

WHAT'S INSIDE

1	What tesamorelin actually is	1	9	Liver fat and metabolic claims	9
2	What FDA approval actually means	2	10	What happens when treatment stops	10
3	What the human trials actually show	3	11	Does it need bedtime, fasting or cycles?	11
4	Dosing: studied vs community	4	12	Does it need a second peptide?	12
5	Why 2 mg, 1.4 mg and 1.28 mg all exist	5	13	Safety, IGF-1 and glucose	13
6	What visceral fat actually is	6	14	Evidence summary	14
7	Visceral fat vs visible belly fat	7	15	Sources	15
8	Muscle and recomposition claims	8			

01 What Tesamorelin Actually Is

Tesamorelin is a synthetic version of growth hormone-releasing hormone, the signal your body uses to tell the pituitary gland to release growth hormone. It works on the pituitary, so the body puts out its own growth hormone, and IGF-1 rises downstream as a result. Its strongest human evidence is unusually specific: reducing excess visceral abdominal fat in adults with HIV-associated lipodystrophy.

The story people often hear online

“Tesamorelin burns stubborn belly fat, builds or preserves muscle, works best fasted before bed, and should be cycled or stacked with Ipamorelin.” Some pieces of that have a biological rationale behind them. Several are not what the clinical trials tested.

THE MOST IMPORTANT SAFETY CORRECTION

“It only releases your own GH, so high IGF-1 is harmless and more is better” is false. Tesamorelin can push IGF-1 well above the normal range for a person's age, can worsen blood sugar control, and has specific situations where it should not be used at all. Because the hormone comes from your own pituitary does not make heavy GH and IGF-1 exposure risk-free.

02 What Is Tesamorelin FDA Approved For?

In the United States, EGRIFTA is approved to reduce excess abdominal fat in HIV-infected adults with lipodystrophy. That is a different thing from obesity treatment, cosmetic belly-fat loss, bodybuilding, anti-aging, or general “GH optimisation.”

APPROVED USE

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

NOT APPROVED FOR

Weight-loss management. The current label describes tesamorelin as weight-neutral.

Current clinical formulations

HISTORICAL TRIAL DOSE

2 mg

The older formulation, used in the pivotal 26-week and 52-week visceral fat trials.

EGRIFTA SV

1.4 mg

FDA data show it puts a similar amount of drug into the bloodstream as the older 2 mg formulation.

EGRIFTA WR

1.28 mg

Also designed to deliver a similar amount of drug as the older 2 mg formulation.

THE FORMULATION TRAP

These are not three strength tiers. Milligrams cannot be compared unless you know which formulation produced the number. A vial labelled “tesamorelin 10 mg” from a research vendor is not automatically equivalent to EGRIFTA WR, EGRIFTA SV, or the historical clinical formulation.

IN SHORT

It is approved for one thing: reducing excess abdominal fat in HIV-infected adults with lipodystrophy. Weight loss, cosmetic fat loss, bodybuilding and anti-aging all sit outside that label, and the three milligram figures are formulations rather than strengths.

03 What the Human Trials Actually Show

Tesamorelin has considerably more human outcome data behind it than most compounds discussed in peptide communities. The results are strongest for visceral fat in HIV lipodystrophy, with narrower signals for liver fat and lean mass.

Pivotal HIV lipodystrophy trials	STRONG POSITIVE	The historical 2 mg dose, injected under the skin daily for 26 weeks. Visceral fat fell by about 18% on average in one pivotal study and 14% in the other. Body weight barely moved.
52-week extension	IMPORTANT	Staying on treatment held the visceral fat effect in place. Switching to placebo after 26 weeks let a substantial amount of that fat come back.
HIV and fatty liver trial	POSITIVE SIGNAL	The historical 2 mg dose daily for 12 months. Liver fat fell substantially compared with placebo in adults who had both HIV and fatty liver disease.
Lean body mass	REAL SIGNAL	The pivotal trials showed roughly 1.2 to 1.3 kg more lean body mass. That is not the same as 1.2 to 1.3 kg of new skeletal muscle.
Healthy lifters	NOT TESTED	There is no dedicated controlled trial showing that tesamorelin builds meaningful muscle or strips subcutaneous fat in healthy people who train.

IN SHORT

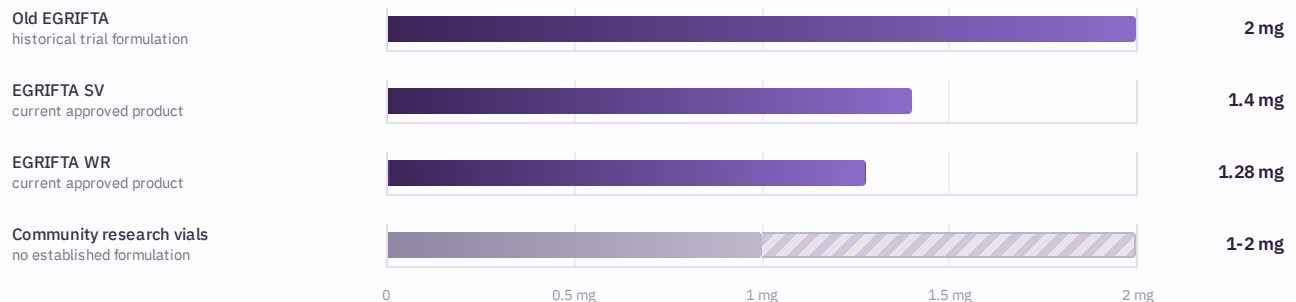
One strong result and two narrower ones. Visceral fat falls substantially in HIV lipodystrophy. Liver fat and lean mass move as well, but by less and in fewer people, and nothing at all has been tested in healthy lifters.

04 Dosing: What Was Studied and What Circulates Online

Tesamorelin dosing is easy to misread because the formulation changed along the way. A milligram number tells you very little unless you also know which formulation produced it.

Pivotal visceral fat trials	2 mg daily	26 weeks, extending to 52 weeks, using the historical EGRIFTA formulation.
HIV and liver fat trial	2 mg daily	6 to 12 months, also using the historical formulation.
Current EGRIFTA SV	1.4 mg daily	The current labelled SV dose, designed to deliver roughly the same drug exposure as the old 2 mg product.
Current EGRIFTA WR	1.28 mg daily	The current labelled WR dose, also designed to deliver roughly the same exposure as the old 2 mg product.
Community research-vial use	1 to 2 mg daily	A common online cluster, often taken fasted and at night. Lower starting amounts and 5-on/2-off schedules also circulate.

THE SAME NUMBERS ON ONE SCALE, FROM DIFFERENT FORMULATIONS



Approved or trial formulations Research vials. The bars show milligrams only. Because the formulations differ, three different milligram numbers were designed to put a similar amount of drug into the bloodstream.

Research-vial caution

The historical 2 mg dose is an efficacy anchor for the historical formulation. It is not proof that 2 mg from an uncharacterised research vial produces the same exposure. Equivalence between the two has never been established.

IN SHORT

The trials used 2 mg of the old formulation. The approved products now use 1.4 mg and 1.28 mg. Online the cluster is 1 to 2 mg. The milligram number means little on its own, because the formulation decides how much drug actually reaches the bloodstream.

05 Why 2 mg, 1.4 mg and 1.28 mg Can All Be “Clinical”

The drug molecule is tesamorelin in every case. What changes is the formulation, and the formulation changes how much of each injection is absorbed. That is why the labelled milligram dose came down while the amount of drug reaching the bloodstream stayed about the same.

OLD EGRIFTA 2 mg daily, under the skin	EGRIFTA SV 1.4 mg daily, under the skin	EGRIFTA WR 1.28 mg daily, under the skin
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All three were designed to put a similar amount of drug into the bloodstream

The pivotal efficacy data come from the 2 mg column. The other two are formulation changes built to match that exposure, not weaker versions of the drug.

What the pharmacology can and cannot settle

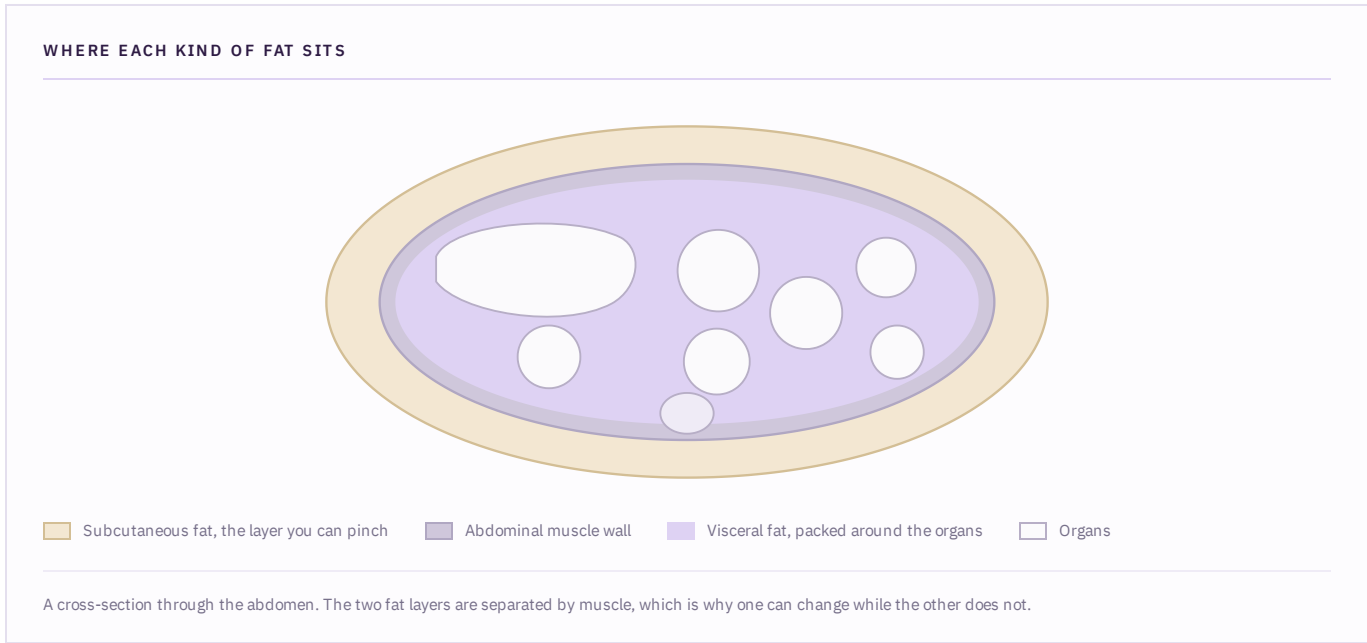
Once daily	SUPPORTED	The clinical programmes and the current products all use once-daily dosing. The short time the drug spends in the blood does not create evidence for splitting the dose or redosing through the day.
5 days on, 2 days off	NO TRIAL BASIS	No evidence shows that weekend gaps improve receptor sensitivity, pituitary function, safety or visceral fat loss.
An 8 to 12 week “cycle”	NOT THE CLINICAL MODEL	The major visceral fat trials ran 26 to 52 weeks, and the liver fat trial ran a full 12 months.
Bedtime	PLAUSIBLE, UNPROVEN	Growth hormone rises naturally as you fall asleep, so bedtime dosing is coherent. No tesamorelin outcome trial has shown it works better.
Fasted	PLAUSIBLE, UNPROVEN	Blood sugar can influence how the pituitary responds, but no clinical trial established a required fasting window for tesamorelin to work.

IN SHORT

Because the formulation changed, not the drug. Each version was designed to put a similar amount of tesamorelin into the bloodstream, so a lower milligram number is not a weaker dose. How often and how long to take it are settled by the trials. Bedtime and fasting are not.

06 What Is Visceral Fat?

Visceral fat is the fat stored deep inside the abdomen, packed around and between the organs. It sits behind the wall of abdominal muscle, which is why you cannot get hold of it from the outside. It is a normal part of the body, and the question is only ever how much of it there is.



Visceral fat compared with the fat you can see

Where it sits	Subcutaneous fat lies directly under the skin, all over the body. Visceral fat lies deeper, behind the abdominal muscle wall and in among the organs.
Can you pinch it	Subcutaneous fat, yes. Visceral fat, no. If you can grab it, it is not visceral fat.
How it is measured	Subcutaneous fat can be estimated with calipers, a tape measure or a DEXA scan. Visceral fat needs a CT or MRI scan to measure properly.
What it does	Subcutaneous fat is mostly a storage depot and is relatively quiet. Visceral fat is more active, releasing fatty acids and inflammatory signals into the vein that runs straight to the liver.
Why it matters	A high amount of visceral fat is associated with insulin resistance, fatty liver and cardiovascular risk more strongly than the same amount of subcutaneous fat.
What you notice	Subcutaneous fat is what shapes the body in the mirror. Visceral fat pushes the abdominal wall outward from the inside, which is why a belly can be firm rather than soft.

IN SHORT	Visceral fat is the deep fat packed around the organs, behind the abdominal muscle wall. It is not the layer you can pinch. The two sit in different places, do different things and are measured in different ways, which is why a drug can shift one and leave the other alone.
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07 Does Tesamorelin Really “Target Belly Fat”? FALSE

Yes, but “belly fat” is too vague a phrase to be useful. The strongest trials measured visceral fat, the deep fat packed around the abdominal organs. They did not show a broad stripping of the surface layer you can actually pinch.

VISCERAL FAT

Strong human evidence in the approved population. Visceral fat fell about 18% and 14% on average at 26 weeks in the two pivotal studies.

SUBCUTANEOUS BELLY FAT

Little to no meaningful reduction in the abdominal fat sitting just under the skin, across the pivotal work.

AVERAGE VISCERAL FAT CHANGE AT 26 WEEKS



Both figures are reductions in visceral fat only. Body weight was essentially unchanged in both studies.

Body weight	ESSENTIALLY NEUTRAL	This is why tesamorelin is not a general weight-loss drug.
“18% visceral fat loss means 18% body-fat loss”	FALSE	Visceral fat is one compartment. A change within it is not a change in whole-body fat percentage.
Injecting into the belly burns fat there	FALSE	Where you inject is a route of administration, not spot reduction. The effect is systemic.
Love handles and lower belly	NOT ESTABLISHED	Those visible areas are mostly subcutaneous fat, the compartment that did not show the same signal.

WHY IT FEELS TRUE

- The starting point is a real result.** Visceral fat genuinely fell 14% to 18%. The claim is built on something that happened, which is what makes it hard to argue with.
- The waist can visibly change.** When the deep fat shrinks, the abdominal wall is pushed out less from the inside, so the midsection can look flatter while the pinchable layer is untouched.
- One phrase covers two tissues.** Trials measured visceral fat. People hear “belly fat” and picture the part they can grab, and the injection going into the stomach area makes local action feel obvious.

IN SHORT

No, not the belly fat people mean. It reduces the deep visceral fat around the organs and left the layer you can pinch largely alone. The abdomen can change shape while the scale barely moves and the surface fat stays put.

08 Does Tesamorelin Build or Preserve Muscle?

FALSE

There is a real lean-mass signal here, but the bodybuilding reading of it runs well ahead of the data. The pivotal HIV trials showed increased lean body mass. They were not muscle-growth trials in healthy people.

LEAN BODY MASS

+1.2 to 1.3 kg

Measured in the pivotal 26-week trials

MUSCLE CT ANALYSIS

Responders

An exploratory secondary analysis, limited to people who responded

TRIALS IN HEALTHY LIFTERS

None

No dedicated muscle-growth trial has been run

Lean mass is not the same as muscle

IMPORTANT

A DEXA scan counts water and other non-fat tissue as lean mass. Drugs that work on the growth hormone axis can also cause fluid retention.

Water gain proves muscle gain

FALSE

Swelling or a quick jump on the scale is not evidence of new muscle, and it can inflate a lean-mass reading.

“The recomp peptide”

OVERSTATED

Losing visceral fat while lean mass ticks up can look like recompensation. It is not proof of real muscle growth plus subcutaneous fat loss in healthy athletes.

Protecting muscle on a GLP-1

UNPROVEN

No pivotal tesamorelin trial shows it is better than anything else at preventing the lean-mass loss that comes with GLP-1 drugs.

WHY IT FEELS TRUE

- The scan number really does go up.** Lean body mass rose 1.2 to 1.3 kg, and “lean mass” sounds like a synonym for muscle even though it counts water and organ tissue too.
- Growth hormone carries a reputation.** Decades of bodybuilding association mean anything that raises GH inherits the assumption that it builds muscle.
- Fluid retention arrives early and feels like progress.** Fuller-looking muscles and a heavier scale in the first weeks read as growth, and fat falling at the same time completes a recompensation story nobody tested.

IN SHORT

No, not on this evidence. Lean body mass rose by about 1.2 to 1.3 kg, but lean mass counts water and other non-fat tissue, and the trial that would show it is muscle has never been run. “May support lean mass” is defensible. “Builds muscle” is not.

09 Does Tesamorelin Reduce Liver Fat? TRUE

Yes, and there is direct human evidence for it, but again in people with HIV. A randomised 12-month trial in adults with HIV and fatty liver disease used the historical 2 mg daily formulation.

LIVER-FAT EFFECT

-37%

Average effect compared with placebo

REACHED UNDER 5% LIVER FAT

35%

Against 4% of people on placebo

HISTORICAL STUDY DOSE

2 mg/day

Under the skin, daily, for 12 months

What the study proves

REAL HUMAN SIGNAL

Tesamorelin can reduce measured liver fat in adults who have HIV and fatty liver disease.

“Tesamorelin heals fatty liver”

TOO BROAD

The result does not establish it as a treatment for fatty liver disease in people without HIV.

“It detoxes the liver”

FALSE FRAMING

Reducing fat in the liver in a defined population is not detoxification.

Prevents scarring or cirrhosis

NOT ESTABLISHED

The available evidence does not prove that long-term liver outcomes are prevented.

Prevents heart attacks

NOT ESTABLISHED

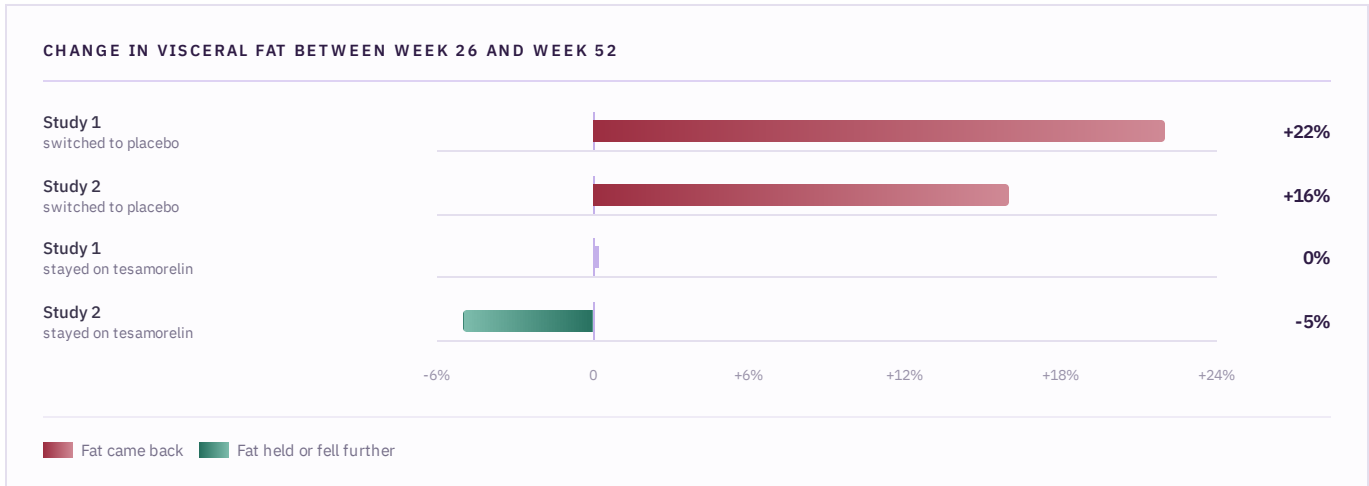
Visceral fat reduction and metabolic changes are stand-in measures. They are not evidence that heart attacks are prevented.

IN SHORT

Yes, in adults who have HIV and fatty liver disease. A randomised 12-month trial cut liver fat by about 37% against placebo, and 35% of people got below the 5% mark. It is not a fatty liver treatment for everyone else, not a detox, and not evidence about scarring or heart attacks.

10 Does the Visceral Fat Stay Gone After a “Cycle”? FALSE

The extension studies tested this directly. They took people who had responded to tesamorelin and stopped their treatment. A substantial amount of the visceral fat came back.



What this shows	REVERSIBLE EFFECT	Tesamorelin behaves like an ongoing drug effect, not a one-time correction that stays put.
A short cycle permanently fixes visceral fat	FALSE	What actually happened after stopping was the fat coming back, not a durable reset.
A planned break protects the pituitary	NO HUMAN EVIDENCE	Continuous daily treatment was studied for up to 52 weeks. No scheduled break has been shown to be necessary or useful.
Antibodies mean the receptor burned out	NOT SUPPORTED	Antibodies to tesamorelin did occur, but responses were not meaningfully different between people who had them and people who did not.

WHY IT FEELS TRUE

- Other fat loss can hold.** Fat lost through diet and training often stays off, so people reasonably assume this behaves the same way once the work is done.
- The reversal is invisible.** Nothing dramatic happens the week you stop, and visceral fat cannot be seen or felt coming back without a scan.
- The word cycle is borrowed.** It arrives from anabolic steroid culture, where the frame is run a cycle and keep the gains. Applied here, that frame quietly assumes the change is permanent.

IN SHORT

No. In the 26 weeks after stopping, visceral fat climbed 22% and 16% in the two studies, while the people who stayed on treatment held theirs. The effect lasts as long as the treatment does, which makes a short cycle the wrong model for this drug.

11 Does Tesamorelin Need Bedtime Dosing, Fasting or Cycles?

FALSE

The protocol you find online usually sounds very precise. The clinical evidence is much simpler. Once-daily treatment is what was studied. Bedtime, fasting windows, weekend breaks and reset cycles were never established as requirements.

Once daily	STRONGEST SUPPORT	The approved products and the pivotal trials all use one injection under the skin per day.
Bedtime	PLAUSIBLE, UNPROVEN	Growth hormone naturally pulses at night, which makes bedtime dosing physiologically coherent. No outcome trial shows better visceral fat loss from it.
Fasted	PLAUSIBLE, UNPROVEN	There is a mechanism behind the idea, but no required fasting window has been clinically validated.
Carbs “cancel” the dose	OVERSTATED	Blood sugar can influence growth hormone physiology. That does not prove a meal makes tesamorelin ineffective.
5 days on, 2 days off	NO TRIAL BASIS	No evidence shows that weekend gaps prevent tolerance or improve safety.
An 8 to 12 week cycle	NOT THE CLINICAL MODEL	The major efficacy studies lasted far longer, and stopping was followed by visceral fat coming back.
Pre-workout or post-workout timing	NO OUTCOME BASIS	Claims that one timing burns fat while another builds muscle have never been tested in a tesamorelin outcome trial.

WHY IT FEELS TRUE

- The physiology underneath is real.** Growth hormone does pulse as you fall asleep, and glucose really can blunt the pituitary response to a GHRH signal. Both rules start from a true mechanism.
- Precision reads as expertise.** “Fasted, 45 minutes before bed” sounds like it came from a protocol rather than a forum, and a specific instruction is easier to trust than a vague one.
- Nothing contradicts it.** Nobody can feel the difference between dosing at 9pm and dosing at noon, so the rule survives indefinitely because personal experience can never disprove it.

IN SHORT

No. Once a day, every day, is what the approved products and every human trial used. Bedtime and fasting are physiologically sensible guesses that no outcome trial has ever tested, and weekend breaks and 8 to 12 week cycles have no basis at all.

12 Does Tesamorelin Need a Second Peptide to Work? FALSE

Community protocols often add tesamorelin to another growth hormone secretagogue, or to a GLP-1 drug. A logical mechanism is not the same thing as controlled outcome evidence.

Tesamorelin + Ipamorelin	MECHANISTICALLY PLAUSIBLE	Stimulating the GHRH receptor and the ghrelin receptor at the same time can amplify growth hormone release. No controlled combination trial proves better visceral fat loss, muscle, sleep or safety.
Tesamorelin + CJC-1295	HEAVY OVERLAP	Both push the same GHRH pathway. A clinical benefit from stacking them has not been established.
Tesamorelin + HGH	OVERLAPPING ENDPOINT	Injected HGH bypasses the pituitary entirely. Running both can push GH and IGF-1 side effects higher with no validated best-of-both protocol.
Tesamorelin + a GLP-1	DIFFERENT PATHWAYS, UNTESTED	The theory is appetite and weight loss from one, visceral fat and lean-mass support from the other. Less redundant than the others, but not validated by any pivotal tesamorelin trial.

THE COMMUNITY THEORY

A GHRH signal plus a ghrelin-receptor signal produces a stronger pulse of growth hormone, usually timed before sleep.

THE EVIDENCE PROBLEM

Tesamorelin earned its approval without Ipamorelin anywhere near it. The stack inherits the mechanism. It does not inherit the clinical validation.

WHY IT FEELS TRUE

- The synergy is genuine in the lab.** Human studies show a GHRH signal plus a ghrelin-receptor signal produces a larger growth hormone pulse than either alone. That part is real. [10,11]
- More hormone sounds like more result.** If tesamorelin works through growth hormone, a bigger pulse seems like it must mean a bigger effect, even though no outcome trial has checked.
- The stack is how it is sold.** Vendors package and price these together, and when results feel slow, adding a second vial feels like doing something about it.

IN SHORT

No. It earned its approval on its own, with no Ipamorelin, CJC or HGH anywhere near it. Every stack borrows a plausible mechanism, and not one of them has been tested for outcomes.

13 What Is Actually Established About Safety?

Tesamorelin does not merely “nudge growth hormone naturally.” It can raise IGF-1 enough that the FDA label calls for monitoring, and it can worsen blood sugar control and cause the fluid-retention symptoms familiar from growth hormone therapy.

IGF-1 goes up	IMPORTANT	At 26 weeks, 47% of treated patients were above the +2 standard deviation mark for their age and 36% were above +3. A reading that stays that high is a reason to reassess treatment.
“Higher IGF-1 is better”	FALSE AND RISKY	IGF-1 is not a score where a bigger number means a better result.
Blood sugar and diabetes	REAL RISK	HbA1c reached 6.5% or higher in 5% of tesamorelin patients, against 1% on placebo, across the clinical programme.
Fluid retention	ESTABLISHED	Swelling, joint pain, pain in the hands and feet, and carpal-tunnel-type symptoms can all occur.
Cancer	CONTRAINDICATED	Active cancer rules the drug out. A history of cancer calls for a careful weighing of risk and benefit.
Pituitary problems	CONTRAINDICATED	Tesamorelin needs a working pituitary to do anything. It is not a substitute for growth hormone in people whose pituitary has failed.
Injection and allergic reactions	ESTABLISHED	Redness, itching, pain, bruising, rash, hives and flushing have all been reported.

DO NOT CONFUSE “NATURAL RELEASE” WITH “RISK-FREE”

The pituitary is involved, but growth hormone and IGF-1 downstream can still rise a long way. Monitoring IGF-1 and blood sugar matters far more than whether the injection went in exactly 45 minutes before bed.

WHY IT FEELS TRUE

- 1 **“Your own hormone” sounds safe.** Something the body already makes feels categorically different from something injected, even when the resulting blood levels are what matters.
- 2 **The pituitary seems self-limiting.** People assume a gland under feedback control cannot overshoot. IGF-1 climbing past the normal range in nearly half of treated patients shows otherwise.
- 3 **Injected HGH is the villain by comparison.** Next to synthetic growth hormone, a secretagogue feels like the gentle option, and side effects such as swelling and joint ache get read as proof it is working.

IN SHORT

Three things. IGF-1 can climb well past the normal range for a person’s age, blood sugar can worsen, and fluid retention is common. Active cancer and a non-working pituitary rule the drug out entirely. The hormone coming from your own pituitary changes none of that.

14 Evidence Summary

Reduces visceral fat	STRONG IN HIV LIPODYSTROPHY	Large randomised trials show substantial visceral fat reduction.
A general weight-loss drug	NO	Body weight was essentially neutral, and the FDA says it is not indicated for weight loss.
Burns visible belly fat	NOT ESTABLISHED	The pivotal data did not show the same reduction in the fat just under the skin.
Builds or preserves muscle	SIGNAL, OFTEN OVERSTATED	Lean body mass increased. There is no dedicated muscle-growth trial in healthy people.
Reduces liver fat	DIRECT HIV SIGNAL	A randomised 12-month trial showed a meaningful reduction in liver fat.
Must be taken fasted at bedtime	PLAUSIBLE, NOT PROVEN	Physiology gives a rationale. Superiority has not been demonstrated in any outcome trial.
Needs 5 on, 2 off	NO TRIAL BASIS	The human efficacy programmes dosed every day.
Needs 8 to 12 week cycles	NO RESET EVIDENCE	Studies ran 26 to 52 weeks, and visceral fat came back after withdrawal.
1.28 mg, 1.4 mg and 2 mg are directly comparable	NO	Different formulations were designed to deliver similar exposure at different milligram doses.
Tesamorelin plus Ipamorelin is better	MECHANISTIC INFERENCE	No controlled combination trial establishes that it is.
Anti-aging or longer life	NOT ESTABLISHED	No tesamorelin trial demonstrates reversal of aging or a longer lifespan.
Higher IGF-1 is better	FALSE	A reading that stays markedly high is a safety concern, not a target to chase.

Conclusion

WHAT IS REAL

Tesamorelin has unusually strong randomised human data for visceral fat reduction in HIV lipodystrophy, plus narrower signals for lean mass and liver fat.

WHAT GETS ADDED ONLINE

Cosmetic spot-fat claims, mandatory bedtime fasting, receptor-reset cycles, untested stacks, “natural GH is safe,” and anti-aging claims.

IN SHORT

One claim holds up strongly: visceral fat reduction in HIV lipodystrophy. Liver fat and lean mass are real but smaller. Almost everything else on the list is unproven or false. For approved products, use the dose that belongs to that formulation, once daily, and watch IGF-1 and blood sugar.

15 Sources Used for This Guide

- 1 **U.S. FDA. EGRIFTA WR Prescribing Information, revised 2025.** Current indication, 1.28 mg once-daily dose, pharmacokinetics, IGF-1, glucose and safety warnings.
- 2 **U.S. FDA. EGRIFTA SV Prescribing Information.** 1.4 mg once-daily formulation and exposure comparison with historical EGRIFTA.
- 3 **Falutz J, et al. N Engl J Med. 2007.** Randomized trial in adults with HIV and abdominal fat accumulation; historical 2 mg SC daily.
- 4 **Falutz J, et al. J Clin Endocrinol Metab. 2010.** Pooled analysis of two Phase 3 tesamorelin trials with 26-week randomized phases and 26-week safety extensions; maintenance with continued therapy and VAT rebound after withdrawal.
- 5 **Stanley TL, et al. JAMA. 2014.** Randomized trial of tesamorelin in adults with HIV; visceral and liver-fat outcomes.
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- 10 **Bowers CY, et al. J Clin Endocrinol Metab. 1990;70:975-982.** PMID 2108187. Human evidence that GHRP and GHRH can act synergistically to stimulate GH release.
- 11 **Hataya Y, et al. J Clin Endocrinol Metab. 2001;86:4552.** PMID 11549707. Human evidence of synergistic GH release from ghrelin plus GHRH.
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- 13 **Valcavi R, et al. Clin Endocrinol (Oxf). 1990;32:539-543.** PMID 2114240. Shows glucose and GHRH interactions are timing-dependent, supporting caution against the claim that carbohydrates simply cancel a dose.
- 14 **Raun K, et al. Eur J Endocrinol. 1998;139:552-561.** PMID 9849822. Establishes Ipamorelin as a selective growth hormone secretagogue acting through the GHS/GHRP pathway; supports the mechanistic premise of Tesamorelin plus Ipamorelin, not clinical superiority.

Community sources reviewed

These record what people are being told online. They are not used as evidence that the claims are true.

- Reddit and peptide-community discussions, reviewed for current 1 to 2 mg dosing, nighttime and fasted use, 5-on/2-off schedules, 8 to 16 week cycles, and stacking claims.
- Commercial peptide protocol pages, reviewed for Tesamorelin plus Ipamorelin, bedtime timing, pre and post-workout claims, and cycle structures.

How the evidence ranks

Highest confidence	FDA labelling plus randomised human trials in HIV lipodystrophy.
Useful but narrower	Exploratory muscle analyses and the HIV fatty-liver studies.
Mechanistic inference	Bedtime, fasting, and GHRH plus ghrelin-receptor synergy.
Community convention	5-on/2-off, reset cycles, workout timing, research-vial dose ladders.

DO NOT FLATTEN THE EVIDENCE

A real clinical dose does not make an untested schedule clinical. A plausible mechanism does not make a stack proven. And “belly fat” does not mean all abdominal fat behaves the same way.