NutriPATH
INTEGRATIVE PATHOLOGY SERVICES

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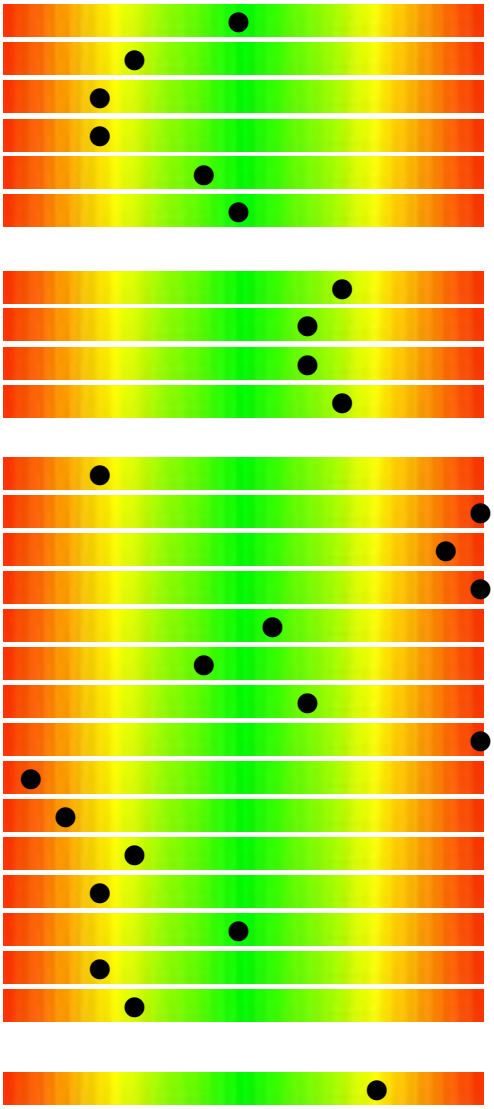
Clinical Notes: Development delays, Aggresive Behaviour, ADD/ADHD

INTEGRATIVE MEDICINE

URINE, SPOT	Result	Range	Units
Advanced Neurotransmitter Profile			
Inhibitory Neurotransmitters			
Tryptophan, Urine	7876	2633 - 12688	ug/gCR
SEROTONIN Urine	201.6	182.2 - 366.8	ug/gCR
5HIAA, Urine	*5331* *L	6331 - 18834	ug/gCR
GABA, Urine	*215.0* *L	270.0 - 633.0	ug/gCR
Glycine.	188	99.0 - 330	ug/gCR
Taurine.	136	7.1 - 293	ug/gCR
Urinary Inflammatory Markers			
Kynurenine	1493.0	108.0 - 1641.0	ug/gCR
Kynurenic Acid	1345.0	437.0 - 1719.0	ug/gCR
3-Hydroxykynurenine	593.0	80.0 - 822.0	ug/gCR
Xanthurenic Acid	2029.0	450.0 - 2175.0	ug/gCR
Excitatory Neurotansmitters			
Glutamine.	27.0	27.0 - 106	ug/gCR
GLUTAMATE Urine	*27064* *H	2157.0 - 4735.	ug/gCR
Histidine.	*121* *H	10.8 - 98.9	ug/gCR
Histamine, Urine	*53.0* *H	14.5 - 38.4	ug/gCR
N-methyl Histamine.	146.0	59.0 - 195.0	ug/gCR
PhenylEthylamine PEA	16.9	6.8 - 32.3	ug/gCR
Tyrosine.	12080	3128 - 15548	ug/gCR
Tyramine	*1237* *H	187 - 910	ug/gCR
DOPAMINE, Urine	*189.0* *L	382.0 - 770.0	ug/gCR
DOPAC.	*307.0* *L	1090.0 - 5203.	ug/gCR
HVA.	8493	7295 - 21929	ug/gCR
NORADRENALIN (Nor-Epinephrine)	*18.4* *L	23.9 - 61.1	ug/gCR
NorMetanephrines	45.0	24.9 - 70.1	ug/gCR
ADRENALIN (Epinephrine)	*1.7* *L	2.8 - 13.0	ug/gCR
VMA.	4854.0	4310.0 - 9207.	ug/gCR
Adrenal Adaptation Index			
Noradrenalin/Adrenalin Ratio	10.8	2.4 - 10.9	RATIO



(*) Result outside normal reference range (H) Result is above upper limit of reference rang (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.

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.

EXCITATORY NEUROTRANSMITTERS

The patient has been diagnosed as ADD/ADHD according to the symptom questionnaire submitted with the sample.
Many patients with a diagnosis of ADD also experience symptoms of multiple ALLERGIES, POOR SLEEP and POOR FOCUS, but not every classification of ADD is clinically similar. If hyperactivity is involved, support of the inhibitory system is essential; 70% of those indicating hyperactivity demonstrate levels of high normal norepinephrine. Moreover, several studies have shown that norepinephrine concentrations positively correlated with degree of hyperactivity of ADHD patients. At least 70% of our patient population indicating moderate to severe ADD have low normal serotonin levels, while 50% have low GABA levels. This underscores the need for inhibitory neurotransmitter support in over half the ADD population. If poor focus is an issue, use concurrent inhibitory support with a boost of the catecholamine pathway to support dopamine's role in focus. 71% of patients indicating moderate to severe issues with focus demonstrate low normal dopamine. Evaluating the levels of serum ferritin may be beneficial, as low levels are associated with ADHD in patients; iron modulates dopamine and norepinephrine production, as well as influences dopamine receptor activity. The presence of allergies is often a result of poor adrenal function (adrenal support useful) while poor sleep may be due to an over-excitation of the HPA axis from an excessive late rise in cortisol or excitatory neurotransmitters.

Of the patient population who indicated moderate to severe focus problems, 71% demonstrate low or low-normal dopamine. When **POOR FOCUS** is a symptom, use concurrent inhibitory support (to prevent over-excitation) with catecholamine pathway support to rebuild dopamine to restore focus and directed attention. Poor focus and memory issues can also be related to chronic stress and

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adrenal dysfunction. Decreased thyroid function is known to impede cognitive function; therefore, consider assessing thyroid hormone levels.

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.

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.

TRYPTOPHAN IS WITHIN THE REFERENCE RANGE:
Tryptophan originates from the diet and serves as a constituent of proteins and a precursor to neurotransmitters. A small fraction is used by the GI tract, with the remaining tryptophan pool distributed to peripheral circulation and transported to tissues such as the brain, heart, and skeletal muscle. Tryptophan not taken up by the upper GI tract is metabolized by resident microbiota.
Tryptophan is a substrate for two biosynthetic pathways relevant to the neuropsychiatric interface: the generation of serotonin and thereafter melatonin, and the formation of kynurenine derivatives and thereafter niacin (vitamin B3).

SEROTONIN LEVELS WITHIN RANGE:

Serotonin levels may be within range, however, if the patient is exhibiting symptoms you may wish to consider that the reported reference range is not optimal for this patient. Optimal Serotonin levels are levels at which serotonin can effectively counterbalance elevated excitatory neurotransmitters (esp Dopamine and Norepinephrine). Even if Serotonin is above the observed reference range but is not proportional to (or able to control) elevations in the catecholamines, then more Serotonin support is needed.
"

5-HIAA IS LOWER THAN THE REFERENCE RANGE:

5-HIAA is the primary metabolite of serotonin via the actions of monoamine oxidase and aldehyde dehydrogenase enzymes. Research shows that urinary 5-HIAA levels are reduced in patients with anorexia nervosa, and MAO deficiency .
TREATMENT CONSIDERATIONS:
Increasing serotonin levels using 5-HTP or tryptophan and supporting conversion from serotonin with adequate levels of copper and vitamin B2 may be beneficial.

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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GLYCINE IS WITHIN NORMAL RANGE:
Glycine is a simple, nonessential amino acid that plays a role in the production of DNA, phospholipids, collagen, creatine, heme and glutathione. Glycine serves as a neurotransmitter that modulates excitatory signals in the brain, and as an anti-inflammatory agent that calms aggression, improves sleep quality, stabilizes blood sugar, and improves metabolic parameters.

TAURINE IS WITHIN NORMAL RANGE:
Taurine is a semi-essential or conditionally essential sulfur-containing amino acid and an inhibitory (calming) neurotransmitter. Taurine improves sleep, relieves anxiety, alleviates fatigue, aids with metabolism and digestion, and promotes glucose control and electrolyte balance.
The main source of taurine is diet. Taurine protects healthy cells and tissues, functions as a potent antioxidant to reduce oxidative stress, mitigates mitochondrial and endoplasmic reticulum stress, inhibits lipid peroxidation, improves energy metabolism, regulates gene expression, and participates in detoxification, calcium homeostasis and osmoregulation processes. By fulfilling all these functions, taurine is therefore protective in cardiovascular health, improves lean body mass and exercise performance. With regard to brain health, taurine serves a neuroprotective role, promotes neural development in embryonic and adult brain tissues, and is an important factor in neurogenesis.

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EXCITATORY NEUROTRANSMITTERS.

GLUTAMINE IS LOW NORMAL:
Glutamine is an essential and should be the most abundant free amino acid in the human body.
Glutamine provides fuel for rapidly dividing cells (lymphocytes, enterocytes and intestinal epithelial cells), helps balance ammonia levels in the body, improves immune system function, contributes to biosynthesis of proteins, amino acids, nucleic acids and glutathione, and protects intestinal lining. Additionally, glutamine increases glutamate and GABA levels in the brain and in the body.
Although the body usually makes enough glutamine to meet all its needs, extreme stress (e.g., strenuous exercise, persistent stress, or injury) can increase the demand for glutamine beyond the amount naturally manufactured. Low circulating glutamine levels are reported after intense exercise, in overtraining syndrome, in diabetes, depression, and in autism spectrum disorder. Low glutamine levels are associated with high oxidative stress.
THERAPEUTIC CONSIDERATIONS:
Consider supplementation with glutamine which comes in capsules or powder. Glutamine is a fairly bland tasting amino acid and easily goes into smoothies. Glutamine is also high in chicken, fish, cabbage, spinach, diary, tofu and lentils among many over foods.

GLUTAMATE LEVELS ELEVATED:
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, improves libido, decreases sleep and contributes to oxidative stress. Chronic stress maintains high levels of glutamate in the brain which may lead to excitotoxicity and even neuronal damage. Research shows that urinary glutamate levels are high in patients with celiac disease and with hyperthyroidism. Clinically, high glutamate is suspected in anxiety, autism, bipolar disorder, depression, and impulsivity, inability to focus (racing thoughts), obsessive compulsive disorder, panic attacks, sleep difficulties, and stroke.
TREATMENT:
GABA, L-theanine, and taurine may be beneficial to counter glutamate actions. Vitamin E and N-Acetyl Cysteine (NAC) may be used to combat oxidative damage. Cofactor supplementation with vitamins B3 and B6, and magnesium and NAC may aid with glutamate metabolism.

HISTIDINE IS ELEVATED:
Histidine is a semi-essential amino acid that gives rise to the neurotransmitter histamine. Histidine protects neurons, assists with making new blood cells, reduces inflammation and oxidative stress, helps with tissue repair and growth. Histidine ameliorates fatigue, promotes clear thinking/concentration, reduces appetite, decreases anxiety, improves sleep and glucose homeostasis.
Research shows that histidine excretion is high with histidine administration, in histidinemia, and in diabetic nephropathy.
THERAPEUTIC CONSIDERATIONS:
As high histamine has not been linked to adverse symptoms, no therapy is thought to be needed.

HISTAMINE IS ELEVATED:

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Histamine is both a neurotransmitter and a modulator of the immune system that has anti-pain properties, plays a neuroprotective role in the brain, and contributes to optimal maintenance of cognition and memory.
Histamine stimulates wakefulness and decreases sleep, stimulates gastric acid production, increases metabolism, suppresses appetite, and prevents weight gain.
Histamine is a potent vasodilator and a pro-inflammatory agent.
Urinary histamine is high in patients with burns, flushing disorder, food allergies, cystitis, polycythemia, and pregnancy.
Clinically, high histamine levels are implicated in allergies, depression, headaches, migraines, OCD, schizophrenia, sensitivity to chemicals, and sleep difficulties.

PEA LEVELS WITHIN REFERENCE RANGE:
PEA, also known as phenethylamine, promotes energy, elevates mood, and regulates attention.
PEA also contributes to aggression, serves as a biomarker for ADHD, and prolongs the signaling of dopamine, norepinephrine, and serotonin.

TYROSINE IS HIGH-NORMAL:
Elevated tyrosine is usually due to nutritional supplementation, but has also been found secondary to hyperthyroidism, diabetes and liver disease. Genetic disorders of tyrosinemia also exist but are uncommon and are usually diagnosed in the first year of life due to unique mental and physical disabilities. Unless due to genetic disorders levels are rarely high enough to cause symptoms.
Tyrosine is obtained from diet (sesame seeds, cheese, soy, meat, nuts and fish) or synthesized in the body from the amino acid phenylalanine. Tyrosine serves as a constituent of proteins and gives rise to neurotransmitters, like dopamine, norepinephrine and epinephrine; and the trace-amine tyramine. Additionally, in the thyroid gland, tyrosine can also be iodinated to give rise to thyroid hormones.
Tyrosine enhances cognitive performance, energy, and alertness, and improves memory after sleep deprivation. Tyrosine also prevents the depletion of central and peripheral catecholamines (dopamine, norepinephrine, epinephrine) induced by acute stress, thereby eliciting protective effects on behavioral and cardiovascular parameters in the body.
THERAPEUTIC CONSIDERATIONS:
In adults, consider checking thyroid, insulin, and liver function labs to screen for problems and address underlying metabolic dysfunction. Decreasing the dosage of supplementation and avoiding foods high in tyrosine may be beneficial.

TYRAMINE IS ELEVATED:
Usually due to eating foods high in this trace amine (protein).
Specifically, tyramine is found in aged, fermented cured or spoiled food where microbes with decarboxylase enzymes convert tyrosine to tyramine. These foods include aged cheeses, smoked fish, cured meats, wine, and some types of beer. In sensitive individuals, high tyramine ingestion can trigger migraines by causing blood vessel restriction and then rebound vasodilation. Additionally, tyramine can trigger norepinephrine release, thereby stimulating sympathetic nervous system and consequently increase blood pressure. Because of this sympathetic mechanism, symptoms of agitation, anxiety, rapid heartbeat, and headaches may be noted.
THERAPEUTIC CONSIDERATIONS:
Avoid tyramine high foods and calm the sympathetic nervous system. Supplements such as SAMe, magnesium, vitamin B2 may aid with promoting norepinephrine metabolism. Additionally, nervines, adaptogens, L-theanine, biofeedback and meditation may help

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quiet down the overactive sympathetic response.

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:
Levels may be low due to the following: Chronic hypoglycaemia, Low epinephrine and chronic drug use, Low epinephrine and adrenal exhaustion, Low epinephrine and low cortisol, Low epinephrine and methyl donor supply (Vitamin B12 and SAMe).

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EXCITATORY METABOLITES

N-METHYLHISTAMINE IS HIGH NORMAL:
N-Methylhistamine is a major metabolite of the neurotransmitter histamine. Research shows that N-methylhistamine excretion is elevated with high protein diet, in gastrointestinal food allergies, in irritable bowel disease, in colitis, in histidinemia, in chronic urticaria, in angioedema, and with interstitial cystitis.
THERAPEUTIC CONSIDERATIONS:
Evaluation for food allergy sources may be warranted.

DOPAC LOWER THAN THE REFERENCE RANGE:
DOPAC is the primary metabolite of dopamine formed via the actions of monoamine oxidase.
Research shows that DOPAC is reduced in the urine of patients with Alzheimer's disease.

HVA IS LOW NORMAL:
HVA is a dopamine metabolite formed through the actions of the monoamine oxidase and catechol-Omethyl transferase enzyme. Research shows that HVA is reduced in the urine of patients with monoamine oxidase enzyme deficiency, polycystic ovarian syndrome and periodic limb movement disorder.

NORMETANEPHRINE IS WITHIN THE REFERENCE RANGE:
Normetanephrine is a norepinephrine metabolite formed via the actions of catechol-O-methyl (COMT) transferase enzyme in response to stress.

VMA IS LOW NORMAL:
VMA is a norepinephrine and epinephrine metabolite formed via the actions of monoamine oxidase (MAO), catechol-O-methyl transferase (COMT), and aldehyde dehydrogenase.
IF Epinephrine is Normal High to High, this suggests reduced epinephrine metabolism (by these 3 enzymes), contributing to higher levels of circulating epinephrine. Studies show that in some patients with depression (self-reported), epinephrine excretion is markedly elevated, without concurrent increases in VMA. Research shows that in rare cases, VMA is reduced in patients with MAO deficiency or on SSRI and SNRI combination therapy. Higher epinephrine levels can stimulate hyperglycaemia and may possibly explain insulin resistance and other cardiovascular events reported to occur in depression.

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INFLAMMATORY MARKERS

KYNURENINE IS HIGH-NORMAL:
Kynurenine is a central metabolite of the amino acid tryptophan with vasodilatory properties.
Kynurenine is utilized by the body in the production of niacin (vitamin B3), eventually leading to the formation of NAD+, which plays a pivotal role in energy metabolism, gene expression, cell death and regulation of calcium homeostasis. More than 90% of the body's tryptophan is metabolized to the kynurenine pathway.
Kynurenine is synthesized by the enzyme tryptophan dioxygenase (TDO), which is expressed primarily but not exclusively in the liver, and indoleamine 2,3-dioxygenase (IDO), which is made in many tissues in response to immune activation by interferons and cytokines, or free radicals. In the brain, approximately ~40% of kynurenine is produced locally, whereas the rest is absorbed from the blood.
Kynurenine degradation generates a series of neuroprotective and neurotoxic compounds that can activate or inhibit N-methyl-d-aspartate (NMDA) glutamate receptors (see kynurenic acid and 3-OH kynurenine). Upregulation of this pathway may be a hallmark of neuroinflammation and is associated with certain disorders.
Research shows that kynurenine is high with tryptophan administration, hydrocortisone treatment, metabolic syndrome, with major coronary events, and in women in pregnancy. High kynurenine levels have been implicated in disorders like Irritable Bowel Syndrome, lupus, Crohn's disease, and Alzheimer's Disease. Additionally, caffeine and regular black tea consumption can elevate kynurenine levels as well.
TREATMENT CONSIDERATIONS:
Reduction of inflammation through diet and supplementation may be beneficial. Glutathione support and modulation of the NMDA receptor (e.g. magnesium) may help reduce symptoms.

KYNURENIC ACID IS HIGH-NORMAL:
Kynurenic acid is a neuroactive metabolite produced from kynurenine. Kynurenine is formed from tryptophan via the enzyme tryptophan dioxygenase (TDO) and indoleamine 2,3-dioxygenase (IDO); and metabolized along two independent pathways to produce kynurenic acid via aminotransferases and 3-OH kynurenine.
Kynurenic acid (unless in excess amounts) is regarded to have a neuroprotective role because it inhibits the N-methyl-d-aspartate (NMDA) glutamate receptor, reduces the neurotransmitter glutamate release and thereby prevents excitotoxicity.
Research shows that urinary kynurenic acid levels are high with tryptophan administration and metabolic syndrome. High kynurenic acid levels are implicated in schizophrenia.
TREATMENT CONSIDERATIONS:
Evaluate tryptophan supplementation and blood sugar regulation. Consider anti-inflammatory diet and supplements to reduce neuroinflammation.

3-HYDROXY-KYNURENINE IS HIGH-NORMAL:
3-Hydroxy Kynurenine (3-OH Kynurenine) is a metabolic intermediate of the kynurenine pathway, one of the major metabolites of tryptophan degradation. Kynurenine is transformed into 3-OH Kynurenine, which acts as a N-methyl-daspartate (NMDA) glutamate receptor agonist and has been demonstrated to exert neurotoxic effects.
Neurotoxicity elicited by 3-OH Kynurenine appears to be also related to generation of oxidative stress produced by reactive radical species, formed as a result of auto-oxidation. Additionally, 3-OH Kynurenine gives rise to neurotoxic metabolites,

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Clinical Notes: Development delays, Aggresive Behaviour, ADD/ADHD

such as quinolinic acid, which activate the NMDA receptor, induce lipid peroxidation and promote oxidative stress.
Research shows that urinary levels of 3-OH Kynurenine are high with hydrocortisone treatment and in women in pregnancy. High 3-OH Kynurenine is implicated in vitamin B6 deficiency and Alzheimer's disease.
TREATMENT CONSIDERATIONS:
Consider glutathione support and antioxidant support to prevent the oxidative stress produced by 3-hydroxyhynurenine. Consider B6 supplementation (under 200 mg/day for safety).

XANTHURENIC ACID IS HIGH-NORMAL:
Xanthurenic acid is a metabolite of the kynurenine pathway, formed directly from 3-OH Kynurenine, and serves as an indirect marker of vitamin B6 status. Vitamin B6 insufficiency leads to elevated levels of xanthurenic acid in urine.
Increased excretion of xanthurenic acid following tryptophan ingestion has been used a measure of vitamin B6 deficiency, which in scientific literature is called the tryptophan loading test (oral 2 g challenge). If xanthurenic acid levels are elevated in the absence of tryptophan administration, vitamin B6 deficiency is considered to be significant.
Vitamin B6 deficiency that contributes to elevated xanthurenic acid levels can also increase oxidative stress in the body. The hydroxylated quinone structure of xanthurenic acid can bind iron and increase DNA oxidative damage. Therefore, elevated xanthurenic acid levels may suggest antioxidant insufficiency.
Research shows that xanthurenic acid is high with vitamin B6 deficiency, with hydrocortisone treatment, rheumatoid arthritis, metabolic syndrome, autism spectrum disorder, and in women in pregnancy.
THERAPEUTIC CONSIDERATIONS:
Vitamin B6 supplementation with antioxidant support may be warranted.

Creatinine, Urine Spot.	**7.1**	5.0 - 25.0	mmol/L	

(*) Result outside normal reference range (H) Result is above upper limit of reference rang (L) Result is below lower limit of reference range