NeuroLab
A Division of Sanesco International

HPA-G Complete Profile (1)

Kacie Doe
Report ID: #3149
Gender: F Age: 63
06/26/1955

Date Collected
12/29/2018

Date Received
01/01/2019

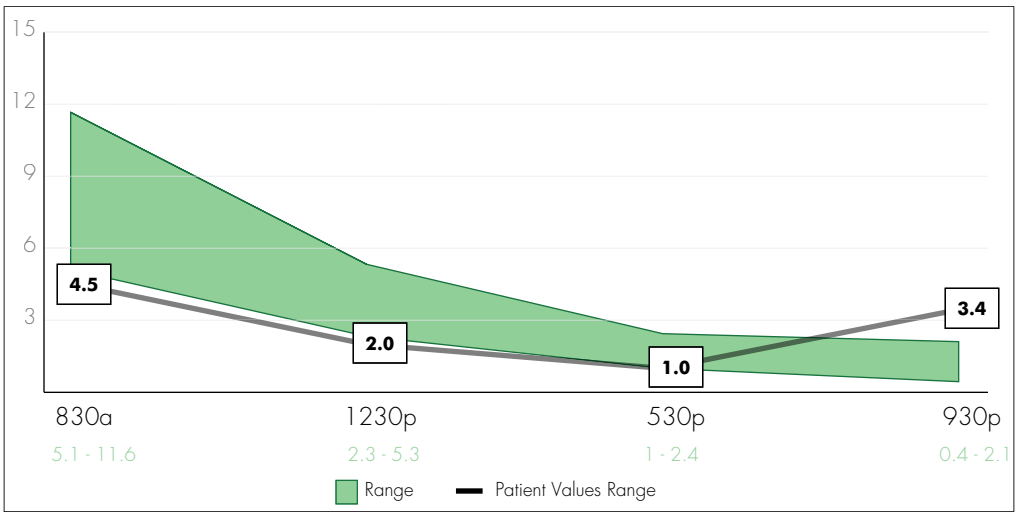
Lab Final
01/07/2019

Sex Hormone Reference Range Type
Post Menopausal + HRT (Progesterone Only)

Sanesco Clinician
1010 Merrimon Ave
Asheville, NC 28803
United States

Marker	Values	Optimal	Reference
INHIBITORY NEUROTRANSMITTERS			
SEROTONIN	**35.2** (L)	125 - 260 mcg/g Cr	50 - 250 mcg/g Cr
GABA	**299.0** (L)	600 - 1100 mcg/g Cr	150 - 700 mcg/g Cr
EXCITATORY NEUROTRANSMITTERS			
DOPAMINE	**202.8** (L)	250 - 400 mcg/g Cr	100 - 350 mcg/g Cr
NOR-EPINEPHRINE	**65.3** (H)	30 - 50 mcg/g Cr	13 - 70 mcg/g Cr
EPINEPHRINE	**12.0**	10 - 15 mcg/g Cr	3 - 20 mcg/g Cr
GLUTAMATE	**14.1** (H)	5 - 10 mg/g Cr	2 - 12 mg/g Cr
PEA	**4.0**	n/a	1.6 - 7.3 mcg/g Cr
ADRENAL ADAPTATION INDEX			
NOREPI/EPI RATIO	**5.4**	n/a	< 13
OTHER MARKERS			
CREATININE, URINE	**100.0**	n/a	mg/dL
ADRENAL HORMONES			
CORTISOL (830a)	**4.5** (L)	n/a	5.1 - 11.6 nM
CORTISOL (1230p)	**2.0** (L)	n/a	2.3 - 5.3 nM
CORTISOL (530p)	**1.0**	n/a	1.0 - 2.4 nM
CORTISOL (930p)	**3.4** (H)	n/a	0.4 - 2.1 nM
DHEA-s (830a)	**2.9**	n/a	1.0 - 6.0 ng/ml
DHEA-s (530p)	**3.0**	n/a	1.0 - 6.0 ng/ml
SEX HORMONES			
ESTRONE (E1)	**12.2**	n/a	10 - 30 pg/mL
ESTRADIOL (E2)	**1.0**	n/a	0.3 - 1.4 pg/mL
ESTRIOL (E3)	**17.0**	n/a	< 32 pg/mL
PROGESTERONE	**102.0**	n/a	34 - 542 pg/mL
TESTOSTERONE	**13.7** (L)	n/a	23 - 69 pg/mL
EQ RATIO			
E3/(E1+E2) Ratio	**1.3**	n/a	< 2.0
Pg/E2 RATIO			
Pg/E2 Ratio	**102.0**	n/a	n/a

Creatinine is used to calculate results and is not to be used diagnostically.
(L) & (H) are based on optimal ranges if available, otherwise they are based on reference range.



HPA-G Complete™ Profile

CORRELATION ANALYSIS REPORT

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The CSM And Your Patient

The Communication System Management™ (CSM) Model provides an analysis of neurotransmitter and hormone values and correlates them with patient complaints represented in a self-scored patient questionnaire. This approach helps practitioners and patients understand how their current problems are related to their laboratory values. In the next section, we introduce the Correlation Analysis Report, where we correlate the patient's lab values with their self-assessment of the health and quality-of-life issues that confront them.

The patient indicted medications and a supplement that may influence lab results.

The patient reported anxiety, irritability, nervousness, poor sleep, and depression that may be related to the levels of deficient serotonin and suboptimal GABA. The inhibitory neurotransmitters serotonin and GABA function together to promote calm, relaxation, and a sense of well-being. Therefore, consider supporting serotonin and GABA with supplementation to help restore optimal inhibitory neurotransmitter function and potentially assist in improving the patient's mood and sleep concerns.

The patient mentioned excessive appetite and low libido that may be related to the suboptimal dopamine level. Dopamine can have an influence on appetite regulation and sex drive as it functions to create a sense of pleasure and reward in the brain and body. The dopamine and testosterone levels seen in this patient are consistent with their relationship to one another; dopamine and testosterone support each other in a positive way. Due to the presence of anxiety and high blood pressure, combined with elevated levels of norepinephrine and glutamate, supplemental catecholamine support is not recommended at this time. Supplemental catecholamine support may be considered upon retesting.

The patient's high glutamate level may be further contributing to feelings of anxiety and depression. Consider assessing this patient's diet for exogenous sources of glutamate (MSG, aspartame, glutamine, and/or processed foods), as chronic high glutamate may be neurotoxic and damaging to neuronal health. Underlying inflammation may also be driving up the glutamate level.

The patient listed decreased stamina and seasonal allergies that may be related to the low cortisol levels. Cortisol functions to maintain energy levels and keep the body engaged during the day. Cortisol may be important for reducing seasonal allergies as it is one of the body's main anti-inflammatory hormones. Therefore, consider supporting the adrenal glands with supplementation to help rebalance cortisol levels and potentially assist in improving the patient's stamina and allergy concerns.

The patient has a slightly elevated level of cortisol at night. This may be contributing to concerns such as abdominal weight gain and poor sleep. Stimulant intake, toxins, exercise, poor sleep, hypoglycemic episodes, and stressors can cause elevations in cortisol. Ensuring adequate sleep, avoiding toxins and allergens, and managing stress may help to maintain adrenal stores.

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The patient's low libido, anxiety, and depression may also be related to the level of low testosterone. Testosterone helps support both dopamine and serotonin levels. It is common for testosterone levels to decrease with age. Bioidentical testosterone replacement therapy may be considered at the practitioner's discretion. Retesting is recommended in nine weeks to assess the rebalancing process and make any necessary adjustments to the suggested therapeutic protocol.

Estrone (E1) level is normal for this range. Estrone is one of the three primary estrogens and the one that is predominant after menopause. It is produced primarily from plasma androstenedione by the enzyme aromatase. E1, as well as DHEA, are precursors to estradiol (our more powerful estrogen). In general, E1 positively influences serotonin, while most of the literature shows an inhibitory influence on GABA production and on the catecholamines (dopamine, norepinephrine and epinephrine). In general, estrogen is considered stimulatory to the HPA axis, increasing CRH and ACTH. Progesterone, however, reduces these stimulatory effects. Contradictions in the literature on estrogen and the HPA axis may be due to which estrogen receptor is being studied. Estrogen receptor alpha may contribute to more stimulation whereas receptor beta may reduce stimulation. Conversely, HPA activation (stress) is suppressive to sex hormone levels in general.

Estradiol (E2) is normal for this range. Estradiol is the primary estrogen during the reproductive years. In addition to its role in developing secondary sex characteristics, estrogen has broad effects throughout the body. In looking at estrogen's influence on neurotransmission, we see global influences. E2 has a primarily inhibitory influence on GABA. On the other hand, E2 sensitizes glutamate's NMDA receptors, increases glutamate release, and affects dendritic connections, leading to increased learning, memory and neuroplasticity.

E2 serves as a vasodilator via reducing arterial norepinephrine (NE), contributing to optimal blood pressure control. However, in certain areas of the brain, E2 has been shown to increase norepinephrine. E2 is known to inhibit MAO and COMT, enzymes that degrade the monoamines (NE, dopamine and serotonin), leading to higher levels of these important neurotransmitters. Estrogen's effect on NE may thus be dependent upon location.

During a woman's reproductive years, E2 tends to promote the HPA stress response—increasing CRF and cortisol. Supplementing with estrogen (ERT) has also been shown to increase the stress response. The effect of age seems to make women's stress response worse, sensitizing both the sympathetic adrenal response and the cortisol response in menopausal women.

Estrogen is known to reduce DHEA levels. DHEA, on the other hand, is a precursor to estrogen and so can increase E2 levels. Both oral and higher-dose vaginal applications of DHEA can increase serum E2. E2 is an excellent support for our serotonin system. It enhances genetic expression of tryptophan hydroxylase, the enzyme that increases serotonin levels. Estrogen increases the cellular resilience of 5-HT neurons (increasing DNA repair) and may prevent serotonin neuron death as women age. It also stimulates serotoninergic activity. Thus, estrogen has been called women's natural mood-booster. Just as E2 supports serotonin, it also supports dopamine in many areas of the brain. It has also been shown to be neuroprotective for dopaminergic neurons.

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Estriol (E3) is normal. Estriol is a pregnancy-specific estrogen, but it also has importance outside of gestation. It has a significant and well-established role in addressing menopausal symptoms. As a vaginal suppository, E3 is especially useful for urogenital disturbances, particularly vaginal dryness, urinary/stress incontinence, recurrent UTIs, hot flashes, night sweats and insomnia. Estriol effects on bone density are equivocal. The best studies showing positive results come from Japan. However, western studies often report no effect. Lindsay et al. reported that it took at least 14 mg of E3 daily to prevent bone loss. E3 also has immunomodulatory benefits and may potentially be useful in reducing autoimmune and inflammatory markers. Stephenson et al. studied the use of a transdermal Biest formula (80% E3, 20% E2) and/or transdermal progesterone in 75 post-menopausal women for 36 months, and reported significant improvement in not only menopausal symptoms but also inflammatory markers (MMP-9, CRP, TNF, IL-6, fibrinogen, antithrombin III, etc.) and metabolic parameters (fasting glucose and triglycerides).

Regarding the safety of estriol use, E3 researcher Dr. Henry Lemon (Univ. of Neb) postulated that estriol was probably the safer estrogen in regards to breast cancer. He noted that low doses and an intermittent dosing schedule of non-conjugated estriol demonstrated the most significant anti-mammary carcinogenic activity of the 22 compounds tested. Regarding the endometrium, a meta-analysis of E3 use intravaginally [once a day] reported sustained levels of estriol and no incidence of endometrial hyperplasia.

Progesterone is normal. Progesterone, manufactured by the ovaries and the adrenal glands, is also made in the brain by neurons and glial cells, where it has neuroprotective effects. It has been shown to increase BDNF, help protect and rebuild the blood-brain barrier, reduce inflammation, and help repair brain injury in cases of stroke and TBI. Progesterone also has pro-myelination ability and has shown promise in cases of multiple sclerosis. Normal levels of progesterone also protect against the proliferative effects of estrogen. Through its main metabolite, allopregnanolone, it has major calming effects on the central nervous system as a GABA-A agonist and inhibitory effects on the HPA axis at the level of both the hypothalamus and hippocampus. It is also known to dampen glutamate excitotoxicity, support dopamine neurotransmission and increase serotonin in particular areas of the brain. Progesterone has also been shown to inhibit NE, thereby reducing sympathetic tone. Progesterone is increased by oxytocin release and has been observed to promote social contact and closeness, as does oxytocin. Progesterone is thus considered a part of the neuroendocrine basis of social bonding.

Testosterone is low. Testosterone generally falls as we age. However, high stress (HPA stimulation) and adrenal and/or ovarian insufficiency can produce a more dramatic decline in levels. ERT (supplemental estradiol) has been shown to reduce testosterone by 42%. In cycling women, low testosterone can interfere with follicle maturation, leading to risk of infertility. Some, but not all PMS, has also been associated with low testosterone. Whether from aging, stress or adrenal/ovarian insufficiency, low testosterone in women, as in men, can produce decreased libido, early senility, memory failure, depression, lack of interest, anxiety/nervousness/irritability, and decreased activity, intellectual agility and concentration. Centrally, testosterone increases nitric oxide synthase which produces nitric oxide, stimulating dopamine release. Loss of dopamine stimulation with low testosterone may contribute to low libido. Because testosterone also supports serotonin, low levels may contribute to lower serotonin and its associated depression, anxiety or irritability.

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Neurotransmitter Comments

Inhibitory Neurotransmitters

Patient indicated symptoms of ANXIETY, NERVOUSNESS, and IRRITABILITY, which are often the result of decreased inhibitory neurotransmission and/or excess excitatory neurotransmission. As the main inhibitory neurotransmitters, GABA, glycine, and serotonin function to promote calm and prevent over-excitation. GABA is the primary inhibitory neurotransmitter in the CNS and can be thought of as "the great balancer" of the nervous system. Serotonin often functions as a modulator of GABA activity. Either low serotonin or depletion of GABA 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 anxiety, irritability, and nervousness concerns. If cortisol (particularly morning levels), NE and/or EPI 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 in excess, thyroid hormones may also generate feelings of nervousness, irritability, and anxiety for the patient. Consider a comprehensive thyroid hormone assessment.

Patient indicated DEPRESSION WITH NERVOUSNESS as a concern. There are multiple pathways in the central nervous system where imbalance can produce depressive symptoms with anxiety, the most well-known of which are the bioamine (serotonin, norepinephrine, dopamine) pathways. Low serotonin levels are often associated with depression accompanied by anxiety, dread and/or insomnia. Low serotonin may present due to inadequacy of its amino precursor tryptophan (usually high sugar/low protein diet) or enhanced hepatic degradation (usually from stress biochemistry). If patient test results show normal or high serotonin, consider that serotonergic or overall inhibitory function is still 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, elevated MAO or high neurotransmitter turnover.

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In addition, elevated catecholamines, particularly norepinephrine (NE), can contribute to depressions with concomitant anxiety. Elevated nocturnal NE has been shown to contribute to insomnia, while epinephrine rises as the counter-regulatory hormone in response to hypoglycemia/high sugar diets. Depression with elevations in excitatory neurotransmission and HPA hyper-reactivity have been described as melancholic depression and need to be addressed differently than those depressions with exhaustion or other "hypo" features. Inhibitory support is indicated. Depression has also been associated with low blood RBC, low serum ferritin levels, and low levels of the essential fatty acid EPA. Serotonin repletion, catecholamine balancing where necessary and EPA supplementation (e.g., high-EPA fish oil) may be warranted with the addition of co-factors required for the pathways, such as the P5P form of B6. Hormone imbalances may also contribute. Optimal thyroid function is paramount to comprehensive treatment of depression. Medical research is replete with references regarding depressive mood and thyroid function. Consider adding a comprehensive thyroid assessment. In cases of low DHEA, supplemental DHEA administration is warranted, as supplemental DHEA has been associated with improvement in symptoms of depression.

Additionally, depression with nervousness is associated in the medical literature with HPA Axis hyperactivity and with elevations in cortisol. It is well known that Corticotropin-Releasing Factor (CRF) is increased in healthy patients with depression, which leads to increased cortisol levels. The literature also describes increased SNS activity with elevated NE in depressed patients, particularly in those with anxiety. For example, both unmedicated unipolar and bipolar depressed patients are shown to have a 'hyperresponsive' noradrenergic system (with elevated NE levels and turnover). This is a common pattern, elevated excitatory (NE) with low inhibitory (serotonin) levels. In addition, much research suggests that both hypothalamic and extrahypothalamic CRF activates the locus coeruleus 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.

Not only impairments in monoaminergic transmission but also increased glutamatergic excitotoxicity and central inflammatory processes are now commonly proposed to explain the etiological basis of depression. Patients with major depression have been found to exhibit increased peripheral blood inflammatory biomarkers, including inflammatory cytokines such as IL-6 and IL-10.

Changes involving altered glutamate signaling have been found in depression in both animal models and clinical trials. Glutamate elevations are known to be caused, at least in part, by inflammatory processes. Inflammatory cytokines have been shown to decrease the expression of glutamate transporters, allowing more available glutamate, and to increase the release of glutamate from astrocytes. Chronic inflammation also alters key metabolic pathways, one of the most important and well-studied of which is the tryptophan-to-kynurenine pathway. Induction of this pathway by inflammation or stress results in reduced formation of serotonin (less tryptophan precursor), and the production of neuroactive metabolites, most importantly, excessive formation of quinolinic acid, an NMDA [glutamate receptor] agonist. Hence, the more stress or inflammation that is present, the less serotonin and the more glutamate, both contributors to depression.

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The patient has indicated problems with SLEEP. The low or low normal serotonin is likely contributory because adequate levels of serotonin reduce anxiety, necessary for restful sleep. In addition, serotonin is the biochemical precursor to melatonin, and increasing serotonin via tryptophan has been shown to consolidate fragmented sleep. High excitatory biochemistry may also contribute to sleep concerns, including elevations of norepinephrine/epinephrine, dopamine, and glutamate. GABA levels must also be adequate since serotonin serves as a modulator at GABA receptors. Deficient GABA itself is associated with hyperarousal and poor sleep, and many of the new-generation sleep medications are GABA receptor agonists. Glycine taken at bedtime has also been shown to improve sleep quality. Serotonin support in this patient, as well as melatonin, GABA and/or glycine support may be warranted. Also, individuals with thyrotoxicosis often present with hypermetabolic features. Consider assessing thyroid hormone levels. Both high and low cortisol levels have been associated with disturbed sleep. Low cortisol can cause nocturnal hypoglycemia, leading to disturbed sleep and nighttime awakenings. Elevated evening or early morning cortisol also commonly causes nighttime awakenings. Addressing any cortisol imbalances may be useful.

The patient indicated HOT FLASHES/NIGHT SWEATS as a concern. For most women, this one symptom signals the menopause transition. Hot flashes are “triggered by small elevations in core body temperature acting within a greatly reduced thermoneutral zone” (area between our sweating and shivering thresholds). High norepinephrine (2AR) is partly responsible for the reduction in this zone, which may be secondary to an estrogen deficiency-related reduction in serotonergic activity and an increase in central NE activity. It is generally thought that it is the drop in peri-menopausal estrogen, causing a transient dysregulation of the entire thermoregulatory system, which is primarily responsible for hot flashes. As estrogen levels begin to decrease, the pituitary releases excess FSH (follicle stimulating hormone) to encourage the ovaries to increase production. FSH increase is shown to be positively associated with hot flashes as well. For about one-third of women, hot flashes/night sweats may continue for up to a decade or more after menopause. Night sweats themselves are associated not only with hot flashes but also panic attacks, obesity, insomnia, and use of certain antidepressants and antihistamines.

While menopause is a natural transition and hot flashes/night sweats are a common occurrence, some factors can increase the intensity and rate of occurrence for patients. Caffeine, hot spices, sugar, alcohol, and stress may trigger hot flashes/night sweats in some women. Many patients experiencing hot flashes/night sweats often report other symptoms related to hormonal imbalance including insomnia, anxiety, fatigue, and migraines. With shifting hormone levels, neurotransmitter levels can also fluctuate in an effort to maintain balance within the neuroendocrine system. The most obvious changes occur in GABA and serotonin due to their synergy with progesterone and estrogen. Serotonin agonists or reuptake inhibitors are commonly used and show modest therapeutic effects on hot flashes. As thyroid function controls metabolism and body temperature, hyperthyroidism may also result in intolerance to heat, which may be interpreted as hot flashes. Consider assessing thyroid hormone levels. Because the neuroendocrine system is an interdependent web, effects may be seen in adrenal function and excitatory neurotransmitters, particularly higher norepinephrine, as well. Working with a skilled practitioner to achieve neuroendocrine balance, and to address stressors and nutritional deficits can bring improvement and possibly alleviation of symptoms for a smoother transition.

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Excitatory Symptoms

Patient checked HIGH BLOOD PRESSURE on questionnaire. Studies show a link between high norepinephrine and elevated blood pressure. Norepinephrine (NE) has vasoconstrictive properties, and when elevated, will increase blood pressure. Elevations in NE may be the result of increased firing from the sympathetic nervous system's response to stress or reduced NE reuptake, or both. High stress levels will trigger Corticotropin Releasing Factor (CRF) release, which in turn will increase cortisol and norepinephrine levels in the body. Metabolic Syndrome's insulin resistance & dyslipidemia are also found more frequently among patients with hypertension. Supporting the inhibitory pathways (serotonin and GABA) may be helpful in regulating catecholamine levels, and can help maintain calm. Supplemental methylation and/or cortisol support can help to lower norepinephrine levels by conversion to epinephrine, a methylation and cortisol-dependent step. Proper nutrition (focused on reducing insulin resistance), exercise, sleep architecture, and stress reducing techniques may also be beneficial. For example, Tai Chi has been found to lower blood pressure. If this patient is using blood pressure medications and has stated that blood pressure is under control, a low or normal norepinephrine level may be present.

Patient indicated excess APPETITE. The appetite control center is located in the hypothalamus. Imbalances in the Appetite Regulating Network (ARN) (made up of complex signals from hormones, neurotransmitters, and neuropeptides) can lead to overweight and obesity. A multi-factorial approach is needed for achieving balance. White adipose tissue (WAT), neurotransmitters such as serotonin and dopamine, and ARN hormones such as cortisol, CCK, ghrelin, and leptin are all involved in hunger signals or satiation. Weight loss has been shown to cause adaptations in WAT that contribute to increased appetite (and weight gain). Ghrelin also powerfully stimulates appetite in various brain centers. ARN satiety signals include leptin, CCK, and insulin. Insulin is shown to be inhibitory to ghrelin and stimulatory to leptin, helping to reduce food intake. Leptin, perhaps the main satiety hormone, messages the hypothalamus regarding levels of stored fuel, contributing to appetite control. Leptin also stimulates TRH and so facilitates normal thyroid function. However, in obesity, where leptin is elevated, leptin resistance develops and renders its normal effects largely absent. To address leptin resistance, studies have shown efficacy for calorie restriction, panax ginseng and acetyl-L-carnitine. Low serotonin levels can increase cravings, especially for carbohydrates. Ensuring sufficient protein, particularly tryptophan, in the diet can help offset such cravings and increase satiation. Serotonin, at optimal levels, is associated with satiation and choosing smaller meals. Dopamine reinforces feeding, lending salience to palatable foods. Low levels of dopamine signaling have been consistently found in the obese, and it is theorized that this contributes to increased appetite/food intake to overcome the effects of dopamine signal loss. Chronic stress can lead to elevated cortisol, which may decrease serotonin and suppress thyroid hormones. Elevated cortisol levels may also interfere with proper sleep. This may prompt the gastric hormone ghrelin levels to rise. Decreased cortisol (Dr. Selye's exhaustion phase) can lead to fatigue, which can increase food intake in an effort to restore energy. Excessive appetite is often present in hyperthyroid patients, which may be explained by the positive association between increased T3 and increases in ghrelin secretion and activity. Because dysfunction of the thyroid gland can result in changes in appetite, consider assessing thyroid hormone levels.

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Patient indicated LOW LIBIDO on symptom questionnaire. Though many neurotransmitters are involved in libido, the most significant are dopamine and serotonin. Optimal dopamine is one of the most important requirements. Low dopamine undermines libido. Of those who marked moderate or severe in this category, 67% measure low or low-normal dopamine. Low libido is also commonly observed in patients on SSRI medications. This may be due to the inverse relationship between serotonin and dopamine. Higher levels of serotonin (SSRI studies) have been shown to decrease dopamine signaling. Testosterone is also a well-known support of libido, particularly in patients in midlife. Another contributing factor to low libido is adrenal insufficiency with low DHEA. DHEA is the precursor to adrenal sex steroids and when depleted, causes deficiencies of androgens and estrogens, which may lead to low libido. Of this patient population, 62% measures low DHEA. Other causes such as medications, illness and psychosocial or emotional conditions should be ruled out as well.

Adrenal Comments

Adrenal function is decreased in this patient and this patient may have moved through Dr. Selye's Alarm Phase of the General Adaptation Syndrome as well as the Adaptation Phase and has now moved closer toward the Exhaustion Phase. The one surge may be due to high glycemic dietary influences that can result in reactive hypoglycemia, causing a surge in cortisol in order to support low blood sugar. Caffeine, use of adrenal glandular or supplemental or prescriptive cortisone products, nicotine, or acute stressors may be other reasons for the surge(s) of cortisol. The patient's clinical picture may commonly involve daily fatigue, allergies, and/or other inflammatory conditions.

Normal DHEA suggests this patient is in the "adaptive phase" of Selye's "General Adaptation Syndrome", however, if the patient is under chronic stressors, overtime, hormone production will be shunted to cortisol at the expense of DHEA and low DHEA levels will result (maladaptive phase). During a sustained stress response, which requires continual cortisol secretion, the organism will begin to adapt - that is, begin to feel that the elevated levels of cortisol and catecholamines are "normal." As time goes on, and if the stressors continue, the adrenals will start to lose their ability to compensate (maladaptive phase) and testing will usually show increased cortisol and decreased DHEA.

Patient has noted a diagnosis of one of the AUTO-IMMUNE DISEASES (AI). AI illness is a most complex condition. Etiologies or contributing factors may include: genetic, HPA/immune, hormonal and environmental factors. Environmental influences, in conjunction with genetic predisposition, include infections (particularly EBV), vaccinations (though considered rare), toxicities (heavy metals) and smoking. Hormonal factors involve, to a large extent, estrogen and prolactin. Estrogens and prolactin seem to be implicated as enhancers of the immune response, though this is not clear-cut, but it seems particularly true for SLE. Estrogen (ER□) in general is shown to inhibit Th1 expression, resulting in anti-inflammatory effects in MS, RA, psoriasis and Hashimoto's thyroiditis. However, in SLE, a Th2-dominant disease, estrogen (ER□) is shown to promote progression.

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In researching the HPA axis, we find a voluminous literature on stress, HPA axis dysfunction and auto-immunity. Since the immune system is not autonomous, it behooves us to study influences from the HPA axis and the sympathetic nervous system (SNS), the two major communication channels through which the CNS influences the immune system. Normally, an upregulated immune response (e.g., from antigenic load) stimulates the HPA axis to produce cortisol for the purpose of lowering inflammation. If this system is dysregulated, and cortisol is low or its function is blunted, it opens the door for inflammatory autoimmune conditions. In many AI diseases, cortisol and DHEA are indeed shown to be low. Shoenfeld, et al, summarizes: "The reduced cortisol and adrenal androgen secretion observed during testing in rheumatoid arthritis patients . . . should be considered as a 'relative adrenal insufficiency' in the presence of a sustained inflammatory process." Low DHEA-s is consistently found in AI conditions and should possibly be supplemented, since it is an inhibitor of both IL-6 and NF□B. Additionally, it has been shown that norepinephrine and epinephrine inhibit Th1 cytokines (involved in most AI conditions) and stimulate Th2 cytokines, thus helping to reduce autoimmune symptomology (with the exception of SLE). Low SNS activity may thus facilitate more AI flareups. Consider thorough functional medicine work up to include standard lab testing for auto-immune disease as well as testing for HPA axis function, neurotransmitter levels, intestinal permeability, vitamin D status and comprehensive stool analysis to assess GI function.

ALLERGIES can be contributed to by 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. Cortisol is an important regulator of allergic disease expression. The literature shows a definite association between inflammatory conditions such as allergies and an attenuated HPA axis responsiveness. It is thought that low cortisol may allow inflammatory conditions, such as allergies, to surface or even worsen. However, exposure to allergens can be a stressor, and may actually elevate cortisol. Thus, normal cortisol levels, in the presence of allergies, may indicate suboptimal adrenal function. DHEA is often found to be low in allergy patients, which may also reflect poor adrenal function. Norepinephrine (NE) may be elevated, contributing to the poor sleep pattern so often seen in allergic individuals. NE has been shown to increase IgE, the antibody active in allergy. It does so via increasing IL-4, contributing to the Th2 dominance associated with allergy. Both adrenal hormone and inhibitory neurotransmitter support may be beneficial for this patient. Allergies could be thought of as a "total load" that a patient is carrying. Thus, in addition to HPA balance, environmental support such as adding a HEPA filter to the bedroom and using allergen-proof covers on mattress and pillow cases can be added to the list of therapies. It may also be of value to eliminate as much clutter (dust) as possible through limiting pillows, stuffed animals, carpets and curtains, etc. Also, anti-inflammatory nutraceuticals such as quercetin, bromelain, and nettle extract may be of value.

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Patient checked FATIGUE/DECREASED STAMINA 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) chronic stress/adrenal fatigue, usually demonstrated by inadequate cortisol, particularly low morning levels. Of our patient population, 87% indicating fatigue of moderate to severe intensity show low a.m. cortisol. Low stores of excitatory neurotransmitters, particularly norepinephrine and epinephrine, can also influence energy levels. Other reasons for fatigue may 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 toxin levels, inactivity, and certainly many medications have fatigue as an adverse side effect. Aging in general and andropause can present with low testosterone which can contribute to decreased stamina. Assessment of thyroid, iron status, blood sugar, and diet may all be warranted.

Patient indicated WEIGHT GAIN on the symptom questionnaire. Weight gain can result from neuroendocrine imbalances—in monoamine neurotransmitter systems, in the HPA axis and/or in thyroid hormones, as well as a myriad of other hunger-related hormones. Obesity itself is associated with deficient serotonin 2C receptor function. In fact, serotonin has been shown to block ghrelin's appetite-inducing function and thus is considered appetite-inhibitory. Serotonin has been also shown to reduce meal size, likely by priming the PVN of the hypothalamus to respond to CCK. Dopamine deficiency also plays a role, since many appetite suppressants work by activation of dopamine in the NAcc (reward pathway). Norepinephrine's effects on weight gain are complex. NE stimulates secretion of ghrelin, boosting appetite, and NE is shown to inhibit leptin production in adipose tissue. However, SNRIs that inhibit NE reuptake are used to suppress food intake and so contribute to weight loss. Balancing the monoamines will be basic to controlling weight gain. There is another important imbalance that heavily predisposes us to weight gain – that of a dysbiotic microbiome. Over the last decade, science has begun to focus on the variations in phyla of intestinal bacteria in health vs. disease. Studies have shown that Firmicutes-predominant flora can extract carbohydrate calories from the diet more efficiently for the host than can Bacteroidetes phyla, resulting in weight gain. Fermented foods in the diet and/or probiotic supplements are recommended. Other factors which may contribute to weight gain include genetics, lack of exercise, fatigue, and excessive alcohol or carbohydrate intake. Excessive dietary carbohydrate intake, over time, (especially when combined with high cortisol from stress) can lead to insulin resistance, directing fat deposition to the abdominal region. Reduced sleep duration is now also considered an independent risk factor for weight gain.

HPA-G Complete™ Profile

THERAPEUTIC RECOMMENDATIONS

Kacie Doe
ID#: 16076
Gender: F Age: 63

Sanesco Clinician
1010 Merrimon Ave
Asheville, NC 28803 United States

Date Reported
01/07/2019

Patient is in: Initial Phase

The following therapeutic protocol is based on patient lab results, gender, age, lifestyle factors, and complaints listed on the patient questionnaire. The goal of this protocol is to help the practitioner begin the three-phase process of restoring balance to the HPA axis. The Initial Phase is the beginning of the rebalancing process. Here, Targeted Nutritional Therapy™ (TNT) is introduced to help address deficiencies and support the system in a trajectory toward wellness. Please note, leaving the patient on the initial protocol longer than suggested may perpetuate imbalance by either over or under employing the nutritional support. This is where retesting can be helpful. Retesting initiates the Restoration Phase and provides value by guiding the practitioner while he or she adjusts or fine-tunes the TNT protocol. In addition, it provides the patient and practitioner with a touchstone or guidepost as the patient's lab values improve.

Overall Summary and Recommendations

Prolent™	*x 1 in the PM for inhibitory support; based on the clinician's assessment and judgement, may increase to x 2 after 10 days.* *Contains: 5-HTP, Suntheanine, Glycine, and Vitamin B6*
Lentra™	*x 1 daily for GABA support; increase to twice daily after 5 days.* *Contains: GABA-A agonists: Magnesium Taurate, Suntheanine, and Lactium*
Adaptacin™	*After 7-10 days, implement x 2 in the AM for adrenal support;* *Do not take after 2 PM as it may disrupt sleep.* *Contains: Bovine Adrenal Cortex, adaptogens, and vitamin cofactors*

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.

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THERAPEUTIC RECOMMENDATIONS

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

These comments are intended to be educational. Any final interpretation and therapeutic intervention are the sole responsibility of the patient's clinician.
For a more detailed interpretation of the individual patient's results, please call our Technical Department at: 866-670-5705.

For Sex Hormone Sample Report
Post Menopausal (Progesterone only)

NeuroLab®

A Division of Sanesco International

Patient Questionnaire

DOB 06 (M M) 26 (D D) 1957 (Y Y Y Y)

Is this your first laboratory test with NeuroLab? ☑ Yes ☐ No

First Name: Kacie | Middle Initial: | Last Name: Doe

Height (ft,in): 5' 4" | Weight (lb): 155 | Gender: ☐ Male ☑ Female

Phone | Email | Doctor/Healthcare Provider Name

IMPORTANT Completely fill out your sample collection times below (when applicable)

Urine Collection	1st saliva	2nd saliva	3rd saliva	4th saliva
Date	Date	Date	Date	Date
Time	Time	Time	Time	Time

Medical History

Fill in where applicable

Cancer History (specify): N/A

Surgical History (specify): C-Section; gall bladder removal

☐ Heart attack or stroke within last 12 months ☐ Alcoholism or other substance abuse ☐ Miscarriage or abortion within last 6 months

Last Menstrual Period (specify date): 2002

Which day of your cycle was your saliva collection on? (Day 1 is first day of your period.)

menopausal

Current Lifestyle Factors

Caffeine: # of cups/bottles per day: 1c | # of alcoholic drinks per week: 2 ☐ Stressful Lifestyle

☐ Vegetarian ☐ Exercise Regularly ☐ Irregular sleep schedule ☐ Regular consumption of soda or energy drinks

☐ Vegan ☐ Smoke ☐ Smokeless Tobacco ☐ IVF (in vitro fertilization)

Medical Diagnosis

Check all that apply

☐ ADD/ADHD ☐ Autism ☐ Bipolar ☐ Anorexia ☐ Bulimia ☐ Psychosis

☐ Hyperthyroidism ☐ Hypothyroidism ☐ Hypergonadism ☐ Hypogonadism ☐ Type I Diabetes ☐ Type II Diabetes

☐ Ovarian Cysts ☐ PCOS ☐ Erectile Dysfunction ☐ Hepatitis ☐ Cirrhosis ☑ Celiac Slightly

☐ Pregnant or breastfeeding ☐ Elevated Homocysteine ☐ Low ☑ High Blood Pressure ☐ Other Chronic Conditions (specify) _____

Current Medications | Indicate the number of months on the medication.

ADD/ADHD Meds		Anti-Inflammatory		Cancer Treatment		Parkinson's Meds	
Adrenal Glandular		Anti-Psychotic Meds		Diabetes Meds		Sleep Meds	sometimes
Allergy Meds		Birth Control		Hormones	Progest only ✓	Seizure Meds	
Anti-anxiety Meds		Blood Pressure Meds	✓	MAO Inhibitors		Thyroid Meds	
Anti-depressant	✓	Cardiac Meds		Pain Meds		Kidney Meds	

REVERSE SIDE REQUIRED ------>

Nutritional Supplements & Herbs

- [ ] Contegra™
- [ ] Lentra™
- [ ] MethylMax™
- [ ] Plenus™
- [ ] Procite-D™
- [ ] Prolent™
- [ ] SomniTR™
- [ ] Tranquilent™
- [ ] Adaptacin™
- [x] DHEA
- [ ] GABA
- [ ] Glutamine
- [x] Melatonin
- [ ] Phosphatidylserine
- [ ] SAMe
- [ ] St. Johns Wort
- [ ] Theanine
- [ ] Trytophan
- [ ] Tyrosine
- [ ] Phenylalanine
- [ ] 5HTP
- [ ] Kava Kava
- [ ] Passion Flower
- [x] Magnesium
- [ ] Probiotics

Please mark symptoms below based on the severity they cause you currently

If no symptoms are present check here ☐ 1 - (mild) 2 - (moderate) 3 - (severe) 4 - (profound) *- rate no more than 10 symptoms*

1	2	3	4	
☐	☐	☐	☐	Addictive behavior
☐	☐	☐	☐	Andropause symptoms
☐	☐	☒	☐	Anxiety
☐	☐	☒	☐	Apathy
☐	☐	☒	☐	Appetite (excessive)
☒	☐	☐	☐	Allergies (seasonal)
☐	☐	☐	☐	Cold Extremities
☐	☐	☐	☒	Decreased Libido
☐	☐	☒	☐	Decreased Stamina
☐	☐	☐	☐	Fatigue
☐	☐	☐	☐	Depression (with exhaustion)
☐	☒	☐	☐	Abdominal Weight Gain
☐	☐	☐	☐	Uterine Fibroids
☐	☐	☐	☐	Increased Facial/Body Hair

1	2	3	4	
☐	☐	☐	☒	Depression (with nervousness)
☐	☐	☐	☐	Headaches
☐	☐	☐	☐	Migraines
☐	☐	☐	☐	Hot Flashes
☐	☒	☐	☐	Night Sweats
☐	☐	☒	☐	IBS Constipation Dominant
☐	☐	☐	☐	IBS Diarrhea Dominant
☐	☐	☐	☐	Insomnia
☐	☐	☒	☐	Poor Sleep
☐	☐	☐	☒	Irritability
☐	☒	☐	☐	Nervousness
☐	☐	☐	☐	General Weight Gain
☐	☐	☒	☐	Vaginal Dryness

1	2	3	4	
☐	☐	☐	☐	Joint Pain
☐	☐	☐	☐	Arthritis
☐	☐	☐	☐	Lack of Focus
☐	☐	☐	☐	Obsessive/Compulsive behavior
☐	☐	☐	☐	Pain (general)
☐	☐	☐	☐	PMS
☐	☐	☒	☐	Poor Memory
☐	☐	☐	☐	Salt Cravings
☐	☐	☐	☐	Shakiness when meal is skipped
☐	☐	☐	☐	Hand Tremors
☐	☐	☐	☒	Sugar Cravings
☐	☐	☐	☐	Unintentional Weight Loss
☐	☐	☐	☐	Fibrocystic Breasts/Breast Tenderness

List the name, dosage, and frequency of all current medications. For hormones, please include method of application.

Progesterone 20 mg 2X/day -cream
Lasix 40mg 1X
Celexa 20mg 1X
Ambien 5mg -occasionally

List the name, dosage, and frequency of all current supplements.

DHEA 15mg 1X

Privacy Statement

"I certify that the information provided in this questionnaire is accurate to the best of my knowledge. I understand that the information contained in this questionnaire may be used for the processing and release of your healthcare services to my provider as detailed in the enclosed notice of privacy practices of Sanesco International Inc. and for anonymous population research. My signature indicates that I have received, reviewed, and understand the above information."

Signature _____ Date _____