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#### Aptose Biosciences Investor Highlights (NASDAQ: APTO)

- Publicly-traded, clinical-stage biotech company developing oral kinase inhibitors to treat cancer patients with life-threatening hematologic malignancies
- **HM43239** lead agent and primary value driver
  - Clinically de-risked (>50 patients in US and S. Korea) once daily, oral, myeloid kinase inhibitor
  - Safely achieved single agent Complete Remissions (CRs) in diverse R/R AML patient populations
    - Strong enough to deliver single agent CRs yet gentle enough for safety in R/R AML patients
    - Intense support from clinical investigators in single agent and drug combination trials
    - Efficacy & safety profile position as a preferred agent for combination therapy and broad commercial use
  - Multiple genetically-defined AML target populations as potential indications for >\$1bn commercial market
    - Targets more genetically-defined AML target populations than SYK inhibitors, IRAK4 inhibitors, or Menin inhibitors
    - Response rates in AML populations of unmet needs that may support single agent Phase 2 accelerated approvals
  - Strong intellectual property estate
- Experienced leadership team with deep expertise in kinase inhibitors & orphan hematologic diseases
- Meaningful near-term upside with value-driving clinical updates and milestones through 2022 and 2023





#### Aptose Leadership Team: Multifaceted Expertise in Therapeutic Development



William G. Rice, PhD

Chairman, President & Chief Executive Officer



#### Rafael Bejar, MD, PhD

Sr. VP & Chief Medical Officer



#### **Fletcher Payne**

Sr. VP & Chief Financial Officer



#### Philippe Ledru

Sr. VP & Chief Commercial Officer





































#### Brian J. Druker, MD

Chair, Scientific Advisory Board



Michael Andreeff, MD, PhD

Scientific Advisory Board



#### Daniel Von Hoff, MD, FACP

Scientific Advisory Board

- Pioneer in the field of precision medicine, Key Role in the development of Gleevec the first targeted kinase inhibitor for cancer
- · Member, National Academy of Medicine, National Academy of Sciences & American Academy of Arts & Sciences
- Winner of Karnofsky Award, Lasker Award, Japan Prize in Healthcare and Medical Technology, Tang Prize in Biopharmaceutical Science, Sjöberg Prize
- · Leader of Inter-institutional Beat AML Initiative
- Renowned hematology specialist, Expert in AML and other hematologic malignancies
- Expert in drug resistance and drug mechanisms
- Professor of Medicine, Paul and Mary Haas Chair in Genetics
- Chief, Section of Molecular Hematology and Therapy, MD Anderson Cancer Center
- Former President of AACR, Board Member of ASCO, Former Presidential Cancer Advisory Board
- Physician in Chief, Tgen, Medical Director of Research for McKesson Specialty Health
- Chief Scientific Officer for US Oncology Research, Professor of Medicine, Mayo Clinic Scottsdale



#### Aptose Biosciences: Clinical Stage Pipeline of Differentiated Myeloid Kinase Inhibitors

#### HM43239 oral myeloid kinase inhibitor clinically validated for R/R AML patients

Clinically Sate & Effective	25-44% ORR in Phase 1/2 Trial with CRs in multiple genetically-defined AML target populations
Near-term Value Creation	Expansion Trials begin 2022 as passage into Registrational Studies planned for 2023

Orphan and Fast Track	Designations earned with impressive clinical responses across AML populations

Clinical Need | Across R/R and front line, fit and unfit, induction and maintenance therapies

**Commercial Opportunity** | Single agent and combination therapy commercial opportunity in excess of \$1B

#### LUXEPTINIB (CG-806) dual lymphoid and myeloid kinase inhibitor

High Value Targets	R-cell cancers	$\Lambda N / I /$	MDS an	d inflammation:	RTK F	EIT3 ICK	IVN Others
nigii value laigets	D-Cell Callcers,	MIVIL/	ווט כעועו	lu IIIIIaiiiiiiatioii.	DIN, F	LIS, LCN	, LIIN, OTHEIS

Activity in III Patients | Difficult to treat R/R B-cell lymphoma/CLL and R/R AML patients

Improved Formulation | G3 formulation being explored to reduce drug substance and increase plasma exposure







### HM43239 "239"

Oral, Daily, Myeloid Kinase Inhibitor for Genetically-Defined AML Target Populations

# 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 <sup>3</sup>	61,090 <sup>1</sup>	51,820 <sup>3</sup>	14,600 <sup>7</sup>	85,400
AML Incidence	20,240 <sup>2</sup>	16,580 <sup>3a</sup>	<b>6,570</b> <sup>7c</sup>	<b>31,430</b> <sup>3b</sup>
5-Year Prevalence (Leukemia) (2020) <sup>3</sup>	187,560	152,230	41,280	241,750
Mortality (Leukemia)	11,400 (AML) <sup>2</sup>	31,690	8,700 <sup>7</sup>	61,690



Deadly and heterogeneous cancer with 5-year survival rate at diagnosis of approx. 29% Relapsed AML patients have a median life expectancy of < 6 months\* with approved therapies Need more effective & better tolerated targeted agents

DURABILITY to achieve lasting remissions and extend meaningful/quality life

SAFETY for maintenance / MRD+ therapy and for drug combination therapy

BREADTH to better treat R/R AML patients and overcome resistance to current agents

Safe and effective agents expected to expand AML market and command significant market share





#### HM43239 Effective and Well Tolerated Targeted Agent Proven Broad Clinical Activity in AML Patients to Treat Significant Unmet Needs



#### **Validated AML Targets**

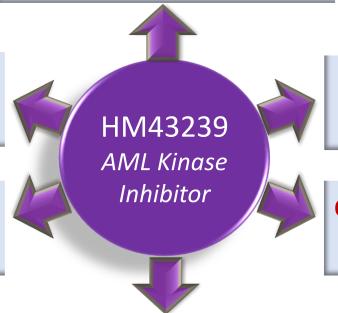
SYK, JAK1/2, FLT3WT/MUT, cKITMUT

#### **Single Agent CRs**

CRs and No DLT at 3 dose levels

#### **ORR 25% in TP53<sup>MUT</sup> R/R AML**

Harboring adverse mutations



#### **Broad Therapeutic Window**

No drug-related SAE, QTc toxicities, or CK increases

#### **ORR 43% in FLT3<sup>MUT</sup> R/R AML**

CRs in patients failed prior FLT3i

Accelerated Paths to Market in R/R Disease

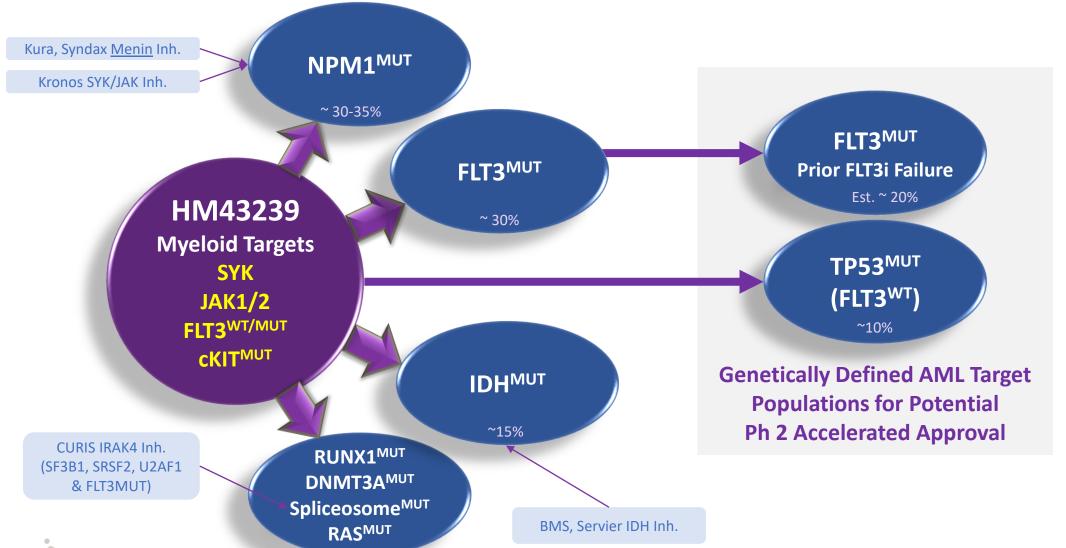
#### **Broad Market Potential**

R/R, 1L, Maintenance/MRD+/Combination





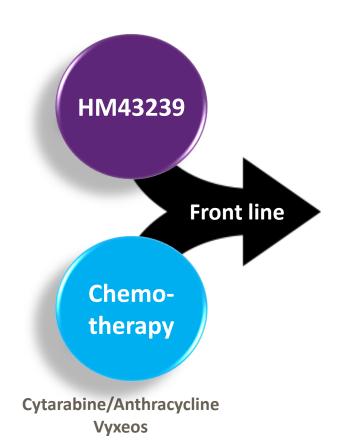
## HM43239: Unlike Any Other Targeted Agent for AML Safely Delivers CRs in Multiple Genetically Defined AML Target Patients

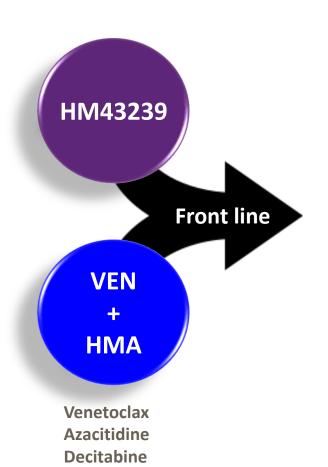


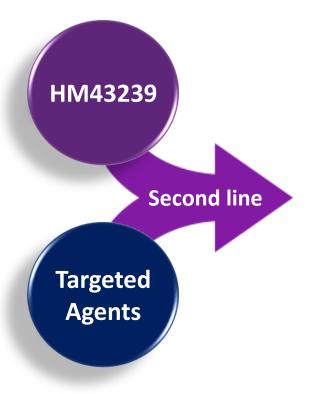
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## HM43239 Effective and Well Tolerated Targeted Agent Potential as Preferred Agent of Choice for Combination Therapy







Anti-CD47 Enasidenib Ivosidenib Menin Inh IRAK4 Inh Glasdegib





# Emerging Clinical Data Support HM43239 as Potential Superior Therapy





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

#### PART A: DOSE ESCALATION (18 Pts Dosed) PART B : DOSE EXPLORATION (39 Pts Dosed) Cohort 6 200 mg QD **Ongoing** 14 Treated → 20 Planned CRc **Completed** 160 mg QD Cohort 5 160 mg QD 16 Treated → 20 Planned (CRC) Cohort 4 120 mg QD **Completed** 120 mg QD Cohort 3 80 mg QD **Completed** 80 mg QD **20 Treated** Cohort 2 40 mg QD **Completed** 40 mg QD **Beginning** Cohort 1 20 mg QD **Completed**

**Favorable safety profile:** No drug related SAE or death and no observed relation between delta-QTc throughout the trial. And no DLT through 160 mg dose level.

**Dose Exploration continues across several cohorts:** currently enrolling patients at 120 mg and 160 mg dose levels and plan to explore 40 mg dose level





#### HM43239 Safety and Efficacy Data Broad Therapeutic Window as a Single Agent in R/R AML Patients

#### Safety Profile Favorable to Date

- No drug related SAE, deaths, or AE of elevated CK (creatinine kinase)
- No drug related AE of QT prolongation No observed relation between  $\Delta$ QTc and dose
- No DLT up to 160 mg and one DLT of muscle weakness (not rhabdomyolysis) at 200 mg

#### Demonstrated Efficacy Across a Diverse Set of R/R AML Patients

- CRc in AML with adverse mutations (FLT3<sup>WT</sup>) incl. TP53-mutant and complex karyotype
- CRc in FLT3-mutant AML (Fast Track) incl. prior failure of other FLT3 inhibitors

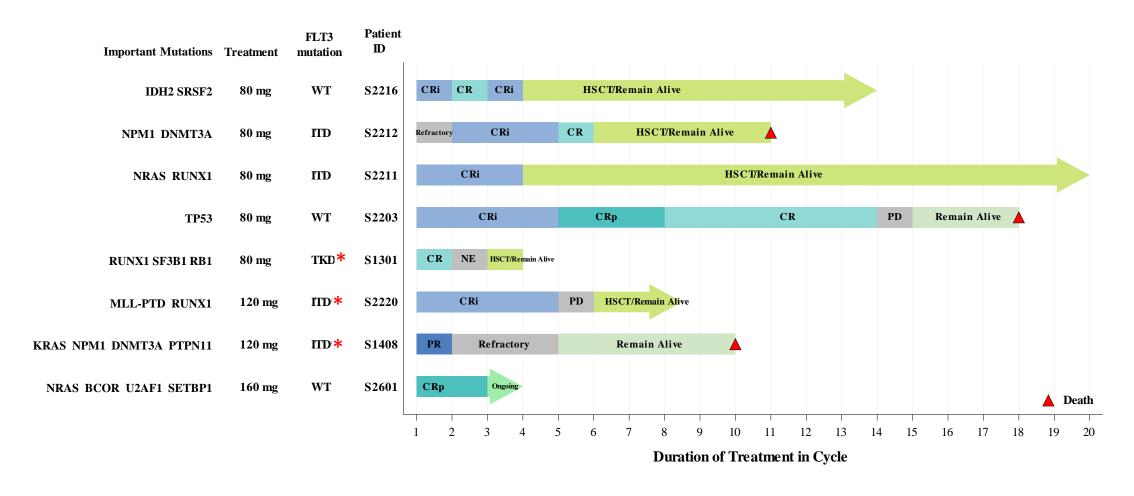
#### Identified a Broad Therapeutic Window

- 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
- Safety profile supports combination therapy with other agents





## HM43239 Diversity of Genetically-Defined R/R AML Patients Who Achieved a Clinical Response to Date in Phase 1/2 Study



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.

BIOSCIENCES



### HM43239 Diversity of Genetically-Defined R/R AML Patients Who Achieved a Clinical Response to Date in Phase 1/2 Study

<b>Important Mutations</b>	FLT3 Status	<u>Dosage</u>	Best Response	<u>HSCT</u>
IDH2 SRSF2	Wild Type	80 mg	CR	Yes
TP53	Wild Type	80 mg	CR	Ineligible
NRAS BCOR U2AF1 SETBP1	Wild Type	160 mg	CRp	In Process
NPM1 DNMT3A	ITD	80 mg	CR	Yes
NRAS RUNX1	ITD	80 mg	CRi	Yes
RUNX1 SF3B1 RB1	<b>TKD</b> Prior FLT3i	80 mg	CR	Yes
MLL-PTD RUNX1	ITD Prior FLT3i	120 mg	CRi	Yes
KRAS NPM1 DNMT3A PTPN11	ITD Prior FLT3i	120 mg	PR	Ineligible

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.

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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.



### HM43239 Overall Response Rate (CRc + PR) 7 CRc and 1 PR to Date in Phase 1 as a Single Agent in R/R AML Patients

Mutation	All Patients			Evaluable Patients			
Status N = 45 Patients	Number Responders	Response Rate	N = 41 Patients	Number Responders	Response Rate		
FLT3+	20	4CRc   1PR	25%	19	4CRc   1PR	26.3%	
FLT3+/prior FLT3i	7	3	42.9% (CRc + PR) 28.6% (CRc only)	7	3	42.9% (CRc + PR) 28.6% (CRc only)	
FLT3-WT	25	3	12%	22	3	13.6%	
TP53+	4	1	25%	3	1	33.3%	

## Overall Response Rate for "All Patients" and "Evaluable Patients" Receiving ≥ 80mg HM43239

- Findings represent a snapshot in time: The reported safety, tolerability, PK, PD and efficacy findings reported herein represent the data available may change as additional patients are assessed and more data are collected.
- "Evaluable Patients" removes those nonevaluable patients who did not have a response evaluation and had no other evidence indicating refractory disease in the peripheral blood.
- Most CRc patients went to HSCT and cannot be evaluated for transfusion independence assessment.

Abbreviation: CR, complete remission; CRc, composite complete remission; CRp, complete remission with incomplete platelet recovery; CRi, complete remission with incomplete hematological recovery; PR, partial remission. Note: efficacy evaluable patients include all patients with at least 80% drug compliance during Cycle 1 or who had reported a DLT during Cycle 1, and who reported relevant data for efficacy interpretation such as bone marrow assessment, CBC counts, reason for treatment termination.



<sup>[1]</sup> Overall response includes CRc and PR.

<sup>[2]</sup> CRc includes CR, CRh, CRp and CRi.

<sup>[3]</sup> The reported prior FLT3 inhibitors include gilteritinib, midostaurin and soranfenib.

#### HM43239 Potential for Accelerated Path Supported by Expansion & Registration Trials

#### Ongoing Dose Escalation/Dose Exploration Phase 1/2 Trial in R/R AML Patients

- Continue Exploration of Highly Adverse Molecular Genotypes (TP53-Mutated, etc.) for Potential Fast Track Designations
- Continue Dose Exploration at 40 mg, 120 mg and 160 mg to Deliver Response Rate Updates & Rolling News Flow

#### **Doses and Patient Populations have been Selected for Expansion Trials**

- Three Safe and Efficacious Doses Identified
  - o **120 mg** planned as **Primary** Single Agent Expansion Dose with **80 mg** and **160 mg** as Bracketing Doses
- Expansion with FLT3 Mutated R/R AML Population (Fast Track Designation)
  - o Includes FLT3+/Prior FLT3i Failure target population for potential accelerated approval
  - o Includes broader FLT3-mutated population to support full approval trials in FLT3-mutated AML
  - o Plan single agent to begin 2H2022 and combination (239+Ven) to begin thereafter
- Expansion with FLT3-Unmutated R/R AML Population (with Adverse Mutations)
  - Including <u>TP53-Mutated</u> target population for <u>potential accelerated approval</u>
  - o Includes broader population to support full approval trials in NPM1/MLL, RUNX1-DNMT3A-Ras and other populations
  - Plan Single Agent to begin 2H2022 and Combination (239+Ven) to begin thereafter
- Expansion Includes Broader Populations to support full approval: NPM1/MLL, RUNX1-DNMT3A-RAS, Broad FLT3+ and Others

#### **Expansion Trials in AML Patients Serve as Segue to Registrational Trials**





# HM43239 Global Dose Expansion Trial Planned to Support Phase 2 Registrational Trials for Accelerated Approval and Drug Combination Trials for Broad Commercialization

**Registrational Single Agent Trials Planned to Begin in Expansion Trials 2022-2023 2023** if Data from Expansion Trial Support Registrational Phase 2 study – Single Agent **HM43239 Single Agent Accelerated** FLT3<sup>MUT</sup> R/R AML with prior FLT3i therapy **FLT3-Mutated Cohort: Approval** Prior FLT3i and Registrational Phase 2 study – Single Agent FIT3i naïve Marketing TP53<sup>MUT</sup> R/R AML, Complex Karyotype, or Other Group **FLT3-Unmutated Cohort:** TP53<sup>MUT</sup>/Complex Karyotype Other **Drug Combination Trial to Support Usage of** 239 with Other Agents in AML Populations HM43239 + Venetoclax Combo **FLT3-Mutated Cohort Combo Phase 2-3 Randomized Study FLT3-Unmutated Cohort** 1L unfit AML: HMA/VEN vs. 239/HMA/VEN + maint 1L fit AML, FLT3-WT: Chemo vs. 239/Chemo + maint • Single agent Expansion studies designed to collect data on a small number **Combo Phase 2 Randomized Study** of patients in "high need" groups and segue into Ph 2 Registrational Trial(s) R/R AML in 2L, FLT3 mutant - 239 vs. 239/VEN vs. GILT • Combination Expansion studies designed to illustrate safety and efficacy of 239 with venetoclax and segue into Phase 2-3 randomized studies and demonstrate 239 can be the preferred agent for combination therapy

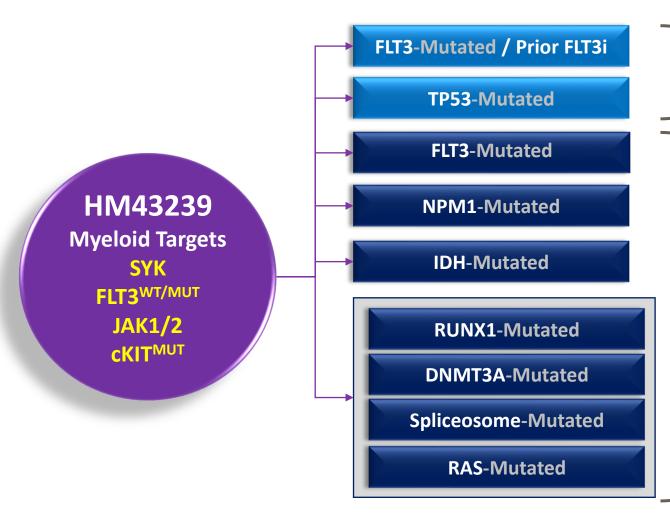


# Clinical Development Plan Sets the Stage for Broad Commercial Success





# HM43239: Positioned for Accelerated Approval & Traditional Development Broad Commercial Opportunities >\$1 billion in Multiple AML Target Populations



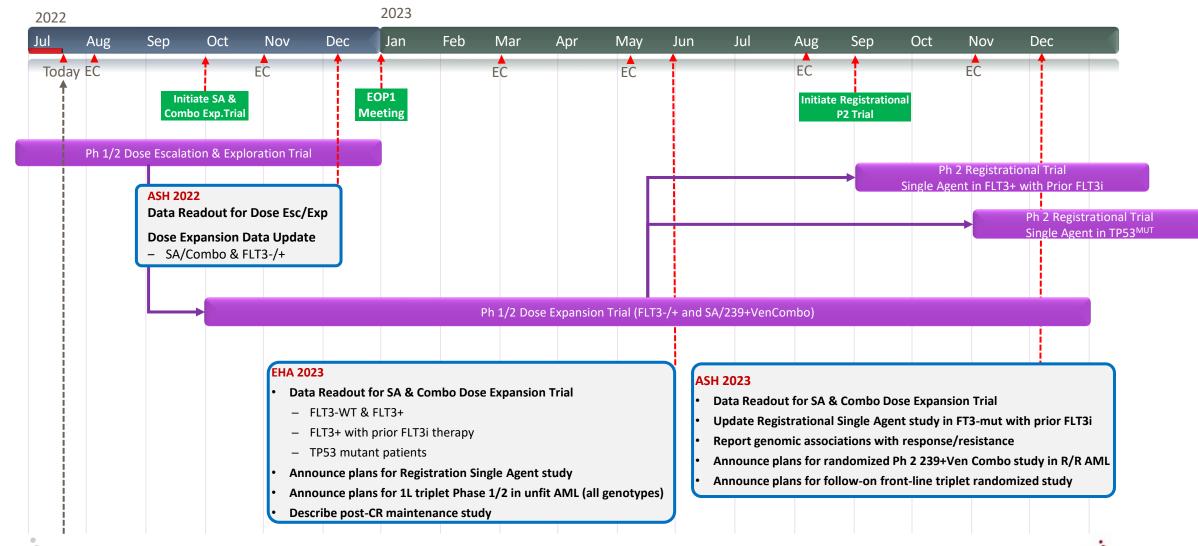
Genetically-defined target populations for potential single agent Phase 2 accelerated approval path



Genetically-defined target populations for potential to expand market through combo and IIT development programs



### HM43239 Clinical Development Timeline, Clinical Data Release and Potential Value Driving Milestones





### HM43239 Clinically Validated, Once Daily, Oral Myeloid Kinase Inhibitor Confidence of Clinical Investigators and KOLs



### Targets Constellation of Kinases Important in AML

- Potent inhibitor of myeloid kinases SYK, FLT3<sup>WT/MUT</sup>, JAK1/2 and mutant forms of c-KIT associated with transformation and resistance
- Potential to treat genetically defined AML patients across multiple lines of therapy & populations
- Safety & efficacy foretell significant market potential for R/R, 1L, FLT3-/+, Fit/Unfit AML populations



### **Clinical Validation Supports Path of Rapid Development for Breadth of AML Patients**

- FLT3-Mutated Patients
  - CRc in patients who failed prior FLT3 inhibitors
     CRc in patients with ITD and TKD mutated FLT3
  - FDA Fast Track received for FLT3<sup>MUT</sup> R/R AML

#### FLT3-Unmutated Patients

- CRc in genetically-defined patients with specific mutations: NPM1, MLL, TP53, DNMT3A, N/KRAS, IDH2, U2AF1, RUNX1, Others
- Broad Therapeutic Window
  - Well tolerated across three active & safe doses
- Preferred Agent Profile for Combination Therapy



### Program Goals Supporting Rapid Development

- Explore Molecular Subgroups for Potential Fast Track Designations
- Single Agent Expansion Trial (239) planned 2H2022
- Combo Expansion Trial (239+Ven) planned 2H2022
- Registrational Ph2 study(ies) planned 2023 from Expansions
- Broad commercialization goals supported by clinical development in diverse patient populations





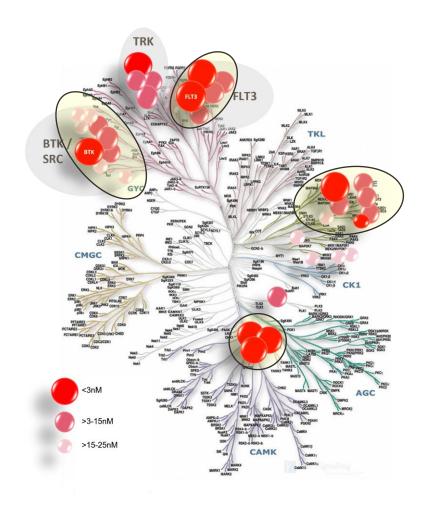


#### LUXEPTINIB

Oral Lymphoid & Myeloid Kinase Inhibitor

CONFIDENTIAL

#### Luxeptinib: Atypical, Dual Lymphoid and Myeloid Kinome Inhibitor



Unique Kinome Targeting Inhibits high value targets: BTK, FLT3, CSF1R, PDGFR $\alpha$ , TRK, AURK 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





#### HM43239 Effective and Well Tolerated Targeted Agent Proven Broad Clinical Activity in AML Patients to Treat Significant Unmet Needs

### Phase 1a/b R/R B-cell leukemias/lymphomas

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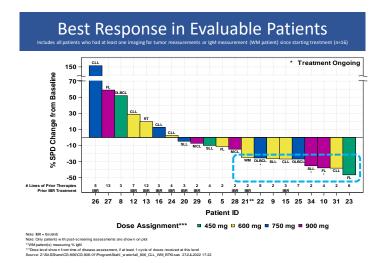
36 Patients dosed

#### Targets Key Kinases in Lymphoid and Myeloid Cancers

- Inhibits BTK, FLT3, CSF1R, PDGFRα, TRK, AURK, others
- Current dosing at 900mg BID with original formulation
- Generally well-tolerated
- To date 64 patients dosed

#### **Antitumor activity in diverse B-cell cancers**

Multiple patients experienced tumor shrinkage below baseline but none with ≥ 50% reduction



Phase 1a/b
R/R AML and MDS

28 Patients dosed



#### **MRD-negative Complete Remission**

Observed in one heavily pretreated relapsed AML patient at 450mg BID dose level





#### **Luxeptinib** Ongoing Activities

- Critical Step for Lux Program is the Transition to an 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
- Following a Single Dose of Lux G3, Patients Continue on Study Using the Original Formulation
- 13 Patients Enrolled Thus far to Test a Single Dose of Lux G3

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4 at 50mg | 4 at 100mg | 4 at 200mg | 1 so far at 10mg
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• The Single Dose PK Data with G3 are being Evaluated with PK Modeling Under Conditions of Once or Twice Daily Continuous Dosing





#### 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 increase exposure and to lower pill burden and drug substance manufacture
- G3 early data are encouraging
- PK modeling of continuous dosing with preliminary PK data has been initiated
- Plan continuous dosing with G3 if the data from single dose and modeling are supportive





# Aptose Biosciences (APTO) Key Financial Highlights Q2/2022

#### **Q2** Financials:

- The company's cash balance at June 2022 was \$62.4M
- Cash burn during Q2 was \$7.1M
- Cash runway into Q1 of 2024
- The company is pre-revenue
- The net loss for the second quarter was \$10.6M,
  - Which is down from \$13.5M in the same quarter last-year
- The net loss YTD was \$22.1M
  - Which is down from \$29.7M in YTD last-year
- Net loss per share Q2 (\$0.11) and YTD (\$0.24)

#### **Upcoming Investor Conferences:**

- Boston: Canaccord
- NYC: HCW 9/13, Cantor 9/28: Piper 11/29; & Others
- Oppenheimer Oncology Summit at MD Anderson
- ASH Early December

#### **Capitalization:**

- The market capitalization is approximately \$80 million
- The recent market capitalization high was \$294M 6/2021
  - Before the acquisition of HM43239, which we acquired for \$12.5M
- Common stock outstanding 92 million as of June 2022
- No debt or warrants

#### **Trading Statistics:**

• The 52-week trading range: high of \$3.13 & low of \$0.73

#### **ATM Program**:

Piper & Canaccord as Co-Agents



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    - Strong enough to deliver single agent CRs yet gentle enough for safety in R/R AML patients
    - Intense support from clinical investigators in single agent and drug combination trials
    - Efficacy & safety profile position as a preferred agent for combination therapy and broad commercial use
  - Multiple genetically-defined AML target populations as potential indications for >\$1bn commercial market
    - Targets more genetically-defined AML target populations than SYK inhibitors, IRAK4 inhibitors, or Menin inhibitors
    - Response rates in AML populations of unmet needs that may support single agent Phase 2 accelerated approvals
  - Strong intellectual property estate
- Experienced leadership team with deep expertise in kinase inhibitors & orphan hematologic diseases
- Meaningful near-term upside with value-driving clinical updates and milestones through 2022 and 2023





