Favipiravir

Cat. No.: HY-14768

CAS No.: 259793-96-9

Molecular Formula: $C_5H_4FN_3O_2$

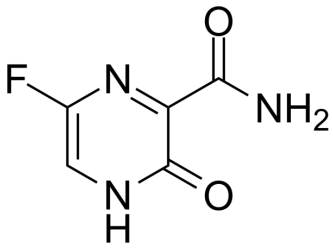
Molecular Weight: 157.1

Target: DNA/RNA Synthesis; Influenza Virus; SARS-CoV; Bacterial

Pathway: Cell Cycle/DNA Damage; 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 (636.54 mM; Need ultrasonic)

H_2O : 6.25 mg/mL (39.78 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	6.3654 mL	31.8269 mL	63.6537 mL
5 mM	1.2731 mL	6.3654 mL	12.7307 mL
10 mM	0.6365 mL	3.1827 mL	6.3654 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 4.55 mg/mL (28.96 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (15.91 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (15.91 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (15.91 mM); Clear solution
5. Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
   Solubility: ≥ 2.5 mg/mL (15.91 mM); Clear solution
6. Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (15.91 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	Favipiravir (T-705) is a potent viral RNA polymerase inhibitor, it is phosphoribosylated by cellular enzymes to its active form, Favipiravir-ribofuranosyl-5′-triphosphate (RTP). Favipiravir-RTP inhibits the influenza viral RNA-dependent RNA polymerase (RdRP) activity with an IC_{50} of 341 nM.
IC_{50} & Target	IC50: 341 nM (RdRP)[1]
In Vitro	Favipiravir (T 705) is an antiviral drug that selectively inhibits the RNA-dependent RNA polymerase of influenza virus. Favipiravir (T 705) is a novel antiviral compound that selectively and potently inhibits the RNA-dependent RNA polymerase (RdRP) of influenza and many other RNA viruses. Favipiravir-RTP does not inhibit the human DNA polymerase α, β or γ with IC_{50}>1 mM. The IC_{50} for the human RNA polymerase II is 905 μM; Favipiravir is therefore 2,650 times more selective for the influenza virus RdRP, consistent with the lack of inhibition of host-cell DNA and RNA synthesis[1]. Favipiravir (T 705) acts as a pro-drug, its cytotoxicity is expected to be cell-line dependent. Favipiravir inhibits in a dose-dependent manner MNV-induced CPE (EC_{50}: 250±11 μM) and MNV RNA synthesis in cell culture (EC_{50}:124±42 μM). Despite this rather modest antiviral activity, Favipiravir (T 705) is able to completely inhibit norovirus replication at a concentration of 100 μg/mL, which is a concentration that has little or no adverse effect on the host cell (cell viability >80%)[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Favipiravir (T 705) (30 mg/kg/day, orally) improves survival compare to placebo. Favipiravir (T 705) also provides significant protection against the A/Duck/MN/1525/81(H5N1) virus at a dose of 33 mg/kg/day or more, regardless of the number of daily doses. When given 4 times a day, all mice survive[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	The antiviral activity of Favipiravir (T 705) is determined using an MTS-based CPE reduction assay in the MNV/RAW 264.7 cell line. To this end, RAW 264.7 cells are seeded (1×10^4 cells/well) in 96-well plates and infected with MNV at an MOI of 0,001 in the presence (or absence) of a dilution series of Favipiravir (T 705) (3.13-200 μg/mL). Following 3 days of incubation, i.e. until complete CPE is observed in infected untreated cells, cell culture supernatants are collected for quantification of viral RNA load by quantitative RT-PCR (qRT-PCR). For the MTS reduction assay an MTS/Phenazine methosulphate (PMS) stock solution (2 mg/mL MTS and 46 g/mL PMS in PBS at pH 6-6.5) is diluted 1/20 in MEM. To each well, 75 μL of MTS/PMS solution is added and the optical density (OD) is read at 498 nm 2 h later. The % CPE reduction is calculated as $[(OD_{treated})_{MNW}-OD_{VC}]/[OD_{CC}-OD_{VC}]\times100$, where OD_{CC} represents the OD of the uninfected untreated cells, whereas OD_{VC} and $(OD_{treated})_{CC}$ represent the OD of infected untreated cells and virus-infected cells treated with a compound concentration, respectively. The EC_{50} is defined as the compound concentration that protected 50% of cells from virus-induced CPE. Adverse effects of the molecule on the host cell are also assessed by means of the MTS-method, by exposing uninfected cells to the same concentrations of Favipiravir for 3 days. The % cell viability is calculated as $(OD_{treated}/OD_{CC})\times100$, where OD_{CC} is the OD of uninfected untreated cells and $OD_{treated}$ are uninfected cells treated with compound. The CC_{50} is defined as the compound concentration that reduces the number of viable cells by 50%. The selectivity index (SI) is calculated as CC_{50}/EC_{50}[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Mice[1] Favipiravir (T 705) has also been shown to protect mice against lethal infection by a variety of influenza virus strains. When Favipiravir is orally administered 2 or 4 times a day for 5 days in mice infected with lethal doses of influenza virus A/Victoria/3/75(H3N2), A/Osaka/5/70(H3N2) or A/Duck/MN/1525/81(H5N1). MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nucleic Acids Res. 2021 Jan 8;49(D1):D1113-D1121.

- Int J Biol Sci. 2020 Oct 16;16(16):3100-3115.
- Cell Chem Biol. 2022 Jun 9;S2451-9456(22)00201-X.
- Antiviral Res. 2023 Aug 21;105703.
- Antiviral Res. 2020 Dec;184:104955.

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REFERENCES

[1]. Furuta Y, et al. Favipiravir (T-705), a novel viral RNA polymerase inhibitor. Antiviral Res. 2013 Nov;100(2):446-54.

[2]. Rocha-Pereira J, et al. Favipiravir (T-705) inhibits in vitro norovirus replication. Biochem Biophys Res Commun. 2012 Aug 10;424(4):777-80.

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