

The Vibrant Longevity Summit

Hormone Optimization

Elevating Lifespan
Through Endocrine
Balance



Session 1
**Dr. Craig
Koniver, MD**



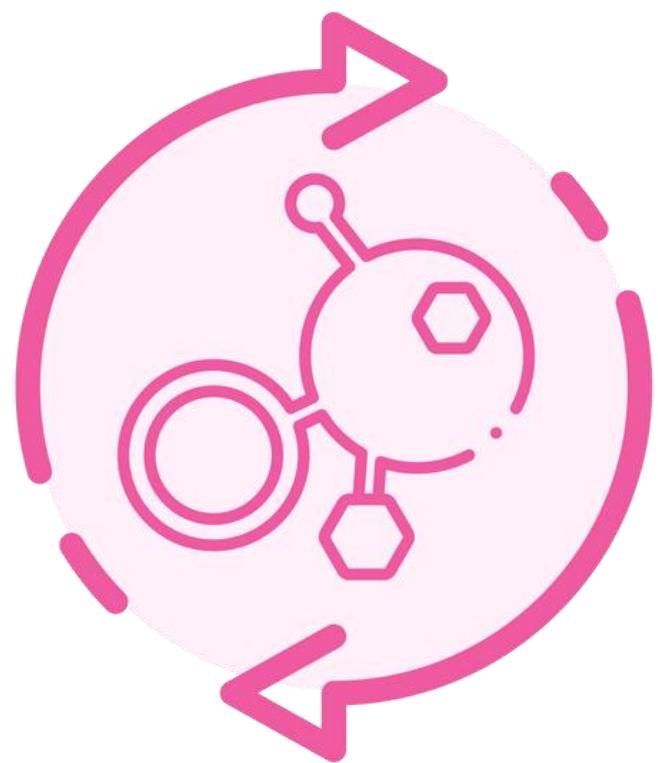
Session 2
**Dr. Tara Scott,
MD, FACOG,
ABAARM,
ABOIM, CNMP**



Session 3
**Dr. Carrie
Jones, ND,
FABNE, MPH,
MSCP**



Session 4
**Dr. Angela D
Mazza, DO,
ABAARM,
FAAMFM, ECNU**



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Session 1

**Craig Koniver,
MD**

Meet Your **Speaker**

Craig Koniver, MD

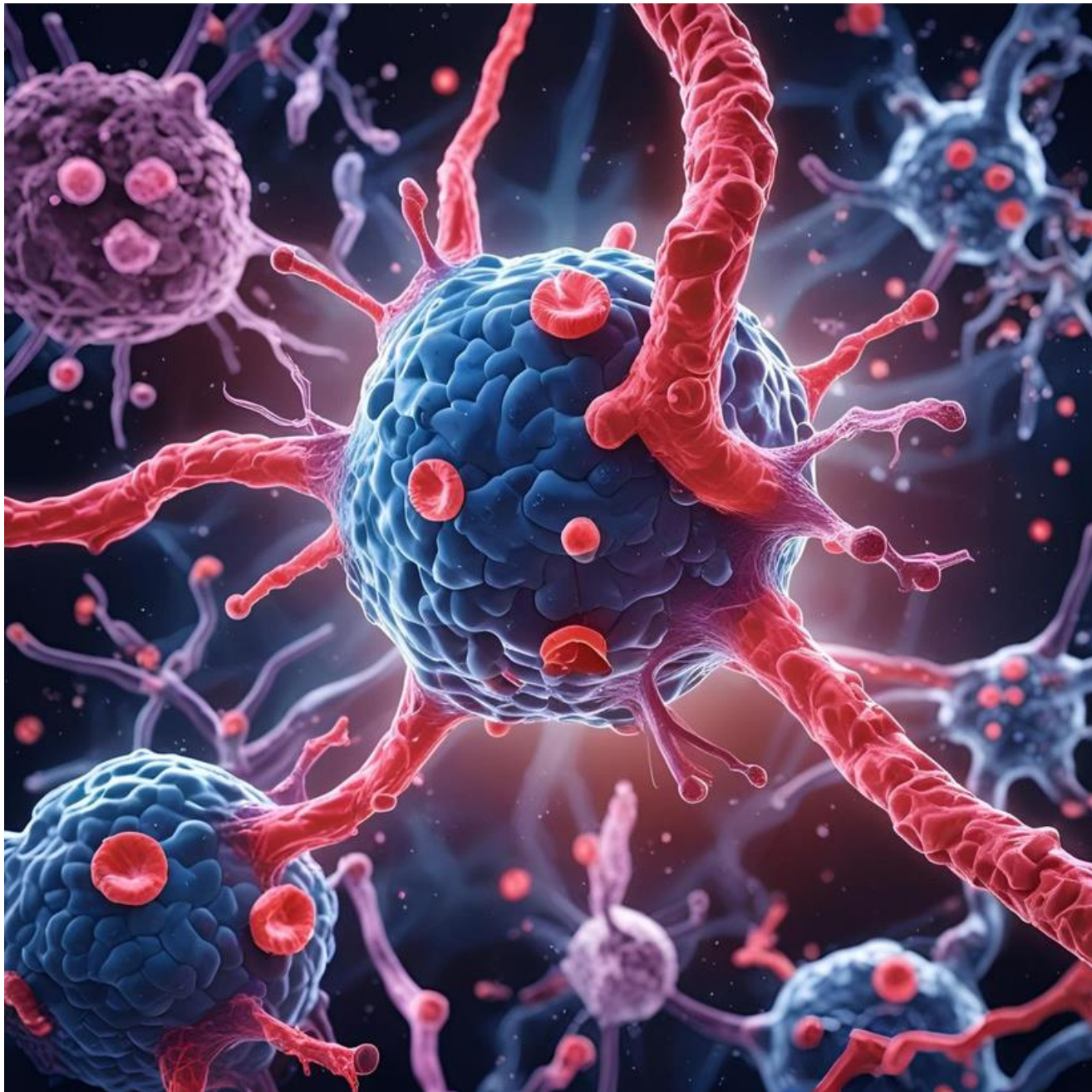
Craig Koniver, MD, stands at the forefront of performance and integrative medicine, merging pioneering science with personalized treatment to optimize physical and cognitive function. His sought-after protocols—including peptide therapy, NAD+ optimization, and bioidentical hormones—have drawn a global clientele ranging from elite athletes and supermodels to Fortune 500 CEOs.



Hormones' Influence on Core Pillars of Longevity

Craig Koniver, MD

Introduction to Hormones and Longevity



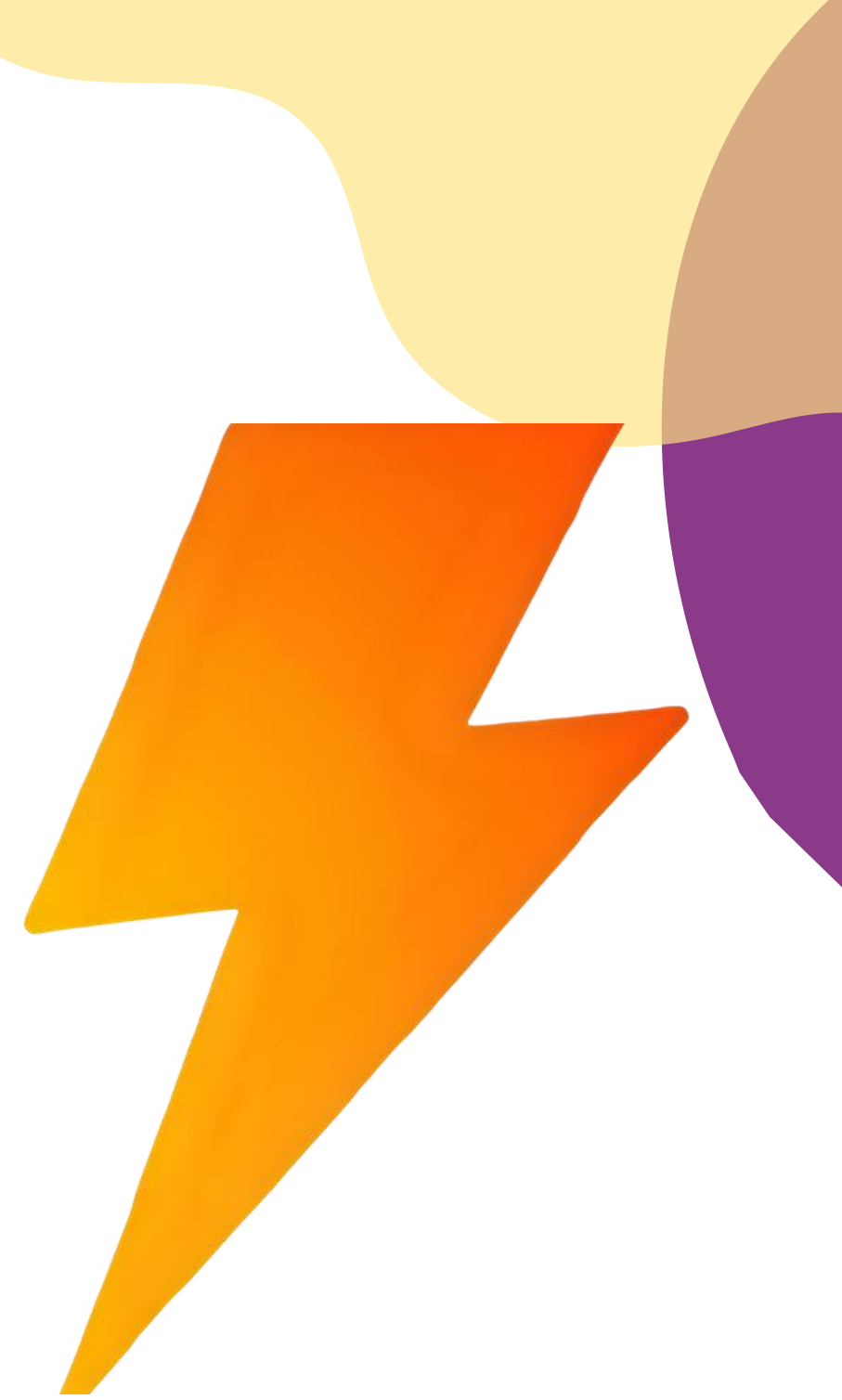
Aging is associated with hormonal declines (e.g., thyroid, sex hormones, growth hormone) that impact metabolic processes and overall health.

- These changes can accelerate age-related declines, but optimizing hormonal balance may promote longevity by preserving metabolic fitness and reducing oxidative stress.
- Key hormones involved: Thyroid (T3/T4), sex hormones (estrogen, testosterone), cortisol, growth hormone (GH), insulin, and melatonin.
- Longevity pillars (energy, cognition, metabolic rate, muscle maintenance, sleep) are interconnected; hormonal dysregulation in one area affects others, influencing lifespan and healthspan.

Hormones and Energy

Thyroid hormones (TH) regulate energy balance by controlling storage and expenditure, with low TH linked to reduced energy levels and metabolic slowdown.

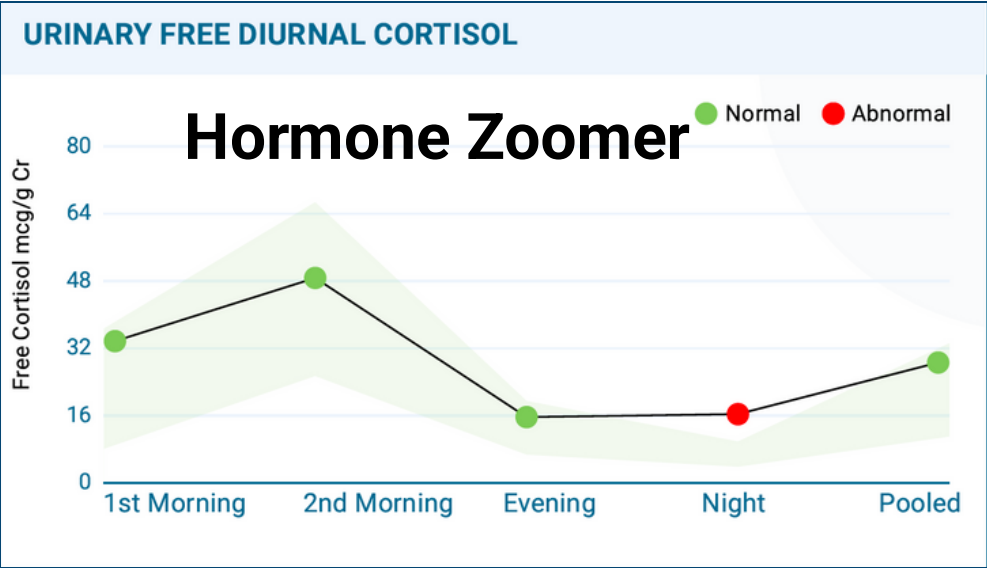
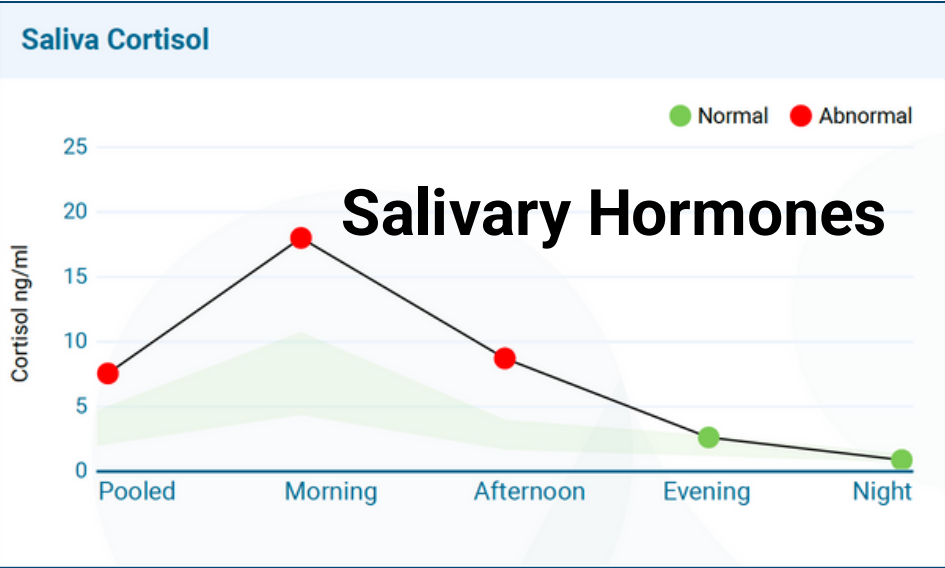
- Cortisol and adrenaline provide acute energy mobilization, but chronic elevation depletes reserves, contributing to fatigue in aging.
- GH and insulin-like growth factor (IGF-1) support energy restoration during recovery, with declines impairing vitality.
- Clinical implication: Hypothyroidism or adrenal insufficiency in elderly patients may manifest as low energy; monitoring TSH and optimizing TH can enhance longevity.



Hormones and Energy:

Vibrant Testing & Peptides

- Markers: T3, T4, TSH (Healthspan Assessment Panel); Cortisol curve (Salivary Hormones and/or Hormone Zoomer); DHEA-S, Melatonin (Hormone Zoomer)
- Peptide Protocols: SS-31, CB4211, NAD+ T3 rate limiting hormone for mitochondrial ETC Fxn.
- Clinical: Identify fatigue drivers → peptide optimization



Thyroid				
Test Name	Current	Previous	Result	Reference
T3 - Triiodothyronine (ng/mL)	0.9			0.8-2.0
T4 - Thyroxine (µg/dL)	6.3			4.5-9.8
Free T3 (pg/mL)	2.5			2.0-4.4
Free T4 (ng/dL)	1.3			0.9-1.7
TSH (µIU/mL)	1.660			0.111-4.91
Anti-TPO (IU/mL)	<12			≤34.0
Reverse T3* (ng/dL)	19			7.0-23.0
Anti-TG (IU/mL)	15.6			≤115.0

Hormones and Cognition

- Sex hormones (estrogen, testosterone) protect cognitive function; declines in aging correlate with memory loss and increased dementia risk.
- Estrogen exerts antioxidant effects, supporting neuroplasticity; hormone replacement therapy (HRT) may preserve cognition in postmenopausal women.
- Thyroid hormones influence brain metabolism; hypothyroidism accelerates cognitive decline.
- Clinical implication: Assess sex hormone levels in patients with mild cognitive impairment; balanced hormones may delay age-related neurodegeneration.



Hormones and Cognition:

Vibrant Testing & Peptides

- Markers: Estradiol, Progesterone, Testosterone (Healthspan Assessment, Hormone Zoomer); 8-OH-dG (Hormone Zoomer)
- Peptide Protocols: Selank, PE 22-28, Methylene Blue, Cerebroylsin

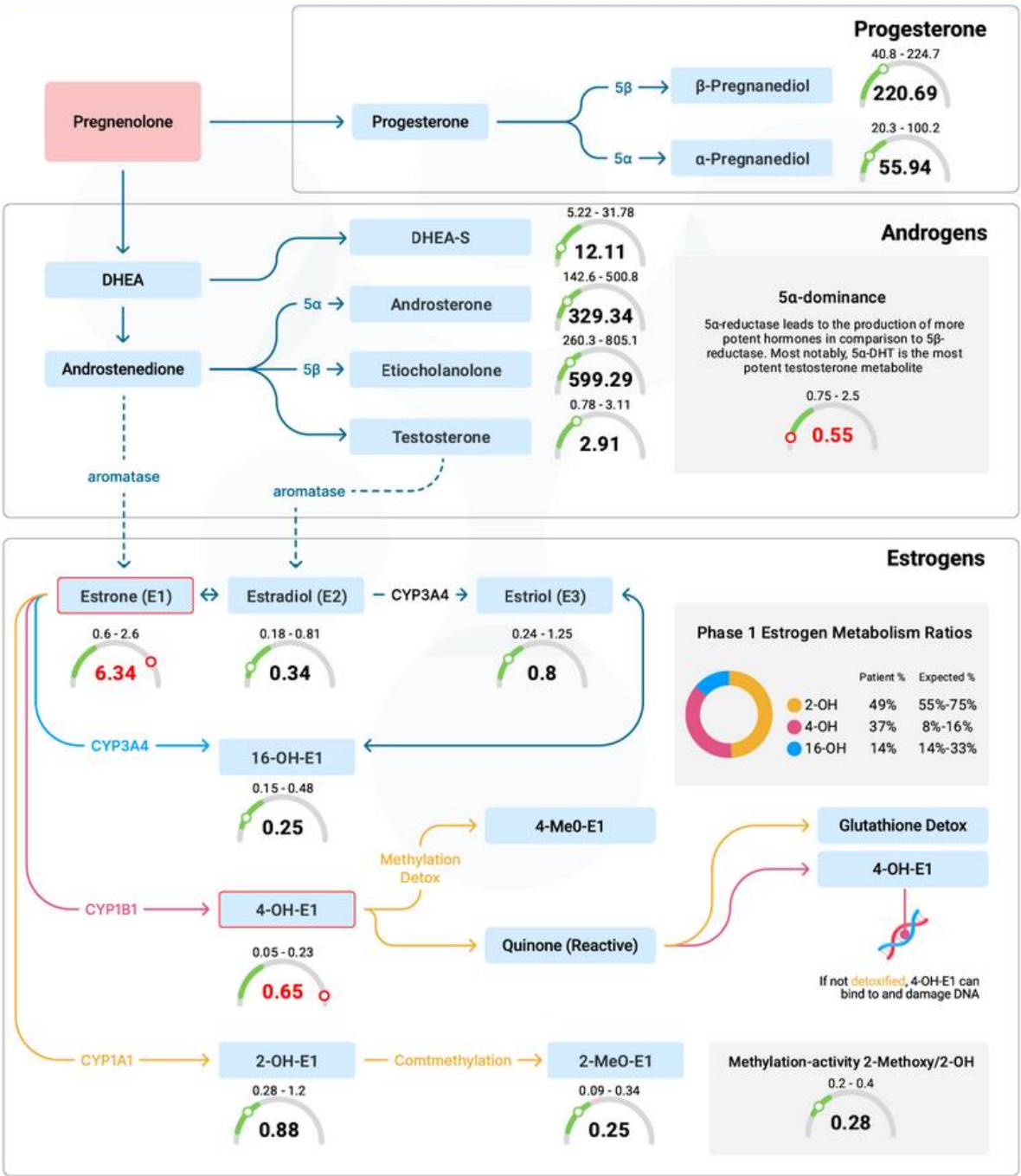
Hormones (all)				
Test Name	Current	Previous	Result	Reference
Estradiol (pg/mL)	314.0			
FSH (mIU/mL)	3.6			
DHEA-S (µg/dL)	500.0			60.9-337.0
LH (mIU/mL)	2.7			
SHBG (nmol/L)	62.6			24.6-122.0
Cortisol (µg/dL)	9.7			6.2-19.4
Testosterone, Total (ng/dL)	45.8			4.5-269.2
Free Testosterone (ng/dL)	0.53			0.03-2.56
Progesterone (ng/mL)	0.285			
Parathyroid Hormone (pg/mL)	49			15.0-65.0
Prolactin (ng/mL)	15.30			4.79-23.3
Dihydrotestosterone* (ng/dl)	20.7			6.5-50.1
Pregnenolone* (ng/mL)	3.69			0.31-3.8

Healthspan Assessment

Hormone Zoomer

Oxidative Stress				
Test Name	Current	Previous	Result	Reference
8-hydroxy-2'-deoxyguanosine (8-OHdG) (mcg/g)	4.44			≤4.77

Sex Hormone - Urine



Hormones and Metabolic Rate

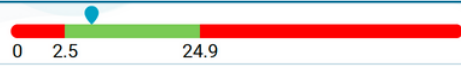
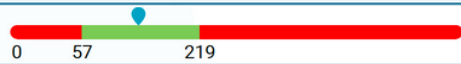
- Thyroid hormones are primary regulators of basal metabolic rate (BMR), with T3/T4 increasing energy expenditure; age-related TH decline slows metabolism.
- Insulin and cortisol modulate glucose metabolism; dysregulation leads to insulin resistance and reduced metabolic efficiency.
- Lower TH levels may promote longevity by reducing oxidative stress and metabolic rate, as seen in centenarians.
- Clinical implication: In obese or diabetic patients, thyroid function tests can guide interventions to optimize metabolic health and extend healthspan.

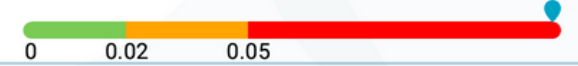
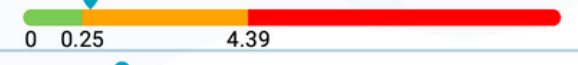





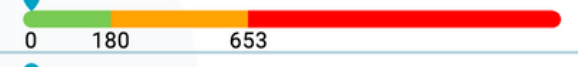



Hormones and Metabolic Rate: Vibrant Testing & Peptides

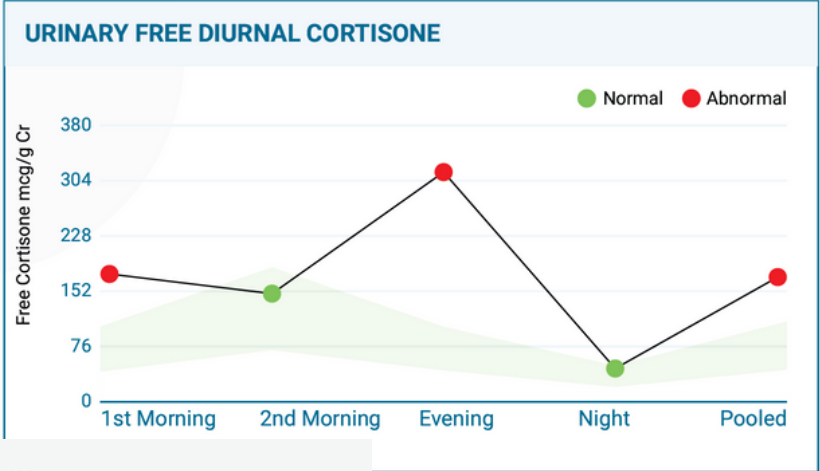
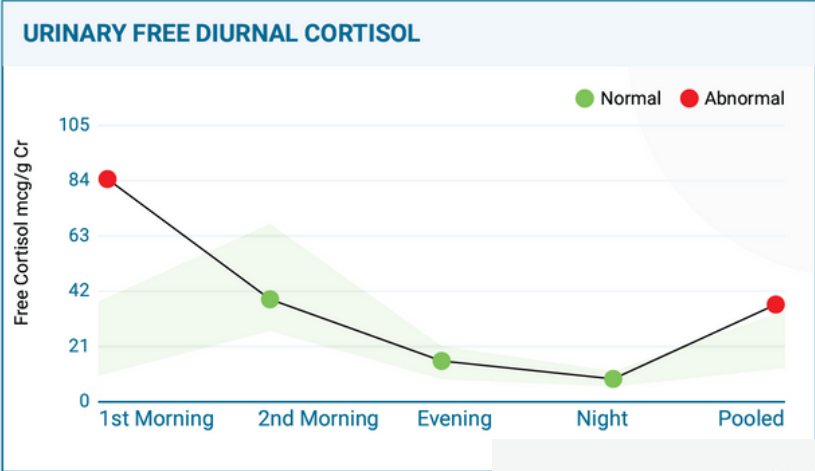
Hormone Zoomer

- Markers: Insulin, IGF-1, Cortisol (healthspan assesment); Endocrine disruptors (hormone zoomer)
- Peptide Protocols: GLP-1s, Tesamorelin, CB4211
- Clinical: Address bottlenecks → optimize metabolism

Healthspan Assesment

Beta Cell Function				
Test Name	Current	Previous	Result	Reference
Insulin (µU/mL)	4.2			2.6-24.9
Other Markers				
Test Name	Current	Previous	Result	Reference
Human IGF-I (ng/mL)	134			58.0-219.0

Endocrine Disruptors	Current	Previous	Result	Reference
Atrazine ^ (ug/g)	0.98			≤0.05
Butylparaben^ (ug/g)	0.45			≤4.39
Perchlorate (PERC)^ (ug/g)	4.30			≤10.7
Glyphosate (ug/g)	0.56			≤7.6
Mono-ethyl phthalate (MEtP)^ (ug/g)	0.94			≤541
Mono-2-ethylhexyl phthalate (MEHP)^ (ug/g)	0.92			≤8.47
Mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)^ (ug/g)	2.89			≤37.7
Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)^ (ug/g)	0.08			≤23.4
Methylparaben^ (ug/g)	0.08			≤653
Propylparaben^ (ug/g)	0.03			≤222
Ethylparaben ^ (ug/g)	0.04			≤99.3



Cortisol/Cortisone

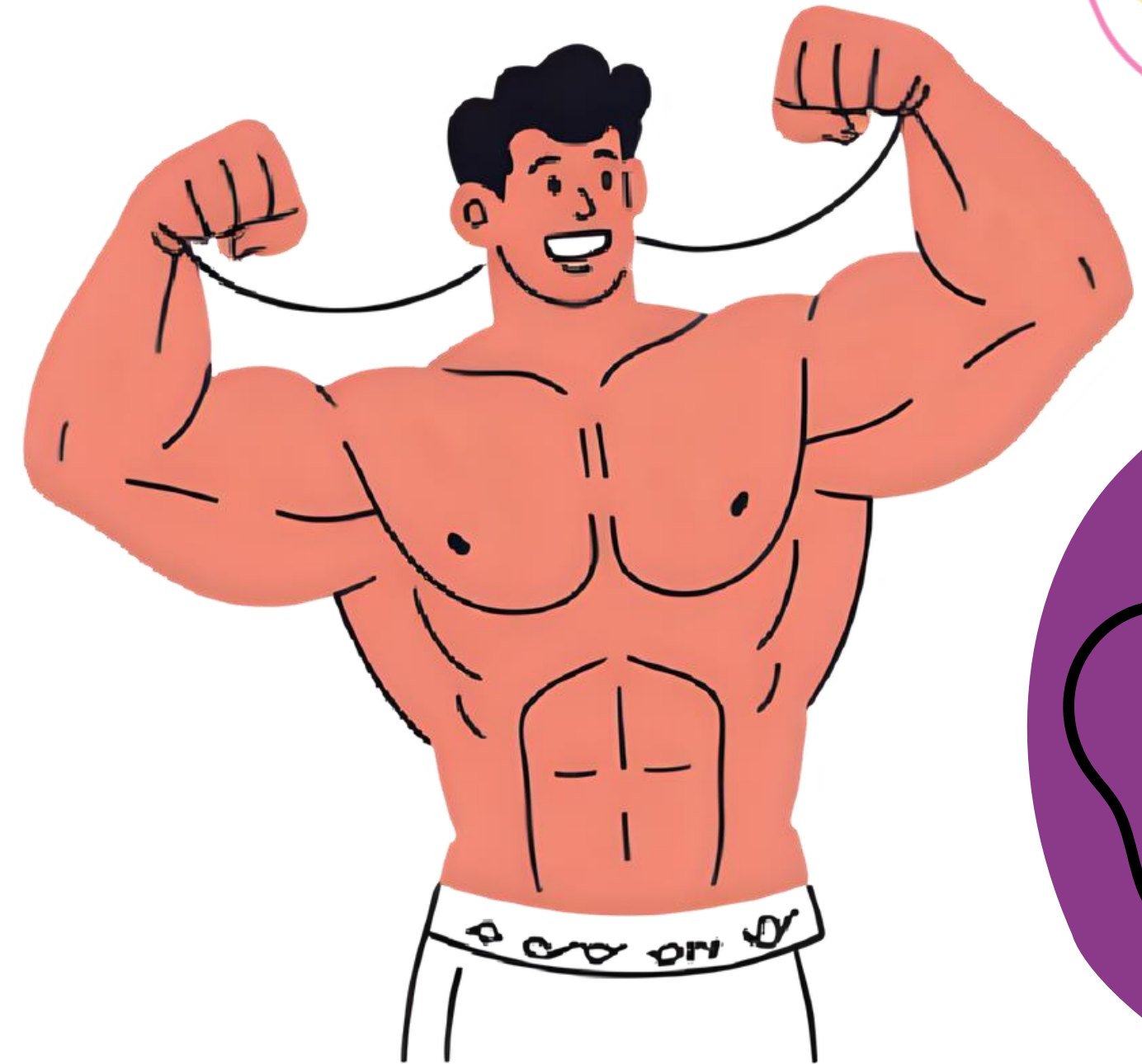
11β-HSD index measures the activity level of the 11β-HSD enzyme which maintains balance between active cortisol and inactive cortisone. Increased cortisol levels has been associated with conditions like metabolic syndrome and insulin resistance.

0.48 - 0.59

0.81

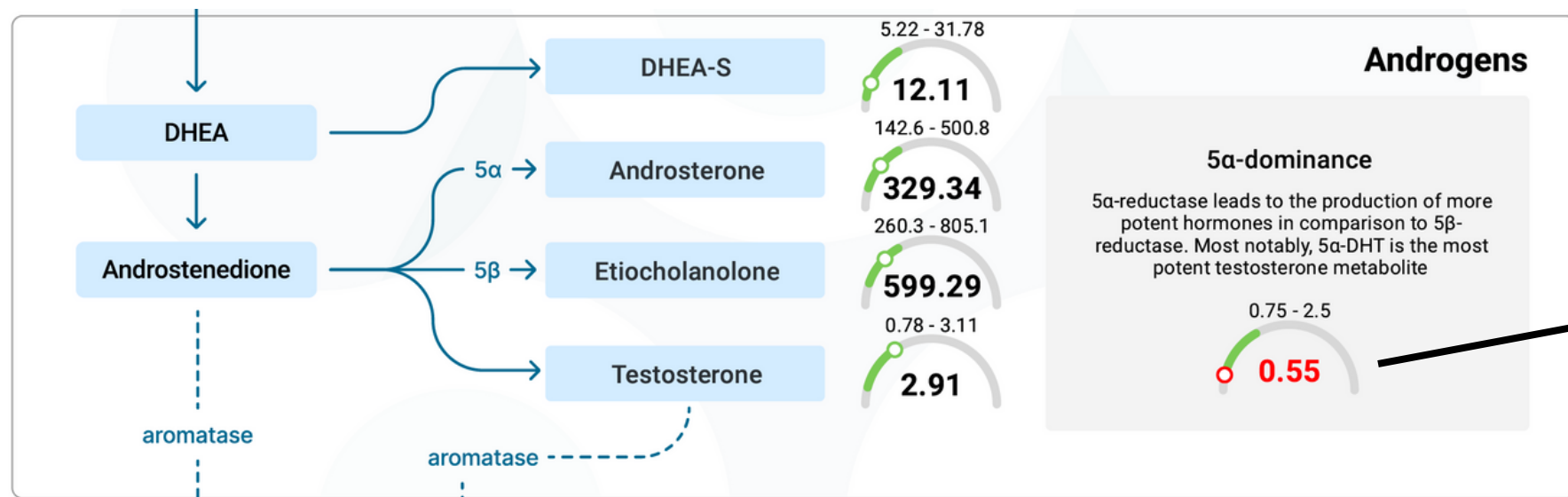
Hormones and Muscle Maintenance

- Testosterone and GH promote muscle protein synthesis; andropause/menopause-related declines accelerate sarcopenia.
- Estrogen supports muscle health via antioxidant and metabolic effects; HRT may mitigate muscle loss in women.
- Cortisol excess catabolizes muscle; chronic stress exacerbates age-related atrophy.
 - Cortisol deficiency (way more common) also leads to impaired catabolic processes
- Clinical implication: Hormone panels (e.g., free testosterone, IGF-1) in frail elderly can inform therapies like resistance training or HRT to preserve muscle mass.



Hormones and Muscle Maintenance: Vibrant Testing & Peptides

- Markers: Total & Free Testosterone, DHT, IGF-1 (healthspan assesment); androgen metabolites (hormone zoomer)
- Peptide Protocols: CJC-1295/ Ipamorelin, PDA, Tesamorelin, Semorelin
- Clinical: Preserve lean mass, support repair



Testosterone				
Test Name	Current	Previous	Result	Reference
Testosterone (T) (mcg/g)	2.91			0.78-3.11
Epi-Testosterone (Epi-T) (mcg/g)	0.54			0.35-1.25
Androstenedione (mcg/g)	4.11			2.58-7.44
Androsterone (mcg/g)	329.34			142.6-500.8
Etiocholanolone (mcg/g)	599.29			260.3-805.1
5a-DHT (mcg/g)	0.55			0.34-1.05
5a,3a-Androstanediol (mcg/g)	16.65			2.46-8.59
5b-Androstanediol (mcg/g)	7.20			4.15-15.66

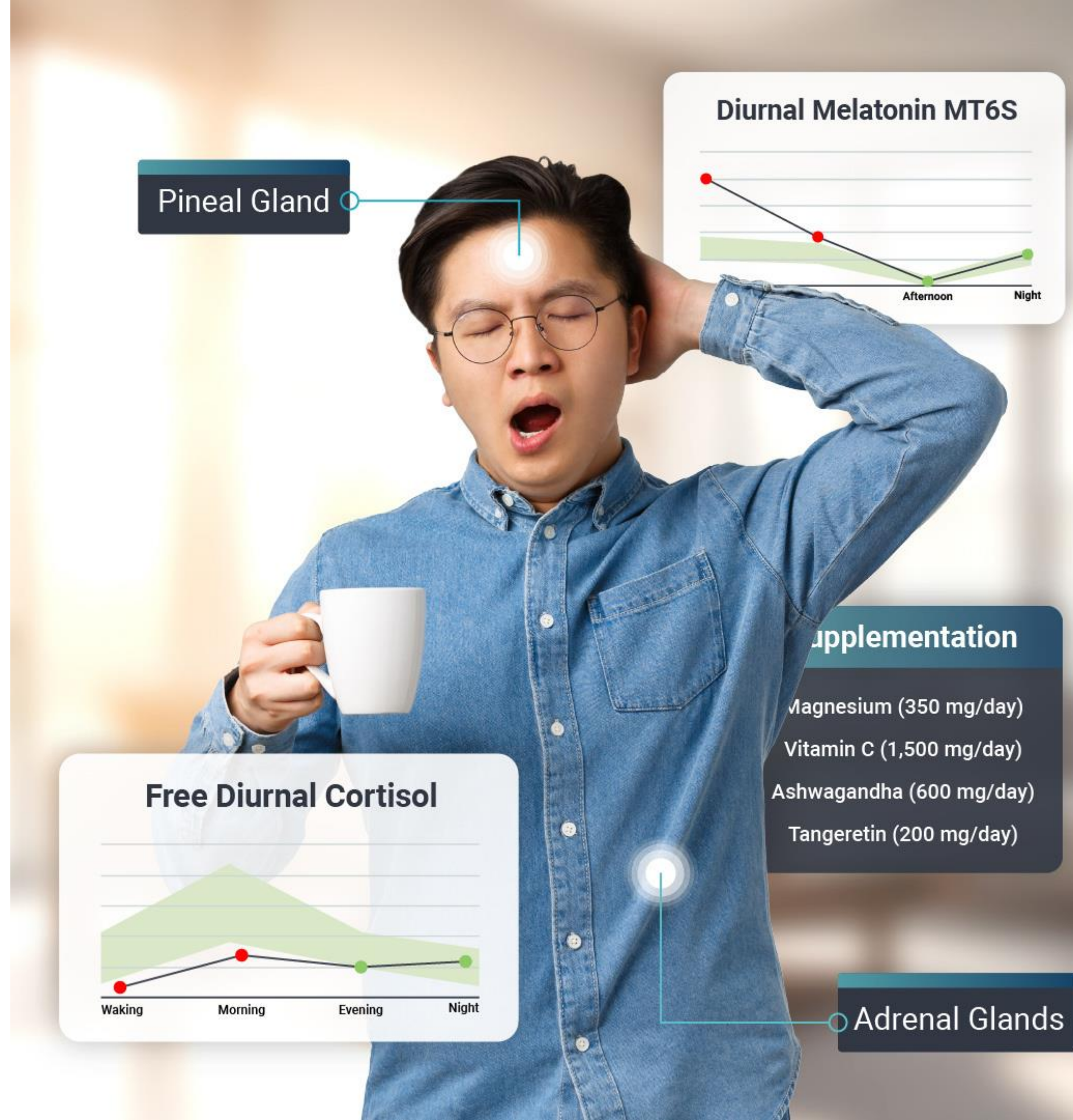
Hormones and Sleep



- Melatonin and cortisol regulate circadian rhythms; age-related melatonin decline disrupts sleep, elevating cortisol and impairing recovery.
- GH is secreted during deep sleep, supporting repair; poor sleep reduces GH, affecting metabolism and longevity.
- Sex hormones influence sleep architecture; estrogen promotes REM sleep, while testosterone aids deep sleep.
- Clinical implication: In insomniac patients, evaluate cortisol and melatonin; interventions like melatonin supplementation may enhance sleep quality and hormonal balance.

Hormones and Sleep: Vibrant Testing & Peptides

- Markers: Cortisol rhythm (salivary hormones); Melatonin (hormone zoomer)
- Peptide Protocols: Pinealon, TA1
- Clinical: Testing directs circadian peptide use



Pillar	Hormonal Link	Impact on Longevity
Energy	Thyroid, cortisol, GH	Sustained energy prevents fatigue-related declines; optimized levels extend vitality.
Cognition	Sex hormones, thyroid	Preserved cognition reduces dementia risk, supporting independent aging.
Metabolic Rate	Thyroid, insulin	Balanced metabolism lowers chronic disease risk, promoting lifespan.
Muscle Maintenance	Testosterone, GH, estrogen	Muscle preservation combats frailty, enhancing mobility and survival.
Sleep	Melatonin, cortisol, GH	Quality sleep restores hormones, reducing inflammation and supporting healthspan.



Lab Testing

- **Serum:** thyroid (all markers), sex hormones, adrenal hormones, pituitary hormones, IGF-1, Insulin
- **Saliva:** cortisol/ cortisol metabolites
- **Urine:** estrogen metabolites, melatonin, toxicity markers, bone metabolites, cortisol metabolites

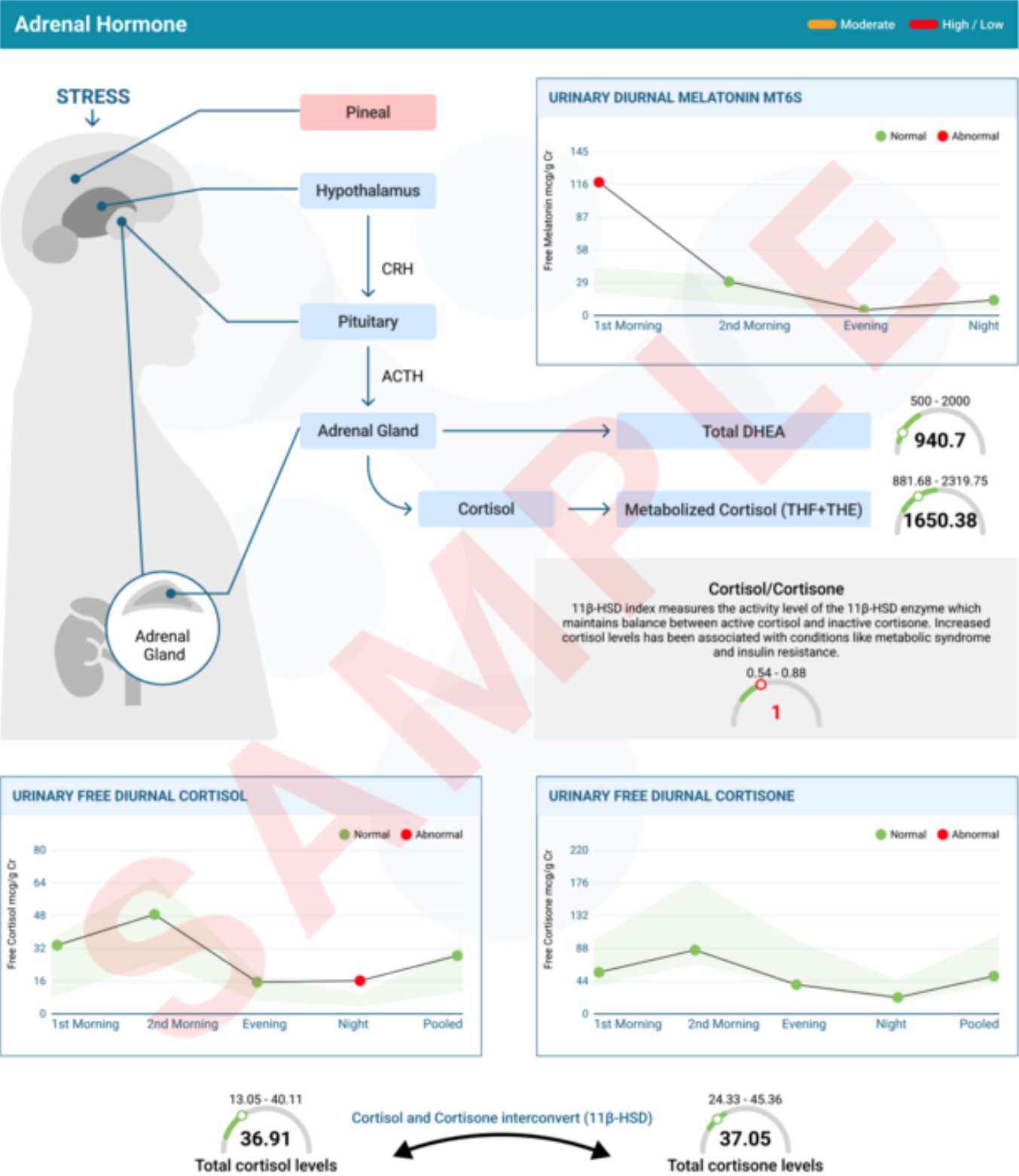
Vibrant Testing Solutions & Peptides

recap of specific test and peptide therapies

- Energy → Vibrant Blood Hormones In healthspan assesment and Salivary Hormones (Thyroid, Cortisol) → SS-31, CB4211, NAD+
- Cognition → Hormone Zoomer (Hormone balance, 8-OH-dG) → Selank, PE 22-28, MB. Cerebrolysin
- Metabolic → Vibrant Blood Hormones In healthspan assesment and Hormone Zoomer (Insulin, IGF-1, Endocrine Disruptors) → GLP-1, CB4211, Tesamorelin
- Muscle → Vibrant Blood Hormones In healthspan assesment and Hormone Zoomer (Testosterone, IGF-1) → CJC1295, GHRPs
- Sleep → Salivary Hormones (Cortisol, Melatonin) → Pinealon, TAI

Vibrant Hormone Zoomer

Urinary Hormone Markers		
Adrenal Hormones	Testosterone	Ratios
<ul style="list-style-type: none">DeoxycorticosteroneCorticosteroneDHEADHEA-STotal CortisolTotal Cortisoneβ-Tetrahydrocortisol (β-THF)α-Tetrahydrocortisol (α-THF)β-Tetrahydrocortisone (β-THE)	<ul style="list-style-type: none">AndrostenedioneAndrosteroneEtiocholanoloneTestosterone (T)Epi-Testosterone (Epi-T)5α-DHT5α,3α-Androstanediol5β-Androstanediol	<ul style="list-style-type: none">2-OH E1/4-OH E1E3/(E1+E2)2-OH (E1 + E2)/16α-OH E12-MeO E1/2-OH E14-MeO E1/4-OH E14-MeO E2/4-OH E2β-Pregnanediol/E2T/Epi-TCortisol/CortisoneMetabolized Cortisol (THF+THE)
Diurnal Cortisol	Diurnal Cortisone	Endocrine Disruptor Markers
<ul style="list-style-type: none">Free Cortisol (1st Morning)Free Cortisol (2nd Morning)Free Cortisol (Evening)Free Cortisol (Night)Free Cortisol (Pooled)	<ul style="list-style-type: none">Free Cortisone (1st Morning)Free Cortisone (2nd Morning)Free Cortisone (Evening)Free Cortisone (Night)Free Cortisone (Pooled)	
Diurnal Melatonin	Creatinine	
<ul style="list-style-type: none">Melatonin (1st Morning)Melatonin (2nd Morning)Melatonin (Evening)Melatonin (Night)Melatonin (Pooled)	<ul style="list-style-type: none">Creatinine (1st Morning)Creatinine (2nd Morning)Creatinine (Evening)Creatinine (Night)Creatinine (Pooled)	<ul style="list-style-type: none">Bisphenol A (BPA)AtrazineGlyphosateMonoethyl PhthalateMono-2-ethylhexyl PhthalateMono-(2-ethyl-5-hydroxyhexyl) PhthalateMono-(2-ethyl-5-oxohexyl) PhthalateMethylparabenEthylparabenButylparabenPropylparabenTriclosanPerchlorate
Estrogen	Progesterone	Bone Health Markers
<ul style="list-style-type: none">Estradiol (E2)Estrone (E1)Estriol (E3)2-OH Estradiol2-OH Estrone4-OH Estradiol4-OH Estrone16α-OH Estrone2-MeO Estradiol2-MeO Estrone4-MeO Estradiol4-MeO EstroneTotal Estrogen	<ul style="list-style-type: none">β-Pregnanediolα-PregnanediolAllopregnanolone3α-Dihydroprogesterone20α-Dihydroprogesterone	<ul style="list-style-type: none">Deoxypyridinoline (DPD) CrosslinksPyridinoline (PYD) Crosslinks
		Oxidative Stress Marker
		<ul style="list-style-type: none">8-Hydroxy-2-deoxyguanosine (8OHdG)



Vibrant Salivary Hormones

Salivary Hormones Markers List

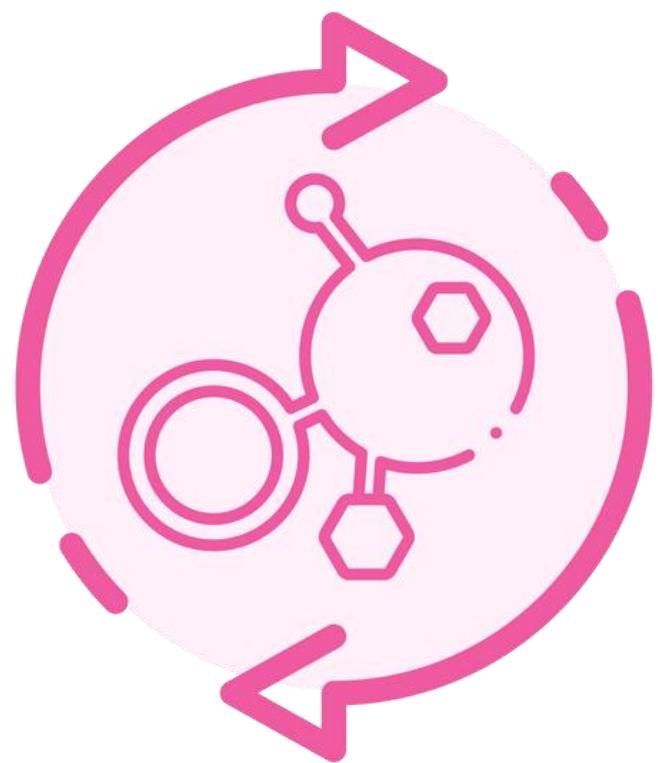
SALIVA STEROIDS HORMONES	SALIVA CORTISOL	SALIVA CORTISONE
Estradiol (E2) Estrone (E1) Estriol (E3) E3/(E1+E2) Ratio Progesterone (Pg) Pg/E2 Ratio Testosterone DHEA-S	Cortisol (Pooled) Cortisol (Morning) Cortisol (Noon) Cortisol (Evening) Cortisol (Night)	Cortisone (Pooled) Cortisone (Morning) Cortisone (Noon) Cortisone (Evening) Cortisone (Night)



Thank You!

Craig Koniver, MD

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Hormone Optimization

Elevating Lifespan
Through Endocrine
Balance



Session 2

**Dr. Tara Scott,
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Meet Your **Speaker**

**Dr. Tara Scott, MD,
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HORMONE
 GURU





Hormone Optimization: Elevating Lifespan and Peak Clinical Performance

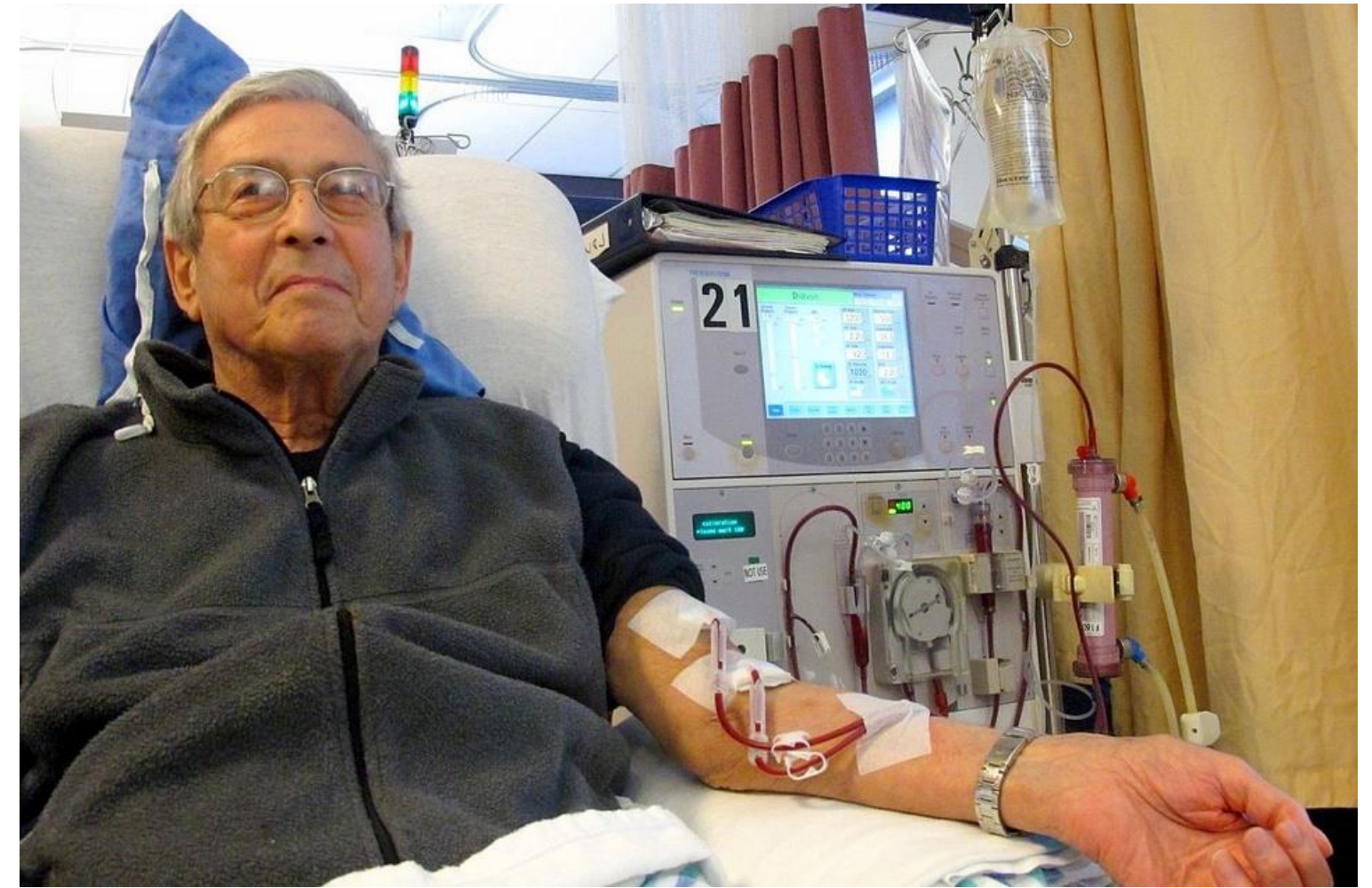
The Test

Suppose I told all of my patients to drink 2 liters of water daily

Marathon Runner in 80 degrees



Dialysis Patient

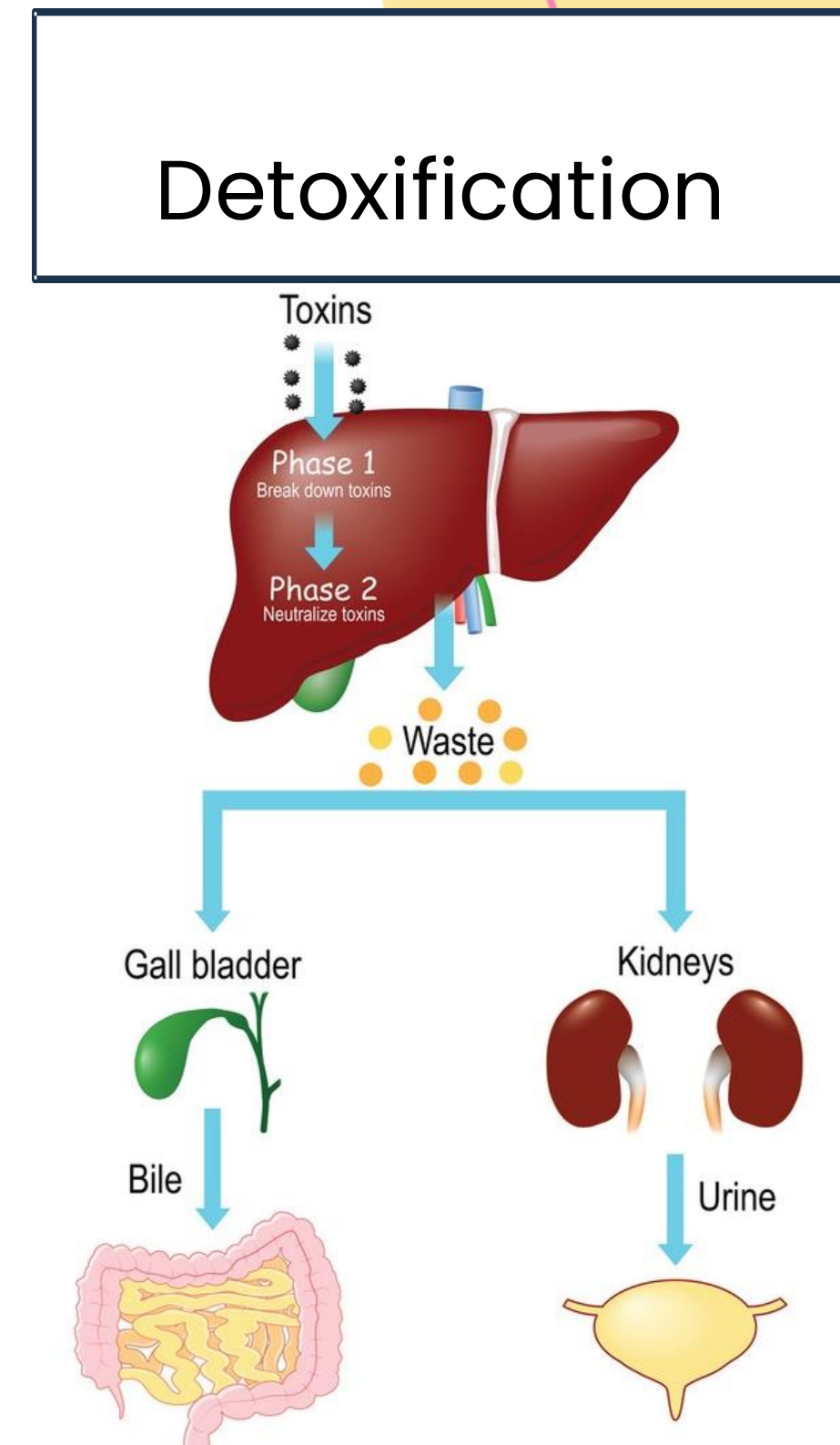


Why is it important to Check Estrogen metabolism?

- Is it really possible to have a randomized placebo controlled trial with hormone therapy?
- You need to consider:
 - Weight, age, oophorectomy status
 - Pharmacokinetics– what the body does to the drug
 - Pharmacodynamics– what the drug does to the body

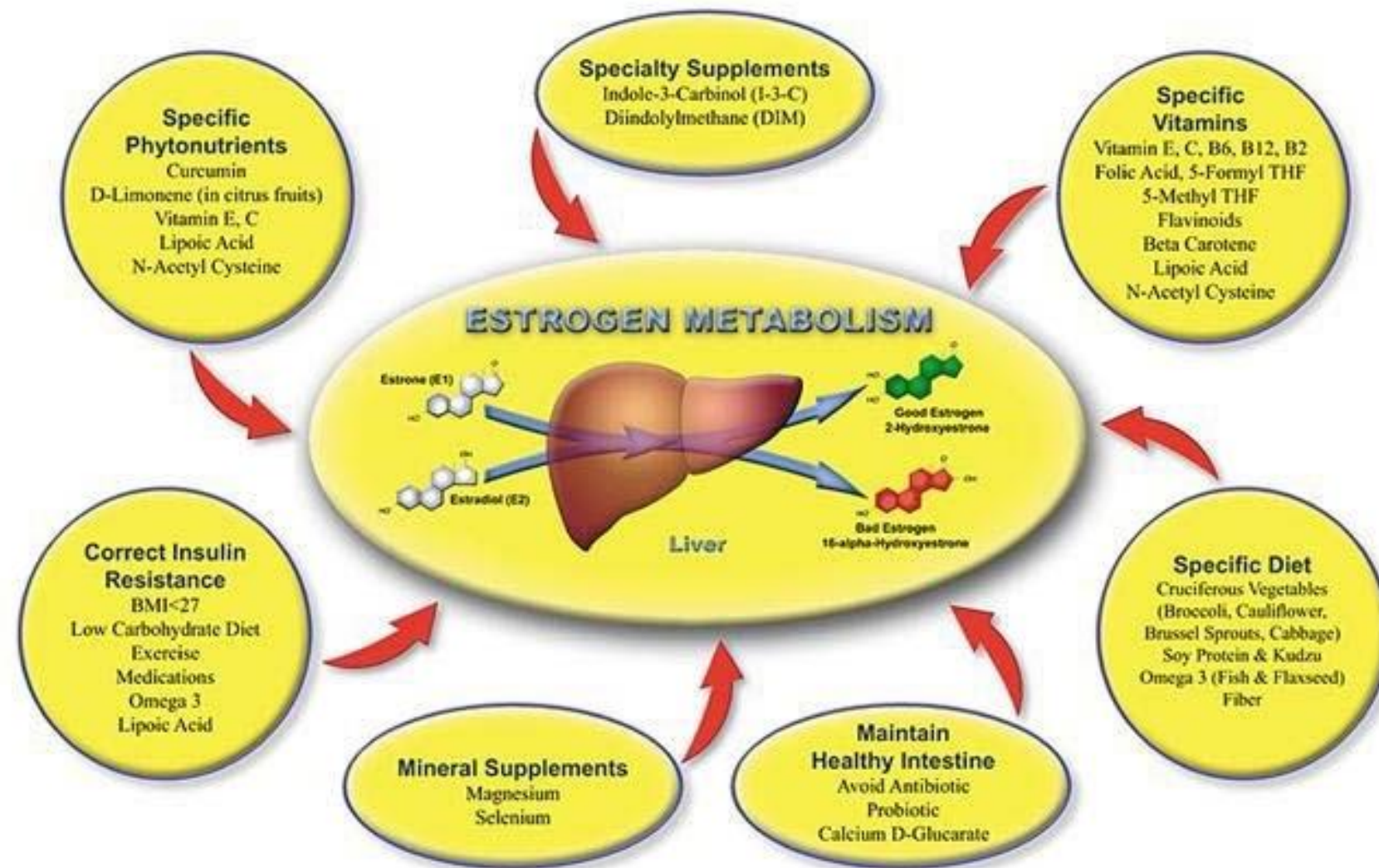
Causes of Detox Imbalances

- Overwhelming load
- Impaired Phase 1
- Impaired Phase 2
- Impaired Phase 3



Healthy Estrogen Metabolism

Promotion of Healthy Estrogen Metabolism



Specific Phytonutrients

Specific Supplements

Specific Vitamins

Specific Diet

Healthy Intestine

Minerals

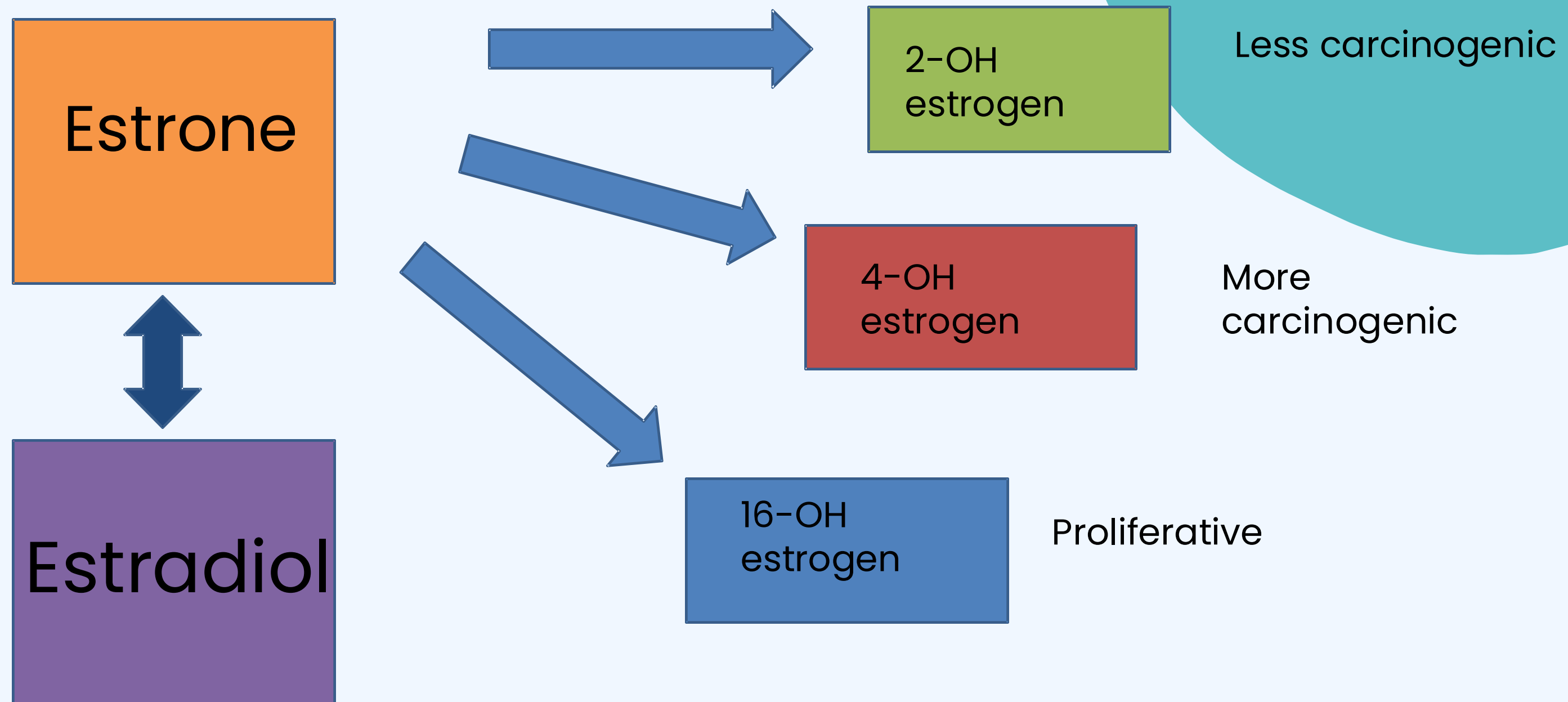
Correct Insulin Resistance

Phase 1 Detoxification

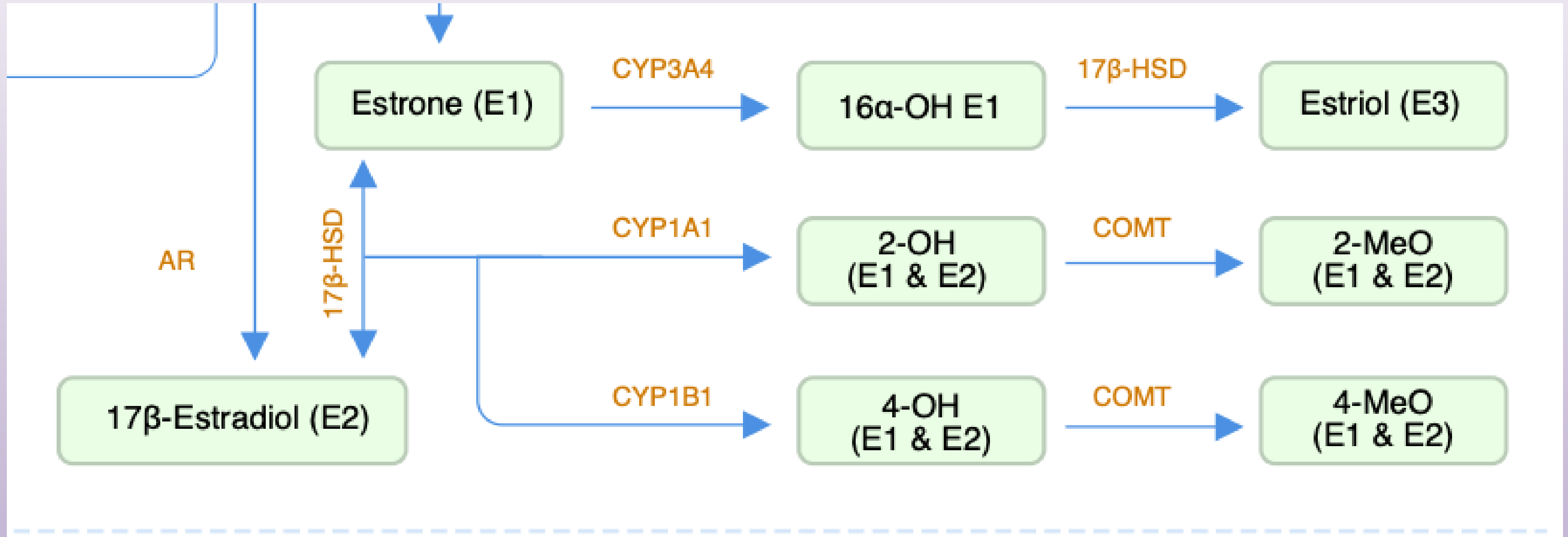


- **Cytochrome P 450**
- **Three Pathways**
 - **CYP 1A1**
 - **CYP 3A4**
 - **CYP 1B1**

Phase 1 Estrogen Detox



Estrogens & Estrogen Metabolites

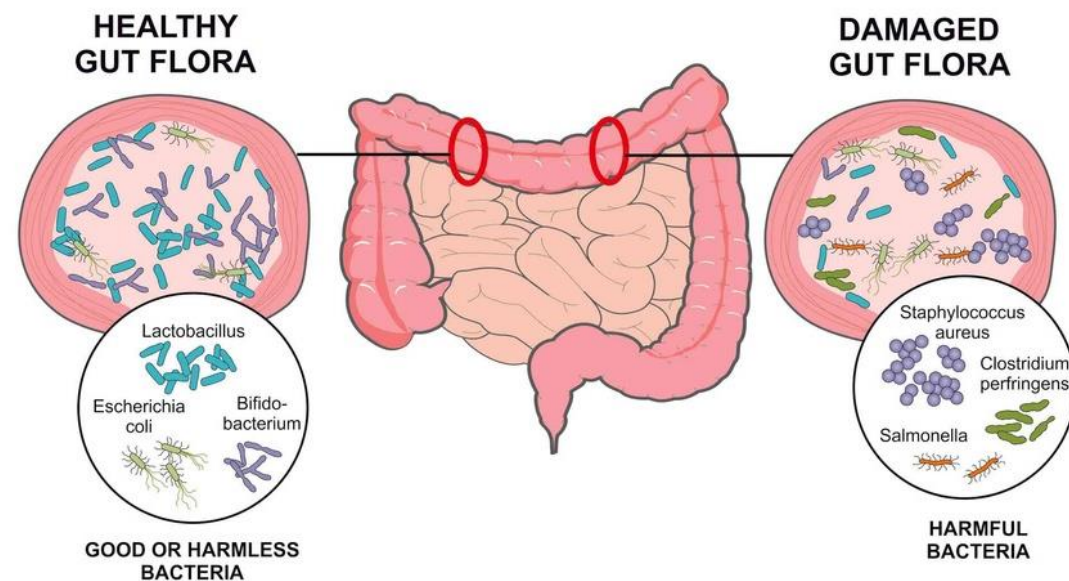


Phase 2 Detoxification

- **Methylation**
- **COMT**
- **17 B HSD to make Estriol**



Phase 3 The Gut and Beta Glucuronidase



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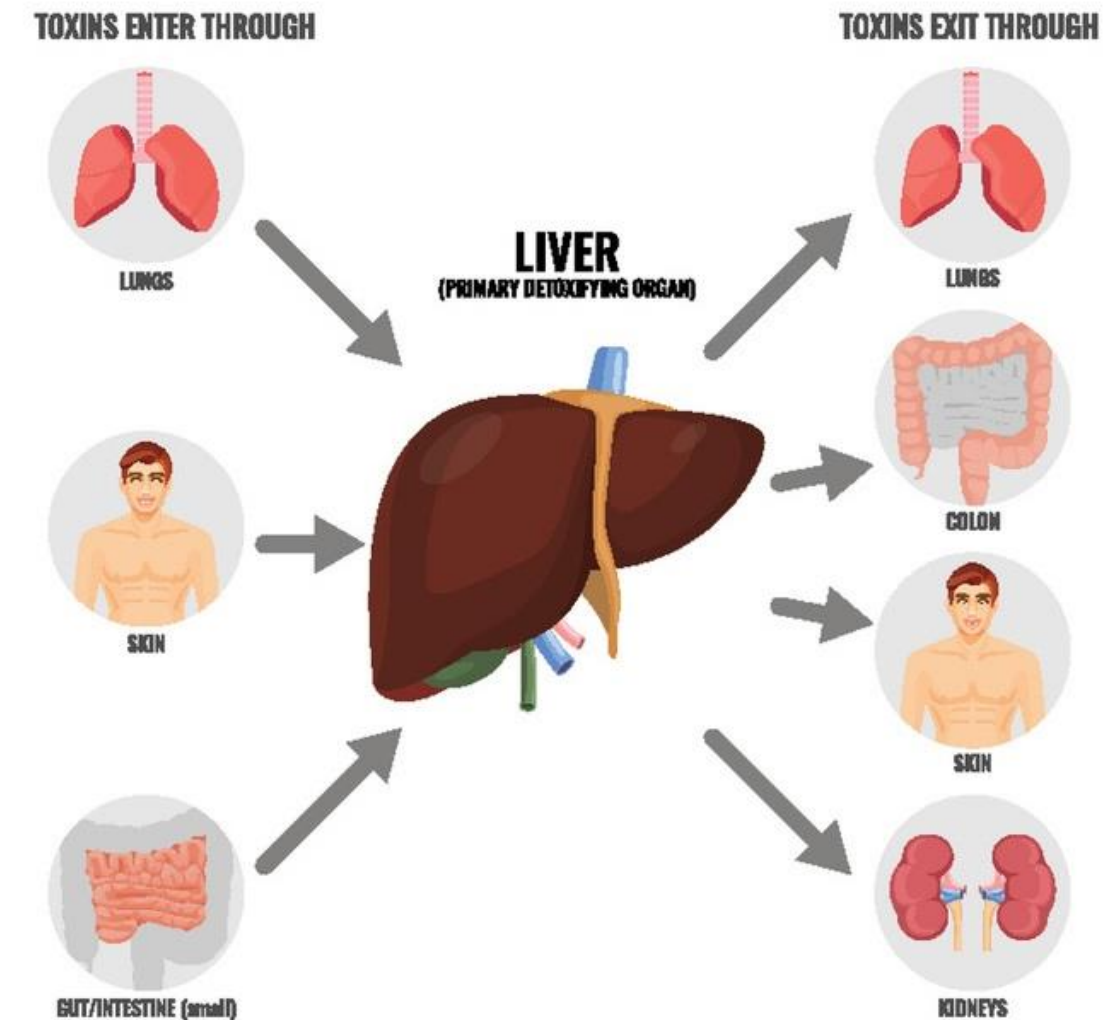
**Can deconjugate estrone and
send back into the circulation**

How Do Toxins Affect Us?

We are all exposed in our environment.

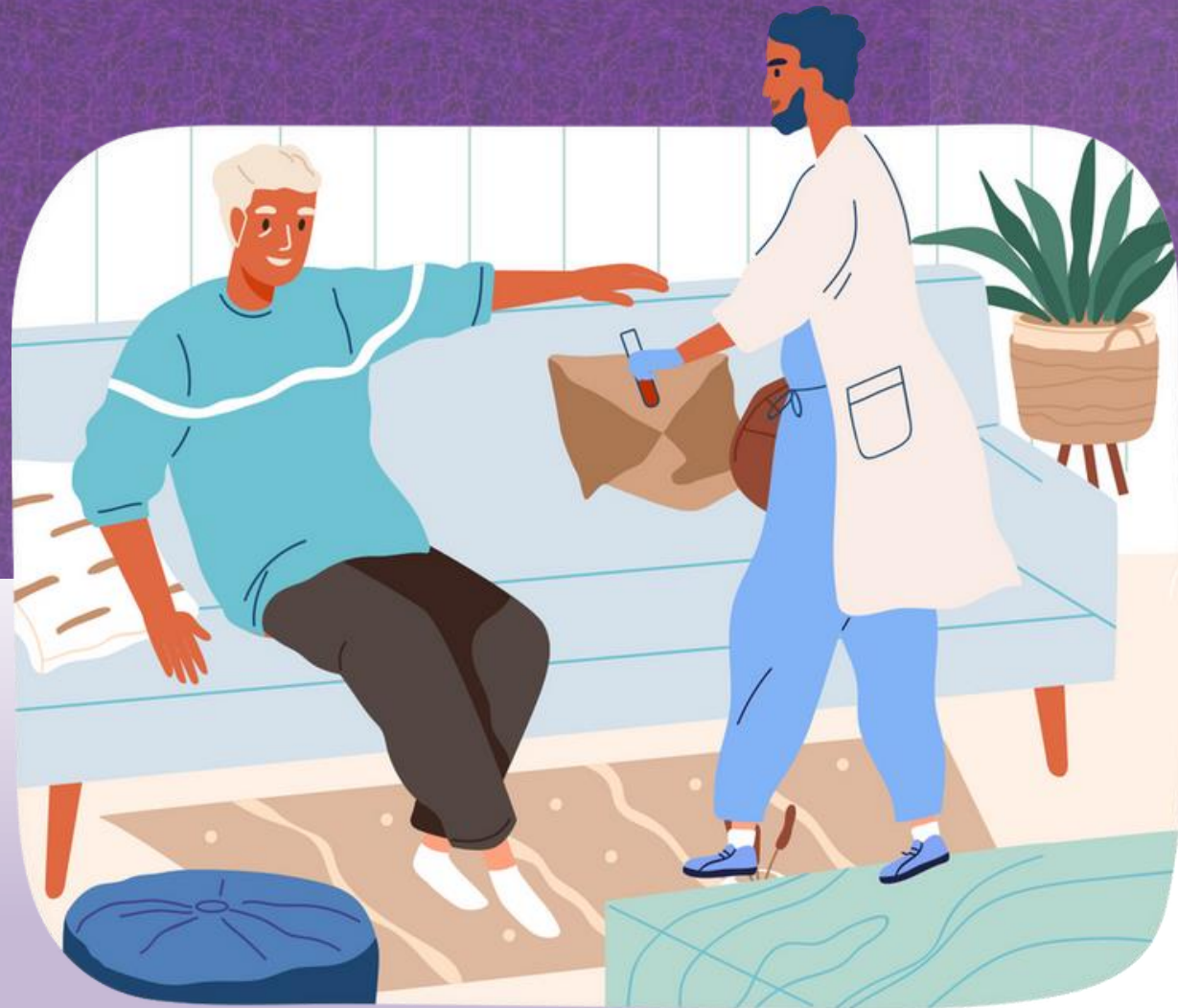
They can come in through breathing, physical contact and consuming food.

THE PROCESS OF DETOXIFICATION AND ELIMINATION



Endocrine Disrupting Chemicals

- EDC's are chemicals or combos of chemicals that interfere with our hormones
- Obesogens: a subset of EDC's that interfere metabolism
- **Types of Obesogens (Metabolism Blockers)**
 - Androgen Disruptors
 - Estrogen Disruptors
 - Thyroid Disruptors
 - Mixed



What do Endocrine Disruptors Do?

- They trick our body
- They can speed up reactions or slow them down
- They pretend to be other hormones (like estrogen)
- They can also block our hormones
- Cause liver damage

Endocrine Disruptors

- BPA mimics estrogenic activates of 17 B estradiol- but has a much lesser affinity for ER, but at low concentrations can induce estrogenic effects
- BPA can also bind to AR and Progesterone receptors
- Phthalates are in plastics, cosmetics, and household products. They bind ER alpha, ER Beta, and AR. They stimulate or inhibit ER but only inhibit AR.

Endocrine Disruptors

- BPA mimics estrogenic activates of 17 B estradiol- but has a much lesser affinity for ER, but at low concentrations can induce estrogenic effects
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- Phthalates are in plastics, cosmetics, and household products. They bind ER alpha, ER Beta, and AR. They stimulate or inhibit ER but only inhibit AR.

Hormone Zoomer



Case Report

- **Late Perimenopausal Female**
- **Using Estradiol patch 0.075mcg**
- **Progesterone 150mg SR**

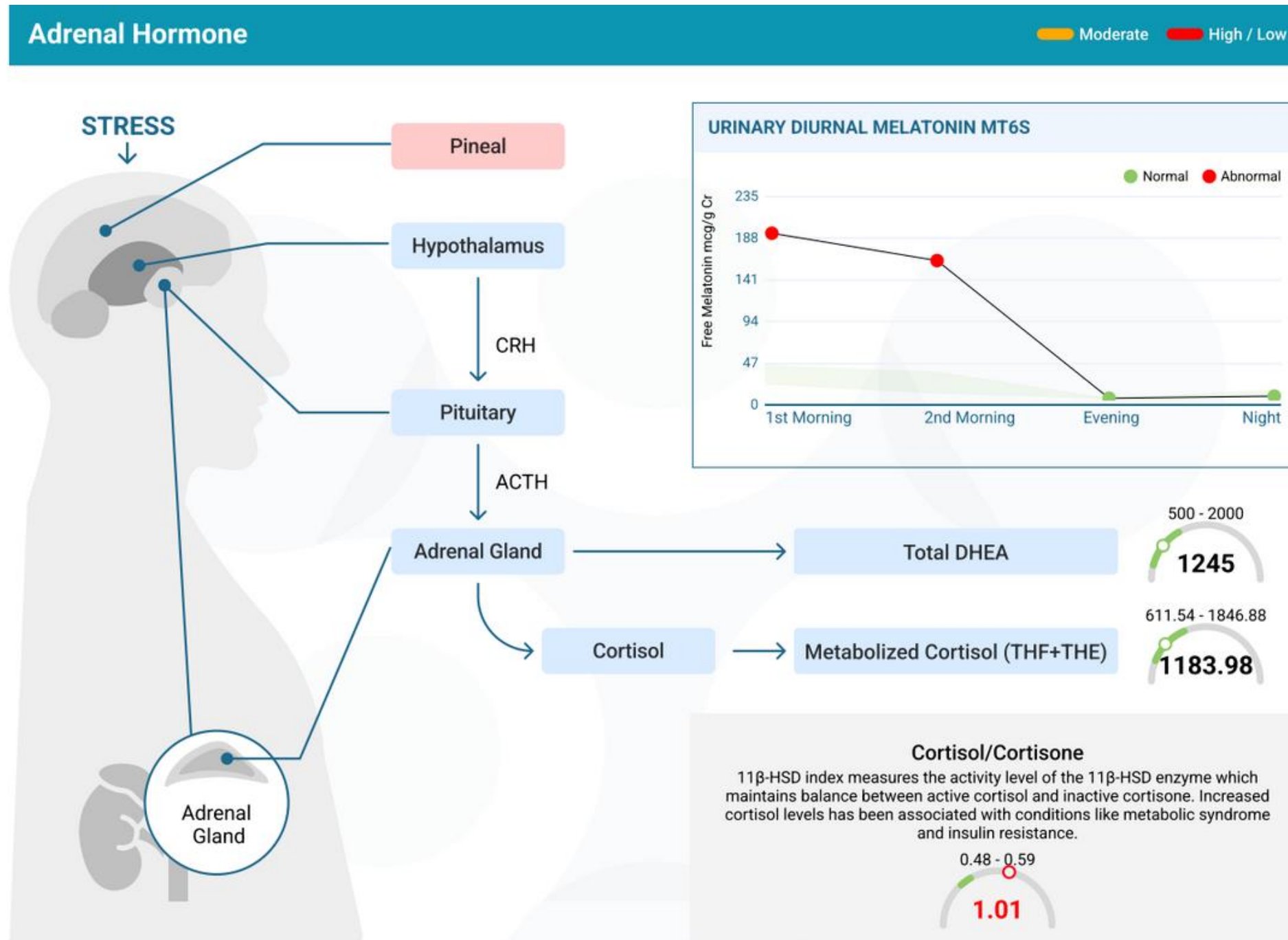


Case Report

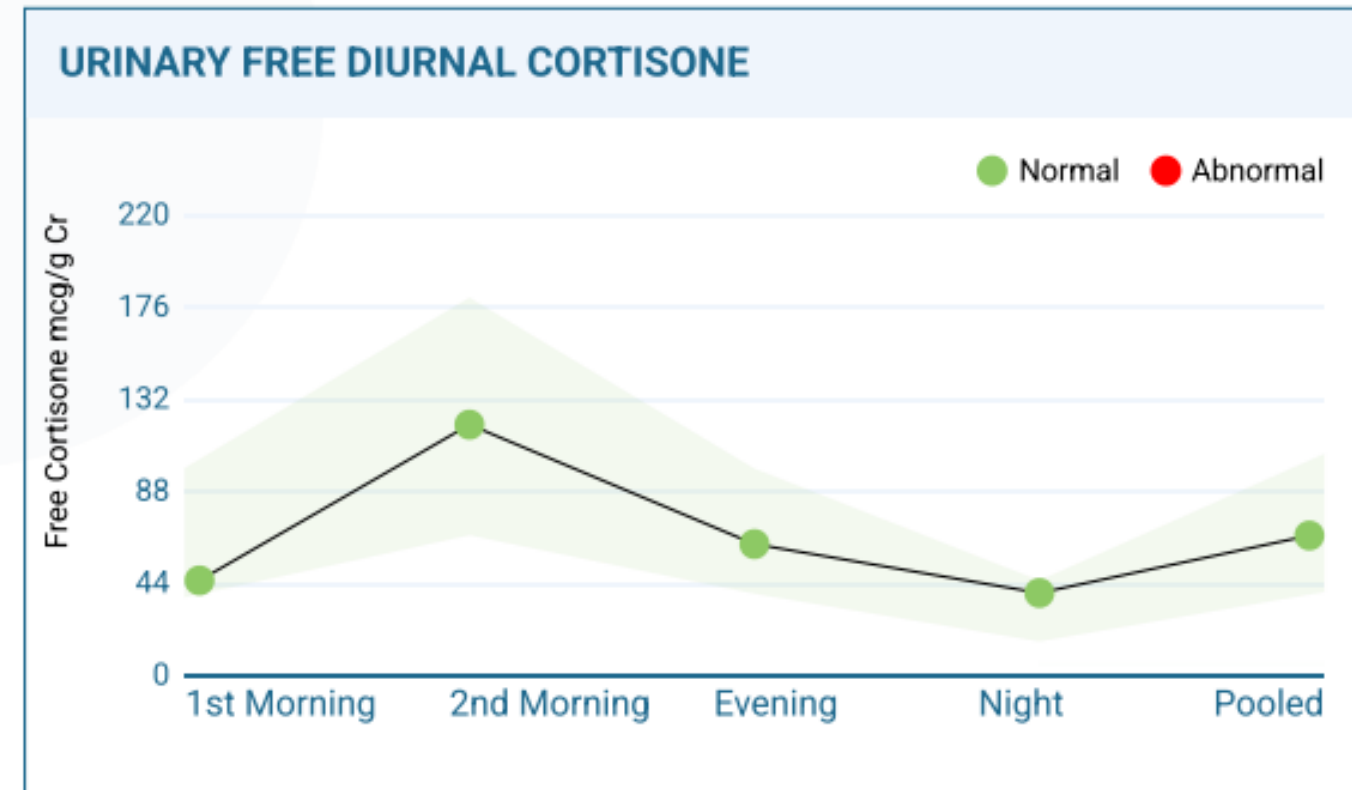
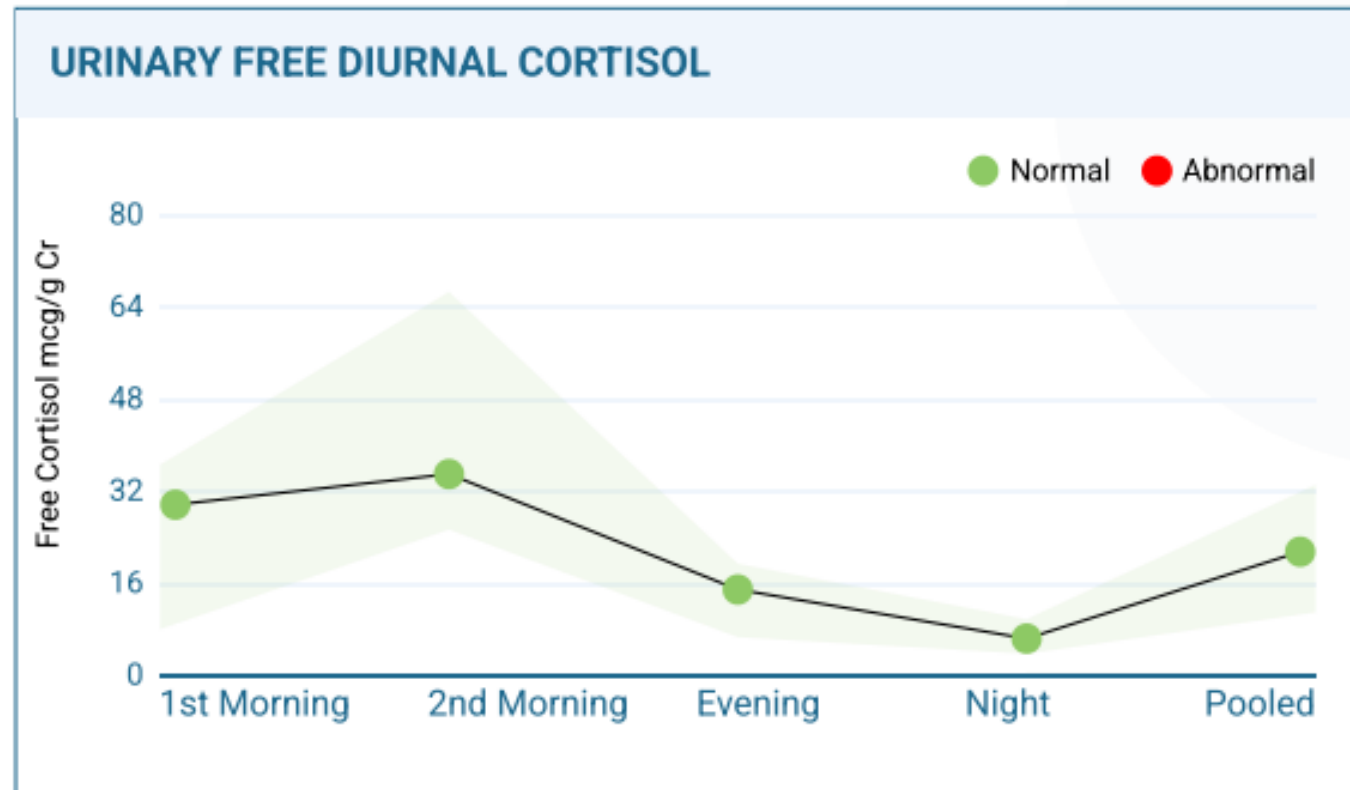
- **Using Estradiol patch 0.075mcg-test mid patch**
- **Progesterone 150mg SR-have them take after nighttime collection**
- **No DHEA x 48 hours**
- **Try to hold melatonin**



Adrenal Hormones







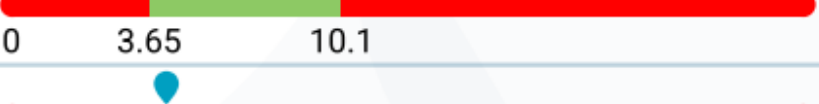

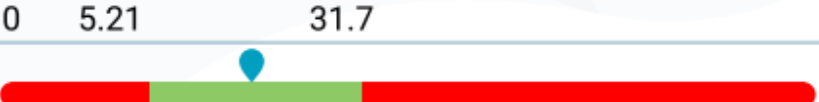

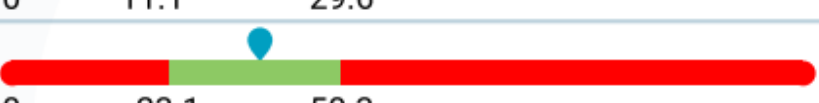
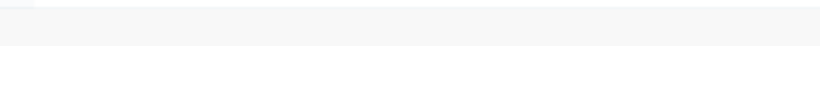
Cortisol and Cortisone



Cortisol and Cortisone interconvert (11β -HSD)

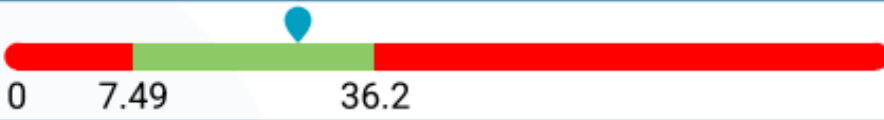






Adrenal Hormones

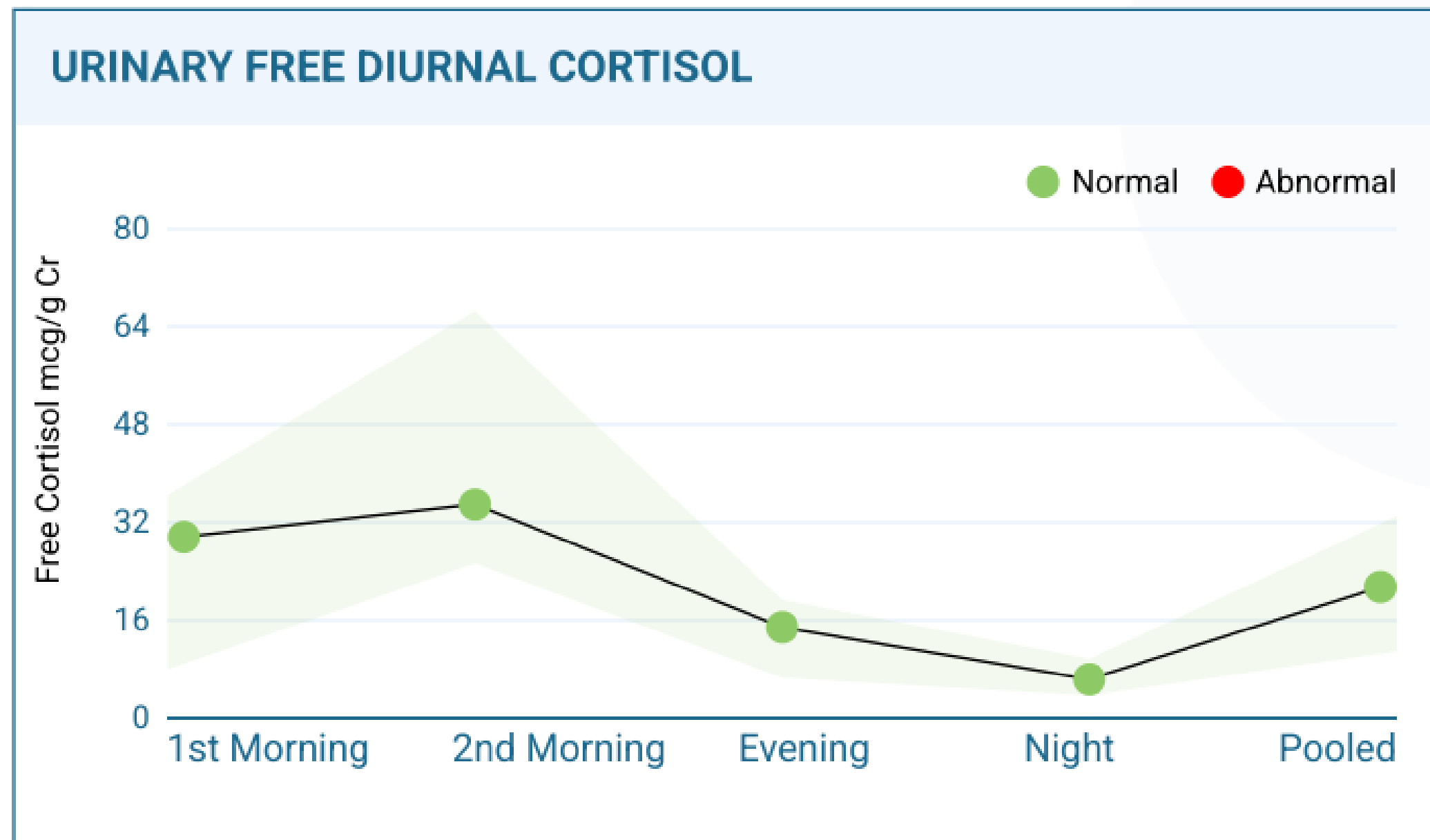
Adrenal Hormones				
Test Name	Current	Previous	Result	Reference
b-Tetrahydrocortisol (b-THF) (mcg/g)	173.87			200.65-500.78
a-Tetrahydrocortisol (a-THF) (mcg/g)	63.29			10.11-67.15
b-Tetrahydrocortisone (b-THE) (mcg/g)	946.83			400.78-1278.95
Deoxycorticosterone (mcg/g)	1.93			0.65-2.18
Corticosterone (mcg/g)	9.44			3.66-10.12
DHEA (mcg/g)	38.26			16.11-138.06
DHEA-S (mcg/g)	86.63			5.22-31.78
Metabolized Cortisol (THF+THE) (mcg/g)	1183.98			611.54-1846.88
Total Cortisol (mcg/g)	38.25			11.18-29.63
Total Cortisone (mcg/g)	37.89			23.11-50.33

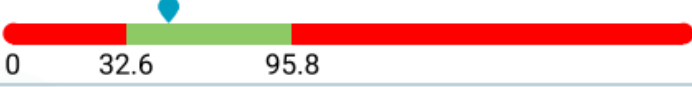

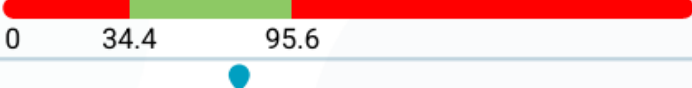

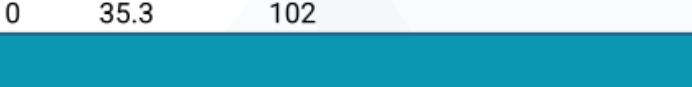

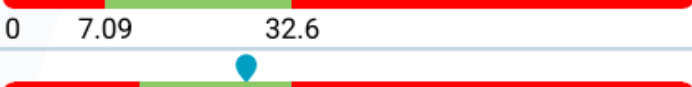


Cortisol Rhythm



Diurnal Cortisol				
Test Name	Current	Previous	Result	Reference
Free Cortisol (1st Morning) (mcg/g)	29.23			7.5-36.2
Free Cortisol (2nd Morning) (mcg/g)	34.58			24.9-66.4
Free Cortisol (Evening) (mcg/g)	14.40			6.1-18.9
Free Cortisol (Night) (mcg/g)	5.83			3.2-9.2
Free Cortisol (pooled) (mcg/g)	21.01			10.43-32.68

Adrenal Hormones- Cortisol



Diurnal Cortisone				
Test Name	Current	Previous	Result	Reference
Free Cortisone (1st Morning) (mcg/g)	40.97			32.7-95.8
Free Cortisone (2nd Morning) (mcg/g)	117.12			63.1-179.2
Free Cortisone (Evening) (mcg/g)	58.74			34.5-95.6
Free Cortisone (Night) (mcg/g)	34.84			11.2-40.9
Free Cortisone (pooled) (mcg/g)	62.92			35.38-102.88
Diurnal Melatonin				
Test Name	Current	Previous	Result	Reference
Melatonin (1st Morning) (mcg/g)	192.10			17.5-40.2
Melatonin (2nd Morning) (mcg/g)	160.56			7.1-32.6
Melatonin (Evening) (mcg/g)	1.79			0.87-2.0
Melatonin (Night) (mcg/g)	4.18			1.9-12.3

SUPPORTIVE SUPPLEMENT SUGGESTIONS *(ENHANCES SPECIFIC ASSOCIATED FUNCTIONS WITHOUT INFLUENCING MARKERS DIRECTLY)*

Vitamin C(90 mg/day): Vitamin C supplementation improves the severity of anxiety in individuals diagnosed with Generalized Anxiety Disorder by increasing serotonin concentrations and decreasing the levels of the inflammatory biomarkers.

Omega-3 fatty acids(950 mg/day): Omega-3 supplements may be efficacious in reducing symptoms of depression and anxiety in adults, particularly as an adjunct to antidepressant medication.

Lavender oil(80 mg/day): Oral lavender oil preparation showed a significant beneficial influence on quality and duration of sleep and improved general mental and physical health .

Phosphatidylserine(400 mg): Phosphatidylserine supplementation is associated with feeling less stressed and having a better mood.



What Adrenal Supplements Would You Recommend to this patient?

Supplement Recommendations

Adrenal Hormones

SUPPLEMENT SUGGESTIONS

Vitamin D(600 IU/day): Vitamin D supplements can lower DHEA-S levels by enhancing the activity of 11 β -hydroxysteroid dehydrogenase type 1 (11 β -HSD1), which converts inactive cortisone to active cortisol. Increased cortisol levels can inhibit DHEA-S production through negative feedback on the adrenal glands. Consequently, this mechanism results in reduced DHEA-S levels in the body.

Magnesium(350 mg/day): Magnesium supplements decrease cortisol by regulating the hypothalamic-pituitary-adrenal (HPA) axis, which controls stress response. Magnesium acts as a cofactor for enzymes involved in neurotransmitter synthesis, promoting GABA activity, and reducing excessive neuronal firing, which helps lower cortisol production. Additionally, magnesium enhances sleep quality, further reducing cortisol levels.

Vitamin C(1500 mg/day): Vitamin C supplementation decreases cortisol levels by reducing the secretion of cortisol in response to stress. It supports the adrenal glands, which produce cortisol, thereby improving their function and reducing excessive cortisol release. Additionally, vitamin C acts as an antioxidant, mitigating oxidative stress that can stimulate cortisol production.

Ashwagandha(600 mg/day): Ashwagandha or its root extract decreases cortisol by inhibiting the activity of the hypothalamic-pituitary-adrenal (HPA) axis, leading to reduced adrenal cortisol production. It enhances the resilience of the body to stress, promoting homeostasis and lowering cortisol levels. Additionally, ashwagandha's bioactive compounds modulate neurotransmitter activity, further aiding in stress reduction.

Tangeretin(200 mg/day): Tangeretin, a polymethoxylated flavone found in citrus peels, decreases cortisol levels by inhibiting the enzyme 11 β -hydroxysteroid dehydrogenase type 1 (11 β -HSD1), which converts inactive cortisone to active cortisol. This inhibition reduces the overall production of cortisol within tissues. Additionally, tangeretin's antioxidant properties may mitigate stress-induced cortisol secretion, further lowering cortisol levels in the body.





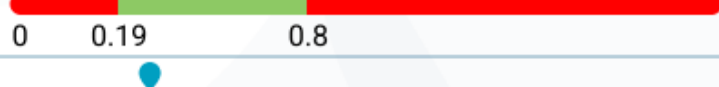



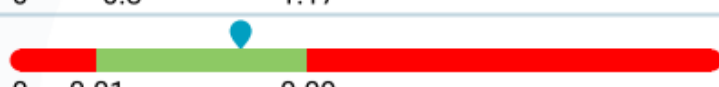
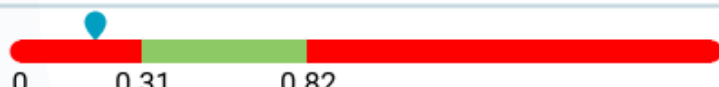



SUPPORTIVE SUPPLEMENT SUGGESTIONS (ENHANCES SPECIFIC ASSOCIATED FUNCTIONS WITHOUT INFLUENCING MARKERS DIRECTLY)

Magnesium(350 mg/day): Magnesium supplements enhance the activity of 11 β -HSD type 2 by increasing intracellular magnesium levels, which stabilize the enzyme's structure, thus promoting its catalytic function. This leads to more efficient conversion of active cortisol to inactive cortisone, thereby modulating the body's stress response and reducing hypertension.

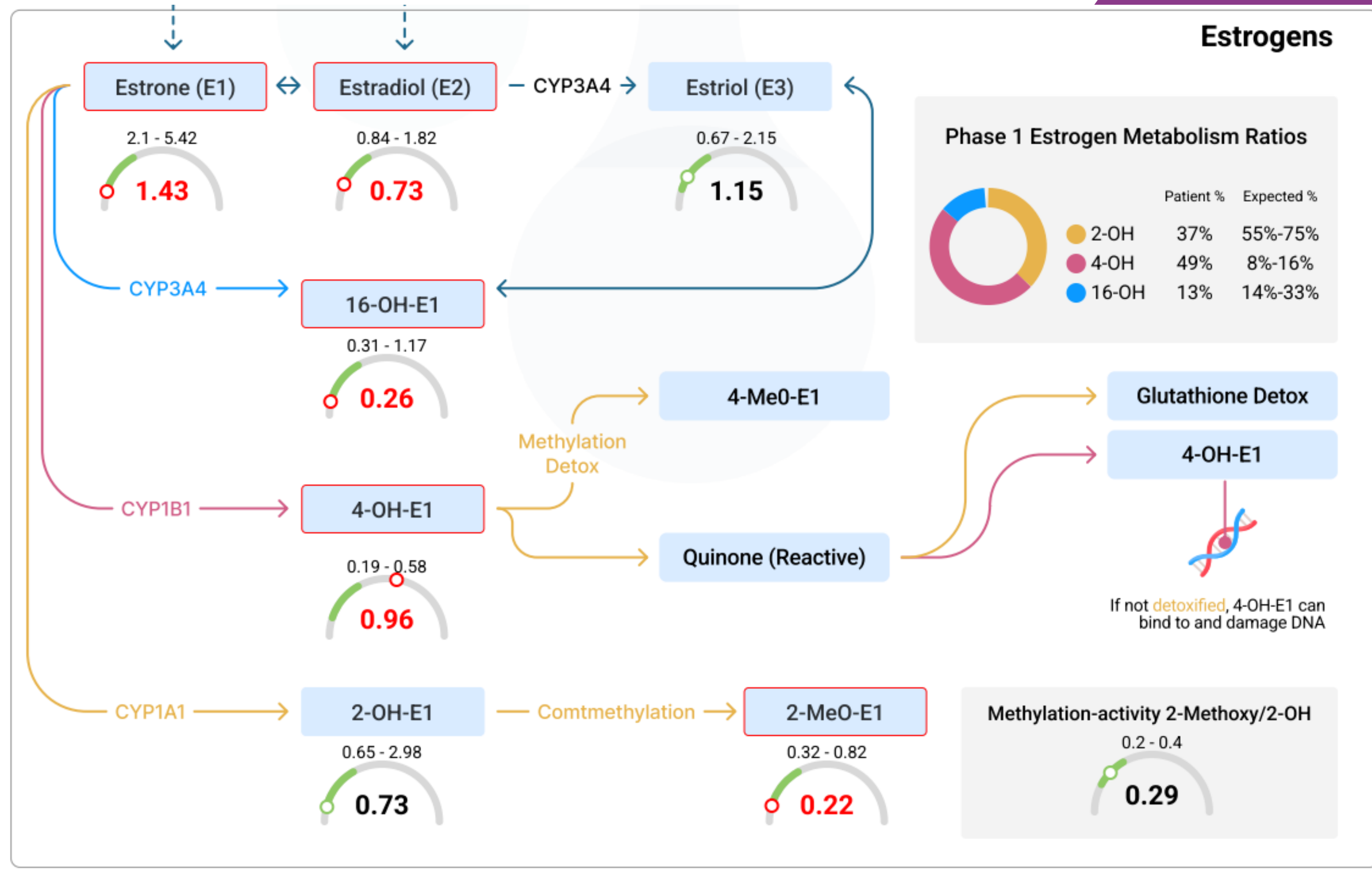
5-HTP(300 mg/day): 5-HT supplements, like 5-HTT, provide a precursor molecule for serotonin synthesis, leading to higher serotonin levels.

Sex Hormones

- *Progesterone Metabolites*
- *Androgen metabolites*
- *5 Alpha Preference*
- *Estrogens*
- *Estrogen Metabolites*
- *Methylation*
- *4 OH- 4ME metabolites*

Estrogen				
Test Name	Current	Previous	Result	Reference
Estradiol (E2) (mcg/g)	0.73			0.84-1.82
Estrone (E1) (mcg/g)	1.43			2.1-5.42
Estriol (E3) (mcg/g)	1.15			0.67-2.15
Total Estrogen (mcg/g)	6.21			5.42-16.13
2-OH Estradiol (mcg/g)	0.11			0.2-0.8
2-OH Estrone (mcg/g)	0.73			0.65-2.98
4-OH Estradiol (mcg/g)	0.51			0.11-0.22
4-OH Estrone (mcg/g)	0.96			0.19-0.58
16a-OH Estrone (mcg/g)	0.26			0.31-1.17
2-MeO Estradiol (mcg/g)	0.07			0.02-0.09
2-MeO Estrone (mcg/g)	0.22			0.32-0.82
4-MeO Estradiol (mcg/g)	<0.01			≤0.05
4-MeO Estrone (mcg/g)	0.03			≤0.05

Estrogen Detox










What is your assessment of Estrogen Detox?

–Any SNPs Suggested?

Sex Hormones

Progesterone				
Test Name	Current	Previous	Result	Reference
Allopregnanolone (mcg/g)	4.56			2.57-19.88
3aDihydroprogesterone (mcg/g)	0.52			0.72-2.5
20aDihydroprogesterone (mcg/g)	13.13			4.11-12.68
b-Pregnanediol (mcg/g)	909.36			450.8-1748.2
a-Pregnanediol (mcg/g)	471.53			120.3-499.8

SUPPLEMENT SUGGESTIONS

Vitamin D(600 IU/day): Vitamin D supplements can lower progesterone levels by modulating the expression of enzymes involved in steroidogenesis. Specifically, Vitamin D may influence the activity of cytochrome P450 enzymes, which are crucial in the synthesis and metabolism of progesterone. This alteration in enzyme function can lead to decreased progesterone production.

SUPPORTIVE SUPPLEMENT SUGGESTIONS (ENHANCES SPECIFIC ASSOCIATED FUNCTIONS WITHOUT INFLUENCING MARKERS DIRECTLY)

Vitamin E(22 IU/day): Vitamin E supplements increase progesterone levels by enhancing the synthesis of this hormone in the ovaries. This effect is attributed to Vitamin E's role as an antioxidant, which reduces oxidative stress and supports hormonal balance. Consequently, it helps in regulating menstrual cycles and supporting reproductive health.

Vitamin B6(200 mg/day): Vitamin B6 supplements enhance progesterone production by promoting the conversion of cholesterol into pregnenolone, a precursor of progesterone. This process is facilitated through the activation of enzymatic pathways involved in steroidogenesis. Additionally, Vitamin B6 helps regulate the balance of estrogen and progesterone, supporting overall hormonal harmony.

Recommendations

SUPPLEMENT SUGGESTIONS

Soy protein isolates(40g/d): Soy protein isolates contain phytoestrogens, particularly isoflavones, which can mimic estrogen by binding to estrogen receptors in the body. This binding stimulates the hypothalamus-pituitary-gonadal axis, leading to increased production of estradiol. Additionally, isoflavones may inhibit the metabolism of estradiol, further increasing its levels.

Isoflavones(2.01 mg/day): Isoflavone supplements increase estrone levels by mimicking estrogen activity due to their structural similarity to estrogen. They bind to estrogen receptors, which can stimulate the synthesis of estrone in target tissues. This estrogenic effect can lead to elevated estrone concentrations in the body.

Soy isoflavones(2.01 mg/day): Soy isoflavones, such as genistein, can influence estrogen metabolism by reducing the formation of genotoxic metabolites and promoting detoxification pathways, which can indirectly support an increase in the relative levels of 2-OH estradiol.




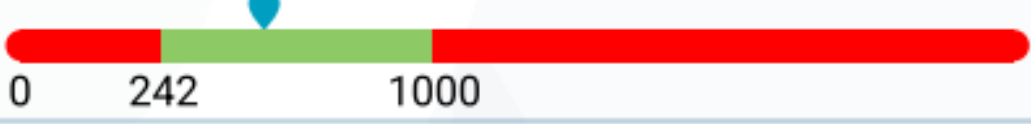
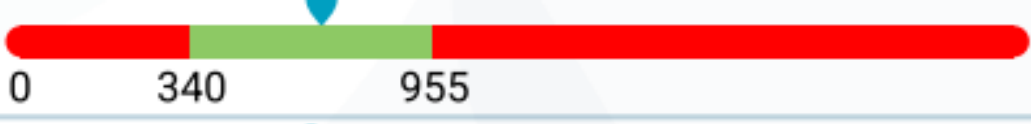
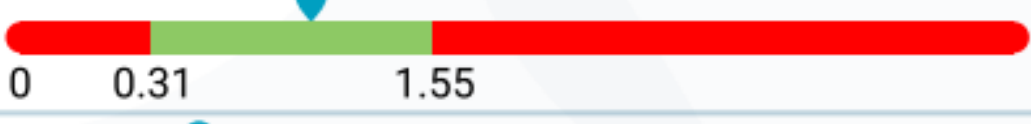


Soy(2.01 mg/day): Soy supplements help reduce 4-OH estrone levels primarily through their content of isoflavones, which act as phytoestrogens. These compounds compete with endogenous estrogens for receptor binding, thereby decreasing the formation of 4-OH estrone. Additionally, soy isoflavones may enhance the activity of liver enzymes involved in estrogen metabolism, promoting the excretion of estrogen metabolites.

Flaxseed(10 g/day): Flaxseed supplements increase 16 α -OH estrone by providing lignans, which are converted into enterolactone and enterodiol in the gut, altering estrogen metabolism, shifting the balance towards 16 α -hydroxylation of estrone and promoting the formation of 16 α -OH estrone through the increased activity of hepatic hydroxylase enzymes.



Any other supplement suggestions for Estrogen Detox?

Testosterone

Testosterone				
Test Name	Current	Previous	Result	Reference
Testosterone (T) (mcg/g)	2.38			1.18-4.11
Epi-Testosterone (Epi-T) (mcg/g)	0.96			1.95-4.61
Androstenedione (mcg/g)	5.05			3.15-15.44
Androsterone (mcg/g)	475.83			242.6-1000.8
Etiocholanolone (mcg/g)	681.71			340.3-955.1
5a-DHT (mcg/g)	1.04			0.32-1.55
5a,3a-Androstanediol (mcg/g)	3.13			2.94-14.59
5b-Androstanediol (mcg/g)	39.18			4.15-15.66

Supplement Suggestions- Testosterone

SUPPORTIVE SUPPLEMENT SUGGESTIONS (ENHANCES SPECIFIC ASSOCIATED FUNCTIONS WITHOUT INFLUENCING MARKERS DIRECTLY)



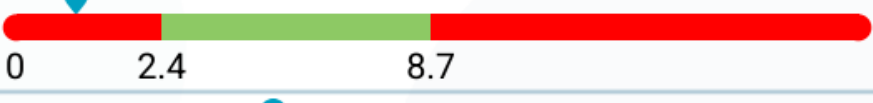


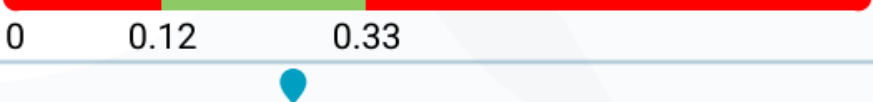


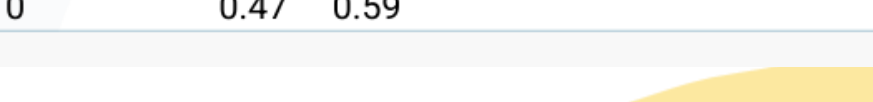
Oral Dehydroepiandrosterone(200 mg/day): Oral dehydroepiandrosterone (DHEA) supplementation increases circulating DHEA and its sulfate (DHEAS), which serve as precursors for testosterone synthesis. Elevated testosterone production raises the testosterone/epitestosterone (T/E) ratio, as epitestosterone remains largely unaffected. This altered ratio is a key marker for detecting exogenous testosterone use in doping tests.

Chromium picolinate(200 µg/day): Chromium picolinate enhances insulin sensitivity by facilitating glucose metabolism, which helps regulate blood sugar levels. This, in turn, can reduce elevated androgen levels linked to hirsutism. Improved insulin sensitivity may contribute to decreased hair growth in individuals with insulin resistance-related hirsutism.

Calcium and vitamin D (Ca/ Vit.D)(1,000 mg of calcium): Calcium and vitamin D supplements prevent hirsutism by maintaining adequate calcium levels and enhancing vitamin D absorption, which are crucial for proper hormonal balance. Vitamin D helps regulate androgen levels, while calcium supports overall endocrine function. This combined action helps mitigate the excessive hair growth associated with hirsutism.

Melatonin(5 mg/day): Melatonin supplements help prevent hirsutism by regulating androgen levels and reducing the activity of hair growth-promoting hormones. Additionally, melatonin exhibits anti-inflammatory effects that can address underlying conditions contributing to excessive hair growth. This combined action helps manage and mitigate the symptoms associated with hirsutism.

Hormone Ratios

Hormone Ratios				
Test Name	Current	Previous	Result	Reference
E3/(E1+E2) Ratio	0.53			≤0.3
2-OH (E1 + E2)/16a-OH E1	3.25			1.5-6.0
2-OH E1 /4-OH E1 (None)	0.8			2.5-8.7
2-MeO E1/2-OH E1	0.29			0.2-0.4
4-MeO E1/4-OH E1	0.03			0.04-0.15
4-MeO E2/4-OH E2	0.02			0.13-0.33
T/Epi-T	2.47			0.4-3.0
b-Pregnanediol/E2	1250.84			536.67-960.55
Cortisol/Cortisone (mcg/g)	1.01			0.48-0.59

Supplement Suggestions

SUPPORTIVE SUPPLEMENT SUGGESTIONS (ENHANCES SPECIFIC ASSOCIATED FUNCTIONS WITHOUT INFLUENCING MARKERS DIRECTLY)

Soy(40 mg/day): Soy supplements contain phytoestrogens like genistein, which compete with estrone for estrogen receptors, reducing estrone's effects. These compounds also influence estrogen metabolism, leading to lower circulating estrone levels. The combined impact helps to modulate estrogenic activity in the body.

Wheat bran(10 g/day): Wheat bran increases dietary fiber intake, which binds estrogens and enhances their excretion through the feces. This process reduces the enterohepatic recirculation of estrogens, leading to lower serum estrone levels. Consequently, decreased serum estrone can also reduce urinary estrone excretion.

Absorbable 3,3'-Diindolylmethane (DIM)(108 mg/day): Absorbable 3,3'-Diindolylmethane (DIM) supplement increases serum 2-OH estrone by enhancing the conversion of estradiol to 2-hydroxyestrone through induction of cytochrome P450 enzymes, specifically CYP1A1 and CYP1B1, which metabolize estrogen into its 2-hydroxy form, potentially reducing the carcinogenicity of estrogen metabolites.

Flaxseed (10 g/day): Flaxseed supplements increase 2-OH estrone levels primarily through the action of lignans, which are converted by gut microbiota into enterolactone and enterodiol. These compounds enhance estrogen metabolism, leading to a higher ratio of 2-OH estrone to its more potent counterpart, 16 α -OH estrone.




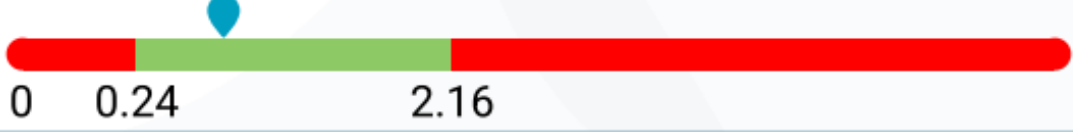

Indole-3-carbinol(200 mg/day): Indole-3-carbinol enhances the production of 2-hydroxyestrone by promoting the activity of cytochrome P450 enzymes, particularly CYP1A1 and CYP1A2. These enzymes increase the hydroxylation of estrogen, leading to the formation of 2-OH estrone. Additionally, these compounds may modulate estrogen metabolism by influencing the balance of estrogen metabolites.


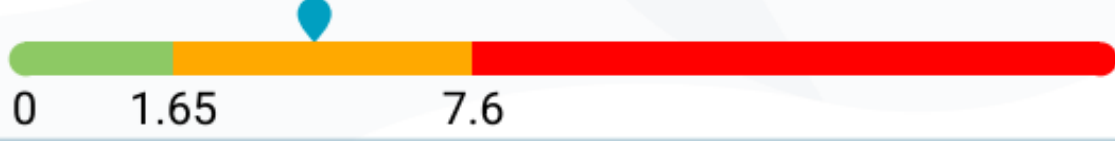

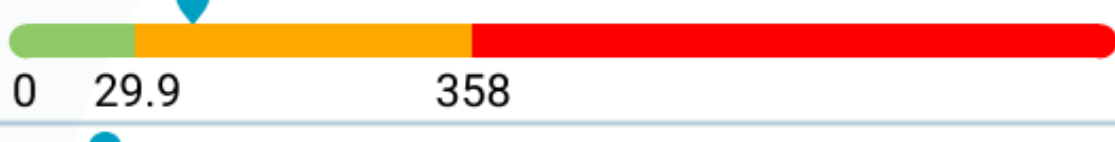
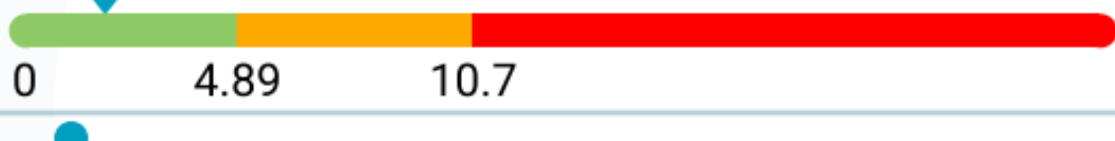



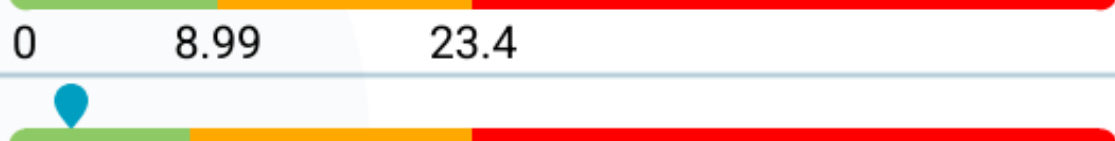
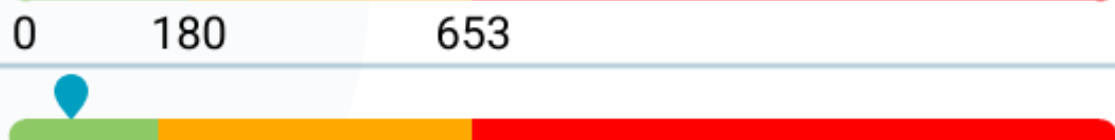

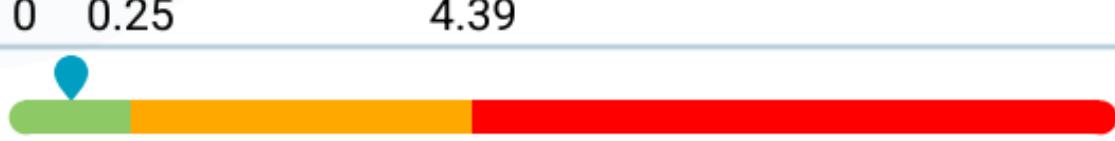
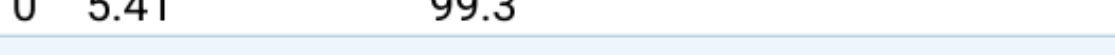
HMR lignan(40 mg/day): HMR lignan supplement enhances the production of 2-hydroxyestrone by promoting the activity of cytochrome P450 enzymes, particularly CYP1A1 and CYP1A2. These enzymes increase the hydroxylation of estrogen, leading to the formation of 2-OH estrone. Additionally, these compounds may modulate estrogen metabolism by influencing the balance of estrogen metabolites.

Pyridoxal 5'-phosphate(30 mg/kg/day): Pyridoxal 5'-phosphate, the active form of vitamin B6, decreases breast cancer by modulating gene expression and inhibiting angiogenesis, thus reducing tumor growth. It also enhances the immune response against cancer cells and induces apoptosis.


Vitamin D(600 IU/day): Vitamin D supplements can lower progesterone levels by modulating the expression of enzymes involved in steroidogenesis. Specifically, Vitamin D may influence the activity of cytochrome P450 enzymes, which are crucial in the synthesis and metabolism of progesterone. This alteration in enzyme function can lead to decreased progesterone production.

Oxidative Stress

Oxidative Stress				
Test Name	Current	Previous	Result	Reference
8-hydroxy-2'-deoxyguanosine (8-OHdG) (mcg/g)	0.85			≤4.77
Creatinine				
Test Name	Current	Previous	Result	Reference
Creatinine (1st Morning) (mg/ml)	1.20			0.25-2.16
Creatinine (2nd Morning) (mg/ml)	2.33			0.25-2.16
Creatinine (Evening) (mg/ml)	0.60			0.25-2.16
Creatinine (Night) (mg/ml)	0.58			0.25-2.16

Endocrine Disruptors	Current	Previous	Result	Reference
Atrazine ^ (ug/g)	0.06			≤0.05
Glyphosate (ug/g)	4.38			≤7.6
Bisphenol A (BPA)^ (ug/g)	2.17			≤5.09
Triclosan (TCS)^ (ug/g)	42.97			≤358
Perchlorate (PERC)^ (ug/g)	1.42			≤10.7
Monoethyl Phthalate (MEP)^ (ug/g)	1.33			≤541
Mono-2-ethylhexyl phthalate (MEHP)^ (ug/g)	0.43			≤8.47
Mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)^ (ug/g)	1.34			≤37.7
Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)^ (ug/g)	4.67			≤23.4
Methylparaben^ (ug/g)	1.74			≤653
Propylparaben^ (ug/g)	0.02			≤222
Butylparaben^ (ug/g)	0.04			≤4.39
Ethylparaben ^ (ug/g)	0.02			≤99.3

Endocrine Disruptors

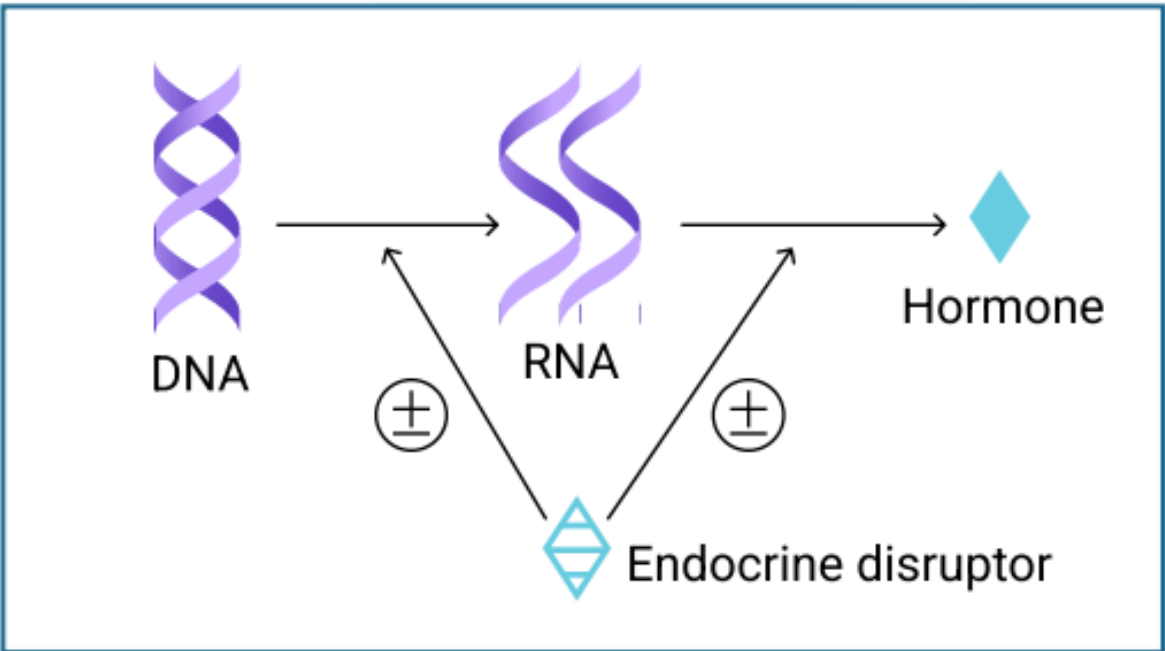
Test Name	Current	Previous	Result		Reference
			75th	95th	
Atrazine ^ (ug/g)	0.06				≤0.05

Atrazine is a chlorinated herbicide that prevents pre and post-emergence broadleaf weeds in crops like maize (corn), soybean, and sugarcane, as well as on turf like golf courses and residential lawns. It used to be the most commonly detected pesticide contaminating drinking water. It is an endocrine disruptor and alters the levels of LH, FSH, testosterone, and estrogen. This can result in delayed puberty and reproduction health problems like low sperm motility and changes in the menstrual cycle. Accumulation of atrazine in the body can affect hormonal function, potentially leading to endocrine disorders and other diseases, which necessitates its elimination to minimize this risk.

HORMONE AFFECTED

Estrogen, LH, FSH

Hormone synthesis



Mechanism

The body can control how hormones are made through feedback mechanisms. For thyroid hormones, this process involves a hormone called TSH activating a receptor and the uptake of iodine, along with other intracellular signals that help activate specific enzymes. Proteins and peptide hormones are stored in vesicles after they are made in the body. In the case of steroid hormones, a precursor (prohormone) is turned into an active hormone by various enzymes. However, some chemicals, known as endocrine disruptors (EDCs), can interfere with hormone production; for example, Atrazine has been shown to boost estrogen production in animals, which is linked to lower testosterone levels and higher estrogen levels in the blood. Additionally, Atrazine increases the activity of aromatase, the enzyme responsible for converting testosterone into estrogen.

SUPPLEMENT SUGGESTIONS

Lycopene(8 mg/day): Lycopene may provide cardioprotective effects and reduce oxidative stress, potentially mitigating atrazine-induced damage, although specific mechanisms against atrazine toxicity are unclear

Spirulina(3 g/day): Spirulina has been shown to reduce oxidative stress and inflammation induced by atrazine (ATZ) in hepatic tissues. It modulates the expression of inflammatory cytokines, up-regulating IL-10 while down-regulating IL-1 β , thereby mitigating hepatotoxic injury.

Vitamin C(75 mg/day): Vitamin C has been shown to ameliorate atrazine-induced oxidative stress and inflammation in hepatic tissues. It helps regulate liver function biomarkers and counteracts apoptosis by enhancing antioxidant defenses.

Soybean(25 g/day): The protective effects of soybeans against atrazine toxicity are not well-documented; however, their isoflavones may provide some antioxidant benefits that could theoretically mitigate oxidative stress.

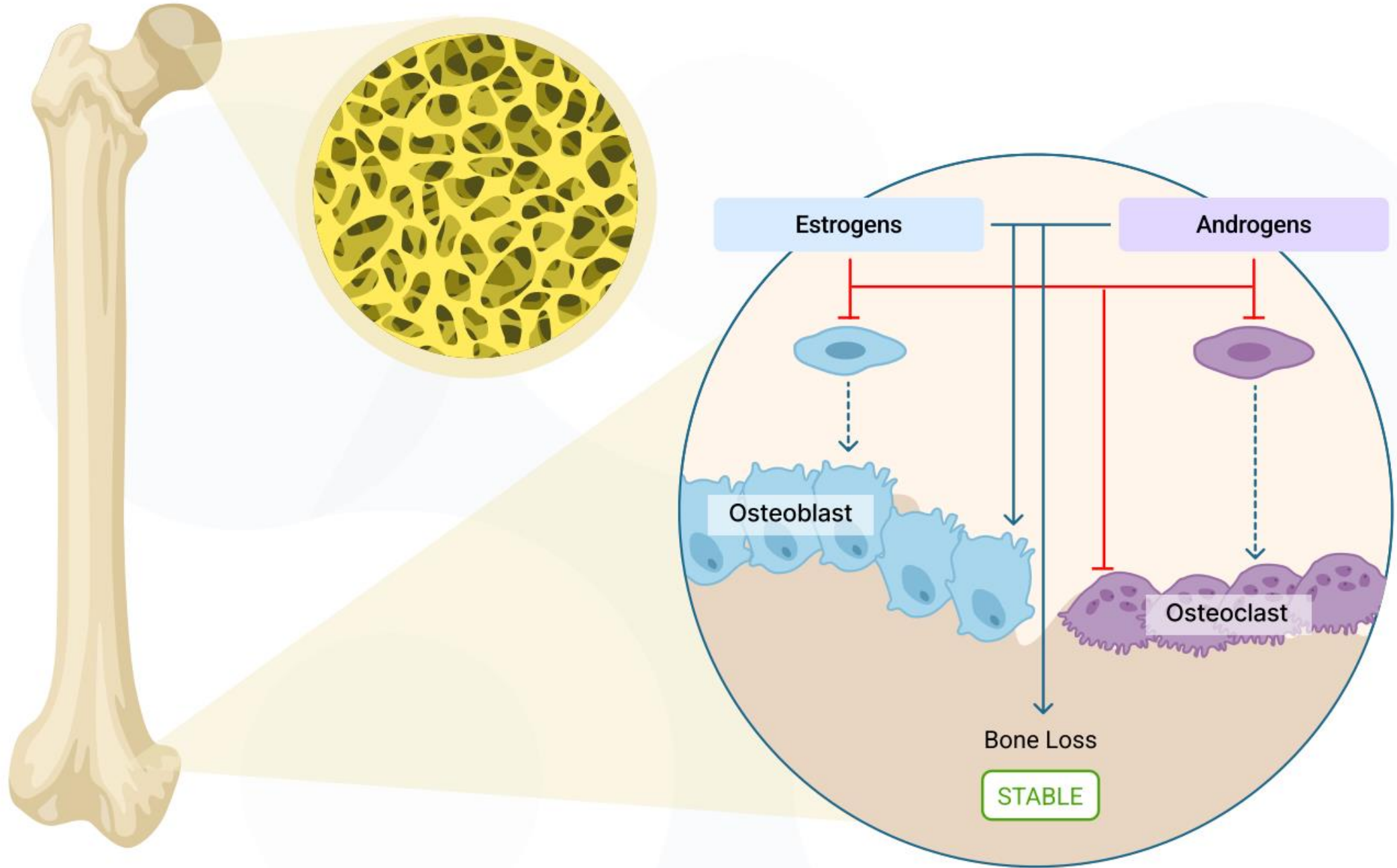
Quercetin(500 mg/day): Quercetin exhibits antioxidant properties that may help reduce oxidative stress and inflammation caused by atrazine exposure, although specific protective effects against atrazine toxicity require further investigation.

Vitamin E(22 IU/day): Vitamin E is known for its antioxidant effects, which can help protect against oxidative damage induced by atrazine; however, specific studies demonstrating its efficacy against atrazine toxicity are limited.

Melatonin(10 mg/day): Melatonin may help mitigate oxidative stress and inflammation associated with atrazine exposure through its antioxidant properties, but specific evidence regarding its protective role against atrazine toxicity is lacking.


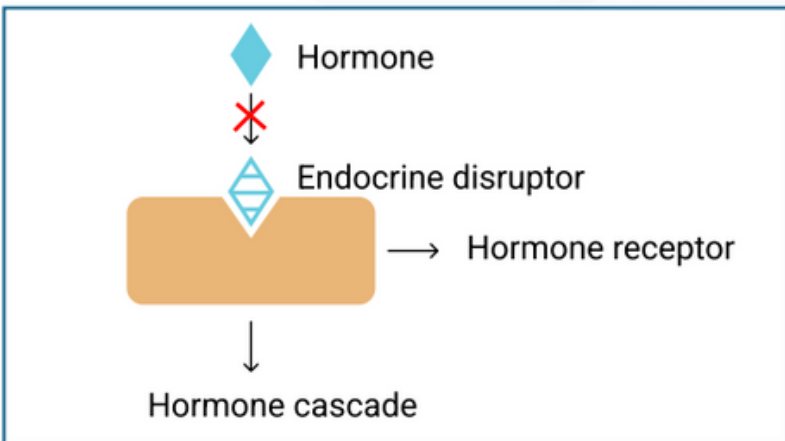
Ginger(15 mg/day): The potential protective effects of ginger against atrazine toxicity are not well-established; however, its anti-inflammatory and antioxidant properties may offer some benefits in reducing oxidative stress related to atrazine exposure.

Curcumin(500 mg/day): Curcumin has shown protective effects against atrazine-induced testicular toxicity by enhancing reproductive hormone levels and improving histological features in studies involving co-treatment with quercetin.




Test Name	Current	Previous	Reference	Test Name	Current	Previous	Reference
Deoxypyridinoline (DPD) (nmol/mmol)	3.53			Pyridinoline (PYD) (nmol/mmol)	20.90		

Endocrine Disruptors

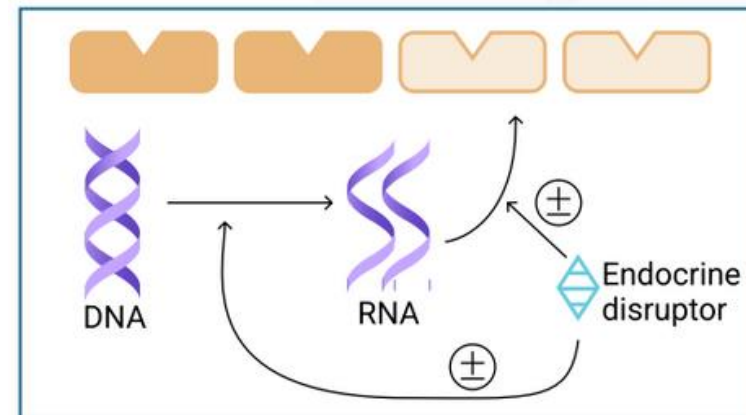
Endocrine Disruptors					
Test Name	Current	Previous	Result		Reference
			75th	95th	
Glyphosate (ug/g)	4.38				≤7.6
<p>Glyphosate is a broad-spectrum systemic herbicide and crop desiccant. It is used to kill weeds, especially annual broadleaf weeds and grasses that compete with crops. Glyphosate interferes with the shikimate pathway, which exists in plants and microorganisms but not in the genomes of mammals, including humans. Exposure by breathing in spray mist from products containing glyphosate may cause irritation in the nose and throat, nausea, and vomiting. Studies report that glyphosate does not induce sensitization and shows no mutagenic, carcinogenic, or teratogenic activity. Hence, glyphosphate exhibits very low toxicity. However, recent studies have shown that glyphosate alters the levels of reporductive and thyroid hormones. Hence, it is essential to adopt relevant measures to remove this toxin from the body upon exposure.</p> <p>HORMONE AFFECTED Estrogen, Progesterone, T4</p>					
Agonist (mimics hormone function)		Mechanism			
		<p>Hormone agonists mimic natural hormones by acting like them; they bind to hormone receptors and trigger similar biological responses. Endocrine-disrupting chemicals (EDCs) act as these mimics, potentially causing harmful effects; Studies indicate that glyphosate can disrupt the hypothalamic-pituitary-thyroid (HPT) axis, leading to increased serum levels of T4 while altering the expression of key regulatory genes involved in thyroid hormone synthesis. This disruption may mimic the effects of natural thyroid hormones, potentially leading to altered metabolic processes. Additionally, glyphosate may affect androgen signaling by interacting with androgen receptors, thereby mimicking the actions of testosterone. In vitro studies show that glyphosate can also activate estrogen receptors such as ERα. However, it does so without directly binding to these receptors, and the precise mechanism remains unclear.</p>			

Endocrine Disruptors

Endocrine Disruptors					
Test Name	Current	Previous	Result		Reference
			75th	95th	
Bisphenol A (BPA)^ (ug/g)	2.17				≤5.09
<p>BPA is an organic synthetic compound used as a starting material for the synthesis of plastics and epoxy resins. BPA-based plastic is clear and tough, and is made into plastic bottles including water bottles, sports equipment, CDs, and DVDs. Epoxy resins containing BPA are used to line water pipes, as coatings on the inside of many food and beverage cans and in making thermal paper such as that used in sales receipts. BPA is an endocrine disruptor and causes fertility problems as well as impotence in males. BPA binds to estrogen and androgen receptors and can disrupt their signalling. They also regulate DNA methylation and gene expression, and interacts with transcription factors. Accumulation of BPA in the body can affect hormonal function, potentially leading to endocrine disorders and other diseases, which necessitates its elimination to minimize this risk.</p>					
HORMONE AFFECTED					
Estrogen, T4, T3, TSH					

Endocrine Disruptors

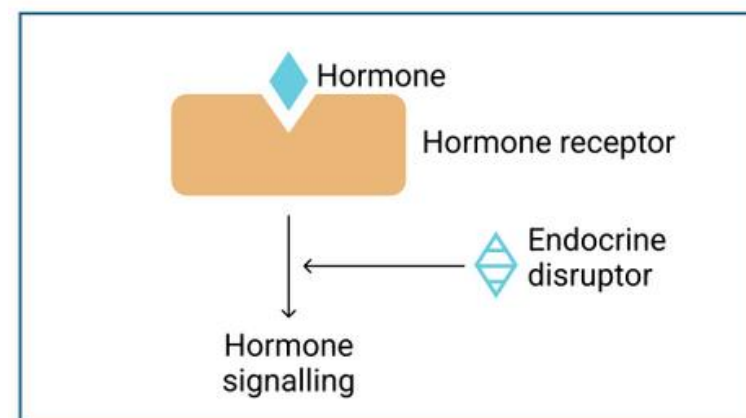
Modifies Receptor Expression



Mechanism

Hormone receptors play a crucial role in how hormones affect the body, and the number of these receptors can influence hormone signaling. Endocrine-disrupting chemicals (EDCs) can interfere with this system by changing the levels of hormone receptors, how they are taken up by cells, and their breakdown, which can ultimately disrupt hormonal signaling; BPA can change the expression of thyroid hormone receptors, leading to abnormal signaling of hormones like T4 and T3. Additionally, thyroid hormones (THs) can influence androgen receptors (ARs), affecting the levels of hormones such as testosterone and dihydrotestosterone. Animal studies show that BPA also alters receptors for other hormones like estrogen, vasopressin, and oxytocin, impacting brain function. Furthermore, BPA has been found to reduce the breakdown of estrogen receptor β (Er β) in lab studies, potentially causing imbalanced hormonal responses.

Blocks hormone signaling



Mechanism

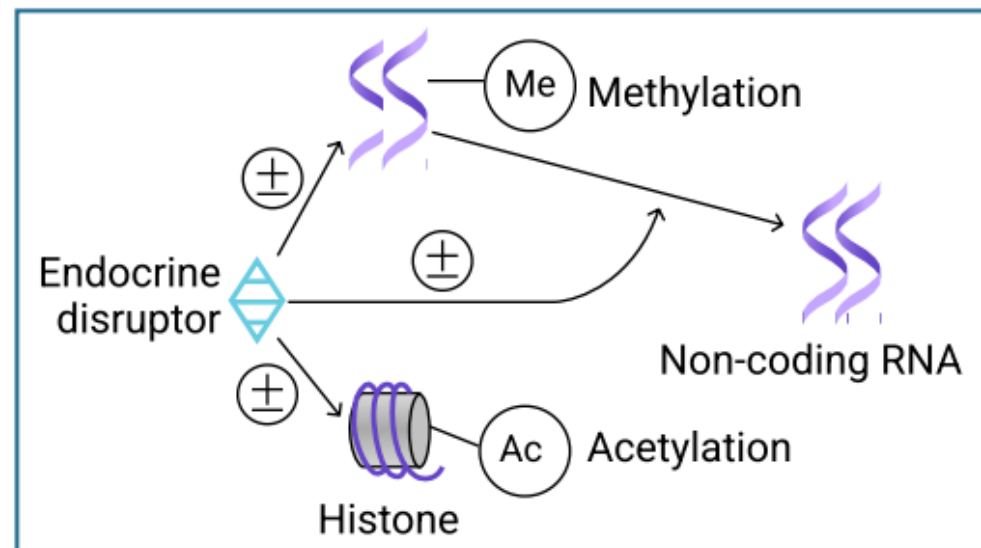
Hormones communicate with cells by binding to specific receptors, triggering a series of responses inside the cell. Endocrine-disrupting chemicals (EDCs) can interfere with these hormone signals and the reactions that follow. They affect different receptors, including those that respond to neurotransmitters and hormones; for instance, BPA disrupts calcium signaling triggered by low glucose levels in animal studies (ionotropic receptors). A key player in this process is the steroid receptor coactivator 1 (SRC1), which helps hormones attach to their receptors. BPA promotes the interaction of SRC1 with estrogen receptors (ER α and ER β) and the thyroid-hormone receptor β (SRC1 is essential for boosting the binding of hormones to these receptors). Research has shown that BPA increases the amount of SRC1 in animal studies and cell lines, including breast cancer cells. Additionally, BPA has been identified as an antagonist of thyroid hormone action, inhibiting T3 binding to its receptor and thus disrupting its signaling pathway. Additionally, BPA affects the androgen receptor's ability to bind testosterone and dihydrotestosterone, potentially altering their biological effects. This interference can lead to reduced efficacy of hormonal actions mediated by these receptors.

Endocrine Disruptors

Endocrine Disruptors

Epigenetic modification of hormone producing cells

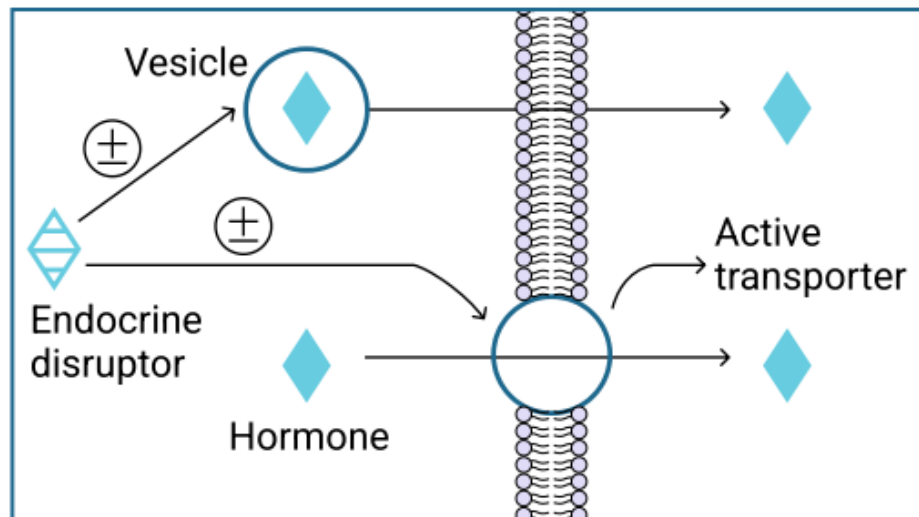
Mechanism



Hormones influence development by changing how genes are expressed through processes like DNA modifications, which include adding chemical tags to DNA (DNA methylation), changing the structure of proteins that help package DNA (histone modifications), and regulating non-coding RNA activity. Endocrine disrupting chemicals (EDCs) can interfere with these hormone functions by either blocking hormones from making these changes or by causing alterations in the epigenetic processes that affect hormone action, such as changing how hormone receptors work or disrupting gene transcription. EDCs can also influence non-coding RNA expression; Bisphenol A (BPA) can change microRNA levels in Sertoli cells, placental cells, and breast cancer cell lines. BPA exposure has been shown to induce the production of HOX antisense intergenic RNA (HOTAIR) in human breast cancer cells. The endocrine receptor-binding area of HOTAIR is modified when exposed to BPA, resulting in specific changes to histone proteins (like trimethylation of lysine 4 on histone 3, known as H3K4), which happens due to the action of particular enzymes. Furthermore, exposure to BPA during development can increase the trimethylation of genes, leading to long-lasting effects on gene expression. BPA exposure has been shown to alter microRNA levels that regulate genes responsive to thyroid hormones. This disruption can lead to long-lasting changes in how thyroid hormones exert their effects on target tissues.

Endocrine Disruptors

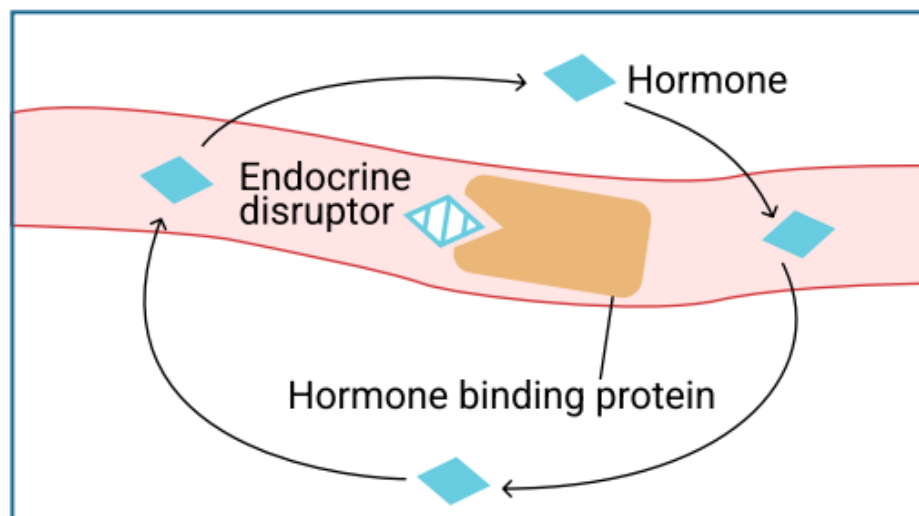
Hormone transport



Mechanism

Steroid hormones, like estrogen and androgens, can easily move across cell membranes due to their lipophilic nature. Other hormones like thyroid hormones are selectively transported in and out of the cell. Both the free and selective movement of hormones in and out of the cells are disrupted by endocrine-disrupting chemicals or EDCs; EDCs like BPA can disrupt the transport of T4 and T3 into cells by interfering with specific membrane transporters. This disruption affects the availability of these hormones at the cellular level and can lead to altered metabolic functions. BPA also disrupts the transport of calcium into pancreatic β -cells in animal models which reduces insulin secretion.

Affects hormone distribution & levels in circulation



Mechanism

Hormones are constantly circulating in the blood at low concentrations. They are either free (not bound to any protein) or bound to serum binding proteins. Endocrine disrupting chemicals or EDCs interfere with distribution and circulating of hormones by displacing the hormones from serum binding proteins thereby affecting their bioavailability and delivery to tissues; Studies have shown that BPA exposure can reduce circulating levels of testosterone and dihydrotestosterone in both humans and animal models. Additionally, BPA has been linked to changes in circulating levels of thyroid hormones due to its interference with binding proteins that transport these hormones in the bloodstream

Endocrine Disruptors

Endocrine Disruptors					
Test Name	Current	Previous	Result		Reference
			75th	95th	
Perchlorate (PERC)^ (ug/g)	1.42		4.89	10.7	≤10.7
Monoethyl Phthalate (MEP)^ (ug/g)	1.33		94.2	541	≤541
Mono-2-ethylhexyl phthalate (MEHP)^ (ug/g)	0.43		2.73	8.47	≤8.47
Mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)^ (ug/g)	1.34		14.1	37.7	≤37.7
Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)^ (ug/g)	4.67		8.99	23.4	≤23.4
Methylparaben^ (ug/g)	1.74		180	653	≤653
Propylparaben^ (ug/g)	0.02		36.7	222	≤222
Butylparaben^ (ug/g)	0.04		0.25	4.39	≤4.39
Ethylparaben ^ (ug/g)	0.02		5.41	99.3	≤99.3

Endocrine Disruptors

SUPPLEMENT SUGGESTIONS

Green Tea(2 cups/day): The polyphenols in green tea, particularly epigallocatechin gallate (EGCG), exhibit antioxidant properties that scavenge reactive oxygen species (ROS) and reduce oxidative stress caused by BPA, thus protecting cellular integrity.

Soy-rich Diet(25 g/day): Isoflavones found in soy, such as genistein, can modulate estrogenic activity and reduce BPA's endocrine-disrupting effects by competing with BPA for binding sites on estrogen receptors, mitigating its toxic impact on hormonal balance.

Grape Juice (Gb)(150 ml/day): Red grape juice contains resveratrol and other polyphenols that counteract BPA-induced apoptosis and oxidative stress by modulating apoptotic pathways and enhancing mitochondrial function, thereby promoting cell survival.

KRG (Korean Red Ginseng)(1 g/day): KRG is known to enhance antioxidant defenses and reduce inflammation through its ginsenosides, which can mitigate oxidative stress and protect against BPA-induced cellular damage in various tissues.

Ginseng(1 g/day): Ginseng extracts possess adaptogenic properties that help stabilize cellular responses to stressors like BPA by enhancing antioxidant enzyme activity and reducing inflammation, thereby protecting cells from oxidative damage.

Resveratrol (RSV)(100 mg/day): Resveratrol has been shown to exert protective effects against BPA toxicity by activating sirtuins, which play a role in cellular stress resistance and mitochondrial function, effectively reducing oxidative damage.

Luteolin(100 mg/day): This flavonoid exhibits strong antioxidant properties that can scavenge ROS generated by BPA exposure, thereby reducing oxidative stress and inflammation in affected tissues.

Lycopene(6 mg/day): Lycopene acts as an antioxidant that can inhibit lipid peroxidation and protect against BPA-induced cellular damage by neutralizing free radicals and modulating inflammatory responses.

AS IV (Astragalus Saponin IV)(100 mg/day): AS IV has been shown to mitigate BPA toxicity through its ability to enhance antioxidant enzyme activities and reduce inflammation, thus supporting cellular health under oxidative stress conditions.

Genistein(40 mg/day): As a phytoestrogen, genistein can compete with BPA for estrogen receptors, reducing the endocrine-disrupting effects of BPA while also providing antioxidant benefits that protect against oxidative stress.

Curcumin(500 mg/day): Curcumin exhibits potent anti-inflammatory and antioxidant properties that can counteract the harmful effects of BPA by reducing oxidative stress and modulating signaling pathways involved in inflammation.

Centella asiatica (600 mg/day): Centella asiatica enhances wound healing and skin health by promoting collagen synthesis and angiogenesis, primarily through its active compounds like asiaticoside, which stimulate fibroblast proliferation and improve blood circulation.

Vitamin D3(600 IU/day): Vitamin D3 supports calcium absorption and bone health while modulating immune function. It also plays a role in reducing inflammation and may improve mood and cognitive function through its effects on neurotransmitter synthesis.



What other suggestions for detoxification can you give this patient?



Support your Detox with food:

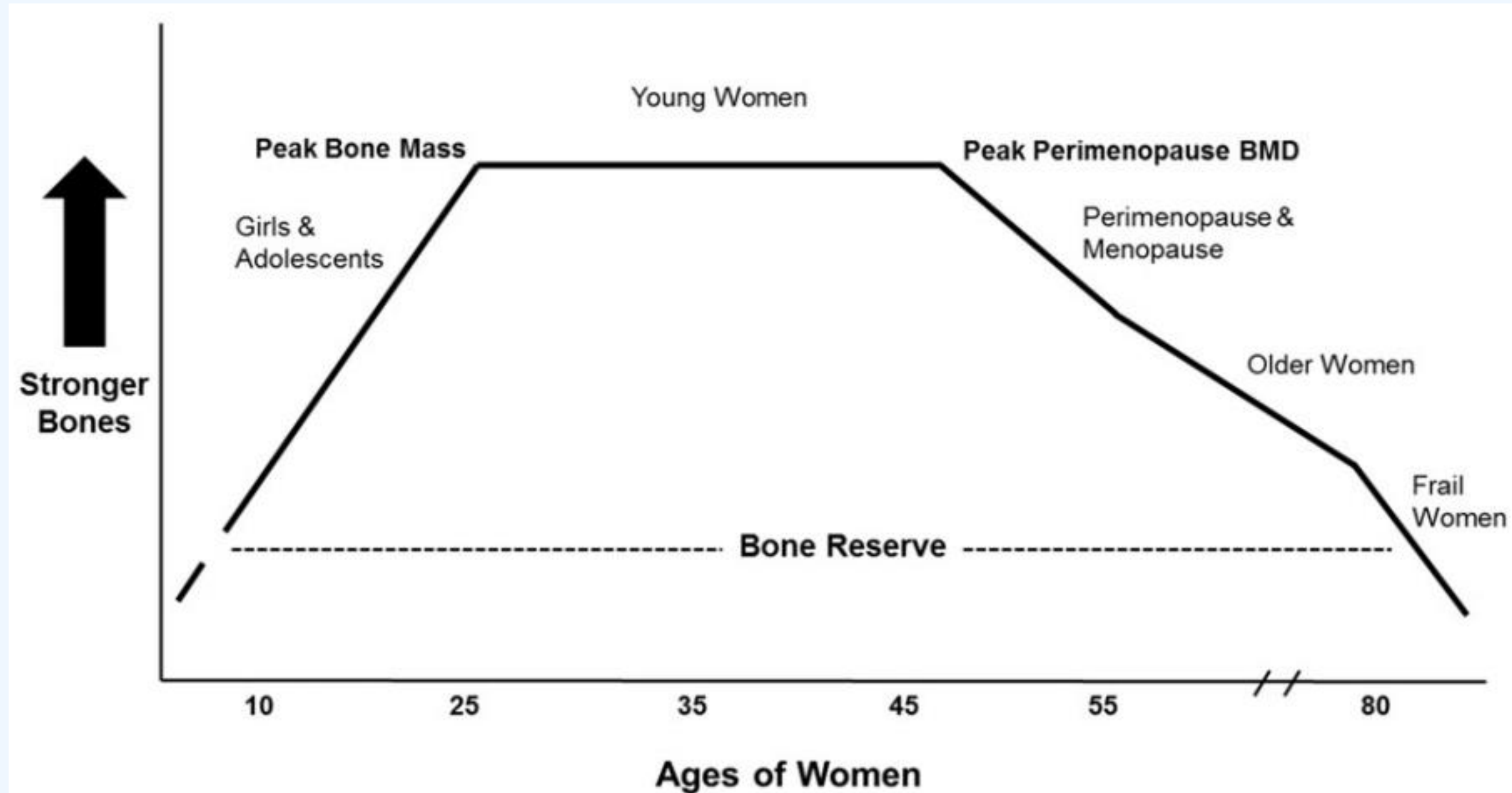
- Increase fiber
- Plenty of lean proteins
- Limit carbohydrates
- Eat the rainbow and eat diverse fruits/veg
- Limit alcohol
- Increase spices
- Add vitamins and minerals when needed
 - This can be assessed
- Support your anti-oxidants and mitochondria

Food preparation:

- Optimize nutrient density:
 - Raw or minimal steam
- Optimizing food preparation to decrease toxins:
 - Cookware
 - Slow, low moist heat
 - Use water rather than oil



Phases of Bone Mass



J. C. Prior (2018) Progesterone for the prevention and treatment of osteoporosis in women, *Climacteric*, 21:4, 366-374

Biomarkers for Bone Remodeling

Formation

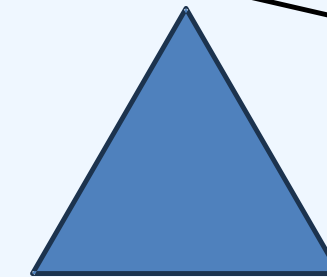
- BONE SPECIFIC ALKALINE PHOSPHATASE ISOENZYME (BSAP)
 - Serum marker for osteoblast derived bone formation
 - There is a circadian rhythm
- PROCOLLAGEN TYPE 1 TERMINAL PROPEPTIDE (P1NP)
 - Marker of osteoblast activity- Bone growth
- OSTEOCALCIN

Resorption

- SERUM AND URINE N-TELOPEPTIDE (NTx)
 - Biomarker for osteoclast derived bone resorption
- C-TELOPEPTIDE (CTX)

P1NP

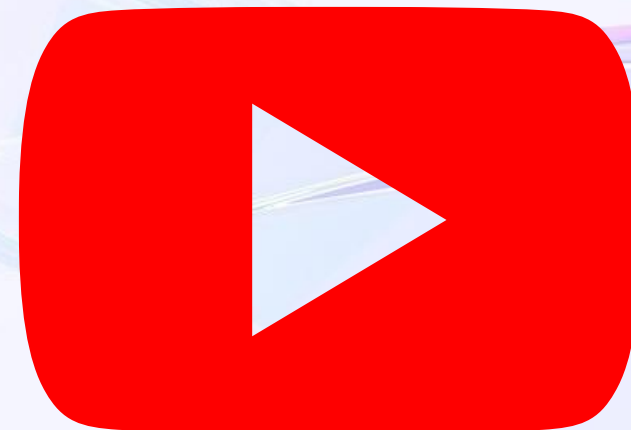
CTX



Thank You!



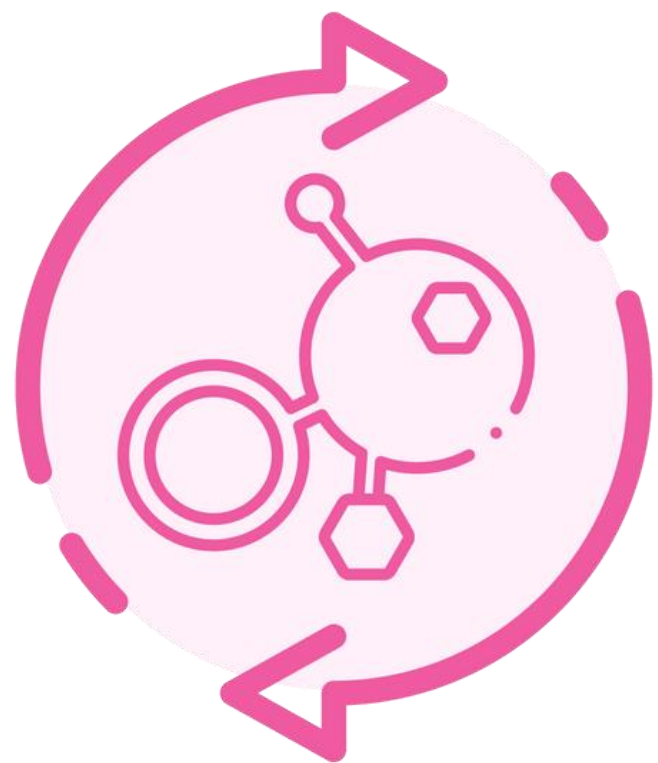
@Hormonegurumd



TaraScottMD



@hormoneguru



Hormone Optimization

Elevating Lifespan
Through Endocrine
Balance



Session 3

**Dr. Carrie
Jones, ND,
FABNE, MPH,
MSCP**

Phase 1 Estrogen Metabolism

Does Everyone Get DIM?





Meet Your Speaker

Dr. Carrie Jones
ND, FABNE, MPH, MSCP

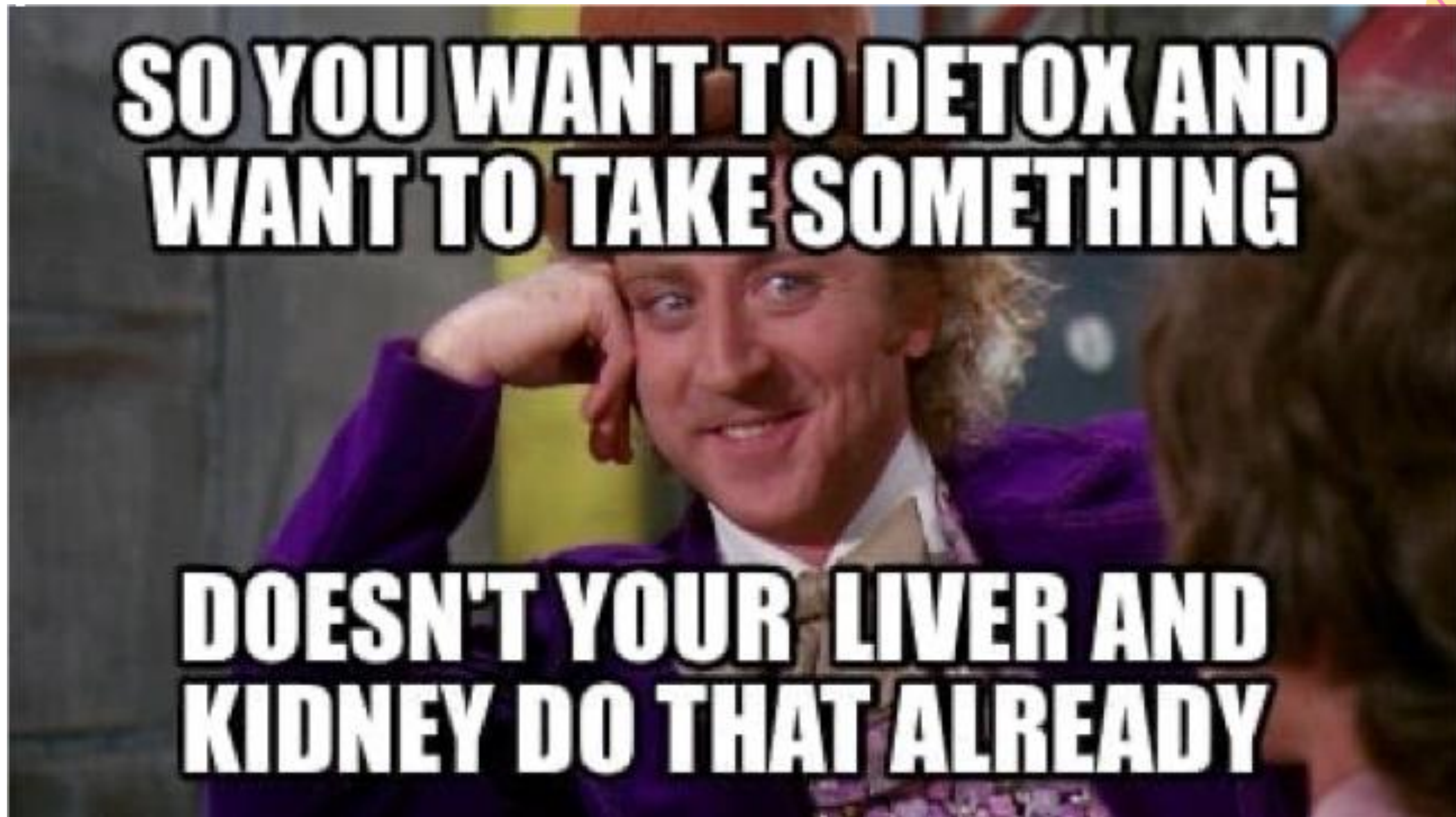
A internationally known women's health & hormone expert with over 20 years of experience and over 12 years in the labs space.

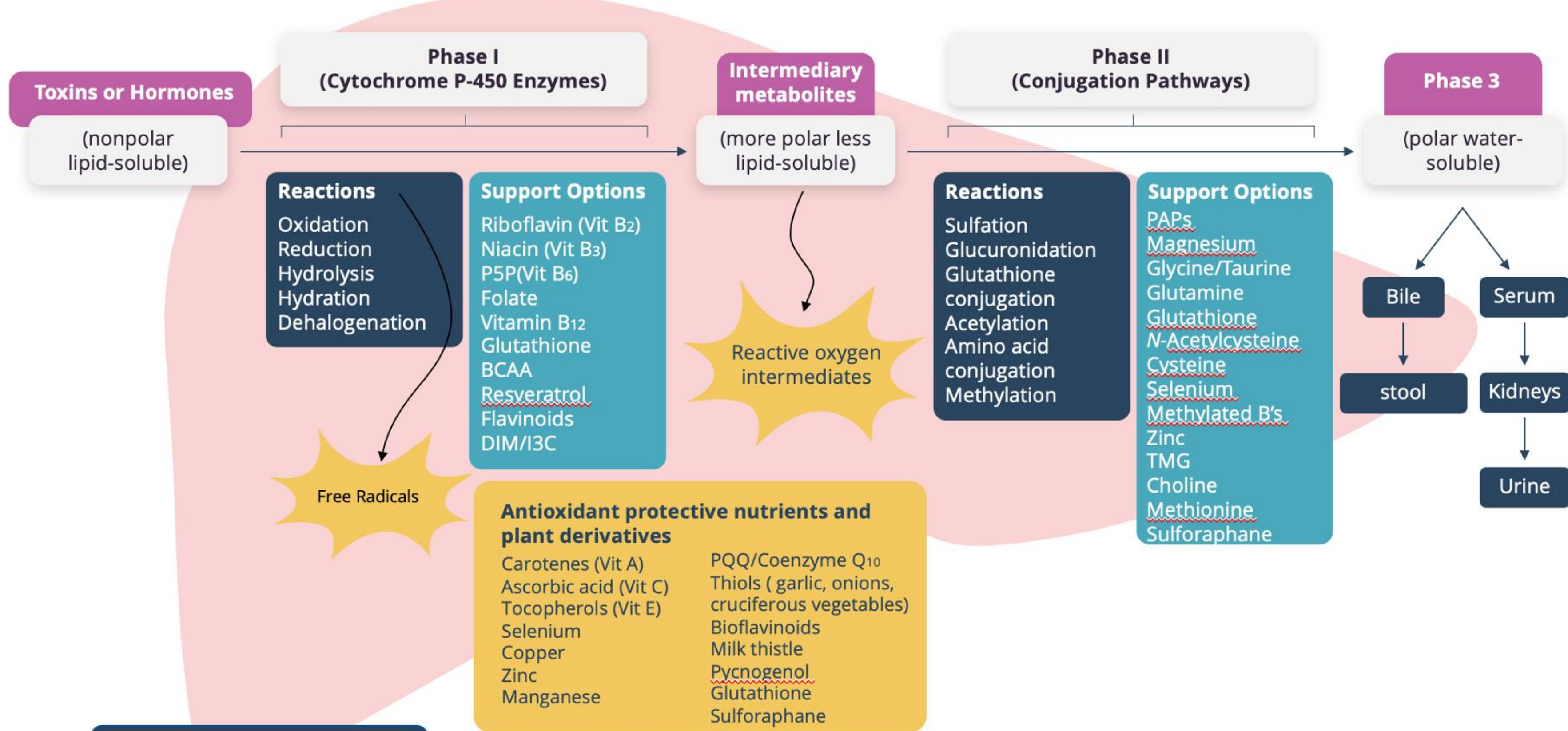
Estrogen Detoxification

Also known as metabolism.

It is done In 2 or 3 phases.

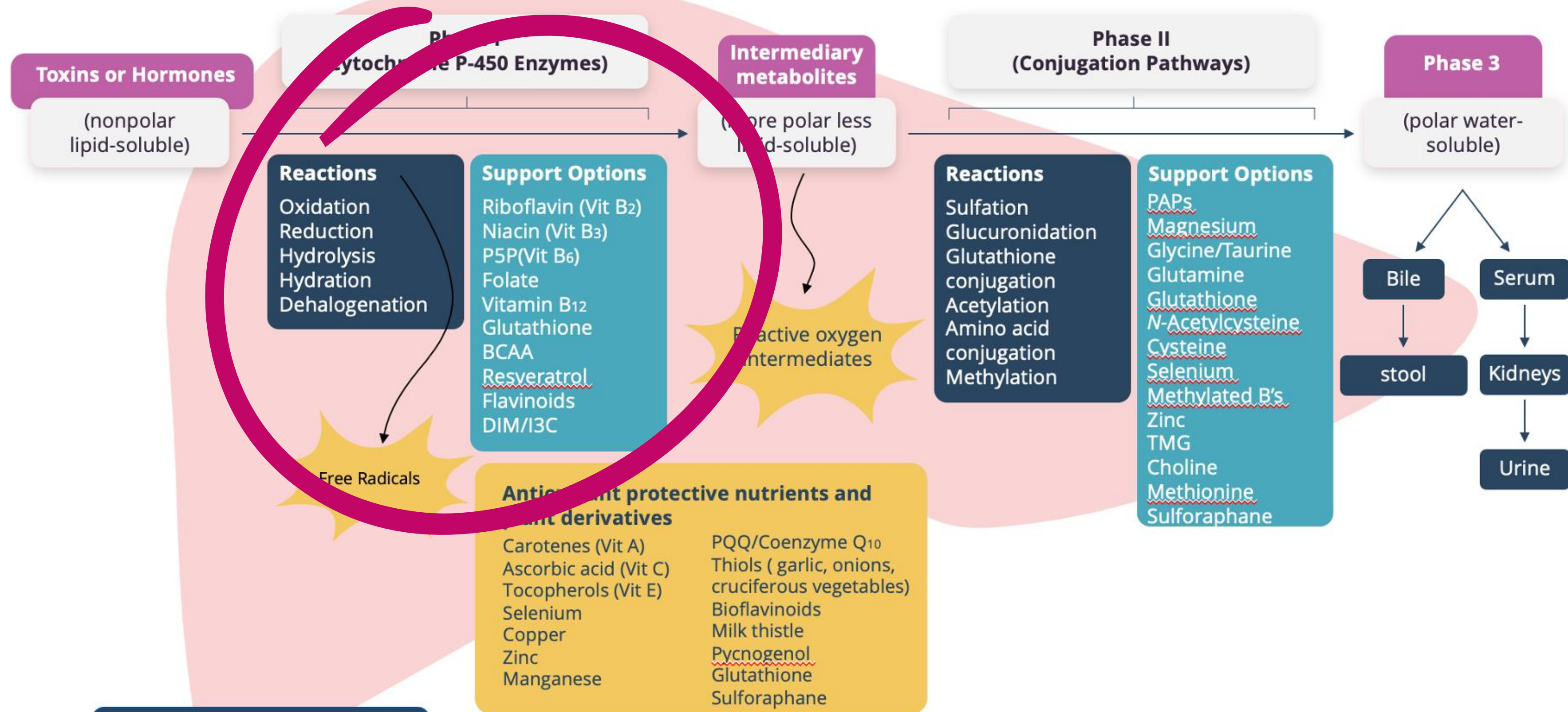
- **Phase 1** = hydroxylation
- **Phase 2** = methylation and/or sulfation or glucuronidation
- **Phase 3** = Excretion





Based on Liska DJ. The detoxification enzyme systems. Altern Med Rev. 1998; 3(3):187-98.

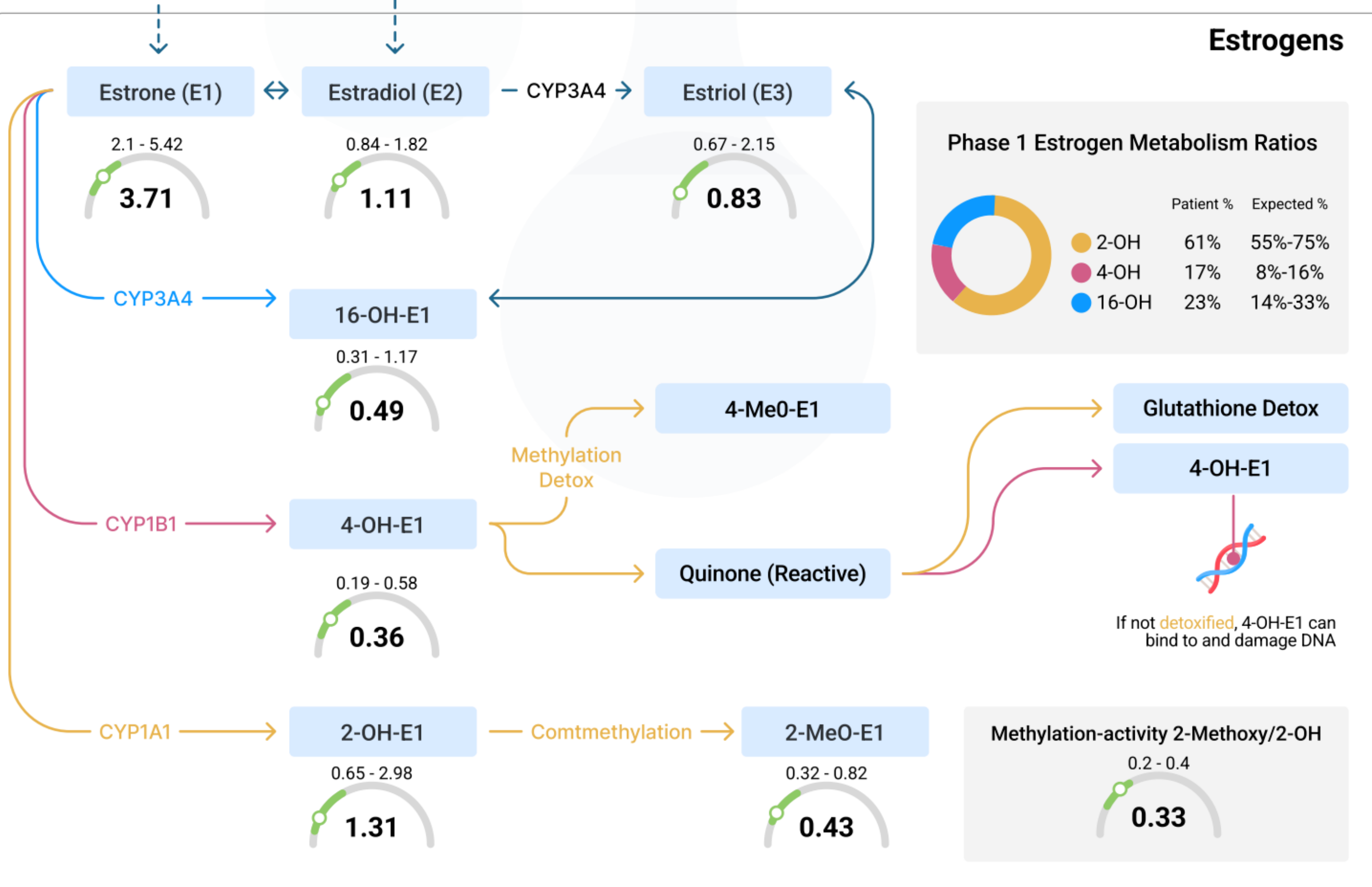
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Based on Liska DJ. The detoxification enzyme systems. Altern Med Rev. 1998; 3(3):187-98.

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Estrogens



Estrogens

Phase 1 Estrogen Metabolism Ratios



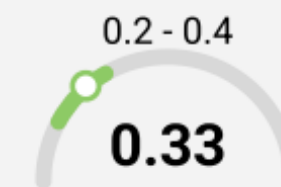
Glutathione Detox

4-OH-E1



If not **detoxified**, 4-OH-E1 can bind to and damage DNA

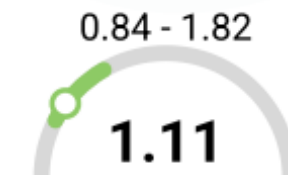
Methylation-activity 2-Methoxy/2-OH



Estriol (E3)



Estradiol (E2)



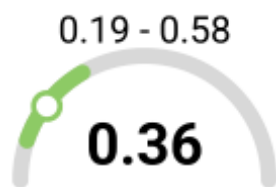
Estrone (E1)



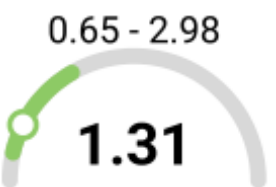
16-OH-E1



4-OH-E1



2-OH-E1



4-MeO-E1

Quinone (Reactive)

2-MeO-E1



Methylation
Detox

Complete Methylation

CYP3A4

CYP1B1

CYP1A1

CYP3A4

Methylation
Detox

Complete Methylation

If not **detoxified**, 4-OH-E1 can bind to and damage DNA

Estrogens

Phase 1 Estrogen Metabolism Ratios

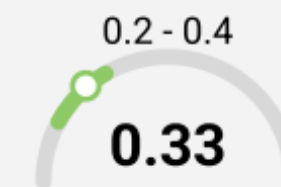


Glutathione Detox

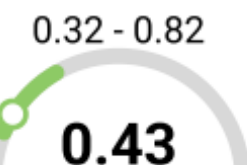
4-OH-E1

If not detoxified, 4-OH-E1 can bind to and damage DNA

Methylation-activity 2-Methoxy/2-OH



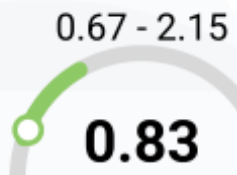
2-MeO-E1



Quinone (Reactive)

4-MeO-E1

Estriol (E3)



Estradiol (E2)



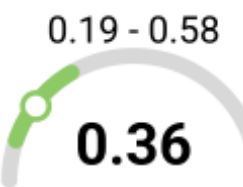
Estrone (E1)



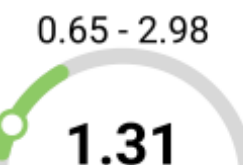
16-OH-E1



4-OH-E1



2-OH-E1



Methylation
Detox

Complete Methylation

CYP3A4

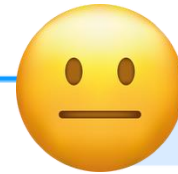
CYP1B1

CYP1A1

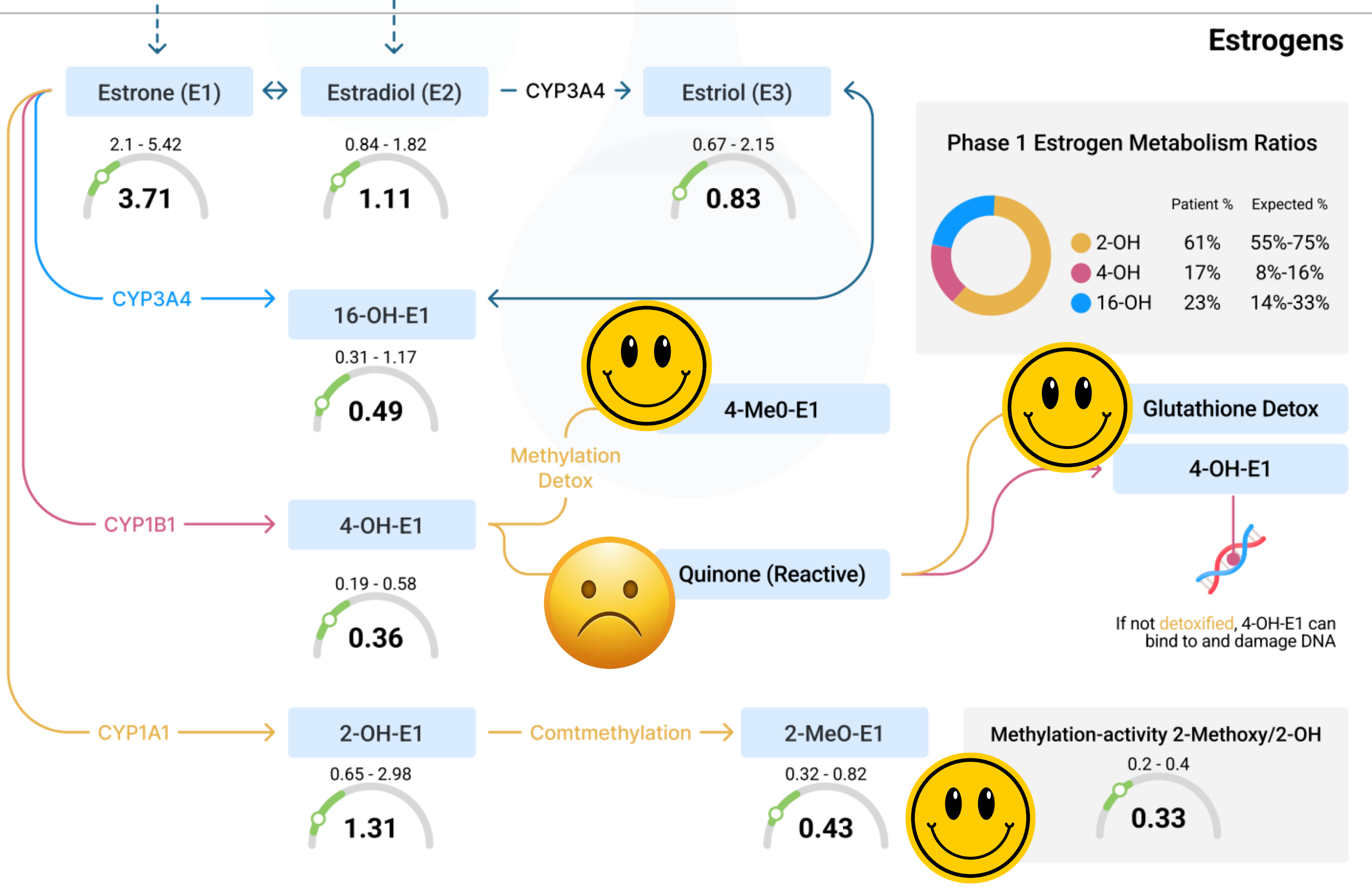
↔

→ CYP3A4 →

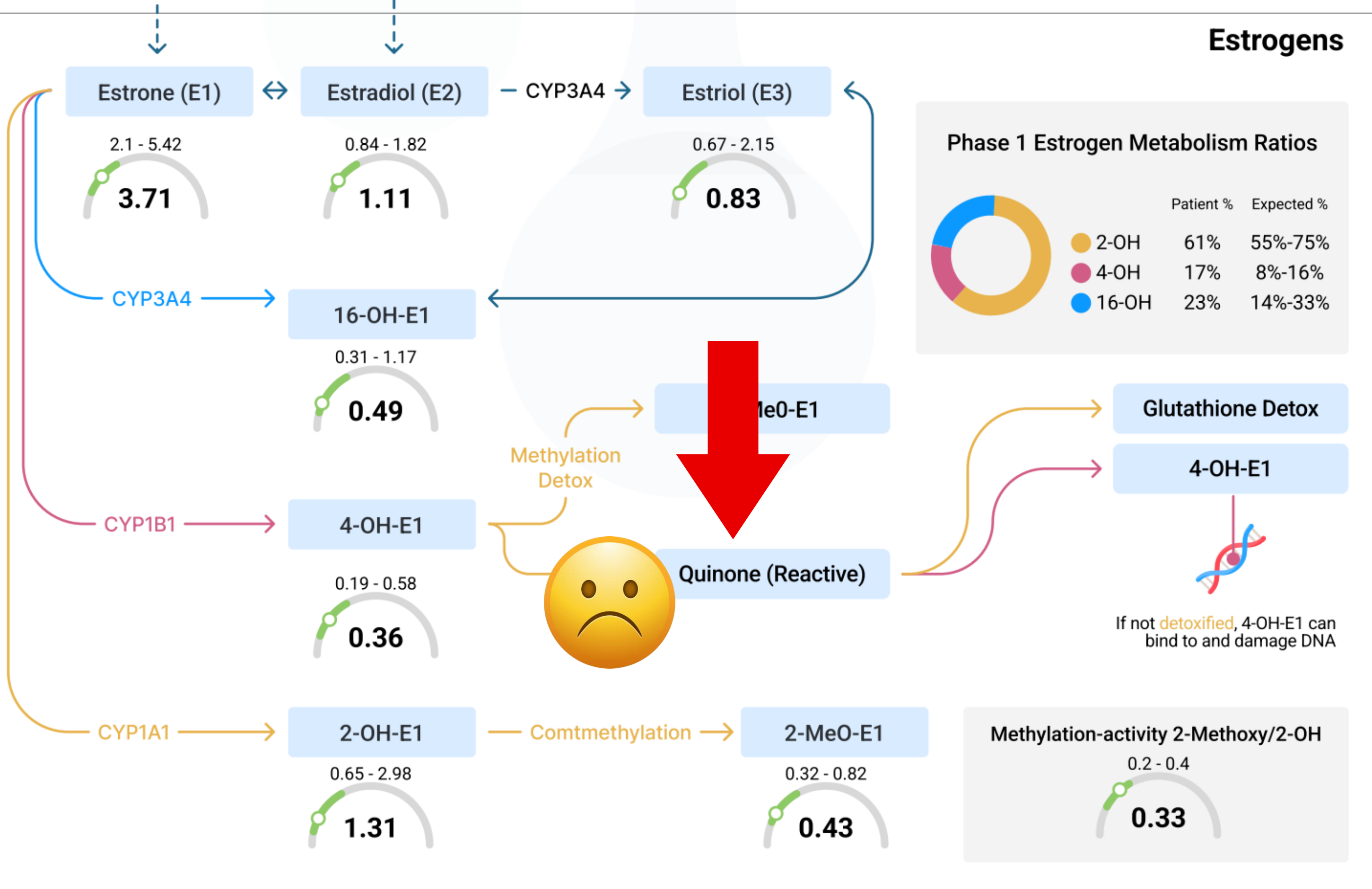
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Estrogens



Estrogens



“Quinones are Michael acceptors, and cellular damage can occur through alkylation of crucial cellular proteins and/or DNA. Alternatively, quinones are highly redox active molecules which can redox cycle with their semiquinone radicals, leading to formation of reactive oxygen species (ROS), including superoxide, hydrogen peroxide, and ultimately the hydroxyl radical. Production of ROS can cause severe oxidative stress within cells through the formation of oxidized cellular macromolecules, including lipids, proteins, and DNA.”

Review > Chem Res Toxicol. 2000 Mar;13(3):135-60. doi: 10.1021/tx9902082.

Role of quinones in toxicology

J L Bolton ¹, M A Trush, T M Penning, G Dryhurst, T J Monks

Affiliations + expand

PMID: 10725110 DOI: [10.1021/tx9902082](https://doi.org/10.1021/tx9902082)

Abstract

Quinones represent a class of toxicological intermediates which can create a variety of hazardous effects in vivo, including acute cytotoxicity, immunotoxicity, and carcinogenesis. The mechanisms by which quinones cause these effects can be quite complex. Quinones are Michael acceptors, and cellular damage can occur through alkylation of crucial cellular proteins and/or DNA. Alternatively, quinones are highly redox active molecules which can redox cycle with their semiquinone radicals, leading to formation of reactive oxygen species (ROS), including superoxide, hydrogen peroxide, and ultimately the hydroxyl radical. Production of ROS can cause severe oxidative stress within cells through the formation of oxidized cellular macromolecules, including lipids, proteins, and DNA.

Formation of oxidatively damaged bases such as 8-oxodeoxyguanosine has been associated with aging and carcinogenesis. Furthermore, ROS can activate a number of signaling pathways, including protein kinase C and RAS. This review explores the varied cytotoxic effects of quinones using specific examples, including quinones produced from benzene, polycyclic aromatic hydrocarbons, estrogens, and catecholamines. The evidence strongly suggests that the numerous mechanisms of quinone toxicity (i.e., alkylation vs oxidative stress) can be correlated with the known pathology of the parent compound(s).

Estrogens

Phase 1 Estrogen Metabolism Ratios



Today's Focus Phase 1

Glutathione Detox

4-OH-E1

If not detoxified, 4-OH-E1 can bind to and damage DNA

Methylation-activity 2-Methoxy/2-OH

0.2 - 0.4
0.33

2-MeO-E1

0.32 - 0.82
0.43

2-OH-E1

0.65 - 2.98
1.31

0.19 - 0.58
0.36

4-OH-E1

0.31 - 1.17
0.49

16-OH-E1

0.84 - 1.82
1.11

Estrone (E1)

2.1 - 5.42
3.71

Estradiol (E2)

Estriol (E3)

0.67 - 2.15
0.83

Com Methylation

CYP1A1

CYP1B1

CYP3A4

CYP3A4

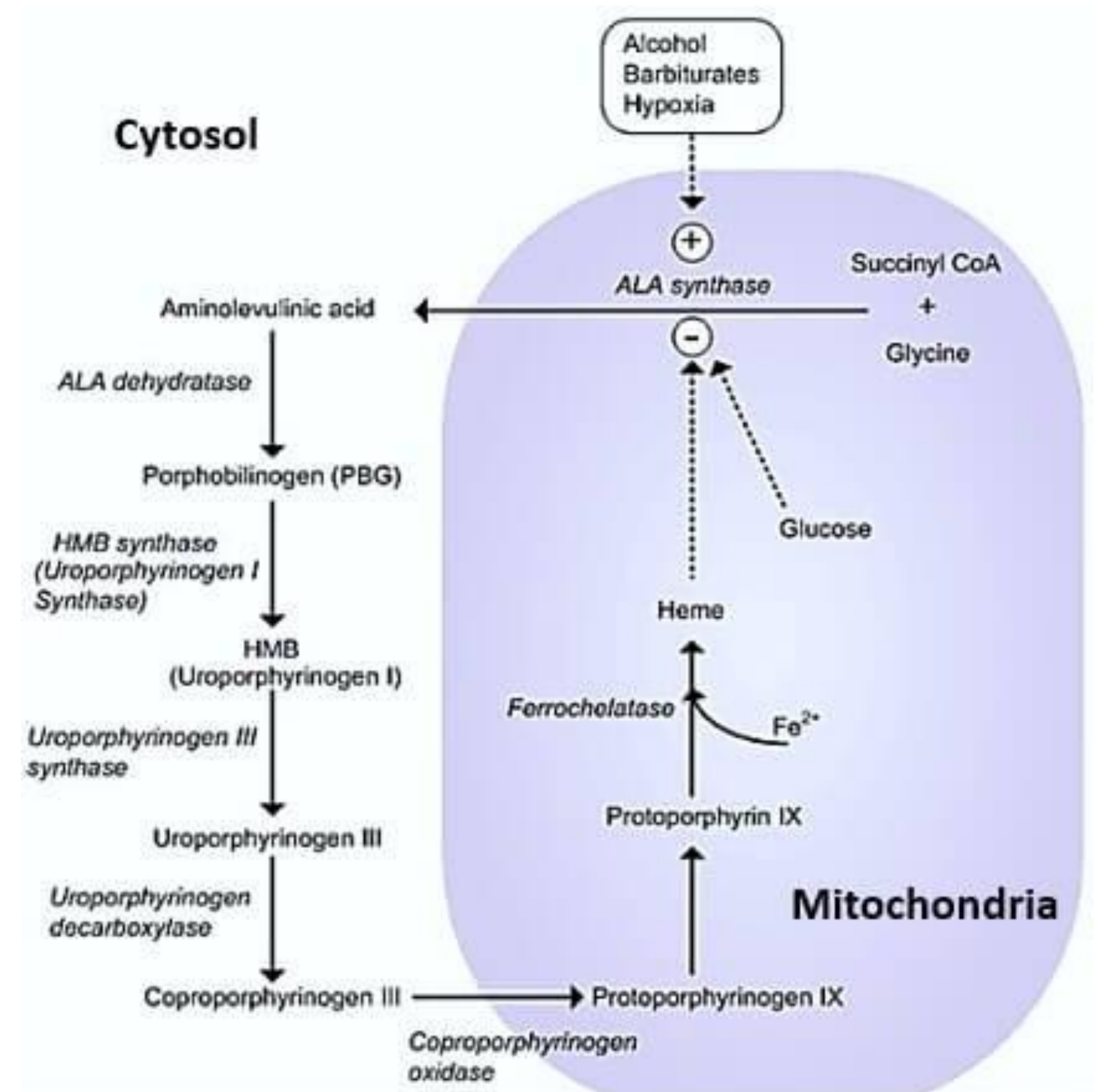
Starts at the Liver

- Be aware of all **medications**
- Be aware of their **alcohol** and/or **drug** consumption
- Educate on **toxicant exposure**/minimization
- **Liver** function tests
- Evaluate for **fatty liver** – estimated 24% of adults have it (NIH)



CYP450 Enzymes are HEME Dependent

- First step = succinyl CoA + glycine + P5P (B6)
- It also requires ferrous Iron



1Ogun AS, Valentine M. Biochemistry, Heme Synthesis. Nih.gov. Published January 31, 2019. <https://www.ncbi.nlm.nih.gov/books/NBK537329/>

1Ogun AS, Valentine M. Biochemistry, Heme Synthesis. Nih.gov. Published January 31, 2019. <https://www.ncbi.nlm.nih.gov/books/NBK537329/>

Estrogens

Phase 1 Estrogen Metabolism Ratios



**Specific for
these enzymes**

Glutathione Detox

4-OH-E1

If not **detoxified**, 4-OH-E1 can
bind to and damage DNA

Methylation-activity 2-Methoxy/2-OH

0.2 - 0.4
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Com Methylation

0.65 - 2.98
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2-OH-E1

CYP1A1

0.19 - 0.58
0.36

4-OH-E1

CYP1B1

0.31 - 1.17
0.49

16-OH-E1

CYP3A4

Estriol (E3)

0.67 - 2.15
0.83

— CYP3A4 →

Estradiol (E2)

0.84 - 1.82
1.11

Estrone (E1)

2.1 - 5.42
3.71

↔

DIM (3,3'-Diindolylmethane)

I3C (Indole-3-Carbinol)

I3C = Formed from the cruciferous family (Glucobrassicin + myrosinase) like broccoli and Brussels sprouts that are chopped or chewed

Needs stomach acid to then convert into 15+ compounds

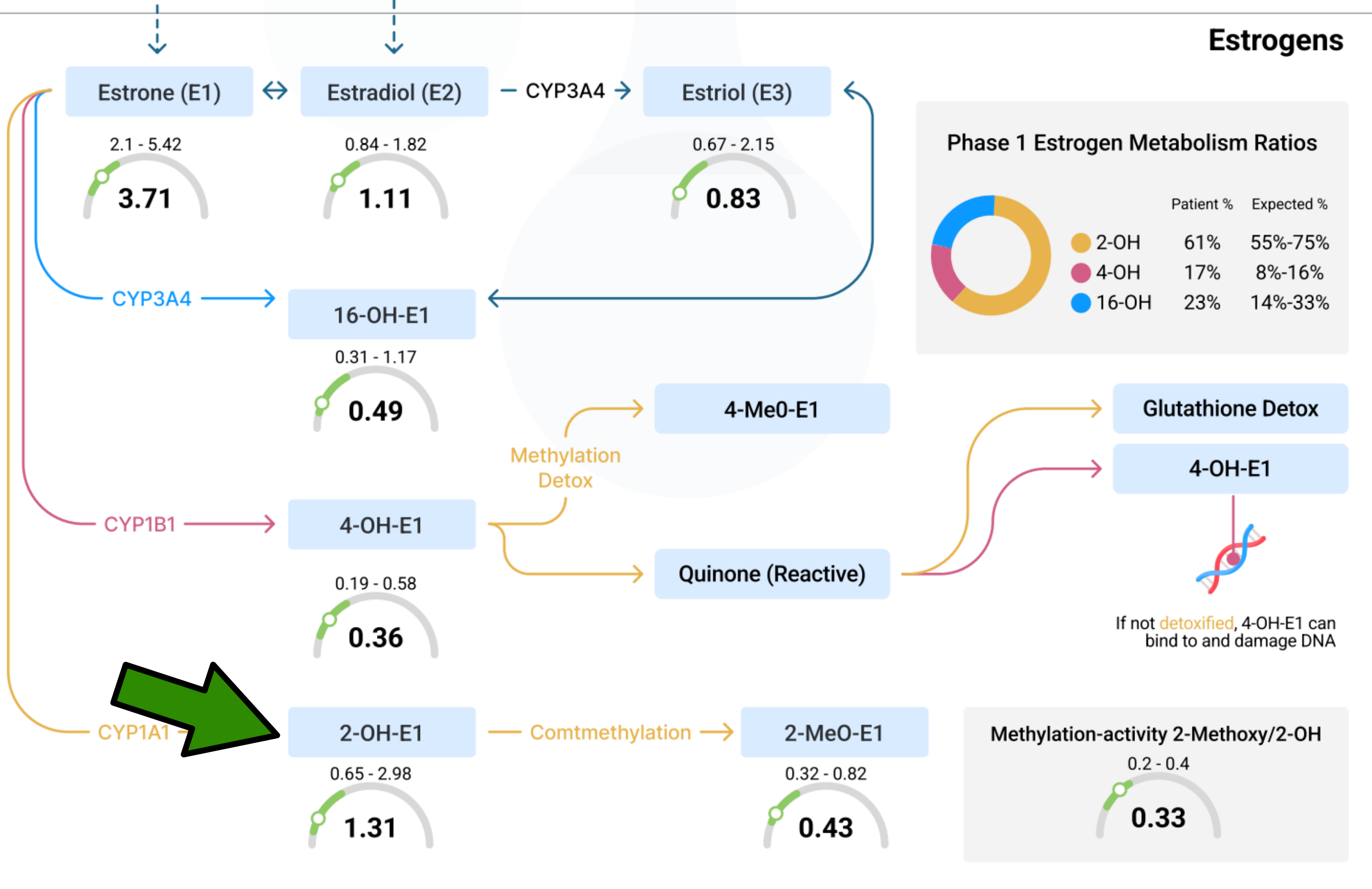
➡ A primary compound is **DIM**

How Does DIM Work Primarily?

- Selective modulator and mild agonist of the Aryl Hydrocarbon Receptor
- This mostly activates **CYP1A1/2-OH** (and less CYP1B1)

- Rajoria S, Suriano R, Parmar PS, et al. 3,3'-Diindolylmethane Modulates Estrogen Metabolism in Patients with Thyroid Proliferative Disease: A Pilot Study. *Thyroid*. 2011;21(3):299-304. doi:<https://doi.org/10.1089/thy.2010.0245>
- Yin XF, Chen J, Mao W, Wang YH, Chen MH. A selective aryl hydrocarbon receptor modulator 3,3'-Diindolylmethane inhibits gastric cancer cell growth. *Journal of Experimental & Clinical Cancer Research*. 2012;31(1):46. doi:<https://doi.org/10.1186/1756-9966-31-46>

Estrogens



Can You Eat Enough?

“On average, 100 g of cruciferous vegetables contains up to 30 mg of glucobrassicin, which is estimated to convert to approximately 2 mg of DIM. However, the variation in DIM content between different cruciferous vegetables is considerable, with differences ranging from 5- to 8-fold. To achieve a biologically relevant exposure, it is suggested that intake would need to be upwards of **600 g/d** and sustained for several years to achieve an anticancer benefit. An intake this high is difficult to attain or maintain through diet alone.”





That's 6-7 cups/day

Thomson CA, Ho E, Strom MB. Chemopreventive properties of 3,3'-diindolylmethane in breast cancer: evidence from experimental and human studies. *Nutrition Reviews*. 2016;74(7):432-443.
doi:10.1093/nutrit/nuw010.

DIM Caveats

- Be careful In low estrogen states
- CYP1A metabolizes more than estrogen
- If CYP1A is upregulated and phase 2/phase 3 are not functioning well = **at more risk** for damage due to the **phase 1 metabolites of toxicants** in circulation

Newman MS, Smeaton J. The impact of 3,3'-diindolylmethane on estradiol and estrogen metabolism in postmenopausal women using a transdermal estradiol patch. Menopause. Published online April 29, 2025. doi:<https://doi.org/10.1097/gme.0000000000002542>

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Quercetin for Phase 1

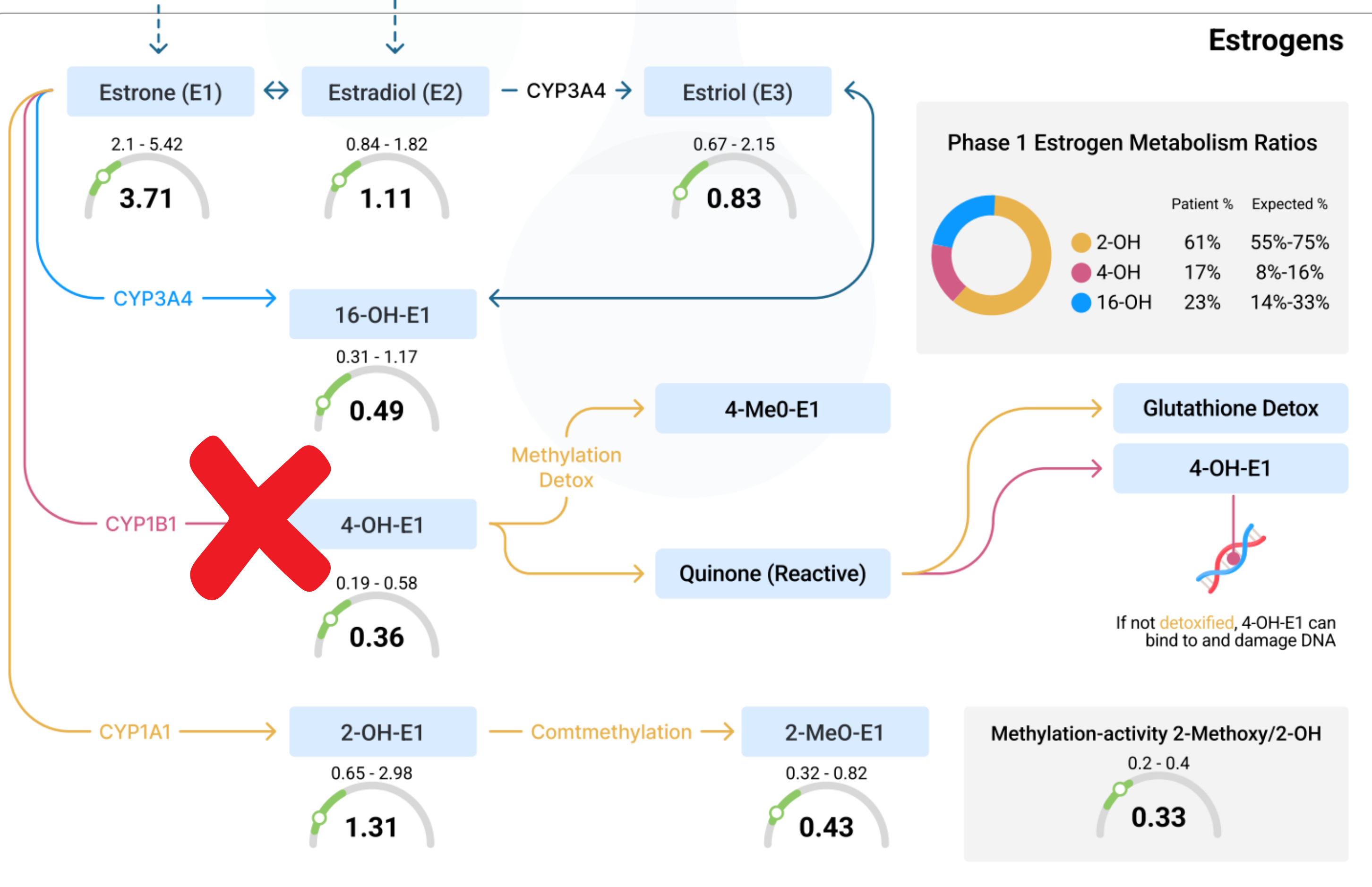
Quercetin

- **Potent CYP1B1 inhibitor** = ↓ 4-OH
- Less effective inhibitor of CYP1A1 and 1A2
- High oral bioavailability of flavonoids (20%)
- Very helpful at many other things including reducing histamine and pro-inflammatory cytokines
- Note: it can slow down COMT (phase 2)
- Typical dose: 250–1000mg/day

• Androutsopoulos VP, Papakyriakou A, Vourloumis D, Spandidos DA. Comparative CYP1A1 and CYP1B1 substrate and inhibitor profile of dietary flavonoids. *Bioorganic & Medicinal Chemistry*. 2011; 19(9):2842–2849.

• Martinez-Perez C, Ward C, Cook G, et al. Novel flavonoids as anti-cancer agents: mechanisms of action and promise for their potential application in breast cancer. *2014; 42(4):1017–1023.*

Estrogens





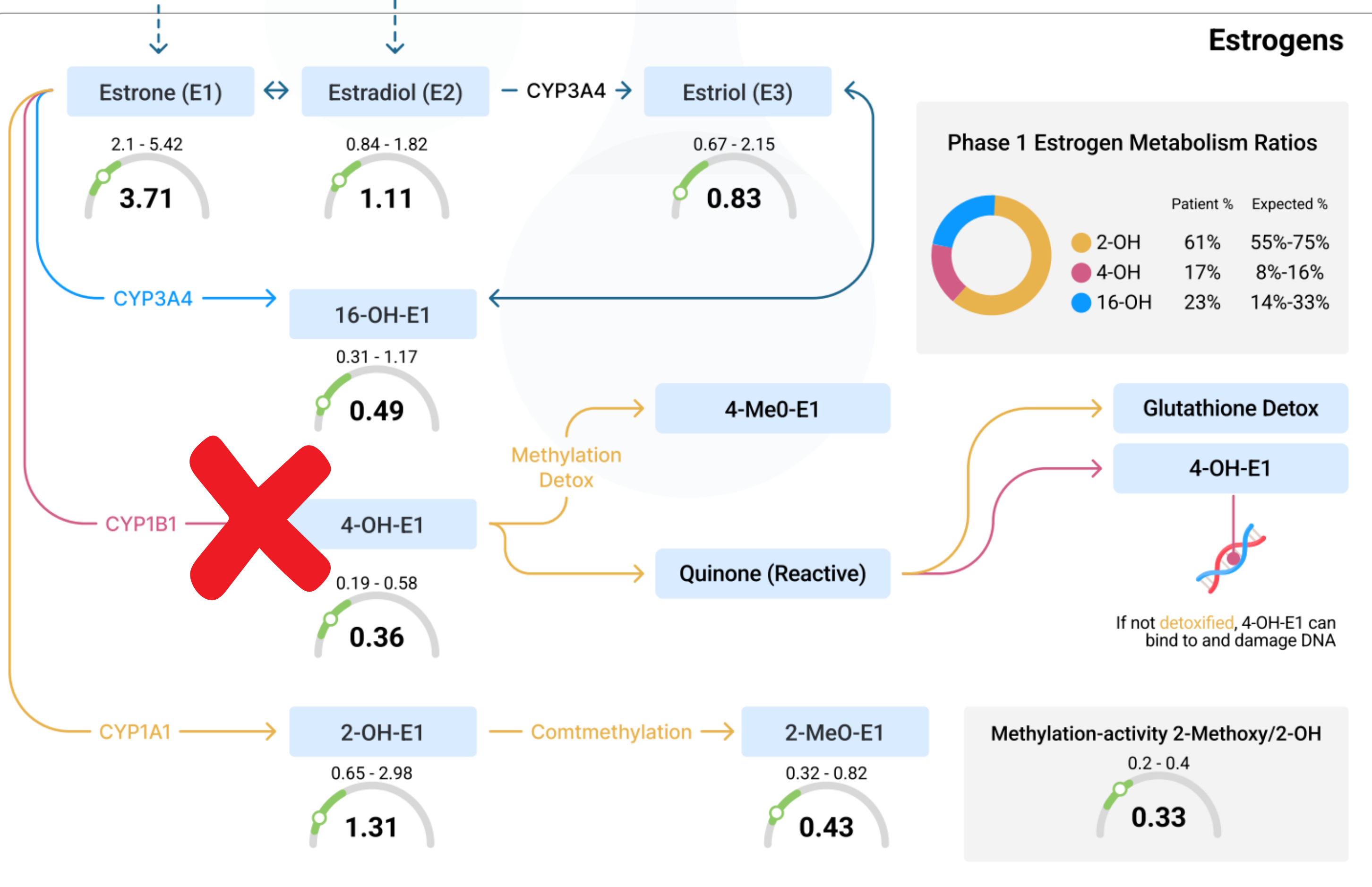
Resveratrol for Phase 1

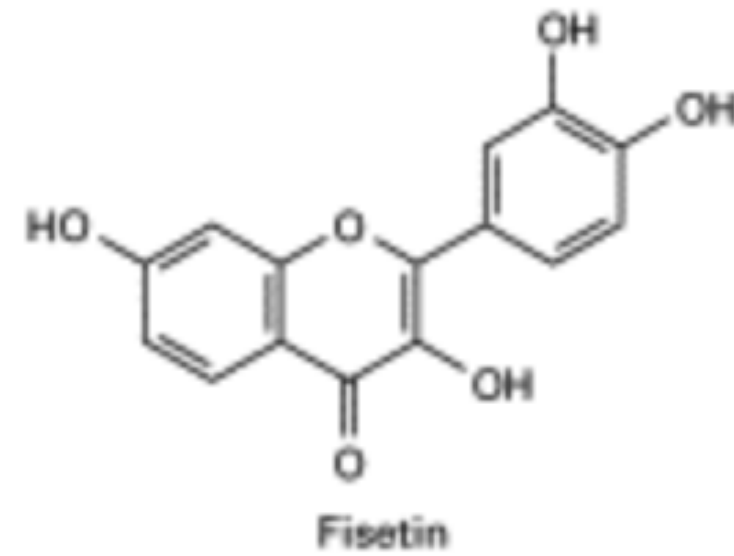
Resveratrol

- It's an **antioxidant**
- **Potent CYP1B1 inhibitor** = ↓ 4-OH
- "Its inhibitory ability for CYP1B1 is over 50-fold greater than against CYP1A1 and 500-fold higher than for CYP1A2" (Li, et al, 2017).
- Note: All from invitro and animal studies

Li F, Zhu W, Gonzalez FJ. Potential role of CYP1B1 in the development and treatment of metabolic diseases Pharmacology & Therapeutics. 2017; 178:18-30.

Estrogens





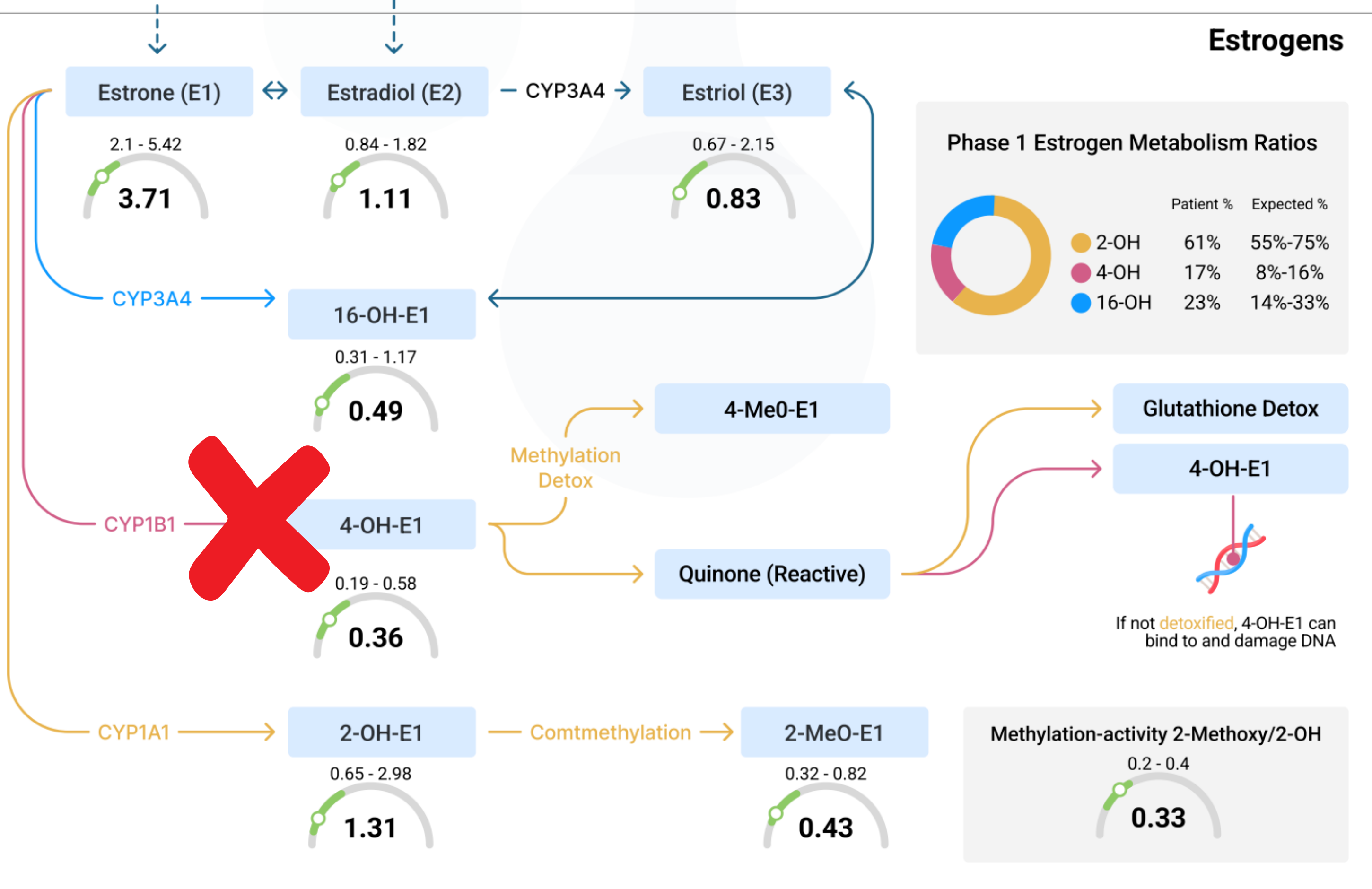
Fisetin for Phase 1

Fisetin

- **Potential CYP1B1 inhibitor** = ↓ 4-OH
- Common ingredient In autophagy, senolytic, longevity blends
- Cell studies

Meng X, Sun H, Yang L, Yin R, Qi L. A hydroxylated flavonol, fisetin inhibits the formation of a carcinogenic estrogen metabolite. Steroids. 2017;119:53-56.
doi:<https://doi.org/10.1016/j.steroids.2017.01.002>

Estrogens





Oroxylin A for Phase 1

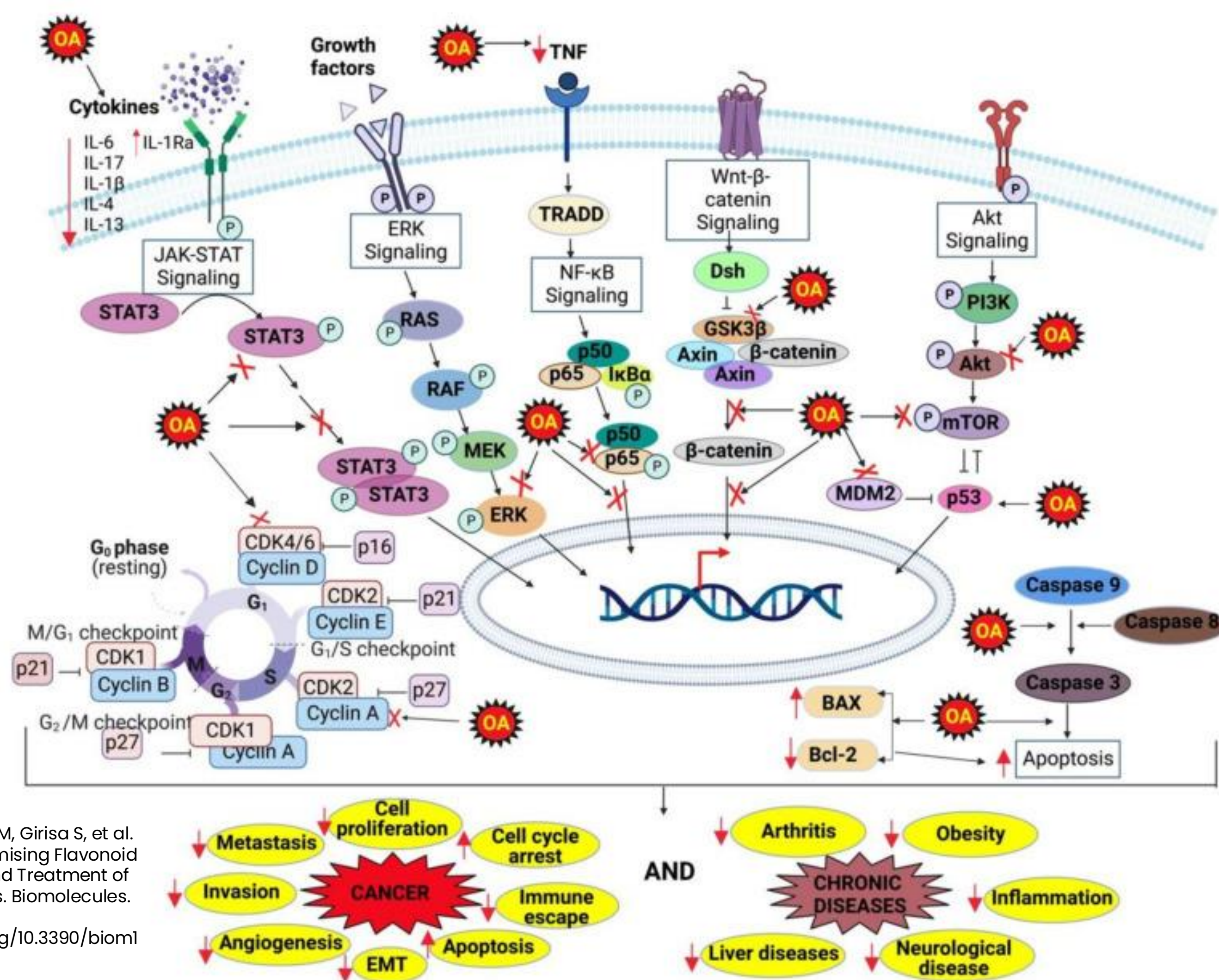
Oroxylin A

- **Flavonoid** in root bark of Scutellaria
- **CYP1B1 inhibitor** = ↓ 4-OH
- **Anti-inflammatory** properties
- Unfortunately has low oral bioavailability
- Numerous preclinical/clinical studies show promising potential of OA against cancer, cardiovascular diseases, inflammation, neurological disorders, rheumatoid arthritis, osteoarthritis, etc.

• An D, Song Z, Yi Y, et al. Oroxylin A, a methylated metabolite of baicalein, exhibits a stronger inhibitory effect than baicalein on the CYP1B1-mediated carcinogenic estradiol metabolite formation. *Phytotherapy Research*. 2019;33(4):1033-1043. doi:<https://doi.org/10.1002/ptr.6297>

• Sajeed A, Hegde M, Girisa S, et al. Oroxylin A: A Promising Flavonoid for Prevention and Treatment of Chronic Diseases. *Biomolecules*. 2022;12(9):1185. doi:<https://doi.org/10.3390/biom12091185>

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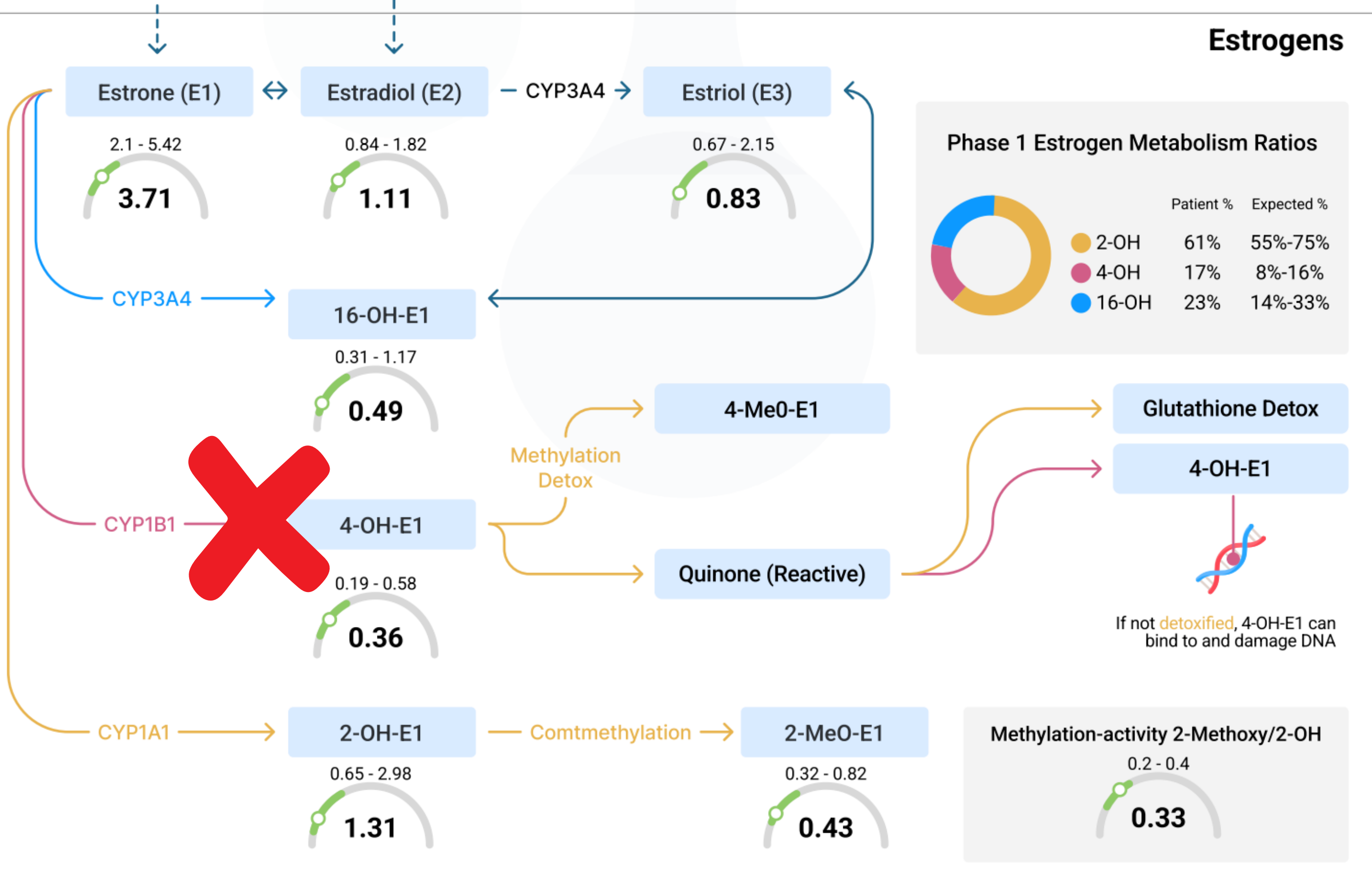


Sajeev A, Hegde M, Girisa S, et al. Oxroxylin A: A Promising Flavonoid for Prevention and Treatment of Chronic Diseases. *Biomolecules*. 2022;12(9):1185. doi:<https://doi.org/10.3390/biom12091185>


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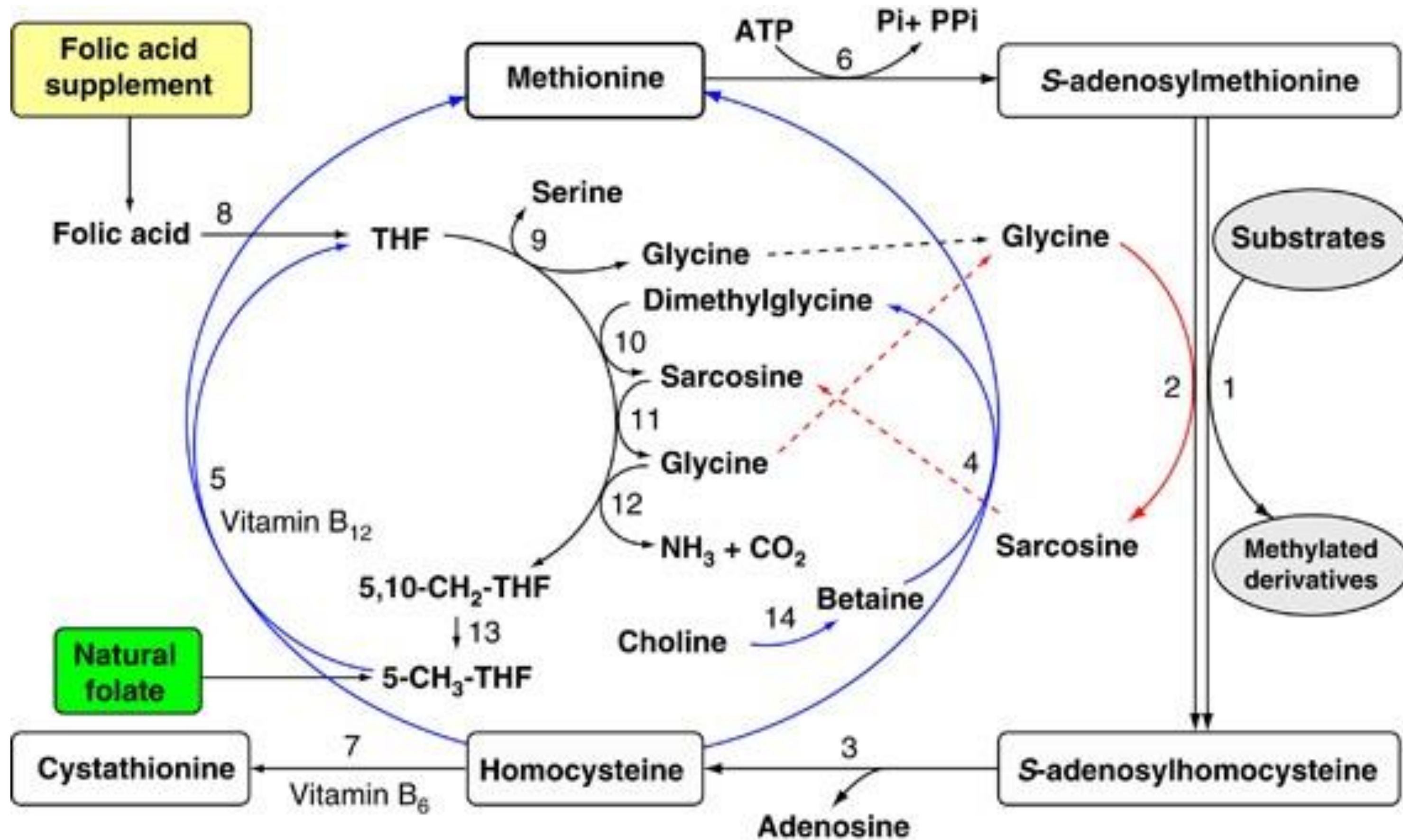
The Vibrant
Longevity
Summit

Estrogens



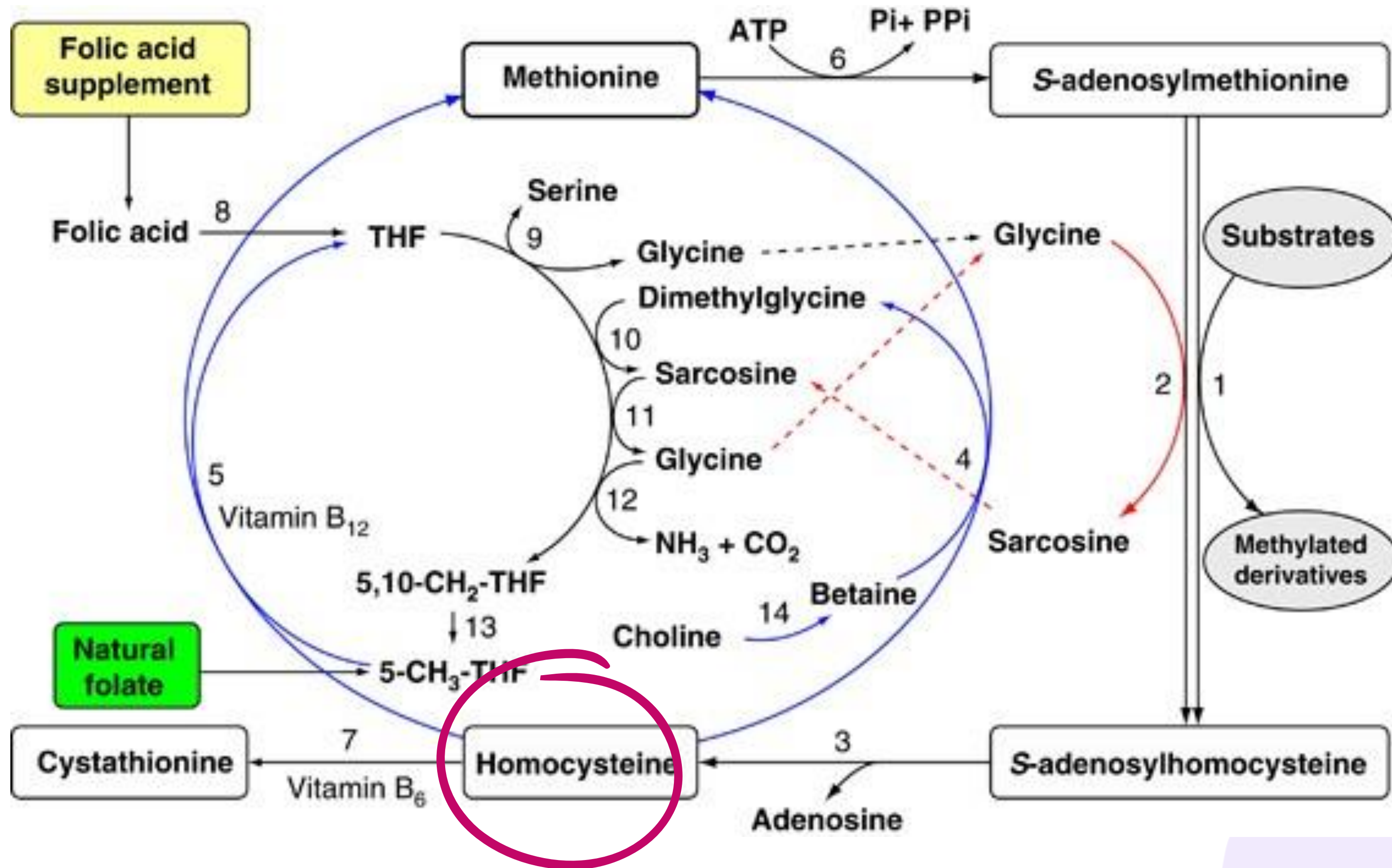
What If she has **low estrogen**?

- Make sure phases 2 and 3 are working well
- Consider skipping DIM/I3C (foods are ok)
- Consider focusing on  4-OH
- Consider glutathione support

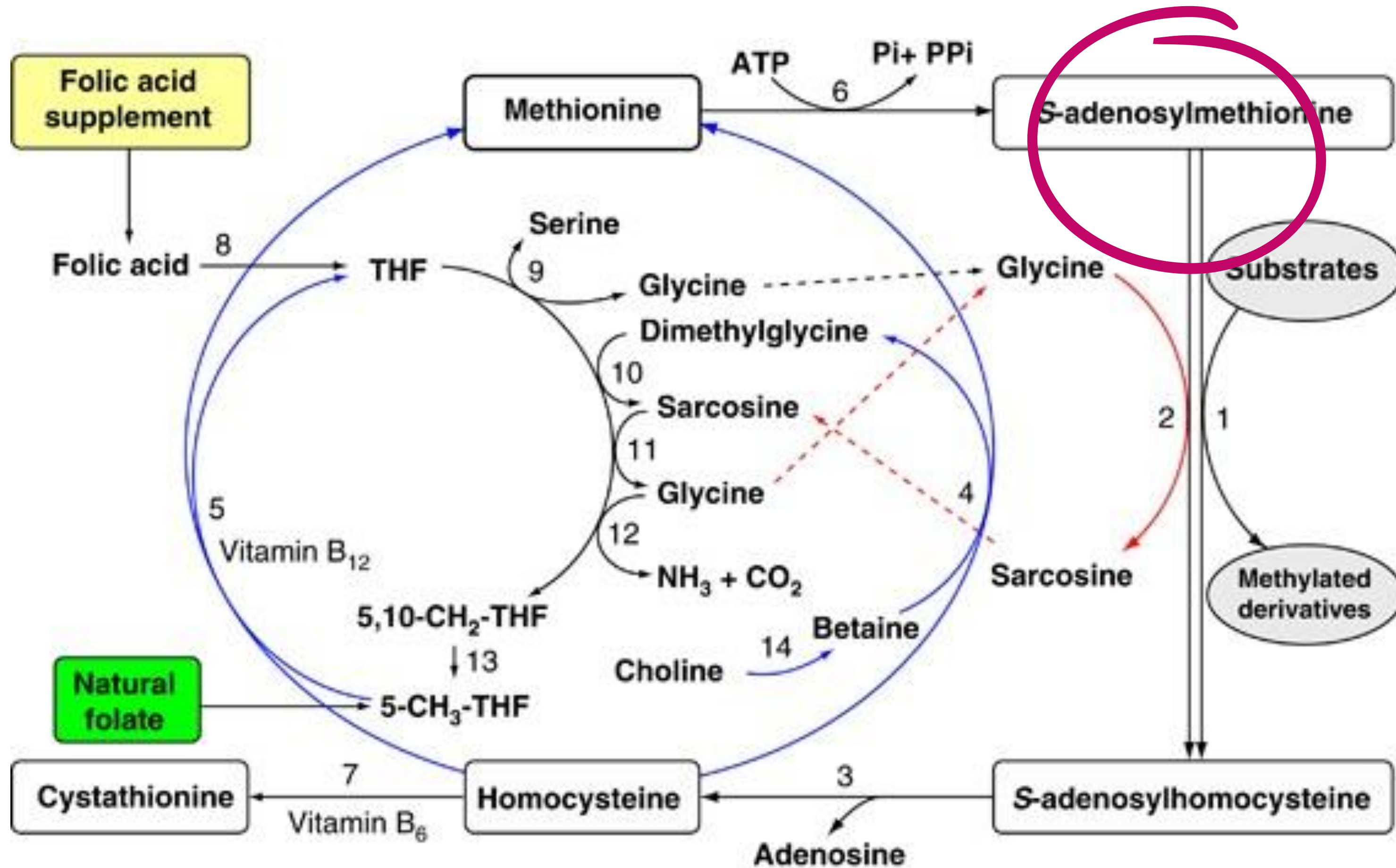


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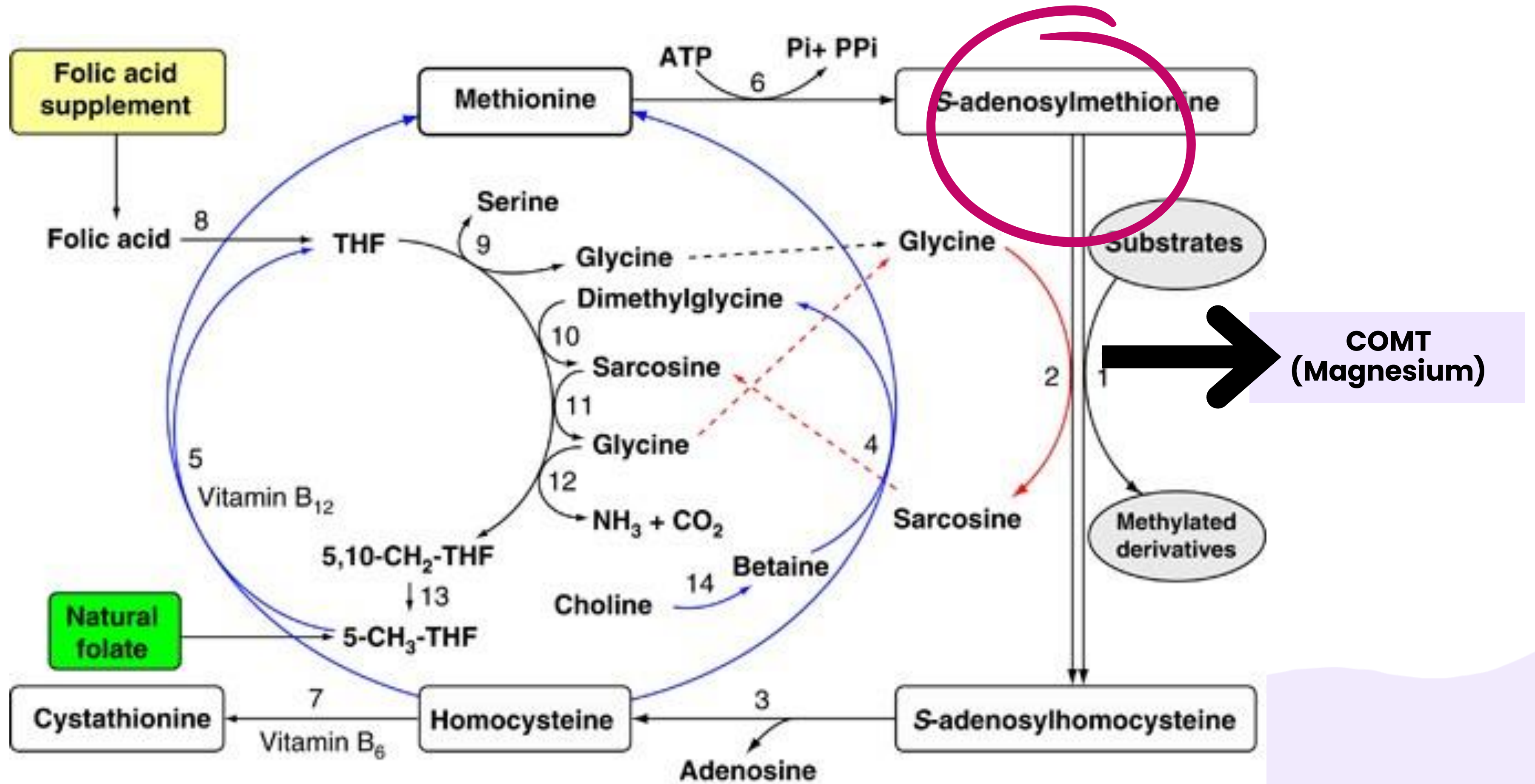
Zhou SS, Zhou YM, Li D, Lun YZ. Dietary methyl-consuming compounds and metabolic syndrome. Hypertension Research. 2011;34(12):1239-1245. doi:<https://doi.org/10.1038/hr.2011.133>



Zhou SS, Zhou YM, Li D, Lun YZ. Dietary methyl-consuming compounds and metabolic syndrome. Hypertension Research. 2011;34(12):1239-1245. doi:<https://doi.org/10.1038/hr.2011.133>



1.
 Zhou SS, Zhou YM, Li D, Lun YZ. Dietary methyl-consuming compounds and metabolic syndrome. Hypertension Research. 2011;34(12):1239-1245. doi:<https://doi.org/10.1038/hr.2011.133>



1.
Zhou SS, Zhou YM, Li D, Lun YZ. Dietary methyl-consuming compounds and metabolic syndrome. Hypertension Research. 2011;34(12):1239-1245. doi:<https://doi.org/10.1038/hr.2011.133>



In Summary:

1. Detox/metabolism starts with phase 1 but make sure phases 2 and 3 are healthy.
2. Phase 1 heavily occurs in the liver – how's liver health?
3. Don't give everyone DIM/I3C
4. Lots of promising research on CYP1B1 Inhibitors

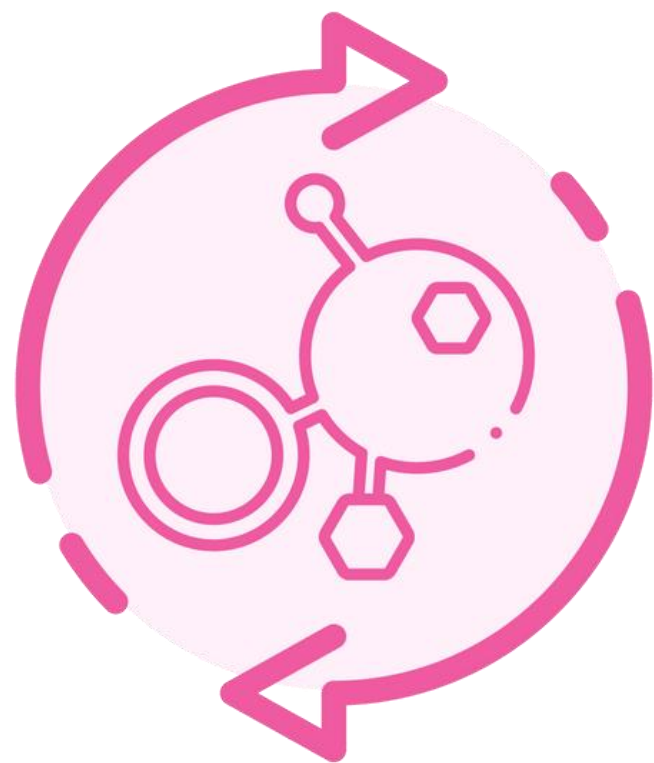


Thank You!

www.drcarriejones.com

For my FREE ESTROGEN DETOX EBOOK





Hormone Optimization

Elevating Lifespan
Through Endocrine
Balance



Session 4

**Dr. Angela D
Mazza, DO,
ABAARM,
FAAMFM,
ECNU**

Meet Your **Speaker**

**Dr. Angela D Mazza,
DO, ABAARM,
FAAMFM, ECNU**

Integrative Endocrinologist
Founder/Medical Director–
Metabolic Center for Wellness
Oviedo, FL





Hormone Optimization:

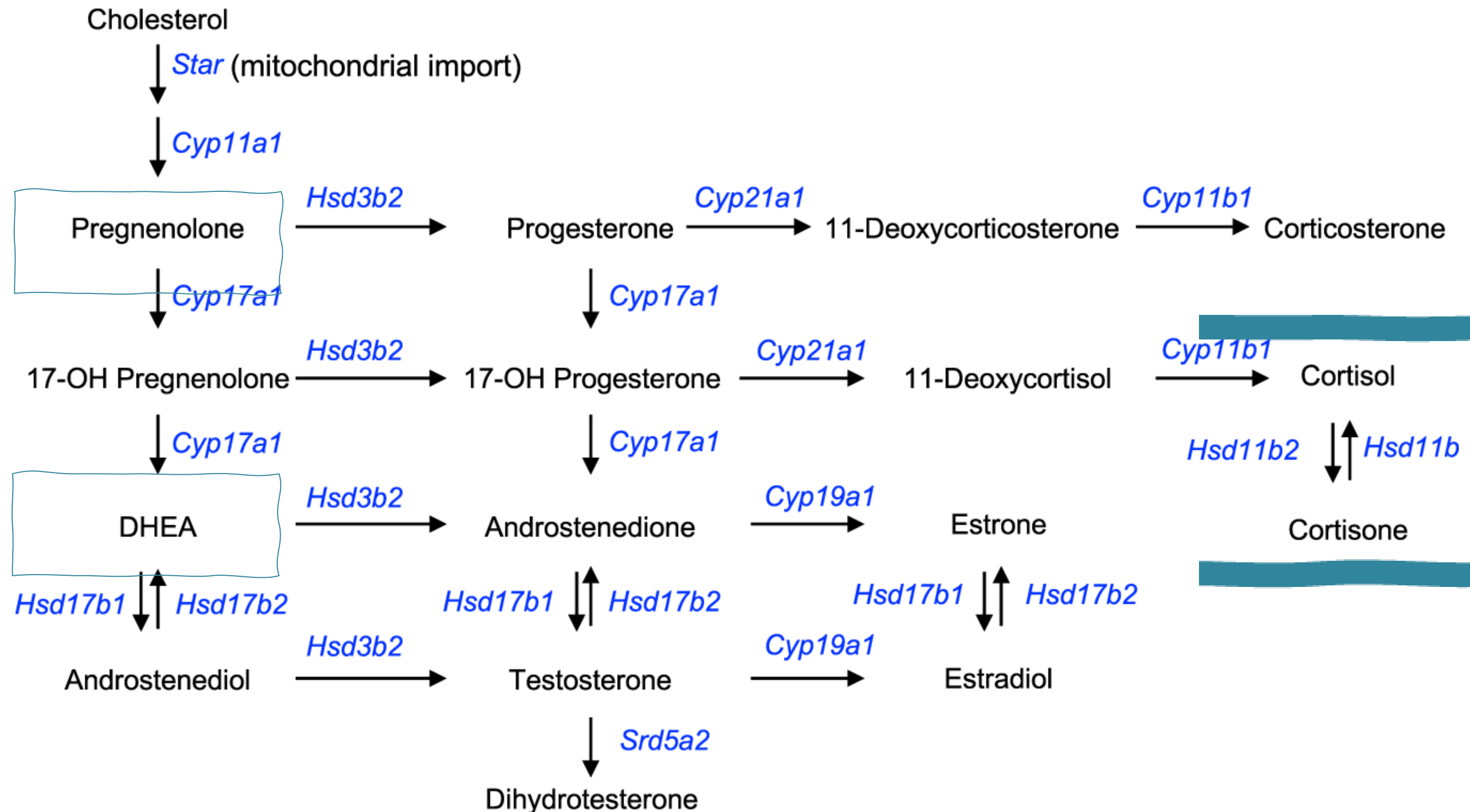
*Elevating Lifespan and Peak Clinical
Performance – Business*

Learning Objectives

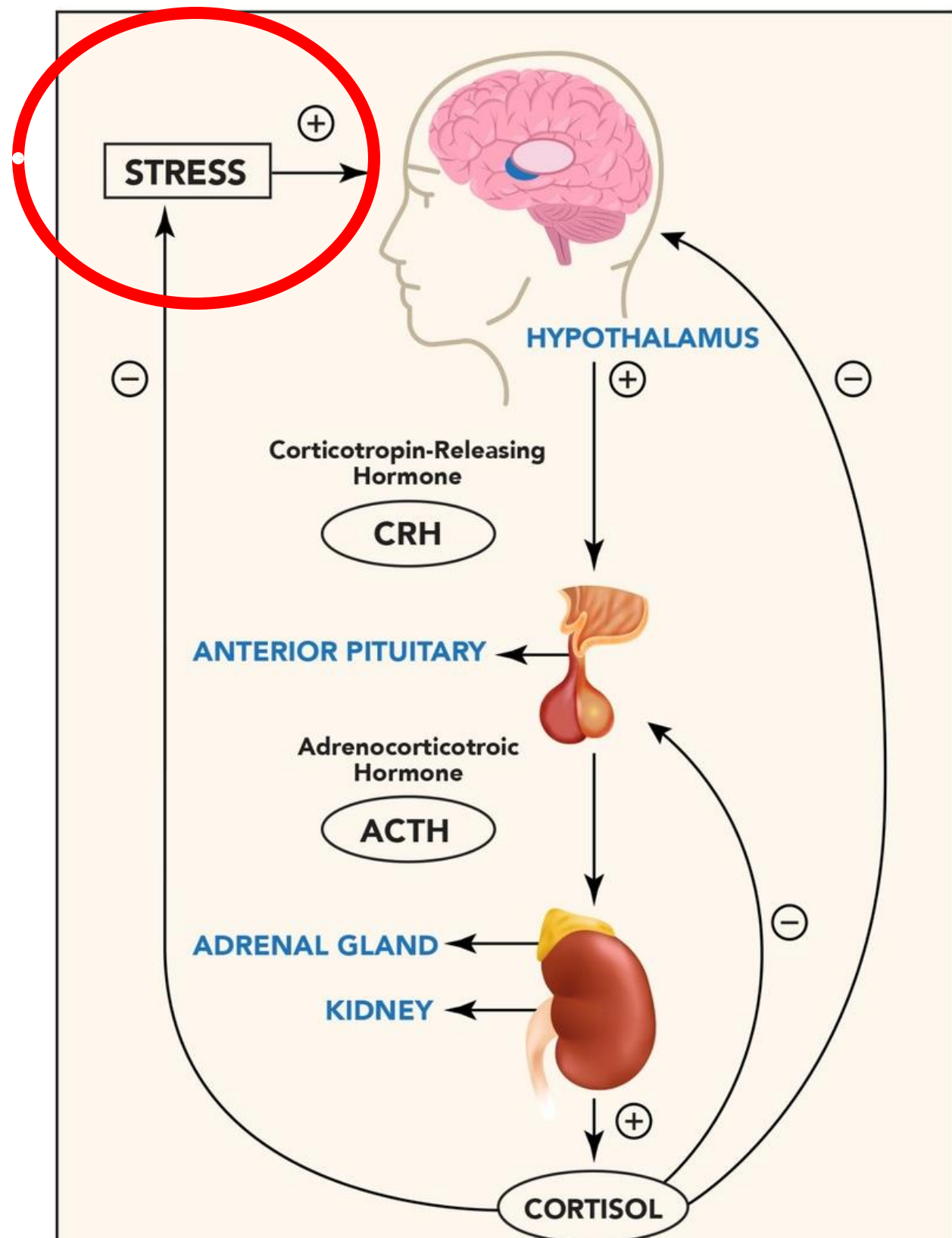
- To understand clinical relevance of hormone metabolism and clearance
- To master lab interpretation with key panels
- To learn protocol design across life stages
- To structure hormone-focused longevity programs for real-world use

Hormone Clearance and Detox Pathways

The Steroidogenic Pathway: the Big Picture



The Hypothalamic-Pituitary-Adrenal (HPA) axis



- Adrenal Inner part - **“medulla”**

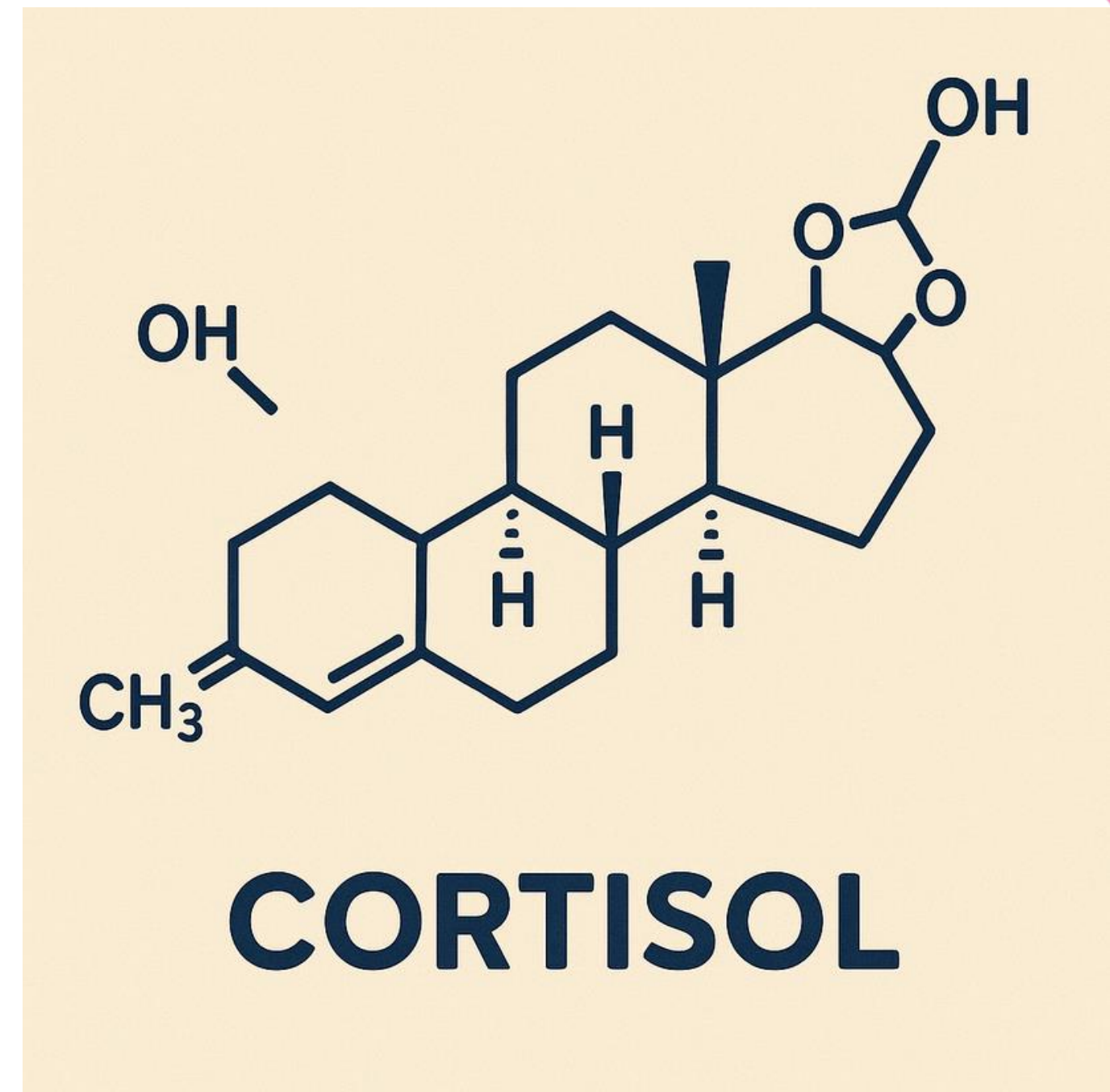
- “Flight or fight” hormones
- Catecholamines
 - Epinephrine (adrenaline)
 - Norepinephrine
 - Dopamine

- Adrenal outer part - **“cortex”**

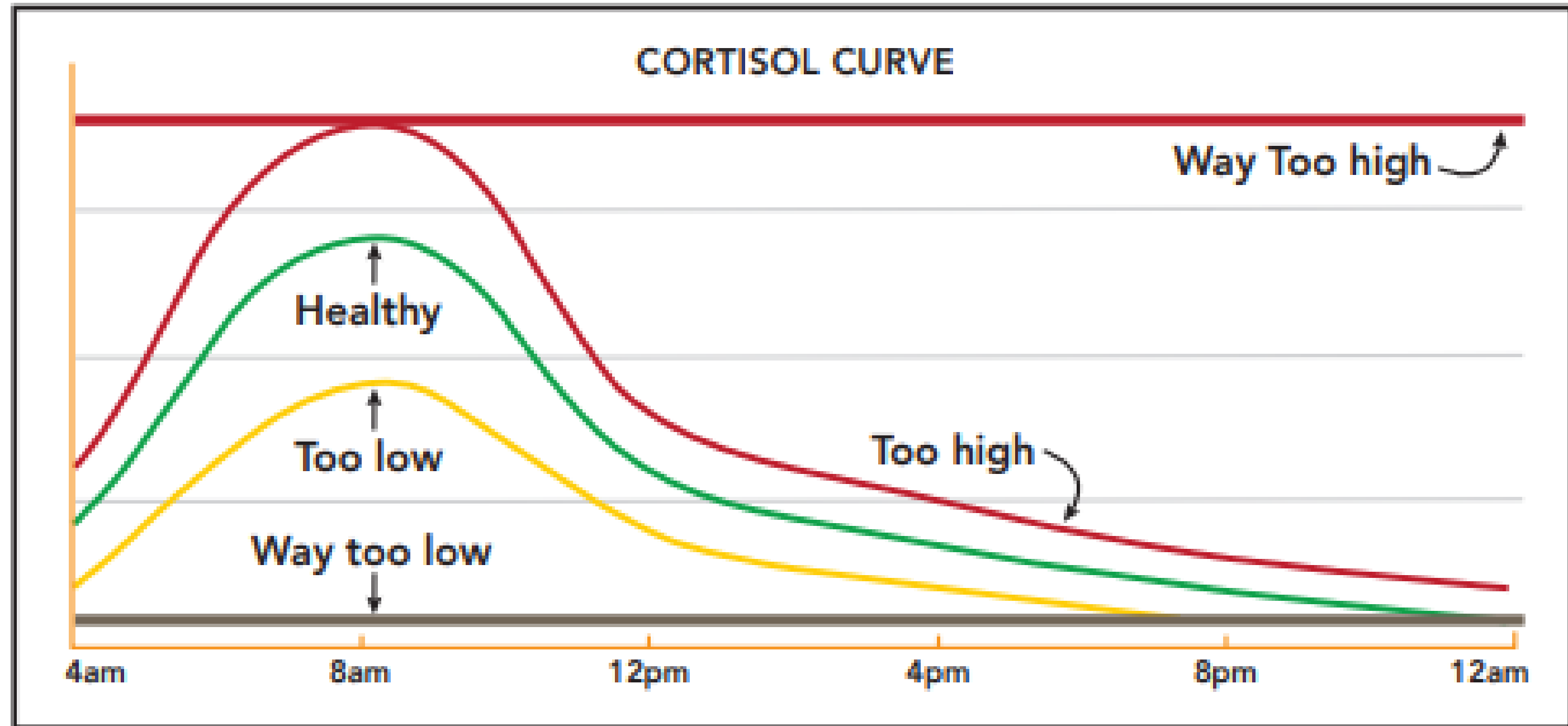
- Cortisol
- Aldosterone
- Sex steroids - DHEA

Cortisol: The Stress Hormone

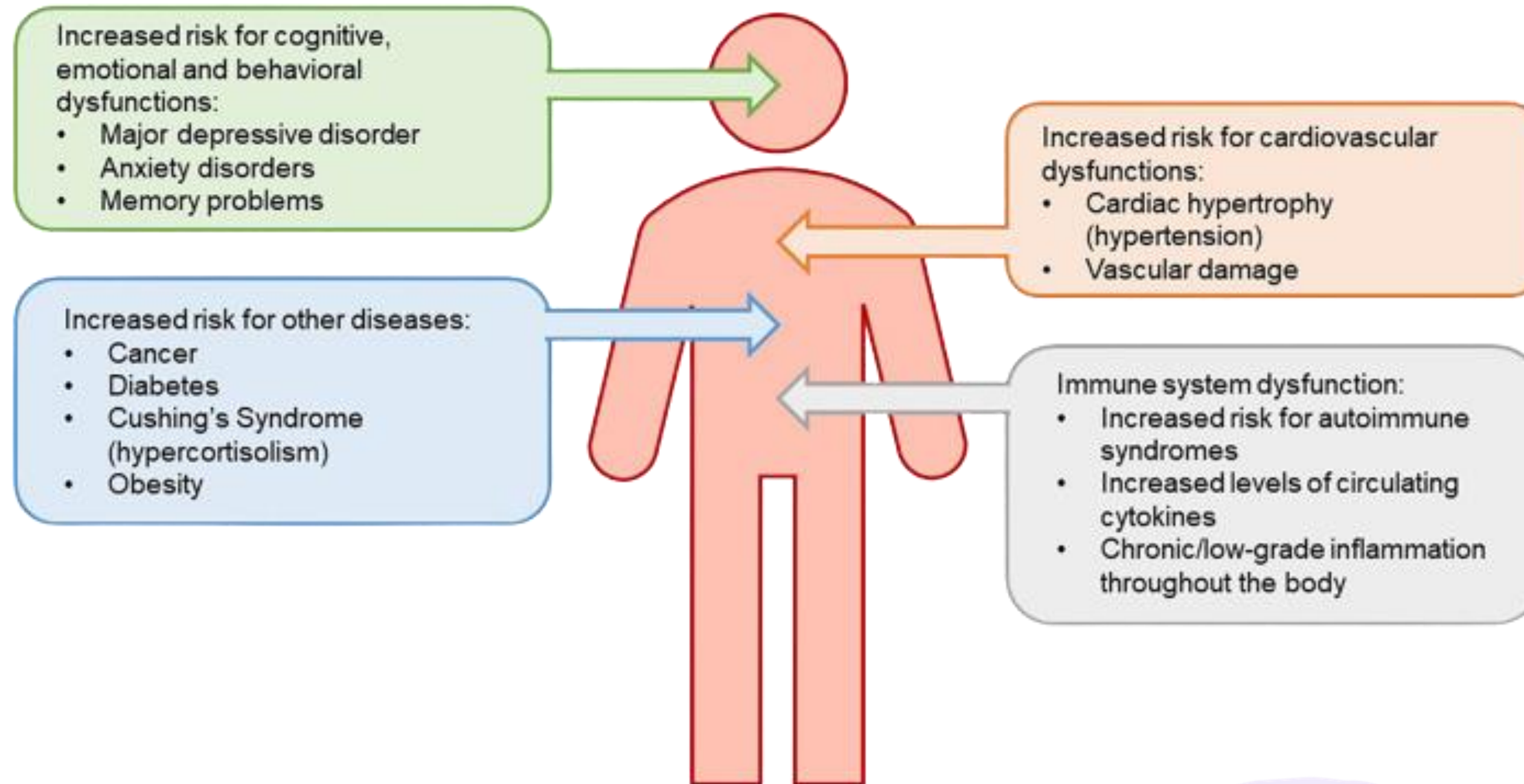
- Released in response to physical, emotional, or psychological stress
- Helps mobilize energy
- Increases blood glucose
- Enhances brain function
- Suppresses non-essential functions (e.g., digestion, reproduction) during acute stress



Characterization of the HPA Axis



HPA Axis Dysfunction Causes Chronic Disease



Sheng JA, et al. *Front Behav Neurosci*. 2021 Jan 13;14:601939.

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Stress is at the Center of Immunometabolism

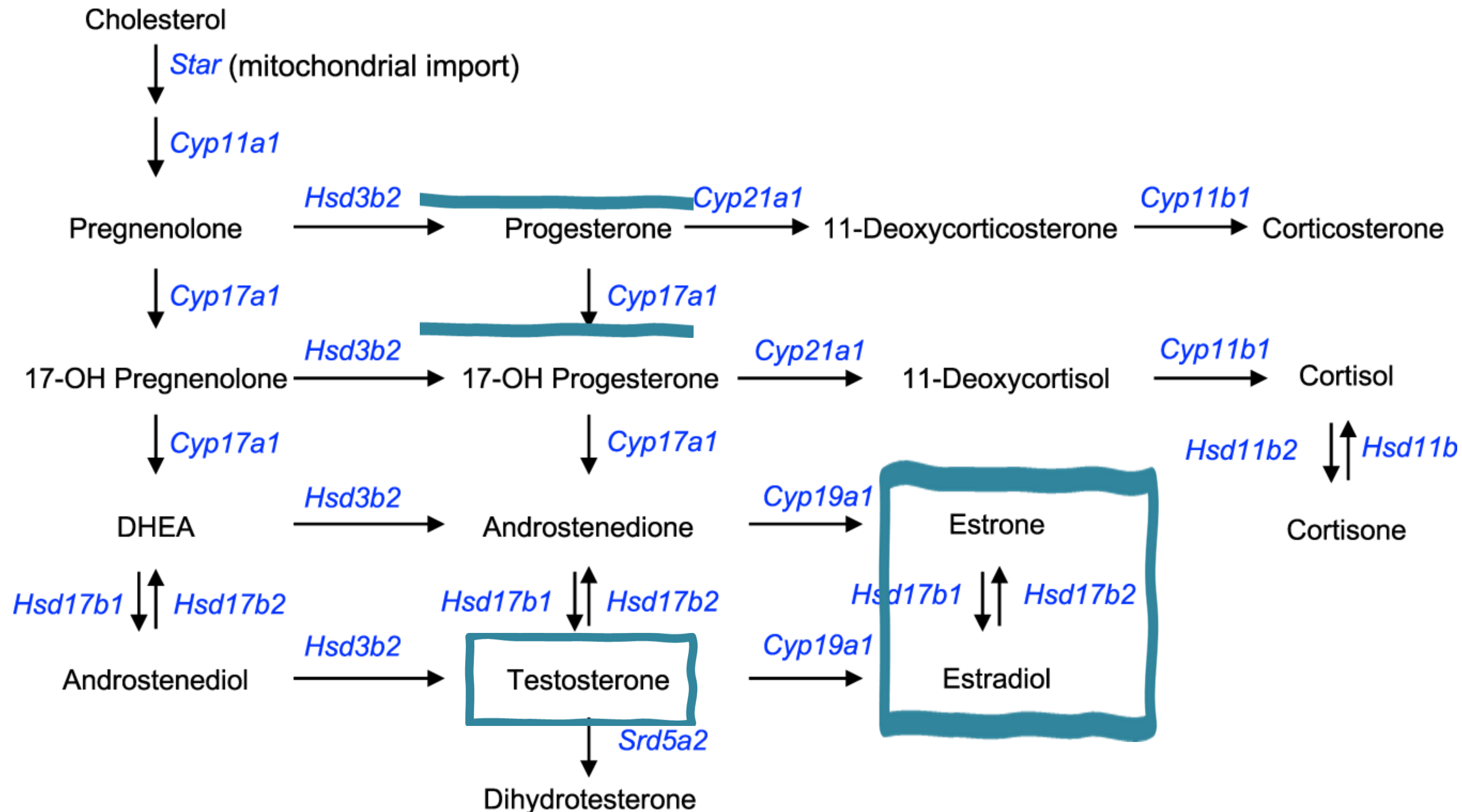
- ***Stress can induce metabolic dysregulation***
 - insulin resistance, altered glucose metabolism, and changes in lipid and protein metabolism.
 - **impaired immune responses and inflammation**
 - **increased oxidative stress**
 - **damage immune cells and impair their function**

Stress impacts the production and clearance of other hormones.

Hotamisligil GS. Foundations of Immunometabolism and Implications for Metabolic Health and Disease. *Immunity*. 2017 Sep 19;47(3):406-420.

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The Steroidogenic Pathway: the Big Picture



Stress and Hormonal Cross-Talk:

Impact on estrogen, progesterone and testosterone

HPA axis activation alters hormonal balance

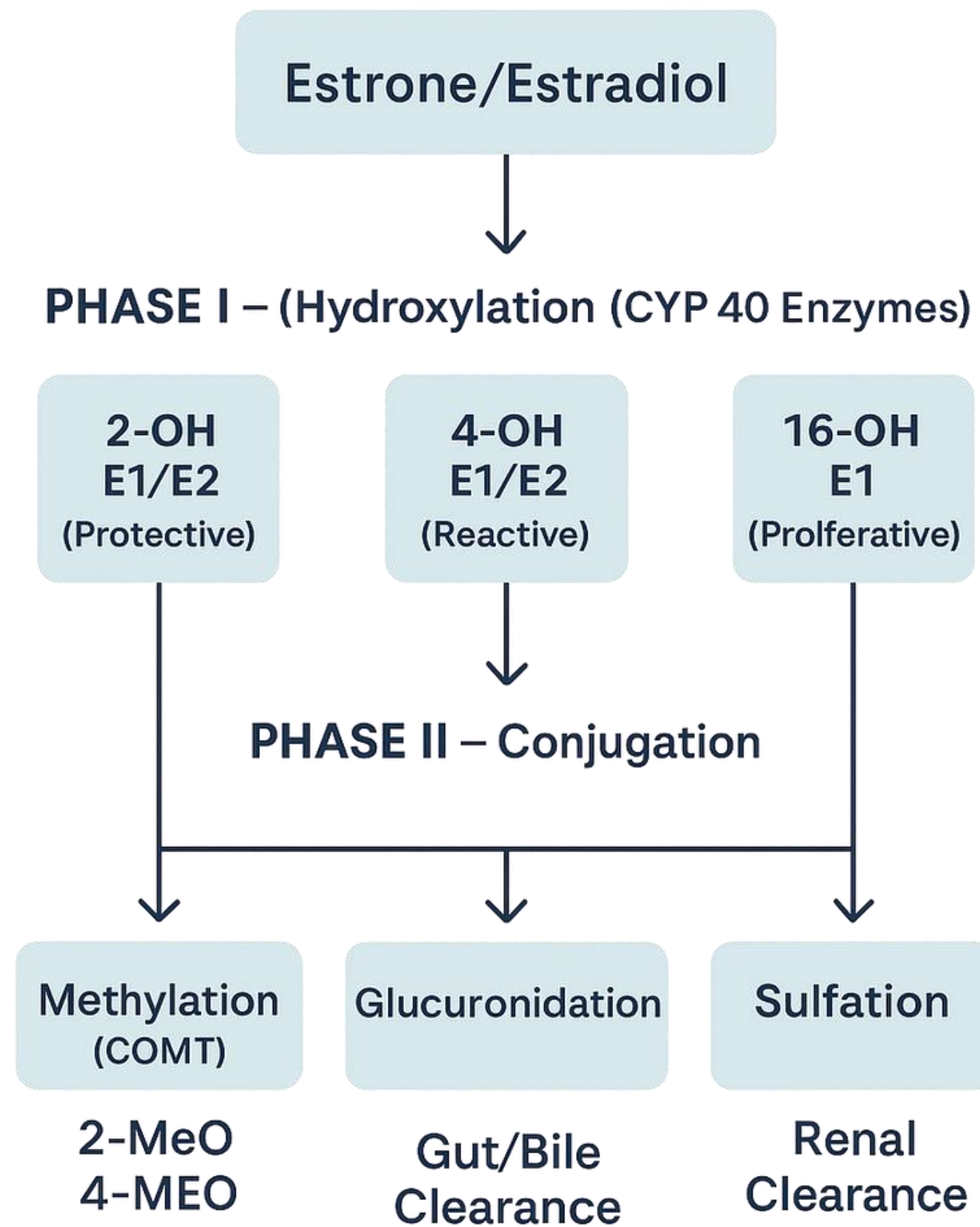
- Cortisol “steals” precursors
- Progesterone suppression
- Estrogen dominance
- Testosterone decline

Disrupted metabolism and receptor sensitivity

- Altered hepatic clearance
- Aromatase upregulation
- Receptor resistance

Berga SL, Loucks TL. . *Minerva Ginecol*. 2005;57(1):45-54.
Panay N, Fenton A.. *Obstet Gynaecol Reprod Med*. 2017;27(5):147-153.
Chrousos GP. . *Nat Rev Endocrinol*. 2009;5(7):374-381.

Estrogen Detoxification Pathways



Zhu, B. T., & Conney, A. H. (1998). *Carcinogenesis*, 19(1), 1–27.

Methylation and Cofactors in Hormone Metabolism

- Why methylation matters–
 - Estrogen clearance (via COMT)
 - Neurotransmitter balance (DOP, SER)
 - Detoxification
 - DNA repair
 - Epigenetic regulation
- Key Cofactors:
B12, Folate, Choline, Magnesium
- Genetic variants
MTHFR (CG77T, A1298C)
COMT (Val158Met)

Deth, R. et al. (2008).. *NeuroToxicology*, 29(1), 190–201.

James, S. J., et al. (2005).. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 135B(1), 43–68.

Zakhari, S. (2013). *Alcohol Research: Current Reviews*, 35(1), 40–46. h

Scaglia, F., & Brunetti-Pierri, N. (2004).. *American Journal of Medical Genetics Part C: Seminars in Medical Genetics*, 125C(1), 49–58.

Environmental Load and Endocrine Disruptors

Environmental Load and Endocrine Disruptors

- **Environmental Load**

- Total burden of external chemical exposures

- **Endocrine disrupting chemicals (EDCs)**

- Exogenous substances that mimic, block, or interfere with body's natural hormones

- Include:

- Plastics – BPA, phthalates
- Heavy metals – mercury, lead
- Pesticides – atrazine, DDT, glyphosates
- Flame retardants (PBDEs)
- Fragrances and solvents

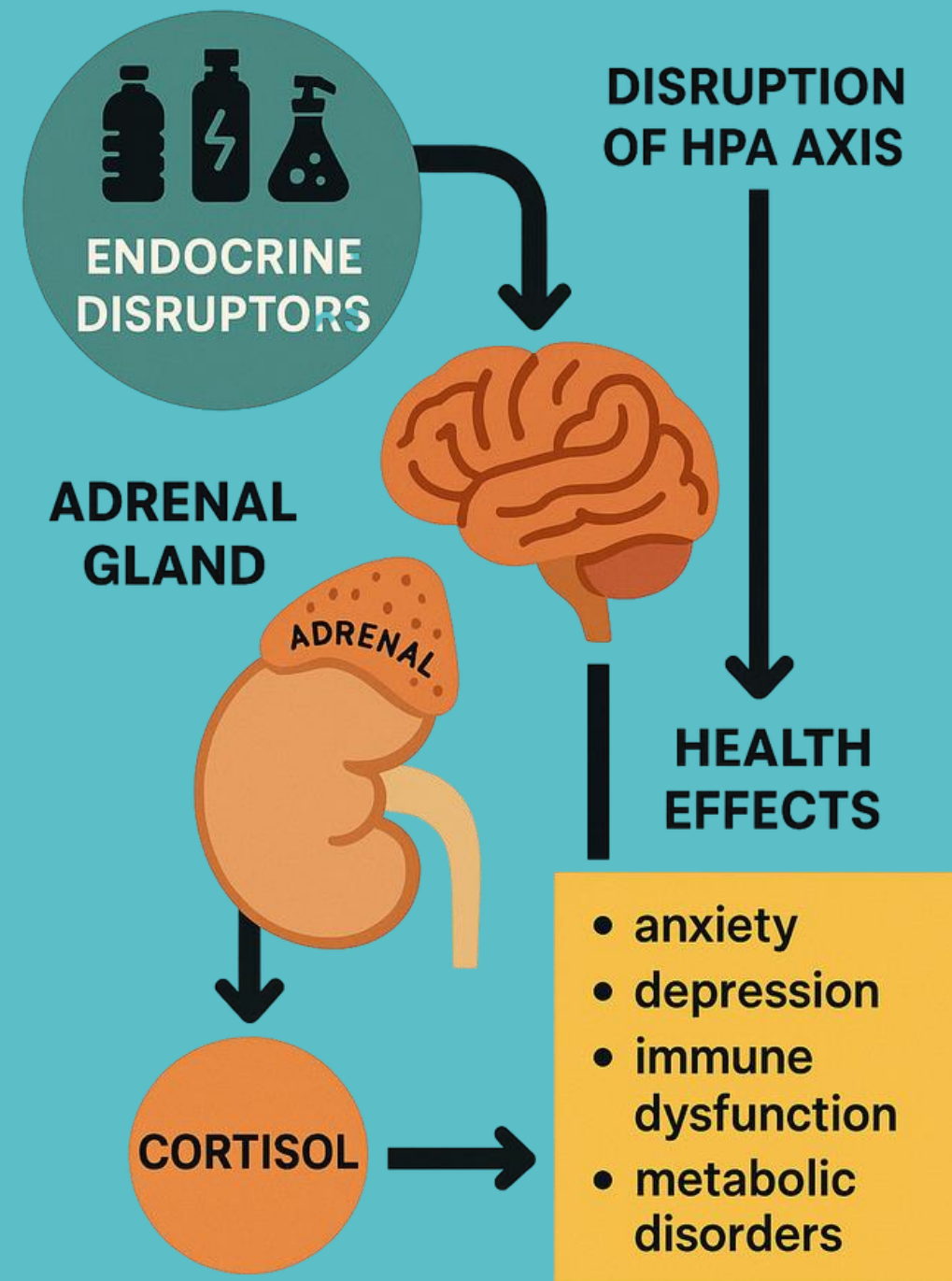
Diamanti-Kandarakis, E., et al. (2009).. *Endocrine Reviews*, 30(4), 293–342.

Gore, A. C., et al. (2015).. *Endocrine Reviews*, 36(6), E1–E150.

Meeker, J. et al (2009). *Philosophical Transactions of the Royal Society B: Biological Sciences*, 364(1526), 2097–2113.

Ko, Y. C., Yang, H. Y., & Chu, C. M. (2021). *Environmental Research*, 195,

ENDOCRINE DISRUPTORS AND CORTISOL



EDCs and Cortisol

- Disruption of HPA axis
 - Chronic overstimulation
 - Blunting of cortisol response
- Direct adrenal effect
 - PCBs and dioxin impair steroidogenesis
 - Pesticides can alter enzymes
- Epigenetic modifications
- Neurodevelopmental effects
- Common culprits
 - BPA, phthalates, PCBs, pesticides

Just a few common EDCs and other hormonal imbalance

EDC	Hormonal Target	Mechanism of disruption
BPAs	Estrogen & Thyroid	Estrogen receptor (ER) agonist → ↑ estrogenic activity; impairs thyroid receptor (TR) binding
Phthalates	Testosterone	↓ Leydig cell function → ↓ testosterone synthesis, ↑ SHBG
Heavy Metals	Thyroid & sex hormones	↓ Thyroid peroxidase (TPO) activity, ↑ oxidative stress, ↓ sex hormone biosynthesis
Atrazine	Estrogen/ Testosterone	Promotes aromatase → ↑ estrogen, ↓ testosterone
PBDEs	Thyroid	Disrupt thyroid hormone transport and binding proteins

Diamanti-Kandarakis, E., et al. (2009).. *Endocrine Reviews*, 30(4), 293–342.

Gore, A. C., et al. (2015).. *Endocrine Reviews*, 36(6), E1–E150.

Meeker, J. et al (2009). *Philosophical Transactions of the Royal Society B: Biological Sciences*, 364(1526), 2097–2113.

Ko, Y. C., Yang, H. Y., & Chu, C. M. (2021). *Environmental Research*, 195,

Detox Support Principles

- **Testing first** for –

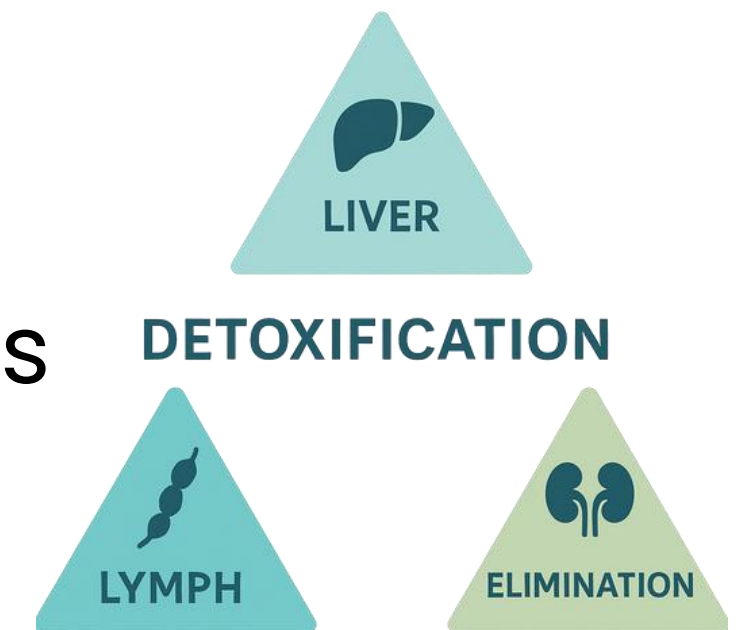
- Total toxin burden panel
- Estrogen metabolism
- Complete thyroid panel
- Complete androgen eval

- **Nutrient Support**

- Glutathione
- NAC
- Fiber
- Cruciferous veggies

- **Lifestyle strategies**

- Saunas
- Binders
- Hydration



Pizzorno, J. (2014). Glutathione! *Integrative Medicine: A Clinician's Journal*, 13(1), 8–12.
Zhang, Y., Tang, L., Gonzalez, V. (2003).. *Cancer Research*, 63(19), 6450–6457.
Crinnion, W. J. (2011). . *Alternative Medicine Review*, 16(3), 215–225.
Liska, D. J., et al. (2006). Fiber and health: An overview. *Nutrition Today*, 41(6), 263–266.

Interpreting and Integrating Advanced Lab Panels

Key Panels for Functional Hormone Insight

- **Urine or salivary testing for comprehensive hormones**

- Cortisol curve
- Estrogen metabolites (2-OH, 4-OH, 16-OH)
- Progesterone
- DHEA
- Testosterone
- Melatonin

- **Methylation Panel**

- **Total tox burden panel**

- **Complete thyroid panel with antibodies**

- **Gut testing**

Kalish, D. (2020). *The Kalish Method: Functional lab interpretation strategies*. Kalish Institute.

Zakhari, S. (2013). Bermuda triangle for estrogen: Role of methylation, metabolism, and microRNA. *Alcohol Research: Current Reviews*, 35(1), 40–46.

James, S. J., et al. (2005). Abnormal redox and methylation capacity in autism spectrum disorders. *Am J Med Genet B*, 135B(1), 43–68.

Fasano, A. (2012). Zonulin and gut permeability: Targeting autoimmune pathways. *Clin Rev Allergy Immunol*, 42(1), 71–78.

Integrating Lab Data Into Protocol Design

- **Map dysfunction**

- Examples:
- *Low progesterone + high 4-OH estrone = poor detox, methylation overload*
- *Flat cortisol + low DHEA = HPA axis dysfunction, mitochondrial compromise*

Integrating Lab Data Into Protocol Design (cont'd)

- **Identify priorities**

- Detox first in toxin-heavy or estrogen-dominant cases
- Calm inflammation in high ab states before adding hormones

- **Layer protocols strategically**

- Phase 1: gut/liver support, binders, methyl donors
- Phase 2: hormone modulation (DIM, P, adaptogens)
- Phase 3: retesting and maintenance based on trending biomarkers

Precision Protocols Across Life Stages – *testing is key*

Adrenal Patterns Over Life's Stages

- *HPA axis governs cortisol, DHEA, aldosterone, and indirectly influences thyroid and sex hormones*
- **Life stage**
 - **Reproductive years** –
key pattern: cortisol spikes, PMS, anxiety
 - **Perimenopause** – key pattern: cortisol flattening, low DHEA
 - **Menopause/post-menopause** –
key pattern: hypocortisolism, sleep issues
 - **Andropause** – key pattern: increased SHBG, decreased free testosterone, decreased AM cortisol

Estrogen Metabolism Support Over Life's Stages

- *Precision protocols should support safe estrogen clearance through all hormonal transitions*
- **Life stage**
 - **Reproductive years** – key focus: PMS, heavy cycles, acne
 - **Perimenopause** – key focus: estrogen dominance, detox slowdown
 - **Menopause/post-menopause** – key focus: HRT support, cancer risk mitigation
 - **Men** – key focus: estrogen detox, SHBG balance

Hormonal Recalibration by Life Stage

- The **hormonal triad** –
 - **Stress** (HPA axis)
 - Elevated cortisol → hormone depletion
 - **Gut microbiome** –
 - Dysbiosis → estrogen recirculation
 - **Thyroid** (metabolism)
 - Regulates SHBG, metabolic rate, hormone receptor expression
- *Essential to address in perimenopause, menopause, and andropause*

Program Structure and Business Implementation

Why Bundle Functional Lab Panels?

- **Root-cause clarity:**
 - Bundling hormone, gut, detox, and toxin panels reveals multisystem imbalances driving hormonal dysfunction
- **Efficiency:**
 - Reduces time to insight by identifying co-contributors (e.g., toxin load + estrogen dominance + gut dysbiosis)
- **Protocol precision:**
 - Enables tailored interventions based on detox capacity, immune status, and clearance pathways
- **Patient engagement:**
 - Improves buy-in by demonstrating interconnected patterns and tracking tangible biomarkers over time

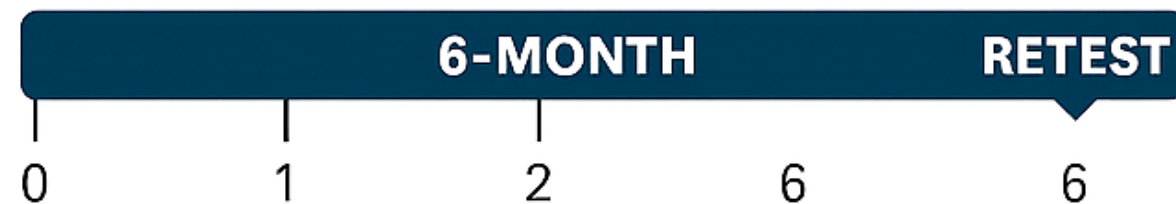
Structuring 3-vs.6-month hormone programs

RETESTING INTERVALS



At 75-90 days

Confirm hormone trends; refine support protocols



At 90 days and 6 months

Evaluate adjustments; ensure long-term recalibration

STAGGER vs. STACK INTERVENTIONS

STAGGERED APPROACH

- Ideal for sensitive or toxic patients
- Phase in adrenal/gut support; then detox, hormones

STACKED APPROACH

- Ideal for resilient or performance patients
- Combine adrenal; hormone and detox support early

Hardy, M. L., & O'Donnell, J. A. (2017). Integrative Medicine Reports, 12(2), 83–91.
Liska, D., et al. (2021). Integrative Medicine: A Clinician's Journal, 20(5), 22–30.
Godfrey, A. R., & Wahls, T. (2023). Journal of Restorative Medicine, 12(1), 14–28.

Follow Up Strategy – Leverage Early Wins!

- **Initial 30–45 days –**
 - Gut and liver support, stress modulation detox, replace micronutrients
 - **Track markers** (eg – energy, sleep, bowel habits, mood)
- **By weeks 8–10** – Share “Before and After” visual or retest previews
- Use data trends to **build buy-in** for next phase (eg – hormone therapy or deeper detox)

Bland, J. S. (2015). *Journal of the American College of Nutrition*, 34(1), 1–9.

Fasano, A. (2012). *Clinical Reviews in Allergy & Immunology*, 42(1), 71–78.

Fitzgerald, K. N., et al. (2021). *Aging (Albany NY)*, 13(7), 9419.

Kalish, D. (2020). The Kalish Method: Functional medicine lab interpretation and sequencing for long-term results. *Kalish Institute White Paper*.

Delivery Models for Hormone Longevity Programs



Concierge Model

- High-touch, personalized experience
- Advanced testing and ongoing access
- Premium monthly or 3–6 month packages



Hybrid/Telehealth Model

- Lab testing + virtual consultations
- Digital support tools
- Remote patient engagement



Group Programs

- Education, community, and accountability
- Modules + live Q&A
- Lower price point, high scalability

Eisenberg, D. M., et al. (2016). *Health Affairs*, 35(3), 361–367.

Anghel, L. A., et al. (2021). *Endocrine Connections*, 10(5), R147–R158.

Kirschner, M. J., & Long, J. M. (2023). *Journal of Integrative and Complementary Medicine*, 29(4), 272–279.

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Case Studies

Case Study 1 – 47-year-old female

- **Occupation:** Real estate agent, mother of 2 teens
- **Presenting:** fatigue, brain fog, constipation, irregular cycles, breast tenderness
- **History:** long-term use of plastics, high-stress job, poor sleep, ultra-processed diet

Case Study 1 – Initial Labs

Hormone Zoomer:

Increase E1/E2 ratio
elevated 4-OH pathways
decreased progesterone
endocrine disruptors:

elevated BPA, phthalates, microplastics

Gut Zoomer: Low *Lactobacillus*, high beta-glucuronidase, candida overgrowth

Thyroid Zoomer: Negative antibodies, mild subclinical hypothyroidism

Case Study 1 – Key Concerns

- Hormonal imbalance
- GI dysbiosis
- Environmental toxin exposure

Case Study 1 – Phased Protocol Overview

Month 1-2: Foundation

- Liver/gut support
 - Broccoli sprouts, DIM, calcium-D-glucarate, NAC
 - Magnesium, psyllium, glycine
- Binders (chlorella, charcoal), sauna 3x/week
- Diet crucifer-rich, gluten/dairy-free, filtered water, protein-focused

Case Study 1 – Phased Protocol Overview

Month 3: Hormone optimization

- Adaptogens:
 - rhodiola, ashwagandha
- Bioidentical progesterone
- Support methylation:
 - methyl b12, 5MTHF, magnesium glycinate

Case Study 1 – Phased Protocol Overview

Month 4–6: Detox Continuation + Retest

- **Retesting:** Hormone Zoomer, Gut Zoomer, thyroid panel
- adjust based on e metabolite shift, gut flora improvement
- Maintenance with glutathione, sauna, high-fiber diet

Early wins (first 45 days) – improved bowel regularity, more restful sleep, reduced breast tenderness, better energy upon waking

Case Study 2 – 42-year-old male

- **Occupation:** tech executive, competitive cyclist
- **Presenting:** fatigue despite high fitness, low libido, poor sleep, reduced muscle recovery, irritability
- **History:** Intermittent fasting, overtraining, high stress, low-carb diet, 2-3 cups of coffee/day

Case Study 2 – Initial Labs

Hormone Zoomer:

- Salivary cortisol (4-point): downward shift of cortisol curve
- Low melatonin
- Total testosterone adequate, but low DHT and high estradiol

Methylation panel: COMT slow variant, ↑ homocysteine

Gut Zoomer: mild dysbiosis, low SCFA

Micronutrient panel: Low zinc, magnesium, CoQ10

Case Study 2 – Key Concerns

- Testosterone resistance (adequate levels, low effect)
- HPA dysfunction (flat curve)
- Mitochondrial depletion
- Sleep fragmentation

Case Study 2 – Phased Protocol Overview

Month 1–2: HPA–Axis & Mitochondria Repair

- **Cortisol support:** adrenal adaptogenic blend, stress management strategies
- **Mitochondrial nutrients:** ubiquinol, B–complex, magnesium
- **Antioxidant support:** NAC, Vitamin C
- **Sleep focus:** GABA, L–theanine, melatonin, screen curfew
- **Dietary recs:** increased fiber and cruciferous vegs, decrease inflammatory foods, limit caffeine

Case Study 2 – Phased Protocol Overview

Month 3: Testosterone Optimization

- Zinc + boron to reduce SHBG
- DIM + calcium-D-glucurate to clear excess estrogen
- Creatine + HIIT cycling blocks for DHT and androgen sensitivity
- Continue adaptogens

Month 4–6: Advanced Detox + Retesting

- **Retest labs:** Hormone Zoomer
- Consider low-dose clomiphene or DHEA if trends plateau
- Continue sauna, fiber, mineral repletion

Takeaway Points

- Hormone imbalance is multisystemic and can be impacted by chronic stress.
- Diurnal cortisol and estrogen metabolites reveal root cause patterns.
- Environmental toxins amplify hormonal resistance.
- Functional lab bundles improve diagnostic clarity and buy-in.
- Protocols must be tailored by life stage.
- Retesting and early wins drive program adherence *better patient results and patient satisfaction.*



Thank You!

Any questions?

angela.mazza@metaboliccenterforwellness.com

metaboliccenterforwellness.com