Corporate Presentation

NASDAQ: CRIS

Forward Looking Statements

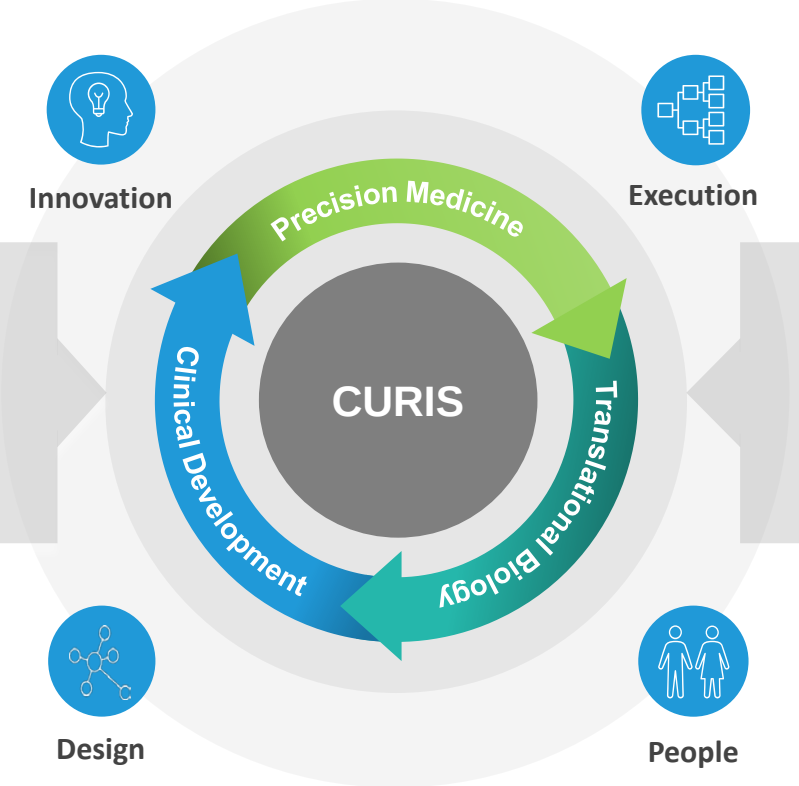
This presentation contains certain forward-looking statements about Curis, Inc. ("we," "us," or the "Company") within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Words such as "expect(s)," "believe(s)," "will," "may," "anticipate(s)," "focus(es)," "plans," "mission," "strategy," "potential," 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

Help patients suffering with cancer to live longer and healthier lives



Strategy

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

Overview

Investment Thesis	Curis develops novel, first-in-class, cancer therapeutics that have blockbuster 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* CA-170: first-in-class suppressor of VISTA *There are no drugs currently approved for VISTA inhibition*
Corporate	• Experienced management team with proven capabilities • Curis R&D pioneered the FDA-approved, first-in-class suppressor of the Hedgehog pathway (Erivedge®) partnered with and commercialized by Genentech and Roche for advanced basal cell carcinoma • Strong financial position, with cash balance of $35M as of June 30, 2019

Evolution of Curis

Progressing through Clinical Studies on the Path to Registration

2018

Regulatory Planning

- Work with investigators and FDA to determine clinical path
- Identify patient populations and open clinical studies

2019

Dose Escalation

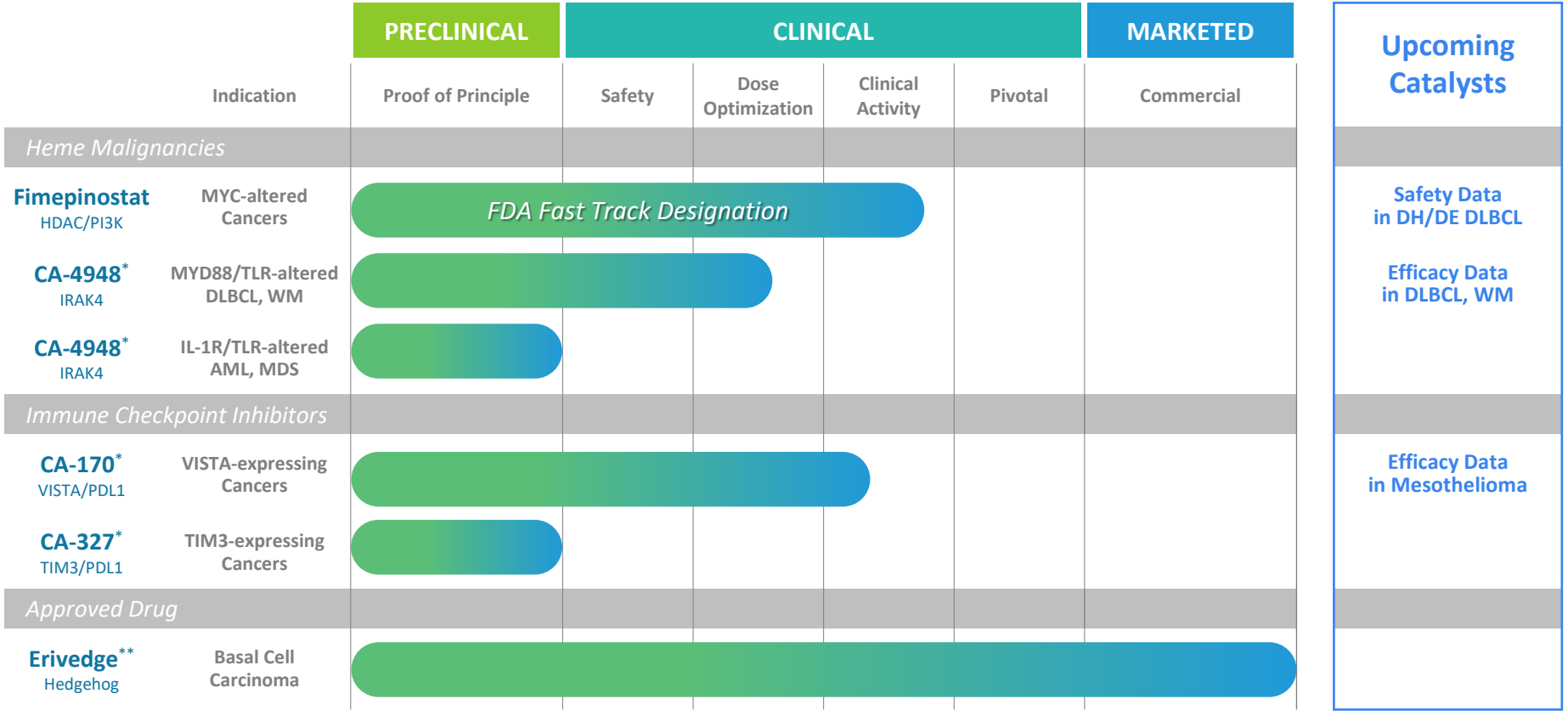
- Report Safety Data for Fimepinostat in DH/DE DLBCL
- Report Efficacy Data for CA-170 in Mesothelioma
- Report Efficacy Data for CA-4948 in MYD88 DLBCL/WM

2020

Expansion

- Expand studies with additional indications and combo-therapy
- Report efficacy data in dose expansion studies
- Outline plan for NDA filing and path to registration

Pipeline of Oncology Drug Candidates



* IP licensed from Aurigene

** IP licensed to Genentech (Curis receives royalty income)

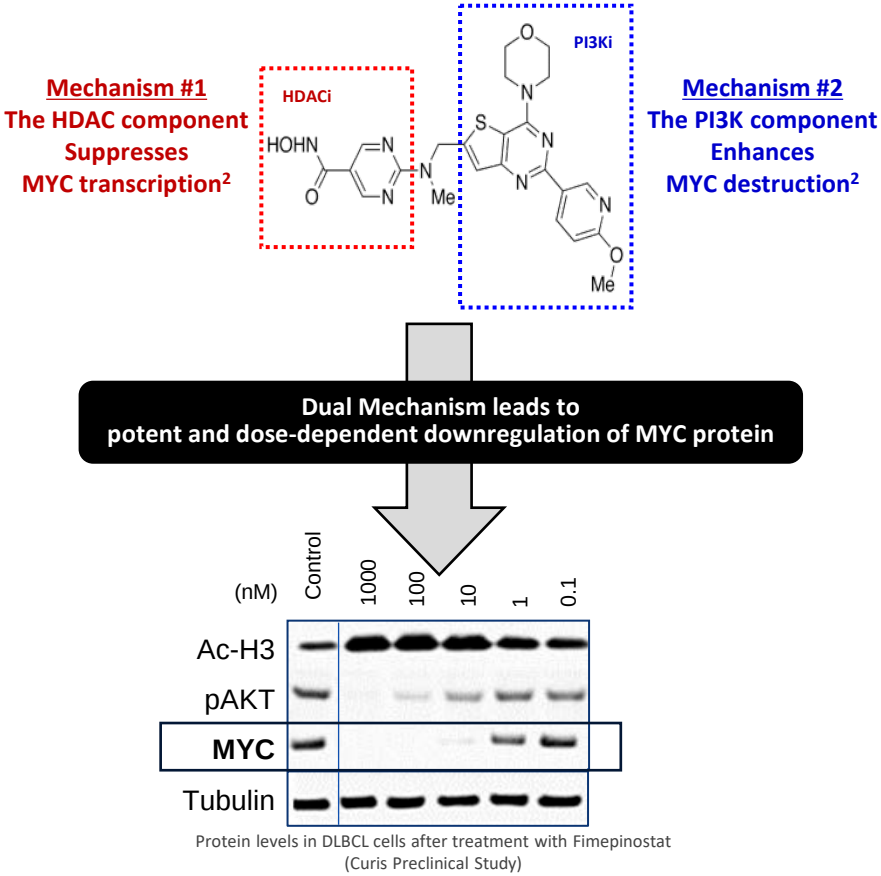
Targeted Drugs in Heme Malignancies

Fimepinostat: For treatment of MYC-altered cancers

Fimepinostat Overview

In development for patients with MYC-altered cancers

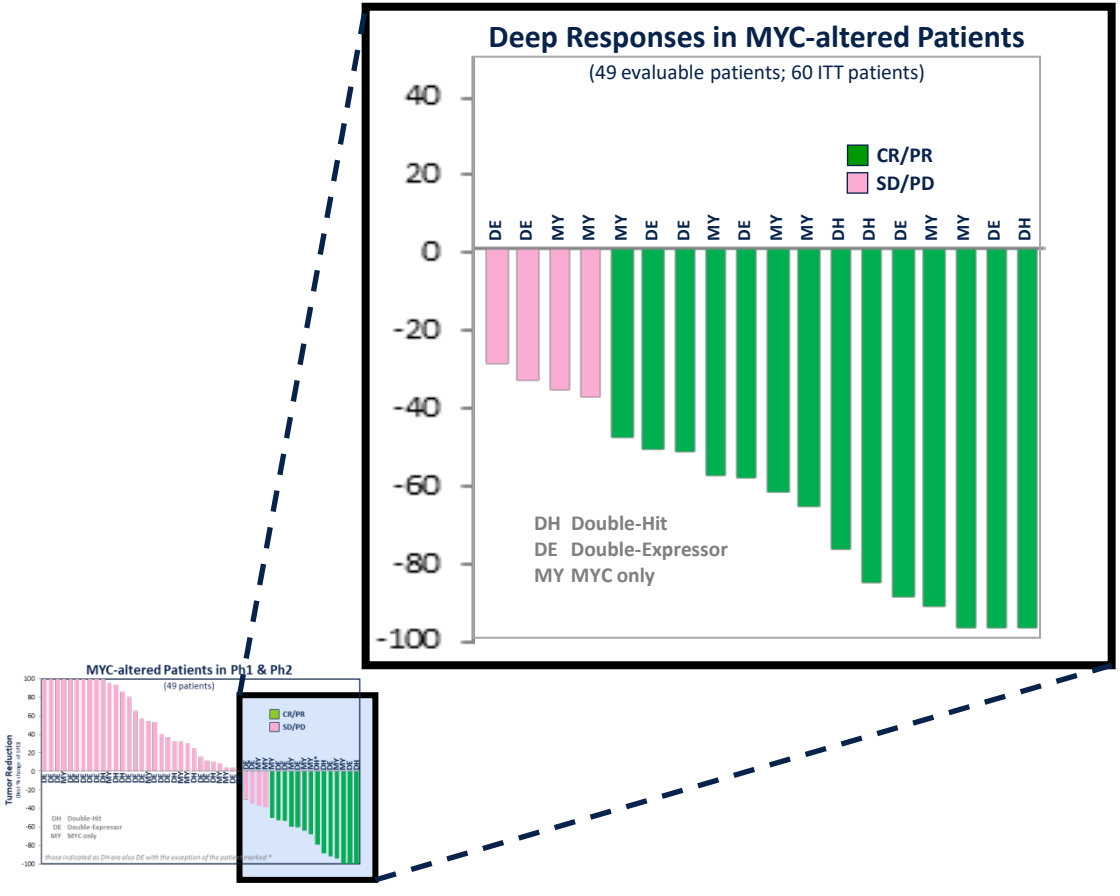
Profile	
Value Proposition	• First-in-class drug with demonstrated anti-tumor activity in MYC-altered patients • Composition-of-matter IP extends into 2032
Population	• Patients with MYC-altered cancer (>50% of all cancers are effected by MYC)[3]
Product Description	• Potent and orally bioavailable dual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



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) Gabay et.al. Cold Spring Harb Perspect Med. 2014

Clinical data provides strong rationale for development in MYC-altered lymphoma



Monotherapy Efficacy

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

- Following FDA review of efficacy data

Fimepinostat + venetoclax are highly synergistic in preclinical models

Fimepinostat + Venetoclax Are Ideal Combination Therapy Partners

Active single-agents in DLBCL

Fimepinostat = 23% ORR with 16.6 month DOR[1]

Venetoclax = 18% ORR[2]

Highly synergistic combination

- Combination index of < 0.1 at multiple doses[3]

High unmet need

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

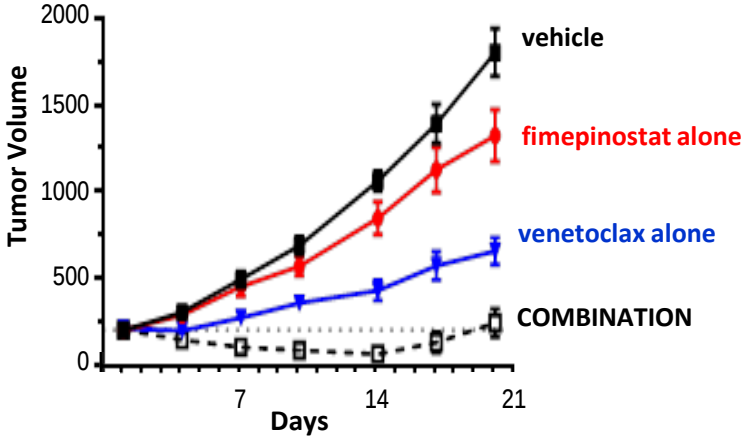
FDA: DHL is high unmet need

Regulatory path

- Possibility for accelerated approval in DHL or other orphan indications
- Full approval with randomized controlled trial

Fimepinostat + Venetoclax Are Highly Synergistic in Preclinical Studies

(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

Phase 1 combination study designed to demonstrate combination is safe

Patient Population

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

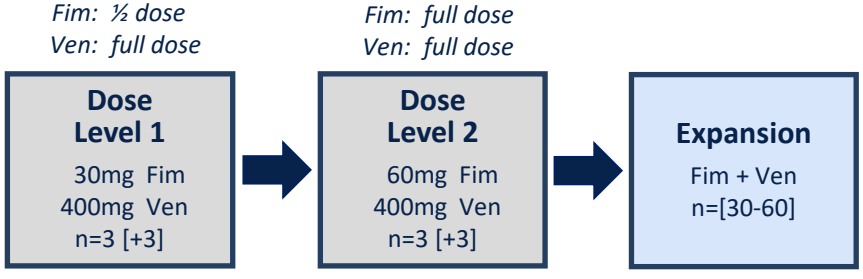
Treatment

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
  - Each single agent independently provides efficacy
  - Strong efficacy synergy in preclinical studies

Expected 2019 Catalyst Safety Data in 2H '19



Targeted Programs in Heme Malignancies

CA-4948: For treatment of TLR-altered cancers

CA-4948 Overview

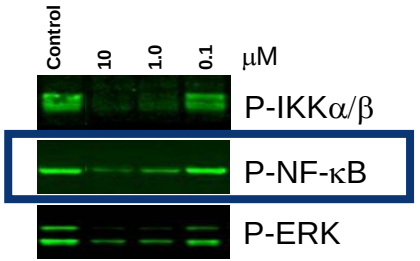
In development for patients with MYD88/TLR-altered disease

Profile	
Value Proposition	• First-in-class IRAK4 inhibitor in cancer • Specific malignancies have overactivity of the myddosome/TLR pathway (dependent upon IRAK4) • Composition-of-matter IP extends into 2035
Population	Lymphoma: IRAK4-dependent pathway activated; Ibrutinib-treated patients (strong synergy) Leukemia: Tumors with splicing mutations that overexpress IRAK4
Product 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)

Mechanism of Action

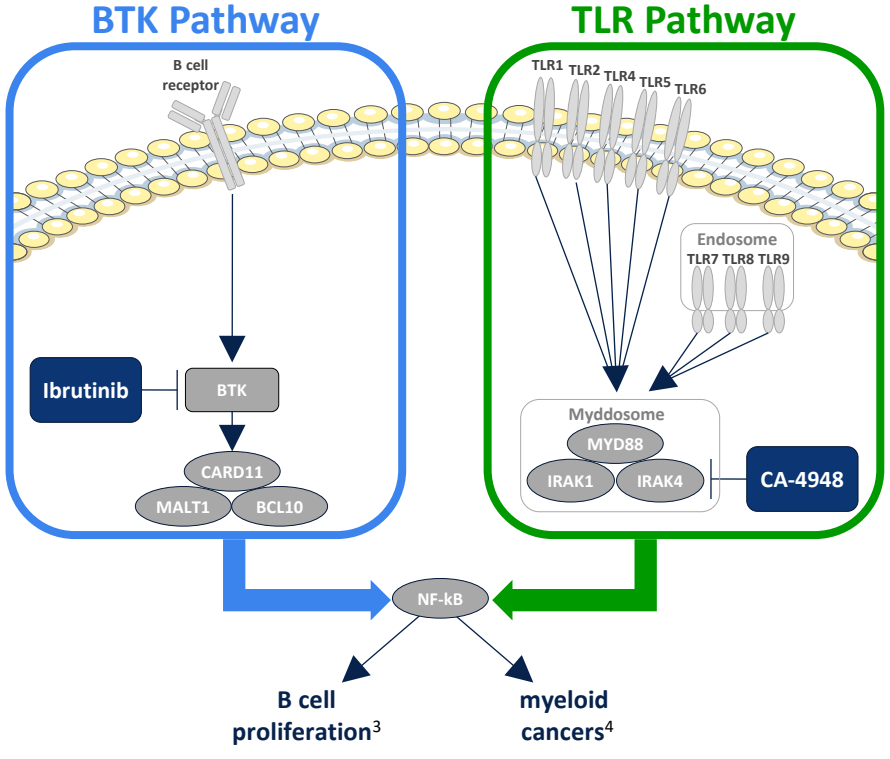
In development for patients with MYD88/TLR-altered disease

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
- Waldenström's macroglobulinemia
- MCL, MZL, CLL

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

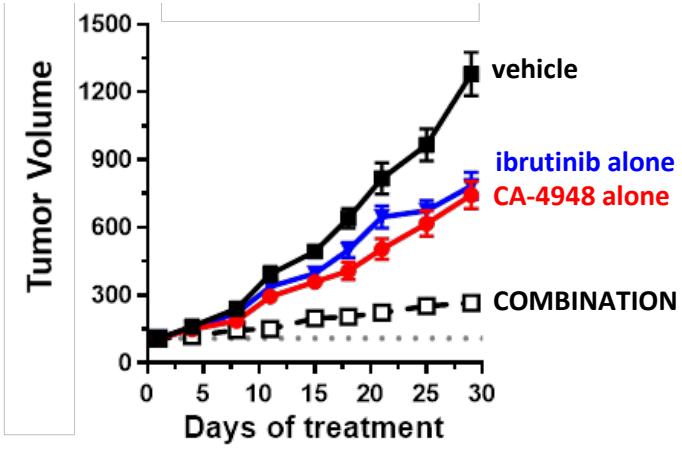
1) IMBRUVICA Package Insert. Rev 08/2018
2) Ngo et al. Nature. 2011 Feb 3;470(7332):115-9
3) Küppers et al. J Exp Med. 2015. 212(13): 2184
4) Smith et al. Nat Cell Biol 2019

Potent preclinical anti-tumor activity

in MYD88-altered DLBCL models

Anti-tumor activity in MYD88-altered DLBCL[1]

(OCI-Ly10)



Preclinical Efficacy

Potent as monotherapy

- Anti-tumor activity in MYD88-altered DLBCL

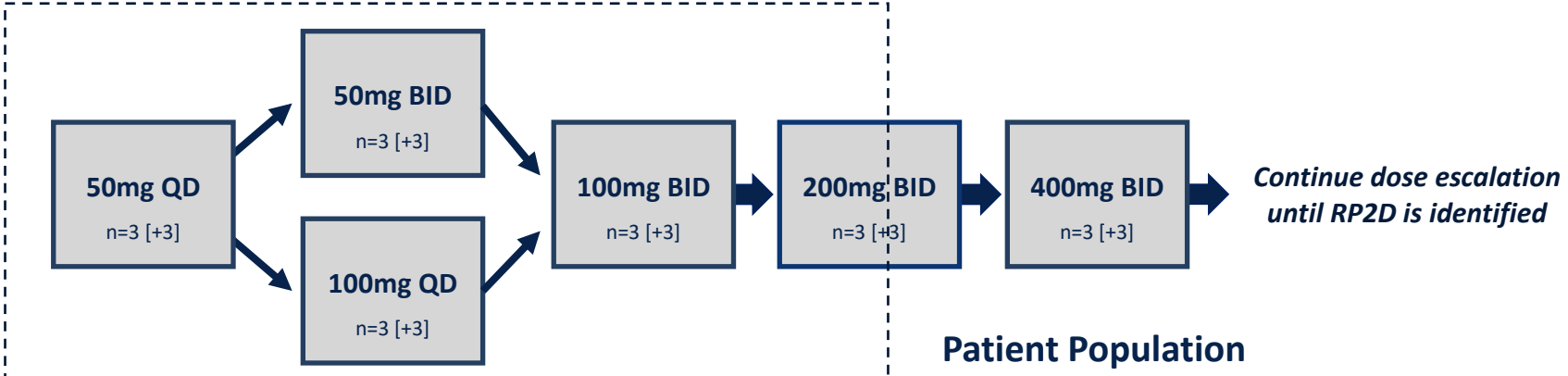
Strong Synergy

- Anti-tumor activity highly synergistic with BTK inhibition

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

CA-4948 Phase 1 Study in R/R Lymphoma

Initial clinical data show safety, PK, PD, and anti-tumor activity



Initial Data Readout

- Target inhibition
- Excellent pharmacokinetic profile
- Well tolerated
- Anti-tumor activity at multiple dose levels

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

Small Molecule Immune Checkpoint Inhibitor

CA-170: For treatment of VISTA/PDL1-expressing cancers

CA-170 Overview

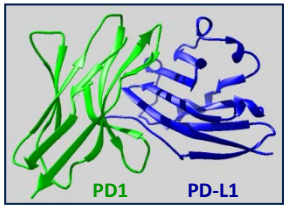
In development for patients with VISTA/PDL1-expressing cancers

Curis is the first to advance an oral small molecule checkpoint inhibitor into the clinic

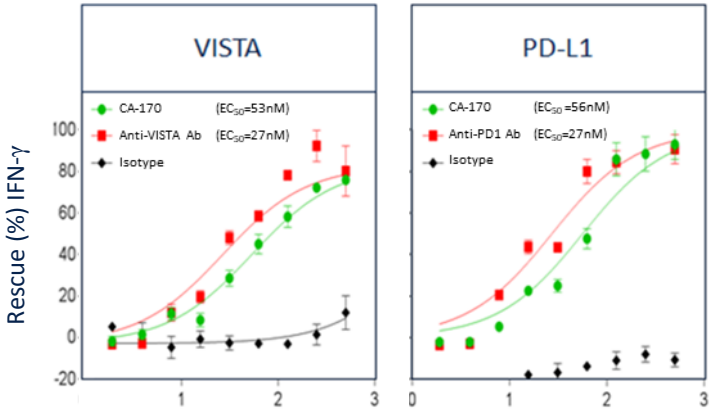
	Profile
Value Proposition	• First-in-class oral inhibitor of VISTA • Only anti-VISTA drug in the clinic • Composition-of-matter IP through 2034
Population	• Patients with VISTA-expressing cancers, including Mesothelioma, NSCLC, and TNBC • Patients whose disease progresses after treatment with immune checkpoint therapy
Product Description	• Orally available, small molecule targeting VISTA and PD-L1 immune checkpoints • Favorable safety profile demonstrated in 59 patients[2]

1) Lazorchak et al. AACR 2016
2) Data from Ph1 (NCT02812875) study

CA-170 binds to the receptor-ligand interaction site



Dose dependent activation of VISTA or PDL1inhibited human T cells *ex-vivo*[1]



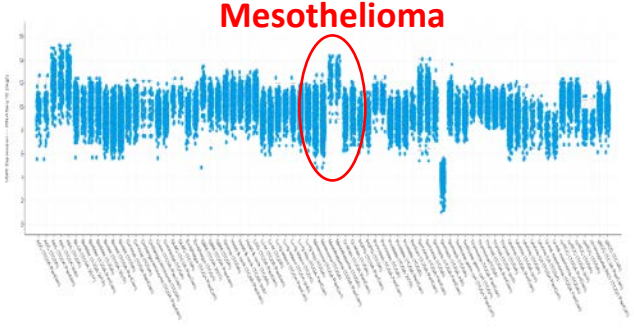
Test Compound Concentration (log nM)

IFN-γ production used as a marker for T cell activation

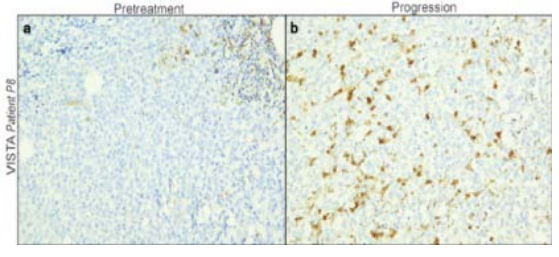
VISTA is highly expressed on tumor cells and infiltrating immune cells

- In addition to T cells, VISTA is expressed on tumor cells in subpopulations of certain cancers
  - ~ 90% of Mesothelioma cells[1]
  - ~ 20% of NSCLC cells[2]
  - ~ 14% of TNBC cells[3]
  - ~ 8% of gastric cancer cells[4]
- VISTA expression on immune cells is up-regulated after checkpoint inhibitor therapy
  - VISTA expression is induced on T Cells and macrophages in response to ipilimumab treatment[5]
  - ~ 60% of melanoma patients show increased VISTA expression at progression[6]

VISTA Gene Expression Analysis (TCGA)[7]



Melanoma[6]



1) Zauderer MG. ID 13232. WCLC. 2018; 2) Villarroel-Espindolai et al. Clinical Cancer Research. 2018; 3) Gruber et al. Poster 4749. AACR. 2018; 4) Boger et al. OncoImmunology. 2017. Volume 6. Number 4; 5) Gao et al. Nature. 2017. 23: 551–555; 6) Kakavand et al. Modern Pathology. 2017; 7) The Cancer Genome Atlas, 2018

CA-170 Phase 1 Efficacy Study in Mesothelioma

Expected 2019 Catalyst
Initial Data in 2H '19

Patient Population

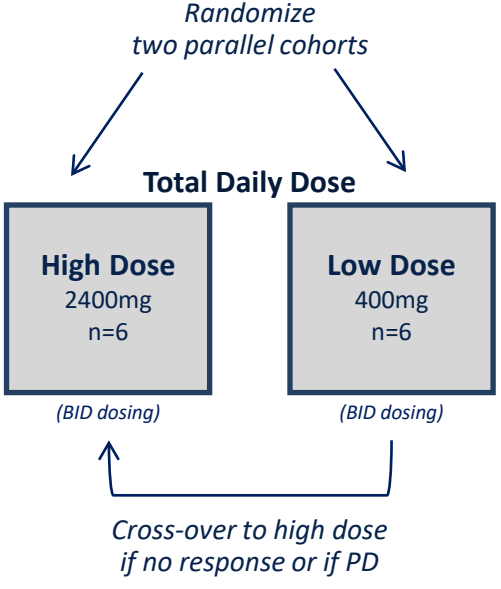
- Patients with Mesothelioma (High VISTA), that have been previously treated and whose disease has been deemed incurable
- 6 Study Sites (US and UK)

Objective

- Anti-cancer efficacy

Treatment

- Randomize to High Dose or Low Dose
- Crossover to High Dose, if no response or if disease progresses (PD)



Summary

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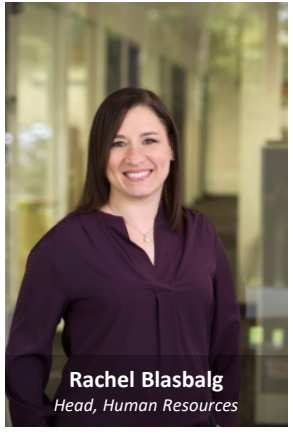
Robust Pipeline	Fimepinostat: first-in-class suppressor of MYC *There are no drugs currently approved for MYC inhibition* CA-4948: first-in-class suppressor of the TLR Pathway *There are no drugs currently approved that block the entire TLR pathway* CA-170: first-in-class suppressor of VISTA *There are no drugs currently approved for VISTA inhibition*
Upcoming Milestones In 2H 2019	• Safety data in fimepinostat-venetoclax combination study • Efficacy data in CA-4948 study in DLBCL/WM • Efficacy data in CA-170 study in Mesothelioma

Curis Leadership Team



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