Meridian Valley Lab

CLIA: 50D063590
6839 Fort Dent Way, Ste 206
Tukwila, WA 98188
Phone: 206.209.4200 / 855.405.TEST (8378)
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PACE24™

Patient Name: **REPORT, SAMPLE**
Accession Number: **514018**
Date of Birth: **1/1/1969** Age: **56** Sex: **F**
External ID: **ABC123**
Comments:

DoctorID: **6206 Dr. Sample Report ND**
The Healing Centre
6839 Fort Dent Way
Green City, WA 98188
United States
Phone: 206-209-4200 Fax: 206-209-4211

Test: **4018**
Date Collected: **7/16/2026**
Date Received: **7/16/2026**
Date Reported: **7/16/2026**
Tech:

Total Volume (TV) **2,600** mL

Notes:

Estrogens

	µg/24hr	Reference Intervals: Luteal	Reference Intervals: Post-Menopausal
Estrone (E1)	**6.1**	5.4 – 24.0	0.6 – 6.0
Estradiol (E2)	**1.8**	2.7 – 10.0	0.3 – 3.0
Estriol (E3)	**16.0**	5.0 – 50.0	0.8 – 7.3
Total Estrogens (E1+E2+E3)	**23.9**	16.0 – 67.0	3.0 – 14.0
Phase I Metabolites			
2-Hydroxy-estrone (2OH-E1)	**0.9**	2.6 – 32.0	0.6 – 14.0
16a-Hydroxy-estrone (16aOH-E1)	**1.2**	1.1 – 6.4	0.1 – 2.3
4-Hydroxy-estrone (4OH-E1)	**0.4**	0.1 – 1.7	0.1 – 1.1
Phase II Metabolites			
2-Methoxy-estrone (2MeO-E1)	**0.3**	(No References Ranges Established)	0.1 – 1.1
4-Methoxy-estrone (4MeO-E1)	**<DL**	(No References Ranges Established)	0.0 – 0.5

Urinary 2-Methoxyestrone and 4-Methoxyestrone are downstream methylated metabolites that are generally detectable only when estrone (E1) production is appreciable. In individuals with low estrone production, including most postmenopausal women not receiving HRT and most men, very low or undetectable levels are expected and typically reflect limited estrone substrate rather than impaired COMT activity or reduced estrogen methylation capacity. Because the Estrogen Methylation Ratio relates 2-methoxyestrone to its precursor, 2-hydroxyestrone, it provides complementary information regarding estrogen methylation that is less dependent on overall estrone production than the absolute methoxy metabolite concentrations.

Progestins

	µg/24hr	Reference Intervals: Luteal	Reference Intervals: Post-Menopausal
5ß-Pregnanediol (PD)	**4,071.0**	1281.0 – 3290.0	129.0 – 800.0

***DL - Detection Limit**

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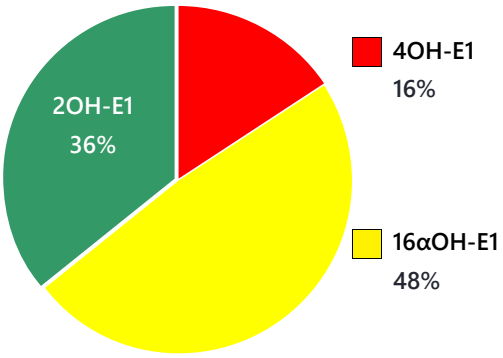
Estrogen Ratios

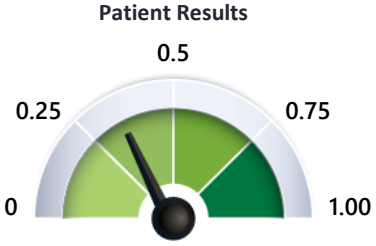
Estrogen Hydroxylation Profile (Phase I)



This graph illustrates the relative distribution of the measured Phase I estrogen hydroxylation metabolites (2-OH-E1, 16α-OH-E1, and 4-OH-E1). Because it displays the relative contribution of each metabolite rather than absolute concentrations, it complements the measured estrogen values by highlighting the distribution of estrogen hydroxylation independent of overall estrogen production.

In general, a favorable metabolic distribution is characterized by 2-OH-E1 contributing the greatest proportion, 16α-OH-E1 an intermediate proportion, and 4-OH-E1 the smallest proportion (typically <10% of total hydroxylated estrogens). The 16α-hydroxylation pathway contributes to normal estrogenic functions, and plays an important role in maintaining bone density as well as reproductive physiology; it should be interpreted in clinical context rather than simply classified as high or low. Because the 4-hydroxylation pathway has greater potential to generate reactive quinone metabolites, maintaining a relatively low contribution is generally considered favorable.

Estrogen Methylation Ratio (Phase II) (2MeO-E1 / 2OH-E1) **0.33**



The Estrogen Methylation Ratio provides insight into Phase II estrogen metabolism by assessing the conversion of 2-hydroxyestrone to 2-methoxyestrone, a reaction catalyzed by the catechol-O-methyltransferase (COMT) enzyme. Methylation facilitates the metabolism of catechol estrogens and contributes to normal estrogen metabolism.

In general, a favorable metabolic profile is characterized by greater conversion of 2-hydroxyestrone to 2-methoxyestrone, resulting in a higher Estrogen Methylation Ratio.

Interpretation is most meaningful when integrated with the Estrogen Hydroxylation Profile (Phase I), as hydroxylation determines catechol estrogen production while methylation facilitates their subsequent metabolism. Clinical interpretation should also consider factors that may influence methylation, including nutritional status, medications, and relevant genetic variants. If methylation is on the low end of the reference range, supplements that provide methyl donors and increase methylation may be helpful.

Androgens

	µg/24hr	Reference Intervals: Luteal	Reference Intervals: Post-Menopausal
Testosterone	**3.6**	5.0 – 18.0	2.0 – 10.0
Androstenedione	**0.3**	(No References Ranges Established)	(No References Ranges Established)
DHEA	**99.0**	20.0 – 1067.0	20.0 – 541.0
Androsterone (AN)	**347.0**	374.0 – 3510.0	303.0 – 1811.0

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		Reference Intervals	
		Luteal	Post-Menopausal
Etiocholanalone (ET)	**267.0**	790.0 – 3127.0	440.0 – 2165.0

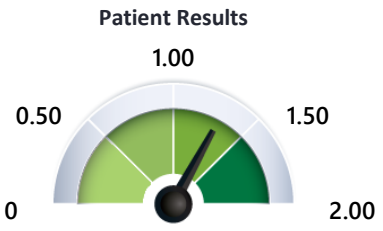
Corticosteroids

	µg/24hr	Luteal	Post-Menopausal
Cortisone (E)	**88.0**	49.0 – 235.0	42.0 – 222.0
Tetrahydrocortisone (THE)	**2,111.0**	487.0 – 4803.0	680.0 – 4839.0
Cortisol (F)	**35.0**	37.0 – 115.0	25.0 – 115.0
Tetrahydrocortisol (THF)	**786.0**	458.0 – 2169.0	540.0 – 2838.0
Allo-Tetrahydrocortisol (5a-THF)	**626.0**	142.0 – 1409.0	142.0 – 1380.0
THE + THF + 5a-THF (THE+THF+5a-THF)	**3,523.0**	1000.0 – 6000.0	1000.0 – 6000.0

Enzyme Activity Phenotype Assessment

5a-Reductase Phenotype

Andro/Etio Ratio (AN / ET) **1.30**



The Androsterone/Etiocholanolone Ratio provides insight into androgen reductase phenotype by reflecting the balance between androsterone, a predominantly 5α-reduced androgen metabolite, and etiocholanolone, a predominantly 5β-reduced androgen metabolite. Their relative formation is influenced by 5α-reductase and 5β-reductase, enzymes that direct androgen metabolism through these competing pathways.

A higher ratio indicates greater relative metabolism through the 5α-reduction pathway, whereas a lower ratio indicates greater relative metabolism through the 5β-reduction pathway. Neither pathway is inherently favorable or unfavorable; rather, the ratio characterizes an individual's predominant androgen metabolic phenotype and should be interpreted in the context of the clinical presentation and therapeutic goals. Interpretation should be integrated with the measured androgen concentrations and broader steroid metabolite profile, as well as factors that may influence androgen metabolism, including 5α-reductase inhibitors, androgen therapy, body composition, and metabolic health. A higher 5α-reduction phenotype is generally associated with greater conversion of testosterone to dihydrotestosterone (DHT), a more potent androgen that binds the androgen receptor with greater affinity than testosterone.

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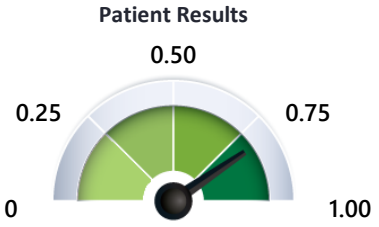
5a-Reductase Phenotype

5a-THF/THF Ratio **0.80**



The 5α-THF/THF Ratio provides insight into glucocorticoid reductase phenotype by reflecting the balance between 5α-tetrahydrocortisol (5α-THF) and tetrahydrocortisol (THF), the principal cortisol metabolites produced through 5α- and 5β-reductive metabolism, respectively. Their relative formation is influenced by 5α-reductase and 5β-reductase, providing complementary insight into glucocorticoid metabolism.

A higher ratio indicates greater relative metabolism through the 5α-reduction pathway, whereas a lower ratio indicates greater relative metabolism through the 5β-reduction pathway. Neither pathway is inherently favorable or unfavorable; rather, the ratio characterizes an individual's glucocorticoid metabolic phenotype and should be interpreted in the context of the clinical presentation and therapeutic goals. Because these ratios evaluate 5α-/5β-reductive metabolism across different steroid classes, interpretation alongside the Androsterone/Etiocholanolone Ratio provides greater confidence when characterizing an individual's overall 5α-reductase phenotype. Interpretation should also be integrated with the broader corticosteroid metabolite profile, as well as medications, supplements, and physiologic factors that may influence glucocorticoid metabolism, including 5α-reductase inhibitors, glucocorticoid therapy, and metabolic health.

11ß-Hydroxysteroid Dehydrogenase (11ß-HSD) Phenotype

Cortisone/Cortisol Ratio (11ß-HSD II) **2.51**

The Cortisone/Cortisol Ratio reflects the balance between cortisone, the less biologically active glucocorticoid, and cortisol, its biologically active counterpart. Their interconversion is regulated by the 11β-hydroxysteroid dehydrogenase (11β-HSD) enzymes. 11β-HSD1 regenerates cortisol fror cortisone in multiple tissues, whereas 11β-HSD2 converts cortisol to cortisone, particularly in the kidney where it protects mineralocorticoid receptors from excessive cortisol exposure.

A higher Cortisone/Cortisol Ratio reflects a greater relative contribution of cortisol inactivation to cortisone, whereas a lower ratio reflects a greater relative contribution of cortisone regeneration to cortisol. Within the expected physiologic range, relatively higher ratios generally reflect a more favorable glucocorticoid metabolic phenotype. Elevated ratios in the setting of adrenal insufficiency or low cortisol concentrations should be interpreted cautiously, as they may reflect reduced cortisol production rather than enhanced cortisol inactivation.

Note: this ratio is inverted from the Cortisol/Cortisone Ratio, the so-called “stress” ratio, as presented on legacy 24-hour urine panels.

Interpretation should be integrated with the measured cortisol, cortisone, and corticosteroid metabolite concentrations, as well as clinical factors that may influence glucocorticoid metabolism, including glucocorticoid therapy, licorice-containing products, chronic physiologic stress, insulin resistance, and alterations in hepatic or renal function.

Luteal reference ranges are derived from 20-29 year-old females. Post-menopause reference ranges are derived from 50-59 year-old females.

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CLIA # 38D0676504 | Frank J. Nordt, Ph.D

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