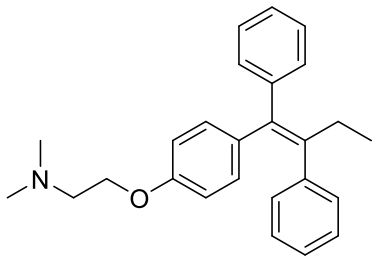
Product data sheet

MedKoo Cat#: 574097 Name: Tamoxifen CAS#: 10540-29-1 (free base) Chemical Formula: $C_{26}H_{29}NO$ Exact Mass: 371.2249 Molecular Weight: 371.52	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
Tamoxifen is a selective estrogen receptor modulator (SERM) and antagonist of ER action in breast tissue and breast cancer cells. It is reported to be effective in the treatment of early breast cancer to prevent tumor growth. Importantly, tamoxifen has been reported to act as an ER agonist in bone and blood vessels, helping to minimize osteoporosis and reduce the risk of cardiovascular disease in post-menopausal women. Also, tamoxifen is a partial ER agonist in uterine tissues and is reported to increase the risk of endometrial carcinoma.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	20.0	53.83
Ethanol	45.0	121.12

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	**1 mg**	**5 mg**	**10 mg**
1 mM	2.69 mL	13.46 mL	26.92 mL
5 mM	0.54 mL	2.69 mL	5.38 mL
10 mM	0.27 mL	1.35 mL	2.69 mL
50 mM	0.05 mL	0.27 mL	0.54 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Weinstock A, Gallego-Delgado J, Gomes C, Sherman J, Nikain C, Gonzalez S, Fisher E, Rodriguez A. Tamoxifen activity against Plasmodium in vitro and in mice. Malar J. 2019 Nov 27;18(1):378. doi: 10.1186/s12936-019-3012-7. PMID: 31775753; PMCID: PMC6882195.
2. Huang S, Wang H, Chen W, Zhan M, Xu S, Huang X, Lin R, Shen H, Wang J. Tamoxifen inhibits cell proliferation by impaired glucose metabolism in gallbladder cancer. J Cell Mol Med. 2020 Jan;24(2):1599-1613. doi: 10.1111/jcmm.14851. Epub 2019 Nov 28. PMID: 31782270; PMCID: PMC6991689.

In vivo study

1. Weinstock A, Gallego-Delgado J, Gomes C, Sherman J, Nikain C, Gonzalez S, Fisher E, Rodriguez A. Tamoxifen activity against Plasmodium in vitro and in mice. Malar J. 2019 Nov 27;18(1):378. doi: 10.1186/s12936-019-3012-7. PMID: 31775753; PMCID: PMC6882195.
2. Mao D, Mi J, Pan X, Li F, Rui Y. Tamoxifen Inhibits the Progression of Trauma-Induced Heterotopic Ossification in Mice. Med Sci Monit. 2019 Oct 21;25:7872-7881. doi: 10.12659/MSM.916733. PMID: 31631887; PMCID: PMC6820362.

Product data sheet

7. Bioactivity

Biological target:

Tamoxifen (ICI 47699) is a selective estrogen receptor modulator (SERM) that is a potent Hsp90 activator and enhances the Hsp90 molecular chaperone ATPase activity as well as inhibits infectious EBOV Zaire and Marburg (MARV) with IC50 of 0.1 μM and 1.8 μM, respectively.

In vitro activity

Gallbladder cancer cells treated with TAM (Tamoxifen) indeed induced AMPK activation, which was accompanied by decreased phosphorylation of mTOR (p‐mTOR) (Figure3E), a critical regulator of cancer cell glycolysis, indicating impaired glycolysis. To determine whether the effect of glycolysis inhibition by TAM was dependent on the upstream activation of ROS, the level of p‐AMPK was measured in GBC cells treated with TAM alone or along with NAC. As shown in Figure 3F, addition of NAC significantly attenuated the phosphorylation of AMPK induced by TAM. Importantly, NAC also recovered the glucose uptake and the production of lactate down‐regulated by TAM treatment, which were consistent with the effect of NAC on TAM‐induced apoptosis (Figure3G). Together, these data provide evidence that TAM promoted GBC apoptosis through impaired glycolysis via ROS production. Since we have demonstrated TAM suppresses glycolysis via activation of AMPK, compound C (AMPK inhibitor) and AMPK knockdown were used to further evaluate whether the AMPK signalling pathway is required for TAM‐induced suppression of GBC cells. As shown, AMPK inhibitor compound C (CC) reversed the pro‐apoptotic effect of TAM (Figure3H,I). In lines with the effect, CC dramatically abrogated AMPK phosphorylation (Figure3J).GBC cells with AMPK knockdown also exhibited stronger resistance to TAM (Figure3K,L). Taken together, these data indicated that TAM inhibited GBC cell growth largely by activating AMPK signalling.

J Cell Mol Med. 2020 Jan; 24(2): 1599–1613. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6991689/

In vivo activity

To further illustrate the curative effect of tamoxifen for different HO progression stages, mice were administered with tamoxifen (9 mg/kg) every other day for a total of 3 weeks post puncture at different stages of HO progression mainly including inflammatory stage (Day 1–Week 3), chondrogenesis stage (Week 4–Week 6), osteogenesis stage (Week 7–Week 9) and maturation stage (Week 10–Week 12) . Analysis of samples scanned by μCT revealed that the bone volume of HO was both significantly decreased in mice with the treatment of tamoxifen from day 1 and week 4, respectively ($p<0.05$) (Figure 3A, 3B), whereas no significant reduction was found at week 7 and week 10 groups compared to vehicle-treated mice (Figure 3B). The bone marrow cavity showed by H&E staining was diminished with the treatment of tamoxifen from day 1 and week 4 relative to week 7, week 10 and vehicle groups (Figure 3C). In addition, the number of Ocn+ osteoblasts (Figure 3D, 3E) and p-Smad2/3+ cells (Figure 3F–3I) were both significantly reduced in mice treated with tamoxifen at inflammatory and chondrogenesis stages relative to vehicle-administered mice ($p<0.05$), showed an inhibitory bone propagation. The downtrends in Ocn and p-Smad2/3 protein levels are consistent with immunohistochemistry results, and administration of tamoxifen at different periods can significantly improve the activity of ERα (Figure 3J). Collectively, all these results demonstrated that HO propagation could be attenuated by tamoxifen at the early stages of inflammation and chondrogenesis accompanied with the TGF-β signaling pathway was suppressed by ERα.

Med Sci Monit. 2019; 25: 7872–7881. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6820362/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.