

RESEARCH MONOGRAPH . COMPOUND PROFILE

PTD-DBM

A research-grade peptide that disrupts the CXXC5-Dishevelled negative regulatory complex on the canonical Wnt pathway, studied for hair follicle stimulation and tissue regeneration.

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AUTHOR	Kodi <i>ac</i> bio <i>l</i> abs Research
COMPOUND CLASS	Wnt pathway activator and CXXC5-Dishevelled interaction inhibitor

RESEARCH USE ONLY

ABSTRACT

PTD-DBM (protein transduction domain conjugated to Dishevelled-binding motif; DBM derived from the Dishevelled-PDZ-binding C-terminus of CXXC5; molecular weight approximately 4 kDa as a synthetic conjugate) is a research-grade peptide developed at Yonsei University in South Korea by Kang-Yell Choi and colleagues as a tool for activating canonical Wnt signaling at the level of the Dishevelled scaffold. The canonical Wnt pathway is a developmental signaling system that drives stem cell self-renewal, tissue regeneration, and (importantly for hair) the anagen phase of the hair follicle cycle. CXXC5 is a negative regulator of canonical Wnt signaling that binds Dishevelled at a defined motif and prevents Dishevelled-mediated signal propagation; loss of CXXC5 in knockout mice produces enhanced Wnt signaling, accelerated hair cycle entry, and resistance to age-related hair thinning. The PTD-DBM peptide is designed to mimic the CXXC5 Dishevelled-binding motif and competitively displace endogenous CXXC5 from Dishevelled, releasing canonical Wnt signal flow. The PTD (HIV-Tat-derived protein transduction domain) enables cell membrane crossing of the otherwise impermeable peptide. Reported activities in rodent and human ex vivo hair follicle models include accelerated anagen induction, increased hair shaft length, and protection against chemotherapy-induced and androgenetic alopecia in rodent models. Topical formulation with the valproic acid co-treatment (KDC-MN-1348) has been explored as a synergistic Wnt-activating combination. The compound is research-grade and not approved by any regulatory authority; clinical translation through Yonsei University spin-out CXC5 has been in early stages. Reconstitution

requires aqueous buffer with co-solvent for the peptide-PTD conjugate; topical delivery in the hair-follicle context is the principal route under investigation.

1. Compound Identification

PTD-DBM is described as wnt pathway activator and cxxc5-dishevelled interaction inhibitor. A research-grade peptide that disrupts the CXXC5-Dishevelled negative regulatory complex on the canonical Wnt pathway, studied for hair follicle stimulation and tissue regeneration.

1.1 Class summary

Wnt pathway activator and CXXC5-Dishevelled interaction inhibitor

1.2 Subtitle

A research-grade peptide that disrupts the CXXC5-Dishevelled negative regulatory complex on the canonical Wnt pathway, studied for hair follicle stimulation and tissue regeneration.

2. Abstract

PTD-DBM (protein transduction domain conjugated to Dishevelled-binding motif; DBM derived from the Dishevelled-PDZ-binding C-terminus of CXXC5; molecular weight approximately 4 kDa as a synthetic conjugate) is a research-grade peptide developed at Yonsei University in South Korea by Kang-Yell Choi and colleagues as a tool for activating canonical Wnt signaling at the level of the Dishevelled scaffold. The canonical Wnt pathway is a developmental signaling system that drives stem cell self-renewal, tissue regeneration, and (importantly for hair) the anagen phase of the hair follicle cycle. CXXC5 is a negative regulator of canonical Wnt signaling that binds Dishevelled at a defined motif and prevents Dishevelled-mediated signal propagation; loss of CXXC5 in knockout mice produces enhanced Wnt signaling, accelerated hair cycle entry, and resistance to age-related hair thinning. The PTD-DBM peptide is designed to mimic the CXXC5 Dishevelled-binding motif and competitively displace endogenous CXXC5 from Dishevelled, releasing canonical Wnt signal flow. The PTD (HIV-Tat-derived protein transduction domain) enables cell membrane crossing of the otherwise impermeable peptide. Reported activities in rodent and human ex vivo hair follicle models include accelerated anagen induction, increased hair shaft length, and protection against chemotherapy-induced and androgenetic alopecia in rodent models. Topical formulation with the valproic acid co-treatment (KDC-MN-1348) has been explored as a synergistic Wnt-activating combination. The compound is research-grade and not approved by any regulatory authority; clinical translation through Yonsei University spin-out CXC5 has been in early stages. Reconstitution requires aqueous buffer with co-solvent for the peptide-PTD conjugate; topical delivery in the hair-follicle context is the principal route under investigation.

3. Mechanism of Action

Competitive displacement of endogenous CXXC5 from Dishevelled at the CXXC5 binding motif. Disinhibition of canonical Wnt signaling. Anagen-phase hair follicle activation, stem cell self-renewal in tissue regeneration models.

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

Topical research dosing 1 to 10 micromolar in vehicle for ex vivo hair follicle culture. Rodent in vivo topical 100 micrograms per application.

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. Lee SH, et al. CXXC5 is a negative-feedback regulator of the Wnt/beta-catenin pathway. Cell Death Differ 2015.
2. Lee SH, et al. PTD-DBM prevents CXXC5 from inhibiting Wnt signaling and induces hair regrowth. EMBO Mol Med 2017.
3. Ryu YC, et al. Combinatorial treatment with PTD-DBM and valproic acid for hair regrowth. Br J Dermatol 2021.

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