



Adrenal Hormone Report; saliva



Order: SAMPLE REPORT



Client #: 12345

Doctor: John Smith, MD

Doctors Data Inc

3755 Illinois Ave

St. Charles, 60175 IL

Patient: Sample Patient

Age: 31 DOB: 01/01/1986

Sex: Male

Sample Collection Date/Time

Date Collected 01/01/2017

Morning 01/01/2017 0800

Noon 01/01/2017 1200

Evening 01/01/2017 1700

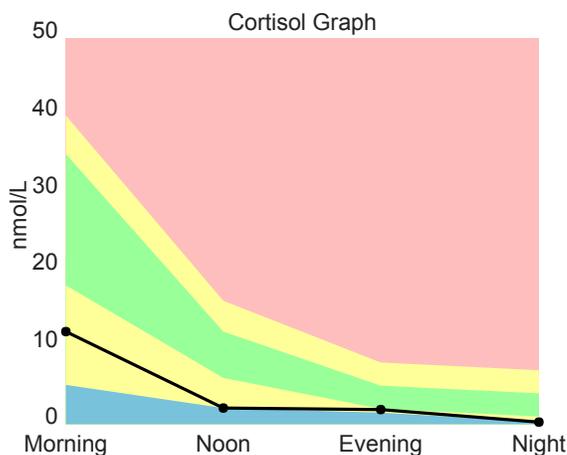
Night 01/01/2017 2100

Wake Up Time 01/01/2017 0800

Date Received 01/04/2017

Date Reported 01/06/2017

Analyte	Result	Unit	L	WR	H	Optimal Range	Reference Interval
Cortisol Morning	12	nmol/L		◆		18 - 35	5.1 - 40
Cortisol Noon	2.1	nmol/L		◆		6.0 - 12	2.1 - 16
Cortisol Evening	1.9	nmol/L		◆		2.0 - 5.0	1.5 - 8.0
Cortisol Night	<0.33	nmol/L	↓			1.0 - 4.0	0.33 - 7.0
DHEA*	125	pg/mL	↓				137 - 336



Hormone Comments:

- Diurnal cortisol pattern and reported symptoms are consistent with evolving (Phase 2) HPA axis (adrenal gland) dysfunction.
- DHEA level is consistent with the expected decline with age (adrenopause). The low DHEA level may warrant supplementation for optimal well-being. Note: Supplementation with DHEA may increase testosterone and/or estradiol levels.

Adrenal Phase: 2



Notes:

L (blue)= Low (below range), WR (green)= Within Range (optimal), WR (yellow)= Within Range (not optimal) H (red)= High (above range)

*This test was developed and its performance characteristics determined by Doctor's Data, Inc. The FDA has not approved or cleared this test; however, FDA clearance or approval is not currently required for clinical use. The results are not intended to be used as the sole means for clinical diagnosis or patient management decisions.

Methodology: Enzyme Immunoassay



Order: SAMPLE REPORT



Client #: 12345

Doctor: John Smith, MD

Doctors Data Inc

3755 Illinois Ave

St. Charles, 60175 IL

Patient: Sample Patient

Age: 31 DOB: 01/01/1986

Sex: Male

Sample Collection Date/Time

Date Collected 01/01/2017

Morning 01/01/2017 0800

Noon 01/01/2017 1200

Evening 01/01/2017 1700

Night 01/01/2017 2100

Wake Up Time 0800

Date Received 01/04/2017

Date Reported 01/06/2017

Analyte	Result	Unit per Creatinine	L	WR	H	Reference Interval
Serotonin	39.54	µg/g				42 - 105
Gamma-aminobutyrate (GABA)	1.1	nmol/g				1.4 - 5
Dopamine	87	µg/g				90 - 220
Norepinephrine	9.9	µg/g				12 - 50
Epinephrine	1.12	µg/g				0.9 - 9.2
Glutamate	54.56	nmol/g				8 - 45
Glycine	3600	nmol/g				280 - 2800
Histamine	22.15	µg/g				10 - 48
Phenethylamine (PEA)	11.9	nmol/g				16 - 146
Norepinephrine / Epinephrine ratio	9.0					< 10
Creatinine	101.54	mg/dL				



Neurotransmitter Comments:

- Urinary neurotransmitter levels provide an overall assessment of the body's ability to make and break down neurotransmitters and are representative of whole body levels. They are required for neurotransmission throughout the body. Direct assessment of neurotransmitter levels and metabolism in the central nervous system is not clinically feasible and approximately twenty percent of the total urinary levels are derived from the brain. The enzymes, cofactors and precursors in neurotransmitter metabolism in general are the same in the periphery and in the central nervous system. Therefore, alterations in urinary neurotransmitter levels assessed in urine provide important clinical information, and may be associated with many symptoms including cognitive and mood concerns, diminished drive, fatigue and sleep difficulties, cravings, addictions and pain.
- Low serotonin may contribute to mood concerns including anxiety, OCD, depression, anger and a sense of discontentment. Low serotonin may also be associated with poor sleep quality and appetite changes, as well as chronic fatigue, rheumatoid arthritis, and over-all lassitude. Production of serotonin requires vitamin D, tetrahydrobiopterin, iron and vitamin B6. Tryptophan is the essential precursor of serotonin. 5-HTP may increase serotonin, and L-theanine may affect serotonin function.
- Low GABA may be associated with anxiety, poor impulse control, major depression, pain, and decreased sleep quality. Low GABA may be seen in individuals deficient in vitamin B6. L-theanine, GABA, and glutamine may positively affect functional GABA activity, and phenibut exerts GABA-like effects (experimental models).
- Low dopamine levels may be associated with anxiety/depression, difficulty concentrating, obesity, reduced social bonding, and other stimulation seeking activities. Production of dopamine requires vitamin D, tetrahydrobiopterin, iron and vitamin B6. L-tyrosine, L-theanine and Mucuna pruriens may influence dopamine signaling.
- Low norepinephrine and low range epinephrine may be associated with depression and mood changes as well as fatigue, difficulty concentrating, decreased ability to stay focused on tasks and diminished sense of personal/professional drive. Norepinephrine is converted from dopamine requiring vitamin C, copper and niacin (B3). L-tyrosine, L-theanine and Mucuna pruriens influence this pathway.
- Elevated glutamate may contribute to anxiety, poor concentration, attention deficits and hyperactive tendencies as well as poor sleep and nighttime awakening. Glutamate may be increased in association with hypoglycemia, Alzheimer's, ALS and chronic compromised blood flow to the brain. Possible sources of increased glutamate include MSG, yeast extract and other hidden sources of free glutamic acid. L-theanine may modulate elevated glutamate levels and attenuate glutamate signaling, and taurine may provide protection from excitotoxicity and neuroinflammation.
- Elevated glycine levels may be associated with diminished intellectual functioning and adaptive behavior. Elevated levels may be seen with glycine supplementation, often used in conjunction with pharmaceutical agents when supporting schizophrenia or psychosis. Lipoic acid may enhance glycine break down. Break down of glycine requires vitamin B6 and tetrahydrofolate as cofactors. Note: High levels of glycine may interact with clozapine and decrease its clinical efficacy.
- Low phenethylamine (PEA) may be associated with depression, attention deficits and hyperactivity (ADHD), Parkinson's disease and bipolar disorder. Phenylalanine is the precursor amino acid to PEA, and vitamin B6 is a required co-factor in the conversion to this primary trace amine. Use of Reserpine can result in depletion of PEA.
- Upper range N/E ratio is consistent with poor conversion of norepinephrine to epinephrine. This conversion is driven by the phenylethanolamine N-methyltransferase (PNMT) enzyme that requires S-AdoMet, magnesium and cortisol (adequate HPA axis function) as cofactors. Suggest interpretation in context of cortisol levels/HPA axis function, with subsequent optimization of HPA axis function when clinically warranted.
- Considerations to address the demonstrated imbalances beyond the identified co-factors and amino acid precursors may include dosage adjustments if indicated, as well as nervine and adaptogenic herbs, methylation support, vitamin D, and gastrointestinal health optimization.
- Note: The reported low to low range monoamine neurotransmitters may be associated with genetic disruptions in methylation and/or suboptimal quantities of required co-factors. Further testing may be warranted.

Notes:

L (blue)= Low (below range), WR (green)= Within Range (optimal), WR (yellow)= Within Range (not optimal) H (red)= High (above range)

Methodology: LCMS QQQ, Creatinine by Jaffe Reaction