

Dihydrotestosterone, Serum

Overview

Useful For

Monitoring patients receiving 5-alpha reductase inhibitor therapy or chemotherapy

Evaluating patients with possible 5-alpha reductase deficiency

Testing Algorithm

For more information see **Steroid Pathways**.

Special Instructions

Steroid Pathways

Method Name

Liquid Chromatography-Tandem Mass Spectrometry (LC-MS/MS)

Portions of this test are covered by patents held by Quest Diagnostics

NY State Available

Yes

Specimen

Specimen Type

Serum

Specimen Required

Collection Container/Tube:

Preferred: Red top
Acceptable: Serum gel
Specimen Volume: 1 mL

Collection Instructions: Centrifuge and aliquot serum into a plastic vial.

Specimen Minimum Volume

0.6 mL

Reject Due To

| Gross | ОК |
|---------------|----|
| hemolysis | |
| Gross lipemia | ОК |
| Gross icterus | ОК |



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Specimen Stability Information

| Specimen Type | Temperature | Time | Special Container |
|---------------|--------------------------|---------|-------------------|
| Serum | Refrigerated (preferred) | 7 days | |
| | Frozen | 90 days | |

Clinical & Interpretive

Clinical Information

The principal prostatic androgen is dihydrotestosterone (DHT). Levels of DHT remain normal with aging, despite a decrease in the plasma testosterone, and are not elevated in benign prostatic hyperplasia (BPH).(1)

DHT is generated by reduction of testosterone by 5-alpha reductase. Two isoenzymes of 5-alpha reductase have been discovered. Type 1 is present in most tissues in the body where 5-alpha reductase is expressed, and is the dominant form in sebaceous glands. Type 2 is the dominant isoenzyme in genital tissues, including the prostate.

Androgenetic alopecia (AGA; male-pattern baldness) is a hereditary and androgen-dependent progressive thinning of the scalp hair that follows a defined pattern.(2) While the genetic involvement is pronounced, but poorly understood, major advances have been achieved in understanding the principal elements of androgen metabolism that are involved. DHT may be related to baldness. High concentrations of 5-alpha reductase have been found in frontal scalp and genital skin and androgen-dependent processes are predominantly due to the binding of DHT to the androgen receptor (AR). Since the clinical success of treatment of AGA with modulators of androgen metabolism or hair growth promoters is limited, sustained microscopic follicular inflammation with connective tissue remodeling, eventually resulting in permanent hair loss, is considered a possible cofactor in the complex etiology of AGA.

Currently available AGA treatment modalities with proven efficacy are oral finasteride, a competitive inhibitor of 5-alpha reductase type 2, and topical minoxidil, an adenosine triphosphate-sensitive potassium channel opener that has been reported to stimulate the production of vascular endothelial growth factor in cultured dermal papilla cells.

Currently, many patients with prostate disease receive treatment with a 5-alpha reductase inhibitor such as finasteride (Proscar) to diminish conversion of DHT from testosterone.

For more information see Steroid Pathways.

Reference Values

Males

Cord blood: < or =100 pg/mL < or =6 months: < or =1,200 pg/mL

Tanner Stages

| Mean | Age | Reference range (pg/mL) |
|--------------------|------------|-------------------------|
| Stage I (>6 months | 7.1 years | < or =50 |
| and prepubertal) | | |
| Stage II | 12.1 years | < or =200 |



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| Stage III | 13.6 years | 80-330 |
|-----------|------------|---------|
| Stage IV | 15.1 years | 220-520 |
| Stage V | 18 years | 240-650 |

>19 years: 112-955 pg/mL

Females

Cord blood: < or =50 pg/mL < or =6 months: < or =1,200 pg/mL

Tanner Stages

| Mean | Age | Reference range (pg/mL) |
|--------------------|------------|-------------------------|
| Stage I (>6 months | 7.1 years | < or =50 |
| and prepubertal) | | |
| Stage II | 10.5 years | < or =300 |
| Stage III | 11.6 years | < or =300 |
| Stage IV | 12.3 years | < or =300 |
| Stage V | 14.5 years | < or =300 |

20-55 years: < or =300 pg/mL >55 years: < or =128 pg/mL

- 1. Pang S, Levine LS, Chow D, et al: Dihydrotestosterone and its relationship to testosterone in infancy and childhood. J Clin Endocrinol Metab 1979;48:821-826
- 2. Stanczyk FZ: Diagnosis of hyperandrogenism: biochemical criteria. Best Pract Res Clin Endocrinol Metab 2006;20(2):177-191

Interpretation

Patients taking 5-alpha reductase inhibitor have decreased dihydrotestosterone (DHT) serum levels.

Patients with genetic 5-alpha reductase deficiency (a rare disease) also have reduced DHT serum levels.

DHT should serve as the primary marker of peripheral androgen production. However, because it is metabolized rapidly and has a very high affinity for sex hormone-binding globulin (SHBG), DHT does not reflect peripheral androgen action. Instead, its distal metabolite, 3-alpha, 17-beta-androstanediol glucuronide, serves as a better marker of peripheral androgen action.

For more information see **Steroid Pathways**.

Cautions

Patients with benign prostatic hyperplasia (BPH) or prostatic cancer may not have elevated dihydrotestosterone (DHT) levels even though growth of the prostate gland may be stimulated by DHT.

Clinical Reference

- 1. Bartsch G, Rittmaster RS, Klocker H: Dihydrotestosterone and the concept of 5 alpha-reductase inhibition in human benign prostatic hyperplasia. World J Urol 2002;19(6):413-425
- 2. Trueb RM: Molecular mechanisms of androgenetic alopecia. Exp Gerontol 2002;37(8-9):981-990
- 3. Singh SM, Gauthier S, Labrie F: Androgen receptor antagonists (antiandrogens): structure-activity relationships. Curr Med Chem 2000;7(2):211-247



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- 4. Rhodes L, Harper J, Uno H, et al: The effects of finasteride (Proscar) on hair growth, hair cycle stage, and serum testosterone and dihydrotestosterone in adult male and female stumptail macaques (*Macaca arctoides*). J Clin Endocrinol Metab 1994;79:991-996
- 5. Gustafsson O, Norming U, Gustafsson S, et al: Dihydrotestosterone and testosterone levels in men screened for prostate cancer: a study of a randomized population. Br J Urol 1996;77:433-440
- 6. van der Veen A, van Faassen M, de Jong WHA, et al: Development and validation of a LC-MS/MS method for the establishment of reference intervals and biological variation for five plasma steroid hormones. Clin Biochem. 2019 Jun;68:15-23. doi: 10.1016/j.clinbiochem.2019.03.013

Performance

Method Description

Deuterated stable isotope of dihydrotestosterone (DHT) is added to a 0.5-mL serum sample as internal standard. The DHT and internal standard are extracted from the sample by solid phase extraction. This is followed by conventional liquid chromatography on a multiplexed LC System and analysis on a tandem mass spectrometer equipped with an electrospray ionizer. (Lagerstedt SA, O'Kane DJ, Singh RJ: Measurement of plasma free metanephrine and normetanephrine by liquid chromatography-tandem mass spectrometry for diagnosis of pheochromocytoma. Clin Chem 2004;50[3]:603-611)

PDF Report

No

Day(s) Performed

Monday, Wednesday, Friday

Report Available

2 to 8 days

Specimen Retention Time

2 weeks

Performing Laboratory Location

Rochester

Fees & Codes

Fees

- Authorized users can sign in to <u>Test Prices</u> for detailed fee information.
- Clients without access to Test Prices can contact <u>Customer Service</u> 24 hours a day, seven days a week.
- Prospective clients should contact their account representative. For assistance, contact <u>Customer Service</u>.

Test Classification



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This test was developed and its performance characteristics determined by Mayo Clinic in a manner consistent with CLIA requirements. It has not been cleared or approved by the US Food and Drug Administration.

CPT Code Information

82642

G0480 (if appropriate)

LOINC® Information

| DHTS Dihydrotestosterone, S 1848-1 | Test ID | Test Order Name | Order LOINC® Value |
|------------------------------------|---------|------------------------|--------------------|
| , , | DHTS | Dihydrotestosterone, S | 1848-1 |

| Result ID | Test Result Name | Result LOINC® Value |
|-----------|------------------------|---------------------|
| 81479 | Dihydrotestosterone, S | 1848-1 |