Female Pattern Hair Loss

A Biogenetix Clinical Presentation

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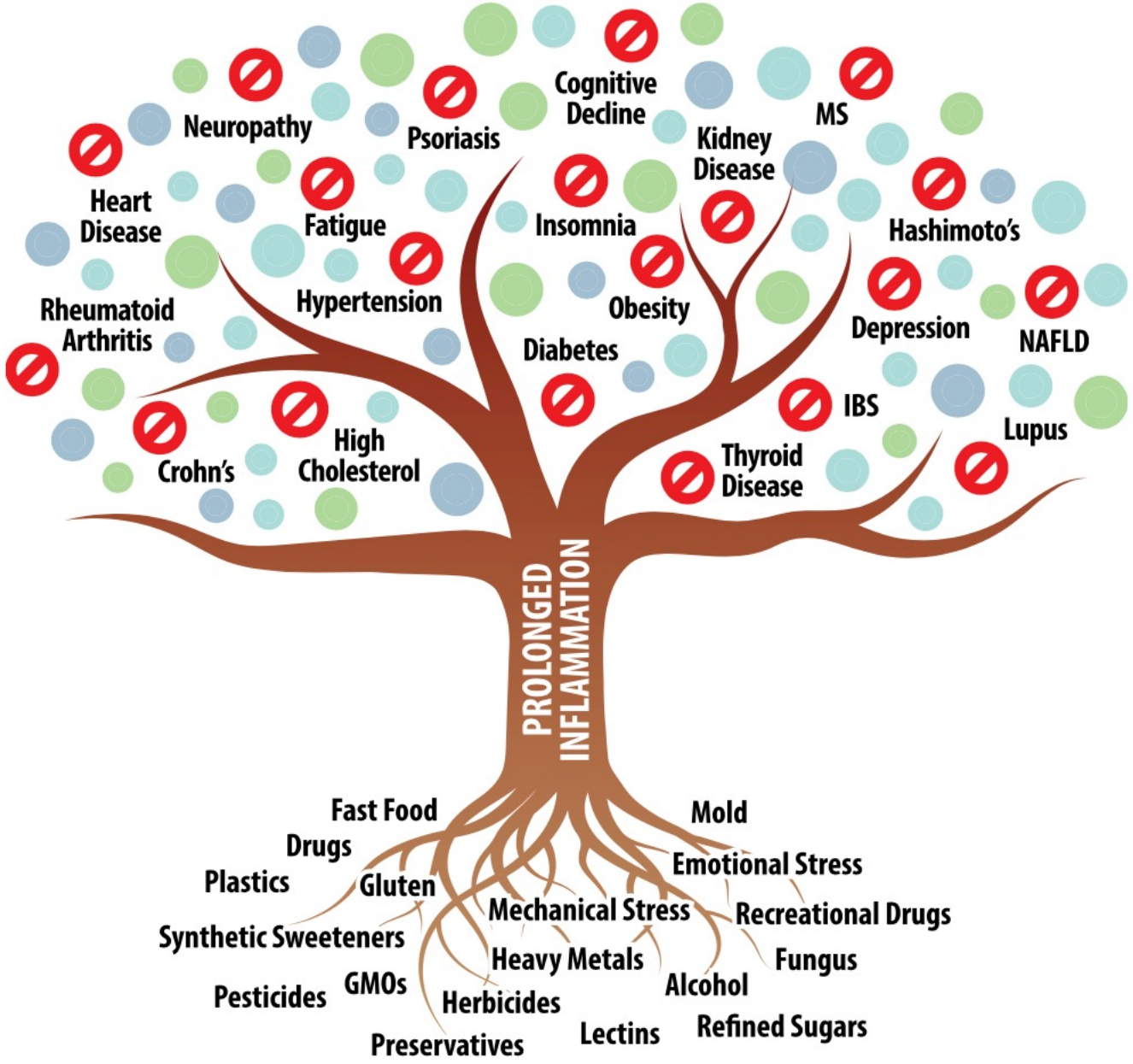
- *The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.*

Epigenetics:

Study of changes in the DNA that do not involve changes in the DNA sequence.


Neuropathy
Psoriasis
Cognitive Decline
MS
Kidney Disease
Heart Disease
Fatigue
Insomnia
Hashimoto's
Hypertension
Obesity
Depression
NAFLD
Rheumatoid Arthritis
Diabetes
IBS
Lupus
Crohn's
High Cholesterol
Thyroid Disease
PROLONGED INFLAMMATION
Fast Food
Mold
Drugs
Emotional Stress
Plastics
Gluten
Mechanical Stress
Recreational Drugs
Synthetic Sweeteners
Heavy Metals
Fungus
Pesticides
GMOs
Alcohol
Herbicides
Lectins
Refined Sugars
Preservatives

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Female pattern hair loss: A clinical, pathophysiologic, and therapeutic review☆☆☆

G. Fabbrocini, M. Cantelli, A. Masarà,* M.C. Annunziata, C. Marasca, and S. Cacciapuoti

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Female pattern hair loss (FPHL) is the most common form of alopecia in women. Affected women may experience psychological distress and impaired social functioning. Early diagnosis and initiation of treatment are desirable because treatments are more effective to avoid the progression of hair loss than stimulating regrowth. Typically, a diagnosis of FPHL can be confirmed by review of a patient's medical history and a physical examination alone. Testing a scalp biopsy is diagnostic but usually not required. In women with signs of hyperandrogenism, an investigation for ovarian or adrenal disorders should be performed. Treatment for FPHL is obscured by myths. The aim of FPHL treatment could be two-fold: Reverse or stabilize the process of hair follicle miniaturization. Mild-to-moderate FPHL in women can be treated with oral antiandrogen therapies (cyproterone acetate and spironolactone) and/or topical minoxidil with good results in many cases. If used correctly, available medical treatments arrest the progression of the disease and reverse miniaturization in most patients with mild-to-moderate FPHL. Hair systems and surgery may be considered for selected cases of severe FPHL.

Ludwig classification scale:

Grade	*Description*	*Clinical aspect*
Grade I:	Perceptible thinning of the hair on the crown, limited in the front by a line situated 1–3 cm behind the frontal hair line.	
Grade II:	Pronounced rarefaction of the hair on the crown within the area seen in Grade I.	
Grade III	Full baldness (total denudation) within the area seen in Grades I and II.	

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6322157/

Olsen’s classification scale:

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The frequency of FPHL varies among population groups and ordinarily increases with age. However, a comparison of prevalence between different studies is hampered by the lack of universally accepted criteria for the disease (Ramos and Miot, 2015). Among healthy women, approximately 6% to 38% experience some degree of frontal and/or frontal-parietal hair loss (Birch et al., 2001).

The age of onset for FPHL is during the reproductive years, which is later than in men. Twelve percent of women first develop clinically detectable FPHL by age 29 years, 25% by age 49 years, 41% by 69 years, and > 50% have some element of FPHL by 79 years (Birch et al., 2002). More severe cases of the disease during puberty are more rarely described. Nevertheless, there is a greater demand for treatment among patients ages 25 to 40 years (Tosti and Piraccini, 2006). In the United Kingdom, 6% of women younger than age 30 years have FPHL for women older than 70 years, FPHL reaches a rate of 42% (Birch et al., 2002). Only 43% of women age > 80 years show no evidence of FPHL (Sinclair and Dawber, 2001).

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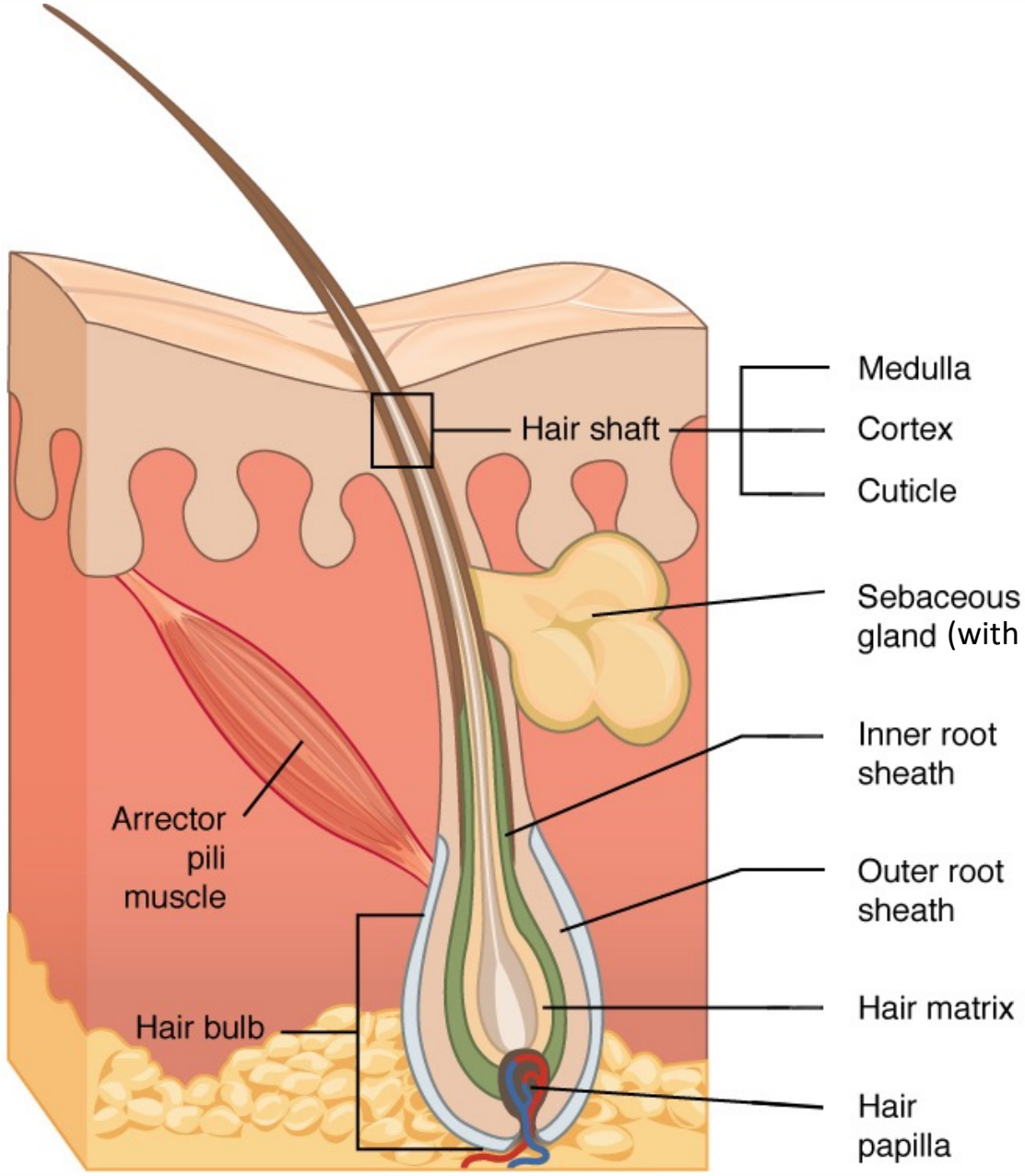
FPHL and male AGA share a final common pathway that causes follicular regression but current knowledge suggests that the etiology is not necessarily the same in both sexes. Although the role of androgens in the pathogenesis of male hair loss has been clearly established, the role of androgens in FPHL is less clear. In fact, FPHL may develop even in the absence of androgens (Herskovitz and Tosti, 2013). However, it is likely that other nonandrogenic factors that are currently unidentified may play a role in the pathogenesis of FPHL (Redler et al., 2017). Therefore, the involvement of these genes in the etiopathogenesis of FPHL cannot be completely excluded.

In women with FPHL who do not have elevated androgen levels, a genetic predisposition may be involved. This genetic disposition permits normal levels of circulating androgen to act on follicular target cells, which are specially sensitized by binding to specific intracellular androgen receptors. In other cases, an androgen-independent mechanism may be involved in the development of FPHL (Orme et al., 1999). Two recent studies by Heilmann-Heimbach et al. (2017) and Pickrell et al. (2016) have substantially found an increased number of gene loci (> 60) associated with male AGA.


Hair shaft
Medulla
Cortex
Cuticle
Sebaceous gland (with Infundibular tract).
Arrector pili muscle
Inner root sheath
Outer root sheath
Hair bulb
Hair matrix
Hair papilla

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Hair loss in women is polygenic and multifactorial with the additional influence of environmental factors. Several studies focused on the importance of several genes related to alopecia (Carey et al., 1993, Hillmer et al., 2008, Randall, 2008). FPHL involves progressive hair follicle miniaturization and subsequently the conversion of terminal follicles into vellus-like follicles. These vellus-like follicles have a shortened hair cycle because of a reduction in the anagen phase, which leads to the production of short and fine hair shafts. Unlike in men, the miniaturization is not uniform and intense in women; therefore, there are no complete areas of baldness except in very rare cases (Birch et al., 2001). Moreover, the miniaturization process may be accompanied by a mild-to-moderate lympho-histiocytic inflammatory infiltrate in the peri-infundibular region. The term "microinflamation" has been used to differentiate this infiltrate from the inflammation that occurs in scarring alopecia (Stefanato, 2010).

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Comorbidities

Go to: ▸

The most common endocrinologic comorbidity that is associated with FPHL is polycystic ovarian syndrome (El Sayed et al., 2016). Metabolic syndrome, which is characterized by obesity, insulin resistance, hypertension, hyperprolactinemia, and raised aldosterone levels, also appears to be frequently associated with FPHL (El Sayed et al., 2016). An increased risk of carotid and coronary artery diseases have also been reported (Arias-Santiago et al., 2010). To further clarify the comorbidity profile of FPHL, systematic studies in larger population-based samples are needed.

An association between ferritin levels and FPHL is controversial. Some studies have demonstrated lower ferritin levels in patients with FPHL compared with controls and antiandrogen therapy seem to work better in patients with ferritin levels > 40 μg/l (Ramos and Miot, 2015).

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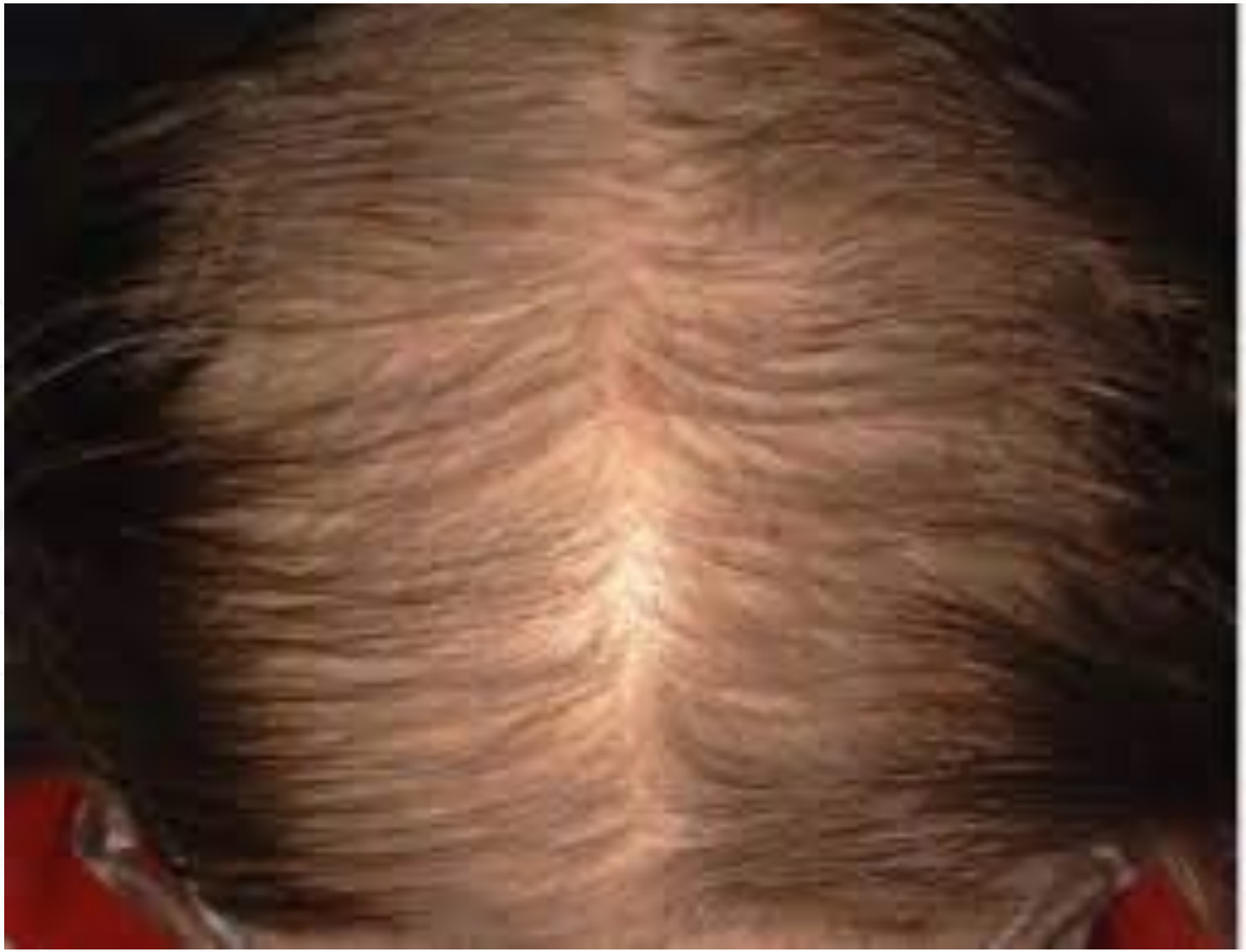
Furthermore, iron acts as a metabolic cofactor for ribonucleotide reductase, which is the rate-limiting enzyme for DNA synthesis of hair growth stems. Therefore, the depletion of iron results in the inhibition of proliferation. Vitamin D has been suggested as an optimal concentration of this micronutrient that is necessary to delay aging phenomena including hair loss. Data from animal models show that vitamin D receptor activation plays an important role in anagen initiation and recent data suggested that vitamin D receptors regulate the expression of genes that are required for hair follicle cycling (Amor et al., 2010, Demay, 2012). Also, the definition of iron deficiency in hair loss remains an important question because some studies suggest that with a serum ferritin level of ≤ 30 μg/l and active hair loss, patients should be treated with iron therapy. However, other studies suggest higher cutoff limits such as 40 μg/l and 70 μg/l or lower cutoff limits such as 10 μg/l to 15 μg/l (Bregy and Trueb, 2008, Deloche et al., 2007, Rushton and Ramsay, 1992).

Other ingredients such as saw palmetto (Nutrafol) or marine protein complexes (Viviscal) may have anti-androgenic and anti-inflammatory properties but their efficacy alone as a monotherapy needs more investigation.



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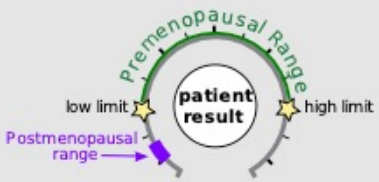
30 yo female. Hair Loss, post partum, Low thyroid.

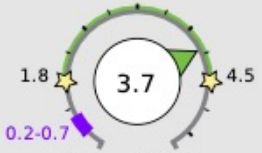
Hormone Testing Summary

Key (how to read the results):

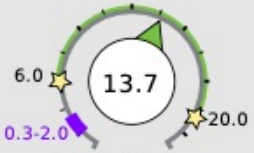


Sex Hormones

See Pages 2 and 3 for a thorough breakdown of sex hormone metabolites



Estradiol(E2)



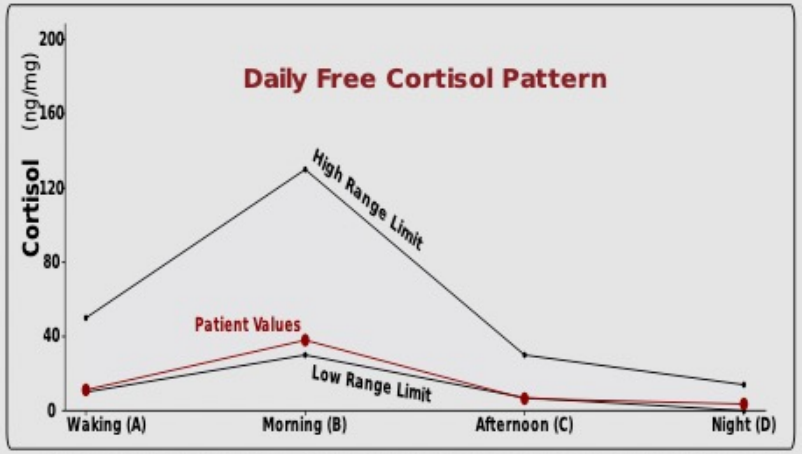
Progesterone
(Serum Equivalent, ng/mL)

Progesterone Serum Equivalent is a calculated value based on urine pregnanediol.

Testosterone

Adrenal Hormones

See pages 4 and 5 for a more complete breakdown of adrenal hormones



Total DHEA Production

Age	Range
20-39	1300-3000
40-60	750-2000
>60	500-1200

Total DHEA Production
(DHEAS + Etiocholanolone + Androsterone)

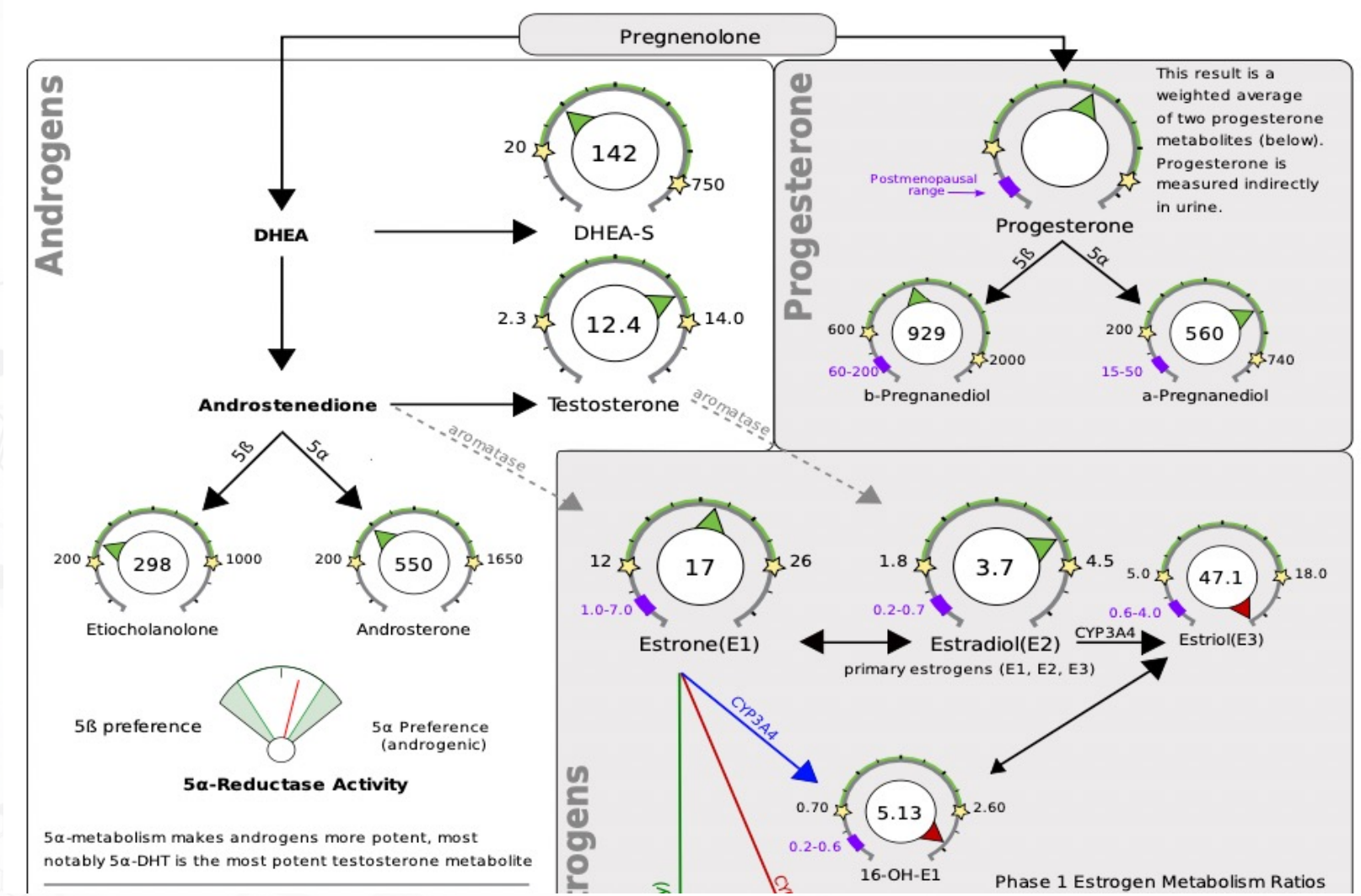
24hr Free Cortisol
(A+B+C+D)

cortisol metabolism

Metabolized Cortisol (THF+THE)
(Total Cortisol Production)

Free cortisol best reflects tissue levels. Metabolized cortisol best reflects total cortisol production.

30 yo female. Hair Loss, post partum, Low thyroid.



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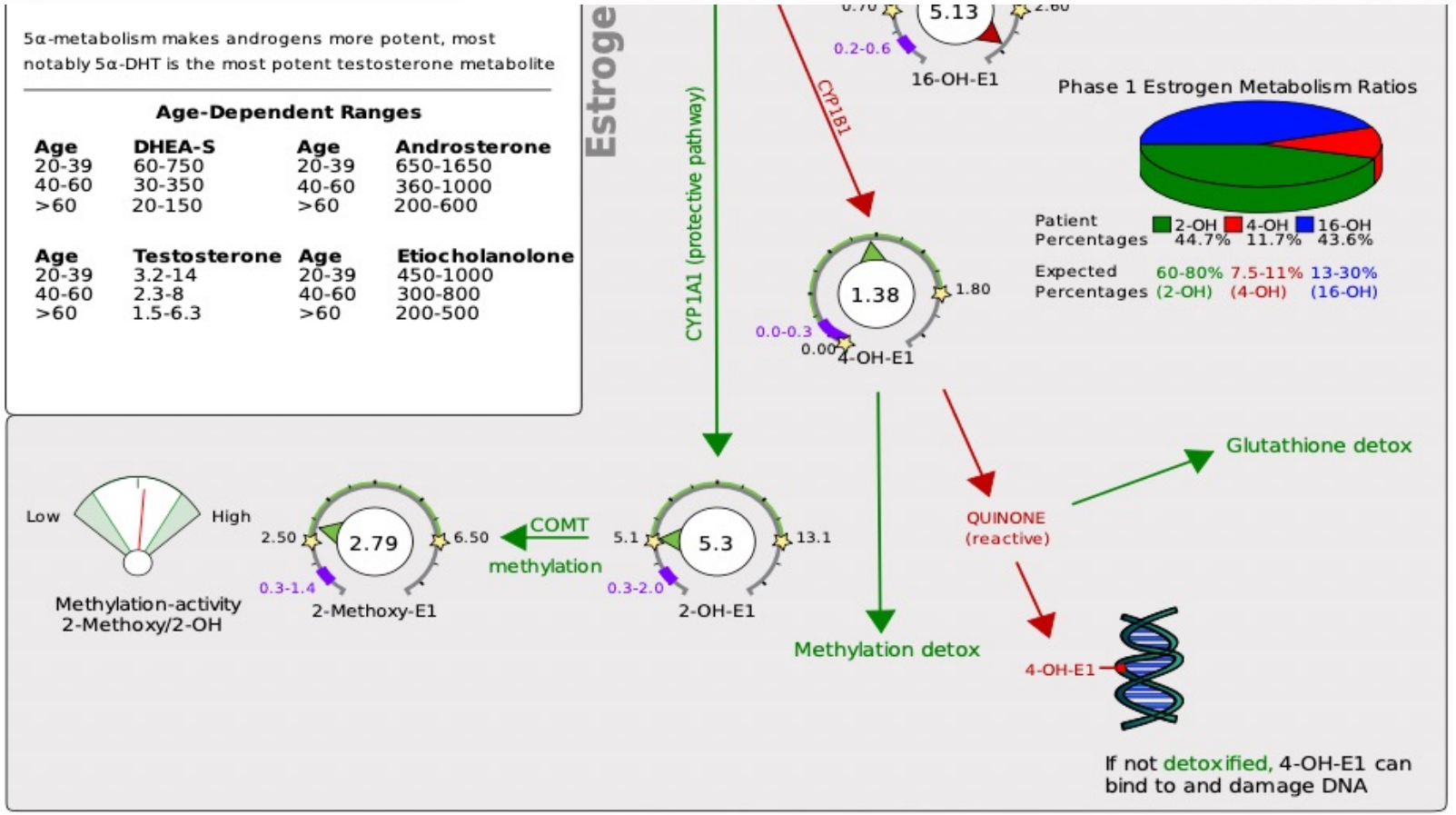

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	Iron [01]	46		ug/dL	27-159
▼	**Iron Saturation**	**14**	**Low**	%	15-55
	Ferritin [01]	105		ng/mL	15-150
▲	**Cholesterol, Total** [01]	**212**	**High**	mg/dL	100-199
	Triglycerides [01]	96		mg/dL	0-149
	HDL Cholesterol [01]	57		mg/dL	>39
	VLDL Cholesterol Cal	17		mg/dL	5-40
▲	**LDL Chol Calc (NIH)**	**138**	**High**	mg/dL	0-99

▲	**C-Reactive Protein, Cardiac** [01]	**3.86**	**High**	mg/L	0.00-3.00

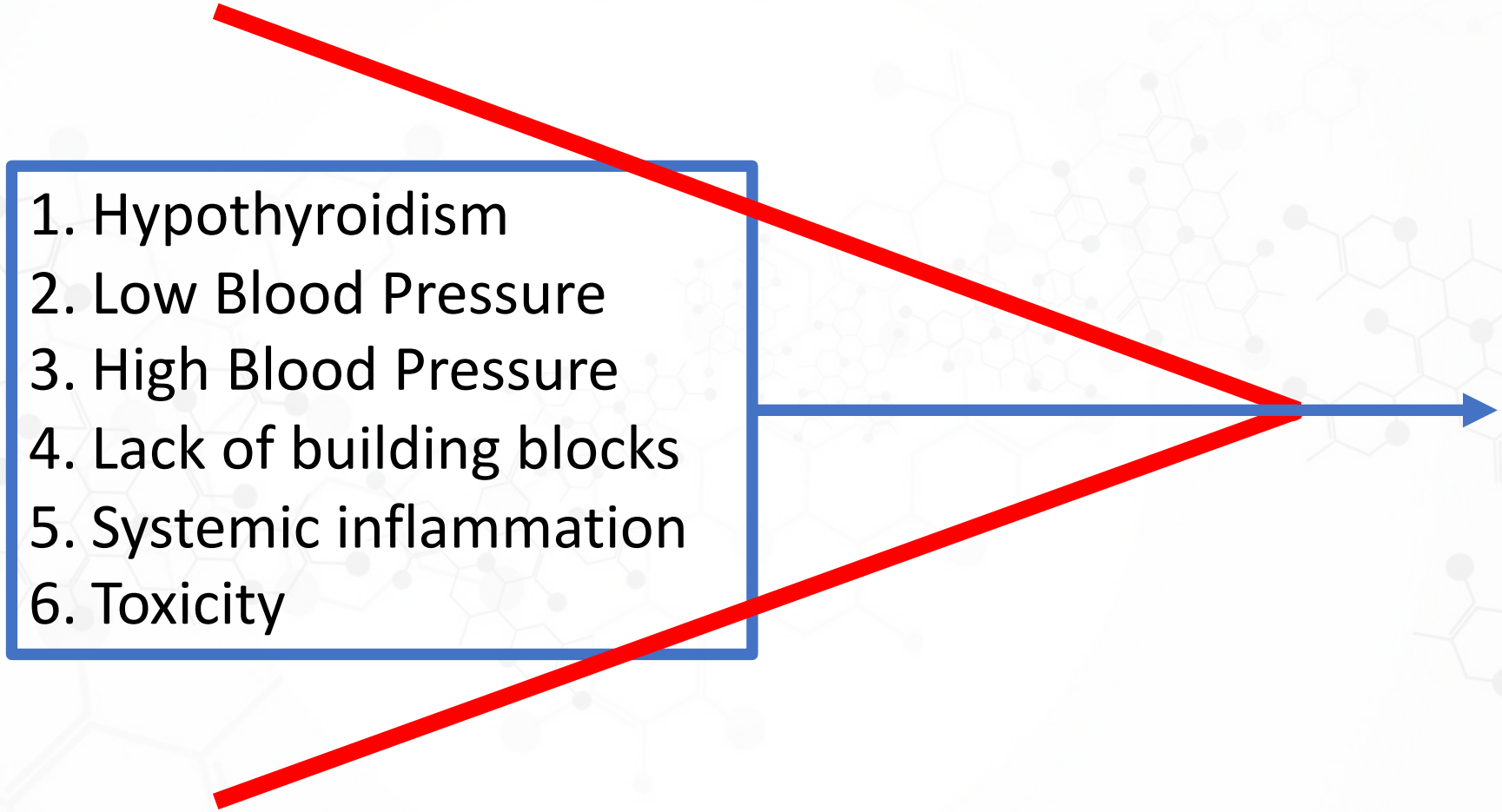
Relative Risk for Future Cardiovascular Event

Low	<1.00
Average	1.00 - 3.00
High	>3.00

	Homocyst(e)ine [01]	8.7		umol/L	0.0-14.5
▲	**TSH** [01]	**13.800**	**High**	uIU/mL	0.450-4.500
	Thyroxine (T4) [01]	4.6		ug/dL	4.5-12.0
	T3 Uptake [01]	26		%	24-39
	Free Thyroxine Index	1.2			1.2-4.9
	Triiodothyronine (T3) [01]	77		ng/dL	71-180
	Triiodothyronine (T3), Free [01]	2.3		pg/mL	2.0-4.4
	Reverse T3, Serum [A,02]	10.6		ng/dL	9.2-24.1
	T4,Free(Direct) [01]	0.85		ng/dL	0.82-1.77
	Thyroid Peroxidase (TPO) Ab [01]	**149**	**High**	IU/mL	0-34
	Thyroglobulin Antibody [01]	<1.0		IU/mL	0.0-0.9

Thyroglobulin Antibody measured by Beckman Coulter Methodology

▼	**Vitamin D, 25-Hydroxy** [01]	**12.5**	**Low**	ng/mL	30.0-100.0


1. Hypothyroidism
2. Low Blood Pressure
3. High Blood Pressure
4. Lack of building blocks
5. Systemic inflammation
6. Toxicity