

TB-500

Thymosin Beta-4 Fragment · Investigational Research Compound ·
Actin-Binding Peptide



A plain-language guide to what TB-500 is, how its actin-regulating signaling is designed to work, and what published peptide research has found so far — written for newcomers to peptide science, not just specialists.

COMPOSITION

TB-500 acetate
10mg

STRUCTURE

7-aa synthetic fragment of the
43-aa protein Thymosin Beta-4

STUDIED HALF-LIFE

~2–3 days for full-length Tβ4;
fragment-dependent, longer
than most repair peptides

1 What Is TB-500?

TB-500 is a synthetic peptide corresponding to the active binding region of Thymosin Beta-4 (Tβ4), a naturally occurring 43-amino-acid protein present in nearly all human cells and particularly abundant in wound fluid and platelets. Researchers identified that a short sequence within Tβ4 — the actin-binding domain — accounts for much of the protein's regenerative activity, and TB-500 was designed as that fragment.

Because Tβ4's primary function is regulating actin, the structural protein cells use to change shape and migrate, TB-500's research profile centers on cell migration and tissue repair rather than on receptor signaling. It is studied across tendon, muscle, cardiac, and corneal repair models, and is frequently paired with BPC-157 in combination research for its complementary mechanism.

NEW TO THIS? KEY TERMS

Thymosin Beta-4 (Tβ4) — a naturally occurring protein that regulates actin, the structural protein cells use to move and rebuild.

Actin — the cytoskeletal protein that allows cells to change shape and migrate to injury sites.

Cell migration — the movement of repair cells (fibroblasts, endothelial cells, stem cells) toward damaged tissue.

Angiogenesis — the formation of new blood vessels, central to tissue repair and wound healing.

2 How It's Designed to Work

TB-500's research profile rests on three linked steps — actin binding at the cytoskeleton, a cell-migration signal that recruits repair cells to injured tissue, and a downstream cascade of vascular and anti-inflammatory effects:

ACTIN BINDING

CYTOSKELETAL REGULATION

Studied as binding actin monomers and promoting actin polymerization, the structural change cells require to alter shape and move.

CELL MIGRATION SIGNAL

REPAIR CELL RECRUITMENT

Enhanced actin mobility is studied as accelerating migration of fibroblasts, endothelial cells, and progenitor cells into damaged tissue.

DOWNSTREAM CASCADE

VASCULAR & ANTI-INFLAMMATORY EFFECTS

Downstream effects studied include new blood vessel formation, reduced inflammatory cytokine activity, and support of muscle fiber regeneration.

The actin binding, migration signaling, and vascular cascade are studied together as one mechanism — a structural peptide fragment that mobilizes the body's own repair cells rather than acting on a receptor.

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3 What the Research Shows

Research on TB-500 and its parent protein Thymosin Beta-4 is largely preclinical, though Tβ4 itself has reached clinical trials in ophthalmology and cardiac repair. Below is a plain-language summary of frequently cited findings.

Cardiac Repair Discovery Bock-Marquette et al., Nature, 2004

A foundational study reported Thymosin Beta-4 promoted cardiac cell migration and survival following myocardial infarction, improving cardiac function and establishing Tβ4 as a regenerative research target.

Wound Healing & Cell Migration Review Sosne et al., Expert Opinion on Biological Therapy, 2010

Reviews of Tβ4 research report promotion of cell migration, reduced inflammation, and support of new blood vessel formation across wound-healing and corneal-repair models, attributed to its actin-regulating activity.

Skeletal Muscle Repair Spurney et al., PLOS Currents Muscular Dystrophy, 2010

In a mouse model of muscular dystrophy, Thymosin Beta-4 treatment reported improved cardiac and skeletal muscle function, supporting its studied role in muscle fiber repair and regeneration.

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4 Effects Observed in Research

Across the studies above, three effects recur most often. These are findings from controlled research settings, not guarantees or claims about any individual outcome.

Muscle & Connective Tissue Repair
Improved muscle fiber regeneration and connective tissue healing reported across studies of Thymosin Beta-4.

Cell Migration & Angiogenesis
Accelerated repair cell migration and new blood vessel formation reported via actin-regulating activity.

Reduced Inflammation
Decreased inflammatory cytokine activity reported across wound-healing, corneal, and cardiac injury models.

5 Storage & Reconstitution Guide

TB-500 is supplied as a lyophilized (freeze-dried) powder that must be reconstituted before use in the lab. The steps below reflect standard peptide-handling practice — always defer to the certificate of analysis (COA) that ships with each vial.

BEFORE RECONSTITUTION
Keep the lyophilized vial refrigerated (2–8°C) for short-term storage, or frozen (–20°C) for longer-term storage, protected from light at all times. The unreconstituted powder is stable for the longest window — reconstitute only what will be used in the near term.

RECONSTITUTING WITH BACTERIOSTATIC WATER
Bring the vial to room temperature. Using a sterile syringe, draw bacteriostatic water and inject it slowly down the inside wall of the vial — not directly onto the powder — to avoid agitating and denaturing the peptide. Gently swirl to dissolve; never shake.

RESULTING CONCENTRATION FOR A 10MG VIAL

Bacteriostatic Water Added	Resulting Concentration
1 mL	10 mg/mL
2 mL	5 mg/mL
3 mL	3.3 mg/mL
5 mL	2 mg/mL

AFTER RECONSTITUTION
Store the reconstituted solution refrigerated at 2–8°C and protected from light. Reconstituted peptide solutions are generally most stable when used within a few weeks — check the COA for the exact window for each lot. Avoid repeated freeze–thaw cycles, label vials with the reconstitution date, and discard any solution that appears cloudy, discolored, or has been left at room temperature for an extended period.

SOURCES

Bock-Marquette, I. et al. Thymosin beta4 activates integrin-linked kinase and promotes cardiac cell migration, survival and cardiac repair · Nature, 2004 · Sosne, G. et al. Thymosin beta 4: a potential novel therapy for neurotrophic keratopathy, dry eye, and ocular surface diseases · Expert Opinion on Biological Therapy, 2010 · Spurney, C.F. et al. Preclinical studies for Thymosin Beta4 as a therapy for muscular dystrophy · PLOS Currents Muscular Dystrophy, 2010.

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