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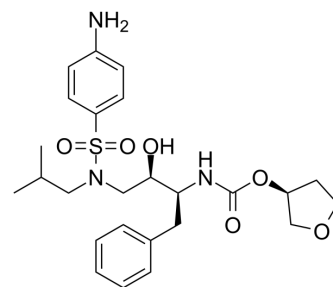
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Amprenavir

Cat. No.:	HY-17430
CAS No.:	161814-49-9
Molecular Formula:	C ₂₅ H ₃₅ N ₃ O ₆ S
Molecular Weight:	505.63
Target:	HIV; HIV Protease; SARS-CoV
Pathway:	Anti-infection; Metabolic Enzyme/Protease
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 : ≥ 100 mg/mL (197.77 mM)
 * "≥" means soluble, but saturation unknown.

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM		1.9777 mL	9.8887 mL	19.7773 mL
	5 mM		0.3955 mL	1.9777 mL	3.9555 mL
	10 mM		0.1978 mL	0.9889 mL	1.9777 mL

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

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
 Solubility: ≥ 2.5 mg/mL (4.94 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
 Solubility: ≥ 2.5 mg/mL (4.94 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
 Solubility: ≥ 2.5 mg/mL (4.94 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Amprenavir (VX-478) is a HIV protease inhibitor (K_i=0.6 nM) used to treat HIV infection. Amprenavir is also a SARS-CoV 3CL^{pro} inhibitor with an IC₅₀ of 1.09 μM.

IC₅₀ & Target

IC₅₀ Value: 0.6 nM (K_i); Against wild-type clinical HIV isolates:14.6 +/- 12.5 ng/mL (mean +/- SD) [1].Target: HIV protease

In Vitro

Amprenavir has an enzyme inhibition constant (K_i = 0.6 nM) that falls within the K_i range of the other protease inhibitors.

Amprenavir's in vitro 50% inhibitory concentration (IC50) against wild-type clinical HIV isolates is 14.6 +/- 12.5 ng/mL (mean +/- SD) [1]. Amprenavir had direct inhibitory effects on invasion of Huh-7 hepatocarcinoma cell lines, inhibiting MMP proteolytic activation [2].

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

In Vivo

Amprenavir was able to promote regression of hepatocarcinoma growth in vivo by anti-angiogenetic and overall anti-tumor activities, independently by PI3K/AKT related pathways that at today is one of the more suggestive hypothesis to explain the anti-tumor effects of the different protease inhibitors [2]. Amprenavir efficiently activated PXR and induced PXR target gene expression in vitro and in vivo. Short-term exposure to amprenavir significantly increased plasma total cholesterol and atherogenic low-density lipoprotein cholesterol levels in wild-type mice, but not in PXR-deficient mice [3]. Amprenavir has been approved for adults and children; the recommended capsule doses are 1200 mg twice daily for adults and 20 mg/kg twice daily or 15 mg/kg 3 times daily for children < 13 years of age or adolescents < 50 kg [1].

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

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.
- J Med Chem. 2021 Mar 11;64(5):2725-2738.
- Antimicrob Agents Chemother. 2020 Aug 20;64(9):e00872-20.

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REFERENCES

- [1]. Sadler BM, Stein DS. Clinical pharmacology and pharmacokinetics of amprenavir. Ann Pharmacother. 2002 Jan;36(1):102-18.
- [2]. Esposito V, Verdina A, Manente L, Amprenavir inhibits the migration in human hepatocarcinoma cell and the growth of xenografts. J Cell Physiol. 2013 Mar;228(3):640-5.
- [3]. Helsley RN, Sui Y, Ai N, Pregnane X Receptor Mediates Dyslipidemia Induced by the HIV Protease Inhibitor Amprenavir in Mice. Mol Pharmacol. 2013 Jun;83(6):1190-9.
- [4]. Qi Sun, et al. Bardoxolone and bardoxolone methyl, two Nrf2 activators in clinical trials, inhibit SARS-CoV-2 replication and its 3C-like protease. Signal Transduct Target Ther. 2021 May 29;6(1):212.

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

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