dutchwebinars

Quiet Decline: Identifying and Addressing Low Androgens in Midlife Men and Women

Allison Smith, ND
Director of Clinical Education
Precision Analytical

7/29/2026

Allison Smith, ND – Clinical Director at Precision Analytical (DUTCH)

Dr. Smith is a licensed naturopathic doctor bringing 16 years of experience including 12 years of working within the hormone lab testing space.

She provides clinical support and professional guidance to practitioners learning to apply functional endocrinology in their practices!

Today's Agenda

1. **Describe the physiological roles of androgens** in both men and women, including their effects on metabolism, musculoskeletal health, cognition, and overall vitality.
2. **Identify typical patterns of androgen decline in midlife**, and recognize how these patterns may differ by sex and individual physiology.
3. **Interpret key androgen-related markers on a dried urine hormone test**, including hormone levels and metabolic patterns relevant to aging and stress physiology.
4. **Recognize stress- and metabolism-related factors** that may contribute to low androgen states and impaired anabolism (ie: cortisol!).
5. **Apply dried urine hormone test findings to clinical case examples** involving low androgens in both female and male patients.
6. **Discuss common therapeutic options** for addressing low androgens, including hormonal and non-hormonal approaches, within a functional medicine context.

Let's Review Androgens!

What are **Androgens**?

- A class of hormones and metabolites that are classically thought of as the "male" hormones as they are responsible in males to drive development of male features and secondary sex characteristics.
- They also have critical function in females!
  - Precursors to production of estrogen.
  - Critical for follicular development and egg quality in fertility.
  - Sexual function and bone health in menopause.

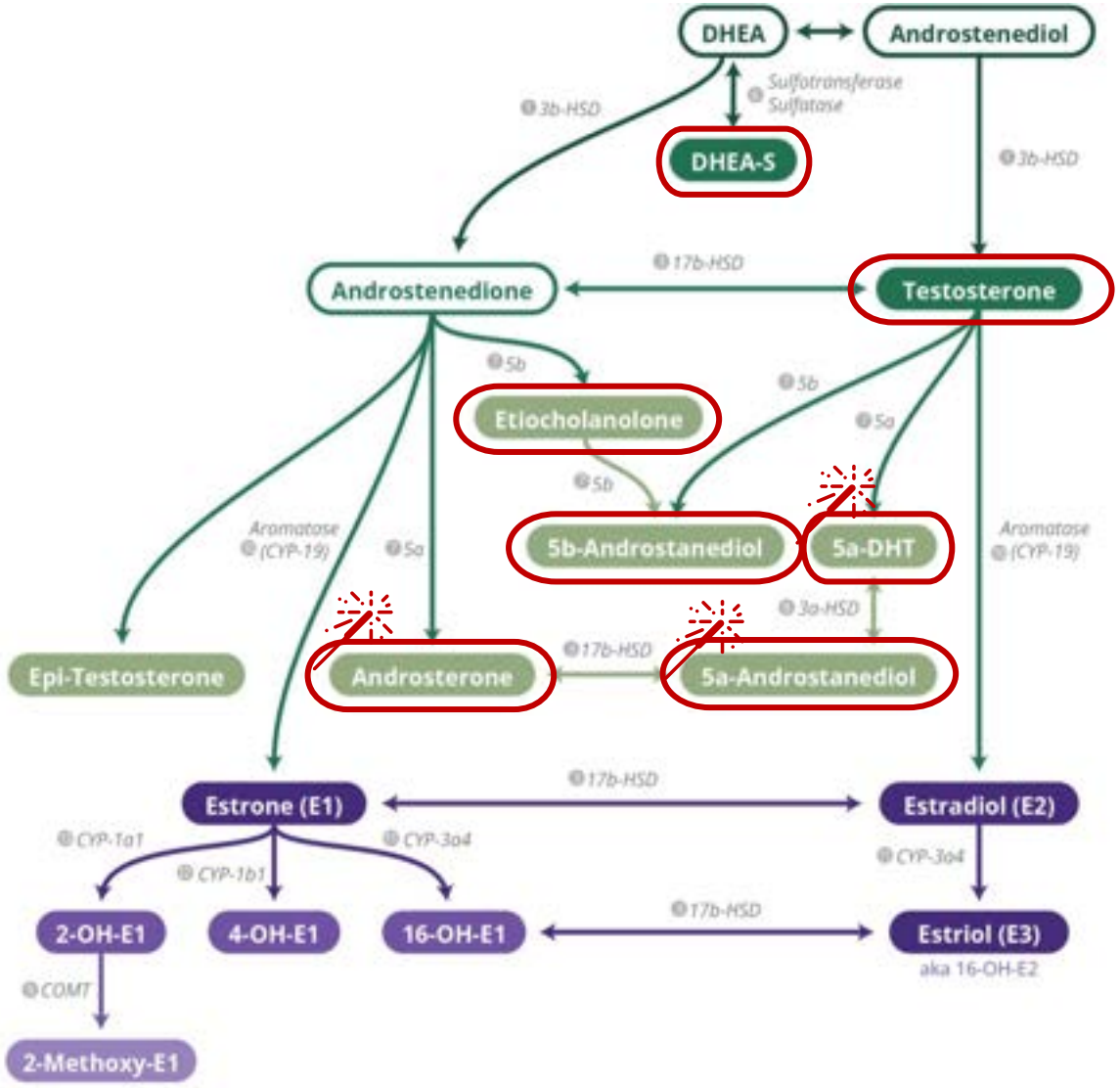
Androgen Production Starts with DHEA

- **DHEA-S** is the bound-up sulfated form of DHEA.
- **Etiocholanolone** is a *beta* DHEA metabolite.
- **Androsterone*** is an *alpha* DHEA metabolite.

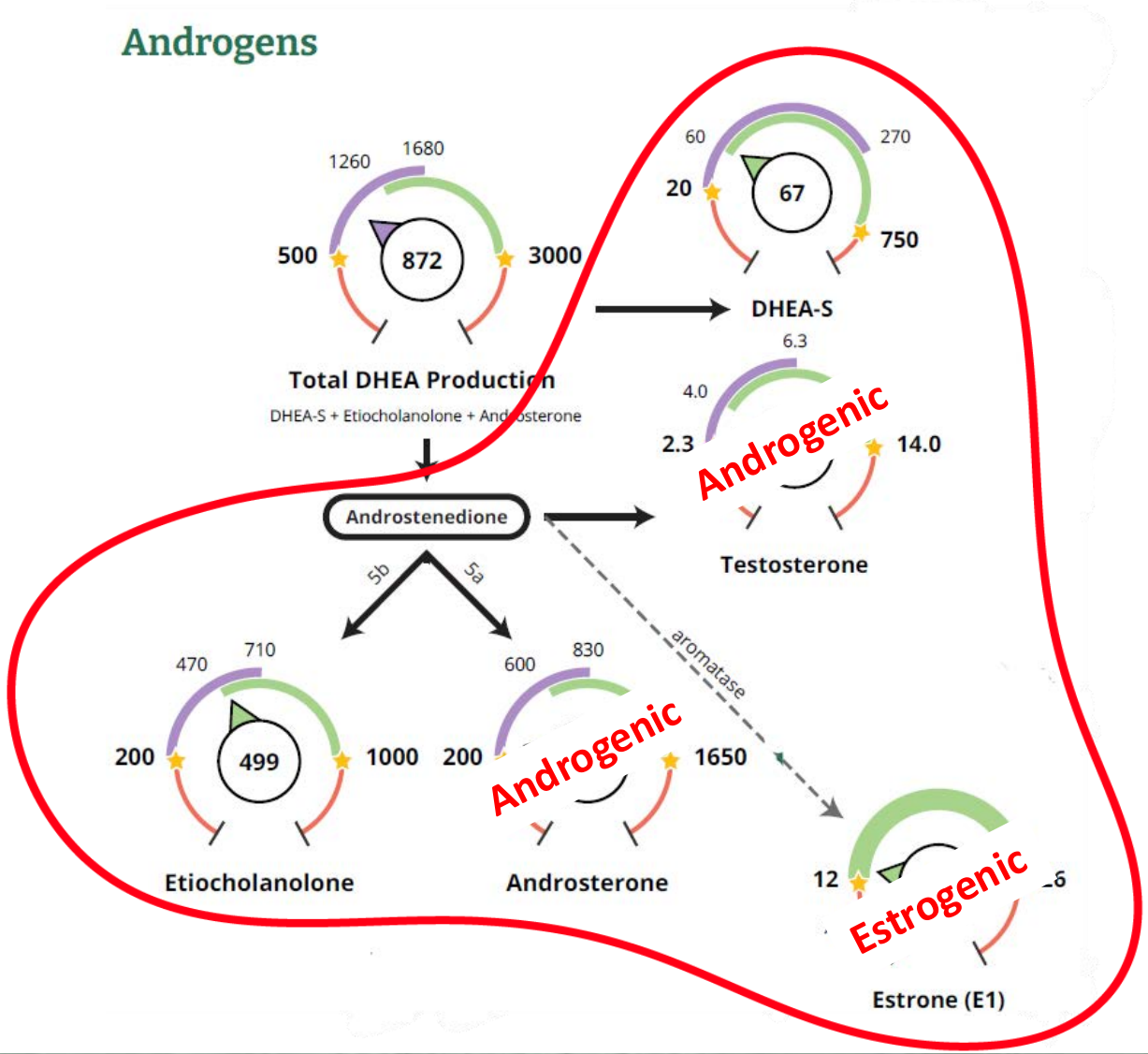
Alpha metabolites are made in **target tissues** and are **active** on the androgen receptors.

Beta metabolites are made in the **liver** and are **NOT active** on the androgen receptors.

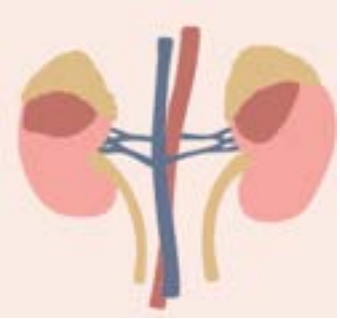
- **Testosterone** is converted into:
- *Alpha* metabolites in the *tissues*:
  - **5a-DHT (androgenic)**
  - **5a-Androstanediol (androgenic)**
- *Beta* metabolites in the *liver*:
  - **5b-Androstanediol (inert)**



DHEA is a Pro Hormone



DHEA (Mostly) Made in the Adrenal Glands



- **DHEA synthesis**
  - Occurs in the adrenal cortex (zona reticularis), gonads, and brain
- **DHEA is a pro-hormone**
  - A precursor to most other steroid hormones
  - All estrogens are synthesized from androgens
- **DHEA is involved in the stress response**
  - An **immune modulator** and **anti-inflammatory hormone**
  - Acute stress biomarker (increases within 1 hour)

Dutheil F, et al. Front Psychiatry. 2021; 12: 688367.

General Androgen Actions in Men and Women

Androgen Actions

- **Brain:** Mood, motivation, energy, sex drive, focus, sense of well-being
- **Bone density**
- **Insulin secretion**
- **Hair (body) growth**
- **Skin:** Moisture, elasticity, sebum production
- **Muscle mass:** Strength, Stamina, Recovery
- **Cardiovascular Health**
- **Immune function**
- **Sexual function:** Vasodilation, tissue elasticity, moisture
- **Fertility**: At optimal levels helps follicle growth in women and sperm production/count in men

DHEA Levels Affect Estrogen and Testosterone Levels

Because DHEA can be converted into androgens and estrogens
it can affect both androgen and estrogen activity in the body!

Estrogen's Actions

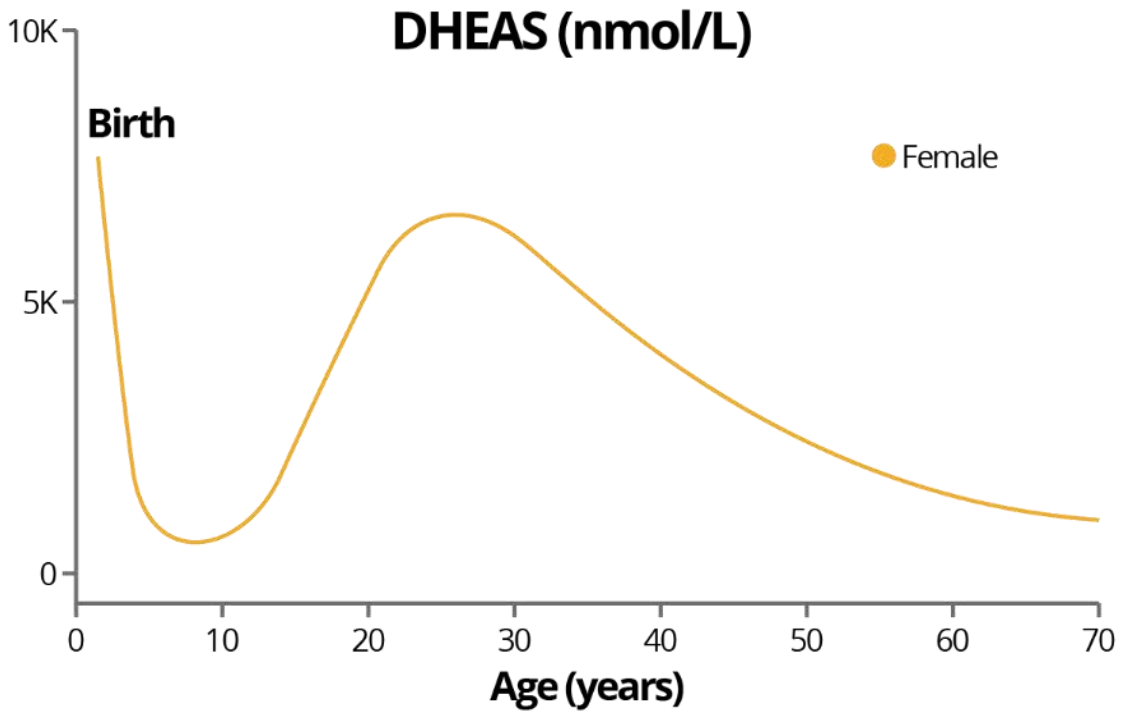
- **Brain:** Mood, cognition, memory, and focus, thermoregulation, sleep, energy
- **Hair (scalp) growth**
- **Skin:** Elasticity, collagen, repair, moisture
- **Muscle mass**
- **Nervous system:** Parasympathetic balance
- **Joint health & Bone density**
- **Breast health**
- **Liver function:** healthy cholesterol
- **Insulin sensitivity**
- **Weight management**
- **Uterine health**
- **Genitourinary system:** Elasticity, microflora, moisture
- **Fertility**
- **Cardiovascular:** endothelial function, etc.

Androgen Actions

- **Brain:** Mood, motivation, energy, sex drive, focus, sense of well-being
- **Bone density**
- **Insulin secretion**
- **Hair (body) growth**
- **Skin:** Moisture, elasticity, sebum production
- **Muscle mass:** Strength, Stamina, Recovery
- **Cardiovascular Health**
- **Immune function**
- **Sexual function:** Vasodilation, tissue elasticity, moisture
- **Fertility**: At optimal levels helps follicle growth in women, sperm development and sperm count in men

DHEA-S Declines with Age in Both Men and Women

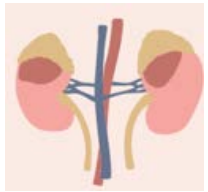
- Androgens **peak in the 20's and 30's** and then decline thereafter.



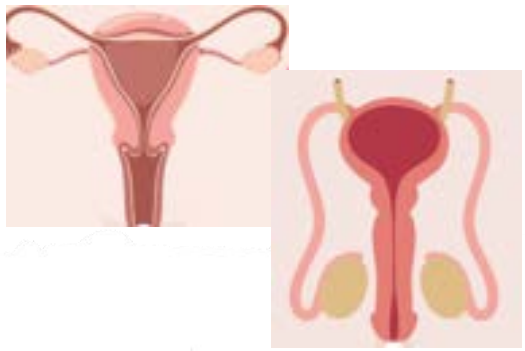
Labrie F, et al. Front Neuroendocrinol. 2001.

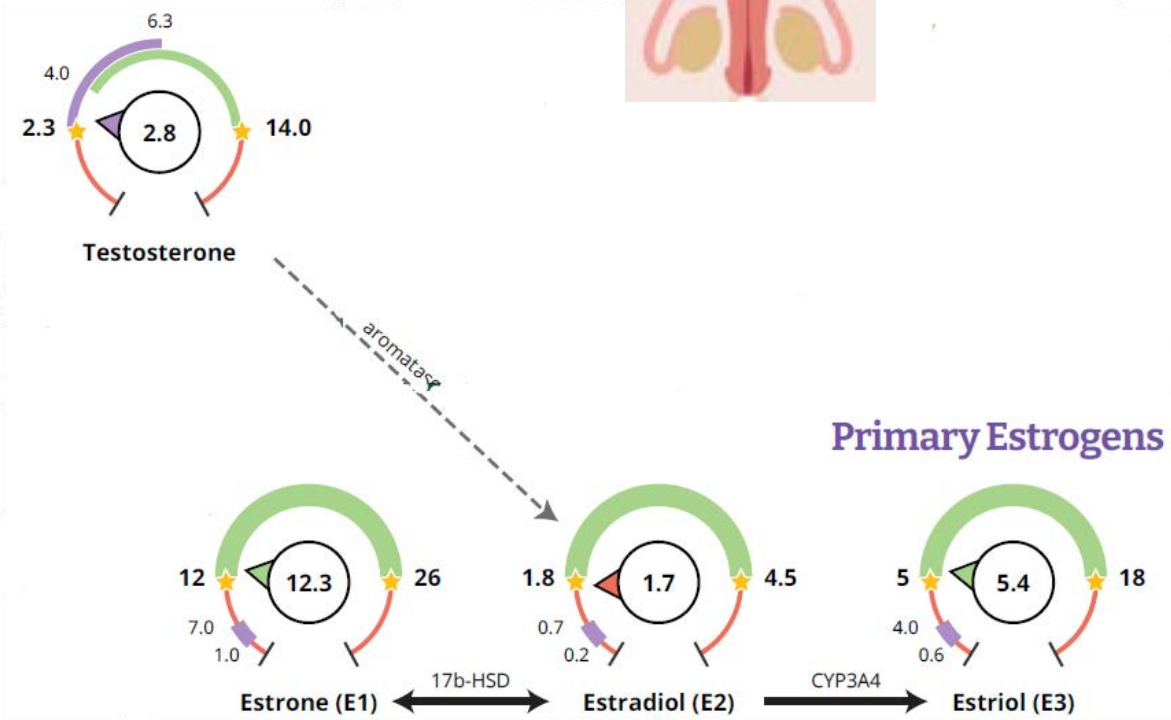
Testosterone (Mostly) Made in the Gonads in Men and Adrenal/Peripheral in Women

- **Testosterone synthesis**
  - Men: Occurs in Leydig cells in the testicles and adrenal cortex (zona reticularis)
  - Cycling women: Occurs in the theca cells in the ovaries of women and from peripheral conversion of adrenal DHEA
  - Menopausal women: Mostly adrenal source – 60% of T from peripheral conversion of adrenal DHEA plus 40% of T from theca/stroma cells in the ovaries for 10+ years after menopause



- **Testosterone forms estrogens via aromatase**
  - Adipose tissue expresses ↑ aromatase
  - All estrogens are synthesized from androgens



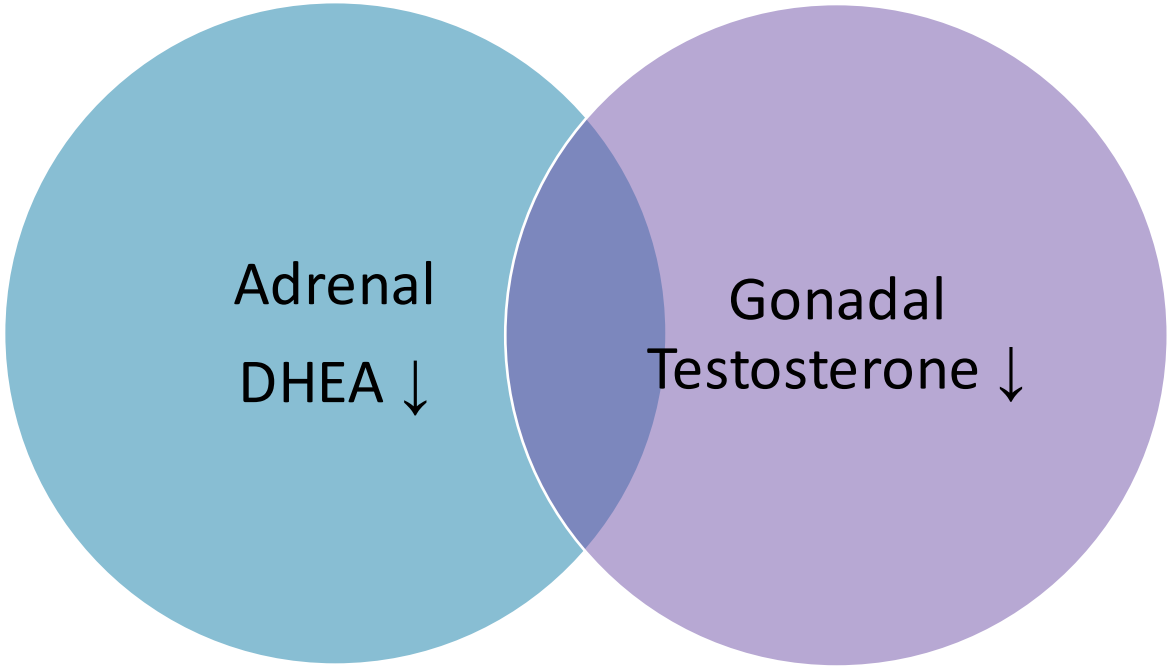


Laughlin GA, et al. H J Clin Endocrinol Metab. 2000.

The Mid-Life Androgen Trajectory and Senescence Begins in Our 30s

In Men

Overlay of Adrenal Aging with Gonadal Aging



>95% of a man's T is gonadal sourced

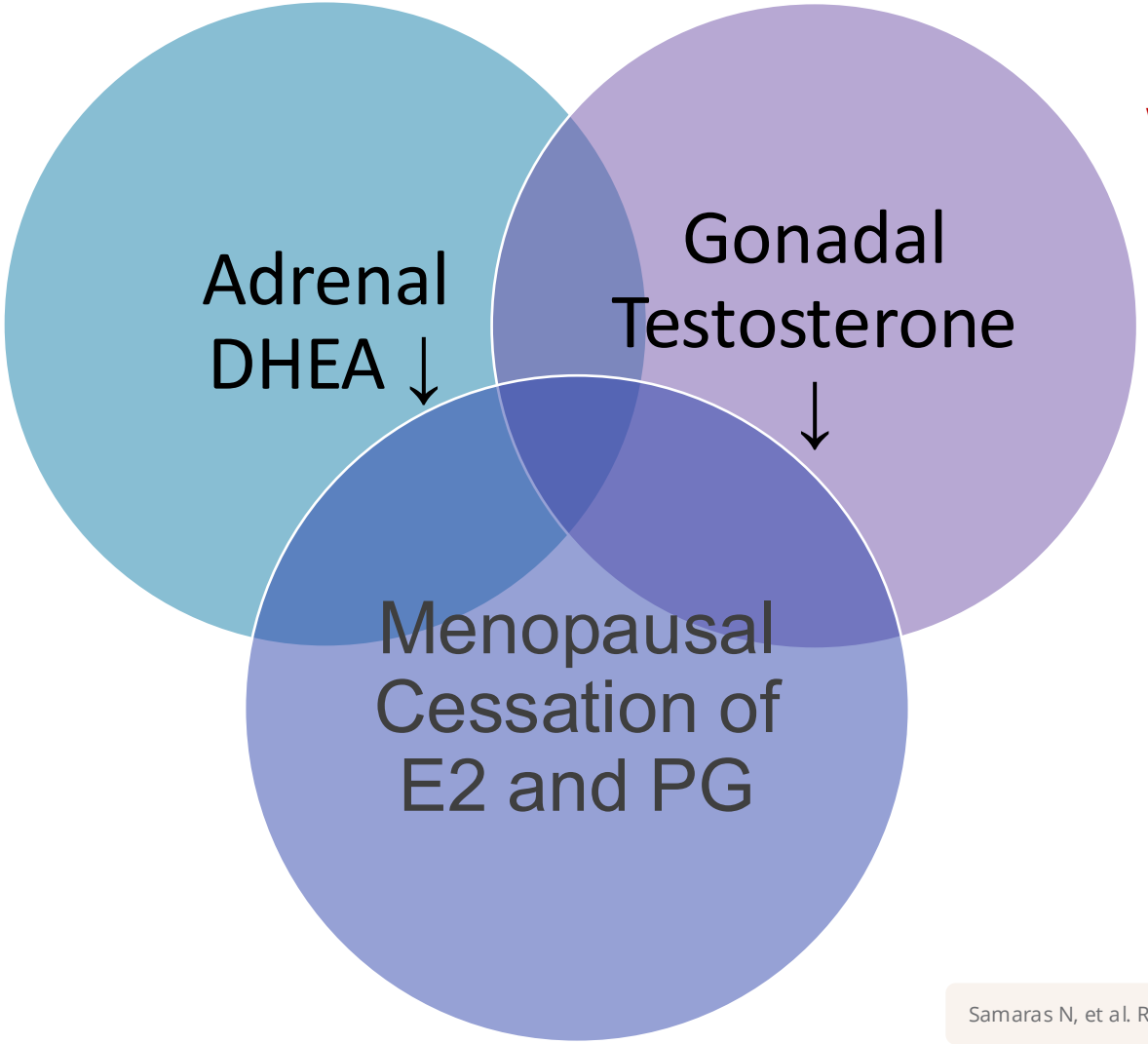
Samaras N, et al. Rejuvenation Res. 2013.

The Mid-Life Androgen Trajectory and Senescence Begins in Our 30s

In Women

Overlay of Adrenal Aging with Gonadal Aging

Added Symptom Impact of Menopause



~25% of a woman's T is gonadal sourced

Samaras N, et al. Rejuvenation Res. 2013.

DHEA Decline is Steady

DHEA levels are dependent on:

Age

- DHEA declines gradually by 2-4% per year beginning in our 30's.

ACTH secretion through the HPA axis

- Like cortisol, DHEA (but not DHEA-S) has a diurnal pattern because it is secreted in response to ACTH signaling from the brain.
- DHEA is involved in the stress response.

Zona reticularis of the adrenal gland

- Function, hypertrophy, atrophy, etc.

Labrie F, et al. Front Neuroendocrinol. 2001.
Schiffer L, et al. Mol Cell Endocrinol. 2018.

Testosterone Decline is Steady

In Males:

- Declines by about 1-2% per year with age
- Sexual symptoms are the most common
  - Can occur without other symptoms

In Females:

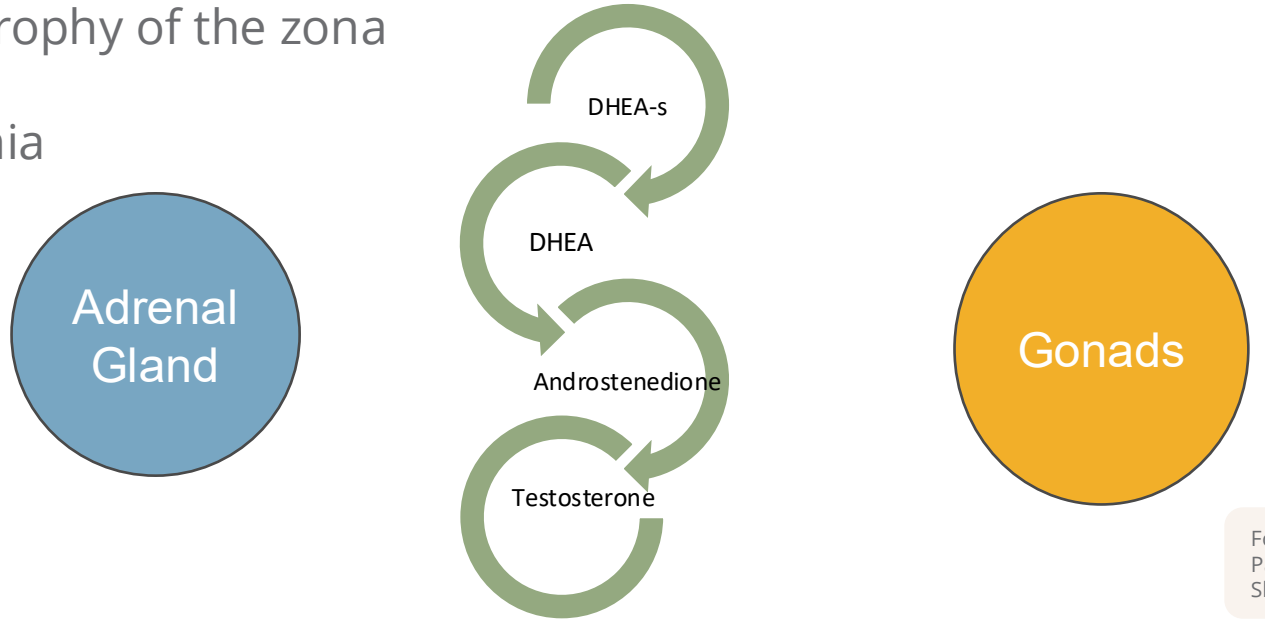
- Declines by about 50% from 20s on
- Hypoactive sexual desire dysfunction
- Body composition changes

Brzozowska M, Lewiński A. Prz Menopauzalny. 2020.

Additive Drivers to Mid-Life Decline in Androgens

Additional factors (besides aging) that lower androgen levels:

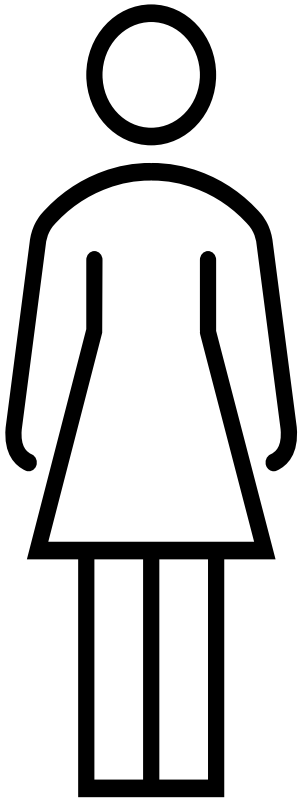
- Oophorectomy (females)
- Medications – statins, chemo, GnRH analogs
- High sex hormone binding globulin (SHBG)
- Chronic stress/HPA axis dysfunction
- Age-related morphological changes in the adrenal cortex: atrophy of the zona reticularis
- Hyperprolactinemia
- Anorexia nervosa



Variation in androgen levels among men and women comea from issues with the adrenal glands and gonads that may be present at any time in life.

Fogle RH, et al. J Clin Endocrinol Met. 2007;92(8):3040-3043.
Papierska L. Menopausal Review. 2017;2:57-60.
Sharma S, Mahajan N. J Midlife Health. 2021;12(1):3-7.

Symptoms Associated with Low Androgens

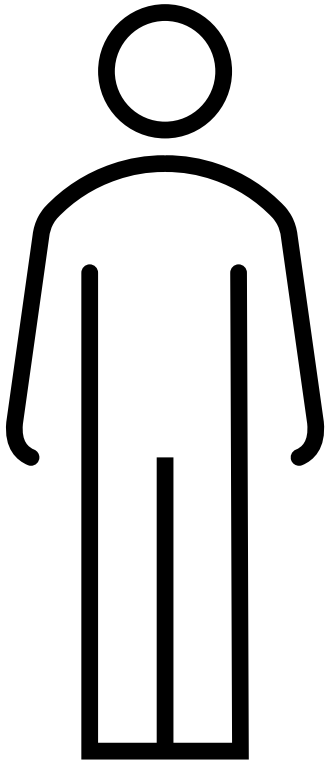


Androgen (both testosterone and DHEA-S) deficiency in women is associated with:

- Low libido
- Low mood
- Osteopenia and osteoporosis
- Reduced muscle mass – including pelvic floor dysfunction
- Vaginal dryness
- Fatigue
- Vasomotor symptoms
- Insomnia
- Depression
- Headaches

Bachmann GA. Fertility and Sterility. 2002;77:72-76.

Male Symptoms Associated with Age-Related Low Androgens



Androgen (both testosterone and DHEA-S) deficiency in men is associated with:

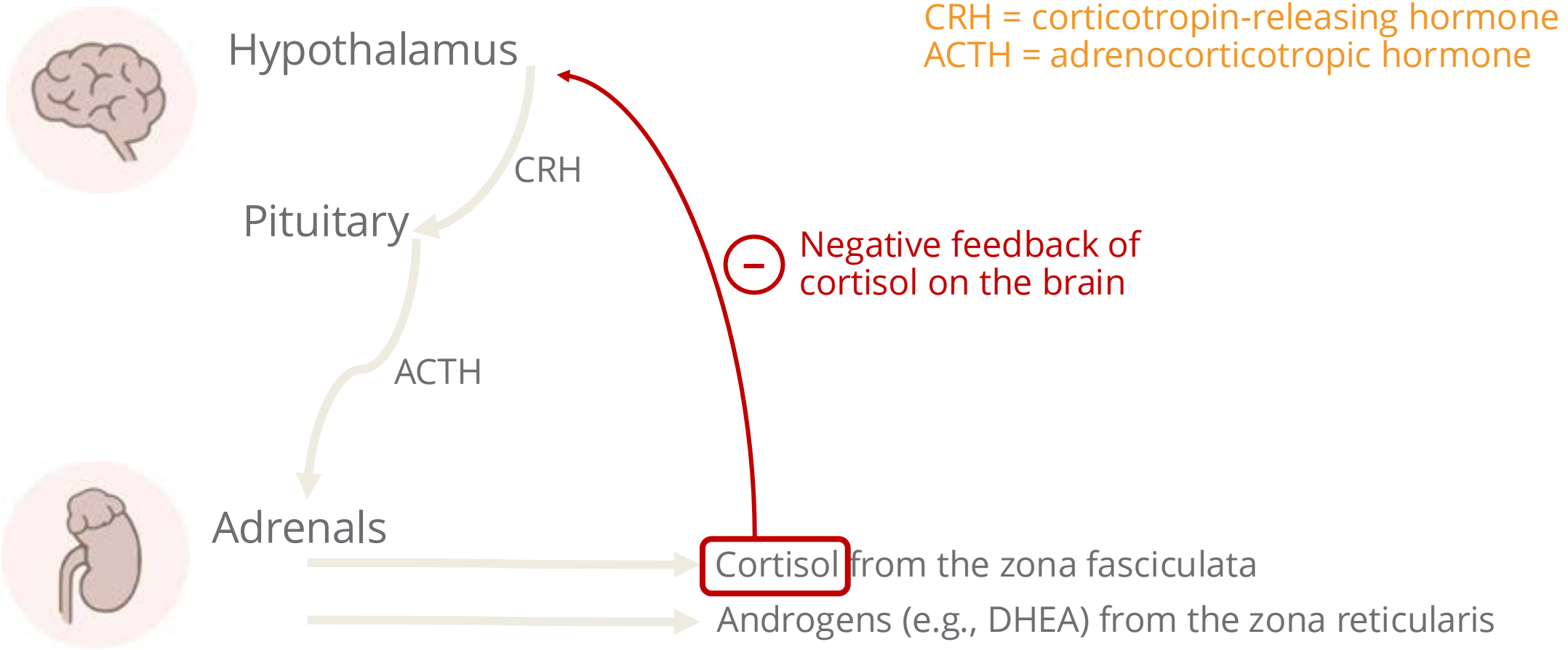
- Low libido – a primary symptom and may occur in isolation esp in men > 50yo (w/o other obvious causes)
- Erectile dysfunction
- Reduced Fertility/Sperm count
- Cognitive issues (brain fog)
- Reduced muscle mass
- Fatigue
- Insomnia
- Depression
- Irritability
- Low motivation

Mok SF, et al. J Gerontol A Biol Sci Med Sci. 2020.

DHEA, Cortisol, and the Stress Response

The Adrenal Stress Response

Hypothalamic-Pituitary-Adrenal (HPA) Axis Communication



All images from https://canva.com

DHEA, Cortisol, and the Stress Response

- **DHEA synthesis**
  - Occurs in the adrenal cortex (zona reticularis), gonads, and brain
- **Cortisol synthesis**
  - Occurs in the adrenal cortex (zona fasciculata)
- **DHEA-S**
  - DHEA-S does not follow a diurnal pattern, it is constant
  - Unsulfated DHEA is diurnal, Cortisol is also diurnal
- **DHEA is a pro-hormone**
  - A precursor to most other steroid hormones
  - All estrogens are synthesized from androgens
- **DHEA and Cortisol are both involved in the acute stress response**
  - DHEA: **immune modulator** and **anti-inflammatory hormone**
  - Cortisol: **immune suppressor, acute anti-inflammatory (chronic pro-inflammatory)**

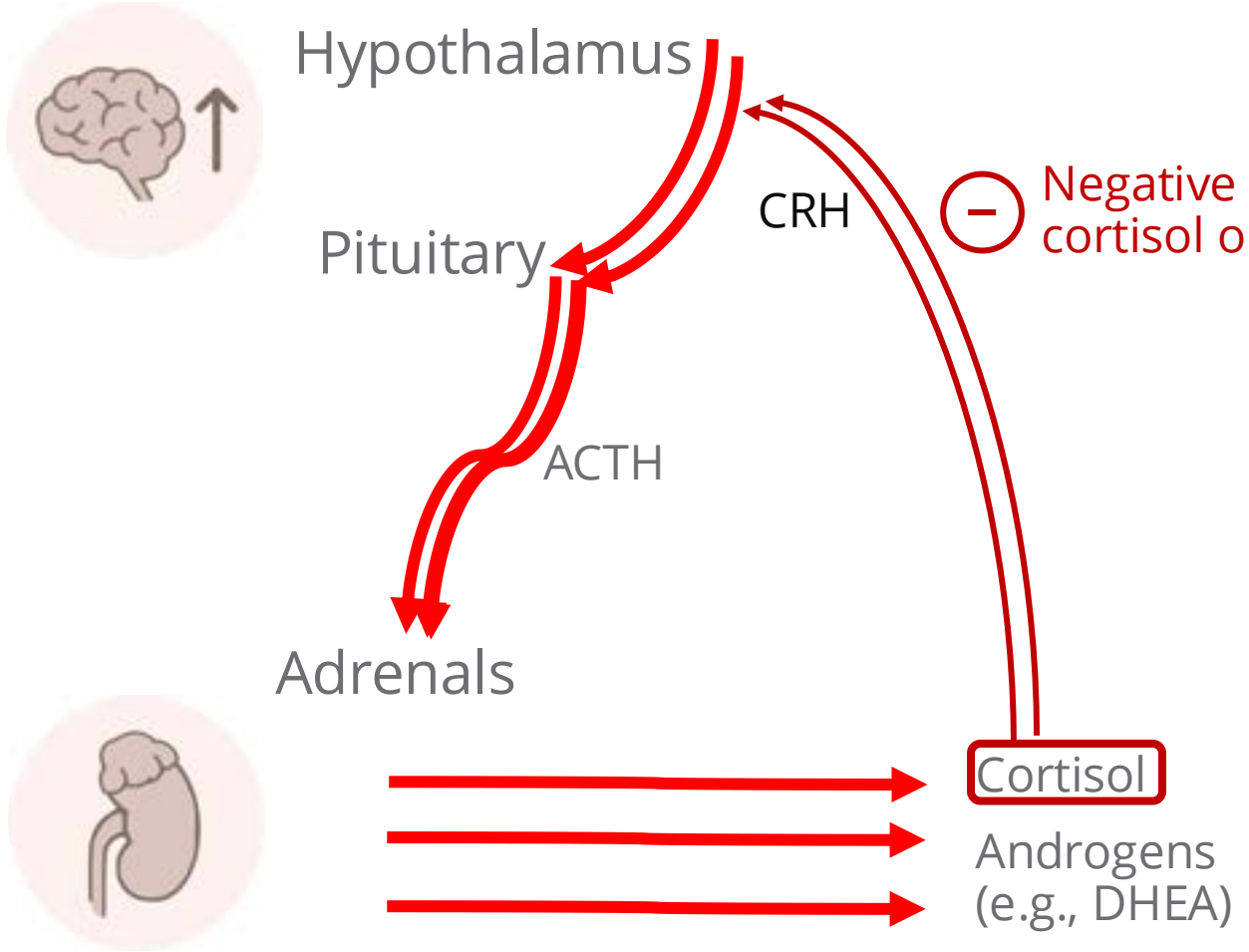
Dutheil F, et al. Front Psychiatry. 2021.

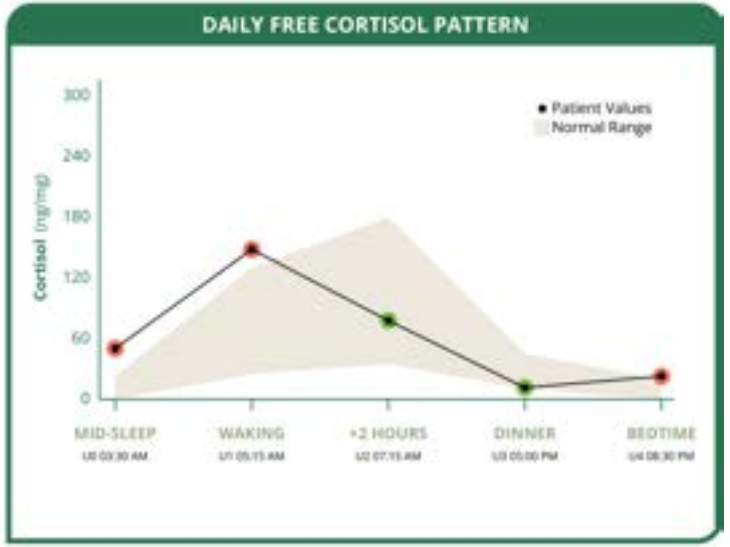
Acute Cortisol Effects

- Maintains glucose levels for energy
  - Gluconeogenesis: mobilizes glucose from fat and liver cells
  - Blocks insulin to maintain blood sugar for energy
- Increased focus: mental and physical
- Increased HR, blood pressure (vasoconstriction), muscle blood flow
- Decreased digestive effort
- Decreased sex hormone response
- Decreased immune response

The Adrenal Stress Response

Over time, chronically high cortisol can lead to…

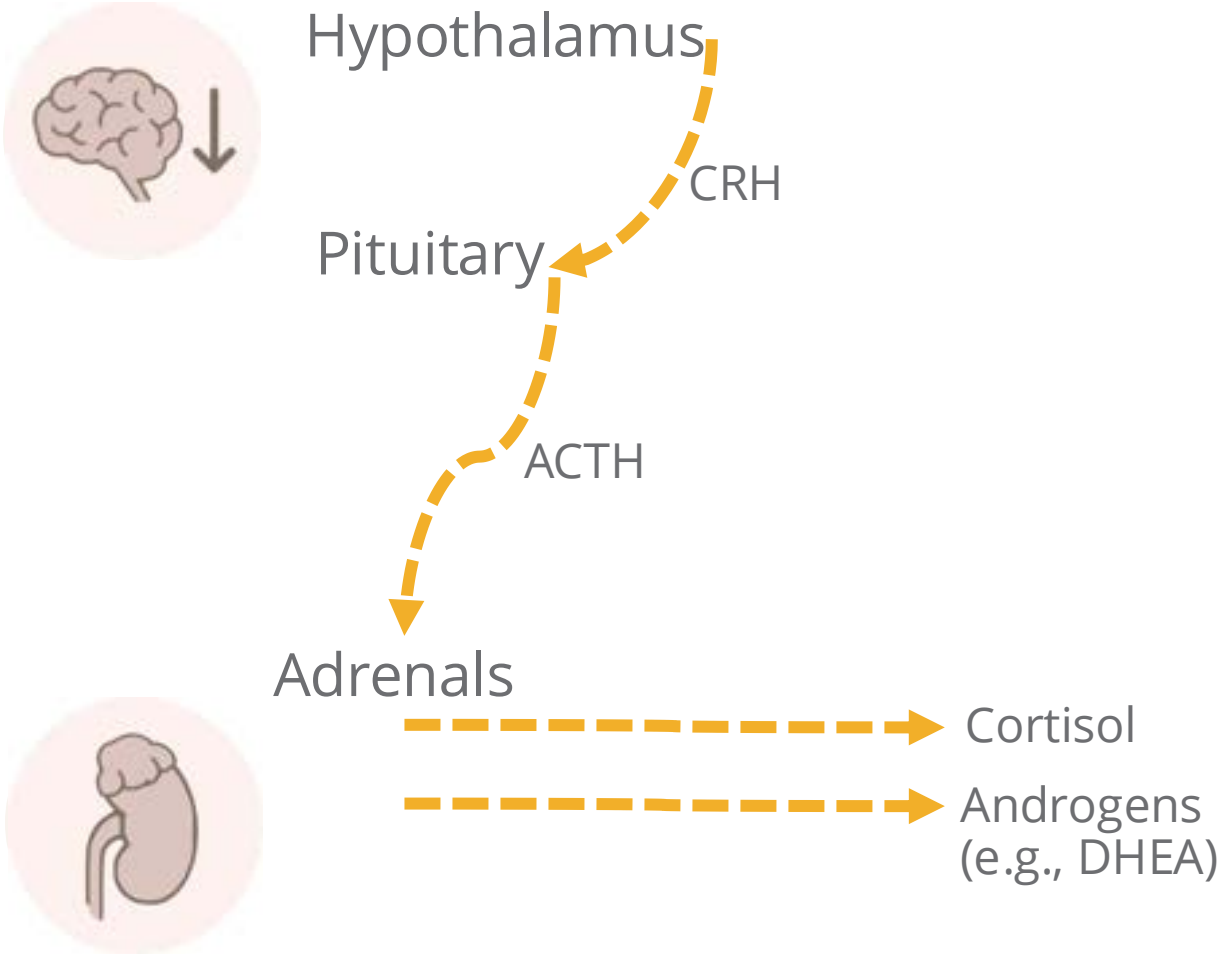


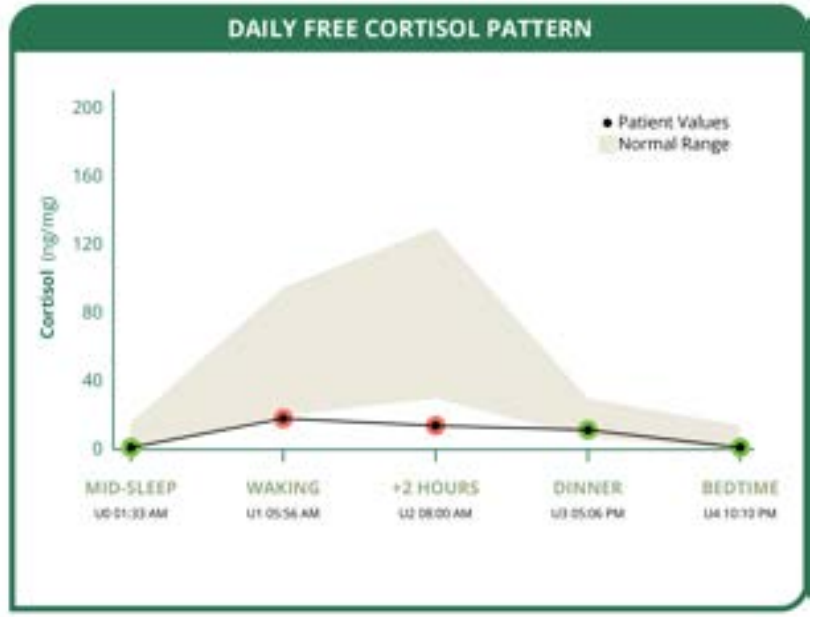


48-year-old male
Significant sleep issues

The Adrenal Stress Response to Chronic Stress

...low cortisol.





49-year-old female
Low libido
Fatigue that worsens throughout the day

Chronic Cortisol Effects

- Insulin dysregulation, dysglycemia, IR/diabetes
- Central adiposity
- Muscle wasting
- Bone loss
- Immune dysregulation, immune suppression, and inflammation
- Chronic fatigue
- Gastrointestinal effects: parasympathetic nervous system suppression
- Cardiovascular effects: HTN, hyperlipidemia, endothelial dysfunction
- Sex hormone imbalance
    - Females: infertility, irregular periods, heavy periods, decreased libido
    - Males: infertility, low testosterone, decreased libido, erectile dysfunction

Low DHEA vs Cortisol

#1 Reason to care about low androgens in mid-life: Cardiovascular Risk!

But why?

Cortisol Levels Are Stable Throughout the Lifespan

With DHEA levels dropping with age, relative shift to relative cortisol dominance and favoring CATABOLISM

- **DHEA** is considered more ***anabolic***.
- **Cortisol** is considered more ***catabolic***.

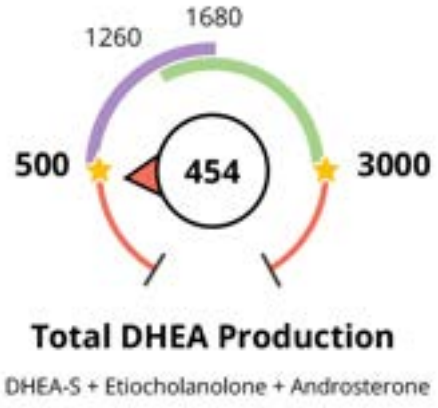
More cortisol relative to DHEA may put a patient in a catabolic state.

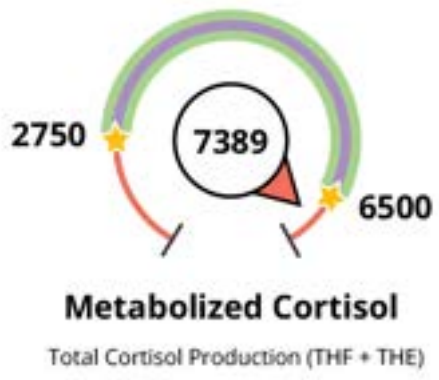
- Excessive catabolism results in the body **breaking down** more tissue than it builds.
- It can result in fatigue, muscle loss, low bone mineral density, weakness, impaired tissue repair, poor wound healing and weakened immune function.

Anabolic-Catabolic Balance Compares Production of DHEA to Cortisol

If a patient has low Total DHEA Production but high Metabolized Cortisol, they are likely in a more catabolic state. ***This is what we want to avoid!***

- It may be more difficult for this person to see gains in muscle mass, maintain muscle mass, exercise and recover from exercise, and recover from illnesses, injuries, and traumas, etc.
- They may experience immunological issues (e.g., get sick more often).





Salivary Cortisol and CVD Mortality: Whitehall II Prospective Cohort Study (2011)

Association of Diurnal Patterns in Salivary Cortisol with All-Cause and Cardiovascular Mortality: Findings from the Whitehall II Study

Meena Kumari, Martin Shipley, Mai Stafford, and Mika Kivimaki

Department of Epidemiology and Public Health, University College London, London WC1E 6BT, United Kingdom

TABLE 3. HR of all-cause, cardiovascular, and noncardiovascular mortality among 4047 participants of the Whitehall II study from phase 7 (2002–2004) through to January 2010 by z-scores of measures of cortisol

	All-cause mortality	Noncardiovascular deaths	Cardiovascular deaths
Waking cortisol	0.94 (0.80–1.12)	0.93 (0.77–1.13)	0.95 (0.67–1.36)
CAR	0.94 (0.80–1.12)	0.90 (0.74–1.10)	1.12 (0.79–1.57)
Slope across the day	1.30 (1.09–1.55)	1.17 (0.96–1.43)	1.87 (1.32–2.64)
Bedtime cortisol	1.33 (1.11–1.59)	1.17 (0.96–1.44)	1.98 (1.39–2.81)

Slope across the day and CV deaths: Z-score: 1.87 ~ p = 0.03 (P < 0.05)
Bedtime cortisol and CV deaths: Z-score: 1.98 ~ p = 0.02 (P < 0.05)

- **First study** to document that daily salivary diurnal cortisol patterns are **predictive of subsequent CV mortality** in men and women
- **Study:** 4047 men and women, average age 61, mean FU 6.1 years
- **Objective:** to examine the association between cortisol patterns, CV and non-CV mortality

Kumari M, et al. J Clin Endocrinol Metab. 2011; 96(5): 1478-1485.

Salivary Cortisol and CVD Mortality: Whitehall II Prospective Cohort Study (2011)

Association of Diurnal Patterns in Salivary Cortisol with All-Cause and Cardiovascular Mortality: Findings from the Whitehall II Study

Meena Kumari, Martin Shipley, Mai Stafford, and Mika Kivimaki

Department of Epidemiology and Public Health, University College London, London WC1E 6BT, United Kingdom

TABLE 3. HR of all-cause, cardiovascular, and noncardiovascular mortality among 4047 participants of the Whitehall II study from phase 7 (2002–2004) through to January 2010 by z-scores of measures of cortisol

	All-cause mortality	Noncardiovascular deaths	Cardiovascular deaths
Waking cortisol	0.94 (0.80–1.12)	0.93 (0.77–1.13)	0.95 (0.67–1.36)
CAR	0.94 (0.80–1.12)	0.99 (0.74–1.19)	1.12 (0.79–1.57)
Slope across the day	1.30 (1.09–1.55)	1.17 (0.96–1.43)	1.87 (1.32–2.64)
Bedtime cortisol	1.33 (1.11–1.59)	1.17 (0.96–1.44)	1.98 (1.39–2.81)

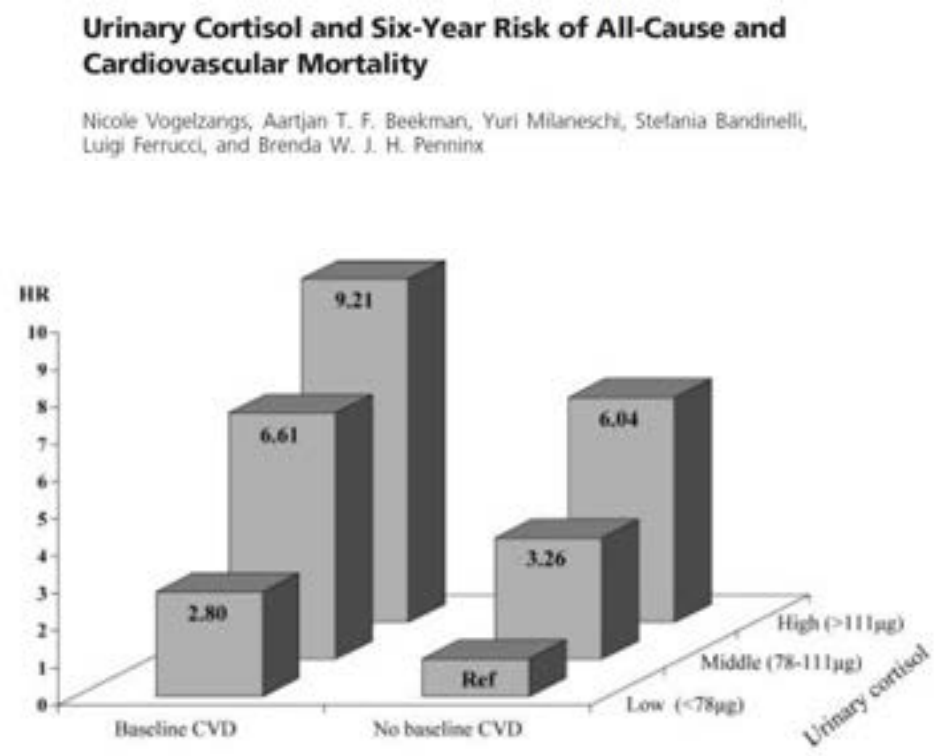
- Slope across the day and CV deaths: Z-score: 1.87, p = 0.03 (P < 0.05)
- Bedtime cortisol and CV deaths: Z-score: 1.98, p = 0.02 (P < 0.05)

- **Results:** A **flattened cortisol curve** was SS associated with **increased CV mortality; elevated PM cortisol was an independent predictor of subsequent CV mortality**
  - No association between waking cortisol, CAR, and mortality

- **Conclusion: A flattened cortisol curve and elevated PM cortisol levels are robust CV mortality predictors in middle-aged adults**

Kumari M, et al. J Clin Endocrinol Metab. 2011; 96(5): 1478-1485.

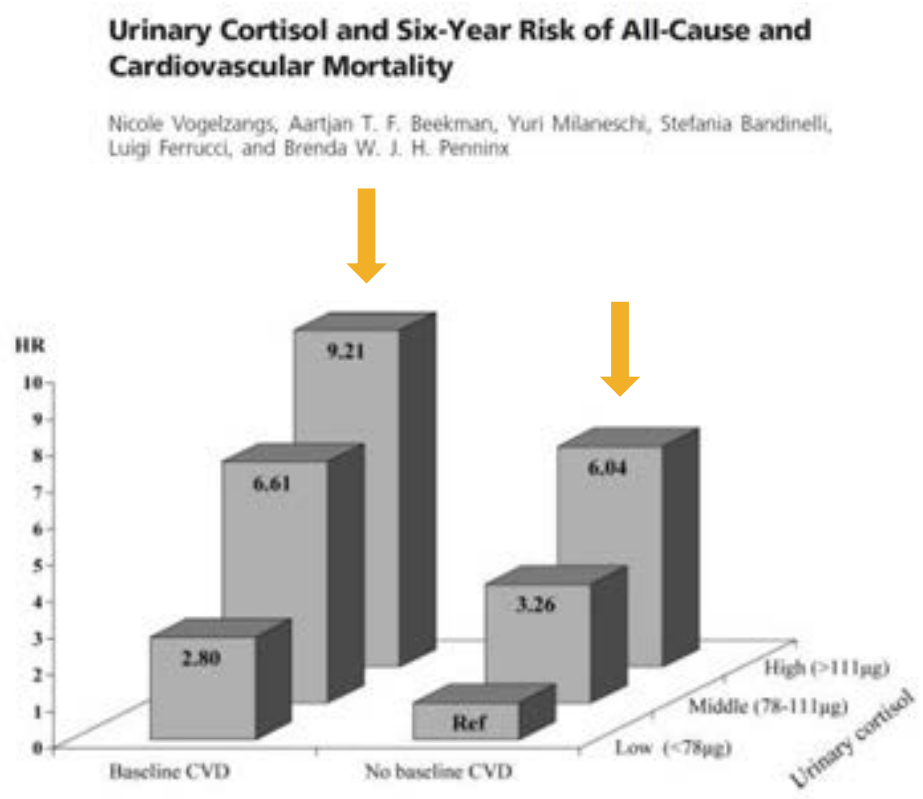
Urinary Cortisol and CVD Mortality: InCHIANTI, a Prospective Cohort Study (2010)



- **First urine study** to document that 24-hour urinary free cortisol (UFC) levels predict CV mortality
- **Study:** 862 older individuals, mean age 74, 55% women; 6-year study; samples at baseline
  - UFC divided into 3 terciles: low < 78μg; moderate: 78-111μg; high: > 111μg
- **Objective:** To determine whether 24-hour UFC levels predict all-cause and CV mortality

Vogelzangs N, et al. J Clin Endocrinol Metab. 2010; 95(11): 4959-4964.

Urinary Cortisol and CVD Mortality: InCHIANTI, a Prospective Cohort Study (2010)



- **Results:** UFC **strongly predicts CV mortality**, not non-CV mortality in persons with and without baseline CVD
  - Risk increased with increasing UFC levels
  - Those in the highest tercile had a 5x increased CVD mortality risk over 6 years
    - No baseline CVD: 6x increased risk of dying from CVD
    - Baseline CVD: 9.2x increased risk of dying from CVD

- **Conclusion: UFC is a strong CVD mortality predictor in persons with and without baseline CVD**

Vogelzangs N, et al. J Clin Endocrinol Metab. 2010; 95(11): 4959-4964.

More Cortisol/CVD Studies Since Whitehall

1. Association of Diurnal Patterns in Salivary Cortisol With All-Cause and Cardiovascular Mortality: Findings From the Whitehall II Study. The Journal of Clinical Endocrinology and Metabolism. 2011. Kumari M, Shipley M, Stafford M, Kivimaki M.

2. Dysregulated Diurnal Cortisol Patterns Are Associated With Cardiovascular Mortality: Findings From the KORA-F3 Study.Psychoneuroendocrinology. 2022. Karl S, Johar H, Ladwig KH, Peters A, Lederbogen F. (Observational)

3. Diurnal Cortisol Features With Cardiovascular Disease in Hypertensive Patients: A Cohort Study. European Journal of Endocrinology. 2022. Gan L, Li N, Heizati M, et al.

4. Diurnal Cortisol Rhythm Is Associated With Adverse Cardiac Events and Mortality in Coronary Artery Bypass Patients. The Journal of Clinical Endocrinology and Metabolism. 2015. Ronaldson A, Kidd T, Poole L, et al.

5. Prolonged Hypothalamic-Pituitary-Adrenal Axis Activation After Acute Coronary Syndrome in the GENESIS-PRAXY Cohort. European Journal of Preventive Cardiology. 2018. Tang AR, Rabi DM, Lavoie KL, et al.

6. Lower Diurnal HPA-axis Activity in Male Hypertensive and Coronary Heart Disease Patients Predicts Future CHD Risk. Frontiers in Endocrinology. 2023. Degroote C, von Känel R, Thomas L, et al.

7. Salivary Cortisol Is Related to Atherosclerosis of Carotid Arteries. The Journal of Clinical Endocrinology and Metabolism. 2008. Dekker MJ, Koper JW, van Aken MO, et al.

8. Diurnal Pattern of Salivary Cortisol and Progression of Aortic Stiffness: Longitudinal Study. Psychoneuroendocrinology. 2021. Ikeda A, Steptoe A, Shipley M, et al.

9. Cortisol and Cardiometabolic Disease: A Target for Advancing Health Equity. Trends in Endocrinology and Metabolism: TEM. 2022. Ortiz R, Kluwe B, Lazarus S, Teruel MN, Joseph JJ. (Review)

10. Cardiac Phenotypes in Secondary Hypertension: JACC State-of-the-Art Review. Journal of the American College of Cardiology. 2022. Januszewicz A, Mulatero P, Dobrowolski P, et al. (Review)

11. Cardiovascular Events and Mortality in Patients With Adrenal Incidentalomas That Are Either Non-Secreting or Associated With Intermediate Phenotype or Subclinical Cushing's Syndrome: A 15-Year Retrospective Study. The Lancet. Diabetes & Endocrinology. 2014. Di Dalmazi G, Vicennati V, Garelli S, et al. (Observational)

Cortisol Key Points re: Cardiac Risk

- Cortisol production is a sign of inflammation
- Chronic stress is significantly associated with HPA axis dysfunction and with MI risk
- Cortisol is a strong predictor of CVD risk, events, and mortality
  - **Salivary** flattened diurnal cortisol pattern and/or high PM cortisol
  - **Urinary Cortisol**: elevated 24-hour UFC strong predictor of CVD mortality in persons with and without preexisting CVD

- **How do we measure this risk?**
  - Saliva and cortisol awakening response (CAR)
  - Urine free cortisol and cortisol/cortisone metabolites
  - For Anabolic/Catabolic balance, we assess Total DHEA Production against Total Cortisol Metabolites

Kumari M, et al. J Clin Endocrinol Metab. 2011; 96(5): 1478-1485.
Vogelzangs N, et al. J Clin Endocrinol Metab. 2010; 95(11): 4959-4964.

General HPA Axis Support

- Correct insulin resistance.
- Encourage weight loss if appropriate.
- Engage in regular aerobic exercise.
- Lower inflammation.
- Lower stress and support parasympathetic activity.
- Manage acute and chronic pain.
- Manage chronic infections.
- Minerals, including magnesium and zinc (balance copper).
- Nutritional and herbal adaptogens (see next page).
- Optimize sleep and the circadian rhythm.

- Probiotics such as Bifidobacterium longum 1714 and Lactobacillus plantarum PS128
- Vitamins, including B vitamins and vitamin C

Adaptogens

Adaptogens are herbs, roots, and other plant substances (like mushrooms) that help the body respond to stress.

Common Name	Latin Name
Ashwagandha	Withania somnifera
Astragalus	Astragalus spp.
Bacopa	Bacopa monnieri
Cordyceps	Cordyceps sinensis or militaris
Fo-ti	Polygonum multiflorum
Gotu kola	Centella asiatica
Holy basil	Ocimum tenuiflorum
Jujube	Ziziphus jujuba
Korean Ginseng	Panax ginseng
Licorice root[1]	Glycyrrhiza glabra
Maca	Lepidium spp.
Magnolia bark	Magnolia officinalis

Common Name	Latin Name
Maitake	Grifola frondose
Mimosa	Albizia julibrissin or lebbeck
Rehmannia	Rehmannia spp.
Reishi	Ganoderma lucidum
Rhodiola	Rhodiola rosea
Schisandra	Schisandra chinensis
Shatavari	Asparagus racemosus
Shiitake	Lentinula edodes
Siberian Ginseng	Eleutherococcus senticosus
Skullcap	Scutellaria lateriflora
Turmeric	Curcuma longa

* Studied for cardiovascular outcomes

References for Cardiovascular Adaptogens

- 1. Pittler MH, et al. Hawthorn Extract for Treating Chronic Heart Failure. The Cochrane Database of Systematic Reviews. 2008.
- 2. Vogel JH, et al. Integrating Complementary Medicine Into Cardiovascular Medicine. A Report of the American College of Cardiology Foundation Task Force on Clinical Expert Consensus Documents (Writing Committee to Develop an Expert Consensus Document on Complementary and Integrative Medicine). Journal of the American College of Cardiology. 2005.
- 3. Liperoti R, et al. Herbal Medications in Cardiovascular Medicine. Journal of the American College of Cardiology. 2017.
- 4. De Smet PA. Herbal Remedies. The New England Journal of Medicine. 2002.
- 5. Jafari A, Mardani H, Abbastabar M, et al. The Effect of Ginseng Supplementation on Cardiovascular Disease Risk Factors: A Comprehensive Systematic Review and Dose-Response Meta-Analysis. The British Journal of Nutrition. 2025.
- 6. Chen Y, et al. Rhodiola rosea: A Therapeutic Candidate on Cardiovascular Diseases. Oxidative Medicine and Cellular Longevity. 2021.
- 7. Han X, et al. Effect of Astragalus Membranaceus on Left Ventricular Remodeling in HFrEF: A Systematic Review and Meta-Analysis. Frontiers in Pharmacology. 2023.
- 8. Nobari H, et al. Ashwagandha (Withania Somnifera): A Comprehensive Narrative Review of Its Role in Enhancing Physical Performance and Health. Nutrition & Metabolism. 2026.
- 9. Chen P, et al. Beneficial Effects of Schisandrin B on the Cardiac Function in Mice Model of Myocardial Infarction. PloS One. 2013.
- 10. Chen X, et al. Ethanol Extract of Schisandrae Chinensis Fructus Ameliorates the Extent of Experimentally Induced Atherosclerosis in Rats by Increasing Antioxidant Capacity and Improving Endothelial Dysfunction. Pharmaceutical Biology. 2018.
- 11. Li Z, et al. Integrated Network Pharmacology and Chinmedomics Reveal the Molecular Mechanisms of Aqueous Extract of Eleutherococcus Senticosus Fruit for Treating Essential Hypertension. Journal of Ethnopharmacology. 2026.
- 12. Yang K, et al. A Comprehensive Review of Ethnopharmacology, Phytochemistry, Pharmacology, and Pharmacokinetics of Schisandra Chinensis (Turcz.) Baill. And Schisandra Sphenanthera Rehd. Et Wils. Journal of Ethnopharmacology. 2022.

Low DHEA and High Cortisol – 35 Year-Old Female

Catabolic or Anabolic?

What if salivary cortisol looks like this?

More worried or less worried now?

Assessing Androgens in Mid-Life Men and Women

Comprehensive Androgen Assessment

What should a complete androgen assessment include?

Likely both serum and urine testing

Measuring Androgens: **Serum**

- Total Testosterone (LC-MS/MS for women and hypogonadal men)
- Free Testosterone (calculated*)
- 5a-DHT
- DHEA-S

***Note:** Calculated free testosterone (Vermeulen equation), derived from total testosterone (LC-MS/MS), SHBG, and albumin, is generally preferred over the standard direct free testosterone immunoassays because it provides a more accurate estimate of free testosterone in most patients.*

Measuring Markers that can Affect Androgen Levels: **Serum**

- LH/FSH
- SHBG
- Thyroid Panel (TSH, free T4, free T3, rT3, antibodies, etc.)
- AM Cortisol
- Blood sugar (fasting glucose and insulin, HbA1c, HOMA-IR, etc.)
- Prolactin, 17-OHP, IGF-1, etc.

Measuring Androgens: **Dried Urine**

Testosterone Metabolites
- Testosterone
- 5a-DHT
- 5a-androstanediol
- 5b-androstanediol

DHEA Metabolites
- DHEA-S
- Androsterone
- Etiocholanolone

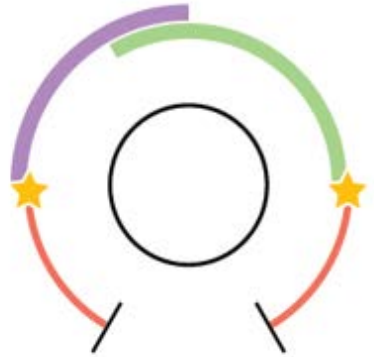
Epi-testosterone (not an active androgen; measured to double check urine testosterone levels – ie: ART, gonadal output assessment)

Range-Embedded Dials (on the DUTCH Test)

Male Androgens

- Normal range, or Age 18-40
- Age 41-60+
- Edge of range

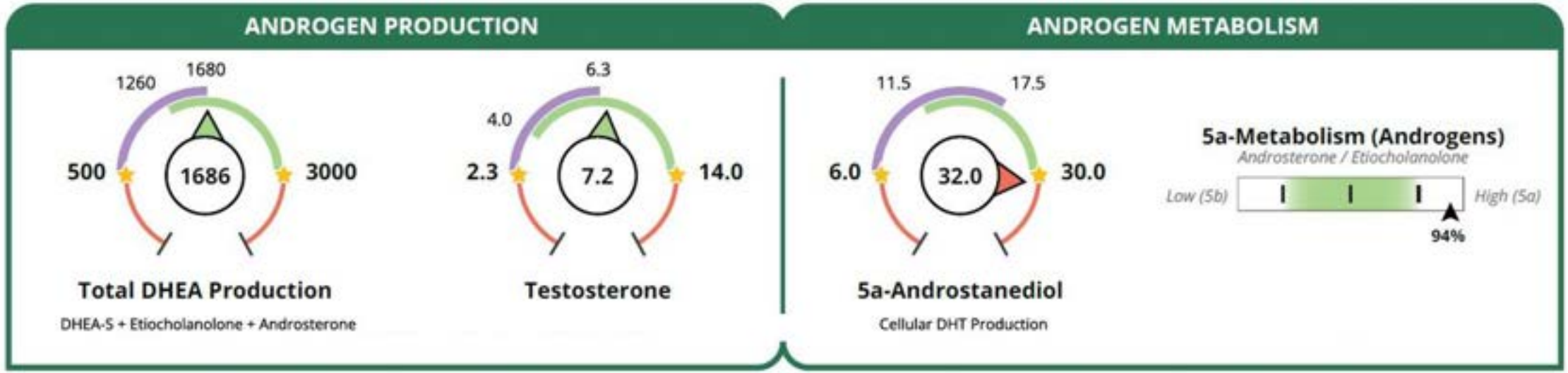
Female Androgens



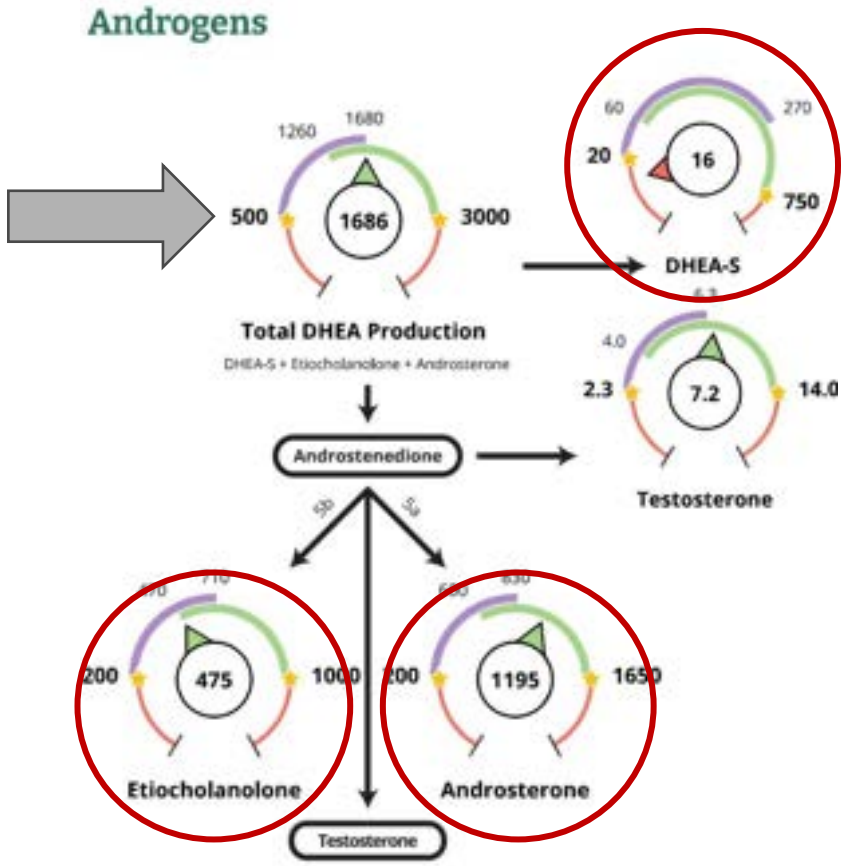
Key to Reading the Dial

- Optimal Premenopausal
- Postmenopausal Range
- Out of Range
- Edge of Range

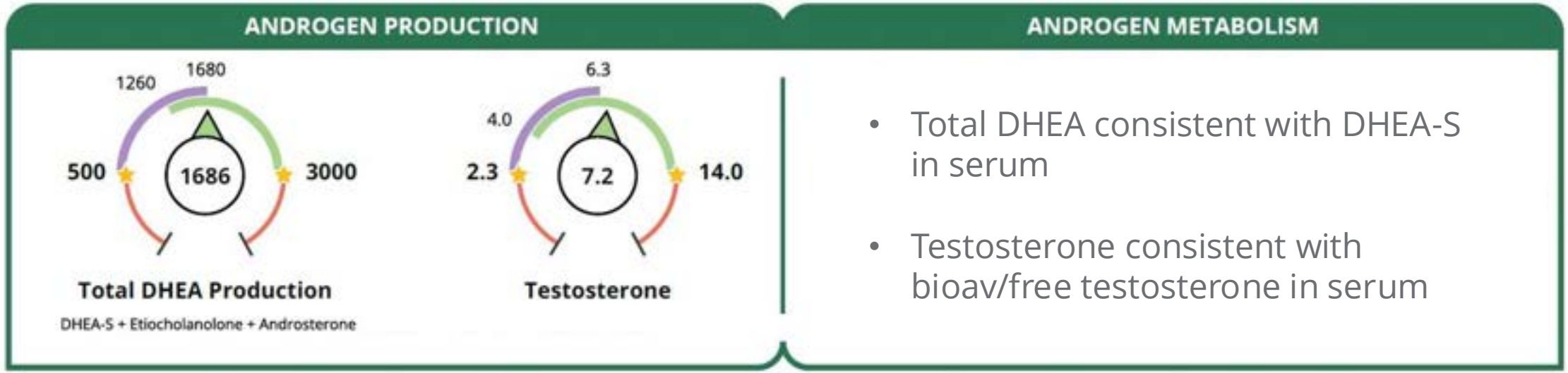
The Androgen Story in Dried Urine



In Urine, Total DHEA Production = DHEA-S + Androsterone + Etiocholanolone



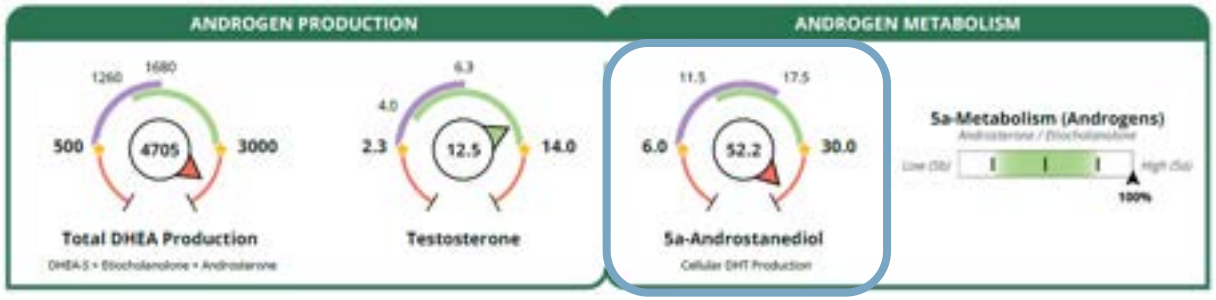
The Androgen Story in Dried Urine



Testosterone›s Metabolites – 5a-R Metabolites Do Testosterone›s Work

- 5a-DHT – **high impact at androgen receptor**
  - Primarily in target tissues by the enzyme 5a-reductase
  - **3x more potent than testosterone**
  - When DHT is robust, it can help with skeletal muscle turnover
  - , increasing muscle development and improving strength.
  - DHT damages hair follicles, increasing body hair and decreasing scalp hair growth.
  - DHT can cause cystic acne development.
  - DHT can act as a neuro-steroid and increase agitation.

- 5a-Androstanediol – **marker of DHT formation, intact/unaffected by UGT deletion**
  - Primarily formed from DHT in target tissues
  - Weakly binds androgen receptors
  - More persistent marker for TISSUE 5a-DHT activity than urinary 5a-DHT itself
  - Correlates with high androgenic symptoms such as acne, body hair, oily skin, irritability, and scalp hair loss.

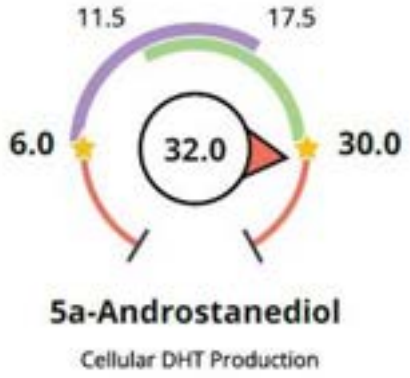


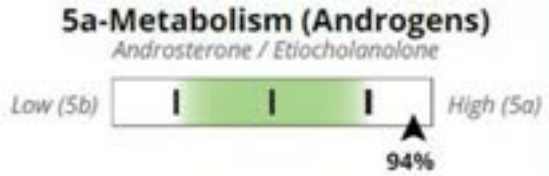
The Androgen Story in Dried Urine

ANDROGEN PRODUCTION

- 5a-Androstanediol represents 5a-DHT activity at the tissue level
- 5a-Metabolism represents 5a-Reductase activation of DHEA to Androsterone

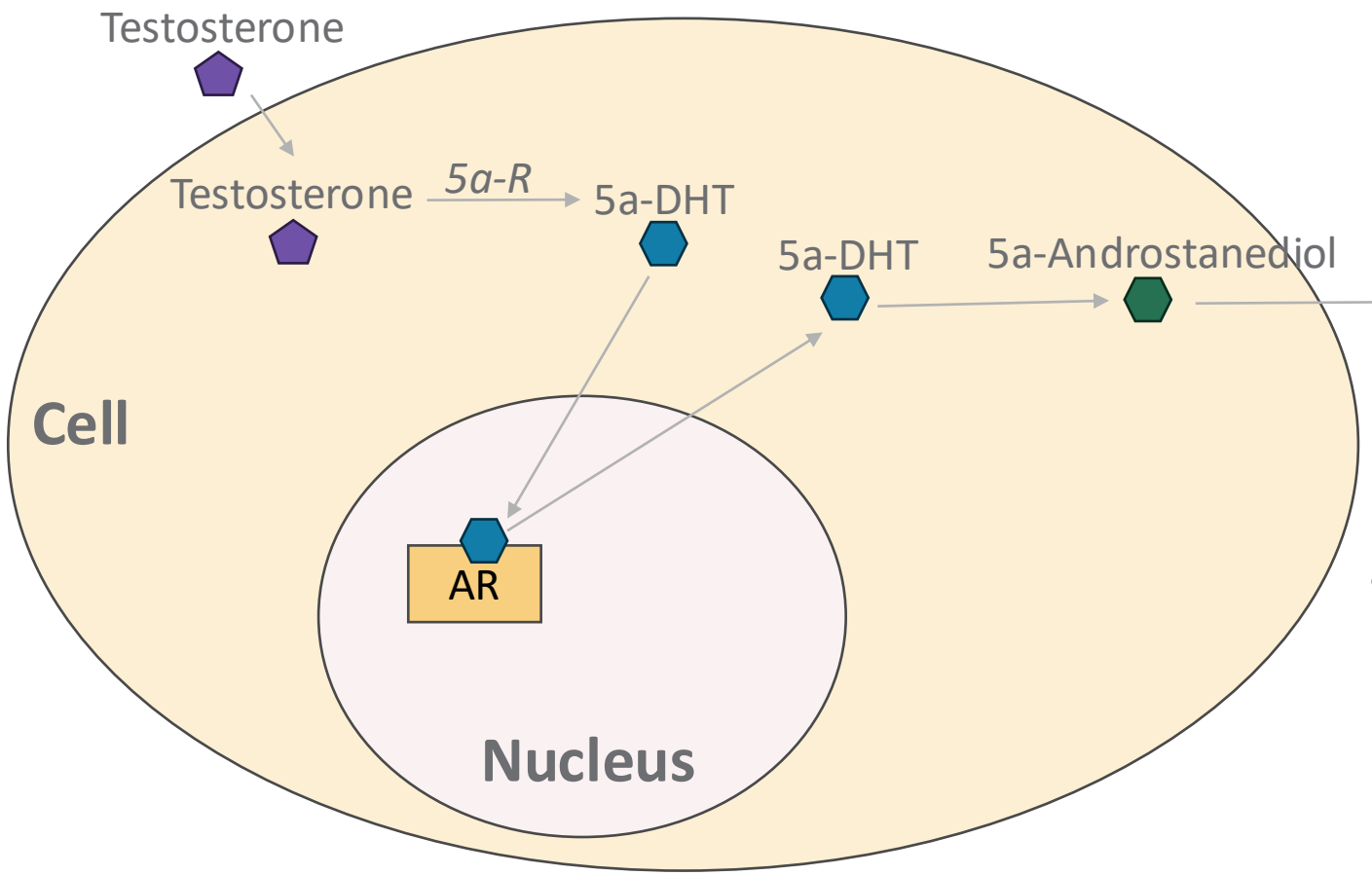
ANDROGEN METABOLISM





The DUTCH Dozen: 7 5a-Androstanediol

- 5a-DHT is used in the tissues and **converted to 5a-Androstanediol**.

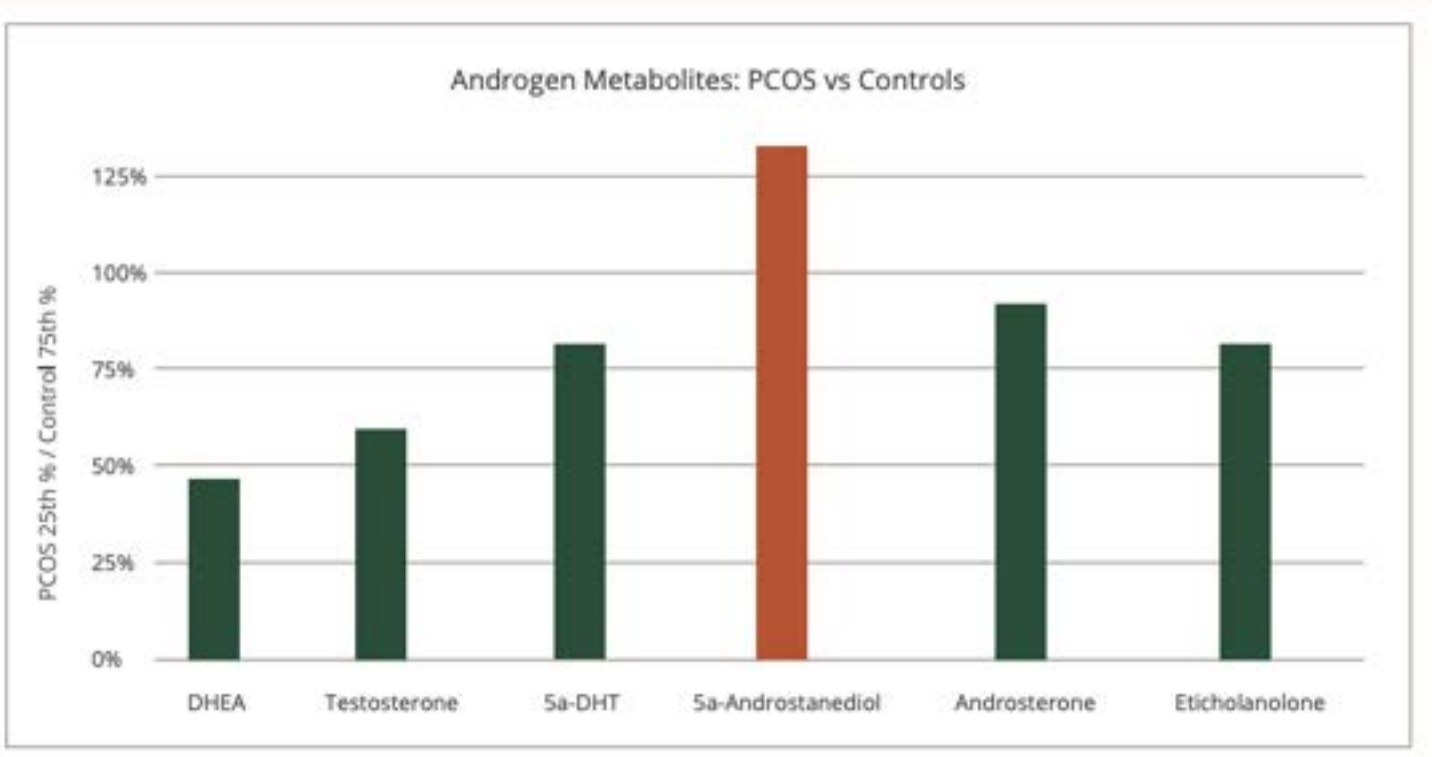


- 5a-Androstanediol circulates more so than 5a-DHT.
- Therefore, **5a-Androstanediol may be a better indicator of 5a-DHT activity in the tissues than 5a-DHT itself**.

Extracellular Matrix (ECM)

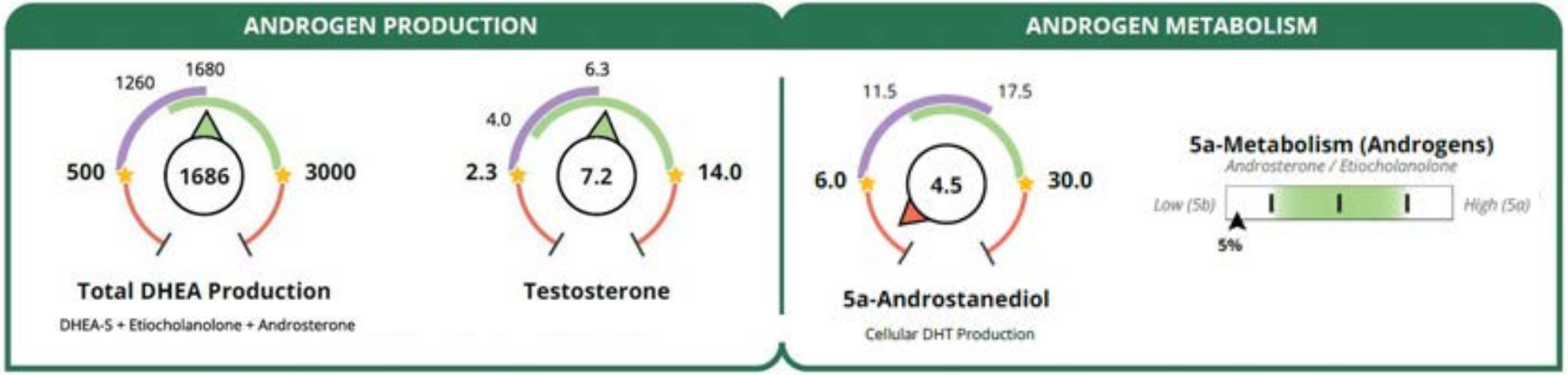
5a-Androstanediol is the most differentiating androgen metabolite

Figure 2: *An analysis of androgen metabolites in 24-hour urine (nmol/24h) in women with or without PCOS shows that, for 5a-androstanediol, the 25th percentile value for PCOS patients is more than 125% of the 75th percentile value for normal controls. Essentially, even the lower end of the PCOS range (25th percentile) exceeds the upper end of the normal range (75th percentile) for healthly controls, suggesting no overlap between the normal range and the PCOS range for 5a-androstanediol. [98]*



Dhayat NA, Marti N, Kollmann Z, et al. Urinary steroid profiling in women hints at a diagnostic signature of the polycystic ovary syndrome: A pilot study considering neglected steroid metabolites. PLOS ONE. 2018; 13(10):e0203903. doi:10.1371/journal.pone.0203903

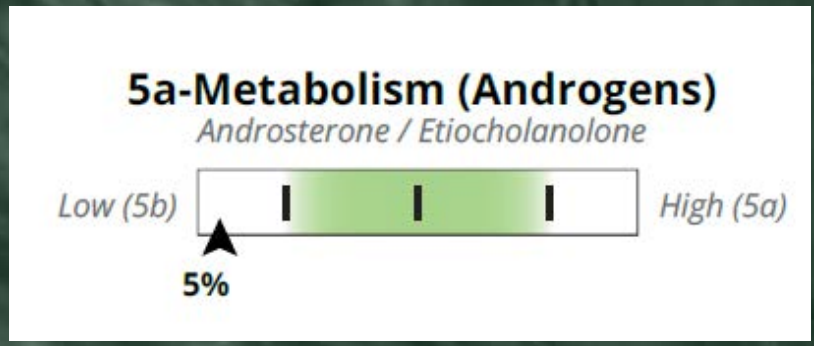
Urine Captures Tissue Androgen Activity



The ISSUE is the TISSUE

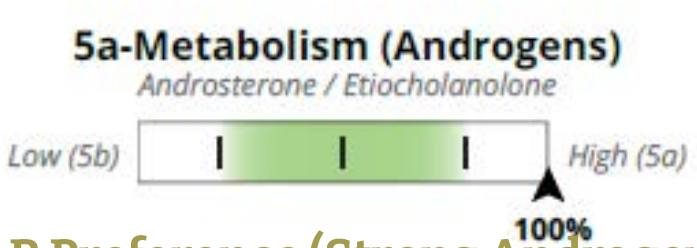
- 5a-Androstanediol best represents tissue DHT
- Parent hormones AND metabolites matter

Low 5a-Reductase Activity Can Worsen Symptoms of Declining Androgens

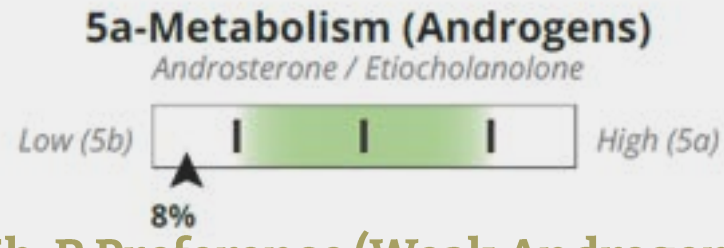


5a-Metabolism Summary

- **5a-reductase** is the enzyme that metabolizes DHEA and testosterone into their **active, androgenic metabolites**.
  - High 5a-Reductase activity associated with acne, scalp hair loss, irritability, facial hair
- Low 5a-reductase activity may be associated with:
  - **Autoimmune disease**
  - **Thyroid disease**
  - **Medications (ie: Finasteride, Dutasteride, Saw Palmetto, Reishi, EGCG, Nettle Root)**
- 5b-reductase preference may lead to low androgen symptoms even when DHEA and Testosterone are within normal ranges.



5a-R Preference (Strong Androgens)



5b-R Preference (Weak Androgens)

Decreasing a 5b-Reductase Preference

In addition to treating the underlying cause (see the DUTCH Interpretive Guide), other potential support considerations for decreasing a 5b-reductase preference (by supporting more 5a-reductase activity) in females and males include:

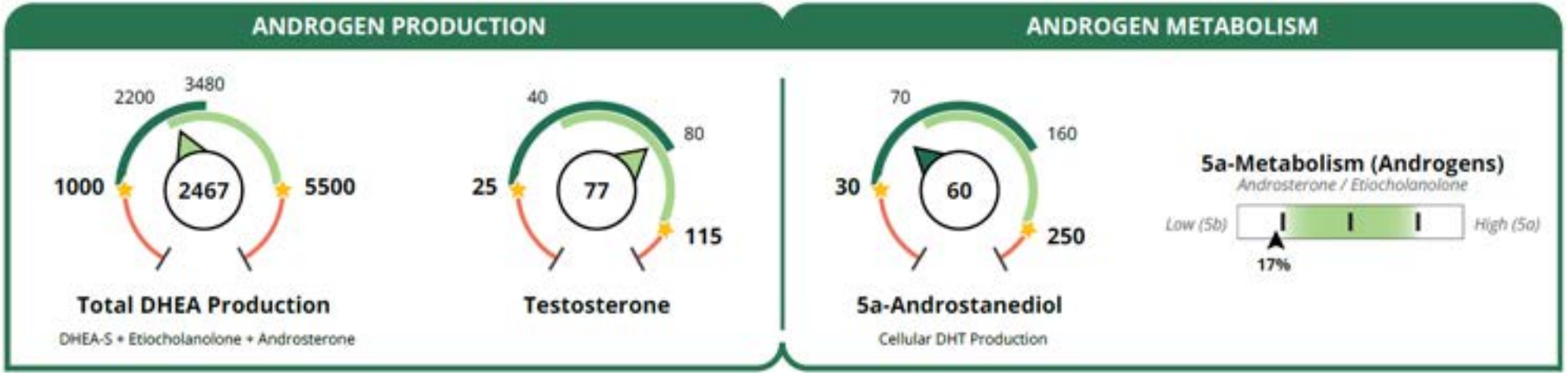
Lifestyle and Diet

- Forskolin
- High intensity interval training (HIIT)
- Pine pollen
- Weight resistance exercises

Avoid 5a-Reductase Blockers

- Beta-sitosterol
- Epigallocatechin gallate (EGCG) from green tea
- Polyunsaturated fatty acids
- Pygeum
- Reishi
- Saw palmetto
- Stinging nettle root
- Zinc (balance copper)
- Rx: Finasteride[6]

What is the androgen status? 57 Yr-Old Male on TD TRT



- This 57yoM struggles with **continued fatigue, low libido, and low mood despite starting transdermal testosterone 3 months ago**. Why is he not responding?
- DHEA and Testosterone both look great for his age but….
  - **5a-Androstanediol**
    - Testosterone's formation of DHT is low compared to testosterone consistent with 5b preference
  - **5a-Reductase Activity Slider shows low 5a-R activity**
    - 5b preference confirms a low tissue androgenic activity

- **Assessment: Normal DHEA and T on therapy but low tissue activation of androgens (low 5a-Metabolism)**

Important Caveat to Interpreting Testosterone in Urine Tests

UGT2B17 Deletion

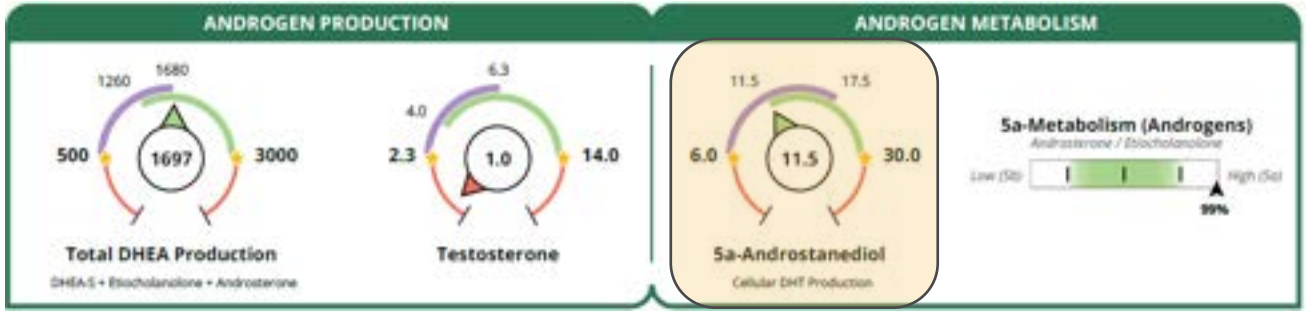
UGT2B17 Gene Deletion

- For those carrying this deletion, urinary testosterone will not correlate with circulating testosterone levels.
- It also diminishes DHT and 5b-Androstanediol in urine.
- Testosterone : Epi-Testosterone ratio is low with UGT2B17 del.
- **Use serum for assessing testosterone in these patients.**

Low →	Testosterone	Below range	1.04	ng/mg	2.3 - 14
Low →	5a-DHT	Within range (But low edge)	0.4	ng/mg	0 - 6.6
	5a-Androstanediol	Within range	11.5	ng/mg	6 - 30
Low →	5b-Androstanediol	Below range	8.3	ng/mg	12 - 75
	Epi-Testosterone	Within range	13.0	ng/mg	2.3 - 14

UGT2B17 Gene Deletion

- 5a-Androstanediol is unaffected, so use it to assess tissue DHT in UGT del carriers.

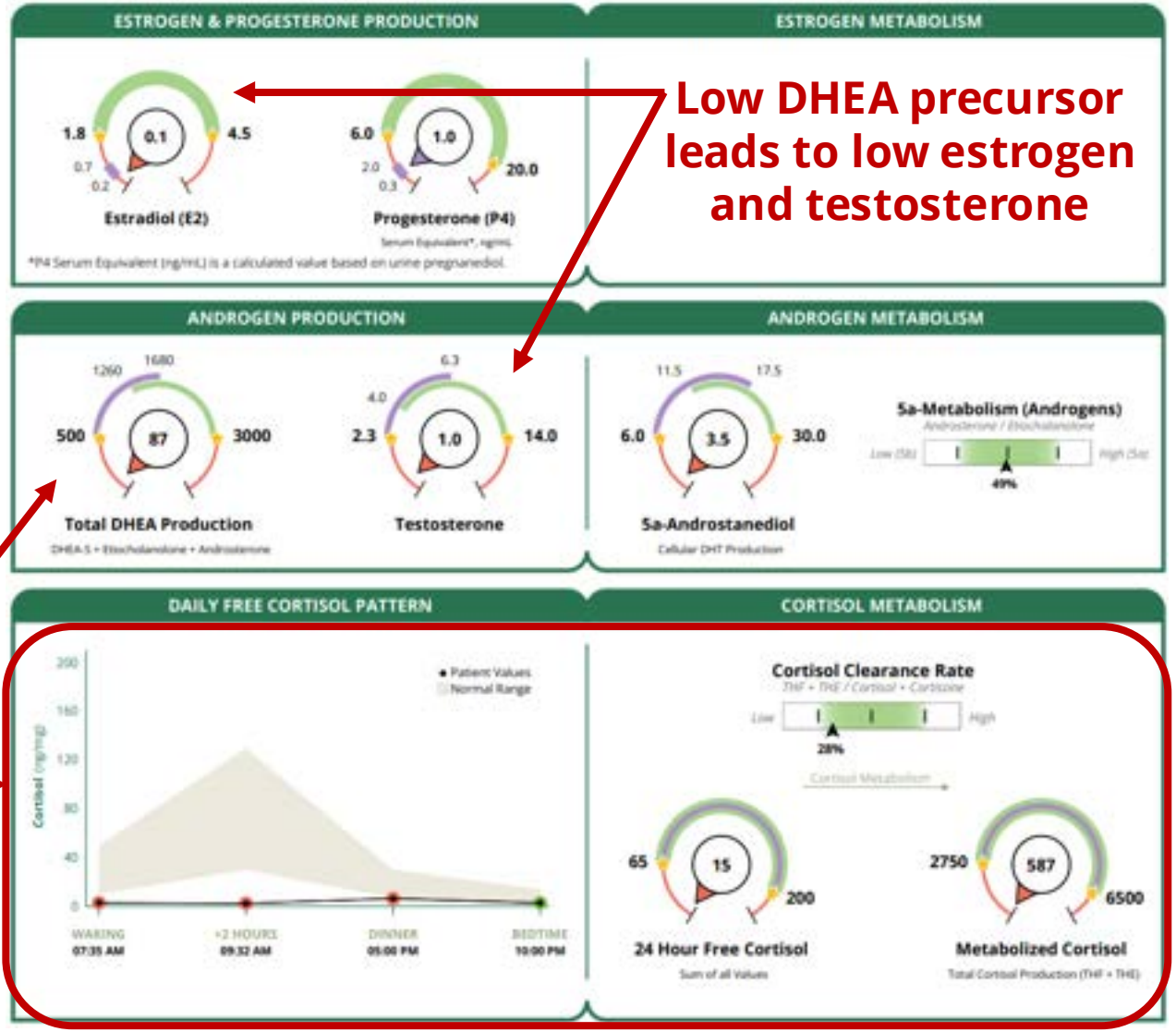


Testosterone	Below range	1.04	ng/mg	2.3 - 14
5a-DHT	Within range	0.4	ng/mg	0 - 6.6
→ 5a-Androstanediol	Within range	11.5	ng/mg	6 - 30
5b-Androstanediol	Below range	8.3	ng/mg	12 - 75
Epi-Testosterone	Within range	13.0	ng/mg	2.3 - 14

62 Year Old Female on Oral Prednisone

Fatigue, sleep disturbed, low libido

62-Year-Old Postmenopausal Female on Oral Prednisone



Low DHEA precursor leads to low estrogen and testosterone

Adrenal Suppression

What hormone might she need to replace while she's on prednisone?

DHEA!

49 Year Old Female in Late Perimenopause

Case: Late Perimenopause

49 Year Old Female

LMP > 6 months prior to collecting samples

Has been using Equol to control hot flashes but it has stopped working well

Difficulty getting to sleep and staying asleep due to hot flashes, feels restless nightly and wakes tired x 1 year

Libido has decreased significantly x 5 years

Weight gain 3-5 pounds per year over the past x 2 years

BMI is normal

Late Perimenopause Baseline

Pertinent Baseline serum labs:

- FSH: 27 mIU/L

All other findings within healthy norms including thyroid panel, CBC, CMP, lipids, and hs-CRP.

Late Perimenopause Baseline

Hot flashes and sleep issues likely due to low Estrogen and Progesterone

Rx ideas?

E2 Patch 0.0375 mg
OMP 100 mg
Phase One Support w/ glucoraphanin

Late Perimenopause Baseline

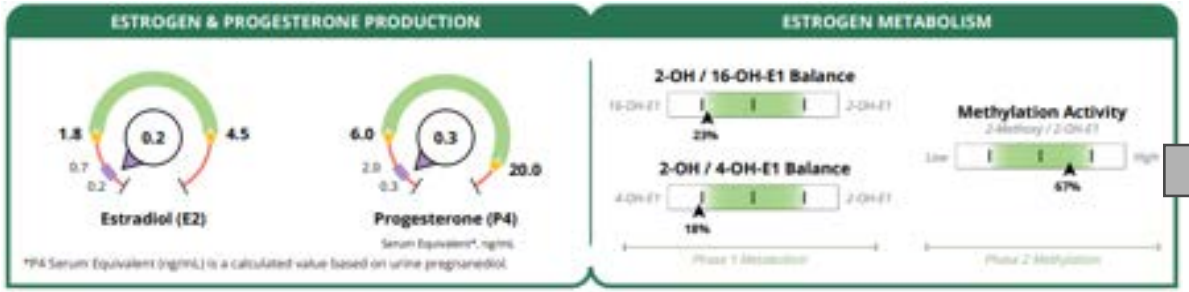
Hot flashes and sleep issues likely due to low Estrogen and Progesterone

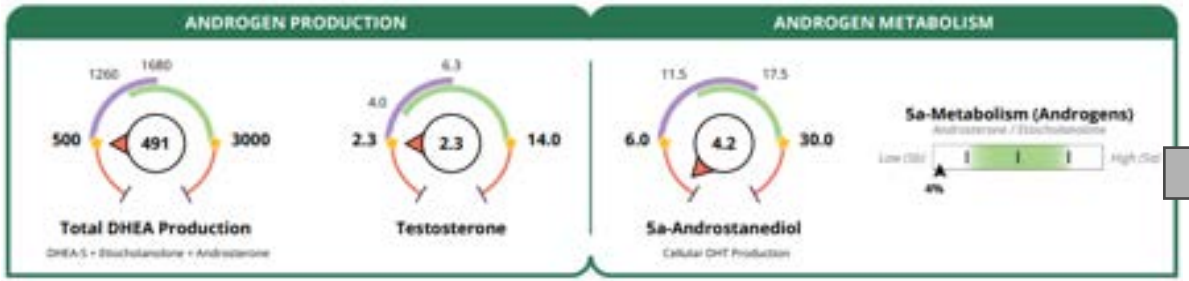
Rx ideas?
E2 Patch 0.0375 mg
OMP 100 mg
Phase One Support w/ glucoraphanin

Low libido and weight gain could be stemming from low androgens and 5B preference

Rx ideas?
DHEA 10 mg
Weight Training 2x wk, HIIT 2x wk

Late Perimenopause Baseline





Hot flashes and sleep issues likely due to low Estrogen and Progesterone

Rx ideas? **E2 Patch 0.0375 mg**
OMP 100 mg
Phase One Support w/ glucoraphanin

Low libido and weight gain could be stemming from low androgens and 5B preference

Rx ideas? **DHEA 10 mg**
Weight Training 2x wk, HIIT 2x wk

Poor sleep is probably the chronic stressor leading to flattening of the cortisol pattern and low cortisol production

Low neurotransmitter metabolites common with low adrenal function – same drivers

Rx ideas? **Adaptogen Botanical Complex w/ Rhodiola and Maca**

Female - Non-Hormonal Therapies for Low Testosterone

LOW TESTOSTERONE IN FEMALES

In addition to treating the underlying cause (see the DUTCH Interpretive Guide), other potential support considerations for low testosterone in females include:

HPO Axis Support (Cycling Females)

- Treat energy deficit from anorexia, low calorie intake, low body weight, and/or extreme exercise.
- Optimize nutrition: B vitamins, choline, inositol, vit. D, and Zn.
- Reduce stress and support parasympathetic activity.
- Optimize sleep and the circadian rhythm.
- Treat hyperprolactinemia.
- Treat hypothyroidism.
- In cycling females, if estrogen and progesterone are also low, consider phytoestrogen and phytoprogestogen support. See
- Avoid endocrine disrupting chemicals (EDCs).

Androgen Supportive

- **Ashwagandha (A)**
- Damiana
- Epimedium
- Fenugreek
- Indian coleus
- **Korean Ginseng (A)**
- **Maca (A)**
- Mucuna[3]
- Sarsaparilla
- **Shatavari (A) (Females)**
- Tongkat Ali
- Tribulus
- Yohimbe

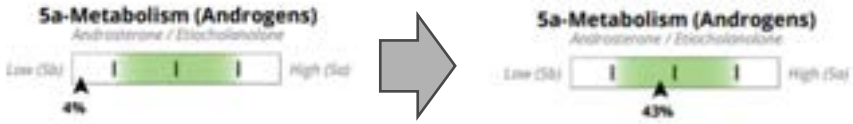
(A) Indicates an Adaptogen

Decreasing a 5b-Reductase Preference

In addition to treating the underlying cause (see the DUTCH Interpretive Guide), other potential support considerations for decreasing a 5b-reductase preference (by supporting more 5a-reductase activity) in females and males include:

Lifestyle and Diet

- Forskolin
- High intensity interval training (HIIT)
- Pine pollen
- Weight resistance exercises



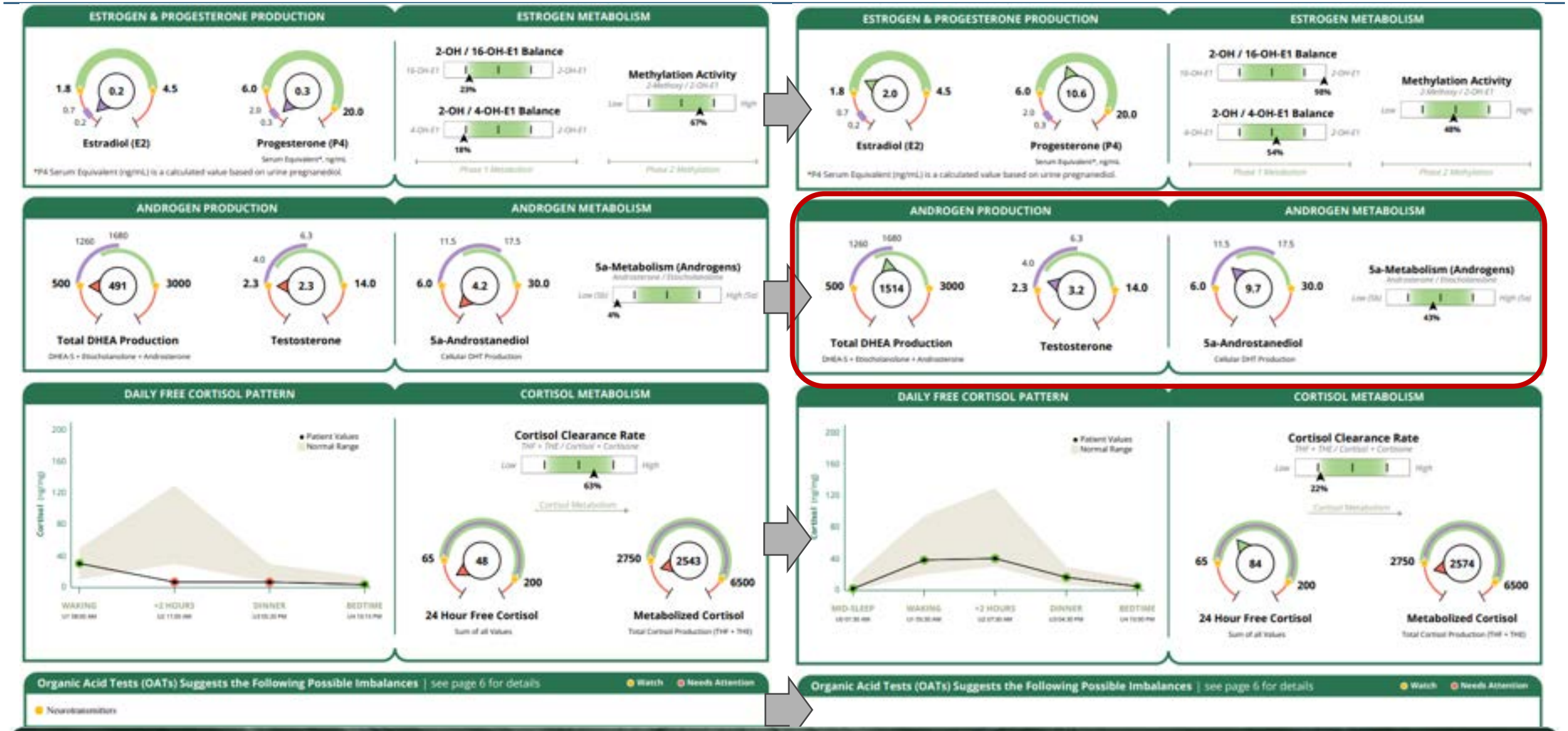
Avoid 5a-Reductase Blockers

- Beta-sitosterol
- Epigallocatechin gallate (EGCG) from green tea
- Polyunsaturated fatty acids
- Pygeum
- Reishi
- Saw palmetto
- Stinging nettle root
- Zinc (balance copper)
- Rx: Finasteride[6]

Female – Hormonal Supports for Low Androgens

Testosterone or Estradiol Pellet	Pellets offer consistent hormone dosing over time for testosterone and estradiol. Research is limited on effects on hot flashes and BMD. Because serum/urine E2 levels match or exceed those seen in patches, E2 pellets are likely to help with hot flashes and BMD.	**Low:** <5 mg Estradiol 20 - 50 mg Testosterone **High:** >12 mg Estradiol >125 mg Testosterone **Most Common:** 5 mg Estradiol 100 mg Testosterone *Inserted every 3 - 4 months*
Vaginal Estrogen or Testosterone	Low doses increase local tissue levels while higher doses also increase systemic levels. Placing in the top 1/3 of the vagina significantly increases uterine levels. Estriol often given in doses 1 - 4 times higher than estradiol. Studies have found that some FDA-approved low dose vaginal E2 formulations (e.g., 0.01 mg E2 insert) may be safe to use without progesterone in a woman with a uterus. Doses higher than this may require concomitant progesterone therapy to prevent endometrial hyperplasia and cancer.	**Low:** 0.01 mg Estradiol 0.25 mg Testosterone **High:** 0.5 mg Estradiol 2 mg Testosterone **Most Common:** 0.1 mg Estradiol 0.25 - 1.0 mg Estriol 0.25 - 1.0 mg Testosterone *Usually used 2-3 times per week at night but may be used nightly.*
Testosterone Cream/Gel	Transdermal testosterone can be used to correct low T and improve sex drive and muscle mass.	**Low:** 0.5 - 2.0 mg **High:** 10 - 20 mg **Most Common:** 1 - 5 mg *Taken daily, at waking or bedtime*
DHEA	Sublingual or oral DHEA will increase systemic levels and also contribute to downstream androgens (testosterone) and estrogens.	**Low:** 1 - 5 mg **High:** 25 - 50 mg **Most Common:** 5 - 10 mg *Usually taken daily*

Late Perimenopause Baseline vs Retest at 6 Months on Therapy



Low Libido and Metabolic Syndrome in a 49 yo Male

Case 2: 49-Year-Old Male Libido/Weight

Chief Complaints

- Low libido, erectile dysfunction
- Trouble losing weight
- Low mood, motivation
- Difficulty falling asleep, wakes
- Daytime fatigue

PMHx

- Prediabetes dx 2022
- Has gained 20 lbs. over the past 3 years
- Offered but refused to take statins

Baseline labs

- CBC & CMP: WNL except fasting glucose = 112
- HbA1c: 5.9
- Fasting insulin: 35
- Lipids: TG: 180, LDL: 120, HDL: 35
- TSH, fT4, fT3, TPO, rT3, anti TG: all WNL
- hs-CRP 2.7 (goal <1)
- SHBG: <15

Physical Exam

- 5'9"
- 202 lbs.
- BMI 29.8 (overweight)
- BP 138/87 mm Hg
- Pulse 82

Medications

- None

KEY = High end | Low end

Male Libido/Weight: Dried Urine Hormone Testing Summary

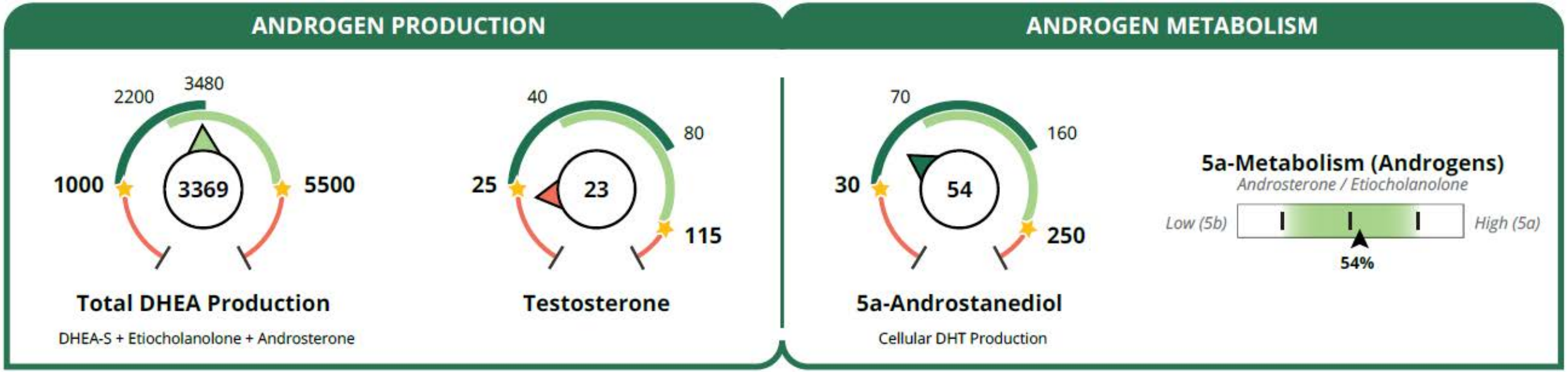
Androgens

Estrogens

Male Estrogens (left dial),
Male Androgens (right dial):

- Normal range, or Age 18-40
- Age 41-60+
- ★ Edge of range

Male Libido/Weight: Androgens



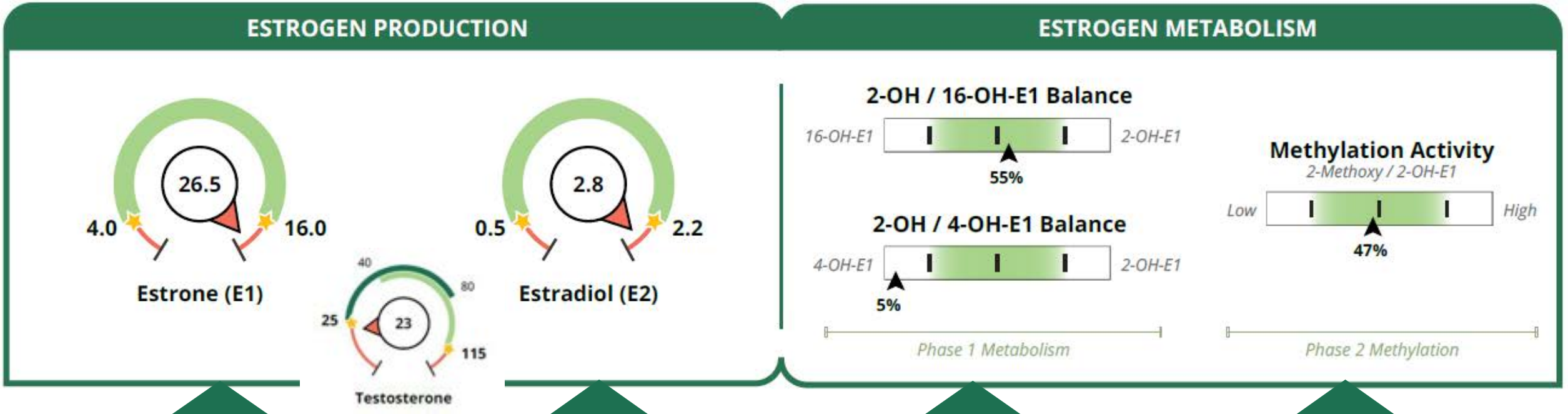
Assess adrenal androgen levels (Total DHEA)

Assess testosterone levels

Assess cellular production of 5a-DHT via 5a-Androstanediol

Assess if there is a preference for the more potent alpha metabolism of the androgens

Male Libido/Weight: Estrogen



Assess estrone level (adipose estrogens)

Assess estradiol level for estrogen dominance over testosterone

Assess 2-OH preference in phase 1 estrogen metabolism

Assess methylation of 2-OH estrogens

Non-Hormonal Options for Promoting Androgen Function in Men

Downregulate Aromatase

Slow aromatase activity if estrogen is high.

- Apigenin
- Chrysin
- Damiana
- Enterolactone
- Genistein
- Grape seed extract (GSE)
- Normalize body fat percentage
- Red wine procyanidin dimers
- Resveratrol
- White button mushroom
- Rx: Anastrozole

Improve Androgen Deficiency Symptoms

Note that these may improve androgen deficiency symptoms without necessarily increasing androgen levels.

- Exercise performance:
  - Korean ginseng
  - Maca
- Erectile function:
  - Arginine
  - Korean ginseng
  - Tribulus
  - Yohimbe
- Muscle mass:
  - Optimal protein consumption
  - Weightlifting

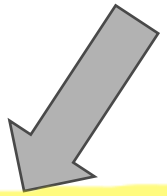
Male - Non-Hormonal Therapies for Low Testosterone

LOW TESTOSTERONE IN MALES

In addition to treating the underlying cause (see the DUTCH Interpretive Guide), other potential support considerations for low testosterone in males include:

HPT Axis Support

- Herbal and nutrient support:
  - Fenugreek
  - Indian coleus
  - Korean ginseng
  - Mucuna[3]
  - Shilajit
  - Tongkat ali
  - Tribulus
  - Vitamin D
  - Zn (balance Cu)



- Metabolic support:
  - Correct insulin resistance.
  - Eat a low glycemic and whole foods diet.
  - Exercise regularly.
  - Maintain healthy body weight.
- Lower stress and increase parasympathetic activity.

- Optimize sleep and the circadian rhythm.
- Decrease inflammation.
- Treat thyroid disorders if present.
- Treat hyperprolactinemia if present.

Avoid endocrine disrupting chemicals (EDCs).

Non-Hormonal Supports for Dopaminergic Tone and Androgens

Dopamine Support

Testosterone supports dopamine production. If symptoms of low dopamine are present, consider:

- Adequate dietary protein intake
- B vitamins
- D,L-phenylalanine (DLPA)[3]
- Mucuna[3] (contains L-DOPA)
- Tyrosine
- Vitamin C

Consider when HVA is low!

Clinical Pearl: HVA is often low in mid-life males with low androgen symptoms

Gonadal Support

- Indian coleus
- Mucuna[3]
- Treat nutrient deficiencies

Androgen Supportive

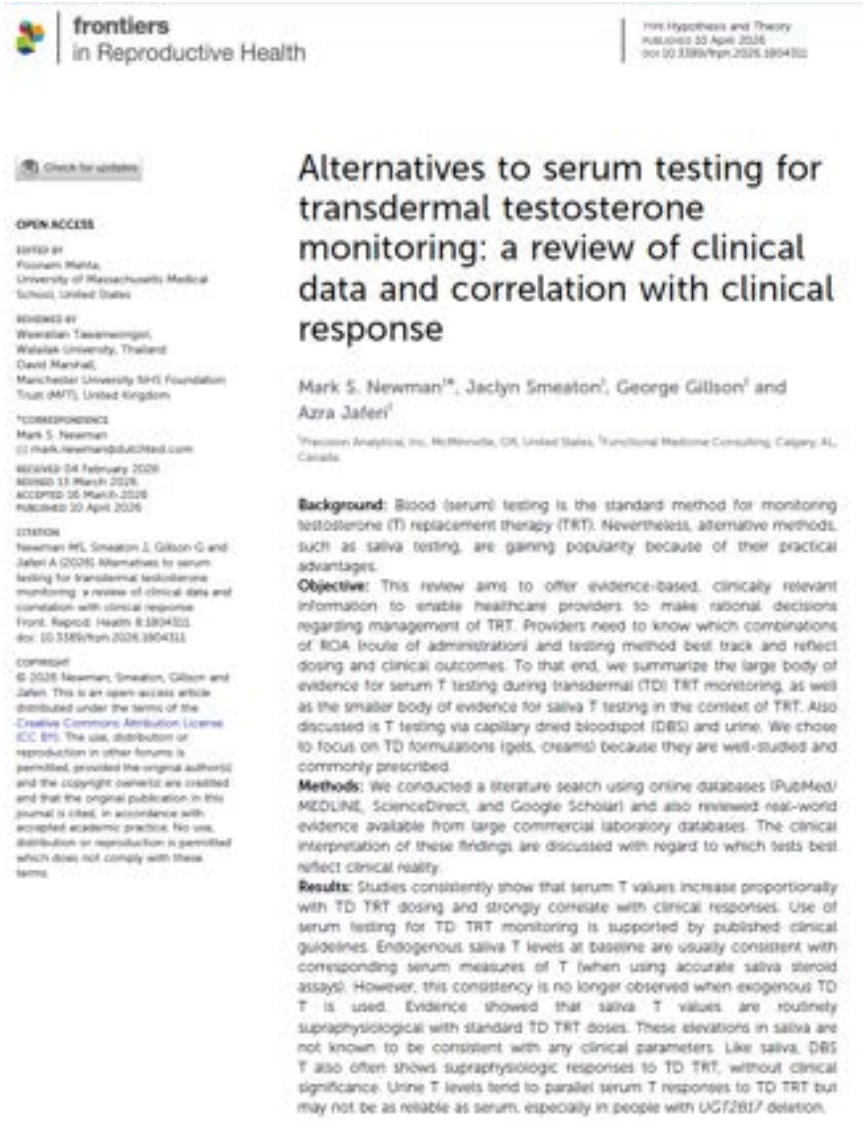
- **Ashwagandha (A)**
- Damiana
- Epimedium
- Fenugreek
- Indian coleus
- **Korean Ginseng (A)**
- **Maca (A)**
- Mucuna[3]
- Sarsaparilla
- **Shatavari (A) (Females)**
- Tongkat Ali
- Tribulus
- Yohimbe

(A) Indicates an Adaptogen

Male - Hormonal Therapies for Low Androgens

Testosterone Pellets	Testosterone pellets offer consistent hormone dosing over time. Most pellet doses tend to suppress endogenous testosterone production. They can be given with aromatase inhibitors if conversion to estrogen is a concern.	**Low:** 400 mg **High:** 1600 - 2000 mg **Most Common:** 800 - 1200 mg *Inserted every 4-6 months*
Testosterone Injections	The most frequently used testosterone injections are testosterone cypionate (8 day half-life) and testosterone enanthate (4-5 day half-life). Injections provide robust testosterone levels for 1-2 weeks typically. Bi-weekly dosing (with lower dosing than weekly injections) may offer improved steady state and less highs and lows.	**Low:** 25 - 100 mg **High:** >300 mg **Most Common:** 100 - 250 mg *Administered biweekly, or every one to two weeks*
Transdermal Testosterone	Testosterone creams and gels are the most popular TRT formulation but can be challenging to dose and monitor effectively. Doses between 50 and 150mg are commonly used in studies in order to see improvements in muscle mass and other clinical parameters. Application is convenient, but patients must also be careful to avoid transference (to partners, children, or pets).	**Low:** 25 - 75 mg **High:** 150 - 250 mg **Most Common:** 50 - 100 mg *Typically applied daily*
DHEA	Even though testosterone is downstream from DHEA, very little testosterone is made from circulating DHEA. The testes make testosterone directly (from cholesterol), so do not give DHEA expecting significant increases in testosterone. Oral or sublingual DHEA is often used. The latter may absorb directly in the mouth and bypass gut/liver metabolism, which may result in less estrogen production.	**Low:** 5 - 10 mg **High:** >100 mg **Most Common:** 10 - 25 mg *Typically taken daily*
HCG or Clomiphene	Human chorionic gonadotropin (hCG) acts as an LH analog and stimulates the Leydig cells to produce testosterone. Clomiphene citrate, a selective estrogen receptor modulator (SERM) can also be used for secondary hypogonadism. By blocking negative feedback of estrogen receptors, it increases gonadotropin levels, indirectly increasing testosterone production. These two options are not advised for primary hypogonadism.	**HCG:** 100 - 250 ug (2000 - 5000 IU) *Taken 2 - 3 times/week* **Clomiphene:** 25 mg *Taken every other day*

Transdermal Testosterone Monitoring



frontiers in Reproductive Health

FRH: Hypothesis and Theory

Alternatives to serum testing for transdermal testosterone monitoring: a review of clinical data and correlation with clinical response

Mark S. Newman[1]*, Jaclyn Smeaton[1], George Gillson[2] and Azra Jaferi[1]

[1]Precision Analytical, Inc., McMinnville, OR, United States, [2]Functional Medicine Consulting, Calgary, AL, Canada

Background: Blood (serum) testing is the standard method for monitoring testosterone (T) replacement therapy (TRT). Nevertheless, alternative methods, such as saliva testing, are gaining popularity because of their practical advantages.

Objective: This review aims to offer evidence-based, clinically relevant information to enable healthcare providers to make rational decisions regarding management of TRT. Providers need to know which combinations of ROA (route of administration) and testing method best track and reflect dosing and clinical outcomes. To that end, we summarize the large body of evidence for serum T testing during transdermal (TD) TRT monitoring, as well as the smaller body of evidence for saliva T testing in the context of TRT. Also discussed is T testing via capillary dried bloodspot (DBS) and urine. We chose to focus on TD formulations (gels, creams) because they are well-studied and commonly prescribed.

Methods: We conducted a literature search using online databases (PubMed/MEDLINE, ScienceDirect, and Google Scholar) and also reviewed real-world evidence available from large commercial laboratory databases. The clinical interpretation of these findings are discussed with regard to which tests best reflect clinical reality.

Results: Studies consistently show that serum T values increase proportionally with TD TRT dosing and strongly correlate with clinical responses. Use of serum testing for TD TRT monitoring is supported by published clinical guidelines. Endogenous saliva T levels at baseline are usually consistent with corresponding serum measures of T (when using accurate saliva steroid assays). However, this consistency is no longer observed when exogenous TD T is used. Evidence showed that saliva T values are routinely supraphysiological with standard TD TRT doses. These elevations in saliva are not known to be consistent with any clinical parameters. Like saliva, DBS T also often shows supraphysiologic responses to TD TRT, without clinical significance. Urine T levels tend to parallel serum T responses to TD TRT but may not be as reliable as serum, especially in people with *UGT2B17* deletion.

Conclusions: Based on the evidence, we conclude that: **(1)** serum T testing remains the most accurate, validated method for monitoring TD TRT; and **(2)** saliva and DBS T testing lack sufficient clinical correlation and should not be used for TD TRT monitoring. In particular, saliva T testing with TD TRT can yield misleading, erroneously high results, which can open the door to underdosing, loss of therapeutic benefit, and safety concerns.

Takeaways:

1. Serum is still best.
2. Saliva levels lose consistency with serum even at low doses.
3. Dried blood spot lacks adequate study.
4. Dried urine is a worthy companion to serum – especially as a way to assess tissue androgen response.

Newman MS. Frontiers, 2026.

Measuring Androgens: Serum and Dried Urine

Including dried urine testing in your work-up can **uncover issues with tissue androgen activity and metabolism**

Hormone and ROA	Serum	Urine
Total T	✓	✗
Free/Bioavailable T	✓ (Vermeulen equation)	✓ (Serum is gold standard*)
T Metabolism	✗ (Limited)	✓
DHEA	✓ (DHEA-S)	✓ (“Total DHEA”)
DHEA Metabolism	✗	✓

*Serum is gold standard due to the possibility of falsely low urinary T when UGT deletion is present
**“Total DHEA” = DHEA-S + Etiocholanolone + Androsterone (4-Spot Urine Test)

Treat ALL Imbalances That Contribute to Low Androgen Symptoms

- Estrogen/Progesterone Imbalance
- High and Low Cortisol
- Hypothyroid
- Gut Dysbiosis
- Metabolic Syndrome
- Chronic Inflammation
- Neurotransmitter imbalances

References

- Babcock MC, et al. Oxidative stress and inflammation are associated with age-related endothelial dysfunction in men with low testosterone. J Clin Endocrinol Metab. 2022.
- Bachmann GA. The hypoandrogenic woman: pathophysiologic overview. Fertility and Sterility. 2002.
- Brzozowska M, Lewiński A. Changes of androgens levels in menopausal women. Prz Menopauzalny. 2020.
- Chauhan S, et al. Effect of standardized root extract of ashwagandha (Withania somnifera) on well-being and sexual performance in adult males: A randomized controlled trial. Health Sci Rep. 2022.
- Cheng H, et al. Age-related testosterone decline: mechanisms and intervention strategies. Reprod Biol Endocrinol. 2024.
- Mok SF, et al. Testosterone for androgen deficiency-like symptoms in men without pathologic hypogonadism: a randomized, placebo-controlled cross-over with masked choice extension clinical trial. J Gerontol A Biol Sci Med Sci. 2020.
- Dutheil F, et al. DHEA as a Biomarker of Stress: A Systematic Review and Meta-Analysis. Front Psychiatry. 2021.
- Fogle RH, et al. Ovarian androgen production in postmenopausal women. J Clin Endocrinol Met. 2007.
- Kumari M, et al. Association of diurnal patterns in salivary cortisol with all-cause and cardiovascular mortality: findings from the Whitehall II study. J Clin Endocrinol Metab. 2011.
- Labrie F, et al. DHEA and its transformation into androgens and estrogens in peripheral target tissues: intracrinology. Front Neuroendocrinol. 2001.
- Laughlin GA, et al. Hysterectomy, oophorectomy, and endogenous sex hormone levels in older women: the Rancho Bernardo Study. J Clin Endocrinol Metab. 2000.

References

- Morgentaler A, et al. Androgen Society Position Paper on Cardiovascular Risk With Testosterone Therapy. Mayo Clinic Proceedings, 2024.
- Newman MS, et al. Comparative estrogen exposure from compounded transdermal estradiol creams and Food and Drug Administration-approved transdermal estradiol gels patches. Menopause. 2023.
- Newman MS, et al. Alternatives to serum testing for transdermal testosterone monitoring: a review of clinical data and correlation with clinical response. Frontiers. 2026.
- Papierska L. Adrenopause – does it really exist? Menopausal Review. 2017.
- Samaras N, et al. A review of age-related dehydroepiandrosterone decline and its association with well-known geriatric syndromes: is treatment beneficial? Rejuvenation Res. 2013.
- Schiffer L, et al. Intracrine androgen biosynthesis, metabolism and action revisited. Mol Cell Endocrinol. 2018;465:4-26.
- Sharma S, Mahajan N. Polycystic Ovarian Syndrome and Menopause in Forty Plus Women. J Midlife Health. 2021.
- Shin JH, et al. Serum level of sex steroid hormone is associated with diversity and profiles of human gut microbiome. Res Microbiol. 2019.
- Uloko, M, et al. The clinical management of testosterone replacement therapy in postmenopausal women with hypoactive sexual desire disorder: a review. Int J Impot Res. 2022.
- Vogelzangs N, et al. Urinary cortisol and six-year risk of all-cause and cardiovascular mortality. J Clin Endocrinol Metab. 2010.
- Wu, T. T., et al. Prognostic Value of Dehydroepiandrosterone Sulfate for Patients With Cardiovascular Disease: A Systematic Review and Meta-Analysis. J Am Heart Assoc. 2017.

Thank You!

info@dutchtest.com

dutchwebinars