NutriPATH
INTEGRATIVE PATHOLOGY SERVICES
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SAMPLE REPORT
09-May-1990 Female

16 HARKER STREET
BURWOOD VIC 3125

Dr.SAMPLE REPORT
TEST HEALTH CENTRE
123 TEST STREET
BURWOOD VIC 3125

LAB ID : 3814217
UR NO. :
Collection Date : 09-May-2022
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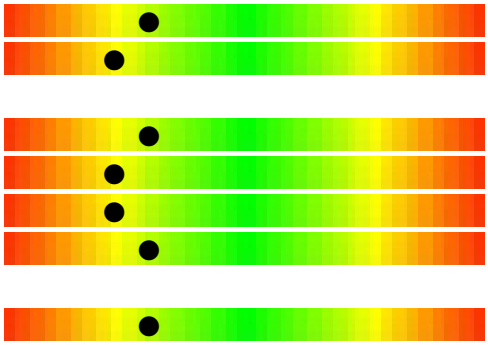
INTEGRATIVE MEDICINE

URINE, SPOT	Result	Range	Units
EXTENSIVE NEUROTRANSMITTER PROFILE			
Inhibitory Neurotransmitters			
SEROTONIN Urine	**57.7**	47.6 - 140.3	ug/gCR
GABA, Urine	***136.0* *L**	167.0 - 463.0	ug/gCR
Excitatory Neurotansmitters			
GLUTAMATE Urine	**1629.0**	1213.0 - 4246.	ug/gCR
DOPAMINE, Urine	***82.0* *L**	103.0 - 282.0	ug/gCR
NORADRENALIN (Nor-Epinephrine)	***7.7* *L**	10.0 - 35.7	ug/gCR
ADRENALIN (Epinephrine)	**1.3**	0.8 - 6.2	ug/gCR
Adrenal Adaptation Index			
Noradrenalin/Adrenalin Ratio	**5.9**	2.9 - 25.2	RATIO



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(L) Result is below lower limit of reference range

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Optimal Ranges Table

Biomarker	Adult Optimal Range (>11 Yrs)	
INHIBITORY TRANSMITTERS:		
Tryptophan	3970 - 8450	ug/gCr
SEROTONIN	100 - 215	ug/gCr
5HIAA	2988 - 5850	ug/gCr
GABA	400 - 600	ug/gCr
Glycine	61 - 159	ug/gCr
Taurine	24.5 - 134	ug/gCr
EXCITATORY TRANSMITTERS:		
Glutamine	37 - 71	ug/gCr
GLUTAMATE	2520 - 3700	ug/gCr
Histidine	19.7 - 58.4	ug/gCr
Histamine	5.2 - 15.3	ug/gCr
PEA	5.3 - 16.1	ug/gCr
Tyrosine	4790 - 10278	ug/gCr
Tyramine	279 - 588	ug/gCr
DOPAMINE	200 - 330	ug/gCr
DOPAC	658 - 1449	ug/gCr
HVA	3737 - 7048	ug/gCr
Noradrenaline	18.5 - 25.5	ug/gCr
Normetanephrine		
VMA	2580 - 4766	ug/gCr
Adrenaline	1.4 - 4.2	ug/gCr
INFLAMMATORY MARKERS:		
Kynurenine	257 - 960	ug/gCr
Kynurenic Acid	639 - 1200	ug/gCr
3-Hydroxykynurenine	147 - 467	ug/gCr
Xanthurenic Acid	694 - 1510	ug/gCr

There are multiple factors that play roles in neurotransmitter levels (Lifestyle, receptors, meds, supplements, diet, stress, etc).
The optimal reference ranges stated above have been determined/derived statistically from historical patient data.
Historically, these levels were achieved in the majority of patients as they experienced symptom relief or improvement.

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CORRELATIONS TO QUESTIONNAIRE

The following section is designed to give you an analysis of neurotransmitter and adrenal hormone values and an observation of how they affect one another. This approach targets the underlying cause of chronic symptoms by addressing the root imbalance. In this section, we will observe trends in the lab values, correlating those with the symptoms that were marked by the patient in the questionnaire.

ADRENAL INFLUENCES
Although the patient chose to only test neurotransmitter levels, an adrenal panel is suggested should any of the following symptoms arise: allergies, symptoms of hypoglycemia (shakiness when a meal is skipped), decreased stamina, fatigue, insulin resistance (sugar cravings, fatigue, abdominal weight gain, poor sleep), decreased libido, stress, salt cravings, which are all related to low adrenal function.

Patient checked ALLERGIES on the questionnaire.
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 be elevated, contributing to the poor sleep pattern so often seen in allergic individuals. Allergies should 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 encasing the mattress and pillow cases and/or eliminating as much clutter as possible, through limiting pillows, stuffed animals, carpets and curtains, etc. may be helpful. Also anti-inflammatory nutraceuticals such as quercetin and nettle extract may be of value.
FURTHER ASSESSMENTS:
An adrenal hormone assessment is highly recommended for this patient.

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) 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.
FURTHER ASSESSMENTS:
Assessment of thyroid, iron status, blood sugar, diet and adrenal function are all warranted.

Patient checked Stress which has system-wide effects on the body's communication system. Chronic stress can become cumulative and may have an especially deleterious effect over time. The perception of stress stimulates immediate release of epinephrine and norepinephrine, followed shortly by release of cortisol and DHEA. The effects of these hormones are beneficial in a short-term, life-threatening situation. Chronic stress, however, generates a cascade of effects. Prolonged stress leads to elevated levels of norepinephrine and epinephrine and decreased turnover in the synaptic space, with chronically high cortisol levels. DHEA levels rise initially but soon decrease. This is significant because DHEA plays a role in protecting nerves from the neurotoxic effects of glucocorticoids, benefiting stress tolerance and resilience. Low levels of DHEA have been associated with chronic illnesses ranging from CFIDS to depression to rheumatoid

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conditions. Continuously elevated cortisol levels contribute to the aging process and are associated with declining immune function. An increased cortisol/DHEA ratio is specifically thought to interfere with T-cell immunity.

Elevated cortisol may damage the overall regulation of the Communication System by interrupting the natural mechanisms of recovery. In addition, elevated cortisol is associated with promoting insulin resistance and weight gain. GABA is the primary inhibitory neurotransmitter. GABA's regulating and calming role is supported by adequate serotonin. Initially, GABA will make a compensatory rise to counter excitatory hormones and neurotransmitters. However, over time, a toll may be taken on GABA stores leading to a state of deficiency. When this happens, feelings of stress and anxiety may not be alleviated. Supporting both GABA and serotonin is recommended.

Avoid supporting excitatory neurotransmitters, even when decreased, before replenishing serotonin and GABA. DHEA support may also be considered.

FURTHER ASSESSMENTS:

An adrenal hormone assessment is highly recommended due to the presence of stress.

* The following are additional recommendations to assist in recovery from or to prevent adrenal fatigue: Adequate nutrient intake including multivitamin/multimineral, B-vitamin (Pantothenic Acid), Vitamin C, Magnesium, and Omega 3 Fatty Acids. Consider hormone support if necessary for DHEA, Pregnenolone, Progesterone, as well as adrenal support. Supportive lifestyle factors include structuring proper sleep hygiene with 8-10 hours per night; avoid stimulants and limit coffee, soda, nicotine, and caffeine; eat a balanced diet of small meals interspersed throughout the day and include lean protein, unprocessed carbohydrates, and healthy fats; increase water consumption to at least 64 oz per day; gentle exercise; make time for quietude.

INHIBITORY NEUROTRANSMITTERS

Patient indicated symptoms of ANXIETY, NERVOUSNESS, and IRRITABILITY.

These symptoms are often the result of decreased inhibitory neurotransmission and/or excess excitatory neurotransmission. Additionally, in the presence of up-regulated adrenal function, anxiety, irritability, and/or nervousness may also be present; therefore, consider assessing adrenal hormone 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. 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 indicated DEPRESSION as a concern on the questionnaire.

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 los, 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.

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

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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.
FURTHER ASSESSMENTS:
Optimal thyroid function is paramount to comprehensive treatment of depression. As medical research is replete with references regarding mood and thyroid function, consider adding a comprehensive thyroid assessment.
As elevated cortisol and low DHEA levels have a known association with presence of depression; consider assessing adrenal hormone levels.

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.

The patient has indicated problems with SLEEP on the questionnaire.
Serotonin function may not be optimal to support proper sleep. Serotonin is the biochemical precursor to melatonin, another very important sleep hormone. 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.
FURTHER ASSESSMENTS:
Assessment of thyroid, sex hormones and adrenal function are all warranted.

SUGAR CRAVINGS may be the result of several factors.
Inadequate levels of serotonin can often be a cause. Where serotonin is low, serotonin support is needed. When there is insulin resistance, adequate blood sugar is not getting into the cells. This can result in cellular signals to the CNS to increase carbohydrate intake. At times, an overgrowth of candida yeast can cause sugar cravings. Consider ruling out this possibility. The adrenal hormones play an important role in blood sugar homeostasis, and glucose/insulin balance; therefore, dysregulation of the adrenal gland can result in sugar cravings. Consider assessing adrenal hormone levels and function.
FURTHER ASSESSMENTS:
Assessment of thyroid, blood sugar, diet and adrenal function are all warranted.

EXCITATORY NEUROTRANSMITTERS
Patient indicated ADDICTIVE BEHAVIORS to be a concern.
Whether the behavior is related to food, alcohol, tobacco, recreational drugs or shopping, there are common patterns often noted in neuroendocrine imbalances. A notable pattern of imbalance is in dopamine levels. As the reward center, dopamine deficiencies often play a role in the cravings that manifest as part of the repetitive cycle in addictive behaviors. Low levels of dopamine may result in a lack of motivation, coupled with cravings, which may affect the patients ability to stop addictive behavior and/or to sustain their recovery. Other imbalances that predominate include decreased serotonin and imbalanced GABA levels that can perpetuate symptoms of agitation, mood swings, increased pain perception and possibly sleep disruption. As a result, excitatory levels may become increased, further promoting the resultant symptoms. Over time, however,

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deficiencies may be noted in all areas of the neuroendocrine system with low thyroid and glucose/insulin imbalances as well as deficiencies in nutrients that are required for neurotransmitter and hormone production. To begin, it is suggested to support neurotransmitter and hormone production while reestablishing nutrition through a whole food diet and supplementation of essential oils, vitamins, and minerals. Overcoming addictions requires the initial and continuing input from a physician/practitioner trained in treating patients with addictive behaviors.

Patient indicated APATHY on symptom questionnaire.
Apathy is defined as lack of interest, ambition, and/or "drive" and is frequently associated with low catecholamines, especially dopamine and norepinephrine. Dopamine is associated with the concept of salience "why we do what we do". Salience is connected with reward-seeking behaviors. Dopamine also strongly influences libido, drive, and focus, while norepinephrine also plays a role in focus, memory, and even sleep. Apathy can be concurrent with symptoms of depression, and balancing serotonin along with catecholamine levels in these particular cases may be beneficial. Although the patients lab results show normal dopamine, the presence of apathy concerns suggest suboptimal catecholaminergic function, indicating a need for support. MTI (minimal thyroid insufficiency) is a mild form of hypothyroidism with discrete somatic manifestations of thyroid deficiency. In some cases, typical depressive symptoms, such as discouragement, lethargy and apathy manifest as a result of decreased thyroid function; therefore, consider assessing thyroid hormone levels. The practitioner might also consider assessing sex hormone levels particularly in light of testosterone and DHEA s role in dopaminergic agonist function.

Patient checked OBSESSIVE/COMPULSIVE behavior on the questionnaire.
OCD is characterized by recurrent, unwanted thoughts, and/or repetitive behaviors with concomitant anxiety and distress. OCD symptoms can worsen with stress indicating a thorough investigation of adrenal gland dysfunction and possible stressors. While the exact mechanisms of the disorder are unknown, studies indicate that serotonin and dopamine may play a role in OCD. Other research indicates elevated CSF glutamate levels in OCD patients. Alterations in the cerebral serotonin receptor system have been linked to patients with OCD, and low availability of dopamines D2 receptor in people with the disorder suggests that this catecholamine may be involved as well. Seventy-eight percent (78%) of patients who ranked OCD as being moderate to severe had low to low normal dopamine and serotonin levels.

Supporting serotonin and dopamine levels and function may be warranted. For some OCD patients, inositol has proven to be beneficial, even in conjunction with SSRI medications.

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 products are not intended to 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 Nutripath on 1300 688 522 with your technical and clinical questions. For further reading and references, please refer to Nutripath's Technical guide and Clinical guide.

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INHIBITORY NEUROTRANSMITTERS.

SEROTONIN LEVELS LOW NORMAL

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), depleted neuron stores (poor nutrition with high demand), Interference from other signaling chemicals, Low carbohydrate diet, High protein competition, Heavy metal toxicity, Inflammation.

Clinically, low serotonin is associated with anxiety, depression, changes in appetite, cravings, excessive worry, heightened sensitivity to pain, hot flashes, hunger, low mood, migraine, obsessive compulsive disorder, panic disorder, sleep disturbances, and worsened PMS symptoms.

TREATMENT:

When serotonin is low, supplementation with cofactors to promote biosynthesis (e.g. vitamin B6), precursors (tryptophan/5-HTP), theanine, SAMe, Carnitine, St Johns Wort, SSRI, Massage, Melatonin, Hydroxy-tryptophan, Vit B6, Fish Oils, and probiotics may be helpful.

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.

GABA LEVELS LOWER THAN THE REFERENCE RANGE.

The brain's major inhibitory neurotransmitter GABA functions as the off switch in the brain. GABA is essential to limiting excitation so that input signals are balanced and not overdone. GABA prevents anxiety, improves mood, promotes sleep, lowers blood pressure, acts as a muscle relaxant, aids in formation and storage of fear memories, increases insulin secretion and decreases blood glucose levels. Clinically, low GABA levels are implicated in anxiety, depression, headaches, menopause symptoms, panic attacks, post-traumatic stress disorder, and sleep difficulties. Low GABA levels may also be associated with adrenal distress and HPA axis dysfunction, and disorders like attention deficit hyperactivity disorder and Tourette syndrome.

TREATMENT:

Supplementation with GABA, L-theanine, cofactor support (e.g. B6), growth hormone-releasing hormone, Ginko biloba, Ashwagandha, Kava, Valerian root, Melissa off (lemon balm), Scutellaria sinensis (skullcap), Gotu Cola, Magnolia and Phellodendron bark, and probiotics may be helpful. Caffeine has been found to inhibit GABA release, so avoidance may be beneficial. Additionally, yoga and meditation increase brain GABA levels.

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EXCITATORY NEUROTRANSMITTERS.

GLUTAMATE LEVELS NORMAL:
The brain's major excitatory neurotransmitter glutamate (also known as glutamic acid) functions as the "on" switch in the brain. Glutamate regulates appetite, thinking (cognition), increases gut motility, optimizes learning, modulates memory, mood and perception of pain, improves libido, and decreases sleep. The brain is the major contributor of glutamate in the body.

DOPAMINE LEVELS LOW:
May be low due to chronic damage to Dopamine neurons, receptors, transporter, excess serotonin levels, chronic use of drugs of abuse, alcohol, nicotine, ADD drugs.
TREATMENT:
Tyrosine, B6, Cocoa, Rhodiola, Green tea, Carnitinem, Theanine, Siberian ginseng, Dopamine inhibitors, Caffeine, Guarana, Yohimbine, Phenylalanine, L-Dopa, folic acid, thiamine, protein-rich diet.

NOREPINEPHRINE LEVELS LOW:
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 sugar, reduces digestive activity, pain, and sleep, prevents bladder emptying, and regulates body temperature. Norepinephrine is very similar in structure and physiological effects to epinephrine. The adrenal gland produces approximately 20% of the total output with 80% produced by the sympathetic nerve fibers.
Urinary norepinephrine is reduced in patients with Alzheimer's disease and may also be low due to the following: Toxic or other damage to Norepinephrine neurons, Depletion of stores (impact of stress and poor diet), Adrenal fatigue/exhaustion, Excess prostaglandin E2.
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, Cocoa, Rhodiola & Green tea and Vitamin C and vitamin B6 may be beneficial.

EPINEPHRINE LEVELS LOW NORMAL.
Epinephrine functions both as a neurotransmitter and a hormone, participating in the body's "fight or flight" response. Approximately 80% of peripheral catecholamine output by the adrenal glands is epinephrine.
Reduced urine epinephrine is seen in Alzheimer's disease, metabolic syndrome, and obesity. Clinically, low epinephrine is implicated in attention impairment, chronic stress, depression, cold body temperature, dizziness, chronic fatigue, hypotension, low mood and libido, and memory issues.
TREATMENT:
Adrenal support may be beneficial to increase epinephrine levels.

Creatinine, Urine Spot.	**10.9**	5.0 - 13.0	mmol/L	

Tests ordered: ENEUM
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