Budesonide

Cat. No.: HY-13580

CAS No.: 51333-22-3

Molecular Formula: $C_{25}H_{34}O_6$

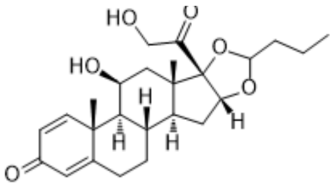
Molecular Weight: 430.53

Target: Glucocorticoid Receptor; ADC Cytotoxin

Pathway: Immunology/Inflammation; Vitamin D Related/Nuclear Receptor; Antibody-drug Conjugate/ADC Related

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 : 215 mg/mL (499.38 mM; Need ultrasonic and warming)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.3227 mL	11.6136 mL	23.2272 mL
5 mM	0.4645 mL	2.3227 mL	4.6454 mL
10 mM	0.2323 mL	1.1614 mL	2.3227 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 (4.83 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.83 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.83 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Budesonide, an inhaled glucocortical steroid, is an orally active glucocorticoid receptor agonist. Budesonide decreases the size of lung tumors, reverses DNA hypomethylation and modulates mRNA expression of genes. Budesonide is an anti-inflammatory agent used for asthma[1][2][3].

In Vitro

Budesonide is selective for human glucocorticoid receptor (hGR; EC_{50}=45.7 pM) over mineralocorticoid receptors (EC_{50} =7,620 pM) in CV-1 cells[1].

	Budesonide (30 min prior to LPS) suppresses the activation of the NLRP3 inflammasome by LPS (100 ng/mL) plus ATP (5 mM) in macrophages (RAW 264.7 cells)[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Budesonide (2.0 mg/kg; orally via their diet; at 2, 7 and 21 days prior to killing) decreases the size of lung tumors[3]. Budesonide (0.5 mg/kg; intranasal administration 1 h before LPS injection (5 mg/kg)) pretreatment dramatically attenuates pathological injury and reduces pathological scores in mice with ALI in adult male C57BL/6 mice[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female strain A/J mice at 8 weeks of age[3]
Dosage:	2.0 mg/kg
Administration:	Orally via their diet; at 2, 7 and 21 days prior to killing (27 weeks)
Result:	Reduced the size of the lung tumors after 2 days and rapidly decreased the size of lung tumors, reversed DNA hypomethylation and modulated mRNA expression of genes.

CUSTOMER VALIDATION

- Mil Med Res. 2022 Jan 29;9(1):7.
- J Control Release. 2019 Dec 28;316:66-78.
- Drug Test Anal. 2020 Aug 27.

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REFERENCES

[1]. Claudia Grossmann, et al. Transactivation via the Human Glucocorticoid and Mineralocorticoid Receptor by Therapeutically Used Steroids in CV-1 Cells: A Comparison of Their Glucocorticoid and Mineralocorticoid Properties. Eur J Endocrinol. 2004 Sep;151(3):397-406.

[2]. Liang Dong, et al. Intranasal Application of Budesonide Attenuates Lipopolysaccharide-Induced Acute Lung Injury by Suppressing Nucleotide-Binding Oligomerization Domain-Like Receptor Family, Pyrin Domain-Containing 3 Inflammasome Activation in Mice. J Immunol Res. 2019 Feb 27;2019:7264383.

[3]. Michael A Pereira, et al. Modulation by Budesonide of DNA Methylation and mRNA Expression in Mouse Lung Tumors. Int J Cancer. 2007 Mar 1;120(5):1150-3.

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