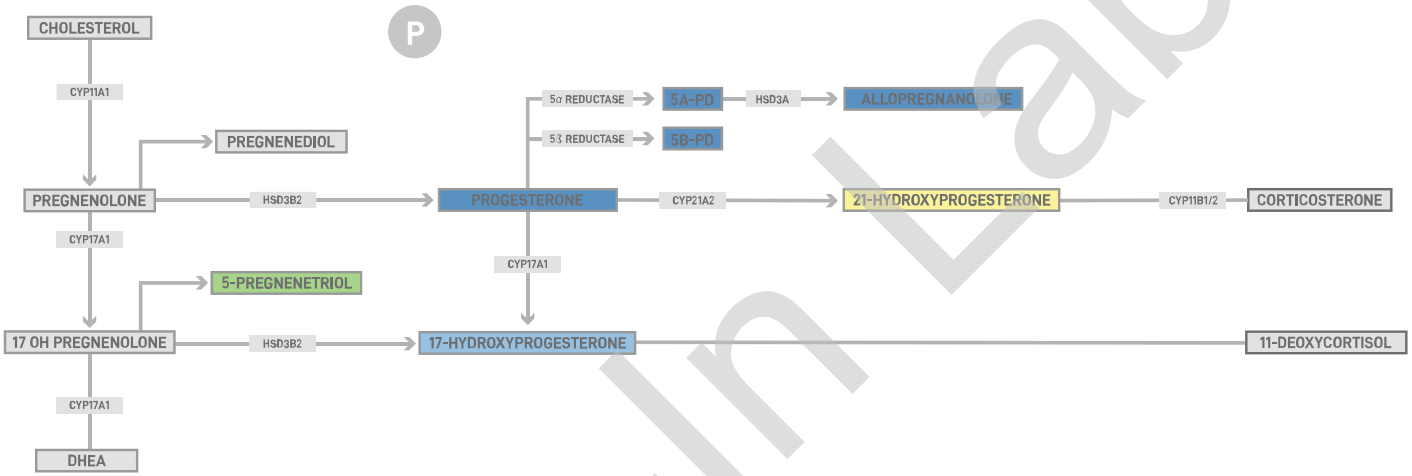
Sex Hormones profile; urine

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: 21 **DOB:** 01/01/2003
Sex: Female
Menopausal Status: Pre-menopausal ,

Sample Collection	Date/Time
Collection Period	Multipoint daily
Date Received	11/22/2024
Date Reported	12/02/2024



Progesterones		Result	Unit	L WRI H	Reference Interval
Progesterone‡	(P4)	0.03	ng/mg Creat/Day		0.10 – 1.10
5α-Pregnanediol‡	(5A-PD)	28	ng/mg Creat/Day		30 – 405
5β-Pregnanediol‡	(5B-PD)	198	ng/mg Creat/Day		300 – 2700
Allopregnanolone‡	(ALLOP)	3.0	ng/mg Creat/Day		3.3 – 110
21-Hydroxyprogesterone‡	(21-OHP)	0.65	ng/mg Creat/Day		0.10 – 0.80
17-Hydroxyprogesterone‡	(17-OHP)	0.21	ng/mg Creat/Day		0.15 – 1.3
5-pregnenetriol‡	(5-PT)	164	ng/mg Creat/Day		70 – 245

Ratios and Calculations		Result	Unit	L WRI H	Reference Interval
5A-PD:5B-PD‡	(alpha vs beta metabolism)	0.14			0.06 – 0.24

Creatinine	Result	Unit	L WRI H	Reference Interval
Creatinine/day	224	mg/dL/Day		30 – 225

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Methodology: LCMS QQQ

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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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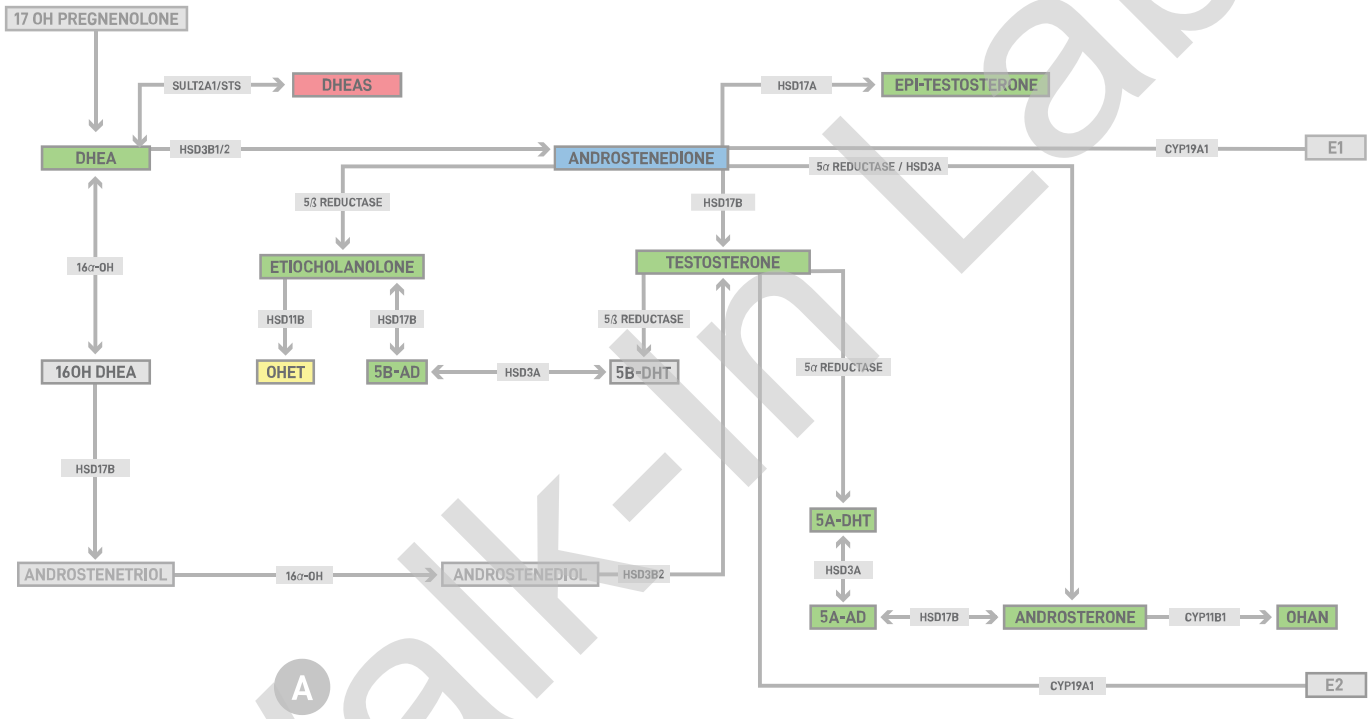

Androgens		Result	Unit	L WRI H	Reference Interval
Androstenedione‡	(A4)	0.85	ng/mg Creat/Day		0.35 – 4.0
EPI-Testosterone‡	(EPI-T)	7.7	ng/mg Creat/Day		0.0 – 15
Testosterone‡	(T)	3.5	ng/mg Creat/Day		1.0 – 12
Androsterone‡	(AN)	1180	ng/mg Creat/Day		390 – 2200
11-hydroxy-Androsterone‡	(OHAN)	539	ng/mg Creat/Day		180 – 800
5α-Androstanediol‡	(5A-AD)	13	ng/mg Creat/Day		4.0 – 25
5α-Dihydrotestosterone‡	(5A-DHT)	0.9	ng/mg Creat/Day		0.4 – 4.0
Etiocholanolone‡	(ET)	973	ng/mg Creat/Day		540 – 2500

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Androgens		Result	Unit	L WRI H	Reference Interval
11-hydroxy-Etiocholanolone‡	(OHET)	408	ng/mg Creat/Day		40 – 470
5β-Androstanediol‡	(5B-AD)	28	ng/mg Creat/Day		9.0 – 110
Dehydroepiandrosterone‡	(DHEA)	43	ng/mg Creat/Day		15 – 190
Dehydroepiandrosterone Sulfate‡	(DHEAS)	3300	ng/mg Creat/Day		45 – 3000

Ratios and Calculations		Result	Unit	L WRI H	Reference Interval
DHEA+DHEAS‡		3300	ng/mg Creat/Day		50 – 2000
Androsterone (5α) / Etiocholanolone (5β)‡	(5α Reductase Activity)	1.2			0.5 – 1.4
Testosterone / EPI-Testosterone‡		0.46			0.1 – 2.0

Creatinine	Result	Unit	L WRI H	Reference Interval
Creatinine/day	224	mg/dL/Day		30 – 225

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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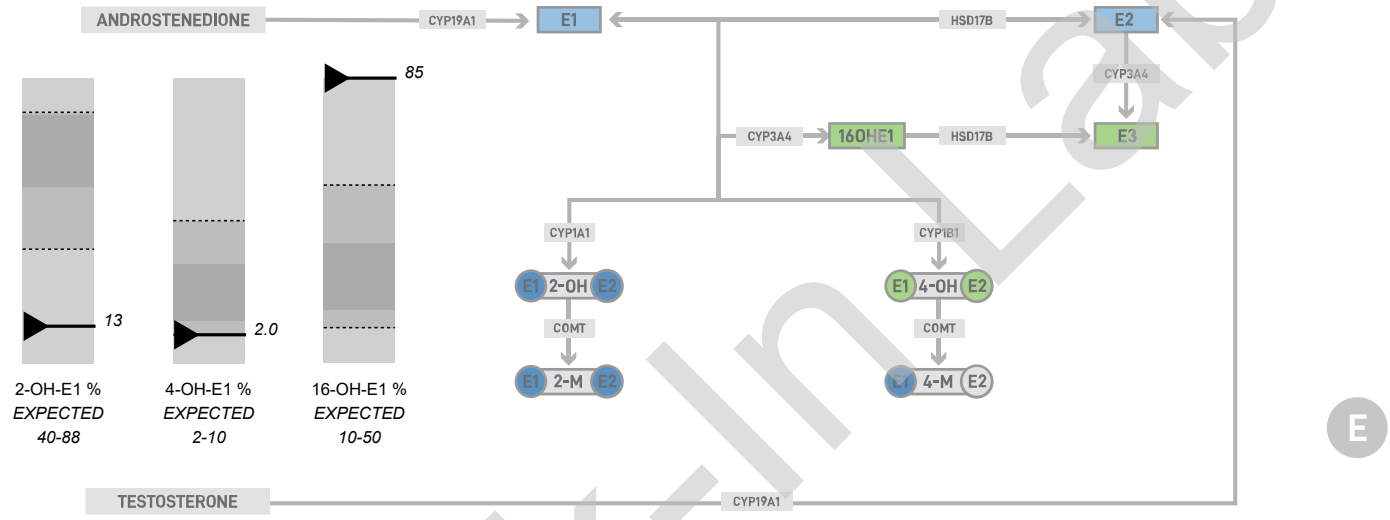

Estrogens		Result	Unit	L WRI H	Reference Interval
Estrone‡	(E1)	3.9	ng/mg Creat/Day		3.8 – 22
2-Hydroxyestrone‡	(2-OH-E1)	1.5	ng/mg Creat/Day		13 – 34
4-Hydroxyestrone‡	(4-OH-E1)	0.24	ng/mg Creat/Day		0.0 – 2.9
16α-Hydroxyestrone‡	(16-OH-E1)	10	ng/mg Creat/Day		1.4 – 15
2-Methoxyestrone‡	(2-M-E1)	0.13	ng/mg Creat/Day		1.0 – 7.10
4-Methoxyestrone‡	(4-M-E1)	0.002	ng/mg Creat/Day		0.005 – 0.060
Estradiol‡	(E2)	1.9	ng/mg Creat/Day		1.5 – 13
2-Hydroxyestradiol‡	(2-OH-E2)	0.10	ng/mg Creat/Day		0.80 – 3.9
4-Hydroxyestradiol‡	(4-OH-E2)	0.079	ng/mg Creat/Day		0.0 – 1.2
2-Methoxyestradiol‡	(2-M-E2)	0.017	ng/mg Creat/Day		0.06 – 0.70
Estriol‡	(E3)	18	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 %)	13	%		40 – 88
4-OH-E1 %‡	(4-OH-E1 %)	2.0	%		2 – 10
16-OH-E1 %‡	(16-OH-E1 %)	85	%		10 – 50
2-M-E1:2-OH-E1‡	(COMT/Methylation activity)	0.080			0.08 – 0.60
2-M-E2:2-OH-E2‡	(COMT/Methylation activity)	0.16			0.06 – 0.80
4-M-E1:4-OH-E1‡	(COMT/Methylation activity)	0.0093			0.004 – 0.10
2-OH-E1:16-OH-E1 ‡		0.15			≥ 0.70
4-OH-E1:2-OH-E1‡		0.16			0.00 – 0.17

Creatinine	Result	Unit	L WRI H	Reference Interval
Creatinine/day	224	mg/dL/Day		30 – 225

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

Progesterone (P4)
In cycling females, progesterone is produced in the corpus luteum of the ovaries, and to a lesser extent the adrenal glands. Menopausal females continue to produce small amounts of progesterone in the adrenal glands. Low/low range levels of progesterone may be due to anovulation, amenorrhea, perimenopause and menopause.

5A-PD
Lower levels of 5A-PD are often not clinically significant, but in research has been associated with amenorrhea, decreased ovarian function, PCOS, and rarely, ovarian neoplastic processes.

5B-PD
Low 5B-PD may not be clinically significant on its own, but in the presence of elevated 5A-PD, low 5B-PD could be associated with cases of amenorrhea, decreased ovarian function, PCOS, or rarely ovarian neoplastic processes.

Allopregnanolone (ALLOP)
Low levels of allopregnanolone can be seen with low progesterone, anovulatory cycles, the use of oral contraceptives containing ethinyl estradiol and levonorgestrel, and decreased 5-alpha reductase or HSD3A activity.

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.

Corticoids

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

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.

2-Methoxyestrone (2-M-E1)
2-M-E1 is considered a non-reactive metabolite. Lower levels indicate possible carcinogenic potential and other negative markers of breast health in females. A genetic variant of the MTHFR enzyme may contribute to decreased methylation. If a variant is suspected, further evaluation may be warranted.

4-Methoxyestrone (4-M-E1)
Lower levels of 4-M-E1 indicate possible carcinogenic potential and other negative markers of breast health in females. Low levels of 4-M-E1 may indicate the possibility that 4-OH metabolites are favoring the quinone/semi quinone pathway which can lead to DNA damage. Increased support of the COMT enzyme (methylation) may be an option. To fully understand this value, it may be beneficial to examine the 4-M-E1 / 4-OH-E1 ratio.

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Estrogens

2-Hydroxyestradiol (2-OH-E2)
Adequate levels of 2-OH-E2, the “safer” estrogen metabolite, have been shown to be a marker for breast health. Low levels of 2-OH-E2 may be due to low levels of estradiol, estrone, or more active CYP34A or CYP1B1 enzymes. Increasing the activity of CYP1A1 to increase 2-OH-E1 is a consideration.

2-Methoxyestradiol (2-M-E2)
2-M-E2 is considered a non-reactive metabolite. Lower levels have been associated with neoplastic risk and other negative markers of breast health in women. Supporting the COMT enzyme (methylation) is a consideration.

2-OH-E1:16-OH-E1
16-OH E1 has been shown to be more estrogenic than 2-OH-E1 with properties similar to estrone. A lower ratio favors the 16-OH-E1 pathway and could indicate an increased carcinogenic potential in breast tissue. Increasing the activity of CYP1A1 to increase 2-OH-E1 is a consideration.

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