# TEST REPORT

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ZRT LABORATORY

**# D2026 05 06 014 B**

**Ordering Provider:**
Getuwell

**Samples Received**
05/06/2026

**Report Date**
05/15/2026

**Samples Collected**
Blood Spot - 05/05/26 12:30

**Patient Name:** Blood Spot LCMS Hormones 7 with SHBG
**Patient Phone Number:**

| Gender | Last Menses | Height | Waist |
|---|---|---|---|
| Female | Unspecified | 5 ft 8 in | Unspecified |
| **DOB** | **Menses Status** | **Weight** | **BMI** |
| 1/1/1960 (66 yrs) | Hysterectomy (ovaries not removed) | 170 lb | 25.8 |

| TEST NAME | RESULTS \| 05/05/26 | RANGE |
|---|---|---|
| **Blood Spot Steroids & Other Analytes (LC-MS/MS)** | | |
| **Estradiol** | 27 L | 32-472 pg/mL topical, SL, troche, vaginal, patch ERT |
| **Estriol** | 4500 H | <40 pg/mL Premeno, Postmeno |
| **Estrone** | <15 | <15-50 pg/mL Topical, SL, troche, vaginal, patch ERT |
| **Progesterone** | 0.9 | 0.5-4.3 ng/mL Oral (100-300mg) |
| **Ratio: Pg/E2** | 33 L | Pg/E2 (bloodspot-optimal 100-500) |
| **Testosterone** | 130 | 29-224 ng/dL Pre/PostMenopausal TRT |
| **DHEAS** | 70 | 17-207 µg/dL |
| **Cortisol** | 7.5 L | 9.1-19.6 µg/dL (morning), 3.3-8.9 (eve/night) |
| **Blood Spot** | | |
| **SHBG** | 75 | 25-104 nmol/L |

***<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

3x/week vaginal Estradiol (compounded) (12 Hours Last Used); oral Progesterone (compounded) (12 Hours Last Used); 3x/week vaginal Testosterone (compounded) (12 Hours Last Used);88mcg oral Levothyroxine (T4) (Pharmaceutical) (10 Hours Last Used);2.5mcg oral Liothyronine (T3) (Pharmaceutical) (10 Hours Last Used);3mg oral Naltrexone (Pharmaceutical) (10 Hours Last Used)





David T. Zava, Ph.D.
Laboratory Director

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

**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 | 35% |
| Estrogen Dominance / Progesterone Deficiency | 27% |
| Low Androgens (DHEA/Testosterone) | 50% |
| High Androgens (DHEA/Testosterone) | 41% |
| Low Cortisol | 56% |
| High Cortisol | 38% |
| Hypometabolism | 38% |
| Metabolic Syndrome | 31% |

| 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 | | | |



The above results and comments are for informational purposes only and are not to be construed as medical advice. Please consult your healthcare practitioner for diagnosis and treatment.

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## Lab Comments

ESTRADIOL (blood spot) is lower than observed range for estrogen replacement therapy, but is within mid-range for a postmenopausal woman. Although the estradiol at time of blood sampling was within the lower range, self-reported symptoms indicate estrogen dominance. This may indicate that the estrogen is excessive and clears rapidly to baseline at time of blood spot collection, or the estrogen is not well balanced with natural progesterone. Consider changing the estrogen delivery and balancing the estradiol with natural progesterone (optimal Pg/E2 ratio: 100-500).

ESTRIOL is much higher than expected for a postmenopausal woman and suggests topical estriol therapy (none indicated) instead of topical estradiol, as reported. Estriol therapy alone has been successfully used to treat vaginal dryness/atrophy. It has very little impact on diminishing vasomotor symptoms (hot flashes and night sweats). For treating vaginal dryness/atrophy, doses of about 0.5 mg of estriol have been used successfully throughout the medical literature for over 60 years. An advantage of estriol over estradiol is that it does not stimulate the growth of breast or uterine epithelial cells unless it is used at doses that exceed about 0.5 mg/day. The very high estriol seen in this patient may suggest dosing is high, or the hands were contaminated with topical estriol prior to finger-stick blood collection.

ESTRONE (blood spot) is low, which is common in postmenopausal women with low estradiol and other estrone precursors (androstenedione, DHEA). Estrone may also be low in postmenopausal women who have discontinued various types (estradiol, DHEA) and delivery (topical, vaginal, troche, pellet) of estrone precursors. In postmenopausal women estrone is the dominant estrogen in the bloodstream. The level of estrone usually exceeds that of estradiol by 2-3 fold as estradiol drops throughout menopause and estrone increases slightly from metabolism of adrenal androgens (DHEA and androstenedione) and estradiol.
Very little estrone is formed with low-dose topical or transdermal (patch) estradiol therapy. Estrone is not increased with estriol therapy; estrone is a precursor to estriol, but estriol does not back-metabolize to estrone or estradiol. Estriol therapy does not contribute to higher levels of estrone.
Estradiol is metabolized to estrone by the enzyme 17BHSD2, which is increased in estrogen target tissues (e.g. liver, breasts, uterus) by progesterone. 17BHSD2 is very high in the liver where progesterone, taken orally or as a sublingual/troche and swallowed, accelerates hepatic metabolism of estradiol to estrone. Estrone is metabolized mostly by phase II sulfatase and glucuronyl transferase to the more stable forms of estrone sulfate and estrone glucuronide, respectively. This helps prevent excessive buildup of estradiol in the bloodstream and in estrogen target tissues such as the breasts and uterus (Bolun, Semin Reprod Med, 2010; Hilborn, Oncotarget, 2017; Martel, J Ster Biochem Mol Bio, 1992).



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PROGESTERONE (blood spot) is within low-normal range for oral progesterone therapy. Oral progesterone results in a rapid increase (30 min-1 hr) and peak in blood progesterone followed by a precipitous decrease to near baseline within 8-24 hr. Peak levels of progesterone are usually within the luteal range or higher for a short time interval but drop rapidly as progesterone is metabolized and removed from the bloodstream and enters target tissues. Clinical research has shown that oral progesterone in the 100-300 mg range is adequate to counter the growth-promoting actions of physiological levels of estradiol from endogenous production or exogenous estrogen therapy. With oral progesterone delivery blood spot collection should be performed at 8-12 hr following supplementation as the lower range is based on the shorter time course. The progesterone/estradiol ratio is expected to be much lower with oral progesterone therapy when progesterone is tested. relative to estradiol, at the 8-12 hr time interval.

TESTOSTERONE (blood spot) is within expected reference range with testosterone therapy. Physiological doses of testosterone in the 0.3-0.5 mg range, delivered through the skin (topical), mucosa (vaginal, troche/sublingual), or released slowly from an im-sc injection or pellet, usually result in blood spot testosterone levels in the physiological range of a young premenopausal woman at 12-24 hr post supplementation (ZRT database). Higher dosing, or a shorter time course (< 12 hr) from last use of a topical or troche/sublingual delivery will often result in a testosterone level higher than physiological range observed in healthy premenopausal woman, but within the expected, and higher, reference range for testosterone therapy. Im-sc injection or sc pellet inserts deliver a steadier state level of testosterone usually over 1-2 weeks, or 3-4 months, respectively. When applied to the skin (topical, patch) or mucosa (sublingual, troche, vaginal), testosterone levels usually peak at about 1-6 hr and may reach levels 5-10 times higher than range at this time interval. With physiological dosing, levels are usually within range at 12-24 hr. Excessive and prolonged exposure to levels of testosterone exceeding the physiological range are more likely to lead to high androgen symptoms such as loss of scalp hair, increased facial/body hair, acne, oily skin, agitation-irritability, sleep disturbances, and more weight gain in the waist. These high-androgen side effects are usually less pronounced when co-supplementing with estrogens or progesterone as both are weak anti-androgens. Symptoms of both androgen deficiency and excess are self-reported with testosterone therapy. High androgen symptoms are caused by testosterone therapy whereas putative low androgen symptoms in the presence of normal testosterone levels more likely are caused by other hormonal imbalances (e.g. low or high cortisol, and/or low thyroid). Evaluation of these hormones is worth considering.

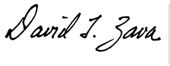
DHEAS (blood spot) is within range. DHEAS is highest during the late teens to early twenties and then declines progressively with age to the lower levels of the range in healthy men and women. Expect DHEAS to be in the high reference range until the mid-twenties, the mid-range during the thirties to 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.

CORTISOL (blood spot-first morning) is lower than range, suggesting HPA axis dysfunction under a period of reported stress. A lower blood cortisol can also result from the use of synthetic cortisol analogues (glucocorticoids) used to treat allergies, inflammation, and asthma. These synthetic cortisol analogues interact with glucocorticoid receptors in target tissues similar to cortisol itself, but are not recognized by the antibodies used to test cortisol. This results in a low cortisol level, but a normal or high glucocorticoid activity. In addition to not being recognized by the test for cortisol, the synthetic derivatives suppress endogenous adrenal cortisol synthesis.. A daily output of cortisol by the adrenal glands (or supplementation with cortisol itself or synthetic analogues) is essential to maintain normal metabolic activity, help regulate steady state glucose levels (important for brain function and energy production), and optimize immune function. Chronic low cortisol production can lead to symptoms of fatigue, allergies (immune dysfunction), chemical sensitivity, cold body temp, and sugar craving. Assuming no cortisol analogues are being used, low adrenal output of cortisol is most commonly caused by chronic stressors which include: psychological stress (emotional), sleep deprivation, poor diet (low protein-particularly problematic in vegetarians), nutrient deficiencies (particularly low vitamins C and B5), physical insults (surgery, injury), diseases (cancer, diabetes), chemical exposure (environmental pollutants, excessive medications), low levels of cortisol precursors (pregnenolone and progesterone) and pathogenic infections (bacteria, viruses and fungi). 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., PhD, "The Cortisol Connection", by Shawn Talbott, Ph.D., "The End of Stress As We Know It" by Bruce McEwen, and "Awakening Athena" by Kenna Stephenson, MD.

SHBG is within mid-normal range. The SHBG level is a relative index of overall exposure to all forms of estrogens (endogenous, pharmaceutical, xeno-estrogens). As the estrogen levels increase in the bloodstream there is a proportional increase in hepatic production of SHBG. Thyroid hormone and insulin also play a role in regulating hepatic SHBG synthesis. Thyroid hormone synergizes with estrogen to increase SHBG production while insulin, in excess (caused by insulin resistance) decreases SHBG synthesis. Thus, in individuals with thyroid deficiency and insulin resistance the SHBG level is usually low. SHBG is an important estradiol and testosterone binding globulin that help increase the half life of these hormones in the bloodstream, and also limit their bioavailability to target tissues. SHBG binds tightly to testosterone and its more potent metabolite dihydrotestosterone (DHT). It also binds tightly to estradiol, the most potent of the endogenous estrogens, but about 5 times weaker than to testosterone and DHT. Thus an increase in SHBG results in proportionately less bioavailable testosterone than estradiol.







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