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): June 4, 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.)

650 Main Street 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 June 4, 2022, Biomea Fusion, Inc. (the "Company") presented preclinical data on its product candidate, BMF-219, in chronic lymphocytic leukemia tumor models at the 2022 American Society of Clinical Oncology (ASCO) Annual Meeting.

On June 5, 2022, the Company presented preclinical data on BMF-219 in two diabetic animal models at the American Diabetes Association (ADA) Scientific Sessions.

Copies of the Company's poster presentations are attached to this Current Report on Form 8-K as Exhibits 99.1 through 99.3 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, 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	Poster presentation titled, "Preclinical Activity of Irreversible menin inhibitor, BMF-219, in Chronic Lymphocytic Leukemia."
99.2	Poster presentation titled, "Oral Long-Acting Menin Inhibitor, BMF-219, Normalizes Type 2 Diabetes Mellitus in Two Rat Models."
99.3	Poster presentation titled, "Oral Menin Inhibitor, BMF-219, displays a significant and durable reduction in HbA1c in a Type 2 Diabetes Mellitus Rat Model."
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: June 6, 2022

By: /s/ Thomas Butler

Thomas Butler
Principal Executive Officer

Preclinical Activity of irreversible menin inhibitor, BMF-219, in Chronic Lymphocytic Leukemia

Priyanka Somanath, PhD[1], Daniel Lu, MS [1] Brian Law, BS [1], Lekha Kumar, MS[1], Tenley Archer, PhD, Tripta Rughwani, MS, Taisei Kinoshita, PhD[1], Mini Balakrishnan, PhD[1] and Thomas Butler, MSc MBA[1]
[1]Biomea Fusion, Inc. Redwood City, CA

We Aim to Cure™

Introduction

- Menin is a scaffold protein that drives oncogenic function through transcriptional modulation directed by its various cofactors.
- A previous report demonstrated that menin regulates a distinct set of gene targets independent of its function with the MLL proteins in hematopoiesis and is essential for B-cell maturation (Li et al.*Blood*.2013;122(12):2039-46.).
- Chronic Lymphocytic Leukemia (CLL) is a disease of malignant B lymphocytes, for which standard-of-care agents are generally well tolerated; however, CLL patients with certain genetic backgrounds demonstrate inferior outcomes to these regimens.

- A major driving feature of CLL is overexpression of the anti-apoptotic marker, BCL2. We previously reported the ability of BMF-219, a selective, covalent menin inhibitor, to downregulate the expression of *BCL2* in acute leukemia cells.
- Additionally, we have reported the synergy of BCL2-targeted agent, venetoclax, with BMF-219 in potent cell killing of diffuse large B-cell lymphoma (DLBCL) preclinical models, prompting our exploration of BMF-219 activity in CLL.
- Here, we provide the first preclinical evidence for menin as a therapeutic target in CLL, by demonstrating high potency of BMF-219 against a diverse collection of CLL patient specimens.

Methods

A comprehensive panel of CLL samples isolated from patients with Rai Stages 1 to 3 disease, including relapsed or refractory disease, were cultured *ex vivo* in the presence of BMF-219 or a clinical reversible menin inhibitor to assess the antileukemic activity of the compounds.

Results

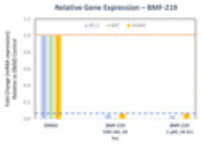
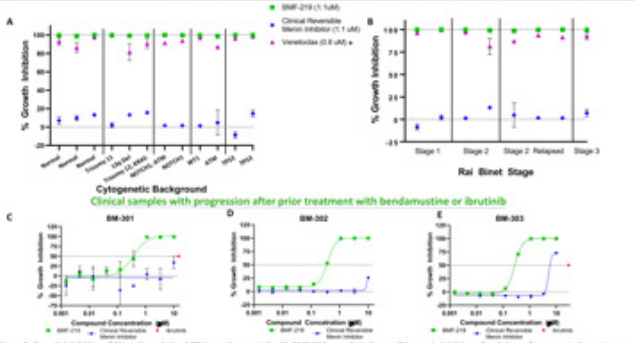


Figure 1. BMF-219 elicits >90% reduction of BCL2 transcript at 24 hours post-treatment in MOLM-13 AML cells. HOXA9 and MYC gene expression was also significantly reduced by >90%. Fold change was calculated relative to vehicle control.

BMF-219 achieves > 98% cell lethality against diverse CLL ex vivo models



Clinical samples with progression after prior treatment with bendamustine or ibrutinib

Figure 2. Growth inhibition of CLL patient-derived PDX samples treated with BMF-219 or a clinical reversible menin inhibitor after 6 days of treatment. Percentage growth inhibition at 1.1 µM BMF-219, 0.8 µM venetoclax and 1.1 µM clinical menin inhibitor are plotted for the PDX samples, as grouped by genetic background (A) or Rai-Binet Stage where data is available (B). Representative dose response curves for BMF-219 or clinical reversible menin inhibitor are shown for PDX samples from CLL patients displaying clinical profiles of progression after prior therapy with bendamustine (C) or ibrutinib (D), or ibrutinib pretreated and subsequently progressed on ibrutinib and venetoclax (E). IC_{50} values are summarized in Table 1. Each data point represents average of at least two replicate values. (*Venetoclax concentration set as standard for positive control). Ibrutinib IC_{50} determined as a standalone experiment.

Table 1. Clinical Profiles of CLL Patient Samples and Response to BMF-219

Sample	Mutation	Cytogenetics	Prior Treatment	Rai Binet Stage	BMF 219 IC_{50} (µM)	BMF 219 % Max Inhibition
BM-301	ATM	Normal	Bendamustine (responded, then progressed)	Stage 3 (Relapsed)	0.373	98.7
BM-302	NOTCH1	Normal	Ibrutinib (responded, then progressed)	Stage 2 (Relapsed)	0.332	99.7
BM-303	TP53	N/A	Ibrutinib (responded), (post-collection: ibrutinib and venetoclax- responded, then progressed)	Stage 1	0.285	99.8
BM-304	None or N/A	46, XX, add(X)(p21), -9, add(9)(p12), +11, der(11;13)(q10;q10), -13,t(15;18)(q15;q21), add(18)(p13.3), add(17)(p11.2) [cp15]/46, XX [5]	Ibrutinib (responded)	Stage 1	0.104	99.9
BM-305	WT1	Normal	Rituximab/Ibrutinib (responded)	Stage 2	0.384	100
BM-306	TP53	Normal	Ibrutinib (responded, no progression)	Stage 3	0.380	100
BM-307	KRAS, KMT2A, TET2	47, XY, +12, t(14;19)(q32;q13.3), t(18;20)(q23;q13.3),q13.1) [13]/46, XY [7]	Rituximab/Methylprednisolone (responded), Ibrutinib	N/A	0.145	99.5
BM-308	None or N/A	46~47, XY, del(6)(q13q25), dic(7;21)(q31;p13), add(15)(q15), del(13)(q12q14), +2mar, inc [cp4]/46, XY [3]	Rituximab/Ibrutinib (responded, continuing)	Stage 2	0.359	99
BM-309	None or N/A	Normal	Ibrutinib (responded)	Stage 3	0.331	100
BM-310	None or N/A	Normal	Ibrutinib (responded)	N/A	0.357	99
BM-311	NOTCH1, ATM	N/A	N/A	Stage 2 (Relapsed)	0.359	100
BM-312	None or N/A	N/A	N/A	N/A	0.384	100

BMF-219 exhibits higher ex vivo potency compared to Standard-of- Care Agents

Sample	Mutation	BMF 219 IC_{50} (µM)	BMF 219 % Max Inhibition	Ibrutinib IC_{50} (µM)	Bendamustine IC_{50} (µM)	Idelasib IC_{50} (µM)
BM-301	ATM	0.373	98.7	14.8	15.6	8.91
BM-302	NOTCH1	0.332	99.7	N/A	N/A	N/A
BM-303	TP53	0.285	99.8	29.1	31.9	37.2
BM-304	None or N/A	0.104	99.9	N/A	N/A	N/A
BM-305	WT1	0.384	100	34.8	17.1	35.7
BM-306	TP53	0.38	100	24.1	6.65	2.21
BM-307	KRAS, KMT2A, TET2	0.145	99.5	18.9	16.1	0.271
BM-308	None or N/A	0.359	99	26.7	13.7	22.7
BM-309	None or N/A	0.331	100	29.0	25.2	38.1
BM-310	None or N/A	0.357	99	12.4	6.84	1.47
BM-311	NOTCH1, ATM	0.359	100	29.1	32.2	35.7
BM-312	None or N/A	0.384	100	N/A	N/A	N/A

Table 2. BMF-219 potency as determined by IC_{50} values in comparison with standard-of-care agents for CLL. CLL ex vivo patient samples were cultured with BMF-219 for 6 days to determine IC_{50} values. IC_{50} values for Ibrutinib (BTK inhibitor), bendamustine (alkylating agent) and idelasib (PI3K inhibitor) were experimentally determined as standalone experiments in these patient models.

Conclusions

- BMF-219 demonstrated high potency, achieving >98% cell lethality at 1.1 µM exposure in all CLL patient samples tested, with IC_{50} values in the range of 0.1 to 0.38 µM, similar to BMF-219 potency in AML and DLBCL ex vivo models.
- Specimens isolated from patients with clinical profiles containing high-risk genetic backgrounds associated with inferior outcomes to standard therapy, such as mutations in TP53 and NOTCH1, and chromosomal aberrations such as del(13q), trisomy 12 and complex karyotype, exhibited high sensitivity to BMF-219 treatment.
- BMF-219 was also highly effective against patient samples with clinical profiles of resistance to bendamustine or ibrutinib therapy.
- A clinical reversible menin inhibitor demonstrated no significant activity across all patient samples tested, with incalculable IC_{50} values and <15% reduction in cell viability at 1 µM exposure.
- Collectively, our data demonstrate the potent preclinical activity of BMF-219 against CLL patient specimens harboring various mutational and cytogenetic backgrounds, including categories of high unmet need, highlighting the unique potential of covalent menin inhibition as a novel therapeutic option for patients with CLL.

References

- Li, W.L., Gan, T., Mayanson, M., et al. Distinct pathways regulated by menin and by MLL1 in hematopoietic stem cells and developing B cells. *Blood* (2013) 122 (12): 2039-2046.
- Somanath, P., Lu, D., Law, B. et al. Novel Irreversible Menin Inhibitor, BMF-219, Shows Potent Single Agent Activity in Clinically Relevant DLBCL Cells. *Blood* (2021). 138 (Supplement 1): 4918.
- Wu, G., Yuan, M., Shen, S. et al. Menin enhances c-Myc-mediated transcription to promote cancer progression. *Nat Commun* **8**, 15278 (2017).

We acknowledge Arpeggio Biosciences for generation of RNA-seq data and Champions Oncology for ex vivo data.

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Oral Long-Acting Menin Inhibitor, BMF-219, Normalizes Type 2 Diabetes Mellitus in Two Rat Models

We Aim to Cure™

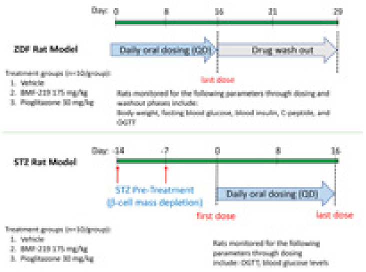
Thomas Butler, MSc MBA[1], Weiqun Li, PhD, Brian Law, BS[1], Tenley Archer, PhD., Taisei Kinoshita, PhD[1], Mini Balakrishnan, PhD[1] and Priyanka Somanath, PhD[1]
[1]Biomea Fusion, Inc. Redwood City, CA

Introduction

- 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 β-cell proliferation.
- *Men1* knockout mice demonstrate increased β-cell mass generation (Yang et al., 2010)
- 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.
- BMF-219 is an orally bioavailable, selective, covalent menin inhibitor that elicits a broad impact on the complexes surrounding menin that also direct its biological function.
- Herein, we report the first evidence that BMF-219 restores glycemic control in Zucker Diabetic Fatty (ZDF) Rat and Streptozotocin-induced Rat (STZ) models of T2DM, with prolonged glycemic control two weeks after dosing in ZDF rats.

Methods

ZDF rats were treated daily with BMF-219, vehicle, or pioglitazone for 16 days and monitored for an additional two weeks post-treatment, until Day 29. STZ rat models were induced through pre-treatment of animals on a high-fat diet with low doses of streptozotocin at day -14 and day -7 prior to starting treatment at Day 0 until Day 16. ZDF and STZ rats were monitored for fasting blood glucose levels, oral glucose tolerance test (OGTT), insulin and C-peptide levels, HOMA-IR (Homeostatic Model Assessment of Insulin Resistance), blood lipemic levels and body weight according to the schematic for each model below. ZDF rats were analyzed for indicated readouts for 15 days post-treatment for OGTT at day 29, and serum insulin and C-peptide at day 31.



BMF-219 demonstrates significant glycemic control in ZDF Rats

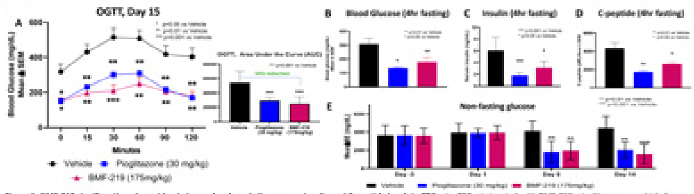


Figure 1. BMF-219 significantly reduces blood glucose levels and alters serum insulin and C-peptide levels in ZDF rats. ZDF rats treated with BMF-219, pioglitazone or vehicle for 16 days were evaluated at various time points. Day 15 OGTT results are shown as time course and AUC (A). Day 17 fasting blood glucose levels (B), fasting insulin (C), and fasting C-peptide (D) are shown. Non-fasting blood sugar was measured weekly on Days -3, 1, 8 and 14 (E). Statistical significance was calculated for treatment groups in comparison with vehicle control.

BMF-219 displays strong glycemic control two weeks after last dose in ZDF Rats

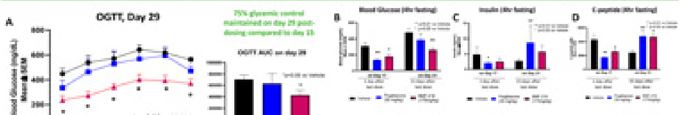


Figure 2. BMF-219 maintains significant impact on blood glucose, insulin and C-peptide levels during drug washout (two weeks after last dose). ZDF rats treated with BMF-219, pioglitazone or vehicle control for 16 days were monitored for blood glucose levels by OGTT on day 29, ~2 weeks after administration of the last dose, displaying an AUC reduction of 40%, (p<0.05) (A), and on day 31 monitored for 4-hour fasting blood glucose (B), fasting serum insulin (C), and fasting C-peptide levels (D). Statistical significance was calculated for treatment groups in comparison to vehicle control.

BMF-219 achieves marked glycemic control in STZ-induced Rats

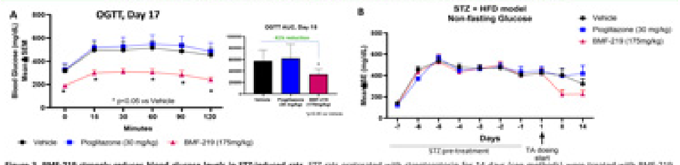


Figure 3. BMF-219 strongly reduces blood glucose levels in STZ-induced rats. STZ rats pretreated with streptozotocin for 14 days (see methods) were treated with BMF-219, pioglitazone or vehicle control for 16 days. Blood glucose was measured by OGTT on Day 17 and displayed an AUC reduction of 41%, (p<0.05) in BMF-219 treated rats only with no change to the pioglitazone treated group (A). Non-fasting glucose levels were measured daily during model establishment (STZ treatment) and weekly during treatment with BMF-219, pioglitazone or vehicle control, displaying reduction of glucose levels in BMF-219 treated rats throughout the duration of treatment (B).

BMF-219 increases insulin sensitivity in ZDF rats

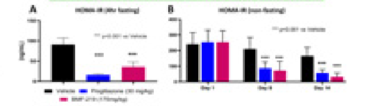


Figure 4. Measurement of HOMA-IR in rats treated with BMF-219 for 16 days. ZDF rats treated with BMF-219, pioglitazone or vehicle were analyzed for HOMA-IR fasting at day 17(A) or non-fasting (B) and values were compared to vehicle control to calculate statistical significance.

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

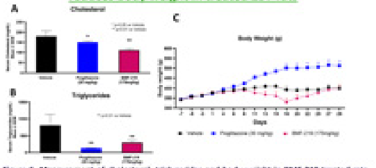


Figure 5. Measurement of cholesterol, triglycerides and body weight in BMF-219 treated rats for 16 days. Cholesterol (A) and triglycerides (B) were measured at Day 17. Body weight was measured daily during treatment and continually monitored two weeks after treatment (C). All groups were treated with vehicle, BMF-219 or pioglitazone and compared to vehicle for statistical analyses. Animals continued to eat a high fat diet until Day 29.

Conclusions

- BMF-219 was tested in rats at clinically relevant exposures.
- BMF-219 treatment resulted in a significant reduction (~50%) in fasting and non-fasting blood glucose levels, significantly reduced serum insulin and C-peptide levels (p<0.05), and reduced HOMA-IR (p<0.001) after two weeks of treatment in ZDF rats.
- BMF-219 showed prolonged glycemic control as evidenced by decreased glucose levels during an oral glucose tolerance test on day 15 (AUC reduction of 54%, p<0.001) and on day 29 during the drug washout period (AUC reduction of 40%, p<0.05, ~2 weeks after the last dose) in the ZDF model, indicating durable glycemic control.
- Strikingly, BMF-219, but not pioglitazone, reduced blood glucose levels by OGTT in STZ animals (AUC reduction of 41%, p<0.05).
- Significant reductions in blood lipemic levels (p<0.01) and body weight were observed in both models.
- Collectively, our data indicate the novel and marked potential of BMF-219 as an oral, long-acting treatment for T2DM.

References

Acknowledgements – We acknowledge Charles River Laboratories for animal studies.
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Oral Menin Inhibitor, BMF-219, displays a significant and durable reduction in HbA1c in a Type 2 Diabetes Mellitus Rat Model

Priyanka Somanath, PhD[1], Sanchita Mourya, MD[1], Weiqun Li, PhD[1], Brian Law, BS[1], Tenley Archer, PhD, Daniel Lo, MS, Trista Rughwani, Lekha Kumar, Taisei Kinoshita, PhD[1], Mini Balakrishnan, PhD[1] and Thomas Butler, MSc MBA[1]
[1]Biomea Fusion, Inc. Redwood City, CA

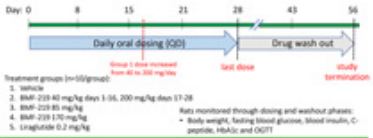
We Aim to Cure™

Introduction

- Menin is a scaffold protein encoded by the gene, *MEN1*, that regulates diverse cellular processes in a tissue-context dependent manner.
- Menin plays a key role in beta-cell proliferation and function, as previously demonstrated though increased beta-cell mass generation in *Men1* knockout mice (Ma et al., 2021).
- The menin-MLL interaction also plays a role in suppressing islet cell growth through control of cell cycle inhibitor expression.
- Importantly, menin inhibition has been shown to improve β-cell proliferation and glycemic control in high fat-induced diabetic mice (Ma et al., 2021).
- BMF-219 is an orally bioavailable, selective, covalent menin inhibitor that elicits a broad impact on the complexes surrounding menin, which direct its biological function.
- Here, we demonstrate the marked potential of an oral menin inhibitor, BMF-219, in achieving durable glycemic control following a short course treatment in a Type 2 Diabetes Mellitus (T2DM) Zucker Diabetic Fatty Rat model.

Methods

Zucker Diabetic Fatty (ZDF) rats were dosed daily with BMF-219, liraglutide or vehicle for 28 days (n=10 per group) and monitored for an additional 28 days post last dose as shown in scheme below. Group 1 dose (40 mg/kg) was increased to 200 mg/kg on day 17 for rest of the dosing phase. Fasting blood glucose, insulin, C-peptide levels, HbA1c, oral glucose tolerance test (OGTT) and body weight were monitored during and post-treatment.



Treatment groups (n=10/group):
1. Vehicle
2. BMF-219 40 mg/kg days 1-16, 200 mg/kg days 17-28
3. BMF-219 85 mg/kg
4. BMF-219 170 mg/kg
5. Liraglutide 0.2 mg/kg

Rats monitored through dosing and washout phases:
- Body weight, fasting blood glucose, blood insulin, C-peptide, HbA1c and OGTT

Results

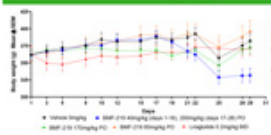


Figure 1. Body weight of ZDF rats during the 28 days of treatment with BMF-219, liraglutide, or vehicle control. Data represents mean SEM for the dose group.

BMF-219 significantly reduces HbA1c and controls blood glucose levels in a 4-week dosing study in ZDF rats

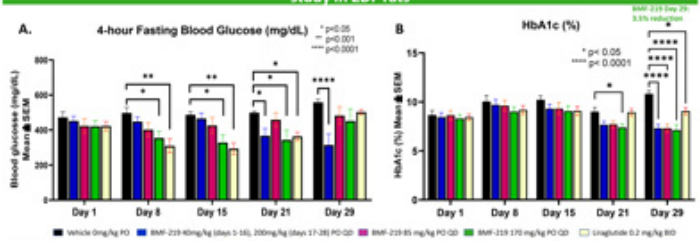


Figure 2. Reduction in fasting blood glucose and HbA1c levels in BMF-219 treated ZDF rats. Rats treated with BMF-219 at indicated doses, liraglutide, or vehicle control were monitored for 4-hour fasting glucose (A) and HbA1c (B) was calculated for treated animals weekly over a 28-day treatment. Changes in blood glucose or HbA1c were compared to vehicle control to calculate statistical significance.

BMF-219 displays durable glycemic control over 4 weeks of dosing

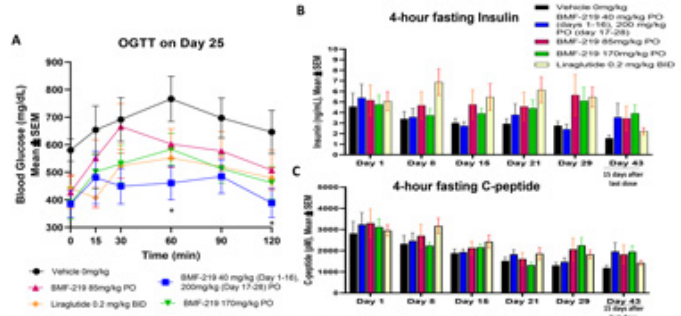


Figure 3. BMF-219 exerts strong glycemic control over 28-days of treatment in ZDF rats. OGTT was conducted on day 25 on rats treated with BMF-219 at indicated doses, liraglutide or vehicle control by measurement of blood glucose at 15 and 30 minute intervals up to 2 hours (A). Fasting insulin (B) and C-peptide (C) levels were measured weekly over 28 days in rats treated with BMF-219 at indicated doses, liraglutide, or vehicle control. Insulin and C-peptide levels were also measured on day 43 (15 days after the last dose was administered).

BMF-219 maintains a significant reduction of HbA1c two weeks after the last dose

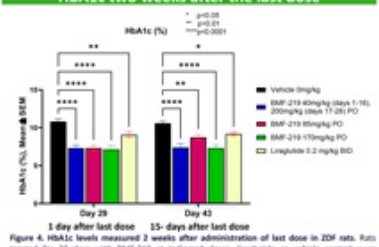


Figure 4. HbA1c levels measured 2 weeks after administration of last dose in ZDF rats. Rats treated for 28 days with BMF-219 at indicated doses, liraglutide or vehicle control were monitored for HbA1c levels on day 1 and day 15 post-dosing. Drug treated groups are compared to vehicle control to calculate statistical significance by two-way ANOVA.

Conclusions

- BMF-219 was tested in rats at clinically relevant exposures.
- All animals tolerated BMF-219 well throughout the study, displaying high activity.
- BMF-219 mid and high-dose arms showed reduction in fasting blood glucose levels similar to liraglutide. On day 29 (one day after treatment stop), BMF-219 high dose group showed sustained and significant reduction in fasting blood glucose.
- BMF-219 treatment reduced HbA1c levels by Day 21 of treatment. Absolute amounts were lower than vehicle group by 3.5% (33% reduced from vehicle) and lower than liraglutide group by 1.8% (20% reduced from vehicle) on day 29, and remained reduced throughout the study, including post-treatment.
- All BMF-219 dose groups showed improved glycemic control by oral glucose tolerance test (OGTT) on day 25, in comparison to vehicle-treated group, with the high dose-treated group showing improved response vs liraglutide.
- Fasting insulin and c-peptide levels were elevated in BMF-219 treated animals up to the last day of dosing, with the effects lasting well into two weeks post last dose.
- BMF-219 induced significant reductions in HbA1c at all doses tested, with the effects lasting 15 days after the last dose.
- Of note, animals in this study, including vehicle group, displayed progressive increase in body weight and fasting blood glucose levels over time, likely from very high food intake. This compromises meaningful data interpretation beyond day 43.
- Collectively, these data demonstrate the novel long-acting potential of BMF-219 as an oral treatment for T2DM, in maintaining glycemic control after short-term dosing.

References

Ma, J. et al. Menin-regulated Pbx1 controls high fat diet-induced compensatory beta cell proliferation. EMBO Mol Med 2021; 13(5):e13526.

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