

WEIGHT MANAGEMENT PROFILE

Weight Management Profile

Estradiol (E2)
 Progesterone (Pg)
 Testosterone (T)
 DHEA-S (DS)
 Diurnal Cortisol (Cx4)
 Thyroid Stimulating Hormone (TSH)
 Vitamin D (D2, D3)
 Insulin (In)
 Hemoglobin A1c (HbA1c)

***OPTIONAL** profiles available at a discounted price when ordered with Weight Management Profile.

*OPTIONAL THYROID

Free Triiodothyronine (fT3)
 Free Thyroxine (fT4)
 Thyroid Peroxidase Antibodies (TPO)

Add on: when symptoms of thyroid deficiency are problematic

Advantage: better estimation of thyroid hormone bioavailability to facilitate effective thyroid therapy

*OPTIONAL CARDIO

High Sensitivity C-Reactive Protein (hsCRP)
 Triglycerides (TG)
 Total Cholesterol (CH)
 LDL Cholesterol (LDL)
 VLDL Cholesterol (VLDL)
 HDL Cholesterol (HDL)

Add on: with abdominal obesity, and symptoms of insulin resistance/ metabolic syndrome

Advantage: early detection of pro-inflammatory CVD risks, metabolic syndrome, and pre-diabetes

An Epidemic

In 2010 the US was ranked as the world's fattest developed nation, and the latest data out of the CDC reports over a third of American adults obese—with the highest prevalence among men and women over 40 years old. It is not a coincidence that this is the age when people start to see the impacts of hormonal imbalance. Providers can help by addressing the hormonal connection to obesity to help patients manage their weight and reduce related disease risks.

ZRT's Weight Management Profile

The Weight Management Profile identifies hormonal imbalances that contribute to obesity, weight gain and difficulty losing or sustaining a healthy weight. Used as a screening tool, the profile also serves as a powerful early indicator of insulin resistance and risks for metabolic syndrome and diabetes.

Purpose

- ▶ Identify hormonal imbalances associated with weight gain and obesity
- ▶ Detect risk markers for insulin resistance, metabolic syndrome and diabetes

What is Included in the Profile?

Estradiol (E2) at optimal physiological levels in women promotes a healthy distribution of fat in hips, thighs, breasts, and subcutaneously. However, in excess, and in the absence of progesterone, estrogen predisposes to unhealthy surplus weight gain in these tissues. Men generally have much lower levels of estradiol and higher testosterone, which is responsible for greater muscle mass and less fat distribution in areas of the body normally seen in women. In overweight men testosterone levels drop and estrogens rise leading to the same problematic weight gain in the hips, thighs, and breasts (referred to as gynecomastia) as seen in women.

Progesterone (Pg) in addition to its primary role in attenuating the effects of excess estrogen in the body by downregulating estrogen receptors, aids weight management by acting as a natural diuretic. Its natural calming effects in the brain may also reduce stress-related overeating and food cravings. As a mineralocorticoid receptor antagonist, progesterone counteracts the effects of mineralocorticoid activation, which include the stimulation of fat cell formation, increased body weight, and release of inflammatory cytokines. However,

Saliva and Dried Blood Spot Testing.

Minimally-invasive home test kit.

excessive supplementation with progesterone to higher than normal levels can increase appetite and also slow the rate of food emptying from the stomach and moving through the digestive tract, causing slower digestion and bloating.

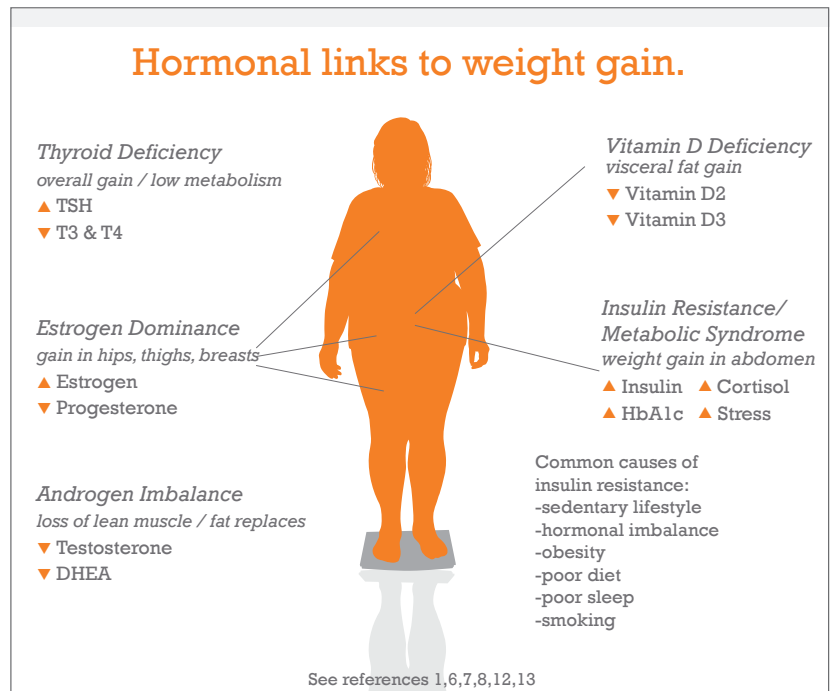
Testosterone (T) and DHEA-S (DS) are androgens that increase lean muscle mass and metabolic rate. As androgen levels decline, muscle mass also decreases with a corresponding increase in adiposity. Low androgens can also reduce vitality and tolerance for exercise. Weight gain itself, with its resulting hormone imbalances, can trigger a drop in testosterone as the aromatase enzyme within fat tissue converts androgens to estrogens. In men this contributes to a female-type body fat distribution, including breast tissue development. In women with polycystic ovarian syndrome (PCOS), high testosterone and DHEA are linked to insulin resistance and weight gain, particularly in the abdomen.

Cortisol (C) imbalances can create problems with blood sugar control, sleep patterns, appetite, food cravings, and tolerance exercise. Under stress, excessive cortisol production particularly in concert with insulin, promotes fat storage in abdominal adipose tissue. This visceral type of fat is closely associated with insulin resistance and metabolic syndrome and thus more hazardous to health. Chronically elevated cortisol is a known risk factor for pre-diabetes and cardiovascular disease.

Thyroid Stimulating Hormone (TSH) elevations, even within the high-normal range, are linked with hypothyroidism, low metabolic rate and obesity. Hypothyroidism is linked to elevated cortisol and can also be a consequence of oral estrogen therapy, which increases the production of binding proteins that reduce thyroid hormone bioavailability.

Vitamin D (D2, D3) deficiency is common in obesity and particularly associated with hyperinsulinemia and visceral fat. Whether by cause or effect, identifying and correcting vitamin D3 deficiency may improve insulin sensitivity.

Fasting Insulin (In), when elevated, is a marker of insulin resistance which precedes metabolic syndrome, PCOS, and type 2 diabetes. Increased levels, particularly in concert with cortisol lead to central obesity and increased inflammatory and other cardiovascular disease markers. Hyperinsulinemia also contributes to decreased testosterone levels in men, but increased testosterone and decreased ovulation in women.



Hemoglobin A1c (HbA1c) is an indirect measure of the average circulating glucose levels over the previous three months. An HbA1c of more than 6% is predictive of type 2 diabetes and cardiovascular disease risk.

Hormone Weight Gain Connection

Estrogen/progesterone imbalance: weight gain in hips, thighs; water retention; low thyroid/metabolism

Testosterone/DHEA imbalance: decreased lean muscle, low metabolic rate; abdominal obesity

Cortisol imbalance: increased appetite, sugar cravings, and belly fat; inhibits thyroid and metabolism

Vitamin D3 deficiency: hyperinsulinemia; visceral fat

TSH elevated: hypothyroidism, low metabolic rate, obesity

Fasting Insulin: insulin resistance, abdominal obesity

HbA1c: predictive of type 2 diabetes

Clinical Utility

The Weight Management Profile allows providers to identify specific hormone imbalances associated with excess weight gain or obesity, vitamin D deficiency, and hypothyroidism in their patients. As a risk assessment panel it allows for early detection of insulin resistance, metabolic syndrome, and type 2 diabetes. The comprehensive test report is designed to help clinicians recommend effective treatments to rebalance hormone levels, address vitamin D and thyroid deficiencies, reduce overall risk for metabolic syndrome, and potentially avoid the onset of type 2 diabetes.

Who Benefits from Profile Testing?

Menopausal women/andropausal men with unexplained weight gain, obesity, abdominal fat, high BMI (body mass index), hypometabolism. Commonly related symptoms include loss of lean muscle, increased appetite and/or sugar cravings, chronic stress, and low thyroid symptoms.

Advantages of Saliva and Blood Spot Testing

- Convenient sample collection at home - no phlebotomist required
- Easy shipment of samples from home to the lab
- Samples stable for several weeks at room temperature
- Excellent correlation with serum/plasma assays

References

1. Björntorp P. The regulation of adipose tissue distribution in humans. *Int J Obes Relat Metab Disord*. 1996;20(4):291-302.
2. McTiernan A, Wu L, Chen C, et al. Women's Health Initiative Investigators. Relation of BMI and physical activity to sex hormones in postmenopausal women. *Obesity (Silver Spring)*. 2006;14(9):1662-77.
3. Pasquali R, Vicennati V, Gambineri A, Pagotto U. Sex-dependent role of glucocorticoids and androgens in the pathophysiology of human obesity. *Int J Obes (Lond)*. 2008;32(12):1764-79.
4. Shi H, Seeley RJ, Clegg DJ. Sexual differences in the control of energy homeostasis. *Front Neuroendocrinol*. 2009;30(3):396-404.
5. Torr ns JI, Sutton-Tyrrell K, Zhao X, et al. Relative androgen excess during the menopausal transition predicts incident metabolic syndrome in midlife women: study of Women's Health Across the Nation. *Menopause*. 2009;16(2):257-64.
6. <http://www.endotext.org/section/obesity/>; Part 12: Endocrine changes in obesity.
7. Knudsen N, Laurberg P, Rasmussen LB, et al. Small differences in thyroid function may be important for body mass index and the occurrence of obesity in the population. *J Clin Endocrinol Metab*. 2005;90(7):4019-24.
8. Cheng S, Massaro JM, Fox CS, et al. Adiposity, cardiometabolic risk, and Vitamin D Status: The Framingham Heart Study. *Diabetes* 2010;59:242-248.
9. Grundy SM, Brewer HB Jr, Cleeman JI, et al; American Heart Association; National Heart, Lung, and Blood Institute. Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Circulation* 2004;109:433-8.
10. Geberhiwot T, Haddon A, Labib M. HbA1c predicts the likelihood of having impaired glucose tolerance in high-risk patients with normal fasting plasma glucose. *Ann Clin Biochem*. 2005;42:193-5.
11. Grant T, Soriano Y, Marantz PR, et al. Community-based screening for cardiovascular disease and diabetes using HbA1c. *Am J Prev Med* 2004;26:271-5.
12. Jones ME, Schoemaker M, Rae M, et al. Changes in estradiol and testosterone levels in postmenopausal women after changes in body mass index. *J. Clin Endocrinol Metab*, 2013;98(7):2967-2974.
13. Yassin A, Doros G. Testosterone therapy in hypogonadal men results in sustained and clinically meaningful weight loss. *Clin Obesity* 2013;3:73-83.
14. Kargi AY, Iacobellis G. Adipose tissue and adrenal glands: novel pathophysiological mechanisms and clinical applications. *Int J Endocrinol*. 2014;2014:614074.

