



<b>Test #</b>	<b>Received</b>	<b>Practitioner Name</b>	Nordic Clinic
<b>Patient #</b>	<b>Tested</b>	<b>Practitioner Address</b>	Nygade 6, 3.sal 1164 Copenhagen Denmark
<b>TST #</b>	<b>Collected</b>		
<b>Patient Name</b>			
<b>Sex</b> Female	<b>Age</b> 62	<b>DOB</b>	

**Menses Status:** Postmenopausal  
**Gender:** Female

**Last Menses:** Unspecified  
**DOB:**

**BMI:** 31.6  
**Height:** 5 ft 6 in  
**Weight:** 196 lb  
**Waist:** Unspecified

Test Name	Result		Units	Range
Estradiol (Blood Spot)	<10		pg/mL	<10-49 Postmenopausal
Progesterone (Blood Spot)	0.2		ng/mL	<0.1-0.8 Postmenopausal
Ratio: Pg/E2 (Blood Spot)	22	L		Pg/E2 (bloodspot-optimal 100-500)
Testosterone (Blood Spot)	<10	L	ng/dL	10-45 Postmenopausal
DHEAS (Blood Spot)	45		ug/dL	40-290
Cortisol (Blood Spot)	8.6		ug/dL	8.5-19.8 (morning), 3.3-8.5 (evening/night)
SHBG (Blood Spot)	75		nmol/L	15-120
Free T4 (Blood Spot)	2.5		ng/dL	0.7-2.5
Free T3 (Blood Spot)	3.7		pg/mL	2.5-6.5
TSH (Blood Spot)	3		µU/mL	0.5-3.0
TPO (Blood Spot)*	350	H	IU/mL	0-150 (70-150 borderline)

\*For research purposes only.

**Therapies**  
None

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**Female Blood Profile II**

**Lab Comments**

Estradiol is within the lower range (<30 pg/ml) that often precipitates symptoms of estrogen deficiency (most common are hot flashes, night sweats, vaginal dryness, sleep disturbances). Some of these symptoms are self-reported. Consider estrogen replacement therapy (contraindicated with cancers of the breast or uterus) in combination 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 low, consistent with symptoms of low androgens (e.g., low libido, incontinence, vaginal dryness, fatigue, memory lapses, depression, and bone loss). The testosterone in the blood spot assay represents the level in whole blood (includes all blood cells that also carry hormones to target tissues), which is very similar to serum or plasma levels in patients not supplementing with testosterone. 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. Low serum testosterone has been correlated with low bone mass in both perimenopausal and postmenopausal women (Oronzo et al. Eur J Epidemiology 16: 907-912, 2000; Slemenda et al. J Clin Invest 97: 14-21, 1996). Low androgens have also been correlated with a higher prevalence of autoimmune problems such as lupus and rheumatoid arthritis (Masi AT. Clin Exp Rheumatol 1995; 13(2):227-240). Because the blood testosterone level is low, it would be worthwhile to evaluate bone density periodically (yearly) and to consider androgen supplementation to prevent long term health issues, particularly osteoporosis and increased fracture risk.

DHEAS (blood spot) is within low-normal 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. DHEAS is expected to be within the lower range in older individuals, which likely accounts for the low-normal DHEAS level in this patient. Low DHEAS is often associated with low testosterone (DHEA is a testosterone precursor) and symptoms of androgen deficiency (fatigue, depression, vaginal dryness, low libido, loss of muscle mass, bone loss, memory lapses). Self-reported symptoms indicate androgen deficiency, consistent with low DHEAS. Consider DHEA therapy if cortisol is within normal range. DHEA therapy can cause a transient suppression of cortisol and exacerbate symptoms of cortisol deficiency if cortisol is low.

Morning cortisol (blood spot) is low-normal, suggesting adrenal fatigue. Self-reported symptoms are also consistent with adrenal fatigue. A daily output of cortisol is essential to maintain normal metabolic activity, help regulate steady state glucose levels (important for brain function and energy production), and optimize immune function. Low cortisol production is consistent with symptoms of fatigue, allergies (immune dysfunction), chemical sensitivity, cold body temp, and sugar craving. Adrenal insufficiency is most commonly caused by 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 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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Free T4 is within normal range but symptoms of thyroid deficiency are self-reported. This usually is due to poor conversion of T4 to T3.

Free T3 is within normal range but symptoms indicate thyroid deficiency. A normal T3 does not exclude the possibility of a "functional" thyroid deficiency caused by other hormonal imbalances such as excess estrogen, low progesterone, low testosterone, low or high cortisol, and low growth hormone (IGF-1). Testing for these hormones is recommended.

TSH is within normal range; however, symptoms suggest thyroid deficiency. A normal TSH does not exclude thyroid deficiency, particularly when stress hormones (cortisol or catecholamines) are elevated (suggest testing salivary cortisol). When stress hormones are high a low level of thyroid hormone (T3) is less likely to stimulate pituitary TSH synthesis (see:

[www.endotext.org/adrenal/adrenal8/adrenalframe8.htm](http://www.endotext.org/adrenal/adrenal8/adrenalframe8.htm)).

Thyroid peroxidase (TPO) antibodies are elevated indicating autoimmune thyroiditis, often referred to as Hashimoto's thyroiditis. This condition is associated with elevated circulating antibodies to thyroid peroxidase, the enzyme found within the thyroid gland responsible for manufacturing thyroid hormones (T4, T3). When the level of TPO antibodies is elevated this can lead to destruction of the thyroid gland and acute release of high levels of thyroid hormones T4 and T3. Continued autoimmune destruction of the thyroid gland eventually results in fibrosis and depletion of the thyroid hormones from the thyroid gland, thus causing an eventual hypothyroid state. Individuals with autoimmune thyroiditis can suffer from symptoms of both thyroid excess and deficiency, depending on the state of the disease (ie, autoimmune attack on the thyroid and hyperthyroidism or post-attack and hypothyroidism). Clinical studies show that selenium supplementation is helpful in decreasing autoimmune destruction of the thyroid gland (Duntas et al. Eur J Endocrinology 148: 389-393, 2003). Normal cortisol levels produced by the adrenal glands help to subdue the autoimmune attack on the thyroid gland. However, if the adrenals are exhausted, resultant low cortisol can lead to intensified autoimmune destruction of the thyroid gland, often referred to as a "thyroid storm". Thus, in addition to selenium supplementation, testing of salivary cortisol levels, stress reduction, and adrenal support should be considered as important components of the treatment strategy for Hashimoto's autoimmune thyroiditis.