TEST ID: DHEA_
DEHYDROEPIANDROSTERONE (DHEA), SERUM

USEFUL FOR
- Diagnosing and differential diagnosis of hyperandrogenism (in conjunction with measurements of other sex steroids).
- An initial screen in adults might include dehydroepiandrosterone/dehydroepiandrosterone sulfate and bioavailable testosterone measurement. Depending on results, this may be supplemented with measurements of sex hormone-binding globulin and occasionally other androgenic steroids (e.g., 17-hydroxyprogesterone).
- An adjunct in the diagnosis of congenital adrenal hyperplasia; dehydroepiandrosterone/dehydroepiandrosterone sulfate measurements play a secondary role to the measurements of cortisol/cortisone, 17 alpha-hydroxyprogesterone, and androstenedione.
- Diagnosing and differential diagnosis of premature adrenarche.

CLINICAL INFORMATION
Dehydroepiandrosterone (DHEA) is the principal human C-19 steroid. DHEA has very low androgenic potency, but serves as the major direct or indirect precursor for most sex steroids. DHEA is secreted by the adrenal gland and production is at least partly controlled by adrenocorticotropic hormone (ACTH). The bulk of DHEA is secreted as a 3-sulfocojugate dehydroepiandrosterone sulfate (DHEAS). Both hormones are albumin bound, but DHEAS binding is much tighter. As a result, circulating concentrations of DHEAS are much higher (> 100-fold) compared to DHEA. In most clinical situations, DHEA and DHEAS results can be used interchangeably. In gonads and several other tissues, most notably skin, steroid sulfatases can convert DHEAS back to DHEA, which can then be metabolized to stronger androgens and to estrogens.

During pregnancy, DHEA/DHEAS and their 16-hydroxylated metabolites are secreted by the fetal adrenal gland in large quantities. They serve as precursors for placental production of the dominant pregnancy estrogen, estriol. Within weeks after birth, DHEA/DHEAS levels fall by 80% or more and remain low until the onset of adrenarche at age 7 or 8 in girls and age 8 or 9 in boys. Adrenarche is a poorly understood phenomenon, peculiar to higher primates, that is characterized by a gradual rise in adrenal androgen production. It precedes puberty, but is not casually linked to it. Early adrenarche is not associated with early puberty or with any reduction in final height or overt androgenization. However, girls with early adrenarche may be at increased risk of polycystic ovarian syndrome as adults and some boys may develop early penile enlargement.

REFERENCE VALUES
- Premature: < 40 ng/mL*
- 0–1 day: < 11 ng/mL*
- 2–6 days: < 8.7 ng/mL*
- 7 days–1 month: < 5.8 ng/mL*
- > 1–23 months: < 2.9 ng/mL*
- 2–5 years: < 2.3 ng/mL
- 6–10 years: < 3.4 ng/mL
- 11–14 years: < 5.0 ng/mL
- 15–18 years: < 6.6 ng/mL
- 19–30 years: < 13 ng/mL
- 31–40 years: < 10 ng/mL
- 41–50 years: < 8.0 ng/mL
- 51–60 years: < 6.0 ng/mL
- ≥ 61 years: < 5.0 ng/mL


ANALYTIC TIME
2 days

CONTENT AND VALUES SUBJECT TO CHANGE. SEE THE MAYO MEDICAL LABORATORIES TEST CATALOG FOR CURRENT INFORMATION.
Following adrenarche, DHEA/DHEAS levels increase until the age of 20 to a maximum roughly comparable to that observed at birth. Levels then decline over the next 40 to 60 years to around 20% of peak levels. The clinical significance of this age-related drop is unknown and trials of DHEA/DHEAS replacement in the elderly have not produced convincing benefits. However, in young and old patients with primary adrenal failure, the addition of DHEA/DHEAS to corticosteroid replacement has been shown in some studies to improve mood, energy, and sex drive.

Elevated DHEA/DHEAS levels can cause symptoms or signs of hyperandrogenism in women. Men are usually asymptomatic, but through peripheral conversion of androgens to estrogens can occasionally experience mild estrogen excess. Most mild-to-moderate elevations in DHEAS levels are idiopathic. However, pronounced elevations of DHEA/DHEAS may be indicative of androgen-producing adrenal tumors. In small children, congenital adrenal hyperplasia (CAH) due to 3 beta-hydroxysteroid dehydrogenase deficiency is associated with excessive DHEA/DHEAS production. Lowerer outerations may be observed in 21-hydroxylase deficiency (the most common form of CAH) and 11 beta-hydroxylase deficiency. By contrast, steroidal acute regulatory protein (STAR) or 17 alpha-hydroxylase deficiency is characterized by low DHEA/DHEAS levels.

See Steroid Pathways in Special Instructions.

**INTERPRETATION**

Elevated dehydroepiandrosterone (DHEA)/dehydroepiandrosterone sulfate (DHEAS) levels indicate increased adrenal androgen production. Mild elevations in adults are usually idiopathic, but levels >5-fold or more of the upper limit of normal can suggest the presence of an androgen-secreting adrenal tumor. DHEA/DHEAS levels are elevated in >90% of patients with such tumors. This is particularly true for androgen-secreting adrenal carcinomas, as they have typically lost the ability to produce downstream androgens, such as testosterone. By contrast, androgen-secreting adrenal adenomas may also produce excess testosterone and secrete lesser amounts of DHEA/DHEAS.

Patients with congenital adrenal hyperplasia (CAH) may show very high levels of DHEA/DHEAS, often 5-fold to 10-fold elevations. However, with the possible exception of 3 beta-hydroxysteroid dehydrogenase deficiency, other steroid analytes offer better diagnostic accuracy than DHEA/DHEAS measurements. Consequently, DHEA/DHEAS testing should not be used as the primary tool for CAH diagnosis. Similarly, discovering a high DHEA/DHEAS level in an infant or child with symptoms or signs of possible CAH should prompt additional testing, as should the discovery of very high DHEA/DHEAS levels in an adult. In the latter case, adrenal tumors need to be excluded and additional adrenal steroid profile testing may assist in diagnosing nonclassical CAH.

See Steroid Pathways in Special Instructions.

**CLINICAL REFERENCE**