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.
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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 medicines
Precision treatment of hematologic malignancies; life-threatening / orphan diseases

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

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**

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

- JAK / STAT pathway
- MAPK / ERK pathway
- NFκB pathway
- PI3K / AKT / mTOR / S6K pathway

Inhibited directly
- CG-806

Inhibited indirectly
- CG-806

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

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

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

Pending FDA Approval
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 enzyme
- Inhibits multiple "oncogenic rescue" pathways to potentially avoid R/R disease

CG-806 Non-Covalent Inhibitor Retains Potency Against Wildtype and C481S-BTK

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.

Expect Superior Safety Profile for CG-806

<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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</tbody>
</table>

<table>
<thead>
<tr>
<th>IC₅₀ (nM)</th>
<th>TEC</th>
<th>EGFR</th>
<th>ErbB2</th>
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<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>
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</tbody>
</table>
CG-806 Exerts Superior Breadth & Potency Compared to Ibrutinib on Patient Samples

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

“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 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 Now in Dose Level 3 of Phase 1a/b Clinical Trial in CLL/NHL

<table>
<thead>
<tr>
<th>Dose Level 1 (150mg BID for 28d) Completed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Only One Patient Required in Dose Level 1</td>
</tr>
<tr>
<td>• R/R-CLL/SLL with TP53 mutation ; Heavily pretreated</td>
</tr>
<tr>
<td>• Challenging Case with TP53 mutation – No DLTs and in Cycle 9</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dose Level 2 (300mg BID for 28d) Completed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Only One Patient Required in Dose Level 2</td>
</tr>
<tr>
<td>• R/R-CLL with unmutated IGHV ; Heavily pretreated</td>
</tr>
<tr>
<td>• Marrow involvement with neutropenia and thrombocytopenia</td>
</tr>
<tr>
<td>• Highly complicated disease to manage – No DLTs and completed Cycle 4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dose Level 3 (450mg BID for 28d) Completed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Three Patients Required in Dose Level 3 – 3 Patients Dosed</td>
</tr>
<tr>
<td>• Collecting Data for Cohort Safety Review Committee Meeting</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dose Level 4 (600mg BID for 28d) Preparing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Three Patients Required in Dose Level 3 – Currently Screening</td>
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</tbody>
</table>

CG-806 Favorable Steady-State Pharmacokinetics ($C_{\text{min}}$)

- Oral absorption and favorable steady-state PK: 0.6-1µM steady state ($C_{\text{min}}$) at Dose Level 2
- Pharmacologic exposure in Dose Level 2: Inhibits 100% P-BTK in PBMC 4hr post-dose
- PIA assay shows exposure-dependent inhibition of phospho-BTK, -ERK, PDGFRα, and -SYK
• **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) completed

• **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
  – **Target Engagement**: Plasma inhibits P-BTK, P-SYK, P-ERK, P-PDGFRα in EOL1
  – **Lymphocytosis**: BTK inhibition in CLL promotes exfiltration

• **Well-behaved Steady-State Oral Pharmacokinetics**
  – Absorption that delivered circa-uM trough plasma exposure levels (steady state)

• **Plan toContinue Dose Escalation + Approaching Starting Dose for AML**
CG-806: A New Class of Drugs
More than Just a BTK Inhibitor for CLL
Only Agent Also to Inhibit FLT3 for AML

Breadth for Difficult-to-Treat CLL and NHL Patients

- Potently inhibits WT-BTK and C481S-BTK, plus multiple oncogenic pathways operative in B-cell cancers
- Potential to treat CLL patients failing covalent & non-covalent BTKi, Bcl-2i, CD-20 antibodies, and others
- Potential to treat Richter’s Transformation, Tx-refractory DLBCL / Follicular Lymphomas / DHLs

Safety: Targets Key Oncogenic Kinases and Avoids Safety Targets

- To date: safe, well-tolerated, and no drug related AEs have been observed
- Does not inhibit TEC, EGFR or ErbB2 kinases that cause toxicities with other BTK inhibitors
- Structurally distinct: assumes unique binding mode in kinase active sites relative to competitor agents

PLUS….Under Development for AML Patients Failing Other Drugs

- Only agent that inhibits BTK and FLT3 and is being developed for CLL/NHL and AML/MDS
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

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\(^1\) 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

<table>
<thead>
<tr>
<th>Drug</th>
<th>IC$_{50}$ (nM)</th>
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<tbody>
<tr>
<td>CG-806</td>
<td>0.8</td>
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<tr>
<td>Quizartinib</td>
<td>8.8</td>
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<tr>
<td>Gilteritinib</td>
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<tr>
<td>Crenolanib</td>
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<tr>
<td>Midostaurin</td>
<td>11</td>
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<tr>
<td>Nexavar</td>
<td>79</td>
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<tr>
<td>Sutent</td>
<td>1</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>
</tr>
<tr>
<td>FLT3 D835Y</td>
<td>4.2</td>
</tr>
<tr>
<td>D835H</td>
<td>2.2</td>
</tr>
<tr>
<td>D835V</td>
<td>7.9</td>
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<tr>
<td>R834Q</td>
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</tr>
<tr>
<td>N841I</td>
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</tr>
<tr>
<td>K663Q</td>
<td>0.55</td>
</tr>
<tr>
<td>ITD / F691L</td>
<td>16</td>
</tr>
</tbody>
</table>

CG-806 Superior to Other FLT3 Inhibitors on AML Cells with FLT3-D835Y Mutation

1. Reaction Biology Corp.
2. Blood. 2009 Dec 1; 114(11): 3244-3252
3. J Clin Oncol 32:5s, 2014 (suppl; abstr 7070)
5. ASH Oral Presentation 2016
N/A – Data not available / Not Applicable.

(1) Ba/F3 isogenic cells kindly provided by Dr. Michael Andreeff at MDACC
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
- IC50 transformed into a Heatmap of Sensitivity
- CG-806 greater potency in killing primary AML cells bearing wild-type FLT3 or FLT3-ITD
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

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

**Survival probability**

- CG-806
- Vehicle
- 10mg/kg
- 30mg/kg
- 100mg/kg
- 300mg/kg

**Number at risk**

- Vehicle
- 10mg/kg
- 30mg/kg
- 100mg/kg
- 300mg/kg

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

CG-806 Efficacy in PDX Model Against AML Patient Cells with FLT3 ITD + D835 Mutations

- Reduced leukemia cell burden
- Reduced splenomegaly
- Extended survival
- Active against patient-derived FLT3-ITD / D835 AML
- Potential to treat emerging FLT3i-resistant AML patients

Model implanted with FLT3 ITD/D835 mutated primary AML cells. CG-806 Tx initiated d27 (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 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

• **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
APTO-253
Phase 1a/b Ongoing
Small Molecule MYC Inhibitor
For the Treatment of AML

1. MYC dysregulation is key driver of AML, certain B-cell cancers and solid tumors
2. MYC gene expression potently inhibited by APTO-253
3. Ph1a/b trial for AML/MDS ongoing with APTO-253
4. APTO-253 first agent to inhibit MYC expression and well tolerated in patients
Tremendous Interest in Targeting MYC as a Cancer Treatment

- **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

**APTO-253** Targets DNA regulatory motif in promoter of MYC gene
- NOT the MYC protein

**APTO-253 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:
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 (40mg/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 (66mg/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
APTO-253
Ongoing Phase 1b Dose Escalating Clinical Trial

- Dose Level 1 (20mg/m^2)  Completed  1 AML Patient
- Dose Level 2 (40mg/m^2)  Completed  1 MDS Patient
- Dose Level 3 (66mg/m^2)  Completed  3 AML Patients
  - Dose Level 4 (100mg/m^2)  Ongoing  3 Patients Required

- To date, well-tolerated & no drug-related SAEs
- Now dosing patient in dose level 4 (100mg/m^2)
<table>
<thead>
<tr>
<th>DRUG</th>
<th>TARGET</th>
<th>RIGHTS</th>
<th>INDICATIONS</th>
<th>Preclinical Stage</th>
<th>Clinical Proof-of-Concept</th>
<th>Pivotal Stage</th>
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</thead>
<tbody>
<tr>
<td>CG-806</td>
<td>Pan-BTK</td>
<td>Aptose: WW CG: Korea</td>
<td>CLL NHL</td>
<td>B-Cell Malignancies</td>
<td></td>
<td></td>
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<tr>
<td>CG-806</td>
<td>Pan-FLT3</td>
<td>Aptose: WW CG: Korea</td>
<td>AML MDS</td>
<td>AML / MDS Planned</td>
<td></td>
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<tr>
<td>APTO-253</td>
<td>MYC</td>
<td>Aptose: WW</td>
<td>AML MDS</td>
<td>AML / MDS Single Agent</td>
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<td>APL-581</td>
<td>BRD4/JAK</td>
<td>Aptose / Ohm</td>
<td>Hematologic Cancers</td>
<td>AML Single Agent</td>
<td></td>
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### 2020 Anticipated Catalysts

<table>
<thead>
<tr>
<th>DRUG</th>
<th>1H: Initiate trial in AML patients</th>
<th>2H: Seek clinical activity in AML patients</th>
<th>2H: Seek clinical activity in B-cell cancer patients</th>
<th>1-2H: Presentation of clinical data during EHA (B-cell) and ASH (B-cell &amp; AML)</th>
<th>1-2H: Continue dose escalation in AML/MDS patients</th>
<th>2H: Explore additional cancer indications</th>
<th>2H: Presentation of clinical data during ASH</th>
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</thead>
</table>
Thank You!