

RESEARCH MONOGRAPH . COMPOUND PROFILE

Harmine

A beta-carboline alkaloid from Peganum harmala; a reversible MAO-A inhibitor and DYRK1A kinase inhibitor of interest in beta-cell biology.

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COMPOUND CLASS	Beta-carboline reversible MAO-A inhibitor (DYRK1A inhibitor)

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ABSTRACT

Harmin (7-methoxy-1-methyl-9H-pyrido[3,4-b]indole; CAS 442-51-3; molecular formula C₁₃H₁₂N₂O; molecular weight 212.25) is a beta-carboline alkaloid from *Peganum harmala* and *Banisteriopsis caapi*. The compound is a reversible MAO-A inhibitor with similar selectivity profile to harmaline but with the additional well-characterized activity of DYRK1A kinase inhibition (IC₅₀ approximately 30 nM). The DYRK1A activity has generated substantial interest in diabetes research: harmine and related DYRK1A inhibitors stimulate human pancreatic beta-cell proliferation in vitro and in vivo, a long-sought target for diabetes regenerative therapy. Phase 1 trials of derivatives are underway. Plasma half-life is approximately 1 to 3 hours. Used as a research probe for both ayahuasca pharmacology and beta-cell regenerative biology.

1. Compound Identification

Harmine is described as beta-carboline reversible mao-a inhibitor (dyrk1a inhibitor). A beta-carboline alkaloid from Peganum harmala; a reversible MAO-A inhibitor and DYRK1A kinase inhibitor of interest in beta-cell biology.

1.1 Class summary

Beta-carboline reversible MAO-A inhibitor (DYRK1A inhibitor)

1.2 Subtitle

A beta-carboline alkaloid from Peganum harmala; a reversible MAO-A inhibitor and DYRK1A kinase inhibitor of interest in beta-cell biology.

2. Abstract

Harmine (7-methoxy-1-methyl-9H-pyrido[3,4-b]indole; CAS 442-51-3; molecular formula $C_{13}H_{12}N_2O$; molecular weight 212.25) is a beta-carboline alkaloid from *Peganum harmala* and *Banisteriopsis caapi*. The compound is a reversible MAO-A inhibitor with similar selectivity profile to harmaline but with the additional well-characterized activity of DYRK1A kinase inhibition (IC₅₀ approximately 30 nM). The DYRK1A activity has generated substantial interest in diabetes research: harmine and related DYRK1A inhibitors stimulate human pancreatic beta-cell proliferation in vitro and in vivo, a long-sought target for diabetes regenerative therapy. Phase 1 trials of derivatives are underway. Plasma half-life is approximately 1 to 3 hours. Used as a research probe for both ayahuasca pharmacology and beta-cell regenerative biology.

3. Mechanism of Action

Reversible MAO-A inhibitor; DYRK1A kinase inhibitor (IC₅₀ ~30 nM). DYRK1A activity stimulates pancreatic beta-cell proliferation.

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. 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. Reported Research Dose Ranges

Research 100 to 300 mg per oral administration; rodent diabetes studies 10 to 30 mg/kg/day.

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. 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. 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. References

1. Wang P, et al. A high-throughput chemical screen reveals that harmine-mediated inhibition of DYRK1A increases human pancreatic beta cell replication. Nat Med 2015.
2. Brierley DI, Davidson C. Developments in harmine pharmacology - implications for ayahuasca use and drug-dependence treatment. Prog Neuropsychopharmacol Biol Psychiatry 2012.
3. Frost D, et al. beta-carboline compounds, including harmine, inhibit DYRK1A and tau phosphorylation. PLoS ONE 2011.

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