



Patient Name : **TEST PATIENT**  
 Date of Birth : December 27, 1966  
 Sex : F  
 Collected : May 11, 2015  
 Lab id : 3389105  
 Referring Physician: Dr TEST TOCTOR

### MICRO SAMPLE ASSAYS

DRIED URINE Result Range Units

#### ADRENAL PROFILE, Dried Urine

**Dried Urine Hormone Comments** Please refer to PDF attached.

**Patient Name:** TEST TEST

**Samples Collected** Urine - 00:00 Urine - 00:00 Urine - 00:00 Urine - 00:00

TEST NAME	RESULTS   10/18/18	RANGE
<b>Urinary Androgens</b>		
DHEA	13.07	9.01-93.80 µg/g Cr
<b>Urinary Glucocorticoids</b>		
Total Cortisol	68.36 H	8.73-28.52 µg/g Cr
Total Cortisone	72.65 H	14.12-42.84 µg/g Cr
Cortisol/Cortisone	0.94 H	0.5-0.7
Tetrahydrocortisol	517	201-597 µg/g Cr
Tetrahydrocortisone	1184 H	330-1126 µg/g Cr
<b>Urinary Free Diurnal Cortisol</b>		
Free Cortisol	36.29 H	7.8-29.5 µg/g Cr (1st Morning)
Free Cortisol	85.57 H	23.4-68.9 µg/g Cr (2nd Morning)
Free Cortisol	24.92 H	6.0-19.2 µg/g Cr (Evening)
Free Cortisol	11.76 H	2.6-8.4 µg/g Cr (Night)
<b>Urinary Free Diurnal Cortisone</b>		
Free Cortisone	174.43 H	31.6-91.6 µg/g Cr (1st Morning)
Free Cortisone	218.88 H	63.3-175.8 µg/g Cr (2nd Morning)
Free Cortisone	203.18 H	30.6-88.5 µg/g Cr (Evening)
Free Cortisone	125.71 H	15.5-44.7 µg/g Cr (Night)
<b>Urinary Creatinine</b>		
Creatinine (pooled)	1.02	0.3-2.0 mg/mL



TEST NAME	RESULTS   10/18/18	RANGE
<b>Urinary Creatinine</b>		
Creatinine	0.89	0.3-2.0 mg/mL (1st morning)
Creatinine	0.88	0.3-2.0 mg/mL (2nd morning)
Creatinine	0.30	0.3-2.0 mg/mL (Evening)
Creatinine	0.48	0.3-2.0 mg/mL (Night)

<dL = Less than the detectable limit of the lab. N/A = Not applicable; 1 or more values used in this calculation is less than the detectable limit. H = High. L = Low.

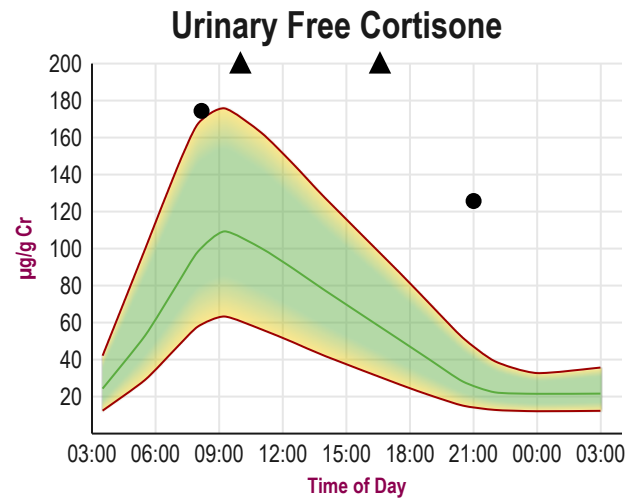
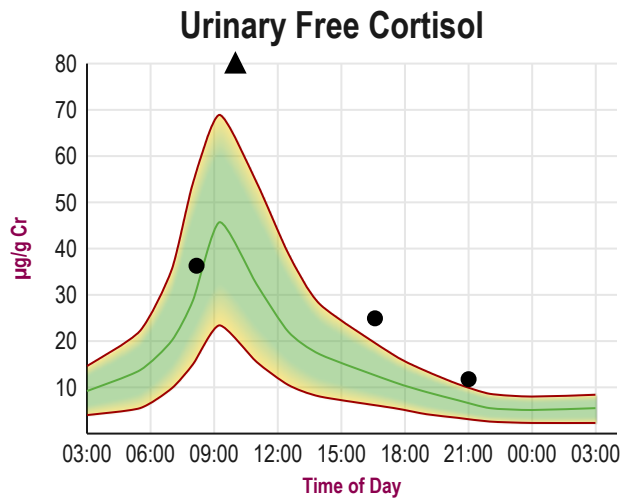
**Therapies**

None Indicated

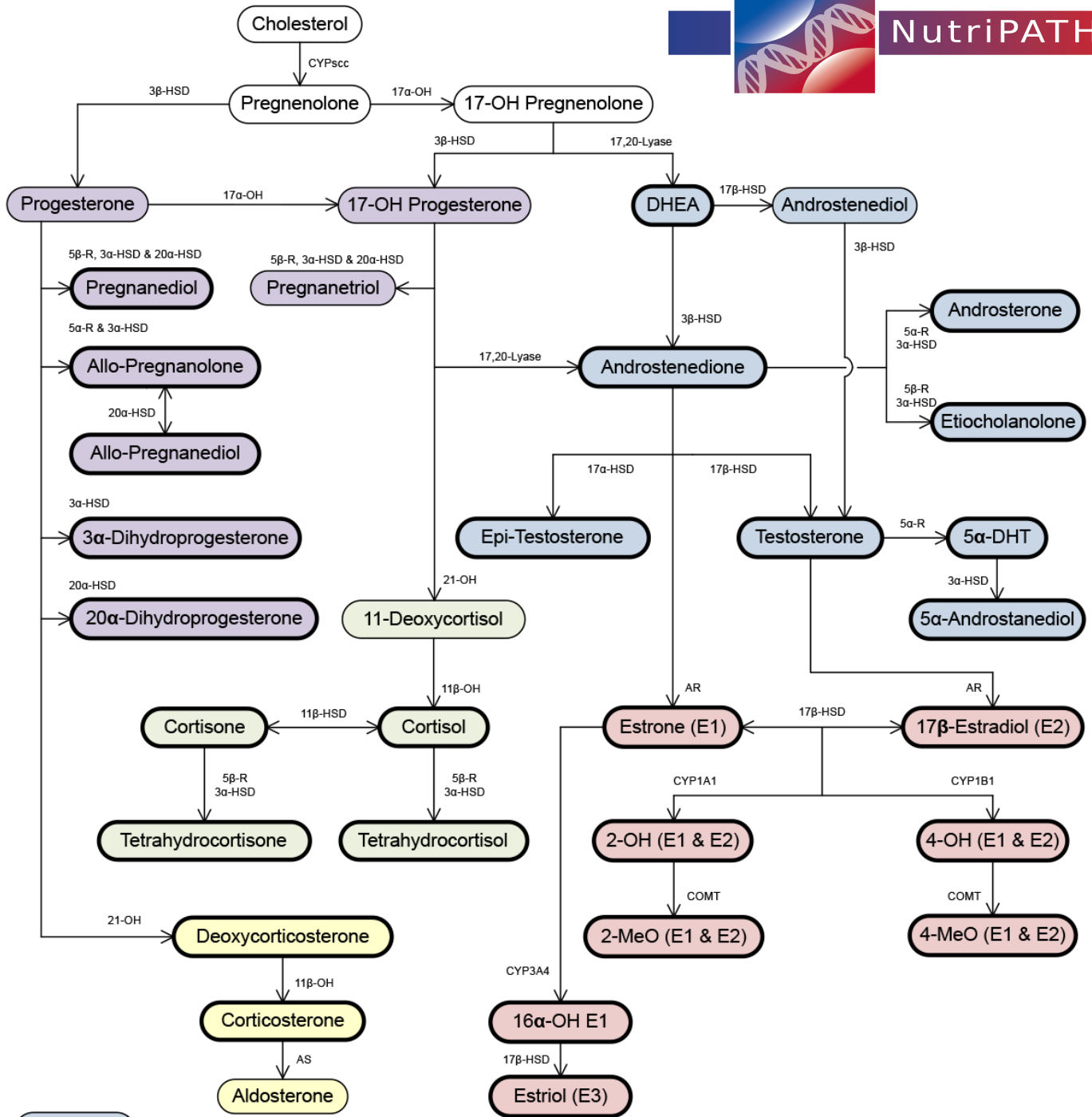
**Graphs**

**Disclaimer:** Graphs below represent averages for healthy individuals not using hormones. Supplementation ranges may be higher. Please see supplementation ranges and lab comments if results are higher or lower than expected.

— Average ▼▲ Off Graph

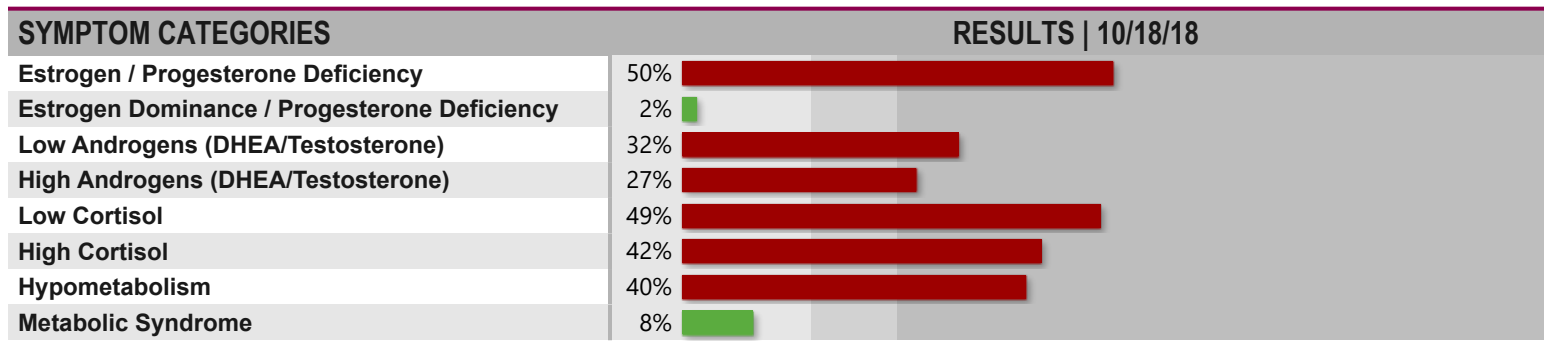


# The Steroid Hormone Cascade



- Androgens
- Estrogens
- Glucocorticoids
- Mineralocorticoids
- Progestogens

Enzyme Abbreviations	
(5α-R) 5α-Reductase	(11β-HSD) 11β-Hydroxysteroid dehydrogenase
(5β-R) 5β-Reductase	(17α-HSD) 17α-Hydroxysteroid dehydrogenase
(11β-OH) 11β-Hydroxylase	(17β-HSD) 17β-Hydroxysteroid dehydrogenase
(17α-OH) 17α-Hydroxylase	(20α-HSD) 20α-Hydroxysteroid dehydrogenase
17,20-Lyase (same enzyme as 17α-OH)	(AR) Aromatase
(21-OH) 21-Hydroxylase	(AS) Aldosterone Synthase
(3α-HSD) 3α-Hydroxysteroid dehydrogenase	(CYP) Cytochrome p450 (scc, 1A1, 1B1 & 3A4)
(3β-HSD) 3β-Hydroxysteroid dehydrogenase	(COMT) Catechol-O-Methyl-Transferase



SYMPTOM CHECKLIST	MILD	MODERATE	SEVERE
Acne			
Aggressive Behavior			
Allergies			
Anxious			
Apathy			
Blood Pressure High			
Blood Pressure Low			
Blood Sugar Low			
Body Temperature Cold			
Bone Loss			
Burned Out Feeling			
Chemical Sensitivity			
Cholesterol High			
Constipation			
Depressed			
Dizzy Spells			
Erections Decreased			
Fatigue - Evening			
Fatigue - Mental			
Fatigue - Morning			
Flexibility Decreased			
Forgetfulness			
Goiter			
Hair - Dry or Brittle			
Hair or Skin Oily			
Headaches			
Hearing Loss			
Heart Palpitations			
Hoarseness			
Hot Flashes			
Infertility			
Irritable			
Joint Pain			
Libido Decreased			
Mental Sharpness Decreased			
Muscle Size Decreased			
Muscle Soreness			
Nails Breaking or Brittle			
Neck or Back Pain			
Nervous			
Night Sweats			

SYMPTOM CHECKLIST	MILD	MODERATE	SEVERE
Numbness - Feet or Hands			
Prostate Cancer			
Prostate Problems			
Pulse Rate Slow			
Rapid Aging			
Rapid Heartbeat			
Ringing In Ears			
Skin Thinning			
Sleeping Difficulty			
Stamina Decreased			
Stress			
Sugar Cravings			
Sweating Decreased			
Swelling or Puffy Eyes/Face			
Triglycerides Elevated	BLANK		
Urinary Urge Increased			
Urine Flow Decreased			
Weight Gain - Breast or Hips			
Weight Gain - Waist			

## Lab Comments

### ANDROGEN & ESTROGEN PRECURSOR (DHEA)

The androgen precursor DHEA is within normal reference range for a male. DHEA(S) levels begin to rise at adrenarche beginning at about age 10 and then peak in the early 20's. Levels remain high in the early 20's and then begin to steadily decline thereafter. Urinary DHEA values from the teens until about age 30 should be within the upper quadrant of the reference range. By age 30-60 DHEA(S) reference values should be within mid-range. In those older than 60 DHEA(S) should be within the mid to lower reference range in individual considered healthy. In persons with insulin resistance/metabolic syndrome/diabetes the DHEA(S) values are usually much lower than age-adjusted reference range values due to a decrease in the activity of the adrenal enzymes 17,20-lyase (converts 17-OH pregnenolone to DHEA)(Ueshiba H et.al. Eur J Endocrinol 146 (3): 375-80, 2002). This shuttles the 17-OH pregnenolone more to 17-OH progesterone and through the cortisol synthesis pathway, leading to low DHEA(S) and high cortisol, a hallmark of diabetes.

DHEA is synthesized primarily in the adrenal glands from 17-OH pregnenolone (see above) and is rapidly sulfated to DHEA-sulfate (DHEAS) before being released into the bloodstream. Sulfation of DHEA extends its half-life in blood. Specific tissues throughout the body remove the sulfate (sulfatase) and convert DHEAS back to DHEA where it can be used as a substrate for creating estrone and testosterone via androstenedione (see Steroid Hormone Cascade). More conversion to estrone (via DHEA to androstenedione), occurs in individuals with higher levels of aromatase, which is higher in adipose (fat) tissue. DHEA supplementation is commonly used to raise testosterone levels in women; however, it is less effective in raising testosterone levels in men.

### TOTAL GLUCOCORTICOIDS

Total cortisol (F) and cortisone (E) are higher than the expected reference ranges, suggesting some type of adrenal stressor. The down-stream metabolites of cortisol and cortisone, tetrahydrocortisol (THF) and tetrahydrocortisone (THE) are within normal levels, indicating some strain on the adrenal glands to keep up with cortisol/cortisone synthesis. The total levels of these glucocorticoids are determined from the average of four urine collections throughout the day and are very similar to the 24 hour urine values. While 24 hr and 4-spot total cortisol urine tests provide useful information about the adrenal glands average capacity to synthesize cortisol and down stream metabolites in a day, they provide no information about the diurnal synthesis of cortisol throughout the day. In healthy individuals cortisol/cortisone synthesis should be high in the morning, drop progressively throughout the day, and be at the lowest level during the night while sleeping. Deviations from this pattern are associated with poor health and disease. Thus, total glucocorticoid production, while important, should be viewed in light of the diurnal cortisol pattern, which can be determined by testing cortisol 4x throughout the day in saliva, or urine (referred to as UFC-Urinary Free Cortisol).

While a high cortisol is a normal and healthy response to an acute stressor, a persistent stressor and chronic high cortisol can lead to multiple dysfunctions and disease. Elevated cortisol is usually caused by different types of stressors (emotional, physical-(e.g. excessive exercise, injury, surgery), chemical-(e.g. environmental pollutants, medications), inflammations-(e.g. cancer, metabolic syndrome), pathogens-(e.g. bacterial, fungal, viral infections).

Typical acute symptoms/signs of high cortisol can include anxiety, nervous-irritability, self-perceived stress, sleep disturbances. More chronic elevated cortisol is commonly associated with the same symptoms seen with acutely high cortisol but also include memory problems, depression, loss of muscle mass, and weight gain in the waist. Insulin resistance and metabolic syndrome are also a consequence and cause of

elevated cortisol, as are the diseases of aging such as diabetes, cardiovascular disease, cancer, and bone loss. When cortisol remains high these symptoms/conditions/syndromes/diseases progressively become more problematic over time.

For additional information about strategies for supporting adrenal health and reducing stress(ors), the following books are worth reading: "Adrenal Fatigue", by James L. Wilson, N.D., D.C., Ph.D.; "The Cortisol Connection", by Shawn Talbott, Ph.D.; "The End of Stress As We Know It" by Bruce McEwen; "Awakening Athena" by Kenna Stephenson, MD.

#### URINARY FREE CORTISOL (F) AND CORTISONE (E)

Urinary free cortisol (F) and cortisone (E) are following a normal circadian rhythm but are higher than the normal reference ranges throughout the day. High levels of both F and E are caused by excessive stress(ors), the most common of which include psychological stressors (emotional), physical insults (surgery, injury, diseases such as cancer), chemical exposure (environmental pollutants, excessive medications), hypoglycemia (low blood sugar), and pathogenic infections (bacterial, viral, fungal).

While increased cortisol synthesis is a normal response to acute stressors, levels return to normal when the stressor is removed. However, persistent stressors and chronic high cortisol production by the adrenal glands over a prolonged period of time (months/years) can lead to dysfunction in most endocrine systems (sex-hormones, thyroid, and growth hormone are diminished) which leads to excessive breakdown of normal tissues (muscle wasting, thinning of skin, bone loss) and immune suppression. High cortisol also leads to insulin resistance and elevated blood sugar, a prelude to diabetes. High cortisol, particularly if it is elevated at night, is associated most commonly with symptoms and conditions such as sleep disturbances, vasomotor symptoms (hot flashes and night sweats despite normal or high estrogen levels-mostly seen in women, but also in men with low androgens), fatigue, depression, weight gain in the waist, bone and muscle loss. Excessive cortisol may also decrease the hypothalamic-pituitary response to TSH production as well as thyroid hormone synthesis in the thyroid gland. High cortisol also interferes with T4 to T3 conversion by thyroid deiodinases within target tissues, and action of thyroid hormone (T3) with the thyroid receptor (excess cortisol down-regulates thyroid receptors) at the target tissue level.

As seen in these results, cortisone is also higher than reference ranges throughout the day, indicating very high cortisol synthesis (cortisone + cortisol). Cortisol is converted to the inactive form, cortisone, by the enzyme 11-beta hydroxysteroid dehydrogenase type II (11-beta HSD-II) (for review see: Seckl JR and Chapman KE Eur J Biochem 249, 361-364, 1997). This enzyme is expressed at high levels in tissues such as the kidneys, liver, lungs, colon, adipose tissue, and salivary glands where it plays an important role in preventing excess buildup of cortisol in tissues. If cortisol is allowed to accumulate to high levels in tissues it will activate the mineralocorticoid receptor (at normal levels cortisol only activates the glucocorticoid receptors) and can lead to mineralocorticoid excess syndrome, causing high blood pressure and low potassium levels.

The activity of 11-beta HSD-II is increased with growth hormone, estrogens, and androgens. Estrogen replacement therapy in women or androgen (testosterone) replacement therapy in men will increase the activity of 11-beta HSD-II and accelerate conversion of cortisol to cortisone. This is why higher physiological levels of estrogens and androgens seen during younger years are associated with a smaller waist circumference (visceral or belly fat) and with menopause and andropause these sex-hormones diminish and waistlines (belly fat) thicken.

Because chronic stressors and associated high night cortisol can have adverse effects on health and wellbeing, it is important to develop strategies to identify and eliminate or reduce the stressors or consider bioidentical hormone replacement therapies, foods, and/or nutritional supplements that help control excessive accumulation of cortisol. For additional information about adrenal dysfunction and strategies for adrenal support and lowering stress/cortisol levels the following books and journal articles are worth reading: "The Role of Stress and the HPA Axis in Chronic Disease Management" by Thomas Guillems, PhD; "Adrenal Fatigue," by James L. Wilson, N.D., D.C., Ph.D.; "The Cortisol Connection," by Shawn Talbott, Ph.D.; "The End of Stress As We Know It," by Bruce McEwen.

Creatinine is within range throughout the day reflecting normal concentration of urine.