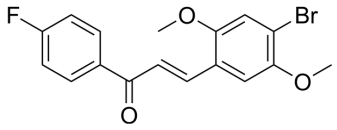
NLRP3-IN-10

Cat. No.:	HY-151343
CAS No.:	2641826-39-1
Molecular Formula:	$C_{17}H_{14}BrFO_3$
Molecular Weight:	365.19
Target:	NOD-like Receptor (NLR)
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 (273.83 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.7383 mL	13.6915 mL	27.3830 mL
	5 mM	0.5477 mL	2.7383 mL	5.4766 mL
	10 mM	0.2738 mL	1.3692 mL	2.7383 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

NLRP3-IN-10 is a potent NLRP3 inhibitor, inhibits IL-1β release with an IC_{50} value of 251.1 nM. NLRP3-IN-10 suppresses NLRP3 inflammasome activation by attenuating ASC speck formation[1].

IC_{50} & Target

NLRP3
251.1 nM (IC_{50})

In Vitro

NLRP3-IN-10 (compound 14c) (0.4, 1.6, 6.4 μM; 40 min) exerts remarkable inhibitory activity on NLRP3 inflammasome activation induced by LPS-MSU (12 h) in THP-1 cells in a dose-dependent manner[1].
NLRP3-IN-10 (0.1-6.4 μM; 1.5 h) shows no cytotoxicity against THP-1 cells and (0.1, and 0.4 μM; 40 min) avoids Nigericin (HY-127019)-induced pyroptosis[1].
NLRP3-IN-10 (0.1, 0.2, and 0.4 μM; 40 min) reduces the processing of caspase-1 p20 and IL-1β, in supernatants in THP-1 cells in a dose-dependent manner[1].
NLRP3-IN-10 (3 μM and 5 μM; 40 min) decreases LPS-induced THF-α, and (0.2 μM and 0.8 μM; 40 min) reduces the rate of THP-1 cells with ASC specks, indicating ASC oligomerization interruptionsup>[1].
NLRP3 inflammasome is regarded as a two-step process, including priming and action. NLRP3-IN-10 (1, 10, and 100 μM; 40

min) suppresses LPS-induced NLRP3 priming through directly interacting with NLRP3[1].

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

In Vivo

NLRP3-IN-10 (compound 14c) (10 mg/kg; i.v.; single dose) reduces peritoneal neutrophil influx in mice and IL-1β in the spleen in the MSU-induced peritonitis in LPS-primed mouse model[1].

NLRP3-IN-10 (10, 30, 90 mg/kg; p.o.; single dose) exhibits extremely low exposure (14.6−23.53 μg·h/L), poor bioavailability (2.47−13.79%), and high plasma clearance (2201.58−5551.12 L/h/kg) after different doses for oral administration[1].

Pharmacokinetics of NLRP3-IN-10 in mouse[1]

Route	Dose (mg/kg)	AUC_{0-t} (μg·h/L)	CL (L/h/kg)	C_{max} (μg/L)	$T_{1/2}$ (h)	T_{max} (h)	F (%)
IV	10	105.88	133.75	81.97	3.13	0.11	
PO	10	14.60	2201.58	3.35	7.43	2.11	13.79
PO	30	15.84	2583.27	16.42	7.92	1.26	4.99
PO	90	23.53	5551.12	13.59	6.08	4.21	2.47

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

Animal Model:	MSU-induced peritonitis in a LPS-primed mouse model (C57BL/6J mice, 7-week-old, male) [1] LPS: 1 mg/kg, i.p.; MSU: 100 mg/kg, i.v.
Dosage:	10 mg/kg
Administration:	Intravenous injection; single dose
Result:	Significantly reduced IL-1β release in the spleen of mice after 6 h treatment. Significantly reduced the increase of peritoneal neutrophil influx compared with the control group.

REFERENCES

[1]. Zhang R, et al. New Highly Potent NLRP3 Inhibitors: Furanochalcone Velutone F Analogues. ACS Med Chem Lett. 2022 Mar 7;13(4):560-569.

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