Flunisolide

Cat. No.: HY-B1121

CAS No.: 3385-03-3

Molecular Formula: $C_{24}H_{31}FO_6$

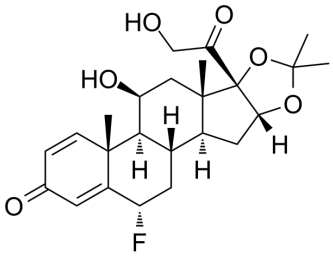
Molecular Weight: 434.5

Target: Glucocorticoid Receptor; Apoptosis

Pathway: Immunology/Inflammation; Vitamin D Related/Nuclear Receptor; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (287.69 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3015 mL	11.5075 mL	23.0150 mL
	5 mM	0.4603 mL	2.3015 mL	4.6030 mL
	10 mM	0.2301 mL	1.1507 mL	2.3015 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (4.79 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.79 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.79 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Flunisolide is a corticosteroid, which is an orally active glucocorticoid receptor activator with anti-inflammatory activity. Flunisolide can induce eosinophil apoptosis, and is used for the research of asthma or rhinitis, and inflammation[1][2].

In Vitro

Flunisolide (0.1-10 μM, 1 h) inhibits lung fibroblast (Isolated from lung) activation[1].
Flunisolide (10 μM, 24 h) reduces MMP-9, TIMP-1, TGF-β and fibronectin release by sputum cells (isolated frommild to moderate asthmatics), and induces sputum eosinophil apoptosis[2].
Flunisolide (0.1-10 μM μM, 24 h) effectively inhibits ICAM-1 expression and GM-CSF and IL-5 release induced by TNF-alpha in

BEAS-2B cells[3].
Flunisolide (115 μM, 0-3 h) can be transported in a polarized way in the apical (ap) to basolateral (bl) direction in Calu-3 cells and is demonstrated to be ATP-dependent[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Apoptosis Analysis[2]

Cell Line:	Eosinophil
Concentration:	10 μM
Incubation Time:	24 h
Result:	Induced sputum eosinophil apoptosis.

In Vivo

Flunisolide (Intranasal administration, 0.3–10 μg/mouse, daily, from days 21-27) inhibits lung inflammation, fibrosis, and airway hyper-reactivity, also improves clearance of silica particles from the lungs in silicotic mice[1].
Flunisolide (Intranasal administration, 0.3–10 μg/mouse, daily, from days 21-27) inhibits silica-induced macrophage and myofibroblast accumulation in the lung tissue[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Swiss Webster mice (instilled, intranasally, with crystalline silica, 10 mg/50 μL, particle size 0.5-10 μm)[1]
Dosage:	0.3-10 μg/mouse, daily, from days 21–27
Administration:	Intranasal administration
Result:	Reduced both granulomatous response, collagen deposition, concerning granuloma formation caused by silica particles. Reduced the number of F4/80 and α-SMA positive cells.

CUSTOMER VALIDATION

- Drug Test Anal. 2020 Aug 27.

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REFERENCES

[1]. Tatiana Paula Teixeira Ferreira, et al. Intranasal Flunisolide Suppresses Pathological Alterations Caused by Silica Particles in the Lungs of Mice. Front Endocrinol (Lausanne). 2020 Jun 17;11:388.

[2]. M Profita, et al. In vitro effects of flunisolide on MMP-9, TIMP-1, fibronectin, TGF-beta1 release and apoptosis in sputum cells freshly isolated from mild to moderate asthmatics. Allergy. 2004 Sep;59(9):927-32.

[3]. S Boero, et al. Modulation by flunisolide of tumor necrosis factor-alpha-induced stimulation of airway epithelial cell activities related to eosinophil inflammation. J Asthma. 2010 May;47(4):381-7.

[4]. B I Florea, et al. Evidence of P-glycoprotein mediated apical to basolateral transport of flunisolide in human broncho-tracheal epithelial cells (Calu-3). Br J Pharmacol. 2001 Dec;134(7):1555-63.

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

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