Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

Tanespimycin

Catalog No: tcsc0161

Available Sizes

Size: 10mg

Size: 25mg

Size: 100mg

Size: 200mg

Specifications

CAS No:
75747-14-7

Formula:
$C_{31}H_{43}N_3O_8$

Pathway:
Autophagy;Metabolic Enzyme/Protease;Cell Cycle/DNA Damage;Autophagy

Target:
Autophagy;HSP;HSP;Mitophagy

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 55 mg/mL (93.91 mM)

Alternative Names:
17-AAG;NSC 330507;CP 127374

Observed Molecular Weight:
585.69

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Product Description

17-AAG is a potent **HSP90** inhibitor with **IC_{50}** of 5 nM, having a 100-fold higher binding affinity for HSP90 derived from tumour cells than HSP90 from normal cells.

IC50 & Target: IC50: 5 nM (HSP90)[5]

In Vitro: 17-AAG causes the degradation of HER2, Akt, and both mutant and wild-type AR and the retinoblastoma-dependent G1 growth arrest of prostate cancer cells. 17-AAG inhibits prostate cancer cell lines with IC_{50}s ranged from 25-45 nM (LNCaP, 25 nM; LAPC-4, 40 nM; DU-145, 45 nM; and PC-3, 25 nM)[1]. Combination of 17-AAG (10 nM) and Trastuzumab induces more effective ErbB2-degradation. 17-AAG (0.1-1 μM) induces a nearly complete loss of ErbB2 on ErbB2-overexpressing breast cancer cells[2]. 17-AAG inhibits cell growth and induces G2/M cell cycle arrest and apoptosis in CCA cells together with the down-regulation of Bcl-2, Survivin and Cyclin B1, and the up-regulation of cleaved PARP[3].

In Vivo: 17-AAG (25-200 mg/kg, i.p.) causes a dose-dependent decline in AR, HER2, and Akt expression in prostate cancer xenografts. 17-AAG treatment at doses sufficient to induce AR, HER2, and Akt degradation results in the dose-dependent inhibition of androgen-dependent and -independent prostate cancer xenograft growth without toxicity[1]. 17-AAG (60 mg/kg) with paclitaxel (60 mg/kg) and rapamycin (30 mg/kg) inhibits A549 and MDA-MB-231 tumor growth far more potently than paclitaxel-containing micelles and effected tumor cures in MDA-MB-231 tumor-bearing animals by tail vein injection[4].