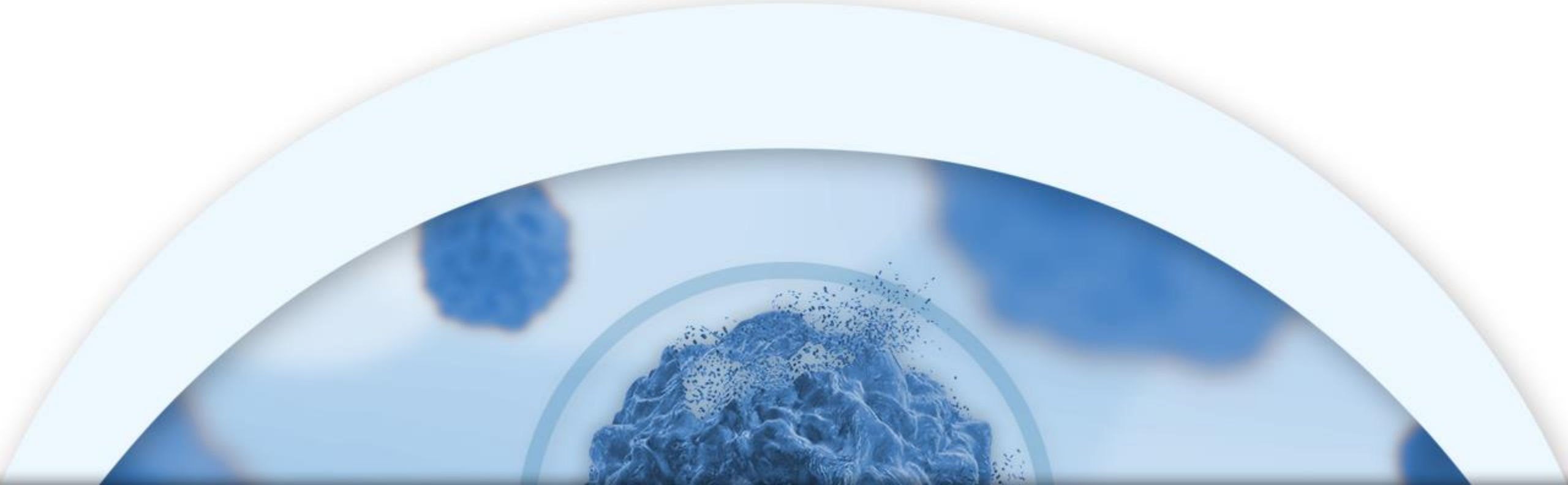
Corporate Presentation

NASDAQ: CRIS

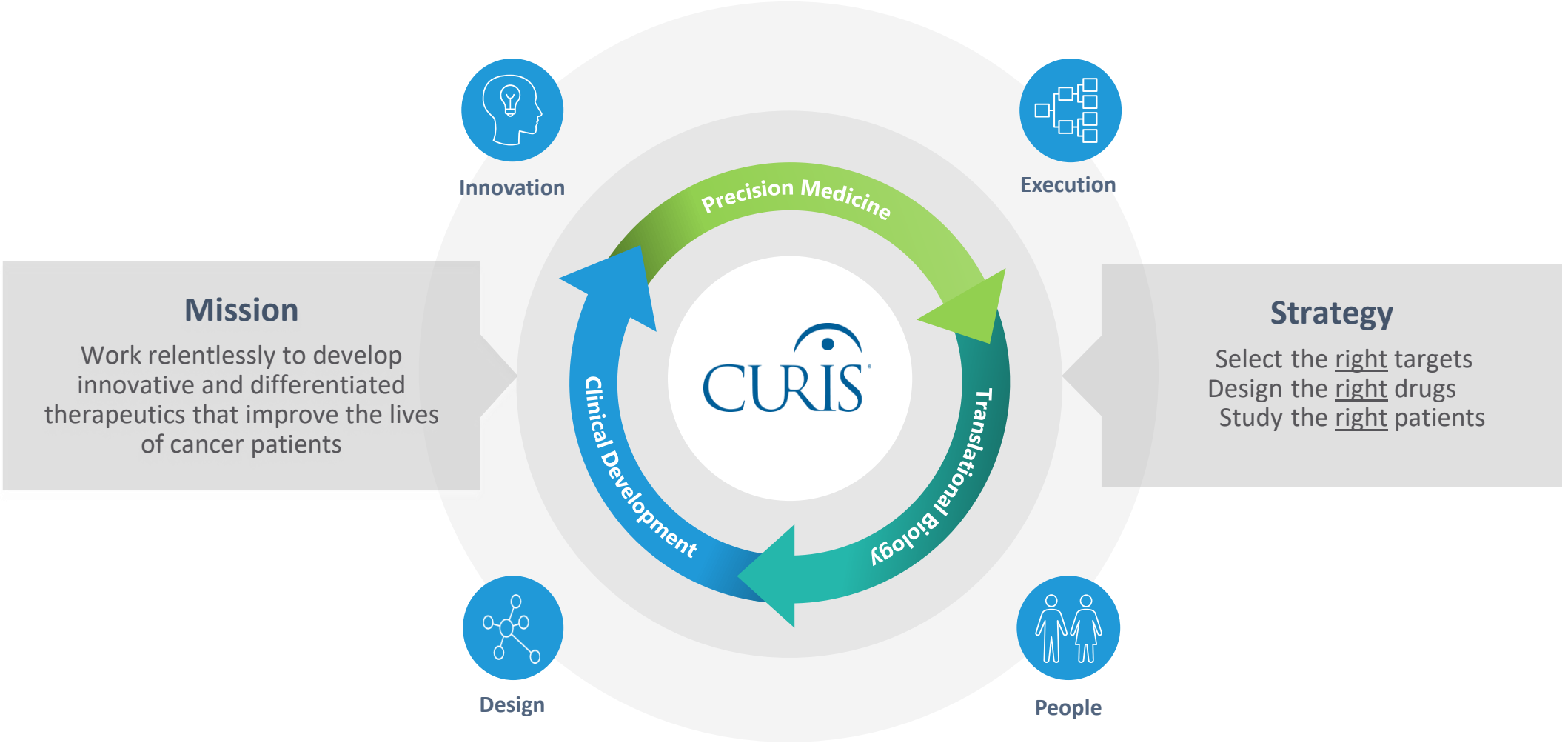


Cautionary Note Regarding Forward Looking Statements

This presentation contains certain forward-looking statements about Curis, Inc. (“we,” “us,” or the “Company”) within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Words such as “expect(s),” “believe(s),” “will,” “may,” “anticipate(s),” “focus(es),” “plans,” “mission,” “strategy,” “potential,” “estimate(s)”, "intend," "project," "seek," "should," "would" and similar expressions are intended to identify forward-looking statements. Forward-looking statements are statements that are not historical facts, reflect management’s expectations as of the date of this presentation, and involve important risks and uncertainties. Forward-looking statements herein include, but are not limited to, statements with respect to the timing and results of future clinical and pre-clinical milestones; the timing of future preclinical studies and clinical trials and results of these studies and trials; the clinical and therapeutic potential of our drug candidates; and management’s ability to successfully achieve its goals. These forward-looking statements are based on our current expectations and may differ materially from actual results due to a variety of important factors including, without limitation, risks relating to: whether any of our drug candidates will advance further in the clinical development process and whether and when, if at all, they will receive approval from the U.S. Food and Drug Administration or equivalent foreign regulatory agencies; whether historical preclinical results will be predictive of future clinical trial results; whether historical clinical trial results will be predictive of future trial results; whether any of our drug candidate discovery and development efforts will be successful; whether any of our drug candidates will be successfully marketed if approved; our ability to achieve the benefits contemplated by our collaboration agreements; management’s ability to successfully achieve its goals; the sufficiency of our cash resources; our ability to raise additional capital to fund our operations on terms acceptable to us or the use of proceeds of any offering of securities or other financing; general economic conditions; competition; and the other risk factors contained in our periodic and interim reports filed with the Securities and Exchange Commission which are available on the SEC website at www.sec.gov. You are cautioned not to place undue reliance on these forward-looking statements that speak only as of the date hereof, and we do not undertake any obligation to revise and disseminate forward-looking statements to reflect events or circumstances after the date hereof, or to reflect the occurrence of or non-occurrence of any events, except as required by law.

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

Company

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	Fimepinostat: first-in-class suppressor of MYC *There are no drugs currently approved for MYC inhibition* 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*
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 $28M as of Sept 30, 2019

Evolution of Curis

Progressing through Clinical Studies on the Path to Potential Registration

2018 | **2019** | **2020**

Regulatory Planning

- ✓ Work with investigators and FDA to determine optimal clinical path
- ✓ Identify patient populations and initiate clinical studies

Dose Escalation

- ✓ Report safety/tolerability data for fimepinostat combination w/ venetoclax
- ✓ Report preliminary efficacy data for CA-170 Ph1 study in Mesothelioma*
- ✓ Report preliminary efficacy data for CA-4948 Ph1 study in NHL

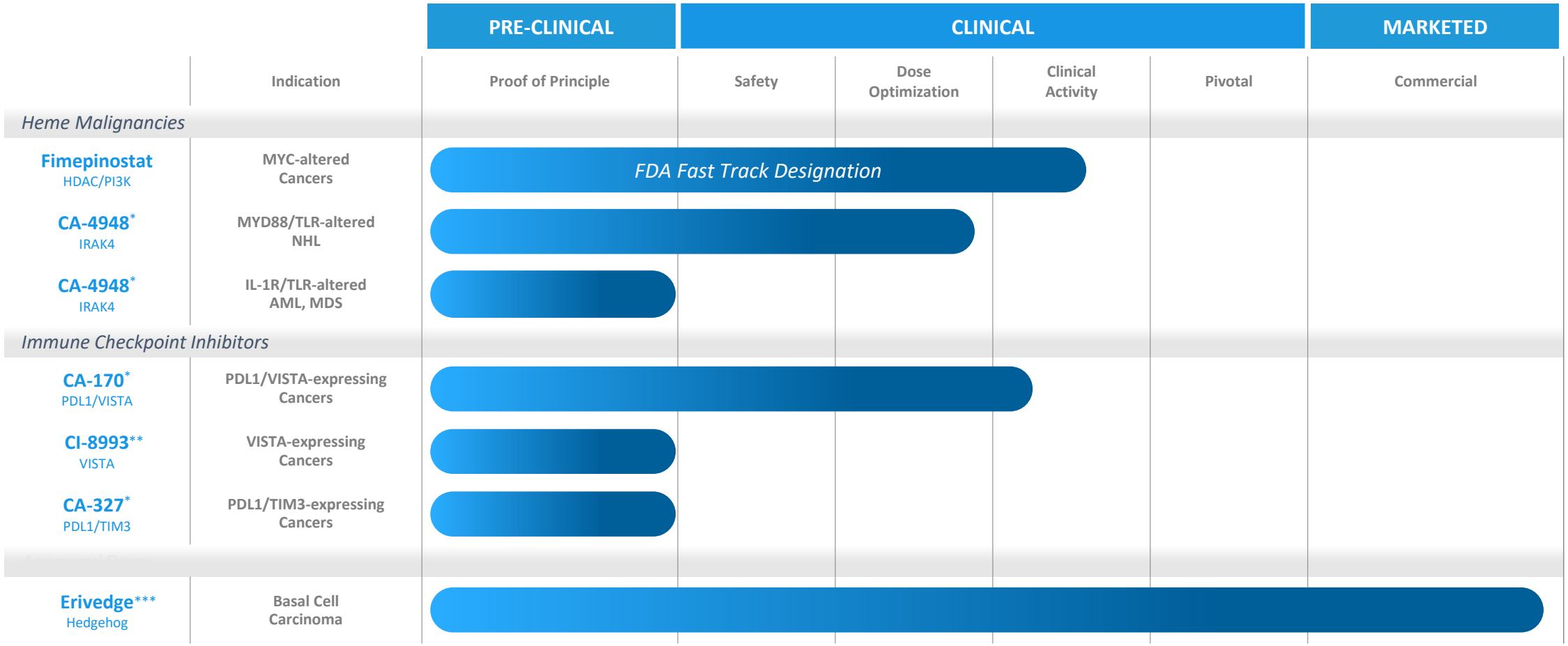
Expansion

- Report efficacy data for combination study of fimepinostat w/ venetoclax
- Continue dose escalation study of CA-4948 to determine MTD or RP2D
- Initiate two new studies of CA-4948:
  1) Single-agent study in AML/MDS patients w/ spliceosome mutations
  2) Combination study w/ BTKi in NHL
- Initiate dose escalation study of CI-8993

**Based on results from preliminary efficacy data, no further patients will be enrolled in this study. We are currently evaluating future studies for CA-170.*

Pipeline

Of Oncology Drug Candidates



* IP licensed from Aurigene
** Option to license IP from ImmuNext
*** IP licensed to Genentech (Curis receives royalty income)

Targeted Programs in Heme Malignancies

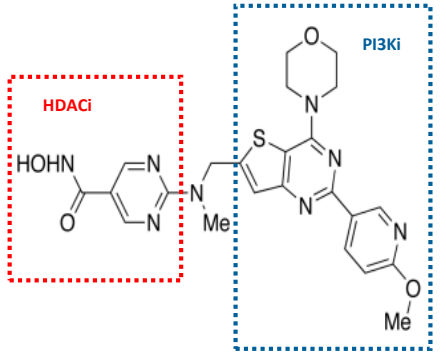
Fimepinostat: In development for treatment of MYC-altered cancers

Fimepinostat Overview

In Development for Patients with MYC-Altered Cancers

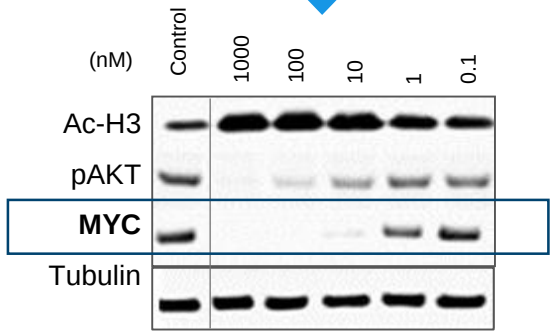
Profile	
Value Proposition	• First-in-class drug candidate with demonstrated anti-cancer activity as a single agent in MYC-altered patients in Ph1 and Ph2 trials • Composition-of-matter IP extends into 2032
Population	• Patients with MYC-altered cancer (>50% of all cancers are effected by MYC)[3]
Product Candidate Description	• Potent and orally bioavailable dual inhibitor of HDAC and PI3K enzymes[1] • Favorable safety profile in over 200 patients

Mechanism #1
The HDAC component Suppresses MYC transcription[2]



Mechanism #2
The PI3K component Enhances MYC destruction[2]

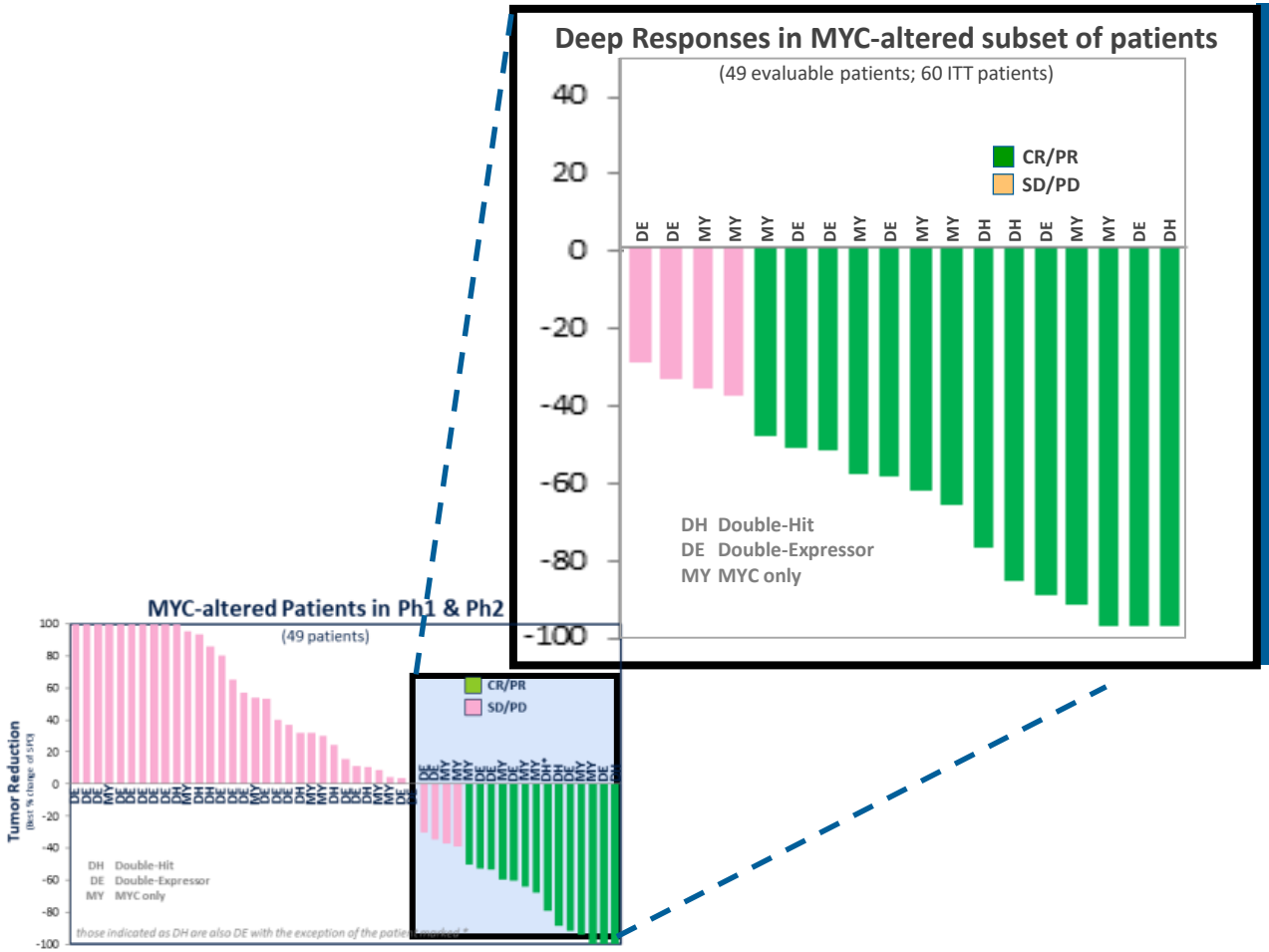
Dual Mechanism leads to potent and dose-dependent downregulation of MYC protein



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
3) Chen et al. Nature. 2018 Feb 23. 3:5

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

Fimepinostat + Venetoclax Appear Highly Synergistic in Preclinical Models

Fimepinostat + Venetoclax

As Combination Therapy Partners

Both drugs are active as single-agents in DLBCL

Fimepinostat = 23% ORR (with 13.6 month DOR[1])

Venetoclax = 18% ORR[2]

Highly synergistic combination

Combination index of < 0.1 at multiple doses[3]

Initial target indication has high unmet need

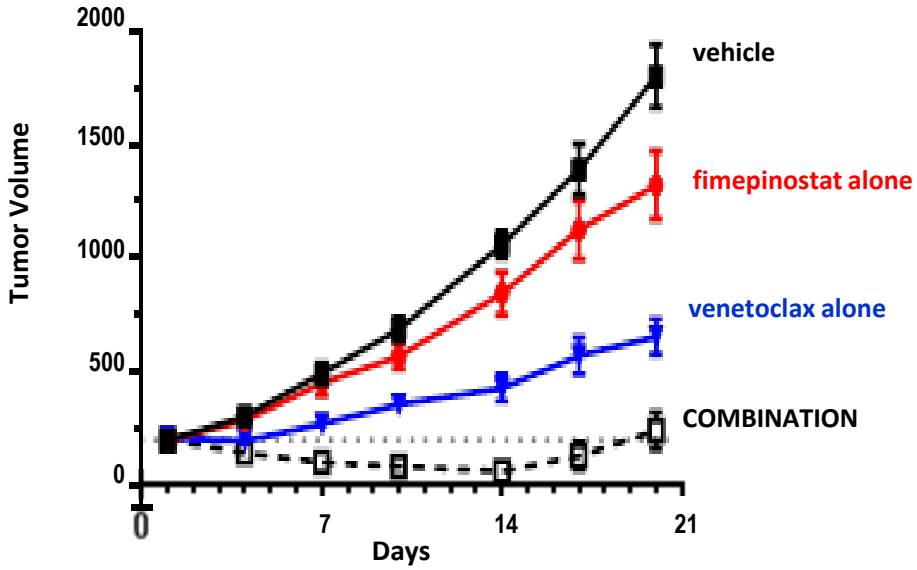
NCCN: Double-hit lymphoma (HGBL) is poor outcome group

FDA: Double-hit lymphoma (HGBL) is high unmet need

Potential for accelerated regulatory path

No drugs are currently approved for HGBL

Fimepinostat + Venetoclax
Appear Highly Synergistic in Preclinical Models
(DH DOHH-2 DLBCL model)[4]



1) 14 PR/CR out of 60 patients in Ph1 & Ph2 (23% ORR)
2) Davids et al. JCO. 2017. 35:826
3) Booher et al. ASH 2016 (poster #4184)
4) Data from Curis preclinical study

Fimepinostat Phase 1 Combination Study Designed to Demonstrate Safety of Combination

Patient Population

- Patients with R/R DLBCL, including DH/DE Lymphoma
- 8 Study Sites (US only)

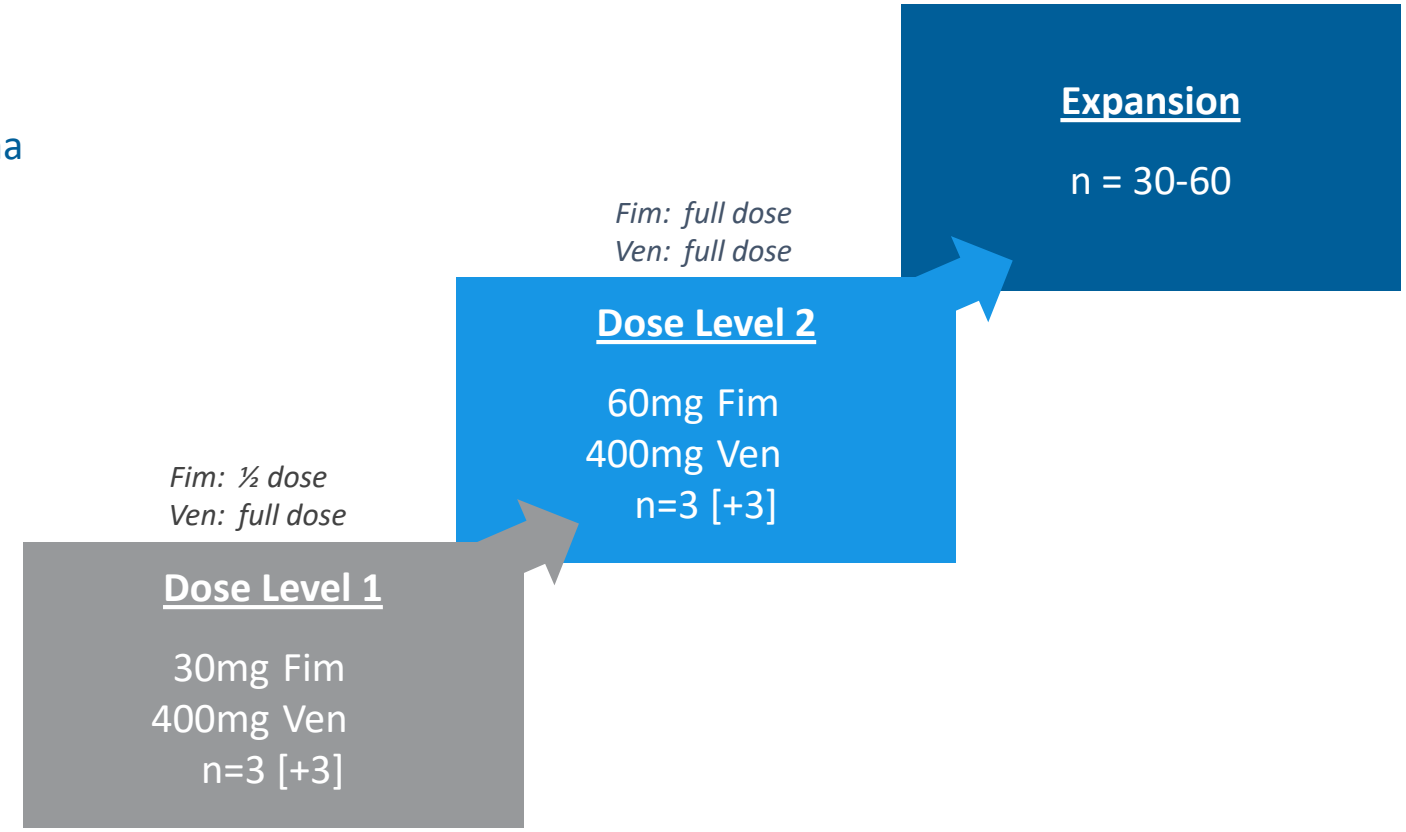
Treatment

Fimepinostat: Oral daily (5 days on, 2-days off)

Venetoclax: Oral daily (with rapid dose ramp-up)

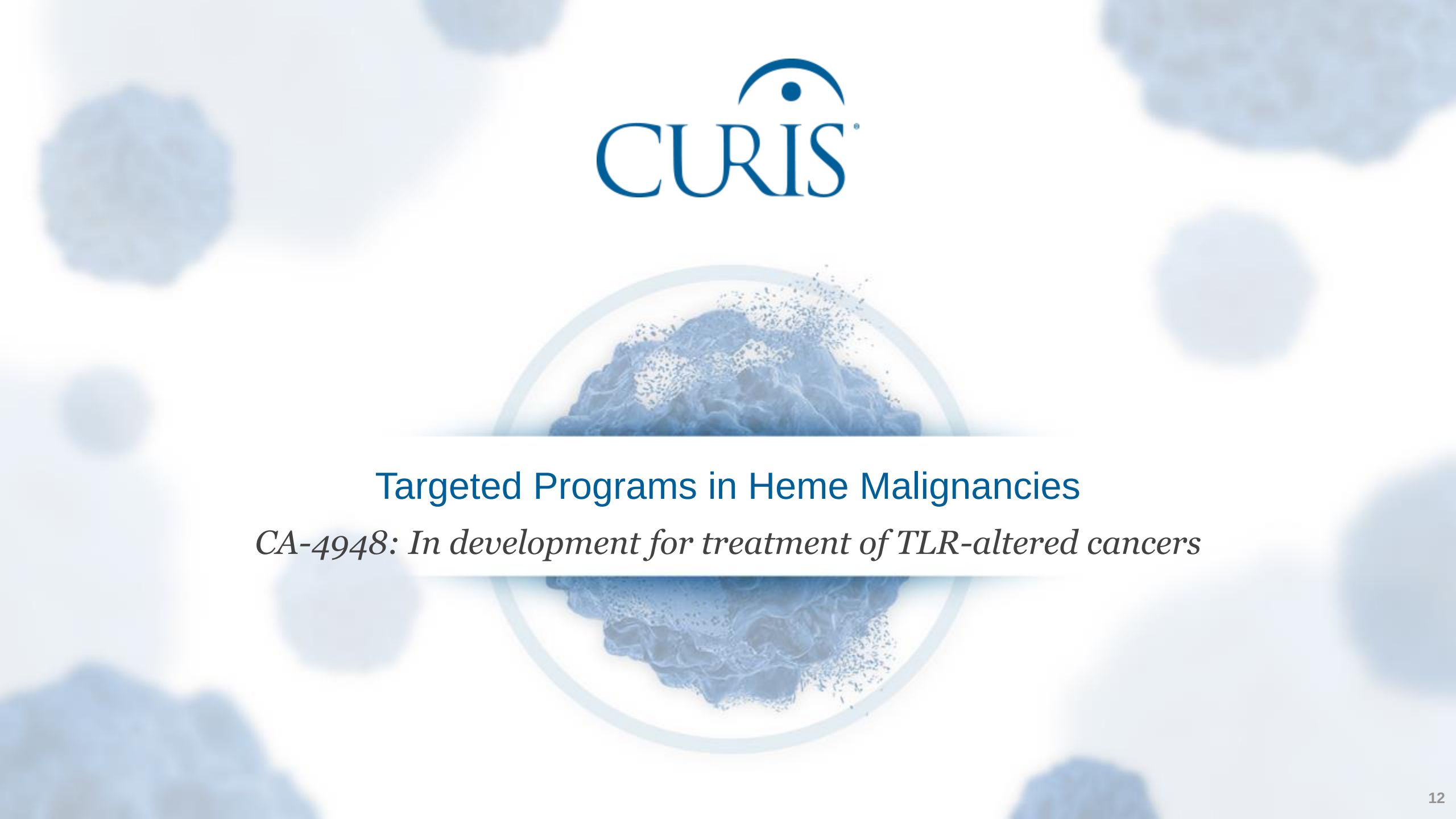
Objective

- Safety/tolerability during dose escalation
- Efficacy during expansion



Preliminary Phase 1 Tolerability Data Readout

- 11 patients enrolled as of December 6, 2019
- Generally well tolerated
- No drug-drug interaction that required dose modification of either agent



Targeted Programs in Heme Malignancies

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

CA-4948 Overview

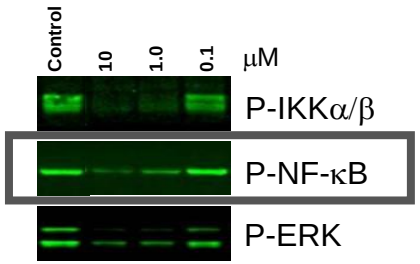
In Development for Patients with MYD88/TLR-Altered Disease

Profile	
Value Proposition	• 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
Population	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
Product Candidate Description	• Potent and orally bioavailable inhibitor of IRAK4 for treatment of MYD88-altered tumors and augmentation of BTK inhibition

Designed to be best-in-class IRAK4 inhibitor[1]

	Affinity
Kinase	K_d (nM)
IRAK4	23
IRAK1	12,000
IRAK2	>20,000
IRAK3	8,500

Potent suppressor of signal transduction[2]



Phospho-protein levels in AML cells after treatment with CA-4948

1) Data from Curis preclinical study
2) Booher et al. AACR 2017 (poster #1168)

CA-4948 Mechanism of Action

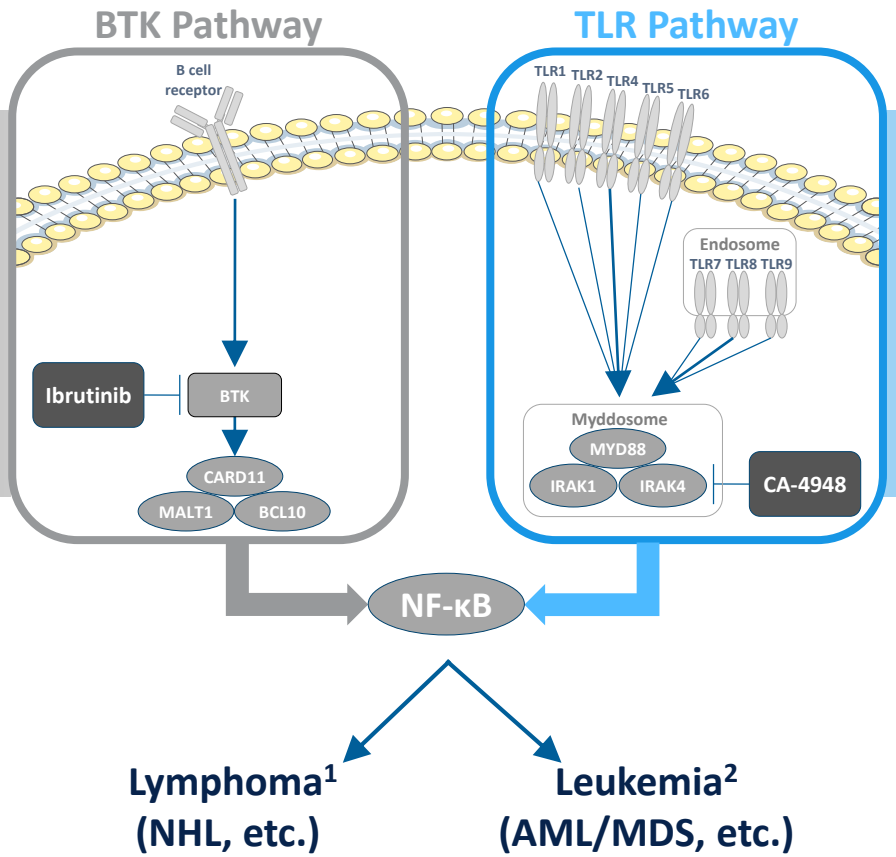
In Development for Patients with MYD88/TLR-Altered Disease

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[1]

BTK inhibition effective
- Ibrutinib is FDA approved



Oncogenic
- Dysregulation drives excessive B cell proliferation[2,3]

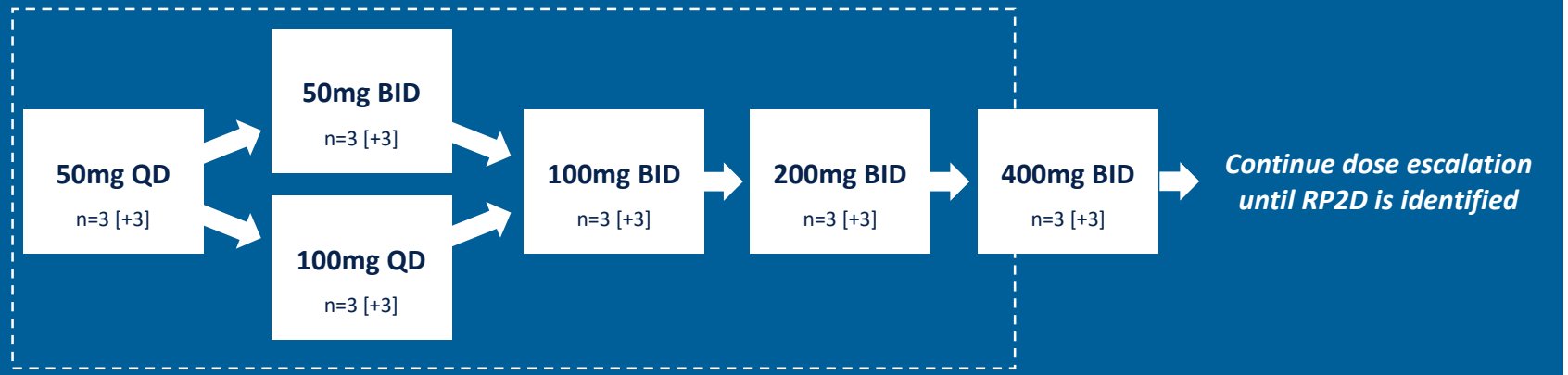
Dependent upon IRAK4
- Signaling requires myddosome, which requires IRAK4

Strong Synergy
- Inhibition highly synergistic with BTK inhibition

1) IMBRUVICA Package Insert. Rev 08/2018
2) Ngo et al. Nature. 2011 Feb 3;470(7332):115-9
3) Küppers et al. J Exp Med. 2015. 212(13): 2184
4) Smith et al. Nat Cell Biol 2019

CA-4948 Phase 1 Study in R/R Lymphoma

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



Preliminary Phase 1 Data Readout

- Generally well tolerated
- Favorable PK profile, PD, and anti-cancer activity
- 5 of 6 patients at 200mg-400mg cohorts have seen reduction

Patient Population

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

Treatment

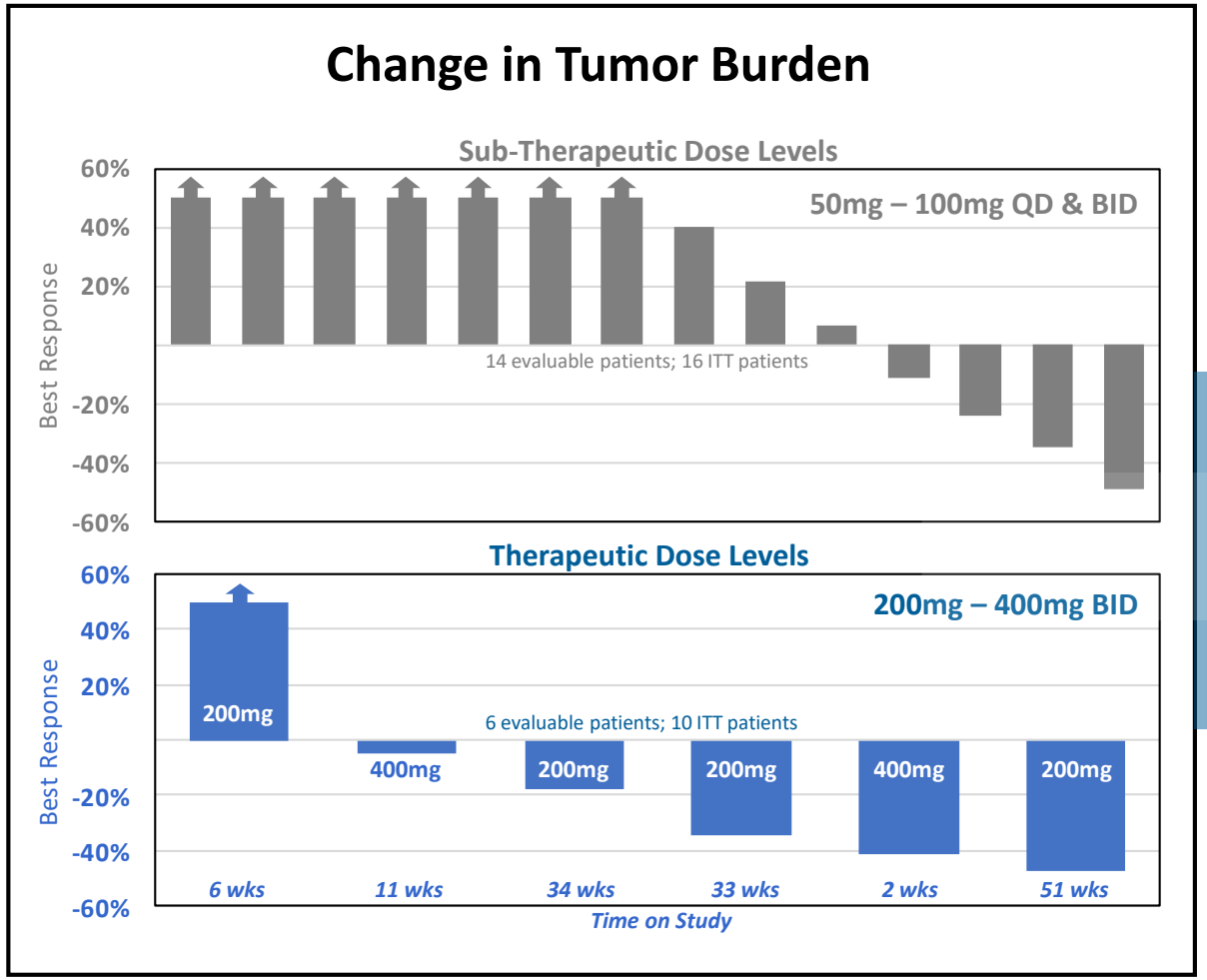
- Oral, once-daily (QD) or twice-daily (BID), dosing in continuous 21-day cycles

Objective

- Safety/tolerability during dose escalation
- Efficacy during expansion

CA-4948 Phase 1 Study in R/R Lymphoma

Dose Response Observed as Study Enrolls at Therapeutic Dose Levels

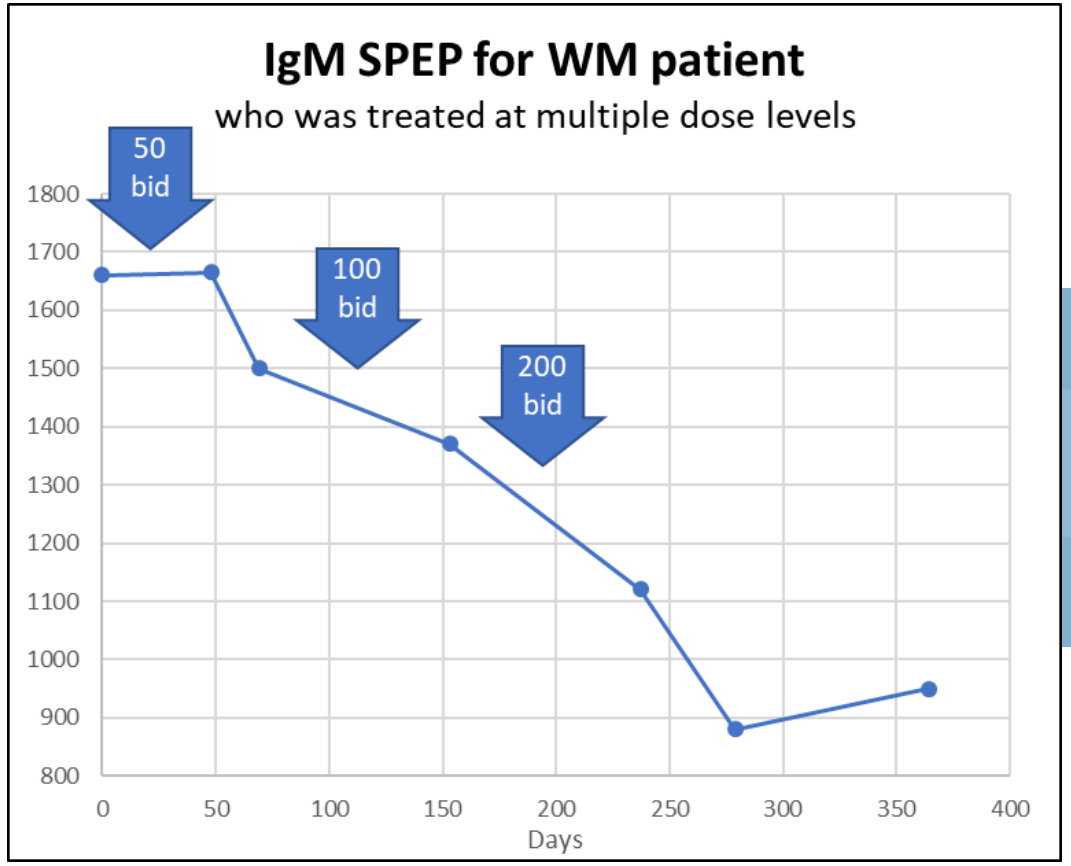


Dose Response Observed

5 of 6 patients experienced tumor reduction as dose increased to therapeutic levels (200mg-400mg)

CA-4948 Phase 1 Study in R/R Lymphoma

Dose Response Observed as Study Enrolls at Therapeutic Dose Levels



Dose Response Observed

Increased tumor reduction observed as patient increased dose

Two CA-4948 Ph1 Studies in NHL

Potent Preclinical Anti-Cancer Activity in MYD88-altered DLBCL

Preclinical Anti-Cancer Activity

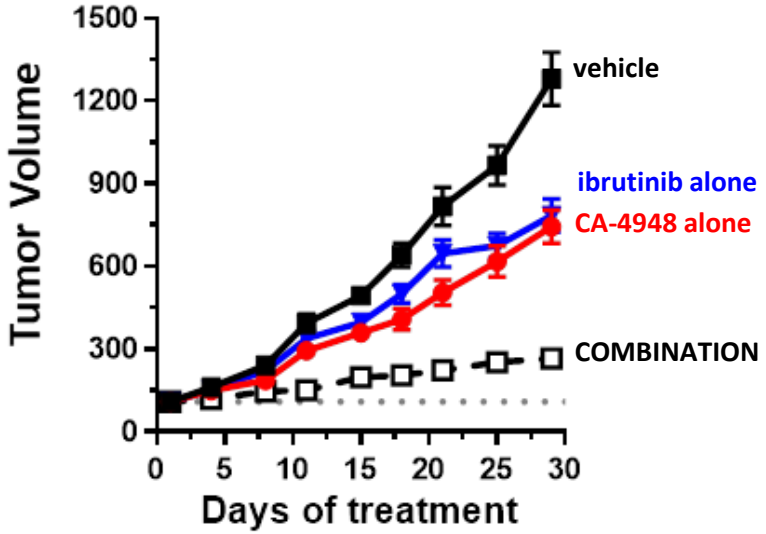
Potent as Single Agent

- 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
- Now that our single-agent study has reached therapeutic dose levels, we are initiating a combination study with BTKi to assess anti-cancer activity

Anti-cancer activity
in MYD88-altered DLBCL[1]
(OCI-Ly10)



1) Data from Curis preclinical study; Booher, et al. 4th Waldenstrom Roadmap Symposium

Additional CA-4948 Ph1 Study in AML/MDS

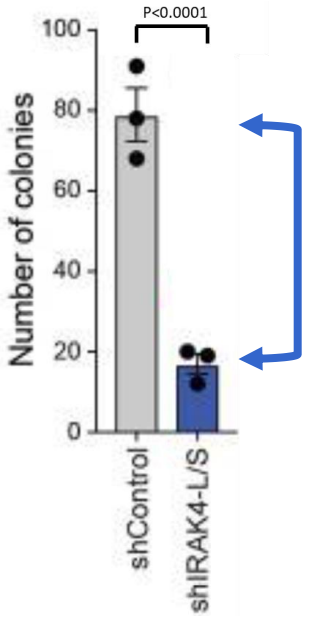
Potential Path to Accelerated Approval

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

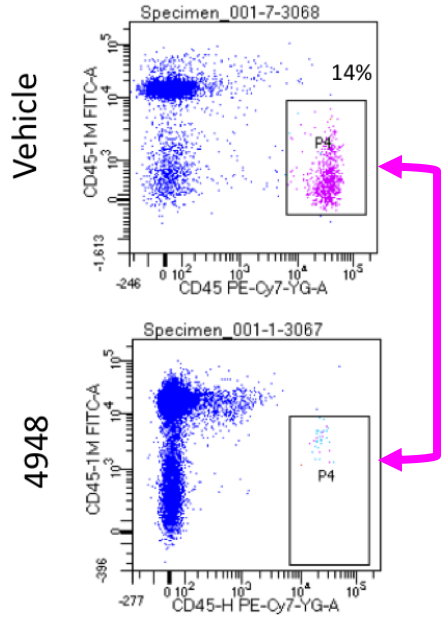


Blocking IRAK4-L reduces the formation of leukemia colonies in preclinical studies

Smith et al. Nat Cell Biol. 2019

CA-4948 Directly Targets IRAK4

Direct inhibition of the protein associated with disease
(IRAK4); we believe may offer a path to accelerated approval



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

Choudhary et al. AACR 2017

Monoclonal Antibody Immune Checkpoint Inhibitor

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

CI-8993 Overview

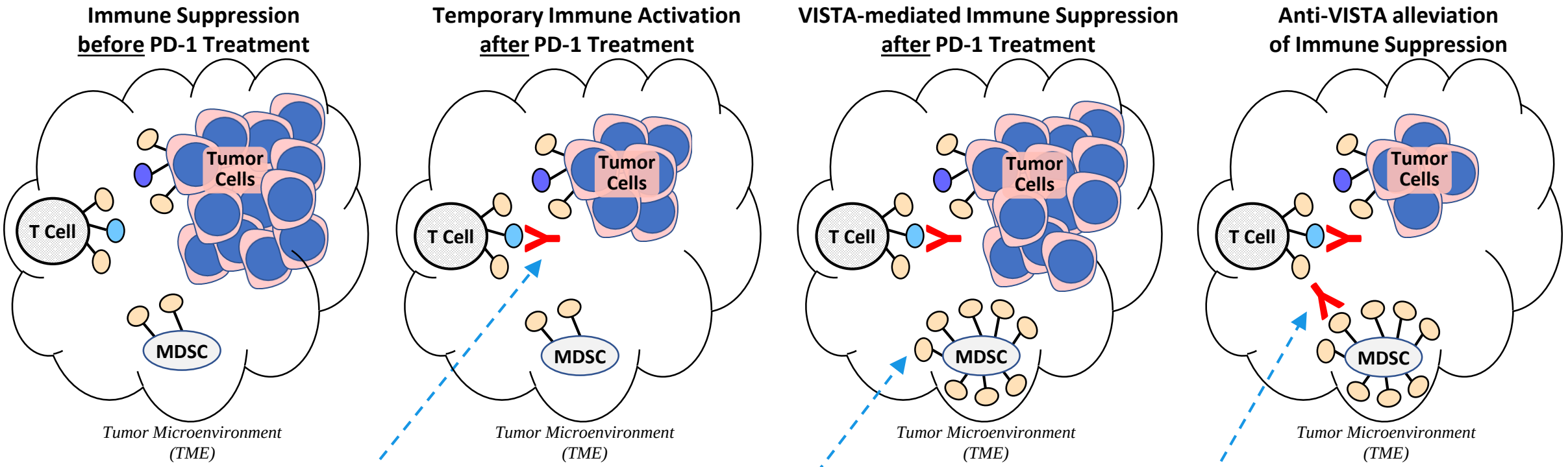
In Development for Patients with VISTA Expressing Cancers

Profile	
Value Proposition	• First-in-class monoclonal antibody antagonist of VISTA • No anti-VISTA antibodies currently in the clinic • Composition-of-matter IP extends into 2034
Population	• Patients with VISTA-expressing cancers, including Mesothelioma, NSCLC, and TNBC • All patients who will receive PD1/PDL1 or CTLA4 antibody therapy • Patients who have developed resistance to PD1/PDL1 or CTLA4 antibody therapy
Product Description	• Option for license entered into in January 2020 • Monoclonal antibody developed by ImmuNext/Janssen in partnership with Randy Noelle's lab at Dartmouth (the co-discoverer of VISTA)

CI-8993

Illustrative Example of VISTA's Role in Immune Suppression

In Pre-Clinical Testing[1], Blocking VISTA Results in:
Reduced suppressive mediators and enhanced antigen presentation of MDSCs
Altered chemotaxis (reduced traffic of MDSCs into the TME)



Blocking PD-1
activates T cells in some patients

Blocking PD-1
also causes up to 5x increase in VISTA expression and re-suppression of T cell activity[2]

Blocking VISTA
re-activates anti-tumor immune function[1]

VISTA
PD-1
PD-L1

[1] Data from ImmuNext preclinical studies
[2] Gao et al. Nature. 2017. 23: 551–555

Company

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	Fimepinostat: first-in-class suppressor of MYC *There are no drugs currently approved for MYC inhibition* 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*
Potential 2020 Catalysts	• Efficacy data for fimepinostat Phase 1 study in combination with venetoclax • Efficacy data for CA-4948 Phase 1 study in combination with BTK inhibitor • Efficacy data for 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

End of Corporate Presentation

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