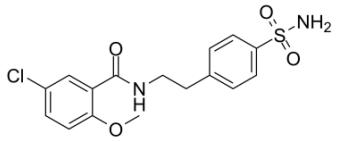
NLRP3-IN-2

Cat. No.:	HY-W011082
CAS No.:	16673-34-0
Molecular Formula:	$C_{16}H_{17}ClN_2O_4S$
Molecular Weight:	368.84
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 : 125 mg/mL (338.90 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.7112 mL	13.5560 mL	27.1120 mL
5 mM	0.5422 mL	2.7112 mL	5.4224 mL
10 mM	0.2711 mL	1.3556 mL	2.7112 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 (5.64 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.64 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

NLRP3-IN-2, an intermediate substrate in the synthesis of glyburide, inhibits the formation of the NLRP3 inflammasome in cardiomyocytes and limits the infarct size following myocardial ischemia/reperfusion in the mouse, without affecting glucose metabolism[1].

In Vivo

NLRP3-IN-2 is well tolerated with no effects on the glucose levels in vivo[1].
NLRP3-IN-2 (100 mg/kg) treatment in a model of AMI due to ischemia+reperfusion significantly inhibits the activity of inflammasome (caspase-1) in the heart by 90% (P<0.01) and reduced infarct size, measured at pathology (by >40%, P<0.01) and with troponin I levels (by >70%, P<0.01) [1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Experimental acute myocardial infarction (AMI) model in mice[1].
Dosage:	100 mg/kg.
Administration:	Intraperitoneal administration 30 minutes prior to surgery, then every 6 hours for 3 additional doses.
Result:	Led to a significant >90% reduction in caspase-1 activity (reflective of the formation of an active inflammasome) in the heart tissue measured 24 hours after ischemia. Led to a significant reduction in the infarct size measured with TTC (>40% reduction) or troponin I levels (>70% reduction) when compared with vehicle alone.

REFERENCES

[1]. Carlo Marchetti, et al. A novel pharmacologic inhibitor of the NLRP3 inflammasome limits myocardial injury after ischemia-reperfusion in the mouse. J Cardiovasc Pharmacol. 2014 Apr;63(4):316-322.

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