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: 55 **DOB:** 01/01/1967
Sex: Female **Body Mass Index:** 20
Menopausal Status: Post-menopausal ,
Supplements: DHEA,E2,E3,P4

Sample Collection	Date/Time
Midsleep	05/13/2023 00:14
Dinnertime	05/12/2023 18:00
Bedtime	05/12/2023 20:35
Waking	05/13/2023 04:30
2 Hr. Post Waking	05/13/2023 06:30
Collection Period	Multipoint daily
Date Received	05/15/2023
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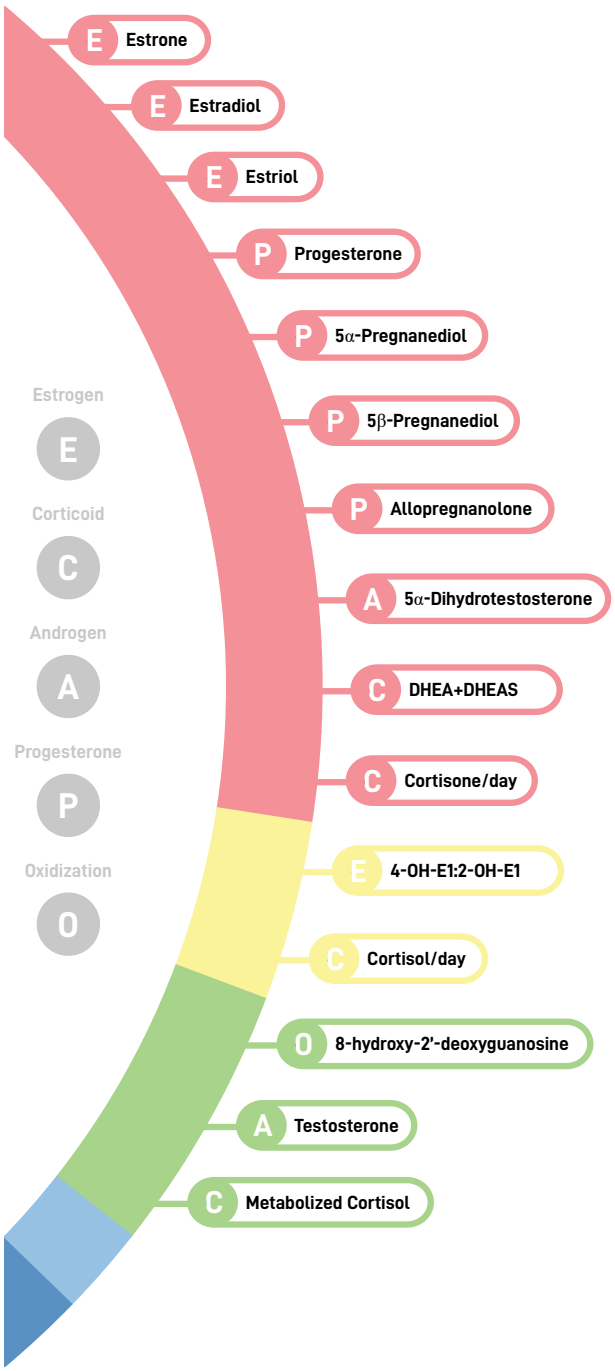
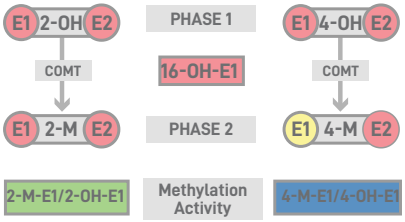


ESTROGENS

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.

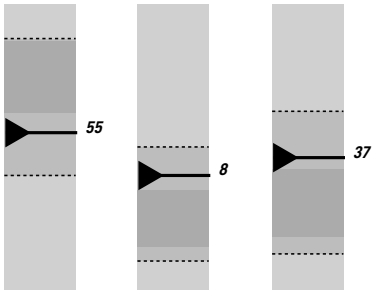
The pathway illustrates phase 1 and phase 2 metabolism of both E1 and E2. Phase 1 metabolites, also known as catechol estrogens, are active and can induce estrogenic actions. Phase 2 metabolism gives insight into a patient's ability to methylate, or potentially inactivate harmful metabolites.



2-OH: *generally considered safest*

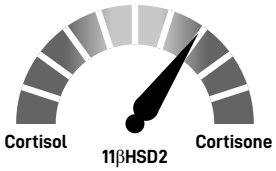
4-OH: *potential for DNA damage*

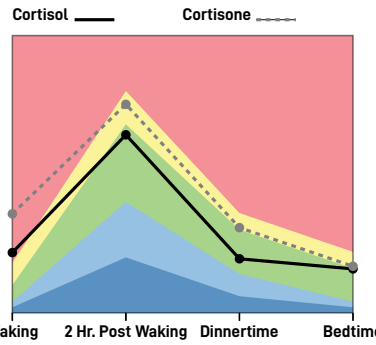
16-OH: *considered highly estrogenic*



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.

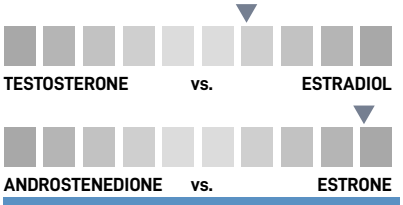
Metabolites in the 5-alpha pathway are more androgenic than their 5-beta counterparts and can be responsible for androgenic symptoms even when hormone levels appear normal.

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

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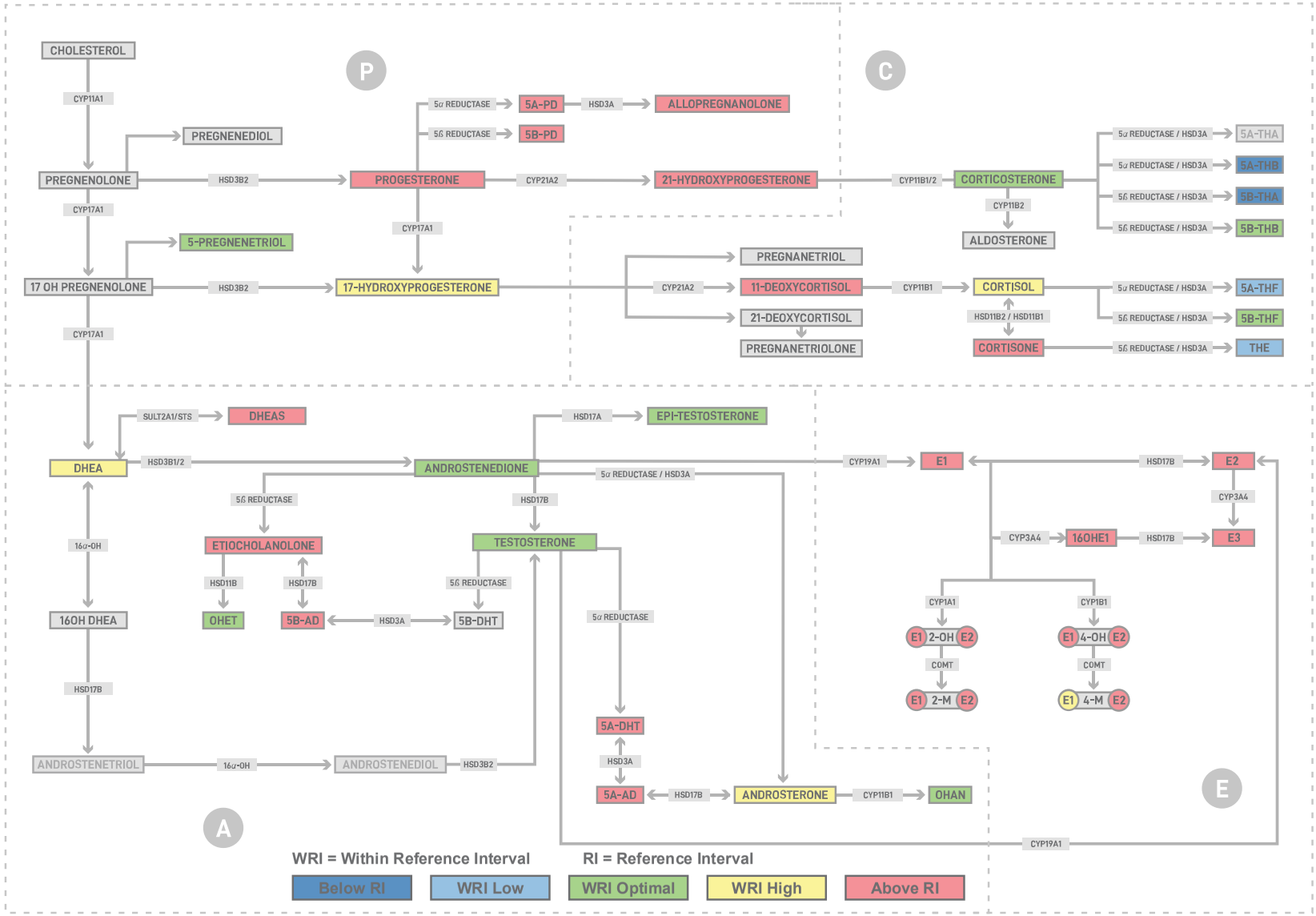
CHOLESTEROL
CYP11A1
PREGNENEDIOL
PREGNENOLONE
HSD3B2
PROGESTERONE
CYP21A2
21-HYDROXYPROGESTERONE
CYP11B1/2
CORTICOSTERONE
CYP17A1
5-PREGNENETRIOL
17 OH PREGNENOLONE
HSD3B2
17-HYDROXYPROGESTERONE
CYP17A1
P
C
5α REDUCTASE
5A-PD
HSD3A
ALLOPREGNANOLONE
5β REDUCTASE
5B-PD
5α REDUCTASE / HSD3A
5A-THA
5A-THB
5β REDUCTASE / HSD3A
5B-THA
5B-THB
CYP11B2
ALDOSTERONE
PREGNANETRIOL
CYP21A2
11-DEOXYCORTISOL
CYP11B1
CORTISOL
21-DEOXYCORTISOL
HSD11B2 / HSD11B1
PREGNANETRIOLONE
CORTISONE
5A-THF
5B-THF
THE
SULT2A1/STS
DHEAS
DHEA
HSD3B1/2
ANDROSTENEDIONE
HSD17A
EPI-TESTOSTERONE
CYP19A1
E1
HSD17B
E2
CYP3A4
16OHE1
E3
5β REDUCTASE
ETIOCHOLANOLONE
HSD17B
TESTOSTERONE
16α-OH
HSD11B
HSD17B
5β REDUCTASE
OHET
5B-AD
HSD3A
5B-DHT
16OH DHEA
5α REDUCTASE
CYP1A1
CYP1B1
E1 2-OH E2
E1 4-OH E2
COMT
COMT
E1 2-M E2
E1 4-M E2
HSD17B
5A-DHT
HSD3A
ANDROSTENETRIOL
16α-OH
ANDROSTENEDIOL
HSD3B2
5A-AD
HSD17B
ANDROSTERONE
CYP11B1
OHAN
A
E
CYP19A1
WRI = Within Reference Interval
RI = Reference Interval
Below RI
WRI Low
WRI Optimal
WRI High
Above RI

Progesterone Metabolites; urine

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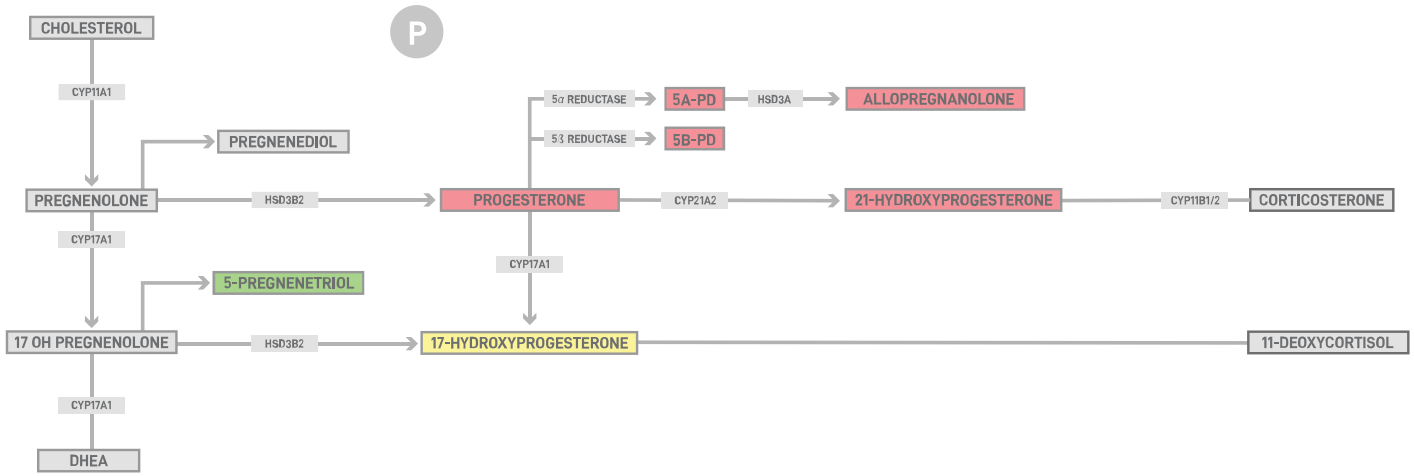

Progesterones		Result	Unit	L WRI H	Reference Interval
Progesterone‡	(P4)	160	ng/mg Creat/Day		0.00 – 0.22
5α-Pregnanediol‡	(5A-PD)	61	ng/mg Creat/Day		5 – 25
5β-Pregnanediol‡	(5B-PD)	419	ng/mg Creat/Day		70 – 320
Allopregnanolone‡	(ALLOP)	11	ng/mg Creat/Day		1.4 – 4.8
21-Hydroxyprogesterone‡	(21-OHP)	1.6	ng/mg Creat/Day		0.3 – 1.4
17-Hydroxyprogesterone‡	(17-OHP)	0.64	ng/mg Creat/Day		0.12 – 0.65
5-pregnenetriol‡	(5-PT)	79	ng/mg Creat/Day		35 – 120

Ratios and Calculations		Result	Unit	L WRI H	Reference Interval
5A-PD:5B-PD‡	(alpha vs beta metabolism)	0.15			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.

Notes:
The white triangle symbol (△) on the graph indicates patient value relative to the premenopausal reference range.
WRI – Within Reference Interval - represented by bracket and stated ranges on report, Dark Blue = Below RI, Light Blue = WRI low, Green = Optimal, Yellow = WRI high, Red = Above RI, <dl = result below detection limit
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Methodology: LCMS QQQ

Analyzed by DOCTOR'S DATA, INC. • 3755 Illinois Avenue, St. Charles, IL 60174-2420 USA • LAB DIR: Erlo Roth, MD • CLIA ID: 14D0646470

Progesterone Metabolites; urine

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Pre-menopausal Reference Intervals (Informational Use Only)

For postmenopausal women who have indicated that they are supplementing we are providing premenopausal reference intervals as a guide to assist the practitioner in assessing treatment. The white triangle symbol (△) on the graph indicates patient value relative to the premenopausal reference range.

Progesterones		Pre-menopausal Reference Interval
Progesterone‡	(P4)	0.18 – 1.8
5α-Pregnanediol‡	(5A-PD)	30 – 405
5β-Pregnanediol‡	(5B-PD)	300 – 2700
Allopregnanolone‡	(ALLOP)	3.3 – 110
21-Hydroxyprogesterone‡	(21-OHP)	0.4 – 5.6
5-pregnenetriol‡	(5-PT)	70 – 245

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Methodology: LCMS QQQ

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Adrenal Corticoid Metabolites; urine

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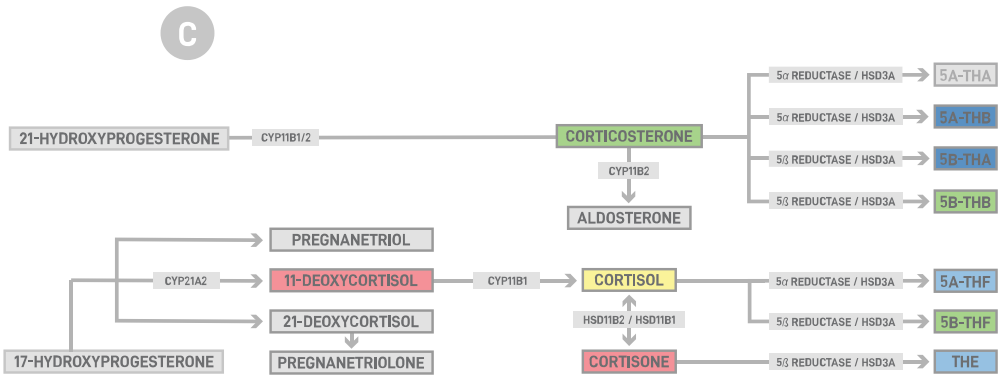

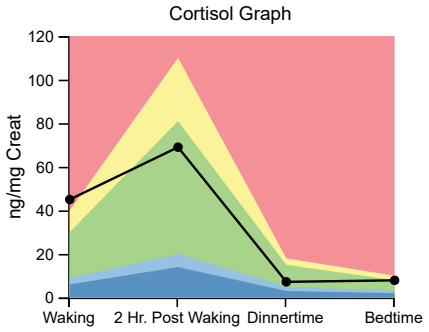

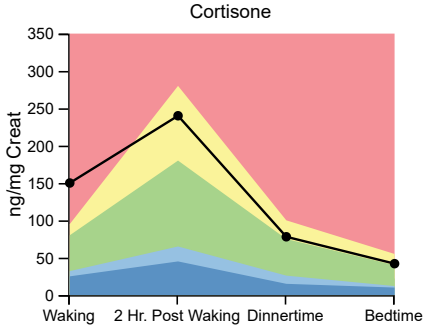

Free Cortisol and Cortisone		Result	Unit	L WRI H	Reference Interval
Cortisol Waking‡		45	ng/mg Creat		6 – 40
Cortisol Waking+2hrs‡		69	ng/mg Creat		14 – 110
Cortisol Dinnertime‡		7.2	ng/mg Creat		3 – 18
Cortisol Bedtime‡		7.9	ng/mg Creat		2 – 10
Cortisol/day‡	(F)	35	ng/mg Creat/Day		9 – 35
Cortisone Waking‡		150	ng/mg Creat		25 – 95
Cortisone Waking+2hrs‡		240	ng/mg Creat		45 – 280
Cortisone Dinnertime‡		78	ng/mg Creat		15 – 100
Cortisone Bedtime‡		42	ng/mg Creat		10 – 55
Cortisone/day‡	(E)	150	ng/mg Creat/Day		30 – 95
Creatinine Waking		34	mg/dL		30 – 225

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Free Cortisol and Cortisone	Result	Unit	L WRI H	Reference Interval
Creatinine Waking+2hrs	28	mg/dL		30 – 225
Creatinine Dinnertime	38	mg/dL		30 – 225
Creatinine Bedtime	12	mg/dL		30 – 225
Creatinine/day	27	mg/dL/Day		30 – 225

Corticoid Metabolites and DHEA		Result	Unit	L WRI H	Reference Interval
Corticosterone‡	(B)	25	ng/mg Creat/Day		6 – 34
Tetrahydrodehydrocorticosterone‡	(5B-THA)	38	ng/mg Creat/Day		44 – 150
5β-Tetrahydrocorticosterone‡	(5B-THB)	83	ng/mg Creat/Day		58 – 240
5α-Tetrahydrocorticosterone‡	(5A-THB)	69	ng/mg Creat/Day		90 – 380
11-Deoxycortisol‡	(11-DOC)	2.5	ng/mg Creat/Day		0.35 – 1.8
5α-Tetrahydrocortisol‡	(5A-THF)	202	ng/mg Creat/Day		150 – 860
5β-Tetrahydrocortisol‡	(5B-THF)	1130	ng/mg Creat/Day		720 – 2050
Tetrahydrocortisone‡	(THE)	1170	ng/mg Creat/Day		1000 – 3000
Dehydroepiandrosterone‡	(DHEA)	71	ng/mg Creat/Day		10 – 120
Dehydroepiandrosterone Sulfate‡	(DHEAS)	599	ng/mg Creat/Day		15 – 320

Ratios and Calculations		Result	Unit	L WRI H	Reference Interval
DHEA+DHEAS‡		670	ng/mg Creat/Day		25 – 370
THE+5A-THF+5B-THF‡	(Metabolized Cortisol)	2500	ng/mg Creat/Day		2000 – 6000
5A-THF+5B-THF/THE‡	(Cortisol/Cortisone Metabolites)	1.1			0.6 – 1.2
Cortisol/Cortisone‡	(11B HSD activity)	0.23			0.18 – 0.60
5A-THF/5B-THF ratio‡	(alpha vs beta metabolism)	0.2			0.15 – 0.65

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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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Androgen Metabolites; urine

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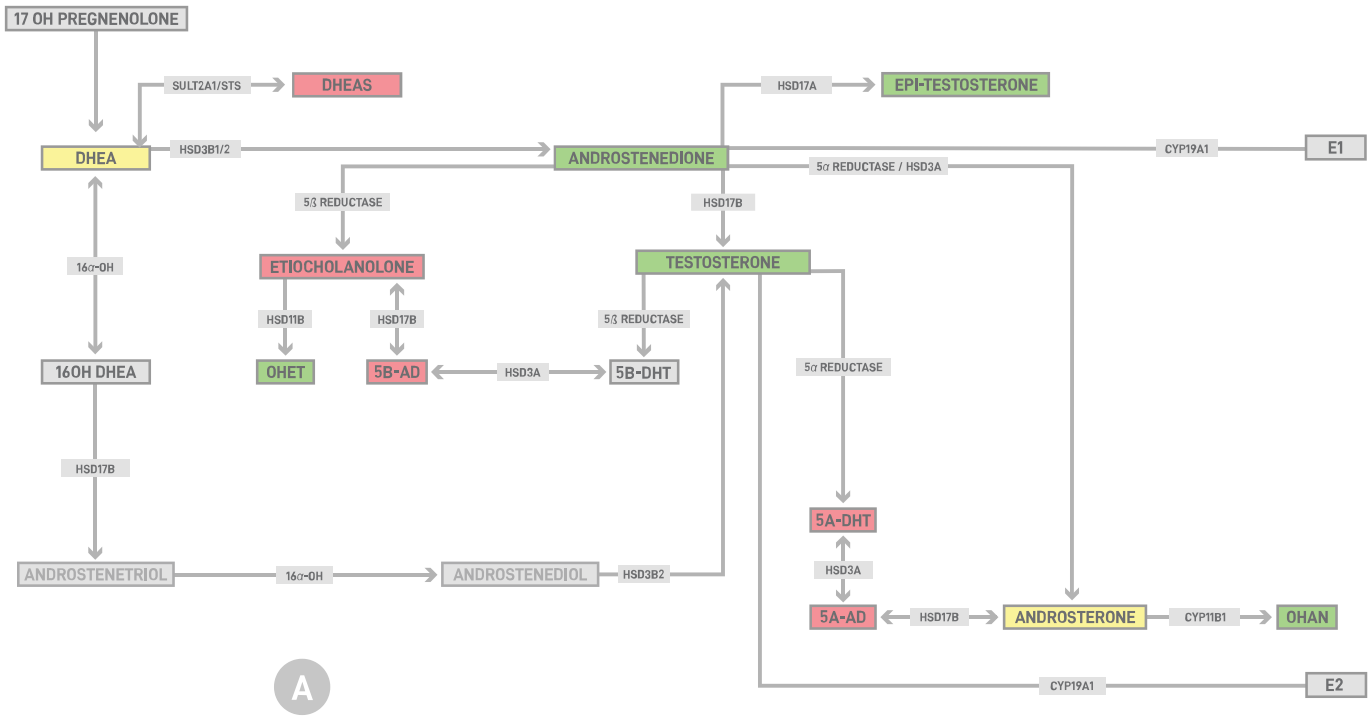

Androgens		Result	Unit	L WRI H	Reference Interval
Androstenedione‡	(A4)	1.5	ng/mg Creat/Day		0.2 – 5.3
EPI-Testosterone‡	(EPI-T)	1.2	ng/mg Creat/Day		0.0 – 5.0
Testosterone‡	(T)	2.9	ng/mg Creat/Day		1.0 – 12
Androsterone‡	(AN)	1070	ng/mg Creat/Day		250 – 1600
11-hydroxy-Androsterone‡	(OHAN)	442	ng/mg Creat/Day		180 – 800
5α-Androstanediol‡	(5A-AD)	30	ng/mg Creat/Day		2.5 – 15
5α-Dihydrotestosterone‡	(5A-DHT)	31	ng/mg Creat/Day		0.4 – 4.0

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Androgens		Result	Unit	L WRI H	Reference Interval
Etiocholanolone‡	(ET)	1850	ng/mg Creat/Day		290 – 1700
11-hydroxy-Etiocholanolone‡	(OHET)	384	ng/mg Creat/Day		40 – 470
5β-Androstanediol‡	(5B-AD)	97	ng/mg Creat/Day		7.0 – 87
Dehydroepiandrosterone‡	(DHEA)	71	ng/mg Creat/Day		10 – 120
Dehydroepiandrosterone Sulfate‡	(DHEAS)	599	ng/mg Creat/Day		15 – 320

Ratios and Calculations		Result	Unit	L WRI H	Reference Interval
DHEA+DHEAS‡		670	ng/mg Creat/Day		25 – 370
Androsterone (5α) / Etiocholanolone (5β)‡	(5α Reductase Activity)	0.6			0.5 – 1.4
Testosterone / EPI-Testosterone‡		2.4			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

Analyzed by DOCTOR'S DATA, INC. • 3755 Illinois Avenue, St. Charles, IL 60174-2420 USA • LAB DIR: Erlo Roth, MD • CLIA ID: 14D0646470

Androgen Metabolites; urine

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: 55 **DOB:** 01/01/1967
Sex: Female **Body Mass Index:** 20
Menopausal Status: Post-menopausal ,

Sample Collection	Date/Time
Midsleep	05/13/2023 00:14
Dinnertime	05/12/2023 18:00
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Collection Period	Multipoint daily
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Pre-menopausal Reference Intervals (Informational Use Only)

For postmenopausal women who have indicated that they are supplementing we are providing premenopausal reference intervals as a guide to assist the practitioner in assessing treatment. The white triangle symbol (△) on the graph indicates patient value relative to the premenopausal reference range.

Androgens		Pre-menopausal Reference Interval
Androstenedione‡	(A4)	0.5 – 9.2
EPI-Testosterone‡	(EPI-T)	0.0 – 15
Androsterone‡	(AN)	390 – 2200
5α-Androstanediol‡	(5A-AD)	4.0 – 25
5β-Androstanediol‡	(5B-AD)	9.0 – 110
Etiocholanolone‡	(ET)	540 – 2500
Dehydroepiandrosterone‡	(DHEA)	40 – 500
Dehydroepiandrosterone Sulfate‡	(DHEAS)	20 – 1200
DHEA+DHEAS‡		40 – 1500

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Estrogen Metabolites; urine

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Supplements: DHEA,E2,E3,P4

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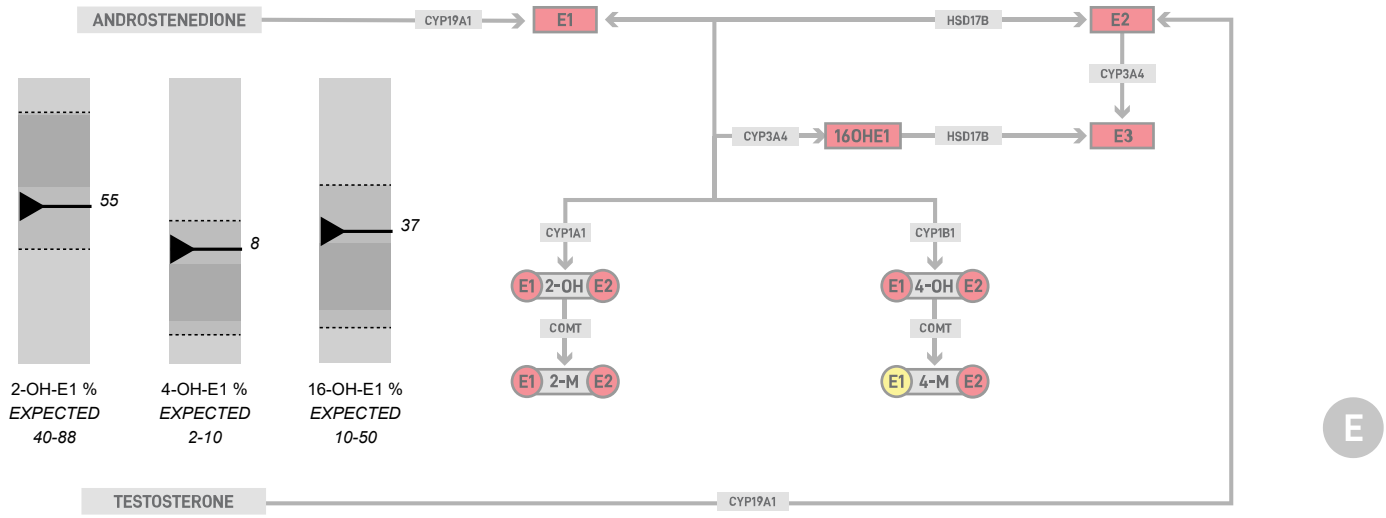

Estrogens		Result	Unit	L WRI H	Reference Interval
Estrone‡	(E1)	18	ng/mg Creat/Day		1.5 – 4.4
2-Hydroxyestrone‡	(2-OH-E1)	22	ng/mg Creat/Day		1.6 – 6.5
4-Hydroxyestrone‡	(4-OH-E1)	3.2	ng/mg Creat/Day		0.0 – 0.3
16α-Hydroxyestrone‡	(16-OH-E1)	15	ng/mg Creat/Day		0.5 – 5.3
2-Methoxyestrone‡	(2-M-E1)	2.6	ng/mg Creat/Day		0.4 – 2.2
4-Methoxyestrone‡	(4-M-E1)	0.13	ng/mg Creat/Day		0.02 – 0.14
Estradiol‡	(E2)	7.2	ng/mg Creat/Day		0.2 – 1.5
2-Hydroxyestradiol‡	(2-OH-E2)	3.0	ng/mg Creat/Day		0.03 – 0.29
4-Hydroxyestradiol‡	(4-OH-E2)	4.1	ng/mg Creat/Day		0.00 – 0.45
2-Methoxyestradiol‡	(2-M-E2)	0.30	ng/mg Creat/Day		0.01 – 0.08
4-Methoxyestradiol‡	(4-M-E2)	0.10	ng/mg Creat/Day		0.009 – 0.024
Estriol‡	(E3)	59	ng/mg Creat/Day		1.0 – 5.4

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Supplements: DHEA,E2,E3,P4

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Ratios and Calculations		Result	Unit	L WRI H	Reference Interval
2-OH-E1 %‡	(2-OH-E1 %)	55	%		40 – 88
4-OH-E1 %‡	(4-OH-E1 %)	8	%		2 – 10
16-OH-E1 %‡	(16-OH-E1 %)	37	%		10 – 50
2-M-E1:2-OH-E1‡	(COMT/Methylation activity)	0.11			0.08 – 0.50
2-M-E2:2-OH-E2‡	(COMT/Methylation activity)	0.10			0.07 – 0.86
4-M-E1:4-OH-E1‡	(COMT/Methylation activity)	0.04			0.09 – 1.0
4-M-E2:4-OH-E2‡	(COMT/Methylation activity)	0.02			0.02 – 0.50
2-OH-E1:16-OH-E1 ‡		1.5			≥ 0.60
4-OH-E1:2-OH-E1‡		0.15			0.00 – 0.17

Oxidative Stress Metabolite		Result	Unit	L WRI H	Reference Interval
8-hydroxy-2'-deoxyguanosine‡	(8-OHdG)	1.7	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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Pre-menopausal Reference Intervals (Informational Use Only)

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Estrogens		Pre-menopausal Reference Interval
Estrone‡	(E1)	3.8 – 22
2-Hydroxyestrone‡	(2-OH-E1)	13 – 34
4-Hydroxyestrone‡	(4-OH-E1)	0.0 – 2.9
16α-Hydroxyestrone‡	(16-OH-E1)	1.4 – 15
2-Methoxyestrone‡	(2-M-E1)	1.0 – 5.9
4-Methoxyestrone‡	(4-M-E1)	0.05 – 0.28
Estradiol‡	(E2)	1.5 – 13
2-Hydroxyestradiol‡	(2-OH-E2)	0.80 – 3.9
4-Hydroxyestradiol‡	(4-OH-E2)	0.00 – 2.3
2-Methoxyestradiol‡	(2-M-E2)	0.04 – 0.50
4-Methoxyestradiol‡	(4-M-E2)	0.049 – 0.11
Estriol‡	(E3)	2.8 – 23

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Progesterones

Progesterone (P4)
In cycling females, progesterone is primarily produced in the corpus luteum of the ovaries, and to a lesser degree in the adrenal glands. Menopausal females continue to produce small amounts of progesterone in the adrenal glands. Elevated levels of progesterone may be due to high dose pregnenolone supplementation, progesterone supplementation, exogenous progesterone exposure, pregnancy, disorders of luteinization, increased HSD3A activity, reduced activity of CYP21A or CYP17A, and rarely thecal cell tumors. In addition, elevations of both progesterone and pregnanediol, progesterone's major metabolite, have been reported in 21 hydroxylase deficiency.

5A-PD
5A-PD is a minor urinary metabolite of progesterone. Increased levels may be due to high levels of progesterone and/or pregnenolone, progesterone supplementation, or adrenocortical hyperplasia. 5A-PD may agonize GABA-A receptors.

5B-PD
5B-PD is the major progesterone metabolite. On its own, elevations may not be clinically significant. However, increased levels could be due to high levels of progesterone and/or pregnenolone, pregnancy, ovarian cyst, pregnenolone and/or progesterone supplementation, or adrenocortical hyperplasia. In addition, elevations of both progesterone and pregnanediol have been reported in 21-hydroxylase deficiency.

Allopregnanolone (ALLOP)
Allopregnanolone is a downstream metabolite of progesterone and is considered a neurosteroid due to its ability to influence the GABA-A receptor, creating anxiolytic effects. Elevated levels can be seen with high endogenous progesterone or 5-alpha reductase preference, as well as exogenous oral supplementation of progesterone.

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

5a-Androstanediol (5A-AD)
5A-AD is a metabolite of 5αDHT and an intermediate in 5αDHT creation within the backdoor pathway. Research suggests elevations of this pathway in females may be due to PCOS and hirsutism.

5α-Dihydrotestosterone (5A-DHT)
5A-DHT is converted from testosterone by 5-alpha reductase in the ovaries and peripherally in fat tissue. Higher levels may be associated with acne, scalp hair loss, and hirsutism.

Etiocholanolone (ET)
Etiocholanolone is a 5-beta reduced isomer of androsterone, and a major metabolite of testosterone and androstenedione, however it is not active as an androgen.

5β-Androstanediol (5B-AD)
5B-AD is the result of the 5-beta reduction of DHT and is a metabolite of etiocholanolone. Elevated levels may be due to an increased conversion via 5-beta reductase, or from DHEA or testosterone supplementation.

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Supplements: DHEA,E2,E3,P4

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Androgens

Dehydroepiandrosterone Sulfate (DHEAS)
Dehydroepiandrosterone sulfate (DHEAS) is the sulfated form of dehydroepiandrosterone (DHEA) and the major steroid precursor in humans. This sulfation is reversibly catalyzed by sulfotransferase 2A1 (SULT2A1) primarily in the adrenals, the liver, and the small intestine. Like DHEA, research suggests DHEAS elevations could be due PCOS, adult-onset adrenal hyperplasia, congenital adrenal hyperplasia, and very rarely adrenal carcinoma. Increased levels of DHEA, as well as pregnenolone, through either supplementation or endogenous excretion, may also contribute to elevated levels of DHEAS.

T:EPI-T
Elevations in the testosterone / EPI-testosterone ratio in females can be caused by exogenous testosterone supplementation and in some cases hormonal contraceptives which lower EPI-T, increasing testosterone / EPI-testosterone ratio.

Corticoids

Tetrahydrodehydrocorticosterone (THA)
5B-THA is a terminal metabolite of corticosterone. This metabolite in combination with other terminal metabolites can be used to estimate metabolism of corticosterone. While research in elevations of single terminal metabolites is limited, assessing metabolism may provide valuable information about enzyme activity.

5α-Tetrahydrocorticosterone (5A-THB)
5A-THB is a terminal metabolite of corticosterone. This metabolite along with the other terminal metabolites can be used to determine metabolism of corticosterone. While research in elevations of single terminal metabolites is limited, assessment of metabolism may provide more information regarding enzyme activity.

11-Deoxycortisol (11-DOC)
11-Deoxycortisol has very little glucocorticoid activity, yet it is helpful to understand its role as an intermediate in cortisol creation and how it can contribute to impairment of the pathway. 11-Deoxycortisol is metabolized via CYP11B (11-beta hydroxylase) to cortisol. Elevations of 11-deoxycortisol may be due to impairment of CYP11B, congenital adrenal hyperplasia, or andrenocortico tumors ir rare cases. Elevations of blood pressure due to a buildup of 11-deoxycortisol have been reported.

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.

DHEA + DHEAS
DHEA and DHEAs are produced in the adrenal gland and serve as precursors to androgens and estrogens. Due to the interconversion between via SULT2A1 and/or STS, the sum of DHEA and DHEAs may be a better representation of total DHEA synthesis.

Estrogens

Estrone (E1)
A component of the estrone level may be due to aromatization of androstenedione and testosterone by CYP19 (aromatase) enzyme in adipose tissue and/or conversion from estradiol due to HSD17B activity. Elevated estrone has been associated with increased risk of carcinogenic potential in breast tissue in postmenopausal women, particularly when accompanied by elevated testosterone. CYP19 enzyme is induced during times of stress, exposure to xeno-estrogens, high glycemic diet, excessive adipose tissue, and alcohol consumption.

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Estrogens

2-Hydroxyestrone (2-OH-E1)
Adequate levels of 2-OH-E1 have been shown to be a favorable marker for breast health. Elevated levels can be due to efficient metabolism or high endogenous estrone as well as exogenous exposure to or supplementation of estrone and/or estradiol. While 2-OH-E1 is considered the “safer” estrogen metabolite, optimizing methylation to support the COMT enzyme can potentiate favorable excretion rates.

4-Hydroxyestrone (4-OH-E1)
Higher levels can indicate slowed methylation and possible carcinogenic potential in breast tissue for in women. Elevation may also be due to an overactive CYP1B1 enzyme or sluggish CYP1A1 or CYP34A. Research indicates CYP1B1 is upregulated by PAHs, PCBs, THC, UV exposure, leptin resistance, inflammation, and insulin resistance. Additional support for the COMT enzyme can help with the conversion toward the inactive metabolite 4-M-E1.

16α-Hydroxyestrone (16-OH-E1)
Higher levels of 16-OH-E1 indicate possible carcinogenic potential and other negative markers of breast health in females. Elevations in 16-OH-E1 may be due to increased metabolism from estrone or a sluggish HSD17B enzyme, keeping 16-OH-E1 from converting into estriol.

2-Methoxyestrone (2-M-E1)
2-M-E1 is considered a non-reactive metabolite. Higher levels correlated with antiproliferative and antiangiogenic effects as well as cardioprotective properties. Depending on other metabolite values, and if excretion from the GI tract is functioning properly, elevations in 2-M-E1 may be considered favorable.

Estradiol (E2)
Elevated estradiol level may be due to exogenous hormone supplementation or aromatization from testosterone in peripheral tissues. The CYP19 enzyme, also known as aromatase, is induced during times of stress, exposure to xeno-estrogens, high glycemic diet, excessive adipose tissue, and alcohol consumption.

2-Hydroxyestradiol (2-OH-E2)
Adequate levels of 2-OH-E2 have been shown to be a favorable marker for breast health. Elevated levels can be due to efficient metabolism or high endogenous estradiol as well as exogenous exposure to or supplementation of estrone and/or estradiol. While 2-OH-E2 is considered the “safer” estrogen metabolite, optimizing methylation to support the COMT enzyme can potentiate favorable excretion rates.

4-Hydroxyestradiol (4-OH-E2)
Elevated levels indicate possible carcinogenic potential and other negative markers of breast health in females. Elevation of 4-OH-E2 may be due to an overactive CYP1B1 enzyme or sluggish CYP1A1 or CYP3A4. Research indicates CYP1B1 is upregulated by PAHs, PCBs, THC, UV exposure, leptin resistance, inflammation, and insulin resistance. Supporting the COMT enzyme (methylation) is a consideration.

2-Methoxyestradiol (2-M-E2)
2-M-E2 is considered non-reactive and protective. Higher levels correlated with antiproliferative, antiangiogenic, and cardioprotective properties. Depending on the other metabolite values, and if excretion from the GI tract is functioning properly, elevations in 2-M-E2 may be considered favorable.

4-Methoxyestradiol (4-M-E2)
Methyl metabolites are considered inactive and are correlated with antiproliferative effects. Proper elimination of 4-M-E2 requires optimal excretion via GI tract optimization. To fully understand this value, it may be beneficial to examine the 4-M-E2 / 4-OH-E2 ratio.

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Estrogens

Estriol (E3)

Estriol is above the reference range which is likely due to individual variance, supplementation, or exogenous exposure. Increased metabolism from 16-OH-E1 via HSD17β may also be a contributing factor. Estriol is considered a safer estrogen due to its inability to convert back to estrone or estradiol. Elevations may have little clinical significance if other metabolite levels seem appropriate.

4-M-E1:4-OH-E1 (COMT/Methylation activity)

The relationship of 4-M-E1 / 4-OH-E1 represents the activity of COMT (methylation) enzyme. A low ratio indicates slower COMT activity, which may mean a higher potential for the creation of quinones, semi-quinones, and depurinating adducts.Increasing COMT enzyme activity is a consideration.

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