TEST REPORT

8605 SW Creekside Place
Beaverton, OR 97008
Phone: 503-466-2445 Fax: 503-466-1636

ZRT LABORATORY

 # D2026 05 08 338 S

Ordering Provider:
Getuwell

Samples Received
05/07/2026

Report Date
05/13/2026

Samples Collected
Saliva - 05/05/26 06:00
Saliva - 05/05/26 12:30
Saliva - 05/05/26 18:00
Saliva - 05/05/26 23:00

Patient Name: Saliva LCMS Hormones 23 with 4-Point Cortisol
Patient Phone Number:

Gender	Last Menses	Height	Waist
Female	Unspecified	5 ft 5 in	Unspecified
DOB	**Menses Status**	**Weight**	
1/1/1971 (55 yrs)	Hysterectomy (ovaries not removed)	Unspecified	

TEST NAME	RESULTS | 05/05/26	RANGE
Salivary Steroids & Other Analytes (LC-MS/ECLIA)		
Estradiol	0.4	0.3-0.9 pg/mL Postmenopausal
Estriol	<0.9	<1.9 pg/mL Postmeno or Premeno-Follicular or Synthetic HRT
Estrone	0.5 L	0.9-3.1 pg/mL Postmeno Premeno-Follicular or Synthetic HRT
Ethinyl Estradiol	<0.4	<0.4 pg/mL
Pregnenolone Sulfate	<1 L	1-23 pg/mL
Progesterone	6	5-34 pg/mL Postmenopausal
Ratio: Pg/E2 (Saliva LCMS)	15 L	23-196
Allopregnanolone	<15.0	<15 pg/mL
17OH-Progesterone	35 H	6-28 pg/mL
Androstenedione	57	36-93 pg/mL
Testosterone	12	7-22 pg/mL
DHT	<7	<7 pg/mL
DHEA	122	77-287 pg/mL
DHEAS	1.4	0.8-8.0 ng/mL
7-keto DHEA	90	39-359 pg/mL
Ratio: DHEA/7keto DHEA	1.4	0.5-2
11-Deoxycortisol	66 H	12-48 pg/mL
Cortisol	9.0 H	2.5-6.2 ng/mL (morning)

CLIA Lic # 38D0960950
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David T. Zava, Ph.D.
Laboratory Director

Alison McAllister, ND.
(Ordering Provider unless otherwise specified on page 1)

TEST NAME	RESULTS | 05/05/26	RANGE
Salivary Steroids & Other Analytes (LC-MS/ECLIA)		
Cortisone	24.0 H	10.0-23.3 ng/mL (morning)
Corticosterone	103 H	11-66 pg/mL
Aldosterone	61	16-63 pg/mL
Melatonin	25 H	3-22 pg/mL
Anastrozole	<5	<5 pg/mL
Finasteride	<5	<5 pg/mL
Letrozole	<5	<5 pg/mL
Cortisol	1.0 L	1.11-2.74 ng/mL (noon)
Cortisol	0.8	0.61-1.33 ng/mL (evening)
Cortisol	0.5	0.25-0.64 ng/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

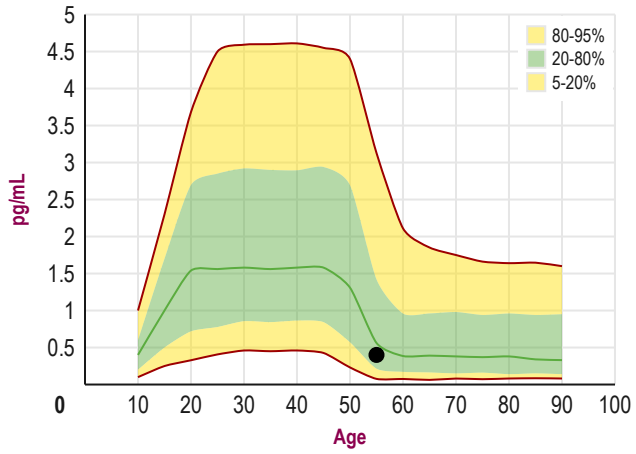
None

Graphs

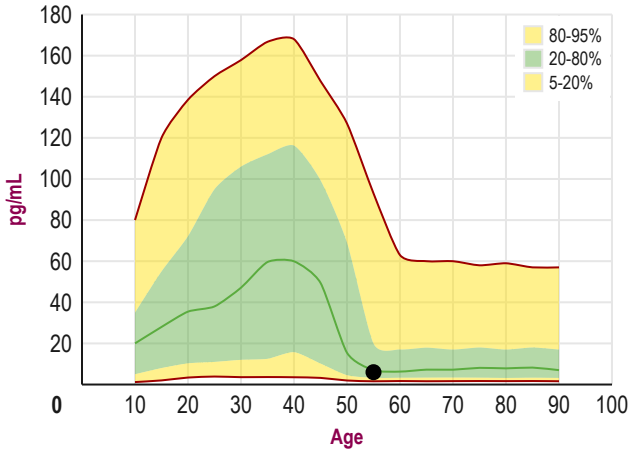
Disclaimer: Graphs below represent averages for healthy individuals not using hormones. Supplementation ranges may be higher. Please see supplementation ranges and lab comments if results are higher or lower than expected.

Average ▼▲ Off Graph

Saliva Estradiol



Saliva Progesterone



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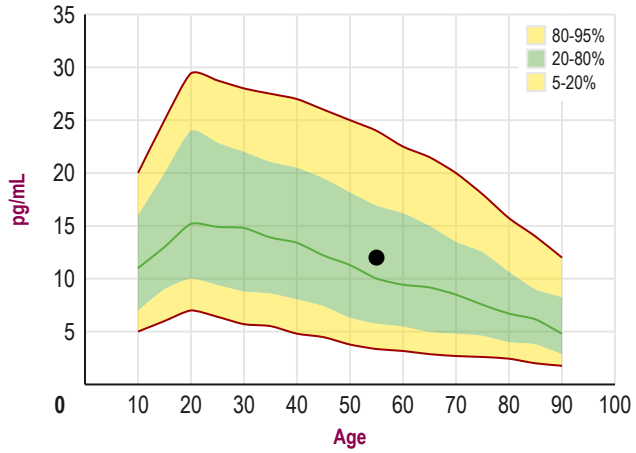
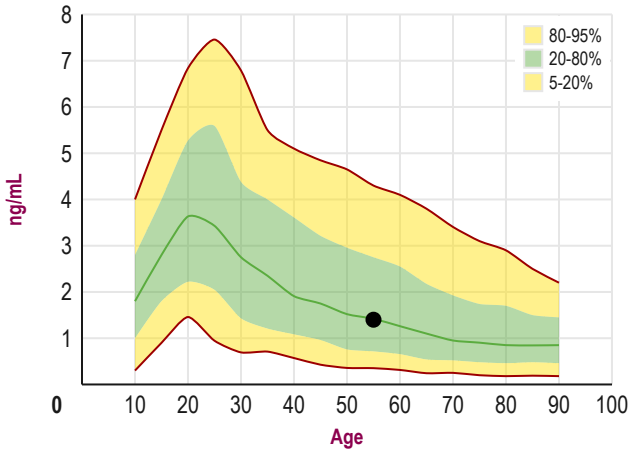
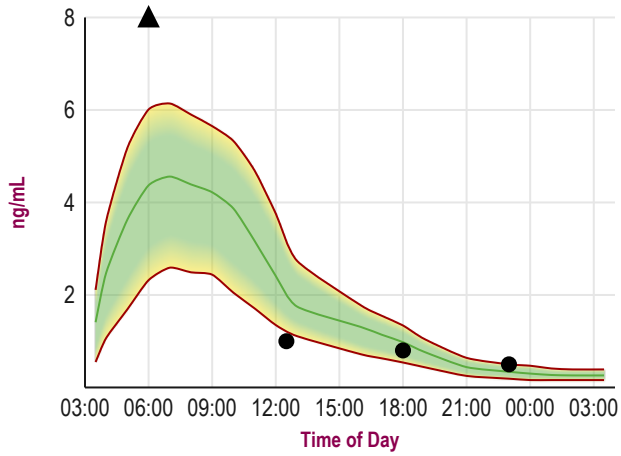
Saliva Testosterone



Saliva DHEAS



Saliva Cortisol



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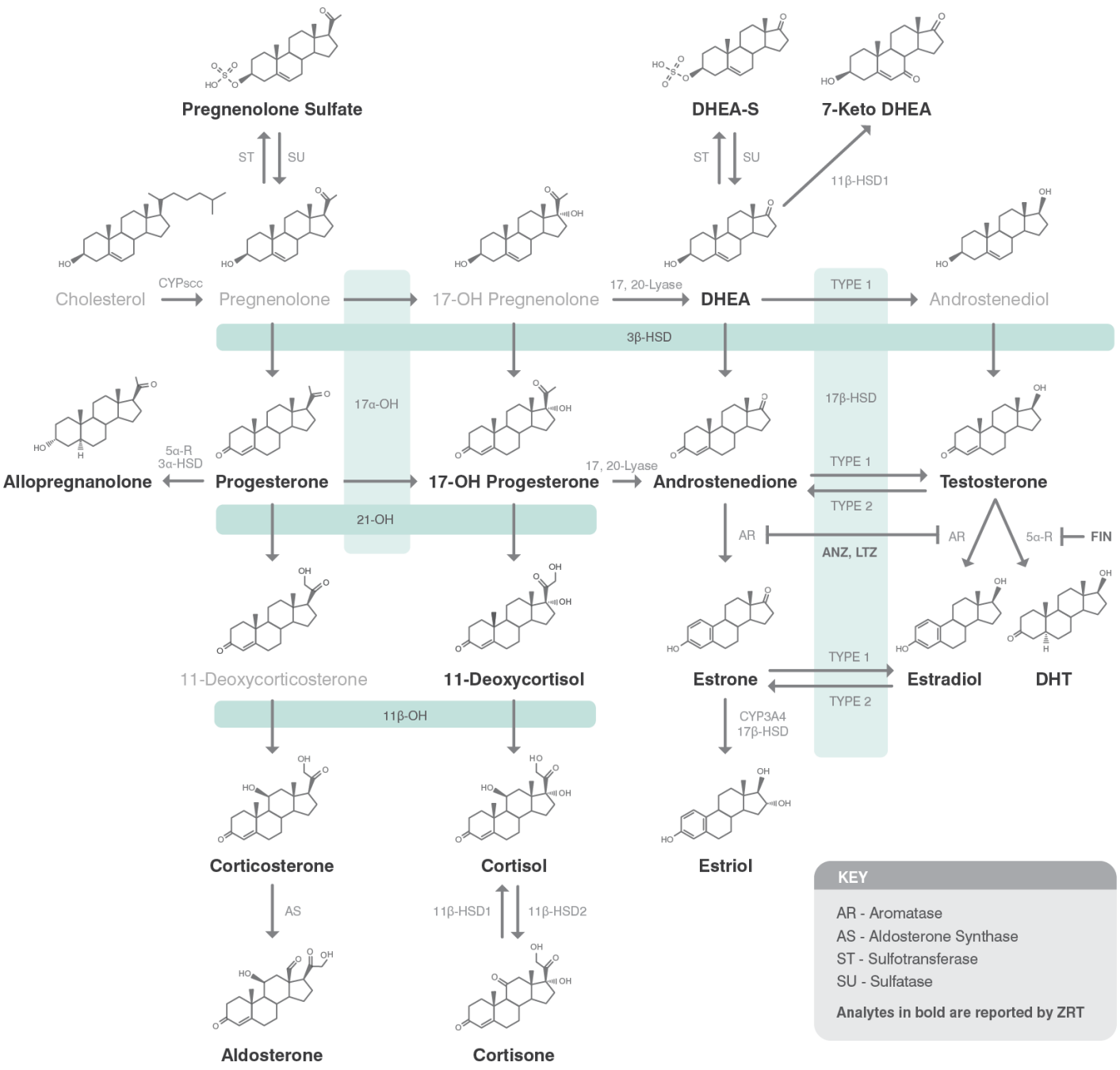
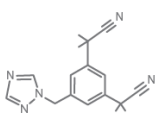
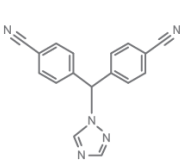
Saliva Steroid Cascade



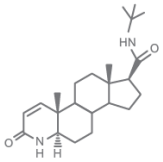
Steroid Synthesis Inhibitors



Anastrozole
ANZ

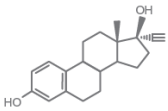


Letrozole
LTZ



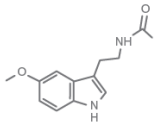
Finasteride
FIN

Synthetic Contraceptive Estrogen



Ethinyl Estradiol

Melatonin



Mel

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Disclaimer: Symptom Categories below show percent of symptoms self-reported by the patient compared to total available symptoms for each category. For detailed information on category breakdowns, go to www.zrtlab.com/patient-symptoms.

SYMPTOM CATEGORIES	RESULTS | 05/05/26
Estrogen / Progesterone Deficiency	20%
Estrogen Dominance / Progesterone Deficiency	19%
Low Androgens (DHEA/Testosterone)	25%
High Androgens (DHEA/Testosterone)	15%
Low Cortisol	36%
High Cortisol	19%
Hypometabolism	20%
Metabolic Syndrome	22%

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			

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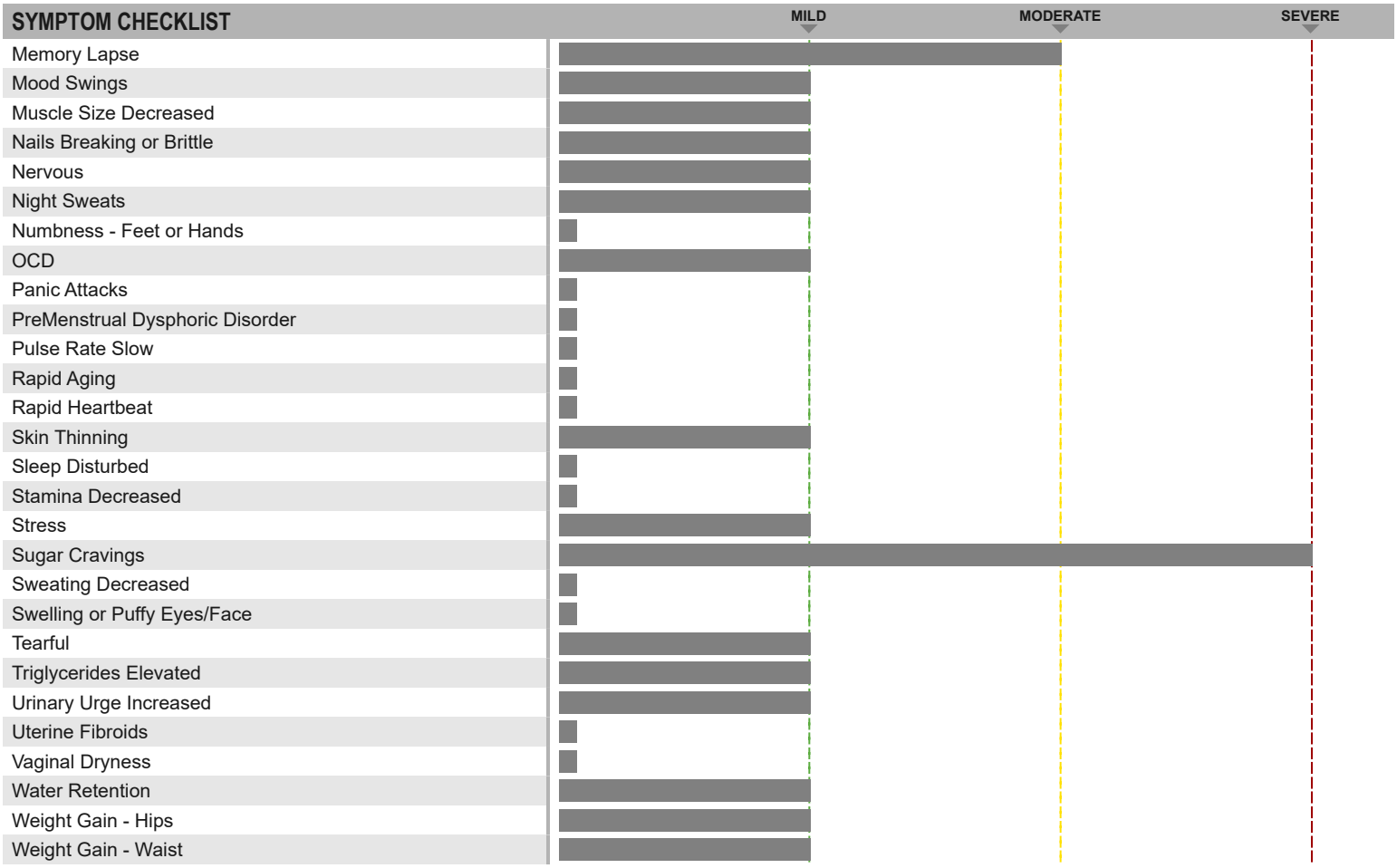

Lab Comments

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

The parent estrogens (estradiol, estrone, estriol), are lower than reference ranges. If symptoms of estrogen deficiency are problematic, consider bioidentical estrogen replacement therapy balanced with natural progesterone, assuming no contraindications. The most common symptoms of estrogen deficiency include hot flashes and night sweats, sleep disturbances, vaginal dryness, incontinence, and long-term effects on bone loss, insulin resistance, and cardiovascular risk. Quality of life issues and the long-term risk/benefit of estrogen deficiency should be carefully evaluated to determine if low-dose natural sex-hormone therapy (estrogen, androgen, progesterone) would be more beneficial than harmful.

PROGESTOGENS: PROGESTERONE, ALLOPREGNANOLONE, PREGNENOLONE SULFATE, 17-HYDROXYPROGESTERONE

Progesterone is lower than expected luteal range for a postmenopausal woman. If estrogen therapy is implemented consideration should be given to balancing it with bioidentical progesterone. Optimally, estradiol should be within mid-range (1-3 pg/ml) and balanced with progesterone at a Pg/E2 ratio of about 50-200. For progesterone to function optimally it is necessary for estradiol to be within luteal range first to induce cellular estrogen and progesterone receptors to enable tissues (e.g. uterus, breasts, brain) to respond optimally to progesterone. Insufficient progesterone in the presence of physiological or high levels of estradiol will not fully blunt the growth promoting actions of estrogens. Conversely, if estradiol is very low, and excess progesterone supplement is used then the benefits of estrogen therapy are less likely as progesterone will suppress tissue estrogen receptors.

Allopregnanolone, the neuroactive and downstream metabolite of progesterone, is lower than detectable range, which is not unexpected when progesterone is low. Allopregnanolone is usually not within measurable range until salivary progesterone is within the upper ranges of luteal phase (> 200 pg/ml). The level of allopregnanolone in saliva and blood is usually about 10% of progesterone but shows individual variability mostly based on the activities of the two enzymes (5-alpha reductase-5aR and 3 beta hydroxysteroid dehydrogenase-3BHSD) that convert progesterone to allopregnanolone. Lower allopregnanolone could contribute to symptoms of estrogen imbalance. Allopregnanolone is a neuroactive steroid that helps balance the stimulating actions of estradiol on the brain function as well as its growth promoting actions in estrogen/progesterone target tissues such as the breasts and uterus. Progesterone supplementation, both oral and topical, significantly raise allopregnanolone to detectable levels, and help create a calming (anxiolytic) effect.

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Pregnenolone Sulfate (PS), a precursor to pregnenolone is lower than range. PS is a precursor to 17 OH-pregnenolone, which is a precursor to 17 OH-progesterone. Low PS could indicate poor sulfation of pregnenolone, or low pregnenolone synthesis, which would result in low levels of all downstream metabolites. Pregnenolone and 17-OH pregnenolone are not measured in these tests, but the metabolite of 17-OH pregnenolone, 17-OH progesterone is measured, and found to be higher than range. This would indicate that while PS is low, pregnenolone is within range, or high as it gives rise to 17-OH progesterone, which is high. The majority (> 95%) of pregnenolone in the bloodstream, and in saliva, is in the form of pregnenolone sulfate. However, in this individual pregnenolone sulfate is low, but its downstream metabolite 17 OH progesterone is, paradoxically, high. This could indicate that pregnenolone sulfate is rapidly desulfated by high levels sulfatase enzyme, converting it to pregnenolone (not tested) and its downstream metabolites 17-OH progesterone (high by testing) and 17-OH-pregnenolone (not tested). Pregnenolone, like progesterone, is a precursor source for allopregnanolone in the brain (see above). Consider pregnenolone supplementation.

17-hydroxy progesterone (17OHP) is higher than reference range. 17OHP is a pivotal precursor to both glucocorticoids, androgens, and estrogens (see Steroid Hormone Cascade). If both glucocorticoids (cortisol, cortisone), androgens (androstenedione, testosterone, DHT) and estrogens (estradiol, estrone) are within range or higher, this indicates that 17OHP is effectively funneled down these metabolic pathways. For conversion to androgens 17OHP is first metabolized via 17,20 lyase to androstenedione and from there to testosterone and DHT. Testosterone serves as a precursor to estradiol. For conversion to glucocorticoids 17OHP is metabolized via 21-hydroxylase to 11-deoxycortisol, and from there metabolized via 11B-hydroxylase to cortisol. High 17OHP is usually a result of high levels of precursors, which may be due to pregnenolone or progesterone supplementation. On the other hand, high 17OHP may be caused by a deficiency in the 21-hydroxylase enzyme if its downstream metabolite 11-deoxycortisol (precursor to cortisol) is low. If 11-deoxycortisol is within normal range, or high, this would indicate that high 17OHP is NOT due to 21-hydroxylase deficiency (common in individuals with Congenital Adrenal Hyperplasia-CAH. 21-hydroxylase deficiency is the most common enzyme deficiency in the steroidal cascade and, if problematic, would result in high 17OHP and low levels of glucocorticoids (cortisol and cortisone) and mineralocorticoids (deoxycorticosterone, corticosterone, and aldosterone).

TESTOSTERONE is within range and symptoms of androgen imbalance are minimal.

ADRENAL ANDROGENS: DHEA, DHEAS, 7-KETO DHEA

The weak adrenal androgen DHEA is within mid-reference range; however, its more stable and abundant sulfate metabolite DHEAS is within the lower quadrant of the reference range, indicating poor sulfation. DHEA and DHEA-S synthesized in the adrenal glands at their highest levels during the late teens and early adulthood and drop steadily about 1% per year with age to the lower end of range. DHEA is a precursor to both androgens and estrogens. If DHEA is within normal range, but androgens or estrogens are low this likely indicates either low enzyme activity that metabolizes DHEA to androstenedione (3 beta-HSD) and/or conversion of androstenedione to testosterone (17beta-HSD type 1).

7-Keto DHEA is also within reference range. 7-keto DHEA is formed in near equal amounts to DHEA in the adrenal glands and the ratio is about 1 and ranges from 0.5-2. Exogenous DHEA supplementation results in an increase of DHEA, but no change in 7-keto DHEA causing an increase in the DHEA/7-keto DHEA ratio. Slight differences outside the expected ratio of 0.5-2 may be normal for an individual or caused by other medications that affect adrenal DHEA synthesis.

7-keto DHEA differs from DHEA in that the former is not a precursor for downstream androgens (testosterone and DHT) or estrogens (estradiol, estrone, estriol) and will not increase levels of these with 7-keto DHEA supplementation. 7-keto DHEA is reported to have many benefits such as boosting the actions of anabolic hormones (thyroid, IGF-1) to improve and strengthen immune function, cognition and memory, energy levels, muscle strength, insulin sensitivity, blood pressure, and weight loss.

Cortisol is high in the morning, low at noon, and returns to normal the remainder of the day. High morning cortisol may be caused by excessive stressors (usually emotional) or low blood sugar levels that result from overnight fasting. A high morning cortisol followed by a low noon level is usually caused by adrenal fatigue, morning use of medications that affect cortisol synthesis or its rate of clearance, or inability to regulate blood sugar levels. Adrenal dysfunction is most commonly caused by stress (emotional), sleep deprivation, poor diet (low protein-particularly problematic in vegetarians), nutrient deficiencies (particularly low vitamins C and B5), and low levels of cortisol precursors (pregnenolone and progesterone) Thyroid or androgen replacement therapy (DHEA or testosterone) in the morning following the first morning saliva collection can cause a transient lowering of adrenal cortisol synthesis (inhibits CRH/ACTH axis) which results in lowered noon cortisol levels. Low blood sugar levels, which often occur with overnight fasting, can result in a higher morning cortisol, followed by lower than normal levels at noon. A normal daily output of cortisol is essential to maintain normal metabolic activity, help regulate steady state glucose levels for the brain, and optimize immune function. Cortisol levels dropping below the normal range at any time during the day may result in fatigue, sugar craving, and some of the symptoms commonly attributed to thyroid deficiency (e.g. feeling cold, low libido, depression, anxiety). Normal physiological levels of cortisol are essential for optimal thyroid function. For additional information about strategies for supporting adrenal health 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.

ALDOSTERONE, CORTICOSTERONE

Aldosterone is within expected reference range but its precursor corticosterone (CC) is higher than reference range. The precursor of CC, deoxycorticosterone was not tested; however, it has weak mineralocorticoid activity and if high could cause pseudo hyperaldosteronism (normal to low aldosterone but hyperaldosteronism symptoms-e.g. high blood pressure).

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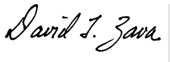
Elevated CC might suggest, on the other hand, that aldosterone could be low (expect low blood pressure) if these metabolites are not converted to aldosterone by aldosterone synthetase and are accumulating instead (see http://omim.org/entry/203400). Aldosterone in this individual is within normal range. Hypoaldosteronism is thought to be caused by a metabolic defect in aldosterone synthesis by the enzyme aldosterone synthetase, which leads to accumulation of the precursor CC, lower aldosterone, and associated symptoms of hypoaldosteronism (low blood pressure and faintness with standing up, frequent urination, salt wasting, hearing problems, fatigue, poor exercise tolerance and painful muscles and joints). In this scenario cortisol and downstream metabolites are usually within normal range. While CC is elevated, aldosterone is within normal range and symptoms of hypo- and hyper-aldosteronism are minimal.

MELATONIN

First morning melatonin is slightly higher than reference range. This is common in children, teens, and younger individuals (< 30 years of age), and in those taking low dose melatonin (none indicated) or melatonin precursors used to raise serotonin levels (e.g. 5-hydroxytryptophan and its precursor tryptophan, or herbs that contain melatonin or increase its synthesis). Early morning salivary melatonin shortly after awakening (within 30 minutes) is somewhat representative of melatonin produced during the dark period throughout the night. Light exposure in the morning will begin to rapidly deplete melatonin levels in the bloodstream and saliva.

Melatonin is known to have many different beneficial effects in the body. It helps slow the aging process, is a potent antioxidant, 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 and decreases estrogens).

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



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