

(Hypothalamic-Pituitary-Adrenal Axis)

Jane Issick

ID#: 146967

Gender: F Age: 63

William Willhelp, MD

123 Anywhere St Anytown, NC 12345 USA **Date Reported** 05/24/2007

SAMPLE REPORT

**Date Collected** 

05/09/2007

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05/15/2007

**Lab Final** 

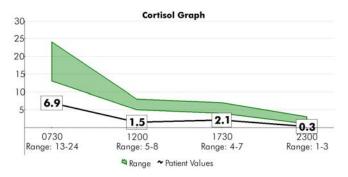
05/23/2007

**Report Final** 

05/24/2007

Marker	Values	Range
INHIBITOI	RY NEUROTRA	NSMITTERS
SEROTONIN	125.0 (L)	150-300 mcg/g Cr
GABA	461.9 (L)	550-750 mcg/g Cr
Marker	Values	Range
EXCITATO	RY NEUROTRA	NSMITTERS
DOPAMINE	405.9 (H)	150-300 mcg/g Cr
NOR-EPINEPHRINE	58.3 (H)	20-45 mcg/g Cr
EPINEPHRINE	6.4	3-20 mcg/g Cr
GLUTAMATE	34.5	15-35 mcg/g Cr
ADRENA	L ADAPTATIO	NINDEX
NOREPI/EPI RATIO	9.1	<10
ADF	RENAL HORMO	NES
CORTISOL (0730)	6.9 (L)	13-24 nM
CORTISOL (1200)	1.5 (L)	5-8 nM
CORTISOL (1730)	<b>2.1</b> (L)	4-7 nM
CORTISOL (2300)	<b>0.3</b> (L)	1-3 nM
DHEA-s (0730)	1.1 (L)	2-10 ng/ml
DHEA-s (1730)	1.1 (L)	2-10 ng/ml
0	THER MARKER	R S
CREATININE, URINE	84.0	

Creatinine is used to calculate results and is not intended to be used diagnostically.



Whenever laboratory data conflict with clinical findings or impressions, clinical judgment should be exercised and additional evaluation undertaken.



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CORRELATION ANALYSIS REPORT

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## The CSM And Your Patient

The objective of the Communication System Management clinical model is to restore function to the HPA axis. This initial test provides an analysis of neurotransmitter and adrenal hormone values. By bringing levels back into established reference ranges, the underlying causes of chronic and acute symptoms are addressed. Monitoring the effects of Targeted Nutritional Therapy through retesting ensures neurotransmitter and adrenal hormone balance, helping the patient to achieve symptom relief and prevent recurrence.

Lab results show deficiencies in all areas of the HPA axis. Adequate serotonin and GABA are needed to maintain mood and calm in the body and to help prevent symptoms of anxiety, depression, and insomnia. Adrenal fatigue may present with low cortisol levels as seen with this patient and can also contribute to fatigue, cravings, and also allergies. Low cortisol and a lack of methyl donors can impede the conversion of norepi to epi, which can exacerbate anxiety. Rebalancing these deficiencies may best begin with inhibitory support to replenish serotonin and GABA. Adrenal support may come next, followed finally by methylation support. Catecholamine support may be recommended after retesting.

ADRENAL HORMONES		
CORTISOL (0730)	<b>6.9</b> (L)	13-24 nM
CORTISOL (1200)	1.5 (L)	5-8 nM
CORTISOL (1730)	<b>2.1</b> (L)	4-7 nM
CORTISOL (2300)	<b>0.3</b> (L)	1-3 nM
DHEA-s (0730)	1.1 (L)	2-10 ng/ml
DHEA-s (1730)	1.1 (L)	2-10 ng/ml

## Adrenal Comments

Adrenal fatigue is indicated in this patient. Consider chronic stressors in the past initiating Dr. Selyes Alarm Phase with an up-regulated HPA axis, followed by the bodyís adaptation and then exhaustion. Low cortisol levels can affect the conversion of nor-epinephrine to epinephrine, resulting in an elevated norepi/epi ratio. Anxiety, burnout and poor blood sugar control are often reflected in an elevated ratio. There are several reasons for low DHEA levels. One of the most common causes of low DHEA is chronic stress. Pregnenolone is the precursor hormone for both DHEA and Cortisol. As stressors become chronic, stores of pregnenolone become less abundant, no longer funding both pathways. Stress requires cortisol, at the expense of the DHEA pathway, and possibly the sex hormone pathway. Symptoms of depression/low mood, lethargy, low libido and poor memory may be present if low levels persist. Low DHEA may also be associated with hypothyroidism and correlates with a higher risk of NIDDM. Adequate DHEA is also neuroprotective, especially of the hippocampus, the epicenter of memory in the brain. Supplementing DHEA in this patient might be of clinical benefit.

The presence of ALLERGIES is often a result of poor adrenal function, where cortisol, the body's anti-inflammatory hormone, is low. Of our patient population marking moderate to severe allergies, 88% have low morning cortisol. Low cortisol can allow inflammatory conditions, such as allergies, to increase. Though cortisol is usually low, it is common to observe a rise in cortisol late in the evening, causing poor sleep, which often accompanies allergies. Excitatory neurotransmitters (e.g., norepinephrine) may also



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be elevated, contributing to the poor sleep pattern so often seen in allergic individuals. Adrenal hormone and inhibitory neurotransmitter support may be beneficial for this patient.

## Neurotransmitter Comments

Marker	Values	Range
INHIBITORY NEUROTRANSMITTERS		
SEROTONIN	125.0 (L)	150-300 mcg/g Cr
GABA	461.9 (L)	550-750 mcg/g Cr

## Inhibitory Neurotransmitters

The patient has indicated problems with SLEEP. The low or low normal serotonin is likely causative because serotonin is the biochemical precursor to melatonin, the sleep hormone. To improve the function of serotonin, GABA levels must be adequate since serotonin generally functions by stimulating GABA at the receptor level. Many of the new generation of sleep medications are GABA receptor agonists in keeping with the role that GABA plays in relation to serotonin and melatonin. In cases of SAD seasonal disorder, serotonin is being utilized at a much higher rate to produce melatonin due to the increased darkness and serotonin stores deplete more quickly.

Symptoms of ANXIETY, NERVOUSNESS, and/or IRRITABILITY may stem from low levels of inhibitory neurotransmitters (serotonin and GABA). Because much of serotonin's inhibitory function is dependent on its binding to GABA receptors in order to carry out inhibition, depletion of either serotonin or GABA may allow excitatory neurotransmitters (glutamate, NE, EPI) to rise. This rise, in turn, may trigger anxiety symptoms. Supporting the inhibitory pathway before addressing excitatory neurotransmitters is recommended.

Patient indicated DEPRESSION and/or DARK THOUGHTS and/or MOODINESS. There are multiple pathways in the central nervous system where imbalance can produce depressive symptoms, the most well-known of which is the serotonin pathway. Low serotonin levels are often associated with depression-74% of depression cases tested measure low to low normal serotonin-particularly depression with concurrent anxiety, dread, and insomnia and often an upregulated HPA axis. For this type of depression, serotonin and often GABA support is necessary. If the patient has high urinary serotonin levels, indicating high loss, serotonin function may still be low. High loss may be due to receptor blockage (medication or heavy metal toxicity), 5-HTP supplementation or high neurotransmitter turnover. Additionally, depression is associated in the literature with elevations in cortisol, particularly evening elevations. Depression is also associated with low dopamine (70% of those with moderate to severe depression tested low), low blood RBC, low serum ferritin levels, and low levels of the essential fatty acid EPA. Dopamine repletion (if necessary) and EPA supplementation (e.g., fish oil) is warranted.

In cases of low DHEA, supplemental DHEA administration is warranted, as supplemental DHEA has been associated with improvement in symptoms of depression.

Thyroid status should always be assessed in treating depression. Low thyroid can reduce serotonin function. We know that in animals with hypothyroidism, serotonin synthesis is decreased and that the administration of T3 increases the brain levels of serotonin. Specifically, thyroid hormones increase the sensitivity of 5-HT2 receptors and also decrease the sensitivity of serotonin 1A autoreceptors that regulate neuronal activity.

The patient's symptoms of SUGAR CRAVINGS, INSOMNIA, FATIGUE and/or ABDOMINAL WEIGHT GAIN may correlate with the



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presence of insulin resistance/metabolic syndrome. Etiologically, insensitive insulin receptors allow rises and falls of blood sugar. Such unstable blood sugar cannot adequately fuel cellular metabolism. If adrenal fatigue is present, assessment may show depleted cortisol and DHEA reserves, reflecting Selye's Adrenal Exhaustion Phase. However, of the patient population indicating moderate to severe abdominal weight gain, over 47% demonstrated evening cortisol levels above 2. These higher levels at night contribute to difficulty falling and/or staying asleep. Low levels during the day mean that cortisol is no longer available to compensate for falling blood sugar levels. Fatigue and other signs and symptoms of reactive hypoglycemia become more severe. Consider assessing patient's glucose and insulin levels (3-hour fasting glucose/insulin test).

Serotonin is necessary for the homeostatic balance of insulin and blood sugar. It is also known that Serotonin often follows blood sugar patterns. As blood sugar fluctuates in insulin resistance from hyperglycemia to hypoglycemia, the brain responds to the slope of the curve-as glucose falls, serotonin also falls and eventually depletes while the catecholamines rise in an effort to force blood sugar back up. Elevated catecholamines produce the irritability, shakiness, and anxiety of low blood sugar.

Marker	Values	Range	
EXCITATORY NEUROTRANSMITTERS			
DOPAMINE	405.9 (H)	150-300 mcg/g Cr	
NOR-EPINEPHRINE	58.3 (H)	20-45 mcg/g Cr	
EPINEPHRINE	6.4	3-20 mcg/g Cr	
GLUTAMATE	34.5	15-35 mcg/g Cr	

#### **Excitatory Neurotransmitters**

Patient checked FATIGUE on questionnaire. Chronic fatigue can be caused by numerous conditions, the most common of which are 1) inadequate sleep, 2) low or high blood sugar, 3) hypothyroidism, and 4) adrenal fatigue, usually demonstrated by inadequate cortisol, particularly low morning levels (87% of patients indicating fatigue of moderate or severe intensity measure low a.m. cortisol). Low stores of excitatory neurotransmitters, such as dopamine, norepinephrine, epinephrine, and glutamate, can also influence energy levels. Other reasons for fatigue involve inadequate dietary protein or B vitamins, dysregulation of mitochondrial function, anemia, depression, acute or chronic illnesses, and certain medications. Assessment of thyroid, iron status, blood sugar, diet and adrenal function are all warranted.

Patient indicated POOR FOCUS to be an issue. Of the patient population who indicated moderate to severe focus problems, 71% demonstrate low or low-normal dopamine. This patient has an elevated dopamine which indicates that there may be inadequate dopamine function. Factors contributing to poor function may include stress/elevated cortisol levels, poor receptor function, or low inhibitory neurotransmitter levels allowing elevations in one or more of the other catecholamines, causing over-excitation. Use inhibitory support to prevent over-excitation, as well as moderate catecholamine support to maintain adequate levels of all of the catecholamines.

Patient's symptoms of LOW LIBIDO may stem from both neurotransmitter and hormonal imbalances. 67% of those who marked moderate or severe in this category measured low or low-normal dopamine. Libido is associated with dopamine function, although many neurotransmitters may influence it, including serotonin. Increased support of catecholamine function may be beneficial. Imbalances in sex, adrenal, or thyroid hormones may be a factor as well. Testosterone is well-known to support libido and assessment may be warranted. Another contributing factor may be low levels of DHEA. DHEA is the precursor to both estrogen and



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testosterone, and is important for proper libido function. Out of the patient population for whom low libido was moderate to severe, 62% had low DHEA. Medications (such as SSRI's), illness, and psychosocial or emotional conditions should be ruled out as causative as well. Supporting both the excitatory pathway and DHEA may be of clinical benefit.



THERAPEUTIC RECOMMENDATIONS

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# Patient is in: Initial Phase

The following therapeutic protocol is based on conclusions derived from patient lab results, clinical data, gender, age, etc, and symptoms listed on the patient questionnaire. The goal of this protocol is to help the doctor begin the three phase process of restoring balance in the HPA axis, while also improving symptoms for patient optimum well-being. The Initial Phase is the beginning of the patients rebuilding process, where TNT is introduced to help move lab values in the right direction. Leaving the patient on the Initial protocol longer than necessary may unbalance the patient. Retesting initiates the Restoration Phase. It provides significant two-fold value in that it serves as a guide in adjusting or fine-tuning the therapy. In addition it allows for monitoring of progress as the patient rebalances their signaling biochemicals on the path toward optimum well-being.

# Overall Summary and Recommendations

Prolent	x 1 in PM, after 5 days increase to $x$ 1 before lunch and dinner to help with sugar cravings Contains: 5HTP, Suntheanine, glycine and B6
Lentra	x 1 BID for GABA support and anxiety help
	Contains: GABA-A agonists Suntheanine, Lactium and magnesium taurate
Adaptacin	After 14 days add x 1 in AM to support adrenal function; after 7 additional
	days increase to $x$ 1 before lunch and $x$ 1 before dinner
	Contains: Bovine adrenal cortex, adaptogens, and vitamin cofactors
MethylMax	When adding Adaptacin, add x 1 in AM
•	Contains: SAMe, TMG, and Folic acid

Retesting is an important part of this process. NT levels need to be monitored. Retesting for this patient is recommended in 9 weeks.

# Additional Recommendations

\* It is recommended that all patients on a program to balance HPA axis function should also supplement with B complex, a multi-mineral and multi-vitamin as well as EPA/DHA.

### **Disclaimers**

- \* These statements have not been evaluated by the Food and Drug Administration. These products are not intended diagnose, treat, cure, or prevent any disease.
- \*The statements above are recommendations to the clinician. All final therapeutic decisions are the responsibility of the treating physician.
- \* Please call Sanesco International at 866-670-5705 with your technical and clinical questions. For further reading and references, please refer to Sanesco's Technical guide and Clinical guide.



Patient Symptom Questionnaire Revised:5/2009

#### PLEASE RETURN THIS QUESTIONNAIRE IN THE BOX WITH YOUR SAMPLE TO NEUROLAB Patient Information: PLEASE PRINT CLEARLY М. Middle Initial: First Name: Last Name Suffix: Female Weight 148 Height 51 Male First Test Doctor's Name: Second Test Third Test or More Lifestyle factors: Please check all that apply to you Medical Diagnoses: Please check all that apply to you Caffeine: # of sups pottles per day ADD/ADHD Blood Pressure: High Low Alcohol: # of drinks 2 days before test Autism Psychosis Consistent difficulty in waking in the AM. ☐ Bipolar Elevated Homocysteine Eating Disorder ☐ Smoke Exercise Regularly Specify: PKU Vegetarian or Vegan Stressful Lifestyle Medications: Please check all that apply to you. LIST DOSE, NAME AND FREQUENCY ON THE BACK OF THIS FORM. ADD/ADHD meds Anti-depressants Blood Pressure meds Hormones Adrenal Glandular Anti-Inflammatory meds MAO Inhibitors Seizure meds Cardiac meds ☐ Allergy meds Anti-Psychotic meds Pain meds Thyroid meds Cancer treatment Anti-Anxiety meds Birth Control Pills Diabetes meds Parkinson's meds If you have used anti-depressants, please list which one(s) worked best for you Supplements and herbs: Please check all that apply to you. LIST DOSE, NAME AND FREQUENCY ON THE BACK OF THIS FORM. ☐ 5HTP Glutamine ☐ Plenus SomniTR Tryptophan Contegra Lentra Procite-D St John's Wort Tyrosine or Phenylalanine ☐ DHEA Melatonin Prolent Theanine ☐ GABA SAMe or MethylMax Tranquilent Phosphatidylserine Please grade each of the symptoms below based on the severity they have caused over the last month. 1 (occasionally); 2 (mild); 3 (moderate); 4 (severe) Allergies Decreased Stamina Nervousness Fatigue (chronic) Andropause symptoms Obsessive/compulsive behavior Anxiety Fibromyalgia Pain (general) Headaches (migraine) **PMS** Apathy Appetite, excessive or uncontrolled Hyperactivity Poor memory Arthritis IBS: Constipation dominant Poor sleep (can't stay asleep) Cold extremities IBS: diarrhea dominant Salt cravings Cycle changes: Insomnia (can't get to sleep) Shakiness when a meal is skipped Shorter Irritability Sugar cravings Dark Thoughts Joint pain Tremor of hands Depression (with exhaustion) Lack of focus Weight gain (abdominal) Depression (with nervousness) Weight gain (general) Menopausal symptoms Decreased libido Moodiness Weight loss

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