Precision oral medicines with enhanced therapeutic index

Corporate Presentation

July 2024

Jubilant Therapeutics: A precision oral therapeutic company focused on oncology

Co-REST Inhibitor

JBI-802 Phase I preliminary clinical proof of principle established – Phase II dosing in H1 2024

- Non Small Cell Lung Cancer (NSCLC) with STK11 mutation **- overcomes immune checkpoint resistance**
- Essential Thrombocythemia (ET), Myelodysplastic/Myeloproliferative Neoplasms (MDS/MPN) with Thrombocytosis - **enables dose dependent platelet reduction**

PRMT5 Inhibitor

JBI-778 IND approved, Phase I dosing in H1 2024

- mEGFR Rx resistant NSCLC with/ without brain mets, mIDH high grade glioma, ACC **- overcomes toxicity of SAM competitive inhibitors through substrate binding, superior brain exposure, addresses larger patient population than MTA cooperative inhibitors**

Strong Discovery Capability

TIBEO (**T**herapeutic **I**ndex and **B**rain **E**xposure **O**ptimization) – structure based discovery engine to address unique patient needs unmet by existing standard of care

Proven track record of partnerships - Lengo Therapeutics (Frazier Healthcare Partners)/ Blueprint Medicines; Collaboration with Jubilant Biosys (preclinical CRO with decades of discovery expertise)

Healthy Financial Position

Backed by Jubilant Pharmova – globally diversified $1B+ healthcare organization

Highly cost efficient precision drug development model

Founded in 2019; Wholly owned assets; Opportunities to maximize partnership

**MTAP: Methylthioadenosine phosphorylase, EGFR: epidermal growth factor receptor, mIDH: isocitrate dehydrogenase (IDH) mutation, ACC: Adenoid Cystic Carcinoma, STK: serine/threonine kinase, SAM: S-adenosylmethionine*

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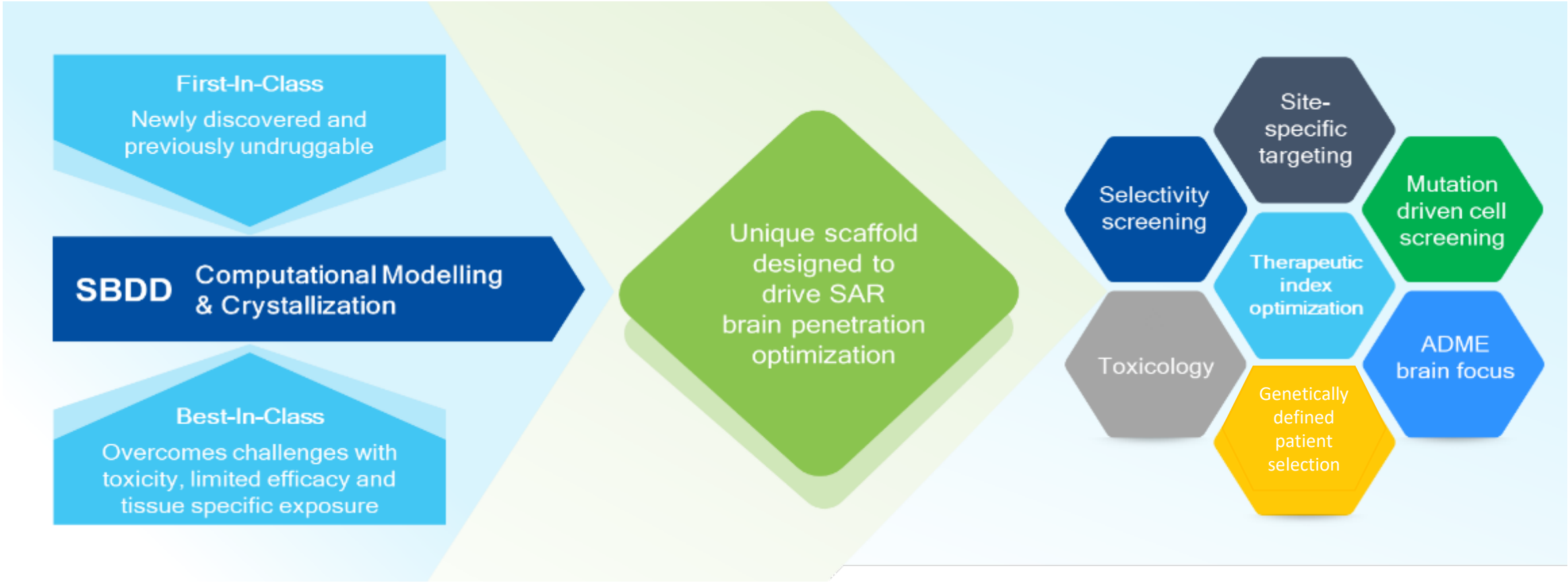
Therapeutic Index & Brain Exposure Optimization (TIBEO)



Jubilant Therapeutics' advanced methodology to develop precision drugs for genetically defined patient requirements

SBDD: Structure-based Drug Discovery; SAR: Structure activity relationship; AI: Artificial intelligence; ADME: Absorption, distribution, metabolism, and excretion

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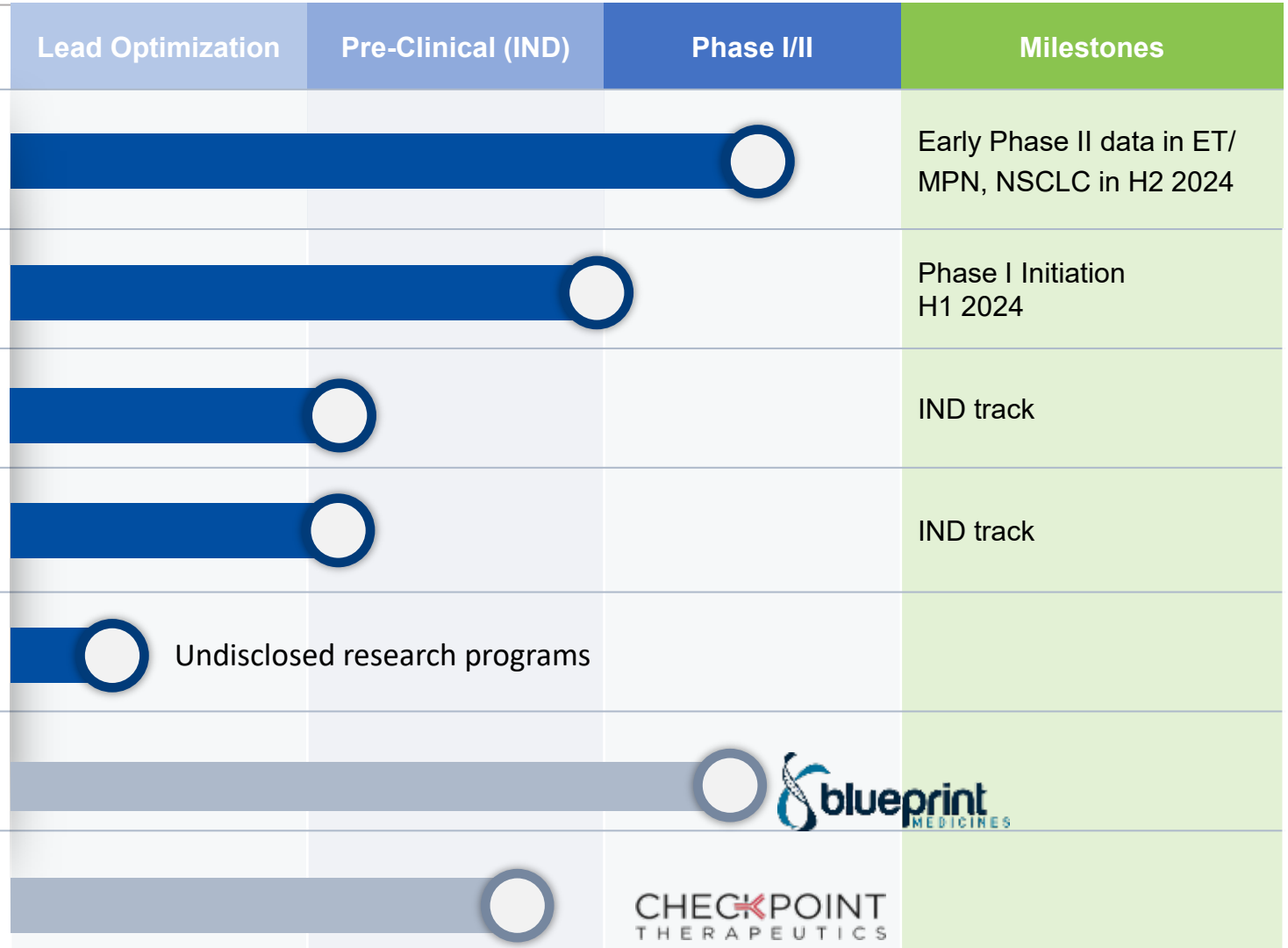
Broad Pipeline of Differentiated Assets with Improved Therapeutic Index

Program	Mechanism	Indications	Milestones
JBI-802	coREST Inhibitor	ET/MPN, NSCLC, post MPN AML	Early Phase II data in ET/ MPN, NSCLC in H2 2024
JBI-778	PRMT5 Inhibitor Brain Penetrant	EGFR mut NSCLC, mIDH high grade glioma, ACC	Phase I Initiation H1 2024
JBI-2174	PD-L1 Inhibitor Brain Penetrant	Brain tumor and metastases	IND track
JBI-1044	PAD4 Inhibitor	Oncology and auto-immune disease	IND track
Other	Various	Various	
EGFR[1,*]		Oncology	
BRD4[*]		Oncology	



- [1]Blueprint Medicines acquired Lengo Therapeutics (Frazier Healthcare entity) for $250M in cash plus $215M in milestone payments
- *Economic rights reside with Jubilant Therapeutics' parent company

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Experienced leadership team guided by strong board

Jubilant Tx Management Team

Syed Kazmi, PhD., MBA
CEO

Melda Dolan, MD
CMO

Shyam Pattabiraman
CFO

Sridharan Rajagopal
Site Head

Scientific Advisory Board

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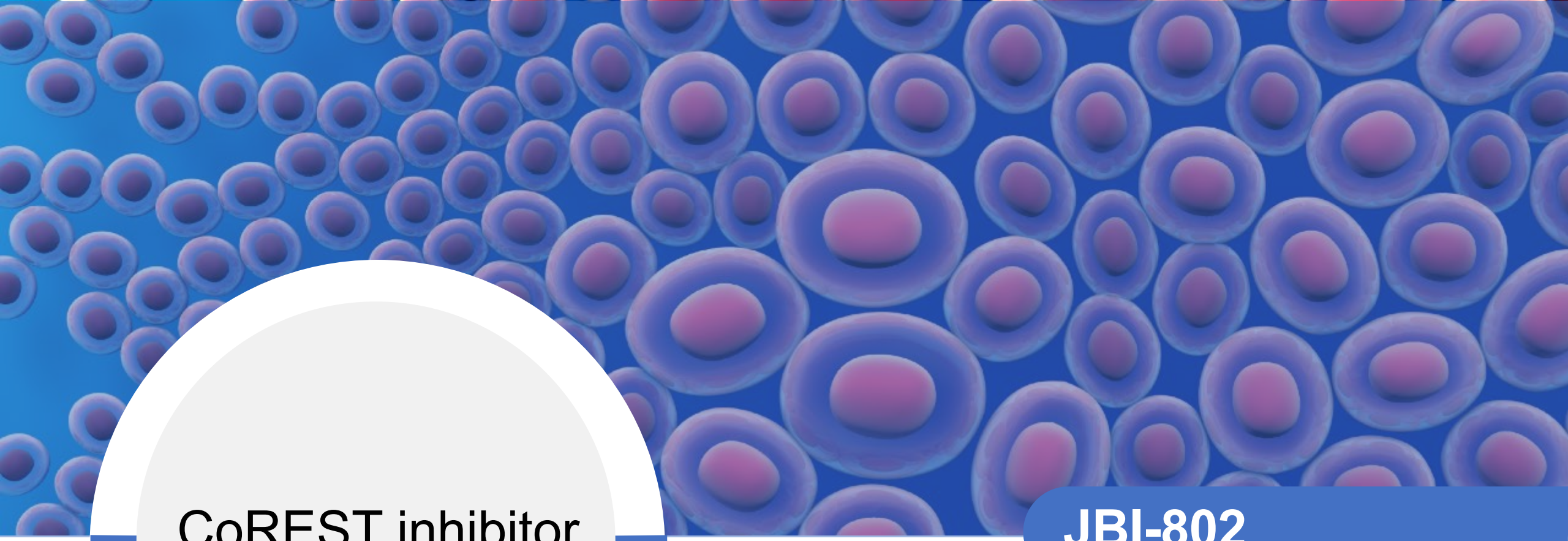


CoREST inhibitor

JBI-802

JBI-802 Highlights: Breakthrough in Epigenetic Therapy

MOA	Novel, oral, potent and selective dual inhibitor of CoREST complex through LSD1 and HDAC6 in low nM range; Targeting stem cell modulation with LSD1; Modulation of immune suppression by inhibition of LSD1 and HDAC6 with 75-100x selectivity
Differentiation	First in class mechanism of inhibiting two epigenetic targets in CoREST complex: Unique pharmacophore leading to faster clearance and better therapeutic index
Clinical opportunities	ET, MDS/MPN with thrombocytosis, NSCLC with STK11 mutation, SCLC and post MPN AML **(combined prevalence of 175k+ patients in US alone)**
Efficacy	Preliminary proof of principle observed from Phase I trials confirmed partial response with no platelet effect at lower dose (NSCLC) and dose dependent platelet reduction at higher dose (potential in heme tumors)
Safety	Favorable safety profile with no significant adverse effects
Milestones	IND accepted by FDA, Phase I proof of principle achieved, early Phase II data expected H2 2024

Clinical opportunity for JBI-802 in select solid tumors

NSCLC

- Prevalence ~ 300k (US)
- ~ 15% have STK11 mutation
- STK11 mutation causes resistance to immune checkpoint inhibitors
- **JBI-802 monotherapy treatment showed Partial response in a NSCLC patient with STK11 mutation and non responder to immune checkpoint therapy**
- Combination of JBI-802 with immune checkpoint is anticipated to have better response in the clinic

SCLC

- Small cell lung cancer (SCLC) comprises ~13–15% of all lung cancers
- 5% of patients survive for 5 years
- **JBI-802 has shown excellent efficacy in various tumor model of SCLC and modulate the key biomarkers of SCLC**
- JBI-802 achieved tumor regression in combination with PD-1 inhibitors in animal model study

Clinical opportunity for JBI-802 in select hematological malignancies

ET+

- Prevalence ~ 100k (US)
- Elevated platelets
- Current SoC Hydroxyurea toxic for long term use
- Only 50% ET cases being treated among which 25% non-responsive/ intolerant
- **LSD1 Inhibitors for ET – Merck acquired Imago for $1.35B at Phase II**
- JBI-802 shows better safety profile compared to competitors (no anemia and dysgeusia)

Post-MPN AML

- Progression from MPN to AML (Acute Myeloid Leukemia) is a serious complication, occurring in about 5-10% of MPN patients.
- No effective therapy available (Survival in adults is only 5 months)
- JBI-802 shows superior efficacy in preclinical in-vivo efficacy studies compared to LSD1 only and HDAC6 only inhibitors
- **Orphan drug designation received from FDA**

JBI-802 Phase I data suggests therapeutic potential in sensitizing immunotherapy resistant tumors and in ET/MPN with thrombocytosis

- 10 patients enrolled, last patient ongoing with ~ 1 year of durable clinical benefit

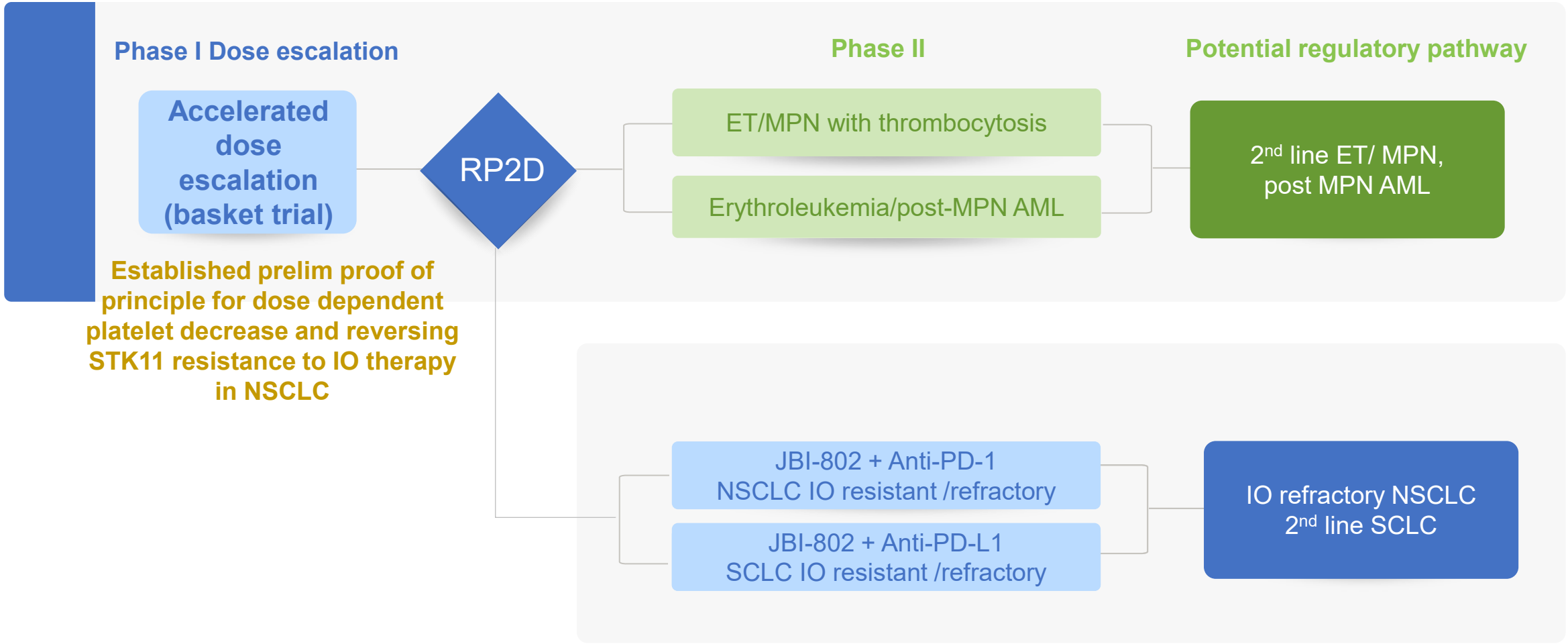
Solid tumors

- 10mg dose is safe, with no platelet decrease and shows biological activity
- 2/2 NSCLC, both IO resistant patients showed tumor-related symptoms improvements
- **Confirmed and durable PR (39% tumor reduction) in NSCLC patient with STK11 mutation, non-responder to immuno-oncology (IO) therapy; STK11 known to induce resistance to IO, KRAS therapy**
  - *"The anti-tumor activity seen in NSCLC patients is remarkable given the poor prognosis based on their genetic and metastatic pattern. The 10 mg dose of JBI-802 was well-tolerated without any clinically significant adverse effects and the initial clinical data suggest a good therapeutic index for JBI-802" – Dr. Starodub, The Christ Hospital, Cincinnati*

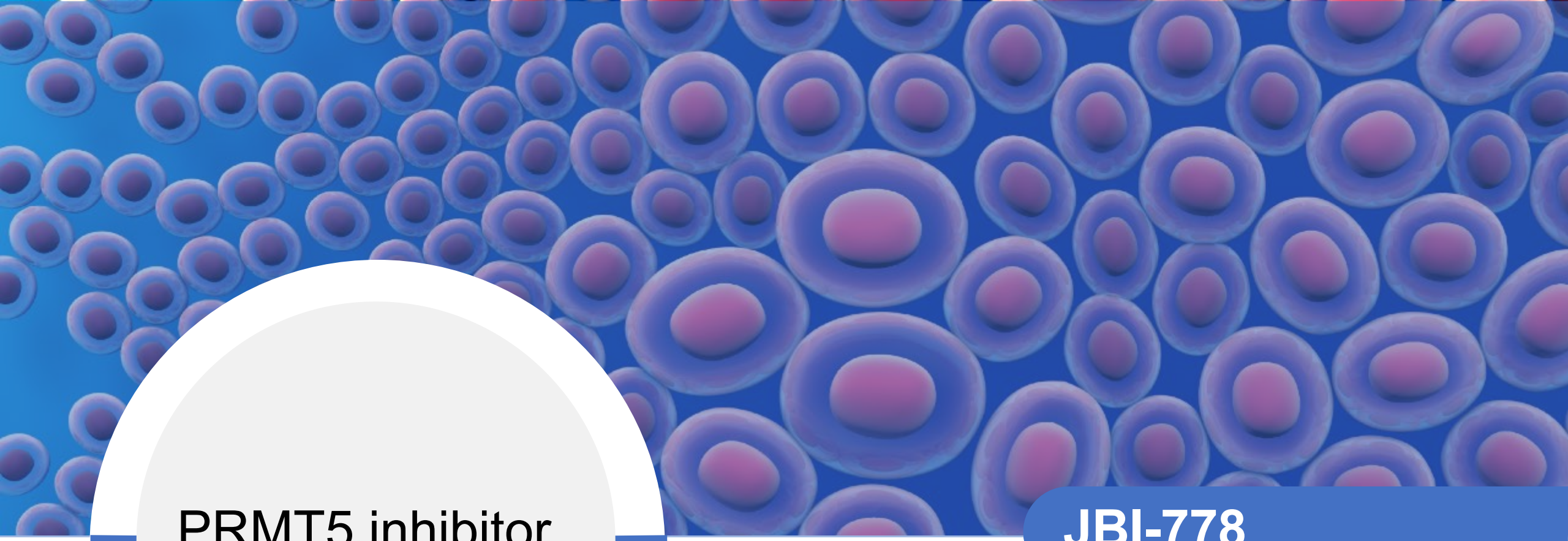
Heme tumors

- **Dose dependent platelet decrease represents proof of principle in hematological malignancies like Essential Thrombocythemia (ET) and other MPN/ MDS characterized by thrombocytosis**

JBI-802 Clinical development strategy: potential in hematologic and solid tumors



MPN: Myeloproliferative neoplasms; SCLC: Small cell lung cancer; NSCLC: non small cell lung cancer; ET: Essential Thrombocythemia



PRMT5 inhibitor

JBI-778

JBI-778 Highlights: Brain penetrant PRMT5i active in both MTAP+/- and spliceosome mutated tumors addressing significantly larger market than other PRMT5 inhibitors

MOA	Substrate competitive, SAM co-operative inhibitor of PRMT5 Synthetic lethality demonstrated in cell lines and patient cells with spliceosome mutation
Differentiation	Orally available across species with excellent brain penetration Equally potent both in MTAP wild type (wt) and MTAP null tumors in-vitro and in-vivo Spliceosome mutation based patient selection possible
Clinical opportunities	mEGFR TKI resistant NSCLC, IDH+ High grade glioma, Adenoid cystic carcinoma, MTAP +/- and spliceosome mutated tumors
Efficacy	Robust anti-proliferative activity across MTAP-wt and MTAP null cell lines In vivo efficacy shown in MTAP mut and MTAP wt models, brain orthotopic models
Safety	Well tolerated in 28-day GLP toxicity in Rat and Dog (No anemia and no changes in platelet observed at the highest dose - supported by lack of effect on HSC; No mortality or morbidity observed)
Milestones	IND approved by FDA, drug product available Clinical trial initiation in H1 2024 in EGFR mutated NSCLC, IDHmutated high grade Glioma and Adenoid cystic carcinoma

Clinical opportunity for JBI-778 in advanced solid tumors

NSCLC

- Prevalence ~ 300k (US)
- ~ 15%-20% patients have EGFR mutation of which ~ 50% develop EGFR Rx resistance; 25-30% develop brain mets during course of disease
- **Non-responders to EGFR inhibitors (such as Osimertinib) have enriched splicing mutations**
- PRMT5 Inhibitors are involved in splicing machinery and sensitive to spliceosome mutant cell line both in-vitro and in-vivo
- **JBI-778 shows strong anti-proliferation activity in NSCLC cell lines and in the brain as well**

Brain tumor

- Prevalence ~ 60k (US)
- **Isocitrate dehydrogenase (IDH) mutant gliomas are the most common adult, malignant primary brain tumors** diagnosed in patients younger than 50, an important cause of morbidity and mortality
- PRMT5 inhibitors shown CR in IDH+ patients but faced tox issues impeding further development
- **JBI-778 has superior brain exposure and substrate competitive binding has established superior safety in preclinical setting**

Adenoid Cystic Carcinoma (ACC)

- Prevalence ~ 11k (US)
- PRMT5 inhibition downregulates ACC gene signature including MYB and NOTCH1 in cancer cell lines
- **Several PRMT5 inhibitors shown favorable response in the clinic but had tox issues due to MOA**
- **JBI-778 has unique substrate competitive MOA that is safer**

Phase I initiation in H1 2024

**ACC Research foundation; Neuro-Oncology, Volume 25, Issue 1, January 2023; European Journal of Cancer, Volume 172, 2022*

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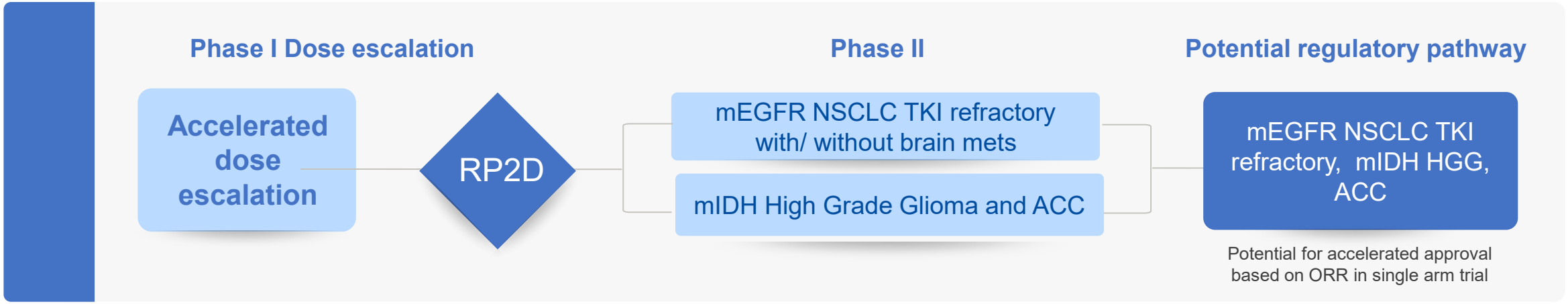
JBI-778 clinical development strategy



***Optimal dose based on PK/PD/Efficacy**

IND cleared by FDA; FIH in H1 2024

NSCLC: Non-small cell lung cancer; ACC: Adenoid cystic carcinoma; HGG: High grade Glioma; IDH: Isocitrate dehydrogenase; EGFR: epidermal growth factor receptor, TKI: Tyrosine Kinase Inhibitors

Upcoming Milestones and Catalysts