

Test Results



8605 SW Creekside Place
Beaverton, OR 97008
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2017 02 24 001 SU



Ordering Provider:

Samples Arrived: 02/24/2017
Date Closed: 02/28/2017

Samples Collected: Saliva: 02/21/17 06:30
Urine: 02/21/17 06:30
Urine: 02/21/17 12:15
Urine: 02/21/17 20:15
Urine: 02/20/17 22:30

Getuwell Clinic
8605 SW Creekside Pl
Beaverton, OR 97008

Menopausal Mary
111 N Main St
Hillsboro, OR 97123

Menses Status: Postmenopausal
Gender: Female

Last Menses: 01/08/2013
DOB: 11/13/1953 (63 yrs) Patient Ph#: 555 555 5555

BMI: 18.5
Height: 5 ft 7 in
Weight: 118 lb
Waist: 25 in

Test Name	Result	Range
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Salivary Steroids

Estradiol	<0.5	L 0.5-1.7 pg/mL Postmenopausal (optimal 1.3-1.7)
Progesterone	20	12-100 pg/mL Postmenopausal
Ratio: Pg/E2	N/A	Optimal: 100-500 when E2 1.3-3.3 pg/mL
Testosterone	30	16-55 pg/mL (Age Dependent)
DHEAS	2.5	2-23 ng/mL (Age Dependent)
Cortisol	5.8	3.7-9.5 ng/mL (morning)

Urinary Inhibitory Neurotransmitters

Serotonin	45.6	L 47.6-140.3 µg/g Cr (Optimal 61.0-103.2)
5-HIAA	4986	2205-11816 µg/g Cr (Optimal 2988-5850)
GABA	234	167-463 µg/g Cr (Optimal 193-367)
Glycine	53	41-295 mg/g Cr (Optimal 61-159)

Urinary Excitatory Neurotransmitters

Glutamate	1424	1213-4246 µg/g Cr (Optimal 1515-2710)
Histamine	11.6	7.6-35.4 µg/g Cr (Optimal 10.1-22.3)
PEA	29.2	3.6-38.8 µg/g Cr (Optimal 5.3-16.1)
Dopamine	111	103-282 µg/g Cr (Optimal 144-240)
DOPAC	1831	495-2456 µg/g Cr (Optimal 658-1449)
HVA	5915	3025-9654 µg/g Cr (Optimal 3737-7048)
Norepinephrine (pooled)	7.2	L 10.0-35.7 µg/g Cr (Optimal 15.0-28.1)
Normetanephrine	13.4	13.4-44.8 µg/g Cr (Optimal 17.9-31.7)
Epinephrine (pooled)	0.7	L 0.8-6.2 µg/g Cr (Optimal 1.4-4.2)
Ratio: Norepi/Epi	10	2.9-25.2 (Optimal 5.2-13.7)
VMA	2913	1996-5939 µg/g Cr (Optimal 2580-4766)

Urinary Free Diurnal Cortisol

Free Cortisol	91.11	H 7.8-29.5 µg/g Cr (1st Morning)
Free Cortisol	48.39	23.4-68.9 µg/g Cr (2nd Morning)
Free Cortisol	4.50	L 6.0-19.2 µg/g Cr (Evening)
Free Cortisol	7.82	2.6-8.4 µg/g Cr (Night)

Urinary Free Diurnal Cortisone

Free Cortisone	213.23	H 31.6-91.6 µg/g Cr (1st Morning)
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The above results and comments are for informational purposes only and are not to be construed as medical advice. Please consult your healthcare practitioner for diagnosis and treatment.

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

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

CLIA Lic # 38D0960950
Composed by: 1165812490 at 4/7/2017 10:20:34 AM

Test Name	Result	Range
Free Cortisone	208.01	H 63.3-175.8 µg/g Cr (2nd Morning)
Free Cortisone	20.36	L 30.6-88.5 µg/g Cr (Evening)
Free Cortisone	40.22	15.5-44.7 µg/g Cr (Night)

Urinary Diurnal Melatonin MT6s

Melatonin	16.45	L 18.0 - 40.9 µg/g Cr (1st Morning)
Melatonin	3.54	L 7.3 - 31.9 µg/g Cr (2nd Morning)
Melatonin	1.83	0.7 - 2.2 µg/g Cr (Evening)
Melatonin	8.64	1.7 - 11.1 µg/g Cr (Night)

Urinary Diurnal Norepinephrine

Norepinephrine	8.35	L 9.4-22.0 µg/g Cr (1st Morning)
Norepinephrine	<dl	L 12.6-38.2 µg/g Cr (2nd Morning)
Norepinephrine	13.98	L 21.1-42.9 µg/g Cr (Evening)
Norepinephrine	5.55	L 16.9-38.8 µg/g Cr (Night)

Urinary Diurnal Epinephrine

Epinephrine	0.63	0.5-1.5 µg/g Cr (1st Morning)
Epinephrine	<dl	L 0.7-6.1 µg/g Cr (2nd Morning)
Epinephrine	1.13	L 2.3-8.1 µg/g Cr (Evening)
Epinephrine	<dl	L 1.2-4.2 µg/g Cr (Night)

Urinary Creatinine

Creatinine (pooled)	0.58	0.3-2.0 mg/mL
Creatinine	0.50	0.3-2.0 mg/mL (1st morning)
Creatinine	0.41	0.3-2.0 mg/mL (2nd morning)
Creatinine	1.42	0.3-2.0 mg/mL (Evening)
Creatinine	0.54	0.3-2.0 mg/mL (Night)

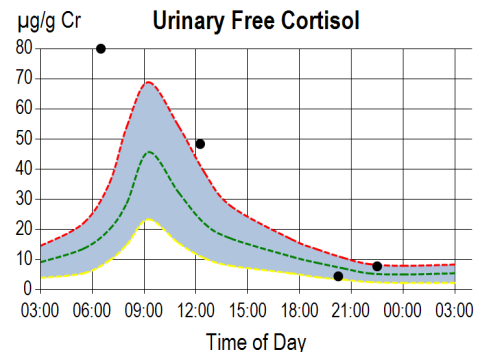
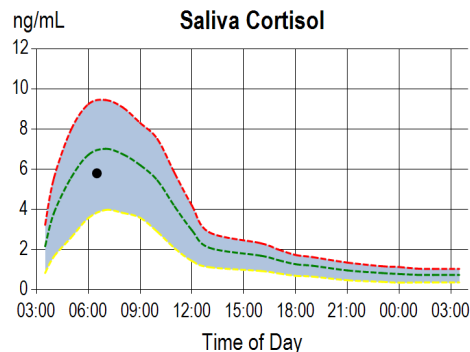
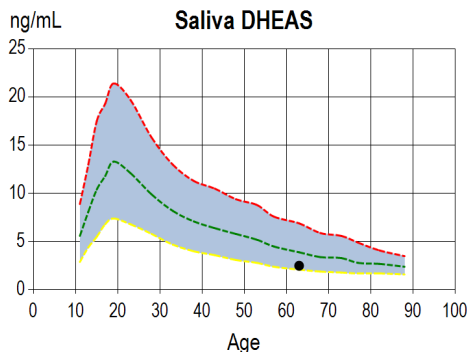
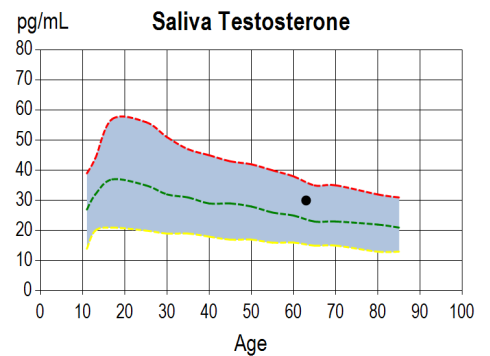
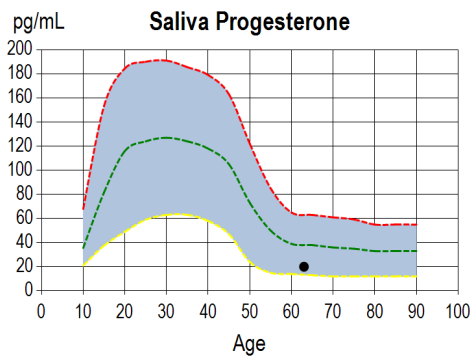
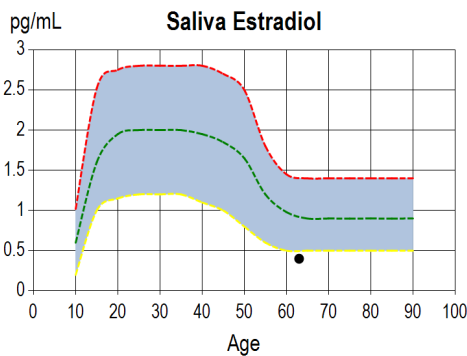
<dl = Less than the detectable limit of the lab.

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

Therapies

Vitex

Disclaimer: Graphs below represent hormone levels in testers not using hormone supplementation and are provided for informational purposes only. Please see comments for additional information if results are higher or lower than expected. Graph key ---High ---Avg ---Low

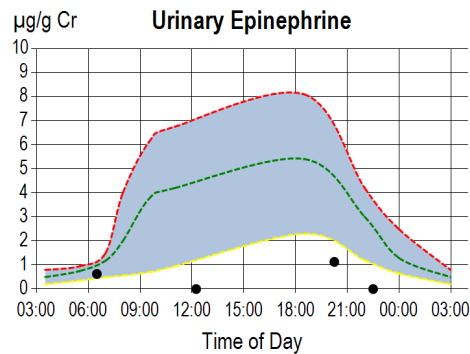
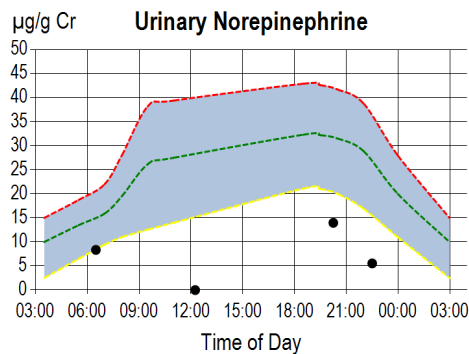
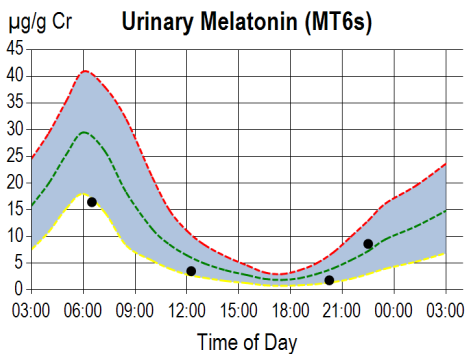
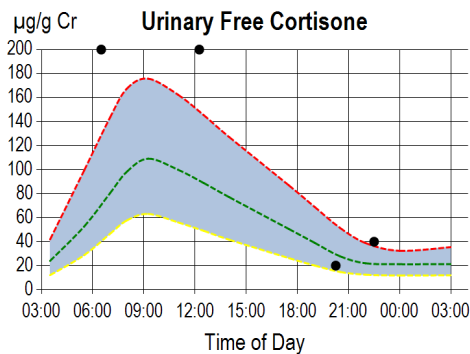


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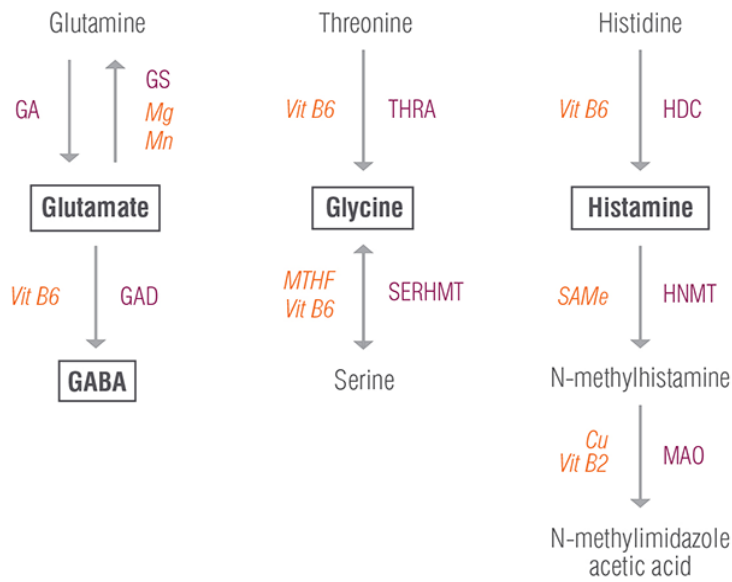
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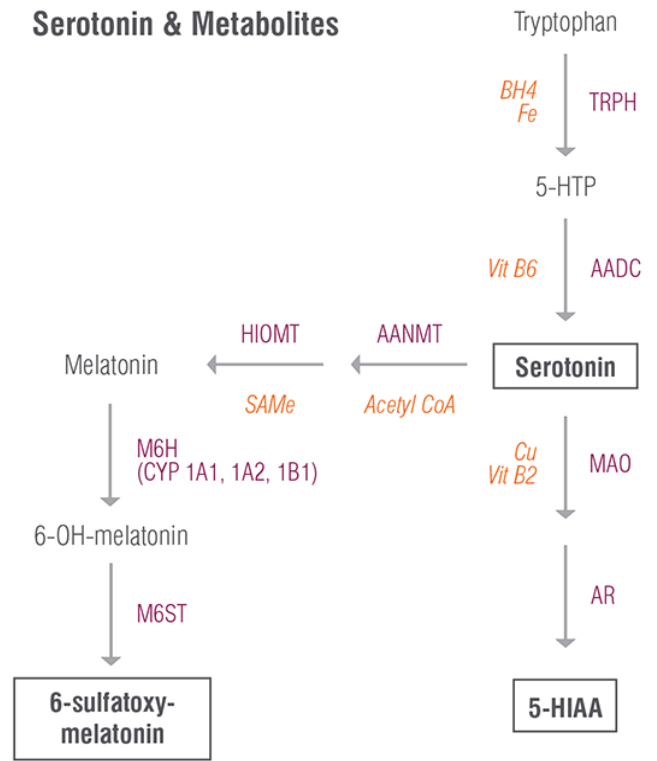


NEUROTRANSMITTER CASCADE

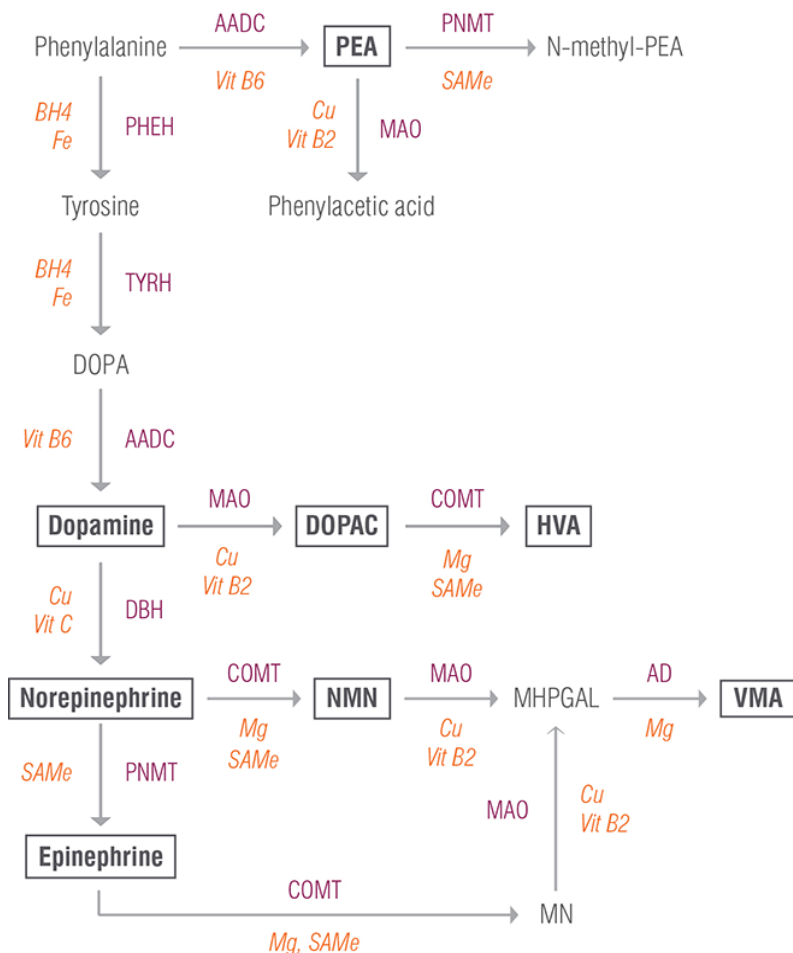
Glutamate/GABA, Glycine & Histidine



Serotonin & Metabolites



Catecholamines & Metabolites



Neurotransmitters & Metabolites:

HVA
NMN
PEA
VMA
5-HIAA

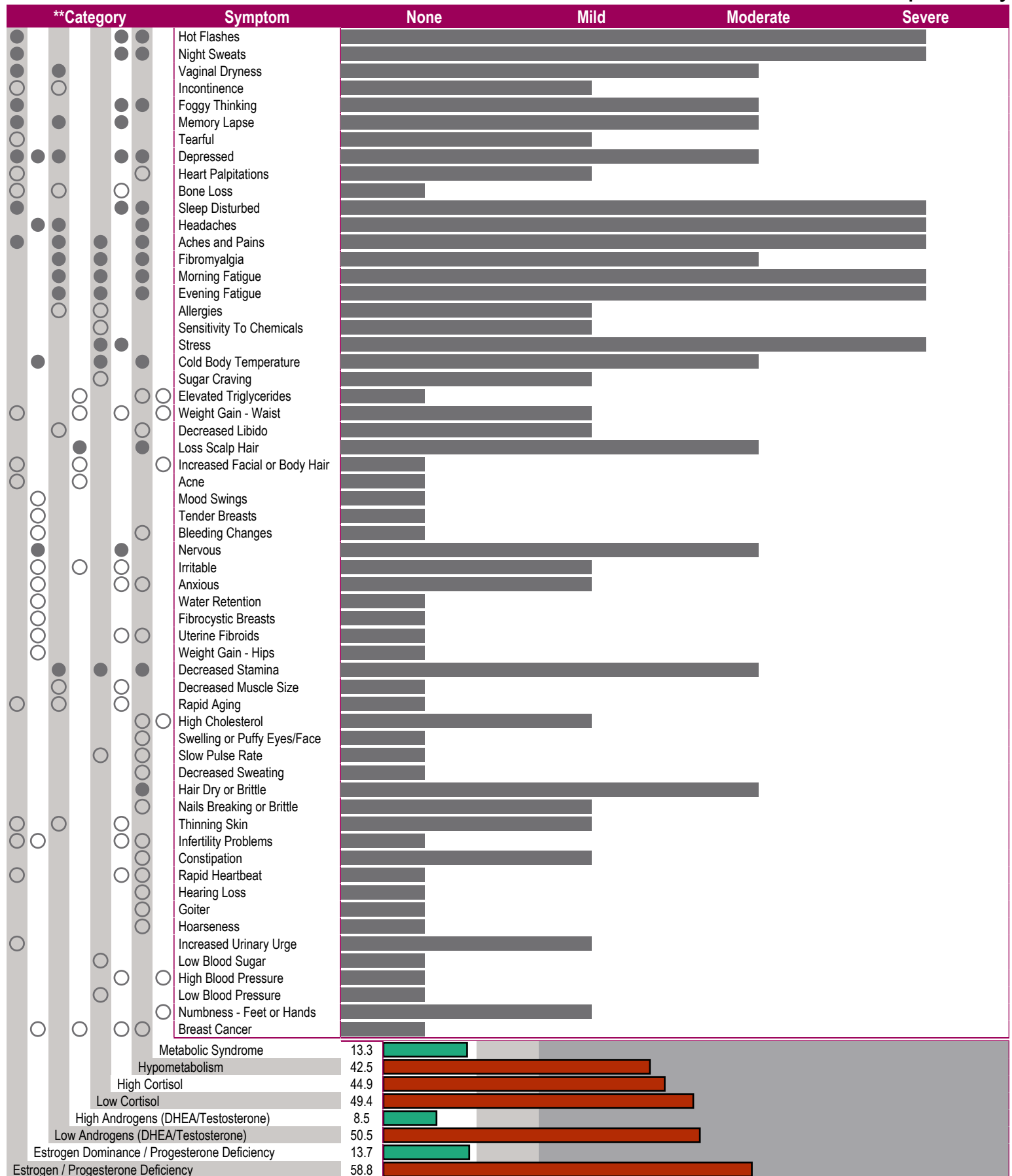
homovanillic acid
normetanephrine
phenethylamine
vanillylmandelic acid
5-hydroxyindole 3-acetic acid

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



**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.

Lab Comments

GENERAL COMMENTS: All monoamine neurotransmitters tested in urine (serotonin, dopamine, norepinephrine, epinephrine) are low or low-normal, whereas their downstream metabolites are within normal ranges. This indicates that the activity of MAOA is very high, which rapidly inactivates serotonin to 5-HIAA, dopamine to DOPAC and HVA, norepinephrine to NMN and VMA, and epinephrine to VMA. PEA, which is metabolized by MAOB is within high-normal range, indicating that MAOA appears to be selectively enhanced. Consider that herbal adaptogens may contribute to high MAOA. In addition, saliva and urine metabolite assays indicate very low estradiol and other endogenous estrogens, which are associated with higher MAOA activity (estrogens suppress MAOA).

INHIBITORY NEUROTRANSMITTERS**SEROTONIN**

Serotonin is lower than the reference range; however, its downstream metabolite 5-HIAA is within normal reference range. This suggests high monoamine oxidase A activity (MAOA), which metabolizes serotonin to 5-HIAA (expect low serotonin/high 5-HIAA). MAOA is inhibited by premenopausal levels of estradiol; therefore, low estradiol as seen in saliva and urine metabolite test results from samples collected at the same time, has likely contributed to higher MAOA. Estradiol is important for not only suppressing MAOA activity, but also for increasing synthesis of serotonin and serotonin receptors in neurons. Severe symptoms experienced by this individual are likely due to a very low level of estradiol and consequent low serotonin and serotonin receptors. Consider estrogen therapy if no contraindications.

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 many of the symptoms self-reported by this individual, which include some of the following: 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.

TREATMENT: In addition to consideration of estrogen therapy, balanced with natural progesterone (assuming no contraindications) also consider supplementation with cofactors to promote serotonin biosynthesis. Vitamin B6, serotonin 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-HIAA

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

GABA is within 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.

GLYCINE

Glycine is low-normal (<20th percentile). 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. Clinically, lower glycine levels are suspected in anxiety.

TREATMENT: Glycine supplementation, B6, Serine support, B6 and MTHF may all support the production of glycine.

EXCITATORY NEUROTRANSMITTERS**GLUTAMATE**

Glutamate is low-normal (< 20th percentile). The brain's major excitatory neurotransmitter glutamate functions as the "on" switch in the brain. Glutamate regulates appetite, thinking, increases gut motility, optimizes learning, modulates memory, improves libido, and decreases sleep. Low urinary glutamate levels have been reported in patients with migraines (Ragginer et al., 2012). Clinically, lower glutamate levels may contribute to agitation, depression, chronic fatigue, lack of concentration, low energy levels, and sleep difficulties.

TREATMENT: L-glutamine may be beneficial to restore glutamate to normal values.

HISTAMINE

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

PEA is high-normal (>80th percentile). 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. PEA is inactivated by MAOB metabolism. Drugs such as Selegiline (L-Deprenyl) cause irreversibly inactivated MAOB, resulting in higher levels of PEA. Urinary PEA levels increase after amphetamine use (Kusaga et al., 2002; Zametkin et al., 1984), exercise (Szabo et al., 2001), Selegiline (L-Deprenyl) 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, sleep difficulties, and toxicity.

TREATMENT: Methylation cofactor support to aid metabolism may be beneficial.

DOPAMINE

Dopamine is low-normal (<20th percentile). 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. While Dopamine is within lower-normal range, its downstream metabolites (DOPAC and HVA) are within the upper-normal range. Dopamine is inactivated by MAOA, which is increased in the absence of estrogens (see explanation above for Serotonin). 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 depression (Field et al., 2010), fibromyalgia (Riva et al., 2012), and periodic limb movement disorder (Cohrs et al., 2004). Clinically, low dopamine is also implicated in apathy, cravings, fatigue, impulse control issues, increased sensitivity to pain, low libido, low mood, memory issues, sleep disturbances, and weight control issues.

TREATMENT: Supplementation with precursors (tyrosine or L-DOPA) and/or cofactors (iron, vitamin B6, tetrahydrofolate) to promote biosynthesis may be beneficial.

DOPAC

DOPAC is high-normal (>80th percentile). DOPAC is the primary metabolite of dopamine formed via the actions of monoamine oxidase. Research shows that DOPAC is elevated in patients with anorexia nervosa (Van Binsbergen et al., 1991).

HVA

Homovanillic acid (HVA) is within reference range. HVA is a dopamine metabolite.

NOREPINEPHRINE

Norepinephrine is lower than the reference range. (See also Diurnal Norepinephrine.) Low Norepinephrine is likely due to low levels of its precursor Dopamine. 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 is implicated in anorexia, attention impairment, depression, fatigue, hypotension, lack of motivation, lethargy, low mood, memory issues, slow pulse rate, and weight issues.

TREATMENT: Precursor supplementation with tyrosine or phenylalanine, or cofactor support with ascorbic acid, iron, tetrahydrofolate, and vitamin B6 may be beneficial.

NORMETANEPHRINE

Normetanephrine is low-normal (<20th percentile). Low normetanephrine may not have clinical utility, but may be reflective of low norepinephrine levels.

EPINEPHRINE

Epinephrine is lower than the reference range. Epinephrine 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. Approximately 80% of peripheral catecholamine output by the adrenal glands accounts for epinephrine.

Research shows that urine epinephrine is decreased in Alzheimer's disease (Liu et al., 2011), metabolic syndrome (Landsberg et al., 1991), and obesity (Landsberg et al., 1991). Clinically, low epinephrine is implicated in attention impairment, chronic stress, depression, dizziness, chronic fatigue, hypotension, low mood and libido, and memory issues.

TREATMENT: Adrenal support may be beneficial to increase epinephrine levels.

NOREPINEPHRINE/EPINEPHRINE RATIO

NE/E ratio is within reference range.

VMA

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.

URINARY FREE CORTISOL (F) AND CORTISONE (E)

Urinary Free Cortisol (F) is following a normal circadian rhythm with the exception of the first morning void, which is higher than reference range. High first morning cortisol suggests high levels during the night as the first void represents the full night production of cortisol. Cortisol returns to a more normal level in the second morning void as well as the evening and at night before bed. Cortisone (E), the inert metabolite of F, is following a similar circadian rhythm and is high in the first two morning voids, but returns to normal levels in the evening and at night before bed.

Cortisol is converted to cortisone by the enzyme 11-beta hydroxysteroid dehydrogenase type II (11-beta HSD-II) (for review see: Seckl JR and Chapman KE Eur J Biochem 249, 361-364, 1997), which is expressed in tissues such as the kidneys, liver, lungs, colon, truncal fat, and salivary glands. This enzyme plays an important role in preventing excess buildup of cortisol, which at high level activates the mineralocorticoid receptor (at normal levels cortisol only activates the glucocorticoid receptors) and can lead to mineralocorticoid excess syndrome, causing high blood pressure and low potassium levels.

The activity of 11-beta HSD-II is increased with growth hormone, estrogens, and androgens. Estrogen replacement therapy in women or androgen (testosterone) replacement therapy in men will increase the activity of 11-beta HSD-II and accelerate conversion of cortisol to cortisone. This is why higher physiological levels of estrogens and androgens seen during younger years are associated with a smaller waist circumference (visceral or belly fat). As women approach menopause their estrogens drop and waistlines thicken. As men age and their testosterone drops, waistlines also increase as a result of increased belly fat. Estrogen and testosterone replacement therapies that return these hormones to youthful levels have been shown to reduce the increase in visceral adipose tissue common to the precipitous drop in estrogens that occur in menopause and the slower drop in androgen levels with aging in men. Some phytochemicals found in fruits and vegetables (e.g. quercetin, genistein), also play a role in regulating HSD activity. For example, licorice contains the compound glycyrrhetic acid that inhibits HSD type I activity, which lowers cortisol by preventing conversion of F to E.

Chronic high cortisol, particularly at night, leads to conditions such as weight gain in the waist, muscle and bone loss, depression, and immune suppression. Dysfunction of other hormones is closely associated with chronic excess cortisol. For example, tissue resistance to insulin, caused by chronically high cortisol, leads to insulin resistance/metabolic syndrome (expect to see symptoms characteristic of metabolic syndrome). Chronic high cortisol may also inhibit the actions of thyroid hormone synthesis and action, resulting in low thyroid symptoms. A persistently high night cortisol can eventually lower melatonin production, which is important for maintaining normal biorhythms and immune function.

Because chronic stressors and associated high night cortisol can have adverse effects on health and wellbeing, it is important to develop strategies to identify and eliminate or reduce the stressors that are causing high cortisol at night (see as high cortisol in the last or first morning voids) or consider bioidentical hormone replacement therapies, foods, and/or nutritional supplements that help control excessive accumulation of cortisol. For additional information about adrenal dysfunction and strategies for adrenal support and lowering stress/cortisol levels the following books and journal articles are worth reading: "Adrenal Fatigue," by James L. Wilson, N.D., D.C., Ph.D.; "The Cortisol Connection," by Shawn Talbott, Ph.D.; "The End of Stress As We Know It," by Bruce McEwen; "The Role of Stress and the HPA Axis in Chronic Disease Management" by Thomas Guillemins, PhD.

MELATONIN METABOLITE 6-SULFATOXYMELATONIN (MT6s)

The melatonin metabolite, 6-sulfatoxymelatonin (MT6s) is within low-normal range in the first and second urine voids, but increases to a high-normal level at night before bed. In a healthy individual MT6s should be at its highest level in the first morning void and then rise again with the onset of night, which is reflective of the dark period (night). During the night melatonin synthesis should peak and result in increased levels of the urinary metabolite MT6s that are measured in the first morning void. Lower levels of MT6s during these early morning time periods may reflect work during a night shift or staying up at night with excessive lighting (e.g. watching television). Some medications such as beta blockers (e.g. propranolol) used to control high blood pressure may also suppress melatonin synthesis during the night. Other medications such as oral contraceptives may increase melatonin synthesis. For a more comprehensive list of medications that can decrease or increase melatonin levels see: <http://umm.edu/health/medical/altmed/supplement-interaction/possible-interactions-with-melatonin>.

Melatonin, produced by the pineal gland in the brain and released into the circulation, rapidly enters tissues throughout the body where it carries out its restorative properties. Melatonin synthesis decreases with aging and calcification of the pineal gland, the latter of which can result in very low production of melatonin. As mentioned above, commonly used medications can decrease (e.g. beta blockers) or increase (hormone contraceptives) melatonin levels.

Melatonin is known to have many different beneficial effects in the body. It helps slow the aging process, is a potent anti-oxidant, inhibits formation and growth of tumors such as breast and prostate cancers, and helps regulate the synthesis of the sex-hormones estradiol and progesterone (melatonin increases progesterone and decreases estrogens) as well as their cellular receptors (decreases estrogen receptors and increases progesterone receptors). Melatonin down-regulates cellular estrogen receptors, which inhibits response of estrogen target tissues (e.g. breast, uterine, and prostate) to estrogens. Pineal calcification, which is accelerated with aging and diseases, including breast cancer, is associated with very low melatonin production at night. Low melatonin has been associated with many different dysfunctions and diseases such as immune dysfunction, neurodegenerative disorders (Alzheimer's disease, senile dementia), pain disorders, cardiovascular disease, cancers of the breast and prostate, and type 2 diabetes (Hardeland R. Aging and Disease 3 (2): 194-225, 2012).

For more general information about melatonin please see: <http://www.nlm.nih.gov/medlineplus/druginfo/natural/940.html>

DIURNAL NOREPINEPHRINE (NE) AND EPINEPHRINE (EPI)

Norepinephrine (NE) and epinephrine (EPI) are not following a normal circadian rhythm and are lower than the expected reference ranges throughout the day. Epinephrine is produced primarily in the adrenal medulla and urinary levels of this stress hormone provide a good assessment of adreno-medullary function. Generally speaking, increases in NE are reflective of psychosocial (e.g. emotional-psychological) perceived stressors, whereas increases in EPI are in response to acute physiological stressors (e.g. exercise, drop in blood glucose). Normal levels of NE and Epi are essential for normal cardiovascular function and maintenance of steady-state levels of blood glucose necessary for optimal brain function. Increases in urinary norepinephrine reflect stimulation of both renal adreno-medullary and peripheral sympathetic nerve endings. Excessive and prolonged exposure to NE and/or EPI can lead to downregulation of beta-adrenergic receptors, increased free radical production, and overall functional decline (Reuben DB. J Gerontology: Medical Sciences 2000, 55A (10), M618-M624). Many of the self-reported symptoms (e.g. mental and physical fatigue) suggest such functional decline and likely result from the inability of the adrenal medulla and peripheral nerves to respond normally to daily stressors (e.g. drop in blood sugar, physical exertion, typical daily emotional stressors). Consider means to support adrenal function (e.g. diet, exercise, nutritional support) and remove, as much as possible, the stressors (emotional, physical, chemical, pathogenic), that have caused the severe depletion of NE and Epi seen in this individual.