CORM-3

Cat. No.: HY-100581

CAS No.: 475473-26-8

Molecular Formula: $C_5H_4ClNO_5Ru$

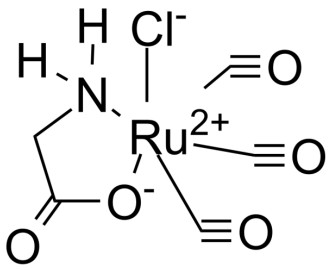
Molecular Weight: 294.61

Target: NF-κB; NOD-like Receptor (NLR)

Pathway: NF-κB; Immunology/Inflammation

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 75 mg/mL (254.57 mM; Need ultrasonic)

H_2O : 50 mg/mL (169.72 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.3943 mL	16.9716 mL	33.9432 mL
5 mM	0.6789 mL	3.3943 mL	6.7886 mL
10 mM	0.3394 mL	1.6972 mL	3.3943 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: 3.75 mg/mL (12.73 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 3.75 mg/mL (12.73 mM); Clear solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 3.75 mg/mL (12.73 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

CORM-3, a carbon monoxide-releasing molecule, attenuates NF-κB p65 nuclear translocation, reduces ROS generation and enhances intracellular glutathione and superoxide dismutase levels. CORM-3 reduces NLRP3 inflammasome activation[1][2][3].

IC_{50} & Target

NLRP3

In Vitro

CORM-3 suppresses caspase-1 activation and the secretion of interleukin (IL)-1β and IL-18 in macrophages in response to

lipopolysaccharide (LPS) and ATP. CORM-3 inhibits the oligomerization of the adaptor protein apoptosis-associated speck-like protein containing a caspase recruitment domain (ASC), which is required for NLRP3-dependent caspase-1 activation[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

CORM-3 (4 mg/kg, ip) reduces NLRP3 inflammasome activation and inhibits hyperglycemia-induced inflammation in mice[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	WT mice (C57BL/6J male, 8e10 weeks old)[2].
Dosage:	4 mg/kg.
Administration:	IP for 3 h before i.p. injection of E. coli LPS (10 mg/kg), and IP injection after 7-day-treatment of S0130.
Result:	Resulted in significantly lower plasma levels of IL-1β and IL-18 in response to LPS challenge in vivo in WT mice relative to that in vehicle-treated control mice, whereas TNF-a levels were unchanged. Had lower expression of cleaved caspase-1 and cleaved IL-1b in response to ATP and nigericin, relative to vehicle control, whereas the expression of procaspase-1 and pro-IL-1 β expression was unchanged.

REFERENCES

[1]. Foresti R, et al. Vasoactive properties of CORM-3, a novel water-soluble carbon monoxide-releasing molecule. Br J Pharmacol. 2004 Jun;142(3):453-60.

[2]. Lee DW, et al. Carbon monoxide regulates glycolysis-dependent NLRP3 inflammasome activation in macrophages. Biochem Biophys Res Commun. 2017 Nov 18;493(2):957-963.

[3]. Huang Y, et al. Carbon monoxide (CO) inhibits hydrogen peroxide (H2O2)-induced oxidative stress and the activation of NF-κB signaling in lens epithelial cells. Exp Eye Res. 2018 Jan;166:29-39.

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