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DEHYDROEPIANDROSTERONE SULPHATE (DHEAS) INFORMATION SHEET

PHYSIOLOGY

Dehydroepiandrosterone (DHEA) and its sulphated form DHEAS are the most abundant steroid products of the adrenal glands. The adrenal is the sole source of these steroids in women but in men there is also a small contribution from the testes. Formation of DHEA is stimulated by ACTH producing a significant diurnal variation in serum concentrations. DHEA is rapidly converted to DHEAS by an enzyme present in the adrenals, liver and small intestine. DHEAS is present at concentrations greater than 200 times that of DHEA and has a longer half-life which largely removes the diurnal variation. Neither form has significant androgenic activity, but they are precursors to about 50% of androgens in men, 75% of active oestrogens in pre-menopausal women and 100% in post-menopausal women.

It is more clinically useful to measure DHEAS rather than DHEA due to the higher serum concentrations and reduced daily variation. There are few indications for the measurement of DHEA.

DHEA is a 19 carbon steroid with a molecular weight of 288 and a half-life in plasma of about 1 - 3 hours. The molecular weight of DHEAS is 371 and the half life is about 10 - 20 hours. DHEA is formed from pregnenolone by the enzyme 17,20 desmolase and metabolised to androstenedione or testosterone by 3 beta- or 17 beta-hydrosteroid dehydrogenase respectively. Hydrosteroid sulphatase converts DHEA to DHEAS and sulphohydrolase reverses this reaction.

PATHOLOGY

Raised levels of DHEAS are found in the plasma of patients with adrenal tumours or with congenital adrenal hyperplasia. DHEAS may also be slightly elevated in patients with polycystic ovaries, supporting an adrenal component to the virilisation seen in this condition. HCG-production tumours in men may lead to increased testicular DHEA production.

DHEAS is usually undetectable with adrenal insufficiency or panhypopituitarism. Concentrations are slightly decreased in pregnancy and with oral contraceptive use and markedly decreased following glucocorticoid administration. There is a gradual decline from early adulthood with values in the 7th decade about 20-30% of young adult values. Low circulating concentrations are seen with severe illness.