

CJC-1295 / Ipamorelin: A Compelling Case

Sermorelin, tesamorelin and the CJC-1295 / ipamorelin pair all ask your pituitary to release more of its own growth hormone. Viewed through a chemistry, manufacturing and controls (CMC) lens, the pair has four structural advantages. Here is the case, residue by residue, and where the evidence stops.

Updated By John Phipps · Revision 2, October 23, 2026 · [Download the PDF](#)

The case in four points

1 Built for solution stability

CJC-1295 replaces the asparagine and methionine weak points of native GHRH. Ipamorelin, at five residues, has neither. Sermorelin carries both unprotected, and tesamorelin relies on formulation to manage them.

[The CMC comparison →](#)

2 Two receptor pathways

CJC-1295 acts on the GHRH receptor and ipamorelin on the ghrelin receptor. Sermorelin and tesamorelin use only the first.

[How the two signals combine →](#)

3 Strong GH stimulation

The ghrelin-receptor arm pushes back on somatostatin, the body's GH brake. In human studies, the two pathways together released about twice the sum of each alone.

[The GH and IGF-1 data →](#)

4 Resists degradation, keeps potency

One D-alanine blocks the enzyme that clips sermorelin within minutes, and the analog design was several times more potent than native GHRH.

[Half-life and potency →](#)

Bottom line. The case for CJC-1295 / ipamorelin is structural and mechanistic, and on those terms it is strong. It is not a clinical case. Tesamorelin is the only one of the three with an FDA approval based on a clinical outcome, and no trial has tested the CJC-1295 / ipamorelin pair for any outcome. Its benefits are proposed rather than proven.

At a glance: three GH-pathway therapies compared

All three work upstream of growth hormone, and none of them is growth hormone. Each asks the pituitary to release its own GH in pulses, so the body's feedback controls, including somatostatin and IGF-1, stay in play.

	Sermorelin	Tesamorelin	CJC-1295 / Ipamorelin two peptides, used together
Receptor	GHRH receptor	GHRH receptor	CJC-1295: GHRH receptor Ipamorelin: ghrelin receptor (GHS-R1a)
What the molecule is	First 29 amino acids of human GHRH, unmodified	Full 44-amino-acid GHRH with a <i>trans</i> -3-hexenoyl cap on the N-terminus	CJC-1295 (no DAC): sermorelin's 29-residue sequence with 4 engineered substitutions Ipamorelin: 5-residue peptide, 3 non-natural residues
Protection from DPP-4	None	Yes, from the cap	Yes (D-Ala2); ipamorelin has no DPP-4 site
Asn8 and Met27 weak points	Both present	Both present	Both removed (Gln8, Leu27); none in ipamorelin
Half-life	Roughly 10–20 min ¹⁴	Roughly 26–38 min ¹⁰	CJC-1295 (no DAC): about 30 min, commonly cited, not formally published Ipamorelin: about 2 h ⁴
IGF-1 response	Restored older men's IGF-1 toward young-adult levels in 14 days ¹²	About +81% after 26 weeks ⁶	1.5- to 3-fold after one dose of the long-acting DAC form ⁷ ; never measured with the pair
Clinical outcome shown	Growth in children with GH deficiency (approved use); small adult studies of lean mass ¹³	About 15% less visceral fat over 26 weeks ⁶	None tested

Figures come from different studies with different populations, doses and durations. They show that each molecule is active on the GH axis, not that the results are interchangeable. Compounded medications are not FDA-approved, and individual results vary.

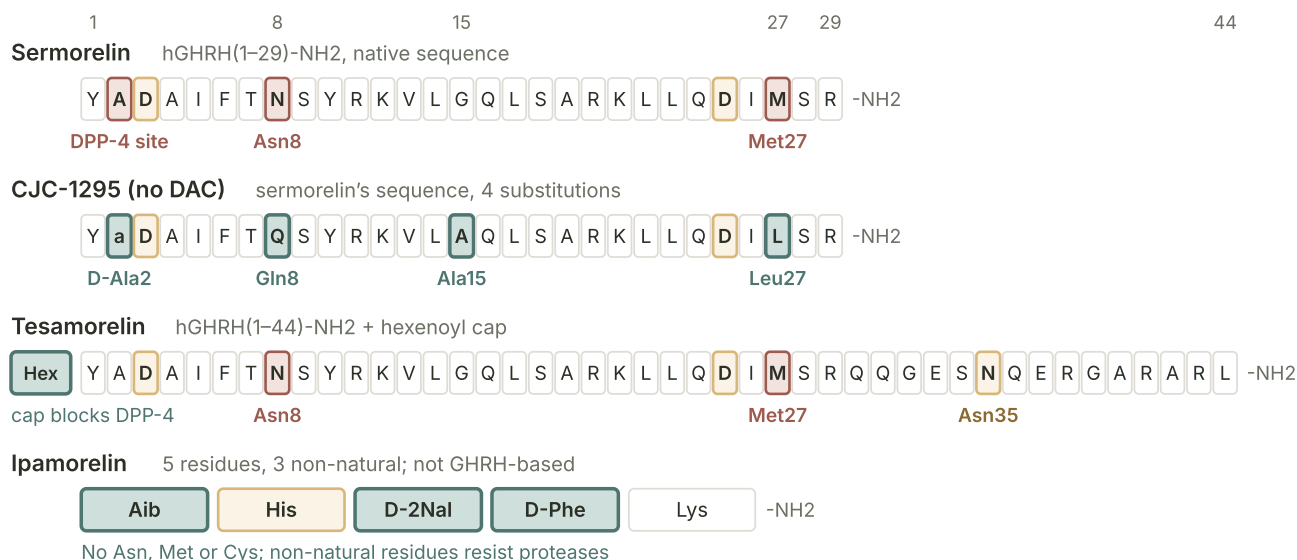
The four molecules, residue by residue

Three of the four are built on human growth hormone–releasing hormone (GHRH). Ipamorelin belongs to a separate class, derived from the ghrelin-mimetic peptide GHRP-1. In the one-letter code below, lowercase **a** is D-alanine, and every molecule ends in a C-terminal amide (-NH₂).

hGHRH (1–44) YADAIFTNSYRKVLGQLSARKLLQDIMSRQQGESNQERGARL - NH₂
Tesamorelin Hex - YADAIFTNSYRKVLGQLSARKLLQDIMSRQQGESNQERGARL - NH₂
Sermorelin YADAIFTNSYRKVLGQLSARKLLQDIMSR - NH₂
CJC-1295 no DAC YaDAIFTQSYRKVLAQLSARKLLQDILSR - NH₂
Ipamorelin Aib-His-D-2NaI-D-Phe-Lys - NH₂

Peptide	Residues	Built from	Key modifications	Mass g/mol, free base
Sermorelin	29	hGHRH(1–29)-NH ₂ , the shortest fully active fragment	None	≈ 3,358
CJC-1295 (no DAC) also called modified GRF(1–29)	29	hGHRH(1–29)-NH ₂	D-Ala2 (blocks DPP-4), Gln8 (no Asn deamidation), Ala15 (binding and potency), Leu27 (no Met oxidation)	≈ 3,368
Tesamorelin	44	Full-length hGHRH(1–44)-NH ₂	<i>trans</i> -3-hexenoyl cap on the N- terminus (blocks DPP-4)	≈ 5,136
Ipamorelin	5	GHRP-1 lineage; not GHRH-based	Aib1, D-2-naphthylalanine3, D-Phe4, C-terminal amide	≈ 712

Degradation hotspot
 Slower liability
 Engineered fix or protecting group



Positions line up vertically, so CJC-1295's four substitutions sit directly under sermorelin's weak points. Red: degradation hotspots (the DPP-4 cleavage site, asparagine 8 deamidation and methionine 27 oxidation). Amber: slower liabilities (aspartate isomerization at positions 3 and 25, asparagine 35 in tesamorelin, histidine oxidation in ipamorelin). Green: engineered substitutions and protecting groups.^{8,9}

1. Built for solution stability

Native GHRH breaks down in water by two main chemical routes. Asparagine at position 8 rearranges (deamidation and isomerization), and methionine at position 27 oxidizes. Both sharply reduce activity.⁸ For a ready-to-use solution, these are the reactions that set the shelf life. The four molecules handle them in four different ways.

Sermorelin is the native sequence, so it carries both weak points unprotected. Its stability in solution depends heavily on formulation.

Tesamorelin keeps the full native 44-residue sequence, so it still contains Asn8 and Met27, plus a second asparagine at position 35. The branded product manages this through formulation rather than structure: it is supplied as a lyophilized powder, and the newest version, Egrifta WR, holds for 7 days at room temperature after reconstitution, with a cyclodextrin excipient.¹⁰

CJC-1295 (no DAC) fixes the same weak points in the sequence itself. Glutamine replaces asparagine 8 and leucine replaces methionine 27. In the original analog work, substitutions of this kind measurably slowed degradation in solution.^{8,9}

Ipamorelin is a five-residue peptide, three of them non-natural (Aib, D-2-naphthylalanine and D-phenylalanine). It has no asparagine, methionine or cysteine, so it lacks the most common chemical degradation sites. Its one histidine is a minor oxidation risk.

Aggregation

Length and lipidation matter as well. At 44 residues with an N-terminal fatty-acid cap, tesamorelin has far more hydrophobic surface than the shorter molecules. Peptides of this kind tend to self-associate in solution, especially at higher concentrations or under agitation or temperature stress. Aggregation lowers the amount of active peptide and can increase the risk of immune reactions. Sermorelin and CJC-1295 (no DAC) are 29 residues with no lipid cap, and ipamorelin is far too small to aggregate in the same way. This is a structural tendency; published studies directly comparing aggregation of these peptides are not available.

Manufacturability

Shorter chains are also easier to make cleanly. Peptides are built one amino acid at a time, and each coupling step leaves a small fraction of incomplete chains. Those losses compound with every residue added.

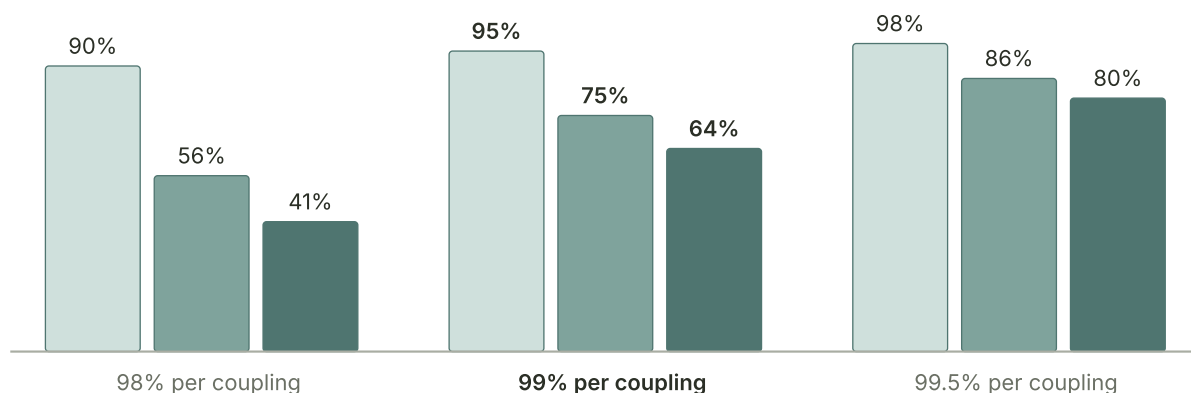
At 99% per coupling, a 44-residue chain keeps 64% full-length product; a 29-mer keeps 75%

Theoretical full-length fraction = coupling efficiency raised to the number of residues, before purification

Ipamorelin, 5 residues

Sermorelin and CJC-1295, 29 residues

Tesamorelin, 44 residues

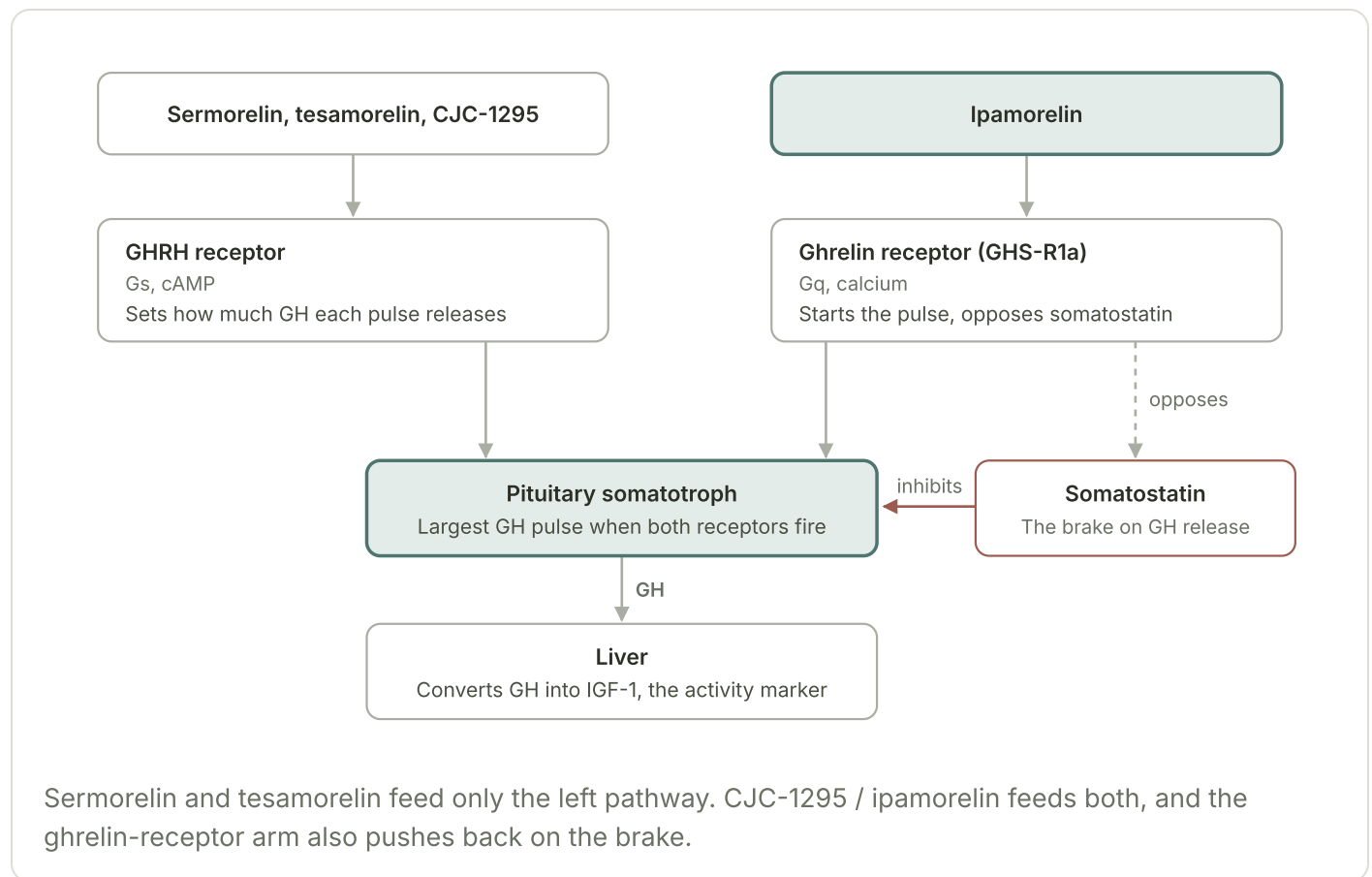


Calculated, not measured: coupling efficiency raised to the number of residues. Real yields depend on the sequence and the process, and every product is purified before release. Tesamorelin also needs an extra step to attach its N-terminal cap.

The CMC bottom line: on structure alone, CJC-1295 and ipamorelin were engineered to avoid liabilities that sermorelin carries unprotected and that tesamorelin relies on formulation to control. Structure predicts stability but does not prove it. For any ready-to-use solution, the deciding evidence is the compounding pharmacy's own stability-indicating assay data supporting the beyond-use date on the label.

2. Two receptor pathways

The pituitary's GH output is governed by two stimulatory signals. GHRH, released from the hypothalamus, acts on the GHRH receptor to set how much GH each pulse releases. Ghrelin acts on a separate receptor (GHS-R1a) to trigger pulses and to oppose somatostatin, the hormone that holds GH back. The two receptors signal through different pathways inside the cell.



The pair puts one agent on each receptor:

- **CJC-1295 (GHRH receptor):** a modified GHRH analog that increases the size of GH pulses. With continuous stimulation it raised baseline and average GH while preserving the body's normal pulse pattern.³

- **Ipamorelin (ghrelin receptor):** a selective agonist that produced a single, dose-dependent GH pulse in healthy volunteers, peaking about 40 minutes after dosing.⁴ In animal studies it released GH without the rises in cortisol and ACTH seen with older ghrelin-receptor peptides.⁵ Ipamorelin does not act on the GHRH receptor.

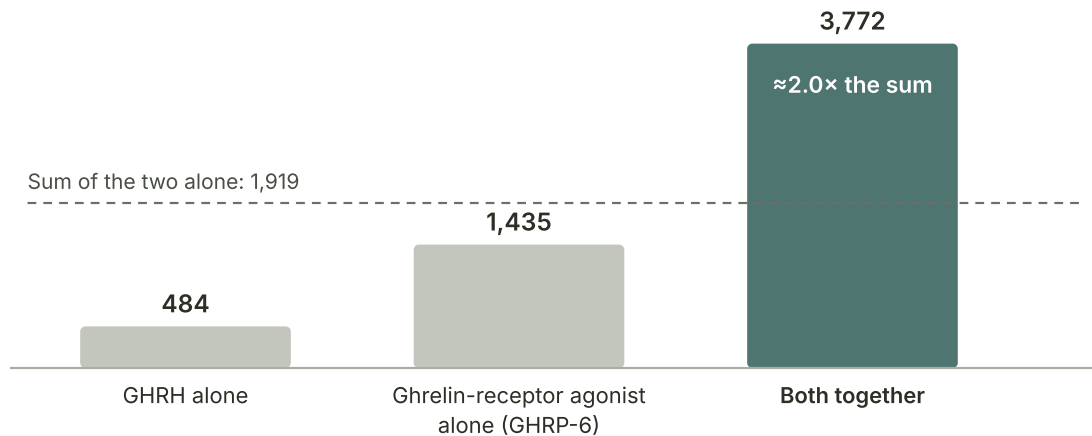
3. Strong GH stimulation, with the brake eased

Somatostatin is the brake on GH release. A GHRH-receptor drug pushes against that brake but cannot ease it. A ghrelin-receptor agonist works on both sides: it starts the pulse and counters somatostatin's hold on the pituitary. Because the two signals work through independent mechanisms, activating both at once does more than add their effects together.

That synergy is well established in humans. In 1990, Bowers and colleagues showed that a ghrelin-receptor peptide and GHRH given together released GH synergistically in healthy men.¹ Popovic and colleagues later measured total GH release in healthy control subjects:²

Together, the two pathways released about twice the sum of each alone

Total GH released (area under the curve) in healthy control subjects



Popovic et al., 1995 (reference 2): healthy control subjects given GHRH, GHRP-6, or both. The dashed line marks what simple addition would predict.

Keep in mind: the published synergy studies used GHRP-6, an older ghrelin-receptor agonist, not ipamorelin. The case for the pair rests on both drugs acting on the same two receptors, not on a direct trial of this pair.

What the GH and IGF-1 numbers show

GH and IGF-1 are the standard markers that the GH axis has been switched on. All three therapies have evidence on them:

- **Tesamorelin:** IGF-1 rose about 81% after 26 weeks of daily dosing in adults with HIV and abdominal fat.⁶
- **Sermorelin:** twice-daily injections for 14 days reversed the age-related drop in GH and IGF-1 in healthy older men.¹² Nightly dosing of a closely related GHRH(1-29) analog for 16 weeks raised nocturnal GH in older men and women, with anabolic effects that favored men.¹³
- **CJC-1295:** a single dose of the long-acting DAC form raised GH 2- to 10-fold for 6 days or more and IGF-1 1.5- to 3-fold for 9-11 days in healthy adults aged 21-61.⁷
- **Ipamorelin:** a dose-dependent GH pulse in healthy volunteers.⁴

These figures support the claim that the pair is biologically active on the same axis as sermorelin and tesamorelin. They do not show that it produces the same clinical results, for three reasons:

- the studies differ in population, duration and dosing
- the CJC-1295 data come from the long-acting DAC form, while most compounded preparations use the short-acting no-DAC form
- no published study has measured IGF-1 with the combined pair

4. Resists degradation, keeps potency

In the blood, the enzyme DPP-4 cuts native GHRH between positions 2 and 3 and inactivates it. That is why sermorelin, which carries the native sequence, has a half-life measured in minutes.¹⁴ Tesamorelin blocks the cut with its N-terminal cap. CJC-1295 blocks it with a single D-alanine at position 2.

CJC-1295 outlasts sermorelin; ipamorelin keeps signaling for about 2 hours

Elimination half-life, minutes. Whiskers show reported ranges; teal marks the pair; the hollow marker is a commonly cited value, not a published measurement.



Sermorelin: reference 14. Tesamorelin: prescribing information (reference 10). Ipamorelin: reference 4. CJC-1295 (no DAC): commonly cited figure; no formal pharmacokinetic study has been published.

CJC-1295's commonly cited half-life of about 30 minutes is roughly two to three times sermorelin's and in the same range as tesamorelin's. Ipamorelin, with no DPP-4 site and three non-natural residues, lasts about 2 hours in people.⁴

Staying intact is only half the story; the design also kept, and improved, activity. The Ala15 substitution strengthens receptor binding, and analogs built this way were several times more potent than native GHRH.⁸ Because Gln8 and Leu27 remove the deamidation and oxidation sites, that potency is also better protected in the vial.⁸

Longer is not automatically better. The short half-lives of all three GHRH-receptor drugs preserve the body's natural on-off GH pulses. The long-acting DAC form of CJC-1295, which lasts days, is a different molecule and is not the one discussed here.

What the evidence does not show

- No randomized trial has tested the combined CJC-1295 / ipamorelin pair.
- No clinical outcome, such as visceral fat, lean mass, sleep or recovery, has been shown for either peptide in humans.
- The strongest CJC-1295 human data are for the long-acting DAC form, not the no-DAC form discussed here.
- The stability advantages are predicted from structure. Published stability data for compounded solutions of these peptides are not available.
- Ipamorelin's one efficacy trial, for bowel recovery after surgery, found it well tolerated but no better than placebo.¹¹
- Sermorelin's adult studies are small and short, and women responded less than men.¹³

- Long-term safety of raising GH and IGF-1 with any of these peptides in healthy adults has not been established.

GH-pathway therapy at AJ Peptide

AJ Peptide offers sermorelin and tesamorelin by prescription, after an evaluation by a licensed clinician from our affiliated medical group, Arora Health & Aesthetics. Both are prepared by a named, licensed 503A compounding pharmacy as a ready-to-use solution, so there is nothing to mix. Your clinician will review your health history and may order baseline IGF-1 and blood sugar tests before deciding whether either is right for you. The evaluation is free.

Sermorelin

The gentler, shorter-acting signal that works with your body's own GH rhythm.

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[Compare sermorelin and tesamorelin side by side →](#)

Prescription required. Treatment is prescribed only if a licensed provider determines it is appropriate for you. Not right for everyone, including people who are pregnant or have active cancer. First-month price applies to your first month of therapy; one code per order.

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Revision history. Revision 2, October 23, 2026: reorganized around four CMC arguments; added the full amino acid sequences, a residue-level stability map, a receptor diagram, and GH-synergy, half-life and synthesis-yield charts. Revision 1, October 9, 2026: original white paper.

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