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Collection Date First: August 02, 2012  
 Collection Date Last: August 31, 2012  
 Sample Received: September 07, 2012  
 Reported On: September 25, 2012

**Month Long Hormone Assessment**

**Accession Number : FFFFFF**

**Healthcare Professional:**

**Client:**

**Age:** 49

**DOB:**

**Gender:** F

**Status:** No menses

**Health #:**

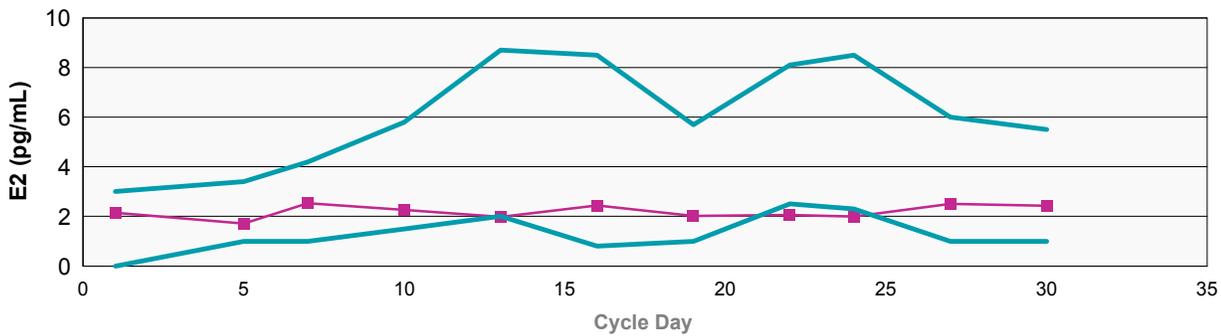
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Phone:

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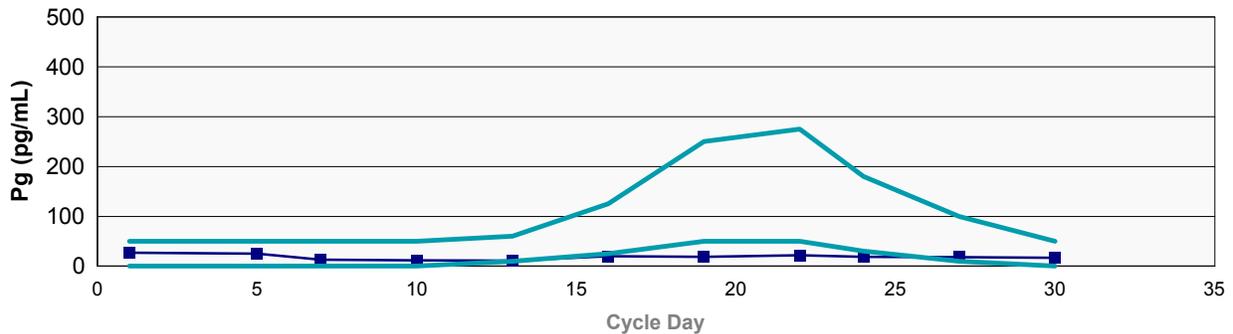
**Estradiol**

Legend ■ Estradiol ■ Normal Range (Upper and lower limits)



**Progesterone**

Legend ■ Progesterone ■ Normal Range (Upper and lower limits)

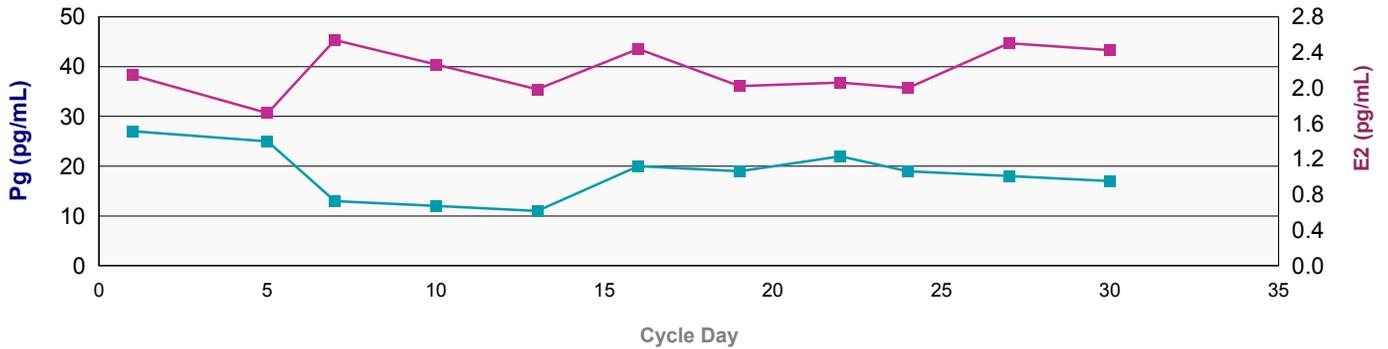


*George Gillson*  
 George Gillson MD, PhD  
 Medical Director

Co-Signing Physician:  
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### Progesterone & Estradiol

Legend ■ Estradiol ■ Progesterone



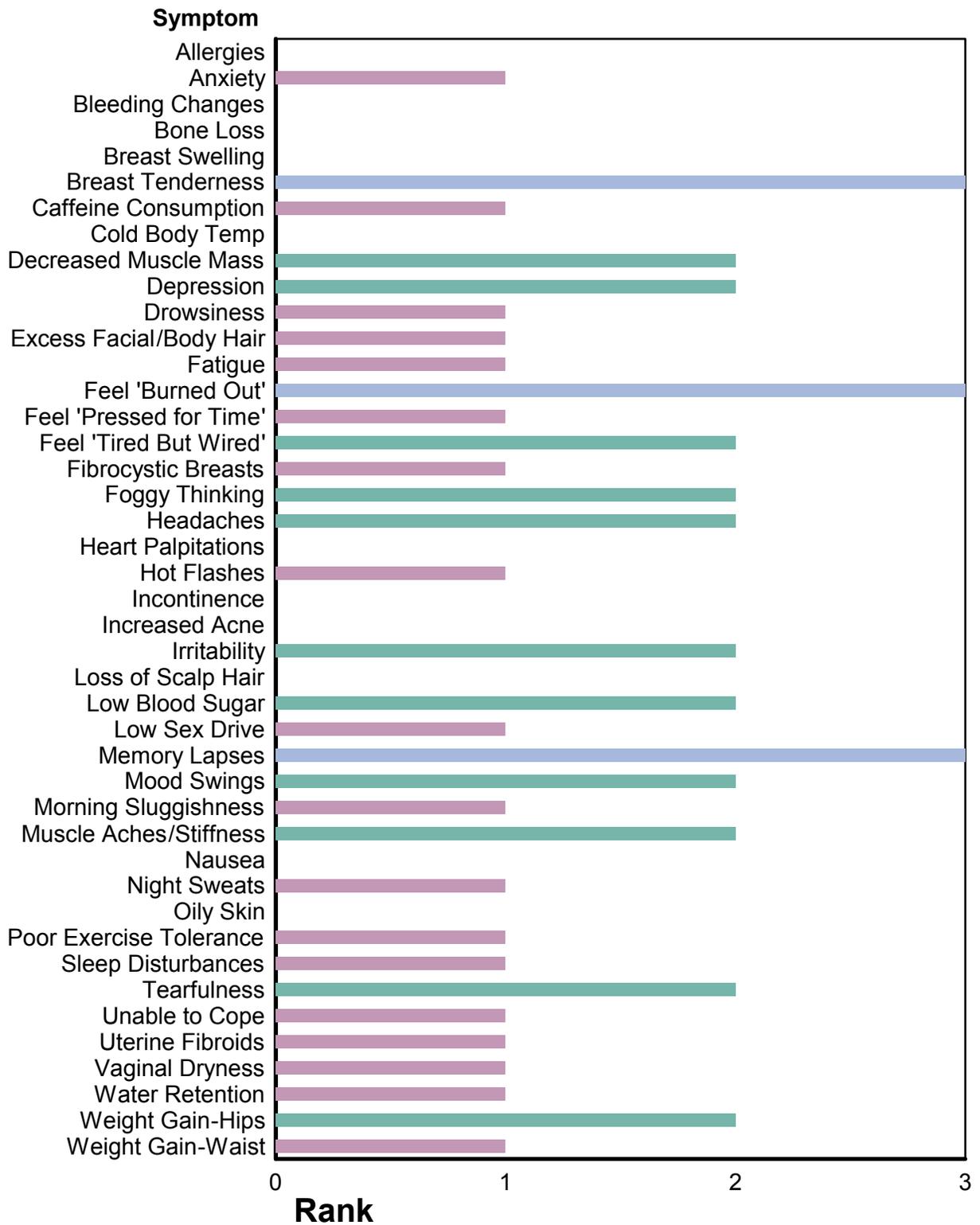
Sample #	1	2	3	4	5	6	7	8	9	10	11
Progesterone pg/mL	27	25	13	12	11	20	19	22	19	18	17
Estradiol pg/mL	2.1	1.7	2.5	2.3	2.0	2.4	2.0	2.1	2.0	2.5	2.4
Pg/E2 Ratio	12.6	14.6	5.1	5.3	5.5	8.2	9.4	10.7	9.5	7.2	7.0

Reference Ranges		
	Follicular	Mid luteal
E2	1 - 5 pg/mL	1 - 9 pg/mL
Pg	<50 pg/ml	50 - 250 pg/ml
Pg/E2	3 - 14	23 - 57

### Testosterone - average

Hormone	Status	Result	Range	Units	Range Applied
Pooled Testosterone	Within range	30	15 - 45	pg/mL	Pooled endogenous testosterone > 30 yrs

Collection Times		
Sample	Date	Time
1	August 02, 2012	7:50 am
2	August 02, 2012	
3	August 06, 2012	7:40 am
4	August 08, 2012	8:15 am
5	August 11, 2012	6:42 am
6	August 14, 2012	7:30 am
7	August 17, 2012	6:55 am
8	August 20, 2012	6:25 am
9	August 23, 2012	6:45 am
10	August 25, 2012	6:30 am
11	August 28, 2012	8:32 am
12	August 31, 2012	5:49 am



\* Indicates symptom left blank.

The patient indicates some problems with sleep. Recognize that there is an optimum range for bedtime cortisol. If bedtime cortisol is too low, there is some evidence that normal sleep architecture is not established (insufficient REM sleep). Conversely, high bedtime cortisol is suppressive for melatonin, and may result in difficulty initiating and sustaining sleep. It may be worth looking at the cortisol profile via saliva, depending on the severity of the situation.

This patient's Body Mass Index (BMI) is 31. Patients with a BMI between 25 and 30 are defined as overweight and obesity is defined as BMI greater than 30. Elevated BMI is often associated with elevated estradiol, testosterone and DHEAS, and may also be associated with high morning cortisol. Metabolic syndrome/insulin resistance are also associated with elevated BMI. Health risks associated with excess weight include increased risk of diabetes, high blood pressure, heart attack, stroke and cancer. Weight reduction and reduced consumption of refined carbohydrates may be worthwhile interventions for some patients with elevated BMI. Note that not all patients with high BMI have metabolic abnormalities. For example, a well-muscled individual may have a BMI in excess of 30, yet be metabolically normal. Note also that not all patients with high androgens have elevated BMI.

Strictly speaking, vasomotor symptoms including hot flashes and night sweats reflect sympathetic nervous system (SNS) instability. Hence these symptoms are dependent on many factors such as stress, brain chemical levels (T3, serotonin, norepinephrine, melatonin, GABA, progesterone, estradiol and cortisol), and HPA axis function. They are not "pure" symptoms of estrogen deficiency (Prior J. *Endocrine Rev* 1998;19:397-428), and in fact, these symptoms may co-exist with symptoms of estrogen dominance. Vasomotor symptoms can be seen with many different patterns of hormone imbalance, such as low progesterone, low testosterone, low or high DHEAS, high estradiol, high cortisol. (Note: A one year trial of progesterone cream demonstrated efficacy compared to placebo, for the control of vasomotor symptoms (Leonetti HB, Longo S, Anasti JN. *Transdermal progesterone cream for vasomotor symptoms and postmenopausal bone loss. Obstet Gynecol.* 1999 Aug;94(2):225-228.) Excessive use of progesterone (higher dose or unbroken daily usage) can also result in vasomotor symptoms by downregulation of estradiol receptors.

Breast tenderness is listed as a complaint for this patient. Diffuse breast tenderness usually indicates an estrogen-progesterone imbalance, with high estradiol or low progesterone. Breast tenderness in the upper outer quadrants, toward the axillae, can be caused by too much progesterone relative to estrogen. For example, breast tenderness is often seen in women who have low salivary estradiol. When both estradiol and progesterone are low, the mechanism for breast tenderness is not well understood. Caffeine and cortisol play a role. Low testosterone may also be a factor; research indicates that testosterone plays a role in balancing the proliferation-inducing effect of estradiol. (Testosterone inhibits estrogen/progestogen-induced breast cell proliferation in postmenopausal women. Hofling et al. *Menopause* 2007; 14:1-8.)

October 2009 results of analysis of the Women's Health Initiative Study (whose participants received oral equine estrogen and oral medroxyprogesterone acetate) indicate that the women on this combination of therapies who had breast tenderness were more likely to develop breast cancer over the course of the study. (New-Onset Breast Tenderness After Initiation of Estrogen Plus Progestin Therapy and Breast Cancer Risk. Crandall CJ, et al. *Arch Intern Med* 2009;169(18):1684-1691. ) We do not know if this association can be extrapolated to women using human identical hormone combinations, nor do we know whether breast tenderness in the absence of hormone therapy has any negative connotation.\*

Symptoms commonly associated with hypothyroidism are present. These may include cold intolerance/feeling cold all the time, depression, weight gain, fatigue, headaches, thinning hair, and aching muscles, although not all these symptoms will be present in every individual. Other symptoms (not inventoried here) can include constipation, dry skin and muscle cramps. (Baiser W, Hertoghe J, Eeckhaut W. *J Nutritional Env Med* 2000;10:105-113.)

Note that these symptoms may be present in the face of normal thyroid studies including TSH. This is known as a functional deficiency state in which the blood level of a hormone is normal, but the action of the hormone at the tissue level is being blocked by other hormone imbalances. For example, unopposed/insufficiently opposed estrogen replacement (especially oral estrogen) and excessive estrogen dosing are common causes of hypothyroid symptoms. High cortisol can oppose the action of T3 at the tissue level, whereas low cortisol can fail

to potentiate the tissue action of T3, even if there is sufficient T3. Many individuals with low cortisol complain of hypothyroid symptoms.

Note also that hypothyroid symptoms may persist despite supplementation with T4 (Synthroid, Eltroxin, L-throxine). In this situation, conversion of T4 to T3 (the more active form) may be blocked by high cortisol, heavy metal toxicity or deficiencies of nutrients including selenium, chromium and zinc.

Some insight into hypothyroid symptoms in the face of normal serum thyroid testing might be had by assessing T3, T4 and selenium in a 24 hour urine specimen. For more information on this test, please contact the laboratory at 866 370 5227.

Symptoms of low cortisol/adrenal fatigue are present. These may include fatigue (especially morning fatigue), difficulty getting going in the morning, feeling flat or "burned out", excessive use of caffeine, depression, allergies, decreased exercise tolerance, feeling cold/cold hands and feet. In women, low cortisol may present with increased facial hair growth. Note that not all of these symptoms are present in every individual with adrenal fatigue. Consideration might be given to additional assessment of adrenal function via 4 point cortisol testing in saliva.

The average progesterone level throughout the month is below 90 pg/ml. It is important to recognize that even though the measured progesterone level may be "normal" for age or circumstance, it may still be suboptimal for any given individual. For example, it is well-recognized that a suboptimal amount of progesterone may lead to a diminished ability of the tissue to respond appropriately to estrogens as well as thyroid hormone. In general, regularly cycling women who do not have any hormonal complaints have a progesterone level above 90 pg/mL when sampled in the middle of the luteal phase. A level below 90 pg/mL might prompt consideration of supplementation. Also, in cases where supplemental estrogen is used, it is common practice to supplement with progesterone, regardless of the endogenous progesterone level, and regardless of whether the patient has had a hysterectomy. A one year, placebo-controlled, randomized trial has demonstrated that topical progesterone is effective for relief of vasomotor symptoms in early menopause. (Leonetti HB, Longo S, Anasti JN. Transdermal progesterone cream for vasomotor symptoms and postmenopausal bone loss. *Obstet Gynecol.* 1999 Aug;94(2):225-228). These women derived benefit from supplementation with progesterone, yet they generally had levels of progesterone which were normal for age, i.e. < 90 pg/mL. Again, the progesterone level in any given situation might be normal for that circumstance, but still not optimal. Ultimately however, treatment decisions must be made by the health care practitioner(s) involved in this patient's care.

The pooled testosterone value is 30 pg/ml and is within normal limits. (Range for cycling women is 15 to 50 pg/ml).

Regularly cycling women sampled in the midluteal phase generally have a ratio of progesterone to estradiol (Pg/E2) between 23 and 57. The ratio Pg/E2 stays at or below the low end of this range for this patient, throughout the collection. A lower ratio may indicate low progesterone relative to normal estradiol levels, or elevated estradiol levels relative to a normal progesterone surge.



George Gillson MD, PhD  
Medical Director

Note: The College of Physicians and Surgeons of Alberta considers saliva hormone testing and some forms of bio-identical hormone replacement to be complementary medicine. The interpretation comments have not been evaluated or approved by any regulatory body. Commentary is provided to clinicians for educational purposes and should not be interpreted as diagnostic or treatment recommendations. \*General treatment suggestions can be found in the Rocky Mountain Analytical Resource Binder.