BIOMOD LABS · RESEARCH PEPTIDE CO.

RESEARCH COMPOUND MONOGRAPH · SERIES 1 · NO. 011

TB-500

A 17-amino-acid fragment of thymosin β-4 corresponding to the actin-binding domain of the parent 43-residue peptide — supplied as a research compound for in-vitro and animal-model studies of cell migration, angiogenesis, and tissue repair.

CAS REGISTRY	CATALOG REFERENCE	CLASS	DATE OF ISSUE
885340-08-9	BM-LYO-011	Synthetic peptide fragment · 17 a.a.	May 2026

TB-500 is the research-peptide designation for a 17-amino-acid fragment of the parent thymosin β-4 protein, corresponding to the central actin-binding domain of the full 43-residue molecule. Thymosin β-4 was first isolated from bovine thymus tissue in 1981 by Teresa L. K. Low and Allan L. Goldstein at the NIH, and subsequent work characterised it as the principal G-actin sequestering protein in mammalian cells — present in essentially every nucleated cell type and at unusually high concentrations at sites of active tissue repair. The TB-500 fragment retains the actin-binding pharmacophore of the parent protein while being more chemically tractable for laboratory synthesis. ***This monograph summarises published preclinical findings for laboratory research reference only.***

01 Compound Profile

COMMON DESIGNATION	TB-500 · Thymosin β-4 fragment · TB4 fragment
PARENT PROTEIN	Thymosin β-4 — 43-residue G-actin-sequestering protein
MOLECULAR MASS	~1900 g · mol^{-1}
PRIMARY MOLECULAR FUNCTION	G-actin sequestration — binds monomeric (G) actin and modulates the G-to-F-actin equilibrium critical for cell migration[1]
PHYSICAL FORM	White lyophilised solid
SOLUBILITY (LAB RECONSTITUTION)	Highly water-soluble in sterile water and bacteriostatic water
STORAGE (RESEARCH HANDLING)	Lyophilised solid: −18 °C, desiccated; reconstituted solution refrigerated 2–8 °C short-term; aliquoted long-term at −18 °C
ANALYTICAL SPECIFICATION	≥ 98 % purity by HPLC (BIOMOD Labs internal release specification)

02 Origin and Chemistry

Thymosin β-4 was originally identified during NIH-led fractionation of thymus-derived peptide preparations in the late 1970s and early 1980s. Subsequent biochemistry established the molecule as the principal G-actin sequestering protein in mammalian cells, present in essentially every nucleated cell type at intracellular concentrations on the order of 0.1–0.5 mM — among the most abundant intracellular proteins in many cell types. The actin-binding domain of thymosin β-4 maps to a defined central region of the 43-residue protein; the TB-500 designation refers to a 17-residue synthetic fragment encompassing this actin-binding pharmacophore.[2]

Products sold under the TB-500 designation in the research-chemical supply chain are not always chemically identical to the recombinant full-length thymosin β-4 used in primary clinical research; the BIOMOD Labs designation corresponds to the synthetic 17-residue actin-binding fragment.

03 Proposed Mechanisms in Preclinical Models

The published preclinical literature on thymosin β-4 and the TB-500 actin-binding fragment attributes observed effects to a constellation of pathways centring on actin biology. **G-actin sequestration** — the molecule binds monomeric (G) actin in 1:1 stoichiometry, modulating the G/F-actin equilibrium that drives cytoskeletal remodelling required for cell migration. **Akt / mTOR signalling** — preclinical work documents upregulation of Akt phosphorylation and downstream mTOR pathway activation in cells exposed to the peptide. **VEGF and angiogenesis** — multiple animal-model studies report elevated VEGF expression and accelerated vascular network formation in

wound and ischaemia preparations. **NF-κB-related inflammation modulation** — preclinical inflammation models report attenuation of inflammatory marker expression. **TGF-β / collagen / fibrosis** — animal fibrosis models report reduced collagen deposition.[1, 3] PRECLINICAL · RODENT

An additional consideration is the natural cleavage product Ac-SDKP (acetyl-Ser-Asp-Lys-Pro), a tetrapeptide derived from the N-terminus of thymosin β-4 that has been independently characterised as a specific inhibitor of hematopoietic stem cell proliferation and as an anti-fibrotic peptide in cardiac and pulmonary preparations.[3]

04 Preclinical Findings

SYSTEM	ANIMAL MODEL / PREPARATION	REPORTED OBSERVATION	REF.
Dermal wound	db/db diabetic mice; aged mice	Accelerated wound closure with full-length thymosin β-4 and synthetic actin-binding-domain peptide	4
Corneal wound	Rat / mouse alkali-burn corneal preparations	Accelerated re-epithelialisation; reduced inflammation	5
Cardiac repair	Mouse myocardial infarction model	Epicardial progenitor cell activation; promoted cardiomyocyte differentiation	1
Septic shock	Rodent endotoxin-induced sepsis	Reduced lethality; downregulation of inflammatory mediators	6
Actin biology	Cell-free biochemistry & cell-culture studies	G-actin sequestration, F-actin assembly regulation, cell migration support	2
Multi-tissue review	Comprehensive preclinical animal-studies review	Wound healing, cardiac repair, CNS injury models, dermal repair across multiple species	1

05 Research Synthesis & Limitations

METHODOLOGICAL NOTES

The thymosin β-4 corpus is one of the broader-based regenerative-peptide literatures, with consistent observations of actin-binding biology, cell migration support, and accelerated histological repair across multiple independent rodent models from independent laboratories. For researchers, the principal considerations are (a) the TB-500 designation refers specifically to a 17-residue actin-binding-domain fragment, which is not chemically identical to the recombinant full-length thymosin β-4 used in primary clinical research — observations made with full-length protein may not translate directly to the fragment, and vice versa; and (b) the Ac-SDKP tetrapeptide cleavage product of thymosin β-4 has distinct biological activity (hematopoietic stem cell modulation, anti-fibrosis) that is not necessarily reproduced by the TB-500 fragment.

06 Laboratory Handling, Reconstitution, and Storage

Lyophilised TB-500 is supplied under research-use specifications. The peptide is highly water-soluble; reconstitution in sterile water for injection or bacteriostatic water is standard practice. Lyophilised storage at −18 °C, desiccated; reconstituted solutions at refrigerated 2–8 °C for short-term work; aliquoted long-term storage at −18 °C with minimised freeze–thaw. Working concentrations are determined by the investigator's experimental design.

07 References

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5. Sosne G, Szliter EA, Barrett R, Kernacki KA, Kleinman H, Hazlett LD. Thymosin beta 4 promotes corneal wound healing and decreases inflammation in vivo following alkali injury. *Exp Eye Res.* 2002;74(2):293–299. pubmed.ncbi.nlm.nih.gov/11950239

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