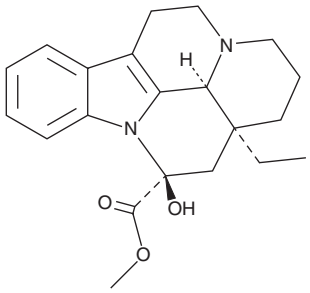
PRODUCT INFORMATION

Vincamine

Item No. 11763

CAS Registry No.:	1617-90-9
Formal Name:	(3α,14β,16α)-14,15-dihydro-14-hydroxy-eburnamenine-14-carboxylic acid, methyl ester
Synonyms:	Angiopac, Devincan, Equipur, Minorin, NSC 91998, Novicet, Oxybral, Perval, Sostenil, Tripervan
MF:	$C_{21}H_{26}N_2O_3$
FW:	354.4
Purity:	≥98%
UV/Vis.:	λ_{max}: 227, 281 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



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

Laboratory Procedures

Vincamine is supplied as a crystalline solid. A stock solution may be made by dissolving the vincamine in the solvent of choice. Vincamine is soluble in dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of vincamine in DMF is approximately 0.25 mg/ml.

Vincamine is sparingly soluble in aqueous solutions. To enhance aqueous solubility, dilute the organic solvent solution into aqueous buffers or isotonic saline. If performing biological experiments, ensure the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. We do not recommend storing the aqueous solution for more than one day.

Description

Vincamine is an indole alkaloid found in the leaves of *V. minor* and *C. roseus* that is used as a peripheral vasodilator to increase blood flow to the brain.[1] Vincamine contracts excised human cerebrovascular smooth muscle *in vitro* with an EC_{50} value of 30 µM and has been explored as a pharmacotherapy to treat cerebral metabolic and vascular diseases.[2,3]

References

1. Lim, C.C. and James, I.M. The effect of an acute infusion of vincamine and ethyl apovincaminate on cerebral blood flow in healthy volunteers. *Br. J. Clin. Pharmacol.* **9(1)**, 100-101 (1980).
2. Young, A.R., Bouloy, M., Boussard, J.-F., *et al.* Direct vascular effects of agents used in the pharmacotherapy of cerebrovascular disease on isolated cerebral vessels. *J. Cereb. Blood Flow Metab.* **1(1)**, 117-128 (1981).
3. Nowicki, J.-P., MacKenzie, E.T., and Spinnewyn, B. Effects of agents used in the pharmacotherapy of cerebrovascular disease on the oxygen consumption of isolated cerebral mitochondria. *J. Cereb. Blood Flow Metab.* **2(1)**, 33-40 (1982).

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