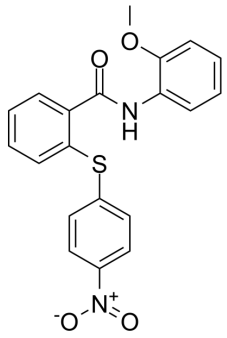
RN-18

Cat. No.:	HY-102014
CAS No.:	431980-38-0
Molecular Formula:	$C_{20}H_{16}N_2O_4S$
Molecular Weight:	380.42
Target:	HIV
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 : 100 mg/mL (262.87 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6287 mL	13.1434 mL	26.2867 mL
	5 mM	0.5257 mL	2.6287 mL	5.2573 mL
	10 mM	0.2629 mL	1.3143 mL	2.6287 mL

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

BIOLOGICAL ACTIVITY

Description

RN-18 is a HIV-1 viral infectivity factor (HIV-1 Vif) inhibitor with an IC_{50} of 6 μM in nonpermissive H9 cells.

IC_{50} & Target

IC50: 6 μM (nonpermissive H9 cell)[1]

In Vitro

RN-18 and RN-19 exhibits potent antiviral activity in the nonpermissive H9 and CEM cells but not in MT4 or CEM-SS cells, confirming that the antiviral activity was Vif specific. RN-18 shows the greater potency (IC_{50}=4.5 μM in CEM cells) and specificity (IC_{50}>100 μM in MT4 cells) among the two compounds[1]. In the presence of the inhibitor, RN-18, reverse transcriptase activity in the nonpermissive H9 and CEM cells decreases substantially and in a dose-dependent manner. RN-18 also exhibits antiviral activity in CEM-SS modified to stably express A3G but does not exhibit antiviral activity in the parental CEM-SS cell line. RN-18 antagonizes Vif function and inhibits HIV-1 replication only in the presence of A3G. RN-18 increases cellular A3G levels in a Vif-dependent manner and increases A3G incorporation into virions without inhibiting general proteasome-mediated protein degradation. RN-18 enhances Vif degradation only in the presence of A3G, reduces viral infectivity by increasing A3G incorporation into virions and enhances cytidine deamination of the viral genome[2].

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

PROTOCOL

Cell Assay [2]

H9 or MT4 cells are treated overnight with 0, 1, 5, 10, 25 or 50 μM RN-18 (all at 0.1% DMSO) and infected with HIV-1. All cells are maintained in the presence of DMSO or RN-18 for 14 d, and viral replication is monitored every 2 d by measuring reverse transcriptase activity in culture supernatants. The average % relative infectivity at day 7 is determined from 3 separate reverse transcriptase assays. Grafit software is used to fit curves and to determine IC_{50}[2].

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

REFERENCES

[1]. Mohammed I, et al. SAR and Lead Optimization of an HIV-1 Vif-APOBEC3G Axis Inhibitor. ACS Med Chem Lett. 2012 Jun 14;3(6):465-469.

[2]. Nathans R, et al. Small-molecule inhibition of HIV-1 Vif. Nat Biotechnol. 2008 Oct;26(10):1187-92.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA