

RESEARCH MONOGRAPH . COMPOUND PROFILE

Pramlintide

A synthetic analog of the pancreatic peptide hormone amylin; used adjunctively with insulin in type 1 and type 2 diabetes for postprandial glucose control.

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AUTHOR	Kodiac biolabs Research
COMPOUND CLASS	Synthetic amylin analog

ABSTRACT

Pramlintide (modified 37-amino-acid amide derived from amylin; CAS 151126-32-8; molecular weight 3949.4) is a synthetic analog of amylin (islet amyloid polypeptide), a pancreatic beta-cell peptide co-secreted with insulin. The native amylin is amyloidogenic; pramlintide substitutes proline for residues at positions 25, 28, and 29 to disrupt amyloid fibril formation while preserving receptor binding. Approved by the FDA in 2005 under the trade name Symlin. Mechanism: amylin receptor agonism produces three principal effects: slowed gastric emptying (delays glucose absorption), suppression of postprandial glucagon (reduces hepatic glucose output), and increased satiety (promotes voluntary caloric reduction). Approved as adjunct to mealtime insulin in type 1 and insulin-using type 2 diabetes; produces approximately 0.4 percent reduction in HbA1c and modest weight loss. Plasma half-life is approximately 50 minutes; subcutaneous administration before each major meal. Used as the canonical amylin analog in metabolic research.

1. Compound Identification

Pramlintide is described as synthetic amylin analog. A synthetic analog of the pancreatic peptide hormone amylin; used adjunctively with insulin in type 1 and type 2 diabetes for postprandial glucose control.

1.1 Class summary

Synthetic amylin analog

1.2 Subtitle

A synthetic analog of the pancreatic peptide hormone amylin; used adjunctively with insulin in type 1 and type 2 diabetes for postprandial glucose control.

2. Abstract

Pramlintide (modified 37-amino-acid amide derived from amylin; CAS 151126-32-8; molecular weight 3949.4) is a synthetic analog of amylin (islet amyloid polypeptide), a pancreatic beta-cell peptide co-secreted with insulin. The native amylin is amyloidogenic; pramlintide substitutes proline for residues at positions 25, 28, and 29 to disrupt amyloid fibril formation while preserving receptor binding. Approved by the FDA in 2005 under the trade name Symlin. Mechanism: amylin receptor agonism produces three principal effects: slowed gastric emptying (delays glucose absorption), suppression of postprandial glucagon (reduces hepatic glucose output), and increased satiety (promotes voluntary caloric reduction). Approved as adjunct to mealtime insulin in type 1 and insulin-using type 2 diabetes; produces approximately 0.4 percent reduction in HbA1c and modest weight loss. Plasma half-life is approximately 50 minutes; subcutaneous administration before each major meal. Used as the canonical amylin analog in metabolic research.

3. Mechanism of Action

Amylin receptor agonist (calcitonin/CGRP-related receptor family); slows gastric emptying, suppresses postprandial glucagon, increases satiety.

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 15 to 120 mcg subcutaneous before each major meal. Rodent studies 1 to 100 mcg/kg.

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. Riddle MC, et al. Randomized comparison of pramlintide or mealtime insulin added to basal insulin treatment for patients with type 2 diabetes. Diabetes Care 2009.
2. Edelman SV, et al. Pramlintide as an adjunct treatment for type 2 diabetes. Endocr Pract 2007.
3. Younk LM, et al. Pramlintide and the treatment of diabetes: a review of the data since its introduction. Expert Opin Pharmacother 2011.

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.