

RESEARCH MONOGRAPH . COMPOUND PROFILE

Dexmedetomidine

A potent selective alpha-2A adrenergic agonist; an intravenous sedative producing arousable sedation without respiratory depression.

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ABSTRACT

Dexmedetomidine ((S)-4-[1-(2,3-dimethylphenyl)ethyl]-1H-imidazole; CAS 113775-47-6; molecular formula C₁₃H₁₆N₂; molecular weight 200.28) is a highly selective alpha-2A adrenergic agonist developed at Farnam and approved by the FDA in 1999 (Precedex). The compound is the active S-enantiomer of medetomidine (used in veterinary anesthesia). Alpha-2A selectivity over alpha-2B and alpha-2C is approximately 8-fold; selectivity over alpha-1 is approximately 1620-fold, much higher than clonidine. The clinical profile is unique: the alpha-2A activation in the locus coeruleus produces sedation that mimics natural sleep architecture, with patients arousable on stimulation; respiratory drive is preserved. Approved indications: ICU sedation, procedural sedation, conscious sedation. Plasma half-life is approximately 2 hours; metabolism is hepatic. Used as the canonical highly selective alpha-2A agonist in anesthesia research.

1. Compound Identification

Dexmedetomidine is described as selective alpha-2a adrenergic agonist (anesthetic). A potent selective alpha-2A adrenergic agonist; an intravenous sedative producing arousable sedation without respiratory depression.

1.1 Class summary

Selective alpha-2A adrenergic agonist (anesthetic)

1.2 Subtitle

A potent selective alpha-2A adrenergic agonist; an intravenous sedative producing arousable sedation without respiratory depression.

2. Abstract

Dexmedetomidine ((S)-4-[1-(2,3-dimethylphenyl)ethyl]-1H-imidazole; CAS 113775-47-6; molecular formula C₁₃H₁₆N₂; molecular weight 200.28) is a highly selective alpha-2A adrenergic agonist developed at Farnos and approved by the FDA in 1999 (Precedex). The compound is the active S-enantiomer of medetomidine (used in veterinary anesthesia). Alpha-2A selectivity over alpha-2B and alpha-2C is approximately 8-fold; selectivity over alpha-1 is approximately 1620-fold, much higher than clonidine. The clinical profile is unique: the alpha-2A activation in the locus coeruleus produces sedation that mimics natural sleep architecture, with patients arousable on stimulation; respiratory drive is preserved. Approved indications: ICU sedation, procedural sedation, conscious sedation. Plasma half-life is approximately 2 hours; metabolism is hepatic. Used as the canonical highly selective alpha-2A agonist in anesthesia research.

3. Mechanism of Action

Highly selective alpha-2A adrenergic agonist (1620-fold over alpha-1, 8-fold over alpha-2B/C). Arousable sedation without respiratory depression via locus coeruleus alpha-2A activation.

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 0.2 to 1.4 mcg/kg/hour intravenous infusion.

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. Riker RR, et al. Dexmedetomidine vs midazolam for sedation of critically ill patients. JAMA 2009.
2. Mantz J, et al. Dexmedetomidine: new insights. Eur J Anaesthesiol 2011.
3. Nelson LE, et al. The alpha-2 adrenoceptor agonist dexmedetomidine converges on an endogenous sleep-promoting pathway to exert its sedative effects. Anesthesiology 2003.

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.