**Casual Friday Series** 

# FM Case Communication Skills

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## Disclaimer

- Information in this presentation is not intended to diagnose, treat, reverse, cure, or prevent any disease. While this presentation is based on medical literature, findings, and text, The following statements have not been evaluated by the FDA.
- The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.





(Lifestyle + Genetics) x Time = Chronic Health Outcomes



### Case of the Day: Weight Loss

POP – Persistent Organic Pollutants

PCB – Polychlorinated Biphenyls

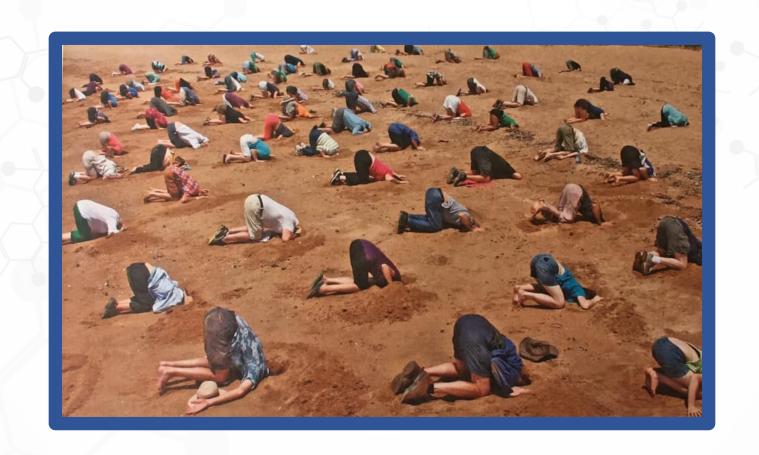
### Fat Soluble Hormones

- Production (Sex and Stress)
- Phyto/Xeno
- THEIR DOCTOR





## Perspectives on Hormone-Induced Obesity





Hum Reprod Update. 2017 May; 23(3): 300-321.

Published online 2017 Mar 2. doi: 10.1093/humupd/dmw045

PMCID: PMC5850121

PMID: 28333235

### Ovarian hormones and obesity

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#### BACKGROUND

Obesity is caused by an imbalance between energy intake, i.e. eating and energy expenditure (EE). Severe obesity is more prevalent in women than men worldwide, and obesity pathophysiology and the resultant obesity-related disease risks differ in women and men. The underlying mechanisms are largely unknown. Pre-clinical and clinical research indicate that ovarian hormones may play a major role.



Brain Res. Author manuscript; available in PMC 2011 Sep 2.

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Brain Res. 2010 Sep 2; 1350: 77-85.

Published online 2010 May 23. doi: 10.1016/j.brainres.2010.04.056

PMCID: PMC2924463

NIHMSID: NIHMS202439

PMID: 20441773

#### Metabolic Impact Of Sex Hormones On Obesity

Lynda M

#### Estrogens Interact with Leptin

Autho

First described in 1994 (Zhang et al., 1994), leptin has proven to be a key metabolic protein with actions throughout the body. Leptin provides a powerful catabolic signal to the brain, resulting in inhibition of food intake and increasing energy expenditure (Ahima et al., 1999; Elias et al., 1999; Elmquist et al., 1999; Schwartz et al., 2000; Woods et al., 2000; Morton et al., 2003; Seeley and Woods, 2003; Balthasar et al., 2004; Schwartz and Porte, 2005). Leptin is secreted from adipose tissue in direct proportion to fat content, and it crosses the blood-brain barrier to interact with leptin receptors in the hypothalamus and brainstem (Tartaglia et al., 1995; Ahima et al., 1996; Seeley et al., 1996; Ahima et al., 1999; Elias et al., 1999; Morton et al., 2003; Schwartz and Porte, 2005). Although there are several splice variants of the leptin receptor, the long form (termed leprb) is the critical variant for regulating energy balance (Chen et al.,

#### Estrogen Regulates Adiposity

Go to: ☑

Go to: ✓

The accumulation of fat centrally (intra-abdominal/visceral adipose tissue) has emerged as a risk factor for the metabolic syndrome (Kannel et al., 1991; Lee et al., 2009). Estrogens promote the accumulation of subcutaneous fat (Krotkiewski et al., 1983); however, visceral fat varies inversely with levels of estrogens (Bouchard et al., 1993). The loss of estrogens with menopause is associated with an increase in intra-abdominal fat accrual (Poehlman et al., 1995; Lee et al., 2009). OVX rats gain fat, specifically visceral fat with no change of subcutaneous fat (Clegg et al., 2006). Furthermore, peripheral or central administration of estradiol-17 $\beta$  to OVX rats changes their body fat distribution to mirror that of intact females. Altering the sex hormone milieu in males with estradiol-17 $\beta$  administration increases subcutaneous fat deposition (Clegg et al., 2006). An important implication from these findings is that estrogens may be critical determinates of body fat distribution.



## Leptin – What is it?

Released by adipocytes (fat) and enterocytes of the small intestine to inhibit hunger and cause fat expenditure.

Your Brain on Leptin: "I'm not hungry, I'll just burn fat."

Your Brain w Leptin Resistance: "I'll have just 1 or 12"

(Melatonin increases leptin in the presence of insulin)

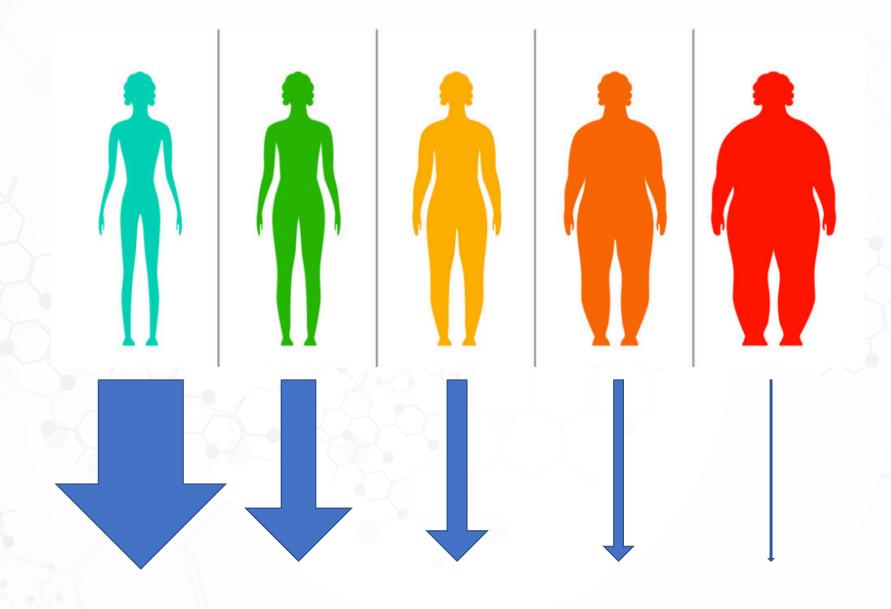


## **Leptin Considerations**

Insulin deficiency = diabetes, type 1 Insulin resistance = diabetes, type 2

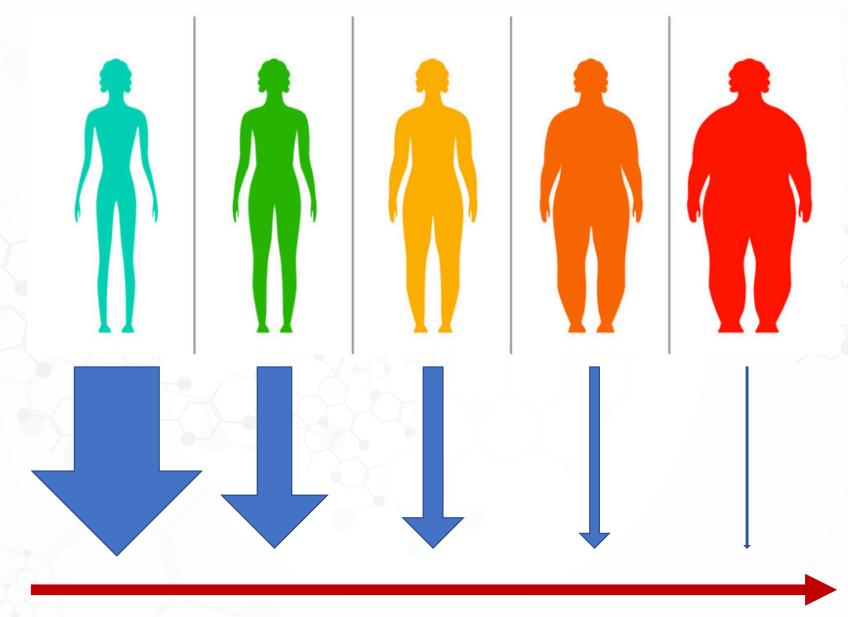
Leptin deficiency = Obesity (1) Leptin resistance = Obesity (2)







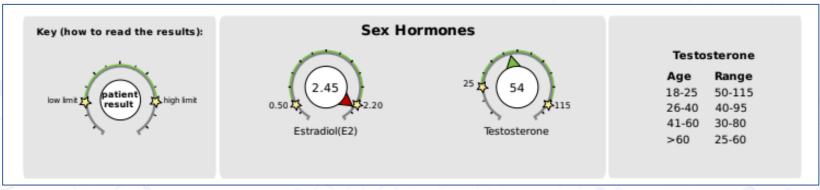
Amount of Hormone Metabolized and Removed





Leptin Production and Resistance Increases with Body Fat Accumulation

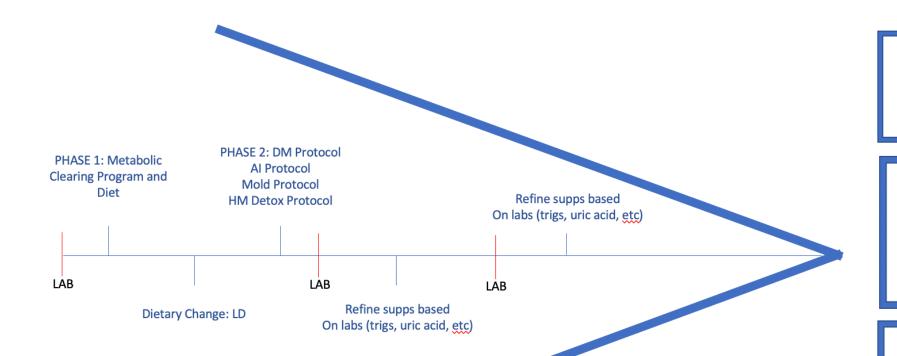
### **DUTCH Test Results**



#### Adrenal Hormones See pages 4 and 5 for a more complete breakdown of adrenal hormones **Total DHEA Production** (6m/6u) **Age** 20-39 Range 1300-3000 750-2000 **Daily Free Cortisol Pattern** 40-60 500-1200 Total DHEA Production (DHEAS + Etiocholanolone + Androsterone) Cortiso 2750 🍕 Low Range Limit Metabolized Cortisol (THF+THE) 24hr Free Cortisol **Patient Values** cortisol (A+B+C+D) (Total Cortisol Production) metabolism Waking (A) Morning (B) Afternoon (C) Night (D) Free cortisol best reflects tissue levels. Metabolized cortisol best reflects total cortisol production.



#### **Supplement and Diet Protocols**



Retest a lab at least every 60 days.

85% of patients will improve with basic structures and healthy eating.

% of problem analysis: this is what the cleanse is for.

Patient's don't finish programs when they don't know where they are.

### Reframe to Refocus:

- Here's where you were...
- Here's where we are...
- Here's where we're going...



Patient's don't finish programs when they can't explain it to their family.

Use the "Kind of Like" strategy:

- Make it personally applicable...
- Involve more than one sense...
- Pretend you're teaching a 5<sup>th</sup> grader...(with respect)



Patient's don't finish programs when they only learn through pictures.

### Reframe to Refocus:

- Here's where you were...
- Here's where we are...
- Here's where we're going...



Patient's don't finish programs when they don't trust their leader.

- You are the product take care of your health.
- Get your ACCT with Evexia Blood Labs.
- "Biogenetix General Screen" 144.

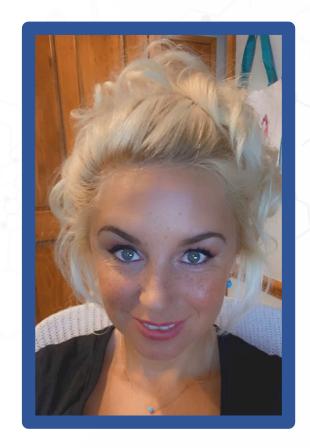
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