

Product Data Sheet

Product Name: Morphothiadin (GLS4)

Cat. No.: GC32073

Chemical Properties

Cas. No. 1092970-12-1

SMILES O=C(C1=C(CN2CCOCC2)NC(C3=NC=CS3)=NC1C4=CC=C(F)C=C4Br)OCC

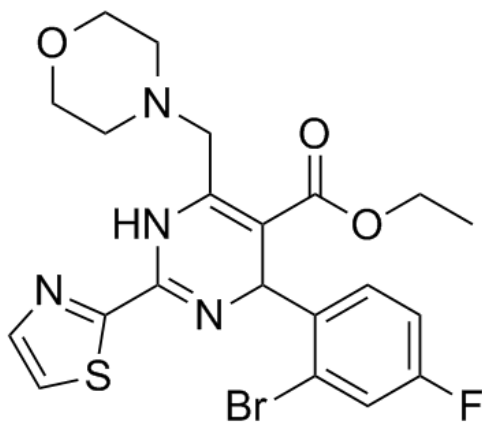
Formula $C_{21}H_{22}BrFN_4O_3S$ M.Wt 509.39

Solubility DMSO : 100 mg/mL (196.31 mM); Water : < 0.1 mg/mL (insoluble) Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines HepAD38 cells

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

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Preparation Method	HepAD38 cells were maintained in DMEM/F12 medium supplemented with 10% fetal bovine serum, and 0.3µg/ml tetracycline (TET). HepAD38 cells were seeded in 96-well microtiter plates (1×10^4 cells/well) for 24h at 37°C and 5% CO ₂ and were treated with media containing different concentrations of Morphothiadin (0, 12.5, 25, 50, 100, 200µM) for 48h, and cell proliferation was measured.
Reaction Conditions	0, 12.5, 25, 50, 100, 200µM; 48h
Applications	Morphothiadin treatment reduced the cell viability of HepAD38 cells in a dose-dependent manner.

Animal experiment [2]:

Animal models	Male C57BL/6 mice
Preparation Method	Male C57BL/6 mice (5-6 weeks old) were kept in temperature-controlled (21°C-25°C) and humidity-maintained (40%-70%) rooms with a 12-h light/dark cycle. Water and food were provided freely. The pAAV/HBV1.2 HDI mouse model was constructed by injecting 10µg pAAV/HBV1.2 plasmid via the tail vein. After 72h, the mice were treated with different therapies for 7 days. The first group (control group) was given daily intragastric administration of olive oil, the second group was given daily Morphothiadin (60mg/kg), the third group was given ritonavir (40mg/kg), and the fourth group was given daily ritonavir (40mg/kg) and Morphothiadin (60mg/kg). During gavage, peripheral blood was collected by retro-orbital bleed for analysis.
Dosage form	60mg/kg/day for 7 days; p.o.
Applications	Morphothiadin treatment combined with ritonavir significantly reduced the HBV DNA levels in the serum of pAAV/HBV1.2 HDI mice.

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References:

- [1] Wu G, Liu B, Zhang Y, et al. Preclinical characterization of GLS4, an inhibitor of hepatitis B virus core particle assembly[J]. Antimicrobial agents and chemotherapy, 2013, 57(11): 5344-5354.
- [2] Zhao L, Xu S, Yao S, et al. GLS4 Induces the Interferon Signaling Pathway During Hepatitis B Virus (HBV) Infection[J]. Journal of Medical Virology, 2026, 98(1): e70777.

Background

Morphothiadin (GLS4) is a potent antiviral agent that inhibits the Hepatitis B virus (HBV) replication with an IC₅₀ value of 12nM [1]. Morphothiadin inhibits HBV replication by interfering with the assembly and disassembly of the viral nucleocapsid, suppresses the production of particles containing HBV pgRNA, and reduces the level of cccDNA[2]. Morphothiadin has been widely used to prevent HBV from infecting cells and to interfere with the formation of the viral core particles[3].

In vitro, Morphothiadin treatment for 48 hours significantly inhibited the viability of primary human hepatocytes and HepAD38 cells, with the CC₅₀ values of 35μM and 26μM, respectively[4].

In vivo, Morphothiadin treatment via daily intragastric administration at a dose of 60mg/kg, and combined with ritonavir (40mg/kg; p.o.) for 7 days significantly reduced the HBV DNA levels in the serum of pAAV/HBV1.2 HDI mice and the expression of HBsAg in the liver, increased the expression of RIG-I in the liver tissue of the mice, and enhanced the IFN signaling pathway[5].

References:

- [1] Zhou X, Gao Z, Meng J, et al. Effects of ketoconazole and rifampicin on the pharmacokinetics of GLS4, a novel anti-hepatitis B virus compound, in dogs[J]. Acta Pharmacologica Sinica, 2013, 34(11): 1420-1426.
- [2] Zhang M, Gao Y, Kong F, et al. Efficacy and safety of GLS4 with entecavir vs entecavir alone in chronic hepatitis B patients: A multicenter clinical trial[J]. Journal of Infection, 2025, 90(3): 106446.
- [3] Wang X Y, Wei Z M, Wu G Y, et al. In vitro inhibition of HBV replication by a novel compound, GLS4, and its efficacy against adefovir-dipivoxil-resistant HBV mutations[J]. Antiviral therapy, 2012, 17(5): 793-803.
- [4] Wu G, Liu B, Zhang Y, et al. Preclinical characterization of GLS4, an inhibitor of hepatitis B virus core particle assembly[J]. Antimicrobial agents and chemotherapy, 2013, 57(11): 5344-5354.

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[5] Zhao L, Xu S, Yao S, et al. GLS4 Induces the Interferon Signaling Pathway During Hepatitis B Virus (HBV) Infection[J]. Journal of Medical Virology, 2026, 98(1): e70777.

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