Aptose Corporate Presentation

March 2022

APTOSE BIOSCIENCES

PRECISION ONCOLOGY FOR THERAPIES OF TOMORROW

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

Disclosure

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Aptose Biosciences (NASDAQ: APTO)

APTOSE

Hematology company focused on precision medicines

Experienced leadership with deep expertise in kinase inhibitors

Orphan hematology programs with broader oncology optionality

Rolling presentation of value-driving clinical findings through 2022

HM43239

Clinically validated *Myeloid Kinome Inhibitor (MKI)*

High value targets: SYK, FLT3, cKIT^{MUT}, JAK

Multiple complete responses (CR) in ongoing Phase 1/2 study of R/R AML

CR in diverse AML patients with NPM1^{MUT}, TP53^{MUT}, N/K-RAS^{MUT}, IDH^{MUT}, FLT3$^{ITD/TKD/WT}$

2022: Select dose(s) and initiate expansion trials as monotherapy and in combination

LUXEPTINIB (Lux, CG-806)

Dual *Lymphoid and Myeloid Kinome Inhibitor (LKI/MKI)*

High value targets: BTK, FLT3, CSF1R, PDGFRα, TRK, AURK

Ongoing parallel dose escalations in patients with B-NHL and AML/MDS

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

2022: Select optimal formulation and dose(s) for continued development

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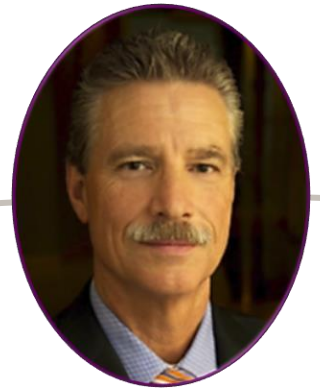
Aptose Leadership Team: Multifaceted Expertise in Therapeutic Development

Rafael Bejar, MD, PhD

Senior Vice President & Chief Medical Officer



William G. Rice, PhD

Chairman, President & Chief Executive Officer

Jotin Marango, MD, PhD

Chief Financial Officer & Chief Business Officer

UC San Diego Moores Cancer Center

mds Myelodysplastic Syndromes (MDS) Foundation

NIH National Cancer Institute

Emory University School of Medicine

Mount Sinai

Dana-Farber Cancer Institute

BWH Brigham and Women's Hospital

Achillion

Cylene Pharmaceuticals

HCW

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Aptose SAB: Distinguished Opinion Leaders with Deep Oncology Expertise

Daniel Von Hoff, MD, FACP

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

Brian J. Druker, MD

Pioneer in the field of precision medicine

Key Role in 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

Michael Andreeff, MD, PhD

Renowned hematology specialist

Professor of Medicine
Paul and Mary Haas Chair in Genetics
Chief, Section of Molecular Hematology and Therapy
MD Anderson Cancer Center

Expert in AML and other hematologic malignancies

Expert in drug resistance and drug mechanisms

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Clinical Stage Pipeline: Oral Kinase Inhibitors that Cover a Broad Spectrum of Hematologic Malignancies

Program	*Target*	*Indication*	*Preclinical*	*Phase 1 Proof-of-Concept*	*Phase 2/3 Registrational*
HM43239	*Myeloid Kinome*	*AML*		**Phase 1/2**	
Luxeptinib	*Myeloid Kinome*	*AML, MDS*		**Phase 1a/b**	
Luxeptinib	*Lymphoid Kinome*	*B-cell Cancers*		**Phase 1a/b**	

- Small molecule kinase inhibitor candidates designed to treat a disease
- Confirmed anti-leukemic activity in dose-escalation studies, with expansion studies planned
- Orphan hematology programs, with broader optionality into solid tumor indications

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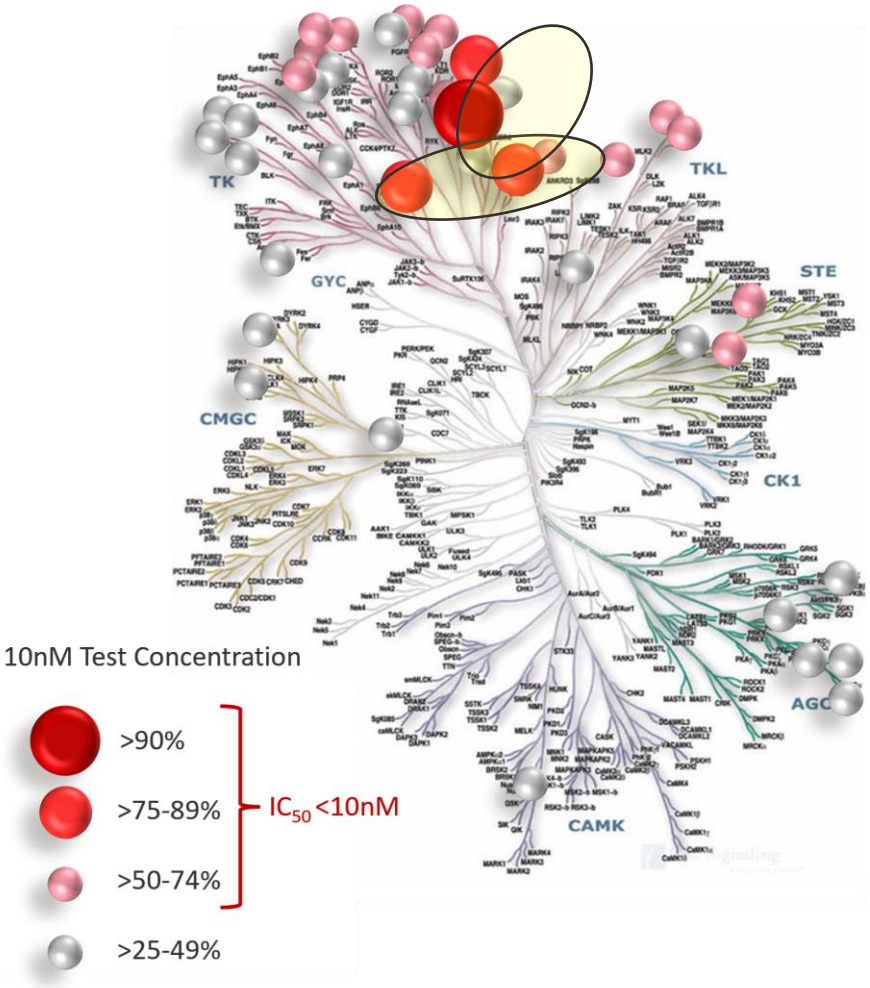
HM43239

Oral Myeloid Kinome Inhibitor

HM43239: Clinically Validated Myeloid Kinome Inhibitor



Targets a constellation of kinases, including SYK, FLT3, cKIT^{MUT}, JAK, others

Suppresses resistance-conferring FLT3 mutations of ITD, activation loop and gatekeeper

Suppresses resistance-conferring growth factor pathways via JAK/STAT and MAPK/ERK

Treating R/R AML patients in an ongoing international dose escalation Phase 1/2 study

Clinically validated: multiple Complete Responses (CRs) with once daily oral dosing

CRs in diverse AML patients: **NPM1^{MUT}, TP53^{MUT}, RAS^{MUT}, IDH2^{MUT}, FLT3^{WT}, FLT3$^{TKD/ITD-MUT}$**

Optionality to cover multiple tumor genotypes and different stages of AML

Acquired in Nov 2021; Assumed clinical oversight of IND and CRO in Jan 2022

2022: **Rolling clinical updates** (medical meetings, investor events, corporate updates)

2022: Plan to select dose and **initiate monotherapy and combination expansion trials**

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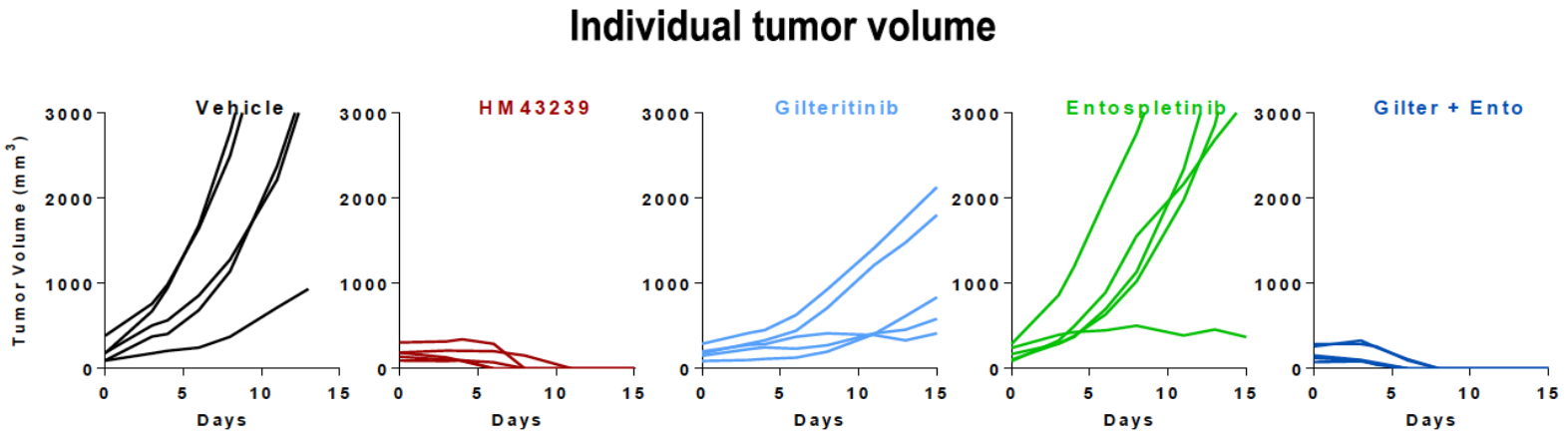
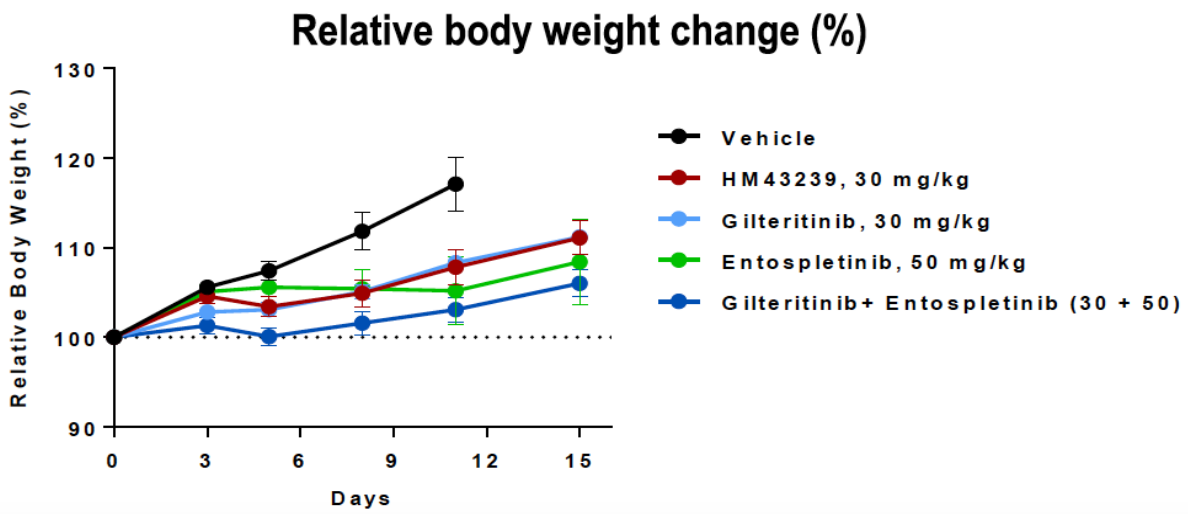
HM43239: *In Vivo* Models Suggest Superior Antitumor Activity and Favorable Tolerability Relative to Established Kinase Inhibitors in AML

MOLM-14$^{ITD/F691L-MUT}$ AML cells used with an *in vivo* murine xenograft model:

- MOLM-14$^{ITD/F691L-MUT}$ is an AML cell harboring the ITD and F691L dual mutant form of FLT3
- Cells resistant to gilteritinib FLT3 inhibitor
- HM43239 inhibits SYK and FLT3 harboring the ITD and F691L
- HM43239 superior antitumor activity to:
  - Gilteritinib FLT3i
  - Entospletinib SYKi
- HM43239 monotherapy overall superior to combination of Gilteritinib plus Entospletinib





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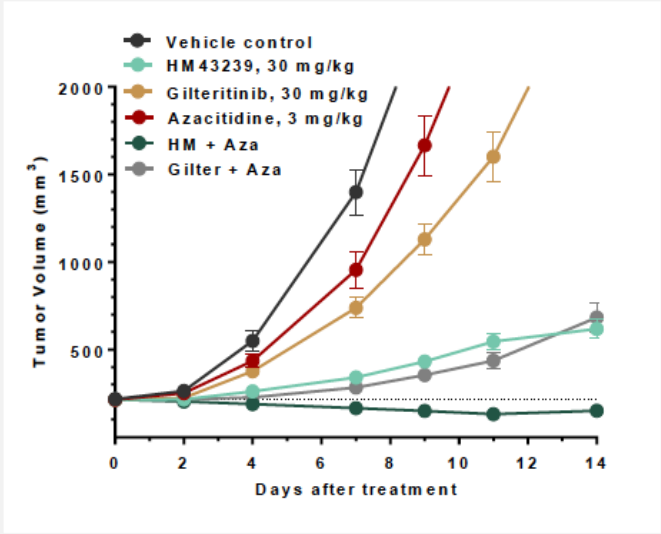
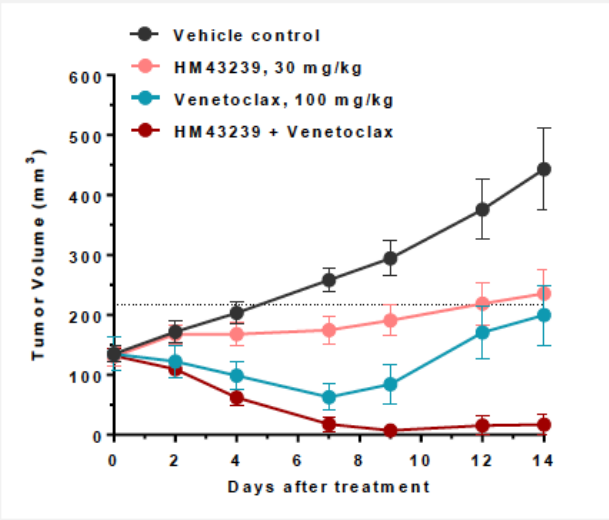
HM43239: *In Vivo* Models Suggest Synergy with Inhibitors of DNMT, BCL-2, or MDM-2, and Combinatorial Optionality in AML

Combo w/ Azacitidine (DNMT)
(MOLM-14-F691L cell model)



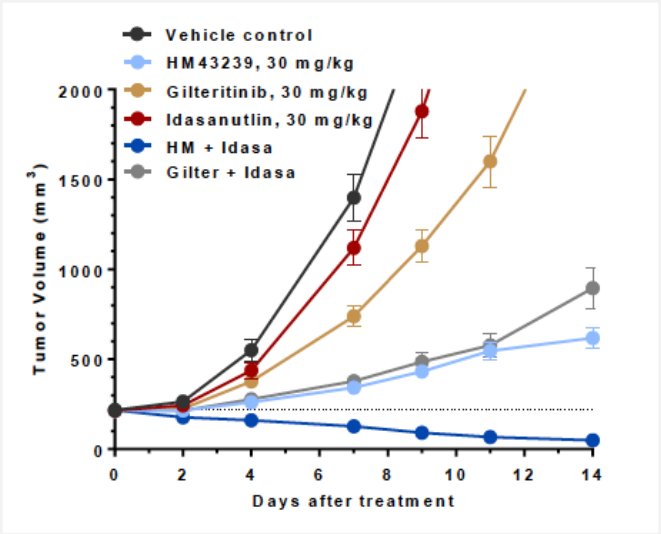
HM43239, in subcutaneous xenograft, superior efficacy to Gilt or Aza alone and combines effectively with each against MOLM-14$^{ITD/F691L\text{-}MUT}$ AML

Combo w/ Venetoclax (BCL-2)
(MV-4-11 cell model)



HM43239, in subcutaneous xenograft, superior efficacy to Venetoclax alone and combines effectively with Ven against MV4-11 AML

Combo w/ Idasanutlin (MDM-2)
(MOLM-14-F691L, NOG mouse n=10)



HM43239, in circulating AML model, superior efficacy to dasanutlin MDM2i alone and combines effectively with Idasanutlin against MOLM-14$^{ITD/F691L\text{-}MUT}$

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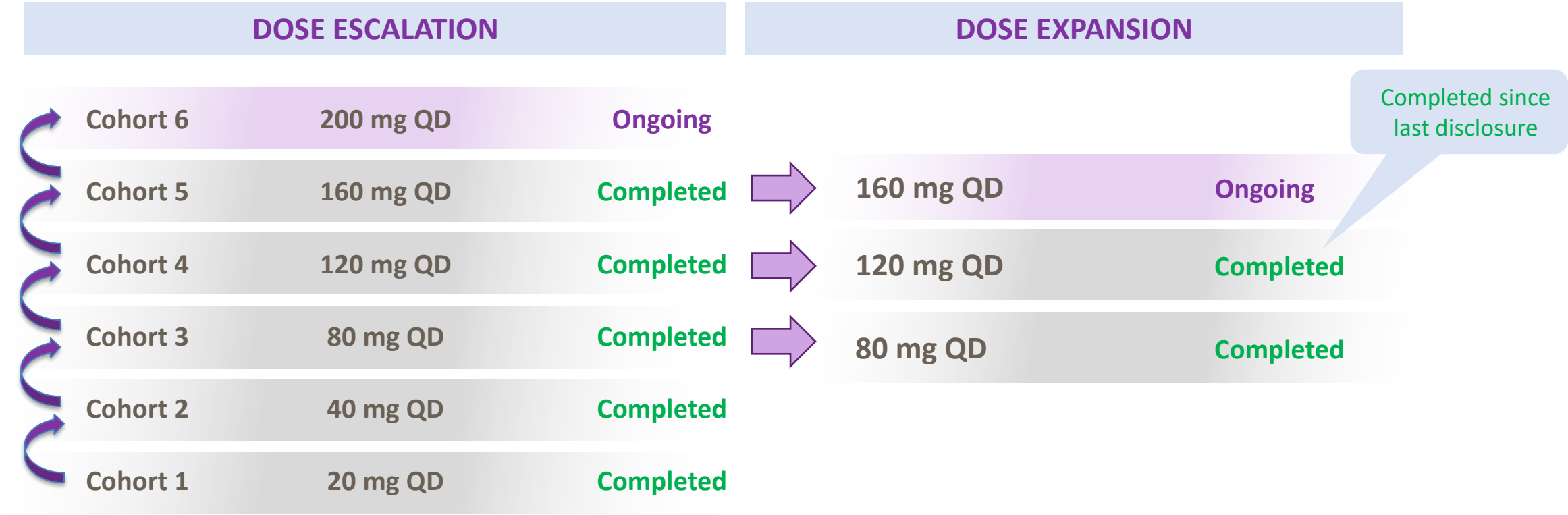
HM43239 Phase 1/2 Study in AML: Ongoing Dose Escalation & Dose Expansion



Favorable safety profile: only mild AEs, no DLTs and no discontinuations from drug related toxicity through the completed 160 mg dose level.

Study ongoing across several cohorts: the dose escalation cohort of 200 mg and the dose expansion cohorts of 120 mg and 160 mg are currently enrolling.

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HM43239 Demonstrates Dose-Dependent PK and Target Engagement

Plasma PK

Daily administered oral doses of 20, 40, 80, 120, 160 and 200mg. Plasma samples not available for all patients to date and all timepoints to date.

FINDINGS:

Generally dose-related increase in plasma exposures

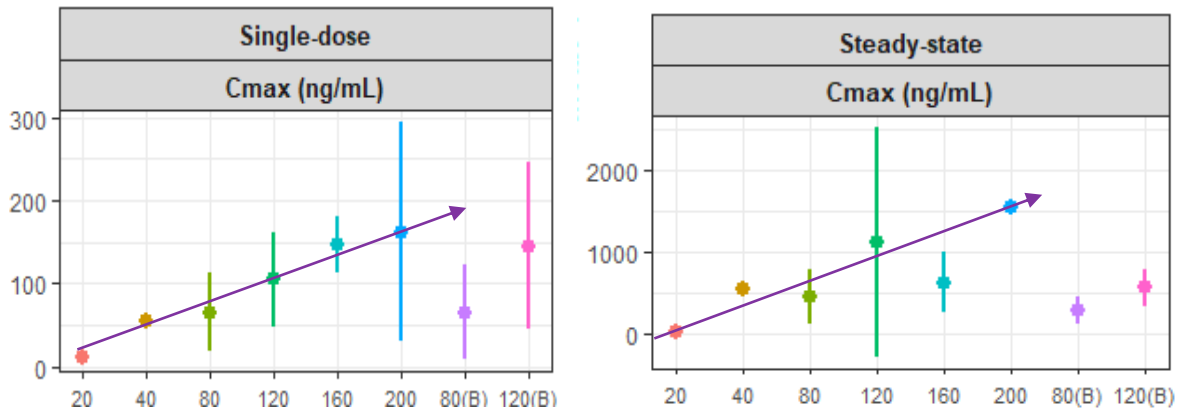
Plasma inhibitory activity (PIA) Assay

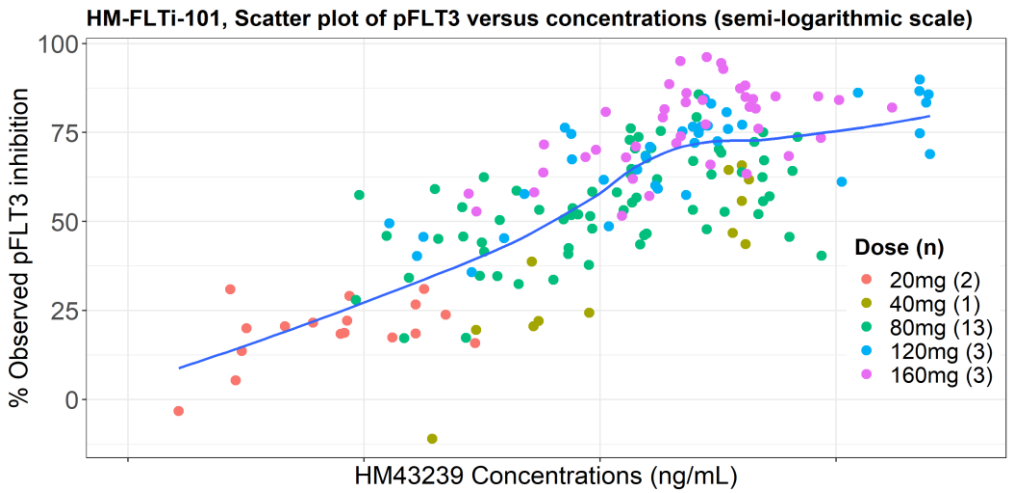
Measures the ability of patient plasma to inhibit phospho-FLT3 in MOLM-14 reporter cell line

FINDINGS:

PIA was dose-dependent with up to 90% phospho-FLT3 inhibition at dose levels ≥ 80 mg.

PK Parameter Mean ± SD





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HM43239 Demonstrates Durable Clinical Benefit in R/R AML (*ASH 2021*)

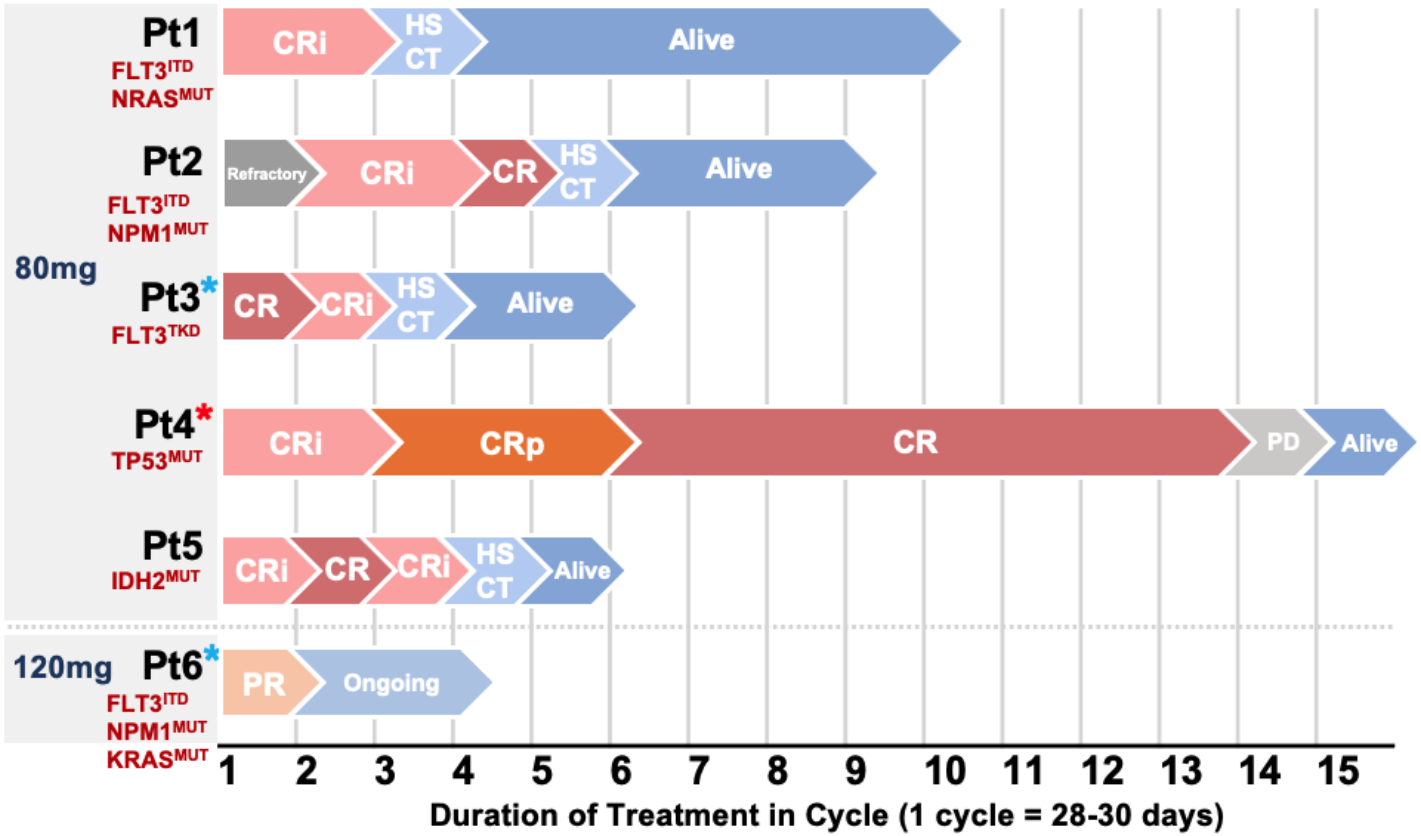
Highlights from 80 mg Composite Cohort
(Dose Escalation + Dose Expansion; n=20)

CRc among FLT3^{MUT} patients at 80mg
- 37.5% (3 of 8) achieved a durable composite complete response (CRc, CR + CRi)
- Includes prior gilteritinib failure patient

CRc among allcomers at 80mg
- 25% CRc rate observed when combining both FLT3^{MUT} *and* FLT3^{UNMUT} AML patients

Meaningful clinical benefit in all 5 CR
- 80% (4 of 5) responders advanced to HSCT
- >1 year duration of response in a relapsed TP53^{MUT} AML patient age-unfit for HSCT

Data cutoff: August 31, 2021



*Patient failed prior gilteritinib therapy
*Patient died ~4 months after progression

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HM43239: Characteristics of AML Patients with Clinical Responses (*ASH 2021*)

Patients	Age/Sex	Dose Level (QD)	AML status	FLT3 mutation* (mt to WT signal ratio)	Other mutation	No. prior therapies	No. prior FLT3i's	Cytogenetics	Best response	Cycles** to first response	Cycles** to best response	Duration on study (weeks)	Reason of discontinuation
Pt1	51/M	80mg	AML NOS	FLT3-ITD† (0.7)	NRAS, RUNX1	3	0	Normal	CRi	1	1	12	HSCT (remains in remission)
Pt2	50/M	80mg	AML NOS	FLT3-ITD (13.5)	NPM1, DNMT3A	1	0	Normal	CR	2	5	20	HSCT (remains in remission)
Pt3	67/F	80mg	AML NOS	FLT3-D835V† (0.11)	RUNX1	2	2# (midostaurin, gilteritinib)	Normal	CR	1	1	7	HSCT (remains in remission)
Pt4	60/M	80mg	AML-MRC	FLT3-WT (0)	TP53	3	0	Abnormal‡	CR	1	8	56	PD → death (remains in remission)
Pt5	63/F	80mg	AML NOS	FLT3-WT† (0)	IDH2	1	0	Abnormal‡	CR	1	2	13	HSCT (remains in remission)
Pt6	54/F	120mg	AML NOS	FLT3-ITD† (23.82)	NPM1, DNMT3A, KRAS, PTPN11	2	2# (midostaurin, gilteritinib)	Normal	PR	1	1	-	Ongoing in PR in C#2

Data cutoff: August 31, 2021

*FLT3 mutation status is based on the results from invivoscribe using the Leukostrat® CDx FLT3 Mutation Assay approved by FDA. **1 Cycle is 28 or 30 days.
†Pt1 mutation status is based on initial diagnosis; Pt3 mutation result was obtained after dosing; Pt5 mutation status at initial diagnosis was FLT3-ITD; Pt6 mutation at initial diagnosis was FLT3-ITD/TKD.
‡Karyotypes for Pt4: Abnormal, Complex, 50~52,XY,del(5)(13q31),-7, dup(8)(q22), dup(9)(q13), +dup(11)(p11.2), -13,-15,+5~7mar[cp14]/46,XY[6]; Karyotypes for Pt5: Abnormal, +8, +13, t(X;9)(q28;p21)
AML NOS: AML not otherwise specified, AML-MRC: AML with myelodysplasia-related changes, HSCT: hematopoietic stem cell transplantation, PD: progression of disease
#Pt3 previously received midostaurin and gilteritinib with no responses; Pt6 previously received midostuarin with CR and gilteritinib with no response.

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), as well as resistance-conferring D835 and gatekeeper (F691) tyrosine kinase domain (TKD) mutations

Activity To Date Suggests Potential Rapid Development

- Broad activity suggests a genotype agnostic agent
- Identified potential *minimum therapeutically effective dose*
- Will explore development as single agent and combination therapy, broadly and in genetic subgroups

Program Goals for 2022 Supporting Rapid Development

- Rolling presentation of clinical findings throughout 2022
- Select optimal Expansion Dose
- Select patient genotypes for further clinical development
- Initiate expansion studies as single agent and in combination
- Plan for registrational studies

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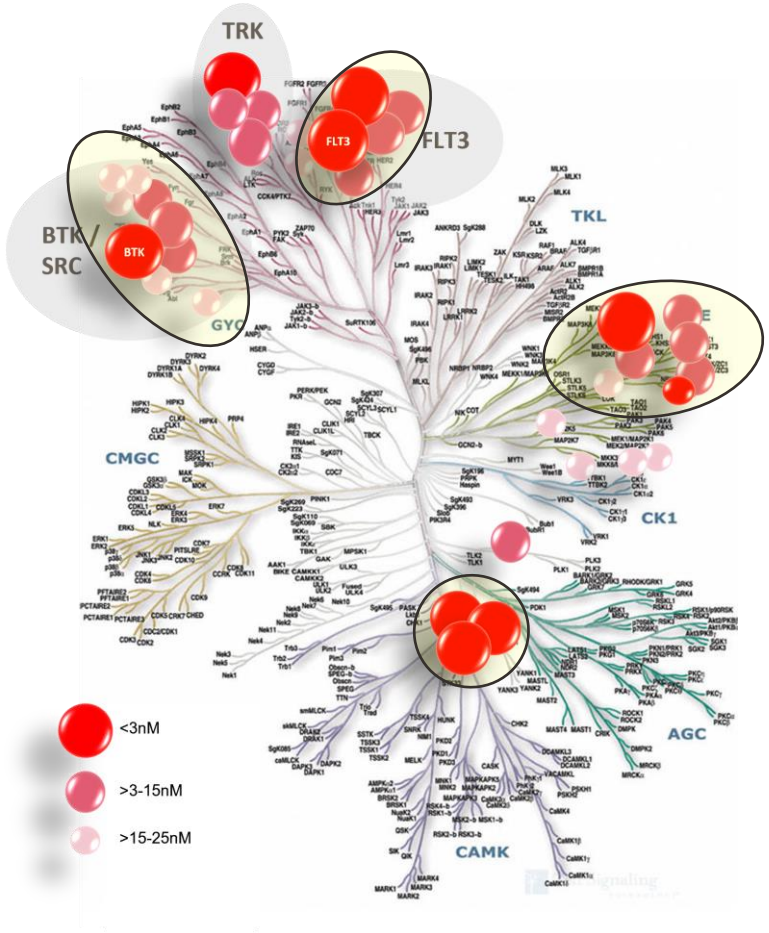
Luxeptinib

Oral Lymphoid & Myeloid Kinome Inhibitor

Luxeptinib: Atypical, Dual Lymphoid and Myeloid Kinome Inhibitor



Unique Kinome Targeting

- Inhibits **high value targets**: BTK, FLT3, CSF1R, PDGFRα, 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

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Luxeptinib: Ongoing Phase 1a/b Study in Heavily Pretreated B-cell Malignancies

Following completion of Cohort 6 with original G1 formulation, additional patients enrolling with new G3 formulation

Cohort 6	900 mg Q12H	**Completed**
Cohort 5	750 mg Q12H	**Completed**
Cohort 4	600 mg Q12H	**Completed**
Cohort 3	450 mg Q12H	**Completed**
Cohort 2	300 mg Q12H	**Completed**
Cohort 1	150 mg Q12H	**Completed**

Objectives

Ongoing Phase 1 a/b, open-label, single arm, multicenter, 3 + 3 dose-escalation clinical study (NCT03893682).

Primary objectives:

- Assess safety and tolerability of luxeptinib (CG-806)
- Determine recommended Phase 2 dose (RP2D)

Key secondary objectives:

- Assess PK profile and PD activity
- Obtain preliminary evidence of antitumor activity
- Characterize the bioavailability (BA) of an automated filled (G2) vs. the original hand-filled (G1) formulations

Dose Escalation Phase

- Patients administered **oral capsules, twice daily** on a **28-day cycle**
- Plan to perform 7 dose levels
- Planned expansion cohorts
- **Accelerated titration** design
- Additional patients may be enrolled (**back filling**) at dose levels previously declared safe
- **Intra-patient dose escalation** is allowed if higher dose is safe in 3 or more patients

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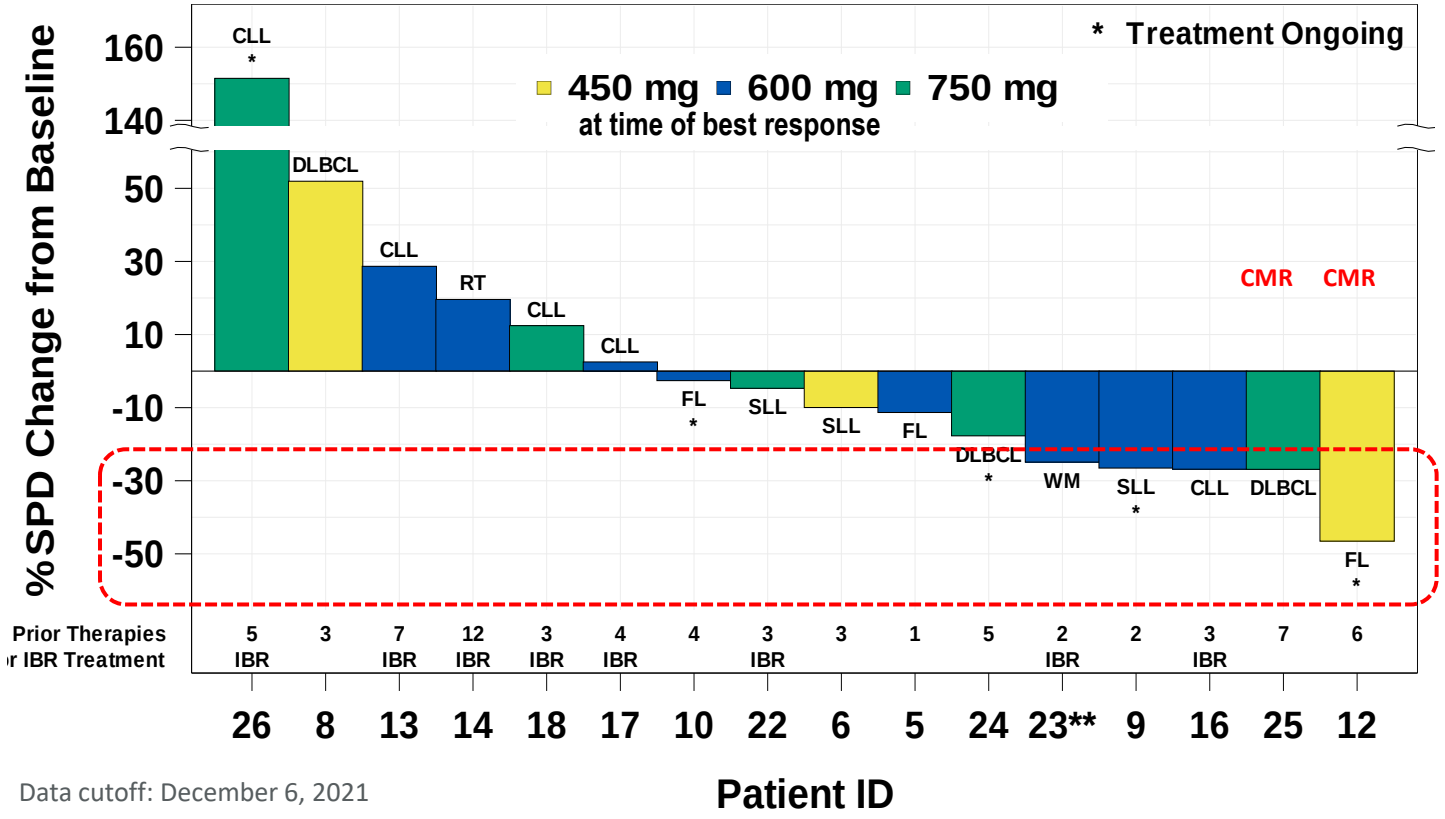
Luxeptinib: Waterfall Plot Shows Encouraging Antitumor Activity Trend in Heavily Pretreated Patients with B-Cell Malignancies (*ASH 2021*)

Encouraging Trends:

- Observing greater antitumor activity with higher dose levels, higher plasma concentrations, and longer time on study drug
- Observing antitumor activity across diverse B-cell cancers

Best Response in Evaluable Patients

Includes all patients who had at least one imaging for tumor measurements or IgM measurement (WM patient) since starting treatment (n=16)



Data cutoff: December 6, 2021

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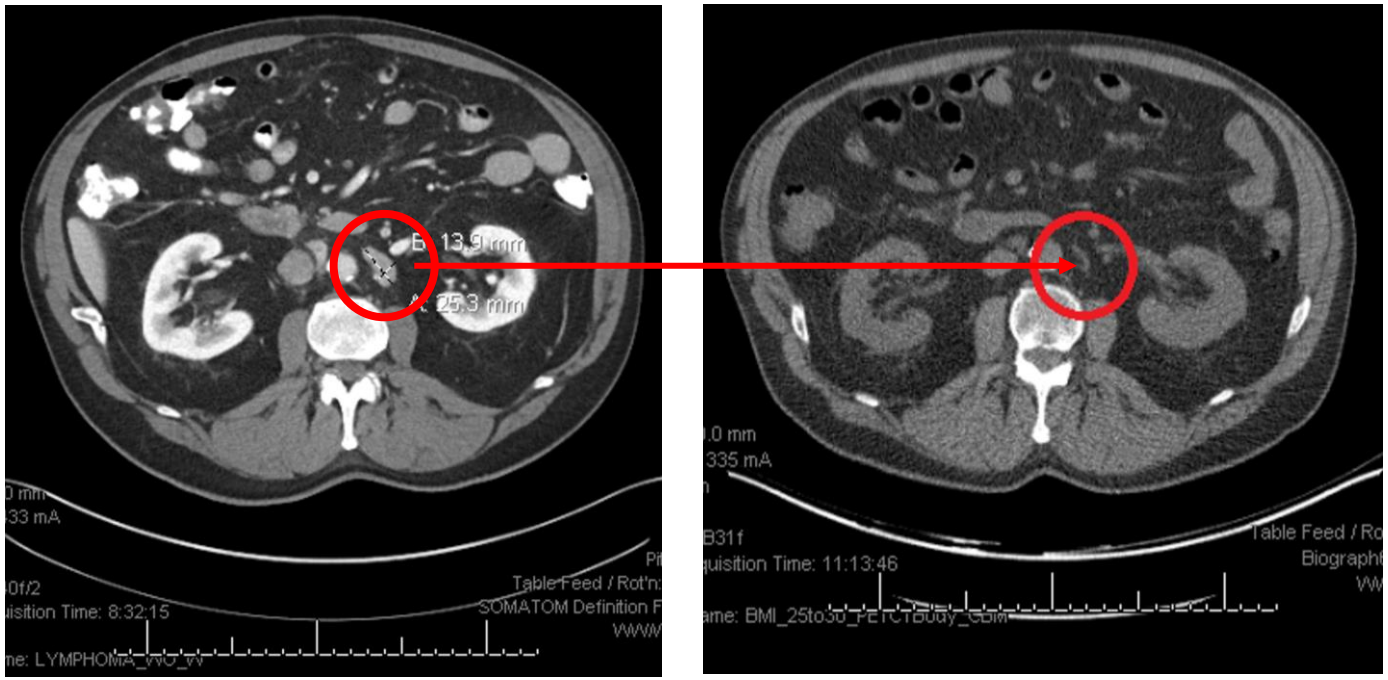
Luxeptinib Case Study: Significant Tumor Reduction (47%) with Accompanying Complete Metabolic Response (CMR) in Patient with Refractory Follicular Lymphoma

Screening



Cycle 5 Day 1

Follicular Lymphoma	Complete Metabolic Response and Tumor Reduction
Patient	72-year-old Male with Follicular Lymphoma : Received 450mg BID Luxeptinib
Prior Therapies Failed	• revlimid+obinutuzumab, obinutuzumab, ublituximab, umbralisib
Response at C5D1	• **47% tumor reduction by SPD (PR requires 50%); 29% Reduction by SLD (PR requires 30%)** • **CMR (Complete Metabolic Response) by Cycle 3**

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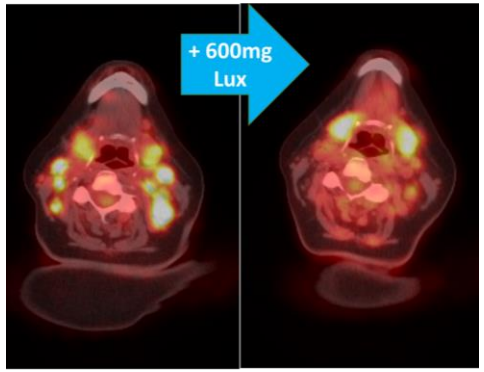
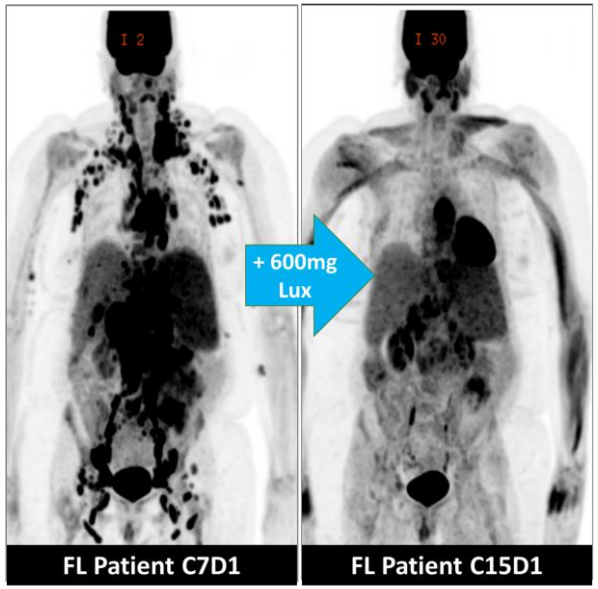
Luxeptinib Case Study: Dose-dependent Anti-tumor Activity in a Patient with Refractory Follicular Lymphoma



FL Patient C7D1
Neck

FL Patient C15D1
Neck



Follicular Lymphoma	Significant Tumor Reduction and Well Tolerated
Demographics	60-year-old female
Diagnosis at Study Entry	Grade 1 FL
Prior Therapies Failed	• bendamustine + obinutuzumab • rituximab
Dose	**450mg** BID 7 cycles, followed by **600mg** BID 8 cycles
Response	**Tumor growth continued, though slowed, while on 450mg BID through 7 cycles:** • SPD increased 28.2%, 10.7% and 8.7% at C3D1, C5D1 and C7D1, respectively, when compared with previous FDG PET-CT scan **43% tumor reduction from peak (12% below baseline) upon dose escalation to 600mg BID:** • Following dose escalation to 600mg in cycle 8, lesion growth arrested, followed by continuous reduction to below baseline • By C15D1, primary lesions shrank by 42.5% and 11.3% when compared with highest measurement (C7D1) and baseline (screening), respectively

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Luxeptinib: Ongoing Phase 1a/b Study in R/R AML and HR MDS

G3 capsules introduced into ongoing cohort 4

Cohort 4	900 mg Q12H	**Ongoing**
Cohort 3	750 mg Q12H	**Completed** ✓
Cohort 2	600 mg Q12H	**Completed** ✓
Cohort 1	450 mg Q12H	**Completed** ✓

PATIENT POPULATION

Relapsed or refractory AML and higher-risk MDS who **failed or are ineligible for / intolerant of** intensive chemotherapy or transplantation

- Patients failed by FLT3i, IDHi, venetoclax, chemotherapy
- Patients unfit for intensive therapy or failed by HSCT
- Patients with WT-FLT3 or mutated TP53 or RAS genes

Objectives

Ongoing Phase 1 a/b, open-label, single arm, multicenter, 3+3 dose-escalation clinical study (NCT04477291).

Primary objectives:

- Assess safety and tolerability of luxeptinib (CG-806)
- Determine maximum tolerated dose (MTD) and / or recommended Phase 2 dose (RP2D)

Key secondary objectives:

- Assess PK profile and PD activity
- Obtain preliminary evidence of antitumor activity

Dose Escalation Phase

- **Oral capsules administered twice daily** on a **28-day cycle**
- Planned expansion cohorts after dose escalation
- Additional patients may be enrolled (**back filling**) at dose levels previously declared safe
- **Intra-patient dose escalation** is allowed if higher dose is safe in 3 or more patients

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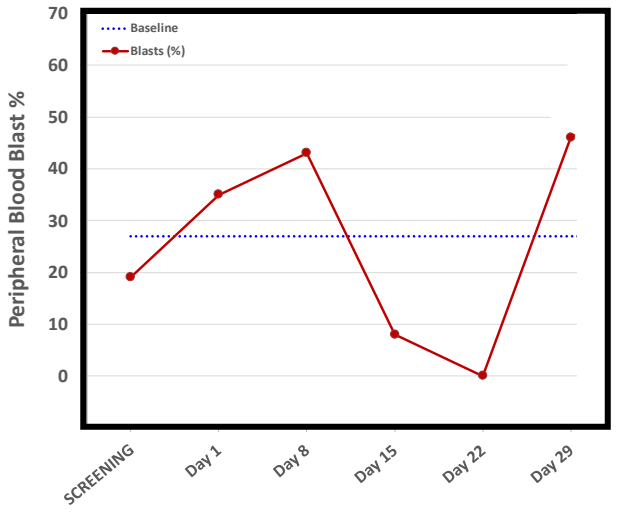
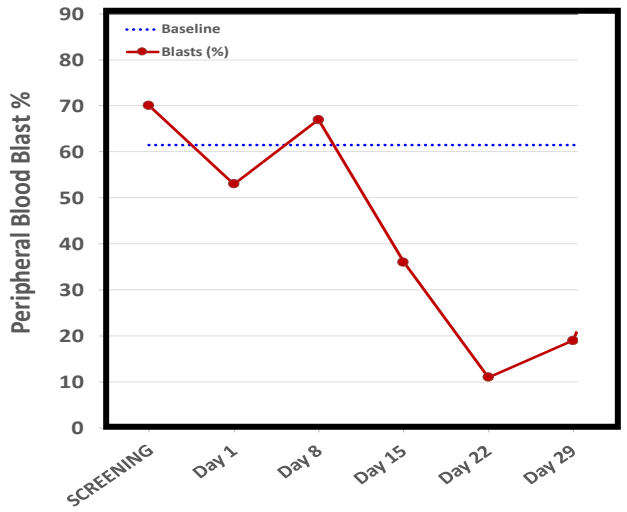
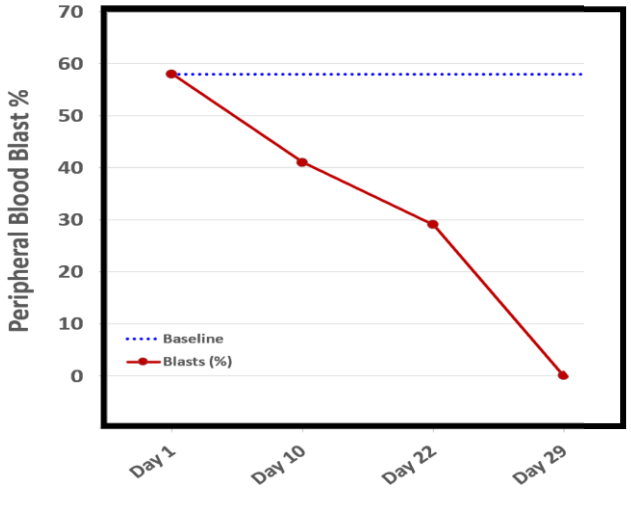
Luxeptinib Case Study: Examples of Blast Reductions in R/R FLT3-ITD+ AML Patients



- **36y, female, treated with Lux 450mg BID**
- **8 prior regimens** including FLT3 inhibitor gilteritinib and crenolanib, venetoclax and alloSCT
- Mutations detected at screening: FLT3-ITD, DNMT3A, NPM1, GATA2, WT1
- **90+% reduction of blasts** in Cycle 1, before disease progression in Cycle 2



- **64y, male, treated with Lux 750mg BID**
- **4 prior regimens** including azacitidine (for MDS), induction chemotherapy, Vyxeos, gilteritinib
- Mutations detected at screening: FLT3-ITD, GATA2, IDH2
- **80+% reduction of blasts** in Cycle 1, before disease progression in Cycle 2



- **61y, female, treated with Lux 750mg BID**
- **4 prior regimens** including induction and salvage chemotherapy, azacitidine, and venetoclax.
- Mutations detected at screening: FLT3-ITD, CBL, SRSF2, RUNX1, WT1
- **90+% reduction of blasts** in Cycle 1, before disease progression in Cycle 2

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Luxeptinib Case Study: Durable MRD-negative CR in FLT3+ Patient with high plasma exposure levels

FLT3-ITD+ R/R AML	CR / MRD-
Demographics	46-year-old male
Diagnosis at Study Entry	**FLT3-ITD+, relapsed de novo AML** with myeloid sarcoma (bone marrow & extra medullary disease)
Prior Therapies	• **Heavily pretreated, failed by chemotherapy / prior-FLT3i / 2 allogeneic transplants** • Induction chemotherapy, followed by salvage chemotherapy + **FLT3i** followed by **HSC** Transplant #1 • Following **HSC** relapse, treated with decitabine + venetoclax + **FLT3i** followed by 2nd **HSC** Transplant • Following 2nd **HSC** relapse & increased BM blast received focal radiation to perispinal mass
Dose	450mg BID luxeptinib
Response	• Abnormal bone marrow blast reduced to 0.6% on C2D1 and remained undetectable thereafter • Patient experienced **no myelosuppression** with blood counts sustained at normal levels • Highly sensitive flow cytometry detected **no abnormal blasts in bone marrow** at C4D1 and C5D3

MRD- CR: FLT3+ patient continues on study in Cycle 13

Luxeptinib: Improved G3 Formulation Entering Clinical Trials in 2022

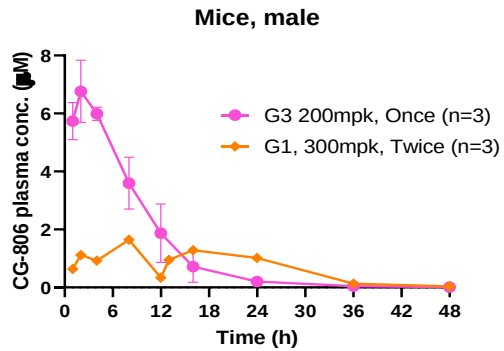
Luxeptinib 1st Generation (G1) Formulation

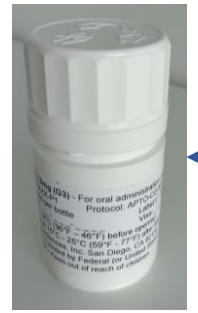
- Demonstrated safety/tolerability
- Clinically active in B-cell cancers
- AML patient MRD- CR with high plasma exposure
- Exposures increased incrementally from 450 to 750mg dose levels

Luxeptinib 3rd Generation (G3) Formulation

Goal to improve absorption reduce pill burden, reduce total API administered, and increase exposures

- G3 self emulsifying drug delivery system developed over 2 years
- PK of oral G3 superior to G1 in preclinical mouse, rat and dog
- G3 increased manufacturing throughput with smaller size capsules
- GMP manufacture, stability, release of first GMP batch complete
- Protocol amendments for AML and CLL/NHL patients complete
- Evaluate single dose PK and then transition to continuous dosing
- Exploring G3 in ongoing human trials





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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
- 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 Development in 2022

- Exploring 900mg and possibly higher doses with original formulation if PK data supports
- Exploring improved G3 formulation to lower pill burden and boost exposure
- Select optimal formulation and dose for monotherapy & drug combination studies

Aptose Biosciences (NASDAQ: APTO)

APTOSE

Hematology company focused on precision medicines

Experienced leadership with deep expertise in kinase inhibitors

Orphan hematology programs with broader oncology optionality

Rolling presentation of value-driving clinical findings through 2022

HM43239

Clinically validated *Myeloid Kinome Inhibitor (MKI)*

High value targets: SYK, FLT3, cKIT^{MUT}, JAK

Multiple complete responses (CR) in ongoing Phase 1/2 study of R/R AML

CR in diverse AML patients with NPM1^{MUT}, TP53^{MUT}, N/K-RAS^{MUT}, IDH^{MUT}, FLT3$^{ITD/TKD/WT}$

2022: Select dose(s) and initiate expansion trials as monotherapy and in combination

LUXEPTINIB (Lux, CG-806)

Dual *Lymphoid and Myeloid Kinome Inhibitor (LKI/MKI)*

High value targets: BTK, FLT3, CSF1R, PDGFRα, TRK, AURK

Ongoing parallel dose escalations in patients with B-NHL and AML/MDS

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

2022: Select optimal formulation and dose(s) for continued development

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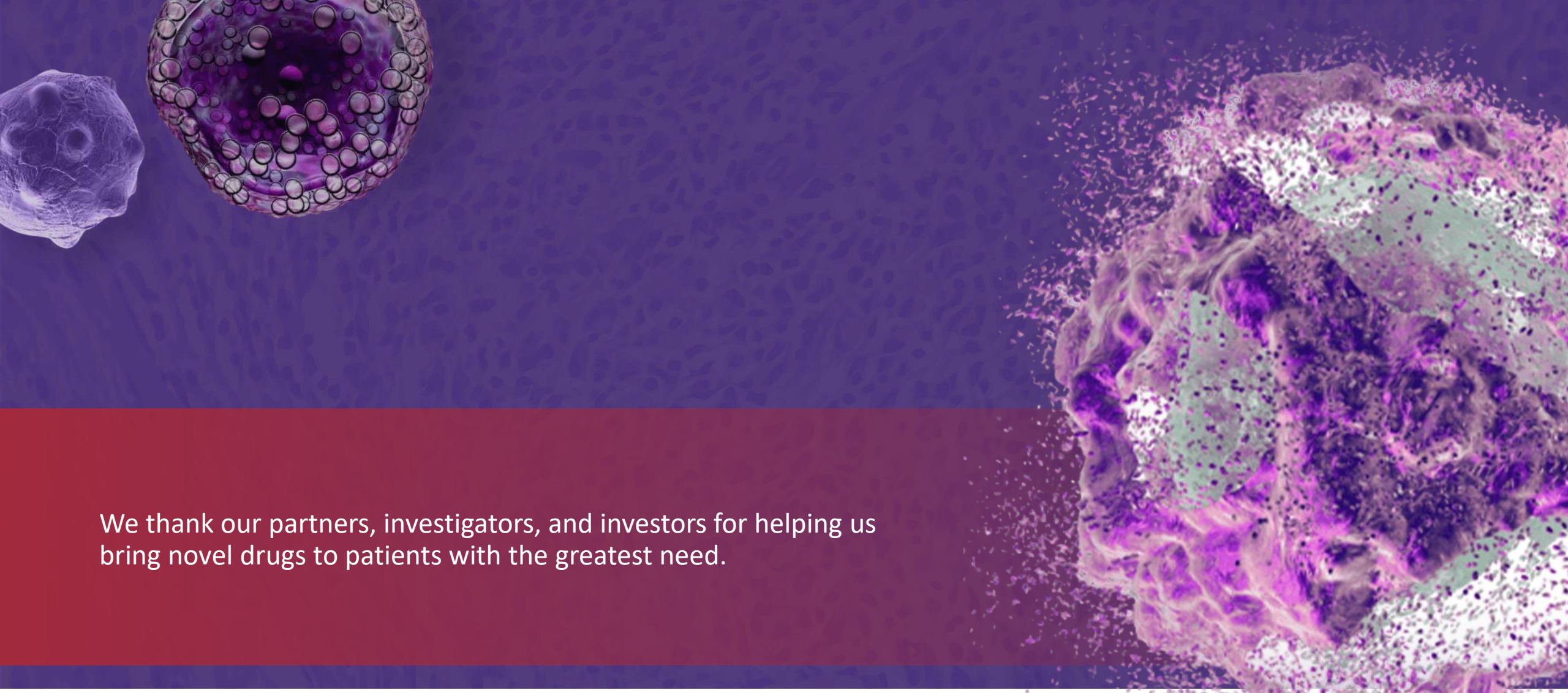

We thank our partners, investigators, and investors for helping us bring novel drugs to patients with the greatest need.

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