

Product Data Sheet

Product Name: Cortisone acetate

Cat. No.: GC16772

Chemical Properties

Cas. No. 50-04-4

Chemical Name [2-[(8S,9S,10R,13S,14S,17R)-17-hydroxy-10,13-dimethyl-3,11-dioxo-1,2,6,7,8,9,12,14,15,16-decahydrocyclopenta[a]phenanthren-17-yl]-2-oxoethyl] acetate

SMILES CC(=O)OCC(=O)C1(CCC2C1(CC(=O)C3C2CCC4=CC(=O)CCC34C)C)O

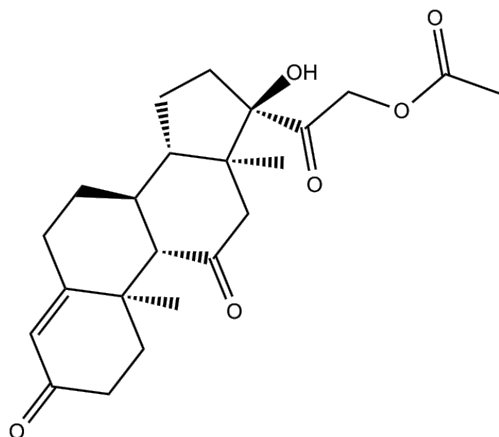
Formula C₂₃H₃₀O₆ M.Wt 402.48

Solubility ≥ 7.03mg/mL in DMSO with gentle warming Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Background

Cortisone acetate (17-hydroxy-11-dehydrocorticosterone), a 21-carbon steroid hormone, is one of the main hormones released by the adrenal gland in response to stress. IC₅₀ Value: Target:

Glucocorticoid Receptor in vitro: Cortisone suppressed this apoptosis at a concentration range of 1-10,000 ng/ml (2.8-28,000 nM) dose-dependently. Suppression of cortisol-induced apoptosis by cortisone was consistently observed in PBMCs derived from 16 healthy subjects. Examination for

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

Product Data Sheet

inhibitory activities of the steroids against [3H]dexamethasone binding to PBMCs suggested that cortisone can bind cellular GC-receptors (GC-Rs), but the affinity of cortisone to GCRs is 1/30 or less than that of cortisol [1]. Apoptosis was also readily induced in primary cultures of third trimester decidual cells when treated with cortisol, cortisone, or dexamethasone (all 100 nM for 24 h) [2]. *in vivo*: The effects of a single dose of cortisone acetate (5 or 10 mg/100 g body weight) on B cells were examined in young chickens. A dose-dependent increase in numbers of circulating B lymphocytes and a change in their Ig-class distribution were followed by parallel increase in splenic plasma cells and serum immunoglobulins. The higher dose of cortisone produced changes in Bmu and Bgamma cells, whereas the lower dose primarily affected Bmu cells [3]. Adult female CD-1 mice received daily injections of cortisone acetate (0--50 mg/kg subcutaneously) and/or amphotericin B (0--12.5 mg/kg intraperitoneally) in a checkerboard combination dosage pattern for 30 days. Dosages of amphotericin B and cortisone acetate that produced little or no mortality individually produced significant (*P* less than 0.005) mortality in combination [4]. **Toxicity:** Oral use of cortisone has a number of potential systemic side-effects: hyperglycemia, insulin resistance, diabetes mellitus, osteoporosis, anxiety, depression, amenorrhoea, cataracts and glaucoma, among other problems.

References:

- [1]. Hirano T, et al. Cortisone counteracts apoptosis-inducing effect of cortisol in human peripheral-blood mononuclear cells. *Int Immunopharmacol*. 2001 Nov;1(12):2109-15.
- [2]. Chan J, et al. Glucocorticoid-induced apoptosis in human decidua: a novel role for 11beta-hydroxysteroid dehydrogenase in late gestation. *J Endocrinol*. 2007 Oct;195(1):7-15.
- [3]. Rusu VM, et al. *In vivo* effects of cortisone on the B cell line in chickens. *J Immunol*. 1975 Nov;115(5):1370-4.
- [4]. Kisch AL, et al. Synergistic nephrotoxicity of amphotericin B and cortisone acetate in mice. *J Infect Dis*. 1978 Jun;137(6):789-94.

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA