

RESEARCH MONOGRAPH . COMPOUND PROFILE

Ladasten

An N-substituted adamantyl bromophenylamine related to bromantane with longer plasma duration, registered in the Russian Federation as Ladasten for asthenic conditions.

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AUTHOR	Kodiac biolabs Research
COMPOUND CLASS	Adamantane-derived actoprotectant and atypical anxiolytic

ABSTRACT

Ladasten (bromantane analog; N-(2-adamantyl)-N-(4-bromophenyl)amine derivative; INN bromantane in some sources but distinguished by the longer-acting Russian preparation; CAS for bromantane parent 40165-90-2) is an adamantane-class atypical psychostimulant and actoprotectant developed at the Russian Academy of Sciences and registered by the Russian Ministry of Health in the early 2000s for the treatment of asthenia and idiopathic chronic fatigue. Ladasten is closely related to bromantane and shares the dual immunomodulatory and dopaminergic profile of that parent compound, but Russian-language pharmacology reports describe a longer plasma half-life and a more pronounced antiasthenic effect at lower mass doses (50 to 100 mg daily versus 100 to 200 mg for bromantane). Mechanism is multifactorial and incompletely characterized in Western pharmacology: principal effects include induction of tyrosine hydroxylase and aromatic L-amino acid decarboxylase transcripts in mesolimbic dopaminergic neurons (driving sustained dopamine biosynthesis rather than release-mediated stimulation), modulation of GABA-A and serotonergic tone, and immunorestorative activity in stress models. The behavioral signature is anxiolysis without sedation combined with restoration of physical and cognitive performance under fatigue conditions, distinguishing the compound from classical psychostimulants and from benzodiazepine anxiolytics. Western clinical trial data are absent. Published Russian Phase 3 data describe efficacy in neurasthenia and post-infectious asthenia at 50 to 100 mg per day for 28 days. The compound is not approved by FDA, EMA, or any regulatory authority outside the Russian

Federation and Eastern European member states; the published evidence base is dominated by a single research-clinical group, which is the principal limitation. Adverse event profile is benign at registered doses, with mild gastrointestinal upset and transient activation reported.

1. Compound Identification

Ladasten is described as adamantane-derived actoprotectant and atypical anxiolytic. An N-substituted adamantyl bromophenylamine related to bromantane with longer plasma duration, registered in the Russian Federation as Ladasten for asthenic conditions.

1.1 Class summary

Adamantane-derived actoprotectant and atypical anxiolytic

1.2 Subtitle

An N-substituted adamantyl bromophenylamine related to bromantane with longer plasma duration, registered in the Russian Federation as Ladasten for asthenic conditions.

2. Abstract

Ladasten (bromantane analog; N-(2-adamantyl)-N-(4-bromophenyl)amine derivative; INN bromantane in some sources but distinguished by the longer-acting Russian preparation; CAS for bromantane parent 40165-90-2) is an adamantane-class atypical psychostimulant and actoprotectant developed at the Russian Academy of Sciences and registered by the Russian Ministry of Health in the early 2000s for the treatment of asthenia and idiopathic chronic fatigue. Ladasten is closely related to bromantane and shares the dual immunomodulatory and dopaminergic profile of that parent compound, but Russian-language pharmacology reports describe a longer plasma half-life and a more pronounced antiasthenic effect at lower mass doses (50 to 100 mg daily versus 100 to 200 mg for bromantane). Mechanism is multifactorial and incompletely characterized in Western pharmacology: principal effects include induction of tyrosine hydroxylase and aromatic L-amino acid decarboxylase transcripts in mesolimbic dopaminergic neurons (driving sustained dopamine biosynthesis rather than release-mediated stimulation), modulation of GABA-A and serotonergic tone, and immunorestorative activity in stress models. The behavioral signature is anxiolysis without sedation combined with restoration of physical and cognitive performance under fatigue conditions, distinguishing the compound from classical psychostimulants and from benzodiazepine anxiolytics. Western clinical trial data are absent. Published Russian Phase 3 data describe efficacy in neurasthenia and post-infectious asthenia at 50 to 100 mg per day for 28 days. The compound is not approved by FDA, EMA, or any regulatory authority outside the Russian Federation and Eastern European member states; the published evidence base is dominated by a single research-clinical group, which is the principal limitation. Adverse event profile is benign at registered doses, with mild gastrointestinal upset and transient activation reported.

3. Mechanism of Action

Tyrosine hydroxylase and AADC transcriptional upregulation produces sustained dopamine biosynthesis. Secondary GABA-A and serotonergic modulation. Immunorestorative activity in stress models.

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

Russian clinical use 50 to 100 mg per oral administration once daily for 28 days. Research literature reports up to 150 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. Mikhaylova MA, et al. Antiasthenic activity of Ladasten. Bull Exp Biol Med 2010.
2. Vakhitova YV, et al. Bromantane upregulates dopamine biosynthesis in mesolimbic neurons. Neurochem Res 2005.
3. Yarkova MA, et al. Pharmacology of N-substituted adamantanes. Pharm Chem J 2018.

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