Desisobutyryl-ciclesonide

Cat. No.: HY-111490

CAS No.: 161115-59-9

Molecular Formula: $C_{28}H_{38}O_6$

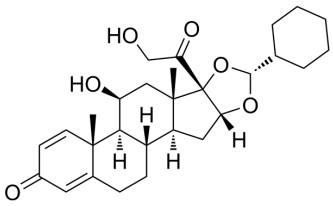
Molecular Weight: 470.6

Target: Glucocorticoid Receptor

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

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 : 100 mg/mL (212.49 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1249 mL	10.6247 mL	21.2495 mL
	5 mM	0.4250 mL	2.1249 mL	4.2499 mL
	10 mM	0.2125 mL	1.0625 mL	2.1249 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: ≥ 2.5 mg/mL (5.31 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.31 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Desisobutyryl-ciclesonide is the active metabolite of Ciclesonide. Desisobutyryl-ciclesonide has affinity for the glucocorticoid receptor.

IC_{50} & Target

Glucocorticoid receptor[1]

In Vitro

Ciclesonide, an inhaled corticosteroid with almost no affinity for the glucocorticoid receptor, is highly effective in downregulating in vitro pro-inflammatory activities of airway parenchymal cells when converted into the active metabolite Desisobutyryl-ciclesonide. Peripheral blood mononuclear cell proliferation to C. albicans is dose-dependently inhibited by 0.3-3.0 μM Ciclesonide and Desisobutyryl-ciclesonide but inhibition by Desisobutyryl-ciclesonide is higher. A significant proliferation to PhlP5 is observed only in cultures from atopic subjects: an effective downregulation is already detected at

0.03 μM Ciclesonide and 0.003 μM Desisobutyryl-ciclesonide (complete inhibition at 3 μM Ciclesonide and 0.03 μM Desisobutyryl-ciclesonide). 3 μM Ciclesonide and Desisobutyryl-ciclesonide reduce the PhlP5-specific T-cell blast proliferation and interleukin 4-producing cell proportion. In PBMCs cultures from atopic patients, both Ciclesonide (CIC) and Desisobutyryl-ciclesonide (des-CIC) induce a dose-dependent downregulation of PhlP5-induced proliferation. The effect is already significantat 0.03 μM Ciclesonide and at 0.003 μM Desisobutyryl-ciclesonide ($p<0.001$, each comparison),with an early complete inhibition observed at 3μM Ciclesonide and at 0.03 μM Desisobutyryl-ciclesonide. The inhibitory activity toward PhlP5-induced PBMC proliferation is higher for Desisobutyryl-ciclesonide than for Ciclesonide at 0.003 μM ($p<0.05$), 0.03 μM ($p<0.001$) and 0.3 μM ($p<0.05$)[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]

Peripheral blood mononuclear cells are isolated from non atopic and atopic asthmatic children sensitized to Phleum pratense (PhlP5). Proliferation toward Candida albicans or PhlP5 in the presence of Ciclesonide or Desisobutyryl-ciclesonide (0.003-3.0 μM) is evaluated as [^{3}H]thymidine incorporation. Modulation of PhlP5-specific T-cell blasts proliferation and PhlP5-induced interleukin 4 expression by Ciclesonide and Desisobutyryl-ciclesonide are measured[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Int J Pharm. 2021 Feb 15;595:120241.

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REFERENCES

[1]. Silvestri M, et al. Ciclesonide modulates in vitro allergen-driven activation of blood mononuclear cells and allergen-specific T-cell blasts. Immunol Lett. 2012 Jan 30;141(2):190-6.

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