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): September 22, 2022

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, CA**	**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, $0.0001 par value	**BMEA**	**The Nasdaq Global Select Market**

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

Emerging growth company ☒

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

Item 8.01. Other Events.

On September 22, 2022, Biomea Fusion, Inc. (the "Company") issued a press release titled, "Biomea Fusion Presents New Translational Data at the European Association for the Study of Diabetes (EASD) 2022 in Animal Models and Ex-vivo Human Donor Islets Further Supporting BMF-219's Potential as an Oral, Long-Acting Treatment for Type 2 Diabetes." The information described in the press release was also presented in a presentation at the 58th European Association for the Study of Diabetes (EASD) Annual Meeting, which is taking place September 19-23, 2022 in Stockholm, Sweden.

Copies of the press release and the Company's presentation are attached to this Current Report on Form 8-K as Exhibits 99.1 and 99.2 and incorporated herein by reference.

Forward-Looking Statements

Statements made or incorporated by reference in this Current Report on Form 8-K may include statements which are not historical facts and are considered forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended (the "Securities Act"), and Section 21E of the Securities Exchange Act of 1934, as amended (the "Exchange Act"). These statements may be identified by words such as "aims," "anticipates," "believes," "could," "estimates," "expects," "forecasts," "goal," "intends," "may," "plans," "possible," "potential," "seeks," "will," and variations of these words or similar expressions that are intended to identify forward-looking statements. Any such statements in this press release that are not statements of historical fact, including statements regarding the clinical and therapeutic potential of the Company's product candidates and development programs, including BMF-219, the potential of BMF-219 as a treatment for various types of cancer and diabetes, the Company's research, development and regulatory plans, including the Company's pursuit of BMF-219 in metabolic diseases, its plans to submit an IND application for BMF-219 in type 2 diabetes, and the timing of such events, may be deemed to be forward-looking statements. The Company intends these forward-looking statements to be covered by the safe harbor provisions for forward-looking statements contained in Section 27A of the Securities Act and Section 21E of the Exchange Act and is making this statement for purposes of complying with those safe harbor provisions.

Any forward-looking statements made or incorporated by reference in this Current Report on Form 8-K are based on the Company's current expectations, estimates and projections only as of the date of this Current Report on Form 8-K are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward-looking statements, including the risk that the Company may encounter delays in patient enrollment and in the initiation, conduct and completion of its planned clinical trials and other research and development activities. These risks concerning the Company's business and operations are described in additional detail in its periodic filings with the U.S. Securities and Exchange Commission (the "SEC"), including its most recent periodic report filed with the SEC and subsequent filings thereafter. The Company explicitly disclaims any obligation to update any forward-looking statements except to the extent required by law.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	Description
99.1	Press release titled, "Biomea Fusion Presents New Translational Data at the European Association for the Study of Diabetes (EASD) 2022 in Animal Models and Ex-vivo Human Donor Islets Further Supporting BMF-219's Potential as an Oral, Long-Acting Treatment for Type 2 Diabetes.
99.2	Presentation titled, "Oral Long-acting Menin Inhibitor Normalizes Type 2 Diabetes in Two Rat Models."
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURES

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

BIOMEA FUSION, INC.

Date: September 22, 2022

By: /s/ Thomas Butler

Thomas Butler
Principal Executive Officer

Biomea Fusion Presents New Translational Data at the European Association for the Study of Diabetes (EASD) 2022 in Animal Models and Ex-vivo Human Donor Islets Further Supporting BMF-219's Potential as an Oral, Long-Acting Treatment for Type 2 Diabetes

September 22, 2022, at 9:30 AM EDT

- Treatment with BMF-219 led to an increase in beta cell mass in ex-vivo experiments with human donor islets
- BMF-219 showed improved pancreatic beta-cell function and beta cell area, insulin sensitivity, blood lipid levels, weight decline, and glycemic control in the rat models (ZDF (Zucker Diabetic Fatty Rat) and the STZ (streptozotocin)) during the dosing period, and importantly, both glycemic and lipemic control were maintained after the dosing period ended
- BMF-219 treatment resulted in a sustained increase in beta cell area and function in the ZDF diabetic rats observed at the end of treatment and two weeks following succession of therapy, compared to rats treated with vehicle or active control pioglitazone, which showed a decline in beta cell area and function
- BMF-219 demonstrated, after 16 days of treatment, a recovery of beta-cell activity in STZ rats compared to rats treated with vehicle control or pioglitazone
- Biomea remains on track to file an IND to explore the utility of BMF-219 in type 2 diabetes in the second half of 2022

REDWOOD CITY, Calif., Sept. 22, 2022 (GLOBE NEWSWIRE) — Biomea Fusion, Inc. (Nasdaq: BMEA), a clinical-stage biopharmaceutical company dedicated to discovering and developing novel covalent small molecules to treat and improve the lives of patients with genetically defined cancers and metabolic diseases, presented "Oral long-acting menin inhibitor normalizes type 2 diabetes in two rat models" at the European Association for the Study of Diabetes Annual Meeting (EASD).

The presentation highlighted BMF-219's ability to improve pancreatic beta cell area and function in two preclinical rat models of diabetes. This data showed BMF-219's robust and prolonged glycemic control, insulin sensitization, and reduction of weight and lipid levels in preclinical rat models.

In addition, and for the first time, Biomea Fusion presented data showing an increase in beta cell function based on HOMA-B determination in the two rat models and an increase in beta-cell area in the ZDF rat model and an increase in beta cell mass in an ex-vivo human donor beta-cell islet model. This data provides further support for the potential of BMF-219 as a long-acting, disease modifying treatment for type 2 diabetes. The preclinical presentations from EASD can be viewed on Biomea's website at https://biomeafusion.com/publications.

"Today we reported direct evidence of BMF-219's ability to increase beta cell area, another key feature of the novel mechanism. BMF-219 also displayed the ability to restore and preserve beta cell function in various type 2 diabetes models. In addition, we have continued to expand our translational work and presented a preview of the results today, showing treatment with BMF-219 led to a substantial expansion of human beta cells in an ex-vivo donor islet model," said Thomas Butler, CEO and Chairman of Biomea Fusion.

Diabetes is a multi-factorial disease that typically requires daily treatment with multiple agents with various mechanisms of action to achieve glycemic control. The global burden of diabetes has been rapidly increasing with over 400 million people currently struggling with diabetes globally.

"These results, along with additional work, increases our understanding and our confidence in BMF-219's ability to restore and balance beta cell mass effectively and efficiently. We are excited about the potential impact a treatment like this can have on the lives of the millions of patients worldwide with diabetes. Today, available therapies only provide symptomatic control whereas BMF-219 has the potential to be disease modifying. We look forward to updating you on our clinical development plan in diabetes in the coming months," commented Mr. Butler

The data presented at EASD shows that BMF-219 was superior to pioglitazone in an oral glucose tolerance test (OGTT), an assessment of glucose metabolism and processing, in the STZ beta cell ablation model during the treatment period. In this model, BMF-219 but not active comparator, pioglitazone, restored non-fasting glucose levels to near normal baseline by treatment day eight and significantly reduced blood glucose levels vs. vehicle and pioglitazone during the OGTT in STZ rats (mean AUC reduction of 41%, $p<0.05$) at day 15, suggesting that BMF-219 rapidly

induced pancreatic beta cell regrowth and function. Furthermore, BMF-219 administration resulted in a near doubling of HOOMA-B value compared to vehicle control and pioglitazone in the STZ rat model.

In the other gold standard type 2 diabetes model, ZDF rats treated with BMF-219 exhibited an increase in beta cell mass and function while on BMF-219 and for two weeks after BMF-219 was removed. ZDF rats treated with vehicle and pioglitazone saw a reduction in IHC-Insulin staining, a proxy for beta cell mass of -33% and -28%, respectively, while rats treated with BMF-219 saw an increase of +2%.

Two weeks after BMF-219 treatment, the ZDF rats continued to see a normalization of their HOMA-B score, with approximately 350% improvement compared to vehicle treated ZDF rats, which remained highly diabetic with poor beta cell function (~75% below the normal range). In the ZDF model, BMF-219 and pioglitazone showed similar glycemic control during an OGTT while drug was present (AUC reduction of 54%, $p<0.001$) but only BMF-219 treated rats saw weight loss during treatment with BMF-219 and maintained glycemic control two weeks after washout (AUC reduction of 40%, $p<0.05$), indicating prolonged glycemic control. In addition to OGTT, blood glucose, insulin, C-peptide, Hemoglobin A1C (HbA1c), lipemic levels, and weight were also assessed.

We believe these data collectively demonstrate the novel, long-acting potential of BMF-219 as a single agent, disease modifying oral treatment for type 2 diabetes.

About Menin in Diabetes

Loss of functional beta-cell mass is a core component of the natural history in both types of diabetes — type 1 diabetes (mediated by autoimmune dysfunction) and type 2 diabetes (mediated by metabolic dysfunction). Beta-cells are found in the pancreas and are responsible for the synthesis and secretion of insulin. Insulin is a hormone that helps the body use glucose for energy and helps control blood glucose levels. In patients with diabetes, beta-cell mass and function are diminished, leading to insufficient insulin secretion and hyperglycemia. 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 regeneration of normal healthy beta-cells. Based on these and other scientific findings, Biomea explored the potential for menin inhibition as a viable therapeutic approach to permanently halt or reverse progression of type 2 diabetes.

About Biomea Fusion

Biomea Fusion is a clinical stage biopharmaceutical company focused on the discovery and development of covalent small molecules to treat patients with genetically defined cancers and metabolic diseases. A covalent small molecule is a synthetic compound that forms a permanent bond to its target protein and offers a

number of potential advantages over conventional non-covalent drugs, including greater target selectivity, lower drug exposure, and the ability to drive a deeper, more durable response. The company is utilizing its proprietary FUSION™ System to advance a pipeline of covalent-binding therapeutic agents against key oncogenic drivers of cancer and metabolic diseases. Biomea Fusion's goal is to utilize its capabilities and platform to become a leader in developing covalent small molecules in order to maximize the clinical benefit when treating various cancers and metabolic diseases.

Forward-Looking Statements

Statements we make in this press release may include statements which are not historical facts and are considered forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended (the "Securities Act"), and Section 21E of the Securities Exchange Act of 1934, as amended (the "Exchange Act"). These statements may be identified by words such as "aims," "anticipates," "believes," "could," "estimates," "expects," "forecasts," "goal," "intends," "may," "plans," "possible," "potential," "seeks," "will," and variations of these words or similar expressions that are intended to identify forward-looking statements. Any such statements in this press release that are not statements of historical fact, including statements regarding our cash runway, the clinical and therapeutic potential of our product candidates and development programs, including BMF-219, the potential of BMF-219 as a treatment for various types of cancer and diabetes, our research, development and regulatory plans, including our pursuit of BMF-219 in metabolic diseases, and the timing of such events, may be deemed to be forward-looking statements. We intend these forward-looking statements to be covered by the safe harbor provisions for forward-looking statements contained in Section 27A of the Securities Act and Section 21E of the Exchange Act and are making this statement for purposes of complying with those safe harbor provisions.

Any forward-looking statements in this press release are based on our current expectations, estimates and projections only as of the date of this release and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward-looking statements, including the risk that we may encounter delays or unforeseen results in preclinical development, IND-filing and acceptance, patient enrollment and in the initiation, conduct and completion of our planned clinical trials and other research, development and regulatory activities. These risks concerning Biomea Fusion's business and operations are described in additional detail in its periodic filings with the U.S. Securities and Exchange Commission (the "SEC"), including its

most recent periodic report filed with the SEC and subsequent filings thereafter. Biomea Fusion explicitly disclaims any obligation to update any forward-looking statements except to the extent required by law.

Contact:

Sasha Blaug
SVP Corporate Development
sb@biomeafusion.com
(650) 460-7759

Presentation # 197

Exhibit 99.2

Oral long-acting menin inhibitor normalizes type 2 diabetes in two rat models

*Thomas Butler, Sanchita Mourya, Weiqun Li, Tenley Archer, Taisei Kinoshita, Mini Balakrishnan, Priyanka Somanath

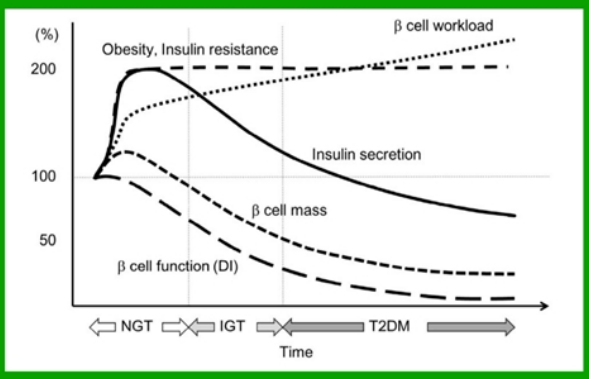
EASD Annual Meeting 2022
Oral Presentation
Golgi Hall
Session: OP 33 Therapy outside the box

September 22, 2022

biomea FUSION

Our understanding of diabetes has evolved; Progression of type 1 and type 2 diabetes are both driven by beta cell loss

Progression of Type 2 Diabetes



*Insulin Resistance leads to an increase in Beta Cell Workload, which ultimately leads to Beta Cell Failure and Death and the progression of Type 2 Diabetes.

"Understanding of Diabetes has evolved"

	Type 1 diabetes	Type 2 diabetes
Past	β cell destruction β cell mass ↓↓ Insulin secretion ↓↓	Obesity Insulin resistance Hyperinsulinemia
Now	Type 1 diabetes	Type 2 diabetes
	β cell destruction β cell mass ↓↓ Insulin secretion ↓↓	β cell loss β cell mass ↓ Insulin secretion ↓
Causes	Autoimmune	Insulin resistance β cell overwork

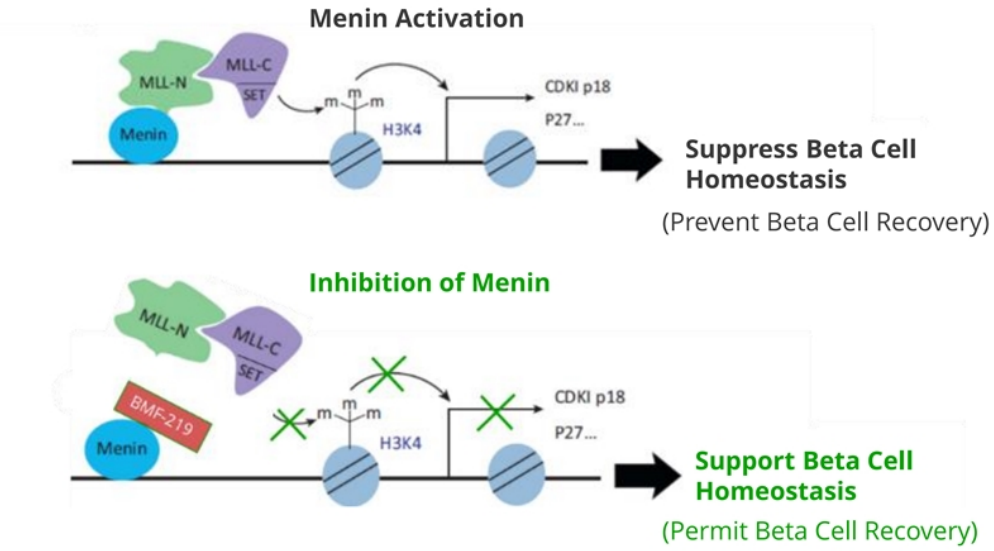
*Both Type 1 Diabetes and Type 2 Diabetes disease results in Beta Cell loss and reduction in Beta Cell Mass

Disease Modifying Potential: BMF-219 drives Beta Cell Proliferation

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

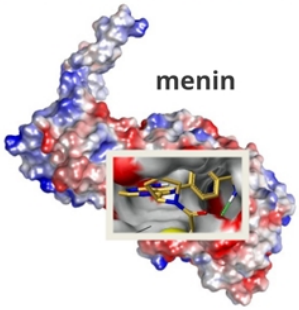
Menin: A key checkpoint for beta cell homeostasis; an important target for type 1 and type 2 diabetes

- Menin functions in a histone methyltransferase protein complex containing MLL
- This complex promotes trimethylation of histone H3 on lysine 4 (H3K4), which is associated with transcriptionally active chromatin and..
- Menin dependent histone methylation maintains expression of p27 and p18, two key members of cyclin-dependent kinase (CDK) inhibitor family that prevent β-cell proliferation.

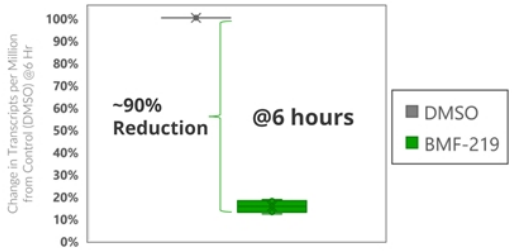


Sources: Karnik et al., Science, Nov 2007, Vol 318 P806-809

BMF-219, A potent & selective covalent menin inhibitor



MEN1 Gene Expression Decreases w/ BMF-219 Treatment



BMF-219 exerts transient changes in Menin Protein

Menin Half Life Varies By Compartment

Half Life in Cytoplasm: <1hr

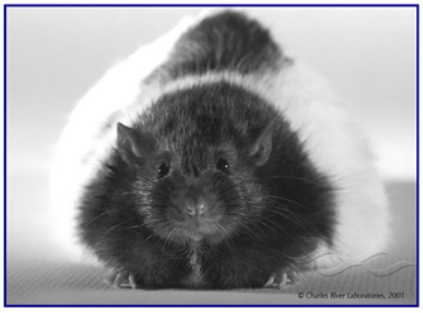
Half Life in Nucleus: 6-8 hrs

Menin's half-life in nucleus is most relevant for pharmacological intervention

- BMF-219 produces **robust decrease in expression of target protein** (Menin)
- **Effect continues beyond established nuclear half-life** of menin, indicating robust effect that is not impacted by protein turnover

Zucker Diabetic Fatty Rat –A model of insulin resistance

THE ZDF RAT



- The ZDF rat is a model of pancreatic exhaustion, thus mimicking some aspects of human diabetes.
- Pioglitazone and metformin provide therapeutic efficacy in this model.
- The ZDF rat is a translatable model for studying the development of T2D.

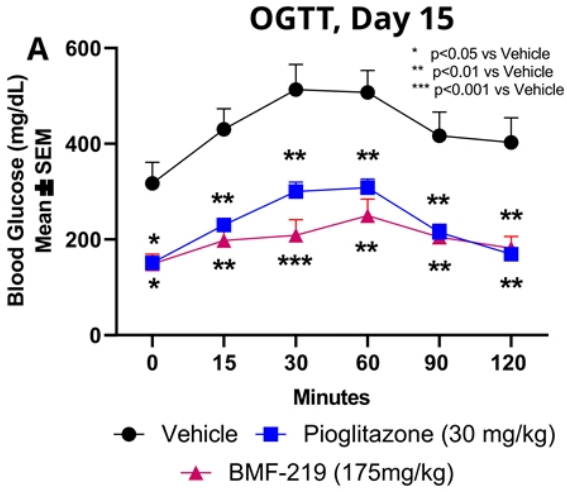
Study Design

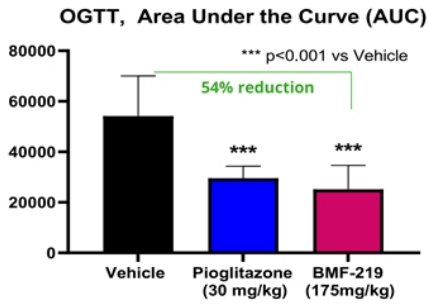
Rats monitored for the following parameters through dosing and washout phases include:
Body weight, fasting blood glucose, blood insulin, C-peptide, and OGTT

Treatment groups (n=10/group):

1. Vehicle
2. BMF-219 175 mg/kg
3. Pioglitazone 30 mg/kg

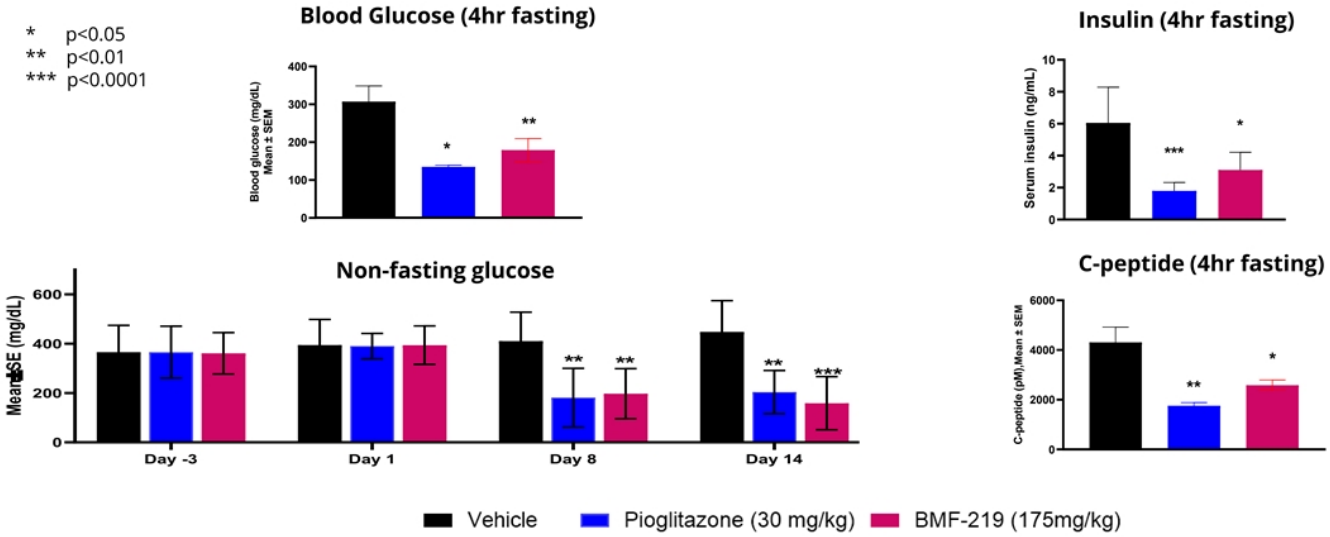
BMF-219 significantly reduces blood glucose by Oral Glucose Tolerance Test (OGTT) in ZDF rats





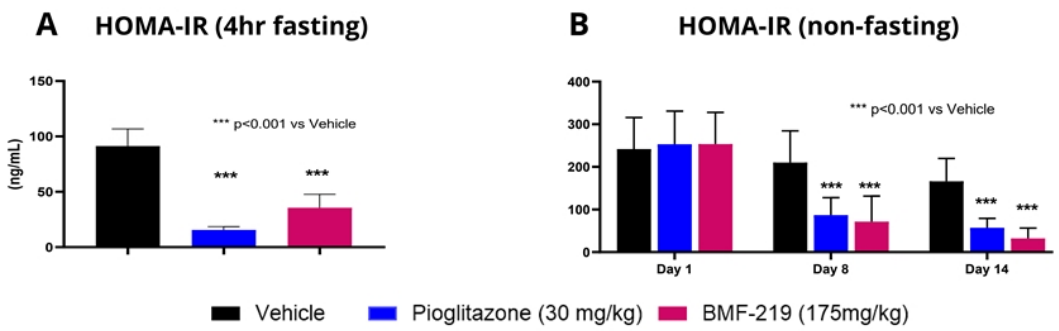
BMF-219 significantly reduces blood glucose levels by OGTT at Day 15 of treatment

BMF-219 significantly reduces blood glucose, insulin, and c-peptide levels in ZDF Rats (After 2 Weeks of Dosing)



BMF-219 significantly reduces blood glucose levels and alters serum insulin and C-peptide levels in ZDF rats @ Day 17

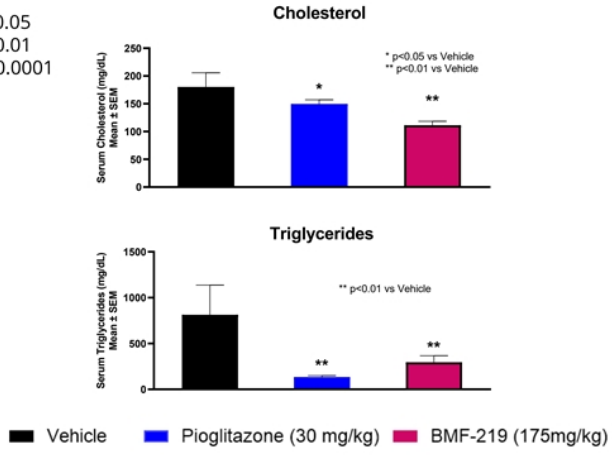
BMF-219 increases insulin sensitivity in ZDF rats

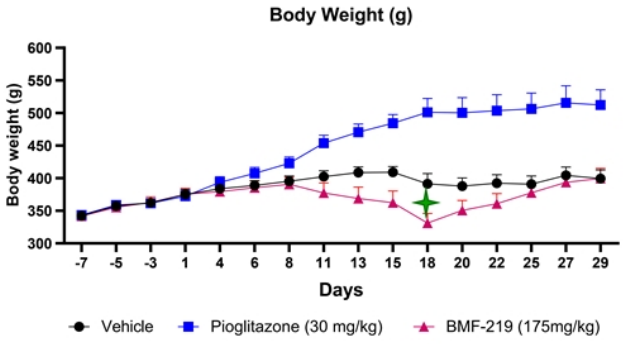


Measurement of HOMA-IR in rats treated with BMF-219 for 16 Days

BMF-219 significantly reduces blood lipemic levels and reduces body weight at day 17

* p<0.05
** p<0.01
*** p<0.0001



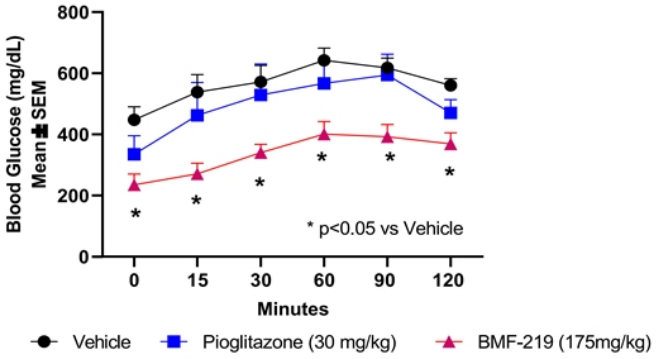


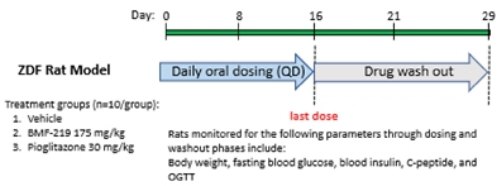
15% reduction after 2 weeks dosing

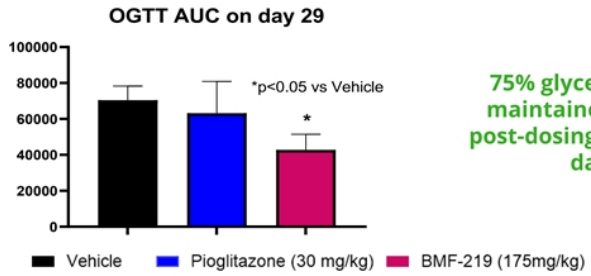
Measurement of cholesterol, triglycerides and body weight in BMF-219 treated rats for 16 days. (Animals continued to eat a high caloric diet until Day 31).

BMF-219 displays durable glycemic control during drug washout, two weeks after the last dose

After 2-week Drug Washout





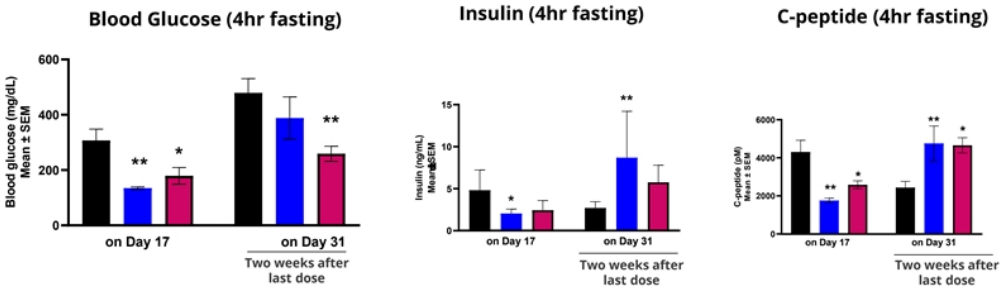


75% glycemic control maintained on day 29 post-dosing compared to day 15

ZDF rats treated with BMF-219, pioglitazone or vehicle control for 16 days were monitored for blood glucose levels by OGTT on day 29, ~2 weeks after administration of the last dose, displaying an AUC reduction of 40%, ($p<0.05$).

BMF-219 maintains a strong impact on blood glucose, insulin and c-peptide levels during drug washout, two weeks after the last dose

Day 31: After 2-week Drug Washout



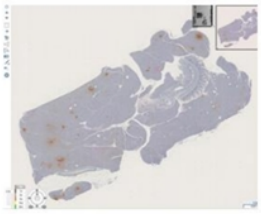
* p<0.05
** p<0.01
*** p<0.0001

BMF-219 maintains significant impact on blood glucose, insulin and c-peptide levels during drug washout (two weeks after last dose)

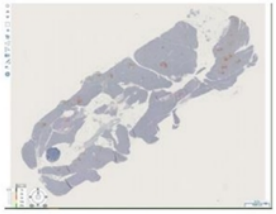
(Animals continued to eat a high caloric diet until Day 31)

BMF-219 increases β-islets in pancreas sections of ZDF diabetic model

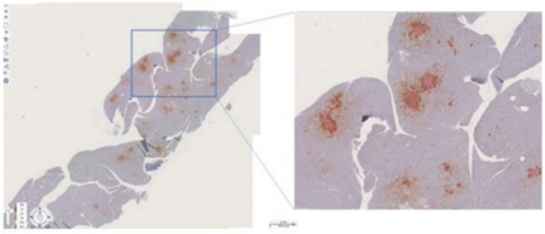
A. Vehicle; Day 31



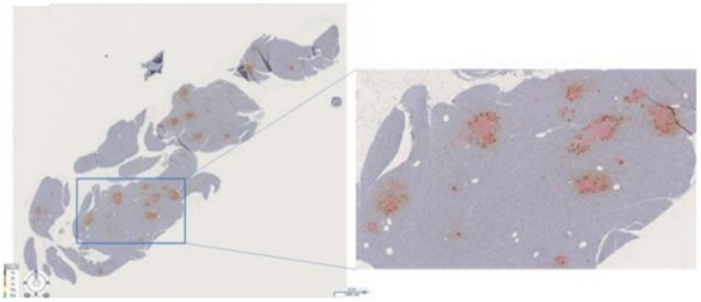
B. Pioglitazone; Day 17



C. BMF-219; Day 17

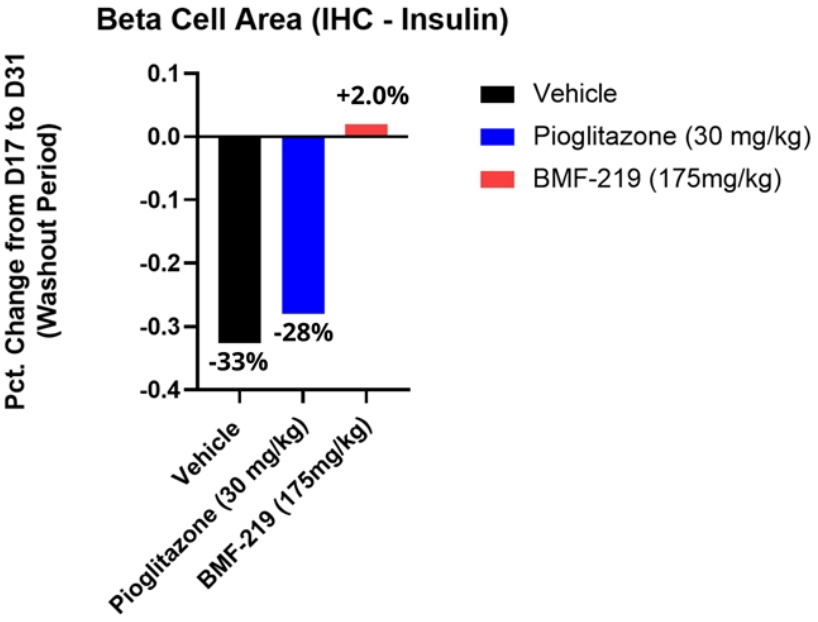


D. BMF-219; Day 31



A) Vehicle-treated animal, Day 31. Beta islets display low congregation and growth, while alpha cells dominate. **B)** Pioglitzaone-treated animal, Day 17. Beta islets display congregation and growth. **C)** BMF-219 treated animal, Day 17. In contrast to the pioglitazone-treated animal shown in Panel B, note that BMF-219 treatment results in high congregation and growth of the beta islets. **D)** BMF-219 treated animal, Day 31. Beta islets display high congregation and continue to increase and mature. Red is insulin-beta islets, brown is glucagon-alpha cells.

BMF-219 increases β-islets in pancreas sections of ZDF diabetic model

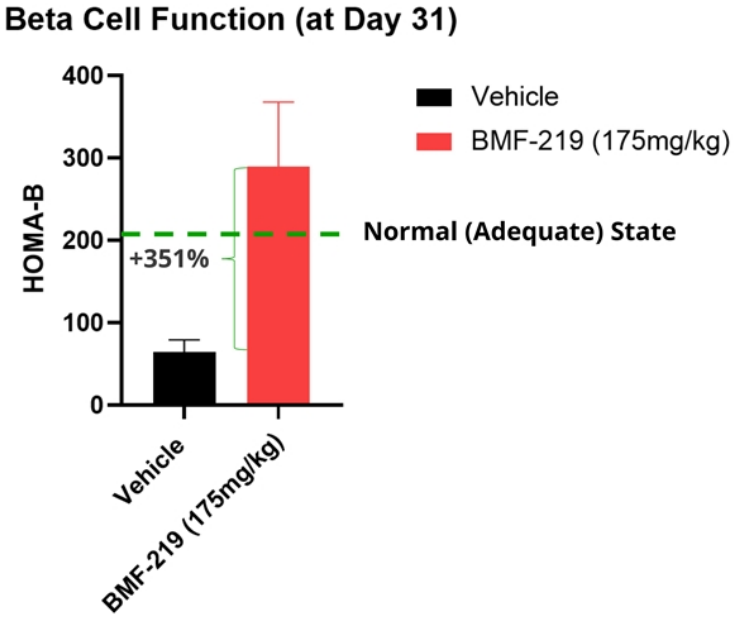


Quantitative Analysis of pancreatic islet tissue cross sections shows BMF-219 treated animals show novel effects in Beta Cell Area growth and maintenance.

BMF-219 was able to maintain Beta Cell function and prevent Beta Cell Area Loss in an Insulin Resistance Type 2 Diabetes Model.

Importantly, Beta Cell Area is maintained, despite cessation of dosing.

BMF-219 demonstrates strong β-cell activity, supporting quantitative analysis



HOMA-Beta, a measurement of Beta Cell Function, was analyzed using 4-hr fasting glucose and insulin levels in animal plasma.

BMF-219 displayed a significant level of Beta Cell function compared to vehicle at Day 31 in an Insulin Resistance Type 2 Diabetes Model.

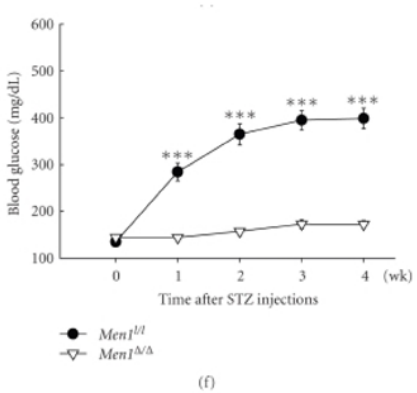
This data supports the observed results from the Beta Cell Area Quantitative Analysis using IHC. Importantly, Beta Cell Function is observed despite cessation of dosing.

O.J. Fasipe et al. / Can J Diabetes 44 (2020) 663e669

Streptozotocin (STZ) Rat, A type 2 diabetes model of beta cell loss

MEN1 Excision Prevents Development of STZ-induced Hyperglycemia

- Menin is a scaffold protein, encoded by the gene *MEN1*, that has been recently recognized for its role in Type 2 Diabetes Mellitus (T2DM) as a key regulator of b-cell proliferation.
- *Men1* knockout mice demonstrate increased B-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.

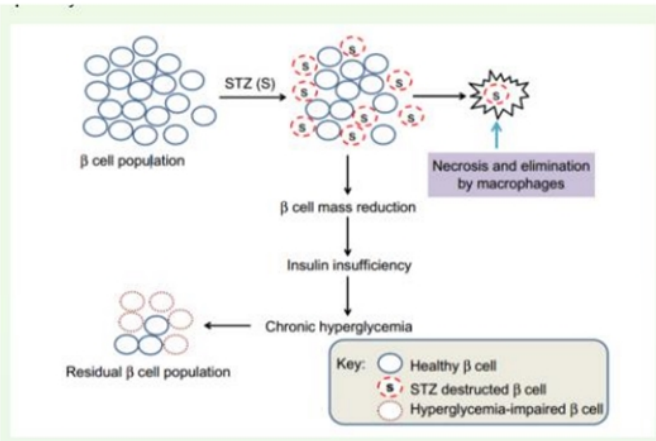


(f)

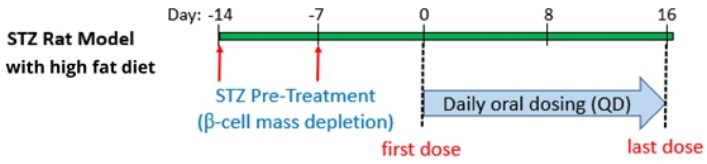
***Men1*-excised mice did not develop hyperglycemia in STZ model, which was observed in the control group**

STZ Rat Model- Study Design

The Streptozotocin (STZ)-Induced Rat Model
Only direct insulin injection shows an effect in this model



Study Design

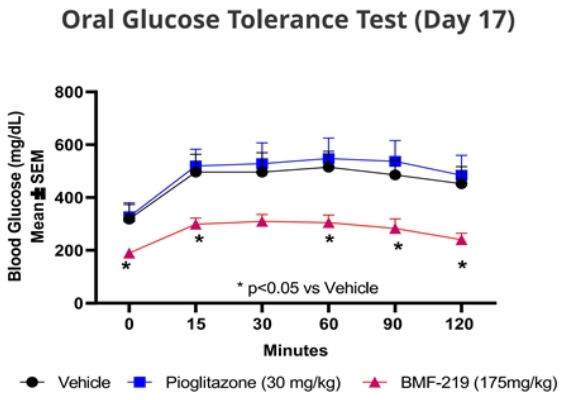


Treatment groups (n=10/group):
1. Vehicle
2. BMF-219 175 mg/kg
3. Pioglitazone 30 mg/kg

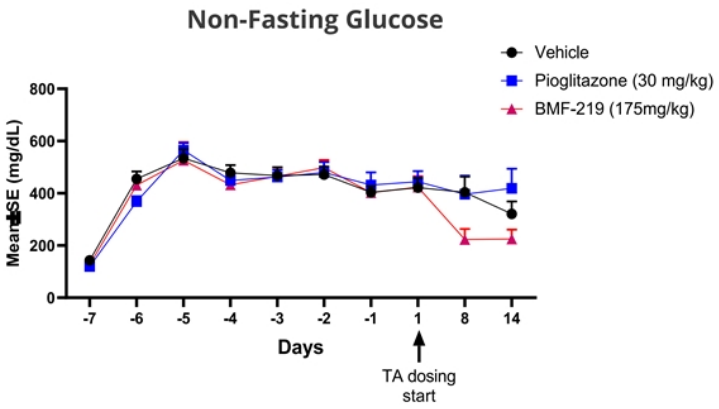
Rats monitored for the following parameters through dosing include: OGTT, blood glucose levels

STZ treatment typically results in ~50% Beta Cell Loss

BMF-219 significantly reduced blood glucose by Oral Glucose Tolerance Test (OGTT) in an STZ-induced rat model



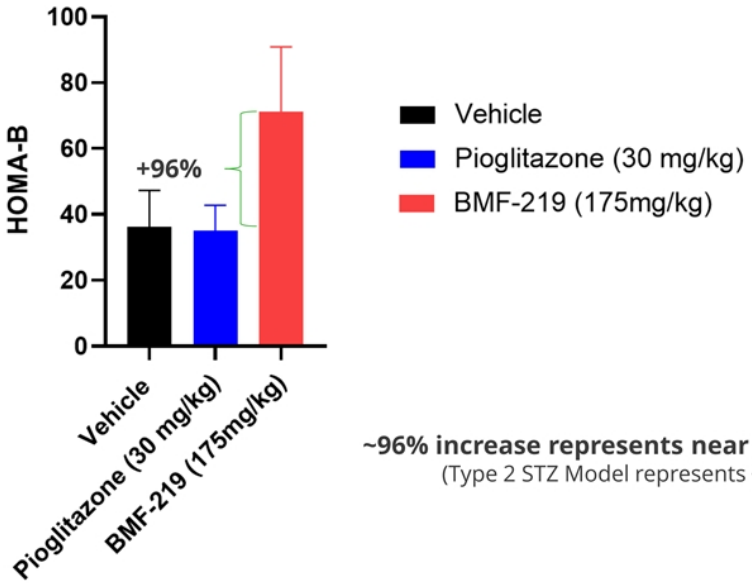
Only BMF-219 significantly reduces blood glucose by OGTT in STZ rats



Only BMF-219 lowered non-fasting glucose in STZ rats

BMF-219 demonstrates recovery of β-cell activity

Beta Cell Function (at Day 17)



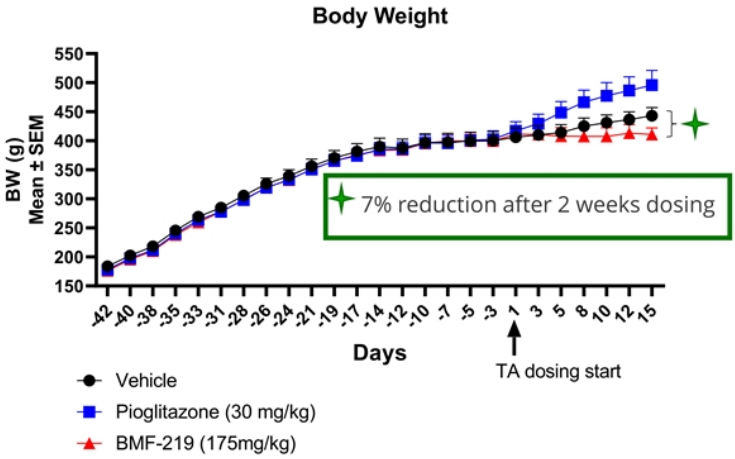
HOMA-Beta, a measurement of Beta Cell Function, was analyzed using 4-hr fasting glucose and insulin levels in animal plasma.

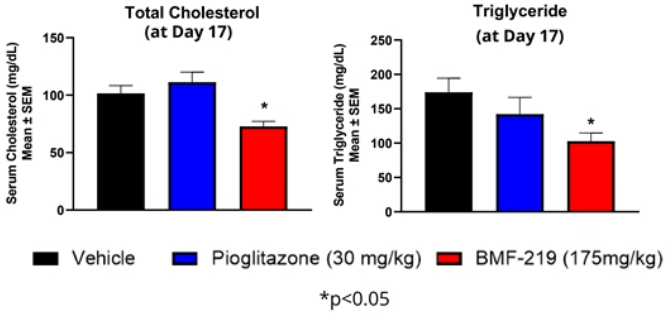
BMF-219 displayed a significant level of Beta Cell function compared to vehicle at Day 17 in a Beta Cell Type 2 Diabetes Model.

This data supports the observed results from the Beta Cell Mass Quantitative Analysis using IHC. Importantly, Beta Cell Function is observed despite cessation of dosing.

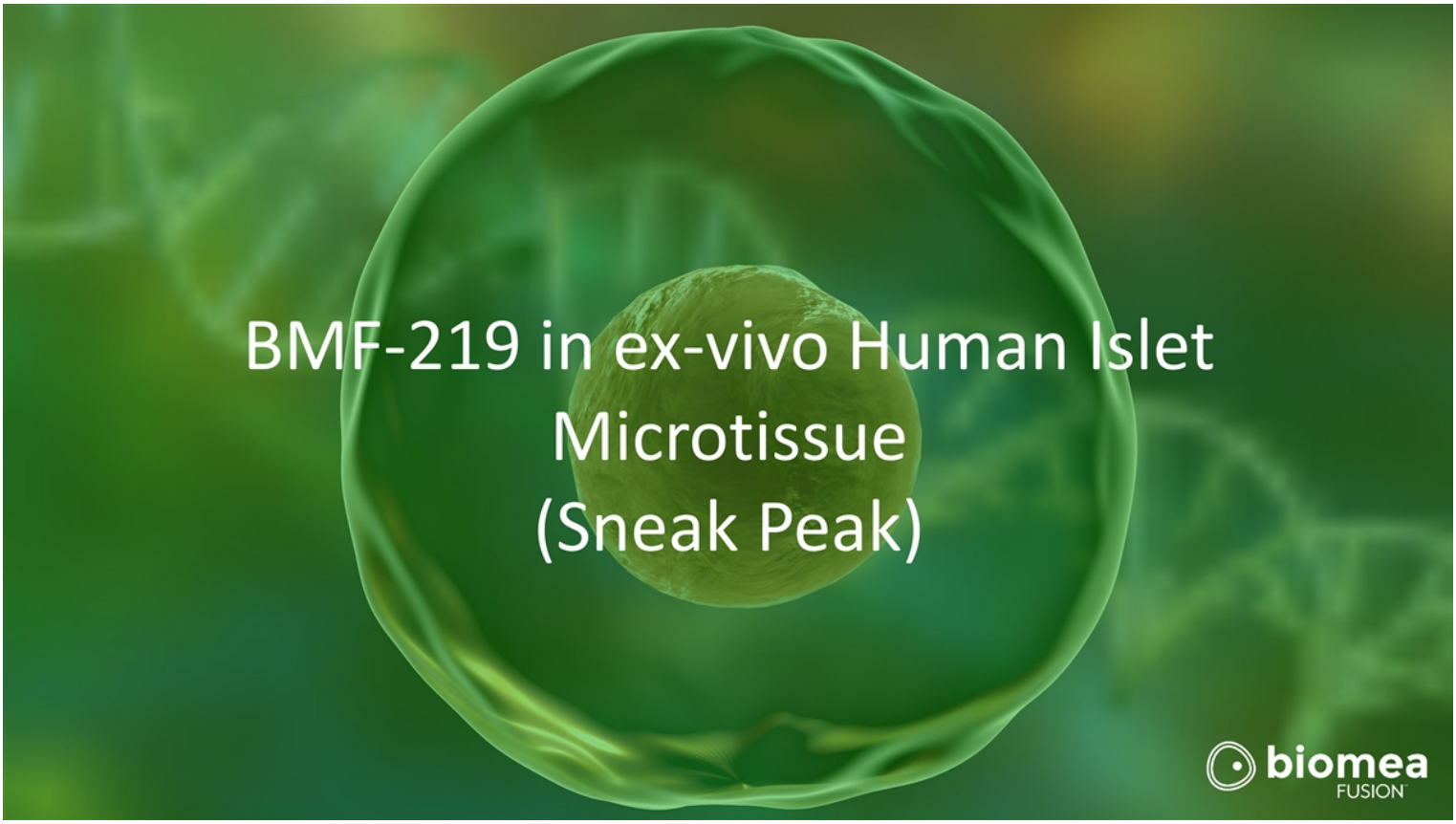
~96% increase represents near doubling of beta cell function
(Type 2 STZ Model represents ~ 50% Beta Cell Destruction)

BMF-219 significantly reduces blood lipemic levels and body weight in STZ rats

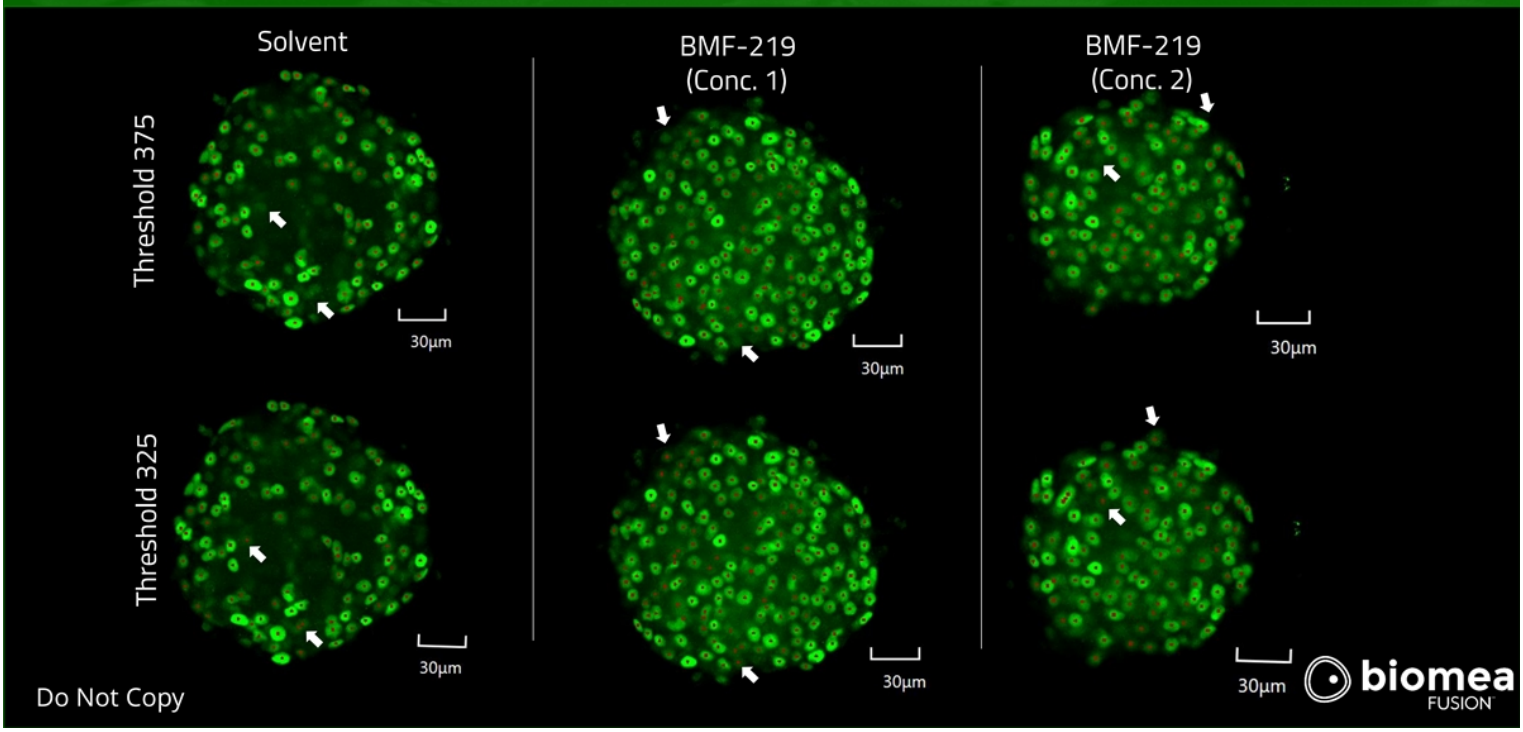




*p<0.05


BMF-219 in ex-vivo Human Islet Microtissue (Sneak Peak)
biomea FUSION

Human Donor Islets (Ex-Vivo): Statistically significant increase in beta cells with BMF-219



Conclusions & Next Steps

- BMF-219 was tested in two type 2 diabetic in-vivo models at clinically relevant exposures.

BMF-219 treatment in STZ-induced diabetic rat model:

- Improved glucose metabolism (OGTT AUC reduction 41%, $p<0.05$) and reduction in blood lipemic levels and body weight. Minimal efficacy for pioglitazone.
- BMF-219 but not pioglitazone increased HOMA-B.

BMF-219 treatment in ZDF rat model:

- Significant reduction (~50%) in fasting and non-fasting blood glucose levels,
- Significant reduction in serum insulin, C-peptide ($p<0.05$), and HOMA-IR ($p<0.001$) after two weeks of treatment.
- Prolonged glycemic control as evidenced by decreased OGTT glucose levels on day 15 (AUC reduction of 54%, $p<0.001$) and on day 29 (AUC reduction of 40%, $p<0.05$, ~2 weeks after the last dose).
- Significant reductions in blood lipemic levels ($p<0.01$) and body weight.

- Next Steps: Type 1 Model – Can BMF-219 re-establish Beta Cells from a severely diminished pool?


THANK YOU
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