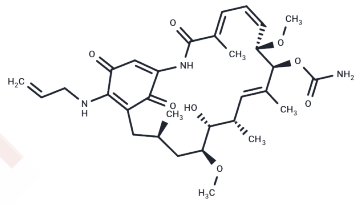
Data Sheet (Cat.No.T6290)

TargetMol

Tanespimycin

Chemical Properties



CAS No. : 75747-14-7

Formula: C31H43N3O8

Molecular Weight: 585.69

Storage: Keep away from direct sunlight,Keep away from moisture,Store at low temperature
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	Tanespimycin (KOS 953) is an Hsp90 inhibitor (IC50=5 nM) and is selective. Tanespimycin depletes intracellular STK38/NDR1 and decreases STK38 kinase activity. Tanespimycin also downregulated stk38 gene expression.
Targets(IC50)	Apoptosis,Mitophagy,HSP,Antibacterial,Antibiotic,Autophagy
In vitro	**METHODS**: Human A-431, A549, BGC-823, HepG2, HUVEC, L02, and MDA-MB-231 cells were treated with Tanespimycin (0-10 μM) for 72 hours, and the cell growth inhibition was detected by MTT assay. **RESULTS**: Tanespimycin inhibited A-431 (IC50=89 nM), A549 (IC50=81 nM), BGC-823 (IC50=847 nM), HepG2 (IC50=91 nM), HUVEC (IC50=282 nM), and L02 (IC50=99) nM), MDA-MB-231 (IC50=0.28 μM) cell growth. [1] **METHODS**: CCA cells were treated with Tanespimycin (0.6 μM) for 72 hours, and the expression levels of target proteins were detected by Western Blot. **RESULTS**: Tanespimycin downregulated Bcl-2, Survivin and Cyclin B1 and upregulated cleaved PARP. [2]
In vivo	**METHODS**: To study the antitumor activity of Tanespimycin, lymphoma-inoculated mice were intraperitoneally injected with Tanespimycin (5-40 mg/kg) every other day for three weeks. **RESULTS**: Tanespimycin inhibited lymphoma in vivo. [3]
Kinase Assay	Purified native Hsp90 protein or cell lysates in lysis buffer (20 mM HEPES, pH 7.3, 1 mM EDTA, 5 mM MgCl2, 100 mM KCl) were incubated with or without 17-AAG for 30 min at 4 °C, and then incubated with biotin-GM linked to streptavidin magnetic beads for 1 h at 4 °C. Tubes were placed on a magnetic rack, and the unbound supernatant removed. The magnetic beads were washed three times in lysis buffer and heated for 5 min at 95 °C in SDS-PAGE sample buffer. Samples were analyzed on SDS protein gels, and western blots done using indicated antibodies. Bands in the western blots were quantified, and the percentage inhibition of binding of Hsp90 to the biotin-GM was calculated. The IC50 reported is the concentration of 17-AAG needed to cause half-maximal inhibition of binding. For in vitro reconstitution, 5 μM of purified Hsp90 was combined with 1 μM each of Hsp70, Hsp40, p23, and Hop purified proteins [1].

Cell Research	Cells were seeded in 96-well plates at 2,000 cells per well in a final culture volume of 100 μl for 24 h before the addition of increasing concentrations of 17-AAG that was incubated for 5 days. Viable cell number was determined using the Celltiter 96 AQueous Nonradioactive Cell Proliferation Assay. The value of the background absorbance at 490 nm (A490) of wells not containing cells was subtracted. Percentage of viable cells ? (A490 of 17-AAG treated sample/A490 untreated cells) × 100. The IC50 was defined as the concentration that gave rise to 50% viable cell number [1].
Animal Research	B10.BR mice were inoculated with 5×10^5 lymphoma cells through intraperitoneal injection. Seven days following tumor implantation, the mice were I.P. injected with 17-AAG or vehicle (10% DMSO + 40% Cremophor EL: Ethanol (3:1) (v/v) + 50 % PBS) every other day for three weeks. At the cessation of treatment, mice were monitored up to 80 days post tumor cell injection. To determine the effects of 17-AAG on lymphoma initiation in vivo, secondary B10.BR recipient mice were implanted by intraperitoneal injection of 1×10^5 lymphoma cells from the spleens of first-round mice that had been treated with 17-AAG or vehicle. These mice were followed up to 160 days post tumor cell injection to monitor differences in tumor initiation between the mice [4].

Solubility Information

Solubility	DMSO: 50.5 mg/mL (86.22 mM),Sonication 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: 1 mg/mL (1.71 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	1.7074 mL	8.5369 mL	17.0739 mL
5 mM	0.3415 mL	1.7074 mL	3.4148 mL
10 mM	0.1707 mL	0.8537 mL	1.7074 mL
50 mM	0.0341 mL	0.1707 mL	0.3415 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

Li Z,et al. Discovery of diamine-linked 17-aroylamido-17-demethoxygeldanamycins as potent Hsp90 inhibitors. Eur J Med Chem. 2014 Nov 24;87:346-63.
Wu Z, Geng Y, Lu X, et al. Chaperone-mediated autophagy is involved in the execution of ferroptosis. Proceedings of the National Academy of Sciences. 2019 Feb 19;116(8):2996-3005
Xiong J, Wang L, Feng Y, et al.Geldanamycin confers fungicidal properties to azole by triggering the activation of succinate dehydrogenase.Life Sciences.2024: 122699.
Zhang J, et al. The heat shock protein 90 inhibitor 17-AAG suppresses growth and induces apoptosis in human cholangiocarcinoma cells. Clin Exp Med. 2013 Nov;13(4):323-8.
Chen H, He A, Li H, et al. TSSK4 upregulation in alveolar epithelial type-II cells facilitates pulmonary fibrosis through HSP90-AKT signaling restriction and AT-II apoptosis. Cell Death & Disease. 2021, 12(10): 1-1
Cheng Y, Wang Q, Zhang Z, et al. Saucerneol attenuates nasopharyngeal carcinoma cells proliferation and metastasis through selectively targeting Grp94. Phytomedicine. 2022: 154133
Newman B,et al. HSP90 inhibitor 17-AAG selectively eradicates lymphoma stem cells. Cancer Res. 2012 Sep 1;72 (17):4551-61.
Newman B, et al. HSP90 Inhibitor 17-AAG Selectively Eradicates Lymphoma Stem Cells.Cancer Res. 2012 Sep 1;72 (17):4551-61. Epub 2012 Jun 29.
Zuo Y, Xu H, Chen Z, et al. 17-AAG synergizes with Belinostat to exhibit a negative effect on the proliferation and invasion of MDA-MB-231 breast cancer cells. Oncology Reports. 2020, 43(6): 1928-1944.
Peng Y C, Wang S, Zhang Y, et al. Hsp90β inhibitors prevent GLT-1 degradation but have no beneficial efficacy on absence epilepsy. Journal of Asian Natural Products Research. 2018 Nov 17:1-11
Peng Y C, Wang S, Zhang Y, et al. Hsp90β inhibitors prevent GLT-1 degradation but have no beneficial efficacy on absence epilepsy[J]. Journal of Asian natural products research. 2018 Nov 17:1-11.
Zuo Y, Xu H, Chen Z, et al. 17-AAG synergizes with Belinostat to exhibit a negative effect on the proliferation and invasion of MDA-MB-231 breast cancer cells[J]. Oncology Reports. 2020, 43(6): 1928-1944.
Qiu C, Shen X, Lu H, et al.Combination therapy with HSP90 inhibitors and piperlongumine promotes ROS-mediated ER stress in colon cancer cells.Cell Death Discovery.2023, 9(1): 375.
Wang C, Wang T, Hu R, et al.9-Butyl-Harmol Exerts Antiviral Activity against Newcastle Disease Virus through Targeting GSK-3β and HSP90β.Journal of Virology.2023: e01984-22.
Wu Z, Geng Y, Lu X, et al. Chaperone-mediated autophagy is involved in the execution of ferroptosis[J]. Proceedings of the National Academy of Sciences. 2019 Feb 19;116(8):2996-3005.
Zhang M, Tan H, Gong Y, et al.TRIM26 restricts Epstein-Barr virus infection in nasopharyngeal epithelial cells through K48-linked ubiquitination of HSP-90β.The FASEB Journal.2024, 38(1): e23345.
Huang Z, Li S, Zhong L, et al.Effect of resveratrol on herpesvirus encephalitis: evidences for its mechanisms of action.Phytomedicine.2024: 155476.