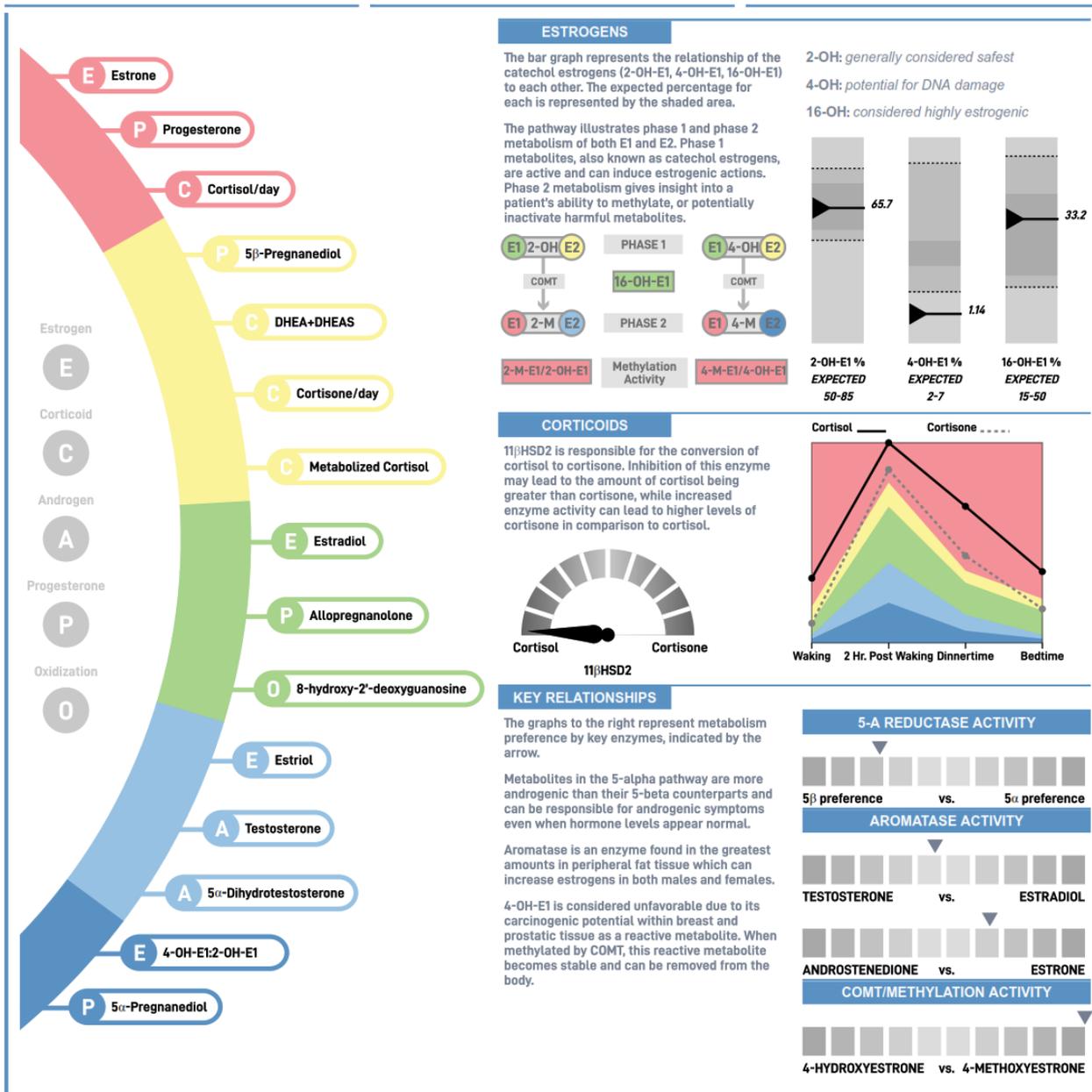
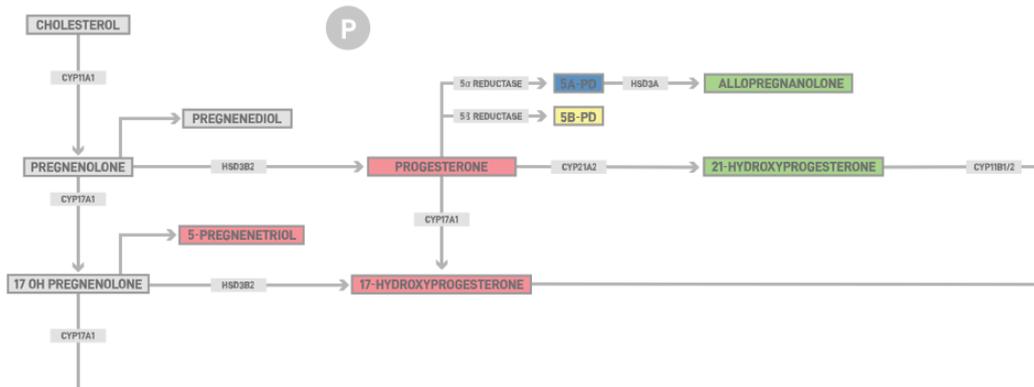


TEST NAME: HuMap (Hormone & Urinary Metabolites Assessment Profile)



TEST NAME: HuMap (Hormone & Urinary Metabolites Assessment Profile)

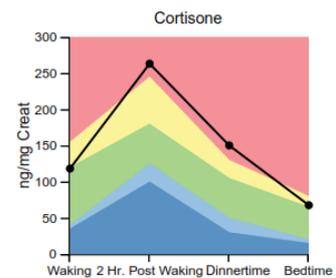
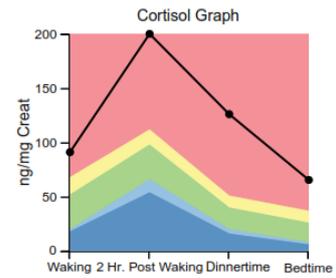
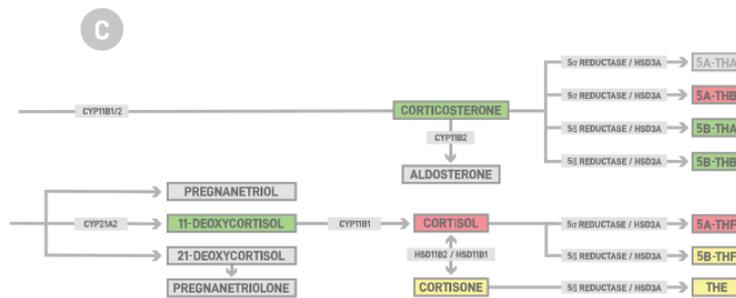
Progesterone Metabolites; urine



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

TEST NAME: HuMap (Hormone & Urinary Metabolites Assessment Profile)

Adrenal Corticoid Metabolites; urine



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

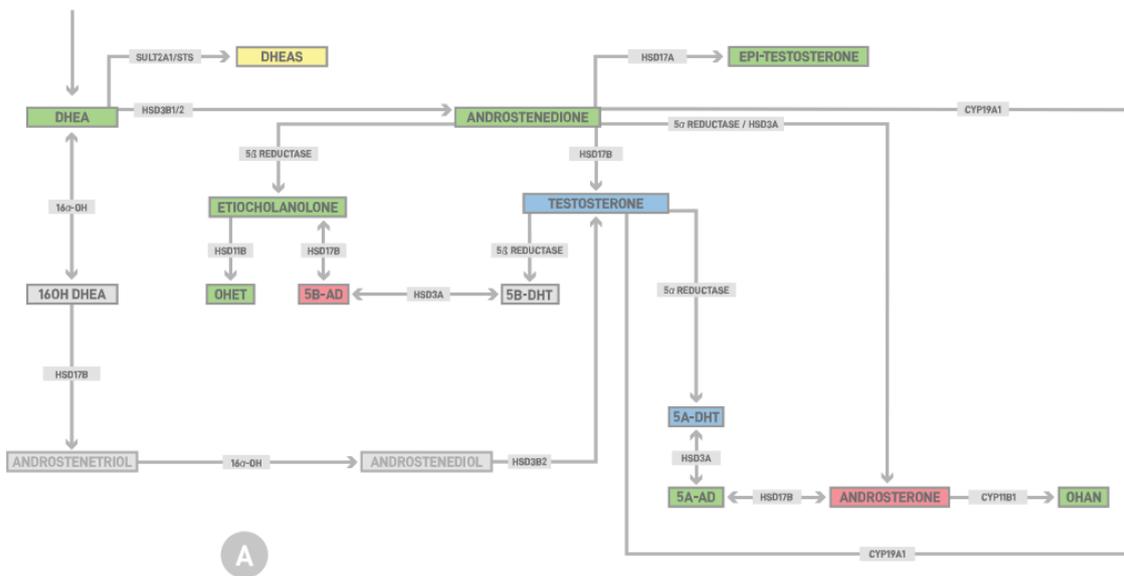
TEST NAME: HuMap (Hormone & Urinary Metabolites Assessment Profile)

Adrenal Corticoid Metabolites; urine

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

TEST NAME: HuMap (Hormone & Urinary Metabolites Assessment Profile)

Androgen Metabolites; urine



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



PATIENT: **XXXXXXXXXXXXXXXXXXXX**

TEST REF: **TST-NL-XXXX**

TEST NUMBER: T-NL-XXXXX (XXXXXXXXXXXX)

COLLECTED: XX/XX/XXXX

PRACTITIONER:

GENDER: XYZ

RECEIVED: XX/XX/XXXX

XXXXXXXXXXXXXXXXXXXX

AGE: XX

TESTED: XX/XX/XXXX

XXXXXXXXXXXXXXXXXXXX

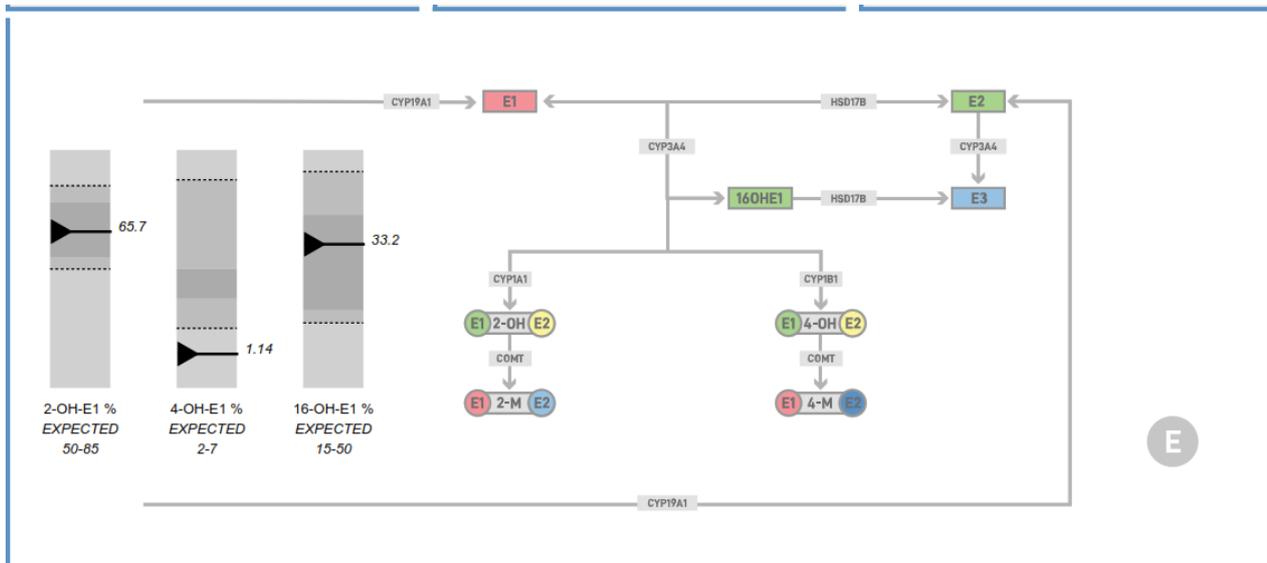
TEST NAME: HuMap (Hormone & Urinary Metabolites Assessment Profile)

Androgen Metabolites; urine

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 (5 α) / Etiocholanolone (5 β) (5 α Reductase Activity)	0.864					0.8 – 2.6
Testosterone / EPI-Testosterone	1.38					0.7 – 3

TEST NAME: HuMap (Hormone & Urinary Metabolites Assessment Profile)

Estrogen Metabolites; urine



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

TEST NAME: HuMap (Hormone & Urinary Metabolites Assessment Profile)

Estrogen Metabolites; urine

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

TEST NAME: HuMap (Hormone & Urinary Metabolites Assessment Profile)

Commentary

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 pregnenediol, progesterone's major metabolite, have been reported in 21 hydroxylase deficiency.

↓ 5A-PD

Lower levels of pregnenediol 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 virilizing adrenal hyperplasia. Additionally, hyperinsulinemia and hyperglycemia (metabolic syndrome) push 17-hydroxylation of progesterone.

↑ Pregnenetriol (5-PT)

5-pregnenetriol is a metabolite of 17 α -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 HSD3 β and HSD17 β 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.

↑ 5 β -Androstenediol (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

↑ 5 α -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.

TEST NAME: HuMap (Hormone & Urinary Metabolites Assessment Profile)

Commentary

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.

TEST NAME: HuMap (Hormone & Urinary Metabolites Assessment Profile)

Commentary

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.