**Product Data Sheet**

**Tanespimycin**

**Cat. No.:** HY-10211  
**CAS No.:** 75747-14-7  
**Molecular Formula:** C₃₁H₄₃N₃O₈  
**Molecular Weight:** 585.69  
**Target:** HSP; Autophagy; Mitophagy; Apoptosis  
**Pathway:** Cell Cycle/DNA Damage; Metabolic Enzyme/Protease; Autophagy; Apoptosis  
**Storage:** 4°C, protect from light  
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)

### SOLVENT & SOLUBILITY

**In Vitro**  
DMSO : ≥ 55 mg/mL (93.91 mM)  
H₂O : < 0.1 mg/mL (insoluble)  
* *≥* means soluble, but saturation unknown.

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent Concentration</th>
<th>Mass 1 mg</th>
<th>Mass 5 mg</th>
<th>Mass 10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>1.7074 mL</td>
<td>8.5369 mL</td>
<td>17.0739 mL</td>
<td></td>
</tr>
<tr>
<td>5 mM</td>
<td>0.3415 mL</td>
<td>1.7074 mL</td>
<td>3.4148 mL</td>
<td></td>
</tr>
<tr>
<td>10 mM</td>
<td>0.1707 mL</td>
<td>0.8537 mL</td>
<td>1.7074 mL</td>
<td></td>
</tr>
</tbody>
</table>

Please refer to the solubility information to select the appropriate solvent.

### BIOLOGICAL ACTIVITY

**Description**  
Tanespimycin (17-AAG) is a potent HSP90 inhibitor with an IC₅₀ of 5 nM, having a 100-fold higher binding affinity for tumour cell derived HSP90 than normal cell derived HSP90. Tanespimycin (17-AAG) depletes cellular STK38/NDR1 and reduces STK38 kinase activity. Tanespimycin (17-AAG) also downregulates the stk38 gene expression.

**IC₅₀ & Target**  
<table>
<thead>
<tr>
<th>HSP90</th>
<th>Autophagy</th>
<th>Mitophagy</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 nM (IC₅₀)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**In Vitro**  
Tanespimycin causes the degradation of HER2, Akt, and both mutant and wild-type AR and the retinoblastoma-dependent G1 growth arrest of prostate cancer cells. Tanespimycin inhibits prostate cancer cell lines with IC₅₀s ranged from 25-45 nM (LNCaP, 25 nM; LAPC-4, 40 nM; DU-145, 45 nM; and PC-3, 25 nM)[1]. Tanespimycin (0.1-1 μM) induces a nearly complete loss of ErbB2 on ErbB2-overexpressing breast cancer cells[2]. Tanespimycin 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].

[2] Tanespimycin 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**

Tanespimycin (25-200 mg/kg, i.p.) causes a dose-dependent decline in AR, HER2, and Akt expression in prostate cancer xenografts. Tanespimycin 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\(^1\). Tanespimycin (60 mg/kg) with Rapamycin (30 mg/kg) inhibits A549 and MDA-MB-231 tumor growth and effects tumor cures in MDA-MB-231 tumor-bearing animals by tail vein injection\(^4\).

**PROTOCOL**

**Cell Assay**\(^1\)

For the Alamar Blue proliferation assay, 2-4×10^3 cells are plated in 96-well plates. Later (48 h), cells are treated with Tanespimycin for 96 h or 0.01% DMSO as control. On day 4, Alamar Blue viability assay is performed as described elsewhere. IC\(_{50}\) and IC\(_{90}\) are calculated as the doses of Tanespimycin required to inhibit cell growth by 50 and 90%, respectively. Cell cycle distribution is assayed as described previously with a Becton Dickinson fluorescence-activated cell sorter and analyzed by the Cell Cycle Multicycle system.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

**Animal Administration**\(^1\)

Tanespimycin is dissolved in an EPL vehicle. To aid in the identification of an optimal dose and schedule, nontumor bearing mice are treated by i.p. injection with 25-200 mg/kg of Tanespimycin 5 days/week for 3 weeks or by the EPL vehicle alone. Serum samples are taken from each group, and equal volumes are pooled on days 5, 10, and 15 of treatment for serum chemistry and liver function analysis. At sacrifice, plasma samples are collected for complete blood count. A gross necropsy is performed on all of the mice, and a complete necropsy, including histopathology, is performed on 1 animal/group.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

**CUSTOMER VALIDATION**


See more customer validations on [www.MedChemExpress.com](http://www.MedChemExpress.com)

**REFERENCES**


