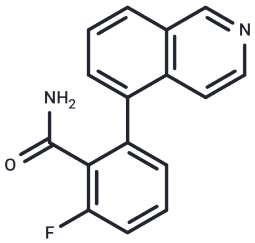
Data Sheet (Cat.No.T204819)

TargetMol

GHP-88309

Chemical Properties



CAS No. : 1269267-87-9

Formula: C16H11FN2O

Molecular Weight: 266.27

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	GHP-88309 is an orally active broad-spectrum anti-paramyxovirus agent that targets viral polymerase and interrupts viral RNA synthesis. GHP-88309 inhibits respiratory syncytial virus (RSV), measles virus (MeV), and canine distemper virus (CDV), with EC50 values ranging from 0.06 to 1.2 μM. In mouse models, GHP-88309 demonstrates significant anti-infective activity. The antiviral profile of GHP-88309 supports the application of GHP-88309 in virology research, viral replication studies, antiviral agent development, and investigations of paramyxovirus biology.
Targets(IC50)	DNA/RNA Synthesis
In vitro	Method: The antiviral activity of GHP-88309 was evaluated in dose-response assays against recombinant human parainfluenza virus type 3 strain JS, human parainfluenza virus type 1 strain 5F6, Sendai virus F1R-eGFP, measles virus strain Alaska.USA/16.00, and canine distemper virus strain 5804p in virus-permissive cultured cells. Rresult: GHP-88309 inhibited the five viruses with EC50 values of 0.4, 0.8, 2.0, 0.6, and 0.4 μM, respectively.[1] Method: Wild-type Nipah virus polymerase and an H1165Y L-protein mutant were evaluated in a minigenome reporter assay in the presence of graded concentrations of GHP-88309. Rresult: Wild-type Nipah virus polymerase was poorly susceptible to GHP-88309, with an EC50 of 314 μM. The H1165Y substitution increased susceptibility approximately 60-fold and reduced the EC50 to 5.3 μM.[1] Method: Vero cells expressing bat CD150 were infected with Myotis bat morbillivirus and treated with increasing concentrations of GHP-88309. Measles virus was evaluated as a susceptible morbillivirus comparator. Rresult: GHP-88309 dose-dependently inhibited Myotis bat morbillivirus and measles virus growth, with EC50 values of 3.0 and 0.6 μM, respectively.[2] Method: The inhibitory activity of GHP-88309 was evaluated against authentic recombinant Cedar virus and tetracistronic transcription- and replication-competent minigenome systems for Cedar virus, Ghana virus, and Nipah virus in BSRT7 cells. Rresult: GHP-88309 inhibited authentic recombinant Cedar virus, the Cedar virus minigenome, and the Ghana virus minigenome with IC50 values of 1.6, 5.7, and 1.7 μM, respectively. The Nipah virus minigenome was not susceptible to GHP-88309.[3] Method: GHP-88309 was docked into structural models of the PIV3 polymerase, wild-type Nipah virus polymerase, and the Nipah virus H1165Y polymerase mutant to investigate the structural basis of differential drug susceptibility.

In vitro	Rresult: In the susceptible PIV3 and Nipah virus H1165Y models, the tyrosine residue was predicted to form a polar interaction with the ring nitrogen of GHP-88309, whereas wild-type Nipah virus H1165 could not form this interaction. Structural analysis further suggested that GHP-88309 may sterically obstruct template-RNA exit and impair efficient polymerase elongation.[4] Method: GHP-88309 was evaluated against recombinant Cedar virus in Vero76 cells, recombinant Pichinde virus and pseudotyped Ebola virus in A549 cells, authentic Ebola virus in HeLa cells, and authentic Nipah virus. Cytotoxicity was evaluated in Vero76 cells. Rresult: GHP-88309 inhibited recombinant Cedar virus with an EC50 of 1.31 ± 0.10 μM and authentic Ebola virus with an EC50 of 9.70 ± 2.55 μM. It produced approximately 50% inhibition of recombinant Pichinde virus at 50 μM, showed an EC50 greater than 100 μM against pseudotyped Ebola virus and greater than 50 μM against authentic Nipah virus, and had a CC50 greater than 100 μM in Vero76 cells.[5] Method: GHP-88309 was applied to cells carrying replication-defective persistent Sendai virus vectors to evaluate whether pharmacological inhibition of Sendai virus polymerase could facilitate vector removal. BRN4-expressing Sendai virus vector persistence was also examined in neural stem cells after enforced differentiation of embryonic stem cells. Rresult: GHP-88309 effectively inhibited Sendai virus replication and enabled removal of persistent Sendai virus vectors. It completely eliminated the BRN4-expressing Sendai virus vector from differentiated neural stem cells.[7]
In vivo	Method: Female 129X1/SvJ mice were infected intranasally with a lethal dose of recombinant Sendai virus and received GHP-88309 orally at 150 mg/kg twice daily, with therapeutic treatment initiated 48 h after infection. Rresult: Therapeutic GHP-88309 treatment provided complete protection against lethal Sendai virus infection, reduced respiratory viral loads, and prevented severe clinical disease.[1] Method: Ferrets received repeated or single oral doses of GHP-88309 to determine nonrodent tolerability and systemic exposure. Rresult: Repeated dosing at 50 mg/kg was tolerated and produced a day-7 plasma Cmax of 43.5 μM and an exposure of 270 μM·h. Repeated dosing at 150 mg/kg caused animals to reach the study endpoint after the second dose and produced a marked reduction in platelet counts. A single 150 mg/kg dose caused transient paralysis and hypothermia, whereas 500 mg/kg caused death within 4 h.[6]

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.7556 mL	18.7779 mL	37.5559 mL
5 mM	0.7511 mL	3.7556 mL	7.5112 mL
10 mM	0.3756 mL	1.8778 mL	3.7556 mL
50 mM	0.0751 mL	0.3756 mL	0.7511 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Cox RM, et al. Orally efficacious broad-spectrum allosteric inhibitor of paramyxovirus polymerase. Nat Microbiol. 2020;5(10):1232-1246.
Ikegame S, et al. Metagenomics-enabled reverse-genetics assembly and characterization of myotis bat morbillivirus. Nat Microbiol. 2023;8:1108-1122.
Haas GD, et al. Tetracistronic minigenomes elucidate a functional promoter for Ghana virus and unveils Cedar virus replicase promiscuity for all henipaviruses. J Virol. 2024;98(10).
Hu S, et al. Structural and functional analysis of the Nipah virus polymerase complex. Cell. 2025;188:688-703.
Gaisina IN, et al. Discovery and optimization of first-in-class furopyrimidine-based inhibitors of henipaviruses. VeriXiv. 2025;2:290. Preprint.
Lieber CM, et al. Developmental candidate GHP-88310/EIDD-3608 with high tolerability and oral efficacy in measles and respiratory paramyxovirus models. Sci Adv. 2026;12.
Osei Mensah E, et al. Enhancing the utility of Sendai virus-based vectors through antiviral compound-mediated removal. J Biotechnol. 2026;414:176-185.

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