APTOSE Corporate Update Event

December 06, 2020

14:00 PDT / 17:00 EDT

APTOSE BIOSCIENCES

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.

Although the Company believes that the views reflected in these forward-looking statements are reasonable, such statements involve significant risks and uncertainties, and undue reliance should not be placed on such statements. Certain material factors or assumptions are applied in making these forward-looking statements, and actual results may differ materially from those statements. Those factors and risks include, but are not limited to, our ability to raise the funds necessary to continue our operations, changing market conditions, the successful and timely completion of our clinical studies including delays, the demonstration of safety and efficacy of our drug candidates, our ability to recruit patients, the establishment and maintenance of corporate alliances, the market potential of our product candidates, the impact of competitive products and pricing, new product development, changes in laws and regulations, uncertainties related to the regulatory approval process and other risks detailed from time to time in the Company's ongoing quarterly filings and annual reports.

Forward-looking statements contained in this document represent views only as of the date hereof and are presented for the purpose of assisting potential investors in understanding the Company's business, and may not be appropriate for other purposes. The Company does not undertake to update any forward-looking statements, whether written or oral, that may be made from time to time by or on its behalf, except as required under applicable securities legislation. Investors should read the Company's continuous disclosure documents available at www.sedar.com and EDGAR at www.sec.gov/edgar.shtml, especially the risk factors detailed therein.

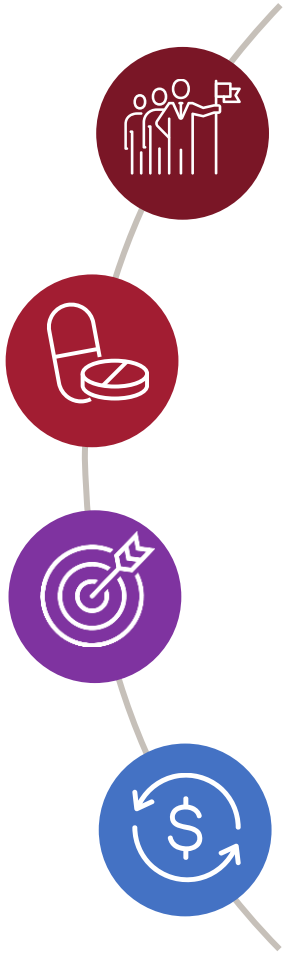
APTOSE BIOSCIENCES

Meeting Agenda

1. Welcome to Aptose Corporate Update Event & Introduction
2. APTO-253 AML/MDS Clinical Trial Update – Poster Yesterday
3. CG-806 CLL/NHL Clinical Trial Update – Poster Today
4. CG-806 AML Clinical Trial Update – First Update
5. Q&A Session

APTOSE BIOSCIENCES

Aptose Highlights



APTOSE

Clinical stage biotech company developing 1st-in-class, small molecule, targeted medicines
Precision treatment of hematologic malignancies; life-threatening / orphan diseases
Multiple assets addressing multiple cancers indications for optionality and value creation

CG-806
Oral FLT3 / BTK Kinase Inhibitor
FDA Orphan Drug Designation in AML

Precision to suppress multiple validated cancer targets, yet spares safety targets
Inhibits all forms of BTK → Phase 1a/b trial ongoing with CLL & NHL patients
Inhibits all forms of FLT3 → Phase 1a/b trial ongoing with AML patients
Potential for the treatment of a broad range of hematologic malignancies

APTO-253
MYC Inhibitor
FDA Orphan Drug Designation in AML

Only clinical stage agent known to directly target G-Quadruplex of notable MYC oncogene
Phase 1b trial ongoing for AML & MDS demonstrating safety and MYC inhibition

Serving the Needs of Patients

Seek to demonstrate near-term clinical POC with AML and CLL
Seek to deliver new agents that serve broadly the needs of patients with CLL and AML
Seek to deliver agents that avoid the rapid emergence of drug resistance

APTOSE BIOSCIENCES

APTOSE BIOSCIENCES

Clinical Update: APTO-253

Phase 1a/b Study in AML & MDS

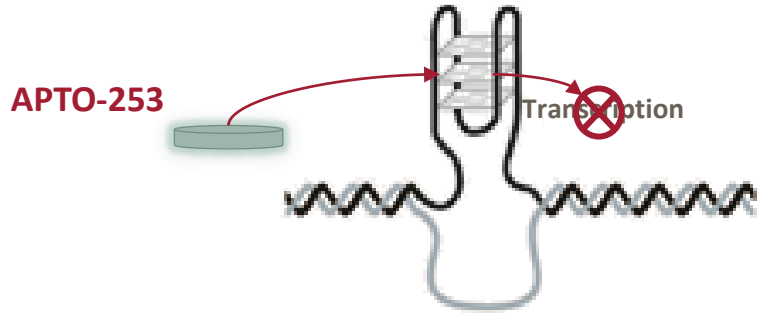
Significant Interest in Targeting MYC as a Cancer Treatment

MYC protein

MYC protein regulates multitude of key biological processes	Transcription factor alters thousands of genes
Dysregulated in >50% of all human cancers	Reprograms signaling pathways to support survival of cells
Direct targeting of MYC *protein* is challenging	Generally considered "undruggable" – no active site

APTO-253 Targets MYC Gene

APTO-253 Targets DNA regulatory motif in promoter of MYC gene	NOT the MYC protein
APTO-253 Inhibits MYC gene expression (mRNA)	Depletes cells of MYC protein → induces apoptosis



- APTO-253 inhibits MYC expression
- Causes induction of p21
- Triggers cell cycle arrest/apoptosis

APTOSE BIOSCIENCES

APTO-253 Phase 1b Dose Escalating Trial in Patients with AML or hr-MDS: Patients Receive Weekly IV Administration on 28D Cycles

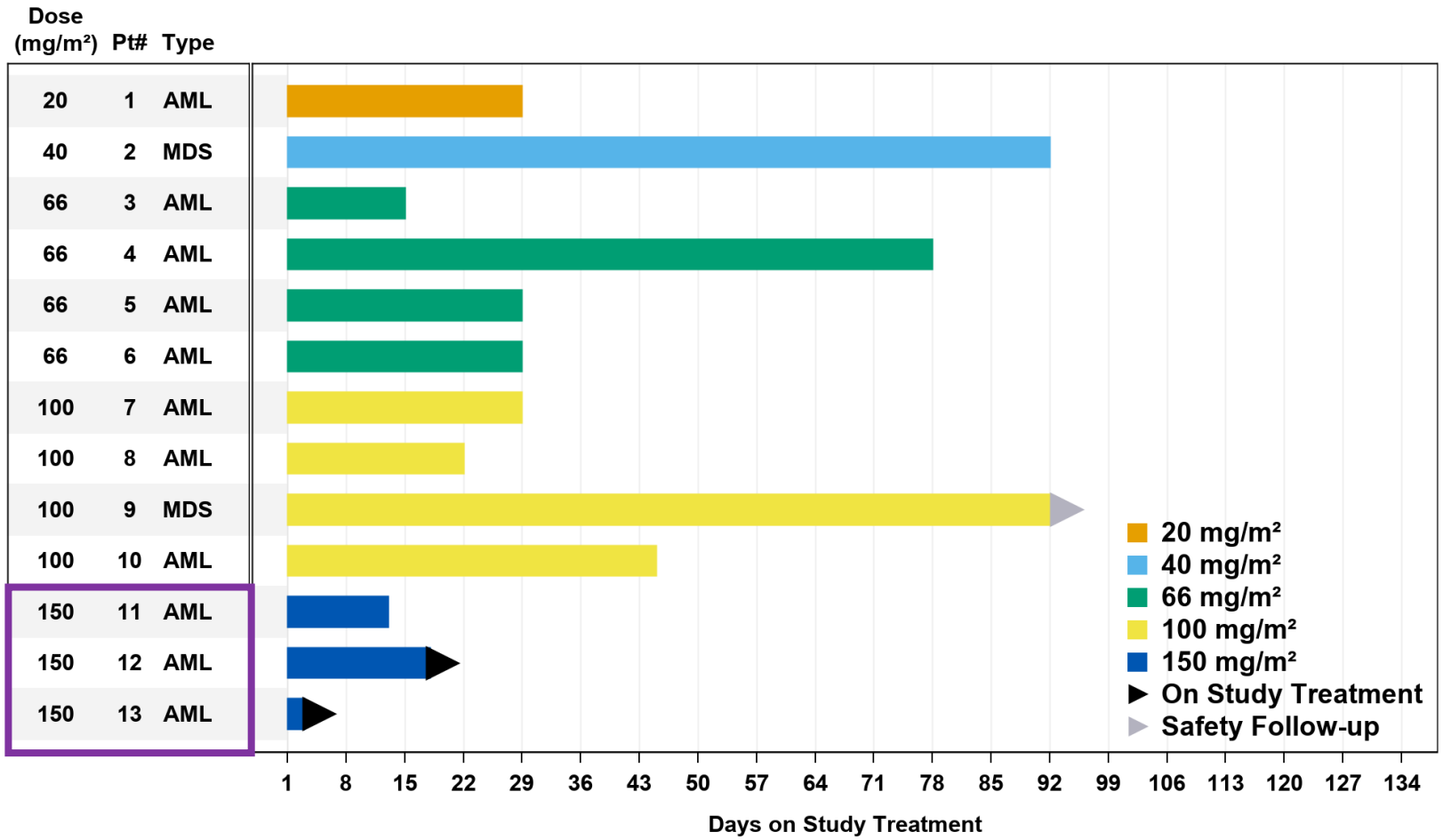
Dose Level	Dose	Status	Patients
1	20 mg/m^2	✓ Completed	AML
2	40 mg/m^2	✓ Completed	MDS
3	66 mg/m^2	✓ Completed	AML
4	100 mg/m^2	✓ Completed	AML & MDS
5	150 mg/m^2	Ongoing	AML to Date
6	210 mg/m^2	Planned	

To date, safe and well-tolerated

Now enrolling at 150mg/m^2 dose level 5

APTOSE BIOSCIENCES

APTO-253 has been Administrated to Five Cohorts Over Multiple Cycles



APTOSE BIOSCIENCES

APTO-253 Safety and Tolerability Profile

APTO-253 Generally Well Tolerated

- Only 1 TEAE of grade 3 or greater (fatigue, considered possibly drug-related) has occurred as of Oct 5, 2020
- No DLT or APTO-253 related SAEs in patients treated at dose levels 1 to 4
- No safety trends of concern to date

Events	Cohorts 1 to 4 (N=10)
Any Treatment Emergent Adverse Events (TEAEs)	10 (100%)
Any TEAEs ≥ Grade 3	8 (80.0%)
Any APTO-253 Related TEAEs ≥ Grade 3	1 (10.0%)
TEAE Leading to Treatment Discontinuation	0 (0.0%)
TEAE Leading to Death	5 (50.0%)*
Any Serious Adverse Events	9 (90.0%)*

*Unrelated to APTO-253

APTO-253 Related Treatment Emergent Adverse Events		
Preferred Term	Cohorts 1 to 4 (N=10)	
	Any Grade, N (%)	Grade 3, N (%)*
Fatigue	2 (20.0%)	1 (10.0%)
Hyperuricaemia	2 (20.0%)	0
Alanine aminotransferase increased	1 (10.0%)	0
Aspartate aminotransferase increased	1 (10.0%)	0
Blood alkaline phosphatase increased	1 (10.0%)	0
Blood creatinine increased	1 (10.0%)	0
Decreased appetite	1 (10.0%)	0
Dizziness	1 (10.0%)	0
Haematoma	1 (10.0%)	0
Hypoalbuminaemia	1 (10.0%)	0
Hypocalcaemia	1 (10.0%)	0
Hypokalaemia	1 (10.0%)	0
Muscle spasms	1 (10.0%)	0
Pyrexia	1 (10.0%)	0
Thrombophlebitis	1 (10.0%)	0
Upper respiratory tract infection	1 (10.0%)	0

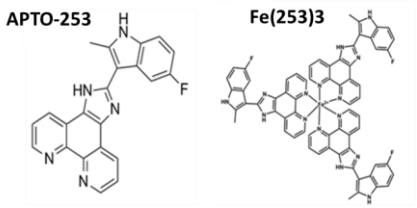
* No APTO-253 Related TEAEs ≥ Grade 4 as of Oct. 5, 2020

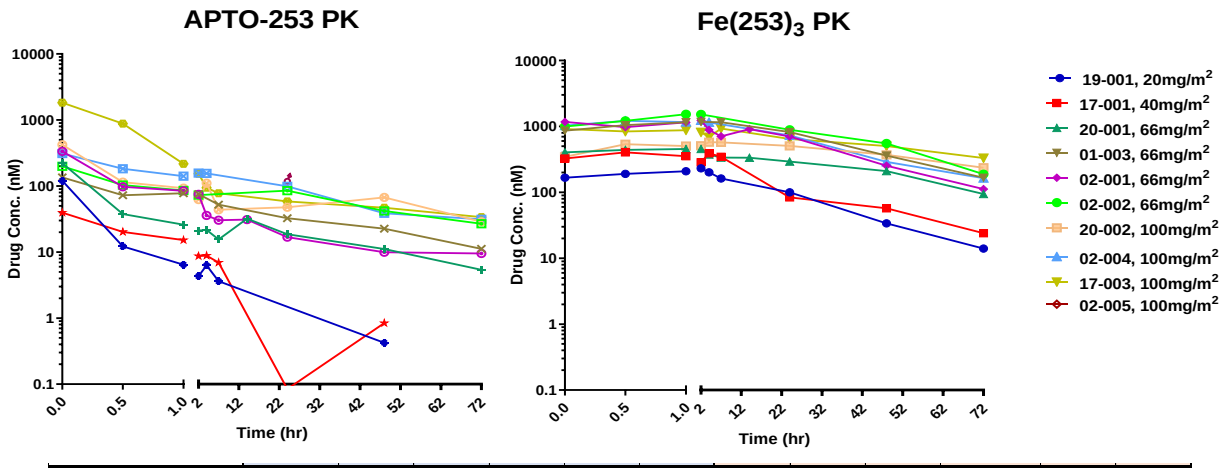
APTOSE BIOSCIENCES

Parent APTO-253 and Iron Adduct $Fe(253)_3$ are Active on MYC
PK Findings and PD (MYC Levels) Target Engagement after Dosing

APTO-253 and $Fe(253)_3$ Pharmacokinetics

Parent drug binds iron in plasma to form adduct and both are active on MYC gene
Adduct maintains 2-3µM plasma exposure for first 24hr & high levels through 72hr



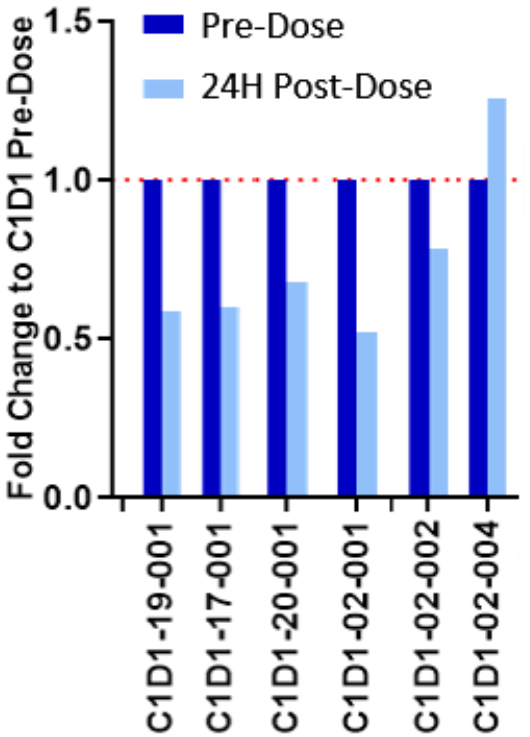


Note: 1. LC-MS/MS was performed at UCSD. The values of APTO-253 are not exactly as generated by ICON but are comparable.

2. GraphPad Prism software was used to analyze $AUC_{0\text{-}72h}$, C_{max} and T_{max}, which would not be the same values as analyzed by WinNolin software.

Drives MYC Reduction at 24hr on C1D1

MYC suppression in total PBMC fraction at 24hr with most patients



qRT-PCR using mRNA isolated from the whole blood cells

APTOSE BIOSCIENCES

Key Takeaways from APTO-253 Phase 1a/b Trial in Patients with R/R AML and High-risk MDS

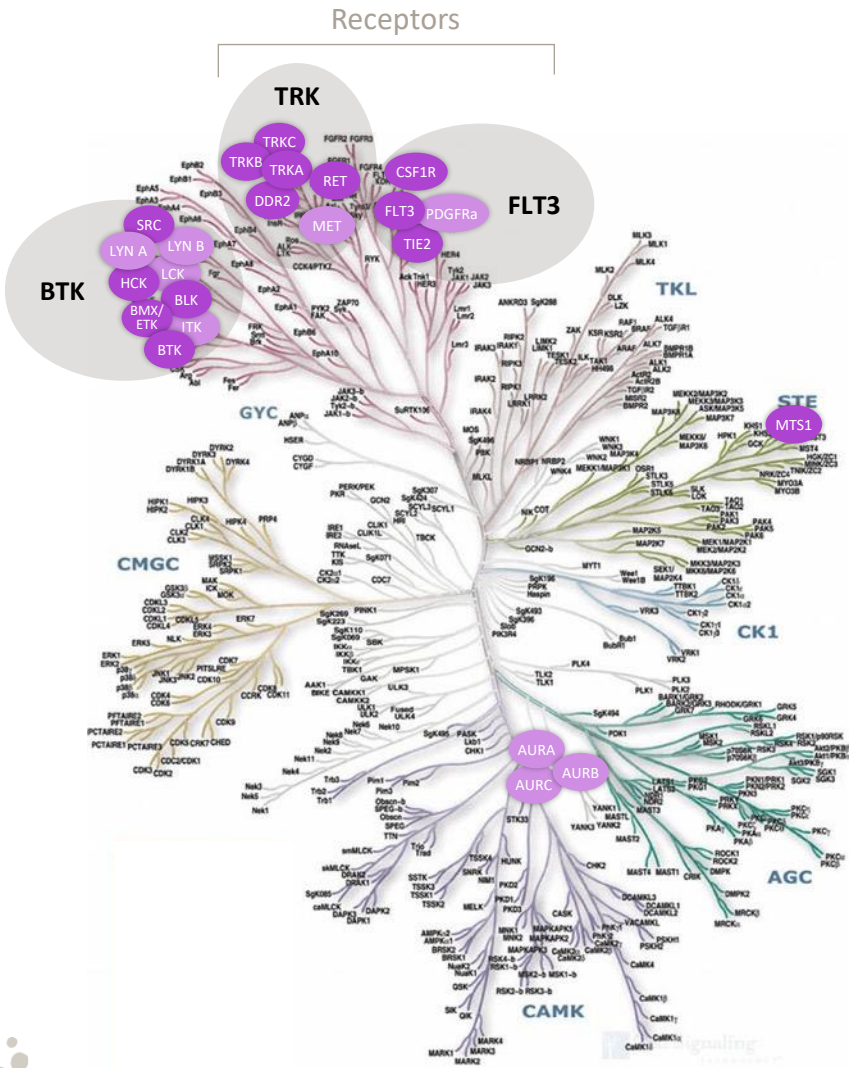
- **APTO-253 Targets the MYC Gene to Treat AML & hr-MDS Patients Failing Standard Therapies**
- **Favorable Safety and Tolerability Profiles to Date**
- **Encouraging Pharmacokinetic Profile of Parent Drug and the Fe(253)3 Complex**
  - Dose-related exposures, with sustained Fe(253)3 complex in the 2-3µM range at the dose of 100mg/m^2
- **Pharmacodynamic Profile Shows MYC Suppression in PBMC 24hr After Dosing**
  - Developed methodologies to assess tumor cell associated MYC levels by flow cytometry
- ***New Research Heightens Interest in Development of a Small Molecule Suppressor of MYC Oncogene***
  - MYC activation modulates genetic programming to drive cancer proliferation in hematologic and solid tumors
  - Recent report demonstrates that *even transient reductions of MYC expression sensitize tumors* and microenvironment to treatment with chemotherapies [Sodir et. al., *Cancer Discov* 2020; 10: 588-607]
- **Believe APTO-253 Program can Bring Value → Plan to Continue Dose Escalation and Seek Responses**

APTOSE BIOSCIENCES

Clinical Update: CG-806, Part 1

Phase 1a/b Study in CLL & NHL

CG-806 is an “Oral, Cluster-Selective Kinase Inhibitor” Potently and Selectively Inhibits Clusters of Related Kinases



Unique Kinome Targeting Profile

- Selectively inhibits clusters of kinases
- Only agent to potently inhibit the validated **BTK** and **FLT3** targets
- Only kinase inhibitor under development for 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
- NOT a “dirty” kinase inhibitor

APTOSE BIOSCIENCES

CG-806 Phase 1a/b Clinical Trial in Heavily Pretreated R/R CLL & NHL: Now Dosing Cohort 5 (750 mg BID)

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 CG-806
- Determine recommended Phase 2 dose (RP2D)

Key secondary objectives:

- Assess PK profile and PD activity
- Obtain preliminary evidence of antitumor activity
- Identify recommended starting dose for a separate study in patients with R/R AML

Dose Escalation Phase

- Patients administered **oral capsules, twice daily** on a **28-day cycle**
- Plan to perform 6 dose levels and expansion cohorts
- **Accelerated titration** design to **rapidly reach higher exposures**
- **Watch for leading indicators of activity as CG-806 progresses through dose escalation and increasing plasma exposures**

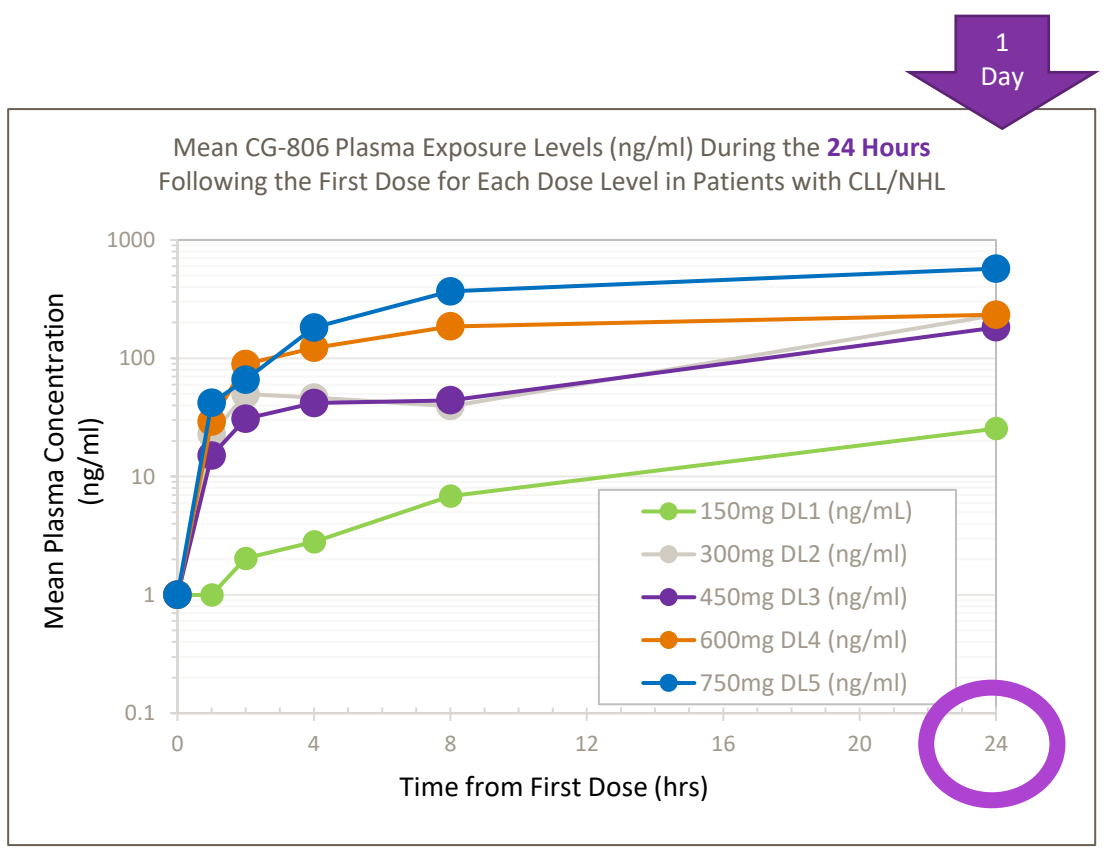
Patient Population

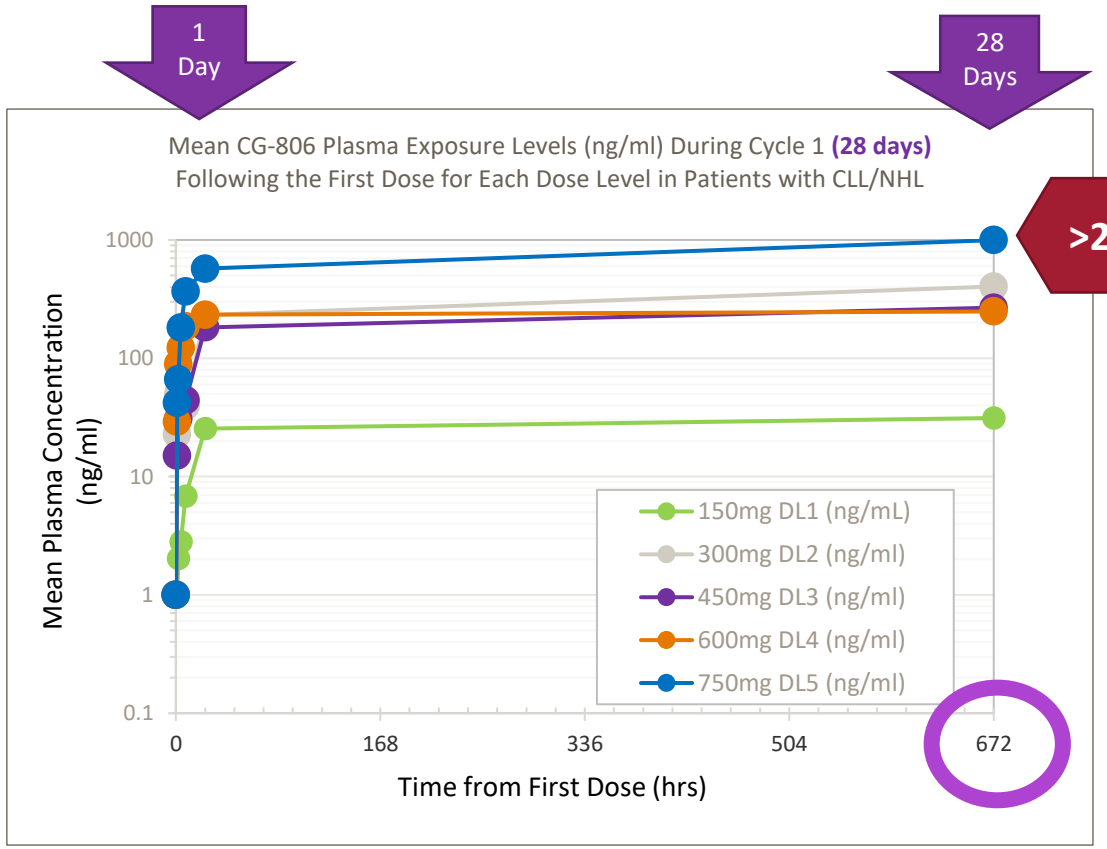
Relapsed or refractory CLL/SLL & NHL who **failed or are intolerant** to 2 or more lines of established therapy, or for whom no other treatment options are available

Cohort	Dose	Status
1	150 mg BID	Completed
2	300 mg BID	Completed
3	450 mg BID	Completed
4	600 mg BID	Completed
5	750 mg BID	Ongoing
6	900 mg BID	Planned

APTOSE BIOSCIENCES

CG-806 Dose Related PK Profile in Patients with CLL/NHL Malignancies: Continued Increases in Steady State Plasma Exposures as Increase Dose Levels

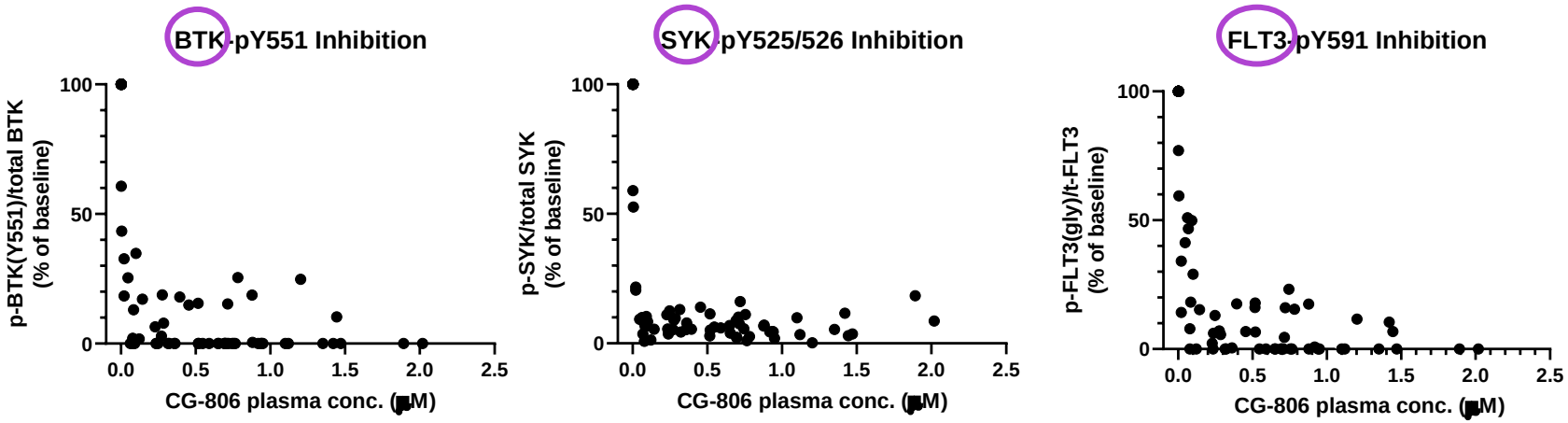




- CG-806 achieved dose related pharmacokinetics during the first 24 hours after initiation of dosing and the end of Cycle 1
- CG-806 achieved a steady state plasma concentration >2µM at the end of Cycle 1 at the dose of 750mg BID

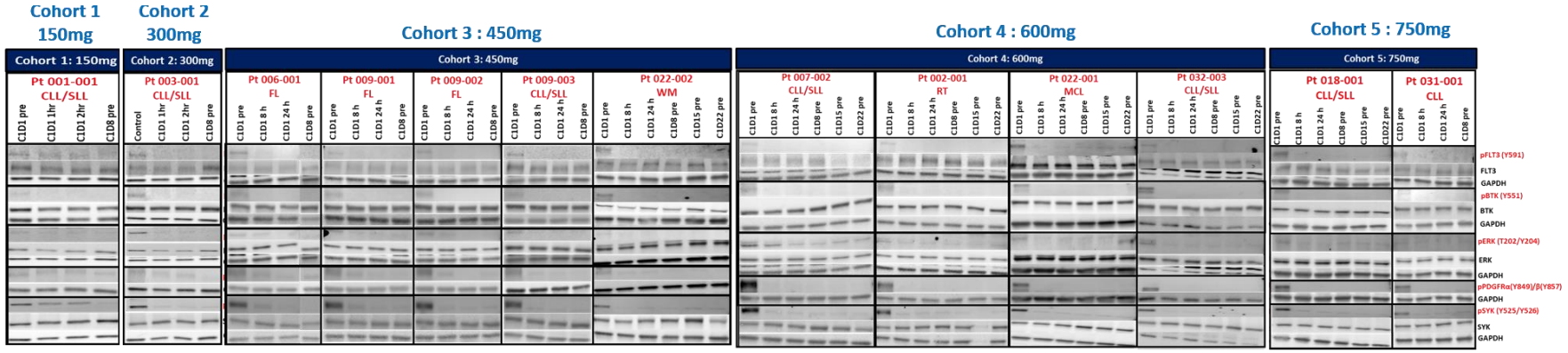
APTOSE BIOSCIENCES

CG-806 Pharmacodynamic Inhibition on Target Kinases BTK, FLT3, ERK, PDGFRα and SYK as demonstrated by a Plasma Inhibitory Activity (PIA) Assay



Plasma collected from patients in Cohorts 1 – 5 (n=13) were used in PIA assay:

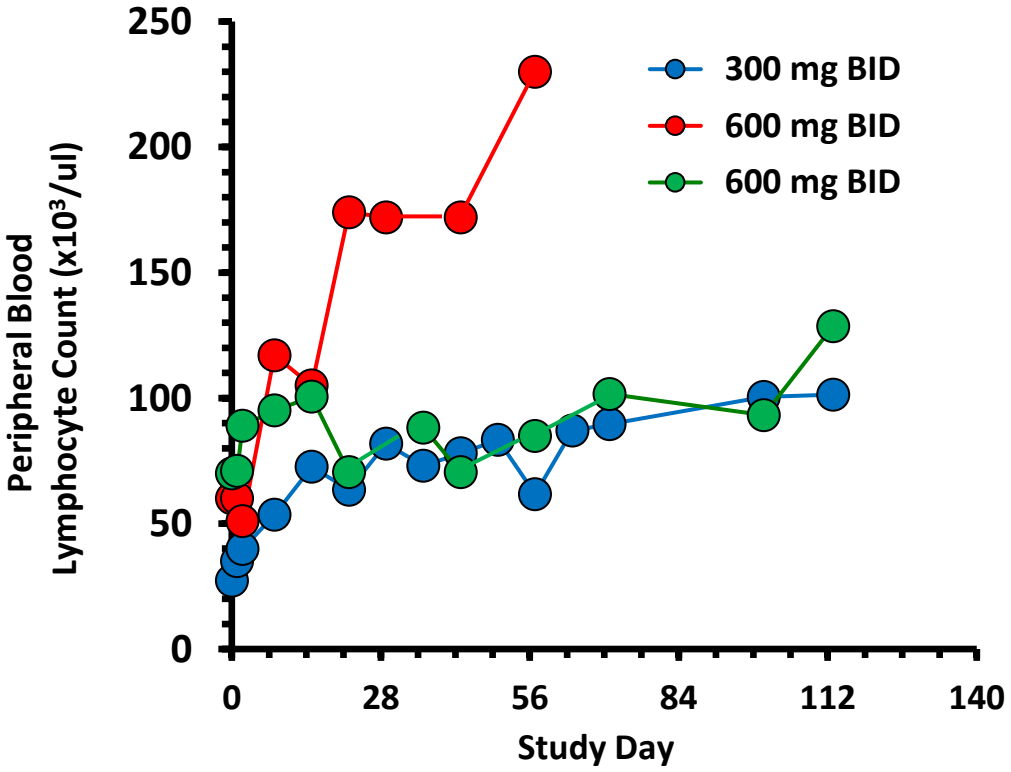
- EOL-1 cells express many of the kinases targeted by CG-806 and are used as a reporter cell line. Cells were treated for 6 hours with plasma collected from patients at the indicated timepoints and then subjected to whole cell lysis and immunoblotting.
- Densitometry analysis determines kinase activity as a function of dose



APTOSE BIOSCIENCES

CG-806 Demonstrates Pharmacologic On-Target Lymphocytosis and Modest Reductions in Tumor Size in CLL/SLL Patients

On-Target Lymphocytosis in CLL/SLL Patients



Treatment Case Studies in Different B-Cell Malignancies

Data-cut date: 2 Nov 2020

Case A: Patient with **follicular lymphoma**, who received 2 prior regimens (bendamustine/obinutuzumab, rituximab), received CG-806 at 450mg BID for 7 cycles, and was then dose-escalated to 600mg BID.

- After 5 weeks at 600mg, scan showed cessation of tumor growth.
- After 13 weeks at 600mg, scan showed an actual reduction in tumor size compared to the prior scan, but not to the level of a PR.

Case B: Patient with **SLL**, who received 3 prior regimens (bendamustine / rituximab, idelalisib / rituximab, lenalidomide / obinutuzumab), was treated with CG-806.

- After 8 weeks at 450mg BID, patient showed a 10% tumor reduction.
- The patient left study prior to Data-cut date.

Case C: Patient with 17p-deleted **CLL**, who received 4 prior lines of treatment (including veltuzumab, alemtuzumab, and ibrutinib), received CG-806 at 600mg BID.

- Immediately, on-target lymphocytosis was observed.
- After 8 weeks, scan showed a 27% tumor reduction.
- At 16 weeks, the tumor size remains below baseline.
- Since then, the patient discontinued treatment.

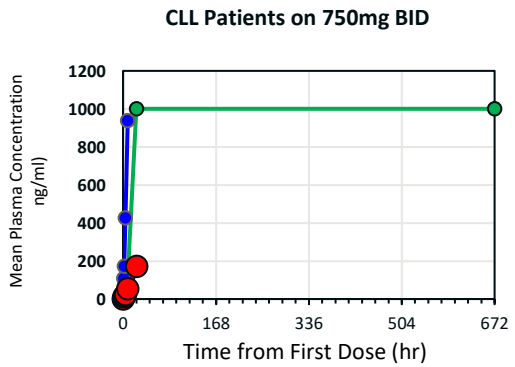
APTOSE BIOSCIENCES

CG-806 Dose Level 5 (750mg) with CLL Patients

- **One CLL Patient Completed 28d Cycle 1 : >2uM Steady State Plasma Level (green spheres)**
- **Two Rapidly Progressing CLL Patients Did Not Complete Even One Week of Dosing**

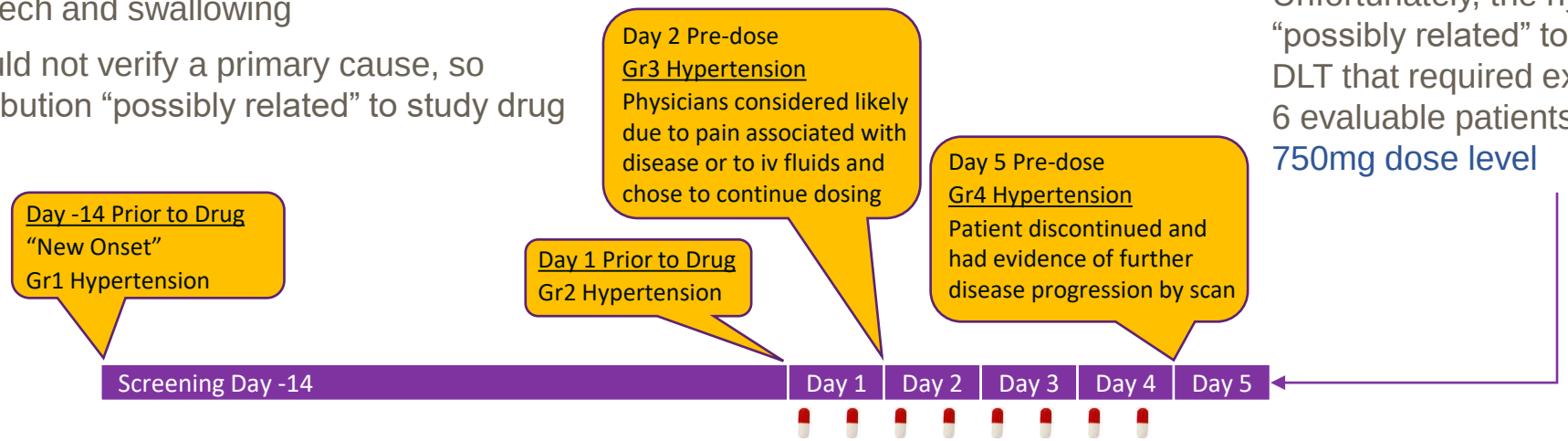
1st Patient Experienced Grade 2 Dysarthria

- PK approaching 2µM at 24hr (blue spheres)
- Puzzling: No evidence of stroke, vasculitis, or local nerve injury elsewhere
- No neurologic deficits in any other patients
- Patient history of lymphadenopathy in the neck adjacent to cranial nerves controlling speech and swallowing
- Could not verify a primary cause, so attribution "possibly related" to study drug



2nd Patient Experienced Grade 3-4 Hypertension

- Exposure (red spheres) was low relative to other patients receiving 750 mg and less than many patients on lower dose levels
- Analysis of all patients identified no correlative effect on blood pressure with exposure levels at any dose level or at any time
- Unfortunately, the hypertension was scored as "possibly related" to study drug and therefore a DLT that required expansion of the dose level to 6 evaluable patients – expansion underway at the 750mg dose level



APTOSE BIOSCIENCES

CG-806 Phase 1a/b Targeting CLL Patients at 750mg Dose Level

- **Targeting an Expanding CLL Population with a Distinct Phenotype**
  - Failed chemotherapy, anti-CD20 antibodies, venetoclax, covalent BTKi, and non-covalent BTKi
  - Likely have mutations in RAS/ERK/MAPK pathways and p53 pathways = Non-BTK escape mechanisms
  - Difficult to treat, but in need of a drug like CG-806 targeting BTK plus multiple pathways simultaneously
  - Deep R/R patients being treated with CG-806 today are more refractory than patients treated by other drugs

- **When Can Responses be Expect with CG-806 in These Patients?**
  - History teaches that non-covalent BTK inhibitors typically deliver PRs (50% tumor reductions) in R/R-CLL patients once a threshold plasma exposure is reached
  - Prior to that threshold plasma exposure, the leading indicators of drug activity evolve sequentially and include:
    1. Inhibition of BTK
    2. On-target lymphocytosis
    3. Modest (<50%) tumor reductions
  - CG-806 delivered clinical leading indicators: BTK inhibition / lymphocytosis / modest tumor reductions
  - CG-806, as moves to higher dose levels, may be approaching the threshold exposures that drive formal responses

APTOSE BIOSCIENCES

CG-806 CLL/NHL Phase 1a/b Synopsis

- **Accelerated Enrollment : Since the Data-cut Date, Two Additional Patients Dosed and Two Additional Patient Consented**
- **Safety and Tolerability : Generally Well Tolerated & See No Toxicity Trends to Date that Would Prevent Escalation**
  - Two rapidly-progressing patients did not complete even one week of Dose Level 5
  - One patient scored as a DLT, leading to expansion of Cohort 5 to 6 patients – Data inconsistent with attribution to study drug
- **PK Findings : Plasma Exposure Levels Generally Increase as the Dose is Increased**
  - The CG-806 with 750mg Dose Level 5 sustained steady state concentration (trough levels) > 2uM range
- **Pharmacodynamic and Pharmacologic Findings : Consistent with BTK inhibition**
  - Plasma of all patients receiving >300 mg BID demonstrated robust inhibition of key phospho-kinases
  - All classic CLL patients experienced on-target lymphocytosis
- **Scans of Tumor Measurements : Modest Tumor Reductions but No Formal Responses to Date**
  - *Three* patients experienced modest tumor reductions (max 27%) but not to the level of a PR (50% decrease)
- **All Leading Indicators of Activity are Trending Toward Responses with the Right Dose in the Right Patients**
- **Plan to Continue Dose Escalation to Deliver as much Drug as Possible and Safely to Treat Deep R/R-Patients**

APTOSE BIOSCIENCES

Clinical Update: CG-806, Part 2

Phase 1a/b Study in AML

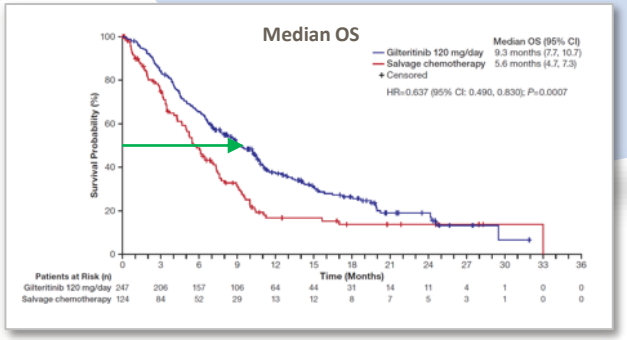
CG-806 belongs to a New Class of Drugs: Only Agent to Inhibit BTK for CLL *and* FLT3 for AML

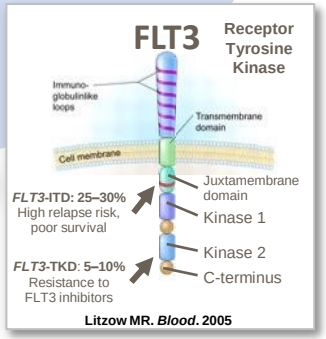
AML: Highly Aggressive and Deadly Cancer of Blood/Bone Marrow

- ~19,940 diagnosed this year ~11,180 deaths this year[1]
- The 5-year survival rate for AML patients is ~25%
- **R/R-AML median OS is under 6 months**

Limitations of Current FLT3 Inhibitors

- **FLT3-ITD mutation** is key driver in **25-35% of AML patients**[2,3]
- Current "Dirty" agents (Midostaurin, etc.) are limited → **Toxicity**
- Current "Selective" agents (Gilteritinib) less durable → **Resistance**
- Current agents **susceptible** to mutations in TP53, Ras, FLT3





Desperate Need for Improved FLT3i → CG-806

- CG-806 potently inhibits *all* WT & mutant forms of FLT3
- CG-806 suppresses multiple oncogenic signaling pathways to avoid resistance
- CG-806 combines effectively with other therapies, including venetoclax

(1) ACS Cancer Facts & Figures diagnosed with AML in US; : [2] *Cancer*. 2014 July 15; 120(14): 2142-2149 : [3] *Blood* 2016;128(5);686-698.

APTOSE BIOSCIENCES

CG-806 Inhibits All Forms of FLT3 & Kills Cells with FLT3-D835Y Mutation More Potently than Other FLT3 Inhibitors

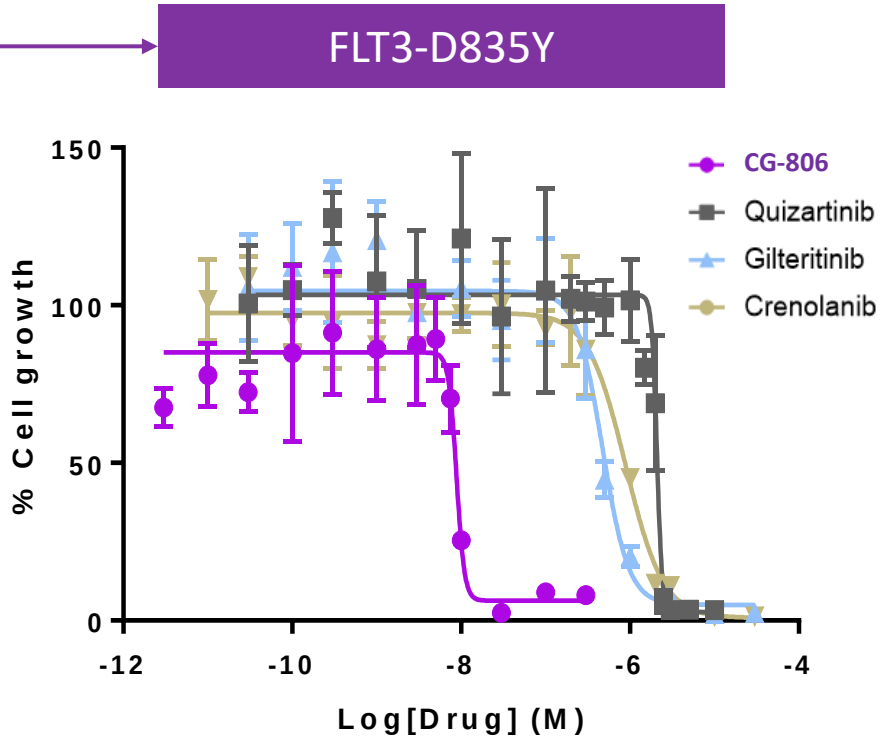
CG-806 Superior to Other FLT3-ITD Inhibitors

Drug	IC_{50} (nM)
CG-806[1]	0.8
Quizartinib[2]	8.8
Gilteritinib[3]	0.9
Crenolanib[4]	2
Midostaurin[2]	11
Nexavar[2]	79
Sutent[2]	1

CG-806 Potent (Kd) FLT3 WT/Mutants

FLT3 Proteins (Fragments)	CG-806 Kd (nM)
FLT3 WT	0.24
FLT3 ITD	3.1
FLT3 D835Y	**4.2**
D835H	2.2
D835V	7.9
R834Q	6.4
N841I	0.8
K663Q	0.55
ITD / F691L	16

CG-806 Superior to Other FLT3 Inhibitors on AML Cells with FLT3-D835Y Mutation



(1) Ba/F3 isogenic cells kindly provided by Dr. Michael Andreeff at MDACC

1. Reaction Biology Corp. 2. Blood. 2009 Oct 1; 114(14): 2984–2992 3. J Clin Oncol 32:5s, 2014 (suppl; abstr 7070)
*4. Blood 2014 Jan 2; 123(1): 94-100 ; AACR Poster 2012 5. ASH Oral Presentation 2016. **N/A** – Data not available / Not Applicable.*

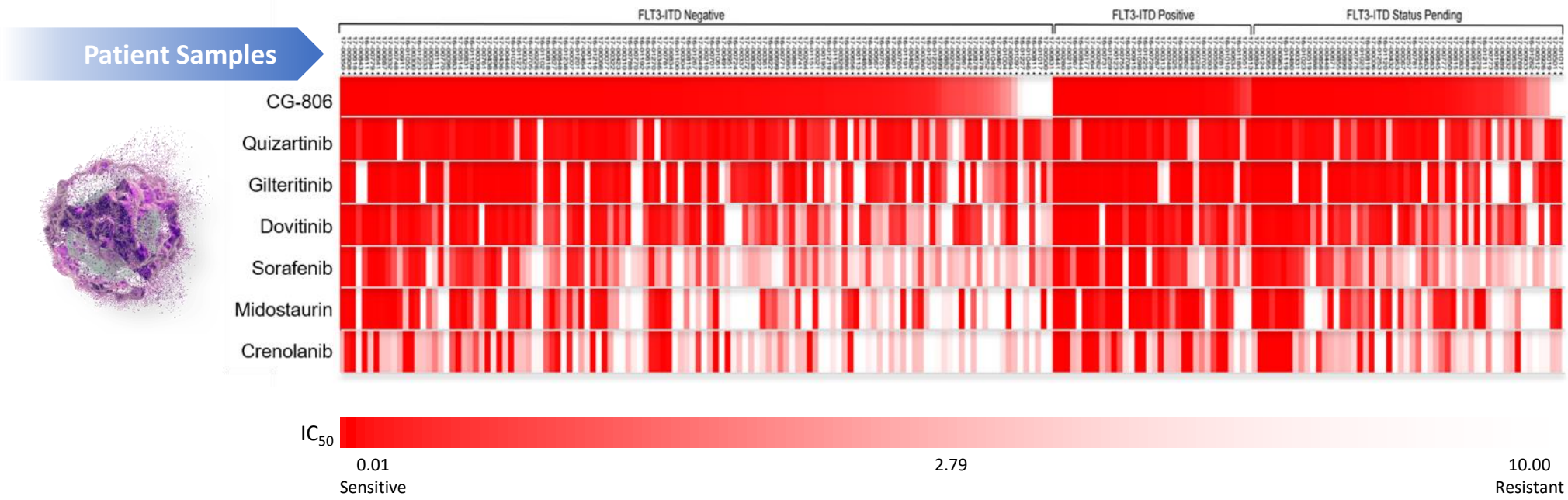
APTOSE BIOSCIENCES

CG-806 Exerts Broad & Superior Killing Potency Compared to Various FLT3i on AML Patient Samples

OHSU KNIGHT CANCER Institute | LEUKEMIA & LYMPHOMA SOCIETY® | hhmi Howard Hughes Medical Institute

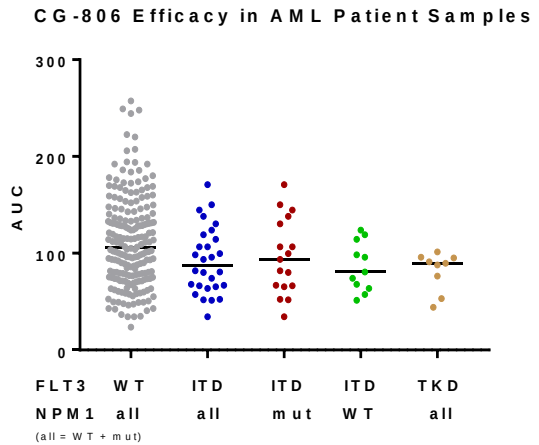
Measured IC_{50} of CG-806 and Other FLT3i's to Kill Ex Vivo Primary Cells from >200 AML Patients; IC_{50} transformed into a Heatmap of Sensitivity

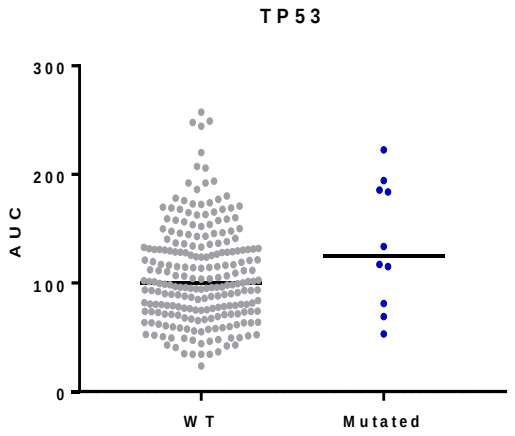
CG-806 greater potency in killing primary AML cells with wild-type FLT3 or FLT3-ITD

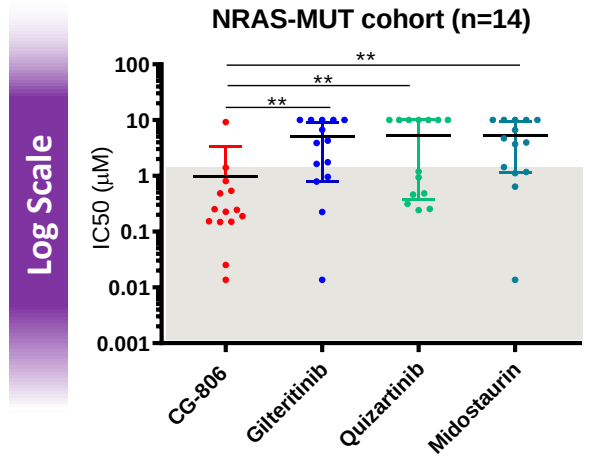


APTOSE BIOSCIENCES

Patient Sample Analyses Reveal High-Value AML Populations Sensitive to CG-806: NPM1, TP53, RAS Mutants







AML patient samples with…

- FLT3 mutations (ITD or TKD), with or without concurrent mutations of NPM1, are highly sensitive to CG-806
- mutated IDH1 are more sensitive to CG-806 relative to the IDH WT or IDH2 mutations ($p < 0.05$)

- TP53 WT and TP53 mutations equivalently sensitive to CG-806
- ASXL1 WT and ASXL1 mutations equivalently sensitive to CG-806
- NRAS WT and NRAS mutations equivalently sensitive to CG-806

Preclinical Profile Supports Developing CG-806 for the Treatment of AML

Strong Rationale to Develop for AML with High Potential Value

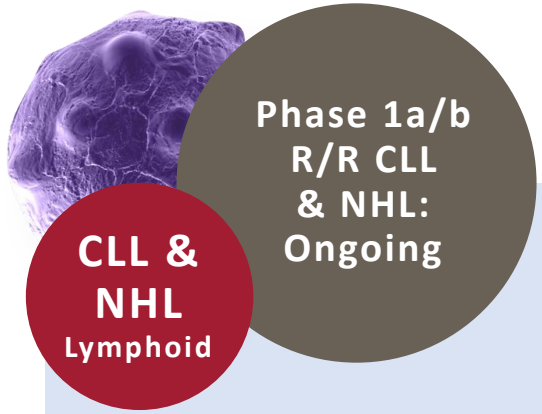
- Broadly potent against AML cells
  - Patients with mutated FLT3, TP53, IDH1, IDH2, SRF2, ASXL1 and RAS
  - Patients with WT-FLT3 (approximately 70% of R/R AML patients)
- More potent than other FLT3 inhibitors on >200 AML patient samples
- Delivers cures in xenograft models of human AML without toxicity

Phase 1: Include R/R AML Patients with Unmet Needs

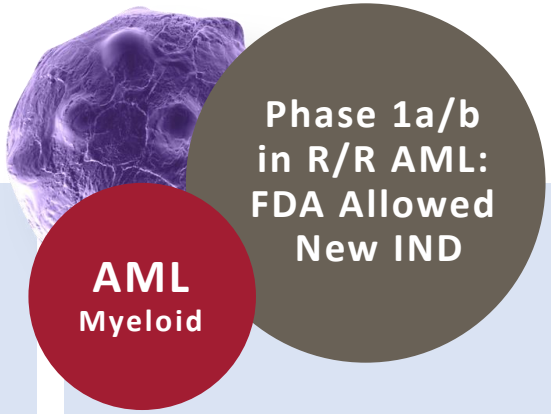
- Patients who failed other FLT3 inhibitors
- Patients who failed IDH-1 inhibitors
- Patients who failed venetoclax
- Patients with mutated p53, mutated RAS
- Patients with wild type-FLT3 (2/3 pts)
- Patients unfit for intensive therapies

- Initiating dosing with 450mg BID CG-806 as a potentially therapeutically active dose
- R/R-AML patient population is more refractory than the population treated with gilteritinib

CG-806 Phase 1 Clinical Development Plan for Patients with Lymphoid (CLL/NHL) and Myeloid (AML) Malignancies



- Dose escalation began at 150mg BID dose level 1 and currently treating at 750mg BID dose level 5
- Seek to define safety, tolerance, PK, PD and RP2D
- Seek to (1) inhibit phospho-BTK, (2) induce lymphocytosis, (3) observe responses in CLL patients



- Dose escalation began at 450mg BID as a dose likely to be active – Critically ill AML patients
- Continue escalation to identify an optimal dose
- Seek to define safety, tolerance, PK, PD and RP2D
- Seek to (1) inhibit phospho-FLT3, (2) decrease PB blasts and (3) observe responses in AML patients

APTOSE BIOSCIENCES

CG-806 Phase 1a/b Clinical Trial in AML: Now Dosing Cohort 1 (450mg BID)
AML and CLL Patients Show Equivalent PK Exposures with 450mg BID

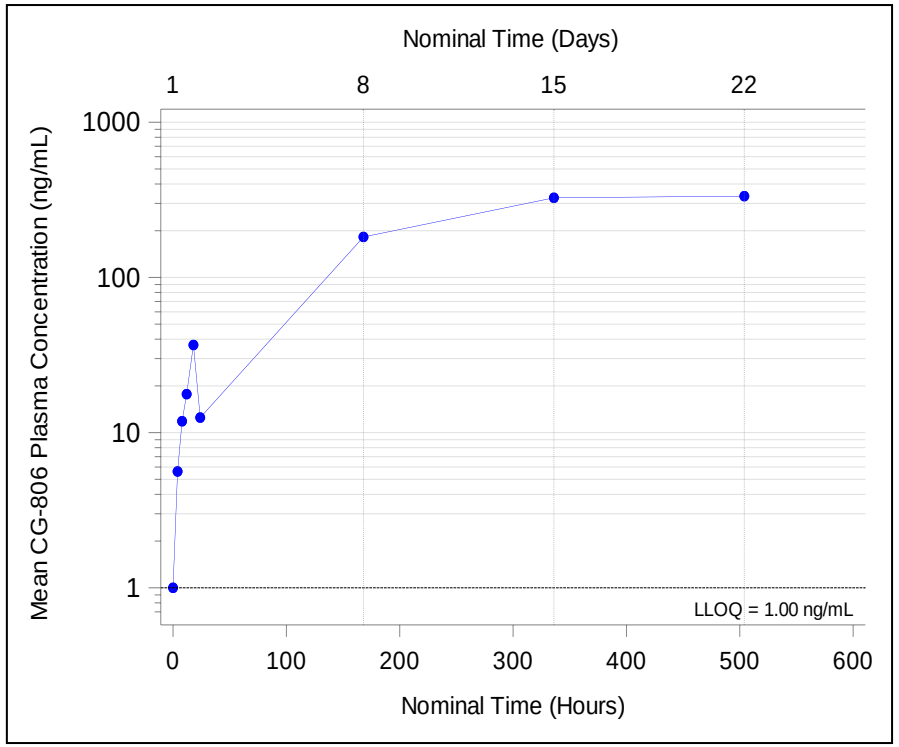
Cohort	Dose	Status
1	450 mg BID	Ongoing
2	600 mg BID	Planned
3	750 mg BID	Planned
4	900 mg BID	Planned

Trial has enrolled briskly: To date, 4 AML patients have been enrolled at the 450mg dose level

Patients receive a single dose on day 1 to collect single dose PK and BID thereafter

Intra-patient dose escalation allowed if higher dose is safe in 3 or more patients

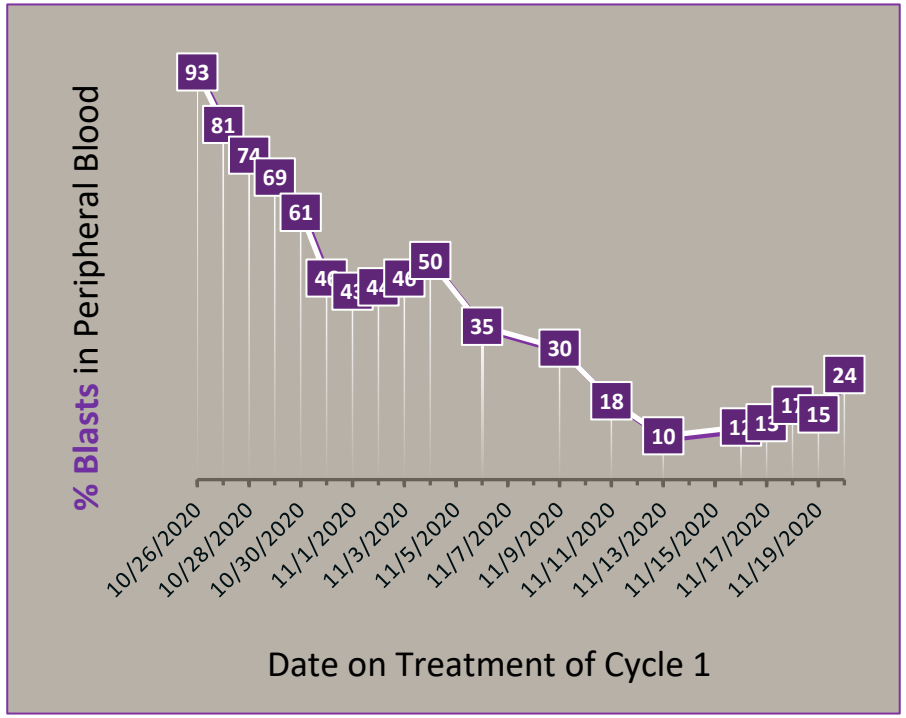
Mean CG-806 Plasma Exposure Levels (ng/ml) **After 21 days** of Cycle 1 Following the First Dose of **450mg** in 3 Patients with **AML**



APTOSE BIOSCIENCES

Case Study of Heavily Pretreated FLT3+ AML Patient on 450mg BID Demonstrates Clinical Anti-Leukemic Activity

- Heavily pretreated patient having relapsed AML
  - –Mutated NPM1, DNMT3A, and FLT-ITD
- Previously progressed after chemotherapy, alloSCT, and two FLT3 inhibitors (gilteritinib, crenolanib)
- During Cycle 1 of 450mg BID CG-806
  - –Observed Reduction in %Blasts from 93% to 10%
- Demonstrates clinical anti-leukemic activity
- Near end of Cycle 1, the %Blasts began to rise likely due to persistent AML clones not controlled by this dose of CG-806
- Hope higher optimal dose can clear persistent AML cells in future patients



APTOSE BIOSCIENCES

Key Takeaways from CG-806 Phase 1 Trial in AML Patients

- **Initiated dosing with 450mg BID in AML patients with FLT3+(ITD) and FLT3-WT**
- **Rapidly enrolled 4 patients on study drug : Additional patients are awaiting a slot**
- **PK data consistent with exposures observed with 450mg dose level in CLL/NHL patients**
- **Case Study of a FLT3+ AML patient**
  - Heavily-pretreated with chemotherapy, alloSCT, and two FLT3 inhibitors (gilteritinib FLT3i, crenolanib FLT3i)
  - Likely killing of subset of AML cells followed by out-growth of “persistent AML”
  - Data demonstrate anti-leukemic activity on AML cells at 450mg dose level
- **Plan to continue dose escalation to identify an optimal dose**
- **Additional sites to be initiated soon**

Key APTO Takeaway in Dec 2020: Programs Evolving with Attractive Value Proposition

Science Heavy Foundation

APTO-253 selective MYC repressor with preclinical and clinical mechanistic PoC

CG-806 kinase inhibitor is neither selective nor dirty, but covers *goldilocks* clusters of kinases that matter

Deep preclinical portfolio suggests **CG-806** is a different type of drug than any others in development in liquid and solid oncology

Steady Clinical Progress

APTO-253 AML/MDS study gaining momentum

CG-806 B-Cell study has now checked off BTK inhibition, lymphocytosis, PoC tumor reductions

CG-806 AML study showing blast reduction in high-bar R/R patient

Now plan to dose optimal patients @ optimal exposures and watch for formal responses

Attractive Value Proposition

Pureplay orphan hematology programs & further oncology optionality

Well funded to 2023, with no plans to finance

Growing base of sophisticated investors

Value-creating clinical updates from key studies poised for 1H2021

Focus: Push through the lower dose levels to get the right types of patients on the right dose!

APTOSE BIOSCIENCES

We thank you, our employees, the principal investigators, clinical site staff, and most importantly, our patients and their families for their participation in these clinical trials, and our colleagues at CrystalGenomics in South Korea.

APTOSE BIOSCIENCES

PRECISION ONCOLOGY FOR THERAPIES OF TOMORROW

APTOSE BIOSCIENCES

Q & A