

RESEARCH MONOGRAPH . COMPOUND PROFILE

Teriparatide

*Recombinant parathyroid hormone
fragment 1-34; the first true anabolic agent
for osteoporosis.*

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ABSTRACT

Teriparatide (parathyroid hormone (1-34) recombinant; CAS 52232-67-4; 34-amino-acid peptide; molecular weight approximately 4117 Da) is a recombinant fragment of human parathyroid hormone (the biologically active N-terminal portion), approved by the FDA in 2002 (Forteo). Mechanism is paradoxical: chronic continuous high PTH (as in hyperparathyroidism) drives net bone resorption via osteoclast activation, but pulsatile once-daily PTH 1-34 administration drives net bone formation via direct osteoblast activation and reduced osteoblast apoptosis. The mechanism explanation involves PTH receptor-G protein signaling kinetics that favor osteoblast over osteoclast effects under intermittent stimulation. Plasma half-life is approximately 1 hour. Approved for severe osteoporosis with high fracture risk. The 2-year cumulative-dose limitation reflects an osteosarcoma signal observed in rat carcinogenicity studies (subsequently not reproduced in humans). Used as the canonical anabolic bone agent in osteoporosis research.

1. 1. Compound Identification

Teriparatide is described as recombinant pth 1-34 anabolic bone agent. Recombinant parathyroid hormone fragment 1-34; the first true anabolic agent for osteoporosis.

1.1. 1.1 Class summary

Recombinant PTH 1-34 anabolic bone agent

1.2. 1.2 Subtitle

Recombinant parathyroid hormone fragment 1-34; the first true anabolic agent for osteoporosis.

2. 2. Abstract

Teriparatide (parathyroid hormone (1-34) recombinant; CAS 52232-67-4; 34-amino-acid peptide; molecular weight approximately 4117 Da) is a recombinant fragment of human parathyroid hormone (the biologically active N-terminal portion), approved by the FDA in 2002 (Forteo). Mechanism is paradoxical: chronic continuous high PTH (as in hyperparathyroidism) drives net bone resorption via osteoclast activation, but pulsatile once-daily PTH 1-34 administration drives net bone formation via direct osteoblast activation and reduced osteoblast apoptosis. The mechanism explanation involves PTH receptor-G protein signaling kinetics that favor osteoblast over osteoclast effects under intermittent stimulation. Plasma half-life is approximately 1 hour. Approved for severe osteoporosis with high fracture risk. The 2-year cumulative-dose limitation reflects an osteosarcoma signal observed in rat carcinogenicity studies (subsequently not reproduced in humans). Used as the canonical anabolic bone agent in osteoporosis research.

3. Mechanism of Action

Recombinant PTH 1-34; intermittent once-daily administration drives osteoblast activation (anabolic effect) rather than the resorptive effect of chronic PTH excess.

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. 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. 5. Reported Research Dose Ranges

Clinical 20 mcg subcutaneous daily for up to 2 years.

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. 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. 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. 9. References

1. Neer RM, et al. Effect of parathyroid hormone (1-34) on fractures and bone mineral density in postmenopausal women with osteoporosis. N Engl J Med 2001.
2. Vahle JL, et al. Skeletal changes in rats given daily subcutaneous injections of recombinant human parathyroid hormone (1-34). Toxicol Pathol 2002.
3. Cosman F, et al. Treatment sequence matters: anabolic and antiresorptive therapy for osteoporosis. J Bone Miner Res 2017.

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