Beclometasone

Cat. No.: HY-B1540

CAS No.: 4419-39-0

Molecular Formula: $C_{22}H_{29}ClO_5$

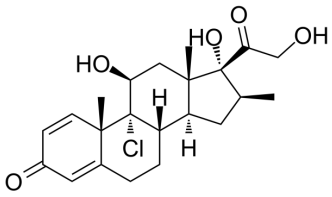
Molecular Weight: 408.92

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	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 32 mg/mL (78.25 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4455 mL	12.2273 mL	24.4547 mL
5 mM	0.4891 mL	2.4455 mL	4.8909 mL
10 mM	0.2445 mL	1.2227 mL	2.4455 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: ≥ 2.25 mg/mL (5.50 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.25 mg/mL (5.50 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.25 mg/mL (5.50 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Beclometasone (Beclomethasone) is a prototype glucocorticoid receptor agonist.

IC_{50} & Target

Glucocorticoid Receptor[1]

In Vitro

An inhibition of the normal physiological neutrophil migration and of neutrophil chemotaxis directed by wounding-induced inflammation is detected at 4 h after administration of 25 μM Beclomethasone. Tumour cell invasion and micrometastasis is

also reduced in embryos incubated in 25 μM Beclomethasone 4 h before implantation. In addition, the lysyl oxidase inhibitor β-aminoproprionitrile (βAPN) largely reduces fibrillar collagen and enhances the CHT-TF transmigration of neutrophils, leading to a significant increase of tumour cell invasion and subsequent formation of micrometastases. Notably, βAPN inhibits neutrophil chemotaxis induced by inflammation, indicating that the increase of tumour cell invasion in βAPN-treated embryos is correlated with enhanced non-pathological neutrophil migration, but not with inflammation[1].

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

PROTOCOL

Cell Assay [1]

The transgenic lines Tg(fli1:GFP) and Tg(mpx:GFP) are used in this study. 0.2 mM N-phenylthiourea (PTU) is applied to prevent pigment formation from 1 day post-fertilization (dpf). For Pu.1 knockdown, Pu.1 MO (1 mM for partial knockdown and 2 mM for complete knockdown is injected into the yolk at the one-cell stage. For pharmacological inhibition, the VEGFR tyrosine kinase inhibitors KRN633 (0.1-1 μM) or Sunitinib (0.1-1 μM), Beclomethasone (25 μM) and β-amino-proprionitrile (β APN, 500 μM) are applied directly to the egg water and refreshed every 2 days. For pharmacological inhibition, Beclomethasone is applied to the embryos 4 h before implantation and KRN633, Sunitinib and βAPN are applied 4-6 h post-implantation. For each cell line or condition, data are representative of ≥ three independent experiments, with ≥30 embryos/group. Experiments are discarded when the survival rate of the control group is <90%[1].

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

CUSTOMER VALIDATION

- Nature. 2021 Jan;589(7843):620-626.
- Pharm Res. 2017 Dec;34(12):2454-2465.
- Drug Test Anal. 2020 Aug 27.

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REFERENCES

[1]. He S, et al. Neutrophil-mediated experimental metastasis is enhanced by VEGFR inhibition in a zebrafish xenograft model. J Pathol. 2012 Aug;227(4):431-45.

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