

RESEARCH MONOGRAPH . COMPOUND PROFILE

Vinpocetine

A synthetic alkaloid derivative of vincamine from Vinca minor; a cerebrovascular agent and PDE1 inhibitor used as a nootropic.

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ABSTRACT

Vinpocetine ((3- α ,16- α)-eburnamenine-14-carboxylic acid ethyl ester; CAS 42971-09-5; molecular formula C₂₂H₂₆N₂O₂; molecular weight 350.46) is a synthetic alkaloid derivative of vincamine from *Vinca minor* (lesser periwinkle), developed at Gedeon Richter (Hungary) in the 1960s and approved in many European countries as Cavinton for cerebrovascular insufficiency. Mechanism is multifactorial: cerebral vasodilation through smooth muscle relaxation, weak phosphodiesterase 1 (PDE1) inhibition, sodium channel block in neurons, and modest antioxidant activity. The combined effect produces improved cerebral blood flow and reduced ischemic neuronal injury in animal stroke models. Plasma half-life is approximately 1 to 2 hours; metabolism is hepatic via CYP2C9 and CYP3A4. Approved indications in Europe include cerebrovascular disorders, age-related cognitive decline, and tinnitus. In the US, the compound is sold as a dietary supplement with FDA reservations about that designation given its drug-like history. Used as a cerebrovascular nootropic and PDE1 inhibitor in research.

1. 1. Compound Identification

Vinpocetine is described as synthetic vincamine derivative / cerebrovascular agent. A synthetic alkaloid derivative of vincamine from Vinca minor; a cerebrovascular agent and PDE1 inhibitor used as a nootropic.

1.1. 1.1 Class summary

Synthetic vincamine derivative / cerebrovascular agent

1.2. 1.2 Subtitle

A synthetic alkaloid derivative of vincamine from Vinca minor; a cerebrovascular agent and PDE1 inhibitor used as a nootropic.

2. 2. Abstract

Vinpocetine ((3- α ,16- α)-eburnamenine-14-carboxylic acid ethyl ester; CAS 42971-09-5; molecular formula C₂₂H₂₆N₂O₂; molecular weight 350.46) is a synthetic alkaloid derivative of vincamine from *Vinca minor* (lesser periwinkle), developed at Gedeon Richter (Hungary) in the 1960s and approved in many European countries as Cavinton for cerebrovascular insufficiency. Mechanism is multifactorial: cerebral vasodilation through smooth muscle relaxation, weak phosphodiesterase 1 (PDE1) inhibition, sodium channel block in neurons, and modest antioxidant activity. The combined effect produces improved cerebral blood flow and reduced ischemic neuronal injury in animal stroke models. Plasma half-life is approximately 1 to 2 hours; metabolism is hepatic via CYP2C9 and CYP3A4. Approved indications in Europe include cerebrovascular disorders, age-related cognitive decline, and tinnitus. In the US, the compound is sold as a dietary supplement with FDA reservations about that designation given its drug-like history. Used as a cerebrovascular nootropic and PDE1 inhibitor in research.

3. Mechanism of Action

Cerebral vasodilation; weak PDE1 inhibition; sodium channel block; modest antioxidant activity. Improves cerebral blood flow.

The mechanistic profile described above represents the consensus position from peer-reviewed literature where available; for compounds with sparse formal characterization, this section reflects vendor-grade reports and structurally-related class pharmacology and should be interpreted with appropriate caution.

4. 4. Pharmacokinetics and Routes of Administration

Pharmacokinetic data are summarized within the abstract for compounds with formal Phase 1 records. Compounds without published human pharmacokinetic studies are noted as such; investigators should treat the absence of data as a research limitation.

5. 5. Reported Research Dose Ranges

Clinical 5 to 10 mg per oral administration three times daily. Rodent studies 1 to 10 mg/kg.

Doses are scaled from clinical use where the compound is approved as a medicine, from Phase 2/3 trials where development advanced, or from rodent dose-response data scaled by allometric assumptions where the compound has no human clinical record. Investigators should treat dose ranges from research-chemical-grade vendors as starting points for their own pharmacokinetic and tolerability characterization rather than validated dosing recommendations.

6. Sourcing and Quality Verification

Investigators should obtain analytical confirmation of identity (mass spectrometry; for peptides, additionally amino acid analysis or N-terminal sequencing) and purity (HPLC area-percent, ideally greater than 95 percent) on every lot. Vendor certificates of analysis vary in quality; third-party analytical confirmation through independent laboratories (e.g., Janoshik Analytical, Anabolic Lab) is the gold standard for research-grade peptides and small molecules.

For compounds with established pharmaceutical-grade marketing in any jurisdiction, sourcing from the marketed product (where legal and accessible) provides the highest quality assurance.

7. 7. Reconstitution and Handling

Peptides should be reconstituted in bacteriostatic water or sterile water for injection in the volume appropriate to the desired concentration; lyophilized peptides should be allowed to come to room temperature before reconstitution to minimize precipitation. Reconstituted peptides should be stored refrigerated and used within the timeframe specified by the manufacturer (typically 14 to 30 days for bacteriostatic water reconstitutions, shorter for sterile water).

Small molecules: storage conditions vary by compound; consult the certificate of analysis for the specific product. Most are stable at room temperature in dry, dark conditions; oxidation-sensitive compounds (e.g., catecholamine precursors) require cool storage and protection from light.

8. 8. Stack Interaction Considerations

Investigators combining this compound with other research substances should consider receptor-level interactions (additive effects at shared targets), enzyme-level interactions (CYP-mediated drug-drug interactions where applicable), and pharmacodynamic interactions (additive sedation, additive serotonergic activity, additive hemodynamic effects).

The stack interaction section in published monographs of well-characterized compounds is necessarily incomplete for any specific combination; investigators should treat novel combinations with elevated caution and titrate gradually.

9. 9. References

1. Bonoczk P, et al. Role of sodium channel inhibition in neuroprotection: effect of vinpocetine. Brain Res Bull 2000.
2. Patyar S, et al. Role of vinpocetine in cerebrovascular diseases. Pharmacol Rep 2011.
3. Szilagyi G, et al. Effects of vinpocetine on the redistribution of cerebral blood flow and glucose metabolism in chronic ischemic stroke. J Neurol Sci 2005.

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