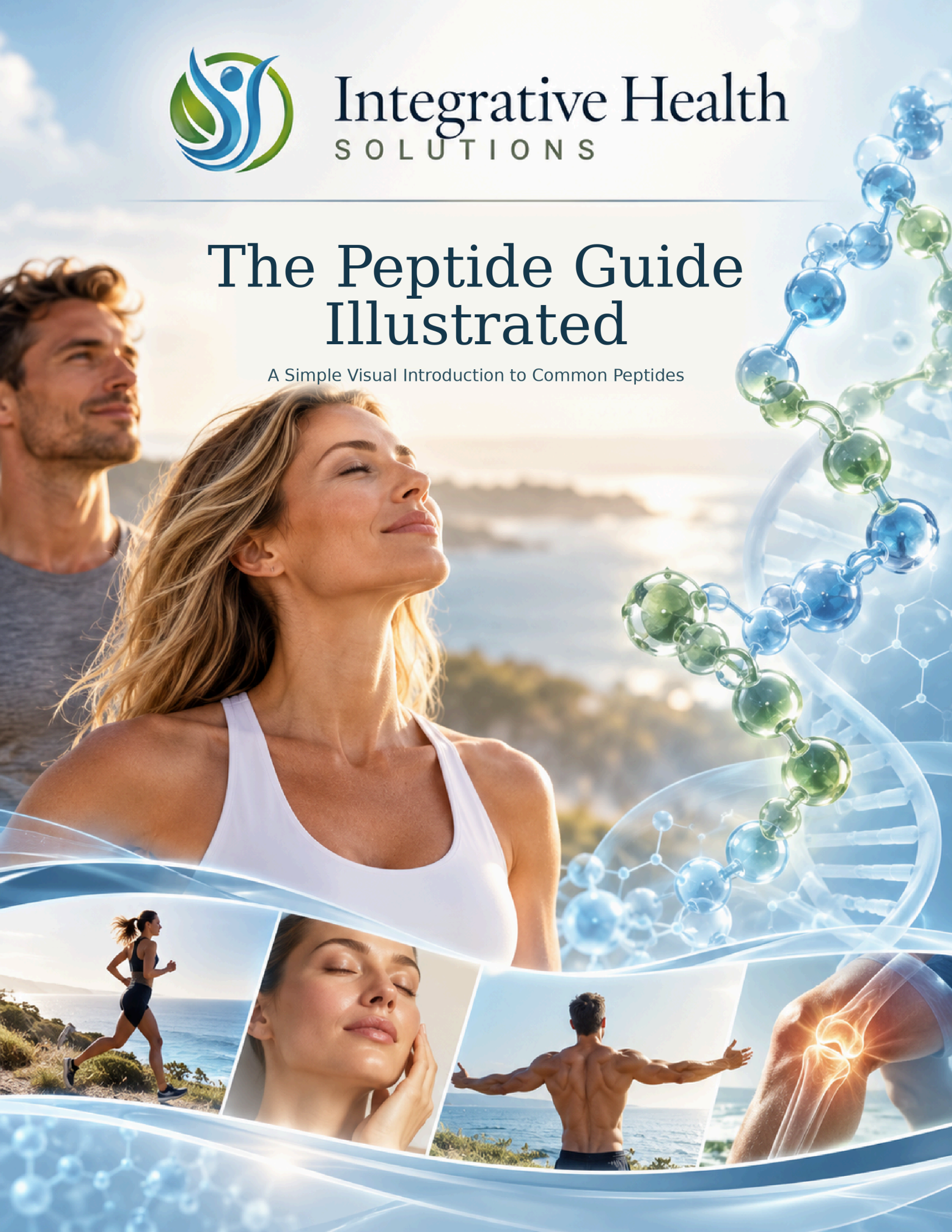
Integrative Health
SOLUTIONS

The Peptide Guide Illustrated

A Simple Visual Introduction to Common Peptides



CONTENTS

Explore the peptides

Therapy infographics and mechanisms

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Explore the peptides

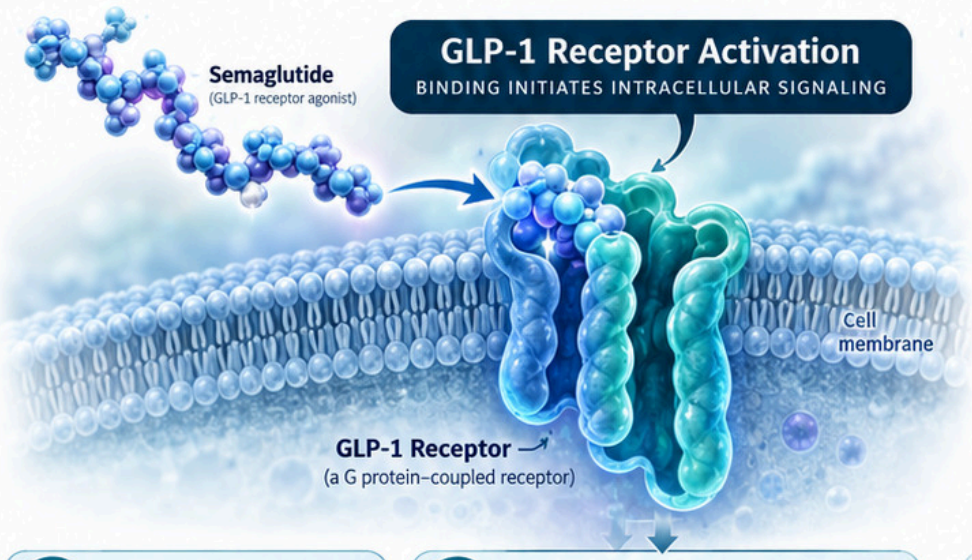
Therapy infographics and mechanisms

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Semaglutide

Mechanism, Physiology, and Clinical Effects

A GLP-1 RECEPTOR AGONIST FOR METABOLIC HEALTH



Pharmacokinetics

PREDICTABLE, LONG-ACTING EXPOSURE



Subcutaneous dosing
Once weekly



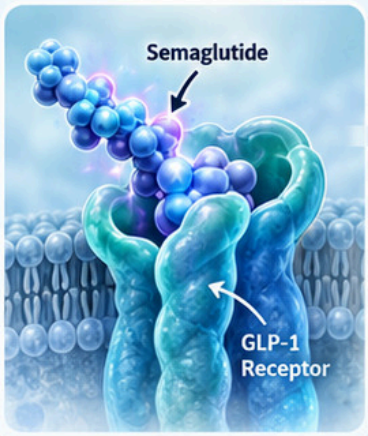
Long half-life
≈ 1 week
(~165–184 hours)



Steady-state
Achieved with repeated weekly dosing (≈ 4–5 weeks)

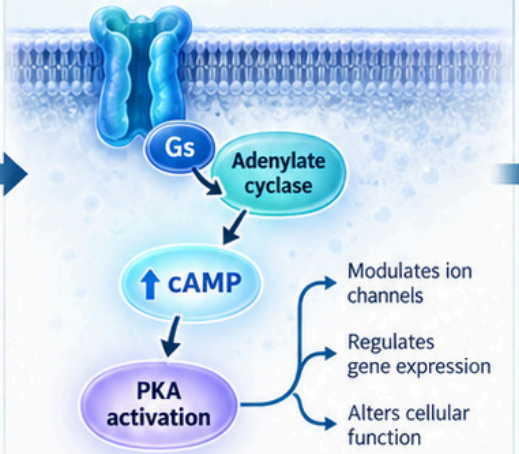
1 Drug binding

SEMAGLUTIDE BINDS TO THE GLP-1 RECEPTOR



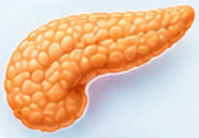
2 Intracellular signaling

ACTIVATES cAMP–PKA PATHWAY



3 Physiologic effects

MULTI-ORGAN ACTIONS



Pancreas
- ↑ Glucose-dependent insulin secretion
- ↓ Glucagon secretion



Stomach
- ↓ Slows gastric emptying



Hypothalamus
- ↑ Satiety
- ↓ Hunger

4 Clinical outcomes

TRANSLATES PHYSIOLOGY INTO MEANINGFUL HEALTH BENEFITS



Reduced appetite
Lower caloric intake



Weight loss
Sustained, clinically meaningful reductions in body weight



Improved glycemic control
Lower HbA1c and better metabolic health

! Common Side Effects

MOSTLY GASTROINTESTINAL AND OFTEN TRANSIENT



Nausea



Vomiting



Constipation



Abdominal discomfort

Evidence Level

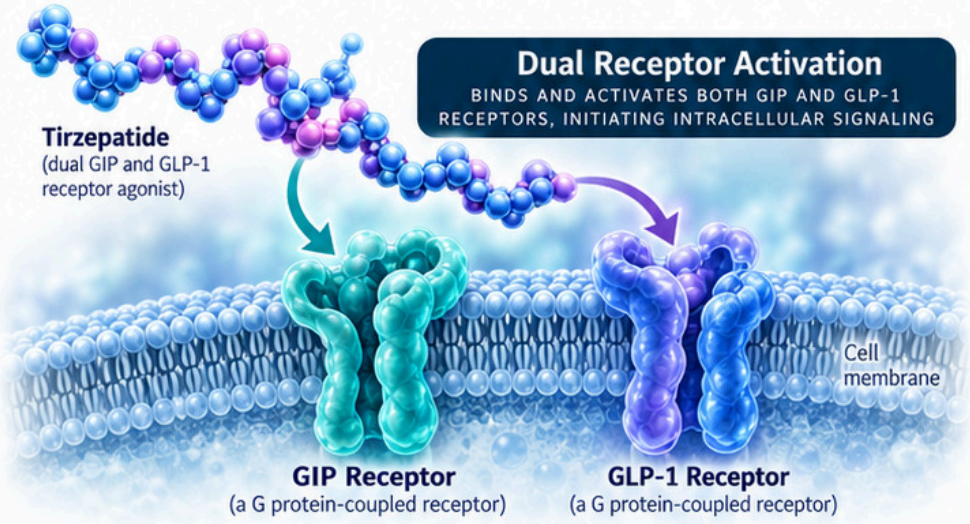


Strong human randomized trial evidence
Consistent benefits for weight loss and glycemic control in multiple large RCTs.

Tirzepatide

Mechanism, Physiology, and Clinical Effects

A DUAL GIP AND GLP-1 RECEPTOR AGONIST FOR METABOLIC HEALTH



Dual Receptor Activation

BINDS AND ACTIVATES BOTH GIP AND GLP-1 RECEPTORS, INITIATING INTRACELLULAR SIGNALING

Pharmacokinetics

PREDICTABLE, LONG-ACTING EXPOSURE



Subcutaneous dosing
Once weekly



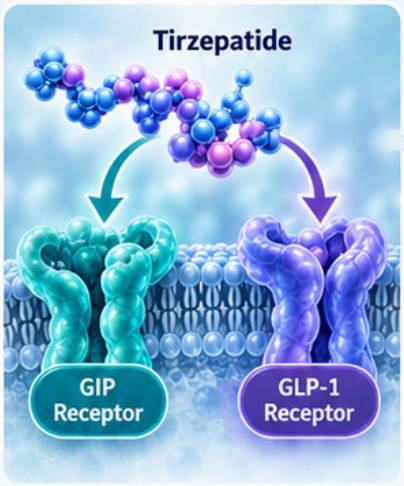
Long half-life ≈ 5 days
(~120 hours)



Steady-state
Achieved with repeated weekly dosing (≈ 4–5 weeks)

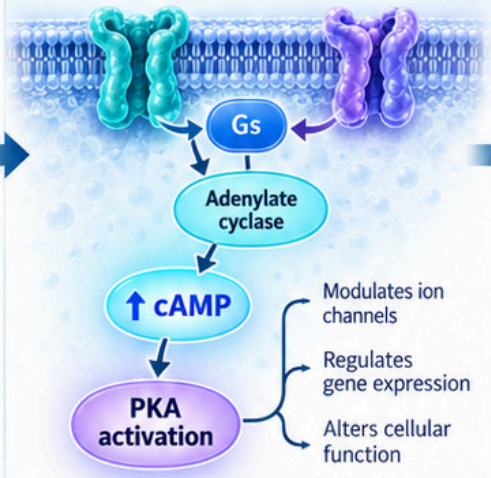
1 Drug binding

TIRZEPATIDE ACTIVATES BOTH GIP AND GLP-1 RECEPTORS



2 Intracellular signaling

ACTIVATES cAMP–PKA PATHWAY VIA Gs PROTEIN



3 Physiologic effects

MULTI-ORGAN ACTIONS



Pancreas
- ↑ Glucose-dependent insulin secretion
- ↓ Glucagon secretion



Stomach
- ↓ Slows gastric emptying



Hypothalamus (brain)
- ↑ Increased satiety
- ↓ Reduced hunger



Adipose tissue & liver
- ↑ Improved metabolic handling (↑ insulin sensitivity, ↓ ectopic fat)

4 Clinical outcomes

TRANSLATES PHYSIOLOGY INTO MEANINGFUL HEALTH BENEFITS



Reduced appetite
Lower caloric intake



Major weight loss
Substantial, sustained reductions in body weight



Improved glycemic control
Lower HbA1c and better overall metabolic health

Common Side Effects

MOSTLY GASTROINTESTINAL AND OFTEN TRANSIENT



Nausea



Vomiting



Diarrhea



Constipation
Abdominal discomfort

Evidence Level



Strong human randomized trial evidence
Consistent benefits for weight loss, glycemic control, and cardiometabolic health in multiple large RCTs.

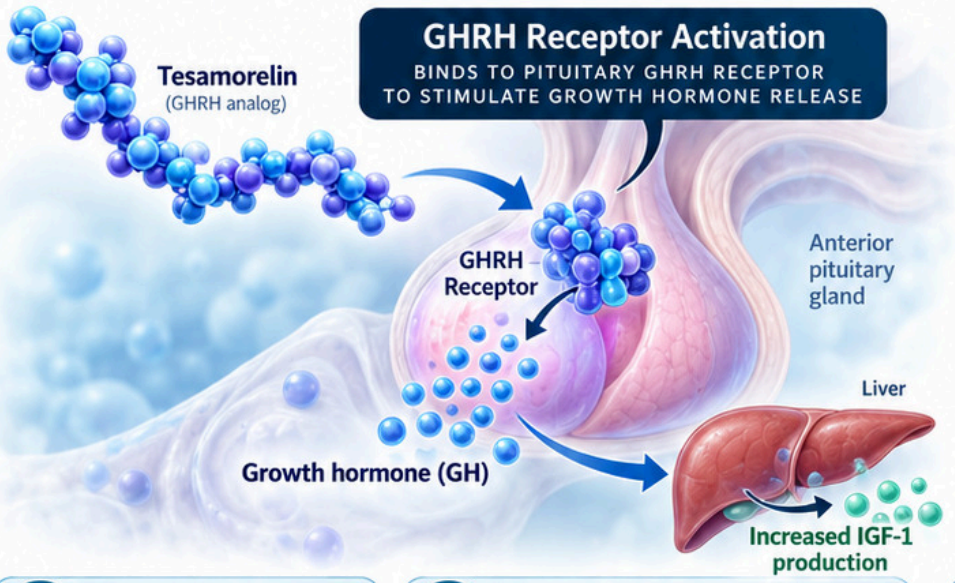
Tesamorelin

Mechanism, Physiology, and Clinical Effects

A GROWTH HORMONE–RELEASING HORMONE ANALOG FOR VISCERAL FAT REDUCTION

GHRH Receptor Activation

BINDS TO PITUITARY GHRH RECEPTOR TO STIMULATE GROWTH HORMONE RELEASE



Pharmacokinetics

PREDICTABLE, SHORT-ACTING EXPOSURE



Subcutaneous dosing
Once daily (preferably at bedtime)



Short plasma half-life
~30–60 minutes



Downstream IGF-1 effects persist
Increased IGF-1 levels can last for hours to days beyond drug exposure

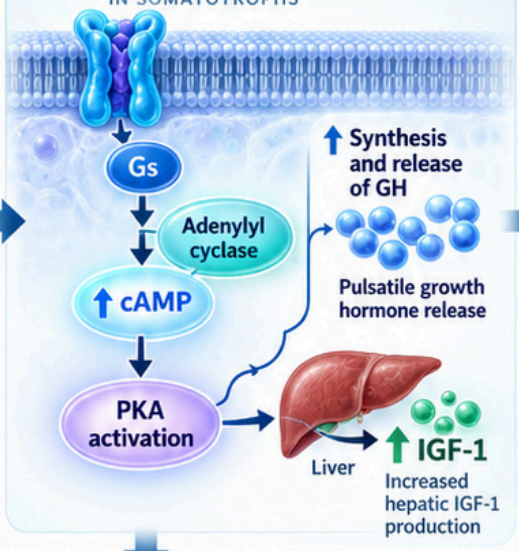
1 Drug binding

TESAMORELIN ACTIVATES PITUITARY GHRH RECEPTOR



2 Intracellular signaling

ACTIVATES cAMP PATHWAY IN SOMATOTROPHS



3 Physiologic effects

GH/IGF-1 SIGNALING AND ADIPOSE TISSUE



Pituitary
- Increased GH secretion (pulsatile)
- Higher circulating GH and IGF-1 levels



Liver
- Increased IGF-1 production
- Mediates anabolic and lipolytic effects



Adipose tissue
- Enhanced lipolysis
- Reduction in visceral adipose tissue (VAT)
- Improved body composition

4 Clinical outcomes

TRANSLATES PHYSIOLOGY INTO MEANINGFUL HEALTH BENEFITS



Reduced visceral fat
Significant reduction in abdominal VAT (measured by CT)



Improved body composition
Decreased waist circumference
Increased lean body mass



Possible metabolic benefits
Improvements in lipids and insulin sensitivity in some studies

Common Side Effects

GENERALLY MILD TO MODERATE

- **Injection-site reactions** — Redness, pain, itching
- **Arthralgia** — Joint pain
- **Edema** — Fluid retention (peripheral edema)
- **Muscle pain** — Myalgia
- **Glucose changes** — Transient increases in fasting glucose in some patients

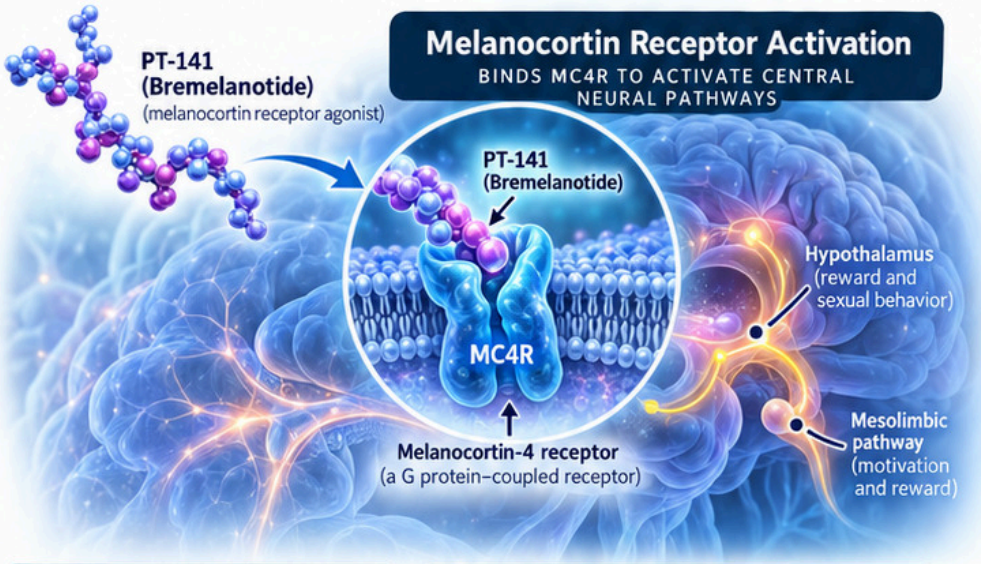
Evidence Level

Established human randomized trial evidence for HIV-associated lipodystrophy
More limited evidence for other uses (e.g., age-related body composition).

PT-141 (Bremelanotide)

Mechanism, Physiology, and Clinical Effects

A MELANOCORTIN RECEPTOR AGONIST FOR SEXUAL DESIRE PATHWAYS



Melanocortin Receptor Activation

BINDS MC4R TO ACTIVATE CENTRAL NEURAL PATHWAYS

Pharmacokinetics

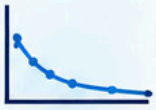
PREDICTABLE, INTERMITTENT USE



Subcutaneous dosing
As needed (on-demand)



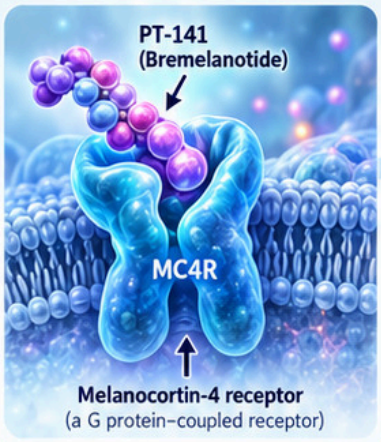
Onset of effect
Typically over 1–2 hours



Short systemic exposure
Plasma half-life ~2–3 hours

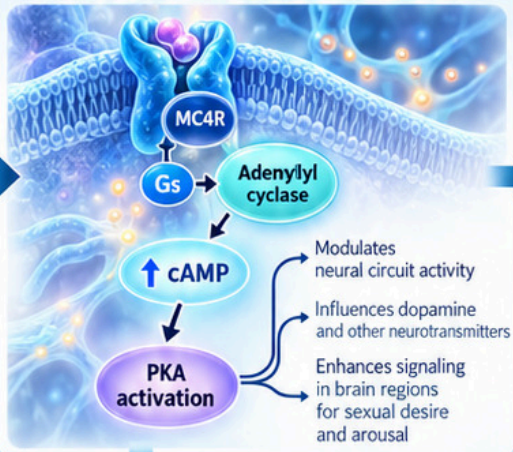
1 Drug binding

MELANOCORTIN RECEPTOR AGONISM



2 Intracellular signaling

ACTIVATES CENTRAL NEURAL PATHWAYS RELATED TO DESIRE AND AROUSAL



3 Physiologic effects

BRAIN/CENTRAL NERVOUS SYSTEM PATHWAYS



Increases sexual desire

- Activates hypothalamic and mesolimbic pathways
- Enhances motivation and sexual arousal
- May improve response to sexual cues



Minimal direct vascular effect

Acts primarily on central neural pathways rather than directly increasing blood flow (unlike PDE5 inhibitors)

4 Clinical outcomes

IMPROVES SEXUAL DESIRE AND RESPONSE IN APPROPRIATE PATIENTS



Increased sexual desire

Greater interest in sexual activity in appropriate patients



Improved sexual response

May increase arousal and satisfaction



Effective in appropriate patients

Indicated for acquired, generalized hypoactive sexual desire disorder (HSDD) in premenopausal women

Common Side Effects

USUALLY MILD TO MODERATE AND TRANSIENT



Nausea



Flushing (warmth, redness)



Headache



Injection-site reactions (redness, pain)



Transient blood-pressure rise (usually small and short-lived)

Evidence Level



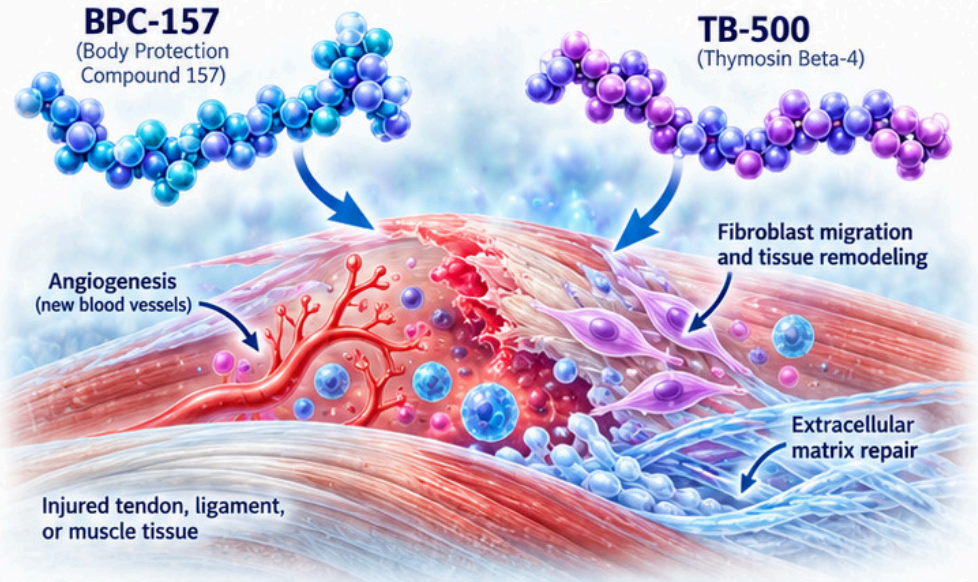
Strong human phase 3 evidence for approved indication

Consistent benefits in randomized, placebo-controlled phase 3 trials in premenopausal women with HSDD.

BPC-157 / TB-500

Mechanism, Physiology, and Clinical Effects

EXPERIMENTAL PEPTIDES ASSOCIATED WITH TISSUE REPALIR AND RECOVERY



Pharmacokinetics

HUMAN DATA NOT WELL ESTABLISHED

Human pharmacokinetics not well established
Absorption, distribution, metabolism, and elimination in humans are not fully known.

Exposure data limited
Only limited human data available from small studies and case reports.

Investigational use
Not approved for medical use. Considered experimental research peptides.

1 Drug actions

DISTINCT BUT COMPLEMENTARY MECHANISMS

BPC-157

- Associated with cytoprotection
- Promotes angiogenic signaling
- Supports tissue survival and repair
- Modulates inflammatory responses

TB-500

- Associated with actin dynamics
- Enhances cell migration
- Supports tissue remodeling
- May reduce inflammation and promote repair

2 Intracellular / tissue signaling

ACTIVATES REPAIR AND REGENERATION PATHWAYS

Angiogenesis
Increases VEGF signaling and new blood vessel formation

Fibroblast migration
Promotes fibroblast activity and cell migration to injury site

Extracellular matrix remodeling
Supports collagen synthesis, ECM organization, and tissue remodeling

Anti-inflammatory signaling
Modulates cytokine production and reduces excessive inflammation

3 Physiologic effects

SUPPORTS MULTIPLE TISSUES

Tendon support

- May enhance tendon healing and strength
- Supports collagen repair

Ligament support

- Promotes ligament repair and remodeling
- May improve structural integrity

Muscle recovery

- Supports muscle regeneration
- May reduce recovery time after injury

Wound-healing support

- May accelerate wound closure
- Supports tissue regeneration and re-epithelialization

4 Clinical outcomes

POTENTIAL RECOVERY SUPPORT, BUT CLINICAL EFFICACY REMAINS UNCERTAIN

May support recovery
Potentially faster recovery from soft tissue injuries (tendon, ligament, muscle).

Tissue-healing benefits
May enhance tissue repair, collagen organization, and functional recovery.

Clinical efficacy uncertain
More high-quality human studies are needed to confirm effectiveness and optimal use.

Common Side Effects

LIMITED HUMAL SAFETY DATA

Injection-site irritation
Redness, pain, or swelling

Limited safety data
Overall safety profile in humans is not well defined.

Uncertain long-term risks
Potential long-term effects are unknown.

Evidence Level

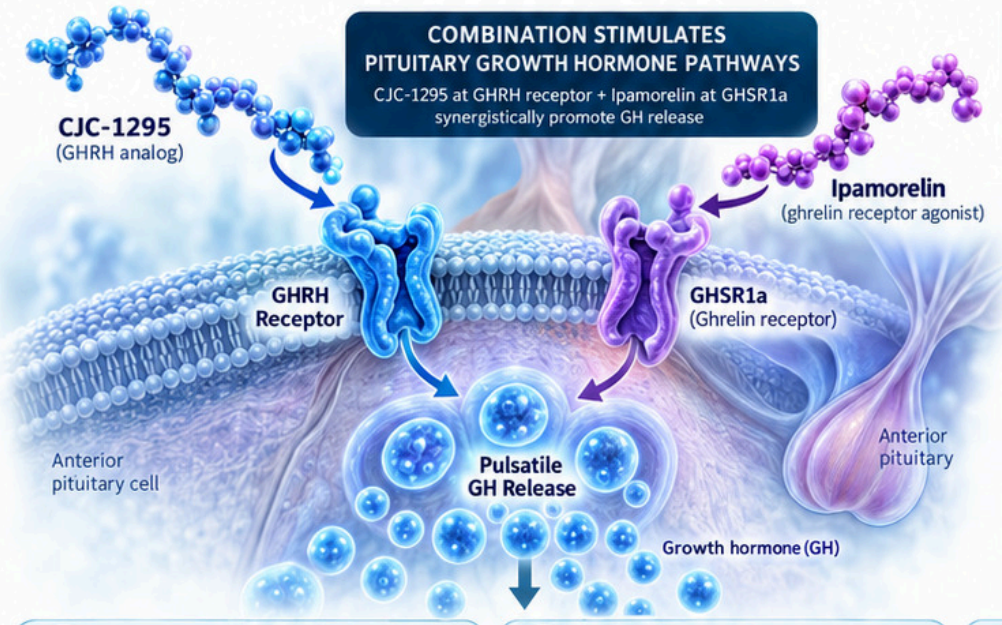
PRIMARILY ANIMAL AND PRECLINICAL EVIDENCE

Predominantly animal and preclinical evidence
Limited robust human clinical evidence. Larger, well-designed human trials are needed.

CJC-1295 / Ipamorelin

Mechanism, Physiology, and Clinical Effects

A GHRH ANALOG PLUS GHRELIN RECEPTOR AGONIST FOR GROWTH HORMONE SIGNALING



Pharmacokinetics

DISTINCT PROFILES, COMPLEMENTARY EFFECTS

Subcutaneous dosing
Once daily or per protocol (e.g., nightly)

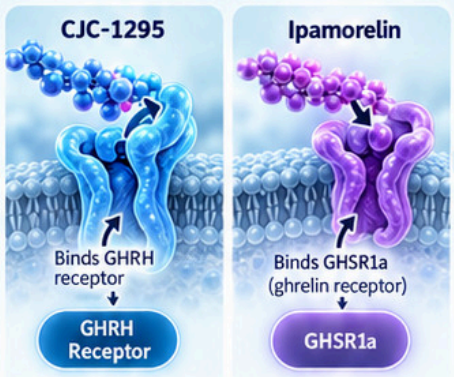
CJC-1295 prolonged effect
Duration depends on formulation (e.g., DAC: ~5–8 days; without DAC: shorter)

Ipamorelin short-acting effect
Ghrelin-mimetic activity ~1–3 hours

Downstream changes
↑ Growth hormone (GH)
↑ IGF-1 (primarily via liver)

1 Drug binding

EACH PEPTIDE BINDS ITS TARGET ON PITUITARY SOMATOTROPH CELLS

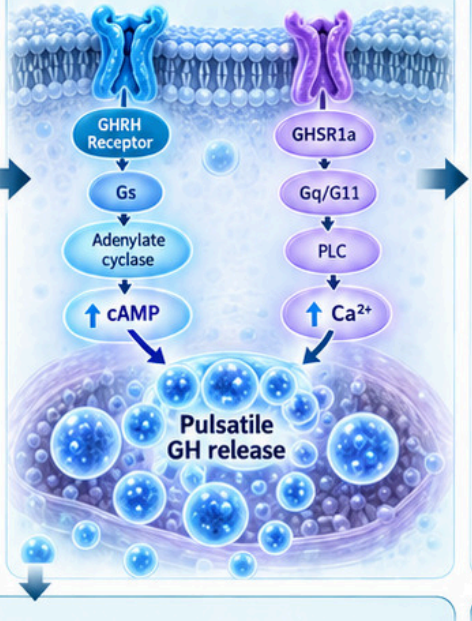


Stimulates GH release (similar to endogenous GHRH)

Stimulates GH release via ghrelin receptor pathway

2 Intracellular signaling

ACTIVATES GH SECRETORY PATHWAYS



3 Physiologic effects

MULTI-TISSUE ACTIONS OF GH AND IGF-1

Anterior pituitary
↑ Growth hormone release
More physiological, pulsatile secretion

Liver
↑ IGF-1 production
Mediates many anabolic effects

Muscle
↑ Protein synthesis
Supports lean mass, strength, and recovery

Adipose tissue
↑ Lipolysis
Supports improved body composition

Brain / Sleep
May support sleep quality
Influences GH neuroregulation

4 Clinical outcomes

POTENTIAL BENEFITS BASED ON PHYSIOLOGY AND LIMITED HUMAN DATA

Lean mass support
May help maintain or increase lean body mass

Enhanced recovery
Supports recovery and adaptation from training

Improved body composition
Potential reductions in fat mass with increased lean mass

Sleep support
May support deeper, more restorative sleep

Clinical evidence for body composition and performance outcomes is limited, with most data from small studies. More high-quality randomized trials are needed.

Common Side Effects

USUALLY MILD AND TRANSIENT

- Edema (fluid retention)
- Paresthesia (tingling)
- Headache
- Flushing
- Increased appetite
- Changes in glucose regulation

Evidence Level

LIMITED HUMAN EVIDENCE

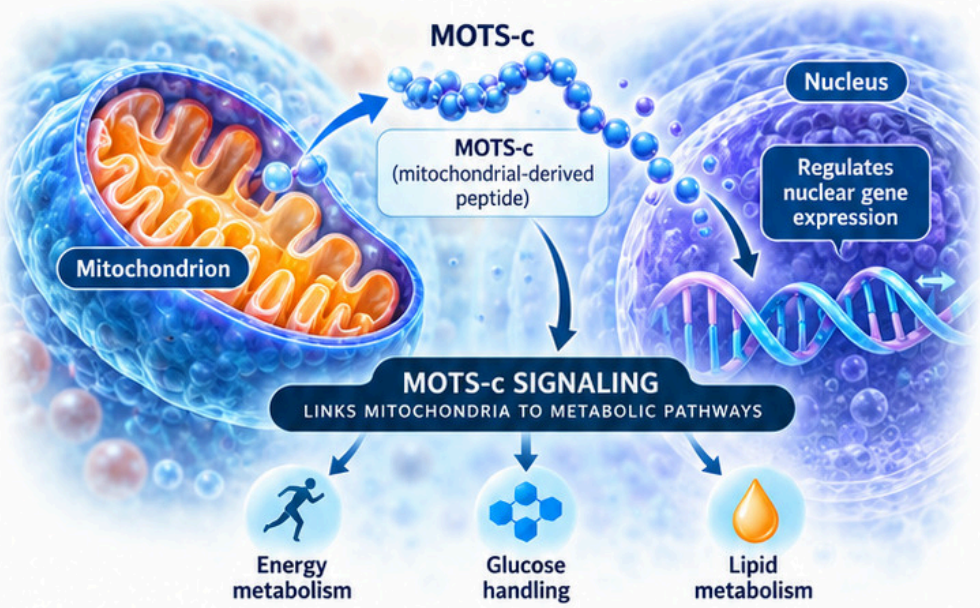
Limited clinical evidence
Stronger physiologic rationale than outcome-trial support

Biologic plausibility and consistent increases in GH/IGF-1, but more large, randomized studies are needed to confirm clinical benefits for body composition, performance, and recovery.

MOTS-c

Mechanism, Physiology, and Clinical Effects

A MITOCHONDRIAL-DERIVED PEPTIDE LINKED TO. METABOLIC HOMEOSTASIS



Pharmacokinetics

HUMAN DATA ARE LIMITED

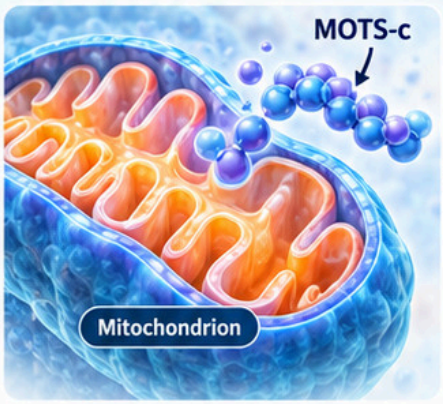
Human pharmacokinetic data are limited
Absorption, distribution, metabolism, and excretion not well defined.

Investigational peptide
Not approved for clinical use.

Exposure characteristics not well defined
Dose, half-life, and bioavailability in humans are unknown.

1 Peptide origin and action

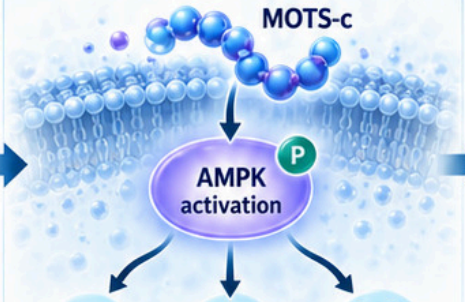
MITOCHONDRIAL-DERIVED SIGNALING PEPTIDE



MOTS-c is a short peptide encoded within mitochondrial DNA that acts as a retrograde signal from mitochondria to the nucleus and other tissues.

2 Intracellular signaling

ACTIVATES AMPK AND METABOLIC STRESS ADAPTATION



Metabolic stress adaptation

Glucose and lipid handling

Nuclear gene regulation
Alters expression of metabolic genes

3 Physiologic effects

MULTI-ORGAN ACTIONS



Skeletal muscle
- ↑ Metabolic function
- ↑ Exercise adaptation
- ↓ Insulin resistance (conceptual)



Liver
- ↑ Glucose metabolism
- ↑ Lipid metabolism



Adipose tissue
- ↑ Healthy adipose function
- ↑ Lipid utilization



Systemic effects
- ↑ Metabolic flexibility
- ↑ Insulin sensitivity (conceptual)

4 Clinical outcomes

POTENTIAL SUPPORT FOR METABOLIC HEALTH AND EXERCISE ADAPTATION

Metabolic health support
Potential to support glucose and lipid metabolism.

Exercise-related adaptation
May enhance metabolic adaptation to exercise and physical activity.

Clinical outcomes not firmly established
Human clinical benefits remain to be confirmed in well-designed clinical trials.

Common Side Effects

LIMITED HUMAN SAFETY DATA

Limited human safety data
Safety profile not well defined in humans.

Investigational status
Not approved for clinical use.

Evidence Level

MOSTLY PRECLINICAL EVIDENCE

Mainly preclinical/animal evidence with emerging human relevance
Promising metabolic effects in animal studies and early human data, but larger, controlled human trials are needed.

AOD-9604

Mechanism, Physiology, and Clinical Effects

A GROWTH HORMONE FRAGMENT ASSOCIATED WITH FAT METABOLISM



Pharmacokinetics

HUMAN DATA ARE LIMITED

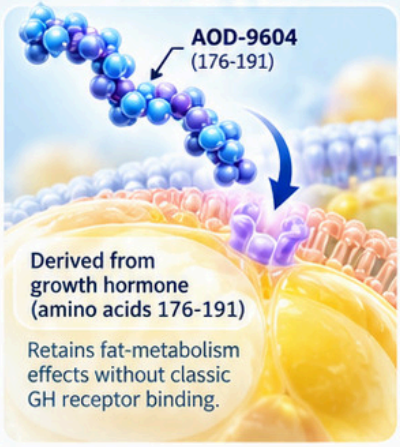
Human pharmacokinetic data are limited
Absorption, half-life, and distribution are not well characterized.

Dosing practices vary
No standardized dosing regimen; protocols differ across studies and settings.

Investigational or non-approved use
AOD-9604 is investigational and not approved for clinical use in many settings.

1 Drug action

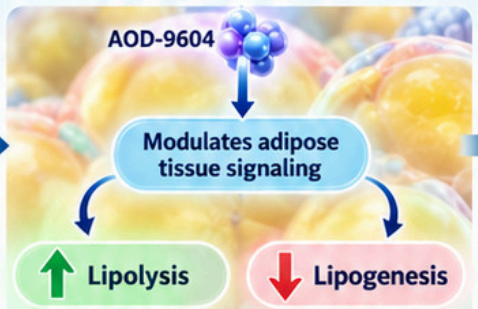
GROWTH HORMONE FRAGMENT 176-191



Retains fat-metabolism effects without classic GH receptor binding.

2 Signaling

PROMOTES LIPOLYSIS AND MAY REDUCE LIPOGENESIS



Increases breakdown of stored triglycerides and release of free fatty acids

May reduce fat storage and adipocyte lipid accumulation

3 Physiologic effects

ADIPOSE-FOCUSED ACTIONS

Increases fat mobilization
Promotes lipolysis and release of free fatty acids from adipose tissue

May reduce fat storage
Potential to decrease lipogenesis and lipid accumulation in adipose tissue

Less emphasis on classic GH-wide effects
Minimal direct anabolic effects on muscle, bone, or IGF-1 compared with GH

4 Clinical outcomes

POTENTIAL FAT-LOSS SUPPORT, BUT HUMAN EVIDENCE IS MIXED

Potential fat loss support
May help reduce body fat, particularly in combination with diet and exercise

Improved body composition
Potential reductions in fat mass with preservation of lean mass

Human efficacy evidence is mixed
Some studies suggest modest benefits, while others show limited or no significant effects

Common Side Effects

LIMITED SAFETY DATA

Injection-site reactions
Redness, swelling, or discomfort

Headache
Reported in some users

Uncertainty around long-term outcomes
Long-term safety data are limited

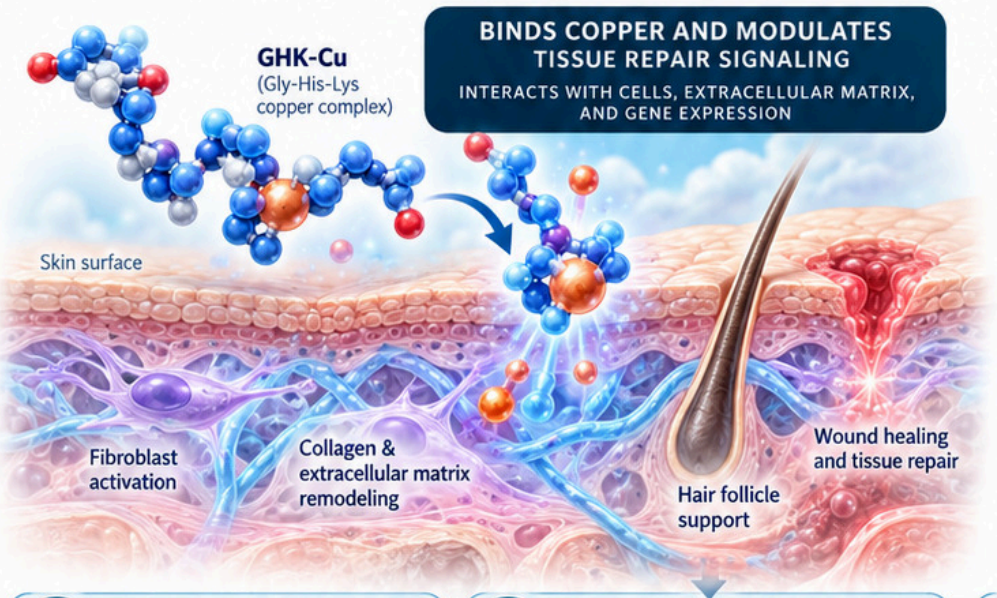
Evidence Level

Limited human evidence; mixed results
Stronger mechanistic and preclinical rationale than definitive clinical proof. More high-quality human studies are needed.

GHK-Cu

Mechanism, Physiology, and Clinical Effects

A COPPER-BINDING TRIPEPTIDE ASSOCIATED WITH REPAIR, SKIN, AND HAIR BIOLOGY



Pharmacokinetics

USE PATTERNS AND CURRENT KNOWLEDGE



Topical use
Commonly used in skincare and cosmetic formulations



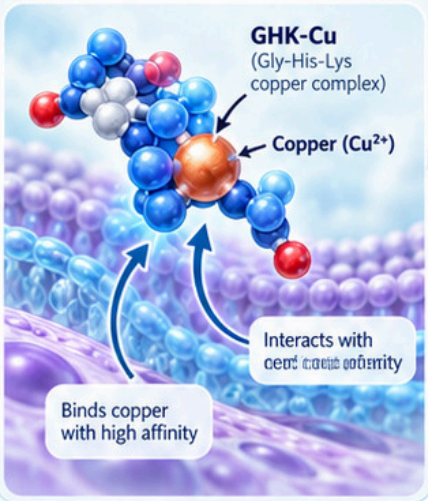
Injectable use
Subcutaneous or intradermal use patterns exist (in research and clinical settings)



Systemic data
Human pharmacokinetic data are limited, including absorption, distribution, metabolism, and half-life

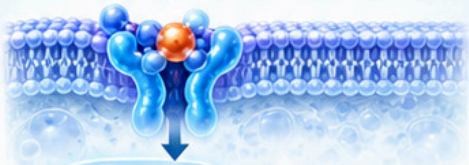
1 Drug binding/action

COPPER-BINDING TRIPEPTIDE INTERACTS WITH TISSUE REPAIR BIOLOGY



2 Intracellular signaling

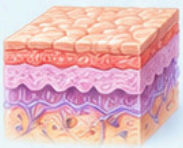
INFLUENCES REPAIR, ECM REMODELING, AND ANTIOXIDANT DEFENSES



- **Gene expression** → Upregulates wound-healing genes (e.g., collagen, MMPs, TIMPs)
- **Extracellular matrix remodeling** → Increases collagen and elastin synthesis; Supports ECM integrity
- **Antioxidant defense** → Enhances cellular antioxidant enzymes; Reduces oxidative stress

3 Physiologic effects

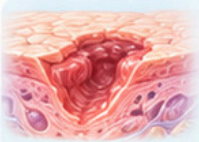
SUPPORTS SKIN, HAIR, AND WOUND-HEALING BIOLOGY



Skin
- ↑ Collagen production
- ↑ Skin elasticity
- ↑ Skin barrier function
- ↑ Improved skin appearance



Hair follicle
- ↑ Supports hair follicle health
- ↑ May promote hair growth environment
- ↑ Strengthens hair quality



Wound healing
- ↑ Accelerates tissue repair
- ↑ Supports re-epithelialization
- ↑ Reduces inflammation in wound environment

4 Clinical outcomes

POTENTIAL BENEFITS FOR SKIN QUALITY, RECOVERY, AND HAIR-SUPPORT APPLICATIONS



Skin quality and appearance
Improved skin tone, elasticity, and texture (primarily from topical and cosmetic use)



Faster recovery
Supports wound healing and skin repair (in clinical and preclinical studies)



Hair support applications
May support hair density and quality (limited human data)

While preclinical and early human studies are promising, definitive clinical outcome data in large, well-controlled trials are still limited.

Common Side Effects

MOSTLY LOCAL; SYSTEMIC DATA ARE LIMITED



Local irritation
Redness, itching, or stinging (especially topical)



Sensitivity reactions
Allergic contact dermatitis (rare)



Limited systemic data
Long-term safety data in humans are limited

Evidence Level



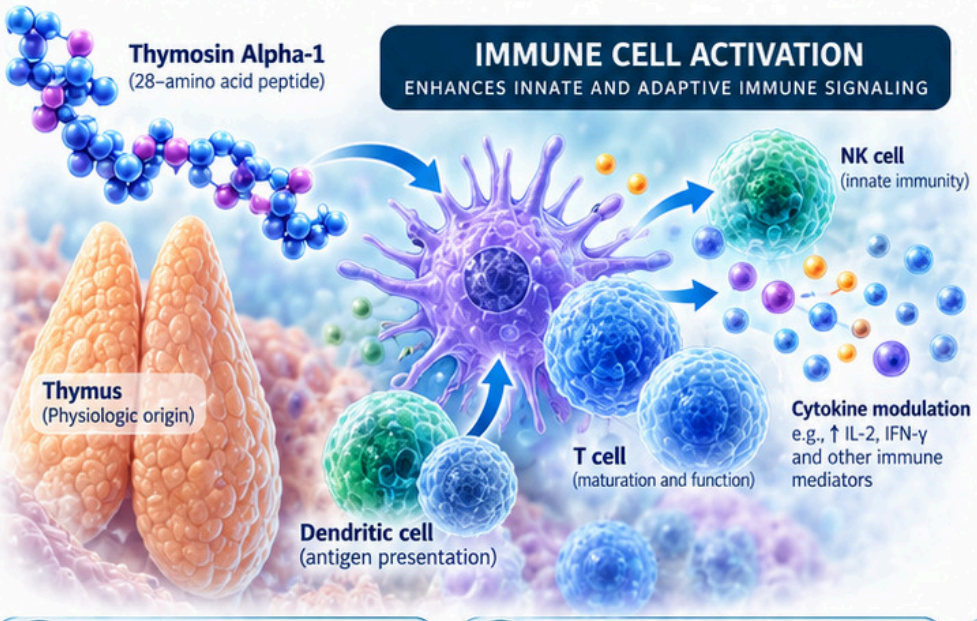
Preclinical evidence plus limited human and cosmetic-use evidence

Strong preclinical and mechanistic evidence, with limited human clinical data and widespread use in cosmetic formulations.

Thymosin Alpha-1

Mechanism, Physiology, and Clinical Effects

AN IMMUNOMODULATORY PEPTIDE AFFECTING INNATE AND ADAPTIVE IMMUNITY



Pharmacokinetics

PREDICTABLE, SHORT EXPOSURE WITH LASTING IMMUNE EFFECTS



Subcutaneous dosing
Typically 0.8–1.6 mg once or twice weekly
(regimen varies by indication)



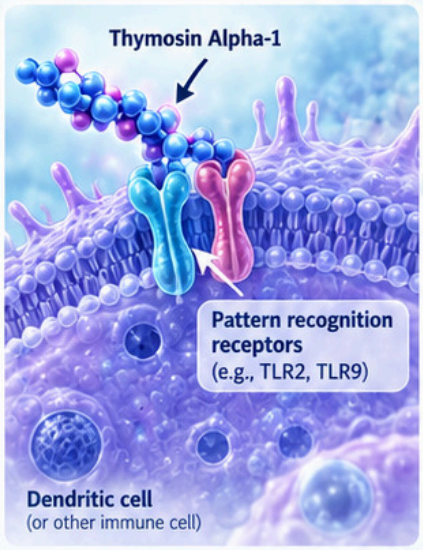
Short plasma half-life
~ 1–4 hours
(rapid clearance)



Biologic immune effects extend beyond measured exposure
Immune modulation persists after clearance due to downstream effects

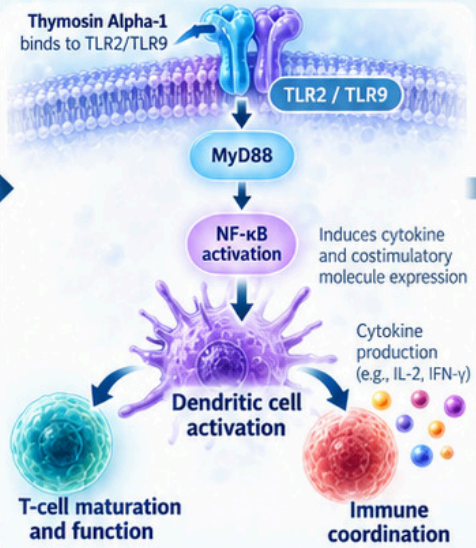
1 Drug action

AN IMMUNOMODULATORY PEPTIDE



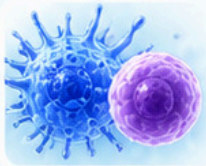
2 Intracellular signaling

ACTIVATES TLR-RELATED PATHWAYS

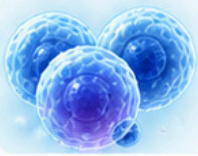


3 Physiologic effects

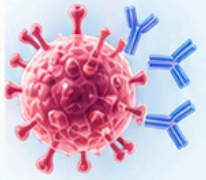
ENHANCES IMMUNE SYSTEM FUNCTION



Improved innate immune signaling
- ↑ Dendritic cell activity
- ↑ NK cell function
- ↑ Cytokine production (e.g., IL-2, IFN-γ)



Supports adaptive immune immunity
- ↑ T-cell maturation
- ↑ Th1 immune responses
- ↑ Antigen-specific immunity



Antiviral and immune-support effects
- Enhances host defense against viral infections
- May improve immune response in selected clinical contexts

4 Clinical outcomes

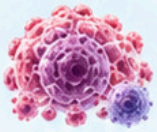
IMMUNE-SUPPORT APPLICATIONS IN SELECTED CLINICAL CONTEXTS



Support in viral infections
Adjunctive use in chronic viral infections (e.g., hepatitis B/C, HIV) in some studies



Immune support in respiratory illness
May enhance immune responses in certain respiratory infections (evidence varies)



Adjunct in oncology care
Used in some settings to support immune function during cancer therapy (indication-specific evidence varies)

Common Side Effects

GENERALLY WELL TOLERATED

Injection-site reactions
Redness, swelling, or discomfort

Fatigue
Usually mild and transient

Mild flu-like symptoms
Low-grade fever, myalgias

Limited adverse-effect burden overall
Serious side effects are uncommon

Evidence Level

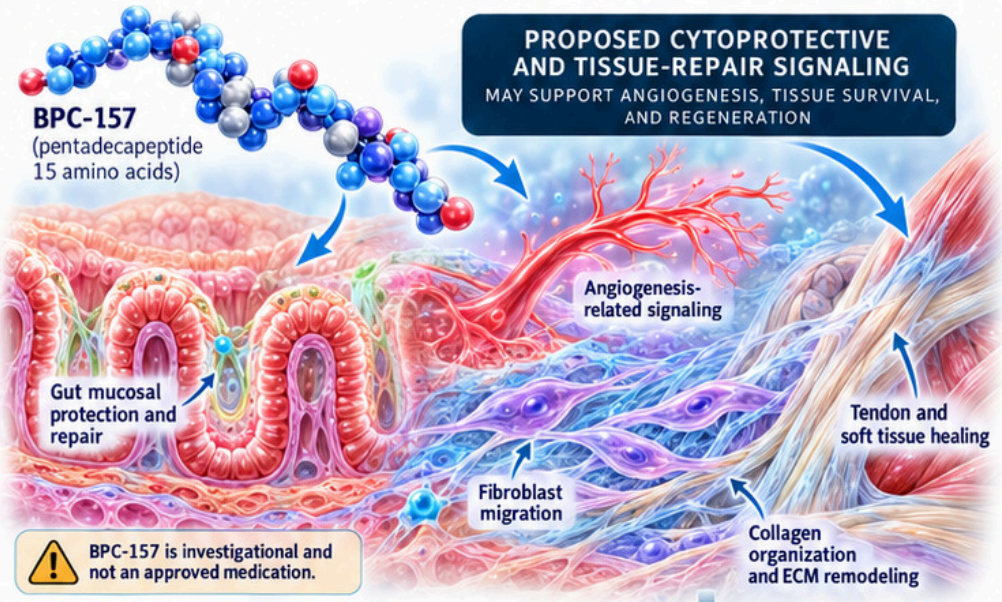
Human clinical evidence exists in selected medical settings
Supportive data in chronic viral infections, some respiratory conditions, and as an adjunct in oncology care.

Broader wellness claims are less established
More high-quality, large-scale studies are needed for general immune enhancement in healthy populations.

BPC-157

Mechanism, Physiology, and Clinical Effects

AN EXPERIMENTAL PENTADECAPEPTIDE ASSOCIATED WITH CYTOPROTECTION AND TISSUE-REPAIR BIOLOGY



Pharmacokinetics

HUMAN PHARMACOKINETICS NOT WELL ESTABLISHED

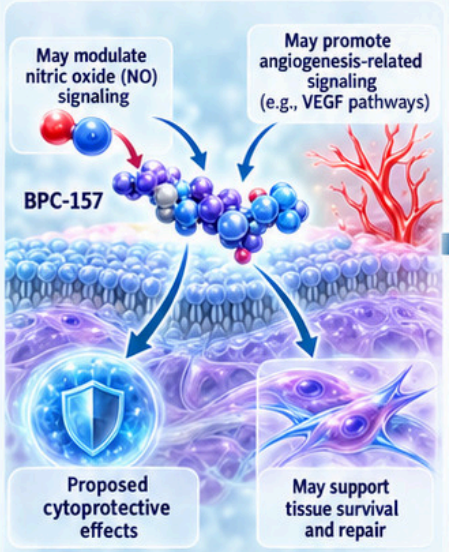
Investigational use
Not approved for medical use in humans (in research and experimental settings).

Human pharmacokinetics not well established
Absorption, distribution, and elimination in humans are not fully characterized.

Limited data
Pharmacokinetic profiles, bioavailability, half-life, and tissue distribution remain unclear.

1 Drug action

PROPOSED MECHANISMS ASSOCIATED WITH TISSUE PROTECTION AND REPAIR



2 Intracellular / tissue signaling

ASSOCIATED PATHWAYS AND BIOLOGIC EFFECTS (BASED ON PRECLINICAL EVIDENCE)



VEGF signaling
- May promote angiogenesis
- Supports new blood vessel formation
- Improves tissue perfusion (in preclinical models)

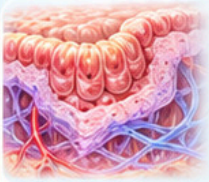
Fibroblast migration
- May enhance fibroblast migration and proliferation
- Supports collagen synthesis
- Aids extracellular matrix remodeling

Epithelial restitution
- Supports re-epithelialization
- Helps maintain mucosal barrier integrity
- May reduce intestinal permeability (preclinical)

Anti-inflammatory effects
- May downregulate pro-inflammatory cytokines
- Modulates inflammatory signaling pathways
- Supports tissue homeostasis

3 Physiologic effects

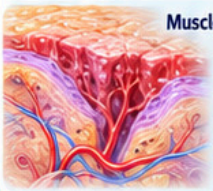
SUPPORTS TISSUE REPAIR, PROTECTION, AND REGENERATION (MULTI-TISSUE)



Gastrointestinal support
- Supports gastric and intestinal mucosal healing
- May reduce mucosal injury (e.g., ulcers, inflammation)
- Helps maintain barrier integrity and gut lining health



Tendon, ligament, and soft tissue support
- Supports tendon and ligament healing
- May improve tissue strength and organization
- Supports musculoskeletal recovery (preclinical)



Muscle and wound-healing support
- May enhance wound closure
- Supports muscle regeneration
- Promotes angiogenesis and tissue remodeling
- May reduce scar formation (preclinical)

4 Clinical outcomes

POTENTIAL BENEFITS BASED ON PRECLINICAL EVIDENCE (HUMAN DATA LIMITED)

Tissue recovery support
May help support recovery from soft tissue, tendon, ligament, and muscle injuries (in preclinical studies).

Gastrointestinal support
May support healing of mucosal injury and gastrointestinal health (in preclinical studies).

Broad tissue protection
May support cytoprotection, angiogenesis, and repair across multiple tissues (preclinical evidence).

High-quality human clinical studies are limited, and definitive evidence of safety and efficacy in humans is not yet established.

Common Side Effects

LIMITED HUMAN SAFETY DATA

Possible injection-site irritation
Redness, pain, or swelling (reported anecdotally)

Gastrointestinal effects (rare)
Nausea or digestive discomfort (reported anecdotally)

Long-term risks unknown
Limited human data; long-term safety effects are not well characterized.

Evidence Level

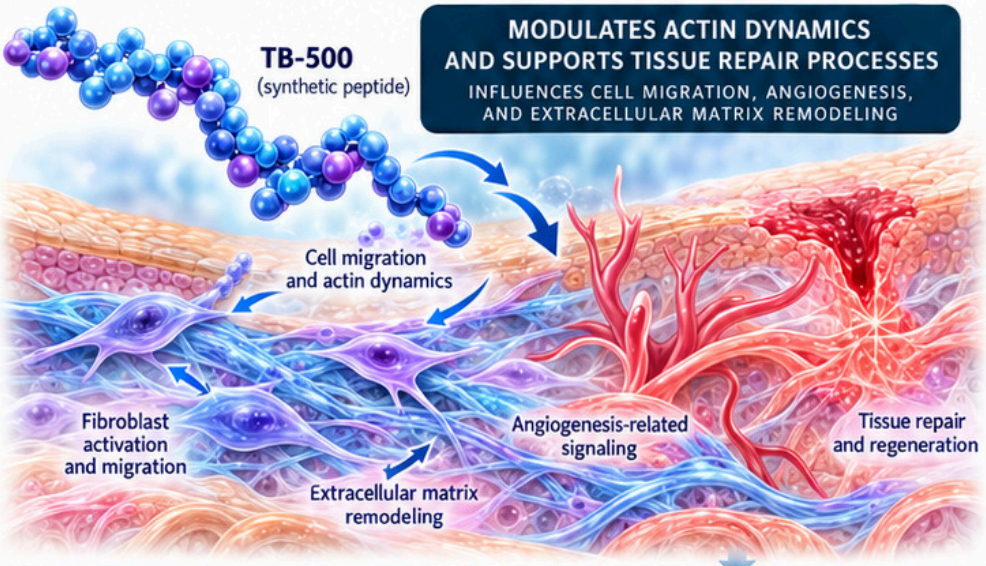
PREDOMINANTLY PRECLINICAL EVIDENCE

Strong preclinical and animal evidence; very limited robust human clinical evidence.
Most data are from in vitro and animal studies. Well-controlled human clinical trials are needed to determine safety, optimal dosing, pharmacokinetics, and efficacy.

TB-500

Mechanism, Physiology, and Clinical Effects

A SYNTHETIC PEPTIDE RELATED TO THYMOSIN BETA-4 AND ASSOCIATED WITH TISSUE REMODELING BIOLOGY



MODULATES ACTIN DYNAMICS AND SUPPORTS TISSUE REPAIR PROCESSES

INFLUENCES CELL MIGRATION, ANGIOGENESIS, AND EXTRACELLULAR MATRIX REMODELING

Pharmacokinetics

HUMAN PHARMACOKINETICS NOT WELL ESTABLISHED



Investigational use
Not an approved medication for human use (currently used in research and off-label settings)



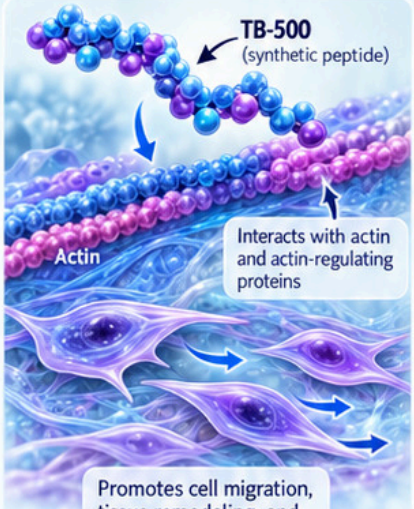
Human PK unknown
Absorption, distribution, metabolism, and half-life have not been well characterized in humans



Limited exposure data
Existing human data are sparse and variable (dose, route, and duration not standardized)

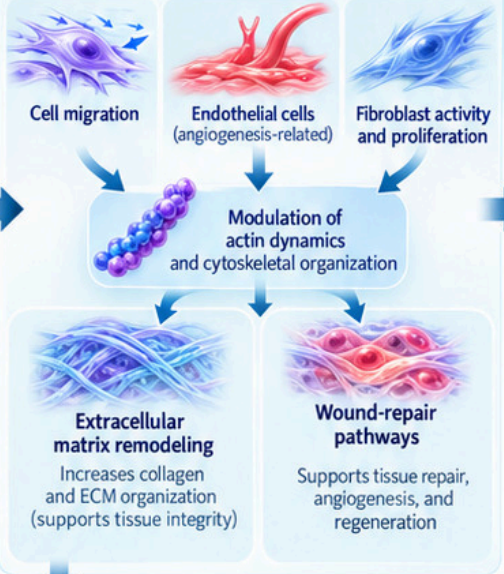
1 Drug action

ASSOCIATED WITH ACTIN BINDING BIOLOGY AND CELL MIGRATION



2 Intracellular / tissue signaling

INFLUENCES CELL MIGRATION, ANGIOGENESIS, AND EXTRACELLULAR MATRIX REMODELING

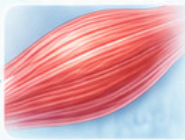


3 Physiologic effects

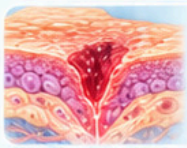
SUPPORTS TISSUE REPAIR AND RECOVERY



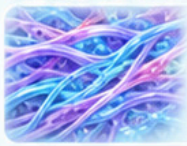
Tendon / ligament recovery
- Supports tendon and ligament healing
- May improve tissue strength and elasticity



Muscle recovery
- Supports muscle repair after injury
- May reduce inflammation and scar tissue formation



Wound-healing support
- Supports re-epithelialization and tissue repair
- May enhance local vascularization



Tissue remodeling
- Supports extracellular matrix organization
- May promote functional recovery of soft tissues

4 Clinical outcomes

POTENTIAL BENEFITS WITH LIMITED HUMAN EVIDENCE



Support recovery after soft tissue injury
May support healing of tendons, ligaments, muscles, and other soft tissues



Improved functional recovery
May help restore mobility, range of motion, and physical function



Clinical efficacy remains uncertain
Human clinical trials are limited, and definitive efficacy has not been established

TB-500 is investigational and not an approved medication for human use.
Clinical evidence is limited, with most data from preclinical and early research studies.

Common Side Effects

LIMITED HUMAN SAFETY DATA



Injection-site irritation
Redness, pain, or swelling



Gastrointestinal symptoms
Nausea or discomfort (rare)



Limited safety data
Long-term risks are unknown



Other potential effects
Full side effect profile is not well established

Evidence Level

PREDOMINANTLY PRECLINICAL EVIDENCE

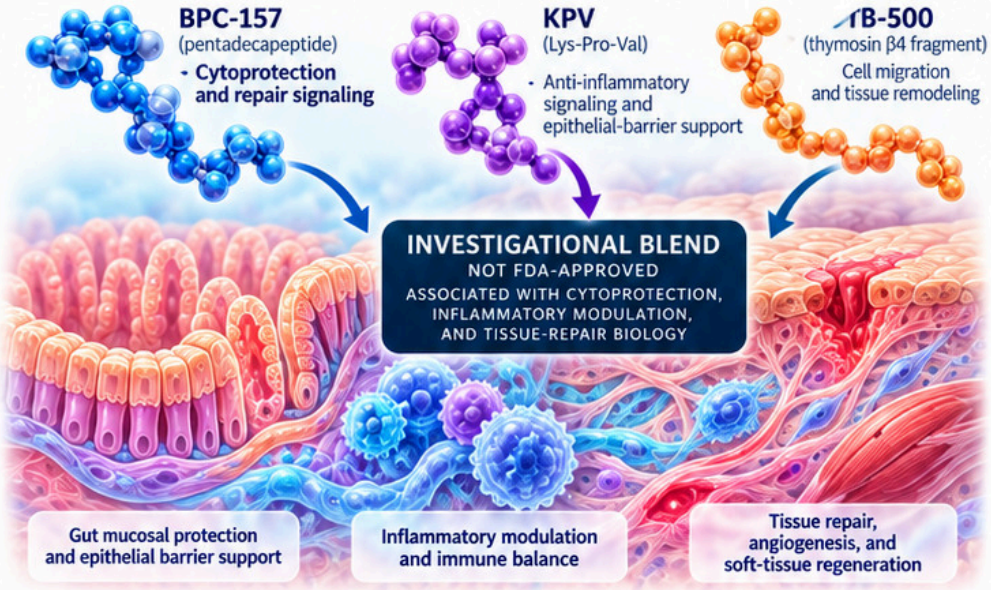


Strong preclinical evidence with limited human data
Most data are from animal and laboratory studies. Human clinical trials are limited, and more research is needed to confirm safety and efficacy.

BPC-157 / KPV / TB-500

Mechanism, Physiology, and Clinical Effects

AN INVESTIGATIONAL BLEND ASSOCIATED WITH CYTOPROTECTION, INFLAMMATORY MODULATION, AND TISSUE-REPAIR BIOLOGY



Pharmacokinetics

HUMAN PK NOT WELL ESTABLISHED



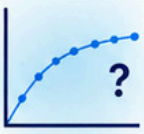
Investigational use

The BPC-157 / KPV / TB-500 blend is not FDA-approved and is used in research and compounding settings.



Human pharmacokinetics

Absorption, distribution, metabolism, and clearance in humans are not well established.



Component-specific data are limited

Exposure and half-life data for each peptide vary by formulation and are based primarily on preclinical studies.

1 Component actions

EACH PEPTIDE CONTRIBUTES DISTINCT BIOLOGICAL ACTIVITIES



BPC-157

- Cytoprotection
- Supports tissue repair
- Gastrointestinal mucosal protection
- Angiogenesis-related signaling



KPV

- Anti-inflammatory signaling (NF-κB modulation)
- Supports epithelial barrier integrity
- May modulate cytokines and immune response

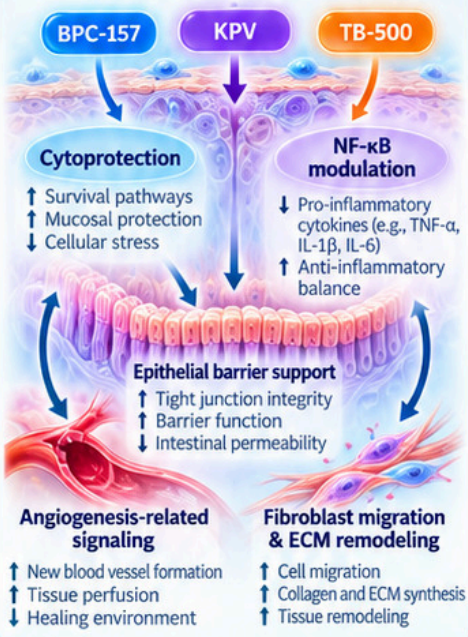


TB-500

- Enhances cell migration and tissue remodeling
- Supports angiogenesis
- Promotes extracellular matrix remodeling
- Associated with soft-tissue repair and recovery

2 Intracellular / tissue signaling

COMPLEMENTARY PATHWAYS SUPPORT REPAIR, PROTECTION, AND BALANCE



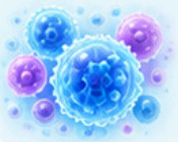
3 Physiologic effects

MULTI-TISSUE ACTIONS



Gut lining support

- ↑ Mucosal protection
- ↑ Barrier integrity
- ↑ Digestive comfort



Inflammatory-balance support

- ↓ Pro-inflammatory cytokines
- ↑ Immune balance
- ↓ Tissue inflammation



Wound-healing & soft-tissue support

- ↑ Tissue repair and regeneration
- ↑ Angiogenesis
- ↑ Collagen and extracellular matrix remodeling



Skin support

- ↑ Wound healing
- ↑ Skin barrier integrity
- ↑ Tissue strength and resilience

4 Clinical outcomes

POTENTIAL BENEFITS BASED ON PRECLINICAL DATA; HUMAN EFFICACY DATA ARE LIMITED



Potential gut comfort/support

May support gastrointestinal mucosal health and epithelial barrier function



Potential recovery support

May aid soft-tissue repair, recovery, and tissue resilience



Potential inflammatory-balance support

May help modulate inflammatory processes and support immune balance

Human clinical evidence for the BPC-157 / KPV / TB-500 blend is limited, with most data from preclinical and early research studies. More high-quality human trials are needed.

Common Side Effects

LIMITED HUMAN SAFETY DATA



Local injection irritation

Redness, soreness, or swelling (depending on formulation)



Gastrointestinal discomfort

Possible nausea or GI upset (reported anecdotally)



Uncertain long-term safety

Long-term safety data in humans are not established

Evidence Level

MOSTLY PRECLINICAL; LIMITED HUMAN EVIDENCE



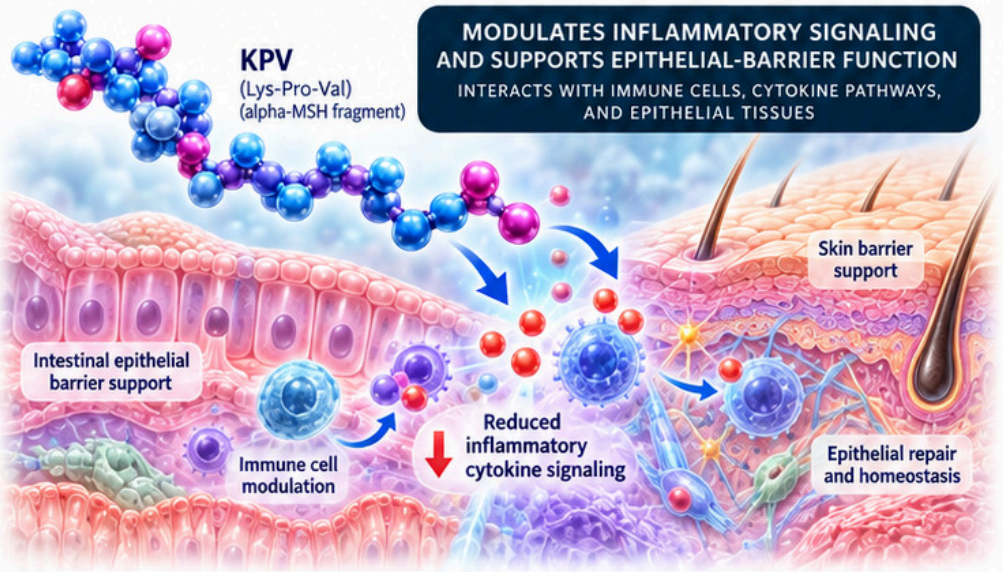
Strong preclinical and mechanistic rationale with limited direct human evidence

Each component has been studied in laboratory and animal models, but well-controlled human trials are limited, especially for the combined blend.

KPV

Mechanism, Physiology, and Clinical Effects

A TRIPEPTIDE DERIVED FROM ALPHA-MSH ASSOCIATED WITH INFLAMMATORY MODULATION AND EPITHELIAL-BARRIER SUPPORT



Pharmacokinetics

HUMAN PK NOT WELL ESTABLISHED



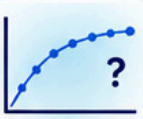
Route-dependent exposure

May be used via subcutaneous, oral, or topical routes (in research settings)



Human PK not well established

Absorption, distribution, metabolism, and half-life are not well characterized in humans

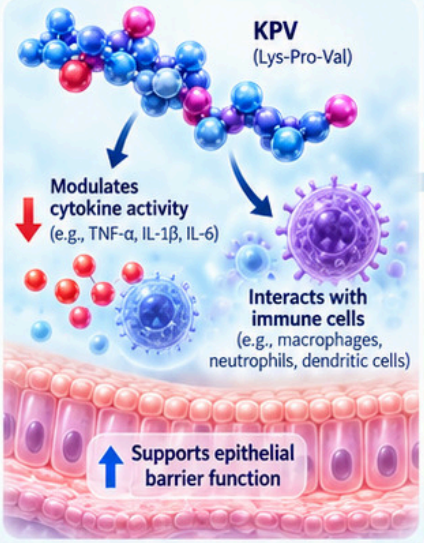


Investigational use

Pharmacokinetic data in humans are limited and variable across routes and formulations

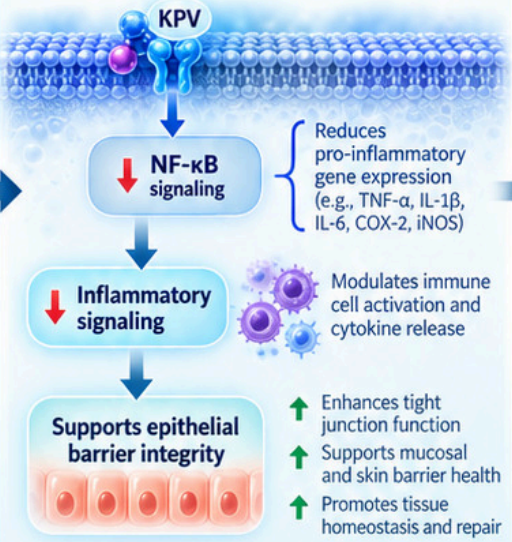
1 Drug action

ANTI-INFLAMMATORY SIGNALING AND EPITHELIAL SUPPORT



2 Intracellular / tissue signaling

MODULATES INFLAMMATORY PATHWAYS AND SUPPORTS BARRIER INTEGRITY



3 Physiologic effects

SUPPORTS GUT AND SKIN BARRIER FUNCTION AND INFLAMMATORY BALANCE



Gastrointestinal tract

- Supports epithelial barrier integrity
- Helps maintain mucosal homeostasis
- May reduce inflammatory signaling in gut tissues



Skin

- Supports skin barrier function
- May help modulate cutaneous inflammation
- Supports skin repair and tissue homeostasis



Inflammatory balance

- Helps down-modulate pro-inflammatory cytokines
- Supports a balanced immune response
- May promote resolution of inflammatory processes

4 Clinical outcomes

POTENTIAL SUPPORT IN INFLAMMATORY GUT OR SKIN SETTINGS (LIMITED DIRECT HUMAN EFFICACY DATA)



Inflammatory gut conditions

May help support barrier function and reduce inflammatory signaling (e.g., IBD)

Preclinical evidence is promising, but human clinical data are limited.



Inflammatory skin conditions

May help reduce inflammation and support barrier repair (e.g., atopic dermatitis, psoriasis, acne)

Potential benefits are based mainly on preclinical and early human data.



Barrier and immune support

May support ongoing tissue homeostasis and a balanced inflammatory response

More high-quality, randomized clinical trials are needed.

Common Side Effects

LIMITED HUMAN SAFETY DATA



Generally well-tolerated in early reports, but safety data are limited.

Long-term safety is not well characterized.

Evidence Level

MAINLY PRECLINICAL EVIDENCE

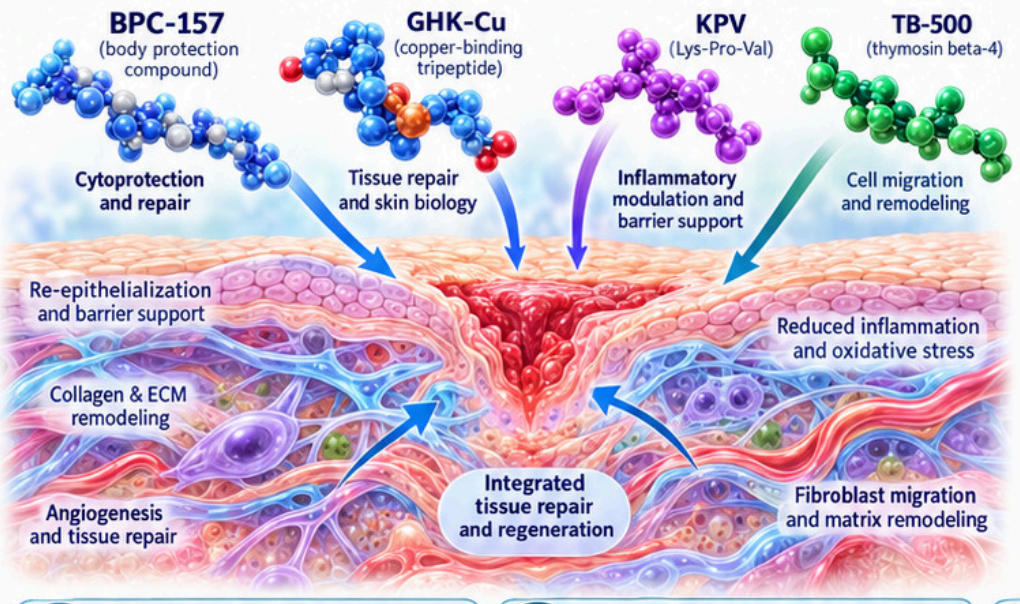
Strong preclinical evidence for anti-inflammatory and barrier-support effects, with limited direct human clinical evidence. More well-designed, randomized clinical trials are needed to establish efficacy and safety.

KPV is an investigational peptide. The information presented is based on preclinical studies and limited human data, and should not be interpreted as established clinical evidence or medical advice.

BPC-157 / GHK-Cu / KPV / TB-500

Mechanism, Physiology, and Clinical Effects

AN INVESTIGATIONAL MULTI-INGREDIENT BLEND ASSOCIATED WITH TISSUE REPAIR, INFLAMMATORY MODULATION, AND SKIN / SOFT-TISSUE SUPPORT



Pharmacokinetics
INCOMPLETELY CHARACTERIZED

Component PK
Individual pharmacokinetics vary and are not fully characterized.

Blend PK
Pharmacokinetics for the combination are not established.

Typical use (investigational)
Commonly used via subcutaneous or topical routes in research and compounding settings.

Systemic data
Human pharmacokinetic data are limited, and long-term effects are unknown.

1 Component actions
EACH INGREDIENT CONTRIBUTES DISTINCT BUT COMPLEMENTARY EFFECTS

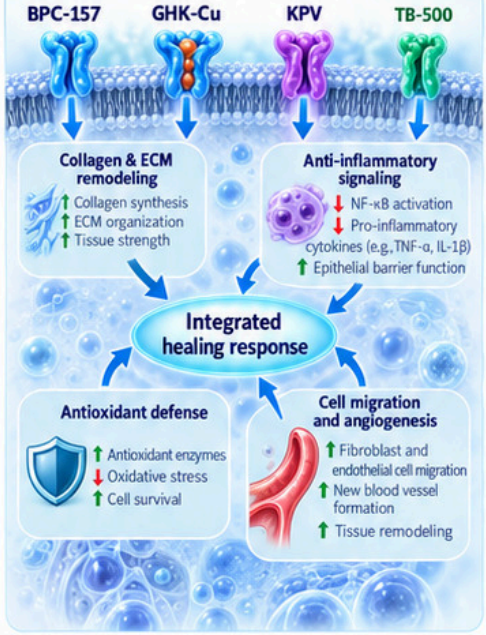
BPC-157
- Cytoprotection
- Enhances tissue repair
- Supports angiogenesis
- Protects gastrointestinal mucosa and epithelial cells

GHK-Cu
- Binds copper
- Stimulates collagen & ECM remodeling
- Supports skin and connective tissue biology
- Modulates gene expression

KPV
- Anti-inflammatory
- Modulates NF-κB signaling
- Supports epithelial barrier function
- Helps regulate immune cell activity

TB-500
- Promotes cell migration and tissue remodeling
- Supports angiogenesis
- Enhances actin cytoskeleton dynamics
- Aids recovery of muscle, tendon, and soft tissue

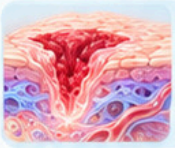
2 Intracellular / tissue signaling
SYNERGISTIC PATHWAYS SUPPORTING REPAIR AND REGENERATION



3 Physiologic effects
MULTI-TISSUE SUPPORT



Skin quality support
- ↑ Collagen and elastin
- ↑ Skin elasticity
- ↑ Barrier function
- ↑ Healthy skin appearance



Wound-healing support
- ↑ Faster re-epithelialization
- ↑ Granulation tissue formation
- ↑ Tissue strength and integrity
- ↓ Inflammation at injury site



Gut / epithelial support
- ↑ Mucosal protection
- ↑ Epithelial barrier integrity
- ↓ Intestinal inflammation
- ↑ GI tissue repair



Soft-tissue recovery support
- ↑ Muscle, tendon, and ligament recovery
- ↑ Tissue remodeling
- ↑ Reduced inflammation
- ↑ Functional recovery

Common Side Effects
MOSTLY LOCAL; SYSTEMIC DATA ARE LIMITED

Local irritation or sensitivity
Redness, swelling, or discomfort (at injection or topical site)

Limited systemic data
Systemic side effects are not well defined for the combination.

Uncertain long-term risks
Long-term safety data for the blend are not available.

4 Clinical outcomes
POTENTIAL BENEFITS BASED ON MECHANISTIC DATA AND LIMITED HUMAN EVIDENCE

Enhanced recovery and repair
May support faster recovery from soft-tissue injury, surgery, or tissue damage

Improved skin appearance
May support skin tone, elasticity, and overall skin health

Gut and mucosal support
May help maintain epithelial barrier function and gastrointestinal comfort

Soft-tissue function
May support muscle, tendon, and ligament recovery, mobility, and function

Evidence Level
MIXED MECHANISTIC AND PRECLINICAL RATIONALE

Promising preclinical evidence, limited direct human data
Each component has mechanistic and preclinical support, but clinical evidence for the combined formulation remains limited. More well-controlled human studies are needed.

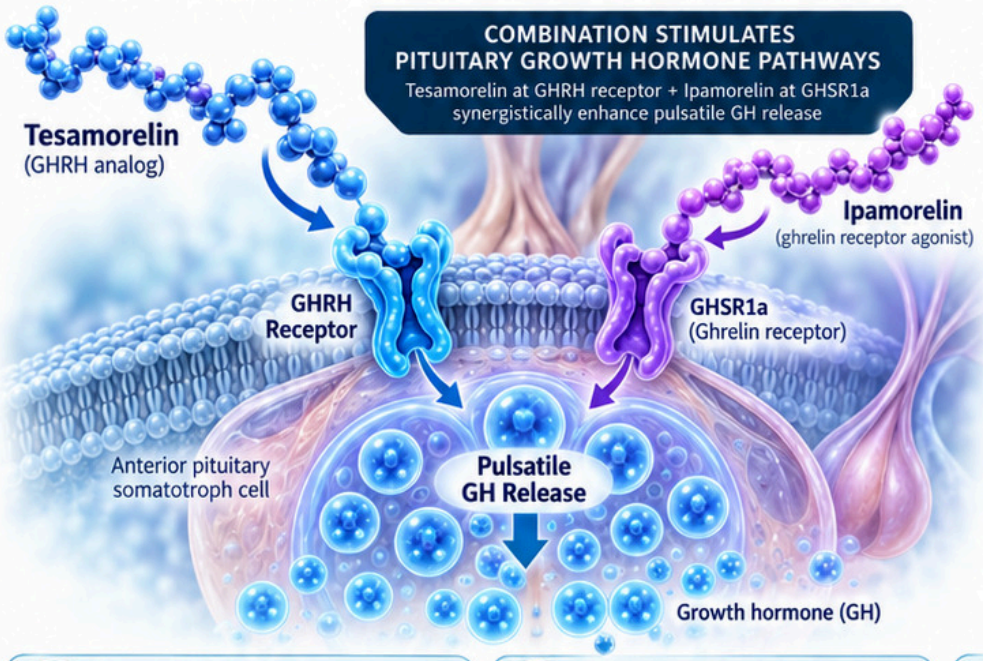
This combination is investigational and not approved by the FDA for any medical use.
The information presented is based on preclinical research, mechanistic data, and limited human studies.

Tesamorelin / Ipamorelin

Mechanism, Physiology, and Clinical Effects

A GHRH ANALOG PLUS GHRELIN-RECEPTOR AGONIST ASSOCIATED WITH GROWTH-HORMONE SIGNALING

COMBINATION STIMULATES PITUITARY GROWTH HORMONE PATHWAYS
Tesamorelin at GHRH receptor + Ipamorelin at GHSR1a synergistically enhance pulsatile GH release



Pharmacokinetics
DISTINCT PROFILES, COMPLEMENTARY EFFECTS

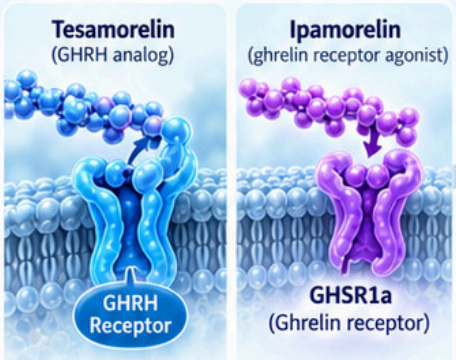
Subcutaneous dosing
Typically once or twice daily (e.g., nightly) protocols vary

Tesamorelin half-life ~ 30–60 minutes
(short plasma half-life); IGF-1 effects persist for hours

Ipamorelin half-life ~ 1–2 hours
(short-acting)

Combination
Used together in some protocols; optimal dosing and long-term data are limited

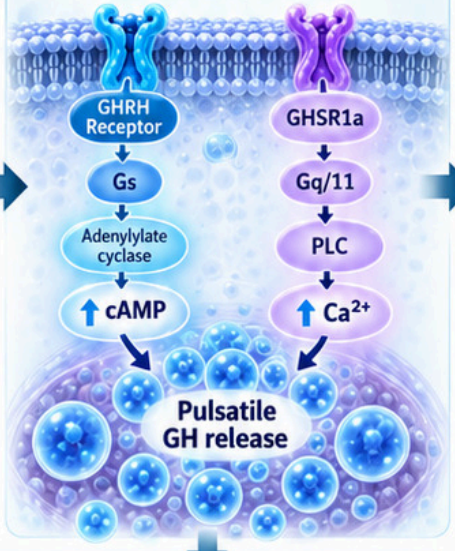
1 Drug binding
EACH PEPTIDE BINDS ITS TARGET ON PITUITARY SOMATOTROPH CELLS



Binds GHRH receptor (similar to endogenous GHRH)

Binds GHSR1a (similar to endogenous ghrelin)

2 Intracellular signaling
ACTIVATES COMPLEMENTARY PATHWAYS THAT STIMULATE GH RELEASE



3 Physiologic effects
MULTI-TISSUE ACTIONS OF GH AND IGF-1

Anterior pituitary
↑ Growth hormone release
More physiologic, pulsatile secretion

Liver
↑ IGF-1 production
Mediates many anabolic effects

Adipose tissue
↑ Lipolysis
Supports improved body composition (reduced visceral fat)

Brain / Sleep
↑ May support sleep quality and recovery
Influences GH neuroregulation

4 Clinical outcomes
POTENTIAL BENEFITS, WITH STRONGER EVIDENCE FOR TESAMORELIN ALONE

Reduced visceral fat
Tesamorelin is FDA-approved for HIV-associated lipodystrophy, with robust RCT evidence for reduced abdominal fat

Improved body composition
May reduce fat mass and increase lean mass, particularly with tesamorelin; data for the combination are limited

Recovery and physical performance
Potential support for recovery, exercise adaptation, and sleep quality (more data needed for combination therapy)

Common Side Effects
MOSTLY MILD AND TRANSIENT

- Injection-site reactions (redness, pain)
- Edema (fluid retention)
- Headache (dizziness)
- Arthralgia (joint pain)
- Changes in glucose metabolism (usually mild)

Evidence Level

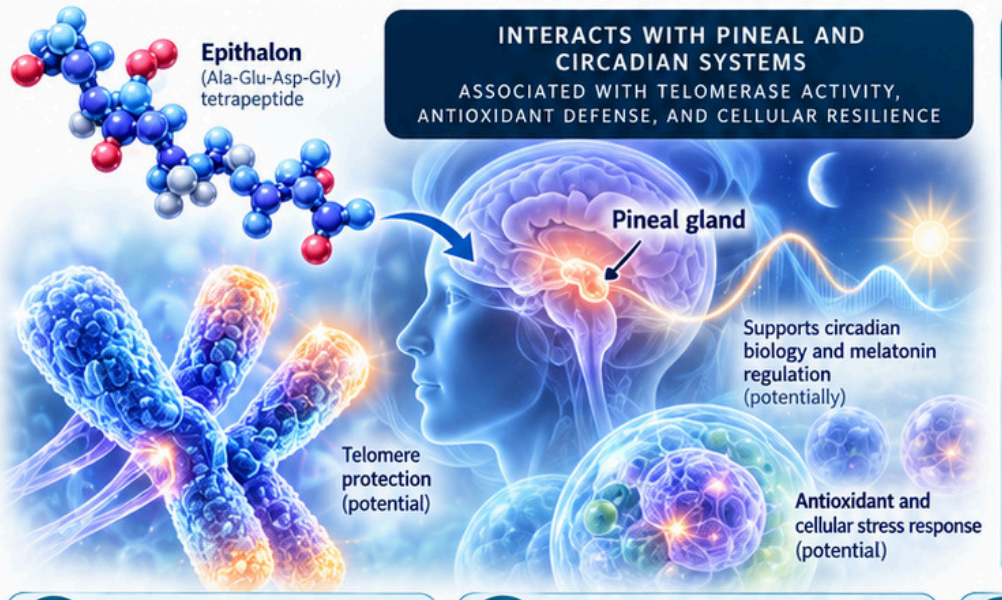
Tesamorelin alone
Strong clinical evidence
Multiple randomized controlled trials (RCTs) show reduced visceral/abdominal fat in HIV-associated lipodystrophy (FDA-approved).

Tesamorelin + Ipamorelin (combination)
↑ Limited clinical evidence
Preclinical and early human data suggest for the combination are lacking.

Epithalon

Mechanism, Physiology, and Clinical Effects

A SYNTHETIC TETRAPEPTIDE ASSOCIATED WITH PINEAL AND HEALTHY-AGING BIOLOGY



Pharmacokinetics

HUMAN PK NOT WELL ESTABLISHED

Route of administration
Most often studied via subcutaneous injection (in research settings)

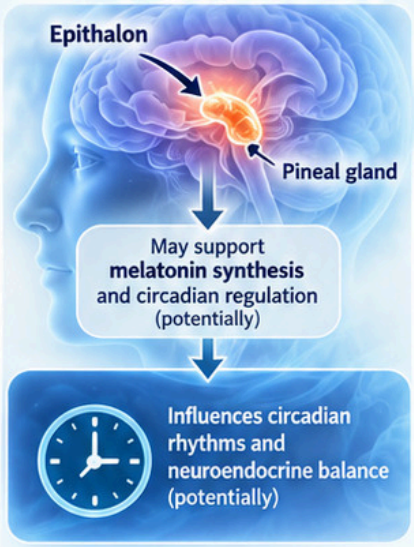
Half-life
Not well established in humans
Likely short (estimated hours), but limited data

Bioavailability
Human pharmacokinetic data are limited

Use status
Investigational compound
Not approved for medical use in most countries

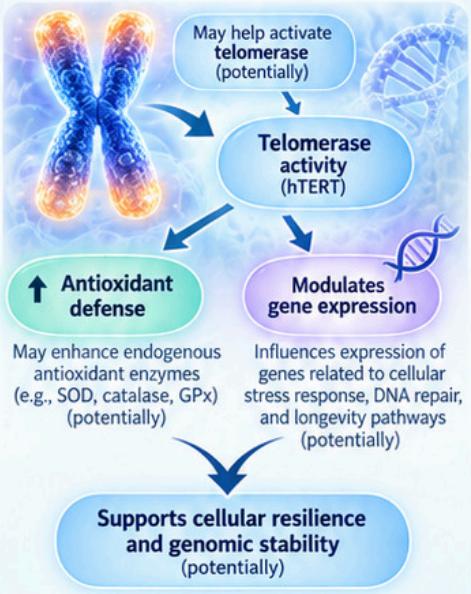
1 Drug action

PROPOSED INTERACTIONS WITH PINEAL AND CIRCADIAN BIOLOGY



2 Intracellular signaling

PROPOSED MECHANISMS BASED ON PRECLINICAL AND EARLY HUMAN DATA



3 Physiologic effects

POTENTIAL MULTI-SYSTEM EFFECTS ASSOCIATED WITH HEALTHY AGING

Circadian system
- May support melatonin production
- May improve sleep quality and circadian rhythm
- May help normalize age-related circadian changes

Cellular longevity
- Associated with telomerase activity (preclinical/early data)
- May support genomic stability and healthy aging processes

Cellular resilience
- May enhance antioxidant defenses
- May reduce oxidative stress and cellular damage

Neuroendocrine balance
- May support overall neuroendocrine health
- Potential benefits for age-related changes in brain function (investigational)

4 Clinical outcomes

POTENTIAL BENEFITS BASED ON PRECLINICAL AND LIMITED HUMAN DATA

Sleep and circadian support
May improve sleep quality and circadian rhythm (limited human data)

Healthy aging support
Potential support for longevity and age-related functional decline (investigational)

Cellular health
May support genomic stability and resilience to cellular stress (potentially)

Cognitive function (possible)
May support age-related cognitive health (very limited human data)

Common Side Effects

LIMITED HUMAN SAFETY DATA

Local injection reactions
Redness, swelling, or discomfort (at injection site)

Other side effects
Not well characterized
Overall safety profile in humans is not established

Evidence Level

MAINLY PRECLINICAL OR LIMITED HUMAN DATA

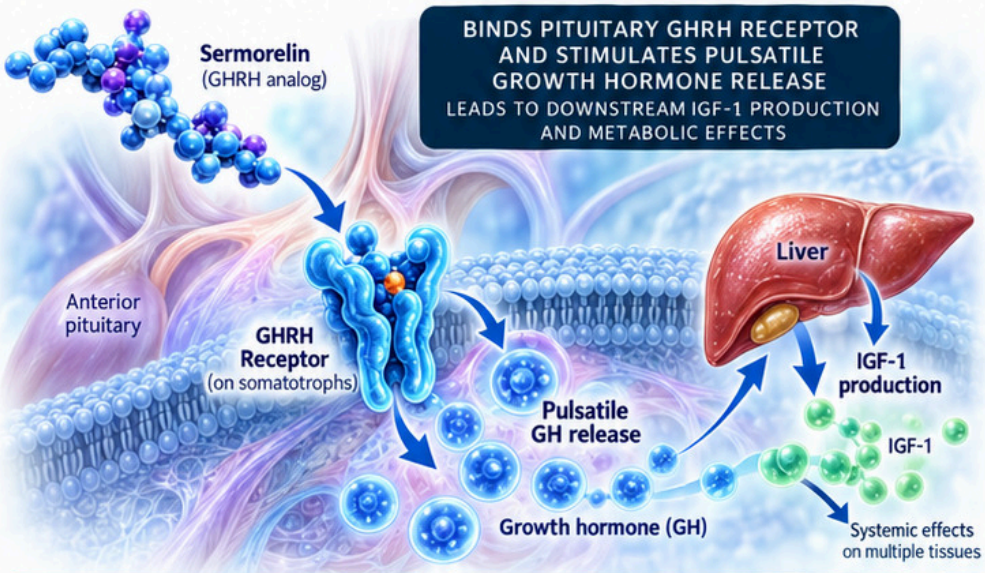
Preclinical studies and small human studies
Suggest potential benefits, but high-quality, large, randomized controlled trials are lacking. Many claims remain investigational and not definitively proven in humans.

Epithalon is an investigational compound. Most of the proposed mechanisms and clinical benefits are based on preclinical studies and limited human data. It is not approved for medical use in most countries, and more high-quality research is needed.

Sermorelin

Mechanism, Physiology, and Clinical Effects

A GROWTH HORMONE–RELEASING HORMONE ANALOG ASSOCIATED WITH PHYSIOLOGIC GH SIGNALING



Pharmacokinetics

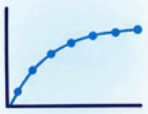
PREDICTABLE, SHORT HALF-LIFE



Subcutaneous dosing
Typically once or twice daily (often at bedtime)



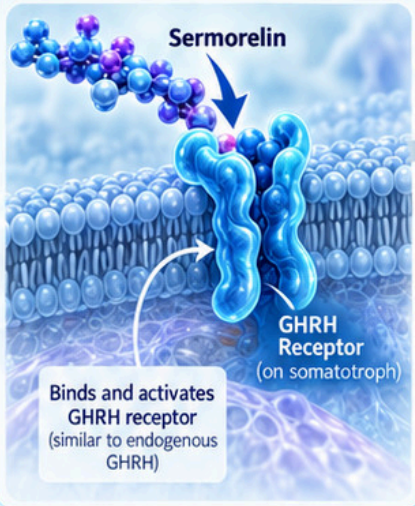
Short half-life
~ 10–30 minutes (varies by study)



Downstream effects persist
Stimulates endogenous GH and IGF-1, which remain elevated beyond direct drug exposure

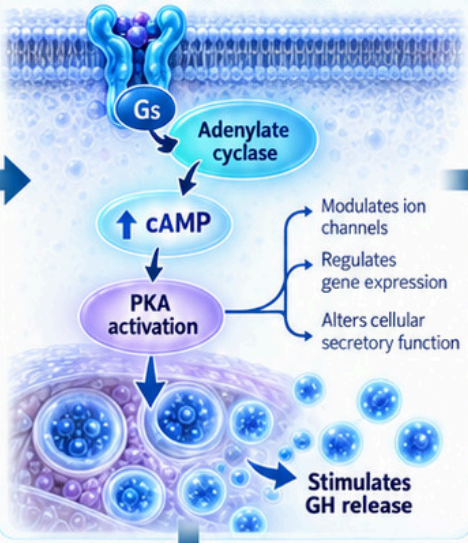
1 Drug binding

SERMORELIN ACTIVATES THE PITUITARY GHRH RECEPTOR ON SOMATOTROPHS



2 Intracellular signaling

ACTIVATES cAMP–PKA PATHWAY LEADING TO GH SECRETION



3 Physiologic effects

INCREASES GH SIGNALING AND DOWNSTREAM IGF-1



Anterior pituitary
↑ Growth hormone release
Restores or enhances pulsatile GH secretion (similar to endogenous GHRH)



Liver
↑ IGF-1 production
Mediates many anabolic and metabolic effects



Body composition
↑ Supports lean mass
May reduce fat mass (in appropriate settings)



Recovery and sleep
↑ May support sleep quality
Supports recovery and overall well-being

4 Clinical outcomes

MAY SUPPORT GH-AXIS FUNCTION AND IGF-1 IN APPROPRIATE SETTINGS



Supports GH-axis function
May increase endogenous GH and IGF-1 levels (in specific populations, e.g., GH deficiency, aging-related decline)



Body composition support
May help support lean mass and reduce fat mass (in conjunction with lifestyle factors)



Sleep and recovery support
May support sleep quality, recovery, and overall well-being through physiologic GH/IGF-1 enhancement

While Sermorelin can increase GH and IGF-1 levels, broader wellness and anti-aging outcomes are supported by limited direct, high-quality clinical evidence.

Common Side Effects

MOSTLY MILD AND TRANSIENT



Injectionsite reactions
Redness, swelling or discomfort



Flushing
Warmth or redness (especially facial)



Headache
Usually mild and transient



Edema
Fluid retention (usually mild)



Glucose changes
May affect glucose metabolism (in some individuals)

Evidence Level

ESTABLISHED PHYSIOLOGY, LIMITED OUTCOME DATA

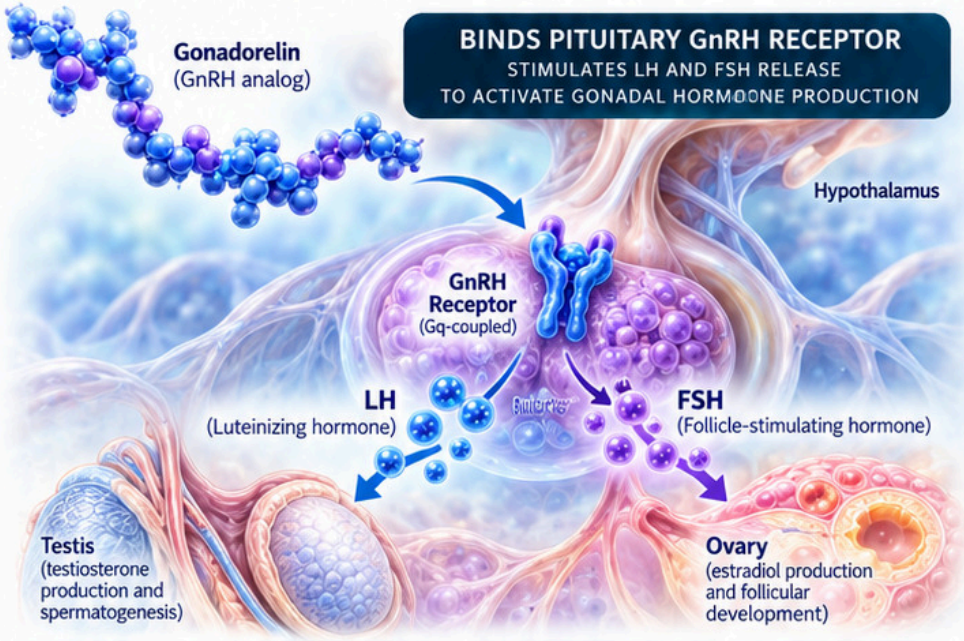


Established physiologic rationale and evidence for GH stimulation
Multiple clinical studies demonstrate increases in GH and IGF-1 levels. However, fewer robust, large-scale trials evaluate broader wellness and anti-aging outcomes.

Gonadorelin

Mechanism, Physiology, and Clinical Effects

A GONADOTROPIN-RELEASING HORMONE ANALOG ASSOCIATED WITH PITUITARY–GONADAL SIGNALING



Pharmacokinetics

SHORT HALF-LIFE, PULSATILE PHYSIOLOGY



Route and dosing
Typically administered subcutaneously or intravenously; dosing varies by indication (e.g., diagnostic or reproductive protocols)



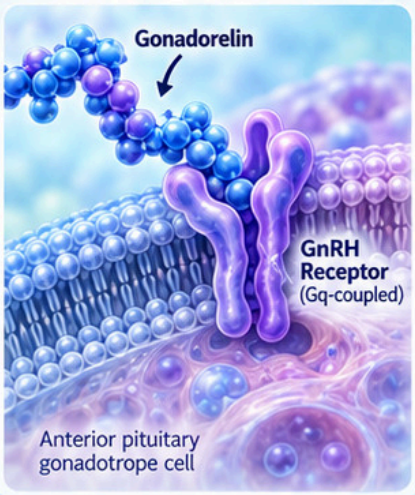
Very short half-life
~ 2–10 minutes
(depending on route)
Rapidly cleared from circulation



Pulsatile physiology
Endogenous GnRH is released in pulses; clinical use often employs pulsatile or intermittent dosing to mimic physiology

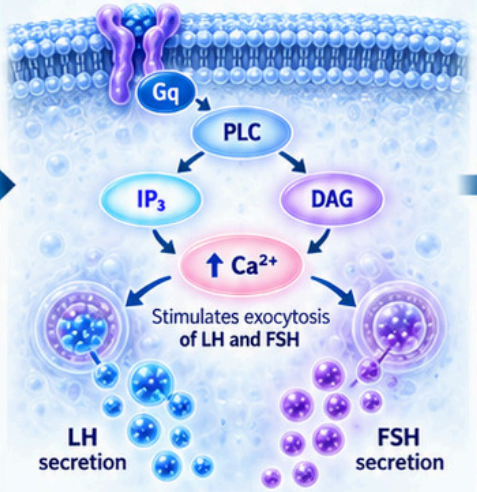
1 Drug binding

GONADORELIN ACTIVATES THE PITUITARY GnRH RECEPTOR



2 Intracellular signaling

ACTIVATES PLC–IP_3–CALCIUM PATHWAY

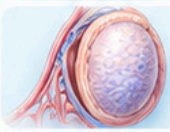


3 Physiologic effects

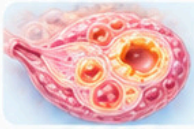
PITUITARY–GONADAL AXIS ACTIVATION



Anterior pituitary
- ↑ LH release
- ↑ FSH release
- ↑ Maintains physiologic pulsatile signaling



Testis (males)
- ↑ Testosterone production
- ↑ Sertoli cell activity
- ↑ Spermatogenesis



Ovary (females)
- ↑ Estradiol production
- ↑ Follicular development
- ↑ Ovulation support



HPG axis
Supports normal hypothalamic–pituitary–gonadal signaling

4 Clinical outcomes

ESTABLISHED DIAGNOSTIC AND REPRODUCTIVE APPLICATIONS



Diagnostic testing
Used in GnRH stimulation tests to assess pituitary LH and FSH reserve



Male reproductive health
Supports evaluation and management of certain forms of hypogonadotropic hypogonadism and infertility



Female reproductive health
Used in fertility protocols (e.g., to induce ovulation or support assisted reproductive techniques)



Potential HPG-axis support
May support normal reproductive hormone signaling in selected clinical contexts; broader wellness use is not well established

Common Side Effects

USUALLY MILD AND TRANSIENT



Evidence Level



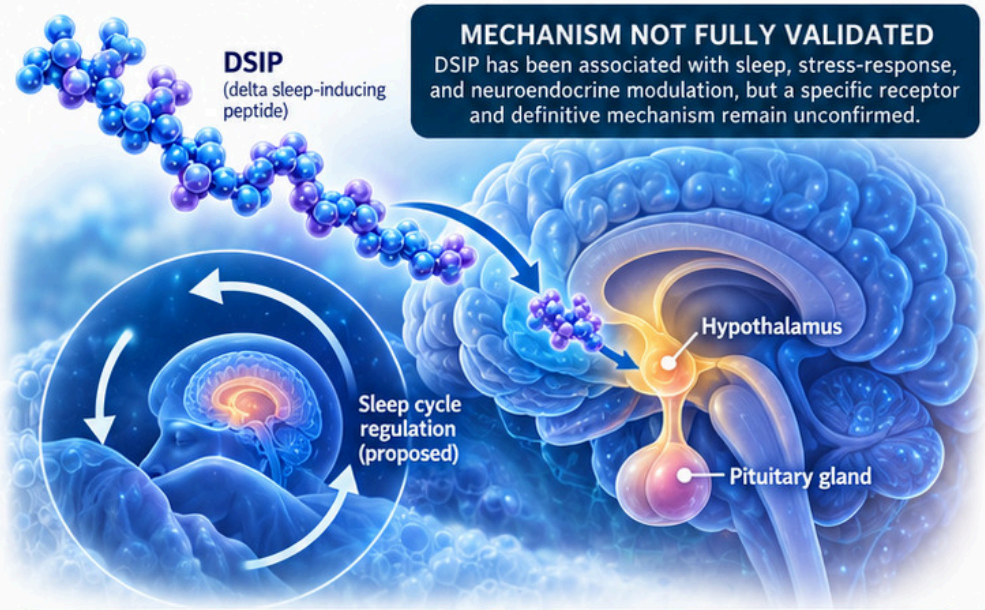
Established endocrine physiology and clinical uses in selected diagnostic and reproductive settings

Strong evidence for pituitary–gonadal effects and clinical utility in specific indications, but limited evidence for generalized wellness or anti-aging use.

DSIP

Mechanism, Physiology, and Clinical Effects

A DELTA SLEEP-INDUCING PEPTIDE ASSOCIATED WITH SLEEP-REGULATION AND NEUROENDOCRINE HYPOTHESES



MECHANISM NOT FULLY VALIDATED

DSIP has been associated with sleep, stress-response, and neuroendocrine modulation, but a specific receptor and definitive mechanism remain unconfirmed.

Pharmacokinetics

HUMAN PK NOT WELL ESTABLISHED



Investigational peptide

Usually used by injection in specialty settings



Human PK limited

Absorption, distribution, metabolism, and clearance in humans are not well established.

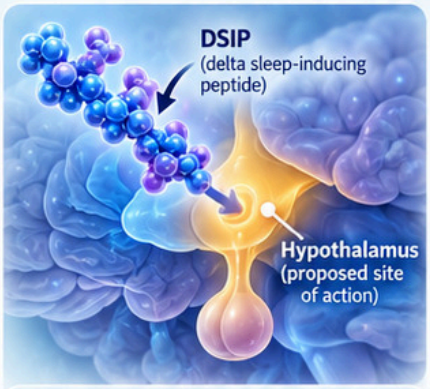


Limited data

Exposure, half-life, and optimal dosing are not well established and may vary by study design and population.

1 Drug action

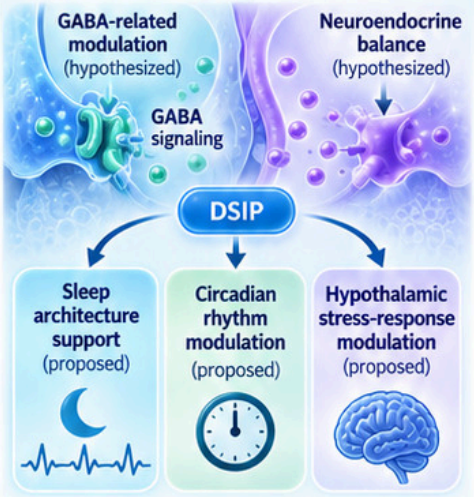
PROPOSED SLEEP-REGULATION AND STRESS-RESPONSE MODULATION



DSIP is proposed to act within the central nervous system to influence sleep-regulation and stress-response pathways, but the specific receptor(s) remain unconfirmed.

2 Intracellular / tissue signaling

HYPOTHESIZED PATHWAYS (UNCONFIRMED)



THESE PATHWAYS ARE HYPOTHETICAL AND NOT FULLY VALIDATED

3 Physiologic effects

POTENTIAL MULTI-SYSTEM EFFECTS



Sleep quality support

- May promote sleep onset
- May improve sleep quality and delta sleep (proposed)
- May support healthy sleep architecture



Stress resilience

- May modulate stress-response systems
- May support a calmer physiologic state



Neuroendocrine balance (possible)

- May influence neuroendocrine signaling (e.g., HPA axis)
- May help maintain hormonal balance (proposed)

4 Clinical outcomes

POTENTIAL BENEFITS, BUT DIRECT HUMAN EFFICACY EVIDENCE IS LIMITED



Potential sleep support

May improve sleep onset, sleep quality, and restorative sleep (proposed)



Potential recovery support

May support better rest and daily functioning through improved sleep (proposed)



Potential stress-resilience

May support adaptation to stress and overall well-being (proposed)

Common Side Effects

MOSTLY TRANSIENT; BASED ON LIMITED DATA



Sedation
Drowsiness



Dizziness
Lightheadedness



Headache
Mild to moderate



Injection-site irritation
Redness, soreness, or swelling

Uncertain long-term safety
Limited data on long-term use in humans

Evidence Level

MAINLY PRECLINICAL OR LIMITED HUMAN STUDIES



Limited human evidence

Mostly preclinical research, with a small number of early human studies. More high-quality, well-controlled clinical trials are needed.

GHK-Cu / Epithalon

Mechanism, Physiology, and Clinical Effects

AN INVESTIGATIONAL COMBINATION ASSOCIATED WITH SKIN / TISSUE BIOLOGY, ANTIOXIDANT DEFENSE, AND HEALTHY-AGING HYPOTHESES



GHK-Cu

(glycyl-L-histidyl-L-lysine copper complex)

- Copper-dependent tissue-remodeling biology
- Skin and wound-healing support (preclinical)
- Antioxidant and gene-modulatory effects



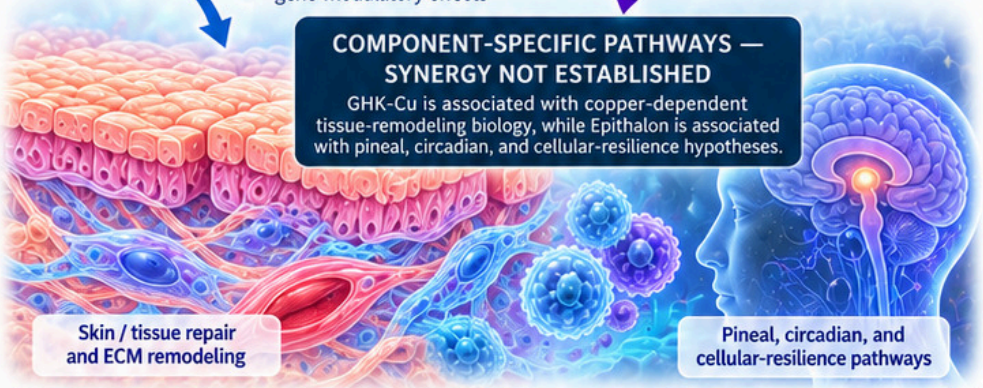
Epithalon

(Ala-Glu-Asp-Gly)

- Pineal and circadian biology hypotheses
- Telomerase-associated activity (preclinical)
- Antioxidant defense and cellular resilience

COMPONENT-SPECIFIC PATHWAYS — SYNERGY NOT ESTABLISHED

GHK-Cu is associated with copper-dependent tissue-remodeling biology, while Epithalon is associated with pineal, circadian, and cellular-resilience hypotheses.



Pharmacokinetics

COMPONENT PK INCOMPLETELY CHARACTERIZED



GHK-Cu

- Often topical or injectable depending on formulation
- Human pharmacokinetics not well established



Epithalon

- Studied mainly as an investigational injectable peptide
- Human pharmacokinetics are limited



Combined formulation

- Pharmacokinetics of the GHK-Cu / Epithalon blend are not established
- Absorption, distribution, metabolism, and clearance in humans are unknown

1 Component actions

EACH PEPTIDE CONTRIBUTES DISTINCT BIOLOGICAL ACTIVITIES



GHK-Cu

- Collagen and ECM remodeling
- Wound-healing and skin biology
- Copper-binding effects
- Antioxidant and gene-modulatory activity

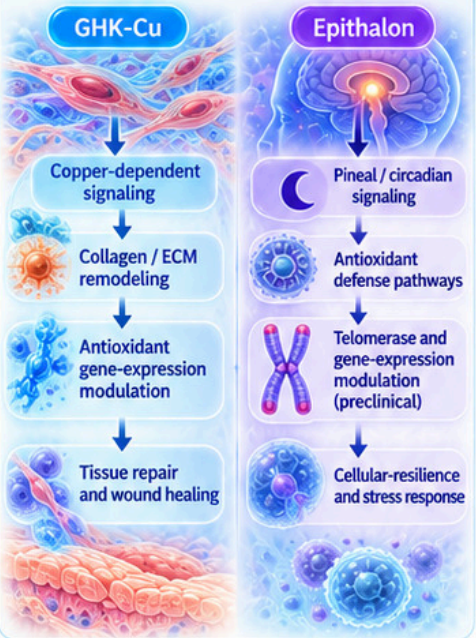


Epithalon

- Circadian and pineal biology hypotheses
- Telomerase-associated activity (preclinical)
- Antioxidant defense
- Cellular resilience and stress response support

2 Intracellular / tissue signaling

COMPLEMENTARY PATHWAYS — SYNERGY IS HYPOTHETICAL



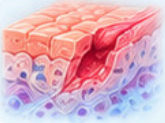
3 Physiologic effects

MULTI-SYSTEM ACTIONS (HYPOTHETICAL)



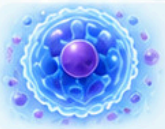
Skin quality support

- ↑ Collagen and ECM remodeling
- ↑ Skin texture and elasticity
- ↑ Skin barrier support



Wound-healing support

- ↑ Tissue repair
- ↑ Epithelial regeneration
- ↑ Reduced inflammation (preclinical)



Cellular-resilience support

- ↑ Antioxidant defense
- ↑ Stress response
- ↑ Healthy cellular function (preclinical)



Circadian / sleep support

- ↑ Pineal and circadian biology
- ↑ Sleep quality (hypothesized)
- ↑ Melatonin regulation (preclinical)



Healthy-aging hypotheses

- ↑ Telomerase-related activity (preclinical)
- ↑ Cellular longevity pathways
- ↑ Overall tissue resilience

4 Clinical outcomes

POTENTIAL BENEFITS BASED ON PRECLINICAL DATA; HUMAN EFFICACY DATA ARE LIMITED



Potential skin and tissue support

May support skin health, wound healing, and tissue repair (primarily preclinical)



Possible sleep or healthy-aging support

May support circadian rhythms, cellular resilience, or healthy aging pathways (hypothesized)



Unclear added benefit of combined formulation

Human clinical evidence for the GHK-Cu / Epithalon combination is limited, and additional high-quality studies are needed

Common Side Effects

LIMITED HUMAN SAFETY DATA



Local irritation

Skin irritation, redness, or sensitivity (especially with topical use)



Incomplete systemic safety data

Long-term safety, optimal dosing, and systemic effects are not well established

Uncertain long-term risks

Potential unknown risks with chronic use or combined formulation

Evidence Level



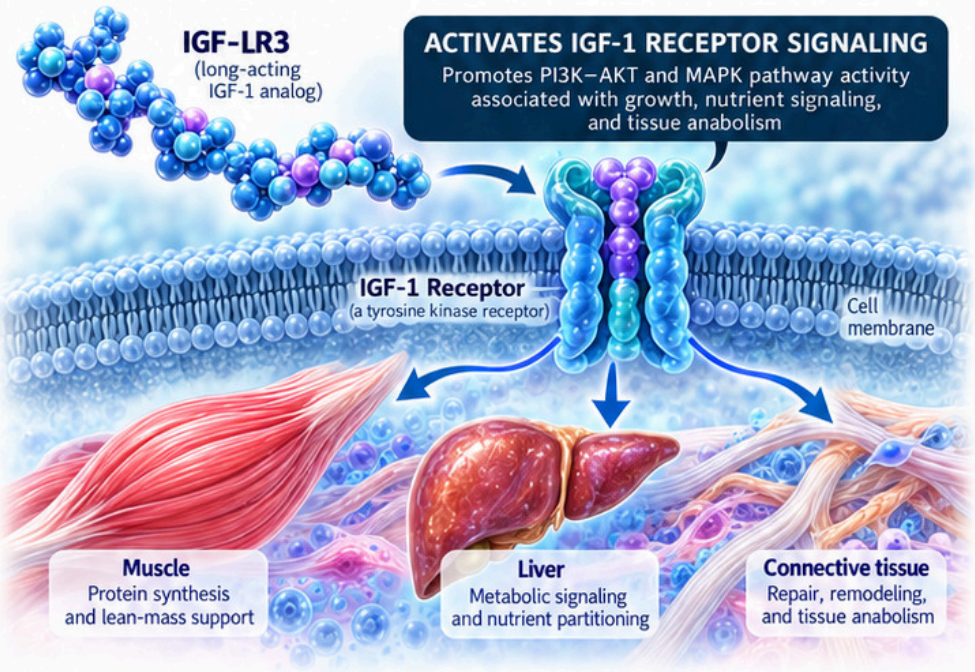
Mixed mechanistic and preclinical rationale

Each component has preclinical and early research support, but direct human clinical evidence for the GHK-Cu / Epithalon combination is limited.

IGF-LR3

Mechanism, Physiology, and Clinical Effects

A LONG-ACTING IGF-1 ANALOG ASSOCIATED WITH IGF-1 RECEPTOR SIGNALING AND ANABOLIC BIOLOGY



Pharmacokinetics

INVESTIGATIONAL; NOT WELL ESTABLISHED



Investigational use
Usually administered subcutaneously in research or off-label settings.



Longer half-life than native IGF-1
Reduced affinity for some IGF binding proteins (IGFBPs) leads to prolonged circulating levels.



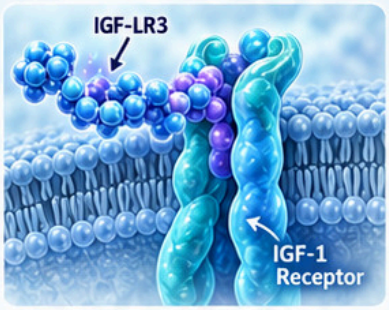
Not well standardized
Human pharmacokinetics and optimal dosing schedules are not well established and may vary by formulation and population.



Systemic metabolic exposure
- Can influence glucose metabolism; risk of hypoglycemia, especially with higher doses or in combination with other agents.

1 Drug binding

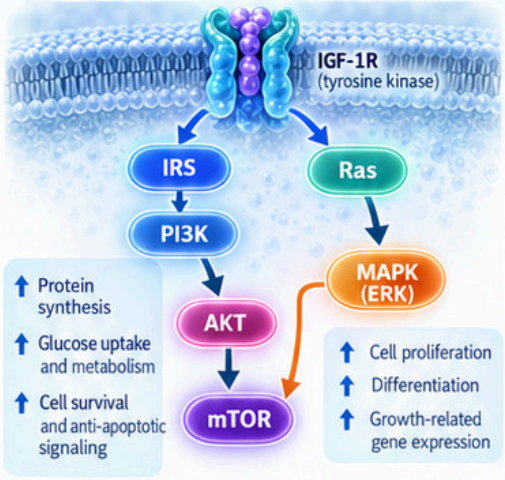
IGF-LR3 BINDS THE IGF-1 RECEPTOR



- IGF-LR3 binds to the IGF-1 receptor with high affinity, similar to native IGF-1.
- Engineered modifications reduce binding to some IGF binding proteins (IGFBPs), increasing free circulating levels and prolonging signaling relative to native IGF-1.

2 Intracellular signaling

ACTIVATES PI3K–AKT AND MAPK PATHWAYS

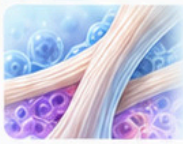


3 Physiologic effects

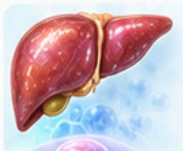
MULTI-TISSUE ACTIONS



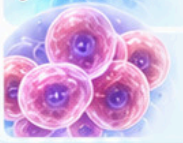
Muscle protein synthesis and lean-mass support
- ↑ Protein synthesis (mTOR)
- ↑ Lean-mass accretion (potential)



Connective tissue and recovery support
- ↑ Collagen synthesis
- ↑ Tendon/ligament repair
- ↑ Exercise recovery (potential)



Metabolic signaling and nutrient partitioning
- ↑ Glucose uptake and utilization
- ↑ Nutrient partitioning to muscle
- ↑ Possible improvements in insulin sensitivity



Possible cell-growth effects
- ↑ Cell survival and proliferation
- ↑ Tissue growth and remodeling in multiple tissues

4 Clinical outcomes

POTENTIAL BENEFITS BASED ON MECHANISM AND LIMITED DATA



Potential lean-mass support
May support increases in lean body mass in research and anecdotal use.



Potential exercise recovery support
May aid recovery from exercise-induced stress and support nitrogen balance.



Potential body-composition support
May support favorable changes in body composition (e.g., lean mass with reduced fat mass).

Clinical efficacy and safety remain uncertain outside of limited research and specialty contexts. High-quality human trials are needed.

Common Side Effects

BASED ON RESEARCH, ANECDOTAL EXPERIENCE, AND CLASS EFFECTS



Hypoglycemia
Low blood sugar, especially at higher doses



Edema
Fluid retention (swelling)



Headache
Headache or dizziness



Jaw or hand tingling
Paresthesia (numbness/tingling)



Injection-site irritation
Redness, soreness, or swelling



Theoretical proliferative risk
Caution in individuals with current or prior malignancy.

Evidence Level

LIMITED DIRECT HUMAN EVIDENCE

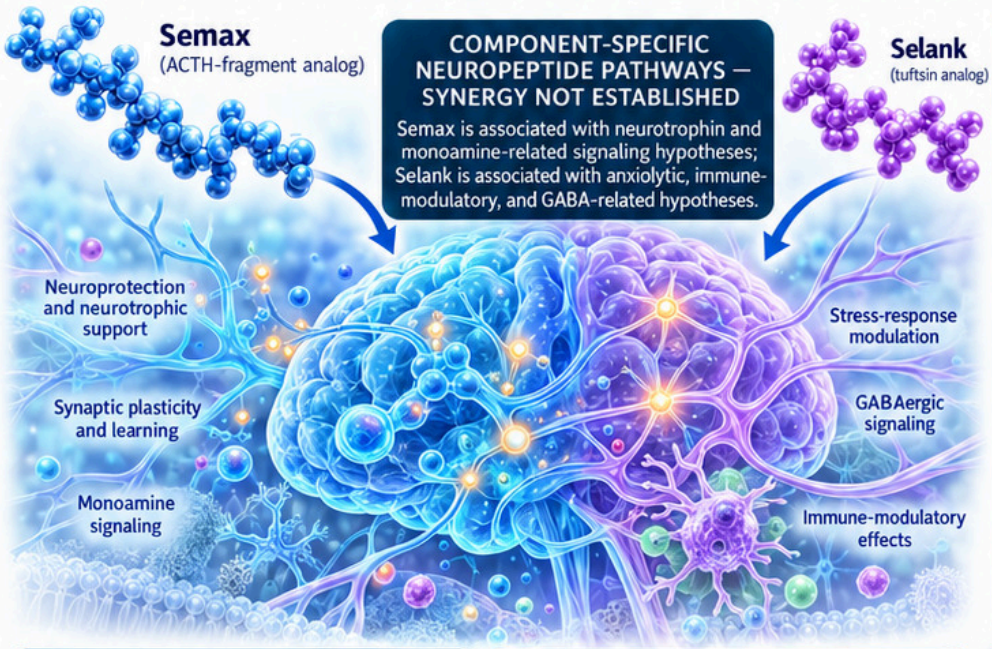


Stronger mechanistic rationale than robust outcome data.
Human clinical evidence for compounded or off-label IGF-LR3 use is limited, with most data from small studies, case reports, or research settings. Well-designed clinical trials are needed to define efficacy and long-term safety.

Semax / Selank

Mechanism, Physiology, and Clinical Effects

AN INVESTIGATIONAL NEUROPEPTIDE COMBINATION ASSOCIATED WITH STRESS-RESPONSE, NEUROTROPHIC, AND COGNITIVE-SUPPORT BIOLOGY



Pharmacokinetics

LIMITED HUMAN DATA; NOT WELL ESTABLISHED

Commonly used intranasally
in research and specialty settings

Human PK incompletely characterized
Absorption, distribution, metabolism, and clearance are not well established.

Onset may be rapid
but duration varies by route and formulation.

Combination PK not established
Pharmacokinetics for Semax + Selank together have not been determined.

1 Component actions

EACH PEPTIDE CONTRIBUTES DISTINCT BIOLOGICAL ACTIVITIES (PROPOSED MECHANISMS)

Semax
(ACTH-fragment analog)

- Proposed BDNF / neurotrophin support
- Monoamine-related signaling hypotheses (e.g., dopamine, serotonin)
- Associated with cognitive-processing and neuroprotection in preclinical studies

Selank
(tuftsin analog)

- Proposed anxiolytic and stress-modulating effects
- Possible GABAergic signaling effects
- Immune-modulatory effects (e.g., cytokine balance)
- Associated with emotional balance and stress resilience in preclinical and limited human studies

2 Intracellular / tissue signaling

COMPLEMENTARY PATHWAYS (PROPOSED) SUPPORT NEURAL FUNCTION AND RESILIENCE

Semax → **Neurotrophin signaling**
↑ BDNF expression and TrkB signaling (proposed)

Selank → **GABAergic and stress-response modulation**
Modulates GABA signaling, HPA axis activity, and stress hormone balance (proposed)

Neurotransmitter balance
Dopamine, serotonin, and other monoamine systems (proposed)

Synaptic plasticity
Supports learning, memory, and neural adaptation (proposed)

Neuroimmune balance
Modulates cytokine signaling and neuroinflammation (proposed)

PROPOSED MECHANISMS — INVESTIGATIONAL

These pathways are based on preclinical research and limited human data. The extent of interaction between Semax and Selank is not established.

3 Physiologic effects

MULTI-SYSTEM ACTIONS (POTENTIAL BENEFITS)

Cognitive support
- ↑ Learning and memory
- ↑ Cognitive processing
- ↑ Mental clarity and focus (investigational)

Stress resilience
- ↑ Stress response modulation
- ↑ Emotional balance
- ↑ Anxiety-related symptoms (investigational)

Mood balance
- ↑ Positive mood and well-being
- ↓ Stress and anxiety
- ↓ Emotional reactivity (investigational)

Attention / mental performance support
- ↑ Attention and concentration
- ↑ Mental performance
- ↑ Resistance to mental fatigue (investigational)

4 Clinical outcomes

POTENTIAL BENEFITS BASED ON LIMITED HUMAN AND PRECLINICAL DATA

Potential cognitive support
Improvements in learning, memory, and cognitive performance in limited studies

Potential anxiolytic support
May reduce stress and anxiety-related symptoms in some studies

Potential neuroprotective and resilience support
May support neural health, stress adaptation, and emotional balance

Efficacy remains uncertain for generalized use. Most data are from preclinical studies and small human trials. More high-quality, large-scale clinical studies are needed.

Common Side Effects

USUALLY MILD AND TRANSIENT (BASED ON AVAILABLE DATA)

Nasal irritation · Headache · Agitation or fatigue · Dizziness · Uncertain long-term safety

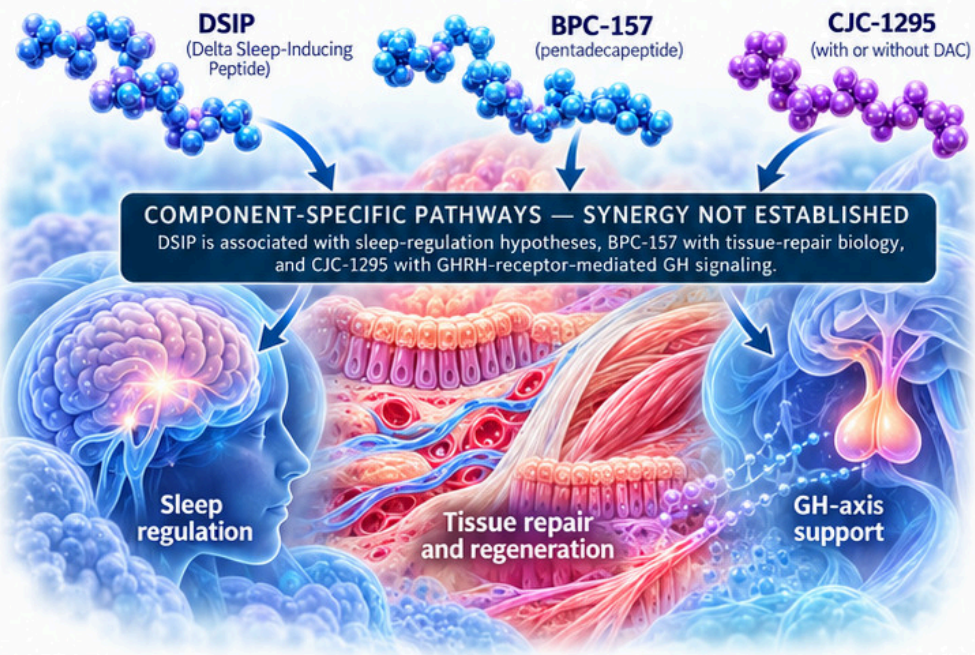
Evidence Level

Limited and heterogeneous human evidence
Preclinical data provide stronger mechanistic and specialty-use rationale. Human studies are limited in size, design, and consistency. Large, robust clinical trials are lacking.

DSIP / BPC-157 / CJC-1295

Mechanism, Physiology, and Clinical Effects

AN INVESTIGATIONAL COMBINATION ASSOCIATED WITH SLEEP-REGULATION, TISSUE-REPAIR, AND GH-AXIS SUPPORT HYPOTHESES



Pharmacokinetics

COMPONENT PK VARIES WIDELY

Component PK varies
Absorption, distribution, metabolism, and clearance differ for each peptide.

DSIP
Human pharmacokinetics are limited and not well established.

BPC-157
Human pharmacokinetics are not well established.

CJC-1295
Duration depends on formulation and may be prolonged with DAC (drug affinity complex).

Blend PK not established
The pharmacokinetics of a DSIP / BPC-157 / CJC-1295 combination have not been studied in humans.

1 Component actions

EACH PEPTIDE CONTRIBUTES DISTINCT BIOLOGICAL ACTIVITIES

DSIP

- Sleep-regulation hypotheses
- May influence neuroendocrine function
- Potential stress and relaxation support

BPC-157

- Cytoprotection
- Supports tissue repair and regeneration
- Gastrointestinal mucosal protection
- Angiogenesis-related signaling

CJC-1295

- GHRH receptor activation
- Stimulates GH release
- Increases IGF-1 (indirectly)
- May support body composition, recovery, and tissue anabolism

2 Intracellular / tissue signaling

COMPONENT-SPECIFIC PATHWAYS
INVESTIGATIONAL AND NOT SYNERGISTIC

DSIP (sleep-regulation)

Modulates neurotransmitter systems (GABAergic, cholinergic, serotonergic)
↓
Influences hypothalamic and pituitary signaling
↓
Sleep-regulation effects

- ↑ Sleep quality
- ↑ Stress resilience
- ↑ Neuroendocrine balance

BPC-157 (tissue-repair)

Modulates cytoprotective and pro-repair pathways (e.g., VEGF, NO, FAK)
↓
Angiogenesis
Fibroblast migration
Epithelial barrier support
↓
Tissue repair and regeneration

- ↑ Blood vessel formation
- ↑ Collagen synthesis
- ↑ Epithelial integrity
- ↑ Tissue remodeling

CJC-1295 (GH-axis support)

Hypothalamus
↓
GHRH receptor activation
↓
Gs
↓
↑ cAMP
↓
↑ GH release (from anterior pituitary)
↓
↑ IGF-1 (in liver and tissues)
↓
GH-axis activation

- ↑ Linear growth signaling
- ↑ Protein synthesis
- ↑ Lean body mass
- ↑ Recovery and repair processes

3 Physiologic effects

MULTI-SYSTEM ACTIONS

Sleep and recovery support

- ↑ Sleep quality and duration
- ↑ Stress resilience
- ↑ Neuroendocrine balance
- ↑ Subjective recovery

Tissue-repair support

- ↑ Mucosal integrity (gastrointestinal)
- ↑ Wound healing and tissue regeneration
- ↑ Angiogenesis and blood flow
- ↑ Collagen and extracellular matrix remodeling

GH-axis / body-composition support

- ↑ GH and IGF-1 levels (indirectly via CJC-1295)
- ↑ Lean body mass
- ↑ Fat mass reduction potential
- ↑ Bone and connective tissue support
- ↑ Recovery and physical performance

4 Clinical outcomes

POTENTIAL BENEFITS BASED ON PRECLINICAL DATA; HUMAN EVIDENCE IS LIMITED

Improved sleep and recovery
Better sleep quality and recovery processes may support overall health.

Tissue recovery and resilience
May support healing, tissue repair, and resilience in injured or stressed tissue.

Body composition and metabolic support
May support lean mass, metabolic health, and recovery during training or catabolic states.

While each component has demonstrated potential benefits individually, robust direct clinical evidence for the combined use of DSIP / BPC-157 / CJC-1295 is lacking.

Common Side Effects

BASED ON HUMAN USE OF INDIVIDUAL COMPONENTS

- **Injection-site irritation** — Redness, soreness, or swelling
- **Headache** — Mild to moderate (headache.
- **Glucose changes** — Altered glucose tolerance (possibly with CJC-1295)
- **Edema** — Fluid retention (possibly with CJC-1295)
- **Sedation or vivid dreams** — (especially with DSIP)
- **Uncertain long-term safety** — Long-term effects are not well established

Evidence Level

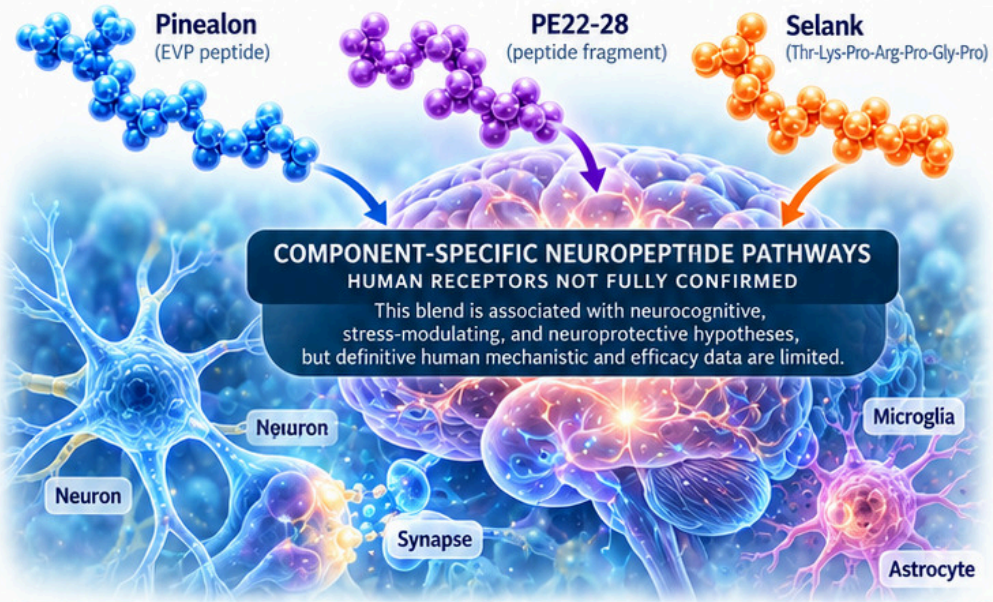
MIXED MECHANISTIC AND PRECLINICAL RATIONALE

Limited direct human evidence for the combination
Each component has mechanistic and preclinical support, but well-designed human studies of the DSIP / BPC-157 / CJC-1295 combination are lacking.

Pinealon / PE22-28 / Selank

Mechanism, Physiology, and Clinical Effects

AN INVESTIGATIONAL NEUROPEPTIDE BLEND ASSOCIATED WITH NEUROREGULATION, STRESS-RESPONSE, AND COGNITIVE-SUPPORT HYPOTHESES



Pharmacokinetics

HUMAN PK NOT WELL ESTABLISHED



Often used intranasally or by injection
In specialty settings



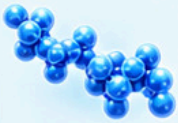
Human PK data for each component are limited
Absorption, distribution, metabolism, and clearance not well defined



Blend PK not established
Exposure and half-life may vary by formulation, dose, and route of administration

1 Component actions

EACH PEPTIDE CONTRIBUTES DISTINCT PROPOSED BIOLOGICAL ACTIVITIES



Pinealon

- Proposed neuroprotective and antioxidant effects
- May influence gene expression and neuronal resilience
- Often studied for aging-related neurobiology



PE22-28

- Experimental neuropeptide fragment
- Associated with cognition and neuronal signaling hypotheses
- May influence synaptic plasticity and neurite growth (preclinical data)



Selank

- Stress and anxiolytic neuroimmune modulation
- May regulate GABAergic, dopaminergic, and serotonergic signaling
- Associated with reduced anxiety-like behaviors (preclinical and regional clinical studies)

2 Intracellular / tissue signaling

PROPOSED PATHWAYS SUPPORT NEUROREGULATION AND BRAIN HOMEOSTASIS



Potential mechanistic effects

- Modulates neurotransmitter systems (GABA, dopamine, serotonin)
- Supports BDNF and neurotrophic signaling (preclinical)
- May influence gene expression and cellular stress pathways
- Regulates neuroinflammation (preclinical)

Tissue and network effects

- Enhanced synaptic plasticity and learning-related signaling
- Neuroprotection and oxidative stress reduction
- Modulation of HPA axis and stress-response
- Support for neuronal network function and brain homeostasis

3 Physiologic effects

MULTI-SYSTEM NEUROLOGIC ACTIONS (HYPOTHESIZED)



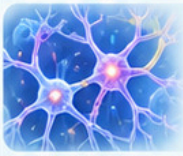
Cognitive support

- ↑ Memory and learning
- ↑ Focus and attention
- ↑ Mental clarity
- ↑ Neuroplasticity (hypothesized)



Emotional / stress balance

- ↑ Stress resilience
- ↑ Reduced anxiety-like responses (preclinical)
- ↑ Emotional well-being



Neuroresilience

- ↑ Neuroprotection
- ↑ Support for healthy neuronal function
- ↑ Adaptive stress response



Sleep or recovery support

- ↑ Sleep quality (hypothesized)
- ↑ Recovery from stress
- ↑ Overall neurocognitive well-being

Common Side Effects

REPORTED IN ANECDOTAL AND LIMITED HUMAN DATA



Nasal irritation



Headache



Dizziness



Fatigue or overstimulation

Uncertain long-term safety

4 Clinical outcomes

POTENTIAL BENEFITS BASED ON PRECLINICAL AND LIMITED HUMAN DATA



Potential memory support
May support learning and memory (preclinical and limited human data)



Potential focus and cognitive support
May improve attention and mental performance (limited human data)



Stress-resilience support
May reduce stress and anxiety-like responses (preclinical and regional human studies)



Overall neurocognitive support
May support brain health, neuroplasticity, and healthy aging (preclinical)

Direct, well-controlled human clinical evidence for the Pinealon / PE22-28 / Selank blend is limited. Most data are from preclinical studies and regional clinical literature. More high-quality human trials are needed.

Evidence Level

MAINLY PRECLINICAL; LIMITED HUMAN EVIDENCE

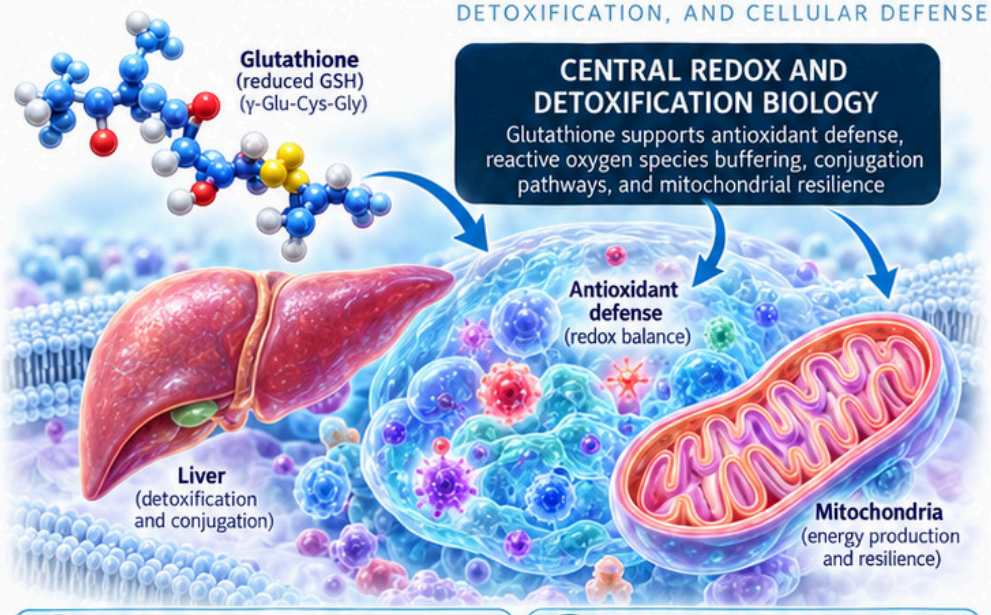


Mechanistic, preclinical, or limited regional human literature
Evidence for the blend is based on preclinical studies and limited clinical data. Large, well-controlled human trials are needed to establish safety and efficacy.

Glutathione

Mechanism, Physiology, and Clinical Effects

A MASTER INTRACELLULAR ANTIOXIDANT ASSOCIATED WITH REDOX BALANCE, DETOXIFICATION, AND CELLULAR DEFENSE

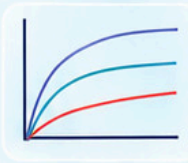


Pharmacokinetics

MULTIPLE ROUTES; VARIABLE EXPOSURE



Can be administered orally, liposomally, intravenously, or by other routes (depending on product)



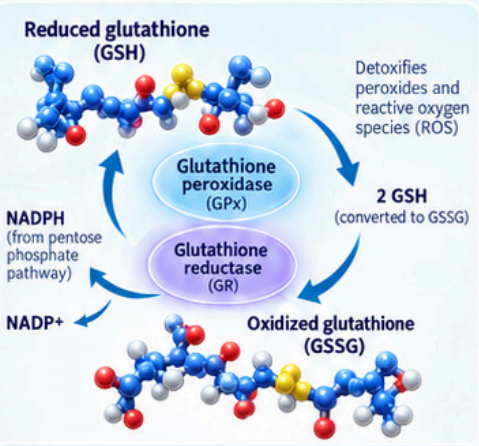
Pharmacokinetics vary by route
IV administration rapidly increases circulating exposure, while intracellular handling is complex.



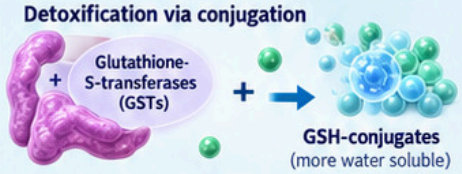
Metabolism and tissue uptake
are route-dependent, with variable half-life and bioavailability.

1 Molecule action

REDOX CYCLING AND DETOXIFICATION



Detoxification via conjugation

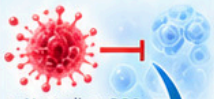


Conjugates toxins, drugs, and endogenous compounds for elimination

2 Intracellular signaling

SUPPORTS MULTIPLE PROTECTIVE PATHWAYS

Antioxidant defense and redox balance



- Neutralizes ROS and maintains redox homeostasis

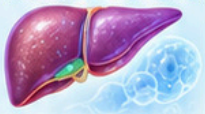
Mitochondrial support



- Reduces oxidative mitochondrial stress
- Supports mitochondrial function and integrity

Glutathione

Phase II detoxification
(Glutathione-S-transferases)



- Conjugates xenobiotics, drugs, and endogenous metabolites
- Supports elimination via bile and urine

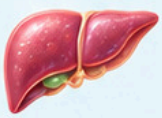
Cellular protection and repair



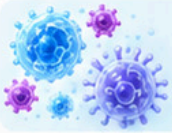
- Supports protein, lipid, and DNA protection
- Helps maintain cellular function and resilience

3 Physiologic effects

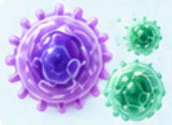
MULTI-ORGAN AND SYSTEMIC ACTIONS



Liver support
- Supports detoxification and conjugation pathways
- Helps manage oxidative stress in the liver



Cellular antioxidant defense
- Neutralizes reactive oxygen species
- Helps maintain redox balance and cellular integrity



Immune support
- Supports immune cell function and resilience
- May modulate inflammatory responses



Skin / pigment and oxidative-stress support
- Helps counteract oxidative stress in skin cells
- Supports skin health and may influence pigmentation balance



Metabolic resilience
- Supports mitochondrial function
- Helps maintain cellular energy and metabolic health

4 Clinical outcomes

POTENTIAL SUPPORT IN OXIDATIVE-STRESS AND RECOVERY CONTEXTS

Oxidative-stress support
May help reduce markers of oxidative stress in specific populations and contexts

Recovery support
May support recovery in settings of increased oxidative stress (e.g., illness, exercise, environmental exposures)

Population-specific uses
Investigated in various conditions, but evidence varies widely by indication, route of administration, and population

Common Side Effects

GENERALLY WELL TOLERATED; USUALLY MILD



Evidence Level

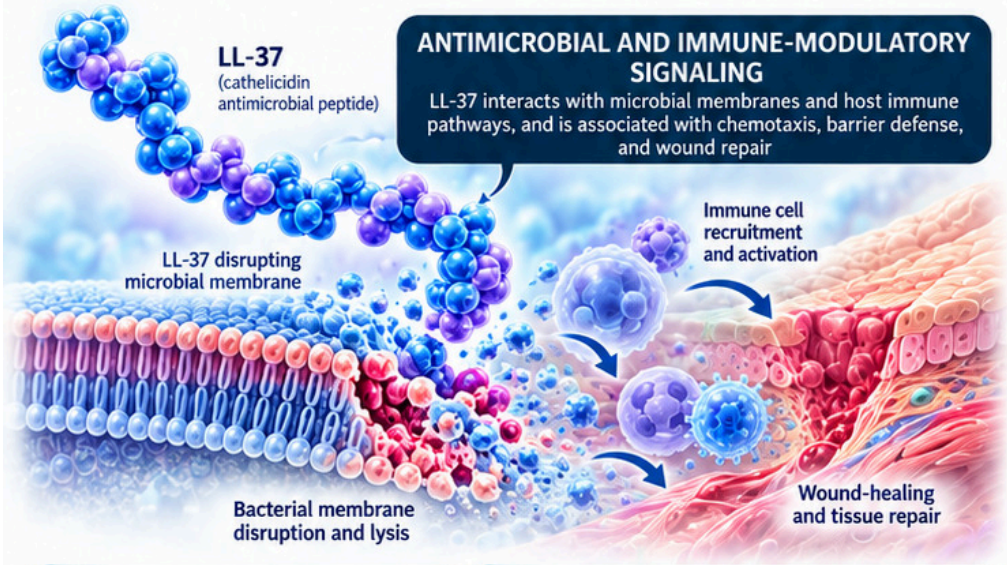
Strong foundational biology but variable direct clinical evidence depending on indication.

Robust mechanistic, preclinical, and biochemical evidence supports glutathione's roles, while human clinical evidence varies by route, population, and specific use.

LL-37

Mechanism, Physiology, and Clinical Effects

A CATIONIC CATHELICIDIN ANTIMICROBIAL PEPTIDE ASSOCIATED WITH INNATE IMMUNITY AND WOUND-HEALING BIOLOGY



Pharmacokinetics

INVESTIGATIONAL USE; NOT WELL ESTABLISHED

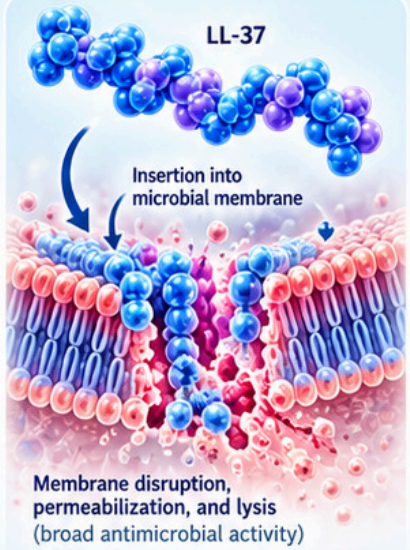
Investigational peptide
Often used in research or specialty contexts (e.g., topical or local administration).

Human pharmacokinetics not well defined
Systemic exposure, half-life, and optimal dosing are not well standardized.

Limited data
Pharmacokinetic and dose-ranging data in humans are limited and formulation-dependent.

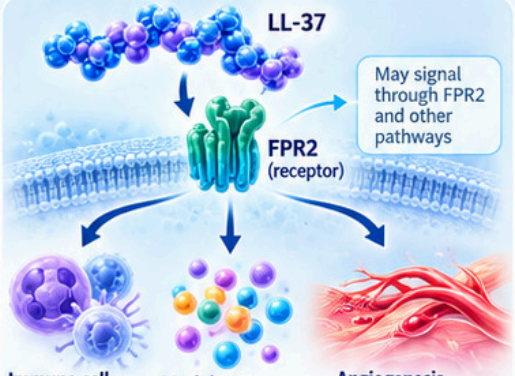
1 Drug action

MICROBIAL MEMBRANE DISRUPTION AND HOST-DEFENSE ACTIVITY



2 Intracellular / tissue signaling

ACTIVATES INNATE IMMUNE PATHWAYS AND SUPPORTS TISSUE REPAIR



Immune cell recruitment and chemotaxis
- Neutrophils
- Monocytes
- Dendritic cells
- T cells

Modulates cytokine signaling
- May reduce excessive inflammation
- Supports immune balance
- Influences antimicrobial responses

Angiogenesis and wound-healing
- Promotes cell migration and proliferation
- Supports re-epithelialization
- May enhance collagen deposition and tissue repair processes

3 Physiologic effects

MULTI-TISSUE ACTIONS

Antimicrobial defense support
- Disrupts microbial membranes
- Broad-spectrum activity (bacteria, fungi, some viruses)

Wound-healing support
- Supports re-epithelialization
- Promotes angiogenesis
- May enhance collagen production and tissue repair

Skin and barrier support
- Supports epithelial integrity
- Enhances barrier defense
- May help maintain mucosal and skin health

Immune-response modulation
- Modulates cytokine production
- Supports innate immunity
- May help resolve inflammation and promote immune balance

4 Clinical outcomes

POTENTIAL BENEFITS BASED ON PRECLINICAL AND LIMITED CLINICAL DATA

Support for difficult wounds
May aid healing in chronic or non-healing wounds (e.g., diabetic ulcers, skin injuries)

Host-defense support
Potential benefit in settings of impaired barrier function or recurrent infections

Skin and mucosal health support
May support epithelial barrier integrity and tissue homeostasis

Robust human clinical evidence for off-label LL-37 peptide use is limited, with most data from preclinical and early clinical studies. More high-quality human trials are needed.

Common Side Effects

MOSTLY LOCAL; SYSTEMIC SAFETY UNCERTAIN

Local irritation
Redness, soreness, or stinging (at application site)

Inflammatory flare
Transient increase in inflammation (possible)

Discomfort
Burning or sensitivity (e.g., topical use)

Uncertain systemic effects
Long-term safety data in humans are limited

Evidence Level

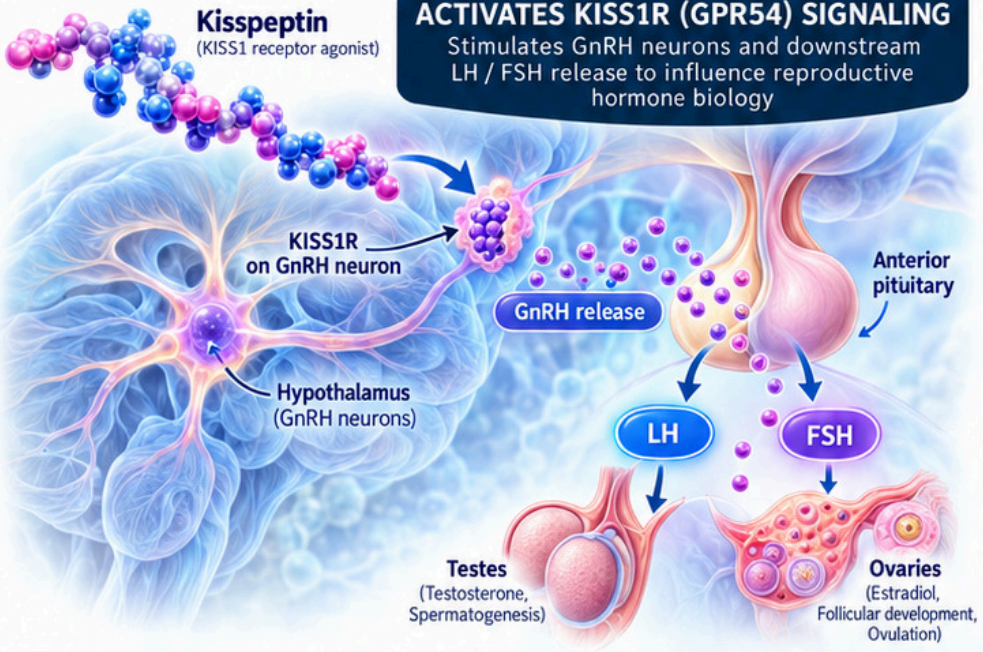
Promising mechanistic and preclinical data with limited clinical evidence

Multiple biological mechanisms support potential benefit, but robust human clinical trials are limited. Additional well-designed studies are needed.

Kisspeptin

Mechanism, Physiology, and Clinical Effects

A KISS1 RECEPTOR AGONIST ASSOCIATED WITH HYPOTHALAMIC–PITUITARY–GONADAL AXIS ACTIVATION



ACTIVATES KISS1R (GPR54) SIGNALING

Stimulates GnRH neurons and downstream LH / FSH release to influence reproductive hormone biology

Pharmacokinetics

RESEARCH AND FERTILITY-FOCUSED USE



Administration
Often subcutaneously or intravenously in research and reproductive settings



Half-life
Generally short-acting with route-dependent pharmacokinetics (typically minutes to hours)



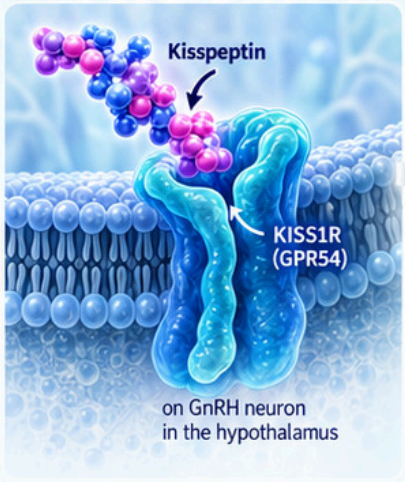
Pulsatile physiology
Kisspeptin signaling is naturally pulsatile, which is relevant for reproductive function



Clinical use
Mainly investigational or fertility-focused; not widely used for general wellness

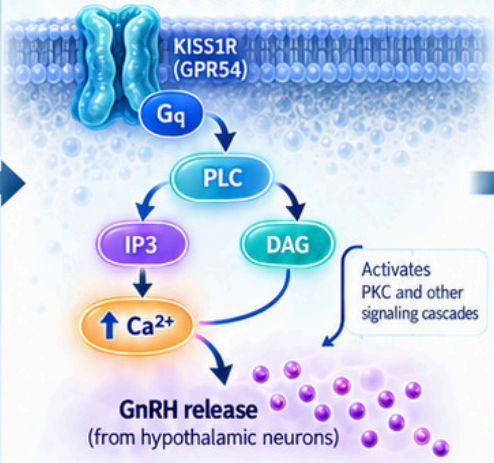
1 Drug binding

KISSPEPTIN BINDS KISS1 RECEPTOR ON GnRH-NEURON PATHWAYS



2 Intracellular signaling

KISS1R ACTIVATION TRIGGERS Gq SIGNALING AND INCREASES INTRACELLULAR Ca^{2+}



3 Physiologic effects

ACTIVATES THE HYPOTHALAMIC–PITUITARY–GONADAL (HPG) AXIS



Anterior pituitary activation
- ↑ LH secretion
- ↑ FSH secretion



Male reproductive hormone support
- ↑ Testosterone synthesis
- ↑ Spermatogenesis support (in selected settings)



Female ovulatory / follicular support
- ↑ Follicular development
- ↑ Ovulatory function
- ↑ Estradiol production



HPG-axis regulation
Regulates GnRH, LH, and FSH secretion and helps coordinate reproductive hormone balance

4 Clinical outcomes

POTENTIAL BENEFITS IN SELECTED CLINICAL AND RESEARCH SETTINGS



Fertility support
May support reproductive function in men and women (in fertility and reproductive medicine settings)



Reproductive-axis evaluation
Used in research and clinical testing to assess HPG-axis function (e.g., GnRH / LH / FSH response)



Potential hypogonadotropic support
May be considered in selected settings (e.g., hypothalamic hypogonadism) under specialist care

Generalized wellness outcomes, such as improved energy, mood, or body composition, are not well established.

Common Side Effects

GENERALLY MILD AND TRANSIENT


Flushing


Headache


Nausea


Injection-site irritation


Abdominal discomfort

Evidence Level

STRONG PHYSIOLOGIC RATIONALE; LIMITED BROAD USE

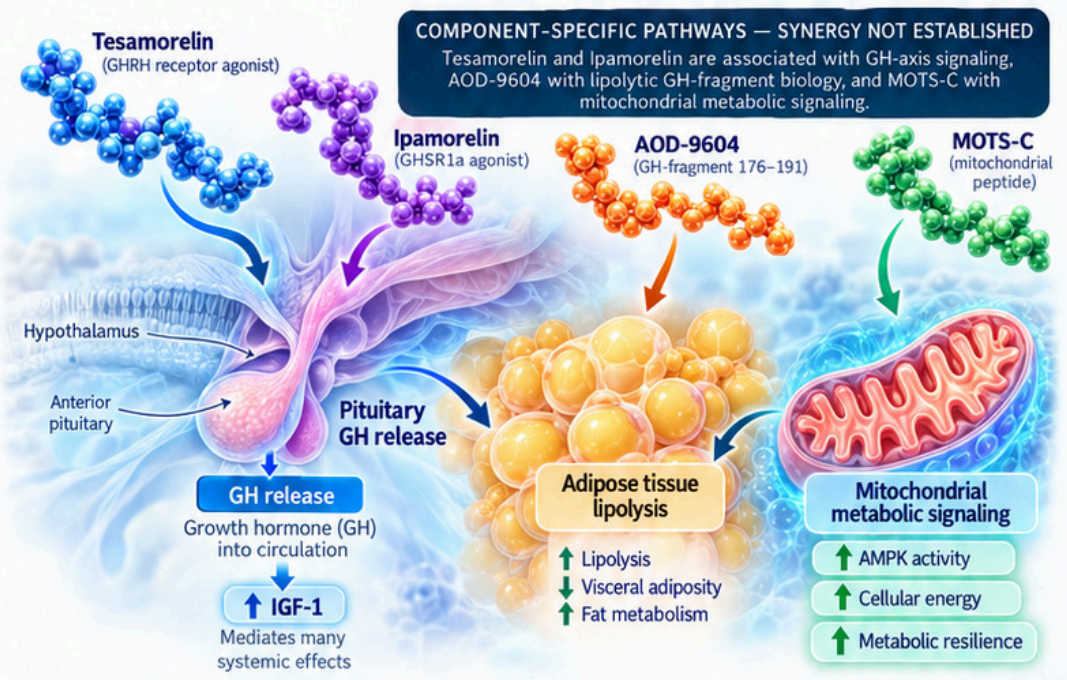


Strong endocrine physiology and selected clinical research applications, but limited broad wellness evidence.

Physiq

Tesamorelin / Ipamorelin / AOD-9604 / MOTS-C
Mechanism, Physiology, and Clinical Effects

AN INVESTIGATIONAL MULTI-INGREDIENT BLEND ASSOCIATED WITH GH-AXIS SIGNALING, LIPOLYTIC BIOLOGY, AND METABOLIC / MITOCHONDRIAL SUPPORT



Pharmacokinetics

INVESTIGATIONAL BLEND; COMPONENT PK VARIES

Subcutaneous investigational blend
Typically administered once daily (research use)

Component PK differs substantially
Each peptide has unique absorption, distribution, and half-life characteristics.

Tesamorelin
- Short plasma half-life (~30–60 minutes)
- Longer-lasting IGF-1 effect (~12–24 hours)

Ipamorelin
- Short-acting (~1–2 hours)

AOD-9604
- Human PK data limited ($t_{1/2}$ not well established)

MOTS-C
- Human PK data limited ($t_{1/2}$ not well established)

PK for the full Physiq blend is not established.

1 Component actions

EACH PEPTIDE TARGETS DISTINCT BIOLOGICAL PATHWAYS

Tesamorelin
- GHRH receptor agonism
- Stimulates pituitary GH release
- Increases circulating IGF-1

Ipamorelin
- GHSR1a agonism
- Stimulates pituitary GH release
- Minimal effect on cortisol and prolactin

AOD-9604
- GH-fragment (176–191) biology
- Associated with lipolysis and fat-metabolism effects
- Does not stimulate GH release

MOTS-C
- Mitochondrial metabolic signaling
- AMPK-related stress-response biology
- Supports metabolic health and cellular resilience

2 Intracellular / tissue signaling

COMPONENT-SPECIFIC PATHWAYS

Tesamorelin (GHRH receptor)
Gs → Adenylate cyclase → ↑ cAMP → ↑ GH release → ↑ IGF-1
Regulates growth, body composition, and metabolism

Ipamorelin (GHSR1a receptor)
Gq → PLC → ↑ IP3 / DAG → ↑ Ca^{2+} → ↑ GH release → ↑ IGF-1
Stimulates GH release with minimal cortisol and prolactin effects

AOD-9604 (GH-fragment 176–191)
Adipose tissue → ↑ cAMP / PKA → Hormone-sensitive lipase (HSL) → ↑ Lipolysis
- Mobilizes stored fat
- Supports visceral fat reduction

MOTS-C (mitochondrial signaling)
AMPK activation → ↑ Mitochondrial function → ↑ Fatty acid oxidation → ↑ Metabolic resilience
Supports cellular energy and metabolic flexibility

3 Physiologic effects

MULTI-SYSTEM ACTIONS

Body composition support
- ↑ Lean mass preservation
- ↑ Reduced fat mass
- ↑ Improved body composition

Visceral-fat reduction support
- ↑ Lipolysis and fat metabolism
- ↓ Visceral adiposity
- ↑ More favorable fat distribution

Metabolic flexibility
- ↑ Fat utilization
- ↑ Insulin sensitivity (potential)
- ↑ Healthy metabolic function

Exercise recovery support
- ↑ Recovery and performance adaptation
- ↓ Exercise-induced fatigue
- ↑ Muscle preservation

Mitochondrial / energy support
- ↑ Mitochondrial function
- ↑ Cellular energy production
- ↑ Metabolic resilience and healthy aging

4 Clinical outcomes

POTENTIAL BENEFITS BASED ON AVAILABLE EVIDENCE; FULL BLEND DATA ARE LIMITED

Improved body composition
Reduced fat mass and preserved lean mass

Visceral fat reduction support
Decreases visceral adiposity and supports a healthier fat distribution

Metabolic health support
Greater metabolic flexibility and fat utilization

Exercise recovery and physical function
Supports recovery, performance adaptation, and muscle preservation

Tesamorelin has the strongest direct human evidence (e.g., improved body composition in HIV-associated lipodystrophy). Robust direct evidence for the full Physiq blend is limited, with most data from preclinical and early research studies.

Common Side Effects

MOSTLY MILD TO MODERATE; BLEND SAFETY NOT FULLY ESTABLISHED

- Injection-site reactions
- Edema (fluid retention)
- Headache
- Flushing
- Changes in glucose regulation
- Nausea
- Uncertain long-term safety — Long-term safety data for the blend are not established

Evidence Level

MIXED — STRONGER EVIDENCE FOR TESAMORELIN INDIVIDUALLY

Tesamorelin has well-established clinical evidence for body composition benefits. Evidence for ipamorelin, AOD-9604, and MOTS-C, and for the full Physiq blend, is more limited and primarily preclinical or early research.