Verteporfin

Cat. No.: HY-B0146
CAS No.: 129497-78-5
Molecular Formula: C₈₂H₈₄N₈O₁₆
Molecular Weight: 718.79
Target: YAP; Autophagy; Apoptosis
Pathway: Stem Cell/Wnt; 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: 100 mg/mL (139.12 mM; Need ultrasonic)
DMF: 10 mg/mL (13.91 mM; ultrasonic and warming and heat to 60°C)

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent</th>
<th>Mass</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 mM</td>
<td>1.3912 mL</td>
<td>6.9561 mL</td>
<td>13.9123 mL</td>
<td></td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td>0.2782 mL</td>
<td>1.3912 mL</td>
<td>2.7825 mL</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td>0.1391 mL</td>
<td>0.6956 mL</td>
<td>1.3912 mL</td>
<td></td>
</tr>
</tbody>
</table>

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 5 mg/mL (6.96 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 5 mg/mL (6.96 mM); Clear solution

**BIOLOGICAL ACTIVITY**

Description
Verteporfin (CL 318952) is a photosensitizer for photodynamic therapy to eliminate the abnormal blood vessels in the eye associated with conditions such as age-related macular degeneration. Verteporfin is a YAP inhibitor which disrupts YAP-TEAD interactions. Verteporfin induces cell apoptosis[1]. Verteporfin is an autophagy inhibitor that blocks autophagy at an early stage by inhibiting autophagosome formation[3].

IC₅₀ & Target
IC50: YAP-TEAD interaction

In Vitro
Verteporfin is specifically selected by PDX-cell screening. The concentrations to cause 50% growth inhibition (GI₅₀) for PhLO, PhLH, and PhLK are 228 nM, 395 nM, and 538 nM, respectively, whereas GI₅₀ for ALL-1, TCC-Y/sr, and NPHeA1 are 3.93 µM, 2.11 µM, and 5.61 µM, respectively. GSH significantly reduces the sensitivity of 2 out of 3 PDX cells to verteporfin. Verteporfin
reduces the mitochondrial membrane potential in PDX cells[1]. Verteporfin reduces the PTX-resistance on HCT-8/T cells by inhibiting YAP expression and combination therapy with verteporfin and NSC 125973 shows synergism on inhibition of YAP and cytotoxicity to HCT-8/T[2].

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

### In Vivo

Verteporfin (10 mg/kg, c.s.c.) and BMS-354825 significantly reduces the leukemia cell ratio, and combined therapy further reduced the number of leukemia cells in the spleen[1].

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

### PROTOCOL

#### Cell Assay [1]

PDX cells co-cultured with S17 cells are treated with 16 combinations of verteporfin (60 nM, 120 nM, 180 nM, and 240 nM) and BMS-354825 (12 nM, 24 nM, 36 nM, and 48 nM). The viabilities of cells treated with each combination are measured after 48 h using FACS Aria flow cytometer. In order to estimate drug interaction between verteporfin and BMS-354825, a normalized isobologram and fraction affected combination index (CI) plot are made using CompuSyn software. CI values greater than 1.0 indicated antagonistic effects, equal to 1.0 additive effects, and below 1.0 synergistic effects.

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#### Animal Administration [1]

Mice: PhLO cells (1.0×10^7/mouse) are injected intravenously into 6-week-old male NOG mice, which are then treated with vehicle, verteporfin (140 mg/kg/day), BMS-354825 (20 mg/kg/day), and a combination of these drugs from days 22 to 28. Verteporfin is administered by continuous subcutaneous infusion (c.s.c.) using Alzet osmotic pumps. An intraperitoneal injection (i.p.) is performed for BMS-354825. All mice are sacrificed on day 28 and the chimerism of leukemia cells is investigated by flow cytometer using an anti-human CD19 antibody and antimouse CD45 antibody. Blood concentrations of verteporfin are calculated by LCMS-2020.

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### REFERENCES


