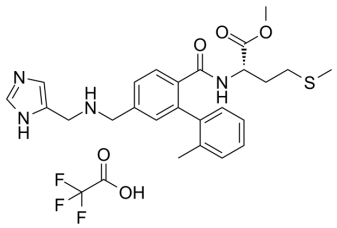
FTI-2153 TFA

Cat. No.:	HY-123242A
CAS No.:	2820151-01-5
Molecular Formula:	$C_{27}H_{31}F_3N_4O_5S$
Molecular Weight:	580.62
Target:	Farnesyl Transferase
Pathway:	Metabolic Enzyme/Protease
Storage:	-20°C, stored under nitrogen * In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 90 mg/mL (155.01 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.7223 mL	8.6115 mL	17.2230 mL
	5 mM	0.3445 mL	1.7223 mL	3.4446 mL
	10 mM	0.1722 mL	0.8611 mL	1.7223 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: ≥ 2.25 mg/mL (3.88 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.25 mg/mL (3.88 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.25 mg/mL (3.88 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

FTI-2153 TFA is a potent and highly selective inhibitor of farnesyltransferase (FTase), with an IC_{50} of 1.4 nM. FTI-2153 TFA is >3000-fold more potent at blocking H-Ras (IC_{50}, 10 nM) than Rap1A processing. Anti-cancer activity[1].

In Vitro

FTI-2153, inhibits bipolar spindle formation during mitosis independently of transformation and Ras and p53 mutation status in two human lung cancer cell lines[2].
FTI-2153 increases the percentage of prometaphase cells with ring-like DNA morphology in transformed and non-transformed cells[2].
FTI-2153 (15 μM) inhibits T-24 and Calu-1 cell growth by 38 and 36%, respectively. NIH3T3, HFF and HT-1080 are less sensitive and are inhibited by only 8, 8 and 13%, respectively. A-549 and OVCAR3 cell growth is inhibited by 25 and 22%,

respectively. Thus, even though T-24 and Calu-1 cells are equisensitive to FTI-2153 cell growth inhibition, FTI-2153 inhibits bipolar spindle formation only in Calu-1 cells. HFF and NIH3T3 cells are both resistant to FTI2153 growth inhibition, yet only NIH3T3 cells are resistant to FTI-2153 inhibition of bipolar spindle formation[2].

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

Cell Viability Assay[2]

Cell Line:	NIH3T3, HFF, HT1080, T-24, OVCAR3, A-549 and Calu-1 CELLS.
Concentration:	48 h.
Incubation Time:	15 μM.
Result:	When A-549 cells were treated with FTI-2153 (15 μM for 48 h), the proportion of cells at prometaphase increased relative to the other phases of mitosis. FTI-2153 accumulated cells at prometaphase with a rosette-like morphology where chromosomes form a ring surrounding a monoaster of microtubules. In all cells, except for T-24 and NIH3T3, FTI-2153 treatment increased the proportion of mitotic cells in prometaphase and decreased the percentage of cells in telophase/cytokinesis. In HT1080 cells, the percentage of cells in prometaphase and telophase/ cytokinesis were 5 and 85% in control cells and 55 and 35% in Treated cells, respectively. Similarly results were also found in HFF cells. Calu-1 and A-549 cells, as described previously, had similarly large changes, whereas OVCAR3 had smaller changes. In contrast, FTI-2153 did not significantly affect the distribution of the different phases of mitosis in T-24 and NIH3T3 cells.

REFERENCES

[1]. Sun J, et al. Antitumor efficacy of a novel class of non-thiol-containing peptidomimetic inhibitors of farnesyltransferase and geranylgeranyltransferase I: combination therapy with the cytotoxic agents cisplatin, Taxol, and gemcitabine. Cancer Res. 1999 Oct 1;59(19):4919-26.

[2]. N C Crespo, et al. The farnesyltransferase inhibitor, FTI-2153, inhibits bipolar spindle formation during mitosis independently of transformation and Ras and p53 mutation status. Cell Death Differ. 2002 Jul;9(7):702-9.

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