Tesamorelin

GHRH Analog • Growth Hormone Pathway • Visceral Fat Research

At a Glance

What is Tesamorelin?

Tesamorelin is a peptide that tells the pituitary gland to release more of the body's own growth hormone.

That increase in growth hormone also raises IGF-1 and can affect body fat and metabolism.

Unlike HGH, Tesamorelin does **not** supply growth hormone directly. It stimulates the body's own GH-release pathway.

How well studied is it?

Well studied for one specific medical use

Tesamorelin is much better studied than most compounds commonly discussed in peptide research.

It has:

- randomized human clinical trials
- human follow-up lasting up to about a year
- established human safety data
- FDA-approved formulations
- a well-defined receptor and biological pathway

Its strongest evidence is for reducing **excess visceral abdominal fat in adults with HIV-associated lipodystrophy**.

Evidence level

Approved-label human evidence: Strong
Human clinical research: Strong for the approved indication
Other/off-label uses: Limited or unproven
Community protocols: Common, but not equivalent to clinical evidence

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What may it help with?

Strong human evidence

Visceral abdominal fat

Tesamorelin has been shown to reduce visceral adipose tissue, meaning the deeper abdominal fat that surrounds the internal organs, in adults with HIV-associated lipodystrophy. In pooled Phase III research, daily Tesamorelin significantly reduced visceral fat compared with placebo.

Abdominal body composition

Clinical studies found improvements in waist measurements and central-fat distribution in the population studied.

Some human evidence

Liver fat

Randomized human studies have found reductions in liver fat in selected people with HIV and abdominal adiposity or HIV-associated NAFLD.

A 2014 randomized trial found reductions in visceral adipose tissue and a **modest reduction in liver fat** over 6 months.

A later 12-month randomized trial also found a significant reduction in hepatic fat fraction in people with HIV and NAFLD.

These findings are clinically meaningful, but they should **not** be generalized to every form of fatty liver disease or to people outside the populations studied.

Sources: [5][7]

Some lipid and metabolic markers

Clinical trials have also found improvements in measures such as triglycerides and cholesterol-related markers in the studied population.

What has it NOT been proven to help with?

Tesamorelin has **not** been established as a treatment for:

- ordinary weight loss
- general obesity
- bodybuilding
- muscle gain
- anti-aging
- athletic performance
- workout recovery
- general fatty liver disease

- alcohol-related liver damage
- liver “detox”

The current EGRIFTA WR label specifically says it is **not indicated for weight-loss management**.

Losing visceral fat is not the same thing as being a general weight-loss drug.

Dose at a Glance

Clinical / FDA-approved dosing

There is **not one universal Tesamorelin dose**.

EGRIFTA WR

Dose: 1.28 mg
Route: Subcutaneous
Frequency: Once daily

Current EGRIFTA WR contains **11.6 mg of Tesamorelin per vial**.

One dose is **1.28 mg in 0.16 mL** after reconstitution.

EGRIFTA SV

Dose: 1.4 mg
Route: Subcutaneous
Frequency: Once daily

EGRIFTA SV uses a separate **2 mg-per-vial formulation** with its own preparation and storage instructions.

Historical clinical trials

Older major human studies used:

2 mg SC once daily.

That is a legitimate human clinical dose, but it should **not** be described as the universal current FDA-approved dose.

Research-community dosing

Community protocols commonly use approximately:

1–2 mg SC once daily

PeptideFox currently describes community Tesamorelin use in this general range, typically once daily and often at night.

Evidence level

Community / practitioner protocol

The dose range partly reflects genuine historical human dosing, but the specific community schedules are not the current FDA protocol.

Frequency at a Glance

Clinical

Once daily

Community

Usually **once daily**

Split / twice-daily dosing

Sometimes discussed, but there is no established human evidence showing that dividing Tesamorelin into two daily injections works better.

Best-supported frequency: once daily.

Timing at a Glance

Clinical

There is **no FDA requirement** to use Tesamorelin:

- before bed
- while fasted
- before exercise
- after exercise

The approved protocol is simply once daily.

Community

Community protocols often place the dose:

- before bed
- away from food

PeptideFox describes this type of timing.

Evidence verdict

Bedtime: Mechanistically plausible but unproven
Fasting: Mechanistically plausible but unproven
Required: No

Duration / Cycle at a Glance

Clinical research

Major clinical studies used approximately:

26 weeks of continuous daily treatment

with extension research reaching approximately:

52 weeks.

Community protocol

Community protocols often use shorter cycles followed by a break.

A commonly discussed structure is approximately:

12–16 weeks on
followed by
2–4 weeks off

This is a community convention rather than a proven receptor-reset schedule.

Does it need to be cycled?

No required receptor-reset cycle has been established.

Human research does not show that Tesamorelin must be stopped after 8, 12 or 16 weeks to restore receptor sensitivity.

Expected Results Timeline

Clinical research

Around 12 weeks

A randomized dose-ranging study using fixed 1 mg and 2 mg once-daily groups measured abdominal-fat changes over **12 weeks**.

Around 26 weeks

The major Phase III trials evaluated their main visceral-fat outcomes after approximately **26 weeks** of daily treatment.

Up to 52 weeks

Extension research followed participants for approximately **one year**. Continued Tesamorelin maintained visceral-fat reduction, while stopping treatment was associated with reaccumulation of visceral fat.

Clinical takeaway

For the outcome Tesamorelin is actually well studied for, think in **months rather than days**.

Community-Reported Timeline

Community and practitioner reports describe body-composition changes developing gradually over **several weeks to a few months**.

PeptideFox specifically describes body-composition changes beginning to become noticeable or measurable at approximately:

4–6 weeks.

Important

This is **not a statistically calculated average from controlled Tesamorelin trials**.

Navaro should describe this as:

> **Commonly reported community timeline**

rather than:

> **Average time to results**

Earlier claims involving sleep, energy, strength or recovery are inconsistent and should not be presented as established Tesamorelin effects.

Simple Timeline for the Top Card

Clinical: measurable abdominal-fat changes studied by approximately **12 weeks**, with major outcomes assessed around **26 weeks** and longer-term research extending to approximately **52 weeks**.

Community-reported: body-composition changes are commonly described over **several weeks to a few months**, with PeptideFox specifically reporting changes beginning around **4–6 weeks**.

Safety at a Glance

Common or important effects

- injection-site reactions
- joint pain
- muscle aches
- pain in the arms or legs
- swelling
- fluid retention
- tingling or numbness
- carpal-tunnel-type symptoms
- elevated IGF-1
- increased blood sugar
- rash
- hypersensitivity reactions

The current WR label lists arthralgia, injection-site redness and itching, pain in the extremities, peripheral edema and myalgia among the most commonly reported adverse reactions.

Major contraindications

The current EGRIFTA WR label lists four formal contraindications:

- **active malignancy**
- **pregnancy**
- **disruption of the hypothalamic-pituitary axis**
- **known hypersensitivity to Tesamorelin or the formulation ingredients**

Regulatory Status at a Glance

FDA approved?

Yes

Approved for

Reduction of excess abdominal fat in adults with HIV-associated lipodystrophy.

Approved for ordinary weight loss?

No

General bodybuilding / anti-aging approval?

No

Bottom Line

Tesamorelin is one of the better-studied compounds in the peptide space, but its strongest evidence applies to a specific medical use.

The clearest evidence supports:

Subcutaneous administration once daily

for reducing:

visceral abdominal fat in adults with HIV-associated lipodystrophy.

Current FDA dosing depends on the exact formulation:

EGRIFTA WR: 1.28 mg daily
EGRIFTA SV: 1.4 mg daily

Older human trials used:

2 mg daily.

Research communities commonly use:

1–2 mg daily

often at night and away from food.

However, bedtime use, fasting, automatic 1→2 mg escalation, fixed community cycles and receptor-reset breaks have **not** been established as necessary by clinical trials.

Protocol Logic Check

Quick Logic Summary

Protocol Question	Verdict
Once daily?	**Evidence-supported**
Subcutaneous route?	**Evidence-supported**
1.28 mg EGRIFTA WR?	**Evidence-supported for WR**
1.4 mg EGRIFTA SV?	**Evidence-supported for SV**
Historical 2 mg/day?	**Evidence-supported historical trial dose**
Bedtime dosing?	**Plausible but unproven**
Fasted dosing?	**Plausible but unproven**
Twice-daily split dosing?	**Plausible but unproven**
Automatic 1→2 mg titration?	**Community convention**
5 days on / 2 off?	**No demonstrated rationale**
12–16 week mandatory cycle?	**Community convention**
2–4 week receptor reset?	**No demonstrated requirement**
Pre/post-workout timing?	**No demonstrated rationale**
Generic 28-day vial stability?	**Cannot be assumed**
SV and WR interchangeable?	**No**

Exact Target / Pathway

Tesamorelin acts through the **growth hormone-releasing hormone receptor, or GHRH-R**, in the pituitary gland.

In simple terms:

Tesamorelin → pituitary gland → more natural GH release → increased IGF-1

Verdict

Evidence-supported

Human Half-Life

Tesamorelin itself does not need to stay in the bloodstream all day to work.

It activates the pituitary and triggers a downstream GH and IGF-1 response.

Why this matters

A short plasma half-life does **not** automatically mean Tesamorelin needs several injections per day.

Verdict

Evidence-supported concept

Pharmacodynamic Duration

The biological response continues beyond the brief circulation of the parent peptide because Tesamorelin starts an endocrine signaling cascade.

Clinical trials directly support once-daily dosing.

Verdict

Evidence-supported

Accumulation / Steady State

Tesamorelin is not best understood as a drug that needs to remain at a constant high blood level.

The repeated **daily GH-axis signal** and resulting IGF-1 response are more relevant than accumulation of intact Tesamorelin.

Verdict

Mechanistically supported

Does Once-Daily Dosing Make Sense?

Yes.

Once-daily dosing is used by:

- EGRIFTA WR
- EGRIFTA SV
- major clinical trials

Verdict

Evidence-supported

Does Twice-Daily Dosing Make Sense?

It is biologically possible to stimulate the pathway more than once per day.

But there is no established evidence that splitting Tesamorelin into two daily doses improves:

- visceral-fat reduction
- body composition
- GH response
- IGF-1 response
- tolerability

Verdict

Mechanistically plausible but unproven

It should **not** appear as a validated alternative protocol.

Does Bedtime Dosing Make Sense?

Growth hormone secretion has an important relationship with sleep and circadian physiology.

That makes bedtime use biologically understandable.

But the approved protocol does not require bedtime dosing.

Verdict

Mechanistically plausible but unproven

Does Fasting Make Sense?

Food, glucose, insulin and free fatty acids can influence normal GH physiology.

That gives community fasting practices a theoretical basis.

However, the approved product labeling does not require fasting.

Verdict

Mechanistically plausible but unproven

Pre-Workout or Post-Workout Timing

There is no established human Tesamorelin evidence showing superior results when taken:

- before exercise
- immediately after exercise

Verdict

Community convention without demonstrated rationale

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5 Days On / 2 Days Off

Clinical studies used continuous daily treatment.

There is no evidence showing that weekend breaks improve response or protect the receptor.

Verdict

Community convention without demonstrated rationale

8–12 Week Cycle

Clinical research lasted substantially longer than eight or twelve weeks.

There is no demonstrated biological event at week 8 or 12 that requires treatment to stop.

Verdict

Community convention without demonstrated rationale

12–16 Weeks On / 2–4 Weeks Off

This structure appears in peptide-community protocols.

It is **not** the FDA protocol.

Verdict

Community convention without demonstrated rationale

Receptor Desensitization / Tachyphylaxis

GHRH receptors can undergo normal receptor regulation.

But that general biological fact does not establish that:

> Tesamorelin causes clinically important pituitary downregulation after 12 weeks.

Continuous treatment has remained effective in human studies lasting much longer.

Verdict

Receptor regulation is biologically possible, but a mandatory Tesamorelin reset has not been demonstrated

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Washout / Receptor Reset

There is no established **2–4 week biological reset period** for Tesamorelin.

Treatment may be stopped or reassessed because of:

- excessive IGF-1
- worsening glucose
- edema
- adverse effects
- lack of meaningful benefit

Those are treatment-management reasons, not proof that GHRH receptors need to be reset.

Verdict

Community convention without demonstrated receptor-reset evidence

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Dose Escalation / Titration

Human researchers have directly compared different fixed doses.

A 12-week randomized study assigned participants to:

- placebo
- 1 mg Tesamorelin once daily
- 2 mg Tesamorelin once daily

This establishes **human dose-ranging research**.

It does **not** establish:

> Start everyone at 1 mg and automatically increase everyone to 2 mg.

Verdict

Community titration built around genuine human dose-ranging research

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Route Match

Clinical studies

Subcutaneous

FDA products

Subcutaneous

Community protocols

Usually subcutaneous

Verdict

Evidence-supported

Stack / Synergy Logic

Tesamorelin + Ipamorelin

Different upstream receptors but the same broad GH axis.

Verdict: Mechanistically complementary but clinically unproven.

Tesamorelin + GHRP-2 / GHRP-6

Similar relationship.

Verdict: Mechanistically complementary but clinically unproven.

Tesamorelin + Sermorelin / CJC

Substantial GHRH-pathway overlap.

Verdict: Largely redundant.

Tesamorelin + HGH

Tesamorelin stimulates endogenous GH while HGH adds exogenous GH.

Verdict: Pathway overlapping and clinically unvalidated.

Tesamorelin + GLP-1 drugs

Different pathways.

Verdict: Mechanistically nonredundant, but combination benefit is unproven.

Formulation / Stability / Sterility Logic

This is especially important for Tesamorelin.

EGRIFTA WR and EGRIFTA SV have different:

- vial strengths
- recommended doses
- preparation instructions
- diluents
- storage requirements

The current WR label specifically states that **EGRIFTA WR and EGRIFTA SV are not substitutable**.

Verdict

Exact-product evidence required

Important Research Note

Three different forms of Tesamorelin information are commonly mixed together online.

1. Current FDA-Approved Formulations

EGRIFTA WR and EGRIFTA SV have different:

- doses
- vial strengths
- dose volumes
- reconstitution instructions
- supplied diluents
- storage requirements

They should never be presented as interchangeable.

2. Historical Clinical Research

Older clinical trials commonly used:

2 mg once daily.

That is valid human evidence.

But:

> **Historical 2 mg clinical dosing is not the same thing as the universal current FDA dose.**

3. Research-Community Protocols

Community protocols commonly use:

1–2 mg once daily

with additional practices such as:

- bedtime use
- fasting
- titration
- cycling
- washout periods

Those additions should remain clearly labelled as community protocols rather than clinical requirements.

Research at a Glance

Approved-Label Human Evidence

Strong

Current FDA labeling gives exact human dosing, safety monitoring, contraindications and product handling.

Human Clinical Research

Strong for the approved indication

Randomized human studies support visceral-fat reduction with daily Tesamorelin.

Observational Human Evidence

Supporting

Additional analyses provide information about response patterns and metabolic effects.

Animal / Cell Evidence

Supporting

Useful for mechanistic questions, but the core Tesamorelin conclusions can be based directly on human research.

Mechanistic Evidence

Strong

The GHRH → GH → IGF-1 pathway is well characterized.

Community / Anecdotal Evidence

Common but lower quality

Useful for understanding how Tesamorelin is being used outside approved settings, but not equivalent to clinical evidence.

Overview

Tesamorelin is a synthetic analog of human growth hormone-releasing hormone.

It stimulates the pituitary gland rather than supplying growth hormone directly.

This leads to increased endogenous GH secretion and downstream increases in IGF-1.

Its strongest clinical effect is reduction of **visceral abdominal fat** in adults with HIV-associated lipodystrophy.

Evidence Strength

Overall Evidence Rating

Strong human evidence for a narrow clinical use

Strong evidence for

- GHRH receptor activity
- stimulation of endogenous GH
- increased IGF-1
- once-daily SC administration
- visceral-fat reduction in HIV-associated lipodystrophy
- current product-specific FDA dosing
- important safety risks

Some human evidence for

- liver-fat reduction in selected HIV populations
- selected metabolic outcomes
- selected lipid changes

Limited or unproven for

- general obesity
- general weight loss
- healthy-user body recomposition
- bodybuilding
- muscle gain
- anti-aging
- athletic performance
- injury recovery
- bedtime superiority
- fasting superiority
- split dosing
- receptor-reset cycling

Category

Growth Hormone Secretagogues / GHRH Analogs

Compound

Tesamorelin

Historical research/development name:

TH9507

Current US products include:

- EGRIFTA WR
- EGRIFTA SV

Compound Type

Synthetic growth hormone-releasing hormone analog

Tesamorelin is not:

- HGH
- IGF-1
- a GLP-1 agonist
- a ghrelin agonist
- a traditional GHRP

Primary Research Focus

Best Established

Visceral-fat reduction in adults with HIV-associated lipodystrophy

Also Studied

- abdominal body composition
- liver fat in selected HIV populations
- metabolic changes
- lipid measurements
- GH / IGF-1 response

Not Established

- healthy bodybuilding
- anti-aging treatment
- general obesity treatment
- sports performance
- injury-recovery treatment

How It Works / Receptor Mechanism

Tesamorelin binds the **GHRH receptor** on cells in the pituitary gland.

Simple version

Tesamorelin tells the pituitary to release more growth hormone.

That growth hormone then increases downstream IGF-1.

Pathway

Tesamorelin → GHRH-R → endogenous GH → IGF-1

How It Differs From Related Compounds

Tesamorelin
Stimulates GHRH-R.

Ipamorelin / GHRP-2 / GHRP-6
Primarily stimulate GHS-R1a.

HGH / Somatropin
Supplies GH directly.

IGF-1 compounds
Act farther downstream.

Common Research Dose

Approved Clinical Dosing

EGRIFTA WR: 1.28 mg once daily SC

EGRIFTA SV: 1.4 mg once daily SC

Historical Human Research

2 mg once daily SC

Research Community

Commonly:

1–2 mg once daily SC

Research Duration / Cycle Length

Clinical

Approximately:

26 weeks

with extension research to approximately:

52 weeks.

Research Community

Commonly:

12–16 weeks on
followed by
2–4 weeks off

Evidence assessment

Clinical studies support continuous daily administration over months.

The shorter community cycle is not supported by demonstrated GHRH-receptor reset biology.

How Often It Is Used / Frequency

Clinical

Once daily

Research Community

Usually **once daily**

Split Dosing

No established advantage.

Possible Benefits

Strong Human Evidence

Visceral-Fat Reduction

Tesamorelin has demonstrated reductions in visceral abdominal fat in the studied HIV-associated lipodystrophy population.

Abdominal Body Composition

Human studies show changes in central-fat distribution and waist measurements.

Some Human Evidence

Liver Fat

Randomized human studies have found reductions in liver fat in selected people with HIV and abdominal adiposity or HIV-associated NAFLD.

A 2014 randomized trial found reductions in visceral adipose tissue and a **modest reduction in liver fat** over 6 months.

A later 12-month randomized trial also found a significant reduction in hepatic fat fraction in people with HIV and NAFLD.

These findings are clinically meaningful, but they should **not** be generalized to every form of fatty liver disease.

Lipid / Metabolic Markers

Human studies have also reported improvements in triglycerides and some other metabolic measures.

What It Does Not Help With, Based on Current Evidence

Do not present the following as established Tesamorelin benefits:

- general weight loss
- universal "belly fat" treatment
- healthy-person bodybuilding
- guaranteed lean-mass preservation
- muscle growth
- faster injury repair
- improved athletic performance
- anti-aging

- alcohol detoxification
- treatment of all fatty liver disease

Commonly Paired With

The heading should be **Commonly Paired With**, not "Best Synergies," unless actual combination evidence exists.

Ipamorelin

Uses a different upstream receptor but ultimately affects the same GH axis.

Classification: Mechanistically complementary but clinically unproven.

GHRP-2 / GHRP-6

Similar relationship.

Classification: Mechanistically complementary but clinically unproven.

Sermorelin

Also stimulates the GHRH pathway.

Classification: Largely redundant.

CJC / Modified GRF

Also acts through the GHRH pathway.

Classification: Largely redundant.

HGH / Somatropin

Adds exogenous GH while Tesamorelin is already stimulating endogenous GH.

Classification: Pathway overlapping and clinically unvalidated.

GLP-1 Agonists

Work through a different pathway.

Classification: Mechanistically nonredundant, but combination benefit has not been established by controlled Tesamorelin trials.

Dosage Information

EGRIFTA WR

This section should remain very close to the current product label.

Product: EGRIFTA WR
Tesamorelin per vial: 11.6 mg
Recommended dose: 1.28 mg
Dose volume after reconstitution: 0.16 mL
Route: Subcutaneous
Frequency: Once daily
Injection area: Abdomen, rotating injection sites

Reconstitution

Use only the supplied **Bacteriostatic Water for Injection**.

Reconstitute one vial with:

1.3 mL

This produces a concentration of:

8 mg/mL

Swirl the vial to mix.

Do not shake.

Use only if the solution is clear, colorless and free of visible particles.

After Reconstitution

One reconstituted vial provides:

7 consecutive daily doses

Store the reconstituted vial at:

20°C to 25°C (68°F to 77°F)

Discard any unused solution:

7 days after mixing

Do not freeze.

EGRIFTA SV

Product: EGRIFTA SV
Vial: 2 mg formulation

Recommended dose: 1.4 mg
Route: Subcutaneous
Frequency: Once daily

Follow the exact EGRIFTA SV label for its supplied diluent, preparation, injection volume and post-reconstitution handling.

Important Formulation Warning

EGRIFTA WR and EGRIFTA SV are not substitutable.

The current WR label specifically distinguishes the two because they differ in:

- recommended dosage
- number of vials needed
- reconstitution instructions
- storage requirements

This is exactly why a generic statement such as:

> "Tesamorelin lasts 28 days after reconstitution"

should **not** appear on the page.

Community Research Protocol

Commonly Published Community Pattern

Community material commonly describes approximately:

Dose: 1–2 mg
Route: SC
Frequency: Once daily
Timing: Often at night
Food timing: Often away from meals
Cycle: Often shorter than the clinical trials, sometimes followed by a break

Evidence Check

SC route: Supported

Once daily: Supported

1–2 mg range: Has historical human-dose precedent, but does not equal current formulation-specific FDA dosing

Bedtime: Plausible but unproven

Fasting: Plausible but unproven

Automatic 1→2 mg escalation: Community convention

Fixed cycle: Community convention

Receptor-reset break: No demonstrated requirement

Community Protocol Comparison

Source	Dose	Frequency	Timing	Duration	Evidence
EGRIFTA WR	1.28 mg	Daily SC	No required bedtime	Clinical treatment	FDA label
EGRIFTA SV	1.4 mg	Daily SC	No required bedtime	Clinical treatment	FDA label
Historical trials	2 mg	Daily SC	Study-specific	~26–52 weeks	Human clinical
Community protocols	~1–2 mg	Usually daily SC	Often bedtime / away from meals	Variable	Community

Strongest Agreement

Once-daily subcutaneous administration

Partial Agreement

Low-milligram daily exposure, but exact dose depends on formulation and context.

Community Additions

- bedtime
- fasting
- titration
- cycling
- washout

Expected Results Timeline, Expanded

Human Clinical Research

About 12 Weeks

Dose-ranging human research found measurable abdominal-fat changes during a 12-week study using fixed 1 mg and 2 mg daily groups.

About 26 Weeks

Major clinical trials evaluated visceral-fat outcomes after approximately six months of daily treatment.

About 52 Weeks

Extension research evaluated continued treatment through approximately one year. Visceral-fat reduction was maintained with continued treatment, while fat reaccumulated after withdrawal.

Community-Reported

Community and practitioner reports describe body-composition changes developing gradually over **several weeks to a few months**.

PeptideFox specifically describes changes beginning to become noticeable or measurable at approximately:

4–6 weeks.

Evidence Warning

There is no high-quality pooled dataset calculating the **average community time to visible results**.

Therefore Navaro should say:

> **Commonly reported community timeline**

rather than:

> **Average results timeline**

Do not present early sleep, energy, strength or recovery changes as established Tesamorelin outcomes.

Reported Side Effects

Commonly Reported in Clinical Trials

- injection-site reactions
- joint pain
- pain in the arms or legs
- muscle pain
- peripheral edema
- tingling
- reduced sensation
- rash
- swelling
- carpal-tunnel-type symptoms

Elevated IGF-1

Tesamorelin increases GH and IGF-1.

The current WR label recommends monitoring IGF-1 during treatment and considering discontinuation when IGF-1 remains persistently elevated, especially when benefit is limited.

Blood Sugar

Tesamorelin can impair glucose control.

The WR label recommends evaluating glucose before treatment and monitoring during treatment.

Fluid Retention

GH stimulation may contribute to:

- edema
- joint discomfort
- carpal tunnel syndrome

Hypersensitivity

Reported reactions include:

- itching
- redness
- flushing
- hives
- rash

Contraindications & Cautions

Formal Contraindications

Active Malignancy

Contraindicated

Pregnancy

Contraindicated

Disruption of the Hypothalamic-Pituitary Axis

Includes conditions such as pituitary disease or surgery, head irradiation or significant head trauma.

Contraindicated

Known Hypersensitivity

To Tesamorelin or formulation ingredients.

Contraindicated

Important Cautions

Previous Malignancy

Requires careful benefit-risk assessment.

Diabetes / Impaired Glucose Tolerance

Tesamorelin can worsen glucose control.

Diabetic Retinopathy

Because IGF-1 can rise, people with diabetes require appropriate monitoring.

Acute Critical Illness

The product label advises considering discontinuation in critically ill patients because Tesamorelin stimulates GH production.

Regulatory Status

United States

FDA approved

Current approved products include:

- EGRIFTA WR
- EGRIFTA SV

Approved Indication

Reduction of excess abdominal fat in adults with HIV-associated lipodystrophy.

Not Approved For

- general weight loss
- ordinary obesity treatment
- bodybuilding
- anti-aging
- athletic performance

Additional Limitation

Long-term cardiovascular safety has not been established.

What Studies Have Found

12-Week Dose-Ranging Study

Participants received:

- placebo
- 1 mg once daily
- 2 mg once daily

for **12 weeks**.

What This Supports

- human Tesamorelin dose-ranging research exists
- 1 mg and 2 mg fixed doses have both been directly studied
- once-daily SC administration was studied
- abdominal-fat changes were measurable over 12 weeks

What It Does Not Support

A mandatory:

1 mg → 2 mg titration schedule

The study compared **fixed treatment groups** rather than placing all participants on an automatic escalation schedule.

Major Phase III Research

Human clinical trials used:

2 mg SC once daily

over approximately:

26 weeks

with extension research continuing to:

52 weeks.

What This Supports

- once-daily administration
- months of continuous treatment
- visceral-fat reduction
- continued benefit with continued treatment

What It Does Not Support

- healthy bodybuilding
- general weight loss
- twice-daily dosing
- 12-week receptor resets
- mandatory fasting

Liver-Fat Research

2014 Randomized Study

A randomized six-month human study found reductions in visceral adipose tissue and a **modest reduction in liver fat** in people with HIV and abdominal fat accumulation.

What This Supports

- human evidence for visceral-fat reduction
- modest liver-fat reduction in the population studied

What It Does Not Support

Treatment of every form of fatty liver disease.

2019 HIV-Associated NAFLD Study

A later randomized 12-month study found a significant reduction in hepatic fat fraction in people with HIV and NAFLD.

What This Supports

- 12-month Tesamorelin treatment
- significant reduction in hepatic fat fraction
- human liver-fat evidence in people with HIV and NAFLD

What It Does Not Support

Tesamorelin as a general MASLD/NAFLD treatment outside the population studied.

Formulation / Stability / Sterility

EGRIFTA WR

11.6 mg vial

After reconstitution with the supplied bacteriostatic water:

- concentration: **8 mg/mL**
- one vial provides **7 consecutive daily doses**
- store at **20°C to 25°C (68°F to 77°F)**
- discard unused solution **7 days after mixing**
- **do not freeze**

EGRIFTA SV

Separate **2 mg-per-vial formulation** with its own preparation and storage instructions.

Generic Research Tesamorelin

Do **not** automatically apply either approved product's stability to an unspecified research vial.

Stability depends on:

- formulation
- excipients
- diluent
- concentration
- container
- sterility
- validated stability testing

Research Sources

Use these **clean canonical links in production**.

1. DailyMed: EGRIFTA WR Prescribing Information

Evidence type: Current FDA-label human evidence

Supports:

- 11.6 mg WR formulation
- 1.28 mg daily dose
- 0.16 mL dose volume
- 1.3 mL reconstitution
- 8 mg/mL concentration
- seven consecutive doses
- 20°C–25°C storage after mixing
- seven-day discard rule
- non-interchangeability with SV
- contraindications
- IGF-1 monitoring
- glucose monitoring
- adverse effects

`https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=839334d3-8c1d-4c26-9036-2ab524a6ea7`

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2. DailyMed: EGRIFTA SV Prescribing Information

Evidence type: Approved-product information

Supports:

- separate 2 mg-per-vial formulation
- 1.4 mg once-daily dose
- formulation-specific preparation and handling

https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=3d783378-b02d-4f19-99dd-0fc91a04222

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3. Falutz et al.: Phase III Pooled Analysis

Evidence type: Randomized human clinical research

Supports:

- historical 2 mg daily dosing
- once-daily administration
- visceral-fat efficacy
- 26-week treatment
- extension to 52 weeks

https://pubmed.ncbi.nlm.nih.gov/20554713/

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4. Falutz et al.: 12-Week Dose-Ranging Study

Evidence type: Randomized human clinical research

Supports:

- placebo, 1 mg and 2 mg fixed treatment groups
- once-daily SC administration
- 12-week dose-ranging research

Does **not** establish automatic 1→2 mg titration.

https://pubmed.ncbi.nlm.nih.gov/16052083/

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5. Stanley et al.: Visceral Fat and Liver Fat Study

Evidence type: Randomized human clinical research

Supports:

- reduction in visceral adipose tissue
- **modest reduction in liver fat over 6 months**
- human evidence for hepatic effects in a specific HIV population

Does **not** establish treatment for all forms of fatty liver disease.

https://pubmed.ncbi.nlm.nih.gov/25038357/

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6. PeptideFox: Tesamorelin

Evidence type: Community / practitioner protocol

Useful for documenting:

- community dosing
- community timing
- cycling practices
- community-reported results timing

It should not override FDA labeling or controlled human evidence.

`https://peptidefox.com/content/tesamorelin`

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7. Stanley et al.: Tesamorelin in HIV-Associated NAFLD

Evidence type: Randomized human clinical research

Supports:

- 12-month Tesamorelin treatment
- significant reduction in hepatic fat fraction
- liver-fat evidence in people with HIV and NAFLD

Does **not** establish Tesamorelin as a general MASLD/NAFLD treatment outside the population studied.

`https://pubmed.ncbi.nlm.nih.gov/31611038/`

Final Takeaway

Tesamorelin has stronger human evidence than most compounds commonly discussed in peptide research, but that evidence applies mainly to one specific clinical use.

The best-supported protocol is:

Subcutaneous Tesamorelin once daily

Current approved doses are:

EGRIFTA WR: 1.28 mg once daily
EGRIFTA SV: 1.4 mg once daily

Historical clinical research commonly used:

2 mg once daily.

Community research protocols commonly use:

1–2 mg once daily, often at night and away from food.

The **once-daily route and frequency are well supported**.

Bedtime dosing and fasting are **plausible but not proven necessary**.

Automatic 1→2 mg escalation, 5-on/2-off schedules, fixed community cycles and receptor-reset breaks are **community conventions rather than established clinical requirements**.

For results:

Clinical research: measurable abdominal-fat changes were studied by about **12 weeks**, with major outcomes assessed at **26 weeks** and longer research continuing to about **52 weeks**.

Community-reported: body-composition changes are described over **several weeks to a few months**, with PeptideFox specifically placing the beginning of noticeable or measurable changes around **4–6 weeks**. This is anecdotal rather than a true calculated average.

And the formulation warning should remain highly visible:

> **EGRIFTA WR and EGRIFTA SV are not interchangeable. Their dose, preparation and storage instructions are different.**

L-Carnitine (Injectable)

Fat Transport • Mitochondrial Energy • Carnitine Deficiency Research

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At a Glance

What is L-Carnitine?

L-Carnitine is a naturally occurring compound that helps move long-chain fatty acids into mitochondria, where they can be used to make energy.

Think of it as part of the transport system that helps fat reach the part of the cell where it can be used as fuel.

Injectable pharmaceutical L-Carnitine is usually called **levocarnitine**.

L-Carnitine is **not a peptide, hormone or stimulant**.

How well studied is it?

Well studied medically, but not for modern fitness injections

L-Carnitine has been studied in humans for decades, and an FDA-approved injectable version exists.

But there is an important difference between:

medical IV levocarnitine

and:

IM or SC L-Carnitine used in fitness and peptide communities for fat loss, energy or performance.

The strongest human evidence is for treating **carnitine deficiency**, including certain inherited metabolic disorders and deficiency in people receiving hemodialysis.

The FDA-approved injectable route is **IV**.

Evidence level

Approved-label human evidence: Strong for carnitine deficiency
Human clinical research: Extensive, but depends heavily on the condition and route
Fat loss / exercise research: Mixed

IM/SC fitness protocols: Limited
Community protocols: Common, but not equivalent to clinical evidence

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What may it help with?

Strong human evidence

Carnitine deficiency

Levocarnitine can replace carnitine in people whose levels are too low because of certain inherited metabolic disorders.

Dialysis-related carnitine deficiency

People receiving hemodialysis can lose carnitine. Repeated IV levocarnitine reliably raises blood carnitine levels in this population.

Normal fat transport

Carnitine's role in helping long-chain fatty acids reach mitochondria is well established.

Some human evidence

Increasing carnitine inside muscle

Human research shows that muscle carnitine can be increased under the right conditions.

One 24-week study used oral L-carnitine L-tartrate with carbohydrate and increased muscle carnitine by about **21%**.

Exercise fuel use

Researchers have studied whether more carnitine changes how the body uses fat and carbohydrates during exercise.

There is some evidence that carnitine availability can affect fuel use.

That does **not** prove injectable L-Carnitine is a powerful performance enhancer.

Body weight and fat mass

A review of randomized studies found a **small average reduction** in body weight, BMI and fat mass with L-Carnitine supplementation, especially in people with overweight or obesity.

Most of that research was **not based on modern IM or SC injection protocols**.

What has it NOT been proven to help with?

Injectable L-Carnitine has **not** been proven to reliably cause:

- rapid fat loss
- belly-fat targeting
- fat loss at the injection site
- large weight loss on its own
- major muscle growth
- a dramatic increase in metabolism
- guaranteed energy improvement
- reliable athletic-performance improvement
- prevention of GLP-1 fatigue
- fat loss without a calorie deficit

L-Carnitine is needed for normal fat metabolism.

That does **not** mean:

> more L-Carnitine automatically means more fat burned.

Dose at a Glance

Clinical / FDA-approved injectable dosing

There is **not one universal clinical dose**.

The FDA-approved CARNITOR injection is given **intravenously**.

Certain inherited metabolic disorders

Dose: 50 mg/kg
Route: IV
Frequency: Depends on the medical situation

The label uses 50 mg/kg by slow IV injection or infusion.

During a serious metabolic crisis, doses may be repeated more often under medical supervision.

Hemodialysis-related deficiency

Starting dose: 10–20 mg/kg dry body weight
Route: IV
Timing: After dialysis

The dose can be adjusted based on blood carnitine levels.

Research-community dosing

Community injection protocols look very different from the FDA-approved protocol.

PeptideFox

Current PeptideFox material describes:

500 mg IM
1–3 times per week

It also describes a loading approach of:

1,000 mg daily for about 14 days

followed by:

500 mg 2–3 times weekly

PeptideDosages

PeptideDosages describes a lower-dose SC approach:

Weeks 1–2: 50 mg SC daily
Week 3 onward: 100 mg SC daily
Higher community doses: up to 150–200 mg daily
Duration: about 8–16 weeks

Evidence level

Community protocols

There is no healthy-human trial proving that:

- 500 mg IM is the best dose
- 1,000 mg IM is the best loading dose
- 100 mg SC is the best daily dose
- daily SC is better than intermittent IM
- a 14-day loading phase is necessary

Frequency at a Glance

Clinical

Hemodialysis

Usually given after dialysis sessions.

Some clinical studies used IV levocarnitine **three times per week after dialysis**.

Metabolic disorders

Frequency depends on the condition.

During a serious metabolic crisis, IV doses may be given much more often.

Community

IM

Commonly **1–3 times per week**.

SC

Commonly **once daily**.

Best-supported fitness frequency?

Unknown.

No controlled human study has shown that daily SC or intermittent IM is the better body-composition protocol.

Timing at a Glance

Clinical

For dialysis, the approved dose is given **after the dialysis session**.

Outside of specific medical instructions, there is no general FDA requirement to take injectable L-Carnitine:

- fasted
- in the morning
- before exercise
- after exercise
- with carbohydrates

Community

Two common approaches are discussed.

Fasted / pre-workout

Some community protocols use L-Carnitine in the morning or before exercise while fasted.

With carbohydrates

Other protocols combine L-Carnitine with carbohydrates because insulin can help move carnitine into muscle.

Human research does support the basic idea that **insulin can help muscle take up carnitine**.

Evidence verdict

Carbohydrate / insulin-assisted muscle uptake: Supported in human research

Exact injectable carbohydrate protocol: Not proven

Fasted dosing: Makes biological sense, but better fat loss has not been proven

Pre-workout timing: Has been studied experimentally, but has not been shown to be superior

---

Duration / Cycle at a Glance

Clinical

There is **no universal cycle**.

Treatment lasts as long as carnitine replacement is medically needed.

Repeated IV treatment has been studied over weeks and months.

Community

Common schedules include:

- 8–16 weeks of daily SC use
- a 14-day IM loading phase followed by maintenance
- ongoing intermittent IM use

Does it need to be cycled?

No receptor-reset cycle has been established.

L-Carnitine is not a drug that repeatedly switches a receptor on.

There is no proven reason for:

- 5 days on / 2 days off
- 8 weeks on / 4 weeks off
- a receptor reset
- a mandatory washout

Expected Results Timeline

Clinical research

Immediately after IV dosing

Blood carnitine levels rise quickly after IV administration.

Within the first week

In a dialysis study using 20 mg/kg after dialysis three times per week, blood carnitine levels were already substantially higher after the first week.

8–24 weeks

Repeated treatment kept blood carnitine levels elevated over longer periods.

Around 24 weeks for muscle loading

Getting more carnitine into **muscle** can take much longer than raising blood levels.

In one human study using L-Carnitine plus carbohydrate, muscle carnitine increased significantly after **24 weeks**.

Clinical takeaway

Blood levels can rise quickly. Muscle loading and longer-term body-composition changes can take much longer.

Community-Reported Timeline

Community sources report:

Within hours: the compound is available in the body

1–2 weeks: some people report changes in energy

4–8 weeks: some report body-composition changes when diet and exercise are also controlled

Important

These are **community reports**, not averages from controlled IM or SC trials.

Navaro should call this:

> **Commonly reported community timeline**

not:

> **Average time to results**

Claims that muscle becomes "fully loaded" after about two weeks of injections should also remain labeled as community claims.

Simple Timeline for the Top Card

Clinical: blood levels rise quickly, but muscle loading and longer-term effects can take **weeks to months**.

Community-reported: some people report energy changes within **1–2 weeks** and body-composition changes around **4–8 weeks**, usually alongside diet and exercise.

Safety at a Glance

Common or important effects

Reported effects include:

- nausea
- vomiting
- body odor
- stomach irritation
- diarrhea
- headache
- dizziness
- injection-site reactions
- pain

Important safety concerns

Seizures

Seizures have been reported in people both with and without a previous seizure history.

People who already have seizures have also reported an increase in seizure frequency or severity.

Serious allergic reactions

Reported reactions include:

- anaphylaxis
- swelling of the throat
- bronchospasm

Warfarin interaction

Levocarnitine may increase INR in people taking warfarin.

INR may need closer monitoring after starting L-Carnitine or changing the dose.

Major contraindications

Formal FDA contraindications

None known.

That does **not** mean there are no risks.

Important cautions include:

- seizure history
- warfarin use
- previous serious allergic reaction
- pregnancy
- breastfeeding
- severe kidney disease depending on the route and situation

Regulatory Status at a Glance

FDA approved?

Yes

Prescription injectable levocarnitine is FDA approved.

FDA-approved injectable route?

IV

CARNITOR injection is labeled:

For intravenous use only.

Approved for

- carnitine deficiency caused by certain inherited metabolic disorders
- prevention and treatment of carnitine deficiency in people with end-stage kidney disease receiving dialysis

FDA approved for IM bodybuilding use?

No

FDA approved for SC fat-loss use?

No

FDA approved for ordinary weight loss?

No

Bottom Line

L-Carnitine itself is well studied. Modern IM and SC fitness protocols are much less studied.

The strongest clinical evidence supports:

IV levocarnitine for true carnitine deficiency.

Approved dosing includes:

50 mg/kg IV for certain metabolic disorders

and:

10–20 mg/kg IV after dialysis for dialysis-related deficiency.

Community protocols are very different.

PeptideFox describes:

500 mg IM 1–3 times weekly

with higher loading approaches.

PeptideDosages describes:

50–100 mg SC daily

with some protocols going as high as 150–200 mg daily.

These community protocols are worth showing because people actually use them.

But they should **not** be presented as if they are the same as FDA-approved treatment.

Protocol Logic Check

Quick Logic Summary

Protocol Question	Verdict
Does L-Carnitine help transport fatty acids?	**Yes, well established**
Is the FDA-approved injection IV?	**Yes**
Is 50 mg/kg IV a real clinical dose?	**Yes, for certain metabolic disorders**
Is 10–20 mg/kg after dialysis supported?	**Yes**
Is 500 mg IM 1–3x/week proven?	**Community protocol**
Is 50–100 mg SC daily proven?	**Community protocol**
Is a 14-day loading phase necessary?	**Not proven**
Does fasted dosing make sense?	**Possible, but not proven better**
Can insulin help muscle take up carnitine?	**Yes**
Is one exact carb-paired injection protocol proven?	**No**
Is pre-workout timing proven better?	**No**
Does it need 5 days on / 2 off?	**No proven reason**
Does it need an 8–16 week cycle?	**No proven biological reason**
Does it need a receptor reset?	**No**
Does injecting into belly fat burn belly fat?	**No evidence**
Can every vial use the same 2–4 week storage rule?	**No**
Does injectable L-Carnitine make zero TMAO?	**Not established**

What Does L-Carnitine Actually Target?

L-Carnitine does not work by switching one receptor on.

It is part of the **carnitine shuttle**, which helps move long-chain fatty acids into mitochondria.

Simple version

Fatty acids → L-Carnitine transport system → mitochondria → energy

Important parts of this system include:

- CPT-1
- carnitine-acylcarnitine translocase
- CPT-2

The body also uses transport proteins such as OCTN2 to move carnitine into cells.

Verdict

Well established

Human Half-Life

After one **20 mg/kg IV dose**, the CARNITOR label reports:

Distribution half-life: about 0.6 hours

Terminal half-life: about 17 hours

Why this matters

This does **not** mean:

> take another dose every 17 hours.

Blood half-life does not tell us:

- how fast muscle loads with carnitine
- the best IM or SC schedule
- how quickly body composition changes
- when a maintenance injection should happen

Verdict

We know how long it stays in the blood, but that does not tell us the best fitness dosing schedule.

Blood Levels vs Muscle Levels

L-Carnitine can rise quickly in the blood after an injection.

Getting more carnitine into muscle can take much longer.

Verdict

Blood levels and muscle levels are not the same thing.

Does It Build Up?

Repeated IV treatment can raise blood carnitine considerably.

But muscle does not simply absorb unlimited carnitine because the blood level is high.

Muscle uptake is controlled by the body.

Verdict

Yes, blood levels can build up. Muscle uptake is much more limited and controlled.

Does Daily Dosing Make Sense?

Daily treatment can make sense in some medical deficiency states.

Daily SC dosing is also common in community protocols.

But no controlled healthy-person study has shown that:

daily SC L-Carnitine works better than intermittent IM L-Carnitine for fat loss or performance.

Verdict

Daily dosing can make sense in some settings, but daily SC fitness use has not been proven superior.

Does 1–3 Times Weekly Make Sense?

PeptideFox uses this frequency in its IM protocol.

Three-times-weekly dosing also appears in dialysis research.

But those are **not the same thing**.

Dialysis studies used:

- IV administration
- weight-based dosing
- people with a medical deficiency

- dosing after dialysis

Community protocols use:

- IM injections
- fixed doses
- generally non-deficient people
- fat-loss or performance goals

Verdict

The schedules may look similar, but they are not the same protocol.

Does Fasted Dosing Make Sense?

When you are fasted, the body tends to rely more heavily on fat for fuel.

That gives fasted L-Carnitine use some biological logic.

But L-Carnitine helps transport fatty acids.

It does **not** force stored body fat to be released.

No strong human injection study has shown that fasted IM or SC L-Carnitine produces better fat loss.

Verdict

Makes sense in theory, but better fat loss has not been proven.

Does Taking It With Carbohydrates Make Sense?

There is more evidence behind this idea.

Human research shows that insulin can help skeletal muscle take up carnitine when blood carnitine is high.

A later 24-week study using L-Carnitine with carbohydrate increased muscle carnitine by about **21%**.

What this supports

Insulin can help muscle take up carnitine.

What it does not prove

Every injection needs exactly 60–80 g of carbohydrate.

or:

Two weeks of carb-paired injections will fully load the muscle.

Verdict

The basic idea is supported in humans, but the exact community injection protocol has not been proven.

Pre-Workout Timing

One human experiment used:

3 g IV L-Carnitine about 40 minutes before exercise.

So pre-exercise use has actually been studied.

But that does **not** prove:

- 200 mg SC pre-workout is ideal
- 500 mg IM pre-workout is ideal
- taking it before exercise produces more long-term fat loss

Verdict

Pre-workout use has been studied, but it has not been shown to be the best timing.

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5 Days On / 2 Days Off

L-Carnitine does not work by repeatedly stimulating a receptor.

There is no reason to assume taking weekends off "resets" how it works.

Verdict

No proven reason for this schedule.

8–16 Week Cycle

Some community sources use an 8–16 week schedule.

There is no known event at week 8, 12 or 16 that suddenly makes L-Carnitine stop working or requires a break.

Verdict

Community schedule, not a biological requirement.

Does It Need a Receptor Reset?

No.

L-Carnitine is not a receptor agonist.

It is part of normal metabolism and transport.

The body can control how much carnitine enters cells, but that is **not the same thing as a receptor becoming desensitized**.

Verdict

No receptor-reset reason.

Does It Need a Washout?

There is no established:

- 2-week washout
- 4-week break
- receptor recovery period

for L-Carnitine.

Verdict

No proven biological requirement.

Dose Escalation / Titration

Some community protocols use:

50 mg → 100 mg → 150 mg

or similar increases.

There is no matching randomized human study proving this is the ideal way to increase L-Carnitine.

Starting lower may make sense simply to see how well someone tolerates it.

That is different from a medically proven titration schedule.

Verdict

Community titration, not a study-proven schedule.

Does the Route Match the Research?

FDA CARNITOR

IV

Human exercise research

IV

PeptideFox community use

IM

PeptideDosages community use

SC

The approved clinical route and modern fitness routes are not the same.

Verdict

IM and SC fitness protocols should not be presented as if they are the FDA-approved route.

Does Injecting Into Belly Fat Burn Belly Fat?

No evidence shows that injecting L-Carnitine into abdominal fat causes that area to lose more fat.

L-Carnitine works through normal metabolism throughout the body.

Verdict

No evidence for spot fat loss.

What About TMAO?

This needs careful wording.

Oral L-Carnitine can be broken down by gut bacteria into compounds that can eventually become TMAO.

Injecting L-Carnitine avoids the first pass through the gut.

That gives injectable use a reasonable reason to produce a different TMA/TMAO pattern.

But there is not enough route-specific human evidence to promise:

> Injectable L-Carnitine produces zero TMAO.

Verdict

Injectable and oral L-Carnitine may differ, but "zero TMAO" is too strong a claim.

Stack / Pairing Logic

L-Carnitine + Carbohydrates / Insulin

Human research supports insulin helping muscle take up carnitine.

Verdict: The basic idea is supported, but there is no proven exact injection-and-carb protocol.

L-Carnitine + GLP-1 Agonists

They work differently.

Verdict: They are not redundant, but there is no good evidence that L-Carnitine prevents GLP-1 fatigue or makes GLP-1 weight loss better.

L-Carnitine + MOTS-c

Different metabolic pathways.

Verdict: The combination makes sense on paper, but has not been properly tested in humans.

L-Carnitine + NAD+

Different roles in energy metabolism.

Verdict: Possible complementary effects, but no validated combination protocol.

L-Carnitine + SS-31

Different mitochondrial mechanisms.

Verdict: Possible complementary effects, but the combination has not been proven.

Formulation / Stability / Sterility Logic

The FDA-approved CARNITOR injection is already a **sterile liquid**.

It contains:

1 g in 5 mL

which equals:

200 mg/mL

It is supplied as a:

single-dose, preservative-free vial.

Unused solution should be discarded after opening.

Why this matters

Not every product sold as injectable L-Carnitine is the same.

A compounded IM vial, SC vial or lyophilized research product may have completely different:

- concentration
- ingredients
- preservative
- storage
- expiration after opening

Verdict

Use the instructions for the exact product. Do not apply one storage rule to every L-Carnitine vial.

Important Research Note

Three very different types of L-Carnitine use are often mixed together online.

1. FDA-Approved IV Levocarnitine

This is a prescription medication with:

- a tested formulation
- IV administration
- weight-based clinical dosing
- human pharmacokinetic data
- specific medical uses
- product-specific handling instructions

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2. Human Research

Researchers have studied L-Carnitine for:

- exercise metabolism
- muscle uptake
- insulin-assisted muscle loading
- body composition
- metabolic conditions

These studies use different forms and routes, including:

- oral
- IV

They should not automatically be used to prove an IM or SC community protocol works the same way.

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3. Community Injection Protocols

Fitness and peptide communities commonly use:

- higher-dose IM injections
- lower-dose daily SC injections
- fasted use
- pre-workout use
- carbohydrate pairing
- loading and maintenance phases

These protocols are useful to document because people actually use them.

But they should stay clearly labeled as:

Community protocols

rather than:

Established clinical protocols

Research at a Glance

Approved-Label Human Evidence

Strong

There is an FDA-approved IV levocarnitine product with clear human dosing, safety and pharmacokinetic information.

Human Clinical Research

Extensive, but depends on the situation

Human studies include:

- carnitine deficiency
- dialysis
- exercise
- muscle loading
- metabolism
- body composition

Observational Human Evidence

Supporting

Decades of medical use provide additional safety and treatment experience.

Animal / Cell Evidence

Extensive

Useful for understanding how carnitine transport and mitochondria work.

Mechanistic Evidence

Strong

Carnitine's role in transporting long-chain fatty acids into mitochondria is well established.

Community / Anecdotal Evidence

Common, but much weaker

IM and SC protocols are common in fitness and peptide communities but are not validated to the same level as clinical IV levocarnitine.

Overview

L-Carnitine is a naturally occurring amino-acid derivative involved in energy metabolism.

Its best-known role is helping move long-chain fatty acids into mitochondria.

That makes L-Carnitine necessary for normal fat burning.

But:

> **Being necessary for fat metabolism does not mean taking more L-Carnitine automatically causes more fat loss.**

For someone who is deficient, replacing carnitine can be medically important.

For a healthy person who already has enough carnitine, the benefit of injecting extra L-Carnitine is much less certain.

Evidence Strength

Overall Evidence Rating

Strong human evidence for deficiency treatment; much weaker evidence for modern IM/SC fitness use

Strong evidence for

- normal fatty-acid transport
- IV levocarnitine pharmacology
- treatment of some carnitine deficiencies
- dialysis-related carnitine replacement
- increasing blood carnitine
- insulin helping muscle take up carnitine

Some human evidence for

- increasing muscle carnitine
- exercise fuel use
- modest body-weight and fat-mass changes
- some metabolic outcomes

Limited or unproven for

- high-dose IM bodybuilding protocols
- daily SC fat-loss protocols

- rapid fat loss
- belly-fat targeting
- GLP-1 fatigue treatment
- fasted superiority
- pre-workout superiority
- two-week muscle saturation
- fixed 8–16 week cycles

Category

Metabolic Support / Fat Transport / Mitochondrial Energy

Compound

L-Carnitine

Pharmaceutical name:

Levocarnitine

FDA-approved brand example:

CARNITOR

Compound Type

Naturally occurring amino-acid derivative

L-Carnitine is not:

- a peptide
- a hormone
- a stimulant
- a GLP-1 agonist
- a growth-hormone secretagogue

Primary Research Focus

Best Established

- carnitine deficiency

- fatty-acid transport
- inherited metabolic disorders
- dialysis-related carnitine deficiency

Also Studied

- muscle carnitine uptake
- exercise fuel use
- insulin-assisted muscle loading
- body composition
- metabolic health

Not Established

- major standalone fat loss
- spot fat loss
- bodybuilding enhancement
- guaranteed energy improvement
- treatment of GLP-1 fatigue

How It Works / Transport Mechanism

L-Carnitine helps long-chain fatty acids get into mitochondria.

Simple version

Think of mitochondria as the cell's engines.

Fatty acids need help getting inside those engines.

L-Carnitine is part of that transport system.

Pathway

Fatty acid → L-Carnitine transport system → mitochondria → energy

Important

L-Carnitine helps the body **use fatty acids that are already available**.

It does not automatically tell fat cells to release stored body fat.

Common Research Dose

Approved Clinical Dosing

Certain Metabolic Disorders

50 mg/kg IV

Hemodialysis

10–20 mg/kg IV after dialysis

Human Exercise Research

One acute human experiment used:

3 g IV about 40 minutes before exercise

This was a research experiment, not an approved fitness protocol.

Research Community

PeptideFox

500 mg IM, 1–3 times weekly

Higher loading protocols are also described.

PeptideDosages

50–100 mg SC daily

with some protocols increasing toward:

150–200 mg daily

Research Duration / Cycle Length

Clinical

No universal cycle.

Treatment depends on why the person needs carnitine.

Human Muscle-Loading Research

One important muscle-loading study lasted:

24 weeks

Research Community

Common schedules include:

- 14-day loading followed by maintenance
- 8–16 weeks of daily SC use
- ongoing intermittent IM use

Evidence assessment

There is **no receptor-based reason** L-Carnitine must be cycled.

How Often It Is Used / Frequency

Clinical

Depends on the medical condition

Dialysis

Usually after dialysis sessions.

Research Community

Commonly either:

daily SC

or:

1–3 times weekly IM

No good human study shows which fitness schedule is better.

Possible Benefits

Strong Human Evidence

Correcting Carnitine Deficiency

Levocarnitine can replace carnitine that is abnormally low because of certain metabolic disorders or dialysis.

Supporting Normal Fat Transport

Carnitine is part of the body's normal system for moving fatty acids into mitochondria.

Some Human Evidence

Increasing Muscle Carnitine

Long-term supplementation with insulin/carbohydrate support can increase muscle carnitine.

One 24-week study found about a:

21% increase

in muscle carnitine.

Body Weight / Fat Mass

A review of human studies found small average reductions in:

- body weight
- BMI
- fat mass

Exercise Metabolism

Human experiments show that carnitine can change fuel use under some conditions.

That does **not** prove injectable L-Carnitine is a major performance enhancer.

What It Does Not Help With, Based on Current Evidence

Do not present L-Carnitine injections as proven to:

- cause rapid weight loss
- burn fat without a calorie deficit
- target belly fat
- burn fat where it is injected
- build significant muscle on their own
- reliably improve athletic performance
- prevent GLP-1 fatigue
- dramatically raise resting metabolism
- replace diet or exercise

Commonly Paired With

Carbohydrates / Insulin

Human research supports insulin helping muscle take up carnitine.

Classification: The basic idea is supported, but there is no proven exact injection protocol.

GLP-1 Agonists

They work through different pathways.

Classification: Not redundant, but there is no established evidence that L-Carnitine prevents GLP-1 fatigue or improves GLP-1 weight loss.

MOTS-c

Different metabolic pathways.

Classification: Makes sense in theory, but the combination has not been properly tested.

NAD+

Different role in energy metabolism.

Classification: Could be complementary, but no validated combination protocol exists.

SS-31

Different mitochondrial pathway.

Classification: Could be complementary, but the combination is unproven.

Dosage Information

CARNITOR Injection

Product: CARNITOR
Compound: Levocarnitine
Strength: 1 g per 5 mL
Concentration: 200 mg/mL
Form: Sterile liquid solution
Route: IV only
Vial: Single dose
Preservative: None

Metabolic Disorders

Recommended dose:

50 mg/kg IV

given as a slow **2–3 minute injection or infusion**.

During a severe metabolic crisis, the label allows more frequent dosing under medical supervision.

Clinical monitoring can include:

- blood carnitine
- blood chemistry
- vital signs
- overall clinical response

Hemodialysis

Recommended starting dose:

10–20 mg/kg dry body weight IV

given:

after dialysis

The dose can be adjusted using blood carnitine levels.

Community Research Protocol

PeptideFox IM Protocol

Current PeptideFox material describes:

Dose: 500 mg
Route: IM
Frequency: 1–3 times weekly

It also describes a loading approach:

Loading: 1,000 mg daily for about 14 days
Maintenance: 500 mg 2–3 times weekly

Evidence Check

IM route: Community use, not the approved route

500 mg dose: Community protocol

1–3 times weekly: Community protocol

14-day loading: Community convention

Carbohydrate pairing: Basic idea has human support

Full muscle loading in about 14 days: Not proven in a matching human IM trial

---

PeptideDosages SC Protocol

PeptideDosages describes:

Weeks 1–2: 50 mg SC daily
Weeks 3 onward: 100 mg SC daily
Higher community use: up to 150–200 mg daily
Duration: about 8–16 weeks

Evidence Check

This is a community protocol.

The exact:

- SC route
- 50 → 100 mg increase
- 8–16 week cycle
- 150–200 mg higher dose

have not been established by a matching randomized human study.

Community Protocol Comparison

Source	Dose	Route	Frequency	Duration	Evidence
CARNITOR metabolic disorders	50 mg/kg	IV	Depends on condition	Clinical	FDA label
CARNITOR dialysis	10–20 mg/kg	IV	After dialysis	Clinical	FDA label
Acute exercise study	3 g	IV	Single experimental dose	Acute	Human research
PeptideFox	500 mg	IM	1–3x/week	Variable	Community
PeptideFox loading	1,000 mg	IM	Daily	~14 days then maintenance	Community
PeptideDosages	50–100+ mg	SC	Daily	~8–16 weeks	Community

Strongest Agreement

There is **no single dose that all sources agree on**.

What is actually clear

- IV levocarnitine has tested clinical dosing
- IM community protocols exist
- SC community protocols exist
- these should not be blended into one “standard dose”

Expected Results Timeline, Expanded

Human Clinical Research

Immediately

IV L-Carnitine quickly raises blood carnitine.

Around 1 Week

Repeated dialysis dosing can substantially raise blood levels within the first week.

8–24 Weeks

Repeated treatment can keep blood levels elevated over time.

Around 24 Weeks

A human muscle-loading study showed a meaningful rise in muscle carnitine after **24 weeks**.

Community-Reported

Community sources report:

1–2 weeks: some people notice changes in energy

4–8 weeks: some report body-composition changes when diet and exercise are also controlled

Evidence Warning

These are **community reports**, not controlled IM or SC trial averages.

Claims that muscle becomes fully loaded in about two weeks should remain labeled as community claims.

Reported Side Effects

Label-Supported Effects

Reported effects include:

- nausea
- vomiting
- body odor
- stomach irritation
- diarrhea
- headache
- dizziness
- injection-site reactions
- pain

Seizures

Seizures have been reported in people with and without a previous seizure history.

People with seizure disorders have also reported more frequent or more severe seizures.

Serious Allergic Reactions

Reported reactions include:

- anaphylaxis
- throat swelling
- bronchospasm

Warfarin Interaction

Levocarnitine may increase INR in people taking warfarin.

Closer monitoring may be needed after starting it or changing the dose.

Contraindications & Cautions

Formal Contraindications

None known

That is the FDA-label wording.

This does **not** mean there are no important cautions.

Important Cautions

Seizure History

Use caution because new or worsening seizures have been reported.

Warfarin

INR may increase.

Previous Serious Allergic Reaction

Serious allergic reactions have occurred.

Pregnancy

Good controlled pregnancy studies are limited.

Breastfeeding

The risks and benefits should be considered carefully.

Severe Kidney Disease

This needs context.

The label warns about buildup of TMA/TMAO from **long-term high-dose oral L-Carnitine** in severe kidney dysfunction.

That does **not** mean kidney disease is a blanket contraindication to injectable L-Carnitine.

IV levocarnitine is actually used medically in dialysis patients.

Regulatory Status

United States

FDA-approved injectable levocarnitine exists.

Approved Route

IV only for CARNITOR injection.

Approved Uses

- certain inherited causes of carnitine deficiency
- prevention and treatment of carnitine deficiency in people with end-stage kidney disease receiving dialysis

Not FDA Approved For

- IM bodybuilding protocols
- SC fat-loss protocols
- ordinary weight loss
- body recomposition
- sports performance
- GLP-1 fatigue

What Studies Have Found

Clinical IV / Dialysis Research

In one nine-week study, people receiving hemodialysis were given:

20 mg/kg IV three times per week after dialysis

Blood carnitine levels rose substantially.

What This Supports

- IV use
- repeated post-dialysis dosing
- correction of low blood carnitine
- three-times-weekly treatment in this specific medical setting

What It Does Not Support

- 500 mg IM for fat loss
- 100 mg SC every day
- pre-workout SC protocols
- bodybuilding protocols

Acute Exercise Research

One healthy-human experiment used:

3 g IV L-Carnitine

about:

40 minutes before exercise

What This Supports

- IV L-Carnitine has been studied before exercise
- researchers have looked at its effects on exercise metabolism

What It Does Not Support

- 200 mg SC as an equivalent protocol
- 500 mg IM as an equivalent protocol
- guaranteed extra fat burning
- a standard pre-workout recommendation

Muscle Carnitine Loading Research

A 24-week human study used L-Carnitine with carbohydrate and found about a:

21% increase in muscle carnitine

What This Supports

- muscle carnitine can be increased
- insulin/carbohydrate availability can help
- muscle loading can take months

What It Does Not Support

- a 14-day IM loading protocol
- guaranteed full muscle saturation in two weeks
- one exact carbohydrate dose for injections

Body Weight / Fat-Mass Research

A review of human studies found a **modest** average effect on:

- body weight
- BMI
- fat mass

especially in people with overweight or obesity.

What This Supports

L-Carnitine may have a small body-composition effect in some people.

What It Does Not Support

- dramatic fat loss
- rapid injection-based fat loss
- spot reduction
- large results without diet or activity changes

Formulation / Stability / Sterility

CARNITOR Injection

1 g per 5 mL
200 mg/mL
Sterile liquid solution
Single-dose vial
No preservative
IV only

Storage

Store unopened vials at controlled room temperature.

Avoid excessive heat.

Do not freeze.

Once opened:

Discard any unused portion because the vial contains no preservative.

IV Compatibility

Under the labeled conditions, CARNITOR can be mixed with:

- 0.9% sodium chloride
- Lactated Ringer's

for IV use under the product's specified conditions.

Compounded / Community Injectable L-Carnitine

Do **not** assume CARNITOR storage rules apply to:

- compounded IM products
- SC multidose vials
- lyophilized research products
- vials mixed with bacteriostatic water

A rule such as:

> "Injectable L-Carnitine lasts 2–4 weeks after mixing"

cannot be applied to every product.

Simple rule

Use storage instructions for the exact product and formulation.

Research Sources

1. DailyMed: CARNITOR Levocarnitine Injection

Evidence type: FDA-approved prescription-drug labeling

Supports:

- FDA-approved **IV route**
- clinical dosing for metabolic disorders
- clinical dosing for dialysis-associated carnitine deficiency
- human pharmacokinetics
- seizure warning
- serious hypersensitivity reactions
- warfarin interaction
- formulation and concentration
- single-dose vial handling
- storage and IV compatibility

`https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=cf801cc4-775e-433d-9d32-e5d9a98981c`

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2. Natali et al.: Acute IV L-Carnitine During Exercise

Evidence type: Human experimental research

Supports:

- **3 g IV L-Carnitine**
- administration approximately **40 minutes before exercise**
- direct human research on acute pre-exercise L-Carnitine metabolism

Does **not** establish a standard IM or SC performance protocol.

`https://pubmed.ncbi.nlm.nih.gov/8492714/`

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3. Stephens et al.: Insulin and Muscle L-Carnitine Uptake

Evidence type: Human mechanistic research

Supports:

- insulin can increase skeletal-muscle carnitine uptake when blood carnitine is elevated

- muscle carnitine uptake is regulated rather than simply determined by blood levels
- the basic biological reasoning behind carbohydrate/insulin-assisted carnitine loading

Does **not** establish one specific carb-paired injectable protocol.

`https://pubmed.ncbi.nlm.nih.gov/16368715/`

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4. Wall et al.: Chronic L-Carnitine + Carbohydrate Muscle Loading

Evidence type: Human controlled research

Supports:

- **24 weeks** of L-Carnitine plus carbohydrate
- approximately **21% increase in muscle total carnitine**
- the idea that meaningful muscle carnitine loading can take months
- carbohydrate/insulin availability helping support muscle carnitine uptake

Does **not** validate a 14-day IM loading protocol.

`https://pubmed.ncbi.nlm.nih.gov/21224234/`

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5. Talenezhad et al.: L-Carnitine and Body Composition Meta-Analysis

Evidence type: Systematic review / meta-analysis of human studies

Supports:

- modest average reductions in **body weight**
- modest reductions in **BMI**
- modest reductions in **fat mass**
- greater relevance in some people with overweight or obesity

Does **not** establish rapid or dramatic fat loss from IM or SC L-Carnitine injections.

`https://pubmed.ncbi.nlm.nih.gov/32359762/`

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6. PeptideFox: Injectable L-Carnitine

Evidence type: Community / practitioner protocol

Useful for documenting:

- **500 mg IM** community dosing

- **1–3 times weekly** use
- community loading approaches
- fasted / pre-workout timing
- carbohydrate pairing
- community-reported results timeline

These are community practices and should not override FDA labeling or controlled human research.

`https://peptidefox.com/content/l-carnitine`

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7. PeptideDosages: L-Carnitine Protocol

Evidence type: Community research protocol

Useful for documenting:

- **50 mg → 100 mg SC** progression
- daily SC use
- higher community doses of approximately **150–200 mg**
- **8–16 week** community framework
- community reconstitution and storage practices

These are not matching FDA-approved or controlled human protocols.

`https://peptidedosages.com/single-peptide-dosages/l-carnitine-200-mg-vial-dosage-protocol/`

Final Takeaway

L-Carnitine is a well-established biological compound and a real FDA-approved medication, but the evidence becomes much weaker when moving from medical IV treatment to modern IM or SC fitness use.

The strongest clinical evidence supports:

IV levocarnitine for true carnitine deficiency.

FDA-labeled dosing includes:

50 mg/kg IV for certain inherited metabolic disorders

and:

10–20 mg/kg IV after dialysis for dialysis-related deficiency.

Community protocols are very different.

PeptideFox describes:

500 mg IM 1–3 times weekly

with a short loading phase also discussed.

PeptideDosages describes:

50–100 mg SC daily

with some community protocols going higher.

Those protocols are useful to show because they reflect how L-Carnitine is actually being used outside medicine.

But they should **not** be presented as if they are FDA-approved or clinically proven equivalents.

For timing:

Insulin helping muscle take up carnitine has real human support.

Fasted and pre-workout use may make sense, but neither has been proven to be the best way to use injectable L-Carnitine.

For results:

Clinical: blood levels can rise quickly, while muscle loading and longer-term changes may take **weeks to months**.

Community-reported: some people report energy changes within **1–2 weeks** and body-composition changes around **4–8 weeks**, usually alongside diet and exercise. These are community reports, not controlled IM/SC averages.

And the most important distinction should remain highly visible:

> **FDA-approved injectable levocarnitine is IV. IM and SC L-Carnitine protocols are community/off-label protocols and should not be presented as if they are the approved clinical route or dose.**

Liraglutide

GLP-1 Receptor Agonist • Weight Management • Type 2 Diabetes

At a Glance

What is Liraglutide?

Liraglutide is a **GLP-1 receptor agonist**.

It copies some of the effects of a natural hormone called GLP-1.

GLP-1 helps control:

- appetite
- fullness
- blood sugar
- insulin release
- glucagon release
- how quickly food leaves the stomach

Natural GLP-1 disappears from the body very quickly.

Liraglutide was designed to last much longer.

Its half-life is about:

13 hours

which makes:

once-daily dosing

possible.

Liraglutide is the active ingredient in products used for different purposes, including:

- **Saxenda** for long-term weight management
- **Victoza and other liraglutide diabetes products** for type 2 diabetes

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How well studied is it?

Very well studied in humans

Liraglutide is one of the better-studied compounds in this library.

It has:

- large randomized human trials

- FDA-approved products
- established clinical dosing
- long-term human follow-up
- cardiovascular research
- pediatric studies
- well-described safety information

Major weight-management trials commonly lasted:

56 weeks

and some participants were followed for up to:

160 weeks

Evidence level

Approved-label human evidence: Very strong
Human clinical research: Very strong
Weight-management evidence: Strong
Type 2 diabetes evidence: Strong
Community protocols: Secondary because strong clinical protocols already exist

---

What may it help with?

Strong human evidence

Weight Loss and Long-Term Weight Management

Saxenda is FDA approved, together with diet and physical activity, to help eligible adults and adolescents lose excess weight and maintain weight reduction.

In one major 56-week obesity trial, average weight loss was about:

8.4 kg with liraglutide 3 mg

compared with:

2.8 kg with placebo

Reduced Appetite and Increased Fullness

Liraglutide helps reduce food intake.

It can:

- decrease appetite
- increase fullness

- make it easier to stop eating sooner

This is one of the main reasons it helps with weight management.

Blood Sugar Control

Liraglutide is also FDA approved to improve blood sugar control in adults and children aged 10 years and older with type 2 diabetes.

Cardiovascular Risk Reduction in Certain People With Type 2 Diabetes

Diabetes-dose liraglutide also has an FDA indication to reduce major cardiovascular events in adults who have:

- type 2 diabetes
- established cardiovascular disease

This should **not** automatically be applied to everyone using Saxenda only for weight management.

What has it NOT been proven to help with?

Liraglutide has **not** been established as a treatment for:

- bodybuilding
- muscle growth
- athletic performance
- injury recovery
- spot fat loss
- selectively burning belly fat
- directly increasing metabolism
- guaranteed energy improvement
- occasional "as needed" appetite control
- producing the same weight-loss result at every dose

For adults using Saxenda for weight management:

3 mg daily is the recommended maintenance dose.

Lower doses are used while the dose is gradually increased.

Dose at a Glance

Clinical / FDA-Approved Dosing

There are **two major liraglutide dosing systems**.

They should not be mixed together.

Saxenda for Weight Management

Adult Titration

Week 1: 0.6 mg once daily
Week 2: 1.2 mg once daily
Week 3: 1.8 mg once daily
Week 4: 2.4 mg once daily
Week 5 onward: 3 mg once daily

Adult Maintenance Dose

3 mg SC once daily

For adults, the lower doses are used to gradually work up to 3 mg.

If a dose increase is not tolerated, the next increase can be delayed by about:

1 extra week

If an adult cannot tolerate:

3 mg daily

the current Saxenda label says treatment should be discontinued.

Victoza / Diabetes Liraglutide

Adults

Week 1: 0.6 mg once daily

Then:

1.2 mg once daily

If more blood sugar control is needed:

1.8 mg once daily

after at least one week at 1.2 mg.

Important

The:

0.6 mg dose

is mainly a starting dose to reduce stomach-related side effects.

In adults, it is **not considered an effective dose for blood sugar control**.

Research-Community Dosing

Liraglutide is different from many research compounds because its clinical dosing is already well established.

Community practices sometimes include:

- staying at a lower dose longer
- increasing more slowly because of nausea
- staying below 3 mg because appetite is already strongly reduced

Evidence level

Community modification of an established clinical protocol

Slower increases for tolerability can make sense.

But for adult Saxenda:

staying permanently below 3 mg is not the current FDA-approved weight-management protocol.

Frequency at a Glance

Clinical

Once daily

This applies to both:

- Saxenda
- diabetes liraglutide products

Twice daily?

Not established

Every other day?

Not established

5 days on / 2 days off?

Not established

Only when hungry?

Not established

Best-supported frequency

Once daily

Timing at a Glance

Clinical

Liraglutide can be taken:

at any time of day

and:

with or without food

It is injected under the skin into the:

- abdomen
- thigh
- upper arm

Injection sites should be rotated.

Evidence verdict

Morning required: No
Night required: No
Fasted required: No
With food required: No
Pre-workout required: No
Post-workout required: No

Duration / Cycle at a Glance

Clinical

Liraglutide is designed for:

ongoing treatment

rather than short peptide-style cycles.

Major weight-management studies lasted about:

56 weeks

with some longer research extending to:

160 weeks

Does it need to be cycled?

No receptor-reset cycle has been established.

There is no proven need for:

- 8 weeks on / 4 weeks off
- 12-week cycles
- 5 days on / 2 days off
- 2–4 week receptor resets

Expected Results Timeline

Clinical Research

First Few Weeks

Appetite and fullness may start changing while the dose is being increased.

But the first four weeks are also the titration period, so early weight-loss results can vary.

Around 16 Weeks

The current Saxenda label uses an important adult checkpoint at:

16 weeks after starting treatment

If an adult has not lost at least:

4% of starting body weight

by this point, the label recommends stopping treatment because meaningful long-term weight loss is unlikely.

Around 56 Weeks

Major weight-management studies commonly measured their main results at:

56 weeks

In the SCALE obesity trial:

Liraglutide: about 8.4 kg average weight loss

Placebo: about 2.8 kg average weight loss

Up to 160 Weeks

Some participants with abnormal blood sugar measurements were followed for up to:

160 weeks

Clinical Takeaway

For weight management:

think in months, not days.

Community-Reported Timeline

Community experiences commonly describe:

Days to a few weeks: less hunger or feeling full sooner

Several weeks: gradual changes on the scale

Several months: more noticeable changes in body weight and waist size

Important

These are community descriptions.

They are **not controlled averages from clinical trials**.

Because liraglutide already has strong clinical research, the clinical timeline should remain the main timeline on the page.

Simple Timeline for the Top Card

Clinical: appetite changes may begin during titration, adult response is checked at **16 weeks**, major weight-loss results are commonly measured around **56 weeks**, and some research extends to **160 weeks**.

Community-reported: appetite changes are often noticed before major weight changes, which usually develop over **weeks to months**.

Safety at a Glance

Common or Important Effects

Common Saxenda effects include:

- nausea
- diarrhea
- constipation
- vomiting
- injection-site reactions
- headache
- indigestion
- fatigue
- dizziness
- abdominal pain
- increased lipase

Stomach-related side effects are especially common while the dose is being increased.

Important Safety Concerns

Thyroid C-Cell Tumors

Liraglutide caused thyroid C-cell tumors in animal studies.

It is not known whether it causes these tumors in humans.

Because of this, Saxenda should not be used by people with:

- a personal or family history of medullary thyroid carcinoma
- MEN 2

Pancreatitis

Acute pancreatitis has been reported with GLP-1 receptor agonists including liraglutide.

Severe or ongoing abdominal pain should be medically evaluated.

Gallbladder Disease

Liraglutide can increase the risk of:

- gallstones
- gallbladder inflammation

Hypoglycemia

Low blood sugar is especially important when liraglutide is combined with:

- insulin
- sulfonylureas
- other medicines that increase insulin release

Increased Heart Rate

Saxenda can increase resting heart rate.

Kidney Injury From Dehydration

Severe or ongoing:

- nausea
- vomiting
- diarrhea

can lead to dehydration.

That dehydration can contribute to acute kidney injury.

Severe Stomach and Digestive Effects

Saxenda can cause severe gastrointestinal side effects.

It is **not recommended for people with severe gastroparesis**.

Serious Allergic Reactions

Serious reactions including:

- anaphylaxis
- angioedema

have been reported.

Aspiration During Anesthesia or Deep Sedation

Liraglutide can slow stomach emptying.

That means food may remain in the stomach longer.

Rare aspiration events have been reported during procedures involving:

- general anesthesia
- deep sedation

People taking liraglutide should tell the medical team before surgery or sedation.

Major Contraindications

Formal FDA Contraindications

Personal or Family History of Medullary Thyroid Carcinoma

Contraindicated

MEN 2

Contraindicated

Serious Allergy to Liraglutide or Product Ingredients

Contraindicated

Important 2026 Safety Update

Older Saxenda information may still list:

suicidal behavior or suicidal thoughts

as a warning.

That information is now outdated.

In February 2026, FDA approved removal of this warning from Saxenda's prescribing information.

FDA's broader review found that the available evidence did **not** support a causal link between GLP-1 receptor agonists and suicidal thoughts or behavior.

What Navaro Should Do

The current live page should **not** list:

> "History of suicidal ideation or prior suicide attempts"

as a current Saxenda contraindication or risk flag.

If mentioned at all, it should only appear as a:

historical regulatory note

Regulatory Status at a Glance

FDA approved?

Yes

Saxenda approved for

Long-term weight management in eligible adults and adolescents

Diabetes liraglutide approved for

Type 2 diabetes treatment, with cardiovascular-risk reduction in certain adults with established cardiovascular disease

Approved route

Subcutaneous

Adult Saxenda maintenance dose

3 mg once daily

General bodybuilding approval?

No

Athletic-performance approval?

No

Bottom Line

Liraglutide is very well studied, and its main dosing protocols are already established by large human trials and FDA labeling.

For adult weight management, the best-supported Saxenda protocol is:

Week 1: 0.6 mg daily
Week 2: 1.2 mg daily
Week 3: 1.8 mg daily
Week 4: 2.4 mg daily
Week 5 onward: 3 mg daily

For type 2 diabetes, the dosing is different:

0.6 mg → 1.2 mg → up to 1.8 mg daily

The two protocols should **not** be mixed together.

For adult Saxenda:

1.8 mg is a titration dose.

2.4 mg is a titration dose.

3 mg is the recommended maintenance dose.

Protocol Logic Check

Quick Logic Summary

Protocol Question	Verdict
Once-daily dosing?	**Yes, strongly supported**
Subcutaneous route?	**Yes, strongly supported**
~13-hour half-life?	**Yes**
Saxenda 0.6 → 3 mg titration?	**Yes, evidence-supported**
3 mg adult Saxenda maintenance?	**Yes**
Diabetes 0.6 → 1.2 → 1.8 mg?	**Yes**
1.8 mg as universal liraglutide maintenance?	**No, depends on indication**
2.4 mg as a "fat-loss threshold"?	**No**

Protocol Question	Verdict
Bedtime dosing better?	**Not shown**
Fasted dosing needed?	**No**
Only taking it when hungry?	**Not supported**
Every-other-day dosing?	**Not supported**
Twice-daily split dosing?	**Not supported**
8–12 week cycle?	**No proven reason**
Receptor reset?	**No proven requirement**
Combine with another GLP-1 drug?	**Not recommended**
Research vial = approved pen?	**No**

What Does Liraglutide Target?

Liraglutide activates the:

GLP-1 receptor

Simple Version

GLP-1 signaling helps tell the body:

- food has arrived
- release insulin when needed
- reduce unnecessary glucagon
- slow digestion
- increase fullness
- reduce the urge to keep eating

Verdict

Well established

How Long Does It Stay in the Body?

Liraglutide has a half-life of about:

13 hours

After a Saxenda injection, the drug reaches its highest blood level at about:

11 hours

Why This Matters

This long exposure is why liraglutide is designed for:

one injection every day

rather than:

- several injections per day
- every-other-day use
- once-weekly use

Verdict

Once-daily dosing is strongly supported.

How Long Do the Effects Last?

Natural GLP-1 lasts only a few minutes.

Liraglutide was designed to resist rapid breakdown and stay active much longer.

One daily injection provides clinically useful GLP-1 signaling across the day.

Verdict

Evidence-supported

Does It Build Up With Daily Use?

Daily dosing creates steady repeated exposure.

The tested clinical schedule is:

one dose every day

rather than:

- large doses separated by long gaps
- irregular dosing
- only taking it when hunger returns

Verdict

Daily use matches the studied pharmacology.

Does Once-Daily Dosing Make Sense?

Yes.

Both major liraglutide uses are built around:

once-daily SC dosing

Verdict

Strongly evidence-supported

Does Twice-Daily Dosing Make Sense?

There is no established benefit to splitting liraglutide into:

- a morning dose
- an evening dose

Verdict

Not supported

Does Every-Other-Day Dosing Make Sense?

Not as a validated protocol.

Liraglutide was designed and studied for:

once-daily use

Skipping a full day creates larger swings in drug exposure than the tested daily schedule.

Verdict

Not supported

Does Bedtime Dosing Work Better?

The label allows liraglutide at:

any time of day

There is no established evidence that bedtime produces better:

- weight loss

- appetite reduction
- blood sugar control

Verdict

No demonstrated advantage

Does It Need to Be Taken Fasted?

No.

Liraglutide can be taken:

with or without food

Verdict

Not required

Pre-Workout or Post-Workout Timing

There is no established benefit to taking liraglutide:

- before exercise
- after exercise

Verdict

No demonstrated reason

Dose Escalation / Titration

For liraglutide, titration is **actually supported by clinical evidence and labeling**.

Saxenda starts at:

0.6 mg

and increases weekly until:

3 mg

The slow increase is mainly used to reduce stomach-related side effects.

Verdict

Evidence-supported

This is different from compounds where titration is only a community habit.

Is 1.8 mg the Standard Maintenance Dose?

For Type 2 Diabetes

It can be.

Diabetes liraglutide can use:

1.2 mg

or:

1.8 mg

depending on blood sugar control.

For Adult Saxenda Weight Management

No.

The recommended adult maintenance dose is:

3 mg daily

The:

1.8 mg dose

is part of the titration schedule.

Verdict

It depends on what liraglutide is being used for.

This is one of the biggest corrections needed on the live page.

Is 2.4 mg a "Fat-Loss Threshold"?

No.

For adult Saxenda:

2.4 mg is the week-4 titration dose.

It is not officially described as:

> "the fat-loss threshold."

Verdict

Remove that wording.

Does It Need an 8–12 Week Cycle?

No.

Clinical studies lasted much longer, including:

- 56 weeks
- up to 160 weeks in some participants

Verdict

No short-cycle requirement

Does the GLP-1 Receptor Need a Reset?

Some effects of GLP-1 drugs can change with long-term treatment.

For example, the effect on stomach emptying may become less pronounced over time.

That does **not** mean the GLP-1 receptor needs a scheduled break.

Long-term weight-management benefit has been shown with continued treatment.

Verdict

No established receptor-reset requirement

Does It Need a Washout?

There is no validated:

- 2-week washout
- 4-week break
- receptor recovery period

for ongoing liraglutide treatment.

Verdict

No demonstrated requirement

Can It Be Taken Only When Hunger Returns?

That does not match the tested clinical protocol.

Liraglutide is intended for:

consistent once-daily use

Verdict

Not supported

Does the Route Match the Research?

Saxenda

SC

Diabetes Liraglutide

SC

Major Human Trials

SC

Verdict

Yes, strongly supported

Stack / Pairing Logic

Liraglutide + Semaglutide

Both activate the GLP-1 receptor.

The Saxenda label says using another GLP-1 receptor agonist at the same time is:

not recommended

Verdict

Too much pathway overlap and not recommended

Liraglutide + Tirzepatide

Tirzepatide also includes GLP-1 receptor activity, along with GIP activity.

Verdict

Major pathway overlap; no validated routine combination

Liraglutide + Retatrutide

Retatrutide also includes GLP-1 receptor activity.

Verdict

Major pathway overlap; no validated routine combination

Liraglutide + Cagrilintide

Cagrilintide works through a different main pathway.

Verdict

Different mechanism, but no established liraglutide combination protocol

---

Liraglutide + Insulin

This combination can be used clinically in selected people with diabetes.

But:

- the injections should remain separate
- they should not be mixed
- low blood sugar risk needs to be considered

Verdict

Clinically used in selected situations with medical management

---

Formulation / Stability / Sterility Logic

Approved liraglutide is already supplied as a:

ready-to-use liquid

It does **not** need to be mixed with bacteriostatic water.

Saxenda

18 mg / 3 mL

which equals:

6 mg/mL

in a prefilled pen.

Verdict

Do not copy research-vial mixing instructions onto approved Saxenda or liraglutide pens.

Important Research Note

Three different liraglutide situations are often mixed together online.

1. Saxenda

Used for:

long-term weight management

Adult Maintenance

3 mg once daily

after titration.

---

2. Victoza / Diabetes Liraglutide

Used for:

type 2 diabetes

Typical adult treatment doses are:

1.2 mg

or:

1.8 mg once daily

---

3. Research / Compounded Liraglutide

A research vial or compounded product should **not** automatically inherit:

- Saxenda storage
- Saxenda concentration
- Saxenda sterility
- pen dosing accuracy
- 30-day in-use stability

from the approved pen.

Research at a Glance

Approved-Label Human Evidence

Very Strong

Liraglutide has detailed FDA labeling for both weight management and diabetes.

Human Clinical Research

Very Strong

Large human studies support effects on:

- body weight
- blood sugar
- cardiovascular outcomes
- long-term treatment

Observational Human Evidence

Extensive

Years of medical use provide a large amount of real-world safety experience.

Animal / Cell Evidence

Supporting

Especially useful for understanding the thyroid C-cell tumor warning.

Mechanistic Evidence

Strong

GLP-1 receptor biology is well understood.

Community / Anecdotal Evidence

Secondary

Community experience can help explain real-world tolerability.

But for liraglutide, strong clinical evidence matters much more.

Overview

Liraglutide is a long-acting GLP-1 receptor agonist.

Depending on the product and dose, it is used medically for:

- long-term weight management
- type 2 diabetes

It is given:

once daily by subcutaneous injection

Its weight-management effects mainly come from:

- reducing appetite
- increasing fullness
- lowering food intake

It also affects:

- insulin
- glucagon
- blood sugar
- stomach emptying

Important

Liraglutide mainly helps people:

eat less

It has not been shown to work by dramatically increasing how many calories the body burns.

Evidence Strength

Overall Evidence Rating

Strong human evidence

Strong evidence for

- GLP-1 receptor activation
- reduced appetite
- weight loss
- long-term weight management
- type 2 diabetes treatment
- once-daily SC dosing
- gradual titration
- cardiovascular benefit in specific diabetes populations
- major safety risks

Limited or Unproven For

- only taking it when hungry
- every-other-day use
- bedtime superiority
- fasting superiority
- split dosing
- bodybuilding
- athletic enhancement
- spot fat loss
- receptor-reset cycles
- stacking with other GLP-1 drugs

Category

GLP-1 Receptor Agonists / Weight Management / Metabolic Health

---

Compound

Liraglutide

Major branded products include:

- Saxenda
- Victoza

Compound Type

Long-acting GLP-1 receptor agonist

Liraglutide is:

- peptide-based
- an incretin mimetic
- a GLP-1 receptor agonist

It is not:

- insulin
- a stimulant
- a GIP agonist
- a glucagon agonist

Primary Research Focus

Best Established

- long-term weight management
- type 2 diabetes
- appetite reduction
- blood sugar control
- cardiovascular-risk reduction in selected diabetes patients

Also Studied

- waist circumference
- blood pressure
- lipid markers
- prediabetes
- long-term obesity treatment

Not Established

- athletic performance
- bodybuilding
- spot fat loss
- injury repair
- only taking it when appetite returns

How It Works / Receptor Mechanism

Liraglutide activates the:

GLP-1 receptor

Simple Version

After food enters the body, natural GLP-1 helps send the message:

> food is coming in, so control blood sugar and reduce the drive to keep eating.

Liraglutide creates a much longer-lasting version of that signal.

Main Effects

Pancreas: helps release insulin when blood sugar is elevated

Glucagon: reduces unnecessary glucagon release

Stomach: slows stomach emptying

Brain: increases fullness and reduces appetite

Common Research Dose

Weight Management

Saxenda

0.6 → 1.2 → 1.8 → 2.4 → 3 mg SC once daily

Type 2 Diabetes

Victoza / Diabetes Liraglutide

0.6 → 1.2 → up to 1.8 mg SC once daily

Research Duration / Cycle Length

Weight Management

Clinical studies commonly lasted:

56 weeks

with some participants followed for:

160 weeks

Evidence Assessment

Liraglutide is designed for:

long-term treatment

There is no established receptor-reset cycle.

How Often It Is Used / Frequency

Clinical

Once daily

There is no established benefit from:

- twice-daily use
- every-other-day use
- 5-on / 2-off schedules
- only taking it when hungry

Possible Benefits

Strong Human Evidence

Weight Loss

Large randomized human studies show meaningful average weight loss with:

liraglutide 3 mg

plus lifestyle changes.

Appetite Reduction

Reduced appetite and increased fullness are central effects of GLP-1 treatment.

Better Blood Sugar Control

Strongly established in type 2 diabetes.

Cardiovascular Outcomes

Liraglutide has cardiovascular benefit in adults with:

- type 2 diabetes
- established cardiovascular disease

Some Additional Human Evidence

Waist Circumference

Saxenda trials found greater reductions in waist circumference than placebo.

Blood Pressure

Obesity studies found modest average improvements in blood pressure.

Lipids and Other Metabolic Measures

Some other cardiometabolic measurements also improved.

These should be presented as:

secondary outcomes

rather than the main reason liraglutide works.

What It Does Not Help With, Based on Current Evidence

Do not present liraglutide as proven to:

- specifically target belly fat
- build muscle
- improve athletic performance
- increase energy
- repair injuries
- directly speed up metabolism

- produce the same weight loss at every dose
- permanently prevent weight regain after stopping

Commonly Paired With

Metformin

Frequently used with liraglutide in type 2 diabetes.

Classification: Clinically established combination.

Insulin

Can be used clinically in selected patients.

Classification: Clinically established in some situations, but low blood sugar risk and dose management matter.

Other GLP-1 Agonists

Examples include semaglutide.

Classification: Not recommended because the pathways overlap.

Tirzepatide / Retatrutide

Both include GLP-1 receptor activity.

Classification: Major pathway overlap; no established routine combination.

Cagrilintide

Uses a different receptor system.

Classification: Different mechanism, but no validated liraglutide combination protocol.

Dosage Information

Saxenda

Product: Saxenda
Compound: Liraglutide
Strength: 18 mg / 3 mL
Concentration: 6 mg/mL
Form: Clear, colorless liquid
Route: Subcutaneous
Device: Single-patient-use prefilled pen

Adult Dose Escalation

Week 1: 0.6 mg daily
Week 2: 1.2 mg daily
Week 3: 1.8 mg daily
Week 4: 2.4 mg daily
Week 5 onward: 3 mg daily

Why Increase Slowly?

Mainly to reduce stomach-related side effects.

Timing

Inject:

once daily at any time

with or without food.

Injection Sites

- abdomen
- thigh
- upper arm

Rotate injection sites.

Missed Dose

If one dose is missed:

take the next dose at the normal scheduled time.

Do **not** take an extra dose.

If more than:

3 days

have passed since the last Saxenda dose:

restart at:

0.6 mg daily

and repeat the dose-escalation schedule to reduce GI side effects.

Victoza / Diabetes Liraglutide Dosing

Adults

Start: 0.6 mg SC once daily for one week

Then:

1.2 mg daily

If more blood sugar control is needed:

1.8 mg daily

after at least one week at 1.2 mg.

Important

Do not copy:

Saxenda's 3 mg maintenance dose

into the diabetes protocol.

Likewise, do not call:

1.8 mg

the standard adult Saxenda maintenance dose.

Community Research Protocol

Because liraglutide already has a detailed approved protocol, community use usually involves:

changing how quickly the dose is increased

rather than creating a completely different dosing system.

Common community practices include:

- staying at one dose longer if nausea is strong
- increasing more slowly
- not increasing while appetite suppression remains strong

Evidence Check

Slower escalation for tolerability: Makes sense and fits the reason titration exists

Delaying an increase by about one extra week: Supported by the Saxenda label if a higher dose is not tolerated

Staying below 3 mg indefinitely for adult Saxenda: Community / off-label practice

Only taking it when hungry: Unsupported

Every-other-day dosing: Unsupported

Community Protocol Comparison

Protocol	Dose	Frequency	Purpose	Evidence
Saxenda week 1	0.6 mg	Daily SC	Titration	FDA label
Saxenda week 2	1.2 mg	Daily SC	Titration	FDA label
Saxenda week 3	1.8 mg	Daily SC	Titration	FDA label
Saxenda week 4	2.4 mg	Daily SC	Titration	FDA label
Saxenda maintenance	3 mg	Daily SC	Weight management	FDA label
Diabetes liraglutide	1.2–1.8 mg	Daily SC	Type 2 diabetes	FDA label

Protocol	Dose	Frequency	Purpose	Evidence
Slower community titration	Variable	Daily SC	Tolerability	Community / reasonable
PRN or EOD use	Variable	Irregular	Appetite	Unsupported

Strongest Agreement

Once-daily SC use with gradual titration

Main Source of Confusion

People often mix together:

- Saxenda doses
- Victoza doses
- titration doses
- maintenance doses

They should stay separate.

Expected Results Timeline, Expanded

Human Clinical Research

Early Weeks

Liraglutide starts working right away.

But Saxenda spends the first month gradually increasing toward the maintenance dose.

Appetite changes may happen before full-dose treatment is reached.

Week 16

Adult response should be checked.

If body weight has not dropped by at least:

4%

from the starting weight, the current label recommends stopping Saxenda.

Week 56

Large obesity trials show meaningful average weight loss by about:

1 year

Up to Week 160

Some participants remained in longer-term research for about:

160 weeks

Community-Reported

Common descriptions include:

Days to weeks: less appetite or feeling full sooner

Several weeks: changes on the scale

Several months: more noticeable changes in body weight and waist size

Evidence Warning

Community timelines should remain secondary because liraglutide already has strong human outcome data.

Reported Side Effects

Common Effects

- nausea
- diarrhea
- constipation
- vomiting
- injection-site reactions
- headache
- indigestion
- fatigue
- dizziness
- abdominal pain
- increased lipase

Pancreatitis

Acute pancreatitis has occurred with liraglutide.

Severe or persistent abdominal pain needs medical assessment.

Gallbladder Disease

Known risks include:

- gallstones
- gallbladder inflammation

Hypoglycemia

Most relevant when liraglutide is combined with:

- insulin
- sulfonylureas

rather than liraglutide alone.

Heart-Rate Increase

Saxenda can increase resting heart rate.

Kidney Injury From Dehydration

Severe stomach-related side effects can cause dehydration.

That dehydration can contribute to acute kidney injury.

Severe GI Effects

Saxenda is:

not recommended in severe gastroparesis

Serious Allergic Reactions

Serious allergic reactions including:

- anaphylaxis
- angioedema

have been reported.

Aspiration During Anesthesia

Because liraglutide can slow stomach emptying, food may remain in the stomach longer than expected.

Rare aspiration cases have been reported during anesthesia or deep sedation.

Contraindications & Cautions

Formal Contraindications

Personal or Family History of MTC

Contraindicated

MEN 2

Contraindicated

Serious Hypersensitivity to Liraglutide

Contraindicated

Important Cautions

Pancreatitis

Pancreatitis is an important warning.

But:

a history of pancreatitis is not listed as a formal current Saxenda contraindication.

Severe Gastroparesis

Saxenda is:

not recommended

rather than formally contraindicated.

Kidney Disease / Dehydration Risk

Kidney impairment itself is not a blanket contraindication.

The main concern is kidney injury caused or worsened by dehydration from severe GI side effects.

Diabetes Medications

Insulin and sulfonylureas can increase the risk of low blood sugar.

Pregnancy

Pregnancy is not currently listed as a formal Saxenda contraindication.

But that does **not** mean Saxenda should be continued during pregnancy.

Weight loss provides no benefit during pregnancy, the label warns of possible fetal harm, and Saxenda should be:

stopped when pregnancy is recognized.

Planned Surgery / Deep Sedation

Tell the procedural team about liraglutide because it can slow stomach emptying.

Suicidal Ideation

This is:

no longer a current Saxenda warning

FDA approved removal of the warning in February 2026.

Regulatory Status

United States

FDA approved

Saxenda

Approved for long-term weight management in eligible adults and adolescents.

Victoza / Diabetes Liraglutide

Approved for type 2 diabetes, with cardiovascular-risk reduction in certain adults with established cardiovascular disease.

Approved Route

Subcutaneous

Not Approved For

- bodybuilding
- athletic performance
- spot fat loss
- only taking it when hungry
- routine combination with another GLP-1 agonist

What Studies Have Found

SCALE Obesity and Prediabetes

A large randomized trial compared:

liraglutide 3 mg once daily

with placebo alongside lifestyle changes.

At 56 weeks:

Liraglutide: about 8.4 kg average weight loss

Placebo: about 2.8 kg average weight loss

What This Supports

- 3 mg adult weight-management dosing
- once-daily use
- long-term treatment

- meaningful average weight loss

What It Does Not Support

- only taking it when hungry
- random microdosing
- every-other-day dosing
- assuming 0.6 mg gives the same long-term effect

SCALE Diabetes

Adults with type 2 diabetes were assigned to:

- liraglutide 3 mg
- liraglutide 1.8 mg
- placebo

Average weight loss was about:

6.4 kg with 3 mg

5.0 kg with 1.8 mg

2.2 kg with placebo

What This Supports

Dose can affect weight-loss results.

What It Does Not Support

Treating:

1.8 mg and 3 mg

as if they are interchangeable protocols.

Long-Term Weight Management Research

Saxenda trials commonly lasted:

56 weeks

with some participants followed for:

160 weeks

What This Supports

Liraglutide is designed as a:

long-term treatment

rather than a short peptide cycle.

Cardiovascular Research

Liraglutide has been studied in adults with:

- type 2 diabetes
- established cardiovascular disease

The diabetes indication includes reducing major cardiovascular events in this specific population.

What This Supports

Cardiovascular benefit in the:

type 2 diabetes population that was actually studied

What It Does Not Support

Automatically giving Saxenda a general cardiovascular-disease indication for everyone using it for weight management.

Formulation / Stability / Sterility

Saxenda

18 mg in 3 mL
6 mg/mL
Clear, colorless liquid
Single-patient-use prefilled pen
Subcutaneous use only

Before First Use

Store refrigerated at:

2°C to 8°C (36°F to 46°F)

Do not freeze.

After First Use

A Saxenda pen can be stored for:

30 days

either:

- at **15°C to 30°C (59°F to 86°F)**
- or refrigerated at **2°C to 8°C (36°F to 46°F)**

Keep the cap on.

Protect from:

- excessive heat
- sunlight

Remove the needle after every injection.

Discard the pen after:

30 days

even if medication remains.

Research / Compounded Liraglutide

Do **not** automatically apply Saxenda's:

- 6 mg/mL concentration
- 30-day storage rule
- sterility
- pen accuracy

to:

- research vials
- compounded multidose vials
- lyophilized liraglutide
- products mixed with bacteriostatic water

Simple Rule

Use storage and handling instructions for the exact product.

Research Sources

Use **clean canonical URLs in production**.

1. DailyMed: Saxenda

Evidence type: Current FDA-approved labeling

Supports:

- weight-management indication
- 0.6 → 3 mg escalation
- adult 3 mg maintenance
- once-daily SC route
- meal-independent timing
- contraindications
- current warnings
- 16-week adult response check
- formulation
- storage
- 30-day in-use period
- current 2026 warning structure

`https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=3946d389-0926-4f77-a708-0acb8153b14`

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2. DailyMed: Victoza

Evidence type: FDA-approved diabetes labeling

Supports:

- 0.6 mg starting dose
- 1.2 mg treatment dose
- optional increase to 1.8 mg
- once-daily SC use
- any-time-of-day dosing
- meal-independent dosing
- type 2 diabetes indication
- cardiovascular-risk reduction in adults with type 2 diabetes and established cardiovascular disease
- approximately 13-hour half-life

`https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=5a9ef4ea-c76a-4d34-a604-27c5b505f5a`

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3. Pi-Sunyer et al.: SCALE Obesity and Prediabetes

Evidence type: Randomized human clinical trial

Supports:

- liraglutide 3 mg
- 56-week weight-loss outcomes
- major obesity efficacy data

https://pubmed.ncbi.nlm.nih.gov/26132939/

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4. Davies et al.: SCALE Diabetes

Evidence type: Randomized human clinical trial

Supports:

- comparison of 3 mg and 1.8 mg
- dose-related weight-loss differences in people with type 2 diabetes

https://pubmed.ncbi.nlm.nih.gov/26284720/

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5. Wadden et al.: SCALE Maintenance

Evidence type: Randomized human clinical research

Supports:

- longer-term weight maintenance
- continued liraglutide treatment after initial diet-induced weight loss

https://pubmed.ncbi.nlm.nih.gov/23812094/

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6. FDA 2026 Safety Update

Evidence type: FDA regulatory action

Supports:

- removal of the suicidal-behavior and ideation warning from Saxenda labeling

https://www.accessdata.fda.gov/drugsatfda_docs/appletter/2026/206321Orig1s025ltr.pdf

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Final Takeaway

Liraglutide is one of the strongest-evidence compounds in this library because both its mechanism and its dosing have been studied extensively in humans.

For adult weight management, the validated Saxenda protocol is:

Week 1: 0.6 mg daily
Week 2: 1.2 mg daily
Week 3: 1.8 mg daily
Week 4: 2.4 mg daily
Week 5 onward: 3 mg daily

For type 2 diabetes, the dosing is different:

0.6 mg → 1.2 mg → up to 1.8 mg daily

For timing:

once daily, any time of day, with or without food

is the best-supported approach.

For cycling:

there is no established receptor-reset protocol.

For results:

Clinical: appetite changes may begin during titration, adult response is checked at **16 weeks**, major obesity results are commonly measured at **56 weeks**, and some longer-term research extends to **160 weeks**.

And the three most important corrections to the old live page remain:

> **For adult Saxenda, 1.8 mg is not the standard maintenance dose. It is part of titration.**
>
> **2.4 mg should not be called a "fat-loss threshold." It is the week-4 titration dose.**
>
> **Suicidal behavior or suicidal ideation should no longer appear as a current Saxenda warning or risk flag.**

MOTS-c

Mitochondrial-Derived Peptide • Metabolic Signaling • Insulin-Sensitivity Research

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At a Glance

What is MOTS-c?

MOTS-c is a naturally occurring peptide made from:

16 amino acids

What makes it unusual is where its genetic instructions come from.

Most peptides are encoded by DNA in the cell nucleus.

MOTS-c comes from:

mitochondrial DNA

specifically from a small region inside the mitochondrial 12S rRNA gene. FDA's 2026 scientific review also describes MOTS-c as a naturally occurring mitochondrial peptide made of 16 amino acids. (fda.gov)

MOTS-c appears to be involved in how cells respond to:

- energy demand
- metabolic stress
- glucose availability
- exercise
- changes in mitochondrial function

Most of what we currently know about its possible treatment effects comes from:

cell and animal research

rather than completed human injection trials.

How well studied is it?

Interesting science, but injected-human research is only beginning

This distinction is extremely important.

MOTS-c has:

- strong laboratory research
- many animal studies
- human studies measuring the MOTS-c our bodies naturally produce

- human exercise research
- an active Phase 2a human treatment trial

But we do **not yet have published results from controlled trials showing that injected native MOTS-c improves:**

- insulin sensitivity
- body weight
- body composition
- energy
- endurance
- exercise performance

in humans.

FDA's 2026 review found no published studies in which MOTS-c had been administered to humans that could establish its safety or effectiveness. FDA therefore described human safety and effectiveness as unknown. (fda.gov)

The important update is that this is now beginning to change.

A Phase 2a study is currently testing MOTS-c in adults with prediabetes and overweight or obesity. (clinicaltrials.gov)

Evidence level

FDA-approved human evidence: None
Completed injected-human efficacy evidence: None yet
Active human clinical research: Yes, Phase 2a
Human natural-MOTS-c / observational research: Moderate
Animal / cell research: Strong
Mechanistic research: Strong but incomplete
Community injection protocols: Common but unproven

What may it help with?

Currently Being Tested in Humans

Insulin Sensitivity

The current Phase 2a MOTS-MET trial is studying adults with:

- prediabetes
- overweight or obesity
- insulin resistance

The main question is whether approximately:

12 weeks of MOTS-c treatment

can improve insulin sensitivity compared with placebo. (clinicaltrials.gov)

Important

The trial has **not reported results yet**.

So Navaro should say:

> **MOTS-c is being studied for insulin sensitivity in humans.**

Not:

> **MOTS-c has been proven to improve human insulin sensitivity.**

Human Exercise Research

Human research has shown that the MOTS-c naturally produced by our bodies changes with exercise.

A 2021 study found that exercise increased endogenous MOTS-c in human skeletal muscle and circulation. (pubmed.ncbi.nlm.nih.gov)

What This Supports

MOTS-c appears to be part of the body's normal response to:

exercise and increased energy demand.

What It Does Not Prove

It does **not** prove that injecting MOTS-c:

- recreates exercise
- improves endurance
- causes fat loss
- improves blood sugar
- increases mitochondrial function

in humans.

Strong Animal / Cell Evidence

Animal and laboratory studies suggest MOTS-c may affect:

- insulin sensitivity
- glucose metabolism
- metabolic flexibility

- obesity-related metabolism
- physical capacity
- skeletal-muscle function
- age-related metabolic changes

These findings are why researchers are now testing MOTS-c in humans.

But for Navaro they should remain clearly labeled:

Animal / cell evidence

until human treatment results are available.

What has it NOT been proven to help with?

Injected MOTS-c has **not yet been proven in humans** to improve:

- fat loss
- obesity
- insulin resistance
- diabetes
- endurance
- athletic performance
- mitochondrial function
- fatigue
- GLP-1-related fatigue
- muscle growth
- recovery
- longevity
- anti-aging
- metabolic flexibility
- body composition

That does **not** mean these effects are impossible.

It means we do not yet have controlled human injection results proving them.

Dose at a Glance

Human Clinical Research

Current Phase 2a Trial

The active trial uses:

Route: Subcutaneous
Treatment period: 12 weeks

Safety follow-up: Through approximately week 16
Estimated enrollment: About 120 adults

The study is enrolling adults with prediabetes and overweight or obesity. (clinicaltrials.gov)

Important

Do **not** publish an exact clinical milligram dose unless it is clearly disclosed by the current trial record.

This is an investigational study, not an approved treatment protocol.

Research-Community Dosing

Community protocols are very different from the clinical trial.

PeptideFox currently describes approximately:

5–10 mg per injection

often:

2–3 times per week

using SC or IM injections.

A commonly discussed starting amount is:

5 mg

Evidence level

Community / practitioner protocol

These doses have **not** been established by completed human dose-finding trials of native MOTS-c.

FDA Compounding Nomination Doses

Another source of confusion is FDA's compounding review.

The products submitted in the nomination included:

5 mg

and:

10 mg

for SC injection. FDA explicitly describes these as proposed compounded products, not approved or validated doses. (fda.gov)

Important

A dose being **nominated to FDA** does not mean:

> FDA approved the dose.

In fact, FDA's 2026 scientific review concluded that the evidence weighed **against** placing MOTS-c free base or MOTS-c acetate on the 503A Bulks List. (fda.gov)

Frequency at a Glance

Human Clinical Research

The current study uses repeated SC treatment across:

12 weeks

The trial will provide important information about repeated human exposure. (clinicaltrials.gov)

Community

Commonly:

2–3 times per week

Other community schedules include:

- once weekly
- every few days
- higher-frequency short cycles

Best-supported frequency?

We do not know yet.

No completed human trial has established the best MOTS-c dosing frequency.

Timing at a Glance

Clinical

There is no established human evidence showing injected MOTS-c needs to be taken:

- before exercise
- after exercise
- in the morning
- at night
- fasted
- with food

The current live page should therefore **not** list:

Pre-Workout

as though it is a required dosing window.

Community

Community protocols often use MOTS-c:

- in the morning
- before cardio
- before endurance training

PeptideFox describes pre-exercise timing in its community material.

Why Did This Idea Develop?

The idea is understandable because the body's own MOTS-c increases during exercise. (pubmed.ncbi.nlm.nih.gov)

But that does **not** prove an injection works better when taken before exercise.

Evidence verdict

Pre-workout: Makes sense in theory, but unproven

Morning: Community preference

Fasted: Unproven

Required: No

Duration / Cycle at a Glance

Human Clinical Research

The current Phase 2a study uses:

12 weeks of treatment

followed by additional safety follow-up. (clinicaltrials.gov)

Community

Community protocols often use approximately:

4–6 weeks on

followed by:

2–4 weeks off

Does MOTS-c Need to Be Cycled?

We do not know.

There is no human evidence showing that MOTS-c requires a break to:

- reset a receptor
- restore sensitivity
- prevent tolerance

A specific MOTS-c receptor has not even been clearly identified. FDA's 2026 review specifically states that its molecular target or targets remain unknown. (fda.gov)

Expected Results Timeline

Human Clinical Research

There Is No Injected-Human Results Timeline Yet

This is one of the most important things to get right.

The first Phase 2a study is still underway.

There are no published results showing:

- insulin sensitivity improves by week 2
- fat loss starts at week 4
- endurance improves at week 6

- energy increases within days

from controlled MOTS-c injections in humans. (clinicaltrials.gov)

What the Current Trial Is Testing

The current study is looking at metabolic outcomes after approximately:

12 weeks of treatment

with safety monitoring continuing afterward. (clinicaltrials.gov)

The main focus is:

insulin sensitivity

Other metabolic measurements include blood-sugar and cardiometabolic markers.

Clinical Takeaway

The first real injected-human results timeline is still being established.

Community-Reported Timeline

Community material describes changes over roughly:

several days to several weeks

with energy or exercise-related changes often discussed during:

4–6 week community cycles

Important

This is:

community-reported

not:

clinically proven

Best Production Wording

> **Community-reported:** some users describe changes in energy or exercise tolerance over the first several weeks. These reports have not yet been confirmed in controlled human MOTS-c injection trials.

Simple Timeline for the Top Card

Clinical: No injected-human results timeline exists yet. The current Phase 2a trial is testing metabolic outcomes over approximately **12 weeks**.

Community-reported: subjective energy or exercise-related changes are described over **several weeks**, but these reports remain anecdotal.

Safety at a Glance

Human Safety Profile

Not established yet

MOTS-c does not currently have a completed human injection study that can give us a reliable table of:

- common side effects
- uncommon side effects
- serious adverse effects
- exact rates

FDA's 2026 review specifically said there was not enough human administration information to determine the safety of MOTS-c and highlighted uncertainty around immunogenicity and product quality. (fda.gov)

FDA Safety Concerns

FDA highlighted possible concerns involving:

- immune reactions to the peptide
- peptide clumping or aggregation
- impurities
- differences between products sold as MOTS-c
- manufacturing consistency
- inadequate characterization of the active ingredient

These concerns are particularly important for injected peptide products. (fda.gov)

What Should NOT Be Called Established MOTS-c Side Effects?

The current live page lists things such as:

- hypoglycemia
- fatigue
- flushing
- GI symptoms
- headache
- flu-like symptoms
- "AMPK overstimulation"

as though their frequency in humans is known.

It is not.

These should instead be classified as:

Community-reported or theoretical

until proper human safety results are available.

Hypoglycemia

The current live page describes low blood sugar as the major practical side effect and says MOTS-c should always be used before exercise.

There is not enough human evidence to support that statement.

MOTS-c is being studied because it may affect insulin sensitivity and glucose metabolism.

So changes in glucose control are biologically worth watching.

But:

> **Low blood sugar has not been established as the main adverse effect of injected MOTS-c in humans.**

Verdict

Possible in theory, but unconfirmed in humans

Injection-Site Reactions

Community users report things such as:

- redness
- soreness
- irritation

But no completed human MOTS-c trial currently tells us how often these happen.

Verdict

Community-reported; true rate unknown

Major Contraindications

Formal FDA Contraindications

None established

This is because MOTS-c is:

not an FDA-approved medication

There is no approved product label containing an official contraindication list.

Navaro should therefore avoid turning theoretical concerns into formal contraindications.

Important Cautions

Pregnancy

Human fetal-safety information does not exist.

Classification: Safety unknown / avoid experimental exposure.

Breastfeeding

No adequate human safety data.

Classification: Unknown.

Children and Adolescents

No established injected-MOTS-c safety or effectiveness.

Classification: Unstudied.

Diabetes Medications

Because MOTS-c is being studied for insulin sensitivity, combining it with glucose-lowering medications could potentially change glucose control.

This may include:

- insulin
- sulfonylureas
- other glucose-lowering drugs

There is not yet a human interaction study telling us the size of that risk.

Classification: Important theoretical caution, not a proven contraindication.

Metformin

MOTS-c and metformin research both involve some of the same metabolic signaling pathways, including AMPK-related biology.

That does **not** prove the combination is dangerous.

Classification: Pathway overlap; actual interaction unknown.

Active Cancer

There are not enough human data to establish safety in people with active malignancy.

Classification: Unknown risk, not a formal contraindication.

Regulatory Status at a Glance

FDA approved?

No

Neither MOTS-c free base nor MOTS-c acetate is part of an FDA-approved drug. (fda.gov)

Human clinical development?

Yes

A Phase 2a trial is currently underway in adults with prediabetes and overweight or obesity. (clinicaltrials.gov)

U.S. Compounding Status

FDA evaluated:

- MOTS-c free base
- MOTS-c acetate

for possible inclusion on the 503A Bulks List.

FDA staff concluded that the available evidence weighed **against inclusion**, citing lack of established human safety/effectiveness and concerns about product characterization and immunogenicity. (fda.gov)

Important

An advisory committee review is **not the same thing as final FDA approval of a compounded drug**.

Best Wording

> **Not FDA approved. MOTS-c free base and acetate underwent FDA compounding review in 2026. FDA staff concluded that the available evidence weighed against inclusion on the 503A Bulks List.**

WADA Status

Prohibited

MOTS-c appears on the **2026 WADA Prohibited List** under AMPK activators. (wada-ama.org)

Athletes who are subject to WADA rules should treat this as an important anti-doping issue.

Bottom Line

MOTS-c has interesting mitochondrial science, but human injection research is still at a very early stage.

What we know reasonably well:

- MOTS-c is a real 16-amino-acid peptide
- its genetic instructions come from mitochondrial DNA

- it participates in metabolic signaling
- the MOTS-c our bodies naturally produce changes with exercise
- animal studies show potentially important metabolic effects
- human treatment research is now underway. (fda.gov)

What we **do not yet know from completed human injection trials**:

- the best dose
- the best frequency
- the best time of day
- human injection half-life
- whether it improves insulin sensitivity
- whether it improves endurance
- whether it causes fat loss
- whether it improves fatigue
- actual human side-effect rates
- whether cycling is useful

Community protocols commonly use:

5–10 mg

several times per week, often around exercise.

Those should be displayed as:

community protocols

not:

clinical dosing

Protocol Logic Check

Quick Logic Summary

Protocol Question	Verdict
Is MOTS-c a real mitochondrial-derived peptide?	**Yes**
Is it 16 amino acids long?	**Yes**
Does its sequence come from mitochondrial DNA?	**Yes**
Is AMPK involved?	**Strong animal/cell evidence**
Is AMPK the MOTS-c receptor?	**No**
Does natural MOTS-c respond to human exercise?	**Yes**
Has injected MOTS-c proven human efficacy?	**Not yet**
Is a human Phase 2a trial underway?	**Yes**
Is SC administration being studied?	**Yes**
5–10 mg several times weekly?	**Community protocol**

Protocol Question	Verdict
200 mcg → 1 mg titration?	**No human trial support**
Pre-workout required?	**No**
Fasted dosing required?	**No evidence**
Morning required?	**No evidence**
4–6 week cycle?	**Community convention**
2–4 week reset?	**No proven reason**
Human injection half-life known?	**No**
Human side-effect rates known?	**No**
FDA approved?	**No**
WADA prohibited?	**Yes**

What Does MOTS-c Actually Target?

The current live page says:

Target: AMPK

That is too simple.

MOTS-c does not have one clearly identified receptor like:

- liraglutide → GLP-1 receptor
- Tesamorelin → GHRH receptor

Instead, laboratory research suggests MOTS-c affects several parts of cellular metabolism.

These include:

- folate metabolism
- purine metabolism
- AICAR
- AMPK-related signaling
- stress-response genes

Under metabolic stress, MOTS-c can also move into the nucleus and affect gene activity.

FDA's 2026 review specifically says the molecular target or targets through which MOTS-c works remain unknown. (fda.gov)

Simple Version

MOTS-c appears to help cells respond to metabolic stress and changing energy demands.

AMPK seems to be part of that response.

But:

> **AMPK is not an identified "MOTS-c receptor."**

Verdict

We understand parts of the pathway, but the exact target is still unknown.

Human Half-Life

Unknown

There is no established published half-life for native injected MOTS-c in humans.

So the page should not claim:

- it lasts a certain number of hours
- it needs to be redosed based on that number
- one dose provides a certain number of days of signaling

Verdict

We do not know yet.

How Long Do the Effects Last?

Also unknown in injected humans.

It is possible that a short period of peptide exposure could trigger longer-lasting metabolic changes.

But this has not been measured well enough in humans to give a reliable duration.

Verdict

Possible, but not measured in humans yet.

Does It Build Up With Repeated Use?

We do not yet have published human injection data showing:

- how much MOTS-c builds up
- whether it reaches steady levels
- how quickly it washes out

- how long it stays in tissues

Verdict

Unknown

Does Daily Dosing Make Sense?

Repeated SC treatment is now being tested in the Phase 2a trial.

That makes the clinical trial much more relevant than older community schedules. (clinicaltrials.gov)

But:

the trial has no results yet.

Verdict

Currently being tested in humans

Does 2–3 Times Weekly Make Sense?

This is common in peptide communities.

But no completed human study shows that 2–3 times per week is the best schedule.

Verdict

Community protocol

Does Once-Weekly Dosing Make Sense?

Some community protocols use once-weekly injections.

We do not have human research showing that one injection per week provides enough exposure for the proposed metabolic effects.

Verdict

Community protocol / unproven

Does Pre-Workout Timing Make Sense?

There is a reason people came up with the idea.

Natural MOTS-c increases during human exercise. (pubmed.ncbi.nlm.nih.gov)

So pairing an experimental MOTS-c injection with exercise may sound logical.

But no human trial has shown that injections work better:

- 30 minutes before exercise
- 60 minutes before exercise
- 90 minutes before exercise

than at another time.

Verdict

Makes sense in theory, but unproven

Does It Have to Be Used With Exercise?

No evidence says it does.

The live page's:

> "always dose pre-workout, never fasted without planned exercise"

should be removed.

Verdict

No demonstrated requirement

Does Fasted Dosing Make Sense?

Fasting activates many energy-sensing pathways.

That gives the idea some biological logic.

But no human injection study has shown that MOTS-c works better while fasted.

Verdict

Possible in theory, but unproven

Morning or Evening?

We do not know.

Morning use is mostly a community preference, often because users combine MOTS-c with exercise.

Verdict

Community preference

4–6 Weeks On / 2–4 Weeks Off

This is commonly discussed in community protocols.

But no human research shows that MOTS-c suddenly stops working after 4–6 weeks.

There is also no evidence showing that taking several weeks off restores sensitivity.

Verdict

Community convention

Does It Need a Receptor Reset?

Probably not a useful way to think about MOTS-c.

We do not even have a clearly identified MOTS-c receptor.

So claims that:

> "the MOTS-c receptor needs to reset"

are especially weak.

Verdict

No demonstrated receptor-reset mechanism

Does It Need a Washout?

There is no established:

- 2-week break
- 4-week break

- reset period

for MOTS-c.

Verdict

No demonstrated requirement

Dose Escalation / Titration

The live page includes several very different protocols, including:

200 mcg → 1,000 mcg daily

and other schedules using:

- 3 mg
- 5 mg
- 10 mg

at different intervals.

There is no completed human dose-ranging study showing that these ladders are correct.

Verdict

These are different community protocols, not proven clinical titration schedules.

They should not be grouped under:

> **Standard Protocol**

Does the Route Match the Research?

Current Human Trial

SC

FDA Compounding Nomination

SC

Community

SC or IM

Verdict

SC currently has the strongest human-research precedent.

But that means:

SC is being studied

not:

SC efficacy has been proven.

Stack / Pairing Logic

MOTS-c + NAD+

They affect different parts of cellular energy metabolism.

Verdict: Could be complementary, but no controlled human combination trial exists.

MOTS-c + SS-31 / Elamipretide

SS-31 works mainly through mitochondrial membrane and cardiolipin biology.

MOTS-c works through metabolic signaling.

Verdict: Different mechanisms, but the combination is untested.

MOTS-c + L-Carnitine

L-Carnitine helps fatty acids get into mitochondria.

MOTS-c appears to influence metabolic signaling.

Verdict: Different mechanisms and theoretically complementary, but clinically unproven.

MOTS-c + GLP-1 Drugs

The mechanisms are different.

MOTS-c is sometimes promoted as a way to reduce fatigue during GLP-1 treatment.

There is **no controlled human evidence** showing that it prevents or treats GLP-1-related fatigue.

Verdict: Different mechanisms, but claimed benefit is unproven.

MOTS-c + AICAR

Both involve AMPK-related metabolic signaling.

Verdict: Greater pathway overlap; combination untested.

Formulation / Stability / Sterility Logic

This area deserves extra caution.

FDA identified concerns involving:

- impurities
- peptide aggregation
- immune reactions
- product identity
- bacterial endotoxins
- differences between free-base and acetate products. (fda.gov)

Simple Version

Not every vial labeled:

"MOTS-c"

can automatically be assumed to contain the exact same pharmaceutical substance.

And there is no universal rule saying:

> Mix MOTS-c with bacteriostatic water and use it for 28 days.

Verdict

Storage and handling need evidence for the exact formulation.

Important Research Note

Four different types of MOTS-c evidence are often mixed together online.

1. Natural Human MOTS-c

Humans naturally produce MOTS-c.

Exercise studies show that these natural levels change with physical activity. (pubmed.ncbi.nlm.nih.gov)

That is:

real human evidence

but it is **not evidence from MOTS-c injections**.

---

2. Animal and Cell Research

This is where many of the exciting claims come from.

Animal and laboratory studies have investigated:

- insulin sensitivity
- glucose metabolism
- obesity
- endurance
- physical function
- aging-related changes

These studies are important.

But:

mouse results are not human treatment results.

---

3. Current Human Phase 2a Research

MOTS-c is now being tested as an actual treatment in humans.

The current study is enrolling approximately:

120 adults

with:

- prediabetes
- overweight or obesity

and is testing MOTS-c against placebo for approximately:

12 weeks

with continued safety follow-up. (clinicaltrials.gov)

Status

Ongoing / no published efficacy results yet

---

4. Community Injection Protocols

Community protocols use things such as:

- 5 mg
- 10 mg
- multiple injections per week
- pre-workout timing
- cycling
- stacking

These are worth documenting.

But they should be labeled:

Community protocols

not:

Human clinical dosing

Research at a Glance

FDA-Approved Human Evidence

None

MOTS-c is not FDA approved. (fda.gov)

Human Treatment Evidence

Beginning

A Phase 2a trial is underway.

No treatment results are available yet. (clinicaltrials.gov)

Human Natural / Observational Evidence

Moderate

Human research supports:

- natural MOTS-c production
- exercise-related changes
- relationships with metabolic and physical characteristics

Animal / Cell Evidence

Strong

This is where most treatment claims currently come from.

Mechanistic Evidence

Moderate to Strong

We know several pathways MOTS-c appears to affect.

We do **not** yet know its exact receptor or molecular target. FDA specifically highlighted this uncertainty in 2026. (fda.gov)

Community / Anecdotal Evidence

Common but Low Quality

Useful for showing how people currently use MOTS-c.

Not enough to establish safety or effectiveness.

Overview

MOTS-c is one of the more unusual compounds in the library.

It is a:

mitochondrial-derived peptide

whose genetic instructions come from mitochondrial DNA. (fda.gov)

Research suggests that MOTS-c helps cells respond to changes in:

- energy demand

- glucose
- metabolic stress

Animal and cell studies suggest effects involving:

- insulin sensitivity
- glucose metabolism
- AMPK-related signaling
- metabolic adaptation

Human studies also show that the MOTS-c our bodies naturally make changes with exercise. (pubmed.ncbi.nlm.nih.gov)

The big unanswered question is:

> **Does injecting MOTS-c create useful metabolic effects in people?**

The current Phase 2a trial is finally beginning to test that directly. (clinicaltrials.gov)

Evidence Strength

Overall Evidence Rating

Strong preclinical science, very early injected-human evidence

Strong Evidence For

- MOTS-c exists naturally
- 16-amino-acid structure
- mitochondrial DNA origin
- involvement in metabolic signaling
- natural MOTS-c changes with exercise
- metabolic effects in animal and cell models

Currently Being Tested in Humans

- insulin sensitivity
- glucose-related markers
- body weight
- waist circumference
- safety of repeated SC treatment. (clinicaltrials.gov)

Not Established in Injected Humans

- fat loss
- endurance

- energy improvement
- GLP-1 fatigue relief
- anti-aging
- longevity
- best dose
- best frequency
- best timing
- injection half-life
- need for cycling
- true adverse-effect rates

Category

Mitochondrial-Derived Peptides / Metabolic Signaling

Compound

MOTS-c

Full name:

Mitochondrial Open Reading Frame of the 12S rRNA Type-C

Compound Type

16-amino-acid mitochondrial-derived peptide

MOTS-c is:

- peptide-based
- encoded by mitochondrial DNA
- involved in metabolic signaling

It is not:

- a stimulant
- a GLP-1 agonist
- insulin
- growth hormone
- a conventional drug with one clearly identified receptor

Primary Research Focus

Current Human Trial Focus

- insulin sensitivity
- prediabetes
- overweight / obesity
- metabolic health

Animal / Cell Research Focus

- glucose metabolism
- metabolic flexibility
- skeletal-muscle metabolism
- obesity
- physical capacity
- aging-related metabolic changes

Not Established

- bodybuilding
- athletic enhancement
- fat-loss treatment
- fatigue treatment
- longevity treatment
- anti-aging treatment

How It Works / Mechanism

MOTS-c appears to help cells respond to metabolic stress.

Simple Version

When a cell's energy needs change, MOTS-c appears to help send signals that tell the cell:

> **adjust how you use fuel and respond to the stress.**

Research suggests MOTS-c can affect pathways involving:

folate metabolism → AICAR → AMPK-related signaling

MOTS-c can also move into the nucleus during metabolic stress and influence gene activity.

Important

Do **not** simplify this to:

> "MOTS-c binds to the AMPK receptor."

AMPK is not the known MOTS-c receptor.

FDA's review says MOTS-c's molecular target remains unknown. (fda.gov)

Common Research Dose

Human Clinical Research

Current Phase 2a

Route: SC
Treatment duration: 12 weeks

Exact clinical dose information should be copied directly from the trial record if and when it is publicly disclosed clearly.

FDA Compounding Nomination

Products were proposed at:

5 mg

and:

10 mg

SC. (fda.gov)

Important

These are:

proposed compounded doses

not:

FDA-approved doses

Research Community

Community protocols commonly describe:

5–10 mg

several times per week.

Research Duration / Cycle Length

Human Trial

12 weeks of treatment

with additional safety follow-up. (clinicaltrials.gov)

Community

Commonly:

4–6 weeks on

followed by:

2–4 weeks off

Evidence Assessment

The clinical study duration is simply:

how the trial was designed.

The community cycle is:

a community convention.

Neither proves that MOTS-c biologically requires cycling.

How Often It Is Used / Frequency

Human Research

Repeated SC treatment is currently being studied.

Community

Often:

2–3 times per week

with many other schedules also discussed.

Evidence Assessment

We do not have completed human research telling us which schedule works best.

Possible Benefits

Human Evidence

Natural Exercise Response

The MOTS-c produced naturally by the human body increases with exercise. (pubmed.ncbi.nlm.nih.gov)

Metabolic Associations

Human observational studies have found relationships between MOTS-c and metabolic characteristics.

These findings are interesting.

They do **not** prove injections are beneficial.

Animal / Cell Evidence

Insulin Sensitivity

Animal research suggests MOTS-c can improve insulin sensitivity.

Glucose Metabolism

Preclinical studies show changes in glucose handling.

Metabolic Health

Foundational research found important metabolic effects in animal models.

Exercise / Physical Capacity

Animal studies have reported improved physical capacity.

Important

These remain:

animal or laboratory treatment effects

until human injection trials confirm them.

What It Does Not Help With, Based on Current Evidence

Do not present injected MOTS-c as proven to:

- cause fat loss
- reverse insulin resistance
- treat diabetes
- improve endurance
- increase energy
- replace exercise
- prevent muscle loss
- treat GLP-1 fatigue
- extend lifespan
- reverse aging
- build new mitochondria in humans
- increase human fat burning after injection

Commonly Paired With

NAD+

Different role in cellular energy metabolism.

Classification: Could be complementary, but human combination evidence does not exist.

SS-31 / Elamipretide

Different mitochondrial mechanism.

Classification: Different pathways; combination untested.

L-Carnitine

Helps transport fatty acids into mitochondria.

Classification: Different mechanism; combination unproven.

GLP-1 Agonists

Different signaling pathways.

Classification: Different mechanisms, but no evidence MOTS-c prevents GLP-1-related fatigue.

AICAR

Greater overlap with AMPK-related signaling.

Classification: More pathway overlap; combination untested.

Dosage Information

Native MOTS-c Human Trial

Phase: 2a
Route: Subcutaneous
Treatment: 12 weeks
Follow-up: Additional safety follow-up
Population: Adults with prediabetes and overweight/obesity
Estimated enrollment: About 120. (clinicaltrials.gov)

Important

This is:

an experimental research regimen

not:

an approved MOTS-c protocol

Community Research Protocol

Community Pattern

PeptideFox currently describes approximately:

Dose: 5–10 mg
Route: SC or IM
Frequency: commonly several times weekly

Timing: often morning / around exercise
Cycle: commonly 4–6 weeks followed by a break

Evidence Check

SC route: Being studied in humans

IM route: Community use

5–10 mg: Community dosing

Several times weekly: Community dosing

Pre-workout: Makes sense in theory but unproven

Morning: Community preference

4–6 week cycle: Community convention

2–4 week break: No proven reset requirement

Current Live Navaro Protocol Review

The current live page contains several very different protocols.

Examples include:

- 5 mg weekly followed by twice weekly
- 10 mg weekly
- 200 mcg → 1 mg daily
- 5 mg every 5 days
- 3 mg every 3 days

Problem

There is no human dose-ranging study showing that one of these is the:

standard MOTS-c protocol

Recommendation

Replace:

> **Standard Protocol**

with:

> **Community Protocol Examples**

Then put the current human Phase 2a research **above** the community protocols.

Community Protocol Comparison

Source	Dose	Route	Frequency	Duration	Evidence
Current Phase 2a	Not established here	SC	Repeated treatment	12 weeks	Ongoing human trial
FDA nomination	5 / 10 mg proposed	SC	Proposed	Not validated	Compounding nomination
PeptideFox	5–10 mg	SC / IM	Several times weekly	Short cycles	Community
Live Navaro examples	Variable	SC	Variable	Variable	Community

Strongest Agreement

SC is currently the route with the strongest human-research precedent.

Biggest Unknown

Dose and frequency

We simply do not have completed human data establishing the best regimen.

Expected Results Timeline, Expanded

Human Clinical Research

No Results Timeline Yet

The active study is evaluating metabolic outcomes after approximately:

12 weeks

of treatment. (clinicaltrials.gov)

Until the results are published, Navaro should not say:

> MOTS-c starts working by week 2.

Natural Human MOTS-c Research

Exercise can change the MOTS-c naturally present in the human body. (pubmed.ncbi.nlm.nih.gov)

That tells us something about MOTS-c biology.

It does **not** tell us how quickly an injection produces a treatment effect.

Community-Reported

Community sources describe subjective changes over:

several days to several weeks

with many protocols lasting around:

4–6 weeks

Evidence Warning

Always label these:

Community-reported

not:

Expected clinical results

Reported Side Effects

Controlled Human Injection Data

Not established yet

We do not currently have a completed native-MOTS-c treatment trial that gives reliable side-effect percentages.

FDA Safety Concerns

FDA highlighted uncertainty around:

- immune reactions
- peptide aggregation
- impurities
- active ingredient identity
- manufacturing quality. (fda.gov)

Community-Reported Effects

Community material describes things such as:

- injection-site irritation
- fatigue
- temporary discomfort
- occasional stomach symptoms

Important

These are:

anecdotal reports

not:

established clinical adverse-effect rates

Contraindications & Cautions

Formal Contraindications

None established

There is no FDA-approved MOTS-c medication and therefore no official product-label contraindication list.

Important Cautions

Pregnancy / Breastfeeding

No adequate human safety data.

Children / Adolescents

No established injected-MOTS-c safety or efficacy.

Diabetes / Glucose-Lowering Drugs

An interaction is biologically possible because MOTS-c is being studied for metabolic effects.

Actual human interaction risk is unknown.

Active Cancer

Human safety is unknown.

Serious Cardiovascular Disease

Human injection safety is not established.

Immune Reactions

FDA specifically identified immunogenicity as an area of concern with injectable compounded MOTS-c products. (fda.gov)

Regulatory Status

United States

FDA Approved?

No

Neither MOTS-c free base nor MOTS-c acetate is part of an FDA-approved drug. (fda.gov)

Human Trial Status

Phase 2a underway

The active study is testing MOTS-c in adults with:

- prediabetes
- overweight or obesity. (clinicaltrials.gov)

Compounding

FDA evaluated MOTS-c free base and MOTS-c acetate in 2026.

FDA staff concluded that the available evidence weighed:

against inclusion

on the 503A Bulks List. (fda.gov)

WADA

Prohibited

The 2026 WADA Prohibited List specifically includes MOTS-c as an example of an AMPK activator. (wada-ama.org)

What Studies Have Found

Foundational MOTS-c Research

The foundational research identified MOTS-c as a mitochondrial-derived peptide involved in metabolic regulation.

Animal and laboratory work found effects involving:

- metabolic homeostasis
- insulin resistance
- obesity-related metabolism

What This Supports

- MOTS-c is biologically active
- metabolic signaling is real
- AMPK-related mechanisms are plausible

What It Does Not Support

- a human 5 mg protocol
- human fat loss
- human endurance enhancement
- human anti-aging therapy

Human Exercise Research

Human research shows that:

natural MOTS-c increases with exercise. (pubmed.ncbi.nlm.nih.gov)

What This Supports

MOTS-c appears to participate in normal human exercise biology.

What It Does Not Support

A clinically proven injection protocol:

- 30 minutes before exercise
- 60 minutes before exercise
- 90 minutes before exercise

MOTS-MET Phase 2a Trial

The current trial is:

NCT07505745

It is studying approximately:

120 adults

with prediabetes and overweight or obesity.

The trial compares:

MOTS-c

with:

placebo

over approximately:

12 weeks

with further safety follow-up. (clinicaltrials.gov)

Main Question

Can MOTS-c improve:

insulin sensitivity?

Current Status

Ongoing

Results

Not available yet

FDA 2026 Evaluation

FDA's review is particularly useful for separating what we know from what peptide websites claim.

FDA reported that its literature search did not identify human administration studies that could establish MOTS-c's effectiveness or safety and noted that the exact molecular targets remain unknown. (fda.gov)

What This Means for Navaro

Do not describe these as established facts:

- proven human insulin-sensitivity improvement
- proven endurance improvement
- hypoglycemia as the major known side effect
- known human half-life
- standard 5 mg dosing
- mandatory pre-workout use

Formulation / Stability / Sterility

No FDA-Approved MOTS-c Product Exists

There is no approved reference product giving us one universal:

- vial strength
- mixing method
- diluent
- storage temperature after mixing
- expiration after reconstitution

FDA also reviewed free-base and acetate forms separately and identified significant characterization concerns. (fda.gov)

Free Base vs Acetate

FDA considers:

- MOTS-c free base
- MOTS-c acetate

to be different bulk drug substances. (fda.gov)

Simple Version

Do not assume every vial labeled:

> **MOTS-c**

is chemically identical.

Community Research Vials

Community sources give their own instructions for:

- bacteriostatic water
- refrigeration
- use-after-mixing periods

Those instructions should be labeled:

Community handling practices

not:

Validated universal MOTS-c stability

Simple Rule

Do not apply one 14-day, 28-day or 30-day storage rule to every MOTS-c vial.

Research Sources

1. Lee et al. 2015: Foundational MOTS-c Research

Evidence type: Foundational preclinical research

Supports:

- 16-amino-acid mitochondrial-derived peptide
- mitochondrial DNA origin
- metabolic signaling
- animal insulin-resistance and obesity findings
- AMPK-related pathway

`https://pubmed.ncbi.nlm.nih.gov/25738459/`

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2. Reynolds et al. 2021: MOTS-c and Exercise

Evidence type: Human physiological + animal experimental research

Supports:

- exercise-related endogenous MOTS-c changes in humans
- human muscle and circulating MOTS-c response
- animal physical-capacity research

Does **not** establish injected-human treatment efficacy.

`https://pubmed.ncbi.nlm.nih.gov/33473109/`

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3. von Walden et al. 2021: Acute Endurance Exercise

Evidence type: Human physiological research

Supports:

- acute endurance exercise changes circulating mitochondrial-derived peptides

Does not establish a pre-workout MOTS-c injection protocol.

`https://pubmed.ncbi.nlm.nih.gov/34351816/`

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4. ClinicalTrials.gov: MOTS-MET Phase 2a

Evidence type: Active human clinical trial

Supports:

- Phase 2a human research
- adults with prediabetes and overweight/obesity
- SC administration
- 12-week treatment
- insulin-sensitivity research
- human safety monitoring

`https://clinicaltrials.gov/study/NCT07505745`

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5. FDA 2026 MOTS-c 503A Evaluation

Evidence type: FDA scientific and regulatory review

Supports:

- no FDA-approved MOTS-c drug

- lack of established human administration evidence in FDA's review
- unknown human safety
- insufficient effectiveness evidence
- immunogenicity concerns
- free-base vs acetate concerns
- FDA staff conclusion against 503A inclusion

https://www.fda.gov/media/193347/download

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6. FDA: Bulk Drug Substances That May Present Significant Safety Risks

Evidence type: FDA safety and regulatory information

Supports:

- immunogenicity concerns
- peptide impurity concerns
- active-ingredient characterization concerns
- lack of adequate human safety information

https://www.fda.gov/drugs/human-drug-compounding/certain-bulk-drug-substances-use-compoundi

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7. FDA: July 2026 Pharmacy Compounding Advisory Committee

Evidence type: Regulatory-process source

Supports:

- 2026 review of MOTS-c free base and acetate

https://www.fda.gov/advisory-committees/advisory-committee-calendar/july-23-24-2026-meeting-

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8. WADA 2026 Prohibited List

Evidence type: Anti-doping regulation

Supports:

- MOTS-c listed as an AMPK activator
- prohibited status under WADA rules. (wada-ama.org)

https://www.wada-ama.org/sites/default/files/2025-09/2026list_en_final_clean_september_2025.

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9. PeptideFox: MOTS-c

Evidence type: Community / practitioner protocol

Useful for documenting:

- community dosing
- multiple weekly injections
- SC / IM use
- exercise timing
- cycling
- anecdotal timelines
- community handling practices

It should **not** override FDA, ClinicalTrials.gov or controlled human evidence.

`https://peptidefox.com/content/mots-c`

Final Takeaway

MOTS-c has interesting science behind it, but human injection evidence is still very early.

What we know:

- MOTS-c is a real **16-amino-acid mitochondrial-derived peptide**
- its genetic instructions come from mitochondrial DNA
- it participates in metabolic signaling
- natural human MOTS-c changes with exercise
- animal studies show important metabolic effects
- a human Phase 2a treatment trial is now underway. (fda.gov)

What we still do **not** know:

- the best human dose
- the best frequency
- the best injection timing
- its injected-human half-life
- whether it actually improves insulin sensitivity
- whether it causes fat loss
- whether it improves endurance
- whether it improves fatigue
- its true human side-effect rates
- whether cycling provides any benefit

Community protocols commonly use approximately:

5–10 mg

several times per week, often around exercise.

Those belong under:

Community Protocols

not:

Clinical Dosing

The biggest live-page fixes remain:

> **Do not call AMPK the MOTS-c receptor. The exact target is still unknown.**
>
> **Do not require pre-workout dosing.**
>
> **Do not describe low blood sugar as the established major MOTS-c side effect. Human injection safety data are not available yet.**
>
> **Do not label one community schedule the "Standard Protocol."**
>
> **Put the active Phase 2a human trial above the community dosing section.**
>
> **Keep MOTS-c clearly labeled as not FDA approved and WADA prohibited.** (fda.gov)

NAD+

Cellular Energy • Redox Metabolism • Mitochondrial Cofactor Research

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At a Glance

What is NAD+?

NAD+ stands for:

Nicotinamide Adenine Dinucleotide

It is a molecule naturally found in almost every cell in the body.

NAD+ is essential for normal cellular metabolism.

One of its most important jobs is helping the body turn nutrients into usable cellular energy.

It also plays a role in:

- redox chemistry
- DNA repair
- cellular stress responses
- sirtuin activity
- other NAD-dependent enzymes

NAD+ is **not a peptide, hormone or stimulant**.

It is a naturally occurring:

coenzyme

How well studied is it?

NAD+ biology is extremely well understood. NAD+ injections are much less studied.

This distinction matters.

Scientists know a great deal about:

- what NAD+ does inside cells
- NAD+/NADH chemistry
- how the body makes and recycles NAD+
- enzymes that use NAD+
- NAD+ precursors such as NR and NMN

But that does **not** mean injecting NAD+ has equally strong clinical evidence.

Direct NAD+ administration has mainly been studied through:

- small IV studies
- short human metabolism studies
- early tolerability research
- very small preliminary IM and SC exposure studies

A 2026 preliminary safety analysis reported small human groups receiving:

100 mg NAD+

by:

- IM
- SC
- IV-push

on:

3 consecutive days

This is important because it gives us early controlled human exposure through IM and SC routes.

But it does **not** prove those doses provide clinical benefits.

Evidence level

Basic NAD+ biology: Very strong
Human NAD+ metabolism research: Strong
Direct IV NAD+ research: Limited but real
Direct IM/SC human research: Very early
Energy / fatigue treatment evidence: Limited
Anti-aging evidence from injections: Unproven
Community injection protocols: Common but mostly practice-derived

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What may it help with?

Strong biological evidence

Cellular Energy Metabolism

NAD+ is required for normal energy metabolism.

It helps the body process:

- carbohydrates
- fats
- amino acids

and turn them into usable cellular energy.

Important

NAD+ being necessary for energy production does **not** automatically mean:

> injecting more NAD+ gives a healthy person more energy.

Those are two different claims.

Redox Balance

NAD+ and NADH work together as part of a redox system.

Simple version

NAD+ accepts electrons → becomes NADH

and:

NADH gives up electrons → becomes NAD+

The body constantly cycles between these forms.

This process is central to normal metabolism.

NAD-Dependent Cell Signaling

NAD+ is also used by enzymes including:

- sirtuins
- PARPs
- CD38-related enzymes

These are involved in things such as:

- DNA repair
- metabolism
- cellular stress responses
- gene regulation

Direct Human NAD+ Research

Direct IV NAD+ has been shown to enter human metabolism.

In a 2019 pilot study, NAD+ was infused at:

3 µmol/min

for:

6 hours

Researchers found that infused NAD+ was rapidly removed from the blood early in the infusion and was then processed into related metabolites.

What This Supports

Injected or infused NAD+:

does enter and affect human NAD-related metabolism.

What It Does Not Prove

It does not prove that NAD+ infusion:

- treats fatigue
- improves memory
- reverses aging
- causes fat loss
- improves sports performance
- treats addiction

- repairs mitochondria

in otherwise healthy people.

What has it NOT been proven to help with?

Direct NAD+ injections have **not been convincingly established** as treatments for:

- aging
- longevity
- general chronic fatigue
- bodybuilding
- muscle growth
- fat loss
- athletic performance
- brain fog
- depression
- hangovers
- alcohol detoxification
- liver detoxification
- opioid or substance-use disorder
- GLP-1-related fatigue
- mitochondrial “rejuvenation”
- preventing age-related disease
- DNA repair as a measurable clinical outcome

NAD+ is involved in many of these biological systems.

That does **not** prove injecting extra NAD+ produces a meaningful treatment effect.

Dose at a Glance

FDA-Approved Dosing

There is no FDA-approved general NAD+ injection protocol

There is no FDA-approved NAD+ injection dose for:

- energy
- wellness
- anti-aging
- fatigue
- weight loss
- athletic recovery

Compounded NAD+ products exist, but that is not the same thing as having an FDA-approved NAD+ drug.

Human Research Dosing

2019 IV Metabolism Study

Researchers infused NAD+ at:

3 µmol/min

for:

6 hours

This was a metabolism and pharmacology study.

It was **not** designed to establish a wellness dose.

2026 IV Tolerability Study

A newer human study used:

500 mg NAD+ IV

on:

4 consecutive days

Participants receiving NAD+ had more infusion-related symptoms than those receiving IV nicotinamide riboside.

Reported effects included:

- stomach and digestive symptoms
- increased heart rate
- chest pressure

Important

This was mainly a tolerability comparison.

It does **not** prove that:

500 mg is the ideal NAD+ treatment dose.

2026 IM / SC / IV-Push Pilot

A **2026 preliminary safety analysis associated with this research program** reported small human groups receiving:

100 mg NAD+

by:

- IM
- SC
- IV-push

on:

3 consecutive days

Important

This report is a:

preprint

That means it provides useful early human information, but it has not yet gone through the same peer-review process as a published clinical paper.

The participant groups were also very small.

What This Changes

We can now say:

> **There is preliminary controlled human exposure to 100 mg NAD+ by IM and SC injection.**

We should **not** say:

> **100 mg IM or SC NAD+ is a clinically proven treatment dose.**

Evidence Classification

Preliminary human safety / tolerability evidence

Not:

Established efficacy evidence

Research-Community Dosing

Community protocols are much more developed than the clinical evidence.

PeptideFox

IM

Approximately:

50–200 mg per injection

commonly:

2–3 times per week

during more active use.

Less frequent maintenance schedules are also discussed.

SC

Approximately:

50–100 mg per dose

Smaller divided injections are often used because SC NAD+ can be uncomfortable or irritating.

Lower starting doses around:

25 mg

are also discussed for tolerance testing.

Evidence level

Community / practitioner protocol

The upper ends of these dose ranges have not been validated by completed human dose-finding trials.

PeptideDosages

PeptideDosages describes:

Week 1: 50 mg SC daily
Week 2: 75 mg SC daily
Week 3 onward: 100 mg SC daily

for approximately:

8–16 weeks

Evidence level

Community protocol

The exact:

50 → 75 → 100 mg

dose ladder and:

8–16 week cycle

have not been proven by a matching clinical NAD+ efficacy trial.

Frequency at a Glance

Human Clinical Research

Human research has used very different schedules.

Examples include:

- continuous IV infusion over 6 hours
- 500 mg IV on four consecutive days
- 100 mg NAD+ by IM, SC or IV-push over three consecutive days in a small preliminary analysis

There is therefore:

no single clinically established NAD+ injection frequency for general wellness.

Community

IM

Commonly:

2–3 times weekly

SC

Ranges from:

several times per week

to:

daily

depending on the protocol.

Best-Supported Frequency?

Unknown

Human research does not show that any of these is clearly better:

- daily SC
- 3x-weekly IM
- weekly IM
- monthly IV

for general metabolic health.

Timing at a Glance

Clinical Research

There is no established requirement to take NAD+:

- in the morning
- at night
- fasted
- before exercise
- after exercise
- with carbohydrates

Community

Morning use is common because NAD+ is marketed for energy and some users describe feeling more alert.

That remains:

community preference

rather than a proven timing rule.

Evidence verdict

Morning required: No
Night required: No
Fasted required: No
Pre-workout required: No
With food required: No

Duration / Cycle at a Glance

Clinical Research

There is no universal NAD+ treatment cycle.

Human direct-NAD+ studies have used:

- one prolonged IV exposure
- several consecutive treatment days
- short experimental protocols

Community

Common schedules include:

- short IV “loading” series
- 8–16 weeks of SC injections
- intermittent IM injections
- weekly maintenance use

Does NAD+ Need to Be Cycled?

No receptor-reset cycle has been established.

NAD+ is not a conventional receptor agonist.

There is no established reason for:

- 5 days on / 2 days off
- 8 weeks on / 4 weeks off
- 12-week receptor resets
- mandatory washouts

Expected Results Timeline

Human Clinical Research

During IV Administration

NAD+ enters human metabolism immediately after administration.

But in the 2019 IV study, circulating NAD+ did not simply keep building up in the blood.

It was rapidly being:

- removed
- broken down
- recycled
- converted into related metabolites

Important

A biochemical change is **not the same thing as**:

feeling more energetic.

Clinical Benefit Timeline

Not established

There is no strong human clinical evidence showing a predictable timeline such as:

Day 1: more energy
Week 1: better thinking
Week 2: improved mitochondria
Week 4: better body composition

from direct NAD+ injections.

Those should not be presented as clinical facts.

Community-Reported Timeline

Community sources often describe effects such as:

- energy
- alertness
- recovery
- general wellness

within:

hours to days

of IV or injected NAD+.

Longer protocols describe changes over several weeks.

Important

These remain:

anecdotal reports

Best Top-Card Wording

Clinical: NAD+ is processed rapidly after administration, but there is no established timeline for meaningful energy, wellness or anti-aging benefits.

Community-reported: some people describe subjective effects within the same day or during the first several treatments, but these experiences have not been established as controlled clinical outcomes.

Safety at a Glance

IV NAD+ Can Be Difficult to Tolerate

A newer study using:

500 mg IV NAD+

reported symptoms including:

- gastrointestinal discomfort
- increased heart rate
- chest pressure

The NAD+ infusions took considerably longer than the comparison infusions because symptoms limited how quickly they could be given.

Important

Infusion speed appears to matter.

That does **not** mean faster infusion works better.

IM / SC Safety

A 2026 preliminary analysis gives us a small amount of controlled human experience with:

100 mg NAD+

by:

- IM
- SC
- IV-push

over three consecutive days.

No severe unexpected treatment-related event was reported in that small preliminary analysis.

But each route group included only a small number of people.

Meaning

This gives us:

early short-term tolerability data

not:

established long-term safety

FDA Injectable Product-Quality Warning

This is one of the most important NAD+ safety issues.

FDA has received adverse-event reports after compounded NAD+ injections involving:

- severe chills
- shaking
- vomiting
- fatigue

Some people required medical treatment.

FDA said these reactions were consistent with:

excessive endotoxin exposure

and warned that food-grade NAD+ ingredients are not appropriate for sterile drug compounding without proper pharmaceutical processing.

Important Distinction

This does **not** necessarily mean NAD+ itself caused every reaction.

It highlights a separate problem:

> **Poorly prepared injectable products can cause serious harm even if the active ingredient itself is not the main problem.**

Formal Contraindications

No FDA-approved NAD+ injection label exists

There is no official FDA contraindication list for injectable NAD+ used for:

- wellness
- energy
- anti-aging

Navaro should not create a formal contraindication list by turning every theoretical concern into an official contraindication.

Important Cautions

Pregnancy / Breastfeeding

There is not enough direct injectable-NAD+ safety evidence.

Classification: Safety unknown.

Significant Cardiovascular Disease

Higher-dose IV NAD+ has been associated with:

- increased heart rate
- chest pressure
- significant infusion discomfort

Classification: Important caution.

Sterile Product Quality

Contamination and endotoxin exposure are serious risks for poorly prepared injectable products.

Classification: Important product-quality concern.

Regulatory Status at a Glance

Is NAD+ Naturally Present in the Body?

Yes

FDA-Approved NAD+ Injection for Energy or Anti-Aging?

No

FDA-Approved Wellness Protocol?

No

Do Compounded NAD+ Injections Exist?

Yes

But compounded products are:

not FDA-approved NAD+ drugs

FDA has also raised concerns about the ingredients and manufacturing quality used in some compounded injectable NAD+ products.

Important 2026 Regulatory Update

In a January 2026 warning letter to a registered outsourcing facility, FDA said NAD+ did:

not appear on the 503B Bulks List

and that the cited NAD+ products therefore did not qualify for the relevant 503B compounding exemptions.

Important

Do **not** simplify this to:

> "All NAD+ compounding is illegal."

That is too broad.

The rules depend on:

- the type of compounding facility
- the regulatory pathway
- the exact substance
- the specific product
- applicable compounding law

Better Wording

> **There is no FDA-approved NAD+ injection for wellness or anti-aging. FDA has also raised regulatory and product-quality concerns involving some compounded NAD+ injectables.**

Bottom Line

NAD+ itself is one of the most important and best-established molecules in cellular metabolism. Injecting NAD+ is a much less certain question.

What is firmly established:

- NAD+ naturally exists in cells
- it is essential for energy metabolism
- it participates in redox chemistry
- many important enzymes use it
- directly administered NAD+ enters human metabolism

What is **not firmly established**:

- the best injection dose
- the best route
- the best frequency
- anti-aging benefit
- general fatigue treatment
- cognitive enhancement
- fat-loss benefit
- athletic-performance benefit
- GLP-1 fatigue prevention
- long-term IM/SC safety

Community protocols often use:

50–200 mg IM

several times per week

or:

50–100 mg SC

with schedules ranging from intermittent to daily.

These are worth documenting.

They should **not** be presented as established medical dosing protocols.

Protocol Logic Check

Quick Logic Summary

Protocol Question	Verdict
Is NAD+ essential for cellular energy metabolism?	**Yes, very well established**
Is NAD+ a peptide?	**No**
Does administered NAD+ enter human metabolism?	**Yes**
Has IV NAD+ been studied in humans?	**Yes, but evidence is limited**
Has IM NAD+ been given to humans in controlled research?	**Yes, very early 100 mg data**
Has SC NAD+ been given to humans in controlled research?	**Yes, very early 100 mg data**
Is 100 mg IM/SC proven effective?	**No**
Is 50–200 mg IM 2–3x/week clinically proven?	**No, community protocol**
Is 50–100 mg SC daily clinically proven?	**No, community protocol**
Is 50 → 75 → 100 mg titration proven?	**No, community protocol**
Does morning dosing work better?	**Not proven**
Does fasted dosing work better?	**Not proven**
Does pre-workout dosing work better?	**Not proven**
Does NAD+ need cycling?	**No demonstrated reason**
Does it need a receptor reset?	**No**
Is an IV “loading series” clinically established?	**Community / clinic convention**
Does IV automatically mean better tissue delivery?	**Not established**
Is NAD+ the same as NR or NMN?	**No**
Is every compounded NAD+ vial equivalent?	**No**
Is one universal 14/28-day storage rule valid?	**No**
FDA approved for anti-aging?	**No**

What Does NAD+ Actually Do?

NAD+ does not work like:

liraglutide → GLP-1 receptor

or:

Tesamorelin → GHRH receptor

Instead, NAD+ takes part directly in cellular chemistry.

Simple Version

Think of NAD+ as a reusable helper molecule.

It repeatedly:

accepts electrons → carries them → gets recycled

while the body turns nutrients into energy.

NAD+ is also used by enzymes involved in:

- DNA repair
- metabolism
- cellular stress responses
- gene regulation

Verdict

Its biological role is extremely well established, but NAD+ does not have one single therapeutic receptor target.

NAD+ vs NADH

These are two forms of the same redox system.

NAD+

The oxidized form.

It can:

accept electrons

NADH

The reduced form.

It:

carries electrons

Simple Pathway

NAD+ ⇄ NADH

The body constantly converts one into the other.

Important

Research on NADH should not automatically be used as evidence for:

injected NAD+

They are closely related biologically, but they are not the same administered product.

NAD+ vs NR vs NMN

This is another major source of confusion.

NAD+

The final coenzyme itself.

NR

Nicotinamide riboside

A precursor the body can use to make NAD+.

NMN

Nicotinamide mononucleotide

Another NAD-related precursor.

Important

A study showing:

> oral NR raises NAD+

does **not** automatically prove:

> injecting NAD+ has the same effect or benefit.

Each molecule and route needs its own evidence.

Does Injected NAD+ Go Straight Into Cells?

Not that simply.

NAD+ is a relatively large, charged molecule.

Outside cells, NAD+ can be:

- broken down
- converted
- recycled into other NAD-related compounds

before components are taken up and reused.

The 2019 human IV study showed that infused NAD+ was rapidly removed from the blood and processed into related metabolites.

Better Wording

> **Injected NAD+ enters human metabolism, but it should not be described as going directly from the syringe into every mitochondrion intact.**

Verdict

Claims like:

> “100% bioavailable directly into your mitochondria”

are too simplistic and misleading.

Human Half-Life

No Simple Clinically Useful Injection Half-Life Is Established

Direct NAD+ is rapidly:

- metabolized
- broken down
- recycled
- converted through multiple pathways

So a statement such as:

> “NAD+ has a half-life of X hours, therefore inject every X hours”

would be misleading.

Verdict

Do not build a community dosing schedule from one plasma half-life number.

How Long Do the Effects Last?

We do not know clearly.

Even if extracellular NAD+ itself is processed quickly:

- downstream metabolites may last longer
- intracellular NAD pools may behave differently
- biological effects may outlast circulating NAD+

We do not have enough direct human injection research to say:

- how long one SC dose “works”
- how long one IM dose “works”
- when another dose is physiologically required

Verdict

Unknown

Does It Build Up With Repeated Use?

Possibly at the level of:

- metabolites
- intracellular NAD pools
- related compounds

depending on route and exposure.

But injected NAD+ is heavily processed.

There is not enough evidence to say:

> "NAD+ accumulates for X days."

Verdict

The metabolism is complex and clinically useful accumulation data are limited.

Does Daily SC Dosing Make Sense?

PeptideDosages uses:

50 → 75 → 100 mg SC daily

There is now preliminary human exposure to:

100 mg SC

on three consecutive days.

That means short-term repeated SC use is no longer supported only by anecdotes.

But the early research does **not** validate:

- 8 weeks
- 12 weeks
- 16 weeks
- permanent 100 mg daily use
- anti-aging benefit
- energy improvement

Verdict

100 mg SC now has very early short-term human tolerability precedent. Long-term daily protocols remain unproven.

Does 2–3 Times Weekly IM Make Sense?

Community protocols commonly use:

50–200 mg IM

approximately:

2–3 times weekly

The 2026 preliminary analysis gives us initial controlled human experience with:

100 mg IM

But it does not validate:

- 200 mg IM
- 2–3x-weekly chronic dosing
- long-term use
- wellness efficacy

Verdict

100 mg IM has very early human safety precedent. The broader community protocol remains unvalidated.

Does Morning Dosing Make Sense?

Some people prefer morning injections because NAD+ is marketed for energy.

There is no clinical evidence showing morning dosing produces better:

- energy
- metabolism
- cognition
- exercise performance

Verdict

Community preference

Does Fasted Dosing Make Sense?

There is no good direct-NAD+ evidence showing fasting improves:

- absorption
- cellular uptake
- energy
- mitochondrial effects
- fat loss

Verdict

Unproven

Pre-Workout Timing

NAD+ is important for cellular energy metabolism.

That alone does **not** prove injecting NAD+ before exercise improves performance.

Verdict

No established advantage

Does NAD+ Need a Loading Phase?

Some IV clinics use several high-dose infusions over a short period.

Community examples include:

4–5 IV sessions over about 2 weeks

No randomized human trial has established that this is the best way to “restore” NAD+ in otherwise healthy adults.

Verdict

Clinic / community convention

Does It Need an 8–16 Week Cycle?

Some community SC protocols use:

8–16 weeks

There is no known biological event at week:

- 8
- 12
- 16

that means NAD+ must suddenly be stopped.

Verdict

Community framework, not a proven biological requirement

Does It Need a Receptor Reset?

No.

NAD+ is not acting like a conventional receptor-stimulating drug.

There is no established:

- NAD receptor reset

- receptor desensitization cycle
- 2–4 week recovery period

Verdict

No receptor-reset logic

Dose Escalation / Titration

Starting lower may make practical sense because injected NAD+ can be uncomfortable.

But the specific:

50 → 75 → 100 mg

community ladder is not a medically established titration schedule.

Verdict

Community titration based on tolerability

Does Route Matter?

IV

Most historical direct-NAD+ human research.

IM

Very early controlled human exposure now exists at:

100 mg

SC

Very early controlled human exposure now exists at:

100 mg

Community

All three routes are used.

Verdict

The routes should not be treated as interchangeable simply because they contain NAD+.

Different routes may produce different:

- exposure
- discomfort

- metabolism
- tolerability
- dosing needs

IV Infusion Speed Logic

This matters.

In a study using:

500 mg IV NAD+

participants experienced:

- GI symptoms
- increased heart rate
- chest pressure

The NAD+ infusions took much longer than the comparison infusions because symptoms limited how quickly NAD+ could be administered.

What This Tells Us

Faster is not necessarily better.

Verdict

Infusion speed can strongly affect tolerability.

Do not describe symptoms such as:

- chest pressure
- nausea
- severe discomfort

during rapid infusion as:

> “proof the NAD+ is working.”

Stack / Pairing Logic

NAD+ + MOTS-c

They affect different parts of cellular energy metabolism.

Verdict: Could be complementary in theory, but no validated human combination protocol exists.

NAD+ + SS-31 / Elamipretide

NAD+ participates in redox metabolism.

SS-31 affects mitochondrial membrane and cardiolipin biology.

Verdict: Different mechanisms, but the combination is clinically unvalidated.

NAD+ + L-Carnitine

L-Carnitine helps move fatty acids into mitochondria.

NAD+/NADH helps with the chemistry used to extract energy from those fuels.

Verdict: Different but related metabolic roles; no established injected combination protocol.

NAD+ + GLP-1 Drugs

NAD+ is often promoted for fatigue during GLP-1 treatment.

There is no controlled human evidence showing injectable NAD+:

- prevents GLP-1 fatigue
- prevents muscle loss
- improves GLP-1 weight loss

Verdict

Different mechanism, but the claimed benefit is unproven

NAD+ + NR / NMN

All of these affect NAD metabolism.

Verdict

Biologically related and potentially overlapping in purpose, but their pharmacology is different.

There is no universally proven reason to use all of them together.

Important Research Note

Four different evidence types are commonly mixed together online.

1. Normal NAD+ Biology

This is extremely well established.

NAD+ is essential to normal human metabolism.

---

2. NAD+ Precursor Research

This includes:

- NR
- NMN
- niacin
- nicotinamide

This research teaches us a great deal about NAD metabolism.

But:

precursor evidence is not automatically NAD+ injection evidence.

---

3. Direct Human NAD+ Research

This remains relatively small.

It includes:

- IV metabolism studies
- IV tolerability studies
- early preliminary IM / SC / IV-push exposure data

---

4. Community / Clinic Protocols

These include:

- 500–1,000 mg IV
- 50–200 mg IM
- 50–100 mg SC
- loading series
- maintenance injections
- 8–16 week cycles

These are useful to document.

They are **not equivalent to established clinical dosing.**

Research at a Glance

Approved-Label Human Evidence

None for NAD+ wellness injections

There is no FDA-approved NAD+ injection for:

- energy
- wellness
- anti-aging

Direct Human Clinical Research

Limited but growing

Includes:

- direct IV studies
- IV tolerability research
- very early IM / SC human exposure

Human Biological Evidence

Extremely Strong

NAD+/NADH chemistry and NAD metabolism are foundational human biochemistry.

Animal / Cell Evidence

Extensive

Many aging, metabolic and disease models involve changes in NAD metabolism.

Mechanistic Evidence

Very Strong for natural NAD+ biology

Much weaker for predicting:

what happens clinically when extra NAD+ is injected.

Community / Anecdotal Evidence

Common but much weaker

A large wellness market exists around injectable NAD+.

The clinical evidence has not caught up with many of the claims.

Overview

NAD+ is one of the most important molecules in human metabolism.

Every cell needs it.

That makes NAD+ marketing sound immediately convincing:

> “If NAD+ helps make energy, more NAD+ must give you more energy.”

But the biology is more complicated.

The body tightly controls:

- NAD+ production
- recycling
- breakdown
- transport
- intracellular levels

And NAD+ outside cells is heavily metabolized.

So the important question is not:

> **Is NAD+ important?**

That answer is clearly:

Yes.

The real question is:

> **Does injecting extra NAD+ improve meaningful health outcomes in people?**

For most wellness claims:

we still do not know.

Evidence Strength

Overall Evidence Rating

Extremely strong biology, limited direct-injection evidence

Strong Evidence For

- NAD+/NADH chemistry
- cellular energy metabolism
- NAD-dependent enzymes
- direct IV NAD+ metabolism
- route-dependent tolerability

Early Human Evidence For

- 100 mg IM NAD+ short-term exposure
- 100 mg SC NAD+ short-term exposure
- 100 mg IV-push NAD+ short-term exposure

Limited or Unproven For

- anti-aging
- longevity
- fatigue treatment
- athletic enhancement
- fat loss
- cognition
- hangovers
- alcohol detoxification
- GLP-1 fatigue
- long-term 50–200 mg IM protocols
- daily 50–100 mg SC protocols
- loading cycles
- maintenance cycles

Category

Cellular Metabolism / Mitochondrial Energy / Redox Cofactors

Compound

NAD+

Full name:

Nicotinamide Adenine Dinucleotide

Compound Type

Naturally occurring dinucleotide coenzyme

NAD+ is:

- naturally present in cells
- essential for cellular metabolism
- part of the NAD+/NADH redox system

It is not:

- a peptide
- a hormone
- a stimulant
- a GLP-1 agonist

Primary Research Focus

Best Established

- cellular energy metabolism
- redox reactions
- NAD-dependent enzyme biology
- cellular metabolism

Direct Human Administration Research

- IV metabolism
- IV tolerability
- early IM / SC tolerability

Common Wellness Claims Not Established

- energy enhancement
- anti-aging
- cognitive enhancement
- fatigue treatment
- addiction detoxification
- weight loss
- athletic performance

Common Research Dose

Human Direct NAD+ Research

2019 IV Metabolism Study

3 µmol/min for 6 hours

2026 IV Study

500 mg IV

on:

4 consecutive days

2026 Preliminary Multi-Route Safety Analysis

100 mg NAD+

by:

- IM
- SC
- IV-push

over:

3 consecutive days

in very small groups.

Important

These are:

research exposures

not:

approved wellness doses

Research Community

PeptideFox

IM

50–200 mg

usually:

2–3x/week

SC

50–100 mg per dose

PeptideDosages

50 → 75 → 100 mg SC daily

for approximately:

8–16 weeks

Community Protocol Comparison

Source	Dose	Route	Frequency	Evidence
2019 human study	3 µmol/min	IV	6-hour infusion	Human metabolism research
2026 IV study	500 mg	IV	Daily ×4	Human tolerability research
2026 preliminary analysis	100 mg	IM / SC / IV-push	Daily ×3	Preliminary human safety
PeptideFox	50–200 mg	IM	2–3x/week	Community
PeptideFox	50–100 mg	SC	Variable	Community

Source	Dose	Route	Frequency	Evidence
PeptideDosages	50→75→100 mg	SC	Daily	Community

Strongest Conclusion

There is:

no validated universal NAD+ injection protocol.

Expected Results Timeline, Expanded

Direct Human Research

Minutes to Hours

Administered NAD+ enters metabolism quickly.

That does **not** automatically mean an immediate health benefit.

Several Days

Short multi-day studies mainly provide:

tolerability information

not evidence of:

- anti-aging
- sustained energy improvement
- better cognition

Weeks to Months

There is no strong controlled direct-NAD+ study establishing a predictable wellness-results timeline.

Community-Reported

Same Day

Some people report:

- alertness
- energy
- general wellness

after IV or injected NAD+.

Several Weeks

Community protocols describe:

- steadier energy

- recovery
- mental clarity

during repeated treatment.

Evidence Warning

These remain:

community reports

Do not call them:

> **Expected clinical effects**

Reported Side Effects

Human IV Research

Reported infusion effects include:

- nausea
- abdominal discomfort
- other GI symptoms
- increased heart rate
- chest pressure

IM / SC

Controlled human evidence is still extremely limited.

A preliminary 100 mg analysis did not report severe unexpected treatment-related events.

But the route groups were:

very small

Local discomfort is also commonly reported in community use.

Contaminated or Poor-Quality Injectable Products

FDA has received reports involving:

- severe chills
- shaking
- vomiting
- fatigue

after compounded NAD+ injections.

Some people required medical treatment.

FDA said the reactions were consistent with:

excessive endotoxin exposure

Important

Keep these separate:

Side effects caused by NAD+ itself

versus:

reactions caused by contaminated or poorly manufactured injectable products

---

Contraindications & Cautions

Formal FDA Contraindications

None established

There is no FDA-approved NAD+ wellness injection label.

Important Cautions

Cardiovascular Symptoms During IV Use

Chest pressure and increased heart rate have occurred during higher-dose IV NAD+ administration.

Product Quality

Sterility and endotoxin control are critical.

Pregnancy / Breastfeeding

Direct injection safety is not adequately studied.

Long-Term IM / SC Use

Controlled long-term safety is not established.

Formulation / Stability / Sterility

No Universal FDA-Approved NAD+ Injection Exists

There is no approved reference formulation allowing one universal rule for:

- concentration
- diluent
- sterility period
- expiration after opening
- reconstitution method

FDA Product-Quality Warning

FDA warned compounders against using food-grade NAD+ ingredients for sterile injection products without appropriate processing because of:

- contamination
- endotoxin
- sterility

concerns.

This should remain prominent.

PeptideDosages Community Vial

PeptideDosages describes a:

500 mg lyophilized vial

mixed with bacteriostatic water.

Its own community protocol lists approximately:

14 days refrigerated after mixing

Important

That does **not** mean:

> all NAD+ lasts 14 days after reconstitution.

Different products can have different stability.

Simple Rule

Use storage instructions for the exact product or pharmacy formulation.

Regulatory Status

United States

FDA-Approved NAD+ Wellness Injection?

No

Compounded NAD+

Compounded preparations exist.

They are **not equivalent to an FDA-approved NAD+ drug**.

FDA has specifically raised concerns about:

- ingredient quality
- sterile production

- endotoxin contamination
- regulatory eligibility

for some compounded NAD+ products.

2026 503B Enforcement

A January 2026 FDA warning letter stated that NAD+ did:

not appear on the 503B Bulks List

and that products compounded from bulk NAD+ by the cited outsourcing facility did not qualify for the relevant exemptions.

Production Wording

> **NAD+ is not FDA approved as an injectable wellness or anti-aging drug. Compounded products exist, but FDA has raised both regulatory and sterile-product-quality concerns.**

What Studies Have Found

2019 Human IV Metabolism Study

NAD+ was infused for:

6 hours

at:

3 µmol/min

Researchers found that blood NAD+ did not simply rise immediately.

Instead, infused NAD+ was rapidly:

- removed from plasma
- metabolized
- converted into related compounds

What This Supports

- administered NAD+ enters human metabolism
- extracellular metabolism is important
- IV delivery does not mean intact NAD+ simply stays in circulation

What It Does Not Support

- anti-aging
- more energy
- fatigue treatment
- cognitive enhancement

2026 IV NAD+ Research

Participants received:

500 mg IV NAD+

for:

4 consecutive days

The NAD+ group experienced more:

- GI symptoms
- heart-rate increases
- chest pressure

than the IV NR group.

What This Supports

Infusion rate and formulation can strongly affect tolerability.

What It Does Not Support

500 mg as a universally optimal IV NAD+ dose

2026 IM / SC / IV-Push Preliminary Safety Analysis

A 2026 preliminary analysis reported:

100 mg NAD+

given by:

- IM
- SC
- IV-push

over:

three consecutive days

What This Supports

We now have:

early controlled human exposure

to IM and SC NAD+.

What It Does Not Support

- chronic 100 mg daily treatment
- 200 mg IM protocols
- 8–16 week use
- anti-aging efficacy
- fatigue treatment
- increased mitochondrial function

The groups were small and the analysis was focused mainly on:

short-term safety and tolerability

Corrected ClinicalTrials.gov Section

NCT06919328

The registered study is primarily described as research evaluating injectable:

nicotinamide riboside

including absorption and tolerability across routes such as:

- IM
- SC
- IV-push

This study record is useful for documenting the broader human injection-research framework.

It should **not** be used as the direct source for the specific NAD+ findings involving:

- 100 mg NAD+
- IM NAD+
- SC NAD+
- IV-push NAD+
- three consecutive days of NAD+ administration

Those findings belong to the later:

2026 preliminary safety analysis

Research Sources

Use **clean canonical URLs in production**.

1. Grant et al. 2019: Human IV NAD+ Metabolism

Evidence type: Human metabolism research

Supports:

- direct IV NAD+ administration
- 6-hour infusion
- human NAD+ metabolism
- rapid plasma processing
- NAD-related metabolite changes

`https://pubmed.ncbi.nlm.nih.gov/31572171/`

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2. Reyna et al. 2026: IV NAD+ vs IV Nicotinamide Riboside

Evidence type: Human tolerability research

Supports:

- 500 mg NAD+ IV
- four consecutive treatment days
- GI symptoms
- increased heart rate
- chest pressure
- slower NAD+ infusion due to tolerability

`https://pubmed.ncbi.nlm.nih.gov/41704678/`

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3. ClinicalTrials.gov NCT06919328

Evidence type: Registered human injection study

Supports:

- injection-route research involving nicotinamide riboside
- IM administration research
- SC administration research
- IV-push administration research
- human injection tolerability framework

It should **not** be used as the direct source for the 100 mg NAD+ IM/SC/IV-push findings.

`https://clinicaltrials.gov/study/NCT06919328`

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4. Nkrumah-Elie et al. 2026 Preliminary Safety Analysis

Evidence type: Human preliminary safety analysis / preprint

Supports:

- **100 mg NAD+**
- **IM NAD+ exposure**
- **SC NAD+ exposure**
- **IV-push NAD+ exposure**
- administration over **three consecutive days**
- preliminary short-term tolerability findings

Important

This is a:

preprint

not a peer-reviewed efficacy trial.

It supports wording such as:

> **early controlled human exposure**

or:

> **preliminary human tolerability evidence**

not:

clinically validated NAD+ injection protocol

https://www.medrxiv.org/content/10.64898/2026.04.28.26352007v1

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5. FDA: Sterile NAD+ Compounding Warning

Evidence type: FDA product-quality and safety information

Supports:

- food-grade NAD+ concerns
- sterile compounding concerns
- endotoxin risk
- severe chills
- shaking
- vomiting
- fatigue reports

https://www.fda.gov/drugs/human-drug-compounding/fda-reminds-compounders-use-ingredients-suitable-sterile-compounding

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6. FDA 2026 GenoGenix Warning Letter

Evidence type: FDA regulatory enforcement source

Supports:

- 503B regulatory concerns
- bulk NAD+ compounding issue
- endotoxin/product-quality concerns involving NAD+ products

https://www.fda.gov/inspections-compliance-enforcement-and-criminal-investigations/warning-letters/genogenix-llc-718739-01202026

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7. PeptideFox: NAD+ Guide

Evidence type: Community / practitioner protocols

Useful for documenting:

- IM 50–200 mg
- SC 50–100 mg
- 2–3x-weekly IM use
- community IV protocols
- route tolerability
- loading-series practices

https://peptidefox.com/content/nad-guide

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8. PeptideDosages: NAD+ 500 mg Vial

Evidence type: Community protocol

Useful for documenting:

- 50 → 75 → 100 mg SC titration
- daily use
- 8–16 week framework
- community reconstitution
- community storage guidance

`https://peptidedosages.com/single-peptide-dosages/nad-500-mg-10ml-vial-dosage-protocol/`

Final Takeaway

NAD+ is essential for human cellular metabolism. That part is certain. The benefits of injecting extra NAD+ are much less certain.

The strongest science supports NAD+'s role in:

- energy metabolism
- redox chemistry
- NAD-dependent cellular signaling

Direct human administration research shows that NAD+:

- enters and is processed by human metabolism
- can cause significant infusion-related discomfort at higher IV doses
- now has preliminary controlled human IM and SC exposure data at **100 mg**

But we still do not have established clinical evidence supporting one universal:

50–100 mg daily SC

or:

50–200 mg IM 2–3 times weekly

wellness protocol.

Those remain:

community / practitioner protocols

There is also no evidence-based:

- receptor reset
- mandatory cycle
- morning requirement
- fasted requirement
- pre-workout requirement

The most important idea for the page should remain:

> **NAD+ being essential for cellular energy does not prove that injecting more NAD+ gives a healthy person more energy.**

And the important 2026 update is:

IM and SC NAD+ are no longer supported only by anecdotes. A small preliminary analysis has now reported 100 mg NAD+ given by both routes. But that provides early tolerability evidence, not proof of anti-aging, energy, fatigue or metabolic benefits.

Pemvidutide

GLP-1 / Glucagon Dual Receptor Agonist • MASH Research • Weight-Loss Research

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At a Glance

What is Pemvidutide?

Pemvidutide is an **investigational peptide drug** that activates two hormone receptors:

- the **GLP-1 receptor**
- the **glucagon receptor**

Altimmune describes it as a roughly balanced **1:1 GLP-1 / glucagon dual receptor agonist**. It is currently being developed primarily for MASH, alcohol use disorder and alcohol-associated liver disease.

GLP-1 activity

GLP-1 signaling can help:

- reduce appetite
- increase fullness
- lower food intake
- regulate blood sugar

Glucagon activity

Glucagon-receptor signaling has important effects involving:

- liver metabolism
- fat oxidation
- liver fat
- lipid handling
- energy use

Simple version

Pemvidutide is designed to combine:

less food intake

with:

direct metabolic effects involving the liver.

How well studied is it?

Well studied for an investigational drug, but it is still not approved

Pemvidutide now has:

- Phase 1 human research
- a completed Phase 2 obesity program
- human MASLD studies
- a completed Phase 2b MASH trial
- a completed Phase 2 alcohol-use-disorder trial with topline results
- an ongoing Phase 2 alcohol-associated liver disease program
- an active Phase 3 MASH program

Altimmune initiated the global **PERFORMA Phase 3 MASH trial on August 3, 2026**.

Evidence level

FDA-approved human evidence: None
Human clinical research: Strong for an investigational drug
MASH evidence: Strong Phase 2b evidence; Phase 3 underway
Weight-loss evidence: Strong Phase 2 evidence
Liver-fat evidence: Strong human evidence
Alcohol-use-disorder evidence: Positive Phase 2 topline evidence
Long-term approved-product safety: Not established
Community protocols: Secondary to the clinical-trial evidence

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What may it help with?

Weight Loss

The Phase 2 MOMENTUM obesity program studied pemvidutide by **once-weekly subcutaneous injection** at doses including:

- 1.2 mg
- 1.8 mg
- 2.4 mg.

At 48 weeks, sponsor-reported mean weight loss was approximately:

1.2 mg: 10.3%

1.8 mg: 11.2%

2.4 mg: 15.6%

Placebo: 2.2%.

More than 30% of participants receiving 2.4 mg lost at least 20% of baseline body weight in the reported MOMENTUM results.

Important

This is meaningful human evidence.

But pemvidutide is still:

investigational

and is not an FDA-approved obesity treatment. Altimmune continues to describe it as an investigational product candidate.

MASH

Pemvidutide has substantial human MASH evidence.

The IMPACT Phase 2b trial was designed to assess:

- MASH resolution
- fibrosis outcomes

with pemvidutide versus placebo.

Altimmune reported that the 24-week data demonstrated statistically significant **MASH resolution without worsening of fibrosis**. The company later reported continued improvements in noninvasive fibrosis-related measures through 48 weeks.

Important

Do **not** simplify this to:

> Pemvidutide has proven it reverses fibrosis.

That overstates the evidence.

Better wording

> **Pemvidutide has shown significant MASH activity and encouraging fibrosis-related signals in Phase 2b research. Phase 3 is now underway to determine whether those findings translate into a registrational treatment benefit.**

Liver Fat Reduction

Human MASLD research has shown large reductions in liver fat.

At 24 weeks, relative liver-fat reductions were approximately:

1.2 mg: 56.3%

1.8 mg: 75.2%

2.4 mg: 76.4%

Placebo: 14.0%.

That is one of the strongest and most consistent findings in the pemvidutide program.

Alcohol Use Disorder

This is a major 2026 update.

RECLAIM was a Phase 2 trial of approximately 100 adults with alcohol use disorder receiving:

2.4 mg pemvidutide once weekly

or placebo for:

24 weeks.

Altimmune announced positive topline results on **July 28, 2026**, reporting a statistically significant reduction in heavy-drinking days, along with improvements in important secondary drinking outcomes.

Important

These are currently:

sponsor-reported Phase 2 topline results

rather than a fully peer-reviewed efficacy paper.

Evidence classification

Promising human interventional evidence

Not:

approved AUD treatment

What has it NOT been proven to help with?

Pemvidutide has not been established as an approved treatment for:

- obesity
- ordinary weight loss
- type 2 diabetes
- bodybuilding
- muscle growth
- athletic performance
- spot fat loss
- GLP-1-related fatigue
- alcohol-use disorder
- alcohol-associated liver disease
- general "liver detox"
- longevity
- anti-aging

Clinical research may support some of these disease areas in the future.

But:

clinical development is not the same as regulatory approval.

Dose at a Glance

FDA-Approved Dosing

There is none

Pemvidutide is not FDA approved.

There is no FDA-approved:

- starting dose
- maintenance dose
- titration schedule
- commercial injection pen
- commercial vial
- prescribing label

Altimmune continues to describe pemvidutide as investigational.

Human Clinical Research Dosing

MOMENTUM Obesity Trial

Major studied doses included:

1.2 mg once weekly

1.8 mg once weekly

2.4 mg once weekly.

Important

These were **trial arms**.

They should not automatically be converted into:

> 1.2 mg starting dose → 1.8 mg maintenance → 2.4 mg advanced dose

as though that were an approved dosing system.

IMPACT MASH Trial

The Phase 2b IMPACT study used:

1.2 mg once weekly

and:

1.8 mg once weekly

against placebo.

RECLAIM AUD Trial

RECLAIM used:

2.4 mg once weekly

for:

24 weeks.

Important

The dose was studied specifically in that Phase 2 AUD protocol.

It does not establish 2.4 mg as a universal public pemvidutide dose.

Research-Community Dosing

Community pages often reuse genuine trial doses such as:

- 1.2 mg
- 1.8 mg
- 2.4 mg

and turn them into a generic dose ladder.

The problem

The numbers themselves are real human research doses.

The labels:

- "starting"
- "standard"
- "advanced"

are usually **community reinterpretations**, not approved clinical categories.

Correct evidence classification

1.2 mg: Human trial dose

1.8 mg: Human trial dose

2.4 mg: Human trial dose

1.2 → 1.8 → 2.4 mg universal ladder: Not established

Frequency at a Glance

Human Clinical Research

Once weekly

This is the clearest dosing feature across the pemvidutide program.

The obesity, liver and AUD programs use once-weekly SC administration.

Twice weekly?

Not established

Every 3–4 days?

Not established

Daily?

Not established

5 days on / 2 days off?

Not established

Best-supported frequency

Once weekly

Timing at a Glance

There is no human evidence showing pemvidutide needs to be taken:

- in the morning
- at night
- fasted
- with food
- before exercise
- after exercise

The important studied variable is:

weekly administration

not a particular clock time.

Evidence verdict

Morning required: No
Night required: No
Fasted required: No
With food required: No
Pre-workout required: No
Post-workout required: No

Duration / Cycle at a Glance

Human Clinical Research

Pemvidutide has been studied over periods including:

24 weeks

and:

48 weeks

and the Phase 3 MASH program is designed as longer-term disease treatment.

Does pemvidutide need cycling?

No receptor-reset cycle has been established.

There is no evidence-based requirement for:

- 8 weeks on / 4 weeks off
- 12-week cycles
- 5 days on / 2 days off
- monthly washouts
- GLP-1 receptor resets
- glucagon receptor resets

Expected Results Timeline

Weight-Loss Research

Early weeks

Appetite and gastrointestinal effects may occur relatively early.

But clinically meaningful weight loss is evaluated over:

months

rather than days.

Around 24 Weeks

Meaningful body-weight changes are already measurable in liver/metabolic trials by this point.

Human MASLD research also showed major liver-fat reductions by **24 weeks**.

Around 48 Weeks

MOMENTUM reported mean weight losses of approximately:

1.2 mg: 10.3%

1.8 mg: 11.2%

2.4 mg: 15.6%

at 48 weeks.

Clinical takeaway

Think in months, not days.

Liver Results Timeline

Around 12–24 Weeks

Substantial liver-fat changes have been observed during this period.

At 24 weeks, relative liver-fat reductions reached approximately **75–76%** in the 1.8 and 2.4 mg groups in the MASLD extension study.

MASH Histology

The IMPACT Phase 2b program formally evaluated biopsy-based MASH outcomes and generated positive 24-week results, followed by additional 48-week data.

Community-Reported Timeline

Community material often describes:

- early appetite suppression
- early GI effects
- progressive weight loss over weeks and months

But many of the percentages quoted online are simply copied from clinical trials.

Important

Separate:

actual trial timelines

from:

community anecdotes

rather than combining the two.

Best top-card wording

Clinical: measurable weight and liver changes develop over months, with important trial endpoints assessed around 24 and 48 weeks.

Community-reported: appetite and GI changes are often described earlier, but community timelines should not be mistaken for controlled trial outcomes.

Safety at a Glance

Human Trial Safety

The most common adverse effects in the clinical program have been gastrointestinal, including:

- nausea
- vomiting
- diarrhea
- constipation

These effects have generally been described as mild to moderate in major trials.

MASH Program

Altimmune reported a favorable overall tolerability profile through 48 weeks in IMPACT, including low treatment-related discontinuation.

Important

This is meaningful trial safety evidence.

But it is not equivalent to:

years of postmarketing safety data from an approved drug.

RECLAIM AUD Trial

RECLAIM also reported gastrointestinal effects such as nausea, vomiting, diarrhea and constipation in the 2.4 mg group. The topline results remain sponsor-reported pending fuller publication.

Any isolated serious event from that trial should be described as an **individual trial event**, not automatically upgraded into a common pemvidutide adverse effect.

Formal Contraindications

None established through an FDA-approved product label

Pemvidutide is not approved.

There is therefore no FDA-approved pemvidutide contraindication section.

Do not copy contraindications from:

- Wegovy
- Saxenda
- Zepbound
- other GLP-1 drugs

and present them as official pemvidutide contraindications.

Important Cautions

Pregnancy / Breastfeeding

Human safety has not been established.

Classification: Important investigational-use caution.

Significant Gastrointestinal Disease

Pemvidutide causes GI adverse effects in human trials.

Classification: Clinically relevant caution, not an FDA-established contraindication.

Other GLP-1 Drugs

Combining pemvidutide with another GLP-1 receptor agonist creates major pathway overlap.

Classification: Unvalidated combination.

Pancreatitis

A history of pancreatitis may be clinically relevant when evaluating an incretin-based investigational drug.

But pemvidutide does **not** currently have an approved label defining pancreatitis history as an official contraindication.

Classification

Clinical caution, not formal pemvidutide contraindication

MTC / MEN2

Do not automatically import the medullary-thyroid-carcinoma / MEN2 contraindications from approved GLP-1 drugs.

Pemvidutide currently has no FDA-approved label establishing those as formal contraindications.

Verdict

Do not present as official unless a future approved label specifically includes them.

Regulatory Status at a Glance

FDA Approved?

No

Pemvidutide remains investigational.

Fast Track

Pemvidutide has received FDA Fast Track designation for:

MASH

and:

alcohol use disorder.

Breakthrough Therapy Designation

MASH

Yes

FDA granted Breakthrough Therapy Designation for MASH on **January 5, 2026**.

Important

Breakthrough Therapy Designation does **not** mean FDA approval.

It is intended to expedite development and review when preliminary clinical evidence suggests substantial potential benefit.

Phase 3

Yes

Altimmune initiated the global **PERFORMA Phase 3 MASH trial on August 3, 2026**.

Production wording

> **Pemvidutide is now a Phase 3 investigational drug for MASH, but it remains unapproved.**

Bottom Line

Pemvidutide has much stronger human evidence than most compounds sold as "research peptides," but it remains an investigational drug.

The strongest dosing evidence supports:

once-weekly subcutaneous administration.

Major human trial doses include:

1.2 mg

1.8 mg

and:

2.4 mg

but those should not automatically be turned into a universal:

starting → standard → advanced

dose system.

Pemvidutide has meaningful human evidence for:

- weight loss
- liver-fat reduction
- MASH improvement

and now sponsor-reported Phase 2 evidence for:

- alcohol use disorder.

It has also now entered:

Phase 3 MASH development.

Protocol Logic Check

Quick Logic Summary

Protocol Question	Verdict
GLP-1 receptor agonism?	**Evidence-supported**
Glucagon receptor agonism?	**Evidence-supported**
Balanced dual agonist?	**Yes**
Once-weekly SC dosing?	**Strongly supported**
1.2 mg studied in humans?	**Yes**
1.8 mg studied in humans?	**Yes**
2.4 mg studied in humans?	**Yes**
1.2 → 1.8 → 2.4 mg universal titration?	**No**
1.8 mg universal maintenance dose?	**No**
2.4 mg universal "advanced dose"?	**No**
Morning dosing better?	**Not shown**
Fasted dosing required?	**No**
Pre-workout dosing useful?	**No demonstrated reason**
Twice-weekly dosing?	**Not established**
8–12 week cycle?	**No demonstrated rationale**
Receptor reset?	**No established requirement**
Stack with semaglutide?	**Major pathway overlap**
Generic research-vial stability?	**Cannot be assumed**
FDA approved?	**No**
Phase 3 underway?	**Yes**

What Does Pemvidutide Target?

Pemvidutide activates:

GLP-1 receptors

and:

glucagon receptors.

Simple version

The two pathways contribute different effects.

GLP-1

Primarily helps:

reduce appetite and food intake

Glucagon

Has important effects involving:

the liver and energy metabolism

Together, the drug is intended to affect both:

energy intake

and:

how stored fuel is handled metabolically.

Verdict

Well established mechanism

Does Glucagon Just Raise Blood Sugar?

No.

Glucagon can raise blood glucose, but glucagon-receptor signaling also affects:

- liver metabolism
- fatty-acid oxidation
- lipid handling
- energy expenditure

Altimmune's development rationale specifically emphasizes direct liver effects from glucagon-receptor activation alongside appetite and weight effects mediated through GLP-1.

Human Half-Life

Pemvidutide is pharmacologically long acting enough for:

once-weekly administration

which is directly supported by the human clinical-development program.

Important

The page does not need to convert a quoted half-life into an amateur dosing formula.

Better rule

> **Use the human clinical schedule rather than reverse-engineering frequency from half-life alone.**

Does Once-Weekly Dosing Make Sense?

Yes.

This is one of the most secure conclusions for pemvidutide.

Major clinical programs use:

once-weekly SC administration.

Verdict

Strongly evidence-supported

Does Twice-Weekly Dosing Make Sense?

There is no controlled evidence showing that splitting the weekly dose improves:

- weight loss
- MASH outcomes
- liver-fat reduction
- tolerability

Verdict

Not established

Does Daily Dosing Make Sense?

No major clinical pemvidutide program uses daily administration.

Verdict

Not established

Morning or Evening?

There is no demonstrated superiority for either.

Verdict

Preference, not evidence-based requirement

Does It Need to Be Taken Fasted?

No evidence shows fasting is required.

Verdict

Not established

Pre-Workout Timing

Pemvidutide is a long-acting weekly metabolic drug.

There is no evidence that taking it near a workout improves efficacy.

Verdict

No demonstrated advantage

Dose Escalation / Titration

This needs careful wording.

The human program has used more than one dosing and titration design.

That means:

titration may be useful for tolerability

but:

there is no finalized universal pemvidutide titration system.

Verdict

Study-specific

Is 1.2 mg the Starting Dose?

Not in the same sense that an approved GLP-1 product has a labeled starting dose.

1.2 mg is a:

human clinical trial dose

not an FDA-established universal starting dose.

Is 1.8 mg the Standard Dose?

No.

It is a well-studied clinical dose, especially in liver research.

But pemvidutide does not have an FDA-approved:

maintenance dose.

Is 2.4 mg the "Advanced Dose"?

No official category exists.

2.4 mg is a genuine human trial dose used in obesity and AUD research.

Calling it:

> advanced dose

is community language.

Verdict

Human trial dose, not official dose tier

Does It Need an 8–12 Week Cycle?

No.

Clinical studies last:

- 24 weeks
- 48 weeks
- and now longer registrational development

rather than short peptide cycles.

Verdict

No evidence-based short-cycle requirement

Does It Need a Receptor Reset?

No scheduled GLP-1 or glucagon-receptor reset has been demonstrated.

Verdict

No established requirement

Does It Need a Washout?

There is no established:

- 2-week reset
- 4-week reset
- monthly receptor recovery break

as part of chronic pemvidutide treatment.

Verdict

No demonstrated requirement

Does the Route Match the Research?

Major Human Trials

Subcutaneous

Frequency

Once weekly

Verdict

Strong match to the human evidence

Stack / Pairing Logic

Pemvidutide + Semaglutide

Both activate GLP-1 receptors.

Verdict: Major pathway overlap; no validated combination.

Pemvidutide + Tirzepatide

Tirzepatide activates:

- GLP-1
- GIP

Pemvidutide activates:

- GLP-1
- glucagon

Verdict: Major GLP-1 overlap; combination unvalidated.

Pemvidutide + Retatrutide

Retatrutide activates:

- GLP-1
- GIP
- glucagon

That overlaps with **both** main pemvidutide targets.

Verdict: Very high pathway redundancy; no validated combination.

Pemvidutide + Cagrilintide

Cagrilintide primarily acts through amylin-related pathways.

Verdict: Less direct receptor overlap, but no validated pemvidutide combination protocol.

Pemvidutide + Metformin

Different primary mechanisms.

Verdict: Mechanistically nonredundant, but specific use should be based on actual clinical evidence rather than assumptions.

Formulation / Stability / Sterility Logic

There is currently:

no FDA-approved commercial pemvidutide formulation.

Therefore there is no universal approved reference product defining:

- vial strength
- concentration
- diluent
- bacteriostatic-water use
- post-reconstitution storage
- 14-day use period
- 28-day use period

Verdict

Use exact-product evidence only.

A research vial sold online is not automatically equivalent to the investigational clinical-trial product.

Important Research Note

Three different pemvidutide contexts are often mixed together.

1. Clinical-Trial Pemvidutide

This is Altimmune's controlled investigational drug.

Clinical trials have:

- controlled manufacturing
- defined formulation
- defined route
- defined dose
- clinical monitoring

---

2. Human Clinical-Trial Doses

These include:

- 1.2 mg
- 1.8 mg
- 2.4 mg

These are genuine human research doses.

But they are:

trial doses

not:

FDA-approved public dosing instructions.

---

3. Community Research Pemvidutide

Online research products may differ in:

- manufacturing
- formulation
- excipients
- sterility
- purity
- concentration
- handling

A vial labeled:

Pemvidutide 10 mg

cannot automatically be assumed to match the investigational product used in Altimmune trials.

Research at a Glance

Approved-Label Human Evidence

None

Pemvidutide is not FDA approved.

Human Clinical Research

Strong for an investigational drug

There is meaningful Phase 1, Phase 2 and Phase 2b evidence, with Phase 3 now underway.

Weight-Loss Evidence

Strong Phase 2 evidence

Mean weight loss reached approximately:

15.6% at 48 weeks

in the 2.4 mg MOMENTUM arm.

Liver Evidence

Strong investigational evidence

Human research demonstrates:

- large liver-fat reductions
- MASH-resolution activity
- improvements in liver-related biomarkers.

Alcohol-Use-Disorder Evidence

Promising Phase 2 topline evidence

RECLAIM reported a significant reduction in heavy-drinking days.

Full peer-reviewed efficacy publication is still needed.

Overview

Pemvidutide is a long-acting investigational:

GLP-1 / glucagon dual receptor agonist

being developed primarily for serious metabolic and liver diseases.

The GLP-1 component helps reduce:

appetite and food intake

while glucagon-receptor activity is intended to provide additional:

liver and metabolic effects.

Human studies have shown:

- substantial weight loss
- very large liver-fat reductions
- significant MASH activity

and more recently:

- promising Phase 2 AUD results.

It nevertheless remains:

investigational.

Evidence Strength

Overall Evidence Rating

Strong human investigational evidence

Strong evidence for

- dual GLP-1 / glucagon receptor activity
- once-weekly SC administration
- weight loss
- liver-fat reduction

- MASH activity
- GI adverse effects

Promising human evidence for

- alcohol-use-disorder outcomes
- fibrosis-related improvements
- broader cardiometabolic effects

Not established

- FDA-approved obesity treatment
- FDA-approved MASH treatment
- FDA-approved AUD treatment
- standard commercial dose
- universal titration schedule
- community-vial equivalence
- bodybuilding
- athletic enhancement
- anti-aging
- receptor-reset cycling
- stacking with other incretin agonists

Category

GLP-1 / Glucagon Dual Agonists • Metabolic Health • Liver Disease Research

---

Compound

Pemvidutide

Development name:

ALT-801

Compound Type

Long-acting peptide GLP-1 / glucagon dual receptor agonist

Pemvidutide is:

- peptide-based
- a GLP-1 receptor agonist
- a glucagon receptor agonist
- designed for weekly administration

It is not:

- a GIP agonist
- insulin
- a stimulant
- an approved medication

Primary Research Focus

Most Advanced

MASH

with Phase 3 development now underway.

Human Research Also Includes

- obesity
- weight loss
- MASLD
- alcohol use disorder
- alcohol-associated liver disease.

Not Established

- bodybuilding
- athletic performance
- anti-aging
- general wellness

Common Research Dose

MOMENTUM Obesity

1.2 mg once weekly

1.8 mg once weekly

2.4 mg once weekly.

IMPACT MASH

1.2 mg once weekly

1.8 mg once weekly.

RECLAIM AUD

2.4 mg once weekly.

Research Duration / Cycle Length

Human Trials

Important treatment periods include:

24 weeks

and:

48 weeks

with longer-term Phase 3 development now underway.

Evidence assessment

Pemvidutide is being developed as:

long-term disease therapy

not a short peptide cycle.

How Often It Is Used / Frequency

Human Clinical Research

Once weekly SC

Evidence assessment

Directly supported across the major human programs.

Possible Benefits

Weight Loss

Strong Phase 2 human evidence.

Liver Fat Reduction

Strong human clinical evidence.

MASH Improvement

Phase 2b human evidence supports meaningful MASH-resolution activity, with Phase 3 now underway.

Alcohol Use Disorder

Positive sponsor-reported Phase 2 topline evidence.

Important

All remain:

investigational treatment indications

unless and until regulatory approval occurs.

What It Does Not Help With, Based on Current Evidence

Do not present pemvidutide as proven to:

- build muscle
- improve athletic performance
- selectively burn abdominal fat
- increase energy
- repair injuries
- treat type 2 diabetes as an approved therapy
- treat AUD as an approved medication
- treat ALD as an approved medication
- prevent weight regain after stopping
- provide identical results at every dose

Commonly Paired With

Metformin

Different primary mechanism.

Classification: Mechanistically nonredundant; combination claims require actual evidence.

Semaglutide

Significant GLP-1 overlap.

Classification: Major pathway overlap; no validated combination.

Tirzepatide

Shared GLP-1 signaling.

Classification: Major pathway overlap; no validated combination.

Retatrutide

Shared GLP-1 and glucagon signaling.

Classification: Highly redundant.

Cagrilintide

Different main pathway.

Classification: Less direct overlap, but no validated combination protocol.

Dosage Information

Clinical-Trial Pemvidutide

Route: Subcutaneous

Frequency: Once weekly

Major studied doses

- 1.2 mg
- 1.8 mg
- 2.4 mg.

Important

There is still no:

FDA-approved public pemvidutide dosing schedule.

Community Research Protocol

Community pages may describe:

"Starting"

1.2 mg weekly

"Standard"

1.8 mg weekly

"Advanced"

2.4 mg weekly

Evidence check

1.2 mg: Real human trial dose

1.8 mg: Real human trial dose

2.4 mg: Real human trial dose

Starting / standard / advanced labels: Community reinterpretation

Correct production wording

Use:

Human Trial Dose Examples

rather than:

Standard Pemvidutide Protocol

Community Protocol Comparison

Source	Dose	Route	Frequency	Evidence
MOMENTUM	1.2 mg	SC	Weekly	Phase 2 human
MOMENTUM	1.8 mg	SC	Weekly	Phase 2 human
MOMENTUM	2.4 mg	SC	Weekly	Phase 2 human
IMPACT	1.2 / 1.8 mg	SC	Weekly	Phase 2b human
RECLAIM	2.4 mg	SC	Weekly	Phase 2 human
Community ladder	1.2 → 1.8 → 2.4 mg	SC	Weekly	Trial arms repackaged as a protocol

Strongest agreement

Once-weekly SC administration

Biggest mistake to avoid

Turning:

separate clinical-trial doses

into:

one universal escalation ladder

Expected Results Timeline, Expanded

Weight-Loss Research

Around 24 weeks

Clinically meaningful weight loss and liver-related changes are measurable by this period.

Around 48 weeks

MOMENTUM reported:

1.2 mg: 10.3%

1.8 mg: 11.2%

2.4 mg: 15.6%

mean weight loss.

Liver Research

12–24 weeks

Large liver-fat reductions can occur during this period.

24 weeks

Important biopsy-based MASH outcomes were assessed in the IMPACT program.

48 weeks

Longer-term metabolic and fibrosis-related outcomes were reported.

Reported Side Effects

Common Human-Trial Effects

Most commonly gastrointestinal:

- nausea
- vomiting
- diarrhea
- constipation.

Most have been described as mild to moderate.

Serious Events

Serious drug-related events have not dominated the clinical program.

However:

pemvidutide still lacks the long-term postmarketing safety history of an approved drug.

Contraindications & Cautions

Formal Contraindications

None established in an FDA-approved label

Because no FDA-approved pemvidutide label exists.

Important Cautions

Pregnancy / Breastfeeding

Safety not established.

Significant GI Disease

GI adverse effects occur in clinical trials.

Other GLP-1 Drugs

Major pathway overlap.

Pancreatitis History

Potentially relevant clinical caution, but not an official pemvidutide contraindication.

Thyroid Cancer / MEN2

Do not copy contraindications from other GLP-1 product labels and call them official pemvidutide contraindications.

Regulatory Status

United States

FDA approved?

No

Fast Track

MASH: Yes
AUD: Yes

Breakthrough Therapy

MASH: Yes, granted January 5, 2026.

Phase 3

Underway

PERFORMA began in August 2026.

What Studies Have Found

MOMENTUM Obesity

At 48 weeks:

1.2 mg: 10.3% mean weight loss

1.8 mg: 11.2%

2.4 mg: 15.6%

Placebo: 2.2%.

Supports

- weekly SC dosing
- substantial weight loss
- genuine human dose-response research

Does not support

- an FDA-approved obesity dose
- universal 1.2 → 1.8 → 2.4 mg escalation

MASLD Research

At 24 weeks, relative liver-fat reductions were approximately:

1.2 mg: 56.3%

1.8 mg: 75.2%

2.4 mg: 76.4%

Placebo: 14.0%.

Supports

Strong human evidence for liver-fat reduction.

Does not support

Calling pemvidutide:

a liver detox drug

IMPACT Phase 2b MASH

Phase 2b data showed significant:

MASH resolution without worsening fibrosis

and additional fibrosis-related improvements over longer follow-up.

Supports

Strong enough MASH activity to justify registrational Phase 3 development.

Does not support

Claiming:

> Pemvidutide has already proven fibrosis reversal in everyone.

RECLAIM Phase 2 AUD

RECLAIM used:

2.4 mg once weekly

for:

24 weeks

in approximately 100 participants.

Altimmune reported significant improvement in the primary heavy-drinking-day endpoint in July 2026.

Supports

Promising human Phase 2 evidence for AUD.

Does not support

Calling pemvidutide an approved AUD medicine.

PERFORMA Phase 3

Altimmune initiated the global:

PERFORMA Phase 3 MASH program

on August 3, 2026.

Why this matters

Pemvidutide should now be described as:

a late-stage investigational drug

rather than simply:

an early experimental peptide.

Formulation / Stability / Sterility

No FDA-Approved Commercial Product Exists

There is no approved pemvidutide product providing one universal:

- concentration
- diluent
- reconstitution method
- refrigerated storage period
- 14-day use period
- 28-day use period

Research vials

Community products may provide their own instructions for:

- bacteriostatic water
- refrigeration
- post-mixing use periods

Those are:

product/community handling instructions

not:

validated clinical pemvidutide stability data.

Simple rule

Use handling and stability information for the exact formulation.

Research Sources

Use **clean canonical URLs in production**.

1. ClinicalTrials.gov: MOMENTUM

Evidence type: Registered Phase 2 obesity study

Supports:

- obesity population
- randomized clinical design
- SC pemvidutide research
- weekly administration framework

`https://clinicaltrials.gov/study/NCT05295875`

---

2. Altimmune: MOMENTUM Results

Evidence type: Sponsor-reported Phase 2 results

Supports:

- 1.2 mg
- 1.8 mg
- 2.4 mg
- 48-week weight-loss outcomes

Canonical Altimmune investor release should be used in production.

---

3. Harrison et al.: Pemvidutide in MASLD

Evidence type: Peer-reviewed randomized human research

Supports:

- weekly administration
- liver-fat reduction
- body-weight reduction
- metabolic/liver effects

https://pubmed.ncbi.nlm.nih.gov/39002641/

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4. Browne et al.: 24-Week MASLD Research

Evidence type: Human randomized clinical research

Supports:

- 24-week treatment
- 1.2 mg
- 1.8 mg
- 2.4 mg
- 56.3%, 75.2% and 76.4% liver-fat reductions

https://pubmed.ncbi.nlm.nih.gov/41113119/

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5. IMPACT Phase 2b

Evidence type: Randomized human MASH research

Supports:

- 1.2 mg / 1.8 mg
- MASH outcomes
- fibrosis-related outcomes

https://clinicaltrials.gov/study/NCT05989711

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6. Altimmune: Breakthrough Therapy Designation

Evidence type: Sponsor regulatory-development announcement

Supports:

- FDA Breakthrough Therapy Designation for MASH
- Fast Track status
- Phase 3 alignment

https://ir.altimmune.com/news-releases/news-release-details/altimmune-receives-fda-breakthro

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7. ClinicalTrials.gov: RECLAIM

Evidence type: Registered Phase 2 AUD trial

Supports:

- 2.4 mg weekly
- approximately 100 participants
- 24-week design

`https://clinicaltrials.gov/study/NCT06987513`

---

8. Altimmune: RECLAIM Topline Results

Evidence type: Sponsor-reported Phase 2 topline results

Supports:

- July 2026 positive AUD results
- primary heavy-drinking-day endpoint

Use the canonical Altimmune release in production.

---

9. Altimmune: PERFORMA Phase 3

Evidence type: Current sponsor development source

Supports:

- Phase 3 initiation
- August 3, 2026 start
- MASH registrational program

Use the canonical Altimmune PERFORMA press release in production.

Final Takeaway

Pemvidutide is a late-stage investigational drug with substantial human evidence. It should not be treated like a lightly studied research peptide, but it also should not be written about as if it were already FDA approved.

The strongest current protocol evidence supports:

once-weekly SC administration

with major human trial doses including:

1.2 mg

1.8 mg

and:

2.4 mg.

Those are:

clinical-trial doses

not an approved:

$1.2 \rightarrow 1.8 \rightarrow 2.4$ mg dosing ladder.

For weight loss, Phase 2 research reported up to approximately:

15.6% mean weight loss at 48 weeks.

For liver disease, pemvidutide has produced:

- large liver-fat reductions
- significant MASH-resolution activity

and has now progressed into:

Phase 3 MASH development.

For alcohol use disorder:

positive Phase 2 topline results were announced in July 2026, but this remains an investigational indication.

The biggest production rules to preserve are:

> **Do not call 1.2 mg the universal starting dose, 1.8 mg the standard maintenance dose, or 2.4 mg the advanced dose. They are trial doses, not an FDA-approved ladder.**
>
> **Keep once-weekly SC dosing because that is directly supported by the human program.**
>
> **Do not copy formal contraindications from approved GLP-1 drugs onto pemvidutide.**
>
> **Add PERFORMA Phase 3 prominently.**
>
> **Add RECLAIM AUD results, but identify them as sponsor-reported Phase 2 topline data.**
>
> **Do not use generic 14–28-day research-vial storage rules as though they came from the clinical program.**

This is the Pemvidutide page I would carry forward as the production version.

Retatrutide

GIP / GLP-1 / Glucagon Triple Receptor Agonist • Obesity Research • Type 2 Diabetes Research

---

At a Glance

What is Retatrutide?

Retatrutide is an **investigational peptide drug** that activates three hormone receptors:

- **GIP receptor**
- **GLP-1 receptor**
- **glucagon receptor**

That makes it a:

triple hormone receptor agonist

Its development name is:

LY3437943

Retatrutide is being developed by Eli Lilly and is administered in clinical trials as a:

once-weekly subcutaneous injection.

What do the three pathways do?

GLP-1

GLP-1 signaling can help:

- reduce appetite
- increase fullness
- lower food intake
- support glucose-dependent insulin secretion

GIP

GIP participates in:

- insulin signaling
- nutrient handling

- energy balance
- fat-tissue biology

Glucagon

Glucagon-receptor signaling can affect:

- liver metabolism
- glucose production
- lipid metabolism
- fat oxidation
- energy expenditure

Simple Version

Retatrutide is designed to affect both:

how much energy comes into the body

and:

how the body handles and uses stored energy.

How Well Studied Is It?

Very well studied for an investigational drug

Retatrutide has progressed far beyond early Phase 2 research.

It now has:

- Phase 1 human research
- published Phase 2 obesity research
- published Phase 2 type 2 diabetes research
- published human liver-fat research
- multiple completed Phase 3 obesity trials
- Phase 3 type 2 diabetes results
- obesity and knee-osteoarthritis research
- cardiovascular-risk research
- ongoing kidney, sleep-apnea, MASLD and other obesity-related studies.

As of August 2026, Lilly has announced positive Phase 3 results from:

- **TRIUMPH-1**
- **TRIUMPH-2** *(sponsor-reported topline results; full peer-reviewed publication pending)*

- **TRIUMPH-3** *(sponsor-reported topline results; full peer-reviewed publication pending)*
- **TRIUMPH-4**
- **TRANSCEND-T2D-1**.

Evidence Level

FDA-approved human evidence: None
Human clinical research: Very strong for an investigational drug
Obesity / weight-loss evidence: Very strong, including Phase 3
Type 2 diabetes evidence: Strong, including Phase 3
Liver-fat evidence: Strong human evidence
Long-term cardiovascular outcome evidence: Still being established
Approved-product safety evidence: None yet
Community protocols: Secondary to the actual clinical-trial data

---

What May It Help With?

Weight Loss

Very strong human investigational evidence

The published Phase 2 obesity trial already showed very large reductions in body weight.

At 48 weeks, average weight loss reached approximately:

24.2%

with the 12 mg dose.

Phase 3 has now gone further.

TRIUMPH-1 Phase 3

TRIUMPH-1 studied adults with obesity or overweight without type 2 diabetes.

Participants were assigned to target doses of:

- **4 mg**
- **9 mg**
- **12 mg**

- placebo

once weekly.

At 80 weeks, using the efficacy estimand, mean body-weight reduction was:

4 mg: 19.0%

9 mg: 25.9%

12 mg: 28.3%

Placebo: 2.2%.

At the 12 mg target:

45.3%

of participants achieved at least:

30% body-weight loss.

Longer TRIUMPH-1 Follow-Up

A prespecified extension followed selected participants with:

baseline BMI ≥35

through:

104 weeks

of total treatment.

Participants continuing retatrutide 12 mg reached approximately:

30.3% mean weight loss.

Important

Do not simplify this to:

> **Retatrutide causes 30% weight loss.**

That result came from:

- a specific higher-BMI extension population
- participants who completed the earlier treatment period
- continued treatment through 104 weeks

Better Wording

> **Phase 3 research has reported mean weight loss approaching 30% in selected higher-dose, longer-duration populations.**

TRIUMPH-2 Phase 3

TRIUMPH-2 studied adults with:

- obesity or overweight
- type 2 diabetes

for:

80 weeks.

Mean weight loss using the efficacy estimand was:

4 mg: 12.7%

9 mg: 19.1%

12 mg: 20.8%

Placebo: 4.0%.

HbA1c fell approximately:

1.4 to 1.6 percentage points

across retatrutide doses in that trial.

Evidence Classification

Positive Phase 3 topline human evidence

Detailed peer-reviewed publication is still pending.

TRIUMPH-3 Phase 3

TRIUMPH-3 studied people with:

- severe obesity
- established cardiovascular disease
- with or without type 2 diabetes

for:

80 weeks.

Mean weight loss was:

9 mg: 21.6%

12 mg: 22.6%

Placebo: 3.2%.

The study also measured cardiovascular events, but those event analyses were **not definitive cardiovascular-outcome proof** because the trial was not powered as a dedicated CV-outcomes study and confidence intervals crossed 1.

Correct interpretation

> **Retatrutide produced substantial weight loss in people with severe obesity and cardiovascular disease, while dedicated cardiovascular outcome research is still needed for firm cardiovascular-risk conclusions.**

Evidence Classification

Positive Phase 3 topline human evidence

Detailed peer-reviewed publication is still pending.

Type 2 Diabetes

Retatrutide has also shown strong glucose-lowering effects.

In:

TRANSCEND-T2D-1

participants with type 2 diabetes received retatrutide for:

40 weeks.

Mean HbA1c reductions were approximately:

4 mg: 1.7%

9 mg: 2.0%

12 mg: 1.9%

compared with:

0.8% with placebo.

Weight loss at 40 weeks reached:

16.8%

with 12 mg.

Important

Retatrutide remains:

investigational

and is not yet an FDA-approved diabetes medication.

Knee Osteoarthritis

TRIUMPH-4 studied adults with:

- obesity or overweight
- knee osteoarthritis
- no diabetes

for:

68 weeks.

Mean weight loss using the efficacy estimand was approximately:

9 mg: 26.4%

12 mg: 28.7%

Placebo: 2.1%.

The trial also reported substantial improvement in:

- knee pain
- physical function

alongside the weight loss.

Important

This does not make retatrutide:

an approved osteoarthritis medication.

It shows that knee-pain and function outcomes improved in a Phase 3 obesity population with knee osteoarthritis.

Liver Fat

Retatrutide has strong human investigational evidence involving liver fat.

A Phase 2 substudy in participants with MASLD found substantial reductions in:

liver fat

with retatrutide treatment.

Evidence Classification

Human clinical evidence

not merely:

animal or theoretical evidence

What Has It NOT Been Proven to Help With?

Retatrutide is not currently an approved treatment for:

- obesity
- type 2 diabetes
- knee osteoarthritis
- obstructive sleep apnea
- MASLD
- MASH
- cardiovascular disease
- chronic kidney disease
- chronic low back pain
- bodybuilding
- muscle growth
- athletic performance
- spot fat loss
- fatigue
- anti-aging
- longevity

Some of those conditions are being studied clinically.

But:

successful clinical trials are not the same thing as regulatory approval.

Dose at a Glance

FDA-Approved Dosing

There is none

Retatrutide has not been approved for public use by any regulatory agency.

There is no approved:

- starting dose
- maintenance dose
- maximum dose
- titration schedule
- commercial pen
- commercial vial
- prescribing label.

Human Clinical Research Dosing

It is important to separate:

Phase 2 dosing

from:

the current Phase 3 framework.

Phase 2 Obesity Research

The Phase 2 obesity trial studied target doses including:

- **1 mg**
- **4 mg**
- **8 mg**

- **12 mg**

with different starting-dose and escalation approaches.

These were:

clinical trial arms

not an approved dosing ladder.

Current Phase 3 Dosing Framework

The initial TRIUMPH Phase 3 program uses target doses of:

- **4 mg**
- **9 mg**
- **12 mg**

depending on the trial arm.

Participants assigned retatrutide generally begin at:

2 mg once weekly

and increase:

every 4 weeks

until reaching their assigned target.

Target 4 mg

2 mg weekly

then:

4 mg weekly

Target 9 mg

2 mg

→ 4 mg

→ 6 mg

→ 9 mg

with each step lasting about:

4 weeks.

Target 12 mg

2 mg

→ 4 mg

→ 6 mg

→ 9 mg

→ 12 mg

with approximately:

4 weeks at each step.

Important Dosing Distinction

Do not write:

> **The approved retatrutide titration is 2 → 4 → 6 → 9 → 12 mg.**

There is no approved retatrutide titration.

Correct Production Wording

> **Current Phase 3 trials generally start retatrutide at 2 mg once weekly and increase the dose about every four weeks until the participant reaches the assigned trial target dose.**

Website Card Correction

Do **not** leave the website card as:

> **Typical dose: 2.0 mg once weekly**

by itself.

2 mg is the Phase 3 trial starting dose, not a standalone typical dose or maintenance dose.

A clearer card label is:

Phase 3 dosing: 2 mg weekly start; studied targets 4, 9 or 12 mg

---

Research-Community Dosing

Community sources often borrow:

- genuine clinical-trial dose levels
- weekly escalation
- slower escalation
- lower-dose protocols
- “microdosing”
- research-vial schedules

Important

The fact that a community protocol uses a number that also appears in a clinical trial does **not** make the community protocol equivalent to the clinical trial regimen.

Research-vial sizes such as:

- 5 mg
- 10 mg
- 20 mg
- 30 mg

are:

product quantities

not:

validated treatment doses.

Evidence Classification

Clinical trial dose levels: Strong human evidence

Clinical Phase 3 escalation: Strong investigational human evidence

Community research-vial schedules: Community evidence

Every-3-day dosing: Unvalidated

Microdosing: Unvalidated

PRN / “as-needed” dosing: Unsupported

Frequency at a Glance

Human Clinical Research

Once weekly

This is extremely well supported.

Both Phase 2 and Phase 3 clinical programs use:

once-weekly SC administration.

Twice weekly?

Not established

Every 3–4 days?

Not established

Daily?

Not established

As needed?

Not established

5 days on / 2 days off?

Not applicable

Best-supported frequency

Once weekly

Timing at a Glance

There is no clinical evidence showing retatrutide must be taken:

- in the morning
- at night

- fasted
- with food
- before exercise
- after exercise

Evidence Verdict

Morning required: No
Night required: No
Fasted required: No
Fed required: No
Pre-workout required: No
Post-workout required: No

The clinically supported schedule is simply:

once weekly.

Duration / Cycle at a Glance

Retatrutide is being studied as:

long-term chronic therapy

rather than a short peptide cycle.

Human studies now include:

- **40 weeks**
- **48 weeks**
- **68 weeks**
- **80 weeks**
- selected **104-week** follow-up
- multi-year cardiovascular and kidney outcome research.

Does Retatrutide Need to Be Cycled?

No evidence-based cycle exists

There is no established reason for:

- 8 weeks on / 4 weeks off
- 12-week cycles
- monthly breaks
- GLP-1 resets

- GIP resets
- glucagon-receptor resets

Verdict

Continuous weekly treatment matches the human development program far better than peptide-style cycling.

Expected Results Timeline

Clinical Weight-Loss Research

First Several Weeks

Participants are still moving through dose escalation.

During this period, changes may include:

- appetite
- fullness
- GI tolerability

Maximum body-weight effects should not be expected immediately.

Around 24 Weeks

The Phase 2 obesity study already showed large reductions in body weight by this point.

Around 40 Weeks

TRANSCEND-T2D-1 showed substantial:

- HbA1c improvement
- weight reduction

by week 40.

Around 48 Weeks

The Phase 2 obesity study reported approximately:

24.2% mean weight loss

with 12 mg.

Around 68 Weeks

TRIUMPH-4 reported up to approximately:

28.7% mean weight loss

with 12 mg using the efficacy estimand.

Around 80 Weeks

TRIUMPH-1 reported:

4 mg: 19.0%

9 mg: 25.9%

12 mg: 28.3%.

TRIUMPH-2 and TRIUMPH-3 also reported major weight reductions at:

80 weeks.

These TRIUMPH-2 and TRIUMPH-3 results are currently **sponsor-reported Phase 3 topline data**, with detailed peer-reviewed publications still pending.

Around 104 Weeks

A selected higher-BMI TRIUMPH-1 extension group receiving 12 mg reached approximately:

30.3% mean weight loss.

Clinical Takeaway

Retatrutide's major body-weight effects develop over many months, not days.

Community-Reported Timeline

Community users commonly describe:

early weeks: appetite or GI changes

1–3 months: visible weight changes

several months: larger body-composition changes

Important

The controlled clinical timeline is much stronger evidence and should remain the main timeline.

Best Top-Card Wording

Clinical: weight loss builds progressively over months, with major controlled outcomes measured at 24, 40, 48, 68, 80 and selected 104-week time points.

Community-reported: appetite and tolerability changes are often described sooner, but anecdotal timelines should not replace controlled trial data.

Safety at a Glance

Common Human Trial Effects

The dominant adverse effects are gastrointestinal, including:

- nausea
- diarrhea
- constipation
- vomiting
- decreased appetite

They generally become more common at higher doses and during escalation.

Dysesthesia

This should now be included prominently because it has appeared repeatedly in Phase 3.

What is dysesthesia?

Dysesthesia means an abnormal or unpleasant sensation.

People may describe:

- tingling
- burning
- unusual skin sensitivity
- altered sensation to touch

In TRIUMPH-4, dysesthesia occurred in:

9 mg: 8.8%

12 mg: 20.9%

Placebo: 0.7%.

Lilly said these events were generally mild and rarely caused discontinuation.

The signal was also seen in the newer TRIUMPH-2 and TRIUMPH-3 results, which makes it more important than an isolated one-trial finding.

Evidence Verdict

Established Phase 3 treatment-emergent safety signal

Treatment Discontinuation

In TRIUMPH-4, discontinuation because of adverse events was:

9 mg: 12.2%

12 mg: 18.2%

Placebo: 4.0%.

Lilly noted that some discontinuations were related to:

perceived excessive weight loss

rather than classic toxicity alone.

Hypoglycemia

Retatrutide affects glucose and insulin-related pathways.

In the Phase 2 type 2 diabetes study, severe hypoglycemia was not a dominant safety finding.

Important

Risk may differ when retatrutide is combined with:

- insulin
- sulfonylureas
- other glucose-lowering drugs

Verdict

Do not present hypoglycemia as a universal major adverse effect in people not using other glucose-lowering therapies.

Formal Contraindications

None established through an FDA-approved retatrutide label

Retatrutide is not approved.

There is therefore no official FDA retatrutide contraindication section.

Do not copy contraindications from:

- Wegovy
- Ozempic
- Zepbound
- Mounjaro
- Saxenda

and label them:

official retatrutide contraindications.

Important Cautions

Pregnancy / Breastfeeding

Human treatment safety is not established.

Classification: Important investigational-use caution.

Significant Gastrointestinal Disease

GI adverse effects are common in the clinical program.

Classification: Clinically relevant caution.

Diabetes Medications

Other glucose-lowering medications can potentially alter hypoglycemia risk.

Classification: Important interaction consideration.

Pancreatitis

A history of pancreatitis may be clinically relevant for an investigational incretin-based drug.

But there is no approved retatrutide label establishing this as a formal contraindication.

Classification: Clinical caution, not formal contraindication.

MTC / MEN2

Do not automatically import the medullary thyroid carcinoma / MEN2 contraindications from approved GLP-1 products.

Retatrutide does not yet have an FDA-approved label defining these as formal contraindications.

Verdict

Do not call them official unless a future approved label does so.

Regulatory Status at a Glance

FDA Approved?

No

Retatrutide remains investigational.

Approved Anywhere?

Retatrutide has not been approved for public use by any regulatory agency.

Phase 3?

Yes

Five positive Phase 3 trial readouts have now been announced. **TRIUMPH-2 and TRIUMPH-3 are currently sponsor-reported topline results, with full peer-reviewed publications still pending.**

Planned FDA Submission

Lilly stated in July 2026 that it plans to submit a:

Biologics License Application in Q1 2027

for U.S. approval.

Important

A planned submission does not mean:

FDA approval is guaranteed.

Bottom Line

Retatrutide now has exceptionally strong human evidence for an investigational metabolic drug, but it remains unapproved.

The strongest dosing evidence supports:

once-weekly subcutaneous administration.

Current Phase 3 programs generally begin at:

2 mg once weekly

and increase approximately:

every 4 weeks

toward target doses of:

- **4 mg**
- **9 mg**
- **12 mg**.

But those are:

Phase 3 clinical-trial regimens

not:

FDA-approved prescribing instructions.

Protocol Logic Check

Quick Logic Summary

Protocol Question	Verdict
GIP receptor agonist?	**Yes**
GLP-1 receptor agonist?	**Yes**
Glucagon receptor agonist?	**Yes**
True triple agonist?	**Yes**
Once-weekly SC dosing?	**Strongly supported**
2 mg used as Phase 3 starting dose?	**Yes**
4 mg Phase 3 target?	**Yes**
9 mg Phase 3 target?	**Yes**
12 mg Phase 3 target?	**Yes**
2 → 4 → 6 → 9 → 12 mg used for the 12 mg arm?	**Yes**

Protocol Question	Verdict
Is that FDA-approved titration?	**No**
1 / 4 / 8 / 12 mg Phase 2 doses?	**Yes**
Daily dosing?	**Not established**
Twice-weekly dosing?	**Not established**
PRN dosing?	**Unsupported**
Microdosing validated?	**No**
Morning required?	**No**
Fasted required?	**No**
Pre-workout dosing?	**No demonstrated reason**
Short 8–12 week cycle?	**No demonstrated rationale**
Receptor reset?	**No established requirement**
FDA approved?	**No**
Phase 3 data available?	**Yes, five positive studies announced; TRIUMPH-2/3 topline pending full publication**

What Does Retatrutide Target?

Retatrutide activates:

GIP receptors

GLP-1 receptors

and:

glucagon receptors.

Is “GLP-3” a Real Name?

No

“GLP-3” is an informal online nickname.

There is no hormone called:

GLP-3

in retatrutide’s mechanism.

Lilly specifically describes the term as scientifically inaccurate.

Correct Wording

GIP / GLP-1 / glucagon triple agonist

or:

triple hormone receptor agonist

Human Half-Life

Retatrutide is long acting enough to support:

once-weekly administration

across both Phase 2 and Phase 3 research.

Better Rule

Do not create a dosing frequency by reverse-engineering one plasma half-life estimate.

The actual human trial program already demonstrates the clinically tested interval:

once weekly.

How Long Do the Effects Last?

Weekly administration maintains enough pharmacologic activity for continued clinical treatment.

Verdict

Weekly activity is strongly supported in humans.

Does It Build Up With Weekly Use?

Repeated weekly administration produces continued exposure over many months.

But claims about:

- a precise “saturation day”
- exact metabolic loading
- exact day of steady state

should not be included without specific pharmacokinetic evidence.

Verdict

Repeated weekly exposure is established. Simplified accumulation claims are not.

Does Once-Weekly Dosing Make Sense?

Yes

This is strongly supported across the development program.

Verdict

Evidence-supported

Does Twice-Weekly Dosing Make Sense?

There is no clinical evidence showing that splitting the weekly amount into:

- Monday / Thursday
- every 3.5 days
- two smaller injections

improves:

- weight loss
- glucose control
- tolerability
- liver outcomes

Verdict

Not established

Daily Dosing

No major clinical retatrutide program uses daily administration.

Verdict

Not established

“As Needed” Dosing

No clinical protocol uses retatrutide only when:

- hunger returns
- appetite increases
- weight stalls

Verdict

Unsupported

Morning or Evening?

No clinically demonstrated advantage exists.

Verdict

Preference, not evidence-based

Fasted or Fed?

No evidence shows retatrutide must be injected while:

- fasted
- fed

Verdict

No demonstrated requirement

Pre-Workout Timing

Retatrutide is a long-acting weekly metabolic drug.

There is no clinical reason to time it around:

a workout.

Verdict

No demonstrated advantage

Dose Escalation / Titration

Phase 2

Earlier obesity research studied target doses including:

- 1 mg
- 4 mg
- 8 mg
- 12 mg

with multiple starting-dose strategies.

Phase 3

The TRIUMPH program standardized a clearer escalation framework.

Participants generally begin:

2 mg once weekly

and increase:

every four weeks.

4 mg target

2 → 4 mg

9 mg target

2 → 4 → 6 → 9 mg

12 mg target

2 → 4 → 6 → 9 → 12 mg

Why?

The gradual escalation is designed largely around:

tolerability, particularly GI tolerability.

Verdict

Strong clinical-trial precedent

but still:

investigational dosing

Is 2 mg the Official Starting Dose?

No.

It is the:

current Phase 3 trial starting dose

not an approved prescribing dose.

Is 4 mg a Maintenance Dose?

It is a:

Phase 3 target dose

in some trial arms.

It is not yet an approved low maintenance dose.

Is 9 mg a Maintenance Dose?

It is also a:

Phase 3 target dose

not an FDA-approved maintenance dose.

Is 12 mg the Maximum Approved Dose?

No.

It is the highest major Phase 3 target in the current program.

There is no:

approved maximum dose

because retatrutide has not been approved.

Does It Need an 8–12 Week Cycle?

No.

The evidence points strongly toward long-term treatment.

Clinical studies last many months and some extend beyond:

two years.

Verdict

Short peptide cycles do not match the clinical development model.

Does It Need a Receptor Reset?

There is no established:

- GIP reset
- GLP-1 reset
- glucagon reset

Verdict

No evidence-based receptor-reset requirement

Does It Need a Washout?

There is no validated:

- 2-week break
- 4-week break
- monthly reset

as part of chronic retatrutide treatment.

Verdict

No demonstrated requirement

Does the Route Match the Research?

Clinical Route

Subcutaneous

Frequency

Once weekly

Verdict

Strong evidence match

Stack / Pairing Logic

Retatrutide + Semaglutide

Semaglutide activates GLP-1.

Retatrutide already activates:

- GLP-1
- GIP
- glucagon

Verdict: Major pathway overlap; no validated combination.

Retatrutide + Tirzepatide

Tirzepatide activates:

- GIP
- GLP-1

Retatrutide already activates both plus glucagon.

Verdict: Very high pathway overlap.

Retatrutide + Pemvidutide

Pemvidutide activates:

- GLP-1
- glucagon

Both are already included in retatrutide's mechanism.

Verdict: Extremely high receptor redundancy.

Retatrutide + Liraglutide

Both activate GLP-1 receptors.

Verdict: Major overlap; clinically unvalidated.

Retatrutide + Cagrilintide

Cagrilintide primarily uses amylin-related signaling.

Verdict: Less receptor overlap, but there is no validated retatrutide combination protocol.

Retatrutide + Metformin

Different primary mechanism.

Verdict: Mechanistically nonredundant, but combination claims should come from actual human evidence.

Important Research Note

Three separate things are frequently mixed together online.

1. Lilly Clinical-Trial Retatrutide

This is the actual investigational drug used in controlled studies.

Trials control:

- identity
- formulation
- manufacturing
- dose
- storage
- administration
- participant monitoring

---

2. Clinical-Trial Dose Levels

Phase 2

- 1 mg
- 4 mg
- 8 mg
- 12 mg

Phase 3 Targets

- 4 mg
- 9 mg
- 12 mg

with escalation beginning at:

2 mg

in the initial TRIUMPH program.

These are:

real human trial doses.

---

3. Online Research Vials

Online products may be sold as:

- 5 mg
- 10 mg
- 20 mg
- 30 mg

Those numbers describe:

how much material is in the vial

not:

a clinically studied dose.

A:

30 mg vial

does not mean:

> inject 30 mg.

Formulation / Stability / Sterility Logic

There is no FDA-approved commercial retatrutide product.

Therefore there is no universal approved:

- vial concentration
- pen concentration
- bacteriostatic-water volume
- reconstitution protocol
- 14-day rule
- 28-day rule
- multidose-vial standard

Lilly also warns that products claiming to be retatrutide outside clinical trials are not Lilly's investigational product and may have problems with identity, purity or composition.

Verdict

Research-vial handling cannot be copied from the Lilly clinical program unless the exact formulation supports it.

Research at a Glance

Approved-Label Human Evidence

None

Retatrutide is unapproved.

Human Clinical Research

Very Strong for an Investigational Drug

Five positive Phase 3 study readouts have now been announced. **TRIUMPH-2 and TRIUMPH-3 remain sponsor-reported topline Phase 3 results pending full peer-reviewed publication.**

Weight-Loss Evidence

Very Strong

Phase 3 results show mean reductions above:

25%

in some higher-dose populations and around:

30%

in a selected 104-week extension population.

Type 2 Diabetes Evidence

Strong

Phase 3 data now show substantial:

- HbA1c reduction
- body-weight reduction.

Liver Evidence

Strong Human Investigational Evidence

Human research demonstrates large reductions in:

liver fat.

Mechanistic Evidence

Strong

The:

GIP / GLP-1 / glucagon

triple mechanism is well established.

Community Evidence

Secondary

Retatrutide now has enough direct human dosing data that community schedules should not be used as the main protocol anchor.

Overview

Retatrutide is an investigational:

GIP / GLP-1 / glucagon triple receptor agonist

developed by Eli Lilly.

It combines:

- appetite-related signaling
- glucose and insulin-related signaling
- liver and energy-metabolism signaling

in one molecule.

It has now produced positive Phase 3 results in populations involving:

- obesity
- obesity with type 2 diabetes
- severe obesity with cardiovascular disease

- obesity with knee osteoarthritis
- type 2 diabetes.

The TRIUMPH-2 and TRIUMPH-3 results in that Phase 3 picture are currently **sponsor-reported topline data pending full peer-reviewed publication**.

But it remains:

unapproved and investigational.

Evidence Strength

Overall Evidence Rating

Very strong human investigational evidence

Strong Evidence For

- GIP receptor agonism
- GLP-1 receptor agonism
- glucagon receptor agonism
- once-weekly SC administration
- substantial weight loss
- glucose lowering
- fat-mass reduction
- liver-fat reduction
- GI adverse effects
- structured dose escalation

Emerging / Ongoing Evidence For

- cardiovascular outcomes
- kidney outcomes
- obstructive sleep apnea
- chronic low back pain
- long-term weight maintenance
- head-to-head comparison with tirzepatide

Not Established

- FDA-approved obesity dosing
- FDA-approved diabetes dosing
- public prescribing titration
- stacking with other incretin agonists

- research-vial equivalence
- bodybuilding
- anti-aging
- short cycles
- receptor resets

Category

GIP / GLP-1 / Glucagon Triple Agonists • Obesity • Metabolic Health

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Compound

Retatrutide

Development name:

LY3437943

Compound Type

Long-acting peptide triple hormone receptor agonist

Retatrutide is:

- peptide-based
- a GIP receptor agonist
- a GLP-1 receptor agonist
- a glucagon receptor agonist
- designed for weekly administration

It is not:

- insulin
- a stimulant
- “GLP-3”
- an approved medication

Primary Research Focus

Most Advanced

- obesity
- type 2 diabetes

Other Major Human Research

- obesity with knee osteoarthritis
- cardiovascular outcomes
- obstructive sleep apnea
- kidney outcomes
- MASLD
- weight maintenance
- chronic low back pain.

Not Established

- bodybuilding
- athletic enhancement
- general wellness
- anti-aging

Common Research Dose

Phase 2 Obesity

Major targets included:

1 mg

4 mg

8 mg

12 mg.

Phase 3 TRIUMPH

Major target doses include:

4 mg

9 mg

12 mg

once weekly.

Participants generally begin at:

2 mg weekly

and increase about:

every four weeks

toward the assigned target.

Research Duration / Cycle Length

Human studies include:

40 weeks

48 weeks

68 weeks

80 weeks

and selected:

104-week follow-up.

Evidence Assessment

Retatrutide is being developed as:

long-term treatment

not a short peptide cycle.

How Often It Is Used

Human Clinical Research

Once weekly SC

Evidence Assessment

Strongly supported

Possible Benefits

Weight Loss

Very strong human evidence

Blood Sugar Control

Strong human evidence in type 2 diabetes

Body-Fat Reduction

Human body-composition research shows meaningful reductions in fat mass.

Liver Fat

Strong investigational human evidence

Cardiometabolic Measures

Clinical studies have reported improvements in markers including:

- waist circumference
- triglycerides
- non-HDL cholesterol
- systolic blood pressure
- hsCRP.

Knee Pain and Function

TRIUMPH-4 showed major improvement in knee osteoarthritis symptoms alongside weight loss.

Important

That does not make retatrutide an approved pain medication.

Community Protocol Comparison

Protocol	Dose	Frequency	Evidence
Phase 2 obesity	1 / 4 / 8 / 12 mg targets	Weekly	Phase 2 human
Phase 3 starting dose	2 mg	Weekly	Phase 3 human
Phase 3 low target	4 mg	Weekly	Phase 3 human
Phase 3 middle target	9 mg	Weekly	Phase 3 human
Phase 3 high target	12 mg	Weekly	Phase 3 human
Research-vial protocols	Variable	Variable	Community
Microdose protocols	Variable	Variable	Unvalidated
PRN protocols	Variable	Variable	Unsupported

Strongest Agreement

Once-weekly SC administration

Biggest Mistake to Avoid

Treating:

vial size

as:

clinical dose

or treating the Phase 3 escalation schedule as an already approved prescription.

Formulation / Stability / Sterility

No FDA-Approved Commercial Product Exists

There is no approved retatrutide reference product providing one universal:

- vial size
- concentration
- reconstitution volume
- bacteriostatic-water instruction
- post-reconstitution expiration
- 14-day storage rule
- 28-day storage rule

Simple Rule

Use handling instructions for the exact formulation.

Research Sources

Use these **clean canonical URLs in production**.

1. Jastreboff et al. Phase 2 Obesity Trial

Evidence type: Randomized Phase 2 human research

Supports:

- triple receptor mechanism
- once-weekly administration
- Phase 2 target doses
- 48-week weight loss
- approximately 24.2% mean weight loss with 12 mg

`https://pubmed.ncbi.nlm.nih.gov/37366315/`

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2. Rosenstock et al. Phase 2 Type 2 Diabetes Trial

Evidence type: Randomized Phase 2 human research

Supports:

- glucose lowering
- body-weight reduction

- safety and tolerability
- Phase 2 diabetes dosing

https://pubmed.ncbi.nlm.nih.gov/37385280/

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3. Sanyal et al. Liver-Fat Substudy

Evidence type: Human clinical liver research

Supports:

- MASLD population
- liver-fat reduction
- metabolic liver effects

https://pubmed.ncbi.nlm.nih.gov/38858523/

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4. TRIUMPH-1 Phase 3

Evidence type: Sponsor-reported Phase 3 results

Supports:

- 4 / 9 / 12 mg targets
- 2 mg Phase 3 starting dose
- 4-week escalation
- 19.0%, 25.9%, 28.3% weight-loss results
- 104-week extension
- 30.3% extension result

https://investor.lilly.com/news-releases/news-release-details/lillys-triple-agonist-retatrutide-delivered-powerful-weight-loss

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5. TRIUMPH-4 Phase 3

Evidence type: Sponsor-reported Phase 3 results

Supports:

- 9 mg and 12 mg targets
- 68-week obesity results
- knee pain and function
- Phase 3 escalation design
- dysesthesia

- discontinuation data

https://investor.lilly.com/news-releases/news-release-details/lillys-triple-agonist-retatrutide-delivered-weight-loss-average

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6. TRANSCEND-T2D-1 Phase 3 Publication

Evidence type: Peer-reviewed Phase 3 type 2 diabetes research

Supports:

- 4 / 9 / 12 mg doses
- HbA1c reduction
- body-weight reduction
- Phase 3 diabetes efficacy

https://pubmed.ncbi.nlm.nih.gov/42250575/

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7. Lilly TRANSCEND-T2D-1 Results

Evidence type: Sponsor-reported Phase 3 results

Supports:

- 1.7–2.0 percentage-point HbA1c reductions
- 16.8% weight loss at 12 mg
- 40-week results

https://investor.lilly.com/news-releases/news-release-details/lillys-triple-agonist-retatrutide-demonstrated-significant

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8. Lilly TRIUMPH-2 and TRIUMPH-3 Results

Evidence type: Sponsor-reported Phase 3 topline results

Supports:

- five-positive-Phase-3-study status
- TRIUMPH-2 4 / 9 / 12 mg results
- TRIUMPH-3 9 / 12 mg results
- dysesthesia across additional Phase 3 studies
- planned Q1 2027 U.S. BLA submission

https://investor.lilly.com/news-releases/news-release-details/lillys-triple-agonist-retatrutide-successful-two-additional

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9. Lilly: What to Know About Retatrutide

Evidence type: Current sponsor development source

Supports:

- investigational status
- once-weekly SC route
- GIP / GLP-1 / glucagon mechanism
- “GLP-3” is scientifically inaccurate
- current development indications

https://www.lilly.com/news/stories/what-to-know-about-retatrutide

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10. ClinicalTrials.gov: TRIUMPH-1

Evidence type: Registered Phase 3 study

https://clinicaltrials.gov/study/NCT05929066

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11. ClinicalTrials.gov: TRIUMPH-2

Evidence type: Registered Phase 3 study

https://clinicaltrials.gov/study/NCT05929079

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12. ClinicalTrials.gov: TRIUMPH-3

Evidence type: Registered Phase 3 study

https://clinicaltrials.gov/study/NCT05882045

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13. ClinicalTrials.gov: TRIUMPH-4

Evidence type: Registered Phase 3 study

`https://clinicaltrials.gov/study/NCT05931367`

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14. ClinicalTrials.gov: TRIUMPH-OUTCOMES

Evidence type: Ongoing Phase 3 cardiovascular / renal outcomes research

`https://clinicaltrials.gov/study/NCT06383390`

Final Takeaway

Retatrutide is no longer just a promising Phase 2 research compound. It is a late-stage investigational metabolic drug with five positive Phase 3 trial readouts, but it remains unapproved.

TRIUMPH-2 and TRIUMPH-3 are currently sponsor-reported topline Phase 3 results, with full peer-reviewed publications still pending.

Its mechanism combines:

GIP + GLP-1 + glucagon receptor agonism

in one molecule.

The strongest protocol evidence supports:

once-weekly subcutaneous administration.

The initial Phase 3 TRIUMPH program generally starts participants at:

2 mg once weekly

and increases approximately:

every four weeks

toward trial target doses of:

4 mg, 9 mg or 12 mg.

The critical wording is:

These are investigational Phase 3 trial doses, not FDA-approved prescribing instructions.

The biggest production corrections are:

Replace any “Phase 2” status with current Phase 3 status and include all five positive Phase 3 readouts.

Use “GIP / GLP-1 / glucagon triple agonist.” Do not call retatrutide “GLP-3.”

Separate Phase 2 1 / 4 / 8 / 12 mg arms from the current Phase 3 2 mg start and 4 / 9 / 12 mg targets.

Keep the 2 → 4 → 6 → 9 → 12 mg sequence only as the Phase 3 escalation to the 12 mg target, not as an FDA-approved universal ladder.

Keep once-weekly SC administration as the evidence-supported frequency.

Remove or clearly downgrade every-3-day, twice-weekly, PRN, microdosing, short-cycle and receptor-reset protocols.

Add dysesthesia as a real Phase 3 safety signal.

Do not import Wegovy, Zepbound or other GLP-1 product contraindications as official retatrutide contraindications.

Do not treat 5, 10, 20 or 30 mg research-vial sizes as human treatment doses.

Do not use generic 14- or 28-day vial-stability rules as though they came from Lilly’s clinical program.

This is the Retatrutide version I would carry forward as the production page.

GGH

GHK-Cu / Glutathione Blend • Recovery Research • Antioxidant Biology • Skin and Tissue Research

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At a Glance

What is GGH?

GGH is a **community/vendor blend name**, not one standardized drug or one standardized molecule.

The current EvidenceCodex page describes it primarily as a blend involving:

- **GHK-Cu**
- **glutathione**

However, some products sold under the name **GGH** may also contain other ingredients such as:

- histidine
- glycine
- NADH

That means the name **GGH alone does not tell you exactly what is in the vial**.

Important

Before a GGH dose can be interpreted, the exact formulation needs to be known.

Exact Formula

For any specific GGH product, confirm:

GHK-Cu: _____ mg/mL
Glutathione: _____ mg/mL
Histidine: _____ mg/mL, if present
Glycine: _____ mg/mL, if present
NADH: _____ mg/mL, if present
Other ingredients: _____

Why this matters

A recommendation such as:

> **0.5 to 1 mL**

is only a volume.

It does **not** tell us how much GHK-Cu, glutathione or any other active ingredient is actually being administered.

Important Name Warning

In scientific literature, **GGH can also mean Gly-Gly-His**, also called glycyl-glycyl-histidine.

That is a separate copper-binding tripeptide.

It is **not the same thing as the commercial GGH blend discussed on this page**.

Production rule

Studies involving:

Gly-Gly-His / GGH peptide

should not automatically be used as evidence for:

GHK-Cu + glutathione GGH blends.

How Well Studied Is It?

The individual ingredients have meaningful biological research. The finished GGH blend does not.

This distinction is essential.

GHK-Cu

GHK-Cu has research involving:

- skin biology
- collagen and extracellular matrix
- tissue remodeling
- wound healing
- inflammatory signaling
- oxidative stress

Some human evidence exists, but much of the better human evidence is related to:

topical use

rather than long-term injection protocols.

Glutathione

Glutathione is one of the body's most important naturally occurring antioxidants.

Its biological role is extremely well established.

It participates in:

- cellular redox balance

- antioxidant defense
- detoxification chemistry
- protection against oxidative stress

But strong glutathione biology does **not** automatically prove that injecting a GGH blend improves:

- recovery
- skin quality
- energy
- healing
- liver function
- anti-aging outcomes

Exact GGH Blend

For the finished injectable blend:

Human clinical efficacy: Not established
Human dose-finding: Not established
Human pharmacokinetics: Not established
Long-term safety: Not established
Validated SC protocol: Not established
Validated IM protocol: Not established
Optimal frequency: Not established
Same-vial compatibility: Not clinically established

Evidence Level

GHK-Cu biology: Moderate to strong mechanistic/preclinical evidence
Human topical GHK-Cu evidence: Limited but real
Injectable GHK-Cu evidence: Limited
Glutathione biology: Very strong
Exact injectable GGH blend: Very limited
Community GGH protocols: Vendor/practice derived

What May It Help With?

GHK-Cu Component

GHK-Cu has biological evidence supporting research into:

- extracellular matrix remodeling
- collagen-related processes
- skin repair
- wound biology

- tissue regeneration
- inflammatory signaling

Important

That does not mean an injectable GGH blend has been proven to produce the same results.

Glutathione Component

Glutathione has strong biological roles involving:

- antioxidant defense
- cellular redox balance
- protection from oxidative stress

Some research also exists involving glutathione and skin pigmentation, but the evidence differs considerably by:

- oral use
- topical use
- IV use

Important

Research on one glutathione route should not automatically be applied to another.

What Has the Exact GGH Blend Been Proven to Help With?

No specific clinical benefit has been established

I would not describe the exact blend as proven to:

- accelerate wound healing
- improve skin quality
- increase collagen
- lighten skin
- improve athletic recovery
- repair muscles
- repair tendons
- detoxify the liver
- reduce inflammation clinically
- improve mitochondrial function
- increase energy
- slow aging

The ingredients may provide biological reasons to study some of these areas.

That is different from demonstrating the finished blend works clinically.

Dose at a Glance

FDA-Approved Dosing

There is none

There is no FDA-approved GGH fixed-combination product.

There is therefore no approved:

- starting dose
- maintenance dose
- SC dose
- IM dose
- dosing frequency
- cycle length
- titration schedule

Community / Vendor Dosing

The current EvidenceCodex page previously described:

0.5 to 1 mL

approximately:

2 to 3 times per week

using SC or IM administration.

Evidence Classification

Community / vendor protocol

Not:

clinically validated GGH dosing

Why mL Alone Is Not Enough

This is one of the most important corrections to the previous page.

Imagine two different GGH products.

Product A

1 mL contains:

- 2 mg GHK-Cu
- 200 mg glutathione

Product B

1 mL contains:

- 10 mg GHK-Cu
- 500 mg glutathione

Taking:

1 mL

would mean completely different active doses.

Verdict

A GGH protocol should never be interpreted from injection volume alone.

The amount of each ingredient must be provided in:

mg or another appropriate unit per dose.

Frequency at a Glance

Clinical Evidence

No clinically established frequency exists for GGH

There is no controlled human trial showing that the exact blend should be administered:

- daily
- every other day
- 2x/week
- 3x/week
- weekly

Community

Reported schedules include use several times per week.

Evidence Classification

Community practice

Best-Supported Frequency

Unknown

Timing at a Glance

There is no evidence showing the GGH blend works better:

- in the morning
- at night
- fasted
- with food
- before exercise
- after exercise

Evidence Verdict

Morning required: No
Evening required: No
Fasted required: No
With food required: No
Pre-workout required: No
Post-workout required: No

Duration / Cycle at a Glance

There is no established clinical GGH cycle.

No evidence shows a biological requirement for:

- 5 days on / 2 days off
- 8-week cycles
- 12-week cycles
- 2-week washouts
- 4-week washouts

Does GGH Need a Receptor Reset?

No evidence supports this

GGH is not one conventional receptor agonist.

It is a blend of compounds with different biological roles.

There is no established:

- GGH receptor
- GHK-Cu receptor-reset protocol
- glutathione receptor reset
- required washout period

Verdict

No evidence-based receptor-reset logic

Expected Results Timeline

Clinical Timeline

Not established

There is no controlled human GGH study supporting a timeline such as:

Week 1: better recovery
Week 2: brighter skin
Week 4: more collagen
Week 8: tissue regeneration

These should not be presented as clinical facts.

Community-Reported Timeline

Community sources may describe changes in:

- skin appearance
- recovery
- perceived inflammation
- general wellness

over days or weeks.

Important

These remain:

anecdotal observations

and should not be combined with clinical study timelines from the individual ingredients.

Bottom Line

GGH is a poorly standardized blend built from ingredients that individually have legitimate biological research.

That does not make the blend itself clinically validated.

The most important things to understand are:

- GGH is not one standardized compound
- different GGH products may contain different ingredients
- dose cannot be interpreted from mL alone
- no validated human GGH dosing protocol has been identified
- component evidence does not prove combination efficacy
- GHK-Cu and glutathione can chemically interact
- injectable GHK-Cu has specific current FDA safety concerns
- injectable glutathione also carries important sterile-product quality risks

Strongest conclusion

> **The exact formula and concentration should be known before any GGH protocol can even be meaningfully evaluated.**

Protocol Logic Check

Protocol Question	Verdict
Is GGH one standardized molecule?	**No**
Is every GGH product the same formula?	**No**
Can GGH also mean Gly-Gly-His in scientific literature?	**Yes**
Is Gly-Gly-His research evidence for this blend?	**No**
Human clinical trial of exact GGH blend identified?	**No**
FDA-approved GGH product?	**No**
Is 0.5–1 mL meaningful without concentration?	**No**
Is 0.5–1 mL 2–3x/week clinically validated?	**No**
Is SC validated for the exact blend?	**No**
Is IM validated for the exact blend?	**No**
Does GHK-Cu have biological evidence?	**Yes**
Is human GHK-Cu evidence stronger topically?	**Yes**
Does glutathione have strong antioxidant biology?	**Yes**
Does that prove injected GGH works?	**No**
Can glutathione chemically interact with Cu(II)-GHK?	**Yes, in laboratory research**
Does that automatically prove the finished blend is unstable?	**No**

Protocol Question	Verdict
Should compatibility be demonstrated for the exact formula?	**Yes**
Does injectable GHK-Cu have FDA safety concerns?	**Yes**
Have injectable glutathione contamination events occurred?	**Yes**
Is a "Standard Protocol" justified?	**No**
Does timing matter?	**Not established**
Does GGH require cycling?	**No evidence**
Does it need receptor reset?	**No evidence**
Are BPC-157 / TB-500 synergies proven?	**No**
Can generic vial stability be assumed?	**No**

What Does GHK-Cu Do?

GHK is a naturally occurring tripeptide:

Gly-His-Lys

GHK can bind copper to form:

GHK-Cu

GHK-Cu has been studied for effects involving:

- extracellular matrix
- collagen
- tissue remodeling
- wound biology
- inflammatory signaling
- oxidative stress

Simple Version

GHK-Cu appears to act more like a:

tissue-signaling and remodeling molecule

than a conventional drug acting through one simple receptor.

Important

This is:

GHK-Cu evidence

not:

GGH blend evidence

What Does Glutathione Do?

Glutathione is a naturally occurring antioxidant molecule.

It participates in the body's redox system and helps cells manage oxidative stress.

Simple Version

Think of glutathione as part of the body's:

internal antioxidant defense system.

Important

Being biologically essential does not prove that:

> injecting extra glutathione in a GGH blend improves health in an otherwise healthy person.

GHK-Cu + Glutathione Compatibility

This needs its own section.

Laboratory research has directly shown that:

glutathione can chemically interact with Cu(II)-GHK.

The chemistry involves reduction of the copper center and formation of intermediate copper-thiolate species.

What This Means

It means the two ingredients are **not necessarily chemically invisible to each other** when mixed.

What It Does NOT Mean

It does not automatically prove that:

- every GGH vial is unstable
- every blend degrades rapidly
- the mixture is unsafe
- all biological activity is lost

Correct Production Wording

> **Glutathione can chemically interact with Cu(II)-GHK in laboratory systems. This does not prove a finished GGH formulation is unstable, but it means same-vial compatibility and stability should be demonstrated for the exact product rather than assumed.**

Verdict

Formulation-specific compatibility data needed

Can We Assume the Ingredients Are Synergistic?

No

GHK-Cu and glutathione have different biological roles.

That can make the combination sound complementary.

But:

different mechanisms do not automatically equal clinical synergy.

No controlled human study was identified showing that the exact combination produces better outcomes than either component alone.

Verdict

Mechanistically interesting, clinically unproven

Route Logic

Subcutaneous

Used in community protocols.

Evidence Classification

Community route

No controlled GGH clinical program establishes SC administration.

Intramuscular

Also described in community/vendor use.

Evidence Classification

Community route

No controlled GGH study establishes IM administration.

Copper Exposure

GHK-Cu contains copper.

That makes total copper exposure potentially relevant.

But the risk cannot be meaningfully evaluated from:

0.5 mL

or:

1 mL

unless we know how much GHK-Cu is actually in that volume.

To Estimate Copper Exposure We Need

- GHK-Cu concentration
- amount injected
- exact molecular form
- frequency

Verdict

Copper exposure cannot be quantified from the current mL-only protocol.

FDA Injectable GHK-Cu Safety Warning

This deserves prominent placement.

FDA has specifically identified **injectable GHK-Cu** as presenting potential safety concerns.

The concerns include possible:

- immunogenicity
- peptide aggregation
- peptide-related impurities

FDA also notes that human safety information for injectable GHK-Cu is limited.

Important

This is not the same as saying:

> "GHK-Cu injections are proven dangerous."

It means the injectable route has unresolved product-quality and immune-response concerns that should not be ignored.

Production Wording

> **FDA has specifically identified injectable GHK-Cu as raising potential immunogenicity concerns related to aggregation and peptide impurities, with limited human safety data.**

Important GHK-Cu Regulatory Distinction

FDA's current compounding framework treats:

GHK-Cu except injectable routes

differently from:

injectable GHK-Cu.

Why?

Injectable formulations raise additional concerns involving:

- sterility
- aggregation
- impurities
- immunogenicity

Important

Do not simplify this into:

> "GHK-Cu is FDA approved."

It is not.

And do not simplify it into:

> "All GHK-Cu is banned."

That is also inaccurate.

Injectable Glutathione Product-Quality Risk

FDA has documented serious adverse events after improperly prepared compounded injectable glutathione.

Reported symptoms included:

- nausea
- vomiting
- lightheadedness
- chills
- body aches

One person developed:

- low blood pressure
- difficulty breathing

and required hospital transfer.

FDA testing identified:

excessive bacterial endotoxin

in the glutathione material involved.

Critical Distinction

These reactions show the danger of:

poor sterile-product quality

They should not automatically be described as:

normal pharmacologic side effects of glutathione itself.

Production Wording

> **FDA has documented endotoxin-related reactions from improperly sourced glutathione used in compounded sterile injections. Product quality is therefore a separate safety issue from glutathione's normal pharmacology.**

Safety at a Glance

Exact GGH Blend

Long-term safety is unknown

No robust clinical safety program was identified for the exact formulation.

Possible Local Effects

Community use may involve:

- injection-site discomfort
- redness
- irritation

Classification

Plausible / community reported

not established exact-blend clinical adverse effects.

Metallic Taste

Sometimes discussed around injectable products.

Classification

Community / uncertain

Do not list as a proven GGH adverse effect.

Lightheadedness

Classification

Community / uncertain

Not established as a characteristic GGH pharmacologic effect.

Headache

Classification

Nonspecific / uncertain

Should not be presented as an established GGH side effect.

Skin Lightening

If glutathione affects pigmentation, this would be better described as:

a possible cosmetic effect

rather than simply a side effect.

Evidence varies substantially by route.

Copper Accumulation

Theoretical concern if copper exposure becomes excessive.

Important

Risk cannot be estimated without knowing:

actual GHK-Cu exposure.

Formal Contraindications

None established through an FDA-approved GGH label

No approved GGH product exists.

Therefore the page should not create its own formal contraindication list.

Important Cautions

Copper Metabolism Disorders / Wilson Disease

GHK-Cu contains copper.

People with disorders of copper handling may require particular caution.

Classification

Important biological/clinical caution

Not:

formal GGH contraindication

Pregnancy / Breastfeeding

Safety of the exact blend is not established.

Classification

Safety unknown

Concurrent Copper-Altering Therapy

Could potentially affect interpretation of copper exposure.

Classification

Clinical caution

Active Malignancy

Do not call active cancer a formal contraindication based only on:

- tissue remodeling
- angiogenesis
- wound-healing biology

The biology is too complex to justify that conclusion.

Classification

Uncertain / theoretical caution if retained

not:

established contraindication

Commonly Paired With

BPC-157

Often discussed in recovery-oriented community stacks.

Verdict

Community combination

No controlled human GGH combination evidence.

TB-500

Also commonly paired in tissue-recovery communities.

Verdict

Community combination

No controlled human GGH combination evidence.

Additional Glutathione

This may create duplication if the GGH blend already contains glutathione.

Verdict

Check exact formula first

GHK-Cu Alone

Could also duplicate one of the blend's main components.

Verdict

Potential ingredient overlap

Important Research Note

Three very different evidence categories are being mixed together when people discuss GGH.

1. GHK-Cu Research

This includes legitimate research involving:

- skin
- collagen
- wound healing
- tissue remodeling

But much of the stronger human evidence is:

topical

rather than injectable.

---

2. Glutathione Research

Glutathione has strong biological evidence and separate human research.

But evidence differs between:

- oral
- topical
- IV
- other injection routes

---

3. GGH Blend Evidence

This is the weakest category.

The finished combination has not been shown to inherit every proven or proposed benefit of its individual ingredients.

Simple Rule

> **Component evidence is not combination evidence.**

Research at a Glance

Approved-Label Evidence

None

No FDA-approved GGH fixed-combination product exists.

Exact-Blend Human Research

Very limited / not established

No controlled exact-blend efficacy program was identified.

GHK-Cu Human Evidence

Limited but real

Strongest in topical and skin-related research.

Injectable GHK-Cu Evidence

Limited

Human safety and efficacy evidence remains much thinner than community use suggests.

Glutathione Human Evidence

Route dependent

Biological role is extremely well established.

Therapeutic evidence depends on:

- indication
- dose
- route
- formulation

Mechanistic Evidence

Moderate to strong for the individual components

Much weaker for predicting what happens when they are:

mixed and injected together.

Community Evidence

Primary source of exact GGH protocols

This is much weaker than clinical dose-finding evidence.

Overview

GGH is best understood as:

a community/vendor blend built from separately researched ingredients.

GHK-Cu has a legitimate scientific literature involving tissue and skin biology.

Glutathione has extremely well-established antioxidant and redox biology.

But the most important question is not:

> **Do GHK-Cu and glutathione each do something biologically?**

They clearly do.

The important question is:

> **Has combining them in this exact injectable formulation been shown to provide a safe and effective clinical benefit?**

At present:

that has not been established.

Evidence Strength

Overall Evidence Rating

Strong component biology, very weak exact-blend evidence

Stronger Evidence For

- glutathione's endogenous antioxidant role
- GHK-Cu tissue-remodeling biology
- topical GHK-Cu research
- chemical interaction between glutathione and Cu(II)-GHK
- injectable-product quality concerns

Limited Evidence For

- injectable GHK-Cu clinical benefit
- injectable glutathione for many wellness/cosmetic claims

Not Established

- exact GGH clinical efficacy
- optimal dose
- optimal frequency
- SC superiority
- IM superiority
- cycling
- receptor reset
- BPC-157 synergy
- TB-500 synergy
- same-vial long-term stability
- generic post-reconstitution expiration

Category

Recovery Blends • Skin / Tissue Research • Antioxidant Research

---

Compound

GGH

Important

GGH here refers to a:

GHK-Cu / glutathione-based vendor or community blend

not:

Gly-Gly-His

unless specifically stated otherwise.

Compound Type

Multi-ingredient research blend

Potential ingredients may include:

- GHK-Cu
- glutathione
- histidine
- glycine
- NADH

depending on the exact product.

Verdict

Formula-dependent

Primary Research Focus

Community GGH products are generally marketed around:

- recovery
- skin
- antioxidant support
- tissue repair
- general regeneration

Evidence Classification

These are primarily:

community/vendor research goals

rather than established indications for the finished blend.

Common Research Dose

Cannot be established without the exact formula

The previous:

0.5–1 mL

figure should not be described as a meaningful dose on its own.

Correct Production Wording

> **Community protocols may describe 0.5–1 mL per injection, but the actual dose depends entirely on the concentration of each active ingredient. No clinically validated GGH dose has been established.**

Research Duration / Cycle Length

Clinical

Not established

Community

Variable.

Biological Requirement for Cycling

None demonstrated

How Often It Is Used

Clinical

Not established

Community

Often reported several times per week.

Evidence Assessment

Community practice only

Community Protocol Comparison

Protocol	Dose	Route	Frequency	Evidence
Current/older GGH page	0.5–1 mL	SC / IM	2–3x weekly	Community/vendor
Standalone GHK-Cu community protocols	Usually expressed in mg	SC	Variable	Community
Exact GGH human clinical protocol	None established	N/A	N/A	No validated protocol identified

Biggest Problem

0.5–1 mL cannot be compared across formulations without knowing mg/mL.

Formulation / Stability / Sterility

No Standardized GGH Formulation Exists

There is no approved product defining one universal:

- ingredient list
- ingredient ratio
- concentration
- diluent
- pH
- reconstitution method
- storage temperature
- post-reconstitution expiration

Same-Vial Stability

Because GHK-Cu and glutathione can chemically interact:

same-vial stability should not be assumed.

The exact formulation may depend on factors including:

- pH
- excipients
- ingredient ratios
- oxygen exposure

- temperature
- storage time
- copper oxidation state

Verdict

Exact formulation data required

Generic 14 / 28-Day Rules

Do not use them

There is no universal evidence showing every GGH vial remains:

- chemically stable
- sterile
- potent

for:

14 days

or:

28 days

after reconstitution.

Simple Rule

Use stability information for the exact formulation and product.

Regulatory Status

FDA-Approved GGH Product?

No

Injectable GHK-Cu

FDA has specifically raised safety concerns involving injectable compounded GHK-Cu.

Glutathione

Glutathione has separate compounding and sterile-product considerations.

Exact GGH Blend

No approved fixed-combination product identified

Component regulatory status does not automatically determine:

finished-blend status.

What Studies Have Found

GHK-Cu Research

Research supports biological roles involving:

- tissue remodeling
- extracellular matrix
- collagen
- wound biology
- inflammation
- oxidative stress

What This Supports

GHK-Cu is a legitimate biologically active compound.

What It Does Not Support

A specific:

GGH injection protocol

Glutathione Research

Glutathione is essential to normal redox biology.

Human research also exists around:

- supplementation
- oxidative stress
- pigmentation

What This Supports

Glutathione has important biological activity.

What It Does Not Support

That:

injecting glutathione as part of GGH creates a proven recovery or anti-aging treatment.

GHK-Cu + Glutathione Chemistry

Laboratory research directly demonstrates interaction between:

Cu(II)-GHK

and:

glutathione

What This Supports

The ingredients can chemically affect each other.

What It Does Not Support

That every finished GGH formulation:

- rapidly degrades
- is ineffective
- is unsafe

Correct Interpretation

Compatibility must be demonstrated, not assumed.

Research Sources

Use clean canonical URLs in production.

1. Current EvidenceCodex GGH Page

Evidence type: Current-page audit source

`https://evidencecodex.pages.dev/library/ghk-cu-glutathione-ggh`

---

2. FDA: Certain Bulk Drug Substances That May Present Significant Safety Risks

Evidence type: FDA safety source

Supports:

- injectable GHK-Cu concerns
- immunogenicity
- aggregation
- peptide-related impurities
- limited human safety information

https://www.fda.gov/drugs/human-drug-compounding/certain-bulk-drug-substances-use-compounding

---

3. FDA 503A Bulk Drug Substances Update

Evidence type: FDA regulatory source

Supports current distinctions involving:

- GHK-Cu
- injectable GHK-Cu
- glutathione

Use the current FDA 503A bulk-substances document in production.

---

4. FDA: Injectable Glutathione Sterile-Compounding Alert

Evidence type: FDA product-quality safety source

Supports:

- endotoxin contamination
- nausea
- vomiting
- lightheadedness
- chills
- body aches
- hypotension
- breathing difficulty

and the distinction between:

contamination risk

and:

glutathione pharmacology

https://www.fda.gov/drugs/human-drug-compounding/fda-highlights-concerns-using-dietary-ingred

---

5. Ufnalska et al. 2021

Intermediate Cu(II)-Thiolate Species in the Reduction of Cu(II)GHK by Glutathione

Evidence type: Laboratory chemistry research

Supports:

- direct chemical interaction between Cu(II)-GHK and glutathione
- copper reduction chemistry
- need to avoid assuming inert same-vial compatibility

`https://pubmed.ncbi.nlm.nih.gov/34781677/`

---

6. Pickart et al.

Regenerative and Protective Actions of the GHK-Cu Peptide

Evidence type: GHK-Cu biological review

Supports:

- tissue-remodeling biology
- wound-related mechanisms
- collagen / extracellular-matrix rationale

`https://pubmed.ncbi.nlm.nih.gov/29986520/`

---

7. GHK-Cu / GHK Anti-Aging Review

Evidence type: Biological/mechanistic review

Useful for separating:

GHK-Cu biology

from:

exact-blend evidence

`https://pubmed.ncbi.nlm.nih.gov/35083444/`

---

8. PeptideDosingProtocols: GHK-Cu

Evidence type: Community / evidence-reconciliation source

Useful for documenting:

- current GHK-Cu community practice
- topical versus injectable evidence distinction

https://www.peptidedosingprotocols.com/protocol/ghk-cu

---

9. PeptideDosages: GHK-Cu

Evidence type: Community dosing source

Useful for standalone GHK-Cu community dose examples.

It does **not** validate the exact GGH blend.

https://peptidedosages.com/single-peptide-dosages/ghk-cu-100-mg-vial-dosage-protocol/

---

10. Glutathione Skin-Lightening Systematic Review

Evidence type: Human evidence review

Useful for separating:

- oral
- topical
- IV

glutathione evidence.

https://pubmed.ncbi.nlm.nih.gov/39444151/

---

11. PeptidePlotter: GGH

Evidence type: Community/vendor blend documentation

Useful for documenting that one current product called GGH contains:

- GHK-Cu
- glutathione
- histidine
- glycine
- NADH

It is not clinical efficacy evidence.

https://peptideplotter.com/compounds/ggh

Final Takeaway

GGH should not be presented as a standardized injectable therapy.

It is a blend name used in the research/community market.

Its ingredients may have legitimate biological research individually, especially:

GHK-Cu

and:

glutathione

But there is a major difference between:

> **"These ingredients each have biological effects."**

and:

> **"This exact combined injection has been clinically shown to work."**

The second claim has not been established.

The biggest production rules are:

> **Do not call 0.5–1 mL a meaningful dose unless the exact mg/mL of every active ingredient is shown.**
>
> **Replace "Standard Protocol" with "Community / Vendor Protocol Example."**
>
> **Do not treat separate GHK-Cu or glutathione studies as clinical evidence for GGH.**
>
> **Clearly distinguish the commercial GGH blend from Gly-Gly-His, which is also abbreviated GGH in scientific literature.**
>
> **Keep the GHK-Cu + glutathione chemical-interaction warning, but do not claim this proves the finished blend is unstable or unsafe.**
>
> **Prominently include FDA's injectable GHK-Cu concerns involving immunogenicity, aggregation and peptide impurities.**
>
> **Keep endotoxin-contaminated glutathione reactions under product-quality risk rather than presenting them as normal glutathione side effects.**
>
> **Use "Commonly Paired With," not "synergistic," for BPC-157, TB-500 or similar compounds.**
>
> **Do not create formal GGH contraindications when no approved GGH label exists.**
>
> **Do not use generic 14-day or 28-day stability rules. The exact formulation needs its own evidence.**

This version is now the one I would consider **publish-ready**, with one remaining product-specific field to fill whenever you know the actual Navaro GGH formulation: the **exact ingredient amounts/concentrations**.

GLOW Audit

I audited GLOW against the current live Navaro page and the same source hierarchy we have been using for the other compounds: FDA, PubMed, ClinicalTrials.gov/current human literature, plus PeptideFox and PeptideDosages for community-protocol comparison.

This page needs a major revision before production. The current page has a good skeptical disclaimer, but several of the actual protocol, safety, and mechanism statements underneath it are much more confident than the evidence supports. (navarohealth.com)

Current Page

GLOW (BPC-157 + TB-500 + GHK-Cu)

Current live Navaro page:

```
https://navarohealth.com/library/glow-blend
```

The page currently describes GLOW as an aesthetics/repair blend and lists:

Route: Subcutaneous Typical dose: 2.8 mg Timing: Anytime Targets: VEGFR2; G-Actin; MMP. (navarohealth.com)

It gives a “Standard Protocol” of:

2.8 mg daily

for:

12 weeks

with:

5 days on / 2 days off

and says this schedule is used:

> “to prevent copper toxicity.” (navarohealth.com)

It also gives a lower-dose 1.4 mg daily protocol and makes several strong claims about zinc depletion, copper accumulation, dopamine effects, cancer risk, liver enzymes, and mandatory supplementation. (navarohealth.com)

Overall Verdict

Major revision needed

The biggest problems are:

- GLOW is not a standardized clinical product
- the exact product identity, particularly TB-500 versus full-length thymosin β4, must be clarified
- no human clinical trial validates the exact fixed-ratio GLOW blend
- “Standard Protocol” is too strong
- 2.8 mg daily is a community protocol, not a clinical dose
- the 5-days-on / 2-days-off rule has no demonstrated copper-toxicity rationale
- the claim that injected GHK-Cu progressively depletes zinc through intestinal absorption competition is not supported and is mechanistically mismatched to SC administration
- mandatory 15–30 mg/day zinc supplementation is not justified

- “receptor resetting” is not supported
- several listed side effects are speculative community claims
- “active cancer = absolute contraindication” is too strong
- full-length thymosin β4 human evidence cannot be transferred automatically to TB-500 fragment LKKTETQ
- generic 4-week post-reconstitution stability is not validated for every GLOW formulation
- same-vial compatibility of the three ingredients has not been clinically established
- FDA currently has significant safety concerns involving all three injectable components in the way these community blends are marketed. (fda.gov)

---

1. First Problem: What Exactly Is in “GLOW”?

The current page calls the blend:

BPC-157 + TB-500 + GHK-Cu. (navarohealth.com)

A common 2026 community formulation is:

GHK-Cu: 50 mg BPC-157: 10 mg Thymosin β4 / “TB” component: 10 mg

for:

70 mg total

or a:

5 : 1 : 1 mass ratio.

PeptideFox currently describes its GLOW formulation specifically as 50 mg GHK-Cu + 10 mg BPC-157 + 10 mg full-length thymosin β4, and explicitly says it means the full 43-amino-acid thymosin β4 molecule rather than the shorter TB-500 fragment. (peptidefox.com)

PeptideDosages, however, describes a 70 mg formulation as:

GHK-Cu + BPC-157 + TB-500. (peptidedosages.com)

This distinction matters enormously.

“GLOW” is therefore a:

community/vendor formulation name

not one scientifically standardized fixed-dose drug.

Production requirement

Near the top of the page, identify the exact formulation:

GHK-Cu: ___ mg BPC-157: ___ mg Third peptide: ___ mg Exact identity of third peptide: full-length thymosin β4 OR TB-500 fragment

Do not leave that last part ambiguous.

---

2. TB-500 and Full-Length Thymosin β4 Are Not Automatically the Same Thing

This is probably the most important scientific source-matching issue on the page.

FDA currently defines:

TB-500

in its compounding review as:

thymosin β4 fragment LKKTETQ.

FDA states that it has not identified human exposure data for drug products containing this thymosin β4 fragment. (fda.gov)

Full-length thymosin β4 is a different molecule.

There is human clinical research involving full-length thymosin β4, including wound-healing research. Phase 2 studies have investigated the full-length peptide in patients with difficult-to-heal wounds. (pubmed.ncbi.nlm.nih.gov)

Therefore:

If the GLOW vial contains:

full-length thymosin β4

then full-length Tβ4 literature may be relevant as component evidence.

If the vial contains:

TB-500 / LKKTETQ

then those full-length human trials cannot simply be assigned to it.

Production wording

> “TB-500” is often used loosely online, but FDA uses the name for the shorter thymosin β4 fragment LKKTETQ. Full-length thymosin β4 has separate research and should not automatically be treated as the same administered compound. Confirm the actual sequence in the GLOW formulation.

This needs to be prominent.

---

3. No Human Clinical Trial Validates the Exact GLOW Blend

The existing page actually acknowledges this in its evidence section, which is good. It says component evidence does not prove combined safety, compatibility, concentration, or route. (navarohealth.com)

That principle needs to control the rest of the page.

The exact:

GHK-Cu + BPC-157 + thymosin β4/TB-500

fixed-ratio injectable formulation does not have a matching approved label or established human efficacy program.

Even PeptideFox's current GLOW comparison page explicitly states that no controlled study has tested GLOW as a combined fixed-ratio intervention. (peptidefox.com)

Evidence rating

Exact GLOW blend human efficacy: Not established Dose-finding: Not established Long-term safety: Not established Human PK: Not established Optimal frequency: Not established Same-vial compatibility: Not established Validated cosmetic efficacy: Not established

---

4. The Current “Typical Dose 2.8 mg” Is Community Dosing

The 2.8 mg number is mathematically coherent if the vial actually contains the 50/10/10 formulation.

At that ratio:

2.8 mg total blend

contains approximately:

- 2 mg GHK-Cu
- 400 mcg BPC-157
- 400 mcg third peptide

That matches the current Navaro page and PeptideFox's standard community dose. (navarohealth.com)

But that only proves:

the blend math works.

It does not prove:

2.8 mg is clinically optimal.

PeptideDosages uses a different community protocol:

2.33 mg daily

which provides approximately:

- 1.7 mg GHK-Cu
- 330 mcg BPC-157
- 330 mcg TB-500

for four weeks. (peptidedosages.com)

Verdict

Change:

> Typical dose: 2.8 mg

to something like:

> Common community dose example: 2.8 mg total blend, formulation-dependent

And always display the component breakdown.

---

5. “Standard Protocol” Is Too Strong

Current Navaro:

2.8 mg daily, 5 days on / 2 days off for 12 weeks. (navarohealth.com)

PeptideFox currently uses a different phased community framework:

- Weeks 1–4: daily
- Weeks 5–8: 5x/week
- Weeks 9–12: 2–3x/week. (peptidefox.com)

PeptideDosages uses:

- daily for 4 weeks
- then 2–4 weeks off. (peptidedosages.com)

That disagreement is informative.

There is no standardized GLOW protocol.

Correct heading

Replace:

Standard Protocol

with:

Community Protocol Examples

Then compare them honestly.

---

6. The “5 Days On / 2 Days Off Prevents Copper Toxicity” Claim Should Be Removed

The current page says the weekend break is:

> “strictly” required to prevent copper toxicity. (navarohealth.com)

I found no clinical evidence establishing that:

five days of GLOW followed by two days off prevents copper accumulation or toxicity.

There is no demonstrated copper threshold that suddenly resets over a weekend.

Verdict

Unsupported biological rationale

The schedule may be a community convention.

It should not be presented as a toxicity-prevention mechanism.

Better wording

> Some community protocols use 5 days on / 2 days off, but no human study has demonstrated that this schedule is necessary to prevent copper toxicity.

---

7. The Zinc-Depletion Section Is One of the Biggest Errors on the Current Page

The page currently says injectable GHK-Cu:

- competes with zinc for intestinal absorption
- progressively depletes zinc
- makes zinc deficiency a contraindication
- requires 15–30 mg/day zinc supplementation. (navarohealth.com)

That mechanism is backwards and route-mismatched.

The established nutritional interaction is:

> high zinc intake can interfere with copper absorption in the intestine.

NIH specifically warns that prolonged high-dose zinc can produce copper deficiency. (ods.od.nih.gov)

That is not the same thing as:

> injected copper causes zinc deficiency by outcompeting intestinal zinc absorption.

An SC injection also does not pass through the intestinal absorption step in the first place.

Major correction

Remove:

- “zinc deficiency is a contraindication”
- “GHK-Cu progressively depletes zinc”
- “mandatory zinc supplementation”
- “15–30 mg zinc required throughout all cycles”

unless there is direct clinical evidence for the exact claim.

Important safety point

Unnecessary zinc supplementation itself is not harmless. NIH notes that sustained high-dose zinc can impair copper absorption and reduce copper status. (ods.od.nih.gov)

Verdict

Current zinc logic should be removed.

---

8. Copper Exposure Still Matters, but It Needs to Be Discussed Correctly

GHK-Cu does contain copper.

Therefore repeated systemic exposure can reasonably raise questions about:

- total copper exposure
- copper metabolism disorders
- hepatic handling

- exact dose

But the current page jumps too quickly from that fact to specific toxicity-prevention schedules and mandatory zinc therapy.

Better approach

For the exact product, document:

- mg GHK-Cu per injection
- estimated copper content
- injection frequency
- duration
- other copper sources

Then state:

> The clinical significance of chronic systemic copper exposure from injectable GHK-Cu has not been well established.

That is more accurate.

---

9. FDA's Injectable GHK-Cu Warning Needs to Be Much More Prominent

FDA currently states:

> compounded injectable GHK-Cu may pose immunogenicity risk because of potential aggregation and peptide-related impurities.

FDA also says human data are limited. (fda.gov)

The 2026 503A update makes an important route distinction:

GHK-Cu except injectable routes

is currently being evaluated separately, while injectable GHK-Cu carries the safety concerns above. (fda.gov)

Production wording

> FDA has specifically identified compounded injectable GHK-Cu as raising potential immunogenicity concerns involving aggregation and peptide-related impurities, with limited human safety data.

This belongs near the top safety summary.

---

10. BPC-157 Evidence Is Mostly Preclinical

BPC-157 has a substantial animal and mechanistic literature.

Recent reviews describe effects involving:

- angiogenesis
- fibroblast activity

- nitric oxide signaling
- tendon and muscle repair
- inflammatory pathways. (pubmed.ncbi.nlm.nih.gov)

But human evidence remains extremely small.

A 2025 pilot IV study administered BPC-157 to only:

two healthy adults

at doses up to:

20 mg IV

and reported no adverse effects in those two participants. (pubmed.ncbi.nlm.nih.gov)

That is interesting early human exposure.

It is nowhere near enough to establish:

- daily 400 mcg SC efficacy
- cosmetic skin benefits
- chronic safety
- inclusion in GLOW

FDA continues to say it has only limited safety-related information for proposed BPC-157 routes and highlights possible immunogenicity and characterization concerns. (fda.gov)

Evidence classification

Preclinical: substantial Human: extremely limited Injected skin/aesthetic efficacy: unproven GLOW combination efficacy: unproven

---

11. The BPC-157 “Capillary Supply” Story Is Mechanistically Plausible but Overstated

PeptideFox says BPC-157:

> “sprouts new small blood vessels into the dermal repair area.” (peptidefox.com)

Preclinical evidence does support angiogenic signaling involving VEGFR2 and nitric-oxide pathways. (pubmed.ncbi.nlm.nih.gov)

But there is no human GLOW trial demonstrating that injected BPC-157:

- increases dermal capillary supply
- improves skin collagen because of that effect
- improves GHK-Cu efficacy

Better wording

> BPC-157 has angiogenesis and vascular-repair effects in preclinical models, which provides a theoretical reason for including it in the blend. Whether that improves human skin outcomes in GLOW has not been demonstrated.

---

12. GHK-Cu Evidence Is Real, but Mostly Not Injectable GLOW Evidence

GHK-Cu has substantial research involving:

- tissue remodeling
- wound biology
- collagen
- extracellular matrix
- antioxidant signaling. (pubmed.ncbi.nlm.nih.gov)

But human evidence is much stronger for:

topical exposure

than community SC injection protocols.

Even a human topical study following laser resurfacing did not show every proposed benefit, which is a useful reminder not to oversell the component. (pubmed.ncbi.nlm.nih.gov)

Verdict

GHK-Cu component evidence belongs on the page.

But the page must repeatedly distinguish:

GHK-Cu biology

from:

injected GLOW efficacy.

---

13. Full-Length Thymosin β4 Has Human Research

Unlike TB-500 fragment LKKTETQ, full-length thymosin β4 does have human wound research.

Clinical work has included difficult-to-heal:

- pressure ulcers
- stasis ulcers
- other wound settings

and has reported accelerated healing in some studies. (pubmed.ncbi.nlm.nih.gov)

But:

That does not validate:

- 400 mcg SC daily
- cosmetic skin use
- the GLOW ratio
- 12-week GLOW cycles

Evidence verdict

Full-length Tβ4 has human component evidence.

GLOW does not inherit a clinical protocol from those trials.

---

14. FDA's TB-500 Review Is a Major 2026 Update

FDA's 2026 review of TB-500-related bulk substances is highly relevant.

FDA defines the substance under review as the:

LKKTETQ thymosin β4 fragment

and concluded that available information was insufficient to establish human safety and effectiveness.

FDA proposed that:

- TB-500 free base
- TB-500 acetate

not be included on the 503A Bulks List. (fda.gov)

The July 23, 2026 PCAC meeting considered both BPC-157 and TB-500. Advisory committee recommendations are nonbinding, and FDA rulemaking/status should not be described as final approval simply because a committee considered or voted on a substance. (fda.gov)

Production rule

Do not say:

> “FDA approved compounded TB-500.”

Do not say:

> “TB-500 is FDA approved because PCAC recommended it.”

It is not.

---

15. The Exact Thymosin Species Must Match the Evidence

This deserves its own Protocol Logic Check row.

If Navaro's blend actually contains:

TB-500 fragment LKKTETQ

then:

full-length Tβ4 human wound studies ≠ TB-500 human evidence.

If it contains:

full-length thymosin β4

then FDA's “no identified human exposure” statement for the LKKTETQ fragment should not be falsely applied as though it describes full-length Tβ4.

Verdict

Exact sequence/form must be confirmed before assigning evidence.

---

16. “Receptor Resetting” Should Be Removed

PeptideDosages says its 4-week schedule is followed by 2–4 weeks off to:

> “allow receptor resetting.” (peptidedosages.com)

There is no established single:

GLOW receptor

to reset.

The blend contains mechanistically different compounds:

- GHK-Cu
- BPC-157
- thymosin β4/TB-500

None creates a demonstrated clinical need for a scheduled 2–4 week receptor reset.

Verdict

Unsupported

---

17. There Is No Evidence-Based 8- or 12-Week Cycle

Current Navaro uses:

12 weeks. (navarohealth.com)

PeptideFox uses:

12 weeks. (peptidefox.com)

PeptideDosages uses:

4 weeks. (peptidedosages.com)

That difference itself tells us:

the cycle is community convention, not biologically established.

Verdict

Do not claim:

- 4 weeks is required
- 8 weeks is required
- 12 weeks is required
- weekend breaks are required
- washouts are required

---

18. Daily Use Is Community-Derived

Daily administration is common across community GLOW protocols.

But there is no exact-blend human dose-frequency trial establishing that daily administration is superior to:

- 5x/week
- 3x/week
- another schedule

Verdict

Community precedent

not:

clinically optimized frequency

---

19. Morning / Evening / Fasted / Exercise Timing

There is no controlled evidence showing GLOW should be administered:

- morning
- evening
- fasted
- fed
- before exercise
- after exercise

Current Navaro's:

Anytime

is actually acceptable as long as it is understood to mean:

no timing advantage has been demonstrated. (navarohealth.com)

---

20. “Dopamine Buffer” Should Be Removed

The current profile includes:

Properties: Dopamine Buffer. (navarohealth.com)

That is not a clinically recognized pharmacological classification for GLOW.

The page then attributes:

- lethargy
- sedation
- anhedonia

to dopaminergic effects of BPC-157/TB-500. (navarohealth.com)

BPC-157 has experimental literature involving several neurotransmitter systems, but that does not establish:

GLOW as a dopamine-buffering treatment

or make sedation/anhedonia established blend adverse effects.

Verdict

Remove:

Dopamine Buffer

from the compound profile.

---

21. Several Current Side Effects Are Overstated

The current page lists things such as:

- sedation
- lethargy
- progressive zinc depletion
- copper accumulation
- headache
- elevated ALT/AST
- temporary inflammation
- anhedonia. (navarohealth.com)

For the exact GLOW blend, there is no controlled safety dataset establishing that profile.

Better classifications

Injection-site pain / irritation: Plausible and community reported

Nausea: Possible but not established as a characteristic exact-blend effect

Headache: Nonspecific/community reported

Fatigue: Community/uncertain

Anhedonia: Community/uncertain, not established GLOW toxicity

Elevated liver enzymes: Not established as a normal expected GLOW adverse effect

Zinc depletion: Remove as currently framed

Copper toxicity: Theoretical exposure concern requiring actual dose/formulation context

---

22. “The Blend Was Designed to Reduce GHK-Cu Injection Pain” Is Not Established

The current page says the blend's:

> “primary design purpose”

is to reduce GHK-Cu injection pain. (navarohealth.com)

I found no clinical formulation study demonstrating that adding BPC-157 and thymosin β4:

- lowers GHK-Cu injection pain
- changes pH enough to improve tolerance
- produces a safer formulation

Verdict

Remove unless a formulation-specific source exists.

---

23. Active Cancer Should Not Be an “Absolute Contraindication”

The existing page gives an extremely confident explanation that BPC-157 angiogenesis and GHK-Cu growth signaling would accelerate tumor growth and calls active malignancy an:

absolute contraindication. (navarohealth.com)

That is too strong.

Mechanistic concerns around:

- angiogenesis
- tissue remodeling
- growth signaling

can reasonably justify caution.

But there is no approved GLOW label or clinical oncology dataset establishing:

active malignancy as a formal absolute contraindication.

Correct classification

Theoretical / clinically important caution

not:

formal contraindication

---

24. Wilson Disease / Copper-Metabolism Disorders

This one deserves more respect than the cancer statement because the blend contains a copper complex.

Wilson disease causes abnormal copper accumulation.

Adding a systemic copper-containing experimental product would raise obvious clinical concerns.

But:

There is still no FDA-approved GLOW label.

Classification

Strong biological caution

rather than pretending it is an official GLOW-label contraindication.

---

25. Pregnancy / Breastfeeding

No good exact-blend pregnancy safety data exist.

Classification

Safety unknown / avoid extrapolation

not a formal FDA GLOW contraindication because no such label exists.

---

26. “Severe IBD Flare” Claims Need Removal

The current page gives detailed instructions about GLOW being acceptable for:

> remission maintenance

but inappropriate for acute severe IBD. (navarohealth.com)

There is no controlled human GLOW program establishing:

- treatment of IBD remission
- flare management
- GLOW maintenance therapy

Verdict

Remove this disease-management language.

It exceeds the evidence by a large margin.

---

27. Combination “Synergy” Is Unproven

The current page lists:

- Ipamorelin
- AOD-9604
- Matrixyl
- SNAP-8
- KPV

as “Reported synergistic.” (navarohealth.com)

No controlled evidence establishes those GLOW combinations as synergistic.

Replace with

Commonly Paired With

Then classify each combination.

For example:

KPV: adds a different anti-inflammatory mechanism in community KLOW formulations, but no human GLOW-vs-KLOW trial exists. (peptidefox.com)

Matrixyl / SNAP-8: topical cosmetic peptides; different route/context; no clinical injectable-GLOW synergy established.

Ipamorelin: unrelated GH-secretagogue pathway; no validated GLOW combination.

AOD-9604: unrelated metabolic/fat-loss research context; no demonstrated synergy.

---

28. Duplicate-Ingredient Stacking Should Be Flagged

Adding separate:

- BPC-157
- TB-500/Tβ4
- GHK-Cu
- KLOW

on top of GLOW could duplicate ingredients.

The current page correctly flags some of these as incompatible, but “contraindicated” is probably too strong.

Better label

Ingredient overlap / redundancy

Because the problem is primarily:

you are doubling components whose actual combined exposure may be unclear.

---

29. Same-Vial Compatibility Is Not Established

Even if all three compounds have separate stability data, that does not prove:

the three-peptide mixture remains chemically and physically stable together.

Blend stability depends on factors such as:

- pH
- excipients
- concentration
- oxidation
- temperature
- storage time
- peptide adsorption

- aggregation

There is no FDA-approved reference GLOW formulation.

Verdict

Formulation-specific compatibility data required

---

30. Generic Four-Week Stability Should Not Be Used as Clinical Fact

PeptideDosages says reconstituted GLOW should be refrigerated and used within:

4 weeks. (peptidedosages.com)

That is community product-handling guidance.

It is not universal clinical stability evidence for every GLOW formulation.

Correct wording

> Some community sources use a four-week refrigerated window, but no universal validated post-reconstitution stability period exists for all GLOW formulations. Follow exact formulation-specific stability information.

---

31. “Pre-Mixed: No” Is Inconsistent With the Way GLOW Is Actually Defined

Current Navaro says:

Pre-mixed: No. (navarohealth.com)

But GLOW is generally discussed in current community sources specifically as a:

single-vial fixed-ratio blend.

PeptideFox explicitly calls it a pre-mixed 70 mg blend. (peptidefox.com)

Verdict

If Navaro means the GLOW entry itself:

Pre-mixed should probably be Yes / formulation-dependent.

If the site means users could assemble the components separately, that should be explained instead.

---

32. FDA 2026 Regulatory Picture

This needs a much stronger section.

BPC-157

FDA says compounded BPC-157 may raise:

- immunogenicity

- peptide impurity
- API-characterization

concerns and says it has limited safety information for proposed routes. (fda.gov)

In July 2026, FDA evaluated BPC-157 free base and acetate for possible inclusion on the 503A Bulks List and proposed against inclusion in its briefing materials. (fda.gov)

TB-500

FDA's current review defines TB-500 as:

thymosin β4 fragment LKKTETQ

and states it has not identified human exposure data for drug products containing that fragment. (fda.gov)

FDA's July 2026 evaluation concluded that information on:

- chemical characterization
- clinical safety
- effectiveness

was insufficient and proposed that TB-500 free base and acetate not be placed on the 503A Bulks List. (fda.gov)

GHK-Cu

For injectable GHK-Cu, FDA highlights:

- possible immunogenicity
- aggregation
- peptide-related impurities
- limited human safety information. (fda.gov)

Noninjectable GHK-Cu is currently being evaluated separately under FDA's 503A framework. (fda.gov)

Exact GLOW Blend

FDA approved?

No

Approved fixed-ratio GLOW product?

No

FDA-approved dosing?

No

FDA-approved cosmetic indication?

No

Protocol Logic Check

Question	Verdict
Is GLOW one standardized approved drug?	No
Common 50/10/10 mg formulation documented in community sources?	Yes
Is every GLOW vial necessarily 50/10/10?	No
Exact third peptide must be confirmed?	Yes
Is full-length Tβ4 the same as TB-500 LKKTETQ?	No
Human full-length thymosin β4 research exists?	Yes
FDA identified human exposure data for TB-500 fragment?	No
Human trial of exact GLOW blend found?	No
2.8 mg blend dose mathematically coherent for 50/10/10?	Yes
2.8 mg clinically validated?	No
Daily dosing clinically validated?	No
5 days on / 2 days off clinically validated?	No
Does weekend skipping prevent copper toxicity?	Not established
Is a 12-week cycle required?	No
Does GLOW need receptor resetting?	No evidence
Does injected GHK-Cu cause zinc deficiency through intestinal competition?	Unsupported / route-mismatched
Is mandatory 15–30 mg zinc established?	No
Does GHK-Cu have legitimate component evidence?	Yes
Is human GHK-Cu evidence stronger topically?	Yes
Does BPC-157 have strong human efficacy evidence?	No
Does full-length Tβ4 have some human wound evidence?	Yes
Does that validate injected GLOW for skin?	No
Is “Dopamine Buffer” a validated GLOW property?	No
Is active cancer an official absolute contraindication?	No
Copper disorders warrant caution?	Yes
Exact-blend same-vial stability established?	No
Universal 28-day stability established?	No
FDA approved?	No

Community Protocol Comparison

Current Navaro

2.8 mg total

providing:

- 2 mg GHK-Cu

- 400 mcg BPC-157
- 400 mcg third peptide

daily, with:

5 days on / 2 days off

for:

12 weeks. (navarohealth.com)

Classification

Community protocol

The component math is valid for a 5:1:1 blend.

The cycle logic is not clinically validated.

PeptideFox

PeptideFox describes a 70 mg blend containing:

- 50 mg GHK-Cu
- 10 mg BPC-157
- 10 mg full-length thymosin β4

with a standard:

2.8 mg total dose

and a phased 12-week schedule:

Weeks 1–4: daily Weeks 5–8: 5x/week Weeks 9–12: 2–3x/week. (peptidefox.com)

Important

PeptideFox itself states:

no controlled GLOW trial establishes the results timeline and the complete blend has not been clinically tested. (peptidefox.com)

Classification

Community/practitioner protocol

PeptideDosages

PeptideDosages describes:

2.33 mg SC daily

for:

4 weeks

followed by:

2–4 weeks off.

It uses a 70 mg formulation and calculates roughly:

- 1.7 mg GHK-Cu
- 330 mcg BPC-157
- 330 mcg TB-500

per injection. (peptidedosages.com)

It explicitly invokes:

receptor resetting

for the break. (peptidedosages.com)

Audit verdict

The dose is useful as a community protocol example.

The receptor-reset claim should not be carried into Navaro.

Strongest Community Agreement

The sources agree fairly well on:

- approximately 50/10/10 formulation
- subcutaneous community use
- daily or near-daily early use
- GHK-Cu as the largest component
- skin/tissue-remodeling positioning

They do not agree on:

- exact daily dose
- frequency
- cycle length
- maintenance schedule
- washout
- third-peptide nomenclature
- storage period

That is exactly why Navaro should not call one schedule:

the standard clinical protocol.

Recommended Evidence Ladder

Exact GLOW blend: Very limited Human clinical dose for GLOW: Not established Human efficacy for injected GLOW: Not established GHK-Cu biology: Moderate/strong mechanistic and preclinical evidence Human topical GHK-Cu: Limited but real Injectable GHK-Cu: Limited, with FDA safety concerns BPC-157 preclinical: Substantial BPC-157 human research: Extremely limited Full-length thymosin β4 human evidence: Limited but real, including wound research TB-500 LKKTETQ human exposure evidence: FDA says none identified Community GLOW protocols: Common Same-vial compatibility: Not established FDA-approved GLOW: No

---

What the Current Page Gets Right

I would keep these concepts:

- exact blend is not independently clinically validated
- evidence strength should remain very limited
- component studies do not prove the combination
- subcutaneous use is community/research context
- no FDA-approved regimen exists
- long-term data are limited
- blend compatibility requires caution. (navarohealth.com)

The page's foundational skepticism is good.

The problem is that the later protocol and safety sections then become much more definitive than that evidence rating allows.

What I Would Remove

Remove:

> “strictly 5 days on / 2 days off to prevent copper toxicity.”

Remove:

> “daily GHK-Cu progressively depletes zinc.”

Remove:

> “zinc supplementation 15–30 mg/day is required.”

Remove:

> “zinc deficiency is a contraindication.”

Remove:

> “active cancer is absolutely contraindicated because BPC-157 would accelerate tumor growth.”

Replace with a cautious mechanistic discussion.

Remove:

> “Dopamine Buffer.”

Remove established-sounding claims that:

- GLOW causes anhedonia
- GLOW predictably causes sedation
- GLOW commonly raises ALT/AST
- weekend breaks prevent copper toxicity
- cycle limits prevent copper accumulation
- the blend was specifically engineered to reduce GHK-Cu injection pain

unless exact supporting evidence is available.

Remove:

> receptor reset

from any protocol.

What Needs to Be Added

Exact Formula / Identity Box

> GLOW is not a standardized drug name. Confirm the amount and exact identity of all three components. In particular, determine whether the third ingredient is full-length thymosin β4 or the shorter LKKTETQ fragment commonly called TB-500.

TB-500 vs Thymosin β4 Warning

> Full-length thymosin β4 and TB-500 fragment LKKTETQ are not interchangeable evidence sources. Human studies of full-length thymosin β4 should not be cited as proof of efficacy or safety for the TB-500 fragment.

Dose Classification

> 2.8 mg is a common community dose for a 50/10/10 mg GLOW formulation, not a clinically validated treatment dose.

Zinc Correction

> The established nutritional interaction runs mainly in the opposite direction: high oral zinc can reduce intestinal copper absorption. There is no established evidence that SC GHK-Cu requires routine zinc supplementation because it “outcompetes zinc” in the intestine. (ods.od.nih.gov)

FDA Component Warning

> All three components require regulatory caution. FDA has raised immunogenicity/characterization concerns for BPC-157 and injectable GHK-Cu, while FDA's TB-500 review found no identified human exposure data for the LKKTETQ fragment. (fda.gov)

Sources I Would Use for the Production Page

1. Current Navaro GLOW Page

For auditing the existing claims. (navarohealth.com)

2. FDA Significant Safety Risks Page

Primary regulatory source for:

- BPC-157
- injectable GHK-Cu
- TB-500 LKKTETQ

safety/evidence limitations. (fda.gov)

3. FDA July 2026 BPC-157 / TB-500 PCAC Materials

For current 503A regulatory evaluation.

FDA proposed against including BPC-157 and TB-500-related substances in its briefing materials; the advisory process itself should not be confused with drug approval. (fda.gov)

4. FDA May 2026 503A Update

For the important injectable versus noninjectable GHK-Cu distinction. (fda.gov)

5. BPC-157 2025 Human IV Pilot

For the very small amount of direct human exposure data.

Only two participants were included. (pubmed.ncbi.nlm.nih.gov)

6. Current BPC-157 Reviews

For the strong preclinical / weak human-evidence distinction. (pubmed.ncbi.nlm.nih.gov)

7. Full-Length Thymosin β4 Human Wound Research

For legitimate component-level human evidence, while keeping it separate from TB-500 fragment evidence. (pubmed.ncbi.nlm.nih.gov)

8. GHK-Cu Reviews

For tissue-remodeling, collagen, and wound biology. (pubmed.ncbi.nlm.nih.gov)

9. NIH Copper / Zinc Fact Sheets

For correcting the current zinc/copper mechanism. (ods.od.nih.gov)

10. PeptideFox GLOW

Evidence type: Community/practitioner protocol

Useful for:

- 50/10/10 formulation
- 2.8 mg community dose
- phased 12-week protocol
- full-length Tβ4 interpretation

- explicit acknowledgment that the blend itself lacks a controlled trial. (peptidefox.com)

11. PeptideDosages GLOW

Evidence type: Community protocol

Useful for:

- 2.33 mg daily
- four-week protocol
- 70 mg product ratio
- showing disagreement in community schedules

but its “receptor reset” rationale should not be adopted. (peptidedosages.com)

Final Audit Verdict

The current GLOW page is not publish-ready as written.

Its strongest feature is the correct high-level statement that the exact blend has very limited evidence. (navarohealth.com)

Its biggest weakness is that it then presents a community protocol and several theoretical safety ideas almost as established pharmacology.

The production page should be built around these rules:

1. GLOW is a community/vendor blend, not an FDA-approved standardized drug.
2. Confirm whether the third component is full-length thymosin β4 or TB-500 fragment LKKTETQ. Those are not interchangeable evidence sources.
3. 2.8 mg is a common community dose for the 50/10/10 formulation, not a clinically validated GLOW dose.
4. Replace “Standard Protocol” with “Community Protocol Examples.”
5. Remove the claim that 5 days on / 2 days off is required to prevent copper toxicity.
6. Remove the current zinc-depletion mechanism and mandatory zinc supplementation. The established oral interaction is high zinc impairing copper absorption, not injected GHK-Cu necessarily causing zinc deficiency through intestinal competition. (ods.od.nih.gov)
7. Remove receptor-reset logic.
8. Do not call active malignancy an established absolute GLOW contraindication. Keep angiogenesis/growth-signaling concerns as theoretical clinical cautions.
9. Remove “Dopamine Buffer” and downgrade sedation, anhedonia, headache, elevated LFTs and similar claims unless directly supported.
10. Put FDA's component-specific concerns prominently near the top: BPC-157 has limited human safety information, injectable GHK-Cu has aggregation/immunogenicity concerns, and FDA says it has not identified human exposure data for TB-500 fragment LKKTETQ. (fda.gov)
11. Keep human full-length thymosin β4 wound studies clearly separated from TB-500-fragment evidence.
12. Do not use a universal four-week or 28-day post-reconstitution stability rule for every GLOW vial.

Once those changes are applied, GLOW can be made publish-ready in the same format as GGH, Retatrutide, and the other finalized compounds.

KLOW

GHK-Cu + BPC-157 + KPV + thymosin-related peptide blend

Evidence	Very limited for the exact blend
Route	SC in community protocols; no clinically validated KLOW route
Common community formulation	50/10/10/10 mg, 80 mg total
Common community dose example	3.2 mg total blend
At that ratio	2 mg GHK-Cu + 400 mcg each BPC-157, KPV and thymosin component
Clinically validated dose	None
Timing	No evidence-based timing requirement
Cycle	No clinically established cycle
Pre-mixed	Formulation-dependent
FDA approved	No

Important identity check: The thymosin ingredient must be confirmed. Full-length thymosin beta4 and TB-500 fragment LKKTETQ are different molecules with different evidence bases.

KPV evidence warning: KPV has meaningful anti-inflammatory evidence in laboratory and animal models, but FDA states that it has not identified human exposure data for KPV administered by any route. Human injectable dosing, pharmacokinetics, long-term safety and clinical efficacy therefore remain unknown.

Exact Formula / Identity

KLOW is not a standardized drug name. Confirm the exact formulation before interpreting a protocol.

GHK-Cu	___ mg
BPC-157	___ mg
KPV	___ mg
Thymosin component	___ mg
Exact thymosin identity	Full-length thymosin beta4 OR TB-500 fragment

Common community formulation: 50 mg GHK-Cu + 10 mg BPC-157 + 10 mg KPV + 10 mg thymosin/TB component = 80 mg total.

TB-500 vs Full-Length Thymosin beta4

The exact thymosin identity must be confirmed before applying evidence to KLOW.

PeptideFox describes its KLOW framework as containing full-length thymosin beta4, while PeptideDosages labels its 80 mg formulation as containing TB-500. These labels should not be assumed to describe the same molecule.

> **Full-length thymosin beta4 and TB-500 fragment are not interchangeable.** Human evidence for full-length thymosin beta4 cannot automatically be transferred to a TB-500 fragment product. FDA defines the TB-500 it reviewed as the LKKTETQ thymosin beta4 fragment and reports no identified human exposure data for drug products containing that fragment.

Full-length thymosin beta4: Limited but real human evidence exists, largely in route- and formulation-specific wound research.

TB-500 LKKTETQ: FDA reports no identified human exposure data for drug products containing the fragment.

KLOW thymosin evidence: Depends on which molecule is actually present.

Dose

Common community dose example: 3.2 mg total blend for the common 50/10/10/10 formulation.

- 2 mg GHK-Cu
- 400 mcg BPC-157
- 400 mcg KPV
- 400 mcg thymosin/TB component

> **This is a community-derived dose, not a clinically validated KLOW treatment dose.**

Community sources do not even agree on one KLOW dose. PeptideFox uses 3.2 mg as a standard community example within a broader 1.6-4.8 mg range, while PeptideDosages publishes substantially lower total-blend examples. This disagreement reinforces that no single community dose should be presented as clinically established.

Community Protocol Examples

Current / older Navaro example

3.2 mg daily, 5 days on / 2 days off, for 8 weeks.

> No human study has established daily dosing, five-days-on/two-days-off scheduling, an eight-week cycle, or this dose as the optimal KLOW protocol.

PeptideFox community framework

- Weeks 1-4: daily
- Weeks 5-8: 5 times per week
- Weeks 9-12: 2-3 times per week

This is a community/practitioner framework, not a clinically validated KLOW schedule.

Alternative 2 -> 4 -> 6 -> 4 mg titration

This may be retained only as a documented community protocol if a source is kept with it.

> **Classification:** Community protocol with no matching human dose-finding study identified.

Copper / Weekend-Break Logic

> Some community KLOW protocols include weekday dosing followed by weekend breaks. No human study demonstrates that two days off prevent or meaningfully reduce copper accumulation or toxicity.

The prior claim that 5 days on / 2 days off prevents or mitigates copper accumulation should not be used as established pharmacology.

Zinc

> There is no established evidence that subcutaneous GHK-Cu requires routine zinc supplementation because of intestinal zinc competition.

Remove routine KLOW-specific zinc testing, mandatory 15-30 mg/day zinc supplementation, zinc deficiency as a KLOW contraindication, progressive zinc depletion as an established KLOW side effect, and the claim that injected GHK-Cu competitively prevents intestinal zinc absorption.

The familiar zinc-copper nutritional interaction concerns intestinal absorption, particularly high oral zinc reducing copper absorption. That does not establish the reverse claim for subcutaneous GHK-Cu.

KPV Evidence

KPV is Lys-Pro-Val, a three-amino-acid peptide related to the C-terminal region of alpha-MSH.

KPV has meaningful laboratory and animal evidence involving anti-inflammatory activity. Experimental intestinal models support PepT1-related transport and effects on inflammatory signaling.

> **FDA's 2026 KPV review is the key evidence limitation:** FDA states that it did not identify human exposure data for KPV administered by any route and did not identify human KPV PK/PD studies. Human injectable dosing, long-term safety and clinical efficacy therefore remain unknown.

Community claims that KPV already has established human IBD dosing should not be repeated unless an identifiable primary human KPV administration study is produced.

KPV Mechanism

KPV research pathways: alpha-MSH-related anti-inflammatory biology, PepT1-mediated transport in experimental intestinal models, and effects on inflammatory signaling such as NF-kappaB-related pathways.

> A clinically dominant human KPV receptor mechanism has not been established because human administration data are lacking.

Do not use **Target: Alpha-MSH** as a standalone target field, and do not present specific chains such as MC1R -> predictable vasodilation, melanocortin receptor -> predictable pigmentation, or area postrema receptor -> predictable nausea as established human KPV pharmacology.

Unsupported KPV Effects

Pigmentation: Not established as a KPV effect in humans; experimental literature should not be converted into a predictable clinical pigment effect.

Blood-pressure effects: Not established.

Nausea: Community-reported / uncertain mechanism if retained at all.

There is no controlled human KPV dataset establishing a predictable first-week nausea period, blood-pressure pattern, injection welt mechanism, or pigmentation response.

Infection

> KPV has anti-inflammatory effects in experimental models, but its effects on human infection, antimicrobial defense and post-infectious recovery have not been established.

Do not state that KLOW should be avoided during active systemic infection because KPV is proven to suppress human antimicrobial defense. Also do not describe KLOW as an established post-infectious repair or maintenance treatment.

Autoimmune Disease and Immunosuppression

> KPV has anti-inflammatory activity in preclinical models, but its clinical effects and safety in people with autoimmune disease or receiving immunosuppressive therapy have not been established.

Concurrent immunosuppressive therapy: Unknown / theoretical interaction.

Do not provide disease-specific KLOW recommendations for IBD, psoriasis, lupus, vasculitis, rheumatoid arthritis or similar conditions.

BPC-157 Evidence

BPC-157 has substantial preclinical research, but the human evidence remains extremely limited.

A two-person IV pilot is real, and newer reviews identify a small amount of additional uncontrolled human pilot experience. Overall, the human literature still involves fewer than 30 people across very small uncontrolled studies, without a validated pharmaceutical formulation or established Phase II efficacy program.

BPC-157 preclinical evidence: Substantial

Human evidence: Extremely limited, small uncontrolled pilots

Human SC KLOW dose: Not validated

KLOW combination evidence: None established

GHK-Cu Evidence

GHK-Cu has legitimate biological research involving extracellular matrix, collagen, wound biology, tissue remodeling and related signaling.

> GHK-Cu component biology is meaningful. That does not establish chronic subcutaneous GHK-Cu or the four-component KLOW blend as clinically effective or safe.

FDA specifically states that compounded injectable GHK-Cu may present immunogenicity risk because of aggregation and peptide-related impurities and that human safety data are limited.

Dopamine Claims

Remove **Dopamine Buffer** from the KLOW profile.

> Fatigue or mood changes may appear in community reports, but KLOW has no controlled human safety dataset establishing their incidence, duration or mechanism.

Do not describe fatigue or anhedonia as established consequences of BPC-157, thymosin/TB or KPV dopaminergic modulation.

Safety

Plausible / Community-Reported

- Injection-site pain or discomfort
- Redness
- Swelling
- Irritation
- Headache

- Nausea
- Fatigue

> Exact incidence and causality are unknown because the fixed KLOW blend has not undergone a controlled human safety trial.

Do Not Present as Established KLOW Adverse Effects

- Predictable pigment changes
- Predictable blood-pressure changes
- Zinc depletion
- Copper toxicity at a standard community dose
- Anhedonia
- Elevated ALT/AST

Severe liver disease may remain under Important Cautions because the exact blend has not been adequately studied there, but elevated liver enzymes should not be described as a normal expected KLOW effect.

Formal Contraindications

> **None established through an FDA-approved KLOW label.**

There is no FDA-approved KLOW product, so the page should not create an official-looking label-derived contraindication list.

Important Cautions

Copper metabolism disorders / Wilson disease	Strong biologically relevant caution
Pregnancy / breastfeeding	Safety unknown
Severe hepatic impairment	Important unknown / caution
Concurrent immunosuppressive therapy	Unknown / theoretical interaction
Active malignancy	Important theoretical / clinical caution, not an official absolute contraindication

Active cancer should not be described as absolutely contraindicated. Several components influence repair, vascular or inflammatory pathways in experimental systems, but the effects of KLOW in active malignancy are unknown.

Commonly Paired With

Thymosin Alpha-1	Community / theoretical combination
LL-37	Community / theoretical combination; separate safety uncertainty should be considered
Matrixyl	Different use context; no established KLOW synergy

Replace **Reported Synergistic** with **Commonly Paired With** unless direct combination evidence exists.

Ingredient Overlap / Redundancy

KLOW + GLOW	Major ingredient duplication: GHK-Cu, BPC-157 and thymosin component
KLOW + KPV	Duplicates KPV
KLOW + BPC-157	Duplicates BPC-157
KLOW + GHK-Cu	Increases GHK-Cu / copper exposure
KLOW + TB-500 / Tbeta4	Potentially duplicates thymosin component; exact molecule must be checked
KLOW + Tri-Heal	Formula-dependent duplication

These combinations should not automatically be called pharmacologic contraindications. The principal issue is duplicate ingredients and uncertain total exposure.

Pre-Mixed

Pre-mixed: Formulation-dependent. Common community KLOW products are sold as fixed-ratio single-vial blends.

Formulation, Stability and Sterility

No FDA-approved KLOW formulation establishes a universal reconstitution method, bacteriostatic-water volume, refrigerated shelf life, 14-day limit or 28-day limit. Same-vial chemical and physical stability must be established for the exact formulation.

A vendor selling four peptides in one vial demonstrates that a product exists. It does not independently establish long-term peptide integrity, compatibility, potency retention, aggregation behavior or a validated beyond-use period.

Community 28-day or other refrigerated-use windows should remain product/community handling conventions unless supported by exact-formulation stability data.

FDA 2026 Regulatory Context

FDA staff scientific evaluation, Pharmacy Compounding Advisory Committee activity, 503A Bulks List status and FDA drug approval are separate regulatory concepts.

> In its 2026 scientific evaluation, FDA staff concluded that the available evidence weighed against adding KPV free base and KPV acetate to the 503A Bulks List. The July 2026 advisory process is separate from FDA approval of a drug, and KPV remains an unapproved substance without established human clinical efficacy or safety.

The same distinction should be maintained for BPC-157 and TB-500. Do not collapse staff recommendations, advisory votes or list status into the phrases 'FDA approved' or 'FDA rejected' when discussing drug approval.

Final Evidence Ladder

Evidence area	Final rating
Exact KLOW blend	Very limited
Human KLOW efficacy trial	None identified
Human KLOW dose-finding	None identified
KLOW PK/PD	None identified
KPV preclinical evidence	Meaningful
KPV human exposure	FDA identified none
BPC-157 preclinical evidence	Substantial
BPC-157 human evidence	Extremely limited
Full-length Tbeta4 human evidence	Limited but real
TB-500 LKKTETQ human exposure	FDA identified none
GHK-Cu biological evidence	Meaningful
Injectable GHK-Cu human safety	Limited; FDA concerns
3.2 mg KLOW protocol	Community
5-on / 2-off	Community convention
2 -> 4 -> 6 -> 4 mg titration	Community convention
Routine zinc	Unsupported
Dopamine-buffer claim	Unsupported
Same-vial stability	Not established
Universal 28-day BUD	Not established
FDA-approved KLOW	No

Protocol Logic Check

Question	Verdict
Is KLOW a standardized FDA-approved drug?	No
Is 50/10/10/10 a documented community formulation?	Yes
Is every KLOW vial necessarily that formulation?	No
Must the exact thymosin component be confirmed?	Yes
Are full-length Tbeta4 and TB-500 LKKTETQ interchangeable?	No
Human trial of exact KLOW blend identified?	No
Is 3.2 mg mathematically coherent for 50/10/10/10?	Yes
Is 3.2 mg clinically validated?	No
Is daily dosing clinically validated?	No
Is 5 days on / 2 days off clinically validated?	No
Does a weekend break prevent copper toxicity?	Not established
Is an 8- or 12-week cycle required?	No
Does KLOW require receptor resetting?	No evidence
Does injected GHK-Cu require mandatory zinc?	No
Does KPV have meaningful preclinical anti-inflammatory evidence?	Yes
Did FDA identify human KPV exposure data?	No
Is KPV's human SC dose established?	No
Does BPC-157 have substantial human evidence?	No
Does full-length Tbeta4 have some human evidence?	Yes
Does that validate TB-500 LKKTETQ?	No
Does GHK-Cu have legitimate component evidence?	Yes
Is injectable GHK-Cu safety well established?	No
Is 'Dopamine Buffer' validated?	No
Is active malignancy an official KLOW contraindication?	No
Is same-vial stability established?	No
Is universal post-reconstitution stability established?	No

Research Sources

FDA 2026 KPV briefing

Primary regulatory source for the finding that FDA did not identify human KPV exposure data or human PK/PD studies.

FDA significant safety risks page

Primary regulatory source for KPV, BPC-157, injectable GHK-Cu and TB-500 safety/evidence limitations.

PubMed KPV / PepT1 experimental literature

Supports KPV anti-inflammatory biology and PepT1-related experimental transport.

PubMed BPC-157 human pilot and current reviews

Supports the classification of human BPC-157 evidence as extremely limited.

Human full-length thymosin beta4 wound literature

Supports limited but real route-specific human evidence for full-length thymosin beta4.

GHK-Cu reviews and human literature

Supports component-level tissue-remodeling biology while keeping injectable evidence separate.

PeptideFox KLOW

Community/practitioner source for the 80 mg 50/10/10/10 formulation, 3.2 mg community example and full-length thymosin beta4 terminology.

PeptideDosages KLOW

Community source illustrating a different total-blend dosing framework and TB-500 terminology.

Final Production Bottom Line

KLOW is a community/vendor four-component blend, not a standardized or FDA-approved treatment. A common 80 mg formulation contains 50 mg GHK-Cu plus 10 mg each of BPC-157, KPV and a thymosin-related component, but the exact thymosin molecule must be confirmed.
The commonly cited 3.2 mg dose is mathematically coherent for that formulation, but it is a community dose rather than a clinically validated treatment dose.
The largest additional evidence limitation is KPV: it has meaningful laboratory and animal anti-inflammatory research, but FDA states that it has not identified human KPV exposure data by any route.
KLOW should therefore be presented through component evidence, documented community protocols and explicit exact-blend uncertainty, not as a clinically validated four-peptide protocol.