

Product Data Sheet

Product Name: Allopregnanolone

Cat. No.: GC14192

Chemical Properties

Cas. No. 516-54-1

Chemical Name 1-((3R,5S,8R,9S,10S,13S,14S,17S)-3-hydroxy-10,13-dimethylhexadecahydro-1H-cyclopenta[a]phenanthren-17-yl)ethanone

SMILES CC(=O)[C@H]1CC[C@@H]2[C@@]1(CC[C@H]3[C@H]2CC=C4[C@@]3(CC[C@@H](C4)O)C[C@H]5CC[C@@H]2[C@]5(C)C)C

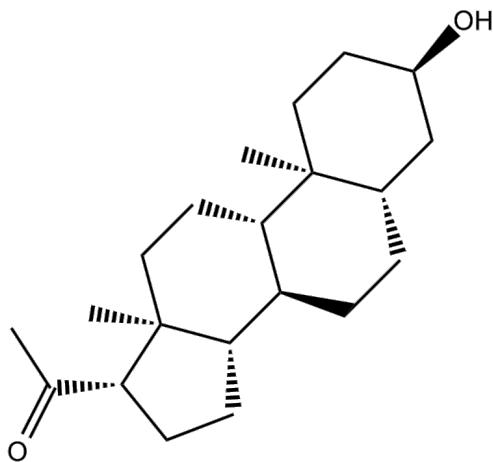
Formula $C_{21}H_{34}O_2$ M.Wt 318.49

Solubility Acetonitrile: 1 mg/ml, Ethanol: 1 mg/ml, Methanol: 1 mg/ml Storage Store at RT

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 sizes: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

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Cell lines Stably transfected differentiated Neuro-2A cells

Stably transfected differentiated Neuro-2A cells, at final confluence of about 80%, were treated with vehicle, Allopregnanolone, THDOC, PGL, PGL-S, DHEA, DHEA-S at concentrations from 0.01 to 30 μ M for 5 days. Next, the Preparation Method influence of all the above-listed neurosteroids at the same concentrations (0.01-30 μ M) present in the medium for 5 days, on forskolin-stimulated CAT activity was measured in Neuro-2A cells. Forskolin (25 μ M) was added to the culture medium 24 h before harvesting of the cells.

Reaction
Conditions 0.01 to 30 μ M for 5 days

Applications Allopregnanolone and THDOC treatment inhibited in a concentration-dependent manner (1-30 μ M) against the CRR-CAT activity. Lower concentrations of Allopregnanolone and THDOC (0.01-0.3 μ M) were inactive.

Animal experiment [2]:

Animal models Female Sprague Dawley rats

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Preparation Method To test whether suppressed HPA axis responses to IL-1 β could be mimicked in virgin rats with allopregnanolone treatment, blood samples and brains were collected from virgin rats treated with allopregnanolone (3 and 1 mg/kg, s.c., 20 and 2 h before IL-1 β , respectively) or vehicle (15% ethanol in corn oil; 0.5 ml/kg). To verify that the effects of finasteride on HPA responses to IL-1 β seen in pregnant rats were attributable to the actions of allopregnanolone, blood samples and brains were also collected from pregnant (day 20-21) rats treated with finasteride and either allopregnanolone or vehicle.

Dosage form 3 and 1 mg/kg, s.c.

Applications There was a significant effect of allopregnanolone treatment on ACTH secretion in the virgin rats. There was no difference in basal plasma ACTH concentration between vehicle and allopregnanolone-pretreated virgin rats. IL-1 β significantly increased ACTH secretion within 15 min in both the vehicle-treated virgins (4.0-fold increase) and the allopregnanolone treated virgins (2.0-fold increase); the response was markedly reduced in the allopregnanolone-treated group (57% of the peak response in the vehicle group).

References:

- [1] :Budziszewska B, Zajac A, Basta-Kaim A, et al. Effects of neurosteroids on the human corticotropin-releasing hormone gene[J]. Pharmacological Reports, 2010, 62(6): 1030-1040.
[2] :Brunton P J, McKay A J, Ochędalski T, et al. Central opioid inhibition of neuroendocrine stress responses in pregnancy in the rat is induced by the neurosteroid allopregnanolone[J]. Journal of Neuroscience, 2009, 29(20): 6449-6460.

Background

Allopregnanolone, a 3 α , 5 α progesterone metabolite, acts as a potent allosteric modulator of the γ -aminobutyric acid type A (GABAA) receptor, exerts antidepressant and neuroprotective action [1]. The levels of this neuroactive steroid as well as its effects are sexdimorphic [2].

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Allopregnanolone (3 μ M) present in the culture medium for 5 days did not change the amount of active, phosphorylated form of protein kinase B (PKB, Akt) and extracellular signal-regulated kinase (ERK). Allopregnanolone and THDOC inhibited basal and forskolin-induced CRH gene promoter activity in the differentiated Neuro-2A cells [1].

Allopregnanolone treatment exerted protective effects also in experimental models of neurodegeneration. For instance, Allopregnanolone (2 mg/kg, i.p.) is protective against kainic acid-induced excitotoxicity in the hippocampus in vivo [3]. In the nucleus accumbens of learned helplessness rats (i.e., an experimental model of depression) the astroglial glutamate transporter-1 and glutamine synthetase system is normalized by Allopregnanolone treatment [4]. In mood and anxiety disorders, Allopregnanolone (3 mg/kg and 1 mg/kg, s.c) treatment shows sex specific features. This neuroactive steroid attenuates in females, but not in males, the HPA axis responses to interleukin-1 β in adult prenatally stressed rats [5].

References:

- [1]. Budziszewska B, Zając A, Basta-Kaim A, et al. Effects of neurosteroids on the human corticotropin-releasing hormone gene[J]. Pharmacological Reports, 2010, 62(6): 1030-1040.
- [2]. Diviccaro S, Cioffi L, Falvo E, et al. Allopregnanolone: An overview on its synthesis and effects[J]. Journal of Neuroendocrinology, 2022
- [3]. Ciriza I, Carrero P, Frye C A, et al. Reduced metabolites mediate neuroprotective effects of progesterone in the adult rat hippocampus. The synthetic progestin medroxyprogesterone acetate (Provera) is not neuroprotective[J]. Journal of neurobiology, 2006, 66(9): 916-928.
- [4]. Nangaku M, Yoshino K, Oda Y, et al. Astroglial glutamate transporter 1 and glutamine synthetase of the nucleus accumbens are involved in the antidepressant-like effects of allopregnanolone in learned helplessness rats[J]. Behavioural Brain Research, 2021, 401: 113092.
- [5]. Brunton P J, Donadio M V, Yao S T, et al. 5 α -Reduced neurosteroids sex-dependently reverse central prenatal programming of neuroendocrine stress responses in rats[J]. Journal of Neuroscience, 2015, 35(2): 666-677.

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