

RESEARCH MONOGRAPH . COMPOUND PROFILE

Sodium Oxybate (GHB)

Sodium gamma-hydroxybutyrate; an endogenous neurotransmitter and prescription medication for narcolepsy and (controversially) alcoholism.

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ABSTRACT

Sodium oxybate (sodium 4-hydroxybutanoate, GHB; CAS 502-85-2; molecular formula $C_4H_7NaO_3$; molecular weight 126.09) is the sodium salt of gamma-hydroxybutyrate, an endogenous short-chain fatty acid neurotransmitter and metabolite of GABA. The compound binds two distinct receptors: high-affinity GHB receptors (K_d approximately 50 nM, low capacity) and low-affinity GABA-B receptors (K_i approximately 5 mM, high capacity); pharmacologically meaningful effects at clinical doses are predominantly GABA-B-mediated. Approved indications: narcolepsy with cataplexy and excessive daytime sleepiness; clinical effect involves consolidation of slow-wave sleep. Used in some European countries (notably Italy, France) for alcohol withdrawal and dependence treatment. Schedule III when prescribed as Xyrem; Schedule I as illicit GHB. The narrow therapeutic index makes overdose easy and dangerous; recreational misuse with predatory drug-facilitated assault has been a significant public health issue. Plasma half-life is approximately 0.5 to 1 hour. Used as the canonical GHB receptor and GABA-B partial agonist in academic neuroscience.

1. Compound Identification

Sodium Oxybate (GHB) is described as ghb receptor agonist / gaba-b partial agonist. Sodium gamma-hydroxybutyrate; an endogenous neurotransmitter and prescription medication for narcolepsy and (controversially) alcoholism.

1.1 Class summary

GHB receptor agonist / GABA-B partial agonist

1.2 Subtitle

Sodium gamma-hydroxybutyrate; an endogenous neurotransmitter and prescription medication for narcolepsy and (controversially) alcoholism.

2. Abstract

Sodium oxybate (sodium 4-hydroxybutanoate, GHB; CAS 502-85-2; molecular formula $C_4H_7NaO_3$; molecular weight 126.09) is the sodium salt of gamma-hydroxybutyrate, an endogenous short-chain fatty acid neurotransmitter and metabolite of GABA. The compound binds two distinct receptors: high-affinity GHB receptors (K_d approximately 50 nM, low capacity) and low-affinity GABA-B receptors (K_i approximately 5 mM, high capacity); pharmacologically meaningful effects at clinical doses are predominantly GABA-B-mediated. Approved indications: narcolepsy with cataplexy and excessive daytime sleepiness; clinical effect involves consolidation of slow-wave sleep. Used in some European countries (notably Italy, France) for alcohol withdrawal and dependence treatment. Schedule III when prescribed as Xyrem; Schedule I as illicit GHB. The narrow therapeutic index makes overdose easy and dangerous; recreational misuse with predatory drug-facilitated assault has been a significant public health issue. Plasma half-life is approximately 0.5 to 1 hour. Used as the canonical GHB receptor and GABA-B partial agonist in academic neuroscience.

3. Mechanism of Action

Endogenous short-chain fatty acid neurotransmitter; high-affinity GHB receptor and low-affinity GABA-B receptor agonism. Consolidates slow-wave sleep.

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

Clinical 4.5 to 9 g per oral administration nightly, in divided doses.

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. Snead OC 3rd, Gibson KM. gamma-Hydroxybutyric acid. N Engl J Med 2005.
2. Mamelak M. Narcolepsy and depression and the neurobiology of gammahydroxybutyrate. Prog Neurobiol 2009.
3. Caputo F, et al. Pharmacological treatment of alcohol use disorder: an update. Eur Neuropsychopharmacol 2018.

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