UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

Form 6-K

REPORT OF FOREIGN PRIVATE ISSUER PURSUANT TO RULE 13a-16 OR 15d-16 OF THE SECURITIES EXCHANGE ACT OF 1934

For the month of July 2018

Commission File Number: 001-32001

Aptose Biosciences Inc.
(Translation of registrant's name into English)

251 Consumers Road, Suite 1105
Toronto, Ontario M2J 4R3
Canada
(*Address of principal executive offices*)

Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F.
Form 20-F ☐ Form 40-F ☒

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(1) ☐

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(7) ☐

INCORPORATION BY REFERENCE

N/A

DOCUMENTS FILED AS PART OF THIS FORM 6-K

See Exhibit Index hereto.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

Aptose Biosciences Inc.

Date: July 9, 2018

By: /s/ Gregory Chow
Name: Gregory Chow
Title: Senior Vice President and Chief Financial Officer

EXHIBIT LIST

99.1	Corporate Presentation

Exhibit 99.1

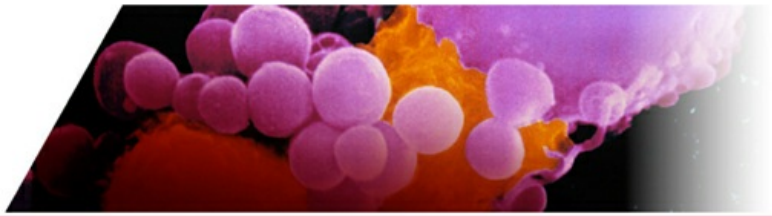
CORPORATE PRESENTATION

JULY 09, 2018

APTOSE BIOSCIENCES

NASDAQ: APTO TSX: APS

APTOSE BIOSCIENCES



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 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. (6/30/2018)	Basic: 34.4 MM; FD: 38.8 MM *No Warrants / No Preferred Stock / No Debt*
3 Month ADTV	~400,000 Shares
Market Cap (6/30/2018)	~$130 Million
Cash Position (3/31/18)	US$16.2 Million / CA$20.9 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

APTO-253 : MYC Inhibitor

FDA Orphan Drug Designation in AML

- Only clinical stage agent directly targeting MYC oncogene
- Currently at Phase Ib stage for acute myeloid leukemia (AML)

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

FDA Orphan Drug Designation in AML

- Potent inhibitor of wild type & all mutant FLT3 >> AML
- Potent inhibitor of wild type & all mutant BTK >> B-cell Cancers
- IND planned 2018 to support AML and B-cell cancer FIH trials

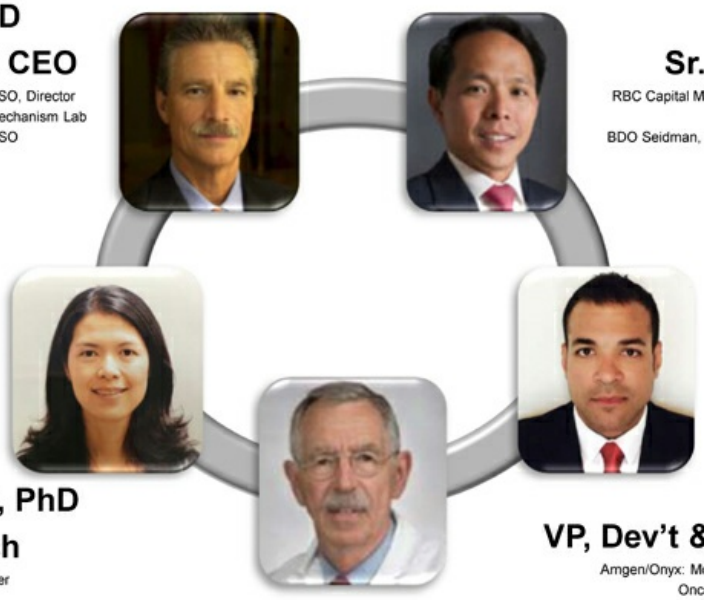
$1B+ commercial opportunity in lead indications (AML and CLL)

Strong leadership team of industry, financial and clinical research professionals

Aptose Leadership Team – See *www.aptose.com*

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

Aptose Program Pipeline

DRUG	TARGET	INDICATION	DISCOVERY	PRE-CLINICAL	PHASE 1	PHASE 2
APTO-253	MYC	AML / MDS				
CG-806	Pan-FLT3	AML				
CG-806	Pan-BTK (WT/C481S) Non-Covalent	B Cell Cancers (CLL/MCL/DLBCL)				
APL-581	BRD/Kinase	Hematologic Malignancies (Program Partnered with Ohm/LALS)				

APTO-253

Phase 1b Stage

First-in-Class Inhibitor of MYC Expression

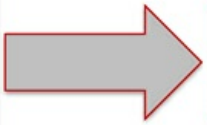
APTOSE BIOSCIENCES

APTO-253 Phase 1b Clinical Development Plan

Phase 1b Dose Escalation Trial
Dosing Planned to Commence Shortly

R/R AML and High Risk MDS

- Planned up to 20 Patients
- 1º Endpts: MTD, DLT, RP2D
- 2º Endpts: PK, Biomarkers, Efficacy, Transfusions



Single Agent Expansions
AML – Up to 15 Patients
ORR, Efficacy, Biomarkers, Safety

Single Agent Expansions
MDS – Up to 15 Patients
ORR, Efficacy, Biomarkers, Safety

Drug Combination Trials
Selection/Design Underway

- Patient Populations: R/R-AML Aggressive Cancer of Blood/Bone Marrow and hr-MDS
- Dosing Levels Planned: 20 (1pt), 40 (1pt), 66 (3x3pts), 100, 140, 180, 220mg/m^2
- Drug Product Employs Newly Modified Formulation/Manufacturing Procedures
- Dosing Schedule: Day 1 of Each Week on 28-day Cycle
- Clinical Sites: Up to 15 Elite Sites Planned to Participate
- Potential Other Indications: B Cell Malignancies

Note: Phase 1b expansion cohorts and Phase 2 trials contingent on Phase 1b outcomes

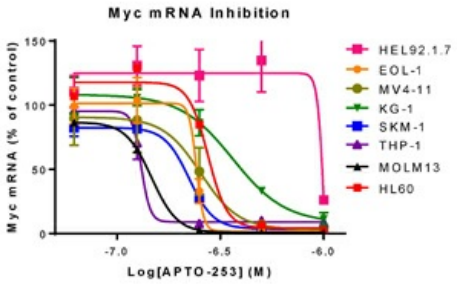
APTO-253 Inhibits Expression of MYC

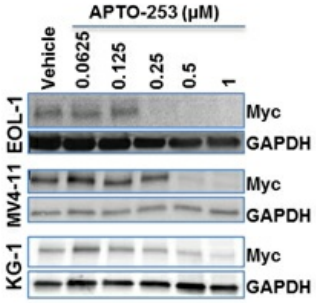
MYC Oncogene — Regulates cell growth, proliferation, apoptosis

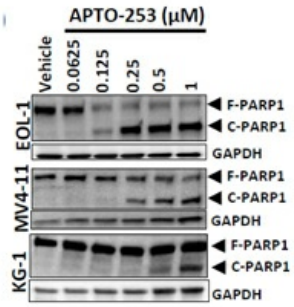
- Dysregulated in numerous hematologic cancers, especially AML
- Notoriously difficult to inhibit MYC expression/signaling

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

- Targets a regulatory motif in the promoter region of MYC gene (NOT the MYC protein)
- Inhibits MYC gene expression (mRNA) → depletes cells of MYC protein → induces apoptosis







APTO-253 — Value as Fresh Approach to Inhibit MYC

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

(1) ASH 2016 Poster #1716

APTO-253 Mechanism & Therapeutic Indications: AACR Publications and 2018 AACR Presentation

APTO-253 stabilizes G-quadruplex DNA, inhibits MYC expression and induces DNA damage in acute myeloid leukemia cells

Andrea Local, Hongying Zhang, Khalid D Benbatoul, Peter Folger, Xia Sheng, Cheng-Yu Tsai, Stephen B. Howell, and William G. Rice

AACR Journal: *Molecular Cancer Therapeutics*, June 2018 (Volume 17, Number 6)

Binding/Stabilizing G-quadruplex DNA (G4) motif in MYC promoter silences MYC gene expression

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

Cheng-Yu Tsai, Si Sun, Hongying Zhang, Andrea Local, Yongxuan Su, Larry A Gross, William Rice, and Stephen B. Howell

AACR Journal: *Molecular Cancer Therapeutics*, June 2018 (Volume 17, Number 6)

Binding of G4 can destabilize telomeres and stall replication forks resulting in DNA damage response

Cancer cells harboring BRCA1/2 mutations hypersensitive – new solid tumor path

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

Cheng-Yu Tsai, Si Sun, Hongying Zhang, Andrea Local, William Rice, and Stephen B. Howell

2018 AACR Abstract and Poster Presentation

Aptose Program Pipeline

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CG-806

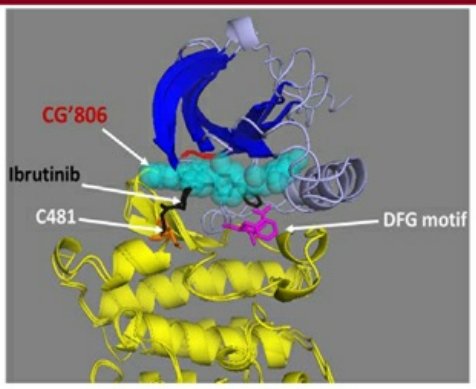
First-in-Class
Pan-FLT3 / Pan-BTK

"Multi-Cluster" Kinase Inhibitor

APTOSE
BIOSCIENCES

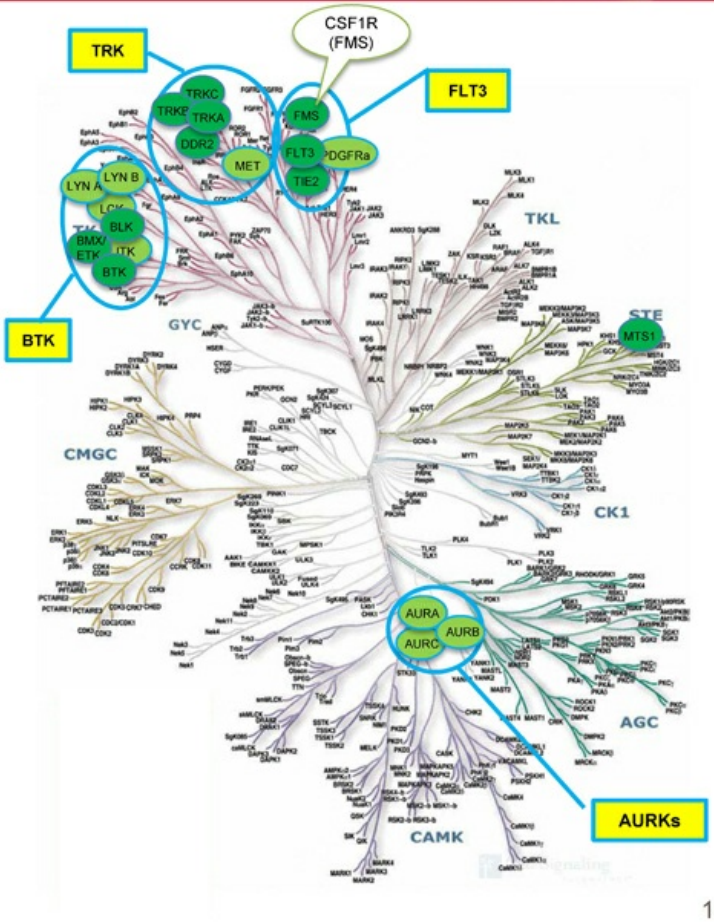
CG-806 Selectively and Potently Targets "Clusters of Related Kinases"

- **NOT** a "Dirty Kinase Inhibitor"
- **Targets Clusters of Oncogenic Kinases** operative in **Cancers Derived from Bone Marrow**
- Selectivity for clusters linked to rigidity, functionalization, and "unique binding modes"



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

Atypical Type II Binding Mode



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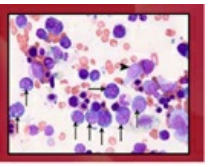
APL-581	BRD/Kinase	Hematologic Malignancies (Program Partnered with Ohm/LALS)				

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

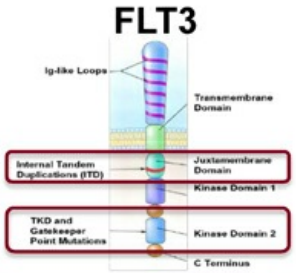


Aggressive & Heterogeneous Cancer of Blood and Bone Marrow

- **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; Quizartinib & 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 Plus, Targets Additional Rescue Pathways

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

	IC_{50} (nM)	
Drug	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

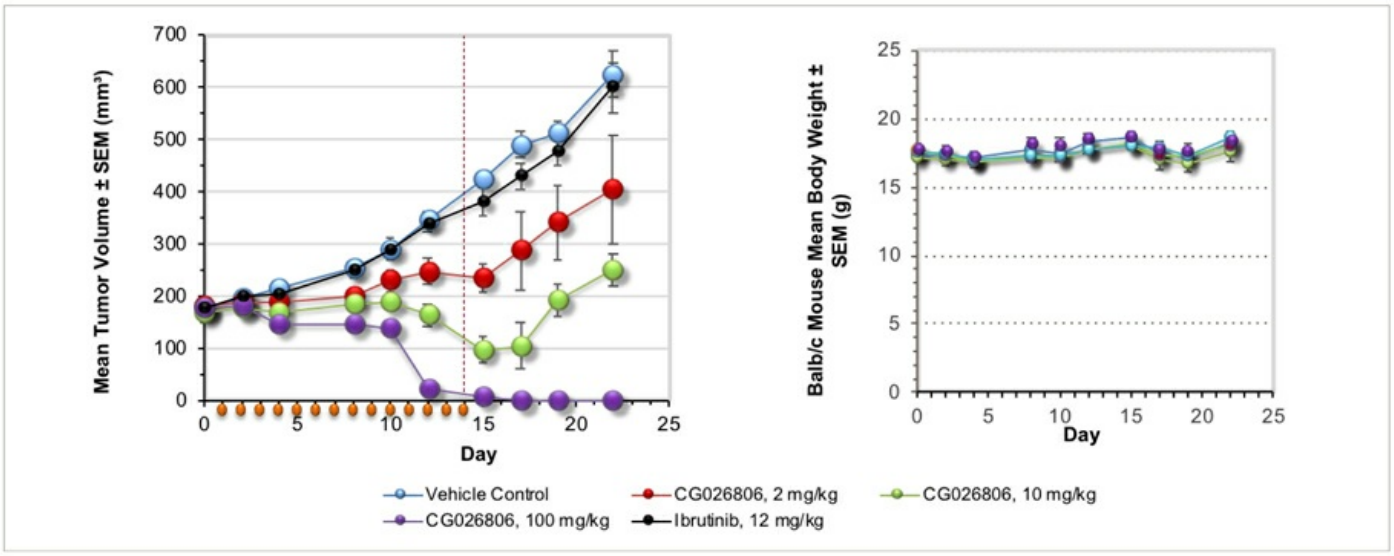
MV4-11 (FLT3-ITD Driven AML) in Balb/c Mice

EFFICACY

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

SAFETY

Complete Tumor Elimination with No Observed Toxicity at These Doses



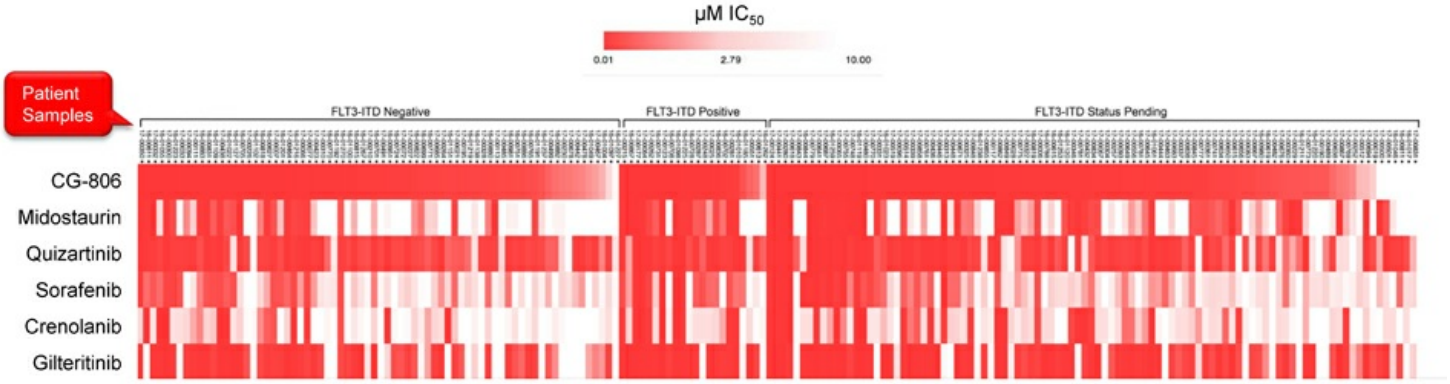
CG-806 Exerts Broad and Superior Potency Against AML Patient Samples

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

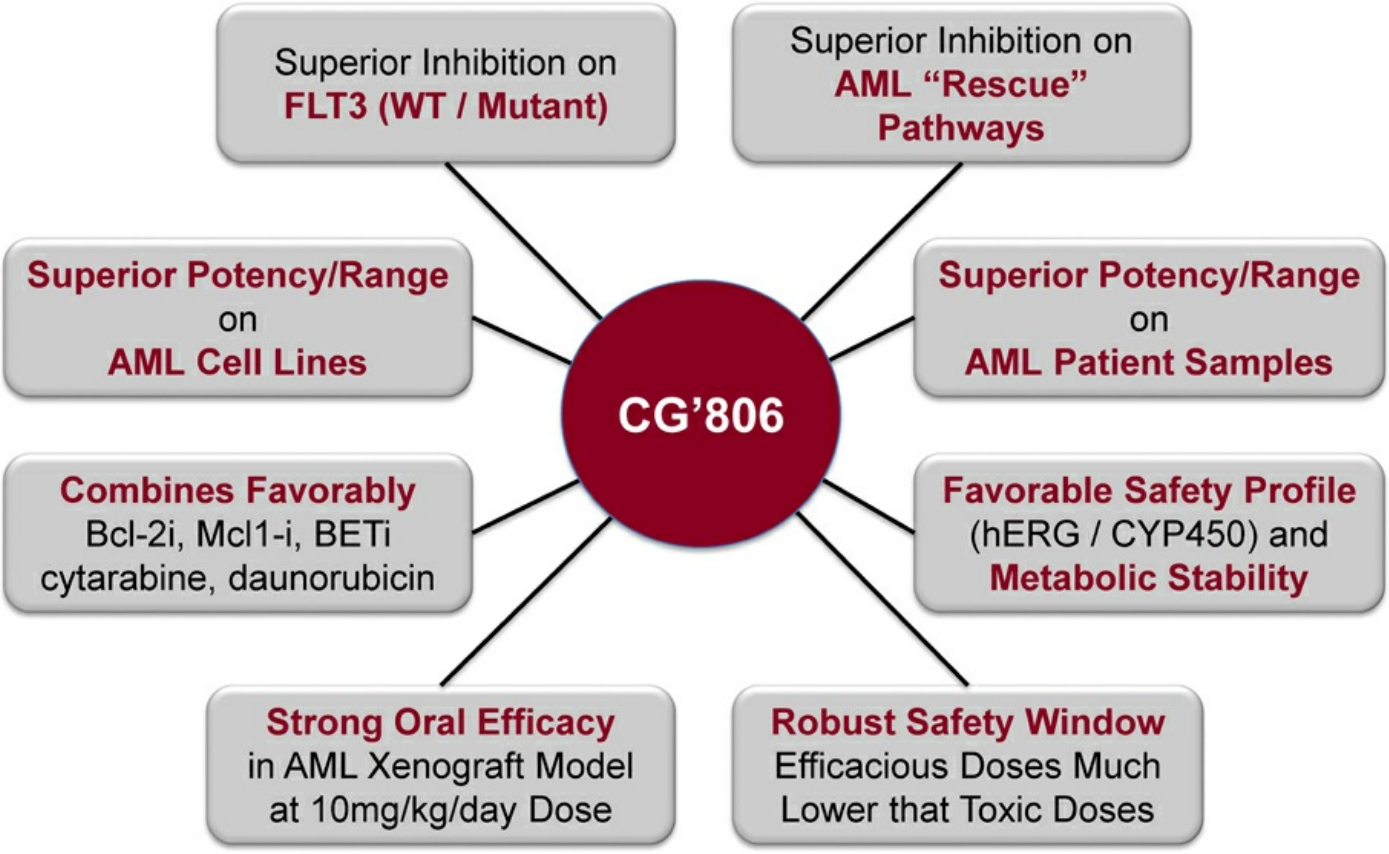
Ex Vivo Drug Sensitivity Assay

- Inhibitors assessed by ex vivo assay to determine sensitivities of **fresh bone marrow 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



Aptose Program Pipeline

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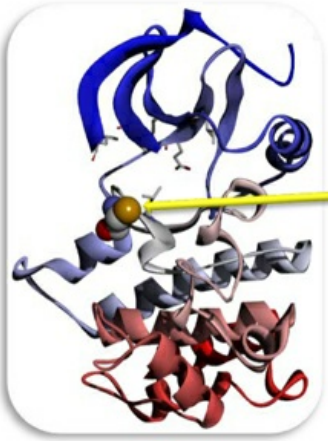
Medical Need for Next Generation BTK Inhibitor



Overexpressed BTK Drives Signaling in B Cell Malignancies

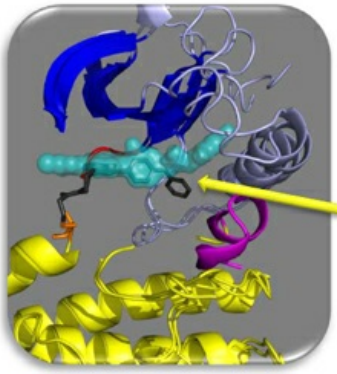
- CLL, MCL, DLBCL

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 Potently Inhibits Kinases in the BTK Cluster but NOT Kinases Related to Ibrutinib Side Effects

CG-806 Inhibits Kinases of BTK Cluster

Kinase	CG-806 IC50 (nM)
BTK-WT	5.0
BTK-C481S	2.5
BTK-P190K	6.5
BTK-E41K	14.5
BLK	0.7
ITK	4.3
LCK	0.7
LYNA	2.0
LYNB	6.9
BMX/ETK	14.5

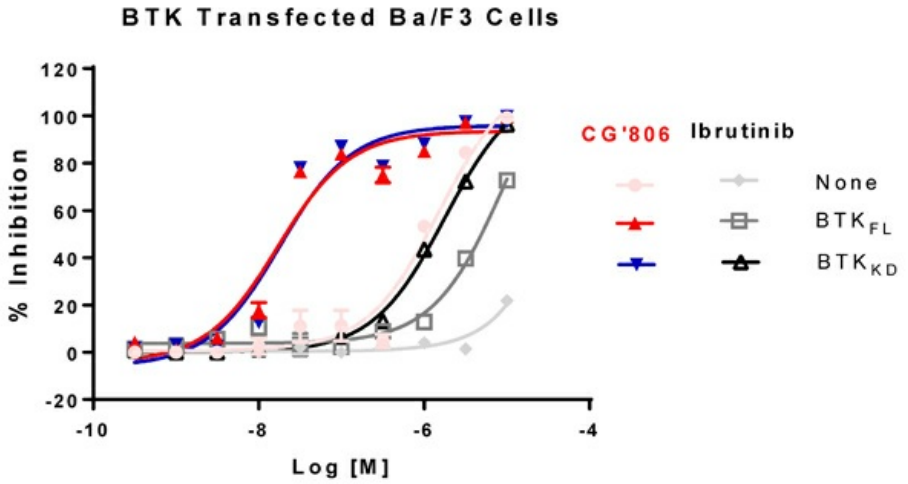
B Cell Cancer Rescue Pathways Potently Inhibited by CG-806

Kinase / Pathway
FLT3-WT / ITD / GK & TKD Mutants
CSF1R Receptor
AKT Pathway
ERK Pathway
AURK - A / B / C
H3S10 Epigenetic

CG-806 Does Not Inhibit Kinases Related to Ibrutinib Side Effects

	TEC	EGFR	ErbB2	ErbB4
CG-806	>1,000	>1,000	>1,000	>1,000
Ibrutinib	78	5.6	9.4	NA

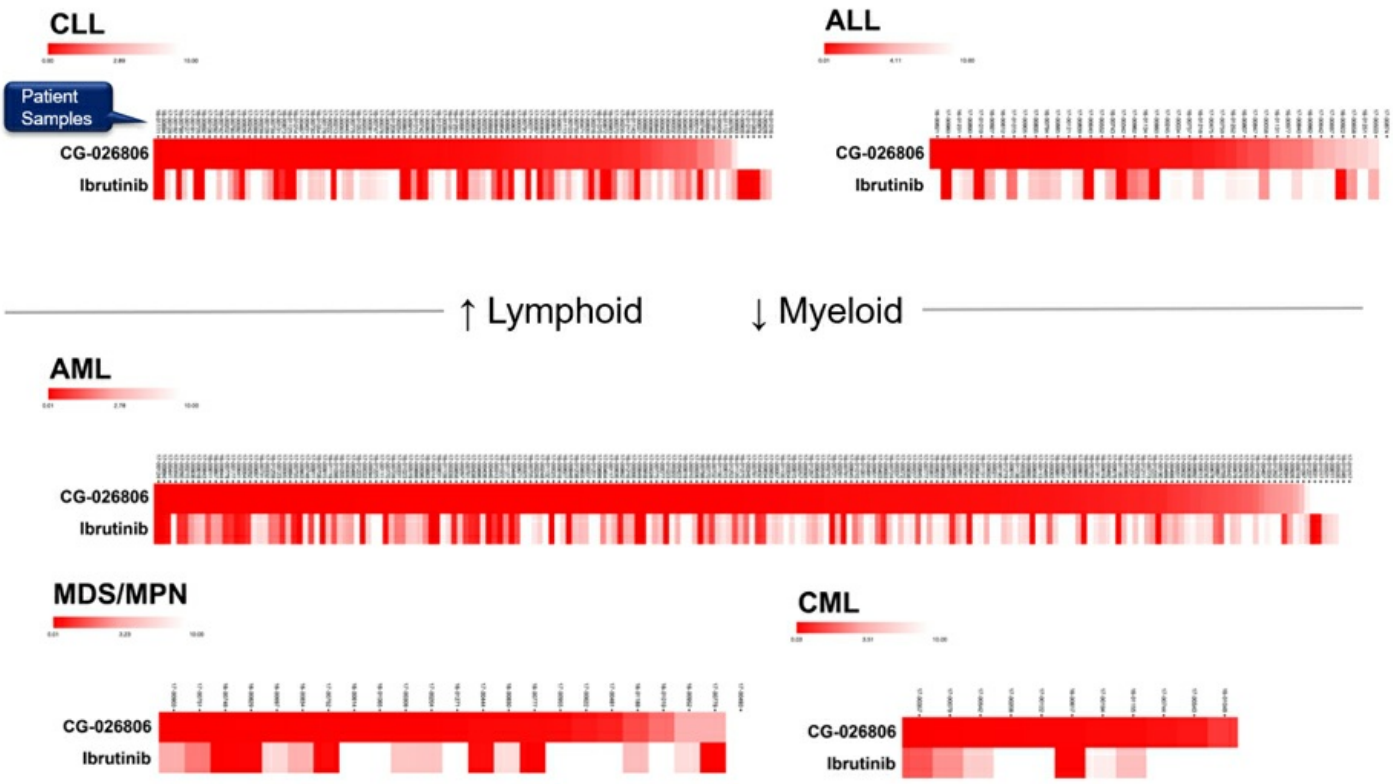
CG-806 Killing Potency Superior to Ibrutinib in Ba/F3 Cell Transfected with BTK



IC50 (nM)	CG'806	Ibrutinib
BTK_{FL}	22.8	4555.0
BTK_{KD}	23.2	1295.0
None	1001.0	> 10,000

Ba/F3 cells are murine lymphoma cells dependent upon exogenous IL-3 for survival. Isogenic Ba/F3 cells transfected with specific oncogenic kinases can be constructed so the only difference in the cells is the form of a particular kinase that is expressed and upon which cell survival is dependent (in the absence of IL-3). Ba/F3 cells were transfected with WT (Full Length of Kinase Domain only) forms of BTK. CG-806 effectiveness against isogenic murine Ba/F3 cells was compared to ibrutinib. CG-806 killed Ba/F3 cells transfected with BTK WT full length (WT-FL) or the WT kinase domain (WT-KD) at equal potency, while ibrutinib required higher concentrations to kill the cells. Note that CG-806 is more effective than ibrutinib at killing the non-transfected Ba/F3 lymphoma cells in the presence of IL-3.

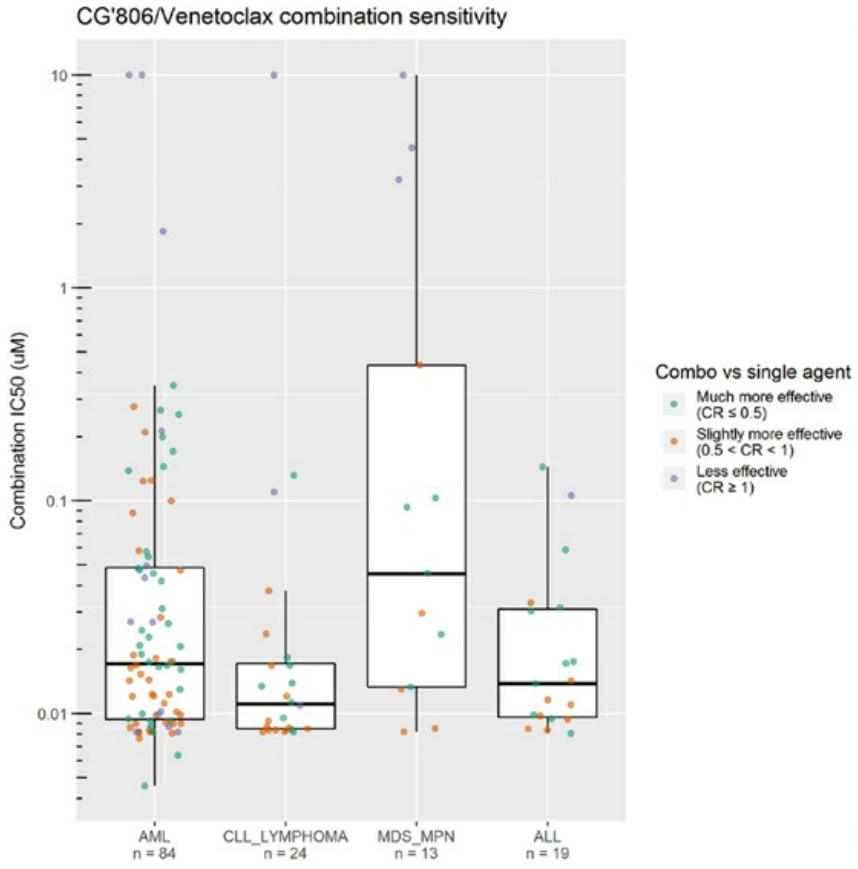
CG-806 Exerts Broad and Superior Killing Potency Compared to Ibrutinib Against Patient Samples



Presented at European Hematology Association 2018

CG-806, A NON-COVALENT PAN-FLT3/PAN-BTK INHIBITOR, EXHIBITS UNIQUE BINDING TO WILD TYPE AND C481S MUTANT BTK AND GREATER POTENCY THAN IBRUTINIB AGAINST MALIGNANT B CELLS Authors: Zhang, Local, Benbatoul, Folger, Sheng, Kurtz, Tyner, Howell, Rice

CG-806 plus Venetoclax : Enhanced Killing of Bone Marrow Samples from Patients with Hematologic Malignancies



- CG-806 and Venetoclax individually are highly active cancer agents
- CG-806 + Venetoclax Combination Studies
  - Enhanced ex vivo killing of patient bone marrow cells in most samples (green and orange spheres)
- Suggests the combination of CG-806 and Venetoclax may become a preferred drug combination for patients with AML, MDS/MPN, CLL, ALL and other hematologic malignancies

CG-806 Differentiates from Other BTK Inhibitors (Covalent and Non-Covalent)

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 is "More than Just a Non-covalent BTK Inhibitor"

- **Inhibits clusters of oncogenic kinases operative in B cell malignancies**
- **Results in greater potency than ibrutinib in killing B-cell cancer cells**
- **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: Steps Toward the Clinic

- ✓ **Completed manufacture of GLP API and formulation of drug product**
- ✓ **Completed dose range finding studies for pre-IND meeting with FDA**
- ➢ **IND-enabling GLP toxicology studies in two species ongoing**
- ➢ **Target IND Submission to FDA during 2018**
- ➢ **Planned Clinical Trials Following IND**
  - Acute Myeloid Leukemia (AML)
  - B Cell Malignancies (MCL, CLL and DLBCL)

Aptose Executive Summary

Developing Highly Differentiated / Targeted Drugs for Blood Cancers

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

- Expect to commence screening and dosing of R/R-AML / hr-MDS patients soon
- Potential to expand into B cell malignancy patients (role of MYC)
- New modified formulation creates potential for new IP

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
- Exercised Exclusive License Agreement and Captured China Rights in 2018
- Expect to file IND for AML and CLL clinical trials in 2018

Announced Licensing Deal for Our Dual BET/Kinase Program

Strong Leadership and KOL Support

Strengthened Financial Foundation

- Cash runway >12 months

Thank You!

APTOSE BIOSCIENCES