



Hormone & Urinary Metabolites Assessment Profile



Order: 999999-9999



Test: X999999-9999-1

Client #: 999999

Doctor: Sample Doctor, MD

Doctors Data Inc

123 Main St.

St. Charles, IL 60174 USA

Patient: Sample Patient

Id: 999999

Age: 33 DOB: 01/01/1991

Sex: Female

Menopausal Status: Pre-menopausal,
LMP: 10/16/2024,

Sample Collection Date/Time

Midsleep 11/11/2024 02:30

Dinnertime 11/10/2024 18:00

Bedtime 11/10/2024 21:36

Waking 11/11/2024 08:25

2 Hr. Post Waking 11/11/2024 10:55

Collection Period Multipoint daily

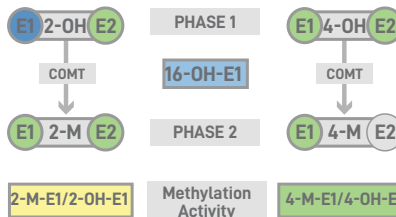
Date Received 11/14/2024

Date Reported 11/21/2024

ESTROGENS

The bar graph represents the relationship of the catechol estrogens (2-OH-E1, 4-OH-E1, 16-OH-E1) to each other. The expected percentage for each is represented by the shaded area.

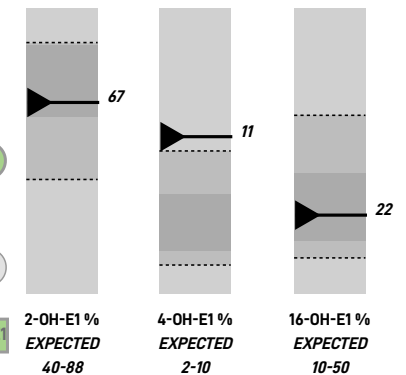
The pathway illustrates phase 1 and phase 2 metabolism of both E1 and E2. Phase 1 metabolites, also known as catechol estrogens, are active and can induce estrogenic actions. Phase 2 metabolism gives insight into a patient's ability to methylate, or potentially inactivate harmful metabolites.



2-OH: generally considered safest

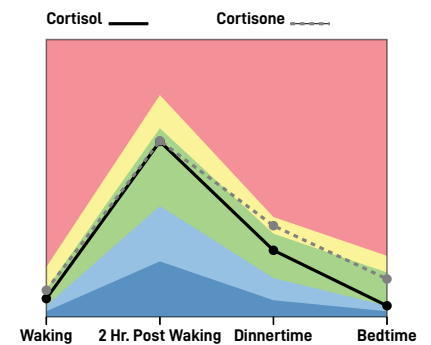
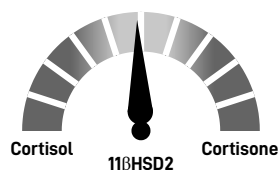
4-OH: potential for DNA damage

16-OH: considered highly estrogenic



CORTICOIDS

11βHSD2 is responsible for the conversion of cortisol to cortisone. Inhibition of this enzyme may lead to the amount of cortisol being greater than cortisone, while increased enzyme activity can lead to higher levels of cortisone in comparison to cortisol.



KEY RELATIONSHIPS

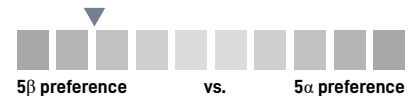
The graphs to the right represent metabolism preference by key enzymes, indicated by the arrow.

Metabolites in the 5-alpha pathway are more androgenic than their 5-beta counterparts and can be responsible for androgenic symptoms even when hormone levels appear normal.

Aromatase is an enzyme found in the greatest amounts in peripheral fat tissue which can increase estrogens in both males and females.

4-OH-E1 is considered unfavorable due to its carcinogenic potential within breast and prostatic tissue as a reactive metabolite. When methylated by COMT, this reactive metabolite becomes stable and can be removed from the body.

5-A REDUCTASE ACTIVITY

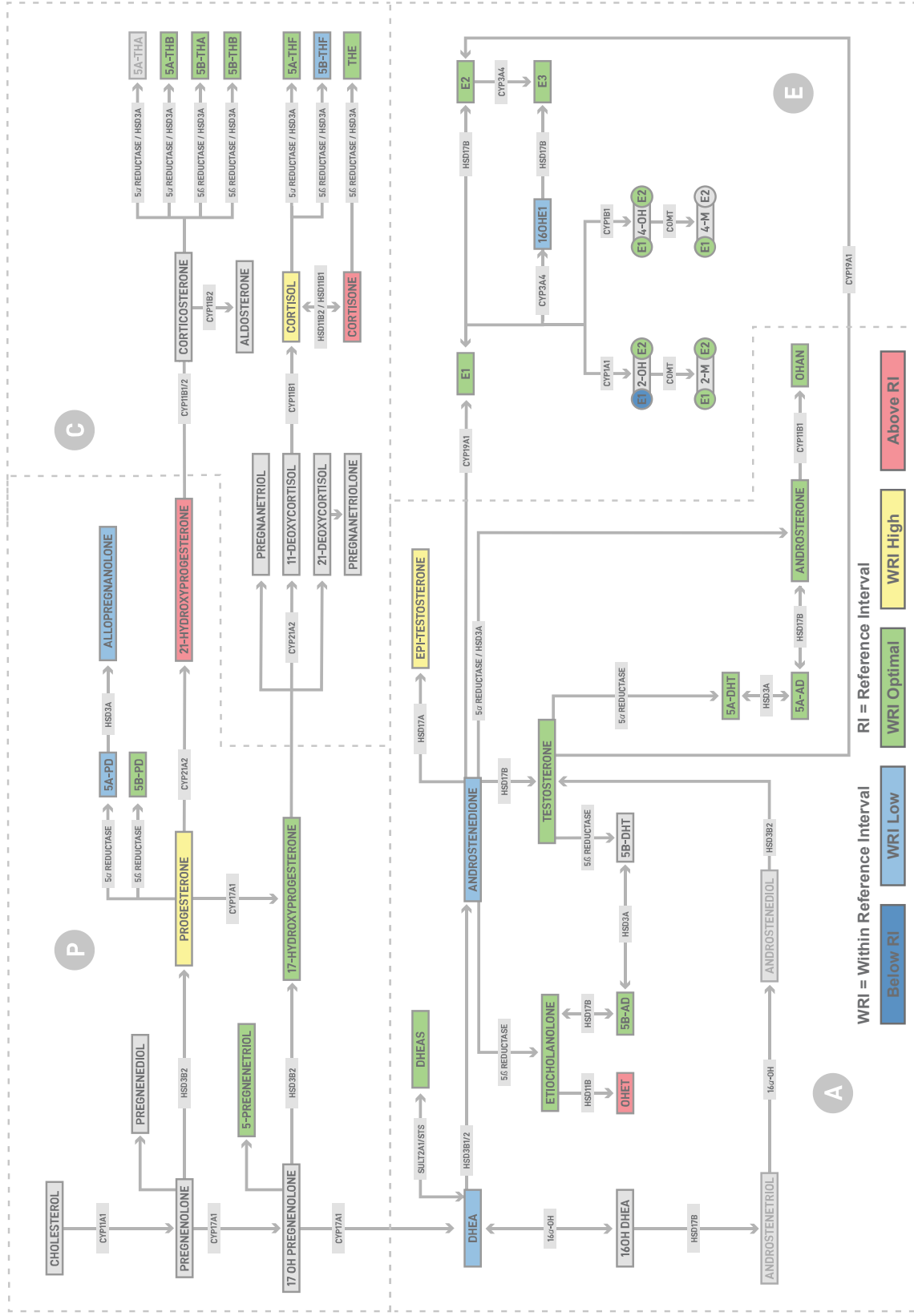


AROMATASE/CYP19A1 ACTIVITY



COMT/METHYLATION ACTIVITY







Progesterone Metabolites; urine



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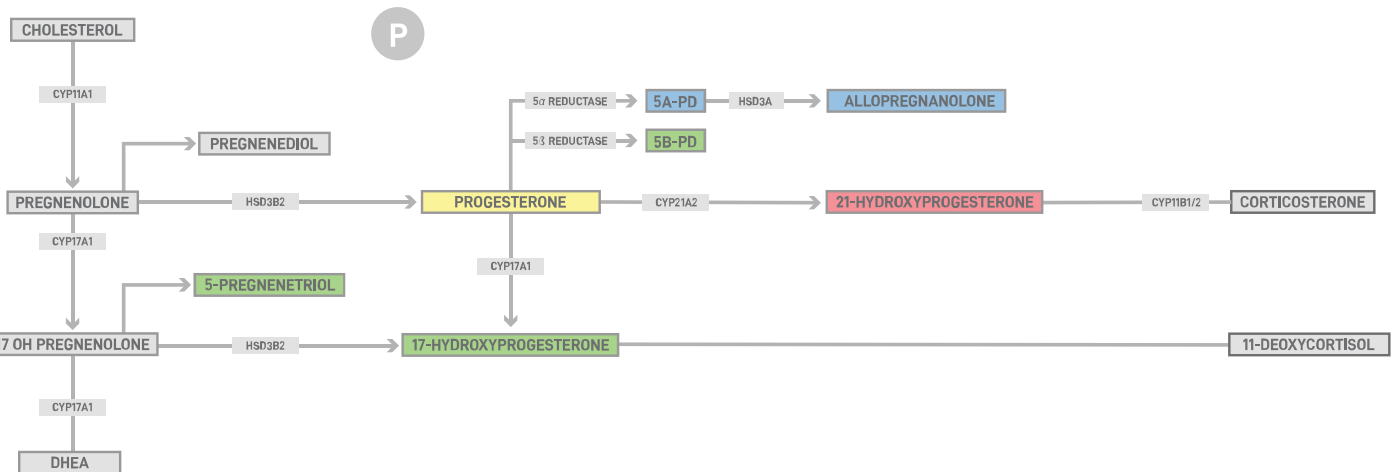
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Progesterones		Result	Unit	L	WRI	H	Reference Interval
Progesterone [‡]	(P4)	0.72	ng/mg Creat/Day				0.10 – 1.10
5α-Pregnanediol [‡]	(5A-PD)	144	ng/mg Creat/Day				30 – 405
5β-Pregnanediol [‡]	(5B-PD)	2250	ng/mg Creat/Day				300 – 2700
Allopregnanolone [‡]	(ALLOP)	28	ng/mg Creat/Day				3.3 – 110
21-Hydroxyprogesterone [‡]	(21-OHP)	0.86	ng/mg Creat/Day				0.10 – 0.80
17-Hydroxyprogesterone [‡]	(17-OHP)	0.39	ng/mg Creat/Day				0.15 – 1.3
5-pregnenetriol [‡]	(5-PT)	91	ng/mg Creat/Day				70 – 245

Ratios and Calculations		Result	Unit	L	WRI	H	Reference Interval
5A-PD:5B-PD [‡]	(alpha vs beta metabolism)	0.064					0.06 – 0.24



Progesterone Metabolites Information

Progesterone is excreted in urine in small quantities. Majority of progesterone is metabolized to 5β-pregnanediol (typically highest), 5α-pregnanediol, and subsequently to allopregnanolone. This test measures progesterone and its metabolites. Allopregnanolone concentrations are useful in the context of oral progesterone use due to its GABA-like effects for sleep and anxiety relief. 17-hydroxyprogesterone and 21-hydroxyprogesterone results are also reported. They reflect endogenous cortisol and corticosterone production.

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Methodology: LCMS QQQ



Adrenal Corticoid Metabolites; urine



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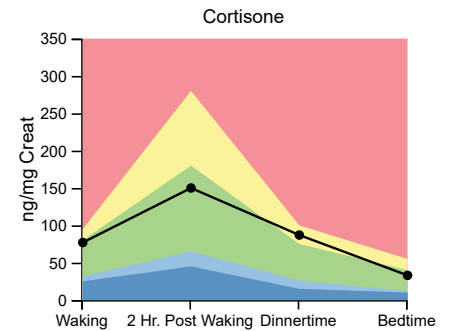
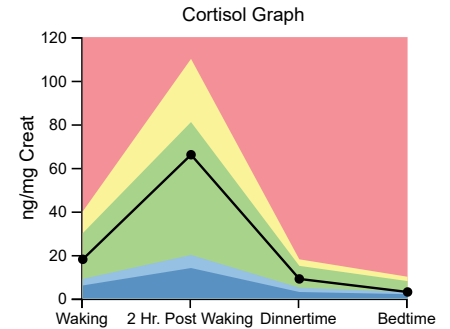
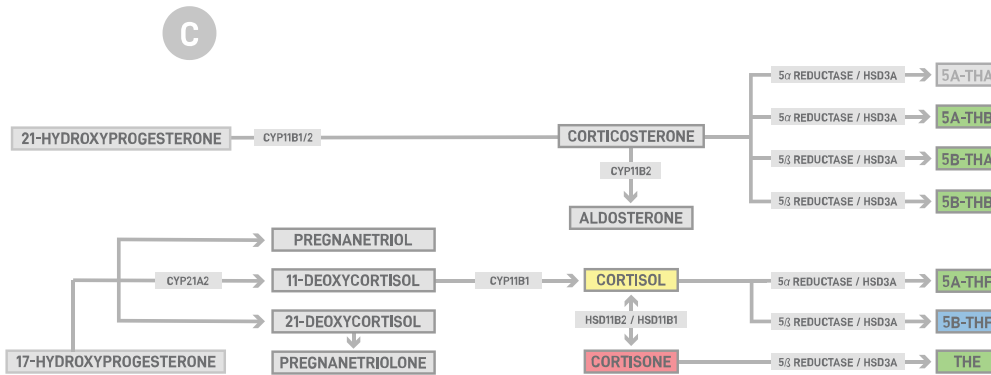
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Free Cortisol and Cortisone	Result	Unit	L	WRI	H	Reference Interval
Cortisol Waking [‡]	18	ng/mg Creat				6 – 40
Cortisol Waking+2hrs [‡]	66	ng/mg Creat				14 – 110
Cortisol Dinnertime [‡]	9	ng/mg Creat				3 – 18
Cortisol Bedtime [‡]	3	ng/mg Creat				2 – 10
Cortisol/day [‡]	(F) 33	ng/mg Creat/Day				9 – 35
Cortisone Waking [‡]	77	ng/mg Creat				25 – 95
Cortisone Waking+2hrs [‡]	150	ng/mg Creat				45 – 280
Cortisone Dinnertime [‡]	87	ng/mg Creat				15 – 100
Cortisone Bedtime [‡]	33	ng/mg Creat				10 – 55
Cortisone/day [‡]	(E) 97	ng/mg Creat/Day				30 – 95
Creatinine Waking	91.5	mg/dL				30 – 225

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Creatinine Waking+2hrs		115	mg/dL				30 – 225
Creatinine Dinnertime		54.3	mg/dL				30 – 225
Creatinine Bedtime		38.3	mg/dL				30 – 225
Creatinine/day		75.5	mg/dL/Day				30 – 225
Corticoid Metabolites and DHEA		Result	Unit	L	WRI	H	Reference Interval
Tetrahydrodehydrocorticosterone [‡]	(5B-THA)	90	ng/mg Creat/Day				40 – 130
5β-Tetrahydrocorticosterone [‡]	(5B-THB)	120	ng/mg Creat/Day				58 – 240
5α-Tetrahydrocorticosterone [‡]	(5A-THB)	210	ng/mg Creat/Day				90 – 380
5α-Tetrahydrocortisol [‡]	(5A-THF)	502	ng/mg Creat/Day				450 – 1300
5β-Tetrahydrocortisol [‡]	(5B-THF)	812	ng/mg Creat/Day				720 – 2050
Tetrahydrocortisone [‡]	(THE)	2580	ng/mg Creat/Day				1650 – 4000
Dehydroepiandrosterone [‡]	(DHEA)	17	ng/mg Creat/Day				15 – 190
Dehydroepiandrosterone Sulfate [‡]	(DHEAS)	600	ng/mg Creat/Day				45 – 3000
Ratios and Calculations		Result	Unit	L	WRI	H	Reference Interval
DHEA+DHEAS [‡]		620	ng/mg Creat/Day				50 – 2000
THE+5A-THF+5B-THF [‡]	(Metabolized Cortisol)	3890	ng/mg Creat/Day				2600 – 7200
5A-THF+5B-THF/THE [‡]	(Cortisol/Cortisone Metabolites)	1					0.6 – 1.2
Cortisol/Cortisone [‡]	(11B HSD activity)	0.34					0.18 – 0.60
5A-THF/5B-THF ratio [‡]	(alpha vs beta metabolism)	0.62					0.19 – 0.82



Adrenal Corticoid Metabolites Information

Under stress, the HPA axis controls the secretion of cortisol from the adrenal cortex. In saliva and blood, cortisol levels are the highest 30 minutes after waking and gradually decline throughout the day (measured by "cortisol awakening response" – CAR). When testing cortisol in urine throughout the day, highest value is typically seen during the second timed collection. Adrenal corticoid page provides four different aspects of cortisol metabolism and excretion: graphical pattern of cortisol and cortisone excretion, average cortisol and cortisone per day, metabolized cortisol, and metabolic preference for cortisol or cortisone. Cortisol and cortisone output is graphed in a diurnal pattern over the course of the day. Metabolized cortisol calculation includes the daily metabolites of cortisol (5A-THF, 5B-THF) and cortisone (THE) which may be a better representation of daily cortisol output than measuring cortisol and cortisone alone.

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Methodology: LCMS QQQ





Androgen Metabolites; urine



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Androgens		Result	Unit	L	WRI	H	Reference Interval
11-hydroxy-Etiocholanolone [‡]	(OHET)	504	ng/mg Creat/Day				40 – 470
5β-Androstenediol [‡]	(5B-AD)	33	ng/mg Creat/Day				9.0 – 110
Dehydroepiandrosterone [‡]	(DHEA)	17	ng/mg Creat/Day				15 – 190
Dehydroepiandrosterone Sulfate [‡]	(DHEAS)	600	ng/mg Creat/Day				45 – 3000
Ratios and Calculations		Result	Unit	L	WRI	H	Reference Interval
DHEA+DHEAS [‡]		620	ng/mg Creat/Day				50 – 2000
Androsterone (5α) / Etiocholanolone (5β) [‡]	(5α Reductase Activity)	0.52					0.5 – 1.4
Testosterone / EPI-Testosterone [‡]		0.19					0.1 – 2.0



Androgen Metabolites Information

Androgens play a significant role in structure and function of muscle, bone, and connective tissue, metabolic homeostasis and reproduction in both men and women. When evaluating the androgens, it is important to look at unconjugated hormones, enzymes, metabolites, and clinical symptoms to gain an understanding of the complete clinical picture. The key areas of focus within the androgen pathway are androstenedione, DHEA, testosterone, 5-alpha and 5-beta reductase, and aromatase (CYP19). Monitoring 5-alpha vs 5-beta activity is of particular interest as 5-alpha metabolites are more androgenic. Symptoms associated with higher androgen levels are often seen when levels of 5-alpha reductase and its corresponding metabolites are elevated. 5-beta reductase and its corresponding metabolites are much less androgenic.

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Methodology: LCMS QQQ



Estrogen Metabolites; urine



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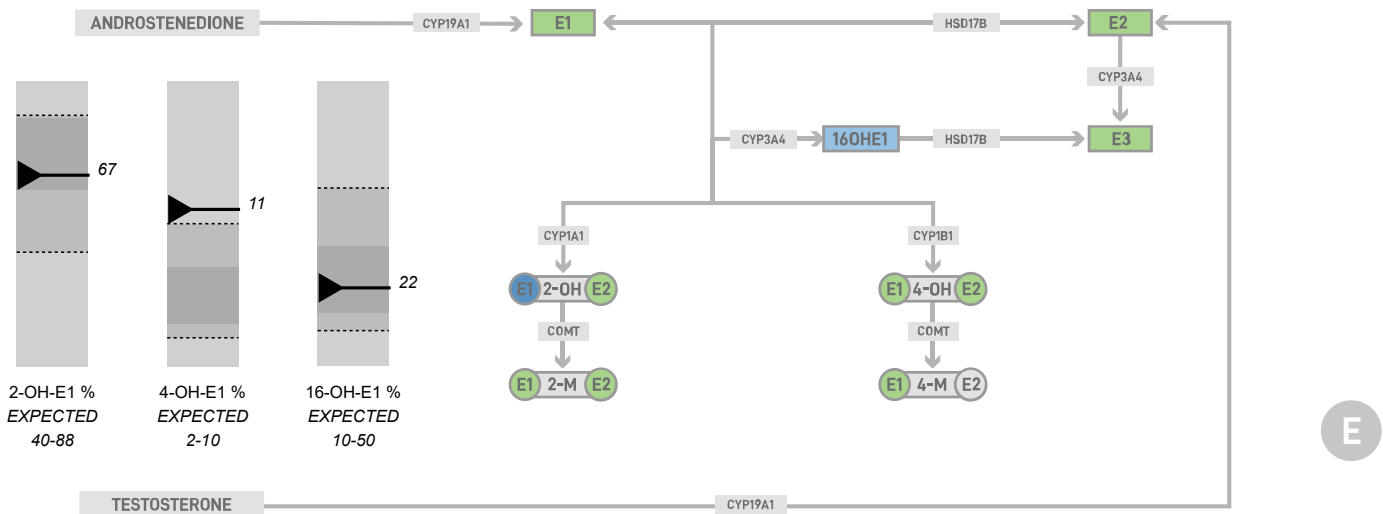
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Estrogens	Result	Unit	L	WRI	H	Reference Interval
Estrone [‡]	(E1) 7.9	ng/mg Creat/Day				3.8 – 22
2-Hydroxyestrone [‡]	(2-OH-E1) 10	ng/mg Creat/Day				13 – 34
4-Hydroxyestrone [‡]	(4-OH-E1) 1.6	ng/mg Creat/Day				0.0 – 2.9
16α-Hydroxyestrone [‡]	(16-OH-E1) 3.4	ng/mg Creat/Day				1.4 – 15
2-Methoxyestrone [‡]	(2-M-E1) 2.4	ng/mg Creat/Day				1.0 – 7.10
4-Methoxyestrone [‡]	(4-M-E1) 0.015	ng/mg Creat/Day				0.005 – 0.060
Estradiol [‡]	(E2) 3.7	ng/mg Creat/Day				1.5 – 13
2-Hydroxyestradiol [‡]	(2-OH-E2) 1.7	ng/mg Creat/Day				0.80 – 3.9
4-Hydroxyestradiol [‡]	(4-OH-E2) 0.54	ng/mg Creat/Day				0.0 – 1.2
2-Methoxyestradiol [‡]	(2-M-E2) 0.24	ng/mg Creat/Day				0.06 – 0.70
Estriol [‡]	(E3) 11	ng/mg Creat/Day				2.8 – 23

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Ratios and Calculations		Result	Unit	L	WRI	H	Reference Interval
2-OH-E1 % [‡]	(2-OH-E1 %)	67	%				40 – 88
4-OH-E1 % [‡]	(4-OH-E1 %)	11	%				2 – 10
16-OH-E1 % [‡]	(16-OH-E1 %)	22	%				10 – 50
2-M-E1:2-OH-E1 [‡]	(COMT/Methylation activity)	0.22					0.08 – 0.60
2-M-E2:2-OH-E2 [‡]	(COMT/Methylation activity)	0.13					0.06 – 0.80
4-M-E1:4-OH-E1 [‡]	(COMT/Methylation activity)	0.0087					0.004 – 0.10
2-OH-E1:16-OH-E1 [‡]		3.0					≥ 0.70
4-OH-E1:2-OH-E1 [‡]		0.16					0.00 – 0.17
Oxidative Stress Metabolite		Result	Unit	L	WRI	H	Reference Interval
8-hydroxy-2'-deoxyguanosine [‡]	(8-OHdG)	5.7	ng/mg Creat/Day				0.0 – 7.5



Estrogen Metabolites Information

Evaluation of the estrogen metabolism pathway relies on understanding several key steps of metabolism: the amount of unconjugated estrogens, hydroxylation of E1 and E2 (phase I), methylation of hydroxy estrogens (phase II), and the function of key enzymes. Estrogen is metabolized down three phase I pathways: 2-OH (considered the safest), 4-OH (considered the most genotoxic), and 16-OH (considered the most estrogenic). In phase II, estrogens are methylated, making them less reactive and ready for excretion. The ratio of 4-M E1/E2 to 4-OH E1 / 2 and 2-M E1/E2 to 2-OH E1/E2 can help determine if adequate methylation of catechol estrogens is occurring. The higher the ratio, the higher the likelihood of metabolizing toward the pathway with lower harm potential, and therefore less reactive quinone formation. Even if 4-OH metabolites are elevated, adequate methylation can indicate these metabolites are being detoxified, rendering them potentially less harmful.

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Progesterones

↑ 21-OH Progesterone (21-OHP)

21-Hydroxyprogesterone is a steroid hormone with mineralocorticoid properties produced in the adrenal gland which serves as a precursor hormone to aldosterone. Elevated levels may not be clinically significant on their own, but could lead to mineralocorticoid hypertension. Elevations have been associated with chronic exposure to ACTH, Cushing's disease, type 2 diabetes, congenital adrenal hyperplasia or rarely adrenocortical carcinoma.

Androgens

↑ 11-hydroxy-Etiocholanolone (OHET)

OHET is the product of cortisol metabolism as well as 11-oxygenated androgens produced from the adrenal gland. Levels tend to reflect levels of etiocholanolone.

Corticoids

↑ Cortisone

Cortisone is the inactive form of cortisol. Elevations of cortisone may reflect high cortisol production, excessive 11B-HSD2 activity, or insufficient conversion by 11B-HSD1.

Estrogens

↓ 2-Hydroxyestrone (2-OH-E1)

Adequate levels of 2-OH-E1 have been shown to be a favorable marker for breast health. Low levels of 2-OH E1 may be due to low levels of estrone, or more active CYP3A4 or CYP1B1 enzymes. Increasing the activity of CYP1A1 to increase 2-OH-E1 is a consideration.