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TEST PATIENT

GUa d'Y'HYgh'BUa Y
 Sex : :
 DUHY Collected : 00-00-0000
 111 H9GH'ROAD TEST SUBURB
@AB =8: 00000000 UR#:0000000

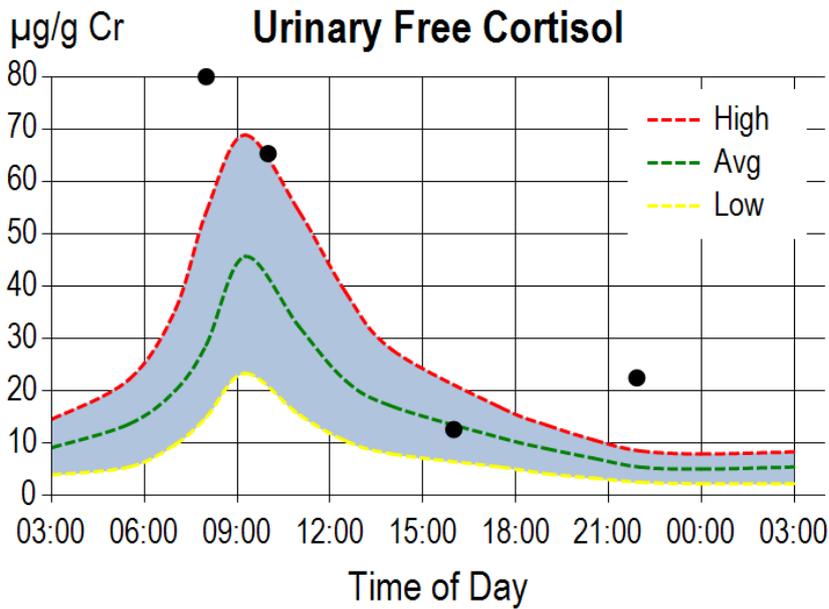
TEST PHYSICIAN

DR JOHN DOE
 111 CLINIC STF 99H
 7@B=7 'GI 6I F6 'J =7 ' \$\$\$

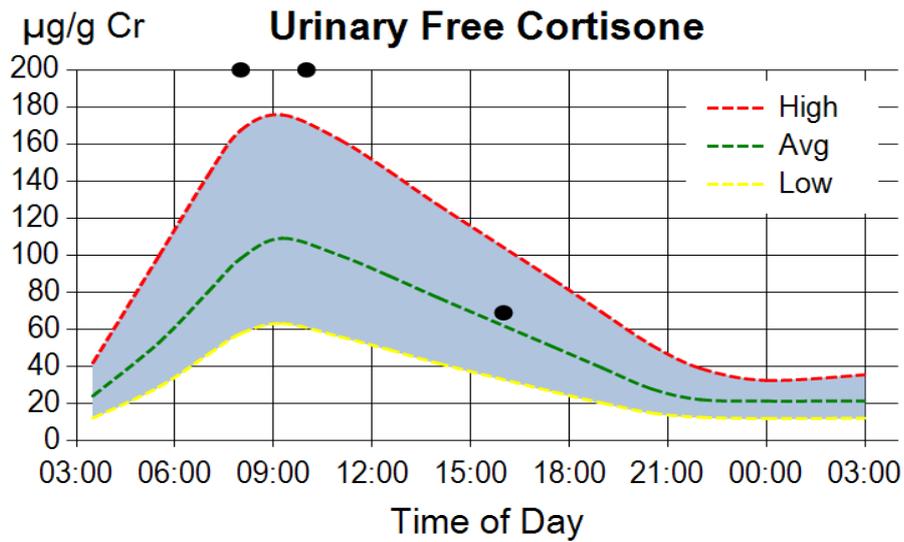
MICRO SAMPLE ASSAYS

DRIED URINE Result Range Units
ADRENAL PROFILE, Dried Urine

FREE CORTISOL AT A GLANCE



FREE CORTISONE AT A GLANCE





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DRIED URINE TESTING -INTEGRATIVE MEDICINE

Samples Collected: Urine: 00/00/0000 08:00 Urine: 00/00/0000 10:00 Urine: 00/00/0000 16:00 Urine: 00/00/0000 21:55

Menses Status: Postmenopausal
 Gender: Female

BMI: 00
 Height: 0 ft in
 Weight: 00 kg

Test Name	Result	Range
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Urinary Androgens (µg/g Cr)

DHEA (Urine) 15.86 8.63-37.28 Postmenopausal

Urinary Glucocorticoids (µg/g Cr)

Total Cortisol (Urine)	40.09	H 13.23-39.26 Postmenopausal
Total Cortisone (Urine)	75.86	H 23.32-59.61 Postmenopausal
Cortisol/Cortisone (Urine)	0.53	0.5-0.7
Tetrahydrocortisol (Urine)	441	281-711 Postmenopausal
Tetrahydrocortisone (Urine)	1525	H 551-1474 Postmenopausal

Urinary Free Diurnal Cortisol (µg/g Cr)

Free Cortisol (Urine)	88.14	H 23.4-68.9 (2nd Morning)
Free Cortisol (Urine)	65.32	23.4-68.9 (2nd Morning)
Free Cortisol (Urine)	12.66	6.0-19.2 (Evening)
Free Cortisol (Urine)	22.51	H 2.6-8.4 (Night)

Urinary Free Diurnal Cortisone (µg/g Cr)

Free Cortisone (Urine)	264.31	H 63.3-175.8 (2nd Morning)
Free Cortisone (Urine)	249.59	H 63.3-175.8 (2nd Morning)
Free Cortisone (Urine)	69.09	30.6-88.5 (Evening)
Free Cortisone (Urine)	133.42	H 15.5-44.7 (Night)

Urinary Creatinine (mg/mL)

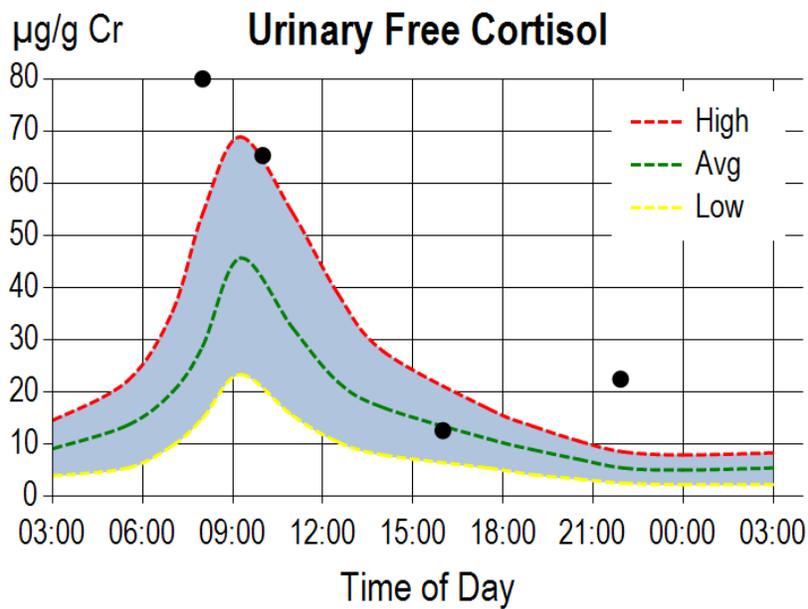
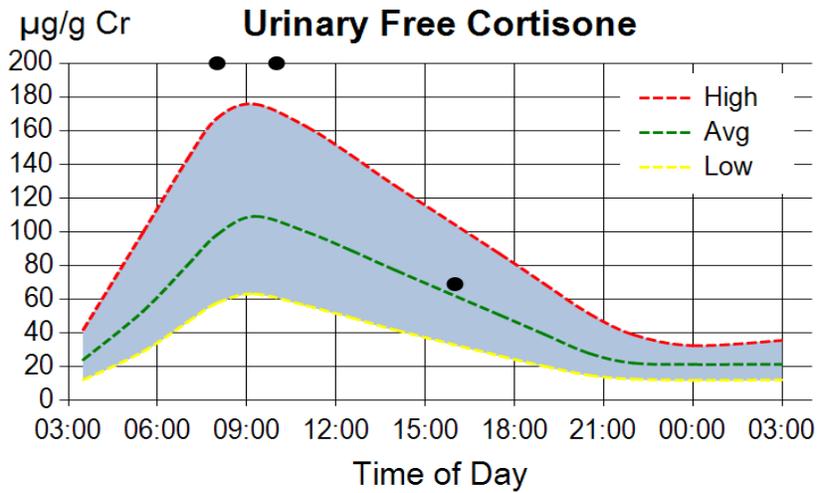
Creatinine (pooled) (Urine)	0.60	0.3-2.0
Creatinine (Urine)	0.41	0.3-2.0 (2nd morning)
Creatinine (Urine)	0.30	0.3-2.0 (2nd morning)
Creatinine (Urine)	1.04	0.3-2.0 (Evening)
Creatinine (Urine)	0.18	L 0.3-2.0 (Night)

<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.

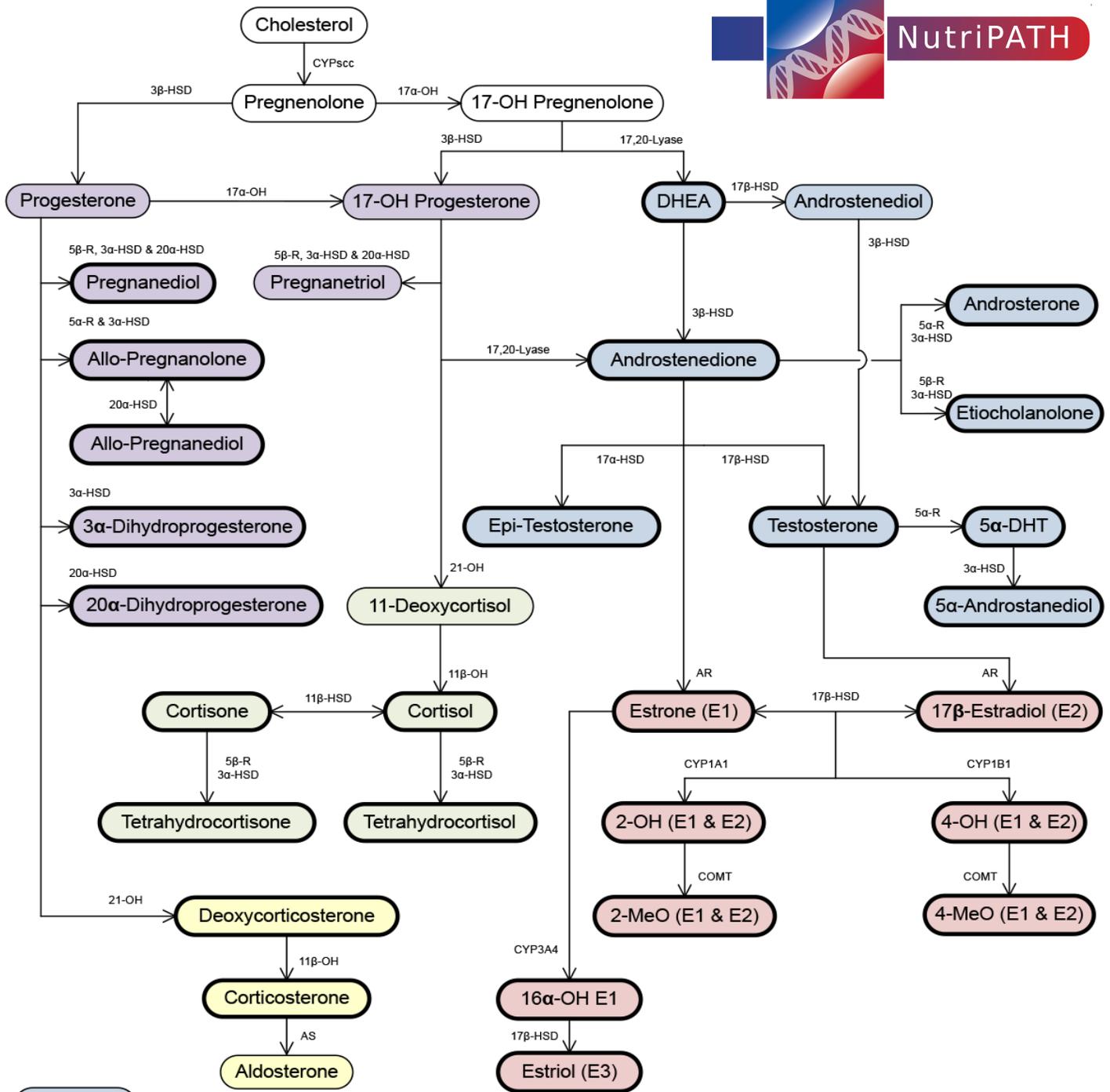
Therapies

None Indicated





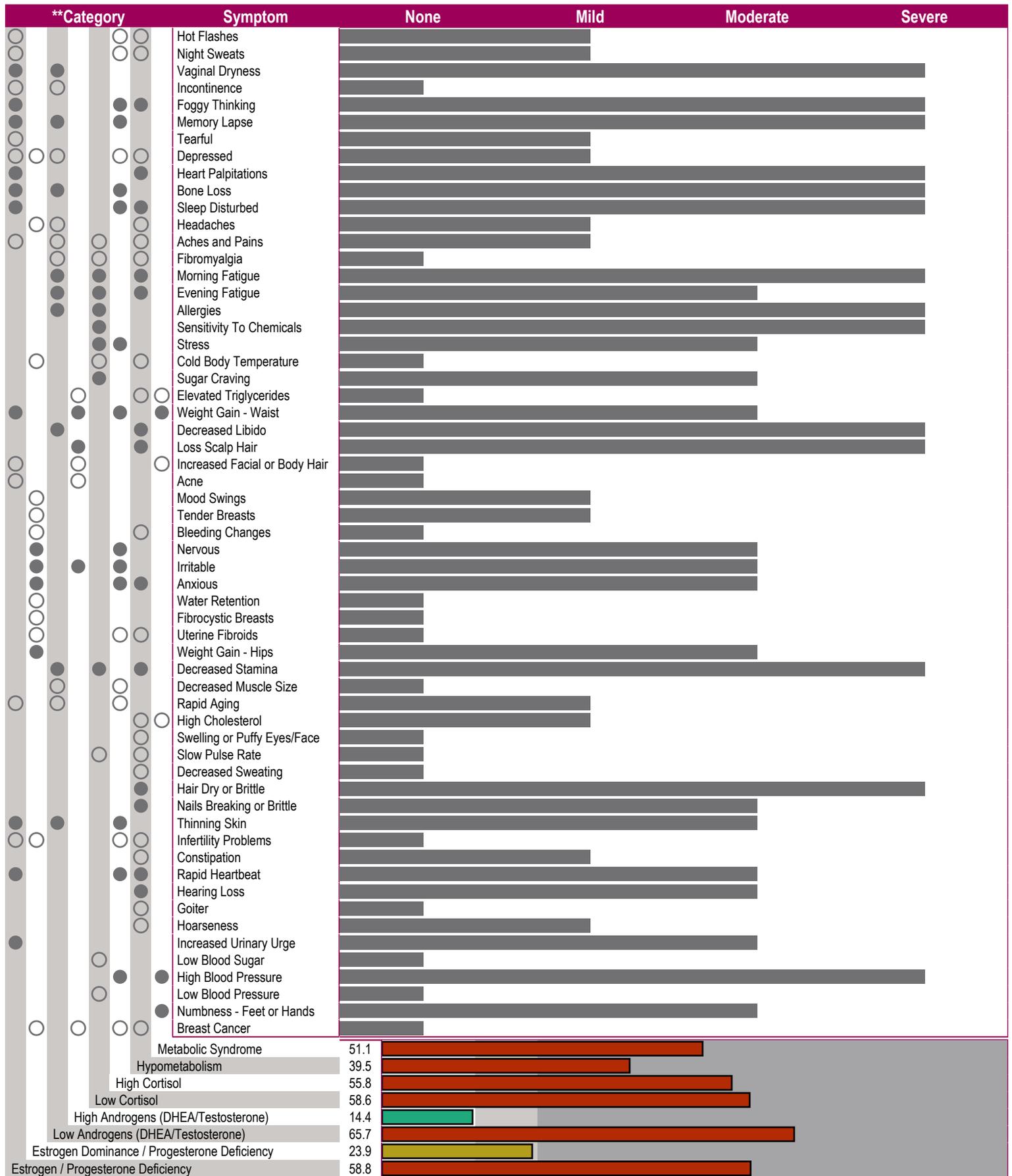
The Steroid Hormone Cascade



- 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





**Category refers to the most common symptoms experienced when specific hormone types (eg estrogens, androgens, cortisol) are out of balance, i.e., either high or low.

Lab Comments**ANDROGEN PRECURSOR (DHEA/S)**

Total urinary DHEA is within normal reference range. DHEA is synthesized in the adrenal glands and is rapidly sulfated to DHEA-sulfate (DHEAS) to extend its half-life in blood. DHEA is converted to androstenedione and then to testosterone and Epi-testosterone in near equal amounts in most individuals, or into estrone. More conversion to the estrogen, estrone, occurs in individuals with higher amounts of adipose (fat) tissue.

DHEA is considered a universal precursor to both androgens (androstenedione, testosterone, DHT), and estrogens (estradiol and estrone). DHEA is commonly used as a supplement to raise both DHEA and testosterone levels in women. Much less DHEA is converted to T and DHT in men.

DHEA itself has very little androgenic activity and serves mostly as a precursor to other downstream more potent metabolites (androgens and estrogens). In the sulfated form DHEA sulfate (DHEAS) plays an important role in the integrity of the immune system via binding to specific DHEAS binding sites on lymphocytes. In the brain DHEAS acts as a neuroactive steroid where it modifies dopaminergic pathways responsible for uplifting mood and increasing feeling of wellbeing.

TOTAL GLUCOCORTICOIDS

Total cortisol (F) and cortisone (E), and their down-stream metabolites, tetrahydrocortisol (THF) and tetrahydrocortisone (THE), are higher than the expected reference ranges for a postmenopausal woman. 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. Under stress situations the adrenal glands normally respond by increasing cortisol output. While the total daily cortisol metabolites are high it does not reveal if levels are only high at certain times of the day, or are persistently high all day long. Please see the Urinary Free Cortisol (UFC) to determine which of these scenarios pertains to this patient.

While a high cortisol is a normal and healthy response to an acute stressor, high cortisol caused by a persistent stressor 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) that lead to high cortisol, 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 (UFC)

The UFC is elevated throughout most of the day, particularly in the early morning with first void, and before bed at night. High urinary cortisol suggests adrenal stressors that raise endogenous cortisol synthesis by the adrenal glands, or less likely, the use of a glucocorticoid (e.g. hydrocortisone or other anti-inflammatory steroids commonly use for asthma or allergies)(none indicated).

In a normal individual without significant stressors, cortisol begins to rise in the morning shortly after awakening and steadily drops throughout the day, reaching the lowest level during sleep in the very early morning about 2 am. The first urine void is representative of the overnight production of cortisol and should be low if no stressors were occurring during the night, or early in the morning just before rising. The second void, which optimally is collected about 2 hours after waking, is representative of the awakening response and should be the highest level of the four collections. It is equivalent to a first morning saliva or blood cortisol measurement. Differences in the first and second morning samples are equivalent to a salivary Cortisol Awakening Response (CAR). If the adrenal glands are functioning normally, and stressors are minimal the second void should be significantly higher than the first void, but remain within the reference range. Excessive stressors that occur during the morning may cause the second cortisol measurement to rise above the reference range. If the adrenals are healthy and the stressor acute the cortisol should drop by the third and fourth measurement, but remain within normal reference range. If the stressor persists, the cortisol levels will usually remain above the reference range throughout the remainder of the day. However, if the adrenals are exhausted, or substrate (17-OH progesterone) and nutrients (e.g. Vitamins B5 and C) are unavailable for cortisol synthesis, a second morning cortisol may be high, normal, or low, but cortisol usually drops to low levels in the evening and night

voids.

The most common stressors that can raise cortisol levels include psychological stressors (emotional), physical insults (surgery, injury, diseases), chemical exposure (environmental pollutants, excessive medications), hypoglycemia (low blood sugar), and pathogenic infections (bacterial, viral, fungal). Acute stressors such as exercise are expected to raise cortisol levels over the interval of the stressor, which is a normal response to the stressor(s) and is essential for optimal health. However, chronic and persistent stressors and chronic high cortisol production by the adrenal glands over a prolonged period of time (months/years) can lead to excessive breakdown of normal tissues (muscle wasting, thinning of skin, bone loss) and immune suppression. Chronic high cortisol, particularly if it is elevated throughout the day or high 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), fatigue, depression, weight gain in the waist, bone and muscle loss. Many of these symptoms associated with both high cortisol are self-reported, suggesting that this condition is likely chronic.

For additional information about strategies for supporting adrenal health and reducing stressors, 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; "Thyroid Power", by Richard Shames, MD.