

**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): December 12, 2022**

**Biomea Fusion, Inc.**

(Exact name of Registrant as Specified in Its Charter)

**Delaware**  
(State or Other Jurisdiction  
of Incorporation)

**001-40335**  
(Commission  
File Number)

**82-2520134**  
(IRS Employer  
Identification No.)

**900 Middlefield Road, 4th Floor**  
**Redwood City, CA**  
(Address of Principal Executive Offices)

**94063**  
(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 December 12, 2022, Biomea Fusion, Inc. (the “Company”) issued a press release titled, “Biomea Fusion Presents at the 2022 ASH Annual Meeting Preclinical Data on BMF-500 Supporting its Potential as the Most Potent and Selective FLT3 Inhibitor to Date.” The information described in the press release was also presented in a presentation at the 64<sup>th</sup> American Society of Hematology (ASH) Annual Meeting, which took place December 10-13, 2022 in New Orleans, Louisiana.

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 Company’s cash runway, the clinical and therapeutic potential of the Company’s product candidates and development programs, including BMF-219 and BMF-500, the potential of BMF-500 as a treatment for various types of cancer, the Company’s research, development and regulatory plans, including the Company’s plans to submit an IND for BMF-500 and to initiate clinical trials of BMF-500 as a single agent and in novel combinations, and the timing of such events, may be deemed to be forward-looking statements. The Company intends these forward-looking statements to be covered by the safe harbor provisions for forward-looking statements contained in Section 27A of the Securities Act and Section 21E of the Exchange Act and is making this statement for purposes of complying with those safe harbor provisions.

Any forward-looking statements made or incorporated by reference in this Current Report on Form 8-K are based on the Company’s current expectations, estimates and projections only as of the date of this Current Report on Form 8-K and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward-looking statements, including the risk that the Company may encounter delays in the initiation, conduct and completion of preclinical studies, including IND-enabling studies, the submission and clearance of IND applications, and the Company’s 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**

<u>Exhibit Number</u>	<u>Description</u>
99.1	<a href="#">Press release titled, “Biomea Fusion Presents at the 2022 ASH Annual Meeting Preclinical Data on BMF-500 Supporting its Potential as the Most Potent and Selective FLT3 Inhibitor to Date.”</a>
99.2	<a href="#">Presentation titled, “BMF-500: An Orally Bioavailable Covalent Inhibitor of FLT3 with High Selectivity and Potent Antileukemic Activity in FLT3-Mutated AML.”</a>
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: December 13, 2022

By: \_\_\_\_\_  
/s/ Thomas Butler  
**Thomas Butler**  
**Principal Executive Officer**

**Biomea Fusion Presents at the 2022 ASH Annual Meeting Preclinical Data on BMF-500 Supporting its Potential as the Most Potent and Selective FLT3 Inhibitor to Date**

- BMF-500, an investigational third generation covalent FLT3 inhibitor, demonstrated preclinically:
  - Picomolar affinity to activating FLT3 mutations including FLT3-ITD and various tyrosine kinase domain (TKD) mutations
  - Multi-fold higher potency and increased cytotoxicity than commercially available non-covalent FLT3 inhibitor gilteritinib
  - Complete tumor regression in mouse models of FLT3-ITD acute myeloid leukemia (AML) and maintenance of effect without continued exposure
- Biomea Fusion remains on track to file an IND for BMF-500 in the first half of 2023

REDWOOD CITY, Calif., December 12, 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 preclinical data today for BMF-500, an investigational covalent FLT3 inhibitor, at the 64<sup>th</sup> American Society of Hematology (ASH) Annual Meeting. The preclinical poster can be viewed at Biomea's website at <https://biomeafusion.com/publications>.

BMF-500, a novel, orally bioavailable, highly potent and selective covalent small molecule inhibitor of FLT3, was discovered and developed in-house at Biomea using the company's proprietary FUSION™ System and designed to have a therapeutic profile to allow for combinations with standard of care and/or novel targeted agents like BMF-219, Biomea's investigational covalent menin inhibitor. The company is on track to submit an investigational new drug (IND) application for BMF-500 in the first half of 2023 and, subject to successful IND clearance, plans to initiate clinical trials evaluating BMF-500 as a single agent and in novel combinations shortly thereafter.

“The majority of late-stage and approved FLT3 inhibitors fall short of providing sufficient and sustained inhibition of FLT3 signaling required for maximal benefit. We believe the exquisite potency, selectivity and durability of BMF-500, all of which are possible through the design of this novel covalent molecule with our proprietary FUSION system, have the potential to translate to deep and durable remissions in patients with FLT3-mutant AML, representing nearly a third of all AML patients,” said Steve Morris, M.D., Biomea's Chief Medical Officer. “We look forward to advancing BMF-500 toward the clinic in 2023, which will mark our second clinical-stage novel covalent small molecule program for cancer and further solidify our position as a leader in next-generation covalent medicines.”

“The impressive profile of BMF-500 speaks to the bench strength of our Discovery Team and to our ability to design and deliver novel therapeutics. We believe that targeting FLT3 with a covalent inhibitor, as we did with menin, represents another significant opportunity to improve the outcomes that reversible inhibition provide. BMF-500 has the potential to be an effective option as a monotherapy as well as in combination,” said Thomas Butler, Biomea’s Chief Executive Officer and Chairman of the Board.

The data presented at the ASH conference today showed that in FLT3-driven AML cell lines, three-hour exposure to BMF-500 produced complete phospho-FLT3 inhibition, which was maintained following washout of the compound. The commercially available reversible (i.e., non-covalent) FLT3 inhibitor gilteritinib required 16 times higher concentration than BMF-500 and continuous exposure for 96 hours to produce the same effect. The covalent small molecule inhibitor BMF-500 exhibited potent inhibition of FLT3 receptor kinase and marked cell killing in FLT3-ITD AML cell lines, as demonstrated by IC<sub>90s</sub> at physiologically relevant doses. In addition, the kinase profile of BMF-500 revealed high target selectivity and selective cytotoxicity profile against a panel of non-target cancer cell lines suggesting the potential for minimal off-target liabilities. As part of the poster presentation, Biomea also presented the results of two preclinical animal xenograft models in which BMF-500 demonstrated antitumor activity with sustained tumor regression and improved survival while being well tolerated, with body weight maintenance across treatment groups.

#### **About BMF-500**

BMF-500 is a novel orally bioavailable, highly potent and selective covalent small molecule inhibitor of FLT3 in preclinical development, which has been shown to exhibit favorable durability, selectivity and tolerability in preclinical studies in comparison to commercially available FLT3 inhibitor gilteritinib. BMF-500 was discovered and developed in-house by Biomea using its proprietary FUSION™ System and designed to have a therapeutic profile to allow for combinations with standard of care and novel targeted agents like BMF-219, Biomea’s investigational covalent menin inhibitor. The kinase profile of BMF-500 showed high target selectivity, suggesting the potential for minimal off-target liabilities.

### **About FLT3 (fms-like tyrosine kinase 3)**

FLT3 is a receptor tyrosine kinase (RTK) that plays a central role in the survival, proliferation, and differentiation of immature blood cells. Notably, FLT3 gene mutations are common in patients with AML and are associated with a poor prognosis. Nearly 30% of AML patients have a FLT3 mutation, representing more than 6,000 incident patients in the United States each year. While FLT3-specific and pan-tyrosine kinase inhibitors are approved by the FDA across various lines of therapy in AML, these agents have produced relatively low rates of durable responses and overall survival remains an unmet need.

### **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 is a leader in advancing next-generation covalent small molecule medicines designed to maximize clinical benefit to treat 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 and BMF-500, the potential of BMF-500 as a treatment for various types of cancer, our research, development and regulatory plans, including our plans to submit an IND for BMF-500 and to initiate clinical trials of BMF-500 as a single agent and in novel combinations, 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.

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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 in the initiation, conduct and completion of preclinical studies, including IND-enabling studies, the submission and clearance of IND applications, and our planned clinical trials and other research and development 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.

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# BMF-500: An Orally Bioavailable Covalent Inhibitor of FLT3 with High Selectivity and Potent Antileukemic Activity in FLT3-Mutated AML

We Aim to Cure

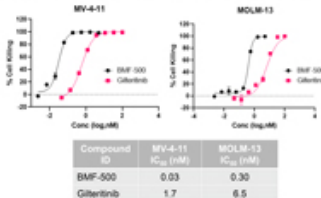
Brian Law, BS<sup>1</sup>, Tripta Rughwan, MS<sup>1</sup>, Tenley Archer PhD<sup>1</sup>, Lekha Kumar, MS<sup>1</sup>, Daniel Lu, MS<sup>1</sup>, Priyanka Somnath, PhD<sup>1</sup>, Yan Ma, PhD<sup>1</sup>, Xiaodong Wang, MS<sup>1</sup>, David Sperandio, PhD<sup>1</sup>, Steve Morris, MD<sup>1</sup>, Thorsten Kirschberg, PhD<sup>1</sup>, Mini BalaKrishnan, PhD<sup>1</sup> and Thomas Butler, MSc MBA<sup>1</sup>  
<sup>1</sup>Biomea Fusion, Inc., Redwood City, CA

## Introduction

- Fms-like tyrosine kinase 3 (FLT3), a member of the receptor tyrosine kinase family, is overexpressed on AML blasts and widely expressed in hematopoietic progenitor cells (Carow et al., 1996).
- Activating mutations of FLT3 are the most frequent genetic alterations in AML, leading to FLT3 ligand-independent signaling and cellular proliferation, which account for approximately 30% of newly diagnosed adult AML patients and are associated with poor prognosis (Papayannoulou et al., 2016).
- Although, several FLT3 inhibitors have entered clinical trials and reached commercialization; however, prolonged FLT3 target duration coverage, adverse events and dose-limiting toxicities often restrict the therapeutic window and limit their long term use and efficacy (Kennedy et al., 2020).
- BMF-500 is a novel orally bioavailable, highly potent and selective covalent small molecule inhibitor of FLT3 with best-in-class potential, given its efficacy, durability and selectivity in comparison to an existing FLT3 inhibitor.

## Potent Target Inhibition

### A. Potent Cell-Based Activity in AML Cell Lines with FLT3 Mutations



### B. NanoBRET Target Engagement Assay, IC<sub>50</sub> (nM)

Compound ID	FLT3 WT	FLT3 (D835H)	FLT3 (D835V)	FLT3 (D835Y)
BMF-500	0.31	0.18	0.22	0.25
Gilteritinib	23.4	1.45	1.1	1.4

### C. FLT3 Inhibitor Resistance Mutations Coverage, IC<sub>50</sub> (nM)

Compound ID	FLT3-ITD	FLT3-ITD-D835Y	FLT3-ITD-F691L
BMF-500	2 nM	5 nM	7 nM
Gilteritinib	7 nM	19 nM	98 nM

Figure 1. A) Potent Cell-Based Activity in AML Cell Lines with FLT3 Mutations. Potency of BMF-500 and gilteritinib against FLT3 (WT) and FLT3-ITD enzyme-based biochemical assays (left) and cell-based 4-day CTO readout using MV-4-11 and MOLM-13 target cell lines. Representative dose response curves are shown for MV-4-11 and MOLM-13 cell killing. B) NanoBRET Target Engagement Assay, IC<sub>50</sub> (nM). C) FLT3 Inhibitor Resistance Mutations Coverage, IC<sub>50</sub> (nM). Transformed cells expressing wild-type (WT) FLT3 (D835H/Y) variants, FLT3-ITD, FLT3-ITD-D835Y, and FLT3-ITD-F691L mutations were profiled via NanoBRET target engagement and cell-based assays, respectively.

## Potent and Durable Target Inhibition Leading to Effective Cell Killing

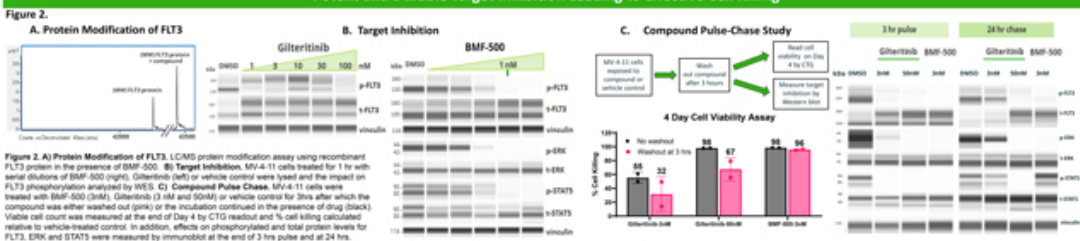


Figure 2. A) Protein Modification of FLT3. LCMSE protein modification assay using recombinant FLT3 protein in the presence of BMF-500. B) Target Inhibition. MV-4-11 cells treated for 1 hr with serial dilutions of BMF-500 (right). Gilteritinib (left) or vehicle control were lysed and the impact on FLT3 phosphorylation analyzed by Western blot. C) Compound Pulse Chase. MV-4-11 cells were treated with BMF-500 (3 nM), Gilteritinib (3 nM and 50 nM) or vehicle control for 3 hrs after which the compound was either washed out (pink) or the incubation continued in the presence of drug (black). Viability cell count was measured at the end of Day 4 by CTO readout and % cell killing calculated relative to vehicle-treated control. In addition, effects on phosphorylated and total protein levels for FLT3, ERK and STAT5 were measured by immunoblot at the end of 3 hr pulse and at 24 hrs.

## In Vivo Efficacy in Mouse Xenograft Models

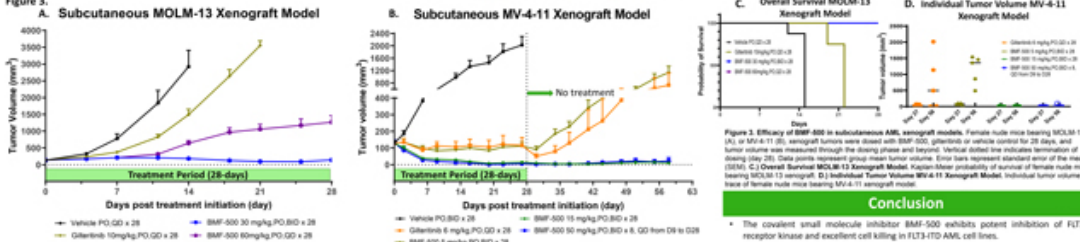


Figure 3. Efficacy of BMF-500 in subcutaneous AML xenograft models. Female nude mice bearing MOLM-13 (A) or MV-4-11 (B) xenograft tumors were dosed with BMF-500, gilteritinib or vehicle control for 28 days, and tumor volume was measured through the dosing phase and beyond. Vertical dotted line indicates termination of dosing (day 28). Data points represent group mean tumor volume. Error bars represent standard error of the mean (SEM). C) Overall Survival MOLM-13 Xenograft Model. Kaplan-Meier probability of survival of female nude mice bearing MOLM-13 xenograft. D) Individual Tumor Volume MV-4-11 Xenograft Model. Individual tumor volume trace of female nude mice bearing MV-4-11 xenograft model.

## Conclusion

- The covalent small molecule inhibitor BMF-500 exhibits potent inhibition of FLT3 receptor kinase and excellent cell killing in FLT3-ITD AML cell lines.
- Transformed cells expressing key FLT3 inhibitor resistance variants, including D835Y/VH activation loop and F691L gatekeeper point mutations show high sensitivity to BMF-500 highlighting a clear differentiation to gilteritinib.
- Covalent modification of FLT3 by BMF-500 leads to constitutive on-target inhibition of phospho-FLT3 and its downstream signaling in a dose dependent manner in the target cell.
- In MV-4-11 cells, 3-hour exposure followed by washout of BMF-500 was sufficient to produce longer duration of response and greater cell killing that outperforms continuous exposures of gilteritinib at 3 nM.
- BMF-500 demonstrates antitumor activity with sustained tumor regression, improves survival and is well tolerated with body weight maintenance across treatment groups in two preclinical mouse xenograft models.
- The kinase profile of BMF-500 revealed high target selectivity and selective cytotoxicity profile against a panel of non-target cancer cell lines suggesting minimal off target liabilities.

## References

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