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): January 14, 2026

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

Delaware	001-40335	82-2520134
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(IRS Employer Identification No.)

1599 Industrial Road San Carlos, CA	94070
(Address of Principal Executive Offices)	(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 January 14, 2026, Biomea Fusion, Inc. (the "Company") presented at the 44th Annual J.P. Morgan Healthcare Conference, which took place from January 12-15, 2026 in San Francisco, California. The Company posted a copy of the presentation (the "Conference Presentation") to the "Investors & Media" section of the Company's website at *www.biomeafusion.com*. A copy of the Conference 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.

Item 8.01. Other Events.

On January 14, 2026, the Company updated its corporate presentation to be used from time to time with investors, analysts and other third parties (the "Corporate Presentation").

A copy of the Company's Corporate Presentation is attached to this Current Report on Form 8-K as Exhibit 99.2 and is incorporated herein by reference.

Forward-Looking Statements

Statements made or incorporated by reference in this Current Report on Form 8-K 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, including statements regarding the clinical and therapeutic potential of the Company's product candidates and development programs, their mechanism of action, and their potential relative to approved products marketed by third parties; the potential benefits to future trial design and program development of subtyping diabetes patients and their potential to be used in combination with approved products marketed by third parties; the Company's research, development and regulatory plans, including the Company's plans to engage with the U.S. Food and Drug Administration, the progress of the Company's ongoing and planned clinical trials, including anticipated data readouts from such trials, and the timing of such events may be deemed to be forward-looking statements. 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 or incorporated by reference in this Current Report on Form 8-K 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 Corporation Presentation at the 44th Annual J.P. Morgan Healthcare Conference, dated January 14, 2026
99.2	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: January 14, 2026

By: /s/ Michael J.M. Hitchcock

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

Exhibit 99.1

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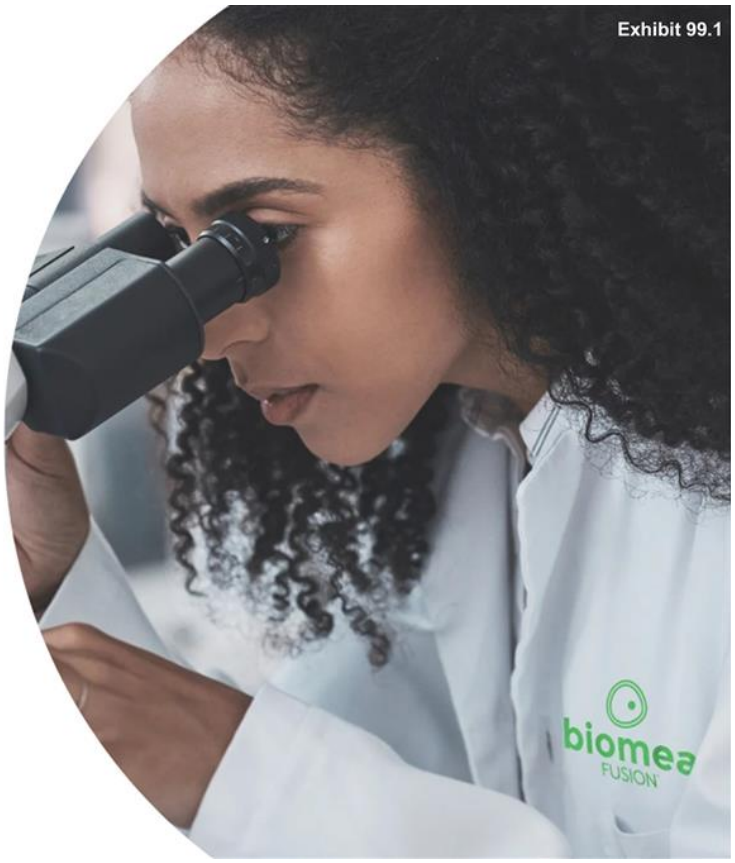
2026 J.P. Morgan Healthcare Conference



Mick Hitchcock, CEO
Biomea Fusion

January 14, 2026

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, the initiation, timing and results of pending or future preclinical studies and clinical trials, the actual or potential actions of the 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.

Biomea Fusion – Development Candidates

Icovamenib

- **Diabetes**
- **Oral**
- **Small molecule**

COVALENT-111 (Phase 2)

- Insulin-deficient Type 2 Diabetes (T2D)
- GLP-1 inadequately controlled

BMF-650 (GLP-1 RA)

- **Weight loss**
- **Oral**
- **Small molecule**

GLP-131 (Phase 1)

Obese (BMI ≥30kg/m^2)

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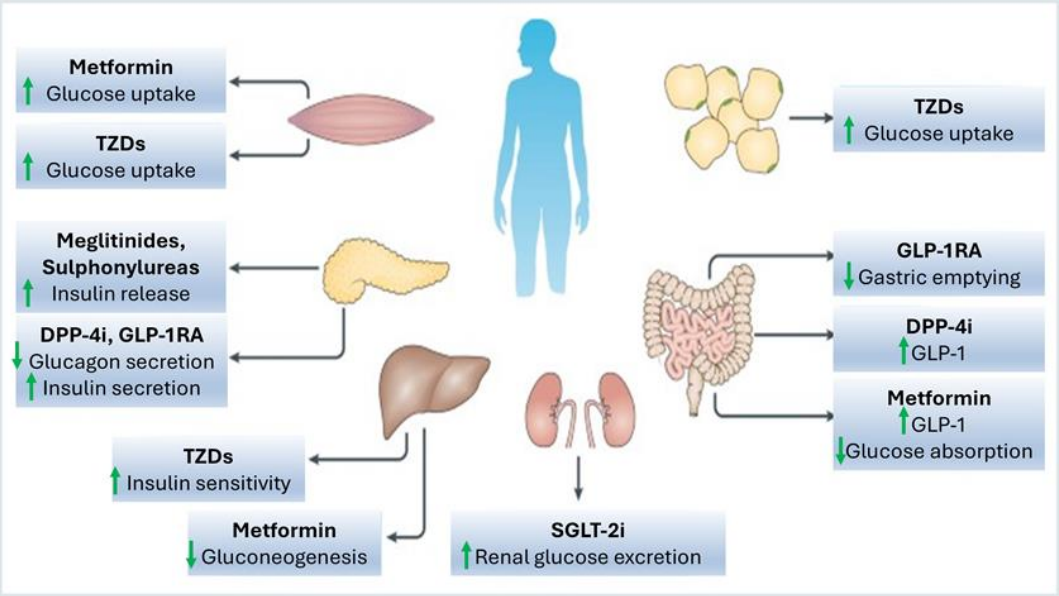
Existing T2D Treatments Address Symptoms, Not Disease Progression



- Existing T2D therapies primarily address downstream metabolic symptoms
- **Icovamenib** is a selective and partial menin inhibitor, targeting a previously unaddressed pathway

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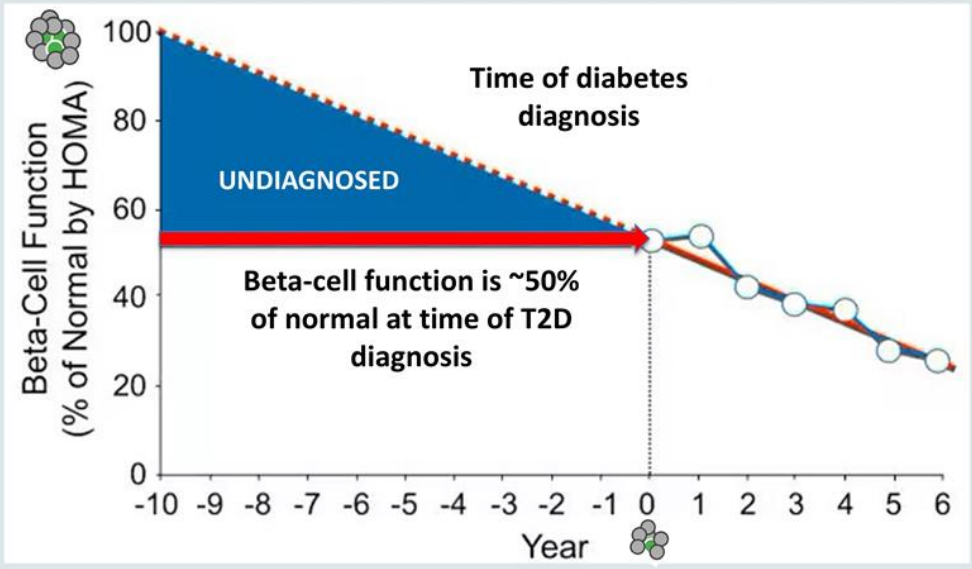
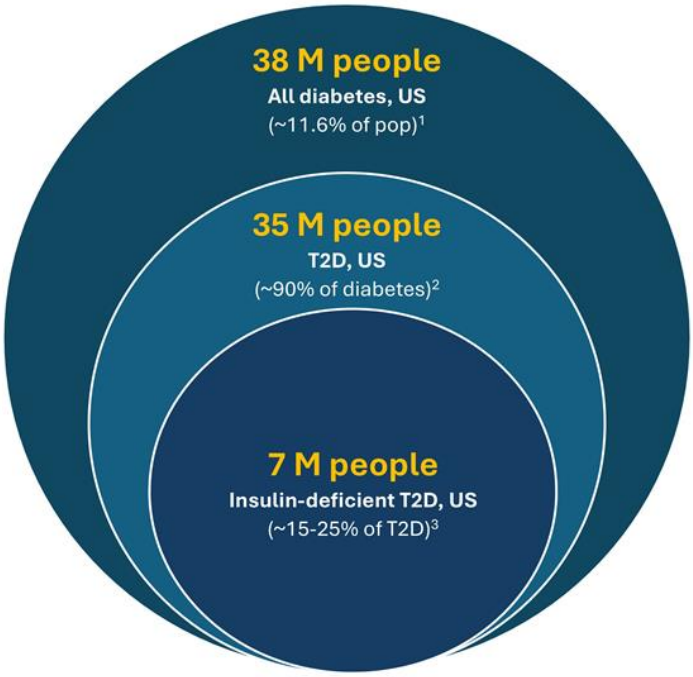
Zhou, Nat Rev Endocrinol, 2016

The Root Cause of Diabetes: T2D is driven by progressive beta-cell failure



Adapted from Lebovitz, Diabetes Rev, 1999

T2D Remains a Growing Health Burden in the US



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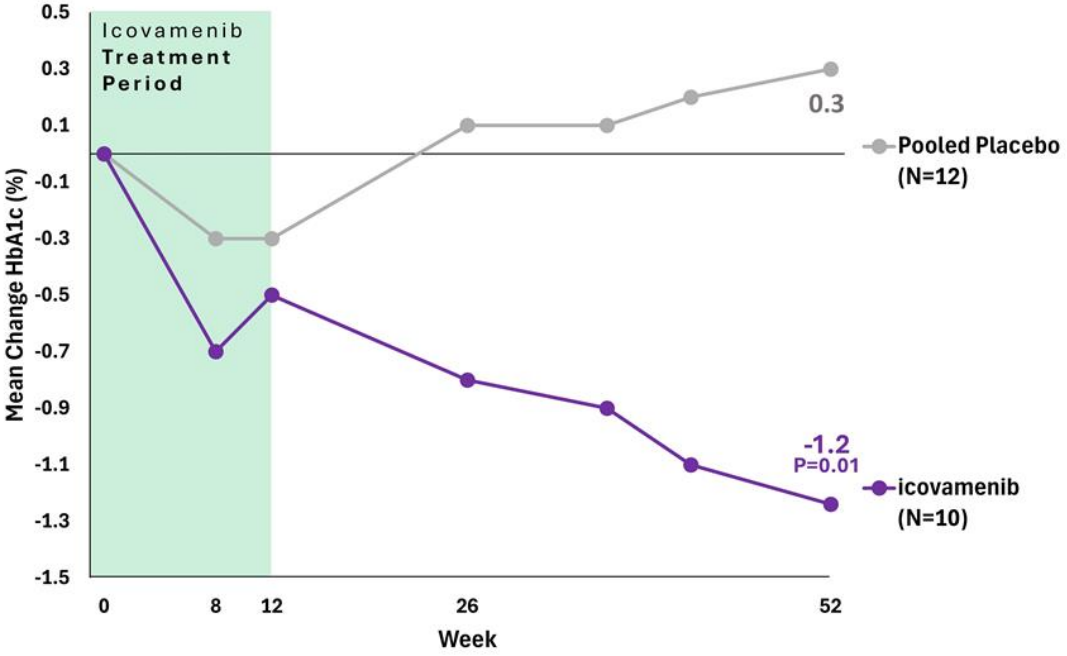
[1]CDC, Natl. Diabetes Stat. Rep., 2022; [2]ADA, Standards of Care in Diabetes, Diabetes Care, 2024; [3]Ahlqvist, Lancet Diabetes Endocrinol., 2018

Severe Insulin-Deficient Diabetes Patients | 12 Weeks of icovamenib Dosing

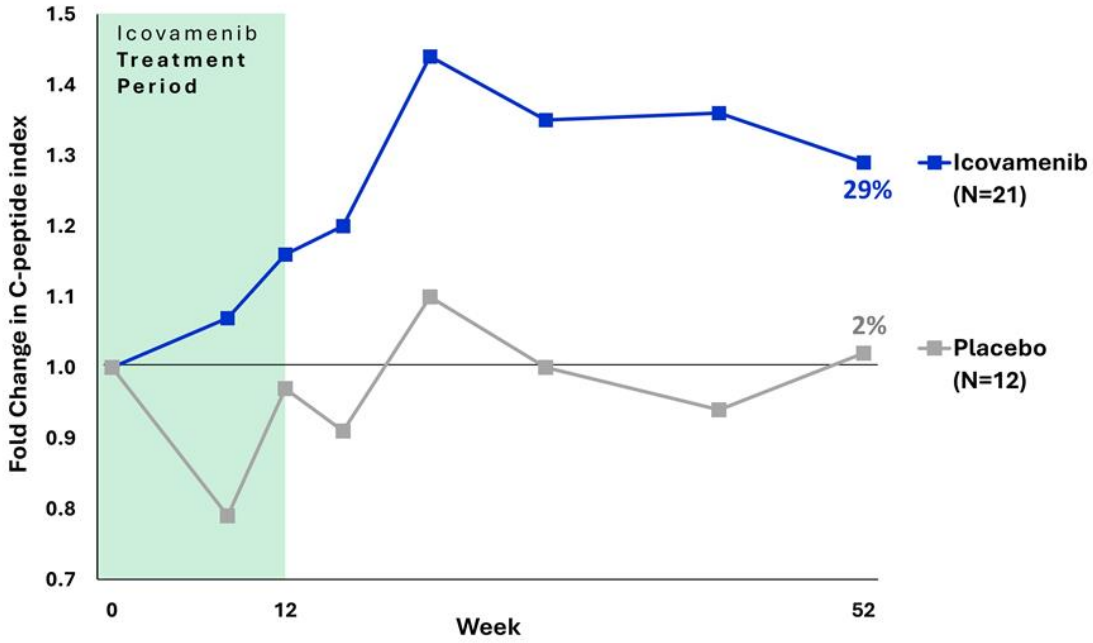
(9 Months After Last Dose)



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

All severe insulin-deficient patients (All Arms)



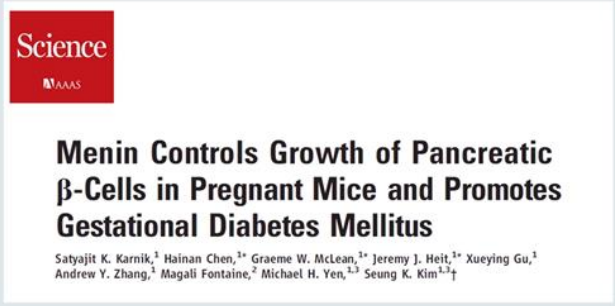
Data censored at onset of rescue medication, defined as any modification in antihyperglycemic therapy

Why did HbA1c decrease beyond 12 weeks?

- **Menin suppressed beta-cell proliferation and function**
  - Demonstrated to act as a biological brake on insulin-producing cells in T2D
- **Menin inhibition was shown to lift this brake**
  - Enabled control on beta-cell regeneration and restoration of insulin secretory capacity
- **A short 12-week dosing period induced durable epigenetic reprogramming**
  - Allowed beta-cell functional regeneration to continue post-treatment and driving sustained HbA1c improvements

Physiologic Suppression of Menin Can Expand Beta-Cell Mass

- Physiologic states such as pregnancy and lactation suppress menin, enabling beta-cell expansion and increased insulin output
- Preclinical and human data consistently link reduced menin signaling to improved beta-cell mass and function
- **Icovamenib** has been shown to directly inhibit menin, aiming to pharmacologically replicate a naturally occurring, validated biologic process


Science
AAAS

Menin Controls Growth of Pancreatic β-Cells in Pregnant Mice and Promotes Gestational Diabetes Mellitus

Satyajit K. Karnik,[1] Hainan Chen,[1*] Graeme W. McLean,[1*] Jeremy J. Heit,[1*] Xueying Gu,[1] Andrew Y. Zhang,[1] Magali Fontaine,[2] Michael H. Yen,[1,3] Seung K. Kim[1,3]†

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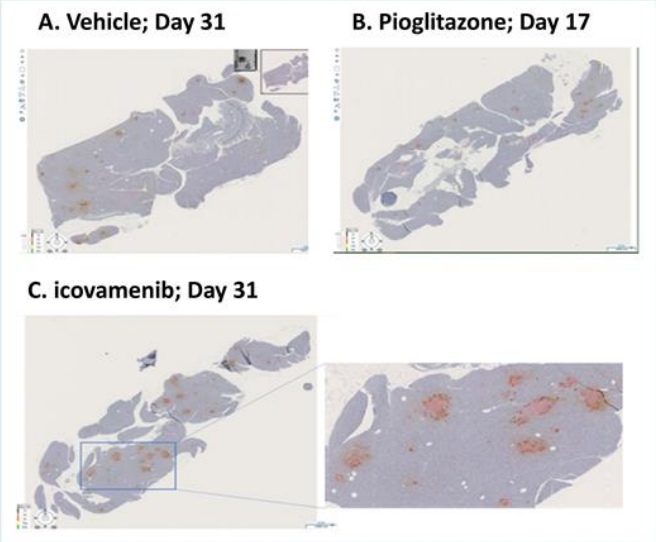
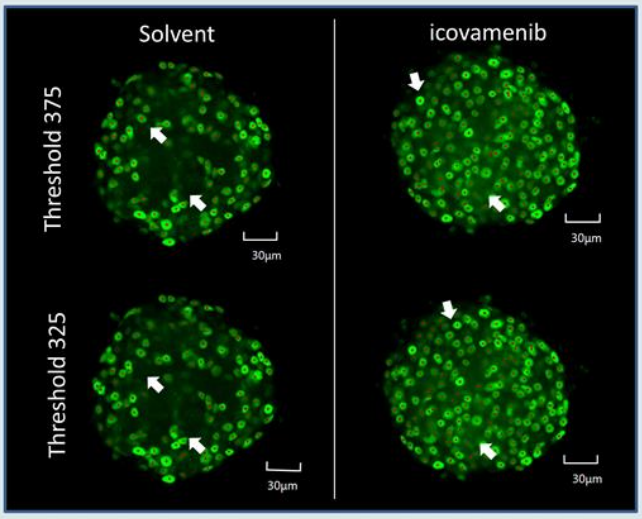
Karnik, Science, 2007

ICOVAMENIB

Inhibiting Menin in Rats and in Human Islet Cells Demonstrates Proliferative Effect



ZDF Diabetic Model: A) Vehicle-treated animal, Day 31. Beta islets display low congregation and growth, while alpha cells dominate. B) Pioglitzaone-treated animal, Day 17. Beta islets display congregation and growth. C) Icovamenib treated animal, Day 31. Beta islets display high congregation and continue to increase and mature. Red is insulin-beta islets, brown is glucagon-alpha cells.

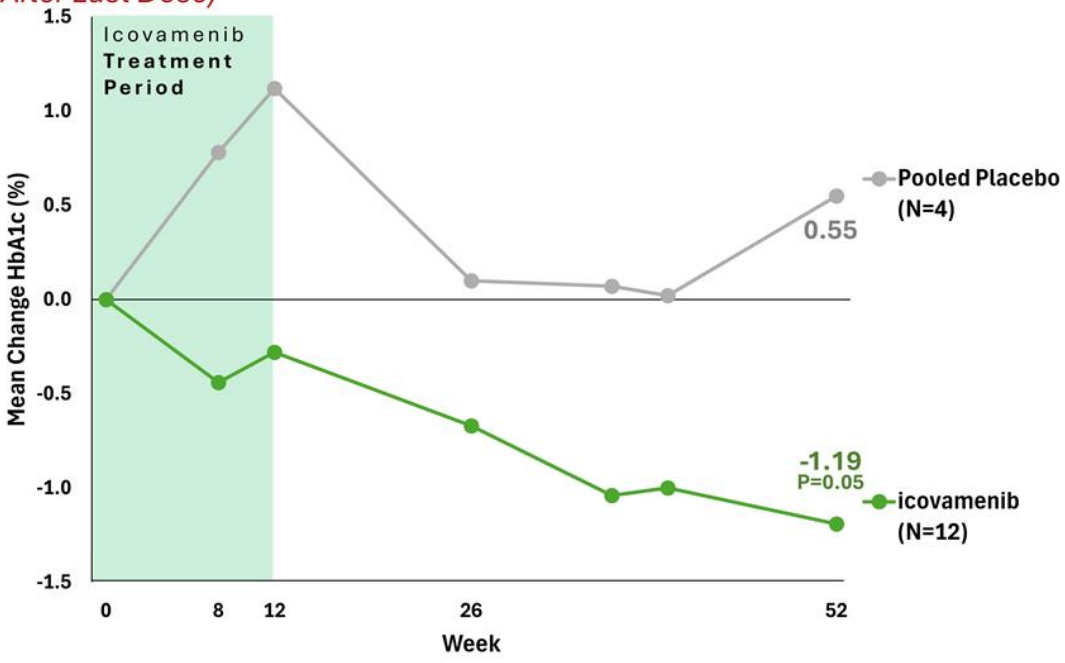


Human Donor Islets (Ex Vivo): Statistically significant increase in beta cells with icovamenib

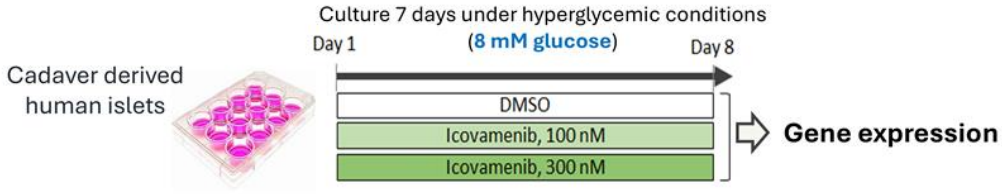
Increased Beta-Cell Mass and Function

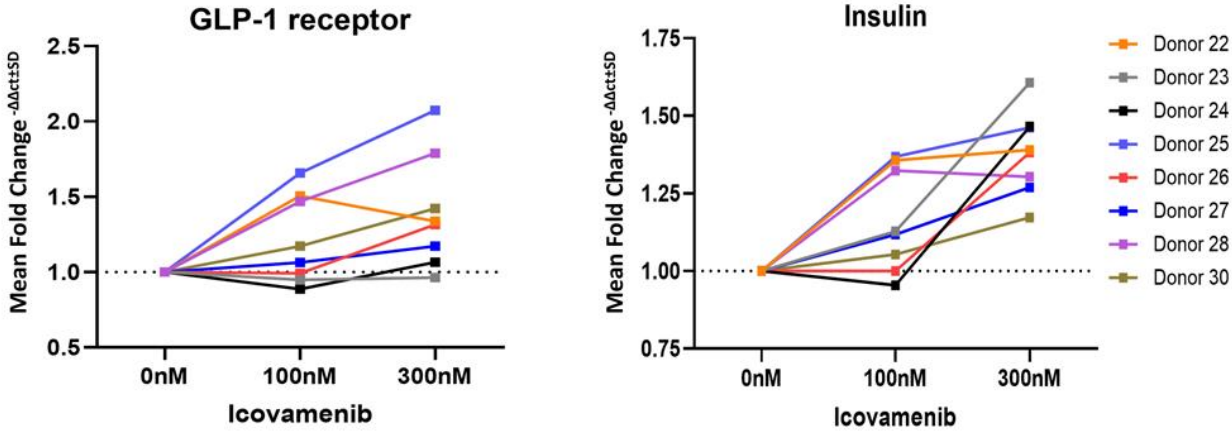
Post-hoc Analysis of Patients on a GLP-1 Based Therapy Not Achieving Target HbA1c <7% at Enrollment

(9 Months After Last Dose)



Icovamenib Enhanced GLP-1 Receptor and Insulin Transcript Levels





Overview of Treatment Emergent Adverse Events (TEAEs) Through 52 Weeks

Parameter	Placebo Combined arms (N=66)	Icovamenib Combined arms (N=201)	Arm A icovamenib (N=67)	Arm B icovamenib (N=67)	Arm C icovamenib (N=67)
Patients with ≥1 TEAE, N (%)	18 (27)	55 (27)	19 (28)	22 (33)	14 (21)
Treatment-Related SAEs, N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
SAEs*, N (%)	1 (1)	2 (1)	1 (1)	0 (0)	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)
Deaths, N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)

Data are n (%) TEAE = Treatment Emergent Adverse event SAE = Serious Adverse Event

*Arm A had an SAE of atrial fibrillation. Unrelated to study treatment and occurred during the treatment period. Subject required hospitalization and was discharged in 3 days. Subject continued in the study.

*Arm C had an SAE of COVID-19. Unrelated to study treatment and occurred during the treatment period. Subject required hospitalization and was discharged in 3 days. Subject continued in the study.

*Placebo Arm had an SAE of nephrolithiasis. Unrelated to study treatment and occurred during the treatment period. Subject required hospitalization and was discharged in 3 days. Subject continued in the study.

Treatment Emergent Adverse Events (TEAEs) Occurring in ≥5% in Any Study Arm and TEAEs Reported for ALT and/or AST Elevations

Parameter	Placebo Combined arms (N=66)	icovamenib Combined arms (N=201)	Arm A icovamenib (N=67)	Arm B icovamenib (N=67)	Arm C icovamenib (N=67)
Diarrhea, N (%)	0	7 (3)	4 (6)	2 (3)	1 (1)
Urinary tract infection, N (%)	3 (5)	1 (0)	0	1 (2)	0
Hyperglycemia, N (%)	3 (5)	8 (4)	2 (3)	5 (7)	1 (1)
Headache, N (%)	2 (3)	5 (2)	0	4 (6)	1 (1)
ALT increase, N (%)	0	5 (2)	3 (4)	0	2 (3)
AST increase, N (%)	0	4 (2)	3 (4)	0	1 (1)
Resolution of ALT/AST w/o interruption in study treatment, %	N/A	100	100	100	100

Data are n (%) of TEAE with ≥5% frequency in any arm and ALT or AST increase irrespective of incidence; Safety population
TEAE, treatment-emergent adverse event; ALT, alanine aminotransferase; AST, aspartate aminotransferase
Diarrhea: In the icovamenib arms, all 7 events were Grade 1.
Nausea: In the icovamenib arms, 6 of 7 events were Grade 1 and 1 event was Grade 2 (Arm B). In the placebo arm, the 1 event was Grade 1.
Hyperglycemia: In the icovamenib arms, 5 of 6 events were Grade 2 and 1 event was Grade 1 (Arm C). In the placebo arm, all 3 events were Grade 2.
Headache: In the icovamenib arms, 3 of the 4 events were Grade 1 and 1 event was Grade 2 (Arm B). In the placebo arm, 2 of the 3 events were Grade 1 and 1 event was Grade 2.
ALT increase: In the icovamenib arms, 3 of the 4 events were Grade 1 and 1 event was Grade 2 (Arm A).
AST increase: In the icovamenib arms, all 3 events were Grade 1.

Icovamenib in T2D: 52-Week Highlights

- **Durable treatment effect in severe insulin-deficient T2D**
  Continued benefit observed in severe insulin-deficient diabetes patients

- **Higher icovamenib exposure (PK) led to improved responses**
  PK analysis shows that greater HbA1c reductions occurred in patients with higher drug exposure

- **Icovamenib increased insulin secretion (C-peptide Index) in severe insulin-deficient T2D**

- **Treatment effect in GLP-1 "failures" continued to improve**
  Demonstrated durable and clinically significant improvements in HbA1c in participants on GLP-1 therapy at baseline

- **Favorable safety profile continued through Week 52**
  Icovamenib was generally well-tolerated, with no adverse-event related discontinuations and no related serious adverse events

COVALENT-111: Potential to Alter the Insulin Treatment Trajectory

COVALENT-111 highlights the potential to restore endogenous insulin production capacity in patients who would otherwise progress to chronic insulin therapy—offering the possibility of short-term oral treatment rather than lifelong injectable management.

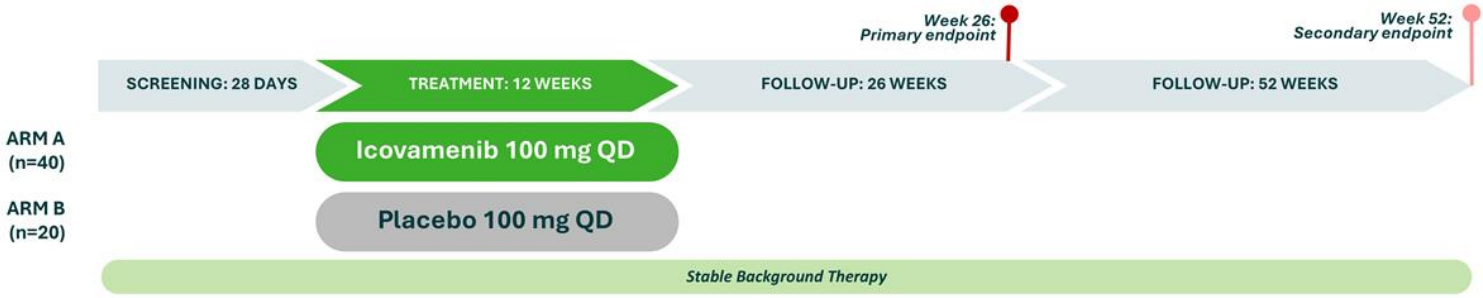
COVALENT-211: A Phase II Trial of Icovamenib in Participants with T2D Who Are Not Achieving Glycemic Targets

Key Inclusion Criteria

Adult participants with T2D treated with 1-3 antidiabetic medications

- HbA1c 7.5 to 10.5%
- BMI ≤ 32 kg/m^2

Background therapy maintained unless rescue required



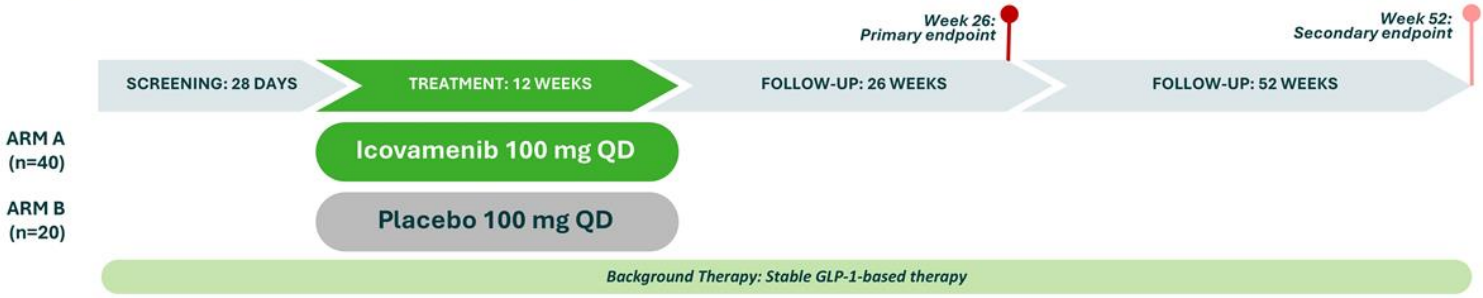
COVALENT-212: A Phase II Trial of Icovamenib in Participants with T2D Mellitus Who Are Not Achieving Glycemic Targets While Using GLP-1-Based Therapy

Key Inclusion Criteria

Adult participants with T2D who are not achieving glycemic targets despite GLP-1-based therapy

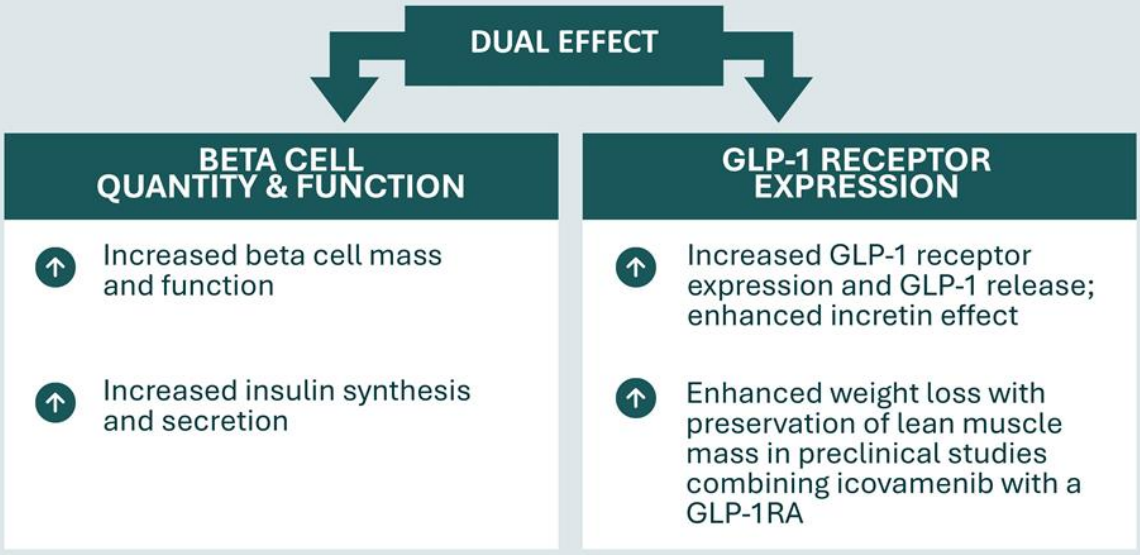
- HbA1c ≥ 7.5 and ≤9.5%
- BMI 25 to 40 kg/m^2

Treatment policy estimand; background therapy maintained unless rescue required



ICOVAMENIB

Mechanism of Action of Icovamenib – a Selective and Partial Menin Inhibitor



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Biomea Fusion – BMF-650

BMF-650 (GLP-1 RA)

- **Weight loss**
- **Oral**
- **Small molecule**

GLP-131 (Phase 1)

Obese (BMI ≥30kg/m^2)

Designed for better bioavailability
>>> More consistent efficacy

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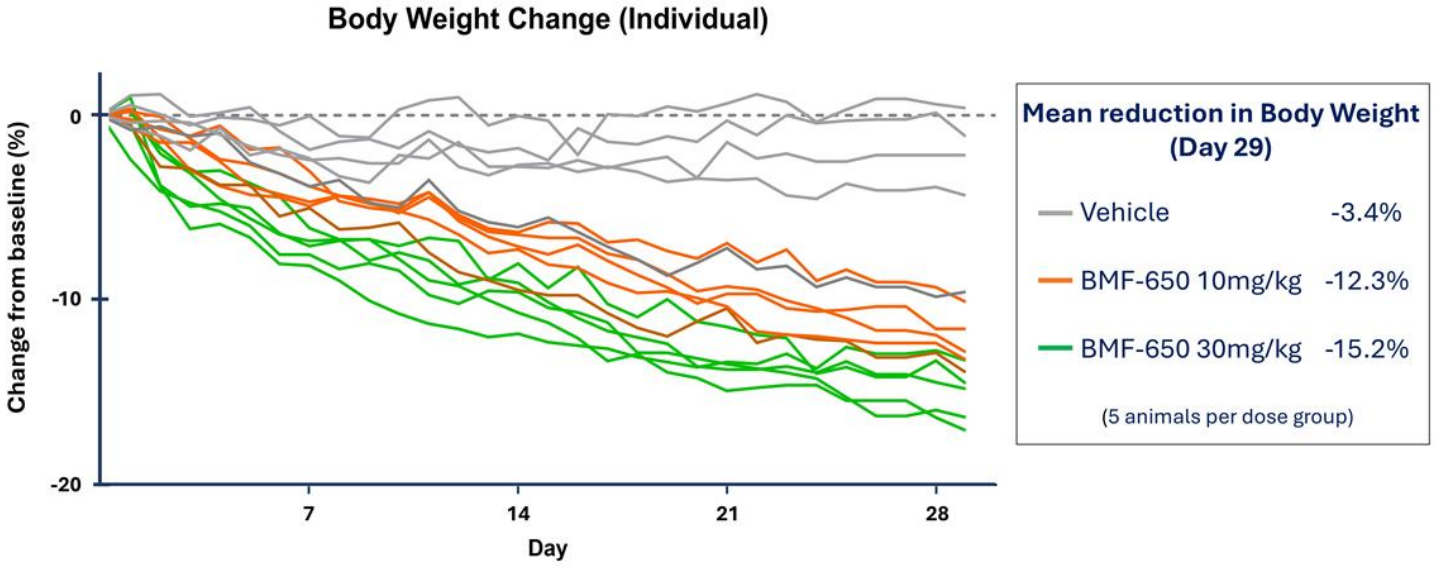
Oral BMF-650 Promoted Body Weight Reduction in Obese Cynomolgus Monkeys



BMF-650 Has Demonstrated Favorable Liver Safety to Date

Chugai chemotype / orforglipron:

- 3000+ patients dosed in ATTAIN 1 / 2 studies[1,2]
  No LFT signals for orforglipron
- **BMF-650** is a member of this chemotype

Pfizer chemotype / danuglipron

- Danuglipron and lotiglipron discontinued[3,4]
  TERN-601 discontinued[5]
- Observation of LFTs elevations

- **Daily oral dosing in cynomolgus monkeys (healthy and obese) for up to 6 weeks**
- **No ALT or AST elevations observed across all preclinical studies**
- **First-in-human study ongoing with no ALT or AST elevations observed to date**

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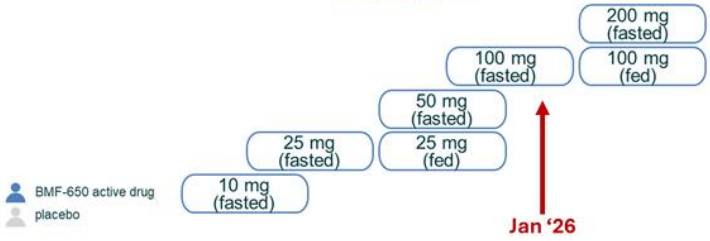
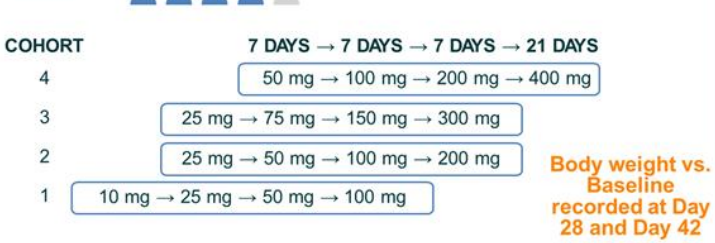
[1]Horn, Lancet, 2026; [2]Knudsen, Cell Chem Biol, 2021; [3]Pfizer press release (danuglipron discontinued, Apr 2025); [4]Pfizer press release (lotiglipron terminated, Jun 2023); [5]Terns Phase 2 topline press release (Oct 2025

GLP-131: A Randomized, Double-blind, Placebo-controlled, FIH Study of an Oral Non-peptide GLP-1 Receptor Agonist

Part 1: single ascending dose (SAD) study | **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





Overview of Key Program Activities

ICOVAMENIB

COVALENT 211 (Phase IIB)

- Insulin-deficient T2D (20% of US T2D population)
- First patient enrollment planned in **1Q 2026**
- 26-Week readout expected in **4Q 2026**

COVALENT 212 (Phase II)

- T2D patients not controlled on GLP-1 based medicines (70% of US GLP-1 population)
- First patient enrollment planned in **1Q 2026**
- 26-Week readout in **4Q 2026**

BMF-650

GLP-131 (Phase I)

- Obese, otherwise healthy volunteers
- Currently dosing single ascending dose
- 28-Day weight loss study results expected in **2Q 2026**

Q&A


THANK YOU
For questions or inquiries, please reach out to Meichiel Weiss at ir@biomeafusion.com
www.biomeafusion.com
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Exhibit 99.2

1ST QUARTER 2026

Biomea Fusion Corporate Presentation

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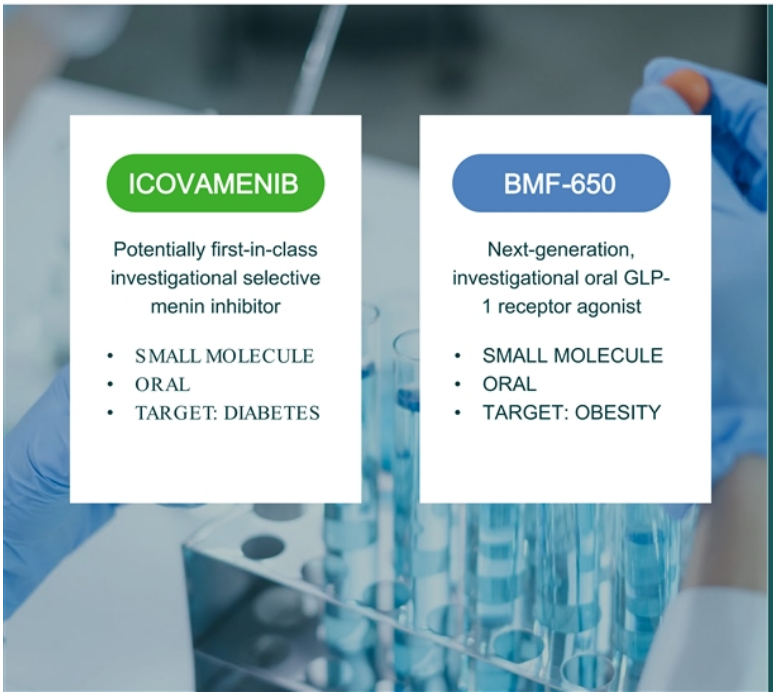
Transformative Oral Therapies for Metabolic Disease



ICOVAMENIB

Potentially first-in-class investigational selective menin inhibitor

- SMALL MOLECULE
- ORAL
- TARGET: DIABETES

BMF-650

Next-generation, investigational oral GLP-1 receptor agonist

- SMALL MOLECULE
- ORAL
- TARGET: OBESITY

Biomea Fusion

(NASDAQ:BMEA) is a diabetes and obesity medicines company developing oral small molecules with the potential to restore beta cell function and reduce body weight while providing convenient, patient-friendly treatment options.

Biomea Pipeline

Biomea Fusion retains full worldwide rights across all its programs

STUDY NAME	INDICATION	PRECLINICAL	PHASE I	PHASE II	PHASE III	NEXT MILESTONE
COVALENT-111	Type 2 Diabetes	ICOVAMENIB (study completed)				52-week follow-up data presented Oct/Dec 2025
COVALENT-211	Type 2 Diabetes *Insulin-Deficient Patients*	ICOVAMENIB (study initiated)				Commencement of Phase IIb (First patient enrollment planned 1Q 2026)
COVALENT-212	Type 2 Diabetes *Patients not controlled on GLP-1-based therapies*	ICOVAMENIB (study initiated)				Commencement of Phase II (First patient enrollment planned 1Q 2026)
GLP-131	Obesity	BMF-650 (study enrolling)				Phase I weight reduction data expected 2Q 2026


ICOVAMENIB
Potential First -in-Class
Oral Menin Inhibitor
Target: DIABETES
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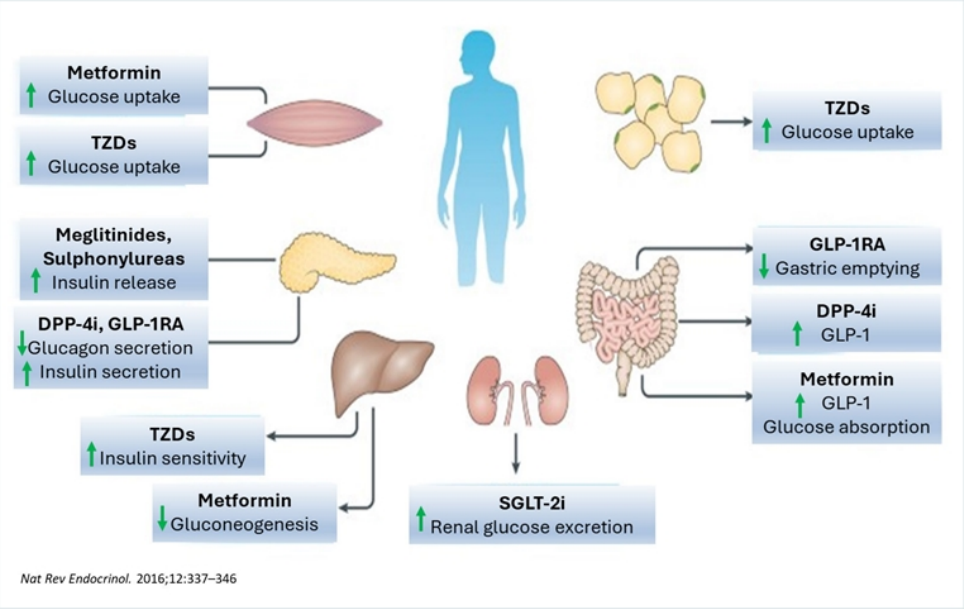
Current Type 2 Diabetes Therapies do not Address the Root Cause of Diabetes - All are Targeting the Symptoms of Hyperglycemia



Nat Rev Endocrinol. 2016;12:337–346

- Existing Type 2 Diabetes therapies primarily address downstream metabolic symptoms
- **Icovamenib** is a first-in-class menin inhibitor, targeting a previously unaddressed pathway

Current Type 2 Diabetes Therapies Require Chronic Treatment and Typically Do not Maintain Long-term Glucose Control

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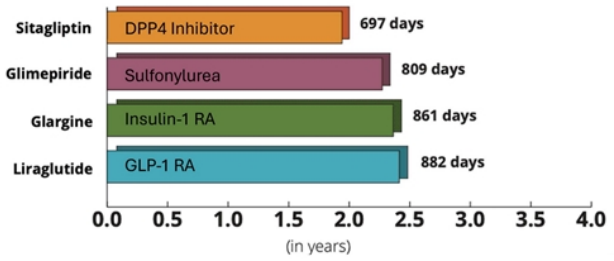
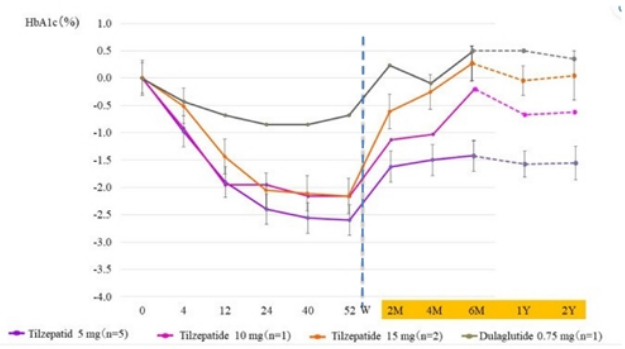
Mean Time to Loss of Glucose Control (HbA1c>7%)



Nathan, et al. N Engl J Med 2022;387:1063-107

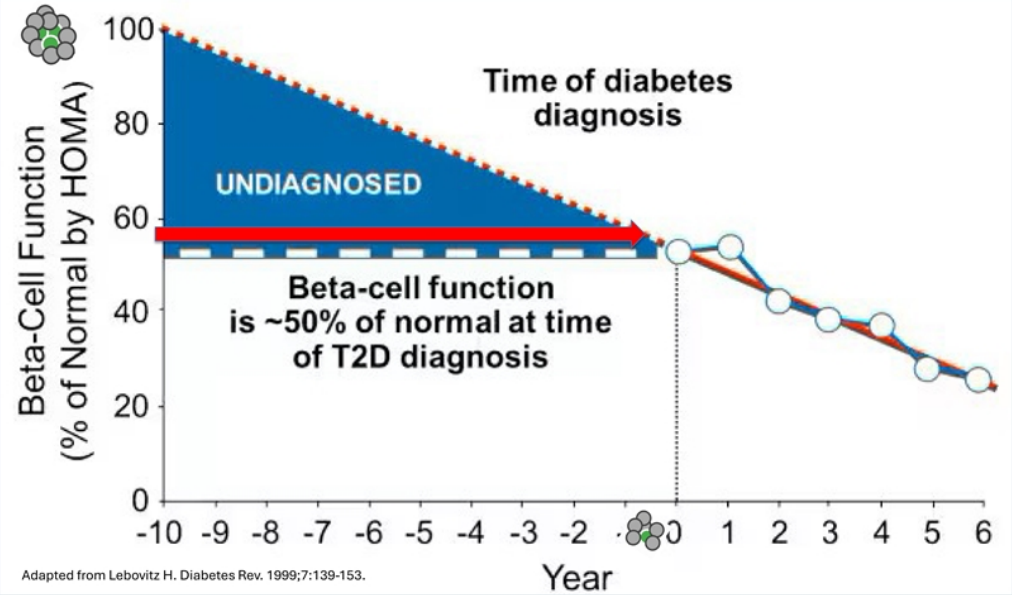
Impact of tirzepatide on HbA1c: Sustained Reduction During Treatment, Rebound After Discontinuation



Kubota M, et al. Cureus. 2023 Oct 4;15(10)

Despite over 60 approved therapies in the US for Type 2 Diabetes, 50% of people with diabetes remain poorly controlled, highlighting the urgent need for novel, durable therapies to improve long-term outcomes

Progressive Decline in Beta-Cell Mass and Function is the Root Cause of Diabetes



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

ICOVAMENIB

Addressing Diabetes at the Root Cause Level

Icovamenib is a potentially **first-in-class investigational oral therapy** designed to be taken for 12 weeks to restore the body's natural insulin production by regenerating beta cells - offering the potential for durable, **disease-modifying benefits for diabetes patients.**

ONCE-DAILY ORAL THERAPY

NON-CHRONIC

GENERALLY FAVORABLE SAFETY-PROFILE OBSERVED TO DATE

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High Unmet Need in Patients with Diabetes

60+

All approved Type 2 Diabetes therapies are chronic agents, none address the root cause of the disease

80m

Type 2 Diabetes US/EU diagnosed patients

15-25%

Type 2 Diabetes patients are severely insulin-deficient. This group has the highest failure rate among all diabetes subgroups [2]

approx. 20m

Addressable U.S./EU target patients [1]

TARGET POPULATION FOR ICOVAMENIB

$20b

Estimated U.S./EU revenue potential

(based on 10% penetration, at 10k p.a.)

1.Zohu Lancet 2024; 404: 2077-93 (adjusted by company to account for **Severe Insulin-deficient patients and those failing a GLP-1 based therapy**)
2.Fendo 2022 doi: 10.3389/fendo.2022.927661 https://doi.org/10.1371/journal.pone.0304036

Menin is Naturally Inhibited During Pregnancy & Breastfeeding Allowing for Beta Cell Regeneration & Reduced Diabetes Risk

Menin inhibition with icovamenib may phenocopy the marked reduction in Type 2 Diabetes incidence observed during pregnancy and after breastfeeding

- 2007 Stanford study found menin regulates islet growth during pregnancy [1]
- Elevated prolactin (during pregnancy and breastfeeding) lowers menin, promoting beta cell growth [1]
- Nursing mice show higher beta cell mass than mice separated from their offspring immediately postpartum [2]
- In humans, lactation lowers the lifetime maternal T2D risk by up to 50% [3-5]
- Reduced Type 2 Diabetes risk persists for up to 30 years postpartum [5]

1. Karnik SK et al._2007_Science_Menin controls growth of pancreatic beta-cells in pregnant mice and promotes gestational diabetes mellitus.
2. Hens JR et al. Protective Effects Of Lactation On Maternal Metabolism. J Endocrine Soc, Volume 7, Issue Supplement_1, Abst ract citation ID: bvad114.737, Diabetes And Glucose Metabolism, THU302, October–November 2023.
3. Kim SY_2018_KJFM_Breastfeeding can reduce the risk of developing diabetes.
4. Pinho-Gomes A-C et al._2021_Diabetes Obes Metab_Association of lactation with maternal risk of type 2 diabetes - A systematic review and meta-analysis of observational studies.
5. Gunderson EP et al._2018_JAMA Int Med_Lactation duration and progression to diabetes in women across the childbearing years - The 30-year CARDIA study

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Mechanism of Action of Icovamenib – a Selective and Partial Menin Inhibitor

DUAL EFFECT

BETA CELL QUANTITY & FUNCTION

- Increased beta cell mass and function
- Increased insulin synthesis and secretion

GLP-1 RECEPTOR EXPRESSION

- Increased GLP-1 receptor expression and GLP-1 release; enhanced incretin effect
- Enhanced weight loss with preservation of lean muscle mass in preclinical studies combining icovamenib with a GLP-1RA

DIFFERENTIATING FEATURES

- ORAL
- NON-CHRONIC
- GENERALLY WELL TOLERATED
- COMPLIMENTARY TO OTHER AGENTS USED


ICOVAMENIB
52-Week Results
COVALENT-111 Phase II Study
Icovamenib in Type 2 Diabetes – All Comers
Reported in 4Q 2025
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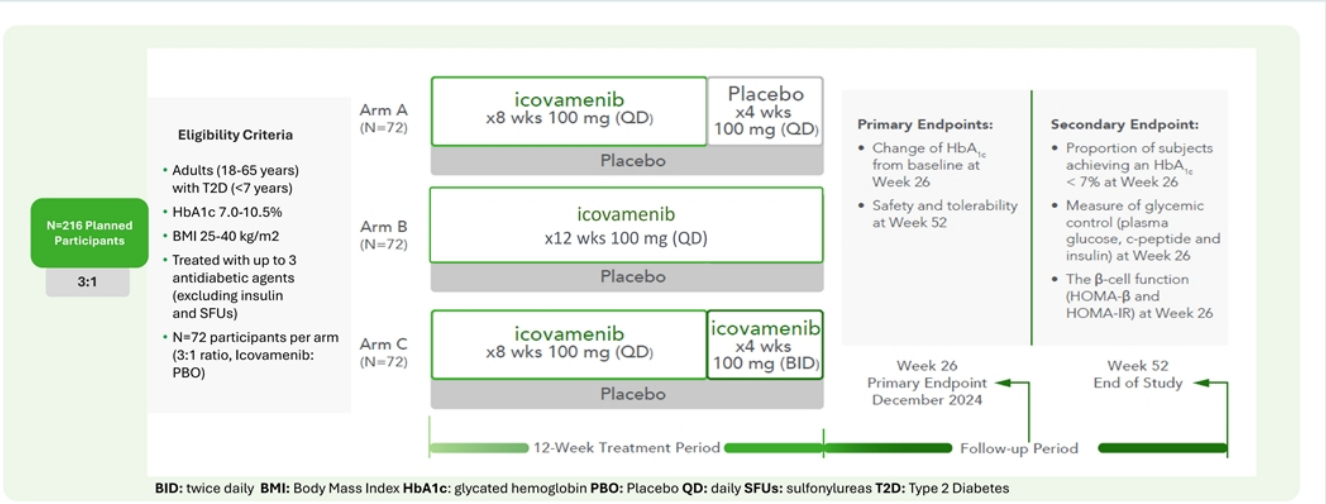
DIFFERENTIATING FEATURES

- ORAL
- NON-CHRONIC
- GENERALLY WELL TOLERATED
- COMPLIMENTARY TO OTHER AGENTS USED

COVALENT-111 Trial Design

Phase 2a Randomized, Double-Blind, Placebo-controlled Study in Participants with T2D, All Comers



BID: twice daily **BMI:** Body Mass Index **HbA1c**: glycated hemoglobin **PBO:** Placebo **QD:** daily **SFUs:** sulfonylureas **T2D:** Type 2 Diabetes

COVALENT-111 Statistical Analysis Plan

Read Out of Insulin-Deficient & Insulin Resistant Subgroups at Weeks 26 and 52

Arm A, B, and C primary analysis:
Change in HbA1c

Prespecified subgroup analysis to include assessment of HbA1c change within each T2D subgroup:

- Severe Insulin Deficient Diabetes
- Mild Age-Related Diabetes
- Mild Obesity Diabetes
- Severe Insulin Resistant Diabetes

Subgroup analysis based on algorithm established per Ahlqvist et al.

(Lancet Diabetes Endocrinol. 2018;6:361-369)

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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)

DEMOGRAPHICS AND BASELINE CHARACTERISTICS WERE WELL MATCHED BETWEEN ICOVAMENIB AND PLACEBO-TREATED PARTICIPANTS

*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.

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Antihyperglycemic Agents at Baseline

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

Parameter		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 100mg BID) (N=33)	Combined Arms icovamenib (N=114)	Combined Arms placebo (N=49)
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)
Metformin Monotherapy, n (%)		36 (80)	18 (50)	22 (67)	76 (67)	38 (78)
SGLT2i, n (%)		6 (13)	12 (33)	8 (24)	26 (23)	7 (14)
DPP4i, n (%)		3 (7)	4 (11)	3 (9)	10(9)	2 (4)
GLP-1 based medicines, n (%)		3 (7)	3 (8)	5 (15)	11 (10)	4 (8)

MOST PARTICIPANTS WERE TREATED WITH METFORMIN MONOTHERAPY, WITH APPROXIMATELY 20% TREATED WITH SGLT2I, 10% WITH DPP4I, AND 10% WITH GLP -1 BASED MEDICINES

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T2D Subtype at Baseline

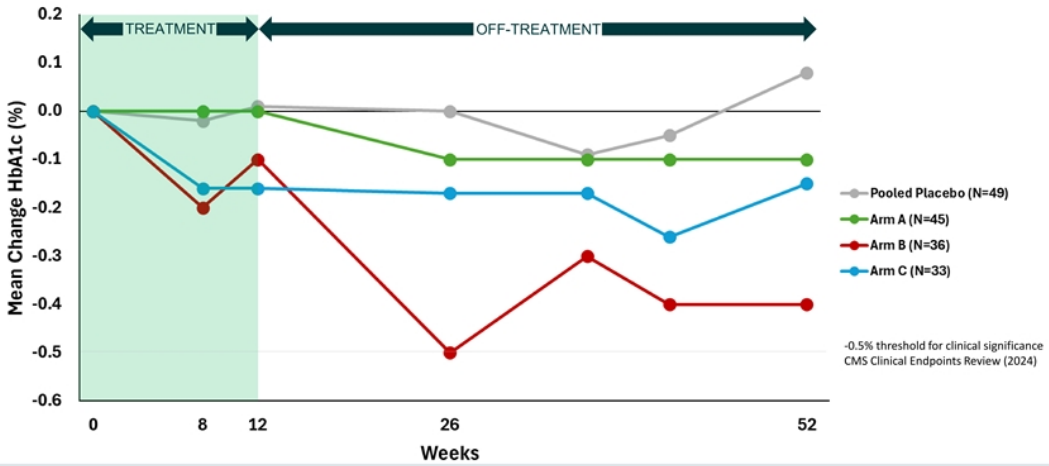
Per Protocol Population on 1 or More Antihyperglycemic Agents at Baseline (N=163)

Parameter	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)
SIDD, n (%)	11 (24)	6 (17)	4 (12)	21 (18)	12 (24)
MARD, n (%)	11 (24)	6 (17)	5 (15)	22 (19)	8 (16)
MOD, n (%)	21 (47)	22 (61)	22 (67)	65 (57)	24 (49)
SIRD, n (%)	2 (4)	2 (6)	2 (6)	6 (5)	5 (10)

SIDD = Severe Insulin-Deficient Diabetes
MARD = Mild Age-Related Diabetes
MOD = Mild Obesity-Related Diabetes
SIRD = Severe Insulin-Resistant Diabetes

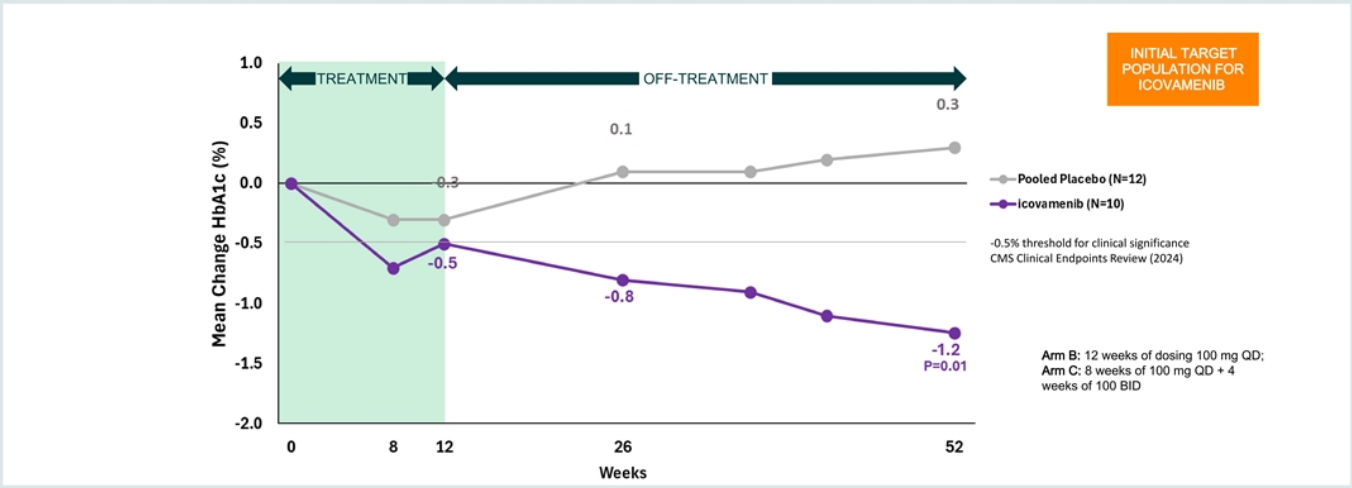
Change in HbA1c from Baseline through Week 52 – All Subtypes

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.

12 Weeks of Dosing (Arms B & C) Delivered Lasting Benefit Through 52 Weeks for Severe Insulin-Deficient Diabetes Patients - 9 Months After Last Dose



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

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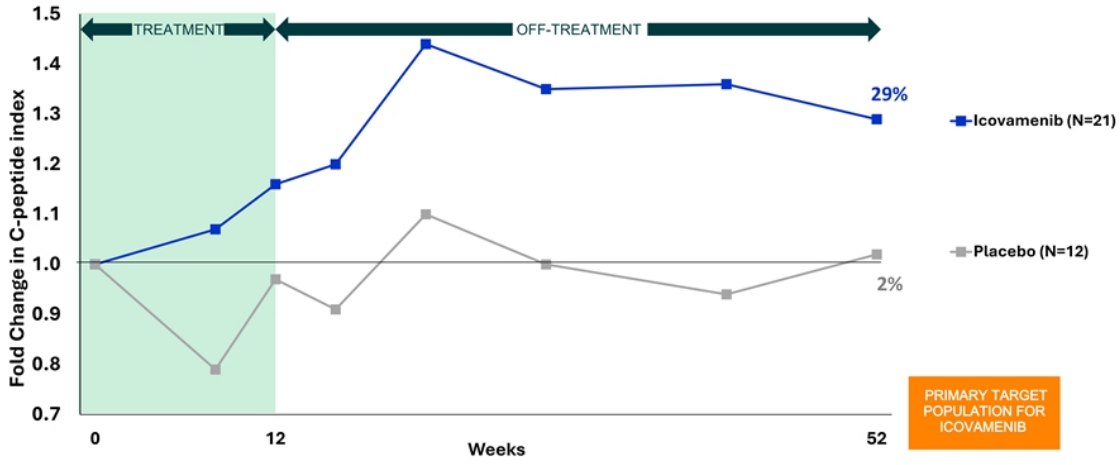
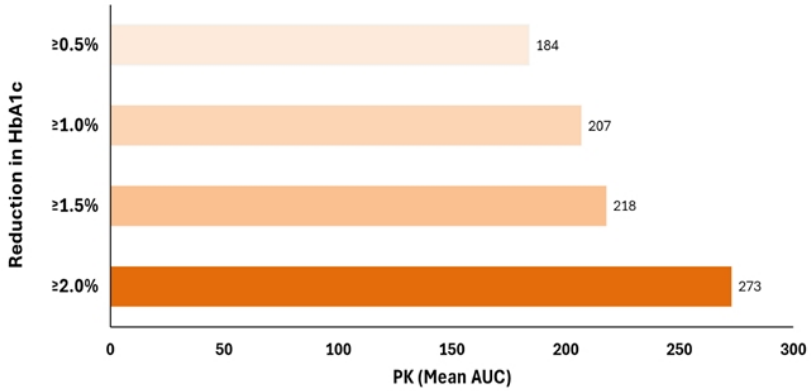
Icovamenib increased Insulin Secretion as Measured by C-peptide Index
All severe insulin-deficient participants (All Arms A, B, C)



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 can 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 inform the dosing strategy for Phase II studies

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Mechanism of Action of Icovamenib – a Selective and Partial Menin Inhibitor

DUAL EFFECT

BETA CELL QUANTITY & FUNCTION

- Increased beta cell mass and function
- Increased insulin synthesis and secretion

GLP-1 RECEPTOR EXPRESSION

- Increased GLP-1 receptor expression and GLP-1 release; enhanced incretin effect
- Enhanced weight loss with preservation of lean muscle mass in preclinical studies combining icovamenib with a GLP-1RA

DIFFERENTIATING FEATURES

- ORAL
- NON-CHRONIC
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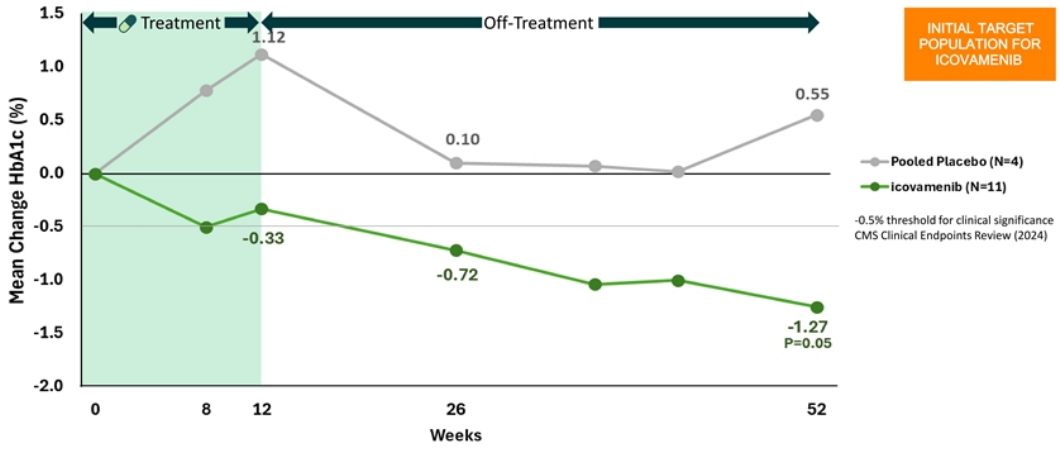
- COMPLIMENTARY TO OTHER AGENTS USED

ICOVAMENIB | COVALENT-111

Post-hoc Analysis: Patients on a GLP-1 Based Therapy at Enrollment (n=11, All Obese Patients) Showed Durable & Clinically Meaningful Response

9 Months After Last Dose



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Short Treatment with Icovamenib Delivered HbA1c Reductions Comparable to Chronic Injectable & Oral Standards of Care

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

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

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

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

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.

Overview of Treatment Emergent Adverse Events (TEAEs) Through 52 Weeks

(Safety Population, N=267)

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)
Deaths, N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)

Data are n (%) TEAE = Treatment Emergent Adverse event. SAE = Serious Adverse Event.
*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.

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Treatment Emergent Adverse Events (TEAEs) Occurring in ⩾5% in Any Study Arm and TEAEs Reported for ALT and/or AST Elevations

(Safety Population, N=267)

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)
Diarrhea, N (%)	4 (6)	2 (3)	1 (1)	7 (4)	0
Nausea, N (%)	2 (3)	3 (4)	2 (3)	7 (4)	1 (2)
Hyperglycemia, N (%)	2 (3)	5 (7)	1 (1)	8 (4)	3 (5)
Headache, N (%)	0	4 (6)	1 (1)	5 (3)	2 (3)
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 interruption in study treatment, %	100	100	100	100	N/A

Data are n (%) of TEAE with ⩾5% frequency in any arm. ALT (alanine aminotransferase) or AST (aspartate aminotransferase increase) irrespective of incidence %.

Diarrhea: In the icovamenib arms, all 7 events were Grade 1.

Nausea: In the icovamenib arms, 6 of 7 events were Grade 1, and 1 event was Grade 2. In the placebo arm, the 1 event was Grade 1.

Hyperglycemia: In the icovamenib arms, 2 of 8 events were Grade 1, and 6 events was Grade 2. In the placebo arm, all 3 events were Grade 2.

Headache: In the icovamenib arms, 4 of the 5 events were Grade 1, and 1 event was Grade 2. In the placebo arm, 1 of the 2 events was Grade 1, and 1 event was Grade 2.

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.

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Key Findings Through Week 52 After Only a Short Treatment Course

Menin Inhibition Potentially Leads to Increased Clinical Benefit

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

Durable Clinical Activity in Insulin Deficient T2D

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

Favorable Safety

Generally well tolerated, no treatment-related serious adverse events

NEXT STEPS: COVALENT-211 PHASE IIB IN SEVERE INSULIN DEFICIENT T2D, FIRST PATIENT PLANNED FOR ENROLLMENT IN 1Q 2026. COVALENT-212 PHASE II T2D PATIENTS NOT CONTROLLED ON GLP-1 BASED THERAPIES. FIRST PATIENT PLANNED FOR ENROLLMENT IN 1Q 2026.

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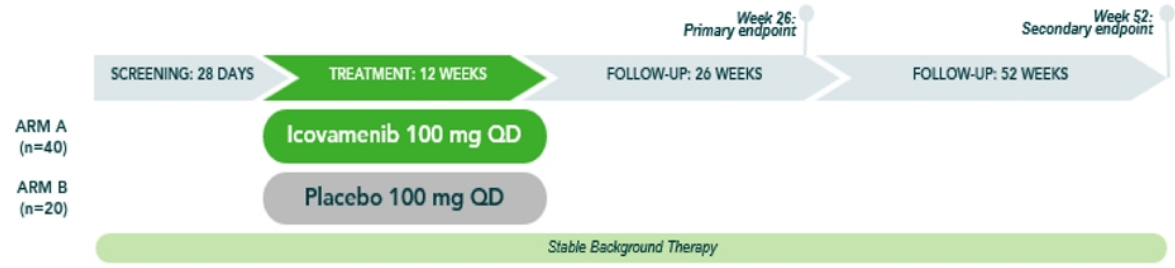
A Phase II Trial of Icovamenib in Insulin Deficient Participants with Type 2 Diabetes Who Are Not Achieving Glycemic Targets

N=60 2:1 randomization

Objectives	Endpoints	Inclusion Criteria
To demonstrate that icovamenib 100 mg once daily for 12 weeks is superior to placebo for glycemic control at Week 26 and Week 52	• Mean change in HbA1c from baseline at Week 26 and 52 • Mean change in fasting plasma glucose (FPG) from baseline at Week 26 • Safety	• Adult participants with Type 2 Diabetes who were treated with 1-3 antidiabetic medications • HbA1c 7.5%-10.5% • BMI ≤ 32 kg/m2 • Background therapy maintained unless rescue required

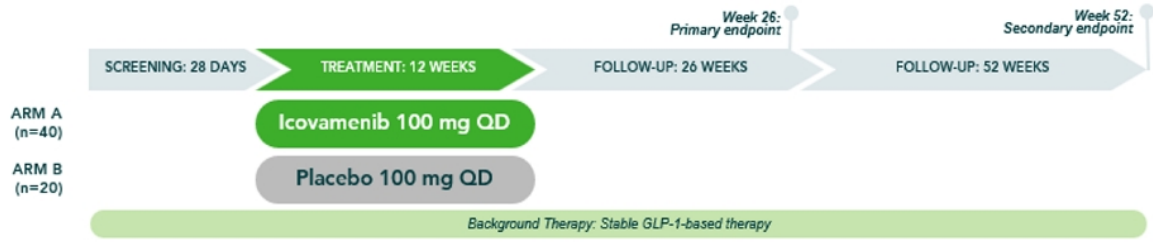


ICOVAMENIB | COVALENT-212

A Phase II Trial of Icovamenib in Participants with Type 2 Diabetes Who Are Not Achieving Glycemic Targets While Using GLP-1-Based Therapy

N=60 2:1 randomization

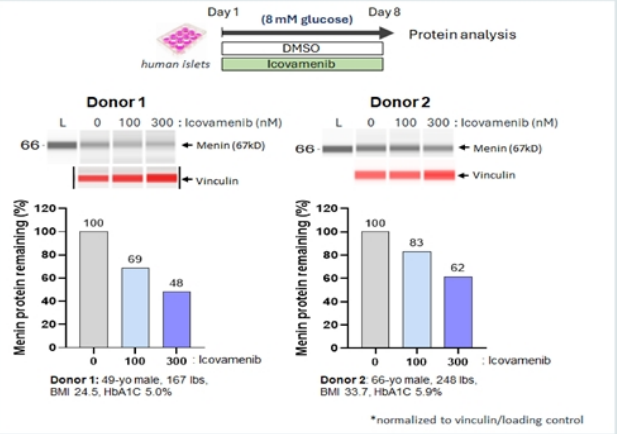
Objectives	Endpoints	Inclusion Criteria
To demonstrate that icovamenib 100 mg once daily for 12 weeks is superior to placebo for glycemic control at Week 26 and Week 52	• Mean change in HbA1c from baseline at Week 26 and 52 • Mean change in fasting plasma glucose (FPG) from baseline at Week 26 • Safety	• Adult participants with T2D who are not achieving glycemic targets despite GLP-1-based therapy • HbA1c ⩾7.5% and ≤9.5% • BMI 25 to 40 kg/m2 • Background therapy maintained unless rescue required



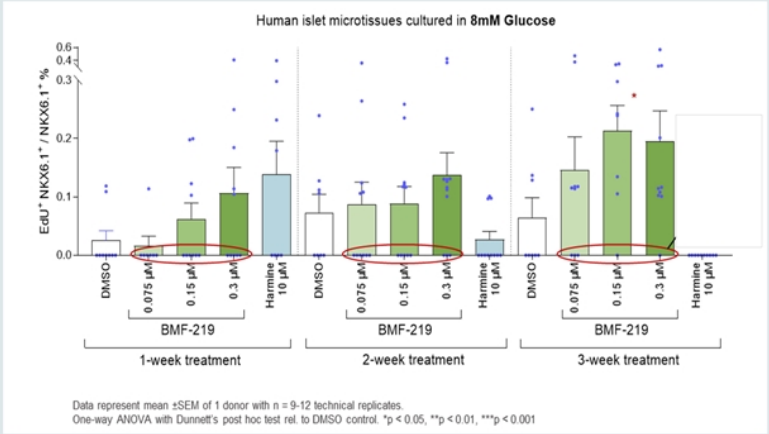

ICOVAMENIB
Preclinical Study Results
Icovamenib in Combination with
GLP-1 Based Therapies
FLAMMABLE
KEEP FIRE
AWAY
CABINET L
biomea
FUSION

Icovamenib Downregulated Menin Protein Levels and Proliferated Beta Cells In Preclinical Human Islet Experiments

Post Icovamenib Treatment
Menin Levels are Downregulated



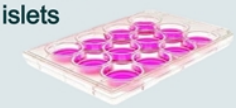
Post Icovamenib Treatment
Beta Cells Proliferate

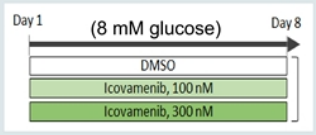


ICOVAMENIB WITH GLP-1RA

Icovamenib Enhanced GLP-1 Receptor & Insulin Expression

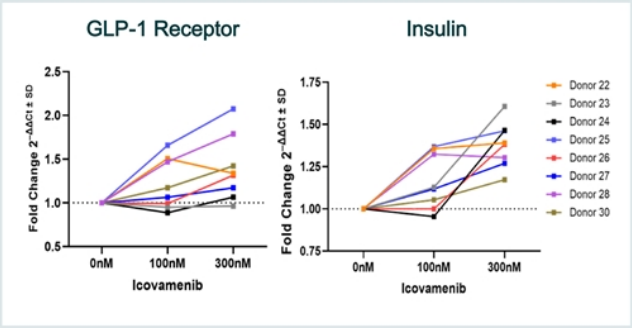
Cadaver derived human islets



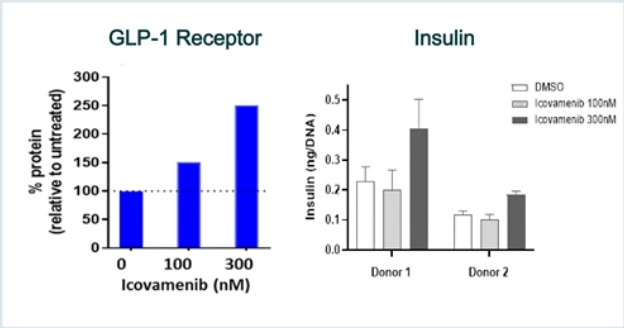


Gene expression
& Protein analysis

Post Icovamenib Treatment
Increased Gene Expression GLP-1 Receptor and Insulin



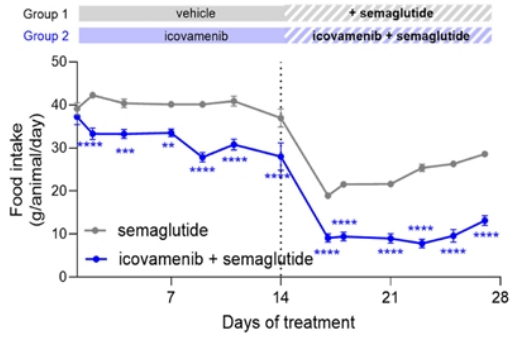
Post Icovamenib Treatment
Increased Protein Levels of GLP-1 Receptor and Insulin



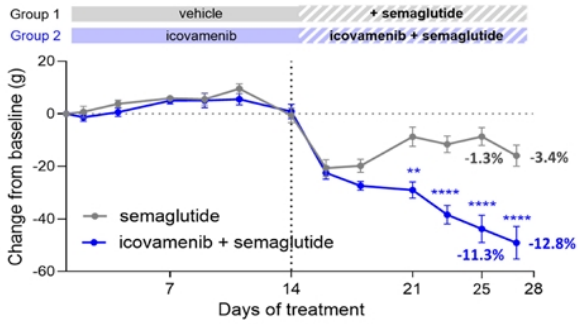
ICOVAMENIB WITH GLP-1RA

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

APPETITE SUPPRESSION



BODY WEIGHT REDUCTION



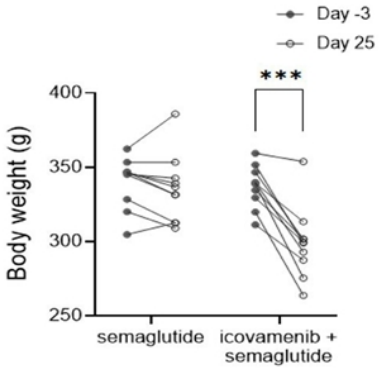
OBSERVED SUPERIOR APPETITE SUPPRESSION WITH ABOUT 10% GREATER BODY WEIGHT REDUCTION THAN LOW-DOSE SEMAGLUTIDE ALONE

THE OBSERVED BODY WEIGHT LOSS WAS PRIMARILY DUE TO FAT MASS REDUCTION WITH COMPLETE PRESERVATION OF LEAN MASS

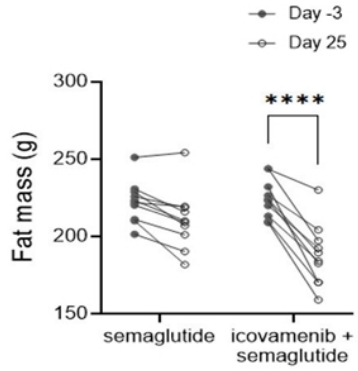
ICOVAMENIB WITH GLP-1RA

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

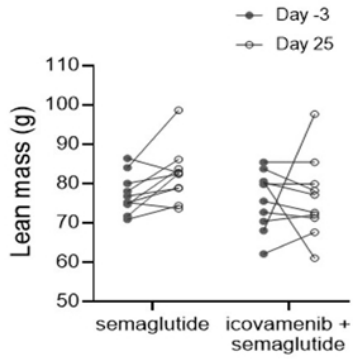
BODY WEIGHT



FAT MASS



LEAN MASS




BMF-650
Next-Generation Oral GLP -1 Receptor Agonist
Preclinical Results & Clinical Overview
Target: OBESITY
biomea FUSION

BMF-650

Designed to Deliver Strong Efficacy with Improved Oral Tolerability

A Next-Generation Oral GLP-1 Receptor Agonist

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

Differentiated properties exhibited by BMF-650

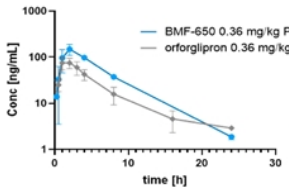
"Why is a greater therapeutic window important?"

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

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

Pharmacokinetics of BMF-650 Showed Very Good Preclinical Bioavailability with Low Inter-Individual Variability

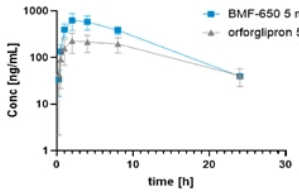
CYNOMOLGUS MONKEY PO PK



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

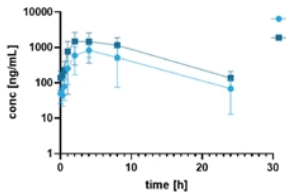
SPRAGUE-DAWLEY RAT PO PK



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



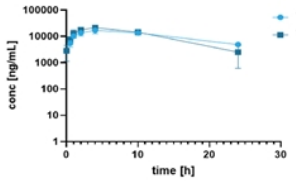
Dose Proportionate Exposure

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

Preclinical Weight-Loss Study in Obese Cynomolgus Monkeys

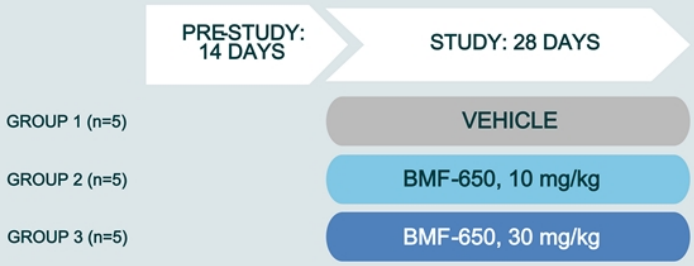
STUDY OVERVIEW

STUDY DESIGN

- 14-day acclimation; 3 groups (n=5)
- QD dosing via oral gavage for 28 days
- BMF-650: 10 or 30 mg/kg, or vehicle

ASSESSMENTS

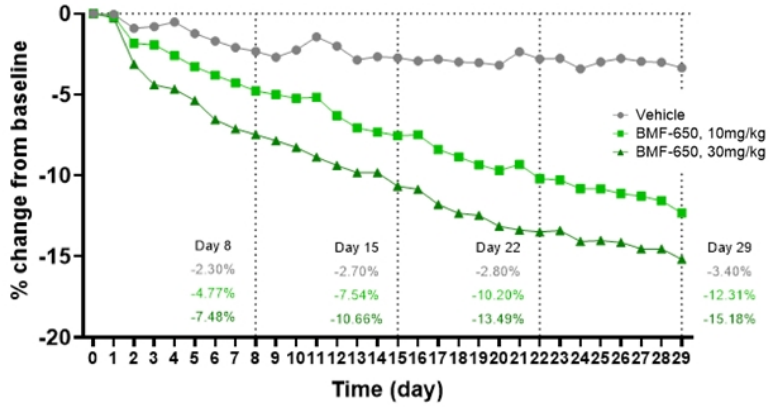
- Food provided as breakfast, fruit snack, and dinner; intake tracked
- Body weight & physicals recorded daily
- Lab values measured on days -5, 13, 20, 29
- Study captured daily food intake & weight change



Oral BMF-650 Weight Loss Study in Obese Cynomolgus Monkeys

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

BMF-650 BODY WEIGHT CHANGE



CT-996 BODY WEIGHT CHANGE

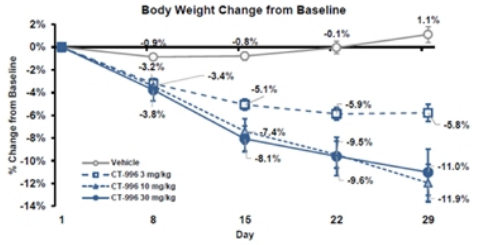


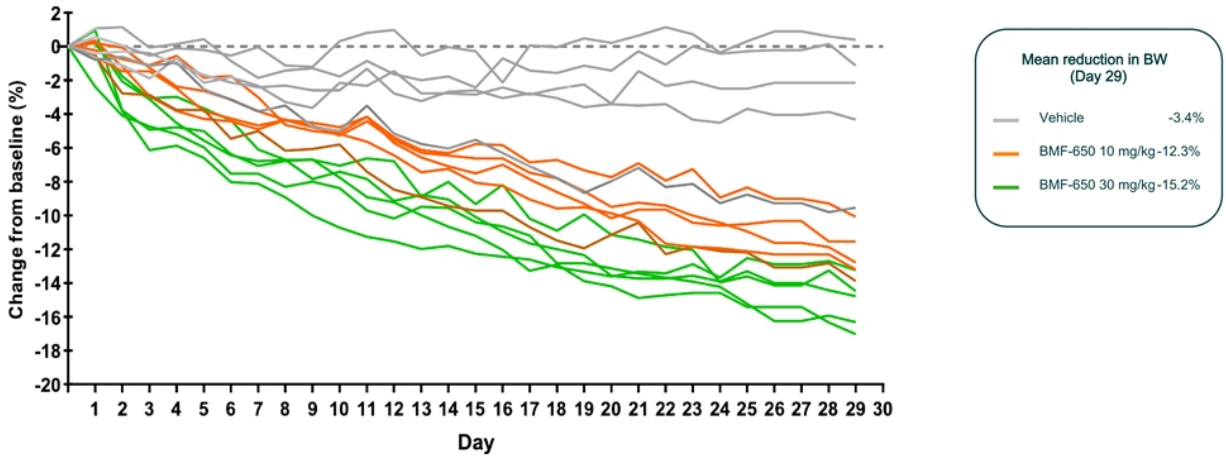
Figure 6. Effects of CT-996 on body weight in obese cynomolgus monkeys following once-daily oral administration. Weekly body weight percent change is represented as mean (± SE) from baseline. N = 6/group.

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.

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

BODY WEIGHT CHANGE (individual obese monkey)



BMF-650 | PRECLINICAL

Oral BMF-650 Generally Well Tolerated in 28 Day Preclinical Animal Study

Low rate of emesis events, mostly in one animal, that decreased rapidly over time

WEEK	EMESIS (Events per 70 weekly dosing occurrences)
1	16%*
2	4.2%
3	4.2%
4	1.4%

*One monkey (#3) accounted for 8 of the 18 total events

SAFETY SUMMARY

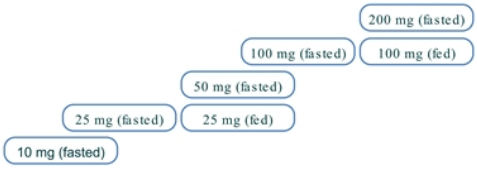
- BMF-650 generally well tolerated with no elevations of AST or ALT
- With 420 dosing occurrences (280 active/140 placebo) there were only a total of 18 (6.4%) events of emesis in the active group
- Most events occurred early, with a marked decline after the first week
- Study was run without a titration scheme, once daily dosing over 28 days

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	50 mg → 100 mg → 200 mg → 400 mg
3	25 mg → 75 mg → 150 mg → 300 mg
2	25 mg → 50 mg → 100 mg → 200 mg
1	10 mg → 25 mg → 50 mg → 100 mg

Body weight vs. Baseline recorded at Day 28 and Day 42

BMF-650

Allowance for Intellectual Property of BMF-650 Received

BMF-650 Intellectual Property:

Our US and PCT applications for BMF-650 are published and proceeding through examination

We received allowance in mid December 2025 for our US patent application covering BMF-650

Additional details, including the list of allowed claims, are available through the U.S. Patent and Trademark Office (USPTO).

BMF-650 | GLP-131

Oral BMF-650 Preclinically Demonstrated a Strong Profile with Consistent Exposure & Weight Loss Effect

Phase I study currently enrolling

- Similar to the broader orforglipron chemotype
- Superior oral bioavailability observed vs. orforglipron (across species)
- Intrinsic potency
- Robust appetite suppression and weight reduction in primate models
- Projected clinical dose aligned with other leading oral GLP-1 agents
- Generally well tolerated with no safety concerns identified to date

CLINICAL STUDY

- Phase I study in obese, otherwise healthy volunteers is currently enrolling
- 28-day weight reduction data anticipated in 2Q 2026

Advancing Multi Program Clinical Pipeline Through 2026

ICOVAMENIB

COVALENT-121

Food Effect Study

+ Optimize Dosing Criteria
+ Started September 2025
+ Completed in December 2025

ICOVAMENIB

COVALENT-211

Phase IIb Insulin Deficient T2D Patients

+ Evaluate icovamenib in Insulin Deficient T2D Patients
+ First Patient Enrollment Panned in 1Q 2026

ICOVAMENIB WITH GLP-1RA

COVALENT-212

Phase II T2D Patients not controlled on GLP -1 based therapies

+ Evaluate icovamenib added to GLP-1 based Therapies in T2D
+ First Patient Enrollment Panned in 1Q 2026

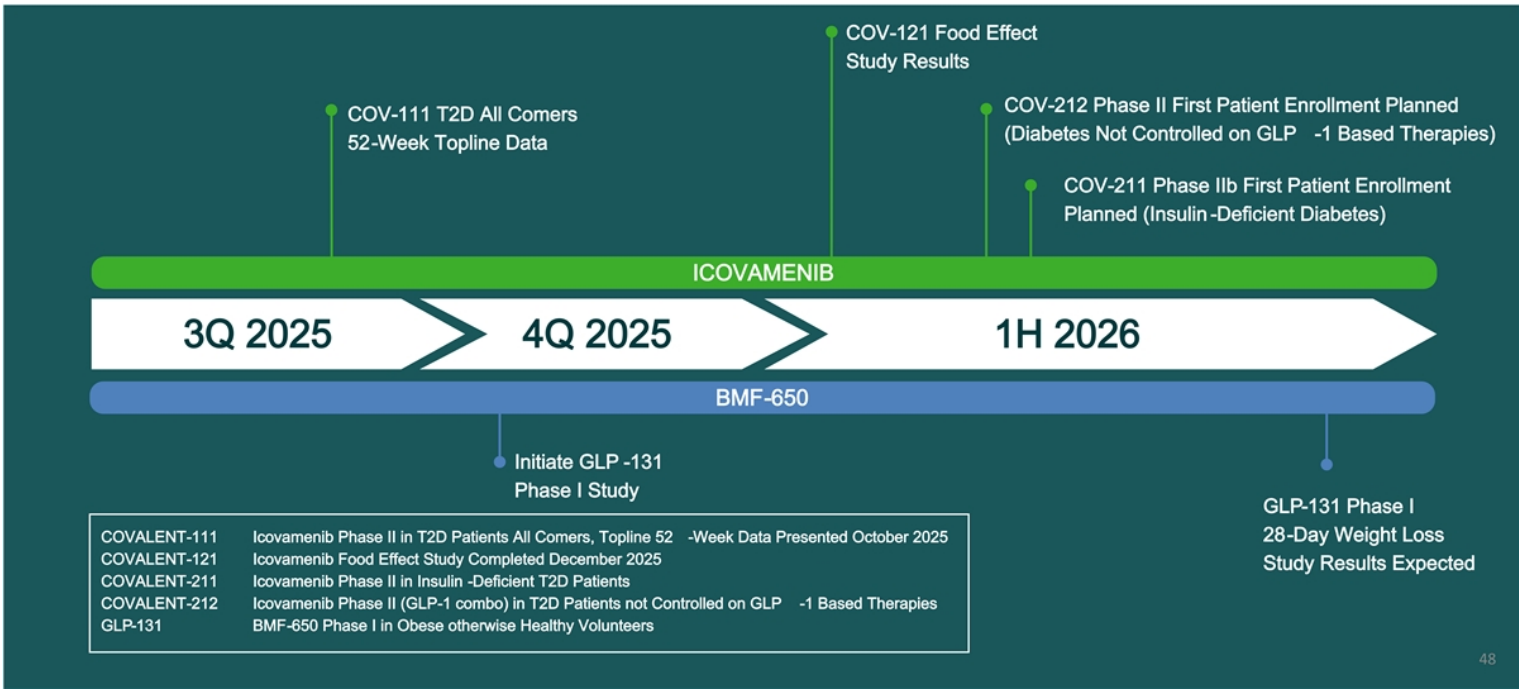
BMF-650

GLP-131

Phase I Obesity Study

+ Evaluate Safety of BMF-650 and 28-Day Weight Reduction in Obese Patients
+ Study Enrollment Completion Expected 2Q 2026

Timeline Overview of Key Program Activities



COVALENT-111	Icovamenib Phase II in T2D Patients All Comers, Topline 52 -Week Data Presented October 2025
COVALENT-121	Icovamenib Food Effect Study Completed December 2025
COVALENT-211	Icovamenib Phase II in Insulin -Deficient T2D Patients
COVALENT-212	Icovamenib Phase II (GLP-1 combo) in T2D Patients not Controlled on GLP -1 Based Therapies
GLP-131	BMF-650 Phase I in Obese otherwise Healthy Volunteers

Key Opinion Leader 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 Leader 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


THANK YOU
For questions or inquiries, please reach out to
Meichiel Weiss at ir@biomeafusion.com
www.biomeafusion.com
biomea
FUSION