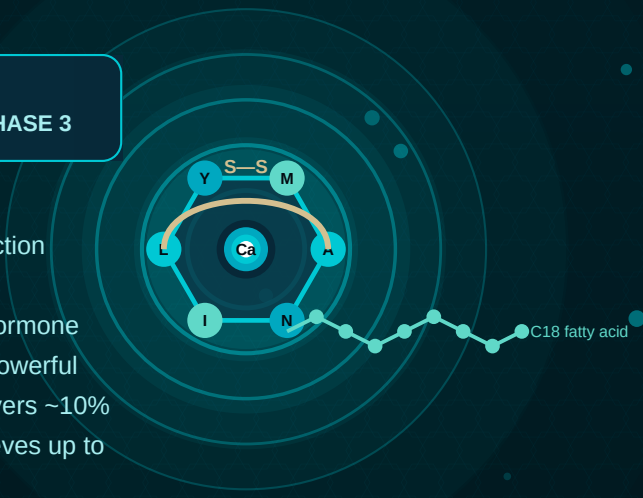
LONG-ACTING AMYLIN ANALOGUE · ONCE-WEEKLY INJECTION · NOVO NORDISK

CAGRI-LINTIDE

A Different Path to Weight Loss.

The Amylin Advantage.

AMYLIN RECEPTOR · NOVEL PATHWAY · COMBO POTENTIAL · PHASE 3



Cagrilintide is a **long-acting amylin analogue** — a once-weekly injection developed by Novo Nordisk that works through a completely different mechanism to GLP-1 and GIP agents. By mimicking the pancreatic hormone **amylin**, it slows gastric emptying, suppresses glucagon, and sends powerful satiety signals directly to the brain's area postrema. On its own it delivers ~10% weight loss — but combined with semaglutide as **CagriSema**, it achieves up to **22.7%**, rivalling the best in class.

~10%	~23%	Amylin	Weekly
SOLO WEIGHT LOSS	WITH CAGRISEMA	NOVEL PATHWAY	CONVENIENT DOSING

What Is Cagrilintide?

The only long-acting amylin analogue — and why the pancreas holds a hidden weight-loss key

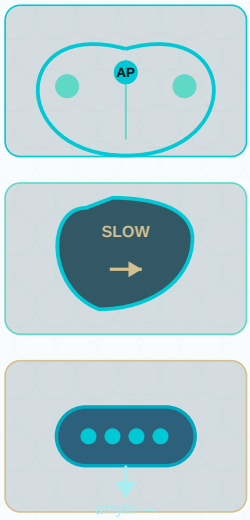
Amylin — The Forgotten Satiety Hormone

Every time you eat, your pancreatic beta cells release two hormones together: **insulin** and **amylin**. While insulin has been the focus of diabetes medicine for a century, amylin's role in weight and metabolism has only recently been appreciated. Amylin slows the rate at which food leaves the stomach, suppresses the glucagon spike that follows a meal, and — crucially — signals directly to the **area postrema** in the brain stem to produce a strong, sustained sense of fullness. In people with obesity and type 2 diabetes, amylin signalling is impaired. Cagrilintide restores it.

From Hours to a Week — Engineering Durability

Natural amylin has a half-life of just a few minutes. Pramlintide — the first synthetic amylin — required injections three times a day with meals, limiting its clinical uptake. Cagrilintide solves this with a **C18 fatty acid chain** that binds to albumin in the bloodstream, dramatically extending its half-life to approximately one week. This makes it the first and only amylin analogue suitable for once-weekly dosing — and the first to truly unlock amylin's therapeutic potential for long-term weight management.

How Cagrilintide Works — Three Key Actions



BRAIN SATIETY

Activates area postrema receptors in the brain stem — producing a direct, sustained satiety signal independent of GLP-1 pathways.

GASTRIC SLOWDOWN

Slows gastric emptying, extending the time food remains in the stomach and prolonging the feeling of fullness after a meal.

PANCREAS SUPPORT

Suppresses the post-meal glucagon surge from the pancreas, reducing excess glucose release and supporting stable blood sugar.

The CagriSema Advantage

Because cagrilintide and semaglutide work through **entirely separate receptors** — amylin vs GLP-1 — combining them produces additive, non-overlapping effects. The SCALE NEXT trial showed CagriSema 2.4/2.4 mg delivered average weight loss of **22.7%** over 68 weeks — comparable to tirzepatide — while offering a complementary mechanism that may prove advantageous in patients who develop tolerance or partial response to GLP-1 agonists alone.

★ A COMPLETELY DIFFERENT MECHANISM TO GLP-1 DRUGS

Cagrilintide doesn't compete with GLP-1 agonists — it complements them at the receptor level. This makes it uniquely valuable for combination therapy and potentially for patients who respond inadequately to GLP-1 treatment alone, opening a new lane in obesity pharmacology.

The Benefits of Cagrilintide

A novel amylin pathway — standalone benefits and game-changing combination potential

01 Meaningful Standalone Weight Loss

As a monotherapy, cagrilintide delivers approximately 10% weight loss over 26 weeks — significant for an amylin analogue acting through a single novel pathway, and well-tolerated compared to GLP-1 agents.

02 Transformative Combination Potential

When paired with semaglutide as CagriSema, weight loss reaches ~22.7% — driven by the additive, non-overlapping effects of dual amylin + GLP-1 receptor activation. This combination may outperform either agent alone more than proportionally.

03 Brain-Mediated Satiety — A Different Feeling of Fullness

Cagrilintide's satiety signal acts via the area postrema in the brainstem — producing a slower, more sustained feeling of fullness that is qualitatively different from the GLP-1-mediated appetite suppression, potentially addressing different dimensions of hunger.

04 Slowed Gastric Emptying

By slowing the rate of gastric emptying, cagrilintide extends the post-meal satiety window, reduces post-prandial blood sugar spikes, and makes patients feel satisfied with smaller meal portions for longer.

05 Post-Meal Glucagon Suppression

Cagrilintide suppresses the glucagon surge that typically follows a meal — preventing excess hepatic glucose release and contributing to improved overall glycaemic control, complementary to the insulin-stimulating effects of GLP-1 agonists.

06 Potential for GLP-1-Resistant Patients

Because cagrilintide acts through a completely separate receptor system, it may provide meaningful weight loss benefit in patients who show partial or inadequate response to GLP-1 or GIP/GLP-1 therapies — addressing a significant unmet clinical need.

07 Favourable Tolerability Profile

Clinical data show cagrilintide is generally well tolerated, with a GI side effect profile that is broadly comparable to semaglutide — and when used in combination, the tolerability of CagriSema has been acceptable across the SCALE NEXT trial population.

08 Expanding the Obesity Medicine Toolkit

Cagrilintide represents the first entirely new mechanism class for obesity pharmacotherapy since GLP-1 agonists. Its unique amylin pathway diversifies the tools available for combination treatment strategies and opens a new chapter in metabolic medicine.

Cagrilintide doesn't just add another GLP-1 drug to the shelf. It opens an entirely new receptor pathway — one that works with, not against, the established agents. Whether alone or as CagriSema, it represents a genuinely new approach to metabolic medicine at a moment when the field needs exactly that.