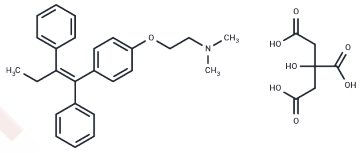
Data Sheet (Cat.No.T0835)

TargetMol

Tamoxifen Citrate

Chemical Properties



CAS No. : 54965-24-1

Formula: C32H37NO8

Molecular Weight: 563.65

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Tamoxifen Citrate is an orally active selective estrogen receptor modulator (SERM) that blocks estrogen activity in breast cells while activating estrogen signaling in cells such as those of the bone, liver, and uterus. As an Hsp90 activator, it enhances the ATPase activity of the Hsp90 chaperone complex, induces autophagy and apoptosis, and can be used to generate gene knockout models in CreER(T2) transgenic mice as well as liver injury models.
Targets(IC50)	Apoptosis,Estrogen Receptor/ERR,HSP,Estrogen/progestogen Receptor,Autophagy
In vitro	Tamoxifen displays antitumor effect due to its antiestrogenic activity (ER). Values for the apparent affinity of Tamoxifen for the ER range between 30 and 0.01% of that obtained for estradiol, dependent on different ER source (species), protein concentration and condition used for assay. Binding of Tamoxifen to ER further leads to inhibition expression of estrogen-regulated genes, including growth factors and angiogenic factors secreted by the tumor that may stimulate growth by autocrine or paracrine mechanisms. Tamoxifen also directly induces programmed cell death. [1] Tamoxifen produces an inhibitory effect on MCF-7 cell [3H]thymidine incorporation and DNA polymerase activity as well as causing a reduction in DNA content of cultures and cell numbers. This inhibitory effect of Tamoxifen on MCF-7 cell growth can be readily reversed by addition of estradiol to the culture medium. 2 and 6 μM Tamoxifen reduces the proportion of cells in S phase and increases the number of cells in G1. At 10 μM, Tamoxifen causes cell death within 48 hr. [2] Tamoxifen inhibits MCF-7 growth with IC50 of ~10 nM after 10 days treatment. Tamoxifen inhibits plasminogen activator activity of MCF-7, and suppresses estradiol-stimulation of plasminogen activator activity. Tamoxifen also evokes minimal increases in cellular progesterone receptor levels. [3] Tamoxifen is able to inhibit the growth of prostate cancer cell PC3, PC3-M, and DU145 with IC50 ranged from 5.5-10 μM, which is related to its inhibition of protein kinase C and induction of p21(waf1/cip1). [4]
In vivo	Tamoxifen administration to rapidly growing, estradiol-stimulated MCF-7 xenografts results in a dose-dependent retardation or cessation of tumor growth by significantly decreasing tumor cell proliferation in tumor. Tamoxifen treatment results in a slowing of tumor growth (tumor doubling time, 12 days), a significant increase in tumor potential doubling time (Tpot) (6.6 days), and a decrease in labeling index (%LI) (to 8%) by 23 days posttreatment, compared with untreated mice which shows a volume doubling time of 5 days, a Tpot of 2.3 days, and a %LI of 23%. [5] Tamoxifen has not only

In vivo	antiestrogenic but also estrogenic properties depending on the species, tissue, and gene. Tamoxifen displays favorable effects on bone and serum lipid concentrations and stimulation endometrium. [1]
Kinase Assay	Competitive binding assays: Cells are harvested from 150-sq cm T-flasks, and cytosol is prepared at a protein concentration of approximately 2 mg/mL in phosphate buffer. Aliquots of this 180,000 ×g supernatant are then incubated with various concentrations of Tamoxifen and 2.5 nM [3H]estradiol for 16 hr at 0-4 ℃. The free steroids are absorbed by dextran-charcoal [l0 μL of 0.5% Dextran C-5% Norite A in TE buffer] for 1 hr at 0 ℃, and aliquots are counted after centrifugation at 800 ×g, 30 min. The relative binding ability of each competitor is taken as the ratio of the concentration of radioinert estradiol/competitor required to inhibit one-half of the specific [3H]estradiol binding, with the affinity of estradiol set at 100%.
Cell Research	MCF-7 cells are seeded into T-25 flasks (1.5×105 cells/flask) and grown for 2 days in the MEM supplemented with 10 mM HEPES buffer, gentamicin (50 μg/mL), penicillin (100 units/mL), streptomycin (0.1 mg/mL), bovine insulin (6 ng/mL), hydrocortisone (3.75 ng/mL), and 5% calf serum that has been treated with dextran-coated charcoal for 45 min at 55 ℃ to remove endogenous hormones. The medium is then changed to MEM supplemented as described above, except that it contains 2% charcoal dextran-treated calf serum and various concentrations of Tamoxifen. At the end of incubation, cell numbers are counted. (Only for Reference)

Solubility Information

Solubility	DMSO: 250 mg/mL (443.54 mM),Sonication is recommended. Ethanol: 2.8 mg/mL (4.97 mM),Heating is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2 mg/mL (3.55 mM),Sonication is recommended. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	1.7742 mL	8.8708 mL	17.7415 mL
5 mM	0.3548 mL	1.7742 mL	3.5483 mL
10 mM	0.1774 mL	0.8871 mL	1.7742 mL
50 mM	0.0355 mL	0.1774 mL	0.3548 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Osborne CK. N Engl J Med, 1998, 339(22), 1609-1618.
Yan X, Luo C, Yang J, et al.Antiviral Activity of Selective Estrogen Receptor Modulators against Severe Fever with Thrombocytopenia Syndrome Virus In Vitro and In Vivo.Viruses.2024, 16(8): 1332.
Furr BJ, et al. Pharmacol Therm, 1984, 25(2), 127-205.
Katzenellenbogen BS, et al. Cancer Res, 1984, 44(1), 112-119.
Rohlff C, et al. Prostate, 1998, 37(1), 51-59.
Sarkaria JN, et al. Cancer Res, 1993, 53(18), 4413-4417.

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