Biomea Fusion Corporate Presentation

Q2 2026

biomea FUSION™

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 funded through key clinical readouts for icovamenib and BMF-650 into 1Q of 2027

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

ICOVAMENIB

Potential first-in-class oral small molecule targeting menin - the control switch to beta cell restoration
Restores functional beta-cell mass and improve endogenous insulin production in both type 1 and type 2 diabetes

Critical unmet need: In **type 1 diabetes**, there are no approved therapies that directly restore or preserve beta-cell function, and patients rely on lifelong insulin therapy.[1] In **type 2 diabetes**, approximately 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.[2-4] In **obesity** and overweight individuals, current therapies have substantially improved weight loss, but may also result in loss of lean muscle mass, raising concerns about preservation of physical function and quality of life.[5]

BMF-650

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

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

1. www.breakthrought1d.org Scherer et al., Scientific Reports, 2020; 2. Nichols et al., Diabetes Care, 2015; 3.UKPDS Group. Lancet, 1998; 4. Prime Therapeutics & Magellan Rx Management, 2023 real-world claims analysis. 5. Neeland IJ, et al. Changes in lean body mass with glucagon-like peptide-1-based therapies and mitigation strategies. Diabetes Obes Metab. 2024.

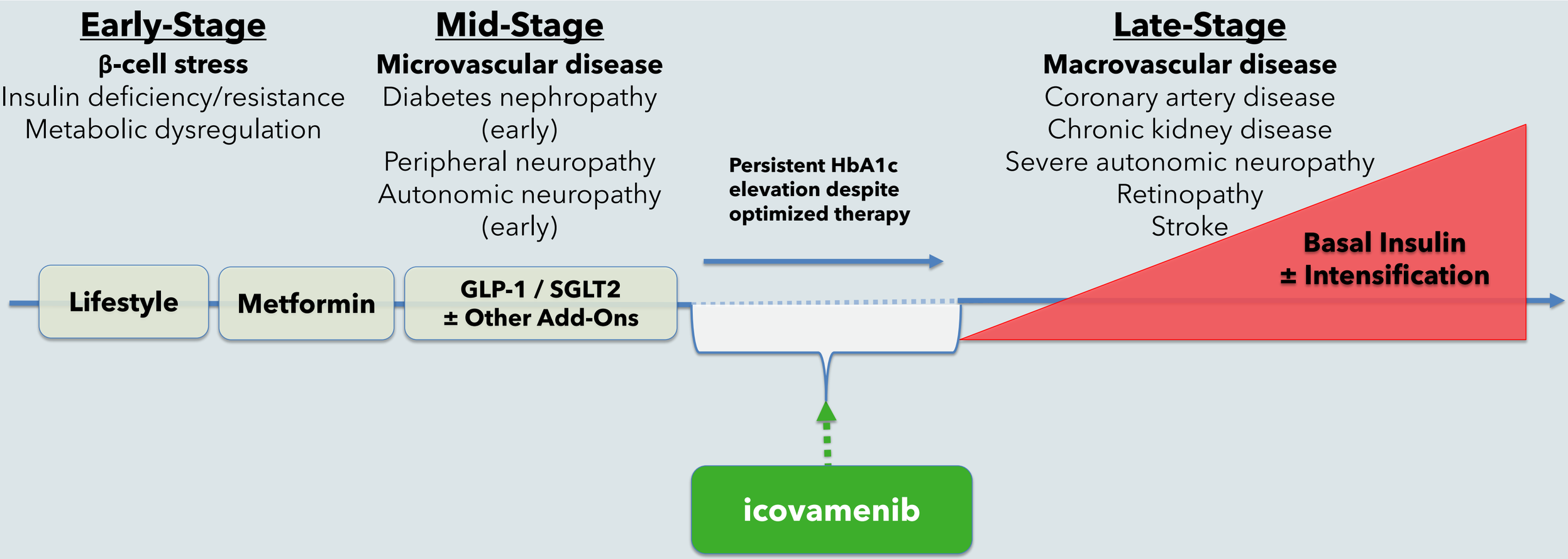
Biomea pipeline

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 2 diabetes** Patients with insulin deficiency *(~7M U.S. Patients)* [1]	**COVALENT-211** (study enrolling)			Phase II 26-week data (primary endpoint) anticipated **4Q 2026**
	Type 2 diabetes Patients not controlled on GLP-1 based therapies *(15-45% U.S. Patients on GLP-1RA)* [2,3]	**COVALENT-212** (study enrolling)			Phase II 26-week data (primary endpoint) anticipated **4Q 2026**
ICOVAMENIB with low dose Semaglutide	**Obesity/Overweight** *(>190M U.S. Patients)* [5] Sponsored by Leicester Diabetes Center	**OPAL Study**			Phase II initiation anticipated in **2H 2026**
BMF-650 Potential best-in-class oral GLP-1 RA	**Obesity** *(>100M U.S. Patients)* [5]	**GLP-131** (study enrolling)			Phase I 28-day weight reduction data anticipated in **3Q 2026**

1.International Diabetes Federation. IDF Diabetes Atlas www.diabetesatlas.org (Based on company calculations)
2.NHANES analyses of glycemic control among U.S. adults with diabetes (JAMA; Diabetes Care);
3.SUSTAIN, AWARD, and SURPASS clinical trial programs for GLP-1 receptor agonists
4.Mayer-Davis et al., NEJM / CDC updates
5.National Center for Health Statistics August 2023. Accessed June 10, 2026

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



In the U.S., >50% of patients with diabetes remain above HbA1c targets ≥7%[1]
Depending on the GLP-1 RA agent, 15-45% do not achieve HbA1c < 7% in clinical trials[2]

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

Diabetes patients are poorly controlled with 1:3 U.S. diabetes patients estimated to require insulin

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

80%

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

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

12-14 years

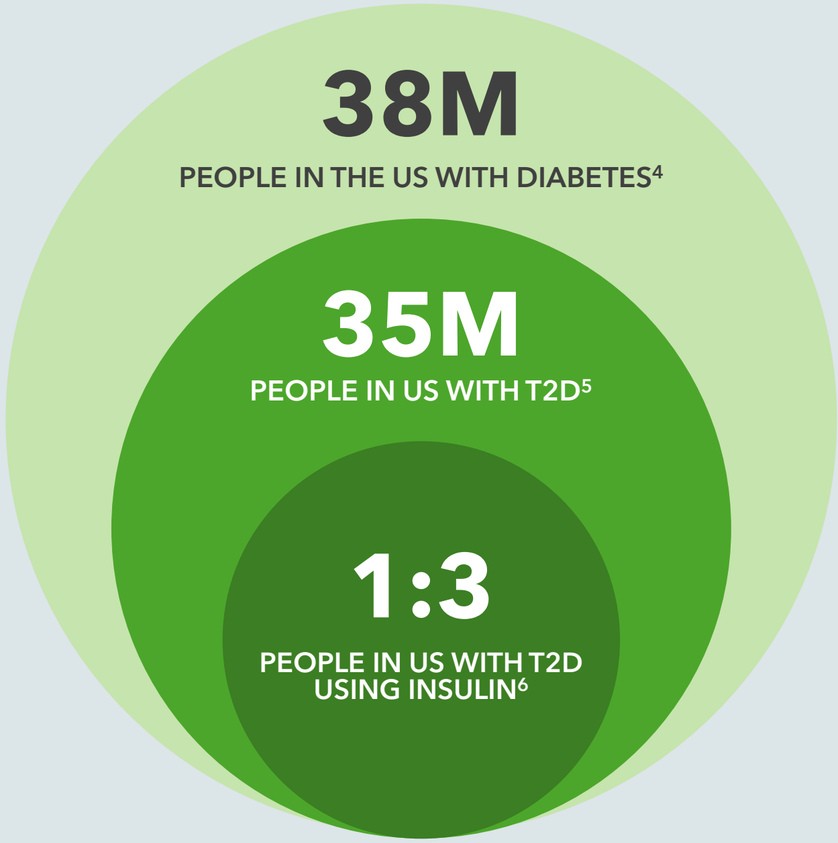
of life lost from diabetes[2]

Diabetes today remains poorly controlled in 50% of patients treated with standard of care agents[3] The burden to the healthcare system is immense.

60+

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

No current therapy restores beta-cell function



1.Tabish Int J Health Sci. 2007 Jul;1(2):V–VIII
2.National library of Medicine 1(2); 2007 Jul PMC3068646
3.Zohu Lancet 2024; 404:2077–93
4. CDC, Natl. Diabetes Stat. Rep., 2022
5. ADA, Standards of Care in Diabetes, Diabetes Care, 2024
6. Li J Diabetes Complications 2012;26(1):17-22

Type 1 diabetes at-a-glance

T1D is caused by autoimmune destruction of insulin -producing pancreatic islet beta cells

~50%

Annual loss of beta cell capacity[1]

Patients with symptomatic T1D (Stage 3) typically lose yearly ~50% of their beta cell capacity over the first 7 years

~9.5M

People live with T1D globally in 2025[2]

~1.8M in the U.S.[3]

~513K

New diagnoses T1D per year globally in 2025[2]

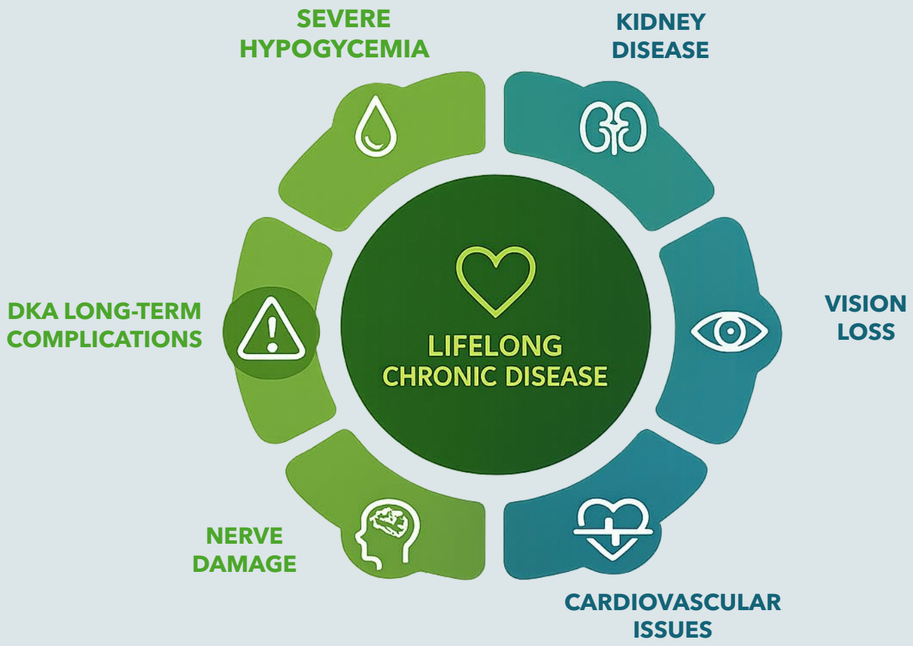
~64K new diagnoses/year in the U.S.[4]

0

Approved therapies for Stage 3[5]

There are no approved therapies other than exogenous insulin that address the dysglycemia associated with the progressive decline of C-peptide in Stage 3

T1D is considered a lifelong chronic disease and carries substantial acute risk



1. *Diabetes Care. 2018 Jun 7;41(7):1486–1492*
2. *Ogle, et al. Diabetes Research and Clinical Practice 2025, 225, 112277*
3. *Centers for Disease Control and Prevention. National Diabetes Statistics Report, 2023*
4. *Mayer-Davis et al., NEJM / CDC updates*
5. *Front. Endocrinol., 05 November 2024*
6. *American Diabetes Association. Standards of Care in Diabetes–2025*

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

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

42%

Of U.S. adults have obesity[1]

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

50-70%

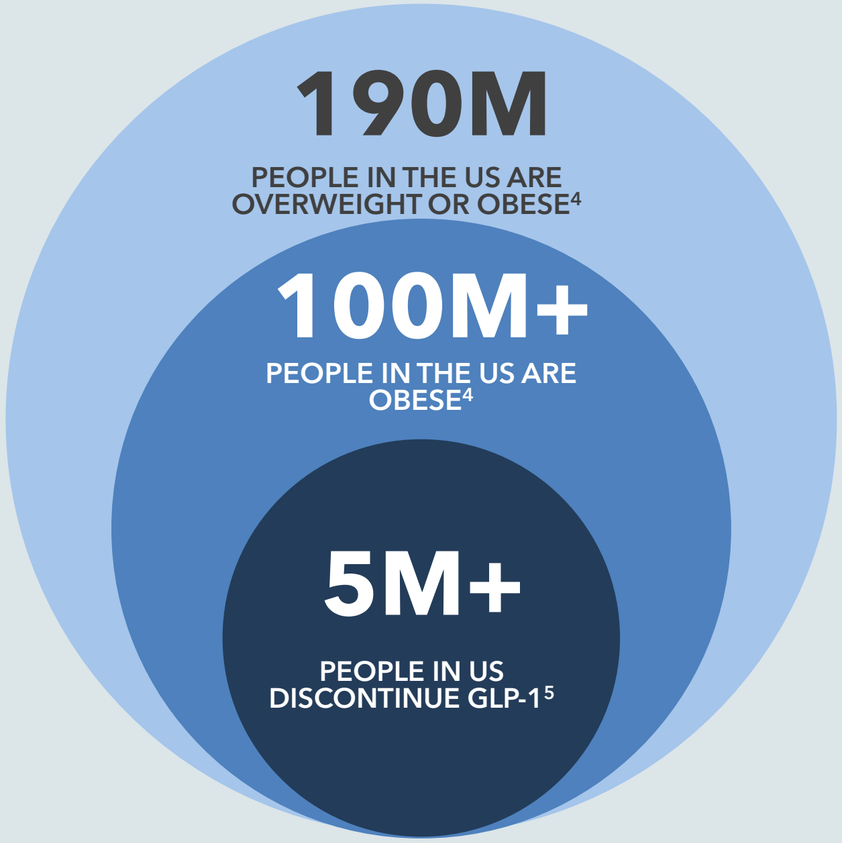
Of patients discontinue GLP-1 therapy within 12 months[2]

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

>60%

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

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



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

ICOVAMENIB

Potential first-in-class menin inhibitor for diabetes

Preclinical results

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Menin is naturally inhibited during pregnancy and breastfeeding

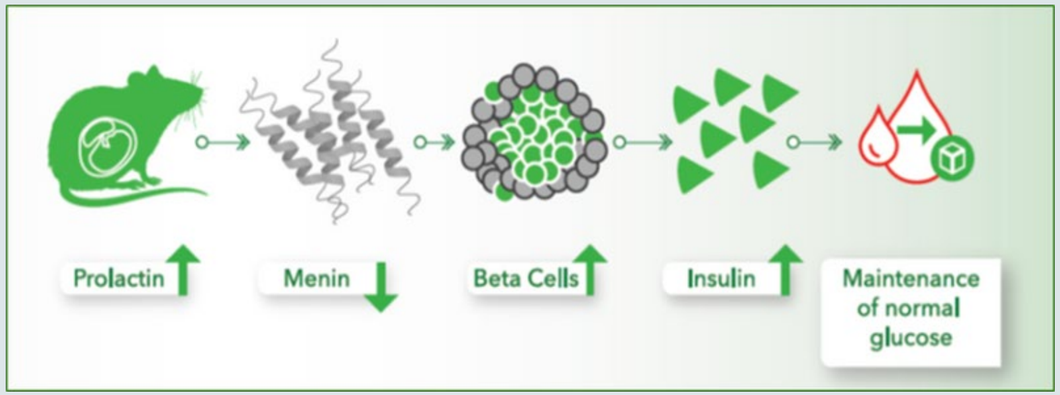
- allowing for adaptive beta cell mass increase & reduced diabetes risk

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

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

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

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



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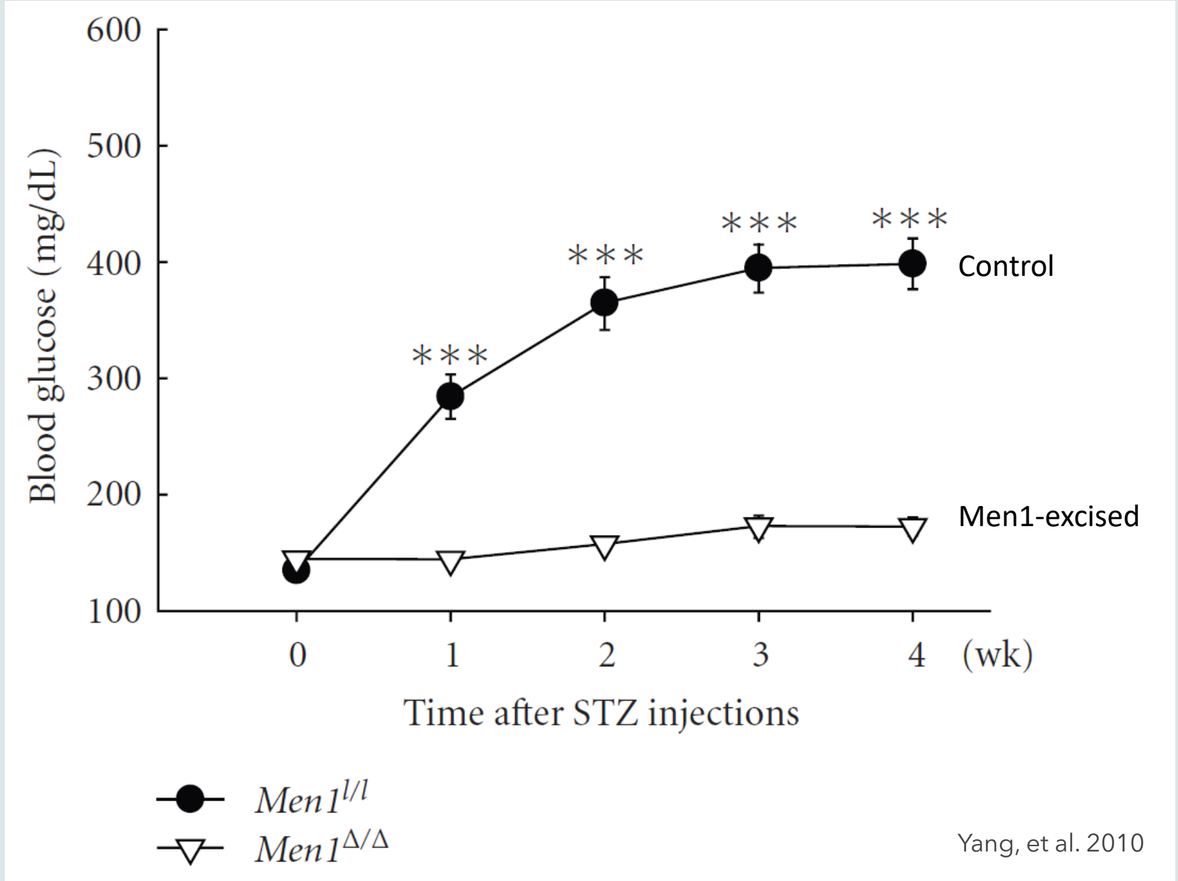
Potential for Menin Inhibition Demonstrated by Beta Cell Ablation Diabetes Model in MEN1-Excised Mice

MEN1 Excision Prevents Development of STZ-induced Hyperglycemia

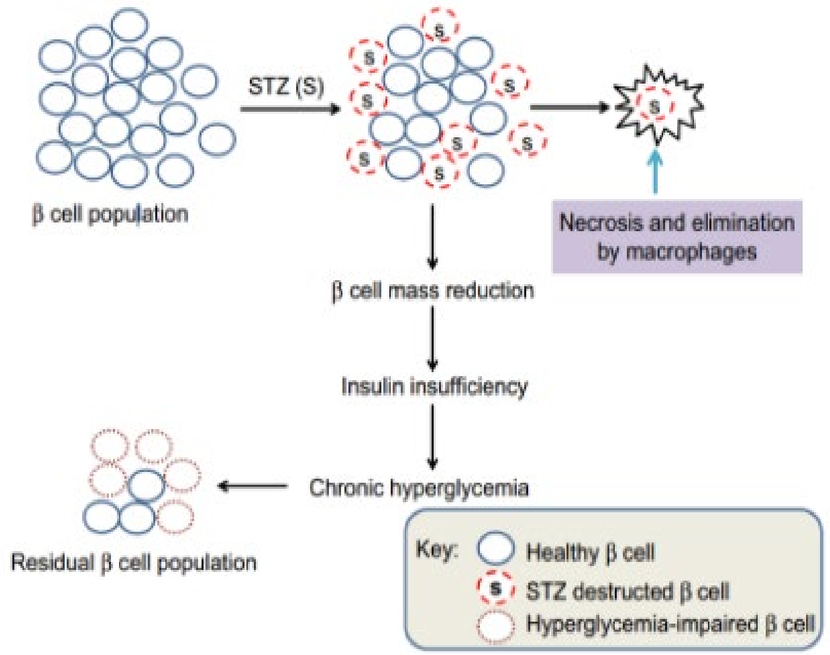
- Menin is a scaffold protein, encoded by the gene MEN1, that has been recognized for its role in Type 2 Diabetes Mellitus (T2DM) as a key regulator of beta-cell proliferation.
- Men1 knockout mice demonstrate increased beta-cell mass generation (Yang et al., 2010) and menin inhibition has previously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



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

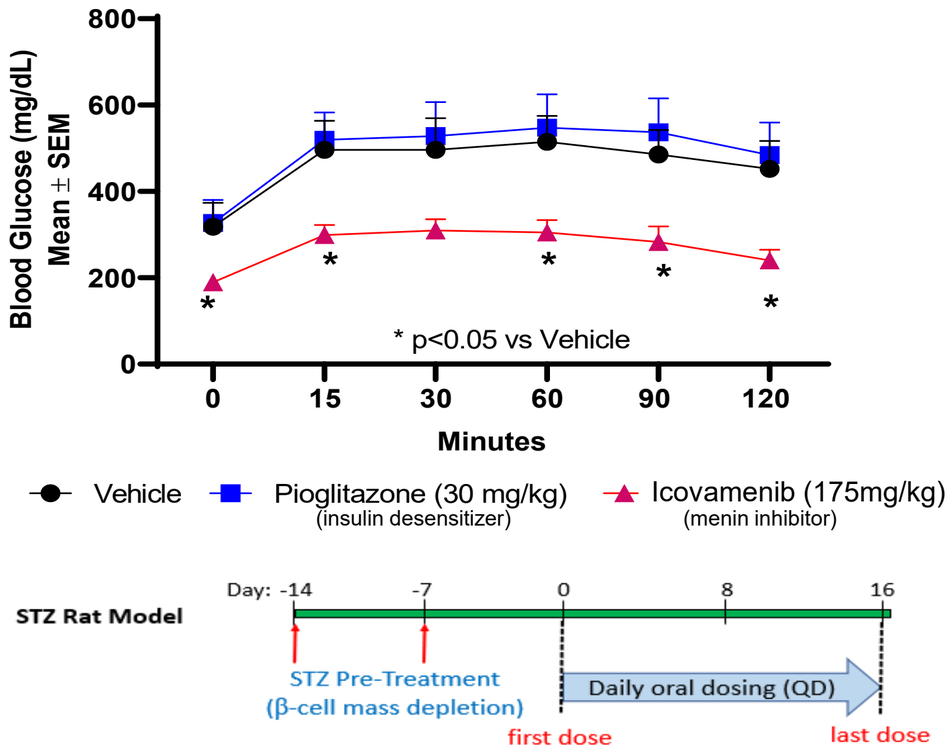
Icovamenib significantly reduced blood glucose in STZ rats (a model in which only insulin decreases blood glucose levels)

STZ TREATMENT TYPICALLY RESULTS IN ~50% BETA CELL LOSS



STZ=Streptozotocin, an antibiotic that produces pancreatic islet beta cell destruction and is widely used experimentally to produce a model in diabetes

ORAL GLUCOSE TOLERANCE TEST (DAY 17)

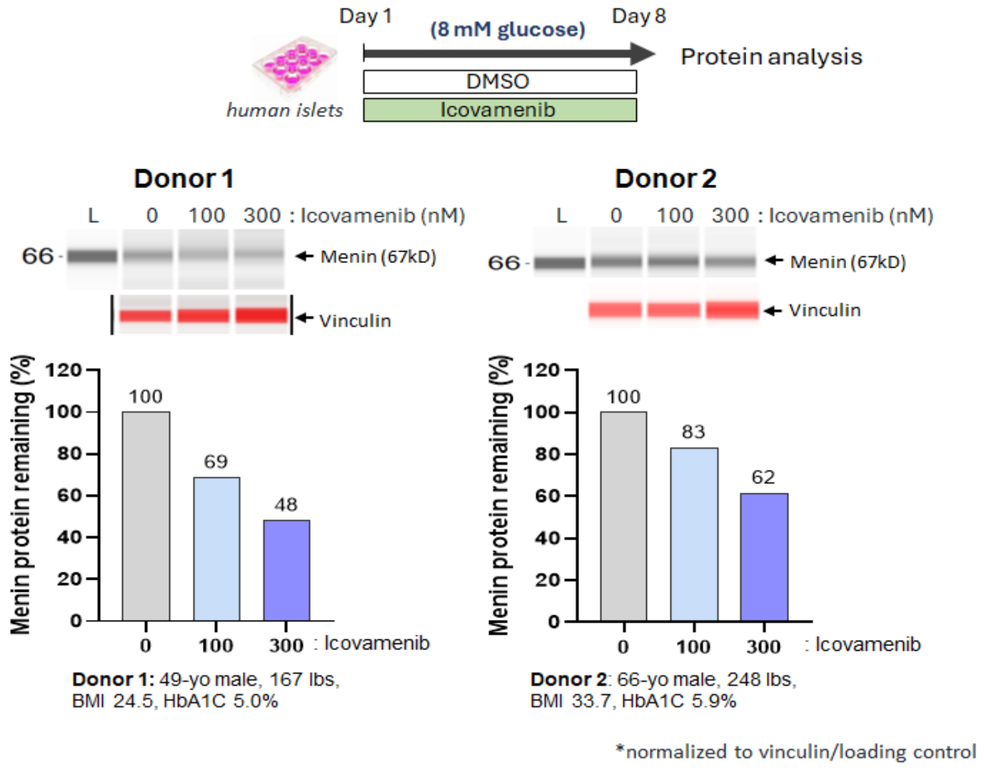


Butler, et al. (EASD) Diabetologia 65 (Suppl 1), 1–469 (2022) presentation #197

ICOVAMENIB | PRECLINICAL

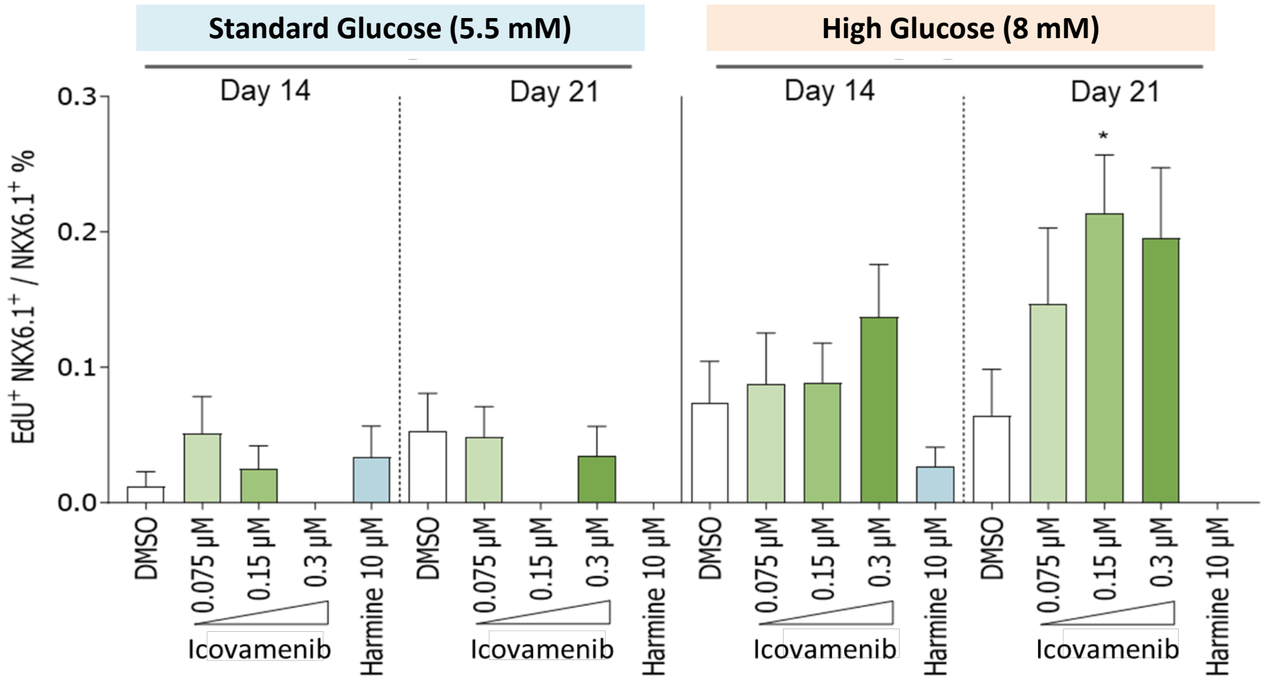
Icovamenib downregulated menin protein levels & promoted beta cell proliferation in ex vivo human islet cultures

MENIN LEVELS DOWNREGULATED



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

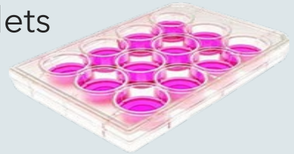
ICOVAMENIB CONDITIONALLY PROMOTED BETA CELL PROLIFERATION ONLY UNDER HYPERGLYCEMIC CONDITIONS



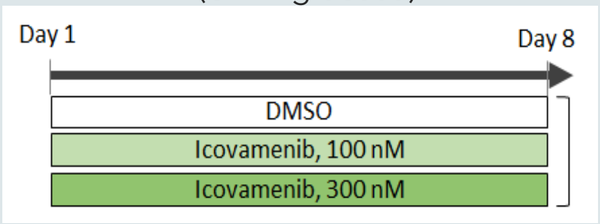
Frias, et al. (WCIRDC) Metabolism,Vol153, Supplement,2023,#88

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

Cadaver derived human islets

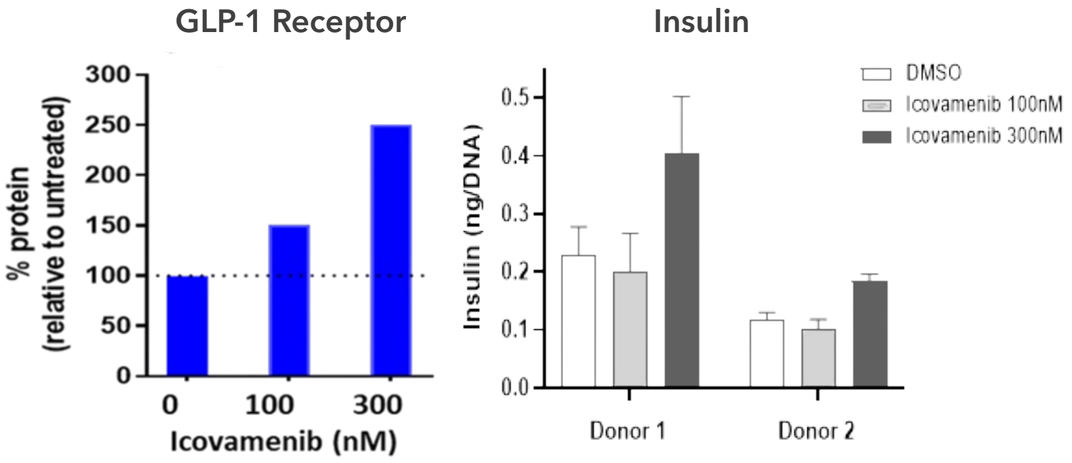


Culture 7 days under glucotox conditions (8mM glucose)



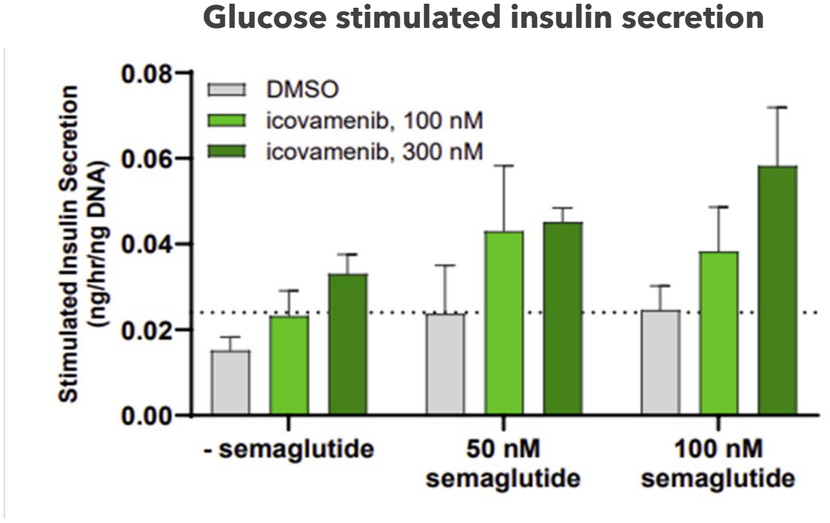
- Gene expression & Protein analysis
- Glucose Stimulated Insulin Secretion -/+ Semaglutide

ICOVAMENIB INCREASED GLP-1 RECEPTOR AND INSULIN PROTEIN LEVELS



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

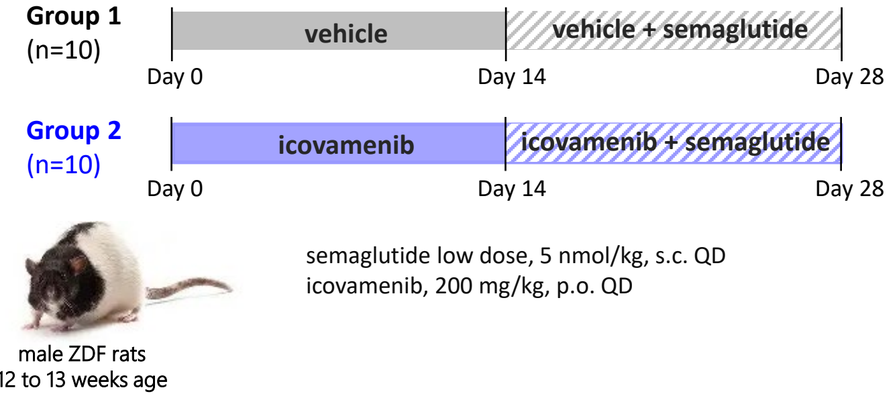
ICOVAMENIB ENHANCES THE RESPONSIVENESS OF HUMAN ISLETS TO GLP-1 RECEPTOR AGONISTS

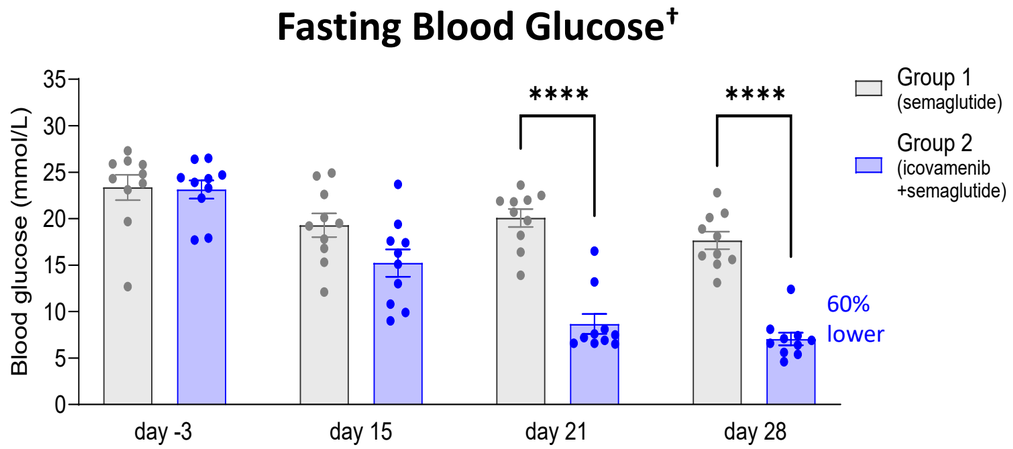


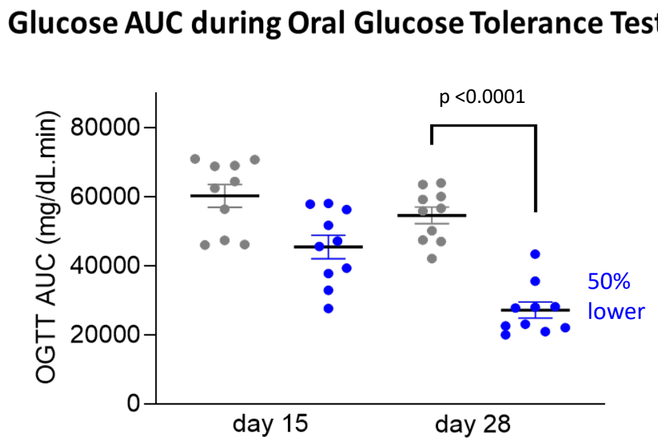
Somanath, et al. (EASD) Diabetologia 68 (Suppl 1), 1–754 (2025). Oral presentation #66

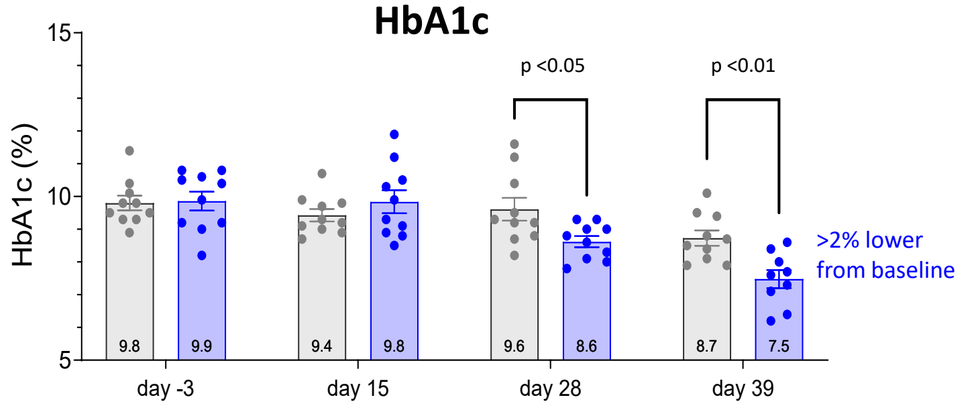
ICOVAMENIB WITH GLP-1RA

Combination treatment of icovamenib & low-dose semaglutide improved glycemic control in ZDF rats









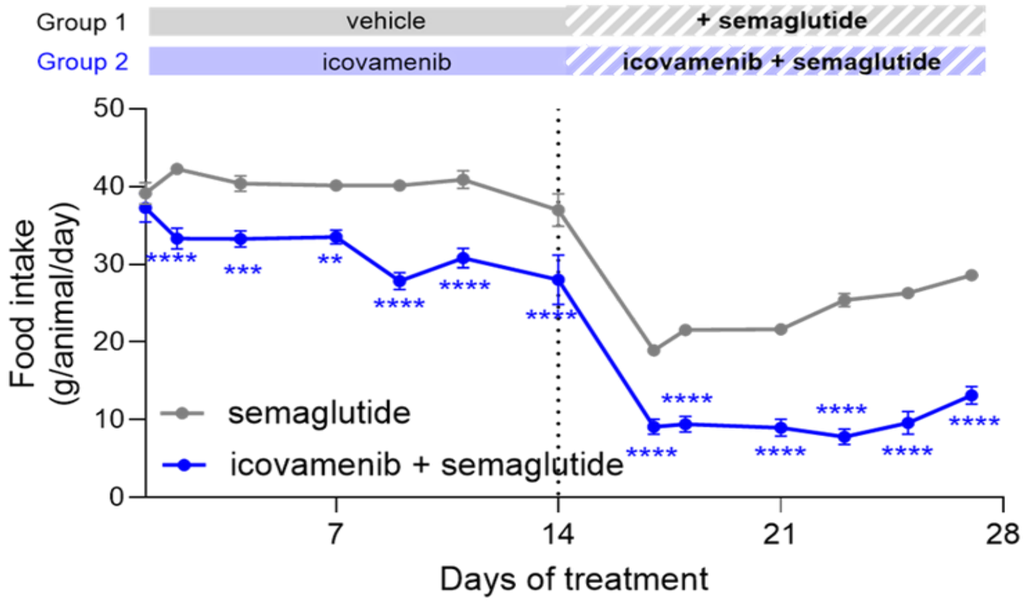
Plots represent mean ± SEM
Dots represent data for individual animal
† 6hr fasting on days -3 and 21, overnight fasting on days 15 and 28.

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

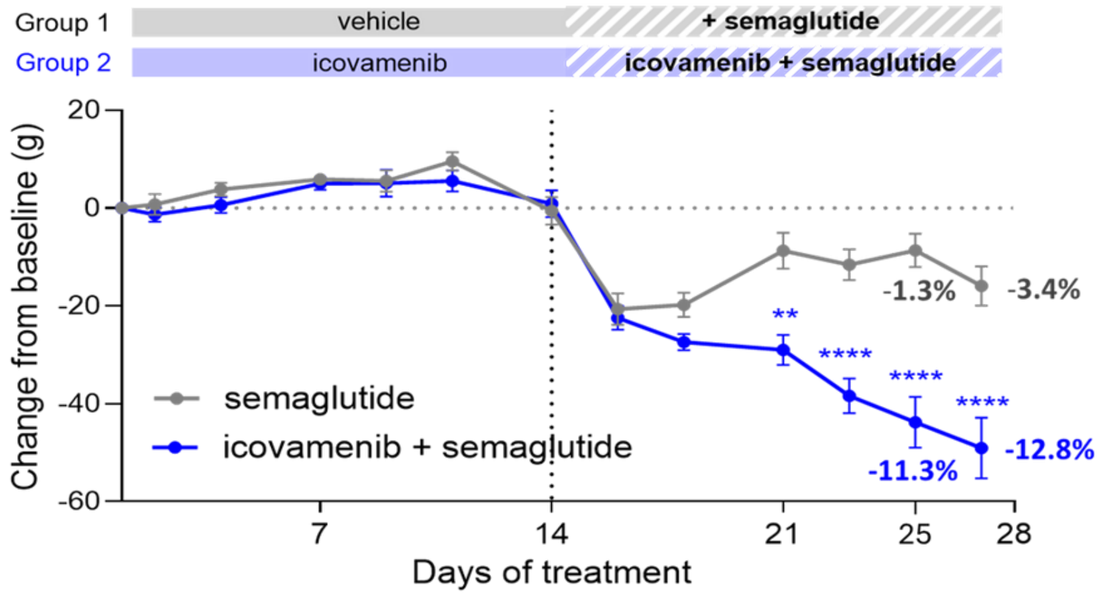
ICOVAMENIB WITH GLP-1RA

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

APPETITE SUPPRESSION



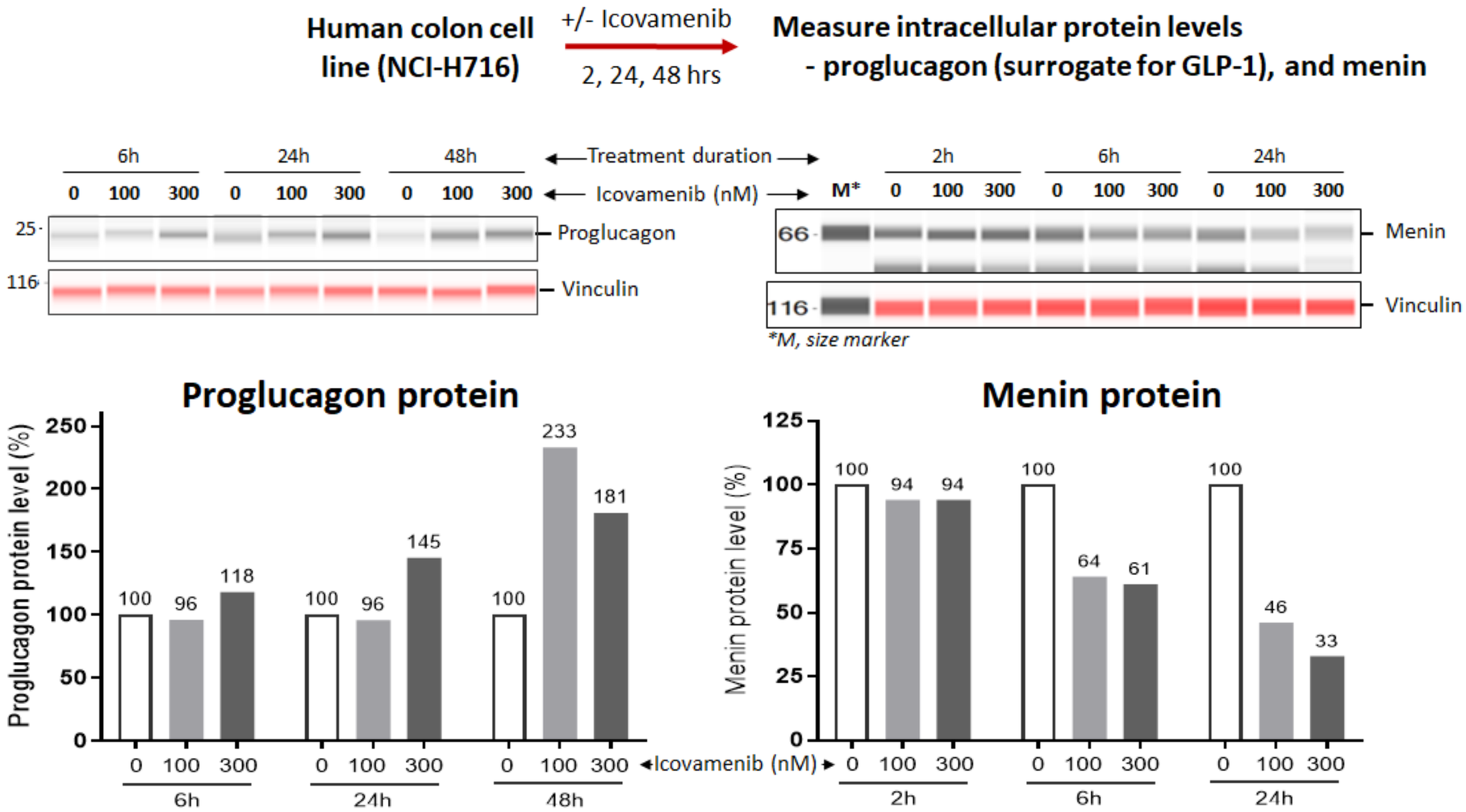
BODY WEIGHT REDUCTION



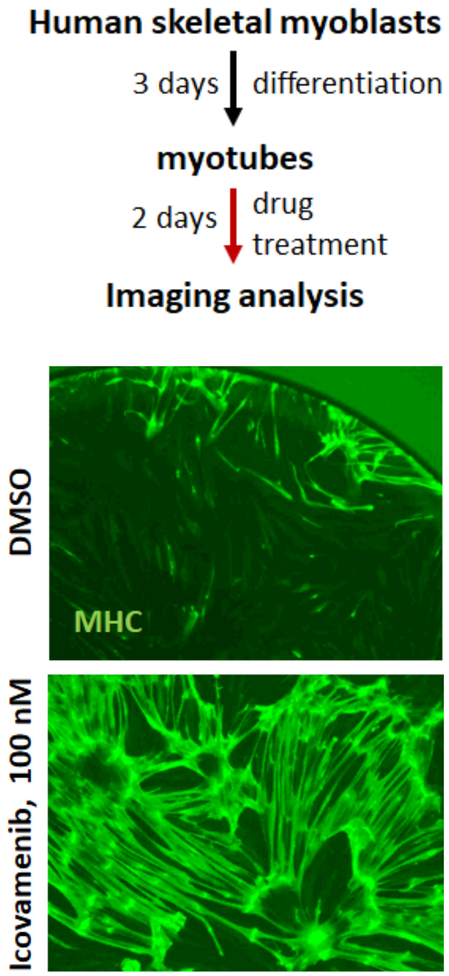
- ❑ SUPERIOR APPETITE SUPPRESSION WITH ABOUT 10% GREATER BODY WEIGHT REDUCTION THAN LOW-DOSE SEMAGLUTIDE ALONE
- ❑ THE OBSERVED BODY WEIGHT REDUCTION WAS PRIMARILY DUE TO FAT MASS LOSS WITH PRESERVATION OF LEAN MASS

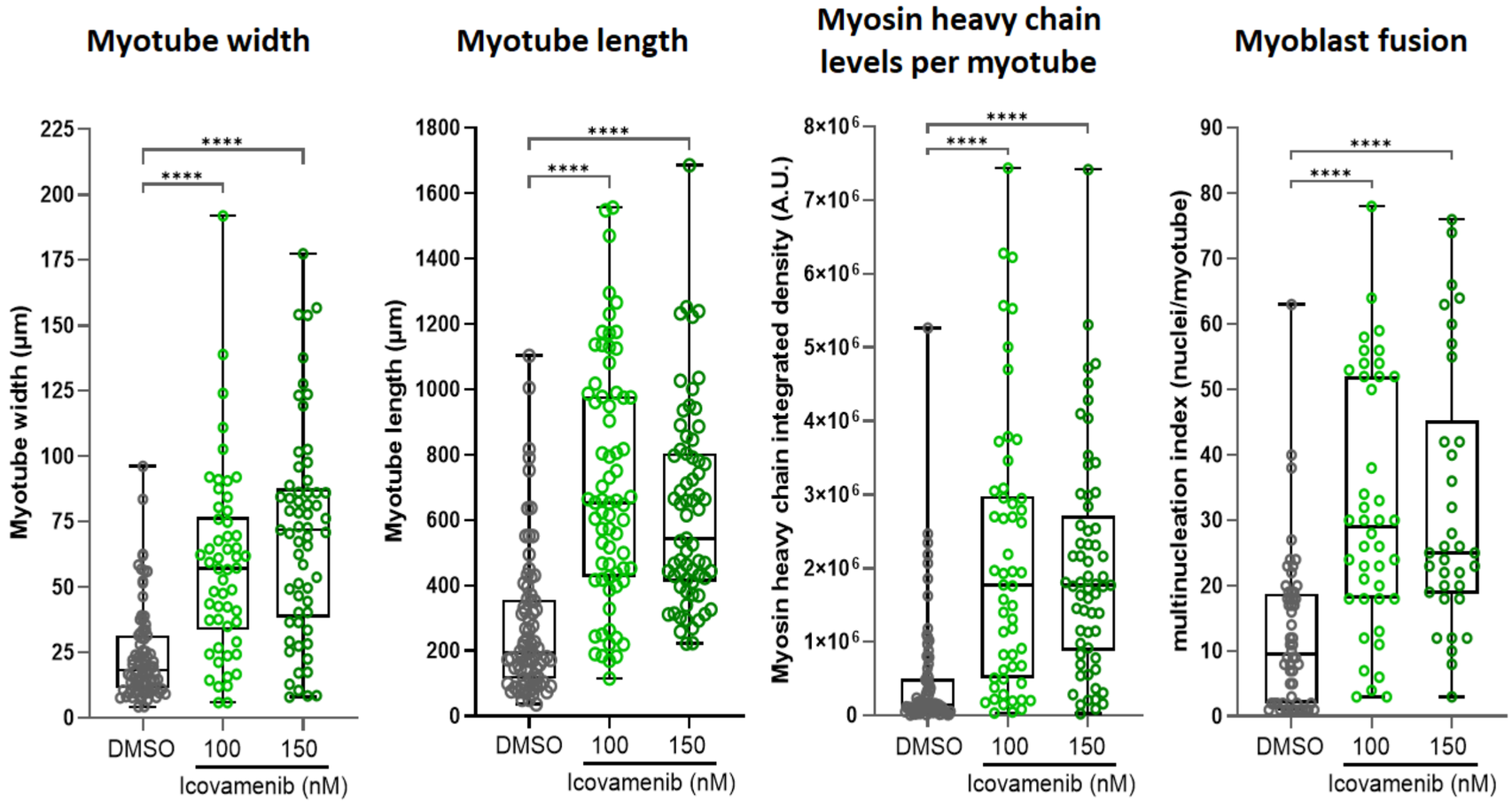
Somanath, et al. (ADA) Diabetes 2025;74 (Supplement_1):870-P

Icovamenib Enhanced Intracellular Proglucagon Expression in a Human Colon L-cell Model

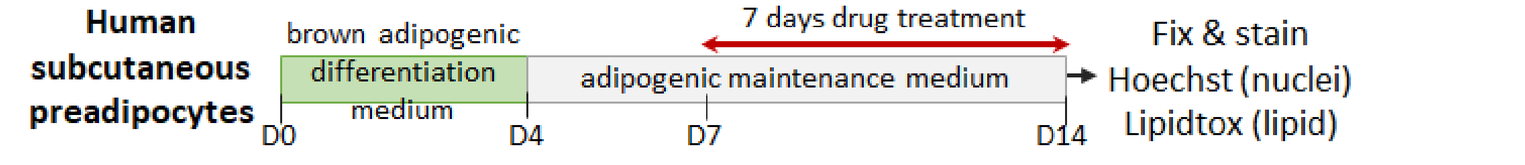


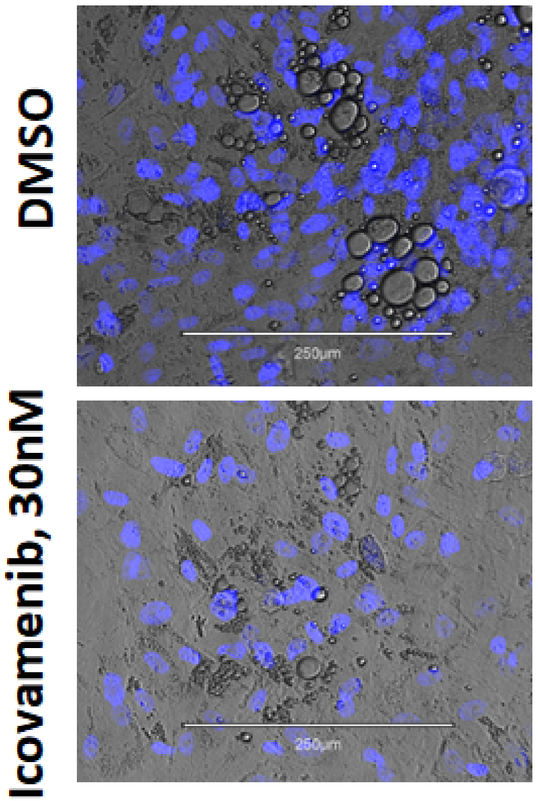
Icovamenib Induced Myogenic Effects in Human Skeletal Myoblast-derived Myotubes

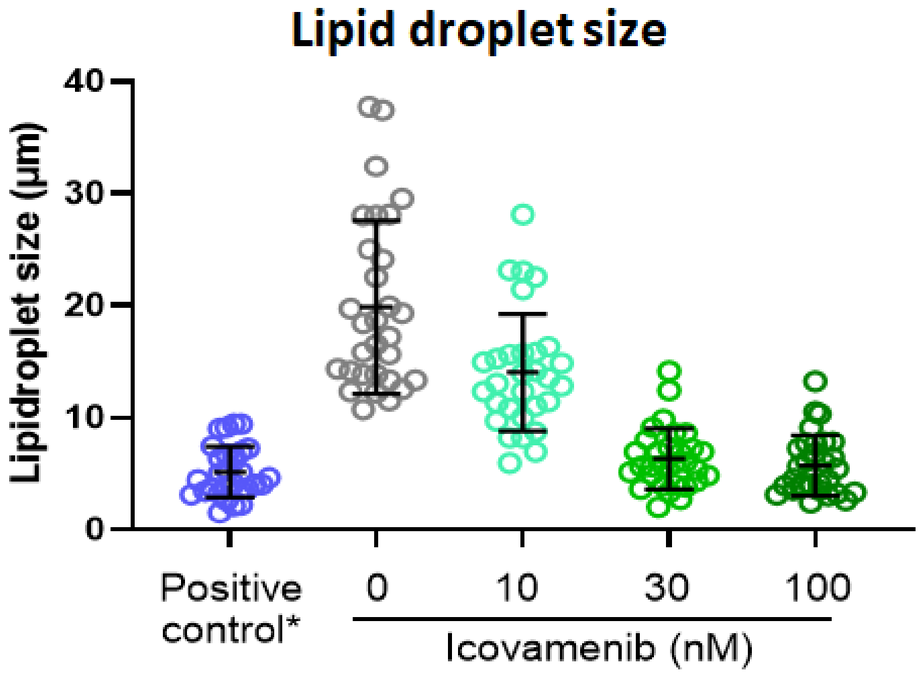




Icovamenib Promoted Lipolysis in Primary Human Adipocytes



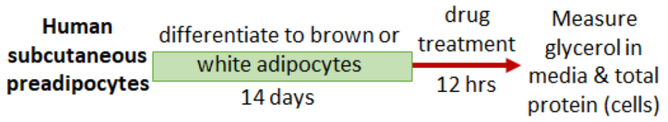


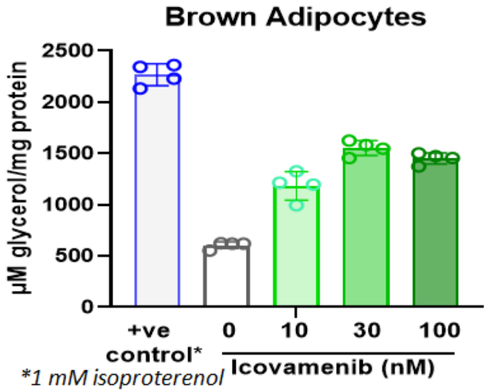


**differentiation media (D7 to D14)*

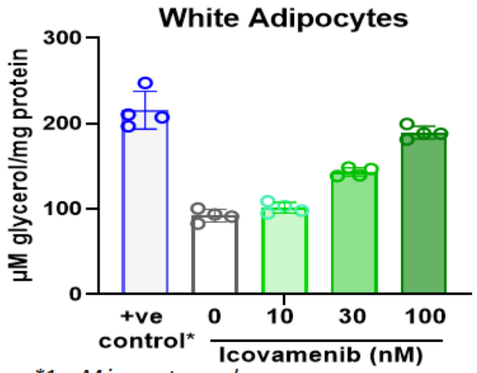
Icovamenib Promoted Effects Indicative of Lipolysis and Fat Browning in Primary Human Adipocytes

Enhanced Glycerol Release





*1 mM isoproterenol

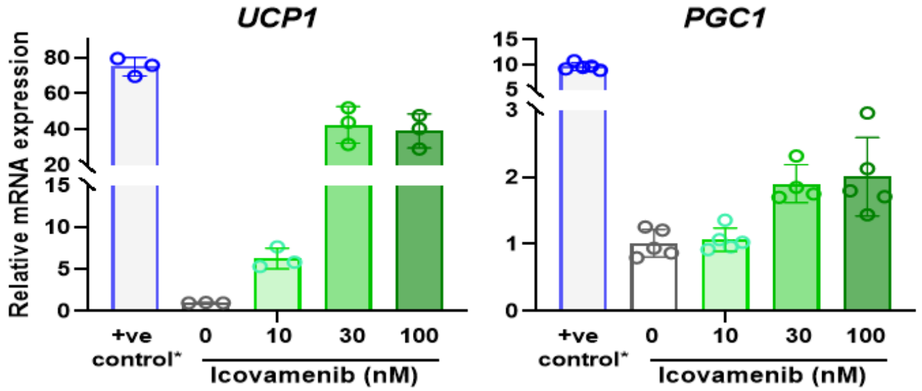


*1 mM isoproterenol

Upregulation of thermogenic and lipolytic genes

Human Brown Adipocytes

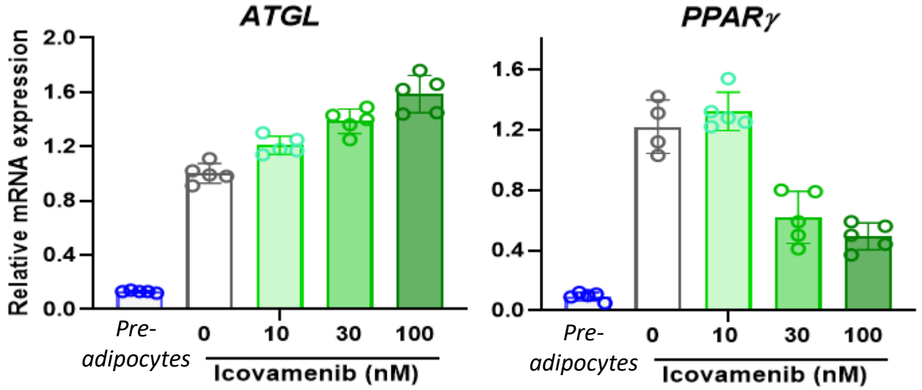
(*differentiated from primary subcutaneous preadipocytes*)



*differentiation media (D7 to D14)

Human White Adipocytes

(*differentiated from primary subcutaneous preadipocytes*)



PPIA (Cyclophilin A) was used as housekeeping gene for normalization of data

ICOVAMENIB | COVALENT-112

Potential first-in-class menin inhibitor for diabetes

Clinical results in Type1 Diabetes

biomea FUSION™

Treatment Landscape:
Limitations of current approaches in stage 3 T1D

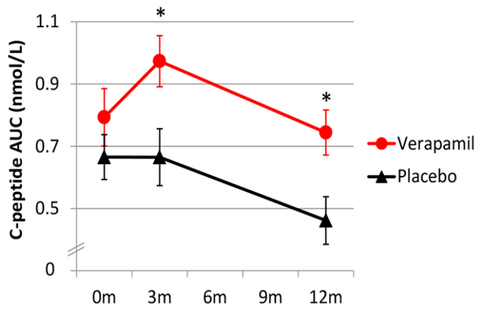
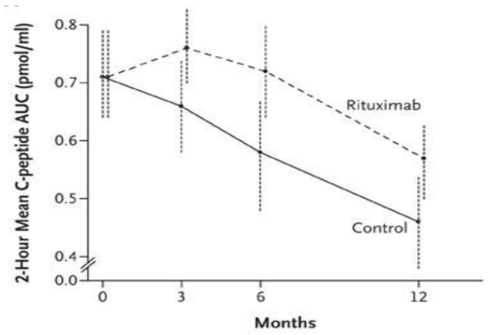
- Most investigational therapies in T1D focus on immune modulation to slow autoimmune destruction or on preserving residual beta cell function[1]
- C-peptide area under the curve (AUC) has become the accepted endpoint, driving enrollment early after diagnosis (<90 days, new-onset T1D) to preserve residual beta-cell function[2]
- To date, most investigational therapies have not demonstrated durable restoration of beta cell function or sustained increases in C-peptide, outside of cell-based transplantation approaches[3]

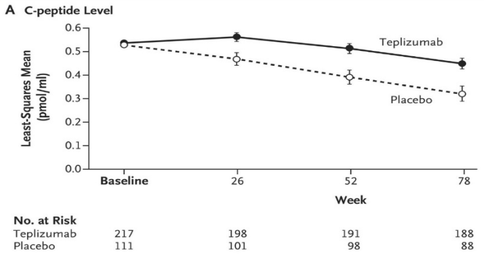
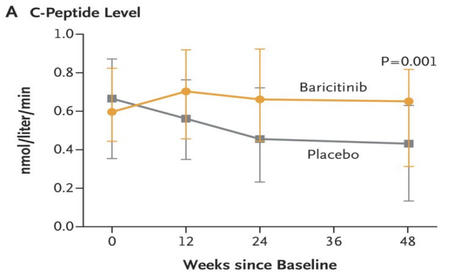
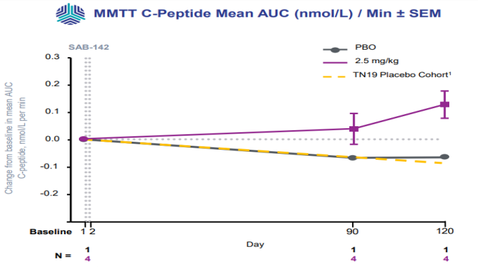
The Next Frontier:

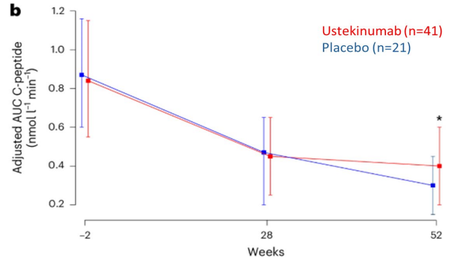
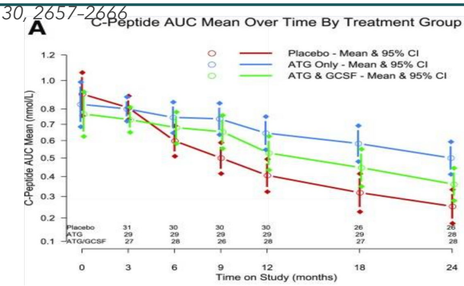
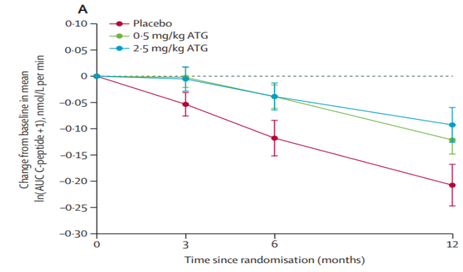
- Restoring beta cell function and mass, beyond only slowing the decline of C-peptide
- Expanding the treatment window beyond early, new onset T1D populations
- Enabling persistence of newly generated beta cells despite autoimmune pressure

1. *Zarei M et al. Diabetes Epidemiology and Management 2025;17*
2. *Diabetes Care 2025*
3. *NIDDK. Diabetes in America, 2024*

Most therapies in development for stage 3 T1D show limited and non-durable C-peptide impact

DRUG	Mean C-PEPTIDE AUC VS PLACEBO
Verapamil	 *Nature. 2018 Aug;24(8):1108-1112*
Rituximab	 *NEJM. 2009 361:2143–2152*

DRUG	Mean C-PEPTIDE AUC VS PLACEBO
Teplizumab (Tzield)	 *ESPE Yearbook of Paediatric Endocrinology (2024) 21 10.2*
Baricitinib	 *N Engl J Med 2023;389:2140-2150*
SAB-142	 *SAB Bio website corporate presentation (March 10, 2026) https://www.sab.bio/*

DRUG	Mean C-PEPTIDE AUC VS PLACEBO
Ustekinumab	 *Nature Medicine 2024 vol 30, 2657–2666*
ATG (TrialNet)	 *Diabetes Care 2018 Jul 16;41(9):1917–1925*
MELD-ATG	 *Lancet 2025 Sep 27;406(10510):1375-1388*

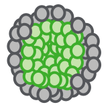
**Ladarixin and Diamyd,* both Immune modulating, not mentioned here as they demonstrated no meaningful difference compared to placebo

Icovamenib's mechanism of action

Icovamenib promotes selective and partial reduction in menin levels

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

Induction of beta cell proliferation



Pancreatic islet

IMPROVED BETA CELL MASS & FUNCTION FOR TYPE 1 AND TYPE 2 DIABETES

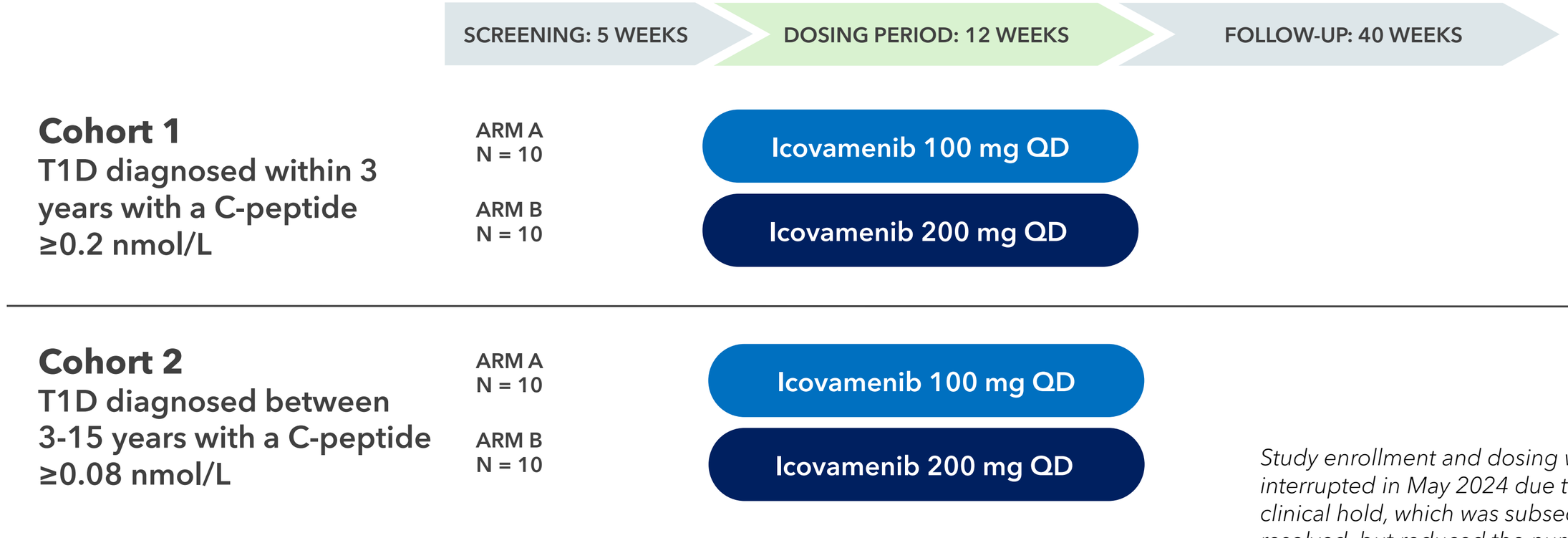
- Increase in insulin/C-peptide
- Decrease in blood glucose (HbA1c)

IMPROVED INCRETIN EFFECTS

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

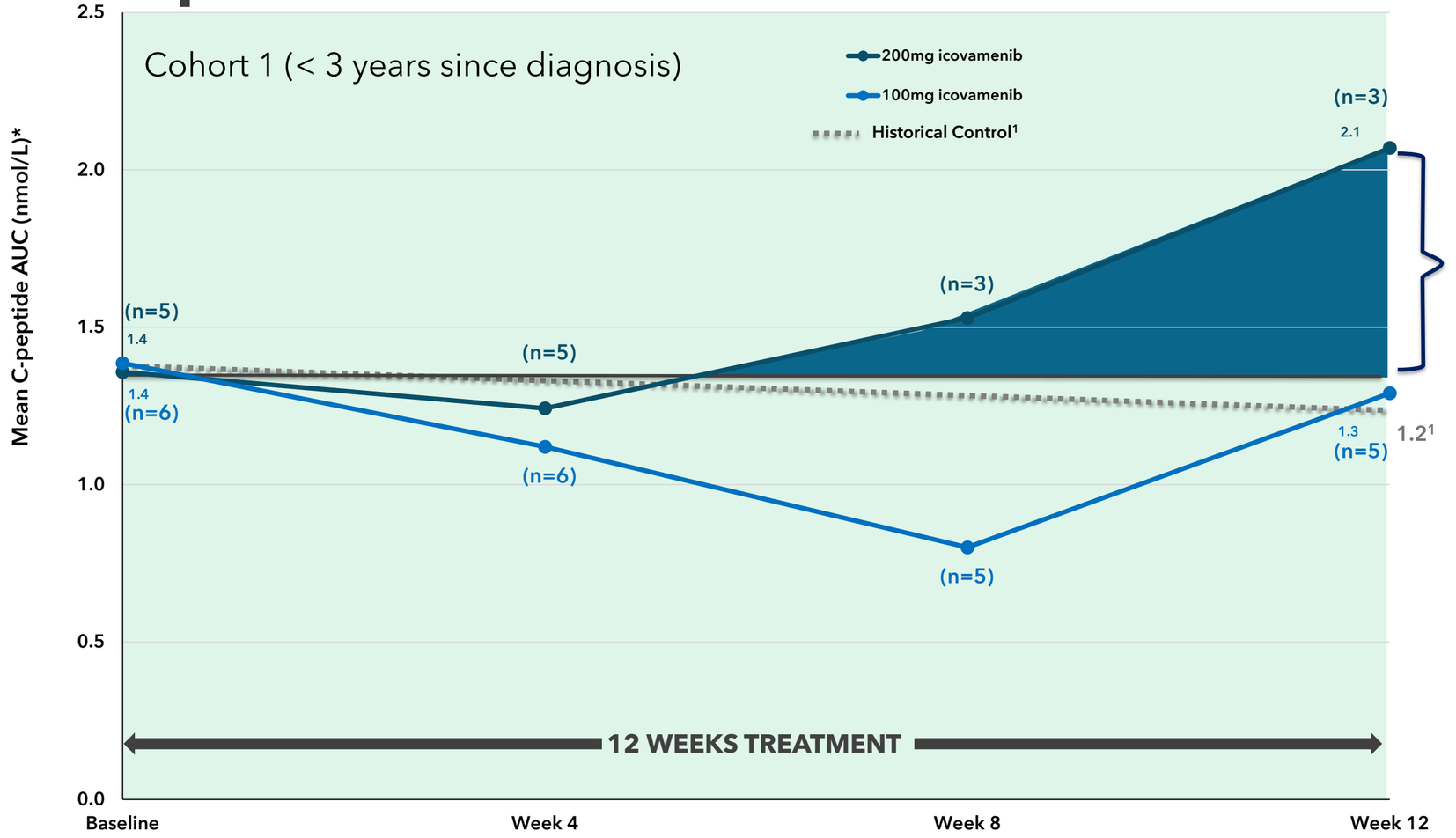
Study Design

COVALENT-112 (NCT06152042) was a Phase 2 trial designed to examine beta cell function (as measured by C-peptide change and the change of exogenous insulin usage) and glucose and lipid metabolism in participants with T1D treated with standard of care insulin and icovamenib.



Study enrollment and dosing were interrupted in May 2024 due to an FDA clinical hold, which was subsequently resolved, but reduced the number of patients enrolled and followed through to the 52-week readout.

52% mean increase in C-peptide during the 12 weeks treatment period of icovamenib



52% mean increase from baseline

+0.7
P<0.001

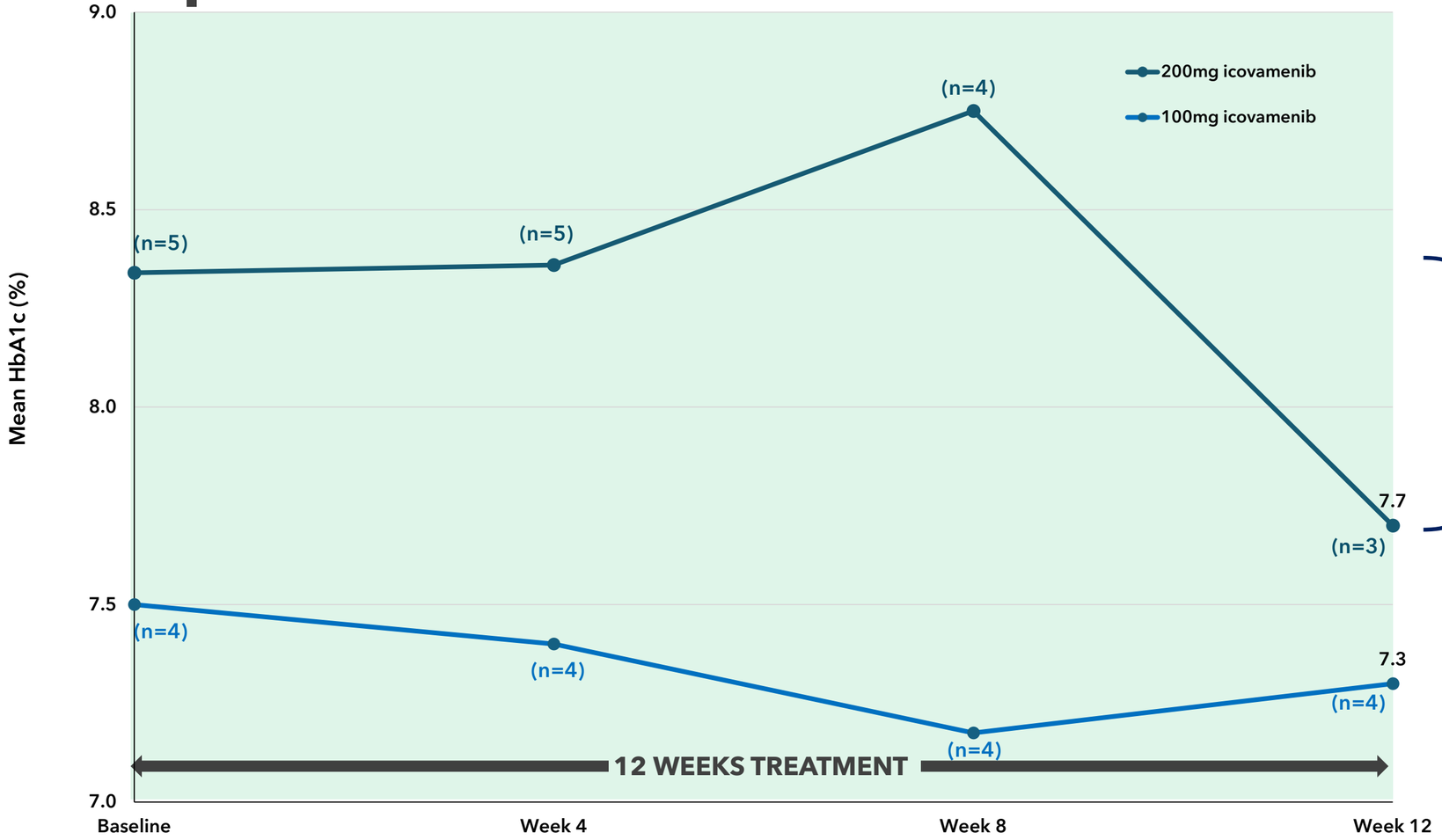
Data represents post-hoc analysis of patients who received per statistical analysis plan, 80% of planned doses

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

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

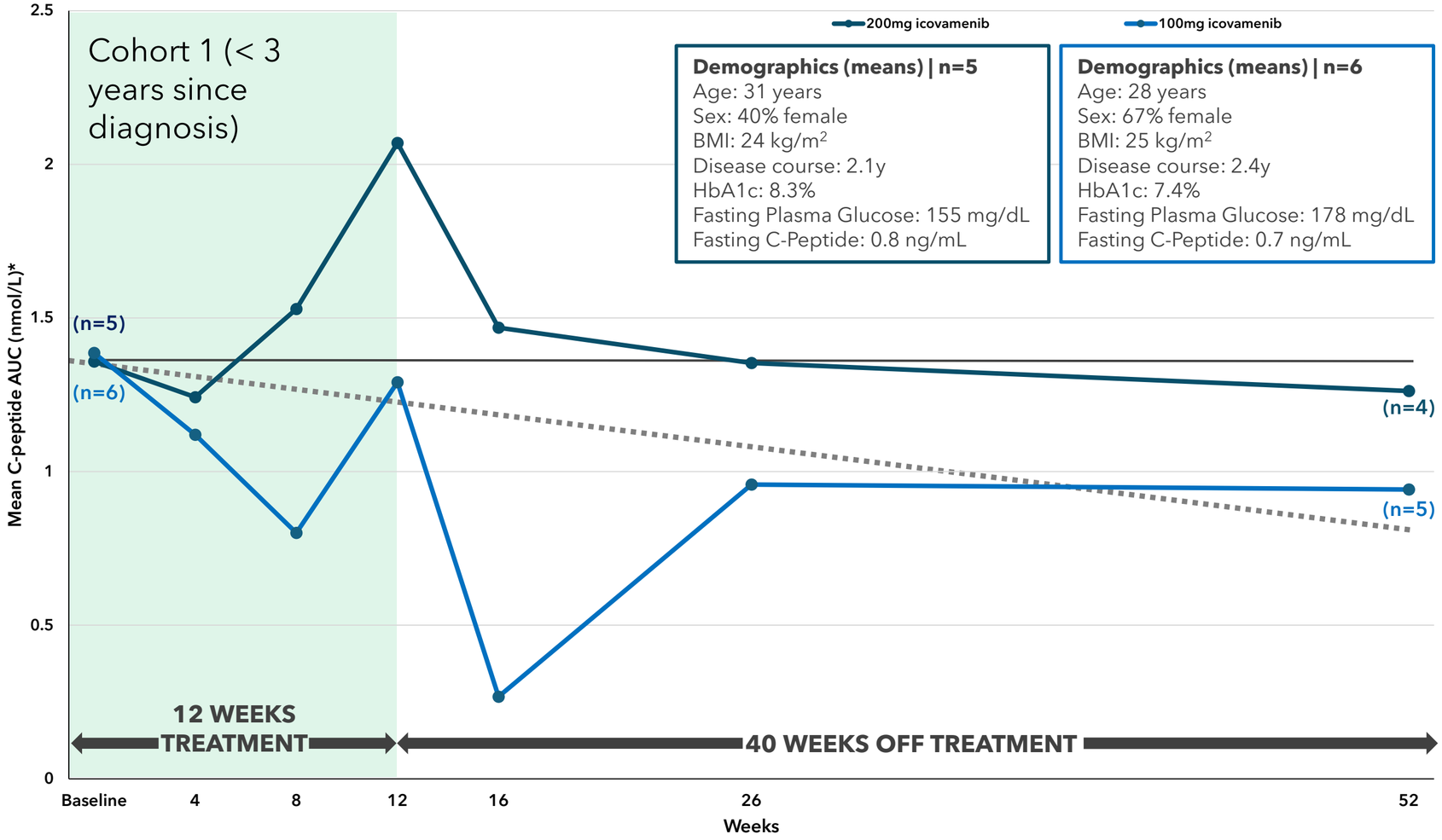
ICOVAMENIB | COVALENT-112

0.6% mean reduction in HbA1c during the 12 weeks treatment period of icovamenib



0.6% reduction in mean HbA1c from baseline

Baseline C-peptide levels sustained through week 52 with minimal decline (only -7.1%) observed post 12 weeks of 200mg daily icovamenib



Change in mean C-peptide AUC at Week 52 vs baseline

-7%

-32%

-47%[1]

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

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

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

Cytokine Profiling of Cohort 1 (<3 year diagnosis) participants receiving 200mg icovamenib

	Week 12			Week 26			Week 52		
	pg/mL	change	status	pg/mL	change	status	pg/mL	change	status
Il-1β	1.07	0.40	Non-Inflammatory	1.00	0.27	Non-Inflammatory	0.70	-0.27	Non-Inflammatory
IL-2	3.43	-0.83	Non-Inflammatory	3.57	-1.20	Non-Inflammatory	1.20	-3.23	Non-Inflammatory
IL-6	3.27	0.70	Non-Inflammatory	0.53	-1.70	Non-Inflammatory	0.53	-1.70	Non-Inflammatory
IL-8	6.57	0.77	Non-Inflammatory	7.17	-0.07	Non-Inflammatory	5.37	-1.18	Non-Inflammatory
IL-10	1.30	-0.03	Non-Inflammatory	1.33	0.00	Non-Inflammatory	1.30	-0.03	Non-Inflammatory
IFN-γ	-	-	Non-Inflammatory	-	-	Non-Inflammatory	-	-	Non-Inflammatory
TNF-α	7.53	-0.17	Non-Inflammatory	7.73	-1.35	Non-Inflammatory	4.07	-5.33	Non-Inflammatory

Mean values were assessed for all patients for each cytokine (n=3) relative to baseline.

- All cytokines remained classified as Non-Inflammatory from baseline through Week 52, indicating no evidence of systemic immune activation in participants receiving 200 mg icovamenib
- Small Week 12 increases in IL-1β, IL-6, and IL-8 were transient and not associated with increases in IL-2 or TNF-α
- By Week 52, most pro-inflammatory cytokines were stable or decreased from baseline
- **The increase in C-peptide was not accompanied by a measurable systemic inflammatory cytokine response but rather led to a stabilization and mild reduction of inflammatory markers over time**

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Favorable 52-week safety profile

	Cohort 1			Cohort 2		
	Arm A 100 mg QD (N = 8)	Arm B 200 mg QD (N = 9)	Cohort 1 Total (N = 17)	Arm A 100 mg QD (N = 9)	Arm B 200 mg QD (N = 10)	Cohort 2 Total (N = 19)
Patients with ≥1 TEAE, N (%)	3 (38)	0 (0)	3 (18)	1 (11)	3 (30)	4 (21)
Treatment-Related SAEs, N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
SAEs*, N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Treatment Discontinuation due to TEAE, N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Study Discontinuation due to TEAE, N (%)	0	0	0	0	0	0
Deaths, N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Diarrhea, N (%)	1 (13)	0	1 (6)	1 (11)	0	1 (5)
Nausea, N (%)	1 (13)	1 (11)	2 (12)	1 (11)	1 (10)	2 (11)
Hyperglycemia, N (%)	0	0	0	0	1 (10)	1 (5)
Headache, N (%)	1 (13)	0	1 (6)	0	1 (10)	1 (5)
AST/ALT increase, N (%)	3 (38	2 (22)	5 (29)	1 (11)	7 (70)	8 (42)
Resolution of ALT/AST w/o interruption in study treatment, %	100	100	100	100	80	90

Optimal dose pool and target population identified for T1D phase 2 program

T1D insights:

- ✓ Dose response: 200 mg demonstrated stronger clinical activity vs 100 mg
- ✓ Potential early intervention advantage: Patients with T1D dosed ≤3 years showed greater response vs those 3-15 years since diagnosis
- ✓ 12-week treatment showed continuous and improved responses, supporting potential for greater benefit with extended dosing
- ✓ Cytokine profiling showed no evidence of systemic immune activation, with inflammatory markers remaining stable or reduced through Week 52
- ✓ Preclinical chronic toxicology studies support longer term dosing
- ✓ Generally well-tolerated, with a favorable safety profile maintained through the 52-week observation period

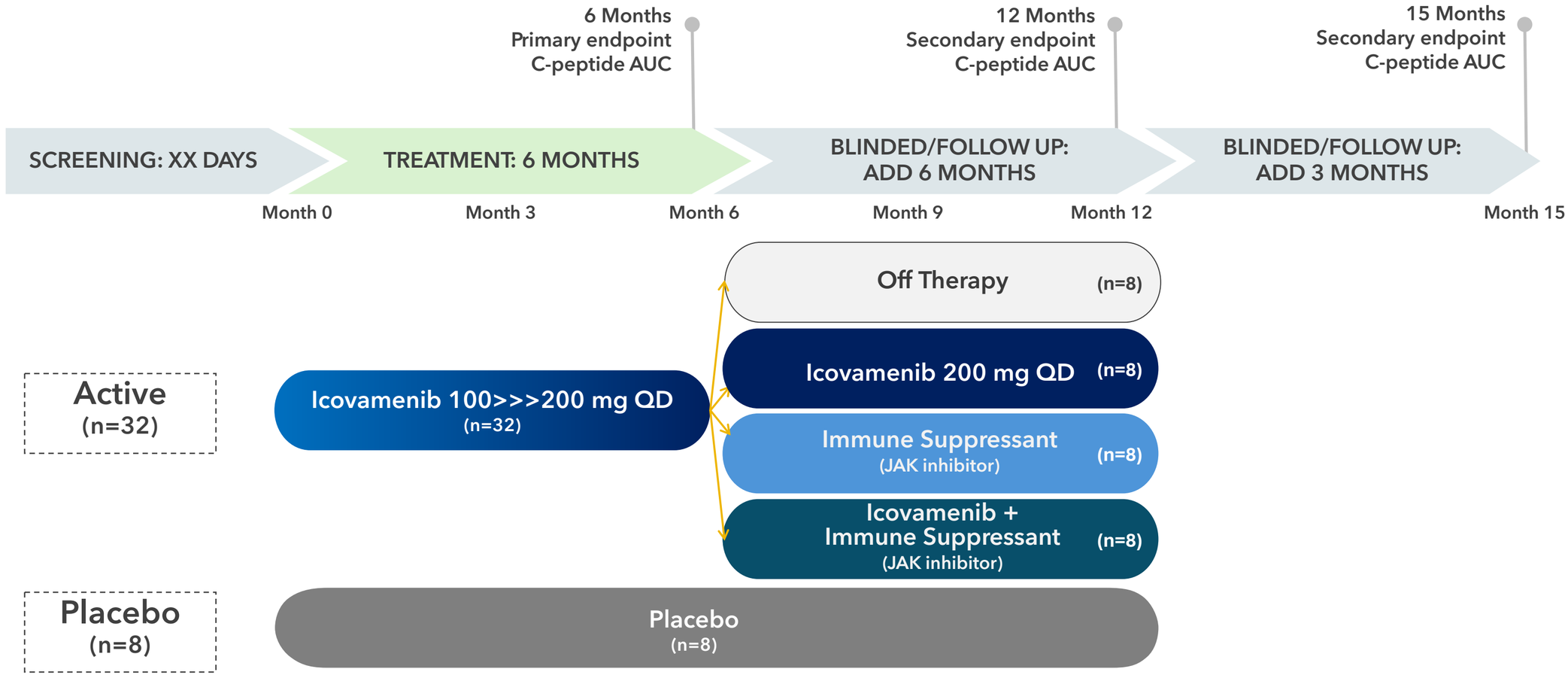
T1D development focus:

- Potential to further improve C-peptide AUC in T1D with extended or continuous dosing
- Opportunity to potentially enhance outcomes through combination with immunomodulation therapies

Proposed phase 2 trial design*

Inclusion Criteria

- Adult participants with T1D diagnosed within 3 years with a C-peptide ≥0.2 nmol/L
- Background therapy maintained unless rescue required



*Subject to regulatory and investigator alignment, and feedback from applicable health authorities.

Study investigators *

Primary Investigator

Peter Gottlieb, MD

Professor of Pediatrics and Medicine | Barbara Davis Center, University of Colorado

- Professor of Pediatrics and Medicine and holds the Orr Family Endowed Chair in Adult Diabetes at the Barbara Davis Center, one of the world's leading centers for Type 1 diabetes research and clinical care.
- Long-standing member of Type 1 Diabetes TrialNet and chairs its Collaborative Mechanistic Studies Panel, helping drive pivotal immunotherapy trials across all stages of T1D.
- With 190+ publications, his work focuses on T- and B-cell–driven autoimmunity.

Anschutz | Barbara Davis Center for Diabetes

Sub-Investigators

Jason Gaglia, MD, MMSc

Ass. Professor | Joslin Diabetes Center, Harvard Medical School

Worlds leading diabetes center (36,000 patients annually, 150 MDs/PhDs)

Joslin Diabetes

David Baidal, MD

Ass. Professor | Diabetes Research Institute, University of Miami Miller School of Medicine

DRI Diabetes Research Institute FOUNDATION

Ralph A. DeFronzo, MD

Professor and Chief of the Diabetes Division | UT Health of San Antonio

UT Health San Antonio Long School of Medicine

**Subject to regulatory and investigator alignment, and feedback from applicable health authorities.*

KOL perspectives across clinical significance, biology, and future development in T1D

Efforts to intervene against type 1 diabetes (T1D) have historically focused on preserving remaining insulin secretion in people just diagnosed with T1D. These icovamenib data are unique in showing increased C-peptide-reflected insulin secretion in patients with established T1D during dosing and persistence of this effect after treatment was stopped. In people with established T1D, endogenous insulin secretion progressively declines to very low levels. Any evidence of improvement in endogenous insulin secretion—even among a few T1D individuals—is unprecedented and of immense biologic and clinical significance. These findings warrant rigorous and longer-term evaluation.

G. Alexander "Zan" Fleming, MD

FOUNDER & EXECUTIVE CHAIRMAN, KINEXUM
FORMER FDA SENIOR MEDICAL OFFICER AND DIVISION LEADER FOR METABOLIC & ENDOCRINE DRUGS, INVOLVED IN THE REVIEW OF LANDMARK DIABETES AND METABOLIC THERAPIES INCLUDING METFORMIN, THE FIRST RAPID-ACTING INSULIN ANALOGS, EARLY STATINS, AND PPAR AGONISTS

The new data presented today with icovamenib in patients with type 1 diabetes suggest a potential new therapeutic avenue in a disease where fundamental unmet need has long persisted. To date, approved therapies have not directly addressed the progressive loss of functional beta cells that underlies diabetes. Biomea has made critical progress in identifying and characterizing this molecule, which has demonstrated the ability to reduce menin protein levels and activate pathways associated with beta cell function. Today's icovamenib type 1 data further validates and deepens our understanding of icovamenib's mechanism of action. Congratulations to the Biomea team on reaching this important therapeutic milestone.

Rohit Kulkarni, MD, PhD

PROFESSOR OF MEDICINE, HARVARD MEDICAL SCHOOL | SENIOR INVESTIGATOR & SECTION CO-HEAD (ISLET CELL & REGENERATIVE BIOLOGY), JOSLIN DIABETES CENTER

What stands out to me in the icovamenib diabetes data is not only the emerging signal of biological activity, but also the safety profile observed to date with using icovamenib in diabetes studies. That combination is important, because safety ultimately determines whether rational combination strategies can be explored as the program moves forward. Looking ahead, future studies will be critical in determining whether the improvements observed in beta cell function of these Type 1 diabetes patients can be maintained over time, particularly in the presence of ongoing immune activity. It will also be important to understand whether combination approaches—including immunomodulatory therapies— are needed and can further enhance or stabilize the observed effects. These are key questions that will inform the long term clinical potential of this approach.

David Baidal, MD

ASSISTANT PROFESSOR DIABETES RESEARCH INSTITUTE, UNIVERSITY OF MIAMI MILLER SCHOOL OF MEDICINE

KOL perspectives across clinical significance, biology, and future development in T1D

"The icovamenib data in Type 1 diabetes naturally makes us pause and reflect on what it could ultimately mean for people living with Type 1 diabetes. While these early findings require confirmation, they suggest a different way of thinking about treatment, one that extends beyond glucose management and begins to engage underlying disease biology. For younger individuals in particular, the possibility of preserving or improving endogenous beta cell function over time could have meaningful implications for lifelong disease burden.
Results like these invite consideration of how the treatment landscape in Type 1 diabetes may evolve if such approaches prove durable and safe.

Alice Cheng, MD

ENDOCRINOLOGIST, ASSOCIATE PROFESSOR OF MEDICINE UNIVERSITY OF TORONTO

"The icovamenib data in type 1 diabetes are encouraging, this is particularly interesting as icovamenib targets a pathway that has not been meaningfully explored in this disease. Despite advances in insulin delivery and glucose monitoring, disease-modifying options remain limited for patients. These findings support the need for focused, proof-of-concept studies in well-characterized patient populations to better understand this signal, its durability, and the underlying biology.
An important next step will be examining the interplay between beta cell effects and the autoimmunity inherent in type 1 diabetes, and whether combination approaches with immunomodulatory therapies could further enhance or stabilize these beta cell effects.

Jason Gaglia, MD, MMSc

ASSISTANT PROFESSOR OF MEDICINE, HARVARD MEDICAL SCHOOL | STAFF PHYSICIAN, JOSLIN DIABETES CENTER – ONE OF THE WORLD'S LEADING DIABETES CENTERS

"While insulin therapy is life saving for people with Type 1 diabetes, chronic exogenous insulin use is not without consequence. Over time, it is associated with well recognized iatrogenic risks, including hypoglycemia, diabetic ketoacidosis, weight gain, lipohypertrophy, and increased cardiovascular burden. Targeting menin with icovamenib represents a fundamentally new therapeutic approach in diabetes. Rather than simply replacing insulin, it seeks to improve endogenous beta cell function. The early results we are seeing in Type 1 diabetes are highly encouraging.
I am excited to explore longer dosing periods to fully assess the potential of enabling patients to regain their own beta cell–mediated glucose control which is something no current therapy has been able to achieve. If this approach is confirmed, this could represent a meaningful step towards allowing patients to live their daily lives with greater physiological stability and far less constant fear of their disease.

Ralph DeFronzo, MD

ENDOCRINOLOGIST, PROFESSOR OF MEDICINE UTHSCSA

ICOVAMENIB | COVALENT-111

Potential first-in-class menin inhibitor for diabetes

Clinical results in Type 2 Diabetes

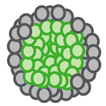
biomea FUSION™

Icovamenib's mechanism of action

Icovamenib promotes selective and partial reduction in menin levels

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

Induction of beta cell proliferation



Pancreatic islet

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

IMPROVED INCRETIN EFFECTS

- Synergistic with GLP-1 based therapies
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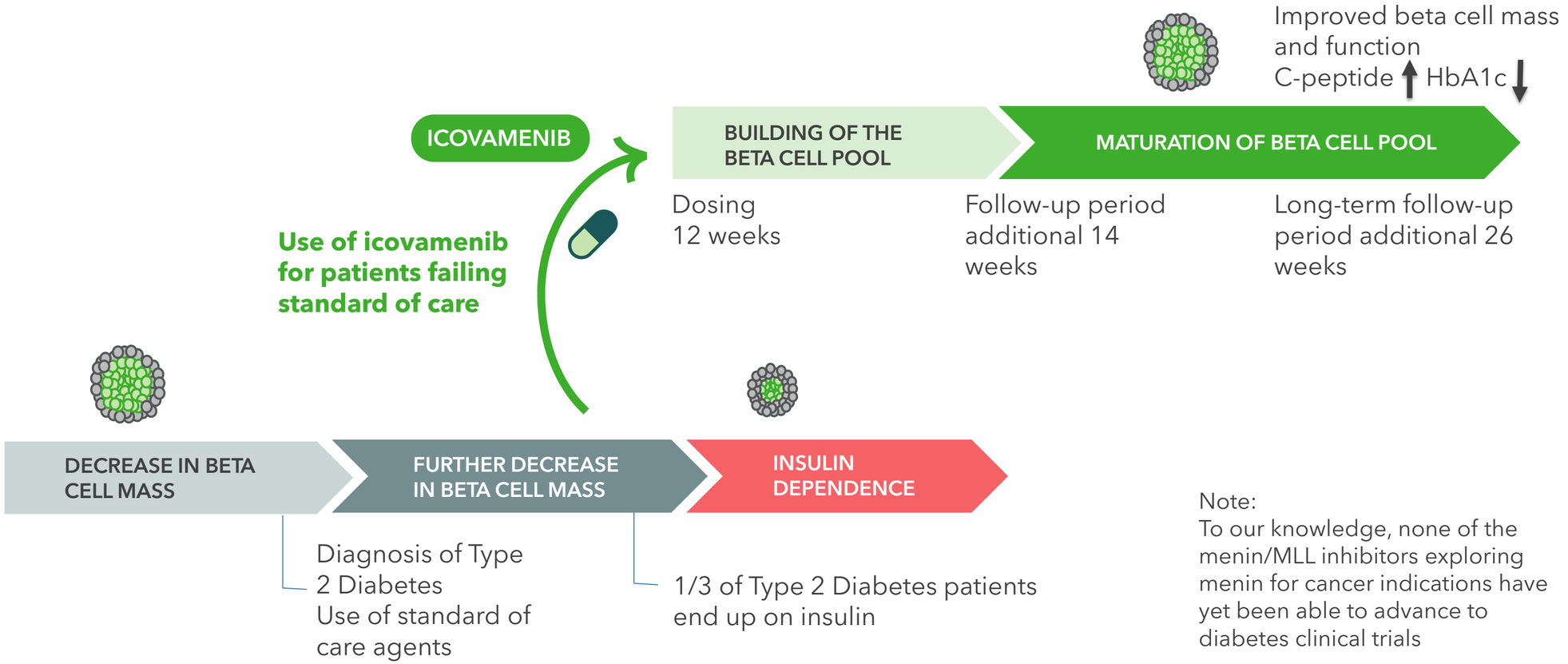
Baseline demographics & characteristics

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)
Number of T2D Medications, n (%)					
1	39 (87)	23 (64)	23 (70)	85 (75)	41 (84)
2	4 (9)	11 (31)	7 (21)	22 (19)	6 (12)
3	2 (4)	2 (6)	3 (9)	7 (6)	2 (4)

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

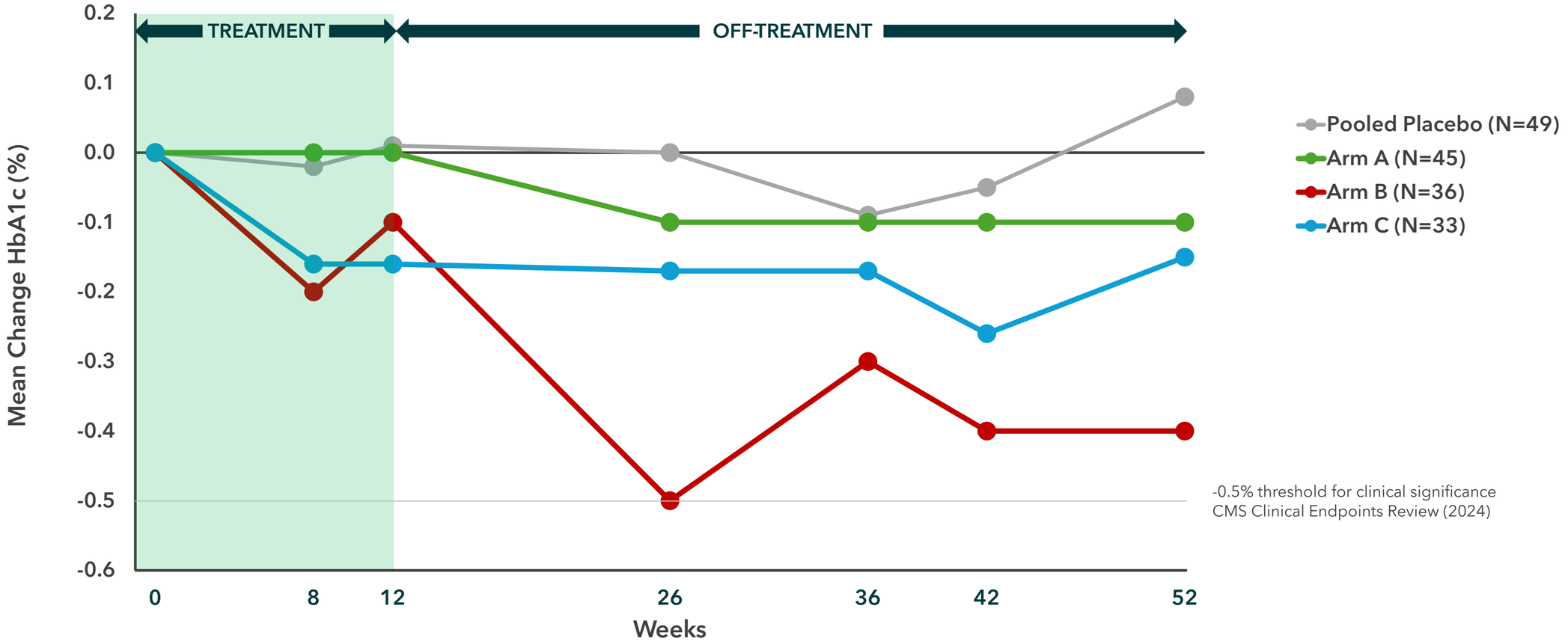
Icovamenib increased beta cell quantity, function & GLP-1 receptor expression following a short treatment period



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

Change in HbA1c from baseline through week 52 – all subtypes

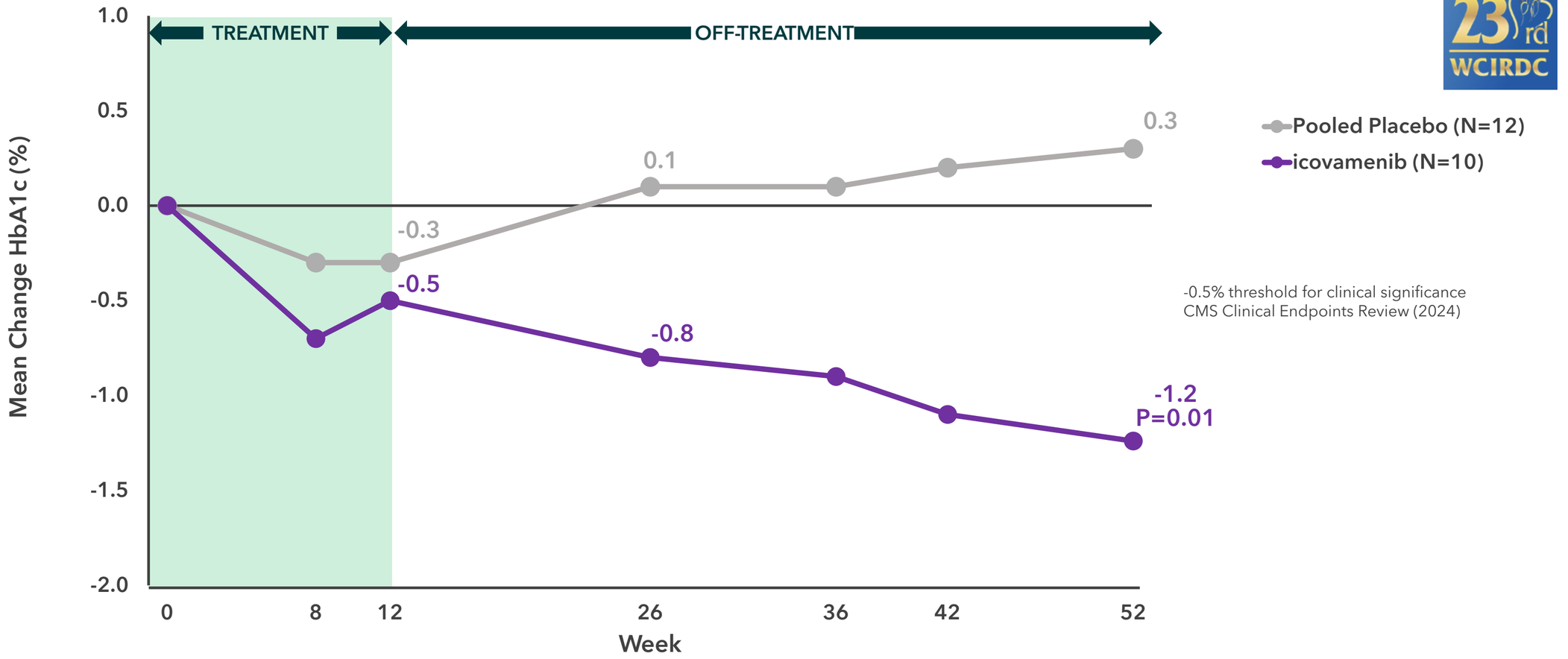
Across treatment durations (Arm A = 8 weeks 100 mg, Arm B = 12 weeks 100 mg, Arm C = 8 weeks 100 mg 4 weeks at 200 mg) per protocol participants taking one or more antihyperglycemic medications at baseline



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

ICOVAMENIB | COVALENT-111

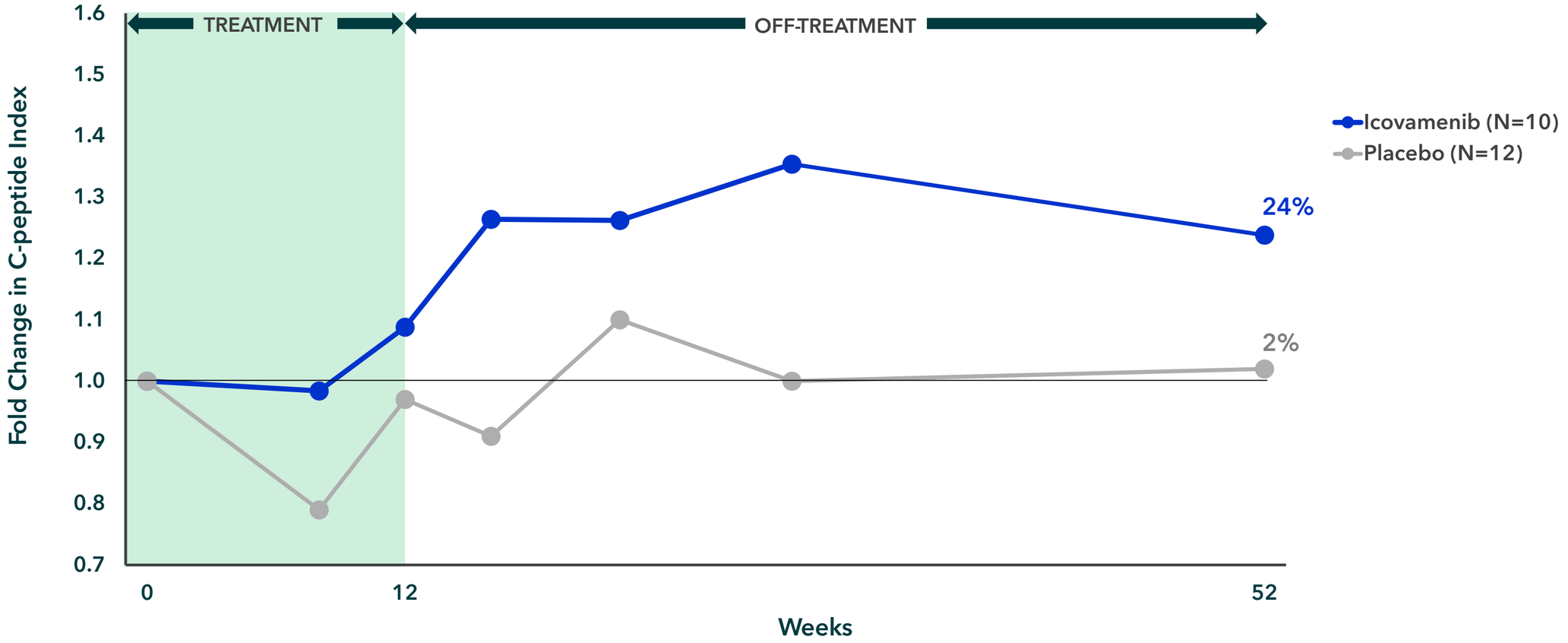
12 weeks of dosing (arms B&C) delivered lasting benefit through 52 weeks for severe insulin-deficient diabetes patients



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

ICOVAMENIB | COVALENT-111

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



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

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Icovamenib promotes selective and partial reduction in menin levels

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

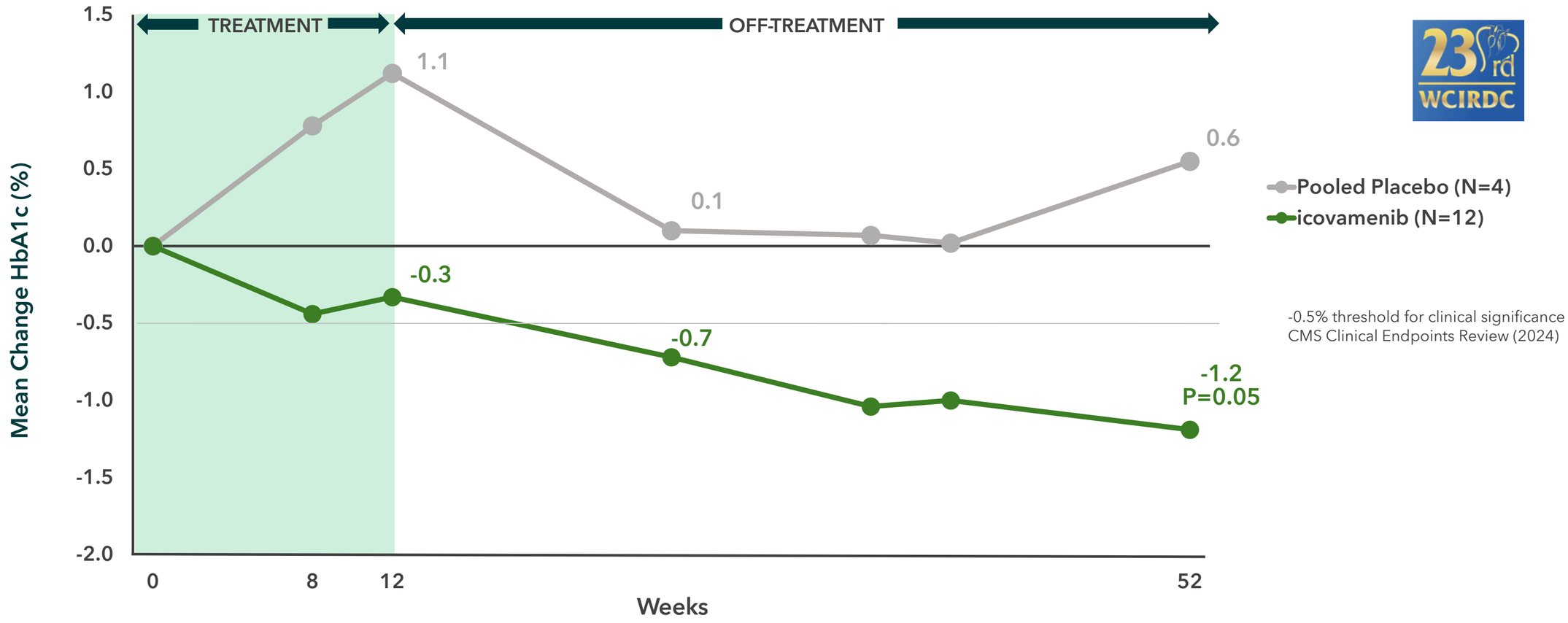
Increased expression of
- **GLP-1**
- **GLP-1 receptor**

IMPROVED INCRETIN EFFECTS

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

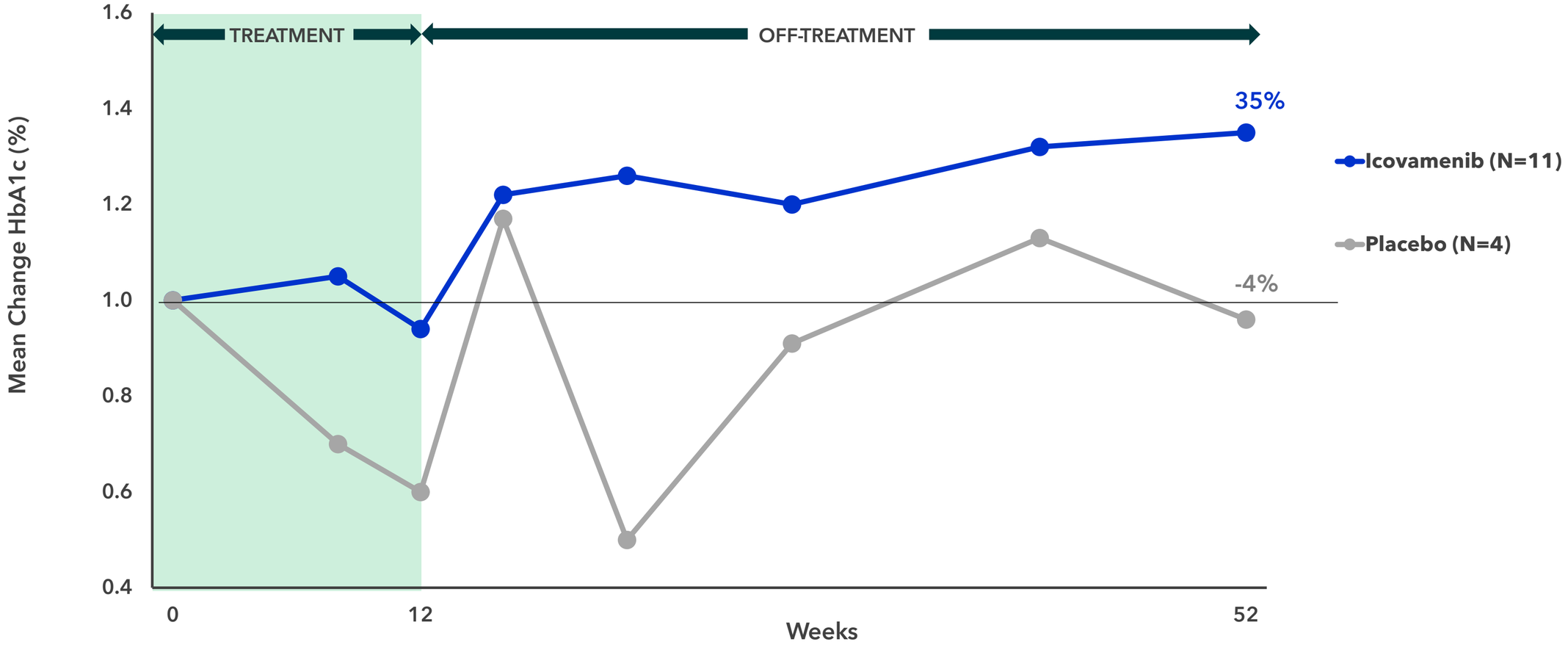
ICOVAMENIB | COVALENT-111

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



ICOVAMENIB | COVALENT-111

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



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

ICOVAMENIB | COVALENT-111

Favorable 52-week safety profile

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

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

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

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

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

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

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

All incidences of ALT and AST elevations resolved without interruption.

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

ICOVAMENIB I CLINICAL

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

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

THERAPY	DOSING REGIMEN	ADMINISTRATION ROUTE	OBSERVATION PERIOD	MEAN HbA1c REDUCTION (PLACEBO ADJ. %)
Ozempic (GLP-1 Agonist)	**Chronic dosing**	Injectable	Week 52 (SUSTAIN 8)	-1.5 (1mg)
Mounjaro (GLP-1/GIP Agonist)	**Chronic dosing**	Injectable	Week 40 (SURPASS 1)	-1.9 (5mg) -2.1 (15 mg)
Jardiance (SGLT2 Inhibitor)	**Chronic dosing**	Oral	Week 52 (Extension study)	-0.6 (10mg) -0.6 (25mg)
Januvia (DPP4 Inhibitor)	**Chronic dosing**	Oral	Week 52 (Sitagliptin)	-0.5 (100mg)

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

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

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

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

ICOVAMENIB

Potential first-in-class oral menin inhibitor for diabetes

Ongoing Phase II Studies

biomea FUSION™

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

ICOVAMENIB

Phase IIa key derisking-insights:

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

Direct application in Phase II Studies

COVALENT-211

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

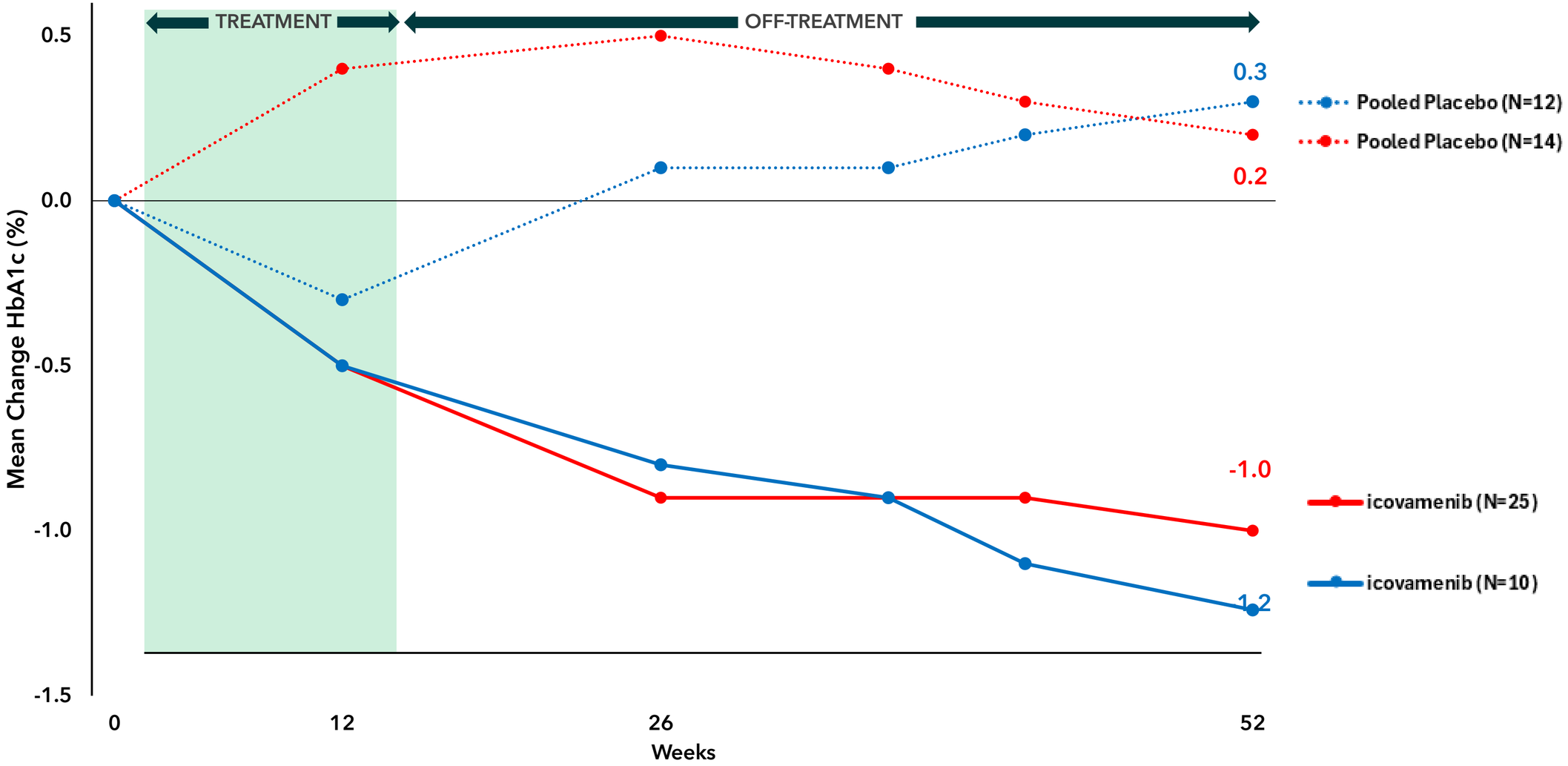
- Adult participants with T2D who were treated with 1-3 antidiabetic medications
- HbA1c 7.5%-10.5% and BMI ≤ 32 kg/m^2
- Background therapy maintained unless rescue required

COVALENT-212

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

- Adult participants with T2D who are not achieving glycemic targets despite GLP-1-based therapy
- HbA1c ≥7.5% and ≤9.5% and BMI 25 to 40 kg/m^2
- Background therapy maintained unless rescue required

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



BMF-650

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

Preclinical results and clinical overview

biomea FUSION™

BMF-650

Developed to deliver strong efficacy with improved oral tolerability

An Investigational Next-Generation Oral GLP-1 Receptor Agonist

Proposed differentiated properties of BMF-650

Improved PK Profile

Greater oral exposure with lower variability observed in preclinical studies

Generally Favorable Safety Profile

Better tolerability associated with higher plasma protein binding in preclinical models

Patient Friendly Design

Oral delivery with the potential for simplified dose escalation

Greater therapeutic window matters

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

Intellectual Property

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

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

BMF-650 | PRECLINICAL

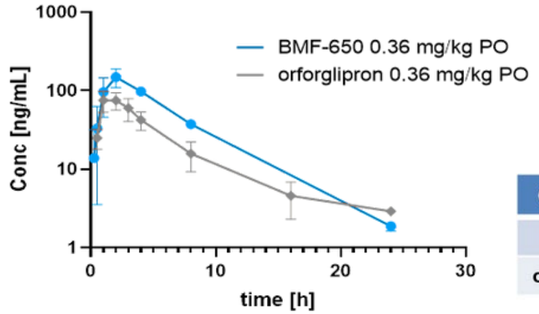
BMF-650 showed favorable in vitro on-target activity and off-target selectivity

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

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

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

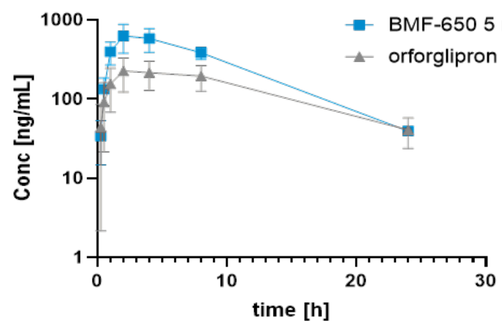
CYNOMOLGUS MONKEY PO PK



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

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

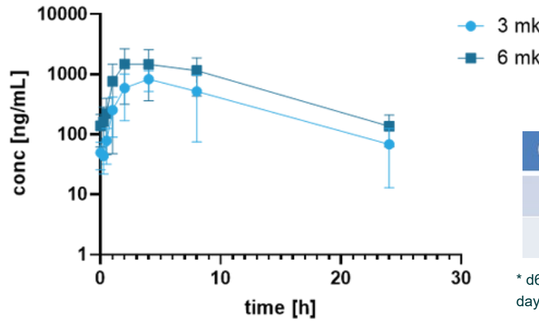
SPRAGUE-DAWLEY RAT PO PK



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

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

CYNOMOLGUS MONKEY PK DAY 6 BMF -650



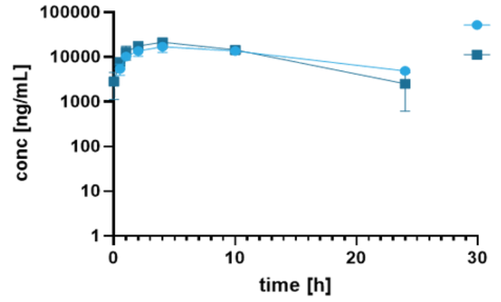
Dose Proportionate Exposure

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

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

PO =per oral

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



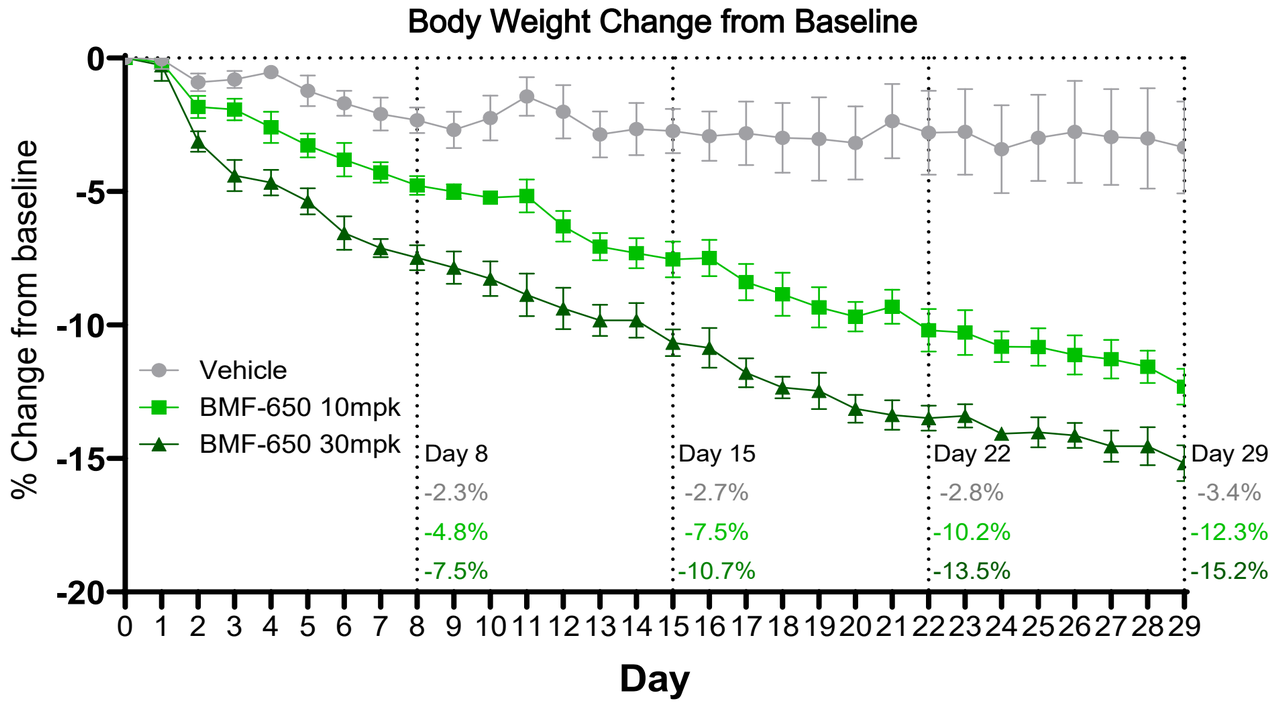
Continuous Exposure after multiple days

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

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

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

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



CT-996 body weight change

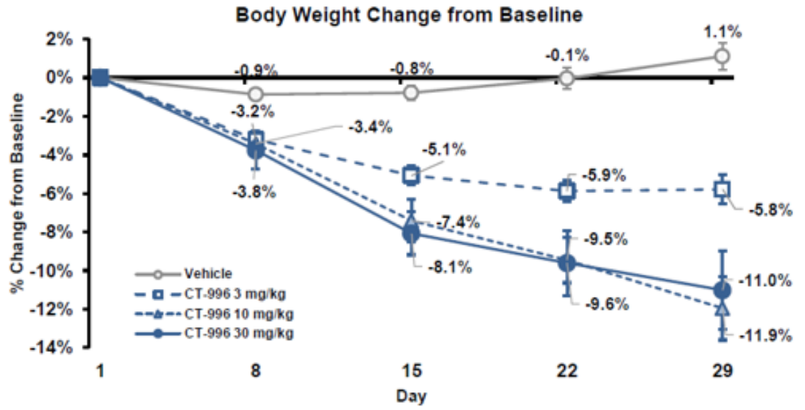


Figure 6. Effects of CT-996 on body weight in obese cynomolgus monkeys following once-daily oral administration. Weekly body weight percent change is represented as mean (± SE) from baseline. N = 6/group.

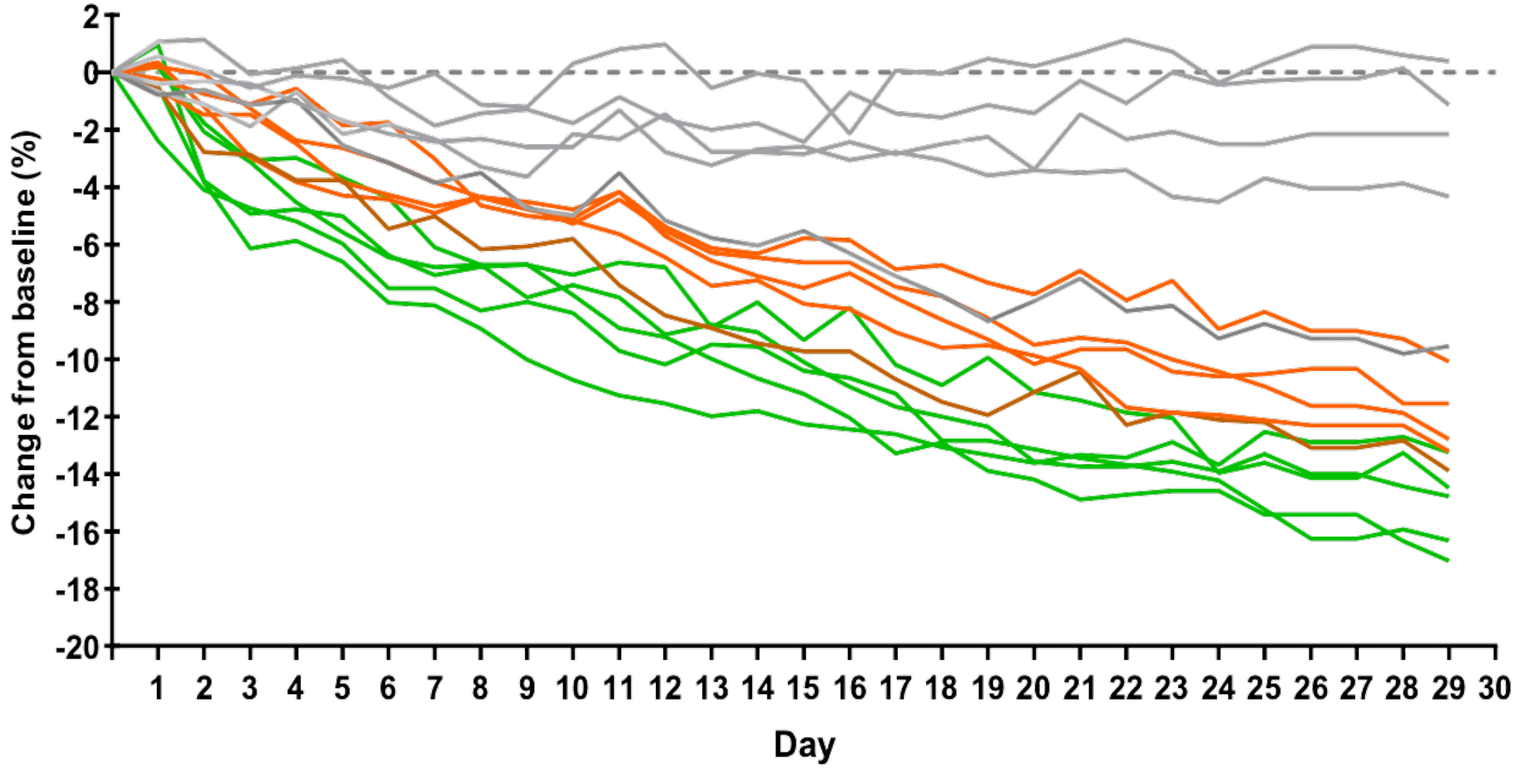
Literature data; Carmot Therapeutics (now part of the Roche group), ADA 2024.

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

BMF-650 | PRECLINICAL

Oral BMF-650 demonstrates strong dose dependent body weight reduction in obese cynomolgus monkeys

BODY WEIGHT CHANGE (individual obese monkey)



Mean reduction in body weight (Day 29)

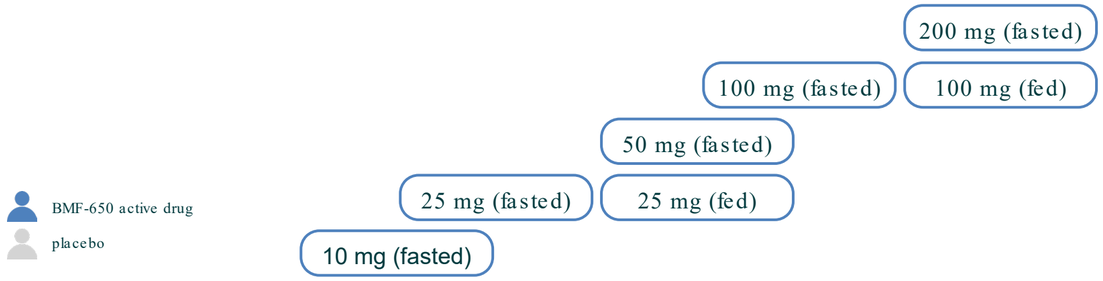
Vehicle	-3.4%
BMF-650 10 mg/kg	-12.3%
BMF-650 30 mg/kg	-15.2%

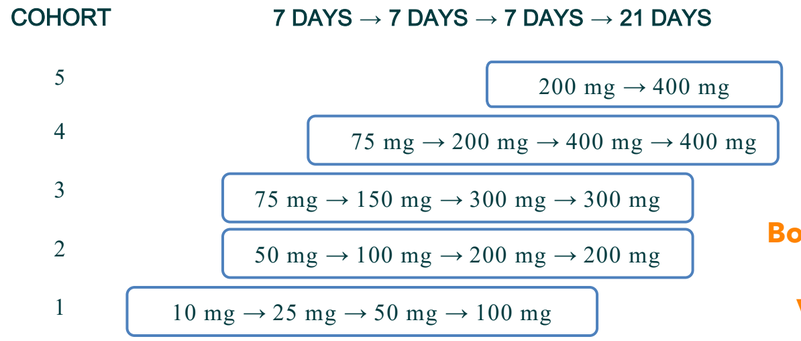
BMF-650 | GLP-131

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

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

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





Body weight at Day 29 and Day 43 versus Baseline

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

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 2 diabetes** Patients with insulin deficiency *(~7M U.S. Patients)* [1]	**COVALENT-211** (study enrolling)			Phase II 26-week data (primary endpoint) anticipated **4Q 2026**
	Type 2 diabetes Patients not controlled on GLP-1 based therapies *(15-45% U.S. Patients on GLP-1RA)* [2,3]	**COVALENT-212** (study enrolling)			Phase II 26-week data (primary endpoint) anticipated **4Q 2026**
ICOVAMENIB with low dose Semaglutide	**Obesity/Overweight** *(>190M U.S. Patients)* [5] Sponsored by Leicester Diabetes Center	**OPAL Study**			Phase II initiation anticipated in **2H 2026**
BMF-650 Potential best-in-class oral GLP-1 RA	**Obesity** *(>100M U.S. Patients)* [5]	**GLP-131** (study enrolling)			Phase I 28-day weight reduction data anticipated in **3Q 2026**

1.International Diabetes Federation. IDF Diabetes Atlas www.diabetesatlas.org (Based on company calculations)
2.NHANES analyses of glycemic control among U.S. adults with diabetes (JAMA; Diabetes Care);
3.SUSTAIN, AWARD, and SURPASS clinical trial programs for GLP-1 receptor agonists
4.Mayer-Davis et al., NEJM / CDC updates
5.National Center for Health Statistics August 2023. Accessed June 10, 2026

THANK YOU

(NASDAQ: BMEA)

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

www.biomeafusion.com

biomea FUSION™

KEY OPINION LEADERS HIGHLIGHT ICOVAMENIB'S POTENTIAL TO TRANSFORM DIABETES TREATMENT

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

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

Steve Edelman, M.D.

ENDOCRINOLOGIST, PROFESSOR OF MEDICINE UCSD / VA SAN DIEGO

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

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

Ralph DeFronzo, M.D.

ENDOCRINOLOGIST, PROFESSOR OF MEDICINE UTHSCSA

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

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

Melanie Davies, M.D.

DIABETOLOGIST, PROFESSOR OF DIABETES MEDICINE AT THE UNIVERSITY OF LEICESTER

KEY OPINION LEADERS HIGHLIGHT ICOVAMENIB'S POTENTIAL TO TRANSFORM DIABETES TREATMENT

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

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

Alice Cheng, M.D.

ENDOCRINOLOGIST, ASSOCIATE PROFESSOR OF MEDICINE
UNIVERSITY OF TORONTO

"The icovamenib data are quite interesting because of the continued effects despite having stopped it for 14 weeks.

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