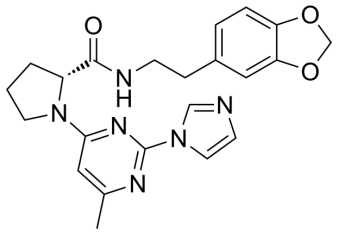
BBS-4

Cat. No.:	HY-12124
CAS No.:	402934-09-2
Molecular Formula:	$C_{22}H_{24}N_6O_3$
Molecular Weight:	420.46
Target:	NO Synthase
Pathway:	Immunology/Inflammation
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 : 100 mg/mL (237.83 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3783 mL	11.8917 mL	23.7835 mL
	5 mM	0.4757 mL	2.3783 mL	4.7567 mL
	10 mM	0.2378 mL	1.1892 mL	2.3783 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 (5.95 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (5.95 mM); Clear solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.5 mg/mL (5.95 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

BBS-4 is a potent and selective inducible nitric oxide synthase (NOS2) dimerization inhibitor, with an IC_{50} of 0.49 nM. BBS-4 can protect mice from the cardiovascular dysfunction of sepsis[1].

IC_{50} & Target

iNOS

In Vitro

BBS-4 exhibits ~300–2000-fold selective for inhibiting iNOS dimerization in cells versus CYP-3A4 (~150 nM in a microsomal benzyloxyresorufin assay; ~1 μM in a cell-based testosterone hydroxylase assay)[2].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	BBS-4 (10 mg/kg; i.p.; 1 h after endotoxin administration) prevents endotoxin-induced hypotension in mice[1]. BBS-4 (30 mg/kg; i.p.; 1 h after endotoxin administration) prevents endotoxin-induced myocardial dysfunction in mice[1]. BBS-4 (10 mg/kg; i.p.; 1 and 8 h after endotoxin administration) prevents endotoxin-induced impairment of murine hypoxic pulmonary vasoconstriction (HPV)[1]. BBS-4 (10 mg/kg; i.p.; 1 and 8 h after endotoxin administration) does not affect the endotoxin-induced increase in pulmonary NOS2 gene expression, but it (30 mg/kg) prevents cardiac and pulmonary NOS2 protein dimerization and increases plasma nitrate and nitrite (NOx) concentration in mice[1]. BBS-2 (30 mg/kg; s.c. twice daily for 10 d) does not affect agonist-stimulated NOS3-dependent aortic relaxation ex vivo[1]. BBS-4 (10-30 mg/kg; i.p.) does not improve mortality rate in endotoxemic mice[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Ichinose F, et, al. A selective inducible NOS dimerization inhibitor prevents systemic, cardiac, and pulmonary hemodynamic dysfunction in endotoxemic mice. Am J Physiol Heart Circ Physiol. 2003 Dec; 285(6): H2524-30.

[2]. https://pubmed.ncbi.nlm.nih.gov/12907425/

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