (ETBE), which are gasoline additives used as octane enhancers. MTBE has been found to pollute large quantities of groundwater when gasoline with MTBE is spilled or leaked at gas stations. In addition, MTBE and ETBE are volatile and may be inhaled or absorbed through the skin by drivers during fueling or from exhaust exposure. MTBE and its metabolites have been shown to be to cause hepatic, kidney and central nervous system toxicity, peripheral neurotoxicity, and cancer in animals. Excretion half-lives in humans range from 10 to 28 hours. Reduce exposure if possible. Elimination is accelerated by sauna therapy, by the Hubbard detoxification protocol employing niacin supplementation to aid in MTBE and ETBE excretion, or by treatment with glutathione (reduced) supplementation (oral, intravenous, transdermal, or precursors such as N-acetyl cysteine [NAC]). MTBE and ETBE are also formed endogenously as a product of branched-chain amino acid degradation and ketogenesis. High values have been reported in both isovaleric acidemia and multiple acyldehydrogenase deficiency.

Monoethylphthalate (MEP) (Marker 2) is the result of exposure to phthalates and the major metabolite of diethyl phthalate. Diethyl phthalate makes plastics more flexible and appears in many common household products including food packaging, tools, toothbrushes, toys, aftershave lotions, aspirin, bath products, cosmetics, detergents, eye shadows, hairsprays, insecticides, mosquito repellants, nail extenders, nail polish, nail polish removers, skin care products, hairstyling products, and auto parts . Adults and children are exposed to phthalates through everyday contact with these products as well as contact with indoor air and dust. When mouthed, chewed or sucked in the course of normal play, phthalates leach from toys into children's' mouths. Phthalates have been linked to premature birth, reproductive defects, and early onset puberty. Phthalates have been linked to cancer, autoimmunity, and organ damage in laboratory tests on rodents. Children's' allergies have been linked to phthalate exposure. Phthalate exposure in pregnant women changed the anogenital distance in neonatal boys; a change that in rodents exposed to phthalates was associated with genital abnormalities. Use of infant lotion, infant powder, and infant shampoo were associated with increased infant urine concentrations of phthalate metabolites. Individuals with high values, especially women who want to have children or children who have been exposed, may wish to dramatically reduce their exposures to these substances. Virtually all phthalates may cause quinolinic acid elevation because of interference with tryptophan metabolism. Seven European countries have outlawed two major types of the compounds in cosmetics and baby toys. Elimination of MEP, diethyl phthalate, and all phthalates can be accelerated by sauna treatment, by the Hubbard detoxification protocol employing niacin supplementation, or by glutathione (reduced) supplementation (oral, intravenous, transdermal, or precursors such as N-acetyl cysteine [NAC]).

Methylhippuric acid (2,-3,4-MHA) (Marker 3) is the result of exposure to the solvent xylene that is widespread in the environment. Xylene is found in paints, lacquers, cleaning agents, pesticides, and gasoline. It is also used in the pathology laboratory for tissue processing. High exposure to xylene may cause nausea, vomiting, dizziness, incoordination, central nervous system depression, and even death. An exposure to 100 ppm xylene in the air resulted in a urine value of 3140 µg/g creatinine for methylhippuric acid. Rats given xylene experienced a significant decrease in locomotor activity, deficits in learning ability and memory loss. These xylene-induced behavioral changes were associated with a decrease in beta-endorphins. Treatment begins with removing all potential sources of exposure. Elimination of xylene can be accelerated by sauna treatment, the Hubbard detoxification protocol employing niacin supplementation, supplementation with glycine to encourage metabolism of xylene to methylhippuric acid in the liver, or by glutathione (reduced) supplementation (oral, intravenous, transdermal, or precursors such as N-acetyl cysteine [NAC]).

Phenylglyoxylic acid (PGO) (Marker 4) usually results from exposure to styrene. Reduce exposure by eliminating plastic and styrofoam containers for cooking, reheating, eating or drinking (especially warm or hot) food or beverages. Replace these containers with glass, paper, or stainless steel whenever possible. Elimination of styrene can be accelerated by sauna treatment, reduced glutathione supplementation (oral, intravenous, transdermal, precursors such as N-acetyl cysteine [NAC]). Elimination of styrofoam products is recommended, especially with hot foods.

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N-acetyl phenyl cysteine (NAP) (Marker 5) is the result of exposure to the solvent benzene which is widespread in the environment from cigarette smoke, gasoline, and as a byproduct of all types of combustion, including motor vehicle exhaust. Benzene also outgases from synthetic materials (carpet, drapes, and furniture), glues, and detergents. Numerous industrial processes release this pollutant. Benzene causes hematological abnormalities as well as being mutagenic and carcinogenic. High exposure to benzene may cause nausea, vomiting, dizziness, poor coordination, central nervous system depression, and even death. N-acetyl phenyl cysteine (NAP) is also a metabolic byproduct of potassium sorbate or sorbic acid, a common and safe food preservative. Remove sources of exposure if possible. The solvent can be eliminated by sauna treatment, by the Hubbard detoxification protocol employing niacin supplementation, or by glutathione (reduced) supplementation (oral, intravenous, transdermal, or precursors such as N-acetyl cysteine [NAC]).

2-hydroxyethyl mercapturic (HEMA) (Marker 9)
High HEMA may be due to exposure to ethylene oxide, which is used in many different industries including agrochemicals detergents, pharmaceuticals, and personal care products. Ethylene oxide is also used as a sterilant on rubber, plastics, and electronics. Chronic exposure to ethylene oxide has been determined to be mutagenic to humans. Multiple agencies have reported it as a carcinogen. Studies of people exposed to ethylene oxide show an increased incidence of breast cancer and leukemia. Ethylene oxide may be difficult to detect since it is odorless at toxic levels.

High HEMA may also due to exposure to vinyl chloride, an intermediate in the synthesis of several major commercial chemicals, including polyvinyl chloride, and used in the past as an aerosol propellant. Exposure to vinyl chloride has been associated with increased incidence of autism. High concentrations of vinyl chloride may cause central nervous system depression, nausea, headache, dizziness, liver damage and liver cancer, degenerative bone changes, thrombocytopenia, enlargement of the spleen and even death. To reduce exposure to vinyl chloride, eliminate use of plastic containers for cooking, reheating, eating or drinking (especially warm or hot) food or beverages. Replace these containers with glass, paper, or stainless steel whenever possible. Elimination of vinyl chloride can also be accelerated by sauna treatment, the Hubbard detoxification protocol employing niacin supplementation, vitamin B-12 therapy, by glutathione (reduced) supplementation (oral, intravenous, transdermal, or precursors such as N-acetyl cysteine [NAC]).

Dimethylphosphate (DMP) (Marker 14) indicates exposure to an organophosphate insecticide. Approximately 340 million kilograms of pesticide active ingredient is used agriculturally in the United States annually, and 85% of U.S. households store at least one pesticide for home use. These insecticides kill insects (and mammals such as humans) by the inhibition of the enzyme acetyl-cholinesterase and other enzymes in which serine is part of the active site, such as dipeptidyl peptidase IV. When acetylcholine breakdown is inhibited, overstimulation can lead to constant nerve transmission or overstimulation of neurons or muscles, resulting in excessive salivation, abnormal behavior, diarrhea, urinary incontinence, vomiting, tremors, muscle paralysis, and even death. High exposure levels have been associated with attention deficit, memory impairment and pervasive developmental disorders. Exposure has also been linked to violent behavior, depression, suicide and may have played a role in the onset of Gulf War syndrome. If levels are high, toxicity can be measured by decreased cholinesterase or pseudocholinesterase activity in plasma. Acute toxicity is treated with atropine and/or pralidoxime. DMP is a major metabolite of the following pesticides: methyl azinphos, methyl chlorpyrifos, dichlorvos, dicrotophos, dimethoate, fenitrothion, fenthion, methyl isazaphos, malathion, methidathion, methyl parathion, naled, methyl oxydemeton, phosmet, and methyl pirimiphos. (The complete list is on the report.) Organophosphate exposure can be reduced by eating organic foods, avoiding using pesticides in house or garden, avoiding residence near agricultural areas or golf courses, and staying indoors if insecticides are being sprayed. Lice shampoo, pet flea collars, and flea spray are also major sources of organophosphates. Remove sources of exposure if possible. Elimination of organophosphates can be accelerated by sauna treatment, by the Hubbard detoxification protocol employing niacin supplementation, or by glutathione (reduced) supplementation (oral, intravenous, transdermal, or precursors such as N-acetyl cysteine [NAC]).

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Patient Age:	*Time of Collection:*
Sex:	*Print Date:*

Diethylphosphate, (DEP) (Marker 15) indicates exposure to an organophosphate insecticide. Approximately 340 million kilograms of pesticide active ingredient is used agriculturally in the United States annually, and 85% of U.S. households store at least one pesticide for home use. These insecticides kill insects (and mammals such as humans) by the inhibition of the enzyme acetyl-cholinesterase and other enzymes in which serine is part of the active site such as dipeptidyl peptidase IV. When acetylcholine cannot be broken down, overstimulation can lead to constant nerve transmission or overstimulation of neurons or muscles, resulting in excessive salivation, abnormal behavior, diarrhea, urinary incontinence, vomiting, tremors, muscle paralysis, and even death. High exposure levels have been associated with attention deficit, memory impairment and pervasive developmental disorders. Exposure has also been linked to violent behavior, depression, suicide and may have played a role in the onset of Gulf War syndrome. If levels are high, toxicity can be measured by decreased cholinesterase or pseudocholinesterase activity in plasma. Acute toxicity is treated with atropine and/or pralidoxime. DEP is a major metabolite of the following pesticides: chlorethoxyphos, chlorpyriphos, coumaphos, diazinon, disulfoton, ethion, parathion, and phorate. (The complete list is on the report.) Organophosphate exposure can be reduced by eating organic foods, avoiding using pesticides in house or garden, avoiding residence near agricultural areas or golf courses, and staying indoors if insecticides are being sprayed. Lice shampoo, pet flea collars, and flea spray are also major sources of organophosphates. Remove sources of exposure if possible. Elimination of organophosphates can be accelerated by sauna treatment, by the Hubbard detoxification protocol employing niacin supplementation, or by glutathione (reduced) supplementation (oral, intravenous, transdermal, or precursors such as N-acetyl cysteine [NAC]).

2,4-dichlorophenoxyacetic acid (2,4-D) (Marker 16) can result from exposure to this very common herbicide that was a part of a chemical mixture called Agent Orange <http://envirocancer.cornell.edu/factsheet/pesticide/fs14.2_4-d.cfm>, used by the U.S. during the Vietnam War to increase the visibility for war planes by destroying plant undergrowth and crops. Mean urinary levels of 2,4-D among workers involved in mixing, loading, and applying this herbicide ranged from 5 to 837 µg/L. Median urine 2,4-D concentrations at baseline and a day after application of this herbicide were 2.1 and 73.1 µg/L for farming applicators, and 1.5 and 2.9 µg/L for their children. Herbicides are chemical agents intended to kill unwanted vegetation such as broadleaf weeds and woody plants. They are used in agriculture and on residential properties. People can be exposed to herbicides by breathing them or by skin contact from their residential use or living near application sites, and by eating contaminated food and drinking contaminated water. 2,4-D has a half-life of approximately 12-36 h. Neuritis, weakness, nausea, abdominal pain, headache, dizziness, peripheral neuropathy, stupor, seizures, brain damage, and impaired reflexes have been associated with dermal or oral exposure. 2,4-D is a known endocrine disruptor, and can block hormone distribution and cause glandular breakdown. It is linked to immune system damage, birth defects and reproductive issues possibly due to its frequent contamination with dioxins. Small amounts of 2,4-dichlorophenoxyacetic acid may be found in many urine samples because of widespread environmental contamination. The risk factors associated with low-level exposure are not well established. High values may be treated by removing the person from likely sources of exposure. Elimination of 2,4-D can also be accelerated by sauna treatment, the Hubbard detoxification protocol employing niacin supplementation, vitamin B-12 therapy, by glutathione (reduced) supplementation (oral, intravenous, transdermal, or precursors such as N-acetyl cysteine [NAC]).

If children have high values, parents should avoid lawn chemicals and prevent their children from playing on lawns that use such chemicals.

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Requisition #:	*Physician Name:*
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3-phenoxybenzoic acid (3PBA) (Marker 17) is the result of exposure to pyrethroid insecticides (pyrethrins). Pyrethrins are the collective name for a group of pesticidal compounds derived from pyrethrum flowers in the genus *Chrysanthemum* that includes permethrin, cypermethrin, deltamethrin, cyhalothrins, fenpropathrin and trihalomethrin. Pyrethroids are synthetic analogs of pyrethrins. Pyrethroids may affect neurological development, disrupt hormones, induce cancer, and suppress the immune system. Pyrethroids are axonic poisons that work by keeping the sodium channels open in the neuronal membranes. Inhaling high levels of pyrethrins or pyrethroids may bring about asthmatic breathing, sneezing, nasal stuffiness, headache, nausea, incoordination, tremors, convulsions, facial flushing and swelling, and burning and itching sensation. A 37-year-old woman died of cardiorespiratory arrest after shampooing her dog with pyrethrin shampoo. Individuals who have ragweed sensitivity are especially vulnerable to allergic reactions to these products. Mothers of children with autism spectrum disorder (ASD) were twice as likely to have reported using pet shampoos containing pyrethrins as those who had healthy children; the effect was most severe if exposure was during the second trimester of pregnancy. In addition, parents of ASD children have reported the first onset of autistic behavior after the use of pyrethrin insecticide sprays in the home.

Increased incidence of hyperactivity was associated with any detectable amount of 3-phenoxybenzoic acid in the urine. Most formulations of pyrethrins and pyrethroids also contain piperonyl butoxide, which inhibits cytochrome P-450, increasing the insecticidal efficacy by slowing the metabolic breakdown of pyrethrins and pyrethroids. Thus, the toxicity of such products may be potentiated by simultaneous exposure to piperonyl butoxide. Exposures of animals to these chemicals cause abnormal behaviors and neurological symptoms. 30 million households in the U.S. are estimated to have pyrethrin and pyrethroid products. Remove all sources of exposure. Elimination is accelerated by sauna treatment, by the Hubbard detoxification protocol employing niacin supplementation, or by glutathione (reduced) supplementation (oral, intravenous, transdermal, or precursors such as N-acetyl cysteine [NAC]).

Tiglylglycine (TG) (Marker 18) is associated with both mitochondrial and/or genetic disorders. Toxic chemical exposure may be one of the most common causes of mitochondrial dysfunction. In mitochondrial disorders of the respiratory chain, TG values are usually more moderately increased than in the genetic disorders. In the medical literature, a normal value is less than 3.8 mmol/mol creatinine in children.

It is an intermediate product of the catabolism of isoleucine and ketone bodies. TG is found at variable high concentration in the urine of patients with 2-methylacetoacetyl-CoA thiolase or 2-methyl-3-hydroxybutyryl-CoA dehydrogenase (MHBD) deficiencies, which are inherited neurometabolic disorders affecting isoleucine catabolism. Biochemically, 2-methylacetoacetyl-CoA thiolase deficiency is characterized by intermittent ketoacidosis and urinary excretion of 2-methyl-acetoacetate (MAA), 2-methyl-3-hydroxybutyrate (MHB) and Tiglylglycine (TG), whereas in MHBD deficiency only MHB and Tiglylglycine accumulate. Typical clinical symptoms in both disorders include intermittent ketoacidotic episodes, seizures, and retardation. These diseases can be treated by switching to a diet low in protein and without isoleucine. In some cases, patients were asymptomatic until provoked by vaccinations or viral infection. In both disorders, biochemical abnormalities became more pronounced after a 100mg/kg oral isoleucine challenge. Tiglylglycine is also moderately elevated in short-chain acyl dehydrogenase (SCAD) deficiency, in propionyl CoA carboxylase, in methylmalonic aciduria, in the mitochondrial disorder Pearson syndrome (caused by mitochondrial DNA deletion), and in disorders of the respiratory chain in mitochondria.

Abnormal results may be confirmed by advanced mitochondrial DNA testing. Normal values of lactate and pyruvate do not rule out the presence of mitochondrial disorders; elevated TG should be considered a better marker of mitochondrial dysfunction than lactate or pyruvate levels. Extremely elevated values are likely due to genetic chromosomal mutations. Confirmation of genetic disorders requires DNA and/or enzyme testing at advanced biochemical genetics centers. Coenzyme Q-10 (300-600 mg), NAD 25 mg, L-carnitine and acetyl-L-carnitine (1000-2000 mg), riboflavin (40-80 mg), nicotinamide (40-80 mg), biotin (4-8 mg), and vitamin E (200-400 IU's) per day may improve mitochondrial dysfunction. Hyperbaric oxygen therapy (HBOT) may also be beneficial.

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