Precision Oncology for Therapies of Tomorrow

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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**Investment Highlights**

**APTOSE**
- Strong leadership with expanded management team
- Approximately 2 years of cash to advance clinical programs
- Clinical stage biotech company developing 1st-in-class targeted agents
- Treating 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 & 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
1\textsuperscript{st}-in-Class
Oral FLT3 / BTK Inhibitor
Phase 1a/b Ongoing

1. Non-covalent “reversible” inhibitor with unique kinome targeting profile
2. Potent inhibitor of all forms of BTK (WT / C481S) driver kinase
3. Potent inhibitor of all forms of FLT3 (WT / ITD or TKD mutated) driver kinase
4. Suppresses multiple signaling pathways essential for cancer cell survival
5. Precision spares safety targets & pathways associated with toxicity
6. Ongoing trial Ph1a/b for CLL & NHL B-cell malignancies
7. Planning trial Ph1a/b for AML/MDS myeloid malignancies
“Multi-Cluster Kinase Inhibitor”: CG-806 Potently and Selectively Inhibits Clusters of Related Kinases

- **Mutation Agnostic**
  - Inhibits all forms of FLT3
  - Inhibits all forms of BTK
  - Simultaneously suppresses multiple 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 Hematologic Malignancies**
  - FLT3 cluster → AML & MDS
  - BTK cluster → CLL & NHL
CG-806 Suppresses Key Oncogenic Targets and Pathways in Myeloid & Lymphoid Malignancies

- FLT3mut
- FLT3i
- CLL
- AML
- BTKupreg
- BTKi
- MYC
- NFκB
- PI3K / AKT / mTOR / S6K pathway
- MAPK / ERK pathway
- JAK / STAT pathway
- NFκB pathway
- Cell Growth and Proliferation

Pathways and Targets:
- FLT3
- CSF1R
- PDGFRα
- STAT
- JAK / STAT pathway
- MAPK / ERK pathway
- NFκB pathway
- PI3K / AKT / mTOR / S6K pathway
- MYC
- BCR
- BTK
- TEC
- SRC
- LYN
- SYK
- SRC
- LYN
- SYK
- PI3K
- AKT
- mTOR
- S6K
- FLT3i
- BTKi
CG-806 for the Treatment of CLL / SLL / 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

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
- Well tolerated and inhibits multiple “oncogenic rescue” pathways
- Potently and directly kills CLL and other B-cell cancer cells

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 IC$_{50}$ (nM)</th>
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<tbody>
<tr>
<td>BTK-WT</td>
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<td>BTK-C481S</td>
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<table>
<thead>
<tr>
<th>IC$_{50}$ (nM)</th>
<th>TEC</th>
<th>EGFR</th>
<th>ErbB2</th>
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<tr>
<td>Ibrutinib</td>
<td>78</td>
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<tr>
<td>CG-806</td>
<td>&gt;1,000</td>
<td>&gt;1,000</td>
<td>&gt;1,000</td>
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</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: 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

Development Plan for Severe Unmet Needs in B Cell Tumors

**CLL Patients Resistant or Intolerant to:**
- Covalent BTK inhibitors
- 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, DHL

**Dose Escalation Phase**
- Patients administered oral capsules
- Every 12 hours on a 28-day cycle
- Plan to include 6 dose levels
- Accelerated titration design
- Planned expansion cohorts

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 in Dose Level 3 of Phase 1a/b Clinical Trial in CLL/NHL
(Clinical Data Cutoff Dec 31, 2019)

Dose Level 1 (150mg BID for 28d) Completed
Only One Patient Required in Dose Level 1

- R/R-CLL/SLL with TP53 mutation; Heavily pretreated
- Challenging Case with p53 Mutation

Dose Level 2 (300mg BID for 28d) Completed
Only One Patient Required in Dose Level 2

- R/R-CLL with unmutated IGHV; Heavily pretreated
- Marrow involvement with neutropenia and thrombocytopenia
- Highly Complicated Disease to Manage
CG-806 Favorable Steady-State Pharmacokinetics ($C_{\text{MIN}}$)

- Oral absorption, dose-related exposure, predictable steady-state PK
- Achieving 0.6-1µM steady state ($C_{\text{MIN}}$) levels in Patient at Dose Level 2
- Approaching what we believe is active exposure in Dose Level 2
- Asked if the exposure at Dose Level 2 could inhibit P-BTK
**Phospho-BTK Inhibition in PBMC**

- Whole blood collected **four hours after administration** of the first dose
- **ELISA assay** to determine Phospho-BTK and total-BTK levels
- **BTK-pTyr223** completely inhibited at 4 hrs

**Phospho-BTK Inhibited in Reporter Cells by Plasma**

- Plasma inhibitory assay (PIA) using plasma collected from patient
- EOL-1 reporter cells **in vitro** treated 6hrs with plasma from patient
- Reduction of key phospho-proteins in EOL-1 reporter cells
  - phospho-BTK (pTyr551)
  - phospho-SYK (pTyr5525/Tyr526)
- PD responses correlated with CG-806 concentration in plasma
CG-806 Delivered Evidence Safety, Target Engagement and Clinical Activity in Patient #2 (300mg BID)

**Evidence of Safety with No Unexpected Toxicities**
- No myelosuppression; stabilized platelets and neutrophils
- No drug-related SAEs; No dose-limiting toxicities

**Evidence of Target Engagement with ↓P-BTK**
- Inhibition of P-BTK, P-SYK, others: PIA Assay
- 100% inhibition of P-BTK in PBMC: ELISA Assay

**Evidence of Clinical Activity in R/R CLL**
- Marked lymphocytosis
  - BTK inhibition in patients leads to CLL cell exfiltrated from lymphoid tissues
  - Observed immediately upon initiation of dosing in Cycle 1

**Well-behaved Oral Steady-State Pharmacokinetics**
- Absorption that delivered near-uM exposures
CG-806 Now in Dose Level 3 of Phase 1a/b Clinical Trial in CLL/NHL

**Dose Level 1 (150mg BID for 28d) Completed**
Only One Patient Required in Dose Level 1

- R/R-CLL/SLL with TP53 mutation; Heavily pretreated
- Challenging Case with p53 Mutation – No DLTs and completed Cycle 6

**Dose Level 2 (300mg BID for 28d) Completed**
Only One Patient Required in Dose Level 2

- R/R-CLL with unmutated IGHV; Heavily pretreated
- Marrow involvement with neutropenia and thrombocytopenia
- Highly Complicated Disease to Manage – No DLTs and completed Cycle 4.5

**Dose Level 3 (450mg BID for 28d) Dosing Ongoing**
Three Patients Required in Dose Level 3
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 molecule that inhibits BTK and FLT3 and is being developed for CLL/NHL and AML/MDS
Aggressive Cancer of Blood/Bone Marrow (Orphan Disease)

- FLT3-ITD mutation is key driver in 25-35% of AML patients\textsuperscript{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 $\Rightarrow$ Toxicity
- “Selective” agents don’t provide durable responses $\Rightarrow$ 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\textalpha, CSF1R, BTK, SYK, ERK, AKT, JAK/STAT, MAPK, MYC pathways

\textsuperscript{1} American Cancer Society : \textsuperscript{2} Cancer. 2014 July 15; 120(14): 2142-2149 : \textsuperscript{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&lt;sub&gt;50&lt;/sub&gt; (nM)</th>
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<tr>
<td>CG-806(1)</td>
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<tr>
<td>Quizartinib(2)</td>
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<td>Gilteritinib(3)</td>
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<td>Crenolanib(4)</td>
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<tr>
<td>Midostaurin(2)</td>
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<tr>
<td>Nexavar(2)</td>
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<tr>
<td>Sutent(2)</td>
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### CG-806 Potent (Kd) FLT3 WT/Mutants

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<thead>
<tr>
<th>FLT3 Proteins (Fragments)</th>
<th>CG-806 Kd (nM)</th>
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<tr>
<td>FLT3 WT</td>
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<tr>
<td>FLT3 ITD</td>
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<tr>
<td>FLT3 D835Y</td>
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<tr>
<td>D835H</td>
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<tr>
<td>D835V</td>
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<td>R834Q</td>
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<tr>
<td>K663Q</td>
<td>0.55</td>
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<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) Ba/F3 isogenic cells kindly provided by Dr. Michael Andreeff at MDACC

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(1) Reaction Biology Corp.
(2) Blood. 2009 Oct 1; 114(14): 2984–2992
(3) J Clin Oncol 32(5): 2014 (suppl): abstr 7070
(4) Blood 2014 Jan 2; 123(1): 94-100; AACR Poster 2012
(5) ASH Oral Presentation 2016

N/A – Data not available / Not Applicable.
CG-806 Exerts Broad & Superior Killing Potency 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
Sensitivity of AML Patients Samples to CG-806: AACR 2019
Enhanced Potency in Samples with FLT3 and IDH1 Mutations
Retains Potency in Samples with NPM1, p53, N-RAS and ASXL1 Mutations

- AML patient samples with FLT3 mutations (ITD or TKD), with or without concurrent mutations of NPM1, are highly sensitive to CG-806
- Sensitivity of AML patient samples generally related to FLT3 ITD high allelic ratio (IC50 = 0.03 µM) vs. low allelic ratio (IC50 = 0.11 µM)
- 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 TP53 mutations were resistant to most other FLT3 inhibitors
- 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 Rapid and Sustained Antitumor Activity in Mouse Model of AML After Oral Dosing for 28 Days

Mean Tumor Volume ± SEM

- **100mg/kg BID**
  - 5 of 11 mice cured with 1st course
  - "Uncured" mice at d88 were treated with 300mg/kg BID for 2nd course of 28 days beginning d88 and those tumors responded to treatment

- **300mg/kg BID**
  - 10 of 11 mice cured with 1st course
  - "Uncured" mouse at d88 was treated with 300mg/kg BID for 2nd course of 28 days beginning d88 and that tumor responded to treatment

- **No weight loss or toxicity** at any dose level
- **Significant cure rates** with two highest doses
- **Re-challenge of uncured mice** with large tumors
  - Active on large tumors and no resistance observed

**MV4-11 FLT3-ITD AML**
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 (70% AML pts) driven by other mutations
  – 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 and hr-MDS Patients with Unmet Needs
  – Patients who failed other FLT3 inhibitors
  – Patients who failed IDH-1 inhibitors
  – Patients who failed venetoclax / mutated RAS
  – 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

• Plan to Initiate Trial with an Active Dose
  – R/R-AML patients are acutely ill and we do not wish to dose sub-therapeutically
  – Continue to dose escalate in B-cell cancer patients and identify likely therapeutic dose for AML patients
  – Seek approval from FDA to initiate trial at a dose with likely “therapeutic exposure’ for 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 (20 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

• **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
APTO-253
Ongoing Phase 1b Dose Escalating Clinical Trial

- Dose Level 1 (20mg/m2) Completed 1 AML Patient
- Dose Level 2 (40mg/m2) Completed 1 MDS Patient
- Dose Level 3 (66mg/m2) Completed 3 AML Patients

- To date, well-tolerated & no drug-related SAEs
- Now screening for patients in dose level 4 (100mg/m2)
<table>
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<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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<tr>
<td>CG-806</td>
<td>Pan-BTK</td>
<td>Aptose: WW CG: Korea</td>
<td>CLL NHL</td>
<td>B-Cell Malignancies</td>
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<td>APTO-253</td>
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<td>AML Single Agent</td>
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**2020 Anticipated Catalysts**

**CG-806**
- **1H:** Initiate trial in AML patients
- **2H:** Seek clinical activity in AML patients
- **2H:** Seek clinical activity in B-cell cancer patients
- **1-2H:** Presentation of clinical data during EHA and ASH

**APTO-253**
- **1H:** Continue dose escalation in AML/MDS patients
- **2H:** Explore additional cancer indications
- **2H:** Presentation of clinical data during ASH
KOL Symposium on CG-806 FLT3 / BTK Inhibitor for Acute Myeloid Leukemia

Hosted by Aptose Biosciences Inc. (Nasdaq: APTO)

The luncheon symposium with Key Opinion Leaders in hematology/oncology will review the treatment landscape and the evolution of kinase inhibitors as anticancer drugs in myeloid leukemias, and highlight the potential for the mutation-agnostic FLT3/BTK inhibitor CG-806 to address unmet medical needs in these patient populations.

Additionally, the Aptose management team will provide an overview of the rationale and strategy for the development of CG-806 in myeloid malignancies. CG-806 is currently in an ongoing Phase 1a/b clinical trial for the treatment of patients with relapsed / refractory B-cell malignancies, including CLL and NHL, and in 1H / 2020 is planned to enter a separate clinical trial in patients with relapsed / refractory AML and high-risk MDS.

Additional information will be provided closer to the date of the event.

Wednesday, February 5th, 2020
Noon - 1:30 PM EST

Lotte New York Palace
455 Madison Ave
Thank You!