# **TEST REPORT**

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# D2024 04 01 079 SB

**Ordering Provider:** 

Getuwell

Samples Received 04/01/2024

Report Date 04/17/2024

Samples Collected

Saliva - 03/27/24 06:01 Saliva - 03/27/24 12:11 Saliva - 03/27/24 16:30 Saliva - 03/27/24 22:30 Blood Spot - 03/27/24 06:10

Patient Name: Comprehensive Female Profile II

**Patient Phone Number:** 

| Patient Phone Number:                           |  |                         |   |  |
|---|--|-------------------------|---|--|
| <b>Gender</b><br>Female                         | <b>Last Menses</b> 01/01/2016          | <b>Height</b> 5 ft 4 in | <b>Waist</b><br>40 in                                     |  |
| <b>DOB</b> 5/21/1973 (50 yrs)                   | <b>Menses Status</b><br>Postmenopausal | <b>Weight</b><br>140 lb | <b>BMI</b> 24.0   |  |
| TEST NAME                                       | RESULTS   03/27/                       | 24 RAN                  | GE  |  |
| Salivary Steroids                               |  |                         |   |  |
| Cortisol  | 4.8                                    | 3.7-9.                  | .5 ng/mL (morning)  |  |
| Cortisol  | 0.7 L                                  | 1.2-3.                  | .0 ng/mL (noon)   |  |
| Cortisol  | 1.2                                    | 0.6-1.                  | .9 ng/mL (evening)  |  |
| Cortisol  | 0.8                                    | 0.4-1.                  | .0 ng/mL (night)  |  |
| Blood Spot Steroids & Other Analytes (LC-MS/MS) |  |                         |   |  |
| Estradiol                                       | 13                                     | <10-2                   | 26 pg/mLPost or Premeno + Synthetic E                     |  |
| Estriol   | <40                                    | <40 p                   | g/mL Premeno, Postmeno                                    |  |
| Estrone   | <15                                    | <15-3                   | 34 pg/mL Postmenopausal, early follicular                 |  |
| Progesterone                                    | 0.2                                    | <0.1-0                  | 0.9 ng/mL Postmeno, Premeno-Follicular or Premeno + Syn P |  |
| Ratio: Pg/E2                                    | 15 L                                   | Pg/E2                   | 2 (bloodspot-optimal 100-500)                             |  |
| Testosterone                                    | 18                                     | 13-38                   | ng/dL Postmeno or Premeno + Synthetic E                   |  |
| DHEAS   | 120                                    | 17-20                   | 7 μg/dL   |  |
| Blood Spot                                      |  |                         |   |  |
| SHBG  | 27                                     | 15-12                   | 20 nmol/L   |  |
| Blood Spot Thyroids                             |  |                         |   |  |
| TSH   | 1.5                                    | 0.5-3.                  | .0 μU/mL  |  |
| Free T3   | 2.1 L                                  | 2.4-4.                  | .2 pg/mL  |  |
| Free T4   | 0.9                                    | 0.7-2.                  | .5 ng/dL  |  |



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| TEST NAME                  | RESULTS   03/27/24 | RANGE                           |
|----------------------------|--------------------|---------------------------------|
| <b>Blood Spot Thyroids</b> |                    |                                 |
| TPOab                      | 6                  | 0-150 IU/mL (70-150 borderline) |

<dI = 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.</p>

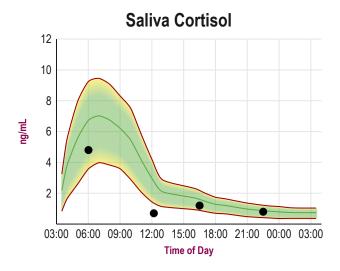
### **Therapies**

None Indicated

#### **Graphs**

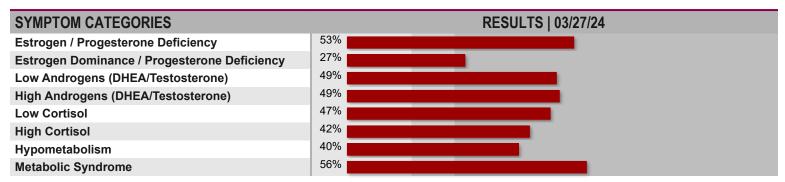
Disclaimer: Graphs below represent averages for healthy individuals not using hormones. Supplementation ranges may be higher. Please see supplementation ranges and lab comments if results are higher or lower than expected.

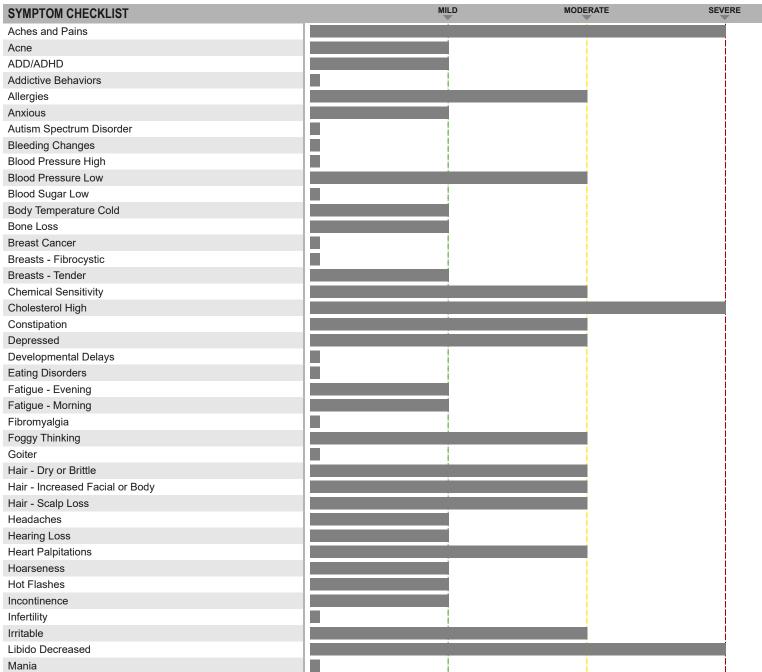
Average ▼▲ Off Graph

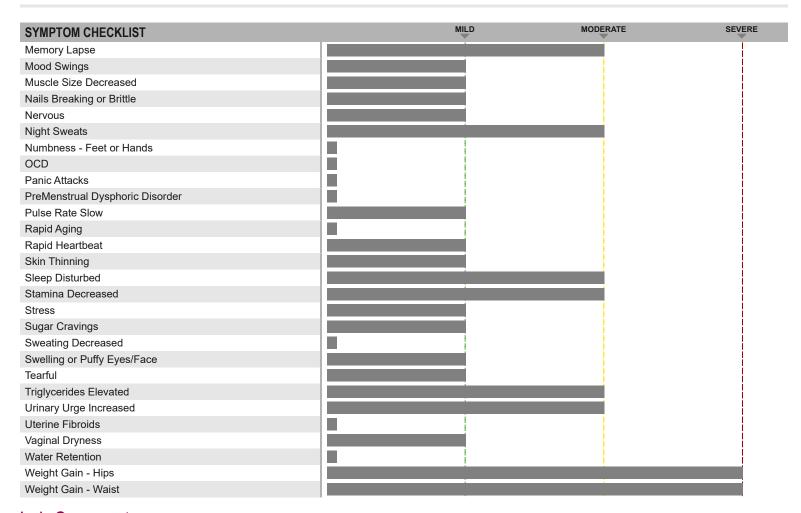


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Disclaimer: Symptom Categories below show percent of symptoms self-reported by the patient compared to total available symptoms for each category. For detailed information on category breakdowns, go to www.zrtlab.com/patient-symptoms.







## Lab Comments

Cortisol is within normal range in the morning, drops to a low level at noon, recovers to normal in the evening and then rises to a high-normal level at night. Lower noon cortisol is often indicative of the use of medications (eg. thyroid, androgens, hormonal contraceptives) that can cause a transient suppression of adrenal cortisol synthesis (androgens, hormonal contraceptives, anti-inflammatory synthetic glucocorticoids, herbal adaptogens) or increase cortisol clearance (eg. thyroid). Higher evening/night cortisol indicates some form of stressor. The most common stressors that can cause raise cortisol include: psychological stress (emotional), sleep deprivation, physical insults (surgery, injury, diseases), chemical exposure (environmental pollutants, excessive medications), and pathogenic infections (bacterial, viral, fungal). Chronic high evening/ night cortisol is commonly associated with sleep disturbances, fatigue, depression, weight gain in the waist, bone loss, and anxiety. This condition can also impair the actions of other hormones such as insulin and thyroid, causing symptoms of their deficiency, even though the levels of these hormones may be within normal range (i.e., insulin resistance and thyroid deficiency). For additional information about strategies for supporting adrenal health and reducing stressors, 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; "The End of Stress As We Know It" by Bruce McEwen.

Estradiol (blood spot) is within the low-norml range for a postmenopausal woman, but much lower than the range seen in premenopausal women or postmenopausal women on physiological estrogen dosing. Self-reported symptoms indicate significant estrogen deficiency (e.g. hot flashes, night sweats, vaginal dryness, sleep disturbances). Other symptoms are characteristic of estrogen excess, which may be due to erratic fluctuations in the levels of estrogens or recent discontinuation of estrogen therapy (none listed). Consider estrogen replacement therapy balanced with natural progesterone..

Progesterone (blood spot) is within the expected lower range for a postmenopausal woman. In women NOT supplementing with progesterone the postmenopausal level is expected to be less than 1 ng/ml. In postmenopausal women supplementing with estrogens, progesterone therapy is often helpful in preventing symptoms of estrogen imbalance when the progesterone/estradiol ratio is optimal (100-500).

Testosterone (blood spot) is within the lower end of the normal range for a postmenopausal woman. In females, testosterone level is highest during youth and drops steadily with age. Symptoms are consistent with both androgen deficiency (e.g. low libido, incontinence, vaginal dryness, fatigue, memory lapses, depression, and bone loss) as well as androgen excess (e.g. loss of scalp hair, increased facial/body hair, acne, oily skin and hair, more aggressive behavior). Low androgen symptoms may be exacerbated if estrogens or cortisol are high (excessive estrogens

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## TEST REPORT | Comments continued

down-regulate cellular androgen receptors and increase SHBG, which decreases bioavailability of T; high cortisol down-regulates androgen receptors). Low androgen symptoms may also be caused by hormonal imbalances other than low testosterone, e.g. high estrogen, high progesterone, low thyroid, and high or low cortisol. High androgen symptoms, on the other hand, are more commonly caused by high androgens (testosterone, DHEA, or synthetic androgen therapy), but also by low estrogens and progesterone (magnifies actions of testosterone) or low cortisol (cortisol suppresses androgen synthesis and actions at the cellular level). Testosterone is an anabolic hormone essential for creating energy, maintaining optimal brain function (memory), regulating the immune system, and building and maintaining the integrity of structural tissues such as skin, muscles, and bone. Consider androgen replacement therapy (e.g. testosterone or testosterone precursor DHEA) if symptoms persist.

SHBG is within range, but on the low end of the normal range. SHBG is an indirect index of estrogen interaction with the liver. As the estrogen levels increase there is a proportional increase in SHBG by the liver, which is released into the bloodstream. The ability of estrogens to increase hepatic production of SHBG is also dependent on normal levels of thyroid hormone (T3) and insulin. When T3 is low, due to hypothyroidism, or insulin is high, due to insulin resistance, SHBG synthesis by the liver is impaired. Thus, hypothyroidism (low thyroid) and insulin resistance lead to low SHBG and potential for estrogen dominance (more bioavailable estrogens). When SHBG is low/low-normal range it is worthwhile to evaluate thyroid hormones and insulin.

DHEAS (blood spot) is within range. DHEAS is highest during the late teens to early twenties and then declines progressively with age to the lower levels of the range in healthy men and women. Expect DHEAS to be in the high reference range until the mid-twenties, the mid-range during the thirties to early fifties and in the lower normal range thereafter. Low age-related DHEAS is often associated with low testosterone (DHEA is a testosterone precursor) and symptoms of androgen deficiency (fatigue, depression, low libido, loss of muscle mass, bone loss, memory lapses). Symptoms of androgen deficiency may be caused by low age-related DHEAS. Consider DHEA therapy if DHEA and/or testosterone are lower than age-expected levels.

Free T4 is low-normal and free T3 is low, consistent with symptoms of thyroid deficiency. TSH is low-normal and not reflective of the lower T3 and T4, which can be caused by impaired feedback response to the pituitary. This often is caused by high stress and associated high cortisol or catecholamines (norepinephrine), which suppresses the hypothalamic/pituitary production of TSH and decreases hepatic conversion of T4 to T3. It should be noted that stress is listed as moderate/severe. High cortisol impairs GI absorption of minerals (ie, zinc and selenium) necessary for the liver enzyme, thyroid deiodinase, to convert T4 to T3. Chronic high stress/cortisol also directs hepatic conversion of T4 to bio-inert reverse T3. Thyroid therapy with T4 alone, and in many cases combination T4/T3 therapy often are not effective when stress/cortisol is high due to excessive conversion of T4 to reverse T3, which impedes the cellular actions of bioactive T3. Consider replacement with a thyroid medication that contains both T4 and T3, or T3 alone (slow release). In addition, saliva testing for cortisol, minimally am and pm testing but preferentially 4x throughout the day, is STRONGLY recommended prior to commencing thyroid therapy since normal cortisol levels are essential for normal tissue response to T3. For an excellent review explaining the effects of stress on the intricate interplay of thyroid and steroid hormones please see the following: www.endotext.org/adrenal.

Thyroid peroxidase antibodies (TPO) are low indicating that Hashimoto's thyroiditis is unlikely.

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