Norisoboldine

Cat. No.: HY-N0586

CAS No.: 23599-69-1

Molecular Formula: $C_{18}H_{19}NO_4$

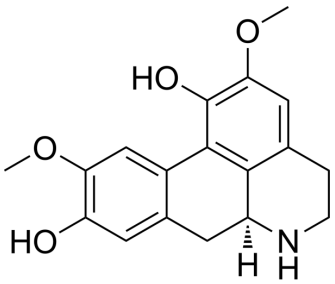
Molecular Weight: 313.35

Target: Adenosine Receptor

Pathway: GPCR/G Protein

Storage: -20°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 62.5 mg/mL (199.46 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.1913 mL	15.9566 mL	31.9132 mL
5 mM	0.6383 mL	3.1913 mL	6.3826 mL
10 mM	0.3191 mL	1.5957 mL	3.1913 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 (6.64 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (6.64 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Norisoboldine is an orally active natural aryl hydrocarbon receptor (AhR) agonist. Norisoboldine, as a major isoquinoline alkaloid present in Radix Linderae, can be used for the research of Rheumatoid arthritis and Ulcerative colitis[1][2].

IC_{50} & Target

AhR[2]

In Vitro

Norisoboldine (1~30 μM; 0~24 hours; CD4+T cells) activates AhR under hypoxic microenvironment and significantly downregulates mRNA expression of miR-31[2].

Norisoboldine (30 μM; 0~24 hours; CD4+T cells) inhibits glycolysis in hypoxia[2].

Norisoboldine (1~30 μM; 0~72 hours; Treg cells) promotes Treg differentiation in hypoxia[2].

Norisoboldine (10, 30 μM) facilitates the disassociation of HSP90/AhR complexes, the nuclear translocation of AhR, and the

formation of AhR/ARNT complexes. Norisoboldine induces generation of Treg cells in hypoxia is independent of miR-31[2].

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

Western Blot Analysis[2]

Cell Line:	CD4+T cells
Concentration:	1~30 μM
Incubation Time:	24 hours
Result:	Activated AhR in cells under hypoxic microenvironment.

RT-PCR[2]

Cell Line:	CD4+T cells
Concentration:	1~30 μM
Incubation Time:	24 hours
Result:	Significantly downregulated mRNA expression of miR-31.

Immunofluorescence[2]

Cell Line:	CD4+T cells
Concentration:	30 μM
Incubation Time:	24 hours
Result:	Inhibited glycolysis in hypoxia.

Cell Differentiation Assay[2]

Cell Line:	Treg cells
Concentration:	1~30 μM
Incubation Time:	72 hours
Result:	Promoted Treg differentiation in hypoxia.

In Vivo

Norisoboldine (10~40 mg/kg; p.o.; 20 days) significantly reduces the severity of joint swelling and erythema during the course of the experiment[1].

Norisoboldine (40 mg/kg; i.g.; 10 days) induces enhancement of CYP1A1 expression and suppresses expressions of Glut1 and HK2 in colons[2].

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

Animal Model:	Male ICR mice (18–22 g)
Dosage:	10~40 mg/kg
Administration:	P.o.
Result:	Significantly reduced the severity of joint swelling and erythema during the course of the experiment.

Animal Model:	Female C57BL/6 mice (18–22 g)
Dosage:	40 mg/kg
Administration:	I.g.
Result:	Induced enhancement of CYP1A1 expression and suppressed expressions of Glut1 and HK2 in colons.

REFERENCES

[1]. Luo Y, et al. Therapeutic effect of norisoboldine, an alkaloid isolated from Radix Linderae, on collagen-induced arthritis in mice. Phytomedicine. 2010;17(10):726-731.

[2]. Lv Q, et al. Norisoboldine, a natural AhR agonist, promotes Treg differentiation and attenuates colitis via targeting glycolysis and subsequent NAD+/SIRT1/SUV39H1/H3K9me3 signaling pathway. Cell Death Dis. 2018;9(3):258. Published 2018 Feb 15.

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