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TEST PATIENT

GUa d'Y'HYghBUa 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

Basic Hormones, 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 11/20/18	RANGE
Urinary Estrogens		
Estradiol	0.18 L	0.78-1.79 µg/g Cr Premeno-luteal or ERT
Estrone	0.60 L	2.27-5.22 µg/g Cr Premeno-luteal or ERT
Estriol	0.45 L	0.78-1.98 µg/g Cr Premeno-luteal or ERT
E3/(E1+E2)	0.59	>0.3 (> median value)
Urinary Progestogens		
Pregnanediol	41 L	465-1609 µg/g Cr Premeno-luteal or PgRT
Allopregnanolone	0.18 L	2.23-14.87 µg/g Cr Premeno-luteal or PgRT
Pgdiol/E2	247.06 L	1000-1500 (Optimal Luteal Only)
Urinary Androgens		
DHEA	14.86 L	15.82-129.17 µg/g Cr Premeno-luteal or DHEAT
Androstenedione	3.12 L	3.93-13.53 µg/g Cr Premeno-luteal or ART
Testosterone	2.29	1.22-3.97 µg/g Cr Premeno-luteal or ART
Epi-Testosterone	1.23	0.39-1.32 µg/g Cr Postmenopausal
T/Epi-T	1.86	0.5-3.0
5α-DHT	0.93	0.28-1.52 µg/g Cr Premeno-luteal or ART
Urinary Glucocorticoids		
Total Cortisol	29.57	13.23-39.26 µg/g Cr Postmenopausal
Urinary Creatinine		
Creatinine (pooled)	1.17	0.3-2.0 mg/mL

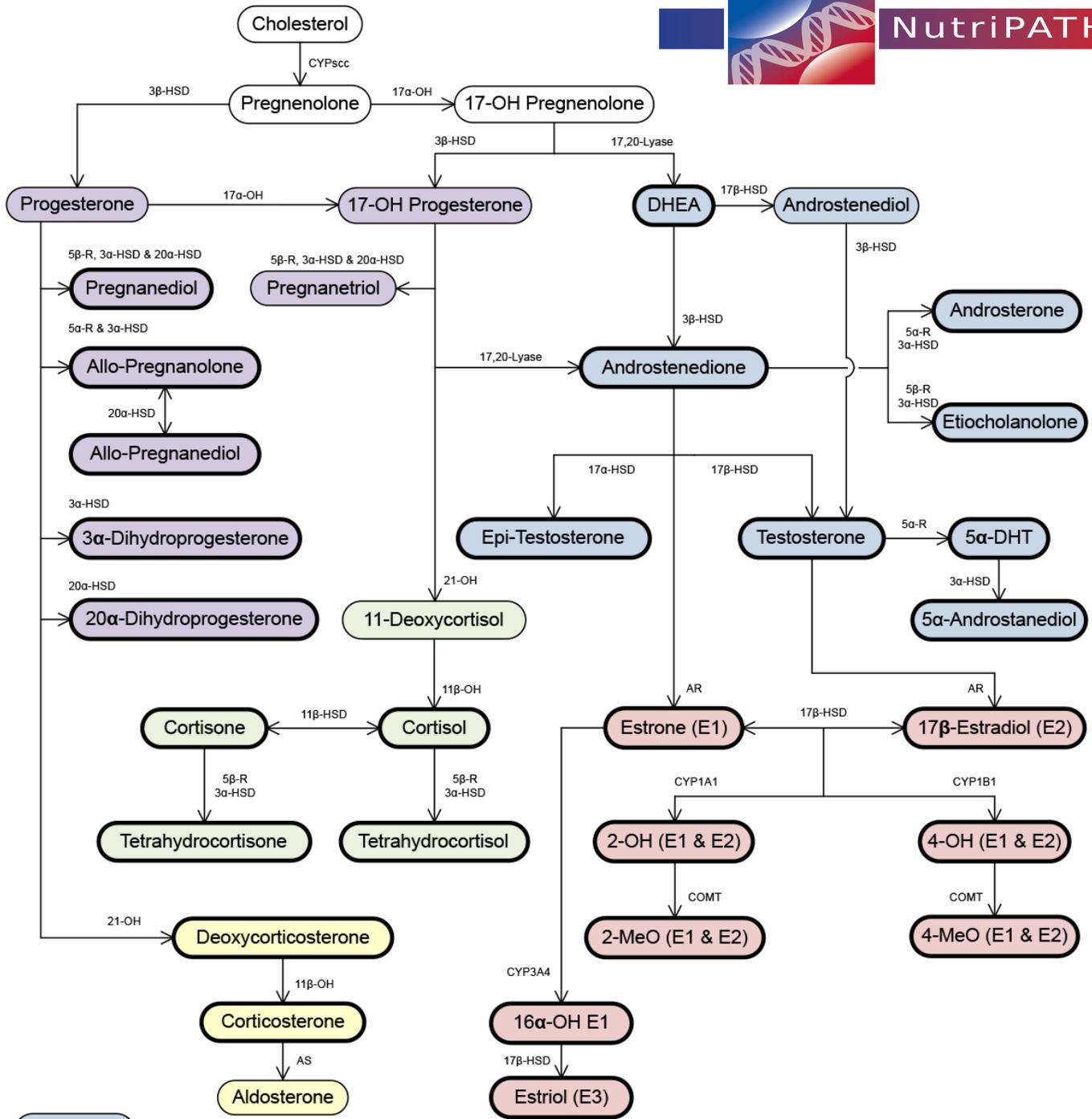


TEST NAME	RESULTS 11/20/18	RANGE
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<dL = Less than the detectable limit of the lab. N/A = Not applicable; 1 or more values used in this calculation is less than the detectable limit. H = High. L = Low.

Therapies

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 CATEGORIES	RESULTS 11/20/18	
Estrogen / Progesterone Deficiency	57%	<div style="width: 57%;"></div>
Estrogen Dominance / Progesterone Deficiency	26%	<div style="width: 26%;"></div>
Low Androgens (DHEA/Testosterone)	73%	<div style="width: 73%;"></div>
High Androgens (DHEA/Testosterone)	28%	<div style="width: 28%;"></div>
Low Cortisol	33%	<div style="width: 33%;"></div>
High Cortisol	50%	<div style="width: 50%;"></div>
Hypometabolism	30%	<div style="width: 30%;"></div>
Metabolic Syndrome	33%	<div style="width: 33%;"></div>

SYMPTOM CHECKLIST	MILD	MODERATE	SEVERE
Aches and Pains	<div style="width: 100%;"></div>		
Acne	<div style="width: 5%;"></div>		
Allergies	<div style="width: 100%;"></div>		
Anxious	<div style="width: 5%;"></div>		
Bleeding Changes	<div style="width: 5%;"></div>		
Blood Pressure High	<div style="width: 5%;"></div>		
Blood Pressure Low	<div style="width: 5%;"></div>		
Blood Sugar Low	<div style="width: 5%;"></div>		
Body Temperature Cold	<div style="width: 5%;"></div>		
Bone Loss	<div style="width: 100%;"></div>		
Breast Cancer	<div style="width: 5%;"></div>		
Breasts - Fibrocystic	<div style="width: 5%;"></div>		
Breasts - Tender	<div style="width: 5%;"></div>		
Chemical Sensitivity	<div style="width: 5%;"></div>		
Cholesterol High	<div style="width: 100%;"></div>		
Constipation	<div style="width: 100%;"></div>		
Depressed	<div style="width: 100%;"></div>		
Fatigue - Evening	<div style="width: 100%;"></div>		
Fatigue - Morning	<div style="width: 100%;"></div>		
Fibromyalgia	<div style="width: 5%;"></div>		
Foggy Thinking	<div style="width: 100%;"></div>		
Goiter	<div style="width: 5%;"></div>		
Hair - Dry or Brittle	<div style="width: 100%;"></div>		
Hair - Increased Facial or Body	<div style="width: 100%;"></div>		
Hair - Scalp Loss	<div style="width: 5%;"></div>		
Headaches	<div style="width: 5%;"></div>		
Hearing Loss	<div style="width: 100%;"></div>		
Heart Palpitations	<div style="width: 5%;"></div>		
Hoarseness	<div style="width: 5%;"></div>		
Hot Flashes	<div style="width: 100%;"></div>		
Incontinence	<div style="width: 5%;"></div>		
Infertility	<div style="width: 5%;"></div>		
Irritable	<div style="width: 100%;"></div>		
Libido Decreased	<div style="width: 100%;"></div>		
Memory Lapse	<div style="width: 100%;"></div>		
Mood Swings	<div style="width: 100%;"></div>		
Muscle Size Decreased	<div style="width: 100%;"></div>		
Nails Breaking or Brittle	<div style="width: 100%;"></div>		
Nervous	<div style="width: 5%;"></div>		
Night Sweats	<div style="width: 100%;"></div>		
Numbness - Feet or Hands	<div style="width: 5%;"></div>		

SYMPTOM CHECKLIST	MILD	MODERATE	SEVERE
Pulse Rate Slow	█		
Rapid Aging	██████████		
Rapid Heartbeat	█		
Skin Thinning	██████████	██████████	██████████
Sleep Disturbed	██████████	██████████	
Stamina Decreased	██████████	██████████	██████████
Stress	██████████	██████████	██████████
Sugar Cravings	██████████	██████████	██████████
Sweating Decreased	█		
Swelling or Puffy Eyes/Face	██████████	██████████	██████████
Tearful	█		
Triglycerides Elevated	█		
Urinary Urge Increased	█		
Uterine Fibroids	BLANK		
Vaginal Dryness	██████████	██████████	██████████
Water Retention	██████████	██████████	██████████
Weight Gain - Hips	██████████	██████████	██████████
Weight Gain - Waist	██████████	██████████	██████████

Lab Comments

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

The parent estrogens E2 and E1 are low or lower than the median of the expected reference ranges seen in postmenopausal women supplementing with most forms of estrogen replacement therapy (e.g. estrogen patch, oral estrogens) other than topical estrogens. Topical estrogens increase urinary levels of estrogens very little, but result in a much higher dose-associated increase in salivary and capillary blood levels of supplemented estrogens. Topical estrogens do, however, increase urinary estrogens slightly from postmenopausal baseline levels to low premenopausal levels. The 20-80 percentile range for topical ERT is 0.40-1.51 for E2, 1.14-3.86 for E1, and 0.35-1.53 for E3. Very little of the topically delivered estrogens enter urine and are more likely to be excreted in bile/feces. A more accurate way of evaluating the bioavailability of these topically delivered estrogens is by testing saliva or capillary blood.

If symptoms of estrogen deficiency are NOT problematic with this form of estrogen delivery then dosage is likely adequate, or excessive, if symptoms indicate estrogen dominance. However, if estrogen deficiency symptoms persist despite physiological (< 0.1 mg) or pharmacological (> 0.1 mg) daily dosing, as seen in these results, this is more likely due to excessive levels of estrogens in tissues, rather than a deficiency. Because pharmacological levels of topical estrogens are used (> 0.1 mg) it is possible that vasomotor symptoms are more likely caused by too much, rather than too little estrogen. However, an alternative explanation is that the topical estrogen was poorly absorbed. If salivary or capillary blood estradiol is low, this would confirm poor absorption; however, if elevated, this would indicate higher tissue levels of estrogens that are precipitating paradoxical estrogen deficiency symptoms (e.g. hot flashes and night sweats). Because symptoms of estrogen deficiency are significantly problematic (most moderate to severe) despite a more pharmacological topical ERT, it would be worthwhile to test saliva and/or capillary blood for estradiol, and if high to consider a different route of estrogen administration (e.g. transdermal patch) that provides a lower and steadier delivery of estrogen.

PROGESTERONE METABOLITES (PREGNANEDIOL-PgDiol, ALLOPREGNANOLONE-AlloP)

The urinary levels of the progesterone metabolites pregnanediol (PgDiol) and allopregnanolone (AlloP), a neuroactive steroid, are lower than ranges expected with topical progesterone supplementation. NutriPATH's urinary PgDiol reference range for topical progesterone (10-30 mg dosing) in postmenopausal women is 85-403 ug/g creatinine, which is not much higher than the urinary PgDiol level seen in premenopausal women during the follicular phase of the menstrual cycle (92-346 ug/mg Cr).

It is important to recognized that topical progesterone raises urinary and serum levels of PgDiol very little. In sharp contrast, topical progesterone results in a precipitous increase in saliva and capillary blood (DBS) progesterone. While topical progesterone does raise the level of urinary PgDiol slightly above the baseline level seen in premenopausal women during the early follicular phase of the menstrual cycle, it does NOT raise the level of PgDiol to levels seen during the luteal phase of the menstrual cycle.

The conversion of progesterone to AlloP is important as this is a neuroactive steroid that freely enters the brain from the bloodstream, where it binds to GABA receptors and contributes to progesterone's well recognized calming (anxiolytic) and sleep inducing effects. Insufficient metabolism of progesterone to AlloP can have a paradoxical anxiogenic effect, increasing symptoms such as anxiety and premenstrual dysphoric disorder (PMDD) and premenstrual syndrome (PMS). Only high levels of AlloP, achieved at peak of an optimal luteal phase, during pregnancy, and with progesterone therapy, have the anxiolytic effects on GABAa receptors in the brain.

Because urine PgDiol is not reflective of topical progesterone delivery to tissues, salivary or capillary blood may be a better means to assess the effects of topically applied progesterone [Du..Zava, et.al. Menopause 20(11): 1169-1175, 2013; O'Leary et.al. Clin Endocrinol 53: 615-620, 2000)]. It is also important to note that the ratio of PgDiol/E2, derived from endogenous PgDiol and estradiol metabolites, is not applicable for exogenously delivered progesterone, topical or oral.

ANDROGEN PRECURSORS (ANDROSTENEDIOL, DHEA)

The androgen precursors, DHEA and its down-stream metabolite androstenedione (A4), are lower than normal reference ranges for a postmenopausal woman supplementing with topical DHEA.

DHEA was delivered in a topical cream/gel, which results in direct uptake into the bloodstream, circumventing the liver. This results in less DHEA metabolism to the sulfated form, DHEAS, and less excretion by the kidneys into urine. While DHEA and A4 may not increase much in urine with topical DHEA therapy, levels of these hormones increase significantly in saliva and capillary blood (measured by finger stick Dried Blood Spot testing), which is more representative of the hormones delivered to tissues, as opposed to those that are excreted as metabolites in urine. DHEA in the systemic circulation can be taken up by target tissues and converted directly into A4, testosterone, and DHT. Thus, the low urinary DHEA value seen in these test results may not accurately reflect how much DHEA is getting to target tissues and converting to down-stream androgens such as A4, testosterone, and DHT. If these urinary androgens are within normal range and urinary DHEA is low, this suggests that the DHEA effectively converted to these androgens systemically.

Low urinary DHEA, despite topical DHEA therapy, indicates that the baseline endogenous level of DHEA is low, suggesting adrenal fatigue. Low DHEA is often associated with symptoms of androgen deficiency (fatigue, depression, low libido, loss of muscle mass, bone loss, memory lapses). If the topical DHEA therapy is associated with normal (premenopausal) levels of down-stream androgens such as T and DHT, and urinary DHEA levels remain low, this likely indicates the DHEA is converting to these androgens systemically in target tissues. Consider other methods of DHEA delivery (e.g. oral) if low androgen symptoms remain problematic.

ANDROGENS (Testosterone-T, Epi-Testosterone-EpiT, 5-alpha-dihydrotestosterone-DHT)

Testosterone (T), Epi-testosterone (Epi-T), and 5-alpha DHT, are within low-normal expected ranges for a postmenopausal woman supplementing with testosterone, which is within the range for a premenopausal woman. Despite T therapy symptoms are suggestive of low androgens. Topical testosterone as increases urinary T and its downstream metabolite DHT, but has no effect on Epi-T levels, usually resulting in a higher (> 3) T/Epi-T ratio. T has remained in the lower range with T therapy, and the ratio of T/Epi-T is < 3.

Lower tissue levels of the proximate androgen DHT and/or lower androgen receptors in target tissues may result from excessive levels of other supplemented hormones that lower T to DHT conversion and tissue levels of androgen receptors. For example, progesterone in excess competitively inhibits conversion of T to DHT by blocking the actions of 5-alpha reductase, the enzyme that converts T to DHT, and progesterone to 5-alpha dihydroprogesterone. In addition, high estrogens from therapy down regulate cellular androgen receptors, resulting in low tissue response to T or DHT. High cortisol also has anti-androgenic actions by down-regulating cellular androgen receptors, suppressing androgen (testosterone) synthesis, and decreasing testosterone by increasing its conversion to estrogens via aromatase (induced by high cortisol in adipose tissue). Low androgen symptoms also overlap with low thyroid symptoms, which are also self-reported as problematic.

If estrogen and progesterone therapies are used in concert with testosterone, and estrogens (particularly estradiol) and/or progesterone metabolites (pregnanediol and allopregnanolone) are high, consider reducing the dosing to create a better balance. If cortisol is elevated, and symptoms of high cortisol are problematic also consider means to reduce stressors that raise cortisol. Consider that high or low cortisol, or low thyroid hormones may also contribute to the symptom profile seen in this individual, and therapeutic interventions may be needed to correct their imbalances.

TOTAL CORTISOL (F)

Total cortisol (F) is within the expected reference range; however, symptoms suggest adrenal dysfunction (see suggestions below for 4x circadian rhythm testing in four urine samples submitted).

The total level of F is determined from the average of four urine collections throughout the day and is equivalent to a 24 hour urine value. While 24 hr and 4-spot total cortisol urine tests provide useful information about the adrenal glands average capacity to synthesize cortisol and downstream metabolites in a day, they provide no information about the diurnal synthesis of cortisol throughout the day. In healthy individuals cortisol 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 symptoms typical of adrenal dysfunction, 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 in the four urine samples used here to determine total cortisol.

Adrenal dysfunction can include many of the self-reported signs/symptoms such as anxiety, nervous-irritability, self-perceived stress, excessive fatigue, and sleep disturbances. Chronic abnormal cortisol levels (both high and low, or not showing a normal circadian rhythm) might also include memory problems, depression, loss of muscle mass, and weight gain in the waist. High cortisol is closely associated with insulin resistance and metabolic syndrome, 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. Excessive chronic high cortisol lowers synthesis of testosterone, growth hormone, thyroid hormones and their tissue receptors.

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; "The Role of Stress and the HPA Axis in Chronic Disease Management" by Thomas Guilliams, PhD.