biomea FUSION™

Corporate Presentation - December 2022

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We aim to cure

- Experienced Management Team
- Novel FUSION™ System
- BMF-219 - Clinical Stage Lead Asset
- BMF-500 and additional Programs

biomea FUSION™ We Aim to Cure™

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

Developing some of the most impactful medicines of our time

A long history of developing successful drugs together

Thomas Butler
Chairman & CEO

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

Veklury® remdesivir 100 MG FOR INJECTION

Co-inventor of
Remdesivir at Gilead

Ramses Erdtmann
President & COO

15+ years in Life Science
Pharmacyclics
Oxygen Investments
Commerzbank
University of Münster,
Master's in Banking & Corp 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 & Mgmt

Steve Morris MD
Chief Medical Officer

25+ years in Life Science
HealthChart LLC
Insight Genetics
St. Jude Children's Research Hospital
Board certified internist (Univ. of Texas SW HSC) and medical oncologist (Yale University School of Medicine)

Franco Valle
Chief Financial Officer

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

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

HARVONI® ledipasvir / sofosbuvir

Co-lead of Ledipasvir at Gilead

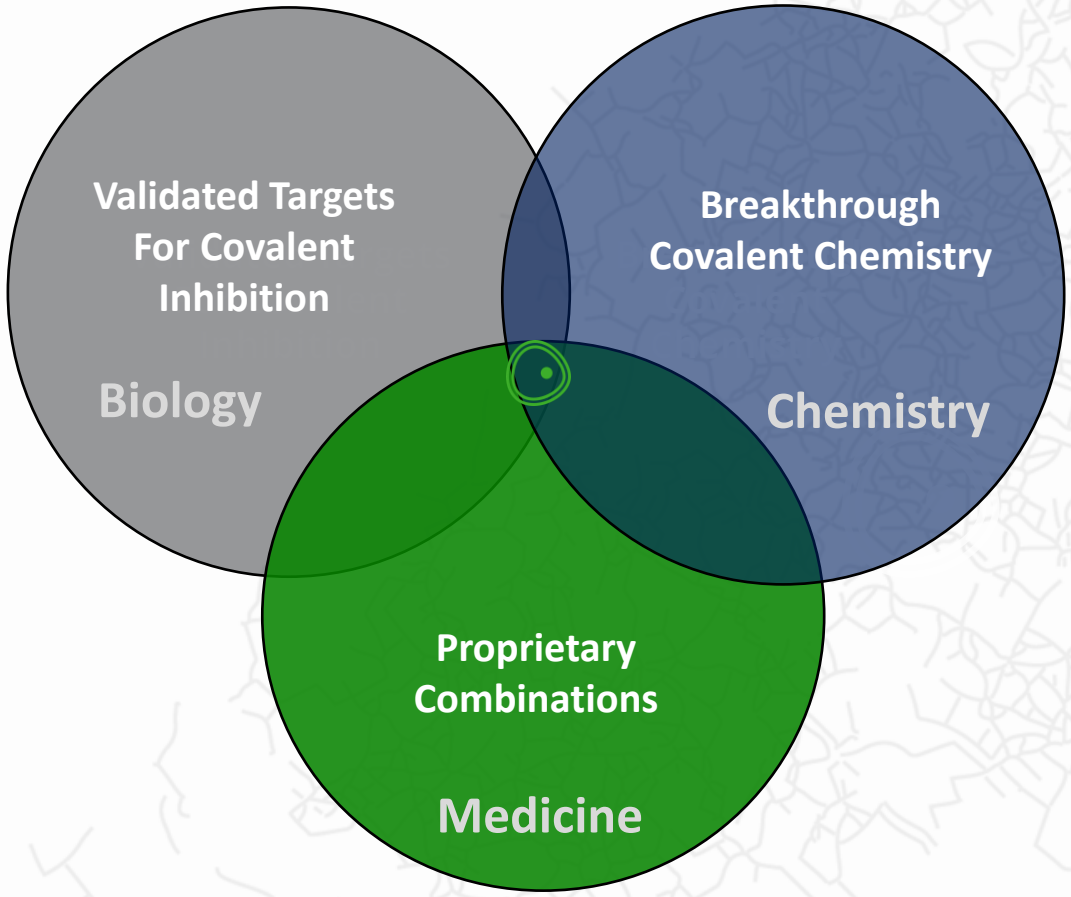
Jim Palmer
VP of Drug Discovery

30+ years in Life Science
Biota Ltd
Cytopia Ltd.
Rigel, Inc.
Celera Genomics
Prototek Inc.
Purdue University
Ph.D. Organic Chemistry

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

Co-inventor of
ibrutinib at Celera

Biomea's Development Principles



Validated Disease Targets

Drugs pursuing **validated targets** have a ~2x higher likelihood of approval than molecules pursuing a new mechanism of action

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

Covalent Sm. Mol. Inhibitors

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

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

Proprietary Combinations

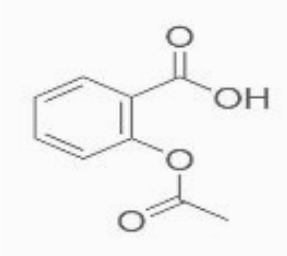
Combination therapy with non-overlapping resistance mechanisms results in more durable responses and better outcomes

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

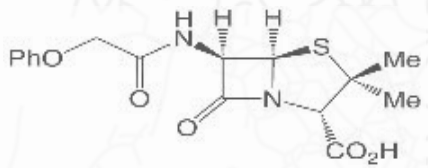
Covalent Inhibitors - a History of Medical & Commercial Success

Notable Covalent Inhibitors

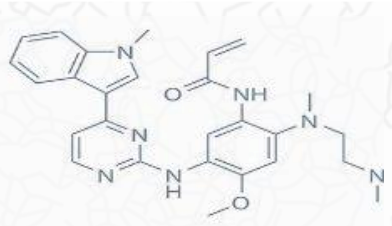
Aspirin

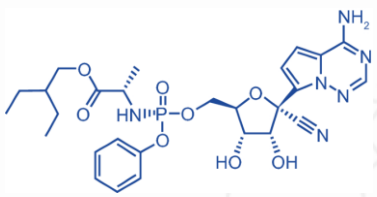


Penicillin

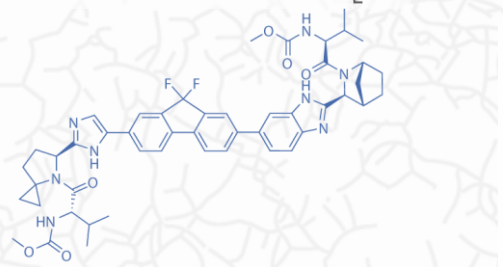


Osimertinib (TAGRISSO)

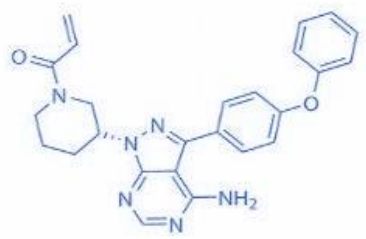




Remdesivir (VEKLURY)



Ledipasvir (HARVONI)

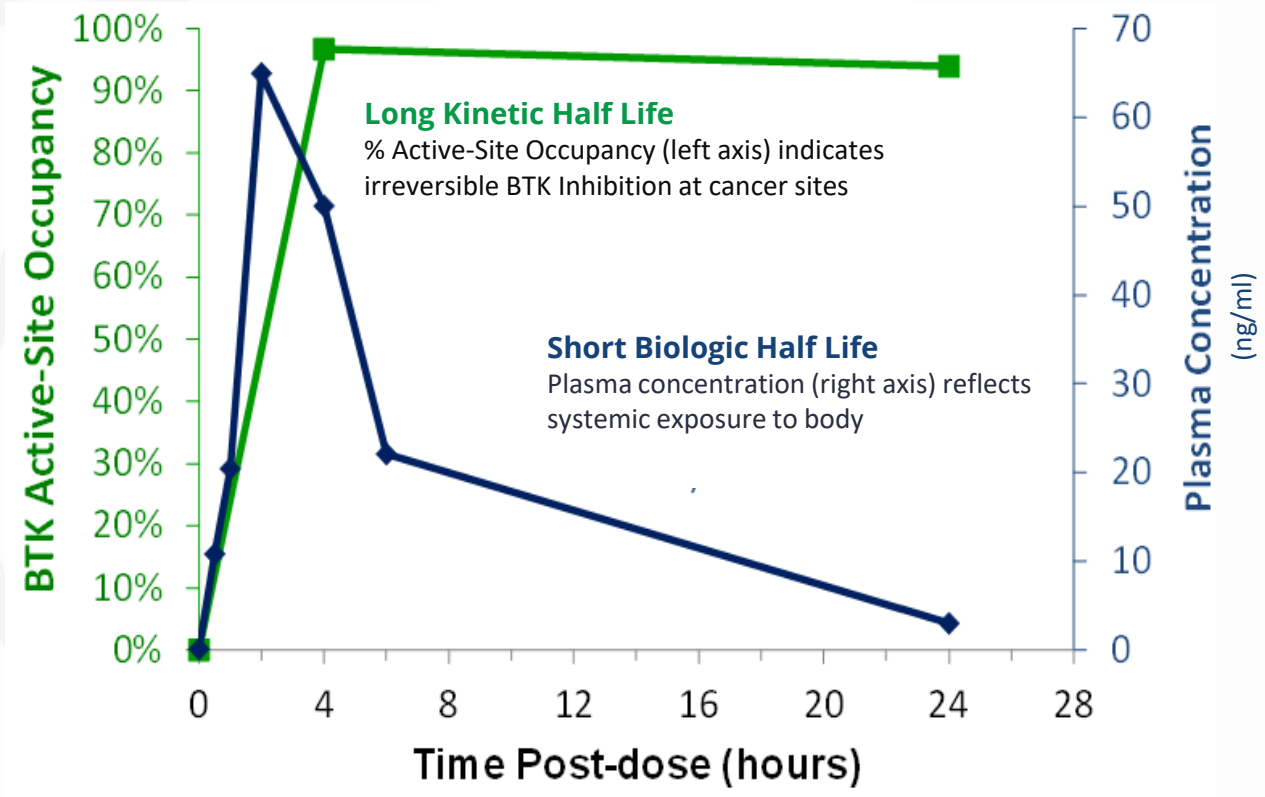


Ibrutinib (IMBRUVICA)

Compounds in blue were invented by Biomea Fusion senior leadership

- **Aspirin** was the first commercialized covalent drug
- Notable precision oncology and infectious disease programs leverage covalent mechanisms
  - Precision Oncology**:**
    Osimertinib and **Ibrutinib** both target kinases and are used in subpopulations with specific genetic biomarkers
  - Antivirals**:**
    Remdesivir and **Tenofovir** both target reverse transcriptases and are leveraged to treat HCV and other viruses including HIV and COVID-19

Imbruvica – a Covalent Inhibitor with Long Kinetic but very Short Biological Half Life



High Selectivity

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

Deep Target Inactivation

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

Greater Therapeutic Window

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

Our Technology Platform – The FUSIONTM SYSTEM

Target identification to IND candidate in 18 months

Target to Hit

Target validation
Visual integration of crystal structures of target and reactive cysteine

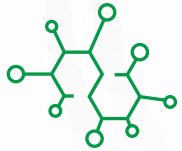
Utility:
Differentiated insights from X-ray crystal structures, identifying target cysteines

Custom Lead

Library of custom engagers
Proprietary AI platform with VR validation matches novel DRUG LIKE PROBES to cysteines; we do not screen via library probes.

Utility:
Library of covalent scaffolds provide for ~1,000 de novo scaffolds for AI/VR scoring

Lead Optimization



Custom scaffold creation
Custom built Synthesis to create candidates with desired

Utility:
AI/VR program platform yields over 300 scaffolds, which are synthesized for in vitro testing

IND

Refinement
Building in drug-like properties, optimizing PK/PD profile, and maintaining specificity

Utility:
Scaffolds are further refined with Mass spec, animal, and cell-based assays to two IND candidates

Meaningful Clinical Progress in 2022

All Corporate Milestones as Presented at JPM January 22 on track or completed

BMF-219 – Liquid Tumors

IND Clearance	Completed ✓
DLBCL Preclinical ASH 2021 Abstract	Completed ✓
BMF-219 Ph. I AML Trial Initiation	In Progress ✓
Additional Preclinical Data in DLBCL/MM	H1 2022 ✓
BMF-219 Ph. I DLBCL/MM Trial Initiation	H1 2022 ✓

BMF-219 – Solid Tumors

Additional Preclinical Data in KRAS Tumors	H1 2022 ✓
IND Filing	H2 2022 ✓
BMF-219 Ph. I KRAS Mut. Trial Initiation	Q4 2022

Menin Inh. – Diabetes

Diabetes Menin Pathway Validation	H1 2022 ✓
IND Filing	H2 2022
Ph. I Diabetes Trial Initiation	H2 2022 ✓

Additional Programs

NEW 2nd Pipeline Candidate Announced	H1 2022 ✓
NEW 3rd Pipeline Candidate Announced	To be announced

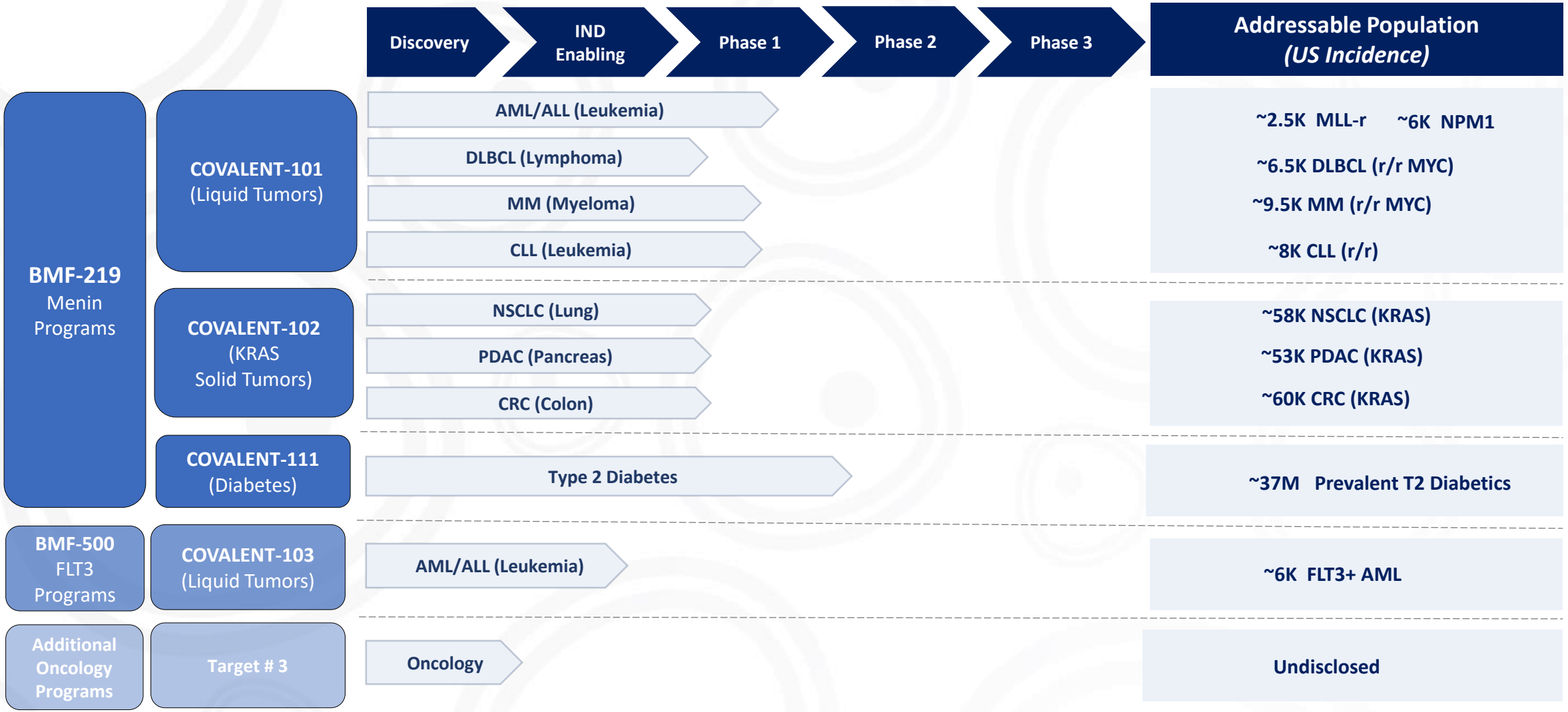
✓ Completed

In Progress

biomea

Biomea Expanding into Eight Different Solid and Liquid Tumors as well as Type 2 Diabetes

Pipeline



Multiple Clinical Read Outs over the coming Quarters

Near Term Milestones – Biomea Fusion (NASDAQ: BMEA)

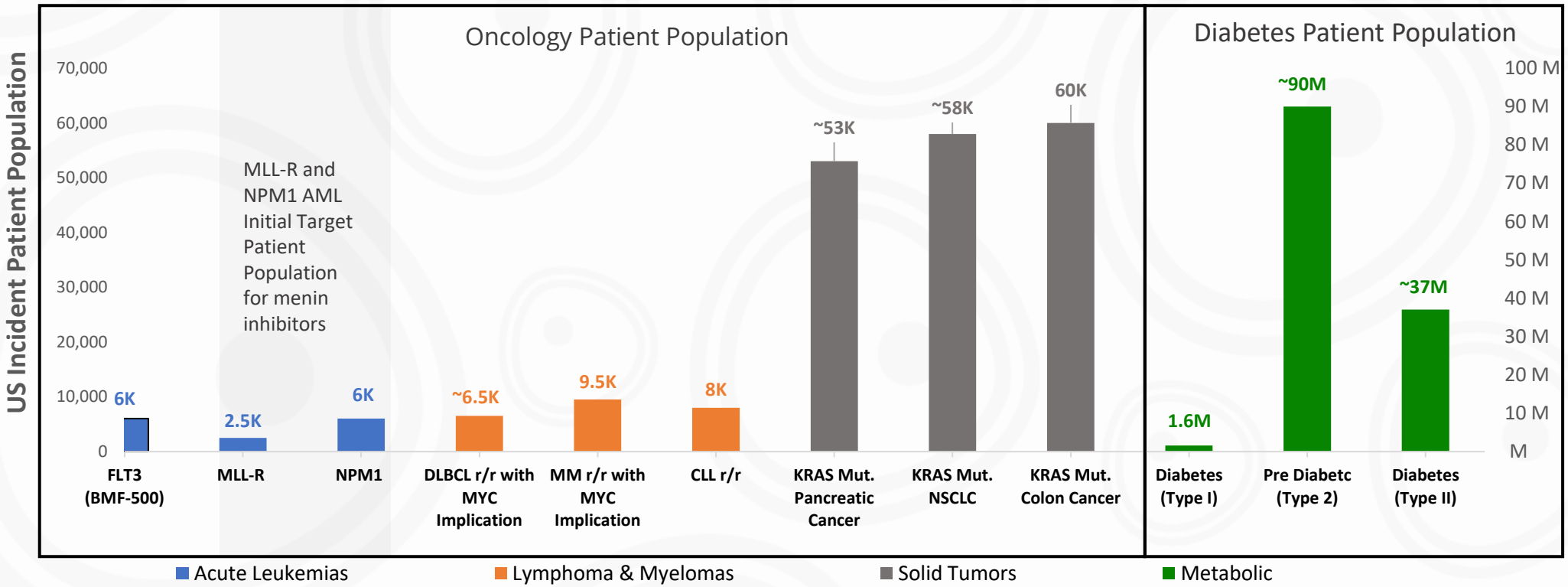
			Key Milestones	Expected Timeline
BMF-219 Menin Programs	COVALENT-101 (Liquid Tumors)	AML/ALL (Leukemia)	Phase I: Clinical Data in AML	1H 2023
		DLBCL (Lymphoma)	Initiation of Phase I additional Cohort in DLBCL	In Progress
		MM (Lymphoma)	Enrolling in Phase I additional Cohort in MM	In Progress
		CLL (Lymphoma)	Enrolling in Phase I additional Cohort in CLL	In Progress
	COVALENT-102 (KRAS Solid Tumors)	NSCL (Lung)	Initiation of Phase I KRAS Study in CRC, PDAC, NSCLC	In Progress
		PDAC (Pancreas)		
		CRC (Colon)		
	COVALENT-111 (Diabetes)	Type 2 Diabetes	Healthy Volunteers of PI/II COVALENT-111 Trial	Completed
			Phase II: Clinical Data in Type 2 Diabetes	In Progress
BMF-500 FLT3 Programs	COVALENT-103 (Liquid Tumors)	AML/ALL (leukemia)	Preclinical Data Presentation	ASH 2022
			IND Filing	1H 2023
Additional Oncology Programs	Target # 3	Oncology	Progress Update	1H 2023

biomea FUSION™ We Aim to Cure™

BMF-219 and BMF-500 Patient Populations in the US

Cancer Indications: >200K and Diabetes: >125M

Addressable Annual US Patient Population for BMF-219



Sources: Jovanović, K. K., Roche-Lestienne, C., Ghobrial, I. M., Facon, T., Quesnel, B., & Manier, S. (2018). Targeting MYC in multiple myeloma. Leukemia, 32(6), 1295–1306. https://doi.org/10.1038/s41375-018-0036-x ; Riedell, P. A., & Smith, S. M. (2018). Double hit and double expressors in lymphoma: Definition and treatment. Cancer, 124(24), 4622–4632. https://doi.org/10.1002/cncr.31646; Kempf, E., Rousseau, B., Besse, B., & Paz-Ares, L. (2016). KRAS oncogene in lung cancer: focus on molecularly driven clinical trials. European respiratory review : an official journal of the European Respiratory Society, 25(139), 71–76. https://doi.org/10.1183/16000617.0071-2015; Lanfredini, S., Thapa, A., & O'Neill, E. (2019). RAS in pancreatic cancer. Biochemical Society transactions, 47(4), 961–972. https://doi.org/10.1042/BST20170521; Serna-Blasco, R., Sanz-Álvarez, M., Aguilera, Ó., & García-Foncillas, J. (2019). Targeting the RAS-dependent chemoresistance: The Warburg connection. Seminars in cancer biology, 54, 80–90. https://doi.org/10.1016/j.semcancer.2018.01.016; Park, W., Chawla, A., & O'Reilly, E. M. (2021). Pancreatic Cancer: A Review. JAMA, 326(9), 851–862. https://doi.org/10.1001/jama.2021.13027; NCI SEER Estimated 2021 Incidence <seer.cancer.gov>

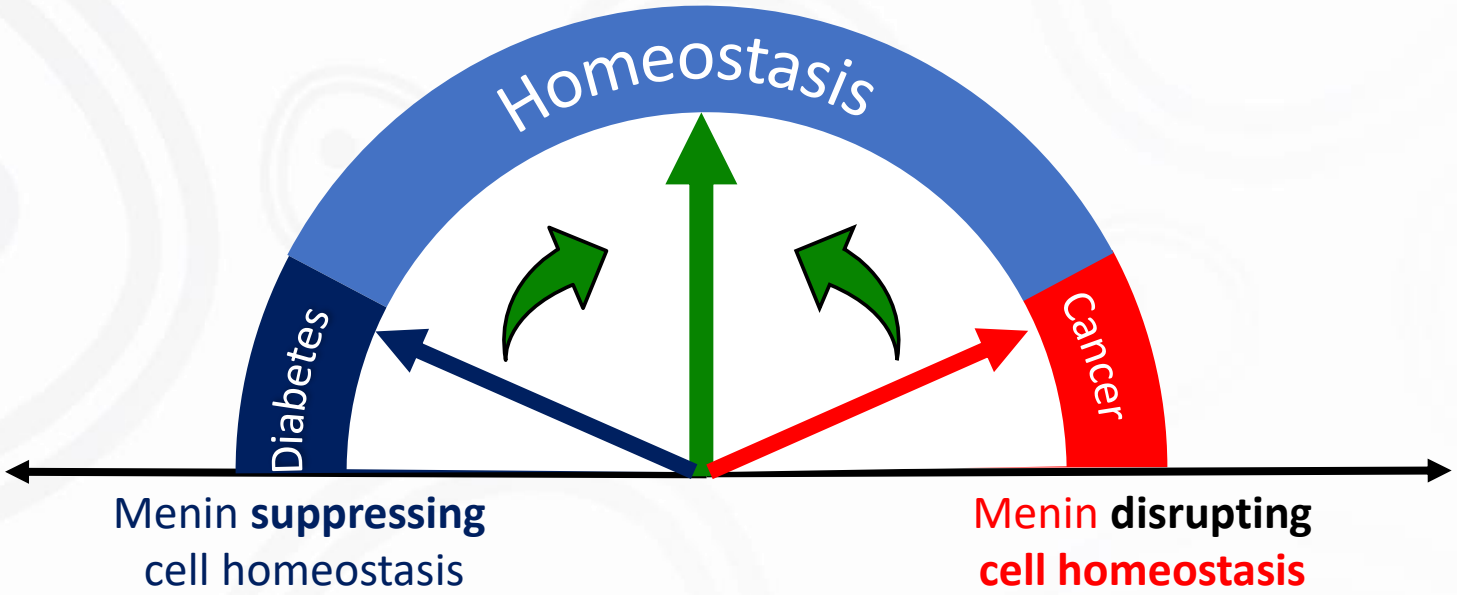
Restoring Balance in Menin Dependent Diseases

Treating Diabetes

BMF-219 selectively **enables cell homeostasis** of menin dependent beta cells

Treating Cancer

BMF-219 selectively **enables cell homeostasis** of menin dependent cancer cells



BMF-219 Was Highly Selective in Key Screening and Safety Panels

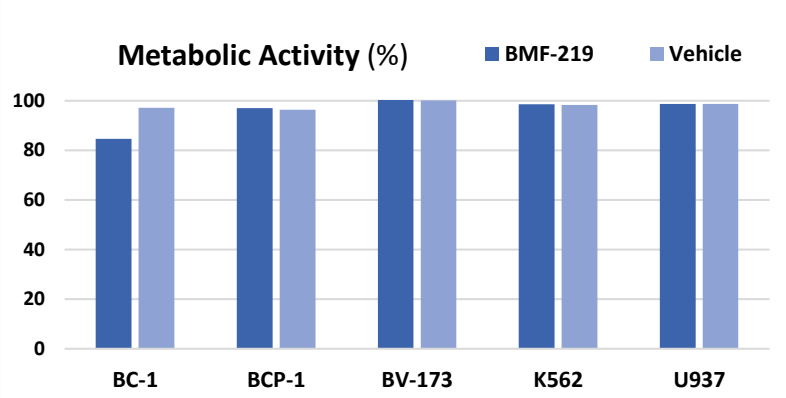
No Histopathology Findings Were Observed with BMF-219 in GLP and non-GLP IND-Enabling Toxicology Studies

✓ Kinase Screening

169 kinases screened; only **two** showed >50% inhibition with BMF-219

✓ Oncopanel Screen

Minimal impact of BMF-219 on cell metabolism in leukemia and lymphoma cell lines that have **wild type MLL1**



✓ Safety Screen

SafetyScreen44 panel (CEREP/Eurofins Discovery)* showed **no meaningful impact** (>50% activation or inhibition)

*SafetyScreen44 *in-vitro* panel of 44 common selected targets to identify significant off-target interactions

✓ Glutathione Reactivity

BMF-219 had **less reactivity than the approved covalent drugs omeprazole and neratinib**

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

Novel Covalent Inhibitor of Menin

BMF-219

Pipeline-in-a-Pill – Single Agent for Multiple Indications

MLL Fusion & NPM1 Driven Tumors

Initial clinical validation in r/r acute leukemias with MLL fusions in addition to NPM1 mutations

MYC Addicted and MYC Driven Tumors

Expansion into r/r diffuse large b cell lymphoma, r/r multiple myeloma and r/r chronic lymphocytic leukemia

RAS/RAF Driven Solid Tumors

Further expansion into KRAS and RAS mutant colorectal, lung, and pancreatic cancer

Diabetes

Pathway and clinical validation of covalent menin inhibition

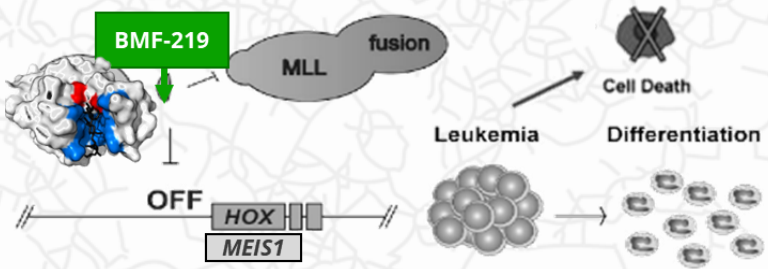
First Development Success with BMF-219 in MLL Fusion and NPM1 Driven Tumors

In Acute Leukemia

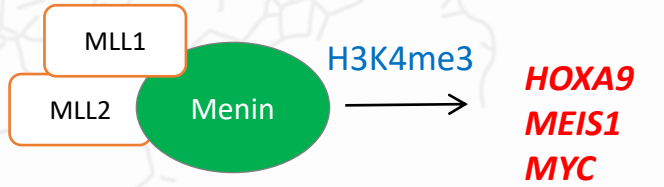
Development Stage:
Phase I Clinical Trial (COVALENT-101) enrolling patients with relapsed/refractory acute leukemia

Key Facts	MOA	Relevant Pathway
Estimated Addressable Population	BMF-219 covalently blocks menin / MLL interaction	Menin / MLL interaction can modify chromatin, activating key leukemic genes

Acute Leukemia (Mutation)	Estimated US Patient Population (Annual Incidence)
MLL-r	~2,500
NPM1 mutant	~6,000
Ras Driven	~6,000



- BMF-219 directly inhibits MLL-menin interaction and was optimized to cause cell killing, rather than cell differentiation.
- In preclinical studies, BMF-219 shows robust cell killing and reduction of expression of key genes (including MYC, MEIS1, HOXA9, and BCL2)

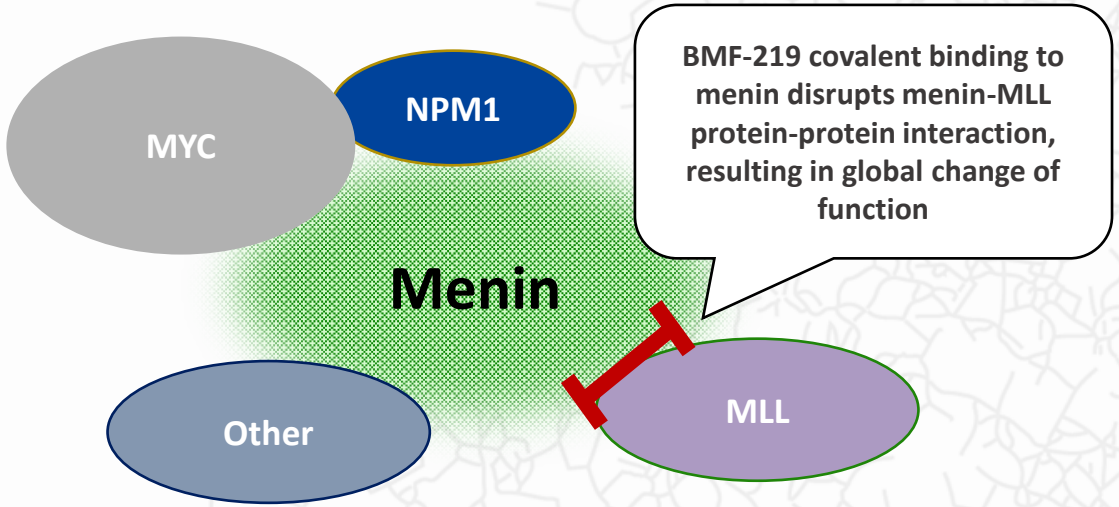


Menin / MLL complex forms and modifies chromatin at histone H3, activating *HOXA9* and *MEIS1*

First Development Success with BMF-219 in MLL Fusion and NPM1 Driven Tumors

BMF-219 has the Potential to Impact Important Binding Partners in Multiple Tumors

Mechanism of Action



Resulting change of function of menin impacts important binding partners involved in oncogenesis

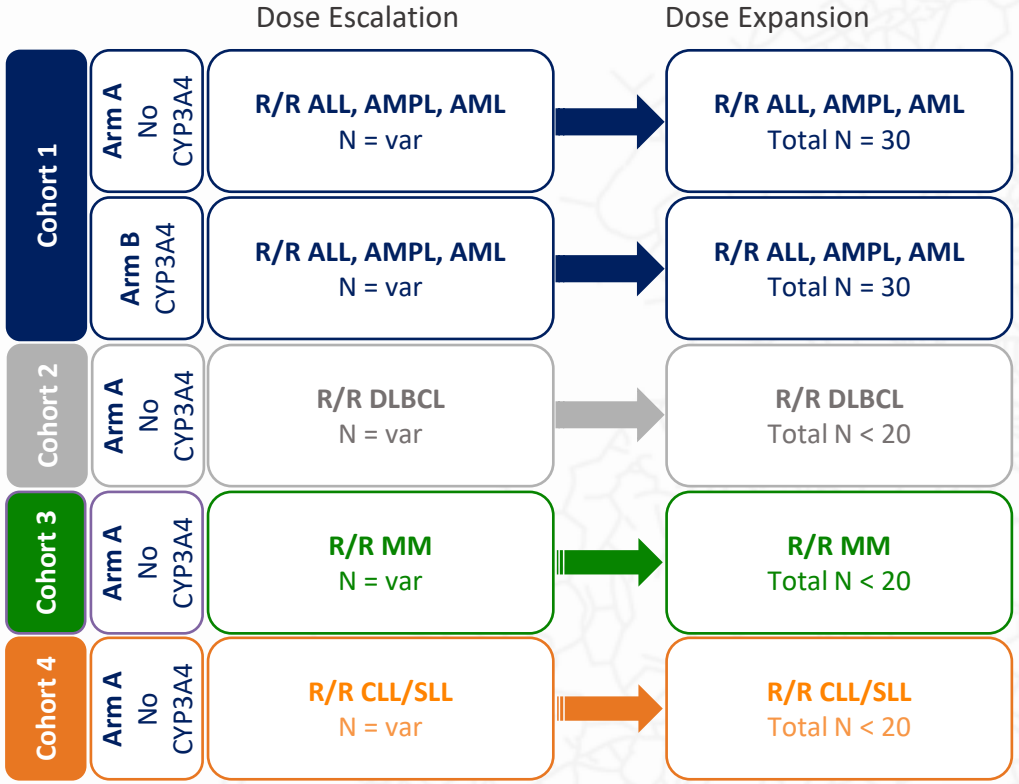
Target Patient Population

- Acute Leukemia: MLL-r
- Acute Leukemia: NPM1 mutant
- Acute Leukemia: Ras mutant
- DLBCL: DHT / DEL
- Multiple Myeloma: MYC addicted
- KRAS mutant Solid Tumors:
  Colorectal
  Lung
  Pancreatic
- CLL: r/r population
- Liquid and Solid Tumors

BMF-219 has the potential to address additional patient populations with tumors that are dependent on menin or some of its binding partners

COVALENT-101 (ENROLLING 4 COHORTS)

Phase I first-in-human dose-escalation and dose-expansion study of BMF-219 enrolling adult patients with r/r acute leukemia, r/r diffuse large B cell lymphoma, r/r multiple myeloma, and r/r chronic lymphocytic leukemia (CLL) (NCT05153330)



Accelerated titration design followed by classical 3+3

Cohort 1 for R/R AML/AMPL/AML patients
Cohort 2 for R/R DLBCL with ≥ 2L of prior therapy
Cohort 3 for R/R MM with ≥ 3L of prior therapy
Cohort 4 for R/R CLL/SLL with ≥ 2L of prior therapy

Study Treatment: BMF-219

- A covalent small molecule menin inhibitor, administered orally daily in 28-day cycles

Objectives

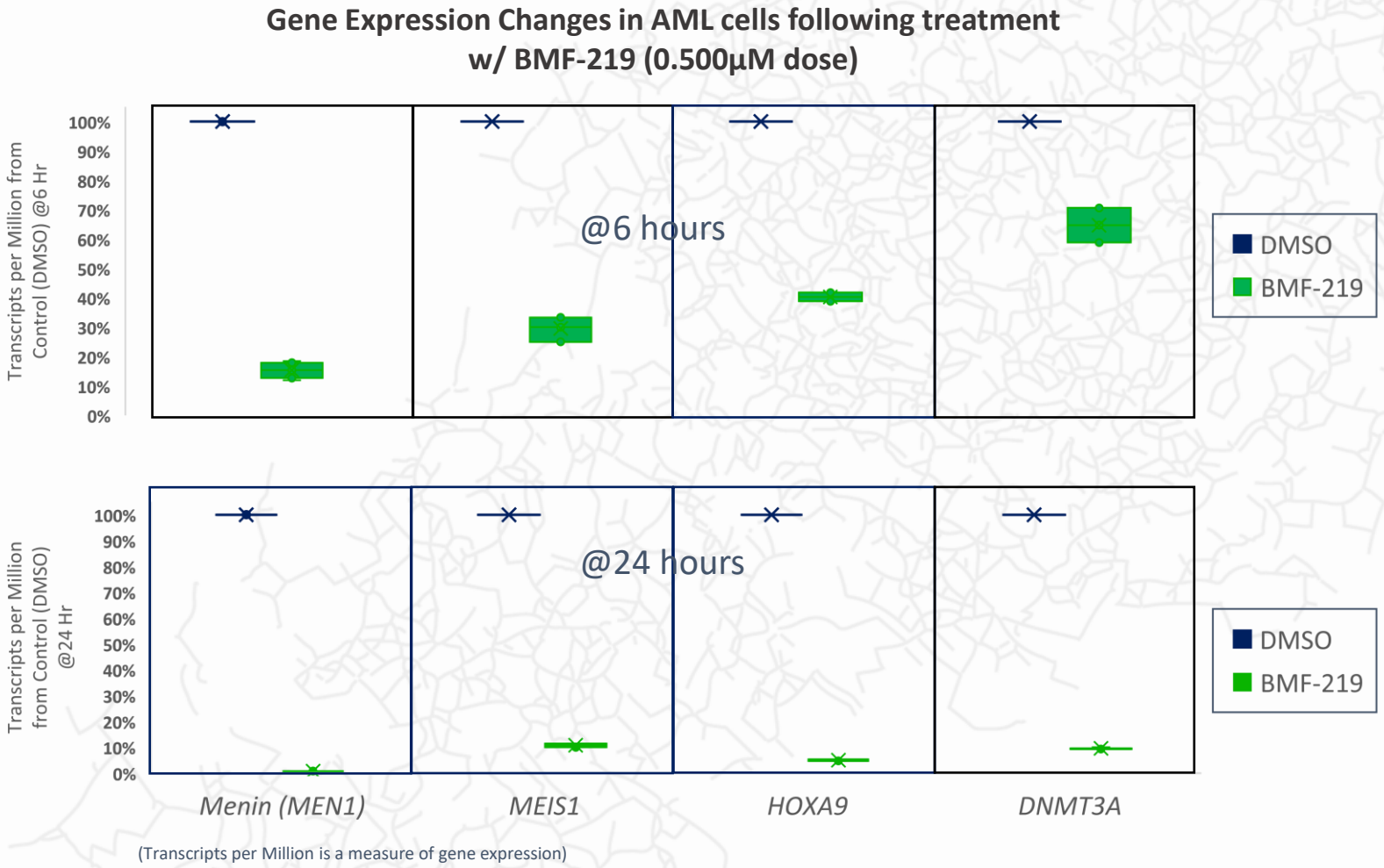
- Primary: Determine OBD & RP2D of BMF-219 monotherapy independently for each Cohort and Arm
- Secondary: Evaluate safety and tolerability of BMF-219
  Determine PK/ PD parameters of BMF-219
  Explore additional evidence of efficacy and antitumor activity

BMF-219 is being studied in seven different blood cancers. The design of COVALENT-101 is the following: Dose escalation of each cohort is done in parallel followed by independent dose selection and dose expansion phase.

Abbreviations: ALL Acute Lymphoblastic Leukemia AML Acute Myeloid Leukemia AMPL Acute Mixed-Phenotype Leukemia CYP3A4 Cytochrome 450 OBD Optimal biologic dose DLBCL diffuse large B-cell lymphoma MM multiple myeloma R/R Relapsed/Refractory

First Development Success with BMF-219 in MLL Fusion and NPM1 Driven Tumors

BMF-219 Demonstrated Rapid and Near Complete Reduction of Expression of Oncogenes

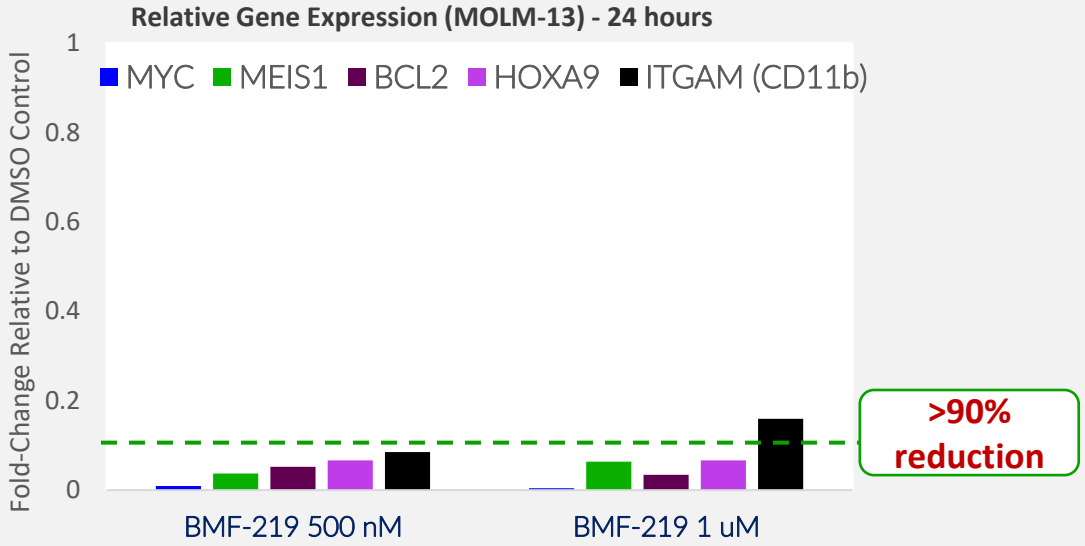


(Transcripts per Million is a measure of gene expression)

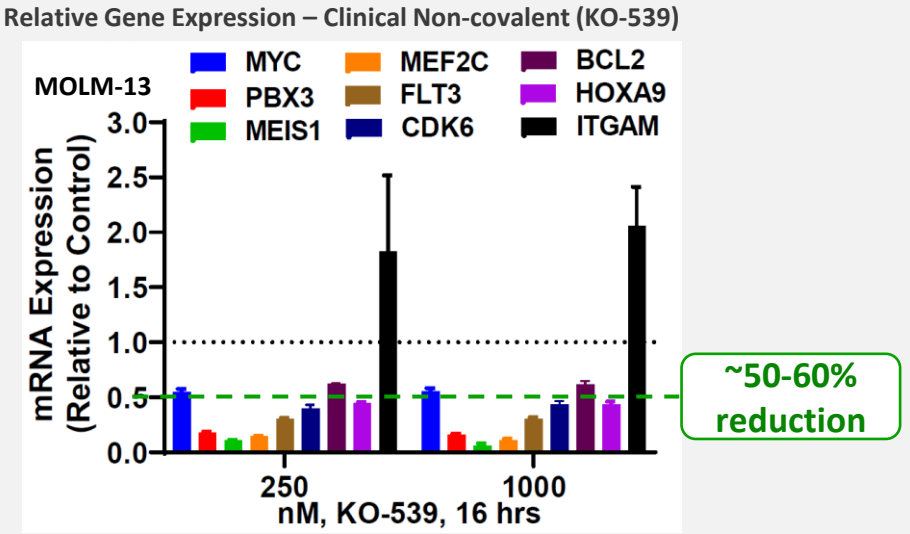
- Covalent inhibitor, BMF-219, downregulates expression of Menin (via the target *MEN1* gene) and critical leukemogenic genes (e.g., *MEIS1* and *HOXA9*)
  - *MEIS1* is a gene that can be an accelerator of leukemic transformation (along with *HOXA9*)
  - *HOXA9* is a gene involved in myeloid differentiation and can be leukemogenic
  - *DNMT3A* is a gene that codes for a methyltransferase, which can be leukemogenic when mutated
- BMF-219 demonstrated up to 80% reduction in readout genes by 6 hours and approximately 90%+ reduction at 24 hours

First Development Success with BMF-219 in MLL Fusion and NPM1 Driven Tumors

BMF-219 Displayed Superior Impact on Key Gene Signatures in MLL-rearranged AML Cell Line



Somanath et al., ASCO 2022 Abstract 7541



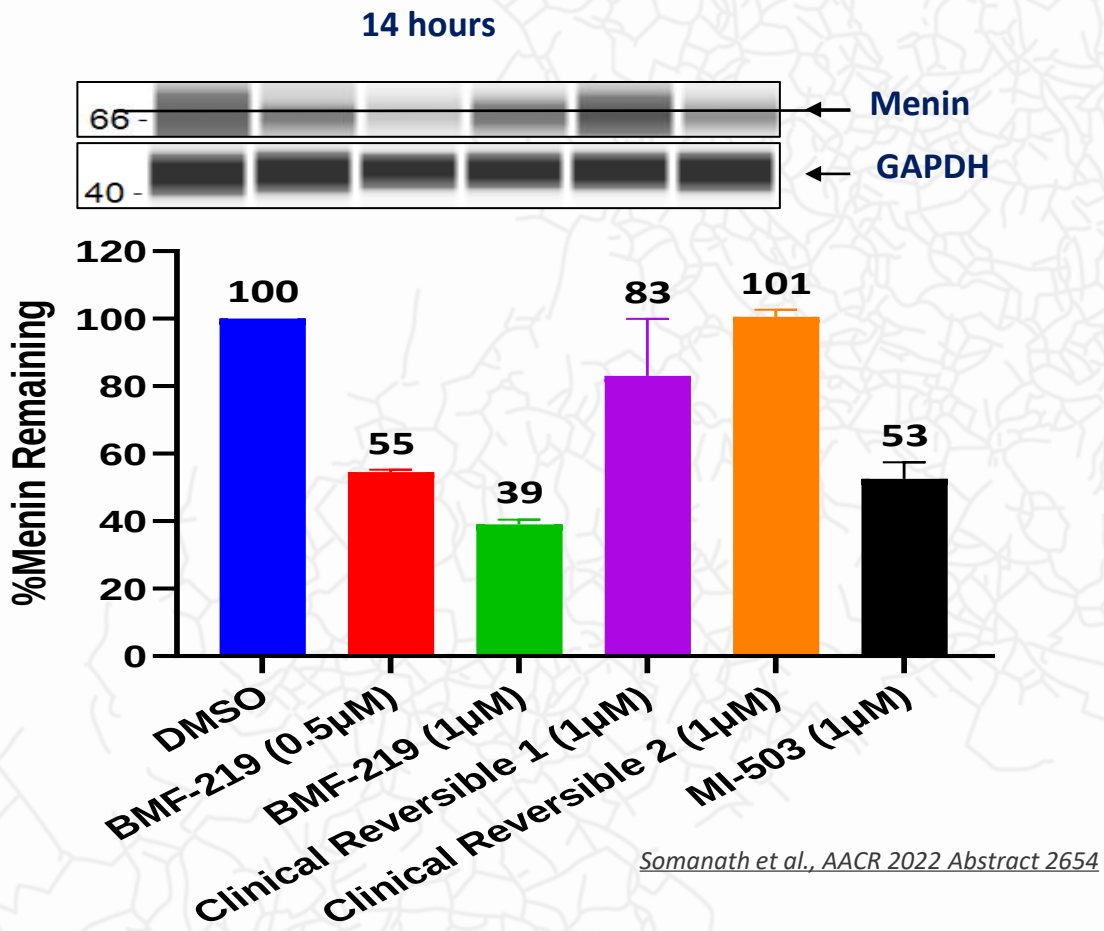
Blood (2021) 138 (Supplement 1): 3357

- Differentiation marker, *ITGAM (CD11b)*, expression increases 2 to 3-fold at 6 hours, followed by ***~8 to 10-fold*** reduction at 24 hours with BMF-219
- *MEIS1* expression is reduced ***~10 to 20-fold*** at 24 hrs with BMF-219
- *HOXA9* expression decreases ***~15-fold*** at 24 hrs with BMF-219
- *BCL2* expression decreases ***~20 to 30-fold*** at 24 hrs post-treatment with BMF-219
- *MYC* expression is reduced ***~100 to 200-fold*** at both 6 and 24 hrs post-treatment with BMF-219

First Development Success with BMF-219 in MLL Fusion and NPM1 Driven Tumors

BMF-219 Significantly Reduces Menin Protein in DLBCL Cell Line

Menin Protein Levels in BMF-219 Toledo (DLBCL- DHL) cell line

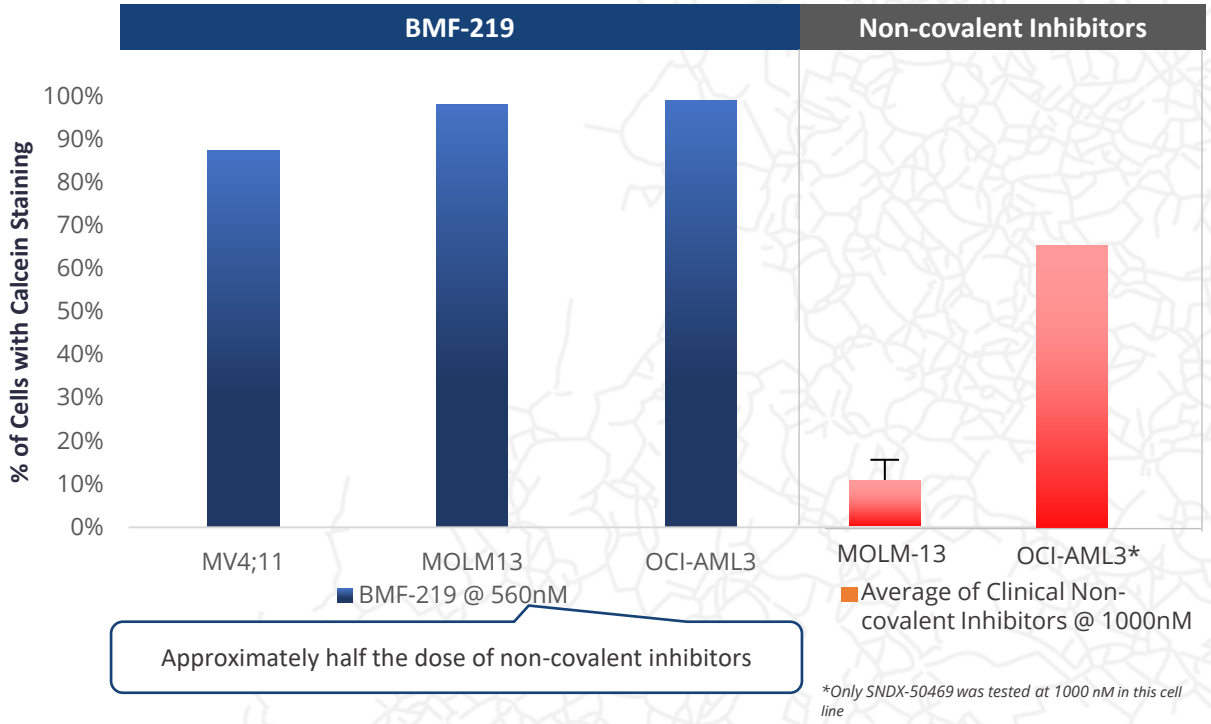


Somanath et al., AACR 2022 Abstract 2654

- Covalent inhibitor, BMF-219, at 1µm concentration achieves >60% reduction of menin protein at 14hrs
- Clinical reversible (non-covalent) inhibitors of menin achieved less than 20% reduction of menin protein at the same concentration

First Development Success with BMF-219 in MLL Fusion and NPM1 Driven Tumors

BMF-219 Superior Cell killing of the Target AML Cell Lines at Half the Dose vs Reversible Competitive Menin Inhibitors



*Only SNDX-50469 was tested at 1000 nM in this cell line

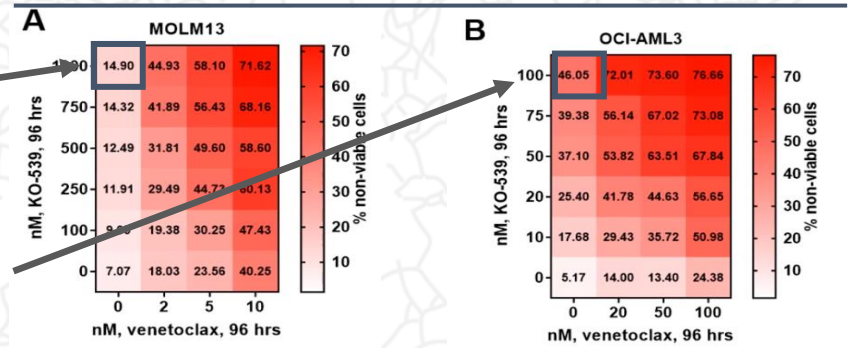
- BMF-219 **killed >90% of AML cells** in MLL-rearranged and NPM1 mutant cell lines at 4 days post-treatment
- Non-covalent menin inhibitors generally report significantly less cell killing of AML cell lines as a single agent

Source Data from ASH 2021 Publications

KO-539 is 15% effective @ 1000nM in MOLM-13 cells and 46% effective @ 1 100nM in OCI-AML3 cells

15% effective @ 1000nM

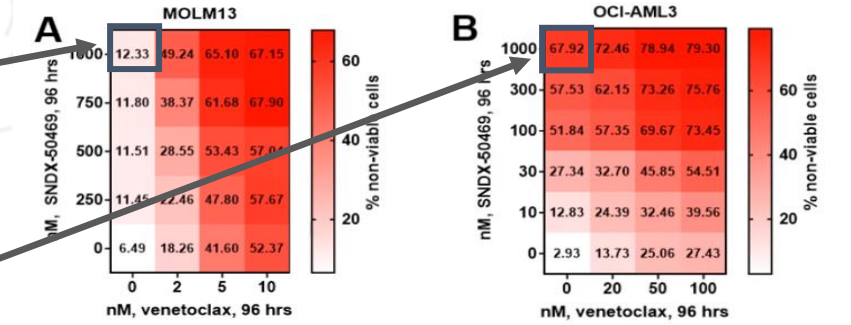
46% effective @ 100nM



SNDX-50469 is 12% effective @ 1000nM in MOLM-13 cells And 68% effective @ 1000nM in OCI-AML3 cells

12% effective @ 1000nM

68% effective @ 1000nM

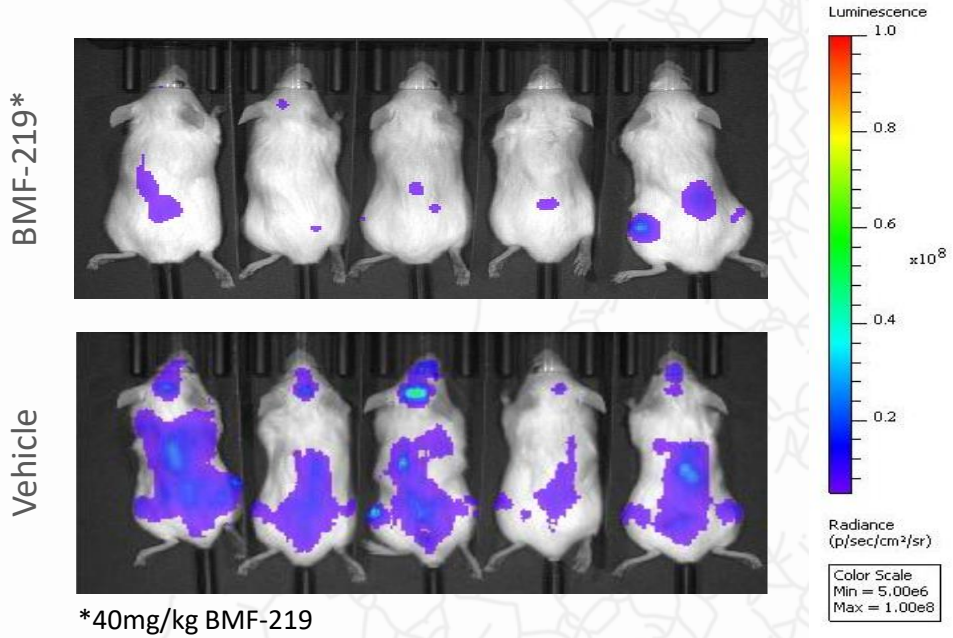


Blood (2021) 138 (Supplement 1): 3340., ASH 2021.

First Development Success with BMF-219 in MLL Fusion and NPM1 Driven Tumors

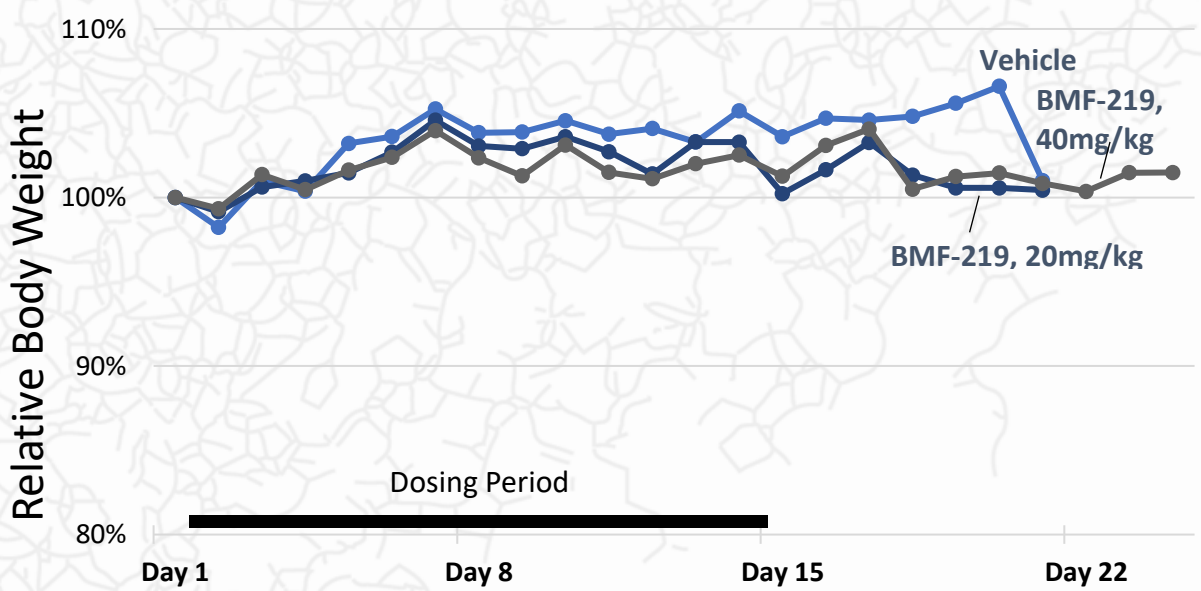
BMF-219 Achieved Significant Survival Benefit in A Disseminated Leukemia Xenograft Model

Anti-Tumor Effect



*40mg/kg BMF-219

Body Weight



- Mice were inoculated with xenograft cancer cells at high levels ($1x10^7$ MV4;11-luc) with greater than 90% viability
- BMF-219 treatment showed notable reduction in tumor burden and **survival benefit over vehicle control (72% at 20mg/kg and 94% at 40mg/kg)**
- Daily dosing for 14 days was well-tolerated and caused **minimal body weight changes**

Novel Covalent Inhibitor of Menin

BMF-219

Pipeline-in-a-Pill – Single Agent for Multiple Indications

MLL Fusion & NPM1 Driven Tumors

Initial clinical validation in r/r acute leukemias with MLL fusions in addition to NPM1 mutations

MYC Addicted and MYC Driven Tumors

Expansion into r/r diffuse large b cell lymphoma, r/r multiple myeloma and r/r chronic lymphocytic leukemia

RAS/RAF Driven Solid Tumors

Further expansion into KRAS and RAS mutant colorectal, lung, and pancreatic cancer

Diabetes

Pathway and clinical validation of covalent menin inhibition

First Development Success with BMF-219 in MYC Addicted and MYC Driven Tumors

In Diffuse Large B-cell Lymphoma (DLBCL), Multiple Myeloma (MM) and Chronic Lymphocytic Leukemia (CLL)

Development Stage: Phase I Clinical Trial (COVALENT-101) enrolling patients with relapsed/refractory DLBCL, MM and CLL

Key Facts

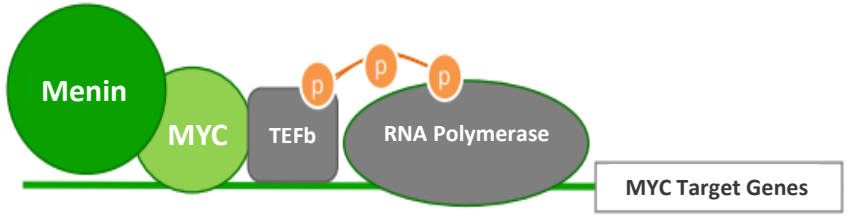
Estimated Addressable Population

Disease (r/r with MYC Implication)	Estimated US Patient Population (Annual Incidence)
DLBCL	~6,500
MM	~9,500

- MYC addiction tends to increase with stage and line of therapy
- ~20-50% MYC dysregulation or translocations in newly diagnosed MM patients
- ~50-70% of advanced r/r MM patients have MYC dysregulation
- ~10,000 (40%) of DLBCL patients are Double and Triple Hit and Double expressors (BCL2 and MYC overexpression)
- >50% of relapsed/refractory DLBCL are double expressors

MOA

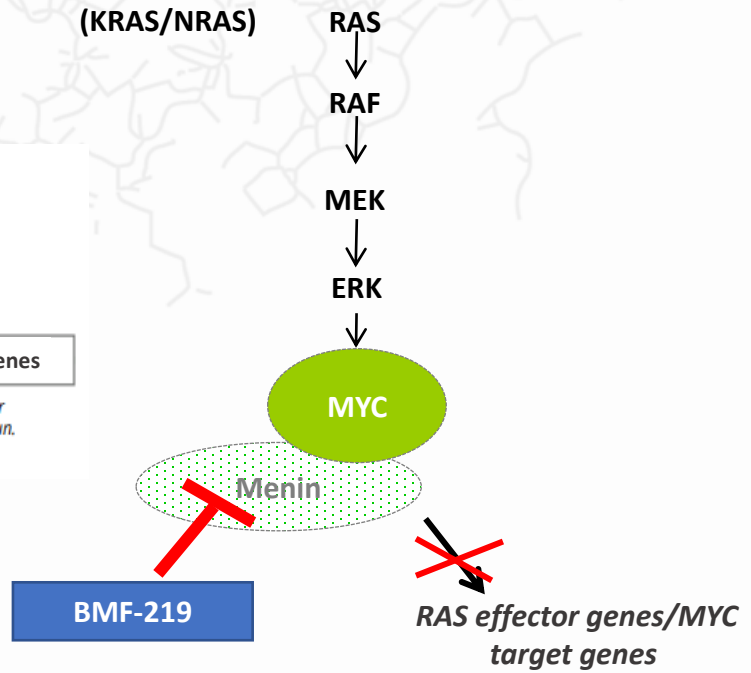
Menin complexes with MYC in the expression of MYC target genes. BMF-219 robustly decreases MYC gene expression and genomic function.
(Blood (2021) 138 (Supplement 1): 4318.)



Source: *Madden et al., Molecular Cancer volume 20, Article number: 3 (2021); Martínez-Martín et al. Cancer Drug Resist 2021;4:842-65; Xia Y. et al., Acta Haematol 2020;143:520–528; Zhu L., et al. (2017).. Nat. Commun. 8, 15278.; Musti et al., Oncogene . 2002 Sep 19;21(42):6434-45.*

Relevant Pathway

Tumor leverages MAPK pathway



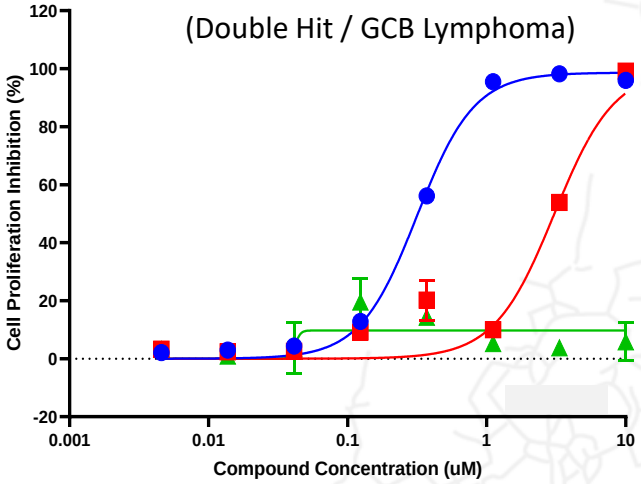
First Development Success with BMF-219 in MYC Addicted and MYC Driven Tumors

BMF-219 Led to near Complete Inhibition of Growth at 1µM in DLBCL Cell Lines

BMF-219 Growth Inhibition in DLBCL Cell Lines, ASH 2021

DB

(Double Hit / GCB Lymphoma)

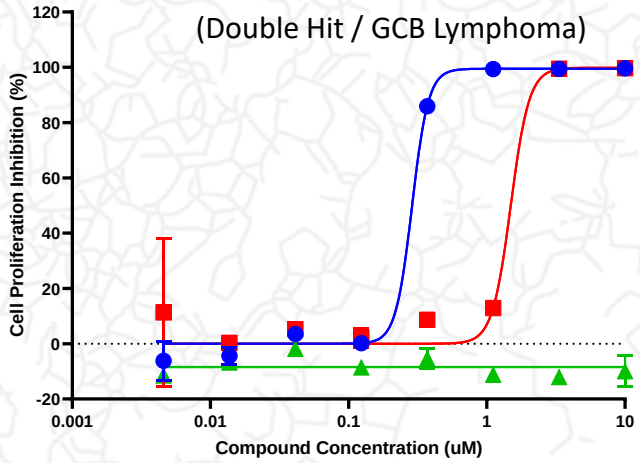


	IC50 (µM)	%Max
BMF-219	0.316	98.64
Clinical Non-covalent Inhibitor-1	3.07	100
Clinical Non-covalent Inhibitor-2	No Resp.	9.7

DB and Toledo cells were incubated with compounds for 4 days

TOLEDO

(Double Hit / GCB Lymphoma)



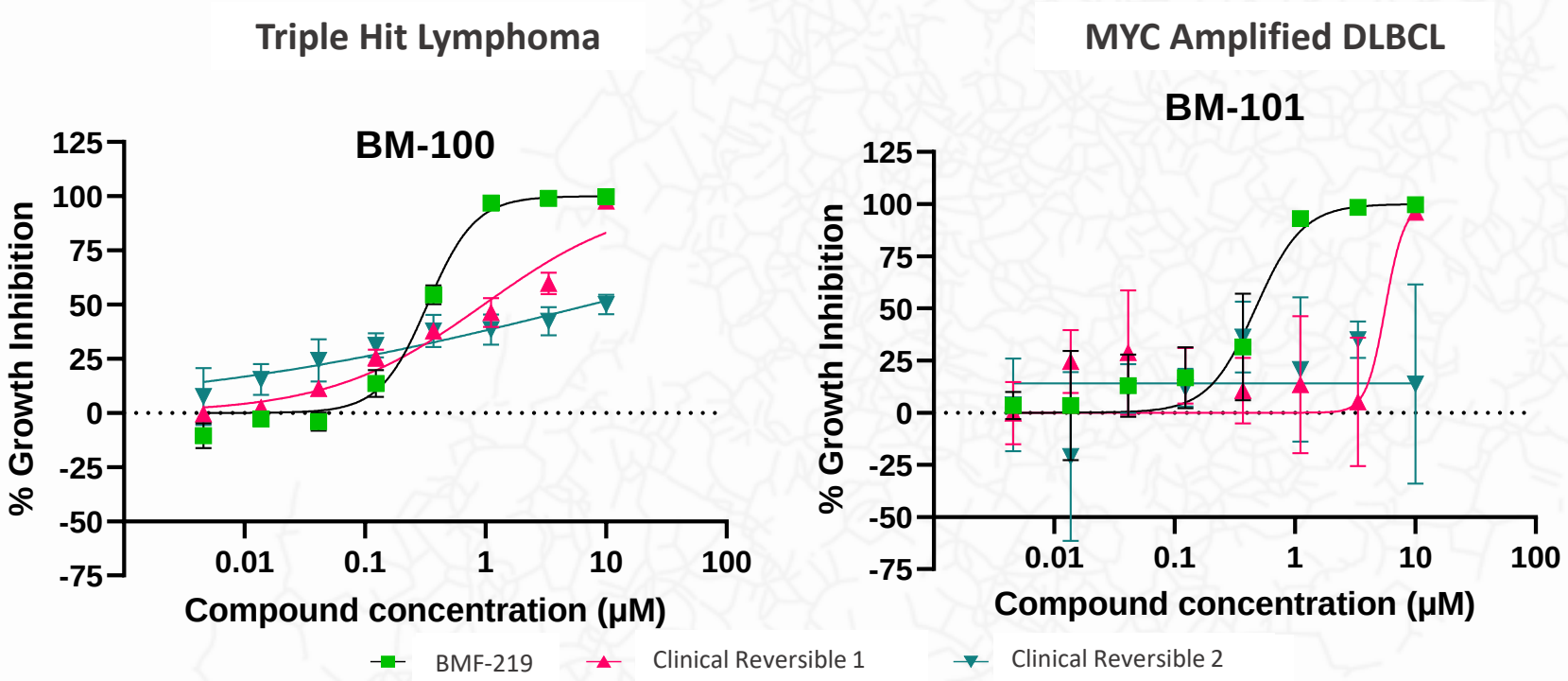
	IC50 (µM)	%Max
BMF-219	0.2877	99.47
Clinical Non-covalent Inhibitor-1	1.49	99.84
Clinical Non-covalent Inhibitor-2	No Resp.	-8.4

Source: Blood (2021) 138 (Supplement 1): 4318. ASH, 2021.

- Covalent menin inhibition by BMF-219 led to marked growth inhibition in multiple DLBCL cell lines
- We believe this is due to disruption of Menin-MYC
- One of the clinical stage non-covalent menin inhibitors tested displayed activity, but at 5-10x higher concentration
- The other clinical non-covalent inhibitor did not achieve IC50 in the tested cell lines at any concentration tested

Cell Lines	Cell Type	Translocations
DB	GCB-DLBCL	MYC/BCL2
TOLEDO	GCB-DLBCL	MYC/BCL2

BMF-219 Led to near Complete Inhibition of Growth at 1µM in DLBCL in ex-vivo Samples



Treatment	Growth Inhibition IC_{50} (mM)	
	BM100	BM101
BMF-219	0.250	0.151
Clinical Reversible-1	0.969	5.63
Clinical Reversible-2	6.31	Max killing <30%

- At ~1µM exposure, BMF-219 produces robust growth inhibition in both THL (triple hit lymphoma) and MYC amplified DLBCL ex-vivo cell lines
- BMF-219 responses were superior to clinical reversible (non-covalent) inhibitors with respect to cell growth inhibition at the concentrations tested

Somanath et al., AACR 2022 Abstract 2654

First Development Success with BMF-219 in MYC Addicted and MYC Driven Tumors

BMF-219 Exerts Potent Lethality Against Representative DLBCL (Toledo & U2932) & MM Cell Lines (SKMM1 & OPM2)

% Cell Death	SKMM1					OPM2				
	BMF-219			Clin Rev	MI-503	BMF-219			Clin Rev	MI-503
Conc.	0.4 µM	0.5 µM	1 µM	1 µM	3 µM	0.4 µM	0.5 µM	1 µM	1 µM	3 µM
14 hr	-	15	25	0	13	-	8	57	0	14
72 hr	27	-	86	4	33	22	-	80	3	21

% Cell Death	TOLEDO					U2932				
	BMF-219			Clin Rev	MI-503	BMF-219			Clin Rev	MI-503
Conc.	0.4 µM	0.5 µM	1 µM	1 µM	3 µM	0.4 µM	0.5 µM	1 µM	1 µM	3 µM
14 hr	-	18	12	0	11	-	19	36	0	7
72 hr	32	-	97	0	35	29	-	86	3	34

Lu et al., IMS 2022

To measure cell killing, cells were cultured in the presence of menin inhibitor for 72hr or 14hr and viable cell count measure by CTG readout. The % cell killing relative to untreated cultures was measured at 72hr and 14hr. Data tabulated was averaged from 2 independent experiments.

BMF-219 at 1 uM induced potent killing inducing 80-97% cell death following 72 hr drug treatment. In comparison, the reversible menin inhibitors MI-503 and a clinical reversible menin inhibitor were significantly less effective (20-35% cell killing with 3 uM MI-503)

First Development Success with BMF-219 in MYC Addicted and MYC Driven Tumors

In Chronic Lymphocytic Leukemia (CLL)

Development Stage: Phase I Clinical Trial (COVALENT-101) enrolling patients with relapsed/refractory CLL

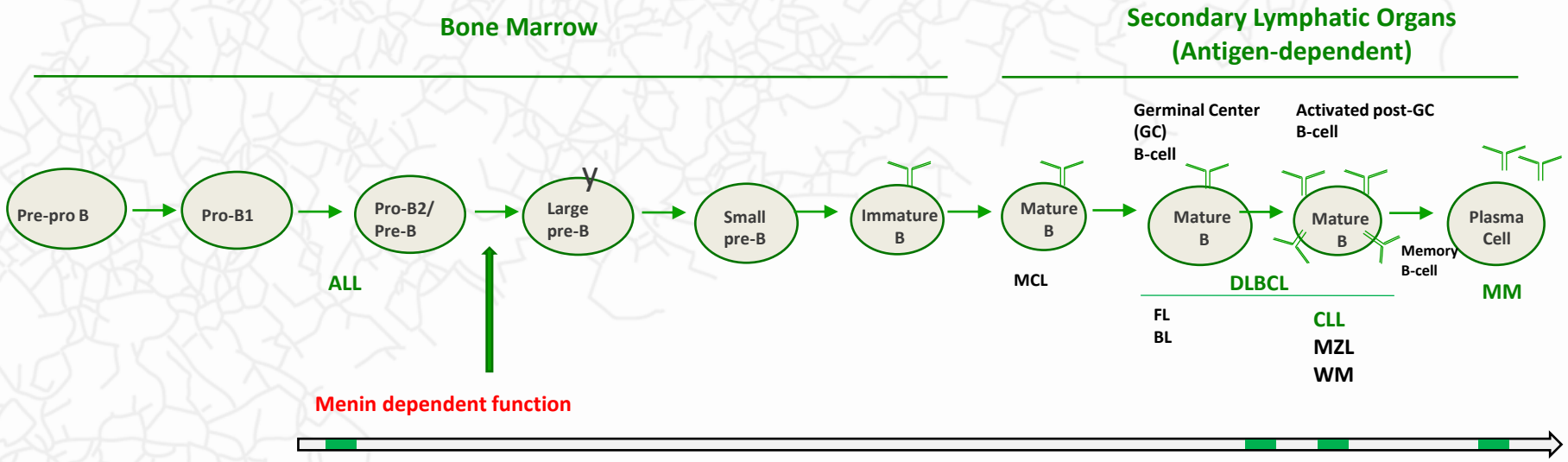
Key Facts

Disease	Estimated US Patient Population (Annual Incidence)
CLL	~20,000

MOA

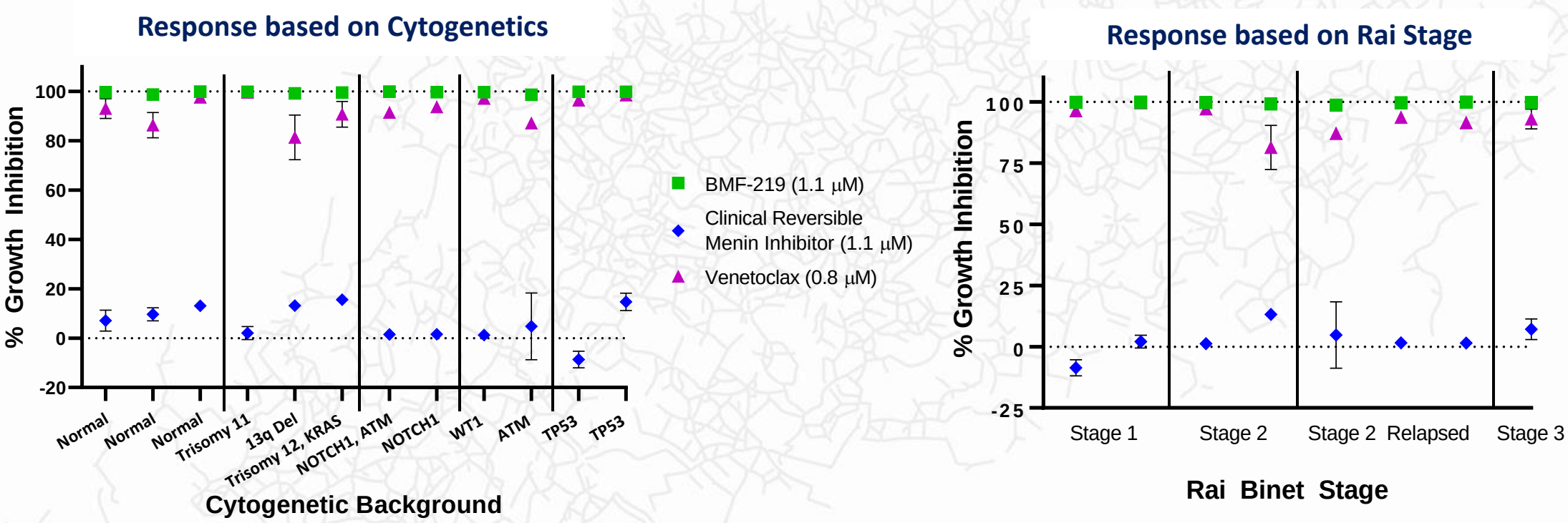
Menin complexes with MYC in expression of MYC target genes. BMF-219 robustly decreases MYC gene expression and downregulates the expression of BCL2 in cells.

Relevant Pathway



BMF-219 Achieves >98% Cell Lethality Against Diverse CLL ex vivo models

Growth inhibition of BMF-219 in CLL *ex vivo* models grouped by genetic background and Rai stage



Somanath et al., ASCO 2022 Abstract 7541

Novel Covalent Inhibitor of Menin

BMF-219

Pipeline-in-a-Pill – Single Agent for Multiple Indications

MLL Fusion & NPM1 Driven Tumors

Initial clinical validation in r/r acute leukemias with MLL fusions in addition to NPM1 mutations

MYC Addicted and MYC Driven Tumors

Expansion into r/r diffuse large b cell lymphoma, r/r multiple myeloma and r/r chronic lymphocytic leukemia

RAS/RAF Driven Solid Tumors

Further expansion into KRAS and RAS mutant colorectal, lung, and pancreatic cancer

Diabetes

Pathway and clinical validation of covalent menin inhibition

First Development Success with BMF-219 in RAS/RAV Driven Solid Tumors

In KRAS Mutant Solid Tumors (Lung, Colon, Pancreatic)

Development Stage: IND Stage in relapsed/refractory KRAS mutant Solid Tumors

Key Facts

Estimated Addressable Population

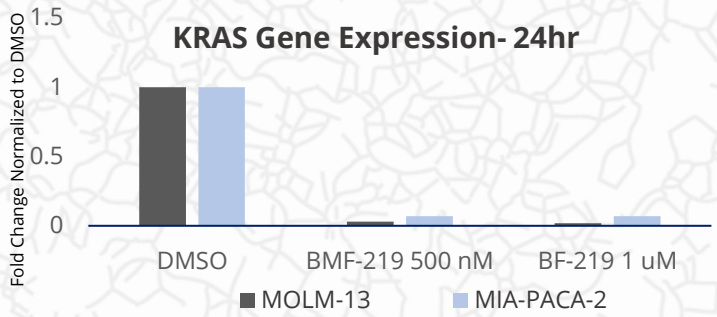
Tumor Type (KRAS Mutant)	Estimated US Patient Population (Annual Incidence)
Lung (NSCLC)	~58,000
Colon (CRC)	~60,000
Pancreatic (PDAC)	~53,000

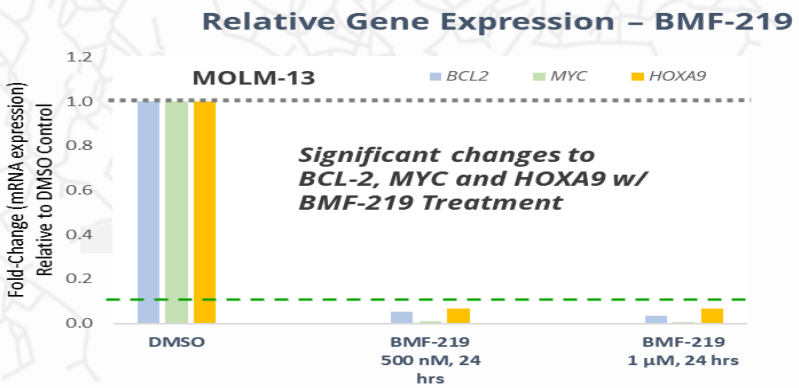
- MYC is a major downstream effector of the MAPK pathway in KRAS-activated tumors
- BMF-219 robustly decreases MYC gene expression and genomic function and drives cell killing in numerous MYC driven ex-vivo tumor samples.

MOA

BMF-219 inhibits the menin/ MYC interaction and downregulates expression of MYC and MYC target genes, including KRAS

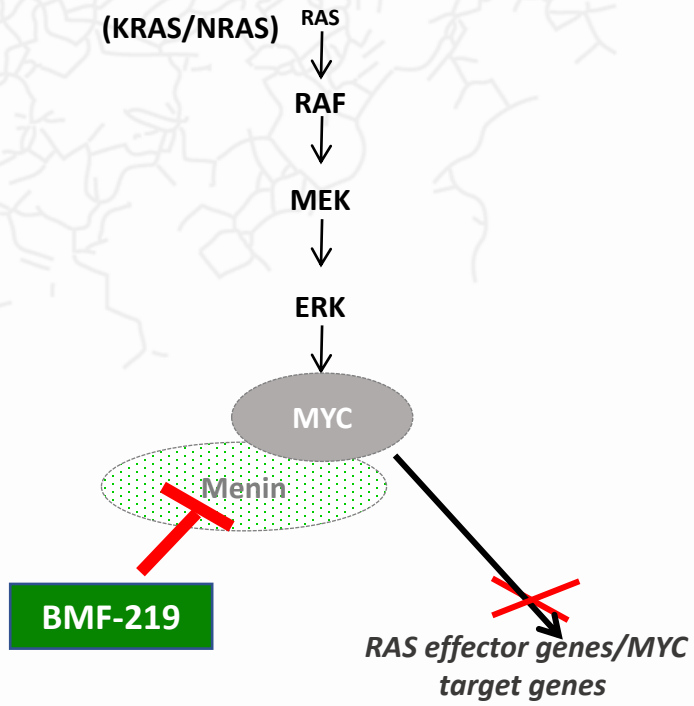
(Blood (2021) 138 (Supple. 1): 4318.)





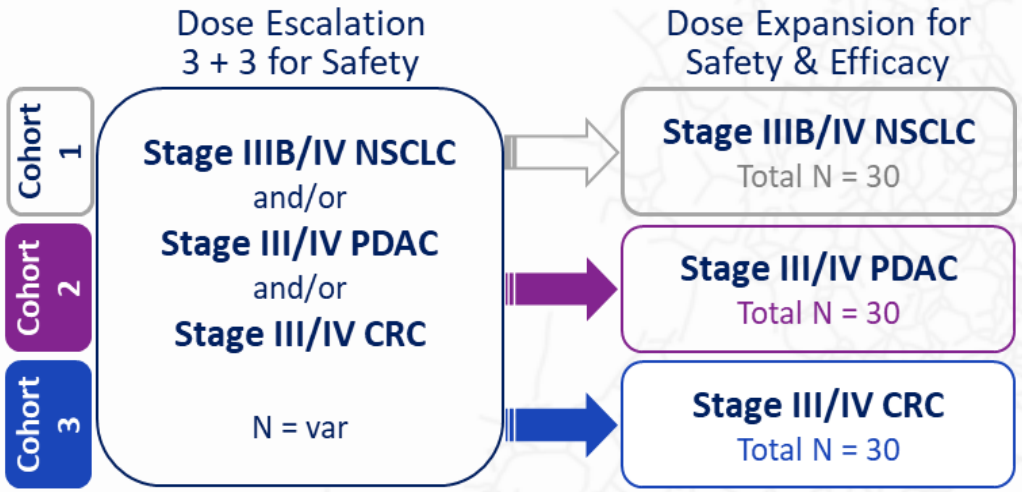
Relevant Pathway

Tumor leverages MAPK pathway

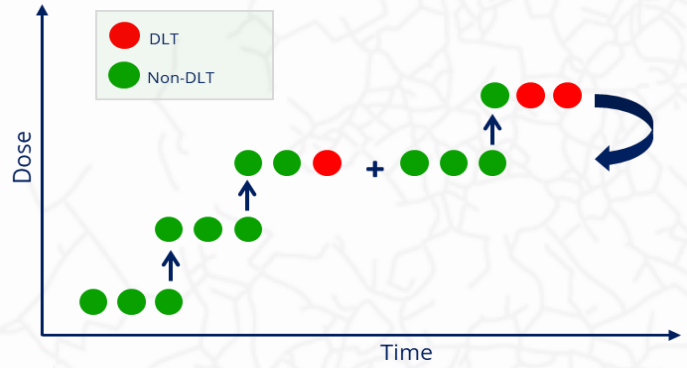


COVALENT-102 (SITE ACTIVATION)

Phase I/Ib Study of BMF-219, an oral covalent menin inhibitor, in patients with KRAS Mutant, Unresectable, Locally Advanced, or Metastatic Non-Small Cell Lung Cancer (NSCLC), Pancreatic Cancer (PDAC), and Colorectal Carcinoma (CRC)



Classical 3+3 dose escalation design



Study Treatment: BMF-219

- A covalent small molecule menin inhibitor, administered orally daily in 28 day cycles

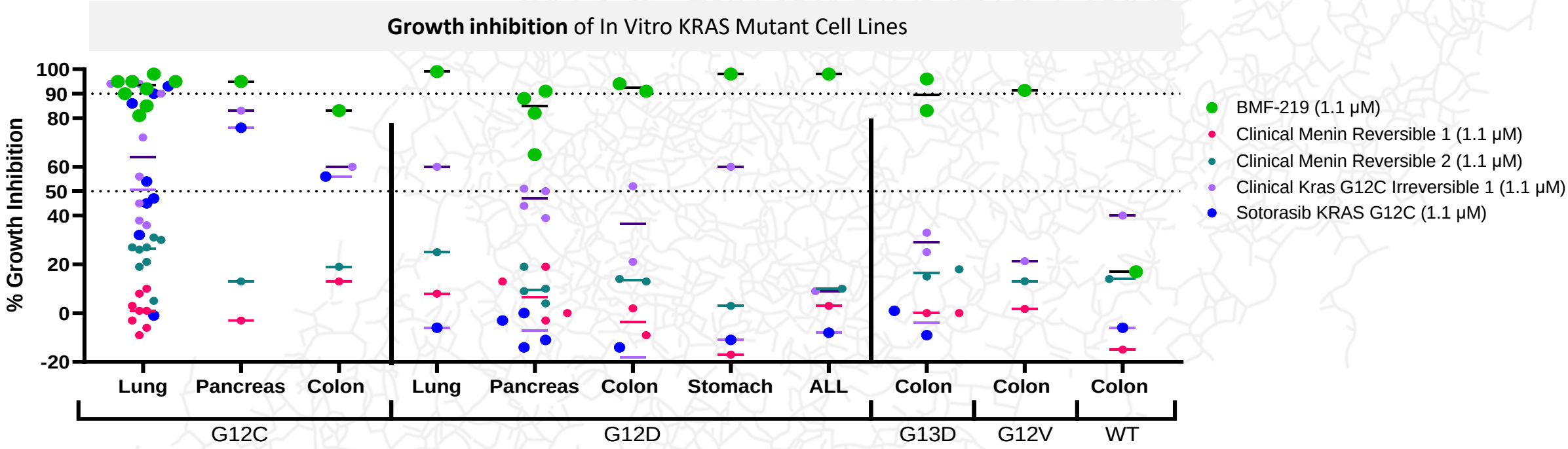
Objectives

- Primary:
  - Determine OBD & RP2D of BMF-219 monotherapy independently for each Cohort / Indication
- Secondary:
  - Evaluate safety and tolerability of BMF-219
  - Determine PK/ PD parameters of BMF-219
  - Explore additional evidence of efficacy and antitumor activity

Abbreviations: NSCLC Non-Small Cell Lung Cancer PDAC Pancreatic Cancer CRC Colorectal Carcinoma OBD optimal biologic dose RP2D recommended phase 2 dose PK/PD pharmacokinetic/pharmacodynamic ECOG Eastern Cooperative Oncology Group var variable L prior line of systemic therapy

First Development Success with BMF-219 in RAS/RAV Driven Solid Tumors

BMF-219 Produced Near Complete Inhibition of Growth at 1.1µM Across KRAS G12C, G12D, G13D, and G12V Mutant Cell Lines but not WT KRAS

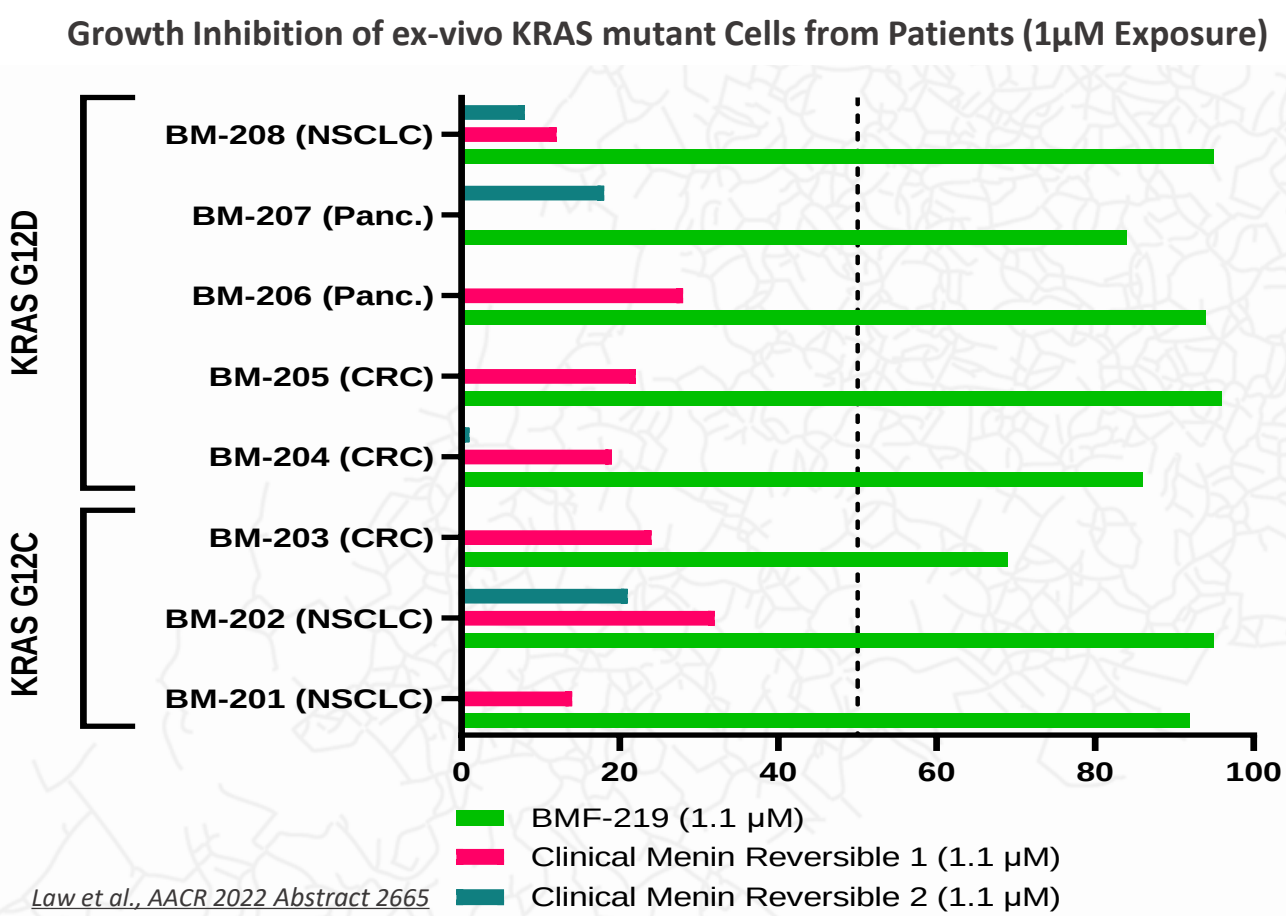


- Covalent menin inhibition by BMF-219 led to robust growth inhibition, comparable to clinical G12C inhibitors in G12C cell lines
- In non-G12C cell lines, BMF-219 achieved robust growth inhibition, higher than clinical KRAS G12C inhibitors
- Clinical reversible (non-covalent) inhibitors did not achieve greater than 30% growth inhibition in any cell lines at the concentrations tested

Law et al., AACR 2022 Abstract 2665

First Development Success with BMF-219 in RAS/RAV Driven Solid Tumors

BMF-219 Produced Near Complete Inhibition of Growth at 1.1µM in KRAS G12C and G12D ex-vivo Samples



Law et al., AACR 2022 Abstract 2665

- 1.1µM exposure of BMF-219 produces robust growth inhibition in both G12C and G12D ex-vivo cell lines
- BMF-219 responses were superior to clinical reversible (non-covalent) inhibitors with respect to cell growth inhibition at the concentrations tested

Novel Covalent Inhibitor of Menin

BMF-219

Pipeline-in-a-Pill – Single Agent for Multiple Indications

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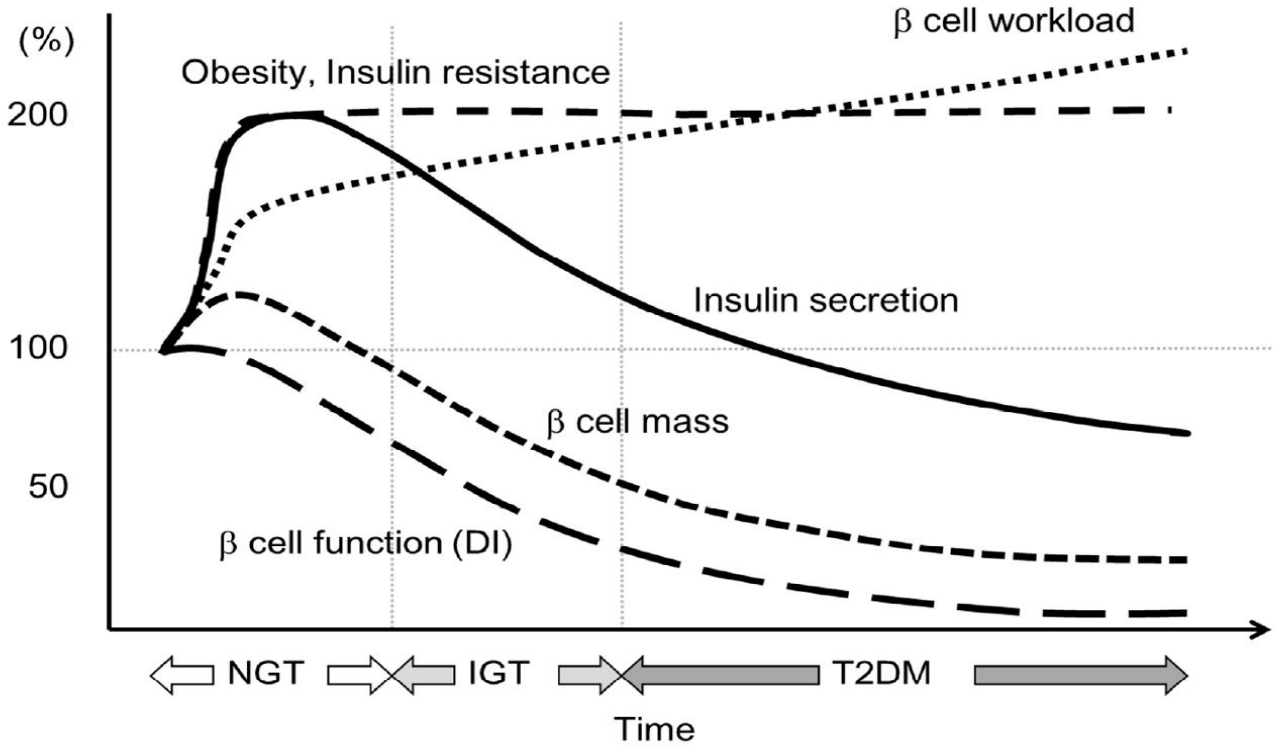
Further expansion into KRAS and RAS mutant colorectal, lung, and pancreatic cancer

Diabetes

Pathway and clinical validation of covalent menin inhibition

First Development Success with BMF-219 in Type II Diabetes

Diabetes Progression of Type 1 and Type 2 Driven by Beta Cell Loss



Prior Paradigm	Type 1 diabetes	Type 2 diabetes
	β cell destruction β cell mass ↓↓ Insulin secretion ↓↓	Obesity Insulin resistance Hyperinsulinemia
Current Paradigm	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

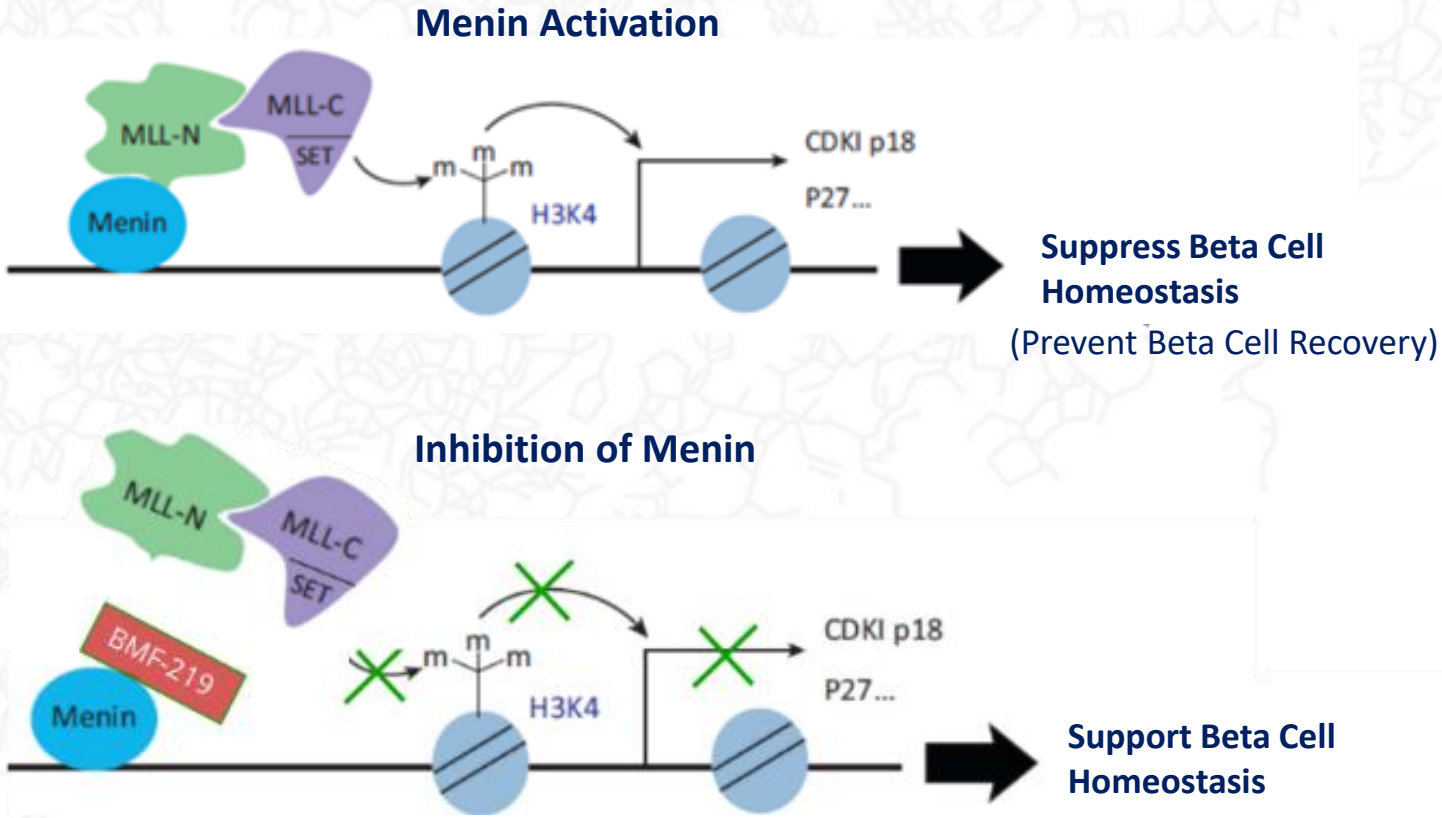
Insulin Resistance leads to an increase in Beta Cell Workload which ultimately leads to Beta Cell Failure and Death and the Progression of Type 2 Diabetes.

Type 1 and Type 2 Diabetes results in Beta Cell Loss and Reduction in Beta Cell Mass

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.



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

COVALENT-111 (ENROLLING)

A Phase 1/2 Randomized, Double-Blind, Placebo-Controlled Single and Multiple Ascending Dose Study to Evaluate the Safety, Tolerability, Pharmacokinetics, and Pharmacodynamics of BMF-219, an Oral Covalent Menin Inhibitor, in Healthy Adult Subjects and in Adult Subjects with Type 2 Diabetes Mellitus

Phase 1 (SAD)

SAD C1 to SAD C4 (HVs)
Total N=40
Dose [100, 200, 400, and 600 mg]

Phase 2 (MAD)

MAD C1 (HVs)
Total N = 16

MAD C2 to MAD C8 (T2D)
Total N=108

Dose [100, 200, 300, 400, 600 mg]

In the Phase 2, COVALENT-111 will enroll subjects with a HbA1C of 7-10% despite being on standard of care, up to three agents of therapy.

Study Treatment: BMF-219

- A covalent small molecule menin inhibitor, administered orally daily in 28 day cycles

Primary Objective:

- Evaluate safety and tolerability of BMF219

Secondary Objectives:

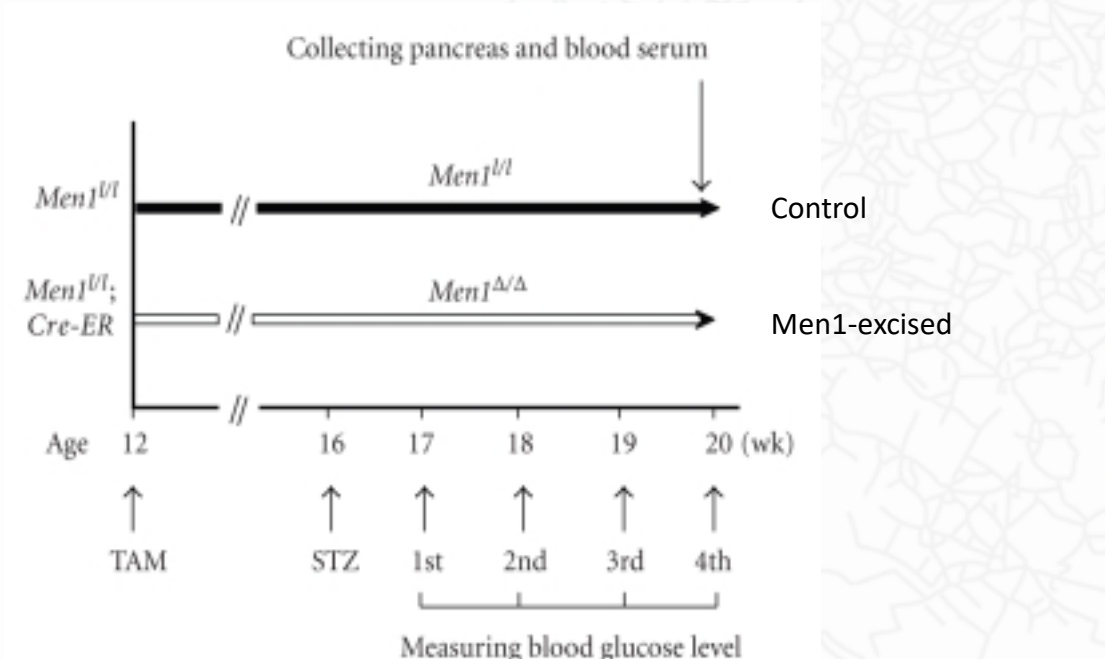
- Evaluate PK of BMF-219
- Evaluate the effect on BMF-219 on glycemic parameters (HbA1C, PG) and few additional parameters using OGTT, 7-day CGM
- Evaluate the changes in beta cell function
- Evaluate impact on lipid parameters, body weight etc.

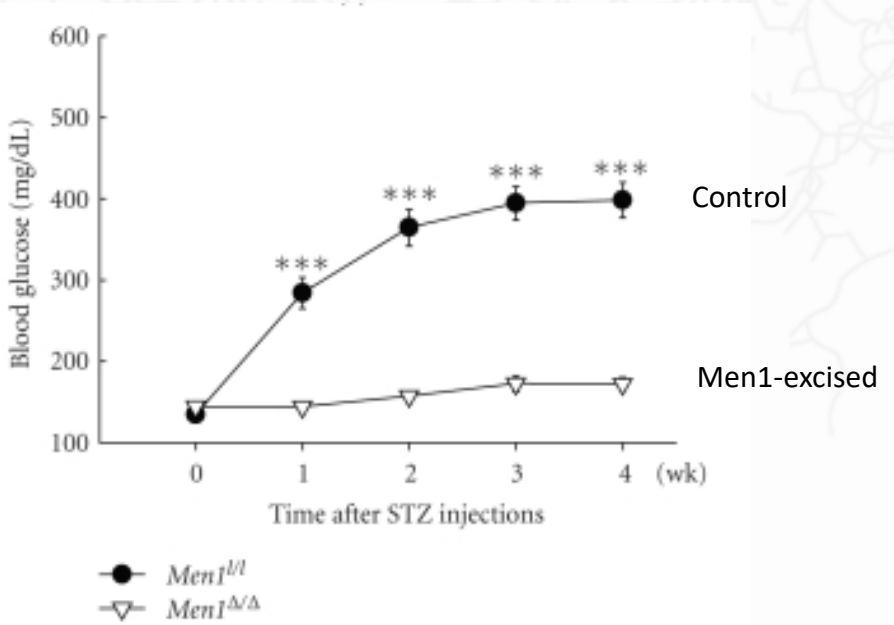
Exploratory Objectives:

- To assess the durability of response to glycemic parameters

Potential for Menin Inhibition Demonstrated by Beta Cell Ablation Diabetes Model in MEN1 Excised Mice

MEN1 Excision Prevents Development of STZ-induced Hyperglycemia





Multiple low-dose streptozotocin (MLD-STZ) administered to the control and *Men1*-excised mice to induce beta cell damage and a diabetes-like environment

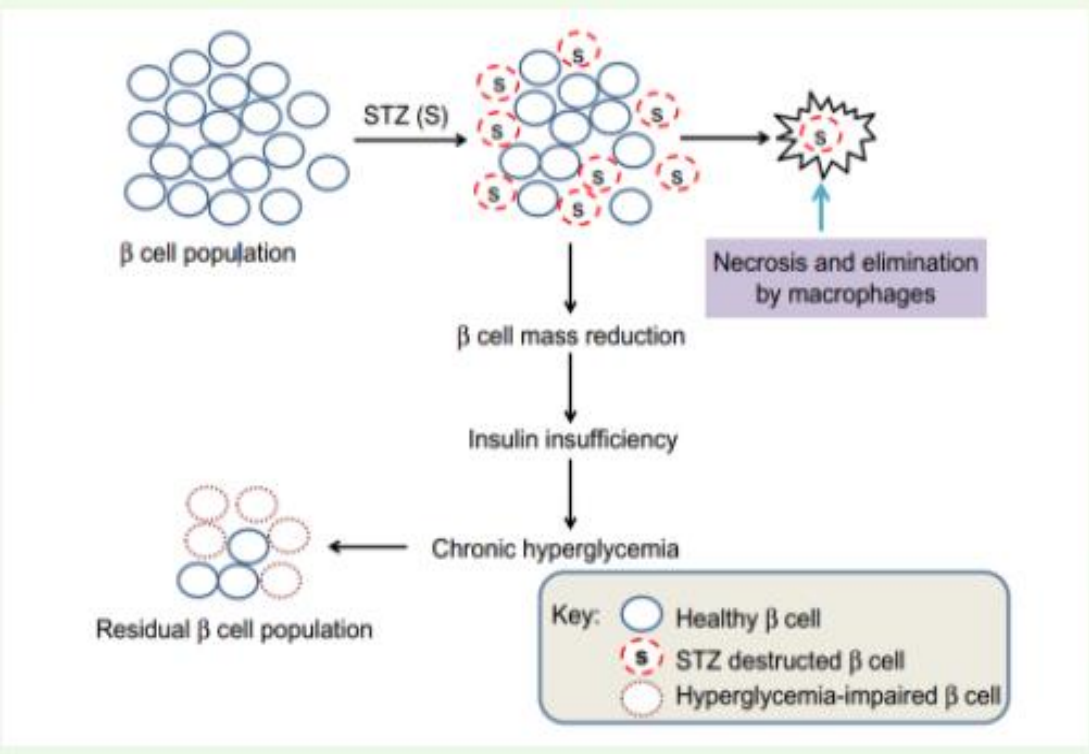
***Men1*-excised mice did not develop hyperglycemia in STZ model, which was observed in the control group**

Sources: Yang et al. (2010) Deletion of theMen1Gene Prevents Streptozotocin-Induced Hyperglycemia in Mice. Experimental Diabetes Research, 2010, 1–11. doi:10.1155/2010/876701

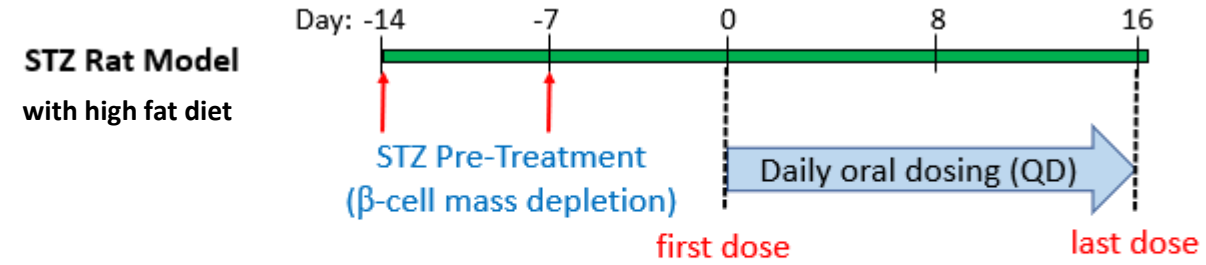
STZ Rat Model Study Design

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

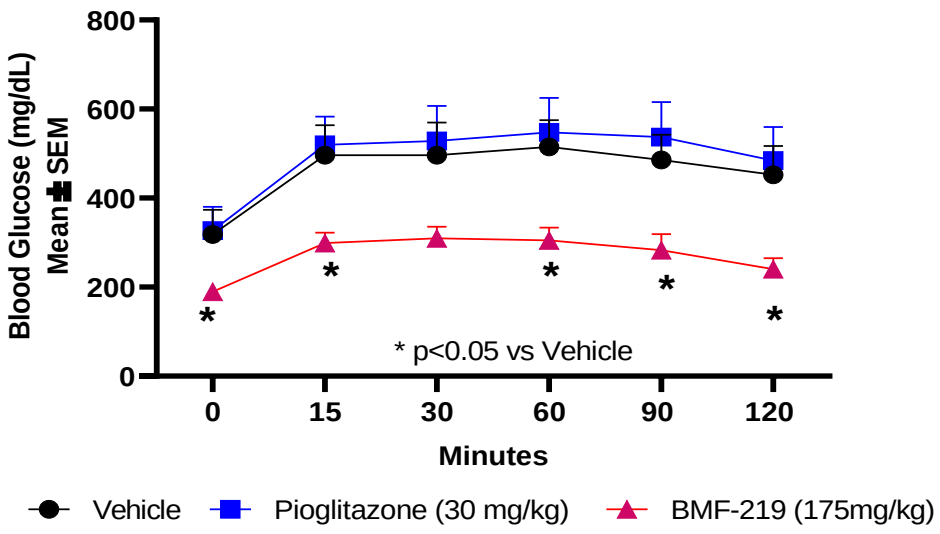
STZ treatment typically results in ~50% Beta Cell Loss

First Development Success with BMF-219 in Type II Diabetes

BMF-219 Demonstrates Strong Efficacy in Beta Cell Loss Animal Model (STZ Rat)

BMF-219 Achieves Glycemic Control in STZ (Beta Cell Loss) Rat Model

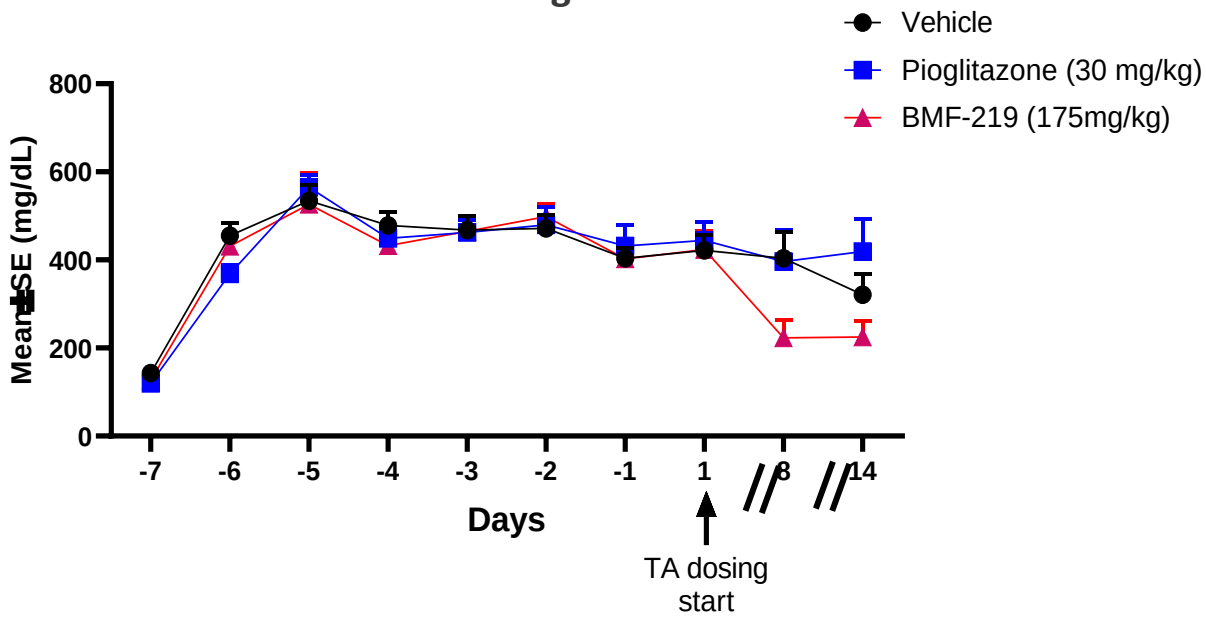
Oral Glucose Tolerance Test (Day 17)



BMF-219 achieves lower glucose level than pioglitazone at all timepoints in OGTT (day 17) in STZ rat model

Butler et al., ADA 2022 (P-851)

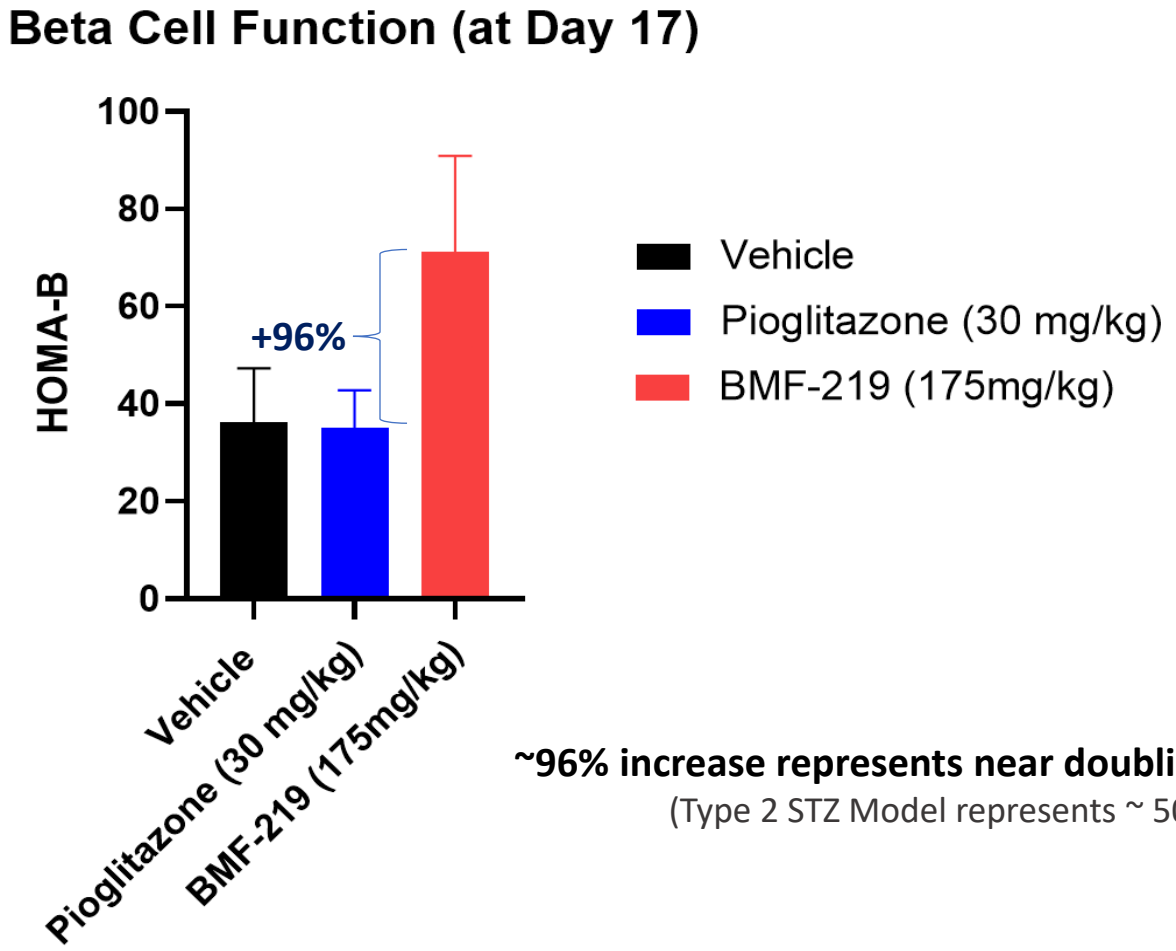
Non-Fasting Glucose



BMF-219 achieves lower non-fasting glucose than pioglitazone at day 8 and day 14 in STZ rat model

First Development Success with BMF-219 in Type II Diabetes

BMF-219 Demonstrates Recovery of Beta Cell Activity



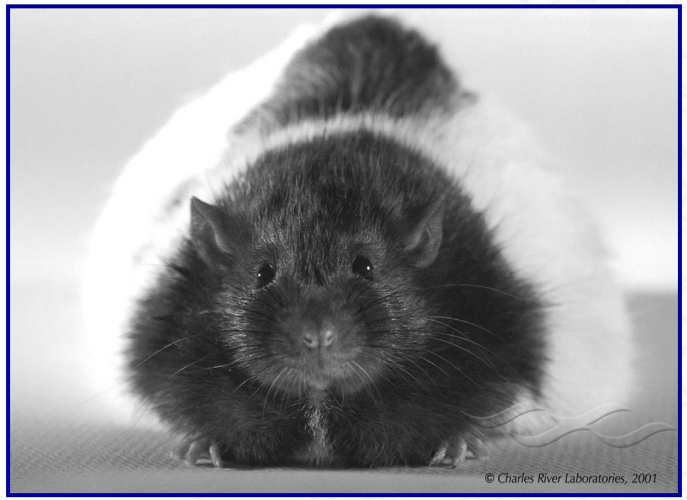
- HOMA-Beta, a measurement of Beta Cell Function, was analyzed using 4-hr fasting glucose and insulin levels in animal plasma.
- BMF-219 displayed a significant level of Beta Cell function compared to vehicle at Day 17 in a Beta Cell Type 2 Diabetes Model.
- This data supports the observed results from 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)

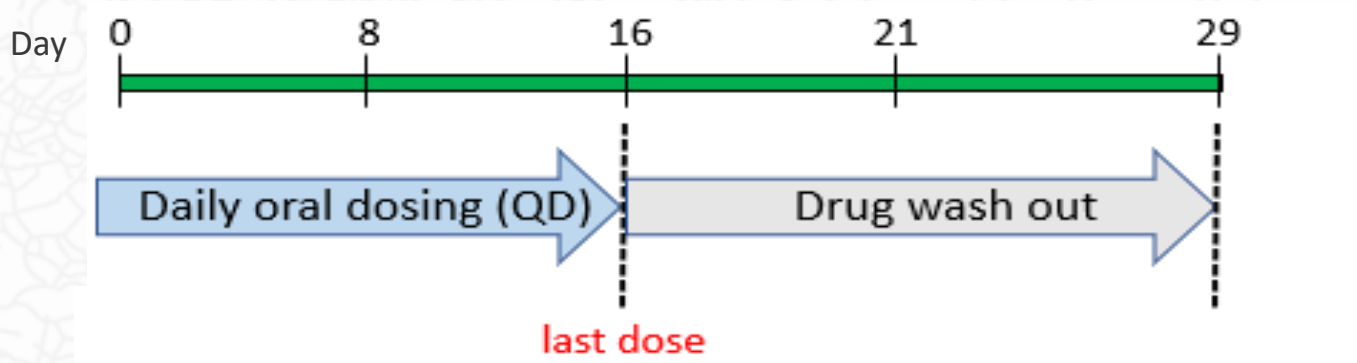
Zucker Diabetic Fatty Rat - a Model of Insulin Resistance

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

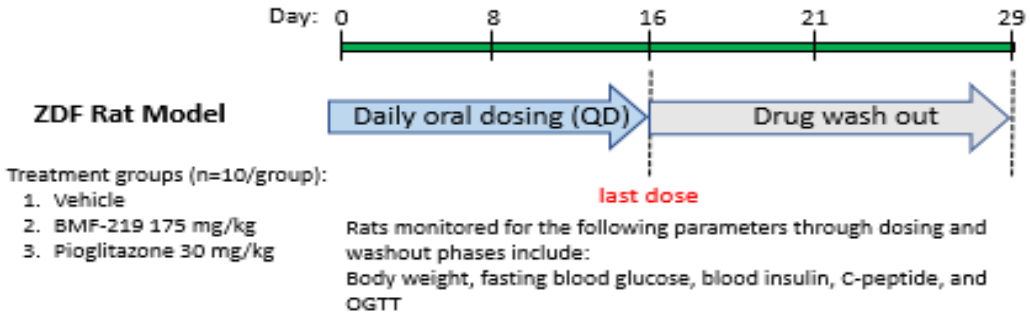
Treatment groups (n = 10/group):

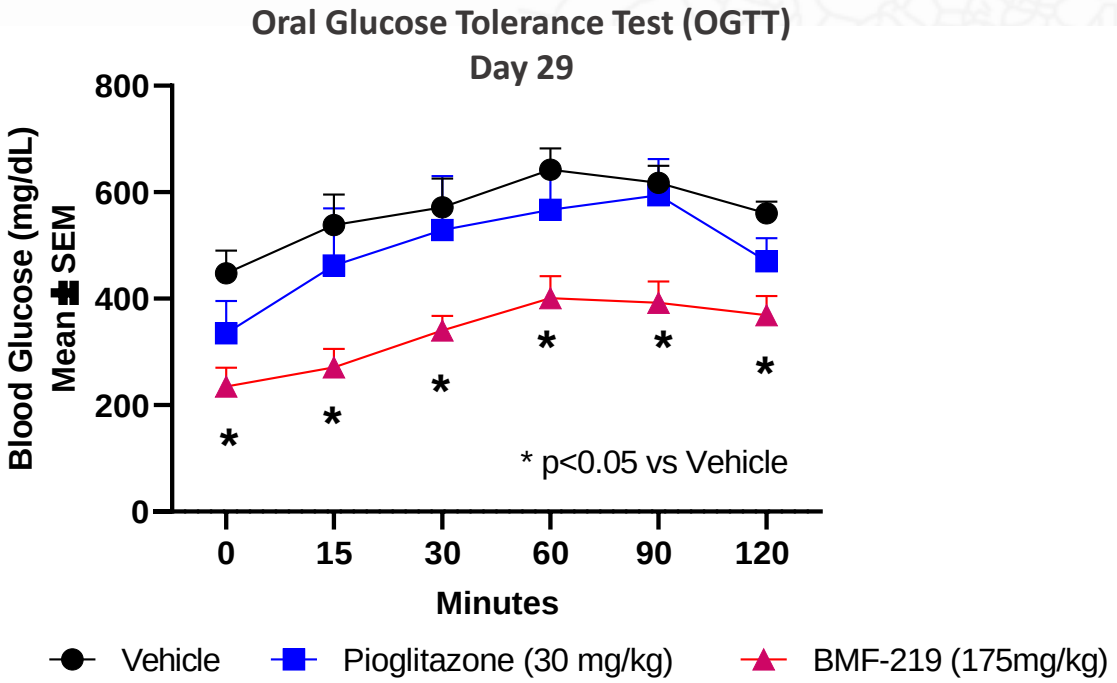
1. Vehicle
2. BMF-219 175 mg/kg
3. Pioglitazone 30 m g/kg

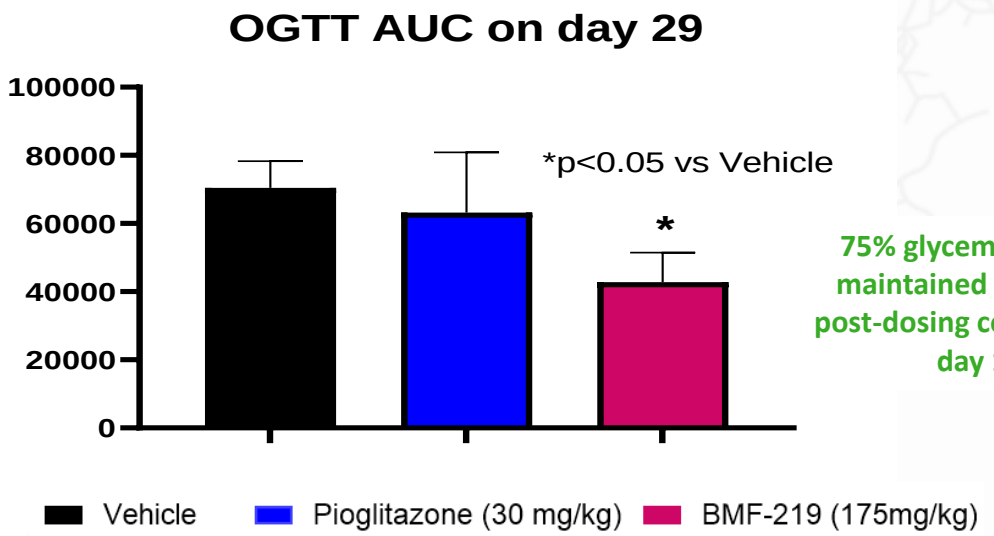
First Development Success with BMF-219 in Type II Diabetes

BMF-219 Displays Durable Glycemic Control during Drug Washout and Two Weeks after the Last Dose

After 2-week Drug Washout





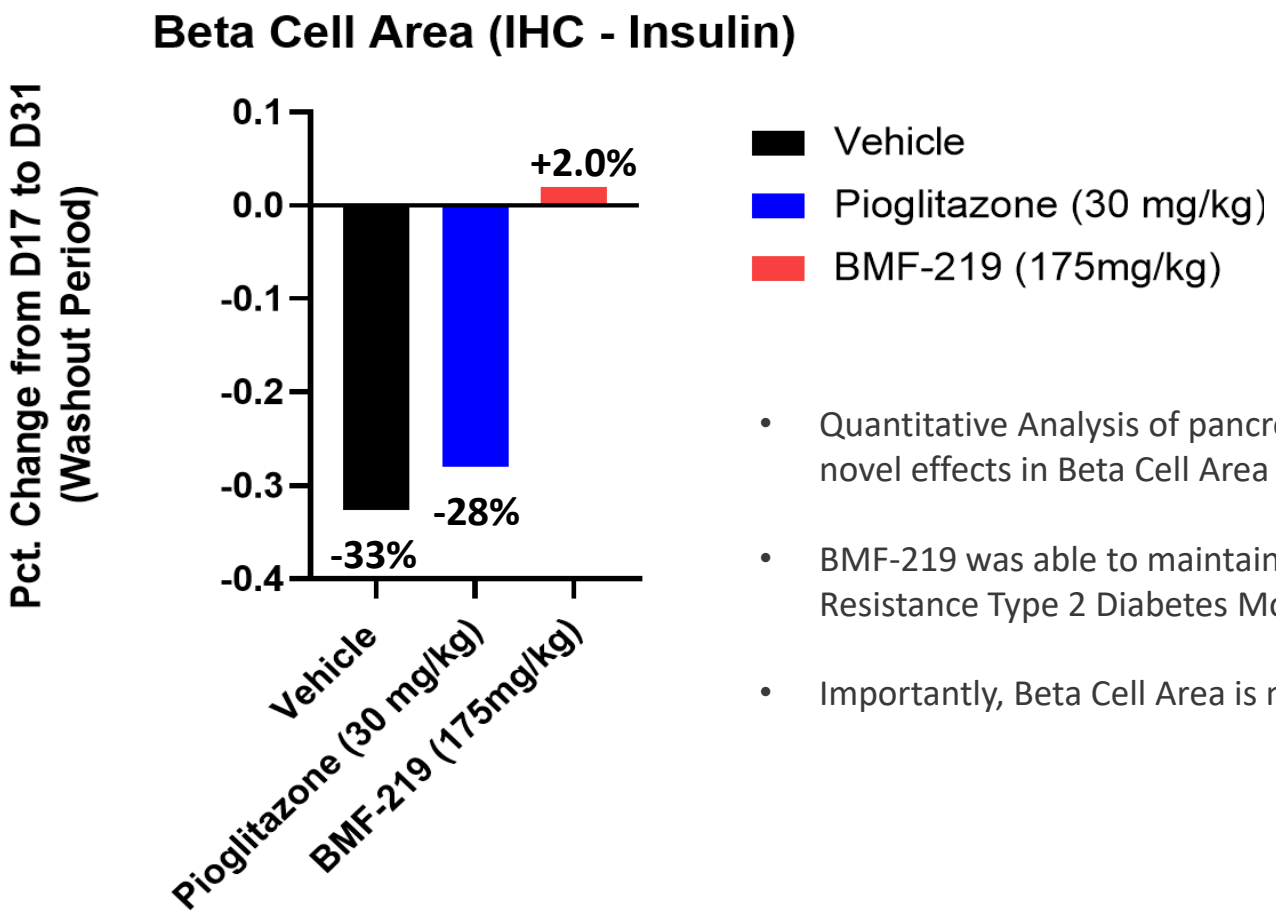


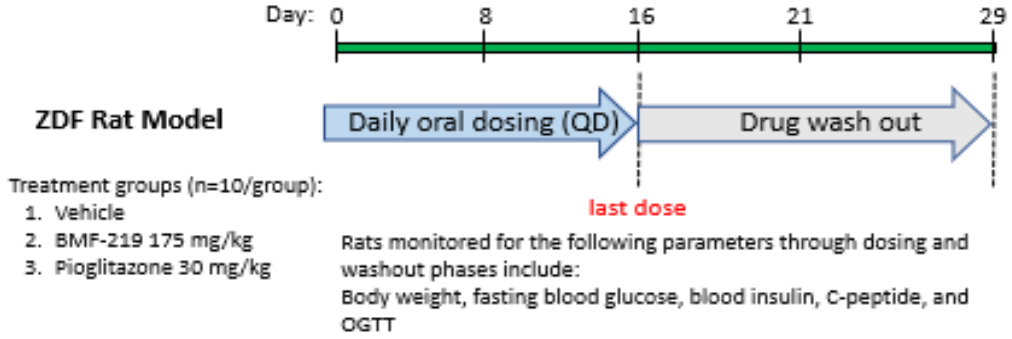
75% glycemic control maintained on day 29 post-dosing compared to day 15

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%, ($p<0.05$).

First Development Success with BMF-219 in Type II Diabetes

BMF-219 Increases B-islets in Pancreas Sections of ZDF Diabetic Model

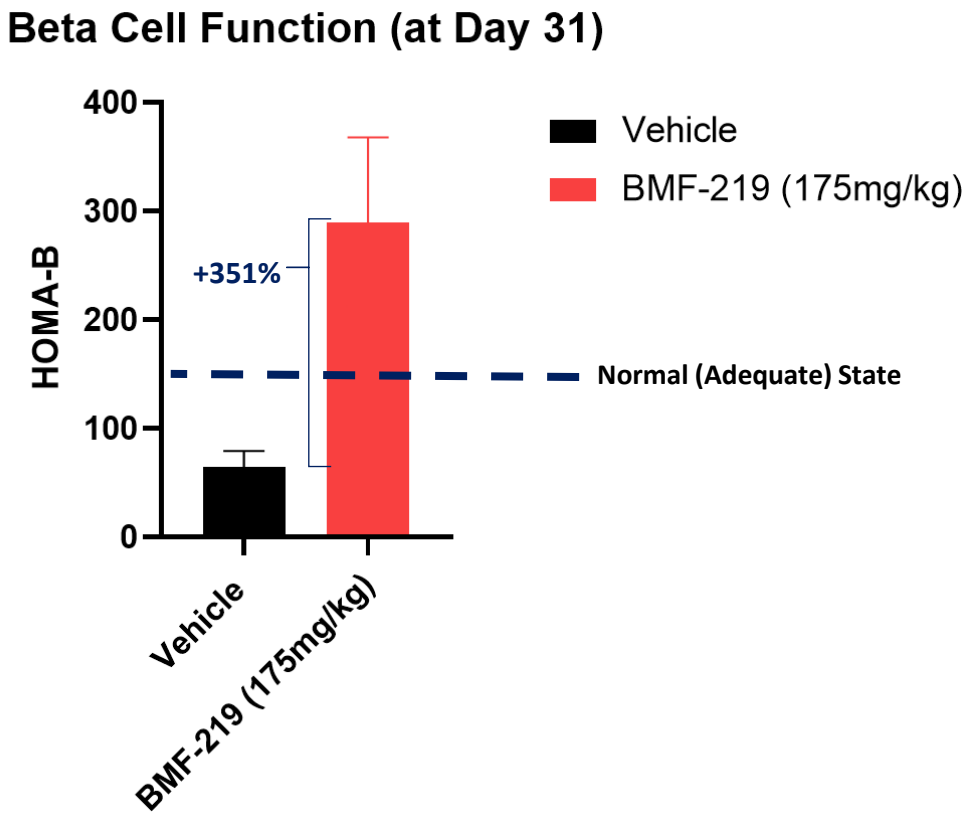




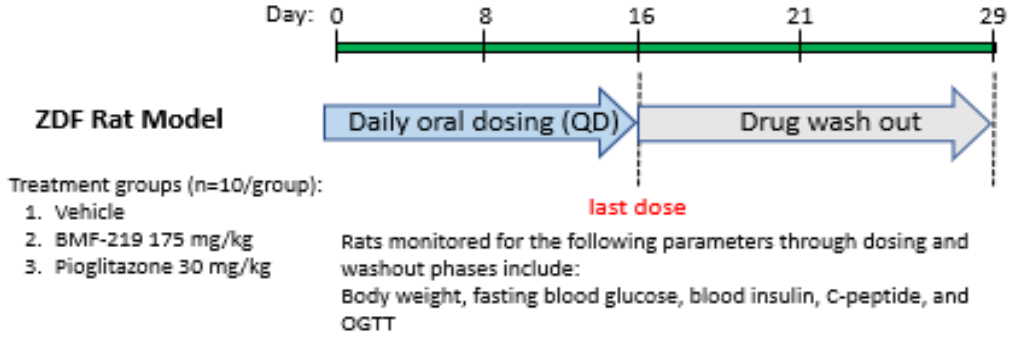
- Quantitative Analysis of pancreatic islet tissue cross sections shows BMF-219 treated animals show novel effects in Beta Cell Area growth and maintenance.
- BMF-219 was able to maintain Beta Cell function and prevent Beta Cell Area Loss in an Insulin Resistance Type 2 Diabetes Model.
- Importantly, Beta Cell Area is maintained, despite cessation of dosing.

First Development Success with BMF-219 in Type II Diabetes

BMF-219 Demonstrates Strong B-cell Activity - Supporting Quantitative Analysis



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

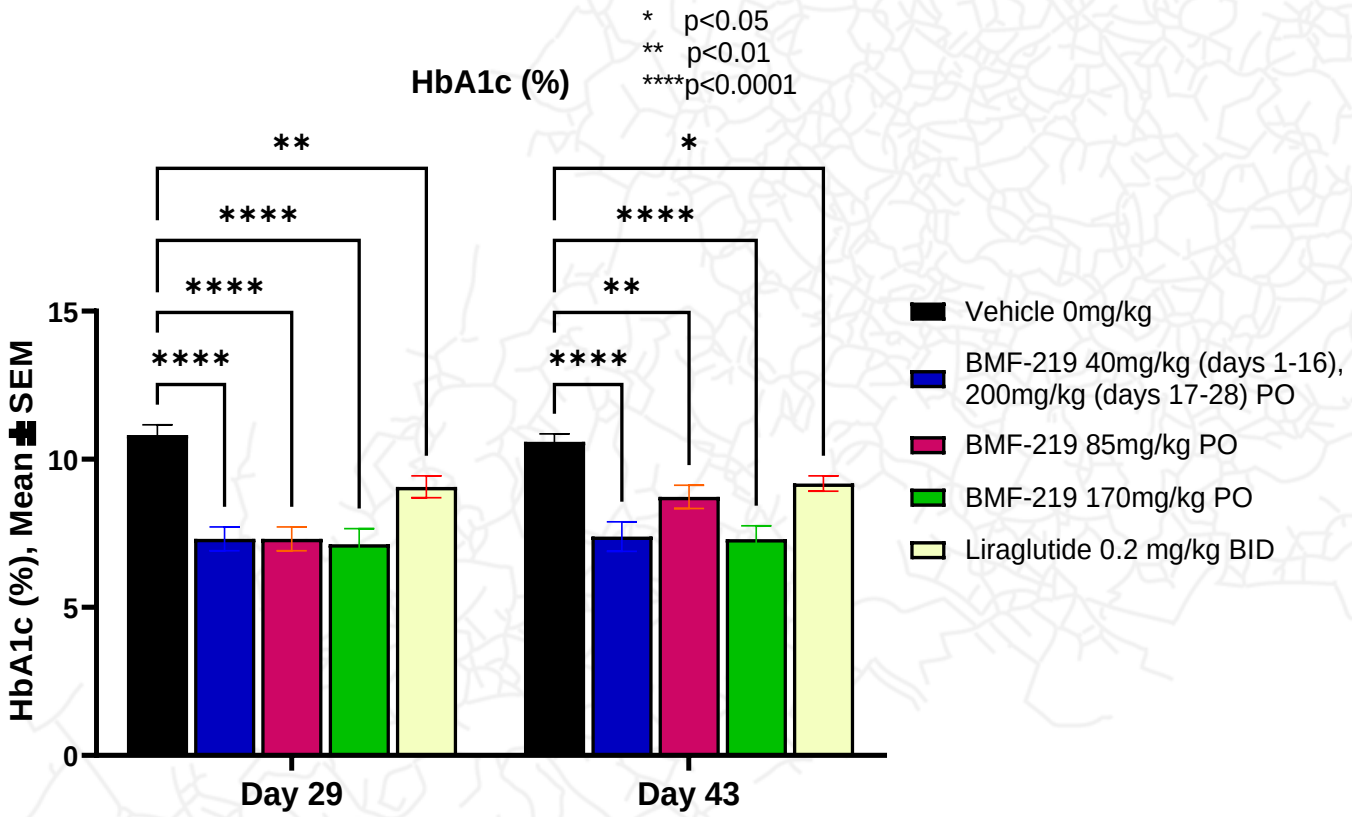


- HOMA-Beta, a measurement of Beta Cell Function, was analyzed using 4-hr fasting glucose and insulin levels in animal plasma.
- BMF-219 displayed a significant level of Beta Cell function compared to vehicle at Day 31 in an Insulin Resistance Type 2 Diabetes Model.
- This data supports the observed results from the Beta Cell Area Quantitative Analysis using IHC. Importantly, Beta Cell Function is observed despite cessation of dosing.

First Development Success with BMF-219 in Type II Diabetes

BMF-219 Demonstrates Strong Efficacy in Insulin Resistant Animal Model (ZDF Rat)

BMF-219 Reduces HbA1c After 28 days of Treatment and Maintains Effect After 14-day Washout



Somanath et al., ADA 2022 (113-LB)

BMF-219 demonstrated significant decrease in HbA1c (-3.5% at day 29) vs. control starting on day 21 of treatment

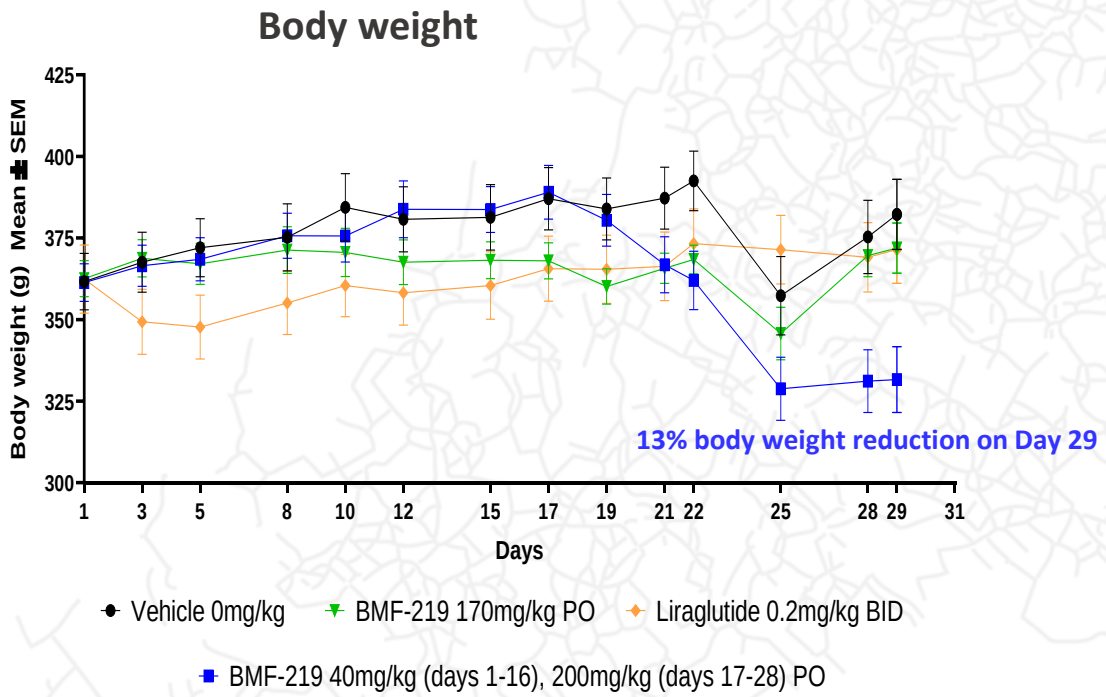
BMF-219 treated group demonstrated significant weight reduction starting at day 25

HbA1c reduction in BMF-219 highest dose groups maintained through washout period

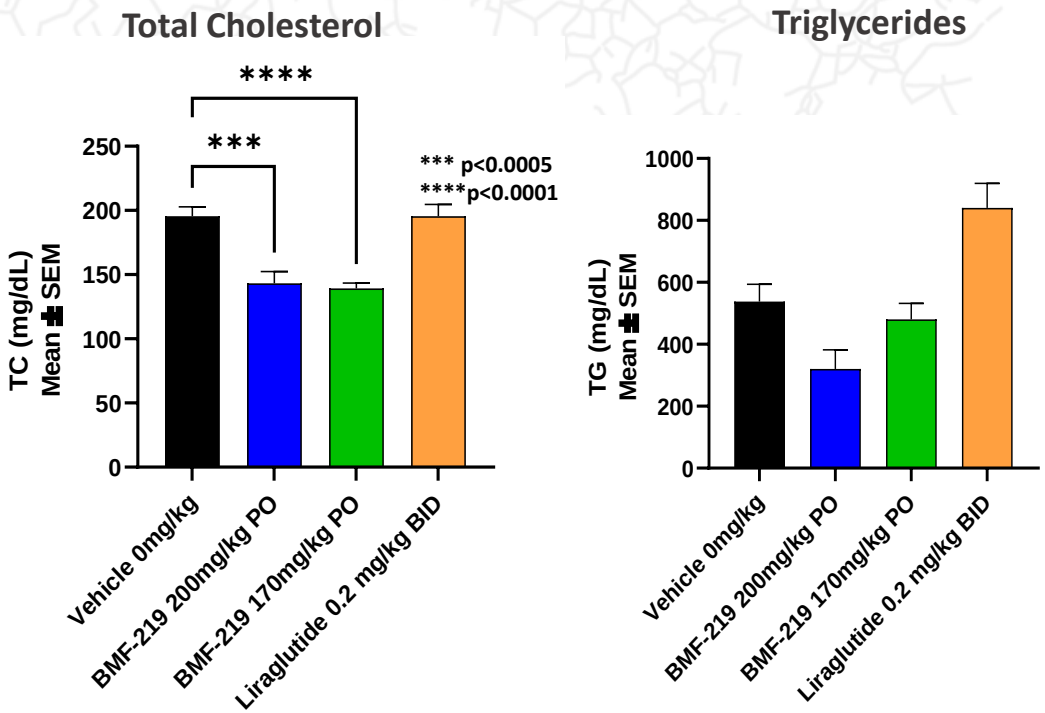
First Development Success with BMF-219 in Type II Diabetes

BMF-219 Treated Groups Display Body Weight and Cholesterol Reduction

BMF-219 200 mg/kg group reduces body weight during treatment in ZDF rats

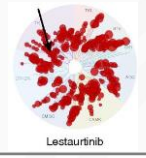
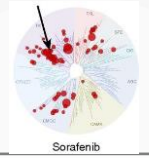
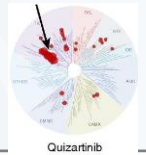
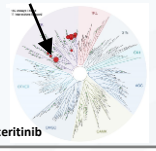
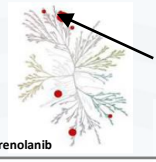


BMF-219 reduces blood lipemic levels measured on Day 29



Second Development Success with BMF-500

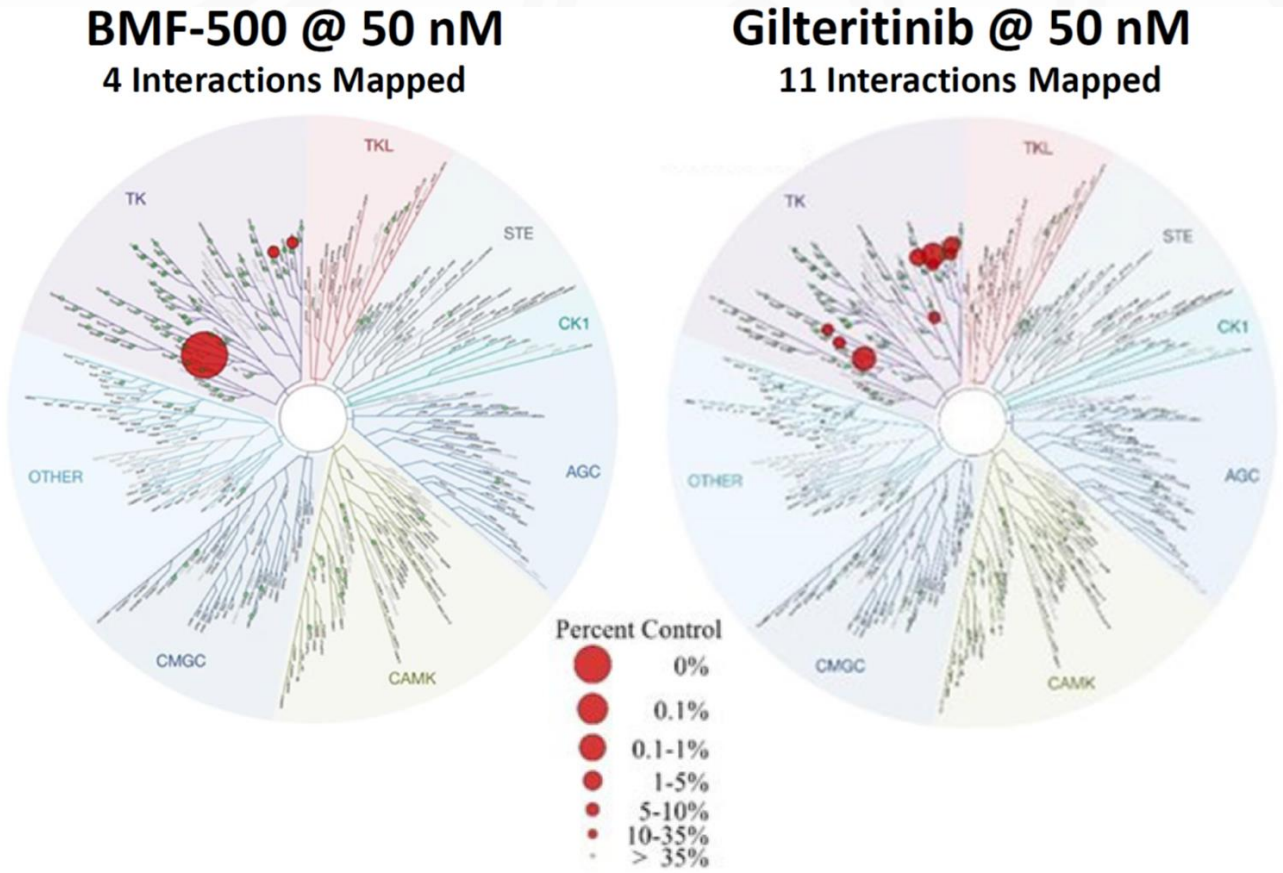
BMF-500 A Third Generation FLT3 Inhibitor

Generation of FLT3 Inhibitor	First Generation FLT3 / multi-kinase Inhibitors			Second Generation FLT3 Inhibitors			Third Generation FLT3 Inhibitors
Products	**Midostaurin** *(FDA Approved as RYDAPT)*	**Lestaurtinib** *(Failed in clinical trials)*	**Sorafenib** *(FDA Approved as NEXAVAR)*	**Quizartinib** *(FDA Rejected due to Cardiotox)*	**Gilteritinib** *(FDA Approved as XOSPATA)*	**Crenolanib** *(Phase 3 in US)*	**BMF-500** *(Covalent Inhibitor, Preclinical)*
Benefits	• *In vitro* potency against FLT3 • Oral route of administration			• More selective for FLT3	• Improved PK properties	• Improved potency D835 • Reduced KIT inhibition	• Drives cell death • Improved FLT3 potency and selectivity • Improved activity in known resistance mechanisms
Challenges	• Poor kinase selectivity • Challenging pharmacokinetic (PK) profile • Low steady state free drug concentration • Low potency resulting from challenging PK at tolerable doses			• Adverse Events • QTc impact • Cytopenia	• Drives Differentiation • Myelo-suppression • Frequent Dose Adj • QTc impact	• TID Dosing • F619 Resistance • Drives Differentiation	• Limited history of covalent FLT3 experience in the clinic • Novel scaffold with emerging profile
Kinome Selectivity	 Midostaurin	 Lestaurtinib	 Sorafenib	 Quizartinib	 Gilteritinib	 Crenolanib	 **BMF-500**

Sources: Levis M. (2017). Midostaurin approved for FLT3-mutated AML. *Blood*, *129*(26), 3403–3406. https://doi.org/10.1182/blood-2017-05-782292; Drugs@FDA.gov

Second Development Success with BMF-500

BMF-500 Highly Selective to FLT3



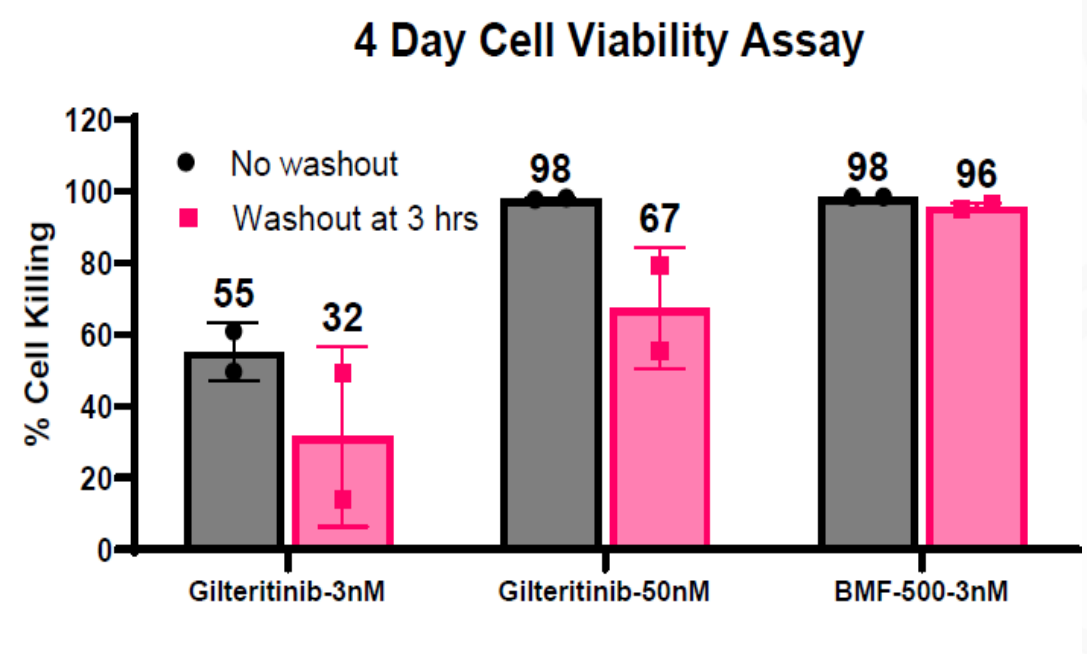
5-Day Cytotoxicity Profile (IC_{50}, uM)

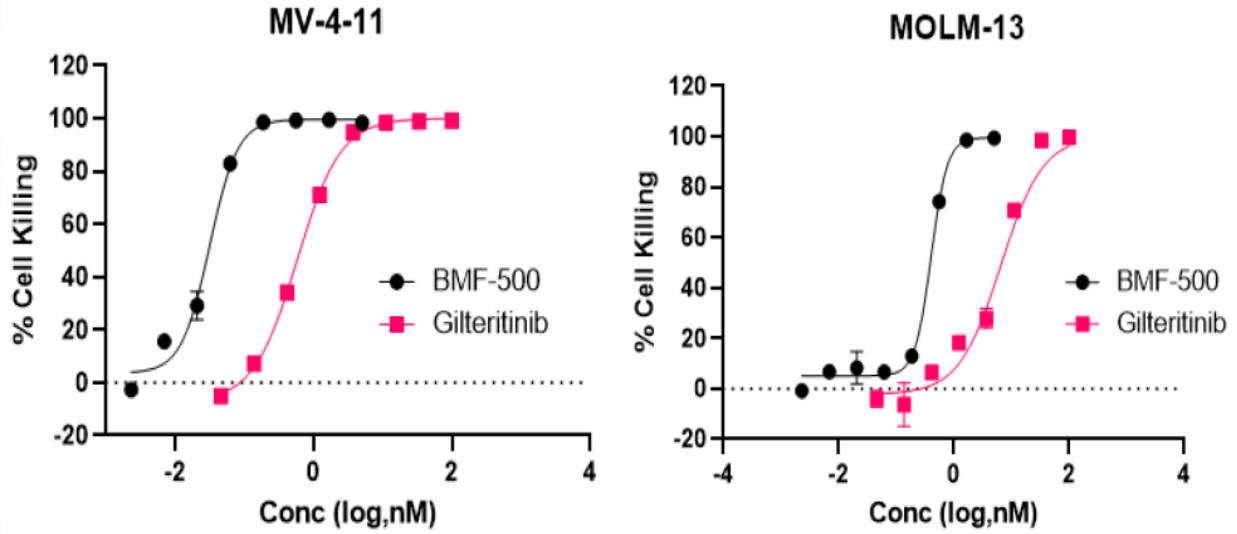
Cell Line	Tumor Type	BMF-500	Gilteritinib
MCF7	Adenocarcinoma	>1	>1
MV-4-11	Leukemia (acute myelomonocytic	**<0.001**	**0.003**
RS4;11	Leukemia (acute lymphoblastic)	>1	0.233
SaOS2	Osteosarcoma	>1	0.236
SK-N-AS	Neuroblastoma	>1	>1
SKOV3	Adenocarcinoma	>1	0.804
Thp1	Leukemia (acute monocytic)	>1	>1
WiDr	Colorectal adenocarcinoma	>1	0.268
CCRFCEM	Leukemia (acute lymphoblastic)	>1	>1
RL95-2	Carcinoma	>1	0.868

Cell Line	Tumor Type	BMF-500	Gilteritinib
SW684	Fibrosarcoma	>1	>1
A549	NSCLC	>1	0.278
BV-173	Leukemia (CML)	>1	0.740
CGTH-W-1	Carcinoma, metastatic	>1	0.455
Daudi	Burkitt's lymphoma	>1	>1
HCT-116	Carcinoma	>1	>1
Jurkat	Acute T-cell leukemia	>1	0.947
HL-60	Leukemia, acute promyelocytic	>1	0.445
LS411N	Carcinoma, Duke's type B	>1	>1
MOLT-4	Leukemia (ALL)	>1	>1

Second Development Success with BMF-500

BMF-500 Highly Effective FLT3 Inhibitor even after Drug Wash-Out

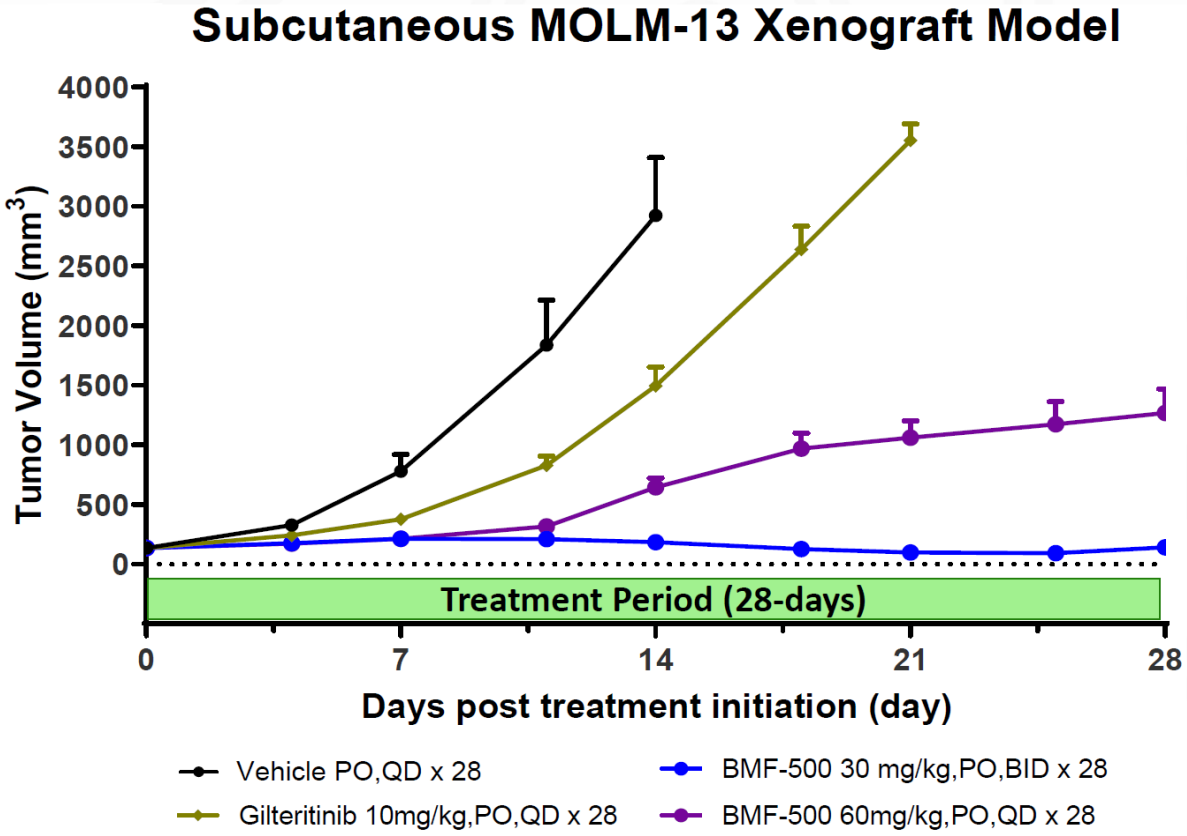


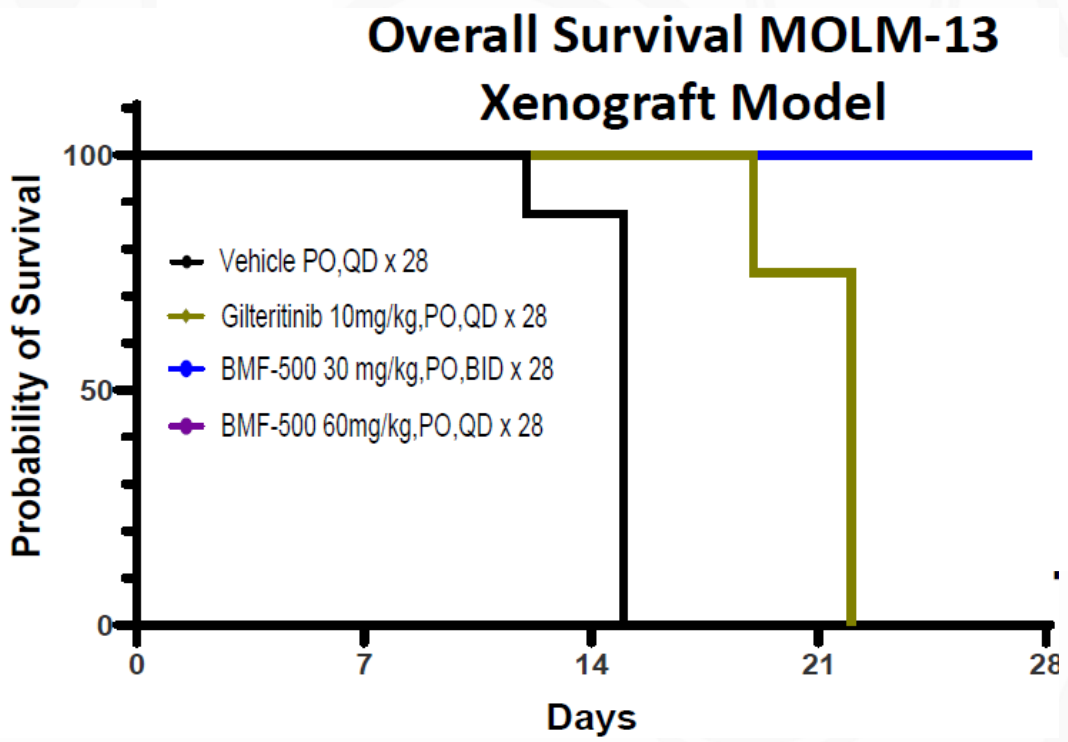


Compound ID	MV-4-11 IC_{50} (nM)	MOLM-13 IC_{50} (nM)
BMF-500	0.03	0.30
Gilteritinib	1.7	6.5

Second Development Success with BMF-500

BMF-500 Highly Potent and Durable FLT3 Inhibitor





Exploring 8 Different Tumor Types and Type II Diabetes in the Clinic

- Established FUSION™ platform technology for discovery of covalent inhibitors
- Lead molecule (BMF-219) with best-in-class potential and favorable safety profile
- Initiate studies with BMF-219 in up to 8 tumor types (liquid and solid) in 2022
- Significant addressable market opportunities for clinically targeted tumor types
- Initiate study with BMF-219 in Type 2 Diabetes in 2022
- Announced second pipeline asset: BMF-500, pM potent covalent FLT3 inhibitor
- Cash as of 30 Sept 2022 $133.8M - Capitalized into 2024

Multiple Clinical Read Outs over the coming Quarters

Near Term Milestones – Biomea Fusion (NASDAQ: BMEA)

			Key Milestones	Expected Timeline
BMF-219 Menin Programs	**COVALENT-101** (Liquid Tumors)	AML/ALL (Leukemia)	Phase I: Clinical Data in AML	1H 2023
		DLBCL (Lymphoma)	Initiation of Phase I additional Cohort in DLBCL	In Progress
		MM (Lymphoma)	Enrolling in Phase I additional Cohort in MM	In Progress
		CLL (Lymphoma)	Enrolling in Phase I additional Cohort in CLL	In Progress
	COVALENT-102 (KRAS Solid Tumors)	NSCL (Lung)	Initiation of Phase I KRAS Study in CRC, PDAC, NSCLC	In Progress
		PDAC (Pancreas)		
		CRC (Colon)		
	COVALENT-111 (Diabetes)	Type 2 Diabetes	Healthy Volunteers of PI/II COVALENT-111 Trial Phase II: Clinical Data in Type 2 Diabetes	Completed 1H 2023
BMF-500 FLT3 Programs	**COVALENT-103** (Liquid Tumors)	AML/ALL (leukemia)	Preclinical Data Presentation IND Filing	ASH 2022 1H 2023
Additional Oncology Programs	**Target # 3**	Oncology	Progress Update	2H 2023

As of September 30, 2022

Company Financials (NASDAQ: BMEA)

	Three Months Ended Sept 30	
	2022	2021
Operating expenses:		
R&D	$ 18,242	$ 7,886
G&A	$ 5,242	$ 4,752
Total Operating Expenses	$ 23,484	$ 12,638
Loss from operations	$ (23,484)	$ (12,638)
Interest and other income, net	$ 594	$ 32
Net loss	$ (22,890)	$ (12,606)
Other comprehensive loss:		
Changes in unrealized gain on short term investments, net	$ 4	-
Comprehensive loss	$ (22,886)	$ (12,606)
Net loss per common share, basic and diluted	$ (0.78)	$ (0.43)
Weighted-average number of common shares used to compute basic and diluted net loss per common share	29,319,042	29,001,213

Cash as of 31 June 2022	$ 150.2M
Net Cash Burn Q3	$ 16.4M
Cash as of 30 Sept 2022	**$ 133.8M**

Clinical Trials

biomea FUSION™ We Aim to Cure™

Program	Molecule	Trial Name	Patient Number	Status and Milestones
Menin	**BMF-219** Oncology	**COVALENT-101** Hematologic Malignancies **Enrolling Now** AML/ALL, DLBCL, MM, CLL	Approximately 140 Patients across 4 Cohorts	**Trial Status:** Enrolling all Cohorts in Dose Escalation **Data:** 1 H 2023 **Dataset:** To include patients in the Acute Leukemia Cohort reporting initial safety, tolerability, and efficacy

BMF-219's ability to disrupt multiple binding partners of menin results in potent activity across multiple tumor types.

AML/ALL: BMF-219 displays in preclinical models differentiated tumor cell killing capacity
DLBCL: BMF-219 displays in preclinical models MYC disruption and potent activity in MYC-driven tumors
MM: BMF-219 displays in preclinical models MYC disruption and potent activity in MYC-driven tumors
CLL: BMF-219 displays in preclinical models key biomarker disruption and potent activity in R/R CLL tumors

Clinical Trials

biomea FUSION™ | We Aim to Cure™

Program	Molecule	Trial Name	Patient Number	Status and Milestones
Menin	**BMF-219** Oncology	**COVALENT-102** KRAS Mutant Solid Tumors **Enrolling Soon** NSCLC, PDAC, CRC	Approximately 120 Patients across 3 Cohorts	**Trial Status:** Site Activation **Data:** To be determined **Dataset:** To be determined

BMF-219's ability to disrupt multiple binding partners of Menin results in potent activity across multiple tumor types Including those driven by RAS.

KRAS G12C:	BMF-219 displays in preclinical models differentiated tumor cell killing capacity versus adegrasib and sotorasib.
KRAS G12D,V,X:	BMF-219 displays in preclinical models MYC disruption and potent activity in KRAS-driven tumors
KRAS G13 D,X:	BMF-219 displays in preclinical models MYC disruption and potent activity in KRAS-driven tumors.
KRAS pan mutations:	BMF-219's activity is independent of the activating mutation of KRAS across multiple tumor types.

BMF-219 in preclinical models have shown great tissue exposure in the target organs being explored in the study.

Clinical Trials

biomea FUSION™ We Aim to Cure™

Program	Molecule	Trial Name	Patient Number	Status and Milestones
Menin	**BMF-219** Metabolic Diseases	**COVALENT-111 Type 2 Diabetes** a double blinded, randomized, placebo controlled Phase I/II study **Enrolling Now** Type 2 Diabetes Patients	Approximately 110 Patients with Type 2 Diabetes across 5 Dosing Cohorts	**Trial Status:** Enrolling first Dosing Cohorts in Dose Escalation **Data:** 1 H 2023 **Dataset:** HV patients (Safety and Tolerability) and T2DM patients (Safety, Tolerability, and Efficacy)

BMF-219 is orally administered, with the goal of being a novel long-acting treatment that achieves and maintains glycemic control in type 2 diabetes.

In the Phase 2, COVALENT-111 will enroll subjects with a HbA1c of 7-10% despite being on standard of care, up to three agents of therapy.

Preclinical Highlights:

- BMF-219 displays in preclinical animal experiments significant glycemic control, outperforming liraglutide in reduction of fasting blood glucose by Day 29 and by OGTT on day 25.
- BMF-219 significantly reduces HbA1c levels (3.5%) in preclinical animal experiments during treatment and after drug washout.
- BMF 219 treatment restores HOMA-B scores in preclinical animal experiments to normal state indicating restored beta-cell function.
- BMF-219 significantly reduced in preclinical experiments body weight (13% at 4 weeks of treatment) and reduced blood lipemic levels

Coming Soon

Clinical Trials

biomea FUSION™ | We Aim to Cure™

Program	Molecule	Trial Name	Patient Number	Status and Milestones
FLT3	**BMF-500** Oncology	**COVALENT-103** Acute Myeloid Leukemia **Planned Enrollment** R/R Acute Leukemia	In Planning	**Trial Status:** IND enabling Studies **Data:** To be determined **Dataset:** To be determined

BMF-500 demonstrated to be a novel FLT3 inhibitor with best-in-class potential, given its efficacy, durability, and selectivity in comparison to existing FLT3 inhibitors

BMF-500 demonstrated in preclinical models potent FLT3 inhibition and high selective cell killing against AML cells harboring FLT3 activating mutations, including MV4-11 and MOLM-13, and engineered cells expressing FLT3 internal tandem duplications (FLT3-ITD) and/or FLT3 tyrosine kinase domain (TKD) mutations.

BMF-500 is a highly potent and selective, covalent, small molecule inhibitor of FLT3, that binds irreversibly to a reactive cysteine in the kinase active site. BMF-500 is a picomolar inhibitor with markedly improved potency and selectivity over gilteritinib, a reversible inhibitor of FLT3.

THANK YOU

biomea FUSION™ | We Aim to Cure™

Biomea Fusion
900 Middlefield Road, 4th floor
Redwood City, CA, 94063
biomeafusion.com

Contact:
Sasha Blaug
SVP Corporate Development
sb@biomeafusion.com
U.S. +1-650-460-7759