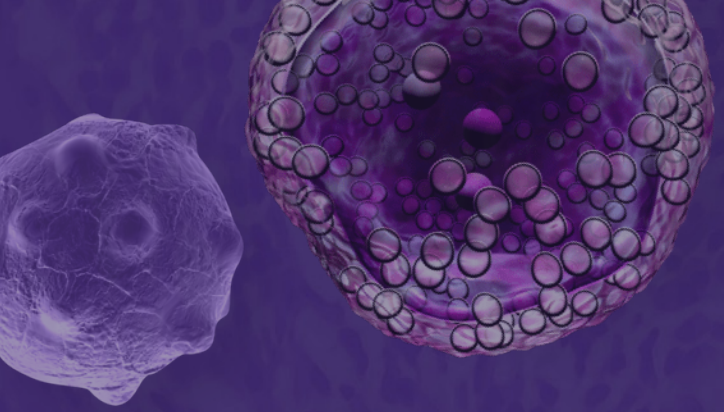

Aptose is a clinical stage oncology company developing oral kinase inhibitors for hematologic malignancies

Aptose KOL Event and Clinical Update

June 02, 2022

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PRECISION ONCOLOGY FOR THERAPIES OF TOMORROW

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

Disclosure

This presentation does not, and is not intended to, constitute or form part of, and should not be construed as, an offer or invitation for the sale or purchase of, or a solicitation of an offer to purchase, subscribe for or otherwise acquire, any securities, businesses and/or assets of any entity, nor shall it or any part of it be relied upon in connection with or act as any inducement to enter into any contract or commitment or investment decision whatsoever.

This presentation contains forward-looking statements, which reflect APTOSE Biosciences Inc.'s (the "Company") current expectations, estimates and projections regarding future events, including statements relating to our business strategy, our clinical development plans, our ability to obtain the substantial capital we require, our plans to secure strategic partnerships and to build our pipeline, our clinical trials and their projected timeline, the efficacy and toxicity of our product candidates, potential new intellectual property, our plans, objectives, expectations and intentions; and other statements including words such as "anticipate", "contemplate", "continue", "believe", "plan", "estimate", "expect", "intend", "will", "should", "may", and other similar expressions. Such statements constitute forward-looking statements within the meaning of securities laws.

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Aptose Biosciences Clinical Stage Agents

HM43239 Oral Myeloid Kinome Inhibitor / Clinically Validated for R/R AML Patients

Targets high value kinases operative broadly in AML patients : $FLT3^{WT/MUT}$, SYK, JAK1/2, $cKIT^{MUT}$

CRs in diverse R/R AML patients: $FLT3^{ITD/TKD/WT}$, $NPM1^{MUT}$, $TP53^{MUT}$, $N/K\text{-}RAS^{MUT}$, MLL, RUNX1, IDH^{MUT}

Orphan Drug Designation for AML and Fast Track Designation for R/R AML patients with $FLT3^{MUT}$

→ Now Transitioning to Expansion Trials planned 2H2022 : Doses and patient populations selected

LUXEPTINIB Dual Lymphoid and Myeloid Kinome Inhibitor

High value targets in B-cell cancers, AML, and inflammation : BTK, FLT3, LCK, LYN, Others

Ongoing parallel dose escalations in patients with B-cell lymphomas/CLL and AML/MDS

Clinically active: anti-tumor activity in high-bar clinical setting of R/R patients

→ Encouraging data with G3 formulation to reduce drug substance and increase plasma exposure

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KOL Event on Aptose's Investigational Treatments for AML
Featuring Elite Key Opinion Leaders

Brian Druker, M.D.

Brian Druker, M.D., has devoted his career to improving the lives of cancer patients. For his contributions to medical research, Dr. Druker was nominated for the Lasker-DeBakey Clinical Medical Research Award in 2009. Dr. Druker is most well-known for his role in developing Gleevec® for patients with chronic myeloid leukemia (CML). Dr. Druker's other career milestones include being named a Howard Hughes Medical Investigator in 2002, becoming a member of the National Academy of Sciences in 2007, winning the Japan Award in 2011, and being elected to the American Academy of Arts and Sciences in 2012. Dr. Druker received his Doctor of Medicine from the School of Medicine at the University of California, San Diego, completed his residency in internal medicine at Washington University in St. Louis, Missouri, and did an oncology fellowship at Dana-Farber Cancer Institute at Harvard Medical School.

From his earliest days, Dr. Druker was a dedicated researcher, winning the President's Undergraduate Research Award at the University of California, San Diego. He is the recipient of a Lifetime Achievement Award from the Leukemia & Lymphoma Society, the Medal of Honor from the American Cancer Society and many other awards.

Naval G. Daver, M.D.

Naval G. Daver, M.D. is an Associate Professor in the Department of Leukemia at the University of Texas MD Anderson Cancer Center. He completed his medical school from Grant Medical College and Sir J group of Hospitals Mumbai, followed by a residency and fellowship in hematology-oncology from Baylor College of Medicine. He is a clinical investigator with a focus on molecular and immune therapies in AML and Myelofibrosis and is principal investigator on >25 ongoing institutional, national and international clinical trials in these diseases. These trials focus on developing a personalized therapy approach by targeting specific mutations or immune pathways expressed by patients with AML, evaluating novel combinations of targeted, immune and cytotoxic agents, and identifying and overcoming mechanism of resistance.

Dr. Daver is especially interested in developing monoclonal and bispecific antibodies, immune checkpoint and vaccine based approaches in AML, MDS, and myelofibrosis and is leading a number of these trials at MD Anderson. Dr. Daver has published >150 peer-reviewed manuscripts and is on the editorial board of numerous hematology specific journals. He has also authored numerous abstracts at national and international conferences.

Brian Andrew Jonas, M.D., Ph.D.

Brian A. Jonas, MD, PhD, FACP is an Associate Professor and clinician scientist in the Division of Hematology and Oncology at UC Davis Comprehensive Cancer Center (UCDCCC), where he specializes in acute myeloid leukemia (AML), myelodysplastic syndrome (MDS), acute lymphoblastic leukemia (ALL), and other hematologic malignancies. He received his medical degree and PhD in biochemistry and molecular biology from UC Davis School of Medicine and completed his internship, residency, and a fellowship in hematology and oncology at Stanford University School of Medicine. Dr. Jonas leads the UCDCCC clinical and translational research program in AML, MDS, and ALL, with an emphasis on early drug development. He is PI on several clinical trials, including multiple investigator-initiated trials and ETCTN trials. He chairs the UCDCCC Hematological Malignancies Working Group and is Chair of the UCDCCC Data and Safety Monitoring Committee. He serves on the National Comprehensive Cancer Network panels for AML, MDS and ALL.

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Format for Aptose KOL Event

- **Clinical Update and Path Forward for HM43239 and Luxeptinib**
  - **Notable Highlights Today with Additional Clinical Data Presented Separately**
  - **Dr. Rafael Bejar and Dr. William Rice**
- **Impact of Kinase Inhibitors on Cancer Treatments and Future Needs of AML Patients**
  - **Dr. Brian Druker**
- **Dr. Druker will Lead Discussions with AML KOLs | Investigators with HM43239**
  - **Dr. Naval Daver**
  - **Dr. Brian Jonas**

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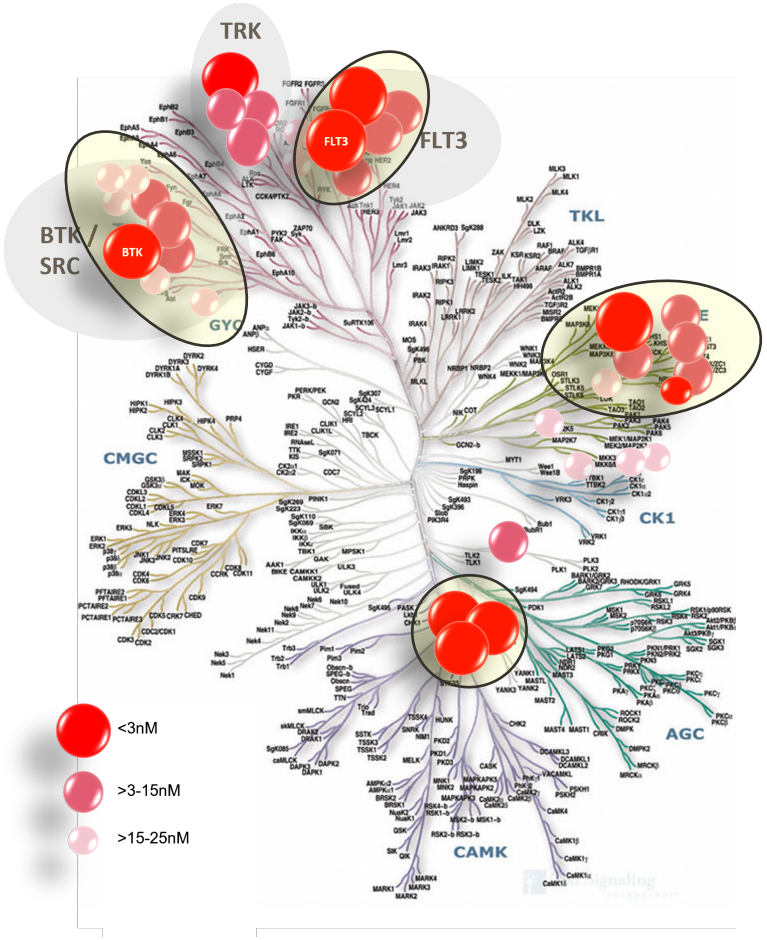
Luxeptinib “Lux”

Oral Lymphoid & Myeloid Kinome Inhibitor

Luxeptinib: Atypical, Dual Lymphoid and Myeloid Kinome Inhibitor



Unique Kinome Targeting

Inhibits constellation of high value targets: BTK, FLT3, Others

Only agent to potently inhibit the validated **BTK** and **FLT3**

In development for the treatment of both lymphoid & myeloid hematologic cancers

Mutation Agnostic

Inhibits **WT and all mutant forms** of BTK

Inhibits **WT and all mutant forms** of FLT3

May avoid rapid emergence of drug resistance

Robust Safety Profile

Simultaneously suppresses multiple oncogenic signaling pathways

Avoids kinases that negatively impact safety

Generally, well tolerated in clinical studies to date

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Luxeptinib Ongoing Activities

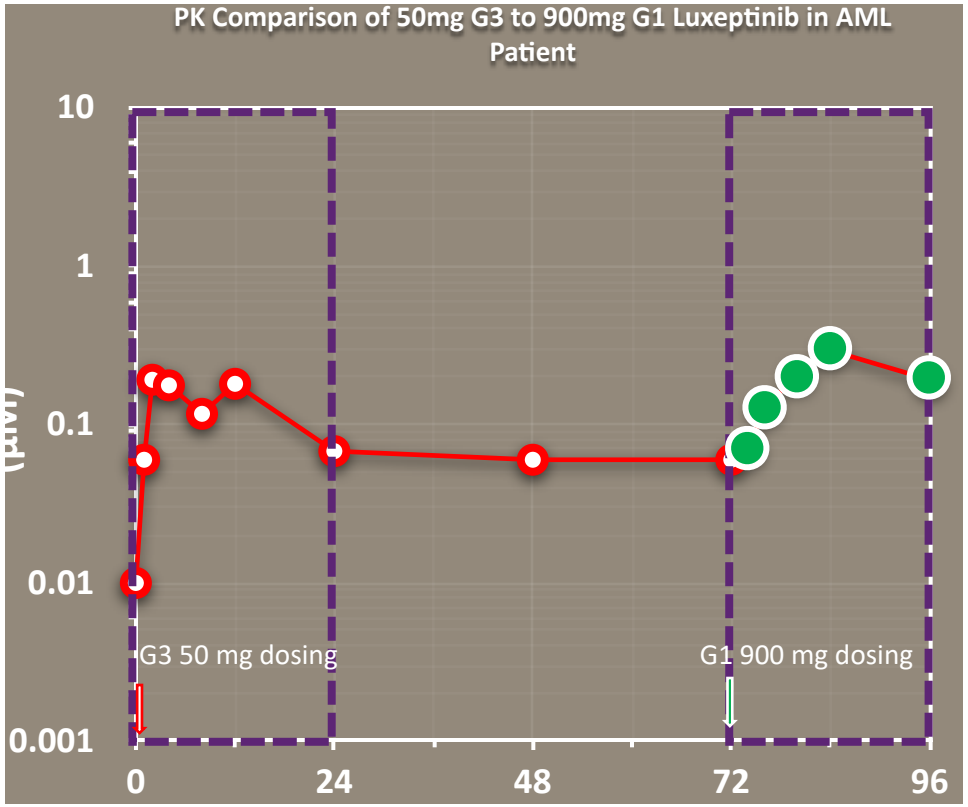
- **Ongoing Clinical Development in Two Separate Trials and Patient Populations**
  - Phase 1 trial in patients with R/R B-cell leukemias|lymphomas • Phase 1 trial in patients with R/R AML
- **Critical Step for Lux Program is the Transition to a Novel and Improved Formulation (G3)**
  - Goals to achieve **greater plasma exposures** with administration of **less drug substance and fewer number of pills**
- **Lux G3 Formulation is Being Tested Relative to the Original Formulation in Both Phase 1 trials**
- **Patients Enrolling Well to Studies, Allowing us to Test a Single Dose of Lux G3 at Multiple Dose Levels**
  - Already dosed G3 formulation at 50 mg, 100 mg and 200 mg
- **Following a Single Dose of Lux G3, Patients Continue on Study Using the Original Formulation**
  - We plan to provide the antitumor data in a corporate slide deck later and then more rigorously during ASH
- **Today We Will Present PK Data with the Lux G3 Formulation**
  - Four patients dosed with 50 mg and 3 patients dosed with 100 mg
  - One patient dosed with 200 mg and others in the queue, but data not yet available

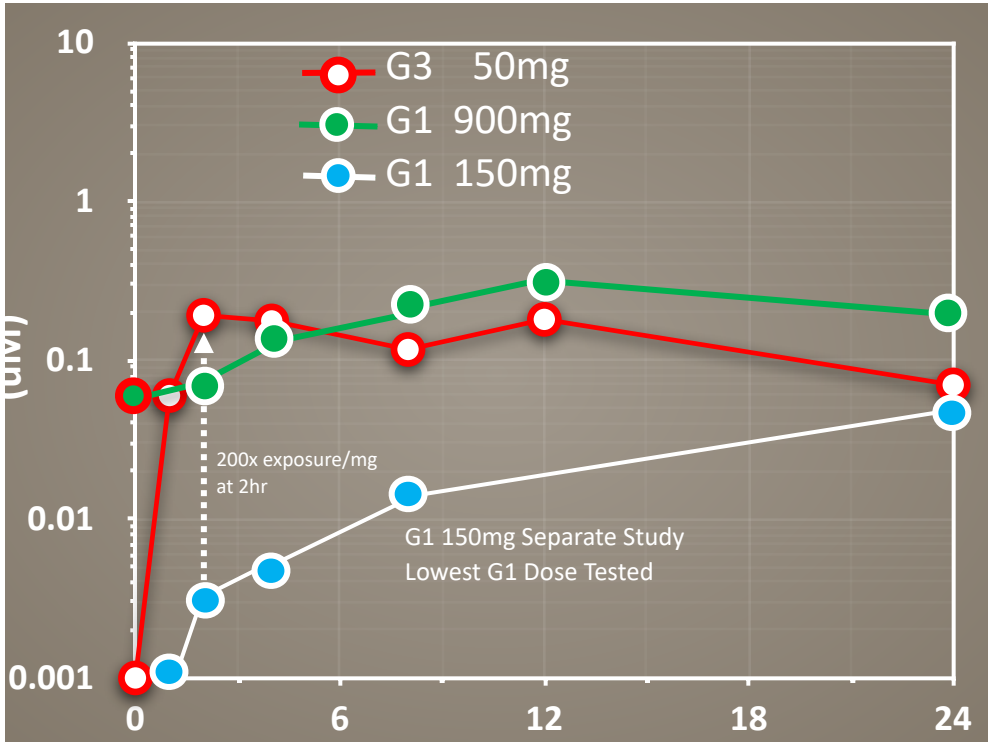
Luxeptinib: G3 Formulation with 50 mg and 100mg Dosages

- **Patients participating in G3 study are derived from:**
  - **Ph 1 R/R B-cell cancers**
  - **Ph 1 R/R AML**
- **72 hr following single dose of G3:**
  - **Some received 750mg G1**
  - **Some received 900mg G1**

G3 Dose	G1 Dose	No. Patients
50 mg	750 mg	1
50 mg	900 mg	3
100 mg	750 mg	2
100 mg	900 mg	1
200 mg	900 mg	1

Luxeptinib: First Patient Dosed with G3 Formulation
PK Comparison of **G3 50mg Single Dose** to G1 900mg Single Dose (18X)





G3 50mg Single Dose PK (72hr), followed by G1 900mg Single Dose PK in Same Patient

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Luxeptinib: Oral Lymphoid and Myeloid Kinome Inhibitor

Targets Kinases Important in Lymphoid *and* Myeloid Cancers

- Inhibits BTK, FLT3, CSF1R, PDGFRα, TRK, AURK, others
- Generally well-tolerated – currently dosing at 900mg BID with original formulation
- Delivered antitumor activity in diverse B-cell cancers
- Delivered **MRD- CR in relapsed AML patient** with high exposure

Findings to Date Identify Needs for Future Development

- Clinical activity and tolerability justify further dose exploration
- Doses of 450-750mg with original formulation provided incremental exposure increases
- Identified **need for consistent and higher exposure levels** in AML & B-cell cancer patients

Next Steps for Luxeptinib in 2022

- Continue exploring improved G3 formulation to increasing exposure and to lower pill burden and drug substance manufacture
- **G3 early data are encouraging**
- Higher dosages may be evaluated
- PK modeling of continuous dosing
- Plan continuous dosing with G3 if the data from single dose and modeling are supportive

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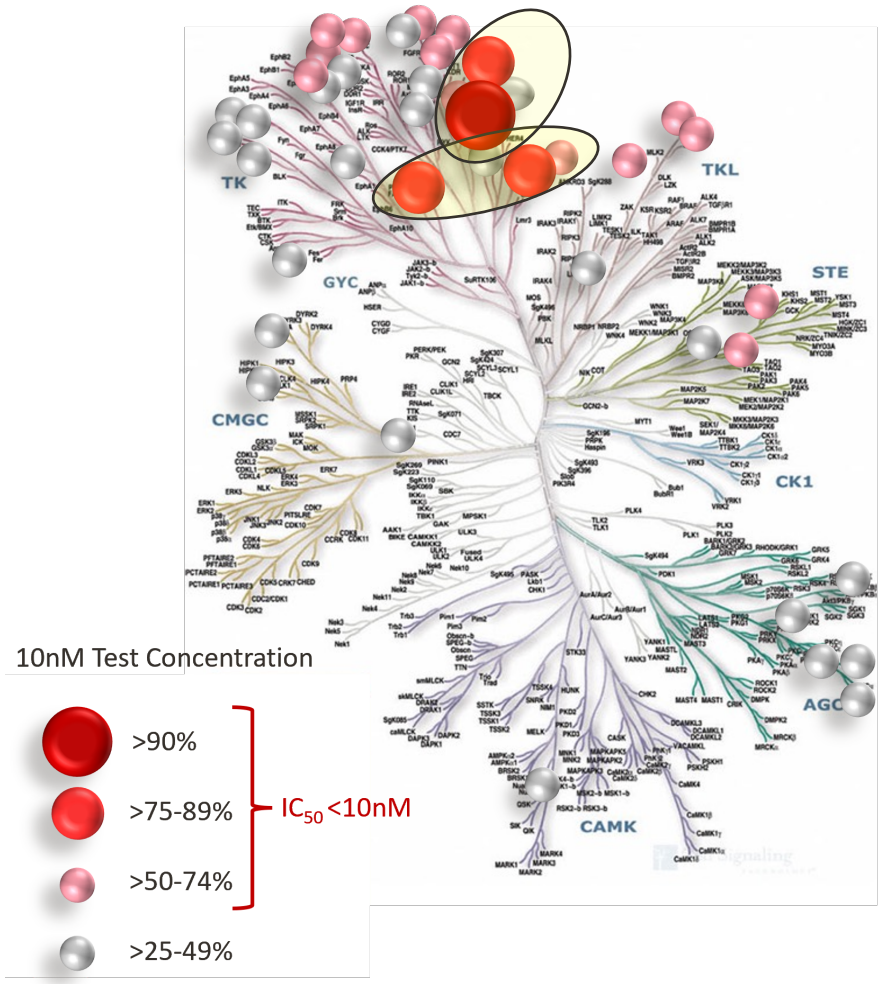
HM43239 “239”

Clinically Validated, Oral Myeloid Kinome Inhibitor Advancing to Next Stage of Clinical Development for the Treatment of Patients with R/R AML

HM43239 Kinase Inhibitory Profile: Predicts Clinical Activity in AML Patients Harboring Mutated FLT3, Unmutated FLT3, and Having a Diverse Collection of Adverse Mutations



Assay Methodology	Kinase	Mutation Type	Activity
Binding Affinity (K_D, nM)	FLT3	WT	0.58
		ITD	0.37
		D835Y	0.29
		D835H	0.4
		ITD/D835V	0.48
		ITD/F691L	1.3
Inhibition of Kinase Enzyme Activity (IC_{50}, nM)	FLT3	WT	1.1
		ITD	1.8
		D835Y	1.0
	SYK	WT	2.9
	JAK	JAK-1	2.8
		JAK-2	6.3
		JAK-2 (V617F)	9.9
	c-KIT	WT	> 500
		D816H	3.6
		D816V	3.5

Potent suppression of driver and compensatory kinases operative in AML

- **All forms of FLT3**: -ITD, -TKD, -GK mutations and FLT3-WT
- **SYK** signal transduction kinase
- **JAK 1/2** signal transduction kinases
- **cKIT^{MUT}** alternative receptor kinases

→ **Serves as a multi drug therapy in a single molecule**

→ **Simultaneously disrupting multiple dysregulated signal transduction pathways that drive AML proliferation and resistance mechanisms.**

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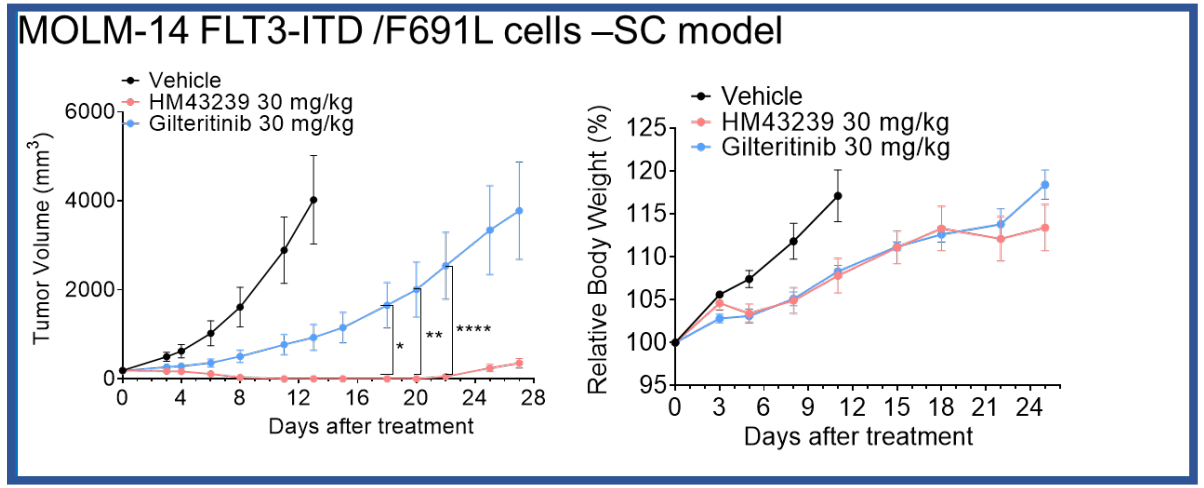
HM43239 Superior to Gilteritinib in AML Models Conducted in Mice: AML with FLT3-ITD/F691L Mutations Resistant to Gilteritinib FLT3 Inhibitor

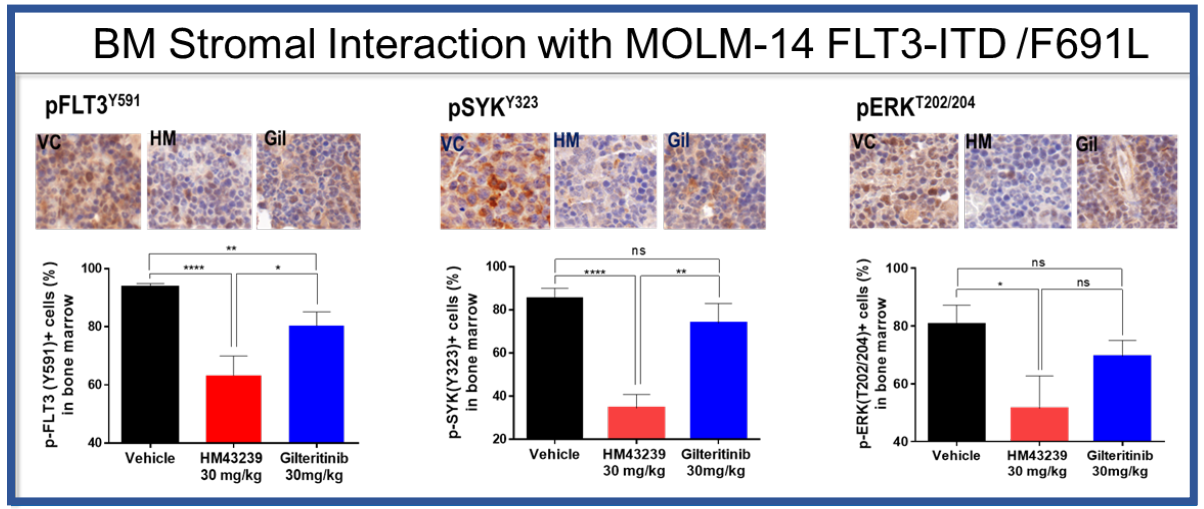
HM43239 more potent than gilteritinib : Subcutaneous AML model resistant to gilteritinib

Three million MOLM-14 FLT3-ITD/F691L cells were implanted SC in nude mice. Fifteen days later they were randomized by their tumor volume into 3 groups of 5 mice each. The mice were then treated orally QD with either placebo, 30mg/kg HM43239 or 30 mg/kg gilteritinib for 28 days. Statistical analysis utilized two-way ANOVA followed by Sidak's test.



HM43239 more potent than gilteritinib : Orthotopic/Systemic AML model resistant to gilteritinib

Mice were administered i.v. MOLM-14 FLT3-ITD/F691L cells and allowed to populate the bone marrow for 7 days, after which drugs were administered orally QD 14 days. Representative images of IHC were collected using a Dako REAL Envision Detection System (400x) and quantified with a Vectra 3 Pathology Imaging Analyzer (200x images). Positive DAB % = DAB positive area pixel / (Hematoxylin pixel + DAB positive area pixel) × 100. ▪ VC, vehicle control; HM, HM43239 30 mg/kg; Gil, Gilteritinib 30mg/kg. ▪ ns, not significant; * p<0.05, **p<0.01, ***p<0.001, ****p<0.0001 (unpaired t test using GraphPad PRISM®, GraphPad Software)



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HM43239 Positioned for Broad Clinical Activity and Success in AML Patients

- **Positioned as Superior to Other FLT3 Inhibitors**
  - Inhibits all forms of FLT3
  - Kills AML cells and treats AML disease in animals resistant to other approved FLT3 inhibitors
- **Positioned as More than FLT3 Inhibitor for AML**
  - SYK inhibitor, JAK inhibitor, and c-KIT inhibitor
  - "Combination therapy in one molecule" that suppresses multiple key targets simultaneously
- **Positioned to Achieve Broad Therapeutic Window**
  - Well tolerated with oral activity in animal models
  - Antitumor activity in animal models across multiple safe dose levels
- **Favorable Pharmaceutical and CMC Properties**
  - Stable as drug substance and drug product
  - Orally administered and absorbed efficiently
  - Current tablet presentation appears acceptable for commercialization
- **Clinically Validated with R/R AML Patients**
  - CRs among R/R AML patients harboring FLT3 mutations ± prior Tx with FLT3 inhibitors
  - CRs among patients with Unmutated FLT3 but harboring a diversity of adverse mutations
  - Now exploring molecular subgroups (genotypes) of R/R AML patients with adverse mutations to understand full scope of activity

HM43239 Phase 1/2 Study in R/R AML: Ongoing Dose Escalation & Dose Exploration

PART A : DOSE ESCALATION			PART B : DOSE EXPLORATION	
Cohort 6	200 mg QD	Ongoing		
Cohort 5	160 mg QD	Completed	160 mg QD	9 Treated → 20 Planned
Cohort 4	120 mg QD	Completed	120 mg QD	12 Treated → 20 Planned
Cohort 3	80 mg QD	Completed	80 mg QD	20 Treated
Cohort 2	40 mg QD	Completed		
Cohort 1	20 mg QD	Completed		

Swimmers' Plots of Patient Study Status
All patients in Part A (dose escalation) and Day 15 in Part B (dose exploration)

Mean Plasma PK Concentrations (+SD) by Dose Cohort (Semi-log Scale)
Single Dose

Mean Plasma PK Concentrations (+SD) by Dose Cohort (Semi-log Scale)
Multiple Doses / Steady State

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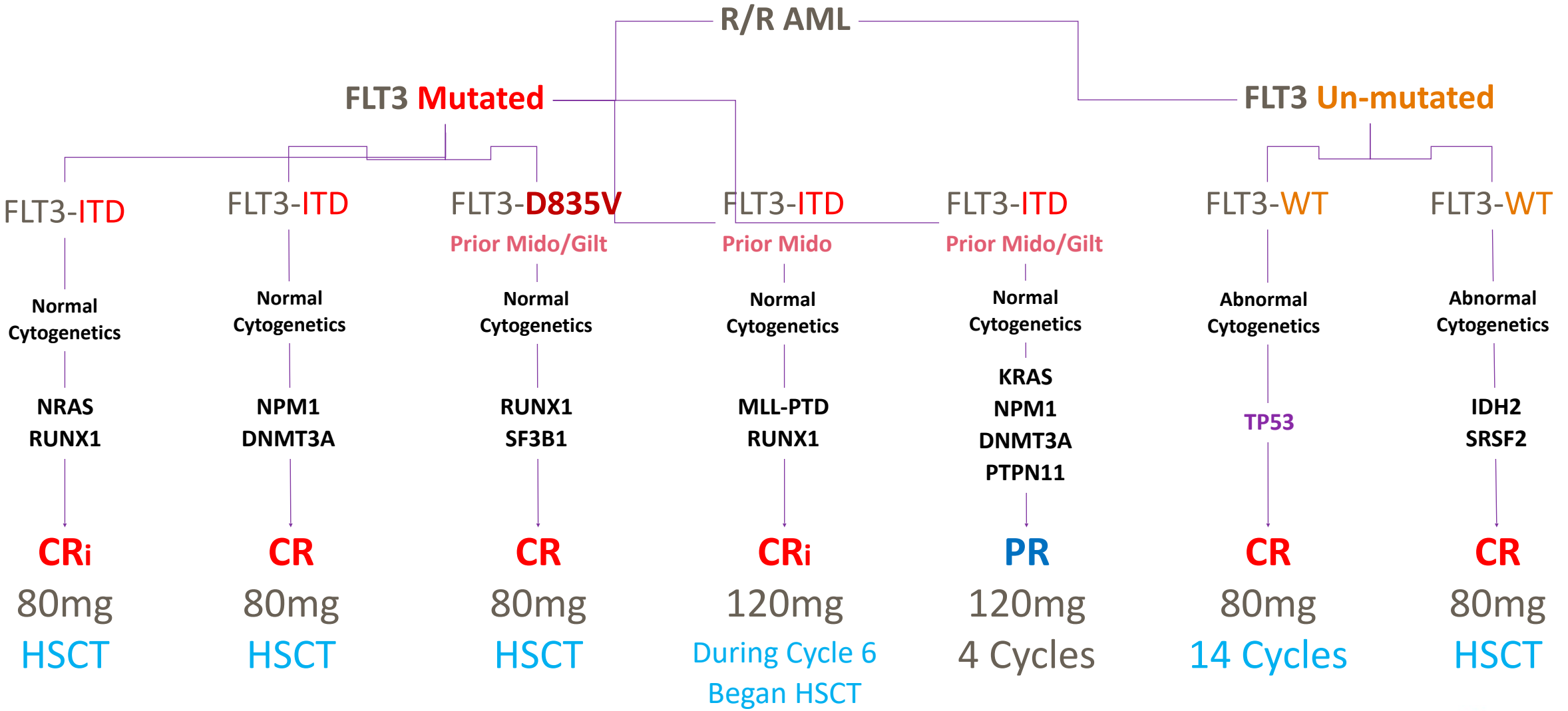
HM43239: Characteristics of Responding AML Patients with Best Clinical Response



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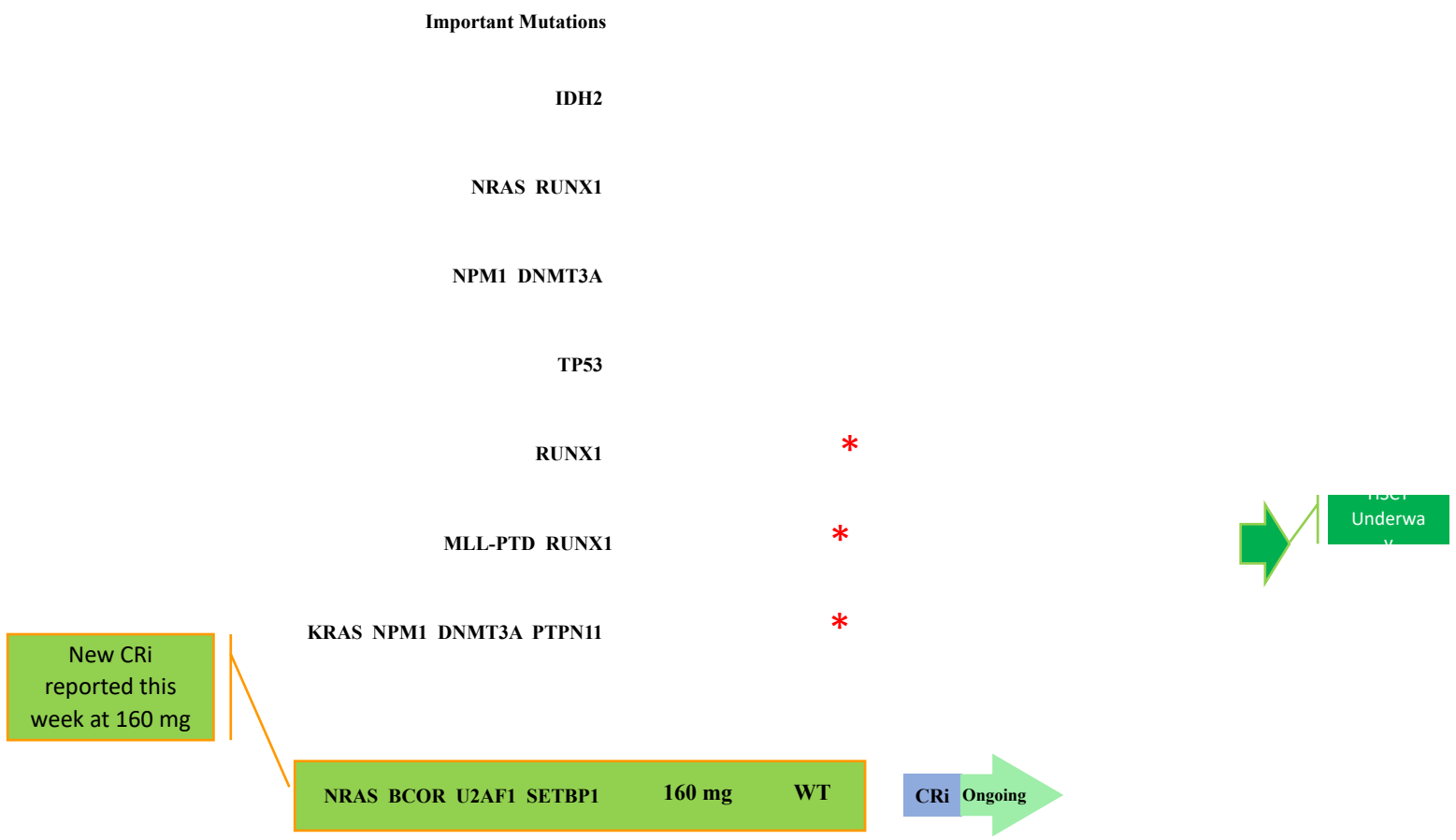
HM43239: Characteristics for Patients Who Achieved a Clinical Response in Phase 1/2 Study in R/R AML Patients

Data filtered through: 26APR2022



Abbreviation: CR, complete response; CRi, complete response with incomplete hematologic recovery; CRp, complete response with incomplete platelet recovery; HSCT, hematopoietic stem cell transplantation; NE, not evaluable; PD, progressive disease; PR, partial remission.
Note: 'Ongoing' means treatment is still ongoing; 'Remain Alive' indicates patients' status in follow-up after treatment termination; 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: Each response assessed at a regular visit is considered to have started 1 cycle before the assessment; however, 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.

▲ Indicates Death

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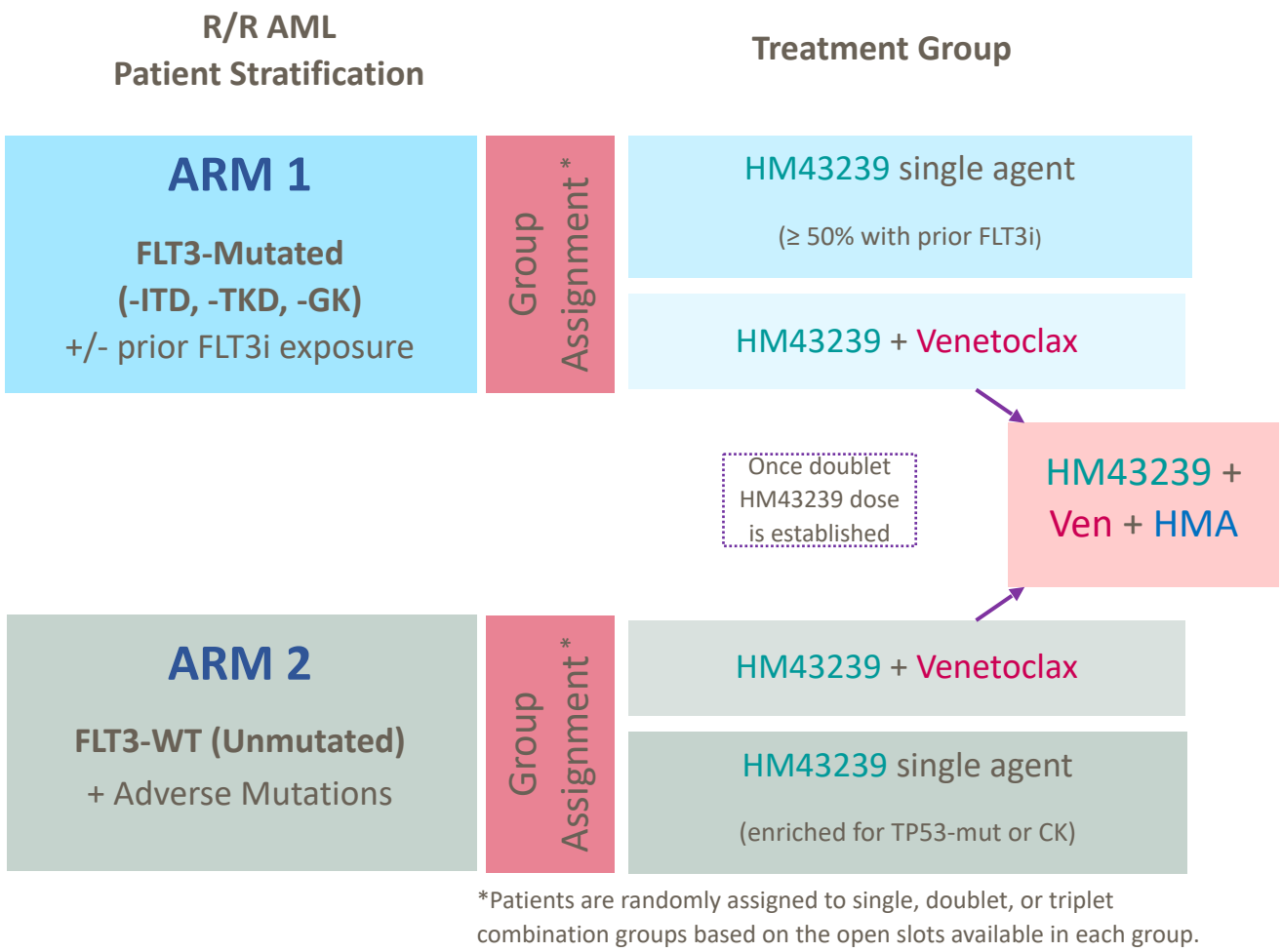
HM43239: Safety and Efficacy Data Revealed a Broad Therapeutic Range

- **Favorable Safety Profile to Date**
  - No drug related SAE
  - No drug related deaths
  - No drug related AE of elevated CK
  - No drug related AE of QT prolongation – No observed relation between ΔQTc and dose
  - No DLT up to 160 mg and one DLT of muscle weakness (not rhabdomyolysis) at 200 mg
- **Identified a Therapeutic Range and Broad Therapeutic Window**
  - Safely achieved **efficacy at 3 separate dose levels** (80 mg, 120 mg, 160 mg) with no DLT
  - Demonstrated **broad therapeutic range** across safe dose levels
- **Study Continuing Across Several Cohorts**
  - **Dose exploration cohort of 120 mg** currently enrolling and planned for a total of 20 patients
  - **Dose exploration cohort of 160 mg** currently enrolling and planned for a total of 20 patients
  - **Dose escalation at 200 mg** dose level planned to continue

HM43239: Teachings from Phase 1 Guides Expansion Clinical Studies

- **Current Dose Escalation/Dose Exploration Phase 1 Trial in R/R AML Patients**
  - Demonstrated **CRs in FLT3-Mutant AML** and received **Fast Track Designation in FLT3-Mutant R/R AML**
  - Selected **3 Expansion Doses** (80 mg, 120 mg, 160 mg) and **Patient Populations** for **Expansion Trials**
  - Continue **Exploration of Molecular Subgroups (Genotypes)** for potential **Fast Track** and **News Flow**
- **Transitioning to Expansion Trials in AML Patients as Prelude to Registrational Trials**
  - Plan **120 mg as primary Single Agent Expansion Dose** with 80 mg and 160mg as bracketing doses
  - Plan **FLT3 Mutated** R/R AML (supported by Fast Track Designation)
  - Plan **FLT3-Unmutated** R/R AML (with Adverse Mutations)
  - Plan **Single Agent** in **FLT3-Mutated** and **FLT3-Unmutated** to **begin 2H2022**
  - Plan **Combination (239+Ven)** in **FLT3-Mutated** and **FLT3-Unmutated** to **begin 1H2023**

HM43239: Next Step Planned as Phase I Expansion to Provide Data Intended to Support Registrational Studies



*Patients are randomly assigned to single, doublet, or triplet combination groups based on the open slots available in each group.

Key Objectives

Primary:

- Safety and tolerability
- Confirm HM43239 single agent RP2D
- Select HM43239 candidate RP2D in combination with venetoclax
- Select HM43239 candidate R2PD dose in combination with venetoclax and HMA

Secondary:

- CR_c rate (CR + CR_h + CR_p + CR_i)
- Best response rate (CR_c + PR)
- Duration of response (DOR)
- Overall survival (OS)

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Major Objectives and AML Target Populations Sought for HM43239

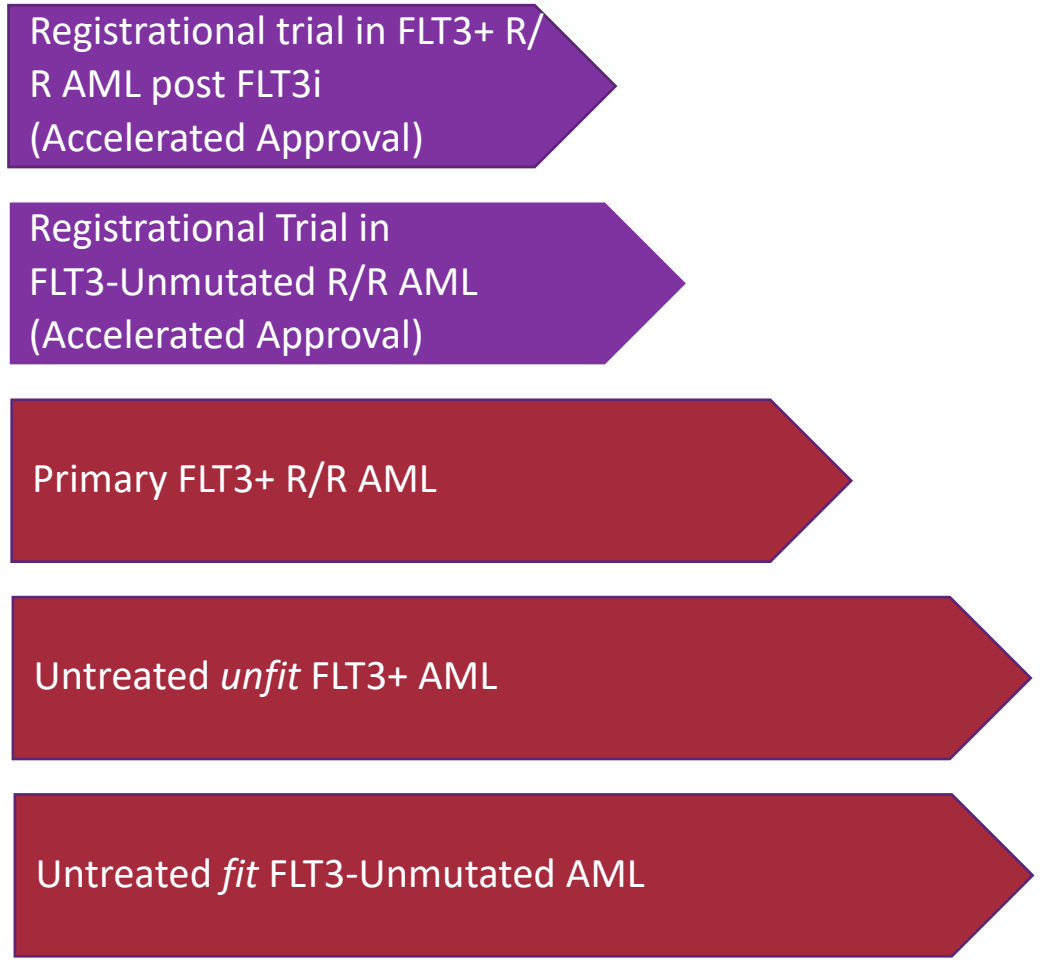
1. R/R AML FLT3+ post FLT3 inhibitor :
   Limited treatment options for patients

2. R/R AML FLT3-Unmutated with adverse mutations:
   Most patients have limited treatment options, especially if they have received BCL2i and HMAs as frontline treatment

3. R/R AML FLT3+ through combination:
   Build upon standard of care for patients in combination with with BCL2i and/or HMAs

4. 1L *unfit* AML FLT3+ patients:
   Provide superior treatment option for untreated not eligible for high dose chemotherapy

5. 1L *fit* AML FLT3-Unmutated:
   Provide superior non high dose chemotherapy option



Phase 1/2 Phase 3

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HM43239: Clinically Validated, Once Daily, Oral Myeloid Kinome Inhibitor

Targets Constellation of Kinases Important in Myeloid Cancers

- Potent inhibitor of kinases associated with malignant transformation and resistance
- Highly active *in vivo* against FLT3 internal tandem duplication (ITD), resistance-conferring tyrosine kinase domain mutations (TKD) , and gatekeeper mutations (F691)
- Highly active on SYK, JAK1/2 and mutant forms of c-KIT

Clinical Validation Supports Path of Rapid Development for AML Patients

- CR breadth in patients with FLT3 mutations, including ITD and D835 TKD, even if failed prior FLT3 inhibitors (midostaurin and gilteritinib)
- CRs in patients with Unmutated FLT3
- CRs in patients with diverse mutations in NPM1, DNMT3A, RUNX1, IDH1/2, N/KRAS, MLL, TP53, others
- Selected three doses (80, 120, 160 mg) in the therapeutic window
- FDA Fast Track in $FLT3^{MUT}$ R/R AML

Program Goals for 2022 Supporting Rapid Development

- Exploring additional adverse genotypes for sensitivity
- Provide rolling presentation of clinical findings throughout 2022
- Plan to initiate Expansion Trial as single agent 2H2022
- Plan to initiate Expansion Trial as combination 1H2023
- Planning for registrational studies

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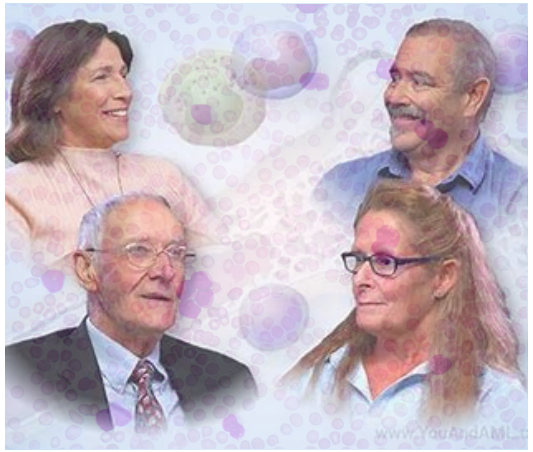
Dr. Brian Druker

Impact of Kinase Inhibitors on Cancer Treatments and Future Needs of AML Patients

AML in the US : Estimated 20,240 new cases and 11,400 deaths in 2021

Continued Unmet Need for More Effective and Safe Therapies

Epidemiology	US (2021)	EU5 (2020)	Japan (2021)	China (2020)
Leukemia Incidence[3]	61,090[1]	51,820[3]	14,600[7]	85,400
AML Incidence	20,240[2]	16,580[3a]	6,570[7c]	31,430[3b]
5-Year Prevalence (Leukemia) (2020)[3]	187,560	152,230	41,280	241,750
Mortality (Leukemia)	11,400 (AML)[2]	31,690	8,700[7]	61,690



- **Most common acute leukemia in adults**
- **5-year survival rate of approx. 30%**
- **Relapsed AML patients have a median life expectancy of < 6 months* with approved therapies**
- **Need new targeted agents to better treat R/R AML patients and to treat resistance to current agents**
- **Need more effective & better tolerated agents to achieve lasting remissions and extend meaningful life**

Sources: 1. SEER_2021 Leukemia; 2. SEER 2021 AML; 3. The Global Cancer Observatory (GLOBOCAN) - IACR (2020) - Projections; 4. Chihara et al. Br J Haematol. 2014; 5. Chen et al. J Hematol Oncol. 2010: 21; 6. Cancer.net; 7. Ganjoho Cancer Statistics in Japan 2021
*https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7486485/ *https://www.frontiersin.org/articles/10.3389/fonc.2021.649209/full

[a]EU5 incidence calculated by applying 32% (AML) on leukemia to obtain incidence of AML[6]
[b]In China, AML accounted for ~36.8% of all leukemias[5] [c]In Japan, AML accounted for ~45% of all leukemias in 2008[4]

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AML is a still a disease with high unmet medical needs : overcome drug resistance, prevent relapse and more tolerable treatment options

Effective Therapies in Relapse / Refractory disease

Combination therapies to overcome resistance and for durable responses

Overcome tolerability and AE concerns with current SoC

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Discussion with KOLs

Dr. Druker to lead discussion:

Dr. Daver and Dr. Jonas as study investigators who have treated patients with HM43239

KOL Event on Aptose's Investigational Treatments for AML Featuring Elite Key Opinion Leaders

Brian Druker, M.D.

Brian Druker, M.D., has devoted his career to improving the lives of cancer patients. For his contributions to medical research, Dr. Druker was nominated for the Lasker-DeBakey Clinical Medical Research Award in 2009. Dr. Druker is most well-known for his role in developing Gleevec® for patients with chronic myeloid leukemia (CML). Dr. Druker's other career milestones include being named a Howard Hughes Medical Investigator in 2002, becoming a member of the National Academy of Sciences in 2007, winning the Japan Award in 2011, and being elected to the American Academy of Arts and Sciences in 2012. Dr. Druker received his Doctor of Medicine from the School of Medicine at the University of California, San Diego, completed his residency in internal medicine at Washington University in St. Louis, Missouri, and did an oncology fellowship at Dana-Farber Cancer Institute at Harvard Medical School.

From his earliest days, Dr. Druker was a dedicated researcher, winning the President's Undergraduate Research Award at the University of California, San Diego. He is the recipient of a Lifetime Achievement Award from the Leukemia & Lymphoma Society, the Medal of Honor from the American Cancer Society and many other awards.

Naval G. Daver, M.D.

Naval G. Daver, M.D. is an Associate Professor in the Department of Leukemia at MD Anderson. He completed his medical school from Grant Medical College and Sir J group of Hospitals Mumbai, followed by a residency and fellowship in hematology-oncology from Baylor College of Medicine. He is a clinical investigator with a focus on molecular and immune therapies in AML and Myelofibrosis and is principal investigator on >25 ongoing institutional, national and international clinical trials in these diseases. These trials focus on developing a personalized therapy approach by targeting specific mutations or immune pathways expressed by patients with AML, evaluating novel combinations of targeted, immune and cytotoxic agents, and identifying and overcoming mechanism of resistance.

Dr. Daver is especially interested in developing monoclonal and bispecific antibodies, immune checkpoint and vaccine based approaches in AML, MDS, and myelofibrosis and is leading a number of these trials at MD Anderson. Dr. Daver has published >150 peer-reviewed manuscripts and is on the editorial board of numerous hematology specific journals. He has also authored numerous abstracts at national and international conferences.

Brian Andrew Jonas, M.D., Ph.D.

Brian A. Jonas, MD, PhD, FACP is an Associate Professor and clinician scientist in the Division of Hematology and Oncology at UC Davis Comprehensive Cancer Center (UCDCCC), where he specializes in acute myeloid leukemia (AML), myelodysplastic syndrome (MDS), acute lymphoblastic leukemia (ALL), and other hematologic malignancies. He received his medical degree and PhD in biochemistry and molecular biology from UC Davis School of Medicine and completed his internship, residency, and a fellowship in hematology and oncology at Stanford University School of Medicine. Dr. Jonas leads the UCDCCC clinical and translational research program in AML, MDS, and ALL, with an emphasis on early drug development. He is PI on several clinical trials, including multiple investigator-initiated trials and ETCTN trials. He chairs the UCDCCC Hematological Malignancies Working Group and is Chair of the UCDCCC Data and Safety Monitoring Committee. He serves on the National Comprehensive Cancer Network panels for AML, MDS and ALL.

APTOSE BIOSCIENCES

Q&A with KOLs

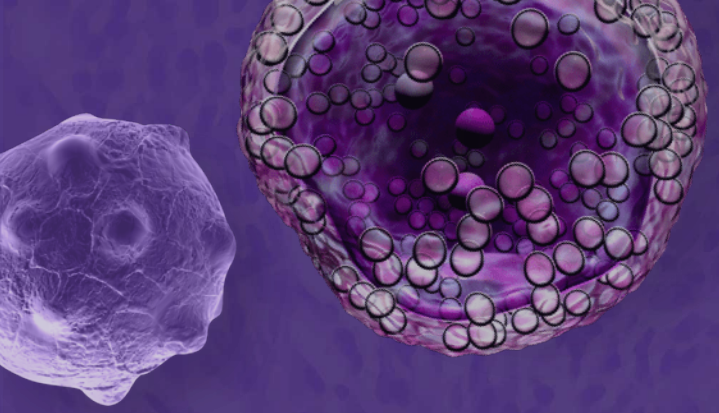
Naval G. Daver, M.D.

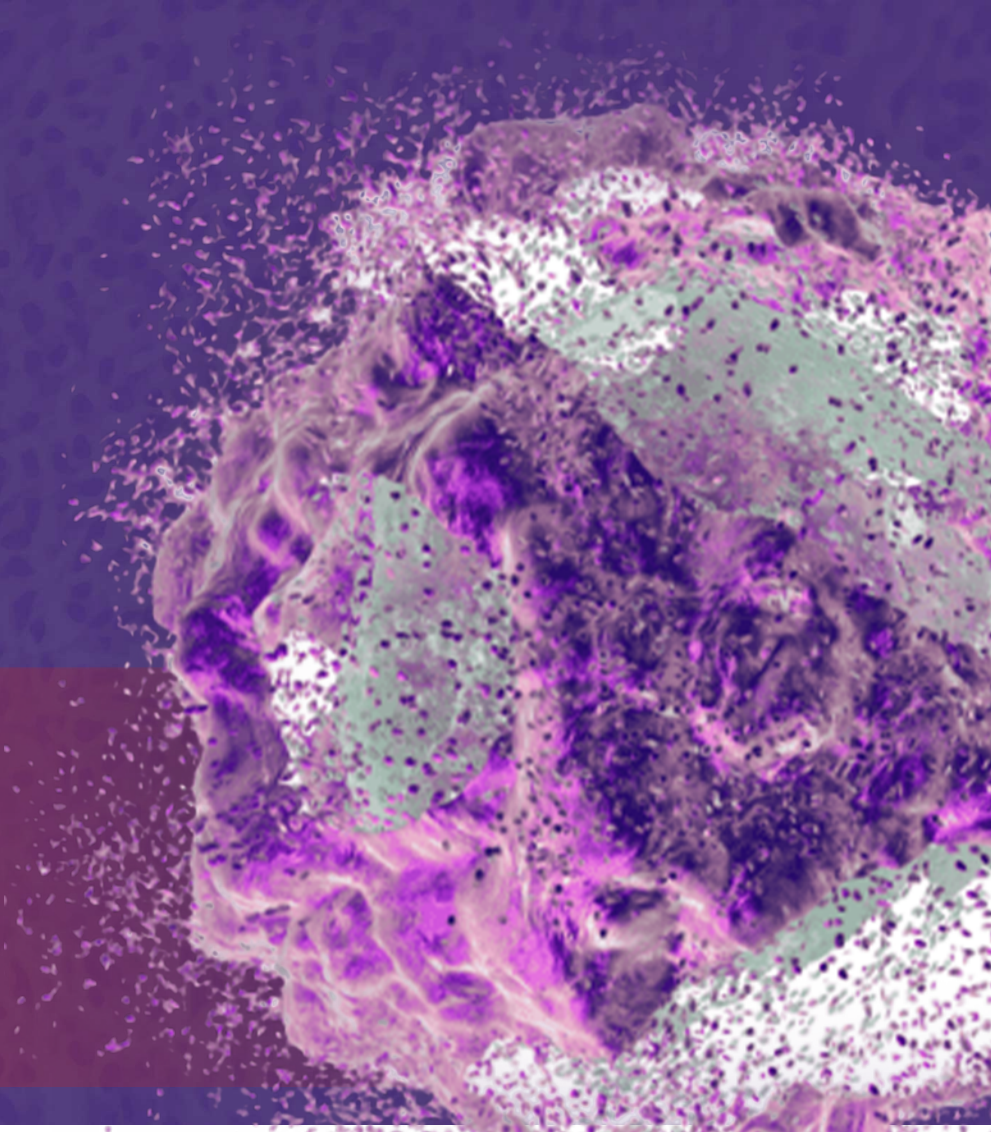
Brian Druker, M.D.

Brian Andrew Jonas, M.D., Ph.D.

Recap of Future Plans

- **HM43239**
  - **HM43239 is Clinically Validated in on FLT3-Mutated and FLT3-Unmutated R/R AML Patients**
  - **Plan to Continue Dose Escalation/Dose Exploration Phase 1**
    - Explore Adverse Genotypes at 120 mg/160 mg to Deliver Additional Fast Track and News Flow
  - **Plan to Initiate Expansion Trials to Position for Registrational Trials**
    - Plan 2H2022 as Single Agent Trials in FLT3-Mut and FLT3-Unmut (Adverse Mutations) Populations
    - Plan 1H2023 as Combination (239+Ven) in FLT-Mut and FLT3-Unmut (Adverse Mutations) Populations
    - Plan 120 mg as Primary Expansion Dose with 80 mg and 160mg as bracketing doses
  - **Planning Subsequent Phase 2 Registrational Studies**
    - Data Permitting, Request Allowance to Move into Genotypes of High Need for Accelerated Approvals
  - **Planning Confirmatory Phase 3** : Perform appropriate trials to support expansion of label
  - **Planning Ultimately for Commercialization :** Include FLT3-Mut and FLT3-Unmut, Unfit and Fit, as well as R/R, 2L, 1L AML
- **Luxeptinib**
  - **Planning to Continue G3 Dosage-PK Exploration and then Transition to Continuous Dosing with G3**





We thank our KOLs, investigators, and investors for helping us bring novel drugs to patients with the greatest need

APTOSE BIOSCIENCES

PRECISION ONCOLOGY FOR THERAPIES OF TOMORROW