SZABO SCANDIC

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Produktinformation

- Forschungsprodukte & Biochemikalien
- Zellkultur & Verbrauchsmaterial
- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

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See the following pages for more information!

Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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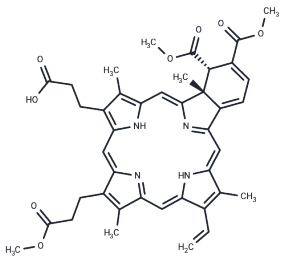
Data Sheet (Cat.No.T3112)

TargetMol

Verteporfin

Chemical Properties



CAS No. : 129497-78-5

Formula: C41H42N4O8

Molecular Weight: 718.79

Appearance: no data available

Storage: keep away from direct sunlight
Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description

Description	Verteporfin (BPD-MA) is a YAP inhibitor that inhibits YAP-TEAD interactions. Verteporfin is also a photosensitizer used in photodynamic therapy. Verteporfin also induces apoptosis and inhibits autophagy.
Targets(IC50)	Apoptosis,YAP,VDA,Autophagy
In vitro	Verteporfin metabolizes into a less active form within the body and is rapidly cleared, primarily excreted via feces and to a lesser extent through urine. Its therapy is effective and selectively prevents fluorescein dye leakage from choroidal neovascularization (CNV) induced in experimental monkeys. Verteporfin quickly accumulates in the choroidal vasculature, retinal pigment epithelium (RPE), and photoreceptors of established rabbit eyes. Upon intravenous injection in mice, peak tissue levels of Verteporfin are reached within 3 hours, followed by a rapid decline within 24 hours.
In vivo	Verteporfin forms a complex with LDL, which may be taken up by proliferating cells (e. g., neovascular endothelial cells) through LDL receptors or endocytosis. The therapy with Verteporfin achieves complete vascular occlusion through thrombosis in the vascular channels following selective endothelial damage. As shown by optical and electron microscopy, Verteporfin therapy selectively induces occlusion in regenerating and detached choroidal capillaries, without altering the overlying photoreceptors or ganglion cells. Verteporfin rapidly exhibits apoptotic changes in conjunction with light, as demonstrated by the activation of caspase-3 and caspase-9 and the cleavage of PARP in HL-60 cells, changes that are inhibited by the broad-spectrum caspase inhibitor ZVAD.fmk.
Kinase Assay	Cells (5 × 103) are plated in 96 well plates. Cells are treated the next day for 24 to 48 hours and then assessed for caspase-3 activity by Caspase-Glo-3/7 Assay, as per manufacturer's instructions and a luminescence plate reader.
Cell Research	Verteporfin is dissolved in DMSO. PDX cells co-cultured with S17 cells are treated with 16 combinations of verteporfin (60 nM, 120 nM, 180 nM, and 240 nM) and dasatinib (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 dasatinib, a normalized isobologram and fraction affectedcombination 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.

Solubility Information

Solubility	Ethanol: < 1 mg/mL (insoluble or slightly soluble), DMSO: 93 mg/mL (129.4 mM), H2O: < 1 mg/mL (insoluble or slightly soluble), (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	1.3912 mL	6.9561 mL	13.9123 mL
5 mM	0.2782 mL	1.3912 mL	2.7825 mL
10 mM	0.1391 mL	0.6956 mL	1.3912 mL
50 mM	0.0278 mL	0.1391 mL	0.2782 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.

Reference

Peng G, Suo S, Cui G, et al. Molecular architecture of lineage allocation and tissue organization in early mouse embryo. Nature. 2019, 572(7770): 528-532.