

Dec 2025



Disclaimer

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Mission and Therapeutic Focus

OUR MISSION is to transform the lives of patients through the development of precision oral medicines with enhanced safety and therapeutic efficacy





Company Highlights



Clinical-stage precision therapeutics company founded in 2019 to discover and develop therapeutics with meaningfully improved safety and efficacy profile against first-in-class and validated but intractable targets



Pipeline generated through in-house Therapeutic Index and Brain Exposure Optimization (TIBEO) discovery engine, validated through partnerships



 JBI-802 and JBI-778 early data readout in 2026 Independent management backed by Jubilant Pharmova a global healthcare organization committed to funding through proof-ofconcept trials



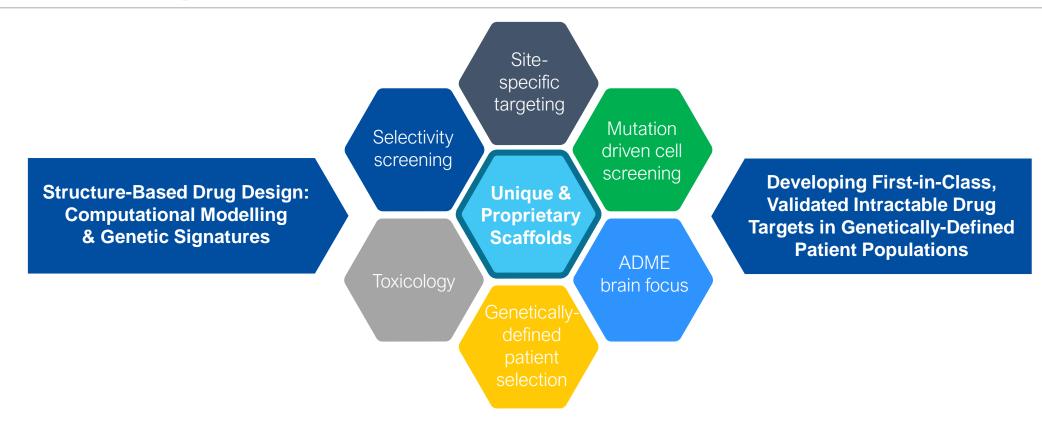
JBI-802 CoREST inhibitor in Phase II trials for heme and solid tumors; JBI-778 PRMT5 inhibitor in Phase I trials for solid tumors; PAD4 inhibitor for autoimmune disease in IND track



FDA Orphan drug designations for JBI-802 and JBI-778

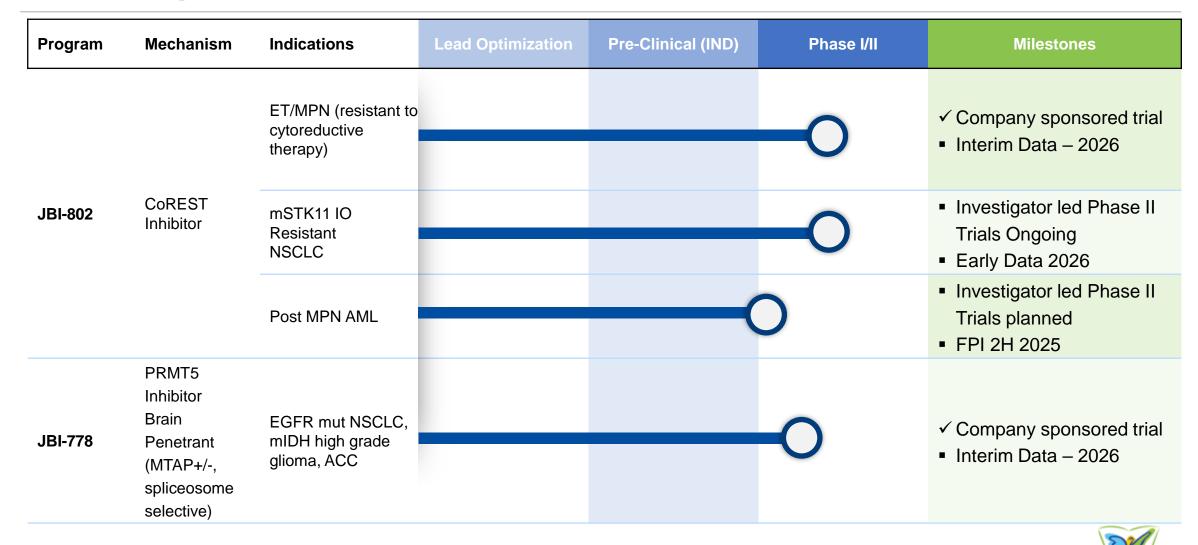


TIBEO Advanced Discovery Engine Enhances the Therapeutic Index and Optimizes Brain Penetration



Jubilant's discovery engine is based on a drug discovery approach validated through successful integrated discovery programs/ partnerships with big pharma, biotech, and healthcare VC and has a team of dedicated scientists

Diverse Clinical Stage Pipeline with Improved Therapeutic Index for Multiple Cancer Indications and Patient Subsets





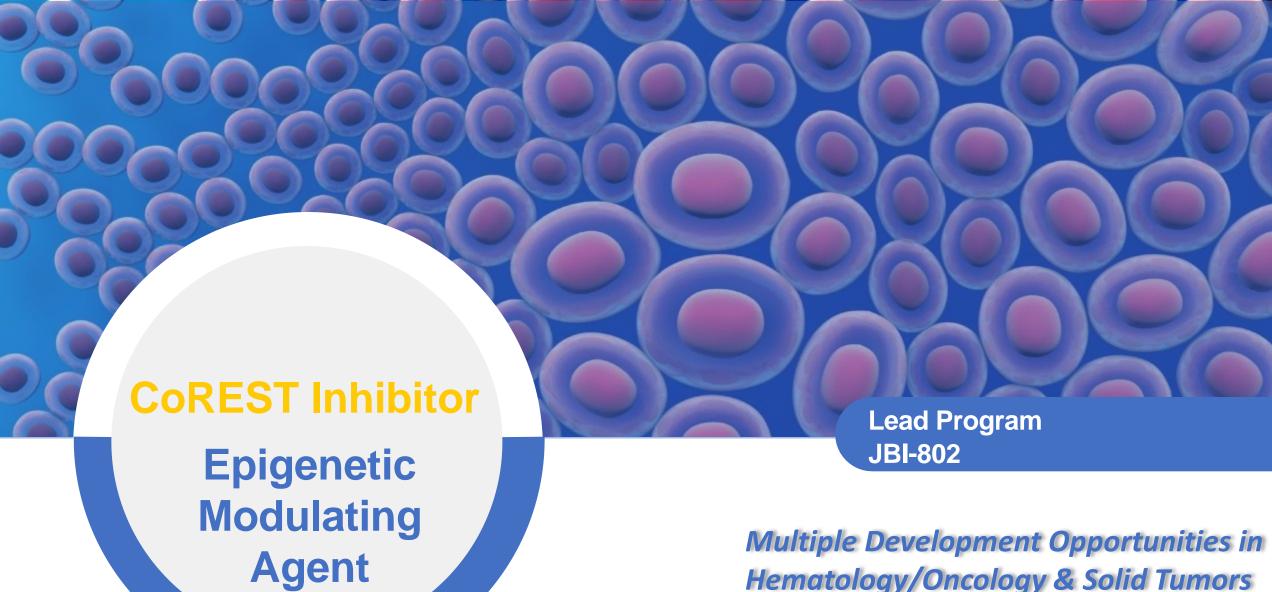
Additional Preclinical Stage Pipeline including First-in-Class Targets such as PAD4

Program	Mechanism	Indications	HIT / Lead Optimization	Pre-Clinical (IND)	Phase I/II	Milestones
JBI-3041	PAD4 Inhibitor	Autoimmune disease and oncology		0		IND track
JBI-2174	PD-L1 Inhibitor Brain Penetrant	Brain tumor and metastases Pemazyre		0		IND track
Pan KRAS	Pan KRAS "ON" inhibitor	Oncology	-0			
EGFR ^{1,*}		Oncology			— Splue	Print
BRD4 [*]		Oncology			CHECKPOINT HERAPEUTICS	

¹Blueprint Medicines acquired Lengo Therapeutics (Frazier Healthcare entity) for \$250M in cash plus \$215M in milestone payments



^{*}Economic rights reside with Jubilant Therapeutics' parent company



Hematology/Oncology & Solid Tumors

JBI-802 Represents Large Market Opportunity validated by Deal Benchmarks

3:

Merck acquired Imago Biosciences' CoREST (LSD1 only Inhibitor) in Phase II stage for \$1.35B in 2023

- Phase I/II in **ET and MPN** with thromocytosis
- Phase II in STK11-mutant NSCLC
- Phase II in Erythroleukemia (Post MPN AML)

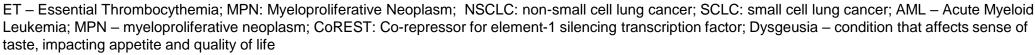
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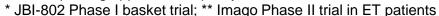
- Potential to **reverse resistance** to immunotherapy in **solid tumors** due to STK11 mutations
- Confirmed partial response in doublet IO refractory STK11-mutant NSCLC patient with Pancoast syndrome

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Potential for superior efficacy and safety compared to LSD1 only inhibitor in hematology/ oncology

- Human proof of principle with dose dependent reduction in platelet levels
- Does not induce dysgeusia or anemia in patients*
- Significant efficacy in post-MPN leukemia (erythroleukemia) model leading to orphan status by FDA

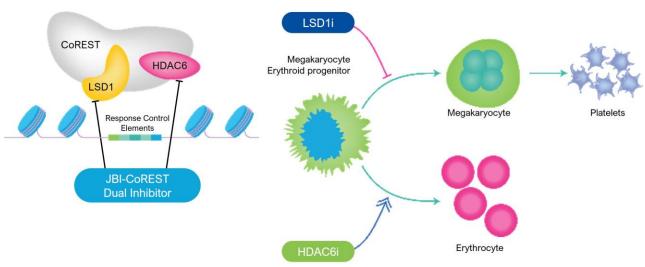






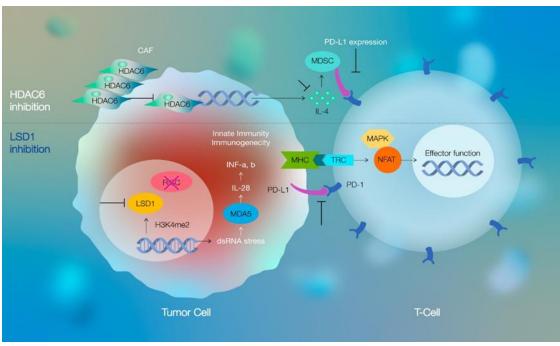
JBI-802 First-in-class CoREST inhibitor applicable in Heme and Solid tumors

Effect on Heme malignancies



LSD1 and HDAC6 are part of the CoREST complex that drives tumorigenesis of lineages such as platelet and erythroid (MEP), thereby creating opportunities for synergistic targeting of REST-driven tumors

Enhancing Anti-Tumor Immunity in Solid Tumors - mSTK11 NSCLC (IO resistant) and SCLC



- HDAC6 inhibition overcomes immunosuppression by decreasing inhibitory activity M2 Macrophage and M-MDSC and increasing NK and T cell infiltration
- LSD1 inhibition stimulates anti-tumor T cell immunity



JBI-802 can potentially address Unmet Medical Need in ET/MPN, Post MPN AML, NSCLC

ET/MPN

Blood Disorders Leading to Cancer

~100k-120k+ patients

Blood disorder causes bone marrow to produce too many platelets leading to stroke and heart attack and eventually cancer – chronic condition requiring long term therapy

Potential better safety and efficacy than Bomedemstat (Merck – currently in Phase III)

Unmet need for efficacious therapy for refractory/non-responders to current therapies (cytoreductive)

Company sponsored trial - Phase II Interim Data - 2026

Post MPN AML Leukemia

~3- 5% of 265k+ MPN patients

MPNs are blood cancers that cause increased production of blood cells, mainly affecting red blood cells, platelets, or white blood cells.

Progression from MPN to AML (Acute Myeloid Leukemia) is a serious complication, occurring in MPN patients.

High-unmet need for effective therapy with survival only for 5 months

Investigator led trial under planning at MSKCC, NY

NSCLC Lung Cancer

~15% of 200k+ NSCLC Patients

Demonstrated clinical response of JBI-802 in two patients one of whom has STK11 mutation (Phase I study)

Disease with high-unmet need with no effective therapy; Patients with STK11 mutations have a lower survival rate and are resistant to immune checkpoint therapy (Keytruda, Atezolizumab, Opdivo+Yervoy) and KRAS therapy

Investigator led trial initiated at Christ Hospital, Cincinnati

Upside in SCLC ~ 10-15% of lung cancer (JBI-802 achieved tumor regression in combination with PD-1 inhibitors in animal models) —**Investigator led trial by SWOG under planning**



Phase I advanced tumor study: Transformative JBI-802 Treatment in NSCLC

From Hospice Care to Tumor Reduction and Better Quality of Life



Initial Condition

- NSCLC patient progressed to last stage after doublet immunotherapy (Opdivo+Yervoy), referred to hospice care
- Suffering with Pancoast syndrome causing severe pain and arm immobility



Treatment Progress

- Over 2 yrs in the study, Pancoast symptoms disappeared
- Patient reported doing very well with no issues



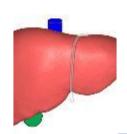
Tumor Reduction

 Confirmed partial response in repeat scans with ~40% tumor reduction



Genetic Insight

- Patient has STK11 mutation, typically resistant to immunotherapy
- Potential reversal with IO+CoREST inhibitor



2nd Doublet IO-Refractory NSCLC patient

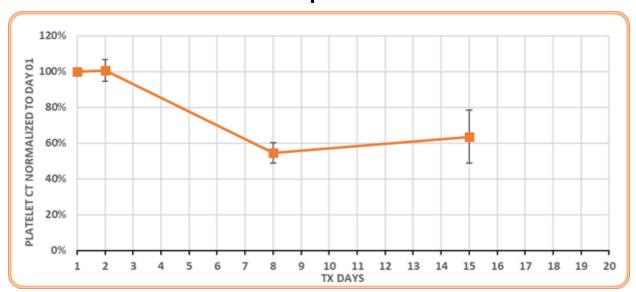
- Both lung lesion and liver metastasis, resistant to immunotherapy with poor prognosis
- JBI-802 treatment resulted in over 50% shrinkage of the patient's liver metastasis and a complete resolution of related portal hypertension, edema and improvement of quality of life

Non-confidential

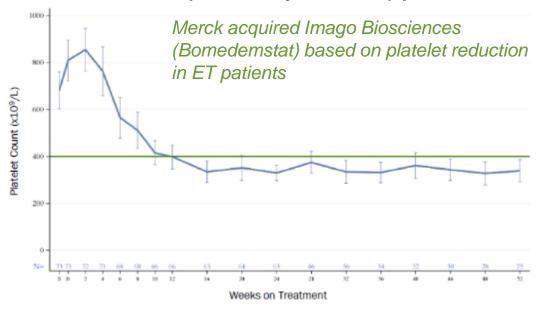


Phase I advanced tumor study: Human Proof of Principle for JBI-802 Indicates better Therapeutic Index than LSD1 Only Inhibitor





Bomedemstat (LSD1 only inhibitor) platelet levels



- Human data confirms JBI-802 can induce dose dependent decrease in platelets without effect on erythroid parameters
 - Proof of Principle for treatment of diseases with elevated platelets such as ET, MDS/MPN with thrombocytosis
- JBI-802 demonstrates superior safety compared to Bomedemstat (LSD1 only inhibitor)
 - Does not induce Dysgeusia in patients* (compared to 55% incidence**)
 - Does not induce Anemia in patients and in animals* (compared to 16% incidence**)
- Preliminary clinical data in ongoing ET trial shows promising clinical benefits for JBI-802



ET and MDS/MPN have total annual revenue potential > \$3B

U.S. Market Opportunity

ET 2nd Line

MDS/MPN-RS-t

~120k

ET Population (U.S.)1

~100k

MDS Population (U.S.)²

~60k

50% cytoreductive therapy (Hydroxyurea)²

~15k

~25% sub-optimal / intolerant to Hydroxyurea³

~1.5k

1.5% of MDS⁴

ET Unmet Needs

Standard of Care

Unmet Need

Hydroxyurea

Anagrelide

Interferon alfa 2A

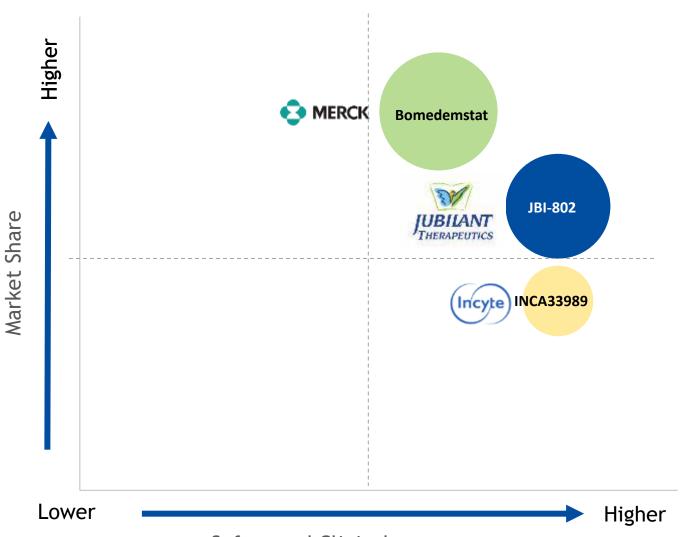
- Lack of disease modifying potential to prevent progression to myelofibrosis or secondary AML
- To mitigate symptomatology

Hydroxyurea Intolerant segment alone translates to annual revenue potential of ~\$3.3B in U.S

Current pipeline with novel mechanisms has potential to address unmet needs



JBI-802: Potential for Superior Safety and Broader Patient Reach Compared to Competitors in ET/MPN



Safety and Clinical Response:

- JBI-802 shows potential to be better than Bomedemstat with no dysgeusia and anemia
- JBI-802 (small molecule) demonstrates potential to have comparable efficacy to Bomedemstat (small molecule) and INCA33989 (biologic)

Patient Size

- JBI-802 and Bomedemstat has potential to address comparable ET patient population with Merck having the first mover advantage
- JBI-802 if successful could also target MDS/MPN with thrombocytosis
- INCA33989 can address only 20% of ET population (CALR mutation)



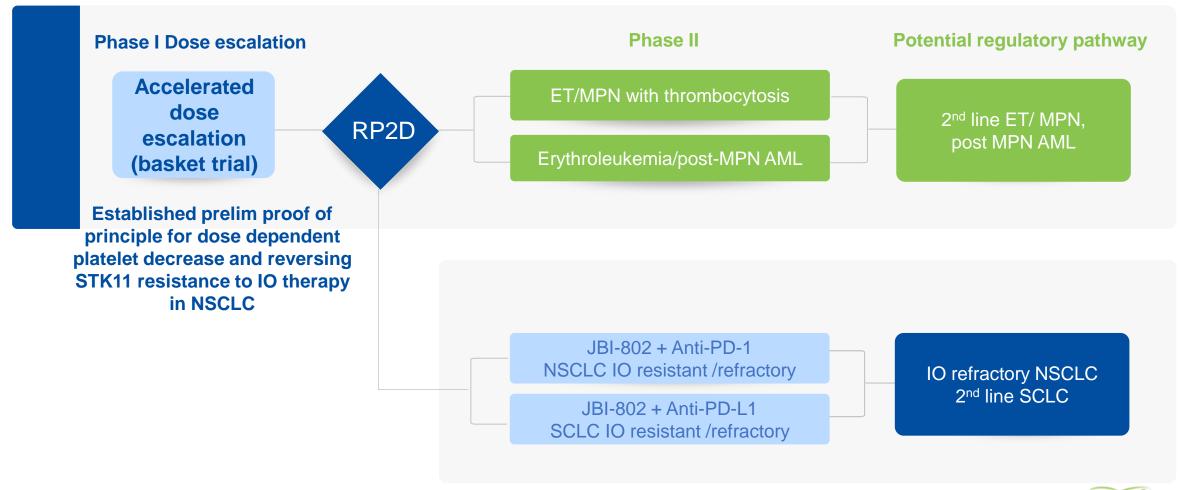
Safety and Clinical response

Clinical Data in multiple indications expected in 2026

Clinical Trials	ET+ MPN	mSTK11 NSCLC (IIT at Christ Hospital, Cincinnati)	Post MPN AML / Erythroleukemia (IIT at Memorial Sloan Kettering)	
Phase	Phase I b/II	Phase I b/II	Phase I b/II	
Number of patients	30 Essential Thrombocythemia (ET):~20 other MPN ~10	20 Arm 1: JBI-802 monotherapy; 10 Patients Arm 2: JBI 802 + anti PD-1 combination; 10 patients	JBI 802 + azacetadine (chemo) combination	
Indications	ET, MPN/MDS, MPN with thrombocytosis	STK11 mutant NSCLC refractory to IO therapy	Relapsed or refractory acute erythroid leukemia and accelerated- or blast-phase myeloproliferative neoplasms	
First Patient In (FPI)	Oct 2024	2H 2025	2H 2025	
Current Status	Enrollments and data collection ongoingInterim data 2026	IND approvedInterim data 2026	IND filing underwayInterim data 2026	



Clinical Development Pathway for JBI-802 in Hematology and Solid Tumors







mIDH high grade glioma, ACC

JBI-778 Shows Potential as Best-In-Class PRMT5 Inhibitor with Superior Brain Penetration

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Bristol-Myers Squibb CVR for Mirati (MTAP null PRMT5 Phase I/II) ~ \$1B

- Oral, highly differentiated, Substrate competitive/ SAM cooperative brain penetrant PRMT5 inhibitor
- Addresses both MTAP +/- tumors (unlike MTA co-operative inhibitors that address only MTAP null tumors)
- IND approved; Phase I ongoing

2

- IND enabling 4-week GLP tox studies completed in rats and dogs
- No mortality in either animal species even at the highest dose
- No effect on platelets

1

- PRMT5 is highly overexpressed in many human cancers
- Glioblastoma (GBM) with splicing dysregulation is selectively sensitive to inhibition of PRMT5
- 3rd Gen EGFRi resistant tumors have enriched RBM10 mutation which are sensitive to PRMT5 inhibition



Opportunity for JBI-778 to address larger market in Difficult-to-Treat Cancers (MTAP +/-) including Brain tumor

NSCLC Lung Cancer

~200k+ NSCLC Patients

High-unmet need with no effective therapy for 3rd Gen EGFRi resistant NSCLC

Genetically defined patient stratification

Preclinical studies suggest an optimal therapeutic window with demonstrated better safety

US FDA approved IND, first-in-human trial ongoing

HGG Brain Tumor

2.5% of ~61k+ Brain Cancer Patients

IDH+ HGG sensitive to PRMT5 inhibition

High unmet need with no treatment option for recurrent HGG

ACC
Head & Neck cancer

Adenoid cystic tumors enriched with specific mutations and are highly sensitive to PRMT5i

High unmet need with chemotherapy as the only current treatment option after surgery and radiation, with low efficacy

Phase I dose escalation studies ongoing



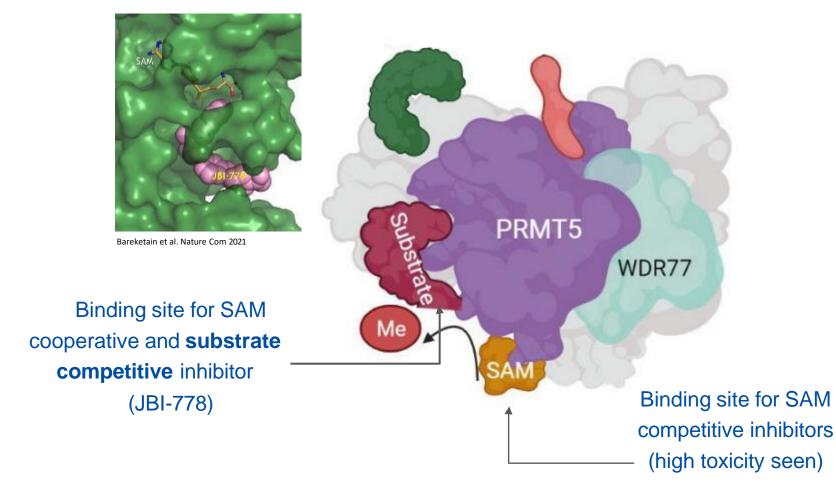
JBI-778 vs. Competitive PRMT5 Inhibitors

Company	Jubilant Therapeutics (JBI-778)	Tango, BMS(Mirati), Amgen	Pfizer, Prelude, J&J		
Product Type	 ✓ Substrate competitive ✓ SAM Cooperative ✓ Spliceosome Selective ✓ Brain Penetrant 	✓ MTA Cooperative*	✓ SAM Competitive		
Mechanism of Action	 Targets substrate site and stabilizes SAM bond to PRMT5 with high biological selectivity Brain penetration in primary tumors as well as brain metastases 	 Stabilizes MTA bond to PRMT5 which is increased in MTAP- deficient tumor Opportunity for patient selection and reduction in toxicity 	 Blocks the binding of SAM cofactor shared among many other methyltransferases 		
Challenges	 Address safety issue of 1st generation Targets broad patient population irrespective of MTAP status Spliceosome-based patient selection 	 MTAP deficiency is present in ~10% patients and may not be applicable to brain since MTA is metabolized in brain 	 Blocks a non-selective cofactor which could explain non-tolerable toxicity Limited patient selection strategy 		
Development Stage	Phase I	Phase I/II	Phase I/II terminated due to toxicity		



JBI-778 Oral, Highly Differentiated, Substrate Competitive PRMT5 Inhibitor

Safe and well tolerated in 4-week GLP tox study with no thrombocytopenia



Different binding and MOA with potential to be less toxic in clinic



mEGFR NSCLC TKI Resistant patients have high unmet needs with total annual revenue opportunity of ~\$2B

mEGFR TKI Resistant NSCLC

USA

India

~200K +

~95k+

 \sim 34k+

17% EGFR mutation²

~25k+

26% EGFR mutation²

~17k+

50% acquire resistance

~12k+

50% acquire resistance

TKI resistant mEGFR NSCLC translates to annual revenue potential of ~\$2B in US

Standard of Care

Unmet Need

Chemotherapy

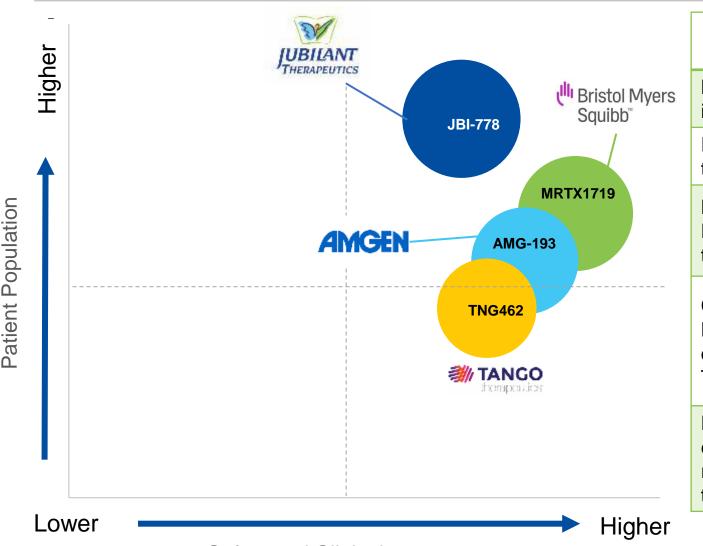
Amivantamab + Chemotherapy

Ivonescimab + Chemotherapy (China)

- Limited therapeutic options which have low efficacy and safety concerns.
- High unmet need for novel efficacious therapy
- Currently multiple mechanisms are in clinic to address unmet needs
- PRMT5i demonstrated partial response in clinic
- MTA co-operative PRMT5 inhibitors can address only 10-15% of these patients
- JBI-778: potential to address larger share of mEGFR NSCLC patients high unmet needs



JBI-778 Demonstrates Signs of Comparable Safety and Efficacy to MTA-PRMT5 Inhibitors, Targeting Distinct Larger Patient Segment



Amgen, BMS and Tango Tx	Jubilant	
MTA co-operative inhibitor	Substrate competitive	
Patients with MTAP del tumors	MTAP intact tumors but can also target MTAP del tumors	

Preclinical Safety

Demonstrated no hematologic toxicities in preclinical toxicology studies, in vivo disease model studies

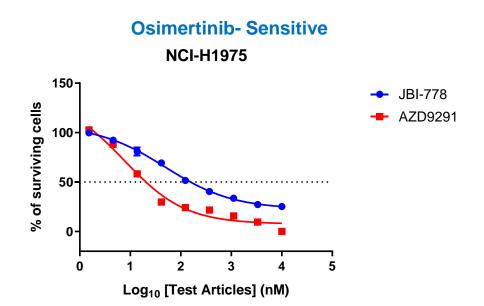
Clinical Safety No hematologic DLT's observed (excluding TNG462) at high dose	Clinical Safety No hematologic toxicity observed at concentration to achieve 90% of PRMT5 inhibition.	
Demonstrated initial efficacy with few partial	Dose escalation Phase I – dat	

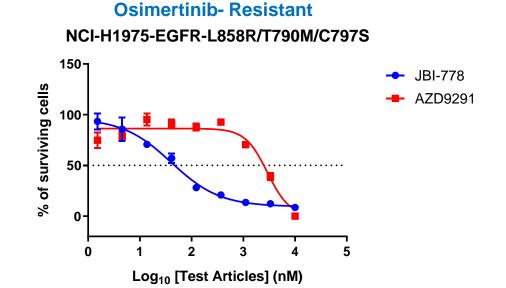
in multiple awaited responses tumors

Safety and Clinical response

Non-confidential

Anti-proliferative activity of JBI-778 in NSCLC – Osimertinib (EGFR TKI) resistant cell line





	Relative IC50 (nM)		
Compound name	NCI-H1975	NCI-H1975-EGFR- L858R/T790M/C797S (Osimertinib resistance)	
JBI-778	49.37	40.58	
AZD9291 (Osimertinib)	7.76	2821	

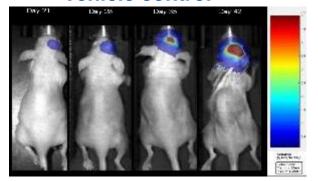
JBI-778 shows strong anti-proliferative activity in NCI-H1975 osimertinib resistant cell line



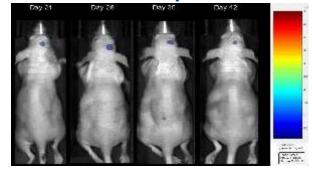
JBI-778 extends survival in preclinical models for Glioblastoma

Survival Plot: U87MG Orthopedic Study 80 -Percent survival 20 Temozolamide: Days 6 12 18 24 30 36 42 48 54 60 66 72 78 Dosed from day 21 to day 48

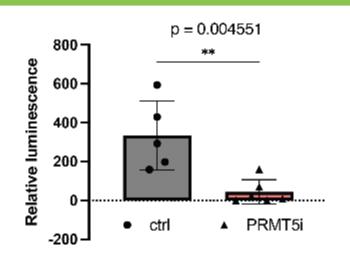




JBI-778-50mpk BID



JBI-778 treatment results in much less tumor burden on 005 GBM



GBM 005 animal model is among the best representation of the human glioblastoma tumor available

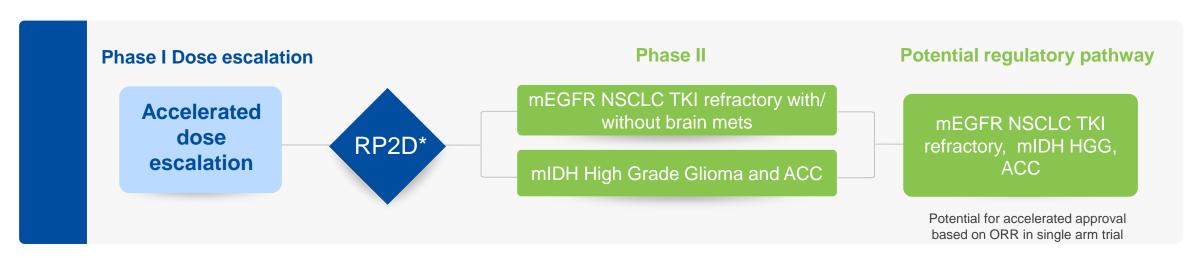


Clinical Trial for mEGFR NSCLC, IDH+ HGG and ACC in progress with early data expected in 2026

Clinical Trial	NSCLC, HGG, ACC	
Phase I		
Number of patients	42 Dose Escalation: 30 Dose Expansion: 12	
Indications mEGFR TKI resistant NSCLC; IDH+ HGG; ACC		
First Patient In (FPI)	Oct 2024	
Current Status • Enrollments and data collection ongoing • Interim data in 2026		



JBI-778 Clinical Development Strategy



*Optimal dose based on PK/PD/Efficacy

IND cleared by FDA; FIH initiated

Non-confidential





PAD4 Inhibitor: First-in-class for Autoimmune Diseases and Cancer

Current Status

Clinical candidate identified with **optimized therapeutic margin** to address acute and chronic autoimmune indications

Differentiation

First-in-class PAD4 inhibitors with mechanism of action affecting broad range of diseases

No observed immune suppression unlike JAK-2 and TNF-α

Clinical opportunities

RA, psoriasis, fibrosis, high unmet niche inflammatory indications like hidradenitis suppurativa (HS) and antibody associated vasculitis (AAV)

Liver mets in colorectal and pancreatic cancer using PAD4 induced MPO as biomarker

Efficacy

Therapeutic activity observed in multiple disease models including RA and other autoimmune/inflammation models including IPF, colitis, diabetic wound healing and psoriasis

Safety

Non-GLP toxicology studies: Well tolerated with excellent therapeutic margin with no observed immune suppression including absence of neutropenia, thrombocytopenia and leukopenia.

Current Stage

9 – 12 months to IND filing



Competitive Landscape: Jubilant PAD4i is the most advanced small molecule inhibitor

	Company	Indications	Target	Stage	Technologies
	AstraZeneca plc	Rheumatoid arthritis	PAD II inhibitor; PAD IV inhibitor	Phase I	Bispecific antibody
	Mitsubishi Tanabe Pharma Corporation	Rheumatoid arthritis	PAD4	Phase I	Antibody
	Citryll	Rheumatoid arthritis and HS	NET inhibitors	Phase I	Antibody
	Jubilant Therapeutics	Rheumatoid arthritis; Solid tumors	PAD IV inhibitor	Preclinical	Small molecule
_	Nagoya City University; Yokohama City University	Rheumatoid arthritis; Lupus	PAD IV inhibitor	Preclinical	Chimeric monoclonal antibody
	Shanghai Qilu Pharmaceutical	Rheumatoid arthritis	PAD II inhibitor; PAD IV inhibitor	Preclinical	Bispecific antibody
	BMS	Rheumatoid arthritis	PAD4 inhibitor	Preclinical	Antibody



Severe RA patients non responders to current SoC: annual revenue opportunity of ~\$6B

RA Market

U.S. India
Prevalent Population

~1.8M

~1.3M

~29% Severe RA

~500K

~377K~

~50% Partial/Non responders to MTX

~250K

~189K

~40% Biologic non responders

~100K

~95K

Severe RA patients not responding to current treatments is a sizeable market opportunity

RA Unmet Needs

Standard of Care

Unmet Need

Biologics (TNF..)

Kinase Inhibitors (JAK2i)

Anti-inflammatory DMARD

(Methotrexate)

- Current SoC biologics (TNFa) and JAK inhibitors cause immune suppression
- Low clinical remission rates
- Non-responders to current SoC

Mechanisms like PAD4 inhibition can address immune suppression challenges and unmet needs

SoC - standard of care

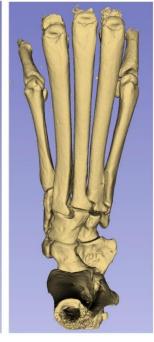


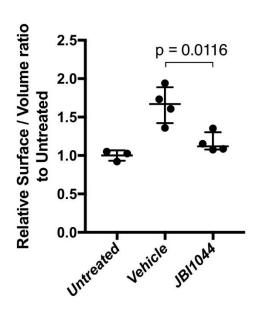
Jubilant PAD4 inhibitor showed superior efficacy in G-CSF modified CIA model at Prof. Wagner's Lab – Boston Children's Hospital

Micro-computed tomography (Micro-CT) image

Vehicle JBI1044

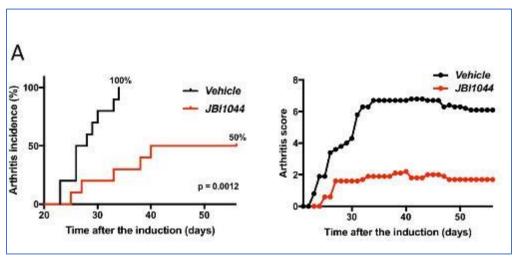




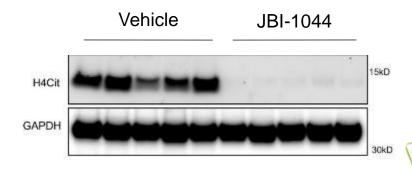


Scanning was performed in air using μ CT 40 (Scanco Medical, Bassersdorf, Switzerland) at 80 kVp, 88 μ A, 1,000 ms integration time, and a voxel size of 6 μ m. 3D reconstructions and analysis of images were performed with 3D Slicer

Granulocyte colony-stimulating factor (G-CSF)-modified CIA model



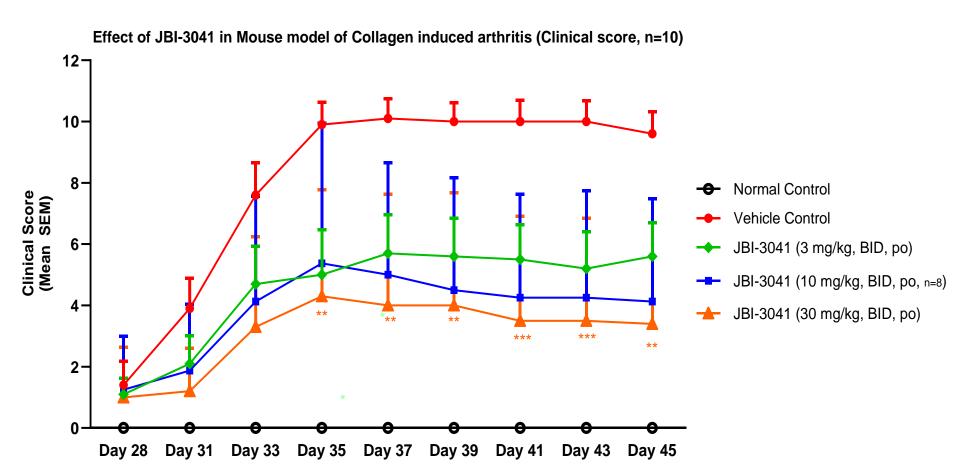
Detection of H4Cit in synovial joint



H4Cit – Histone4 Citrullination; G-CSF – granulocyte colony-stimulating factor CIA – collagen-induced arthritis



JBI-3041 In-vivo efficacy in CIA model

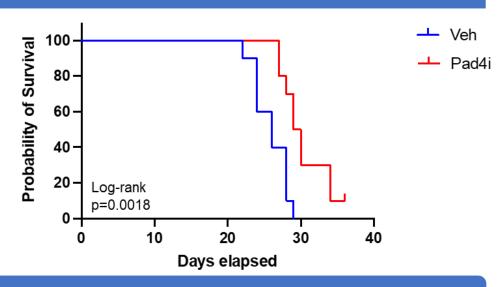


***P<0.001, **P<0.01, *P<0.05 vs Vehicle Control, Two way ANOVA followed by Bonferroni multiple comparisons Test.

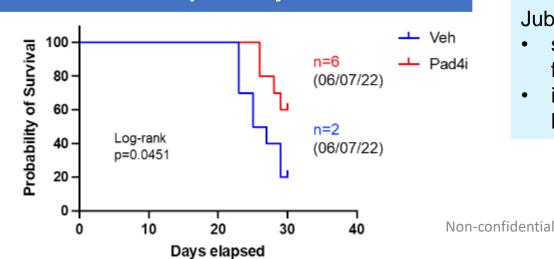


Jubilant PAD4 inhibitors decrease metastasis in multiple models of cancer

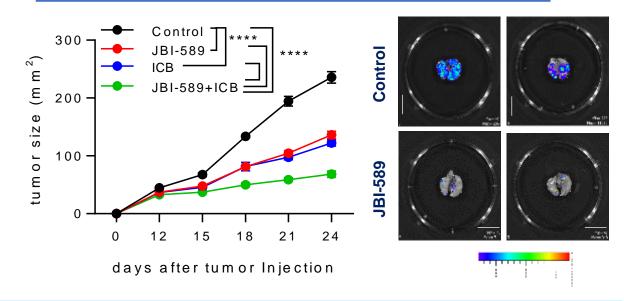
Metastatic pancreatic cancer to liver



MC38 Splenic injection



Metastatic lung cancer



Jubilant PAD4i

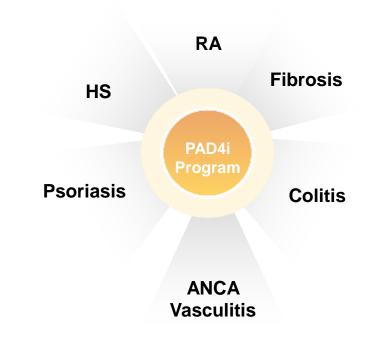
- slows growth of primary tumor and completely abolishes formation of lung metastasis
- inhibits metastatic tumor growth in liver by modulating hepatic tumor microenvironment

Studies done by Wistar Institute, PA, US and Cedars Sinai, CA, US Cancer Res (2022) 82 (19): 3561–3572. https://doi.org/10.1158/0008-5472.CAN-21-4045



PAD4 Inhibition: A Potential Pipeline for Broad Spectrum of Autoimmune Indications and Cancer

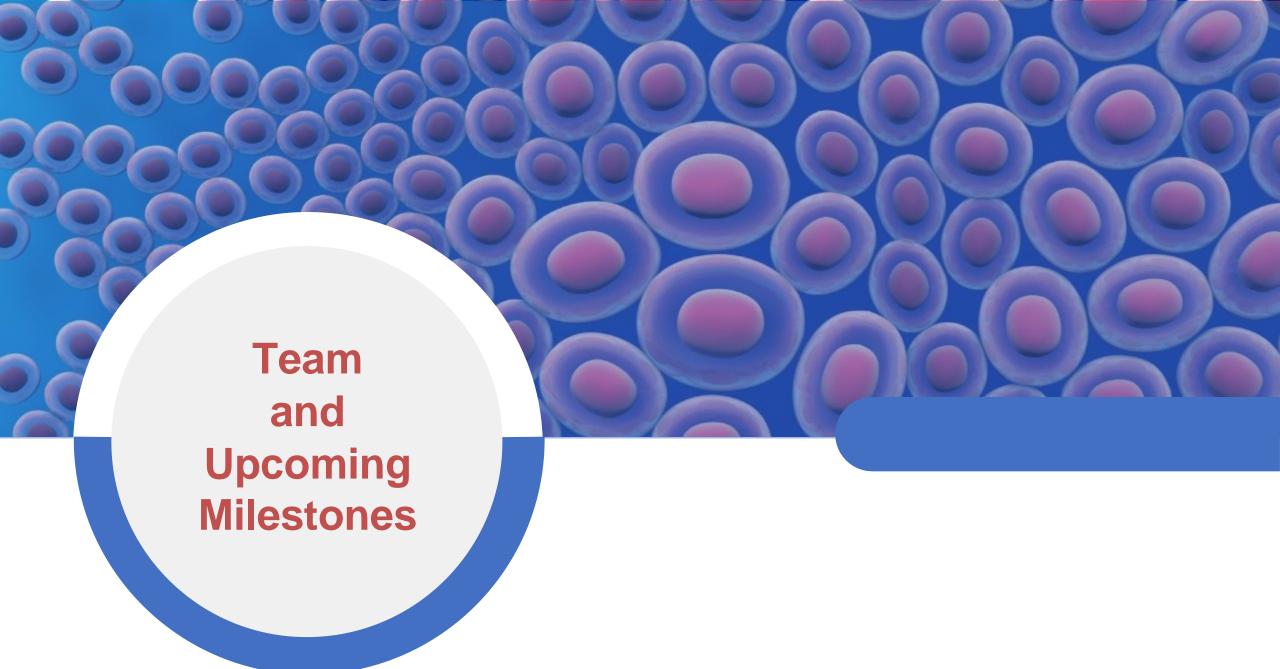
Disease	PAD4 Inhibition Outcomes	Potential Market Size (US)
Rheumatoid Arthritis (RA)	↓ Citrullinated autoantigens↓ ACPA formation↓ Pro-inflammatory cytokines	D2T RA patients ~100k pts. \$6 B
Idiopathic Pulmonary Fibrosis (IPF)	1 J. Pro-inflammatory cytokines	
Psoriasis (PSO)	↓ NETosis↓ TLR4/IL36R pathway activation↓ Skin inflammation	Refractory pts. ~163000 pts. \$9-10B
Hidradenitis Suppurativa (HS)	↓ NET accumulation in lesions↓ Potential reduction in disease severity and lesion inflammation	Moderate to Severe (2L/3L) ~45k pts. \$2 – 3 B
ANCA-Associated Vasculitis (AAV)	 ↓ NETosis by ANCA-activated neutrophils ↓ Circulating NETs ↓ Mitigation of vascular inflammation and disease activity 	Inadequate Responders ~12k pts. \$0.8B



Other Inflammatory/Autoimmune diseases and Cancer

PAD4 inhibition is a targeted mechanism to address refractory/immune compromised autoimmune disease patients without compromising immune integrity





Executive Leadership Driven by Scientific Excellence to Develop First in Class Therapeutic Targets



DANIEL J. O'CONNOR President and Chief Executive Officer



MELDA DOLAN, MD Chief Medical Officer



SHYAM PATTABIRAMAN, MS Chief Financial Officer

pwc

IUBILANT PHARMOVA



SRIDHARAN RAJAGOPAL, PhD VP & Head, Medicinal Chemistry















Scientific Advisory Board:

Dr. William C. Hahn

William Rosenberg Professor of Medicine, Interim EVP & COO, CSO, Dana-**Farber Cancer Institute**

Dr. Santosh Kesari

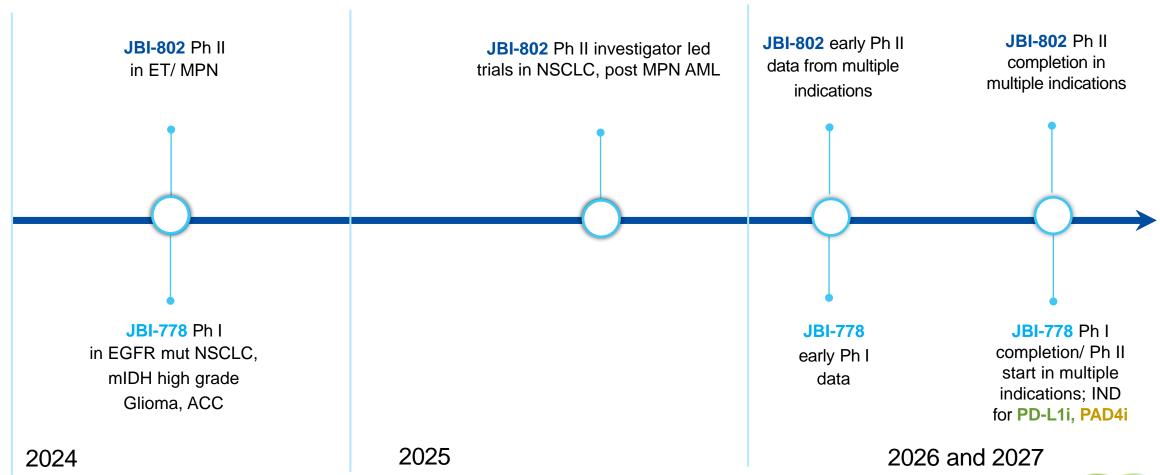
Director, Neuro-Oncology; Professor, **Dept. of Translational Neurosciences**; Director, Research Clinical Institute, **Providence Southern California**

Dr. Neal Rosen

Enid A. Haupt Chair in Medical Oncology, **Memorial Sloan Kettering**



Milestones and Catalysts





Summary

Clinical-Stage Precision Oral Medicine Company Aiming to Improve Patient Outcomes in Hematology Oncology, Solid Tumors, and Immunology



JBI-802 Preliminary Phase II
Clinical Trial Results
expected in 2026



JBI-778 Preliminary Phase I Clinical Trial Results expected in 2026



Value inflection potential with de-risked clinical data



Thank You

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