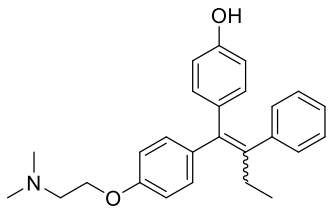
Product data sheet

MedKoo Cat#: 200120 Name: Afimoxifene CAS#: 68392-35-8 Chemical Formula: $C_{26}H_{29}NO_2$ Exact Mass: 387.21983 Molecular Weight: 387.51	
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:

Afimoxifene (4-hydroxytamoxifen) is a selective estrogen receptor modulator which is the active metabolite of tamoxifen. Afimoxifene is a transdermal gel formulation and is being developed by Ascend Therapeutics, Inc. under the trademark TamoGel. Afimoxifene has completed a phase II clinical trial for the treatment of cyclical mastalgia. A study in France on 55 women showed that rubbing afimoxifene on the skin was as good as tamoxifen tablets at slowing breast cancer growth. A US trial will compare 6 weeks use before breast cancer surgery. Skin application can reduce systemic levels by a factor of nine and this is expected to reduce the unpleasant side-effects of tamoxifen. (source: http://en.wikipedia.org/wiki/Afimoxifene).

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	54.11	139.64
DMF	20.0	51.61
Ethanol	20.0	51.61
Ethanol:PBS (pH 7.2) (1:2)	0.3	0.77

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.58 mL	12.90 mL	25.81 mL
5 mM	0.52 mL	2.58 mL	5.16 mL
10 mM	0.26 mL	1.29 mL	2.58 mL
50 mM	0.05 mL	0.26 mL	0.52 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. Kohli L, Kaza N, Coric T, Byer SJ, Brossier NM, Klocke BJ, Bjornsti MA, Carroll SL, Roth KA. 4-Hydroxytamoxifen induces autophagic death through K-Ras degradation. Cancer Res. 2013 Jul 15;73(14):4395-405. doi: 10.1158/0008-5472.CAN-12-3765. Epub 2013 May 30. PMID: 23722551; PMCID: PMC3715566.

In vivo study

1. Heinen A, Gödecke S, Flögel U, Miklos D, Bottermann K, Spychala A, Gödecke A. 4-hydroxytamoxifen does not deteriorate cardiac function in cardiomyocyte-specific MerCreMer transgenic mice. Basic Res Cardiol. 2021 Feb 5;116(1):8. doi: 10.1007/s00395-020-00841-9. PMID: 33544211; PMCID: PMC7864833.

2. El Gebeily G, Fiset C. 4-Hydroxytamoxifen inhibits K(+) currents in mouse ventricular myocytes. Eur J Pharmacol. 2010 Mar 10;629(1-3):96-103. doi: 10.1016/j.ejphar.2009.12.006. Epub 2009 Dec 16. PMID: 20006599.

7. Bioactivity

Biological target:

(E/Z)-4-Hydroxytamoxifen is an estrogen receptor modulator.

In vitro activity

Relative to untreated cells, OHT (Afimoxifene)- treated cells demonstrated a dramatic increase in steady state levels of AVs (Fig.1C). LC3 II can accumulate in response to increased autophagy induction and/or decreased AV degradation. Therefore, autophagic flux was measured in control and OHT-treated cells using BafB1, which inhibits vacuolar ATPase, a molecule active in the late stage of autophagy. OHT- treated cells displayed increased autophagic flux, indicating that the increase in steady state AV levels was due, at least in part, to increased autophagy induction by OHT (Fig.1D). To assess the functional significance of this phenomenon, this study next inhibited the initiation of autophagy by transfecting cells with siRNA targeting Atg7, a critical regulator of AV formation (Fig.1E). Atg7 knockdown partially protected MPNST cells from OHT-induced death (Fig.1F). OHT triggers autophagic death in MPNST cells.

Reference: Cancer Res. 2013 Jul 15; 73(14): 4395–4405. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3715566/

In vivo activity

Fig. 2A illustrates typical examples of a family of total K^+ currents (I_{peak}) obtained in the same voltage-clamped mouse ventricular myocyte before and after 30 min exposure to 10 µM of 4OH-tamoxifen. Fig. 2B summarizes the corresponding current–voltage (I–V) relationships of I_{peak}. At this concentration, 4OH-tamoxifen significantly decreased the density of both the inward and outward portions of I_{peak}. Data presented in Fig. 2B also shows that 10 µM of 4OH-tamoxifen significantly decreased the density of the outward portion of the total K^+ current for voltages ranging between − 30 and + 50 mV (at + 30 mV, control: 61.3 ± 5.1 $pApF^{-1}$; 4OH-tamoxifen: 38.2 ± 4.1 $pApF^{-1}$, n = 12, $P < 0.01$). The density of I_{peak} measured at + 30 mV was reduced when the cells were perfused with 0.5 µM of 4OH-tamoxifen from 63.7 ± 4.4 $pApF^{-1}$ to 49.4 ± 5.4 $pApF^{-1}$ (n = 9; $P < 0.05$). Similarly, 1 µM 4OH-tamoxifen reduced I_{peak} from 62.9 ± 7.0 $pApF^{-1}$ to 45.8 ± 5.5 $pApF^{-1}$ (n = 10; $P < 0.01$). Data presented in this figure were obtained from ventricular myocytes isolated from 7 different female mice.

Reference: Eur J Pharmacol. 2010 Mar 10;629(1-3):96-103. https://pubmed.ncbi.nlm.nih.gov/20006599/

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.