# Product data sheet

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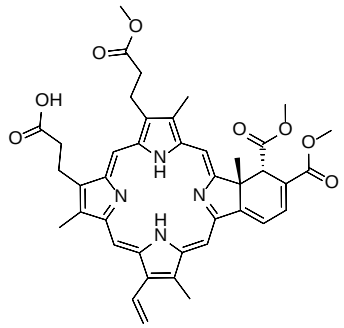
|---|---|
| MedKoo Cat#: 203120<br>Name: Verteporfin<br>CAS#: 129497-78-5<br>Chemical Formula: $C_{41}H_{42}N_4O_8$<br>Exact Mass: 718.3003<br>Molecular Weight: 718.79 |  |
| Product supplied as: | Powder |
| Purity (by HPLC): | ≥ 98% |
| Shipping conditions | Ambient temperature |
| Storage conditions: | Powder: -20°C 3 years; 4°C 2 years. |
| | In solvent: -80°C 3 months; -20°C 2 weeks. |

**1. Product description:**

Verteporfin, also known as Benzoporphyrin derivative monoacid ring A or BPD-MA, is a benzoporphyrin derivative and is a medication used as a photosensitizer for photodynamic therapy to eliminate the abnormal blood vessels in the eye associated with conditions such as the wet form of macular degeneration. Verteporfin accumulates in these abnormal blood vessels and, when stimulated by nonthermal red light with a wavelength of 693 nm in the presence of oxygen, produces highly reactive short-lived singlet oxygen and other reactive oxygen radicals, resulting in local damage to the endothelium and blockage of the vessels. Verteporfin is also used off-label for the treatment of central serous retinopathy.

**2. CoA, QC data, SDS, and handling instruction**

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under “QC And Documents” section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

**3. Solubility data**

| Solvent | Max Conc. mg/mL | Max Conc. mM |
|---|---|---|
| DMSO | 70.0 | 97.38 |
| DMF | 20.0 | 27.82 |
| Water | 0.01 | 0.014 |

**4. Stock solution preparation table:**

| Concentration / Solvent Volume / Mass | 1 mg | 5 mg | 10 mg |
|---|---|---|---|
| 1 mM | 1.39 mL | 6.96 mL | 13.91 mL |
| 5 mM | 0.28 mL | 1.39 mL | 2.78 mL |
| 10 mM | 0.14 mL | 0.70 mL | 1.39 mL |
| 50 mM | 0.03 mL | 0.14 mL | 0.28 mL |

**5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator**

Please refer the product web page under section of “Calculator”

**6. Recommended literature which reported protocols for in vitro and in vivo study**

In vitro study

1. Liang J, Wang L, Wang C, Shen J, Su B, Marisetty AL, Fang D, Kassab C, Jeong KJ, Zhao W, Lu Y, Jain AK, Zhou Z, Liang H, Sun SC, Lu C, Xu ZX, Yu Q, Shao S, Chen X, Gao M, Claret FX, Ding Z, Chen J, Chen P, Barton MC, Peng G, Mills GB, Heimberger AB. Verteporfin Inhibits PD-L1 through Autophagy and the STAT1-IRF1-TRIM28 Signaling Axis, Exerting Antitumor Efficacy. Cancer Immunol Res. 2020 Jul;8(7):952-965. doi: 10.1158/2326-6066.CIR-19-0159. Epub 2020 Apr 7. PMID: 32265228; PMCID: PMC8204534.
2. Wang Y, Wang L, Wise JTF, Shi X, Chen Z. Verteporfin inhibits lipopolysaccharide-induced inflammation by multiple functions in RAW 264.7 cells. Toxicol Appl Pharmacol. 2020 Jan 15;387:114852. doi: 10.1016/j.taap.2019.114852. Epub 2019 Dec 5. PMID: 31812773.

In vivo study

# Product data sheet

1. Liang J, Wang L, Wang C, Shen J, Su B, Marisetty AL, Fang D, Kassab C, Jeong KJ, Zhao W, Lu Y, Jain AK, Zhou Z, Liang H, Sun SC, Lu C, Xu ZX, Yu Q, Shao S, Chen X, Gao M, Claret FX, Ding Z, Chen J, Chen P, Barton MC, Peng G, Mills GB, Heimberger AB. Verteporfin Inhibits PD-L1 through Autophagy and the STAT1-IRF1-TRIM28 Signaling Axis, Exerting Antitumor Efficacy. Cancer Immunol Res. 2020 Jul;8(7):952-965. doi: 10.1158/2326-6066.CIR-19-0159. Epub 2020 Apr 7. PMID: 32265228; PMCID: PMC8204534.

**7. Bioactivity**

Biological target: Verteporfin (CL 318952, Visudyne) is a small molecule that inhibits TEAD–YAP association and YAP-induced liver overgrowth.

In vitro activity

Verteporfin suppressed PD-L1 expression effectively in all 6 cell lines (T cell leukemia; B cell leukemia; ovarian; endometrium n=3) (Fig. 1A, Supplemental Table 1, Supplemental Fig. S1). In an additional panel of 8 human cancer cell lines (ovarian, n=5; osteoblastoma, n=1; and lung cancers, n=2) and 2 murine cancer cell lines (ovarian and lung), verteporfin abolished basal PD-L1 protein expression, including differential glycosylated states as reflected by the double bands on Western Blots, regardless of genetic background, lineage specificity, and basal (intrinsic) PD-L1 levels (Fig. 1A-D). Cell fractionation revealed that verteporfin decreased membrane-associated PD-L1 (functionally relevant PD-L1) in EFE184 cells (endometrial cancer) (Fig. 1E) and flow cytometry showed that verteporfin reduced PD-L1 expression on both the surface of cancer cells (Fig. 1F) and on antigen presenting cells (Supplementary Fig. S1D). Verteporfin suppressed both IFN-induced PD-L1 protein expression (Supplemental Fig. S1B, C, D) and mRNA expression (Fig. 1G). However, in contrast to the marked loss of PD-L1 protein, verteporfin had little effect on intrinsic PD-L1 mRNA expression in the absence of IFN-γ (Fig. 1H). Thus, verteporfin engages at least two independent mechanisms to down-regulate PD-L1 expression.

Reference: Cancer Immunol Res. 2020 Jul;8(7):952-965. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8204534/

In vivo activity

The in vivo therapeutic effects of verteporfin were tested in established immune competent mice bearing ID8 cells intraperitoneally (Fig. 6B). Verteporfin had a modest effect on the survival of ID8-burdened mice. In contrast, the combination of verteporfin and BMN 673 produced a statistically significant improved outcome compared to either monotherapy that was equivalent to the combination of anti-PD-L1 and BMN 673 (Fig. 6B). In LLC (Lewis lung carcinoma) tumors for which tumors could be harvested for immune analysis, verteporfin treatment led to marked decreases in PD-L1 expression and increases in CD8 T cells especially in the combinatorial group of vereporfin and BMN 673 (Fig. 6E, D; Supplemental Fig. S6A, B).

Reference: Cancer Immunol Res. 2020 Jul;8(7):952-965. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8204534/