

APRIL 2023

# CORPORATE PRESENTATION

**A P T O S E**  
BIOSCIENCES

PRECISION ONCOLOGY FOR THERAPIES OF TOMORROW

NASDAQ: **APTO**  
TSX: **APS**

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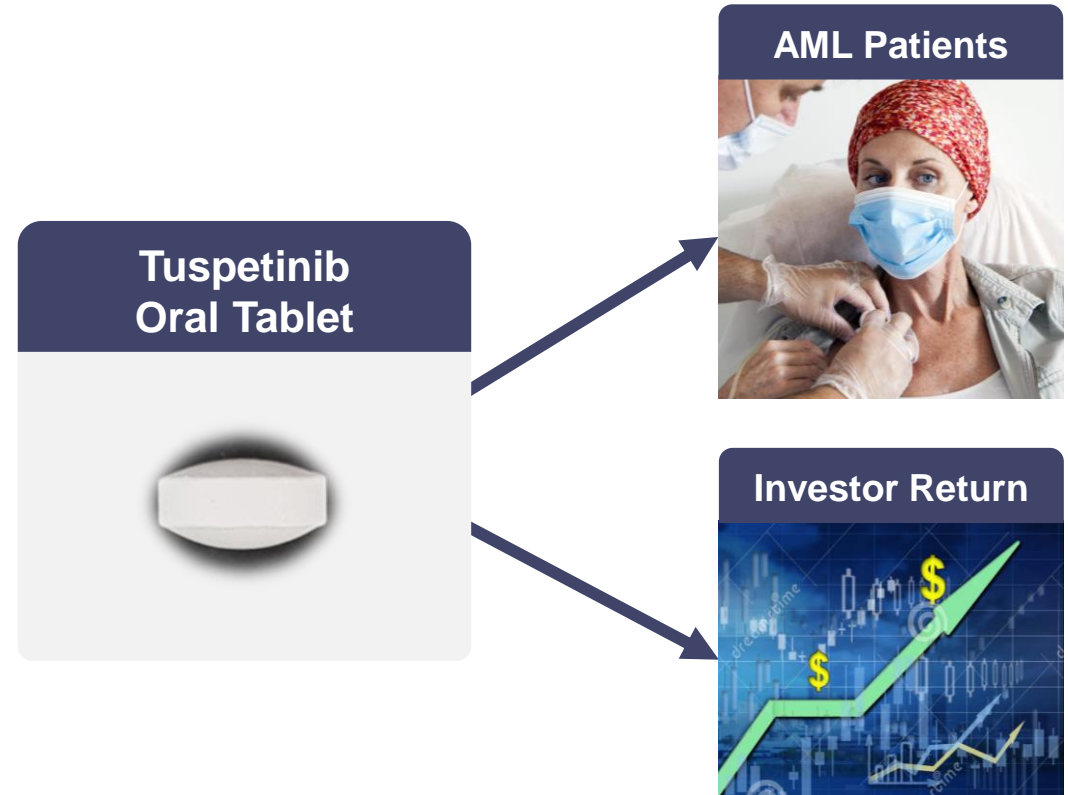
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**Aptose is a precision oncology company developing oral targeted agents to treat hematologic malignancies**



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## **Aptose Investment Highlights**

**Tuspetinib (Tus) myeloid kinase inhibitor: safe and effective, once daily, oral agent to treat AML**

- CRs across 4 dose levels with no DLT
- Favorable safety and non-myelosuppressive
- Broadly active across diverse AML populations
- Accelerated approval paths as monoRx and doublet
- Ideal for 1L triplet therapy and maintenance therapy
- Orphan Drug and Fast Track Status
- \$1B+ market potential

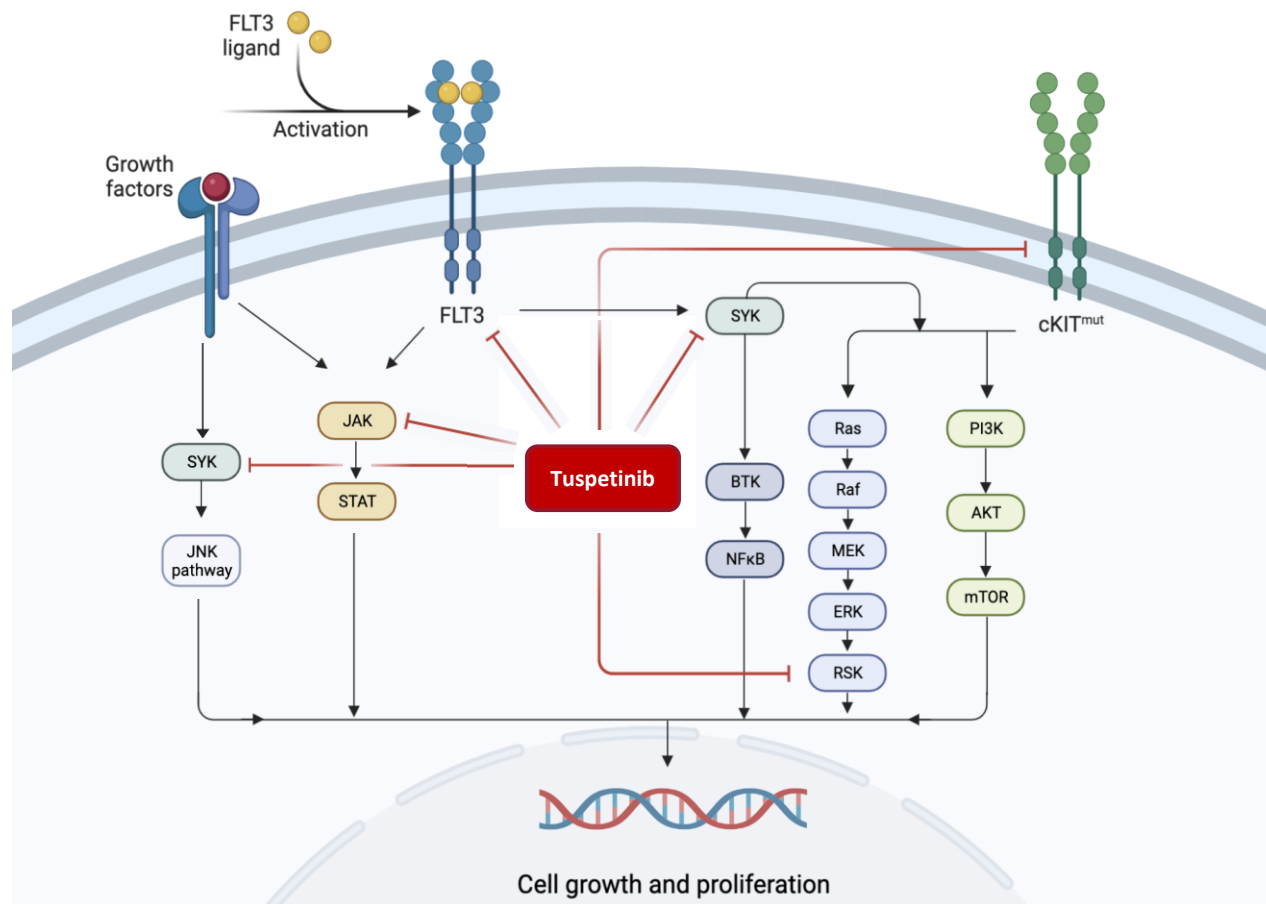
**Luxeptinib (Lux) lymphoid & myeloid kinase inhibitor**

- Clinically active in AML and B-cell cancers
- Exploring new formulation with improved absorption

**Value-driving near-term clinical milestones during 2023**

# Tuspetinib simultaneously targets clinically validated kinases in oncogenic signaling pathways in AML

*Avoids the need to fully suppress any single target – Avoids typical toxicities of other agents*



## Multi-drug therapy in a single tablet

- Uniquely and potently targets **FLT3, SYK, JAK1/2, cKIT<sup>MUT</sup>, and RSK**
- Suppresses multiple dysregulated signal transduction pathways that drive AML proliferation and resistance
- Ideal for **monotherapy, combination therapy, and maintenance therapy**

# Building a long-term strategy for tuspetinib blockbuster potential

Addresses multiple AML patient populations and commercial opportunities

## Monotherapy in Prior FLT Inhibitor Failures

Potential for Accelerated Approval in r/r AML

## Doublet Combination in 2L AML

Potential for Accelerated Approval with Interim Data Analysis

## Triplet Combination for 1L AML

## Maintenance Therapy Post-CR

**Potential Annual Sales\*  $\geq$  \$1B**

Near Term

Long Term

## Tuspetinib blockbuster potential

- Delivers potent single agent CRs among refractory AML regardless of adverse mutation status
- CRs among wildtype FLT3 patients, representing 70% of AML population
- Avoids typical toxicities of other kinase inhibitors, including myelosuppression
- Paths identified for accelerated approval
- Ideal for oral maintenance & combination therapy representing significant markets

# Tuspetinib **monotherapy** in r/r AML who failed prior FLT3i

## Accelerated approval path with fast-to-market opportunity

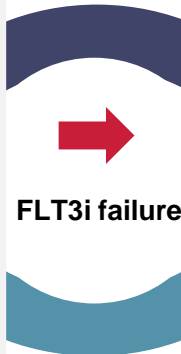
**FLT3<sup>mut</sup> AML failed prior FLT3 inhibitors**  
(3L+ r/r AML population with no approved options):

Gilteritinib (Xospata<sup>®</sup>) *FY 2022E sales: ~\$355M\**

Midostaurin (Rydapt<sup>®</sup>)

Sorafenib (Nexavar<sup>®</sup>)

Quizartinib



### Tuspetinib Monotherapy

- Active against all forms FLT3 & other targets
- Responses in patients who failed prior FLT3i
- Potential to address an unmet medical need
- Fast-to-market strategy in US

**Accelerated development strategy may offer more rapid value creation**

# Tuspetinib **doublet** therapy in 2L AML

Accelerated approval opportunity with large commercial potential

## 2L AML failed all 1L agents (all comer):

- Failed chemotherapy
- Failed 1L HMA + Ven
- Failed 1L FLT3 inhibitor

1L failure

## Tuspetinib + venetoclax doublet therapy

- Active in AML with **FLT3<sup>MUT</sup>**
- Active in **larger AML population** with **FLT3<sup>WT</sup>**
- Potential to address an **unmet medical need**
- Potential for interim analysis and **early approval**
- Option for confirmatory regulatory strategy in US

**Accelerated development strategy may offer more rapid value creation**



# New era of **triplet** therapy with targeted agents for 1L AML creates a significant commercial opportunity for tuspetinib

## Triplet with Targeted Agents for AML | Analogous to HIV Cocktail Therapy

Blood Cancer Journal

www.nature.com/bcj

ARTICLE OPEN

Check for updates

Hypomethylating agent and venetoclax with FLT3 inhibitor  
“triplet” therapy in older/unfit patients with *FLT3* mutated AML

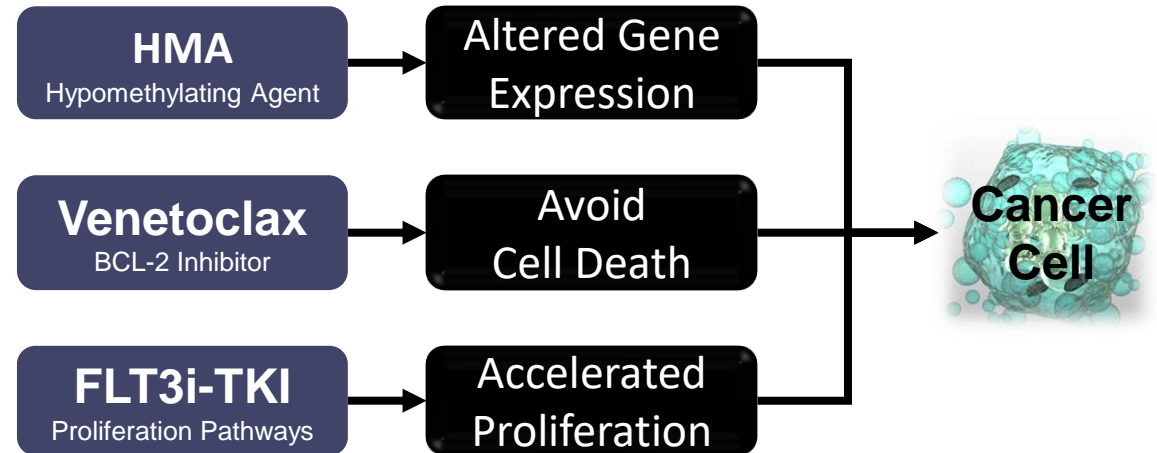
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**Improves CR/CRh/CRi to >90%**

**Improves MRD-negative status**

**Improves OS (survival) and gives hope**



# Tuspetinib for **triplet** therapy in 1L AML due to favorable safety profile

## Large commercial opportunity

### Current triplet: HMA + Venetoclax + FLT3i

**HMA:** Hypomethylating agent

**Venetoclax:** BCL-2 inhibitor (2022 sales: \$2.9B)\*

**FLT3i:** Current FLT3 inhibitors

#### Problems with current triplet: HMA + Venetoclax + FLT3i-TKI

Safety issue with QTc prolongation

Prolonged myelosuppression

Limited breadth of antileukemic activity



### Ideal triplet: HMA + Venetoclax + Tuspetinib

**HMA:** Hypomethylating agent

**Venetoclax:** BCL-2 inhibitor



**Tuspetinib:** Myeloid kinase inhibitor

#### Solution for ideal triplet: HMA + Venetoclax + **Tuspetinib**

**No observed cardiotoxicity**

**No observed differentiation syndrome**

**No myelosuppressive with continuous dosing**

**Active on FLT3<sup>MUT</sup> and FLT3<sup>WT</sup>**

# Tuspetinib **maintenance** therapy post-CR

## Large commercial opportunity

### Patients who achieve a CR

- CR post-Tus Monotherapy
- CR post-Tus triplet Therapy
- CR post-Chemotherapy
- CR post-HSCT

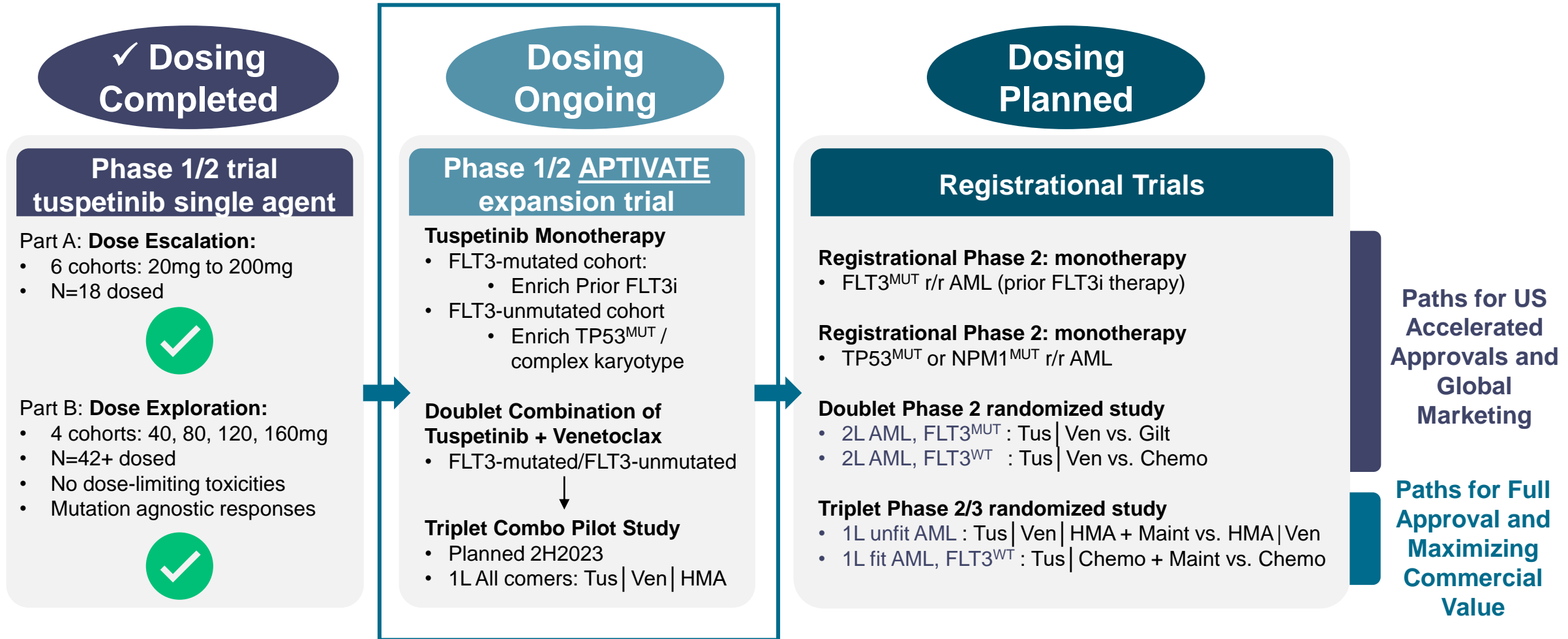


### Tuspetinib maintenance therapy

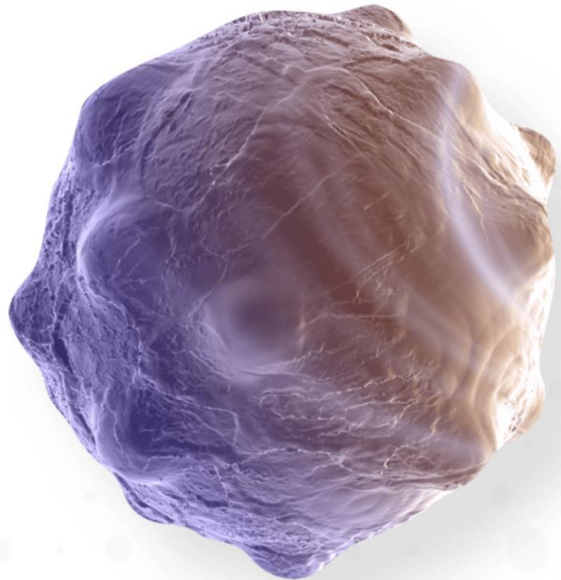
- Tus monotherapy or Tus|Ven or Tus|HMA
- Applicable to larger FLT3<sup>WR</sup> or MUT AML populations
- Maintain patients long term MRD-negative CR
- Extend disease free state and survival

**Maintenance strategy may offer significant long-term value creation**

# Pathway to commercialization: global APTIVATE dose expansion trial ongoing to support registrational studies for accelerated approval



"Fit & unfit patients": The AML subtype, cytogenetics, and molecular features are essential for a patient's prognosis. These can influence the treatment choice. However, the therapeutic choice is also influenced by the physician-assessed "fitness" of the patient's ability to tolerate intensive chemotherapy.



## **Initial Results from the Tuspetinib Phase 1/2 Clinical Trial**

**Relapsed or Refractory AML**



# Tuspetinib Phase 1/2 Study in r/r AML: Dose Escalation & Dose Exploration Completed

## Dose Escalation 18 patients dosed

Cohort 1: 20 mg QD ✓ Completed

Cohort 2: 40 mg QD ✓ Completed

Cohort 3: 80 mg QD ✓ Completed

Cohort 4: 120 mg QD ✓ Completed

Cohort 5: 160 mg QD ✓ Completed

Cohort 6: 200 mg QD ✓ Completed

**Favorable, non-myelosuppressive safety profile across six dose levels:**

- No drug-related SAE or deaths
- No drug-related QTc prolongation
- No DLT through 160 mg dose level
- Plasma  $t_{1/2}$  estimated at 40hrs



## Dose Exploration 42 patients dosed

40 mg QD CRs No DLT Dosing

80 mg QD CRs No DLT ✓ Completed

120 mg QD CRs No DLT ✓ Completed

160 mg QD CRs No DLT ✓ Completed



**Dose escalation and dose exploration completed across six dose cohorts:**

- Total patients dosed in Part A + Part B = 60
- Total evaluable for efficacy in Part A + Part B = 48
- Total evaluable for efficacy at 80/120/160mg = 42
- Additional patients being placed on 40mg dose level

# Phase 1/2 dose escalation and exploration - patient characteristics

Patient Characteristics	
<b>Demographic:</b>	<b>N=60 (%)</b>
Male	35 (58.3%)
<b>Race:</b>	
Asian	32 (53.3%)
White	22 (36.7%)
<b>Median age</b>	61 (range: 18-83)
<b>FLT3 Mutation Status</b>	
FLT3 <sup>MUT</sup>	26 (43.3%)
FLT3 <sup>WT</sup>	33 (55.0%)
Unknown	1 (1.7%)
<b>Prior Lines of AML Therapy - Mean (range)</b>	2.7 (1 to 8)
<b>Type of Prior Therapy</b>	<b>N (%)</b>
Prior Drug Therapy (Chemotherapy/Not Radiation)	60 (100%)
Cytotoxic Chemotherapy	43 (71.7%)
HMA	36 (60.0%)
Venetoclax	30 (50.0%)
HSCT	17 (28.3%)
FLT3 Inhibitor	14 (23.3%)

## Dose escalation and exploration completed across six dose cohorts

- **As of 10/6/22, 60 patients treated across 6 dose levels**
- **Patients heavily pre-treated**
  - Cytotoxic chemotherapy (72%)
  - HMAs (60%)
  - Venetoclax (50%)
  - HSCT (28.3%)
- **Half (50%) of FLT3<sup>MUT</sup> patients failed prior FLT3 inhibitor**

# Tuspetinib favorable safety profile and broad therapeutic window

## Favorable Safety Profile

- No drug-related myelosuppression
- No drug related AE of QTc prolongation
- No observed differentiation syndrome
- No drug related SAE, deaths, or discontinuations
- No DLT from 20 mg level through 160 mg level
- One DLT of muscle weakness at 200 mg
  - Reversibility in patient with high exposure
  - Not rhabdomyolysis | No muscle destruction
  - No AE of elevated creatine phosphokinase (CPK)
- Avoids many of the typical toxicities observed with other TKI and menin inhibitors

## Treatment-emergent AEs (TEAEs), Safety Analysis Set, Parts A and B (N=60)

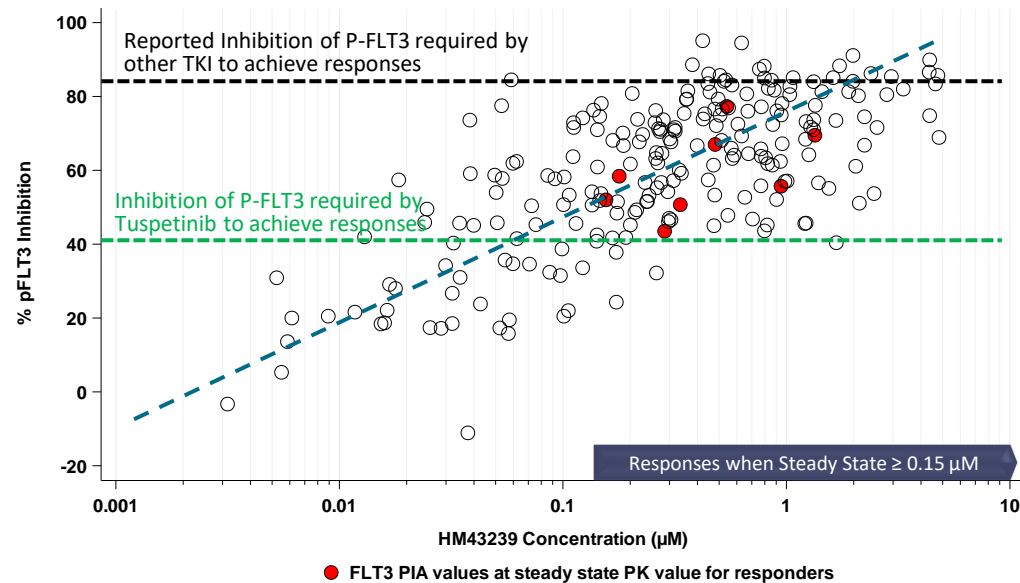
Patients Experiencing TEAEs	N (%)
Any	56 (93.3%)
Most Frequent TEAEs (>15% of patients)	
Pneumonia	18 (30.0%)
Pyrexia	12 (20.0%)
Nausea	11 (18.3%)
Diarrhea	9 (15.0%)
≥ Grade 3	41 (68.3%)
SAEs	31 (51.7%)
Leading to treatment discontinuation	6 (10.0%)
Leading to death	11 (18.3%)
Patients Experiencing TEAEs Related to HM43239	
Any	17 (28.3%)
Most Frequent Related TEAEs (>5% of patients)	
Diarrhea	7 (11.7%)
Nausea	5 (8.3%)
≥ Grade 3	6 (10.0%)
Decreased neutrophil count	2 (3.3%)
Muscle weakness	2 (3.3%)
Decreased white blood cell count	1 (1.7%)
Nausea	1 (1.7%)
Leukopenia	1 (1.7%)
SAEs	0 (0%)
Leading to death	0 (0%)
Dose Limiting Toxicity (DLT)*	1 (1.7%)

# Tuspetinib in patient plasma inhibits multiple kinase targets

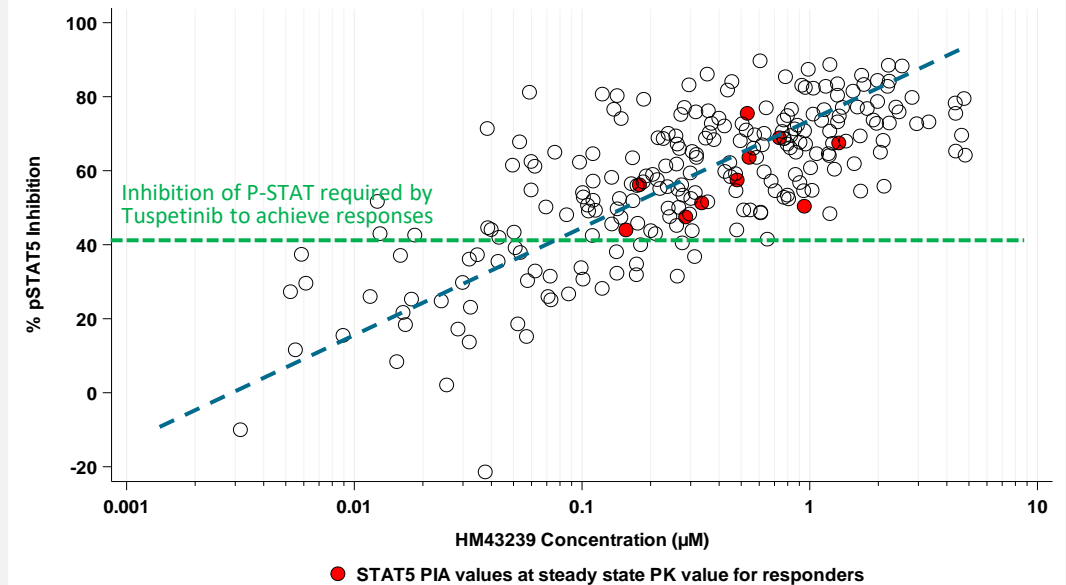
## Full inhibition of each target is not required

*Lower doses needed for responses = fewer toxicities*

### Inhibition of FLT3 activity Measure P-FLT3 in MOLM-14 AML Cells By Patient Plasma in PIA Assay

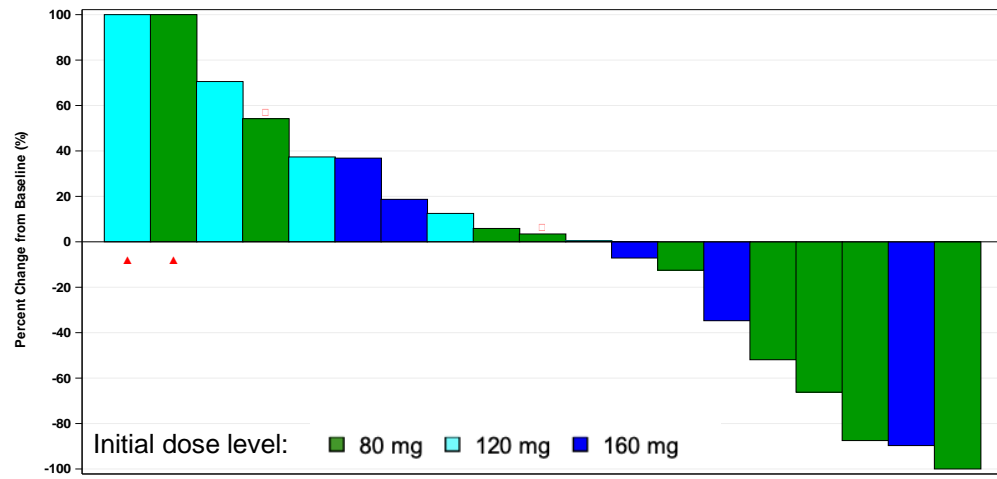


### Inhibition of JAK activity Measure P-STAT5 in MOLM-14 AML Cells Patient Plasma in PIA Assay

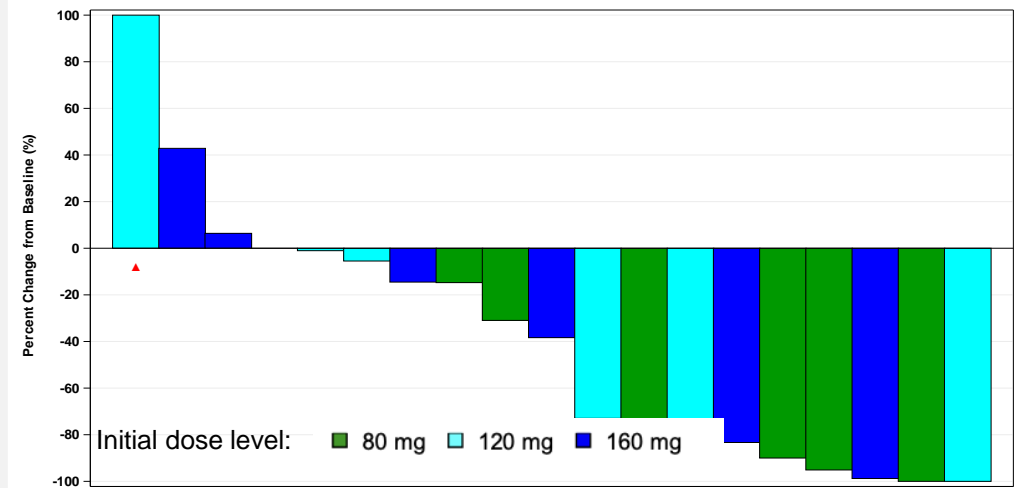


# Clinical activity: bone marrow blast reductions achieved across multiple dose levels in both FLT3<sup>WT</sup> patients and FLT3<sup>MUT</sup> patients

## Blast Reductions | FLT3<sup>WT</sup> Patients



## Blast Reductions | FLT3<sup>MUT</sup> Patients

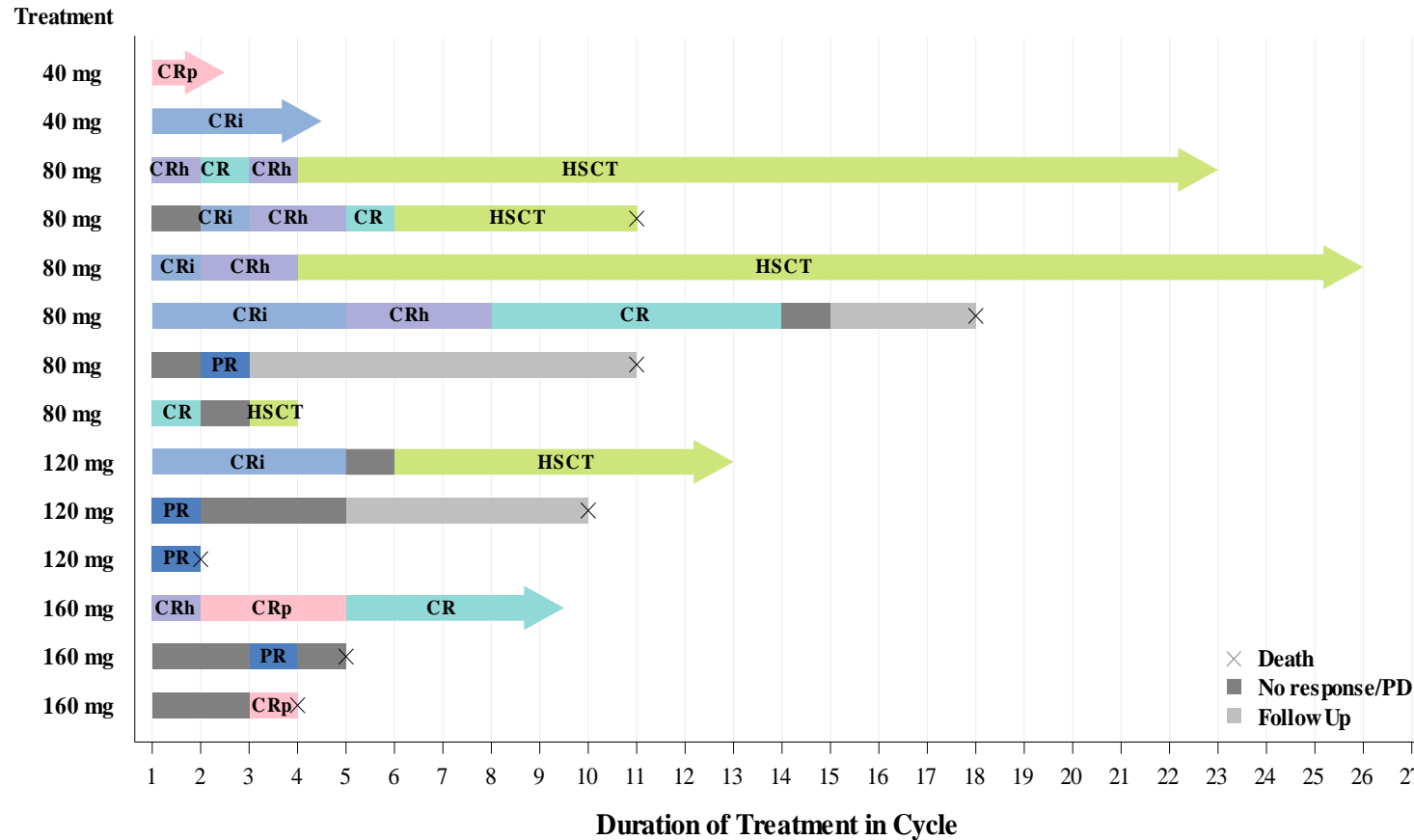


- Blast reductions observed in heavily pretreated r/r AML patients across multiple dose levels
- Several CR achieved with blast clearance accompanied by full recovery of normal blood cells
- Meaningful bone marrow blast reductions highlight the potential of tuspetinib to reach a CR when combined with hypomethylating agents, venetoclax, or other active therapies

\*Indicates patients who administered hydroxyurea within 7 days prior to the lowest marrow blast value.  
 †Indicates patients with percent change from baseline > 100% are shown as 100% and indicated with a triangle.



# R/R AML patients achieving clinical responses with tuspetinib monotherapy



## Responder Analysis

- Responses achieved across four dose levels
- Responses mature over time with ongoing continuous dosing
- Many bridged to potentially life-saving transplant (HSCT)
- Durability observed when no HSCT unavailable

Executed: 08FEB2023

Abbreviation: CR, complete response; CRh, complete response with partial hematologic recovery; CRi, complete response with incomplete hematologic recovery; CRp, complete response with incomplete platelet recovery; HSCT, hematopoietic stem cell transplantation; ND, not done; NE, not evaluable; PD, progressive disease; PR, partial remission.

Note: 'No response' includes refractory or not done. The right arrow at the end of horizontal bar indicates patients are still on study, whereas without the right arrow indicates patients discontinued from study.

Note: The bone marrow aspiration/biopsy date was used as response date. Each response assessed at a regular visit is considered to have started 1 cycle before the assessment; the start of the response is considered the integer part of (study day/28) if the response occurred at the End of Treatment visit.

\*Indicates patients who received prior FLT3 inhibitors, including gilteritinib and/or midostaurin.

# Tuspetinib safely delivers monotherapy responses across diverse AML populations

Pt.	Mutations present							Dose Level	Best Response
	FLT3 <sup>MUT</sup>	RAS	NPM1	DNMT3A	RUNX1	IDH	Other		
1							TP53	80mg	CR
2							TP53, TET2	40 mg	CRp
3	X	X					RUNX1	80mg	CRh
4		X					U2AF1, BCOR, SETBP1	160mg	CR
5	X	X	X	X			PTPN11	120mg	PR
6	X		X	X				80mg	CR
7	X		X					160mg	CRp
8	X		X	X		X		160mg	PR
9						X	SRSF2	80mg	CR
10	X				X		SF3B1, RB1	80mg	CR
11	X				X		MLL-PTD	120mg	CRi
12							Not yet reported	40mg	CRi
13	X						Not yet reported	120mg	PR
14							ASXL1, CBL	80mg	PR

## Mutation Response Analysis

Responses across populations with highly adverse mutations: TP53, RAS, NPM1, FLT3, IDH, DNMT3A, RUNX1, MLL genes

37.5% of CRc Responders are FLT3<sup>WT</sup> (3 of 8)

TP53<sup>MUT</sup> / complex karyotype responders

# Tuspetinib delivers durable complete responses in Phase 1a trial

## Best efficacy response in evaluable r/r AML 80 mg | 120 mg | 160 mg

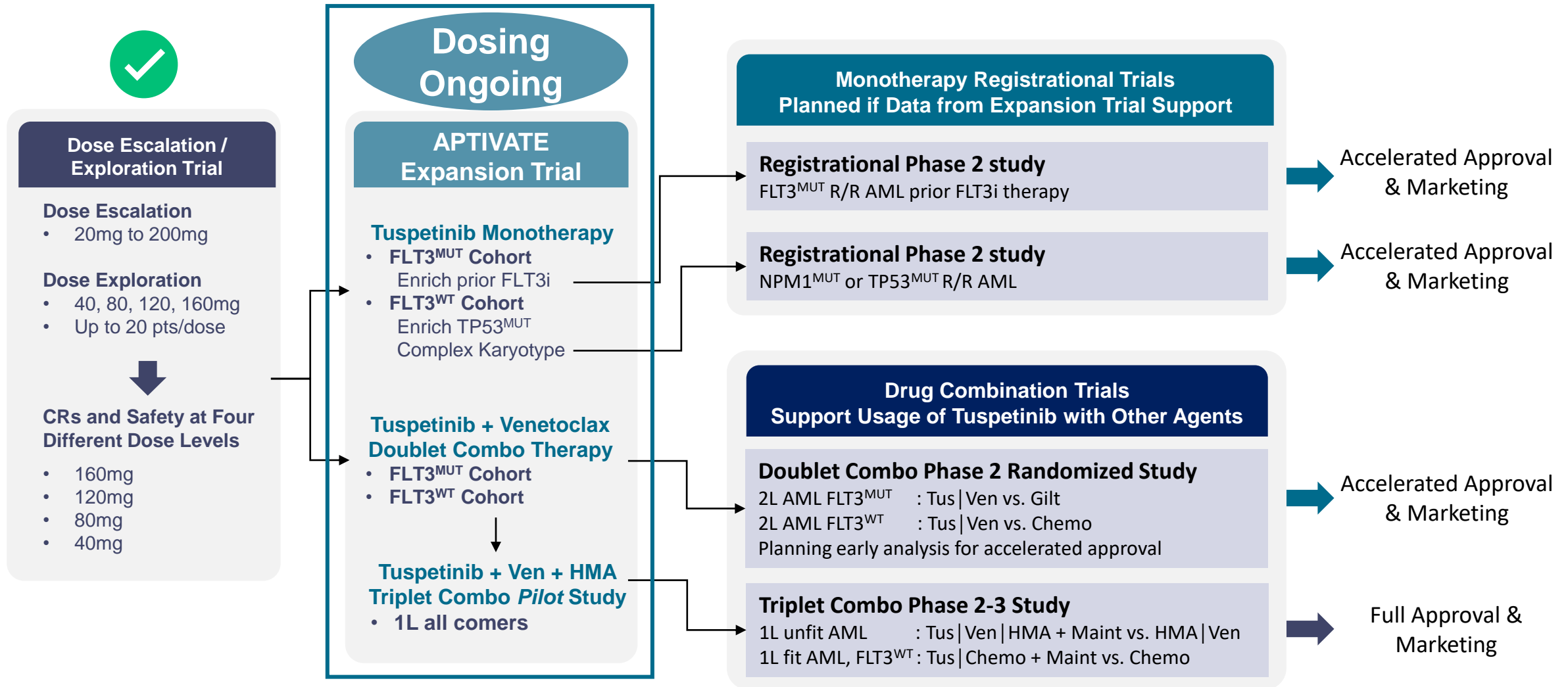
Best Response:	RAS <sup>MUT</sup> n=7	FLT3 <sup>MUT</sup> n=21	FLT3 <sup>WT</sup> n=21	FLT3 <sup>MUT</sup> + prior FLT3i n=11	ALL n=42
CRc	2 (28.6%)	5 (23.8%)	3 (14.3%)	2 (18.2%)	8 (19.0%)
CR/CRh	2 (28.6%)	3 (14.3%)	3 (14.3%)	1 (11.1%)	6 (14.3%)
ORR	3 (42.9%)	8 (38.1%)	4 (19.0%)	3 (27.3%)	12 (28.6%)



## Summary of Efficacy

- ✓ ORR: 29% at dose levels advanced to ACTIVATE expansion trial
- ✓ Responses in heavily pretreated patients (mean 2.7 prior therapies | 3L+)
- ✓ Responses achieved across four dose levels
- ✓ Many responders bridged to potentially life-saving transplant (HSCT)
- ✓ Responses across populations with highly adverse mutations including FLT3<sup>WT</sup> patients
- ✓ Responses in FLT3<sup>MUT</sup> patients who failed prior FLT3 inhibitors

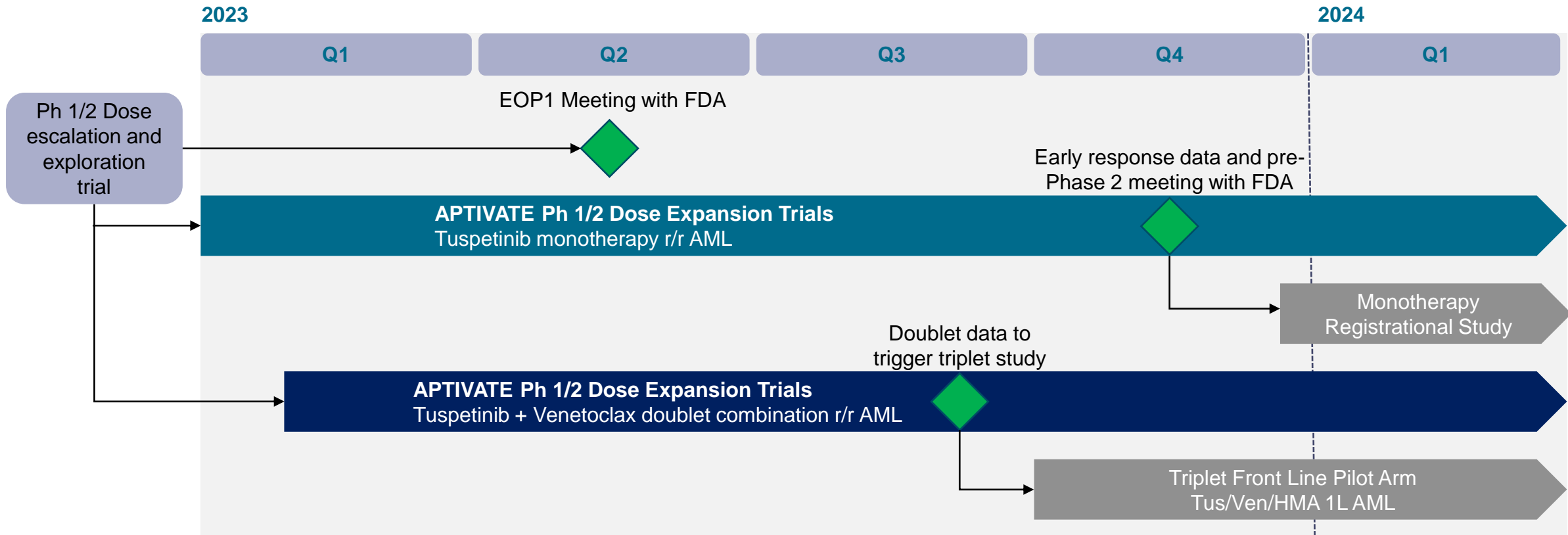
# Tuspetinib APTIVATE global expansion trial ongoing to support registrational trials for accelerated approval and drug combination trials



Single agent Expansion studies designed to collect data on a small number of patients in "high need" groups and segue into Ph 2 Registrational Trial(s)

Combination Expansion studies designed to illustrate safety and efficacy of Tuspetinib with venetoclax and segue into Phase 2-3 randomized trials and demonstrate Tuspetinib can be the preferred agent for combination therapy

# Tuspetinib APTIVATE trial delivering value-creating milestones in 2023



Anticipate data from monotherapy, doublet therapy and triplet therapy during 2023



# Tuspetinib best-in-class TKI for AML

	Tuspetinib	Gilterinib	Quizartinib	Emavusertib	Revumenib	Ziftomenib	Lanraplenib
<b>Targets:</b>	SYK, JAK1/2 FLT3 <sup>ITD/TKD/WT</sup> c-KIT <sup>MUT</sup>	FLT3 <sup>ITD/TKD</sup>	FLT3 <sup>ITD</sup>	IRAK4/FLT3	Menin	Menin	SYK
<b>Safety: broad Tx window</b>	✓	—	✗	✗	—	—	—
<b>Avoids QTc prolongation</b>	✓	✗	✗	✓	✗	✓	✓
<b>Avoids differentiation syndrome</b>	✓	✗	—	✓	✓	✗	✓
<b>Single agent efficacy in AML</b>	✓	✓	—	✓	✓	✓	—
<b>Potential beyond AML</b>	✓	✗	✗	✓	—	—	✗

# Tuspetinib ideal TKI for triplet combination to treat 1L AML

**Ideal Triplet**

- HMA:**  
hypomethylating agent
- +**
- Venetoclax:**  
BCL-2 inhibitor
- +**
- Tuspetinib:**  
myeloid kinase inhibitor

	Tuspetinib	FLT3 Inhibitors		Menin Inhibitors	
		Gilterinib	Quizartinib	Revumenib	Ziftomenib
<b>Targets:</b>	SYK, JAK1/2 FLT3 <sup>ITD/TKD/WT</sup> c-KIT <sup>MUT</sup>	FLT3 <sup>ITD/TKD</sup>	FLT3 <sup>ITD</sup>	Menin	Menin
<b>Avoids myelosuppression</b>	✓	✗	✗	—	—
<b>Avoids QTc prolongation</b>	✓	✗	✗	✗	✓
<b>Avoids differentiation syndrome</b>	✓	✗	—	✓	✗
<b>CRs in FLT3<sup>MUT</sup> and FLT3<sup>WT</sup></b>	✓	✓	—	✓	✓

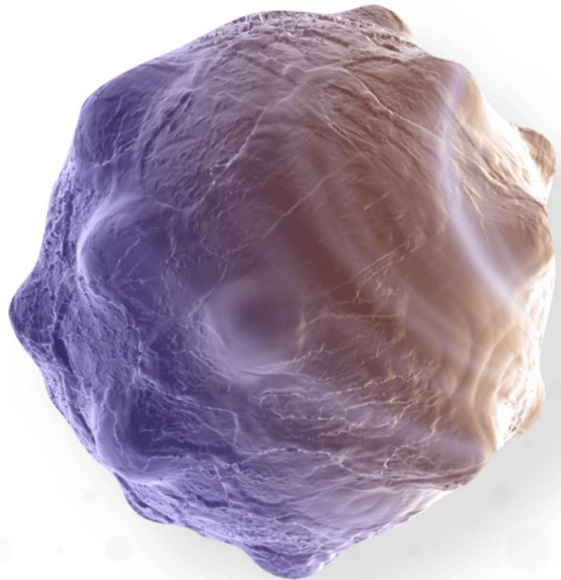
# Tuspetinib vision of “Firsts” that increase survival at each stage of AML

## Tuspetinib is positioned to be 1<sup>st</sup> in the next wave of innovation in AML

Recently approved targeted agents have run their course of the current wave of innovation in AML – their lifecycle is limited by their expected LOE expiration

The patent runway, efficacy profile and safety profile position tuspetinib potentially to become the first regulatory approved drug at each stage of the disease (1L, 2L, post-CR, and deep relapsed/refractory)

r/r AML	1 <sup>st</sup> approved targeted agent in FLT3+ patients who have failed prior FLT3i
2L AML	1 <sup>st</sup> approved targeted agent in Tus+VEN doublet in 2L AML patients
Frontline (1L) AML	1 <sup>st</sup> approved targeted agent in Tus+VEN+HMA triplet in fit & unfit patients
Post-CR Maintenance	Targeted agent of choice in patients with CR following chemo or HSCT



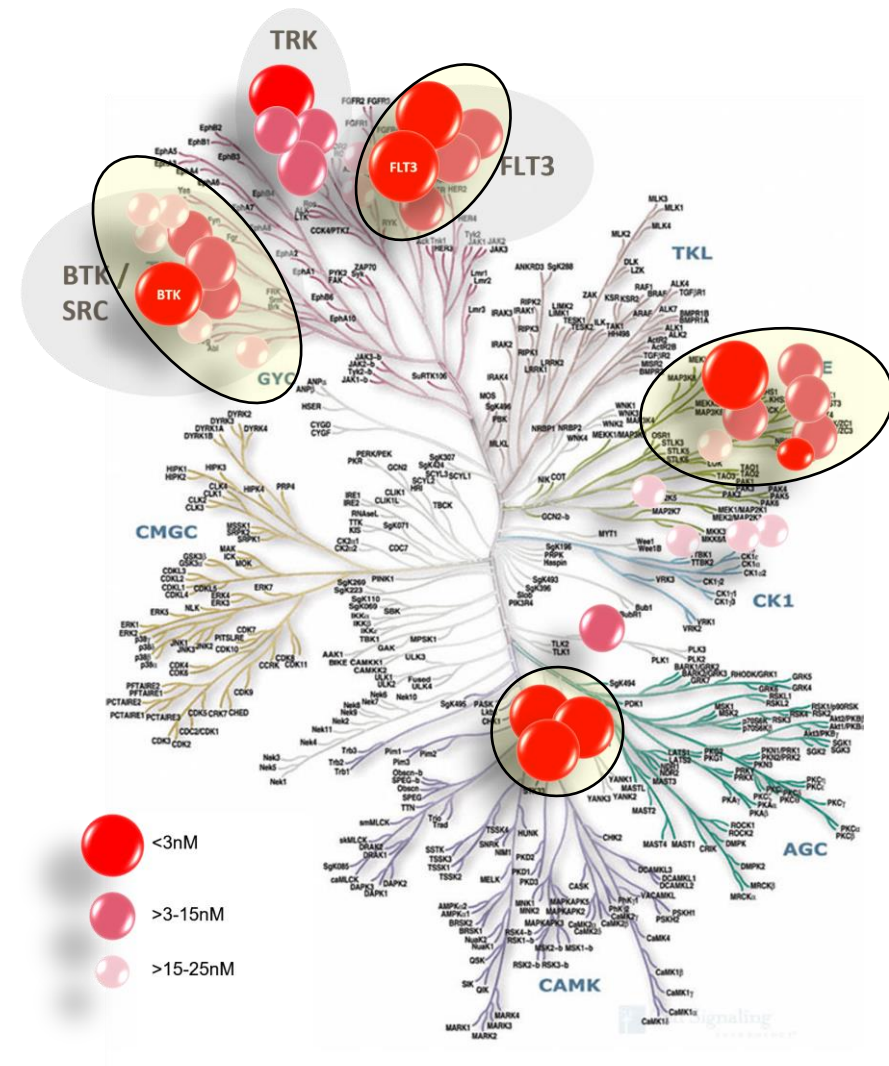
**Luxeptinib (Lux)**

**Oral Lymphoid & Myeloid  
Kinase Inhibitor**

# Luxepatinib: Clinical activity achieved in AML and B-cell cancer patients

## Potent suppression of multiple kinases driving AML and B-cell cancers

- Inhibits BTK, FLT3, CSF1R, PDGFR $\alpha$ , TRK, AURK, and others
- Well-tolerated with dosing at 900mg BID with G1 original formulation
- Antitumor activity in diverse B-cell cancers
- Delivered CR (MRD-) in r/r AML patient
- Exploring G3 new formulation with improved absorption and pharmacokinetics

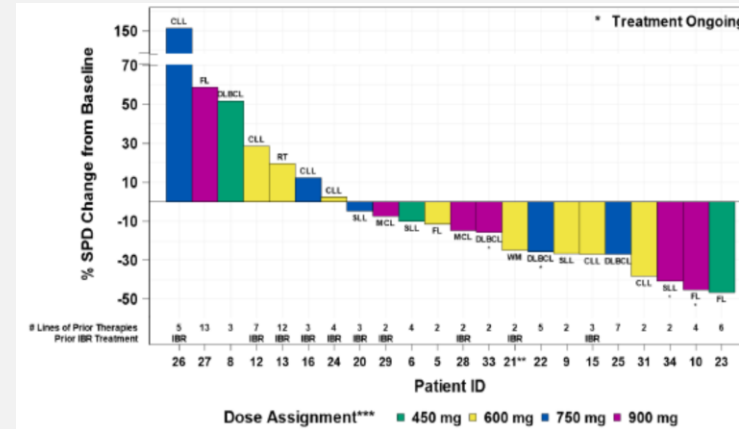


# Luxeptinib: demonstrated clinical activity but needs improved PK

Phase 1a/b in  
r/r B-cell leukemias and lymphomas  
36 patients dosed



## Best responses in evaluable B-cell malignancy patients



✓ CR in DLBCL

Phase 1a/b in  
r/r AML and MDS  
29 patients dosed



## Best responses in evaluable R/R AML patients

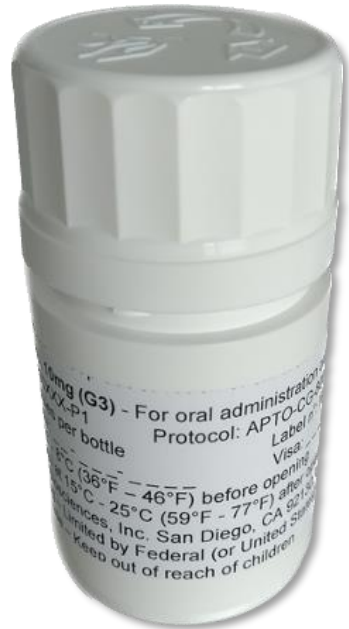
### CR in FLT3+ AML Patient

Achieved CR with 450mg BID dose level of G1  
Achieved MRD-negative status  
Demonstrates active against R/R AML

✓ CR in AML

G1 original formulation active but poorly absorbed and requires administration of high doses

# G3 improved formulation of luxepatinib



## Luxepatinib 3<sup>rd</sup> Generation (G3) Formulation

### Novel self emulsifying formulation

Designed for more rapid absorption (early T<sub>max</sub>), more efficient absorption (use lower doses), longer retention (longer t<sub>1/2</sub>), greater accumulation (higher steady state levels)

### Administered as a single dose to define its PK profile in cancer patients

### PK modeling predicts approx. an 18-fold improvement in bioavailability

Modeling predicts steady state with 50mg G3 Q12h is comparable to 900mg G1 Q12h

### Ongoing continuous dosing – 3x3 dose escalation study with AML patients

Treatment of R/R AML patients with 50mg G3 Q12h is ongoing

### Anticipate preliminary readout of PK properties with continuous dosing 1H 2023



# Aptose Biosciences (APTO) Key Financial Highlights Q4/2022

## Q4 Financial Highlights

Cash balance on Dec. 31, 2022, was \$47M

Cash burn during Q4 was \$8.4M

Cash runway into Q1 of 2024

The net loss during Q4 was \$10M

The net loss FY 2022 was \$41.8M

Net loss per share Q4 (\$0.11) and FY 2022 (\$0.45)

## Capitalization

Market capitalization is approximately \$60 million

Recent market cap. high was \$294M on 6/2021

Tuspetinib was acquired for \$12.5M

Common stock O/S March 23, 2023, was 93,005,278

No debt, warrants, or preferred equity

Commercial est. peak sales in excess of \$1B annually

### FIRM

Bios Research

Canaccord Genuity

Cantor Fitzgerald

H.C. Wainwright

JonesTrading

Oppenheimer & Co.

Piper Sandler & Co.

RBC Capital Markets

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Tuspetinib  
Oral Tablet



AML Patients



Investor Return



**Aptose is a precision oncology company developing oral targeted agents to treat hematologic malignancies**

# Aptose Investment Highlights

**Tuspetinib (Tus) myeloid kinase inhibitor: safe and effective, once daily, oral agent to treat AML**

- CRs across 4 dose levels with no DLT
- Favorable safety and non-myelosuppressive
- Broadly active across diverse AML populations
- Accelerated approval paths as monoRx and doublet
- Ideal for 1L triplet therapy and maintenance therapy
- Orphan Drug and Fast Track Status
- \$1B+ market potential

**Luxeptinib (Lux) lymphoid & myeloid kinase inhibitor**

- Clinically active in AML and B-cell cancers
- Exploring new formulation with improved absorption

**Value-driving near-term clinical milestones during 2023**

# Oppenheimer Expert Call: AML Current Treatments and Future Directions

Held January 27, 2023

## Quotes Regarding Tuspentinib from the KOL: *Dr. Harry Erba MD, PhD* Professor of Medicine, Hematologic Malignancies and Cellular Therapy, Duke Cancer Institute

*“Let's remember, phase 1 studies are for toxicity assessments and finding the recommended phase 2 dose. But of course, we want to see responses, and we see responses, and some of them are surprising! In patients who don't have mutated FLT3.”*

*“...I'll tell you what was most interesting to me when I look at the duration of responses and the way the study was done. **There are responses, clearly responses.** But here's the thing. **The drug did not have to be stopped in order to see a CR or a CRh.** Don't underestimate that. That's really important because it limits our combination partners because of myelosuppression with the FLT3 inhibitors we have.”*

*“**This drug may be better suited for the combinations that we hope to develop than anything we have right now.**”*

*“...when I look at the data for Tus, I'm more **excited about the lack of myelosuppression**”*

*“...at the doses that they can use to get a response, it's not leading to like an 85, 90% inhibition of FLT3 activity. And so I think that might be why they're getting away with less myelosuppression”*

*“**A drug like Tus will have a position mostly because it has a better toxicity profile than the drugs that we're using now in terms of myelosuppression**”*

**“If this drug continues to be safe and effective without the myelosuppression, it could be a game changer” Dr. Naval Daver<sup>1</sup>**

<sup>1</sup> Aptose ASH KOL Event Recording Dec. 11 2022



A microscopic view of a cell cluster, possibly a tumor, with a color gradient from red on the left to blue on the right. The cells are densely packed and have a textured, irregular appearance.

THANK YOU

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APT·OSE  
BIOSCIENCES

PRECISION ONCOLOGY FOR THERAPIES OF TOMORROW