NecroX-7

Cat. No.: HY-124750

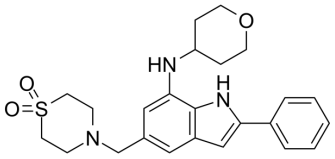
CAS No.: 1120332-55-9

Molecular Formula: $C_{24}H_{29}N_3O_3S$

Molecular Weight: 439.57

Target: TNF Receptor; Interleukin Related; Toll-like Receptor (TLR); Reactive Oxygen Species

Pathway: Apoptosis; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB



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 (227.50 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2750 mL	11.3748 mL	22.7495 mL
	5 mM	0.4550 mL	2.2750 mL	4.5499 mL
	10 mM	0.2275 mL	1.1375 mL	2.2750 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 (5.69 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.69 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.69 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

NecroX-7 is a potent free radical scavenger and a HMGB1 (high-mobility group box 1) inhibitor. NecroX-7 can be used as an antidote to acetaminophen toxicity. NecroX-7 exerts a protective effect by preventing the release of HMGB1 in ischemia/reperfusion injury. NecroX-7 inhibits the HMGB1-induced release of TNF and IL-6, as well as the expression of TLR-4 and receptor for advanced glycation end products. NecroX-7 can be used graft-versus-host disease (GVHD) research[1].

IC_{50} & Target		
	IL-6	TLR4

In Vitro

NecroX-7 (0-40 μM, 3-4 d) suppresses activated or proliferating T cells without causing apoptosis[1].
NecroX-7 (0-40 μM) markedly reduces HMGB1 levels in a dose-dependent manner[1].
NecroX-7 inhibits formation of mitochondria-specific ROS/reactive nitrogen species in H9C2 cells and hepatocytes after induction by tert-butyl hydroperoxide or doxorubicin[1].
NecroX-7 increased regulatory T cell numbers, which may be associated with regulation of differentiation signals independent of HMGB1[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	CD4 T cells
Concentration:	0, 0.625, 1.25, 2.5, 5, 10, 20, and 40 μM
Incubation Time:	3-4 d
Result:	Showed a marked reduction in splenocyte proliferation, in a dose-dependent manner. Modulated alloreactive T cell responses.

In Vivo

NecroX-7 (0-0.3 mg/kg, IV, once injection at 2-d intervals, for 2 weeks) significantly attenuates GVHD-related mortality and inhibits severe tissue damage[1].
NecroX-7 protects mice against lethal GVHD by reciprocal regulation of regulatory T/Th1 cells, attenuating systemic HMGB1 accumulation and inhibiting HMGB1-mediated inflammatory response[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female BALB/c and C57BL/6 mice (Eight-week-old, with GVHD)[1]
Dosage:	0.03, 0.1, and 0.3 mg/kg
Administration:	IV, once injection at 2-d intervals, for 2 weeks
Result:	Observed statistically significant prolonged survival at doses ≥0.1 mg/kg: 30–60% of mice in these treatment groups survived for >50 d. Significantly improved clinical signs and prolonged survival, and the mice showed a reduction in clinical manifestations of acute GVHD, including weight loss, hunched posture, diarrhea, and ruffled fur.

REFERENCES

[1]. Im KI, et al. The Free Radical Scavenger NecroX-7 Attenuates Acute Graft-versus-Host Disease via Reciprocal Regulation of Th1/Regulatory T Cells and Inhibition of HMGB1 Release. J Immunol. 2015 Jun 1;194(11):5223-32.

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