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TEST PATIENT

GUa d'Y HYghBUa Y
 Sex : :
 DUH 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 F 6'J =7'' \$\$\$

DRIED URINE-INTEGRATIVE MEDICINE

DRIED URINE

Androgen Elite, Dried Urine

Samples Collected: Urine: 00/00/000 07:00 Urine: 00/00/000 10:00 Urine: 00/00/000 17:00 Urine: 00/00/000 21:00

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

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

Estradiol (Urine)	0.40	0.18-0.49
Estrone (Urine)	1.54	0.57-1.67
Estriol (Urine)	0.37	0.18-0.64
2-OH Estrone (Urine)	0.82	H 0.16-0.48
16α-OH Estrone (Urine)	0.10	0.06-0.21
2-MeO Estrone (Urine)	0.15	0.05-0.15
2-MeO E1/2-OH E1 (Urine)	0.18	L 0.20-0.38

Urinary Progesterones (µg/g Cr)

Pregnanediol (Urine)	324	H 47-140
Allopregnanolone (Urine)	1.19	0.32-1.20

Urinary Androgens (µg/g Cr)

DHEA (Urine)	158.87	H 9.01-93.80
Androstenedione (Urine)	12.49	H 2.12-9.51
Testosterone (Urine)	17.39	H 3.81-14.21
Epi-Testosterone (Urine)	9.12	H 3.15-8.85
T/Epi-T (Urine)	1.91	0.5-3.0
5α-DHT (Urine)	2.61	H 0.71-2.46
5α,3α-Androstenediol (Urine)	30.00	H 9.48-24.96

Urinary Glucocorticoids (µg/g Cr)

Total Cortisol (Urine)	36.51	H 8.73-28.52
Total Cortisone (Urine)	64.23	H 14.12-42.84
Cortisol/Cortisone (Urine)	0.57	0.5-0.7
Tetrahydrocortisol (Urine)	580	201-597
Tetrahydrocortisone (Urine)	1520	H 330-1126

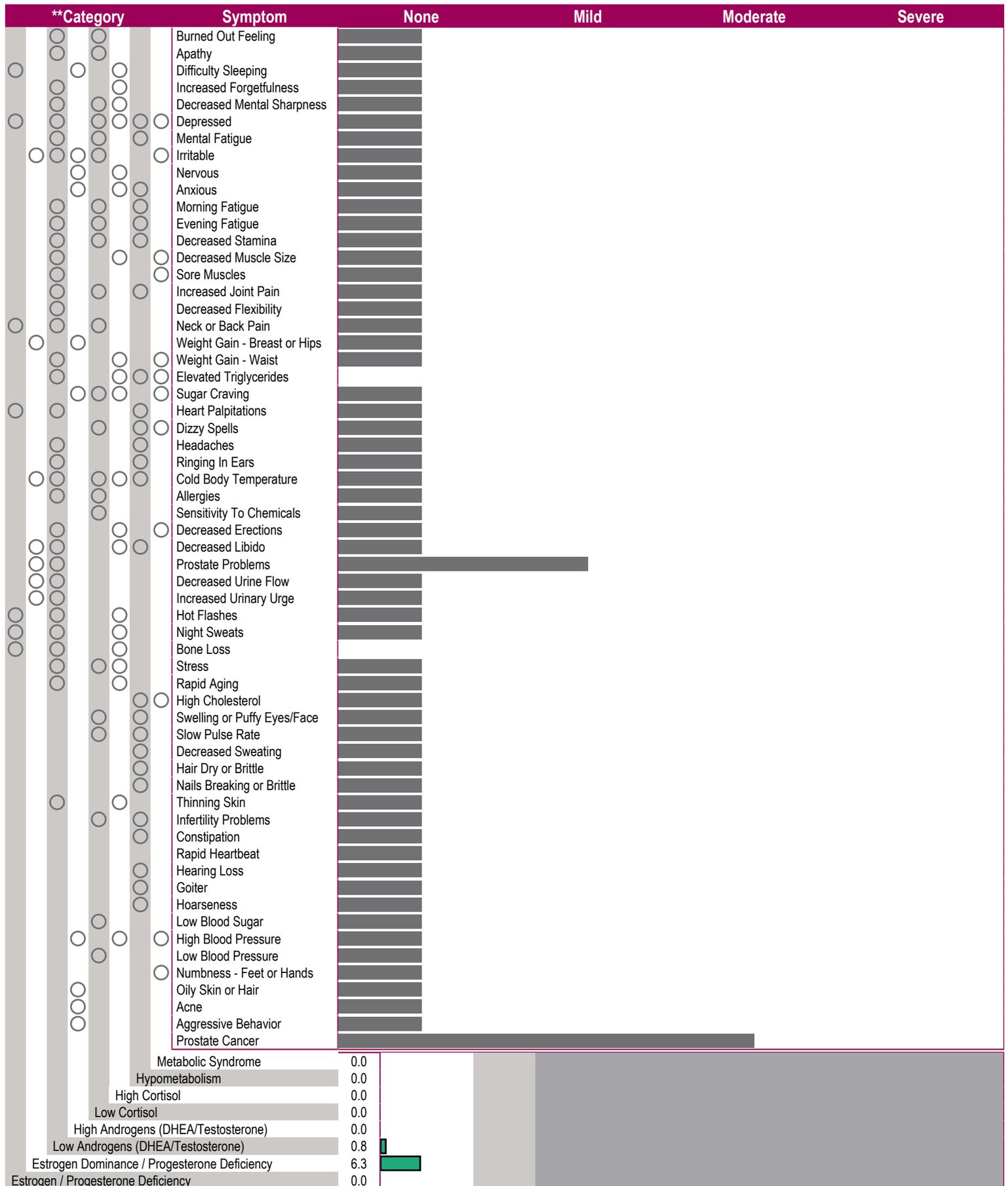
Urinary Creatinine (mg/mL)

Creatinine (pooled) (Urine)	0.76	0.3-2.0
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<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.

Tests ordered: 1504





**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**PARENT ESTROGENS (ESTRADIOL-E2, ESTRONE-E1, ESTRIOL-E3)**

The parent estrogens are within or near the limits of the reference ranges seen in healthy men and symptoms of estrogen imbalance (excess or deficiency) are self-reported as minimal.

ANDROGEN PRECURSORS (ANDROSTENEDIOL, DHEA)

Androstenedione and DHEA(S) are higher than reference ranges. High levels of these androgen precursors usually result from either higher endogenous production by the adrenal glands (often a result of adrenal glandulars), or exogenous supplementation with DHEA.

DHEA is synthesized in the adrenal glands and is rapidly sulfated to DHEA-sulfate (DHEAS) to extend its half-life in blood. DHEA(S) is converted to androstenedione and then to testosterone and Epi-testosterone in near equal amounts in most individuals, or into estrone. More conversion to estrone occurs in individuals with higher amounts of adipose (fat) tissue, which contains high levels of aromatase.

Oral DHEA is commonly used as a supplement to raise testosterone levels. As an androstenedione precursor, DHEA supplementation will also raise androstenedione levels. Oral DHEA supplementation, like oral progesterone, will result in a disproportionate increase in urinary DHEA to levels often higher than reference ranges. Most of the orally supplemented DHEA is converted to inert metabolites (e.g. DHEA sulfate) that circulate in the bloodstream where they are taken up by tissues and converted into more active androgens (testosterone and DHT) excreted into urine.

ANDROGENS AND METABOLITES

Testosterone (T) is higher than the reference ranges for males, as are its up-stream precursors, DHEA and androstenedione. DHT, the more potent down-stream metabolite of T is within normal range, indicating low 5-alpha reductase activity, which is the intracellular enzyme that converts T to DHT. In contrast to high T, Epi-T is within normal reference range. A high T, relative to Epi-T, suggests exogenous androgen therapy (e.g. testosterone or DHEA).

T, Epi-T, and DHT are normally higher in healthy young males following puberty, and levels of these androgens progressively drop with age. While androgens naturally decline with age, they can drop more rapidly when the body is exposed to stressors (psychological, physical, surgical, pathogens), excessive estrogens, and some medications.

The most potent of the androgens is dihydrotestosterone (DHT), which is created from testosterone via the enzyme 5a reductase. DHEA or T therapy can sometimes lead to excessive levels of either DHT or estradiol, both down-stream metabolites of T via the enzymes 5-alpha reductase and aromatase, respectively. These enzymes are higher in tissues and organs such as the skin, seminal vesicles, prostate, and other organs such as the brain. Endogenous testosterone is derived mostly from androstenedione and DHEA. In men most of the testosterone is produced in the testes and a much smaller portion is derived from androstenedione in the adrenal glands.

Testosterone, and particularly its more potent down-stream metabolite DHT, are important anabolic hormones that help to maintain both physical and mental health. They help prevent fatigue, help to maintain a normal sex drive, increase the strength of all structural tissues (skin, bone, muscles, heart) and prevent depression and mental fatigue. Testosterone deficiency, particularly when coupled with high estrogens, is more commonly associated with symptoms such as decreased sex drive, memory lapses, grumpiness, thinning skin, weight gain in the hips and thighs (mostly from high estrogens) and loss of muscle and bone mass. Estrogens in excess can block the beneficial effects of T and DHT.

DHT is the most potent of the androgen metabolites and forms from 5-alpha reductase conversion of T. DHT is formed within cells of target tissues such as the skin and prostate, where it binds to androgen receptors and activates androgen-specific genes. Excessive levels of DHT can result from overexpression of 5-alpha reductase in the skin as well as excessive T-therapy. High DHT, especially formed in the skin from topical T therapy, can cause conditions such as acne and heavier growth of hair on the face and body, but loss of hair on the scalp. In the brain excessive conversion of T to DHT and estradiol may cause agitation and aggressive behavior.

Epi-testosterone (Epi-T) and testosterone (T) are created in about equal amounts from androstenedione. The ratio of T/Epi-T is about 1 under normal circumstances, however this ratio can be much lower in men and women of Asian descent due to deletion polymorphisms in testosterone glucuronidation, which results in less testosterone excreted in urine, but normal levels in serum (Jakobsson J J Clin Endocrinol Metab 91: 687-693, 2006; Strahm E. Br J Sports Med 43: 1126-1130, 2009). Exogenous T therapy usually results in an increase in urinary T, but not Epi-T, the latter of which reflects endogenous T production only. A T/Epi-T ratio of > 6 is nearly always associated with use of exogenous T, and is used by the Olympic Committee to screen athletes for anabolic steroid use.