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Curis Mission & Strategy

Developing the New Generation of Targeted Cancer Drugs

Mission
Work relentlessly to develop innovative and differentiated therapeutics that improve the lives of cancer patients

Strategy
Select the right targets
Design the right drugs
Study the right patients
Overview

Investment Thesis

Curis seeks to develop novel, first-in-class, cancer therapeutics that we believe have significant potential in areas of unmet patient need

Robust Pipeline

- CA-4948: first-in-class suppressor of the TLR Pathway
  *There are no drugs currently approved that block the entire TLR pathway*

- CI-8993: first-in-class antagonist of the VISTA pathway
  *There are no drugs currently approved for VISTA inhibition*

- Fimepinostat: first-in-class suppressor of MYC
  *There are no drugs currently approved for MYC inhibition*

Corporate

- Experienced management team with proven capabilities
- Curis R&D pioneered the first-in-class inhibitor of the Hedgehog pathway (Erivedge®) partnered with and commercialized by Genentech/Roche for advanced basal cell carcinoma
- Cash, cash equivalents and investments of approximately $20.5M as of Dec 31, 2019
### Evolution of Curis

*Progressing through Clinical Studies on the Path to Potential Registration*

<table>
<thead>
<tr>
<th>2018</th>
<th>2019</th>
<th>2020</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Regulatory Planning</strong></td>
<td><strong>Clinical Execution</strong></td>
<td><strong>Expansion</strong></td>
</tr>
<tr>
<td>✓ Work with investigators and FDA to determine optimal clinical path</td>
<td>✓ Report preliminary efficacy data for CA-4948 Ph1 study in NHL</td>
<td>• Continue dose escalation study of CA-4948 to determine MTD or RP2D and report updated efficacy data from Ph1 study</td>
</tr>
<tr>
<td>✓ Identify patient populations and initiate clinical studies</td>
<td>✓ Evaluate additional opportunities for CA-4948 in AML/MDS and work with investigators to design clinical studies</td>
<td>• Initiate new single-agent study of CA-4948 in AML/MDS patients w/ specific spliceosome mutations</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Initiate dose escalation study of CI-8993</td>
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</tbody>
</table>
## Pipeline

### Of Oncology Drug Candidates

<table>
<thead>
<tr>
<th>Indication</th>
<th>PRE-CLINICAL</th>
<th>CLINICAL</th>
<th>MARKETED</th>
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</thead>
<tbody>
<tr>
<td><strong>Heme Malignancies</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CA-4948* IRAK4</td>
<td>MYD88/TLR-altered NHL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CA-4948* IRAK4</td>
<td>IL-1R/TLR-altered AML, MDS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fimepinostat HDAC/PI3K</td>
<td>MYC-altered Cancers</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Immune Checkpoint Inhibitors</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>CI-8993* VISTA</td>
<td>VISTA-expressing Cancers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CA-327* PDL1/TIM3</td>
<td>PDL1/TIM3-expressing Cancers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CA-170* PDL1/VISTA</td>
<td>PDL1/VISTA-expressing Cancers</td>
<td></td>
<td></td>
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<tr>
<td>Erivedge*** Hedgehog</td>
<td>Basal Cell Carcinoma</td>
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<td></td>
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</tbody>
</table>

* IP licensed from Aurigene
** Option to license IP from ImmuNext
*** IP licensed to Genentech (Curis receives royalty income)
Targeted Programs in Heme Malignancies

CA-4948: In development for treatment of TLR-altered cancers
CA-4948 Overview

In Development for Patients with MYD88/TLR-Altered Disease

### Profile

<table>
<thead>
<tr>
<th>Value Proposition</th>
<th>Population</th>
<th>Product Candidate Description</th>
</tr>
</thead>
</table>
| • First-in-class IRAK4 inhibitor in cancer  
  • Specific malignancies have overactivity of the myddosome/TLR pathway (which is dependent upon IRAK4)  
  • Composition-of-matter IP extends into 2035 | Lymphoma: Patients w/ over-activated myddosome/TLR pathway;  
  Patients treated w/ ibrutinib (IRAK4i has strong synergy with BTKi)  
  Leukemia: Patients whose tumors have splicing mutations that overexpress IRAK4 | • Potent and orally bioavailable inhibitor of IRAK4 for treatment of MYD88-altered tumors and augmentation of BTK inhibition |

**Affinity**

<table>
<thead>
<tr>
<th>Kinase</th>
<th>$K_d$ (nM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IRAK4</td>
<td>23</td>
</tr>
<tr>
<td>IRAK1</td>
<td>12,000</td>
</tr>
<tr>
<td>IRAK2</td>
<td>&gt;20,000</td>
</tr>
<tr>
<td>IRAK3</td>
<td>8,500</td>
</tr>
</tbody>
</table>

Designed to be best-in-class IRAK4 inhibitor\(^1\)

Potent suppressor of signal transduction\(^2\)

![Phospho-protein levels in AML cells after treatment with CA-4948](image)

1) Data from Curis preclinical study  
2) Booher et al. AACR 2017 (poster #1168)
CA-4948 Mechanism of Action

In Development for Patients with MYD88/TLR-Altered Disease

Inhibiting either of these two pathways should provide benefit to patients with B cell lymphoma, CA-4948 targets oncogenic activity in the TLR pathway by blocking IRAK4

**Oncogenic**
- Dysregulation drives excessive B Cell proliferation

**Pathway validated**
- MCL, MZL, CLL, Waldenström’s macroglobulinemia

**BTK inhibition effective**
- Ibrutinib is FDA approved

1) IMBRUVICA Package Insert. Rev 08/2018
4) Smith et al. Nat Cell Biol 2019
CA-4948 in Lymphoma

Preliminary Phase 1 Data Demonstrate Tolerability, PK, PD, and Anti-Cancer Activity

Patient Population
- Patients with R/R Lymphoma (incl DLBCL, WM, and patients with MYD88-altered disease)

Treatment
- Oral, once-daily (QD) or twice-daily (BID), dosing in continuous 21-day cycles

Objective
- Safety/tolerability during dose escalation
- Efficacy during expansion

Preliminary Phase 1 Data Readout
- Generally well tolerated
- Favorable PK profile, PD, and anti-cancer activity
- 5 of 6 patients dosed at 200mg or higher have seen reduction
- Report updated safety and efficacy data in 2020
CA-4948 in Lymphoma

Single Agent Dose Response Observed as Phase 1 Study Advances to Therapeutic Dose Levels

Dose Response Observed
5 of 6 patients experienced tumor reduction as dose increased to therapeutic levels (200mg and higher)
CA-4948 in R/R Lymphoma

Single Agent Dose Response Observed as Phase 1 Study Advances to Therapeutic Dose Levels

Dose Response Observed

*Increased tumor reduction observed as patient increased dose*
CA-4948 in R/R Lymphoma

Combination with BTKi leads to Potent Anti-Cancer Activity in MYD88-altered DLBCL Models

Preclinical Anti-Cancer Activity

Potent as Single Agent

- Anti-cancer activity demonstrated in MYD88-altered DLBCL
- Interim readout in dose escalation study shows clear dose response
- We intend to continue dose escalation to further enhance efficacy until MTD / R2PD

Strong Synergy in Combination

- Anti-cancer activity demonstrated to be highly synergistic with BTK inhibition
- Evaluating potential clinical development strategies for CA-4948 with BTKi to assess anti-cancer activity

Preclinical Anti-Cancer Activity in MYD88-altered DLBCL\(^1\)

(OCI-Ly10)

1) Data from Curis preclinical study; Booher, et al. 4th Waldenstrom Roadmap Symposium
CA-4948 in AML/MDS

Additional Regulatory Path Identified in AML/MDS

Specific Genetic Mutations Lead to Expression of the Long Isoform of IRAK4 (IRAK4-L)
Spliceosome mutations (incl. SF3B1 and U2AF1) drive expression of IRAK4-L in >50% of AML/MDS

IRAK4-L is Oncogenic
IRAK4-L provides a genetic link to oncogenic immune signaling in AML/MDS

CA-4948 Directly Targets IRAK4
Direct inhibition of the protein associated with disease (IRAK4) may offer a potential path to accelerated approval

Blocking IRAK4-L reduces the formation of leukemia colonies in preclinical studies

CA-4948 treatment reduces leukemic blasts in patient-derived xenografts

Smith et al. Nat Cell Biol. 2019
Choudhary et al. AACR 2017
CA-4948 in AML/MDS

Overexpression of IRAK4-L is a Strong Predictor of Sensitivity to CA-4948

Overexpression of IRAK4-L is a Strong Predictor of Sensitivity to CA-4948

Smith et al. 2019

Bone Marrow Engraftment

<table>
<thead>
<tr>
<th></th>
<th>Vehicle</th>
<th>CA-4948</th>
</tr>
</thead>
<tbody>
<tr>
<td>(%) of mice</td>
<td>70%</td>
<td>10%</td>
</tr>
</tbody>
</table>

% of mice

Bone Marrow Engraftment

Smith et al. 2019
Monoclonal Antibody Immune Checkpoint Inhibitor

CI-8993: In development for treatment of VISTA-expressing cancers
## CI-8993 Overview

### In Development for Patients with VISTA Expressing Cancers

<table>
<thead>
<tr>
<th>Profile</th>
<th></th>
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<tbody>
<tr>
<td><strong>Value Proposition</strong></td>
<td>• First-in-class monoclonal antibody antagonist of VISTA</td>
</tr>
<tr>
<td></td>
<td>• No anti-VISTA drugs currently in the clinic</td>
</tr>
<tr>
<td></td>
<td>• Composition-of-matter IP extends into 2034</td>
</tr>
<tr>
<td><strong>Population</strong></td>
<td>• Patients with VISTA-expressing cancers, including Mesothelioma, NSCLC, and TNBC</td>
</tr>
<tr>
<td></td>
<td>• All patients who will receive PD1/PDL1 or CTLA4 antibody therapy</td>
</tr>
<tr>
<td></td>
<td>• Patients who have developed resistance to PD1/PDL1 or CTLA4 antibody therapy</td>
</tr>
<tr>
<td><strong>Product Description</strong></td>
<td>• Monoclonal antibody developed by ImmuNext/Janssen in partnership with Randy Noelle’s lab at Dartmouth (the co-discoverer of VISTA)</td>
</tr>
</tbody>
</table>
Illustrative Example of VISTA’s Role in Immune Suppression

Blocking PD-1 activates T cells in some patients

Blocking VISTA re-activates anti-tumor immune function

Blocking PD-1 also causes up to 5x increase in VISTA expression and re-suppression of T cell activity

**In Pre-Clinical Testing**, Blocking VISTA Results in:
Reduced suppressive mediators and enhanced antigen presentation of MDSCs
Altered chemotaxis (reduced traffic of MDSCs into the TME)

1 Data from ImmuNext preclinical studies
Targeted Programs in Heme Malignancies

Fimepinostat: In development for treatment of MYC-altered cancers
Fimepinostat Overview

In Development for Patients with MYC-Altered Cancers

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<td><strong>Value Proposition</strong></td>
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<td>• First-in-class drug candidate with demonstrated anti-cancer activity as a single agent in MYC-altered patients in Ph1 and Ph2 trials</td>
</tr>
<tr>
<td>• Composition-of-matter IP extends into 2032</td>
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<tr>
<td><strong>Population</strong></td>
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<tr>
<td>• Patients with MYC-altered cancer</td>
</tr>
<tr>
<td>• (&gt;50% of all cancers are effected by MYC)³</td>
</tr>
<tr>
<td>• Collaborating with DarwinHealth on characterization of biomarkers and tumor type alignments to identify potential therapeutic opportunities</td>
</tr>
<tr>
<td><strong>Product Candidate Description</strong></td>
</tr>
<tr>
<td>• Potent and orally bioavailable dual inhibitor of HDAC and PI3K enzymes¹</td>
</tr>
<tr>
<td>• Favorable safety profile in over 200 patients</td>
</tr>
</tbody>
</table>

Mechanism #1
The HDAC component Suppresses MYC transcription²

Mechanism #2
The PI3K component Enhances MYC destruction²

Dual Mechanism leads to potent and dose-dependent downregulation of MYC protein

Protein levels in DLBCL cells after treatment with Fimepinostat (Curis Preclinical Study)

1) Qian et al. Clin Cancer Res. 2012. 18: 4104
2) Sun et al. Mol Cancer Ther. 2017. 6: 285
Fimepinostat Clinical Data

Provides Strong Rationale For Development in MYC-Altered Lymphoma

**Monotherapy Anti-Cancer Activity**

Deep responses
- 8 complete responses (CR); 6 partial responses (PR)
- 2 patients able to proceed to transplant

Durable responses
- Median duration = 13.6 months

Fast Track designation received
- Following FDA review of clinical data

Evaluating potential therapeutic opportunities for fimepinostat in research collaboration with DarwinHealth
## Summary

**Investment Thesis**

Curis seeks to develop novel, first-in-class, cancer therapeutics that we believe have significant potential in areas of unmet patient need.

### Robust Pipeline

<table>
<thead>
<tr>
<th>Drug</th>
<th>Description</th>
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<td></td>
<td><em>There are no drugs currently approved for MYC inhibition</em></td>
</tr>
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</table>

### Potential Upcoming Catalysts

- Updated efficacy data for CA-4948 Phase 1 study
- Initiation of CA-4948 Phase 1 study in AML/MDS patients with spliceosome mutations
- Initiation of CI-8993 dose escalation Phase 1 study
Curis

Leadership Team

Rachel Blasbalg
Head, Human Resources

James Dentzer
President & CEO

Christine Guertin
Head, Regulatory

Robert Martell
Head, R&D

Mark Noel
Head, Intellectual Property

Reinhard von Roemling
Head, Clinical Development

Raul Soikes
Head, Portfolio Management

Nancy Soohoo
General Counsel

William Steinkrauss
Chief Financial Officer