

**UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549**

FORM 8-K

**CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934**

Date of Report (Date of earliest event reported): February 25, 2026

Biomea Fusion, Inc.
(Exact name of Registrant as Specified in Its Charter)

Delaware
(State or Other Jurisdiction
of Incorporation)

001-40335
(Commission
File Number)

82-2520134
(IRS Employer
Identification No.)

1599 Industrial Road
San Carlos, CA
(Address of Principal Executive Offices)

94070
(Zip Code)

Registrant's Telephone Number, Including Area Code: (650) 980-9099

Not Applicable
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

- Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
- Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
- Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
- Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, \$0.0001 par value	BMEA	The Nasdaq Global Select Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act.

Item 7.01 Regulation FD Disclosure.

On February 25, 2026, Biomea Fusion, Inc. (the “Company”) updated its corporate presentation to be used from time to time with investors, analysts and other third parties (the “Corporate Presentation”). The Company posted a copy of the presentation to the “Investors & Media” section of the Company’s website at www.biomeafusion.com. A copy of the Corporate Presentation is furnished herewith as Exhibit 99.1 to this Current Report on Form 8-K.

The information contained in Item 7.01 of this Current Report on Form 8-K, including Exhibit 99.1 attached hereto, is being furnished and shall not be deemed to be “filed” for the purposes of Section 18 of the Securities Exchange Act of 1934 (the “Exchange Act”), or otherwise subject to the liabilities of that section and shall not be incorporated by reference in any filing under the Securities Act of 1933 or the Exchange Act, except as shall be expressly set forth by specific reference in such filing.

Forward-Looking Statements

This Current Report on Form 8-K and certain materials furnished within may include statements which are not historical facts and are considered forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended (the “Securities Act”), and Section 21E of the Securities Exchange Act of 1934, as amended (the “Exchange Act”). These statements may be identified by words such as “aims,” “anticipates,” “believes,” “could,” “estimates,” “expects,” “forecasts,” “goal,” “intends,” “may,” “plans,” “possible,” “potential,” “seeks,” “will” and variations of these words or similar expressions that are intended to identify forward-looking statements. Any such statements in this press release that are not statements of historical fact could be deemed forward-looking, including any projections of financial information or profitability, including the Company’s expected cash runway, the initiation, timing and results of pending or future preclinical studies and clinical trials, the actual or potential actions of the U.S. Food and Drug Administration (FDA), the status and timing of ongoing research, development and corporate partnering activities, any statements about historical results that may suggest trends for the Company’s business; any statements of the plans, strategies, and objectives of management for future operations and any statements of expectation or belief regarding future events, potential markets or market size, or technology developments. The Company intends these forward-looking statements to be covered by the safe harbor provisions for forward-looking statements contained in Section 27A of the Securities Act and Section 21E of the Exchange Act and is making this statement for purposes of complying with those safe harbor provisions.

Any forward-looking statements made in this Current Report on Form 8-K or included in certain of the materials furnished herewith are based on the Company’s current expectations, estimates and projections only as of the date of this Current Report on Form 8-K and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward-looking statements, including the risk that preliminary or interim results of preclinical studies or clinical trials may not be predictive of future or final results in connection with ongoing or future clinical trials and the risk that we may encounter delays in preclinical or clinical development, patient enrollment and in the initiation, conduct and completion the Company’s ongoing and planned clinical trials and other research and development activities. These risks concerning the Company’s business and operations are described in additional detail in its periodic filings with the U.S. Securities and Exchange Commission (the “SEC”), including its most recent periodic report filed with the SEC and subsequent filings thereafter. The Company explicitly disclaims any obligation to update any forward-looking statements except to the extent required by law.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	Description
99.1	Biomea Fusion Corporate Presentation
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned thereunto duly authorized.

BIOMEA FUSION, INC.

Date: February 25, 2026

By: /s/ Michael J.M. Hitchcock

Michael J.M. Hitchcock
Interim Chief Executive Officer, Director
(Principal Executive Officer)

1st QUARTER 2026

Biomea Fusion Corporate Presentation



Legal disclaimer & forward-looking statements

Certain statements in this presentation and the accompanying oral commentary are forward-looking statements. These statements relate to future events or the future business and financial performance of Biomea Fusion, Inc. (the "Company") and involve known and unknown risks, uncertainties, and other factors that may cause the actual results, levels of activity, performance or achievements of the Company or its industry to be materially different from those expressed or implied by any forward-looking statements. In some cases, forward-looking statements can be identified by terminology such as "may," "will," "could," "would," "should," "expect," "plan," "anticipate," "intend," "believe," "estimate," "predict," "potential" or other comparable terminology. All statements other than statements of historical fact could be deemed forward-looking, including any projections of financial information or profitability, including our expected cash runway, the initiation, timing and results of pending or future preclinical studies and clinical trials, the actual or potential actions of the U.S. Food and Drug Administration (FDA), the status and timing of ongoing research, development and corporate partnering activities, any statements about historical results that may suggest trends for the Company's business; any statements of the plans, strategies, and objectives of management for future operations and any statements of expectation or belief regarding future events, potential markets or market size, or technology developments. The Company has based these forward-looking statements on its current expectations, assumptions, estimates, and projections. While the Company believes these expectations, assumptions, estimates and projections are reasonable, such forward-looking statements are only predictions and involve known and unknown risks and uncertainties, many of which are beyond the Company's control. For a discussion of these and other risks and uncertainties, and other important factors, any of which could cause our actual results to differ from those contained in the forward-looking statements, see the section entitled "Risk Factors" in our most recent annual report on Form 10-K filed with the Securities and Exchange Commission (the SEC), as well as discussions of potential risks, uncertainties, and other important factors in our other subsequent filings with the SEC. The forward-looking statements in this presentation are made only as of the date hereof. Except as required by law, the Company assumes no obligation and does not intend to update these forward-looking statements or to conform these statements to actual results or to changes in the Company's expectations. This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions, and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk.

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

Designed 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.

Biomea Fusion pipeline - targeting diabetes & obesity



Biomea Fusion retains full worldwide rights across all programs

Program	Indications	Pre-clinical	Phase I	Phase II	Phase III	Key Catalysts
ICOVAMENIB	Type 2 diabetes Patients with Insulin-deficiency		COVALENT-211 (study initiated)			Phase II 26-week data (primary endpoint) anticipated 4Q 2026
ICOVAMENIB	Type 2 diabetes Patients not controlled on GLP-1-based therapies		COVALENT-212 (study initiated)			Phase II 26-week data (primary endpoint) anticipated 4Q 2026
ICOVAMENIB	Type 2 diabetes Patients - all comers Type 1 diabetes Patients - all comers		COVALENT-111 (study completed)	COVALENT-112 (study completed)		52-week follow-up data presented 4Q 2025 52-week follow-up data expected in Q2 2026
BMF-650	Obesity		GLP-131 (study enrolling)			Phase I weight reduction data expected Q2 2026

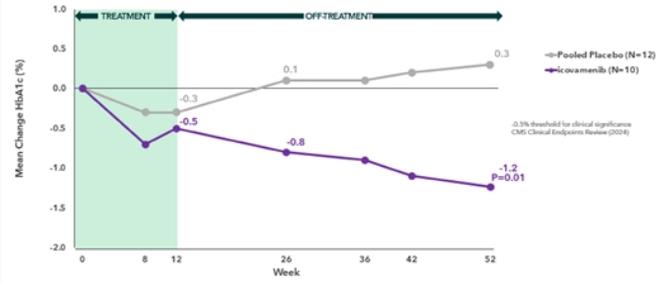
Potential first-in-class menin inhibitor aimed to restore functional beta-cells

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

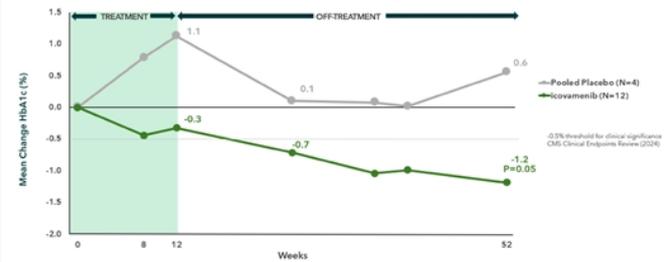
Icovamenib is developed to:

- + 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

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 ⁵

Two transformative phase II trials ongoing

Near term readouts expected in 4Q 2026

ICOVAMENIB

Phase IIa completed and proposed mechanism supported

- Persistent 52-week HbA1c reduction after 12 weeks of treatment
- Increased C-peptide OFF DRUG in both responding patient populations validating mechanism of action of restored beta-cell mass
- Go-forward regimen generally well-tolerated

Two Phase II trials underway

COVALENT-211

Insulin-deficient T2D failing standard of care

- Enrollment ongoing
- 26-week topline data expected 4Q 2026

COVALENT-212

T2D inadequately controlled on GLP-1 therapy

- Enrollment ongoing
- 26-week topline data expected 4Q 2026

Diabetes patients are poorly controlled with over 7M US patients currently needing insulin as a last resort



Icovamenib targets beta-cell restoration and may delay or prevent onset of end-stage disease



80%

of people with diabetes will die from the disease¹

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 diabetes²

Diabetes today remains poorly controlled in 50% of patients treated with standard of care agents³. 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. Ahlqvist, Lancet Diabetes Endocrinol., 2018

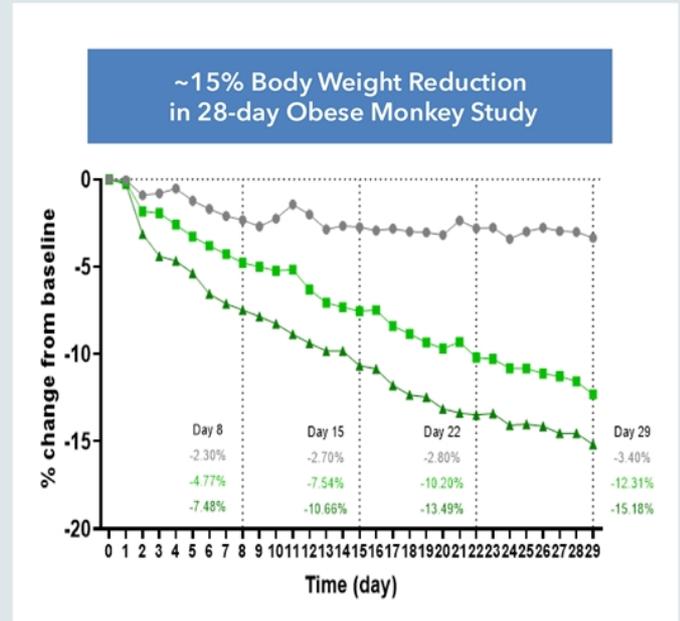
Oral GLP-1 RA developed for improved patient friendly tolerability

Aims to serve a significant unmet need with millions of obese Americans dropping off the available GLP-1 RAs agents within the first year¹

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

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



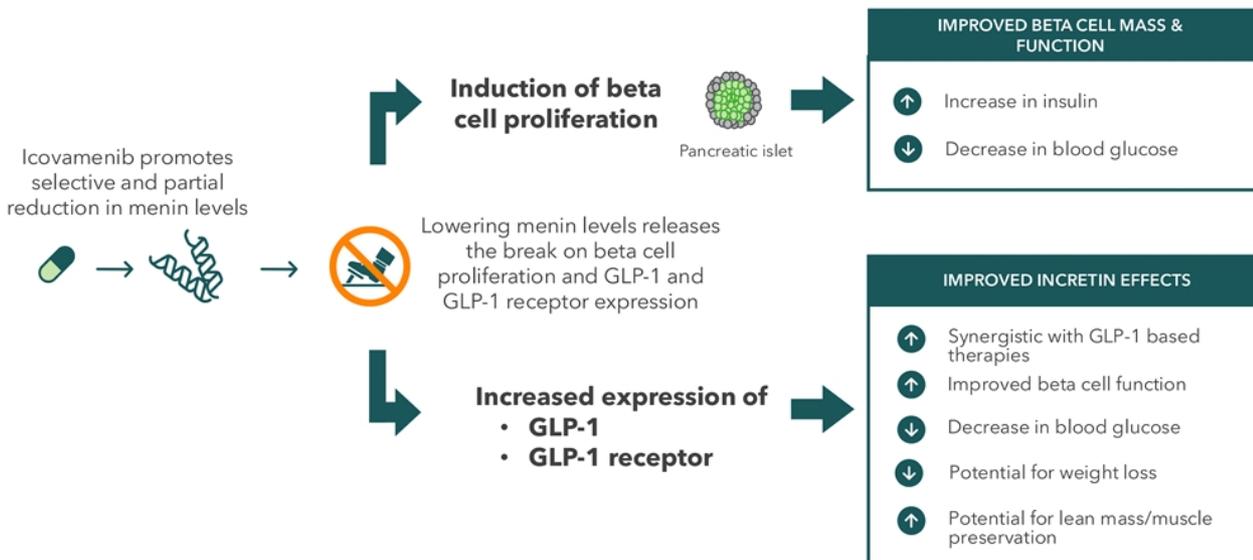
ICOVAMENIB

Biology, mechanism of action & preclinical findings

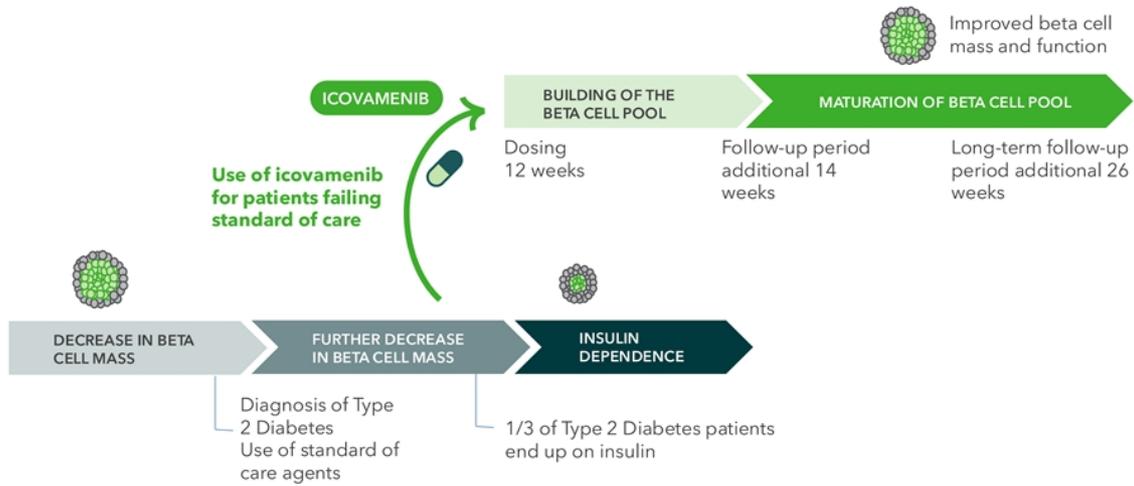
 **biomea**
FUSION™



Icovamenib's mechanism of action

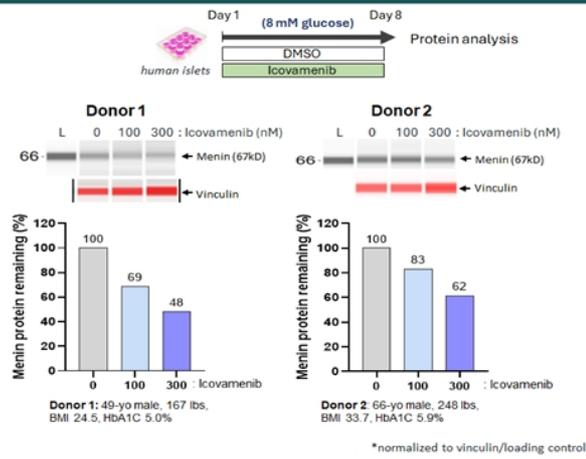


Icovamenib increased beta cell quantity, function & GLP-1 receptor expression following a short treatment period

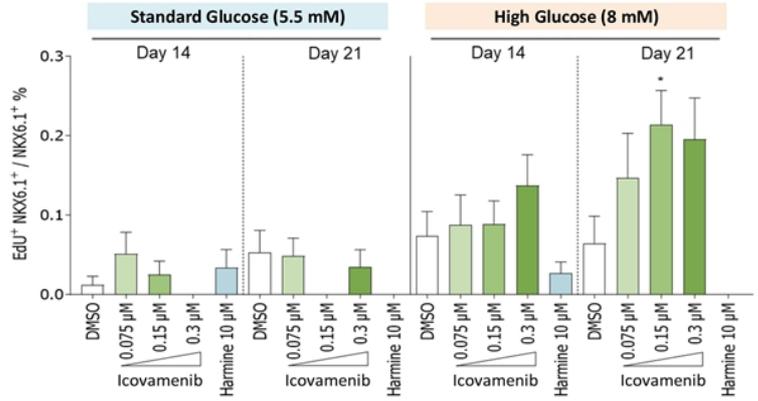


Icovamenib downregulated menin protein levels & promoted beta cell proliferation in ex vivo human islet cultures

MENIN LEVELS DOWNREGULATED



ICOVAMENIB CONDITIONALLY PROMOTED BETA CELL PROLIFERATION ONLY UNDER HYPERGLYCEMIC CONDITIONS

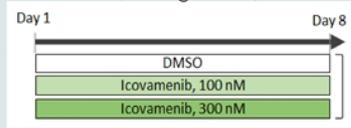


Icovamenib enhanced GLP-1 receptor & insulin expression and demonstrated potential synergy in combination with semaglutide ex vivo

Cadaver derived human islets

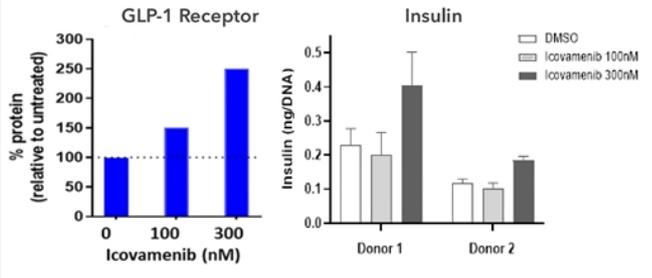


Culture 7 days under glucotox conditions (8mM glucose)

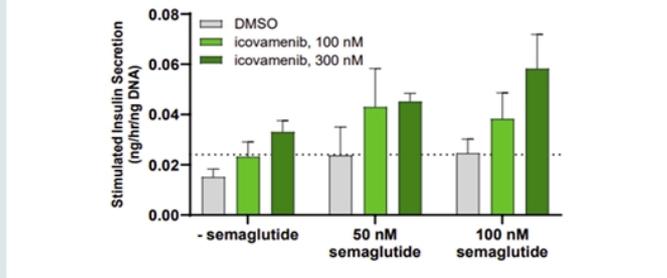


- Gene expression & Protein analysis
- Glucose Stimulated Insulin Secretion +/- Semaglutide (200nM)

ICOVAMENIB INCREASED GLP-1 RECEPTOR AND INSULIN EXPRESSION

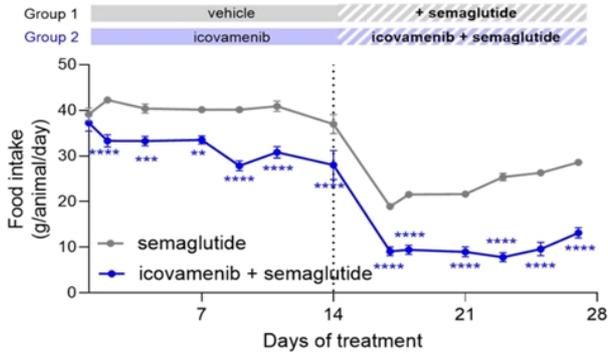


ICOVAMENIB IN COMBINATION WITH SEMAGLUTIDE INCREASED GLUCOSE-STIMULATED INSULIN SECRETION

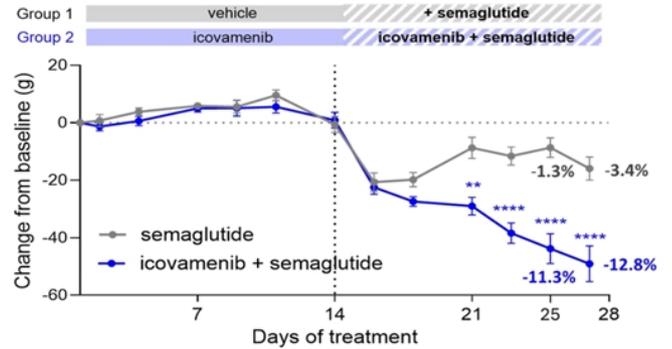


Combination treatment of icovamenib & low-dose semaglutide reduced food intake & body weight

APPETITE SUPPRESSION



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

Potential first-in-class oral menin inhibitor clinical study results

 **biomea**
FUSION

Baseline demographics & characteristics

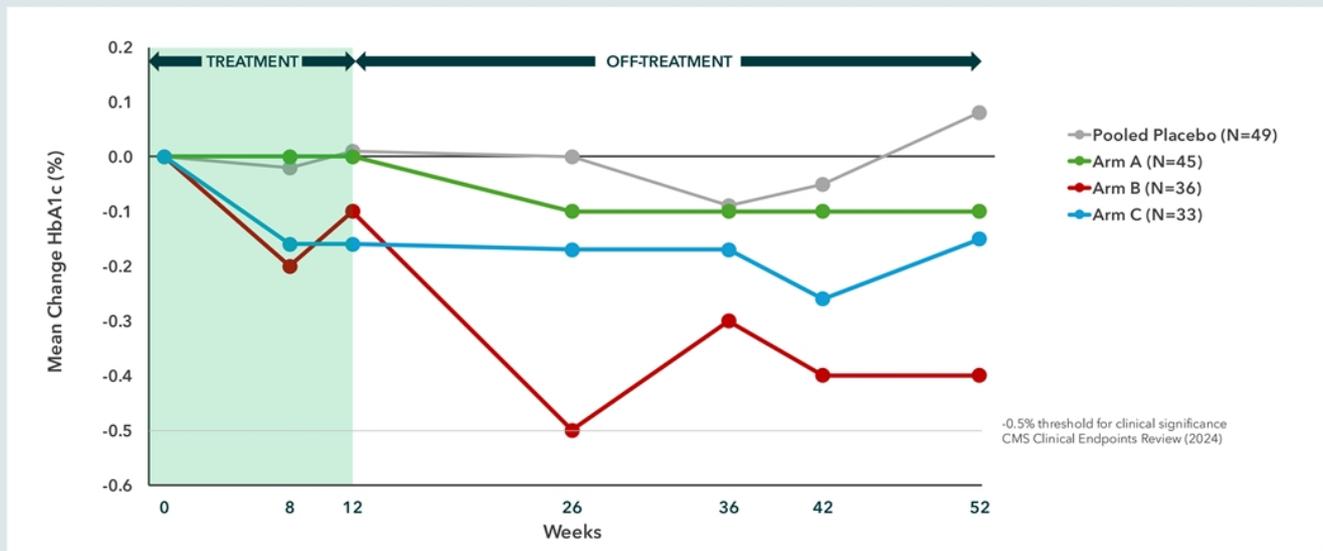
Per Protocol Population* on 1 or More Antihyperglycemic Agents at Baseline (N=163)

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.

Change in HbA1c from baseline through week 52 - all subtypes

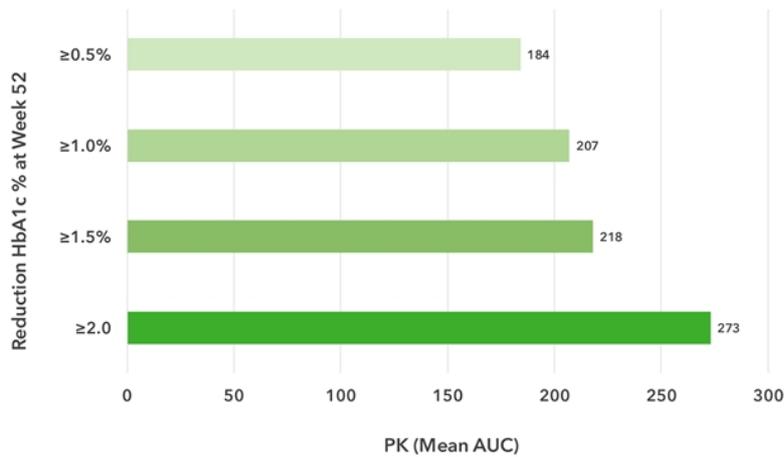
Across treatment durations (Arm A = 8 weeks 100 mg, Arm B = 12 weeks 100 mg, Arm C = 8 weeks 100 mg 4 weeks at 200 mg) per protocol participants taking one or more antihyperglycemic medications at baseline



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.

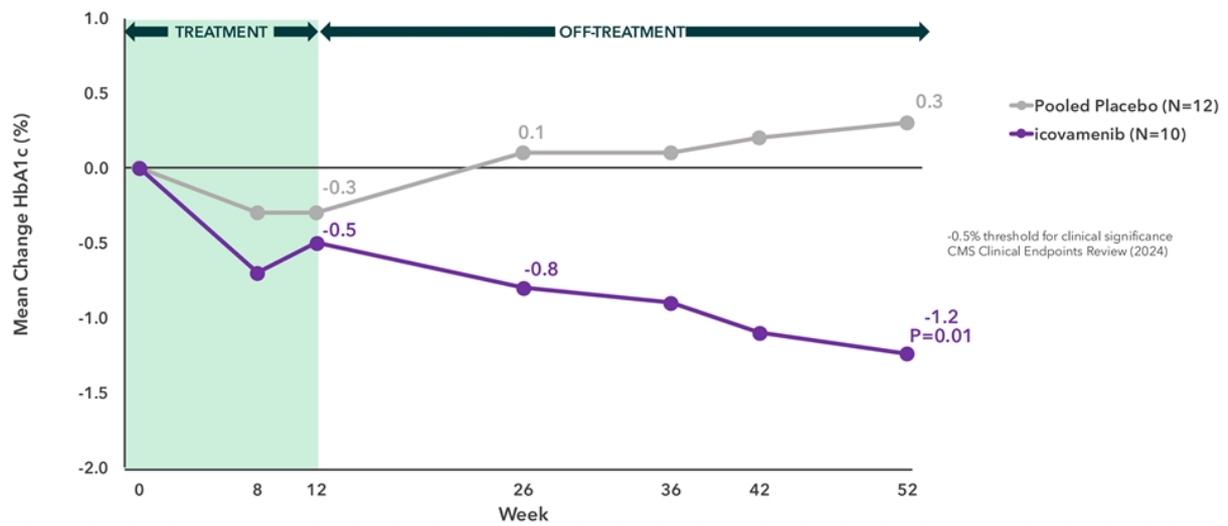
Higher HbA1c reduction was associated with higher icovamenib exposure

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



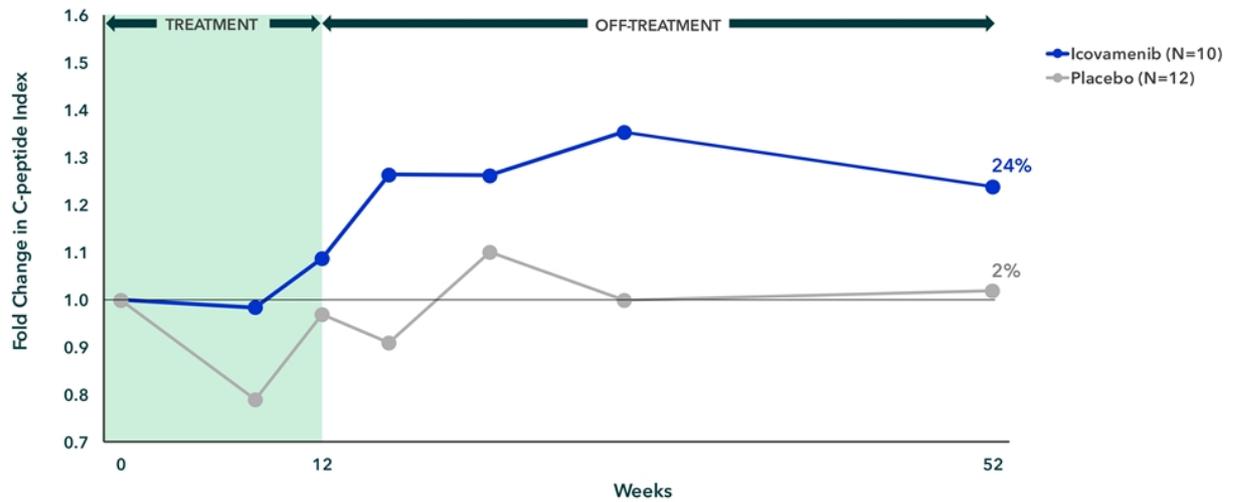
- Dosing timing relative to food will impact icovamenib's pharmacokinetics (PK)
- In a 'Food Effect Study' icovamenib achieved optimal PK exposure when administered within 30 minutes after a meal
- These findings now inform the dosing strategy for the ongoing Phase II studies

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



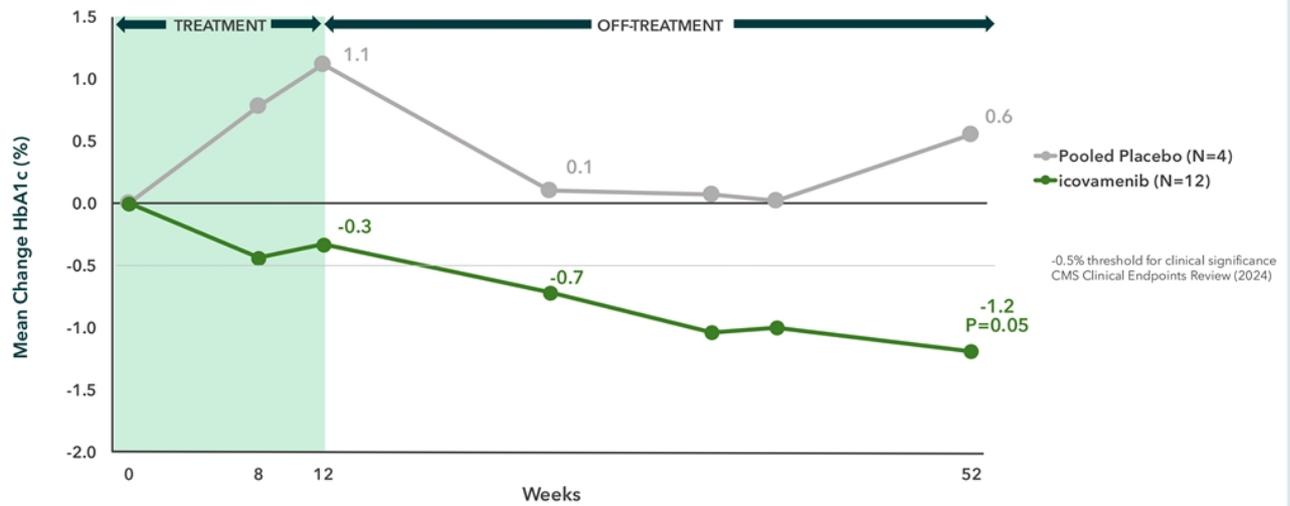
Arm A was excluded from this analysis because it included only 8 weeks of dosing which the company is not planning to pursue.

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

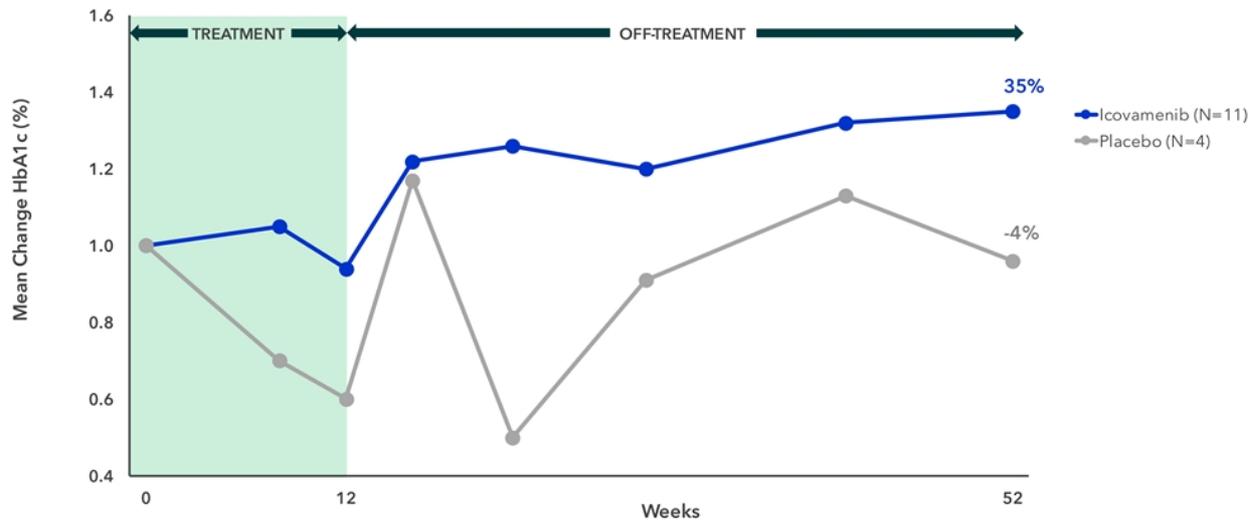


Data censored at onset of rescue medication, defined as any modification in antihyperglycemic therapy

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



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

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 $\geq 5\%$ 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.

Key findings through week 52 after a short treatment course



Menin Inhibition Potentially Leads to Increased Clinical Benefit

Higher exposure aligned with deeper HbA1c reductions. Data also supports potential for exposure improvements



Durable Clinical Activity in Insulin Deficient T2D

1.2% mean HbA1c reduction ($p=0.01$) maintained through Week 52 after 12 weeks of dosing



Durable Clinical Activity in T2D Not Controlled on GLP-1-based Therapies

1.3% mean HbA1c reduction ($p=0.05$) maintained through Week 52 after 12 weeks of dosing



Favorable Safety Profile

Generally well tolerated, no treatment-related serious adverse events

NEXT STEPS:

- COVALENT-211 PHASE IIB IN SEVERE INSULIN DEFICIENT T2D, FIRST PATIENT ENROLLMENT IN 1Q 2026.
- COVALENT-212 PHASE IIT2D PATIENTS NOT CONTROLLED ON GLP-1 BASED THERAPIES, FIRST PATIENT ENROLLED IN 1Q 2026.

ICOVAMENIB

Potential first-in-class oral menin inhibitor

Ongoing Phase II Studies



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

ICOVAMENIB

Phase IIa key derisking-insights

- Optimal dose selected
- 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

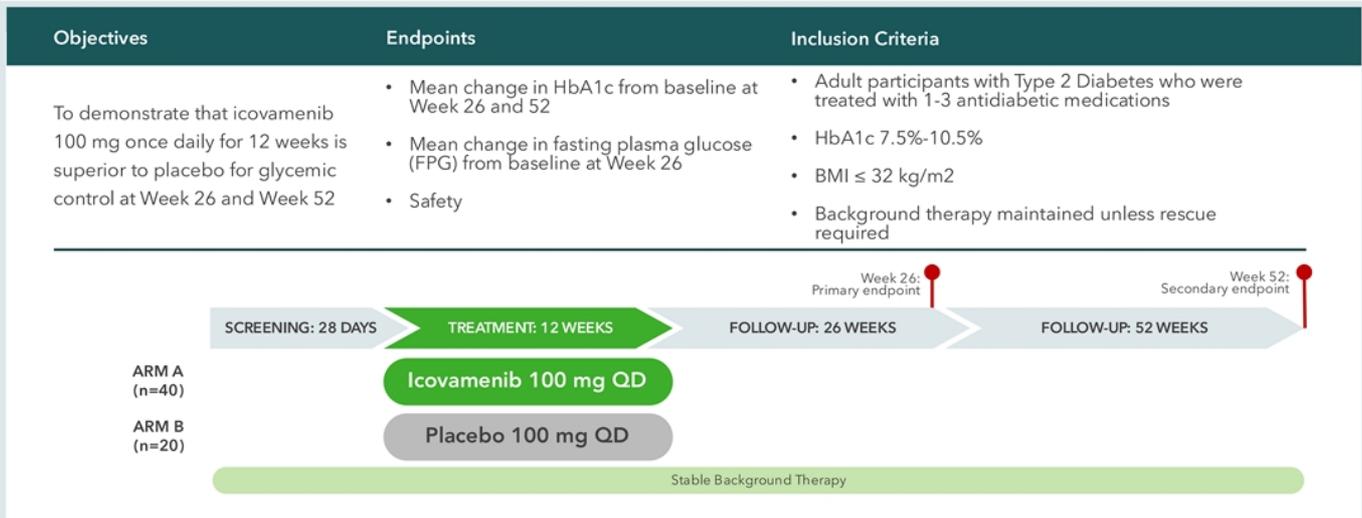
- Adult participants with Type 2 Diabetes who were treated with 1-3 antidiabetic medications
- HbA1c 7.5%-10.5%
- BMI \leq 32 kg/m²
- Background therapy maintained unless rescue required

COVALENT-212

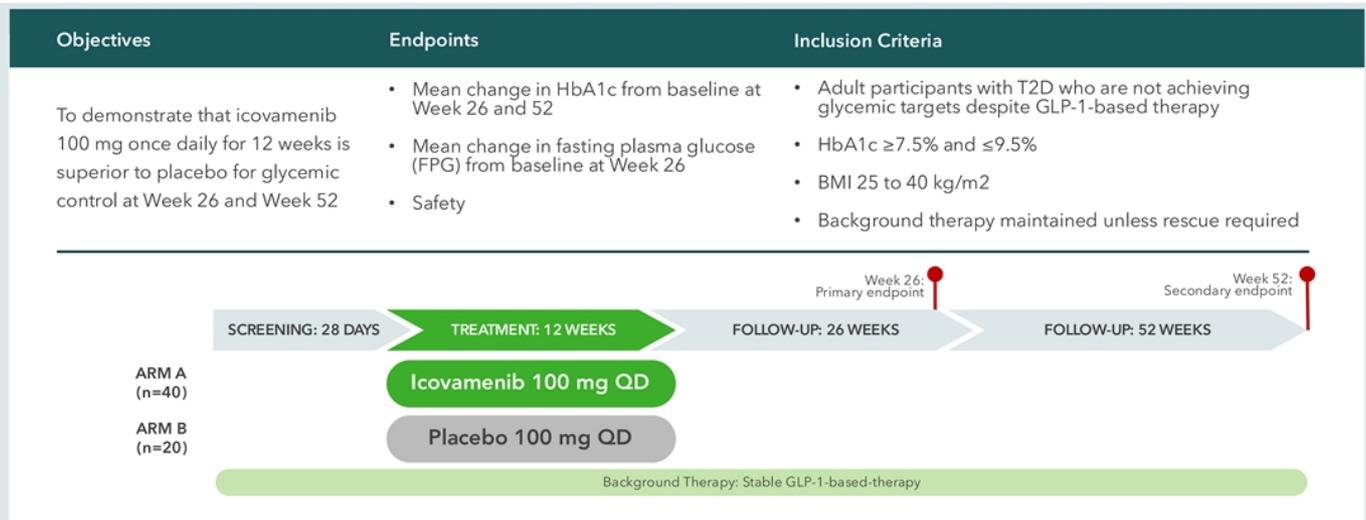
Phase II trial in Type 2 Diabetes Patients failing standard of care while on a GLP-1 RA

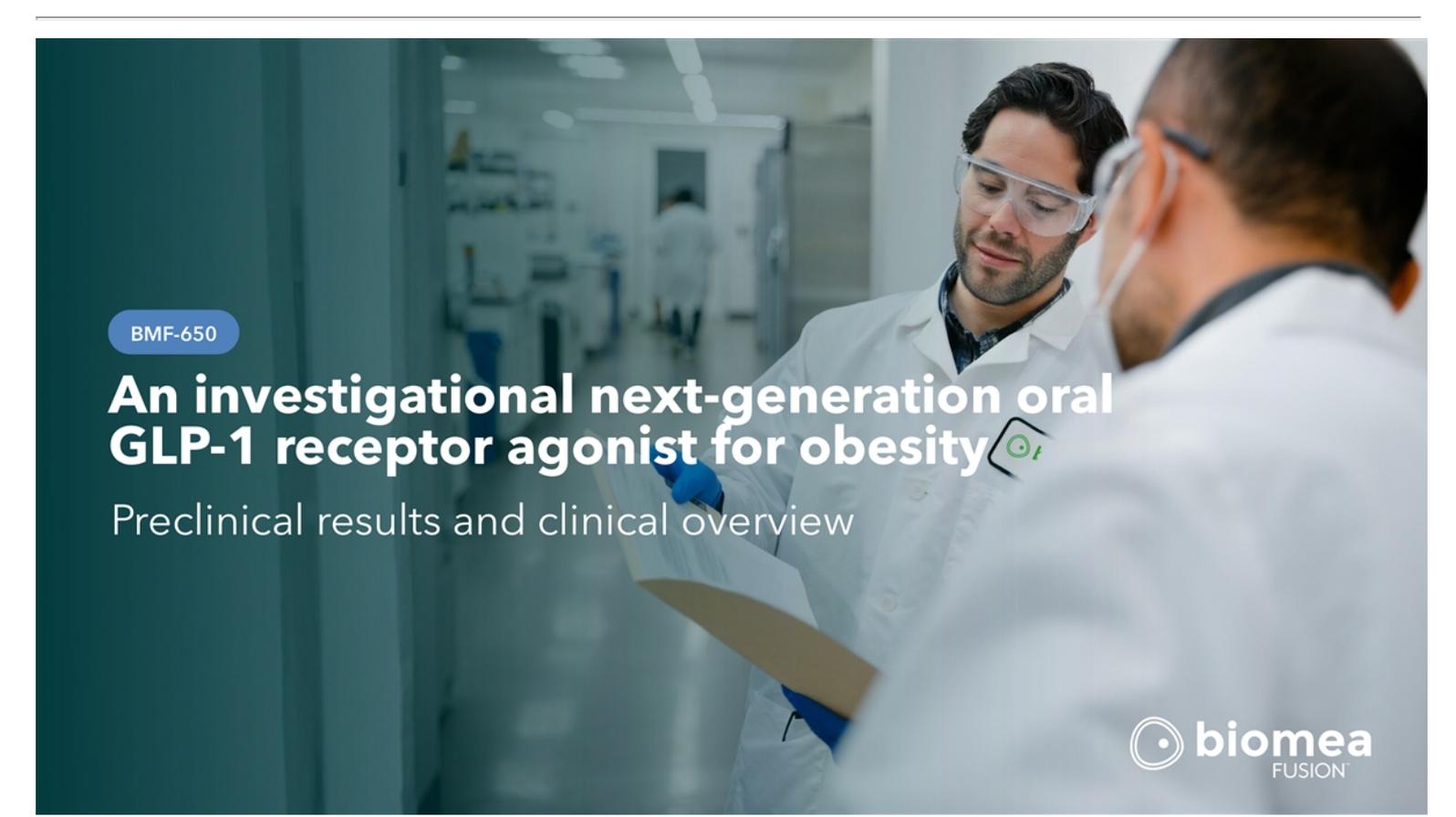
- Adult participants with T2D who are not achieving glycemic targets despite GLP-1-based therapy
- HbA1c \geq 7.5% and \leq 9.5%
- BMI 25 to 40 kg/m²
- Background therapy maintained unless rescue required

A Phase II trial of icovamenib in T2D insulin deficient participants who are not achieving glycemic targets



Phase II trial of icovamenib in participants with T2D who are not achieving glycemic targets while using GLP-1-based therapy





BMF-650

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

Preclinical results and clinical overview

 **biomea**
FUSION

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

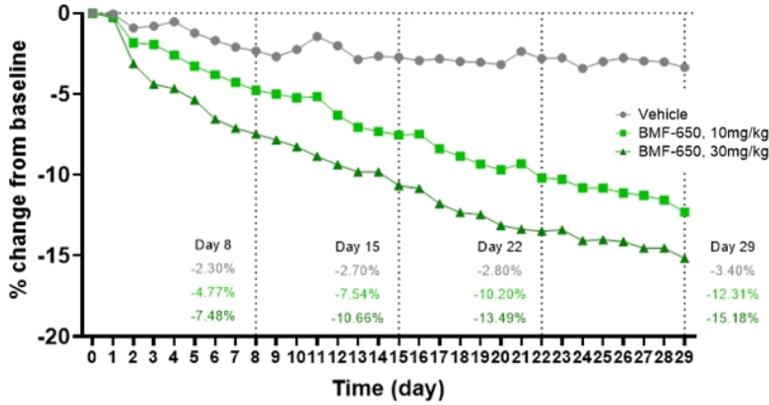
- U.S. patent allowance received December 2025 covering BMF-650 composition.
- U.S. and PCT applications published and proceeding through examination.



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

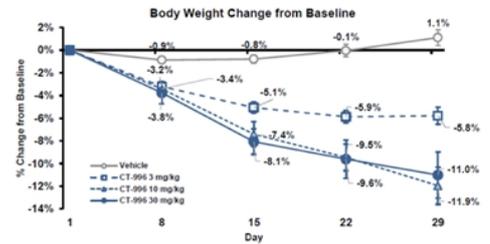


Figure 6. Effects of CT-996 on body weight in obese cynomolgus monkeys following once-daily oral administration. Weekly body weight percent change is represented as mean (± SE) from baseline. N = 6/group.

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.

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/m ²)	Healthy overweight or obese patients (BMI 30.0–45.0 kg/m ²)
Design	<p>N=40 5 cohorts x </p> <p>Legend: BMF-650 active drug, placebo</p>	<p>N=40 4 cohorts x </p> <p>COHORT</p> <p>7 DAYS → 7 DAYS → 7 DAYS → 21 DAYS</p> <p>4: 75 mg → 200 mg → 400 mg → 400 mg</p> <p>3: 75 mg → 150 mg → 300 mg → 300 mg</p> <p>2: 50 mg → 100 mg → 200 mg → 200 mg</p> <p>1: 10 mg → 25 mg → 50 mg → 100 mg</p> <p>Body weight at Baseline versus Day 28 and Day 42 on treatment</p>

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](#)

THANK YOU (NASDAQ: BMEA)

For questions or inquiries, please reach out to
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www.biomeafusion.com



Slowing the clock on diabetes

Turning a healthcare crisis into a huge opportunity



The problem:

Diabetes is a global healthcare crisis, with over 800 million people affected worldwide. The disease is expanding at an estimated ~4% annually. In the United States, one in every four healthcare dollars is spent on people living with diabetes, totaling over \$400 billion per year. More than 100 million Americans have prediabetes; approximately 40% will progress to diabetes, and ~60% are diagnosed only after reaching late-stage disease.

The economic impact:

Late-stage diabetes drives over 50% of healthcare related costs due to complications such as kidney failure, cardiovascular disease, amputations and visual loss. With late-stage diabetes the healthcare system is burdened by additional \$15,000 - \$20,000 per patient. Assuming 10m US diabetes patients are in late stage, delaying the progression could save the system \$150-200billion.

Our breakthrough:

We are developing a novel mechanism of action developed to restore beta-cell function and slow the progression of hyperglycemia. By rebuilding a functional beta-cell pool, the body may regain the ability to regulate its own insulin production. The true burden of diabetes is not early disease, but late-stage progression with irreversible organ damage. Slowing that clock by even a few years could generate societal returns exceeding the annual economic output of the entire pharmaceutical sector. Icovamenib would delay the need for unwieldy and often unreliable insulin therapy for years!

Our vision:

Keeping diabetes patients durably controlled, preventing progression to late-stage disease and avoiding the devastating complications that drive cost, morbidity, and mortality.

Key opinion leaders highlight icovamenib's potential to redefine diabetes care



"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

Key opinion leaders highlight icovamenib's potential to redefine diabetes care



"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