NeuroLab
A Division of Sanesco International

The foremost reference laboratory for the measurement of biomarkers associated with HPA-T axis function.

HPA Profile (2) (Hypothalamic-Pituitary-Adrenal Axis)

Franklin Cook
ID#: 366016
Gender: M Age: 71

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Date Reported
02/05/2016

Date Collected
01/18/2016

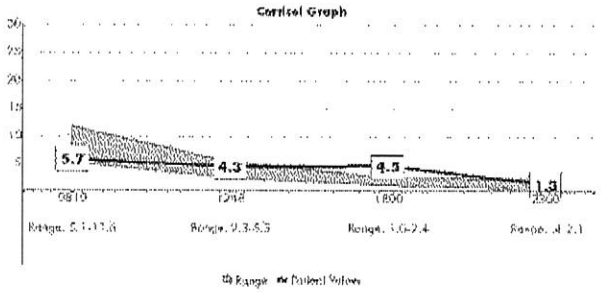
Date Received
01/25/2016

Lab Final
02/01/2016

Report Final
02/05/2016

Marker	Values	Previous Values	Optimal Range	Reference Range
INHIBITORY NEUROTRANSMITTERS				
SEROTONIN	**361.6**	**725.8** (H)	200-415 mcg/g Cr	50-250 mcg/g Cr
GABA	**117.1** (L)	**162.6** (L)	600-1100 mcg/g Cr	150-700 mcg/g Cr
EXCITATORY NEUROTRANSMITTERS				
DOPAMINE	**111.8** (L)	**115.0** (L)	250-400 mcg/g Cr	100-350 mcg/g Cr
NOR-EPINEPHRINE	**22.2** (L)	**109.1** (H)	30-50 mcg/g Cr	13-70 mcg/g Cr
EPINEPHRINE	**9.4** (L)	**9.1** (L)	10-15 mcg/g Cr	3-20 mcg/g Cr
GLUTAMATE	**2.7** (L)	**2.4** (L)	5-10 mg/g Cr	2-12 mg/g Cr
ADRENAL ADAPTATION INDEX				
NOREPI/EPI RATIO	**2.4**	**12.0**	N/A	<13
ADRENAL HORMONES				
CORTISOL (0810)	**5.7**	**9.6**	N/A	5.1-11.6 nM
CORTISOL (1248)	**4.3**	**7.7** (H)	N/A	2.3-5.3 nM
CORTISOL (1800)	**4.5** (H)	**2.7** (H)	N/A	1.0-2.4 nM
CORTISOL (2300)	**1.3**	**2.7** (H)	N/A	.4-2.1 nM
DHEA-s (0810)	**7.9** (H)	**1.7**	N/A	1.0-3.0 ng/ml
DHEA-s (1800)	**>1200** (H)	**1.8**	N/A	1.0-3.0 ng/ml
OTHER MARKERS				
CREATININE, URINE	**179.4**	**46.1**	N/A	mg/dL

Previous Test: HPA Profile (November 2015)
Creatinine is used to calculate results and is not intended to be used diagnostically.
(L) and (H) are based on optimal ranges



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

Results Processed By CLIA #: 34D1054007, NeuroLab Asheville, NC

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REBALANCING

CORRELATION ANALYSIS REPORT

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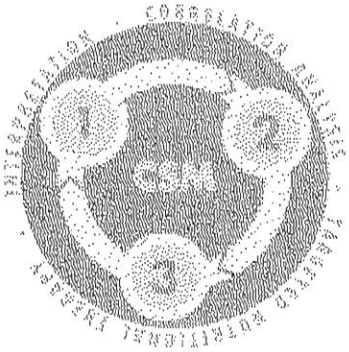

The CSM And Your Patient

The object of the Communication System Management clinical model is to restore function to the HPA axis. One of the cornerstones of the CSM model is to monitor neurotransmitter and hormone levels by retesting the patient throughout the rebalancing process, which is the most effective way of guiding individual therapy. The patient's current response can be measured against previous results and symptoms, allowing for imbalances to be more adequately addressed. Targeted Nutritional Therapy will be adjusted as results are viewed and compared. With each retest, the patient is moving closer to achieving HPA axis and symptom balance.

Note: The lab results have been verified by the laboratory and the patient is taking a supplement that may contribute to the high DHEA values reported. However, if the practitioner and patient determine that a resubmit is necessary, a complimentary kit will be provided if resubmitted within 30 days after practitioner receives this report. To order a resubmit kit, please contact customer service at 866-670-5705. If you have questions, please contact the clinical department at the same number.

Since the last test, the patient has seen some symptoms diminish completely (andropause symptoms, excessive appetite, decreased stamina, night sweats, joint pain) and the continuation of previous symptoms (anxiety, fatigue, depression with exhaustion, headaches, poor sleep, irritability, general pain, poor memory, hand tremors, sugar cravings). The patient's anxiety, headaches, poor sleep, hand tremors, and irritability that may be related to laboratory results showing a decrease in GABA and serotonin. These inhibitory neurotransmitters function together to promote calm, relaxation, and a sense of well-being. Serotonin may be especially important for reducing headaches as it influences pain perception. Evidence suggests that inadequate GABA may also be a factor in the occurrence and recurrence of headaches. Therefore, consider implementing supplemental support for serotonin and GABA to help restore optimal inhibitory function and to potentially assist in alleviating the patient's associated concerns. The patient's depression with exhaustion, fatigue, general pain, and poor memory may be related to current lab values of low excitatory neurotransmitters. Dopamine can have an influence on pain modulation and feelings of ambition, motivation, and energy/drive as it functions to create a sense of pleasure and reward in the brain. Norepinephrine and epinephrine can play roles in energy level maintenance, while glutamate can have an influence on learning and memory. Therefore, consider adjusting/changing supplemental support for the excitatory neurotransmitters to help restore levels and to potentially assist in alleviating the patient's associated concerns. The lab value of high cortisol may be further contributing to the patient's anxiety and irritability. As high cortisol levels can eventually result in adrenal fatigue and are known to suppress serotonin receptor function, it is suggested to identify and appropriately address stressors, diet, and caffeine consumption that may cause surges. Retesting is recommended in 12 weeks to assess the restoration process and to make any necessary adjustments to therapeutic protocol.

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*NOTE: This service is reserved for practitioner use only.
This report was written for you by Nathan Bridges. We strive to create the highest quality reports, and encourage our practitioners to contact our Clinical Support Specialists with any questions or concerns. We can also arrange for an Interpretation of the patient's results, based on your schedule and availability. To reach the report writer directly, dial 866-670-5705 ext. 316.

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ADRENAL HORMONES			
CORTISOL (0810)	**5.7**	**9.6**	5.1-11.6 nM
CORTISOL (1248)	**4.3**	**7.7** (H)	2.3-5.3 nM
CORTISOL (1800)	**4.5** (H)	**2.7** (H)	1.0-2.4 nM
CORTISOL (2300)	**1.3**	**2.7** (H)	.4-2.1 nM
DHEA-s (0810)	**7.9** (H)	**1.7**	1.0-3.0 ng/ml
DHEA-s (1800)	**>1200** (H)	**1.8**	1.0-3.0 ng/ml

Adrenal Comments

This patient has a normal morning cortisol level, with one or more of the other three readings above reference range. Adrenal function is sufficient for the normal morning cortisol rise. Cortisol surges throughout the day may result from stress, high glycemic meals which may result in reactive hypoglycemia causing elevated cortisol to support the low blood sugar. Excess caffeine or nicotine, adrenal glandulars, supplemental or prescriptive cortisone products, pain and/or viral infections can also cause elevated levels of cortisol. Nearly half of insulin resistant patients demonstrate elevated evening cortisol. An elevated evening cortisol level can sometimes contribute to poor sleep and increased sleep latency, i.e. difficulty falling asleep.

High DHEA is most likely the result of a stress response. To confirm this elevation, consider reassessing DHEA level in 2 weeks particularly if DHEA is greater than 7.5. Additionally, if DHEA levels remain elevated, consider the following possibilities in this patient: hyperthyroidism, adrenal hyperplasia, PCOS, or even an adrenal tumor. Nicotine has been noted to raise levels of DHEA. Note: Elevations in DHEA are also associated with suppression of GABA function and potentiation of glutamate.

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Neurotransmitter Comments

Marker	Values	Previous Values	Optimal Range	Reference Range
INHIBITORY NEUROTRANSMITTERS				
SERONTONIN	**361.6**	**725.8** (H)	200-415 mcg/g Cr	50-250 mcg/g Cr
GABA	**117.1** (L)	**182.6** (L)	600-1100 mcg/g Cr	150-700 mcg/g Cr

Inhibitory Neurotransmitters

Patient indicated symptoms of ANXIETY and IRRITABILITY, which are often the result of decreased inhibitory neurotransmission and/or excess excitatory neurotransmission. However, this patient has a normal serotonin level. As mood concerns were noted with normal urinary levels of serotonin, consider that overall inhibitory function may be suboptimal, possibly due to suboptimal glycine and/or GABA levels. As the main inhibitory neurotransmitters, GABA, glycine, and serotonin function to promote calm and prevent over excitation. As GABA is the primary inhibitory neurotransmitter, it can be thought of as "the great balancer" of the nervous system. Also, serotonin often functions as a modulator of GABA activity. Low serotonin or depletion of GABA alone may cause anxiety. Research indicates that inositol and glycine supplementation may be beneficial for those suffering from anxiety, especially acute anxiety and panic disorders. Avoid supporting excitatory neurotransmitter function before restoring serotonin and GABA levels. Elevated adrenal hormone levels are known to contribute to the presence of anxiety, irritability, and nervousness concerns. If adrenal hormones levels are elevated, consider identifying and appropriately managing stressors, dietary components and/or medication, which may be contributing to up-regulated adrenal function and the presence of mood concerns. When up-regulated, thyroid hormones may also generate feelings of nervousness, irritability, and anxiety for the patient; therefore, consider a comprehensive thyroid hormone assessment.

Patient noted the presence of HEADACHES/MIGRAINES. While the exact mechanisms behind migraines are not fully known, some studies have shown that serotonin levels may impact the frequency and severity of this type of headache. One of the ways serotonin may act is through its vasoconstrictive properties. Research indicates that during an attack, serotonin levels decrease rapidly. This decrease may allow the blood vessels to dilate, triggering a migraine. Imbalances in the excitatory and inhibitory neurotransmitters serotonin and norepinephrine, as well as imbalanced sex hormones (especially estrogen) may be causative. New research indicates that many of the tension headaches and/or sinus headaches experienced by patients may actually be variants of migraine headaches. Remember that the serotonin pathway is usually regarded as one of the major components of pain mediation. Serotonin determines "pain" behavior and influences the perception of pain. Consider addressing serotonin levels, and balancing excitatory neurotransmitters if inappropriately high. In addition, sex hormone evaluation may be indicated.

The patient has indicated problems with SLEEP. Although serotonin is within normal range, serotonin function may not be optimal to support proper sleep. Serotonin is the biochemical precursor to melatonin, another very important sleep hormone. High excitatory levels may also contribute to sleep concerns, such as elevated norepinephrine, epinephrine, dopamine, glutamate, and/or cortisol. GABA levels must also be adequate since serotonin serves as a modulator for GABA at the receptor level. That is, without adequate GABA, serotonin cannot function optimally. Most of the new generation sleep medications are GABA receptor agonists. In cases of SAD (seasonal affective disorder), serotonin is being utilized at a much higher rate to produce melatonin due to the shorter days and less daylight. Serotonin stores deplete more quickly during the winter months. Serotonin support in this patient, as well as melatonin support, may be warranted. Individuals with thyrotoxicosis often present hypermetabolic features; therefore, consider assessing thyroid hormone levels. Individuals with thyrotoxicosis often present hypermetabolic features; therefore, abnormalities in sleep regulation may also be associated with thyroid dysfunction.

The patient indicated HAND TREMORS. Imbalances between excitatory and inhibitory neurotransmitters can lead to stress, anxiety, heightened emotional state and/or fatigue that can be causes of hand tremors. High cortisol may also be present, contributing to

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hand tremors, as over-excitation often results in shakiness. Dietary causes may include excess caffeine, other stimulants and alcohol or other substance withdrawal. Blood sugar and thyroid disorders should be ruled out through appropriate testing. Studies have shown that a genetic variance in dopamine function or heavy metal toxicity may contribute to this condition. Neurological conditions that affect the brain function, such as stroke, multiple sclerosis, and traumatic brain injury, can contribute to hand tremors. Proper balancing of excitatory and inhibitory neurotransmitters is recommended. A persistent fine tremor is the most common movement abnormality that presents in thyrotoxicosis, a syndrome resulting from the excess of circulating FT4 and/or FT3; therefore, consider assessing thyroid hormone levels, for more insight into the cause of this symptom.

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EXCITATORY NEUROTRANSMITTERS				
DOPAMINE	**111.8** (L)	**115.0** (L)	250-400 mcg/g Cr	100-350 mcg/g Cr
NOR-EPINEPHRINE	**22.2** (L)	**109.1** (H)	30-50 mcg/g Cr	13-70 mcg/g Cr
EPINEPHRINE	**9.4** (L)	**9.1** (L)	10-15 mcg/g Cr	3-20 mcg/g Cr
GLUTAMATE	**2.7** (N)	**2.4** (L)	5-10 mg/g Cr	2-12 mg/g Cr

Excitatory Neurotransmitters
Patient checked FATIGUE on the questionnaire. Chronic fatigue can be caused by numerous conditions, the most common of which are 1) inadequate sleep (consider sleep pathologies), 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 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, heavy metal toxicity as well as acute and chronic environmental toxins, and certainly many medications. Assessment of thyroid, iron status, blood sugar, and diet are all warranted.

The patient's questionnaire indicated GENERAL PAIN to be an issue. Approximately 70% of patients who marked moderate or severe pain issues measure low serotonin. Both serotonin and norepinephrine are known to provide an inhibitory influence on pain, and under stressful conditions, may provide nearly complete pain inhibition. Norepinephrine can also reduce pain by stimulating beta-endorphin release. If the level of serotonin is deficient, pain perception increases. Often norepinephrine is elevated (if adrenal function is adequate), likely responding to the stress of the painful experience. Another possible area of imbalance is dopamine function. Research indicates altered dopamine activity at the receptors, and an abnormal dopamine response in the experience of pain. Replenishing serotonin, supporting dopamine, and balancing norepinephrine can often be helpful in cases of pain. At times, if pain is severe, the body may perceive this pain as stress, resulting in the up-regulation of adrenal hormone levels.

Patient indicated DEPRESSION as a concern. There are multiple pathways in the central nervous system where imbalance can produce depressive symptoms, the most well-known of which are the bioamine (serotonin, norepinephrine, dopamine) pathways. Low serotonin levels are often associated with depression, particularly depression with concurrent anxiety, dread, and insomnia. If patient shows normal or high serotonin, consider that serotonergic or overall inhibitory function is not adequate; inhibitory support may beneficial despite the normal urinary levels. High urinary levels of serotonin may be indicative of high loss, which may be due to receptor blockage (medication or heavy metal toxicity), 5-HTP supplementation or high neurotransmitter turnover. Depression can also be associated with low dopamine and/or norepinephrine, especially those with vegetative depressions that involve lack of adequate drive, ambition, focus, or energy and typically present with lethargy, fatigue, excess sleep and lowered HPA function.

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If the patient has normal or high urinary bioamine levels, indicating high loss, function may still be low. High loss may be due to receptor blockage (medication or heavy metal toxicity), supplementation or high neurotransmitter turnover. Depression can also be associated with low blood RBC, low serum ferritin levels, and low levels of the essential fatty acid EPA. Bioamine repletion (if necessary) and EPA supplementation (e.g., fish oil) may be warranted with the addition of co-factors required for the pathways, such as B6 as P5P.

Optimal thyroid function is paramount to comprehensive treatment of depression. Medical research is replete with references regarding mood and thyroid function; consider adding a comprehensive thyroid assessment.

Additionally, depression is associated in the literature with elevations in cortisol, particularly evening elevations. It is well known that Cortisol Releasing Factor (CRF) is increased in healthy patients with depression, which lead to increased cortisol levels. The medical literature also supports the fact that unmedicated unipolar and bipolar depressed patients have a 'hyperresponsive' noradrenergic system (with elevated NE levels and turnover). This is a common pattern along with low serotonin levels. In addition, much research suggests that both hypothalamic and extrahypothalamic CRF activates the locus ceruleus in the brain, leading to an increase in norepinephrine. Thus, high CRF activity might lead to both elevated cortisol and norepinephrine levels seen in depressed patients. In cases of low DHEA, supplemental DHEA administration is warranted, as supplemental DHEA has been associated with improvement in symptoms of depression.

Patient Indicated POOR MEMORY. Memory is dependent upon balance among many central neurotransmitters. Adequate glutamate is required for learning and memory; 60% of patients marking moderate or severe memory issues have low/low normal glutamate. Adequate dopamine is also necessary; low levels can impair working memory, in particular. 70% have low or low-normal dopamine. Norepinephrine is also required-both short-term memory and long-term memory depend on adequate NE levels. Acetylcholine is a primary neurotransmitter for the laying down of memory traces and, though not measured, can be supported by increasing dietary choline or supplementing with phosphatidylcholine or DMAE. Serotonin is also required for proper memory (acute tryptophan depletion can directly impair memory). There is evidence in the literature, however, that extreme excesses of norepinephrine, glutamate and serotonin can also impair memory. Additionally, chronic elevations of cortisol damage the hippocampus, center for short-term memory. DHEA should be repleted when low, since it is known to be neuroprotective to the hippocampus. Balance, then, among the neurochemicals, is of utmost importance for establishment and maintenance of memory. Decreased thyroid function is known to impede cognitive function; therefore, consider assessing thyroid hormone levels.

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THERAPEUTIC RECOMMENDATIONS

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Patient is in: Restoration Phase

The following therapeutic protocol is based on conclusions derived from previous and current patient lab results, clinical data, such as gender, age, etc, and previous and current symptoms listed on patient questionnaires. The goal of this phase is the restoration of their optimal ranges as well as their physical and emotional balance, which is the reason it is called Restoration Phase. Depending upon how the patient responded to previous therapies, the protocol will reflect the next stage in the rebalancing process. Continuing the path towards balancing the HPA-T axis and alleviating symptoms is the main goal. Retesting will be important, and it may be necessary to retest more than two or three times until optimum balance is achieved. When the doctor and patient agree that the patient has found their ideal level of balance, Maintenance Phase will begin.

Overall Summary and Recommendations

Tranquilent	***x 1 as needed for mood support, may increase to x 2 as needed*** ***Contains: low doses of 5-HTP and Suntheanine, Myo-Inositol***
Lentra	***x 1 daily for GABA support, may increase to twice daily*** ***Contains: GABA-A agonists: Magnesium Taurate, Suntheanine, and Lactium***
Procite-D	***x 1 in the AM every other day for dopamine support. Do not take in conjunction with other excitatory support.*** ***Contains: Mucuna pruriens, N-acetyl-L-tyrosine, DL-phenylalanine, NAC and B vitamins***

Retesting is an important part of this process. NT levels need to be monitored. Retesting for this patient is recommended in 12 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.

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