CAGRILINTIDE

AM833 | *First-in-Class Long-Acting Amylin Analog* | *Dual CTR/AMYR Agonist*

Novo Nordisk | NDA Filed Dec 2025 | FDA Decision Expected ~October 2026 | REDEFINE & REIMAGINE Programs

1. Peptide Description

- **Also known as:** AM833; NN9838; investigational compound by Novo Nordisk
- **Classification:** First-in-class long-acting amylin analog; dual calcitonin receptor (CTR) / amylin receptor (AMYR) agonist
- **Combination product:** CagriSema — fixed-dose combination of cagrilintide 2.4 mg + semaglutide 2.4 mg; NDA filed December 18, 2025; FDA decision expected ~October 2026
- **Structure:** 37 amino acid peptide (~4 kDa); based on the pramlintide (non-fibrillating) backbone rather than native amylin; key substitutions at N14E, V17R (alpha-helix stabilization via salt bridge) and Y37P (CTR/AMYR dual agonism); C-terminus amidated
- **Acyl chain modification:** C20 diacid lipid (dicarboxylic acid) at N-terminus via gamma-glutamine linker — enables albumin binding and extends half-life to ~7 days (159–195 hours)
- **Half-life:** ~159–195 hours (approximately 7 days); Tmax 24–72 hours post-injection; once-weekly SC dosing
- **Bioavailability:** SC subcutaneous administration; no renal or hepatic dose adjustment studies completed
- **Regulatory status:** NOT yet FDA-approved as of mid-2026; investigational compound; NDA filed December 2025 for CagriSema combination; cagrilintide monotherapy also under review
- **Key programs:** REDEFINE 1–3 (obesity phase 3); REIMAGINE 1–5 (T2D phase 3); 4.5-year extension studies planned
- **Non-fibrillating design:** Pramlintide backbone solves the primary challenge of native amylin drug development — spontaneous fibrillation at injectable concentrations; this design enables the clinical formulation

⚠ **INVESTIGATIONAL:** *Cagrilintide and CagriSema are investigational compounds. Neither is FDA-approved as of the publication of this guide. All clinical data presented reflects investigational trial findings. Not for clinical prescribing outside approved regulatory frameworks.*

2. Modes of Action & Mechanisms

Amylin physiology — the missing piece in incretin therapy

- Amylin is a 37-amino acid peptide co-secreted with insulin from pancreatic beta-cells in response to meals — it acts in parallel to insulin to regulate postprandial glucose
- Amylin is deficient in type 1 diabetes (beta-cell destruction eliminates co-secretion) and dysregulated in type 2 diabetes and obesity — this deficit is not addressed by GLP-1 RAs or GIP agonists
- Native amylin cannot be formulated as a drug due to spontaneous fibrillation; pramlintide (short-acting) was the first amylin analog approved; cagrilintide is the first long-acting weekly amylin analog

- Primary physiologic actions: glucagon suppression (postprandial), gastric emptying delay, hindbrain satiety signaling, and hedonic feeding circuit modulation

Dual CTR/AMYR receptor pharmacology

- **Amylin receptor subtypes:** AMY1R = CTR + RAMP1; AMY2R = CTR + RAMP2; AMY3R = CTR + RAMP3 — cagrilintide is a non-selective agonist at all subtypes
- **CTR (calcitonin receptor) component:** CTR in the brain, in complex with RAMPs, mediates the satiety and appetite-suppressing effects; this is distinct from the peripheral CTR on bone, which is responsible for calcitonin's osteoclast-inhibiting and calcium-lowering effects — cagrilintide does not produce clinically relevant bone or calcium effects
- **AMY1R (CTR + RAMP1):** Highest prevalence in the area postrema; primary mediator of cagrilintide-induced satiety; cFos activation reduced 57% in RAMP1/3 knockout mice
- **AMY3R (CTR + RAMP3):** Also required for weight effects; RAMP3 contributes uniquely to hindbrain amylin signaling; cagrilintide was specifically designed to retain AMY3R affinity
- **Bypass conformation:** Cagrilintide adopts a unique bypass confirmation at the CTR; the Y37P substitution enables this conserved CTR/AMYR engagement — a structurally distinct binding mode from other CTR ligands

Central nervous system circuit — the AP→NTS→LPBN satiety axis

- **Area postrema (AP):** The primary entry point — a circumventricular organ outside the blood-brain barrier that detects circulating peptides including cagrilintide; primary site of AMY1R/AMY3R expression mediating satiety
- **Nucleus tractus solitarius (NTS):** Signal relay from the area postrema; integrates peripheral satiety signals with vagal afferent input
- **Lateral parabrachial nucleus (LPBN):** The amplifier — integrates and propagates the satiety signal; requires both AMY1R and AMY3R; drives the subjective sensation of fullness and mild food aversion
- Hedonic feeding circuit: cagrilintide also modulates reward-driven (non-homeostatic) feeding — addressing the hedonic overconsumption that GLP-1 RAs target less specifically
- Energy expenditure: potential increase via CNS pathways — mechanistically plausible, not yet fully characterized in humans

Why amylin + GLP-1 = more than the sum of parts

- **Anatomically distinct brain targets:** Cagrilintide acts on the hindbrain (AP/NTS/LPBN); GLP-1 RAs (semaglutide) act on the hypothalamic arcuate nucleus and POMC/CART neurons — two separate satiety networks activated simultaneously
- Additive and potentially synergistic suppression of food intake — CagriSema weight loss (−22.7%) exceeds the arithmetic sum of cagrilintide alone (−11.8%) and semaglutide alone (−16.1%)
- Complementary glucagon suppression and gastric emptying delay — both pathways contribute; the combined effect is enhanced postprandial glucose control
- **Hedonic vs. homeostatic feeding:** Cagrilintide addresses reward-driven eating (hedonic); semaglutide addresses hunger signaling (homeostatic) — multi-axis coverage of the behavioral drivers of overconsumption

NOTE: *CagriSema's combination pharmacology is not redundant — it targets two anatomically and functionally distinct brain circuits. This is why the combination significantly exceeds semaglutide monotherapy: not because one drug 'adds to' the other at the same receptor, but because they engage parallel, complementary neural pathways that both constrain food intake.*

3. Main Points of Clinical Relevance

1 REDEFINE 1 — CagriSema achieves −22.7% weight loss, surpassing any approved agent

REDEFINE 1 (n=3,417, BMI ≥30 or ≥27 with comorbidity, no T2D, 68 weeks, NEJM 2025): CagriSema (2.4/2.4 mg weekly) achieved −20.4% weight loss (treatment-policy estimand) and −22.7% in fully adherent patients — vs. −14.9% for semaglutide alone and −11.5% for cagrilintide alone. Critically, 40.4% of CagriSema patients achieved ≥25% weight loss vs. 16.2% for semaglutide and 6.0% for cagrilintide alone. The comparison with semaglutide 2.4 mg is particularly important: CagriSema adds a meaningful ~6–8% additional weight loss on top of the current best-in-class GLP-1 RA, without the need for a fundamentally different receptor class. No weight loss plateau was observed at 68 weeks — the trajectory was still declining.

2 REIMAGINE — superior HbA1c reduction and weight loss in T2D vs. semaglutide alone

REIMAGINE 2 (n=2,728, T2D on metformin ± SGLT2i, Feb 2026): CagriSema achieved −14.2% weight loss and −1.91% HbA1c reduction (from baseline 8.2%); 74% achieved HbA1c ≤6.5% vs. 15.9% with placebo. In the REDEFINE 2 T2D cohort (n=1,206), 43% achieved ≥15% weight loss and 24% achieved ≥20% weight loss — outcomes that significantly exceed semaglutide monotherapy in T2D. These numbers reframe what is achievable in T2D weight management: outcomes previously requiring bariatric surgery are now pharmacologically attainable in a once-weekly injection.

3 A genuinely new mechanism — the amylin axis fills the gap left by incretin therapy

GLP-1 RAs have transformed metabolic treatment, but they engage only one of the body's satiety systems. Native amylin is deficient or dysregulated in the exact populations who need it most — T1D, T2D, and obesity. Cagrilintide restores this missing signal through a distinct hindbrain circuit (AP→NTS→LPBN) that GLP-1 RAs do not reach. This is not a 'more of the same' approach — it is additive pharmacology from a separate anatomical and receptor system. The clinical implication: patients who have reached their plateau on GLP-1 RA monotherapy have a biologically rational escalation path via amylin pathway addition, without simply increasing the GLP-1 dose and its associated GI burden.

4 Hedonic feeding modulation — addressing the behavioral driver GLP-1 RAs underserve

Cagrilintide modulates reward-driven (hedonic) eating behavior via its action on the LPBN and connected reward circuitry. This is distinct from the homeostatic hunger suppression provided by GLP-1 RAs. Hedonic overconsumption — eating driven by pleasure, habit, and food reward rather than hunger — is a major contributor to obesity that persists even when GLP-1-mediated hunger reduction is effective. By addressing both homeostatic (semaglutide) and hedonic (cagrilintide) feeding simultaneously, CagriSema covers the two primary behavioral drivers of caloric excess in obesity.

5 No weight loss plateau at 68 weeks — the trajectory is still active

In both REDEFINE 1 and REIMAGINE trials, no plateau in weight loss was observed at the 68-week endpoint — the curves were still declining. This distinguishes CagriSema from most GLP-1 RA data, where plateau typically occurs at 60–68 weeks. Whether this reflects sustained dual-pathway engagement or continued dose optimization is not yet fully characterized, but the clinical implication is significant: patients may continue to lose weight beyond the studied timeframe, and 4.5-year extension data are expected to clarify the long-term trajectory.

6 GI tolerability — higher absolute rates than GLP-1 RAs alone, but manageable with slow escalation

CagriSema GI adverse events: 79.6% vs. 39.9% placebo (REDEFINE 1) — higher than semaglutide monotherapy rates (~40–44%). Nausea is the most common; vomiting, diarrhea, constipation, and abdominal pain are also reported. Serious adverse events were low and consistent with the GLP-1 RA class. The key clinical reality: virtually all GI adverse events are mild-to-moderate, diminish over time, and are dramatically reduced by slow titration. The 16-week escalation schedule should be treated as the minimum — not the target. Many patients tolerate the combination well at sub-maximal doses (1.0–1.7 mg cagrilintide) without needing to reach 2.4 mg; staying at an effective lower dose is clinically rational.

7 Patient selection: the GLP-1 RA 'plateau' patient is the ideal candidate

The clearest clinical indication for cagrilintide is the patient who has an inadequate response to GLP-1 RA monotherapy — either insufficient weight loss, a plateau reached before goal, or GI intolerance limiting GLP-1 dose escalation. Adding the amylin pathway via cagrilintide provides dual-axis coverage

without increasing the GLP-1 burden. For new patients with obesity and T2D who need both glycemic and weight management, CagriSema as the initial agent offers the highest weight loss and HbA1c reduction of any approved or pending agent. Practical expectation-setting: cagrilintide monotherapy delivers ~10–12% weight loss; CagriSema combination delivers ~20–23%.

4. Dosing Instructions & Delivery Options

Cagrilintide monotherapy escalation

Phase	Dose	Duration	Notes
Initiation	0.25 mg SC weekly	Weeks 1–4	Adaptation dose — not therapeutic; establishes GI tolerance baseline
Escalation 1	0.50 mg SC weekly	Weeks 5–8	First therapeutic dose; advance only if tolerated
Escalation 2	1.0 mg SC weekly	Weeks 9–12	Many patients find effective dose here; consider holding longer
Escalation 3	1.7 mg SC weekly	Weeks 13–16	Approaching maintenance; continue hold if any GI burden
Maintenance	2.4 mg SC weekly	Week 17+	Target dose; many patients achieve goals without reaching this
Max monotherapy	4.5 mg SC weekly	As needed	Phase 2 maximum; only ~50% of trial patients reached max dose

CagriSema combination dosing

Regimen	Detail	Notes
Fixed combination	Cagrilintide 2.4 mg + semaglutide 2.4 mg	Single weekly SC injection; abdomen is the preferred site
Escalation protocol	Same 16-week step schedule as monotherapy	Titrate slowly; combination GI burden is additive — go slower, not faster
Flexible approach	Hold at lower cagrilintide dose (1.0–1.7 mg) if combining with GLP-1 RA	Many combinations work well at sub-maximal cagrilintide doses
Stopping cagrilintide	Can discontinue cagrilintide and continue GLP-1 RA alone at maintenance	Logical de-escalation once metabolic goals achieved

Key dosing principles: The 16-week escalation schedule is a minimum — not a target pace. In clinical practice, slower progression produces dramatically better GI tolerability and no meaningful reduction in long-term efficacy. Target 0.5–0.75 lbs/week weight loss as the titration guide. For combination use, lower cagrilintide doses (1.0–1.7 mg) combined with the GLP-1 RA often achieve excellent outcomes without needing the full 2.4 mg — avoid escalating for its own sake. Approximately 50% of patients in REDEFINE 1 did not reach the 4.5 mg max monotherapy dose; efficacy at lower doses is clinically meaningful. Abdomen is the preferred injection site. Refrigerate; pen may be used at room temperature for up to 21 days. Baseline eye exam at initiation; annual thereafter.

5. Evidence Profile

Evidence tier legend: ● *Human RCT / clinical trial* ○ *Animal / preclinical* ◎ *Structural / in vitro* ✕ *Critical gap* ~ *Theoretical / emerging*

◎ Dual CTR/AMYR agonism confirmed; bypass conformation at CTR; AMY1R/AMY3R binding dynamics; Y37P substitution enabling dual receptor engagement (Cao 2025, Nat Commun; Kruse 2021, J Med Chem)*Structural / in vitro*

○ AMY1R/AMY3R requirement for cagrilintide weight effects confirmed; RAMP1/3 KO mice: cFos −57% in area postrema; weight loss abolished (Oliveira Carvas 2025, EBioMedicine)*Animal study*

● Phase 2 dose-finding (n=706, BMI 27–40, 26 weeks): −10.8% weight at 4.5 mg; 1.2–2.4 mg comparable to liraglutide 3.0 mg; no serious safety signals (Lau 2021, Lancet)*Human RCT*

● Phase 1b combination study: cagrilintide 0.16–4.5 mg + semaglutide 2.4 mg; −15.6% weight (2.4/2.4 mg); no PK interaction; t½ 159–195 hours; supports Phase 3 (Enebo 2021, Lancet)*Human RCT*

● REDEFINE 1 (n=3,417, obesity, no T2D, 68 weeks, NEJM 2025): CagriSema −20.4% (policy) / −22.7% (adherent); sema alone −14.9%; cagrilintide alone −11.5%; 40.4% achieved ≥25% WL (Garvey 2025)*Human RCT — Phase 3 pivotal*

● REDEFINE 1: no weight loss plateau at 68 weeks; 91.4% of CagriSema patients achieved ≥5% weight loss; GI AEs 79.6% vs 39.9% placebo; no QTc prolongation*Human RCT — Phase 3*

● REDEFINE 2 (n=1,206, T2D, BMI ≥27, 68 weeks): CagriSema −13.7% weight (policy) / −15.7% (adherent); 74% achieved HbA1c ≤6.5%; 43% achieved ≥15% WL; 24% achieved ≥20% WL*Human RCT — Phase 3*

● REIMAGINE 2 (n=2,728, T2D on metformin ± SGLT2i, Feb 2026): CagriSema −14.2% weight; HbA1c −1.91% from baseline 8.2%; superior to semaglutide alone on both endpoints*Human RCT — Phase 3*

● QTc safety study: no clinically relevant QTc prolongation with cagrilintide (Haahr 2024, Diabetes Obes Metab)*Human RCT*

~ Cardiovascular outcomes: REDEFINE 3 (dedicated MACE trial) ongoing — results pending*Emerging / investigational*

~ Head-to-head vs. tirzepatide: REIMAGINE 4–5 trials planned — results not yet available*Emerging / investigational*

~ MASH/NASH resolution: biologically plausible given weight loss magnitude; not yet formally studied*Theoretical*

✕ Dedicated CV outcomes trial results — REDEFINE 3 pending*Critical gap*

✕ Long-term safety beyond 68 weeks — 4.5-year extensions planned but not reported*Critical gap*

✕ Lean mass preservation profile — not fully characterized; combination data limited*Critical gap*

✕ Optimal patient phenotype for amylin-based vs. incretin-based therapy — not established*Critical gap*

✕ Post-discontinuation weight regain trajectory — not characterized for cagrilintide specifically*Critical gap*

Comparative weight loss — placing cagrilintide in context

Agent	Mechanism	Weight Loss	Duration	Evidence
CagriSema 2.4/2.4 mg	Amylin + GLP-1	−22.7% (adherent)	68 weeks	Phase 3 RCT (REDEFINE 1)
Tirzepatide 15 mg	GIP + GLP-1	−20.9%	72 weeks	Phase 3 RCT (SURMOUNT-1)
Semaglutide 2.4 mg	GLP-1	−14.9%	68 weeks	Phase 3 RCT (STEP 1)
Cagrilintide 2.4 mg	Amylin analog	−11.8%	68 weeks	Phase 3 RCT (REDEFINE 1)
Liraglutide 3.0 mg	GLP-1	−8.0%	56 weeks	Phase 3 RCT (SCALE)

6. Clinical Considerations

Contraindications

- Personal or family history of medullary thyroid carcinoma (MTC) — contraindicated (class warning shared with GLP-1 RAs)
- Multiple Endocrine Neoplasia type 2 (MEN2) — contraindicated
- Active or recent pancreatitis — use with caution; assess risk-benefit individually
- Severe GI disease or gastroparesis — may be exacerbated by gastric emptying delay; use with caution or avoid
- Concurrent GLP-1 RAs or other amylin analogs (e.g., pramlintide) — not recommended; additive class effects without safety data
- Pregnancy and lactation — insufficient data; not recommended; discontinue before planned conception

Patient selection

- **Primary candidates (investigational):** Obesity (BMI ≥30) or overweight (BMI ≥27 with comorbidity — hypertension, dyslipidemia, T2D, OSA) with inadequate response to GLP-1 RA monotherapy; T2D patients seeking simultaneous glycemic control and meaningful weight loss; patients where GLP-1 dose escalation is GI-limited
- **Strongest clinical case:** The patient already on semaglutide or liraglutide who has plateaued below their weight loss goal — adding cagrilintide engages a separate brain circuit without increasing GLP-1 burden
- **Practical expectation-setting:** Cagrilintide monotherapy ~10–12% weight loss; CagriSema combination ~20–23% — communicate both to set realistic goals based on planned regimen
- **Screen before initiating:** MTC/MEN2 history; pancreatitis history; baseline weight, HbA1c, lipids, BP, waist circumference, CMP, liver enzymes; retinal exam; GI symptom history

Monitoring protocol

Timepoint	Labs / Assessments	Clinical Focus
Baseline	Body weight, BMI, waist circumference, HbA1c (T2D), fasting glucose, CMP, CBC, lipid panel, liver enzymes, BP, heart rate; thyroid history (MTC/MEN2); pancreatitis history; calcitonin if indicated; retinal exam; DEXA or InBody	Exclude contraindications; establish full metabolic baseline
Week 4	Body weight, BP, heart rate, GI symptom diary review	Tolerability check; dose escalation decision
Week 8–12	Body weight, waist circumference, fasting glucose, BP, GI symptom review	Intermediate efficacy; dose progression decision
Month 3	HbA1c (T2D), fasting glucose, body weight, waist circumference, BP, lipid panel	Glycemic and lipid response; dose optimization
Month 6	Full metabolic panel, HbA1c, lipid panel (triglycerides), liver enzymes, eGFR, body composition (DEXA or InBody)	Comprehensive intermediate assessment; hepatic monitoring
Month 12+	Full panel as above + retinal exam + calcitonin if indicated + cardiac assessment if clinically indicated	Annual safety and efficacy review

Timepoint	Labs / Assessments	Clinical Focus
Every visit	Body weight, waist circumference, BP, GI symptom review, hydration status, weight loss rate (target 0.5–0.75 lbs/week)	Ongoing safety, titration pacing, dose appropriateness

Note: CagriSema reduces systolic blood pressure over time — monitor at every visit and adjust antihypertensive therapy proactively. Maintain GI symptom diary during the 16-week escalation period; this is the highest-risk window for adverse events. Hypoglycemia risk is low unless cagrilintide or CagriSema is combined with insulin or sulfonylureas — reduce glucose-lowering agent doses proactively.

Drug interactions & practical cautions

Interaction / Caution	Detail
GLP-1 RA combination	CagriSema is the validated combination; do not add cagrilintide to a different GLP-1 RA without clinical rationale — additive GI effects and no comparative safety data for non-semaglutide combinations
Insulin / sulfonylureas	Reduce doses when initiating CagriSema — glucagon suppression and gastric slowing amplify hypoglycemia risk from these agents; monitor glucose closely
Pramlintide (Symlin)	Concurrent use not recommended — redundant amylin mechanism with additive GI burden and no clinical data supporting dual amylin analog use
SGLT2 inhibitors	Complementary — REIMAGINE 2 included patients on metformin ± SGLT2i; compatible combination; monitor for dehydration given combined GI fluid losses and renal glucose excretion
Oral medications (narrow TI)	Delayed gastric emptying affects oral drug absorption; administer narrow therapeutic index drugs (levothyroxine, warfarin, certain antibiotics) ≥1 hour before injection
Dehydration / AKI	GI adverse events (nausea, vomiting) cause fluid loss; active hydration counseling is essential; monitor eGFR if prolonged GI symptoms occur
Weight loss pace	Target 0.5–0.75 lbs/week to preserve lean mass and minimize aesthetic concerns; faster weight loss should prompt dose evaluation not celebration
Injection technique	Abdomen preferred for both cagrilintide monotherapy and CagriSema; rotate sites; same day each week; refrigerate — pen stable at room temperature (≤30°C) for 21 days

Managing GI side effects — the titration imperative

- Slow titration is the primary GI management strategy — more effective than any antiemetic and more important here than with GLP-1 RA monotherapy given the additive burden of the amylin pathway
- GI adverse events diminish substantially over time at stable doses — counsel patients that the escalation phase is the hardest, and it passes
- Small, frequent meals during escalation; avoid high-fat, greasy, or spicy foods
- Active hydration — GI fluid losses must be replaced; minimum 2L water daily during escalation
- If nausea is limiting: hold current dose for an additional 2–4 weeks rather than escalating; temporary reduction to the prior dose is preferable to discontinuation
- Anti-emetics (short-term) are an acceptable bridge during dose escalation if needed — do not allow avoidable GI burden to derail an otherwise appropriate therapeutic plan

- Approximately 50% of patients in REDEFINE 1 did not reach the 4.5 mg monotherapy maximum — efficacy at 1.0–2.4 mg is clinically meaningful; not reaching maximum dose is not a failure

Clinical bottom line: Cagrilintide is the first clinically validated long-acting amylin analog — and CagriSema is the most efficacious weight management regimen ever studied in a Phase 3 RCT, achieving −22.7% weight loss at 68 weeks in fully adherent patients. It is not a variation on GLP-1 RA pharmacology; it is a genuinely additive mechanism acting on a separate brain circuit (hindbrain AP→NTS→LPBN) that GLP-1 RAs do not reach. The clinical case is clearest for patients who have plateaued on GLP-1 RA monotherapy — adding the amylin pathway without increasing the GLP-1 dose provides a mechanistically rational and evidence-supported escalation. Awaiting FDA approval (~October 2026); prescribe within investigational frameworks only until approved. The titration discipline that applies to all agents in this series is especially critical here: slow escalation, 0.5–0.75 lbs/week weight loss target, and active GI symptom management during the 16-week escalation window.

Final Note: Where Cagrilintide stands in the metabolic peptide landscape and what it means for the series

Cagrilintide completes a picture that GLP-1 RAs and tirzepatide could not finish on their own. Liraglutide established that GLP-1 RAs were more than glucose-lowering drugs. Semaglutide proved they could approach bariatric weight loss outcomes. Tirzepatide added the GIP pathway and pushed weight loss further. Cagrilintide adds the amylin axis — the third major gut-derived satiety hormone — and with it, access to a hindbrain circuit that neither GLP-1 nor GIP reaches.

Head-to-head context: CagriSema (−22.7%) and tirzepatide 15 mg (−20.9%) are now in the same weight loss territory — but via completely different mechanisms. REIMAGINE 4–5 will provide the direct comparison. The mechanistic implication is that combination of tirzepatide with cagrilintide may represent the next frontier, though no data yet supports this. Triple agonist retatrutide (GLP-1/GIP/glucagon) is also in Phase 3, adding thermogenesis to the mix.

In the context of this series: for the practitioner using GH secretagogues (ipamorelin, GHRP-2, MK-677) alongside metabolic agents, cagrilintide and CagriSema represent the highest tier of metabolic optimization currently achievable. Greater weight loss, improved insulin sensitivity, AMPK activation from both the GLP-1 and amylin pathways, and reduced hedonic food drive all create the optimal cellular environment for anabolic peptide strategies to work effectively. The sequencing principle — metabolic optimization first, anabolic support second — has never had a more powerful tool at its disposal than CagriSema.