

R-7050

Cat. No.:	HY-110203		
CAS No.:	303997-35-5		
Molecular Formula:	C ₁₆ H ₈ ClF ₃ N ₄ S		
Molecular Weight:	380.77		
Target:	TNF Receptor		
Pathway:	Apoptosis		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro	DMSO : 25 mg/mL (65.66 mM; Need ultrasonic)					
		Solvent	Mass	1 mg	5 mg	10 mg
	Preparing Stock Solutions	Concentration				
		1 mM		2.6263 mL	13.1313 mL	26.2626 mL
5 mM			0.5253 mL	2.6263 mL	5.2525 mL	
	10 mM		0.2626 mL	1.3131 mL	2.6263 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 (6.57 mM); Suspended solution; Need ultrasonic					
	2. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (6.57 mM); Clear solution					

BIOLOGICAL ACTIVITY

Description	R-7050 (TNF-α Antagonist III) is a tumor necrosis factor receptor (TNFR) antagonist with greater selectivity toward TNFα.
IC₅₀ & Target	CD40
In Vitro	R-7050 (TNF-α Antagonist III) is a potent and fully reversible hit with greater selectivity toward TNFα. In TNFα-induced intercellular adhesion molecule 1 (ICAM-1) expression, R-7050 inhibition potency (EC ₅₀ =0.63 μM) is 2- to 3-fold greater than EC ₅₀ for IL-1β-induced ICAM-1 expression (EC ₅₀ =1.45 2 μM). R-7050 inhibits phosphorylation of both the JNK pathway (MKK4, JNKs, and ATF2) and p38 pathway (MKK3/6, p38, and MAPKAP2) ^[1] . R-7050 is a cell-permeable triazoloquinoxaline compound that selectively inhibits TNF-α induced cellular signaling. Unlike biologic TNF inhibitors (e.g. Infliximab, Etanercept, Adalimumab) that directly bind TNF-α and function as decoy receptors, R-7050 does not affect binding of TNF-α

to TNFR. In contrast, R-7050 selectively inhibits the association of TNFR with intracellular adaptor molecules (e.g. TRADD, RIP), limits receptor internalization, and prevents subsequent cellular responses after TNF- α binding^[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

R-7050 (TNF- α Antagonist III) (6 mg/kg) reduces Evans blue extravasation to 28.7 \pm 5.9 μ g and 30.3 \pm 1.9 μ g Evans blue/g brain tissue when administered at 0.5 h or 2 h post-ICH, respectively (p<0.05 and p<0.01 vs ICH, respectively; not significantly different from sham). Brain water content, a measure of brain edema, increases from 75.6 \pm 0.3% in sham-operated mice to 81.5 \pm 0.5% at 24h post-ICH (p<0.05 vs. sham). 6, 12, or 18 mg/kg R-7050 reduces brain water content to 78.5 \pm 0.3%, 78.3 \pm 0.3%, or 79.3 \pm 0.5%, respectively (all treatments p<0.05 vs. ICH; treatments not significantly different from each other). Notably, mice treated with 18 mg/kg exhibit a reduction in general activity/locomotion. As is observed with Evans blue extravasation, R-7050 (6 mg/kg) significantly reduces brain water content after ICH. Administration of R-7050 at 0.5h or 2h post-ICH attenuates brain water content to levels observed in sham-operated mice (p<0.05 vs ICH, not significantly different from sham)^[2].
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PROTOCOL

Animal Administration ^[2]

Mice^[2]

A mouse collagenase model of ICH is utilized. Briefly, male CD-1 mice (8-10 weeks old) are placed into a stereotactic frame and a 0.5 mm diameter burr hole is drilled over the parietal cortex, 2.2 mm lateral to the bregma. A 26-gauge Hamilton syringe, loaded with 0.04 U of bacterial type IV collagenase in 0.5 μ L saline, is lowered 3 mm deep from the skull surface directly into the left striatum. The syringe is depressed at a rate of 450 nL/min and left in place for several minutes after the procedure to prevent solution reflux and excess diffusion. Sham animals undergo the same surgical procedure, except that saline is stereotactically injected rather than collagenase. After the syringe is removed, bone wax is used to close the burr hole, the incision is surgically stapled, and mice are kept warm until recovery of the righting reflex. R-7050 (6-18 mg/kg) is administered via intraperitoneal route at the time of injury or up to 2 h post-ICH^[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Death Differ. 2021 Sep;28(9):2728-2744.
- J Neuroinflammation. 2023 Jun 1;20(1):137.
- J Neuroinflammation. 2019 Nov 26;16(1):234.
- Cell Rep. 2022 Jul 5;40(1):111033.
- Phytomedicine. 2024 Mar 15, 155482.

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REFERENCES

[1]. Gururaja TL, et al. A class of small molecules that inhibit TNF α -induced survival and death pathways via prevention of interactions between TNF α RI, TRADD, and RIP1. Chem Biol. 2007 Oct;14(10):1105-18.

[2]. King MD, et al. TNF- α receptor antagonist, R-7050, improves neurological outcomes following intracerebral hemorrhage in mice. Neurosci Lett. 2013 May 10;542:92-6.

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

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