Biomea Fusion Corporate Presentation

The Citizens Life Sciences Conference
March 10, 2026

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Transforming diabetes and obesity with novel oral medicines

Biomea Fusion founded in 2017 (public in 2021; NASDAQ: BMEA)
Clinical-stage company advancing two differentiated metabolic investigative programs

ICOVAMENIB

Potential first-in-class oral small molecule targeting menin - the control switch to beta cell restoration
Restores functional beta-cell mass to address disease biology in type 2 diabetes

Critical unmet need: 1/3 of all diabetes patients fail standard of care and progress to insulin dependence driving complications such as kidney disease, nerve damage, vision loss, and cardiovascular issues.[1-3]

BMF-650

Next-generation oral GLP-1 receptor agonist
Designed for consistent exposure, higher bioavailability and improved tolerability with scalable weight reduction

Critical unmet need: Real world evidence indicates that up to 70% of patients on currently available GLP-1 based therapies drop out within the first year due to gastrointestinal adverse events and other tolerability considerations.[4]

Biomea funded through key clinical readouts for icovamenib and BMF-650 into Q1 of 2027.

1. Scherer et al., Scientific Reports, 2020; 2. Nichols et al., Diabetes Care, 2015; 3.UKPDS Group. Lancet, 1998; 4. Prime Therapeutics & Magellan Rx Management, 2023 real-world claims analysis.

ICOVAMENIB

Potential First-in-Class Menin Inhibitor For Diabetes

Preclinical and first clinical results

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Diabetes patients are poorly controlled with 1:3 US diabetes patients estimated to require insulin as a last resort

Icovamenib targets menin to allow for beta-cell restoration which may delay or prevent onset of end-stage disease

80%

of people with diabetes will die from the disease[1]

The end-stage in the evolution of diabetes is insulin-dependence, which drives complications such as kidney disease, nerve damage, vision loss, and cardiovascular issues.

12-14 years

of life lost from diabetes[2]

Diabetes today remains poorly controlled in 50% of patients treated with standard of care agents[3] The burden to the healthcare system is immense. There is no current therapy except for insulin replacement

60+

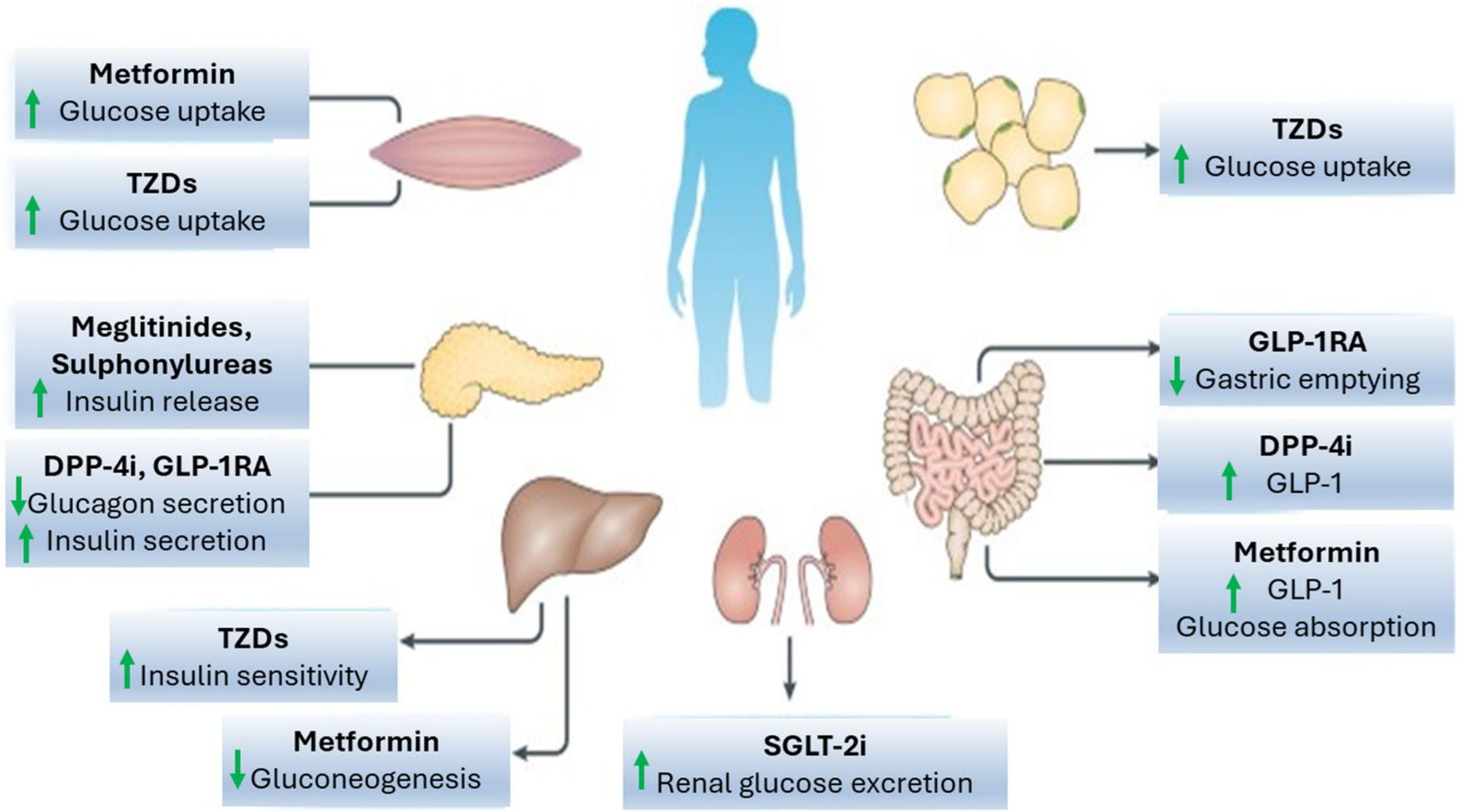
Approved therapies are not adequately resolving the growing problem of type 2 diabetes.

No current therapy restores beta-cell function

1.Tabish Int J Health Sci. 2007 Jul;1(2):V–VIII.
2.National library of Medicine 1(2); 2007 Jul PMC3068646
3.Zohu Lancet 2024; 404:2077–93

4. CDC, Natl. Diabetes Stat. Rep., 2022
5. ADA, Standards of Care in Diabetes, Diabetes Care, 2024
6. Li J Diabetes Complications 2012;26(1):17-22

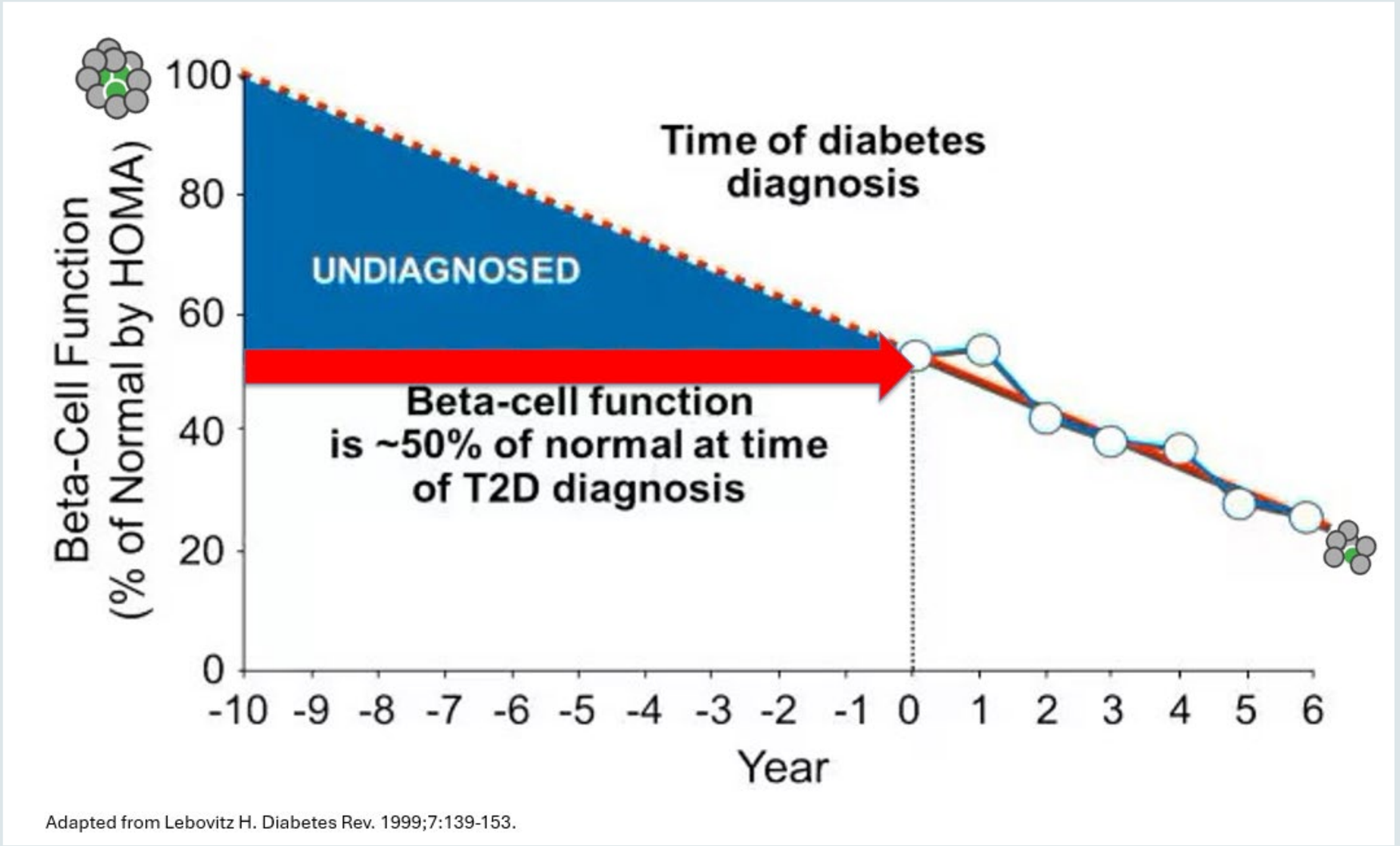
Current type 2 diabetes therapies are targeting the symptoms of hyperglycemia not the root cause



Nat Rev Endocrinol. 2016;12:337–346

- Existing Type 2 Diabetes therapies primarily address downstream metabolic symptoms
- **Icovamenib** is a potentially first-in-class menin inhibitor, targeting a previously unaddressed mechanism.
- **Icovamenib** can induce transient reductions in menin protein levels in relevant tissues (e.g. pancreatic islets) and thereby modulate menin regulated pathways.

Progressive decline in beta-cell mass and function is the root cause of diabetes



Adapted from Lebovitz H. Diabetes Rev. 1999;7:139-153.

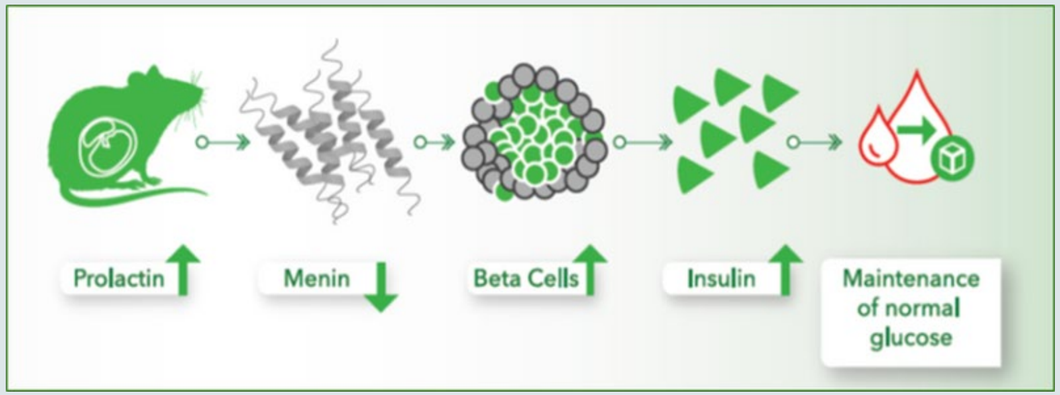
Physiologic suppression of menin can expand beta cell mass

- Physiologic states such as pregnancy and lactation suppress menin, enabling beta-cell expansion and increased insulin output

- Preclinical and human data consistently link reduced menin signaling to improved beta-cell mass and function.

First in a 2005 paper in Proceedings of the National Academy of Sciences (PNAS) by Satyajit K. Karnik et al. titled "Menin regulates pancreatic islet growth by promoting histone methylation and expression of genes encoding p27^Kip1 and p18^INK4c"

- Icovamenib has been shown to directly inhibit menin, aiming to pharmacologically replicate a naturally occurring, validated biologic process



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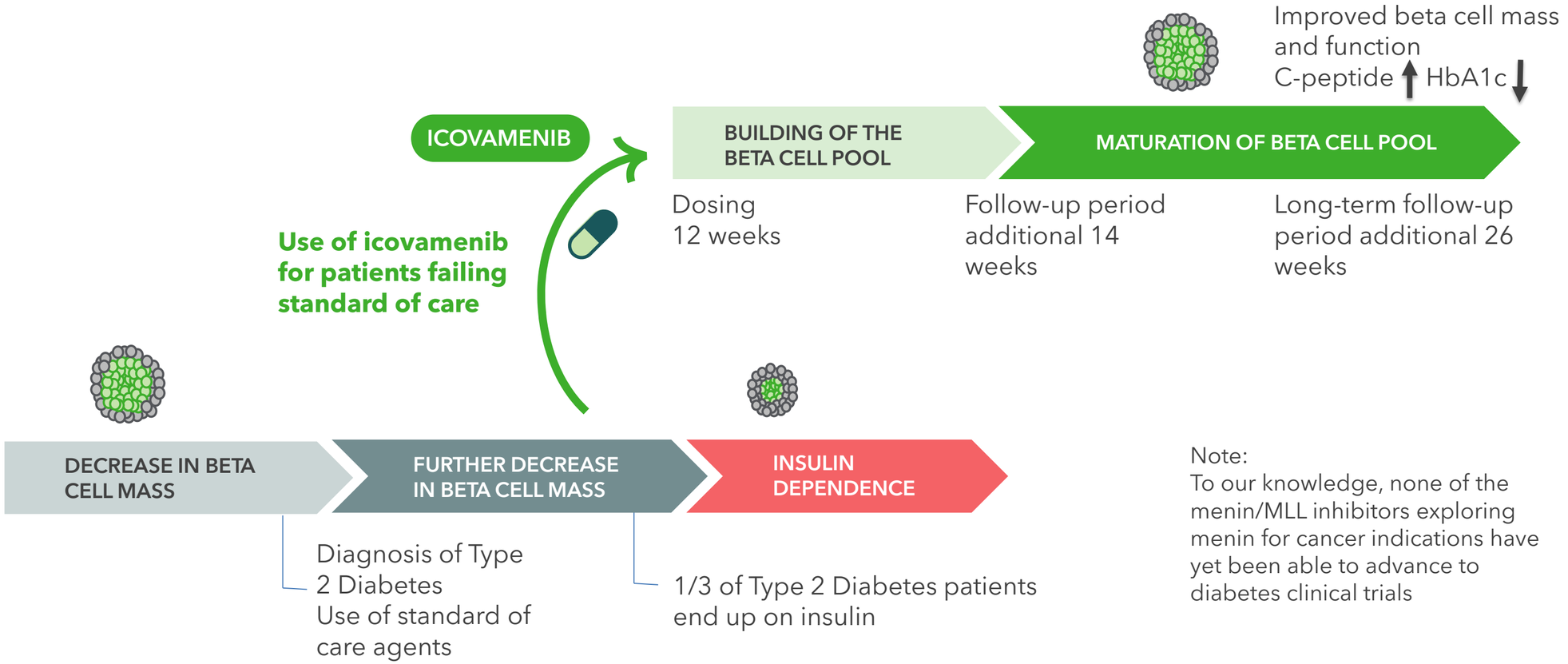
Icovamenib increased beta cell quantity and function

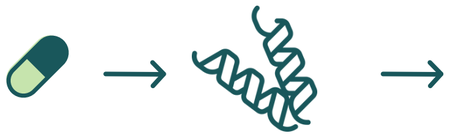
demonstrating long term results post the initial dosing period



Note:
To our knowledge, none of the menin/MLL inhibitors exploring menin for cancer indications have yet been able to advance to diabetes clinical trials

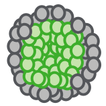
Icovamenib's mechanism of action

Icovamenib promotes selective and partial reduction in menin levels



Lowering menin levels releases the break on beta cell proliferation and GLP-1 and GLP-1 receptor expression

Induction of beta cell proliferation



Pancreatic islet

Increased expression of
- **GLP-1**
- **GLP-1 receptor**

IMPROVED BETA CELL MASS & FUNCTION

- ↑ Increase in insulin/C-peptide
- ↓ Decrease in blood glucose (HbA1c)

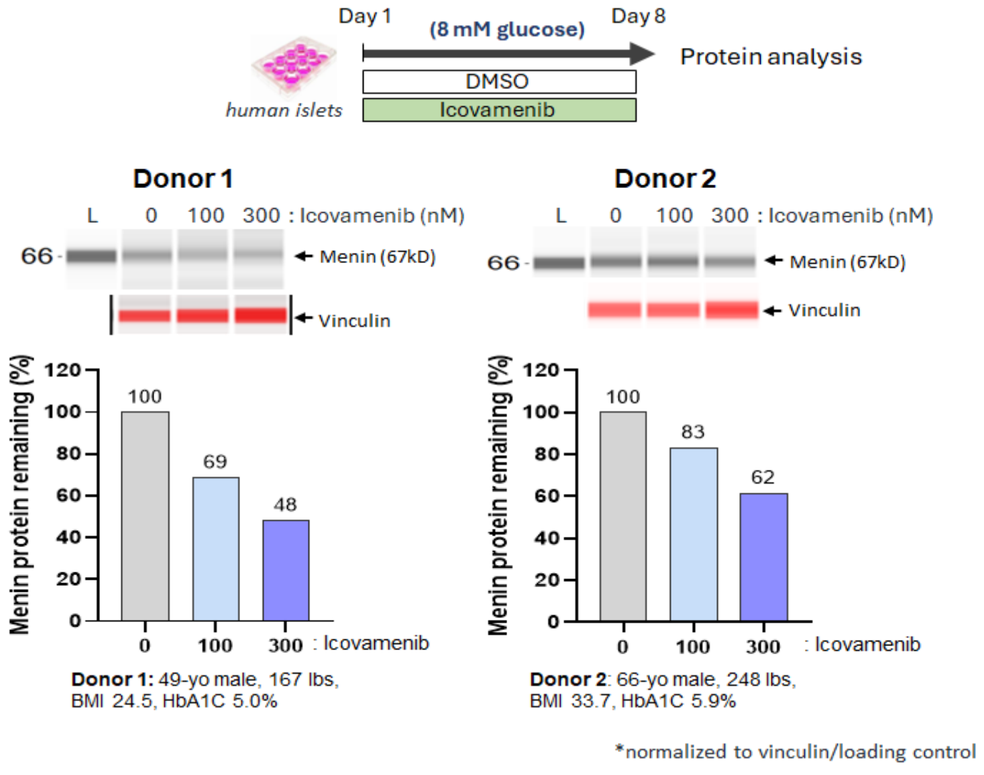
IMPROVED INCRETIN EFFECTS

- ↑ Synergistic with GLP-1 based therapies
- ↑ Improved beta cell function
- ↓ Decrease in blood glucose (HbA1c)
- ↓ Potential for weight loss
- ↑ Potential for lean mass/muscle preservation

ICOVAMENIB | PRECLINICAL

Icovamenib downregulated menin protein levels & promoted beta cell proliferation in ex vivo human islet cultures

MENIN LEVELS DOWNREGULATED

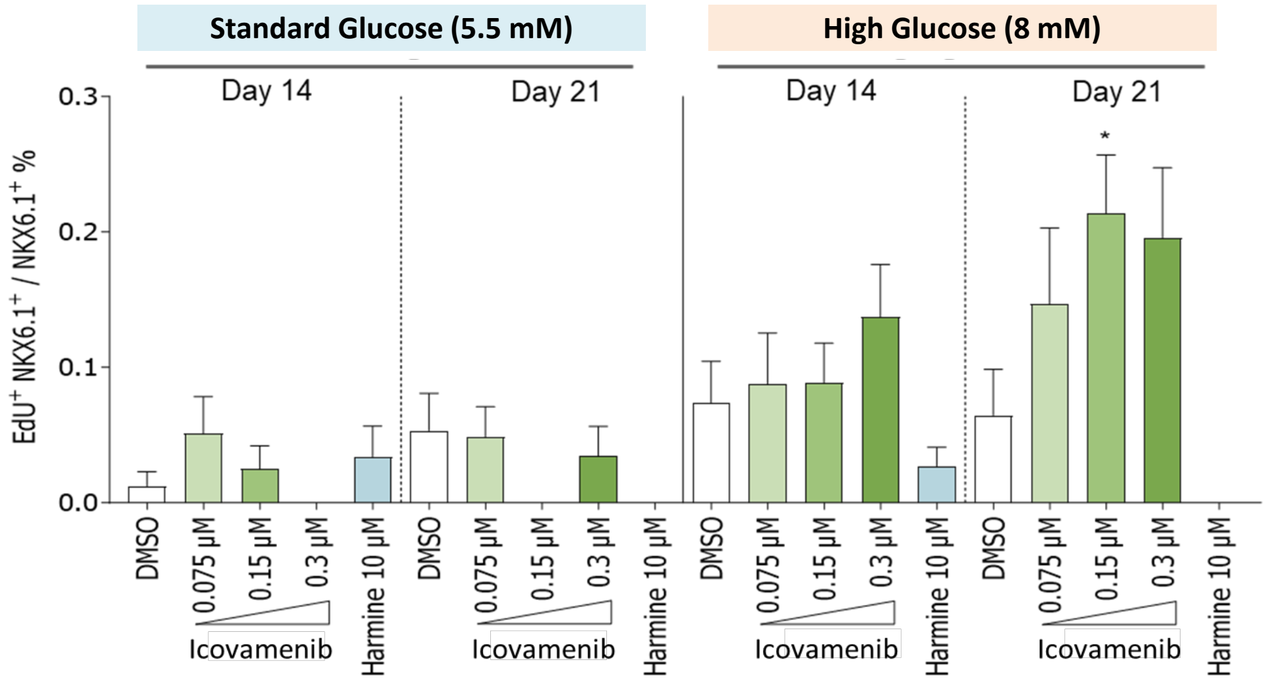


Donor 1: 49-yo male, 167 lbs, BMI 24.5, HbA1C 5.0%

Donor 2: 66-yo male, 248 lbs, BMI 33.7, HbA1C 5.9%

*normalized to vinculin/loading control

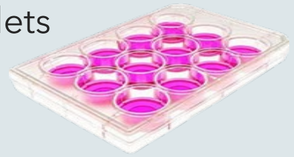
ICOVAMENIB CONDITIONALLY PROMOTED BETA CELL PROLIFERATION ONLY UNDER HYPERGLYCEMIC CONDITIONS

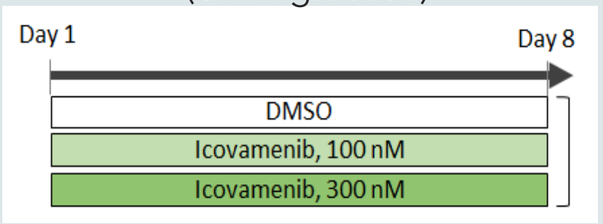


Icovamenib enhanced GLP-1 receptor & insulin expression in combination with semaglutide

Culture 7 days under glucotox conditions (8mM glucose)

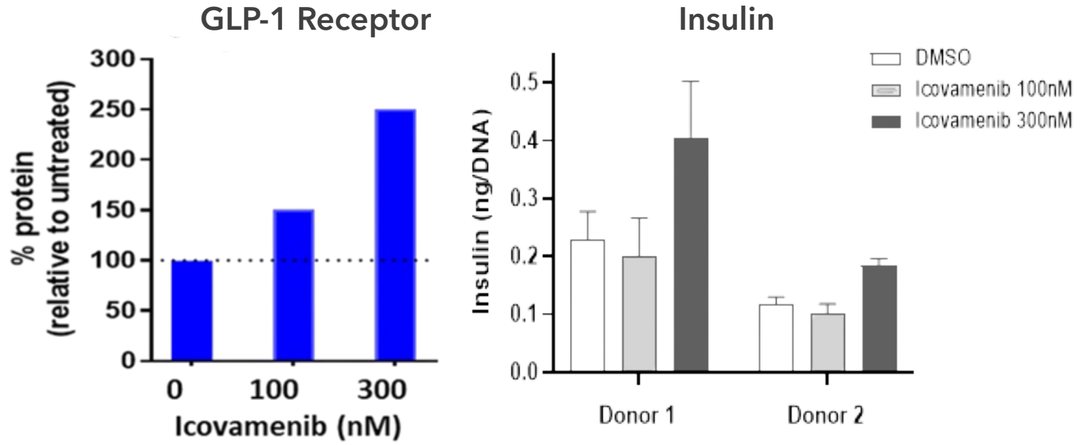
Cadaver derived human islets



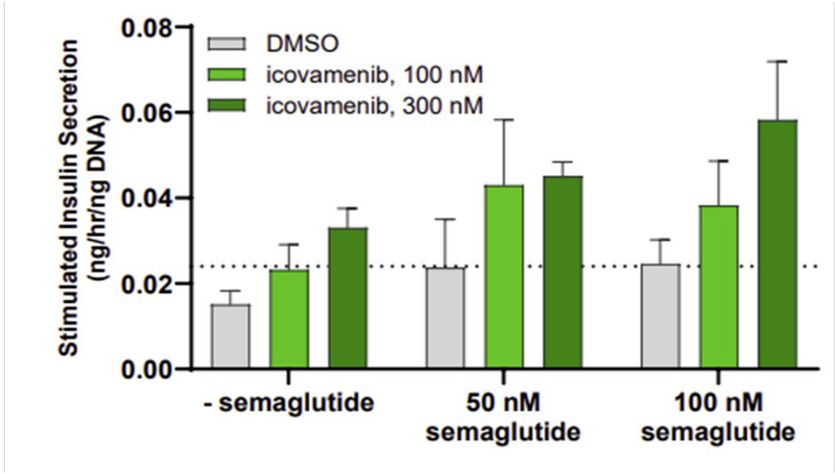


- Gene expression & Protein analysis
- Glucose Stimulated Insulin Secretion -/+ Semaglutide (200nM)

ICOVAMENIB INCREASED GLP-1 RECEPTOR AND INSULIN EXPRESSION

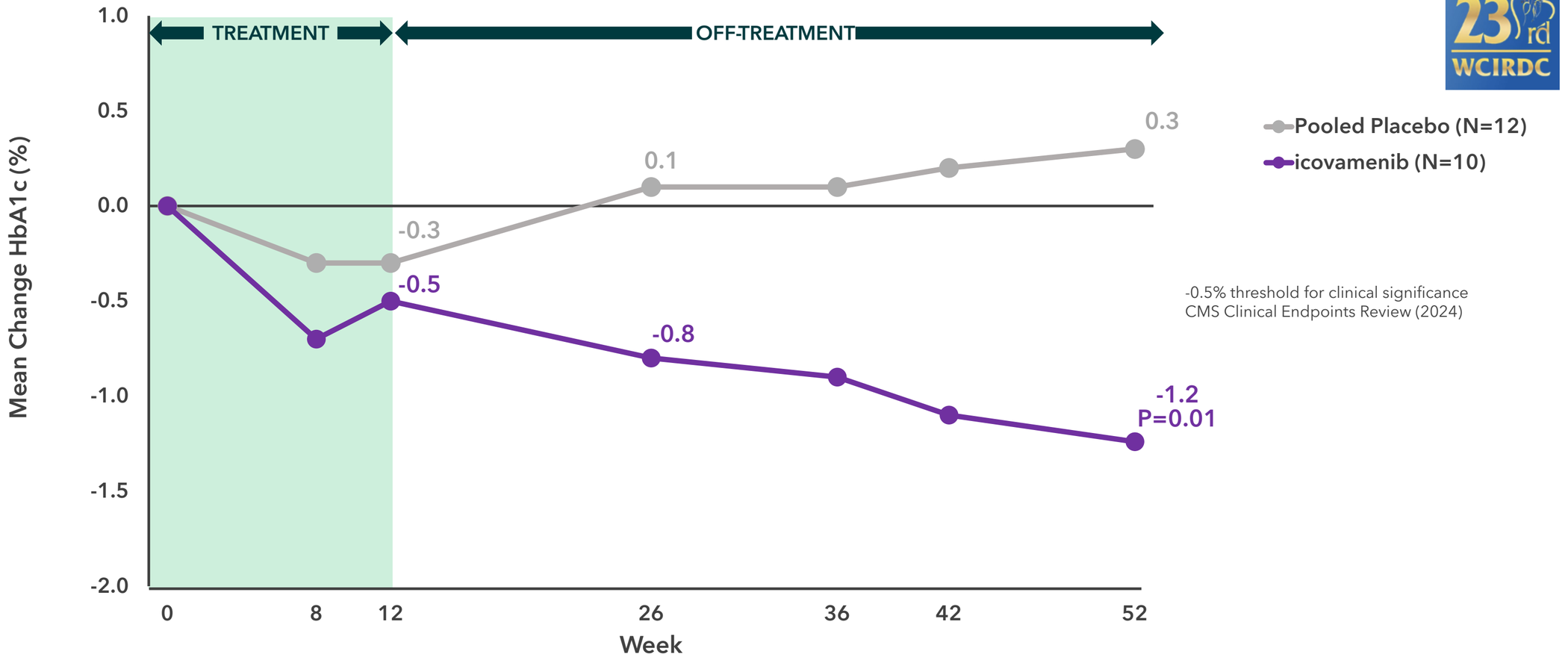


ICOVAMENIB IN COMBINATION WITH SEMAGLUTIDE INCREASED GLUCOSE-STIMULATED INSULIN SECRETION



ICOVAMENIB | COVALENT-111

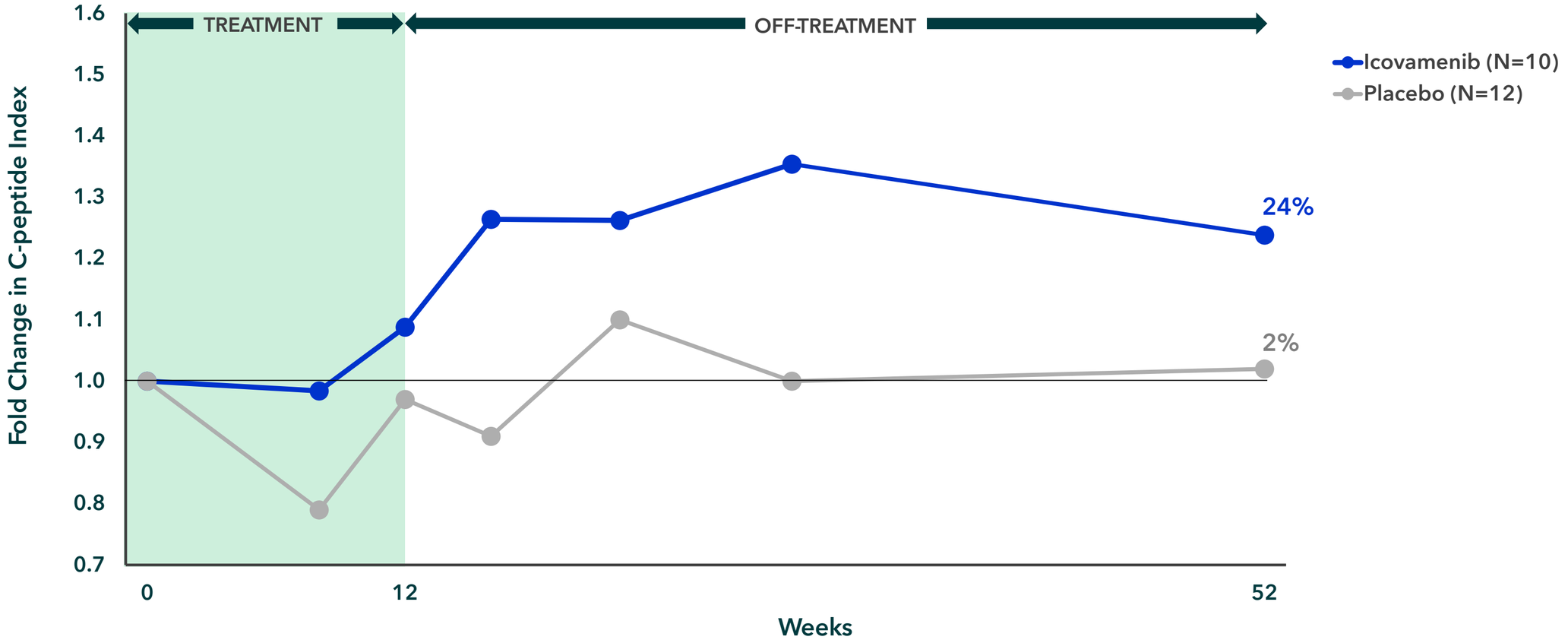
12 weeks of dosing (arms B & C) delivered lasting benefit through 52 weeks for severe insulin-deficient diabetes patients



Arm A was excluded from this analysis because it included only 8 weeks of dosing which the company is not planning to pursue.

ICOVAMENIB

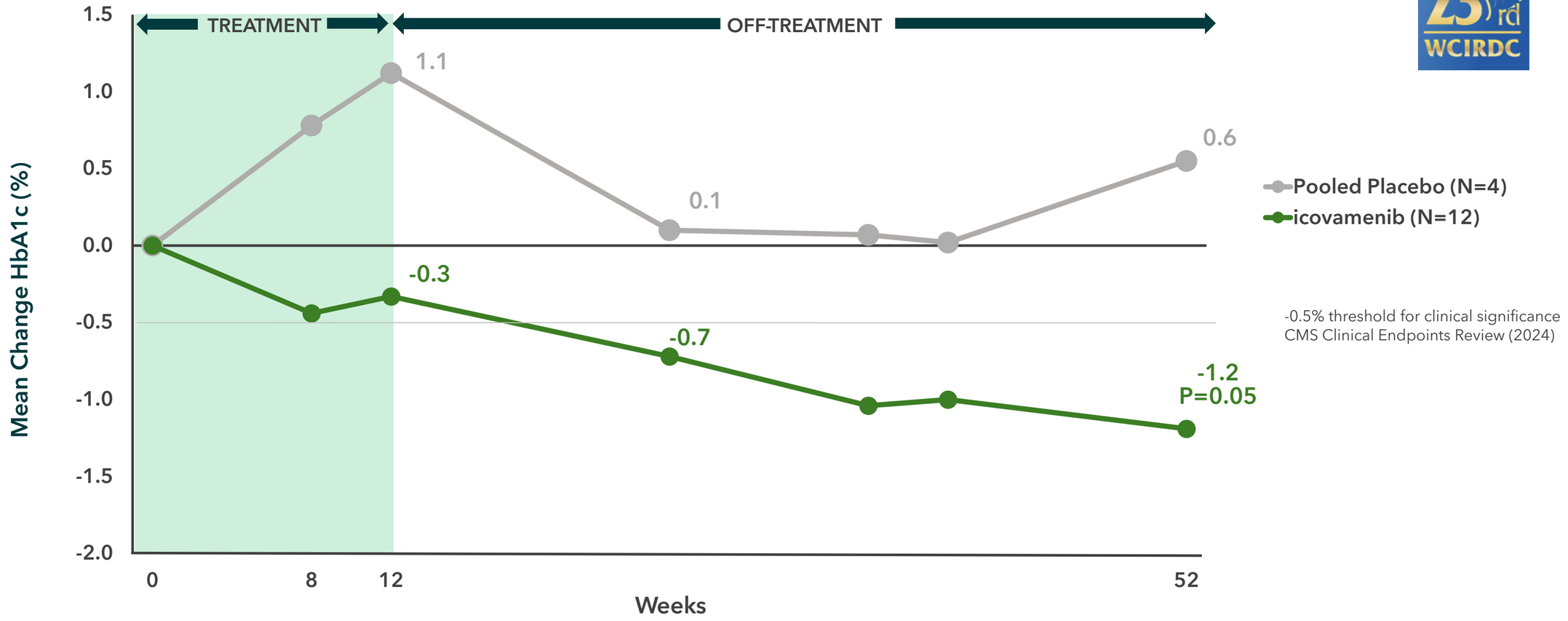
Icovamenib increased insulin secretion as measured by C-peptide index in severe insulin-deficient patients (arms B & C)



Data censored at onset of rescue medication, defined as any modification in antihyperglycemic therapy

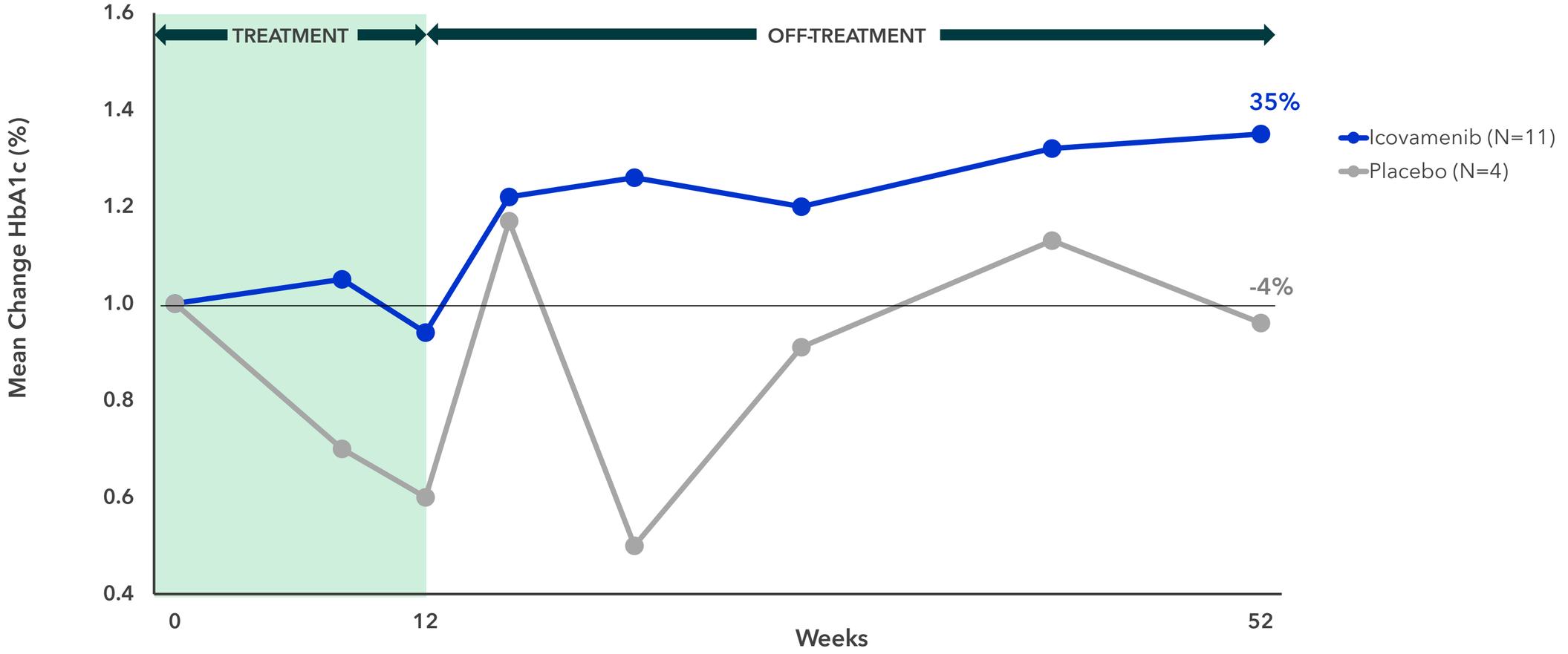
ICOVAMENIB | COVALENT-111

Patients on a GLP-1 based therapy at enrollment showed durable & clinically meaningful response in reduction of HbA1c



ICOVAMENIB

Icovamenib increased insulin secretion as measured by C-peptide index in GLP-1 RA treated patients



Data censored at onset of rescue medication, defined as any modification in antihyperglycemic therapy

ICOVAMENIB | COVALENT-111

Favorable 52-week safety profile

Parameter	Arm A icovamenib (N=67)	Arm B icovamenib (N=67)	Arm C icovamenib (N=67)	Combined Arms icovamenib (N=201)	Combined Arms placebo (N=66)
Patients with ≥1 TEAE, N (%)	19 (28)	22 (33)	14 (21)	55 (27)	18 (27)
Treatment-Related SAEs, N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
SAEs*, N (%)	1 (1)	0 (0)	1 (1)	2 (1)	1 (1)
Treatment Discontinuation due to TEAE, N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Study Discontinuation due to TEAE, N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
ALT increase, N (%)	3 (4)	0	2 (3)	5 (3)	0
AST increase, N (%)	3 (4)	0	1 (1)	4 (2)	0
Resolution of ALT/AST w/o treatment interruption (%)	100	100	100	100	N/A
Deaths, N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)

Data are n (%) TEAE = Treatment Emergent Adverse event. SAE = Serious Adverse Event. Data are n (%) of TEAE with ≥5% frequency in any arm. ALT (alanine aminotransferase) or AST (aspartate aminotransferase) increase irrespective of incidence %.

*Arm A had an SAE of atrial fibrillation, unrelated to study treatment and occurred during the treatment period.

*Arm C had an SAE of COVID-19. Unrelated to study treatment and occurred during the treatment period.

*Placebo Arm had an SAE of nephrolithiasis. Unrelated to study treatment and occurred during the treatment period.

ALT increase: In the icovamenib arms, 4 of the 5 events were Grade 1 and 1 event was Grade 2.

AST increase: In the icovamenib arms, all 4 events were Grade 1.

All incidences of ALT and AST elevations resolved without interruption.

Note:
In AML studies icovamenib demonstrated a well-tolerated safety profile across all dose levels, with up to 500 mg QD / 325 mg BID, and dose durations extending over 1 year

Short treatment with icovamenib delivered HbA1c reductions comparable to chronic injectable & oral standards of care

Comparing icovamenib to currently approved type 2 diabetes agents with chronic dosing

THERAPY	DOSING REGIMEN	ADMINISTRATION ROUTE	OBSERVATION PERIOD	MEAN HbA1c REDUCTION (PLACEBO ADJ. %)
Ozempic (GLP-1 Agonist)	Chronic dosing	Injectable	Week 30	-1.2 (0.5mg) -1.4 (1mg)
Mounjaro (GLP-1/GIP Agonist)	Chronic dosing	Injectable	Week 40	-1.8 (5mg) -1.7 (15mg)
Jardiance (SGLT2 Inhibitor)	Chronic dosing	Oral	Week 24	-0.7 (10mg) -0.9 (25mg)
Januvia (DPP4 Inhibitor)	Chronic dosing	Oral	Week 24	-0.8 (100mg)
Icovamenib (menin inhibitor)	**12 weeks**	**Oral**	**Week 52**	**-1.5% to -1.8% (100 mg)**

Ozempic FDA Label; Mounjaro FDA Label; Jardiance FDA Label; Januvia FDA Label

Disclaimer: The data presented above are based on cross-study comparisons and are not based on any head-to-head clinical trials. Cross-study comparisons are inherently limited and may suggest misleading similarities and differences. The values shown in the cross-study comparisons are directional and may not be directly comparable.

Optimal dose, dose-duration, target population identified for phase IIb program

ICOVAMENIB

Phase IIa key derisking-insights:

- ✓ Optimal dose selected, 100 mg
- ✓ Food Effect Study confirmed optimal PK exposure of icovamenib within 30 minutes after a meal
- ✓ 12-week treatment observed to drive durable and lasting effects, no chronic treatment required
- ✓ Strong clinical activity in insulin-deficient and GLP-1 inadequate responder populations
- ✓ Treatment-emergent AEs comparable to placebo

Direct application in Phase II/IIb's

COVALENT-211

Phase IIb trial in type 2 insulin deficient diabetes patients failing standard of care

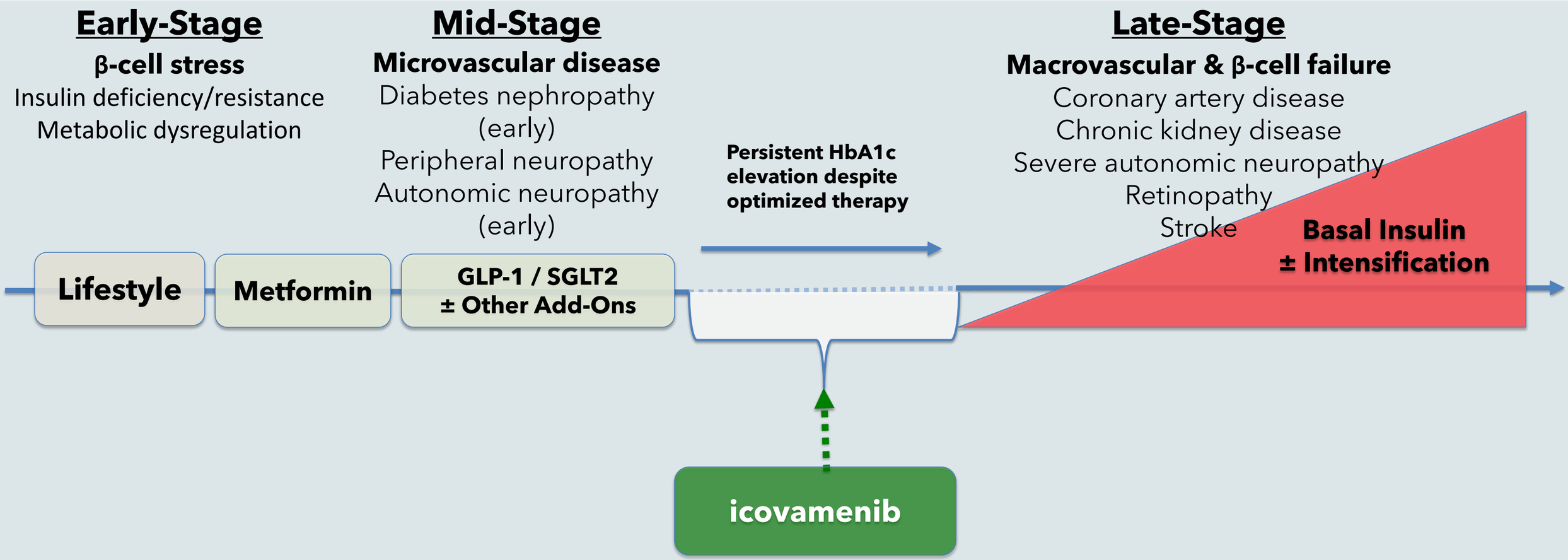
- Adult participants with Type 2 Diabetes who were treated with 1-3 antidiabetic medications
- HbA1c 7.5%-10.5% and BMI ≤ 32 kg/m2
- Background therapy maintained unless rescue required

COVALENT-212

Phase II trial in type 2 diabetes patients failing standard of care while on a GLP-1 RA

- Adult participants with T2D who are not achieving glycemic targets despite GLP-1-based therapy
- HbA1c ≥7.5% and ≤9.5% and BMI 25 to 40 kg/m2
- Background therapy maintained unless rescue required

Icovamenib aims to delay need for insulin therapy
and reduce complications and disease burden



In the U.S., more than half of patients with diabetes remain above HbA1c targets ≥7%[1]
Depending on the GLP-1 RA agent, 15-45% do not achieve HbA1c < 7% in clinical trials[2]

1.NHANES analyses of glycemic control among U.S. adults with diabetes (JAMA; Diabetes Care); 2.SUSTAIN, AWARD, and SURPASS clinical trial programs for GLP-1 receptor agonists

BMF-650

An Investigational Next-Generation Oral GLP-1 Receptor Agonist for Obesity

biomea FUSION™

Obesity remains inadequately controlled despite GLP-1 therapies, with millions discontinuing or failing treatment

Obesity is a chronic, progressive disease associated with cardiometabolic complications and increased mortality

42%

Of U.S. adults have obesity[1]

Obesity is a chronic disease characterized by excess adiposity and metabolic dysfunction. It is strongly associated with type 2 diabetes, cardiovascular disease, fatty liver disease, and certain cancers.

50-70%

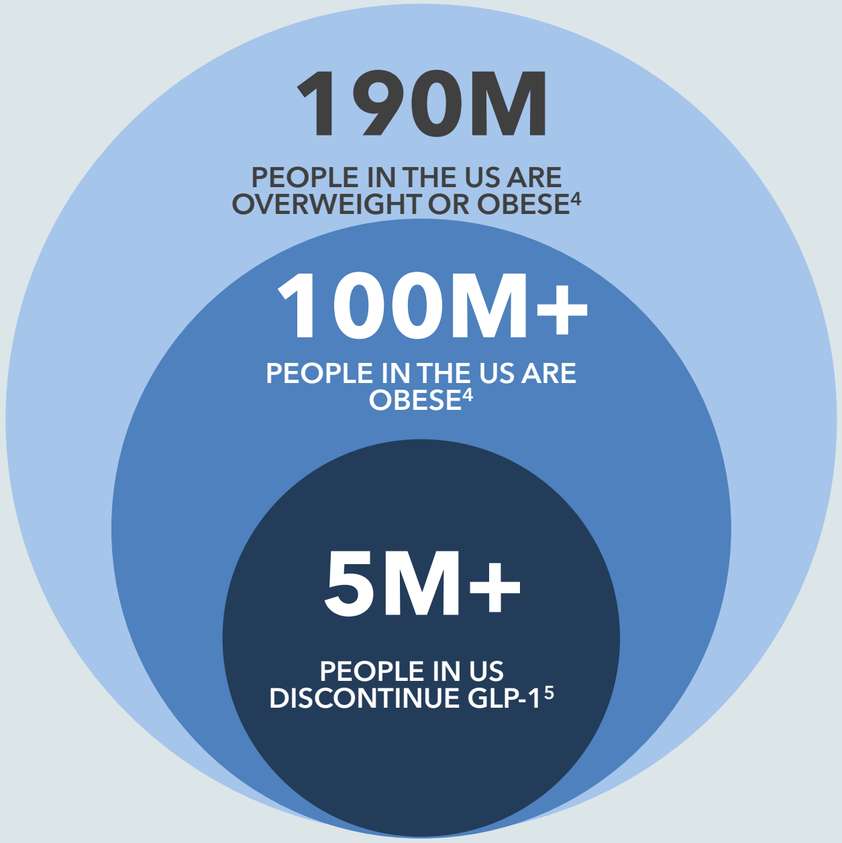
Of patients discontinue GLP-1 therapy within 12 months[2]

Real-world data show high discontinuation rates due to GI side effects, cost, access barriers, and tolerability challenges. Weight regain is common after discontinuation.

>60%

of adults with obesity have at least one obesity-related comorbidity[3]

Despite lifestyle interventions and approved pharmacotherapies, many patients discontinue treatment or fail to achieve sustained weight loss. Long-term disease modification remains an unmet need.



1. CDC Adult Obesity Facts, 2023
2. Real-world GLP-1 discontinuation analyses (claims database studies 2023–2024)
3. STEP and SURMOUNT program responder analyses
4. CDC National Health and Nutrition Examination Survey
5. IQVIA prescription data

BMF-650

Developed to deliver strong efficacy with improved oral tolerability

An Investigational Next-Generation Oral GLP-1 Receptor Agonist

Proposed differentiated properties of BMF-650

Improved PK Profile

Greater oral exposure with lower variability observed in preclinical studies

Generally Favorable Safety Profile

Better tolerability associated with higher plasma protein binding in preclinical models

Patient Friendly Design

Oral delivery with the potential for simplified dose escalation

Greater therapeutic window matters

- Only 3 of 10 patients remain on GLP-1 therapy at one year due to tolerability, GI effects and complexity of use.[1]
- An oral agent with improved tolerability could potentially expand the long-term use.

Intellectual Property

- U.S. patent allowance received December 2025 covering BMF-650 composition.
- U.S. and PCT applications published and proceeding through examination.

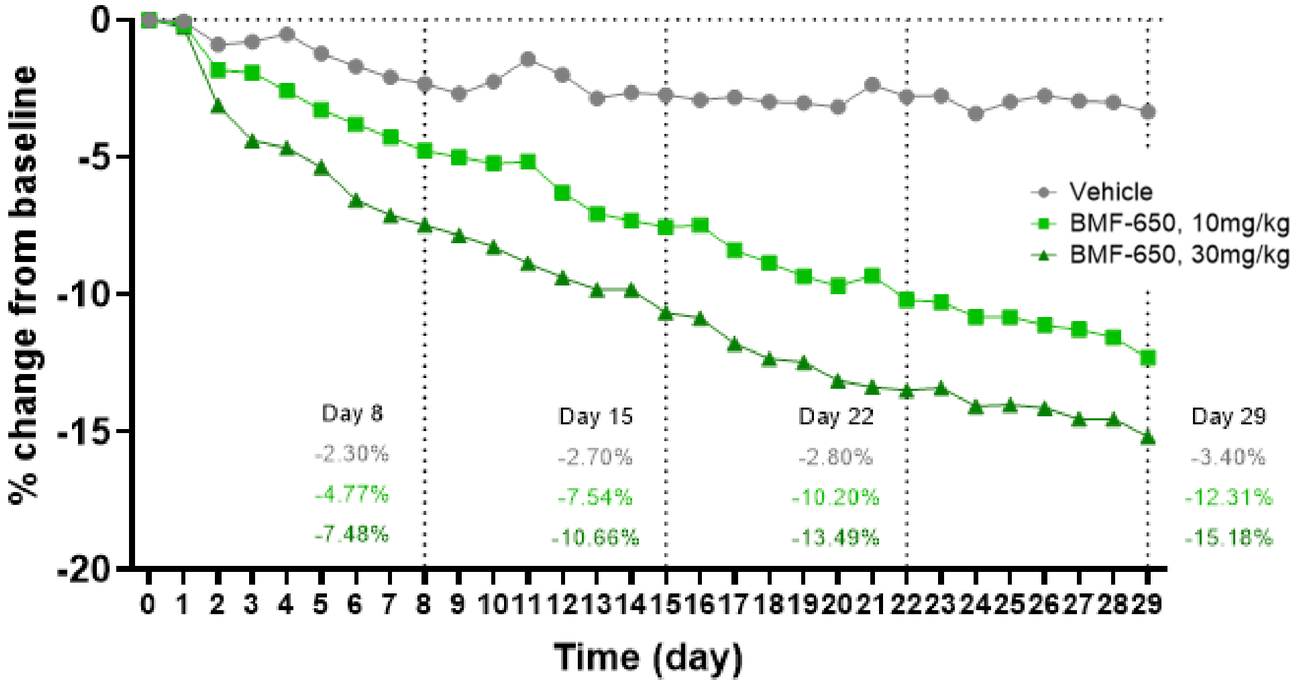
1. Khan, et al. JAMA 2024 doi:10.1001/jama.2024.22284.

BMF-650 | PRECLINICAL

BMF-650 demonstrated robust, dose dependent weight loss in obese monkeys

Weight loss in cross-study comparison with CT-996 (Roche/Carmot), while not head-to-head appeared favorable

BMF-650 up to ~15% body weight reduction after 28-days



CT-996 body weight change

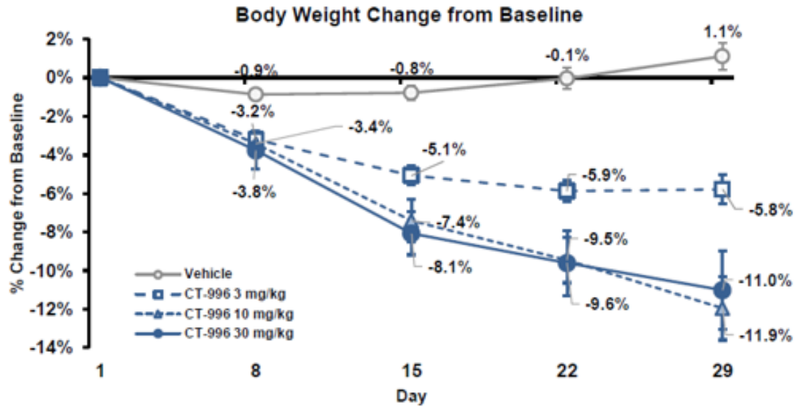


Figure 6. Effects of CT-996 on body weight in obese cynomolgus monkeys following once-daily oral administration. Weekly body weight percent change is represented as mean (± SE) from baseline. N = 6/group.

Literature data; Carmot Therapeutics (now part of the Roche group), ADA 2024.

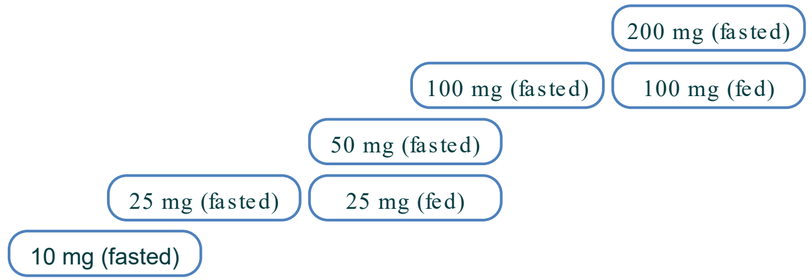
Disclaimer: No head-to-head studies of BMF-650 and CT-996 have been conducted. Comparing results from different preclinical studies may be unreliable due to differences in study designs, study endpoints, and other parameters.

BMF-650 | GLP-131

A randomized, double-blind, placebo-controlled, FIH study of an oral non-peptide GLP-1 receptor agonist

Part 1 is a single ascending dose (SAD) study and Part 2 is a multiple ascending dose (MAD) study.

	Single Ascending Dose (SAD)	Multiple Ascending Dose (MAD)
Objectives	Safety and tolerability, PK, and food effect	Safety and tolerability, and efficacy (weight-loss)
Eligibility	Healthy overweight or obese patients (BMI 25.0–40.0 kg/m²)	Healthy overweight or obese patients (BMI 30.0–45.0 kg/m²)
Design	N=40 5 cohorts x 	N=40 4 cohorts x 



BMF-650 active drug

placebo

COHORT	7 DAYS → 7 DAYS → 7 DAYS → 21 DAYS
4	75 mg → 200 mg → 400 mg → 400 mg
3	75 mg → 150 mg → 300 mg → 300 mg
2	50 mg → 100 mg → 200 mg → 200 mg
1	10 mg → 25 mg → 50 mg → 100 mg

Body weight at Baseline versus Day 28 and Day 42 on treatment

Biomea pipeline

Biomea Fusion retains full worldwide rights across all programs and is currently funded through major catalysts into 1Q 2027

PROGRAM	INDICATION	PHASE I	PHASE II	PHASE III	UPCOMING MILESTONES
ICOVAMENIB Potential first-in-class oral menin inhibitor	**Type 1 diabetes** Patients - All comers *(>2M US Patients)* [1]	**COVALENT-112** (study completed)			52-week follow-up data of those patients who completed dosing expected **2Q 2026**
	Type 2 diabetes Patients with insulin deficiency *(~7M US Patients)* [2]	**COVALENT-211** (study initiated)			Phase II 26-week data (primary endpoint) anticipated **4Q 2026**
	Type 2 diabetes Patients not controlled on GLP-1 based therapies *(15-45% US Patients on GLP-1RA)* [3,4]	**COVALENT-212** (study initiated)			Phase II 26-week data (primary endpoint) anticipated **4Q 2026**
BMF-650 Potential best-in-class oral GLP-1 RA	**Obesity** *(>100M US Patients)* [5]	**GLP-131** (study enrolling)			Phase I 28-day weight reduction data expected **2Q 2026**

1.National Diabetes Statistics Report, Accessed January 28, 2026
2.International Diabetes Federation. IDF Diabetes Atlas www.diabetesatlas.org (Based on company calculations)
3.NHANES analyses of glycemic control among U.S. adults with diabetes (JAMA; Diabetes Care);
4..SUSTAIN, AWARD, and SURPASS clinical trial programs for GLP-1 receptor agonists
5.National Center for Health Statistics August 2023. Accessed January 28, 2026

THANK YOU

(NASDAQ: BMEA)

For questions or inquiries, please reach out to Meichiel Weiss at ir@biomeafusion.com

www.biomeafusion.com

biomea FUSION™

KOLs highlight icovamenib's potential to impact and redefine diabetes treatment

"Icovamenib's recent data has shown an impressive restoration of beta cell function as demonstrated by significant elevations in C-peptide even after the treatment period ended.

This data validates the mechanism of action of this menin inhibitor as a disease modifying agent and helps address the poor adherence and persistence commonly seen in type 2 diabetes."

Steve Edelman, M.D.

ENDOCRINOLOGIST, PROFESSOR OF MEDICINE UCSD / VA SAN DIEGO

"The icovamenib data looks exciting. The data presented today help to confirm icovamenib's mechanism of action. We have not previously seen data like this with any antihyperglycemic agent.

As more trials are conducted, I believe that inhibition of menin may lead to benefits across all subtypes of diabetes. I applaud Biomea for developing a potential new treatment option that may be disease modifying for patients with diabetes."

Ralph DeFronzo, M.D.

ENDOCRINOLOGIST, PROFESSOR OF MEDICINE UTHSCSA

"Great foray into precision medicine. We need to be addressing patients in a much more individualized manner. By addressing insulin-deficient diabetes patients with icovamenib, we have seen post treatment that the beta cell pool is being restored and producing a higher level of insulin, as measured by C-peptide.

This indicates a fundamental and potentially lasting impact on the disease and validates the mechanism of action of menin inhibition."

Melanie Davies, M.D.

DIABETOLOGIST, PROFESSOR OF DIABETES MEDICINE AT THE UNIVERSITY OF LEICESTER

KOLs highlight icovamenib's potential to impact and redefine diabetes treatment

"We do not have an agent today that addresses one of the root cause of diabetes - beta cell dysfunction - icovamenib would be the first.

Patients are achieving lasting benefits without continuous chronic dosing, suggesting that icovamenib may be disease modifying. I am very impressed."

Alice Cheng, M.D.

ENDOCRINOLOGIST, ASSOCIATE PROFESSOR OF MEDICINE
UNIVERSITY OF TORONTO

"The icovamenib data are quite interesting because of the continued effects despite having stopped it.

Usually, one would expect to see the HbA1c levels climb towards baseline when the medication is stopped, but with icovamenib, the HbA1c levels decreased, which is quite intriguing and unprecedented."

Julio Rosenstock, M.D.

DIRECTOR VELOCITY CLINICAL RESEARCH AT MEDICAL CITY DALLAS AND CLINICAL PROFESSOR OF MEDICINE, UNIV. OF TEXAS SOUTHWESTERN MEDICAL CENTER

"Icovamenib is a very interesting molecule that acts quite differently than anything I have seen before. We are observing glucose controlled and beta cell-specific proliferation and an increase in stimulated C-peptide secretion leading to patient benefits that continued after the icovamenib dosage ended.

I am very excited to further explore the many opportunities that the covalent inhibition of menin will provide to patients."

Rohit Kulkarni, M.D., Ph.D.

PROFESSOR OF MEDICINE AT HARVARD MEDICAL SCHOOL