Test Results

ZRT LABORATORY
8605 SW Creekside Place
Beaverton, OR 97008
Phone: 503-466-2445 Fax: 503-466-1636
info@zrtlab.com www.zrtlab.com

D2016 08 25 003 U

Samples Arrived: 08/25/2016
Date Closed: 08/25/2016

Samples Collected:
Urine: 08/22/16 06:45
Urine: 08/22/16 09:00
Urine: 08/22/16 18:30
Urine: 08/22/16 22:45

Ordering Provider:

Getuwell
8605 SW Creekside Pl
Beaverton, OR 97008

Anxious Alishia

BMI: 24.2
Height: 5 ft 6 in
Weight: 150 lb
Waist: Unspecified

Menses Status: Pre-Menopausal
Gender: Female
Last Menses: Unspecified
DOB: 5/27/1984 (32 yrs)
Patient Ph#: Unspecified

Test Name	Result		Range
Inhibitory Neurotransmitters (µg/g Cr)			
Serotonin (Urine)	32	L	47.6-140.3 (Optimal 61.0-103.2)
5-HIAA (Urine)	11800		2205-11816 (Optimal 2988-5850)
GABA (Urine)	142	L	167-463 (Optimal 193-367)
Glycine (Urine)	124		41-295 (Optimal 61-159) (mg/g Cr)
Excitatory Neurotransmitters (µg/g Cr)			
Glutamate (Urine)	5000	H	1213-4246 (Optimal 1515-2710)
Histamine (Urine)	23		7.6-35.4 (Optimal 10.1-22.3)
PEA (Urine)	40	H	3.6-38.8 (Optimal 5.3-16.1)
Dopamine (Urine)	60	L	103-282 (Optimal 144-240)
DOPAC (Urine)	370	L	495-2456 (Optimal 658-1449)
HVA (Urine)	3000	L	3025-9654 (Optimal 3737-7048)
Norepinephrine (pooled) (Urine)	8	L	10.0-35.7 (Optimal 15.0-28.1)
Normetanephrine (Urine)	15		13.4-44.8 (Optimal 17.9-31.7)
Epinephrine (pooled) (Urine)	1		0.8-6.2 (Optimal 1.4-4.2)
Ratio: Norepi/Epi (Urine)	8		2.9-25.2 (Optimal 5.2-13.7)
VMA (Urine)	2500		1996-5939 (Optimal 2580-4766)
Urinary Creatinine (mg/mL)			
Creatinine (pooled) (Urine)	1		0.3-2.0

<dL = Less than the detectable limit of the lab.
N/A = Not applicable; 1 or more values used in this calculation is less than the detectable limit.

Therapies

None

David T. Zava, Ph.D.
(Laboratory Director)

Alison McAllister, ND
(Ordering Provider unless otherwise specified on pg1)

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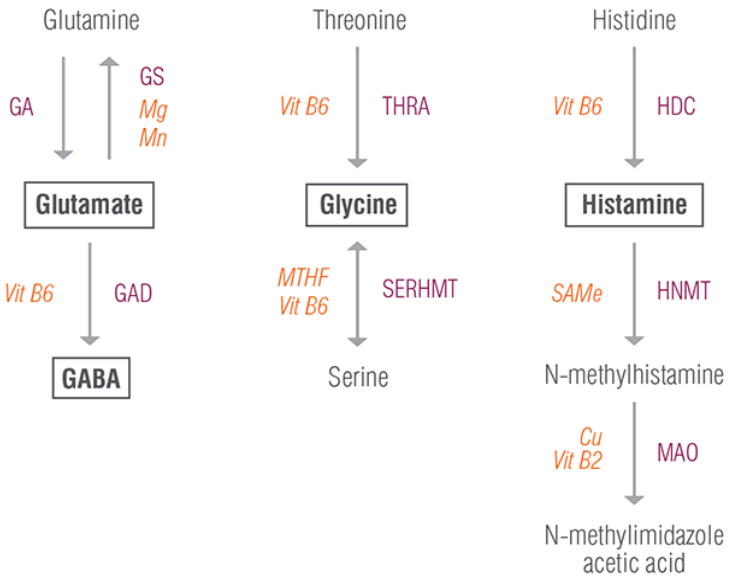
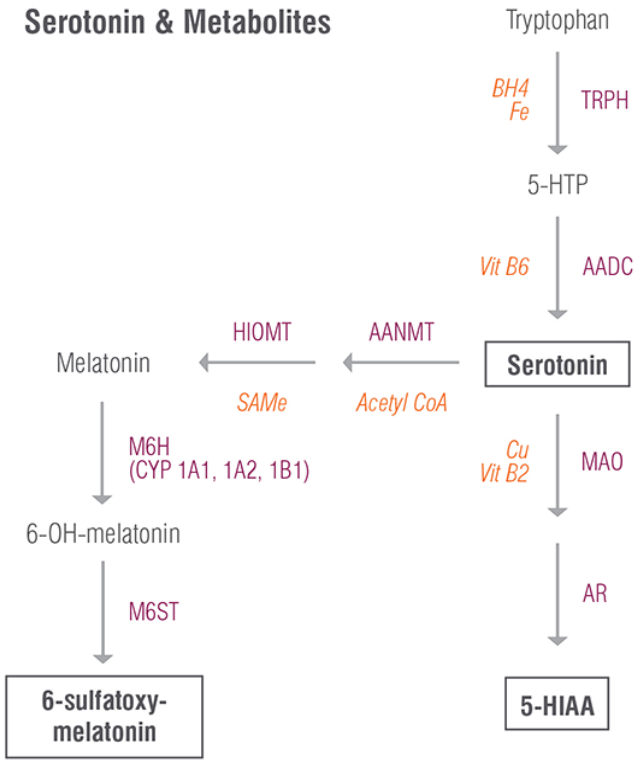
NEUROTRANSMITTER CASCADE

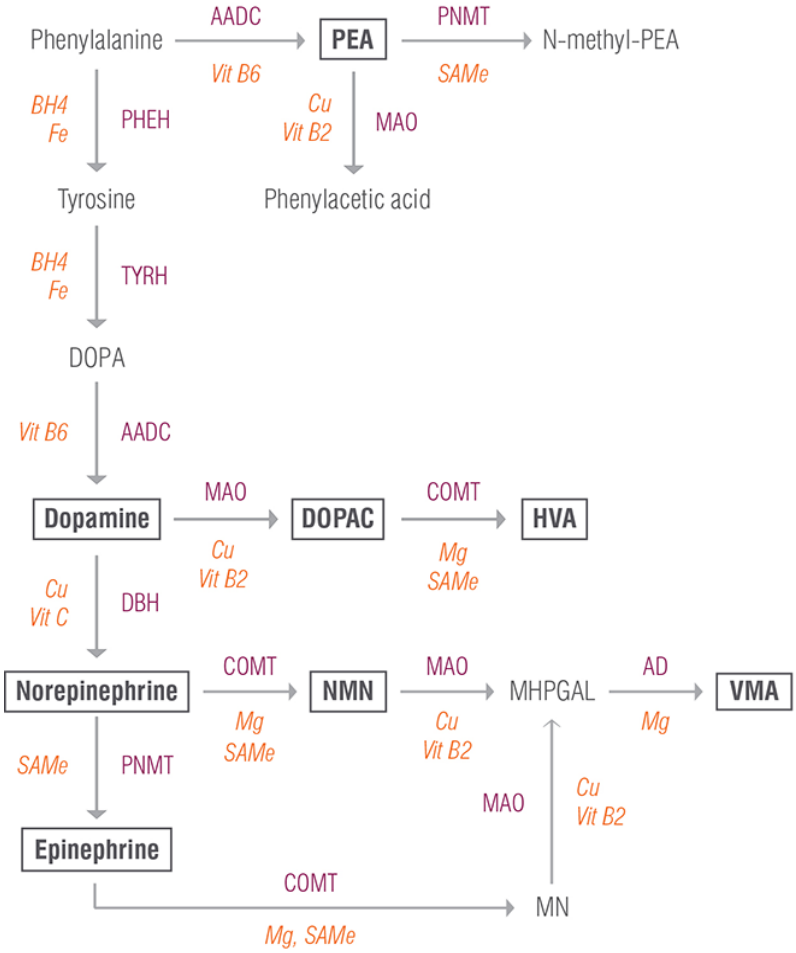
Glutamate/GABA, Glycine & Histidine



Serotonin & Metabolites



Catecholamines & Metabolites



Neurotransmitters & Metabolites:

HVA	homovanillic acid
NMN	normetanephrine
PEA	phenethylamine
VMA	vanillylmandelic acid
5-HIAA	5-hydroxyindole 3-acetic acid

Enzymes:

AADC	aromatic L-amino acid decarboxylase
AANMT	arylalkylamine N-methyltransferase
AD	aldehyde dehydrogenase
AR	aldehyde reductase
COMT	catechol-O-methyltransferase
DBH	dopamine beta hydroxylase
GA	glutaminase
GAD	glutamate decarboxylase
GS	glutamine synthetase
HDC	histidine decarboxylase
HIOMT	hydroxyindole-O-methyltransferase
HNMT	histamine N-methyltransferase
MAO	monoamine oxidase
M6H	melatonin 6 hydroxylase
M6ST	melatonin 6 sulfotransferase
PHEH	phenylalanine hydroxylase
PNMT	phenylethanolamine N-methyltransferase
SERHMT	serine hydroxymethyltransferase
THRA	threonine aldolase
TRPH	tryptophan hydroxylase
TYRH	tyrosine hydroxylase

Cofactors:

BH4	tetrahydrobiopterine
Cu	copper
Fe	iron
Mg	magnesium
Mn	manganese
MTHF	methyltetrahydrofolate
SAMe	S-adenosyl methionine

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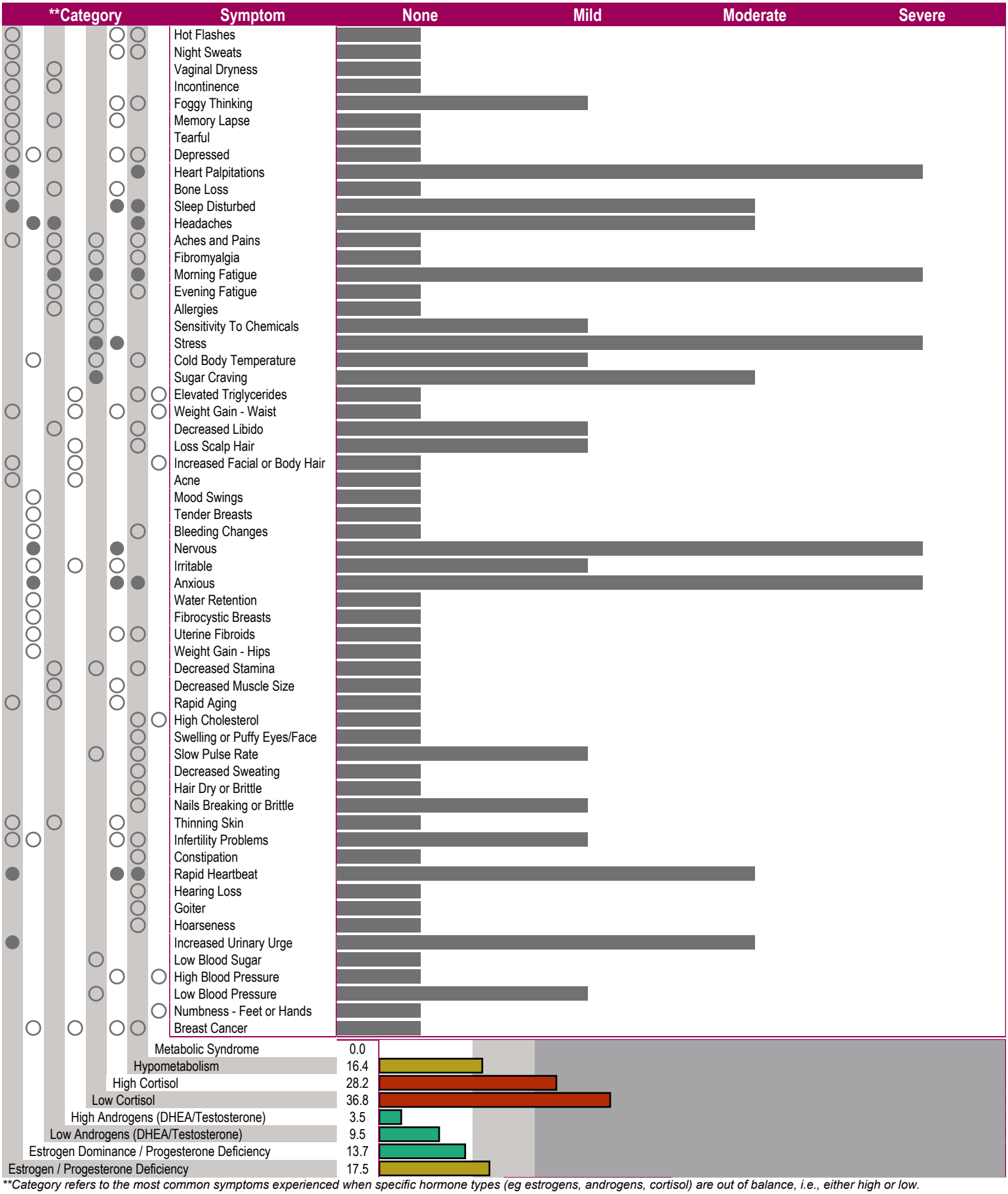

***Category refers to the most common symptoms experienced when specific hormone types (eg estrogens, androgens, cortisol) are out of balance, i.e., either high or low.*

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Lab Comments

Serotonin is lower than the reference range. Generally regarded as the happiness molecule, serotonin has calming effects and contributes to the feelings of well-being. Serotonin elevates mood, decreases anxiety, appetite, and libido, improves sleep and memory, eases depression, and helps regulate body temperature. Most of serotonin in the human body is produced in the gastrointestinal tract, where it stimulates gut motility. Research shows that urinary serotonin levels are reduced in patients with depression (Nichkova et al., 2012). Clinically, low serotonin is associated with anxiety, depression, changes in appetite, cravings, excessive worry, heightened sensitivity to pain, hot flashes, hunger, low mood, migraine, obsessive compulsive disorder, panic disorder, sleep disturbances, and worsened PMS symptoms. When serotonin is low, supplementation with cofactors to promote biosynthesis (e.g. vitamin B6), precursors (tryptophan/5-HTP), L-theanine, and probiotics may be helpful (Patterson et al., 2014;Pamela Wartian Smith, 2008;Strasser et al., 2016). Additionally, lifestyle modifications, such as regular exposure to bright light, healthy diet, sufficient exercise, and positive self-talk are all effective strategies that result in increased serotonin levels (Young, 2007)

5-hydroxyindoleacetic acid (5-HIAA) is within reference range. 5-HIAA is the primary metabolite of serotonin via the actions of monoamine oxidase and aldehyde dehydrogenase enzymes.

GABA is lower than the reference range. The brain's major inhibitory neurotransmitter GABA, functions as the off switch in the brain. GABA is essential to limiting excitation so that input signals are balanced and not overdone. GABA prevents anxiety, improves mood, promotes sleep, lowers blood pressure, acts as a muscle relaxant, aids in formation and storage of fear memories, increases insulin secretion and decreases blood glucose levels. Clinically, low GABA levels are implicated in anxiety, depression, headaches, menopause symptoms, panic attacks, post-traumatic stress disorder, and sleep difficulties. LowGABA levels may also be associated with adrenal distress and HPA axis dysfunction, and disorders like attention deficithyperactivity disorder and Tourette syndrome (Perlmutter and Loberg, 2015). With low GABA, supplementation with GABA, L-theanine, cofactor support (e.g. B6), growth hormone-releasing hormone, Ginko biloba, Ashwagandha, Kava, Valerian root,Melissa off (lemon balm), Scutellaria sinensis (skullcap), Gotu Cola, Magnolia and Phellodendron bark, and probiotics may behelpful (Alramadhan et al., 2012;Awad et al., 2007;Alexeev et al., 2012;Dhakal et al., 2012). Additionally, yoga (Streeter et al.,2012) and meditation (Guglietti et al., 2013) increase brain GABA levels.

Glycine is within normal range. Glycine plays a dual role as a neurotransmitter and a building block of proteins. Glycine serves as an anti-inflammatory agent, calms aggression, improves sleep quality, regulates locomotion, stabilizes blood sugar, and modulates excitatory signals in the brain.

Glutamate is elevated. The brain's major excitatory neurotransmitter glutamate (also known as glutamic acid) functions as the on switch in the brain. Glutamate regulates appetite, thinking (cognition), increases gut motility, optimizes learning, modulates memory, improves libido, decreases sleep and contributes to oxidative stress. Chronic stress maintains high levels of glutamate in the brain which may lead to excitotoxicity and even neuronal damage (Gold, 2015;Popoli et al., 2012). Research shows that urinary glutamate levels are high in patients with celiac disease (MARKO et al., 1960) and with hyperthyroidism (Belanger et al., 1972). Clinically, high glutamate is suspected in anxiety, autism, bipolar disorder, depression, and impulsivity, inability to focus (racing thoughts), obsessive compulsive disorder, panic attacks, sleep difficulties, and stroke. When glutamate is high, calming GABA, L-theanine, and taurine may be beneficial to counter glutamate actions. Vitamin E and N-Acetyl Cysteine (NAC) may be used to combat oxidative damage. Cofactor supplementation with vitamins B3 and B6, and magnesium and NAC may aid with glutamate metabolism.

Histamine is within reference range. Histamine plays a dual role in the body as a neurotransmitter and a modulator of the immune system. Histamine has anti-pain properties, plays a neuroprotective role in the brain, and contributes to optimal maintenance of cognition and memory. Histamine stimulates wakefulness and decreases sleep, stimulates gastric acid production, increases metabolism, suppresses appetite, and prevents weight gain. Histamine is a potent vasodilator and a pro-inflammatory agent.

PEA is elevated. PEA, also known as phenethylamine, promotes energy, elevates mood, and regulates attention. PEA also contributes to aggression, serves as a biomarker for ADHD, and prolongs the signaling of dopamine, norepinephrine, and serotonin. Urinary PEA levels increase after amphetamine use (Kusaga et al., 2002;Zametkin et al., 1984), exercise (Szabo et al., 2001), and in the following disorders: bipolar disorder (Karoum et al., 1982), phenylketonuria (Reynolds et al., 1978), schizophrenia (O'Reilly and Davis, 1994), postpartum period (Taylor et al., 1996), and in severe anxiety and insomnia (DeLisi et al., 1984). High PEA is suspected in the etiology of anxiety, inflammation, inability to focus (racing thoughts), sleep difficulties, and toxicity. When PEA is high, methylation cofactor support to aid metabolism may be beneficial.

Dopamine is lower than the reference range. Dopamine improves attention, focus, and motivation, helps with decision making, modulates movement control, promotes lactation, increases blood pressure, urine output and sodium excretion, and allows for feelings of reward and pleasure. Additionally, the quest for dopamine stimulation plays a central role in the etiology of addiction. Dopamine also serves as the parent precursor to norepinephrine and epinephrine. Research shows that urinary dopamine levels are reduced in patients with Alzheimer's disease (Liu et al., 2011), anorexia nervosa (Van Binsbergen et al., 1991), anxiety with

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depression (Field et al., 2010), fibromyalgia (Riva et al., 2012), and periodic limb movement disorder (Cohrs et al., 2004). Clinically, low dopamine is implicated in addiction, apathy, cravings, depression, fatigue, impulse control issues, increased sensitivity to pain, low libido, low mood, memory issues, sleep disturbances, and weight control issues. When dopamine is low, supplementation with precursors (tyrosine or L-DOPA) and/or cofactors (iron, vitamin B6, tetrahydrofolate) to promote biosynthesis may be beneficial.

DOPAC is lower than the reference range. DOPAC is the primary metabolite of dopamine formed via the actions of monoamine oxidase. Research shows that DOPAC is reduced in the urine of patients with Alzheimer's disease (Liu et al., 2011).

Homovanillic acid (HVA) is lower than the reference range. HVA is a dopamine metabolite formed through the actions of the monoamine oxidase (MAO) and catechol-O-methyl transferase (COMT) enzyme. Research shows that HVA is reduced in the urine of patients with monoamine oxidase enzyme deficiency (Sims et al., 1989), polycystic ovarian syndrome (Shoupe and Lobo, 1984), and periodic limb movement disorder (Cohrs et al., 2004).

Norepinephrine is lower than the reference range. Norepinephrine functions both as a neurotransmitter and a hormone, participating in the body's fight or flight response. Norepinephrine increases alertness, focuses attention, fine-tunes vigilance, increases blood pressure, heart rate, and blood glucose, reduces digestive activity, pain and sleep, prevents bladder emptying, and regulates body temperature. The adrenal gland produces approximately 20% of norepinephrine with 80% produced by the sympathetic nerve fibers. Research shows that urinary norepinephrine is reduced in patients with Alzheimer's disease. Clinically, low norepinephrine isimplicated in anorexia, attention impairment, depression, fatigue, hypotension, lack of motivation, lethargy, low mood, memoryissues, slow pulse rate, and weight issues. If norepinephrine is low, precursor supplementation with tyrosine or phenylalanine, orcofactor support with ascorbic acid, iron, tetrahydrofolate, and vitamin B6 may be beneficial.

Normetanephrine is within reference range. Normetanephrine is a norepinephrine metabolite formed via the actions of catechol-O-methyl (COMT) transferase enzyme in response to stress.

Epinephrine is within reference range. Epinephrine, also called adrenalin, functions both as a neurotransmitter and a hormone, participating in the body's fight or flight response. Epinephrine increases alertness, focuses attention, fine-tunes vigilance, increases blood pressure, heart rate, and blood glucose, reduces digestive activity, pain and sleep, prevents bladder emptying, and regulates body temperature.

Vanillylmandelic acid (VMA) is within reference range. VMA is a norepinephrine and epinephrine metabolite formed via the actions of monoamine oxidase, catechol-O-methyl transferase (COMT), and aldehyde dehydrogenase.

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