

RESEARCH MONOGRAPH . COMPOUND PROFILE

N,N-Dimethyltryptamine (N,N-DMT)

An endogenous tryptamine; the principal psychoactive constituent of ayahuasca; a high-affinity 5-HT_{2A} receptor agonist.

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COMPOUND CLASS	Endogenous tryptamine / 5-HT _{2A} agonist

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ABSTRACT

N,N-Dimethyltryptamine (N,N-DMT; CAS 61-50-7; molecular formula $C_{12}H_{16}N_2$; molecular weight 188.27) is an endogenous and exogenous tryptamine alkaloid identified in many plants (notably *Mimosa hostilis* and *Psychotria viridis*) and at trace concentrations in mammalian tissue. The compound is the principal psychoactive constituent of ayahuasca brews, where harmala beta-carbolines provide MAO-A inhibition that allows oral DMT activity. Pharmacologically, DMT is a high-affinity full agonist at 5-HT_{2A} (K_i approximately 75 nM), 5-HT_{1A}, and 5-HT_{2C} receptors with secondary activity at sigma-1, TAAR1, and other targets. Plasma half-life is extremely short (approximately 15 minutes) owing to rapid MAO-A degradation; oral administration without MAO inhibition is essentially inactive. Behavioral effects include intense visual imagery, altered sense of time, and (at higher doses) sense of communicating with autonomous entities. Approximately 30 active research and clinical trials of DMT and analogs are underway as of 2024 for depression and other psychiatric indications. Schedule I in the US under the CSA. Used as the canonical classical psychedelic in academic neuroscience.

1. Compound Identification

N,N-Dimethyltryptamine (N,N-DMT) is described as endogenous tryptamine / 5-HT_{2A} agonist. An endogenous tryptamine; the principal psychoactive constituent of ayahuasca; a high-affinity 5-HT_{2A} receptor agonist.

1.1 Class summary

Endogenous tryptamine / 5-HT_{2A} agonist

1.2 Subtitle

An endogenous tryptamine; the principal psychoactive constituent of ayahuasca; a high-affinity 5-HT_{2A} receptor agonist.

2. Abstract

N,N-Dimethyltryptamine (N,N-DMT; CAS 61-50-7; molecular formula C₁₂H₁₆N₂; molecular weight 188.27) is an endogenous and exogenous tryptamine alkaloid identified in many plants (notably *Mimosa hostilis* and *Psychotria viridis*) and at trace concentrations in mammalian tissue. The compound is the principal psychoactive constituent of ayahuasca brews, where harmala beta-carbolines provide MAO-A inhibition that allows oral DMT activity. Pharmacologically, DMT is a high-affinity full agonist at 5-HT_{2A} (K_i approximately 75 nM), 5-HT_{1A}, and 5-HT_{2C} receptors with secondary activity at sigma-1, TAAR1, and other targets. Plasma half-life is extremely short (approximately 15 minutes) owing to rapid MAO-A degradation; oral administration without MAO inhibition is essentially inactive. Behavioral effects include intense visual imagery, altered sense of time, and (at higher doses) sense of communicating with autonomous entities. Approximately 30 active research and clinical trials of DMT and analogs are underway as of 2024 for depression and other psychiatric indications. Schedule I in the US under the CSA. Used as the canonical classical psychedelic in academic neuroscience.

3. Mechanism of Action

High-affinity 5-HT_{2A}, 5-HT_{1A}, 5-HT_{2C} agonist; sigma-1 and TAAR1 secondary activity. Endogenous tryptamine; rapid MAO-A degradation requires MAO inhibition for oral activity.

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

Smoked/vaporized 20 to 60 mg; intravenous 0.05 to 0.4 mg/kg in research; oral with MAO inhibitor 30 to 70 mg.

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. Strassman R. DMT: The Spirit Molecule. 2001.
2. Carbonaro TM, Gatch MB. Neuropharmacology of N,N-dimethyltryptamine. Brain Res Bull 2016.
3. D'Souza DC, et al. Exploratory study of the dose-related safety, tolerability, and efficacy of dimethyltryptamine (DMT) in healthy volunteers and major depressive disorder. Neuropsychopharmacology 2022.

This monograph is provided for research use only. Not for medical, diagnostic, or therapeutic purposes. Not for human consumption. Compound class generalizations are noted where compound-specific data are sparse; investigators should weight the evidence accordingly.