Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

DHEA

Catalog No: tcsc1667

Available Sizes

Size: 100mg

Size: 500mg

Specifications

CAS No:
53-43-0

Formula:
$C_{19}H_{28}O_2$

Pathway:
Others;Metabolic Enzyme/Protease

Target:
Androgen Receptor;Endogenous Metabolite

Purity / Grade:
>98%

Solubility:
DMSO : 50 mg/mL (173.36 mM; Need ultrasonic)

Alternative Names:
Prasterone;Dehydroisoandrosterone;Dehydroepiandrosterone

Observed Molecular Weight:
288.42

Product Description

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,

Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

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, P50: 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].