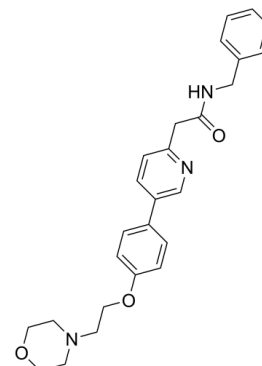


Data Sheet

Product Name:	Tirbanibulin
Cat. No.:	CS-0248
CAS No.:	897016-82-9
Molecular Formula:	C ₂₆ H ₂₉ N ₃ O ₃
Molecular Weight:	431.53
Target:	Microtubule/Tubulin; Src
Pathway:	Cell Cycle/DNA Damage; Cytoskeleton; Protein Tyrosine Kinase/RTK
Solubility:	DMSO : 41.67 mg/mL (ultrasonic)



BIOLOGICAL ACTIVITY:

Tirbanibulin (KX2-391) is an inhibitor of **Src** that targets the peptide substrate site of Src, with **GI₅₀** of 9-60 nM in cancer cell lines. IC₅₀ & 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 (GI₅₀=9 nM), PLC/PRF/5 (GI₅₀=13 nM), Hep3B (GI₅₀=26 nM), and HepG2 (GI₅₀=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 in NIH3T3/c-Src527F and SYF/c-Src527F cells and exhibits GI₅₀ 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: Tirbanibulin (KX2-391) is prepared in DMSO and stored, and then diluted with appropriate medium before use^{[1],[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% CO₂. Cells are seeded at 4.0×10³/190 μL and 8.0×10³/190 μL per well of 96-well plate in basal medium containing 1.5% FBS. These are cultured overnight at 37°C and 5% CO₂ 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 (GI₅₀), and 80% inhibition concentration (GI₈₀) 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].

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)-

SMILES:

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

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: 610-426-3128

Fax: 888-484-5008

E-mail: sales@ChemScene.com

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