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.

Evolution of Curis

Progressing from Pipeline Building to Clinical Execution

Curis has built a novel pipeline with three first-in-class programs.
In 2019, our focus has shifted to efficient clinical execution and the reporting of clinical data.

2014-2017

Pipeline Building

- Identify targets of interest and design first-in-class molecules to hit them
- Establish collaborations to in-license novel technology

2018

Regulatory Planning

- Consult with investigators and regulatory agencies to determine clinical path
- Identify the right patient populations for our programs

2019

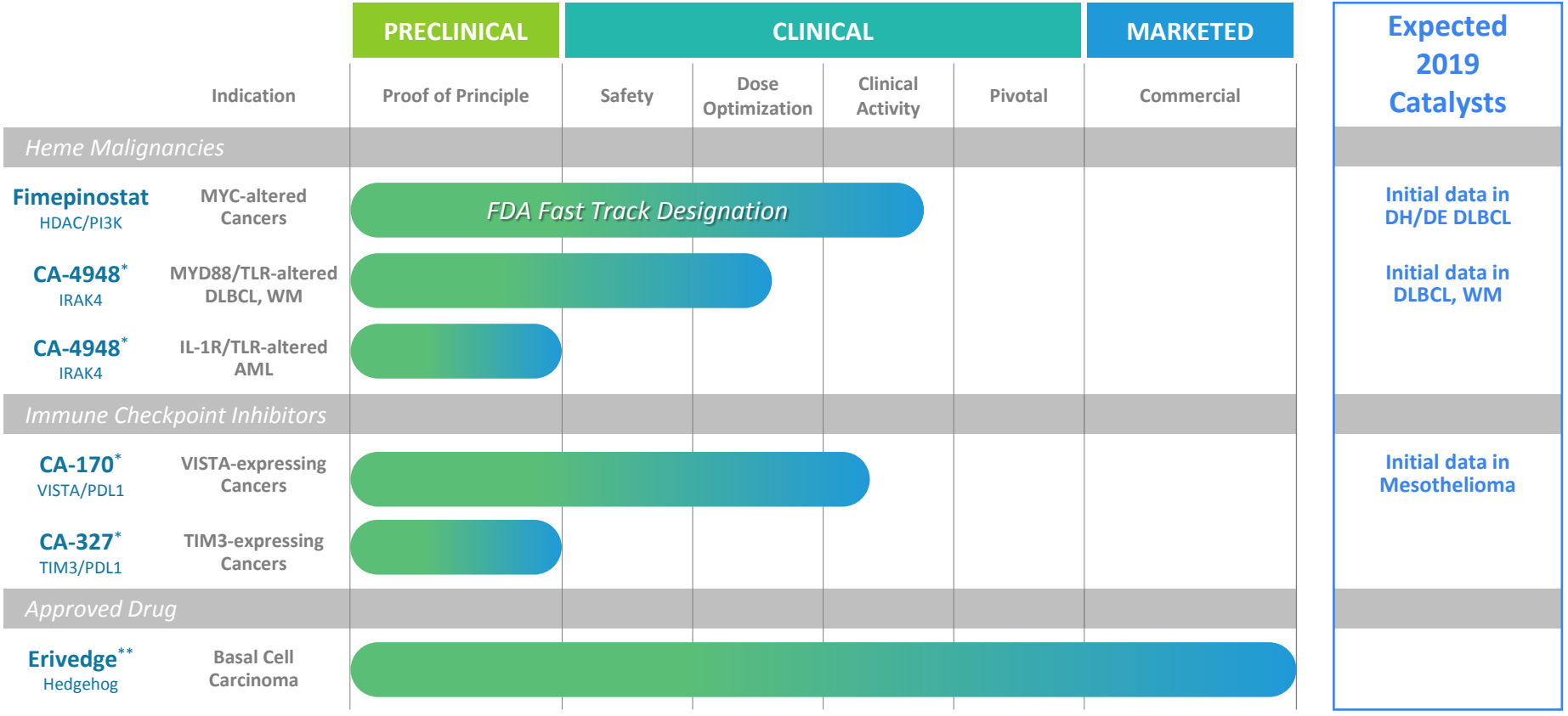
Clinical Execution

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

Note: This slide contains forward-looking statements about Curis's potential 2019 data catalysts within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. The potential 2019 data catalysts 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..

Pipeline of Oncology Drug Candidates

First-in-Class, orally available, targeted small molecules



* IP licensed from Aurigene

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

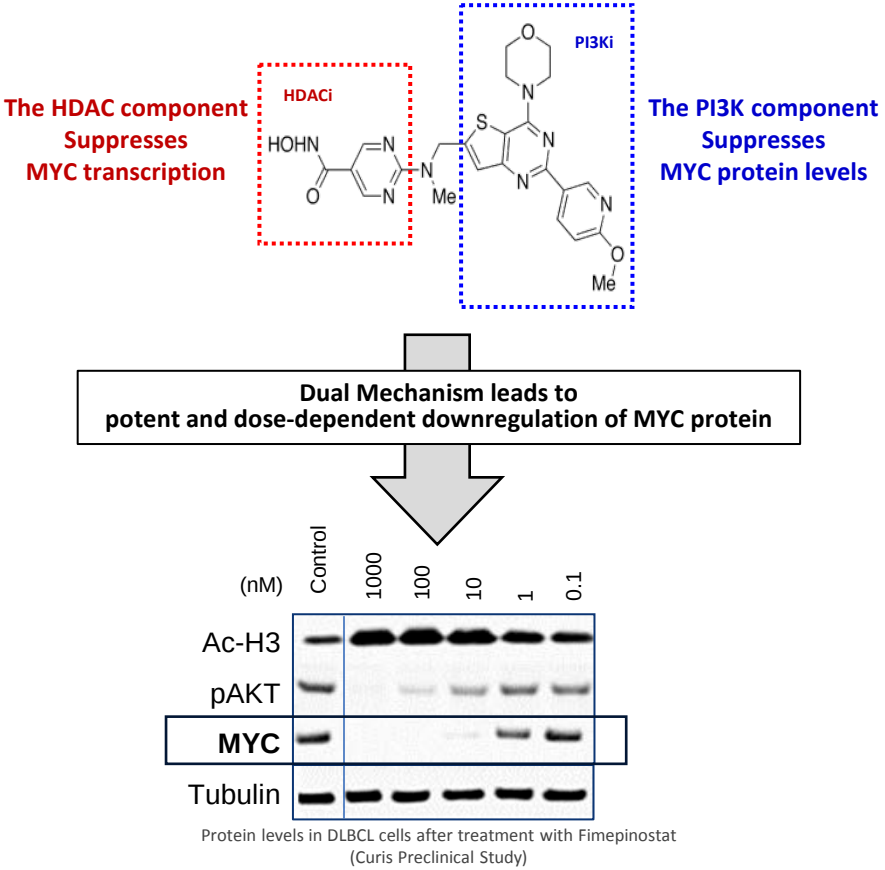
Targeted Drugs in Heme Malignancies

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	• MYC-altered cancers
Product Description	• Potent and orally bioavailable dual inhibitor of HDAC and PI3K enzymes[1] • HDAC component inhibits transcription of MYC and MYC-regulated genes[2] • PI3K component results in ubiquitin mediated MYC protein degradation[2] • Favorable safety profile in over 200 patients

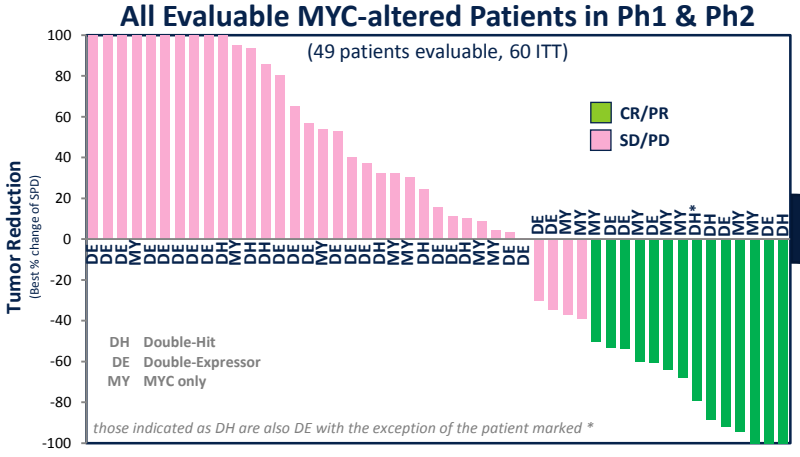


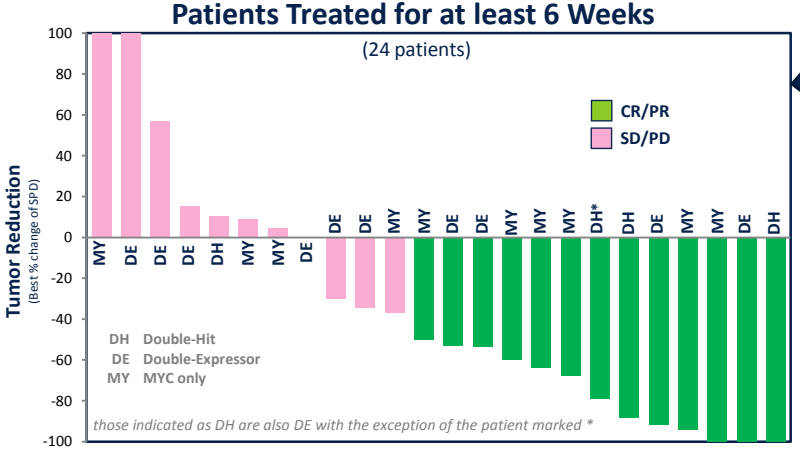
Protein levels in DLBCL cells after treatment with Fimepinostat (Curis Preclinical Study)

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

Fimepinostat Clinical Data

Fast Track designation received after data reviewed by the FDA





Ph 1/2 data show clear efficacy in MYC-altered disease

- FDA review of data led to Fast Track designation
- 8 CR and 6 PR, incl 8 patients with DH/DE
- Responses are durable (mDoR is 13.6 months)

Strong Rationale for Combination Strategy

Combining fimepinostat with another anti-lymphoma agent may allow more patients to remain on drug for ≥ 6 weeks (long enough for MYC suppression to provide benefit)

Patients able to stay on therapy ≥ 6 weeks achieved higher ORR

- Fimepinostat targets reduction in MYC activity and provides increased efficacy with multi-cycle exposure

Note:
Tumor Reduction Data from Ph1 (NCT01742988) and Ph2 (NCT02674750) studies

Regulatory Strategy

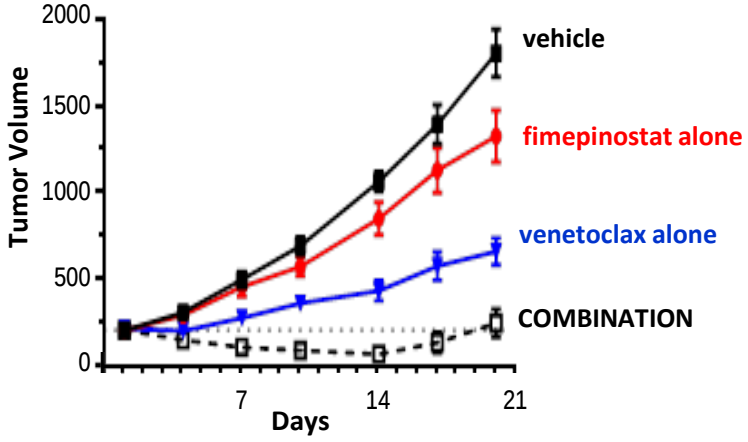
Charting the fastest, highest probability path to FDA approval

Fimepinostat + Venetoclax Are Ideal Combination Therapy Partners

- Targeting both MYC (w/fimepinostat) and BCL2 (w/venetoclax) enables a regulatory path for double-hit lymphoma, an NCCN-designated and FDA-acknowledged disease of high unmet need
- Fimepinostat and venetoclax have different mechanisms of action and are highly synergistic in preclinical models[1]
- Venetoclax has already been tested in DLBCL as a monotherapy (as a monotherapy, venetoclax had an 18% ORR[2])
- Venetoclax may enhance fimepinostat's 23% ORR[3] due to synergy or by delaying disease progression, extending the window for fimepinostat's epigenetic MYC-based mechanism of action

Fimepinostat + Venetoclax Highly Synergistic in Preclinical Studies

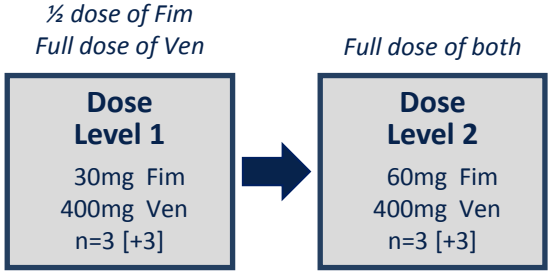
(DH DOHH-2 DLBCL model)[4]



1) Sun et al. Blood. 2016. 128:4184
2) Davids et al. JCO. 2017. 35:826
3) 14 PR/CR out of 60 patients in Ph1 & Ph2 (23% ORR)
4) Data from Curis preclinical study

Regulatory Plan

Initial Combination Study with Venetoclax to show combination is safe



Expected 2019 Catalyst
Initial Data in 2H '19

Key Catalyst for 2019
is to show the combination is safe/tolerable

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 is Safety/Tolerability

- Efficacy of fimepinostat, as a monotherapy, is already higher than chemo-based regimens used in 3rd line treatment
- Given different mechanisms of action, it is presumed that efficacy of the combination may be even higher than monotherapy
- Key question when combining any drug with venetoclax is safety, given the drug's well known risk profile

Targeted Programs in Heme Malignancies

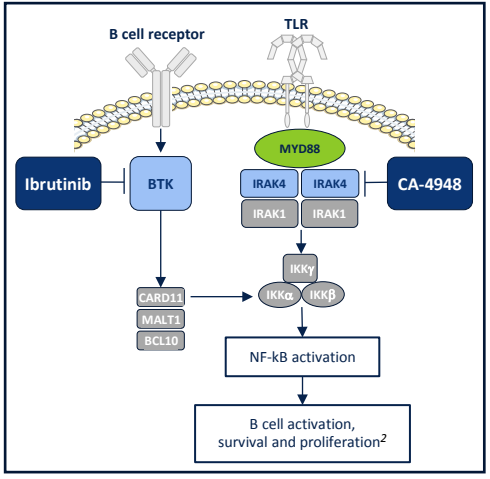
CA-4948: For treatment of TLR-altered cancers

CA-4948 Overview

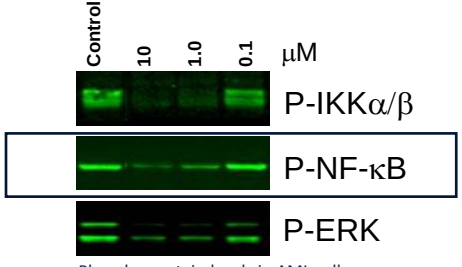
In development for patients with MYD88/TLR-altered disease

Profile	
Value Proposition	• First-in-class IRAK4 inhibitor in cancer • A defined subset of malignancies are driven by overactivity of the myddosome in the TLR pathway, which is dependent upon IRAK4 • Composition-of-matter IP extends into 2035
Population	• Lymphoma: DLBCL, Waldenström's macroglobulinemia, and patients with MYD88-altered disease • Leukemia: TLR-altered AML
Product Description	• Potent and orally bioavailable inhibitor of IRAK4 for treatment of MYD88-altered tumors

Inhibition of this pathway was validated in Waldenström's macroglobulinemia with ibrutinib[1]



Potent and direct inhibitor of NF-kB signal transduction[3]



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

Potent and selective inhibitor of IRAK4 enzyme[4]

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

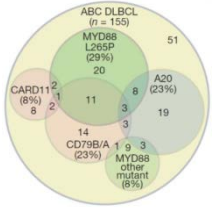
1) IMBRUVICA Package Insert. Rev 08/2018
2) Küppers et al. J Exp Med. 2015. 212(13): 2184.
3) Booher et al. AACR 2017 (poster #1168)
4) Data from Curis preclinical study

CA-4948 Preclinical Data

Potent anti-tumor activity in MYD88-altered DLBCL models

Overlap of Gene Mutations in ABC-DLBCL
(Image adapted from Ngo et al. Nature. 2011.)

MYD88 alterations are prevalent in 29% of ABC-DLBCL[1]

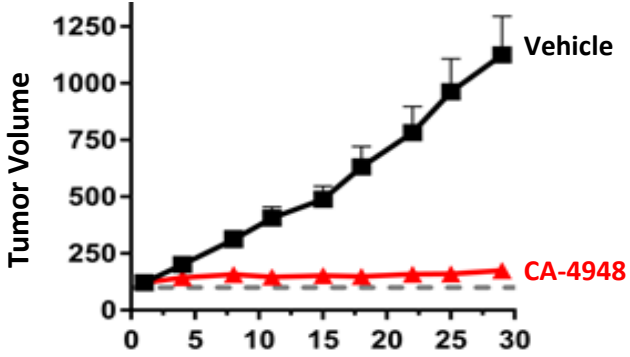


CA-4948 exhibits potent anti-tumor activity in preclinical studies

- MYD88 mutation is associated with constitutive activation of NF-κB signaling[1]
- NF-κB and JAK kinase signaling promote malignant cell survival in ABC-DLBCL[1]
- ABC subtype of DLBCL is the most difficult subtype to treat, despite recent advances in therapy[1]

Anti-tumor Activity in MYD88-altered DLBCL in preclinical studies[3]

(OCI-Ly10)

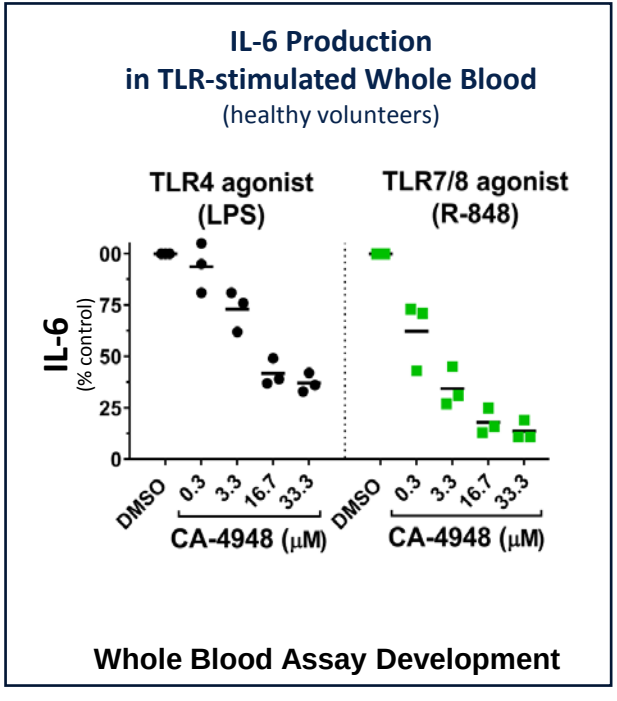


1) Ngo et al. Nature. 2011 Feb 3;470(7332):115-9
2) Caner et al. Genet Test Mol Biomarkers 19, 372–378
3) Data from Curis preclinical study; Booher, et al. CA-4948 IWWM 2018 Poster

CA-4948 Clinical Data

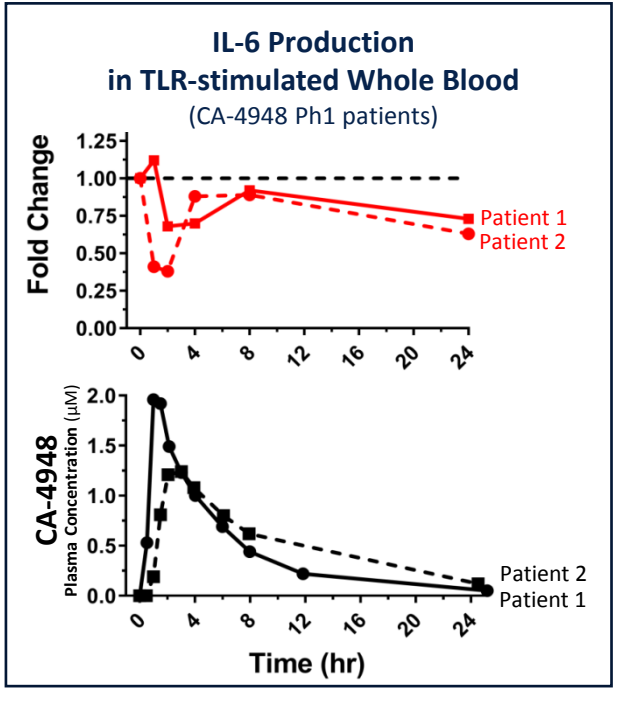
Early clinical data indicated successful target inhibition

In whole blood from healthy volunteers, cytokine production dropped when incubated with CA-4948



(Curis internal analysis performed ex-vivo, n=3)

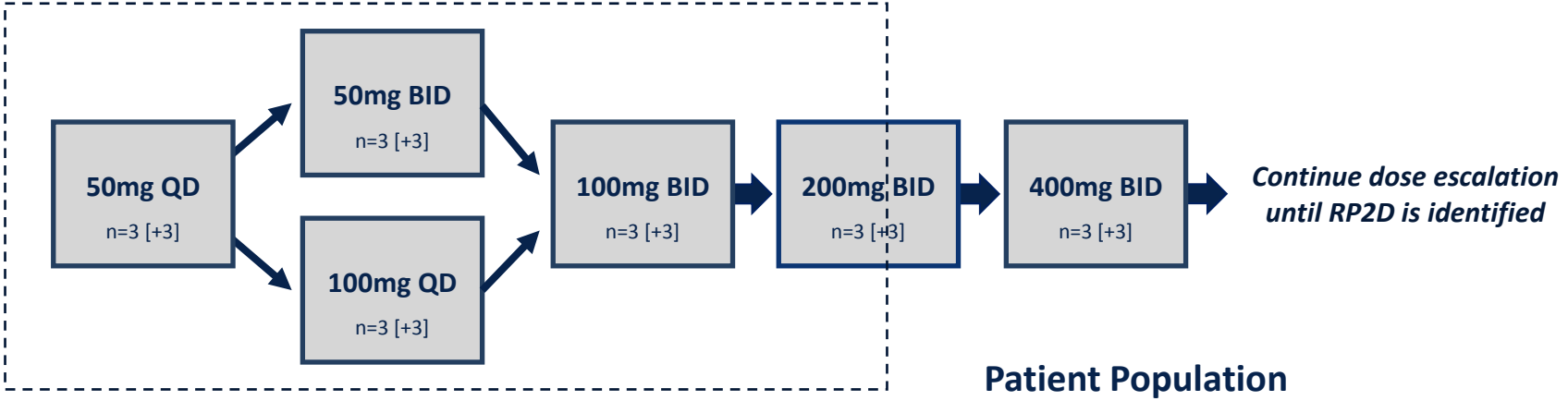
In whole blood from patients treated with CA-4948, cytokine production dropped, mirroring drug exposure



(Curis internal analysis performed ex-vivo, n=2)

CA-4948 Clinical Plan

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



Mid-Study Readout of Initial Data

Mid-Year 2019 Update

Dataset analyzed midway thru 200mg BID cohort show PD target inhibition with dose-proportional increases in PK exposure and Anti-tumor activity at multiple dose levels

Patient Population

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

Objective

- Safety, PK, PD, Anti-tumor activity, Recommended Phase 2 Dose (RP2D)

Treatment

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

Small Molecule Immune Checkpoint Inhibitor

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

- *CA-170 is the first oral small molecule targeting VISTA (and also only anti-VISTA drug in the clinic)*
- *CA-170 is the first oral small molecule targeting PD-L1 in the clinic*

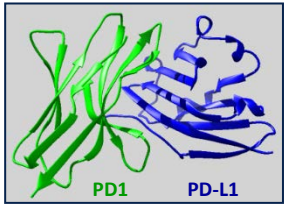
CA-170 Overview

In development for patients with VISTA/PDL1-expressing cancers

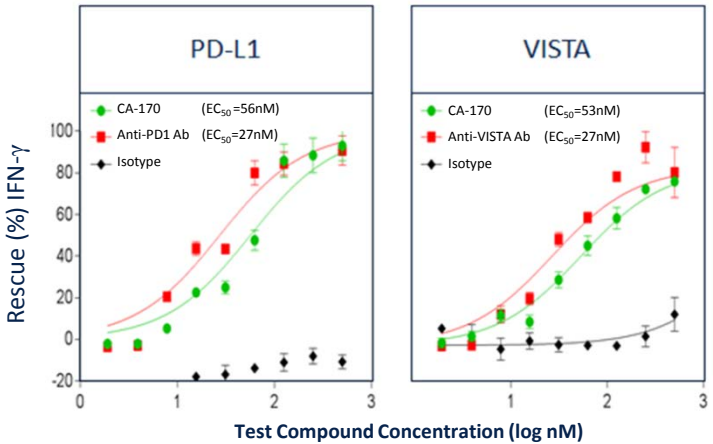
	Profile
Value Proposition	• First-in-class oral inhibitor of VISTA • First-in-class oral inhibitor of PD-L1 • Composition-of-matter IP through 2034
Population	• Patients with VISTA-expressing cancers • Patients with tumors not addressable by anti-PD1 /PD-L1 treatment alone
Product Description	• Orally 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 PD-L1 or VISTA-inhibited human T cells *ex-vivo*[1]



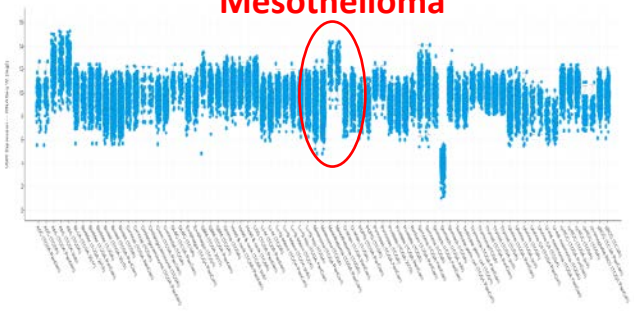
IFN-γ production used as a marker for T cell activation

VISTA Targeting Strategy

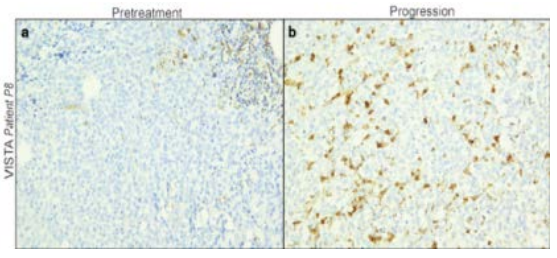
VISTA is highly expressed on tumor cells and infiltrating immune cells

- 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

Clinical Plan for Efficacy Study in Mesothelioma

Expected 2019 Catalyst
Initial Data in 2H '19

Patient Population

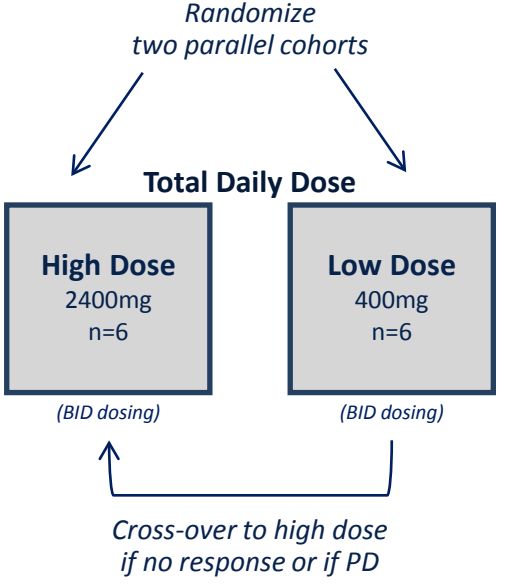
- Patients with Mesothelioma (High VISTA), that is incurable and previously treated
- 6 Study Sites (US and UK)

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

Financial Data as of June 30, 2019	
$35.3M	Cash & Marketable Securities
33.2M	Basic Shares Outstanding
39.1M	Fully Diluted Shares Outstanding

Expected 2019 Catalysts	
Fimepinostat – Venetoclax Combination	Initial safety data in DH/DE DLBCL (HGBL)
CA-4948	Update of efficacy data at an upcoming medical conference
CA-170	Initial efficacy data in Mesothelioma

Fully Diluted Shares = 33.2M Basic Shares + 5.9M Options

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