Galecto

TRANSFORMING TREATMENT

of cancer and liver disease with first-in-class small molecule agents

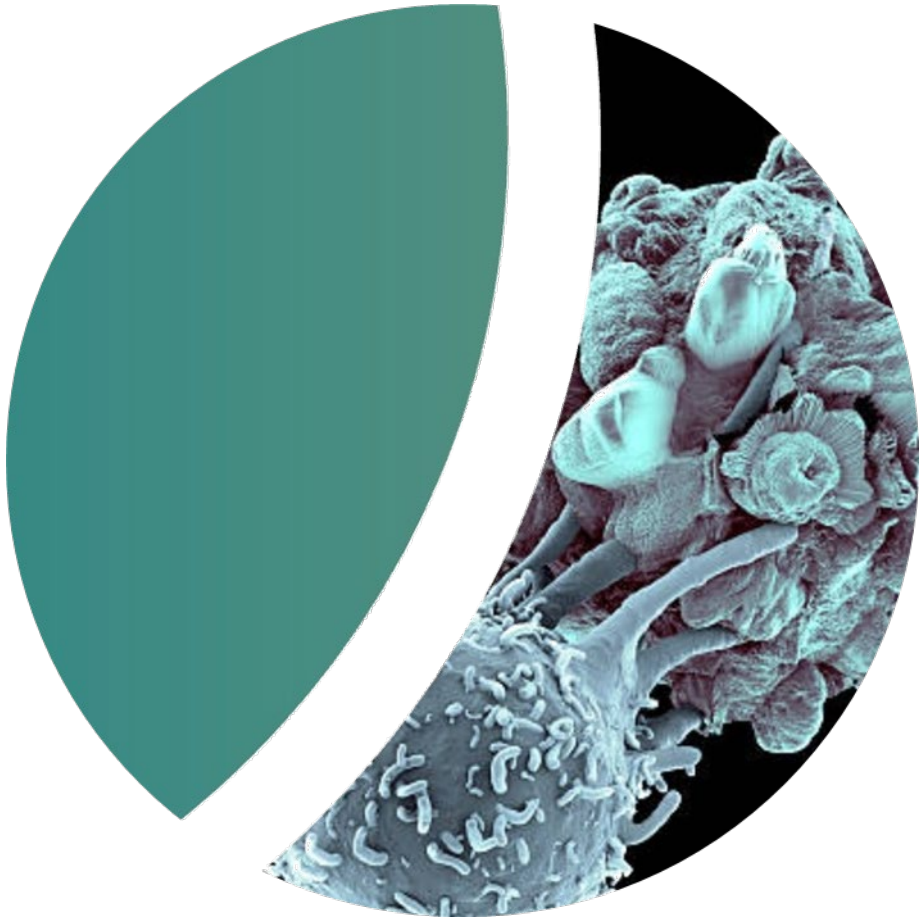
October 2024

Forward-looking statements

This presentation contains forward-looking statements about Galecto, Inc.'s ("Galecto" or the "Company") strategy, future plans, operations and prospects, including, but not limited to, statements regarding the development of Galecto's compounds and potential opportunities, including BRM-1420; the expected timing and reporting of results of Galecto's clinical trials; and Galecto's expected cash runway. These statements involve substantial risks and uncertainties. All statements, other than statements of historical facts, contained in this presentation are forward-looking statements. The words ''anticipate,'' ''believe,'' ''continue,'' ''could,'' ''estimate,'' ''expect,'' ''intend,'' ''may,'' ''plan,'' ''potential,'' ''predict,'' ''project,'' ''target,'' ''should,'' ''would,'' and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. For such statements, Galecto claims the protection of the Private Securities Litigation Reform Act of 1995. The Company may not actually achieve the plans, intentions, or expectations disclosed in these forward-looking statements, and you should not place undue reliance on these forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in these forward-looking statements. Factors that could cause actual results to differ materially from such statements include, without limitation: that drug development is expensive, time consuming, uncertain and susceptible to change, interruption, delay or termination; enrolling patients in clinical trials is competitive and challenging and the expected timing of Galecto's planned readouts for its ongoing clinical trials may be delayed as a result; that the timing and outcome of research, development and regulatory review and feedback is uncertain; Galecto's need to raise additional capital to advance all of its programs; the amount of Galecto's future losses is uncertain and could cause our stock price to fluctuate or decline; top-line data may not accurately reflect the complete results of a particular study or trial; results of clinical trials and other studies are subject to different interpretation and may not be predictive of future results; Galecto's clinical trials may fail to demonstrate adequately the safety and efficacy of any of its drug candidates; Galecto's drug candidates may not advance in development or be approved for marketing; clinical trial and other studies may not proceed at the time or in the manner expected or at all; clinical and nonclinical data is voluminous and detailed, and regulatory agencies may interpret or weigh the importance of data differently and reach different conclusions than Galecto or others, request additional information, have additional recommendations or change their guidance or requirements; data and information related to the Company's programs may not meet regulatory requirements or otherwise be sufficient for further development at all or on the Company's projected timeline; and other risks related to developing, seeking regulatory approval of and commercializing drugs, including regulatory, manufacturing, supply and marketing issues and drug availability. Additional factors that could cause results to differ materially from those stated or implied by Galecto's forward-looking statements are disclosed in its Securities and Exchange Commission (SEC) filings, including its most recent Annual Report on Form 10-K, filed with the SEC on March 8, 2024, under the headings "Risk Factors." In addition, the forward-looking statements included in this presentation represent the Company's views as of the date of this presentation. The Company anticipates that subsequent events and developments will cause its views to change. However, while the Company may elect to update these forward-looking statements at some point in the future, it specifically disclaims any obligation to do so.

Galecto

is working to create a world where patients suffering from cancer and severe liver diseases have effective treatment solutions



Galecto enhances pipeline following strategic review

Cash balance of ~$22.9M as of June 30, 2024 funds ongoing extension trials and acquired program with runway into 2026

Completed Strategic Review Identifies Core Assets and Direction

Following an intensive strategic review process, Galecto has determined to focus on

- cancer and liver disease
- existing clinical stage asset GB1211

Pipeline is bolstered by acquisition of BRM-1420 for AML, which brings novelty and breakthrough potential

- Superior activity in preclinical AML models
- Identified after review of multiple assets, assisted by Leerink

Enhanced Pipeline Focused On Oncology and Liver Disease

Innovative platforms targeting core disease processes

- Pioneers in ENL-YEATS and galectin-3 based pharmacology
- First-in-class highly specific, oral small-molecule inhibitors

All programs address:

- Diseases characterized by clear unmet medical need
- Multi-billion-dollar market opportunities

Early Data Supportive of Drug Activity

ENL-YEATS inhibitor:

- MoA estimated to cover 30%+ of AML population
- Rapid and durable anti-tumor activity in AML models

Galectin-3 Inhibitors:

- Target engagement shown
- Positive biomarker data
- Evidence of tumor microenvironment transformation
- Significant clinical data in cirrhosis

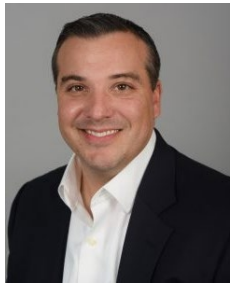
Galecto pipeline post acquisition

PRODUCT CANDIDATE	INDICATION	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	NEXT STEPS	DEMONSTRATED RESULTS
BRM1420	AML	AML				IND ready Q4 2025	Promising preclinical profile showing rapid and durable anti-tumor activity
GB1211	Oncology: NSCLC	GALLANT-1 (Oral Gal-3 inhibitor)				Initiation of Phase 2/3 study TBD*	Evidence for transforming tumor microenvironment and prevention of galectin-3 mediated checkpoint inhibitor resistance
GB1211	Oncology: Melanoma & HNSCC	IIT Phase 2 Trial				Enrollment completion 2025	
FIBROSIS PROGRAM	**INDICATION**	**PRECLINICAL**	**PHASE 1**	**PHASE 2**	**PHASE 3**	**NEXT STEPS**	**DEMONSTRATED RESULTS**
GB1211	Liver Cirrhosis	GULLIVER (Oral Gal-3 inhibitor)				Initiation of long-term cirrhosis trial TBD*	Clinical activity in liver cirrhosis

*Subject to obtaining additional financing

Experienced management team & board of directors

Hans Schambye
CEO



Garrett Winslow
GC

Matt Kronmiller
EVP, Strategy & CBO

Lori Firmani
CFO

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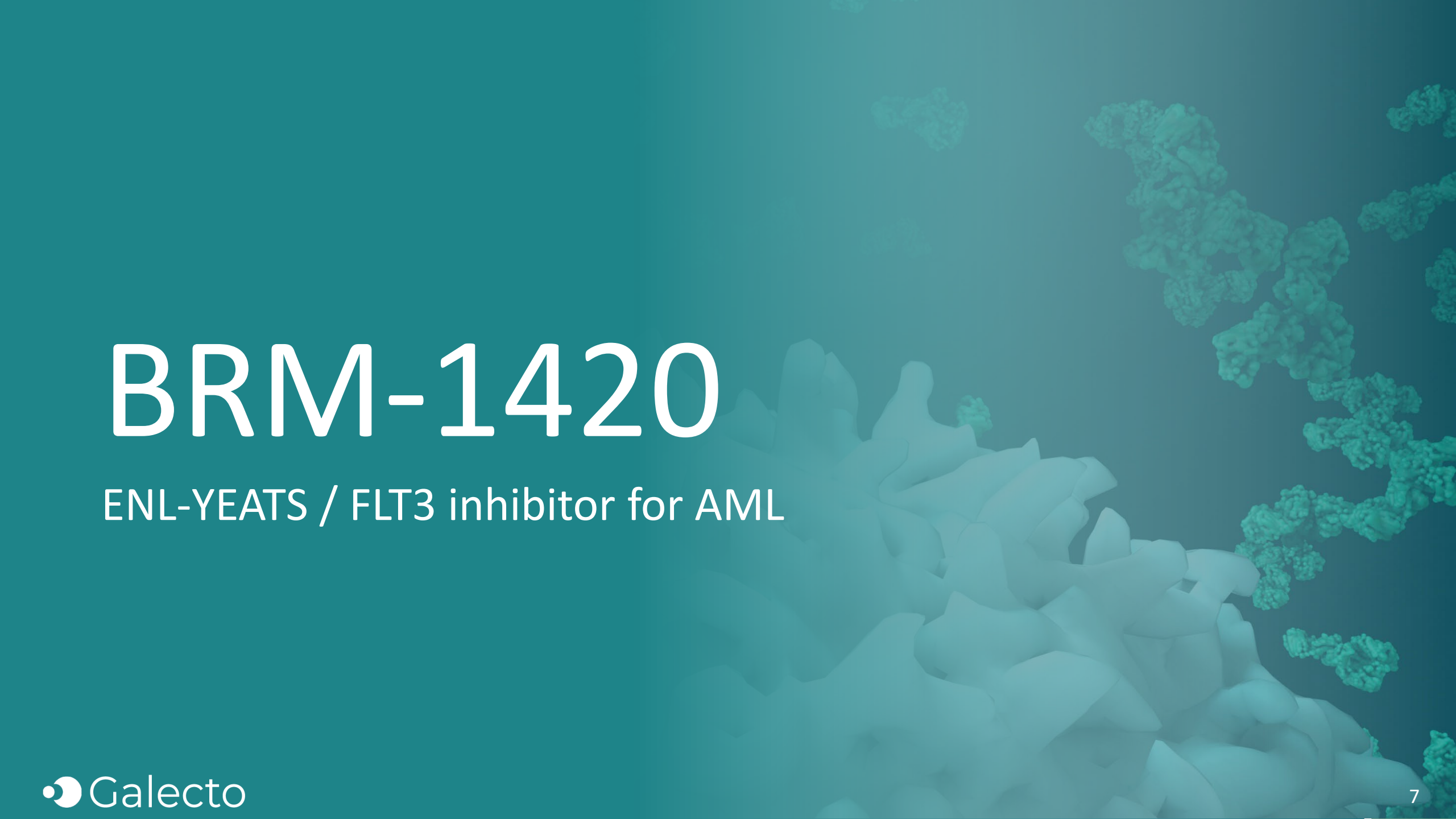
BRM-1420

ENL-YEATS / FLT3 inhibitor for AML



Galecto

BRM-1420 – Newly acquired asset from Bridge Medicines

Novel, orally-active ENL-YEATS / FLT3 inhibitor

Indicated for treatment of multiple genetic subsets of AML

- MoA estimated to cover 30%+ of AML population and potential high-risk genetic drivers
- Ideally positioned to treat both FLT3-inhibitor and menin-resistant patients
- Optimal combination partner with frontline therapies

Demonstrated potential best-in-class status, differentiated from standard of care and current known pipeline opportunities

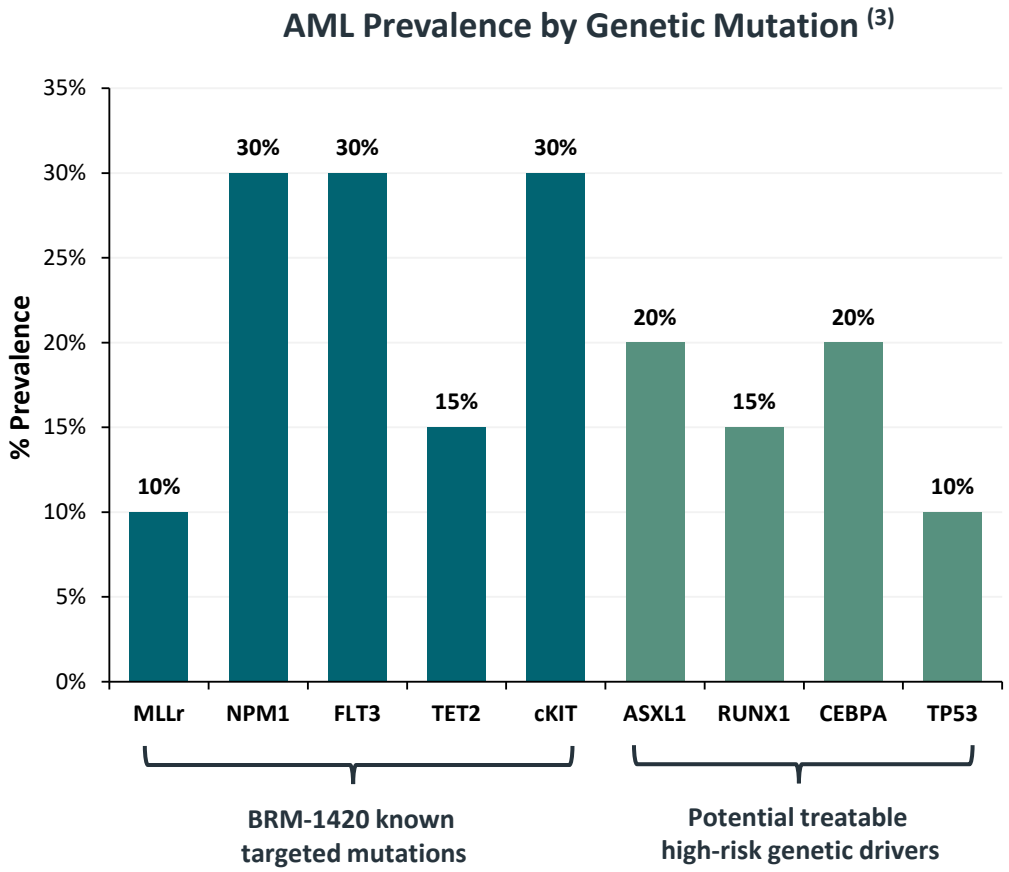
- *In vivo* therapeutic window that demonstrates superior efficacy to FLT3 and menin inhibitors in rigorous models of MLLr AML (KMT2A)
- Excellent cardiovascular safety (no QTc) and toxicology profile
- Synergistic with standard of care molecules and compounds in development

Initial clinical data expected in 2026; Established regulatory pathway

ENL-YEATS / FLT3: Commercial opportunity

BRM-1420 has potential to address a broader population within AML

- >20,000 new cases of AML in the US per year, with prevalence projected to increase and total market size expanding to $10 billion by 2028 (1)
- We estimate that ~7.5% - 10.0% of patients suffer from MLLr AML and ~30% of AML patients possess FLT3 or NPM1 mutations
- High proportion of patients who do not respond with current first-line treatment or relapse
- Unmet medical need remains incredibly high as outcomes continue to be challenging
- **Potential for BRM-1420 to treat a broader patient population within r/r AML, including those who develop resistance to menin inhibitor therapy and those with high-risk mutations**
- **Candidate to treat patients in earlier line settings as a key combination therapy**



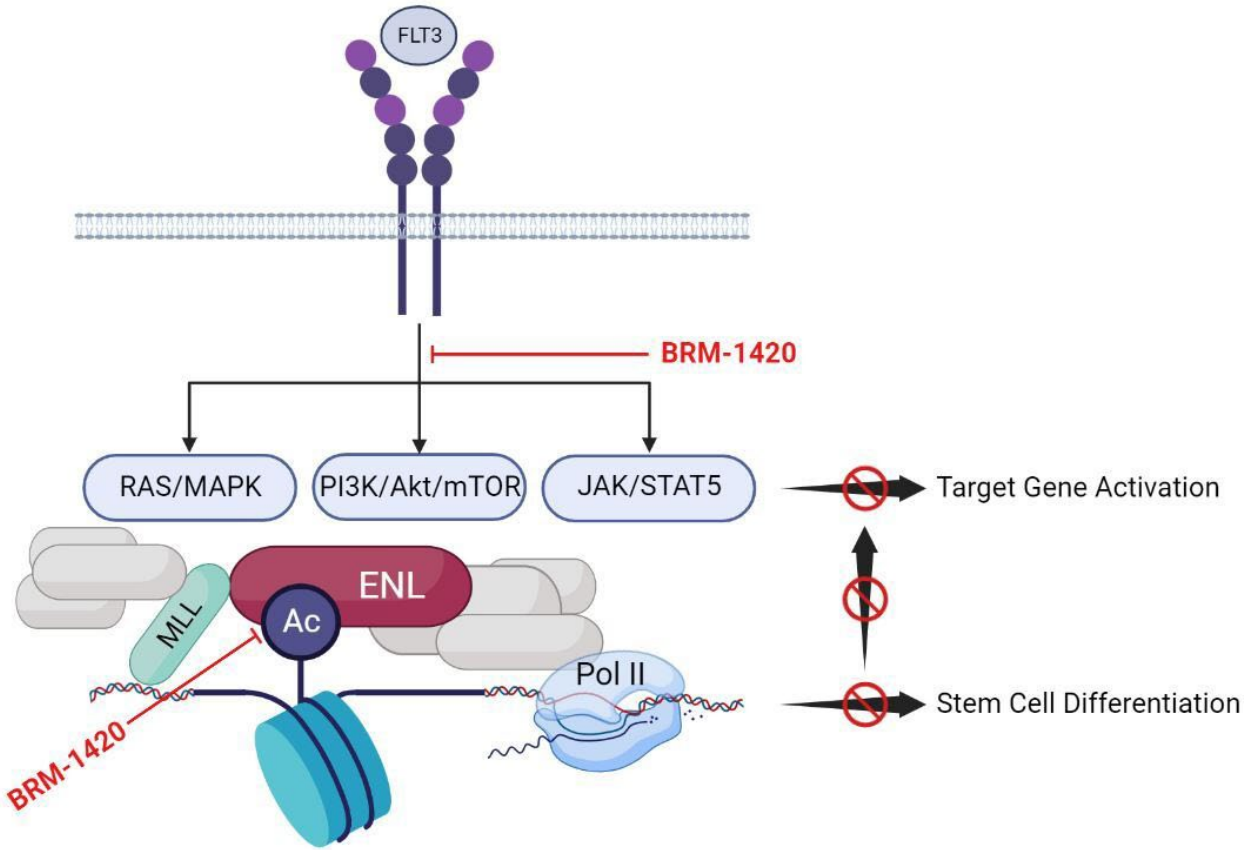
1 *EvaluatePharma* estimated AML therapeutic sales in 2028.

2. Source: "Clinical implications of recurrent gene mutations in acute myeloid leukemia" (Yu, Li, et. al); "Biomarkers in Acute Myeloid Leukemia: Leveraging Next Generation Sequencing Data for Optimal Therapeutic Strategies" (El Achi & Kanagal-Shammana).

BRM-1420 mechanism of action (ENL YEATS / FLT3)

Unique mechanism inhibits both ENL-YEATS and FLT3; this combination expands points of therapeutic intervention and increases potential for enhanced clinical effectiveness versus FLT3 inhibition alone

- Dr. David Allis, along with Dr. Liling Wan (now of University of Pennsylvania, recognized the importance of YEATS domain of ENL/AF9 as a “reader” of acetylated histones on the MLL fusion protein, regulating transcription of key drivers of stem cell differentiation and leukemogenesis
- Concept and rudimentary chemical matter originally licensed from The Rockefeller University in 2020
- Dr. Wan’s discoveries published by *Cancer Discovery* in November 2022
- Broad patent estate of ENL-YEATS inhibitors that also demonstrate activity at a variety of other leukemia targets, including FLT3



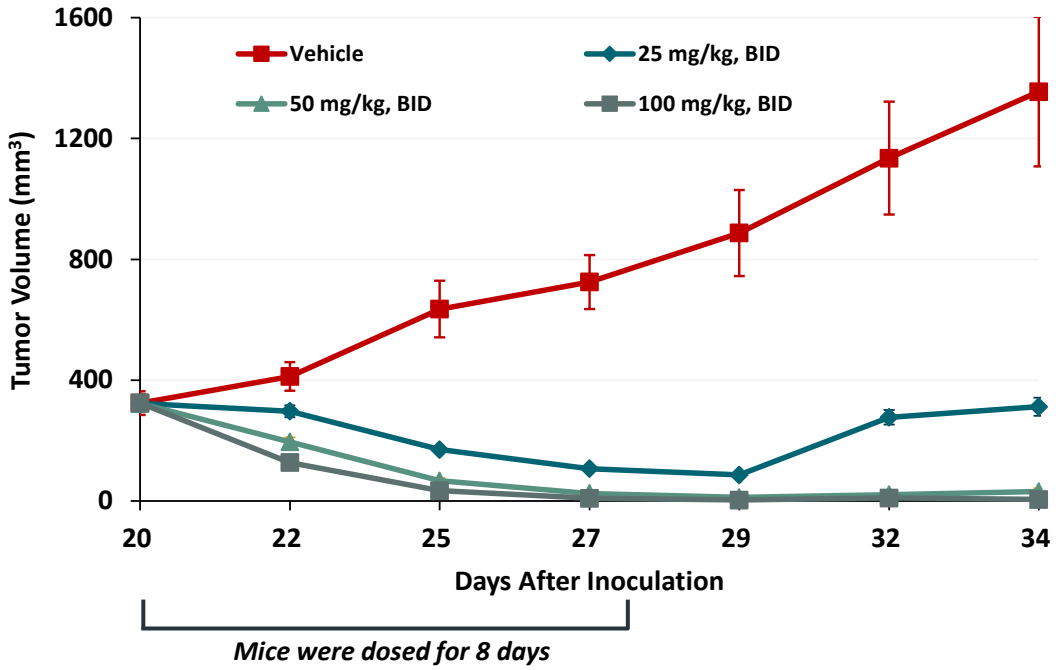
Preclinical data and potential clinical implications

- Rapid and durable anti-tumor activity with survival > comparators
- Tolerability > comparators
- Pronounced effects on key genetic drivers of leukemogenesis and maintenance (HOXA9, MEIS1, MYC)
- Substantial reduction/elimination of blast cells in peripheral blood, bone marrow and spleen
- Immediate cell cycle arrest, differentiation of blasts and apoptosis
- Preclinical data shows potential for rapid onset of action, complete and durable responses, efficacy potentially superior to competitors, and a better safety/tolerability profile

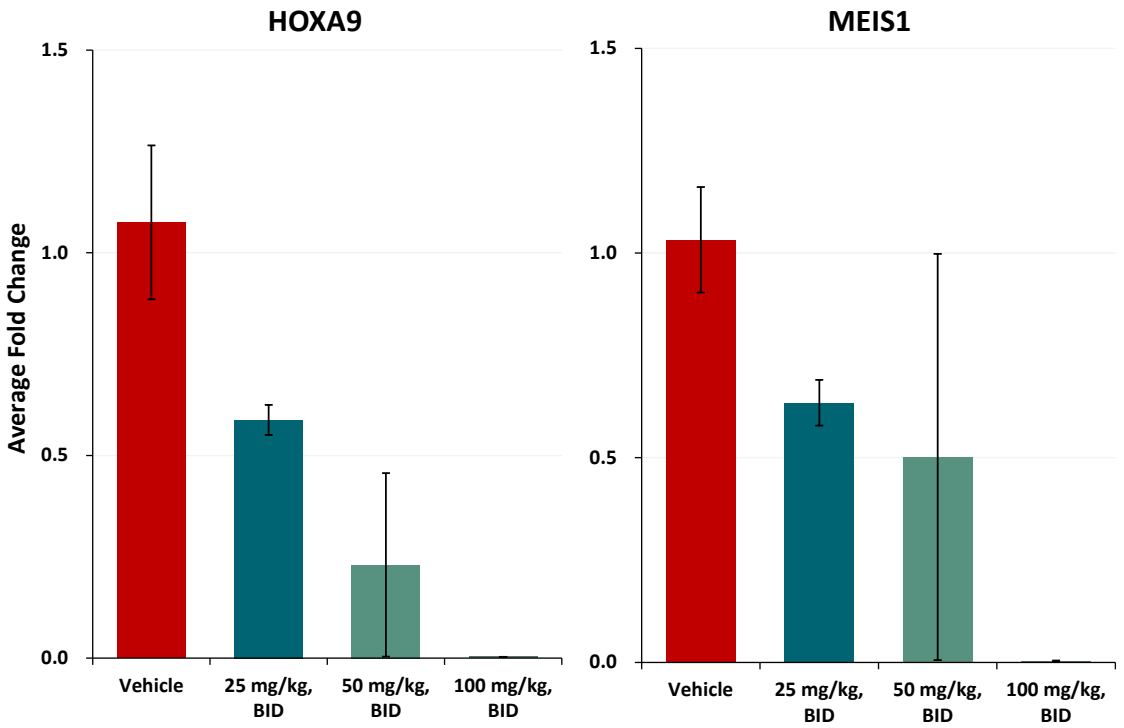
BRM-1420 inhibits tumor growth and reduces critical biomarkers in mouse model

- Potent inhibitor of tumor growth
- Reduction of critical biomarkers indicate suppression of leukemic stem cell development in the bone marrow
- FLT3 promotes rapid onset, ENL-YEATS promotes duration of response

Tumor Volume Reduction



Biomarker Suppression

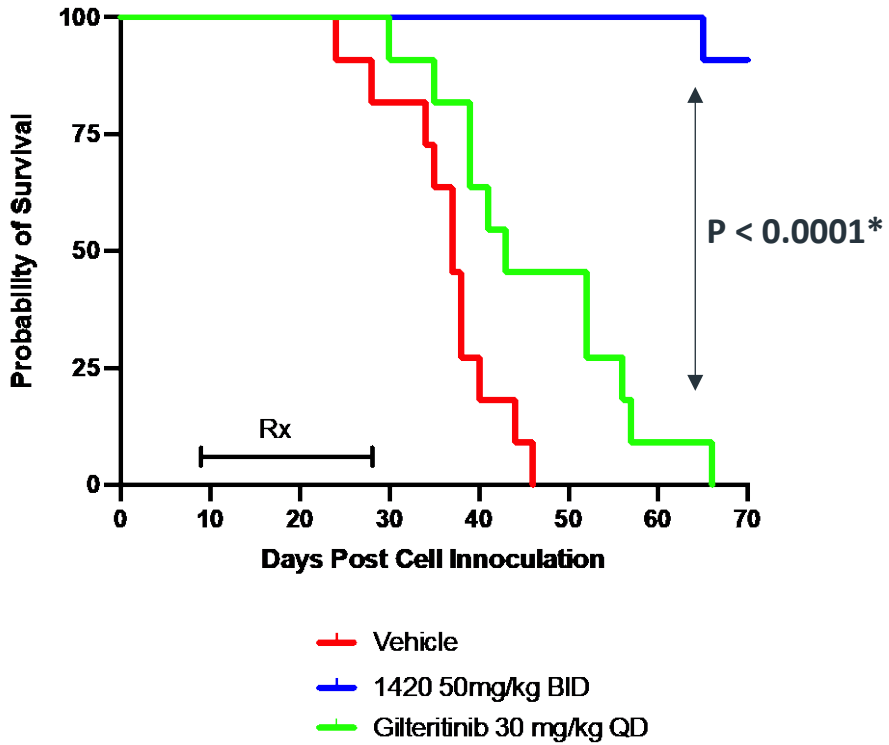


MV4;11 mouse model. Biomarker expression at Day 14.

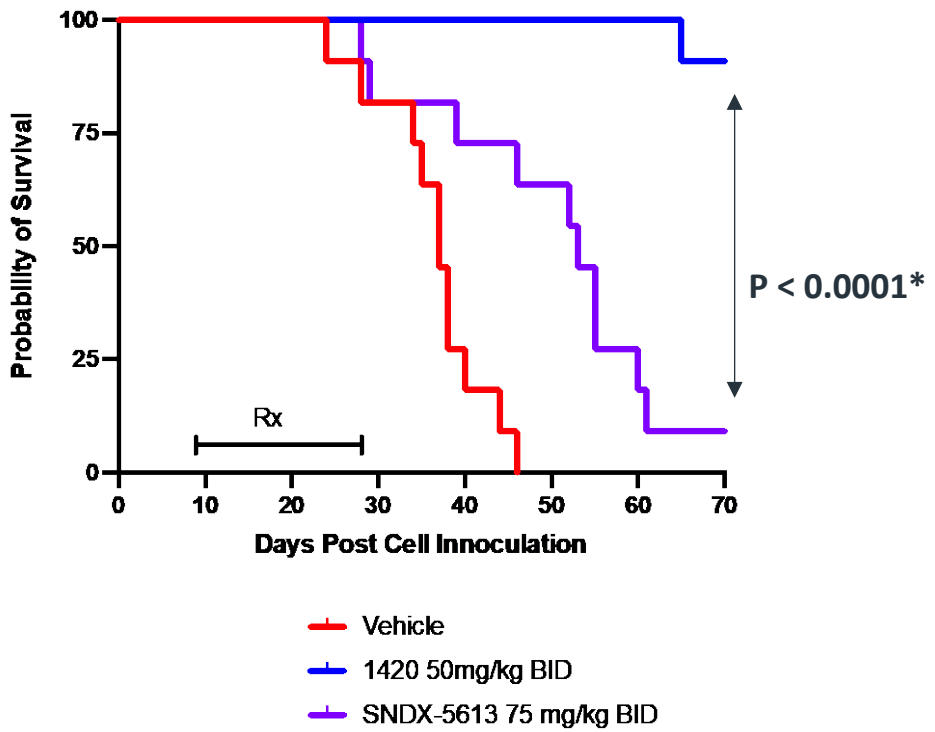
BRM-1420 vs. comparators in survival

BRM-1420 observed to be statistically superior to Gilteritinib and SNDX-5613 in prolonging survival

BRM-1420 / Gilteritinib



BRM-1420 / SNDX-5613

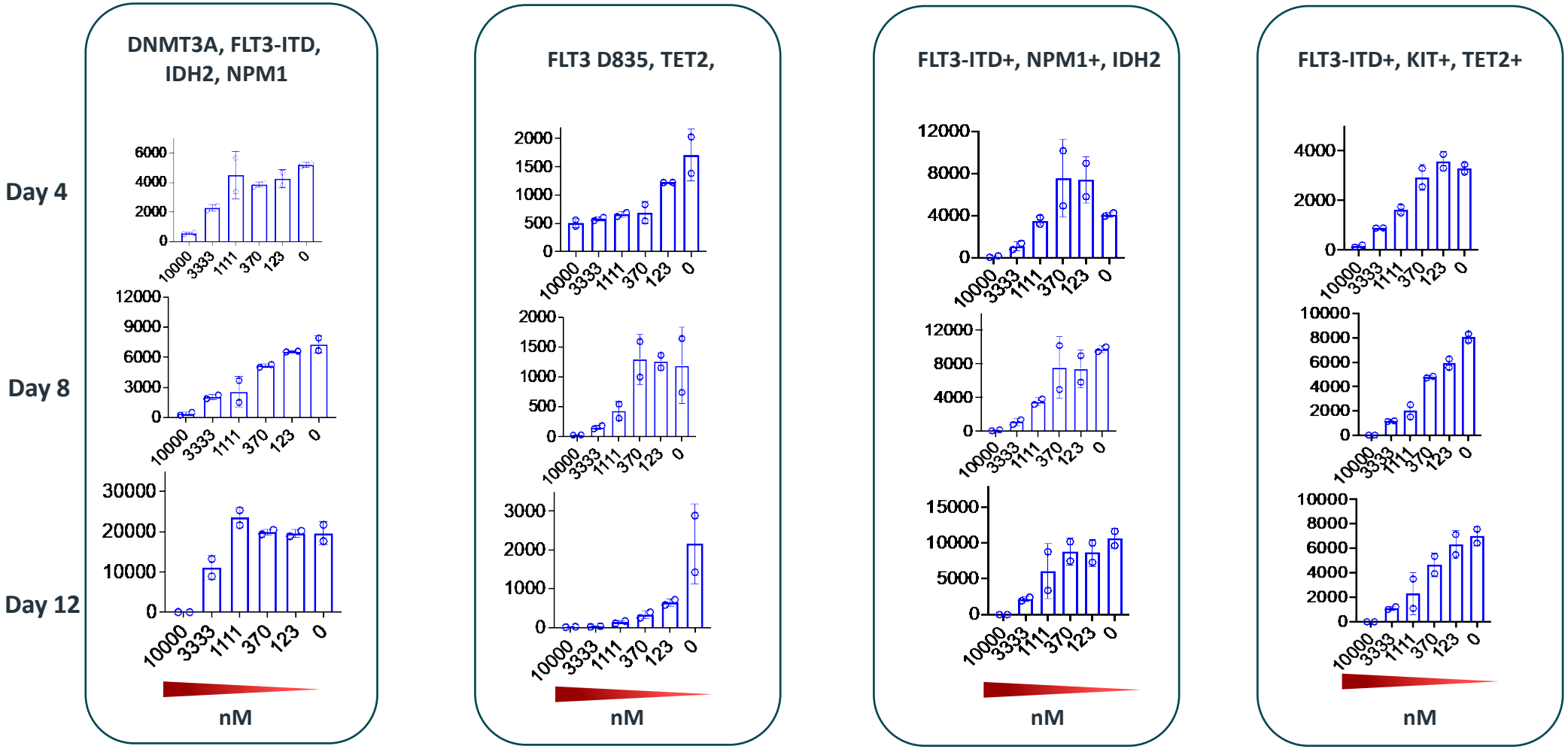


Disseminating MV4;11 mouse model of AML.
* Log-rank (Mantel-Cox) test
Gehan-Breslow-Wilcoxon test

Translation studies in AML patient samples

BRM-1420 highly effective in inhibiting patient sample cell viability; activity against non-MLLr cell lines

Co-mutations included FLT3-ITD+, FLT3 D835, TET2+, KIT+, TP53, ASXL1



MEN1 mutations mediate clinical resistance to menin inhibition

Resistance seen in first generation menin inhibitors

- Phase I first-in-human trial of revumenib showed somatic mutations in MEN1 at the revumenib-menin interface in patients with acquired resistance to menin inhibition
- Mutations represent a conserved mechanism of therapeutic resistance in xenograft model and in unbiased base-editor screen
- Attenuate drug-target binding but no the interaction with the natural ligand MLL1
- Menin is a key scaffolding protein for several targets controlling leukemogenesis and leukemia stem cells, including ENL-YEATS
- Menin mutation occurs rapidly and in a large number of patients
- BRM-1420 targets both FLT3 (WT, D835) and ENL-YEATS

Article

***MEN1* mutations mediate clinical resistance to menin inhibition**

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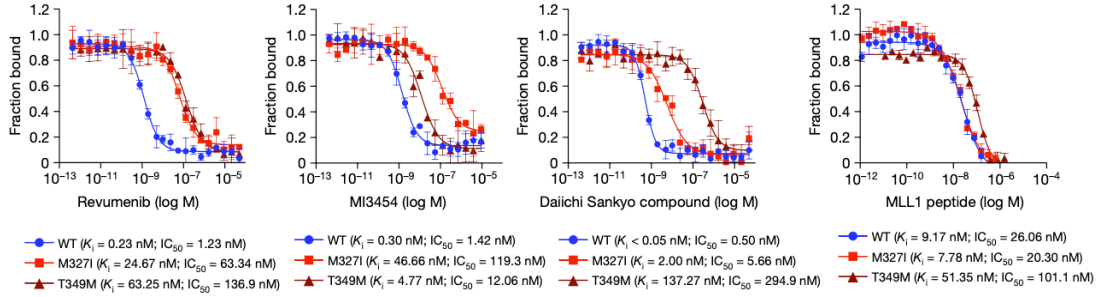


https://doi.org/10.1038/s41586-023-05755-9

Represents attractive market opportunity for BRM-1420

In Vitro combination study examples: 2D and 3D views

BRM-1420 has been tested in combination with SoC and next-generation therapies

- BRM-1420 has been tested *in vitro* in combination with a variety of different standard of care compounds, as well as pipeline candidates
- Demonstrated additive and/or synergistic effects with first-line standard of care (e.g., cytarabine) and emerging standard of care (e.g., Venetoclax)
- Similarly, demonstrated additive/synergistic effects with menin inhibitor

BRM1420 Combination Partner

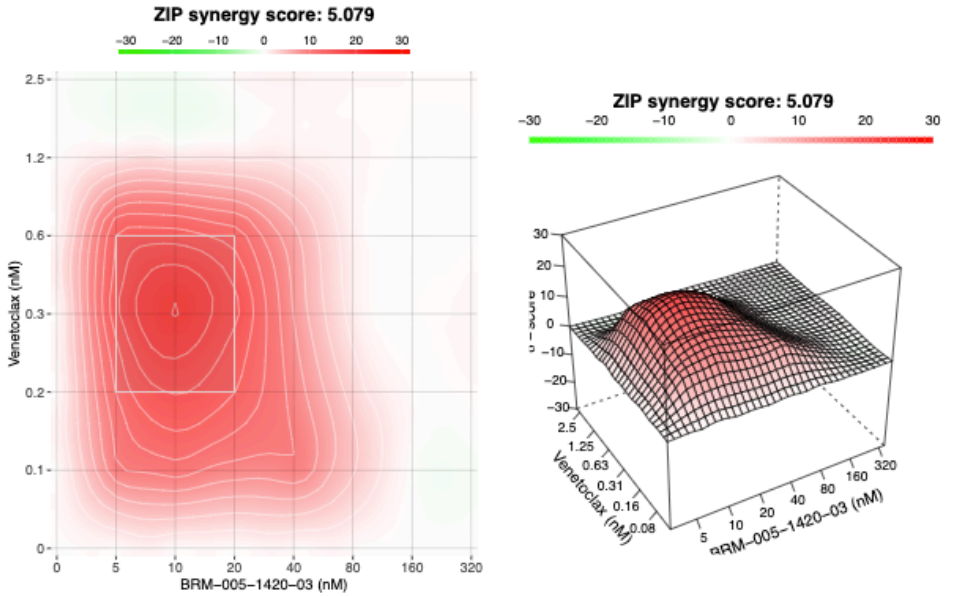
Synergistic

Additive

Cell Line	Cytarabine	Gilteritinib	Venetoclax	SNDX-5613
HL60				
MOLM-13				
MV4;11				
OCI-AML-3				

Illustrative BRM-1420 Synergy Topography

BRM-1420 + Venetoclax in MOLM-13 Cells (Max synergy 15.1)



BRM-1420 toxicology summary

Well-tolerated in both rats and dogs with no QTc liability

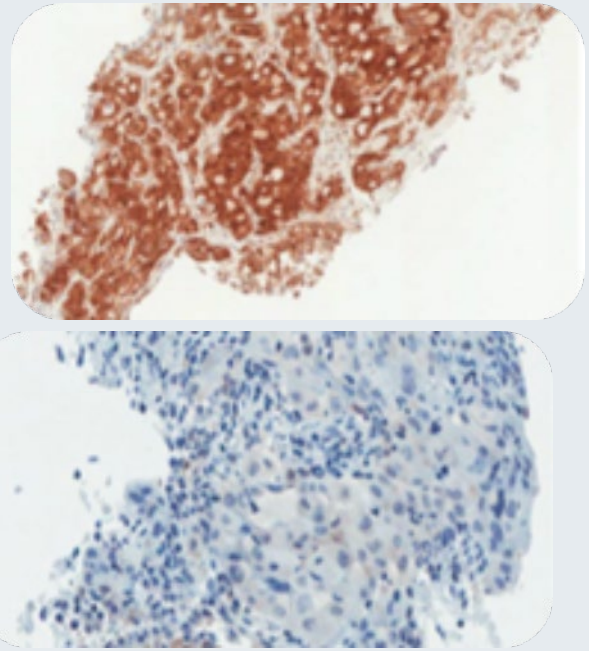
- Current standard of care and compounds in development have encountered safety / PK liabilities
  - Common side effects of FLT3-inhibitor gilteritinib include diarrhea, anemia, fatigue and elevated LFT; warning included about QT prolongation
  - Syndax's menin-inhibitor has issues with tolerability (QT prolongation) and drug-drug interactions (CYP3A4 inhibitors)
- No statistically significant QTc prolongation seen with BRM-1420 up to 100 mg/kg in dog cardiovascular study
- No deaths, target organ toxicity, or changes in clinical chemistry seen in 14-day pilot studies in rats and dogs
- Some effects on WBC counts, hematocrit and hemoglobin that were mild and manageable in a clinical setting
  - Effects on bone marrow / blood cell counts typically more severe with SoC in AML
- Reduction in reticulocytes observed but is easily monitorable in a clinical setting and reversable
  - Effect on reticulocytes may be attributable to inhibition of AF9 and is an indirect surrogate for target engagement
- In dog study, study operator considered 200 mg/kg to be an MTD based on recurrent vomits, presence of soft to liquid feces and body weight loss

GB1211

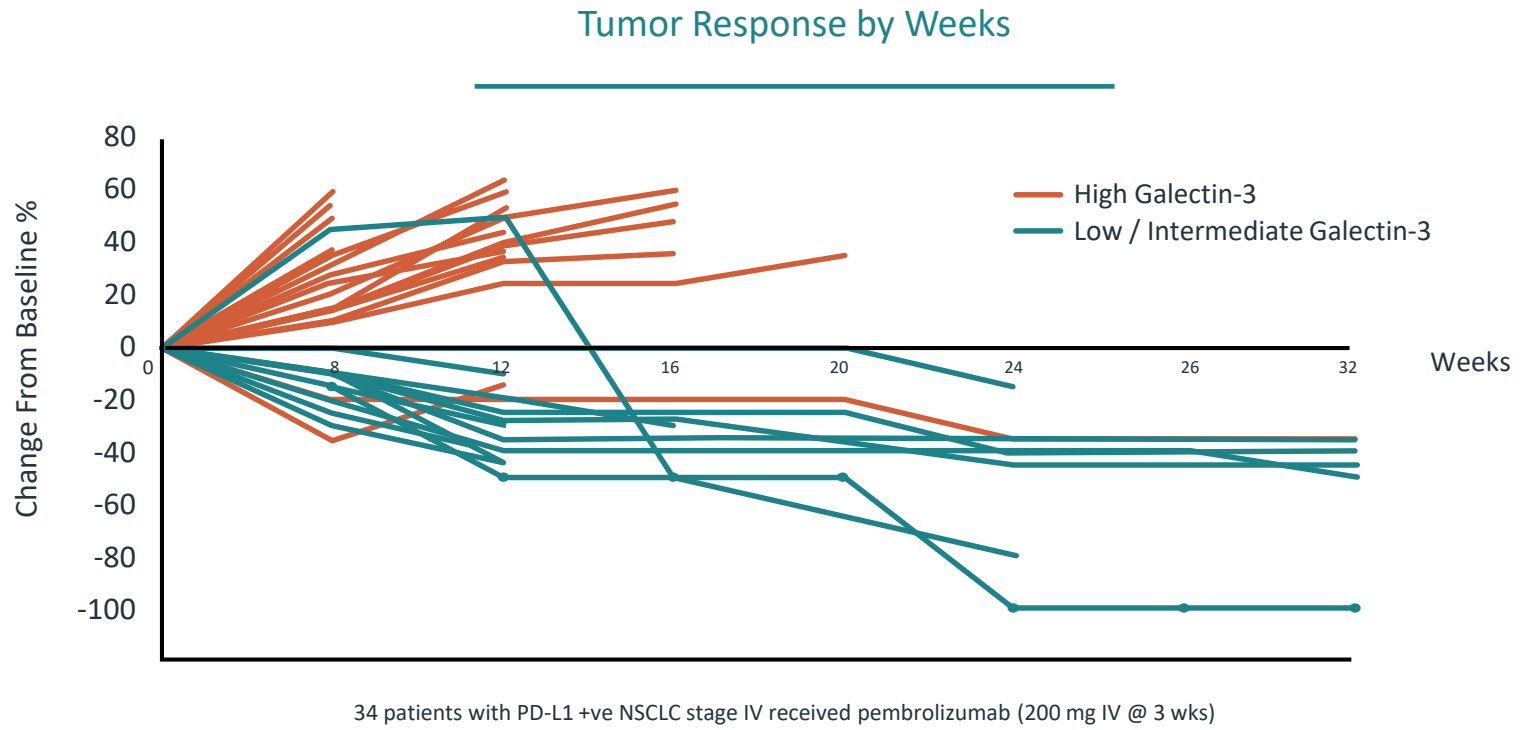
Oral Galectin-3 Inhibitor for Cancer

Galecto

Galectin-3 expression predicts response to pembrolizumab in NSCLC



Galectin-3 expression in NSCLC biopsies



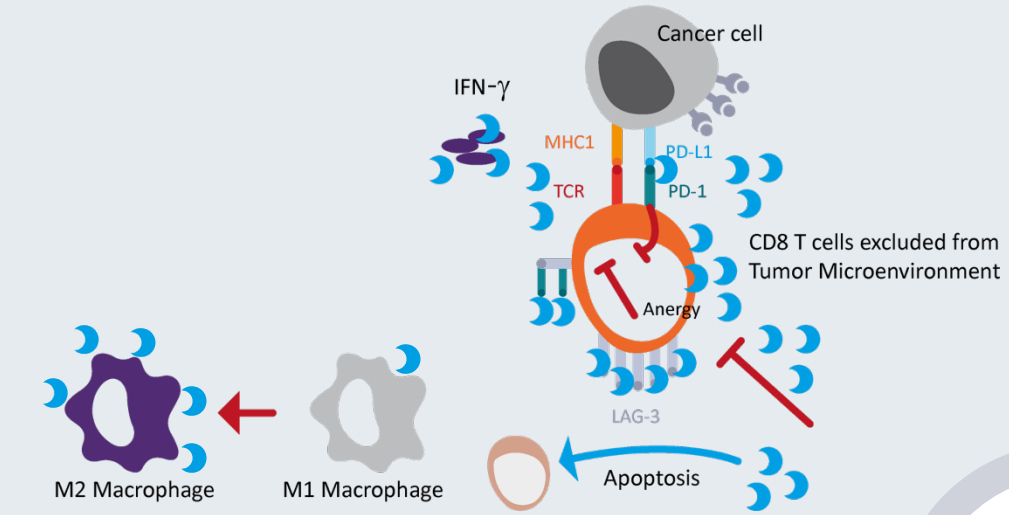
34 patients with PD-L1 +ve NSCLC stage IV received pembrolizumab (200 mg IV @ 3 wks)

- High galectin-3 expression in patients with NSCLC strongly correlated with tumor resistance to pembrolizumab
- A clinical response was seen in tumors with a negative, low or intermediate galectin-3 expression

GB1211 increases CD8+ T cell recruitment and activation in galectin-3 rich tumor microenvironment, and potentiates checkpoint inhibitors

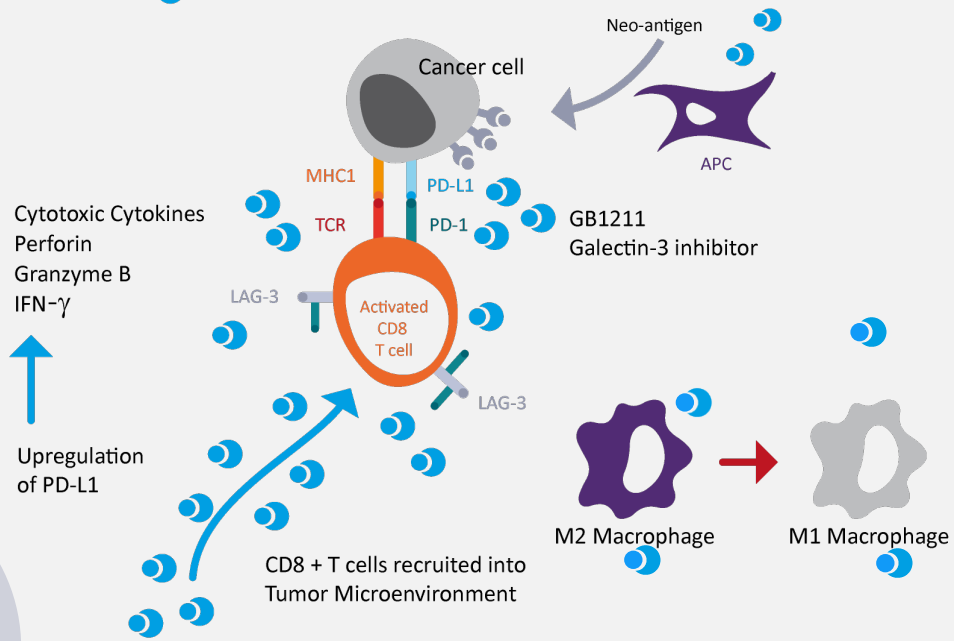
Galectin-3 rich Tumor Microenvironment

Galectin-3



GB1211 blocks Galectin-3 – recruits and activates CD8+T cells

GB1211
Galectin-3 inhibitor

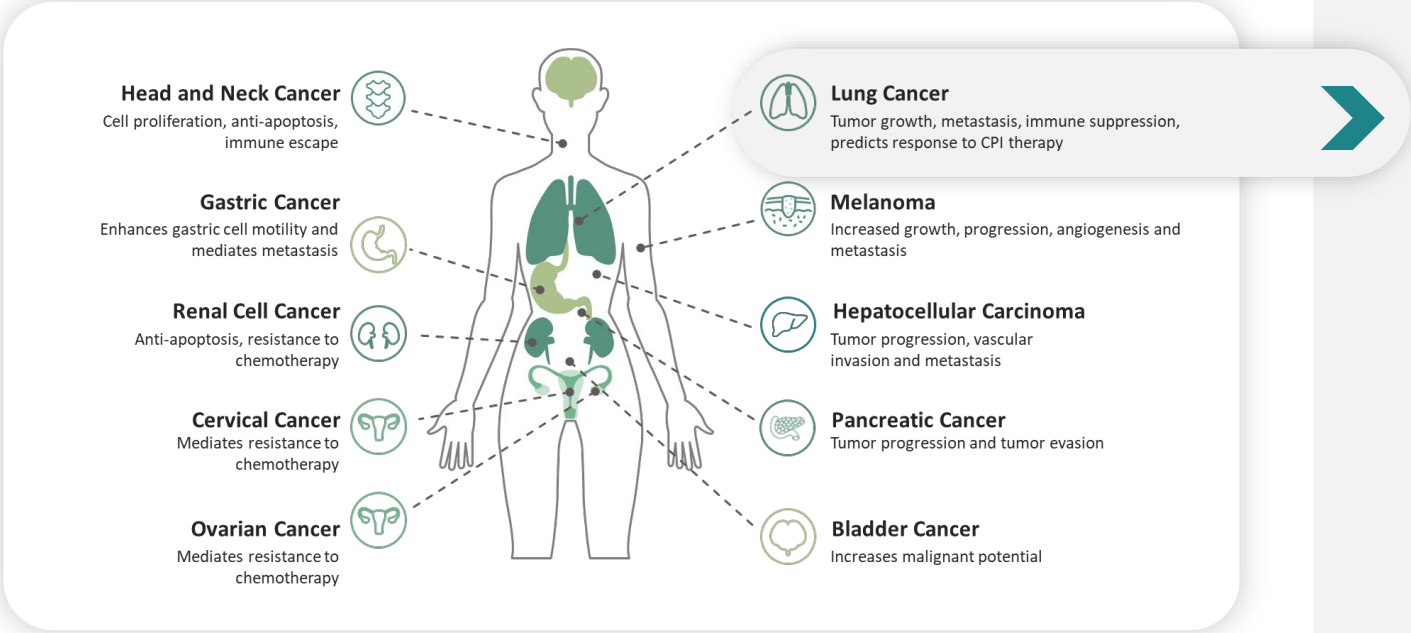


CD8 + T cell

APC (Antigen Presenting Cell), IFN-γ (Interferon-γ), LAG-3 (Lymphocyte Activating Gene 3), MHC1 (major histocompatibility complex 1), TCR (T-cell receptor), PD-L1 (Programmed death-ligand 1), PD-1 (Programmed cell death protein 1),

Galecto chose NSCLC as first development target

NSCLC represents a significant unmet medical need with a strong rationale for anti-Galectin-3 therapy



ASCO: Cancer.net (01-2021)
Ebrahim et al (2014); Ann Transl Med;2(9):88
Kuou et al (2015); Cancer Immunol Res;3: 412
Ou et al (2021); Ther Adv Med Oncol;13: 1
Capalbo et al. (2019); Int. J. Mol. Sci.;20
Vuong et al (2019); Cancer Res;79: 1480

High unmet need

- Lung cancer is 2nd most common cancer and leading cause of cancer death
  - More than 130,000 death/year in US
  - 1.59 million deaths/year globally
- NSCLC has a poor prognosis – 5-year survival <25%
  - Metastatic NSCLC: 5-year survival rate < 7%
- Billion-dollar market opportunity

Galectin-3 is a promising target that

- Predicts overall poor survival
- Predicts response to CPI therapy

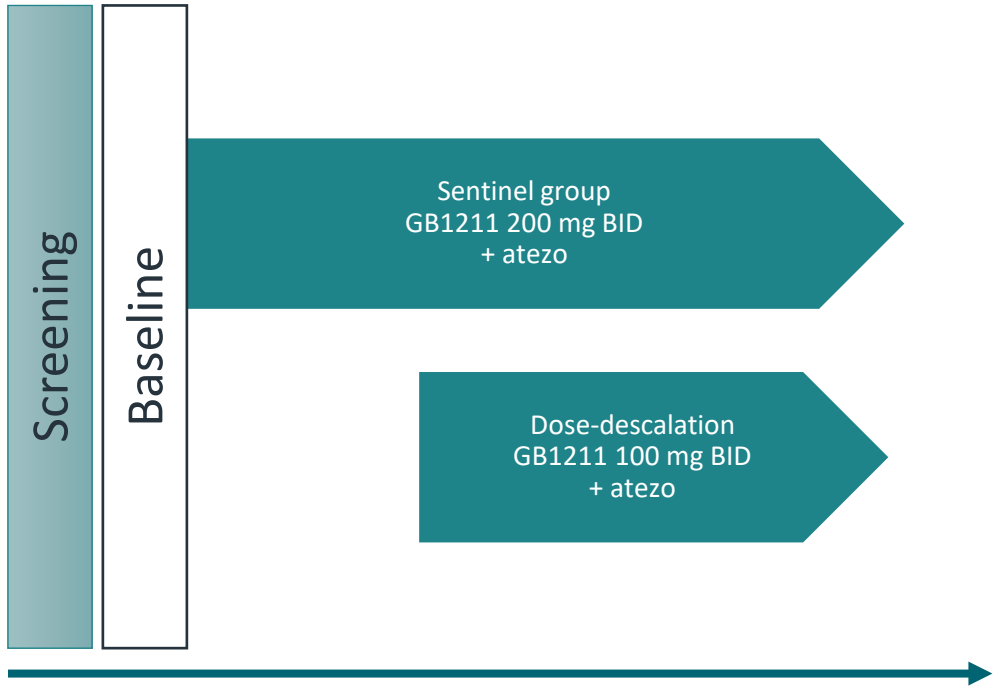
CPI therapy for treatment of NSCLC is well established

- However, 40-60% of patients don't respond to therapy
- Gal-3 inhibitors show:
  - Anti-tumor effects
  - T cell activation – LAG3 blockade
  - Macrophage polarizations
  - Increased apoptosis

GALLANT-1 - Part A/C: atezolizumab + GB1211

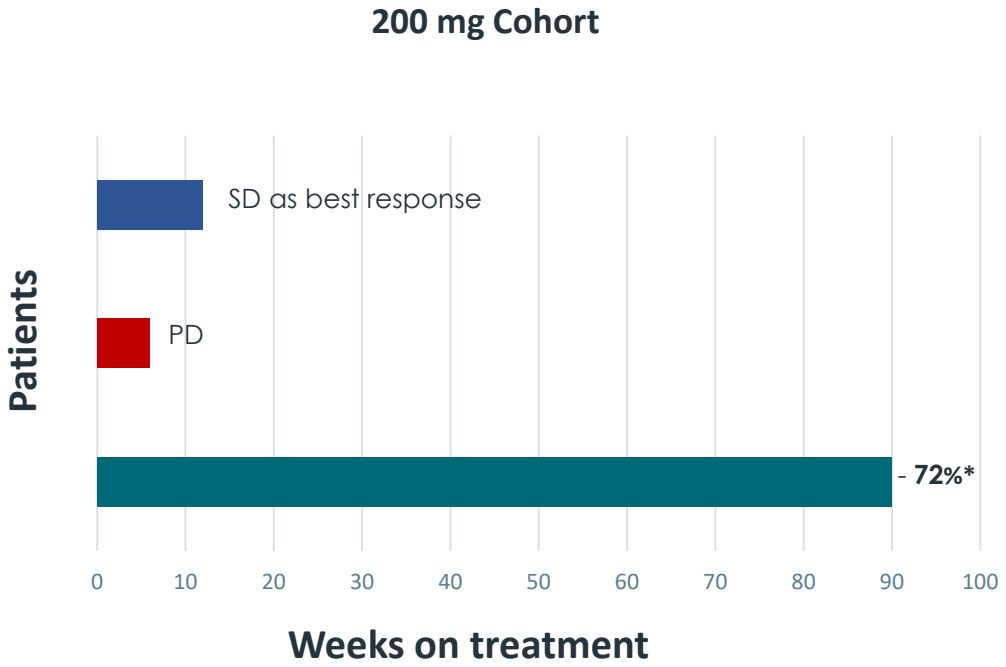
Open label study to investigate the safety and efficacy of GB1211 in combination with atezolizumab in the 1st-line treatment of patients with Non-Small Cell Lung Cancer. **The study was performed in collaboration with Roche**

Patient population: Advanced or metastatic NSCLC expressing PD-L1 on at least 50% of tumor cells
Primary endpoint: Safety and tolerability, tumor shrinkage; Secondary endpoints includes PK measurements, ORR (RECIST)
Exploratory: Effect of GB1211 on pharmacodynamic markers and biomarkers

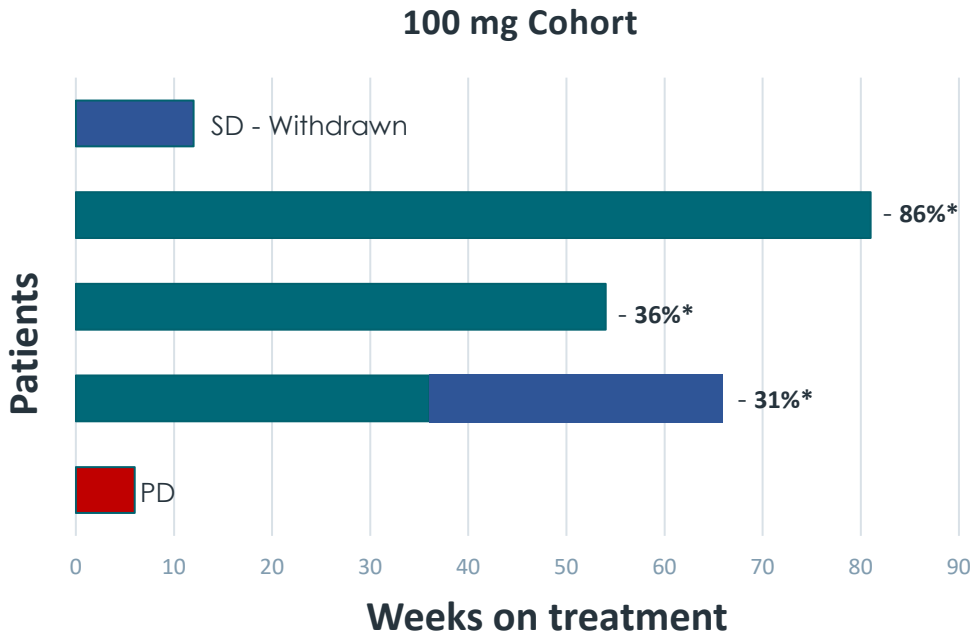


Encouraging tumor response rates observed in GALLANT-1

High ORR and long duration of response were observed at the recommended phase 2 dose level of 100 mg GB1211 BID in combination with atezolizumab in the 1st line treatment of NSCLC



*Best overall tumor volume reduction



*Best overall tumor volume reduction

Partial Response (PR)

Stable Disease (SD)

Progressive Disease (PD)

*Eight evaluable patients with >2 cycles of treatment and at least 1 post-baseline scan (week 6)
*Two patients in the 200 mg group were discontinued from the study due to MoA related toxicity (lymphocyte infiltrated skin rash)
*Three out of five (60%) evaluable patients at the recommended phase 2 dose demonstrated sustained PR

Conclusions - GALLANT-1

GB1211 appears to be well-tolerated with encouraging efficacy in combination with atezolizumab monotherapy

- Dose-limiting skin reactions observed at 200 mg dose indicate lymphocyte activation and hence support MoA for GB1211 in combination with atezolizumab
- Patients included in GALLANT-1 are not suited for chemotherapy-CPI combination and despite ECOG 0-1 performance status, the study population may represent a segment of hard-to-treat patients
- The 100 mg GB1211 BID dose on top of atezolizumab appears safe and well-tolerated
  - No safety concerns in patients on long-term treatment
  - 100 mg dose seems adequate based on dose-scaling modeling
- Responses seem to exceed the expected efficacy of atezolizumab monotherapy
  - ORR at R2PD 60% vs. anticipated 30%
  - The duration of response and PFS already now appears longer than anticipated, with all responding patients receiving treatment for more than 1Y
- As of September 30, 2024, two patients are being treated with GB1211+atezo in the extension phase of the trial

Gulliver-2

GB1211

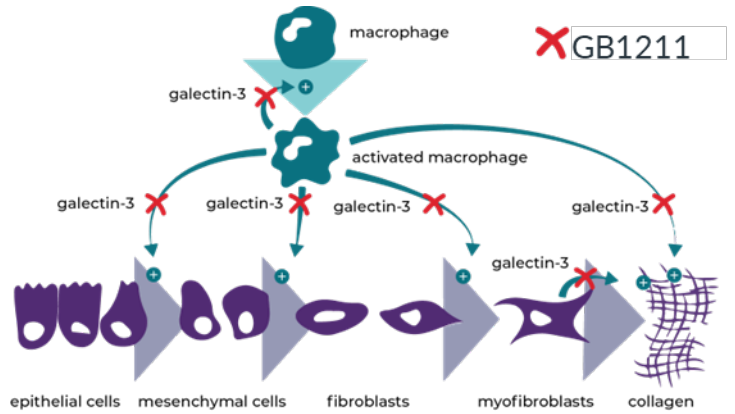
Oral Galectin-3 Inhibitor for Liver Cirrhosis

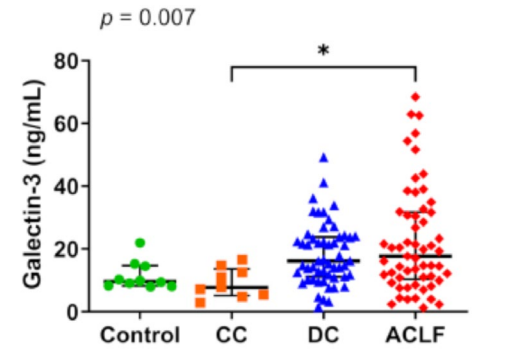
Galecto

GB1211: Oral galectin-3 inhibitor for advanced liver disease and cirrhosis

Evidence links galectin-3 to cirrhosis progression

- Galectin-3 is elevated in decompensated cirrhosis and in acute chronic liver failure
- Galectin-3 is a prognostic biomarker of hepatocellular carcinoma, a known complication of liver cirrhosis
- Data suggests galectin-3 inhibition may address cirrhosis:
  - Inhibition of galectin-3 reduces development of liver inflammation and fibrosis
  - Galectin-3 is required for TGF-ß mediated myofibroblast activation and matrix production in liver fibrosis
  - Pre-clinical and clinical evidence for reduction in transaminases by galectin-3 inhibitors suggesting hepatocyte protection





Serum galectin-3 (ng/mL)

Control (n= 10)	CC (n= 9)	DC (n= 54)	ACLF (n= 57)
9.66 (8.27 – 14.71)	7.85 (5.19 – 13.62)	16.18 (11.16 – 23.85)	17.69 (10.38 – 31.71)

[1] www.thelancet.com/journals/langas/article/PIIS2468-1253(19)30349-8/fulltext Sepanlou, et al Lancet Gastroenterol Hepatol 2020; 5: 245–66

[2] www.hepatitis.va.gov/cirrhosis/background/stages.asp

[3] Cervantes-Alvarez et al., 2022

GULLIVER-2 – Part 2

A randomized, double blind, placebo-controlled 12-week study in Child-Pugh B patients

GULLIVER-2

Part 2: Repeat dose hepatic impairment study (Child-Pugh B)

GB1211 100mg BID (n=15)

Placebo BID (n=15)

Biochemistry on Day 1, 7, 42, and follow up Day 96
PK samples on Day 1, 7, 21, 42, 63, 84

Primary endpoints:

- Safety and tolerability
- PK

Exploratory endpoints

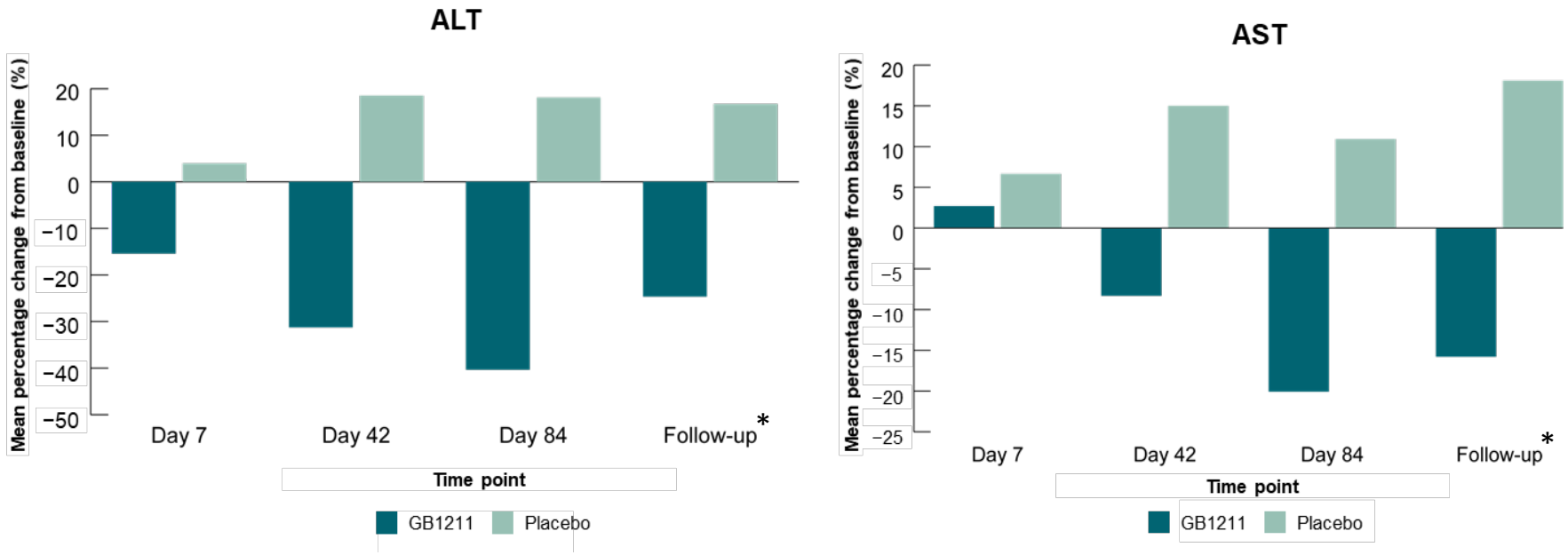
- Biochemistry
- Liver fibrosis (VCTE)
- Steatosis (CAP)
- Exploratory biomarkers

Part 1: Single dose hepatic impairment study (Child-Pugh B) → Part 3: Single dose hepatic impairment study (Child-Pugh C)

• BID, twice a day; CAP, controlled attenuation parameter; PK, pharmacokinetics; VCTE, vibration controlled transient elastography

Liver-related biochemistry results

Consistent and increasing reduction in liver enzymes for GB1211 patients

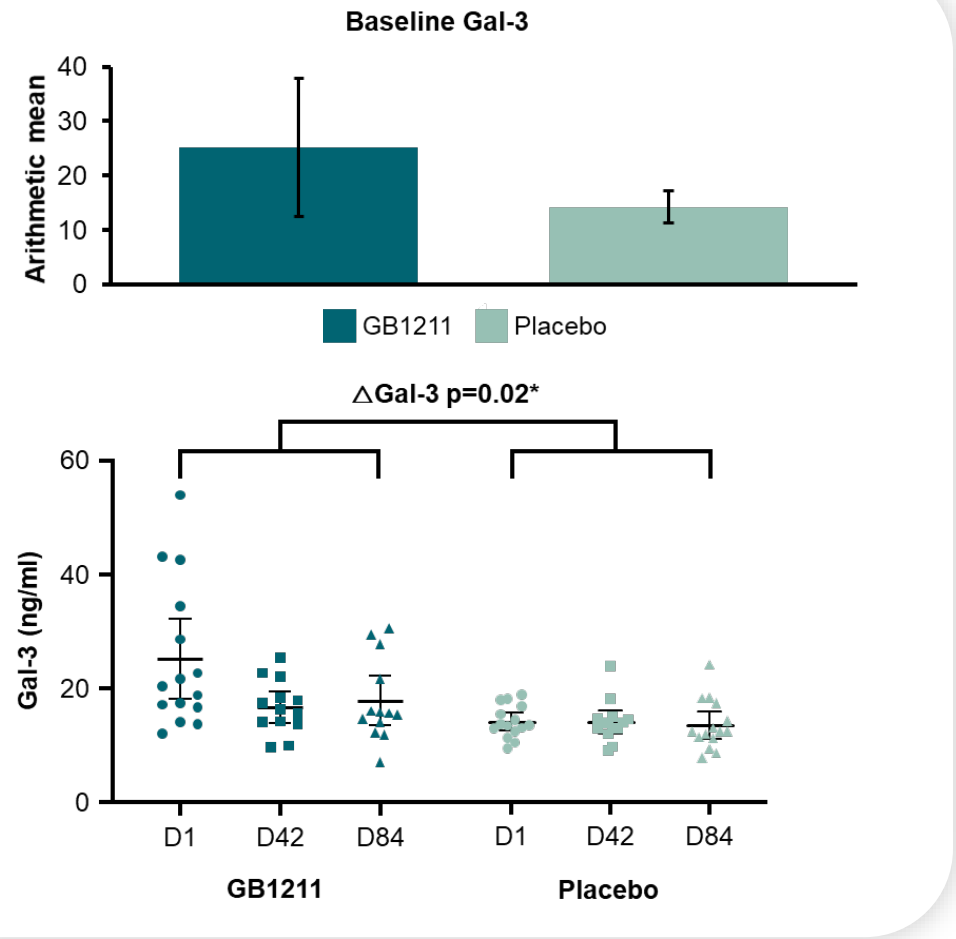


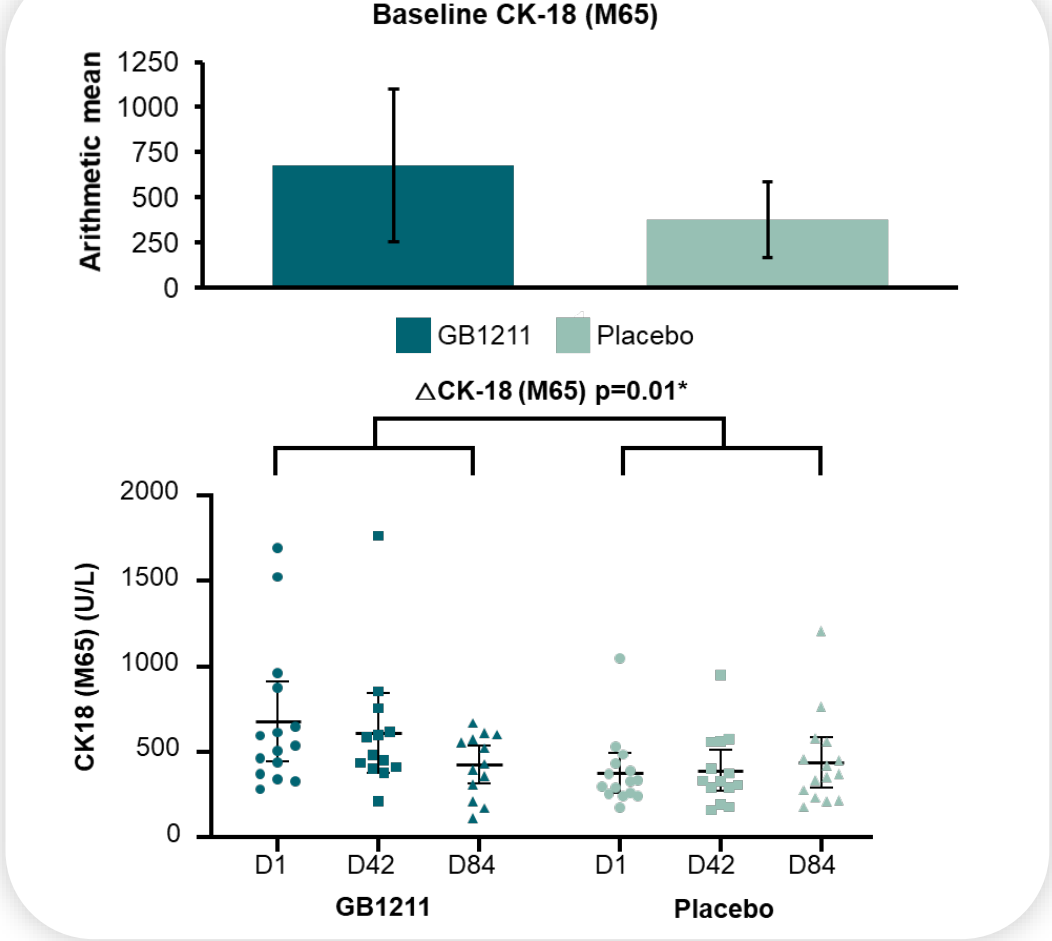
Treatment effect (GB1211-Placebo) [%] at Day 84	ALT	AST	GGT	ALP
Mean	-58.44	-32.40	-37.77	-14.76
95% confidence interval	(-79.00, -37.88)	(-51.63, -13.17)	(-69.47 , -6.06)	(-31.92 , -2.40)
p-value	0.0001	0.002	0.0214	0.0889

*Follow up took place two weeks after the last dose. ALT: alanine transferase; AST: aspartate transferase No adverse changes in standard safety laboratory parameters, including bilirubin albumin, or INR

GB1211 reduces galectin-3 and CK-18 (M65)

GB1211 demonstrated target engagement and potential anti-apoptotic properties





*p value from unpaired t-test. Δ: change; CK-18: cytokeratin-18; D: day; Gal-3: galectin-3

GULLIVER-2 - topline results

Promising study results in a decompensated cirrhosis patient population

Galectin-3 in liver disease

- Carbohydrate binding protein shown to drive fibrosis via TGF-ß receptor
- Elevated in decompensated cirrhosis, alcoholic hepatitis and ACLF
- GB1211 is a potent, selective, oral inhibitor of Galectin-3

Well-tolerated

- GB1211 was well-tolerated with no drug-related adverse events identified
- Predictable PK profile consistent with the option of repeated dosing in patients with hepatic impairment

Clinical

- Galectin-3 reduction demonstrates target engagement
- Consistent and statistically significant reductions in ALT, AST and GGT
- Concordant changes in liver biochemistry, liver stiffness & steatosis observed
- Data suggests that GB1211 improves liver inflammation and reduces liver injury

Results strongly support progressing to phase 2/3 studies in severe liver diseases

Summary

Galecto

Galecto enhances pipeline following strategic review

Cash balance of ~$22.9M as of June 30, 2024 funds ongoing trials and acquired program with runway into 2026

Completed Strategic Review Identifies Core Assets and Direction

Following an intensive strategic review process, Galecto has determined to focus on

- cancer and liver disease
- leveraging its existing clinical stage asset GB1211

Pipeline is bolstered by acquisition of BRM-1420 for AML, which brings novelty and breakthrough potential

- Superior activity in preclinical AML models
- Identified after review of multiple assets, assisted by Leerink

Enhanced Pipeline Focused On Oncology and Liver Disease

Innovative platforms targeting core disease processes

- Pioneers in ENL-YEATS and galectin-3 based pharmacology
- First-in-class highly specific, oral small-molecule inhibitors

All programs address:

- Diseases characterized by clear unmet medical need
- Multi-billion-dollar market opportunities

Early Data Supportive of Drug Activity

ENL-YEATS inhibitor:

- MoA estimated to cover 30%+ of AML population
- Rapid and durable anti-tumor activity in AML models

Galectin-3 Inhibitors:

- Target engagement shown
- Positive biomarker data
- Evidence of tumor microenvironment transformation
- Significant clinical data in cirrhosis