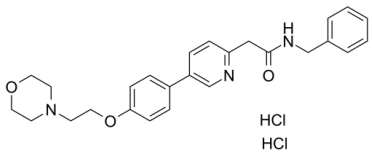
Data Sheet

Product Name:	Tirbanibulin (dihydrochloride)
Cat. No.:	CS-0455
CAS No.:	1038395-65-1
Molecular Formula:	C26H31Cl2N3O3
Molecular Weight:	504.45
Target:	Microtubule/Tubulin; Src
Pathway:	Cell Cycle/DNA Damage; Cytoskeleton; Protein Tyrosine Kinase/RTK
Solubility:	DMSO : 33.33 mg/mL (66.07 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

Tirbanibulin (dihydrochloride) (KX2-391 (dihydrochloride)) is an inhibitor of **Src** that targets the peptide substrate site of Src, with **GI_{50}** of 9-60 nM in cancer cell lines. IC50 & Target: GI50: 9 nM (Src HuH7), 13 nM (Src PLC/PRF/5), 26 nM (Src Hep3B), 60 nM (Src HepG2) **In Vitro:** Tirbanibulin (KX2-391) is a Src inhibitor that is directed to the Src substrate pocket. 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]. **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].

PROTOCOL (Extracted from published papers and Only for reference)

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.

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.

CAIndexNames:

2-Pyridineacetamide, 5-[4-[2-(4-morpholinyl)ethoxy]phenyl]-N-(phenylmethyl)-, hydrochloride (1:2)

SMILES:

O=C(CC1=NC=C(C2=CC=C(OCCN3CCOCC3)C=C2)C=C1)NCC4=CC=CC=C4.Cl.Cl

Tel: 732-484-9848 Fax: 888-484-5008 E-mail: sales@ChemScene.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA