Tirbanibulin

Cat. No.: HY-10340

CAS No.: 897016-82-9

Molecular Formula: $C_{26}H_{29}N_3O_3$

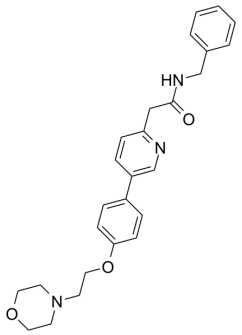
Molecular Weight: 431.53

Target: Src; Microtubule/Tubulin

Pathway: Protein Tyrosine Kinase/RTK; Cell Cycle/DNA Damage; Cytoskeleton

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 : 41.67 mg/mL (96.56 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3173 mL	11.5867 mL	23.1734 mL
	5 mM	0.4635 mL	2.3173 mL	4.6347 mL
	10 mM	0.2317 mL	1.1587 mL	2.3173 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: ≥ 2.08 mg/mL (4.82 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.82 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.82 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Tirbanibulin (KX2-391) is an inhibitor of Src that targets the peptide substrate site of Src, with GI_{50} of 9-60 nM in cancer cell lines.

IC_{50} & Target

GI50: 9 nM (Src, in HuH7 cells), 13 nM (Src, in PLC/PRF/5 cells), 26 nM (Src, in Hep3B cells), 60 nM (Src, in HepG2 cells)[1]

In Vitro

Tirbanibulin (KX2-391) is a Src inhibitor that is directed to the Src substrate pocket. Tirbanibulin (KX2-391) shows steep dose-response curves against Huh7 (GI_{50}=9 nM), PLC/PRF/5 (GI_{50}=13 nM), Hep3B (GI_{50}=26 nM), and HepG2 (GI_{50}=60 nM), four

	hepatic cell cancer (HCC) cell lines[1]. Tirbanibulin (KX2-391) is found to inhibit certain leukemia cells that are resistant to current commercially available drugs, such as those derived from chronic leukemia cells with the T3151 mutation. Tirbanibulin (KX2-391) is evaluated in engineered Src driven cell growth assays inNIH3T3/c-Src527F and SYF/c-Src527F cells and exhibits GI_{50} with 23 nM and 39 nM, respectively[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Orally administered Tirbanibulin (KX2-391) is shown to inhibit primary tumor growth and to suppress metastasis, in pre-clinical animal models of cancer[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Liver cell lines including Huh7, PLC/PRF/5, Hep3B, and HepG2 are routinely cultured and maintained in basal medium containing 2% fetal bovine serum (FBS) at 37°C and 5% CO_2. Cells are seeded at 4.0×10^3/190 µL and 8.0×10^3/190 µL per well of 96-well plate in basal medium containing 1.5% FBS. These are cultured overnight at 37°C and 5% CO_2 prior to the addition of Tirbanibulin (KX2-391), at concentrations ranging from 6,564 to 0.012 nM in triplicates. Treated cells are incubated for 3 days. Ten µLs of 3-(4,5-dimethylthiazol- 2-yl)-2,5-diphenyltetrazolium bromide (MTT) solution (5 mg/mL) is then added to each well on day 3 and cells incubated for 4 hours. The formazan product is dissolved with 10% SDS in dilute HCl. Optical density at 570 nm is measured. For comparison of activity and potency, parallel experiments are performed using Tirbanibulin (KX2-391). Growth inhibition curves, 50% inhibition concentration (GI_{50}), and 80% inhibition concentration (GI_{80}) are determined using GraphPad Prism 5 statistical software. Data are normalized to represent percentage of maximum response as well as reported in optical density at wavelength of 570 nm (OD570) signal format[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Sci Transl Med. 2018 Jul 18;10(450):eaaq1093.
- J Biol Chem. 2019 Nov 29;294(48):18099-18108.
- Brain Res. 2020 Jun 1;1736:146782.
- bioRxiv. 2023 Dec 14.
- Harvard Medical School LINCS LIBRARY

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REFERENCES

[1]. Lau GM, et al. Expression of Src and FAK in hepatocellular carcinoma and the effect of Src inhibitors on hepatocellular carcinoma in vitro. Dig Dis Sci, 2009, 54(7), 1465-1474.

[2]. Fallah-Tafti A, et al. Thiazolyl N-benzyl-substituted acetamide derivatives: synthesis, Src kinase inhibitory and anticancer activities. Eur J Med Chem, 2011, 46(10), 4853-4858.

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