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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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## Corporate Snapshot

<table>
<thead>
<tr>
<th>Public Company</th>
<th>NASDAQ: APTO / TSX: APS</th>
</tr>
</thead>
</table>
| Shares Outstanding (10/30/2019) | Basic: 55.5 MM  
No Warrants / No Preferred Stock / No Debt |
| 3 Month ADTV | ~430,000 Shares |
| Cash Runway | > 12 Months Operating Capital On Hand and Availability |
| Executive Headquarters & Research Laboratories | San Diego, CA |
| Attractive Investment Profile | • Hematology/Oncology Space  
• Active Calendar of Catalysts  
• Strong Cash Balance, Yet Attractive Valuation  
• Two Targeted Clinical Assets with Upside Potential |
Company Highlights

**APTOSE**

Clinical stage biotech company developing first-in-class targeted agents treating hematologic malignancies life-threatening / orphan diseases

**CG-806 Oral FLT3 / BTK Inhibitor**

Inhibits wild type and all mutant forms of FLT3 : Driver of AML & MDS
Inhibits wild type and all mutant forms of BTK : Driver of CLL & NHL
Precision 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**

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

Oral FLT3 / BTK Inhibitor

Phase 1a/b Stage Ongoing
CG-806 Potently Inhibits Clusters of Kinases That Drive CLL / NHL and AML

- **Mutation Agnostic**
  - Inhibits WT and all mutant FLT3
  - Inhibits WT and all mutant BTK
  - Simultaneously suppresses multiple signaling pathways

- **Robust Safety Profile**
  - NOT a “dirty” kinase inhibitor
  - Avoids kinases that impact safety
  - Promising Safety Profile – No drug related AEs seen to date

- **Inhibits Clusters of Driver Kinases**
  - FLT3 cluster \(\rightarrow\) AML & MDS
  - BTK cluster \(\rightarrow\) CLL & NHL
  - Target patients failing other drugs
CG-806 Suppresses Key Oncogenic Targets and Pathways in Myeloid & Lymphoid Malignancies

Cell Growth and Proliferation
CG-806 for the Treatment of CLL / SLL and 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
- Disrupts signaling among CLL cells and lymphoid microenvironment
- Promotes egress of CLL cells from lymphoid tissues and cells die

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

CG-806 May Overcome Shortcomings of Ibrutinib
- “Non-covalent”: Retains activity against WT and C481S-BTK enzyme
- Well tolerated and inhibits “oncogenic rescue” pathways
- Potently and directly kills CLL and other B-cell cancer cells

CG-806 Non-Covalent Inhibitor Retains Potency Against C481S BTK Mutation

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

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

<table>
<thead>
<tr>
<th>Kinase</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>
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</table>

<table>
<thead>
<tr>
<th>IC₅₀ (nM)</th>
<th>TEC</th>
<th>EGFR</th>
<th>ErbB2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ibrutinib</td>
<td>78</td>
<td>5.6</td>
<td>9.4</td>
</tr>
<tr>
<td>CG-806</td>
<td>&gt;1,000</td>
<td>&gt;1,000</td>
<td>&gt;1,000</td>
</tr>
</tbody>
</table>
CG-806 In Vitro Inhibitory Curves for Wild Type and Mutant Forms of BTK

**CG-806 Potent (IC$_{50}$) BTK WT/Mutants**

<table>
<thead>
<tr>
<th>BTK Enzymes</th>
<th>CG-806 IC$_{50}$ (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>BTK-P190K</td>
<td>6.6</td>
</tr>
</tbody>
</table>

**Compound IC50 Data for BTK**

**Compound IC50 Data for BTK (C481S)**

**Compound IC50 Data for BTK (P190K)**
CG-806 Exerts Broad & Superior Killing Potency Compared to Ibrutinib on Patient Samples

- OHSU Measured the Ability of CG-806 or Ibrutinib to Kill Primary Cells from Patients with CLL or B-cell ALL Ex Vivo: IC$_{50}$ transformed into a Heatmap of Sensitivity

```
Patient Samples

CG-026806
Ibrutinib

Sensitivity

Resistant

IC$_{50}$

Sensitive

Resistant

IC$_{50}$

```

“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 Combines Successfully with Venetoclax to Kill Primary Samples from CLL or ALL Patients

- **Combination Studies:**
  - Enhanced ex vivo killing of patient bone marrow cells

- **CG-806 + Venetoclax:**
  - Combination may become the preferred drug combination for patients with CLL/SLL, ALL and other hematologic malignancies

Box plots show median and IQR; width is proportional to number of samples
Drugs are ordered from left to right by increasing median IC50 across all diagnoses
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
- Accelerated titration design
- Plan to include 6 dose levels
- Planned expansion cohorts

**Development Plan for Severe Unmet Needs in B Cell Tumors**

**CLL: Seek to treat patients resistant or intolerant to all:**
- Covalent BTK inhibitors
- BCL2 inhibitors
- Anti-CD20 therapy
- PI3K inhibitors
- Cytotoxins, Other Agents

**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
**Oral CG-806 Phase 1a/b Trial in CLL Patients Heavily Pretreated with Competitor Drugs**

<table>
<thead>
<tr>
<th>Dose Level</th>
<th>Description</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1</strong> (150mg BID for 28d)</td>
<td>Completed</td>
<td>Only One Patient Required in Dose Level 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• R/R-CLL/SLL : No DLTs and currently in Cycle 6 with stable disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Heavily pretreated ; TP53 mutation (strong negative prognostic marker)</td>
</tr>
<tr>
<td><strong>2</strong> (300mg BID for 28d)</td>
<td>Completed</td>
<td>Only One Patient Required in Dose Level 2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• R/R-CLL : No DLTs and currently in Cycle 4 – Challenging case</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Heavily pretreated ; marrow involvement with severe thrombocytopenia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>─ Needs an agent that spares the bone marrow and is unlikely to cause bleeding</td>
</tr>
<tr>
<td><strong>3</strong> (450mg BID for 28d)</td>
<td>Ongoing</td>
<td>Three Patients Required in Dose Level 3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Actively consenting and screening for appropriate patients</td>
</tr>
</tbody>
</table>
Patient #1 on Dose Level 1: Demographics & Prior Therapies

- **Diagnosis:** R/R-CLL/SLL
  - TP53 mutated or deleted
  - Unknown IGHV status

- **Demographic:** White Male Age 79

- **Prior Therapies**
  - Rituxan  Progression
  - Fludarabine  Progression
  - Cytoxan  Progression
  - Ibrutinib  AE
  - Rituxan  Progression
  - Rituxan  Progression
  - Venetoclax  Intolerant
  - Idelalisib  Progression
  - Rituxan  Progression
  - Radiation  May 2019
Patient #1 on Dose Level 1 (150mg BID): Stable Disease

- One Patient with R/R-CLL/SLL
- Heavily Pretreated
  - Failed fludarabine, cytoxan, radiation, ibrutinib, venetoclax, rituximab, idelalisib
- No DLTs: Patient Remains on Study in Cycle 6
Patient #2 on Dose Level 2:
Demographics & Prior Therapies

- **Diagnosis:** R/R-CLL
  - Unmutated IGHV
  - RAI I-V / Binet B-C

- **Demographic:** White Male Age 76
  - Marrow Infiltration with Neutropenia and Thrombocytopenia

- **Prior Therapies**
  - Rituxan / Fludarabine: Progressive disease
  - Rituxan: Progressive disease
  - Rituxan: Progressive disease
  - Rituxan: Progressive disease
  - Gazyva: Progressive disease
  - Arzerra: Progressive disease
  - Rituxan: Progressive disease
  - Ibrutinib: Refused
Patient #2 on Dose Level 2 (300mg BID) :
Status Prior to Entry into CG-806 Trial

- One Patient with R/R CLL
- Heavily Pretreated
  - Failed fludarabine, rituximab, obinutuzumab & ofatumumab and refused ibrutinib
- Marrow Involvement with Severe Thrombocytopenia
  - Acutely ill with need for agent that spares bone marrow and unlikely to cause bleeding

- Completed Cycle 3 with no DLTs
- No drug-related SAEs
- Currently in Cycle 4

Platelet count 25,000/mm$^3$ at screening prior to entry in the trial
Patient #2 on Dose Level 2: Safe, Well-Tolerated and No Evidence of Myelosuppression To Date

No observed myelosuppression to date
- Neutrophils (ANC) stable
- Platelet count stable
Patient #2 Evidence of Response: Lymphocytosis

Evidence of response:

- Observed lymphocytosis: indicator of target engagement by BTK inhibitors
- Stabilization of platelet count without transfusion
CG-806 Favorable Steady-State Pharmacokinetics ($C_{MIN}$)

- Significant Oral Absorption and Predictable PK Profile
- Achieving Approximately 1µM Levels at Steady State in Dose Level 2
Assay to Measure Inhibition of BTK-pTyr223 in Whole Blood

- Whole blood collected four hours after administration of the first dose to each patient and p-BTK and total-BTK levels were determined by ELISA

Patient #1 – Dose Level 1 (150mg BID)

- BTK-pTyr223 not inhibited at 4 hrs in Patient #1
- At that time point, the plasma concentration of CG-806 was 5nM

Patient #2 – Dose Level 2 (300mg BID)

- BTK-pTyr223 demonstrated complete inhibition (compared to the calculated maximum inhibition) at 4 hrs
- At that time point, the plasma concentration of CG-806 was 93nM

BTK-pTyr223 ELISA assay design:
- Whole blood from patients was collected 4 hours following the first dose administers, the blood was lysed with lysis buffer and subjected to ELISA assay to detect level of total and pTyr223 BTK (tBTK and pBTK, respectively) in the samples with and without phosphatase/protease inhibitors (PPI).
- Data were analyzed as ratio of pBTK/tBTK
- Data are presented as % inhibition according to the following analysis protocol:
  - Normalized to maximum inhibition: the ratio of pBTK/tBTK in the presence of PPI adjusted for maximum inhibition by subtraction of the pre-dose ratio in the absence of PPI, and compared with the adjusted pre-dose value; calculation formula as % inhibition = 100 X (ratio of pre-dose with PPI – ratio of pos-dose with PPI) / (ratio of pre-dose with PPI – ratio of pre-dose without PPI)
Pharmacodynamic (PD) Markers: Patient Plasma Inhibited P-BTK, P-SYK and P-ERK in EOL1 Cells : Ex Vivo PIA Assay

Plasma inhibitory activity (PIA) assay design:
Plasma was collected from patients at the indicated times. EOL1 cells were treated with plasma from patients for 6hr, harvested and subjected to immunoblotting for P-proteins or total-proteins. Experimental control (data not shown) of cells treated in healthy donor plasma or culture medium containing various concentrations of CG-806 was run in parallel.

- Plasma from patients treated with CG-806 decreased the level of phosphorylated proteins in EOL1 reporter cells ex vivo
  - phospho-BTK (Y551)
  - phospho-SYK (Y5525/Y526)
  - phospho-ERK (T202/Y204)

- The reduction of phosphorylation correlated with the level of CG-806 detected in the plasma
CG-806 in Patient #2 (300mg BID): Evidence of Response, Well-Tolerated and No Myelosuppression to Date

✅ Evidence of Target Engagement (BTK and Other Key Kinases)
- Inhibition of P-BTK, P-SYK, P-ERK and P-PDGFRα : PIA Assay
- 100% inhibition of P-BTK in PBMC : ELISA Assay

✅ Evidence of Clinical Response in R/R CLL
- Marked lymphocytosis
  - BTK inhibition in CLL patients leads to CLL cell exfiltrated from the lymphoid tissues
  - Observed early in Cycle 1 and continuing through Cycle 3
- FDG PET/CT after cycle 2: consistent with no disease progression
- Platelet stabilization without transfusions to date

✅ Safe and Well Tolerated To Date
- No myelosuppression, drug-related SAEs or dose-limiting toxicities

✅ Steady-State Pharmacokinetics with Favorable Exposures
CG-806: Key Messages from Phase 1 a/b CLL/NHL Trial Findings Through Dose Levels 1 and 2 (Accelerated Titration)

- **Safety:** No Unexpected Toxicities Have Emerged To Date
  - No Myelosuppression, No Drug-related SAE or DLT

- **Evidence of BTK Target Engagement**
  - Inhibition of P-BTK
  - Lymphocytosis in Dose 2

- **Early Evidence of Clinical Response**
  - Lymphocytosis and Platelet Stabilization

- **Significant Oral Absorption and Predictable PK Profile**

- **Achieving Approx. 1µM Levels at Steady State in Dose Level 2**

- **Exposures Likely Therapeutic for AML Patients**
CG-806

Mutation-Agnostic FLT3/BTK Inhibitor

Pending Phase 1a/b in AML & MDS
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

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, PDGFR\(\alpha\), CSF1R, ERK, BTK, SYK, AKT, MYC key pathways suppressed

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

CG-806 Superior to Other FLT3-ITD Inhibitors

<table>
<thead>
<tr>
<th>Drug</th>
<th>IC&lt;sub&gt;50&lt;/sub&gt; (nM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CG-806&lt;sup&gt;(1)&lt;/sup&gt;</td>
<td>0.8</td>
</tr>
<tr>
<td>Quizartinib&lt;sup&gt;(2)&lt;/sup&gt;</td>
<td>8.8</td>
</tr>
<tr>
<td>Gilteritinib&lt;sup&gt;(3)&lt;/sup&gt;</td>
<td>0.9</td>
</tr>
<tr>
<td>Crenolanib&lt;sup&gt;(4)&lt;/sup&gt;</td>
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</tr>
<tr>
<td>Midostaurin&lt;sup&gt;(2)&lt;/sup&gt;</td>
<td>11</td>
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<tr>
<td>Nexavar&lt;sup&gt;(2)&lt;/sup&gt;</td>
<td>79</td>
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<tr>
<td>Sutent&lt;sup&gt;(2)&lt;/sup&gt;</td>
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</table>

CG-806 Potent (Kd) FLT3 WT/Mutants

<table>
<thead>
<tr>
<th>FLT3 Proteins (Fragments)</th>
<th>CG-806 Kd (nM)</th>
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<tbody>
<tr>
<td>FLT3 WT</td>
<td>0.24</td>
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<tr>
<td>FLT3 ITD</td>
<td>3.1</td>
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<tr>
<td>FLT3 D835Y</td>
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</table>

<table>
<thead>
<tr>
<th>Drug</th>
<th>IC&lt;sub&gt;50&lt;/sub&gt; (nM)</th>
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<td>Crenolanib</td>
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<td>Midostaurin</td>
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<td>Nexavar</td>
<td>79</td>
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<td>Sutent</td>
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</tbody>
</table>

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
CG-806 Exerts Broad & Superior Killing 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
CG-806 Combines Successfully with Venetoclax to Kill Primary Samples from AML and MDS/MPN Patients

Box plots show median and IQR; width is proportional to number of samples
Drugs are ordered from left to right by increasing median IC$_{50}$ across all diagnoses

• **Combination Studies:**
  — Enhanced ex vivo killing of patient bone marrow cells

• **CG-806 + Venetoclax:**
  — Combination may become the preferred drug combination for patients with AML, MDS/MPN, and other hematologic malignancies
CG-806 Rapid and Sustained Antitumor Activity in Mouse Model of AML After Oral Dosing for 28 Days

- No weight loss or toxicity at any dose level
- TGI at all dose levels over 28-d of dosing
- Significant cure rates with two highest doses
- Re-challenge of uncured mice with large tumors → Active on large tumors and no resistance observed

Group 4: 100mg/kg BID
5 of 11 mice cured
“Uncured” mice at d88 were treated with 300mg/kg BID for additional 28 days beginning d88 and those tumors responded to treatment

Group 5: 300mg/kg BID
10 of 11 mice cured
“Uncured” mouse at d88 was treated with 300mg/kg BID for additional 28 days beginning d88 and that tumor responded to treatment
**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.
Developing CG-806 for the Treatment of AML

**Strong Rationale:**

- Broadly potent against AML cells
  - Wild type FLT3 and mutated FLT3, TP53, IDH1, IDH2, SRF2 and ASXL1
  - More potent than other FLT3 inhibitors on >200 AML patient samples
  - Delivers cures in xenograft models of human AML without toxicity
  - High “value creation impact”

**Phase 1 Plan: Treat 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
- Patients with wild type-FLT3

- Rapidly differentiate CG-806 from other FLT3i’s
- Plan to initiate dosing with an active dose level
CG-806 Phase 1 Clinical Development Plan: Planned Trial in Patients with AML/MDS

First Phase 1a/b Ongoing: R/R CLL and NHL Patients

Seek to identify a dose in CLL/NHL patients that delivers a plasma “therapeutic exposure” for AML

Planned Second Phase 1a/b: Relapsed/Refractory AML/MDS

AML patients are acutely ill and do not wish to dose sub-therapeutically. Dose escalating Phase I trial – Define safety, tolerance, PK, PD and RP2D

Seek to Treat R/R AML Patients with High Unmet Medical Needs

- Resistant to FLT3 inhibitors
- “Unfit” / Fragile Population
- Resistant to Venetoclax
- Mutations in IDH1/2, p53

Plan to advance CG-806 into an AML study in 1H2020
APTO-253
Phase 1b Ongoing
Small Molecule MYC Inhibitor
For the Treatment of AML and HR-MDS
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

Targets DNA regulatory motif (G-Quadruplex) in promoter of MYC gene

- NOT the MYC protein

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 First Three Dose Levels: Safely Inhibits MYC Expression in AML & MDS Patients

• **AML Patient: Dose Level 1 (20mg/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
### Progress of APTO-253 in Phase 1b Trial in AML and MDS

<table>
<thead>
<tr>
<th>Dose Level</th>
<th>Description</th>
<th>Status</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dose Level 1</td>
<td>(20mg/m²)</td>
<td>Completed</td>
<td>1 AML Patient</td>
</tr>
<tr>
<td>Dose Level 2</td>
<td>(40mg/m²)</td>
<td>Completed</td>
<td>1 MDS Patient</td>
</tr>
<tr>
<td>Dose Level 3</td>
<td>(66mg/m²)</td>
<td>Completed</td>
<td>3 AML Patients</td>
</tr>
</tbody>
</table>

- To date, well-tolerated & no drug-related SAEs
- Now screening for patients in dose level 4 (100mg/m²)
## 2019 Anticipated Catalysts

### CG-806
- **1H:** First B-cell cancer patient dosed with CG-806 ✅
- **2H:** Demonstrate Evidence of Response in CLL ✅
- **2H:** Presentation of clinical data during ASH 2019 ✅
- **TBD:** FDA approval for AML trials: Anticipate 1H 2020

### APTO-253
- **1H:** Successful completion of second dose level ✅
- **2H:** Initiation of AML/MDS patients with third dose level ✅
- **2H:** Presentation of clinical data during ASH 2019 ✅

### Management
- **2H:** Further expansion of management team ✅
Aptose Executive Summary

Developing Highly Differentiated / Targeted Drugs for Blood Cancers

**CG-806  Clinical Mutation-Agnostic FLT3 / BTK Inhibitor**
- Potently Inhibits all forms of FLT3 and BTK, plus multiple oncogenic signaling pathways
- Potential to treat CLL & NHL patients failing covalent BTKi’s / venetoclax / rituximab
- Potential to treat AML patients FLT3i-resistant, IDH1-mutant, and unfit / elderly
- Phase 1 in CLL & NHL underway and subsequent AML trial planned

**APTO-253  Clinical First-in-Class MYC Inhibitor**
- Dosing of R/R-AML / hr-MDS patients ongoing
- Demonstrated MYC inhibition: Watch for PK, safety, tolerance and responses
- Potential to expand into B cell malignancies and solid tumors

**Strong Leadership and KOL Support**
- Executing on our plans, addressing patient needs and building value

**Strong Financial Foundation**
- Cash Balance September 30, 2019: $30.2 MM
- Clean Capital Structure: No Warrants, No Debt, No Preferred Stock
KOL-Driven SAB

Dr. Brian J. Druker, MD
Collaborator & Chair of SAB

Key Role in Dev’t of Gleevec
Member, National Academy of Medicine, National Academy of Sciences & American Academy of Arts & Sciences
Winner of Karnofsky Award, Lasker “America’s Nobel” Award
Winner of Japan Prize in Healthcare and Medical Technology and the Tang Prize in Biopharmaceutical Science
Winner of the prestigious Sjöberg Prize
Leader of Inter-institutional Beat AML Initiative

Dr. Michael Andreeff, MD, PhD
Collaborator & Member of SAB

Professor of Medicine
Paul and Mary Haas Chair in Genetics
Director, Flow Cytometry and Cellular Imaging Facility
Director, Bone Marrow Aspiration Clinic
Chief, Section of Molecular Hematology and Therapy
MD Anderson Cancer Center
Physician Scientist
Expert in AML and other hematologic malignancies
Expert in drug resistance and drug mechanisms
Published over 450 peer-reviewed papers
Published multiple books and chapters

Distinguished KOLs with Domain Expertise in the Hematology/Oncology Space

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
Physician in Chief, TGen
Medical Director of Research for McKesson Specialty Health
Chief Scientific Officer for US Oncology Research
Professor of Medicine, Mayo Clinic Scottsdale, AZ

Dr. Michael Andreeff, MD, PhD
Collaborator & Member of SAB

Professor of Medicine
Paul and Mary Haas Chair in Genetics
Director, Flow Cytometry and Cellular Imaging Facility
Director, Bone Marrow Aspiration Clinic
Chief, Section of Molecular Hematology and Therapy
MD Anderson Cancer Center
Physician Scientist
Expert in AML and other hematologic malignancies
Expert in drug resistance and drug mechanisms
Published over 450 peer-reviewed papers
Published multiple books and chapters

Distinguished KOLs with Domain Expertise in the Hematology/Oncology Space
Thank You!