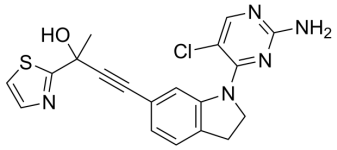
B022

Cat. No.:	HY-120501
CAS No.:	1202764-53-1
Molecular Formula:	$C_{19}H_{16}ClN_5OS$
Molecular Weight:	398
Target:	NF-κB
Pathway:	NF-κB

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (628.14 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.5126 mL	12.5628 mL	25.1256 mL
5 mM	0.5025 mL	2.5126 mL	5.0251 mL
10 mM	0.2513 mL	1.2563 mL	2.5126 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 (5.23 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.23 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

B022 is a potent and selective NF-κB-inducing kinase (NIK) inhibitor (K_i of 4.2 nM; IC_{50}=15.1 nM). B022 protects liver from toxin-induced inflammation, oxidative stress, and injury[1][2]. B022 is a click chemistry reagent, it contains an Alkyne group and can undergo copper-catalyzed azide-alkyne cycloaddition (CuAAc) with molecules containing Azide groups.

IC_{50} & Target

Ki: 4.2 nM (NF-κB-inducing kinase (NIK))[1]

In Vitro

B022 (0-5 μM; 12 hours; Hepa1 cells) treatment suppresses NIK-induced p52 formation in a dose-dependent manner[1].
?B022 (0-5 μM; 12 hours; Hepa1 cells) treatment for 8 h completely blocks NIK-induced expression of TNF-a, IL-6, iNOS, CCL2, and CXCL5[1].
?B022 prevents NIK- or H2O2-induced β cell death and also ameliorates streptozotocin (STZ)-induced β cell death and

hyperglycemia[3].

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

Western Blot Analysis[1]

Cell Line:	Hepa1 cells
Concentration:	0 μM, 0.5 μM, 5 μM
Incubation Time:	12 hours
Result:	Suppressed NIK-induced p52 formation in a dose-dependent manner.

RT-PCR[1]

Cell Line:	Hepa1 cells
Concentration:	0 μM, 0.5 μM, 5 μM
Incubation Time:	12 hours
Result:	Dose-dependently blocked NIK-induced expression of chemokines, cytokines, and iNOS in these cells. Completely blocked NIK-induced expression of TNF-a, IL-6, iNOS, CCL2, and CXCL5.

In Vivo

B022 (30 mg/kg; intravenous injection; twice a day; for 10 days; STOP-NIK male mice) treatment inhibits NIK-triggered liver inflammation and injury in STOP-NIK mice infected with cre adenoviruses[1].

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

Animal Model:	STOP-NIK male mice (8 weeks) infected with Ad-cre[1]
Dosage:	30 mg/kg
Administration:	Intravenous injection; twice a day; for 10 days
Result:	Completely prevents the lethal effect of abnormally high levels of hepatic NIK in mice. Inhibited the majority of the deteriorating effects of aberrant activation of hepatic NIK.

CUSTOMER VALIDATION

- Nat Commun. 2022 Dec 16;13(1):7782.
- Nat Commun. 2022 Nov 12;13(1):6881.
- Int J Pharm. 2022 Nov 1;122361.

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REFERENCES

[1]. Ren X, et al. A small-molecule inhibitor of NF-κB-inducing kinase (NIK) protects liver from toxin-induced inflammation, oxidative stress, and injury. FASEB J. 2017 Feb;31(2):711-718.

[2]. Li Z, et al. Discovery of a Potent and Selective NF-κB-Inducing Kinase (NIK) Inhibitor That Has Anti-inflammatory Effects in Vitro and in Vivo. J Med Chem. 2020;63(8):4388-4407.

[3]. Li X, et al. Activation of NF-κB-Inducing Kinase in Islet β Cells Causes β Cell Failure and Diabetes. Mol Ther. 2020;28(11):2430-2441.

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