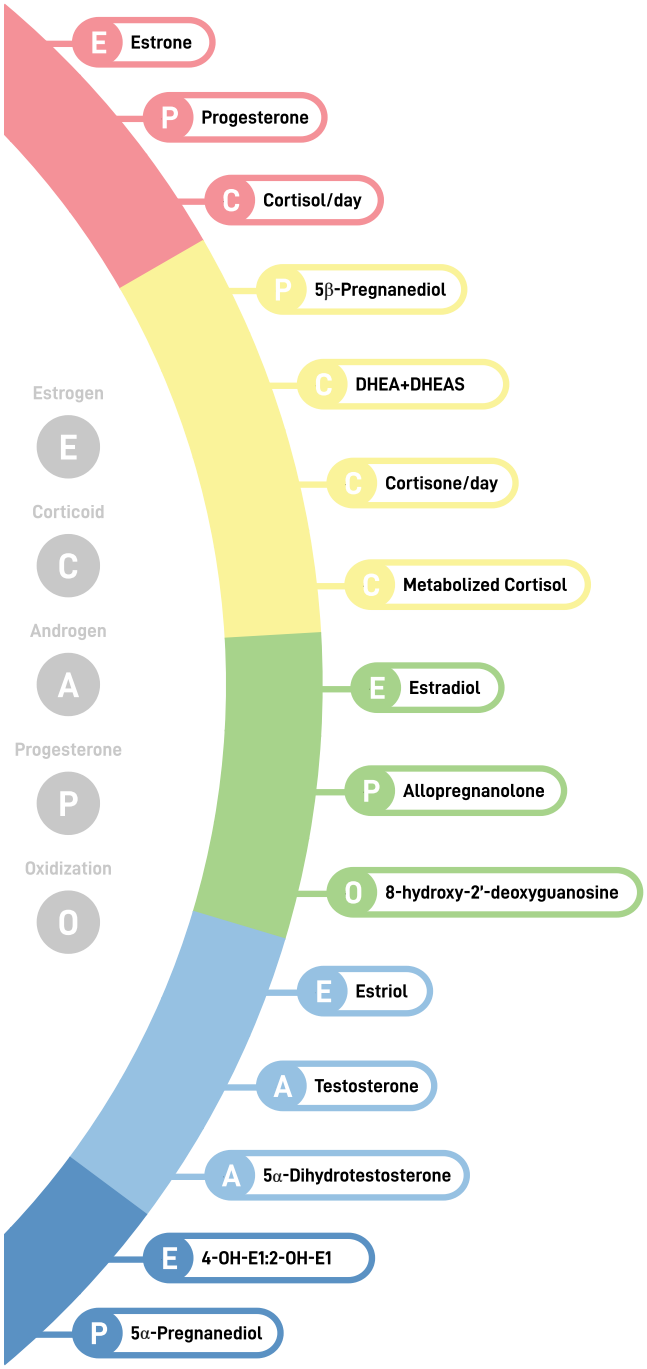
Hormone & Urinary Metabolites Assessment Profile

Order: 999999-9999
Test: X999999-9999-1
Client #: 999999
Doctors Data Inc
123 Main St.
St. Charles, IL 60174 USA

Patient: Sample Patient
Id: 999999
Age: 61 **DOB:** 01/01/1960
Sex: Female
Body Mass Index (BMI): 25

Menopausal Status: Post-menopausal

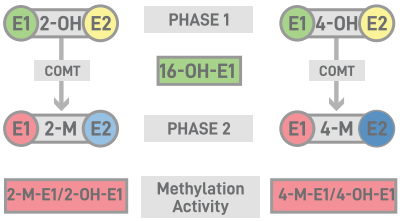
Sample Collection	Date/Time
Dinnertime	12/30/2022 19:20
Bedtime	12/30/2022 22:30
Waking	12/31/2022 07:00
2 Hr. Post Waking	12/31/2022 09:30
Collection Period	Multipoint daily
Date Received	01/01/2023
Date Reported	01/02/2023



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.

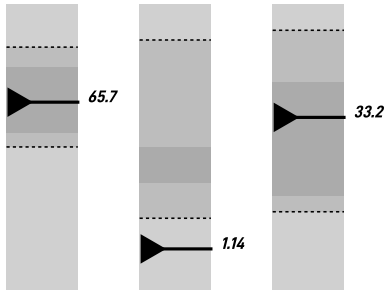
The pathway illustrates phase 1 and phase 2 metabolism of both E1 and E2. Phase 1 metabolites, also known as catechol estrogens, are active and can induce estrogenic actions. Phase 2 metabolism gives insight into a patient's ability to methylate, or potentially inactivate harmful metabolites.



2-OH: *generally considered safest*

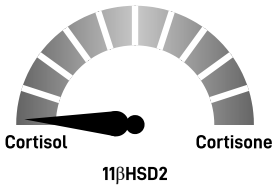
4-OH: *potential for DNA damage*

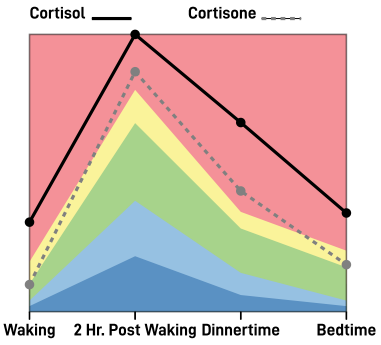
16-OH: *considered highly estrogenic*



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.





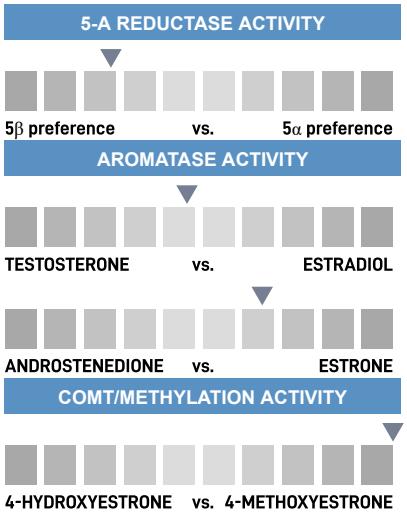
KEY RELATIONSHIPS

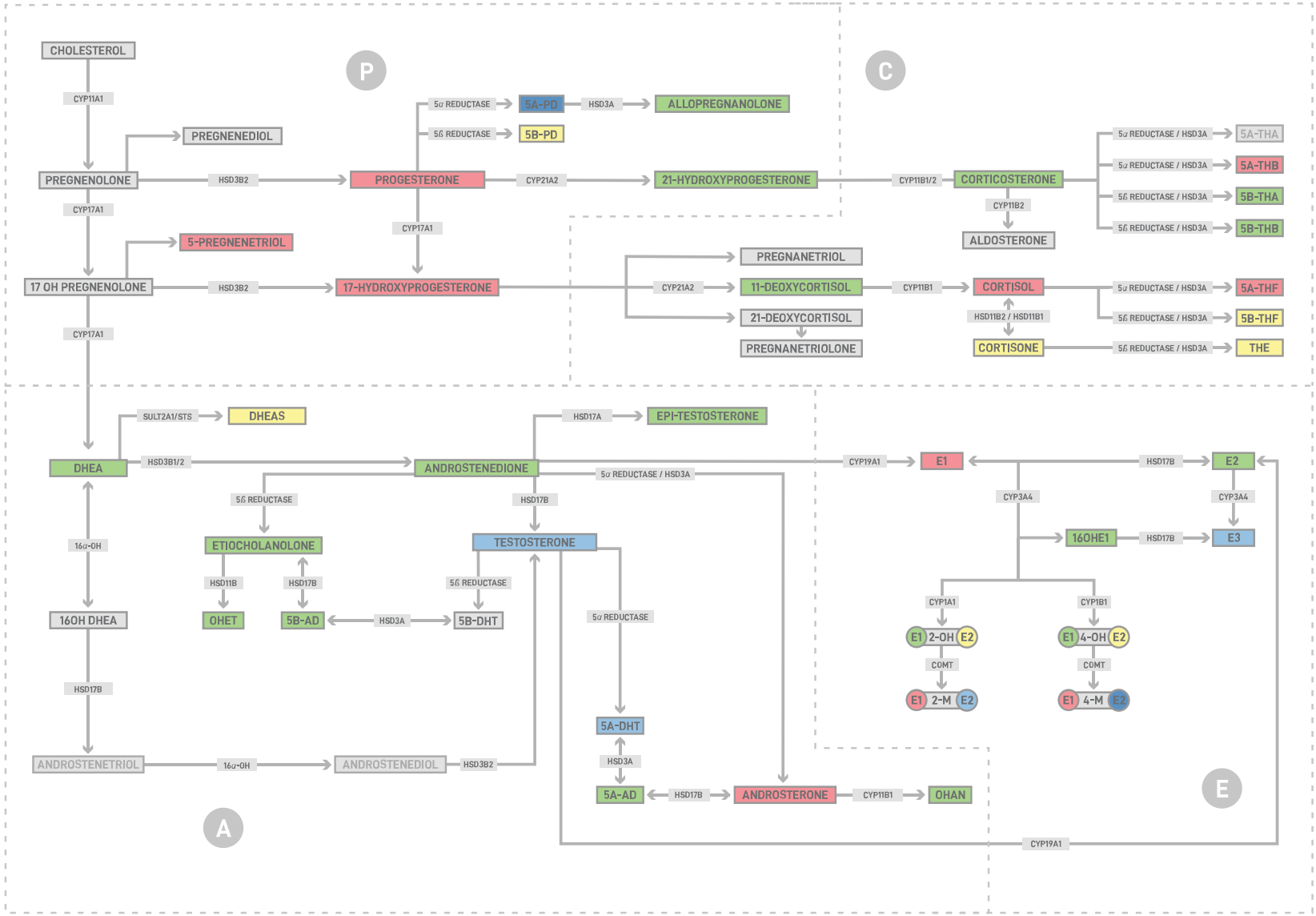
The graphs to the right represent metabolism preference by key enzymes, indicated by the arrow.

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.




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

Progesterone Metabolites; urine

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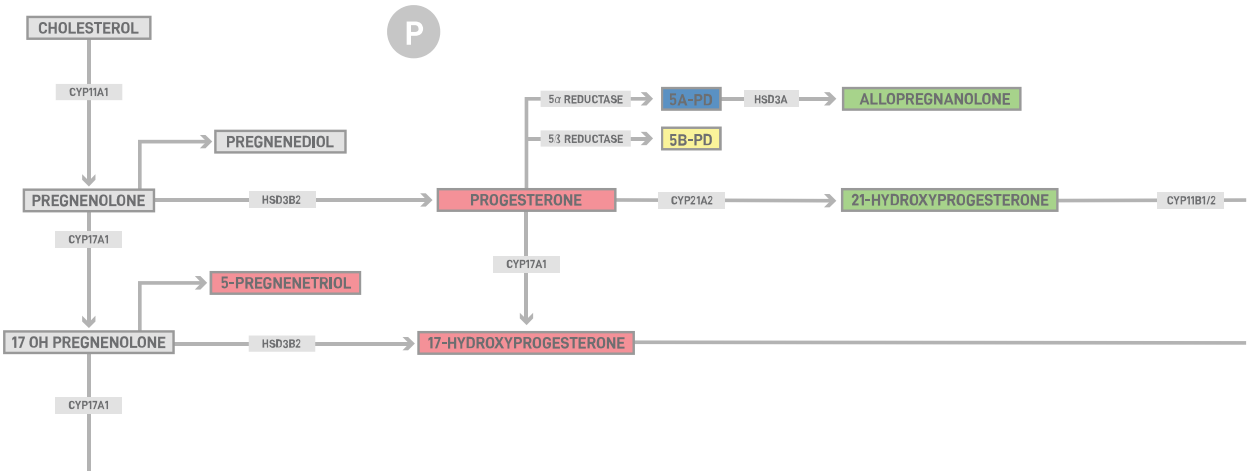

Progesterones		Result	Unit	L WRI H	Reference Interval
Progesterone	(P4)	0.464	ng/mg Creat/Day		0 – 0.22
5α-Pregnanediol	(5A-PD)	18.6	ng/mg Creat/Day		21 – 50
5β-Pregnanediol	(5B-PD)	255	ng/mg Creat/Day		79 – 280
Allopregnanolone	(ALLOP)	2.74	ng/mg Creat/Day		1.4 – 4.8
21-Hydroxyprogesterone	(21-OHP)	0.837	ng/mg Creat/Day		0.3 – 1.4
17-Hydroxyprogesterone	(17-OHP)	0.629	ng/mg Creat/Day		0.17 – 0.55
5-pregnenetriol	(5-PT)	204	ng/mg Creat/Day		35 – 120

Ratios and Calculations		Result	Unit	L WRI H	Reference Interval
5A-PD:5B-PD	(alpha vs beta metabolism)	0.073			0.1 – 0.5

Adrenal Corticoid Metabolites; urine

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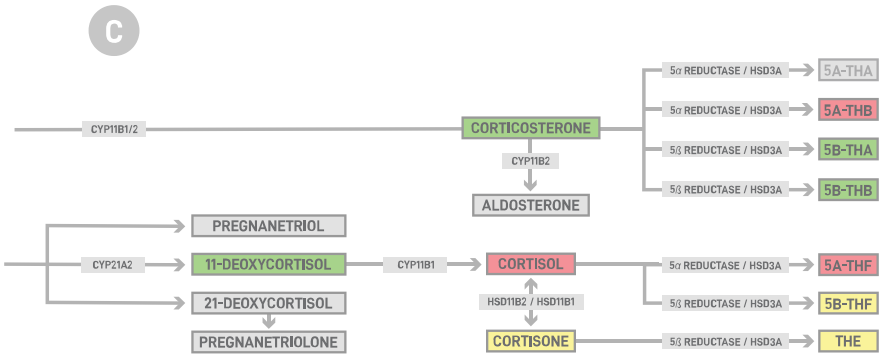

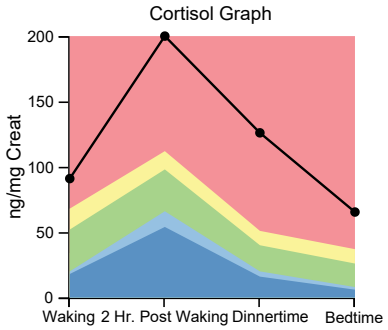

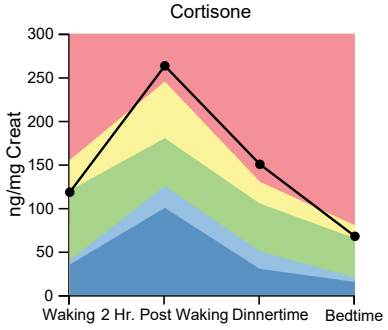

Free Cortisol and Cortisone		Result	Unit	L WRI H	Reference Interval
Cortisol Waking		91.0	ng/mg Creat		18 – 68
Cortisol Waking+2hrs		265	ng/mg Creat		54 – 112
Cortisol Dinnertime		126	ng/mg Creat		16 – 51
Cortisol Bedtime		65.4	ng/mg Creat		6 – 37
Cortisol/day	(F)	130	ng/mg Creat/Day		30 – 90
Cortisone Waking		118	ng/mg Creat		35 – 155
Cortisone Waking+2hrs		263	ng/mg Creat		100 – 245
Cortisone Dinnertime		150	ng/mg Creat		30 – 130
Cortisone Bedtime		67.4	ng/mg Creat		15 – 80
Cortisone/day	(E)	145	ng/mg Creat/Day		60 – 165
Creatinine Waking		68.5	mg/dL		30 – 225
Creatinine Waking+2hrs		115	mg/dL		30 – 225
Creatinine Dinnertime		93.4	mg/dL		30 – 225
Creatinine Bedtime		116	mg/dL		30 – 225
Creatinine/day		104	mg/dL/Day		30 – 225

Adrenal Corticoid Metabolites; urine

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Corticoid Metabolites and DHEA		Result	Unit	L WRI H	Reference Interval
Corticosterone	(B)	27.7	ng/mg Creat/Day		10 – 47
Tetrahydrodehydrocorticosterone	(5B-THA)	111	ng/mg Creat/Day		46 – 220
5β-Tetrahydrocorticosterone	(5B-THB)	202	ng/mg Creat/Day		65 – 240
5α-Tetrahydrocorticosterone	(5A-THB)	515	ng/mg Creat/Day		160 – 430
11-Deoxycortisol	(11-DOC)	0.949	ng/mg Creat/Day		0.35 – 1.8
5α-Tetrahydrocortisol	(5A-THF)	1320	ng/mg Creat/Day		200 – 1300
5β-Tetrahydrocortisol	(5B-THF)	2470	ng/mg Creat/Day		900 – 2600
Tetrahydrocortisone	(THE)	3210	ng/mg Creat/Day		1180 – 4000
Dehydroepiandrosterone	(DHEA)	33.9	ng/mg Creat/Day		10 – 120
Dehydroepiandrosterone Sulfate	(DHEAS)	173	ng/mg Creat/Day		35 – 300

Ratios and Calculations		Result	Unit	L WRI H	Reference Interval
DHEA+DHEAS		207	ng/mg Creat/Day		62 – 283
THE+5A-THF+5B-THF	(Metabolized Cortisol)	7000	ng/mg Creat/Day		2500 – 7900
5A-THF+5B-THF/THE	(Cortisol/Cortisone Metabolites)	1.17			0.7 – 1.4
Cortisol/Cortisone	(11B HSD activity)	0.891			0.4 – 0.8
5A-THF/5B-THF ratio	(alpha vs beta metabolism)	0.537			0.4 – 1.4

Androgen Metabolites; urine

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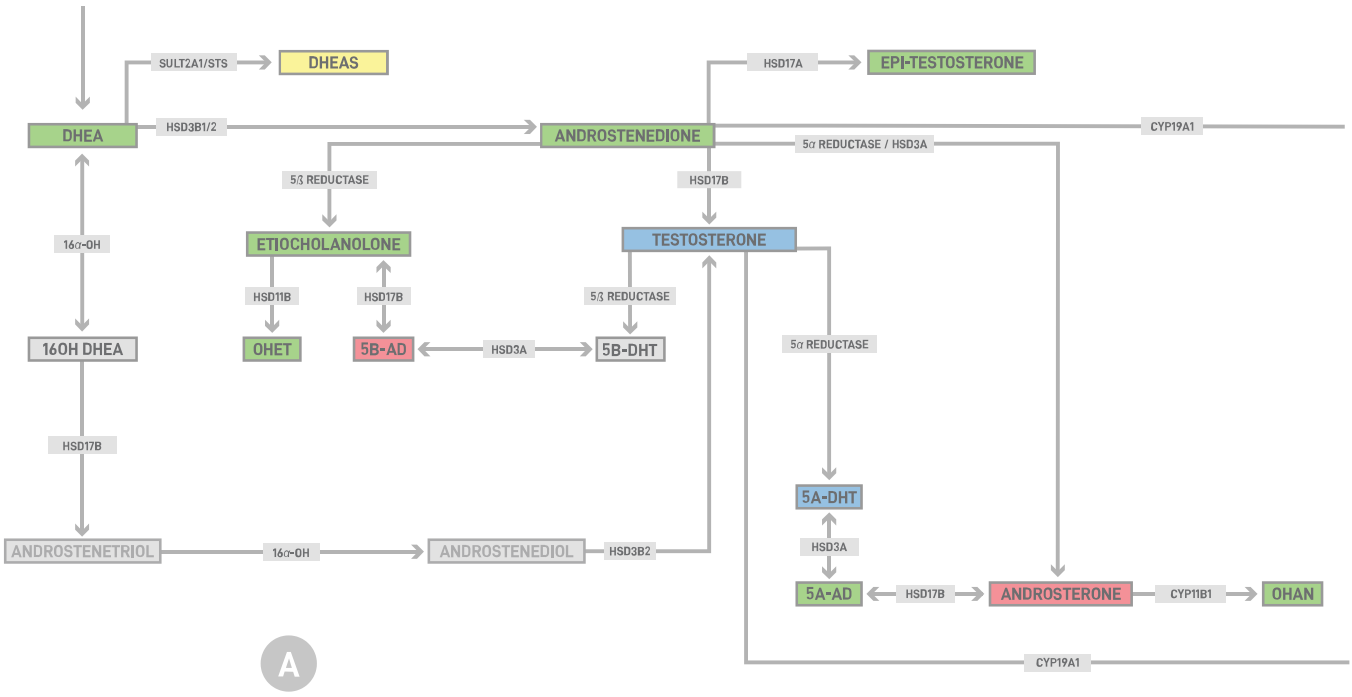

Androgens		Result	Unit	L	WRI	H	Reference Interval
Androstenedione	(A4)	1.15	ng/mg Creat/Day				0.2 – 5.3
EPI-Testosterone	(EPI-T)	1.38	ng/mg Creat/Day				0 – 5
Testosterone	(T)	1.91	ng/mg Creat/Day				0.25 – 10.9
Androsterone	(AN)	928	ng/mg Creat/Day				170 – 850
11-hydroxy-Androsterone	(OHAN)	762	ng/mg Creat/Day				250 – 1000
5α-Androstanediol	(5A-AD)	8.63	ng/mg Creat/Day				4.8 – 16
5α-Dihydrotestosterone	(5A-DHT)	0.391	ng/mg Creat/Day				0.2 – 6
Etiocholanolone	(ET)	1070	ng/mg Creat/Day				240 – 1410
11-hydroxy-Etiocholanolone	(OHET)	83.0	ng/mg Creat/Day				20 – 710
5β-Androstanediol	(5B-AD)	65.4	ng/mg Creat/Day				14 – 62
Dehydroepiandrosterone	(DHEA)	33.9	ng/mg Creat/Day				10 – 120

Androgen Metabolites; urine

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Androgens		Result	Unit	L WRI H	Reference Interval
Dehydroepiandrosterone Sulfate	(DHEAS)	173	ng/mg Creat/Day		35 – 300

Ratios and Calculations		Result	Unit	L WRI H	Reference Interval
DHEA+DHEAS		207	ng/mg Creat/Day		62 – 283
Androsterone (5α) / Etiocholanolone (5β)	(5α Reductase Activity)	0.864			0.8 – 2.6
Testosterone / EPI-Testosterone		1.38			0.7 – 3

Estrogen Metabolites; urine

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123 Main St.
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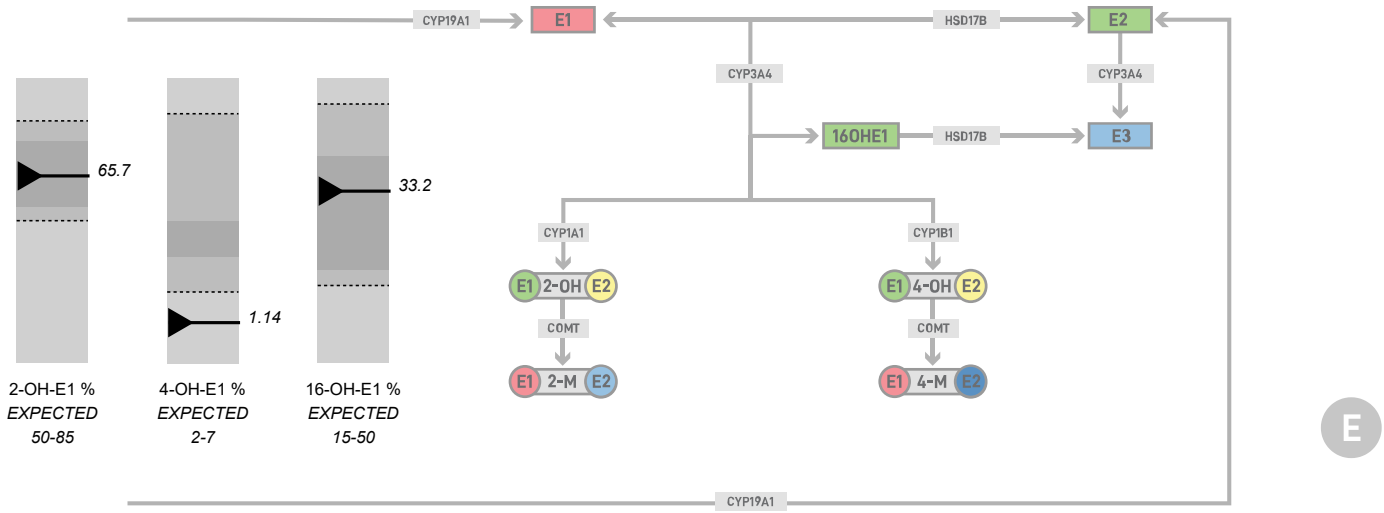

Estrogens		Result	Unit	L WRI H	Reference Interval
Estrone	(E1)	5.62	ng/mg Creat/Day		1.75 – 5.12
2-Hydroxyestrone	(2-OH-E1)	3.97	ng/mg Creat/Day		1.62 – 6.5
4-Hydroxyestrone	(4-OH-E1)	0.069	ng/mg Creat/Day		0 – 0.3
16α-Hydroxyestrone	(16-OH-E1)	2.00	ng/mg Creat/Day		1.05 – 5.3
2-Methoxyestrone	(2-M-E1)	2.36	ng/mg Creat/Day		0.41 – 1.34
4-Methoxyestrone	(4-M-E1)	0.099	ng/mg Creat/Day		0.007 – 0.05
Estradiol	(E2)	1.02	ng/mg Creat/Day		0.2 – 1.6
2-Hydroxyestradiol	(2-OH-E2)	0.288	ng/mg Creat/Day		0.033 – 0.29
4-Hydroxyestradiol	(4-OH-E2)	0.231	ng/mg Creat/Day		0.052 – 0.26
2-Methoxyestradiol	(2-M-E2)	0.018	ng/mg Creat/Day		0.012 – 0.039
4-Methoxyestradiol	(4-M-E2)	0.004	ng/mg Creat/Day		0.009 – 0.024
Estriol	(E3)	3.27	ng/mg Creat/Day		1.61 – 5.6

Ratios and Calculations		Result	Unit	L WRI H	Reference Interval
2-OH-E1 %	(2-OH-E1 %)	65.7	%		50 – 85
4-OH-E1 %	(4-OH-E1 %)	1.14	%		2 – 7

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Ratios and Calculations		Result	Unit	L WRI H	Reference Interval
16-OH-E1 %	(16-OH-E1 %)	33.2	%		15 – 50
2-M-E1:2-OH-E1	(COMT/Methylation activity)	0.568			0.1 – 0.36
2-M-E2:2-OH-E2	(COMT/Methylation activity)	0.061			0.07 – 0.37
4-M-E1:4-OH-E1	(COMT/Methylation activity)	1.37			0.09 – 0.54
4-M-E2:4-OH-E2	(COMT/Methylation activity)	0.015			0.04 – 0.54
2-OH-E1:16-OH-E1		1.98			1.6 – 5.1
4-OH-E1:2-OH-E1		0.017			0.02 – 0.07

Oxidative Stress Metabolite		Result	Unit	L WRI H	Reference Interval
8-hydroxy-2'-deoxyguanosine	(8-OHdG)	2.67	ng/mg Creat/Day		0 – 7.5

Commentary

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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
Lower levels of pregnanediol have been associated with amenorrhea, decreased ovarian function, PCOS, ovarian cancer, and certain complications of pregnancy.

17-Hydroxyprogesterone (17-OHP)
17-Hydroxyprogesterone is the product of progesterone hydroxylation. Elevations are associated with PCOS, idiopathic hirsutism, congenital adrenal hyperplasia, 11-beta-hydroxylase deficiency, and adult onset viralizing adrenal hyperplasia. Additionally, hyperinsulinemia and hyperglycemia (metabolic syndrome) push 17-hydroxylation of progesterone.

Pregnenetriol (5-PT)
5-pregnenetriol is a metabolite of 17α-pregnenolone, an intermediary resulting from the hydroxylation of pregnenolone by CYP 17A1 enzyme. Elevations in urine may be seen in cases of PCOS, Cushing's Syndrome, congenital adrenal hyperplasia, and adrenocortical carcinoma.

5A-PD : 5B-PD
The metabolic prioritization for alpha or beta reductase activity within the progesterone pathway may be confirmatory of a general preference of metabolism. Comparing these results with the metabolic preference of androgens and corticoids may provide additional insight.

Androgens

Androsterone (AN)
Androsterone is the product of androgens metabolized by 5-alpha reductase. It acts as a neurosteroid and a weak potentiator of GABA-A receptor activity. Androsterone may also be converted to DHT via backdoor pathway using HSD3β and HSD17β making it a metabolic intermediate. Potential causes of AN elevation may include PCOS, over supplementation of DHEA or pregnenolone, androgen producing gonadal tumors, congenital adrenal hyperplasia, adult-onset adrenal hyperplasia, serious illness, shock, and burns.

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.

Corticoids

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.

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Corticoids

Cortisol (F)
Cortisol is the main glucocorticoid released from the adrenal gland in response to stress. Elevated levels of cortisol have been reported in cases of Cushing's disease, malnutrition, early life stress, hypothyroidism, depression, alcoholism, PCOS, obesity, and critical illness. Additionally, exogenous exposure to glucocorticoids prior to testing may be a source of cortisol elevations.

5a-Tetrahydrocortisol (5A-THF)
5A- THF is a terminal metabolite of cortisol metabolized via 5 alpha reductase. Combining all the terminal metabolites can be used to estimate metabolized cortisol. While research into single terminal metabolite elevations is limited, it may have more clinical relevance when assessed in combination with the daily output of free cortisol.

Cortisol/Cortisone (11B HSD activity)
Cortisol / cortisone ratio measures activity of HSD11B2 activity and assessment of tissue specific concentration of cortisol, which normally cannot be measured without a biopsy. An elevated ratio indicates suppressed enzyme activity or a low conversion rate of cortisol to cortisone. This can be seen in stress, hypertension, metabolic syndrome, insulin resistance, PCOS, depression, with cortisol supplementation, or high licorice doses.

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 HSD17β activity. Elevated estrone has been associated with increased risk of breast cancer 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.

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 healthy.

4-Methoxyestrone (4-M-E1)
Methyl metabolites are considered inactive and are correlated with protective and antiproliferative effects. Proper elimination of 4-M-E1 requires optimal excretion via the GI tract; optimizing GI health is an option. To fully understand this value, it may be beneficial to examine the 4-M-E1 / 4-OH-E1 ratio.

4-Methoxyestradiol (4-M-E2)
Lower levels of 4-M-E2 is associated with a higher risk of certain cancers and other negative markers for breast health. Low levels of 4-M-E2 may indicate that 4-OH metabolites are favoring the quinone/semi quinone pathway which can lead to DNA damage. Supporting the COMT enzyme (methylation) is a consideration.

2-M-E1:2-OH-E1 (COMT/Methylation activity)
The relationship of 2-M-E1 / 2-OH-E1 represents the activity of COMT (methylation). While 2-OH-E1 is considered a safe metabolite, it is still considered a reactive metabolite until methylated and inactivated. Elevated COMT activity shows more of 2-OH-E1 is being methylated, which is considered favorable. Over time, COMT enzyme may need additional support to keep up with demand. Comparing additional areas of COMT activity (i.e., 4-M-E1/ 4-OH-E1) may give more insight into the function of this enzyme.

Commentary

Order: 999999-9999
Test: X999999-9999-1
Client #: 999999
Doctors Data Inc
123 Main St.
St. Charles, IL 60174 USA

Patient: Sample Patient
Id: 999999
Age: 61 **DOB:** 01/01/1960
Sex: Female
Body Mass Index (BMI): 25

Menopausal Status: Post-menopausal

Sample Collection	Date/Time
Dinnertime	12/30/2022 19:20
Bedtime	12/30/2022 22:30
Waking	12/31/2022 07:00
2 Hr. Post Waking	12/31/2022 09:30
Collection Period	Multipoint daily
Date Received	01/01/2023
Date Reported	01/02/2023

Estrogens

2-M-E2:2-OH-E2 (COMT/Methylation activity)
The relationship of 2-M-E2 / 2-OH-E2 represents the activity of COMT (methylation) enzyme. A low ratio indicates slower COMT activity. While 2-OH-E2 is considered a safe metabolite, it is still considered a reactive metabolite until methylated and inactivated. Comparing additional areas of COMT activity (i.e., 4-M-E1/ 4-OH-E1) may give more insight into the function of this enzyme.

4-M-E1:4-OH-E1 (COMT/Methylation activity)
The relationship of 4-M-E1 / 4-OH-E1 represents the activity of COMT (methylation). 4-OH-E1 is considered unfavorable due to its carcinogenic potential within breast and prostatic tissue. Elevated COMT activity shows more of 4-OH-E1 is being methylated, which is considered favorable. Over time, COMT enzyme may need additional support to keep up with demand. Comparing additional areas of COMT activity (i.e., 2-M-E1/ 2-OH-E1) may give more insight into the function of this enzyme.

4-M-E2:4-OH-E2 (COMT/Methylation activity)
The relationship of 4-M-E2 / 4-OH-E2 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.

4-OH-E1:2-OH-E1
A low ratio can indicate a metabolic preference for the less favorable 4-OH-E1 pathway. Optimizing methylation to support the COMT enzyme can potentiate the more protective 2-OH-E1 pathway. Increasing the activity of CYP1A1 to increase 2-OH-E1 is a consideration.