P:1300 688 522
E:info@nutripath.com.au
A:PO Box 442 Ashburton VIC3142

Please refer to PDF report attached

Practitioner:	RACHEAL LEE (NPINS)
	SHOP 6/115 SHINGLEY DRIVE
	AIRLIE BEACH QLD
	QLD
	4802
Request id:	4080668
Patient:	SHAINA BERRY
	51 LITFINS ROAD
	MOUNT SYLVIA QLD
	QLD
	4343
Date of Birth:	07-Nov-1997
Sex:	F

TEST REPORT

 # 2025 03 17 1053 U

Ordering Provider:
NutriPath

Samples Received
03/17/2025

Report Date
03/22/2025

Samples Collected
Urine - 03/05/25 06:30
Urine - 03/05/25 09:50
Urine - 03/05/25 16:55
Urine - 03/05/25 20:40

Patient Name: Shaina Berry
Patient Phone Number:

Gender	Last Menses	Height	Waist	Basal Body Temperature
Female	02/06/2025	174 cm	73 cm	36.5°
DOB	**Menses Status**	**Weight**	**BMI**	
11/7/1997 (27 yrs)	Pre-Menopausal - Irregular	77 kg	25.4	

TEST NAME	RESULTS | 03/05/25	RANGE
Urinary Estrogens		
Estradiol	0.41 L	0.78-1.79 µg/g Cr Premeno-luteal or ERT
Estrone	1.85 L	2.27-5.22 µg/g Cr Premeno-luteal or ERT
Estriol	0.87	0.78-1.98 µg/g Cr Premeno-luteal or ERT
E3/(E1+E2)	0.38	>0.3 (> median value)
2-OH Estradiol	0.41	0.17-0.70 µg/g Cr Premeno-luteal or ERT
2-OH Estrone	2.02	0.70-2.54 µg/g Cr Premeno-luteal or ERT
4-OH Estradiol	0.06 L	0.10-0.18 µg/g Cr Premeno-luteal or ERT
4-OH Estrone	0.24	0.17-0.47 µg/g Cr Premeno-luteal or ERT
16α-OH Estrone	0.30 L	0.35-1.07 µg/g Cr Premeno-luteal or ERT
2-OH (E1 + E2)/16-α-OH E1	8.1 H	1.29-5.49 Premeno-luteal or ERT
2-MeO Estradiol	0.05	0.03-0.08 µg/g Cr Premeno-luteal or ERT
2-MeO Estrone	0.69 H	0.26-0.68 µg/g Cr Premeno-luteal or ERT
2-MeO E1/2-OH E1	0.34	0.21-0.38 Premeno-luteal or ERT
4-MeO Estradiol	0.04	<0.04 µg/g Cr
4-MeO Estrone	0.02	<0.04 µg/g Cr
4-MeO E1/4-OH E1	0.08	0.05-0.13 Premeno-luteal or ERT
4-MeO E2/4-OH E2	0.67 H	0.10-0.29 Premeno-luteal or ERT
Bisphenol A	<dl L	1.11-3.74 µg/g Cr Premeno-luteal

TEST NAME	RESULTS | 03/05/25	RANGE
Urinary Progestogens		
Pregnanediol	468	465-1609 µg/g Cr Premeno-luteal or PgRT
Allopregnanolone	11.07	2.23-14.87 µg/g Cr Premeno-luteal or PgRT
Allopregnanediol	50.41	14.65-76.71 µg/g Cr Premeno-luteal or PgRT
3α-Dihydroprogesterone	2.75 H	0.67-2.03 µg/g Cr Premeno-luteal or PgRT
20α-Dihydroprogesterone	3.36 L	3.93-11.62 µg/g Cr Premeno-luteal or PgRT
Deoxycorticosterone	0.66 L	0.69-2.23 µg/g Cr Premeno-luteal or PgRT
Corticosterone	2.43 L	3.19-9.59 µg/g Cr Premeno-luteal or PgRT
Pgdiol/E2	1141.46	1000-1500 (Optimal Luteal Only)
Urinary Androgens		
DHEA	9.31 L	15.82-129.17 µg/g Cr Premeno-luteal or DHEAT
Androstenedione	5.34	3.93-13.53 µg/g Cr Premeno-luteal or ART
Androsterone	747	248-937 µg/g Cr Premeno-luteal or ART
Etiocholanolone	326 L	330-960 µg/g Cr Premeno-luteal or ART
Testosterone	3.54	1.22-3.97 µg/g Cr Premeno-luteal or ART
Epi-Testosterone	5.12 H	2.01-4.66 µg/g Cr Premeno-luteal
T/Epi-T	0.69	0.5-3.0
5α-DHT	1.09	0.28-1.52 µg/g Cr Premeno-luteal or ART
5α,3α-Androstanediol	9.76	2.98-13.10 µg/g Cr Premeno-luteal or ART
Urinary Glucocorticoids		
Total Cortisol	17.04	12.26-33.12 µg/g Cr Premeno-luteal
Total Cortisone	26.25	23.27-50.88 µg/g Cr Premeno-luteal
Cortisol/Cortisone	0.65	0.5-0.7
Tetrahydrocortisol	115 L	214-546 µg/g Cr Premeno-luteal
Tetrahydrocortisone	193 L	437-1184 µg/g Cr Premeno-luteal
Urinary Free Diurnal Cortisol		
Free Cortisol	13.10	7.8-29.5 µg/g Cr (1st Morning)
Free Cortisol	20.19 L	23.4-68.9 µg/g Cr (2nd Morning)
Free Cortisol	4.41 L	6.0-19.2 µg/g Cr (Evening)

TEST NAME	RESULTS | 03/05/25	RANGE
Urinary Free Diurnal Cortisol		
Free Cortisol	1.62 L	2.6-8.4 µg/g Cr (Night)
Urinary Free Diurnal Cortisone		
Free Cortisone	68.37	31.6-91.6 µg/g Cr (1st Morning)
Free Cortisone	67.51	63.3-175.8 µg/g Cr (2nd Morning)
Free Cortisone	15.37 L	30.6-88.5 µg/g Cr (Evening)
Free Cortisone	11.34 L	15.5-44.7 µg/g Cr (Night)
Urinary Diurnal Melatonin MT6s		
Melatonin	67.38 H	18.0 - 40.9 µg/g Cr (1st Morning)
Melatonin	17.09	7.3 - 31.9 µg/g Cr (2nd Morning)
Melatonin	2.72 H	0.7 - 2.2 µg/g Cr (Evening)
Melatonin	1.94	1.7 - 11.1 µg/g Cr (Night)
Urinary Creatinine		
Creatinine (pooled)	1.29	0.3-2.0 mg/mL
Creatinine	1.15	0.3-2.0 mg/mL (1st morning)
Creatinine	0.74	0.3-2.0 mg/mL (2nd morning)
Creatinine	0.87	0.3-2.0 mg/mL (Evening)
Creatinine	0.99	0.3-2.0 mg/mL (Night)

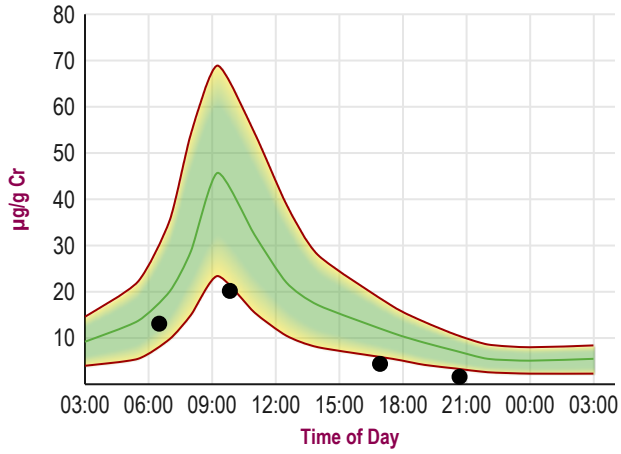
<dl *= Less than the detectable limit of the lab.* ***N/A*** *= Not applicable; 1 or more values used in this calculation is less than the detectable limit.* ***H*** *= High.* ***L*** *= Low.*

Therapies

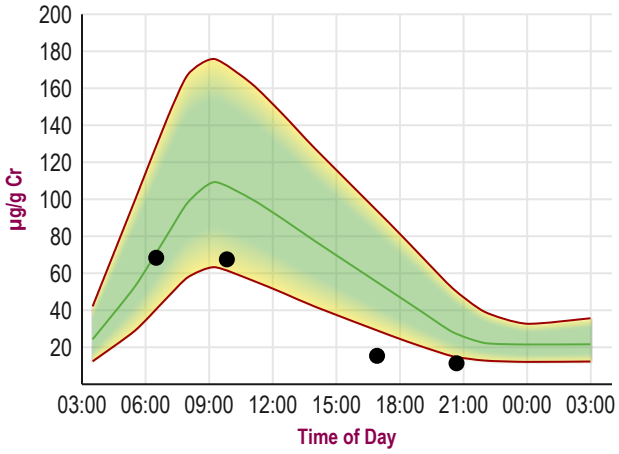
None

Graphs

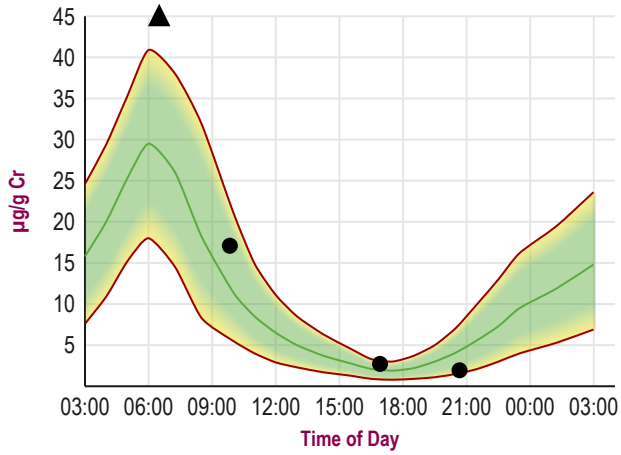
Urinary Free Cortisol



Urinary Free Cortisone



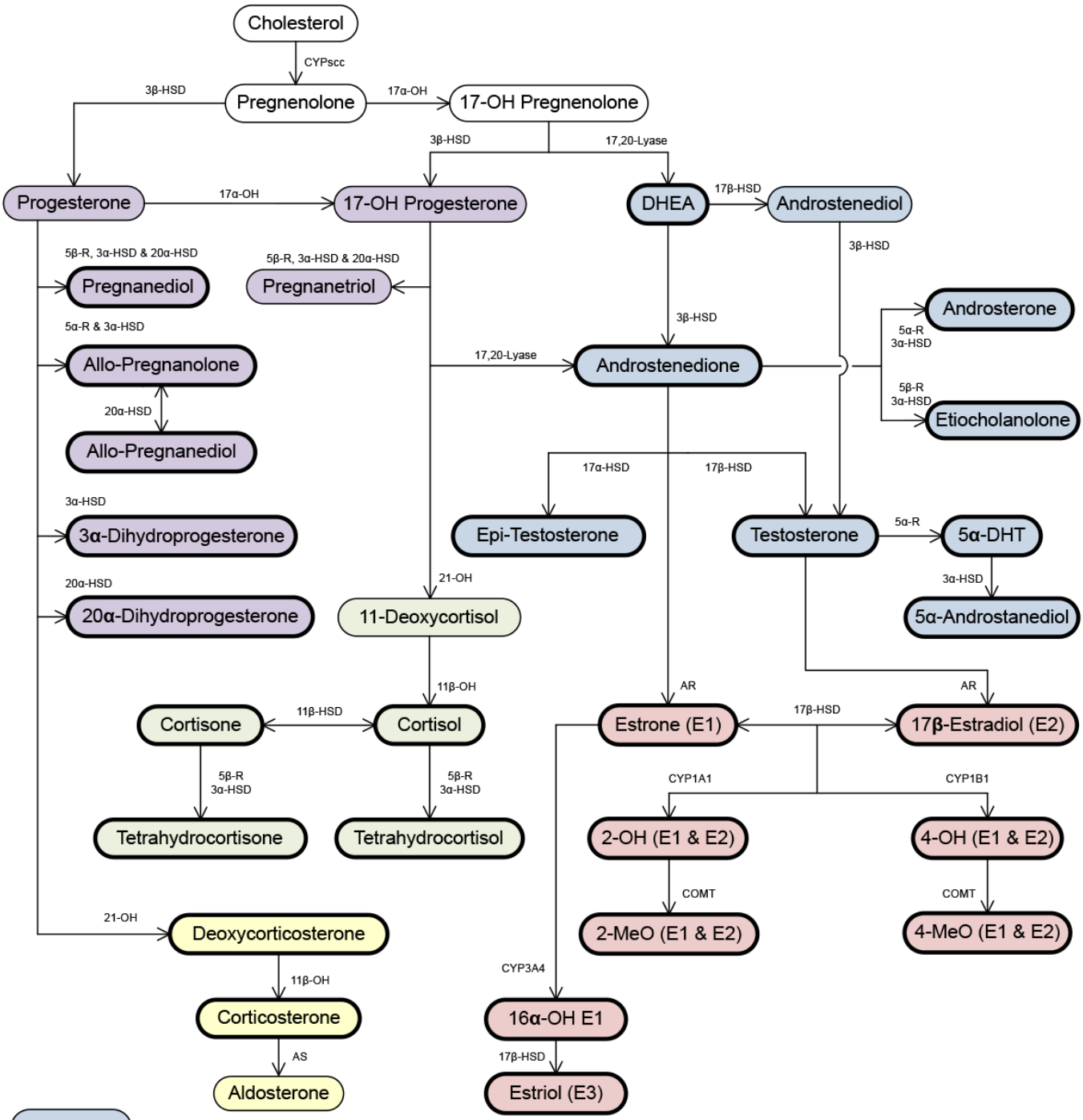
Urinary Melatonin (MT6s)



TEST NAME	WOMEN
Urinary Estrogens	
Estradiol	0.15-0.75 µg/g Cr Postmenopausal; 0.78-1.79 µg/g Cr Premeno-luteal or ERT
Estrone	0.64-2.56 µg/g Cr Postmenopausal; 2.27-5.22 µg/g Cr Premeno-luteal or ERT
Estriol	0.28-1.17 µg/g Cr Postmenopausal; 0.78-1.98 µg/g Cr Premeno-luteal or ERT
E3/(E1+E2)	>0.3 (> median value)
2-OH Estradiol	0.08-0.31 µg/g Cr Postmenopausal; 0.17-0.70 µg/g Cr Premeno-luteal or ERT
2-OH Estrone	0.25-1.00 µg/g Cr Postmenopausal; 0.70-2.54 µg/g Cr Premeno-luteal or ERT
4-OH Estradiol	0.03-0.12 µg/g Cr Postmenopausal; 0.10-0.18 µg/g Cr Premeno-luteal or ERT
4-OH Estrone	0.06-0.22 µg/g Cr Postmenopausal; 0.17-0.47 µg/g Cr Premeno-luteal or ERT
16α-OH Estrone	0.10-0.41 µg/g Cr Postmenopausal; 0.35-1.07 µg/g Cr Premeno-luteal or ERT
2-OH (E1 + E2)/16-α-OH E1	1.47-8.17 Postmenopausal; 1.29-5.49 Premeno-luteal or ERT
2-MeO Estradiol	0.02-0.07 µg/g Cr Postmenopausal; 0.03-0.08 µg/g Cr Premeno-luteal or ERT
2-MeO Estrone	0.06-0.29 µg/g Cr Postmenopausal; 0.26-0.68 µg/g Cr Premeno-luteal or ERT
2-MeO E1/2-OH E1	0.19-0.36 Postmenopausal; 0.21-0.38 Premeno-luteal or ERT
4-MeO Estradiol	<0.04 µg/g Cr
4-MeO Estrone	<0.04 µg/g Cr
4-MeO E1/4-OH E1	0.03-0.38 Postmenopausal; 0.05-0.13 Premeno-luteal or ERT
4-MeO E2/4-OH E2	0.14-0.73 Postmenopausal; 0.10-0.29 Premeno-luteal or ERT
Bisphenol A	1.5-4.5 µg/g Cr Postmenopausal; 1.11-3.74 µg/g Cr Premeno-luteal
Urinary Progestogens	
Pregnanediol	56-220 µg/g Cr Postmenopausal; 465-1609 µg/g Cr Premeno-luteal or PgRT
Allopregnanolone	0.3-1.31 µg/g Cr Postmenopausal; 2.23-14.87 µg/g Cr Premeno-luteal or PgRT
Allopregnanediol	1.38-6.75 µg/g Cr Postmenopausal; 14.65-76.71 µg/g Cr Premeno-luteal or PgRT
3α-Dihydroprogesterone	0.19-0.77 µg/g Cr Postmenopausal; 0.67-2.03 µg/g Cr Premeno-luteal or PgRT
20α-Dihydroprogesterone	0.60-5.53 µg/g Cr Postmenopausal; 3.93-11.62 µg/g Cr Premeno-luteal or PgRT
Deoxycorticosterone	0.37-1.97 µg/g Cr Postmenopausal; 0.69-2.23 µg/g Cr Premeno-luteal or PgRT
Corticosterone	2.32-9.88 µg/g Cr Postmenopausal; 3.19-9.59 µg/g Cr Premeno-luteal or PgRT
Pgdiol/E2	1000-1500 (Optimal Luteal Only)
Urinary Androgens	
DHEA	8.63-37.28 µg/g Cr Postmenopausal; 15.82-129.17 µg/g Cr Premeno-luteal or DHEAT
Androstenedione	2.07-7.94 µg/g Cr Postmenopausal; 3.93-13.53 µg/g Cr Premeno-luteal or ART
Androsterone	152-482 µg/g Cr Postmenopausal; 248-937 µg/g Cr Premeno-luteal or ART
Etiocholanolone	239-777 µg/g Cr Postmenopausal; 330-960 µg/g Cr Premeno-luteal or ART
Testosterone	0.66-2.89 µg/g Cr Postmenopausal; 1.22-3.97 µg/g Cr Premeno-luteal or ART

TEST NAME	WOMEN
Urinary Androgens	
Epi-Testosterone	0.39-1.32 µg/g Cr Postmenopausal; 2.01-4.66 µg/g Cr Premeno-luteal
T/Epi-T	0.5-3.0
5α-DHT	0.26-0.98 µg/g Cr Postmenopausal; 0.28-1.52 µg/g Cr Premeno-luteal or ART
5α,3α-Androstanediol	2.32-8.17 µg/g Cr Postmenopausal; 2.98-13.10 µg/g Cr Premeno-luteal or ART
Urinary Glucocorticoids	
Total Cortisol	13.23-39.26 µg/g Cr Postmenopausal; 12.26-33.12 µg/g Cr Premeno-luteal
Total Cortisone	23.32-59.61 µg/g Cr Postmenopausal; 23.27-50.88 µg/g Cr Premeno-luteal
Cortisol/Cortisone	0.5-0.7
Tetrahydrocortisol	281-711 µg/g Cr Postmenopausal; 214-546 µg/g Cr Premeno-luteal
Tetrahydrocortisone	551-1474 µg/g Cr Postmenopausal; 437-1184 µg/g Cr Premeno-luteal
Urinary Free Diurnal Cortisol	
Free Cortisol	7.8-29.5 µg/g Cr (1st Morning); 23.4-68.9 µg/g Cr (2nd Morning); 6.0-19.2 µg/g Cr (Evening); 2.6-8.4 µg/g Cr (Night)
Urinary Free Diurnal Cortisone	
Free Cortisone	31.6-91.6 µg/g Cr (1st Morning); 63.3-175.8 µg/g Cr (2nd Morning); 30.6-88.5 µg/g Cr (Evening); 15.5-44.7 µg/g Cr (Night)
Urinary Diurnal Melatonin MT6s	
Melatonin	18.0 - 40.9 µg/g Cr (1st Morning); 7.3 - 31.9 µg/g Cr (2nd Morning); 0.7 - 2.2 µg/g Cr (Evening); 1.7 - 11.1 µg/g Cr (Night)
Urinary Creatinine	
Creatinine (pooled)	0.3-2.0 mg/mL
Creatinine	0.3-2.0 mg/mL (1st morning); 0.3-2.0 mg/mL (2nd morning); 0.3-2.0 mg/mL (Evening); 0.3-2.0 mg/mL (Night)

The Steroid Hormone Cascade

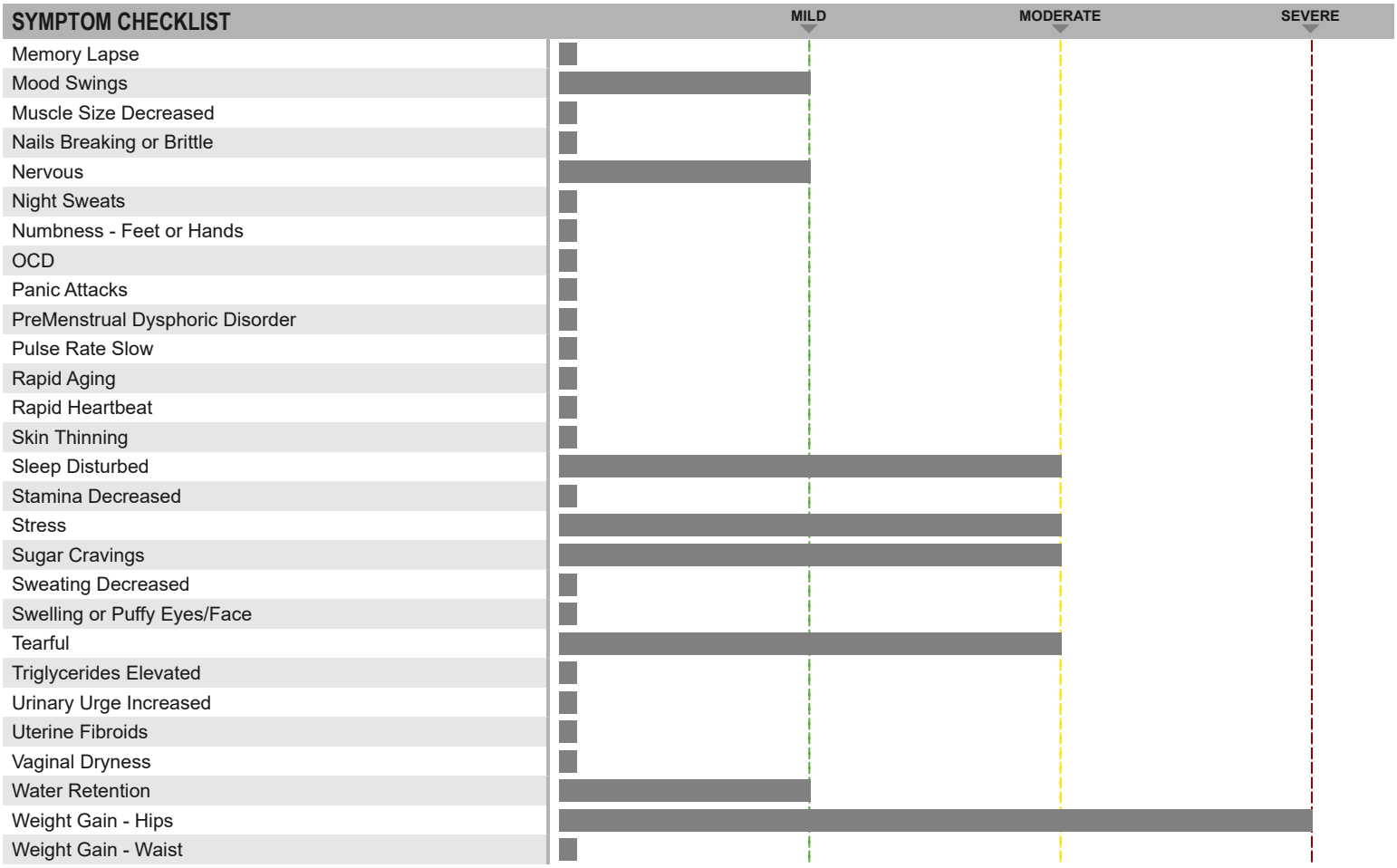


Enzyme Abbreviations

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

SYMPTOM CATEGORIES	RESULTS | 03/05/25
Estrogen / Progesterone Deficiency	14%
Estrogen Dominance / Progesterone Deficiency	32%
Low Androgens (DHEA/Testosterone)	11%
High Androgens (DHEA/Testosterone)	22%
Low Cortisol	17%
High Cortisol	22%
Hypometabolism	13%
Metabolic Syndrome	2%

SYMPTOM CHECKLIST	MILD	MODERATE	SEVERE
Aches and Pains			
Acne			
ADD/ADHD			
Addictive Behaviors			
Allergies			
Anxious			
Autism Spectrum Disorder			
Bleeding Changes			
Blood Pressure High			
Blood Pressure Low			
Blood Sugar Low			
Body Temperature Cold			
Bone Loss			
Breast Cancer			
Breasts - Fibrocystic			
Breasts - Tender			
Chemical Sensitivity			
Cholesterol High			
Constipation			
Depressed			
Developmental Delays			
Eating Disorders			
Fatigue - Evening			
Fatigue - Morning			
Fibromyalgia			
Foggy Thinking			
Goiter			
Hair - Dry or Brittle			
Hair - Increased Facial or Body			
Hair - Scalp Loss			
Headaches			
Hearing Loss			
Heart Palpitations			
Hoarseness			
Hot Flashes			
Incontinence			
Infertility			
Irritable			
Libido Decreased			
Mania			



Lab Comments

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

Estradiol and estrone are lower than the observed reference ranges for a premenopausal woman. This is common at perimenopause (ages usually ranging from 45-56, but younger in some women with premature ovarian failure). Erratic fluctuations in estrogen levels from high to low are more frequent during perimenopause (Prior J. et. al. J Clin Endocrinol Metab 1996: 81: 3127-3128), particularly when progesterone is also low. High ovarian estrogen production followed by a precipitous drop is usually associated with symptoms of both estrogen dominance and deficiency. As menopause progresses the estrogen fluctuations usually cease and estrogen levels drop to a lower baseline level. If symptoms of estrogen deficiency worsen (eg. hot flashes, night sweats, sleep disturbances), it would be worthwhile to consider supplementation with an estrogen (assuming no contraindications such as breast cancer) in combination with natural progesterone.

HYDROXYLATED (CATECHOL) ESTROGENS (2-OH E2 & E1, 4-OH E2 & E1, 16-OH E1) and 2-OH/16-OH RATIO

The hydroxylated estrogens are either low or within normal reference ranges for a premenopausal woman, inferring a lower risk for breast cancer.

The hydroxylation of estradiol and estrone represent the first phase of metabolism and elimination of these estrogens via urine. Following hydroxylation at the 2-, 4-, or -16 position, the estrogens undergo further modification (methylation, sulfation, glucuronidation) that increases their solubility and excretion in urine. The sulfate and glucuronide groups are removed by enzyme hydrolysis, which allows for measurement of the different types of hydroxylated estrogens, in addition to methylation of the hydroxyl groups (see below). The 2- and 4-hydroxylated E1 and E2 are referred to as catechol estrogens.

Research and clinical studies show that the 2-hydroxylated estrogens (2-OH E2 and 2-OH E1) are a safer pathway of hydroxylation than the 4-hydroxyestrogens (4-OH E2 and 4-OH E1), which bind to and damage DNA, leading to mutations that are associated with increased breast cancer risk. For reviews see: Cavalieri EL, Rogan EG Future Oncol 6(1): 75-79, 2010; and Lee, JR, Zava DT What Your Doctor May Not Tell You About BREAST CANCER: How Hormone Balance Can Help Save Your Life: Chapter 7.

2-hydroxylated estrogen metabolism is increased with cruciferous vegetables and extracts of them. The most commonly used are indole-3-

carbinol (I3C) and its metabolite diindolylmethane (DIM). Iodine also increases the 2-hydroxylation of estrogens, with a slight increase in 4-hydroxylation (Stoddard FR et.al. Int J Med Sci 5: 189-196, 2008). The more dangerous 4-hydroxylated estrogen metabolism is enhanced by exposure to environmental toxins, mostly petrochemical-based products but also heavy metals, that induce 4-hydroxylation pathway enzymes (1B1), and cause formation of Reactive Oxygen Species (ROS) that co-oxidize the catechol estrogens to quinones.

16-hydroxyestrone is another pathway of estrone metabolism and is a precursor to estriol (see Steroid Hormone Cascade). Early clinical research in humans suggested that a high urinary level of 16-hydroxyestrone relative to 2-hydroxylated estrogens (i.e. a low 2-OH E1 + 2-OH E2/16-OH E1 ratio), was associated with an increased risk of breast cancer in premenopausal women, but not in postmenopausal women. This has remained controversial and newer research suggests that while higher levels of 16-hydroxy estrone may indeed be slightly associated with increased breast cancer risk in premenopausal women, higher levels are, paradoxically, associated with a decreased risk in postmenopausal women (Huang J et.al. Analytica Chimica Acta 711: 60-68, 2012). Overall, more recent studies have not shown the 2/16 ratio to be useful for predicting breast cancer risk.

METHYLATION OF HYDROXYESTROGENS

The methylated forms of the 2- and 4-hydroxyestrogens (2MeO-E1/E2, and 4-MeO-E1/E2) are within normal to higher than reference ranges, which is considered beneficial. Higher levels of these methylated estrogen metabolites usually are due to healthy methylation pathways. Methylation of the 4-catechol estrogens is considered beneficial as this renders them inert and no longer capable of converting down-stream to the more reactive and dangerous 4-estrogen quinones.

A mid-normal to high ratio of 4-MeO-E1/4-OH-E1 or 4-MeO-E2/4-OH-E2 is desirable as this indicates adequate methylation of the more dangerous 4-hydroxylated catechol estrogens. If this ratio is low for either 4-MeO-E1/4-OH-E1 or 4-MeO-E2/4-OH-E2 then it is possible that either genetic polymorphisms have resulted in defective methylation (low COMT) or nutrients for methylation are low (e.g. B12, B6, folate). When 2- and 4-hydroxylated estrogens are high, this can eventually strain the methylation pathways and it is worthwhile to consider lowering the estrogen burden and balancing estrogen with natural progesterone.

The 2- and 4- hydroxyl catechol estrogens are methylated by the enzyme Catechol-O-Methyl Transferase (COMT), which renders them inert and harmless (Cavalieri EL, Rogan EG Future Oncol 6(1): 75-79, 2010). In this inert form the methylated catechol estrogens are excreted in urine. However, if methylation pathways are inadequate, due to low levels of COMT or lack of precursors of methylation (i.e. vitamins B6, B12, folate, betaine), the 2- and 4-hydroxyl estrogens can take a more insidious and dangerous pathway of metabolism, which is oxidation of the 4-hydroxylated estrogens (4-OH-E1 and 4-OH-E2) to their respective 4-quinones. Estrogen quinones, especially the 4-quinone of estradiol and estrone, are highly electrophilic and bind to DNA forming adducts that lead to permanent mutations in the DNA. Many studies have shown that high urinary levels of these 4-quinones of estradiol and/or estrone are associated with increased breast cancer risk, and research also suggests this same mechanism is responsible for increased risk for prostate cancer.

The 2- and 4-hydroxy estrogens are converted to their more dangerous oxidized quinone forms under oxidizing conditions in the cell, and this occurs rapidly in the presence of oxidized lipids, especially those from trans-hydrogenated fats and oxidizing heavy metals. 4-estrogen quinones, like all oxidized and electron-hungry molecules in the body, are inactivated when inactivated by binding to glutathione, the most ubiquitous antioxidant in the body. If glutathione is low, due to insufficient levels of minerals (selenium, iodine) and vitamins (C and E), the quinone estrogens are less likely to be detoxified (inactivated) and have potential to cause DNA mutations that increase cancer risk.

BISPHENOL A (BPA)

Bisphenol A (BPA) is within reference range. BPA is an endocrine disrupting chemical (EDC) derived from plastics used for making bottles, wraps for foods, and linings for food cans. BPA is not retained in the body for a prolonged period of time and is rapidly excreted into urine. High urinary levels of BPA indicate recent exposure to plastics that released excessive amounts of BPA into food or beverages consumed in the past 24-48 hr.

BPA acts as an EDC by binding to a activating both membrane and nuclear estrogen receptors in a manner similar to estradiol. Thus by mimicking the actions of endogenous estrogens, high levels of BPA can contribute to symptoms of estrogen dominance. High BPA levels have been associated with increased risks for many different health issues, including diabetes, breast cancer, and prostate cancer. When BPA levels are elevated, identification of its source and reducing exposure is worth considering.

PROGESTERONE METABOLITE (PREGNANEDIOL-PGDIOL)

The primary urinary progesterone metabolite, pregnanediol-PgDiol, is within the lower quadrant of the reference range for a premenopausal woman during the luteal phase of the menstrual cycle. In addition, PgDiol is NOT well balanced with the parent estrogens, estradiol and estrone, which are more within premenopausal ranges or higher (low PgDiol/E2 ratio).

In healthy premenopausal women with normal menstrual cycles and higher levels of estrogens, the estrogens are normally counterbalanced with adequate progesterone during the second half of the menstrual cycle. With adequate progesterone production the PgDiol/E2 ratio is usually in the 1000-2000 range. Adequate ovarian production of progesterone during this time prevents the estrogens from overstimulating growth of estrogen sensitive tissues such as the breasts and uterus, and allows for differentiation and specialization of these tissues. When estrogens are elevated relative to progesterone for prolonged periods of time this can increase risk of developing cancers of the breasts and uterus. This

occurs more commonly during the perimenopausal years (about ages 45-55, but varies in individuals) when risk for breast cancer rises sharply.

Consider balancing the higher estrogens with natural progesterone, particularly if symptoms of estrogen dominance are/become problematic.

PROGESTERONE METABOLITES

The urinary progesterone metabolites are within or near normal reference ranges seen in premenopausal women. The urinary progestogen metabolites included encompass the primary urinary metabolite, pregnanediol (Pgdiol), and four other more minor metabolites that belong to the pregnane (Allo-pregnanolone, Allo-pregnanediol) and pregnene (3a-dihydroprogesterone, 20a-dihydroprogesterone) categories. In postmenopausal women the level of pregnanediol is expected to be much lower than in premenopausal women (mean values 81 and 1324 µg/g creatinine, respectively). The mean and range levels for urinary pregnanediol established in premenopausal women during the early follicular and mid-luteal phases of the menstrual cycle are 152 µg/g creatinine (range 92-346) and 1324 µg/g creatinine (range 465-1609 ug/g creatinine), respectively. Thus, about a 10-fold increase in Pgdiol is expected during the progression from the follicular to the luteal phase of the menstrual cycle. The urinary ranges of pregnanediol during the luteal phase are equivalent to a range of about 3-25 ng/mL progesterone in blood (capillary whole blood and venous serum or plasma) and 50-250 pg/mL in saliva. Optimal luteal ovarian production of progesterone is reflected in all three body fluids (urine, blood, salivary), which is roughly > 1300 µg PgDiol/g creatinine in urine, > 10 ng Progesterone/mL in blood, and > 100 pg Progesterone/mL in saliva.

MINERALCORTICOID PRECURSORS (DEOXYCORTICOSTERONE-DOC; CORTICOSTERONE-CC)

Deoxycorticosterone (DOC) and corticosterone (CC) are lower than reference ranges. Progesterone is converted to DOC via 21-hydroxylase, and to CC via 11-beta hydroxylase (see Steroid Hormone Cascade), CC serves as a substrate for aldosterone synthetase, which converts CC to aldosterone. Aldosterone is important for maintaining balanced levels of sodium and potassium and regulating blood pressure.

Low aldosterone, resulting from inadequate DOC and CC precursors and low levels of enzymes 21-hydroxylase, 11-beta-hydroxylase, or aldosterone synthetase is usually associated with low blood pressure. The most common symptoms of low aldosterone often present as fatigue, salt craving, inability to think clearly, dizziness or lightheadedness on standing, and palpitations.

ANDROGEN PRECURSORS (ANDROSTENEDIOL, DHEA)

DHEA(S) is lower than reference range for a premenopausal woman. This usually indicates adrenal exhaustion caused by excessive stressors.

In premenopausal women about half of the androstenedione is derived from the ovaries and the other half from the adrenals. DHEA(S) is produced mostly by the adrenal glands. Because androstenedione (A4) is within normal range and DHEA(S) is low, this suggests that ovarian androgen production is normal.

At menopause, most of the androstenedione derives from DHEA(S) produced by the adrenal glands. DHEA is synthesized in the adrenal glands and is rapidly sulfated to DHEA-sulfate (DHEAS) to extend its half-life in blood. Adrenal DHEA(S) is a precursor of A4, which is further converted into testosterone via 17-beta-hydroxysteroid dehydrogenase (see Steroid Hormone Cascade), Epi-Testosterone via 17-alpha-hydroxysteroid dehydrogenase, or estrone (E1) via aromatase. In most individuals endogenously produced A4 is converted into near equal amounts or T and Epi-T (expect T/Epi-T ratio to be about 1). More conversion of A4 to E1 occurs in individuals with higher amounts of adipose (fat) tissue, which expresses high levels of aromatase.

Low levels of either DHEA(S) or A4 are often associated with symptoms of low androgens, particularly if the more potent down-stream androgen metabolites, testosterone and DHT, are also low. DHEA is commonly used as a supplement to raise testosterone levels in women (less effective in men as it does not raise circulating levels of T and often converts to E1 in individuals with higher body fat). If low androgen symptoms are problematic and T is low or within lower expected reference range, consider supplemental DHEA to raise the level of T.

ANDROGENS AND METABOLITES

Testosterone, 5-alpha DHT, and Epi-T are within expected ranges for a premenopausal woman. If symptoms of androgen deficiency are, or become, problematic, androgen therapy (DHEA or testosterone) is worth considering, assuming no contraindications. DHEA therapy increases both DHEAS and testosterone levels in women, but may also increase estrogens, which need to be countered with natural progesterone if increased by DHEA.

Androgens are important for strengthening structural tissues such as muscles, bone, connective tissue, and skin. They 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. Androgens are also precursors to the estrogens, estradiol and estrone. The most potent of the androgens is dihydrotestosterone (DHT), which is created from testosterone via 5a reductase. Testosterone itself is derived mostly from androstenedione and DHEA. In premenopausal women about half of the testosterone is derived from androstenedione produced by the ovaries, and the other half from peripheral conversion of DHEA manufactured in the adrenals. Following menopause the ovarian contribution of androgens is lower.

EPI-TESTOSTERONE AND RELATIONSHIP TO TESTOSTERONE.

Epi-testosterone (Epi-T) and testosterone (T) are created in about equal amounts from androstenedione and DHEA. The ratio of T/Epi-T should be about 1 under normal circumstances. When testosterone is supplemented with any delivery system except topical, the T/Epi-T ratio increases, which reflects an increase in the exogenous testosterone, but not Epi-T, which represents endogenous production.

5-ALPHA 3-ALPHA ANDROSTANEDIOL (ADIOL)

The downstream metabolite of DHT, 5-alpha 3-alpha androstanediol (Adiol), is within expected reference range. Adiol is considered a neuroactive steroid that can passively enter the brain from the bloodstream through the blood brain barrier.

Adiol binds to GABAa receptors in the brain and has a similar anxiolytic (calming) effect, albeit weaker than allopregnanolone. It also interacts with the dopaminergic pathways in the brain and is associated with the dopamine pleasure and reward pathway. Thus, low levels of Adiol are more likely to be associated with conditions/symptoms common to low dopamine, and high levels with high dopamine. Fibromyalgia and chronic fatigue syndrome (CFS) are common in individuals with low dopamine, as are symptoms of brain fog, achy muscles, and excessive fatigue.

TOTAL GLUCOCORTICOIDS

Total cortisol (F) and cortisone (E), and the ratio of cortisol/cortisone, are within the expected reference ranges. 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. If symptoms of cortisol imbalance are not problematic this likely indicates good adrenal function. However, if symptoms of cortisol imbalance are self-reported as problematic this likely indicates that the total production is an average of both low and high values that have occurred throughout the day. In this case, adrenal function would be more appropriately evaluated based on individual cortisol measurements throughout the day (see Urinary Free Cortisol results).

URINARY FREE CORTISOL (F) and FREE CORTISONE (E)

The Urinary Free Cortisol (F) is not following a normal circadian rhythm and is outside (lower) the normal reference ranges throughout most of the day. First morning F is within normal reference range; however, the second morning void is low, indicating a poor Cortisol Awakening Response (CAR). Cortisol (E), the inert metabolite of F, is within normal reference ranges in the first two morning voids, indicating that F formed is converted to E, which may be partly responsible for the poor CAR. Symptoms are consistent with excessive stressors, which can result in both high and low cortisol synthesis, depending on the timing of the stressors or use of medications that affect adrenal cortisol synthesis.

Low F and E in the evening and at night before bed may also result from suppression of cortisol synthesis by anti-anxiety medications, or the use of synthetic glucocorticoids in the morning (none indicated) that suppress the HPA axis and lower ACTH-cortisol synthesis. Assuming no medications are lowering adrenal cortisol synthesis after the first morning void, lower cortisol levels can be caused by different types of stressors such as emotional/psychological stress, sleep deprivation, low protein diet, nutrient deficiencies (particularly low vitamins C and B5), physical insults (surgery, injury, diseases, inflammatory conditions), chemical exposure, low cortisol precursors (pregnenolone, progesterone) or pathogenic infections (bacterial, viral, fungal).

Adequate sleep and rest, gentle exercise, proper diet (adequate protein), natural progesterone, adrenal extracts, herbs, and nutritional supplements (vitamins C and B5) are some of the natural ways to help support adrenal cortisol production during the day. 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.

MELATONIN METABOLITE: 6-SULFATOXYMELATONIN (MT6s)

The urinary metabolite of melatonin, 6-sulfatoxymelatonin (MT6s), is above normal reference ranges in the first urine void, but returns to levels within expected reference ranges throughout the remainder of the day. Overall, melatonin is following a normal circadian rhythm. Higher melatonin in the first morning void may be normal for this individual (often higher in younger individuals), but may also indicate supplementation with melatonin, a pharmaceutical product that affects melatonin metabolism and clearance, or the use of sleep-inducing herbs the night before collection. Hormone therapies at night might also increase night-time melatonin and result in a higher first morning void.

Melatonin is known to have many beneficial effects in the body. It helps slow the aging process, is a potent anti-oxidant, regulates the immune system, inhibits formation and growth of tumors such as breast and prostate cancers, and helps regulate the synthesis of the sex-hormones estradiol and progesterone (melatonin increases progesterone, decreases estrogens by inhibiting aromatase, and down-regulates cellular estrogen receptors, which diminishes response of estrogen-sensitive tissues to estrogens). Low melatonin, caused by excessive light exposure during the dark hours, or calcification of the pineal gland caused by aging, has been associated with many different dysfunctions and diseases such as immune dysfunction, neurodegenerative disorders (Alzheimer's disease, senile dementia), pain disorders, cardiovascular disease, cancers of the breast and prostate, and type 2 diabetes (Hardeland R. Aging and Disease 3 (2): 194-225, 2012). Low melatonin is also thought to contribute to obesity in people with insomnia or those who do night shift work.

For more general information about melatonin please see: http://www.nlm.nih.gov/medlineplus/druginfo/natural/940.html

Urinary creatinine is within normal reference ranges throughout the day, based on testing diurnal 2x, 4x, or 6x urine collections.

Creatinine values slightly lower than range usually indicate overly dilute urine from excessive water intake shortly before collection, or not spacing collection of multiple urine samples by at least 2 hr (most problematic in second morning urine collection). Creatinine slightly higher than range is usually due to inadequate hydration.
Extreme low or high values may be caused by kidney or other metabolic disorders (e.g. metabolic syndrome and diabetes).