

RESEARCH MONOGRAPH . COMPOUND PROFILE

Thymosin alpha 1 (Thymalfasin)

A 28-amino-acid acetylated peptide originally isolated from thymus tissue, registered as Zadaxin in approximately 35 jurisdictions for chronic hepatitis B and as adjunctive therapy in immunocompromised oncology patients.

DOCUMENT	KDC-MN-1339
ISSUED	2026-05-10
REVISION	1.0
AUTHOR	Kodiac biolabs Research
COMPOUND CLASS	28-residue immunomodulatory thymic peptide

ABSTRACT

Thymosin alpha 1 (Talpha1; Ac-Ser-Asp-Ala-Ala-Val-Asp-Thr-Ser-Ser-Glu-Ile-Thr-Thr-Lys-Asp-Leu-Lys-Glu-Lys-Lys-Glu-Val-Val-Glu-Glu-Ala-Glu-Asn; thymalfasin; CAS 62304-98-7; molecular formula C₁₂₉H₂₁₅N₃₃O₅₅; molecular weight 3108.27) is a 28-residue N-acetylated peptide originally isolated from bovine thymus tissue by Allan Goldstein and colleagues at the Albert Einstein College of Medicine in 1977 as a fraction of the broader thymosin fraction 5 immunomodulator. The synthetic peptide was developed by SciClone Pharmaceuticals as Zadaxin and approved in approximately 35 jurisdictions (including most of Asia, Latin America, and parts of Europe) for chronic hepatitis B, chronic hepatitis C (in combination with interferon and ribavirin), and as adjunctive immune-restorative therapy in immunocompromised oncology patients. The compound is not FDA-approved (Phase 3 trials in hepatitis C did not establish independent efficacy at the threshold required by FDA; the agent is approved on different evidence packages in the jurisdictions where it is marketed). Mechanism is multifactorial immunomodulation: enhancement of T cell maturation through Toll-like receptor 9 signaling on dendritic cells, increased Th1 cytokine production, enhanced natural killer cell cytotoxicity, and a more complex effect on regulatory T cell phenotypes that varies with disease context. Routes of administration are subcutaneous twice weekly at 1.6 mg per dose. The compound is distinct from the broader Thymosin Fraction 5 extract and from other thymosin family peptides (Thymosin beta-4, KDC-MN-003; Thymalin; Thymulin; Thymopentin) in being the principal alpha-class thymic immunomodulator with regulatory approval and a substantial

randomized clinical trial database. Adverse events are mild and dominated by injection site reactions. Clinical use in COVID-19 and sepsis received attention in 2020 to 2022 with mixed efficacy results in randomized trials.

1. 1. Compound Identification

Thymosin alpha 1 (Thymalfasin) is described as 28-residue immunomodulatory thymic peptide. A 28-amino-acid acetylated peptide originally isolated from thymus tissue, registered as Zadaxin in approximately 35 jurisdictions for chronic hepatitis B and as adjunctive therapy in immunocompromised oncology patients.

1.1. 1.1 Class summary

28-residue immunomodulatory thymic peptide

1.2. 1.2 Subtitle

A 28-amino-acid acetylated peptide originally isolated from thymus tissue, registered as Zadaxin in approximately 35 jurisdictions for chronic hepatitis B and as adjunctive therapy in immunocompromised oncology patients.

2. Abstract

Thymosin alpha 1 (Talpha1; Ac-Ser-Asp-Ala-Ala-Val-Asp-Thr-Ser-Ser-Glu-Ile-Thr-Thr-Lys-Asp-Leu-Lys-Glu-Lys-Lys-Glu-Val-Val-Glu-Glu-Ala-Glu-Asn; thymalfasin; CAS 62304-98-7; molecular formula C129H215N33O55; molecular weight 3108.27) is a 28-residue N-acetylated peptide originally isolated from bovine thymus tissue by Allan Goldstein and colleagues at the Albert Einstein College of Medicine in 1977 as a fraction of the broader thymosin fraction 5 immunomodulator. The synthetic peptide was developed by SciClone Pharmaceuticals as Zadaxin and approved in approximately 35 jurisdictions (including most of Asia, Latin America, and parts of Europe) for chronic hepatitis B, chronic hepatitis C (in combination with interferon and ribavirin), and as adjunctive immune-restorative therapy in immunocompromised oncology patients. The compound is not FDA-approved (Phase 3 trials in hepatitis C did not establish independent efficacy at the threshold required by FDA; the agent is approved on different evidence packages in the jurisdictions where it is marketed). Mechanism is multifactorial immunomodulation: enhancement of T cell maturation through Toll-like receptor 9 signaling on dendritic cells, increased Th1 cytokine production, enhanced natural killer cell cytotoxicity, and a more complex effect on regulatory T cell phenotypes that varies with disease context. Routes of administration are subcutaneous twice weekly at 1.6 mg per dose. The compound is distinct from the broader Thymosin Fraction 5 extract and from other thymosin family peptides (Thymosin beta-4, KDC-MN-003; Thymalin; Thymulin; Thymopentin) in being the principal alpha-class thymic immunomodulator with regulatory approval and a substantial randomized clinical trial database. Adverse events are mild and dominated by injection site reactions. Clinical use in COVID-19 and sepsis received attention in 2020 to 2022 with mixed efficacy results in randomized trials.

3. Mechanism of Action

Toll-like receptor 9 agonism on dendritic cells and T cell precursors. Enhanced Th1 cytokine production; increased NK cell cytotoxicity; T cell maturation. Composite immune restoration in immunocompromised states.

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

Approved subcutaneous 1.6 mg twice weekly for chronic hepatitis B. Oncology adjunctive 1.6 mg subcutaneous twice weekly. Pediatric and severe sepsis dosing per published protocols.

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. Goldstein AL, et al. Thymosin alpha 1: chemistry, mechanism of action and clinical applications. Ann N Y Acad Sci 2007.
2. Sjogren MH. Thymalfasin: an immune system enhancer for the treatment of liver disease. J Gastroenterol Hepatol 2004.
3. Garaci E, et al. Thymosin alpha 1: from bench to bedside. Ann N Y Acad Sci 2007.
4. Kumar V, et al. Thymosin alpha 1 in COVID-19. Front Immunol 2021.

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