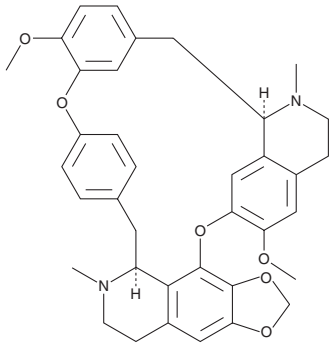
PRODUCT INFORMATION

Cepharanthine

Item No. 19648

CAS Registry No.:	481-49-2
Formal Name:	2,3,13,14,14aS,15,26,26aR-octahydro-22,30-dimethoxy-1,14-dimethyl-1H-4,6:16,19-dietheno-21,25-metheno-12H-[1,3]dioxolo[4,5-g]pyrido[2',3':17,18][1,10]dioxacycloeicosino[2,3,4-ij]isoquinoline
Synonyms:	O-Methylcepharanoline, NSC 623442
MF:	$C_{37}H_{38}N_2O_6$
FW:	606.7
Purity:	≥98%
UV/Vis.:	λ_{max}: 283 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years
Item Origin:	Plant/*Stephania japonica*



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

Laboratory Procedures

Cepharanthine is supplied as a crystalline solid. A stock solution may be made by dissolving the cepharanthine in the solvent of choice, which should be purged with an inert gas. Cepharanthine is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of cepharanthine in these solvents is approximately 2, 5, and 10 mg/ml, respectively.

Cepharanthine is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, cepharanthine should first be dissolved in DMF and then diluted with the aqueous buffer of choice. Cepharanthine has a solubility of approximately 0.25 mg/ml in a 1:3 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Cepharanthine is a biscoclaurine alkaloid extracted from *S. cepharantha* that is cationic and amphipathic and has been reported to decrease the fluidity of biological membranes.[1] It exhibits antiparasitic, antimalarial, antiviral, anti-inflammatory, antimetastatic, and anticancer activities in various cell and animal models.[1-3] Cepharanthine can decrease LPS-stimulated expression of TNF-α, IL-6, and IL-1β in macrophages and prevent the activation of NF-κB, ERK, JNK, and p38 MAPK.[4] It has also been shown to inhibit cell and tumor growth, inducing G_1 phase cell cycle arrest and apoptosis, and decreasing expression of STAT3, Bcl-xL, c-Myc, and cyclin D1 in osteosarcoma models.[5]

References

1. Matsuda, K., Hattori, S., Komizu, Y., *et al. Bioorg. Med. Chem. Lett.* **24(9)**, 2115-2117 (2014).
2. Desgrouas, C., Chapus, C., Desplans, J., *et al. Malar. J.* **13:327**, (2014).
3. Rogosnitzky, M. and Danks, R. *Pharmacol. Rep.* **63(2)**, 337-347 (2011).
4. Huang, H., Hu, G., Wang, C., *et al. Inflammation* **37(1)**, 235-246 (2014).
5. Chen, Z., Huang, C., Yang, Y., *et al. Acta Pharmacol. Sin.* **33(1)**, 101-108 (2012).

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