Endo+

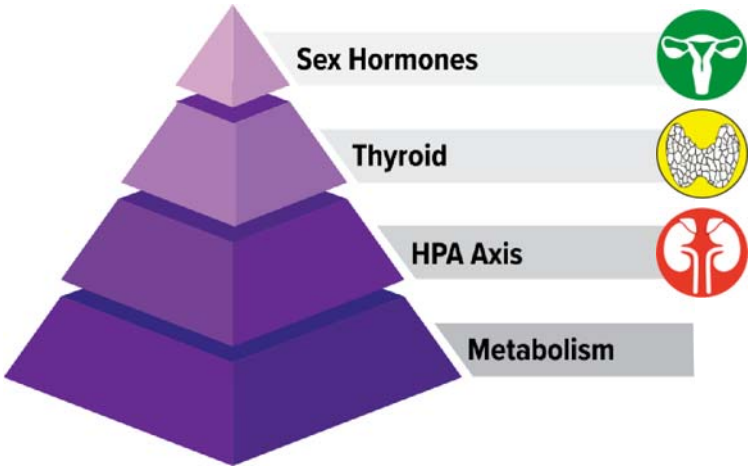
GENOVA DIAGNOSTICS

63 Zillicoa Street
Asheville, NC 28801

Patient: **SAMPLE PATIENT**
DOB:
Sex:
MRN:

4008 Endo+ - Serum, Saliva, Urine

Results Overview



Anabolic/Catabolic Balance

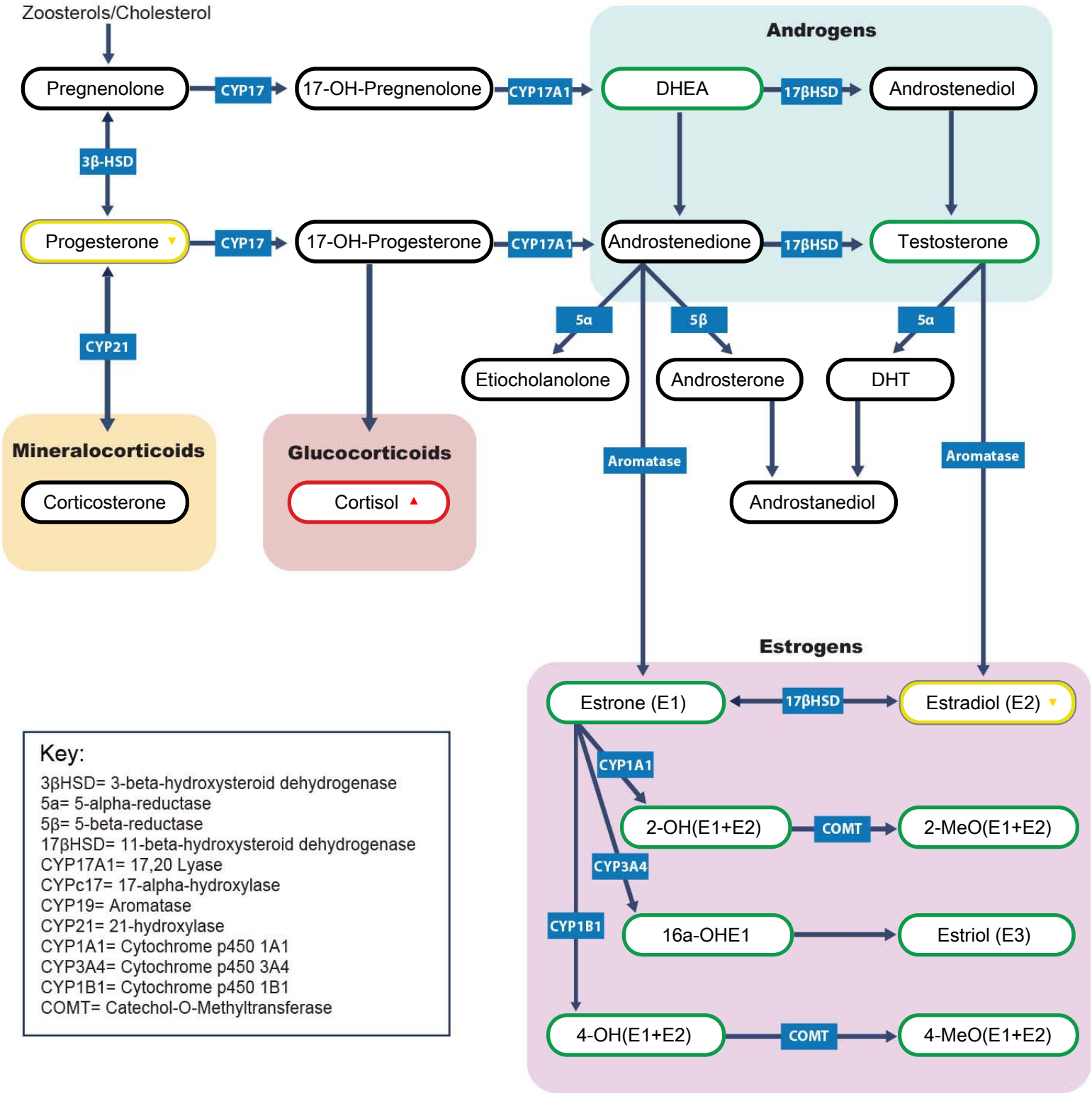
Estrogen Metabolism

Functional Imbalance Scores

Key (< 2) : Low Need for Support | (2-3) : Optional Need for Support | (4-6) : Moderate Need for Support | (7-10) : High Need for Support

	Need For HPA Axis Support	Need For Thyroid Support	Need For Sex Hormone Support	Need For Detoxification Support
	ADRENOCORTEX	THYROID	SEX HORMONES	ESTROGEN METABOLISM
	10	4	1	0
Biomarkers	Morning Cortisol ▲ Cortisol Curve ▲ DHEA ● DHEA:Cortisol ●	Thyroid Antibodies ▲ TSH ● FT3 ● rT3 ●	Progesterone ▽ Estradiol ▽ Estrone ● Testosterone ●	2-OH-E1/E2 ● 4-OH-E1/E2 ● 2/16 Ratio ● Methylation Activity ●
Therapeutic Support Options	• Stress Reduction • Mindfulness Training • HRV Biofeedback • Breathwork Training • Exercise/Yoga/Tai Chi • Herbal Adaptogens • Lower Blood Sugar • B-Vitamins • Phosphoserine • Glandulars	• Minerals: Se, Zn, Iron • Tyrosine • Adequate Iodine • Stress Reduction • Reduce Environmental Exposures • Antioxidant Support • Assess Medications • Smoking Cessation • Consider Thyroid HRT	• Address Thyroid Imbalance • Address HPA Axis • Phytoestrogens (for estrogen support) • Phytoandrogens (for testosterone support) • Consider Glandulars • Consider Hormone Precursors • Consider BHRT	• Methylation Support: B-Vitamins, Mg, Betaine • Cruciferous vegetables, berries, rosemary • Soy isoflavones, DIM, I3C • Bioflavanoids, Glutathione • Reduce sugar, stress, environmental toxicity • Reduce adiposity • Physical activity

Steroidogenic Pathway



Key:

3βHSD= 3-beta-hydroxysteroid dehydrogenase
5a= 5-alpha-reductase
5β= 5-beta-reductase
17βHSD= 11-beta-hydroxysteroid dehydrogenase
CYP17A1= 17,20 Lyase
CYPc17= 17-alpha-hydroxylase
CYP19= Aromatase
CYP21= 21-hydroxylase
CYP1A1= Cytochrome p450 1A1
CYP3A4= Cytochrome p450 3A4
CYP1B1= Cytochrome p450 1B1
COMT= Catechol-O-Methyltransferase

The results shown on pages 1 and 2 default to serum results except for DHEA. If no serum results are available, salivary results are used. If no salivary or serum results are available, urine results are used. For DHEA, salivary results are used unless none are available in which serum DHEA-S is used.

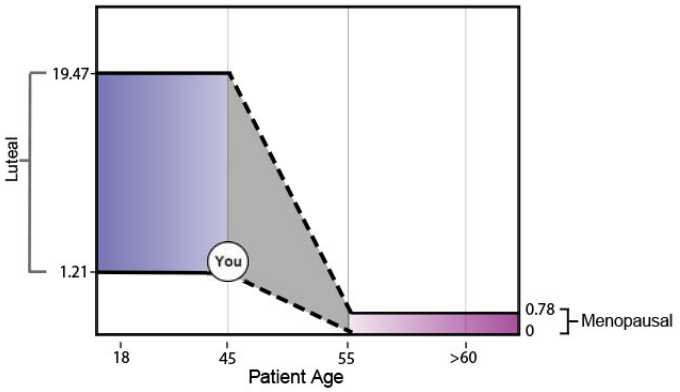
Endo+ Serum

Methodology: Chemiluminescent, RIA

Serum Sex Hormones

Progesterone

Progesterone



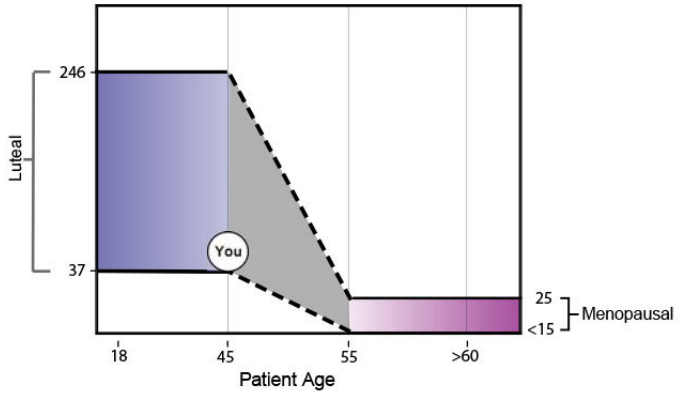
		Reference Range
Progesterone	2.13	See Chart Below

Androgens

DHEA-S	223	19-231 mcg/dL
Testosterone, Total	0.64	<0.10-0.75 ng/mL
Free Androgen Index	1.93	0.43-8.48

Estrogens

Estradiol (E2)



		Reference Range
Estradiol (E2)	57	See Chart Below

Estrone (E1)	132	43-250 pg/mL
Estriol (E3)	<80	<= 80 pg/mL

Binding Proteins

Sex Hormone Binding Globulin	114.7	18.2-135.5 nmol/L

Reference Range Information

Serum Analyte	Premenopause Luteal	Unsupplemented Menopause	Unsupplemented Male	Patient Result
Estrone (pg/mL)	43 - 250	18 - 63	46 - 143	132
Estradiol (pg/mL)	37 - 246	<15 - 25	<15 - 32	57
Estriol (pg/mL)	<=80	<=80	<=80	<80
Progesterone (ng/mL)	1.21 - 19.47	<=0.78	<=2.06	2.13
Testosterone (ng/mL)	<0.10 - 0.75	<0.10 - 0.75	1.75 - 7.81	0.64
Sex Hormone Binding Globulin (nmol/L)	18.2 - 135.5	16.8 - 125.2	13.3 - 89.5	114.7
Free Androgen Index	0.43 - 8.48	0.32 - 6.73	N/A	1.93

These reference ranges are based on luteal premenopausal samples. If patient is menopausal, refer to the chart above to determine the appropriate clinical ranges. Each individual is unique and evaluation of hormone status should be within the context of the patient's clinical picture.
The testosterone reference range is based on the manufacturer's range determined from women ages 21-73.

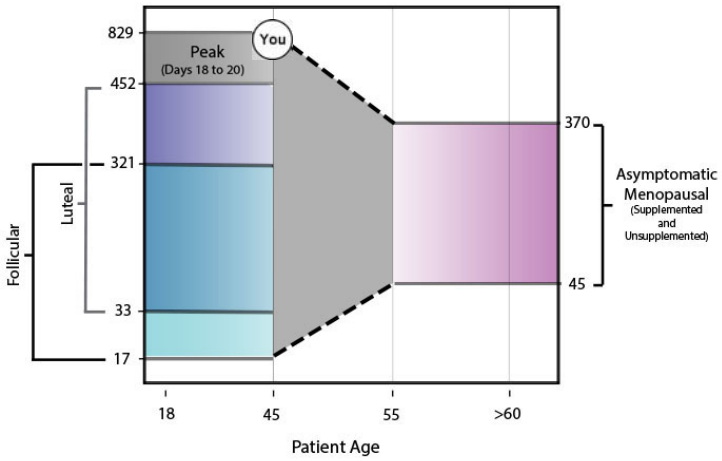
Endo+ Saliva

Methodology: EIA, LIA

Salivary Sex Hormones

Progesterone

Progesterone



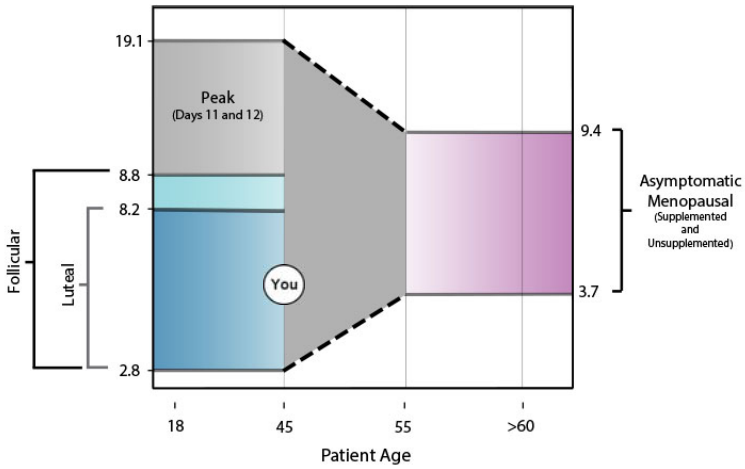
		Reference Range
Progesterone	780	See Chart Below

Androgens

DHEA ◆	289	71-640 pg/mL
Testosterone	70	34-148 pmol/L

Estrogens

Estradiol (E2)



		Reference Range
Estradiol (E2)	4.2	See Chart Below
Estrone (E1) * ◆	77.3	31.9-183.4 pmol/L
Estriol (E3) * ◆	<70	<= 133 pmol/L

**Reference ranges for Estrone and Estriol are based on menopausal patients.*

Reference Range Information

Saliva Analyte	Luteal	Follicular	Menopausal	Male	Patient Result
Estrone (pmol/L)	N/A	N/A	31.9 - 183.4	N/A	77.3
Estradiol (pmol/L)	2.8 - 8.2	2.8 - 8.8	3.7 - 9.4	3.1 - 7.4	4.2
Estriol (pmol/L)	N/A	N/A	<=133	N/A	<70
Progesterone (pmol/L)	33 - 452	17 - 321	45 - 370	31 - 280	780
Testosterone (pmol/L)	34 - 148	34 - 148	34 - 148	110 - 513	70
DHEA (pg/mL)	71 - 640	71 - 640	71 - 640	71 - 640	289

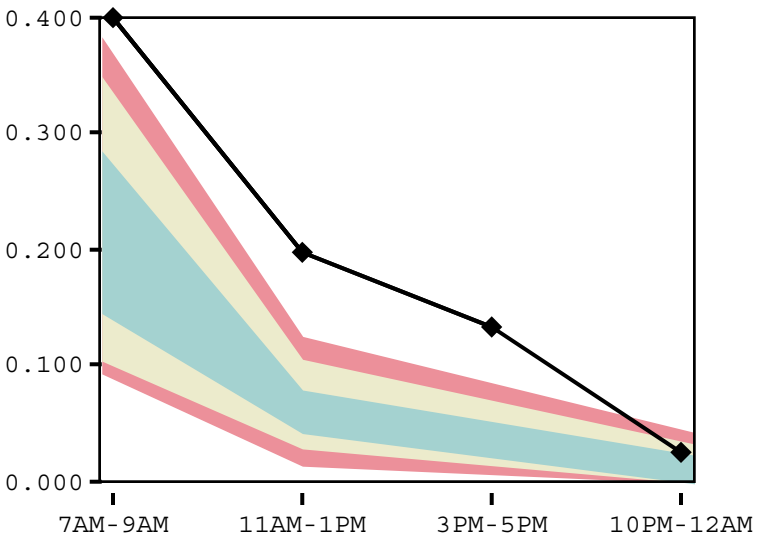
These reference ranges are based on luteal premenopausal samples. If patient is menopausal, refer to the chart above to determine the appropriate clinical ranges. Each individual is unique and evaluation of hormone status should be within the context of the patient's clinical picture.

Endo+ Adrenocortex

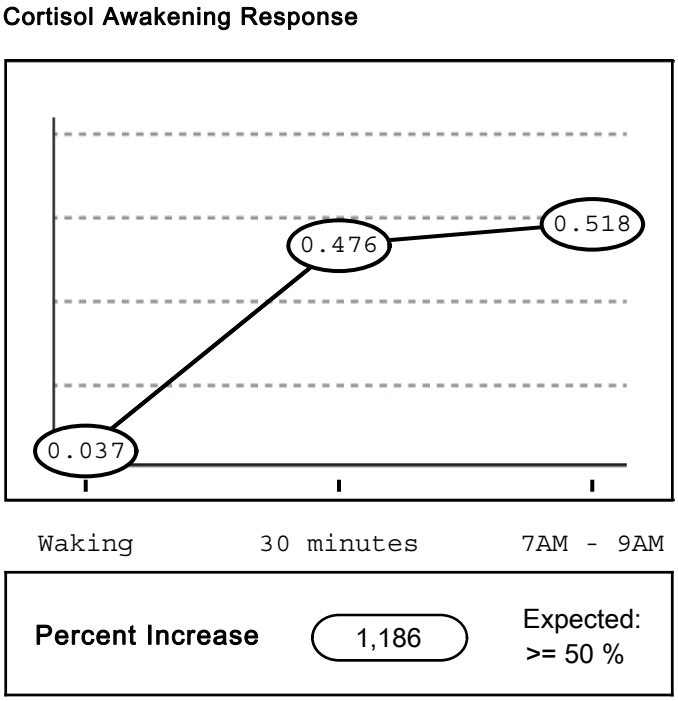
Methodology: EIA

Adrenocortex

Diurnal Cortisol Curve



Cortisol Awakening Response



Cortisol, Free (Salivary)

		Reference Range
7AM-9AM Cortisol	0.518	0.097-0.337 mcg/dL
11AM-1PM Cortisol	0.196	0.027-0.106 mcg/dL
3PM-5PM Cortisol	0.132	0.013-0.068 mcg/dL
10PM-12PM Cortisol	0.024	<= 0.034 mcg/dL

Androgens

		Reference Range
DHEA ◆	289	71-640 pg/mL
DHEA:Cortisol Ratio ◆	558	358-2,538

Results

Cortisol, Free (Salivary)	Waking Cortisol	30 minutes	Morning Cortisol 7AM-9AM**	Midday Cortisol 11AM-1PM**	Afternoon Cortisol 3PM-5PM**	Evening Cortisol 10PM-12AM**
Patient Result (mcg/dL) >>	**0.037**	**0.476**	**0.518**	**0.196**	**0.132**	**0.024**
Reference Range (mcg/dL) **Based on Collection Times	N/A	N/A	0.097-0.337	0.027-0.106	0.013-0.068	<=0.034
Actual Collection Time	3:35AM	4:05AM	7:42AM	12:42PM	4:55PM	10:03PM

Endo+ Urine

Methodology: LC/MS/MS

Estrogen Metabolism

Urine Estrogens

		Reference Range
Estrone (E1) ◆	7.5	2.0-26.2 mcg/g Creat.
Estradiol (E2) ◆	3.2	0.6-11.2 mcg/g Creat.
Estriol (E3) ◆	7.2	0.6-19.9 mcg/g Creat.

Phase 1 Estrogen Metabolites

		Reference Range
2-Hydroxyestrone+ ◆ 2-Hydroxyestradiol 2-OH(E1+E2)	15.3	1.3-36.3 mcg/g Creat.
16α-Hydroxyestrone ◆ 16a-OHE1	5.0	0.5-8.9 mcg/g Creat.
4-Hydroxyestrone+ ◆ 4-Hydroxyestradiol 4-OH(E1+E2)	1.7	<= 5.9 mcg/g Creat.

Enzyme Activity

2-OH(E1+E2)/ ◆ 16α-OHE1 Ratio	3.1	0.3-13.7
Methylation Activity ◆ 2-OH/2-MeO Ratio	3.3 → Poorer Methylation	1.6-10.7

Phase 2 Estrogen Metabolites

2-Methoxyestrone+ ◆ 2-Methoxyestradiol 2-MeO(E1+E2)	4.6	0.2-8.6 mcg/g Creat.
4-Methoxyestrone+ ◆ 4-Methoxyestradiol 4-MeO(E1+E2)	<dl	<= 1.0 mcg/g Creat.

Reference Range Information

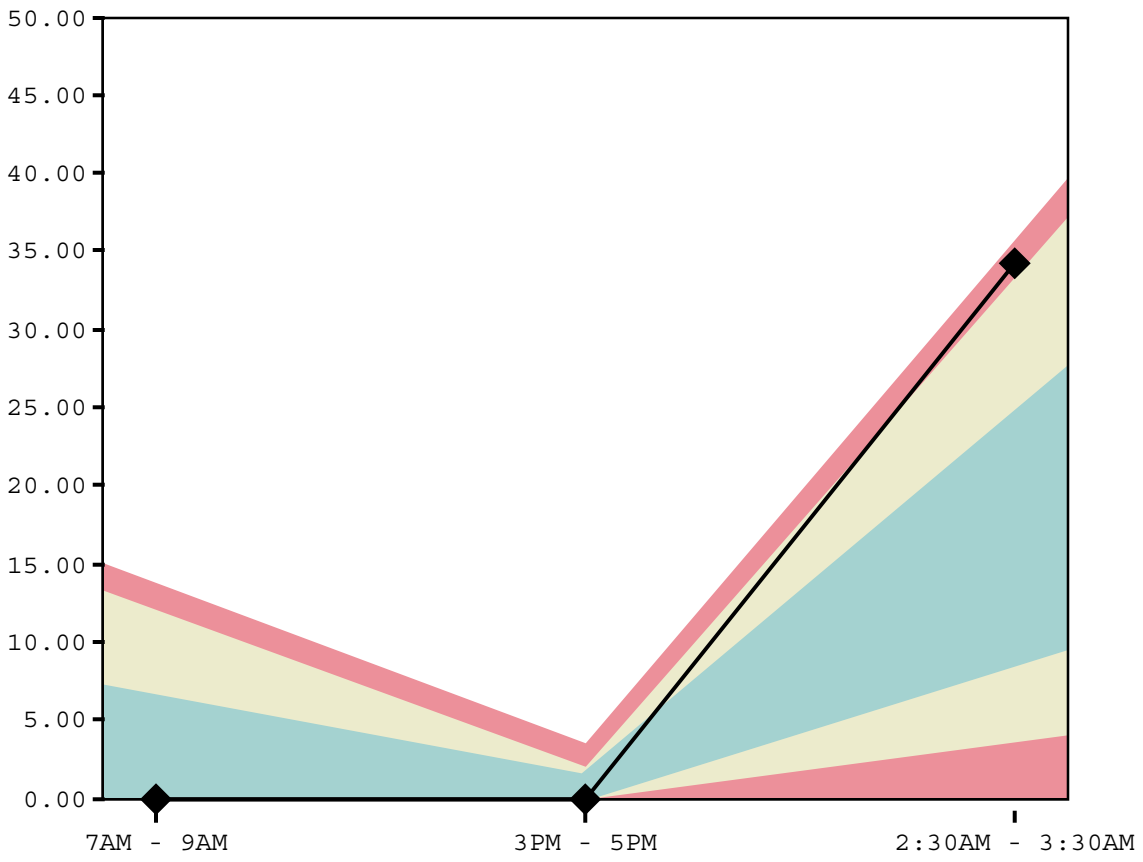
Urine Analyte	Premenopause Luteal	Menopause	Male	Patient Result
Estrone (mcg/g Creat.)	2 - 26.2	1.1 - 26.2	1.6 - 8.6	7.5
Estradiol (mcg/g Creat.)	0.6 - 11.2	0.6 - 15.4	0.8 - 4.3	3.2
Estriol (mcg/g Creat.)	0.6 - 19.9	0.7 - 30.8	0.3 - 5.1	7.2
2-OH(E1+E2) (mcg/g Creat.)	1.3 - 36.3	0.9 - 43.8	0.7 - 12.5	15.3
16a-OHE1 (mcg/g Creat.)	0.5 - 8.9	0.4 - 7.7	<=2.0	5.0
4-OH(E1+E2) (mcg/g Creat.)	<=5.9	<=8.8	<=1.6	1.7
2-MeO(E1+E2) (mcg/g Creat.)	0.2 - 8.6	0.3 - 5.9	0.2 - 2.5	4.6
4-MeO(E1+E2) (mcg/g Creat.)	<=1.0	<=1.0	<=1.0	<dl
2-OH(E1+E2)/16a-OHE1 Ratio	0.3 - 13.7	0.3 - 15.1	0.8 - 12.9	3.1
2-OH/2-MeO Ratio	1.6 - 10.7	0.4 - 11.6	1.0 - 8.8	3.3

These reference ranges are based on luteal premenopausal samples. If patient is menopausal, refer to the chart above to determine the appropriate clinical ranges. Each individual is unique and evaluation of hormone status should be within the context of the patient's clinical picture.

Endo+ Melatonin

Methodology: EIA

Salivary Melatonin



Results

Salivary Melatonin ♦	7AM-9AM**	3PM-5PM**	2:30AM-3:30AM**
Patient Result (pg/mL) >>	**<1.56**	**<1.56**	**34.17**
Reference Range (pg/mL) **Based on Collection Times	<=12.12	<=1.97	3.71-33.38

Endo+ Thyroid

Methodology: Chemiluminescent, RIA

Comprehensive Thyroid Assessment

Central Regulation

		Reference Range			Reference Range
Thyroid Stimulating Hormone (TSH)	1.69	0.40-2.50 microIU/mL	Free Thyroxine (FT4)	1.03	0.61-1.12 ng/dL

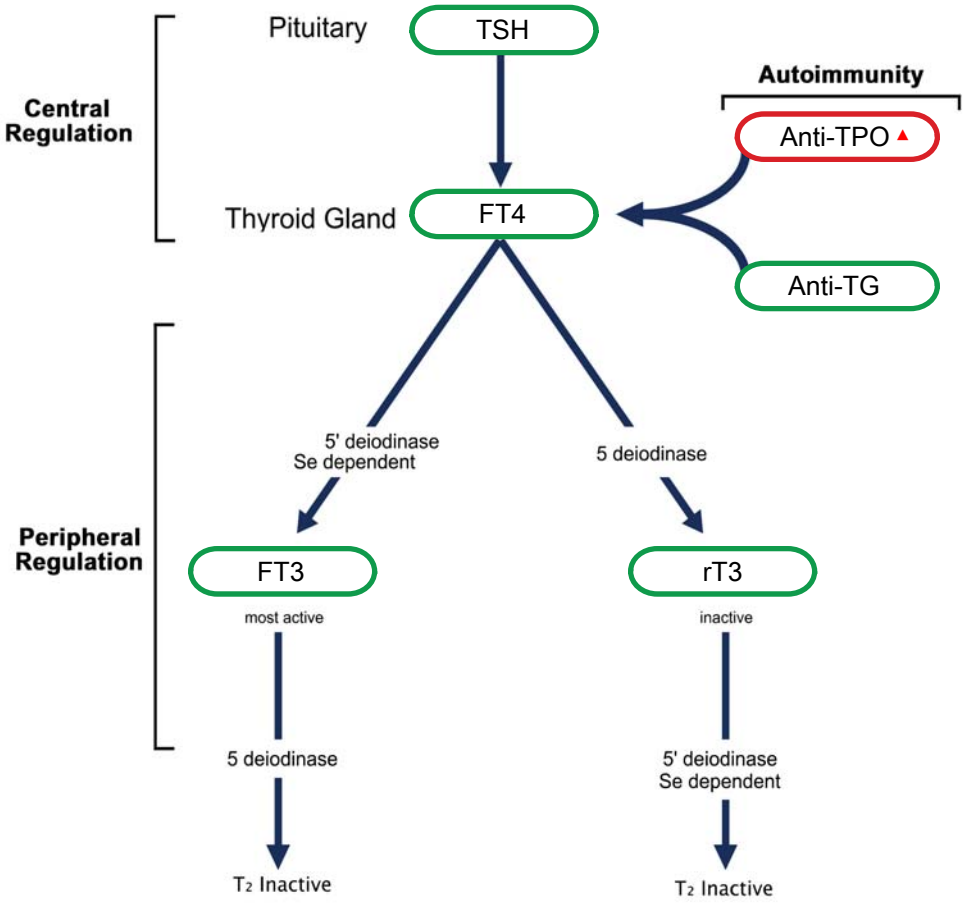
Peripheral Regulation

Free Triiodothyronine (FT3)	3.4	2.5-3.9 pg/mL	Reverse T3 (rT3) ◆	29.5	9.0-35.0 ng/dL

Thyroid Antibodies

Anti-Thyroglobulin Antibody (Anti-TG)	<0.9	<4.0 IU/mL	Anti-Thyroid Peroxidase (Anti-TPO)	19	<9 IU/mL

Thyroid Metabolism At-A-Glance



Additional Tests

Analyte		Reference Range
Secretory IgA (saliva) ♦	146	56-212 mcg/mL
Prolactin (serum) *	13.98	3.34-26.72 ng/mL
Androstenedione (serum) * † ♦	68	41-262 ng/dL
Luteinizing Hormone (serum) *	3.5	1.0-11.4 mIU/mL
Follicle Stimulating Hormone (serum) *	4.2	1.7-7.7 mIU/mL

* Please see commentary section for relevant testing location and reference range details.

† Reference ranges are age dependent.

Analyte	Premenopause Luteal	Unsupplemented Menopause	Male	Patient Results
Prolactin (ng/mL)	3.34 - 26.72	2.74 - 19.64	2.64 - 13.13	13.98
Luteinizing Hormone (mIU/mL)	1.0 - 11.4	7.7 - 58.5	1.7 - 8.6	3.5
FSH (mIU/mL)	1.7 - 7.7	25.8 - 134.8	1.5 - 12.4	4.2

These reference ranges are based on luteal premenopausal samples. If patient is menopausal, refer to the chart above to determine the appropriate clinical ranges. Each individual is unique and evaluation of hormone status should be within the context of the patient's clinical picture.

Commentary

Lab Comments
Testing performed at LabCorp was reported on 12/16/2023.

For more information regarding Endo+ clinical interpretation, please refer to the Endo+ Support Guide at **www.gdx.net/hormoneguide.**

The performance characteristics of all assays have been verified by Genova Diagnostics Inc. Assays are cleared by the U.S. Food and Drug Administration, unless otherwise noted with ♦.

Commentary is provided to the practitioner for educational purposes, and should not be interpreted as diagnostic or as treatment recommendations. Diagnosis and treatment decisions are the practitioner's responsibility.

<dl = Less than detection limit

Cortisol Awakening Response (CAR)

CAR is calculated by a direct percent increase: difference between 30 minutes and wake, divided by wake, then multiplied by 100. In literature, there are several ways to calculate CAR. Expected increases may differ depending on which calculation is used. Most literature demonstrates an expected increase of greater than 50% as a reflection of HPA axis resiliency.[1]

CAR represents the momentum of rising cortisol levels that begins several hours prior to awakening and an additional transient increase. The initial cortisol rise begins due to ACTH-mediated normal HPA axis activities with the additional CAR increase caused by supra-chiasmic nucleus (SCN) light activation.

CAR reflects a person's ability to cope with anticipated challenges and the perceptions of control around chronic stress. CAR is calculated based on the percent cortisol rise from awakening to 30 minutes. A value of approximately 50% is expected.

Approximately 25% of healthy adults do not mount a CAR, and are termed non-responders. Response is defined as an increase of at least 2.5 nmol/l (0.09 mcg/dL) above individual baseline. Any patient with a result less than this is considered a "non-responder" if sampling was performed correctly and the rest of the diurnal curve shows adequate cortisol response.

- Blunted CAR is seen in clinical burnout, self-reported health problems, early loss experiences, material hardship, depression, PTSD, and amnesia.
- Elevated CAR can be adaptive as a reflection of anticipation for daily stress. It may play a literal role in "preparing for action" by stimulating motor function, immunity responses, and alertness.
- If CAR is abnormal, and the rest of the diurnal pattern is not, then this would imply that a CAR-specific mechanism (SCN-related signaling) is implicated instead of a CRH or ACTH-mediated mechanism. Any abnormality of the hippocampus may blunt the CAR response and not affect the diurnal slope.

- If both the CAR and the diurnal rhythm are abnormal, this may represent a more general HPA dysfunction. It may also be useful to look at DHEA for a complete assessment of the HPA axis.

CAR treatment involves HPA axis and adrenal support using lifestyle modification, nutrition and adaptogens. However, insight into blunted or elevated CAR may help direct additional modalities such as behavioral modification and

Commentary

psychological therapies.

References:

1. Clow A, Thorn L, Evans P. Hucklebridge F. The awakening cortisol response: methodological issues and significance. *Stress.* 2004;7(1):29-37.
2. Stalder T, Kirschbaum C, Kudielka BM, et al. Assessment of the cortisol awakening response: Expert consensus guidelines. *Psychoneuroendocrino.* 2016;63:414-432.
3. Wust S, Wolf J, Hellhammer DH, Federenko I, Schommer N, Kirschbaum C. The cortisol awakening response-normal values and confounds. *Noise health.* 2000;2(7):79.
4. Fries E, Dettenborn L, Kirschbaum C. The cortisol awakening response (CAR): facts and future directions. *IntJPsychophysiol.* 2009;72(1):67-73.
5. Saxbe DE. A field (researcher's) guide to cortisol: tracking HPA axis functioning in everyday life. *Health Psychol Rev.* 2008;2(2):163-190.

Secretory immunoglobulin A (sIgA)

Methodology: Immunoturbidimetric

Secretory immunoglobulin A (sIgA) is the dominant immunoglobulin in external secretions that cover the mucosal surfaces (respiratory and gastrointestinal). It is a vital component of the immune system's "first line of defense" against pathogenic microorganisms. SIgA production is affected by a number of factors including stress, emotions, nutritional status, commensal bacteria, pathogens, and inflammation.

Elevated levels of salivary sIgA reflect an immune response to stimulation, such as stress, inflammation, and infection. Acute psychological stress, real and perceived, is associated with increases in sIgA concentration and secretion rate.

Lower salivary secretory IgA levels are seen in chronic stress or excessive exercise. Levels of salivary secretory IgA can decline with advanced age.

References:

1. Jarfarzadeh A, Sadeghi M, et.al. Salivary IgA and IgE levels in healthy subjects: relation to age and gender. *Braz Oral Res.* 2010;24(1):21-27.
2. Tsujita S, Morimoto K. Secretory IgA in saliva can be a useful stress marker. *Environ Health Prev Med.* 1999;4(1):1-8.
3. Phillips AC, Carroll D, Evans P, et al. Stressful life events are associated with low secretion rates of immunoglobulin A in saliva in the middle aged and elderly. *Brain Behav Immun.* 2006;20(2):191-197.
4. Engeland C, Hugo F, Hilgert J, et al. Psychological distress and salivary secretory immunity. *Brain Behav Immun.* 2016;52:11-17.
5. Fahlman MM, Engels HJ, Hall H. SIgA and Upper Respiratory Syndrome During a College Cross Country Season. *Sports Med Int Open.* 2017;1(06):E188-E194.

The Prolactin reference range was determined with serum samples from adult women.

Prolactin Testing Methodology: Chemiluminescent

Androstenedione Testing Methodology: Liquid chromatography/tandem mass spectrometry (LC/MS/MS)

Luteinizing Hormone Testing Methodology: Electrochemiluminescence immunoassay (ECLIA)

Commentary

Follicle Stimulating Hormone Testing Methodology: Electrochemiluminescence immunoassay (ECLIA)

Reference ranges for the following analytes provided by LabCorp:

- Androstenedione
- Luteinizing Hormone
- Follicle Stimulating Hormone

Testing for the following analytes was performed by LabCorp - Regional Lab and Center for Esoteric Testing (CET), 1447 York Court, Burlington, NC 27215:

- Androstenedione
- Luteinizing Hormone
- Follicle Stimulating Hormone

Optional Add-on

Genomic Results

GENOVATIONS™

63 Zillicoa Street
Asheville, NC 28801

Patient: **SAMPLE PATIENT**
DOB:
Sex:
MRN:

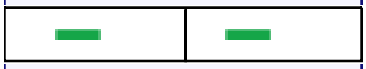
CYP1B1	*Cytochrome p450 1B1 : DETOXIFICATION*
Location: Chromosome 2 **L432V Your Genotype:**	Cytochrome P450 1B1 (CYP1B1) is a Phase I detoxification enzyme responsible for the 4-hydroxylation of estrogen as well as the activation of common environmental toxins such as polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), and aflatoxin B1.
+ ▲ | -	**Health Implications** · Hyper-induction of CYP1B1 upon exposure to its substrates or inducers · Increased production of 4-hydroxyestrogens and potentially carcinogenic compounds · Possible increased risk of breast cancer, especially in women smokers, those exposed to waste incinerator or agricultural pollutants, and women on HT for 4 years or longer (studies are mixed) · Possible increased risk of cancer of the ovary, uterus, prostate, or lung (studies are mixed) · CYP1B1 polymorphisms are generally associated with higher potential for altered hormone levels and greater menopausal symptoms
N453S Your Genotype:	**Clinical Management Considerations** · Minimize exposure to xenobiotics (e.g., PAHs) and xenoestrogens (e.g., organochlorines), which increase CYP1B1 activity · Maintain a diet rich in antioxidants (colorful fruits and vegetables); consider supplementation · Consider redirecting estrogen metabolism away from 4-hydroxylation using cruciferous vegetables and/or agents such as indole 3-carbinol (I3C), diindolylmethane (DIM), fish oils, or rosemary · Caution using long-term estrogen therapy, especially conjugated equine estrogens, which are preferentially 4-hydroxylated; combined estrogen/progestin therapy produces greatest breast density in carriers of the SNP · Carcinogen-induced DNA damage may be minimized by agents such as curcumin, black cohosh, genistein, and DHEA
-	

Key

- \- Neither chromosome carries the genetic variation.
+ \- One chromosome (of two) carries the genetic variation.
+ + Both chromosomes carry the genetic variation.
(You inherit one chromosome from each parent)

+ ▲ Gene activity increased
+ ▼ Gene activity decreased

GENOVA DIAGNOSTICS

MTHFR	*5,10-methyltetrahydrofolate reductase : METHYLATION*
Location: Chromosome 1 **C677T Your Genotype:**	5,10-methylenetetrahydrofolate reductase (MTHFR) is a key enzyme in folate metabolism, facilitating the formation of methyltetrahydrofolate, a required cofactor in the remethylation of homocysteine (Hcy) to methionine.
	**Health Implications** · Heterozygosity for only 1298 (-/+) is associated with baseline "normal" MTHFR enzyme activity, suggesting efficient formation of methyl-THF · Risk of methylation impairment and elevated homocysteine is increased only when C677T polymorphism is also positive
A1298C Your Genotype:	**Clinical Management Considerations** · Ensure adequate intake of dark-green leafy vegetables and other B vitamin-rich foods
	

COMT — Catechol-O-MethylTransferase : METHYLATION

Location:
Chromosome 22.11q
V158M
Your Genotype:

Catechol-O-Methyltransferase (COMT) is a key enzyme involved in the deactivation of catechol compounds, including catecholamines, catechol estrogens, catechol drugs such as L-DOPA, and catechol metabolites of various chemicals and toxins, such as aryl hydrocarbons.

Health Implications

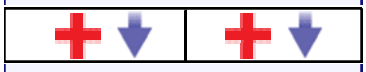
- Most common genotype in individuals of European descent
- Moderately reduced COMT enzyme activity, resulting in slightly impaired methylation
- Strong cognitive stability, e.g., ability to focus (due to higher brain dopamine) but lower cognitive flexibility (e.g., ability to adapt to external changes), compared to the (-/-) genotype
- Cognitive benefit may increase as dopamine levels decline with age
- Acute or chronic stress may increase risk of nervousness and anxiety (esp. when history of childhood trauma), due to higher baseline levels of catecholamines
- Past studies have suggested increased breast cancer risk under certain conditions; however, larger and more recent studies have not confirmed these findings
- Moderately reduced pain threshold, exacerbated by one's experience of pain; slightly increased risk of fibromyalgia and chronic pain syndromes

Clinical Management Considerations

- Ensure adequate B6, B12, folate, magnesium, and methionine to support formation of S-adenosylmethionine and prevent elevated homocysteine; S-adenosylhomocysteine inhibits COMT
- Exercise caution using conjugated equine estrogens such as Premarin®; in-vitro studies suggest show one of its metabolites to inhibit COMT
- Individuals with this genotype might have a superior response to SSRI antidepressants (mixed studies)
- In children with ADHD, methylphenidate (Ritalin®) may be less effective (mixed studies)

VDR — Vitamin D Receptor : HORMONAL BONE FORMATION

Location:
Chromosome 12
BsmI RFLP
Your Genotype:



VDR is an intracellular hormone receptor that specifically binds active vitamin D (calcitriol), interacting with target-cell nuclei to produce effects in a wide variety of biological processes. Due to the extensive functions of vitamin D, VDR polymorphisms may play a role in a range of conditions including osteopenia/osteoporosis, inflammatory states, autoimmune disorders, certain cancers, metabolic syndrome, and coronary artery disease.

Health Implications

- Impaired vitamin D receptor function
- Reduced calcium absorption efficiency with low calcium intake, especially if intake <300 mg/day
- Higher rate of bone turnover, lower bone mineral density (BMD), increased risk of fracture, and increased risk of bone lead accumulation following exposure
- Possible reduced risk of breast, prostate and colorectal cancer

Clinical Management Considerations

- Ensure adequate calcium intake; this genotype is the most sensitive to low calcium status
- These individuals benefit the most from vitamin D supplementation
- Vitamin K may help to compensate for the higher risk of bone loss
- Caffeine intake >300 mg/day may accelerate bone loss, especially with lower calcium intake
- Most favorable bone response to raloxifene and least beneficial response to bisphosphonates and HT

Lab Comments

Testing performed at LabCorp was reported on 12/16/2023.

This test has been developed and its performance characteristics determined by Genova Diagnostics, Inc. It has not been cleared by the U.S. Food and Drug Administration.

Commentary is provided to the practitioner for educational purposes, and should not be interpreted as diagnostic or treatment recommendations. Diagnosis and treatment decisions are the responsibility of the practitioner.

The accuracy of genetic testing is not 100%. Results of genetic tests should be taken in the context of clinical representation and familial risk. The prevalence and significance of some allelic variations may be population specific.

Any positive findings in your patient's test indicate genetic predisposition that could affect physiologic function and risk of disease. We do not measure every possible genetic variation. Your patient may have additional risk that is not measured by this test. Negative findings do not imply that your patient is risk-free.

DNA sequencing is used to detect polymorphisms in the patient's DNA sample. The sensitivity and specificity of this assay is <100%.