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Aptose Corporate Snapshot

Clinical stage company employing a mechanism-driven approach to deliver safer, targeted, first-in-class cancer drugs

<table>
<thead>
<tr>
<th>Public Company</th>
<th>NASDAQ: APTO / TSX: APS</th>
</tr>
</thead>
</table>
| Shares Outstanding (8/31/2018) | Basic: 34.7 MM; FD: 39.3 MM  
No Warrants / No Preferred Stock / No Debt |
| 3 Month ADTV | ~250,000 Shares |
| Market Cap (9/28/2018) | ~$100 Million |
| Cash Position (6/30/18) | US$18.5 Million |
| Cash Runway | > 12 Months |
| Executive Headquarters & Research Laboratories | San Diego, CA |
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
- 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
- Potent inhibitor of wild type & all mutant FLT3 >> AML
- Potent inhibitor of wild type & all mutant BTK >> B-cell Cancers
- Collecting IND data 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 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. 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

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

Scientific Advisory Board Populated with KOLs – Domain Expertise
APTO-253
Phase 1b Stage
Small Molecule
MYC Inhibitor
APTO-253 Inhibits Expression of MYC

MYC Oncogene  Regulates cell growth, proliferation and apoptosis
- Dysregulated in numerous hematologic cancers, especially AML

Upregulated MYC  Reprograms signaling pathways to support survival
- Normal Cell: Reprograms signaling pathways (survival) to transform cells to malignant
- Cancer Cell: Reprograms signaling pathways (rescue) to bypass a pathway targeted by a drug

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
- Fresh approach to MYC inhibition – Not Myelosuppressive
APTO-253 Phase 1b Dose Escalating Clinical Trial Underway for Treatment of R/R-AML and hr-MDS

R/R AML and High Risk MDS
- Planned up to 20 Patients
- 1º Endpts: MTD, DLT, RP2D
- 2º Endpts: PK, Biomarkers, Efficacy, Transfusions

Drug Product Employs Newly Modified Formulation
Dosing Schedule: Day 1 of Each Week on 28-day Cycle
Clinical Sites: Up to 15 Elite Sites Planned to Participate
Dosing Levels Planned: 20 (1pt), 40 (1pt), 66 (3x3pts), 100, 140, 180, 220mg/m²
Anticipate preliminary updates late 2018 and completion of trial during 2019

Note: Phase 1b expansion cohorts and Phase 2 trials contingent on Phase 1b outcomes
CG-806
Approaching IND
First-in-Class
Pan-FLT3 / Pan-BTK Inhibitor
“Multi-Cluster” Kinase Inhibitor
CG-806 Selectively Targets “Clusters of Related Kinases” and Inhibits Driver & Bypass Pathways Simultaneously

<table>
<thead>
<tr>
<th>FLT3 Kinases</th>
<th>CG-806 Kd (nM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FLT3-WT</td>
<td>0.24</td>
</tr>
<tr>
<td>FLT3-ITD</td>
<td>0.31</td>
</tr>
<tr>
<td>FLT3-D835Y</td>
<td>4.2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>BTK Kinases</th>
<th>CG-806 IC₅₀ (nM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BTK-WT</td>
<td>8.4</td>
</tr>
<tr>
<td>BTK-C481S</td>
<td>2.5</td>
</tr>
<tr>
<td>TEC</td>
<td>&gt; 1000</td>
</tr>
<tr>
<td>EGFR</td>
<td>&gt; 1000</td>
</tr>
<tr>
<td>ErbB2</td>
<td>&gt; 1000</td>
</tr>
</tbody>
</table>

√ FLT3 WT / ITD / GK / TKD → AML Driver
√ BTK and BCR Pathway → BCM Driver
√ AKT/mTOR/S6K Pathway
√ ERK Pathway
√ MYC Expression
√ H3S10 Epigenetic
√ Rescue Pathways
CG-806 X-Ray Crystal Structure: Selectivity is Linked to Rigidity, Functionalization, and Binding Modes

X-ray Crystal Structure of CG-806 in BTK WT and C481S mutant. The co-crystal complex of CG-806 bound to BTK- wild type (WT) or -C481S at resolution of 1.84Å and 1.63Å, respectively, revealed a binding mode of an atypical type II inhibitor. The DFG motif occupies an atypical conformation whereby the Phe540 is rotated out of the ATP binding pocket and the Asp539 side chain is tilted to hydrogen bond with CG-806. Moreover, CG-806 interacts with the hinge region and the αC-helix is in a partial out position. No electron density interaction was observed between CG-806 and the C481 residue. A. CG-806 binds BTK WT; B. CG-806 binds BTK C481S mutant.

**CG-806 Selectivity Parameters**

- **Rigid Molecule**
  - Penetrates active site of related kinases
  - Rigidity restricts entry into other kinase active sites

- **Highly Functionalized**
  - Provides extensive H-bonding and Pi stacking
  - Productive binding

- **Unique Binding Modes**
  - Adopts “atypical” Type II binding mode in BTK
  - Type II inhibitor locks kinase in inactive conformation
  - Generally Type II inhibitors show greater selectivity than Type I
CG-806 Targets FLT3 Kinase Driver of Disease and Rescue Pathways Operative in AML

- FLT3 mutations are drivers of AML
- Need for agent that inhibits WT and mutant forms of FLT3 (Pan-FLT3i)
- Need to inhibit Rescue Pathways

CG-806

- Inhibits Kinase Clusters
- Inhibits All FLT3 and BTK
- Inhibits Rescue Pathways
- Robust Safety Profile
CG-806 Inhibits Key Oncogenic Pathways in AML Cells that Utilize Distinct Pathways to Survive

- Potently inhibited FLT3 and BTK clusters of kinases in these cells
- Inhibited key oncogenic pathways (FLT3, BTK/BCR, ERK, MYC, PI3K/AKT/mTOR/S6K, AURK/H3S10) and induced apoptosis
- Each cell uses different pathways for survival …. CG-806 inhibits sufficient number of key pathways (“Whac-a-mole” concept)
CG-806 Exerts Broad & Superior Killing Potency Compared to FLT3i on AML Patient Samples

188 AML Patient Samples: Determined Cell Killing IC$_{50}$

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.

IC$_{50}$ for each drug against each patient sample: Expressed as a Heatmap

CG-806 Targets Driver (FLT3) and Bypass Pathways in AML “More Than Just a FLT3 Inhibitor”
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
Patient information: AML patient (FLT3-ITD) received Sorafenib+Azacitidine Tx and experienced CR after one cycle therapy; relapsed after 3 cycles of treatment and acquired a D835 mutation (now FLT3-ITD+D835)

Patient Derived Xenograft (PDX) Model
- CG’806 reduced leukemia cell burden
- CG-806 reduced splenomegaly

Model implanted with FLT3 ITD+D835 mutated primary AML cells CG’806 (QDx5/wk Orally). hCD45+/mCD45- leukemic cells in peripheral blood were quantitated with flow cytometry.
CG-806 Oral, Small Molecule, Multi-Cluster Inhibitor: Potential Best-In-Class Agent for AML

- Superior Inhibition on FLT3 (WT / Mutant)
- Superior Inhibition on AML “Rescue” Pathways
- Superior Potency/Range on AML Cell Lines
- Superior Potency/Range on AML Patient Samples
- Combines Favorably Bcl-2i, Mcl1-i, BETi, cytarabine, daunorubicin
- Favorable Safety Profile (hERG / CYP450) and Metabolic Stability
- Strong Oral Efficacy in AML Xenograft Model at 10mg/kg/day Dose
- Robust Safety Window Efficacious Doses Much Lower than Toxic Doses
CG-806 Targets BTK Kinase Driver of Disease and Rescue Pathways Operative in B Cell Cancers

- BTK is a driver of B Cell Cancers
- Need for agent that inhibits WT and mutant forms of BTK (Pan-BTKi)
- Need to inhibit Rescue Pathways

Bone Marrow Progenitor Cells

BTK Inhibitors
Covalent or Non-Covalent

Lymphoid Lineage

BTK Driver (kinase)
FLT3, ERK, MYC, AKT, H3S10 Rescue Pwys

B Cell Cancers
CLL, MCL, DLBCL, FL, Others

CG-806
- Inhibits Kinase Clusters
- Inhibits All FLT3 and BTK
- Inhibits Rescue Pathways
- Robust Safety Profile
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

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

IC₅₀ for each drug against each patient sample: Expressed as a Heatmap

CG-806 Targets Driver (BTK) and Bypass Pathways in B Cell Cancers
“More Than Just a BTK Inhibitor”
## CG-806 Oral, Small Molecule, Multi-Cluster Inhibitor: Potential Best-In-Class Agent for B Cell Cancers

### CG-806 Targets Driver (BTK) and Rescue Pathways in B Cell Cancers

<table>
<thead>
<tr>
<th>Agent</th>
<th>Company</th>
<th>Binding</th>
<th>BTK IC&lt;sub&gt;50&lt;/sub&gt; (nM)</th>
<th>Key Off-Targets</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>WT</td>
<td>C481S</td>
<td>ITK</td>
</tr>
<tr>
<td>Ibrutinib&lt;sup&gt;(1)&lt;/sup&gt;</td>
<td>Abbvie</td>
<td>Covalent</td>
<td>0.5</td>
<td>R</td>
</tr>
<tr>
<td>Acalabrutinib&lt;sup&gt;(2)&lt;/sup&gt;</td>
<td>AZ / Acerta</td>
<td>Covalent</td>
<td>5.1</td>
<td>R</td>
</tr>
<tr>
<td>SNS-062&lt;sup&gt;(3)&lt;/sup&gt;</td>
<td>Sunesis</td>
<td>Non-Covalent</td>
<td>4.6</td>
<td>1.1</td>
</tr>
<tr>
<td>ARQ 531&lt;sup&gt;(4)&lt;/sup&gt;</td>
<td>ArQule</td>
<td>Non-Covalent</td>
<td>4.2</td>
<td>NA</td>
</tr>
<tr>
<td>CG-806</td>
<td>APTOSE</td>
<td>Non-Covalent</td>
<td>5.0</td>
<td>2.5</td>
</tr>
</tbody>
</table>

### 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
- Well tolerated and Does NOT inhibit TEC, EGFR or ErbB2/4 kinases associated with bleeding disorders, rash/diarrhea and atrial fibrillation, respectively

### References

(3) Sunesis Corporate Presentation, September 2017
(4) Eathiraj et al, Pan Pacific Lymphoma Conference 2016
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)

- CG-806 + Venetoclax
  - Combination may become a preferred drug combination for patients with AML, MDS/MPN, CLL, ALL and other hematologic malignancies
CG-806: Steps Toward the Clinic

- Completed pre-IND meeting with FDA

- Completed dose range finding studies (rodents and dogs)

- Completed manufacture of GMP drug substance (multiple kg)

- Completed in-life stage of IND-enabling GLP toxicology studies

- Performing studies to support IND Submission to FDA

- 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 dosing of R/R-AML / hr-MDS patients throughout 2H 2018 and into 2019
- 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
- Plan to perform AML and CLL clinical trials throughout 2019

**Announced Licensing Deal for Our Dual BET/Kinase Program**

**Strong Leadership and KOL Support**

**Strengthened Financial Foundation**
- Cash runway >12 months
Thank You!