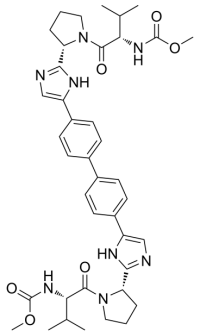
Daclatasvir

Cat. No.:	HY-10466
CAS No.:	1009119-64-5
Molecular Formula:	$C_{40}H_{50}N_8O_6$
Molecular Weight:	738.88
Target:	HCV
Pathway:	Anti-infection
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 : ≥ 40 mg/mL (54.14 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.3534 mL	6.7670 mL	13.5340 mL
5 mM	0.2707 mL	1.3534 mL	2.7068 mL
10 mM	0.1353 mL	0.6767 mL	1.3534 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 (3.38 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.38 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.38 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Daclatasvir (BMS-790052) is a potent and orally active HCV NS5A protein inhibitor with EC_{50}s range of 9-146 pM for multiple HCV replicon genotypes. Daclatasvir is also a organic anion transporting polypeptide 1B (OATP1B) and OATP1B3 inhibitor with IC_{50}s of 1.5 μM and 3.27 μM, respectively[1][2][3].

IC_{50} & Target

EC50: 50 pM (HCV replicon genotype 1a), 9 pM (HCV replicon genotype 1b), 71 pM (HCV replicon genotype 2a), 146 pM (HCV replicon genotype 3a), 12 pM (HCV replicon genotype 4a) and 33 pM (HCV replicon genotype 5a)[1]

	Kd: 8 nM (NS5A33-202) and 210 nM (NS5A26-202)[2] IC50: 1.5 μM (OATP1B) and 3.27 μM (OATP1B3)[3]
In Vitro	Daclatasvir (BMS-790052) demonstrates potent inhibitory activity towards all genotypes tested, with EC_{50} values ranging from 9 pM to 146 pM. Daclatasvir inhibits HCV replicon genotype 1a, 1b, 2a, 3a, 4a and 5a with EC_{50} values of 50 pM, 9 pM, 71 pM, 146 pM, 12 pM and 33 pM, respectively. Daclatasvir is a potent inhibitor of the JFH-1 genotype 2a infectious virus that replicates in cell culture (EC_{50}=28 pM)[1]. Daclatasvir (BMS-790052) binds tightly to NS5A33-202 and NS5A26-202 with K_ds of 8 nM and 210 nM, respectively[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Daclatasvir (BMS-790052; 30 mg/kg; oral administration; daily; for 27 days) treatment reduces serum HCV RNA titers very rapidly by ~1.5 log_{10} at day 3[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	NOD/SCID male mice (5 weeks of age, 18-20 g) bearing HCV RNA-transfected cells[4]
Dosage:	30 mg/kg
Administration:	Oral administration; daily; for 27 days
Result:	Reduced serum HCV RNA titers very rapidly by ~1.5 log_{10} at day 3.

CUSTOMER VALIDATION

- Hepatology. 2019 May;69(5):1861-1872.
- Int J Antimicrob Agents. 2015 Oct;46(4):381-8.
- Cell Rep. 2021 Nov 23;37(8):110049.
- EMBO Rep. 2016 Jul;17(7):1013-28.
- Antiviral Res. 2020 May;177:104734.

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REFERENCES

[1]. Min Gao, et al. Chemical genetics strategy identifies an HCV NS5A inhibitor with a potent clinical effect. Nature. 2010 May 6;465(7294):96-100.

[2]. David B Ascher, et al. Potent hepatitis C inhibitors bind directly to NS5A and reduce its affinity for RNA. Sci Rep. 2014 Apr 23;4:4765.

[3]. Tomomi Furihata, et al. Different interaction profiles of direct-acting anti-hepatitis C virus agents with human organic anion transporting polypeptides. Antimicrob Agents Chemother. 2014 Aug;58(8):4555-64.

[4]. Seung-Hoon Lee, et al. HA1077 displays synergistic activity with daclatasvir against hepatitis C virus and suppresses the emergence of NS5A resistance-associated substitutions in mice. Sci Rep. 2018 Aug 20;8(1):12469.

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

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