Filanesib

Cat. No.: HY-15187

CAS No.: 885060-09-3

Molecular Formula: $C_{20}H_{22}F_2N_4O_2S$

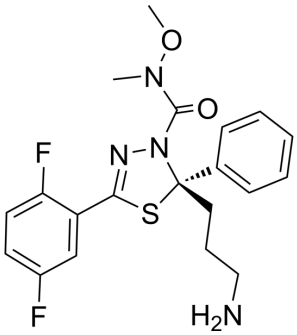
Molecular Weight: 420

Target: Kinesin; Apoptosis

Pathway: Cell Cycle/DNA Damage; Cytoskeleton; Apoptosis

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 (238.10 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.3810 mL	11.9048 mL	23.8095 mL
5 mM	0.4762 mL	2.3810 mL	4.7619 mL
10 mM	0.2381 mL	1.1905 mL	2.3810 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 (5.95 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.95 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.95 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Filanesib (ARRY-520) is a selective and noncompetitive kinesin spindle protein (KSP) inhibitor, with an IC_{50} of 6 nM for human KSP. Filanesib induces cell death by apoptosis in vitro. Filanesib has potent anti-proliferative activity[1].

IC_{50} & Target

KSP

6 nM (IC_{50})

In Vitro

Filanesib induces mitotic arrest in multiple cell lines[1].
Filanesib exhibits anti-proliferative against a broad range of human and rodent tumor cell lines, including a variety of leukemias and solid tumors, with EC_{50}s between 0.4 nM and 14.4 nM[1].
Filanesib (0.001-0.1 nM; 36 hours) induces apoptosis in a dose-dependent manner in HeLa cells[1].
Filanesib (3.13-6.25 nM; 44 hours) causes accumulation of cells in the G2/M phase of the cell cycle in a dose-dependent manner in HeLa cells[1].
Filanesib potently induces cell cycle block and subsequent death in leukemic cells via the mitochondrial pathway and has potential to eradicate AML progenitor cells[2].
Filanesib (3 μM; 6-24 hours) is able to induce caspase-2 activation[3].
Filanesib (0.003-3 μM; 24-48 hours) is cytotoxic in Type II EOC cells[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Apoptosis Analysis[1]

Cell Line:	Hela cells
Concentration:	0.01-0.1 nM
Incubation Time:	36 hours
Result:	Induced the formation of nucleosomes and activation of caspases-3 and 7.

Cell Cycle Analysis[1]

Cell Line:	HeLa cells
Concentration:	0.78 nM, 1.56 nM, 3.13 nM, 6.25 nM
Incubation Time:	44 hours
Result:	Resulted in G2/M arrest.

Western Blot Analysis[3]

Cell Line:	Type II EOC cells
Concentration:	3 μM
Incubation Time:	6 hours, 12 hours, 24 hours
Result:	Induced caspase-2 activation in a time-dependent manner.

Cell Cytotoxicity Assay[3]

Cell Line:	Type II EOC cell lines (A2780, CP70, 01-28)
Concentration:	0.003 μM, 0.03 μM, 0.3μM, 3 μM
Incubation Time:	24 hours , 48 hours
Result:	Effectively decreased cell viability in a time-dependent manner in the Type II EOC cell lines.

In Vivo

Filanesib (20 mg/kg, 30 mg/kg; i.p.; q4dx3) has anti-tumor activitiy in vivo[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female nude mice, EOC mice xenograft model[3]

Dosage:	20 mg/kg, 30 mg/kg
Administration:	Intraperitoneal injection, q4dx3
Result:	Induced a decrease in tumor kinetics in a dose-dependent manner.

CUSTOMER VALIDATION

- Cell Discov. 2022 Sep 14;8(1):92.
- Cancer Lett. 2021 Feb 27.
- Preprints. 2023 Sep 30.
- Methods Mol Biol. 2018;1711:351-398.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Christine Lemieux, et al. ARRY-520, a Novel, Highly Selective KSP Inhibitor with Potent Anti-Proliferative Activity. AACR Annual Meeting. 2007.

[2]. BZ Carter, et al. Inhibition of KSP by ARRY-520 Induces Cell Cycle Block and Cell Death via the Mitochondrial Pathway in AML Cells.

[3]. Ki Hyung Kim, et al. KSP inhibitor ARRY-520 as a substitute for Paclitaxel in Type I ovarian cancer cells. J Transl Med. 2009; 7: 63.

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