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 9, 2024

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.)

900 Middlefield Road, 4th Floor Redwood City, California	94063
(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, par value $0.0001 per share	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 8.01 Other Events.

On January 9, 2024, Biomea Fusion, Inc., or the Company, presented a business update at the 42nd Annual J.P. Morgan Healthcare Conference. A copy of the Company's presentation slides, which has been published on the Company's website, is filed as Exhibit 99.1 to this current report on Form 8-K and is incorporated by reference herein.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit	Description
99.1	Biomea Fusion, Inc.'s Presentation at the 42nd Annual J.P. Morgan Healthcare Conference, dated January 9, 2024
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 hereunto duly authorized.

Biomea Fusion, Inc.

Date: January 10, 2024

By: /s/ Thomas Butler
Thomas Butler
Principal Executive Officer

Exhibit 99.1

biomea FUSION™

JP Morgan 2024 Corporate Presentation

Disclaimer

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 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; any statements of expectation or belief regarding future events, potential markets or market size, or technology developments, unfavorable global economic conditions, including inflationary pressures, market volatility, acts of war and civil and political unrest, and other factors affecting the Company's financial condition or operations. 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, as well as discussions of potential risks, uncertainties, and other important factors in our other subsequent filings with the Securities and Exchange Commission. 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.

Excellent Science - Combining Validated Targets with Breakthrough Chemistry

We Aim to Cure

- Experienced Management Team
- Novel FUSION™ System
- BMF-219 – Phase II Stage
- BMF-500 – Phase I Stage
- Development of Combination Assets

biomea FUSION™ | We Aim to Cure™

Biomea Fusion is a clinical-stage biopharmaceutical company focused on the discovery and development of **oral covalent small-molecule drugs** to treat patients with genetically defined cancers and metabolic diseases. We believe that our approach may lead to significant improvement and extension of life for patients. Our team is engaged in all phases of drug discovery and development, including target selection, small molecule design, and preclinical and clinical studies to develop innovative medicines.

Aiming to Develop Some of the Most Impactful Medicines of Our Time

A Long History of Developing Successful Drugs - Together

Thomas Butler
Chairman & CEO

biomea FUSION
Co-Founder

The FUSION™ SYSTEM
BMF-219*
Co-Inventor

imbruvica (ibrutinib)
560, 420, 280, 140 mg tablets | 140, 70 mg capsules

Veklury remdesivir
Co-Inventor

biomea FUSION We Aim to Cure

Ramses Erdtmann
President & COO

biomea FUSION
Co-Founder

imbruvica (ibrutinib)
560, 420, 280, 140 mg tablets | 140, 70 mg capsules

Juan Frías, M.D.
Chief Medical Officer

once weekly mounjaro (tirzepatide) injection 0.5 mL | 5 mg | 7.5 mg

farxiga (dapagliflozin) 5 mg & 10 mg tablets

Jardiance (empagliflozin) tablets 10 mg/25 mg

once weekly trulicity (dulaglutide) injection 0.5 mL | 0.75 mg | 1.5 mg | 3.0 mg | 4.5 mg

ONCE-WEEKLY wegovy semaglutide injection 2.4 mg

Byetta (exenatide) injection

Invokana (canagliflozin) tablets

OZEMPIC semaglutide injection 0.5 mg, 1 mg, 2 mg

Januvia sitagliptin

Naomi Cretcher
Chief of People

imbruvica (ibrutinib)
560, 420, 280, 140 mg tablets | 140, 70 mg capsules

Heow Tan
Chief Technical & Quality Officer

imbruvica (ibrutinib)
560, 420, 280, 140 mg tablets | 140, 70 mg capsules

Xtampza ER (oxycodone) extended-release capsules II

ZADAXIN

PLENAXIS

HylatopicPlus Cream

Steve Morris, M.D.
Chief Development Officer

XALKORI CRIZOTINIB

ZYKADIA ceritinib 150 mg

LORBRENA LORLATINIB

ALECENSA alectinib 150 mg capsules

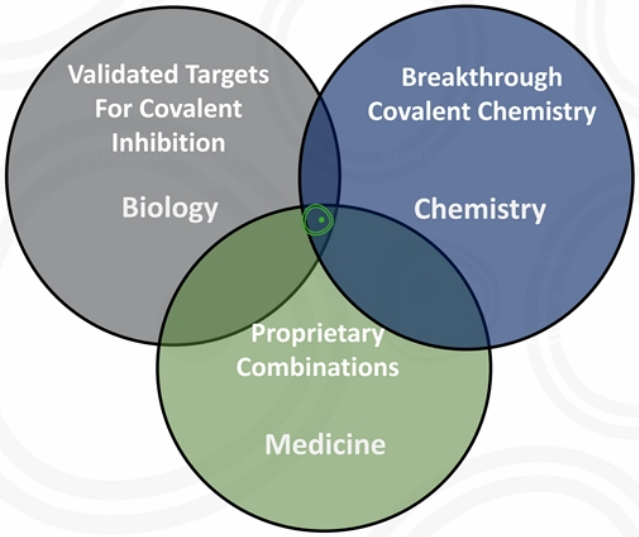
ALUNBRIG BRIGATINIB 30mg TABLETS

Franco Valle
Chief Financial Officer

imbruvica (ibrutinib)
560, 420, 280, 140 mg tablets | 140, 70 mg capsules

*Note: BMF-219 is an investigational new drug

Biomea's Development Principles



Validated Targets

Drugs pursuing **Validated Disease Targets** have a ~2x higher likelihood of approval than molecules pursuing a new mechanism of action

Sources: Nelson et al. (2015) Nat Genet.; Thomas et al. (2016) BIO; In a Landscape of 'Me Too' Drug Development, What Spurs Radical Innovation? HBS Weekly Review (Jun 2018)

Covalent Inhibitors

Covalent Small Molecule Inhibitors provide deep target inactivation and a wider therapeutic window, allowing for longer duration on therapy

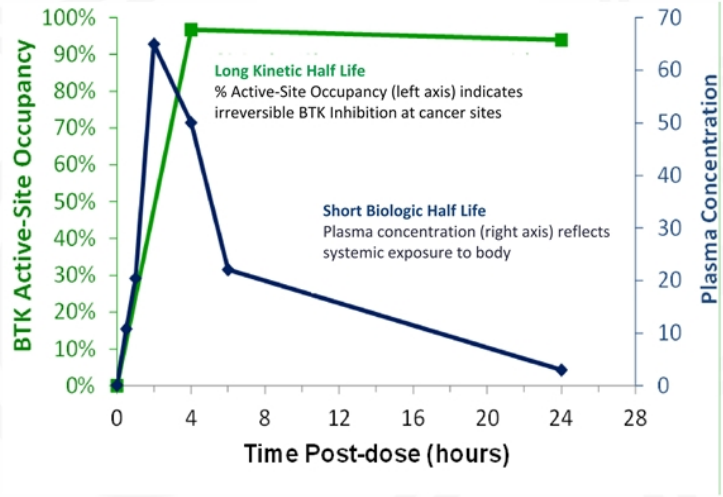
Sources: Singh et al. (2011) Nature Reviews Drug Discovery; Cheng et al. (2020) Journal of Hematology & Oncology; Strelow (2017) SLAS Discovery; Kalgutkar & Dalvie (2012) Expert Opin. Drug Discov.;

Proprietary Combinations

Combination Therapy with non-overlapping resistance mechanisms results in more durable responses and better outcomes

Sources: Palmer et al. (2019) eLife; Mokhtari et al. (2017) Oncotarget

Covalent Inhibitors Have Long Kinetic but Short Biological Half Life



*Pharmacyclics Corporate Deck 2012

High Selectivity

Two-step inhibition: 1) Initial reversible binding followed by 2) covalent interaction, increasing target selectivity

Deep Target Inactivation

Permanent inactivation of bound protein drives target elimination through normal cellular degradation processes

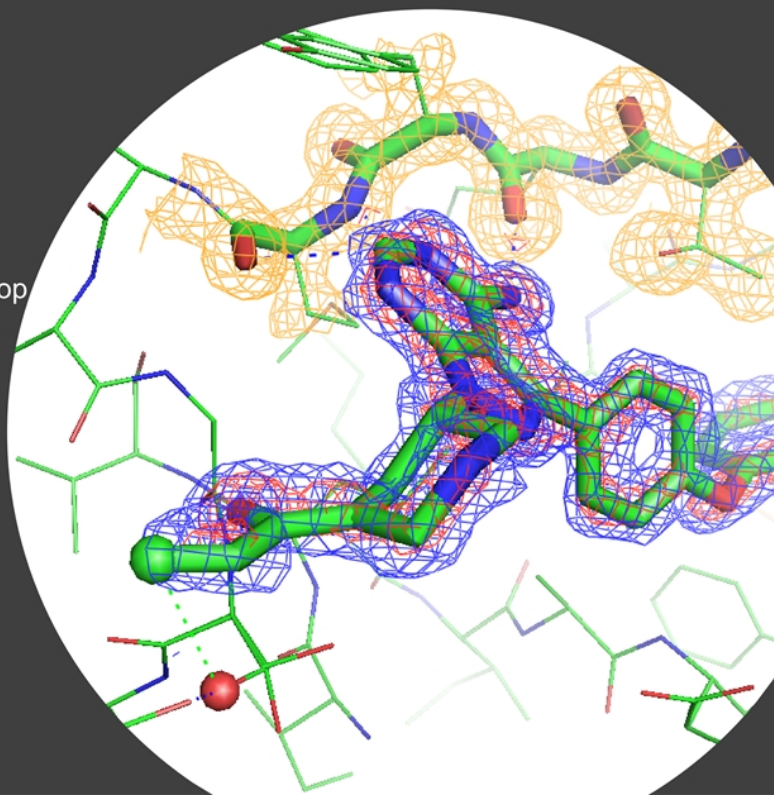
Greater Therapeutic Window

Designed to maintain an effect without sustained systemic exposure, unlike conventional non-covalent inhibitors

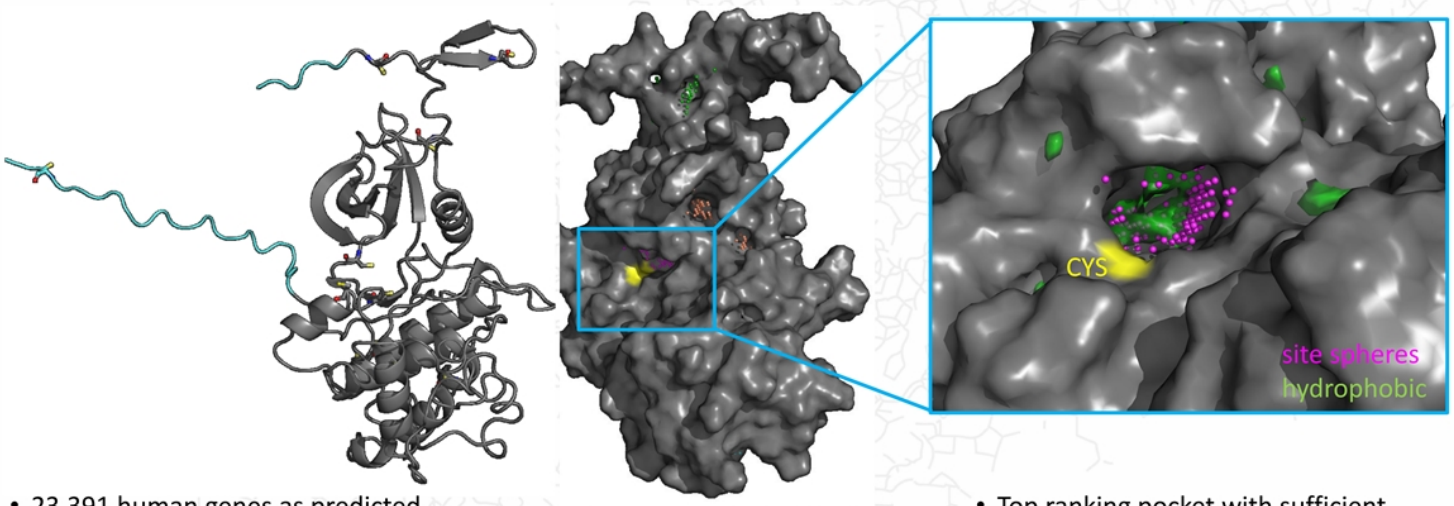
Our FUSION™ System

We leverage our FUSION System to discovery and develop Novel covalent inhibitors against targets essential for many diseases.

- Novel Target Selection Process
- Crystal Structure based Drug Design
- Proprietary Scaffold Construction



Human Genome Wide Covalent Pocket Analysis



- 23,391 human genes as predicted structures; 14,159 novel vs PDB
- Remove spurious N- and C-termini (blue)
- Analyze individual domains if needed – potential artificial inter-domain pockets
- Manual curation for high interest targets

- Analyze Apo structures without ligands
- Pocket identification using established methods SiteMap → "bindability" ranking

- Top ranking pocket with sufficient hydrophobic character

→ Virtual screening for ligands

→ Biomea Linker/Warhead Determination Protocol

→ Lead Molecule(s)

Biomea's Pipeline Expands into 5 Clinical Trials with 2 Novel Agents

Multiple Upcoming Milestones in the Near Term

	Study	Indications	Milestones	Expected Timeline
BMF-219 Menin Program	COVALENT-111	Type 2 Diabetes	Phase II - Dose Escalation Completion, ATTD	1Q 2024
	COVALENT-112	Type 1 Diabetes	Phase II - Initial Proof of Concept	2024
	COVALENT-101	Liquid Tumors	Phase I - Dose Escalation Completion, RP2D	2024
	COVALENT-102	Solid Tumors	Phase I - Dose Escalation Completion, RP2D	2024
BMF-500 FLT3 Program	COVALENT-103	AML/ALL (acute leukemia)	Phase I - Dose Escalation Completion, RP2D	2024
Additional Program	Target # 3	TBA	Progress Update	2024

biomea FUSION We Aim to Cure

Aiming to Develop Some of the Most Impactful Medicines of Our Time

Juan Pablo Frías, M.D. is Appointed as Biomea's Chief Medical Officer

August 31, 2023

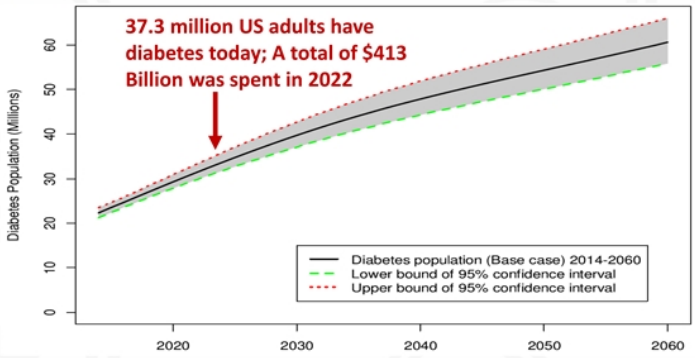
Dr. Frías is a board-certified endocrinologist who has served as principal investigator on over **250 clinical diabetes studies**, with over half of those being Phase III studies, and has participated in the clinical development of more than **20 approved diabetic agents**

- Previous Pharmaceutical Leadership Positions: in Clinical and Medical Affairs at Eli Lilly, Amylin Pharmaceuticals, Pfizer, and Johnson & Johnson, where he served as CMO and Global Vice President of Clinical and Medical Affairs, Diabetes Care.
- Academic Positions:
  - University of Colorado Health Sciences Center, Barbara Davis Center for Diabetes
  - Clinical Faculty at University of California San Diego School of Medicine
  - Published over 125 articles in peer reviewed journals

2 in 5 Americans Will Develop Diabetes during Their Lifetime

- Diabetes is the 7th leading cause of death in the US. 80% of people with diabetes will die from this disease. Premature mortality caused by diabetes results in an estimated 12-14 years of life lost. Source: National library of Medicine 1(2); 2007 Jul PMC3068646

- Diabetes creates one of the largest economic burdens on the US health care system. $1 out of every $4 in US health care costs is being spent on caring for people with diabetes. In 2022 the US spent $412.9 Billion to treat diabetes. On average, people with diagnosed diabetes have medical expenditures 2.6 times higher than would be expected without diabetes.

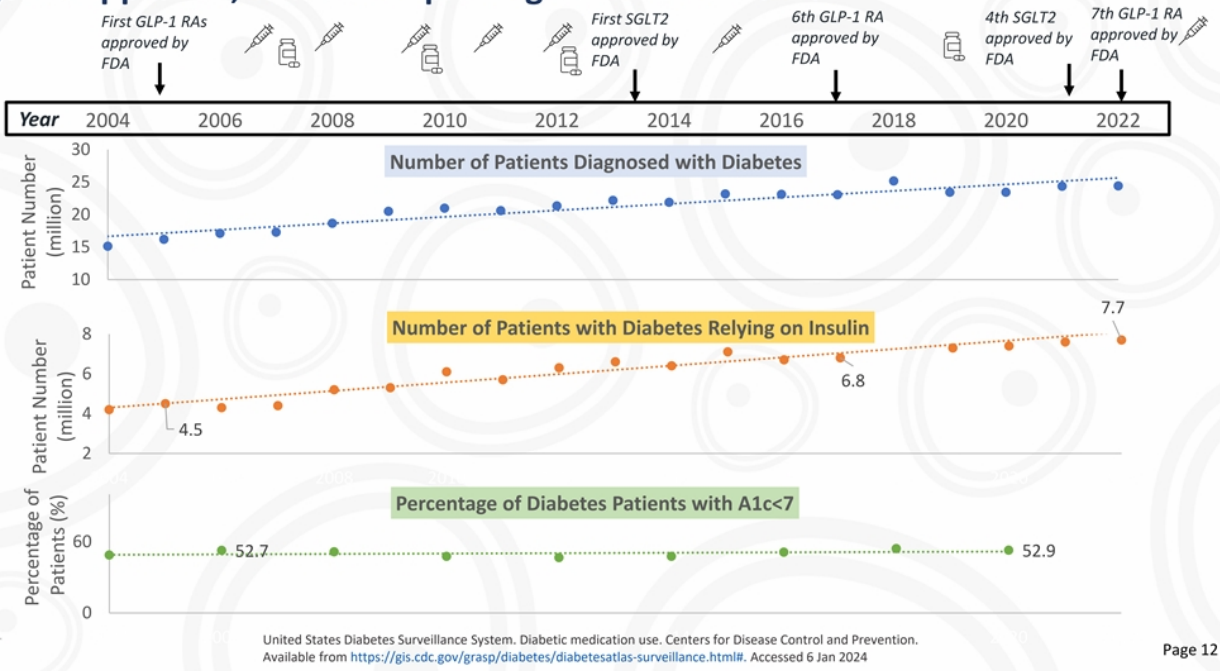


- According to the CDC, worldwide 537 million adults have diabetes. In the United States alone, 37.3 million adults have diabetes, 11.3% of the population. 96 million adults (more than 1 in 3) in the US have prediabetes. CDC.gov - By the Numbers: Diabetes in America

- In a study conducted by Prime Therapeutics, 68% of patients using GLP-1 drugs to address weight loss, stopped using them within the first year, according to 16M insured members.
https://www.primetherapeutics.com/news/real-world-analysis-of-glp-1a-drugs-for-weight-loss-finds-low-adherence-and-increased-cost-in-first-year/

- In a study published by the Obesity Journal, only 59% of adults were still taking GLPs after three months and only 32% after one year (semaglutide was used 40% after 1 year). Early- and later-stage persistence with antiobesitymedications: A retrospective cohort study

Number of Patients with Diabetes Relying on Insulin Continues to Rise despite Novel Diabetic Agents Approved, without Improving the A1c Outcome



United States Diabetes Surveillance System. Diabetic medication use. Centers for Disease Control and Prevention. Available from https://gis.cdc.gov/grasp/diabetes/diabetesatlas-surveillance.html#. Accessed 6 Jan 2024

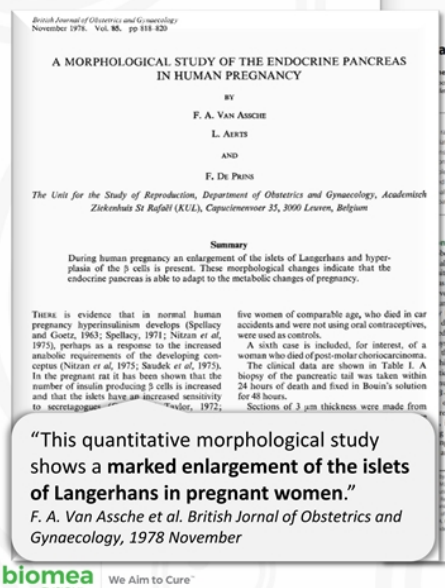
Investigational BMF-219 - A Unique Value Proposition: Beta Cell Health

BMF-219: 1st in Class Potential for Differentiated Profile

- Oral Small Molecule
- Complementary Agent to Available Diabetes Therapies
- Non-Chronic Dosing
- Well-Tolerated Profile After First Read Out
- Disease Modifying Potential Addressing the Root Cause of Diabetes
- Continued Glycemic Control Even After Cessation of Dosing
- Addressable Market May Include All Diabetic Patients

Beta Cells Proliferate

- Just not enough to overcome pre-existing metabolic disorder



British Journal of Obstetrics and Gynaecology
November 1978. Vol. 85. pp 818-820

A MORPHOLOGICAL STUDY OF THE ENDOCRINE PANCREAS IN HUMAN PREGNANCY

BY

F. A. VAN ASSCHE

L. AERTS

AND

F. DE PRINS

The Unit for the Study of Reproduction, Department of Obstetrics and Gynaecology, Academisch Ziekenhuis St Rafaël (KUL), Capucienenvoer 35, 3000 Leuven, Belgium

Summary

During human pregnancy an enlargement of the islets of Langerhans and hyperplasia of the β cells is present. These morphological changes indicate that the endocrine pancreas is able to adapt to the metabolic changes of pregnancy.

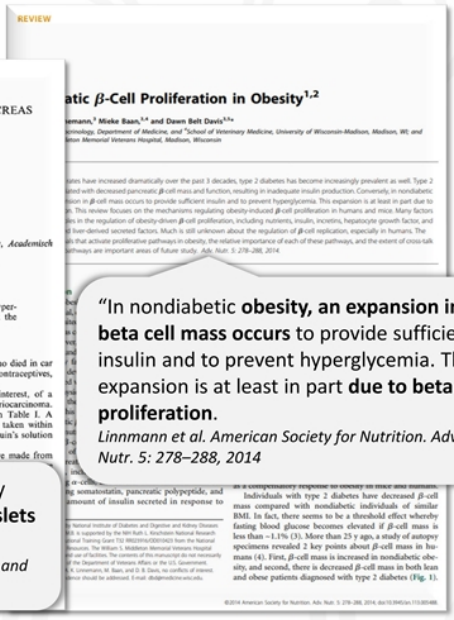
THERE is evidence that in normal human pregnancy hyperinsulinism develops (Spellacy and Goetz, 1963; Spellacy, 1971; Nitzan *et al*, 1975), perhaps as a response to the increased anabolic requirements of the developing conceptus (Nitzan *et al*, 1975; Saudek *et al*, 1975). In the pregnant rat it has been shown that the number of insulin producing β cells is increased and that the islets have an increased sensitivity to secretagogues [illegible]wler, 1972; five women of comparable age, who died in car accidents and were not using oral contraceptives, were used as controls.

A sixth case is included, for interest, of a woman who died of post-molar choriocarcinoma.

The clinical data are shown in Table I. A biopsy of the pancreatic tail was taken within 24 hours of death and fixed in Bouin's solution for 48 hours.

Sections of 3 μm thickness were made from

"This quantitative morphological study shows a **marked enlargement of the islets of Langerhans in pregnant women.**"
F. A. Van Assche et al. British Jornal of Obstetrics and Gynaecology, 1978 November



REVIEW

atic β-Cell Proliferation in Obesity[1,2]

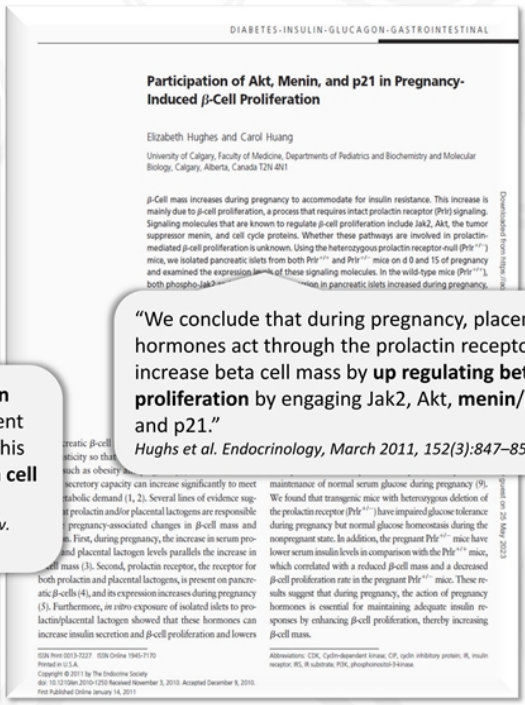
[illegible][3] Mieke Baan,[3,4] and Dawn Belt Davis[3,5]*

[illegible]nology, Department of Medicine, and [5]School of Veterinary Medicine, University of Wisconsin-Madison, Madison, WI; and [illegible]ton Memorial Veterans Hospital, Madison, Wisconsin

[illegible]tes have increased dramatically over the past 3 decades; type 2 diabetes has become increasingly prevalent as well. Type 2 [illegible]ated with decreased pancreatic β-cell mass and function, resulting in inadequate insulin production. Conversely, in nondiabetic [illegible]sion in β cell mass occurs to provide sufficient insulin and to prevent hyperglycemia. This expansion is at least in part due to [illegible]n. This review focuses on the mechanisms regulating obesity-induced β-cell proliferation in humans and mice. Many factors [illegible]ies in the regulation of obesity-driven β-cell proliferation, including nutrients, insulin, incretins, hepatocyte growth factor, and [illegible]l liver-derived secreted factors. Much is still unknown about the regulation of β-cell replication, especially in humans. The [illegible]ays that activate proliferative pathways in obesity, the relative importance of each of these pathways, and the extent of cross-talk [illegible]thways are important areas of future study. *Adv. Nutr. 5: 278–288, 2014.*

Individuals with type 2 diabetes have decreased β-cell mass compared with nondiabetic individuals of similar BMI. In fact, there seems to be a threshold effect whereby fasting blood glucose becomes elevated if β-cell mass is less than ~1.1% (3). More than 25 y ago, a study of autopsy specimens revealed 2 key points about β-cell mass in humans (4). First, β-cell mass is increased in nondiabetic obesity, and second, there is decreased β-cell mass in both lean and obese patients diagnosed with type 2 diabetes (Fig. 1).

"In nondiabetic **obesity, an expansion in beta cell mass occurs** to provide sufficient insulin and to prevent hyperglycemia. This expansion is at least in part **due to beta cell proliferation**.
Linnmann et al. American Society for Nutrition. Adv. Nutr. 5: 278–288, 2014



DIABETES-INSULIN-GLUCAGON-GASTROINTESTINAL

Participation of Akt, Menin, and p21 in Pregnancy-Induced β-Cell Proliferation

Elizabeth Hughes and Carol Huang

University of Calgary, Faculty of Medicine, Departments of Pediatrics and Biochemistry and Molecular Biology, Calgary, Alberta, Canada T2N 4N1

β-Cell mass increases during pregnancy to accommodate for insulin resistance. This increase is mainly due to β-cell proliferation, a process that requires intact prolactin receptor (Prlr) signaling. Signaling molecules that are known to regulate β-cell proliferation include Jak2, Akt, the tumor suppressor menin, and cell cycle proteins. Whether these pathways are involved in prolactin-mediated β-cell proliferation is unknown. Using the heterozygous prolactin receptor-null ($Prlr^{+/-}$) mice, we isolated pancreatic islets from both $Prlr^{+/+}$ and $Prlr^{+/-}$ mice on d 0 and 15 of pregnancy and examined the expression [illegible] of these signaling molecules. In the wild-type mice ($Prlr^{+/+}$), both phospho-Jak2 [illegible] in pancreatic islets increased during pregnancy.

[illegible]creatic β-cell [illegible]sticity so that [illegible]such as obesity [illegible] secretory capacity can increase significantly to meet [illegible]tabolic demand (1, 2). Several lines of evidence sug- [illegible]t prolactin and/or placental lactogens are responsible [illegible] pregnancy-associated changes in β-cell mass and [illegible]n. First, during pregnancy, the increase in serum pro- [illegible]and placental lactogen levels parallels the increase in [illegible]ell mass (3). Second, prolactin receptor, the receptor for both prolactin and placental lactogens, is present on pancreatic β-cells (4), and its expression increases during pregnancy (5). Furthermore, *in vitro* exposure of isolated islets to prolactin/placental lactogen showed that these hormones can increase insulin secretion and β-cell proliferation and lowers maintenance of normal serum glucose during pregnancy (9). We found that transgenic mice with heterozygous deletion of the prolactin receptor ($Prlr^{+/-}$) have impaired glucose tolerance during pregnancy but normal glucose homeostasis during the nonpregnant state. In addition, the pregnant $Prlr^{+/-}$ mice have lower serum insulin levels in comparison with the $Prlr^{+/+}$ mice, which correlated with a reduced β-cell mass and a decreased β-cell proliferation rate in the pregnant $Prlr^{+/-}$ mice. These results suggest that during pregnancy, the action of pregnancy hormones is essential for maintaining adequate insulin responses by enhancing β-cell proliferation, thereby increasing β-cell mass.

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Printed in U.S.A.

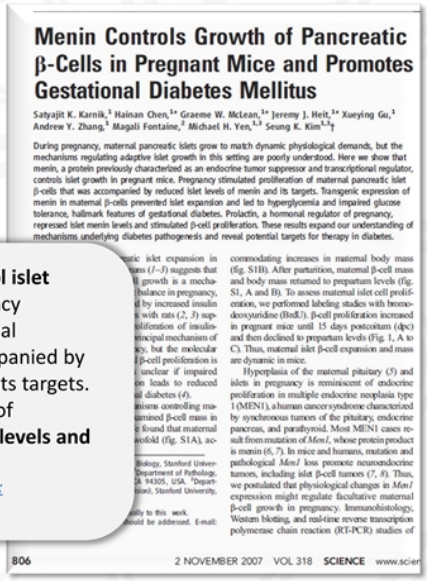
doi: 10.1210/en.2010-1250 Received November 3, 2010. Accepted December 9, 2010.
First Published Online January 14, 2011

Abbreviations: CDK, Cyclin-dependent kinase; CIP, cyclin inhibitory protein; IR, insulin receptor; IRS, IR substrate; PI3K, phosphoinositol-3-kinase.

"We conclude that during pregnancy, placental hormones act through the prolactin receptor to increase beta cell mass by **up regulating beta cell proliferation** by engaging Jak2, Akt, **menin**/p18, and p21."
Hughes et al. Endocrinology, March 2011, 152(3):847–855

Menin Controls Beta-Cell Proliferation and Mass

- Menin is a transcriptional scaffold protein that controls the expression of proteins that regulate beta-cell proliferation.
- Menin is thought to act as a brake on beta cell turnover / beta cell growth, supporting the notion that inhibition of menin could lead to the reactivation, protection, and regeneration of beta cells, which could be a disease-modifying approach to treat type 2 diabetes.



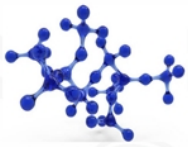
Menin Controls Growth of Pancreatic β-Cells in Pregnant Mice and Promotes Gestational Diabetes Mellitus

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[1,2]†

During pregnancy, maternal pancreatic islets grow to match dynamic physiological demands, but the mechanisms regulating adaptive islet growth in this setting are poorly understood. Here we show that menin, a protein previously characterized as an endocrine tumor suppressor and transcriptional regulator, controls islet growth in pregnant mice. Pregnancy stimulated proliferation of maternal pancreatic islet β-cells that was accompanied by reduced islet levels of menin and its targets. Transgenic expression of menin in maternal β-cells prevented islet expansion and led to hyperglycemia and impaired glucose tolerance, hallmark features of gestational diabetes. Prolactin, a hormonal regulator of pregnancy, repressed islet menin levels and stimulated β-cell proliferation. These results expand our understanding of mechanisms underlying diabetes pathogenesis and reveal potential targets for therapy in diabetes.

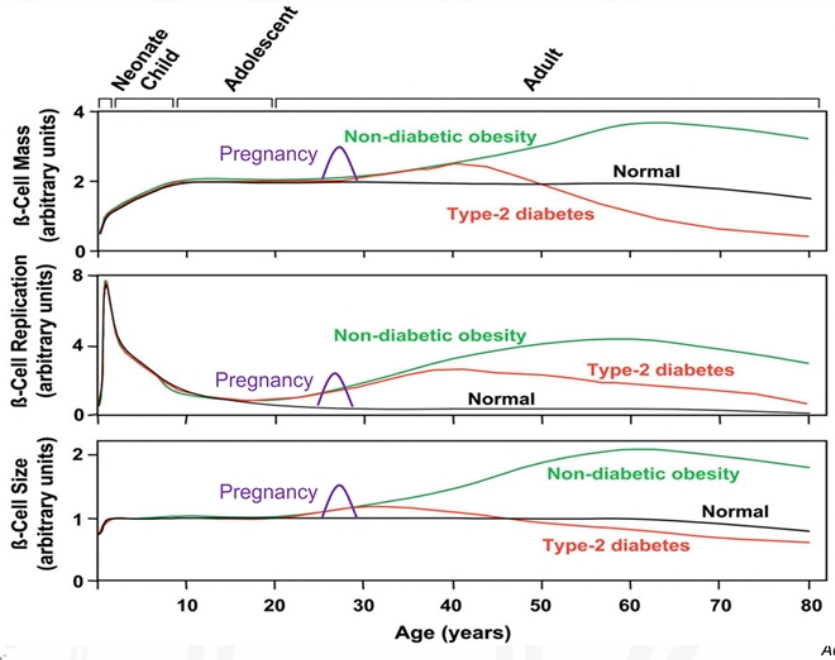
- **Menin has been found to control islet growth** in pregnant mice. Pregnancy stimulated proliferation of maternal pancreatic islet b-cells was accompanied by reduced islet levels of menin and its targets.
- **Prolactin**, a hormonal regulator of pregnancy, **repressed islet menin levels and stimulated b-cell proliferation**.

Dr. Kim, S.K. et al., Science. 2007 Nov 2. doi: 10.1126/science.1146812.



BMF-219 *is a small molecule designed by the Biomea Fusion Team to covalently inhibit menin. Preclinical studies have shown that the inhibition of menin leads to the overall rehabilitation of beta cell health and function, and thereby to increased insulin production and glycemic control. Clinical trials with BMF-219 are under way to investigate oral dosing for a limited time only until the pool of healthy beta cells are restored. The goal is to address diabetes with BMF-219 at the root cause.*

Beta Cell Compensation in Physiological and Pathophysiological States in Mammals



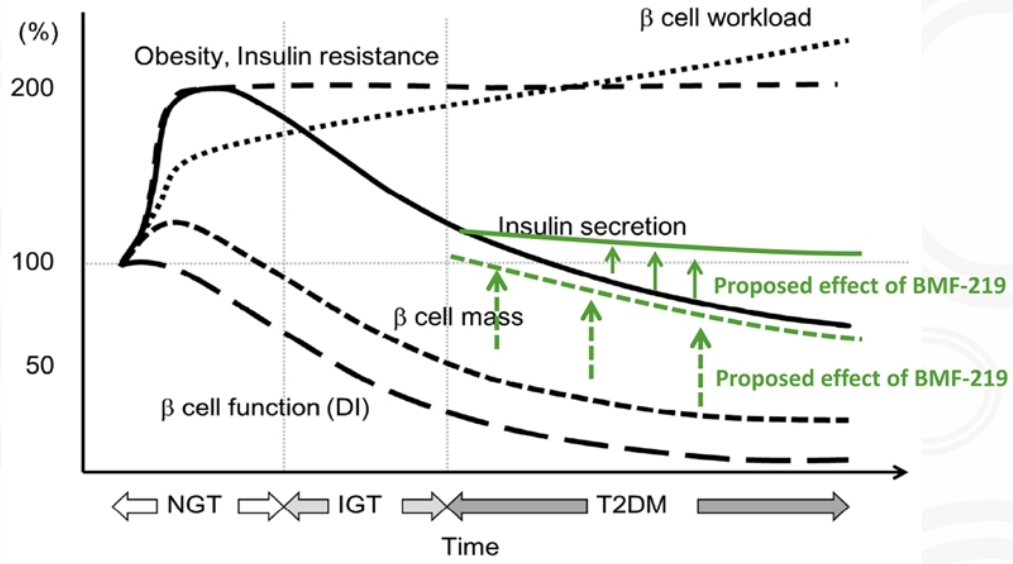
β-Cell Mass

β-Cell Replication

β-Cell Size

Adapted from Science 2005

The Goal for BMF-219 is to Improve Glycemic Control without Continuous Medication

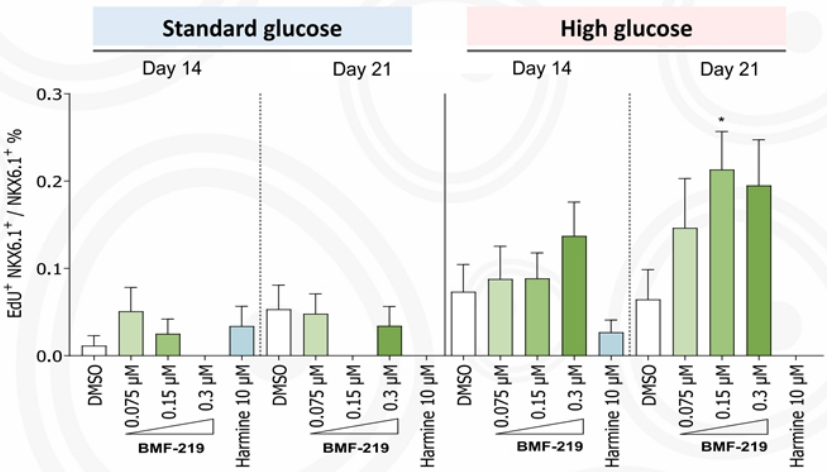


BMF-219 is aimed to increase beta cell mass and function, thereby increase insulin production in order to achieve glycemic control - without the need of continuous medication.

*Int. J. Mol. Sci. 2016, 17, 744; doi:10.3390/ijms17050744

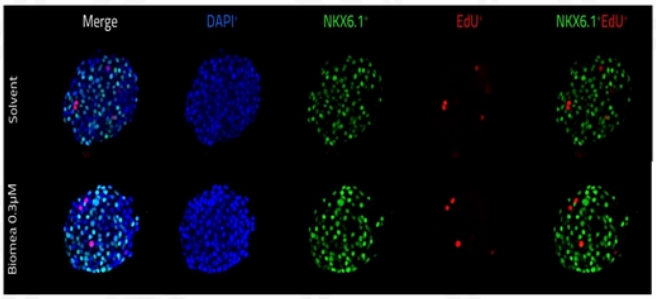
BMF-219 Induced a Glucose-Dependent Enhancement in β-Cell Proliferation

Donor 2 Proliferating beta cells as a fraction of total beta cells



Data represent mean ±SEM of 1 donor with n = 9-12 technical replicates.
One-way ANOVA with Dunnett's post hoc test rel. to DMSO control. *p < 0.05, **p < 0.01, ***p < 0.001

Donor 2; Day 14, High glucose

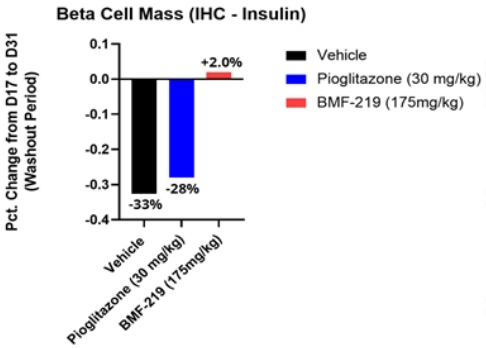


Donor 2	Age	BMI	HbA1c
Caucasian	32	25.0	5.2

Proliferation observed only under elevated glucose conditions, which mimic diabetic levels.

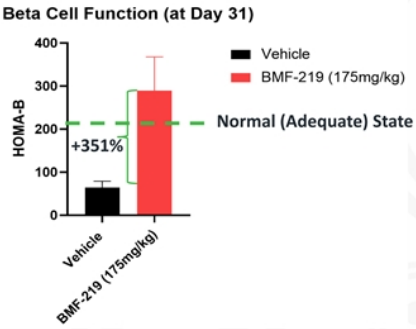
BMF-219 Preserved, Reactivated and Regenerated Beta Cells in Preclinical Studies

Preservation



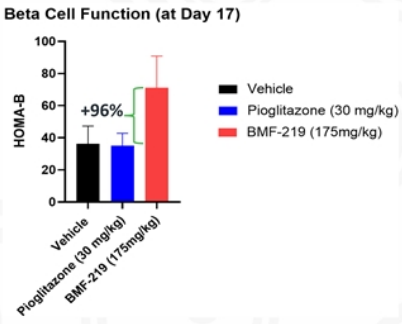
Quantitative Analysis of pancreatic islet tissue cross sections shows BMF-219 treated **ZDF** animals show novel effects in Beta Cell Mass growth and maintenance. BMF-219 was able to maintain Beta Cell function and prevent Beta Cell Mass loss in a model of insulin resistance. Importantly, Beta Cell Mass is maintained, despite cessation of dosing.

Reactivation



BMF-219 demonstrated a significant level of beta cell function compared to vehicle at day 31 in an insulin resistant type 2 diabetes animal model (**ZDF**). Homa B, a measurement of Beta Cell function, was analyzed using 4 h fasting glucose and insulin levels. It increased up to ~351% versus vehicle, despite cessation of therapy.

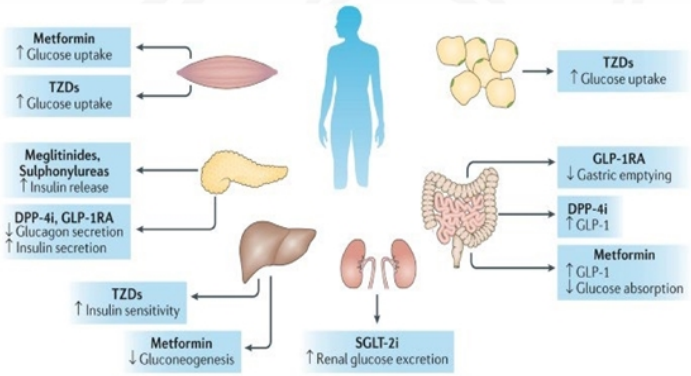
Regeneration



BMF-219 increased HOMA-B by 96% in a type 2 animal model (STZ = 50% Beta Cell destruction). Homa B, a measurement of Beta Cell function, was analyzed using 4 h fasting glucose and insulin levels. BMF-219 in ex-vivo Human Donor Islets (Ex-Vivo) statistically significant increased beta cells with BMF-219.

BMF-219 Mechanism of Action

BMF-219 is a Potential First-in-Class Diabetic Agent – Addressing the Root Cause of Disease



Nat Rev Endocrinol 12, 337–346 (2016).
https://doi.org/10.1038/nrendo.2016.51

Currently approved therapies are primarily targeting the **Symptoms of Type 2 Diabetes: *Hyperglycemia***

BMF-219: Menin Inhibition a Potential New Class of Diabetes Agents

Beta Cell Mass ↑ Beta Cell Health ↑

Control of Glycemia even after Cessation of Dosing

BMF-219 represents a potential new class of diabetes agents addressing the: **Root Cause of Diabetes**
- Loss of Beta Cell Mass and Function -

Additional Dose Levels and Various Dosing Durations Are Being Explored in the Escalation and Expansion Portion of COVALENT-111

Part 1 Dose Escalation,
4 weeks dosing+ 12 weeks follow up

- **Healthy Volunteers** n=16
- **50 mg QD, n=10** x 4 wks
- **100 mg QD, n=20** x 4 wks
- **200 mg QD / 100 mg BID, n=22** x 4 wks
- **200 mg QD,** x 2 wks, **n=10** → **400 mg QD** x 2 wks

Part 2 Dose Expansion, n=216 – 288 incl.
12 weeks dosing + 40 weeks follow-up

Arm A*	**100 mg** x 8 wks	
Arm B	**100 mg** x 12 wks	
Arm C	**100 mg** x 8 wks	→ **200 mg** x 4 wks
Arm D	Anticipated to be added based on data from 400 mg cohort of escalation portion	

*Redosing if required at Week 22 for another 4 weeks.

Baseline Characteristics and Demographics

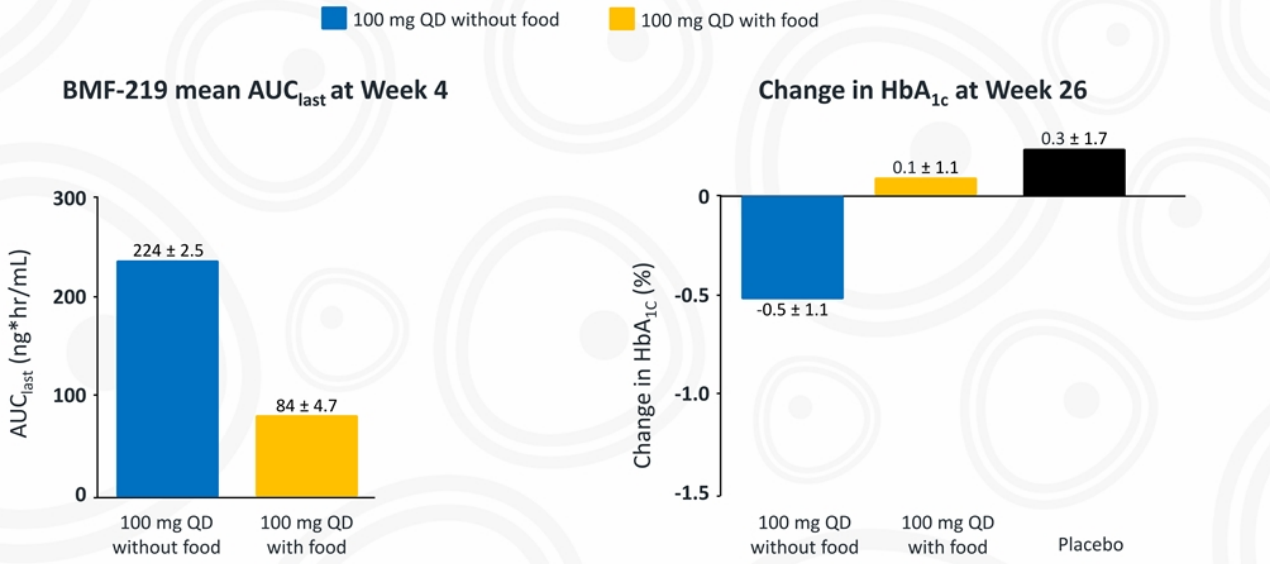
	BMF-219 100mg QD without food (n=10)	BMF-219 100mg QD with food (n=10)	Placebo (n=6)
Age (year, min-max)	52 (38-63)	51 (35-60)	46 (31-61)
Sex (n, M/F)	6/4	7/3	6/0
Duration of diabetes (year, min-max)	4.2 (0.5-9.0)	8.7 (4.0-14.0)	4.2 (1.0, 10.0)
HbA_{1c} (%-point, SD)	8.1 (0.9)	8.0 (0.6)	8.3 (0.7)
Diet and exercise alone (n, %)	0 (0%)	1 (10%)	0 (0%)
1 antihyperglycemic agent (n, %)	9 (90%)	7 (70%)	5 (83%)
2 antihyperglycemic agent (n, %)	0 (0%)	2 (20%)	1 (17%)
3 antihyperglycemic agent (n, %)	1 (10%)	0	0 (0%)

Glycemic Results Summary at Week 26

	BMF-219 100mg QD without food (n=10)	BMF-219 100mg QD with food (n=10)	Placebo (n=6)
Mean change in HbA_{1c}	-0.5%	0.1%	0.3%
Placebo adjusted mean change in HbA_{1c}	-0.8%	-0.2%	-
Percent of participants with ≥1.0% reduction in HbA_{1c}	20%	20%	0%

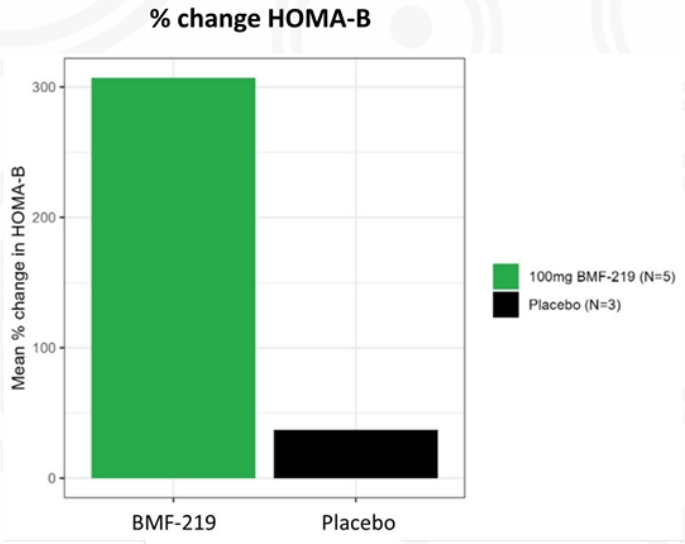
Percent of participants with any reduction in HbA_{1c}: 80% (BMF-219 100mg QD without food) and 40% (BMF-219 100mg QD with food)

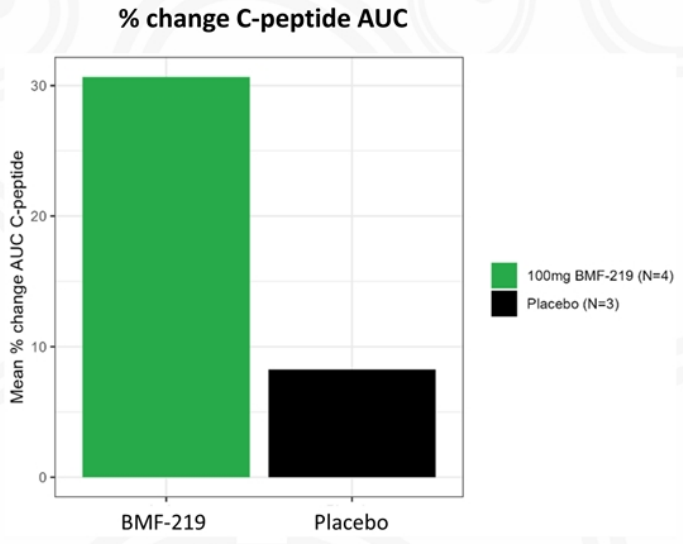
Greater BMF-219 Exposure at Week 4 Resulted in Greater Reduction in HbA_{1c} at Week 26



% Increase in HOMA-B and C-peptide AUC in Responders

Patients with HbA_{1c} reduction ≥0.5% at Week 26 and baseline HOMA-B <200





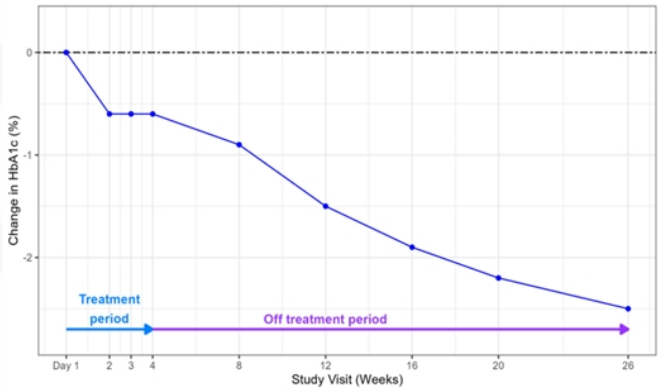
COVALENT-111 Phase 2 Study (Type 2 Diabetes)

Case Study 2: 29-Year-Old Man with 4-Year History of T2D

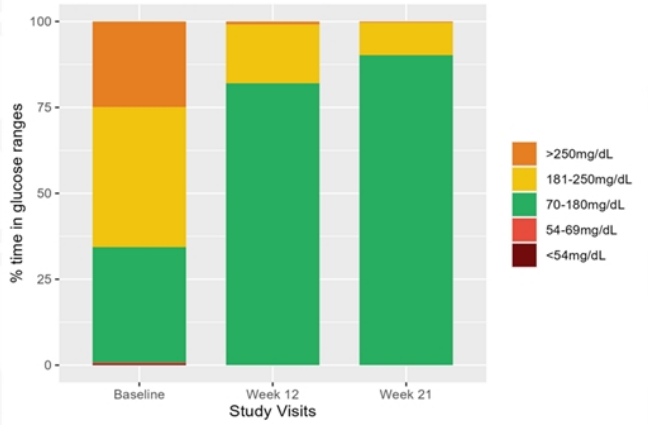
- 29-year-old man with 4-year history of T2D
- Metformin and empagliflozin
- HbA_{1c} 9.5%; FPG 146 mg/dL; BMI 25.6 kg/m^2

- BMF-219 200 mg once daily without food for 4 weeks
- CGM at Week 21 with ~90% $TIR_{70\text{-}180\ mg/dL}$
- No tolerability issues or related adverse events

Change in HbA_{1c} (%)



Continuous Glucose Monitoring



biomea FUSION We Aim to Cure

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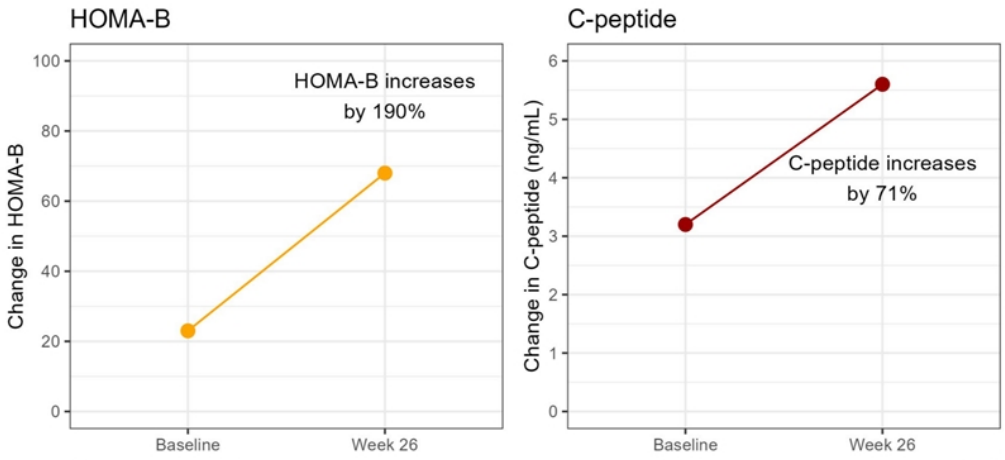
Change at Week 26



2023 Accomplishments

DIABETES

COVALENT-111: Type 2 Diabetes Patients failing standard of care (Metformin, SGLT2, GLP-1, DPP-4)

- 84% of patients responded to BMF-219 while on treatment (any reduction in HbA1c at Week 4)
- 74% of patients continued to respond to BMF-219 despite off-treatment (any reduction in HbA1c at Week 12)
- 20% of patients achieved at least a 1% reduction in HbA1c, 5 months off treatment (100mg @ Week 26)
- 36% of patients achieved at least a 1% reduction in HbA1c, 5 months off treatment (200mg @ Week 26)
- Expansion Cohorts initiated – exploring 8 and 12 weeks of dosing

COVALENT-112: Type 1 Diabetes IND (FDA) & CTA (Health Canada) cleared

ONCOLOGY

COVALENT-101: Relapsed/ Refractory Acute Leukemia

- Initial Phase I topline data with first Complete Responses, including MRD-

COVALENT-103: Relapsed/ Refractory Acute Leukemia

- IND for BMF-500 accepted and first patient in FLT-3 Leukemia enrolled

FUSION™ SYSTEM

- New lab facilities built out to expand in-house capabilities
- Continued development of the Biomea FUSION™ Platform Technology

WE AIM TO CURE

2024 Anticipated Milestones

DIABETES

- COVALENT-111 Phase II - BMF-219 in type 2 diabetes – Dose Escalation Completed
- COVALENT-111 Phase II - BMF-219 in type 2 diabetes - Expansion cohorts fully enrolled (n=216+)
- COVALENT-112 Phase II - BMF-219 in type 1 diabetes - Open Label cohorts fully enrolled (n=40)
- COVALENT-112 Phase II - BMF-219 in type 1 diabetes - Initial proof of concept established

ONCOLOGY

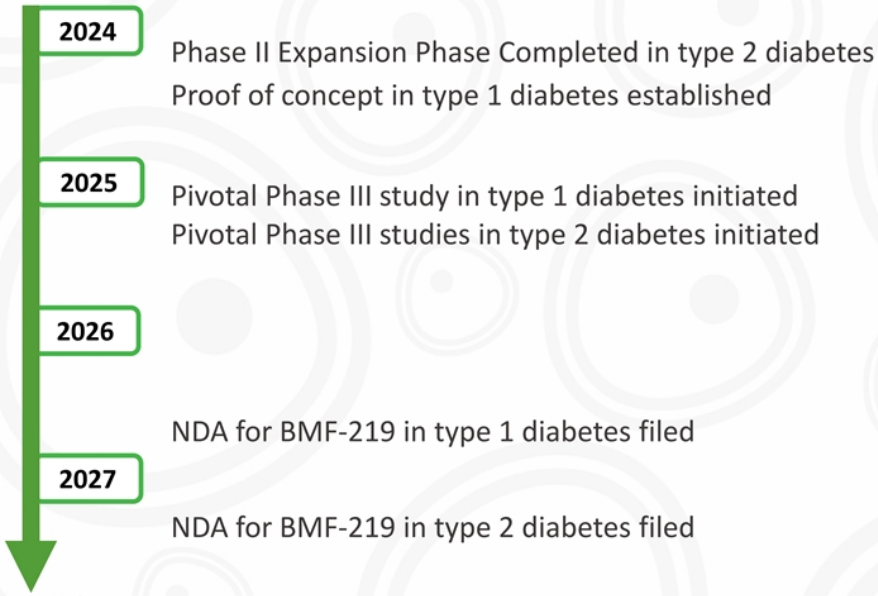
- COVALENT-101 Phase I - BMF-219 in liquid tumors - Dose Escalation Completed and Recommended Phase II Dose established
- COVALENT-102 Phase I - BMF-219 in solid tumors - Dose Escalation Completed and Recommended Phase II Dose established
- COVALENT-103 Phase I - BMF-500 in AML – Dose Escalation Completed and Recommended Phase II Dose established

FUSION SYSTEM

- Third pipeline asset from FUSION™ Platform Technology announced

WE AIM TO CURE

Our Development Plan: Next 4 Years BMF-219 in Diabetes



As of September 30, 2023

Company Financials (NASDAQ: BMEA)

	Three Months Ended September 30, 2023
Operating expenses:	
R&D	$ 25,347
G&A	5,772
Total Operating Expenses	31,119
Loss from operations	(31,119)
Interest and other income, net	2,690
Net loss	$ (28,429)
Other comprehensive loss:	
Changes in unrealized gain on short term investments, net	—
Comprehensive loss	$ (28,429)
Net loss per common share, basic and diluted	$ (0.80)
Weighted-average number of common shares used to compute basic and diluted net loss per common share	35,653,988

Q3 Operating Expenses minus Stock Based Comp	**$24.8 M**
Cash, Cash Equivalents, Investments, and Restricted Cash as of 30 September 2023	**$199.5M**

THANK YOU

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