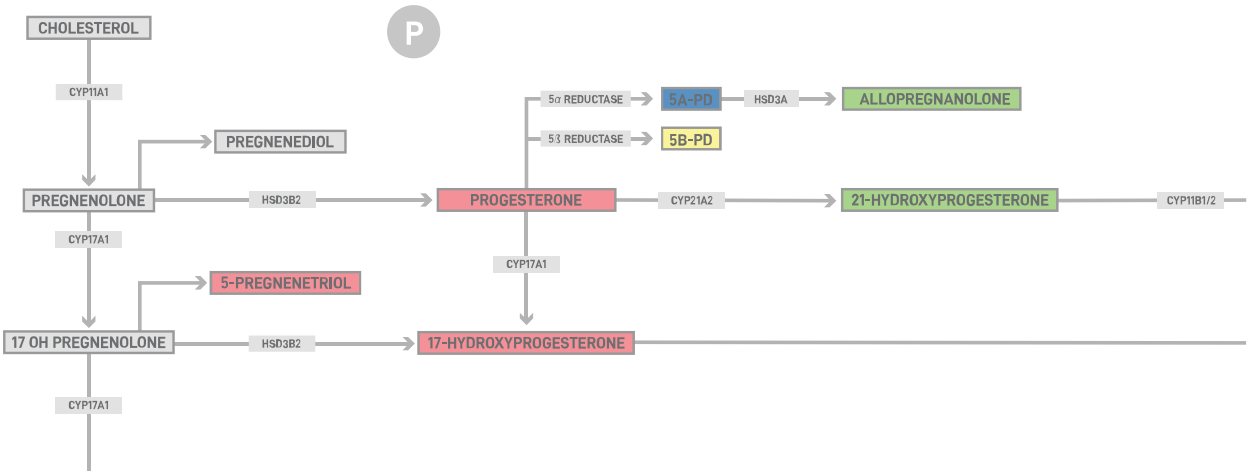
Sex Hormones profile; urine

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



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

Creatinine	Result	Unit	L WRI H	Reference Interval
Creatinine/day	104	mg/dL/Day		30 – 225

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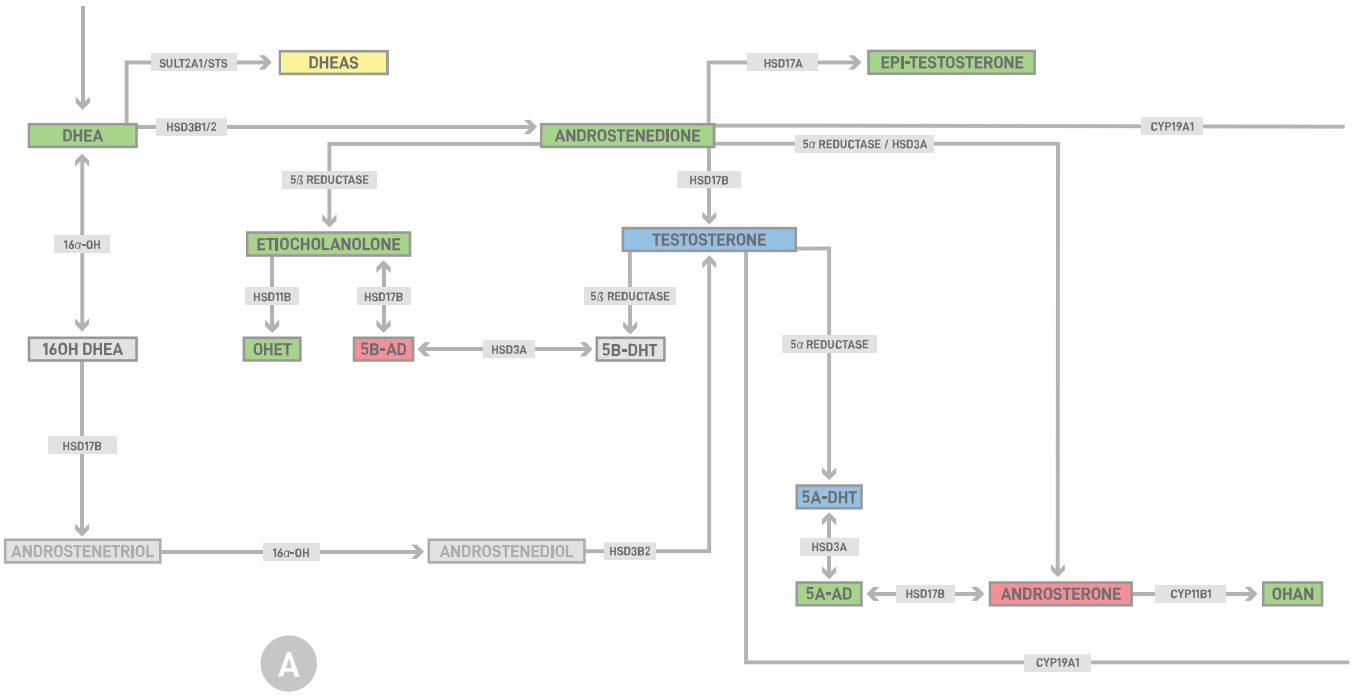

Androgens		Result	Unit	L WRI H	Reference Interval
Androstenedione	(A4)	1.15	ng/mg Creat/Day		0.2 – 5.3
EPI-Testosterone	(EPI-T)	1.38	ng/mg Creat/Day		0 – 5
Testosterone	(T)	1.91	ng/mg Creat/Day		0.25 – 10.9
Androsterone	(AN)	928	ng/mg Creat/Day		170 – 850
11-hydroxy-Androsterone	(OHAN)	762	ng/mg Creat/Day		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

Sex Hormones profile; 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

Creatinine	**Result**	**Unit**	L WRI H	**Reference Interval**
Creatinine/day	104	mg/dL/Day		30 – 225

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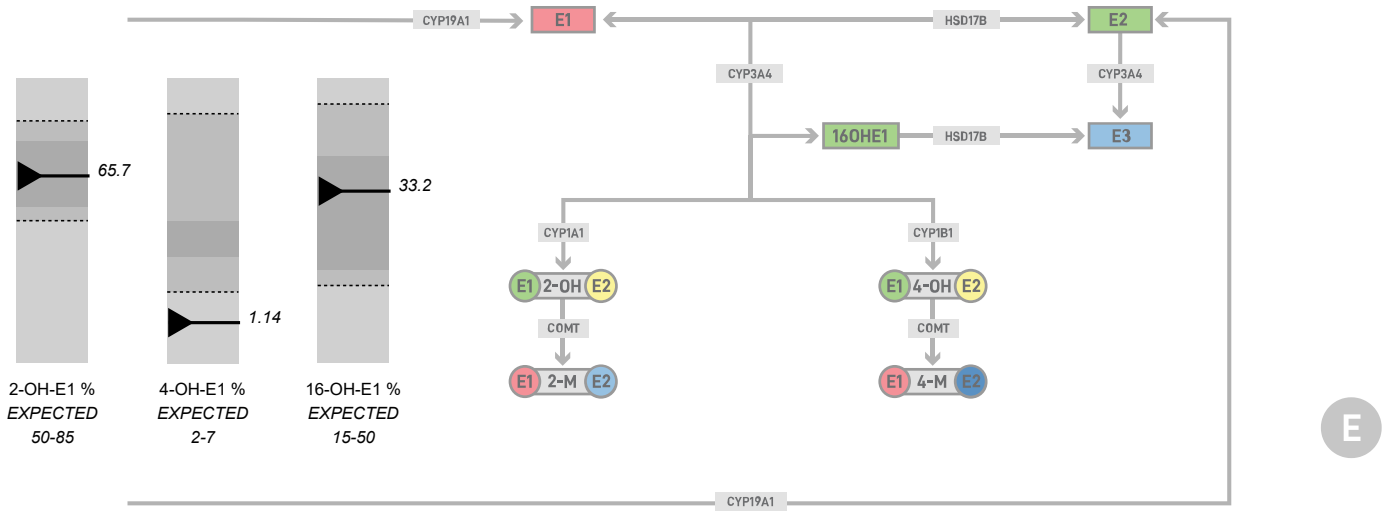

Estrogens		Result	Unit	L WRI H	Reference Interval
Estrone	(E1)	5.62	ng/mg Creat/Day		1.75 – 5.12
2-Hydroxyestrone	(2-OH-E1)	3.97	ng/mg Creat/Day		1.62 – 6.5
4-Hydroxyestrone	(4-OH-E1)	0.069	ng/mg Creat/Day		0 – 0.3
16α-Hydroxyestrone	(16-OH-E1)	2.00	ng/mg Creat/Day		1.05 – 5.3
2-Methoxyestrone	(2-M-E1)	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

Creatinine	Result	Unit	L WRI H	Reference Interval
Creatinine/day	104	mg/dL/Day		30 – 225

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.

Commentary

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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.

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.