

Biomea Fusion Corporate Presentation

Q4 2026

At-a-Glance

biomea FUSION™

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

First-in-class oral small molecule for the treatment of diabetes targeting menin - the natural control switch to beta cell restoration in Type 1 and Type 2 Diabetes

Restores functional beta-cell mass and improve endogenous insulin production

BMF-650

Next-generation oral GLP-1 receptor agonist

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

1. www.breakthrought1d.org 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

Investment Thesis

Biomea is building a leading metabolic disease company with two differentiated programs and multiple near-term catalysts

- First-in-class beta-cell restorative therapy targeting menin in development for diabetes
- Potential best-in-class Type 1 & 2 diabetes agent
- Next-generation oral GLP-1 receptor agonist (BMF-650) with positive Phase I weight loss results and favorable GI tolerability
- Multiple Phase II readouts over the next 12 months
- Experienced leadership team with a clear vision and execution plan
- Cash runway through key upcoming value inflection read outs

Icovamenib milestones – overview next 18 months

Icovamenib in Type 1 Diabetes

PLANNED T1D Investigator Sponsored Study | Phase 2 Study | n=40 | Enrollment anticipated to start 1Q 27

PLANNED T1D Biomea Sponsored Study | Phase 2 Study | n=36 | Enrollment anticipated to start 1Q 27

T1D
FDA End of Phase 2 Meeting and Phase 3 Initiation

Icovamenib in Obesity

LEICESTER DIABETES CENTER in Obesity | Phase 2 Study | n=64 | Topline data anticipated: End of 27

Icovamenib in Type 2 Diabetes

COVALENT-211 in insulin deficient T2D | Phase 2 Study | n=64 | Fully enrolled | Topline data anticipated: 1Q 27

Secondary Endpoint: HbA1c change at Week 52

COVALENT-212 in GLP-1 uncontrolled T2D | Phase 2 Study | n=60 | Start: 2Q 26 | Enrollment ongoing | Topline data anticipated: 2Q 27

Secondary Endpoint: HbA1c change at Week 52

T2D
FDA End of Phase 2 Meeting and Phase 3 Initiation

4Q 2026 | 1Q 2027 | 2Q 2027 | 3Q 2027 | 4Q 2027 | 2028

Current Cash Position
Runway into 2Q 2027

The Opportunity

Growing market with large unmet need

Diabetes

>38M

People living with diabetes in U.S.[1]

>35M

People with T2D in U.S.[2]

1:3

People in U.S. with T2D using Insulin[3]

25%

Of U.S. healthcare dollars go to treat those diabetes[4]



Obesity

>190M

People in U.S. are obese or overweight[5]

100M +

People in U.S. are obese[5]

5M+

People in U.S. discontinue GLP-1[6]

Large unmet need

for durable, well-tolerated therapies

Addressing root biology has the potential to address patient outcomes

1. CDC, Natl. Diabetes Stat. Rep., 2022
2. ADA, Standards of Care in Diabetes, Diabetes Care, 2024
3. Li J Diabetes Complications 2012;26(1):17-22
4. CDC Health and Economic Benefits of Diabetes Interventions
5. CDC National Health and Nutrition Examination Survey
6. IQVIA prescription data

Restoring beta-cell health to delay insulin dependence

and reduce complications and disease burden

Early-Stage

β-cell stress

Insulin deficiency/resistance

Metabolic dysregulation

Mid-Stage

Microvascular disease

Diabetes nephropathy (early)

Peripheral neuropathy

Autonomic neuropathy (early)

Late-Stage

Macrovascular disease

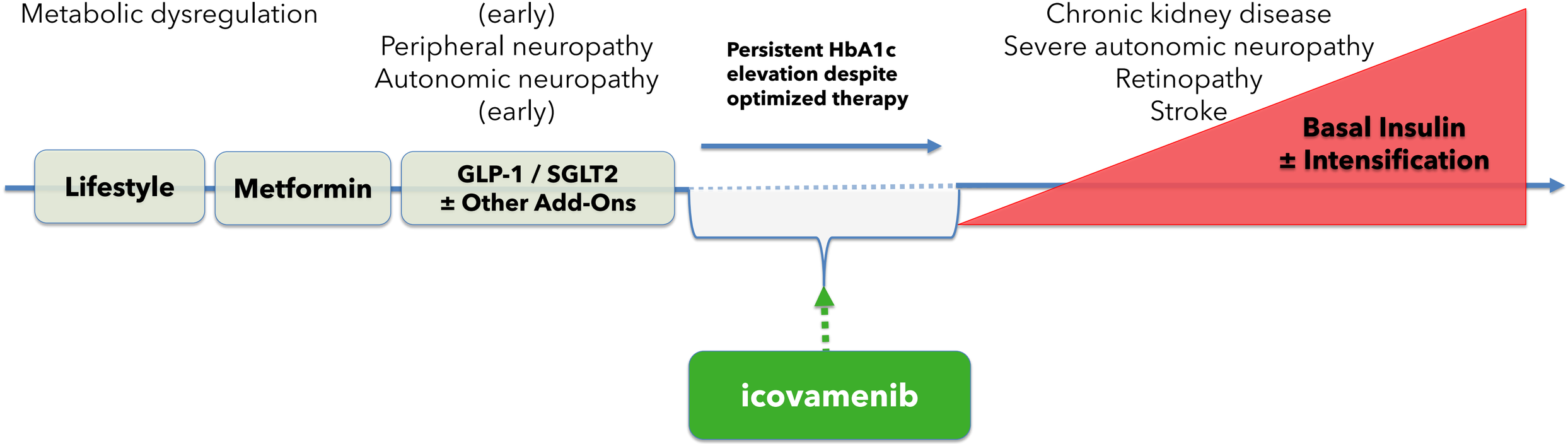
Coronary artery disease

Chronic kidney disease

Severe autonomic neuropathy

Retinopathy

Stroke



In the U.S., >50% 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

First-in-class menin targeting agent in development for the treatment of diabetes – PRECLINICAL RESULTS

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Menin is naturally inhibited during pregnancy and breastfeeding

- allowing for adaptive beta cell mass increase & reduced diabetes risk

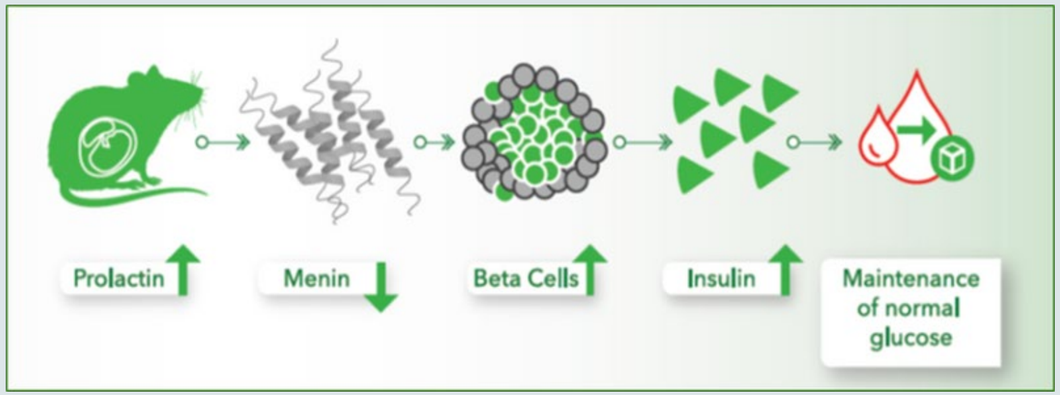
- Physiologic states such as pregnancy and lactation suppress menin, enabling beta-cell expansion and increased insulin output

- 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





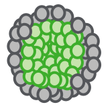
Icovamenib's mechanism of action

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 FOR TYPE 1 AND TYPE 2 DIABETES

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

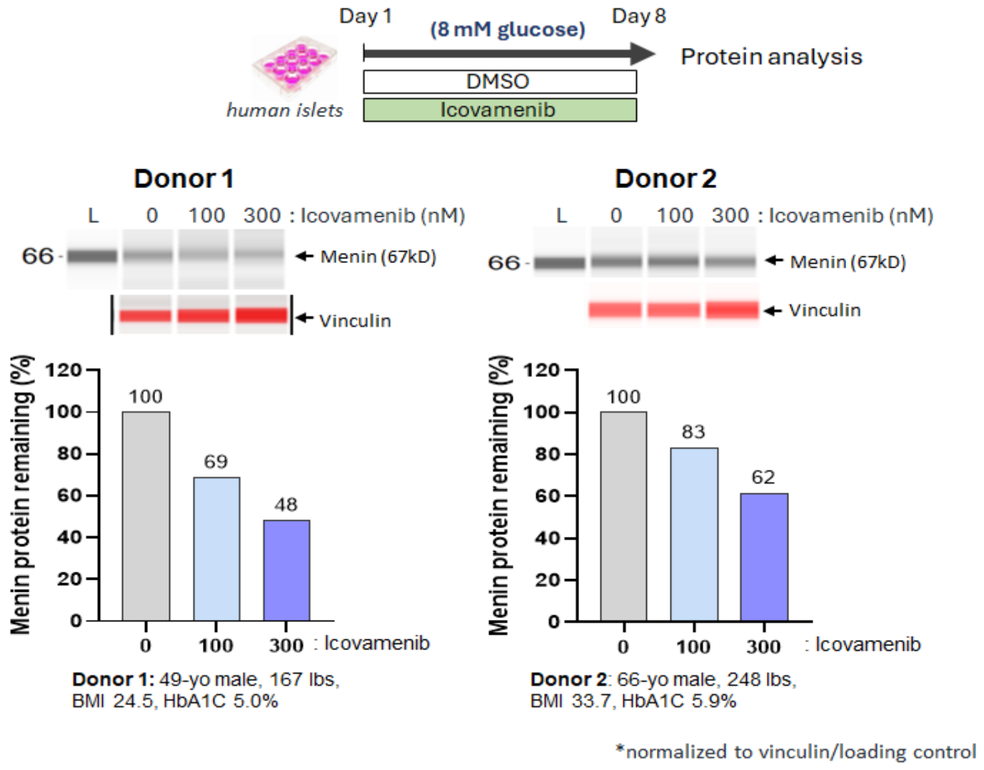
IMPROVED INCRETIN EFFECTS

- ↑ 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

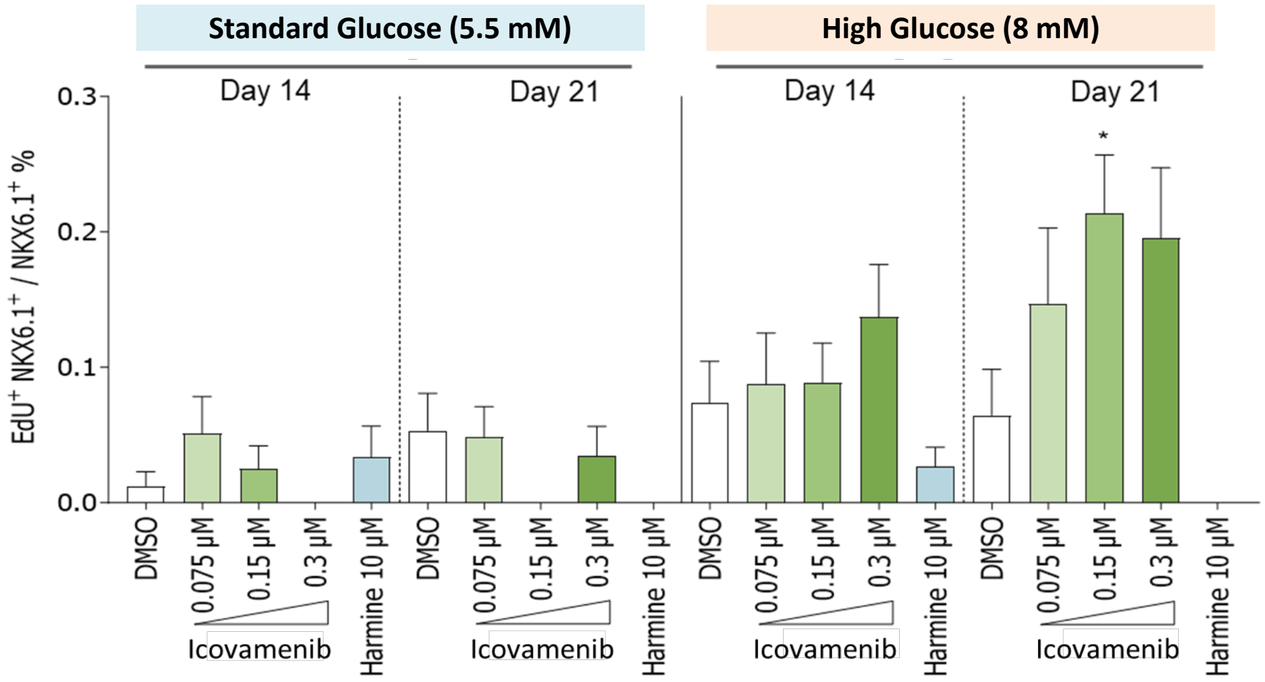
Icovamenib downregulated menin protein levels & promoted beta cell proliferation in ex vivo human islet cultures

MENIN LEVELS DOWNREGULATED



Somanath, et al. (EASD) Diabetologia 68 (Suppl 1), 1–754 (2025). Oral presentation #66

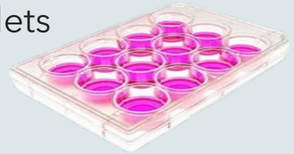
ICOVAMENIB CONDITIONALLY PROMOTED BETA CELL PROLIFERATION ONLY UNDER HYPERGLYCEMIC CONDITIONS



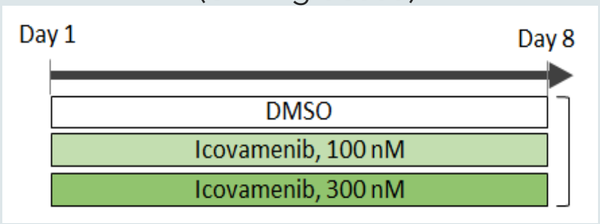
Frias, et al. (WCIRDC) Metabolism,Vol153, Supplement,2023,#88

Icovamenib enhanced GLP-1 receptor & insulin expression in human islets

Cadaver derived human islets

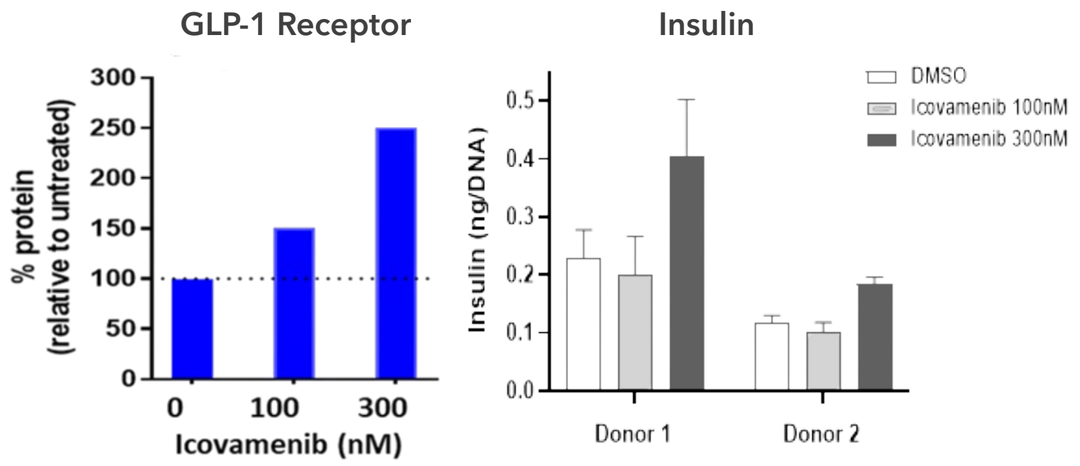


Culture 7 days under glucotox conditions (8mM glucose)



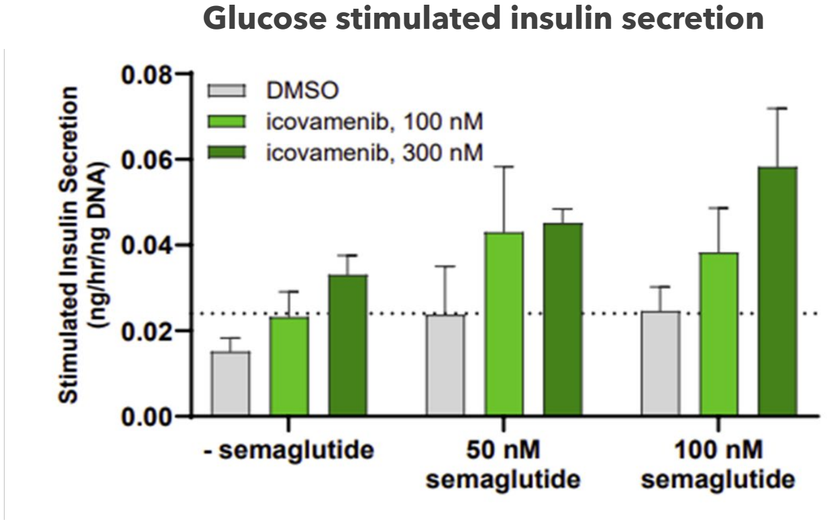
- Gene expression & Protein analysis
- Glucose Stimulated Insulin Secretion -/+ Semaglutide

ICOVAMENIB INCREASED GLP-1 RECEPTOR AND INSULIN PROTEIN LEVELS



Balakrishnan, et al. (WCIRDC) Supplement,2025,156226. Oral presentation

ICOVAMENIB ENHANCES THE RESPONSIVENESS OF HUMAN ISLETS TO GLP-1 RECEPTOR AGONISTS

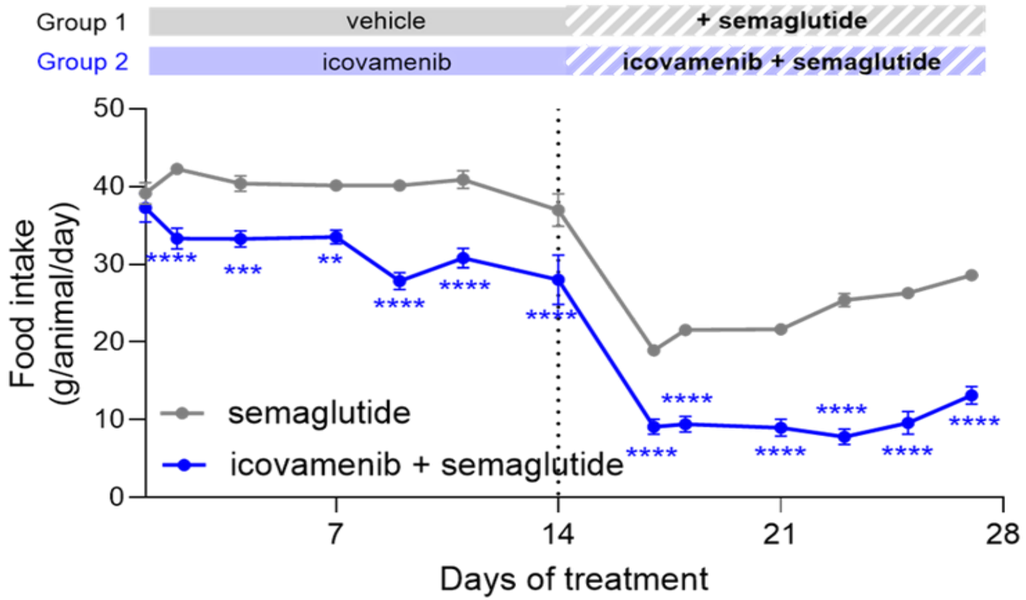


Somanath, et al. (EASD) Diabetologia 68 (Suppl 1), 1–754 (2025). Oral presentation #66

ICOVAMENIB WITH GLP-1RA

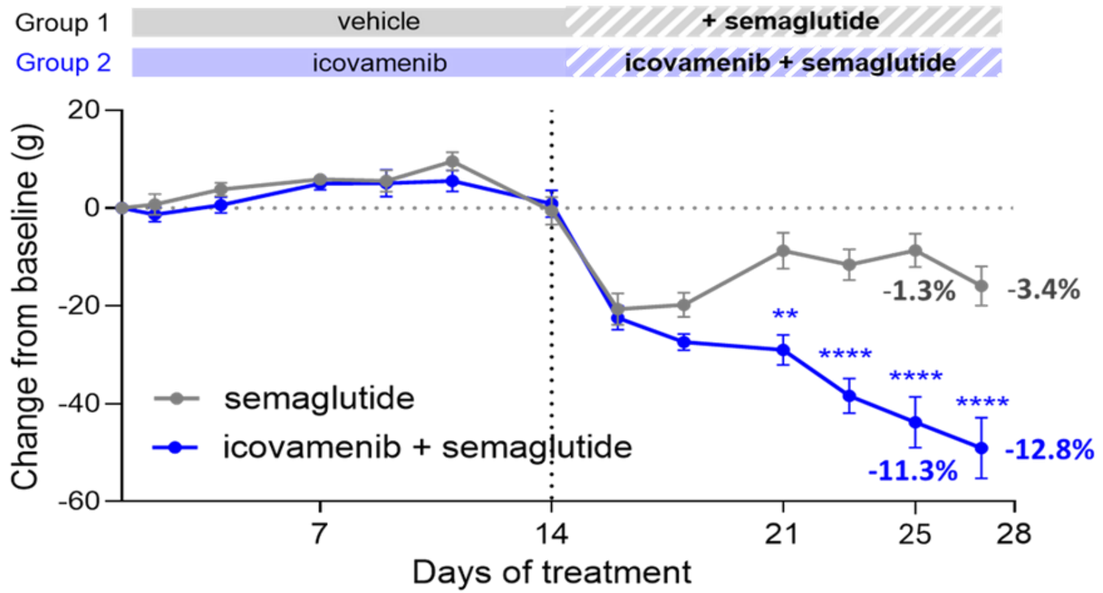
Combination Treatment of Icovamenib & Low-dose Semaglutide Reduces Food Intake & Body Weight

APPETITE SUPPRESSION



Somanath, et al. (ADA) Diabetes 2025;74 (Supplement_1):870-P

BODY WEIGHT REDUCTION



- 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



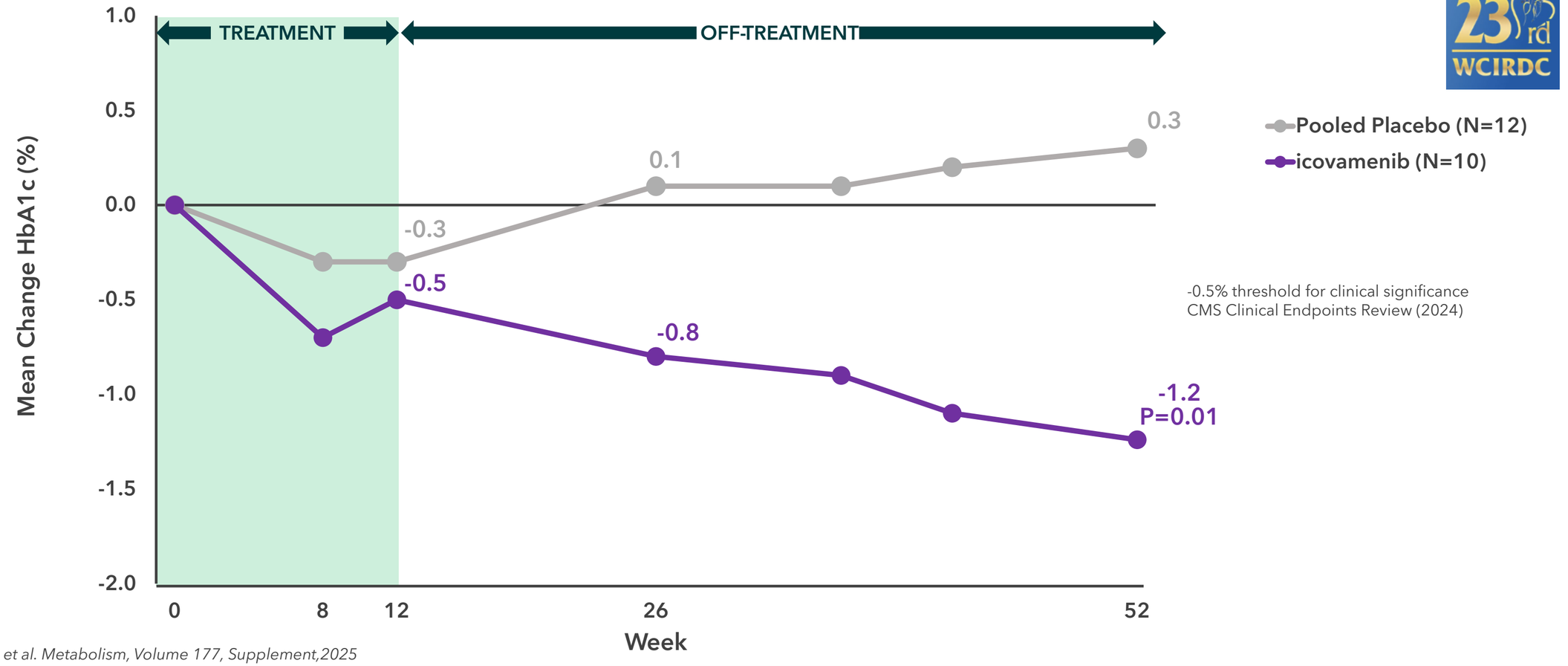
ICOVAMENIB

First-in-class menin targeting agent in development for the treatment of diabetes - TYPE 2 DIABETES

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ICOVAMENIB | COVALENT-111

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

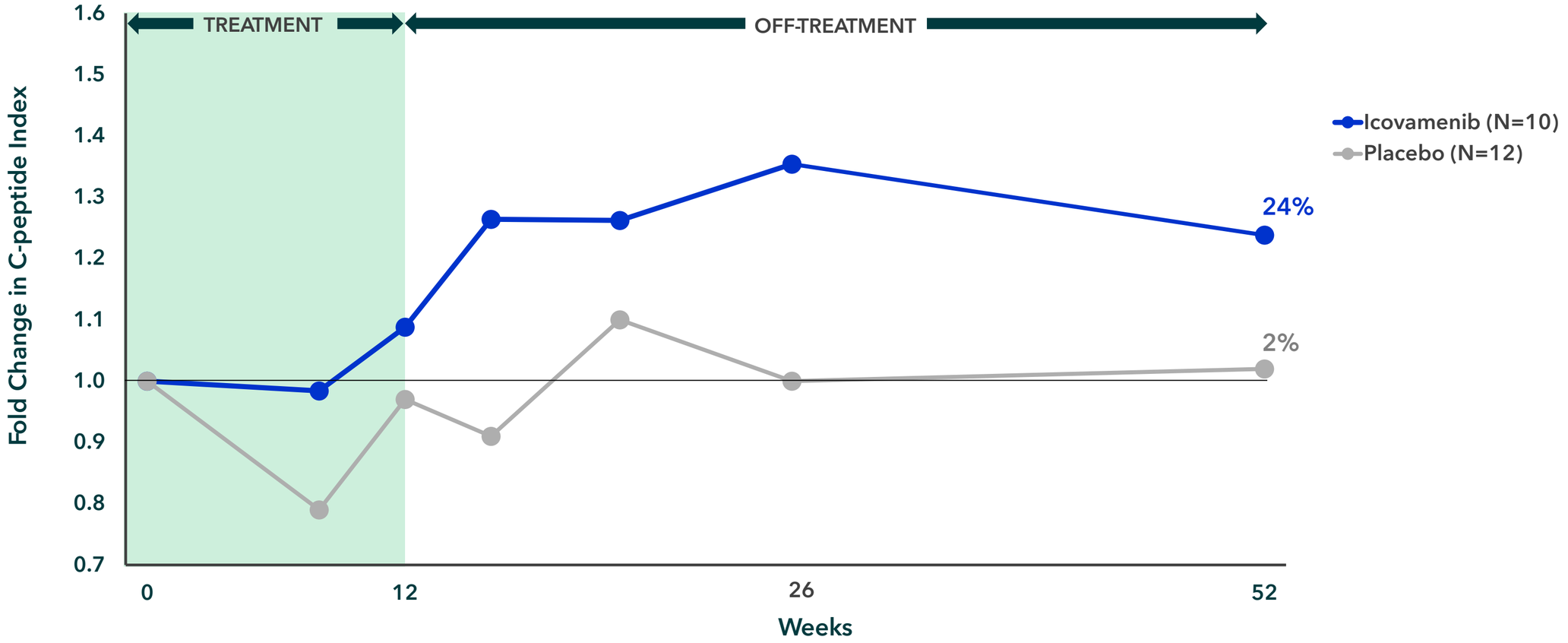


Frias, et al. Metabolism, Volume 177, Supplement,2025

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

ICOVAMENIB | COVALENT-111

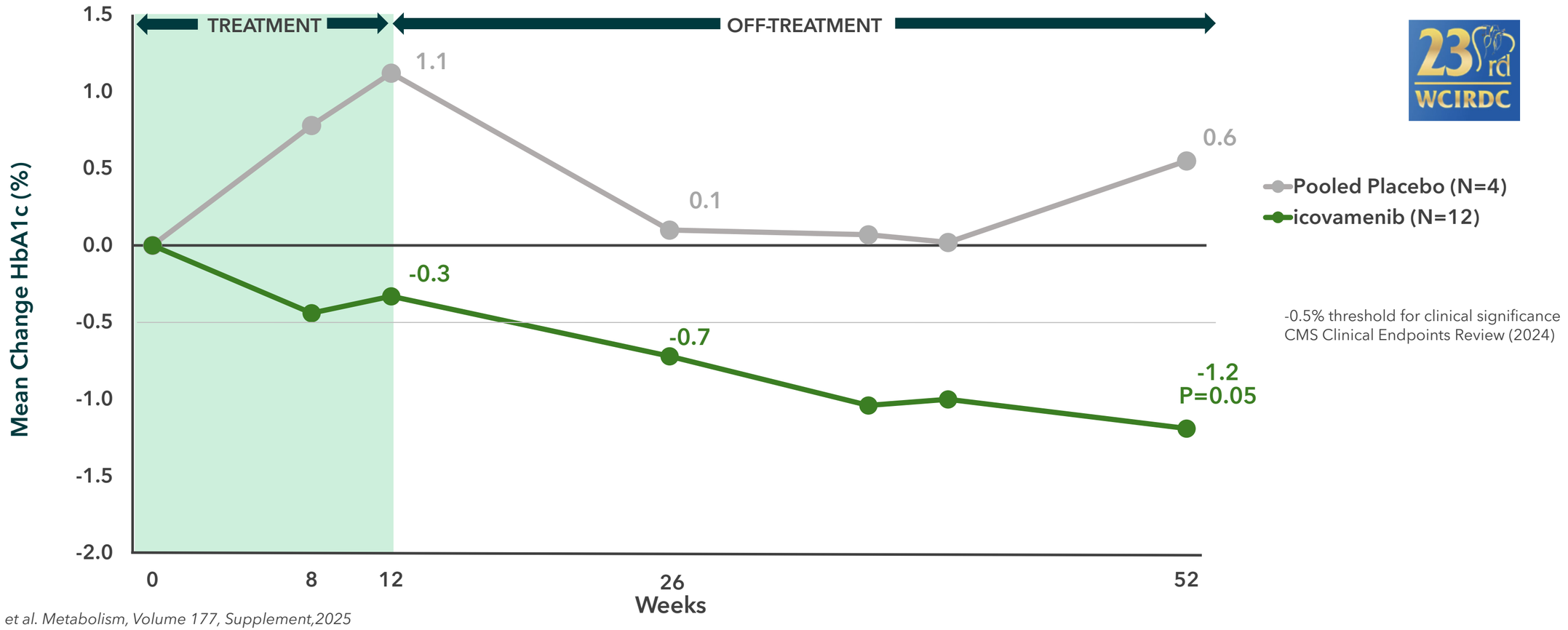
Icovamenib increased insulin secretion as measured by C-peptide index in severe insulin-deficient patients (arms B&C)



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

ICOVAMENIB | COVALENT-111

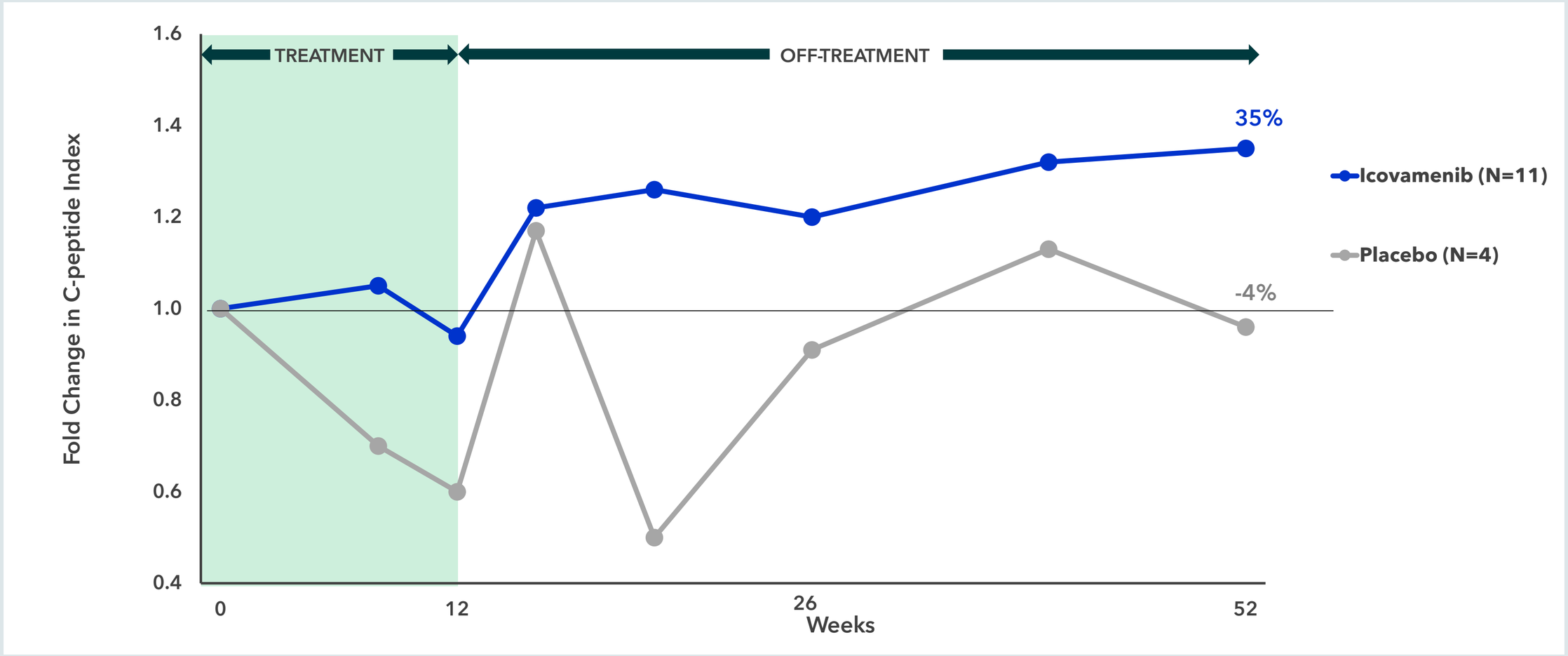
Patients on a GLP-1 based therapy at enrollment showed durable & clinically meaningful response in reduction of blood sugar (HbA1c)



Frias, et al. Metabolism, Volume 177, Supplement,2025

ICOVAMENIB | COVALENT-111

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)

Frias, et al. Metabolism, Volume 177, Supplement,2025

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 | COVALENT-111

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

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

*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.



ICOVAMENIB

First-in-class menin targeting agent in development for the treatment of diabetes - TYPE 1 DIABETES

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Type 1 Diabetes – Opportunity

Established market with large unmet need. Patients suffer from loss of beta cells and require the use of exogenous insulin - Type 1 diabetes untreated is deadly.

Type 1 Diabetes in the US and WW

US>2M WW>10M

People living with Type 1 diabetes [1]

of approved Agents

one

Teplizumab is approved in Stage 2 of T1D and has an accelerated approval in Stage 3 for children 8-17 years old, contingent on confirmatory evidence of clinical benefit.

Investigational Therapies in Development

Focus primarily on immune modulation and preservation

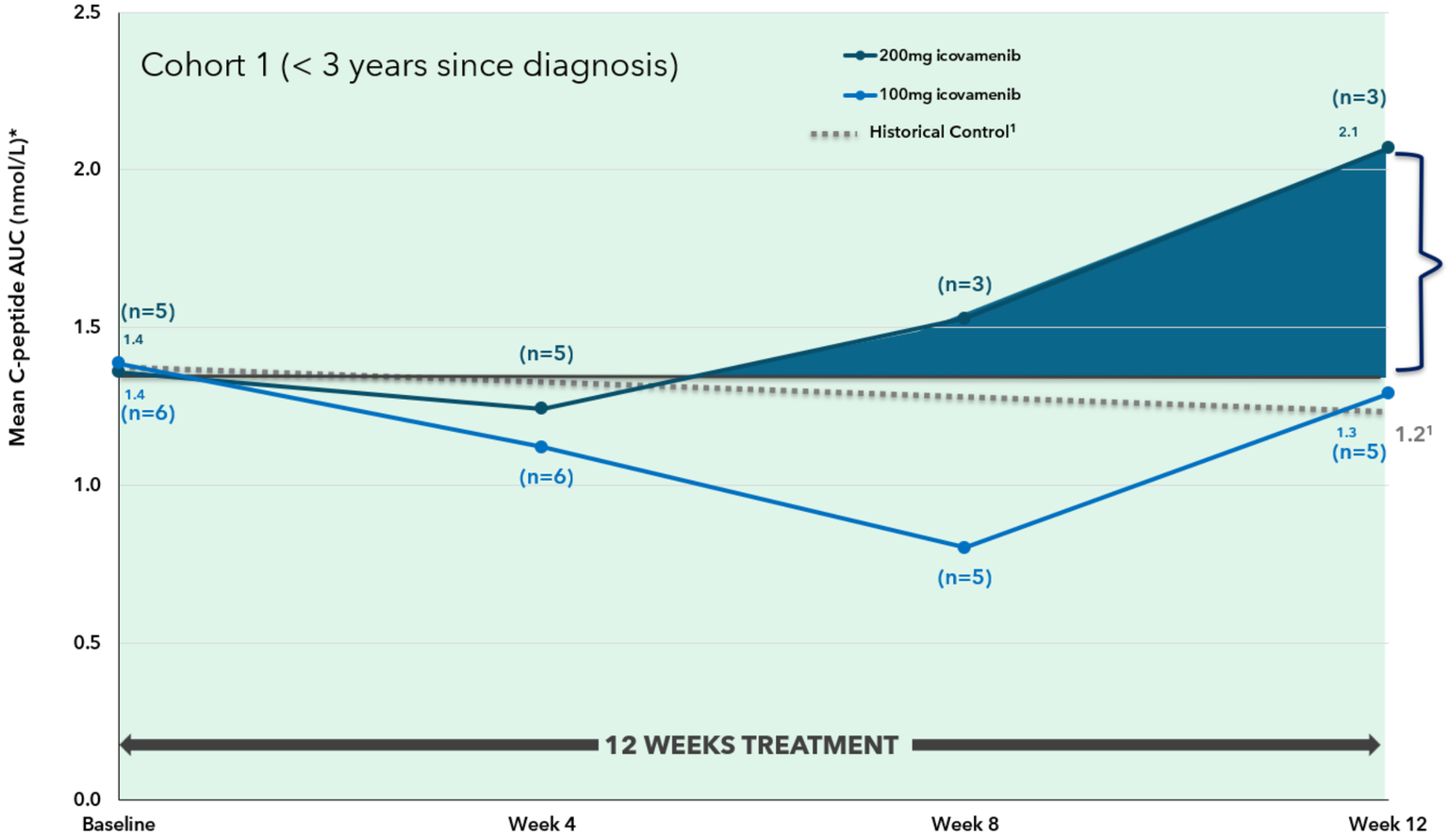


- Immune modulation to slow autoimmune destruction[1]
- Preserve remaining beta-cell function
- Mose effective when started shortly after diagnosis[2] *(<90 days, new onset T1D)*
- Slows decline in C-peptide over time[3]

GOAL: Slow disease progression

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

Compelling Signal of Beta Cell Restoration in T1D with Icovamenib



- 52% Mean increase in C-Peptide AUC* at Week 12
- Durable effect through Week 52 after only 12 weeks of dosing
- Greatest response in patients ≤3 years since diagnosis
- No Systemic immune activation
- Favorable safety profile through 52 weeks

Data represents post-hoc analysis of patients who received per statistical analysis plan, 80% of planned doses

[1] Historical control in T1D patients (n=1549) C-peptide declining over first 7 years at 47% yearly. Diabetes Care. 2018 Jun 7;41(7):1486–1492

** 4-hour Mixed Meal Tolerance Test (MMTT)*

Frias, J. (ADA) Diabetes 2026; 75 (Supplement_1): 2858–LB.

Potential to Expand the Treatable Population in Type 1 Diabetes

Approved Therapy in T1D TEPLIZUMAB (Tzield®)	Key Characterization	ICOVAMENIB
Newly diagnosed Stage 3 T1D	Eligible Population	Newly diagnosed and established T1D with residual beta cell function
0-90 days from diagnosis	Time From Diagnosis	0-3+ years demonstrated clinically
Pediatric only (8 to 17 years)	Age	Adult T1D patients at first with pediatric to follow in development
Immune modulation	Mechanism of Action	Beta cell restoration / preservation
Two 12-day infusion 26 weeks apart	Treatment	Oral, once daily
Delay insulin dependence	Goal	Restore endogenous insulin secretion and preserve beta cell function

A SUBSTANTIALLY LARGER TREATABLE PATIENT POPULATION

Icovamenib potentially expands the treatment window and the patient population by more than 10-fold

CLINICAL DEVELOPMENT TIMELINE

2026
- New Type 1 Diabetes Study Initiation

2027
- Enrollment completion (n=36)
- Phase 2 data readout

2028
- End of Phase II FDA meeting
- Fast Track/ Break-through potential
- Potential to initiate registrational study in broader T1D



BMF-650

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

- Nonclinical and Phase 1 Clinical Results

biomea FUSION™

BMF-650: A potential best-in-class oral GLP-1 receptor agonist

THE CURRENT GLP-1 MARKET

Injectable GLP-1 RAs

- Some patients averse to injections
- Inconvenient
- Injection site reactions

First Generation Oral GLP-1 RAs

- High rates of GI side effects
- Many do not reach maintenance dose
- High discontinuation rates

Long, complex titration
Typically, 4-8+ steps, which can take 3-9 months depending on product

7 out of 10
Patients **discontinue** GLP-1 therapy due to side effects[1]

BMF-650
Designed for sustainable weight loss management

Next Generation Oral GLP-1 RA

Oral administration
Convenience without injections

Designed for improved GI tolerability
Potential for better tolerability and adherence

Two-step titration
Faster path to maintenance dose

Built to improve persistence
Helping more patients stay on therapy

Chronic therapy in mind
Designed for sustainable weight loss management

THE PROJECTED IMPACT

Weight Loss Management
Better tolerability will lead to continuous weight management

Better Patient Experience
Simpler regimen and fewer GI side effects

Higher Persistence
More patients stay on therapy and reach therapeutic benefit

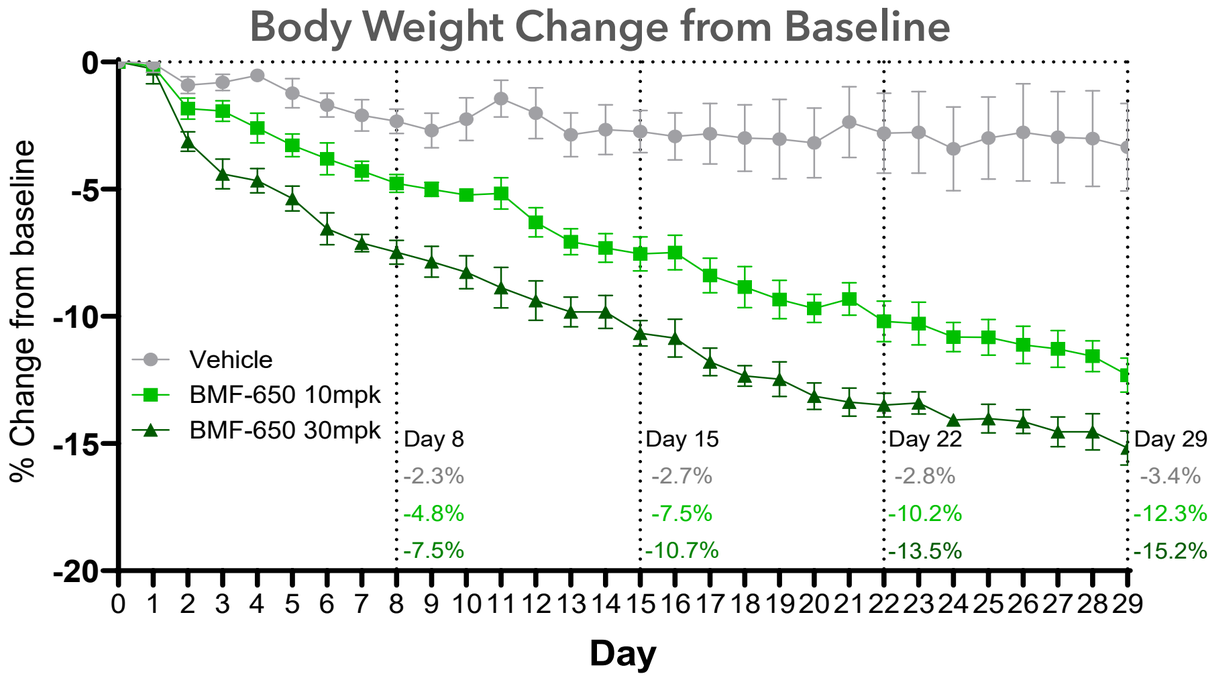
Long-term Health Benefits
Sustained weight management supports cardiometabolic health

BMF-650 is designed to be the oral GLP-1 RA patients can start, stay on, and have long term benefits from

1.Real-world evidence across multiple GLP-RA studies (e.g. N Engl J Med 2021.385.896-907. JAMA Netw Open 2022.5e223433; Diabetes Obe Metab. 2023.25.1254-1264

BMF-650 demonstrated robust, dose-dependent weight loss in obese monkeys with low rates of emesis

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



Wang, et al. Obesity 2025: 33 Sup 2 Poster #085

Week	Emesis (Vomiting)
1	18.2%*
2	7.0%
3	5.6%
4	1.4%

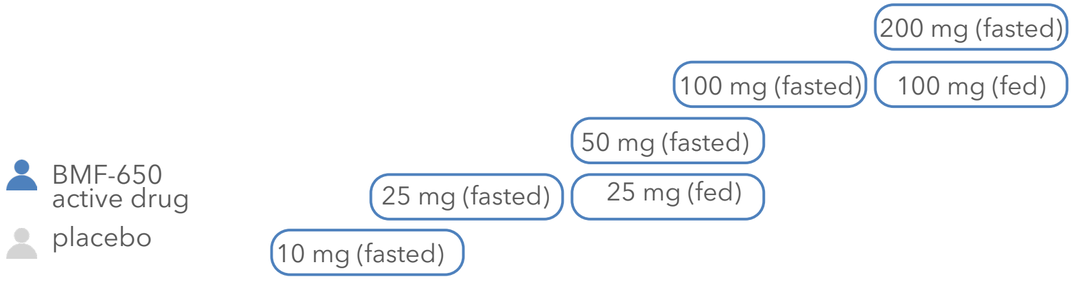
*One monkey (#3) in the low-dose cohort accounted for 12 of the 23 total events. % calculated for 70 dosing events/week

With 280 active dosing occurrences, there were a total of 23 emesis events (8.2%) balanced across both dosing cohorts.

GLP-131: Phase 1 study design + dose cohort 5

Randomized, double-blind, placebo-controlled study, evaluating safety, tolerability, and weight reduction with BMF-650 in overweight or obese participants

	Single Ascending Dose (SAD) Phase 1a	Multiple Ascending Dose (MAD) Phase 1b
Primary & Secondary Objectives	Safety and tolerability, PK, PD and food effect	Safety and tolerability, and efficacy (weight-loss)
Eligibility	Healthy overweight or obese participants (BMI 25.0–40.0 kg/m²)	Healthy obese participants (BMI 30.0–45.0 kg/m²) **Cohort 5 conducted under confinement to evaluate one-step titration regimen**
Design	N=40 5 cohorts x 	N=50 5 cohorts x 



COHORT	Wk 1 → Wk 2 → Wk 3 → Wk 4
5	**200 mg → 400 mg → 400 mg → 400 mg**
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

Baseline demographics & characteristics

Doses for Weeks 1–4	Active BMF-650						
	Cohort 1 10/25/50/100 mg (N=8)	Cohort 2 50/100/200/200 mg (N=8)	Cohort 3 75/150/300/300 mg (N=8)	Cohort 4 75/200/400/400 mg (N=8)	Cohort 5 200/400/400/400 mg (N=8)	MAD BMF-650 Total (N=40)	Pooled Placebo (N=10)
Sex, female, n (%)	**6 (75)**	**6 (75)**	**3 (38)**	**4 (50)**	**5 (63)**	**24 (60)**	**5 (50)**
Age, years, mean	**49.5**	**45.6**	**49.9**	**42.3**	**41.6**	**45.8**	**37.4**
Weight, kg, mean	**97.0**	**109.1**	**96.1**	**97.6**	**102.1**	**100.4**	**107.2**
BMI, kg/m^2, mean	**34.8**	**36.2**	**31.9**	**34.4**	**34.0**	**34.3**	**35.3**

Analysis of weekly weight reduction

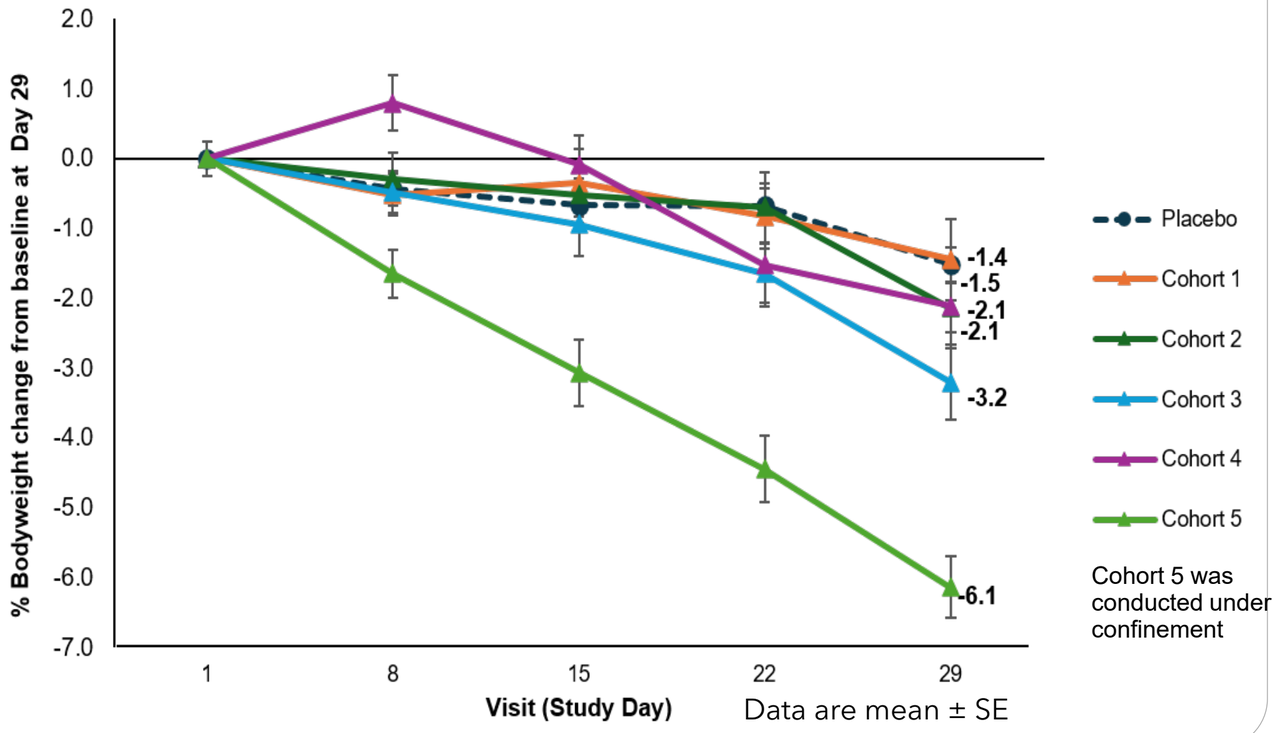
	Pooled PLACEBO n=8	Cohort 1 10 → 25 → 50 → 100	Cohort 2 50 → 100 → 200 → 200	Cohort 3 75 → 150 → 300 → 300	Cohort 4 75 → 200 → 400 → 400	Cohort 5 in-patient confinement: 200 → 400 → 400 → 400
Week 1	+0.33 kg	10 mg -0.47 kg	50 mg -0.41 kg	75 mg -0.45 kg	75 mg +0.76 kg	200 mg -1.72 kg
Week 2	+0.06 kg	25 mg +0.12 kg	100 mg -0.20 kg	150 mg -0.39 kg	200 mg -0.83 kg	400 mg -1.38 kg
Week 3	+0.12 kg	50 mg -0.48 kg	200 mg -0.23 kg	300 mg -0.70 kg	400 mg -1.37 kg	400 mg -1.39 kg
Week 4	-0.82 kg	100 mg -0.63 kg	200 mg -1.57 kg	300 mg -1.47 kg	400 mg -0.63 kg	400 mg -1.71 kg
TOTAL Weight Los	-0.10 kg	-1.46 kg	-2.41 kg	-3.01 kg	-2.07 kg	-6.20 kg

- Cohort 5 used confinement to monitor dosing adherence, with 200 mg once daily for one week, then 400 mg once daily for three weeks.
- Mean weight loss with BMF-650 was **1.55 kg/week**, and similar weight loss with placebo (n=2).
- All participants were provided 2,100 kcal/day. The 2 placebo participants consumed approximately 33% of the presented food, complicating interpretation of weight loss under confinement.

Clinical Efficacy

Weight reduction at Day 29 with participants oral dosing of BMF-650

	Bodyweight % mean change from baseline at Day 29	
Placebo (n=10)	**-1.5%**	
Cohort 1 (n=8) (10/25/50/100 mg)	**-1.4%**	
Cohort 2 (n=8) (50/100/200/200 mg)	**-2.1%**	
Cohort 3 (n=8) (75/150/300/300 mg)	**-3.2%**	
Cohort 4 (n=8) (75/200/400/400 mg)	**-2.1%**	
Cohort 5 (n=8) (200/400/400/400 mg)	**-6.1%**	Exploratory P-value P<0.01



Cohort 5 achieved 6.1% mean weight loss at Day 29
- 75% (6 of 8) participants on BMF-650 in Cohort 5 achieved >5% mean weight loss
- 100% (8 of 8) participants on BMF-650 in Cohort 5 achieved >4% mean weight loss

Primary Endpoint: Overall Treatment-Emergent Adverse Events (TEAEs)

Doses for Weeks 1–4 Participants, n(%)	Active BMF-650						Pooled Placebo (N=10)
	Cohort 1 10/25/50/100 mg (N=8)	**Cohort 2** 50/100/200/200 mg (N=8)	**Cohort 3** 75/150/300/300 mg (N=8)	**Cohort 4** 75/200/400/400 mg (N=8)	**Cohort 5** 200/400/400/400 mg (N=8)	MAD BMF-650 Total (N=40)	
Any TEAE	**6 (75.0)**	**4 (50.0)**	**5 (62.5)**	**6 (75.0)**	**7 (87.5)**	**28 (70.0)**	**4 (40.0)**
Mild (Grade 1)	**3 (37.5)**	**3 (37.5)**	**4 (50.0)**	**5 (62.5)**	**5 (62.5)**	**20 (50.0)**	**4 (40.0)**
Moderate (Grade 2)	**3 (37.5)**	**1 (12.5)**	**1 (12.5)**	**1 (12.5)**	**2 (25.0)**	**8 (20.0)**	**0**

0	**Severe TEAE (Grade 3)**	**0**	**Serious TEAE**	**0**	**TEAE-leading to dose modification**	**0**	**TEAE-leading to study discontinuation**

All TEAEs were mild or moderate and there were no dose modifications or discontinuations related to BMF-650. TEAEs were not associated with dose escalation, dosing cohort, or time at a given dose.

Select Gastrointestinal TEAEs

Doses for Weeks 1–4 **Participants, n (%)**	Active BMF-650						
	Cohort 1 10/25/50/100 mg (N=8)	Cohort 2 50/100/200/200 mg (N=8)	Cohort 3 75/150/300/300 mg (N=8)	Cohort 4 75/200/400/400 mg (N=8)	Cohort 5 200/400/400/400 mg (N=8)	MAD BMF-650 Total (N=40)	Pooled Placebo (N=10)
Nausea	**0**	**3 (37.5)**	**1 (12.5)**	**2 (25.0)**	**1 (12.5)**	**7 (17.5)**	**0**
Constipation	**0**	**1 (12.5)**	**1 (12.5)**	**0**	**2 (25.0)**	**4 (10.0)**	**1 (10.0)**
Vomiting	**1 (12.5)**	**0**	**1 (12.5)**	**0**	**1 (12.5)**	**3 (7.5)**	**0**
Diarrhea	**0**	**1 (12.5)**	**1 (12.5)**	**0**	**0**	**2 (5.0)**	**1 (10.0)**

The select GI-related TEAEs were all Grade 1 AEs in the highest dose level cohort.

BMF-650 | Phase 1b MAD

Phase 1 findings provide support for the BMF-650 design hypothesis

Rapid one-step titration to 400 mg is well tolerated

BMF-650 reached the target dose of 400 mg QD via a single titration step design post week 1, supporting a potentially simple and practical dosing paradigm

Sustained and meaningful weight reduction observed at higher dose regimens

Cohort 5 demonstrated mean weight loss at 400 mg across consecutive weeks (−1.4, −1.4 and −1.7 kg/week), reaching 6.1% at week 4, with no evidence of attenuation over the observed treatment period

Encouraging GI tolerability profile

No serious TEAEs, no TEAE-related dose modifications and no TEAE-related study discontinuations occurred across active MAD cohorts, supporting longer-duration evaluation

BMF-650 | Phase 1b MAD

Next Steps:
Advancing BMF-650 development while optimizing the clinical profile

Clinical Findings	• Highest dose achieved with only a single titration step while maintaining favorable tolerability • Weight loss as expected in line with other GLP-1 agents • Overall, generally well tolerated with no exposure-related safety signals
BMF-650 Next Steps	• Advance Phase 2 development planning incl. a “no-titration” regimen at 400 mg QD • Initiate longer-term toxicology studies to support Phase 2 dosing

THANK YOU

(NASDAQ: BMEA)

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

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