JUBILANT THERAPEUTICS

Developing the Next Wave of Targeted Therapies

for the treatment of life-threatening cancers

Corporate Presentation

July 2026

Non-Confidential

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Jubilant Therapeutics: Private Subsidiary of Jubilant Bhartia Group

Jubilant Bhartia Group circa 1978

Over $10B Enterprise Value | ~43,000 employees

Jubilant Ingrevia	Jubilant Agri & Consumer Products	Jubilant FoodWorks	Jubilant Pharmova	Jubilant Therapeutics	Jubilant Enpro	Jubilant Beverages	Jubilant MotorWorks
Specialty chemicals and life science ingredients	Agri, consumer and industrial products	Food service brands	Global pharmaceuticals, CDMO, and radiopharma	Clinical-stage oncology therapeutics	Business aviation and oil-field services	Bottling and distribution of Coca Cola	Luxury car dealerships e.g., Audi

PUBLICLY LISTED COMPANIES

Jubilant Bhartia Group:

- Multibillion international corporation
- Domino's, Dunkin, Popeye's Indian Licensee
- Strategic partner for Coca Cola in India

UNLISTED COMPANIES

Jubilant Therapeutics

- Private drug discovery and pipeline focused subsidiary
- Leverages cross group preclinical, CMC and regulatory expertise

Internally Discovered, Potential Best-in-Class Therapies Targeting Large Unmet Medical Needs

Target	Molecule	Indications	Pre-Clinical (IND)	Phase 1/2	Status
Dual LSD1 & HDAC6 CoREST Inhibitor	**JBI-802**	Essential Thrombocythemia (ET)/MPNs			• Dose escalation ongoing • Potential registrational study in 2027
		mSTK11 IO resistant NSCLC			Phase 2 IST ongoing
		Post MPN-AML			Planned Phase 1b/ 2 IST with MSK Cancer Center

Co-repressor of Repressor Element-1 Silencing Transcription (CoREST); Essential Thrombocytopenia (ET); Investigator Initiated Trial (IIT) Myeloproliferative neoplasm (MNP); Non-Small Cell Lung Cancer (NSCLS); Epidermal growth factor receptor (EGFR), mitochondrial Lactate Dehydrogenase (mLDH); Adenoid cystic carcinoma (ACC); Memorial Sloan Kettering (MSK).

CoREST Inhibitors: Two is Better than One

LSD1 (lysine-specific demethylase-1)
An enzyme critical for the self-renewal potential of malignant cells and proliferation of blood stem cells. It is essential for the differentiation of mature megakaryocytes and granulocytes

HDAC6 (histone deacetylase 6)
A unique, primarily cytoplasmic enzyme which leads to increase in megakaryocyte differentiation and proplatelet formation. It is progressively expressed in ET, PV, and PMF patients, and leads to increased, activated and dysfunctional platelet production

PMF – Primary Myelofibrosis
ET – Essential Thrombocythemia
PV – Polycythemia Vera

Mechanism

- CoREST inhibitors are small molecules designed to inhibit the CoREST complex
- CoREST complex plays a crucial role in regulating gene expression through histone deacetylation and demethylation
- CoREST inhibitors have been shown to alter chromatin structure and gene expression patterns, potentially reversing immune evasion and proliferation of cancer cells
- Inhibiting this complex enables broad transcriptional reprogramming

Opportunity

- Single target epigenetic agents (LSD1 only) may deliver incomplete transcriptional reprogramming and potentially a narrow therapeutic index
- PK constraints with legacy agents (long half life, wide tissue distribution) challenge safety management
- Several companies developing single target CoREST inhibitors
- LSD1 is validated as a viable CoREST target via Merck's bomedemstat

Call to Action

- JBI-802 is a next-generation **dual targeting (LSD1 & HDAC6) CoREST inhibitor designed as a single integrated small molecule (i.e., not two separate molecules)**
- Dual inhibition of LSD1 + HDAC6 drives deeper, durable transcriptional reprogramming
- Dual inhibition optimizes PK design - shorter half life / lower tissue distribution for more predictable safety

Ismail et al., Sci. Adv. 11, eads6556 (2025), Cell Death and Disease (2025) 16:619; Xu et al., Haematol 2024, 109 (10), *Cancers 2022, 14, 6014.*

Role of HDAC6 Inhibitor

HDAC6 Expression	HDAC6 is highly expressed in platelets[1] and its expression increases in ET, PV, PMF patients[2]
HDAC6 Function	HDAC6 controls HSP90 and tubulin function, and HSP90 sustains mutant JAK2 signaling[3]
Thrombotic Risk	HDAC6 controls platelet shape and activation, and may contribute to platelet dysfunction and thrombotic risk[4-6]
Proplatelet Formation	HDAC6 inhibition may disrupt proplatelet formation to reduce platelet production[7]
JBI-802 Advantage	JBI-802 may promote JAK2 degradation while modulating platelet biology and thrombotic risk

The HDAC6-cortactin axis is a human-specific, actin-dependent regulator of megakaryocyte maturation, and its disruption explains HDAC inhibitor-induced reduction in platelets

Multiple FDA-approved HDAC inhibitors (including vorinostat, panobinostat, romidepsin, and belinostat) cause dose-dependent reduction in platelets in humans through impaired megakaryocyte maturation and proplatelet formation, a mechanism confirmed directly in human cells but not murine systems

[1]Joseph E Aslan et.al. Am J Physiol Cell Phsyiol. 2013; Dec 15 305 (12); C1230-1239; [2]Vibe Skov et.al. Leuk Lymphoma 2012 Jan;53(1):123-9; [3]Wang Y et.al. Blood 2009 Dec 3;114(24):5024-33
[4]Marie Octave et.al. Int J Mol Sci. 2021 Dec 4;22(23):13129; [5]Ana Latorre, Antonio Moscardó. Current Med Chem. 2016;23(35):3966-3974; [6]Karin Sadoul et.al. Blood 2012 Nov 15;120(20):4215-8
[7]Kahia Messaoudi et.al. Nat Commun. 2017 Nov 27;8(1):1786

PMF – Primary Myelofibrosis, ET – Essential Thrombocythemia, PV – Polycythemia Vera

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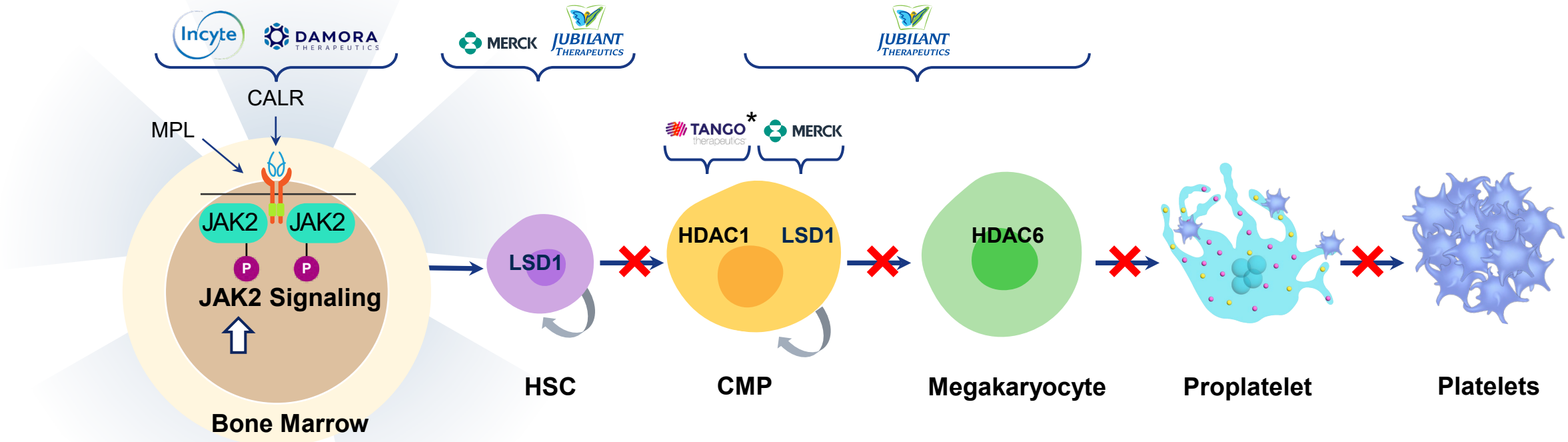
CoREST Dual LSD1 & HDAC6 Inhibition

Potential Disease Modifying Therapy in ET

- LSD1 and HDAC6 activity is elevated in ET
- LSD1 functions as critical downstream effectors of the driver mutations - CALR, MPL and JAK2 in ET
- HDAC6 is highly expressed in platelets and regulates the production and function of platelets, positioning it as a key target to control thrombocytosis in ET
- JBI-802 provides a strong rationale in the treatment of ET



HSC: Hematopoietic Stem cell; CMP: Common myeloid progenitor; CALR: Calreticulin, ER chaperone protein; MPL: Thrombopoietin receptor
**Being studied in NSCLC*
Jonas S. Jutzi, et.al. HemaSphere 2018, Nat com 2017; Joseph E Aslan, et.al. Am J Physiol cell physiol 2013, Blood, 2010; Leukemia and Lymphoma 2012, Nat Comm 2017; Am J Cell Physiol 2013; Blood 2012, Front. Immunol 2019, Yongchao Wang, et.al. Blood 2009; Hematology, 2013

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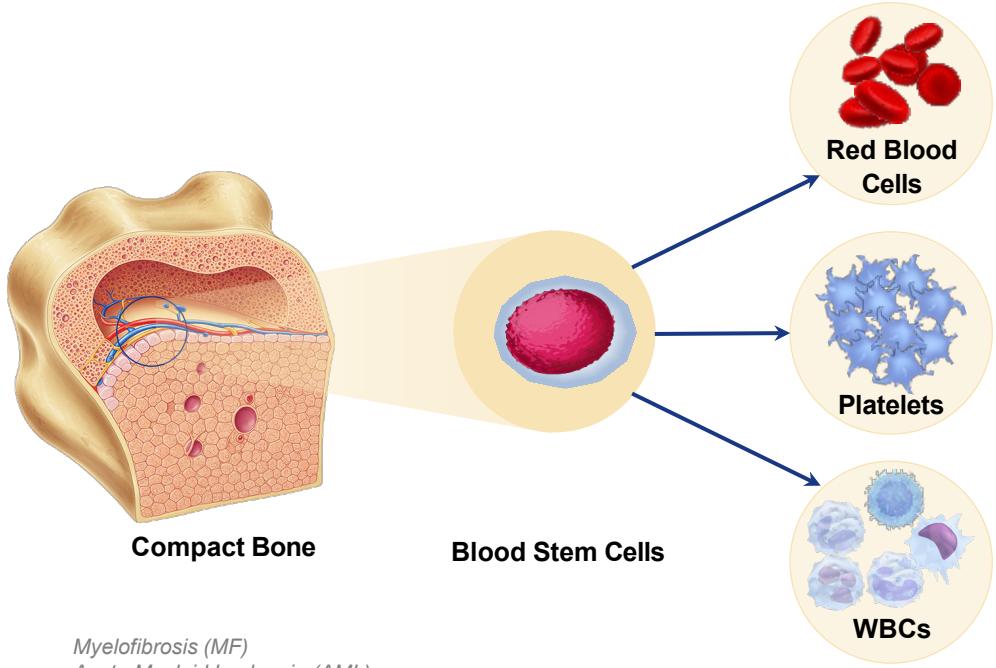
JBI-802 and Essential Thrombocythemia (ET)

Essential Thrombocythemia (ET): A Chronic Disease Characterized by Thrombosis, Thrombocytosis, and Risk of Progression to Both MF and AML

Prevalence	Up to 150,000 patients in the U.S., characterized by platelet overproduction
Clinical Risks	Thrombosis (clots), hemorrhage, impaired organ perfusion, and progression to MF or AML
Diagnosis	Platelet count ≥450,000/μL* and molecular and marrow findings, absence of other myeloid neoplasm
Treatment Goal	Lower platelet count to reduce the risk of thrombosis or hemorrhage
Unmet Need	Existing therapies reduce platelets but have limitations including boxed warnings



ET can result in excess platelet and WBC production

Dual LSD1/HDAC6 inhibition targets the root biology and clinical consequences of ET

Myelofibrosis (MF)
Acute Myeloid Leukemia (AML)
** Platelet count as per WHO guidelines for diagnosis, Normal range of Platelets is 150 to 400 10^9/L*
Yacoub et al, Clin Lymphoma Myeloma and Leuk. 2021
N Engl J Med 2017;376:2168-81.

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Clinical Presentation Varies:
70% of ET Population Prescribed Cytoreductive Therapy

Disease Risk Classification	Risk Factors	Current Therapy as per NCCN Guidelines – ASA to All Pts	JBI-802 Target Population
High-risk: 45-50% of population	• History of thrombosis or • Age > 60 years with JAK2 mutation	Cytoreductive therapy (CR) 1L: Hydroxyurea 2L: peg-interferon or anagrelide	100% of high-risk patients
Intermediate-risk: 15-20% of population	• No history of thrombosis • Age > 60 years, no JAK2 mutation	CR considered for patients with cardiovascular (CV) risk factors, symptomatic thrombocytosis, extreme platelet counts or disease burden	~50% of intermediate-risk patients
Low-risk: 20-25% of population	• No history of thrombosis • Age ≤60 years with JAK2 mutation	Low dose ASA daily, Manage CV risk. CR for significant symptoms or extreme thrombocytosis	~25% of low-risk patients
Very-low-risk: 10% of population	• No history of thrombosis • Age ≤60 years, no JAK2 mutation	Monitor	~10% of very-low risk patients

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Source: Tefferi et al, AJH 2024, Gangat et al, Blood 2024, NCCN Guidelines Version 1.2026, Alvarez-Larran et al, EHA 2023.

Current ET Treatments Control but Do Not Modify Disease

Do not prevent eventual progression to MF or AML

	Mechanism of Action	Unmet Need
Low-dose Aspirin Oral Once Daily	• Blocks microvascular clots leading to headaches, fatigue, erythromelalgia • Prevents serious thrombotic events like TIA, stroke, etc.	• Modest effect on platelet count • Von Willebrand Syndrome – increased bleeding risk with high platelets • No effect on underlying disease
Hydroxyurea Oral Once Daily	• Reduces megakaryocyte proliferation and platelet production • Only therapy proven in randomized trials to prevent major thrombosis in ET • **Boxed Warning**	• No effect on underlying disease • Cytopenias, dermatologic and mucosal toxicity • Potential long-term increased AML transformation risk • Cannot be used in pregnancy
Peg-IFN-α SQ Weekly	• Suppresses megakaryocyte progenitors and normalizes maturation • Reduces malignant clone (JAK2/CALR/MPL), lowering allele burden and potentially altering disease course	• High discontinuation due to flu-like symptoms, CNS toxicity, IRRs, etc. • Frequent monitoring requirements • Slow onset of activity vs HU
Anagrelide (2L) Oral 2-4 times daily	• Inhibits megakaryocyte maturation and platelet release • Short to medium-term platelet control • Approved 30 years ago, remains the only FDA approved therapy • **Boxed Warning**	• No effect on underlying disease • Inferior thrombotic protection vs HU • Cardiovascular toxicity in pre-existing high-risk CV patients
Ruxolitinib (2L) Oral Twice Daily	• Inhibits JAK/STAT pathway reducing inflammatory cytokine and megakaryocyte production • Modest reduction in platelet counts and cardiovascular symptoms • Off-label use to “rescue” patients intolerant to other therapies	• No clear disease modification in ET • Myelosuppression and infection risk • Modest reduction in platelet counts and cardiovascular symptoms

JBI-802: Controlling biology and potentially solving significant unmet need

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Source: FDA labels

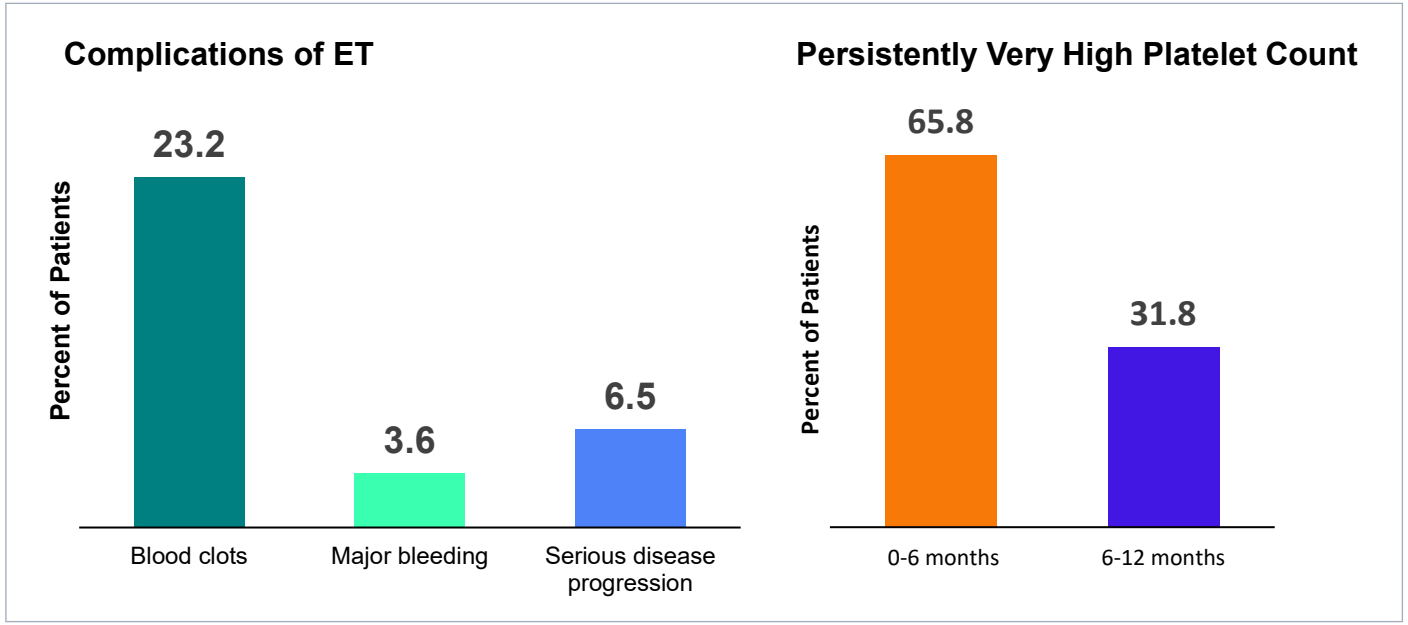
ET: Limited Treatment Options and Poor Long-Term Adherence

Treatments

- First line cytoreductive therapy with hydroxyurea (~90%) in High/Intermediate risk ET patients
- Other medications rarely used: interferon-alpha (1.8%), ruxolitinib (1.5%), anagrelide (1.3%) and busulfan (0.3%)

Poor patient outcomes with current SoC:

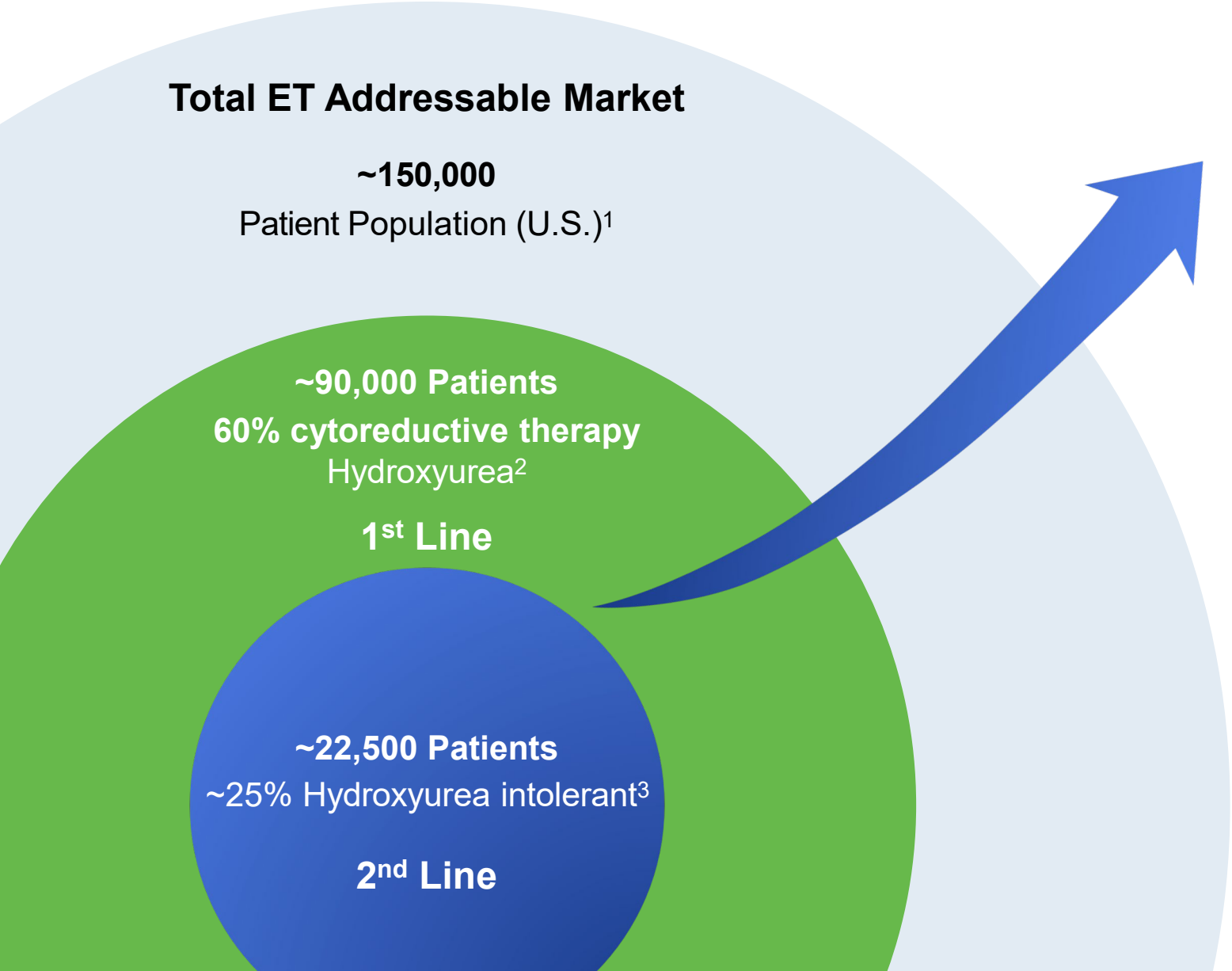
- ~30% of patients experience disease related complications e.g., blood clots, bleeding, or more serious blood disorders/cancers
- ~50% of patients interrupted or discontinued treatment
- 31.8% of patients have persistent >600k/µl platelet counts after 6 months



Managing ET remains challenging and better treatment options are needed to improve the long-term health of these patients

Source: Real-World Treatment Patterns and Clinical Outcomes in Patients With Essential Thrombocythemia Treated With Cytoreductive Therapy: A Retrospective Analysis of Electronic Health Records Data – ASH 2025

ET: Potential Blockbuster US Opportunity in Both 1st and 2nd Line Settings



2nd line Hydroxyurea intolerant segment alone translates to TAM of **~$5B in U.S4**

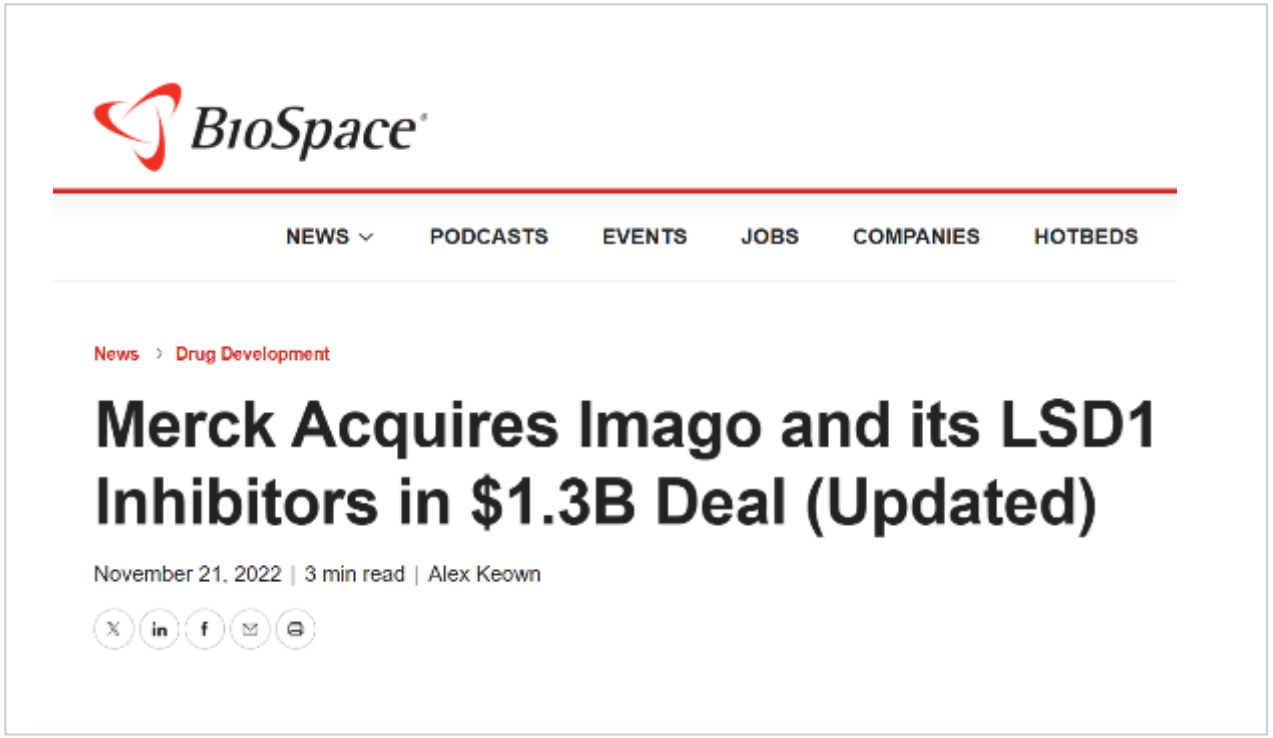
Potential to move to 1st line in ET and broader MPN segments including MF, PV

1. *Yacoub et al, Clin Lymphoma Myeloma and Leuk. 2021*
2. *Larsson,E.A et.al, Eur J Haematol. 2026 Jan 13.; Gangat et al, Blood 2024*
3. *Lancet Haematol. 2025 Nov;12(11):e862-e875*
4. *Total Addressable Market (TAM) based on Company estimate - pricing benchmarked with Jakafi Annual Cost - ~$224k (GlobalData, Myelofibrosis Report)*

Commercial Validation: Merck Acquired Imago in 2023 for $1.3B for its LSD1 Inhibitor

MERCK IMAGO BIOSCIENCES

Phase 3 clinical trials ongoing for Bomedemstat LSD1 inhibitor (MRK-3543) in 1L and 2L ET


BioSpace

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News › Drug Development

Merck Acquires Imago and its LSD1 Inhibitors in $1.3B Deal (Updated)

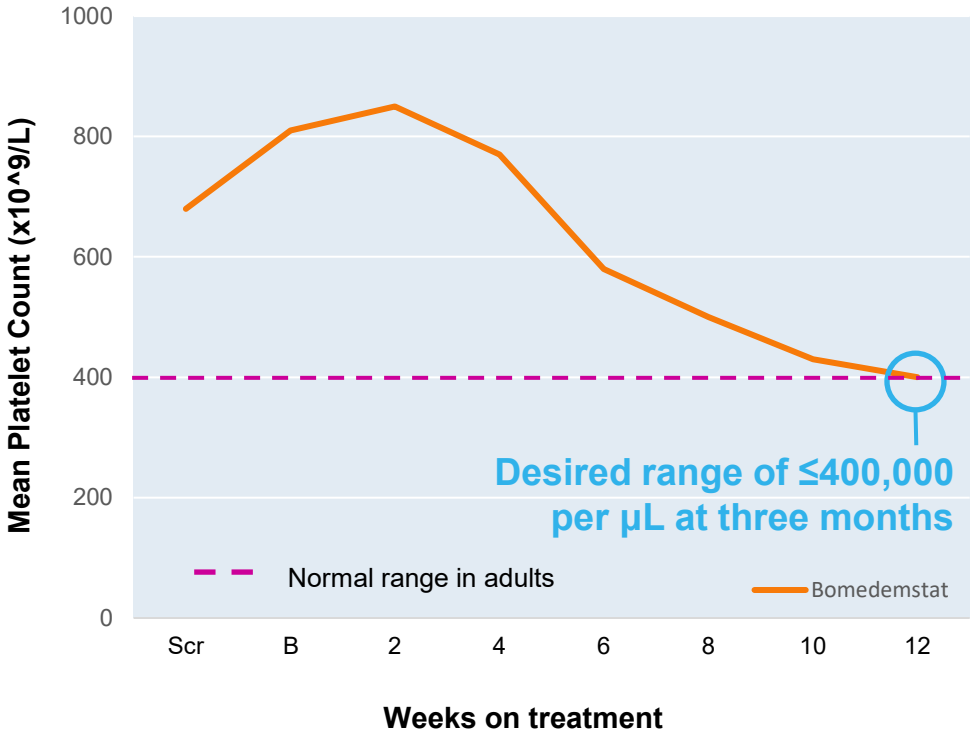
November 21, 2022 | 3 min read | Alex Keown

Strong validation of LSD1 as a target and confirmation of the MOA and commercial opportunity of the LSD1 space

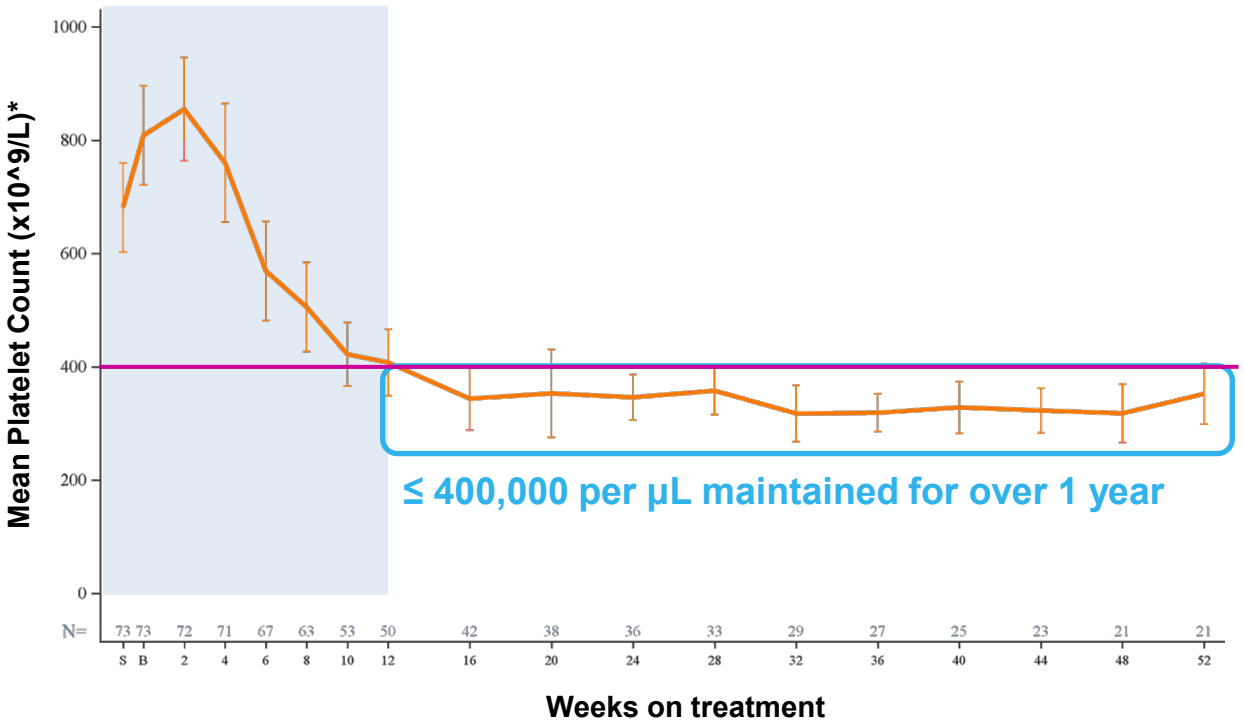
Positive Phase 2 Bomedemstat Data Drove Merck Acquisition

Mean platelet count with bomedemstat treatment

First 12 weeks



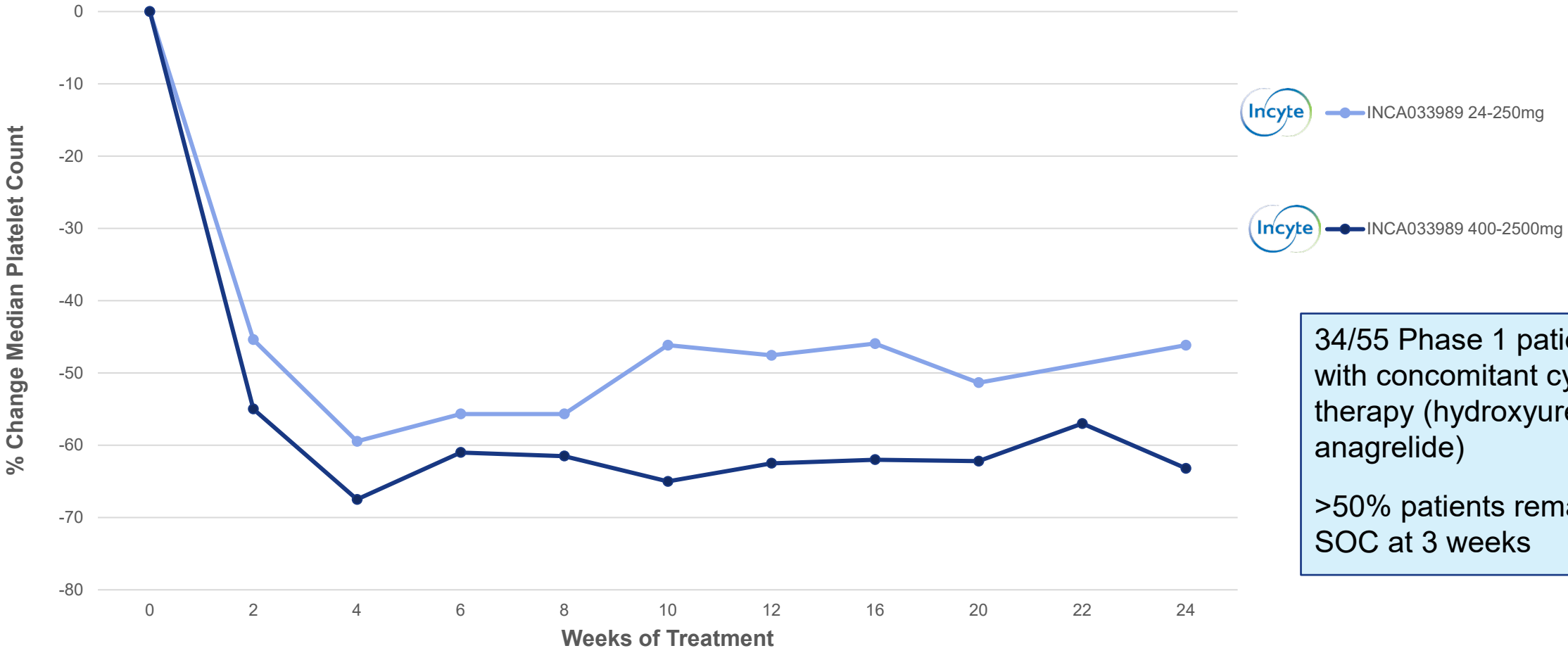
1 year



Normalization of platelet count by three months and maintained through one year

Blood (2022) 140 (Supplement 1): 1784–1787.
S=Screening, B=Last non missing value closest to day 1

INCA033989 Phase 1 Monotherapy Study Included >60% on SOC at Enrollment



34/55 Phase 1 patients enrolled with concomitant cytoreductive therapy (hydroxyurea or anagrelide)

>50% patients remained on SOC at 3 weeks

FDA Breakthrough Designation Granted Dec. 2025

Source: Gupta.V, et, al., Blood (2025) 146 (Supplement 1): 1024; Jubilant Analysis
Any comparisons shown here are for illustrative purposes only. No head-to-head studies have been conducted, and the data should not be interpreted as a direct comparison between product candidates.

Ongoing Phase 1/2, Open Label Multicenter Study of Oral JBI-802 for 2L ET

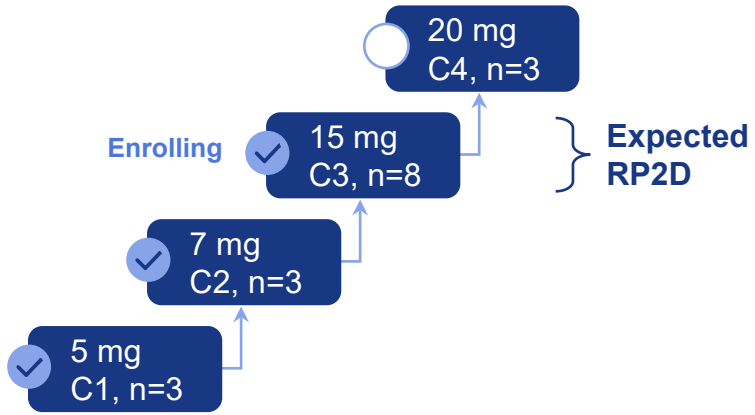
Objectives

- Evaluate safety and tolerability
- Identify MTD and/or establish recommended Phase 2 dose regimen(s)
- Demonstrate preliminary evidence of efficacy at RP2D via reduction of platelet count

Inclusion criteria

ET/MPN patients who relapsed and/or refractory or intolerant to standard of care and that, in the opinion of the Investigator, have no available therapies known to provide clinical benefits

Phase 1: Dose Escalation
i3+3 Design



Phase 2: Dose Expansion
(~15 Patients)

JBI-802 RP2D

Inclusion criteria

ET patients failed at least one standard therapy or being intolerant to standard of care.

Endpoints

- Reduction of platelet count to ≤400 x 10^9/L in the absence of thromboembolic events and progression
- Durability of platelet and WBC count reduction
- Reduction in variant allele frequency (VAF)

https://anzctr.org.au/Trial/Registration/TrialReview.aspx?ACTRN=12624000478516

Attributes of Emerging Targeted Therapies for ET: JBI-802 with Best in Indication Potential

	Bomedemstat – Merck (LSD1 only inhibitor)	Antibodies targeting mCALR: Incyte, Damora	JBI-802: Dual CoREST (LSD1 and HDAC6 inhibitor)
Extreme thrombocytosis i.e. platelets >1,000 x 10^9/L (~ 25%* of ET patients)	**No**	**Yes**	**Yes (Single Agent)**
Leukocytosis i.e. WBC > 11 x 10^9/L (~20%** of ET patients)	**No data**	**No data**	**Yes**
Relevant for all driver mutations	**Yes**	**No**	**Yes**
Disease modifying (reduction in VAF)	**Yes**	**Yes**	**Yes**
Patient Compliance	**Yes** (oral once daily)	**No** (IV once every 2 weeks or subcutaneous)	**Yes** (oral once daily)

*Source: *Blood Adv (2024) 8 (23): 6043–6054. ** Blood Cancer J.* **14**, 11 (2024) ; [&]Clin Med Insights Blood Disord. 2020 Dec 28;13:2634853520978210 ; ^^Carobbio A et al, Blood Advances 2019; Erdos K et al. Blood (2023) 142 (Supplement 1): 1819. ^ *Blood. 2013 Dec 23;123(10):1544–1551: patients with JAK2-mutated ET had higher risk of thrombosis (more than 2 fold) than those with CALR-mutated ET (Hazard Ratio 2.12, CI 95% 1.14-3.92, P = .017)*

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JBI-802 Potential in Other MPNs and Beyond

JBI-802 – Potential Dual Mechanism Advantage in Myelofibrosis (MF)

LSD1 (Bomedemstat) in MF — Clinical Proof[1]

- Spleen volume reduction in 66% of pts
- Symptoms improvement (TSS) in 65% of pts
- Hemoglobin stable/improved in 90% of transfusion independent pts
- Bone marrow fibrosis improvement in 85% of pts
- VAF Reduction in 52% of pts

HDAC6 in MF — Preclinical Evidence

- High HDAC6 expression associated with splenomegaly
- HDAC6i reduces bone marrow fibrosis in preclinical models

Dual CoREST inhibitor potentially augments LSD1 response to provide deeper, more durable control than SOC

Prevalence [2]
~18,000 (US)

50% JAKi NR[3]
~9000

TAM
~$2B

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1. Blood (2022) 140 (Supplement 1): 9717–9720; 2. Myelofibrosis – MPN Research Foundation; 3. Blood (2018) 132 (5): 492–500.
TSS – Total Symptom Score, VAF – Variant Allele Frequency, NR – Non-responders; TAM – Total Addressable Market

JBI-802 – Potential Dual Mechanism Advantage in Polycythemia Vera (PV)

LSD1 (Bomedemstat) in PV — Clinical Proof[1]

- Hematocrit reduction in 85% of pts
- Platelets reduction in 90% of pts
- WBC reduction in 75% of pts
- Spleen volume reduction in 40% of pts

HDAC (Givinostat) in PV — Clinical Proof[2]

- >80% Overall Response Rate with long-term follow-up
- 20–30% reduction in JAK2 VAF
- Phase 3 randomized study vs hydroxyurea ongoing
- Fast Track Designation for PV

Dual LSD1 and HDAC6 inhibition may:

- Improve hematocrit, platelet and WBC control
- Reduce JAK2 signaling via HDAC6/HSP90
- Provide durable disease control with greater reduction in molecular burden vs SOC

Prevalence[3]
~165,000 (US)

70% High Risk[4]
~116,000

20% HU NR[5]
~23,000

TAM
~$5B

1. Phase 2 - ASH abstract # 634. Blood (2025) 146 (Supplement 1): 83; 2. Phase 1/ 2- Givinostat long-term PV studies (Blood Cancer J 2021)
3. Leuk Lymphoma. 2014 Mar;55(3):595-600; 4. Blood (2025) 146 (Supplement 1): 8202.; 5. Leukemia volume 35, pages623–627 (2021)
WBC – White blood cells, VAF – Variant Allele Frequency, NR – Non-responders (Resistant/ Intolerant); TAM – Total Addressable Market

JBI-802 Beyond MPN:
Significant Multi-indication Therapeutic & Commercial Opportunity

Heme-malignancies

Post MPN AML / AEL
(planned IIT at MSK)

- Post MPN AML patients progress from MPN to accelerated or blast phase
- US prevalence: ~7K patients (~3% of MPN), Median overall survival (OS) of 3-6 months
- Acute erythroid leukemia (AEL) is a rare subtype of acute myeloid leukemia (AML)
- US incidence: ~1K/yr (~5% of AML), Median overall survival (OS) of 1.8 months
- **FDA Orphan drug designation obtained**

Solid Tumor

mSTK11 NSCLC
(ongoing IIT at Christ Hospital, Cincinnati)

- mSTK11 NSCLC patients are resistant to immune-checkpoint and KRAS therapies
- US prevalence: ~20K patients (10-15% of NSCLC)
- Early clinical activity with tumor response in 2/2 patients
- Synergistic in combination with IO agents in preclinical models

ES-SCLC

- Extensive Stage (ES)-SCLC is a highly aggressive form of cancer
- Often presents at an advanced stage (Stage IV) with metastases
- US incidence: ~20-25K patients/ yr (70% of SCLC), mPFS with 1st line therapy < 6 months
- **FDA Orphan drug designation obtained**

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JBI-802 and Non Small Cell Lung Cancer (NSCLC)

JBI-802 in mSTK11 NSCLC: Phase 2 Trial

Phase 2 trial IST (~20 patients in Christ Hospital, Cincinnati)

- Open-label, 4 days on/ 3 days off JBI-802
- mSTK11 NSCLC patients: 10 patients with JBI-802 monotherapy and 10 patients with JBI-802 + pembrolizumab

Objectives: To evaluate the overall safety and tolerability of JBI-802 as a single agent and in combination,
To determine antitumor activity of JBI-802 as a single agent and in combination

Endpoints: Incidence of adverse events, Investigator Assessed ORR and PFS as defined by (RECIST) version 1.1

Current Status, Preliminary Insights and Way Forward

- Ongoing; 2 patients enrolled

JBI-802 is Generally Well Tolerated in Patients with mSTK11 NSCLC

- Majority of AEs are low-grade
- No thrombocytopenia or anemia

JBI-802 Monotherapy Demonstrates Encouraging Clinical Activity

- Early data suggest monotherapy antitumor activity

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ORR – Objective Response Rate; PFS- Progression Free Survival; IST – Investigator Study Trial

Transformative JBI-802 Treatment Potential in mSTK11 NSCLC: Patient Case Study

From Hospice Care to Tumor Reduction and Better Quality of Life

Initial Condition	NSCLC patient progressed to last stage after doublet immunotherapy (Opdivo+Yervoy), only hospice care option Suffering with Pancoast syndrome causing severe pain and arm immobility
Treatment Progress	**On treatment for >2.5 yrs with JBI-802 monotherapy**, **Pancoast symptoms disappeared** Patient reported doing very well with no issues
Tumor Response	**~40% tumor reduction**
Genetic Insight	Patient has STK11 mutation, typically resistant to immunotherapy Potential reversal with IO+CoREST inhibitor

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Patient from prior Phase 1

Encouraging Anti-Tumor Activity Observed in IO Refractory NSCLC Patient in Prior Phase 1 Monotherapy Study

Patient Diagnosis:
Stage IV Squamous NSCLC

Patient Treatment History:
Immunotherapy and Chemotherapy (Non-responder)

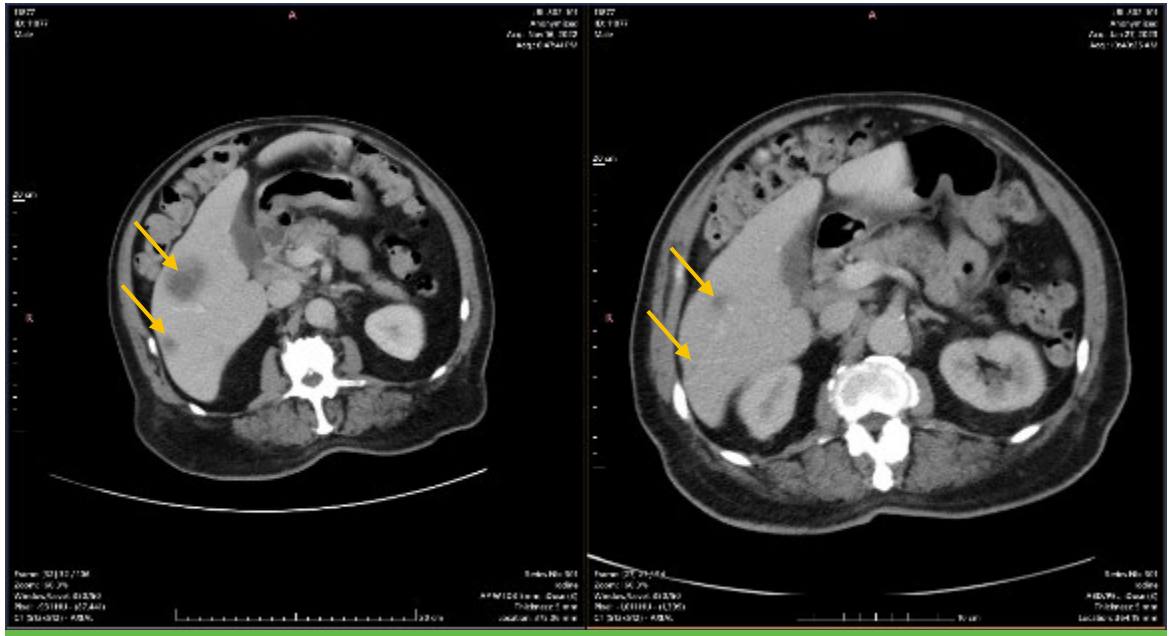
Treatment:
JBI-802 10mg dose, 4 days ON, 3 Days OFF

Response Summary:
Target Lesion – Liver Metastasis – **Stable Disease**

So-what:
Response in difficult-to-treat squamous NSCLC metastasized to liver resistant to IO and Chemotherapy

Safety:
Well tolerated

Baseline (30 mm) Follow-up Scan (15 mm)



~50% shrinkage in liver metastasis, complete resolution of related portal hypertension, edema and improvement of quality of life

JBI-802 Phase I study in Advanced Solid Tumors

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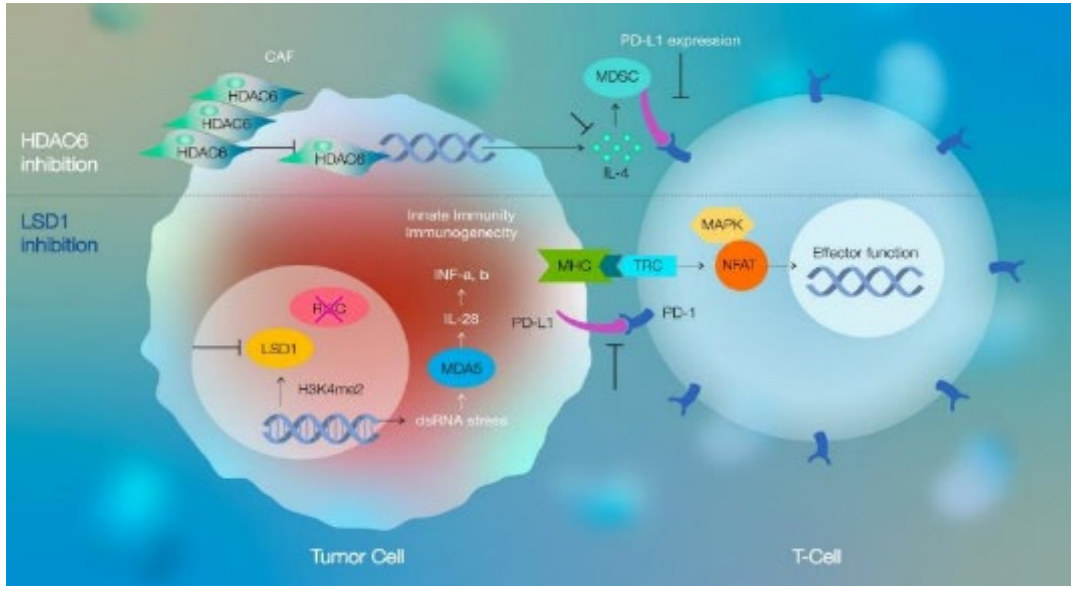
Combination Potential: Dual CoREST Inhibition + PD-1 Blockade Strong In Vivo Synergy Borne Out of Solid Science Observed Preclinically

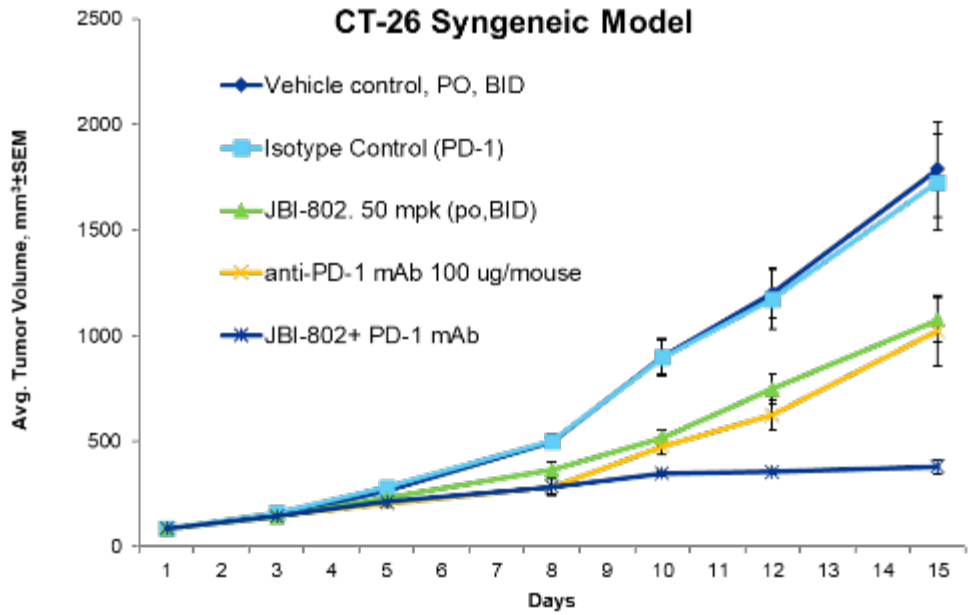
Rationale for Combination: CoREST Inhibition Boosts Anti-Tumor Immunity



Dual inhibitor plus known PD-1 blockade:

- **LSD1 inhibition -** ↑ CD8+ T-cell differentiation & IFN response → enhanced tumor killing
- **HDAC6 inhibition -** ↓ immunosuppressive M2 macrophages & M-MDSCs; ↑ NK & T-cell infiltration

JBI802 + PD-1i – Synergistic Efficacy Signal



- ~80% tumor growth inhibition JBI-802 + anti PD-1
- Complete regression in 3/8 animals
- Generally well tolerated

Sci Rep. 2019 Apr 16;9(1):6136, Cell. 2018 Jul 26;174(3):549-563

AACR Annual Meeting 2019 March 29-April 3, 2019; Atlanta, GA.

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Summary

JBI-802 Investment Highlights Summary

Oral, First-in-Class Dual CoREST	JBI-802 is the only dual CoREST inhibitor targeting both LSD1 and HDAC6 and addresses the limitations of other single target CoREST inhibitors (e.g., Merck, only LSD1i, and Tango Therapeutics, only HDAC1)
Essential Thrombocythemia	Huge unmet need and large commercial opportunity Anagrelide is the only FDA approved drug for ET and has a Boxed Warning, No FDA approvals for ~30 years ~150,000 US patients represents a multi billion annual market opportunity in the U.S.
Demonstrated Clinical Activity	Early Phase 1/2 data demonstrate rapid, durable, dose-dependent platelet reduction with JBI-802, comparing favorably to published bomedemstat (Merck) Phase 2 and ICALR–targeted mAB Phase 1 (Incyte) benchmarks Expected 15mg RP2D dose provides potential superior safety profile to bomedemstat's starting dose~40-50mg
Best-in-indication with Near-Term Regulatory Catalysts	One of only two oral targeted treatments for ET; JBI-802 emerging as potential best-in-indication FDA Orphan and Fast Track designations planned for 2H2026 Orphan designation granted for AML, SCLC
Established Heritage & Powerful IP Portfolio	Discovered by Jubilant Therapeutics, a precision oncology biotech founded in 2019 by Jubilant Bhartia group, a 43,000 employee multi-billion international organization with multiple businesses Composition of matter global patent coverage until at least 2037 - extendable up to 2043 and beyond

Upside in other MPN (PV, MF) and beyond (post MPN AML and solid tumors)

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AML – Acute myeloid leukemia

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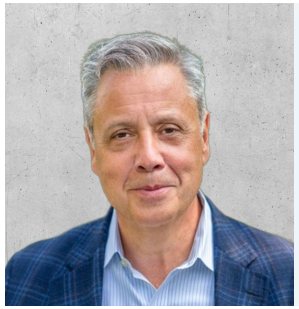
Leadership

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



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JUBILANT THERAPEUTICS

Thank You

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Appendix

Additional clinical and preclinical stage pipeline

Target	Molecule	Indications	HIT / Lead Optimization	Pre-Clinical (IND)	Phase 1/2	Status
PAD4 Inhibitor	JBI-3041	Autoimmune disease and oncology				Candidate Identified
PD-L1 Inhibitor Brain Penetrant	JBI-2174	Brain tumor and metastases				Candidate Identified
Pan KRAS "ON" inhibitor	Pan KRAS	Oncology				Hit Series
EGFR[1,*]		Oncology				blueprint MEDICINES
BRD4[2,*]		Oncology			CHECKPOINT THERAPEUTICS	
PRMT5 MTAP-agnostic brain penetrant	JBI-778	EGFR mut NSCLC, mIDH high grade glioma, ACC				Phase 1 dose escalation ongoing

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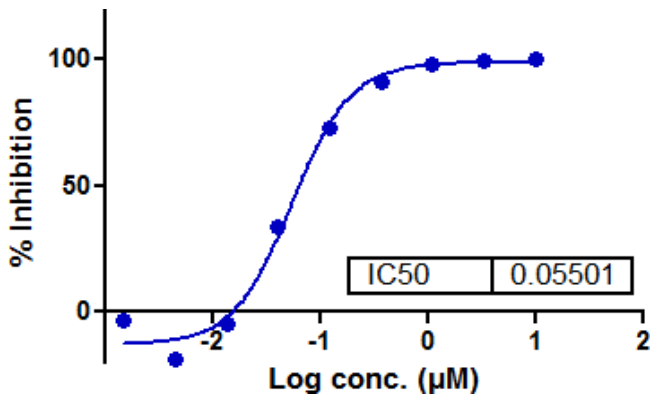
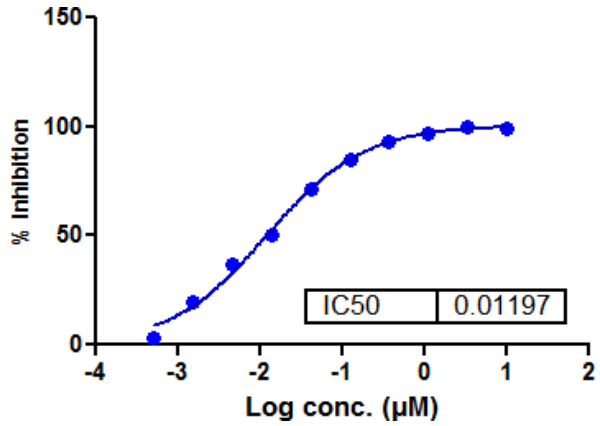
[1]Blueprint Medicines acquired Lengo Therapeutics (Frazier Healthcare entity) for $250M in cash plus $215M in milestone payments. Blueprint Medicines acquired by Sanofi in 2025
[2]Checkpoint Therapeutics acquired by Sun Pharma in 2025
**Economic rights reside with Jubilant Therapeutics' parent company*

JBI-802: Novel, Potent Dual CoREST Inhibitor Optimized for Targeting of LSD1 and HDAC6 in a Single Pharmacophore with Sophisticated Chemistry

LSD1 Potency



HDAC6 Potency



JBI-802 has potency for both LSD1 and HDAC6 in the low nM range

HDAC6 Isoform Selectivity

HDAC Isoform	JBI-802 IC_{50} nM	ACY-1215 IC_{50} nM
HDAC1	923	58
HDAC2	1560	48
HDAC3	1220	51
HDAC4	7840	7000
HDAC5	4450	5000
HDAC6	11.9	4.7
HDAC7	1720	1400
HDAC8	98.3	1000
HDAC9	5710	10000
HDAC10	2220	NA
HDAC11	1330	10000

JBI-802 is highly selective for HDAC6, unlike pan HDAC inhibitors that cause toxicity

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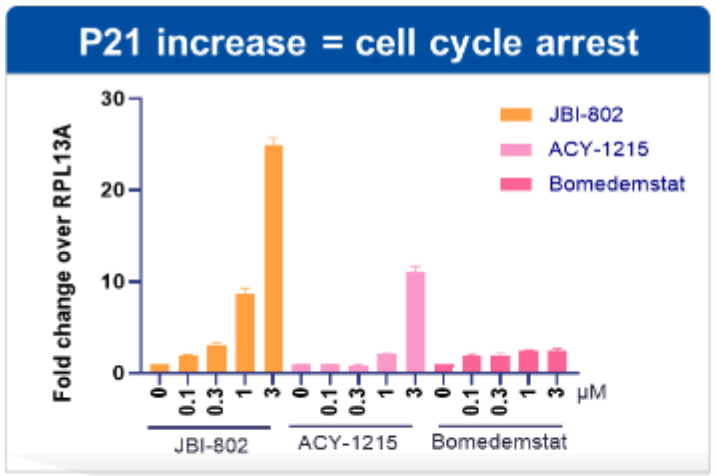
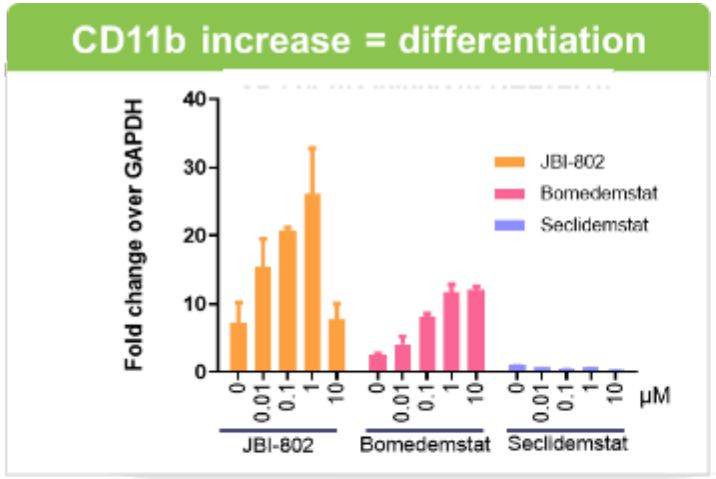
Dual LSD1 and HDAC6 Inhibition Induces Synergistic Antitumor Activity

P21 increase = cell cycle arrest

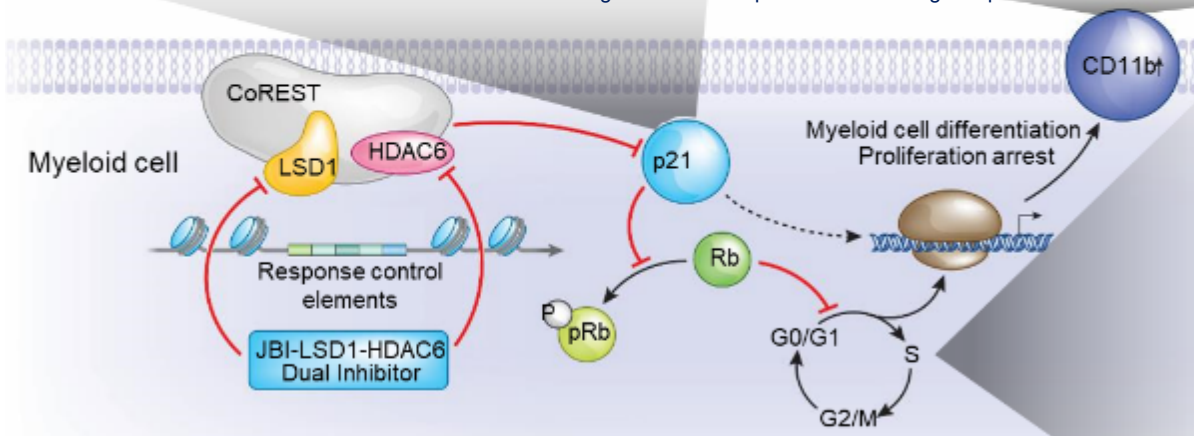


CD11b increase = differentiation



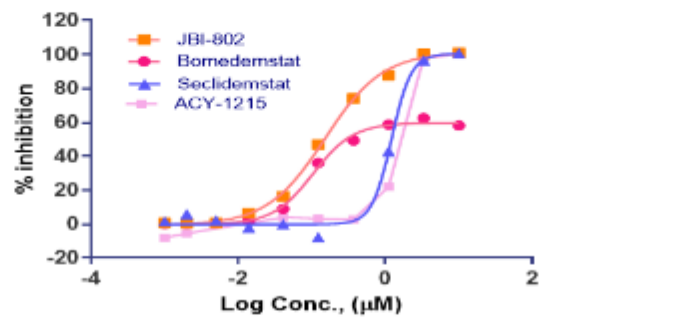
RPL13a and GAPDH are internal controls used for normalizing and accurate quantification of target expression

- Dual inhibition (LSD1 + HDAC6) delivers synergistic anti-tumor activity by directly inhibiting tumor cell proliferation and activating immune response
- HDAC6 inhibition → P21 increase → cell cycle arrest
- LSD1 inhibition → CD11b increase → myeloid cell maturation (in AML) & increase antigen presentation (increase immune response against tumors)
- Targeting superior efficacy vs. single-agent approaches



- P21 inhibits the phosphorylation of Rb protein, resulting in increased active Rb protein that prevents cell cycle from progressing from the G0/G1 to S phase, leading to cell cycle arrest.
- Increase CD11b expression pushes AML cells towards maturation/differentiation, thus stopping leukemic cell proliferation.

Cell proliferation inhibition



μM	JBI-802	Bomedemstat	Seclidemstat	ACY-1215
IC50	0.1512	0.1044	1.216	1.465

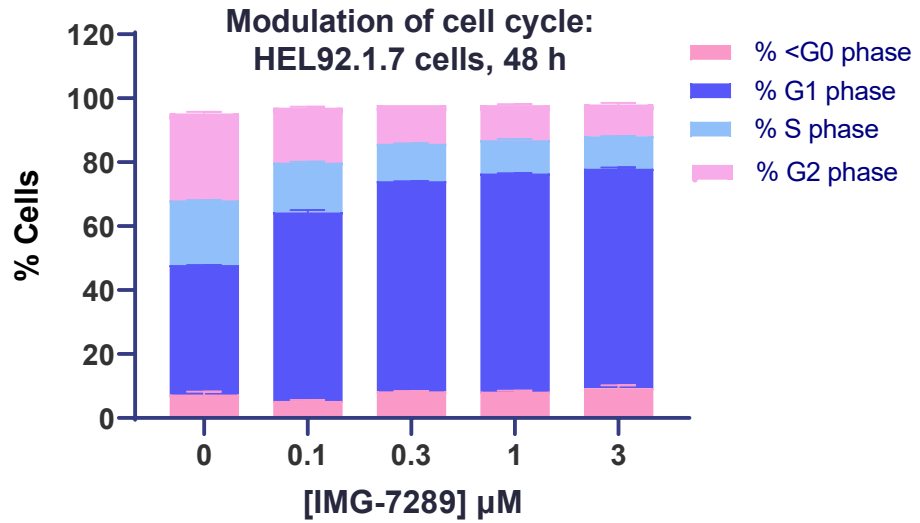
HEL92.1.7 cellular model for post-MPN AML

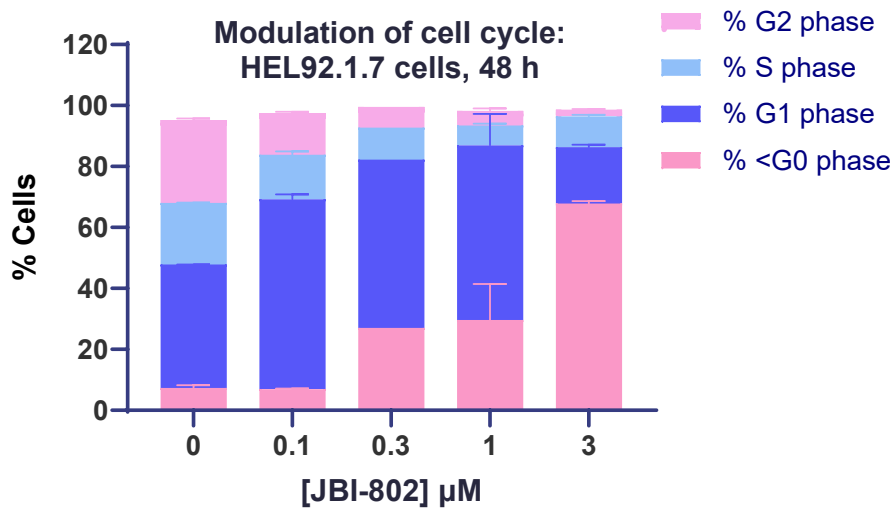
LSD1 Inhibitors: Seclidemstat, Bomedemstat
HDAC6 Inhibitor: ACY-1215
LSD1/HDAC6 Dual Inhibitor: JBI-802

JBI-802 is likely to have higher and better activity than Bomedemstat – requiring less drug in patients

Any comparisons shown here are for illustrative purposes only. No head-to-head studies have been conducted, and the data should not be interpreted as a direct comparison between product candidates.

JBI-802 May Drive Strong Apoptosis and Sub-G0 Arrest – Indicating Potential for Greater Allele Burden Reduction for Disease modification in ET/ MPN patients





- JBI-802 has shown dose-dependent G1 arrest and substantial sub-G0 population, indicating **strong apoptosis** in JAK2V617F mutant cells **(~60% of ET patients have JAK2V617F mutation)**
- Bomedemstat primarily has been shown to induce G1 arrest with only moderate apoptosis at later time points.
- **Why This Matters for VAF**: Apoptosis and sub-G0 enrichment correlate with elimination of mutant clones, suggesting JBI-802 may achieve greater VAF reduction, especially at higher doses.

JBI-802 may drive significant apoptosis and sub-G0 arrest, providing a mechanistic edge for deeper allele burden reduction at higher doses

Any comparisons shown here are for illustrative purposes only. No head-to-head studies have been conducted, and the data should not be interpreted as a direct comparison between product candidates.

JBI-802: Dual CoREST Inhibitor Has Observed Anti-Tumor Activity in NSCLC

	TNG-260 (Tango)	JBI-802 (Jubilant)
Target	**HDAC1 (Mono CoREST inhibitor)**	**LSD1/HDAC6 (Dual CoREST inhibitor)**
NSCLC patients-mSTK11	37	2
Dose	**80 mg**	**10 mg**
Dosing schedule	**7-days continuous**	**4-days on/3-days off**
Single agent outcomes	**Disease progression**	**Anti-tumor activity observed 2/2 patients**
Combination with Anti-PD-1	1 Partial response (20% ORR at efficacious dose)	Not tested (combination is expected to give better response)
Half-Life in hr	**17 hr**	**~1.5 hr**
Adverse effects->Grade 2 at dose shown	**Thrombocytopenia/anemia**	**Well tolerated with no thrombocytopenia and anaemia**
mPFS	**29 weeks**	**>104 weeks (1st patient)** **>12 weeks (2nd patient)**
mSTK11 with KRAS mutations-NSCLC	No efficacy	Not tested
Other cancer with mSTK11-pancreas, ovary, cervical etc	No efficacy	Not tested

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Any comparisons shown here are for illustrative purposes only. No head-to-head studies have been conducted, and the data should not be interpreted as a direct comparison between product candidates.
Tango Corporate Presentation Jan 2026.

BI-802 IP Portfolio Supports Protection Into 2046

Composition of matter, isomer, and method-of-use filings support long-term protection across key markets
Global IP developed in-house (i.e., not in-licensed) and wholly owned by Jubilant Therapeutics

Patent Type	Coverage	Status / Geography	Expected Expiry
Composition of Matter	Novel dual LSD1/HDAC inhibitors	Granted in U.S. and major global markets	**2042***
Composition of Matter (Isomers)	Pharmaceutically active dual LSD1/HDAC inhibitors	Granted in Eurasia; pending in U.S. and other major markets	**2043**
Method of Use	Dual LSD1/HDAC inhibitors for the treatment of myeloproliferative neoplasms	U.S. provisional filed	**2046**
Method of Use	Dual LSD1/HDAC6 inhibitors for the treatment of cancers with STK11 mutations	U.S. provisional filed	**2046**

Layered IP strategy extends coverage from core composition claims to follow-on isomer and indication-specific filings

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** Expiry date includes potential Patent Term Extension, which may extend patent life by up to 5 years*