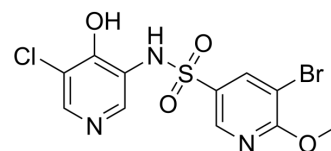


ABR-238901

Cat. No.:	HY-141537		
CAS No.:	1638200-22-2		
Molecular Formula:	C ₁₁ H ₉ BrClN ₃ O ₄ S		
Molecular Weight:	394.63		
Target:	Toll-like Receptor (TLR)		
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 : 33.33 mg/mL (84.46 mM; ultrasonic and warming and heat to 60°C)
 H₂O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	2.5340 mL	12.6701 mL	25.3402 mL
	5 mM	0.5068 mL	2.5340 mL	5.0680 mL
	10 mM	0.2534 mL	1.2670 mL	2.5340 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 (6.34 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

ABR-238901 is an orally active and potent S100A8/A9 blocker and inhibits S100A8/A9 interaction with its receptors RAGE (receptor for advanced glycation endproducts) and TLR4 (toll-like receptor 4). ABR-238901 has the potential for myocardial infarction (MI) research^{[1][2][3]}.

In Vivo

ABR-238901 (30 mg/kg/day; gavage; for 3 weeks) causes less angiogenesis and less IL6 and IL10 in MDSCs^[1].
 ABR-238901 (30 mg/kg/day; gavage) in combination with Bortezomib (0.6 mg/kg; sc; 2 times/week) reduces tumor load compared with treatments of either agent alone^[1].
 ABR-238901 (30 mg/kg; IP for the first 3 d and thereafter continuously p.o.; daily; for 21 days) leads to gradual deterioration of cardiac function and accelerated left ventricular remodeling in C57BL/6NRJ mice with myocardial ischemia induced by permanent coronary artery ligation. Treatment with ABR-238901 during the first 3 days post-myocardial infarction (MI) restricts the inflammatory damage and promotes a reparatory environment^[2].

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

Animal Model:	C57BL/KaLwRij mice with 5T33MMvv cells ^[1]
Dosage:	30 mg/kg
Administration:	Gavage; daily; for 3 weeks
Result:	Caused less angiogenesis. Caused less IL6 and IL10 in myeloid-derived suppressor cells (MDSCs).

CUSTOMER VALIDATION

- Cancer Lett. 2022 Feb 14;532:215598.
- Int Immunopharmacol. 2023 Apr 5;118:110110.
- Exp Ther Med. February 17, 2022.

See more customer validations on www.MedChemExpress.com

REFERENCES

- [1]. Kim De Veirman, et al. Extracellular S100A9 Protein in Bone Marrow Supports Multiple Myeloma Survival by Stimulating Angiogenesis and Cytokine Secretion. Cancer Immunol Res. 2017 Oct;5(10):839-846.
- [2]. Goran Marinković, et al. S100A9 Links Inflammation and Repair in Myocardial Infarction. Circ Res. 2020 Aug 14;127(5):664-676.
- [3]. A. Schiopu, et al. Short-term blockade of the S100A8/A9 alarmin in the immediate post-myocardial infarction period inhibits acute myocardial inflammation and preserves myocardial repair. European Heart Journal, Volume 38, Issue suppl_1, August 2017, ehx504.

Caution: Product has not been fully validated for medical applications. For research use only.

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