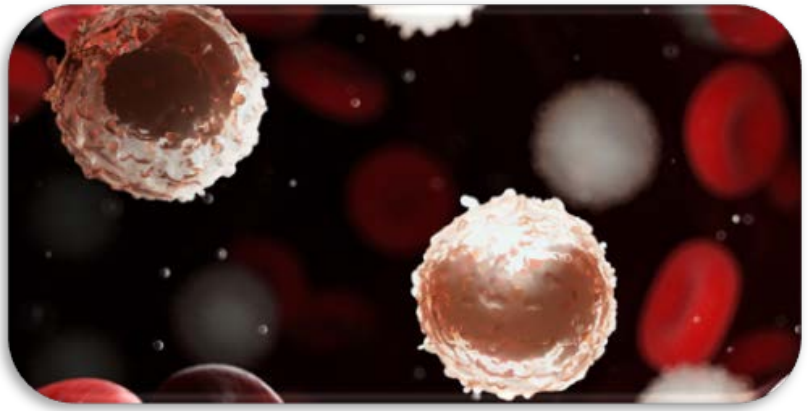
Biotech Showcase Presentation

January 2020

NASDAQ: APTO
TSX: APS

APTOSE BIOSCIENCES



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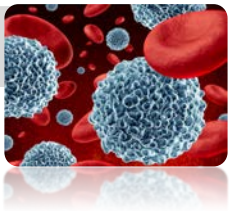
Investment Highlights

APTOSE



Strong leadership with expanded management team

Approximately 2 years of cash to advance clinical programs

Clinical stage biotech company developing 1st-in-class targeted agents

Treating hematologic malignancies; life-threatening / orphan diseases

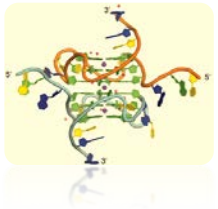
CG-806 Oral FLT3 / BTK Kinase Inhibitor

FDA Orphan Drug Designation in AML

Inhibits all forms of FLT3 and BTK : Drivers of AML, CLL & NHL hematologic cancers

Precision that suppresses multiple oncogenic pathways, yet spares safety targets

Phase 1a/b trial ongoing for CLL & NHL and Phase 1 is planned for AML & MDS



APTO-253 MYC Inhibitor

FDA Orphan Drug Designation in AML

Only clinical stage agent directly targeting G-Quadruplex of notable MYC oncogene

Phase 1b ongoing for AML & MDS demonstrating safety and MYC inhibition

Serving Patients and Market Opportunities

Potential to serve broadly CLL and AML patient needs : $1B+ commercial opportunity

Potential for rapid clinical POC and value creation with hematologic cancers

CG-806

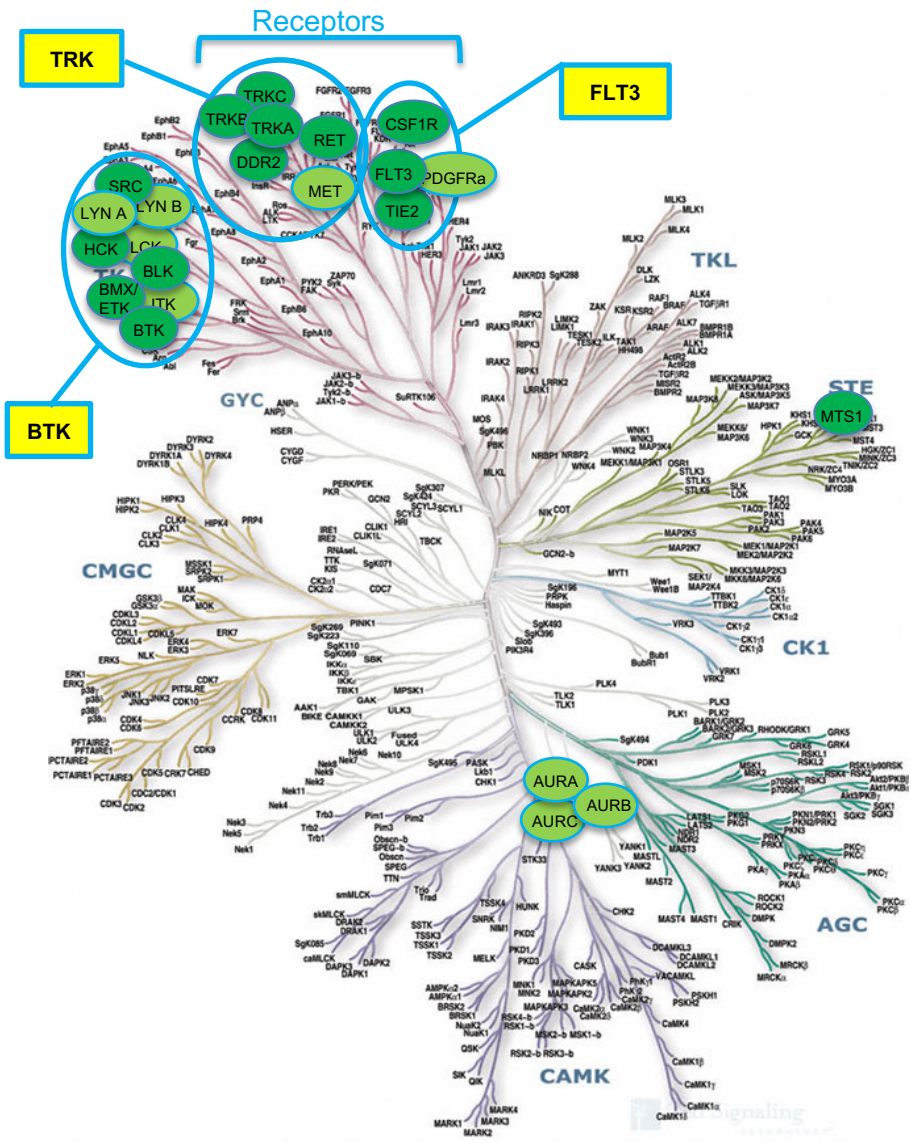
1st-in-Class

Oral FLT3 / BTK Inhibitor

Phase 1a/b Ongoing

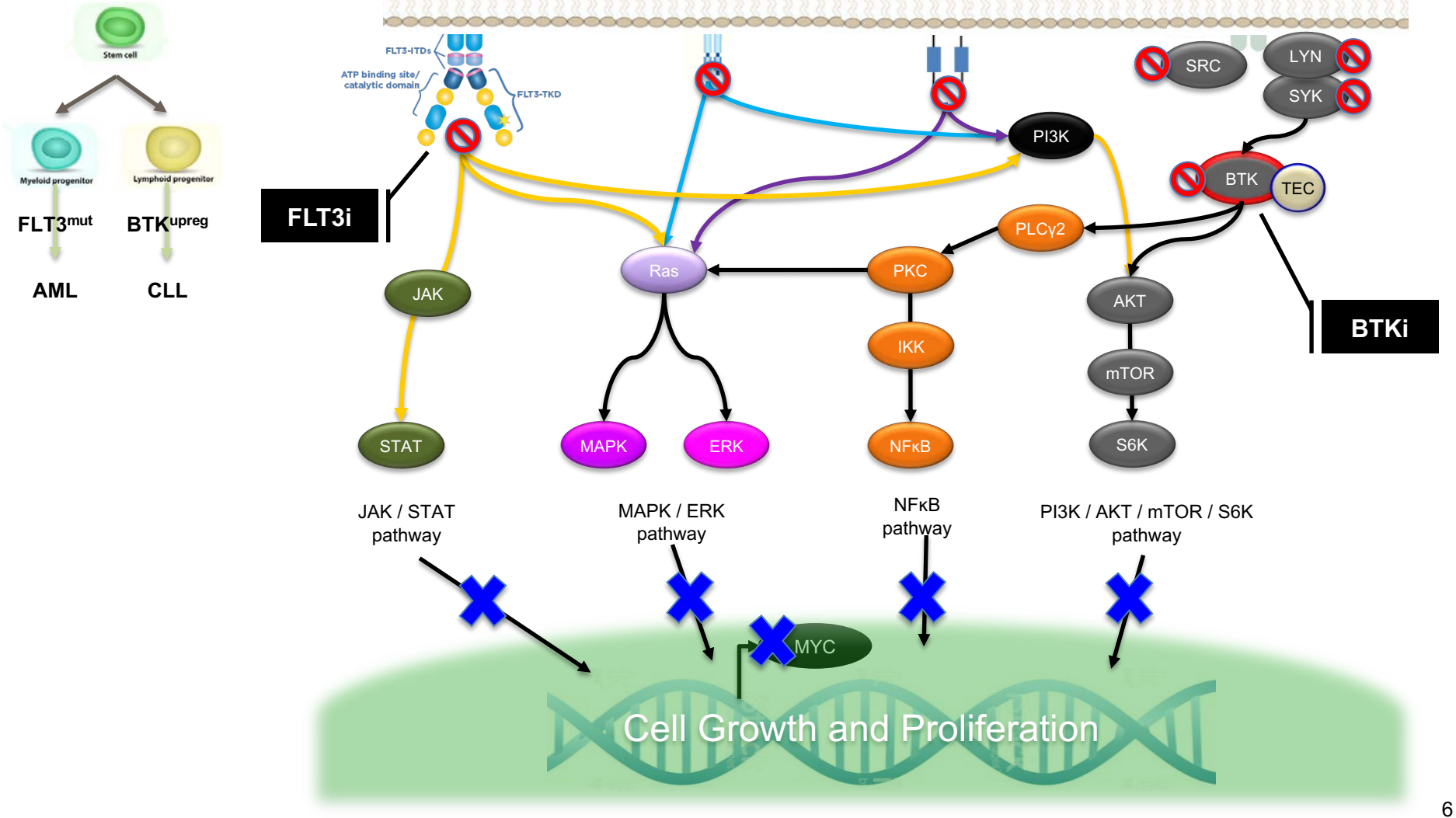
1. Non-covalent “reversible” inhibitor with unique kinome targeting profile
2. Potent inhibitor of all forms of BTK (WT / C481S) driver kinase
3. Potent inhibitor of all forms of FLT3 (WT / ITD or TKD mutated) driver kinase
4. Suppresses multiple signaling pathways essential for cancer cell survival
5. Precision spares safety targets & pathways associated with toxicity
6. Ongoing trial Ph1a/b for CLL & NHL B-cell malignancies
7. Planning trial Ph1a/b for AML/MDS myeloid malignancies

“Multi-Cluster Kinase Inhibitor”: CG-806 Potently and Selectively Inhibits Clusters of Related Kinases



- **Mutation Agnostic**
  - Inhibits all forms of FLT3
  - Inhibits all forms of BTK
  - Simultaneously suppresses multiple signaling pathways
- **Robust Safety Profile**
  - NOT a “dirty” kinase inhibitor
  - Avoids kinases that impact safety
  - No drug-related AEs seen to date
- **Inhibits Clusters of Kinases that Drive Hematologic Malignancies**
  - FLT3 cluster → AML & MDS
  - BTK cluster → CLL & NHL

CG-806 Suppresses Key Oncogenic Targets and Pathways in Myeloid & Lymphoid Malignancies



CG-806 for the Treatment of CLL / SLL / NHL

Overexpressed BTK (Bruton's Tyrosine Kinase)

- Drives B-cell cancers : CLL/SLL and NHL (FL, MCL, DLBCL, others)

Ibrutinib Covalent BTKi : SOC with >$6B Annual Sales

- Chemically targets Cys481 residue in the active site of BTK

Ibrutinib Shortcomings : Patients Discontinuing

- Over half (54%) CLL patients discontinue treatment by 44 months[1,2]
- Patients resistant (C481S mutant), intolerant or refractory to ibrutinib

Patients Failing a Host of Other Agents

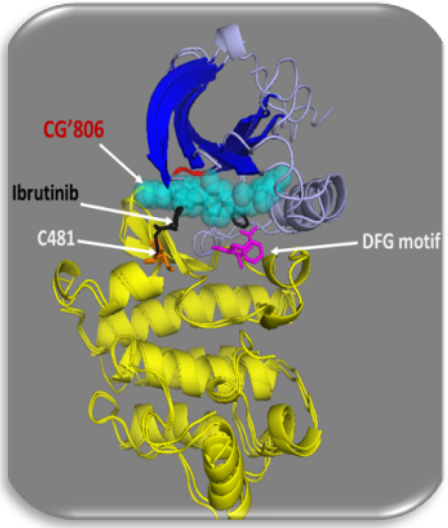
- Covalent BTKi, Non-covalent BTKi, BCL-2i, PI3Ki, Anti-CD20 Abs

CG-806 May Overcome Shortcomings of Ibrutinib & Other Agents

- "Non-covalent" : Retains activity against WT and C481S-BTK enzyme
- Well tolerated and inhibits multiple "oncogenic rescue" pathways
- Potently and directly kills CLL and other B-cell cancer cells

(1) Byrd et al. *Blood* ; 2019: 133(19): 2031-2042 | (2) Woyach et al. *J Clin Oncol*..; 2017: 35; 1-7

CG-806 Non-Covalent Inhibitor Retains Potency Against Wildtype and C481S-BTK



CG-806 Binds Non-Covalently and Productively to BTK

X-ray Crystallographic Analysis:

- Reversibly binds to WT-BTK and C481S-BTK Active Sites
- Atypical Binding Mode Not Reported with Other Drugs
- Chemical Structure Distinct from Ibrutinib/Other BTKi's

Retains potency against C481S-BTK

Kinase	CG-806 IC_{50} (nM)
BTK-WT	8.4
BTK-C481S	2.5

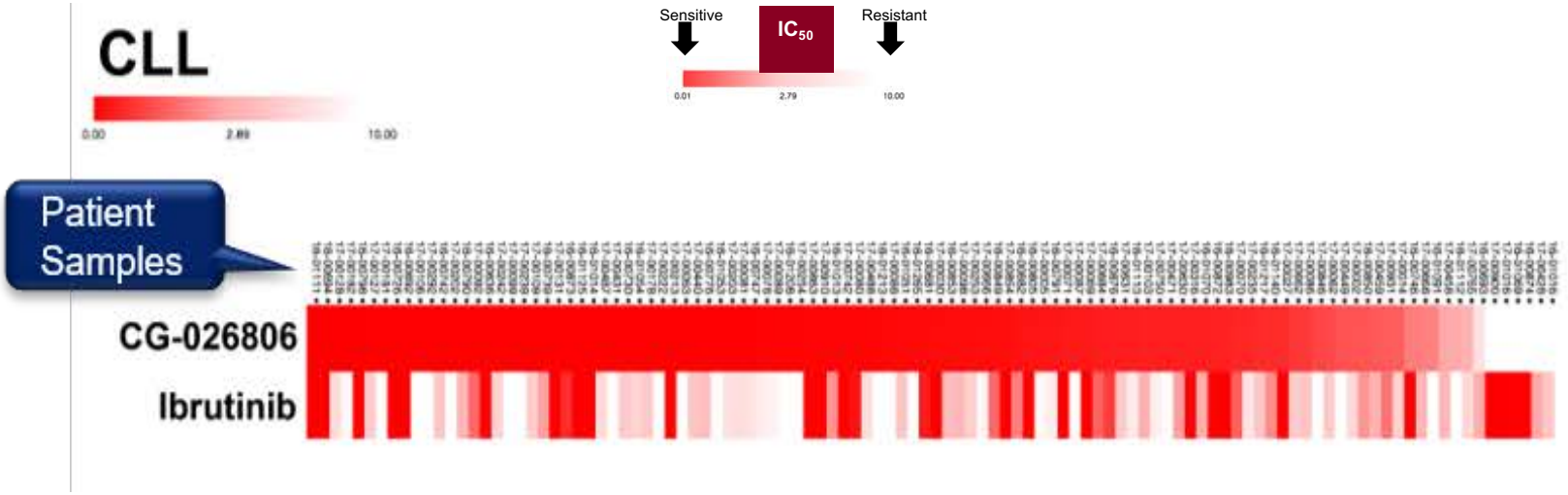
But, does NOT inhibit TEC, EGFR or ErbB2 kinases linked to ibrutinib related toxicities; including bleeding disorders, gut and skin toxicity and atrial fibrillation, respectively.

Expect Superior Safety Profile for CG-806

IC_{50} (nM)	TEC	EGFR	ErbB2
Ibrutinib	78	5.6	9.4
CG-806	>1,000	>1,000	>1,000

CG-806 Exerts Superior Breadth & Potency Compared to Ibrutinib on Patient Samples

- OHSU Measured the Ability of CG-806 or Ibrutinib to Kill Primary Cells from CLL Patients *Ex Vivo* : IC_{50} transformed into a Heatmap of Sensitivity



"CG-806 is More Than Just a BTK Inhibitor"

- **Targets driver (BTK-WT/Mutant) and rescue pathways operative in B-cell cancers**
- **1000x more potent than ibrutinib (SOC covalent BTKi) at killing malignant B-cells**

CG-806 Phase 1a/b Clinical Trial Underway: Initially in Patients with R/R CLL/SLL or NHL

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

Dose Escalation Phase

- Patients administered **oral capsules**
- **Every 12 hours** on a **28-day cycle**
- Plan to include 6 dose levels
- **Accelerated titration** design
- Planned expansion cohorts

Enrollment: 1, 1, 3x3

- *Fewer patients early in the study, but..*
- *Dose escalate quickly to effective dose*

Dose Expansion will occur once MTD or Therapeutic Dose is Reached to Define RP2D

Development Plan for Severe Unmet Needs in B Cell Tumors

CLL Patients Resistant or Intolerant to:

- Covalent BTK inhibitors
- BCL2 inhibitors (venetoclax)
- Anti-CD20 therapy (rituximab)
- PI3K inhibitors (idelalisib)
- Cytotoxic agents
- Non-covalent BTK inhibitors

NHL Patients with Unmet Needs

- Richter's Transformation
- Tx-refractory DLBCL
- Tx-refractory FL, DHL

CG-806 in Dose Level 3 of Phase 1a/b Clinical Trial in CLL/NHL

(Clinical Data Cutoff Dec 31, 2019)

Dose Level 1 (150mg BID for 28d) Completed

Only One Patient Required in Dose Level 1

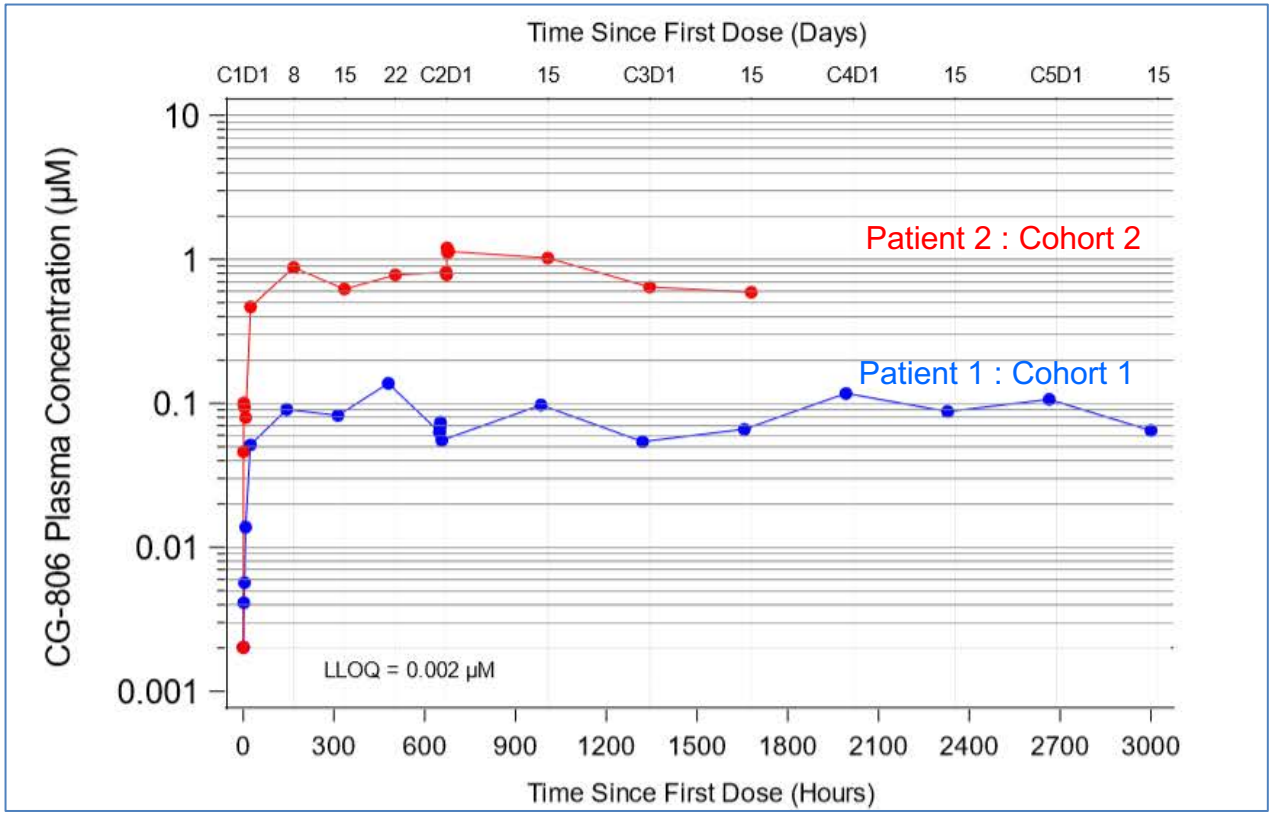
- **R/R-CLL/SLL** with TP53 mutation ; Heavily pretreated
- **Challenging Case with p53 Mutation**

Dose Level 2 (300mg BID for 28d) Completed

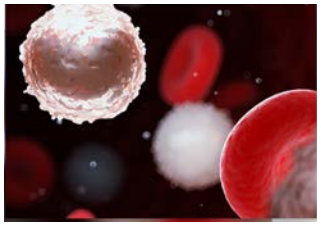
Only One Patient Required in Dose Level 2

- **R/R-CLL** with unmutated IGHV ; Heavily pretreated
- Marrow involvement with neutropenia and thrombocytopenia
- **Highly Complicated Disease to Manage**

CG-806 Favorable Steady-State Pharmacokinetics (C_{MIN})

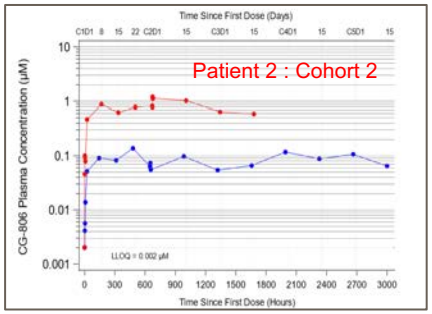


- Oral absorption, dose-related exposure, predictable steady-state PK
- Achieving 0.6-1µM steady state (C_{min}) levels in Patient at Dose Level 2
- Approaching what we believe is active exposure in Dose Level 2
- Asked if the exposure at Dose Level 2 could inhibit P-BTK

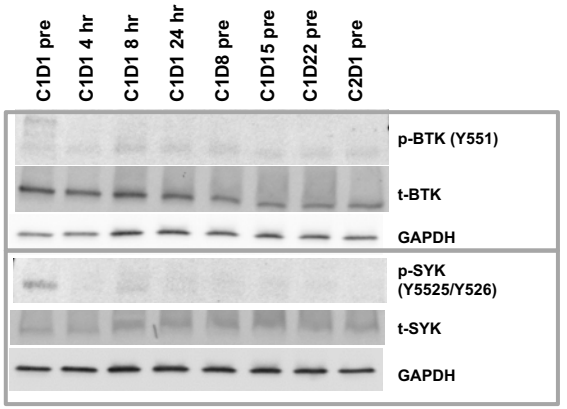


CG-806 Delivered Dose-Related Inhibition of Pharmacodynamic Markers (P-BTK) in CLL Patient #2 (300mg BID)

- **Phospho-BTK Inhibition in PBMC**
  - Whole blood collected four hours after administration of the first dose
  - ELISA assay to determine Phospho-BTK and total-BTK levels
  - BTK-pTyr223 completely inhibited at 4 hrs



- **Phospho-BTK Inhibited in Reporter Cells by Plasma**
  - Plasma inhibitory assay (PIA) using plasma collected from patient
  - EOL-1 reporter cells *in vitro* treated 6hrs with plasma from patient
  - Reduction of key phospho-proteins in EOL-1 reporter cells
    - phospho-BTK (pTyr551)
    - phospho-SYK (pTyr5525/Tyr526)
  - PD responses correlated with CG-806 concentration in plasma



CG-806 Delivered Evidence Safety, Target Engagement and Clinical Activity in Patient #2 (300mg BID)

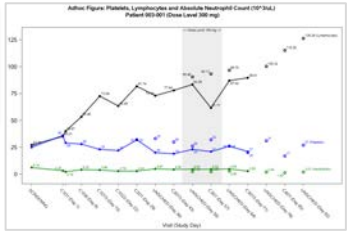
Evidence of Safety with No Unexpected Toxicities

- No myelosuppression ; stabilized platelets and neutrophils
- No drug-related SAEs ; No dose-limiting toxicities

Evidence of Target Engagement with ↓P-BTK

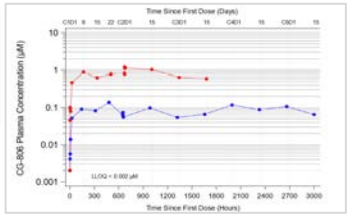
- Inhibition of P-BTK, P-SYK, others : PIA Assay
- 100% inhibition of P-BTK in PBMC : ELISA Assay

Evidence of Clinical Activity in R/R CLL



- **Marked lymphocytosis**
  - BTK inhibition in patients leads to CLL cell exfiltrated from lymphoid tissues
  - Observed immediately upon initiation of dosing in Cycle 1

Well-behaved Oral Steady-State Pharmacokinetics



- Absorption that delivered near-uM exposures

CG-806 Now in Dose Level 3 of Phase 1a/b Clinical Trial in CLL/NHL

Dose Level 1 (150mg BID for 28d) Completed

Only One Patient Required in Dose Level 1

- **R/R-CLL/SLL** with TP53 mutation ; Heavily pretreated
- **Challenging Case with p53 Mutation – No DLTs and completed Cycle 6**

Dose Level 2 (300mg BID for 28d) Completed

Only One Patient Required in Dose Level 2

- **R/R-CLL** with unmutated IGHV ; Heavily pretreated
- Marrow involvement with neutropenia and thrombocytopenia
- **Highly Complicated Disease to Manage – No DLTs and completed Cycle 4.5**

Dose Level 3 (450mg BID for 28d) Dosing Ongoing

Three Patients Required in Dose Level 3

CG-806 : A New Class of Drugs
More than Just a BTK Inhibitor for CLL
Only Agent Also to Inhibit FLT3 for AML

Breadth for Difficult-to-Treat CLL and NHL Patients

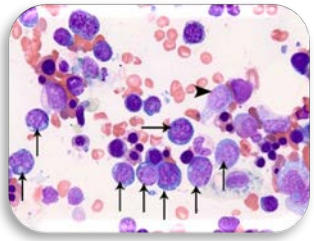
- Potently inhibits WT-BTK and C481S-BTK, plus multiple oncogenic pathways operative in B-cell cancers
- Potential to treat CLL patients failing covalent & non-covalent BTKi, Bcl-2i, CD-20 antibodies, and others
- Potential to treat Richter's Transformation, Tx-refractory DLBCL / Follicular Lymphomas / DHLs

Safety : Targets Key Oncogenic Kinases and Avoids Safety Targets

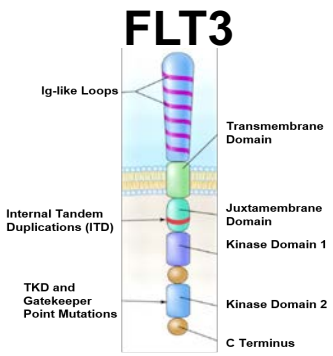
- To date: safe, well-tolerated, and no drug related AEs have been observed
- Does not inhibit TEC, EGFR or ErbB2 kinases that cause toxicities with other BTK inhibitors
- Structurally distinct : assumes unique binding mode in kinase active sites relative to competitor agents

PLUS….Under Development for AML Patients Failing Other Drugs

- Only molecule that inhibits BTK and FLT3 and is being developed for CLL/NHL and AML/MDS



CG-806 For the Treatment of AML (Acute Myeloid Leukemia)



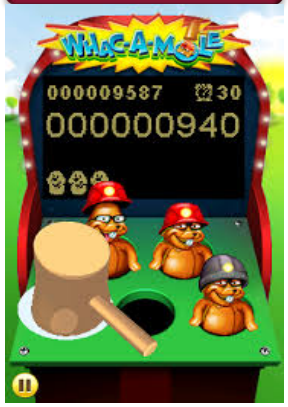
Aggressive Cancer of Blood/Bone Marrow (Orphan Disease)

- **FLT3-ITD mutation** is key driver in **25-35% of AML patients**[2,3]
- Approved: Midostaurin (Rydapt®); Gilteritinib (Xospata®)
- Advanced Development Stage: Quizartinib; Crenolanib

Medical Need For a Superior FLT3 Inhibitor

- "Dirty" agents (Midostaurin, etc.) are limited ➔ Toxicity
- "Selective" agents don't provide durable responses ➔ Resistance
- Need potent drug to inhibit *all* WT and mutant forms of FLT3: ITD/TKD/GK/WT

"Whac-a-mole"



Inhibiting FLT3 Only is Not Enough to Control AML

- Need to suppress multiple other oncogenic signaling pathways that compensate

CG-806 Potently Inhibits All FLT3 + "Rescue" Pathways

- FLT3, PDGFRα, CSF1R, BTK, SYK, ERK, AKT, JAK/STAT, MAPK, MYC pathways

(1) American Cancer Society : [2] *Cancer*. 2014 July 15; 120(14): 2142-2149 : [3] *Blood* 2016;128(5);686-698.

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

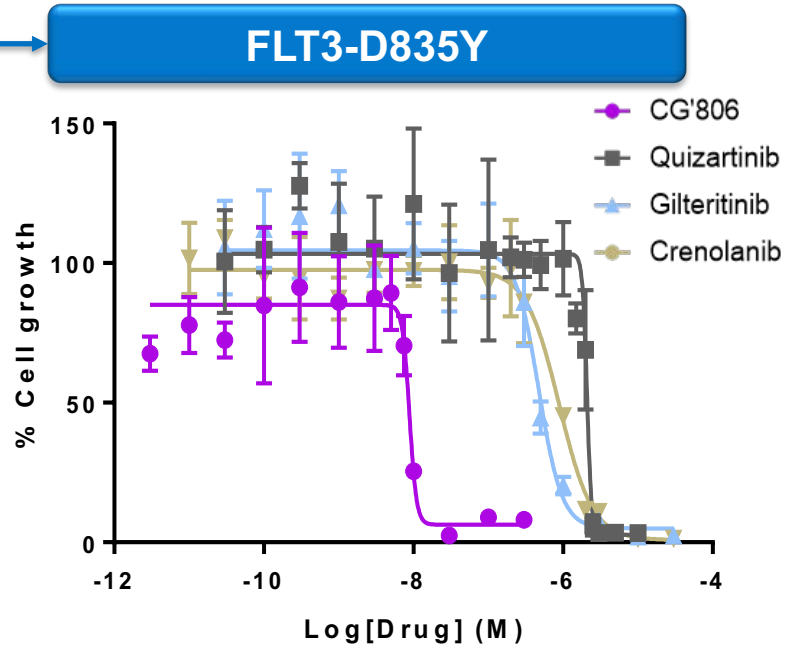
CG-806 Superior to Other FLT3-ITD Inhibitor

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

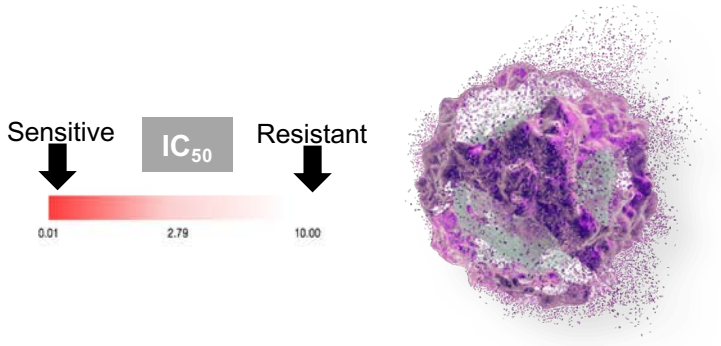


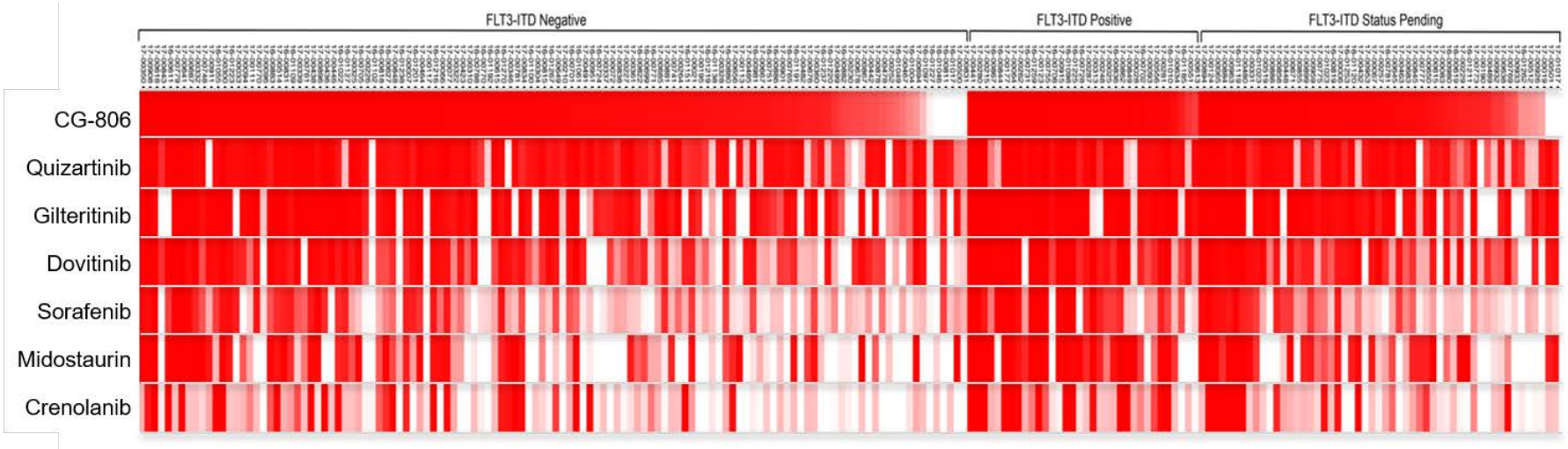
(1)Ba/F3 isogenic cells kindly provided by Dr. Michael Andreeff at MDACC

(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.

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

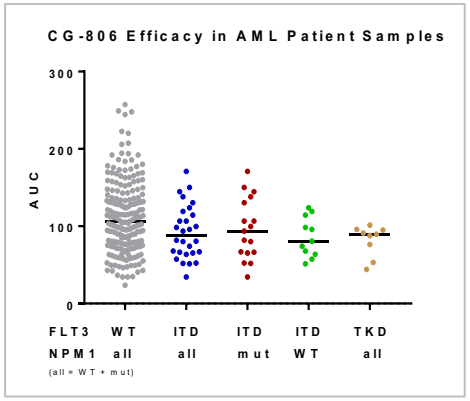
- OHSU Measured the Ability of CG-806 and Various FLT3i's to Kill Ex Vivo the Primary Cells from >200 AML Patients : IC_{50} transformed into a Heatmap of Sensitivity
- **CG-806 greater potency in killing primary AML cells bearing wild-type FLT3 or FLT3-ITD**

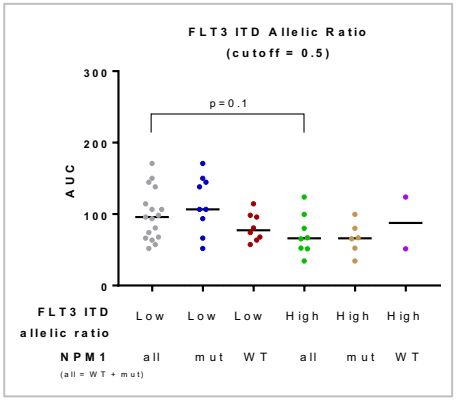


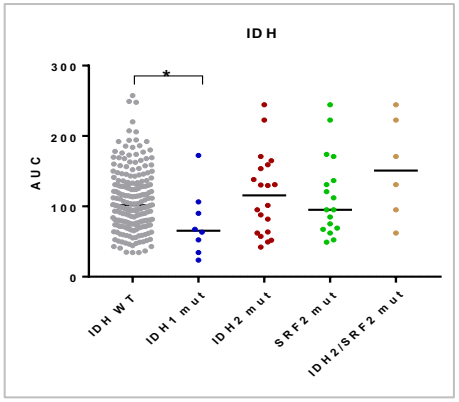


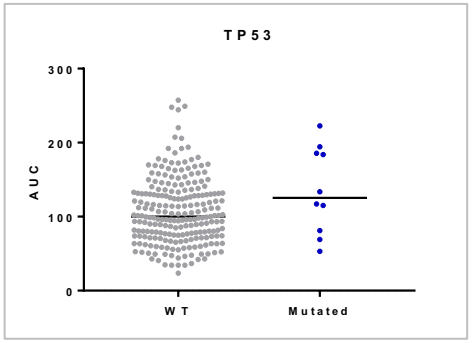
Sensitivity of AML Patients Samples to CG-806: AACR 2019

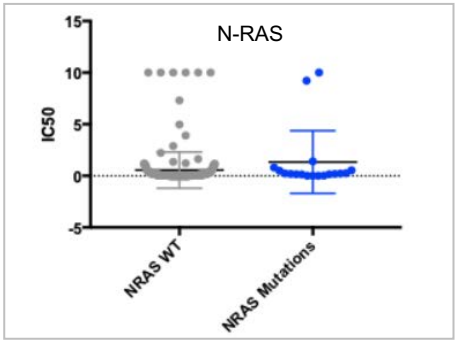
Enhanced Potency in Samples with FLT3 and IDH1 Mutations
Retains Potency in Samples with NPM1, p53, N-RAS and ASXL1 Mutations

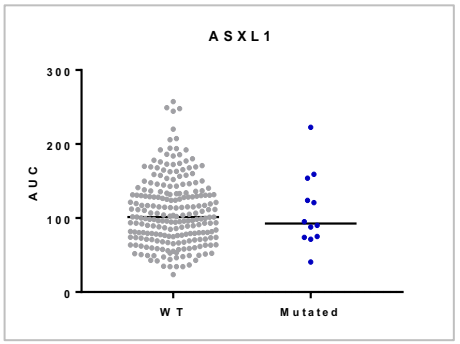






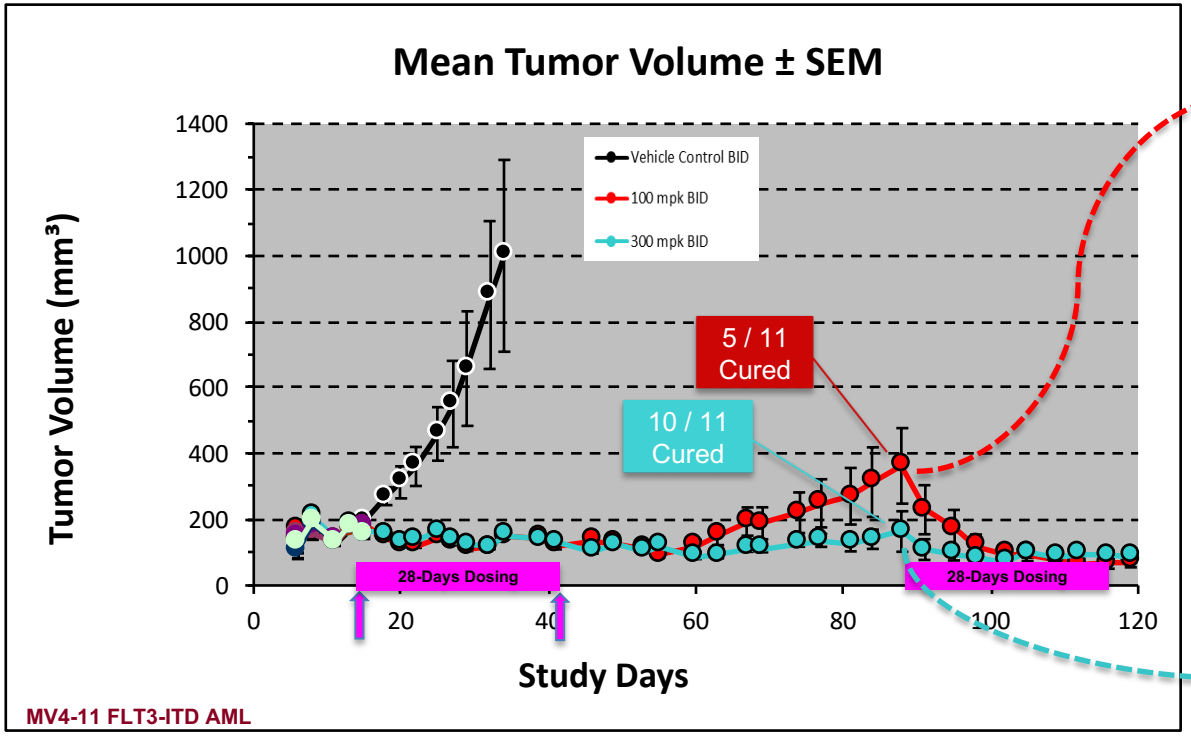


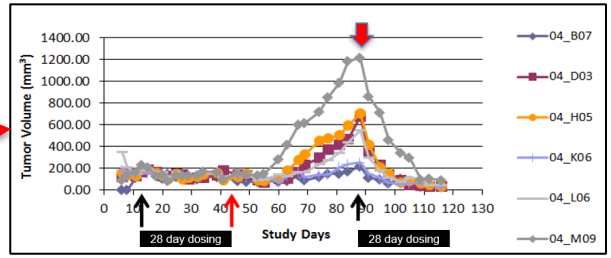




- AML patient samples with FLT3 mutations (ITD or TKD), with or without concurrent mutations of NPM1, are highly sensitive to CG-806
- Sensitivity of AML patient samples generally related to FLT3 ITD high allelic ratio (IC50 = 0.03 μM) vs. low allelic ratio (IC50 = 0.11 μM)
- AML patient samples with mutated IDH1 are more sensitive to CG-806 relative to the IDH WT or IDH2 mutations ($p < 0.05$)
- AML patient samples with TP53 WT and TP53 mutations equivalently sensitive to CG-806
  - AML patient samples with TP53 mutations were resistant to most other FLT3 inhibitors
- AML patient samples with ASXL1 WT and ASXL1 mutations equivalently sensitive to CG-806
- AML patient samples with NRAS WT and NRAS mutations equivalently sensitive to CG-806

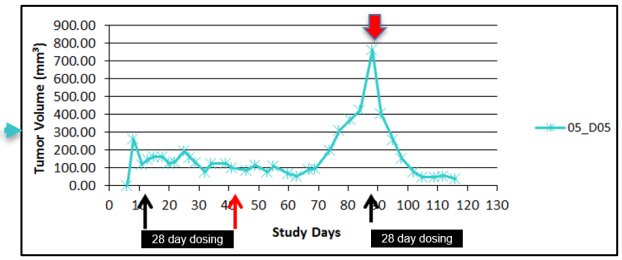
CG-806 Rapid and Sustained Antitumor Activity in Mouse Model of AML After Oral Dosing for 28 Days





100mg/kg BID
5 of 11 mice cured with 1st course

"Uncured" mice at d88 were treated with 300mg/kg BID for 2nd course of 28 days beginning d88 and those tumors responded to treatment



300mg/kg BID
10 0f 11 mice cured with 1st course

"Uncured" mouse at d88 was treated with 300mg/kg BID for 2nd course of 28 days beginning d88 and that tumor responded to treatment

- No weight loss or toxicity at any dose level
- Significant cure rates with two highest doses
- Re-challenge of uncured mice with large tumors
  - ➡ Active on large tumors and no resistance observed

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 (70% AML pts) driven by other mutations
  - More potent than other FLT3 inhibitors on >200 AML patient samples
  - Delivers cures in xenograft models of human AML without toxicity

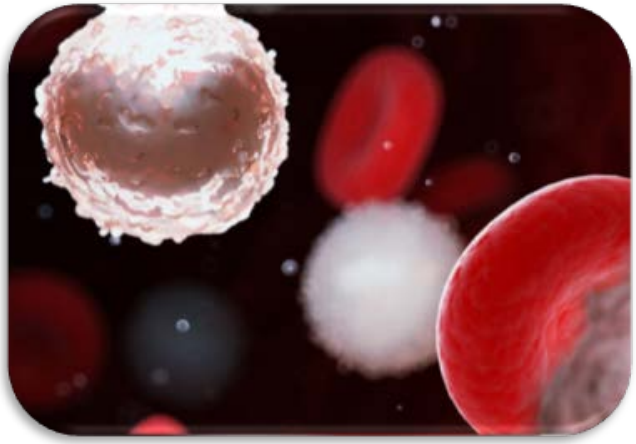
- **Phase 1 Planned : R/R AML and hr-MDS Patients with Unmet Needs**
  - Patients who failed other FLT3 inhibitors
  - Patients who failed IDH-1 inhibitors
  - Patients who failed venetoclax / mutated RAS
  - Patients with mutated p53
  - Patients with wild type-FLT3

  - Rapidly differentiate CG-806 from other FLT3i's

  &

  - Plan to initiate dosing with an active dose level

- **Plan to Initiate Trial with an Active Dose**
  - R/R-AML patients are acutely ill and we do not wish to dose sub-therapeutically
  - Continue to dose escalate in B-cell cancer patients and identify likely therapeutic dose for AML patients
  - Seek approval from FDA to initiate trial at a dose with likely "therapeutic exposure' for AML



APTO-253

Phase 1a/b Ongoing

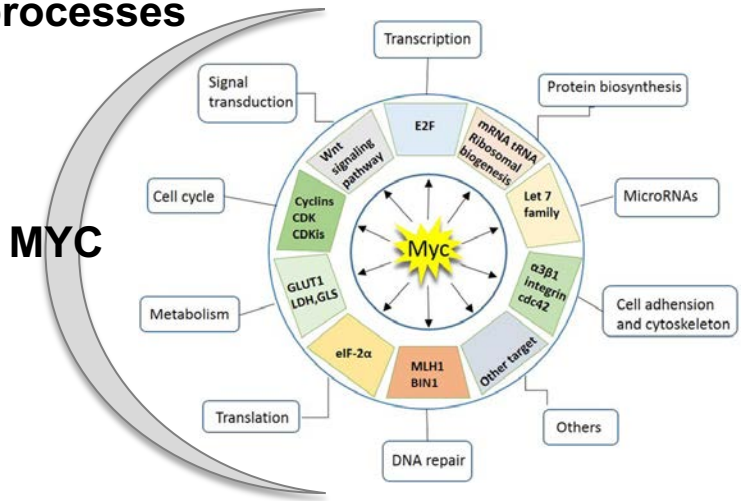
Small Molecule MYC Inhibitor

For the Treatment of AML

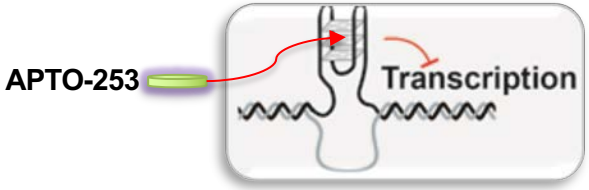
1. MYC dysregulation is key driver of AML, certain B-cell cancers and solid tumors
2. MYC gene expression potently inhibited by APTO-253
3. Ph1a/b trial for AML/MDS ongoing with APTO-253
4. APTO-253 first agent to inhibit MYC expression and well tolerated in patients

Tremendous Interest in Targeting MYC as a Cancer Treatment

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

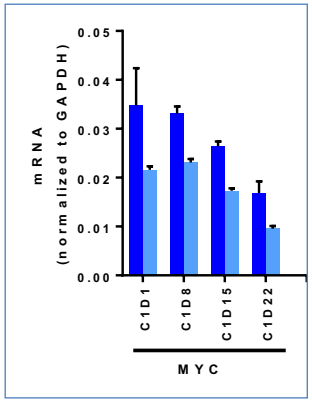


- **APTO-253 Targets DNA regulatory motif in promoter of MYC gene**
  - NOT the MYC protein
- **APTO-253 Inhibits MYC gene expression (mRNA)**
  - Depletes cells of MYC protein → induces apoptosis

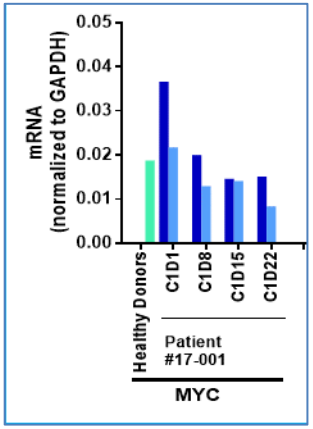


- APTO-253 inhibits MYC expression
- Causes induction of p21
- Triggers cell cycle arrest/apoptosis

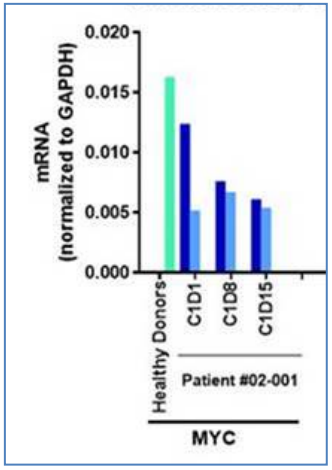
APTO-253 Phase 1 Trial :
Safely Inhibits MYC Expression in AML & MDS Patients



- **AML Patient: Dose Level 1 (20 mg/m2)**
  - Sampled pre-dose and 24 hr. post-dose day 1, 8, 15, 22
- **MYC Suppression & Well Tolerated**
  - Observed inhibition of MYC expression in PBMC



- **MDS Patient: Dose Level 2 (40 mg/m2)**
  - Sampled pre-dose and 24 hr. post-dose day 1, 8, 15, 22
- **MYC Suppression & Well Tolerated**
  - Observed inhibition of MYC expression in PBMC



- **AML Patient: Dose Level 3 (66 mg/m2)**
  - Sampled pre-dose and 24 hr. post-dose day 1, 8, 15, 22
- **MYC Suppression & Well Tolerated**
  - Observed inhibition of MYC expression in PBMC

APTO-253
Ongoing Phase 1b Dose Escalating Clinical Trial

✓ Dose Level 1 (20mg/m2)	Completed	1 AML Patient
✓ Dose Level 2 (40mg/m2)	Completed	1 MDS Patient
✓ Dose Level 3 (66mg/m2)	Completed	3 AML Patients

- To date, well-tolerated & no drug-related SAEs
- Now screening for patients in dose level 4 (100mg/m2)

DRUG	TARGET	RIGHTS	INDICATIONS	Preclinical Stage	Clinical Proof-of-Concept	Pivotal Stage
CG-806	Pan-BTK	Aptose: WW CG: Korea	CLL NHL	B-Cell Malignancies		
CG-806	Pan-FLT3	Aptose: WW CG: Korea	AML MDS	AML / MDS Planned		
APTO-253	MYC	Aptose: WW	AML MDS	AML / MDS Single Agent		
APL-581	BRD4/JAK	Aptose / Ohm	Hematologic Cancers	AML Single Agent		

2020 Anticipated Catalysts

CG-806	1H: Initiate trial in AML patients 2H: Seek clinical activity in AML patients 2H: Seek clinical activity in B-cell cancer patients 1-2H: Presentation of clinical data during EHA and ASH
APTO-253	1H: Continue dose escalation in AML/MDS patients 2H: Explore additional cancer indications 2H: Presentation of clinical data during ASH

KOL Symposium on CG-806 FLT3 / BTK Inhibitor for Acute Myeloid Leukemia

Hosted by Aptose Biosciences Inc. (Nasdaq: APTO)

The luncheon symposium with Key Opinion Leaders in hematology/oncology will review the treatment landscape and the evolution of kinase inhibitors as anticancer drugs in myeloid leukemias, and highlight the potential for the mutation-agnostic FLT3/BTK inhibitor CG-806 to address unmet medical needs in these patient populations.

Additionally, the Aptose management team will provide an overview of the rationale and strategy for the development of CG-806 in myeloid malignancies. CG-806 is currently in an ongoing Phase 1a/b clinical trial for the treatment of patients with relapsed / refractory B-cell malignancies, including CLL and NHL, and in 1H / 2020 is planned to enter a separate clinical trial in patients with relapsed / refractory AML and high-risk MDS.

Additional information will be provided closer to the date of the event.

Wednesday, February 5th, 2020
Noon - 1:30 PM EST

Lotte New York Palace
455 Madison Ave

Thank You!

APTOSE BIOSCIENCES