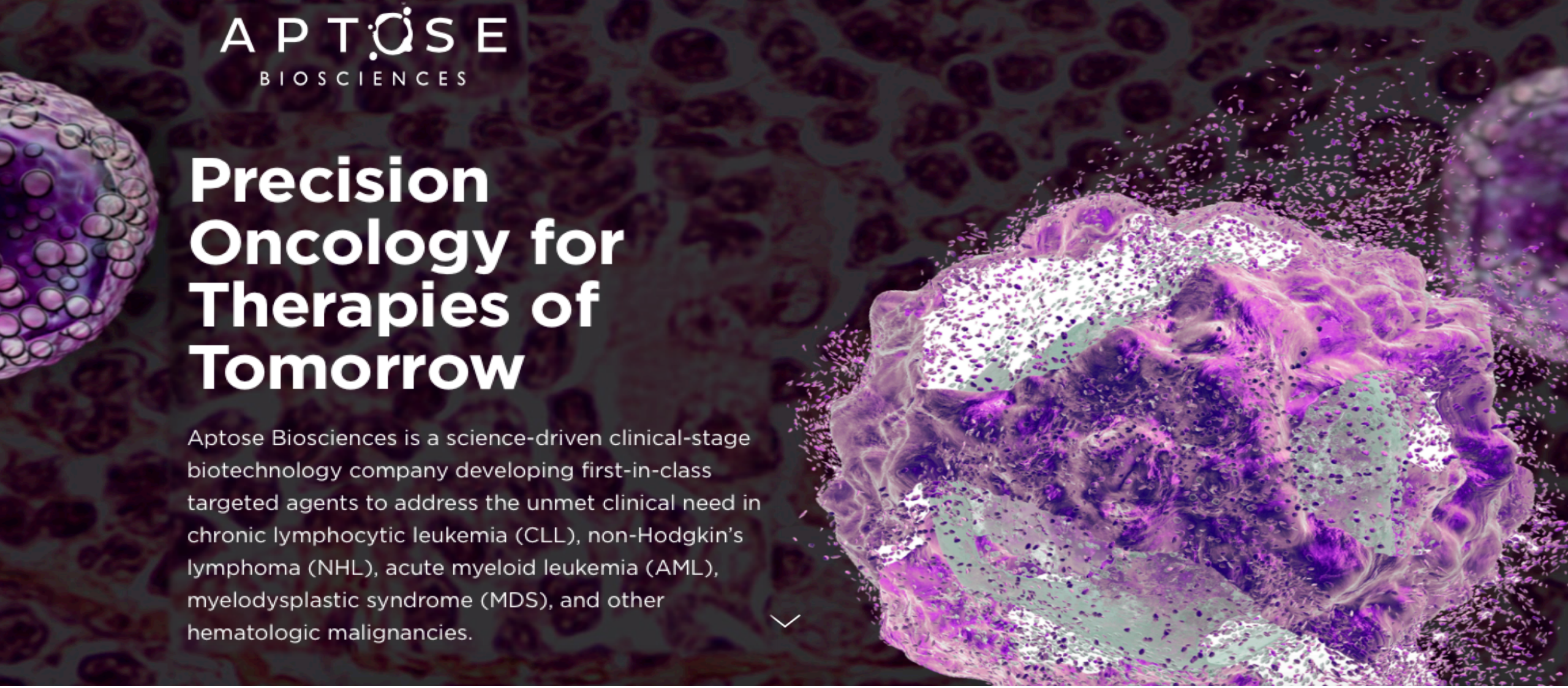
APTOSE BIOSCIENCES

Precision Oncology for Therapies of Tomorrow

Aptose Biosciences is a science-driven clinical-stage biotechnology company developing first-in-class targeted agents to address the unmet clinical need in chronic lymphocytic leukemia (CLL), non-Hodgkin's lymphoma (NHL), acute myeloid leukemia (AML), myelodysplastic syndrome (MDS), and other hematologic malignancies.



BIO CEO & INVESTOR CONFERENCE 2020

NASDAQ: APTO
TSX: APS

Dr. William G. Rice ; Chairman, President, CEO
February 2020

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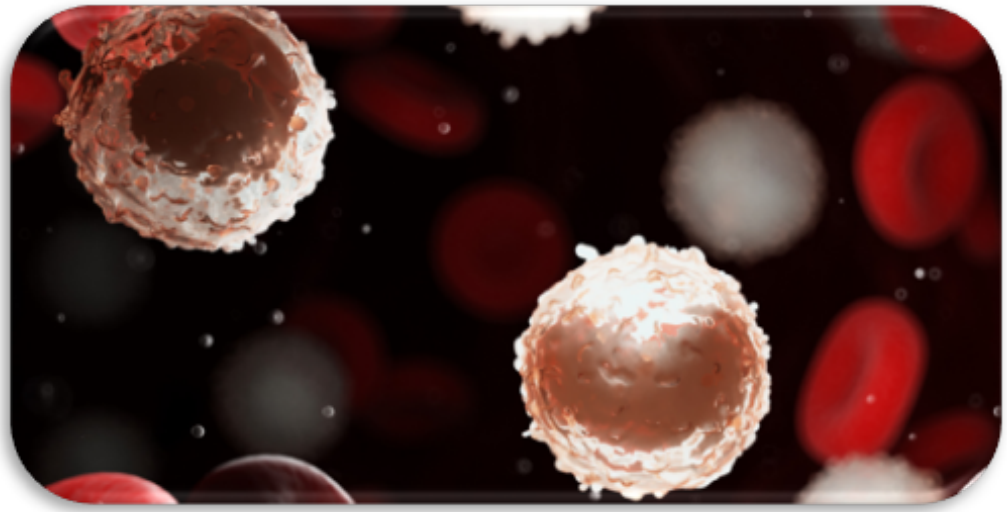

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This presentation contains forward-looking statements, which reflect APTOSE Biosciences Inc.'s (the "Company") current expectations, estimates and projections regarding future events, including statements relating to our business strategy, our clinical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

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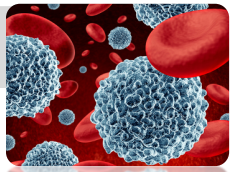
Investment Highlights

APTOSE



Strong leadership and 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

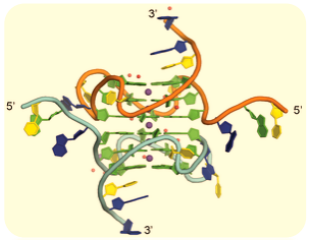
CG-806 Oral FLT3 / BTK Kinase Inhibitor

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



APTO-253 MYC Inhibitor

FDA Orphan Drug Designation in AML

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

Phase 1b in dose level 4 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

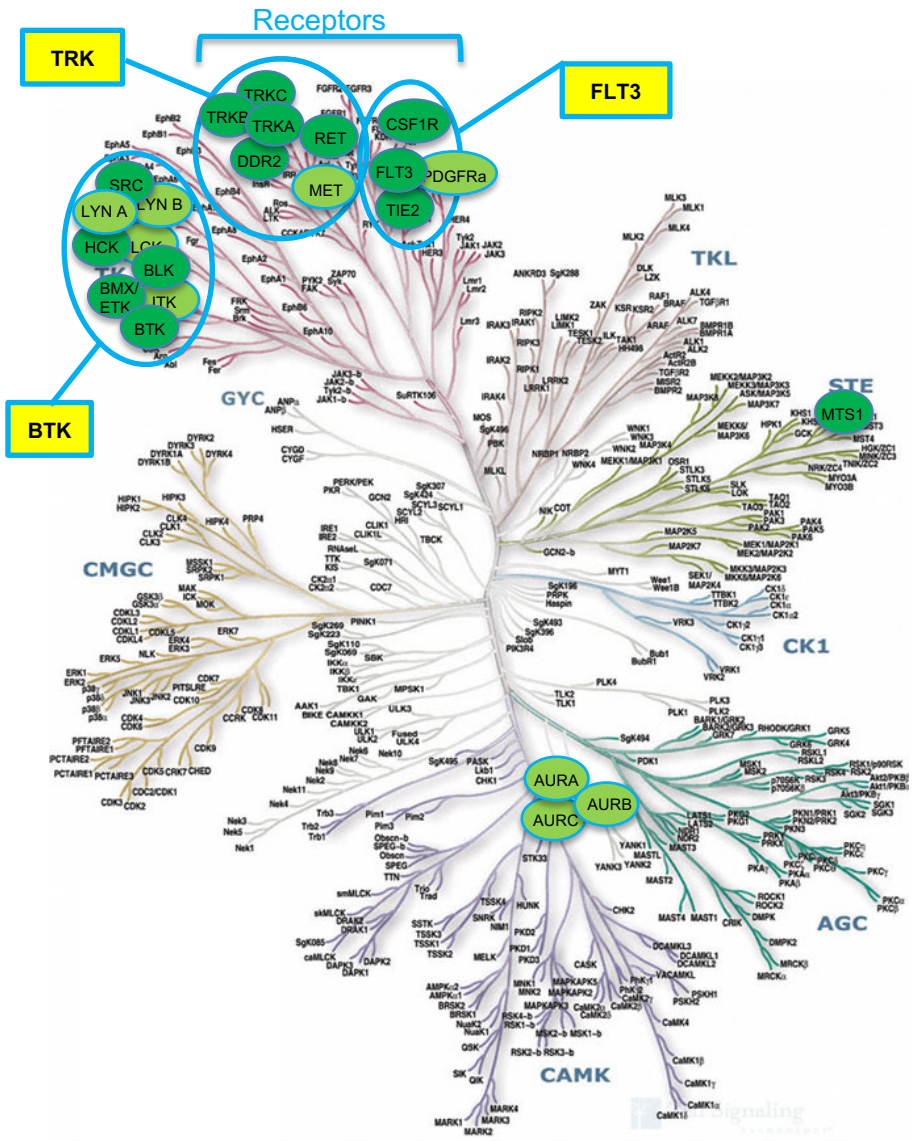
CG-806

1st-in-Class Oral Kinase Inhibitor

- Mutation Agnostic FLT3 Inhibitor
- Mutation Agnostic rBTK Inhibitor

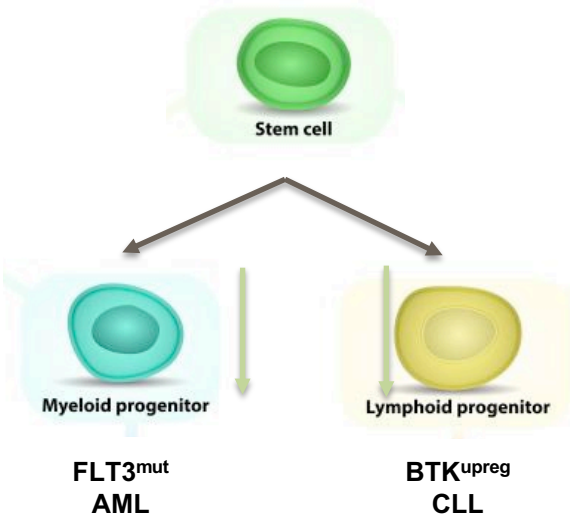
- ❑ Small molecule **"reversible" kinase inhibitor**
- ❑ Cluster-selective and **highly unique kinome targeting profile**
- ❑ Developing across ***spectrum of hematologic malignancies***
  - ***lymphoid*** malignancies (**CLL** & NHL)
  - ***myeloid*** malignancies (**AML**)
- ❑ Retains **potency on CLL & AML cells with mutations** render other agents ineffective
- ❑ Ongoing trial **Ph1a/b for CLL** and lymphoid malignancies as rBTKi
- ❑ Planning trial **Ph1a/b for AML** and myeloid malignancies as FLT3i

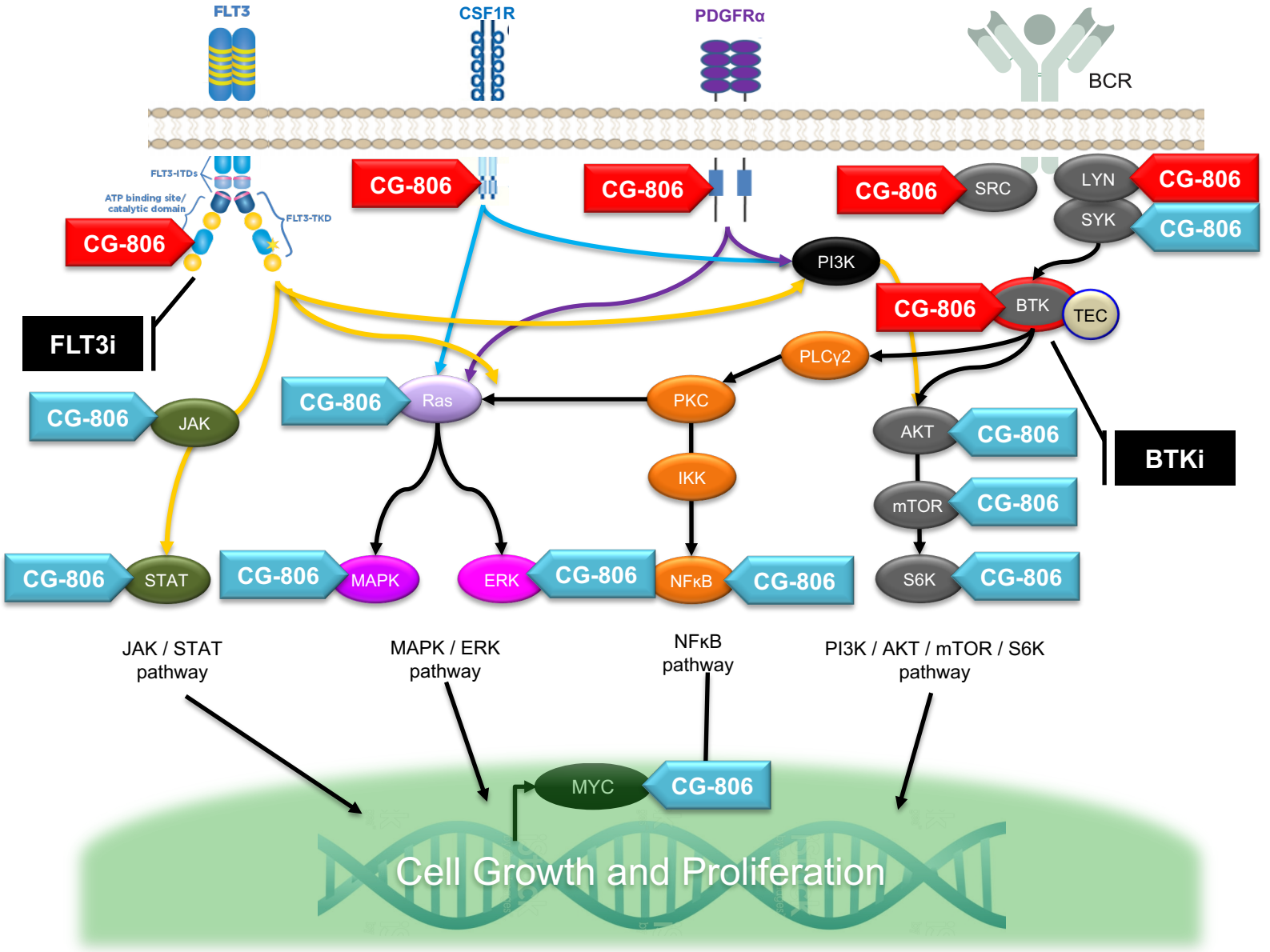
“Cluster-Selective Kinase Inhibitor”: CG-806 Potently and Selectively Inhibits Clusters of Related Kinases



- **Mutation Agnostic**
  - Inhibits WT and all mutant forms of FLT3
  - Inhibits WT and all mutant forms of BTK
  - Simultaneously suppresses multiple oncogenic 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 Lymphoid and Myeloid Hematologic Malignancies**
  - BTK cluster → CLL & NHL
  - FLT3 cluster → AML & MDS

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





CG-806 Inhibited directly

CG-806 Inhibited indirectly

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

CLL & NHL Lymphoid

1st
Phase 1a/b Ongoing in Patients with R/R CLL & NHL

- Define safety, tolerance, PK, PD and RP2D in CLL/NHL patients
- Seek responses in CLL/NHL patients
  - R/R AML patients are acutely ill and we do not wish to dose sub-therapeutically
  - During CLL trial, seek a dose likely to be “therapeutically active” for AML patients

Pending FDA Approval

AML Myeloid

2nd
Perform Phase 1a/b : R/R AML

- Plan to initiate dosing in AML patients at a dose likely active
- Define safety, tolerance, PK, PD and RP2D
- Seek benefit in AML patients quickly

CG-806 for the Treatment of CLL & Lymphoid Malignancies

Overexpressed BTK (Bruton's Tyrosine Kinase) is Driver Kinase

SOC is *Ibrutinib* Covalent BTKi : Targets C481 Residue 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 (IC50 = 2.5nM) enzyme
- Inhibits multiple "oncogenic rescue" pathways simultaneously to avoid resistance
- Does no inhibit TEC, EGFR, ErbB2 kinases that cause ibrutinib intolerances

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

CG-806 Phase 1a/b Clinical Trial Underway: First in Patients with R/R CLL & NHL Lymphoid Malignancies

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**
- **Twice daily** on a **28-day cycle**
- Plan to perform 6 dose levels
- **Accelerated titration** design
- Planned expansion cohorts

Development Plan for Severe Unmet Needs in B Cell Tumors

CLL Patients Resistant or Intolerant to:

- Covalent BTK inhibitors (ibrutinib)
- 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

Enrollment: 1, 1, 3x3

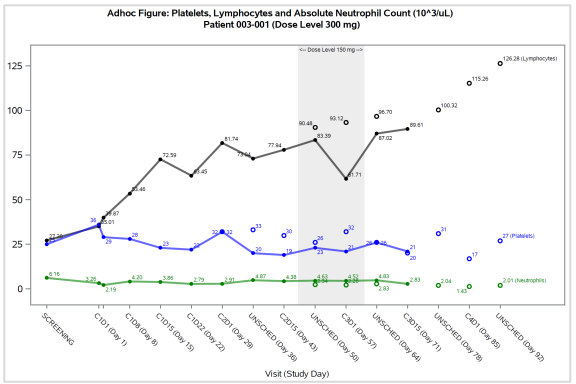
- ***Fewer patients early in the study, but…..***
- ***Dose escalate quickly to effective dose***

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

CG-806 Delivered Clinical Evidence of Safety, Pharmacologic Activity and Favorable Oral Pharmacokinetics

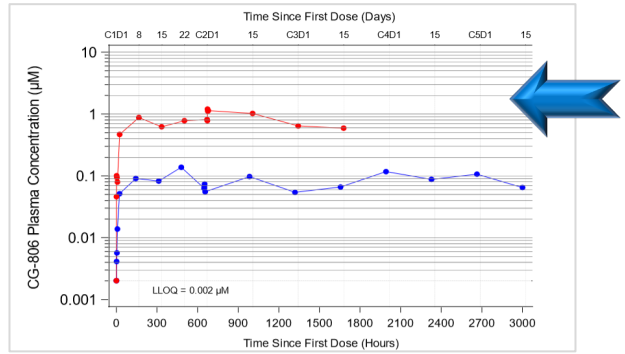
- **Patient Dose Escalation Advancing Efficiently**
  - Dose Level 1 (1 CLL patient at 150mg BID) completed
  - Dose Level 2 (1 CLL patient at 300mg BID) completed
  - Dose Level 3 (3 patients at 450mg BID) fully enrolled

- **Evidence of Safety with No Unexpected Toxicities to Date**
  - No myelosuppression to date ; No drug-related toxicities to date



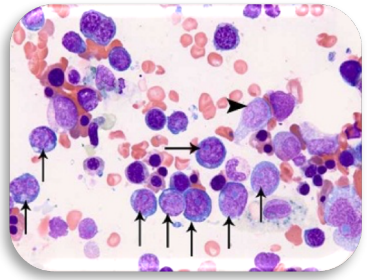
- **Evidence of Pharmacologic Activity as Early as Dose Level 2**
  - **Target Engagement**: 100% inhibition of **P-BTK** in PBMC at 4hrs
  - **Lymphocytosis:** BTK inhibition in CLL promotes exfiltration

- **Well-behaved Steady-State Oral Pharmacokinetics**
  - Absorption that delivered near-uM plasma exposure levels



- **Plan to Continue Dose Escalation + Approaching Starting Dose for AML**

CG-806 for the Treatment of AML & Myeloid Malignancies

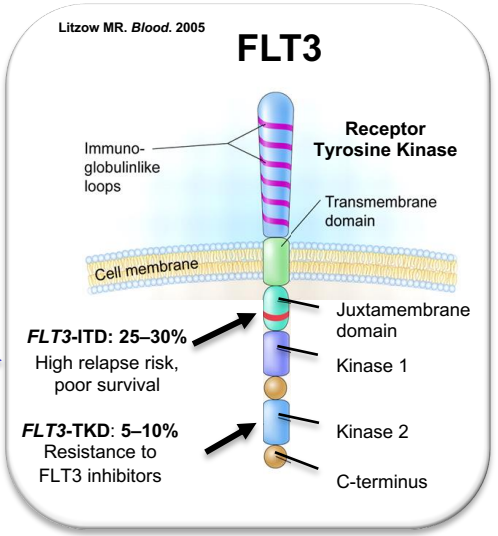


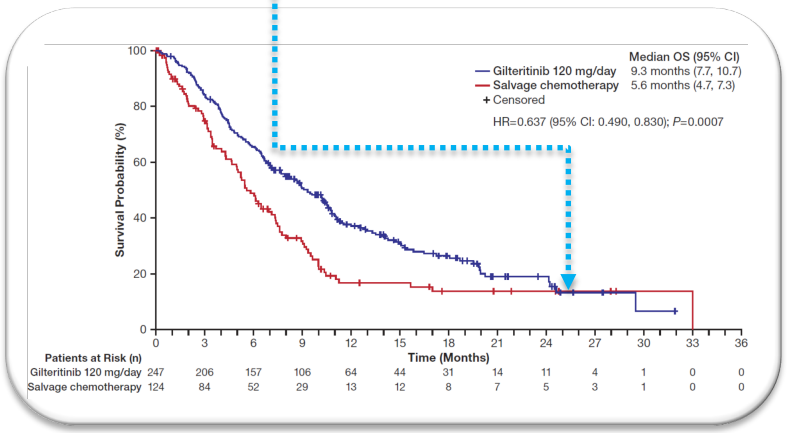
Deadly Cancer of Blood/Bone Marrow (Orphan Disease)

- ~21,450 diagnosed this year / ~10,920 deaths this year[1]
- The 5-year survival rate for patients with AML approximately 28.3%

Limitation of Current FLT3 Inhibitors and Other Agents

- **FLT3-ITD mutation** is key driver in **25-35% of AML patients**[2,3]
- Current "Dirty" agents (Midostaurin, etc.) are limited ➔ Toxicity
- Current "Selective" (Gilteritinib, Quizartinib) agents not durable ➔ Resistance
- Current agents susceptible to mutations in TP53, Ras, FLT3 (ITD/TKD/GK)





Desperate Need for Improved AML Agents → CG-806

- CG-806 potently inhibits *all* WT and mutant forms of FLT3: ITD/TKD/GK/WT
- CG-806 suppresses multiple oncogenic signaling pathways to avoid resistance
- CG-806 retains activity in presence of diverse mutational background
- CG-806 combines effectively with other therapies, i.e. venetoclax

(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

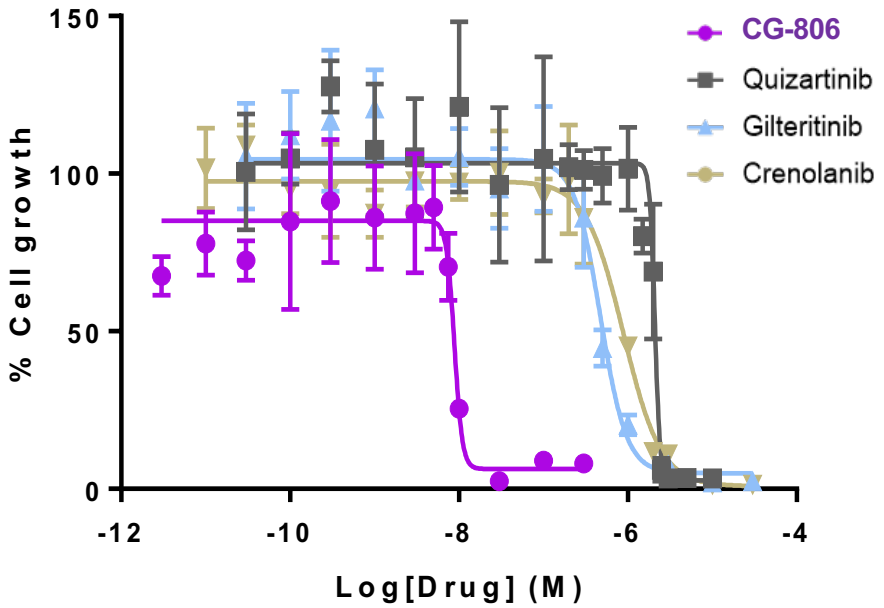
Drug	IC_{50} (nM)
CG-806(1)	0.8
Quizartinib(2)	8.8
Gilteritinib(3)	0.9
Crenolanib(4)	2
Midostaurin(2)	11
Nexavar(2)	79
Sutent(2)	1

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

FLT3-D835Y

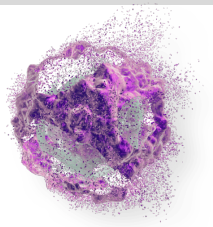


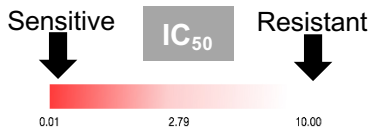
(1)Ba/F3 isogenic cells kindly provided by Dr. Michael Andreeff at MDACC

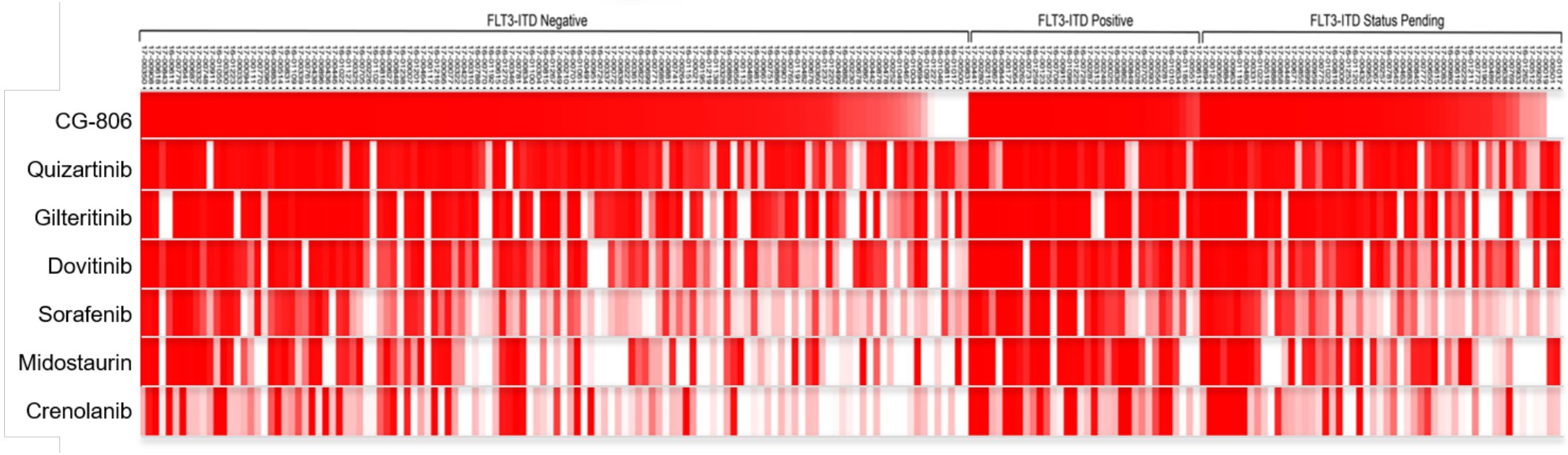
(1) Reaction Biology Corp.
(2) Blood. 2009 Oct 1; 114(14): 2984–2992
(3) J Clin Oncol 32:5s, 2014 (suppl; abstr 7070)
(4) Blood 2014 Jan 2; 123(1): 94-100 ; AACR Poster 2012
(5) ASH Oral Presentation 2016
N/A – Data not available / Not Applicable.

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

- Measured IC50 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 bearing wild-type FLT3 or FLT3-ITD**



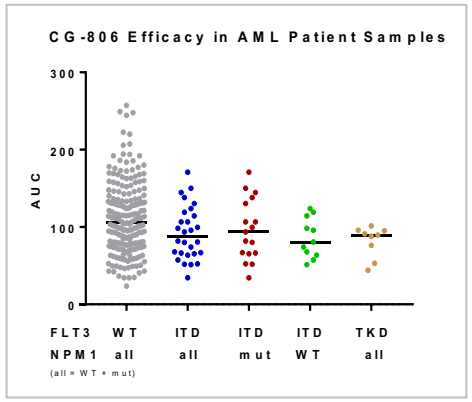


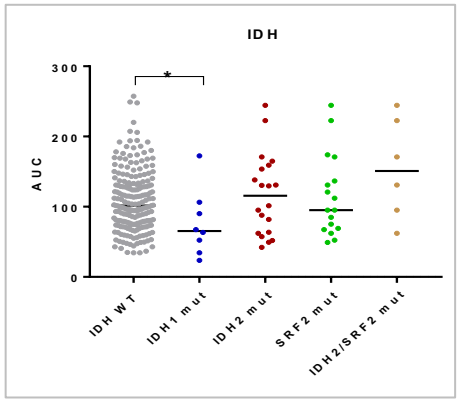


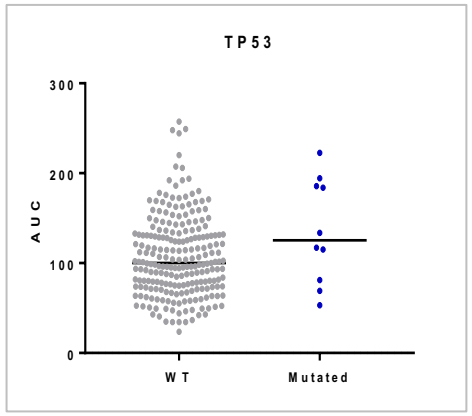
Sensitivity of AML Patients Samples to CG-806: AACR 2019

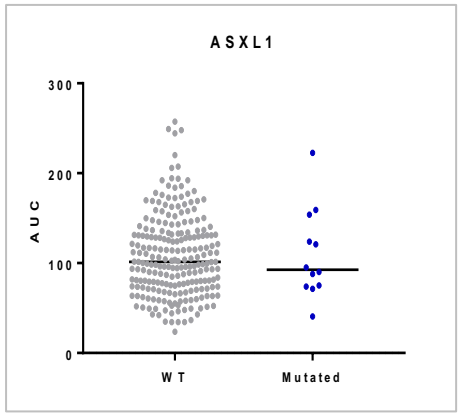
Enhanced Potency in Samples with FLT3 and IDH1 Mutations

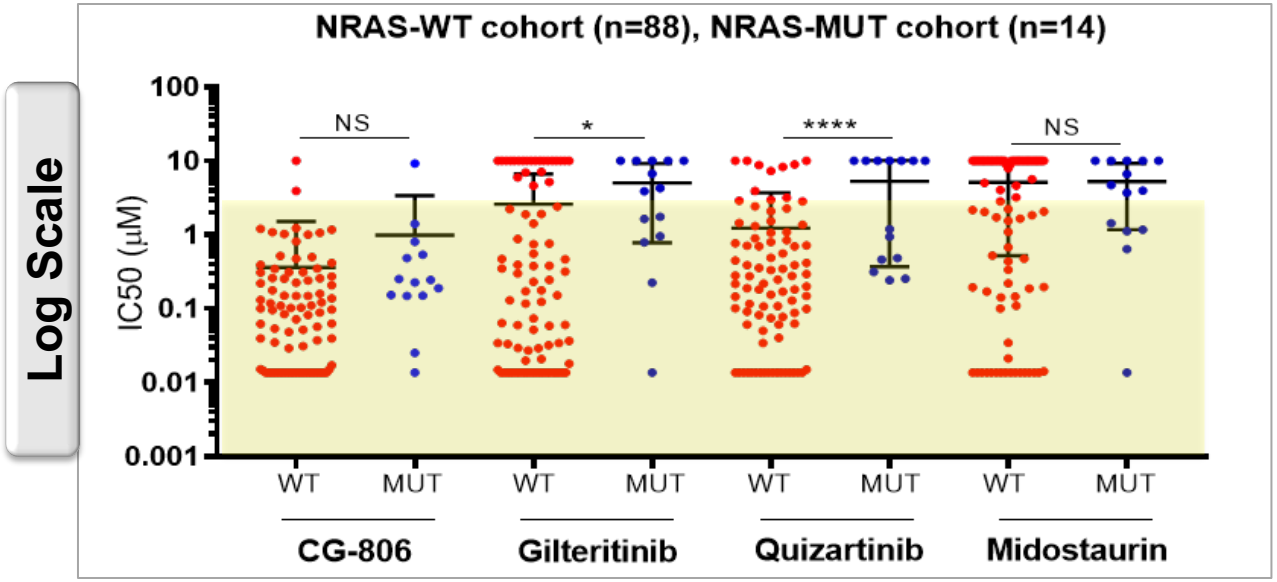
Retains Potency in Samples with NPM1, p53, N-RAS and ASXL1 Mutations





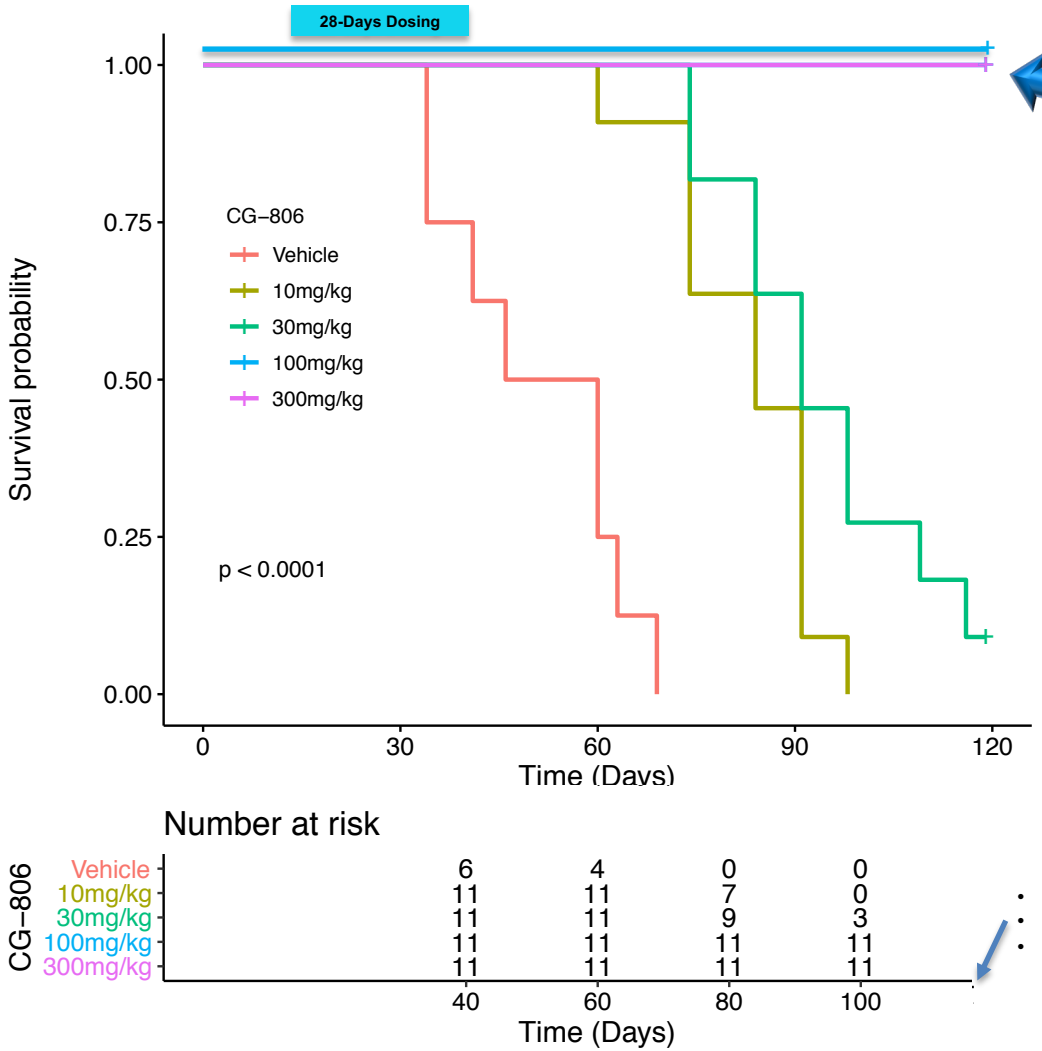






- AML patient samples with FLT3 mutations (ITD or TKD), with or without concurrent mutations of NPM1, are highly sensitive to CG-806
- AML patient samples with mutated IDH1 are more sensitive to CG-806 relative to the IDH WT or IDH2 mutations ($p < 0.05$)
- AML patient samples with TP53 WT and TP53 mutations equivalently sensitive to CG-806
- AML patient samples with 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 Extends Survival in Dose Dependent Way in Mouse Model of AML After Oral Dosing for 28 Days



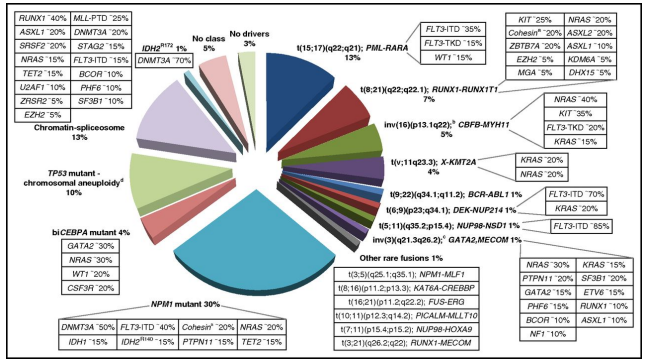
- Study terminated on day 119
- 11 / 11 mice survived in the 100mg/kg group
- 11 / 11 mice survived in the 300mg/kg group

- **100% cure rates** at two highest dose levels
- **No evidence of toxicity** at any dose

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

Cyto/Molecular Heterogeneity of AML



- **Phase 1 Planned : 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
  - Patients unfit for intensive therapies

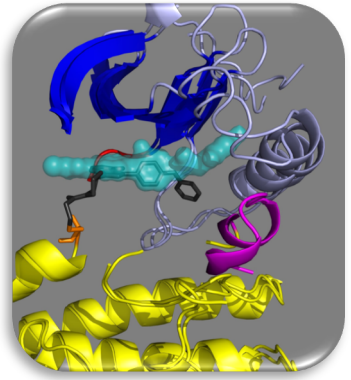
- Plan to initiate dosing with an active dose

&

- Rapidly differentiate CG-806 from other FLT3i's

Developing CG-806 Broadly Across Hematologic Malignancies

- **Uniquely and Selectively Inhibits Clusters of Kinases**
  - Targets kinases that are drivers of hematologic malignancies (lymphoid and myeloid)
  - Yet, avoids kinases generally associated with toxicity
- **Phase 1 Ongoing in R/R CLL & NHL Lymphoid Cancer Patients**
  - Targeting BTK and multiple survival pathways to treat patients failing other agents
  - Observed safety, pharmacologic activity and predictable PK characteristics
  - Continuing to dose escalate and seek safety, PD responses and efficacy responses
- **Phase 1 Planned in R/R AML Myeloid Cancer Patients**
  - Targeting FLT3 and multiple survival pathways to treat patients failing other agents
  - Plan to initiate dosing at active level; Potential for rapid development and value creation



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