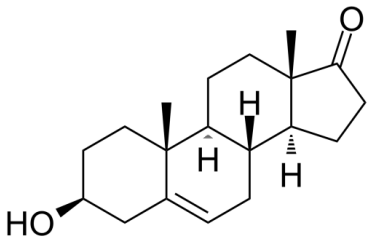
Data Sheet

Product Name:	DHEA
Cat. No.:	CS-1667
CAS No.:	53-43-0
Molecular Formula:	C19H28O2
Molecular Weight:	288.42
Target:	Androgen Receptor; Endogenous Metabolite
Pathway:	Metabolic Enzyme/Protease; Others
Solubility:	Ethanol : 50 mg/mL (173.36 mM; Need ultrasonic); H2O : < 0.1 mg/mL (insoluble); DMSO : 50 mg/mL (173.36 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

DHEA (Prasterone) is one of the most abundant steroid hormones. DHEA (Prasterone) mediates its action via multiple signaling pathways involving specific membrane receptors and via transformation into androgen and estrogen derivatives (e.g., androgens, estrogens, 7α and 7β DHEA, and 7α and 7β epiandrosterone derivatives) acting through their specific receptors. IC50 & Target: Androgen receptor[1] **In Vitro:** DHEA (Prasterone) is an effective antiapoptotic factor, reversing the serum deprivation-induced apoptosis in prostate cancer cells (DU145 and LNCaP cell lines) as well as in colon cancer cells (Caco2 cell line). DHEA (Prasterone) significantly reduces serum deprivation-induced apoptosis in all 3 cancer cell types, quantitated with the APOPercentage assay (apoptosis is reduced from 0.587±0.053 to 0.142±0.0016 or 0.059±0.002 after treatment for 12 hours with DHEA or NGF, respectively; n=3, P<0.01), and by flow cytometry analysis (FACS) for DU145 cells. The antiapoptotic effect of DHEA is dose dependent with an EC50 at nanomolar concentrations (EC_{50}: 11.2±3.6 nM and 12.4±2.2 nM in DU145 and Caco2 cells, respectively)[1]. DHEA (Prasterone) is the principal sex steroid precursor in humans and can be converted directly to androgens. DHEA (Prasterone) (≥1 μM) causes a dose-dependent inhibition of Chub-S7 proliferation, as assessed by thymidine incorporation assays. DHEA (Prasterone) treatment inhibits expression of the key glucocorticoid-regulating genes H6PDH (≥100 nM) and HSD11B1 (≥1 μM) in differentiating preadipocytes in a dose-dependent manner. In keeping with this finding, DHEA (Prasterone) treatment (≥1 μM) results in a marked reduction in 11β-HSD1 oxoreductase activity (≥1 μM) and a concurrent increase in dehydrogenase activity at the highest DHEA dose used (25 μM DHEA) in differentiated adipocytes[2]. **In Vivo:** DHEA (Prasterone) in the diet (0.45 % w/w) of male B6 mice (groups of five mice) treated for 8 weeks led to significant decreases in body temperature compared with mice fed the control AIN-76A diet. A similar comparison indicated that control and pair-fed mice are also significantly different. Animals fed DHEA (Prasterone) have significantly lower temperatures than mice fed the control diet 26/29 times tested; mice pair fed to those on the DHEA (Prasterone) diet are less affected, with 8/29 values significantly lower than in mice fed AIN-76A ad libitum. The temperatures of mice fed DHEA (Prasterone) or pair fed to DHEA (Prasterone) are significantly different 21/29 times tested. Body weights are significantly greater in mice fed the control diet than in mice fed DHEA or pair fed to DHEA (Prasterone). Food intake (grams per day) from cages are averaged for each week (n=7), except for Week 9 (n=3). The amount of food intake is significantly decreased in mice fed DHEA (Prasterone). By design, mice pair fed to DHEA (Prasterone) ate about the same amount. Thus, it appears that DHEA (Prasterone) reduces body temperature by food restriction and by a separate mechanism[3].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: DHEA (Prasterone) is dissolved in DMSO and stored, and then diluted with appropriate medium before use[2].[2]Chub-S7 preadipocytes and human primary preadipocytes are seeded into a 24-well plate at densities 1×10^5 and 2.5×10^5 respectively. Following overnight culture, medium is supplemented with DHEA, androstenediol, or DHEA (Prasterone) (0-100 μM). Following 24-, 48-, or 72 h incubation, cell proliferation is assessed by incubation with radiolabeled thymidine (0.2 μCi/well) for the final 6 h of culture. Proteins are precipitated with TCA, and cells are scraped in NaOH. The respective content of radiolabeled nuclear material in

the resulting lysates is analyzed by scintillation counting[2]. **Animal Administration:** DHEA is prepared in 0.9% NaCl (Mice)[3].[3]Mice[3] Mice are fed Purina Lab Chow until the start of experiments (Day 0). Groups of five mice are then fed pelleted AIN-76A diet containing either no additive or DHEA (0.45% w/w) between 0900 and 1000 hr. Diets are stored at 4°C for no longer than six months to maintain optimal activity. Mice are given the diets ad libitum, except for mice that are pair fed to mice treated with DHEA (Prasterone). The amounts of AIN-76A diet the pair-fed mice received are determined by the weight of food consumed by the DHEA-fed mice on a daily basis. Body weights (grams) are measured at different time points starting at Day 1 and ending at Day 59. Daily food intakes (grams per day) are determined by weighing the food consumed per cage of five mice. The mean±SEM values are calculated for weeks 1 to 8 (n=7); week 9 had only 3 days.

References:

[1]. Anagnostopoulou V, et al. Differential effects of dehydroepiandrosterone and testosterone in prostate and colon cancer cell apoptosis: the role of nerve growth factor (NGF) receptors. Endocrinology. 2013 Jul;154(7):2446-56.

[2]. McNelis JC, et al. Dehydroepiandrosterone exerts anti-glucocorticoid action on human preadipocyte proliferation, differentiation and glucose uptake. Am J Physiol Endocrinol Metab. 2013 Nov 1;305(9):E1134-44.

[3]. Catalina F, et al. Decrease of core body temperature in mice by dehydroepiandrosterone. Exp Biol Med (Maywood). 2002 Jun;227(6):382-8.

CAIndexNames:

Androst-5-en-17-one, 3-hydroxy-, (3β)-

SMILES:

O=C1CC[C@@]2([H])[C@]3([H])CC=C4C[C@@H](O)CC[C@]4(C)[C@@]3([H])CC[C@@]21C

Tel: 732-484-9848 Fax: 888-484-5008 E-mail: sales@ChemScene.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA