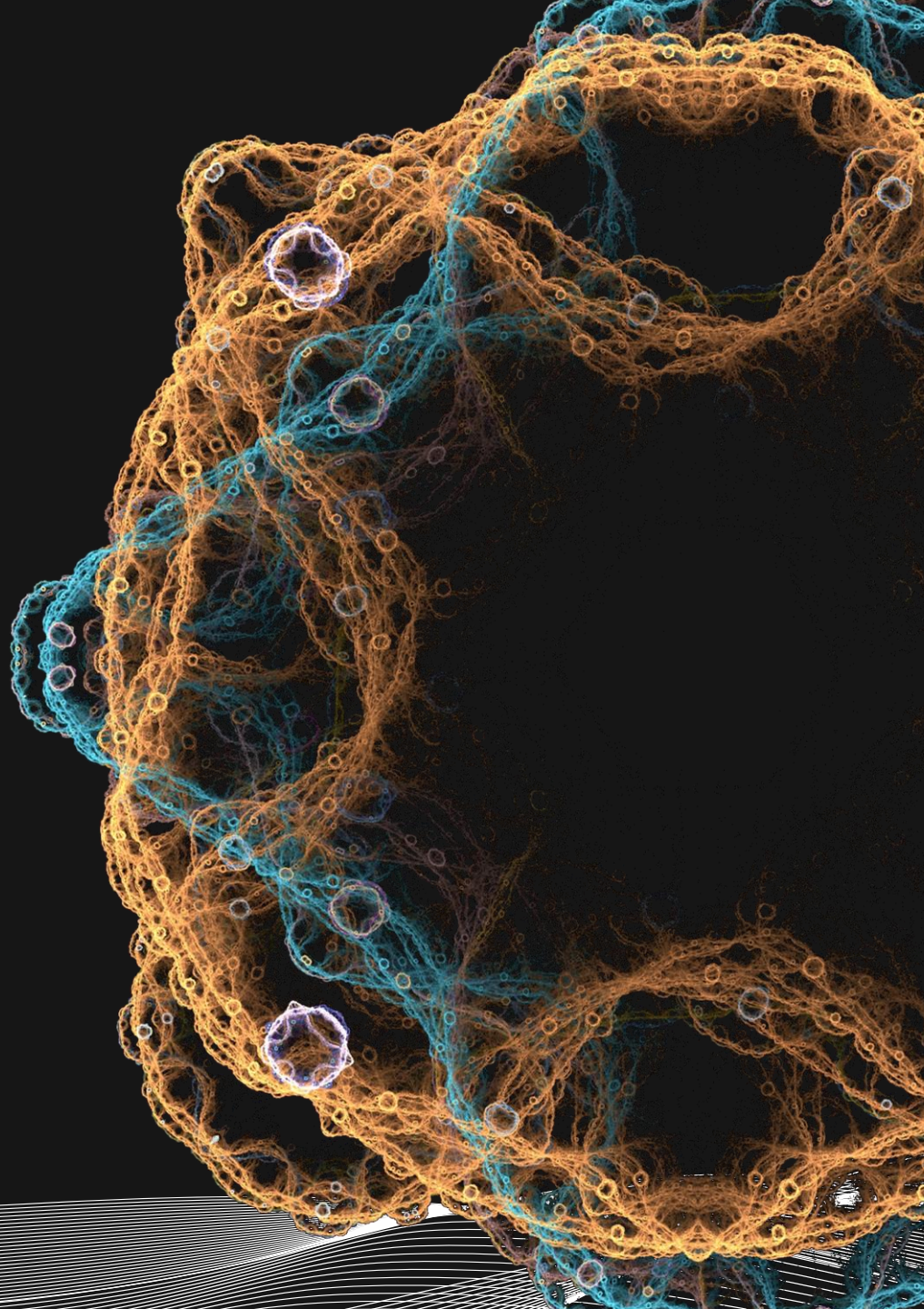
JABEZ BIOSCIENCES

A New Era of Small Molecules for Cancer

Company Overview
2025



Disclosures and Disclaimers

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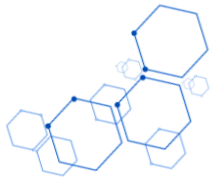
Potential acquirers, investors, or lenders must conduct their own reviews and satisfy themselves in terms of the Business and its prospects for the future. The Directors accept no liability for any loss or damage whatsoever which may occur as a result of reliance on the information in this document.

Company Overview

Jabez Biosciences is a **clinical-stage biotechnology company** that is developing small molecule inhibitors for the treatment of both solid and liquid cancers



JBZ-001:
Our Next-Gen DHODH Inhibitor

Novel, potent, and potentially best-in-class inhibitor of DHODH that displays wide range of therapeutic activity

Clinical Development:
Phase I Clinical Trial in NHL + Solid tumors

We are actively pursuing clinical investigations in various solid and liquid cancer indications.

The Future:
Strategic Combination Therapies

The future of cancer therapy is strategic combinations. Our lead program synergizes with many FDA approved therapies.

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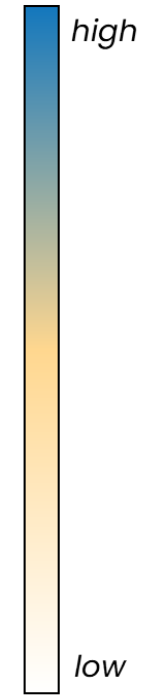
Lead Program
JBZ-001

DHODH INHIBITORS

- First and second-line treatment
- Multitude of indications
- Fast track and orphan designations
- Stable oral dosage form
- Developed by DDI at OSU Cancer Center
- Large patent portfolio

**Enzyme inhibition*

Developer	Platform	IC_{50} (nM)
JABEZ BIOSCIENCES	JBZ-001	0.95
Bayer	BAY	0.97
Servier	A-636	3.38
ASLAN Pharmaceuticals	ASLAN003	3.91
Sanofi	Teriflunomide	26.45
Sanofi	Leflunomide	208.5
PTC Therapeutics	PTC299	686.5



Elgamal and Fobare, et al., JCI Insights 2024

Lead Program
JBZ-001

Designed Mechanism OF ACTION

Exploiting cancer's deregulated metabolism

The drug candidate JBZ-001 binds to and inhibits the rate-limiting enzyme involved in the synthesis of pyrimidine nucleotides, DHODH. Since cancer cells rely on vast quantities of nucleotides for fast growth, they are exquisitely sensitive to DHODH inhibition.

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Lead Program

JBZ-001

Superior Potency

Exhibits a stronger ability to inhibit DHODH compared to other prominent compounds.

Optimized Pharmacokinetics

Orally-bioavailable and has a very long half-life of 29 hours in circulation, enabling convenient dosing.

Enhanced Safety Profile

Has a wider therapeutic window, allowing for potentially greater efficacy without compromising safety.

Efficient Manufacturing

Utilizes the well-established Suzuki Reaction for synthesis, enabling scalable and cost-effective production.

Variety of Powerful Therapeutic Effects

Strong preclinical data suggests JBZ-001 harbors a range of therapeutically relevant mechanisms of action.

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JBZ-001 vs. Leading Competitors

	JABEZ BIOSCIENCES	BAYER	ASLAN PHARMACEUTICALS	CLEAR CREEKBIO	ZENSHINE	genase THERAPEUTICS BV
Drug Candidate	JBZ-001	BAY-2402234	ASLAN-003	BREQUINAR	ZX-9021	GTX-0196
Indication(s)	Solid Tumors, NHL, MM, AML	AML	Alopecia	AML, COVID-19	Solid Tumors	Hematologic Malignancies
Development Stage	Phase I	Inactive (Phase I)	Phase II	Inactive (Phase II)	Preclinical	Preclinical
Combination Strategy	Std of Care; Targeted Tx	-	-	-	-	Undisclosed
Biochemical potency (preferred IC50 <20 nM)						Published Data
Low Cellular Shift (preferred ≤15x)						Published Data
AML MOLM-13 xenograft efficacy (QD, PO)	+38 days (10 mg/kg)	+35 days (4mg/kg)	+24 days (50 mg/kg)	+48 days (10 mg/kg)		
IP Strength						

Provided by: The Ohio State University Center for Innovation Strategies

Good Intermediate Poor Not Determined

Understanding Our DHODH Inhibitor

Therapeutic Mechanisms of Action of JBZ-001

On top of the expected nucleotide depletion-induced cell stress, our DHODH inhibitor, JBZ-001, displays multiple mechanisms of action, opening up the possibilities to be used in **combination** with currently approved therapies.

JBZ-001's Therapeutic Potential

- Metabolic Disruption
- Myeloid Cell Differentiation
- Increased CD38 Expression
- Increased CD47 Expression

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Metabolic Disruption

Myeloid Cell Differentiation

Increased CD38 Expression

Increased CD47 Expression

Biological Effect

Widescale Metabolic Disruption

JBZ-001 inhibits the *de novo* synthesis of pyrimidine nucleotides shutting down the cell's ability to produce these essential building blocks. This induces significant metabolic stress leading to widespread cellular dysfunction and ultimately, cell death.

Therapeutic Implication(s)

Excellent Single-Agent and Combination Therapy Potential

Not only does JBZ-001 display potent single-agent activity against **various preclinical cancer models**, but our preclinical data also suggests it synergizes effectively with other approved treatments, offering a powerful combination strategy.

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Metabolic Disruption

Myeloid Cell Differentiation

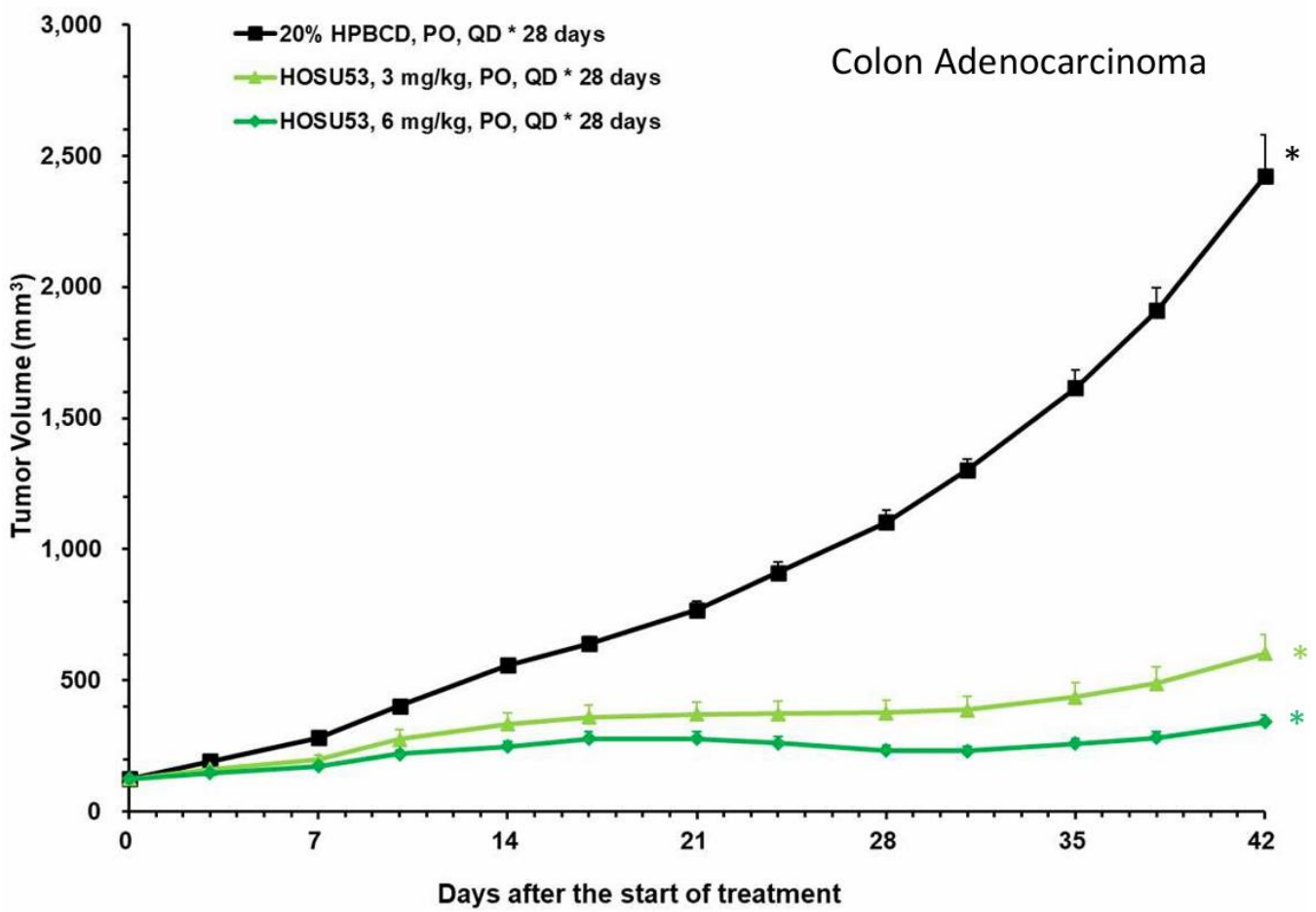
Increased CD38 Expression

Increased CD47 Expression

SINGLE-AGENT

JBZ-001 in model of **CRC**

- Potent **single-agent** efficacy
- Proof of concept data to support JBZ-001's single-agent activity in **solid tumors**



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Metabolic Disruption

Myeloid Cell Differentiation

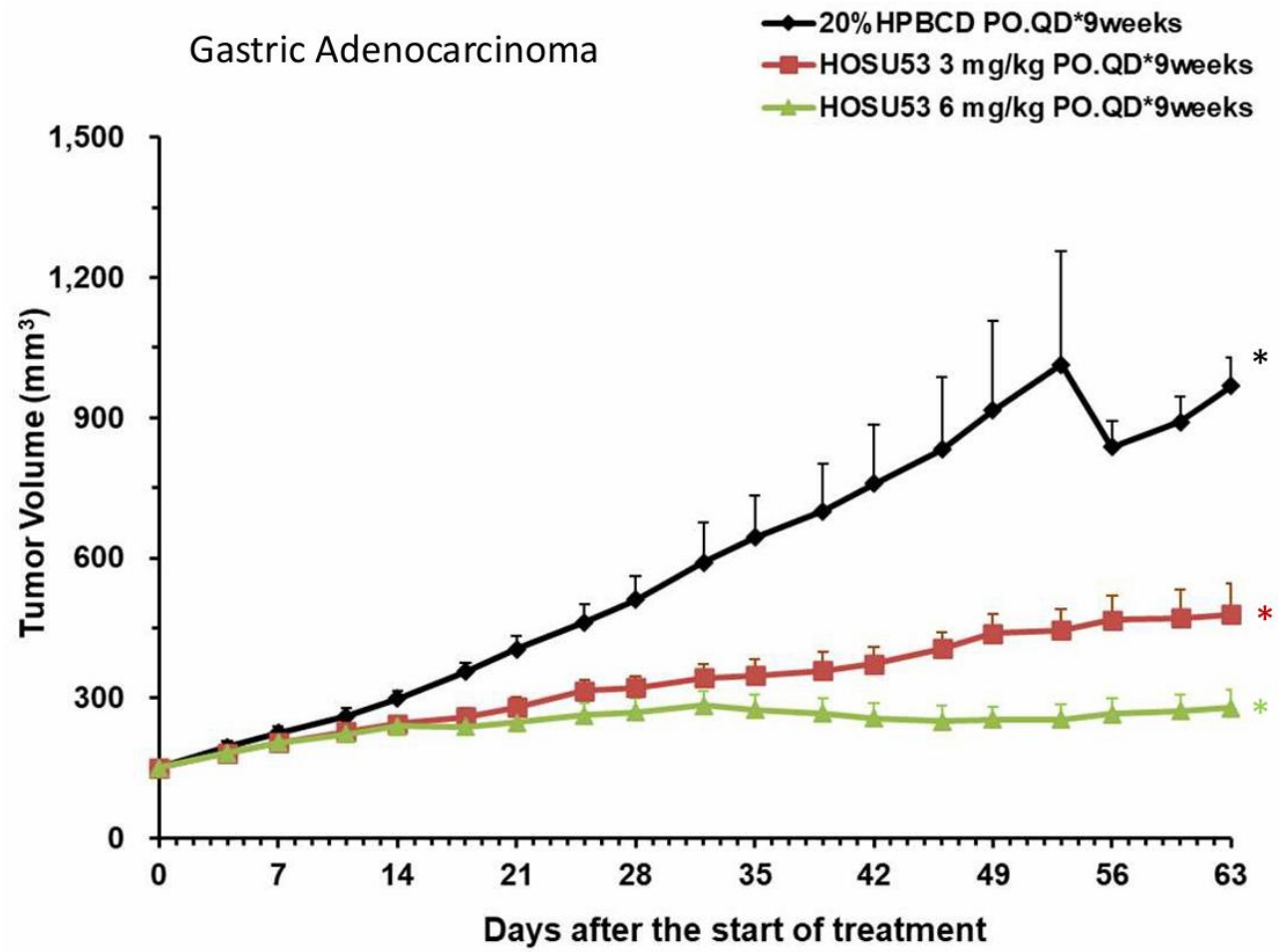
Increased CD38 Expression

Increased CD47 Expression

SINGLE-AGENT

JBZ-001 in model of **GA**

- Potent **single-agent** efficacy
- Further proof of concept data to support JBZ-001's single-agent activity in **solid tumors**



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Metabolic Disruption

Myeloid Cell Differentiation

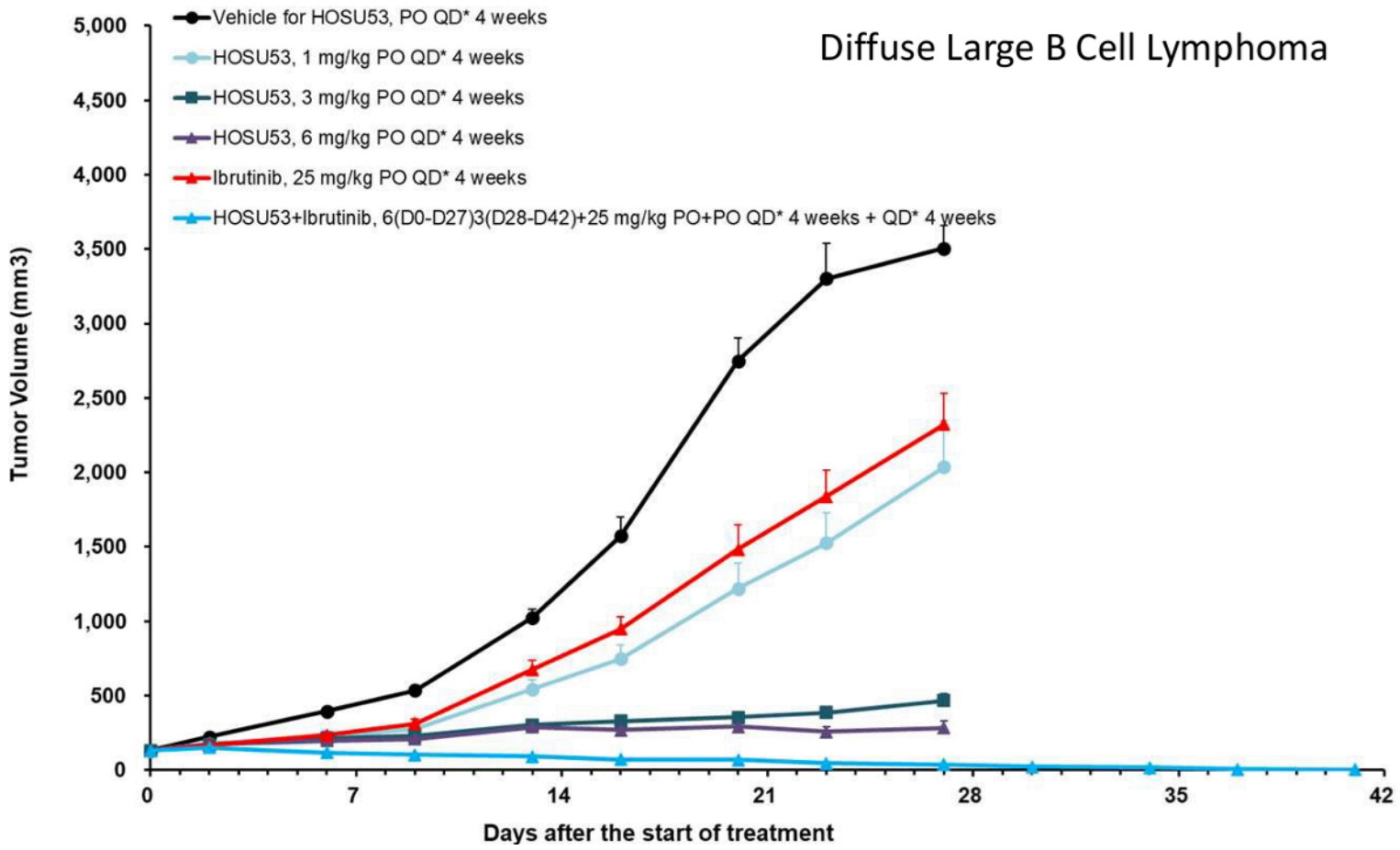
Increased CD38 Expression

Increased CD47 Expression

COMBO THERAPY

JBZ-001 in model of **NHL**

- Potent **single-agent** efficacy
- Significantly enhances therapeutic activity of BTK inhibitor **Ibrutinib** (J&J/Abbvie)
- Clear ability to augment therapy in NHL and potential to transform patient outcomes in this indication



Ibrutinib is an irreversible inhibitor of BTK (Bruton's tyrosine kinase). Developed by J&J/Abbvie **$5B in 2019**

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Metabolic Disruption

Myeloid Cell Differentiation

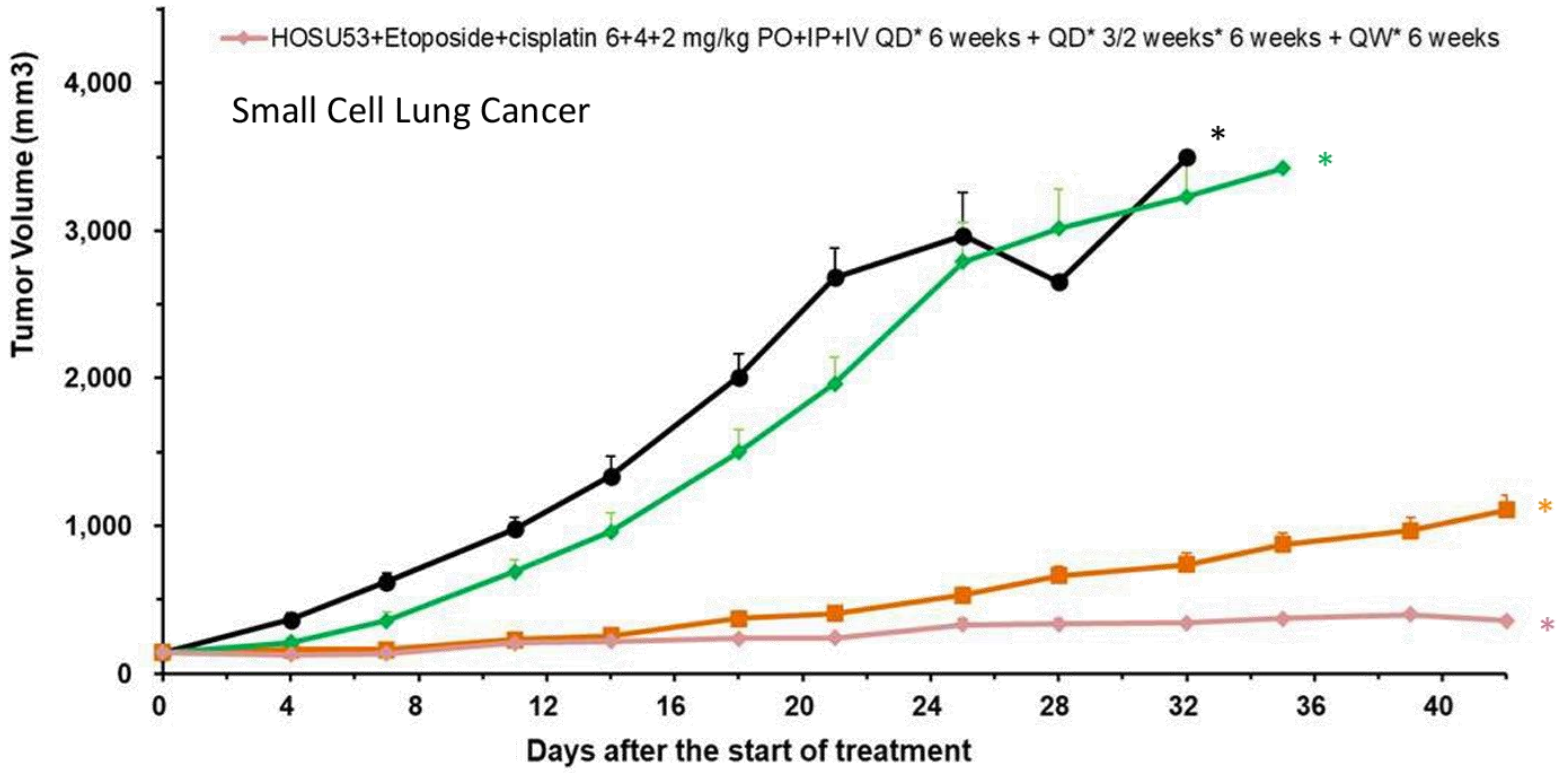
Increased CD38 Expression

Increased CD47 Expression

COMBO THERAPY

JBZ-001 in model of **SCL**

- Potent **single-agent** efficacy
- Significantly enhances therapeutic activity of conventional chemotherapy (Etop. and Cis.
- Clear ability to augment SOC therapy in solid tumors; implications across wide array of indications.



*SOC = Standard of Care

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Metabolic Disruption

Myeloid Cell Differentiation

Increased CD38 Expression

Increased CD47 Expression

Biological Effect

Myeloid Cell Differentiation

JBZ-001 induces the differentiation of immature myeloid cells, driving them toward a more mature, functional state. This is evidenced by morphological changes in AML cells (shown below) and enhanced phagocytic activity in the THP-1 myeloblast cell line, both hallmark indicators of cellular differentiation.

Therapeutic Implication(s)

Targeted Therapy for AML & Combination w/ Immunotherapies

By promoting the differentiation of immature myeloid cells, JBZ-001 holds significant therapeutic potential for **treating myeloproliferative disorders such as AML**. Also, this can suppress MDSC activity, augmenting several forms of immunotherapy.

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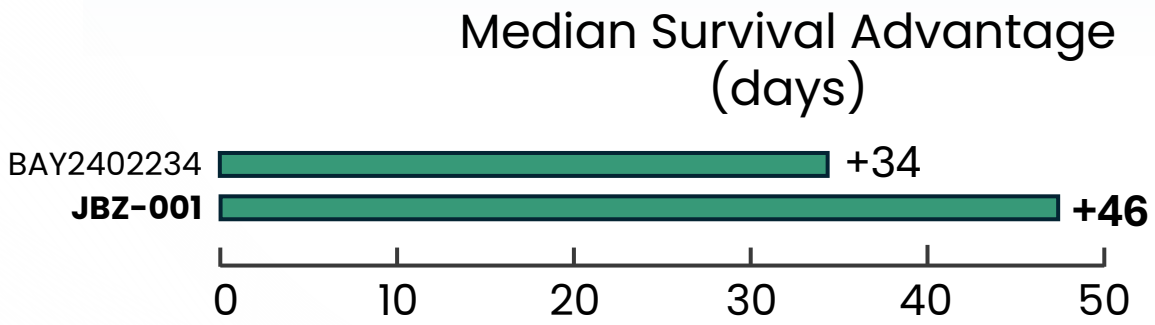
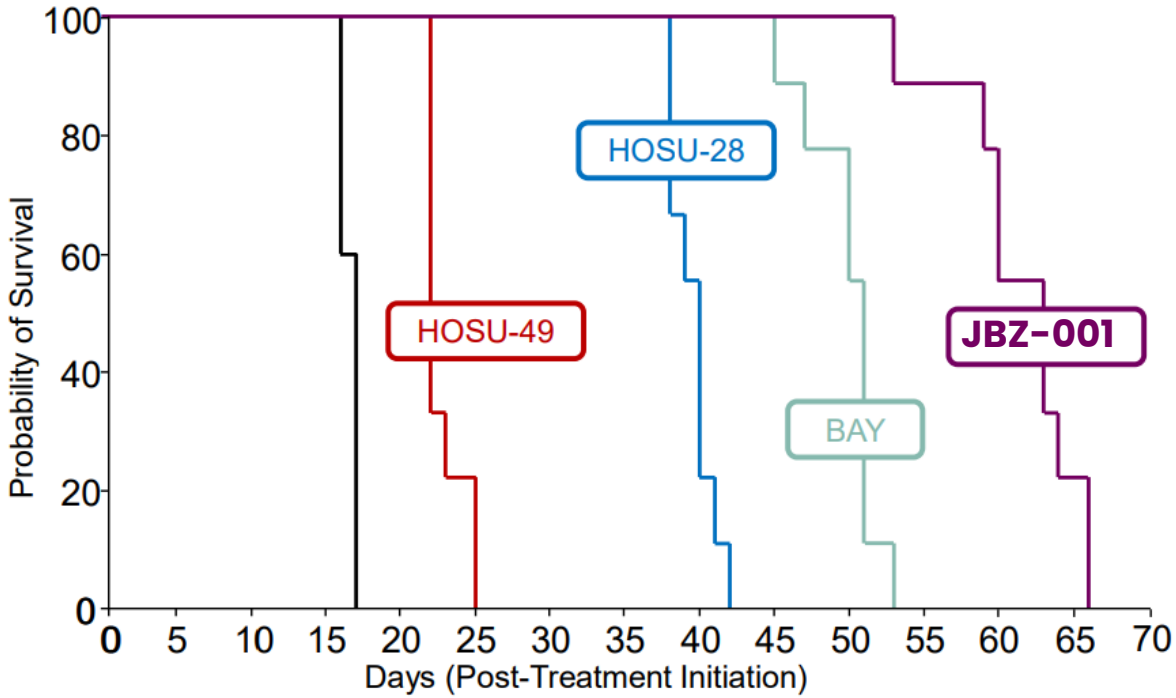
Metabolic Disruption | Myeloid Cell Differentiation | Increased CD38 Expression | Increased CD47 Expression

SINGLE-AGENT

JBZ-001 Displays Potent **Single-Agent** Activity in MOLM-13 AML Model

- Single-agent treatment with JBZ-001 (10mg/kg – purple line) displays potent anti-tumor activity in xenograft model of **AML**
- Single-Agent JBZ-001 displayed superior therapeutic activity than competing compounds including Bayer's DHODH inhibitor, BAY (green line)





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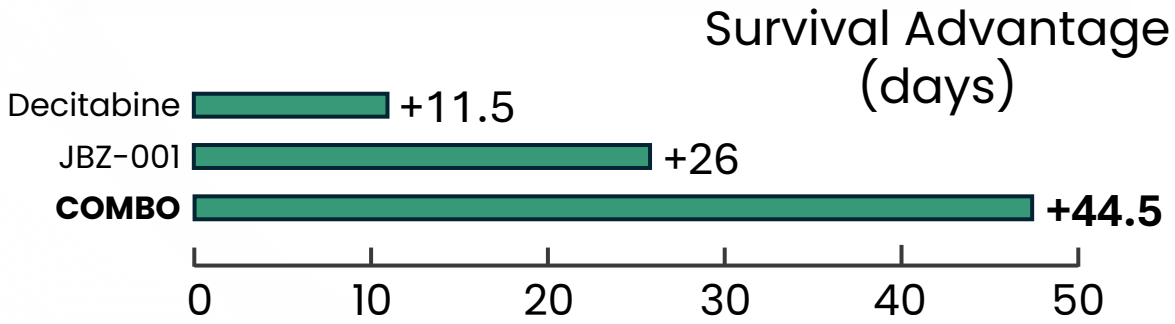
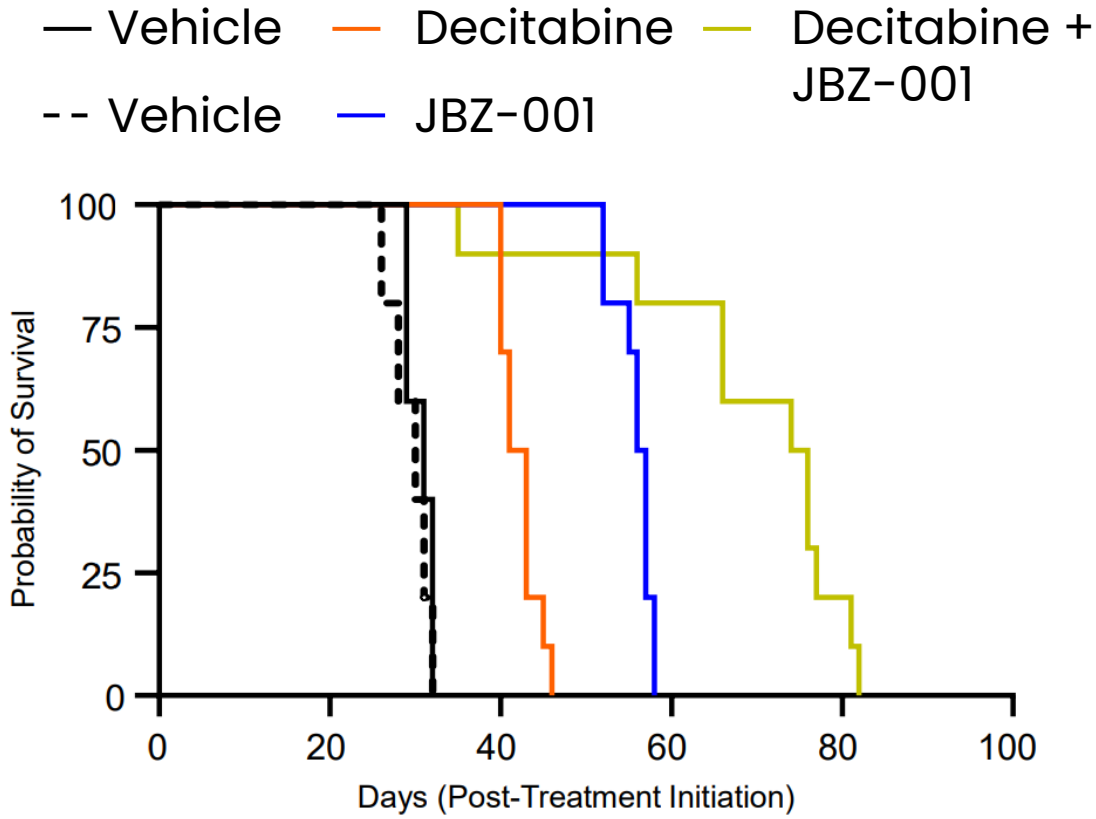
Metabolic Disruption | Myeloid Cell Differentiation | Increased CD38 Expression | Increased CD47 Expression

COMBO THERAPY

JBZ-001 **Synergizes with Decitabine** in HL-60 (p53-mut) xenograft of AML

- JBZ-001 alone displays superior therapeutic activity than Decitabine in p53-mutant AML model (HL-60).
- Combination therapy with JBZ-001 and Decitabine displays robust anti-tumor activity, exceeding either single-agent alone.





JABEZ BIOSCIENCES

Metabolic Disruption

Myeloid Cell Differentiation

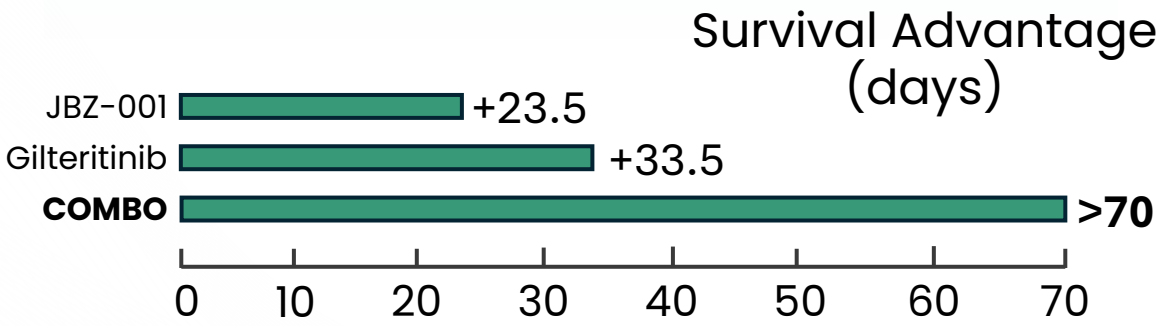
Increased CD38 Expression

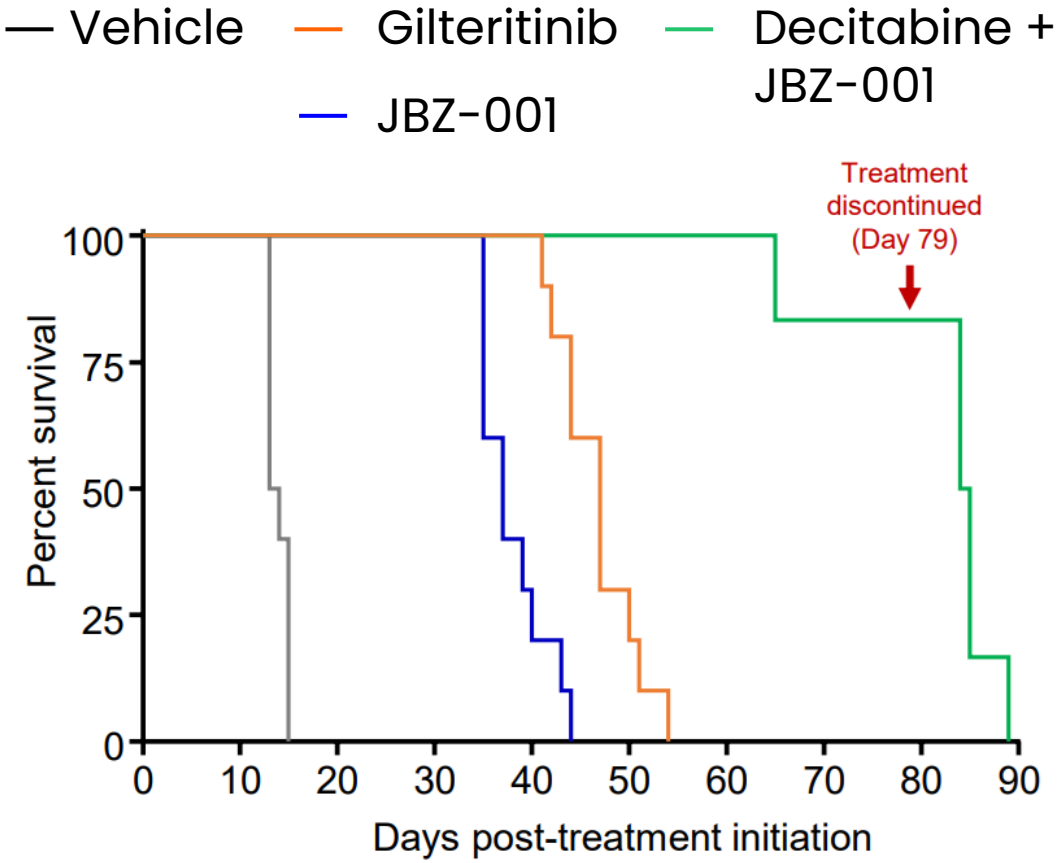
Increased CD47 Expression

COMBO THERAPY

JBZ-001 **Synergizes with Flt3 Inhibitor Gilteritinib** in aggressive AML model

- JBZ-001 boosts therapeutic activity of Flt3 inhibitor Gilteritinib treatment in aggressive model of AML.
- Provides strong preclinical proof-of-concept for use of **JBZ-001 in AML in combination with standard of care therapies**.





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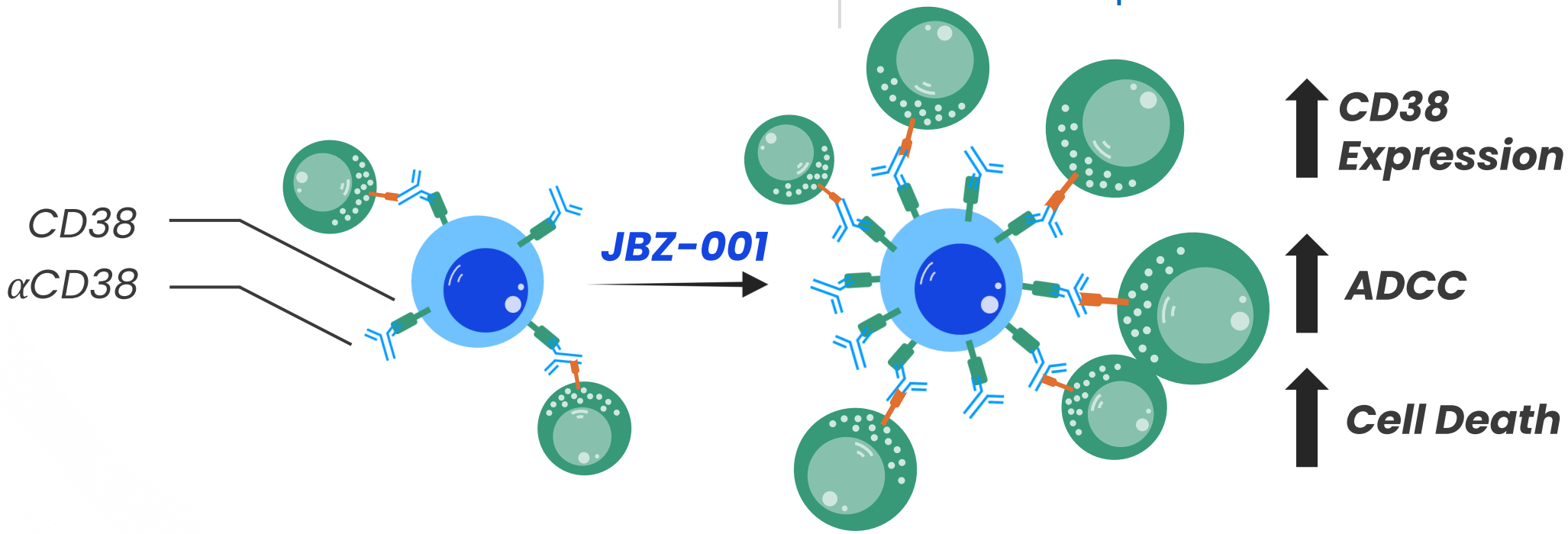
Metabolic Disruption | Myeloid Cell Differentiation | **Increased CD38 Expression** | Increased CD47 Expression

Biological Effect

Increased CD38 Expression on Surface of Cancer Cells

Therapeutic Implication(s)

Potent Synergy w/ CD38-Targeting Therapies such as αCD38 mAbs

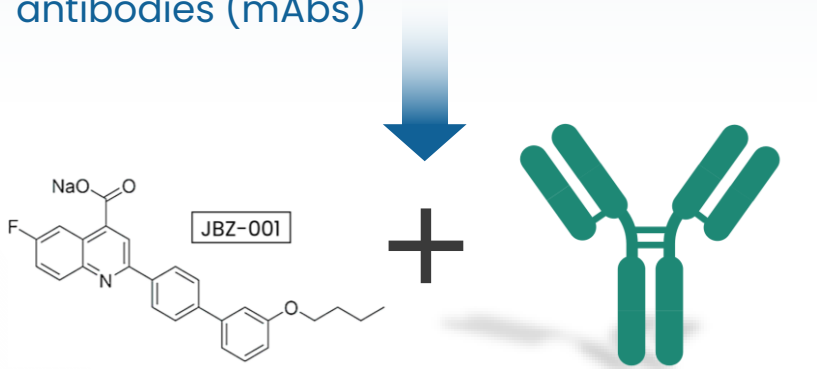


JABEZ BIOSCIENCES

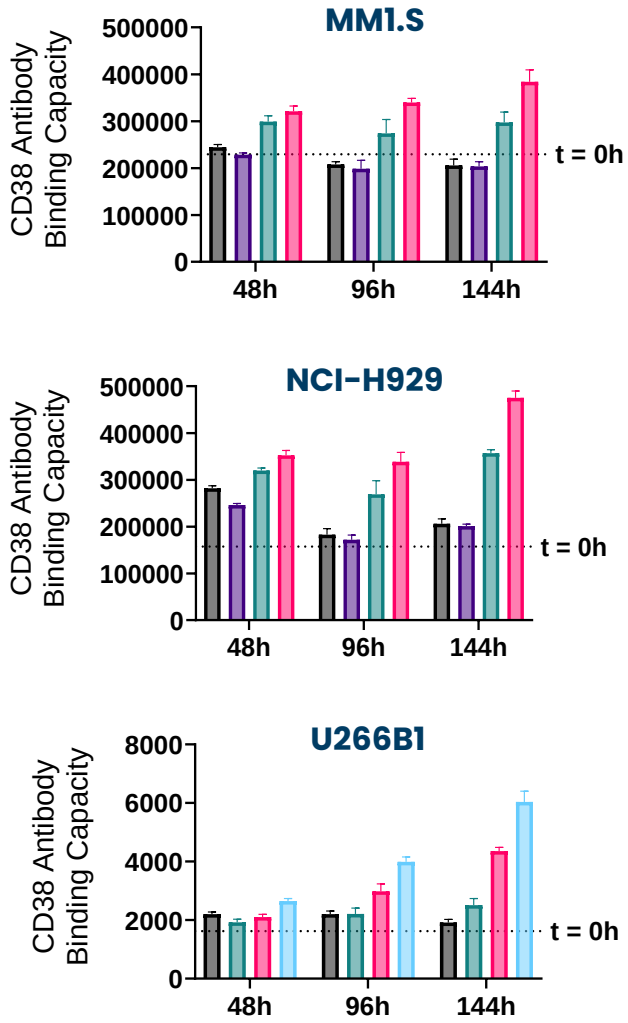
Metabolic Disruption | Myeloid Cell Differentiation | **Increased CD38 Expression** | Increased CD47 Expression

JBZ-001 Increases CD38 Expression

- Treatment with low dose (10-100nM) JBZ-001 significantly **increases surface expression of CD38** on various human MM cell lines
- Suggests rational **combination with αCD38 targeting therapies** such as monoclonal antibodies (mAbs)



JBZ-001 + αCD38 mAb



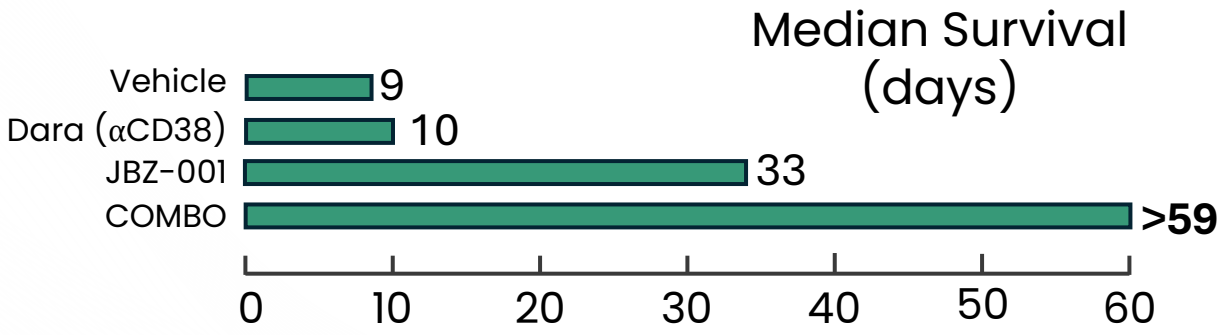
JABEZ BIOSCIENCES

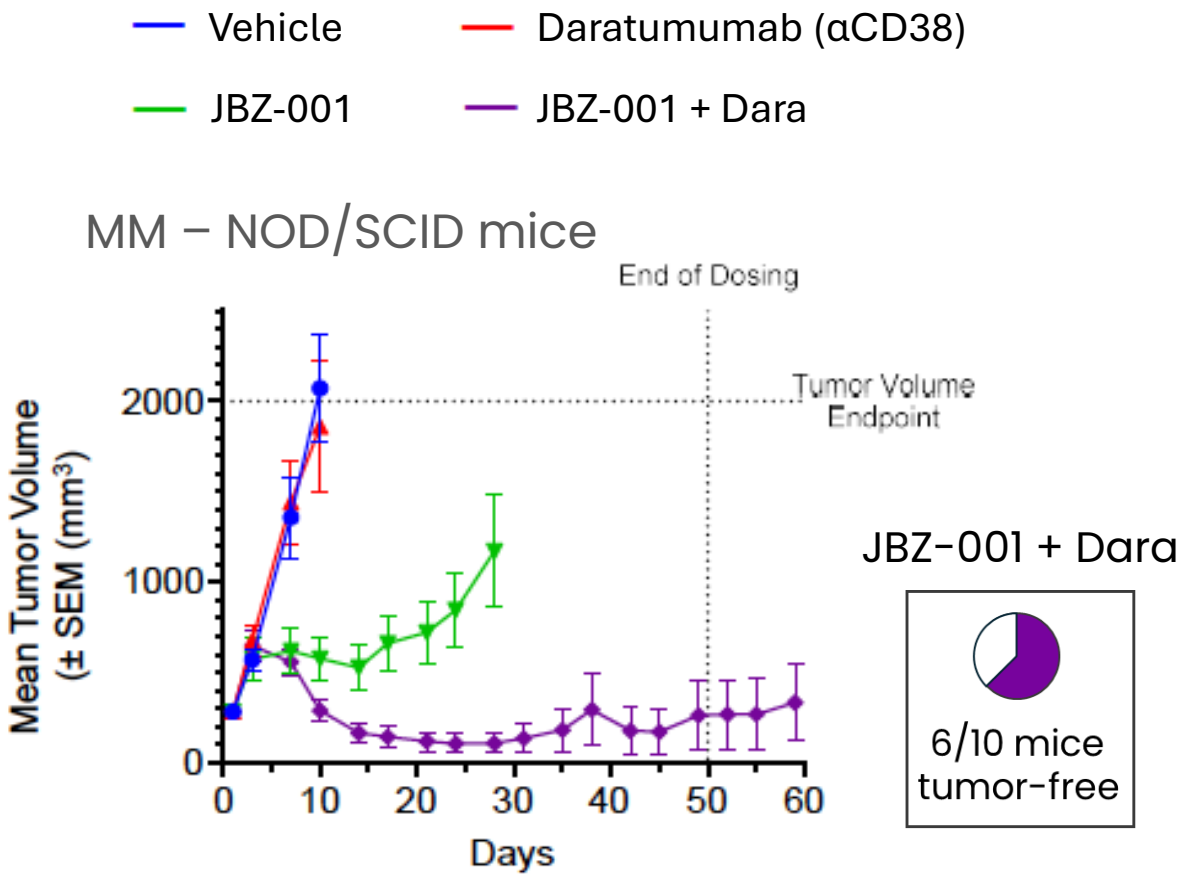
Metabolic Disruption | Myeloid Cell Differentiation | **Increased CD38 Expression** | Increased CD47 Expression

COMBO THERAPY

JBZ-001 + αCD38 mAb (Daratumumab)

- Single-agent treatment with JBZ-001 (10mg/kg – green line) displays potent anti-tumor activity in xenograft model of **Multiple Myeloma**
- JBZ-001 synergizes with αCD38 mAb **Daratumumab**, resulting in extremely potent anti-tumor activity (purple line).





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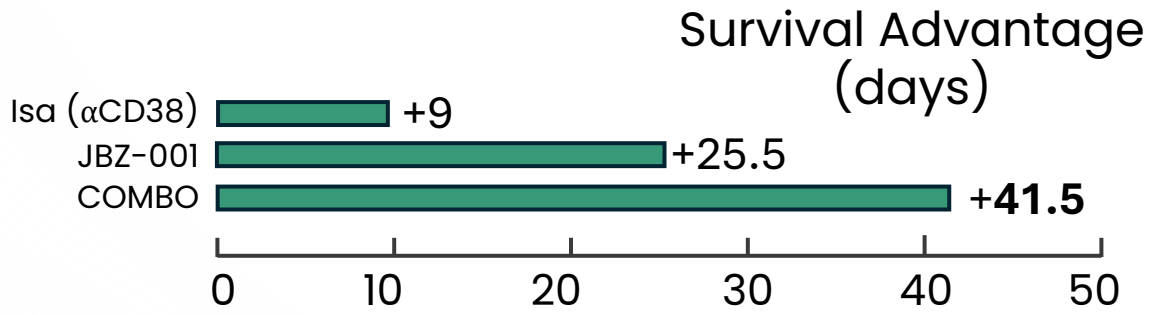
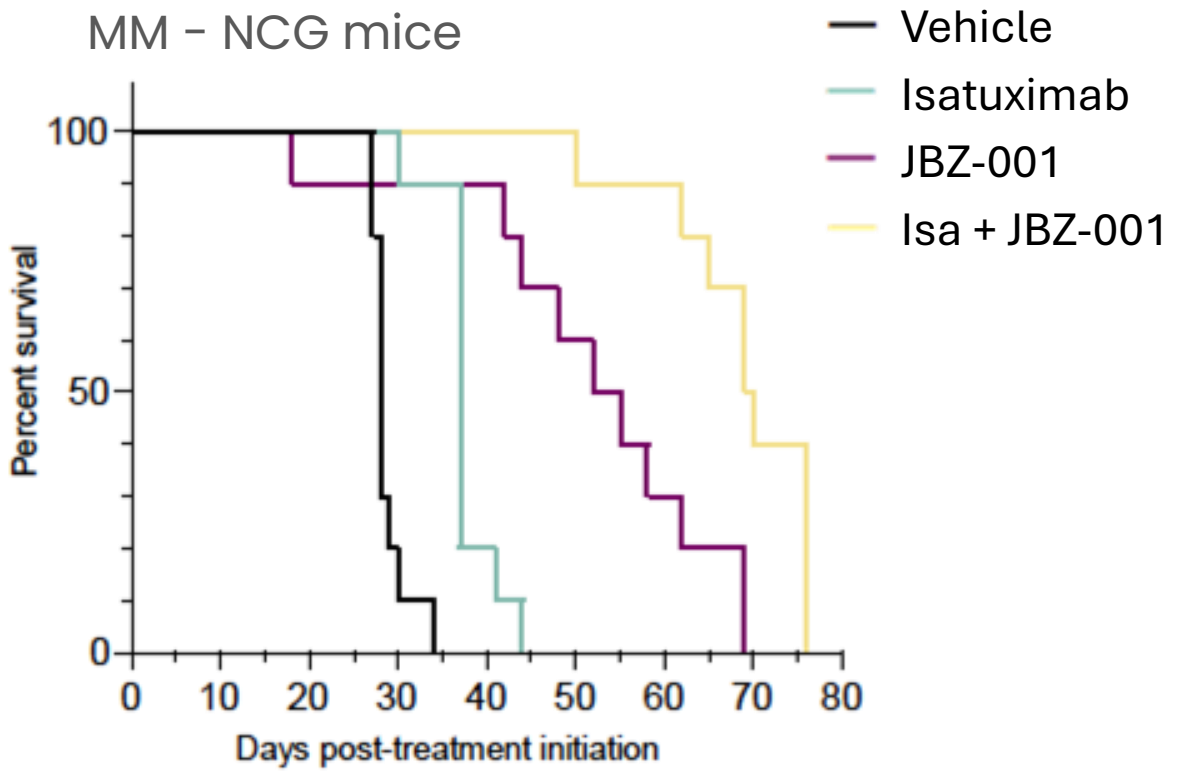
Metabolic Disruption | Myeloid Cell Differentiation | **Increased CD38 Expression** | Increased CD47 Expression

COMBO THERAPY

JBZ-001 + αCD38 mAb (Isatuximab)

- Single-agent treatment with JBZ-001 (10mg/kg – green line) displays potent anti-tumor activity in xenograft model of **Multiple Myeloma**
- JBZ-001 synergizes with αCD38 mAb **Isatuximab**, resulting in superior survival in preclinical model.





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Metabolic Disruption | Myeloid Cell Differentiation | **Increased CD38 Expression** | Increased CD47 Expression

Combination Therapy Targets

janssen
DARZALEX®
(daratumumab)

$9.7B USD in 2023†

- 22% increase from 2022†
- Projected to hit $14.7B by 2030

sanofi
SARCLISA®
(isatuximab-irfc)

$412M USD in 2023

- 37% increase from 2022†

*According to Bloomberg Intelligence
†According to Fierce Pharma

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Metabolic Disruption

Myeloid Cell Differentiation

Increased CD38 Expression

Increased CD47 Expression

Biological Effect

Therapeutic Implication(s)

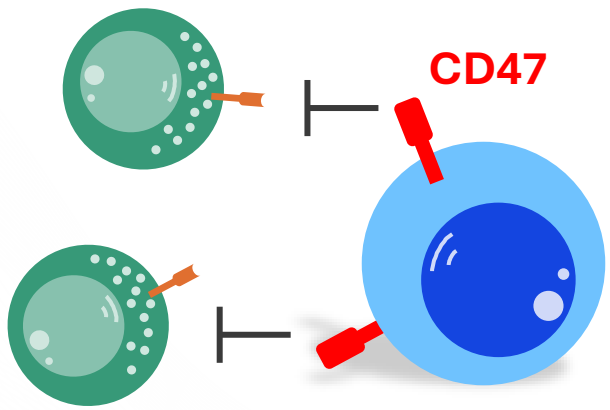
Increased CD47 Expression on Surface of Cancer Cells

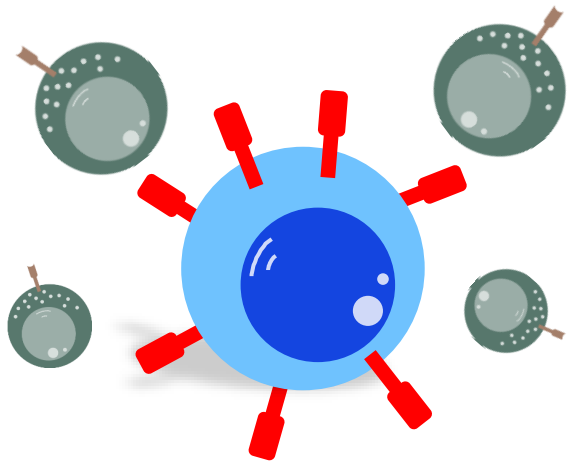
Potent Synergy w/ CD47-Targeting Therapies such as αCD47 mAbs

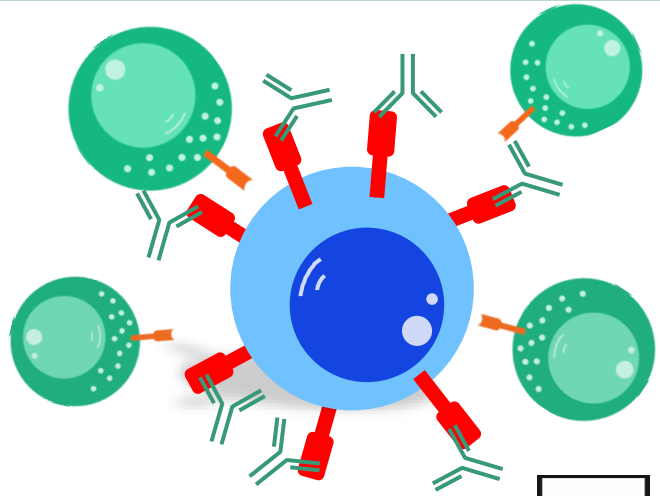
CD47 blocks anti-cancer activity of several immune cell types

Upregulation of CD47 may be causing immune evasion

CD47 block may enhance immune-mediated killing





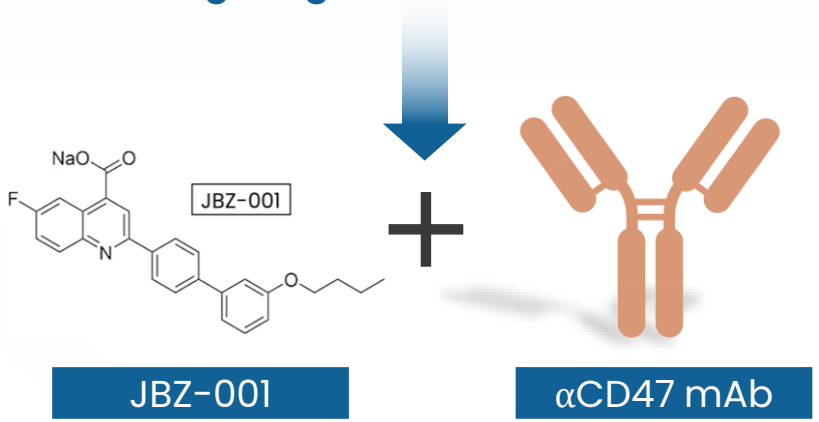


JABEZ BIOSCIENCES

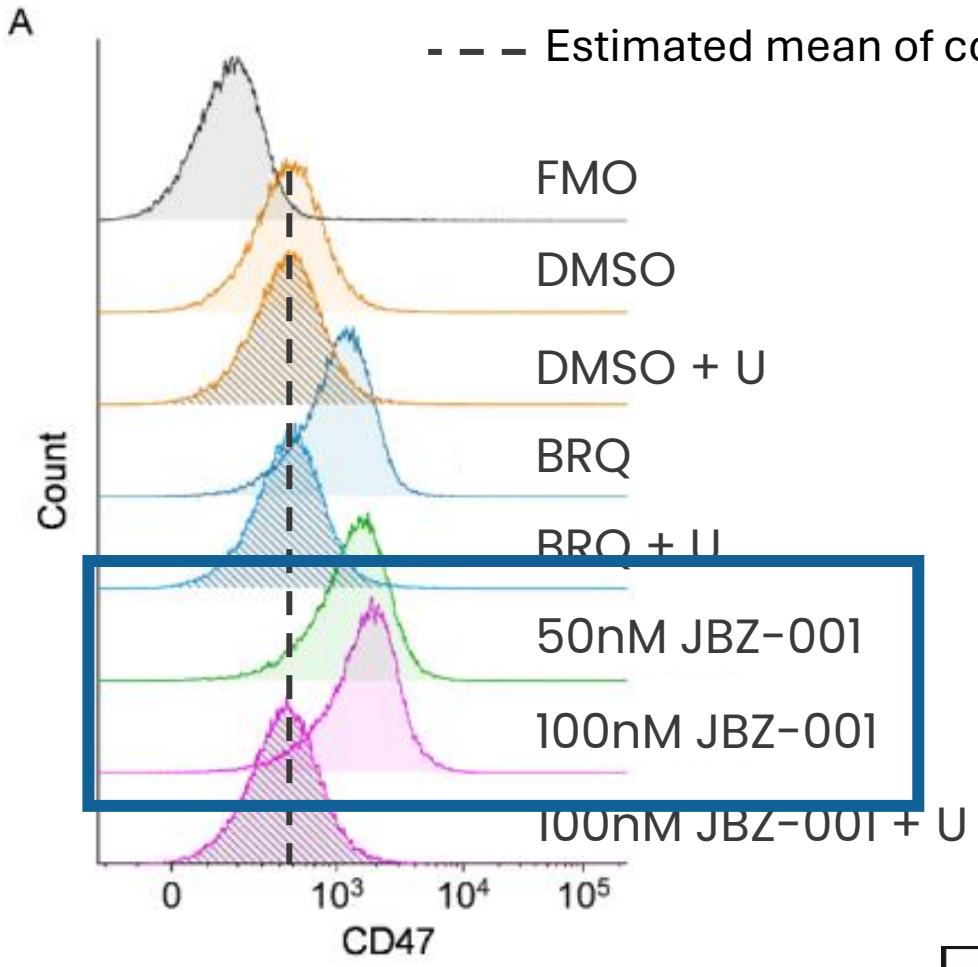
Metabolic Disruption | Myeloid Cell Differentiation | Increased CD38 Expression | **Increased CD47 Expression**

JBZ-001 Increases CD47 Expression

- Treatment with low dose (10-100nM) JBZ-001 significantly **increases surface expression of CD47** on human AML cell line.
- CD47 upregulation may be causing immune evasion; suggests rational **combination with αCD47 targeting mAbs**



JBZ-001 + αCD47 mAb

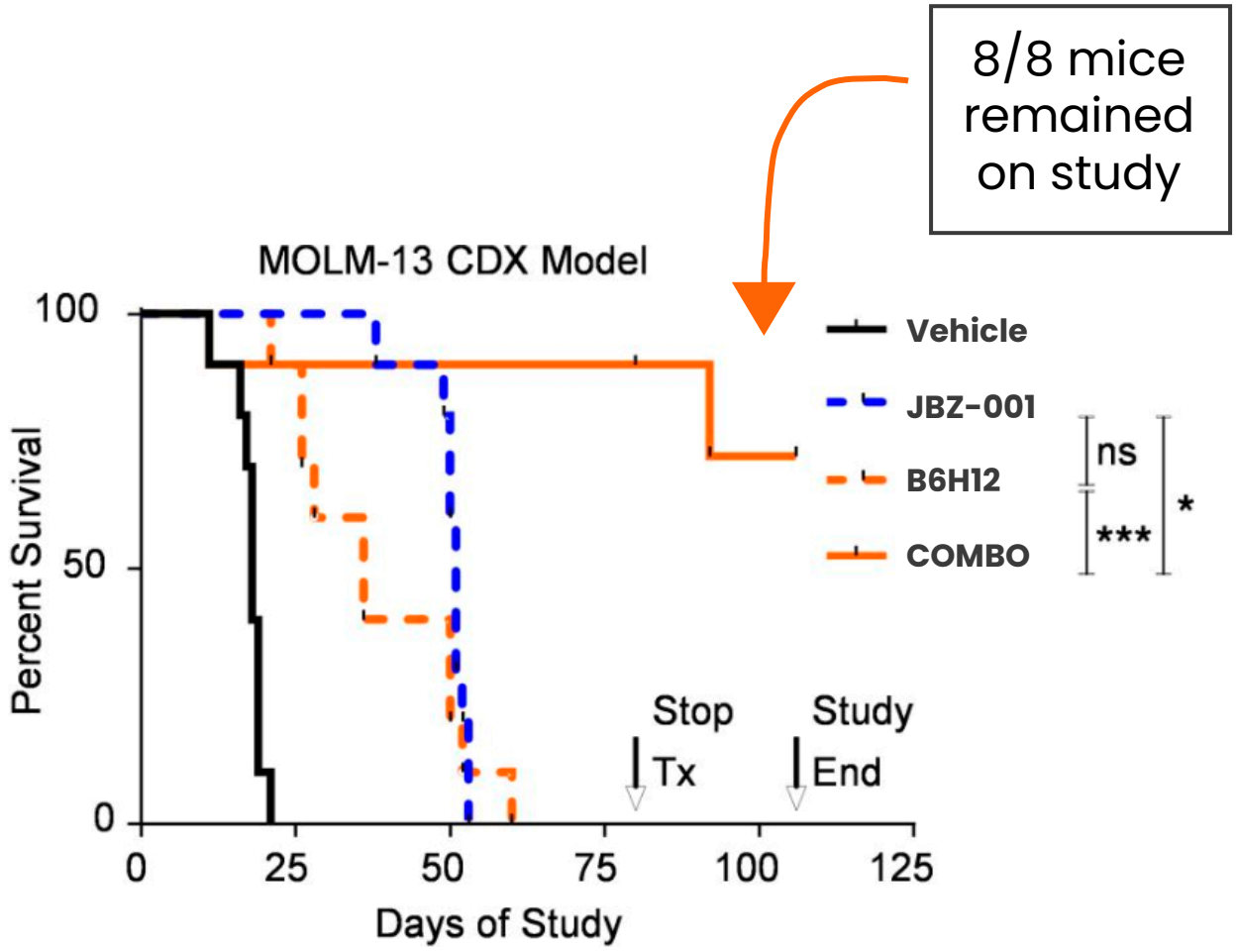


JABEZ BIOSCIENCES

Metabolic Disruption | Myeloid Cell Differentiation | Increased CD38 Expression | Increased CD47 Expression

JBZ-001 Increases CD47 Expression

- Single-agent treatment with JBZ-001 (10mg/kg – dotted blue line) displays potent anti-tumor activity in xenograft model of **AML**
- Combination of JBZ-001 and the αCD47 mAb B6H12 resulted in **impressive, prolonged survival**, with all mice surviving to study end (>80 days)



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Our Clinical Development

Phase 1 Clinical Study

JBZ-001 (Advanced Solid and NHL)

Single-Agent

We have recently begun a phase 1, open-label, dose-escalation and expansion, first-in-human trial to evaluate safety, tolerability, pharmacokinetics, pharmacodynamics, and preliminary efficacy of JBZ-001, in patients with advanced solid and Non-Hodgkin's Lymphoma.

Study Details

Ph1a to include all comers solid tumor + non-Hodgkins Lymphoma (NHL)

- Ph 1 pt 1: Safety and tolerability, prelim efficacy; MTD, 15-25 patients
- Phase 1 part 2: Dose Escalation; up to 4-indications, OBD, 40-80+ patients
- Advanced/metastatic solid tumors may include small cell lung cancer, colorectal cancer, pancreatic cancer, Gastric cancer
- Planned dose Expansion into relapsed/refractory heme malignancies multiple myeloma (MM), Acute myeloid leukemia (AML), Myelodysplastic syndrome (MDS)

Intellectual Property

Targeted Molecules for the Treatment of Cancer
Issued: US, Australia, China, Isreal, S. Africa, Europe
Patent Pending: Canada, S. Korea, Singapore
Published: Hong Kong, India
To be filed: Taiwan
Allowed: Japan, Mexico

Methods and Compositions for Inhibition of Dihydroorotate Dehydrogenase
Patent Pending: US, Australia, Canada, Japan
Published: China, Europe
To be filed: Hong Kong, S. Korea

Methods and Compositions for Inhibition of Dihydroorotate Dehydrogenase in Combination with Anti-CD47 Therapeutic Agent
Patent Pending: US

Uridine Supplementation Increases Tolerability of Treating with DHODH inhibitors
To be Filed: US

Selection of lysine salt of JBZ-001 for clinical development
To be Filed: US

Technology Right – OSIF Technologies

T2018-003—"Targeted molecules for the treatment of cancer"

T2020-047 – "Combination therapy strategies using DHODH inhibitors and antibodies"

T2021-101 – "DHODH inhibitor compositions using 6-membered heteroaryl ring replacements"

T2021-102– "DHODH inhibitor compositions using 5-membered heteroaryl ring replacements"

T2021-103 – "DHODH inhibitor compositions using substitutions of central phenyl ring"

T2021-272 – "Combination Strategies for DHODHi"

T2022-043—"Combination strategies with dihydroorotate dehydrogenase inhibitors and SLAMF7 (CD319) therapeutic antibodies in leukemia"

T2023-185 – "Uridine supplementation increases tolerability of treating with DHODH inhibitors"

T2024-176- "Selection of lysine salt of HOSS-53 for clinical development."

T2024-165—" A series of novel C-3 substituted quinoline derivatives as potent biochemical dihydroorotate dehydrogenase (DHODH) enzyme inhibitors."

T2024-166—" A series of C-3 substituted and C-4 carboxylic acid or its bioisosters quinoline derivatives as potent biochemical dihydroorotate dehydrogenase (DHODH) enzyme inhibitors."

T2024-167—" A series of novel hydantoin and thiohydantoin derivatives as potent biochemical dihydroorotate dehydrogenase (DHODH) enzyme inhibitors."

T2024-168—" A series of novel amide derivatives as potent biochemical dihydroorotate dehydrogenase (DHODH) enzyme inhibitors."

Jabez Biosciences
LEADERSHIP

Tamara Jovonovich, CEO;
PhD—20 years in pharmaceutical development; 15 FDA drug approvals

Robert Lewis, COO;
30 years in pharmaceutical development; 30 FDA drug approvals

Brian Cogley, CFO;
Over 15 years leading companies in various industries including life sciences and financial services

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Thank You!