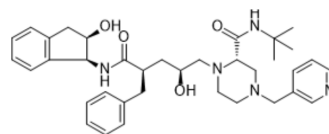


## Indinavir

<b>Cat. No.:</b>	HY-B0689
<b>CAS No.:</b>	150378-17-9
<b>Molecular Formula:</b>	C <sub>36</sub> H <sub>47</sub> N <sub>5</sub> O <sub>4</sub>
<b>Molecular Weight:</b>	613.79
<b>Target:</b>	HIV; HIV Protease; Apoptosis; MMP; SARS-CoV
<b>Pathway:</b>	Anti-infection; Metabolic Enzyme/Protease; Apoptosis
<b>Storage:</b>	Please store the product under the recommended conditions in the Certificate of Analysis.



### BIOLOGICAL ACTIVITY

<b>Description</b>	Indinavir (MK-639 free base) is an orally active and selective HIV-1 protease inhibitor with a K <sub>i</sub> of 0.54 nM for PR. Indinavir exhibits anticancer activity by inhibiting the activation of MMPs-2 hydrolysis, anti-angiogenesis and inducing apoptosis. Indinavir is also a SARS-CoV 3CL <sup>pro</sup> inhibitor <sup>[1][2][3][4]</sup> .																	
<b>IC<sub>50</sub> &amp; Target</b>	HIV-1	MMP-2																
<b>In Vitro</b>	<p>Indinavir (0-50 μM; 18 h) blocks lymphocyte cell cycle in G0/G1 phase in PBMCs cells and impairs lymphoproliferative responses<sup>[1]</sup>.</p> <p>Indinavir (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<sup>[2]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Cell Viability Assay<sup>[1]</sup></p> <table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 30%;">Cell Line:</td> <td>PBMCs (from healthy and HIV-infected volunteers)</td> </tr> <tr> <td>Concentration:</td> <td>0-50 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>18 h (pretreatment; stimulation with anti-CD3 for an additional 48 hours)</td> </tr> <tr> <td>Result:</td> <td>Blocked anti-CD3-induced cell-cycle progression in a dose-dependent manner. Resulted in dose-dependent reduction of lymphoproliferative responses.</td> </tr> </table> <p>Cell Invasion Assay<sup>[2]</sup></p> <table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 30%;">Cell Line:</td> <td>Huh7 and SK-HEP-1 cells</td> </tr> <tr> <td>Concentration:</td> <td>40 μM-40 nM</td> </tr> <tr> <td>Incubation Time:</td> <td>5 days</td> </tr> <tr> <td>Result:</td> <td>Reduced ability to invade an in vitro constituted extracellular matrix for both cell lines treated compared with the untreated cells.</td> </tr> </table> <p>Western Blot Analysis<sup>[2]</sup></p>		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 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.
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	Cell Line:	Huh7 and SK-HEP-1 cells
	Concentration:	40 $\mu$ M-40 nM
	Incubation Time:	48 h
	Result:	Blocked the conversion of latent MMP-2 to its 62/64-kDa active form.
<b>In Vivo</b>	Indinavir (70 mg/kg; i.g.; once a day for 3 weeks) inhibits the growth of hepatocarcinoma cells in vivo <sup>[2]</sup> . 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) <sup>[2]</sup> .
	Dosage:	70 mg/kg
	Administration:	Oral gavage; once a day for 3 weeks.
	Result:	Delayed 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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