Biomedical and Nutritional Intervention Training Week 3

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http://biomedicalnutritionalintervention.com/training/

Week 3

Importance of Functional Medicine Testing

- Crucial role of testing and priority of each
- Recommended testing for families on limited budget
- Organic Acid Testing
- Peptide
- Heavy Metal
- Toxins
- Mycotoxin
- Glyphosate
- Food Intolerances
- Diagnostic Stool
- Hormone
- Thyroid
- Adrenal
- Lyme
- CBC looking at Vitamin D & cholesterol markers

Biomedical Balance of the Body

- Brain
- Sleep
- Bowels
- Eliminate any additional chemicals
- Electromagnetic field exposure/protection
- What you put on your body
- W put in your body
- are products
- from food

Recommended Testing on a Budget

- Organic Acid
- Glyphosate

Organic Acid Test

- Why the Organic Acid test?

It is an accurate assessment of what is going on metabolically in the human body. Organic acids are compounds with acidic properties and helps evaluate 75+ urinary metabolites that can be useful for discovering underlying causes of disorders and chronic illness.

Treatment can then be customized when you gain insight with the test results often leading to improved focus, concentration, energy, sleep and mental health issues, as well as reduced chronic pain and digestive problems and much more!

Who is the OAT test for?

Autism
ADD/ADHD
Autoimmune
Chronic fatigue
Digestive problems
Metabolic disorders
Mental health disorders Neurological disorders

Any individual with a chronic health condition where you suspect metabolic toxins may be a factor overlooked.

Personally, I recommend this test for everyone to get a baseline on how their body is functioning across many levels.

Major Sections of OAT Testing

Yeast and Fungal Markers:

- *Evaluates for invasive candida and mold toxicity*

Bacterial Markers:

- *Evaluates for dysbiosis*

Clostridia Bacteria Markers:

- *Evaluates for various clostridia bacteria toxins*

Oxalate Metabolites:

- *Evaluates for oxalic acid toxicity and endogenous oxalate production problems*.

Glycolytic and Mitochondrial Markers: - *Evaluates for mitochondrial dysfunction*

Major Sections of OAT Testing

Phenylalanine and Tyrosine Metabolites:

- *Evaluates for phenylalanine and tyrosine metabolism which can lead to dopamine and norepinephrine imbalances.*

Tryptophan Metabolites:

- *Evaluates for issues in tryptophan metabolism which contributes to serotonin imbalance and excess quinolinic acid production.*

Pyrimidine Metabolites:

- *Evaluates for uracil and thymine as indicators of folate imbalance.*

Ketone and Fatty Acid Oxidation:

- *Evaluates for fatty acid metabolism problems which can contribute to mitochondrial dysfunction.*

Nutritional Markers:

- *Evaluates for various nutrient imbalances*

Indicators of Detoxification:

- *Measures organic acids linked to glutathione status*

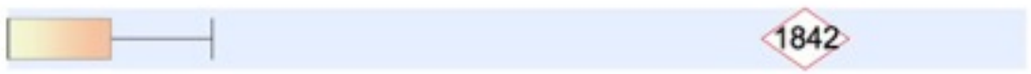
Major Sections of OAT Testing

Amino Acid Metabolites:

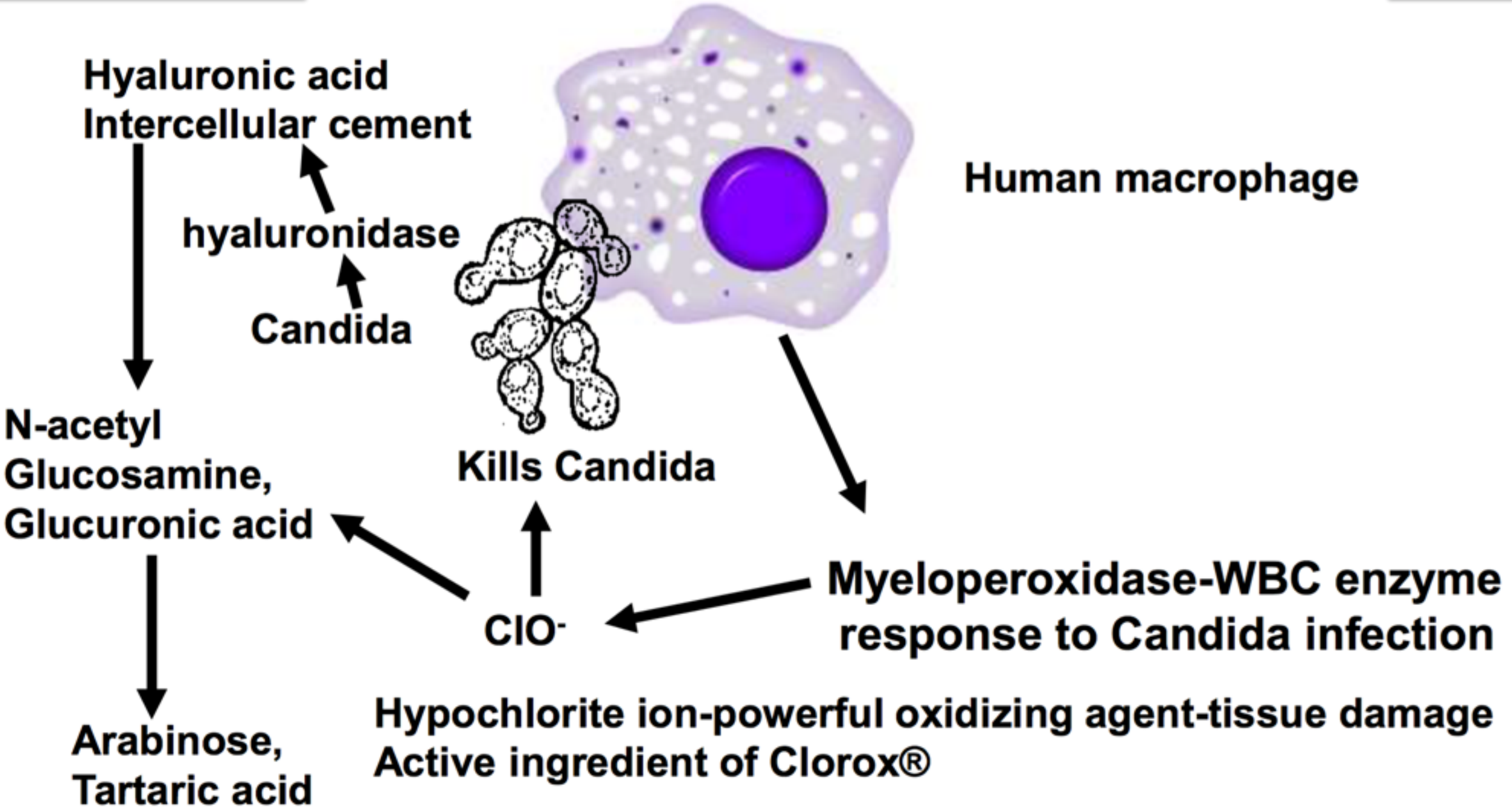
- *Measures organic acids linked to inborn errors of metabolism and other metabolic imbalances.*

Mineral Metabolism:

- *Measures phosphoric acid linked dietary consumption of phosphate (processed foods), as well as bone metabolism and vitamin D status.*

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Value	Reference Population - Males Under Age 13
Intestinal Microbial Overgrowth				
Yeast and Fungal Markers				
1 Citramalic	≤ 5.0		4.0	
2 5-Hydroxymethyl-2-furoic *(Aspergillus)*	≤ 28		5.2	
3 3-Oxoglutaric	≤ 0.46		0	
4 Furan-2,5-dicarboxylic *(Aspergillus)*	≤ 18		2.7	
5 Furancarbonylglycine *(Aspergillus)*	≤ 3.1		0.62	
6 Tartaric *(Aspergillus)*	≤ 6.5		1.1	
7 Arabinose	≤ 50	H	89	
8 Carboxycitric	≤ 25		10	
9 Tricarballylic *(Fusarium)*	≤ 1.3		0.39	
Bacterial Markers				
10 Hippuric	≤ 680	H	1,842	
11 2-Hydroxyphenylacetic	≤ 0.86	H	1.1	
12 4-Hydroxybenzoic	≤ 3.0	H	3.3	
13 4-Hydroxyhippuric	≤ 30	H	44	
14 DHPPA (Beneficial Bacteria)	≤ 0.59		0.33	
Clostridia Bacterial Markers				
15 4-Hydroxyphenylacetic *(C. difficile, C. stricklandii, C. lituseburense & others)*	2.0 - 32		27	
16 HPHPA *(C. sporogenes, C. caloritolerans, C. botulinum & others)*	≤ 220	H	227	
17 4-Cresol *(C. difficile)*	≤ 84		7.7	
18 3-Indoleacetic *(C. stricklandii, C. lituseburense, C. subterminale & others)*	0.60 - 14		2.9	

Production of arabinose, tartaric acids as indirect markers of invasive Candidiasis



Research article

Acute Schizophrenia

Increased urinary excretion of a 3-(3-hydroxyphenyl)-3-hydroxypropionic acid (HPHPA), an abnormal phenylalanine metabolite of *Clostridia* spp. in the gastrointestinal tract, in urine samples from patients with autism and schizophrenia

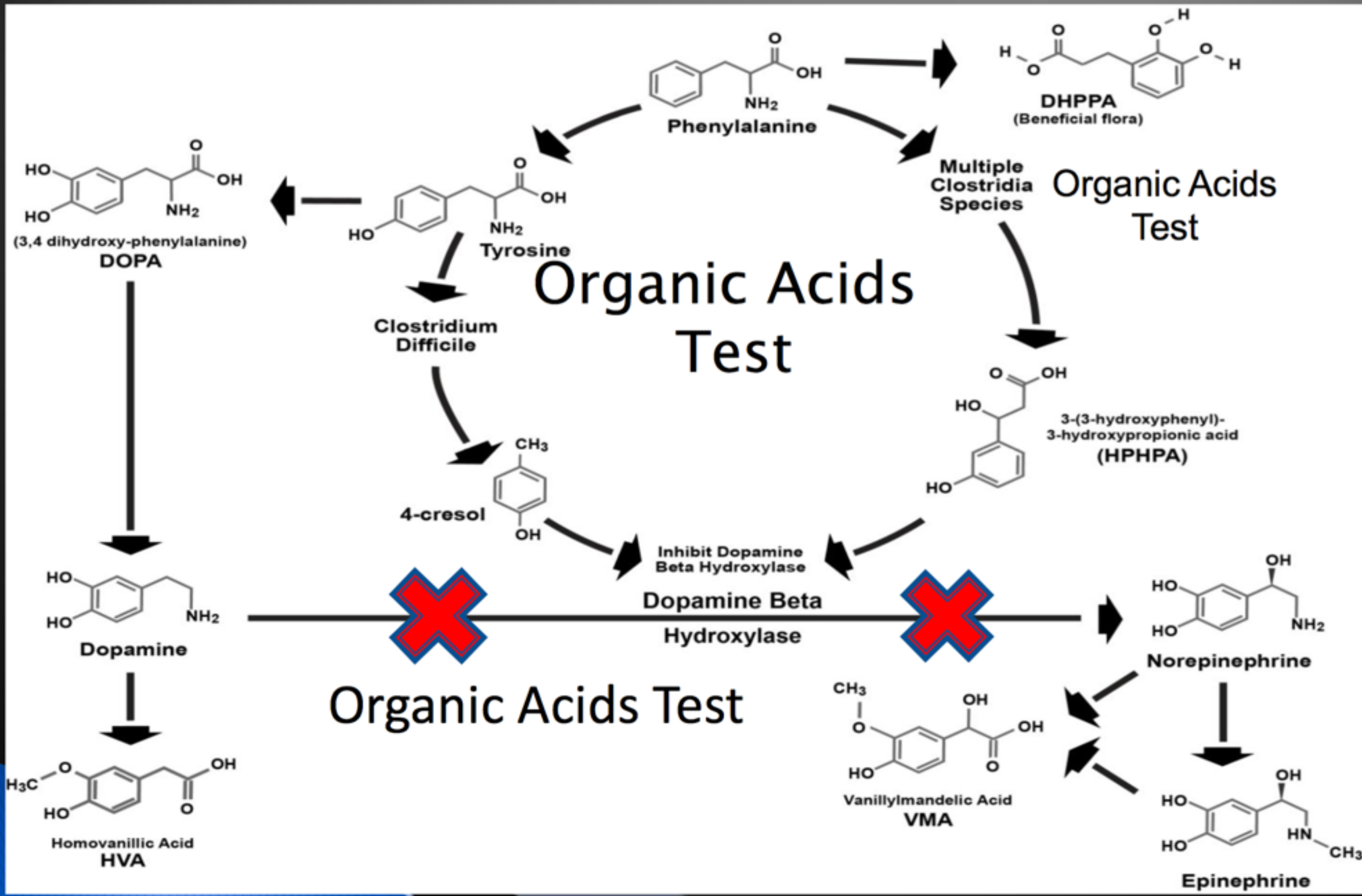
William Shaw

The Great Plains Laboratory, Inc., Lenexa, Kansas, USA

Nutritional Neuroscience 2010 Vol 13 No 3: 1-10

A compound identified as 3-(3-hydroxyphenyl)-3-hydroxypropionic acid (HPHPA) was found in higher concentrations in urine samples of children with autism compared to age and sex appropriate controls and in an adult with recurrent diarrhea due to *Clostridium difficile* infections. The highest value measured in urine samples was 7500 mmol/mol creatinine, a value 300 times the median normal adult value, in a patient with acute schizophrenia during an acute psychotic episode. The psychosis remitted after treatment with oral vancomycin with a concomitant marked decrease in HPHPA. The source of this compound appears to be multiple species of anaerobic bacteria of the *Clostridium* genus. The significance of this compound is that it is a probable metabolite of *m*-tyrosine (3-hydroxyphenylalanine), a tyrosine analog which depletes brain catecholamines and causes symptoms of autism (stereotypical behavior, hyperactivity, and hyper-

Effect Of Certain Intestinal Bacteria Toxins On Neurotransmitters



		(mmol/mol creatinine)			Value	
Oxalate Metabolites						
19	Glyceric	0.74	-	13	12	12
20	Glycolic	27	-	221	107	107
21	Oxalic	35	-	185	H 574	574
Glycolytic Cycle Metabolites						
22	Lactic	2.6	-	48	H 68	68
23	Pyruvic	0.32	-	8.8	6.3	6.3
Mitochondrial Markers - Krebs Cycle Metabolites						
24	Succinic		≤	23	11	11
25	Fumaric		≤	1.8	H 4.1	4.1
26	Malic		≤	2.3	H 4.7	4.7
27	2-Oxoglutaric		≤	96	65	65
28	Aconitic	9.8	-	39	39	39
29	Citric		≤	597	357	357
Mitochondrial Markers - Amino Acid Metabolites						
30	3-Methylglutaric	0.01	-	0.97	H 1.4	1.4
31	3-Hydroxyglutaric		≤	16	H 18	18
32	3-Methylglutaconic		≤	6.9	5.0	5.0
Neurotransmitter Metabolites						
Phenylalanine and Tyrosine Metabolites						
33	Homovanillic (HVA) *(dopamine)*	0.49	-	13	9.4	9.4
34	Vanillylmandelic (VMA) *(norepinephrine, epinephrine)*	0.72	-	6.4	3.5	3.5
35	HVA / VMA Ratio	0.23	-	2.8	2.7	2.7
36	Dihydroxyphenylacetic (DOPAC) *(dopamine)*	0.13	-	4.9	3.4	3.4
37	HVA/ DOPAC Ratio	0.37	-	3.3	2.8	2.8
Tryptophan Metabolites						
38	5-Hydroxyindoleacetic (5-HIAA) *(serotonin)*		≤	11	4.5	4.5
39	Quinolinic	0.48	-	8.8	3.7	3.7
40	Kynurenic		≤	4.2	1.5	1.5

Oxalate Metabolism

Collagen

Protein, Gelatin

Candida collagenase

Glycolate hydroxypyruvate

hydroxyprolin

Glycolate Oxidase (GO)

GRHPR
Glyoxylate reductase
Hydroxypyruvic reductase
Type II Hyperoxaluria

B-6

Glyoxylate Glycerate

Glycine

AGT
Alanine Glyoxylate Amino Transferase
Type I Hyperoxaluria

Protein

LDH
Lactate dehydrogenase

Yeast
Fungi

Ascorbate
Arabinose

diet

Oxalate

Oxalates diet

Ethylene glycol

Other Diseases/Disorders in Which Oxalate May Play a Role

- Arthritis
- Behavior problems in children
- Interstitial cystitis
- Joint pain
- Fibromyalgia
- Heart disease (*atherosclerosis, conduction issues)*
- Heavy metal toxicity
- Osteoporosis
- Thyroid problems

Quinolinic Acid Production

5-HT

Serotonin

Tryptophan

Protein

Tryptophan 2,3-dioxygenase (TDO)

Indoleamine 2,3-dioxygenase (IDO)

N-Formylkynurenine

Interferon, cortisol, and immune overstimulation increases tryptophan conversion to quinolinic acid. Niacin inhibits this conversion.

Kynurenine formamidase

Kynurenine aminotransferase

Kynurenic acid

L-Kynurenine

Kynureninase

Kynurenine 3-hydroxylase

3-Hydroxykynurenine

Anthranilic acid

Non-specific hydroxylation?

Kynureninase

3-Hydroxyanthranilic acid

Causes mitochondria dysfunction by reducing NAD synthesis

Increases neurotoxic Quinolinic acid

Phthalate

3-Hydroxyanthranilic acid oxidase

Non-enzymatic cyclization

Picolinic carboxylase

Picolinic acid

2-Amino-muconic semialdehyde

Non-enzymatic cyclization

Phosphoribosyl pyrophosphate

inhibits

Nicotinic acid mononucleotide

NAD Synthesis

Quinolinate phosphoribosyl transferase

Quinolinic acid

+

Chronic Stress Response

Potential Sources of Stress

Anger - Fear
Worry/Anxiety
Depression
Guilt
Overwork
Physical and Mental strain
Excessive exercise
Sleep deprivation
Light-cycle disruption
Late hours
Surgery
Trauma/Injury
Whiplash – Head injury
Inflammation
Pain
Temperature extremes
Toxic exposure
Infections
Chemicals - Heavy metals
Electromagnetic fields
Radiation
Geophysical
Malabsorption
Maldigestion
Illness
Low blood sugar - Poor diet
Nutritional deficiencies
Allergies
Foods
Mold – Pollens

Sympathetic System ↑↑

Stressor

Epinephrine

NE

HP

Reduced HP Sensitivity to Negative Feedback

NFL

ACTH ↑↑↑

Adrenal Cortex

Total Cortisol ↑↑

Free Cortisol ↑↑

Total DHEA(s)

Free DHEA(s)

Elevated Cortisol to DHEA Ratio* ↑

* Abnormal Ratio of Cortisol to DHEA indicates Pregnenolone Steal (Cortisol Escape)

Energy Production
- Insulin sensitivity↓
- Glucose utilization↓
- Blood sugar↑
- Gluconeogenesis↑

Other Influences
- Osteoporosis (bone loss) ↑
- Fat accumulation (waist)↑
- Protein breakdown↑
- Salt & water retention↑

Immune Activity
- Secretory IgA↓
- Antigen penetration ↑
- Circulating IgG↑
- NK cell activity↓
- Interleukin 2↓
- T-Lymphocytes↓

Clinical Conditions

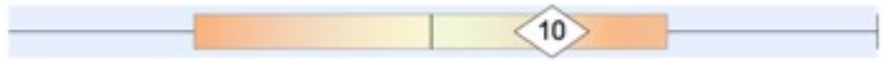
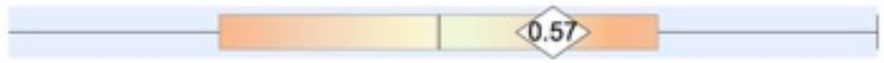
Chronic viral infections (EBV,CMV,Herpes I-II,etc.)
Increased infections
Yeast overgrowth
Allergies
Fatigue
Headaches
Autoimmune disease
Cancer
Cardiovascular disease
Insomnia
Hypoglycemia
Hunger
PMS
Depression
Irritable bowel
Digestive problems
ADD/ADHD

KEY

Association
Stimulus
Outcome
Inhibition

HP = Hypothalamus - Pituitary
NFL = Negative Feedback Loop
NE = Norepinephrine

Pyrimidine Metabolites - Folate Metabolism

41	Uracil		≤	16		10	
42	Thymine		≤	0.91		0.57	

Ketone and Fatty Acid Oxidation

43	3-Hydroxybutyric		≤	4.8	H	1,196	
44	Acetoacetic		≤	10	H	704	
45	Ethylmalonic	0.06	-	4.8	H	5.6	
46	Methylsuccinic		≤	4.0	H	5.3	
47	Adipic	0.19	-	6.5	H	7.3	
48	Suberic		≤	7.0	H	7.5	
49	Sebacic		≤	0.61		0.38	

Nutritional Markers

Vitamin B12

50	Methylmalonic ✸		≤	5.2		1.6	

Vitamin B6

51	Pyridoxic (B6)		≤	53		4.0	

Vitamin B5

52	Pantothenic (B5)		≤	14		5.2	

Vitamin B2 (Riboflavin)

53	Glutaric ✸		≤	1.4	H	2.2	

Vitamin C

54	Ascorbic	10	-	200	L	0.49	

Vitamin Q10 (CoQ10)

55	3-Hydroxy-3-methylglutaric ✸		≤	88	H	101	

Glutathione Precursor and Chelating Agent

56	N-Acetylcysteine (NAC)		≤	0.34		0	

Biotin (Vitamin H)

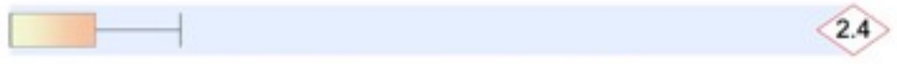
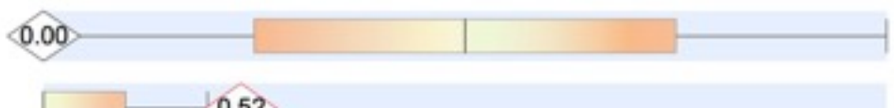
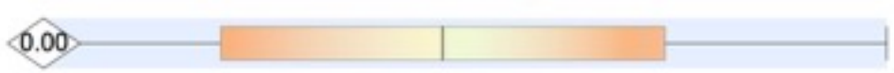
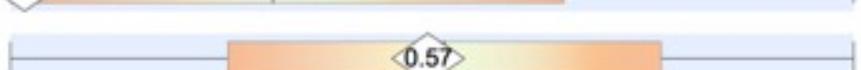
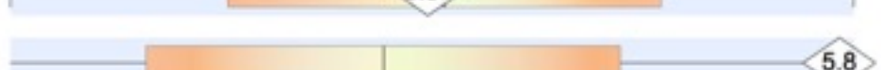
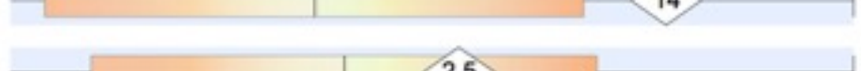
57	Methylcitric ✸		≤	5.7		1.3	

Glutathione						
58	Pyroglutamic ✱	13	- 62		57	
Methylation, Toxic exposure						
59	2-Hydroxybutyric ✱✱	0.19	- 2.0	H	9.0	
Ammonia Excess						
60	Orotic	0.04	- 0.80		0.71	
Aspartame, salicylates, or GI bacteria						
61	2-Hydroxyhippuric		≤ 1.2		0.88	

✱ ***A high value for this marker may indicate a Glutathione deficiency.***

✱✱ ***High values may indicate methylation defects and/or toxic exposures.***

Amino Acid Metabolites

62	2-Hydroxyisovaleric		≤ 0.55	H	2.4	
63	2-Oxoisovaleric		≤ 2.5		0	
64	3-Methyl-2-oxovaleric		≤ 1.1		0	
65	2-Hydroxyisocaproic		≤ 0.68		0	
66	2-Oxoisocaproic		≤ 0.46	H	0.52	
67	2-Oxo-4-methiolbutyric		≤ 0.33		0	
68	Mandelic		≤ 0.30	H	0.63	
69	Phenyllactic		≤ 0.19		0	
70	Phenylpyruvic		≤ 4.0		1.3	
71	Homogentisic		≤ 0.61		0.01	
72	4-Hydroxyphenyllactic	0.05	- 1.1		0.57	
73	N-Acetylaspartic		≤ 5.9		5.8	
74	Malonic		≤ 18		14	
75	4-Hydroxybutyric		≤ 4.7		2.5	

Mineral Metabolism

76	Phosphoric	1,000	- 7,300		3,288	

Krebs Cycle: Steps and regulation of this essential part of cellular physiology

Step 1: Acetyl CoA (two carbon molecule) joins with oxaloacetate (4 carbon molecule) to form citrate (6 carbon molecule).
Step 2: Citrate is converted to isocitrate (an isomer of citrate)
Step 3: Isocitrate is oxidised to alpha-ketoglutarate (a five carbon molecule) which results in the release of carbon dioxide. One NADH molecule is formed. The enzyme responsible for catalysing this step is **isocitrate dehydrogenase.** This is a rate limiting step as isocitrate dehydrogenase is an allosterically controlled enzyme.
Step 4: Alpha-ketoglutarate is oxidised to form a 4 carbon molecule. This binds to coenzyme A forming succinyl CoA. A second molecule of NADH is produced, alongside a second molecule of carbon dioxide.
Step 5: Succinyl CoA is then converted to succinate (4 carbon molecule) and one GTP molecule is produced.
Step 6: Succinate is converted into fumarate (4 carbon molecule) and a molecule of $FADH_2$ is produced.
Step 7: Fumarate is converted to malate (another 4 carbon molecule).
Step 8: Malate is then converted into oxaloacetate. The third molecule of NADH is produced.

Krebs Cycle: Steps and regulation of this essential part of cellular physiology

The primary role of the Krebs cycle is production of NADH and $FADH_2$, it also produces molecules that supply various biosynthetic processes. These enter or exit the cycle at various points depending on demand. For example, alpha-ketoglutarate can leave the cycle to be converted into amino acids, and **succinate** can be converted to haem.

Net Output

Each cycle produces:

- Two molecules of carbon dioxide.
- Three molecules of NADH.
- Three hydrogen ions.
- One molecule of $FADH_2$
- One molecule of GTP.

Each molecule of glucose produces two molecules of pyruvate, which in turn produce two molecules of acetyl-coA. Therefore, each molecule of glucose produces double this.

The TCA Cycle is regulated in a variety of ways including:

- **Metabolites:** The products of the cycle provide negative feedback on the enzymes that catalyse it. For example, NADH inhibits the majority of the enzymes found in the TCA cycle.
- **Citrate:** Inhibits phosphofructokinase, a key enzyme in glycolysis. This reduces the rate of production of pyruvate and therefore of acetyl-coA.
- **Calcium:** Calcium accelerates the TCA cycle by stimulating the link reaction.

Note: Prior to the TCA cycle, glycolysis has occurred. This has generated molecules including pyruvate, ATP and NADH.

Pyruvate is then **decarboxylated** to form acetyl-coA by the **pyruvate decarboxylase complex**. Acetyl-coA is the intermediate that enters the TCA cycle.

Krebs Cycle: Steps and regulation of this essential part of cellular physiology

Note: Prior to the TCA cycle, **glycolysis** has occurred. This has generated molecules including **pyruvate, ATP and NADH**. Pyruvate is then decarboxylated to form acetyl-coA by the pyruvate decarboxylase complex. Acetyl-coA is the intermediate that enters the TCA cycle.

Glycolysis can be considered as a two part process.

Energy is consumed to generate high energy intermediates, which then go on to release their energy during the second phase.

Energy investment phase – requires two ATP molecules to produce high energy intermediates.

Energy pay out phase – The intermediate is metabolized, producing four ATP molecules and two NADH molecules.

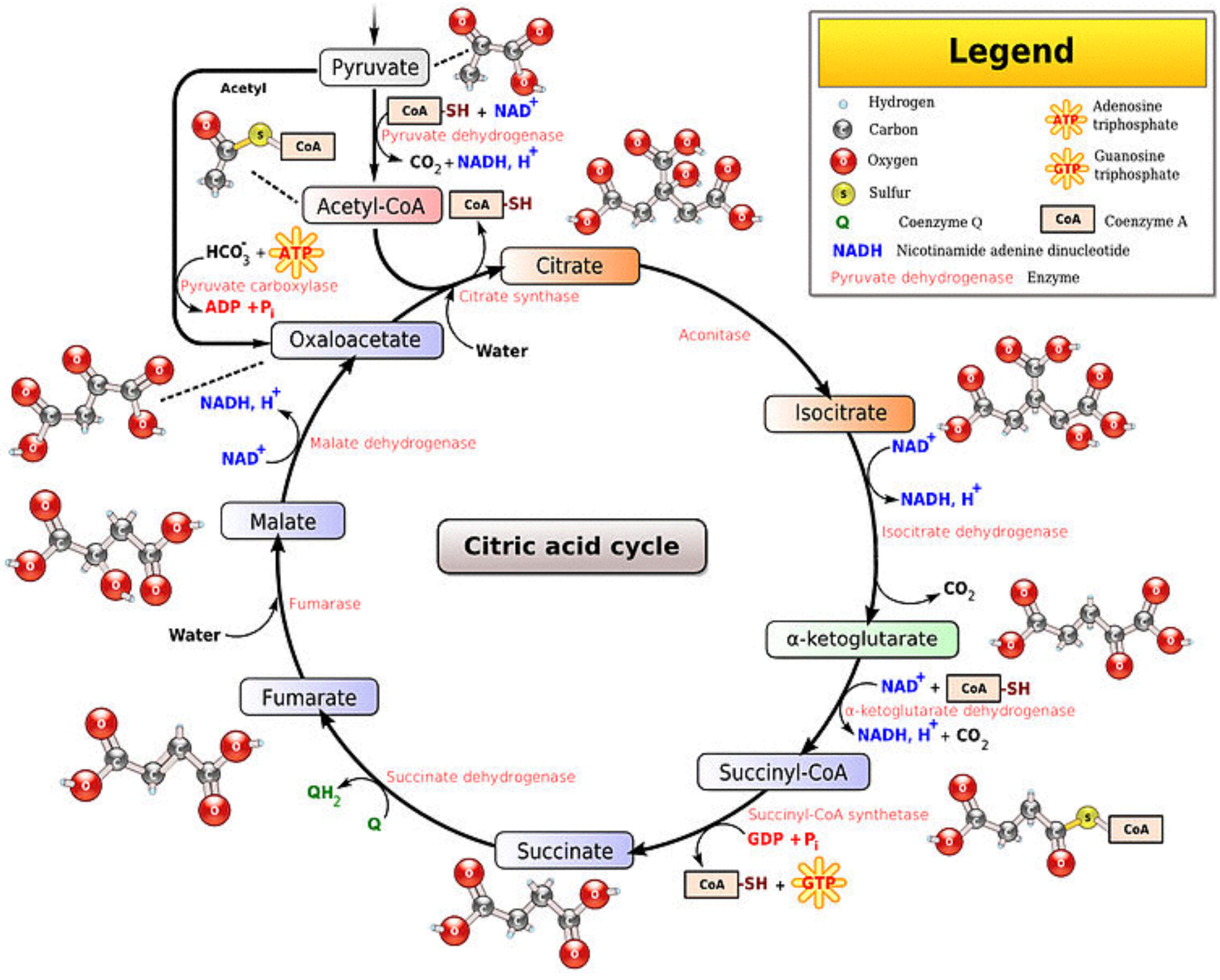
Phosphenolpyruvate is converted into pyruvate by pyruvate kinase, which yields our second molecule of ATP. This is irreversible, and is therefore another key regulatory step.

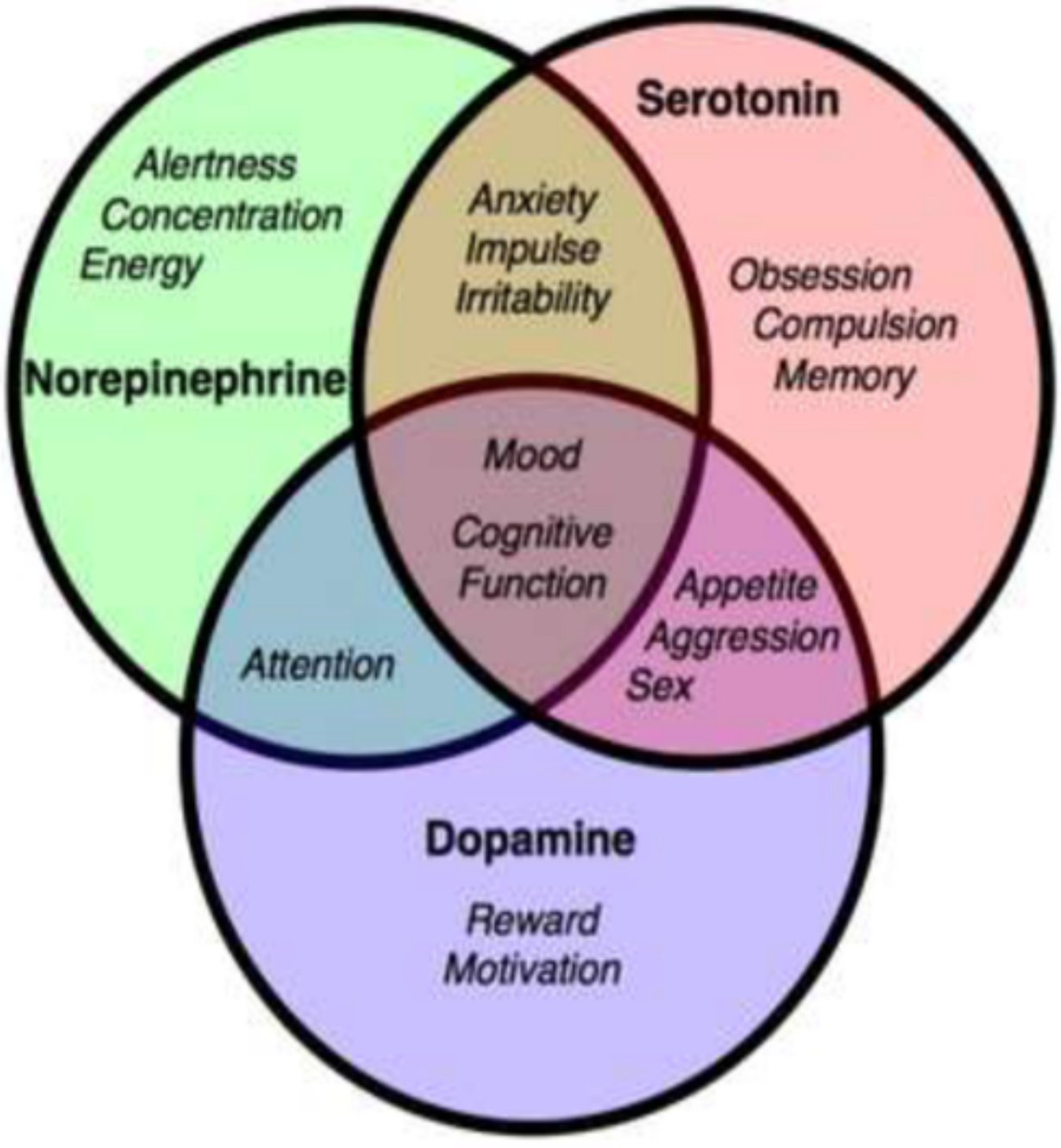
Pyruvate is a versatile molecule which feeds into numerous pathways.

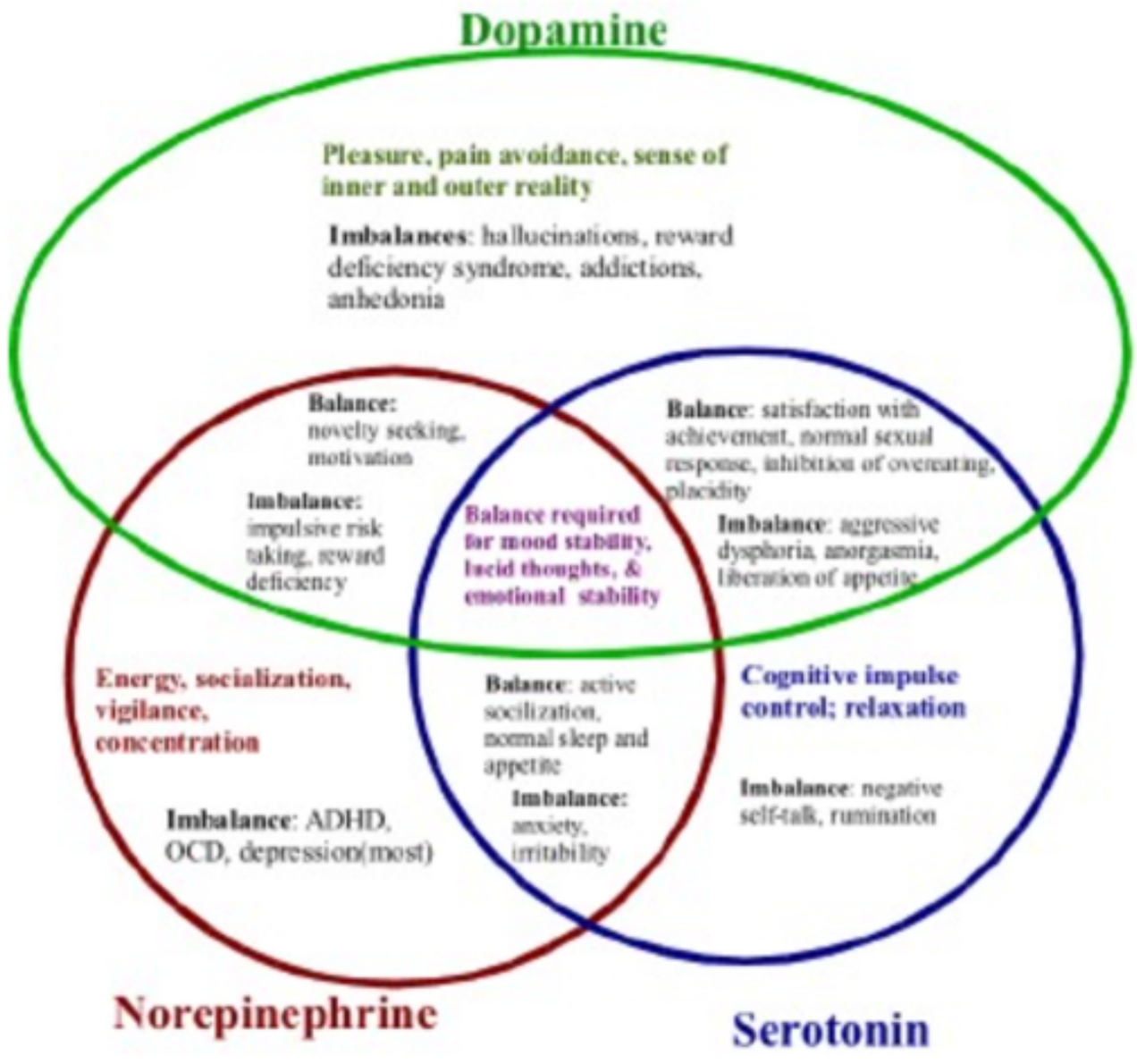
After glycolysis, it can be converted to acetyl-CoA, which has numerous metabolic destinations, including the Krebs cycle. It can also be converted into lactate, which enters the Cori cycle in absence of mitochondria or oxygen.

Other Important Pathway Interactions

DHAP, an intermediate of glycolysis, can be converted to glycerol phosphate in the liver and adipose tissue. This can feed into biosynthetic pathways, such as triglyceride and phospholipid biosynthesis, which also recycles NADH. 1,3-BPG can also be converted to 2,3-BPG in red blood cells to alter the affinity of haemoglobin for O2.


Legend
Hydrogen
Carbon
Oxygen
Sulfur
Q Coenzyme Q
ATP Adenosine triphosphate
GTP Guanosine triphosphate
CoA Coenzyme A
NADH Nicotinamide adenine dinucleotide
Pyruvate dehydrogenase Enzyme
Pyruvate
Acetyl
CoA
CoA-SH + NAD+
Pyruvate dehydrogenase
CO2 + NADH, H+
Acetyl-CoA
CoA-SH
HCO3- + ATP
Pyruvate carboxylase
ADP + Pi
Oxaloacetate
Citrate
Citrate synthase
Water
Aconitase
Isocitrate
NAD+
NADH, H+
Isocitrate dehydrogenase
CO2
α-ketoglutarate
NAD+ + CoA-SH
α-ketoglutarate dehydrogenase
NADH, H+ + CO2
Succinyl-CoA
Succinyl-CoA synthetase
GDP + Pi
CoA-SH + GTP
Succinate
Succinate dehydrogenase
QH2
Q
Fumarate
Fumarase
Water
Malate
NADH, H+
NAD+
Malate dehydrogenase
Citric acid cycle





Modified from Katz, N.B. 2005. Neurotransmitters the bridges of the brain. Institute for Natural Resources. Health Update. Home-Study #1700, 27pp.

Gluten and Casein Peptide Test

- this test is not currently available but may in the future
- Casomorphinis one of the peptides produced from casein, a major protein in all mammalian milk. Digestion of gluten in wheat also produces peptides of similar size, one of which is gliadorphin. Because of structural similarity, these peptides are presumed to a ect the brain similarly.

Heavy Metal Hair Test

Heavy metals toxicity caused by increasing levels of pollution and use of chemicals in industry is a growing threat to our health and development of our children. High levels of toxic metals deposited in body tissues and subsequently in the brain, may cause significant developmental and neurological damage, including autistic symptoms, depression, increased irritability, anxiety, insomnia, hallucinations, memory loss, aggression and many other disorders.

Some possible sources of heavy metals poisoning include chemical products, fertilizers, industrial paint, building materials, fish that is high in mercury, silver dental fillings in biological moms during pregnancy, and preservatives in vaccines, nasal sprays, and many more.

Lead may be found in dirt near roads and is commonly present in paint from older houses. Children with pica or who eat paint chips may develop toxic lead levels. **Cadmium** is used in batteries, steel-belted tires, and plastics. The clear plastics used to cover foods may be a source of cadmium and it is also found in cigarette smoke. **Mercury** is used in batteries, dental amalgam, vacuum pumps, valves, and seals. High amounts of mercury from the dental fillings of the cremated deceased have caused contamination of the surrounding neighborhoods. **Arsenic** is high in seafood and may also be found in animals fed arsenic in their feed. Arsenic is also present in wood preservatives, fungicides, herbicides, corrosion inhibitors, and in lead and copper alloys.

A Metals Hair Test is ideal for checking current exposure to toxic metals. Hair provides important information that can assist the practitioner with an early diagnosis of physiological disorders associated with aberrations in essential and toxic element metabolism.

Heavy Metal Tests

WHY CHOOSE A METALS HAIR TEST

Extensive research established that scalp hair element levels are related to human systemic levels. Many researchers consider hair as the tissue of choice for toxic and several nutrient elements. Unlike blood, hair element levels are not regulated by homeostatic mechanisms. Hair element analysis provides important information which, in conjunction with symptoms and other laboratory values, can assist with an early diagnosis of physiological disorders. As protein is synthesized in the hair follicle, elements are incorporated permanently into the hair with no further exchange with other tissues. Scalp hair is easy to sample, and because it grows an average of one to two cm per month, it contains a "temporal record" of element metabolism and exposure to toxic elements.

Nutrient elements including magnesium, chromium, zinc, copper and selenium are obligatory co-factors for hundreds of important enzymes and also are essential for the normal functions of vitamins. The levels of these elements in hair are correlated with levels in organs and other tissues.

Toxic elements may be 200-300 times more highly concentrated in hair than in blood or urine. Hair is the tissue of choice for detection of recent exposure to elements such as arsenic, aluminum, cadmium, lead, antimony, and mercury. CDC acknowledges the value of hair mercury levels as a maternal and infant marker for exposure to neurotoxic methylmercury from fish.

THE BENEFITS OF METALS TESTING

- Determine if metal toxicity or mineral deficiency is contributing to the disorder
- Monitor the effects of chelation (elimination of heavy metals from the body)
- Identify if supplementation of important minerals may bring about significant improvements

SPECIMEN REQUIREMENTS

Hair (source head): 0.25 grams (approximately 1 tablespoon) of hair is minimum. 1 gram is preferred.

Heavy Metal Tests

A Metals Hair Test is ideal for checking current exposure to toxic metals. Hair provides important information that can assist the practitioner with an early diagnosis of physiological disorders associated with aberrations in essential and toxic element metabolism.

WHY CHOOSE A METALS HAIR TEST

Extensive research established that scalp hair element levels are related to human systemic levels. The strength of this relationship varies for specific elements, and many researchers consider hair as the tissue of choice for toxic and several nutrient elements. Unlike blood, hair element levels are not regulated by homeostatic mechanisms. Thus, deviations in hair element levels often appear prior to overt symptoms and can thereby be a valuable preliminary tool for predicting the development of physiological abnormalities.

Hair element analysis provides important information which, in conjunction with symptoms and other laboratory values, can assist the physician with an early diagnosis of physiological disorders associated with aberrations in essential and toxic element metabolism.
As protein is synthesized in the hair follicle, elements are incorporated permanently into the hair with no further exchange with other tissues. Scalp hair is easy to sample, and because it grows an average of one to two cm per month, it contains a “temporal record” of element metabolism and exposure to toxic elements.
Nutrient elements including magnesium, chromium, zinc, copper and selenium are obligatory co-factors for hundreds of important enzymes and also are essential for the normal functions of vitamins. The levels of these elements in hair are correlated with levels in organs and other tissues.
Toxic elements may be 200-300 times more highly concentrated in hair than in blood or urine. Therefore, hair is the tissue of choice for detection of recent exposure to elements such as arsenic, aluminum, cadmium, lead, antimony, and mercury. The CDC acknowledges the value of hair mercury levels as a maternal and infant marker for exposure to neurotoxic methylmercury from fish.
Through recent vast improvements in technology, instrumentation, and application of scientific protocols, hair element analysis has become a valuable tool in providing dependable and useful data for physicians and their patients. The U.S. Environmental Protection agency stated in a recent report that “...if hair samples are properly collected and cleaned, and analyzed by the best analytic methods, using standards and blanks as required, in a clean and reliable laboratory by experienced personnel, the data are reliable.” (U.S.E.P.A. 600/4-79-049)
Hair element analysis is a valuable and inexpensive screen for physiological excess, deficiency or maldistribution of elements. It should not be considered a stand-alone diagnostic test for essential element function, and should be used in conjunction with patient symptoms and other laboratory tests.

THE BENEFITS OF METALS TESTING

- Determine if metal toxicity or mineral deficiency is contributing to the disorder
- Monitor the effects of chelation (elimination of heavy metals from the body)
- Identify if supplementation of important minerals may bring about significant improvements

SPECIMEN REQUIREMENTS

Hair (source head or pubic): 0.25 grams (approximately 1 tablespoon) of hair is the absolute minimum. 1 gram is preferred.

TOXIC METALS		RESULT µg/g	REFERENCE INTERVAL	PERCENTILE 68th 95th
Aluminum	(Al)	1.5	< 7.0	
Antimony	(Sb)	< 0.01	< 0.066	
Arsenic	(As)	0.027	< 0.080	
Barium	(Ba)	0.29	< 1.0	
Beryllium	(Be)	< 0.01	< 0.020	
Bismuth	(Bi)	< 0.002	< 2.0	
Cadmium	(Cd)	0.021	< 0.065	
Lead	(Pb)	0.82	< 0.80	
Mercury	(Hg)	2.8	< 0.80	
Platinum	(Pt)	< 0.003	< 0.005	
Thallium	(Tl)	< 0.001	< 0.002	
Thorium	(Th)	< 0.001	< 0.002	
Uranium	(U)	0.036	< 0.060	
Nickel	(Ni)	0.31	< 0.20	
Silver	(Ag)	0.08	< 0.08	
Tin	(Sn)	0.05	< 0.30	
Titanium	(Ti)	0.21	< 0.60	
Total Toxic Representation				

SAMPLE

ESSENTIAL AND OTHER ELEMENTS		RESULT µg/g	REFERENCE INTERVAL	PERCENTILE 2.5th 16th 50th 84th 97.5th
Calcium	(Ca)	554	200- 750	
Magnesium	(Mg)	31	25- 75	
Sodium	(Na)	7	20- 180	
Potassium	(K)	3	9- 80	
Copper	(Cu)	64	11- 30	
Zinc	(Zn)	200	130- 200	
Manganese	(Mn)	0.14	0.08- 0.50	
Chromium	(Cr)	0.37	0.40- 0.70	
Vanadium	(V)	0.018	0.018- 0.065	
Molybdenum	(Mo)	0.010	0.025- 0.060	
Boron	(B)	0.82	0.40- 3.0	
Iodine	(I)	0.54	0.25- 1.8	
Lithium	(Li)	< 0.004	0.007- 0.020	
Phosphorus	(P)	126	150- 220	
Selenium	(Se)	0.75	0.70- 1.2	
Strontium	(Sr)	1.4	0.30- 3.5	
Sulfur	(S)	48100	44000- 50000	
Cobalt	(Co)	0.003	0.004- 0.020	
Iron	(Fe)	5.0	7.0- 16	
Germanium	(Ge)	0.033	0.030- 0.040	
Rubidium	(Rb)	< 0.003	0.011- 0.12	
Zirconium	(Zr)	0.035	0.020- 0.44	

REPORT

SPECIMEN DATA	RATIOS

Environmental Toxins Test

Every day, we are exposed to hundreds of toxic chemicals through products like pharmaceuticals, pesticides, packaged foods, household products, and environmental pollution. As we have become more exposed to chemical-laden products and to toxic chemicals in food, air, and water, we have been confronted with an accelerating rate of chronic illnesses like cancer, heart disease, chronic fatigue syndrome, chemical sensitivity, autism spectrum disorders, ADD/AD(H)D, autoimmune disorders, Parkinson's disease, and Alzheimer's disease.

Because exposure to environmental pollutants has been linked to many chronic diseases, a toxic non-metal chemical profile that screens for the presence of 173 different toxic chemicals including organophosphate pesticides, phthalates, benzene, xylene, vinyl chloride, pyrethroid insecticides, acrylamide, perchlorate, diphenyl phosphate, ethylene oxide, acrylonitrile, and more.

This profile also includes Tiglylglycine (TG), a marker for mitochondrial disorders resulting from mutations of mitochondrial DNA. These mutations can be caused by exposure to toxic chemicals, infections, inflammation, and nutritional deficiencies.

Environmental Toxins Test

Phthalates
The most widespread group of toxic chemicals found in our environment. Phthalates are found in after shave lotions, aspirin, cosmetics, detergents, foods microwaved with plastic covers, oral pharmaceutical drugs, intravenous products prepared in plastic bags, hair sprays, insecticides, insect repellents, nail polish, nail polish remover, skin care products, adhesives, explosives, lacquer, janitorial products, perfumes, paper coatings, printing inks, safety glass, 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.

Vinyl Chloride
Vinyl chloride is an intermediate in the synthesis of several commercial chemicals, including polyvinyl chloride (PVC). Exposure to vinyl chloride may cause central nervous system depression, nausea, headache, dizziness, liver damage, degenerative bone changes, thrombocytopenia, enlargement of the spleen, and death.

Benzene
Benzene is an organic solvent that is widespread in the environment. Benzene is a by-product of all sources of combustion, including cigarette smoke, and is released by outgassing from synthetic materials, and is a pollutant released by numerous industrial processes. 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.

Pyrethrins
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.

Xylenes
Xylenes (dimethylbenzenes) are solvents 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 xylene levels may be due to the use of certain perfumes and insect repellents. High exposures to xylene create an increase in oxidative stress, causing symptoms such as nausea, vomiting, dizziness, central nervous system depression, and death.

Styrene
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,*** tiredness and nausea, and irritates the mucous membranes of the eyes, nose, and throat.

Environmental Toxins Test

Organophosphates

Organophosphates are one of the most toxic groups of substances used throughout the world. They are often used as biochemical weapons and terrorist agents, but are most commonly used in pesticide formulations. Organophospates 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.*** A study done in the San Francisco Bay area found that in California agricultural areas, children born to mothers living within 500 meters of fields where organochlorine pesticides were used were more than 6 times more likely to develop autism than children whose mothers did not live near such fields. ASD risk increased with the poundage of organochlorines applied and decreased with distance from field sites. Maternal organophosphate exposure has been associated with various adverse outcomes including having shorter pregnancies and children with impaired reflexes.

MTBE and ETBE

MTBE and ETBE are gasoline additives used to improve octane ratings. Exposure to these compounds is most likely due to ***groundwater contamination***, and 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. Since the metabolites of these compounds are the same, ETBE may be similarly toxic.

2, 4-Dicholorophenoxyacetic (2,4-D)

A very common herbicide that was a part of Agent Orange, used by the US during Vietnam War to increase visibility for war planes, by destroying plant undergrowth and crops. 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.

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***.

Acrylamide

Acrylamide can polymerize to form polyacrylamide. Polyacrylamide is used in many industrial processes such as plastics, food packaging, cosmetics, nail polish, 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, French fries, and many others such as asparagus, potatoes, legumes, nuts, seeds, beef, eggs, and fish. Asparagine, which is found in these foods can produce acrylamide when cooked at high temperature in the presence of sugars.*** High levels of acrylamide can elevate a patient's risk of cancer. In addition, acrylamide is known to cause ***neurological damage.***

Environmental Toxins Test

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 perchlorate. Perchlorate can disrupt the thyroid's ability to produce hormones. The EPA has also labeled perchlorate a likely human carcinogen. If this marker is elevated then use a reverse osmosis water treatment system to remove the chemical from water supply.

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 **r*****ubber playgrounds and athletic fields is quite troubling because children and athletes may be exposed to toxic chemicals this way.***

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.

1-Bromopropane (1-BP)
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.

Ethylene Oxide
Ethylene oxide is used in many different industries including agrochemicals detergents, pharmaceuticals, and personal care products. Ethylene oxide is also used as a sterilizing agent 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. Caution is needed with ethylene oxide because it is odorless at toxic levels.

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.

Acrolein
Acrolein is commonly used as an herbicide to control submersed and floating weeds and algae in irrigation canals. Humans are exposed to acrolein via oral ***(fried foods, alcoholic beverages, and water),*** respiratory (***cigarette smoke and automobile exhaust)***, and dermal routes. In addition, there is also endogenous generation (metabolism and lipid peroxidation) of acrolein. Acrolein has been suggested to play a role in several disease states including spinal cord injury, multiple sclerosis, Alzheimer's disease, cardiovascular disease, diabetes mellitus, and neuro-, hepato-, and nephro-toxicity. On the cellular level, acrolein exposure has diverse toxic effects, including DNA and protein adduction, oxidative stress, ***mitochondrial disruption***, membrane damage, and immune dysfunction.

Environmental Toxins Test

MITOCHONDRIAL DISORDERS

Testing for Tiglylglycine (TG), since it's one of the most specific markers for mitochondrial disorders resulting from mutations of mitochondrial DNA. These mutations can result from exposure to toxic chemicals, infections, inflammation, and nutritional deficiencies. Mitochondria are important in all cells in the body, but are especially important to organs that utilize large amounts of energy, such as the muscles, heart, and brain. The mitochondria also have several other important functions in the cell, including steroid synthesis, calcium regulation, free radical production, and the induction of apoptosis or programmed cell death, all of which are involved in the pathogenesis of numerous disorders. The marker 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.

Environmental Toxins Test

2-Methylhippuric Acid (2MHA), 3-Methylhippuric Acid (3MHA), 4-Methylhippuric Acid (4MHA)
These are metabolites of xylenes, solvents found in paints, lacquers, cleaning agents, pesticides, and gasoline. Exposure to xylenes generates methylhippuric acid isomers.

N-acetyl phenyl cysteine (NAP)
NAP is a metabolite of benzene. Benzene is a solvent that is widespread in the environment. It is found in cigarette smoke and gasoline, and is a byproduct of all types of combustion, including motor vehicle exhaust.

Phenylglyoxylic Acid (PGO)
Exposure to environmental styrene may slightly increase phenylglyoxylic and mandelic acid. Reduce exposure by eliminating the use of plastic and styrofoam containers for cooking, reheating, eating or drinking. Elimination of styrene can be accelerated by supplementing with ***glutathione and N-acetyl cysteine (NAC).***

2-Hydroxyisobutyric Acid (2HIB)
2-Hydroxyisobutyric acid is formed endogenously as a product of branched-chain amino acid degradation and ketogenesis. This compound is also the major metabolite of gasoline octane enhancers such as MTBE and ETBE. Elevated levels indicate environmental exposure and very high values have been reported in genetic disorders.

Monoethyl Phthalate (MEP)
MEP from diethyl phthalate is the most abundant phthalate metabolite found in urine. Diethyl phthalate is used in plastic products. Elevated values indicate exposure from various possible sources. Elimination of phthalates may be accelerated by ***sauna treatment.***

Dimethylphosphate (DMP) & Diethylphosphate (DEP)
DMP and DEP are major metabolites of many organophosphate pesticides. Reduce exposure by eating organic foods and avoiding use of pesticides in your home or garden. Living near agricultural areas or golf courses and areas regularly sprayed with pesticides will increase exposure. Elimination of organophosphates can be accelerated by s***auna treatment.***

3-Phenoxybenzoic Acid (3PBA)
3-Phenoxybenzoic acid is a metabolite of pyrethroid insecticides. Elimination can be accelerated by ***sauna treatment.***

Environmental Toxins Test

2,4-Dichlorophenoxyacetic Acid (2,4-D)
2,4-D was an ingredient in Agent Orange, and is most commonly used in agriculture of genetically modified foods, and as a weed killer for lawns. Reduce exposure by ***eating organic foods and avoiding use of pesticides in your home or garden.***

Tiglylglycine (TG)
TG is a marker for mitochondrial dysfunction. Mutations of mitochondria DNA may result from exposure to toxic chemicals, infections, inflammation, and nutritional deficiencies.

N-acetyl-S-(2-carbamoylethyl)-cysteine
NAE is a metabolite of acrylamide, which is detoxified through a two-step process. First acrylamide is metabolized by the cytochrome P450s. Second it is conjugated to glutathione in order to make it more water soluble. Acrylamide is used in many industrial processes such as plastics, food packaging, cosmetics, nail polish, dyes, and treatment of drinking water. High levels of acrylamide can elevate a risk of cancer and cause neurological damage. ***Supplementation with glutathione*** can assist in the elimination of this compound.

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.***

Perchlorate
Perchlorate is used in the production of rocket fuel, missiles, fireworks, flares, explosives, fertilizers, and bleach. Studies show that perchlorate is often found to contaminate water supplies and food sources. It can disrupt the thyroid's ability to produce hormones. The EPA has also labeled perchlorate a likely human carcinogen. ***Use a reverse osmosis water treatment system to remove perchlorate.***

Environmental Toxins Test

N-Acetyl (3,4-Dihydroxybutyl) Cysteine (NABD)
NADB is a metabolite of 1,3 butadiene, which is evident of exposure to synthetic rubber such as tires. 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.***

N-Acetyl (2,Hydroxypropl) Cysteine (NAHP)
NAHP is a metabolite of propylene oxide which is used in the production of plastics and as a fumigant. It is also used in the preparation of lubricants, surfactants, and oil demulsifiers and as a food additive, an herbicide, a microbicide, an insecticide, a fungicide, and a miticide. Propylene oxide is a probable human carcinogen.

N-Acetyl (Propyl) Cysteine (NAPR)
NAPR is a metabolite of 1-bromopropane. Chronic exposure can lead to ***decreased cognitive function and impairment of the central nervous system.*** Acute exposure can lead to headaches.

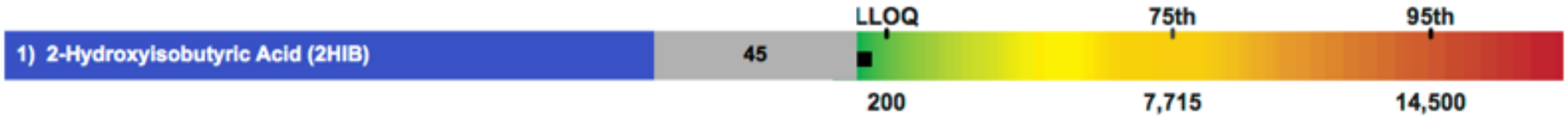
2-Hydroxyethyl Mercapturic Acid (HEMA)
HEMA is a metabolite of ethylene oxide, which is used in the production of agrochemicals, detergents, pharmaceuticals, and personal care products. Chronic exposure to ethylene oxide has been determined to be mutagenic to humans. HEMA is also a metabolite of vinyl chloride and halopropane, which are used in many commercial chemical processes such as ***foam glueing,*** dry cleaning, and in the production of solvents. Supplementation with glutathione should assist in the detoxification process of these chemicals.

N-Acetyl (2-Cyanoethyl) Cysteine (NACE)
NACE is a metabolite of acrylonitrile, which is used in the production of acrylic fibers, resins, and rubber. Acrylonitrile is metabolized by the cytochrome P450s and then conjugated to glutathione. Supplementation with glutathione should assist in the detoxification of acrylonitrile.

N-acetyl-S-(3-hydroxypropyl)-L-cysteine (3-HPMA)
3-HPMA is a metabolite of acrolein. Acrolein is commonly used as an herbicide to control weeds and algae in irrigation canals. Humans are exposed to acrolein via oral (fried foods, alcoholic beverages, and water), respiratory (cigarette smoke and automobile exhaust), and dermal routes. On the cellular level, acrolein exposure has diverse toxic effects, including DNA and protein adduction, oxidative stress, mitochondrial disruption, membrane damage, and immune dysfunction. N-acetylcysteine (NAC) or glutathione (GSH) supplementation is recommended as treatment.

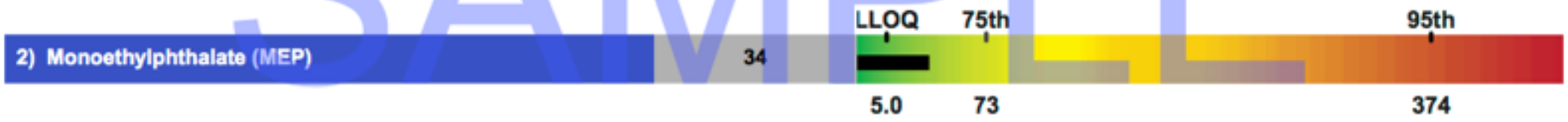
Metabolite	Result µg/g creatinine	Percentile

Industrial Toxicants



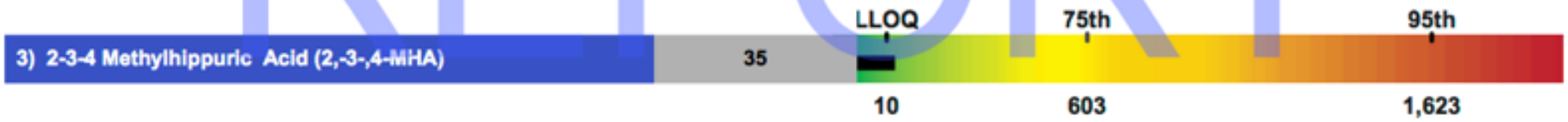
Parent: MTBE/ETBE

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.



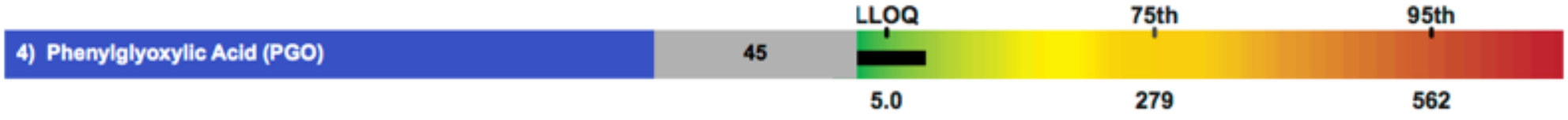
Parent: Diethylphthalates

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.



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.



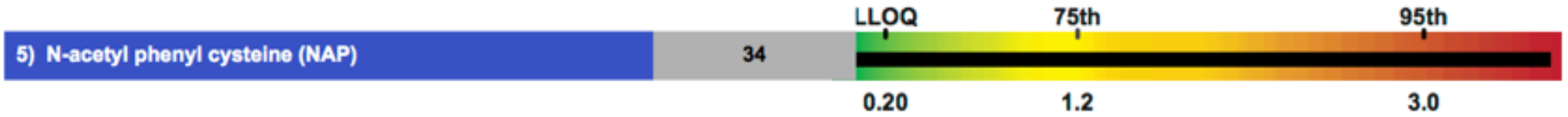
Parent: Styrene/Ethylbenzene

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*

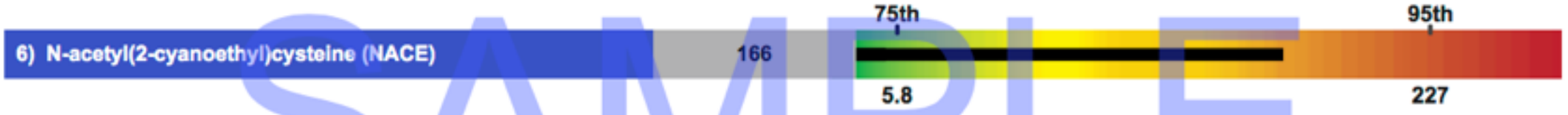
***N.D. - Not Detected*

SAMPLE REPORT



Parent: Benzene

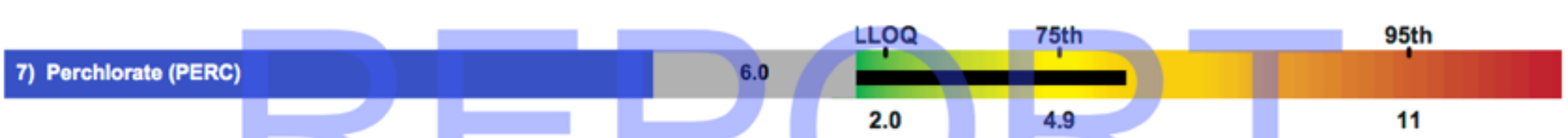
Benzene is an organic solvent that is widespread in the environment. Benzene is a by-product of all types of industrial processes and combustion, including motor vehicle exhaust and cigarette smoke, and is released by outgassing from synthetic materials. Benzene is an extremely toxic chemical that is 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.



SAMPLE

Parent: Acrylonitrile

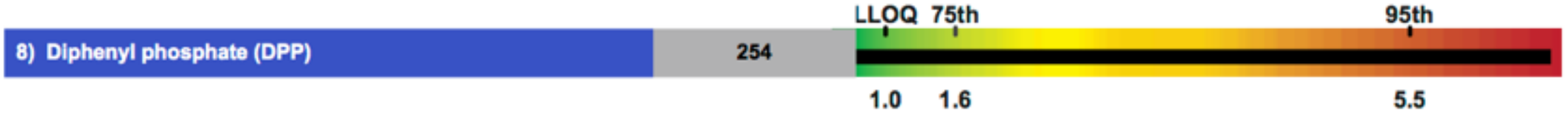
Acrylonitrile is a colorless liquid with a pungent odor. It is used in the production of acrylic fibers, resins, and rubber. Use of any of these products could lead to exposure to acrylonitrile. Smoking tobacco and cigarettes is another potential exposure. Exposure to acrylonitrile can lead to headaches,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



REPORT

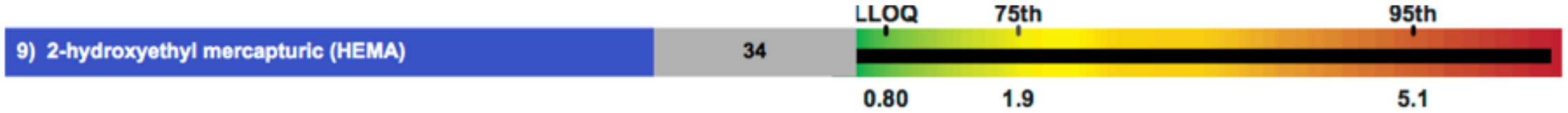
Parent: Perchlorate

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.



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.

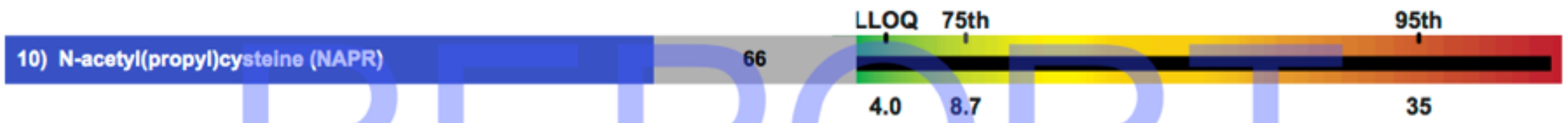


Parent: Ethylene oxide, Vinyl chloride, Halopropane

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]).

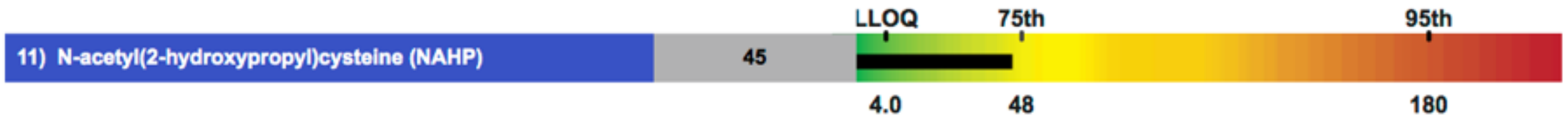
SAMPLE



REPORT

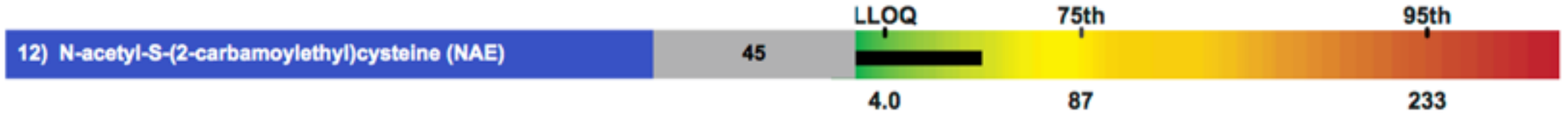
Parent: 1-bromopropane

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.



Parent: Propylene oxide

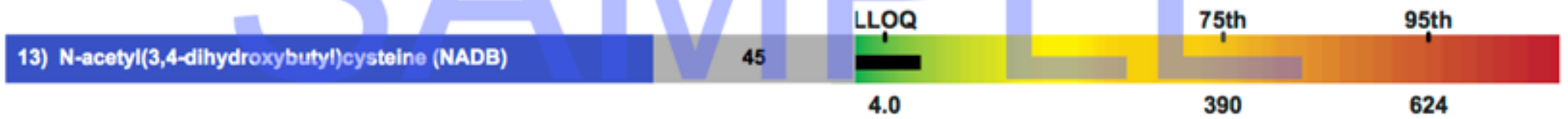
This chemical is used in the production of plastics and is used as a fumigant. Propylene oxide is used to make polyester resins for textile and construction industries. It is also used in the preparation of lubricants, surfactants, and oil demulsifiers. It has also been used as a food additive, an herbicide,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



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.

SAMPLE

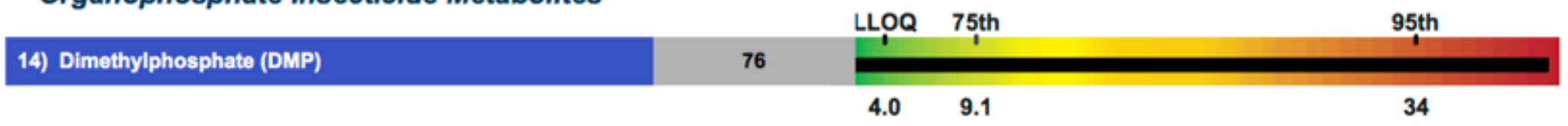


Parent: 1,3 butadiene

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.

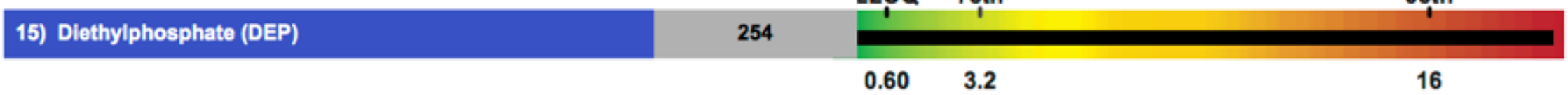
REPORT

Organophosphate Insecticide Metabolites



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.

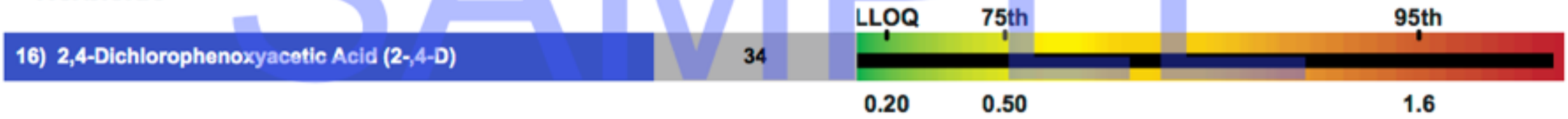


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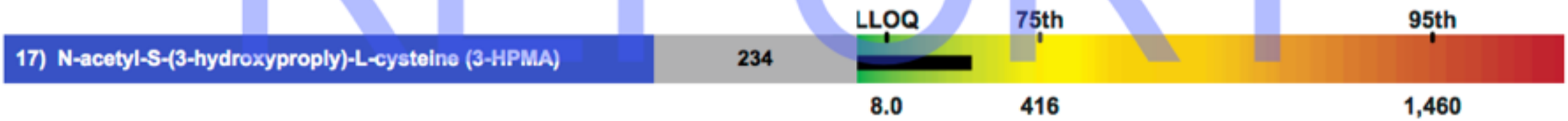
SAMPLE

Herbicide



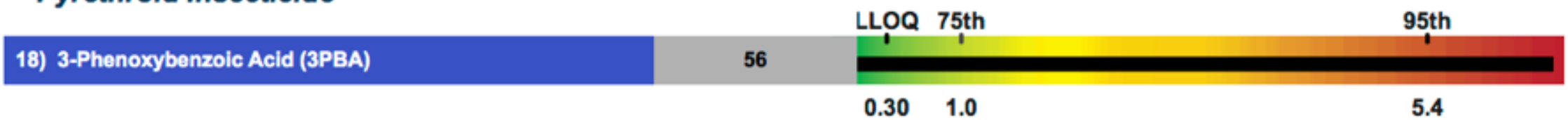
2,4-Dichlorophenoxyacetic Acid (2,4-D) is a very common herbicide that was a part of Agent Orange, which was used by the U.S. in the Vietnam War. It is most commonly used in agriculture on genetically modified foods, and as a weed killer for lawns. Exposure to 2, 4-D via skin or oral ingestion is associated 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.

REPORT



3-HPMA is the main urinary metabolite of acrolein. Acrolein is an environmental pollutant, commonly used as an herbicide and in many different chemical industries. Acrolein is also present in the burning of cigarettes, gasoline, and oil. Certain bacteria produce acrolein, such as Clostridium. Acrolein metabolites are associated with diabetes and insulin resistance.

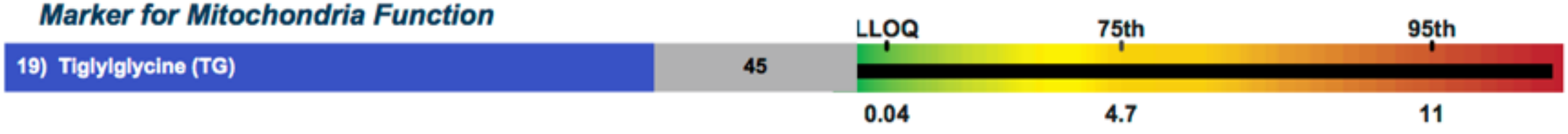
Pyrethroid Insecticide



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

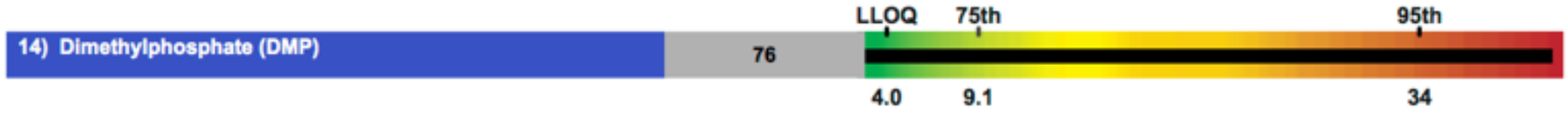
Pyrethrins are widely used as insecticides. Exposure during pregnancy doubles the likelihood of autism. Pyrethrins may affect neurological development, disrupt hormones, induce cancer, and suppress the immune system.

Marker for Mitochondria Function



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.

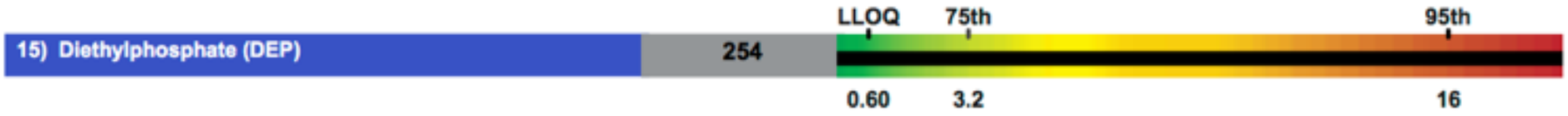
List of Organophosphate Insecticides that are converted to DMP



-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

SAMPLE REPORT

List of Organophosphate Insecticides that are converted to DEP



-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

SAMPLE REPORT

Gentle Detox

Steps to reducing the amount of toxins presently in the body are to switch to eating only organic food and drinking water that has common toxins, including pesticides filtered out. Most conventional food crops are exposed to larger and larger doses of pesticides and herbicides, and by switching to organic you will prevent exposure to hundreds of these toxicants. Many of these chemicals have also contaminated our water supplies. Installing a high-quality water filtration system in the home that eliminates them is important to do and there are several options available.

The next step to avoiding future exposures is to change the products you use on a daily basis – from food and beverage containers to beauty and cleaning products. Instead of using plastic water bottles and food containers, switch to glass or metal. Never microwave food in plastic or styrofoam containers and do not drink hot beverages from plastic or styrofoam cups. Make sure your shampoo, soaps, lotions, and other beauty products are free of phthalates. Use cleaning products made from natural ingredients or make your own at home.

To eliminate toxins from the body, exercise and the use of saunas, especially infrared sauna therapy to rid many chemicals through sweat. Infrared sauna is superior to conventional sauna because it reaches deeper into the body, increasing the circulation in the blood vessels, and causing the body to start to releasing many of the chemicals stored in body fat.

Supplements that are particularly useful in helping the body detoxify are glutathione, or its precursor N-acetyl cysteine. Glutathione is one of the most common molecules used by the body to eliminate toxic chemicals. If you are constantly exposed to toxicants your stores of glutathione could be depleted.

Glyphosate

Glyphosate is the world's most widely produced herbicide and is the primary toxic chemical in Roundup™, as well as in many other herbicides. In addition, it is a broad-spectrum herbicide that is used in more than 700 different products from agriculture and forestry to home use. Glyphosate was introduced in the 1970s to kill weeds by targeting the enzymes that produce the amino acids tyrosine, tryptophan, and phenylalanine. The enzymes of many bacteria are also susceptible to inhibition by this chemical, thus altering the flora of many animals and humans. Usage of glyphosate has since amplified, after the introduction of genetically modified (GMO) glyphosate-resistant crops that can grow well in the presence of this chemical in soil. In addition, toxicity of the surfactant commonly mixed with glyphosate, polyoxyethyleneamine (POEA), is greater than the toxicity of glyphosate alone. Worse yet, in 2014 Enlist Duo™, a herbicide product which contains a 2,4-dichlorophenoxyacetic acid (2,4-D) salt and glyphosate, was approved for use in Canada and the U.S. for use on genetically modified soybeans and genetically modified maize, both of which were modified to be resistant to both 2,4-D and glyphosate. 2,4-D has many toxic effects of its own.

Glyphosate

Recent studies have discovered glyphosate exposure to be a cause of many chronic health problems. It can enter the body by direct absorption through the skin, by eating foods treated with glyphosate, or by drinking water contaminated with glyphosate.

A recent study stated that a coherent body of evidence indicates that glyphosate could be toxic below the regulatory lowest observed adverse effect level for chronic toxic effects, and that it has teratogenic, tumorigenic and hepatorenal effects that can be explained by endocrine disruption and oxidative stress, causing metabolic alterations, depending on dose and exposure time.

The World Health Organization International Agency for Research on Cancer published a summary in March 2015 that classified glyphosate as a probable carcinogen in humans.

Glyphosate

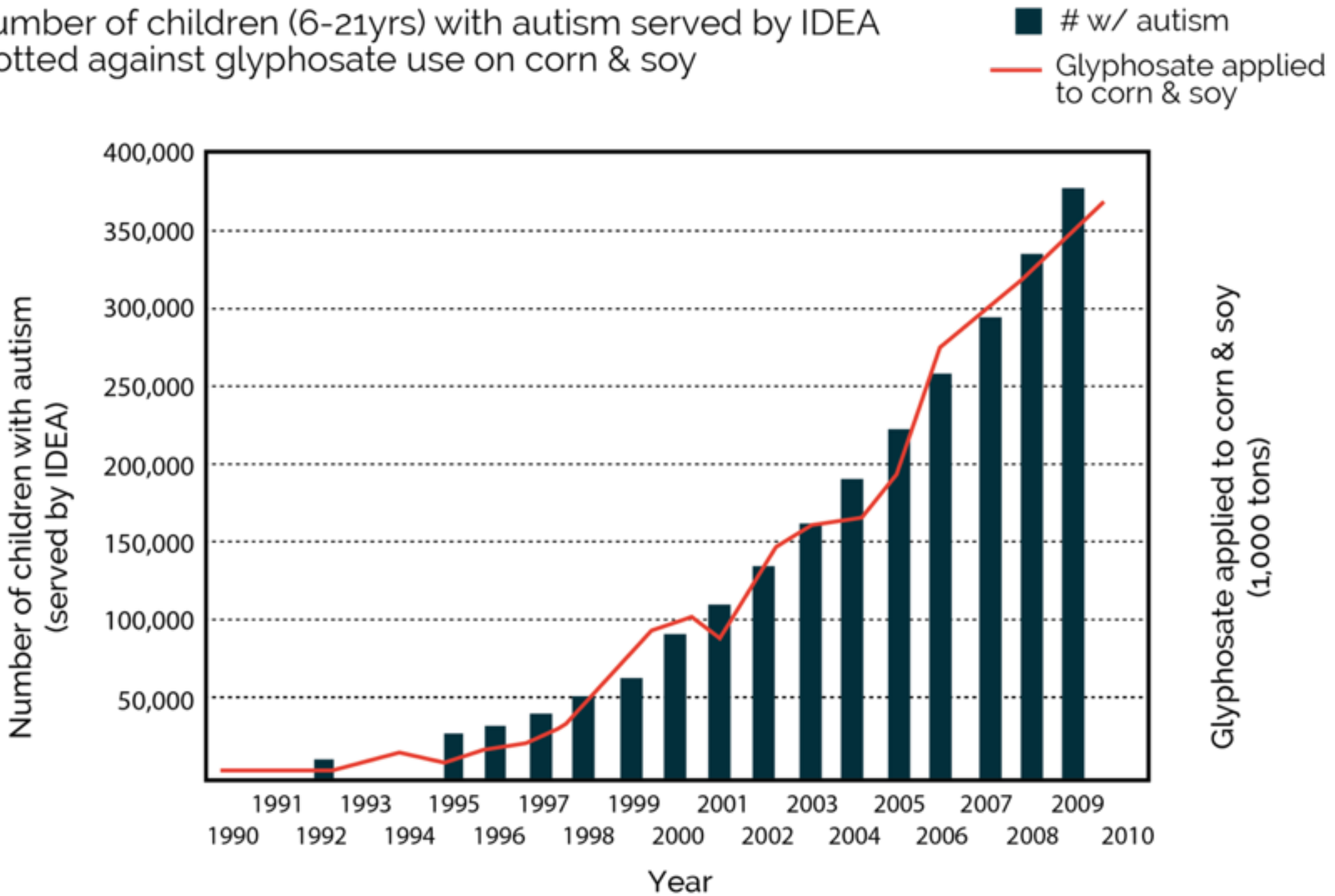
Studies indicated that glyphosate disrupts the microbiome in the intestine, causing a decrease in the ratio of beneficial to harmful bacteria. Thus, highly pathogenic bacteria such as Salmonella *entritidis*, Salmonella*gallinarum*, Salmonella *typhimurium*, Clostridium *perfringens*, and Clostridium *botulinum* ***are highly resistant to glyphosate but most beneficial bacteria*** such as Enterococcus *faecalis*, Enterococcus *faecium*, Bacillus *badius*, Bifidobacterium *adolescentis*, and Lactobacillus *spp*. ***were found to be moderately to highly susceptible.***

The relationship between the microbiome of the intestine and overall human health indicates that disruption of the microbiome could cause diseases such as metabolic disorder, diabetes, depression, autism, cardiovascular disease, and autoimmune disease.

Glyphosate

Glyphosate and Autism*

Number of children (6-21yrs) with autism served by IDEA plotted against glyphosate use on corn & soy



*http://www.examiner.com/article/data-show-correlations-between-increase-neurological-diseases-and-gmos

Glyphosate Profile

Metabolite	Result ug/g creatinine	Reference Range
Glyphosate	3.65	LLOQ 0.38 — 75th 1.8 — 95th 2.5

Glyphosate is the world's most widely produced herbicide. It is a broad-spectrum herbicide that is used in more than 700 different products for agriculture and forestry to home use. Glyphosate was introduced in the 1970s to kill weeds by targeting the enzymes that produce the amino acids tyrosine, tryptophan, and phenylalanine. Usage of glyphosate has since amplified, after the introduction of genetically modified (GMO) glyphosate-resistant crops.

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Treatment of glyphosate toxicity should be centered on determining the route of introduction and avoiding future exposure. Glyphosate is readily metabolized in the body. However, a recent study found that glyphosate accumulates in mammalian bones. Another study found glyphosate to be detectable in mammalian intestine, spleen, liver, muscle, and kidney. Kidney impairment is common in regions where glyphosate may accumulate in ground water as metal chelates. The most effective way to reduce glyphosate exposure is to avoid living in areas where glyphosate is applied and to avoid eating GMO foods or animal products such as milk or meat for which GMO foods were used to feed the animals. Since glyphosate is now commonly combined with the weed killer 2,4-dichlorophenoxyacetic acid (2,4-D), testing for this chemical with the GPL-TOX test may wish to be considered also.

*LLOQ - Lower Limit of Quantitation

Mycotoxin Test

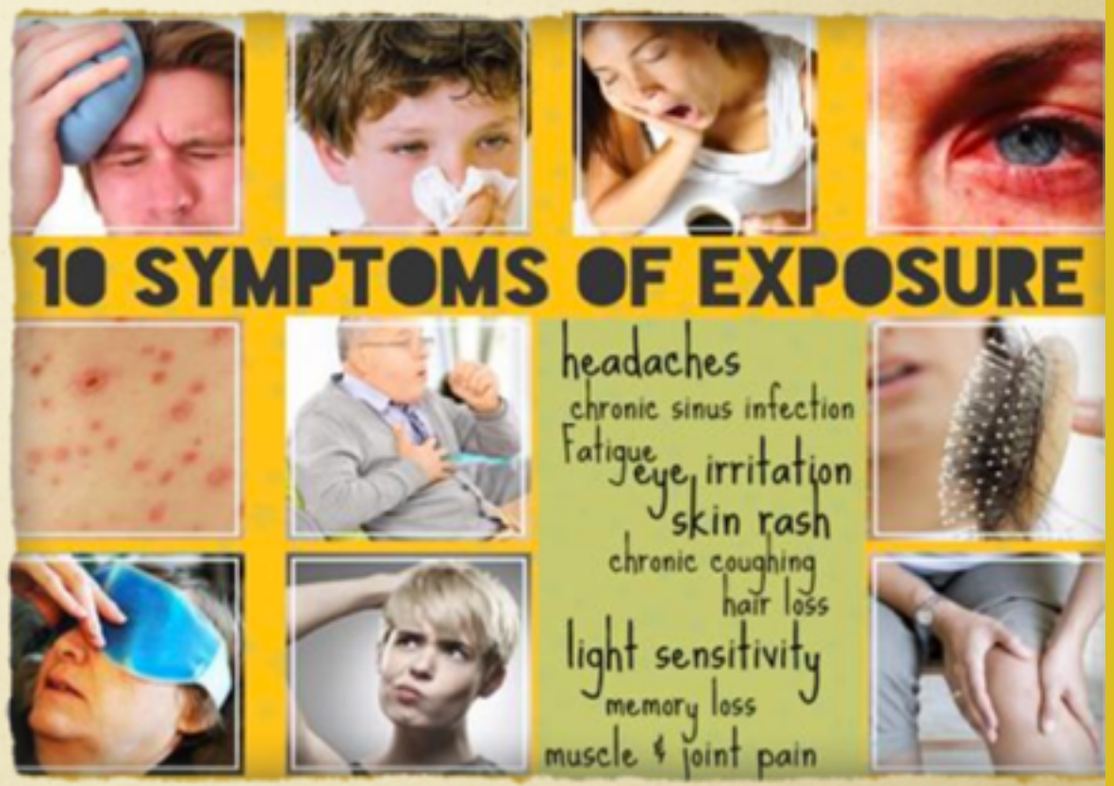
Mycotoxins (toxins from mold) are some of the most prevalent toxins in the environment. Mycotoxins are metabolites produced by fungi like mold, which can infest buildings, vehicles, and foodstuffs. Most mycotoxin exposures in North America, Europe, and Australia are through airborne exposure.

Food may be a major source of mycotoxins in third-world nations that lack government inspection of foods, especially the grains such as rice, corn, wheat, rye, and barley. Many grains that are harvested have been found to be contaminated with mycotoxins but little of such harvests are discarded and are frequently sold for animal feeds such as dog food or feed for cows, pigs, chickens, turkeys, and horses.

Fungi can grow on almost any surface, especially if the environment is warm and wet. Inner wall materials of buildings, wallpaper, fiber glass insulation, ceiling tiles, and gypsum support are all good surfaces for fungi to colonize. These fungi then release mycotoxins into the environment causing symptoms of many different chronic diseases. Diseases and symptoms linked to mycotoxin exposure include fever, pneumonia-like symptoms, heart disease, rheumatic disease, asthma, sinusitis, cancer, memory loss, vision loss, chronic fatigue, skin rashes, depression, ADHD, anxiety, and liver damage.

MOLD SICKNESS IS HARD TO IDENTIFY

- Mold sickness develops slowly over the course of a few months.
- The symptoms are vague, low grade, and likely different from others with the same exposure.
- Because mold can hide inside a building, the symptoms aren't often tied back to mold.
- One clue it might be mold is if symptoms are worse on rainy days, after snow melt, with barometric pressure changes, and after eating carbs.



How Mycotoxins Affect Living Beings

- •Causes inflammation in sinuses, lungs, bladder, and digestive tract
- •Migrates across linings of the respiratory and digestive tracts
- •Absorbs and stores in fat
- •Interferes with vital cellular processes
- •Causes mitochondrial damage
- •Impairs synthesis of protein, RNA, and DNA
- •Depletes the master cell antioxidant called glutathione
- •Accelerates programmed cell death
- •Is poisonous to nerves in the body and brain
- •Is toxic to liver and kidneys
- •Affects medications metabolized by the cytochrome p450 system
- •Inhibits immune defenses
- •Causes some cancers
- •Winnows away the lining of the intestines
- •Crosses into the brain and weakens the blood-brain barrier
- •Rides the olfactory nerve to the hippocampus and frontal lobe
- •Crosses the placenta and becomes more active inside the uterus
- •Is detectable in breast milk

Symptoms of Mold Illness

EYES, EARS NOSE & THROAT (EENT) Sneezing; Runny nose; Post-nasal drip; Chronic sinusitis; Nasal polyps; Bumps at back of the throat; Swollen lymph nodes; Allergies; Hay fever; Ear popping; Ringing in the ears; Hearing loss; Dry eyes; Irritated eyes

RESPIRATORY SYSTEM Shortness of Breath; Wheezing; Asthma; Chronic dry cough; Burning lungs; Heaviness in the chest; Sensitivity to fragrances; Colds go to the lungs easily; Chronic respiratory illnesses; Blood-stained sputum; Smoke & exhaust sensitivity; Aspergillosis

DIGESTIVE SYSTEM Appetite changes; Nausea; Irritable bowels; Diarrhea/ Constipation; Vomiting; Cyclical vomiting syndrome; Bloating; Abdominal pain; Ulcers; Food sensitivities; Sweet cravings

CIRCULATORY SYSTEM Many spider veins; Cherry angiomas; Easy bruising; Easy bleeding; Iron-deficiency anemia; Varicose veins; Raynaud's phenomenon; Irregular heartbeat; Low or reactive blood pressure; Atriovenous malformation

SKIN Sensitive skin; Itchy skin; Burning sensation; Flushing ;Sensitivity to sunlight; Skin rash; Peeling or sloughing skin; Fungal infections

More Symptoms of Mold Illness

BRAIN Brain fog; Confusion; Slowed thinking; Memory loss; Trouble finding the right word; Dementia

NERVOUS SYSTEM Anxiousness; Depression; Incoordination; Headache; Dizziness/Vertigo; Migraine; Slow reflexes; Dysautonomia; Insomnia; Neuropathies; Tremors; Seizures; Daytime sleepiness; Difficulty with balance and walking

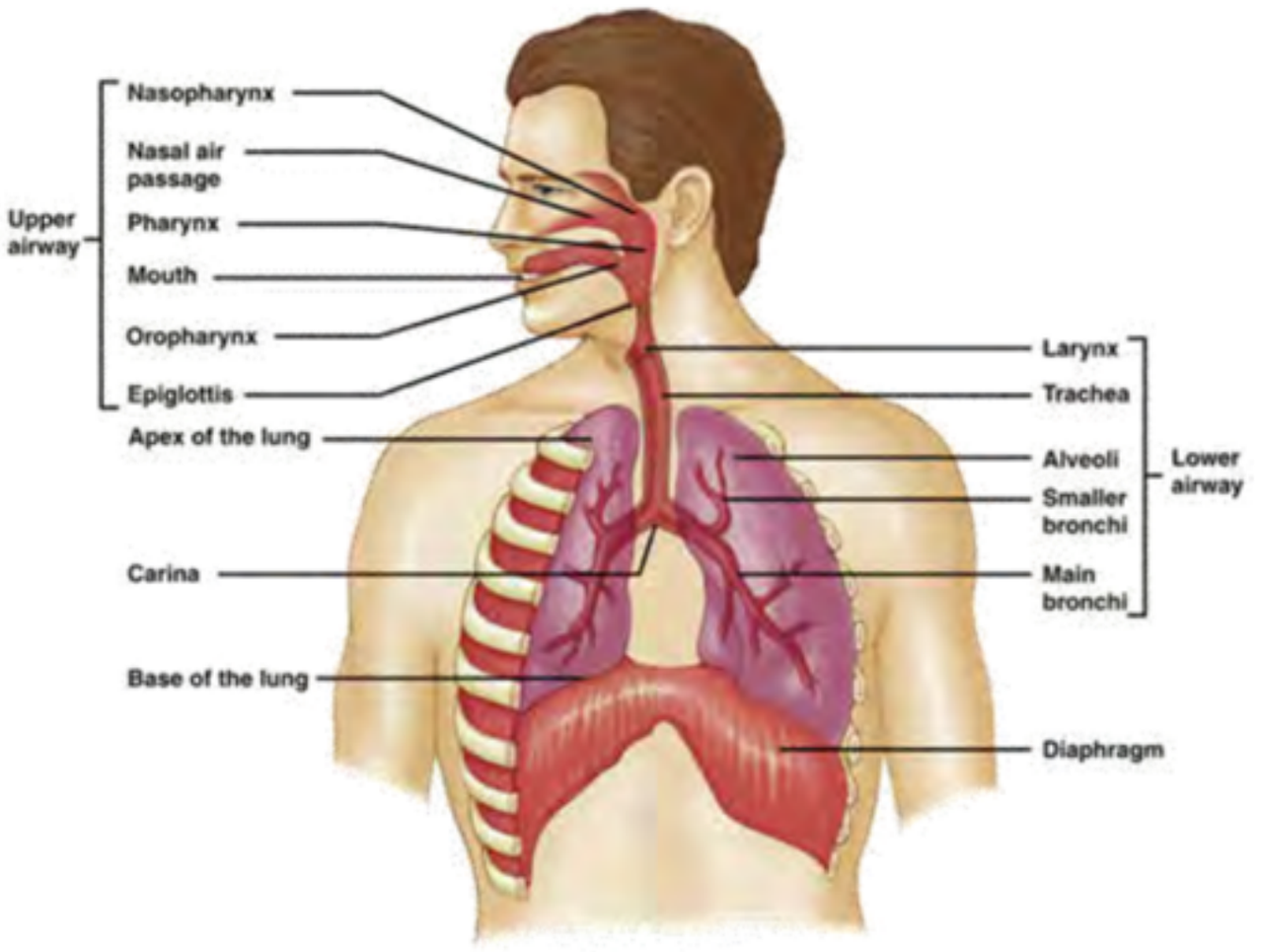
URINARY SYSTEM Overactive bladder; Irritable bladder; Blood in urine; Kidney inflammation; Urinary tract infection symptoms with no identifiable infection

IMMUNE SYSTEM Increased susceptibility to infection; Long-lasting colds; Viral infections become bacterial ;Chronic mono or Epstein-Barr virus

REPRODUCTIVE SYSTEM Changes in menstrual cycle; Infertility in both genders; Vaginal yeast or bacterial infections ;Jock itch

Main Mycotoxin Exposure Pathways

Eating: People are constantly being exposed to significant levels of mold toxins via their diet when they eat grains, meat, farmed fish, poultry and many fruits. Generally the level of mycotoxin exposure through the food chain, at least in the U.S. and Europe (but not so in sub-Saharan Africa), is low enough so that a healthy body's natural detox functions (via liver and kidneys) can adequately clean these toxins from the body quickly enough so they do no damage.



Breathing: The human respiratory tract can be considered to consist of two regions:

(1) Upper airway. Nose through Epiglottis
(2) Lower airway. Larynx through Lungs

People living in mold contaminated homes and/or with mold growth in their AC's, ducting and/or AC closet, breath elevated levels of mold spores and/or mold fragments. In the recent CDC funded research study on mold spore inhalation (Cho et al.) it was shown that the majority of inhaled mold spores land in the upper airway and never reach the lungs.

The (toxin laden) mold spores that land in the upper airway adhere to the airway mucous lining. Once embedded in the mucous, the spores are rapidly cleared from the airway and are deposited in the digestive tract for later excretion.

The majority of inhaled toxin laden mold spores therefore *are effectively eaten*.

Mycotoxins absorbed from the digestive tract will enter the blood stream and directly affect the body.

But often more important is the indirect affect on the body by damage occurring to gut bacteria/ microflora because mycotoxins are strongly bacteriocidal.

Levels of mold toxins that can negatively affect the gut microflora (microbiome) are likely 100's or even thousands of times lower than mycotoxin levels required to directly cause mold related illnesses by toxic impact on human organs/cells.

Therefore gut related problems from mold toxin exposure can be particularly common.

The principal route of exposure from *breathing* mold toxins
is via the gut and not the lungs.

Subsequently, a common result of elevated mycotoxin exposure is
damage to gut microflora.

Neurotoxic Mycotoxins

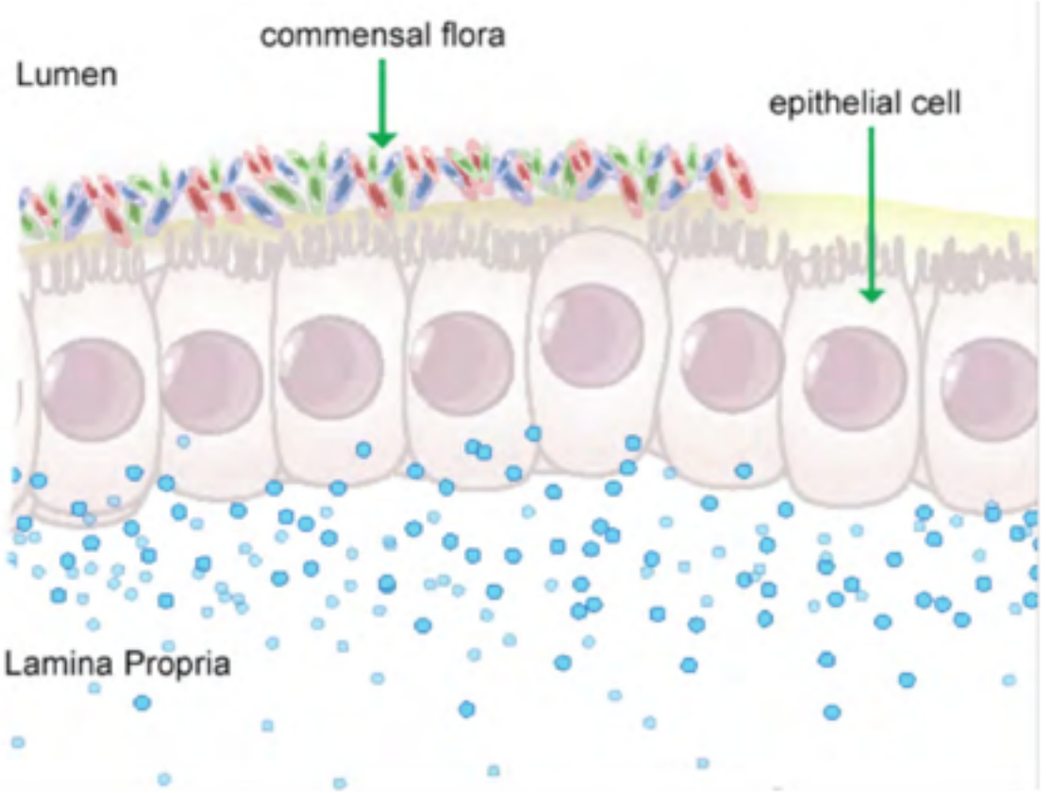
Some commonly occurring mold toxins can cause Neurological problems such as: Hheadaches, short term memory loss, attention deficit problems, anxiety, neurological impairment in children and other problems. It is well established that some mycotoxins (toxins from Stachybotrys and Tricho-derma both molds commonly found in water damaged homes) are neurotoxic and exposure to such neurotoxins can result in some degree of neurological impairment.

The diagnosis of such exposure can be aided by the Visual Contrast Sensitivity (VCS) deficit test developed by Dr. Hudnell of the U.S. EPA, when there is no other reasonable explanation for the illness.

Since there are usually no reliable tests available to identify these neurotoxins in human tissue, blood or urine, proof of the cause of illness is VCS recovery and symptom resolution following treatment with toxin binders. The test can be taken online at: www.chronicneurotoxins.com. Note that unless the individual is removed from the mycotoxin laden environment, or the environment is fixed, the individual cannot heal. If you test positive, call your doctor and your mold remediator.

Mycotoxins and the Gastrointestinal Tract

Gut Microflora: Many or most mycotoxins of concern (including the well known mold toxin Penicillin) exhibit strong anti-bacterial affects (bacteriocidal). Chronic exposure to even very low levels of these bacteriocidal toxins (at mold levels in many homes) can damage the good gut bacteria that make up our gut micro flora. For more on gut flora see: http://en.wikipedia.org/wiki/Gut_flora.



A healthy gut is critical to overall health. Commensal gut microflora are involved in many critical functions including production of essential vitamins; proper and complete food digestion; as well, gut microflora are an important player in the body's detox capability.

When the gut microflora become out of balance as a result of mycotoxin exposure (dysbiosis), many adverse health effects can result including: a shift toward intestinal aerobic bacteria such as observed in inflammatory bowel diseases; obesity (additional obesity links †, ††, †††); as well as yeast/Candida overgrowth in the gut.

Yeast/Candida secretes the powerful immunosuppressive Gliotoxin (the same causative agent produced by Aspergillus molds resulting in Invasive Aspergillosis) and Candida also secretes proteins so it can attach itself permanently to the intestinal lining. Once yeast has taken over the gut, it does not simply go away. A healthy gut cannot be restored without medical treatment. No carb/ no sugar diets, anti-fungals, probiotics and sometimes fecal transplants (all under Doctor supervision) are solutions to yeast over-growth. Again, unless the sick individual is removed from the mycotoxin-laden environment they will not get better no matter how brilliant the doctor!

Mycotoxin Test

ADVANTAGES OF THE MYCOTOXIN PROFILE

- Screens for eleven different mycotoxins, from 40 species of mold, in one urine sample necessary to detect lower levels of these fungal toxins.

11 different mycotoxins tested from a wide variety of 13 mold types (genera) including *Aspergillus, Penicillium, Fusarium, Myrothecium, Stachybotrys, Bipolaris, Gibberella, Chaetomium, Trichoderma, Trichothecium, Cephalosporium, Verticimonosporium*, and *Monascus*. Since each of these genera contain multiple species, this test is likely to detect hundreds of mold species. In addition, poisonous mushrooms also produce some of the same mycotoxins as the molds.

Mycotoxin Test

ASPERGILLUS

Aspergillus is the most prevalent mold group in the environment and there are hundreds of different species. It has caused billions of dollars in damage to crops and livestock. Common *Aspergillus* mycotoxins tested include *aflatoxin, ochratoxin, sterigmatocystin, gliotoxin,* and *citrinin*. The main target of these toxins is the liver. These toxins have been found in all major cereal crops including peanuts, corn, cotton, millet, rice, sorghum, sunflower seeds, wheat, and a variety of spices. They are also found in eggs, milk, and meat from animals fed contaminated grains. Diseases caused by *Aspergillus* are called aspergillosis. Most common route of infection is through the respiratory system. *Aspergillus* can cause severe asthma when the mold colonizes the lung, forming a granulomatous disease.

PENICILLUM

There are over 200 species of the genus *Penicillium* that have been discovered. *Penicillium chrysogenum* is the most common of these species. It is often found in indoor environments and is responsible for many allergic reactions. *Penicillium* is also a known contaminant in many different food items. Many different types of citrus fruits can become contaminated with *Penicillium*, but it can also contaminate seeds and grains. One reason that *Penicillium* is such a common infestation is because of its ability to thrive in low humidity. In the home, *Penicillium* can be found in wallpaper, carpet, furniture, and fiberglass insulation. The most common mycotoxin produced by *Penicillium* is ochratoxin. Ochratoxin is nephrotoxic, which means that it damages the kidneys. It is also carcinogenic.

STACHYBOTRYS

Stachybotrys is a greenish-black mold. This mold can grow on materials with high cellulose and low nitrogen content such as gypsum board, paper, fiberboard, and ceiling tiles. *Stachybotrys* is known for its production of the highly toxic macrocyclic trichothecene mycotoxins. Two of the more common mycotoxins produced by *Stachybotrys* are roridin E and verrucarin. In addition to these mycotoxins, the fungus produces nine phenylspirodrimanes, as well as cyclosporine, which are potent immunosuppressors. These immunosuppressors, along with the mycotoxin trichothecenes may be responsible for the high toxicity of *Stachybotrys*.

FUSARIUM

Fusarium's major mycotoxins are zearalenone (ZEN) and fumonisin. *Fusarium* fungi grow best in temperate climate conditions. They require lower temperatures for growth than Aspergillus. Fusarium grows worldwide on types of grains including corn and wheat. Exposure to mycotoxins from *Fusarium* can lead to both acute and chronic effects. Symptoms can include abdominal distress, malaise, diarrhea, emesis, and death. ZEN possesses estrogenic effects and has been implicated in reproductive disorders.

Mycotoxin Test

AFLATOXIN M1 (AFM1)

Aflatoxin M1 (AFM1) is the main metabolite of aflatoxin B1, which is a mycotoxin produced by different species of the genus *Aspergillus*. Aflatoxins are some of the most carcinogenic substances in the environment. Aflatoxin susceptibility is dependent on multiple different factors such as age, sex, and diet. Aflatoxin can be found in beans, corn, rice, tree nuts, wheat, milk, eggs, and meat. In cases of lung aspergilloma, aflatoxin has been found in human tissue specimens. Aflatoxin can cause liver damage, cancer, mental impairment, abdominal pain, hemorrhaging, coma, and death. Aflatoxin has been shown to inhibit leucocyte proliferation. Clinical signs of aflatoxicosis are non-pruritic macular rash, headache, gastrointestinal dysfunction (often extreme), lower extremity edema, anemia, and jaundice. The toxicity of aflatoxin is increased in the presence of ochratoxin and zearalenone.

OCHRATOXIN A (OTA)

Ochratoxin A (OTA) is a nephrotoxic, immunotoxic, and carcinogenic mycotoxin. This chemical is produced by molds in the *Aspergillus* and *Penicillium* families. Exposure is primarily through contaminated foods such as cereals, grape juices, dairy, spices, wine, dried vine fruit, and coffee. Exposure to OTA can also come from inhalation exposure in water-damaged buildings. OTA can lead to kidney disease and adverse neurological effects. Studies have shown that OTA can cause significant oxidative damage to multiple brain regions and the kidneys. Dopamine levels in the brain of mice have been shown to be decreased after exposure to OTA.

STERIGMATOCYSTIN (STG)

Sterigmatocystin (STG) is a mycotoxin that is closely related to aflatoxin. STG is produced from several types of mold such as *Aspergillus*, *Penicillium*, and *Bipolaris*. It is carcinogenic, particularly in the cells of the GI tract and liver. STG has been found in the dust from damp carpets. It is also a contaminant of many foods including grains, corn, bread, cheese, spices, coffee beans, soybeans, pistachio nuts, and animal feed. In cases of lung aspergilloma, STG has been found in human tissue specimens. The toxicity of STG affects the liver, kidneys, and immune system. Tumors have been found in the lungs of rodents that were exposed to STG. Oxidative stress becomes measurably elevated during STG exposure, which causes a depletion of antioxidants such as glutathione, particularly in the liver.

ZEARALENONE (ZEA)

Zearalenone (ZEA) is a mycotoxin that is produced by species of the mold genera *Fusarium* and *Gibberella*. It has been shown to be hepatotoxic, hematotoxic, immunotoxic, and genotoxic. ZEA is commonly found in several foods in the US, Europe, Asia, and Africa including wheat, barley, rice, and maize. ZEA has estrogenic activity and exposure to ZEA can lead to reproductive changes. ZEA's estrogenic activity is higher than that of other non-steroidal isoflavones (compounds that have estrogen-like effects) such as soy and clover. ZEA exposure can result in thymus atrophy and alter spleen lymphocyte production as well as impaired lymphocyte immune response, which leads to patients being susceptible to disease.

Mycotoxin Test

RORIDIN E

Roridin E is a macrocyclic trichothecene produced by the mold genera *Fusarium, Myrothecium, Trichoderma, Trichothecium, Cephalosporium, Verticimonosporium,* and *Stachybotrys* (i.e. black mold). Trichothecenes are frequently found in buildings with water damage but can also be found in contaminated grain. This is a very toxic compound, which inhibits protein biosynthesis by preventing peptidyl transferase activity. Trichothecenes are considered extremely toxic and have been used as biological warfare agents. Even low levels of exposure to macrocyclic trichothecenes can cause severe neurological damage, immunosuppression, endocrine disruption, cardiovascular problems, and gastrointestinal distress.

VERRUCARIN A

Verrucarin A is a macrocyclic trichothecene mycotoxin produced from *Stachybotrys, Fusarium,* and *Myrothecium* molds. Trichothecenes are frequently found in buildings with water damage but can also be found in contaminated grain. This is a very toxic compound, which inhibits protein biosynthesis by preventing peptidyl transferase activity. Trichothecenes are considered extremely toxic and have been used as biological warfare agents. Even low levels of exposure to macrocyclic trichothecenes can cause severe neurological damage, immunosuppression, endocrine disruption, cardiovascular problems, and gastrointestinal distress.

ENNIATIN B

Enniatin B1 is a fungal metabolite categorized as a cyclohexa-depsipeptides toxin produced by the molds of the *Fusarium* species. These species are common cereal contaminants. Grains in many different countries have recently been contaminated with high levels of enniatin. The toxic effects of enniatin are caused by the inhibition of the acyl-CoA cholesterol acyltransferase, depolarization of mitochondria, and inhibition of osteoclastic bone resorption. Enniatin has antibiotic properties and chronic exposure may lead to weight loss, fatigue, and liver disease.

GLIOTOXIN*

Gliotoxin (GTX) is produced by the mold genus *Aspergillus* and perhaps *Candida* species. *Aspergillus* spreads in the environment by releasing conidia which are capable of infiltrating the small alveolar airways of individuals. To evade the body's defenses *Aspergillus* releases gliotoxin to inhibit the immune system. One of the targets of gliotoxin is PtdIns (3,4,5) P3. This results in the downregulation of phagocytic immune defense, which can lead to the exacerbation of polymicrobial infections. *Gliotoxin* impairs the activation of T-cells and induces apoptosis in monocytes and in monocyte-derived dendritic cells. These impairments can lead to multiple neurological syndromes.

Mycotoxin Test

MYCOPHENOLIC ACID*

Mycophenolic Acid (MPA) is produced by the *Penicillium* fungus. MPA is an immunosuppressant which inhibits the proliferation of B and T lymphocytes. MPA exposure can increase the risk of opportunistic infections such as Clostridia and *Candida*. MPA is associated with miscarriage and congenital malformations when the woman is exposed in pregnancy.

DIHYDROCITRINONE*

Dihydrocitrinone is a metabolite of Citrinin (CTN), which is a mycotoxin that is produced by mold species of the genera *Aspergillus*, *Penicillium*, and *Monascus*. CTN exposure can lead to nephropathy, because of its ability to increase permeability of mitochondrial membranes in the kidneys. The three most common exposure routes are through ingestion, inhalation, and skin contact. CTN has been shown to be carcinogenic in rat studies. Multiple studies have linked CTN exposure to a suppression of the immune response.

CHAETOGLOBOSIN A*

Chaetoglobosin A (CHA) is produced by the mold *Chaetomium globosum* (CG). CG is commonly found in homes that have experienced water damage. Up to 49% of water-damaged buildings have been found to have CG. CHA is highly toxic, even at minimal doses. CHA disrupts cellular division and movement. Most exposure to CG is through the mycotoxins because the spores tend not to aerosolize. Exposure to CHA has been linked to neuronal damage, peritonitis, and cutaneous lesions.

Mycotoxin Test

TREATMENT FOR MYCOTOXIN EXPOSURE

All mycotoxin exposures are basically treated the same.

1) Remove the person from exposure by moving or remediating the home, school, or office.

There are a wide variety of tests that can be used. Just walking around your residence with a critical eye and smelling for a moldy smell is the first thing to be done. Also, a positive urine mycotoxin test is a common indicator of residential or office mold since food contamination with mold is uncommon in the United States. The next step would be to check the internet for a mold inspector who can give references.

2) Treat with antifungals to remove any molds colonizing the GI tract, lungs, or sinuses.

This step is frequently omitted which is, in my opinion, the reason mold related illnesses drag on for years sometimes. Retest the mycotoxins after 6 weeks of treatment. If significant mycotoxins still remain, a new antifungal treatment may be needed, or the dosage may have to be increased.

3) Helping the body remove the mycotoxins.

Treat with binders such as charcoal and/or clay or combinations of charcoal and clay such as GI DETOX. Glutathione or N-acetylcysteine (NAC) at doses of 500 mg per day for adults may also be useful to remove residual mycotoxins.

Mycotox Profile

Creatinine Value: 136.37 mg/dl

Metabolite	Results (ng/g creatinine)	Normal Range	Abnormal Range
Aspergillus			
Aflatoxin-M1	2.53	< 0.5	▲ 0.5
Ochratoxin A	13.08	< 7.5	▲ 7.5
Gliotoxin	< 200.00	< 200	▲ 200
Penicillum			
Sterigmatocystin	0.00	< 0.4	▲ 0.4
Mycophenolic Acid	0.00	< 37.4	▲ 37.4
Stachybotrys			
Roridin E	0.00	< 0.2	▲ 0.2
Verrucarin A	0.00	< 1.4	▲ 1.4

SAMPLE REPORT

Metabolite	Results (ng/g creatinine)	Normal Range	Abnormal Range
Fusarium			
Enniatin B	0.00	< 0.3	▲ 0.3
Zearalenone	0.00	< 3.2	▲ 3.2
Chaetomium globosum			
Chaetoglobosin A	0.00	< 10	▲ 10
Multiple Mold Species			
Citrinin	< 25.00	< 25	▲ 25

SAMPLE REPORT

MOLD CAUSES CRAVINGS

- Mold feeds on carbohydrates, and makes the affected person crave sweets

- If mold is not fed, it begins to die inside your body. As it dies, mold releases toxic chemicals that travel to your brain and affect how you think. Without sweets, mold-sick people describe a sense of deep fog over the brain.

- These chemicals also increase pain all over the body.

- As the mold and yeast eat the food themselves, this reduces nutrient absorption through the intestinal lining.



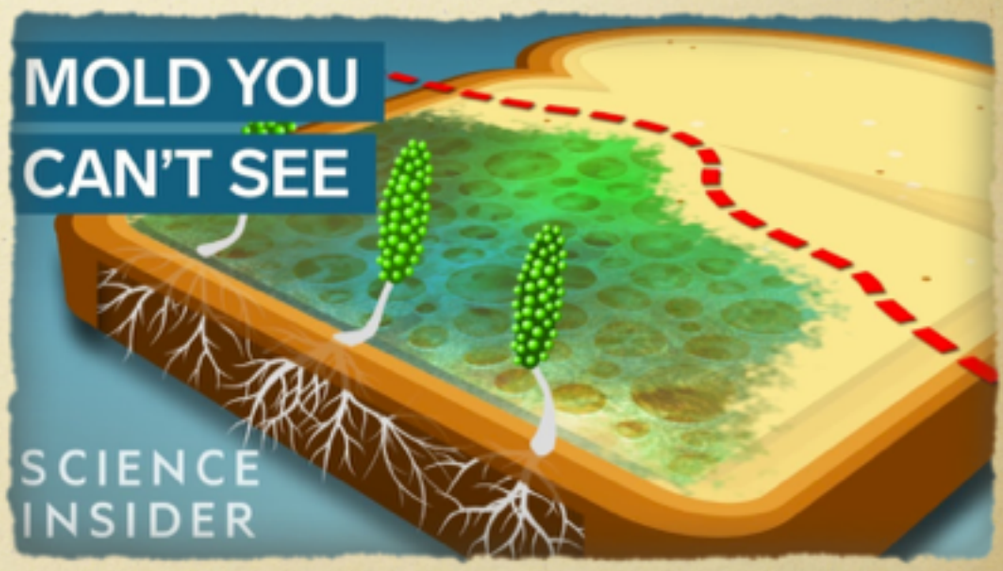
MOLD CAUSES FOOD SENSITIVITIES

- Under the influence of mold, your body begins to view commonly eaten foods as a perpetual threat, something to be attacked. This can cause bloating and inflammation in the intestines.

- Mycotoxins can be ingested in the food we eat. This is true for pets as well. Mycotoxins are most often found in grains, flour, and dried fruit. Mold may be a contributor to the growing trend of grain allergies.

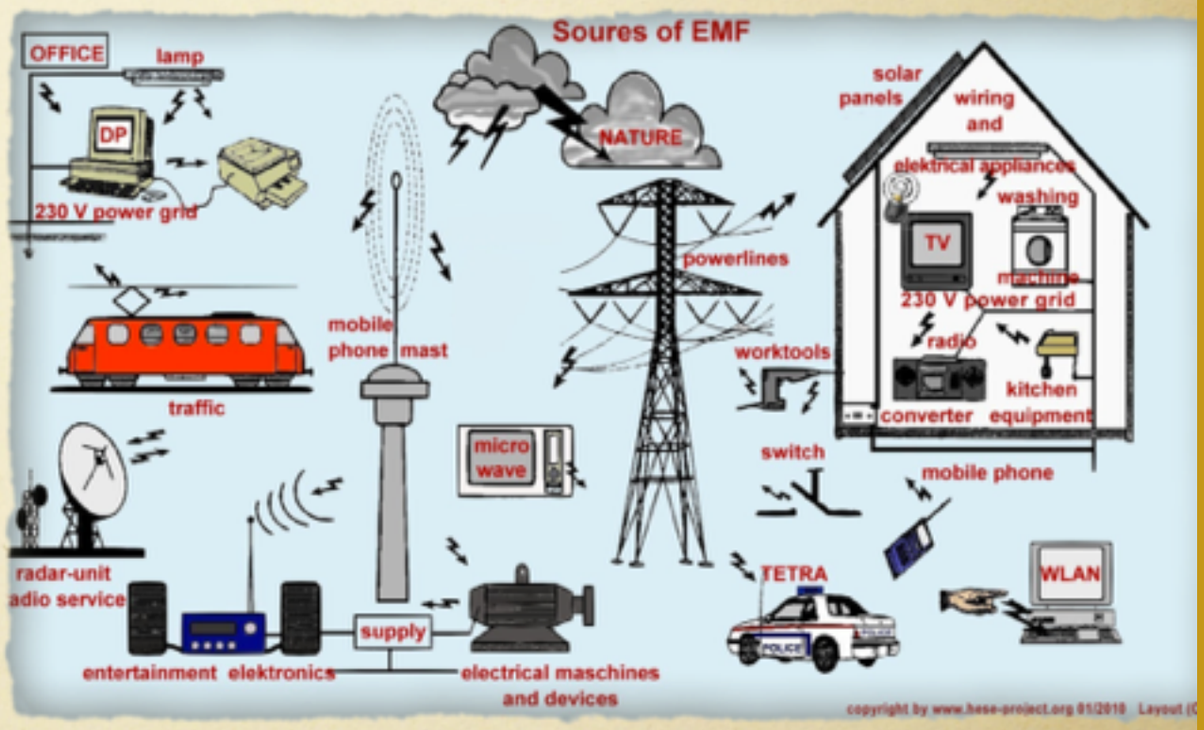
- When mycotoxins travel down the digestive tract, they destroy the intestinal lining. Our smart bodies try to get rid of the toxins by flushing the colon, causing diarrhea. Then constipation may follow. It's not unusual for mold-sick people to develop irritable bowels, alternating constipation with diarrhea,





MOLD MAKES YOU SENSITIVE TO ELECTROMAGNETIC FIELDS

- At the cellular level, mold changes our sensitivity to electrical currents.
- Electromagnetic fields (EMFs), such as Wi-Fi, do the same.
- Mold-sick people have a harder time being around EMFs because mold has impaired their sensitivity to electrical signals.



CHECK **ALL SYMPTOMS** EXPERIENCED IN THE **PAST 3-6 MONTHS**

CATEGORY 1

- ☐ Brain fog
- ☐ Feel tired all the time
- ☐ Frequent runny nose
- ☐ Blow your nose often
- ☐ Sneezing
- ☐ Sinusitis
- ☐ Post-nasal drip
- ☐ Nose bleeds
- ☐ Swollen glands
- ☐ Shortness of breath
- ☐ Frequent yawning or sighing
- ☐ Heart palpitations
- ☐ Headaches
- ☐ Hay fever
- ☐ Eye irritation
- ☐ Blurry vision
- ☐ Frequent change in vision
- ☐ Allergies
- ☐ Dark circles under eyes
- ☐ Sensitivity to sunlight
- ☐ Nervousness/can't settle
- ☐ Low mood or depressed
- ☐ Feeling overwhelmed
- ☐ Episodic/chronic dry cough
- ☐ Irritated lungs
- ☐ Blood-streaked mucous
- ☐ Nasal polyps
- ☐ Coated tongue
- ☐ Sores in the mouth
- ☐ Bumps on back of throat
- ☐ Thrush
- ☐ Sore or itchy ear canals
- ☐ Ringing in the ears
- ☐ Bothered by loud noises
- ☐ Skin rash
- ☐ Burning or itchy skin
- ☐ Easy bruising
- ☐ Spider veins
- ☐ Bothered by tags and seams on clothing
- ☐ Anemia
- ☐ Protruding veins on limbs
- ☐ Lower extremity edema
- ☐ Clear your throat often
- ☐ Sore throat
- ☐ Frequent colds
- ☐ Delayed recovery from colds
- ☐ Exhausted from exercise
- ☐ Frequent static shocks
- ☐ Increased thirst
- ☐ Trouble sleeping
- ☐ Feeling of internal vibration
- ☐ Dizziness
- ☐ Vertigo
- ☐ Drunken feeling
- ☐ Frequent urination
- ☐ Yeast infection
- ☐ Change in appetite
- ☐ Intestinal gas
- ☐ Nausea
- ☐ Feeling bloated
- ☐ Constipation
- ☐ Crave sweets
- ☐ Crave alcohol

TOTAL **CATEGORY 1** BOXES MARKED: _____

0-4 boxes marked = Score 0
5-9 boxes marked = Score 1
10-15 boxes marked = Score 2
16+ boxes marked = Score 3

CATEGORY 1 SCORE _____

CATEGORY 2

- ☐ Wheezing
- ☐ Asthma
- ☐ Burning lungs
- ☐ Recurrent respiratory infections
- ☐ Migraine
- ☐ Allergies aren't well controlled by medication
- ☐ Voice sounds nasally
- ☐ Plugged or clogged ears
- ☐ Chronic sinusitis
- ☐ Vomiting
- ☐ Alternating constipation/diarrhea
- ☐ Diarrhea
- ☐ Irritable bowel
- ☐ Food sensitivities
- ☐ Chemical sensitivities
- ☐ Abnormal reaction to antibiotics
- ☐ Epstein-Barr virus
- ☐ Recurrent yeast infections
- ☐ Bacterial vaginosis
- ☐ Recurrent athlete's foot, jock itch, or toenail fungus
- ☐ Peeling/sloughing skin
- ☐ Episodes of fast heart rate
- ☐ Chest pain
- ☐ Raynaud's syndrome
- ☐ Non-obstructive sleep apnea
- ☐ Difficulty thinking clearly
- ☐ Disorientation
- ☐ Balance issues
- ☐ Slow reflexes
- ☐ Incoordination
- ☐ Numbness or tingling
- ☐ Nerve pains
- ☐ Unexplained menstrual changes
- ☐ Overactive bladder
- ☐ Bladder infection
- ☐ React to musty spaces

TOTAL **CATEGORY 2** BOXES MARKED: _____

0-2 boxes marked = Score 0
3-5 boxes marked = Score 1
6-9 boxes marked = Score 2
10+ boxes marked = Score 3

CATEGORY 2 SCORE _____

Continue to Category 3

CHECK **ALL SYMPTOMS** EXPERIENCED IN THE **PAST 3-6 MONTHS**

CATEGORY 3

- ☐ Daily use of sinus spray, sinus prescription, or Neti pot
- ☐ Sinus surgery at any time in your life
- ☐ Chronic inflammatory response syndrome (CIRS)
- ☐ MARCoNS
- ☐ Peanut allergy
- ☐ Chronic fatigue syndrome
- ☐ Difficulty walking
- ☐ Dysautonomia
- ☐ Postural Tachycardia Syndrome (PoTS)
- ☐ Hearing loss
- ☐ Confusion
- ☐ Dementia
- ☐ Memory loss
- ☐ Tremors
- ☐ Sarcoidosis
- ☐ Asthma that's difficult to control with medication
- ☐ Idiopathic pneumonitis
- ☐ Lung scarring or nodules
- ☐ Respiratory distress
- ☐ Aspergillosis
- ☐ Arrhythmia
- ☐ Coagulation abnormalities
- ☐ Atriovenous abnormalities
- ☐ Churg Strauss Syndrome
- ☐ Histamine intolerance
- ☐ Erythema nodosum
- ☐ Eosinophilic esophagitis
- ☐ Ulcer
- ☐ Non-celiac intestinal disease
- ☐ Blood in stool
- ☐ Cyclical vomiting syndrome
- ☐ Liver pain or swelling
- ☐ Fatty liver
- ☐ Non-alcoholic steatohepatitis (NASH)
- ☐ Interstitial cystitis
- ☐ Kidney pain or swelling
- ☐ Kidney disease
- ☐ Nephritis
- ☐ Chronic pelvic pain
- ☐ Infertility
- ☐ Hepatocellular carcinoma
- ☐ Previous or current cancer diagnosis
- ☐ Mast cell activation syndrome (MCAS)
- ☐ Exposure to water-damaged building any time in your life
- ☐ Exposure to mold
- ☐ Positive Shoemaker tests

TOTAL **CATEGORY 3** BOXES MARKED: _____

Score 1 for each box marked
Boxes marked and score will
be the same for this category

CATEGORY 3 SCORE _____

Continue to Results

TOTAL MOLD RISK **RESULTS**

Gather your Category scores
from the 3 previous categories

CATEGORY 1 SCORE: _____ +
CATEGORY 2 SCORE: _____ +
CATEGORY 3 SCORE: _____ = TOTAL MOLD RISK _____

TOTAL MOLD RISK RESULTS

0-4 = Not Likely Mold Sickness
5-9 = Possible Mold Sickness
10+ = Probable Mold or Biotoxin Sickness

OTHER THINGS TO CONSIDER:

- LYME DISEASE, MSIDS, TICK-BORNE COINFECTIONS (USE HORROWITZ MSIDS-LYME QUESTIONNAIRE)
- OTHER ENVIRONMENTAL TOXINS (IE: MERCURY, LEAD, PM2.5, GLYPHOSATE, PESTICIDES, VOCs)
- INTESTINAL PARASITES, CHRONIC VIRAL SYNDROMES, OR OTHER STEALTH INFECTIONS
- FOOD SENSITIVITIES
- CVIDS OR IMMUNODEFICIENCY SYNDROMES

Diet and Foods to Avoid

- FIRST TIER Sweets of any kind; Dried fruits; Leavened bread; Yeast; Simple carbohydrate; Baked goodies; Mushrooms; Corn; Potatoes; Pickles & pickled foods; Vinegar; Soy sauce; Cantaloupe; Grapes; Aged cheeses; Moldy cheeses; Peanuts; Peanut butter

- FOODS SECOND TIER All fruit; Starchy vegetables; All grains; Fermented foods; Shelled nuts; Condiments made with vinegar or sugar; Sour cream or other soured milk products

- BEVERAGES ANY sweetened beverage; Fruit juice; Oolong and black tea (partially fermented); Moldy coffee (check your company for independent testing); Alcoholic beverages; Fermented beverages, such as cider, kombucha





- Certain supplements and medications have the potential to make a mold-sick person feel worse.
- SUPPLEMENTS | CAUSE FUNGAL OVERLOAD Saccharomyces boulardii; Nutritional yeast; Medicinal mushrooms
- GROWN ON YEAST/MOLD Aspergillus-activated brands Some B-vitamin brands
- MAY CONTAIN MYCOTOXINS Red yeast rice (use only companies that conduct independent testing); Bee propolis
- Make sure you're not taking any supplements that are contaminated with mycotoxins. Most reputable supplement companies regularly test and control for mycotoxin contamination.
- CAUTION MEDICATIONS | TWO CATEGORIES Antibiotics: many classes of antibiotics are mycotoxins. They're based on the antibacterial effect of mold mycotoxins. Use these types of antibiotics only if necessary. Try others first if an antibiotic is needed.
- Strong antifungal herbs and medications: take caution using these without prepping your body first.

Food Intolerance Test

Test 190 Foods

Includes a wide range of foods, common in the Western, Asian, and Mediterranean diets. Added a hemp allergy marker because it is very common now as a food source or as medical CBD. Able to run the IgG Food MAP on both serum and dried blood spot samples.

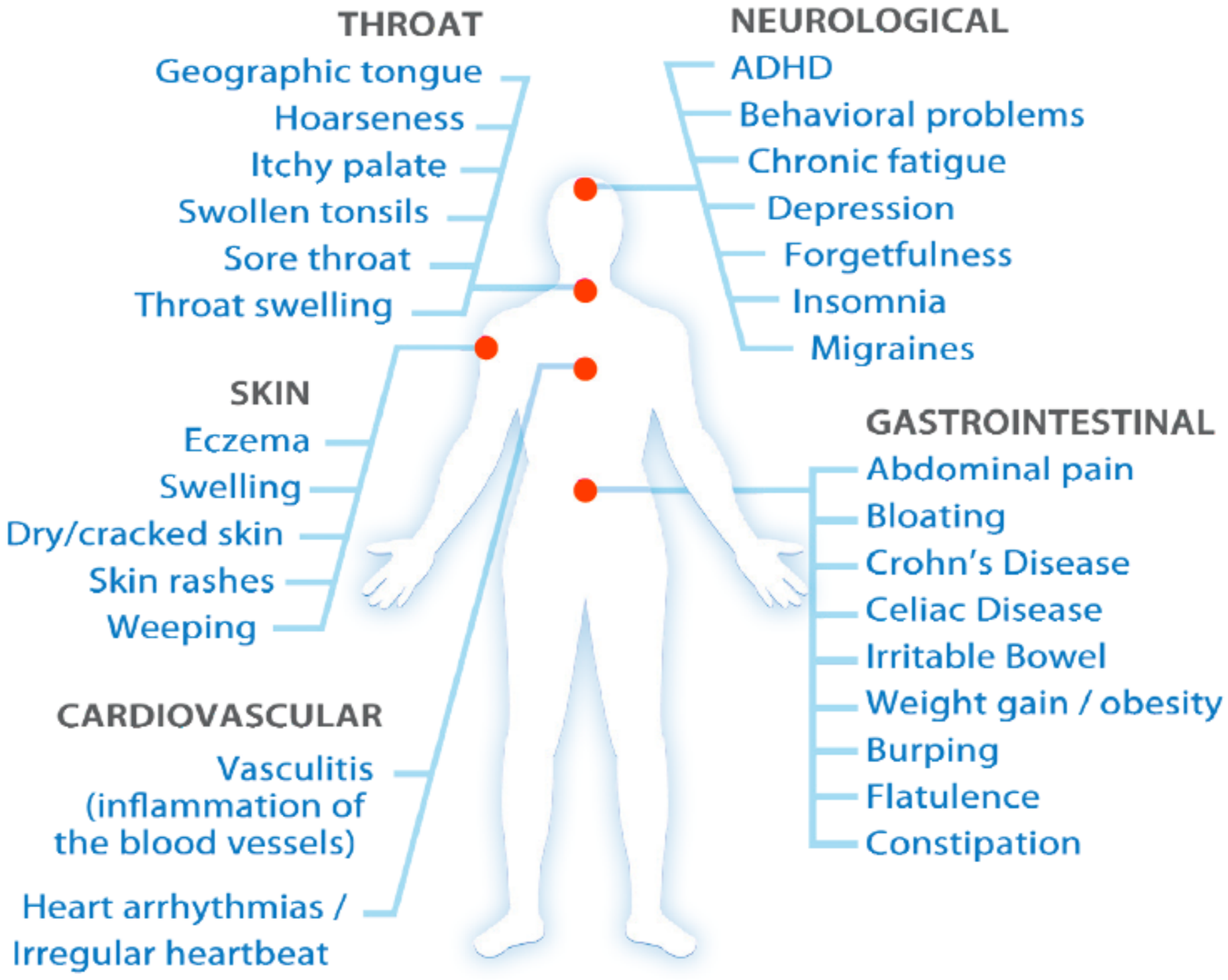
WHY TEST IGG SENSITIVITIES?

Immunoglobulin G (IgG) food testing is a useful guide for structuring elimination diets for people with many chronic conditions. Individuals with neurological, gastrointestinal, movement, and behavioral disorders often suffer from IgG food sensitivities. People may continue to eat offending foods unaware of their potential adverse effects. Symptoms associated with food sensitivities may occur hours or days after the offending food was eaten because IgG food antibodies remain for a much longer time than IgE antibodies.

IgE food allergy causes the release of histamine, producing an immediate hypersensitivity reaction. In contrast, IgG food sensitivity is triggered by the binding of complement to IgG food antigen complexes, causing an inflammatory response. This is a delayed hypersensitivity reaction in which symptoms appear anywhere from hours to days after eating the offending food. Elimination of IgG-positive foods may improve symptoms of irritable bowel syndrome, autism, AD(H)D, cystic fibrosis, rheumatoid arthritis, and epilepsy, according to numerous clinical studies.

The most common IgG food allergies were to cow’s milk, garlic, mustard, egg yolk, tea, and chocolate. A recent study reported that 93% of non-celiac, gluten-sensitive patients showed anti-gliadin IgG antibody disappearance after a six-month adherence to a gluten-free diet. The IgG disappearance was closely related to a significant improvement of both gastrointestinal and extra-intestinal symptoms.

Parts of Body Impacted by Food Sensitivities



Food Intolerance Test

SIGNIFICANCE OF TOTAL IGG VERSUS IGG4

The goal of IgG-mediated food allergy testing is to identify foods that are capable of triggering many adverse reactions. IgG1, IgG2, and IgG3 are all capable of causing inflammation. IgG1, IgG2, and IgG3 antibodies to food antigens form large immune complexes or lattices that activate complement proteins and increase inflammation. IgG4 antibodies to food antigens will not usually trigger inflammation because IgG4 antibodies do not bind complement. However high levels of these antibodies indicate the presence of immune reactions against food antigens. Similarly IgA antibodies are not as clinically significant due to their inability to bind complement and trigger inflammation. Most laboratories only provide a measurement of IgG4 molecules.

Research has revealed a relationship between health of the gastrointestinal tract and overall well-being. Imbalance between good and bad bacteria (dysbiosis) has been shown to worsen behavior disorders, hyperactivity, aggression, and cause fatigue. Evidence is mounting that dysbiosis impairs the immune system and contributes to food allergies, digestive disorders, nutritional deficiencies, and cognitive dysfunction.

Food Intolerance Test

THE BENEFITS OF TESTING

- IgG testing determines if food reactions are contributing to physical or mental symptoms, and much more quickly than eliminating and then reintroducing each suspect food one-by-one over a period of time.
- Removal of highly reactive foods from the diet is a non-invasive therapy that often mitigates symptoms.
- Research and clinical studies suggest food allergies identified by IgG testing can be major contributing factors to many chronic health conditions.
- Eliminating all identified IgG-positive foods after testing can reduce stress on the immune system, decrease inflammation (helping to heal "leaky gut"), resolve food cravings, and reduce the potential for eating disorders.

Dried Blood Spot (DBS): Four full circles of dried blood on the protein saver card is required.

Please note that the elimination diet is based only on IgG testing. To be safe, testing for IgE antibodies to food allergens should be considered PRIOR TO BEGINNING an ELIMINATION DIET. If there are negative results for IgG antibodies, it does not necessarily mean the individual is negative for IgE, IgM, or IgA antibodies.

Dairy

Beta-Lactoglobulin	10.04
Casein	28.29
Cheddar Cheese	19.79
Cow's Milk	20.78
Goat's Milk	4.07
Mozzarella Cheese	16.28
Sheep's Yogurt	2.69
Whey	19.83
Yogurt	27.06

Beans and Peas

Adzuki Bean	8.18
Black Bean	8.47
Garbanzo Bean	5.65
Green Bean	10.21
Green Pea	4.78
Kidney Bean	8.39
Lentil	7.63
Lima Bean	3.47
Mung Bean	12.26
Navy Bean	8.40
Pinto Bean	6.87
Soybean	13.44
Tofu	10.09

Fruits

Acai Berry	5.92
Apple	1.62
Apricot	17.02
Banana	5.23
Blueberry	1.03
Cantaloupe	2.32
Cherry	27.56
Coconut	11.56
Cranberry	1.10
Date	8.42
Fig	15.21
Grape	7.64
Grapefruit	15.12
Guava	2.55
Jackfruit	17.27
Kiwi	4.33
Lemon	10.10
Lychee	2.70
Mango	4.58
Orange	8.41
Papaya	19.20
Passion Fruit	1.79
Peach	13.04
Pear	2.35
Pineapple	9.13
Plum	2.91
Pomegranate	1.27
Raspberry	5.21
Strawberry	1.52
Watermelon	3.44

Grains

Amaranth	6.64
Barley	2.97
Buckwheat	2.39
Corn	6.83
Gliadin	26.82
Malt	1.23
Millet	8.31
Oat	6.23
Quinoa	3.41

Grains Continued

Rice	9.98
Rye	9.33
Sorghum	7.67
Teff	5.45
Wheat Gluten	31.96
Whole Wheat	26.14

Fish/Seafood

Abalone	0.67
Anchovy	0.24
Bass	0.61
Bonito	1.27
Codfish	0.91
Crab	0.38
Halibut	0.32
Jack Mackerel	1.02
Lobster	0.37
Octopus	0.63
Oyster	0.52
Pacific Mackerel (Saba)	3.03
Pacific Saury	0.33
Perch	0.91
Red Snapper	1.27
Salmon	0.81
Sardine	0.04
Scallop	0.60
Shrimp	0.60
Small Clam	0.60
Squid	0.68
Tilapia	0.73
Trout	0.74
Tuna	3.30

Meat/Fowl

Beef	1.06
Chicken	0.27
Duck	0.89
Egg White	47.59
Egg Yolk	24.29
Goose	1.90
Lamb	0.88
Pork	0.64
Turkey	0.84

Nuts/Seeds

Almond	27.96
Brazil Nut	1.62
Cashew	5.81
Chestnut	3.28
Chia Seed	2.24
Flax Seed	7.14
Hazelnut	14.36
Hemp Seed	4.20
Macadamia Nut	7.43
Peanut	12.89
Pecan	0.82
Pine Nut	5.57
Pistachio	9.85
Pumpkin Seed	5.92
Sesame Seed	4.79
Sunflower Seed	13.80
Walnut	9.20

Vegetables

Artichoke	4.16
Asparagus	6.17
Avocado	13.92

Vegetables Continued

Bamboo Shoot	2.18
Bean Sprout	8.41
Beet	3.80
Bell Pepper	21.93
Bitter Gourd	8.15
Broccoli	17.13
Brussel Sprout	19.38
Burdock Root	2.14
Cabbage	43.96
Carrot	6.22
Cauliflower	18.39
Celery	17.75
Chili Pepper	36.89
Cucumber	12.05
Eggplant	9.46
Enoki Mushroom	0.28
Garlic	15.71
Kale	11.97
Leek	9.60
Lettuce	6.33
Lotus Root	1.25
Napa Cabbage	6.57
Olive (Green)	0.28
Onion	10.57
Portabella Mushroom	0.71
Potato	19.01
Pumpkin	14.56
Radish	15.76
Seaweed Kombu Kelp	0.26
Seaweed Nori	0.37
Seaweed Wakame	0.28
Shitake Mushroom	0.34
Spinach	4.35
Sweet Potato	8.62
Tomato	16.52
Yam	4.66
Yellow Squash	18.84
Yuca	0.00
Zucchini	5.46

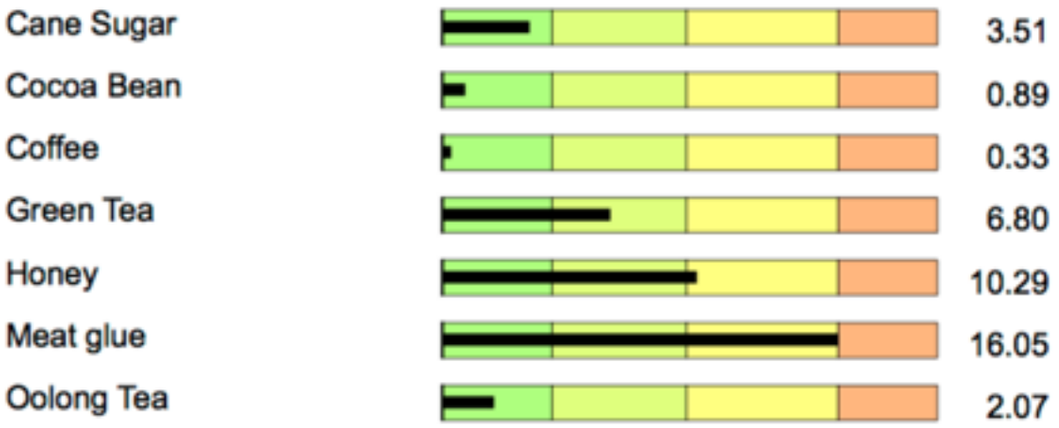
Herbs/Spices

Basil	2.54
Bay Leaf	0.56
Black Pepper	2.97
Cayenne Pepper	10.42
Cilantro	0.97
Cinnamon	1.29
Cloves	2.46
Cumin	11.01
Curry	5.52
Dill	1.59
Ginger	9.13
Hops	4.56
Mint	0.64
Miso	8.91
Mustard Seed	34.01
Oregano	0.27
Paprika	20.00
Rosemary	2.76
Sage	2.67
Tarragon	2.17
Thyme	3.47
Turmeric	0.88
Vanilla Bean	5.21

Miscellaneous

Bromelain	22.50

Miscellaneous — Continued



Food Reactivity Scale	MFI* x 1000
Not Significant	< 4.47
Low	4.47-9.86
Moderate	9.87-15.99
High	>=16

(*) Median Fluorescent Intensity

Reactivity Summary

High

Almond	Apricot	Bell Pepper
Broccoli	Bromelain	Brussel Sprout
Cabbage	Casein	Cauliflower
Celery	Cheddar Cheese	Cherry
Chili Pepper	Cow's Milk	Egg White
Egg Yolk	Gliadin	Jackfruit
Meat glue	Mozzarella Cheese	Mustard Seed
Papaya	Paprika	Potato
Tomato	Wheat Gluten	Whey
Whole Wheat	Yellow Squash	Yogurt

Moderate

Avocado	Beta-Lactoglobulin	Cayenne Pepper
Coconut	Cucumber	Cumin
Fig	Garlic	Grapefruit
Green Bean	Hazelnut	Honey
Kale	Lemon	Mung Bean
Onion	Peach	Peanut
Pumpkin	Radish	Rice
Soybean	Sunflower Seed	Tofu

Low

Acai Berry	Adzuki Bean	Amaranth
Asparagus	Banana	Bean Sprout
Bitter Gourd	Black Bean	Carrot
Cashew	Corn	Curry
Date	Eggplant	Flax Seed
Garbanzo Bean	Ginger	Grape
Green Pea	Green Tea	Hops
Kidney Bean	Leek	Lentil
Lettuce	Macadamia Nut	Mango
Millet	Miso	Napa Cabbage
Navy Bean	Oat	Orange
Pine Nut	Pineapple	Pinto Bean
Pistachio	Pumpkin Seed	Raspberry
Rye	Sesame Seed	Sorghum
Sweet Potato	Teff	Vanilla Bean
Walnut	Yam	Zucchini

Yeast		3.00
Candida Albicans		8.00

Reactivity Summary

Moderate
- Candida Albicans

Low
- Yeast

Not Significant	1.00 - 1.99
Low	2.00 - 3.49
Moderate	3.50 - 4.99
High	>= 5.00

Yeast Saccharomyces Cerevisiae Scale

Not Significant	< 3.49
Low	3.50 - 6.99
Moderate	7.00 - 14.99
High	>= 15.00

Candida Scale

The Candida albicans scale accounts for the observation that background levels of Candida-specific immunoglobulins are normally present in virtually all individuals tested. It is intended to provide a clearer description of its clinical significance and was established according to population percentile ranks obtained from a random subset of 1,000 patients.

Comprehensive Stool Test

- Parameters for digestion & absorption
- Cultures for bacteria and yeast
- Parasite testing
- Sensitivity panels
- Inflammatory markers
- Stool metabolic markers
- Infectious pathogens

THE BENEFITS OF COMPREHENSIVE STOOL ANALYSIS:

- The amount of beneficial bacteria in the GI tract is determined
- The digestive parameters aid in the diagnosis of intestinal dysfunction without invasive procedures
- Inflammation and immune markers will aid in appropriate treatments
- Many different ***pathogens*** have the potential to be isolated and identified

SPECIMEN REQUIREMENTS:

Stool: Sample must be collected on two separate days (at least 12 hours apart). The specimen must be received within 7 days of the first collection. The patient must discontinue digestive enzymes, antacids, iron supplements, vitamin C over 250 mg, aspirin, anti-inflammatories, and large amounts of meat 48 hours prior to the collection of the specimen.

The Biotoxin Pathway

Body acquires biotoxins or toxin-producing organisms from food, water, air, or insect bites

Biotoxin (HLA susceptible)

In genetically susceptible people biotoxin binds to fat cell receptors causing continuing unregulated production of cytokines

Surface ("Toll") receptor

Fat Cell

Capillaries

High cytokine levels in the capillaries attract white blood cells, leading to restricted blood flow and lower oxygen levels Reduced VEGF leads to fatigue muscle cramps and shortness of breath (may be overridden by replacement with erythropoietin

Increased Cytokines

Immune system symptoms

Patients with certain HLA genotypes (immunity related genes) may develop inappropriate immunity Most common are antibodies to
-- Myelin basic protein (often from fungal biotoxins, affects nervous system functions)
-- Gliadin (affects digestion)
-- Cardiolipins (affects blood clotting)
The "complement" alternative immune pathway may be triggered (detectable as an increase in levels of the proteins C3a C4a).

Biotoxin (HLA susceptible)

Nerve cell

Increased Cytokines

Increased Leptin

Fat cells then produce more leptin, leading to obesity (which doesn't respond to exercise and diet)

Excessive cytokine levels can damage leptin receptors in the hypothalamus

Leptin receptor

Hypothalamus

VIP

AVP

Damaged leptin receptors lead to reduced production by the hypothalamus of MSH, a hormone with many functions

Cytokine-related symptoms

High levels of cytokines produce flu-like symptoms Headaches, muscle aches, fatigue unstable temperature, difficulty concentrating High levels of cytokines also result in increased levels of several other immune response related substances, including TNF, MMP9, IL-1B, and PAI-1. MMP9 delivers inflammatory elements from blood to brain, nerve, muscle, lungs, and joints It combines with PAI in increasing clot formation and arterial blockage

Biotoxins have direct effects, including impairment of nerve cell function One result is poor performance on contrast sensitivity test

Biotoxin (not HLA susceptible)

Removal from the body

In most people, biotoxins are either removed from the blood by the liver or attacked by the immune system, broken down and excreted harmlessly In people who don't have the right immune system genes, however, biotoxins can remain in the body indefinitely

Reduced MSH

Sleep disturbance

Production of melatonin is reduced leading to light, non-restorative sleep

Chronic pain

Endorphin production is suppressed This can lead to chronic, sometimes unusual, pain

Gastrointestinal problems

Lack of MSH can cause malabsorption in the gut, resulting in diarrhea This is sometimes called "leaky gut" and resembles (but is not) celiac disease Patients must avoid gluten, whey, and amylose

Prolonged illness

White blood cells lose regulation of cytokine response, so that recovery from other illnesses, including infectious diseases, may be slowed

Resistant Staph bacteria

Colonies of Staph bacteria with resistance to multiple antibiotics may develop in mucous membranes The bacteria produce substances that aggravate both the high cytokine (Patients should avoid steroids such as prednisone, which can lower levels of ACTH) and low MSH levels

Changes in cortisol and ACTH levels

The pituitary may produce elevated levels of cortisol and ACTH in early stages of illness, then drop to excessively low levels later

Reduced ADH

Reduced MSH can cause the pituitary to produce lower levels of antidiuretic hormone (ADH), leading to thirst, frequent urination, and susceptibility to shocks from static electricity

Reduced sex hormones

Reduced MSH can cause the pituitary to lower its production of sex hormones

Stage1: Biotoxin effects

Stage2: Cytokine effects

Stage3: Reduced VEGF

Stage4: Immune effects

Stage5: Low MSH

Stage6: Resistant Staph bacteria

Stage7: Pituitary hormone effects

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Comprehensive Stool Analysis / Parasitology x2

BACTERIOLOGY CULTURE

Expected/Beneficial flora	Commensal (Imbalanced) flora	Dysbiotic flora
4+ *Bacteroides fragilis* group	3+ Alpha hemolytic strep	
1+ *Bifidobacterium* spp.	1+ Beta hemolytic strep, not group A or B	
3+ *Escherichia coli*	3+ Gamma hemolytic strep	
NG *Lactobacillus* spp.	1+ *Pseudomonas chlororaphis* group	
NG *Enterococcus* spp.		
4+ *Clostridium* spp.		

NG = No Growth

BACTERIA INFORMATION

Expected / Beneficial bacteria make up a significant portion of the total microflora in a healthy & balanced GI tract. These beneficial bacteria have many health-protecting effects in the GI tract including manufacturing vitamins, fermenting fibers, digesting proteins and carbohydrates, and propagating anti-tumor and anti-inflammatory factors.

Clostridia are prevalent flora in a healthy intestine. Clostridium spp. should be considered in the context of balance with other expected/beneficial flora. Absence of clostridia or over abundance relative to other expected/beneficial flora indicates bacterial imbalance. If C. difficile associated disease is suspected, a Comprehensive Clostridium culture or toxigenic C. difficile DNA test is recommended.

Commensal (Imbalanced) bacteria are usually neither pathogenic nor beneficial to the host GI tract. Imbalances can occur when there are insufficient levels of beneficial bacteria and increased levels of commensal bacteria. Certain commensal bacteria are reported as dysbiotic at higher levels.

Sample Report

Dysbiotic bacteria consist of known pathogenic bacteria and those that have the potential to cause disease in the GI tract. They can be present due to a number of factors including: consumption of contaminated water or food, exposure to chemicals that are toxic to beneficial bacteria; the use of antibiotics, oral contraceptives or other medications; poor fiber intake and high stress levels.

YEAST CULTURE

Normal flora	Dysbiotic flora
1+ *Candida albicans*	
1+ *Geotrichum* spp.	

MICROSCOPIC YEAST

RESULT	EXPECTED
None	None – Rare

Yeast in stool is expected at a level of none-rare. A microscopic finding of yeast in stool of few, moderate, or many may be helpful in identifying potential yeast overgrowth, or non-viable or dietary yeast.

YEAST INFORMATION

Yeast may normally be present in small quantities in the skin, mouth, and intestine. When investigating the presence of yeast, disparity may exist between culturing and microscopic examination. Yeast are not uniformly dispersed throughout the stool and this may lead to undetectable or low levels of yeast identified by microscopy, despite culture and identified yeast species. Conversely, microscopic examination may reveal a significant amount of yeast present but no viable yeast cultured. Yeast may not always survive transit through the intestines. Nonviable diet-derived yeast may also be detected microscopically. Consideration of clinical intervention for yeast detected microscopically should be made in the context of other findings and presentation of symptoms.

Protozoa	PX1	PX2
Balantidium coli	Not Detected	Not Detected
Blastocystis spp.	Moderate	Moderate
Chilomastix mesnili	Not Detected	Not Detected
Dientamoeba fragilis	Not Detected	Not Detected
Endolimax nana	Rare Cysts/Trophs	Few Cysts/Trophs
Entamoeba coli	Not Detected	Not Detected
Entamoeba hartmanni	Not Detected	Not Detected
Entamoeba histolytica/Entamoeba dispar	Not Detected	Not Detected
Entamoeba polecki	Not Detected	Not Detected
Enteromonas hominis	Not Detected	Not Detected
Giardia duodenalis	Not Detected	Not Detected
Iodamoeba bütschlii	Not Detected	Not Detected
Isospora belli	Not Detected	Not Detected
Pentatrichomonas hominis	Not Detected	Not Detected
Retortamonas intestinalis	Not Detected	Not Detected
Nematodes - Roundworms		
Ascaris lumbricoides	Not Detected	Not Detected
Capillaria hepatica	Not Detected	Not Detected
Capillaria philippinensis	Not Detected	Not Detected
Enterobius vermicularis	Not Detected	Not Detected
Strongyloides stercoralis	Not Detected	Not Detected
Trichuris trichiura	Not Detected	Not Detected
Hookworm	Not Detected	Not Detected
Cestodes - Tapeworms		
Diphyllobothrium latum	Not Detected	Not Detected
Dipylidium caninum	Not Detected	Not Detected
Hymenolepis diminuta	Not Detected	Not Detected
Hymenolepis nana	Not Detected	Not Detected
Taenia	Not Detected	Not Detected
Trematodes - Flukes		
Clonorchis sinensis	Not Detected	Not Detected
Fasciola hepatica/Fasciolopsis buski	Not Detected	Not Detected
Heterophyes heterophyes	Not Detected	Not Detected
Paragonimus westermani	Not Detected	Not Detected
Other Markers		
Yeast	Not Detected	Not Detected
RBC	Not Detected	Rare
WBC	Not Detected	Not Detected
Charcot-Leyden Crystals	Not Detected	Not Detected
Pollen	Not Detected	Not Detected
Immunoassay	**RESULT**	**REFERENCE INTERVAL**
Giardia duodenalis	Negative	Negative
Cryptosporidium	Negative	Negative

Sample Report

Intestinal parasites are abnormal inhabitants of the gastrointestinal tract that have the potential to cause damage to their host. The presence of any parasite within the intestine generally confirms that the patient has acquired the organism through fecal-oral contamination. Damage to the host includes parasitic burden, migration, blockage and pressure. Immunologic inflammation, hypersensitivity reactions and cytotoxicity also play a large role in the morbidity of these diseases. The infective dose often relates to severity of the disease and repeat encounters can be additive.

In general, acute manifestations of parasitic infection may involve diarrhea with or without mucus and or blood, fever, nausea, or abdominal pain. However these symptoms do not always occur. Consequently, parasitic infections may not be diagnosed or eradicated. If left untreated, chronic parasitic infections can cause damage to the intestinal lining and can be an unsuspected cause of illness and fatigue. Chronic parasitic infections can also be associated with increased intestinal permeability, irritable bowel syndrome, irregular bowel movements, malabsorption, gastritis or indigestion, skin disorders, joint pain, allergic reactions, and decreased immune function.

One negative parasitology x1 specimen does not rule out the possibility of parasitic disease, parasitology x3 is recommended. This test is not designed to detect Cyclospora cayetanensis or Microsporidia spp.

DIGESTION / ABSORPTION

	WITHIN	OUTSIDE	REFERENCE INTERVAL
Elastase	233		> 200 µg/mL
Fat Stain	None		None – Few
Muscle fibers	None		None – Rare
Vegetable fibers	Rare		None – Few
Carbohydrates†	Negative		Negative

Elastase findings can be used for the diagnosis or the exclusion of exocrine pancreatic insufficiency. Correlations between low levels and chronic pancreatitis and cancer have been reported.
Fat Stain: Microscopic determination of fecal fat using Sudan IV staining is a qualitative procedure utilized to assess fat absorption and to detect steatorrhea.
Muscle fibers in the stool are an indicator of incomplete digestion. Bloating, flatulence, feelings of "fullness" may be associated with increase in muscle fibers.
Vegetable fibers in the stool may be indicative of inadequate chewing, or eating "on the run".
Carbohydrates: The presence of reducing substances in stool specimens can indicate carbohydrate malabsorption.

INFLAMMATION

Sample Report

	WITHIN	OUTSIDE	REFERENCE INTERVAL
Lactoferrin	<0.[illegible]		≤ 7.[illegible] µg/mL
Calprotectin	<10		≤ 50 µg/g
Lysozyme*	147		≤ 500 ng/mL
White Blood Cells	None		None – Rare
Mucus	Negative		Negative

Lactoferrin and **Calprotectin** are reliable markers for differentiating organic inflammation (IBD) from functional symptoms (IBS) and for management of IBD. Monitoring levels of fecal lactoferrin and calprotectin can play an essential role in determining the effectiveness of therapy, are good predictors of IBD remission, and can indicate a low risk of relapse.
Lysozyme is an enzyme secreted at the site of inflammation in the GI tract and elevated levels have been identified in IBD patients.
White Blood Cells (WBC) and **Mucus** in the stool can occur with bacterial and parasitic infections, with mucosal irritation, and inflammatory bowel diseases such as Crohn's disease or ulcerative colitis

IMMUNOLOGY

	WITHIN	OUTSIDE	REFERENCE INTERVAL
Secretory IgA*		10.1	30 – 275 mg/dL

Secretory IgA (sIgA) is secreted by mucosal tissue and represents the first line of defense of the GI mucosa and is central to the normal function of the GI tract as an immune barrier. Elevated levels of sIgA have been associated with an upregulated immune response.

Comprehensive Stool Analysis / Parasitology x2

SHORT CHAIN FATTY ACIDS

	WITHIN	OUTSIDE	REFERENCE INTERVAL
% Acetate‡	52		50 – 72 %
% Propionate‡	23		11 – 25 %
% Butyrate‡	20		11 – 32 %
% Valerate‡	4.4		0.8 – 5.0 %
Butyrate‡	0.85		0.8 – 4.0 mg/mL
Total SCFA's‡		4.2	5.0 – 16.0 mg/mL

Short chain fatty acids (SCFAs): SCFAs are the end product of the bacterial fermentation process of dietary fiber by beneficial flora in the gut and play an important role in the health of the GI as well as protecting against intestinal dysbiosis. Lactobacilli and bifidobacteria produce large amounts of short chain fatty acids, which decrease the pH of the intestines and therefore make the environment unsuitable for pathogens, including bacteria and yeast. Studies have shown that SCFAs have numerous implications in maintaining gut physiology. SCFAs decrease inflammation, stimulate healing, and contribute to normal cell metabolism and differentiation. Levels of **Butyrate** and **Total SCFA** in mg/mL are important for assessing overall SCFA production, and are reflective of beneficial flora levels and/or adequate fiber intake.

INTESTINAL HEALTH MARKERS

Sample Report

	WITHIN	OUTSIDE	REFERENCE INTERVAL
Red Blood Cells	Rare		None – Rare
pH	6.7		5.8 – 7.0
Occult Blood	Negative		Negative

Red Blood Cells (RBC) in the stool may be associated with a parasitic or bacterial infection, or an inflammatory bowel condition such as ulcerative colitis. Colorectal cancer, anal fistulas, and hemorrhoids should also be ruled out.
pH: Fecal pH is largely dependent on the fermentation of fiber by the beneficial flora of the gut.
Occult blood: A positive occult blood indicates the presence of free hemoglobin found in the stool, which is released when red blood cells are lysed.

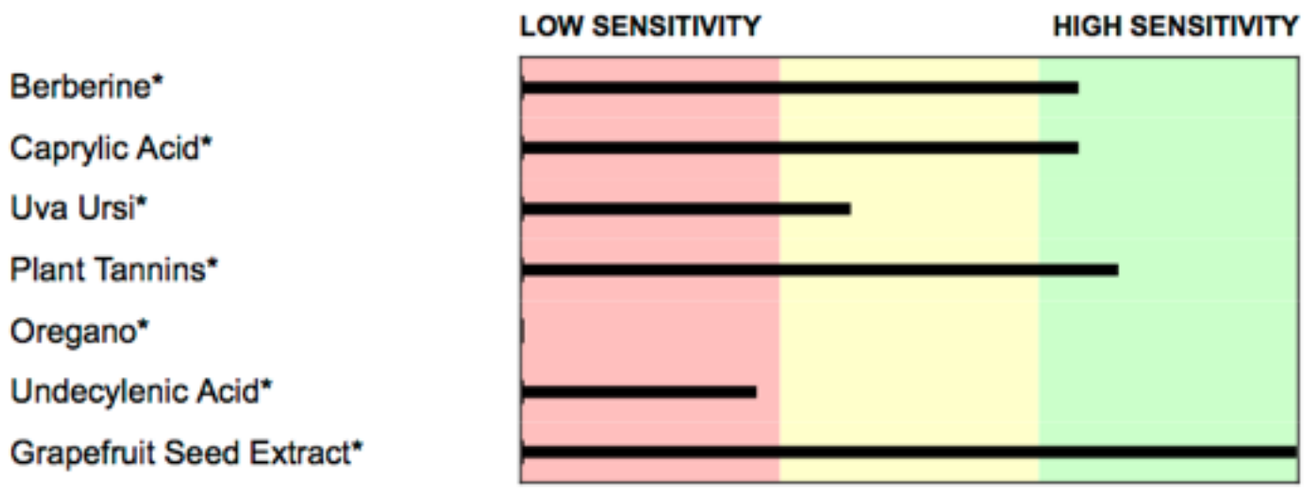
MACROSCOPIC APPEARANCE

	WITHIN	OUTSIDE	EXPECTED
Color	Brown		Brown
Consistency		Loose/Watery	Soft

Color: Stool is normally brown because of pigments formed by bacteria acting on bile introduced into the digestive system from the liver. While certain conditions can cause changes in stool color, many changes are harmless and are caused by pigments in foods or dietary supplements.
Consistency: Stool normally contains about 75% water and ideally should be formed and soft. Stool consistency can vary based upon transit time and water absorption.

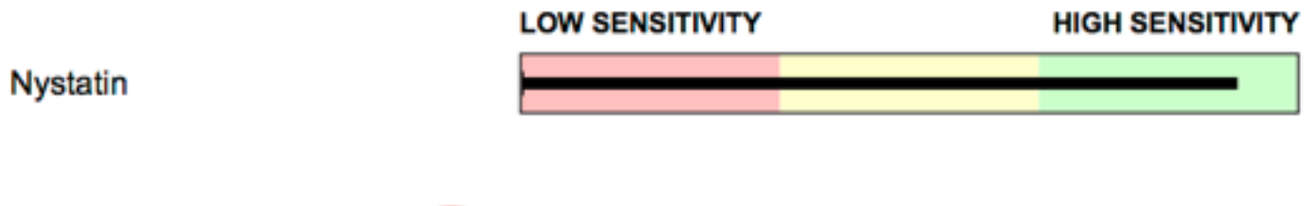
Yeast Susceptibilities; Candida albicans

NATURAL ANTIFUNGALS



Natural antifungal agents may be useful for treatment of patients when organisms display in-vitro susceptibility to these agents. The test is performed by using standardized techniques and filter paper disks impregnated with the listed agent. Relative activity is reported for each natural agent based upon the diameter of the zone of inhibition or no growth zone surrounding the disk. Data based on over 5000 individual observations were used to relate the zone size to the activity level of the agent. A scale of relative activity is defined for the natural agents tested.

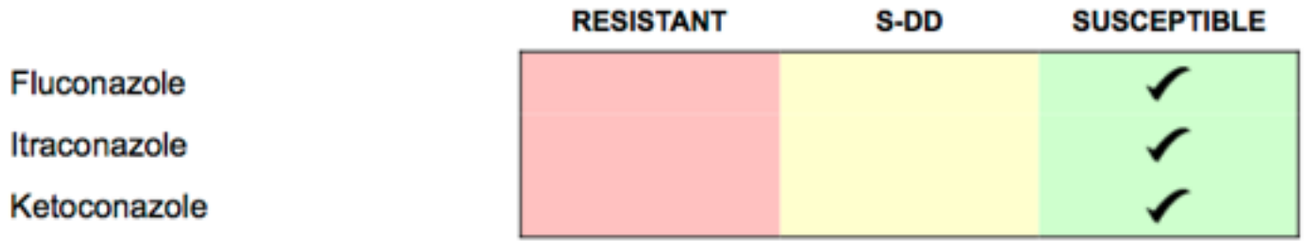
NON-ABSORBED ANTIFUNGALS



Non-absorbed antifungals may be useful for treatment of patients when organisms display in-vitro susceptibility to these agents. The test is performed using standardized commercially prepared disks impregnated with Nystatin. Relative activity is reported based upon the diameter of the zone of inhibition or no growth zone surrounding the disk.

Sample Report

AZOLE ANTIFUNGALS

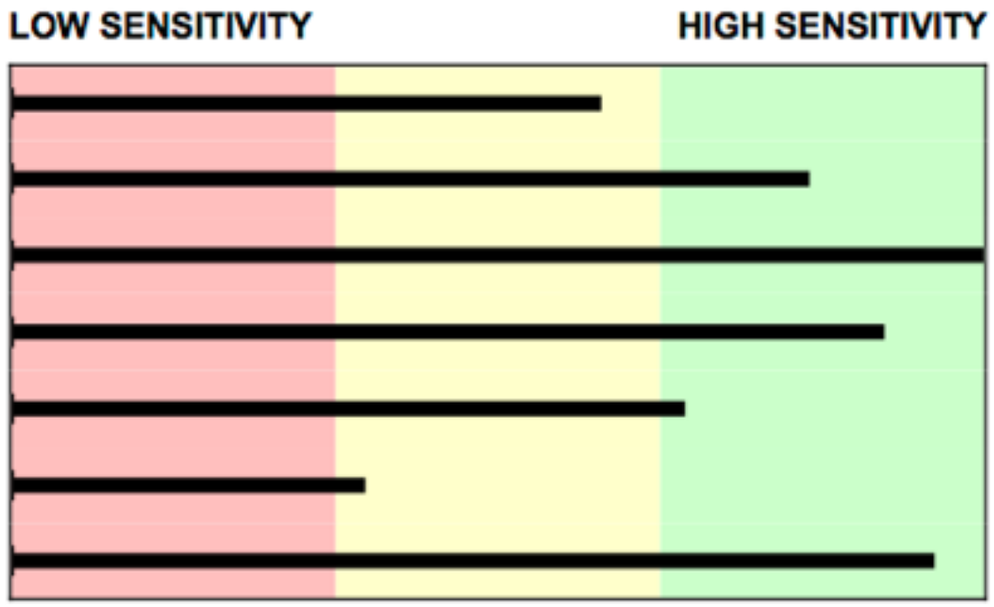


Susceptible results imply that an infection due to the fungus may be appropriately treated when the recommended dosage of the tested antifungal agent is used. **Susceptible - Dose Dependent (S-DD)** results imply that an infection due to the fungus may be treated when the highest recommended dosage of the tested antifungal agent is used. **Resistant** results imply that the fungus will not be inhibited by normal dosage levels of the tested antifungal agent.

Standardized test interpretive categories established for *Candida* spp. are used for all yeast isolates.

Yeast Susceptibilities; Geotrichum spp.

NATURAL ANTIFUNGALS



Natural antifungal agents may be useful for treatment of patients when organisms display in-vitro susceptibility to these agents. The test is performed by using standardized techniques and filter paper disks impregnated with the listed agent. Relative activity is reported for each natural agent based upon the diameter of the zone of inhibition or no growth zone surrounding the disk. Data based on over 5000 individual observations were used to relate the zone size to the activity level of the agent. A scale of relative activity is defined for the natural agents tested.

NON-ABSORBED ANTIFUNGALS



Non-absorbed antifungals may be useful for treatment of patients when organisms display in-vitro susceptibility to these agents. The test is performed using standardized commercially prepared disks impregnated with Nystatin. Relative activity is reported based upon the diameter of the zone of inhibition or no growth zone surrounding the disk.

Sample Report

Cholesterol Test

The concept of "good" and "bad" cholesterol depends highly on the circumstances of the individual person. Although very high blood serum cholesterol values are associated with heart disease, ***low values are associated*** with increased violent behavior, suicide, depression, anxiety, bipolar disease, Parkinson's disease, and increased mortality from cancer.

Low cholesterol values are also associated with manganese deficiency, celiac disease, hyperthyroidism, liver disease, malabsorption, and malnutrition.

Advanced Cholesterol Profile

GENERAL

The concept of "good" and "bad" cholesterol depends highly on the circumstances of the individual person. Although very high blood serum cholesterol values are associated with heart disease, low values are associated with increased violent behavior, suicide, depression, anxiety, bipolar disease, Parkinson's disease, and increased mortality from cancer. Low cholesterol values are also associated with manganese deficiency, celiac disease, hyperthyroidism, liver disease, malabsorption, and malnutrition.



Total Cholesterol

Patient Value mg/dL	Reference Range mg/dL	High/Low Flag
213.00	160.00 - 200.00	H

Total Cholesterol Interpretation

SAMPLE REPORT

High and low values are flagged based on clinical studies that generally indicate significant health impairment with cholesterol values below 160 mg/dL or greater than 200 mg/dL. Values between 160-200 mg/dL are flagged as optimal. Population based reference ranges are given below.

Cholesterol is an essential fat needed for production of steroid hormones and bile salts. In addition, cholesterol is needed for the function of cell membranes and is especially important for myelin, the insulating material for the nerves in the peripheral nervous system and the brain. Cholesterol is needed to maintain serotonergic brain function and helps to modulate GABA (gamma-aminobutyric acid) receptors. In addition, cholesterol is an essential activating factor for the developmental protein called sonic hedgehog. Since there are mixed benefits from both high and low cholesterol, all related factors should be considered in the interpretation of results. All values should be examined in light of the patient's history, not only those flagged as high or low.

Age -years	Males 5 percentile	Males 95 percentile	Females 5 percentile	Females 95 percentile
0-4	114	203	112	200
5-9	121	203	126	205
10-14	119	202	124	201
15-19	113	197	119	200
20-24	124	218	122	216
25-29	133	244	128	222
30-34	138	254	130	230
35-39	146	270	140	242
40-44	151	268	147	252
45-49	158	276	152	265
50-54	158	277	162	285
55-59	156	276	172	300
60-64	159	276	172	297
65-69	158	274	171	303
>70	144	265	173	280

Population based reference ranges for normal people. These values are not used for interpretive range.

Apolipoprotein A-I and Apolipoprotein B

Apolipoprotein A-I Result	g/L	1.37

Reference Range		Result
< 1.25	Low	Normal
1.25-2.15	Normal Range	
> 2.15	High	

SAMPLE REPORT

Apolipoprotein B Result	g/L	1.11

Reference Range		Result
< 0.55	Low	Normal
0.55-1.25	Normal Range	
> 1.25	High	

Apolipoprotein A-I

Apolipoprotein A-I (Apo A-I) is the main protein component of HDL (high density lipoprotein) and accounts for approx 65% of the total protein content of HDL. Apo A-I activates lecithin cholesterol acyltransferase (LCAT) which catalyses the esterification of cholesterol. The resulting esterified cholesterol can then be transported to the liver, metabolized and excreted. Persons with atherosclerotic vascular changes frequently exhibit decreased levels of Apo A-I. Even if the concentrations of Apo B are normal, a decreased Apo A-I level may be a risk factor for atherosclerotic processes. Decreased concentrations of Apo A-I also occur in dyslipoproteinaemias, acute hepatitis, hepatic cirrhosis and in insulin-treated diabetics. Values of apolipoprotein A-I have been shown to decrease during infection.

Lipoprotein (a)

Patient Value		Reference Range
g/L		g/L
0.17	<	2.50

Lp(a) consists of two components, the low-density lipoprotein (LDL) and a glycoprotein, the apolipoprotein(a), which are linked by a disulfide bridge. Lp(a) is not related to apolipoprotein A. The individual concentration of Lp(a) in the serum depends primarily on genetic factors; the range of variation in a population is relatively large. Elevated concentrations of Lp(a) are a risk factor for coronary heart disease (CHD). Lp(a) does not change much over time so values in childhood are predictive of adult values. If Lp(a) values are abnormally elevated, the person needs to focus on changing other risk factors that can be modified by diet and lifestyle changes. Mechanisms of pathogenicity associated with high levels include enhanced blood clot formation and impaired clearance of fibrin. Stable lifelong values of Lp(a) are attained by age two. High epsilon 4 gene allele dose of apolipoprotein E correlated with low lipoprotein (a) levels in autism. High dose of this allele is associated with accelerated brain atrophy, Alzheimer's disease, and coronary artery disease.

SAMPLE REPORT

Values in higher than 2.50-3.00 mg/L in Caucasians are associated with coronary artery blockage. Values of Lp(a) are higher with blockage of all 3 coronary arteries than with blockage of a single coronary artery. Plasma fibrinogen concentration correlated positively and significantly with serum lipoprotein (a) levels in male nonsmokers without cardiovascular disease and in female nonsmokers with cardiovascular disease. In addition, racial differences in Lp(a) values are substantial. Whereas levels of Lp(a) above 3.0 g/L are associated with increased coronary artery disease in Caucasians, no such relationship has been found in black populations. Values are flagged as high when values exceed 2.5 g/L. The tables should be used as a guideline based on the appropriate ethnic group.

Adult Values Lp(a)	Lp(a) g/L range 2.5% - 97.5%	
Group	Males	Females
Caucasian	< 0.02 - 0.74	< 0.02 - 0.72
African-American	0.04 - 1.14	0.02 - 1.08
Hispanic	0.02 - 0.53	0.02 - 0.46

Children's values Lp(a) from USA National Health and Nutrition Examination Survey (NHANES)

Ethnic Group	Age Range	5-95% percentile
NonHispanic White	4-5	< 0.1 - 0.62
	6-11	< 0.1 - 0.65
	12-15	< 0.1 - 0.56
	16-19	< 0.1 - 0.62
NonHispanic Black	4-5	< 0.1 - 0.94
	6-11	< 0.1 - 1.00
	12-15	< 0.1 - 0.95
	16-19	< 0.1 - 0.76
Mexican American	4-5	< 0.1 - 0.48
	6-11	< 0.1 - 0.62
	12-15	< 0.1 - 0.58
	16-19	< 0.1 - 0.52

Homocysteine

Patient Value µmol/L	Reference Range µmol/L	High/Low Flag
10.90	3.40 - 8.80	H

SAMPLE REPORT

Homocysteine is an amino acid derived from methionine, requiring folic acid, vitamin B6 and B12 for further metabolizing. Homocysteine exists in both free but predominately in protein-bound form. Mutations in the genes coding for the enzymes involved in homocysteine metabolism, e.g. MTHFR (5, 10 methylene-tetrahydrofolate reductase), result in hyperhomocysteinemia. Folic acid, vitamin B6 and vitamin B12 are necessary cofactors for a proper breakdown of homocysteine. Deficiencies of these vitamins are associated with increased plasma levels of homocysteine. Homozygous mutations of the CBS (cystathionine-B-synthase) gene cause severe hyperhomocysteinemia along with homocysteinuria.

Hyperhomocysteinemia was found to be associated with increased risk for ischemic heart disease, stroke, peripheral arterial disease and deep venous thrombosis, as well as for neural tube defects and preeclampsia in pregnancy. High concentrations of homocysteine in blood induce endothelial dysfunction, suggesting a causal role in vascular disease. An increased frequency of hyperhomocysteinemia is observed especially in the elderly, smokers, patients with renal disease, diabetes or on a strict vegetarian diet. Higher than normal homocysteine values have been reported in autism. In autism, homocysteine is negatively correlated with glutathione peroxidase activity. High homocysteine in autism is associated with suboptimal vitamin B-12.

Homocysteine Reference Ranges:

Group	Micromoles/L Range	Percentiles
Men and Women	4.9-15.0	2.5%-97.5%
Boys	3.4-8.8	5%-95%
Girls	3.0-8.2	5%-95%
Autism (75% boys)	4.3-15.3	5%-95%

CBC

(CBC: complete blood count) Do you receive a copy of your test results to keep in your files to compare from year to year?
When getting blood done, ***you can check these markers in addition to the regular ones:***

- B12
- Ferritin Iron
- Cortisol
- Vitamin D
- DHEA
- Insulin
- T4 TSH + Free T4 T3 T3 Uptake
- C-Reactive Protein
- Magnesium
- Biotin (Vitamin B7)
- Niacin (Vitamin B3)
- Vitamin A
- Vitamin C
- Zinc
- Testosterone androstenedione and dihydrotestosterone

Here are the blood test markers to include with a CBC to ***detect the presence of inflammation caused by one or more conditions such as infections, tumors, or autoimmune disorders:***

- C-Reactive Protein (CRP)
- Erythrocyte Sedimentation Rate (ESR)
- TSH (thyroid stimulating hormone) and other thyroid testing – hypothyroidism can cause symptoms similar to CFS (Chronic Fatigue Syndrome)
- Cortisol: when low cortisol concentrations & decreased adrenal gland function are suspected
- Epstein-Barr Virus (EBV) Antibody Profile
- Lyme Antibodies Detection Lyme Antibodies IgM/IgG by Western Blot Formal Name Borrelia Antibodies, IgM/IgG; Borrelia DNA Detection by PCR
- Rheumatoid Factor (RF)

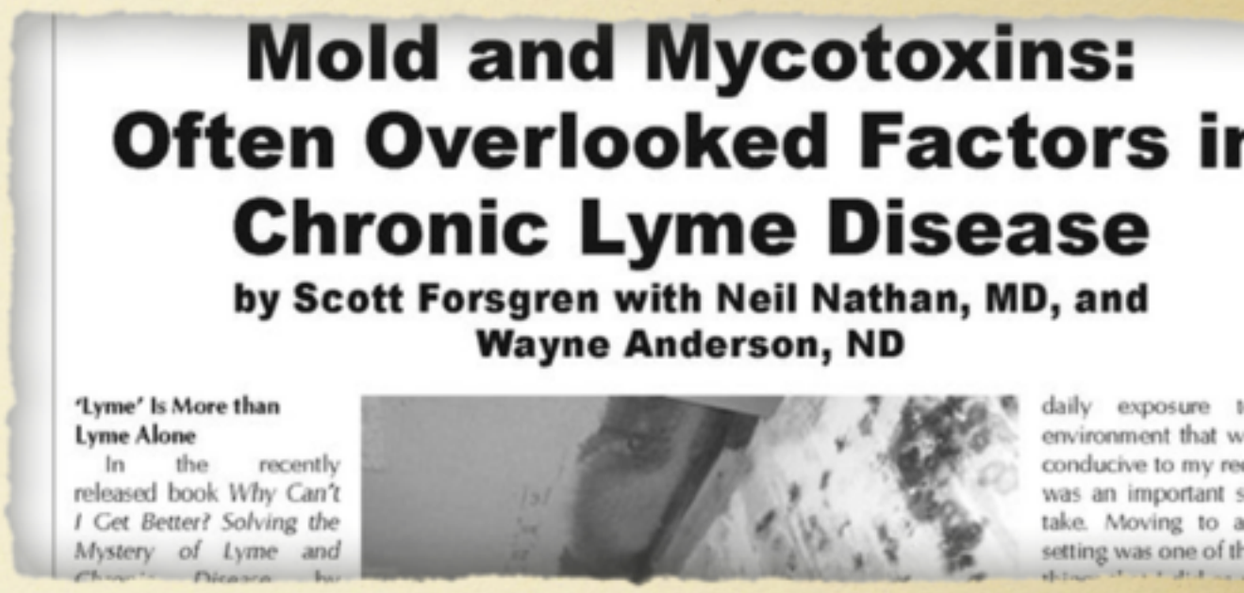
These five blood markers should be part of the extended CBC panel:
Vitamin D, Vitamin B12, Magnesium, Ferritin (Iron), Homocysteine

Blood Tests: Complete Blood Count; Immune globulin reaction to mold and Candida albicans; Vitamin D, Natural Killer cell total count and function, T and B-cell Counts; red blood cell glutathions, Liver enzymes, Creatinine, Glomerular filtration rate, Anti-Diuretic Hormone; Transforming Growth Factor B-1; VEGF; MMP-9; Melanocyte Stimulating Hormone, HLA-DR/DQ; hormonal tests

Mold, Lyme or Both?

- MOLD and Lyme both imitate other conditions; can make preexisting illnesses worse; and alter responses to treatments

- Symptoms of Mold and Lyme are similar but Lyme wanders while Mold symptoms don't; Mold also affects the respiratory system more than Lyme



Mold and Mycotoxins: Often Overlooked Factors in Chronic Lyme Disease

by Scott Forsgren with Neil Nathan, MD, and Wayne Anderson, ND

'Lyme' Is More than Lyme Alone

In the recently released book *Why Can't I Get Better? Solving the Mystery of Lyme and*

daily exposure t
environment that w
conducive to my re
was an important s
take. Moving to a
setting was one of th

- Both mold and Lyme impair the immune system and increase your susceptibility to each condition. If you have mold sickness, you can more easily develop chronic or persisting Lyme disease. Conversely, if you have Lyme disease, you're more susceptible to the effects of mold.

- Both conditions will need treatment; if you have both the one that is predominant should be addressed first -often this is the MOLD.



Labs

- Depending on what state you operate in and where the individual lives will dictate what lab tests are available so you need to call the labs and ask what is allowed in your state. If the individual is sending out the specimen then it depends what state they live in. These regulations change so you must check.

- Doctor's Doctor

- Genova

- Great Plains