



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'' \$\$\$

P: 1300 688 522
 E: info@nutripath.com.au
 A: PO Box 44Z Ashburton VIC 3142

SPECIMEN RECEPTION

URINE, SPOT
SPECIMEN RECEPTION COMMENTS Result Range Units
 Please Note: This is a day 21 specimen.

MICRO SAMPLE ASSAYS

DRIED URINE	Result	Range	Units	
Female Profile, Basic (Dry Urine)				
Estradiol (E2), Dried Urine	1.38	0.78 - 1.96	ug/gCR	
Estrone (E1), Dried Urine	4.05	2.37 - 5.48	ug/gCR	
Estriol (E3), Dried Urine	0.04 *L	0.76 - 2.13	ug/gCR	
PREGNANEDIOL, Dried Urine	403.0 *L	579.0 - 1710.0	ug/gCR	
CORTISOL, Dried Urine	39.97 *H	11.83 - 31.10	ug/gCR	
DHEA, Dried Urine	222.61 *H	16.90 - 54.67	ug/gCR	
ANDROSTENEDIONE, Dried Urine	11.58 *H	3.75 - 11.55	ug/gCR	
TESTOSTERONE, Dried Urine	2.81	0.71 - 3.75	ug/gCR	

(*) Result outside normal reference range

(H) Result is above upper limit of reference rang (L) Result is below lower limit of reference range



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Dried Urine Hormone Reference Ranges

DRIED URINE HORMONE REFERENCE RANGES

Hormone	Pre-Menop/Luteal	Post Menop
Estradiol (E2)	0.78 - 1.96	0.13 - 0.78
Estrone (E1)	2.37 - 5.48	0.59 - 2.70
Estriol (E3)	0.76 - 2.13	0.25 - 1.15
E3 / (E2+E1)	> 0.25	> 0.25
2OH Estradiol	0.15 - 0.82	0.07 - 0.30
2OH Estrone	0.81 - 2.90	0.21 - 1.11
4OH Estradiol	0.11 - 0.24	0.03 - 0.11
4OH Estrone	0.17 - 0.49	0.05 - 0.23
16OH Estrone	0.31 - 1.02	0.08 - 0.42
2OH (E1+E2) / 16OHE1	1.81 - 5.21	1.77 - 13.42
2MeOH Estradiol	0.03 - 0.09	0.02 - 0.05
2MeOH Estrone	0.25 - 0.71	0.06 - 0.27
2MeOHE1 / 2OHE1	0.21 - 0.38	0.19 - 0.36
4MeOH Estradiol	0.02 - 0.04	0.01 - 0.04
4MeOH Estrone	0.01 - 0.03	0.01 - 0.03
4MeOHE1 / 4OHE1	0.05 - 0.13	0.03 - 0.38
4MeOHE2 / 4OHE2	0.10 - 0.29	0.14 - 0.73
Pregnanediol	579 - 1710	43 - 168
Pregnanediol/Estradiol	1000 - 5000	(Optimal Luteal phase only)
Allopregnanolone	2.81 - 16.36	0.22 - 1.08
Allopregnanediol	16.64 - 72.85	0.82 - 4.16
3a-Dihydroprogesterone	0.77 - 1.91	0.13 - 0.56
20a-Dihydroprogesterone	3.09 - 9.30	0.49 - 2.21
Deoxycorticosterone	0.63 - 1.52	0.32 - 1.45
Corticosterone	3.27 - 7.88	1.58 - 6.75
Testosterone	0.71 - 3.75	0.55 - 2.34
Epi-Testosterone	2.21 - 5.12	0.30 - 0.90
Testo/Epi-Testo	0.5 - 3.0	0.5 - 3.0
5a-DiHydroTestosterone (DHT)	0.20 - 1.29	0.18 - 0.72
Androstenedione	3.75 - 11.55	1.98 - 4.75
DHEA	16.90 - 54.67	7.85 - 28.80

Hormone	Pre-Menop	Post Menop
Total Cortisol	11.83 - 31.10	10.22 - 23.92
Total Cortisone	23.70 - 48.92	18.80 - 39.46
Cortisol/Cortisone	0.5 - 0.7	0.5 - 0.7
Tetrahydrocortisol	254 - 733	232 - 518
Tetrahydrocortisone	421 - 1240	421 - 1043

	Free Cortisol	Free Cortisone	Melatonin
1st Morn:	8.50 - 30.5	33.5 - 93.9	18.0 - 40.9
2nd Morn:	20.7 - 56.9	58.8 - 140	7.3 - 31.9
Evening:	7.1 - 17.5	36.9 - 82.1	0.7 - 2.2
Night:	3.1 - 9.0	18.4 - 47.7	1.7 - 11.1



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Dried Urine Hormone Comments

Lab Comments

ESTROGENS (ESTRADIOL-E2, ESTRONE-E1, ESTRIDIOL-E3)

The parent estrogens, estradiol (E2) and estrone (E1) are within/near the expected median to 90 percentile reference ranges seen in postmenopausal women supplementing with topical estradiol replacement therapy. Estriol was not used as a form of ERT and the level is within reference range for a postmenopausal woman. Topically delivered estrogens increase saliva and capillary blood levels of the supplemented estrogens proportionately more than in urine and serum levels. Topically delivered hormones are more likely to be excreted in bile/feces than in urine, which accounts for the minimal increase in urinary estrogens seen with topically delivered hormones.

PROGESTERONE METABOLITES

The progesterone metabolite, pregnanediol (PgDiol), is within the expected reference range for a postmenopausal woman (or premenopausal woman in follicular phase or with luteal phase insufficiency) supplementing with topical progesterone (85-403 ug/g creatinine). Because of its non-polar nature, very little topically delivered progesterone or its down-stream metabolite (PgDiol) is excreted into urine. Some progesterone may be seen in urine with vaginal progesterone supplementation, but this represents direct contamination. Very little of the Pg metabolite (PgDiol) is seen in urine with vaginal Pg delivery.

This individual has indicated supplementation with topical progesterone, which results in an increase in PgDiol to levels slightly higher than seen in postmenopausal women, but lower than levels seen in premenopausal woman during the luteal phase of the menstrual cycle (range: 849-1932 ug/g Cr).. The 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).

While topical progesterone supplementation raises urinary PgDiol very little, it results in high levels of progesterone in saliva, capillary blood, and target tissues (e.g. breasts and uterus). Therefore, salivary or capillary blood may be a better means to assess tissue delivery of TOPICALLY applied progesterone since urine PgDiol is poorly reflective of tissue progesterone distribution.

Important note: the ratio of PgDiol/E2, derived from endogenous PgDiol and estradiol metabolites, is not applicable for exogenously delivered progesterone, topical or oral.

ANDROGEN PRECURSORS (DHEA, Androstenedione)

DHEA is much higher than the reference range for either premenopausal or postmenopausal women. An exceptionally high level of urinary DHEA is consistent with DHEA supplementation. High levels of DHEA and down-stream metabolites, which include androstenedione, testosterone, and 5-alpha DHT, are common with oral DHEA therapy.

Oral and sublingual DHEA are commonly used as a supplement to raise testosterone



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levels in women. DHEA supplementation also raises androstenedione levels, which is likely why androstenedione is also elevated in this individual. Oral DHEA supplementation, like oral progesterone, will result in a disproportionate increase in urinary DHEA to levels higher than reference ranges, but much less increase in salivary or capillary blood levels of DHEA or DHEA-sulfate (DHEAS). 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), or excreted into urine.

ANDROGENS AND METABOLITES (TESTOSTERONE, EPI-TESTOSTERONE, AND 5-ALPHA-DIHYDROTESTOSTERONE)

Testosterone (T) is slightly higher than the expected reference range for a postmenopausal woman, and is > 3x higher than its epimer, Epi-Testosterone (Epi-T), resulting in a high T/Epi-T ratio. A high ratio of T/Epi-T indicates exogenous supplementation with T, or a T precursor (e.g. DHEA, androstenediol, pregnenolone-note patient self-reports supplementation with 100 mg sublingual DHEA). Exogenous supplementation with T, or T precursors (i.e. DHEA), raise the level of T, but not of Epi-T, resulting in a high T/Epi-T ratio.

The more potent down stream metabolite of T, 5-alpha dihydrotestosterone (DHT), is also slightly elevated. Despite the higher androgens, apparently derived from exogenous DHEA therapy, symptoms characteristic of prolonged androgen excess are NOT self-reported as problematic. When progesterone is high as a result of supplementation, it can competitively inhibit 5 alpha reductase mediated conversion of T to DHT. This individual is also supplementing with progesterone, which may account for the lack of high androgen symptoms. When progesterone is high, relative to testosterone, this can result in more conversion of progesterone to 5-alpha-progesterone metabolites (e.g. allopregnanolone and allopregnanediol), than to DHT; however these metabolites also are NOT elevated.

Androgens, particularly the most potent of them, DHT, play an important role in maintaining the integrity of structural tissues such as skin, connective tissues, bone, and muscles. Androgens also play an important role in the brain to increase the level of neurotransmitters such as dopamine, which are important for mood elevation and sex drive. Testosterone, while it is a precursor to DHT, is also a precursor to the estrogens, estradiol and estrone. Recent studies have shown that testosterone, through its conversion to DHT, protects against breast cancer caused by excessive conversion of T to estrogens via the enzyme aromatase (Glaser RL, Maturitas 76: 342-349, 2013).

DHT is the most potent of the androgen metabolites and is formed directly within cells of target tissues such as the skin (pilosebaceous gland) where it binds to androgen receptors and activates androgen-specific genes (e.g. DHT formed in the skin causes skin thickening and stimulates hair growth on the body and face, but more rapid loss of hair on the scalp when 5-alpha reductase is high). Excessive levels of DHT, resulting from genetic polymorphisms that



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overexpress 5-alpha reductase in the skin, can cause conditions such acne and heavier growth of hair on the face and body, but loss of hair on the scalp.

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.