Indinavir sulfate

Cat. No.: HY-B0689A

CAS No.: 157810-81-6

Molecular Formula: $C_{36}H_{49}N_5O_8S$

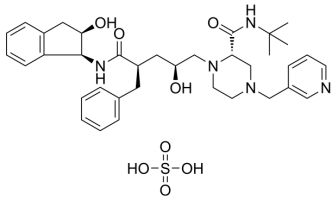
Molecular Weight: 711.87

Target: HIV; HIV Protease; SARS-CoV; Apoptosis; MMP

Pathway: Anti-infection; Metabolic Enzyme/Protease; Apoptosis

Storage: -20°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (140.48 mM)

H_2O : 50 mg/mL (70.24 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.4048 mL	7.0238 mL	14.0475 mL
5 mM	0.2810 mL	1.4048 mL	2.8095 mL
10 mM	0.1405 mL	0.7024 mL	1.4048 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (140.48 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (3.51 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.51 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.51 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Indinavir sulfate (MK-639) is an orally active and selective HIV-1 protease inhibitor with a K_i of 0.54 nM for PR. Indinavir sulfate exhibits anticancer activity by inhibiting the activation of MMPs-2 hydrolysis, anti-angiogenesis and inducing apoptosis. Indinavir sulfate is also a SARS-CoV $3CL^{pro}$ inhibitor[1][2][3][4].

IC_{50} & Target	MMP-2	HIV-1

In Vitro

Indinavir sulfate (0-50 μM; 18 h) blocks lymphocyte cell cycle in G0/G1 phase in PBMCs cells and impairs lymphoproliferative responses[1].
Indinavir sulfate (40 μM-40 nM; 5 days) inhibits cell invasion and (40 μM-40 nM; 48 h) MMPs-2 activation of the Huh7 and SK-HEP-1 hepatocarcinoma cells in vitro[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	PBMCs (from healthy and HIV-infected volunteers)
Concentration:	0-50 μM
Incubation Time:	18 h (pretreatment; stimulation with anti-CD3 for an additional 48 hours)
Result:	Blocked anti-CD3-induced cell-cycle progression in a dose-dependent manner. Resulted in dose-dependent reduction of lymphoproliferative responses.

Cell Invasion Assay[2]

Cell Line:	Huh7 and SK-HEP-1 cells
Concentration:	40 μM-40 nM
Incubation Time:	5 days
Result:	Reduced ability to invade an in vitro constituted extracellular matrix for both cell lines treated compared with the untreated cells.

Western Blot Analysis[2]

Cell Line:	Huh7 and SK-HEP-1 cells
Concentration:	40 μM-40 nM
Incubation Time:	48 h
Result:	Blocked the conversion of latent MMP-2 to its 62/64-kDa active form.

In Vivo

Indinavir sulfate (70 mg/kg; i.g.; once a day for 3 weeks) inhibits the growth of hepatocarcinoma cells in vivo[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Nude mice(s.c. into Huh7 and SK-HEP-1 cells)[2].
Dosage:	70 mg/kg
Administration:	Oral gavage; once a day for 3 weeks.
Result:	Delaied the growth of s.c. implanted hepatocarcinoma xenografts in nude mice compared with placebo.

CUSTOMER VALIDATION

- Signal Transduct Target Ther. 2021 May 29;6(1):212.
- Nat Commun. 2020 Sep 4;11(1):4417.

- Int J Antimicrob Agents. 2019 Dec;54(6):814-819.
- Antiviral Res. 2022 Nov 10;105463.
- Front Pharmacol. 2021 Apr 12;12:634097.

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REFERENCES

[1]. Chavan S, et al. The HIV protease inhibitor Indinavir inhibits cell-cycle progression in vitro in lymphocytes of HIV-infected and uninfected individuals. Blood. 2001 Jul 15;98(2):383-9.

[2]. Esposito V, et al. Evaluation of antitumoral properties of the protease inhibitor indinavir in a murine model of hepatocarcinoma. Clin Cancer Res. 2006 Apr 15;12(8):2634-9.

[3]. Liu F, et al. Kinetic, stability, and structural changes in high-resolution crystal structures of HIV-1 protease with drug-resistant mutations L24I, I50V, and G73S. J Mol Biol. 2005 Dec 9;354(4):789-800.

[4]. Hall DC Jr, et al. A search for medications to treat COVID-19 via in silico molecular docking models of the SARS-CoV-2 spike glycoprotein and 3CL protease. Travel Med Infect Dis. 2020 May-Jun;35:101646.

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

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