CORPORATE PRESENTATION
APRIL 2018

APTOSE BIOSCIENCES

NASDAQ: APTO TSX: APS

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Aptose Corporate Snapshot

Clinical stage company employing a mechanism-driven approach to deliver safer, targeted, first-in-class cancer drugs	
Public Company	**NASDAQ: APTO** TSX: APS
Shares Outs. (3/30/2018)	Basic: 30.7 MM; FD: 35.1 MM *No Warrants / No Preferred Stock / No Debt*
3 Month ADTV	~450,000 Shares
Market Cap (3/29/2018)	~$95 Million
Cash Position (12/31/17)	US$11.4 Million / CA$13.6 Million
Cash Runway	>12 Months
Executive Headquarters & Research Laboratories	San Diego, CA

Aptose Investment Highlights

Clinical stage **biotechnology** company **developing first-in-class targeted agents** to treat life-threatening **hematological malignancies** / orphan opportunities

Two differentiated targeted agents with Strong IP Protection

CG'806 : Oral Pan-FLT3 / Pan-BTK Inhibitor

Non-covalent

FDA Orphan Drug Designation in AML

- Potent inhibitor of wild type & all mutant FLT3 >> AML
- Potent inhibitor of wild type & mutant BTK(C481S) >> CLL
- First-in-human trials planned in 2018 for AML and CLL

APTO-253 : c-Myc Inhibitor

FDA Orphan Drug Designation in AML

- Small molecule inhibitor of c-Myc oncogene expression
- Currently at Phase Ib stage for acute myeloid leukemia (AML)

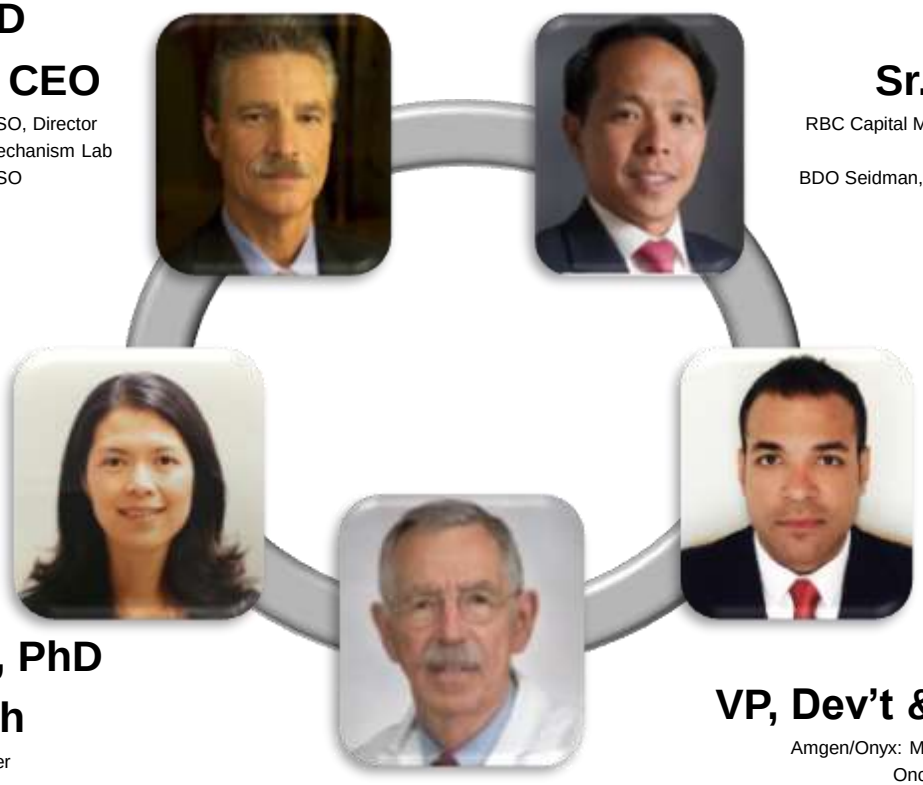
$1B+ commercial opportunity in lead indications (AML and CLL)

Strong leadership team comprised of accomplished industry, financial and clinical research professionals

Aptose Leadership Team

Dr. William G. Rice, PhD
Chairman, President & CEO

Achillion Pharmaceuticals: Founder, CEO, President, CSO, Director
National Cancer Institute-FCRDC: Sr. Scientist, Drug Mechanism Lab
Cylene Pharmaceuticals: Chairman, CEO, President, CSO



Mr. Gregory Chow
Sr. Vice President & CFO

RBC Capital Markets: Director, Led Life Sciences Private Placements
Wells Fargo: Led Private Capital Group
BDO Seidman, LLP: Senior Auditor, CPA (inactive), State of California

Dr. Hannah Zhang, MD, PhD
Sr. Director of Research

Aangstrom Pharm: Project Mgr to Moores Cancer Center
Bio-Quant: Sr. Research Scientist
Guilin Medical College, Guilin, P.R. China: Ob.Gyn.

Mr. Ernest Kitt
VP, Dev't & Technical Operations

Amgen/Onyx: Molecule Lead Director for Kyprolis in Clinical Operations
Oncosec Medical: Executive Director of Clinical Operations
Medicinova Inc: Associate Director of Clinical Operations

Dr. Stephen Howell, MD
Serves as Chief Medical Officer

Distinguished Professor of Medicine, UCSD Moore's Cancer Center
Physician scientist conducting research to address drug resistance
Expertise in pharmacology and design and conduct of clinical trials

Aptose Scientific/Clinical Advisory Team

Dr. Brian J. Druker, MD

Collaborator & Chair of SAB

Key Role in Dev't of Gleevec

Member, National Academy of Sciences

Winner of Karnofsky Award and Lasker "America's Nobel" Award

*Leader of Inter-institutional **Beat AML Initiative***

Dr. Daniel Von Hoff, MD, FACP

Serves as SVP of Medical Affairs

*Winner of 2010 **Karnofsky** Memorial Award*

Prior President of AACR

Board Member of ASCO

Appointed to President's National Cancer Advisory Board

Dr. Michael Andreeff, MD, PhD

Collaborator & Member of SAB

Professor of Medicine, Chair in Genetics,
MD Anderson Cancer Center

Physician Scientist, expert in AML / drug resistance / drug mechanisms, published over 450 peer-reviewed papers / books / chapters

Scientific Advisory Board Populated with KOLs – Domain Expertise

2018 AACR Presentations

CG'806, a first-in-class pan-FLT3/pan-BTK inhibitor, demonstrates superior potency to other FLT3 and BTK inhibitors against primary human samples

Kurtz, Watanabe-Smith, Bottomly, Wilmot, Mcweeney, Local, Zhang, Howell, Rice, **Druker**, Tyner

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CG'806, a first-in-class pan-FLT3/pan-BTK inhibitor, targets multiple pathways to kill diverse subtypes of acute myeloid leukemia and B-cell malignancy cells in vitro

Zhang, Local, Benbatoul, Folger, Sheng, Tsai, Howell, Rice

APTO-253 is a new addition to the repertoire of drugs that can exploit DNA BRCA1/2 deficiency

Tsai, Sun, Zhang, Local, Rice, Howell

CG'806

First-in-Class
Pan-FLT3 / Pan-BTK

"Multi-Cluster" Kinase Inhibitor

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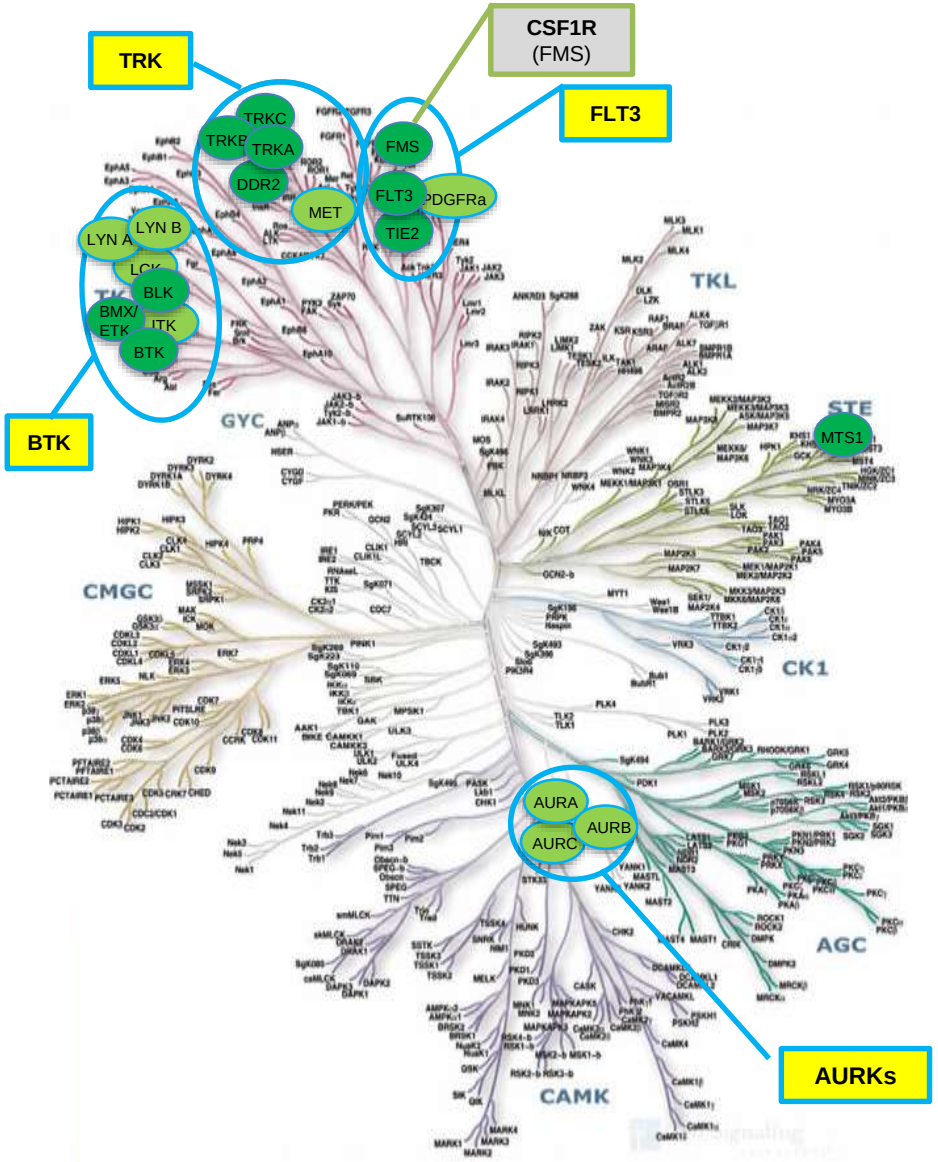
CG’806 Selectively and Potently Targets “Clusters of Related Kinases”

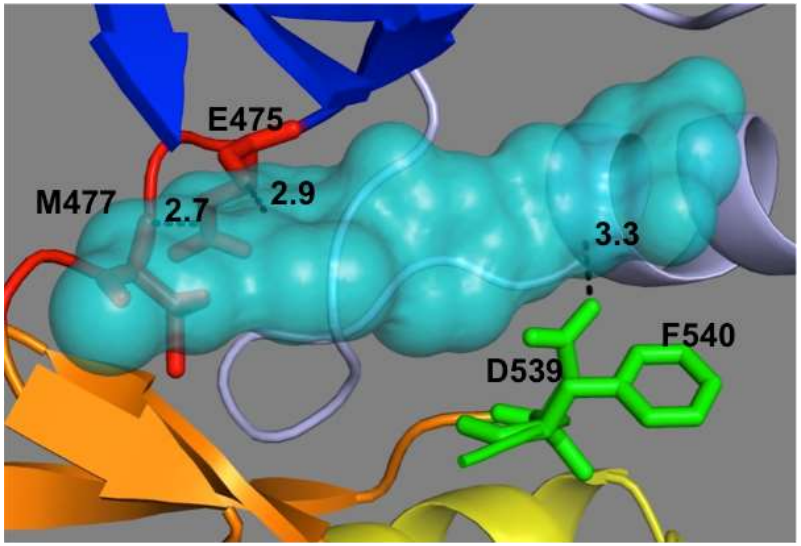
Kinome Tree Image

- **NOT** a “Dirty Kinase Inhibitor”
- **Targets Specific Clusters** of oncogenic kinases operative in **AML** and **B-cell Malignancies**
- Does NOT potently inhibit other clusters or targets associated with toxicity
- How does CG’806 selectively target specific clusters?
- Selectivity may be linked to rigidity, functionalization and “unique binding modes”



CG’806 Demonstrates Unique Binding Modes to Kinase Active Sites

X-ray Crystal Structure
CG’806 in BTK-C481S



Atypical Type II
Binding Mode in BTK

CG’806 Structure Activity Relationship

- **Rigid molecule** : “fits” into active site of structurally related kinases, but... restricted from entering active site of other kinases
- **Highly functionalized** : allows productive binding after entering active site
- **Selectivity for Clusters** : **Appears linked to rigidity, functionalization and “unique binding modes” and reduces potential off-target effects**

Aptose Program Pipeline

DRUG	TARGET	INDICATION	DISCOVERY	PRE-CLINICAL	PHASE 1	PHASE 2
CG'806	Pan-FLT3	AML				
CG'806	BTK-WT/C481S	B Cell Cancers (CLL/MCL/DLBCL)				
APTO-253	c-Myc	AML / MDS				
APL-581	BRD/Kinase	Hematologic Malignancies				

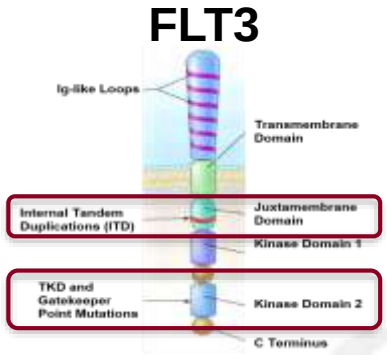
CG'806 For the Treatment of Acute Myeloid Leukemia (AML)

Aggressive & Heterogeneous Cancer of Blood and Bone Marrow

- **21,000 new cases** estimated in US in 2016 and leading to **10,600 deaths**[1]
- **FLT3-ITD mutation** is key driver in **25-35% of AML patients**[2,3]

Medical Need For Superior FLT3 Inhibitor

- Midostaurin (Rydapt®) approved and others in development
- Current agents unable to control *all* mutant FLT3 forms ➔ Resistance
- Need potent drug to inhibit *all* mutant forms of FLT3: ITD/TKD/GK/WT



Targeting Only FLT3 is Not Enough to Control AML

- Multiple pathways operative – compensate for loss of one or few
- Need potent drug to target FLT3 **plus** multiple other "rescue" pathways
- FLT3, CSF1R, ERK, AKT, BTK and AURK as key pathways to cripple

"Whac-a-mole" Concept

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

CG'806: Only Known Pan-FLT3 Inhibitor for AML And, Targets Additional Rescue Pathways

CG'806 more potent FLT3-ITD inhibition relative to competitors

Drug	IC_{50} (nM)	
	FLT3-WT	FLT3-ITD
CG'806[1]	8	**0.8**
Midostaurin[2]	11	11
Quizartinib[2]	4	8.8

CG'806 retains potency against all tested forms of FLT3:

	Kd (nM)
- FLT3-WT	0.2 nM
- FLT3-ITD	3.1 nM
- FLT3-D835Y (TKD Mutant)	4.2 nM
- FLT3-ITD/F691L (GK Mutant)	15 nM

CG'806 Differentiates as "More Than Just a FLT3 Inhibitor"

- Potent inhibitor of all forms of FLT3 operative in AML, plus……
- Potent inhibitor of other oncogenic kinases/pathways operative in AML, including CSF1R (FMS), BTK, AURK, H3S10, ERK Pathway, AKT Pathway

(1) Reaction Biology Corp.
(2) Blood. 2009 Oct 1; 114(14): 2984–2992

CG'806 Induced Rapid and Sustained Tumor Eradication in Mouse Model of AML

ORAL

MV4-11 (FLT3-ITD Driven AML) in Balb/c Mice

EFFICACY

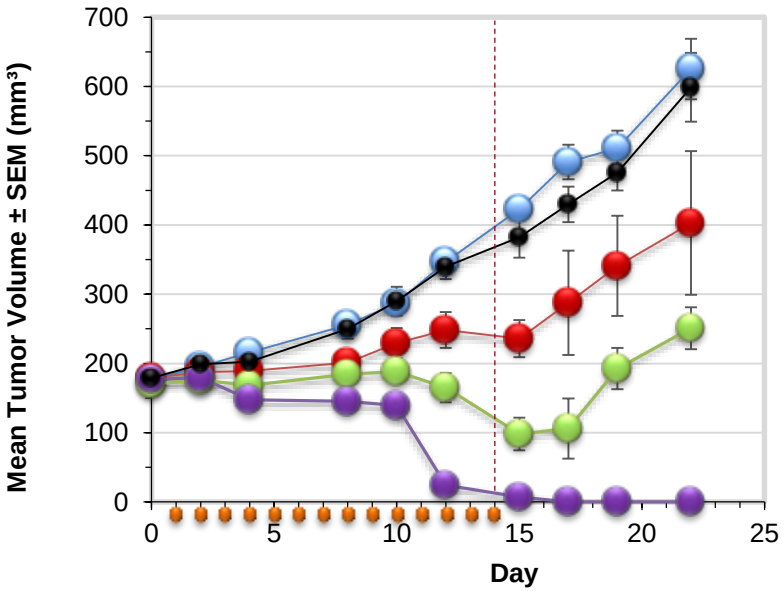
14 DAYS

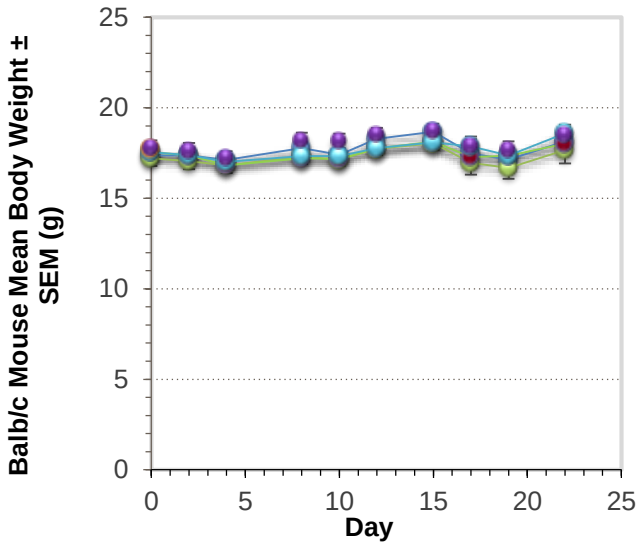
Treated Orally, Once Daily (QD) Dosing for 14 Days

SAFETY

Complete Tumor Elimination with

No Observed Toxicity at Doses up to 1000 mg/kg/day (Micronized/SLS)



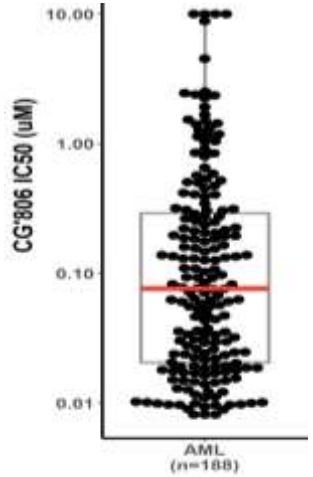


CG'806 Exerts Broad and Superior Potency Against AML Patient Samples

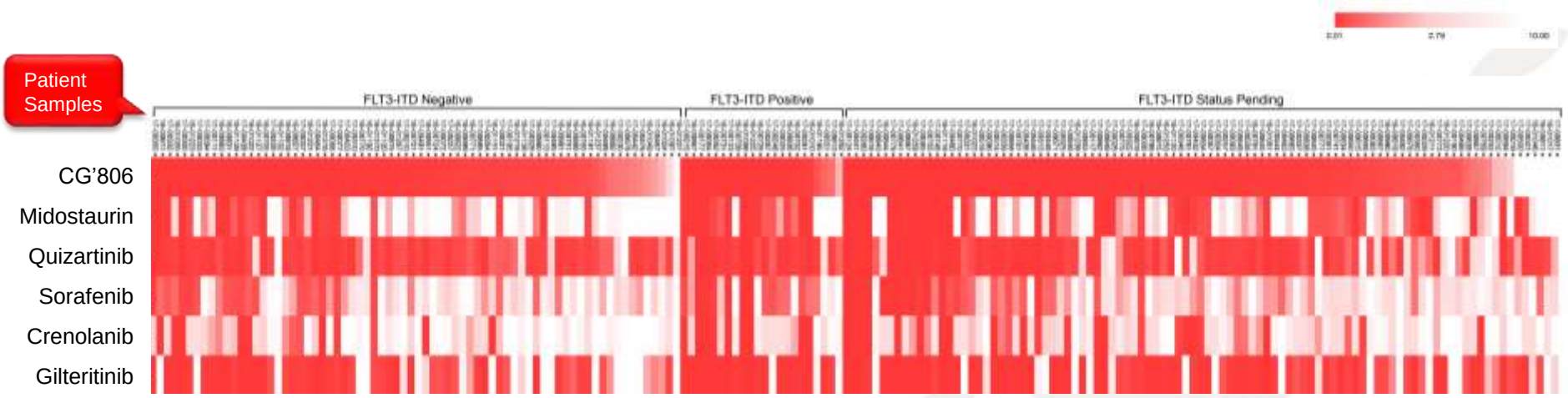
188 AML Patient Samples: Median IC_{50} = 76nM

Ex Vivo Drug Sensitivity Assay

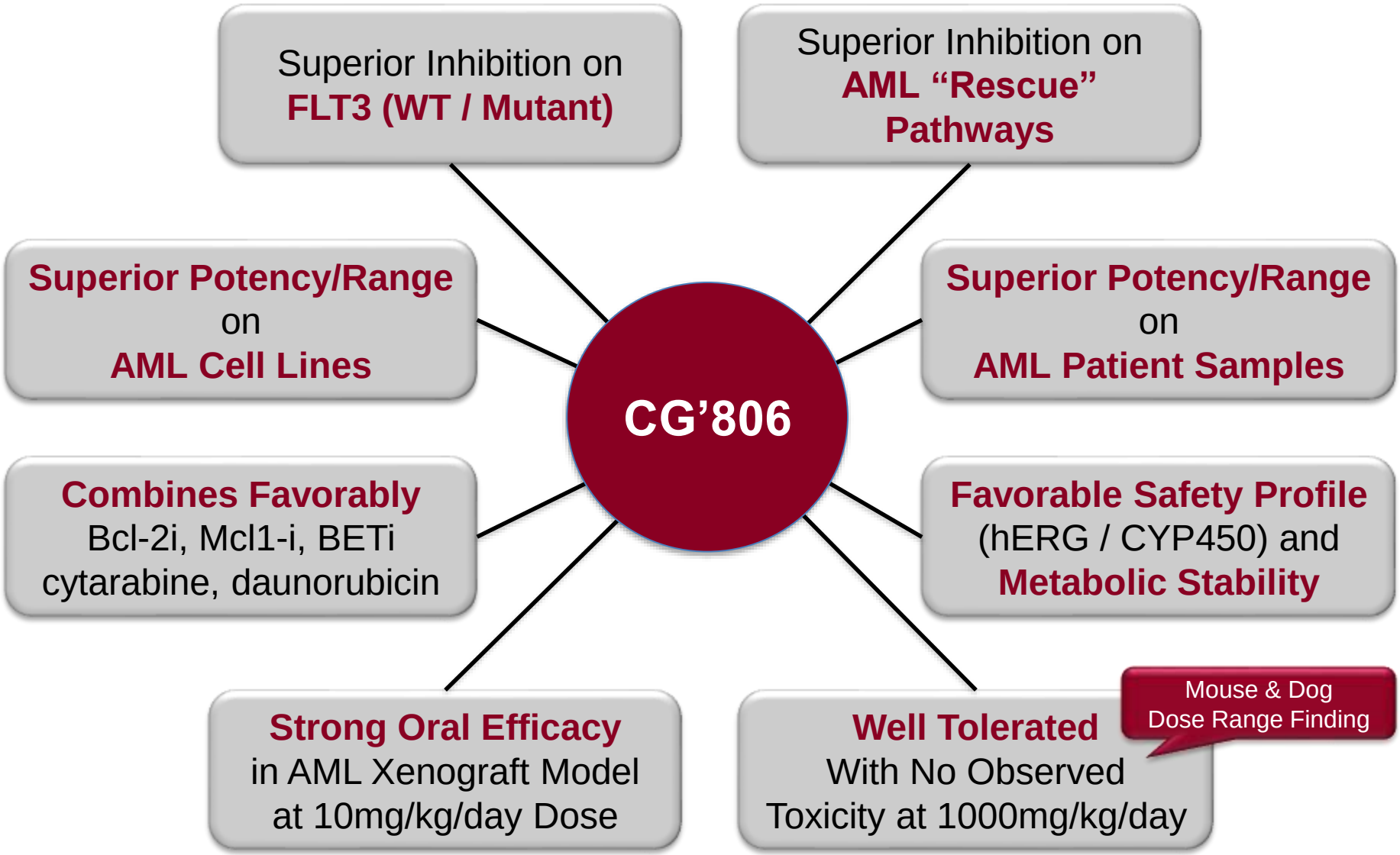
- Inhibitor activity was assessed by an ex vivo assay to determine sensitivities of freshly isolated primary patient samples to CG'806 and other FLT3 inhibitors.
- Cell viability was assessed after 72-hour culture using a tetrazolium-based MTS assay and IC_{50} values calculated as a measure of drug sensitivity. Under the culture conditions used here, the cells retain viability (>90%), but do not proliferate.



Heatmap of CG'806 Versus Other FLT3 Inhibitors on Primary AML Samples



CG'806 Oral, Small Molecule, Multi-Cluster Inhibitor: Potential Best-In-Class Agent for AML



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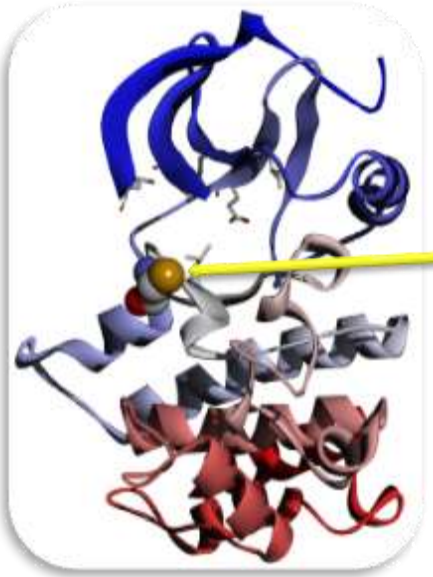
CG'806	BTK-WT/C481S	B Cell Cancers (CLL/MCL/DLBCL)				
APTO-253	c-Myc	AML / MDS				
APL-581	BRD/Kinase	Hematologic Malignancies				

Medical Need for Next Generation BTK Inhibitor



Overexpressed BTK Drives Signaling in B Cell Malignancies

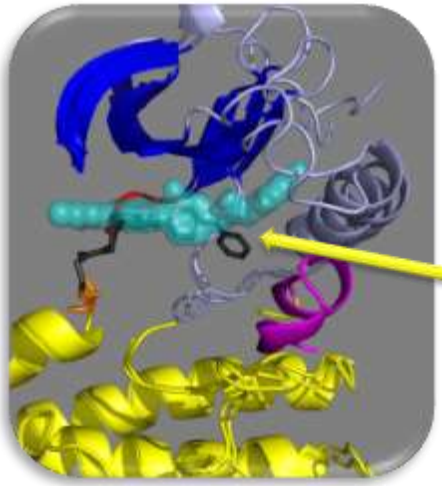
- CLL, MCL, DLBCL

Ibrutinib (Imbruvica®) is Current Standard of Care

- $Multi-billion WW sales in 2017 (Bloomberg)

Ibrutinib Shortcomings – Patients Discontinuing

- 51% CLL Patients: Discontinue treatment with ibrutinib after 3.4yrs (1)
- 5-10% Patients: Resistant (C481S) to ibrutinib Covalent inhibitor
- 40-45% Patients: Intolerant or refractory to ibrutinib



CG'806 Overcomes Shortcomings of Ibrutinib

- **"Non-covalent inhibitor" of BTK (WT & C481S)**
- **Well tolerated in animal toxicology studies**
- **Inhibits multiple "rescue" kinases/pathways**
- **Plan to treat all patients discontinuing ibrutinib**

(1) Woyach et al. *J Clin Oncol*..; 2017: 35; 1-7

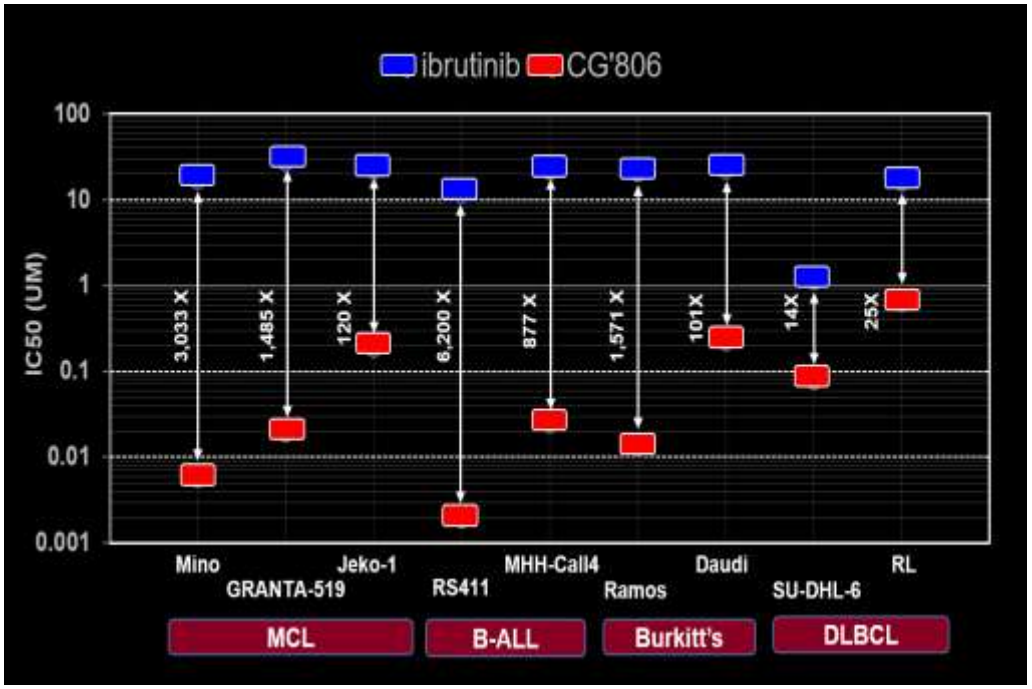
CG'806 More Potent than Ibrutinib in Killing B-Cell Cancer Cell Lines and CLL Patient Samples (Ex Vivo)

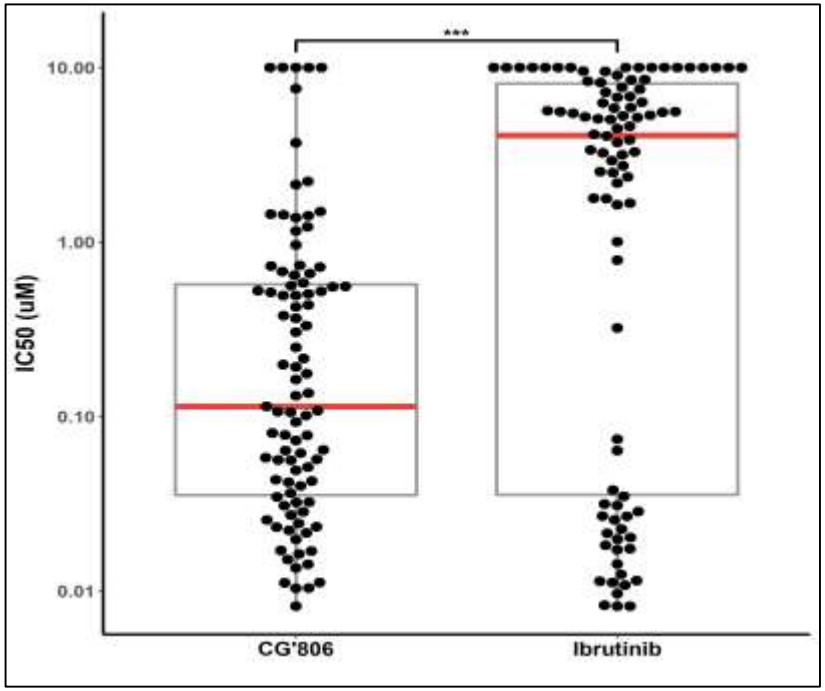
CG'806 kills B-cell cancer cell lines more potently than Ibrutinib

- Range: 14X to 6,200X Greater Potency
- Mean: 1,491X

CG'806 has greater potency than Ibrutinib on CLL patient samples

- Broadly sensitive to CG'806
- Segregate into populations sensitive to or resistant to ibrutinib





CG'806 Profile to Become Best in Class Non-Covalent BTK inhibitor

CG'806 and Other Non-Covalent Agents
Potent Inhibitors of Wild Type and Mutant C481S-BTK, but not EGFR

			BTK IC_{50} (nM)		Key Off-Targets	
Agent	**Company**	**Binding**	**WT**	**C481S**	**ITK**	**EGFR**
Ibrutinib[1]	Abbvie	Covalent	0.5	R	10.7	5.6
Acalabrutinib[2]	AZ / Acerta	Covalent	5.1	R	>1000	>1000
CG'806	**APTOSE**	**Non-Covalent**	**5.0**	**2.5**	**4.3**	**>1000**
SNS-062[3]	Sunesis	Non-Covalent	4.6	1.1	14	>1000
ARQ 531[4]	ArQule	Non-Covalent	4.2	NA	>1000	290

CG'806 Differentiates: More than Just Non-covalent BTK Inhibitor

- **Inhibits cluster of oncogenic kinases operative in B cell malignancies**
- **Results in >1000x more potent than ibrutinib in killing B-cell cancer cells**
- **Well Tolerated: Does NOT inhibit TEC, EGFR or ErbB2/4 kinases associated with bleeding disorders, rash/diarrhea and atrial fibrillation, respectively**

References

(1) Proc Natl Acad Sci U S A. 2010 Jul 20;107(29):13075-80.

(2) N Engl J Med. 2016 Jan 28;374(4):323-32

(3) Sunesis Corporate Presentation, September 2017

(4) Eathiraj et al, Pan Pacific Lymphoma Conference 2016

CG'806: Next Steps

- ✓ **Completed manufacture of GLP API and formulation of drug product**
- ✓ **Completed dose range finding studies for pre-IND meeting with FDA**
- ➢ **Next: IND-enabling GLP toxicology studies in two species**
- ➢ **Target IND Submission to FDA during 2018**
- ➢ **Plan clinical trials to begin in late 2018**
- ➢ **Clinical/commercial development plans**
  - Acute Myeloid Leukemia (AML)
  - B Cell Malignancies (MCL, CLL and DLBCL)

Aptose Program Pipeline

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CG'806	Pan-FLT3	AML				
CG'806	BTK-WT/C481S	B Cell Cancers (CLL/MCL/DLBCL)				
APTO-253	c-Myc	AML / MDS				
APL-581	BRD/Kinase	Hematologic Malignancies				

APTO-253

First-in-Class Inhibitor of MYC Expression

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APTO-253 Inhibits Expression of MYC

MYC Oncogene — Regulates cell growth, proliferation, apoptosis

- Overexpressed in hematologic cancers, including AML
- Notoriously difficult to inhibit MYC expression/signaling

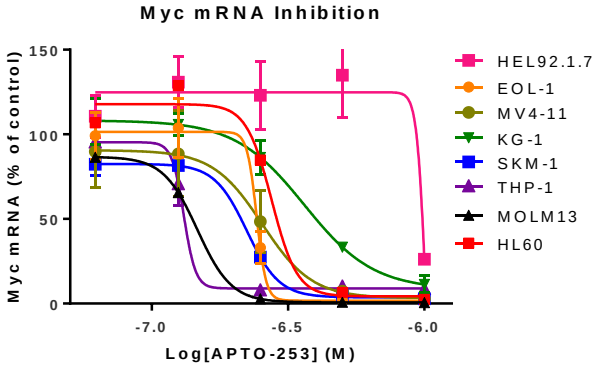

LETTER

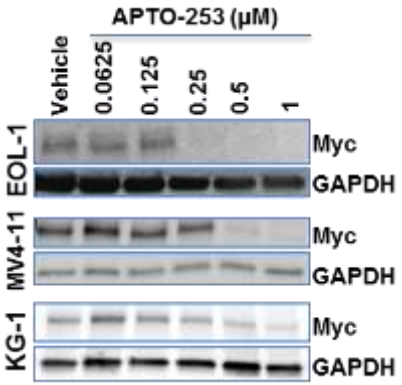
A Myc enhancer cluster regulates normal and leukaemic haematopoietic stem cell hierarchies

25 JANUARY 2018 | VOL 553 | NATURE | 515

APTO-253 — Entirely new class of small molecule MYC inhibitor

- Inhibits MYC gene expression (mRNA) and depletes AML cells of MYC protein





APTO-253 — Value as Fresh Approach to Inhibit MYC

- APTO-253 is safe, combines well with other agents, and ***not*** **myelosuppressive**[1]

(1) ASH 2016 Poster #1716

Steps / Plans to Return APTO-253 to Clinic

✓ **Conducting Phase 1b in Patients with AML**	✓ Observed favorable PK and safety findings ✓ Completed three dose cohorts and entered fourth cohort ✓ **Observed solubility deficiency with drug formulation – Suspended dosing/trial**
✓ **Solved Deficiency with Original Drug Product Formulation**	✓ **Optimized synthesis of API** drug substance ✓ **Optimized liquid formulation** for the drug product ✓ **Optimized drug product manufacturing** processes ✓ **Completed Engineering Run** of drug product manufacture ✓ **Engineering Vials passed formal stability testing**
✓ **Completed Manufacture of "GMP" Drug Product Intended for Clinic**	○ **Performing** stability, sterility, mock infusion studies ○ **Performing** animal bridging studies
Seek to Return APTO-253 to Clinical Trial	○ Plan to present CMC findings to FDA ○ Seek "release of clinical hold" from FDA ○ Seek to re-initiate dosing of patients with AML / hr-MDS

Aptose Executive Summary

Developing Highly Differentiated / Targeted Drugs for Blood Cancers

CG'806 First-in-Class Pan-FLT3 / Pan-BTK Multi-Cluster Inhibitor

- FLT3 inhibitor to treat sizable segment of AML population
- BTK inhibitor to treat B cell cancer patients resistant to / discontinuing Imbruvica
- IND and clinical trials in AML and CLL targeted to begin in 2018

APTO-253 First-in-Class MYC Inhibitor in Phase Ib for AML

- Inhibits MYC oncogene expression without toxicity to normal bone marrow
- Resolved formulation and manufacturing setbacks with drug product
- Manufactured new drug product for return to the clinic

Announced Licensing Deal for Our Dual BET/Kinase Program

Strong Leadership and KOL Support

Strengthened Financial Foundation

- Cash runway >12 months

Thank You!

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