Biomea Fusion Corporate Presentation

Q2 2026

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Legal disclaimer & forward-looking statements

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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.

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

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.

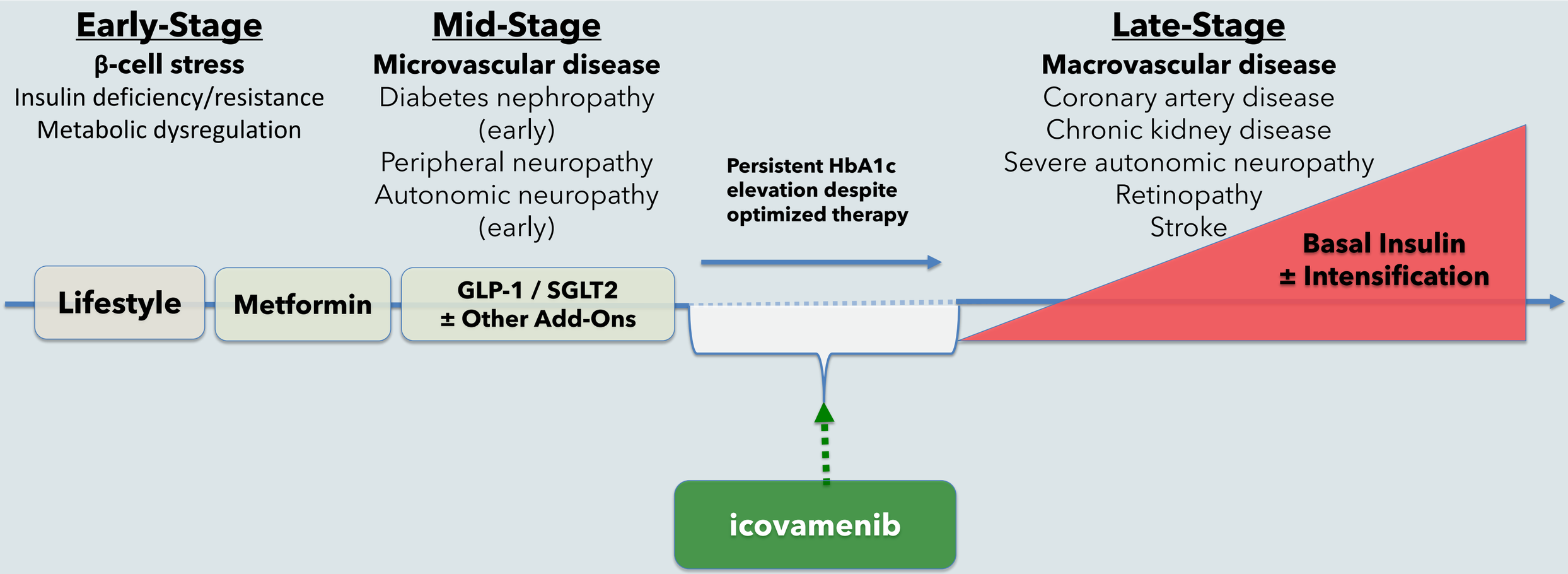
60+

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

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

ICOVAMENIB

Potential first-in-class menin inhibitor aimed to restore functional beta-cells

Aims to serve a significant unmet need for millions of diabetes patients failing on standard of care

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Icovamenib is developed to:

- \+ Employ and enhance body's natural response to hyperglycemia as evidenced in pregnancy
- \+ Conditionally drive beta-cell proliferation and activity only in presence of high glucose levels
- \+ Enhance GLP-1 efficacy by upregulating GLP-1 receptors on the beta-cell surface
- \+ Target beta-cell restoration and potentially delay or prevent onset of end-stage disease

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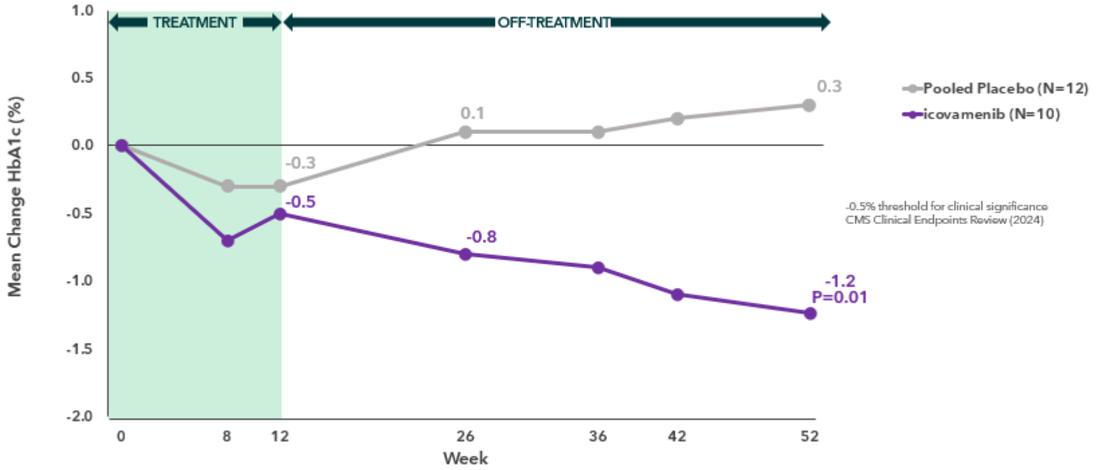
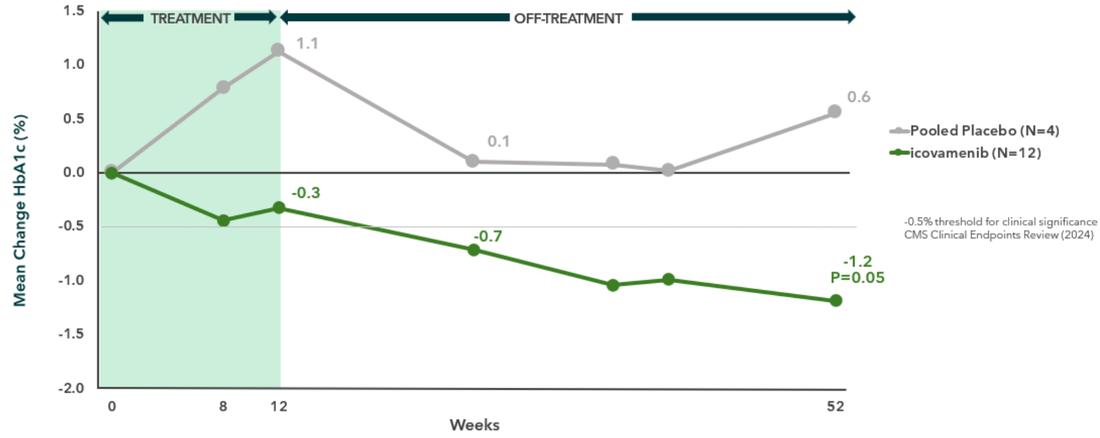
Severe insulin-deficient diabetes patients after 12-weeks of dosing



GLP-1 RA uncontrolled diabetes patients after 12 weeks of dosing



Post-hoc analysis of patients on GLP-1 based therapy not achieving stable HbA1c <7% at enrollment (9 months after last dose)

Early signs of clinical activity with 12 weeks of dosing in diabetes patients failing standard of care therapies

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%

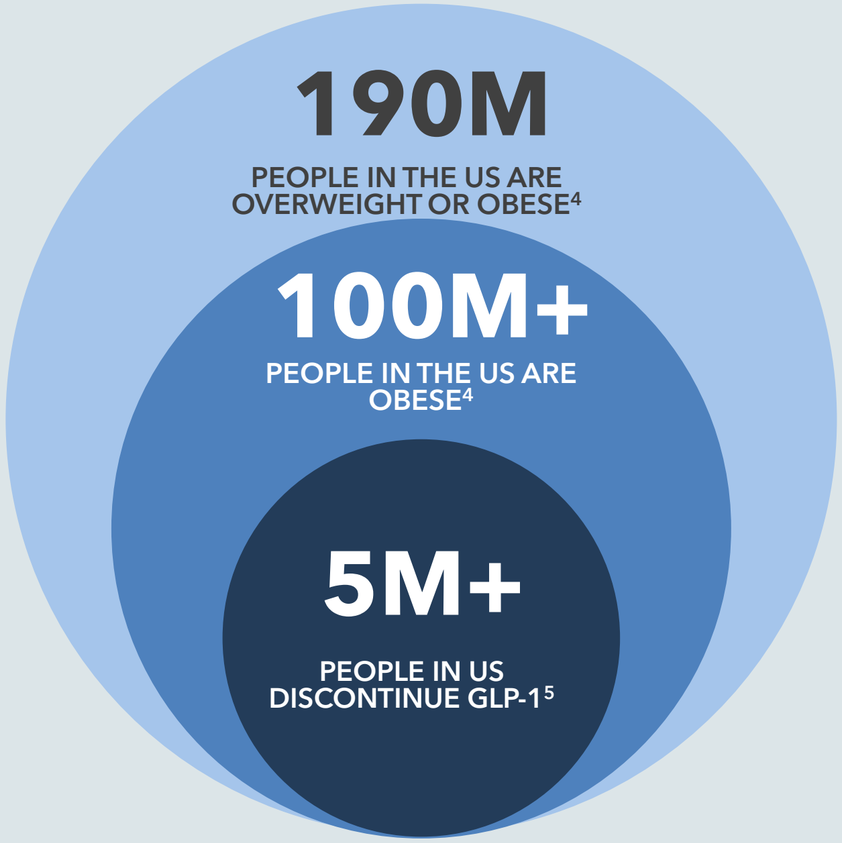
Of patients discontinue GLP-1 therapy within 12 months[2]

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

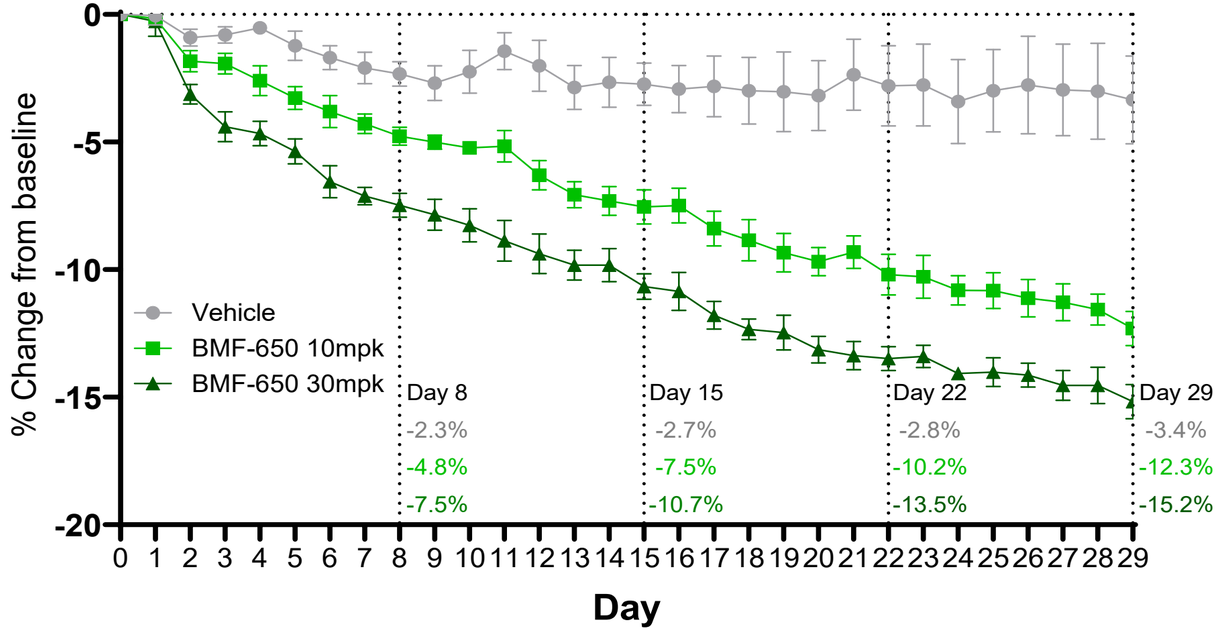
Oral GLP-1 RA developed for improved patient friendly tolerability

Aims to serve a significant unmet need with millions of obese Americans dropping off the available GLP-1 RAs agents within the first year[1]

BMF-650 is developed to:

- \+ Built on the orforglipron scaffold with key structural improvements
- \+ Greater oral exposure and bioavailability with lower variability observed in preclinical models
- \+ Higher plasma protein binding supporting better tolerability
- \+ Potential for simplified dose escalation schedule with generally well-tolerated safety profile

~15% Body Weight Reduction in 28-day Obese Monkey Study



1. Prime Therapeutics & Magellan Rx Management, 2023 real-world claims analysis.

ICOVAMENIB

Potential first-in-class menin inhibitor for diabetes

Preclinical results

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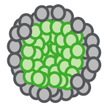
Icovamenib's mechanism of action

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

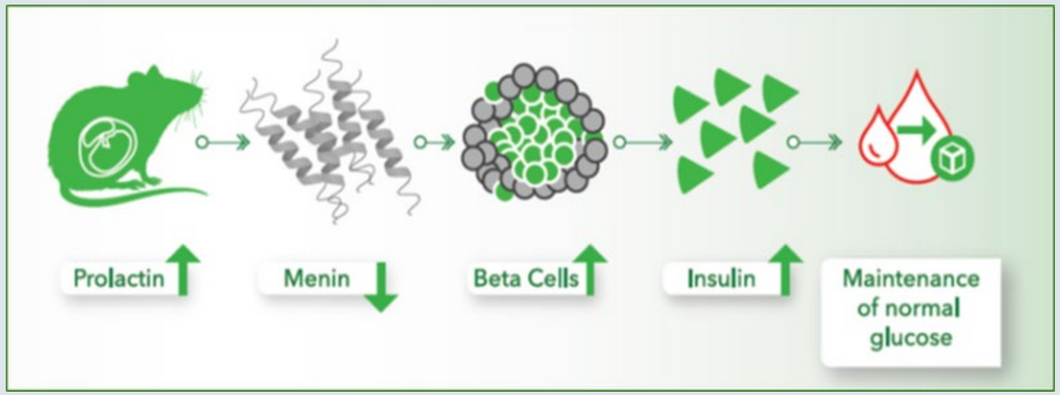
Menin is naturally inhibited during pregnancy and breastfeeding - allowing for beta cell regeneration & reduced diabetes risk

- 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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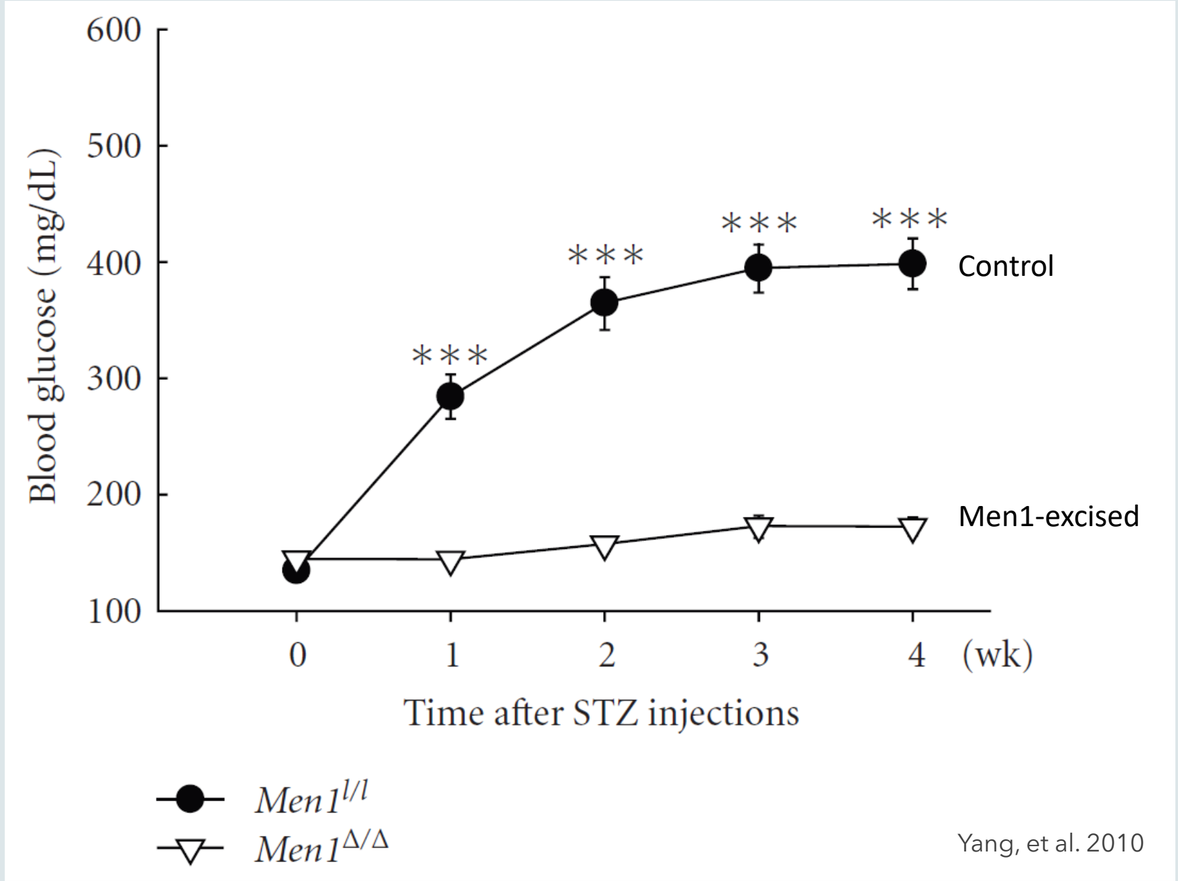
Potential for Menin Inhibition Demonstrated by Beta Cell Ablation Diabetes Model in MEN1-Excised Mice

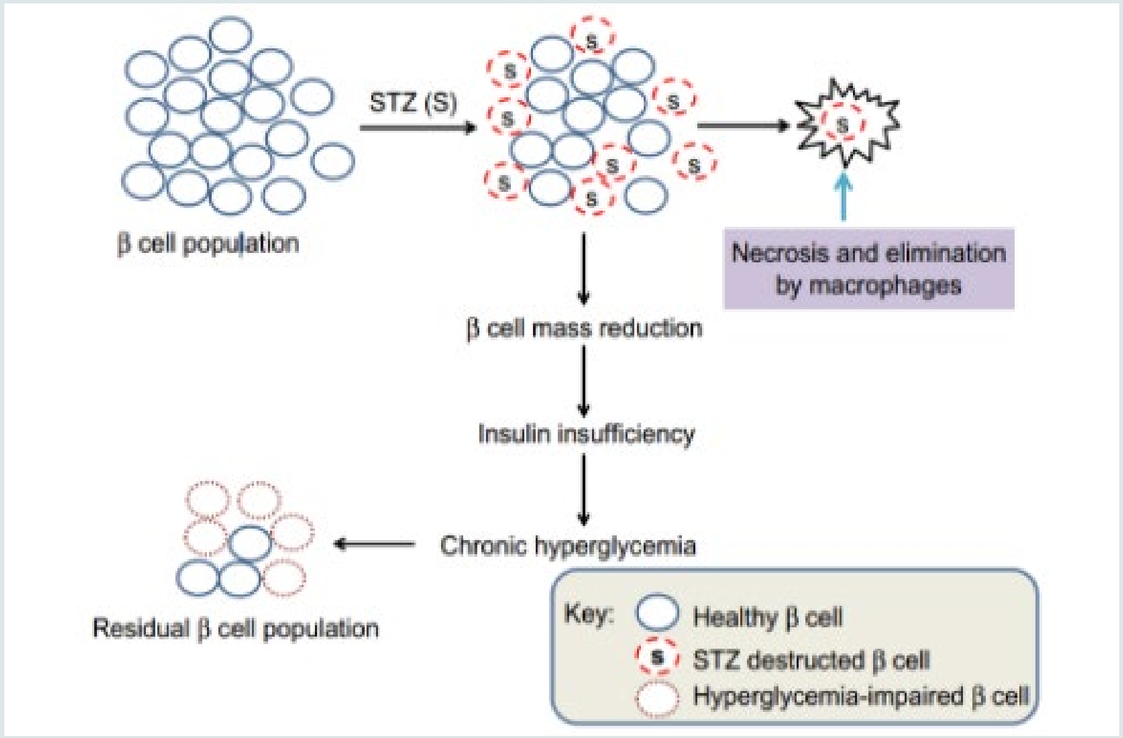
MEN1 Excision Prevents Development of STZ-induced Hyperglycemia

- Menin is a scaffold protein, encoded by the gene MEN1, that has been recognized for its role in Type 2 Diabetes Mellitus (T2DM) as a key regulator of beta-cell proliferation.
- Men1 knockout mice demonstrate increased beta-cell mass generation (Yang et al., 2010) and menin inhibition has previously been shown to improve glycemic control in high fat induced diabetic mice (Ma et al., 2021).
- Men1-excised mice do not develop hyperglycemia in a Streptozotocin-(STZ) induced rat model, which is a model for impaired beta-cell function and insulin production, demonstrating the role of menin in glycemic control.

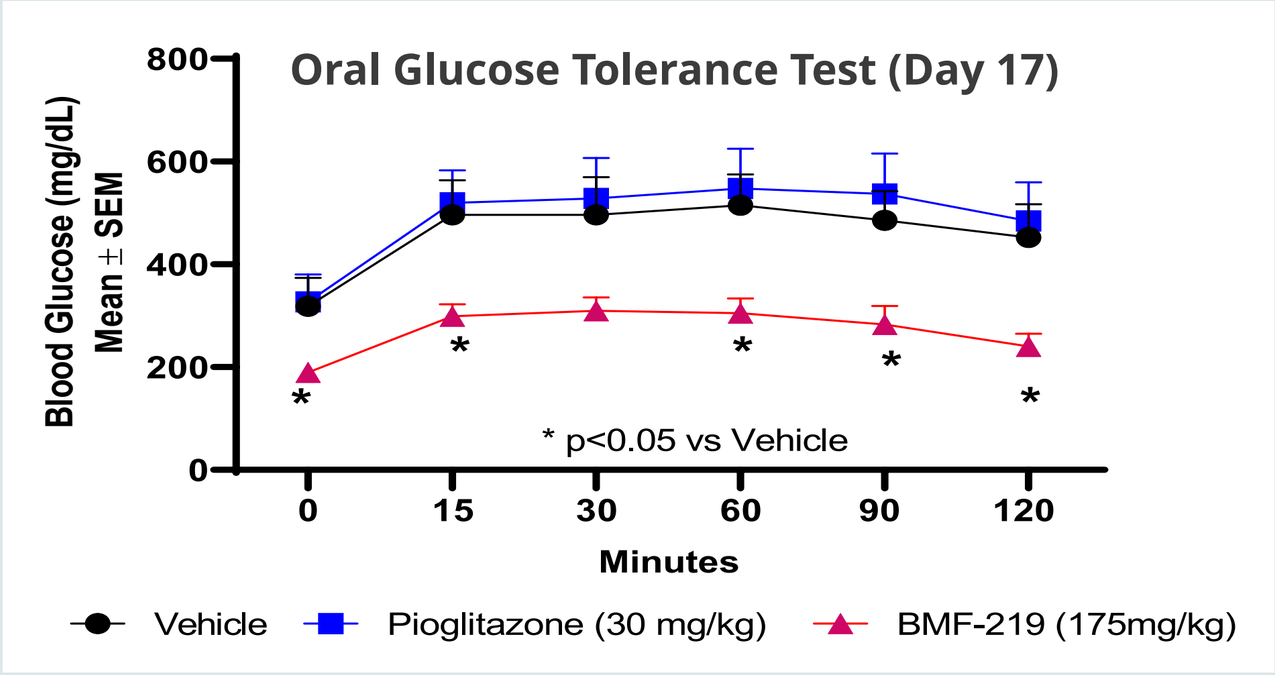


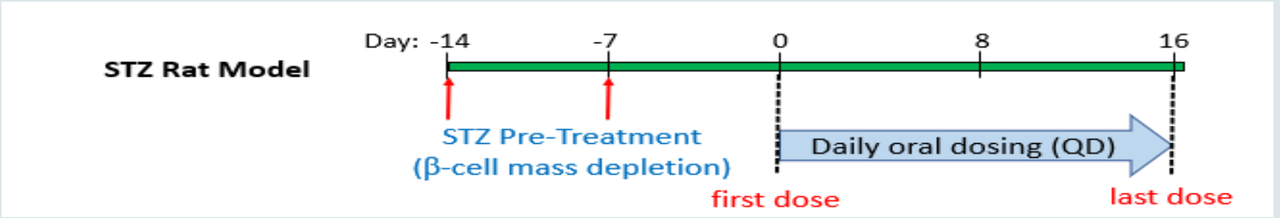
Men1-excised mice did not develop hyperglycemia in the STZ model, which was observed in the control group

BMF-219 (icovamenib) significantly reduces blood glucose levels in STZ rats where typically only insulin decreases blood glucose levels



STZ treatment typically results in ~50% Beta Cell Loss

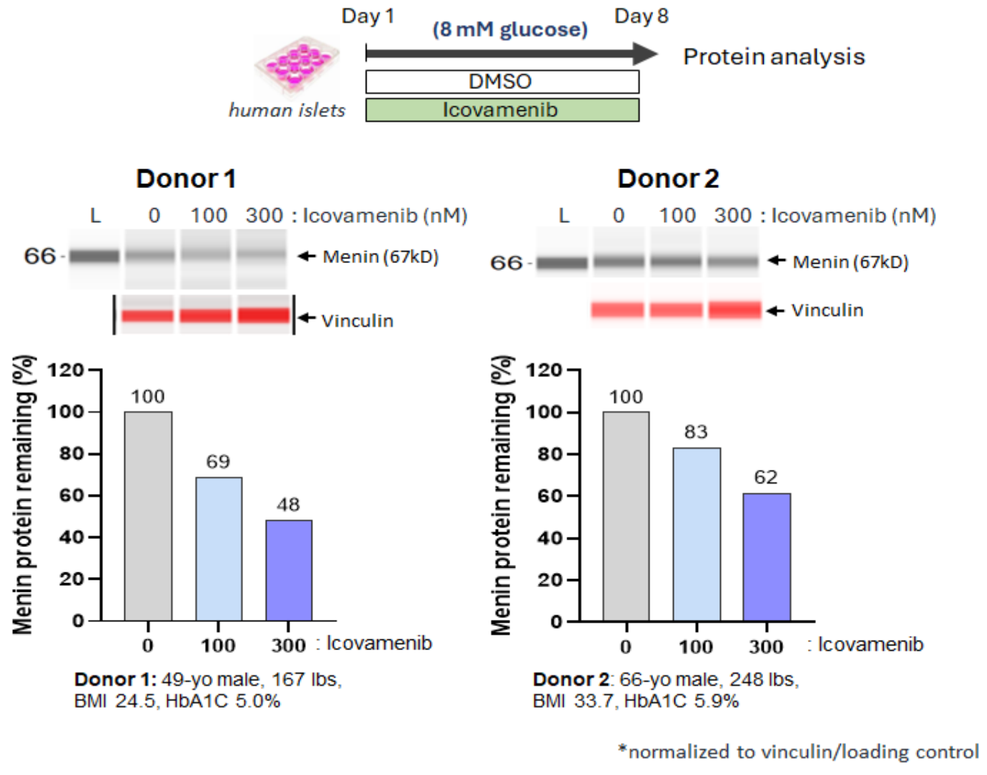




ICOVAMENIB | PRECLINICAL

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

MENIN LEVELS DOWNREGULATED

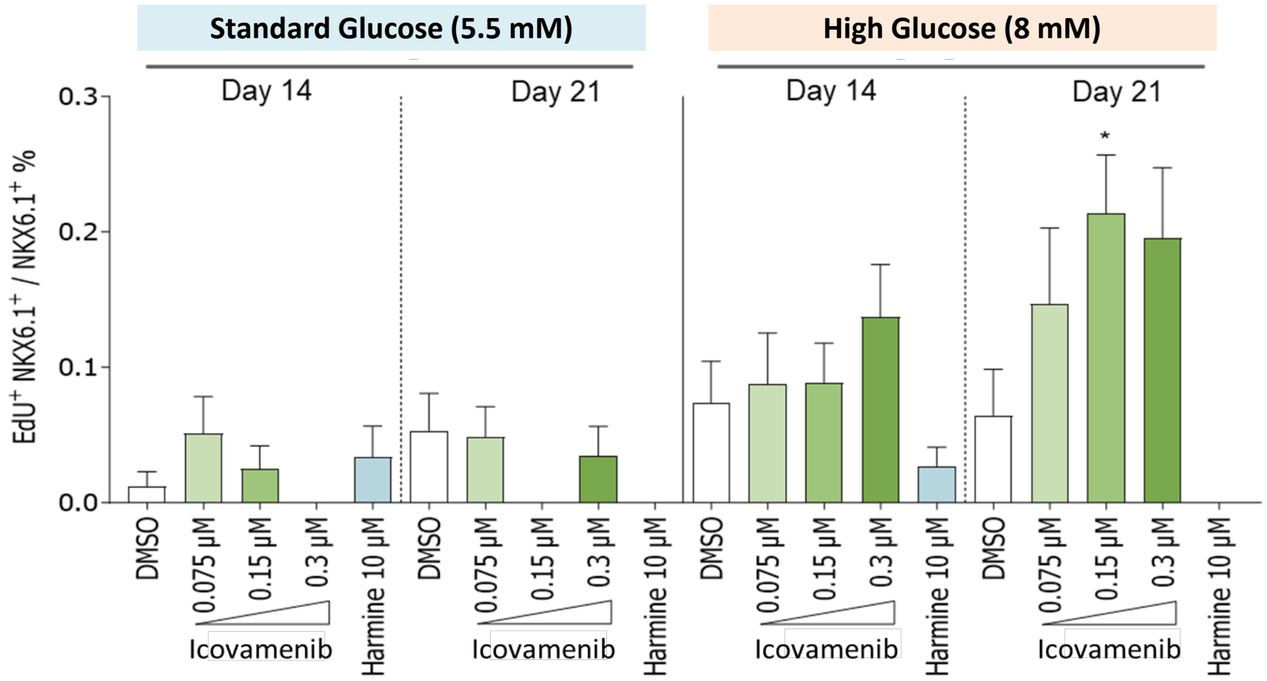


Donor 1: 49-yo male, 167 lbs, BMI 24.5, HbA1C 5.0%

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

*normalized to vinculin/loading control

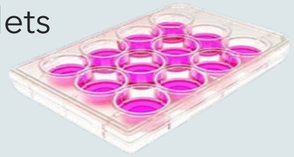
ICOVAMENIB CONDITIONALLY PROMOTED BETA CELL PROLIFERATION ONLY UNDER HYPERGLYCEMIC CONDITIONS

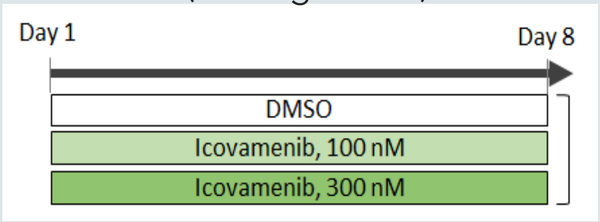


Icovamenib enhanced GLP-1 receptor & insulin expression in combination with semaglutide

Culture 7 days under glucotox conditions (8mM glucose)

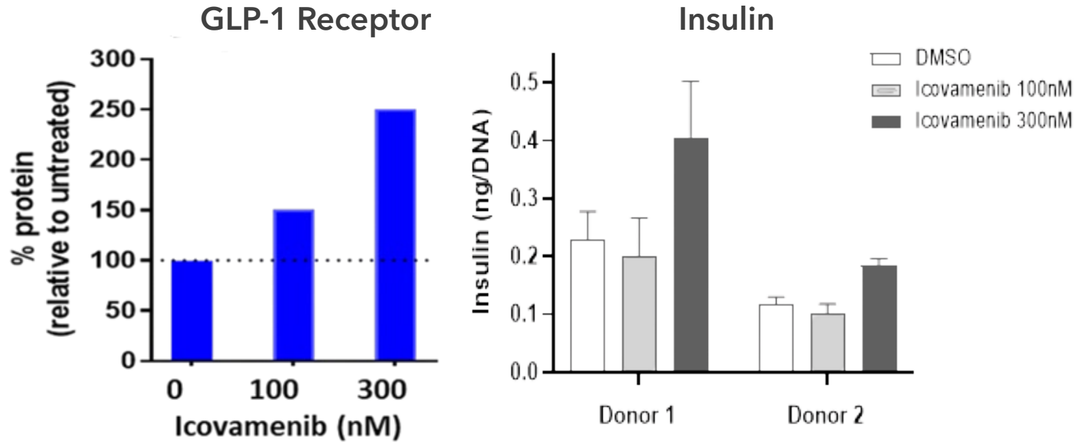
Cadaver derived human islets



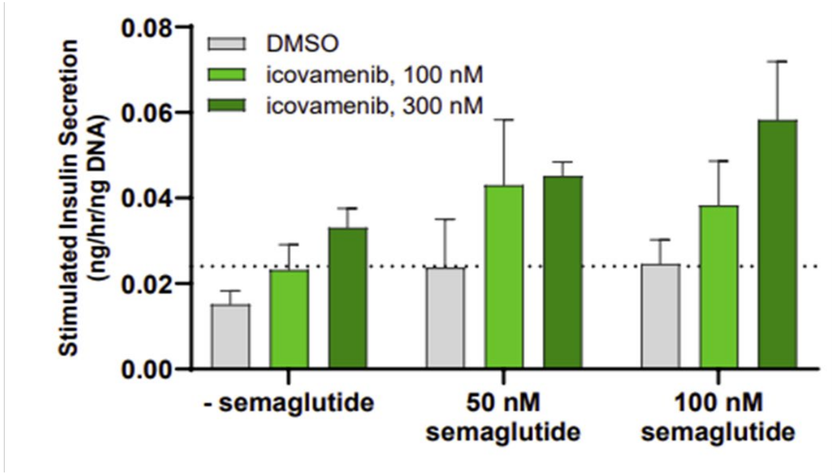


- Gene expression & Protein analysis
- Glucose Stimulated Insulin Secretion -/+ Semaglutide (200nM)

ICOVAMENIB INCREASED GLP-1 RECEPTOR AND INSULIN EXPRESSION



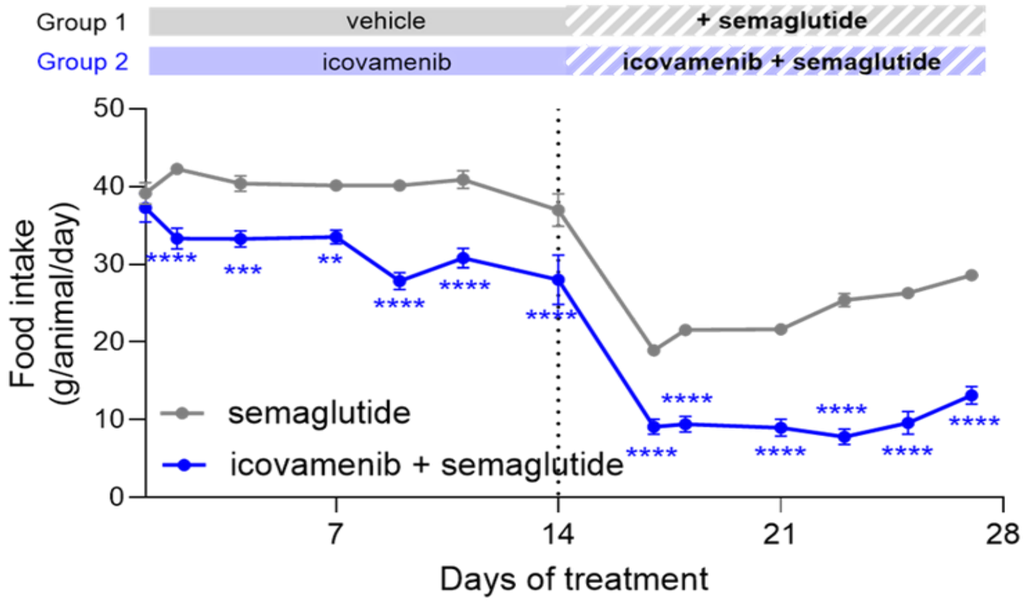
ICOVAMENIB IN COMBINATION WITH SEMAGLUTIDE INCREASED GLUCOSE-STIMULATED INSULIN SECRETION



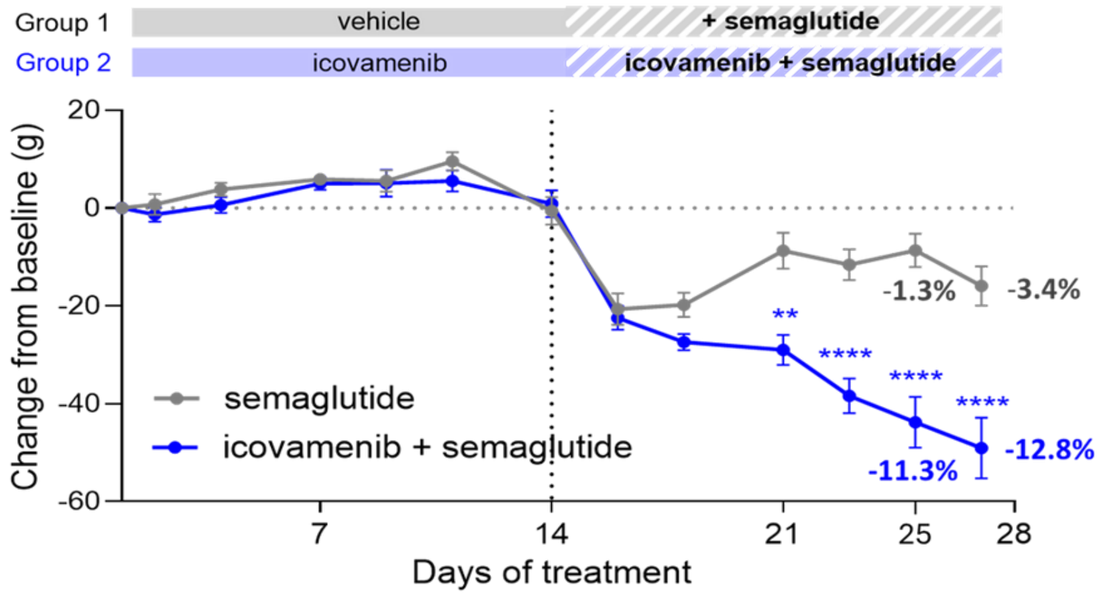
ICOVAMENIB WITH GLP-1RA

Combination Treatment of Icovamenib & Low-dose Semaglutide Reduces Food Intake & Body Weight

APPETITE SUPPRESSION



BODY WEIGHT REDUCTION

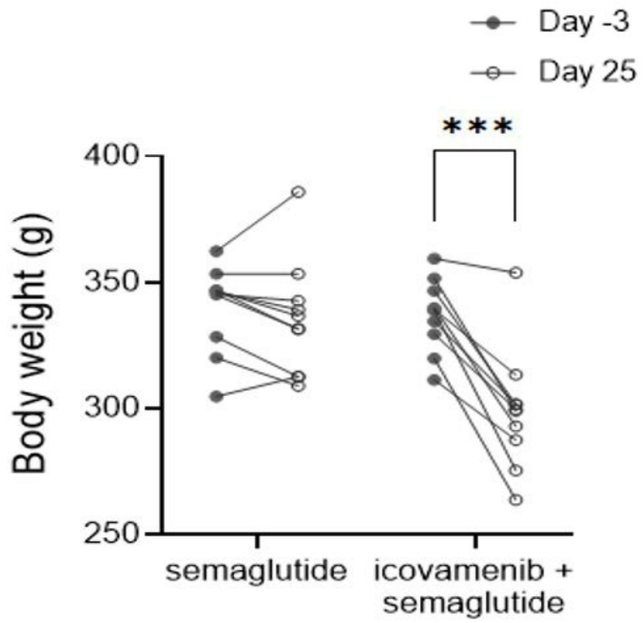


- ❑ OBSERVED SUPERIOR APPETITE SUPPRESSION WITH ABOUT 10% GREATER BODY WEIGHT REDUCTION THAN LOW-DOSE SEMAGLUTIDE ALONE
- ❑ THE OBSERVED BODY WEIGHT REDUCTION WAS PRIMARILY DUE TO FAT MASS LOSS WITH PRESERVATION OF LEAN MASS

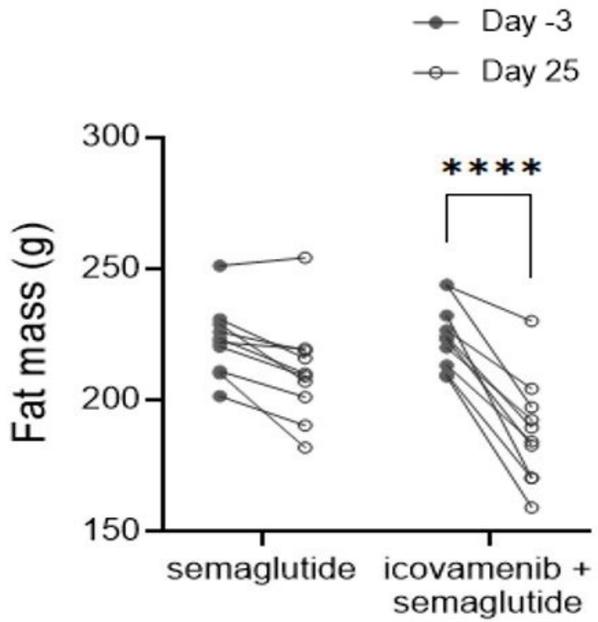
ICOVAMENIB WITH GLP-1RA

Combination of Icovamenib & Low Dose Semaglutide Selectively Promotes Fat Loss with Lean Mass Preservation in ZDF Rats

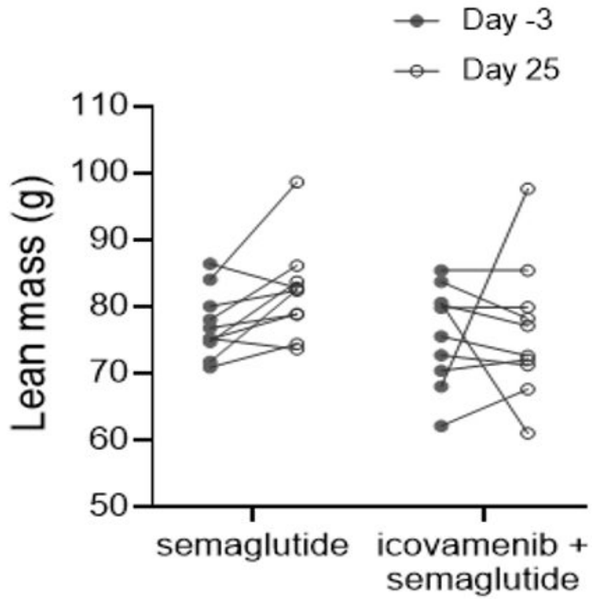
BODY WEIGHT



FAT MASS



LEAN MASS



ICOVAMENIB

Potential first-in-class menin inhibitor for diabetes

First clinical results

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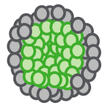
ICOVAMENIB | PRECLINICAL

Icovamenib's mechanism of action

Icovamenib promotes selective and partial reduction in menin levels

Induction of beta cell proliferation



Pancreatic islet

IMPROVED BETA CELL MASS & FUNCTION

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

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

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

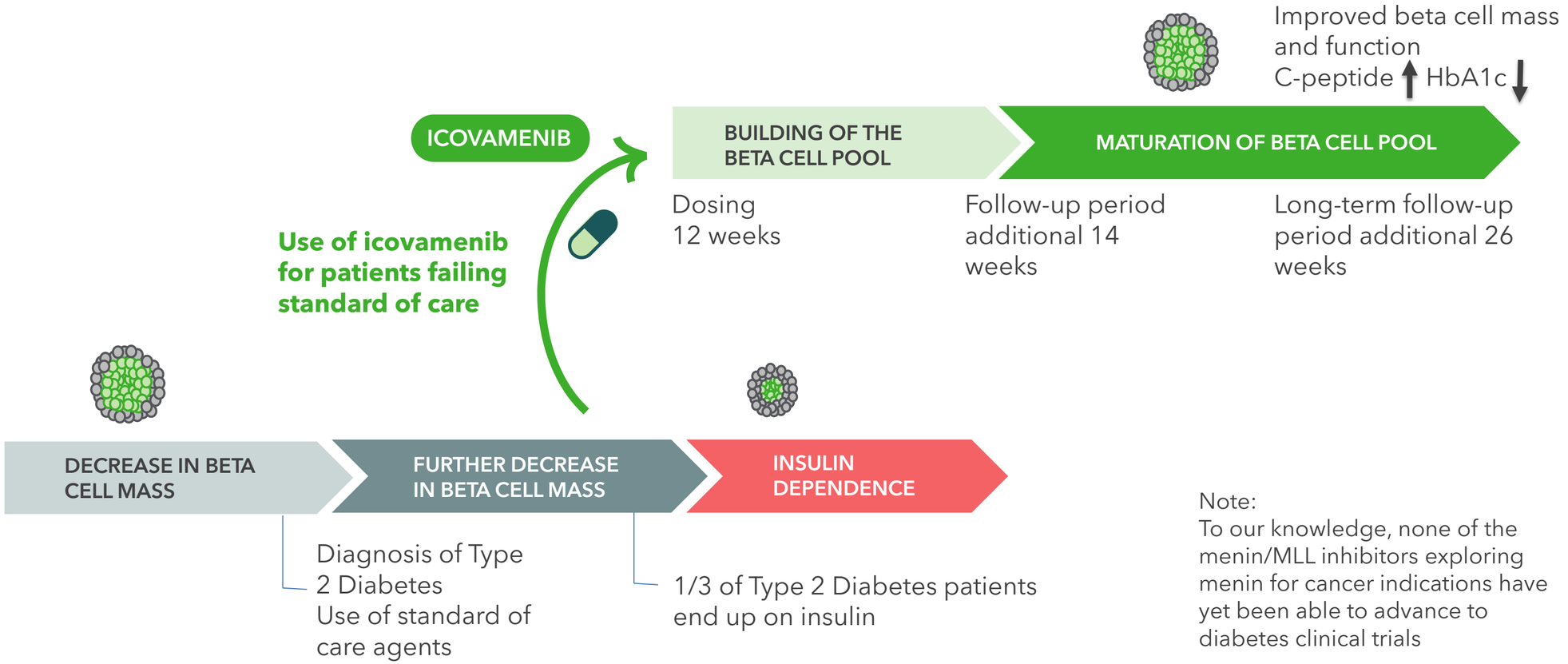
Baseline demographics & characteristics

Per Protocol Population* on 1 or More Antihyperglycemic Agents at Baseline (N=163)

Parameter Mean (SD) or %	Arm A icovamenib (8 wks 100mg QD) (N=45)	Arm B icovamenib (12 wks 100 mg QD) (N=36)	Arm C icovamenib (8 wks 100 mg QD then 4 wks of 100 mg BID) (N=33)	Combined Arms icovamenib (N=114)	Combined Arms placebo (N=49)
Age (yr)	55 (7)	56 (6)	51 (10)	54 (8)	55 (7)
Duration of T2D Diagnosis (yr)	4.3 (1.8)	4.7 (1.8)	4.2 (2.2)	4.4 (1.9)	4.3 (2.0)
Sex (% Female)	(31)	(56)	(36)	(40)	(43)
HbA1c % (SD)	8.3 (1.1)	8.3 (1.0)	8.0 (0.8)	8.2 (1.0)	8.3 (1.0)
Fasting C-peptide (ng/mL)	3.4 (1.2)	3.8 (1.5)	3.7 (1.8)	3.6 (1.5)	3.5 (1.4)
BMI (kg/m^2)	30.9 (4.7)	32.7 (4.5)	32.4 (4.9)	31.9 (4.7)	32.6 (4.2)
BMI <30 kg/m^2 (%)	(49)	(22)	(30)	(35)	(27)
BMI ≥30 kg/m^2 (%)	(51)	(75)	(70)	(64)	(73)
Number of T2D Medications, n (%)					
1	39 (87)	23 (64)	23 (70)	85 (75)	41 (84)
2	4 (9)	11 (31)	7 (21)	22 (19)	6 (12)
3	2 (4)	2 (6)	3 (9)	7 (6)	2 (4)

*Per the COVALENT-111 Protocol the population analyzed includes only subjects who received ≥80% of their planned dosing. A clinical hold interrupted the dosing. Patients were also excluded if they had significant protocol deviation.

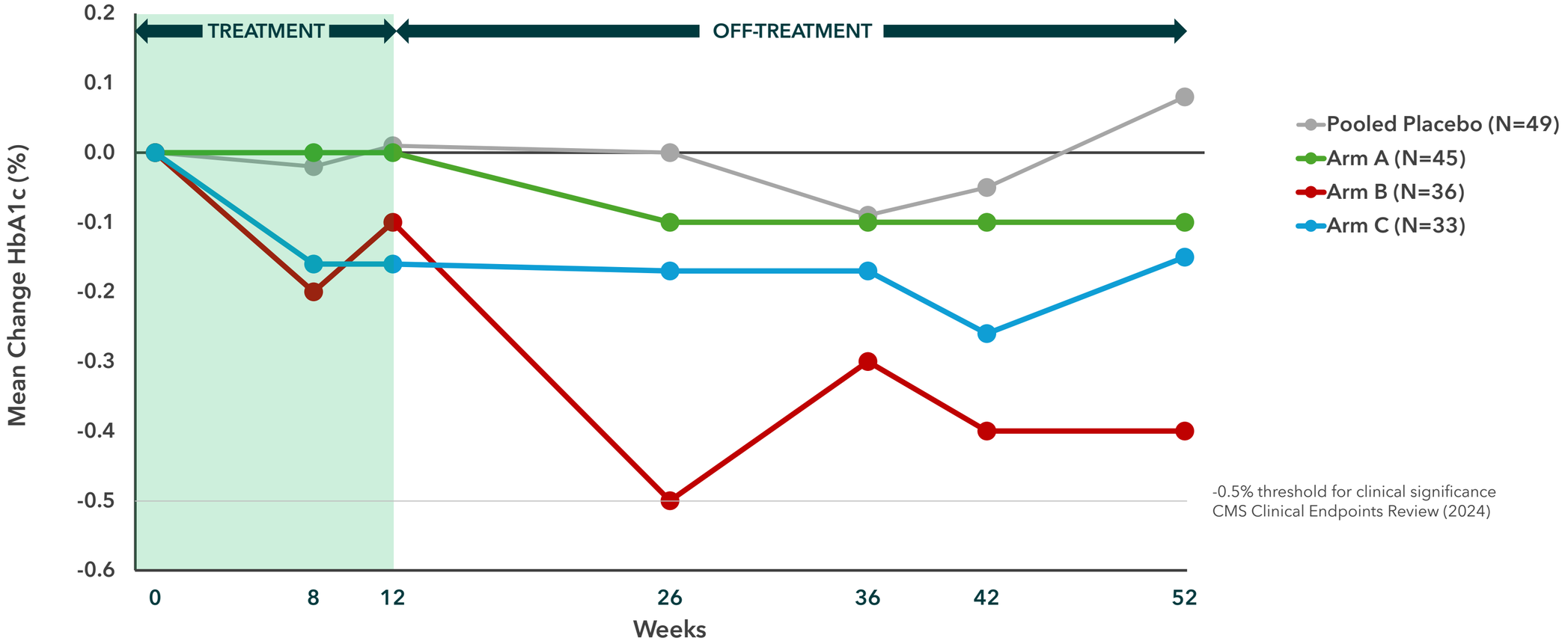
Icovamenib increased beta cell quantity, function & GLP-1 receptor expression following a short treatment period



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

Change in HbA1c from baseline through week 52 – all subtypes

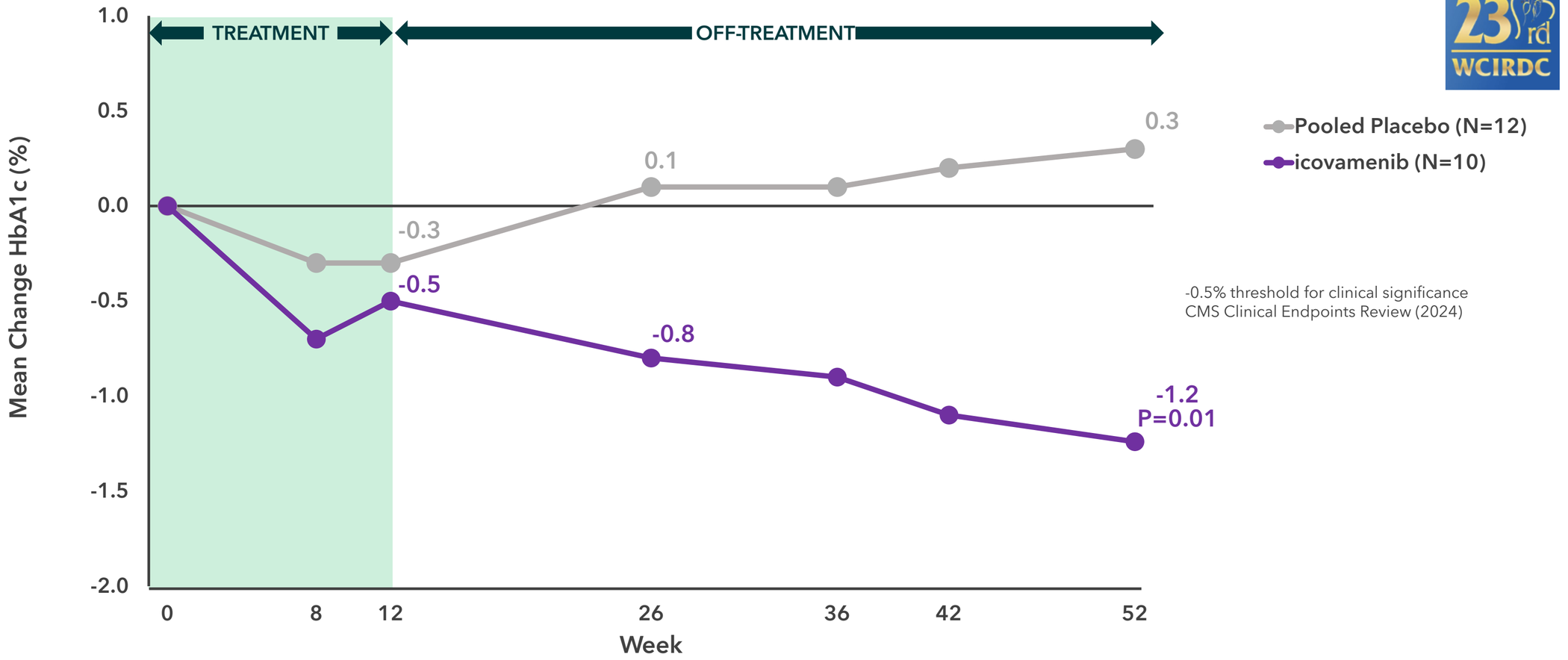
Across treatment durations (Arm A = 8 weeks 100 mg, Arm B = 12 weeks 100 mg, Arm C = 8 weeks 100 mg 4 weeks at 200 mg) per protocol participants taking one or more antihyperglycemic medications at baseline



All presented data utilized a while-on-treatment estimand with mixed model repeated measures (MMRM) analysis and was censored for use of rescue medication, defined as any modification in anti-diabetic therapy.

ICOVAMENIB

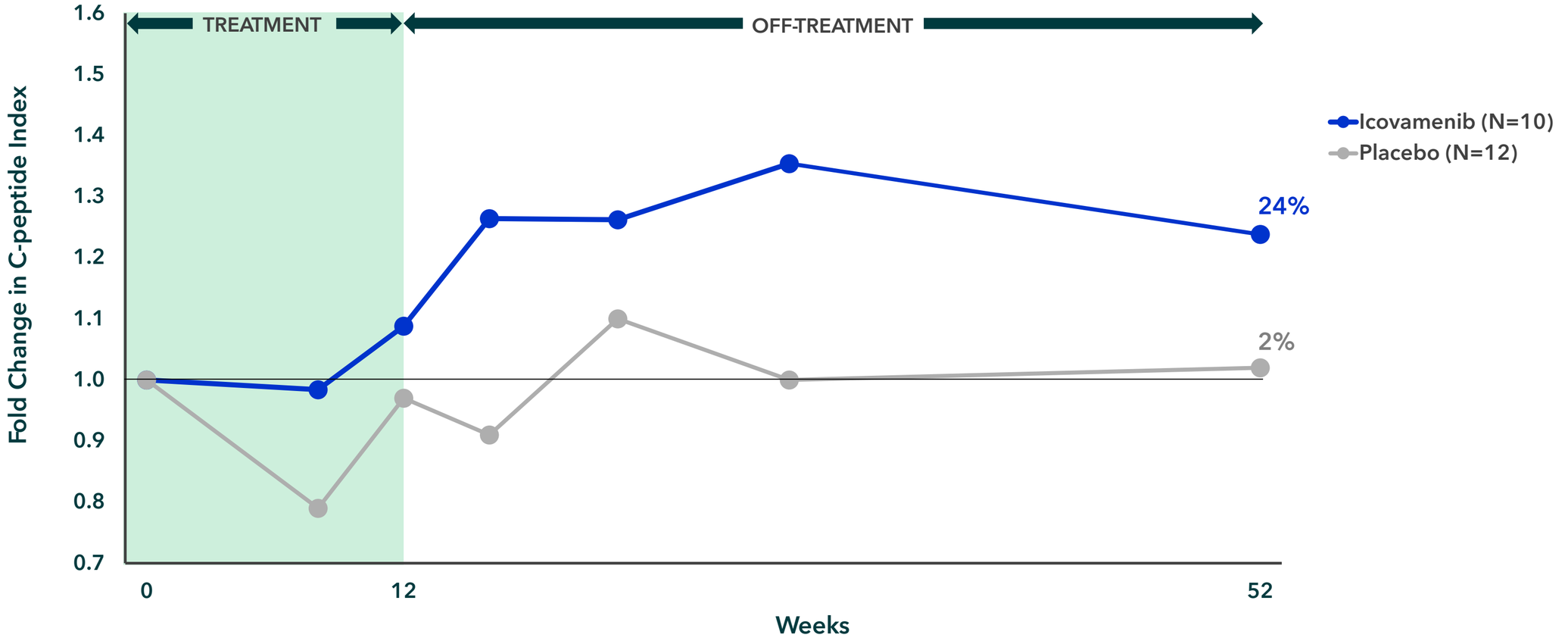
12 weeks of dosing (arms B&C) delivered lasting benefit through 52 weeks for severe insulin-deficient diabetes patients



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'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

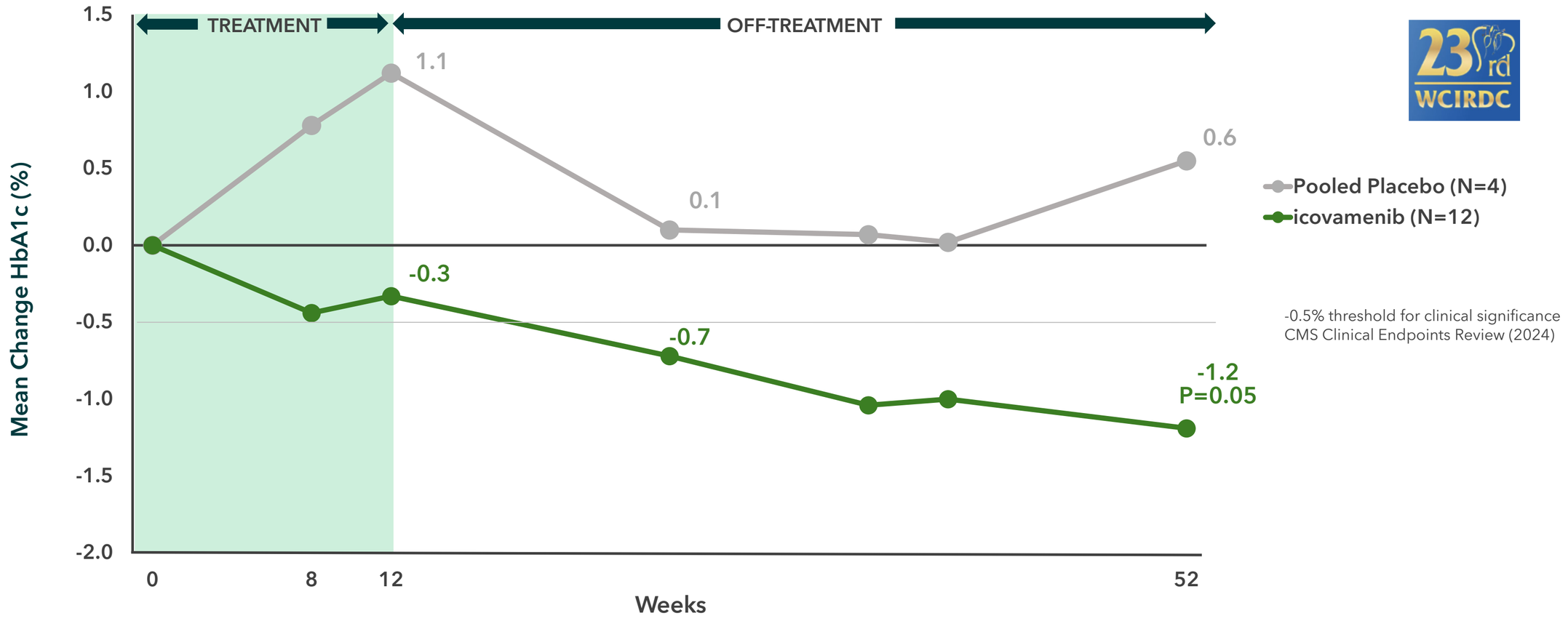
Increased expression of
- **GLP-1**
- **GLP-1 receptor**

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

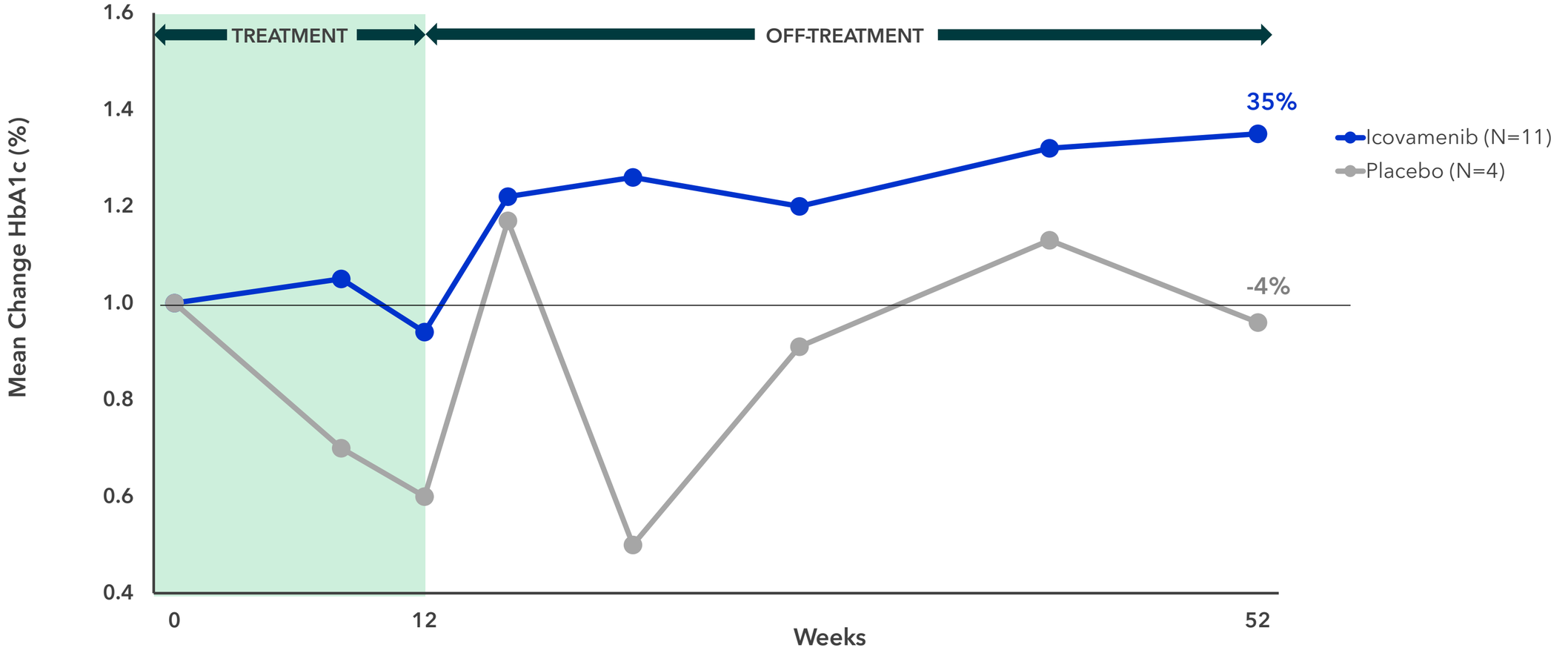
ICOVAMENIB

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



ICOVAMENIB

Icovamenib increased insulin secretion as measured by C-peptide index in GLP-1 RA treated patients – 9 months post last dose



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

ICOVAMENIB I CLINICAL

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 52 (SUSTAIN 8)	-1.5 (1mg)
Mounjaro (GLP-1/GIP Agonist)	**Chronic dosing**	Injectable	Week 40 (SURPASS 1)	-1.9 (5mg) -2.1 (15 mg)
Jardiance (SGLT2 Inhibitor)	**Chronic dosing**	Oral	Week 52 (Extension study)	-0.6 (10mg) -0.6 (25mg)
Januvia (DPP4 Inhibitor)	**Chronic dosing**	Oral	Week 52 (Sitagliptin)	-0.5 (100mg)

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

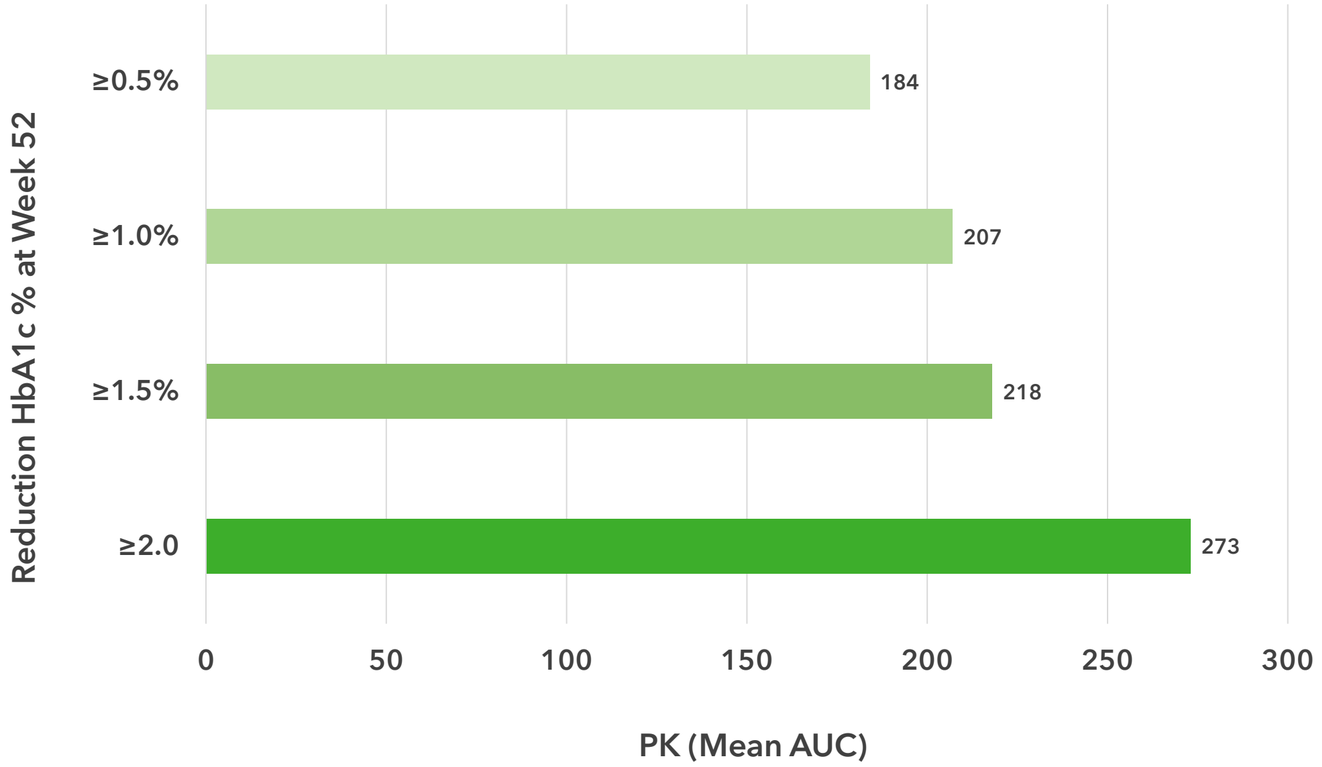
Icovamenib (menin inhibitor)	**12 weeks dosing**	**Oral**	**Week 52 (COVALENT-111)**	**-1.5% to -1.8%* (100 mg)**

*Icovamenib data are from a Phase II study in selected populations: insulin deficient diabetes patients and GLP-1 inadequate responders.

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.

Higher HbA1c reduction was associated with higher icovamenib exposure

Week 52, All Dosing Arms (N=114), HbA1c Reduction vs. Icovamenib Exposure (Mean AUC)



- Dosing timing relative to food will impact icovamenib's pharmacokinetics (PK)
- In a 'Food Effect Study' icovamenib achieved optimal PK exposure when administered within 30 minutes after a meal
- These findings now inform the dosing strategy for the ongoing Phase II studies

ICOVAMENIB

Potential first-in-class oral menin inhibitor for diabetes

Ongoing Phase II Studies

biomea FUSION™

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 Studies

COVALENT-211

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

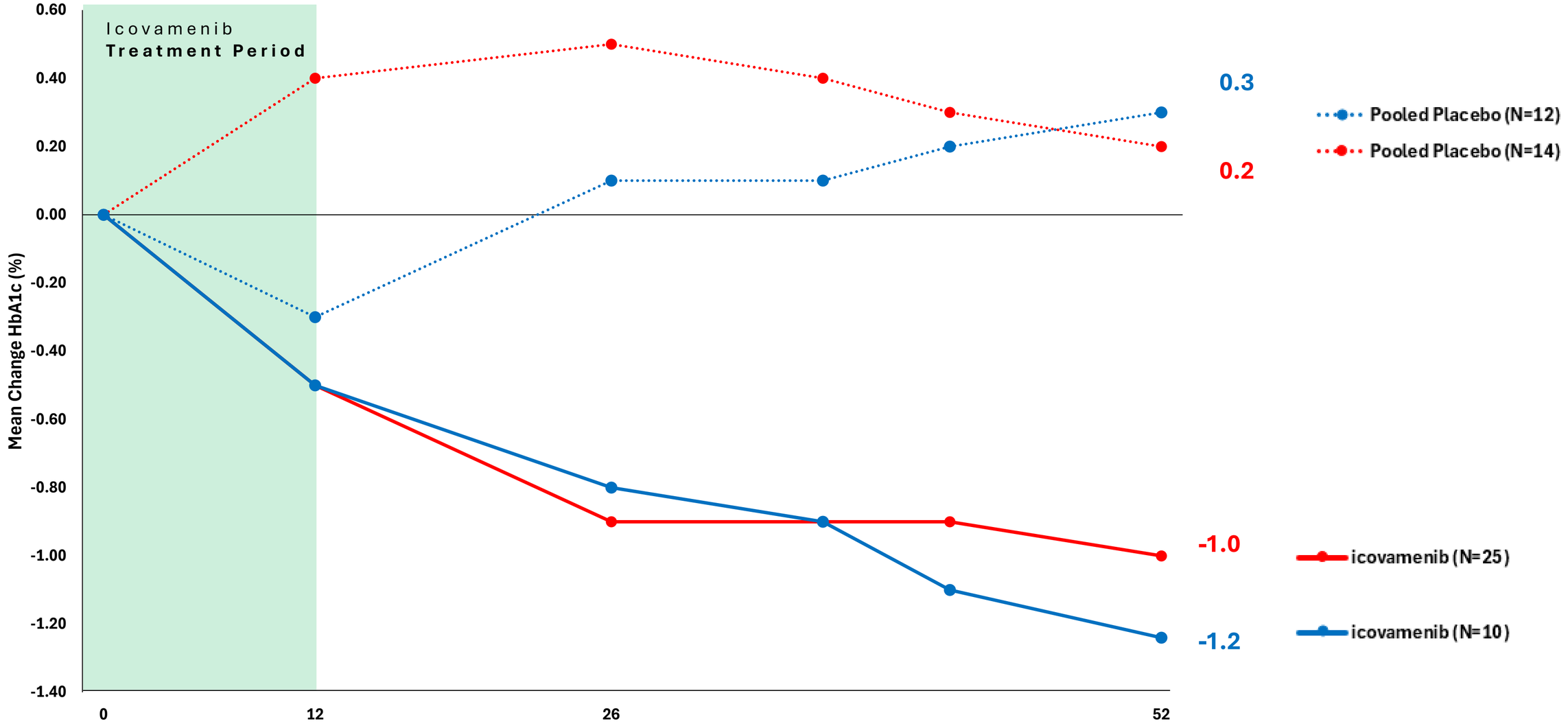
- Adult participants with T2D who were treated with 1-3 antidiabetic medications
- HbA1c 7.5%-10.5% and BMI ≤ 32 kg/m^2
- Background therapy maintained unless rescue required

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/m^2
- Background therapy maintained unless rescue required

Applying enrollment criteria of COVALENT-211 (red) vs published results in SIDDs dosed in COVALENT-111 (blue)



BMF-650

An investigational next-generation oral GLP-1 receptor agonist for obesity

Preclinical results and clinical overview

biomea FUSION™

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.

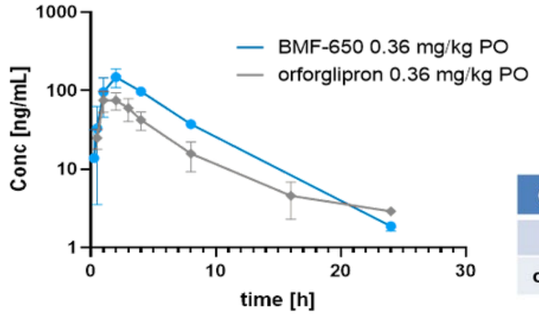
BMF-650 Showed Favorable In Vitro On-Target Activity and Off-Target Selectivity

Compound	GLP-1 human EC_{50}		β-arrestin1 EC_{50}	β-arrestin2 EC_{50}
	25 °C	37 °C		
BMF-650	8.6 nM	2.6 nM	> 10 μM	> 10 μM
orforglipron	2.6 nM	0.1 nM	> 10 μM	> 10 μM

- Good potency on-target to achieve more efficient drug titration
- No off-target concerns from counter-screening assays

Pharmacokinetics of BMF-650 showed very good preclinical bioavailability with low inter-individual variability

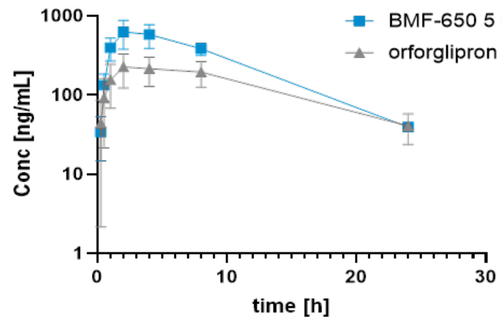
CYNOMOLGUS MONKEY PO PK



BMF-650 showed 2 - to 3 -fold greater oral bioavailability in comparison to orforglipron

Compound	cyno PO	$T_{1/2}$ (h)	%F
BMF-650	0.36 mg/kg	3.66	54.0
orforglipron	0.36 mg/kg	3.70	29.4

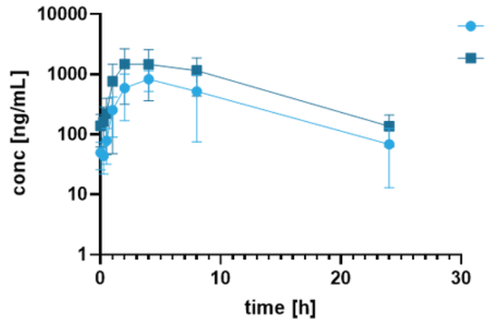
SPRAGUE-DAWLEY RAT PO PK



BMF-650 showed 2 - to 3 -fold greater oral bioavailability in comparison to orforglipron

Compound	rat PO	$T_{1/2}$ (h)	%F
BMF-650	5 mg/kg	5.14	32.6
orforglipron	5 mg/kg	7.44	11.2

CYNOMOLGUS MONKEY PK DAY 6 BMF -650



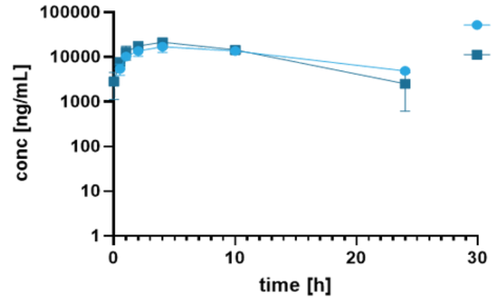
Dose Proportionate Exposure

Compound	cyno PO	Day	AUC**
BMF-650	3 mg/kg	6*	9,353
BMF-650	6 mg/kg	6#	19,918

* d6 of 6 days of daily PO dosing; d6# after 6 additional days of PO dosing at indicated dose level. ** hr*ng/mL

PO =per oral

SPRAGUE-DAWLEY RAT PK DAYS 1, 7 BMF -650



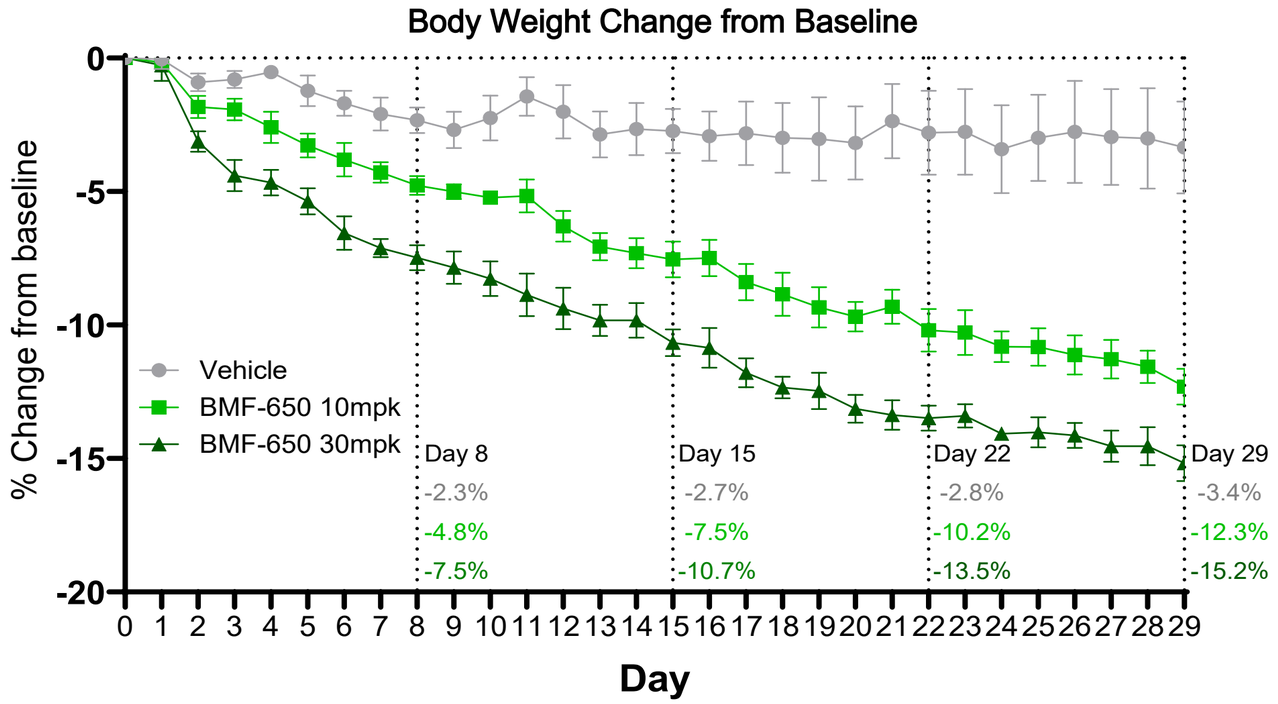
Continuous Exposure after multiple days

Compound	rat PO	day	AUC*
BMF-650	70 mg/kg	1	269,100
BMF-650	70 mg/kg	7	289,370

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

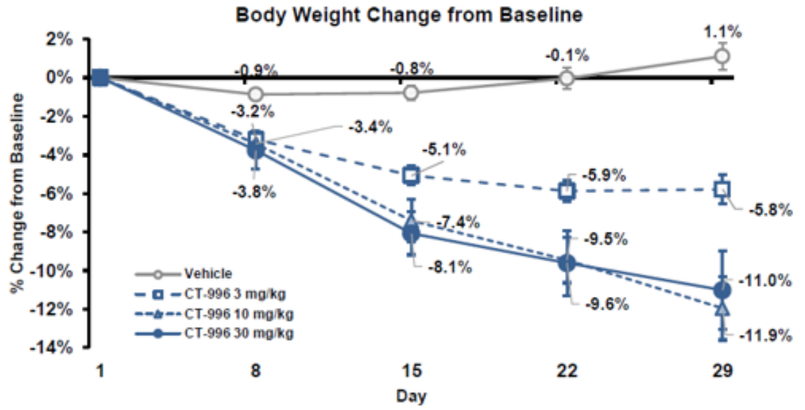


Figure 6. Effects of CT-996 on body weight in obese cynomolgus monkeys following once-daily oral administration. Weekly body weight percent change is represented as mean (± SE) from baseline. N = 6/group.

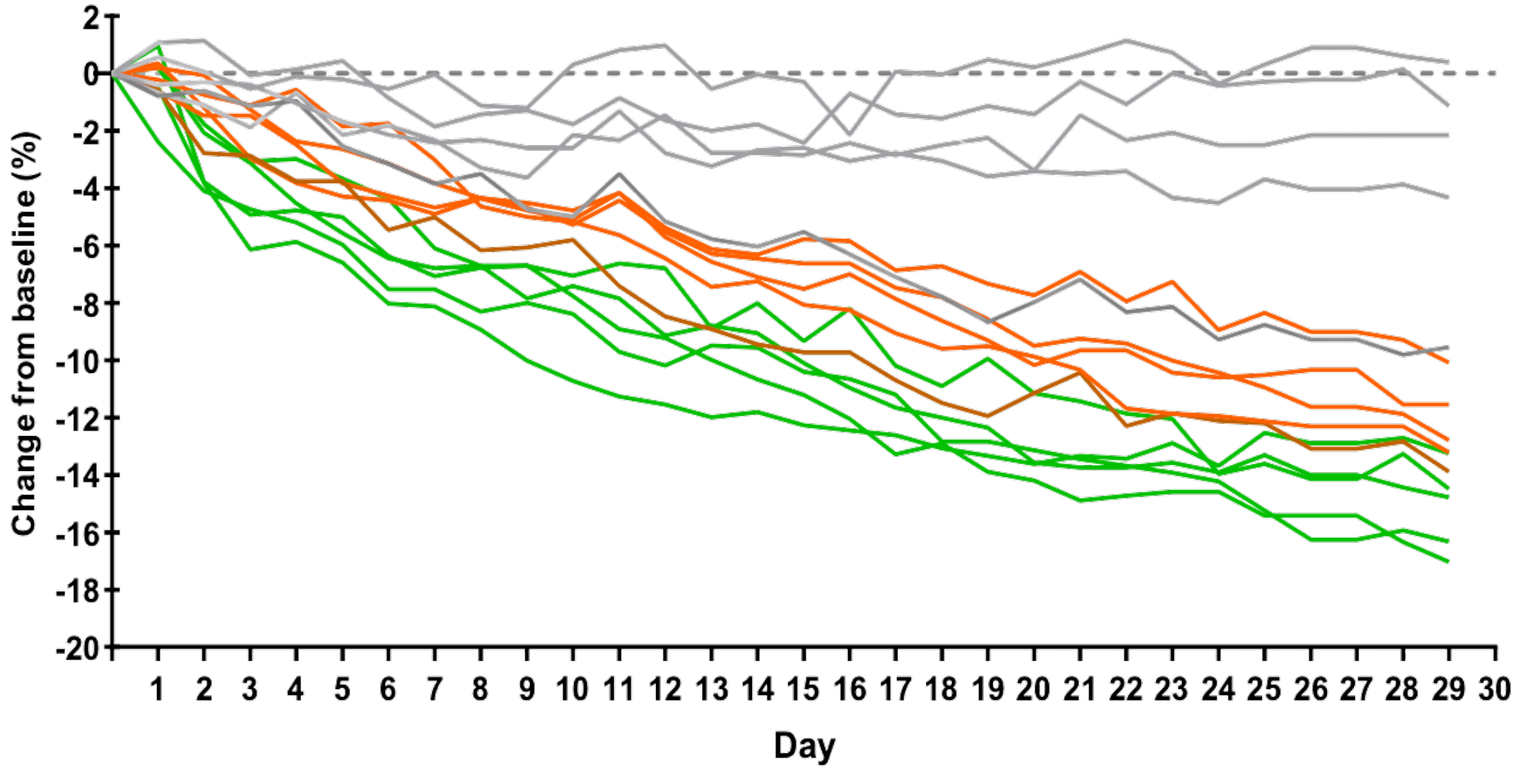
Literature data; Carmot Therapeutics (now part of the Roche group), ADA 2024.

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 | PRECLINICAL

Oral BMF -650 Demonstrates Strong Dose Dependent Body Weight Reduction in Obese Cynomolgus Monkeys

BODY WEIGHT CHANGE (individual obese monkey)



Mean reduction in BW (Day 29)

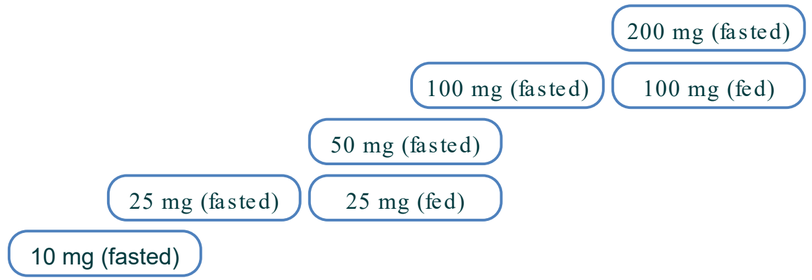
Vehicle	-3.4%
BMF-650 10 mg/kg	-12.3 %
BMF-650 30 mg/kg	-15.2 %

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 



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 *(>3M US Patients)* [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.NCHS Data Brief dated August 2025. Accessed January 28, 2026 (Based on company calculations)
4.Chitnis AS. Clinical effectiveness of liraglutide across body mass index in patients with type 2 diabetes in the United States: a retrospective cohort study. Adv Ther. 2014 Sep;31(9):986-99 (Based on company calculations)
5.National Center for Health Statistics August 2023. Accessed January 28, 2026

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 menin inhibitor - the control switch to beta cell restoration

Restores functional beta-cell mass to address disease biology in type 2 diabetes

- Increased insulin production and synergy with GLP-1 shown in preclinical models
- Durable HbA1c reduction and C-peptide increase through 52 weeks after a 12-week course in the first Phase II trial in T2D patients failing standard of care
- Two Phase II studies underway with 26 weeks primary endpoint data anticipated in 4Q 2026 with the potential to address over 10M U.S. T2D diabetes patients

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

Developed for consistent exposure, higher bioavailability and improved tolerability with scalable weight reduction

- Improved bioavailability, better plasma protein binding, greater oral exposure with lower variability
- Demonstrated weight reduction and generally well tolerated in preclinical models
- Phase I clinical study in obese healthy volunteers ongoing with 28-day weight reduction data anticipated in 2Q 26, aiming to address over 100M U.S. obese patients

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.

THANK YOU

(NASDAQ: BMEA)

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

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