



Order: 999999-9999



Client #: 12345

Doctor: Sample Doctor, MD
Doctors Data Inc.
3755 Illinois Ave.
St. Charles, IL 60174

Patient: Sample Report

Age: 33 DOB: 06/07/1985

Sex: Male

Body Mass Index (BMI): 24.4

Sample Collection Date/Time

Date Collected 08/06/2018

Wake Up Time 0900

Collection Period Second morning

Date Received 08/07/2018

Date Reported 08/07/2018

Analyte	Result	Unit per Creatinine	L	WRI	H	Reference Interval
Phenethylamine (PEA)	27	nmol/g				26 - 70
Tyrosine	112	µmol/g				28 - 75
Tyramine	1.9	µmol/g				1.6 - 3.2
Dopamine	211	µg/g				110 - 200
3,4-Dihydroxyphenylacetic acid (DOPAC)	311	µg/g				330 - 1000
3-Methoxytyramine (3-MT)	175	nmol/g				82 - 174
Norepinephrine	21	µg/g				18 - 42
Normetanephrine	133	µg/g				70 - 275
Epinephrine	4.3	µg/g				1.3 - 7.3
Metanephrine	55	µg/g				44 - 103
Norepinephrine / Epinephrine ratio	4.9					< 12
Tryptamine	0.3	µmol/g				0.10 - 0.75
Serotonin	83	µg/g				50 - 98
5-Hydroxyindolacetic acid (5-HIAA)	1450	µg/g				1600 - 6000
Glutamate	42	nmol/g				9.0 - 40.0
Gamma-aminobutyrate (GABA)	2.8	nmol/g				1.6 - 3.5
Glycine	2805	nmol/g				350 - 1500
Histamine	32	µg/g				12 - 30
Taurine	1111	µmol/g				240 - 900



Neurotransmitter Comments:

- Urinary neurotransmitter levels provide an overall assessment of the body's ability to make and break down neurotransmitters and are representative of whole body levels. They are required for neurotransmission throughout the body. Direct assessment of neurotransmitter levels and metabolism in the central nervous system is not clinically feasible and approximately twenty percent of the total urinary levels are derived from the brain. The enzymes, cofactors and precursors in neurotransmitter metabolism in general are the same in the periphery and in the central nervous system. Therefore, alterations in urinary neurotransmitter levels assessed in urine provide important clinical information, and may be associated with many symptoms including cognitive and mood concerns, diminished drive, fatigue and sleep difficulties, cravings, addictions and pain.
- Tyrosine is the non-essential amino acid precursor for dopamine, norepinephrine and epinephrine. Increased tyrosine may exacerbate migraine headaches and hyperthyroid conditions. Elevated tyrosine levels may occur due to supplementation (phenylalanine or tyrosine), heritable enzyme defects, or liver disease. Tyrosine hydroxylase converts tyrosine into the dopamine precursor L-DOPA; BH4, Vitamin D and iron are cofactors for that enzymatic activity.
- Elevated dopamine may be associated with increased worry, distrust of others and decreased ability to interact socially and is often found in patients with attention deficits and hyperactivity. Medications that may increase dopamine levels include L-dopa, methyl dopa, select antidepressants and some ADD/ADHD medications. L-theanine may modulate catecholamine effects. Metabolism requires vitamins B2, B3, SAMe, magnesium, and iron, while conversion to norepinephrine requires vitamin C, copper and vitamin B3.

Notes:

RI= Reference Interval, L (blue)= Low (below RI), WRI (green)= Within RI (optimal), WRI (yellow)= Within RI (not optimal), H (red)= High (above RI)

Methodology: LCMS QQQ

- DOPAC levels may be low simply if dopamine levels are low. DOPAC is the primary metabolite of dopamine formed by MAO activity. Alternatively low levels of DOPAC may be associated with medications such as adrenergic antagonists, monoamine or norepinephrine reuptake inhibitors. Low DOPAC levels may also indicate low activity of MAO-A. Vitamins B2, B3, B6, and iron are required for optimal dopamine metabolism. Although a low level of urinary DOPAC is absolutely not diagnostic, low DOPAC levels in cerebral spinal fluid have been associated with Parkinson's disease.
- 3-MT may be increased if dopamine is high; rule out use of L-DOPA. 3-MT is formed by direct metabolism of dopamine by COMT. Very high levels of 3-MT may have stimulatory effects. 3-MT levels may increase during acute stress. Herbicides, such as paraquat, have been shown to increase 3-MT levels in animals. Consumption of foods rich in catecholamines (bananas, pineapple, walnuts) may acutely increase urinary levels of 3-MT. Deficiency or inhibition of MAO may increase 3-MT levels. MAO may be inhibited by cigarette smoke or medications such as monoamine oxidase inhibitors. Vitamins C, B2, B3, SAMe, magnesium, copper and iron are required for optimal dopamine metabolism.
- Low 5-HIAA may be associated with mood concerns including depression and anxiety, sleep changes, and poor concentration. Low 5-HIAA may be associated with low precursor serotonin, or compromised metabolism of serotonin by MAO-A. Some medications, including aspirin, MAO-inhibitors, levodopa, and tricyclic antidepressants may decrease 5-HIAA levels. MAO may be inhibited by cigarette smoke. Vitamins B2, B3, B6, magnesium and iron are required for optimal serotonin metabolism. If MAO-A enzyme function is inhibited, serotonin may be elevated and 5-HIAA will be low.
- Elevated glutamate may contribute to anxiety, poor concentration, attention deficits and hyperactive tendencies as well as poor sleep and nighttime awakening. Glutamate may be increased in association with hypoglycemia, Alzheimer's, ALS and chronic compromised blood flow to the brain. Possible sources of increased glutamate include MSG, yeast extract and other hidden sources of free glutamic acid. L-theanine may modulate elevated glutamate levels and attenuate glutamate signaling, and taurine may provide protection from excitotoxicity and neuroinflammation.
- Glycine is a non-essential amino acid that acts as an inhibitory neurotransmitter in the central nervous system. Elevated glycine levels may be associated with compromised cognitive processing. Elevated levels may be seen with glycine supplementation. Glycine may be given in conjunction with pharmaceutical agents when supporting schizophrenia or psychosis. Lipoic acid may enhance glycine break down. Break down of glycine requires vitamin B6 and tetrahydrofolate as cofactors. Note: High levels of glycine may interact with clozapine and decrease its clinical efficacy.
- Elevated histamine may be associated with allergy-like symptoms, gastro-intestinal concerns, skin itch/inflammation (pruritis), increased wakefulness and insomnia, and has been demonstrated in gastrointestinal blastocystis infections. Levels may be elevated due to use of histamine-releasing medications, consumption of allergenic and sulfite-rich foods and/or histamine-rich foods, dysbiotic bacterial production in the intestine and zinc deficiency. High urine (and blood) histamine levels have been associated with cluster and cyclic headaches. Break down of histamine requires SAMe and copper.
- Taurine is an essential amino acid that may have inhibitory effects on CNS neurons. High urinary levels of taurine may be associated with stress reactions, depression, autism and psychosis. Symptoms may include apathy, sleep changes, irritability, recklessness, poor concentration, aches and pains, or social withdrawal. Patients with Cushing's syndrome (high cortisol) may have elevated urinary taurine levels. Urinary taurine levels may be high with acute or chronic kidney damage, inherited kidney disorders, liver inflammation, or gastrointestinal dysbiotic bacterial or yeast over growth. Oral supplementation may raise taurine levels; taurine is an ingredient in many "energy drinks". High taurine levels may compete with glycine N-methyl-D-aspartate receptors (NMDR). Chronically high taurine excretion may deplete intracellular magnesium and calcium.
- Considerations to address the demonstrated imbalances beyond the identified co-factors and amino acid precursors may include dosage adjustments if indicated, as well as nervine and adaptogenic herbs, methylation support, vitamin D, and gastrointestinal health optimization.

