The Great Plains Laboratory, Inc.

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Requisition #:	000000	*Practitioner:*	REGENERUS LAB
Patient Name:	Sample Report	*Date of Collection:*	10/19/2021
Patient Age:	44	*Time of Collection:*	07:00 AM
Patient Sex:	F	*Print Date:*	10/27/2021
Specimen Id.:	000000-2		

Organic Acids Test - Nutritional and Metabolic Profile

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Females Age 13 and Over
Intestinal Microbial Overgrowth			
Yeast and Fungal Markers			
1 Citramalic	≤ 3.6	2.0	
2 5-Hydroxymethyl-2-furoic *(Aspergillus)*	≤ 14	1.8	
3 3-Oxoglutaric	≤ 0.33	0.02	
4 Furan-2,5-dicarboxylic *(Aspergillus)*	≤ 16	2.1	
5 Furancarbonylglycine *(Aspergillus)*	≤ 1.9	0.17	
6 Tartaric *(Aspergillus)*	≤ 4.5	0.69	
7 Arabinose	≤ 29	20	
8 Carboxycitric	≤ 29	22	
9 Tricarballylic *(Fusarium)*	≤ 0.44	0.18	
Bacterial Markers			
10 Hippuric	≤ 613	199	
11 2-Hydroxyphenylacetic	0.06 - 0.66	0.30	
12 4-Hydroxybenzoic	≤ 1.3	0.83	
13 4-Hydroxyhippuric	0.79 - 17	6.0	
14 DHPPA (Beneficial Bacteria)	≤ 0.38	0.24	
Clostridia Bacterial Markers			
15 4-Hydroxyphenylacetic *(C. difficile, C. stricklandii, C. lituseburense & others)*	≤ 19	6.5	
16 HPHPA *(C. sporogenes, C. caloritolerans, C. botulinum & others)*	≤ 208	56	
17 4-Cresol *(C. difficile)*	≤ 75	11	
18 3-Indoleacetic *(C. stricklandii, C. lituseburense, C. subterminale & others)*	≤ 11	1.4	

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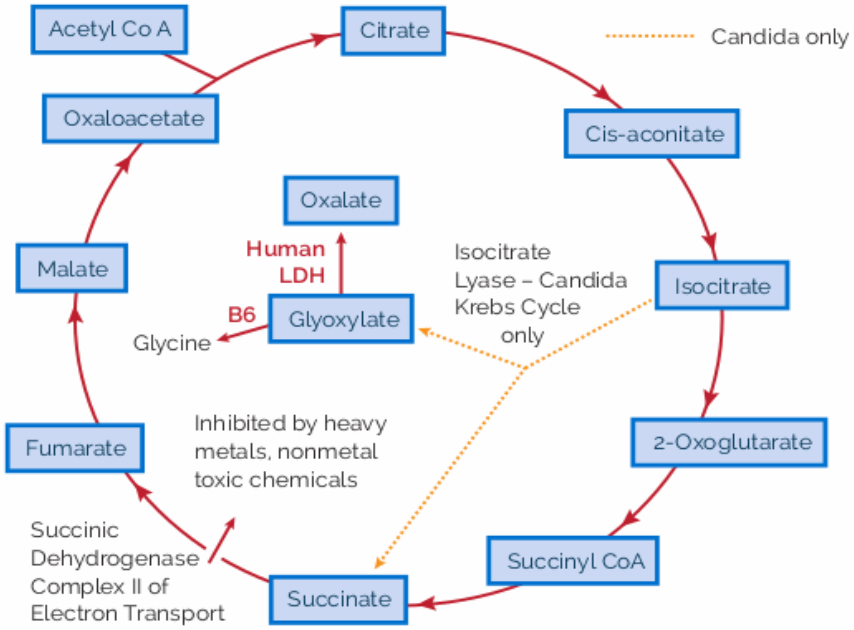
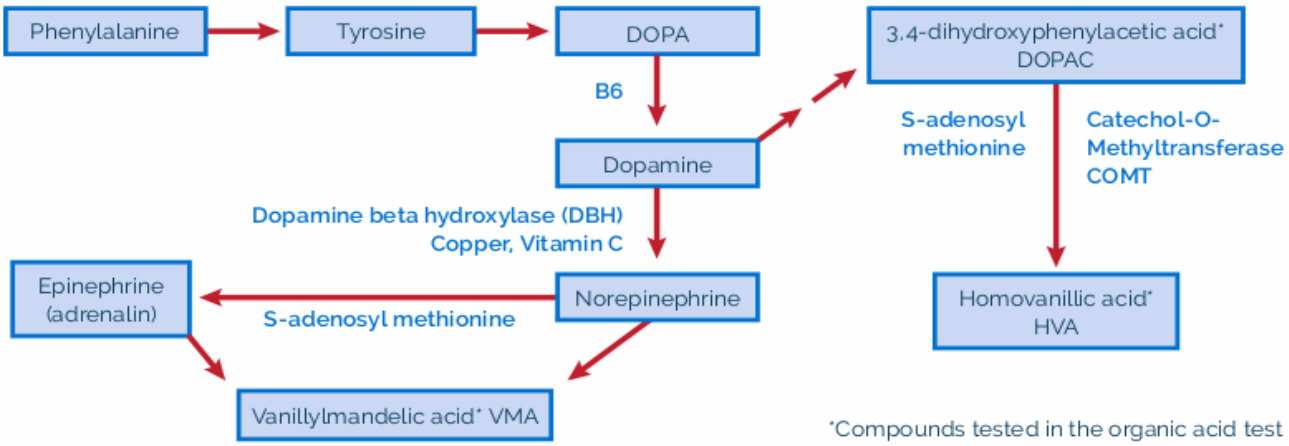
Human Krebs Cycle showing Candida Krebs Cycle variant that causes excess Oxalate via Glyoxylate



Major pathways in the synthesis and breakdown of **catecholamine neurotransmitters** in the absence of microbial inhibitors



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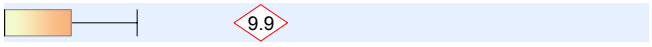
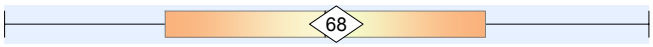
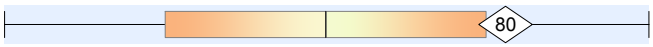
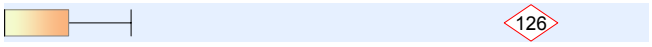
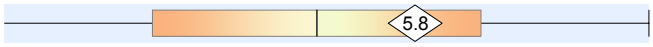
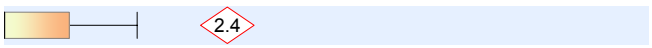
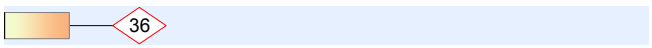
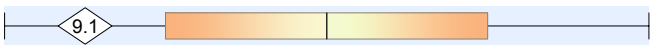
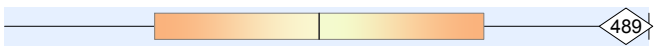
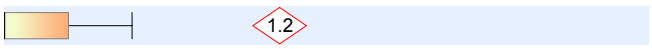
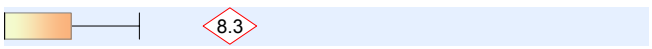
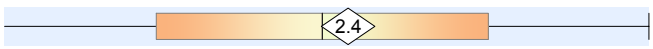
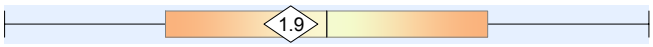
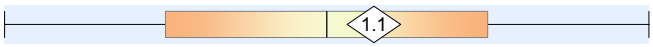
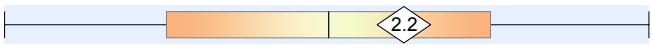
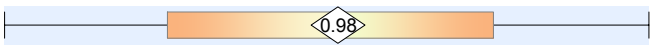
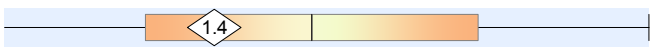
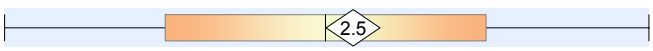
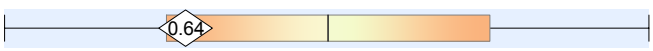
Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Females Age 13 and Over
Oxalate Metabolites			
19 Glyceric	0.77 - 7.0	H 9.9	
20 Glycolic	16 - 117	68	
21 Oxalic	6.8 - 101	80	
Glycolytic Cycle Metabolites			
22 Lactic	≤ 48	H 126	
23 Pyruvic	≤ 9.1	5.8	
Mitochondrial Markers - Krebs Cycle Metabolites			
24 Succinic	≤ 9.3	4.3	
25 Fumaric	≤ 0.94	0.36	
26 Malic	0.06 - 1.8	H 2.4	
27 2-Oxoglutaric	≤ 35	H 36	
28 Aconitic	6.8 - 28	9.1	
29 Citric	≤ 507	489	
Mitochondrial Markers - Amino Acid Metabolites			
30 3-Methylglutaric	≤ 0.76	H 1.2	
31 3-Hydroxyglutaric	≤ 6.2	H 8.3	
32 3-Methylglutaconic	≤ 4.5	2.4	
Neurotransmitter Metabolites			
Phenylalanine and Tyrosine Metabolites			
33 Homovanillic (HVA) *(dopamine)*	0.80 - 3.6	2.2	
34 Vanillylmandelic (VMA) *(norepinephrine, epinephrine)*	0.46 - 3.7	1.9	
35 HVA / VMA Ratio	0.16 - 1.8	1.1	
36 Dihydroxyphenylacetic (DOPAC) *(dopamine)*	0.08 - 3.5	2.2	
37 HVA/ DOPAC Ratio	0.10 - 1.8	0.98	
Tryptophan Metabolites			
38 5-Hydroxyindoleacetic (5-HIAA) *(serotonin)*	≤ 4.3	1.4	
39 Quinolinic	0.85 - 3.9	2.5	
40 Kynurenic	≤ 2.2	0.64	

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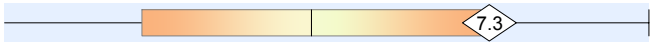
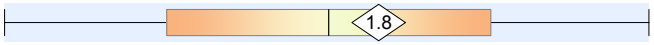
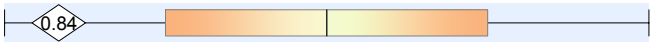
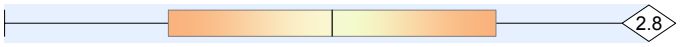
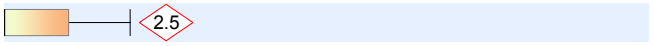
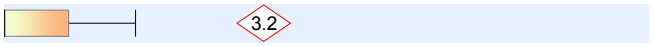
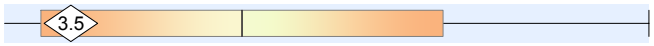
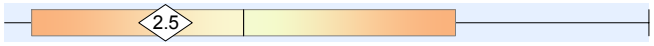
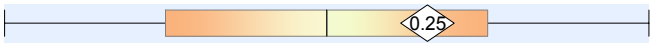
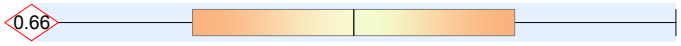
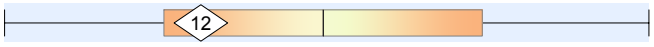
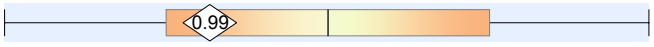
Pyrimidine Metabolites - Folate Metabolism			
41 Uracil	≤ 9.7	7.3	
42 Thymine	≤ 0.56	0.23	
Ketone and Fatty Acid Oxidation			
43 3-Hydroxybutyric	≤ 3.1	1.8	
44 Acetoacetic	≤ 10	0.84	
45 Ethylmalonic	0.44 - 2.8	2.8	
46 Methylsuccinic	0.10 - 2.2	H 2.5	
47 Adipic	0.04 - 3.8	H 6.9	
48 Suberic	0.18 - 2.2	H 3.2	
49 Sebacic	≤ 0.24	0.20	
Nutritional Markers			
Vitamin B12			
50 Methylmalonic ✱	≤ 2.3	2.2	
Vitamin B6			
51 Pyridoxic (B6)	≤ 34	3.5	
Vitamin B5			
52 Pantothenic (B5)	≤ 10	2.5	
Vitamin B2 (Riboflavin)			
53 Glutaric ✱	0.04 - 0.36	0.25	
Vitamin C			
54 Ascorbic	10 - 200	L 0.66	
Vitamin Q10 (CoQ10)			
55 3-Hydroxy-3-methylglutaric ✱	0.17 - 39	12	
Glutathione Precursor and Chelating Agent			
56 N-Acetylcysteine (NAC)	≤ 0.28	0.10	
Biotin (Vitamin H)			
57 Methylcitric ✱	0.19 - 2.7	0.99	

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

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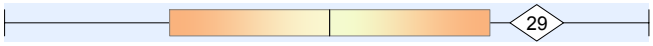
Specimen Id.:	000000-2		

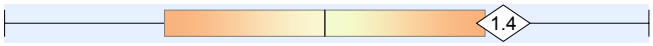
Metabolic Markers in Urine	Reference Range *(mmol/mol creatinine)*	Patient Value	Reference Population - Females Age 13 and Over

Indicators of Detoxification

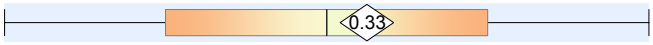
Glutathione

58	Pyroglutamic ✱	10 - 33		29	

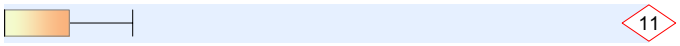
Methylation, Toxic exposure

59	2-Hydroxybutyric ✱✱	0.03 - 1.8		1.4	

Ammonia Excess

60	Orotic	0.06 - 0.54		0.33	

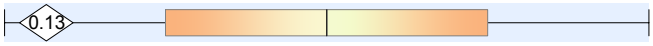
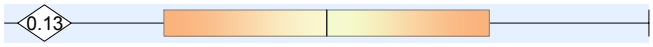
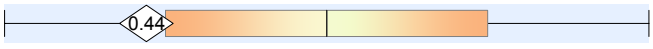
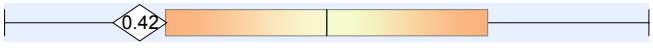
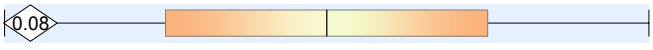
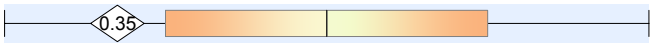
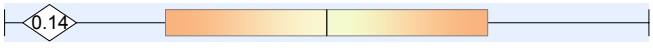
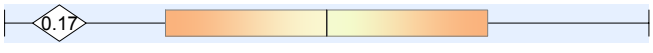
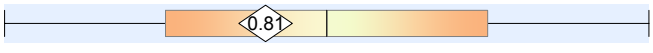
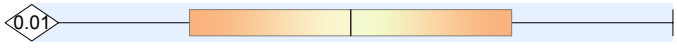
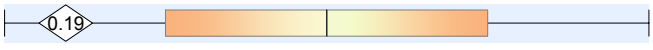
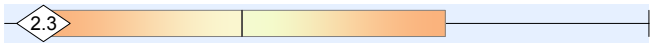
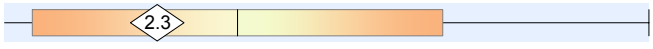
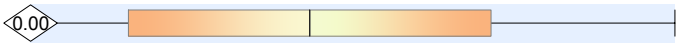
Aspartame, salicylates, or GI bacteria

61	2-Hydroxyhippuric	≤ 1.3	H	11	

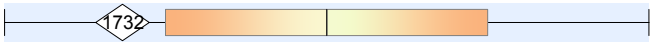
✱ ***A high value for this marker may indicate a Glutathione deficiency.***

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

Amino Acid Metabolites

62	2-Hydroxyisovaleric	≤ 2.0		0.13	
63	2-Oxoisovaleric	≤ 2.1		0.13	
64	3-Methyl-2-oxovaleric	≤ 2.0		0.44	
65	2-Hydroxyisocaproic	≤ 2.0		0.42	
66	2-Oxoisocaproic	≤ 2.0		0.08	
67	2-Oxo-4-methiolbutyric	≤ 2.0		0.35	
68	Mandelic	≤ 2.0		0.14	
69	Phenyllactic	≤ 2.0		0.17	
70	Phenylpyruvic	≤ 2.0		0.81	
71	Homogentisic	≤ 2.0		0.01	
72	4-Hydroxyphenyllactic	≤ 2.0		0.19	
73	N-Acetylaspartic	≤ 38		2.3	
74	Malonic	≤ 9.7		2.3	
75	4-Hydroxybutyric	≤ 4.8		0	

Mineral Metabolism

76	Phosphoric	1,000 - 5,000		1,732	

Requisition #:	000000	*Practitioner:*	REGENERUS LAB
Patient Name:	Sample Report	*Date of Collection:*	10/19/2021
Specimen Id.:	000000-2		

Indicator of Fluid Intake

77	*Creatinine	85	mg/dL	

*The creatinine test is performed to adjust metabolic marker results for differences in fluid intake. Urinary creatinine has limited diagnostic value due to variability as a result of recent fluid intake. Samples are rejected if creatinine is below 20 mg/dL unless the client requests results knowing of our rejection criteria.

Explanation of Report Format

The reference ranges for organic acids were established using samples collected from typical individuals of all ages with no known physiological or psychological disorders. The ranges were determined by calculating the mean and standard deviation (SD) and are defined as ± 2SD of the mean. Reference ranges are age and gender specific, consisting of Male Adult (≥13 years), Female Adult (≥13 years), Male Child (<13 years), and Female Child (<13 years).

There are two types of graphical representations of patient values found in the new report format of both the standard Organic Acids Test and the Microbial Organic Acids Test.

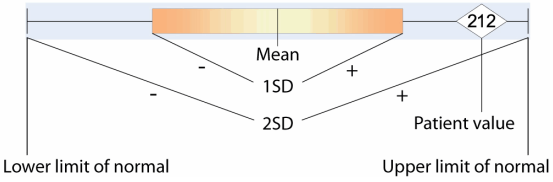
The first graph will occur when the value of the patient is within the reference (normal) range, defined as the mean plus or minus two standard deviations.

The second graph will occur when the value of the patient exceeds the upper limit of normal. In such cases, the graphical reference range is “shrunk” so that the degree of abnormality can be appreciated at a glance. In this case, the lower limits of normal are not shown, only the upper limit of normal is shown.

In both cases, the value of the patient is given to the left of the graph and is repeated on the graph inside a diamond. If the value is within the normal range, the diamond will be outlined in black. If the value is high or low, the diamond will be outlined in red.

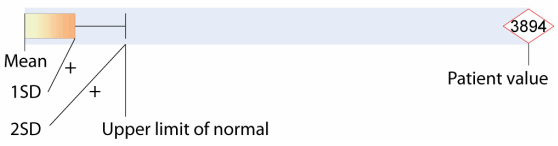
Example of Value Within Reference Range

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Result	Reference Range - Males Age 13 and Under
Intestinal Microbial Overgrowth				
HPHPA (Clostridia marker)	<	219 .9	212	



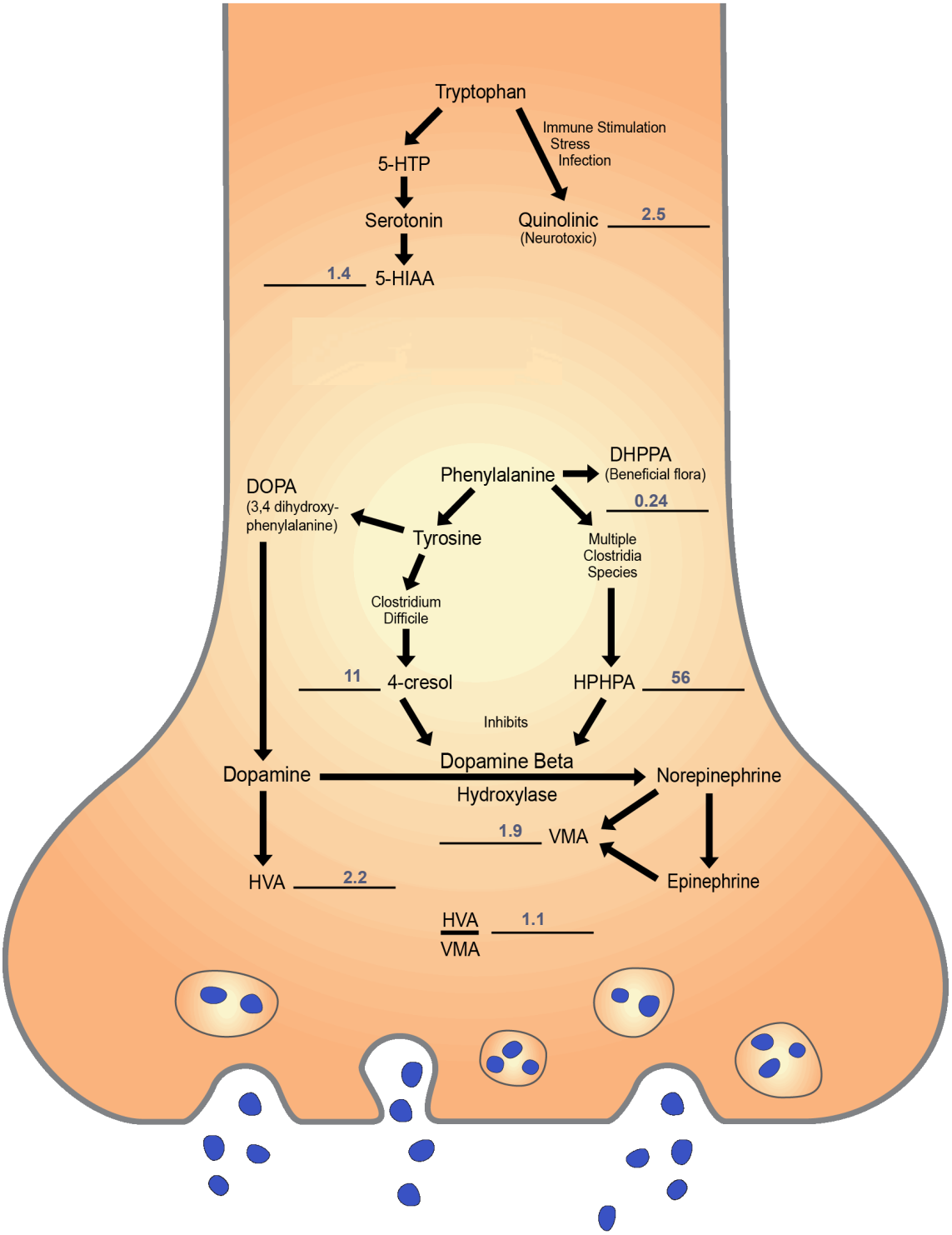
Example of Elevated Value

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Result	Reference Range - Males Age 13 and Under
Intestinal Microbial Overgrowth				
HPHPA (Clostridia marker)	<	219 .9	H 3894	



Requisition #:	000000	***Practitioner:***	REGENERUS LAB
Patient Name:	Sample Report	***Date of Collection:***	10/19/2021
Specimen Id.:	000000-2		

Neurotransmitter Metabolism Markers



The diagram contains the patient's test results for neurotransmitter metabolites and shows their relationship with key biochemical pathways within the axon terminal of nerve cells. The effect of microbial byproducts on the blockage of the conversion of dopamine to norepinephrine is also indicated.

Requisition #:	000000	***Practitioner:***	REGENERUS LAB
Patient Name:	Sample Report	***Date of Collection:***	10/19/2021
Specimen Id.:	000000-2		

Interpretation

High glyceric (19): may be due to microbial sources such as yeast (Aspergillus, Penicillium, Candida) or due to dietary sources containing glycerol/glycerine.

High lactic acid and/or high pyruvic acid (22,23) may be caused by many nonspecific factors, such as vigorous exercise, bacterial overgrowth of the GI tract, shock, poor perfusion, anemia, mitochondrial dysfunction or damage, and many other causes. Conversion of pyruvic acid to acetyl-CoA requires the cofactors coenzyme A (derived from pantothenic acid), lipoic acid, FAD derived from riboflavin, and thiamine. However, the possibility of an inborn error of metabolism increases as the value exceeds 300 mmol/mol creatinine. Values greater than 1000 mmol/mol creatinine indicate a much higher likelihood of an inborn error of metabolism. There are many inborn errors of metabolism that present elevated lactic acid, including disorders of sugar metabolism and pyruvate dehydrogenase deficiency.

High malic acid (26) indicates a greater requirement for the nutrients niacin and coenzyme Q10.* Malic acid simultaneously elevated with citric, fumaric and alpha-ketoglutaric acids may indicate a possible Cytochrome C Oxidase deficiency. Mitochondrial energy pathway dysfunction would be expected.

High 2-oxoglutaric acid (alpha-ketoglutaric acid) (27) may be due to dietary deficiencies of any of several enzyme cofactors or the intake of alpha-ketoglutaric acid (AKG) as a supplement. Conversion of 2-oxoglutaric acid to succinyl-CoA requires the cofactors coenzyme A (derived from pantothenic acid), FAD (derived from riboflavin), and thiamine.* Increased conversion of glutamic acid to AKG is another possible explanation. Extremely high values (ten times the upper limit of normal) may be due to genetic enzyme deficiencies and indicate the need for consultation with a biochemical genetics specialist.

High 3-methylglutaric and/or high 3-methylglutaconic acids (30, 32) may be due to reduced capacity to metabolize the amino acid leucine. This abnormality is found in the genetic disease methylglutaconic aciduria and in mitochondrial disorders in which there are severe deficiencies of the respiratory complexes (Complex I, NADH ubiquinone oxidoreductase and complex IV, cytochrome c oxidase.). Small elevations may be due to impairment of mitochondrial function and may respond to the recommended supplements below. Typical results found in genetic defects are above 10 mmol/mol creatinine. A few non-genetic conditions including pregnancy and kidney failure may also produce elevation of these organic acids in urine. Confirmation of the genetic disease requires enzymes and/or DNA testing. Multiple genetic defects can cause the biochemical abnormality. Confirmation of mitochondrial disorder usually requires tissue biopsy for mitochondria testing. Symptoms differ within different types of genetic disorders, but in severe cases may include speech delay, delayed development of both mental and motor skills (psychomotor delay), metabolic acidosis, abnormal muscle tone (dystonia), and spasms and weakness affecting the arms and legs (spastic quadriparesis). Recommendations include supplementation with coenzyme Q-10, L-carnitine and acetyl-L-carnitine, riboflavin, nicotinamide, and vitamin E.

Requisition #:	000000	***Practitioner:***	REGENERUS LAB
Patient Name:	Sample Report	***Date of Collection:***	10/19/2021
Specimen Id.:	000000-2		

High 3-hydroxyglutaric (31) is a metabolite associated with the genetic disease glutaric aciduria type I, which is due to a deficiency of glutaryl CoA dehydrogenase, an enzyme involved in the breakdown of lysine, hydroxylysine, and tryptophan. Other organic acids elevated include glutaric and glutaconic. This disease has been associated with clinical symptoms ranging from near normal to encephalopathy, cerebral palsy, and other neurological abnormalities. Some individuals with glutaric acidemia have developed bleeding in the brain or eyes that may be mistaken for the effects of child abuse . This abnormality should be confirmed by additional testing of enzyme deficiencies and/ or DNA at a major pediatric medical genetics center (Morton et al. Glutaric aciduria type I: a common cause of encephalopathy and spastic paralysis in the Amish of Lancaster County, Pennsylvania. American J. Med. Genetics 41: 89-95, 1991). Elevated values may also be found in hepatic carnitine palmitoyltransferase I deficiency, short-chain acyl dehydrogenase deficiency (SCAD), and ketosis. Mitochondrial dysfunction induced by glutaric acid metabolites causes astrocytes to adopt a proliferative phenotype, which may underlie neuronal loss, white matter abnormalities and macrocephalia. Values in glutaric aciduria type I range from 60-3000 mmol/mol creatinine. Values higher than normal but less than 60 mmol/mol creatinine may be due to mild glutaric acidemia type I or to the other causes indicated above. Treatment of this disorder includes special diets low in lysine and supplementation with carnitine or acetyl-L-carnitine.

Vanillylmandelic acid (VMA) levels (34) below the mean indicate low production and/or decreased metabolism of the neurotransmitters norepinephrine and epinephrine. Vanillylmandelic acid is a metabolite of the neurotransmitters norepinephrine and epinephrine. Low production of VMA can be due to decreased intake or absorption of norepinephrine's and epinephrine's precursor amino acids such as phenylalanine and/or tyrosine, decreased quantities of cofactors needed for biosynthesis of norepinephrine and epinephrine such as tetrahydrobiopterin and vitamin B6 coenzyme or decreased amounts of cofactors such as S-adenosylmethionine (Sam-e) needed to convert norepinephrine and epinephrine to VMA. In addition, a number of genetic variations such as single nucleotide polymorphisms (SNPs) or mutations in MAO or COMT genes can cause reduced production of VMA. Such SNPs are available on **The Great Plains DNA methylation pathway test** which can be performed on a cheek swab. VMA values below the mean but which are much lower than HVA values are usually due to impairment of dopamine beta hydroxylase due to Clostridia metabolites, the mold metabolite fusaric acid, pharmaceuticals such as disulfiram, or food additives like aspartame or deficiencies of cofactors such as vitamin C or copper. Values may be decreased in patients on monoamine oxidase (MAO) inhibitors. Another cause for a low VMA value is a genetic variation (single nucleotide polymorphism or SNP) of the DBH enzyme. Patients with low VMA due to Clostridia metabolites or genetic DBH deficiency should not be supplemented with phenylalanine, tyrosine, or L-DOPA.

5-hydroxyindoleacetic acid (5HIAA) (38) levels below the mean may indicate lower production and/or decreased metabolism of the neurotransmitter serotonin. 5-hydroxy-indoleacetic acid is a metabolite of serotonin. Low values have been correlated with symptoms of depression. Low production of 5 HIAA can be due to decreased intake or absorption of serotonin's precursor amino acid tryptophan, decreased quantities of cofactors needed for biosynthesis of serotonin such as tetrahydrobiopterin and vitamin B6 coenzyme. In addition, a number of genetic variations such as single nucleotide polymorphisms (SNPs) or mutations can cause reduced production of 5HIAA. Such SNPs are available on **The Great Plains DNA methylation pathway test** which can be performed on a cheek swab. Values may be decreased in patients on monoamine oxidase (MAO) inhibitors that are drugs or foods that contain tyramine such such as Chianti wine and vermouth, fermented foods such as cheeses, fish, bean curd, sausage, bologna, pepperoni, sauerkraut, and salami.

High ethylmalonic, methylsuccinic, adipic, suberic, or sebacic acids (45,46,47,48,49) may be due to fatty acid oxidation disorders, carnitine deficiency, fasting, or to increased intake of the medium-chain triglycerides found in coconut oil, MCT oil, and some infant formulas. The fatty acid oxidation defects are associated with hypoglycemia, apnea episodes, lethargy, and coma. [An acyl carnitine profile (Duke University Biochemical Genetics Laboratory, http://medgenetics.pediatrics.duke.edu) can rule out fatty acid oxidation defects.] Regardless of cause, supplementation with L-carnitine or acetyl-L-carnitine may be beneficial.

Pyridoxic acid (B6) levels below the mean (51) may be associated with less than optimum health conditions (low intake, malabsorption, or dysbiosis). Supplementation with B6 or a multivitamin may be beneficial.

Pantothenic acid (B5) levels below the mean (52) may be associated with less than optimum health conditions. Supplementation with B5 or a multivitamin may be beneficial.

Requisition #:	000000	***Practitioner:***	REGENERUS LAB
Patient Name:	Sample Report	***Date of Collection:***	10/19/2021
Specimen Id.:	000000-2		

Ascorbic acid (vitamin C) levels below the mean (54) may indicate a less than optimum level of the antioxidant vitamin C. Individuals who consume large amounts of vitamin C can still have low values if the sample is taken 12 or more hours after intake. Supplementation with buffered vitamin C taken 2 or 3 times a day is suggested.

High 2-hydroxyhippuric acid (61) may result from ingestion of aspartame (Nutrasweet®), salicylates (aspirin), dietary salicylates, or from GI bacteria converting tyrosine or phenylalanine to salicylic acid. For more information about salicylates in foods go to <http://www.feingold.org/salicylate.php>. 2-Hydroxyhippuric acid is a conjugate of hydroxybenzoic acid (salicylic acid) and glycine. Very high 2-hydroxyhippuric also inhibits dopamine beta-hydroxylase resulting in elevated HVA, decreased VMA, and elevated HVA/VMA ratio.

High quality nutritional supplements can be purchased through your practitioner or at New Beginnings Nutritionals, www.NBNUS.com <http://www.NBNUS.com> , or call 877-575-2467.

The Great Plains Laboratory, Inc.

William Shaw, Ph.D Director | 11813 W. 77th Street, Lenexa, KS 66214 | (913) 341-8949 | Fax (913) 341-6207

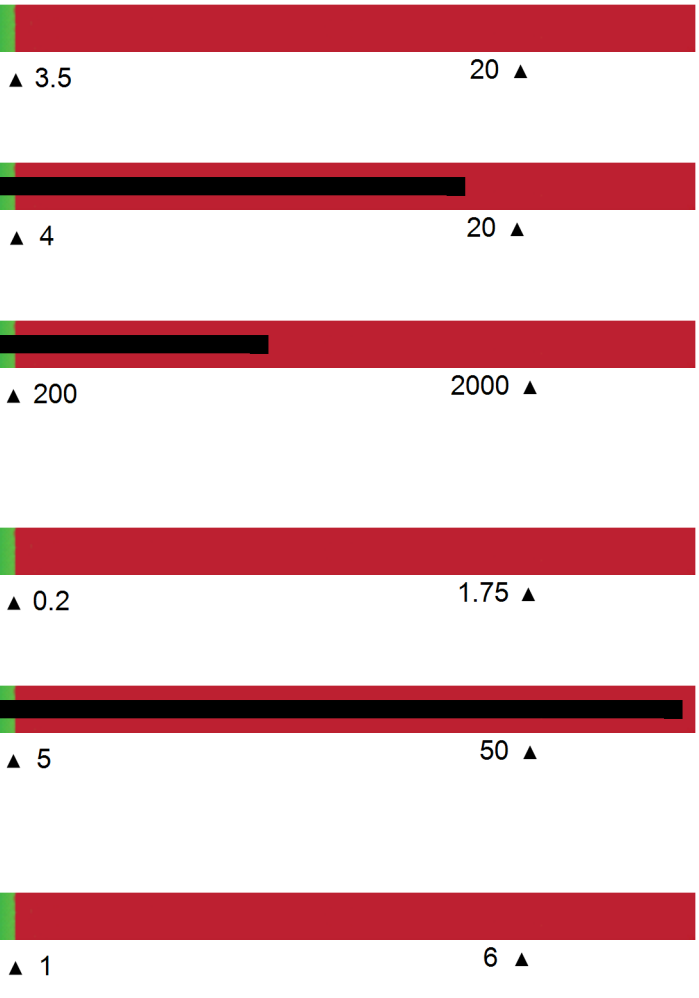
Sample Report CMI42

GPL-MYCOTOX

		Physician Name	REGENERUS LAB
		Date of Collection	6/28/2019
Patient BirthDate	11/17/1977	Time of Collection	7:15 AM
Sex	F	Print Date	7/15/2019

MycoTox Profile

Metabolite	Results (ng/g creatinine)	Common Range of Positive Results
Aspergillus		
Aflatoxin-M1	0.00	3.5 - 20
Ochratoxin A	18.04	4 - 20
Gliotoxin	1055.37	200 - 2000
Penicillum		
Sterigmatocystin	0.00	0.2 - 1.75
Mycophenolic Acid	332.28	5 - 50
Stachybotrys		
Roridin E	0.00	1 - 6



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

The Great Plains Laboratory, Inc.

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Sample Report CMI42

GPL-MYCOTOX

Metabolite	Results (ng/g creatinine)	Common Range of Positive Results
Verrucarin A	0.00	1 - 10

▲ 1 10 ▲

Fusarium

Enniatin B	0.00	0.07 - 1

▲ 0.07 1 ▲

Zearalenone	0.00	0.5 - 10

▲ 0.5 10 ▲

Chaetomium globosum

Chaetoglobosin A	121.01	20 - 80

▲ 20 80 ▲

Multiple Mold Species

Citrinin	0.00	10 - 50

▲ 10 50 ▲

Ochratoxin: Ochratoxin A (OTA) is a nephrotoxic, immunotoxic, and carcinogenic mycotoxin. This chemical is produced by molds in the Aspergillus and Penicillium families. Exposure is done primarily through water damaged buildings. Minimal exposure can occur 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 lead to significant oxidative damage to multiple brain regions and is highly nephrotoxic. Dopamine levels in the brain of mice have been shown to be decreased after exposure to OTA. Some studies have hypothesized that OTA may contribute to the development of neurodegenerative diseases such as Alzheimer's and Parkinson's. Treatment should be aimed at removing the source of exposure. Agents such as oral cholestyramine, charcoal, and phenylalanine can help prevent the absorption of these toxins from food. Antioxidants such as vitamins A, E, C, NAC, rosmarinic acid, and liposomal glutathione alone or in combination have been shown to mitigate the oxidative effects of the toxin. Bentonite or zeolite clay is reported to reduce the absorption of multiple mycotoxins found in food, including OTA. Studies have also shown that OTA is present in sweat, which supports the use of sauna as a treatment to increase the excretion of OTA. (PMID 17195275, 16621780, 16293235, 27521635, 22069626, 24792326, 22253638, 16140385, 2467220, 16844142, 19148691, 22069658, 16019795, 18286403, 15781206, 11439224, 17092826, 32710148)

Gliotoxin: Gliotoxin (GTX) is produced by the mold genus Aspergillus. Aspergillus spreads in the environment by releasing conidia which are capable of infiltrating the small alveolar airways of individuals. In order 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. (PMID: 16712786, 27048806, 21575912, 23278106)

Mycophenolic Acid: Mycophenolic Acid (MPA) 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. (PMID: 28646113, 27809954, 27599910)

Chaetoglobosin A: Chaetoglobosin A (CHA) is produced by the mold Chaetomium globosum (CG). CG is commonly found is 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. PMID: 21196335, 12781669, 17551849

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		Physician Name:	REGENERUS LAB
		Date of Collection:	4/24/2019
Patient BirthDate:	11/25/2011	Time of Collection:	7:20 AM
Sex:	F	Print Date:	5/2/2019

Glyphosate Profile Sample Report CMI47

Metabolite	Result ug/g creatinine	Reference Range
Glyphosate	1.35	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.

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. The World Health Organization International Agency for Research on Cancer published a summary in March 2015 that classified glyphosate as a probably carcinogen in humans. Possible cancers linked to glyphosate exposure include non-Hodgkin lymphoma, renal tubule carcinoma, pancreatic islet-cell adenoma, and skin tumors. Studies have also indicated that glyphosate disrupts the microbiome in the intestine, causing a decrease in the ratio of beneficial to harmful bacteria. The relationship between the microbiome of the intestine and overall human health is still unclear, but current research indicates that disruption of the microbiome could cause diseases such as autism, metabolic disorder, diabetes, depression, cardiovascular disease, and autoimmune disease.

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

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

Requisition #:	000000	***Physician Name:***	REGENERUS LAB
Patient Name:	Sample Report 44	***Date of Collection:***	10/19/2021
Patient Age:		***Time of Collection:***	07:00 AM
Sex:	F	***Print Date:***	11/10/2021
Specimen Id.:	000000-2		

Toxic Compounds

Metabolite	Result µg/g creatinine	Percentile

Industrial Toxicants

		LLOQ	75th	95th
1) 2-Hydroxyisobutyric Acid (2HIB)	6,022	600	22,479	35,724

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.

		LLOQ	75th	95th
2) Monoethylphthalate (MEP)	19	15	218	1,122

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.

		LLOQ	75th	95th
3) 2-3-4 Methylhippuric Acid (2,-3-,4-MHA)	548	30	1,810	4,869

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.

**LLOQ - Lower Limit of Quantitation* ***N.D. - Not Detected*

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

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Requisition #:	000000	*Physician Name:*	REGENERUS LAB
Patient Name:	Sample Report 44	*Date of Collection:*	10/19/2021
Patient Age:		*Time of Collection:*	07:00 AM
Sex:	F	*Print Date:*	11/10/2021
Specimen Id.:	000000-2		

Toxic Compounds

Metabolite	Result µg/g creatinine	Percentile
4) Phenylglyoxylic Acid (PGO)	208	LLOQ 15 / 75th 837 / 95th 1,686

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.

5) N-acetyl phenyl cysteine (NAP)	N.D.	LLOQ 0.60 / 75th 3.6 / 95th 9.1

Parent: Benzene

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

6) N-acetyl(2-cyanoethyl)cysteine (NACE)	N.D.	75th 17 / 95th 681

Parent: Acrylonitrile

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

7) Perchlorate (PERC)	N.D.	LLOQ 6.0 / 75th 15 / 95th 32

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.

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Patient Name:	Sample Report 44	*Date of Collection:*	10/19/2021
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Sex:	F	*Print Date:*	11/10/2021
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Toxic Compounds

Metabolite	Result µg/g creatinine	Percentile
8) Diphenyl phosphate (DPP)	N.D.	LLOQ 3.0, 75th 4.8, 95th 17

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.

Metabolite	Result µg/g creatinine	Percentile
9) 2-hydroxyethyl mercapturic (HEMA)	N.D.	LLOQ 2.4, 75th 5.6, 95th 15

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]).

Metabolite	Result µg/g creatinine	Percentile
10) N-acetyl(propyl)cysteine (NAPR)	N.D.	LLOQ 12, 75th 26, 95th 106

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.

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Toxic Compounds

Metabolite	Result µg/g creatinine	Percentile
11) N-acetyl(2-hydroxypropyl)cysteine (NAHP)	18	LLOQ 12 · 75th 144 · 95th 540

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.

Metabolite	Result µg/g creatinine	Percentile
12) N-acetyl-S-(2-carbamoylethyl)cysteine (NAE)	76	LLOQ 12 · 75th 261 · 95th 699

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.

Metabolite	Result µg/g creatinine	Percentile
13) N-acetyl(3,4-dihydroxybutyl)cysteine (NADB)	195	LLOQ 12 · 75th 1,170 · 95th 1,872

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.

*** We resolved the problem with NADB and it is now back in the profile. Thank you for your patience.

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Toxic Compounds

Metabolite	Result µg/g creatinine	Percentile
Organophosphate Insecticide Metabolites		
14) Dimethylphosphate (DMP)	N.D.	LLOQ 12, 75th 27, 95th 101

Parent: Organophosphates

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

15) Diethylphosphate (DEP)	1.3	LLOQ 1.8, 75th 9.6, 95th 47

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.

Herbicide		
16) 2,4-Dichlorophenoxyacetic Acid (2-,4-D)	N.D.	LLOQ 0.60, 75th 1.5, 95th 4.7

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.

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Toxic Compounds

Metabolite	Result µg/g creatinine	Percentile
17) 3-hydroxypropylmercapturic acid (3-HPMA)	309	

Parent: Acrolein

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

18) 3-Phenoxybenzoic Acid (3PBA)	N.D.	

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

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

Metabolite	Result Creat mmol/mol	Percentile
19) Tiglylglycine (TG)	0.67	

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.

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Metabolite	Result µg/g creatinine	Percentile
14) Dimethylphosphate (DMP)	N.D.	LLOQ 4.0 · 75th 9.1 · 95th 34

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

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Metabolite	Result µg/g creatinine	Percentile
15) Diethylphosphate (DEP)	1.3	LLOQ 0.60 · 75th 3.2 · 95th 16

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

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Interpretation

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

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

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

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

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

N-acetyl (2-cyanoethyl) cysteine (NACE) (Marker 6) is a result of the exposure to acrylonitrile and NACE is the major metabolite. 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. Elimination of acrylonitrile can be accelerated by the supplementation of glutathione (reduced) either oral, intravenous, transdermal, or its precursor N-acetyl cysteine(NAC).

Perchlorate (PERC) (Marker 7) can result from the exposure to this chemical which is used in the production of rocket fuel, missiles, fireworks, flares, explosives, fertilizers, cleansers, and bleach. Studies show that perchlorate is often found in water supplies. Perchlorate has also been found in food, including cow's milk, eggs, vegetables, and fruit. Perchlorate's main target organ is the thyroid gland. Perchlorate inhibits the thyroid's uptake of iodine. Iodine is required as a building block for the synthesis of thyroid hormone. Perchlorate's inhibition of iodide uptake could lead to hypothyroidism. The thyroid hormone plays a critical role in the neurological development of the fetus, so perchlorate exposure in pregnancy could result in neurodevelopmental effects. The EPA has also labeled perchlorate a likely human carcinogen. Patients that are high in perchlorate can use a reverse osmosis water treatment system (or ion exchange) to remove the chemical from their water supply.

Diphenyl phosphate (Marker 8) is a metabolite of the organophosphate flame retardant triphenyl phosphate (TPHP), which is used in plastics, electronic equipment, nail polish, and resins. Exposure can result from PVC piping, rubber, polyurethane, textiles, and pigments, and paints. TPHP can cause endocrine disruption. Studies have also linked TPHP to reproductive and developmental problems. Diphenyl Phosphate is eliminated from the body by the Glucuronosyltransferase enzymes.

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

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

N-acetyl (propyl)cysteine (NAPR) (Marker 10) is a metabolite of 1-bromopropane, which 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. Individuals who have high levels of 1-bromopropane should examine their environment to determine their exposure route. 1- bromopropane elimination can be accelerated by the supplementation of glutathione (reduced) either oral, intravenous, transdermal, or its precursor N-acetyl cysteine(NAC).

N-acetyl (2-hydroxypropyl) cysteine (NAHP) (Marker 11) is a metabolite of propylene oxide. Propylene oxide 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. The National Institute for Occupational Safety and Health (NIOSH) estimates that approximately 209,000 US workers are exposed each year. Health effects include corneal burns, dermatitis, and DNA damage. Propylene oxide elimination can be accelerated by the supplementation of glutathione (reduced) oral, intravenous, transdermal, or its precursor N-acetyl cysteine (NAC).

N-acetyl-S-(2-carbamoylethyl)-cysteine (NAE) (Marker 12) is a metabolite of acrylamide. Acrylamide is used in many industrial processes such as plastics, food packaging, cosmetics, nail polish, dyes, and treatment of drinking water. Acrylamide can also be formed during the frying of starchy foods such as breads and potatoes. Acrylamide can cause skin irritation such as redness and peeling. It has also been tied to neuropathy regarding the central nervous system and the peripheral nervous system. Long term exposure to acrylamide can produce motor and sensory polyneuropathy such as numbness of lower limbs, tingling of the fingers, vibratory loss, ataxic gait, and muscular atrophy. Studies have also shown that acrylamide has carcinogenic properties. Acrylamide elimination can be accelerated by the supplementation of glutathione (reduced) either oral, intravenous, transdermal, or its precursor N-acetyl cysteine (NAC).

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Sex:	F	***Print Date:***	11/10/2021
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N-Acetyl(3,4-dihydroxybutyl) cysteine (NADB) (Marker 13) is a result of the exposure to 1,3 butadiene and NABD is the major metabolite. This metabolite is evidence of exposure to synthetic rubber such as tires. The primary route of exposure is inhalation. Some exposure may occur through ingestion of contaminated food or water or through dermal contact. Newer playgrounds and athletic fields are now made with ground up tires, which may lead to increased exposure for children. 1,3 butadiene is a known carcinogen and has been linked to increased risk of cardiovascular disease. Elimination of 1,3 butadiene can be accelerated by the supplementation of glutathione (reduced) either oral, intravenous, transdermal, or its precursor N-acetyl cysteine(NAC).

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

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

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Requisition #:	000000	**Physician Name:**	REGENERUS LAB
Patient Name:	Sample Report 44	**Date of Collection:**	10/19/2021
Patient Age:		**Time of Collection:**	07:00 AM
Sex:	F	**Print Date:**	11/10/2021
Specimen Id.:	000000-2		

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

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

3-hydroxypropylmercapturic acid (3-HPMA) (Marker 17) Acrolein is converted to the metabolite N-acetyl-S-(3-hydroxypropyl)-L-cysteine which is also termed 3-hydroxypropylmercapturic acid (3-HPMA). Acrolein is commonly used as an herbicide to control submersed and floating weeds and algae in irrigation canals. Crops using this irrigation water may be contaminated with acrolein. Acrolein, a highly reactive unsaturated aldehyde, is a ubiquitous environmental pollutant and its potential as a serious environmental health threat is beginning to be recognized. Humans are exposed to acrolein per 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. Treatment of acrolein should consist of either N-acetylcysteine (NAC) or glutathione (GSH) supplementation, which stimulate the conversion of acrolein to 3-HPMA.

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Patient Name:	Sample Report 44	***Date of Collection:***	10/19/2021
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3-phenoxybenzoic acid (3PBA) (Marker 18) is the result of exposure to pyrethroid insecticides (pyrethrins). Pyrethrins are the collective name for a group of pesticidal compounds derived from pyrethrum flowers in the genus *Chrysanthemum* that includes permethrin, cypermethrin, deltamethrin, cyhalothrins, fenpropathrin and trihalomethrin. Pyrethroids are synthetic analogs of pyrethrins. Pyrethroids may affect neurological development, disrupt hormones, induce cancer, and suppress the immune system. Pyrethroids are axonic poisons that work by keeping the sodium channels open in the neuronal membranes. Inhaling high levels of pyrethrins or pyrethroids may bring about asthmatic breathing, sneezing, nasal stuffiness, headache, nausea, incoordination, tremors, convulsions, facial flushing and swelling, and burning and itching sensation. A 37-year-old woman died of cardiorespiratory arrest after shampooing her dog with pyrethrin shampoo. Individuals who have ragweed sensitivity are especially vulnerable to allergic reactions to these products. Mothers of children with autism spectrum disorder (ASD) were twice as likely to have reported using pet shampoos containing pyrethrins as those who had healthy children; the effect was most severe if exposure was during the second trimester of pregnancy. In addition, parents of ASD children have reported the first onset of autistic behavior after the use of pyrethrin insecticide sprays in the home.

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

Tiglylglycine (TG) (Marker 19) is associated with both mitochondrial and/or genetic disorders. Toxic chemical exposure may be one of the most common causes of mitochondrial dysfunction. In mitochondrial disorders of the respiratory chain, TG values are usually more moderately increased than in the genetic disorders. In the medical literature, a normal value is less than 3.8 mmol/mol creatinine in children.

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

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

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