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): July 19, 2021

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.)**

726 Main Street **Redwood City, CA**	**94063**
(Address of Principal Executive Offices)	**(Zip Code)**

Registrant's telephone number, including area code: (650) 980-9099

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 per share	**BMEA**	**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 7.01. Regulation FD Disclosure.

Biomea Fusion, Inc. (the "Company") from time to time presents and/or distributes to the investment community at various industry and other conferences slide presentations to provide updates and summaries of its business. On July 19, 2021, the Company posted an updated corporate slide presentation in the "Investors & Media" portion of its website at *www.biomeafusion.com*. A copy of its current corporate slide presentation is attached to this Current Report on Form 8-K as Exhibit 99.1. The Company undertakes no obligation to update, supplement or amend the materials attached hereto as Exhibit 99.1.

The information contained in Item 7.01 of this Form 8-K (including Exhibit 99.1 attached hereto) shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly provided by specific reference in such a filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

The following Exhibit 99.1 relating to Item 7.01 shall be deemed to be furnished, and not filed:

Exhibit No.	Description
99.1	Corporate Slide Presentation of Biomea Fusion, Inc. dated July 2021
104	Cover Page Interactive Data File (embedded within the inline XBRL document)

SIGNATURES

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

BIOMEA FUSION, INC.

Date: July 19, 2021

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

Exhibit 99.1

Disclaimer and Forward-Looking Statement

Certain statements in this presentation and the accompanying oral commentary are forward-looking statements. These statements relate to future events or the future 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 clinical trials, the actions or potential action of the FDA, the status and timing of ongoing research, 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, 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. These and other important factors may cause actual results, performance or achievements to differ materially from those expressed or implied by these forward-looking statements. 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.

OUR MISSION

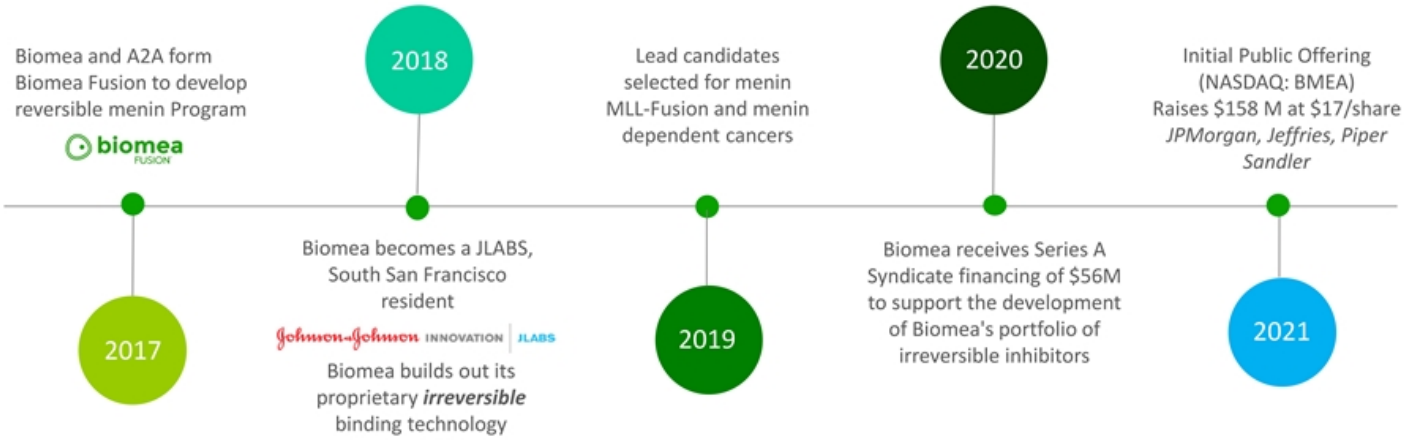
Our Mission is to revolutionize drug development in order to create more effective therapies for patients in need.

Biomea Fusion is a preclinical-stage biopharmaceutical company focused on the discovery and development of **irreversible small-molecule drugs** to treat patients with genetically defined cancers. Our discovery team is engaged in all phases of development, including target selection, small molecule design, and preclinical and clinical studies to develop innovative medicines.

biomea FUSION

Biomea Fusion History

A Discovery Platform in Development since 2017



biomea FUSION

Biomea's Team

Diverse team with significant drug development experience

Thomas Butler
Chairman & CEO

15 years in Life Science
Pharmacyclics
Gilead Sciences
UCLA – MBA Finance
UCSB, MS – Chemistry

Ramses Erdtmann
President & COO

13 years in Life Science
Pharmacyclics
Oxygen Investments
Commerzbank
University of Münster, Master's in Banking & Corp Finance

Franco Valle
Chief Financial Officer

15 years in Life Science
Eidos Therapeutics
Iovance Biotherapeutics
Pharmacyclics
CallidusCloud
PricewaterhouseCoopers
San Jose State University, BS Corporate Finance

Naomi Cretcher
Chief of People

15 years in Life Science
Pharmacyclics
Genentech
UC Irvine, BA Comm
SF State University, Comm

Heow Tan
Chief Technical & Quality Officer

22 years in Life Science
Pharmacyclics
Collegium Pharmaceutical
Praecis Pharmaceuticals
Ohio State University
Santa Clara University
Leavey School of Business, MBA – Finance & Management

Alex Cacovean MD
Exec. Medical Director

15 years in Life Science
Iovance Inc.
Pharmacyclics
PPD Inc.
Henry Ford Hospital
University of Medicine and Pharmacy "Iuliu Hatieganu" Cluj-Napoca, Romania, M.D.

Thorsten Kirschberg
EVP of Chemistry

25 years in Life Science
Terns Pharmaceuticals
Gilead Sciences
Cell Gate
Golden Gate
University, MBA
University of Münster, Ph.D., Chemistry

Taisei Kinoshita
VP of Biology

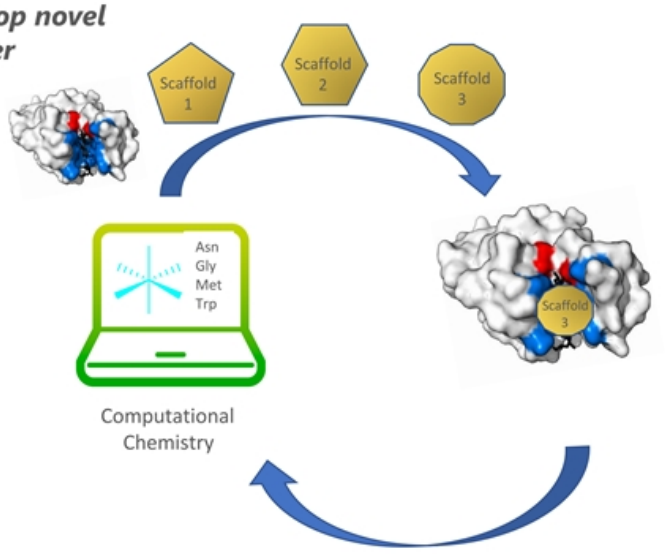
20 years in Life Science
Pharmacyclics
Rigel Pharmaceuticals
University of Tokyo, Ph.D. Biology

Biomea FUSION™ System Discovery Platform

We leverage our FUSION™ System to discover and develop novel irreversible inhibitors against targets essential for cancer

Our FUSION™ System Discovery Platform encompasses the following:

- ***Target selection***: Expertise in structural biology and irreversible binding chemistry.
- ***Scaffold creation:*** Computational approach to exploit unique structural elements of target proteins and create novel scaffolds.
- ***Molecule optimization:*** Proprietary suite of computational technologies, assays, analytical approaches, chemistry to maximize selectivity, potency, safety and convenience of our oral irreversible small molecule product candidates.



Irreversible Drugs offer several potentially significant Advantages

High Selectivity

- Irreversible drugs leverage both non-covalent and covalent interactions to drive selectivity.
- Offers greater potential selectivity versus reversible compounds, which rely on non-covalent bonding alone.
- High selectivity provides potential to reduce non-specific, off-target interactions that often lead to safety and tolerability challenges.

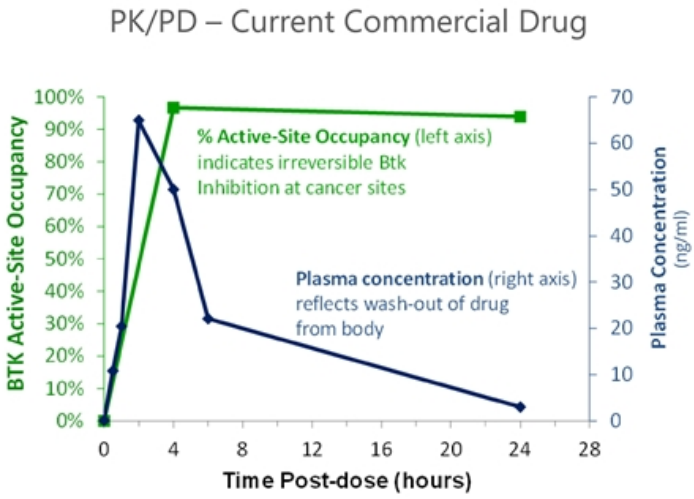
Deep Target-Inactivation

- Irreversible inhibitors can cause permanent inactivation of bound protein.
- Irreversible binding may result in the target elimination through normal cellular degradation processes.
- Target inactivation can trigger rapid apoptosis or differentiation into a normal, mature cell.

Greater Therapeutic Window

- Irreversible drugs are designed to maintain their effect in the absence of sustained drug exposure, unlike conventional reversible drugs, which typically need to be present to provide benefit.
- Uncoupling of drug effects from drug exposure can potentially enable lower drug dosing and less frequent dosing regimens versus reversible approaches.

Irreversible Drugs are designed to uncouple Drug-Effects from Drug-Exposure



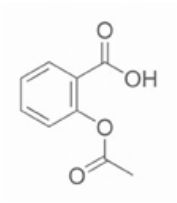
- The chart shows Ibrutinib, an approved, irreversible BTK inhibitor.
- Irreversible drugs can be designed to achieve nearly complete occupancy in short time, and occupancy is intended to be sustainable over 24 hours.
- Irreversible drugs are also designed to be cleared rapidly to minimize off target toxicity.
- Irreversible binding potentially offers:
  - Optimal Effect (Pharmacodynamics (PD)) / Exposure (Pharmacokinetics (PK))
  - Maximum Target Engagement
  - Better Selectivity (Lower Molecular Weight)
  - Better Drug-Like Properties (Lower Molecular Weight)

biomea FUSION

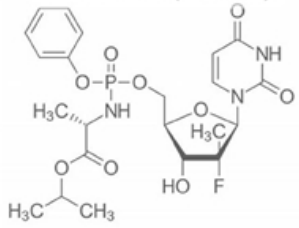
Biomea Irreversible Drugs have a long History in Medicine

Aspirin was the first irreversible drug, discovered in 1899, and is to date the most used medicine in the world

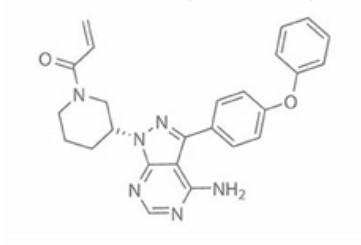
Aspirin

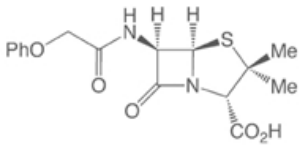


Sofosbuvir (Sovaldi)

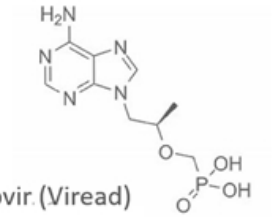


Ibrutinib (Imbruvica)

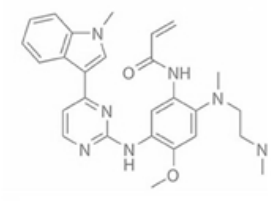




Penicillin



Tenofovir (Viread)



Osimertinib (Tagrisso)

biomea FUSION

High Barriers to Entry to develop Irreversible Drugs

Complexity

The discovery and development of irreversible drugs has been limited by:

- Need for specialized understanding of proteome structural knowledge and medicinal chemistry capabilities, including the ability to construct complex novel chemical scaffolds.
- Limited knowledge and availability of targets as not all disease causing proteins have the properties necessary for the application of irreversible binding.

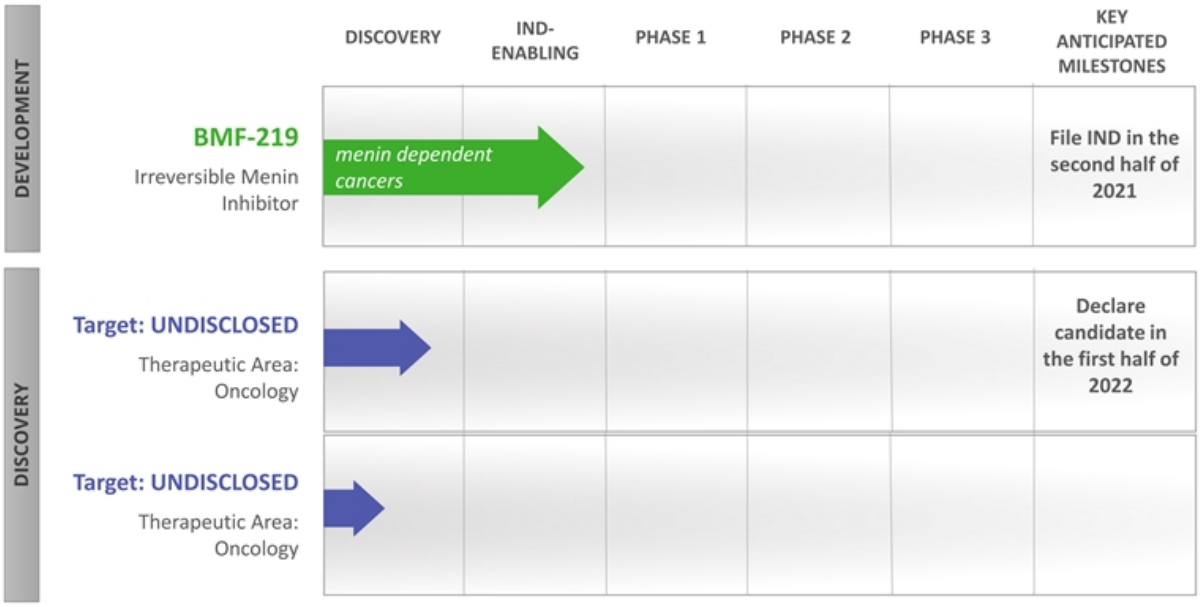
Safety and Toxicity

While the irreversible binding modality can provide a high degree of selectivity, potential risks have presented barriers:

- Irreversible molecules with promiscuous binding profiles can pose risk of significant off-target interactions and safety concerns.
- Drug developers, without the experience and specific capabilities required to develop irreversible binders, have historically not pursued irreversible drugs.

Biomea's Pipeline of Irreversible Proprietary Assets

We are building a platform of irreversible inhibitors in multiple tumor types

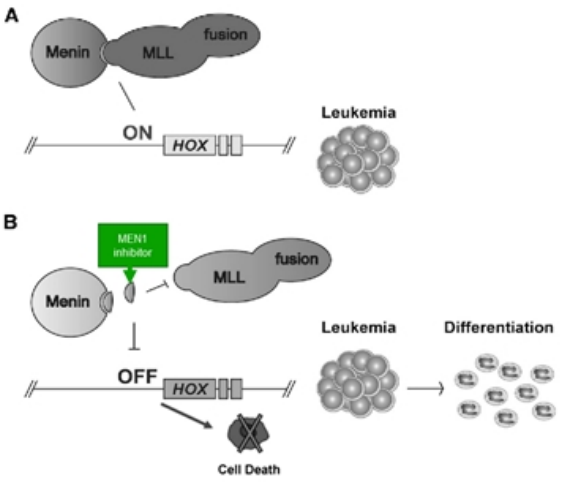


biomea FUSION

Menin – a Protein important to transcriptional Regulation

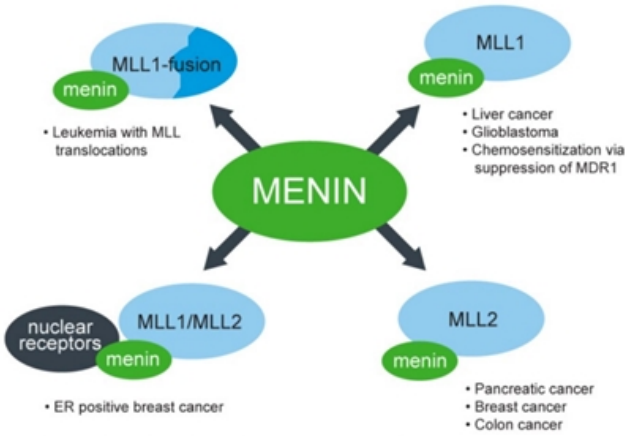
Menin impacts major processes such as cell cycle control, apoptosis, and DNA damage repair

Liquid Tumor Role



Modified after Uckelmann (Scott Armstrong Lab) , ASH 2018, Abstract # 546

Solid Tumor Role



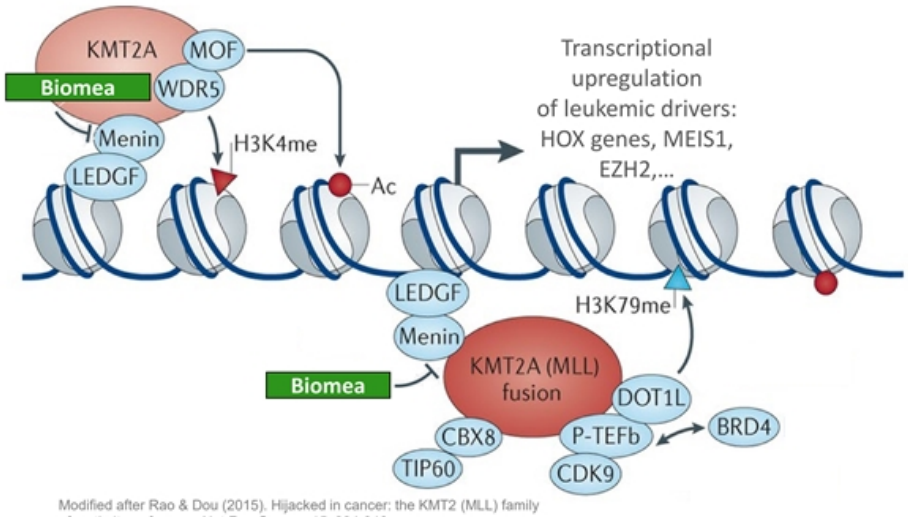
Source: Cierpicki & Grambacka, Future Med. Chem. (2014)

biomea FUSION

Menin-MLL Interaction

Schematic of key proteins and their interactions in the regulation of gene transcription

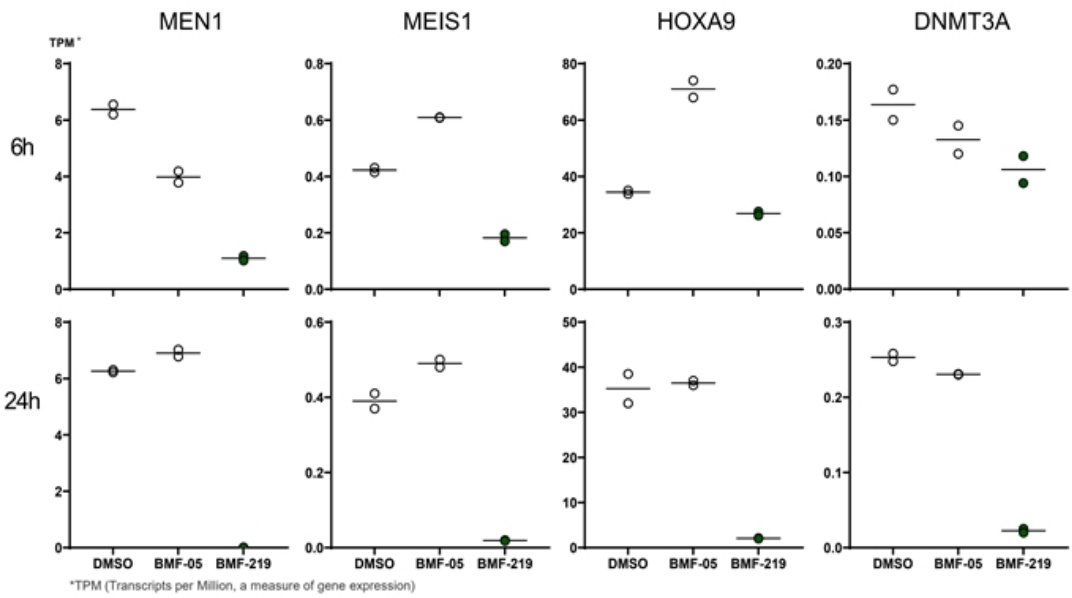
- Inhibition of the menin-MLL interaction leads to reduction in *MEN1* transcription, resulting in down regulation of *MEIS1*, *HOXA9* and *DMNT3A*, and differentiation of leukemic cells into myeloid cells.
- BMF-219, is intended to irreversibly inhibit the interaction between menin and wild type MLL and MLL fusions.



Modified after Rao & Dou (2015). Hijacked in cancer: the KMT2 (MLL) family of methyltransferases. Nat.Rev.Cancer. 15: 334-346

biomea FUSION

BMF-219 has demonstrated rapid Reduction in Menin dependent Gene Expression = Target Engagement

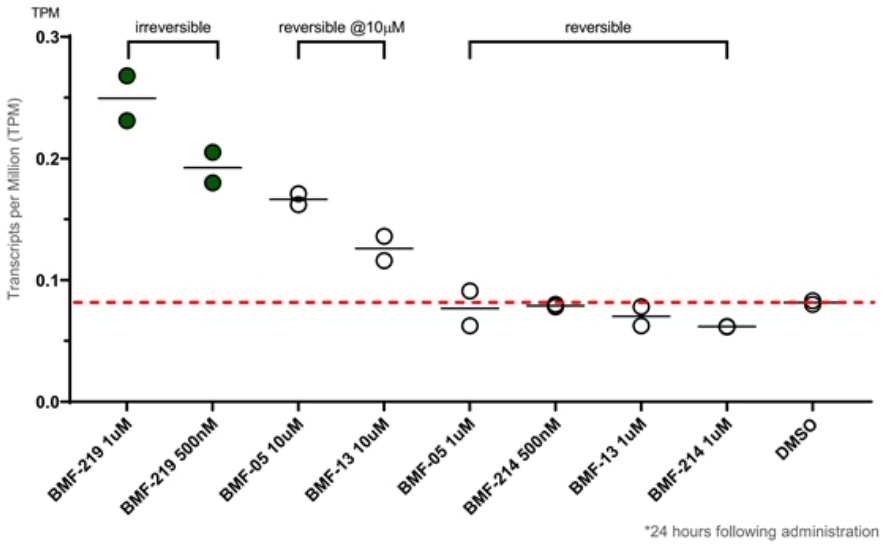


*TPM (Transcripts per Million, a measure of gene expression)

- Molecular responses following treatment with BMF-219 in MOLM-13 cells (an acute myeloid leukemia cell line with a KMT2A-MLLT3 fusion).
- A reversible inhibitor (BMF-05) showed minimal impact on signature genes at these time points as measured in Transcripts per Million (TPM), consistent with published findings.
- Our irreversible inhibitor BMF-219 caused rapid and notable effect, showing up to 80% reduction in readout genes by 6 hours and approximately 95% reduction at 24 hours.

biomea FUSION

BMF-219 has demonstrated Dose Dependent Cell Differentiation

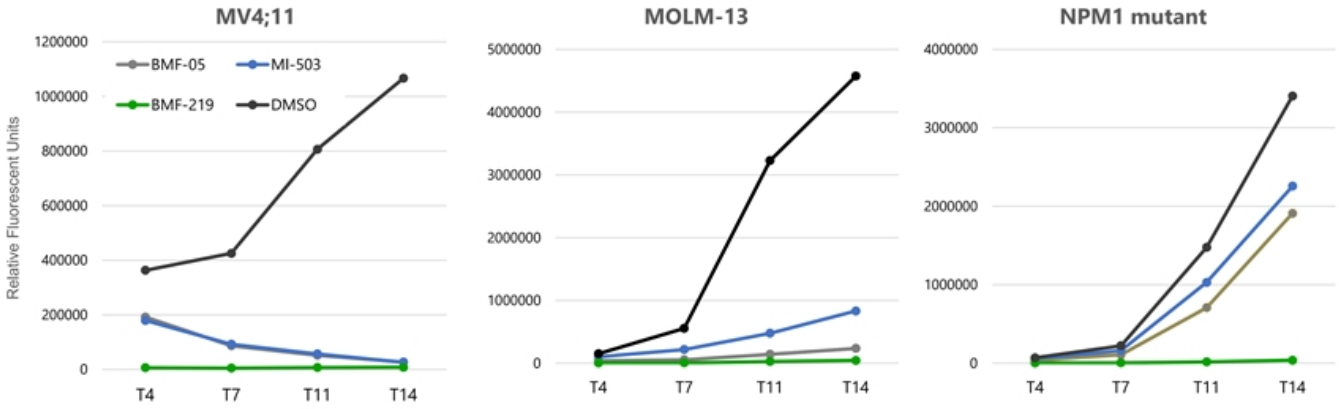


*24 hours following administration

- Dose dependent elevation of myeloid differentiation marker (ITGAM) at 24 hours after administration of BMF-219 demonstrated target engagement in MOLM-13 cells.
- Comparable exposures of reversible menin inhibitors (BMF-05, BMF-13, BMF214) reflected no change from vehicle controls.
- Reversible inhibitors were able to upregulate ITGAM at 10-fold increase in exposure. This supports:
  - Hypothesis for the role of menin in cancer cell proliferation and
  - Highlights potential need for high clinical exposures for reversible inhibitors in order to achieve sufficient menin suppression to affect the disease.

biomea FUSION

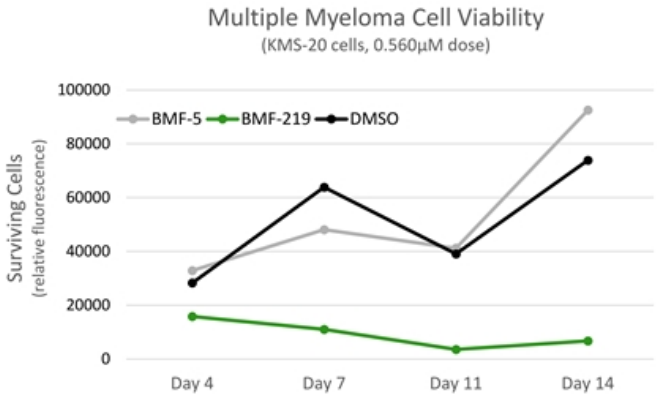
BMF-219 has demonstrated superior, rapid and durable Responses in Leukemia Cell Lines



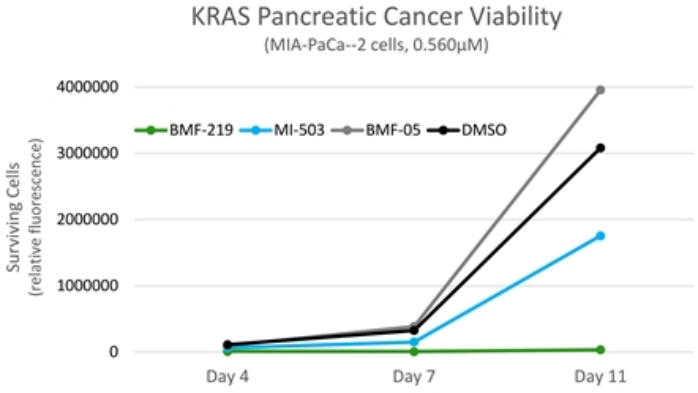
- BMF-219 demonstrated rapid shut down of metabolic activity, sustained over the 14-day study duration.
- BMF-219 responses were superior to tested reversible menin inhibitors (BMF-05, MI-503) with respect to both onset and durability of metabolic suppression.

biomea FUSION

BMF-219 Impaired Cell Survival in Multiple Myeloma and Pancreatic Cancer Models



- Impairment of survival in multiple myeloma model (KMS-20 cell line, 0.56µM doses) by irreversible menin inhibitor BMF-219 versus a reversible inhibitor (BMF-05).



- Impairment of survival in G12C KRAS mutation driven pancreatic cancer line (MIA-PaCa-2, 0.56µM doses) shows the effects of irreversible menin inhibitor BMF-219 versus a reversible inhibitor (BMF-05) at far lower dose levels.

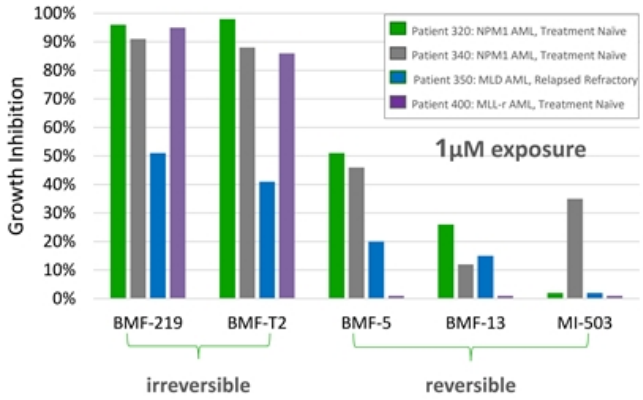
biomea FUSION

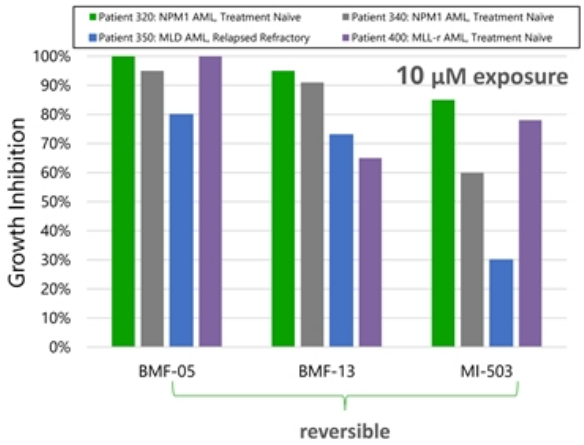
BMF-219 Inhibited Cell Survival in Multiple Cancer Cell Lines

- Responses to BMF-219 were screened across a range of cancer cell lines.
- Potent growth inhibition was observed across a range of cancer models including liquid and solid tumors.

	Cell Line/Tumor Type	IC50 (µM)
Fusion	MOLM-13/AML	0.05
	MV4;11/ALL-AML	0.07
NPM1 Mutation	OCI-AML3/AML	0.14
KRAS	MIA-PaCa-2/Pancreatic	0.23
	NCI-H23/Lung	0.26
Menin Dependent	KMS-20/Plasma Cell Myeloma	0.26
	VCaP/Prostate Adenocarcinoma (met)	0.29
	OE-33/Barrett Esophageal Adenocarcinoma	0.30
	KG-1/AML	0.33
	HC1187/Ductal Breast Carcinoma (TNBC)	0.46
KRAS	Panc 10.05/Pancreatic	0.49
	NCIH23/NSCLC	0.49
Menin Dependent	BT-474/invasive ductal carcinoma NOS	0.52
KRAS	SK-LU-1/Lung	0.59

BMF-219 Inhibited Patient derived AML cells at very low Exposures vs Reversible Inhibitors





- Irreversible inhibition leads to dramatic growth inhibition at 1µM exposure while tested reversible inhibitors showed limited effect.
- At six days, reversible menin inhibitors show similar inhibition of growth at drug exposures 10-fold greater than their respective IC90 values (10µM).
- Findings support relevance of the mechanism in disease and our hypothesis that an irreversible inhibitor could potentially provide greater therapeutic benefit at lower exposure-levels versus reversible inhibitors.

biomea FUSION

Selectivity Profiling of BMF-219 in multiple Safety Models

Kinase screening

- In-house analysis of menin revealed limited structural similarity between targeted binding pocket and tyrosine kinases with known involvement in hematological cancers.
- 169 kinases screened; only two wild type kinases showed greater than 50% inhibition upon treatment with BMF-219.

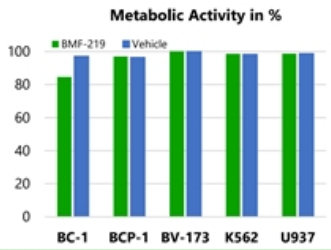
Glutathione reactivity

Drug	Mean half-life (min)
Omeprazole	123.3
Neratinib	197.7
Ibrutinib	>360
BMF-213	322.3
BMF-214	>360
BMF-219	>360

- Drugs with limited non-specific interactions have long half-lives as the drug does not get consumed in a reaction with wildly-used glutathione (GSH).
- BMF-219 showed negligible interaction with the strong nucleophile GSH and showed less reactivity than the approved irreversible drugs omeprazole and neratinib.

Oncopanel screening

- Minimal impact of BMF-219 treatment on cell metabolism in leukemia and lymphoma cell lines that have wild type MLL, but no menin-linked mechanism for disease.
- Findings are consistent with external studies, showing that menin-MLL interaction is not generally cell-essential and only critical to survival in those cells that contain aberrant biology.

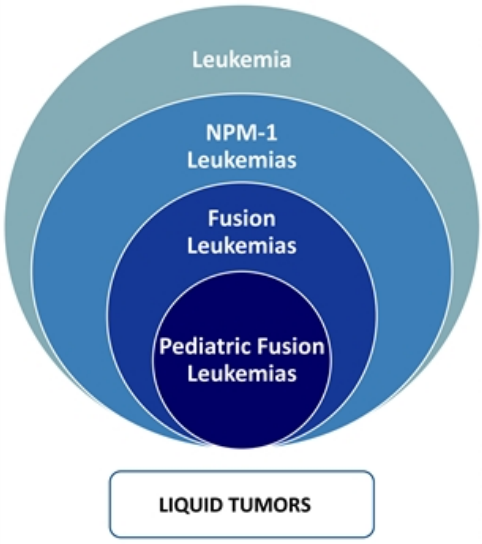


Safety screen

- BMF-219 was also profiled on the SafetyScreen44 panel (CEREP/Eurofins Discovery), an in vitro panel of 44 common selected targets to identify significant off-target interactions.
- Findings showed no meaningful impact (greater than 50% activation or inhibition) of BMF-219 across these key safety assays

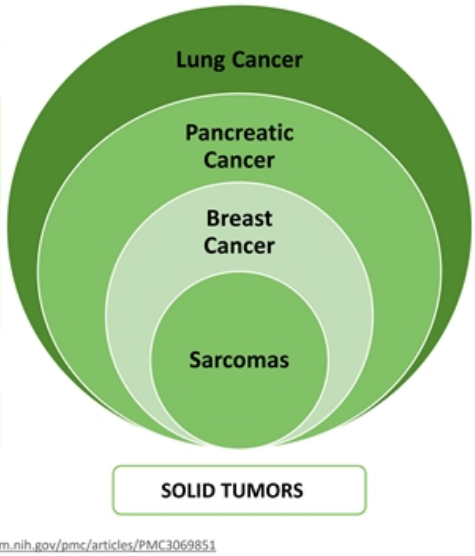
biomea FUSION

Estimated Target Population for Irreversible Menin Inhibitor BMF-219



Liquid Tumors	
Disease	Est. US Patient Population (Annual Incidence)
AML/ALL (MLL-R)	2,500
AML/ALL (NPM1)	7,000
DLBCL*	18,000
MM*	32,000

Solid Tumors	
Disease	Est. US Patient Population (Annual Incidence)
EWS	1,000
KRAS-Lung	34,000
KRAS-Pancreatic	26,000
KRAS-CRC	17,000



* Investigation ongoing to further define menin dependent subset

Patient Numbers see:

AML/ALL/DLBCL/MM/Lung/Pancreatic/Colorectal: https://seer.cancer.gov

EWS: pubmed.ncbi.nlm.nih.gov/18525458/

Mutation Status see:

AML/ALL MLL-R & NPM1 www.ncbi.nlm.nih.gov/pmc/articles/PMC5299633/ and www.ncbi.nlm.nih.gov/pmc/articles/PMC3069851

KRAS NSCLC G12C&D https://err.ersjournals.com/content/25/139/71#

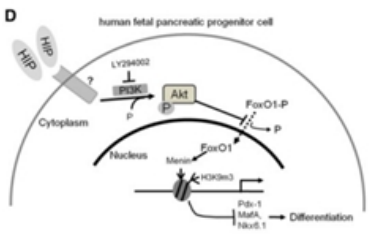
KRAS PDAC G12C&D www.cell.com/trends/biochemical-sciences/pdf/S0968-0004(13)00203-X.pdf

KRAS CRC G12C&D www.ncbi.nlm.nih.gov/pmc/articles/PMC6945179/#!po=15.9091

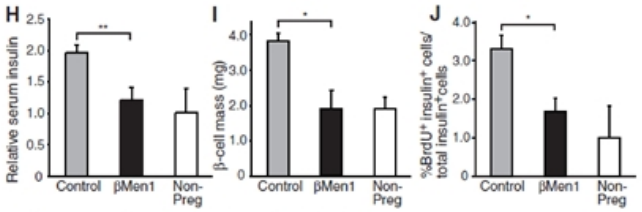
biomea FUSION

Menin Pathway Validation – Type II Diabetes

- Menin (MEN1) controls differentiation of pancreatic stem cells into adult, glucose-responsive Beta Cells
- Expression of MEN1 and MEN1 target genes (p27) is significantly decreased with increasing Concentrations of Glucose
- Glucose induces islet Beta-cell growth while inhibiting MENIN expression both at the Transcription and Translational Level
- MENIN expression also controls Beta Cell Growth and Insulin Levels in pregnant Mice and promotes Gestational Diabetes



Source: Jiang et al., Diabetes (2018)



[1]Department of Developmental Biology, Stanford University, Stanford, CA 94305, USA. [2]Department of Pathology, Stanford University, Stanford, CA 94305, USA. [3]Department of Medicine (Oncology Division), Stanford University, Stanford, CA 94305, USA.

Biomea Fusion Intellectual Property

We believe our Patent Portfolio with novel irreversible Scaffolds is strong and growing

We have a strong Intellectual Property Portfolio (IP) which is growing every month. Our IP consists of several pending US and ex-US patent applications and several trademarks related to our scientific assets.
Our patents cover composition of matter, pharmaceutical composition, and methods of treatment for our reversible and irreversible menin inhibitors.
If issued, we expect our patents to expire between December 2039 to December 2041
(excluding possible patent term extensions which can vary in time and are not guaranteed).

Corporate Milestones

Developing BMF-219 for multiple indications and building out a platform of irreversible inhibitors

2021 and beyond

- ✓ 15 April 2021, IPO. Sale of 9.823 M shares at $17 per share, raising after fees $153M with a total share count of 29.5M. Total Cash at the end of Q1 was $57.5M, together with the funds raised during the IPO, Biomea had a cash balance of about $210M.
- ✓ Completion of IND-enabling studies with irreversibly bound menin inhibitor BMF-219.
- File IND in 2nd Half of 2021.
- Pre-clinical data in DLBCL with BMF 219 in Q1 2022.
- Pathway validation of menin in Type II diabetes in Q1 2022.
- Initiation of first in human trial with BMF-219 expected to produce clinical data for a variety of tumor types in Phase I / II Basket-Study.
- Advancement of irreversible platform to generate one additional IND candidate in 1H of 2022.


Thank You
biomea
FUSION