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Produktinformation

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- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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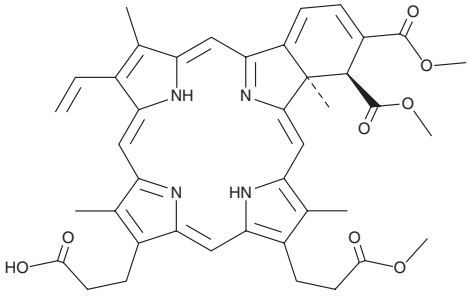
PRODUCT INFORMATION

Verteporfin

Item No. 17334

CAS Registry No.:	129497-78-5
Formal Name:	(4R,4aS)-*rel*-18-ethenyl-4,4a-dihydro-3,4-3,4-*bis*(methoxycarbonyl)-4a,8,14,19-tetramethyl-24H,26H-benzo[b]porphine-9,13-dipropanoic acid, monomethyl ester
Synonyms:	BPD-MA, CL 318,952
MF:	$C_{41}H_{42}N_4O_8$
FW:	718.8
Purity:	≥95%
UV/Vis.:	λ_{max}: 214, 345, 435, 566, 682 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥2 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Verteporfin is supplied as a crystalline solid. A stock solution may be made by dissolving the verteporfin in the solvent of choice. Verteporfin is soluble in organic solvents such as DMSO and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of verteporfin in these solvents is approximately 50 and 3 mg/ml, respectively.

Verteporfin is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, verteporfin should first be dissolved in DMF and then diluted with the aqueous buffer of choice. Verteporfin has a solubility of approximately 0.1 mg/ml in a 1:7 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Verteporfin is a photosensitizer used during photodynamic therapy to eliminate abnormal blood vessels in the eye that are associated with conditions such as macular degeneration. It accumulates in these abnormal blood vessels and, when activated by nonthermal red light with a wavelength of 693 nm in the presence of oxygen, produces a highly reactive short-lived singlet oxygen and other reactive oxygen radicals, generating local damage to the endothelium and vessel occlusion.[1] Verteporfin can also inhibit autophagosome formation by directly targeting and modifying p62, a scaffold and adaptor protein that binds both polyubiquitinated proteins destined for degradation and LC3 on autophagosomal membranes.[2]

References

1. Schmidt-Erfurth, U. and Hasan, T. Mechanisms of action of photodynamic therapy with verteporfin for the treatment of age-related macular degeneration. *Surv. Ophthalmol.* **45(3)**, 195-214 (2000).
2. Donohue, E., Balgi, A.D., Komatsu, M., *et al.* Induction of covalently crosslinked p62 oligomers with reduced binding to polyubiquitinated proteins by the autophagy inhibitor verteporfin. *PLoS One* **9(12)**, 1-30 (2014).

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