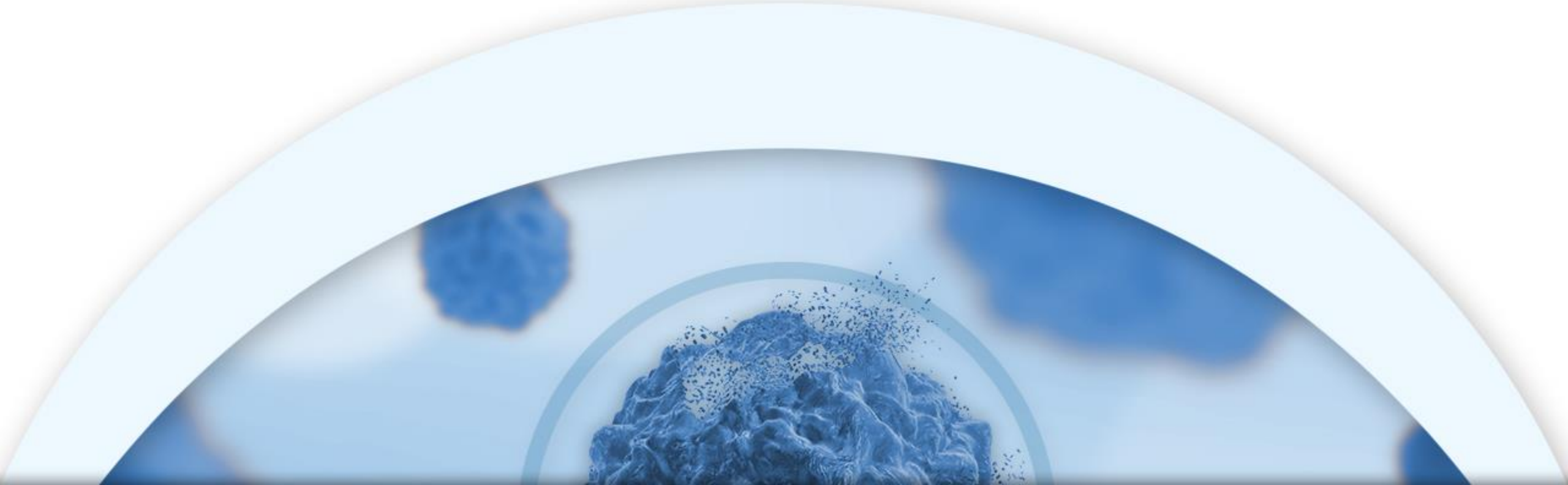
Corporate Presentation

NASDAQ: CRIS



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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.

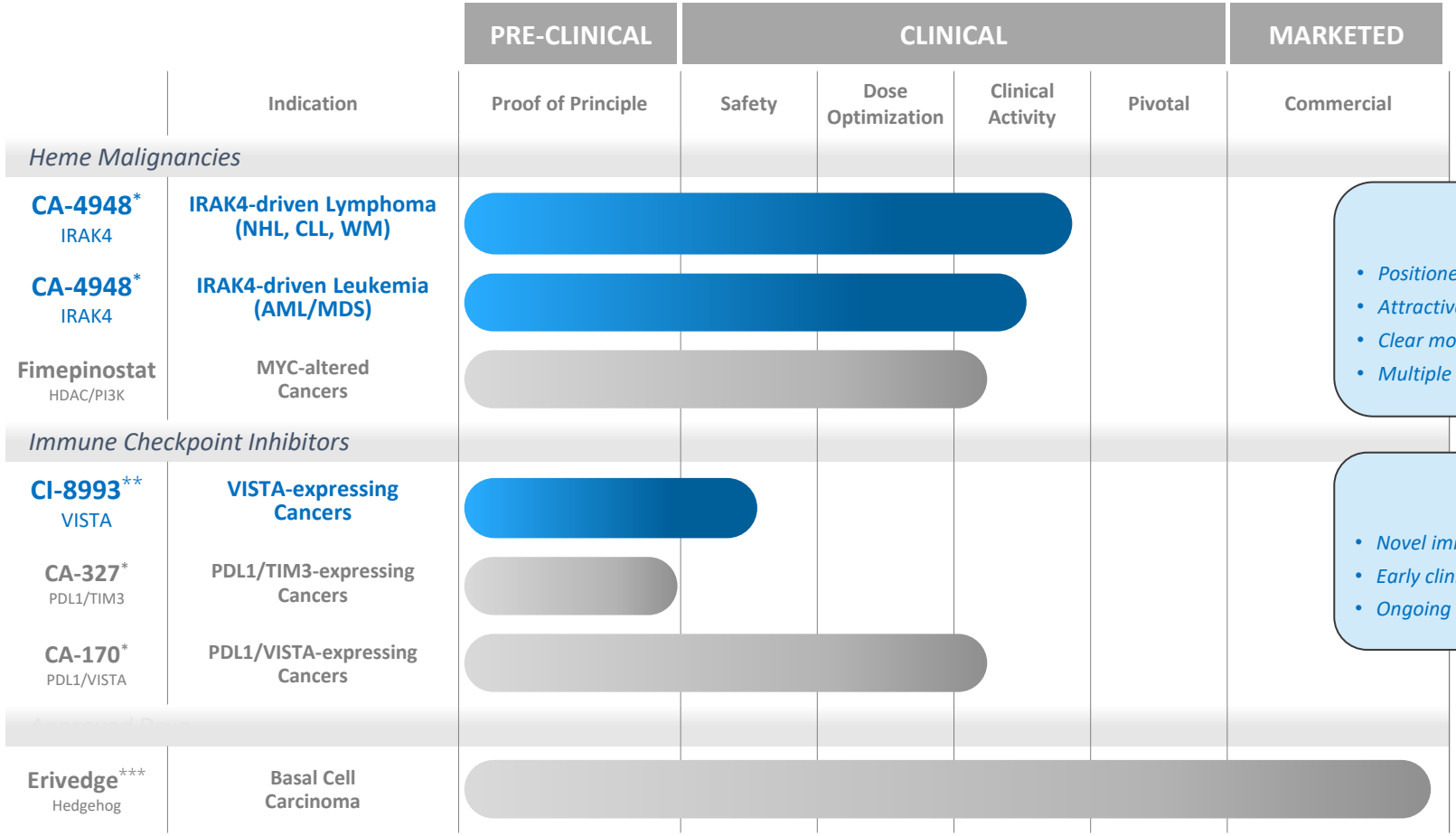
Corporate Overview

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 *Cash and investments of approximately $161M as of June 30, 2021; cash runway into 2024*
Robust Pipeline	CA-4948: first-in-class inhibitor of IRAK4 in oncology *There are no drugs currently approved for IRAK4 inhibition in oncology* CI-8993: first-in-class antagonist of VISTA *There are no drugs currently approved for VISTA inhibition*
Upcoming Milestones	YE 2021: Report safety data in CI-8993 (VISTA) YE 2021: Report additional data in CA-4948 in AML/MDS (spliceosome population) 2022: Discuss potential for rapid approval path for CA-4948 with FDA

Pipeline

Curis develops novel, first-in-class cancer drugs



Two programs are the focus of this presentation

IRAK4 (CA-4948)

- *Positioned to become the cornerstone agent in heme malignancies*
- *Attractive PK/PD exposure correlating with 98% target inhibition*
- *Clear monotherapy activity with consistent marrow blast reduction and complete responses*
- *Multiple clinical paths in targeted and broader B cell populations*

VISTA (CI-8993)

- *Novel immune checkpoint with unique role in T cell activation*
- *Early clinical demonstration of immune activation biology in solid tumor cancers*
- *Ongoing dose escalation study to establish safety, activity, and recommended phase 2 dose*

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

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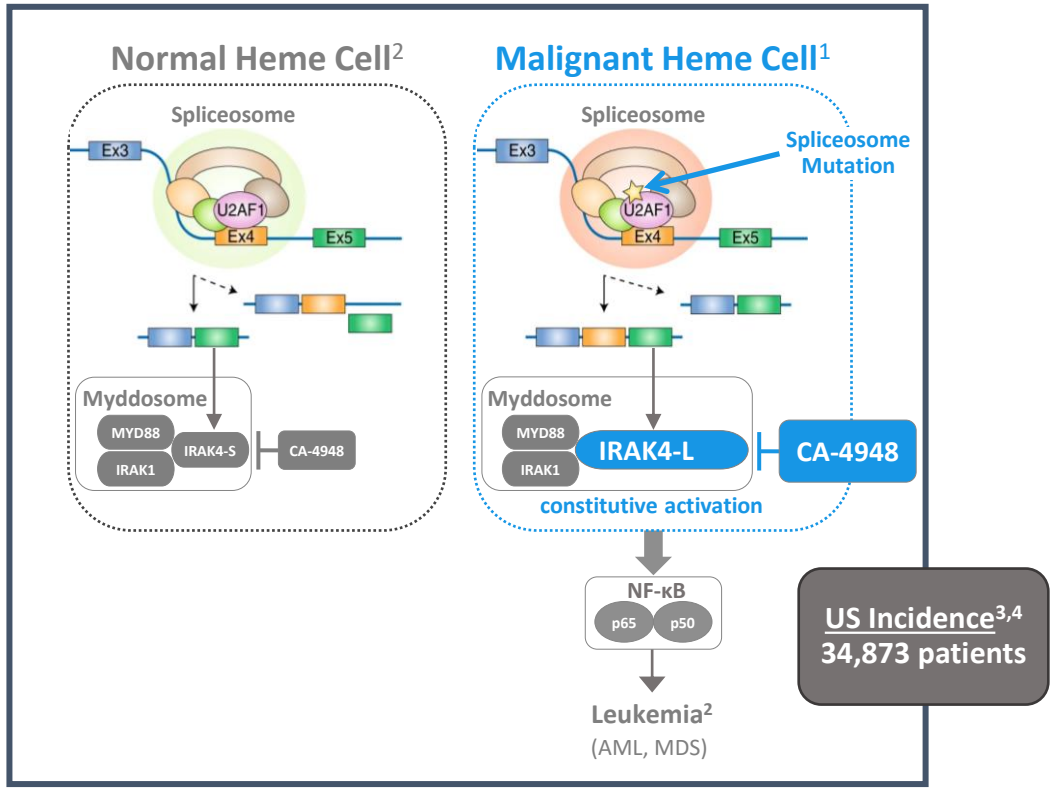
IRAK4 Biology and CA-4948

IRAK4 Biology and CA-4948

IRAK4 is a novel and important target across multiple heme malignancies

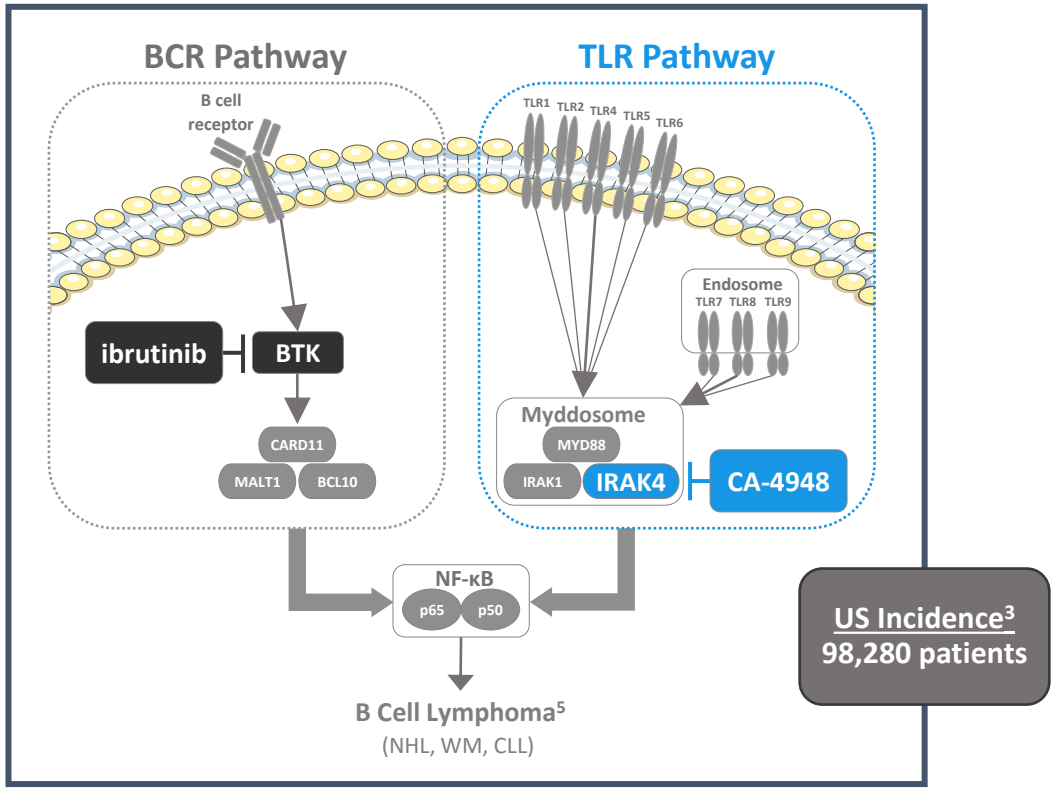
IRAK4 in AML/MDS

Spliceosome mutation drives overexpression of IRAK4-L (which causes constitutive activation of the myddosome)



IRAK4 in B Cell Cancers

TLR Pathway is dependent upon IRAK4 for function (the 2nd pathway driving NF-κB overactivity)



1) Guillamot et al. Nat Cell Biol 2019
2) Smith et al. Nat Cell Biol 2019
3) American Cancer Society, Cancer Facts & Figures 2020
4) Leukemia & Lymphoma Society, Facts and Statistics Overview
5) IMBRUVICA Package Insert. Rev 08/2018

CA-4948 Targeted Design

CA-4948 is the most advanced IRAK4 inhibitor in clinical development for cancer

In Nov 2020, the NCI selected CA-4948, Curis's first-in-class IRAK4 inhibitor, and entered into an agreement ("CRADA") with Curis to conduct both clinical and non-clinical studies of CA-4948 in oncology

CA-4948 inhibits IRAK4 and several additional key oncogenic targets

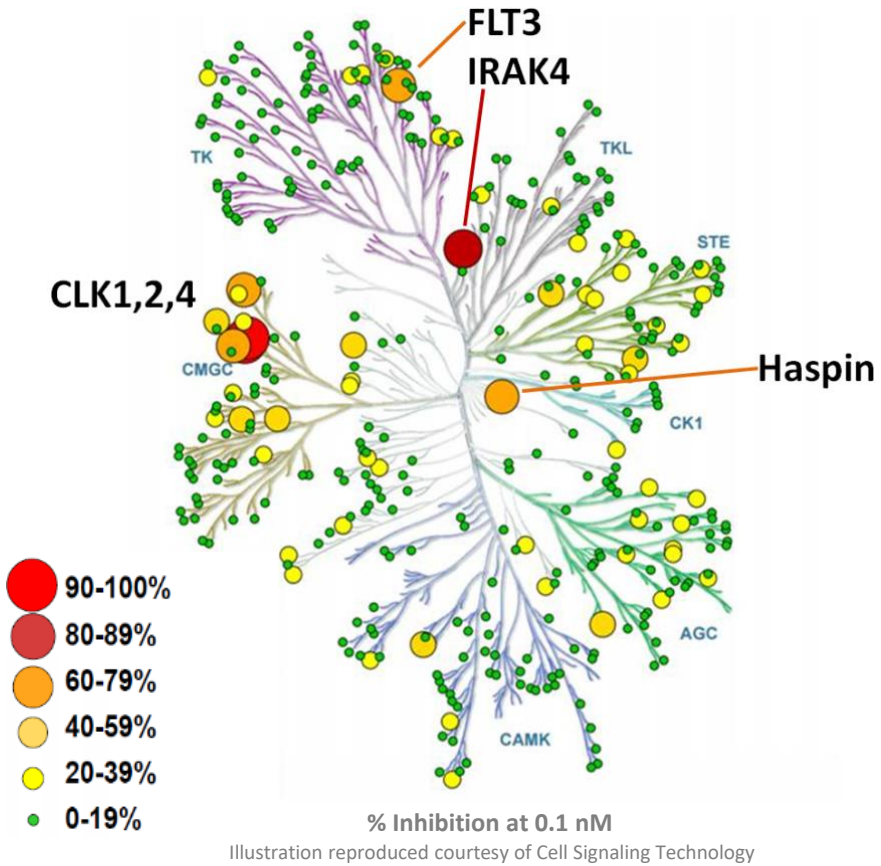


Illustration reproduced courtesy of Cell Signaling Technology

CA-4948 binding affinity

Target	K_d nM
IRAK1	12,000
IRAK2	>20,000
IRAK3	8,500
IRAK4	**23**
DYRK1A	25
FLT3 (D835H)	5
FLT3 (D835V)	44
FLT3 (ITD)	8
FLT3 (K663Q)	47
FLT3 (N841I)	16
Haspin (GSG2)	32
CLK1	10
CLK2	20
CLK3	>20,000
CLK4	14
RET (V804L)	3,000
TrkA	130

DiscoverX Kinase Panel
(378 kinases screened)

CA-4948 PK/PD

Attractive PK profile supports BID dosing and high target suppression

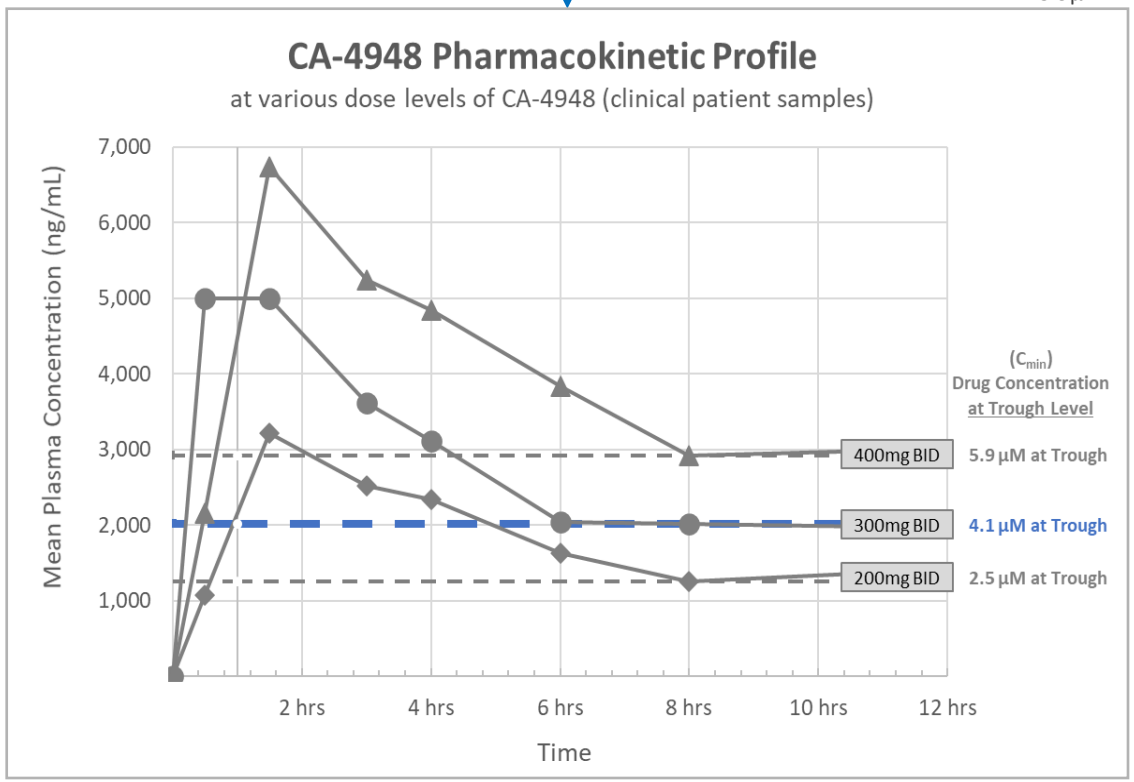
Attractive PK Profile

- Half-life of ~6 hours
- Supports BID dosing regimen

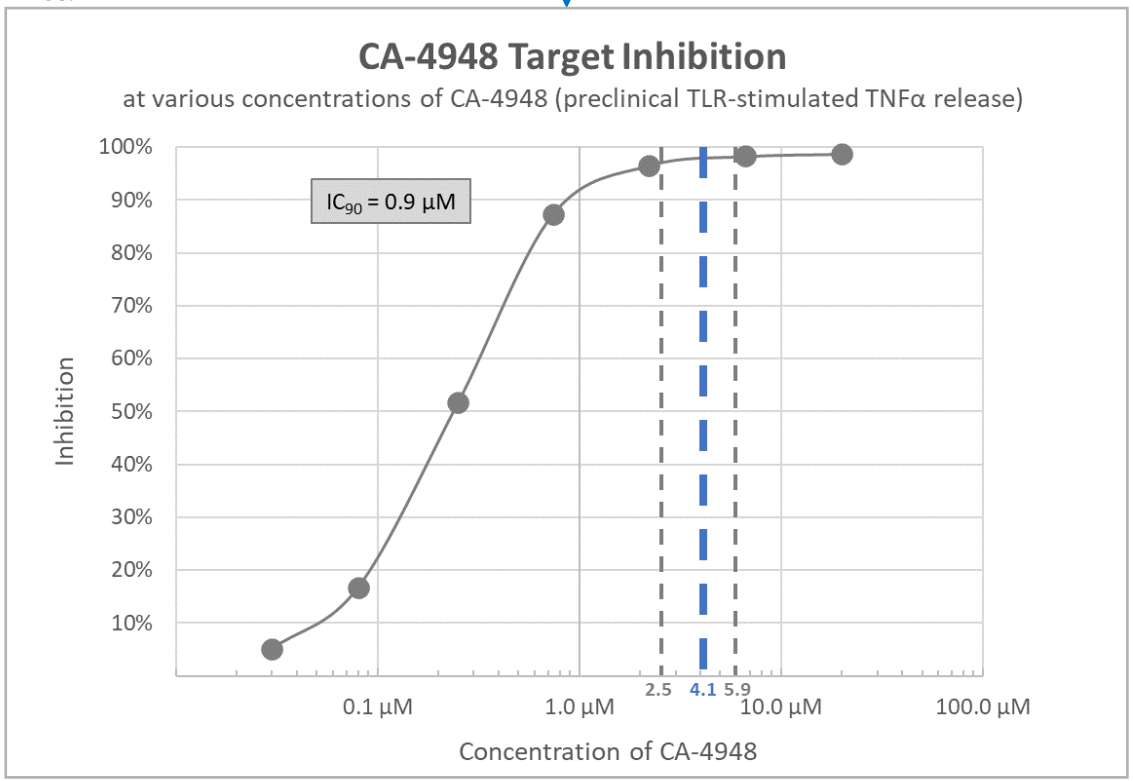
Trough Exposure	Dose	Inhibition
2.5µM	200mg	97%
4.1µM	**300mg**	**98%**
5.9µM	400mg	98%

High Target Suppression

- Exposure at RP2D correlates with 98% inhibition



Data from CA-4948 lymphoma clinical study

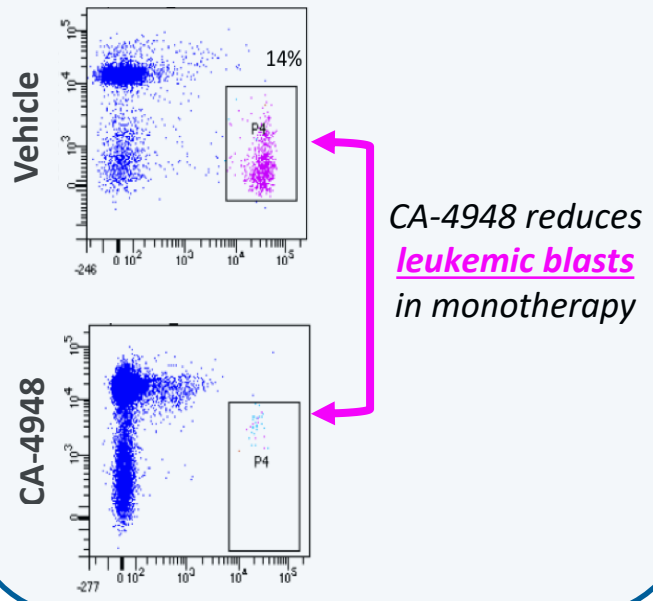


Data from preclinical study of target inhibition

CA-4948 Preclinical Data

Clear anti-cancer activity suggests broad potential across heme malignancies

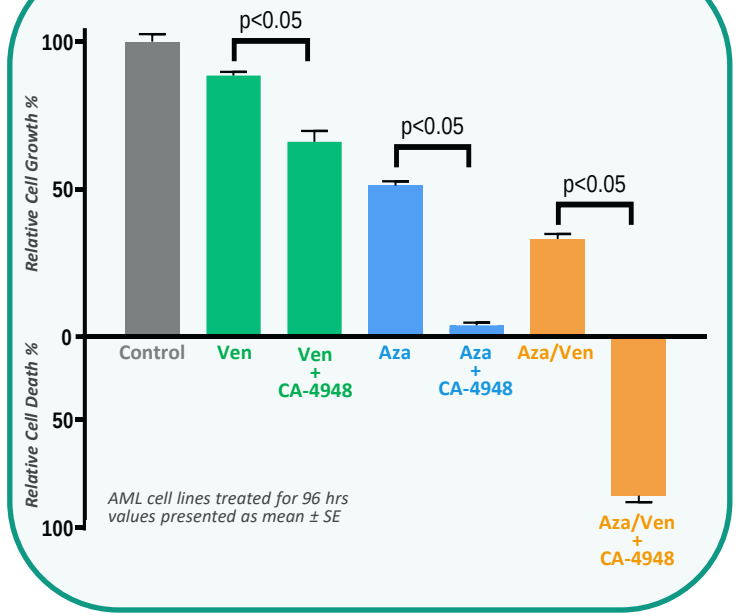
AML/MDS Monotherapy



CA-4948 reduces ***leukemic blasts*** *in monotherapy*

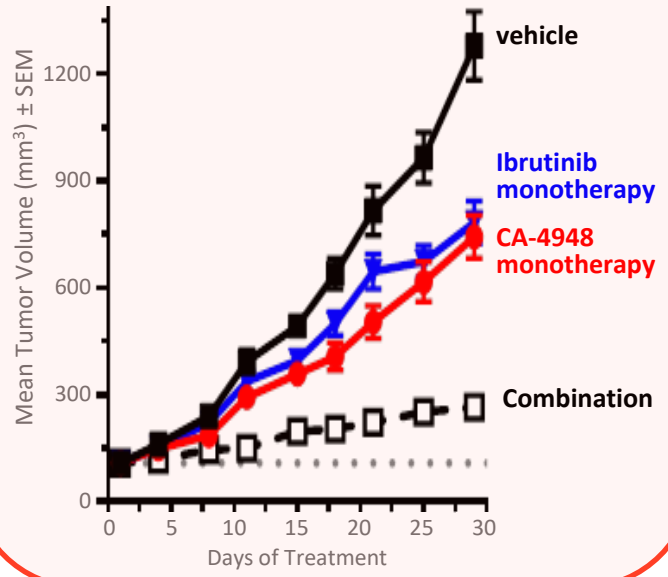
CA-4948 demonstrates monotherapy activity in patient-derived xenografts[1]

AML/MDS Combination



CA-4948 demonstrates synergy with both azacitidine and venetoclax in THP-1 model[2]

B Cell Cancers Monotherapy & Combination



CA-4948 demonstrates monotherapy and combination activity in OCI-Ly10 model[3]

1) Choudhary et al. AACR 2017
2) Curis AML MDS poster, EHA 2021
3) Booher et al. Waldenström Roadmap Symposium 2019

CA-4948 Clinical Plan

Planned clinical studies for AML/MDS and B cell cancers

AML/MDS Monotherapy

Specific Subpopulations

1) Patients with spliceosome mutation
2) Patients with FLT3 mutation

Supports rapid regulatory path

- *Spliceosome mutation is a leading cause of IRAK4-L overexpression[1]*
- *Signaling through IRAK4 is an adaptive response mechanism for FLT3 patients treated with a FLT3 inhibitor[2]*

AML/MDS Combination

Broader Population

1) R/R patients, HMA-naïve
2) R/R patients, venetoclax-naïve

R/R patients who do not have a spliceosome/FLT3 mutation and are ineligible for intensive chemotherapy

Supports use in broad population

- *Clinical data show CA-4948 reduces tumor burden in the significant majority of evaluable patients*
- *Preclinical data demonstrate synergy with azacitidine and venetoclax*

B Cell Cancers Monotherapy & Combination

Monotherapy

No monotherapy studies planned

Combination

1) BTKi naïve, Marginal Zone Lymphoma (MZL)
2) BTKi naïve, Primary CNS Lymphoma (PCNSL)
3) BTKi naïve, ABC-DLBCL
4) Patients w/ adaptive resistance to ibrutinib

Maximizes speed and probability of success

- *MZL, PCNSL, and ABC-DLBCL are aggressive indications which are associated with TLR Pathway activity*
- *If patients R/R to ibrutinib can be brought back into control, it would likely be because CA-4948 was added*

1) Smith et al. Nat Cell Biol 2019
2) Rabik et al. Ann Transl Med 2020

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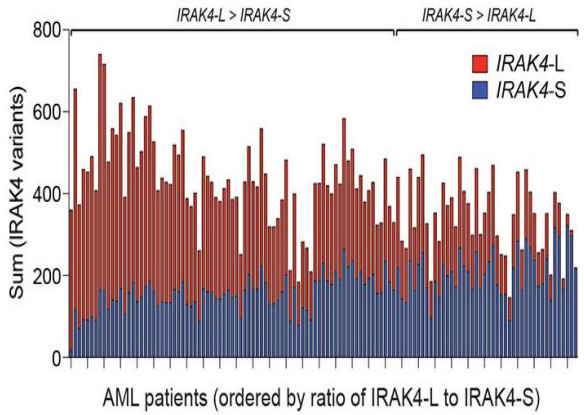
CA-4948 in AML/MDS

CA-4948 in AML/MDS

Clinical studies designed to leverage the role of IRAK4/FLT3 in AML/MDS

Disease Driver	% of Patient Population
IRAK4-L	> 50%[1]
FLT3	25-30%[2]
TET2	10-20%[3]
IDH2	9-13%[4]
IDH1	6-10%[4]
CEBPA	~10%[3]



Rationale for Monotherapy

- IRAK4 / FLT3 is the largest targeted market in AML/MDS[1,2]
- Spliceosome mutation is a leading cause of IRAK4-L overexpression[1]
- Signaling through IRAK4 is an adaptive response mechanism for FLT3 patients treated with a FLT3 inhibitor[5]

Rationale for Combination

- Nearly all patients express some level of IRAK4-L[1]
- Clinical data show CA-4948 reduces tumor burden in the significant majority of evaluable patients
- Preclinical data demonstrate clear synergy with azacitidine and venetoclax
    - *IRAK4 stimulates NF-κB and ultimately an array of anti-apoptotic factors (beyond BCL2), which prevent the effectiveness of anti-leukemic drugs*
    - *Blocking this effect with CA-4948 synergistically enhances the anti-cancer efficacy of those agents in preclinical models*

1) Smith et al. Nat Cell Biol 2019
2) Saygin, et al. J Hematol Oncol. 2017 Apr 18
3) DiNardo, Cortes. Hematology Am Soc Hematol Educ Program. 2016
4) DiNardo et al. N Engl J Med 2018
5) Rabik et al. Ann Transl Med 2020

CA-4948 in AML/MDS

Study design and patient characteristics

Phase 1/2 Study

- Open-label, single arm
- Dose escalation and expansion

Study Objectives

1°: Determine maximum tolerated dose
Determine recommended Phase 2 dose

2°: Pharmacokinetic (PK) profile
Preliminary anti-cancer activity

Study Population

- Relapsed/Refractory AML or High-Risk MDS
- ECOG performance Status of ≤ 2
- Age ≥ 18 years

Dosing

- Oral, Twice Daily (BID) Dosing
- 28-day cycles
- 3+3 escalation

(200mg → 300mg → 400mg → 500mg)
3 patients *6 patients* *10 patients* *3 patients*

>90% of patients enrolled had intermediate or worse cytogenetic risk

Baseline Patient Characteristics		Patients (n=22)
Female n (%) : Male n (%)		5 (23) : 17 (77)
Median Age		74 yrs
ECOG: n 0/1/2		7 / 11 / 4
Cytogenetic Risk[3] n (%)	AML (favorable, intermediate, adverse)	1 (10) , 2 (20) , 7 (70)
	hrMDS (good, intermediate, poor, very poor)	1 (9) , 4 (36) , 3 (27) , 3 (27)
Diagnosis n (%)	AML	11 (50)
	AML	11 (50)
Median platelets ($10^3/mm^3$) (range)		33 (7, 275)
Median ANC ($10^3/mm^3$) (range)		1.2 (0.1, 14.8)
Median lines of prior therapy (range)		2 (1-4)
Prior Therapy n (%)	Azacitidine	14 (64)
	Decitabine	7 (32)
	Cytarabine	3 (14)
	Venetoclax	10 (45)
Relevant Mutations	FLT3	1
	SF3B1	2
	U2AF1	2

Data cut-off: 30Apr2021

CA-4948 in AML/MDS

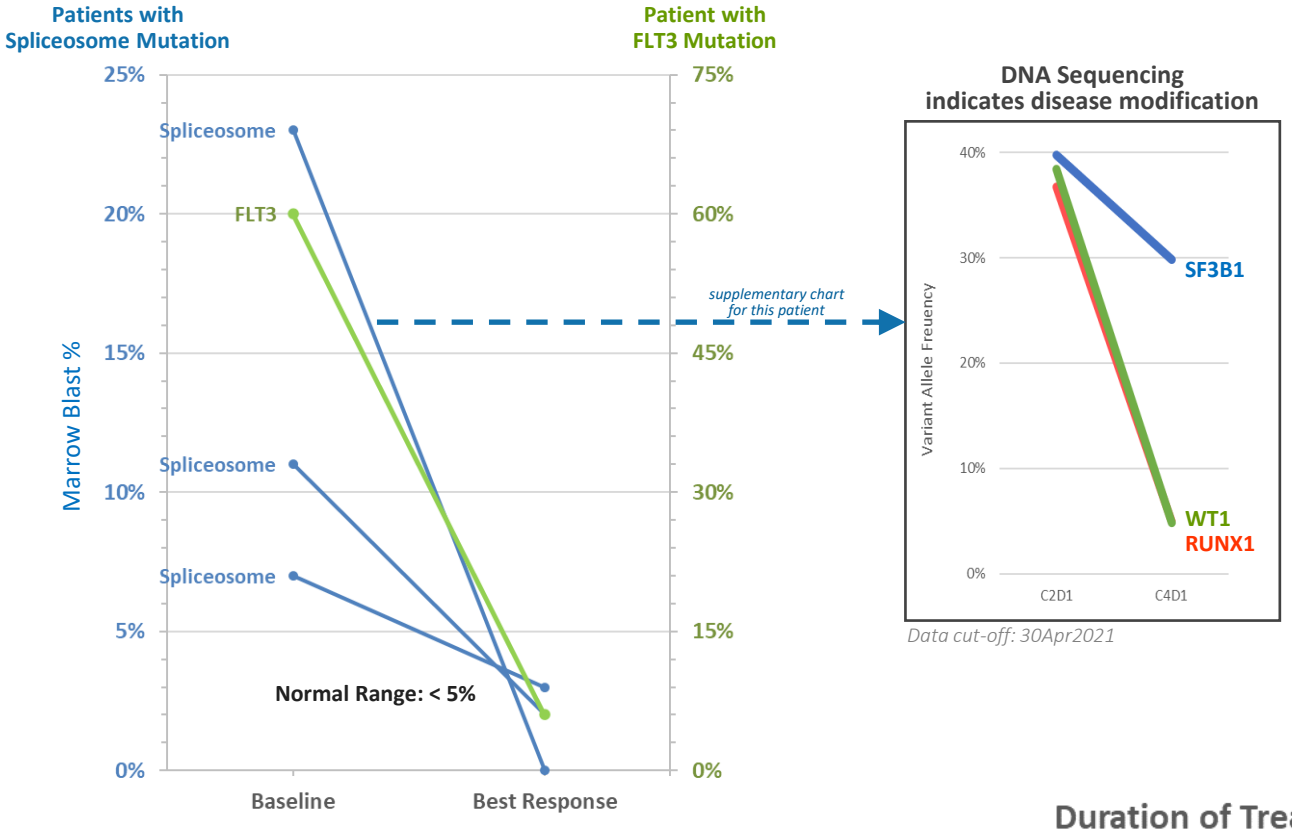
Preliminary safety data suggest differentiated profile

Predictable and manageable safety profile

- MTD not exceeded until 500mg BID
- No dose-limiting toxicities related to myelosuppression
- No overlap in dose-limiting toxicities with azacitidine and venetoclax, which are planned for combination with CA-4948
- Dose-limiting side effect at higher doses consists of uncomplicated rhabdomyolysis (elevated CPK and muscle soreness), was manageable, quickly and easily detected, readily reversible, and did not limit further treatment at a reduced dose level

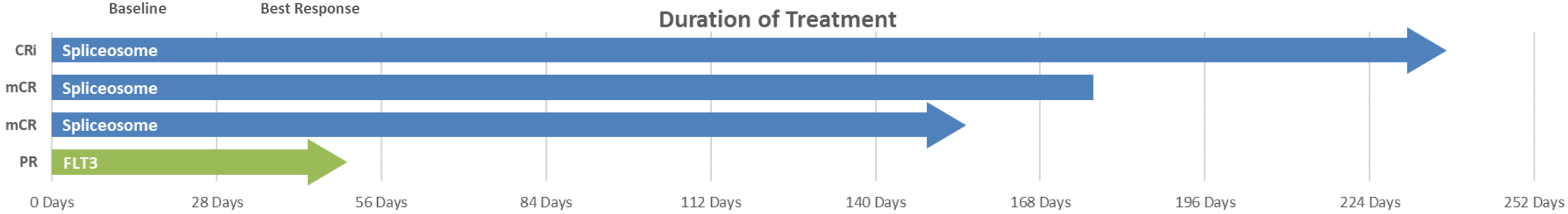
Preliminary Clinical Data: Specific Population in Monotherapy

Clear efficacy highlights potential for rapid regulatory path in spliceosome and FLT3



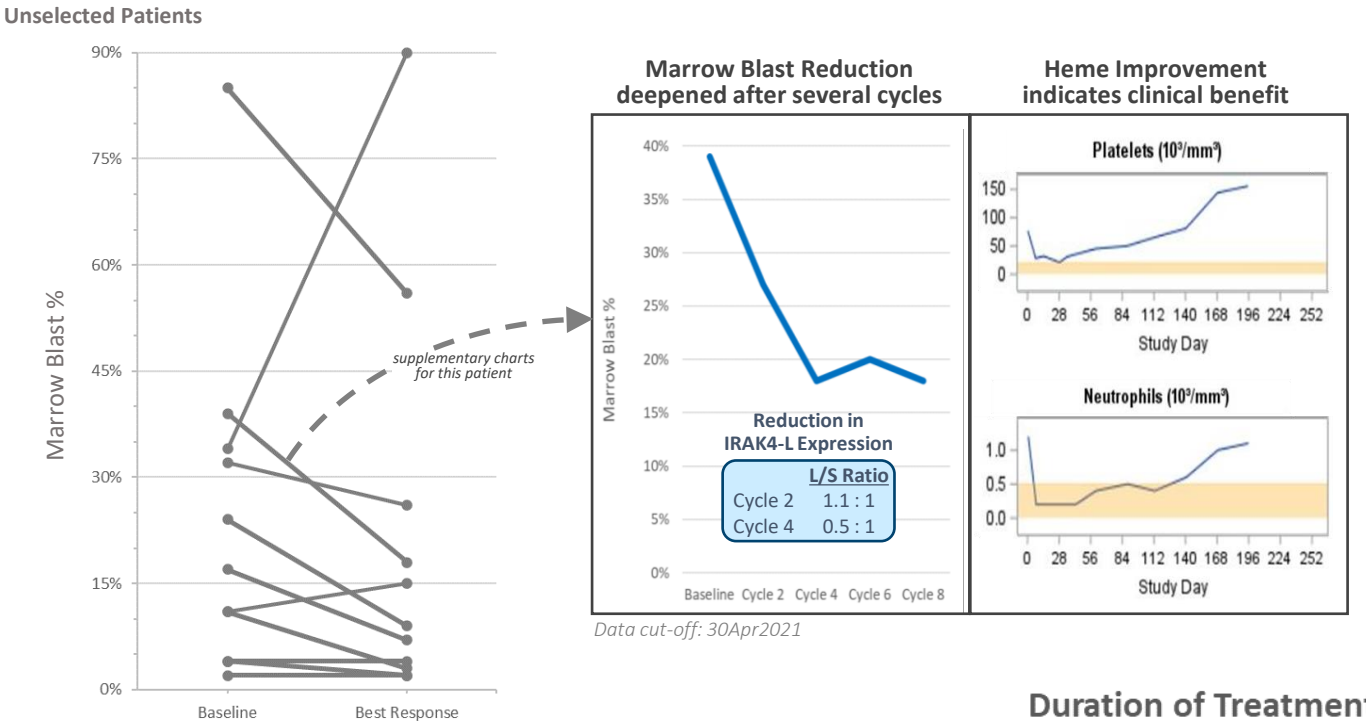
Data cut-off: 30Apr2021

- *4 of 4 evaluable patients w/ targeted mutation achieved objective response*
- *Ablation of cancer confirmed by genetic or morphologic analysis*



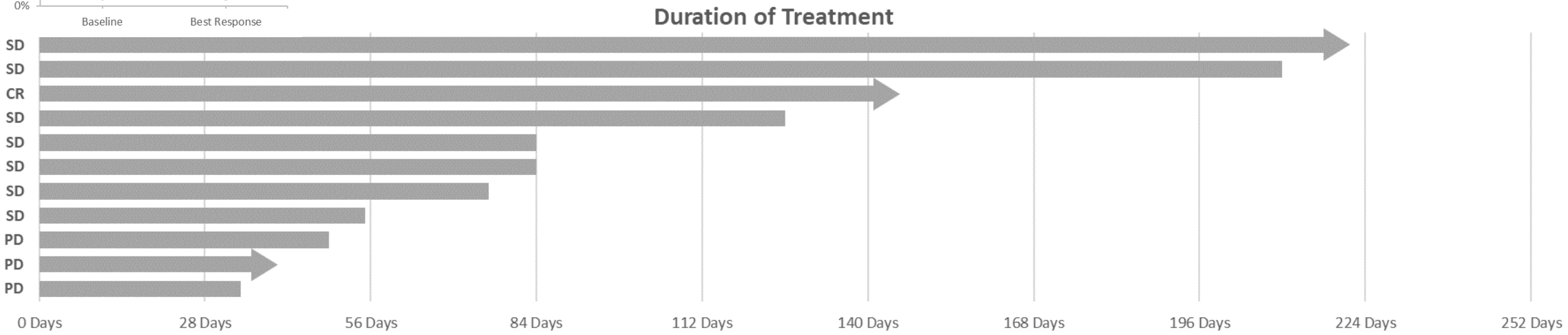
Preliminary Clinical Data: Broader Population in Combination

Efficacy supports differentiated profile even in patients without spliceosome/FLT3 mutation



Data cut-off: 30Apr2021

- *9 of 11 evaluable patients w/o targeted mutation achieved tumor reduction or maintained counts in normal range*



CA-4948 in AML/MDS

Multiple regulatory paths based on clear biologic activity and durable responses

- Preliminary clinical data show clear biologic activity with deepening, durable responses
  - In population targeted for monotherapy, 4 of 4 patients achieved objective response
  - Ablation of cancer confirmed by genetic or morphologic analysis
- Multiple paths to rapid regulatory approval in targeted subpopulations
- Clear anti-cancer activity in broader population suggests expanded opportunity in combination therapy

Next Steps in Expansion

- *Monotherapy: Spliceosome mutation*
- *Monotherapy: FLT3 mutation*
- *Combination: CA-4948 + azacitidine*
- *Combination: CA-4948 + venetoclax*

Plan to discuss potential for rapid approval path with FDA in 2022

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CA-4948 in B Cell Cancers

CA-4948 in B Cell Cancers

Monotherapy Phase 1/2 study design and patient characteristics

Study Objectives

1°: Determine maximum tolerated dose
Determine recommended Phase 2 dose

2°: Pharmacokinetic (PK) profile
Preliminary anti-cancer activity

Study Population

- Relapsed/Refractory B-cell NHL, including WM/LPL
- ECOG performance Status of ≤ 1
- Age ≥ 18 years

Dosing

- Oral, Once Daily (QD) and Twice Daily (BID) Dosing
- 21-day cycles
- 3+3 escalation
  (50mg → 100mg → 200mg → 300mg → 400mg BID)

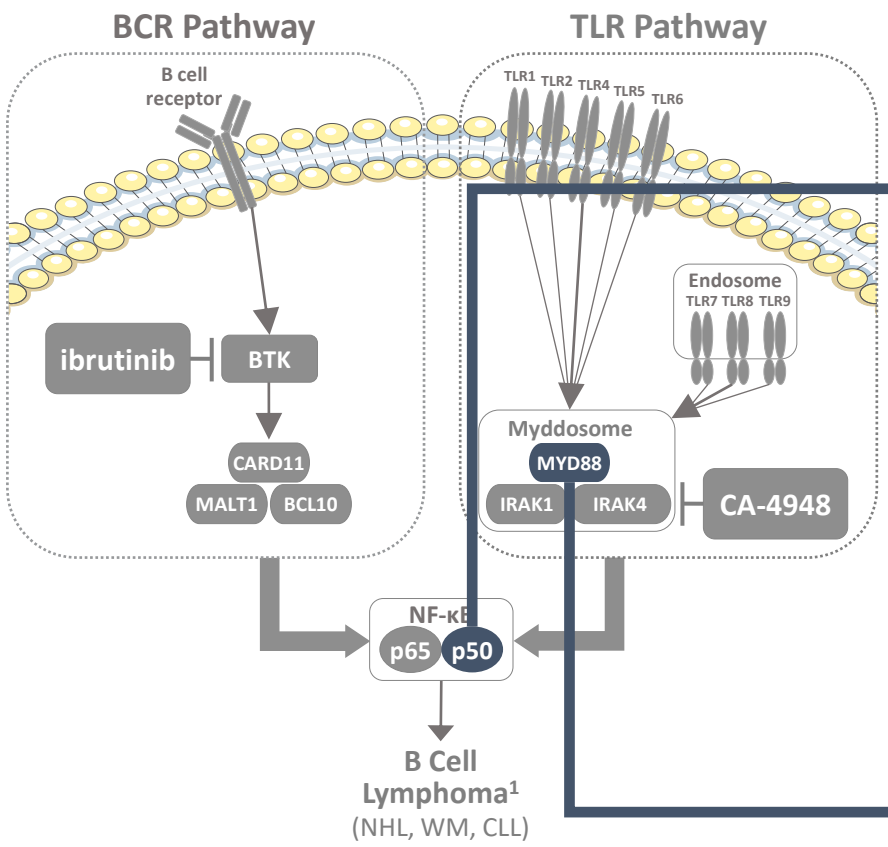
Heavily pre-treated population

Baseline Patient Characteristics		Patients (n=31)
Female n (%) : Male n (%)		26 (84) : 5 (16)
Median Age (range)		69 yrs
Diagnosis n (%)	DLBCL	14 (45)
	Transformed Follicular	6 (19)
	Waldenströms Macroglobulinemia	4 (13)
	Other	7 (23)
Median lines of prior therapy		4
Prior Therapy n (%)	BTK inhibitor	6 (19)
	CAR-T	5 (16)
	ASCT	7 (23)
	Other	13 (42)
MYD88 Status	Positive	2 (6)
	Negative	18 (58)
	Unknown	11 (35)

Source: ASH 2020 Presentation

CA-4948 in B Cell Cancers

Two potential biomarkers may increase probability of success



NF-κB phospho-p50

positive expression of p-p50 indicates the NF-κB complex is active

Baseline testing may support future patient selection

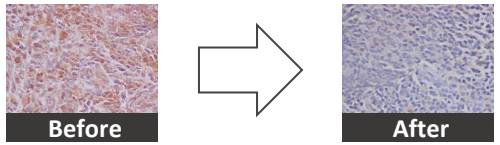
6 of 7 patients testing p-p50 positive at baseline experienced SD or tumor shrinkage

Patient	Best Response	Patient	Best Response
19-1001	-35% SD	12-1002	+86% PD
02-1001	-24% SD	18-2004	+156% PD
02-3003	+22% SD	02-4004	+75% PD
12-5007	-35% SD	12-4004	+125% PD
02-6007	+25% SD	12-5006	+190% PD
02-6008	+16% SD	13-6001	+98% PD
15-1001	+65% PD	01-4002	+7% PD

7 of 7 patients testing p-p50 negative at baseline experienced disease progression

Biomarker data support thesis that CA-4948 "turns off" NF-κB

p-p50 positive at baseline *p-p50 negative after treatment*



Source: ASH 2020 Presentation

MYD88

MYD88 mutation indicates the TLR/myddosome is driving NF-κB activity

Baseline testing may support future patient selection

Patient	Best Response
12-5005	-20% SD
17-3002	-67% PR

both patients testing positive for MYD88 mutation at baseline experienced PR or tumor shrinkage

Observed tumor reduction is consistent with our thesis that patients with MYD88-mutated tumors should benefit from IRAK4 inhibition

Source: ASH 2020 Presentation

1) IMBRUVICA Package Insert. Rev 08/2018

CA-4948 in B Cell Cancers

Promising preliminary safety data

Predictable and manageable safety profile

- MTD not exceeded until 400mg BID
- No overlap in dose-limiting toxicity with ibrutinib, which is planned for combination with CA-4948
- Dose-limiting side effect at higher doses consists of uncomplicated rhabdomyolysis (elevated CPK and muscle soreness), was manageable, quickly and easily detected, readily reversible, and did not limit further treatment at a reduced dose level

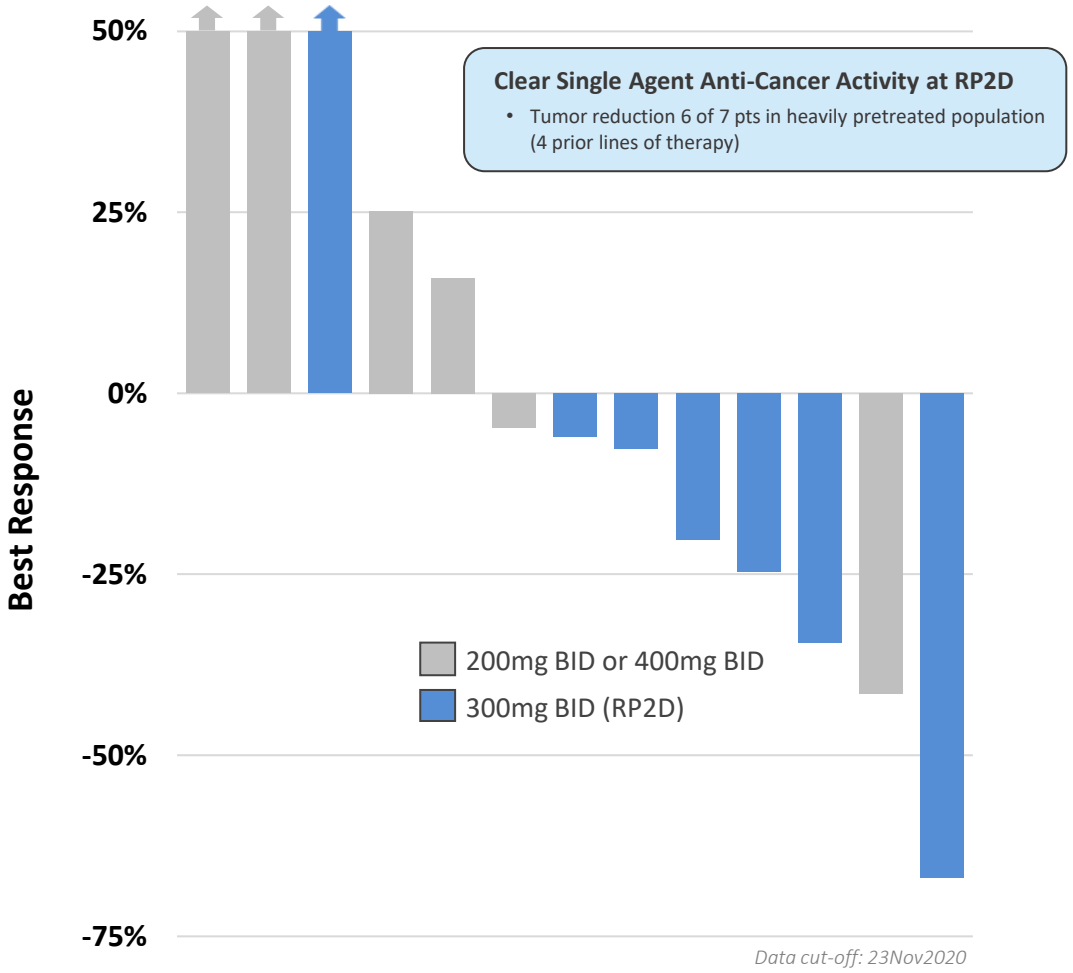
	Adverse Reaction	200 mg BID (n=5); (%)		300 mg BID (n=6); (%)		400 mg BID (n=8); (%)		All (n=30); (%)
		All Grades	Grade 3 or 4	All Grades	Grade 3 or 4	All Grades	Grade 3 or 4	All Grades
Gastrointestinal disorders	Diarrhea	20	0	33	0	25	0	20
	Nausea	20	0	17	0	38	0	27
	Vomiting	20	0	17	17	25	0	20
	Constipation	20	0	0	0	13	0	20
Respiratory	Upper respiratory infection	40	20	0	0	13	0	7
	Dyspnoe	20	0	0	0	13	13	7
	Upper-airway cough	40	0	0	0	0	0	7
General & Other	Fatigue	40	0	0	0	50	0	37
	Oedema	20	0	0	0	0	0	10
	Dehydration	20	0	0	0	13	13	10
Nervous system disorders	Headache	20	0	0	0	13	0	10
	Dizziness	0	0	0	0	25	0	20
	Insomnia	20	0	0	0	13	0	7
	Peripheral sensory neuropathy	0	0	0	0	25	0	7
Musculoskeletal disorders	Back pain	20	0	0	0	13	0	10
	Myalgia	40	0	0	0	38	0	17
	Rhabdomyolysis	0	0	0	0	25	25	7
	Muscle weakness	20	20	0	0	13	0	7
Hematological	Neutropenia	40	40	17	17	25	0	7
	Anemia	20	0	33	0	13	13	20
	Thrombocytopenia	0	0	0	0	13	13	7

Data cut-off: 11Oct2020

CA-4948 in B Cell Cancers

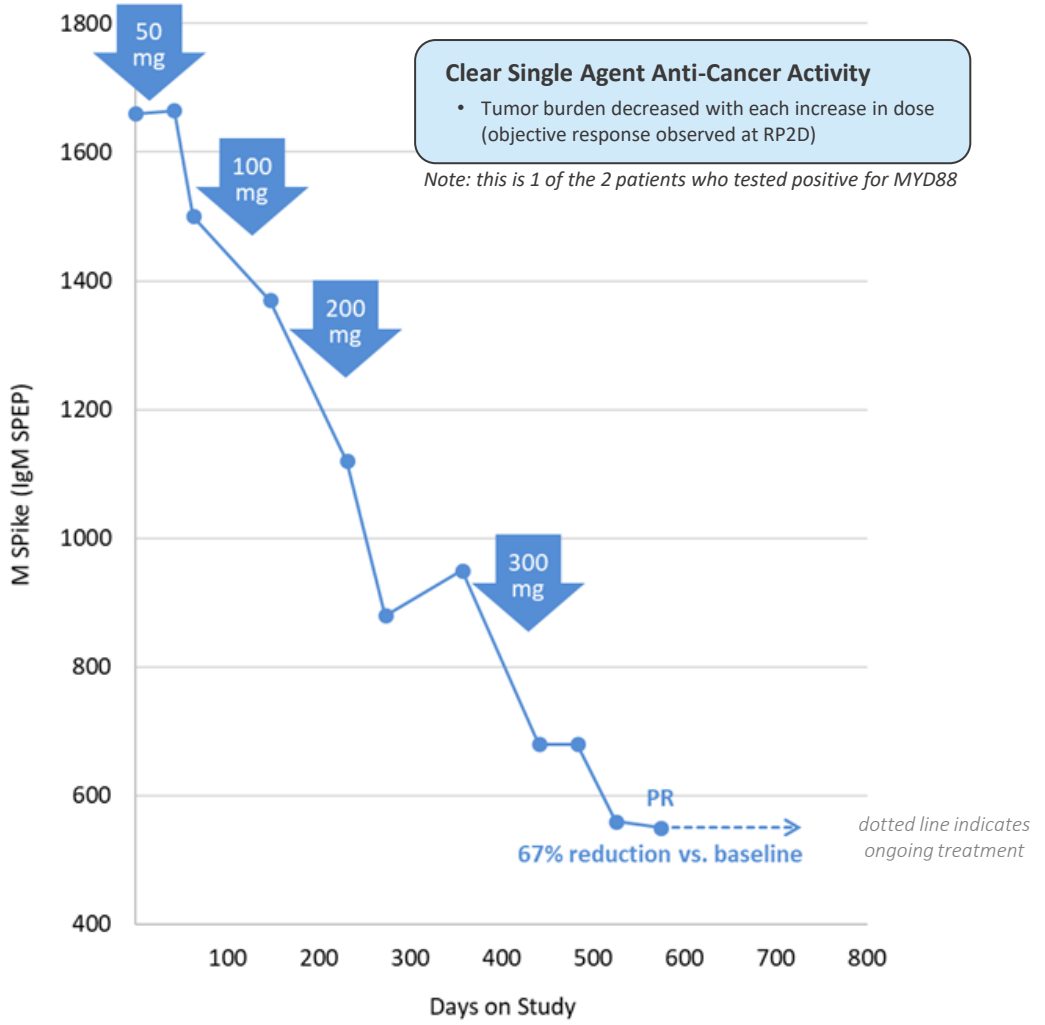
Preliminary clinical data: clear reduction in tumor burden

Change in Tumor Burden for 13 Evaluable Patients
(200mg BID+)



Data cut-off: 23Nov2020

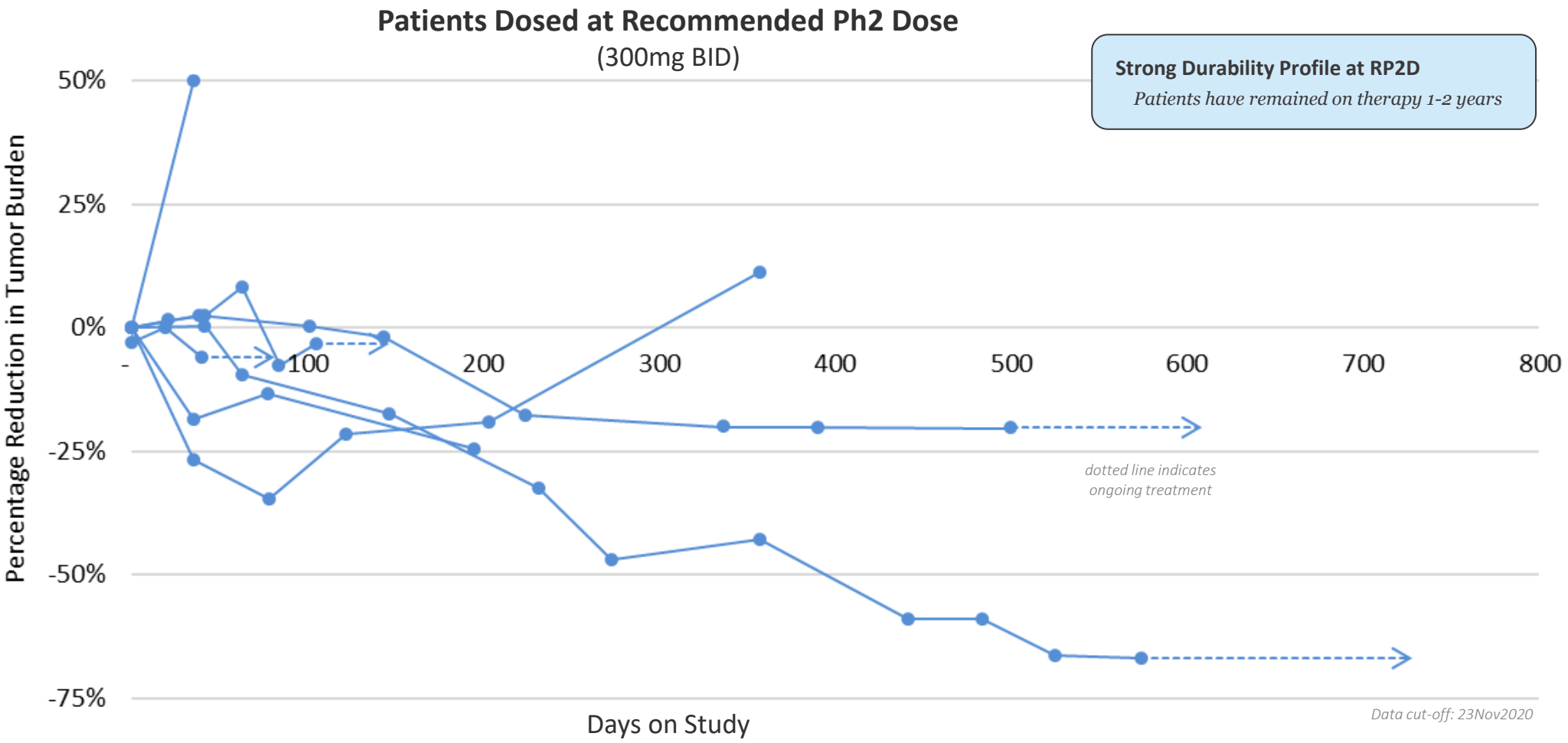
Dose Response Observed in WM Patient
(50mg - 300mg BID)



Note: this is 1 of the 2 patients who tested positive for MYD88

CA-4948 in B Cell Cancers

Preliminary clinical data: strong durability profile



CA-4948 in B Cell Cancers

CA-4948 is the ideal candidate to combine with BTKi to maximize downregulation of NF-κB

- Patients are treated with BTKi because downregulating NF-κB activity provides benefit in B Cell Cancers
- Two pathways drive NF-κB:
  - BCR Pathway: addressed with BTKi
  - TLR Pathway: addressed with IRAK4i
- Preliminary clinical data demonstrate clear reduction in tumor burden, even in heavily pretreated patients

Next Steps in Expansion

- *BTKi naïve, Marginal Zone Lymphoma (MZL)*
- *BTKi naïve, Primary CNS Lymphoma (PCNSL)*
- *BTKi naïve, ABC-DLBCL*
- *Patients with adaptive resistance to ibrutinib*

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VISTA Biology and CI-8993

VISTA Biology

VISTA is an important checkpoint regulator target across multiple malignancies

RESEARCH ARTICLE SUMMARY

T CELLS

VISTA is a checkpoint regulator for naïve T cell quiescence and peripheral tolerance

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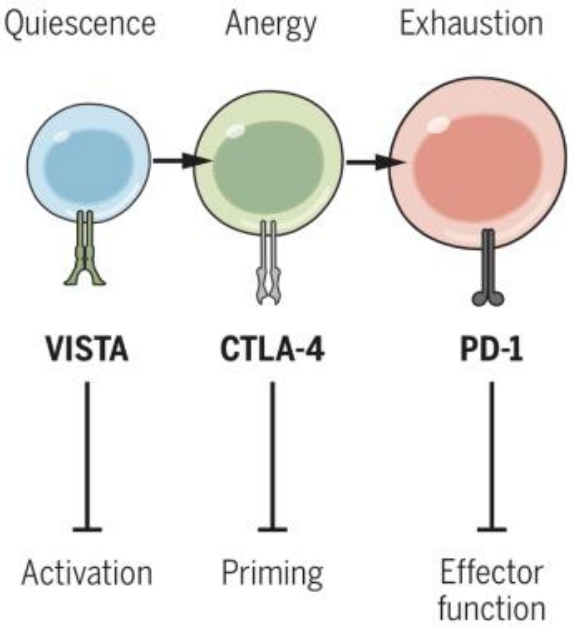
- CTLA-4, PD-1, and VISTA are the three main players in controlling checkpoint blockade
- VISTA controls early T cell activation events
- Blockade of VISTA will allow for an expanded T cell response against tumors

ElTanbouly et al. Science. 2020

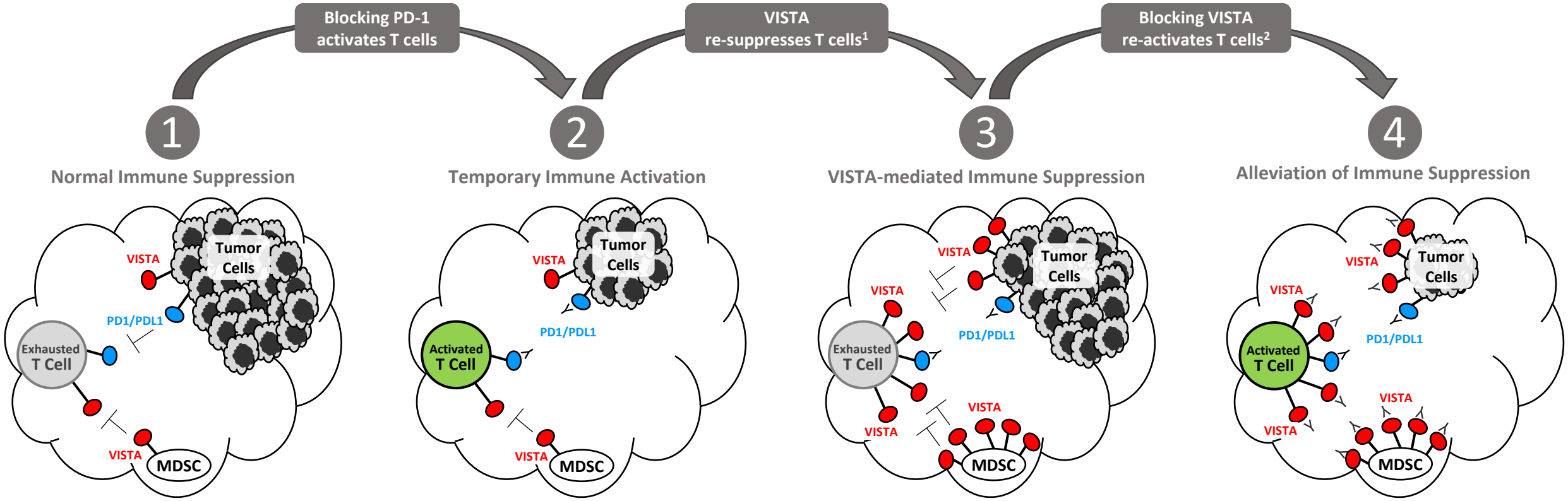
Integration of VISTA with other well-established negative checkpoint regulators of T cell activation



ElTanbouly et al. Science. 2020

VISTA Biology

Role of VISTA in immune suppression in the tumor microenvironment (TME)



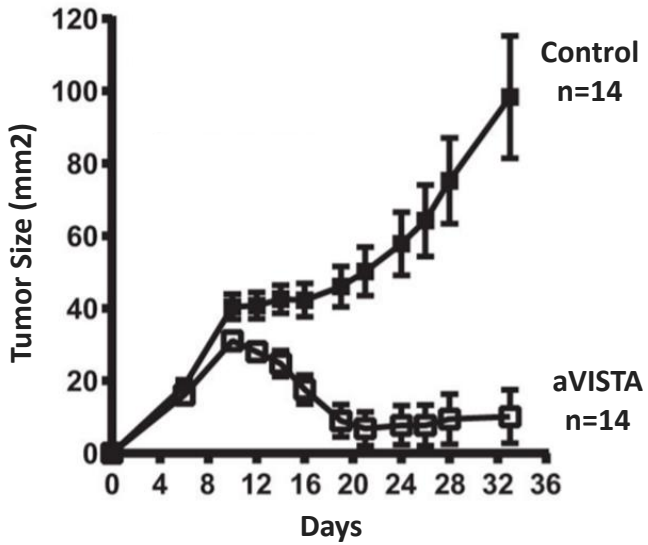
[1] Gao et al. Nature. 2017. 23: 551–555
[2] Data from ImmuNext preclinical studies

CI-8993 Preclinical Data

Clear anti-cancer activity suggest potential transformation of immune-oncology treatment

Monotherapy

Anti-VISTA inhibited tumor growth in B16ova melanoma model[1]



[1] Le Mercier et al. Cancer Res. 2014 Apr 1

Combination Therapy

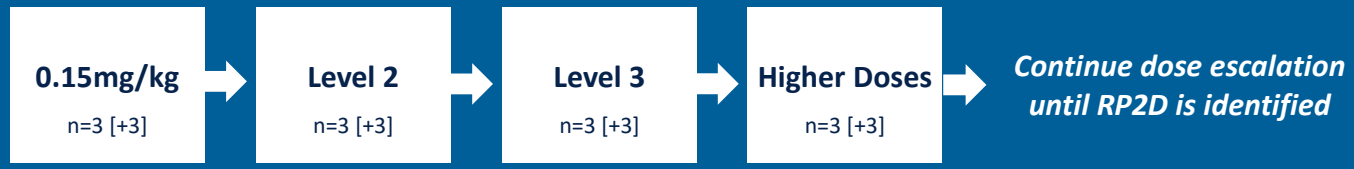
Anti-VISTA inhibited xenograft growth in checkpoint resistant CT26 model[2]

[2] J. Lines, IEBMC Conference 2019

CI-8993 Clinical Plan

Ongoing clinical study to determine safety

Curis Design for Ph1 Dose Escalation Study



Patient Population

- Patients with advanced refractory solid tumors (includes mesothelioma, melanoma, NSCLC, TNBC)

Treatment

- Bi-weekly dosing
- Mitigate potential toxicities by desensitization, premedication, dosing interval and duration

Objective

- Safety, PK/PD, tolerability during dose escalation
- Anti-cancer activity during expansion

CI-8993 is the first anti-VISTA monoclonal antibody to enter the clinic

- Janssen licensed VISTA IP from ImmuNext in 2012 and initiated a Ph1 study in 2016 (JNJ-61610588)
- 12 patients were enrolled; initial dose level was 0.005mg/kg
- Low-grade transient Cytokine Release Syndrome (CRS) seen at 0.15mg/kg and above

Janssen halted study after 1 DLT at sub-therapeutic dose level

- The only patient treated at 0.3mg/kg experienced grade 3 CRS-associated encephalopathy after 36hrs on treatment
- Patient was initially treated w/antibiotics; symptoms resolved after treatment with tocilizumab
- Janssen opted to halt the study and return IP to ImmuNext

Target range for expected anti-cancer activity (0.5 – 2.0mg/kg) was never reached

Curis Study Design Incorporates Key Learnings from Janssen Ph1 Study

- CRS is likely an on-target toxicity; indicates drug is hitting the target and inducing inflammatory response
- Oncology community is now familiar with managing CRS; NCCN guidelines were issued in 2018
- FDA cleared the study IND which outlined our plan for managing CRS and enabling escalation to therapeutic dose levels

CI-8993 in Solid Tumors

CI-8993 has potential to be the leading anti-VISTA therapeutic

- VISTA's role in enforcing T cell quiescence limits the effectiveness of other immune checkpoint therapies
- Preclinical data demonstrate potential to transform treatment with CTLA4/PD1 checkpoint inhibitors
- CI-8993 is the leading anti-VISTA monoclonal antibody in clinical studies

Next Steps in Dose Escalation

- *Confirm that CI-8993 can be administered safely (that CRS can be managed) in dose escalation*

Corporate Overview

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 *Cash and investments of approximately $161M as of June 30, 2021; cash runway into 2024*
Robust Pipeline	CA-4948: first-in-class inhibitor of IRAK4 in oncology *There are no drugs currently approved for IRAK4 inhibition in oncology* CI-8993: first-in-class antagonist of VISTA *There are no drugs currently approved for VISTA inhibition*
Upcoming Milestones	YE 2021: Report safety data in CI-8993 (VISTA) YE 2021: Report additional data in CA-4948 in AML/MDS (spliceosome population) 2022: Discuss potential for rapid approval path for CA-4948 with FDA

Committed, Experienced 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 Roemeling
Head, Clinical Development

Raul Soikes
Head, Portfolio Management

Nancy Soohoo
General Counsel

William Steinkrauss
Chief Financial Officer

End of Corporate Presentation

NASDAQ: CRIS