

Corvus Corporate Presentation

Cowen Healthcare Conference

March 12, 2019

Forward-Looking Statements / Safe Harbor



This presentation and the accompanying oral presentation contain “forward-looking” statements. All statements other than statements of historical facts contained in this presentation, including statements related to the potential safety and efficacy of Ciforadenant (CPI-444), both as a single agent and in combination with anti-PD-1 or anti-PD-(L)1, CPI-006 and CPI-818; the Company’s or Genentech’s ability to develop and advance product candidates into and successfully complete clinical trials, including the Company’s Phase 1/1b clinical trial of Ciforadenant, and Genentech’s Phase 1b/2 clinical trial of Ciforadenant in combination with atezolizumab, and the timing of any future clinical trials including the Company’s Phase 1b/2 clinical trial of Ciforadenant and Phase 1/1b clinical trials of CPI-006 and CPI-818; and the potential utility of preclinical findings. Forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified. In some cases, you can identify forward-looking statements by terminology such as “believe,” “will,” “may,” “estimate,” “continue,” “anticipate,” “intend,” “should,” “plan,” “might,” “approximately,” “expect,” “predict,” “could,” “potentially” or the negative of these terms or other similar expressions. You should not put undue reliance on any forward-looking statements. Forward-looking statements should not be read as a guarantee of future performance or results, and will not necessarily be accurate indications of the times at, or by, which such performance or results will be achieved, if at all. Forward-looking statements are based on information available at the time those statements are made and/or management’s good faith beliefs and assumptions as of that time with respect to future events, and are subject to known and unknown risks and uncertainties that could cause actual performance or results to differ materially from those expressed in or suggested by the forward-looking statements. In light of these risks and uncertainties, the forward-looking events and circumstances discussed in this presentation may not occur and actual results could differ materially from those anticipated or implied in the forward-looking statements. Certain of these risks and uncertainties are described in greater detail in our Annual Report on Form 10-K for the year ended December 31, 2018, filed with the Securities and Exchange Commission on March 7, 2019 as well as other documents that may be filed by the Company from time to time with the Securities and Exchange Commission. Except as required by law, we do not undertake any obligation to publicly update or revise any forward-looking statement, whether as a result of new information, future developments or otherwise.

This presentation concerns products that are under clinical investigation and which have not yet been approved for marketing by the U.S. Food and Drug Administration. Such products are currently limited by Federal law to investigational use, and no representation is made as to its safety or effectiveness for the purposes for which it is being investigated.

An Expanding Pipeline

Adenosine Pathway	DEVELOPMENT STATUS				Status
	Lead Optimization	IND-Enabling	Phase 1/1b	Phase 1b/2	
Adenosine A2A Receptor Antagonist	Combination (Ciforadenant +Tecentriq) and Single-agent (Ciforadenant) RCC				Completed enrollment of Phase 1/1b
	Combination (Ciforadenant+Tecentriq) RCC				Enrolling Phase 1b/2
	Morpheus (Ciforadenant+Tecentriq) NSCLC				Enrolling Phase 1b/2
Adenosine Production Inhibitor Anti-CD73	CPI-006 Monotherapy and Combinations				Enrolling Phase 1/1b
Adenosine A2B Receptor Antagonist	CPI-935				Development candidate selected

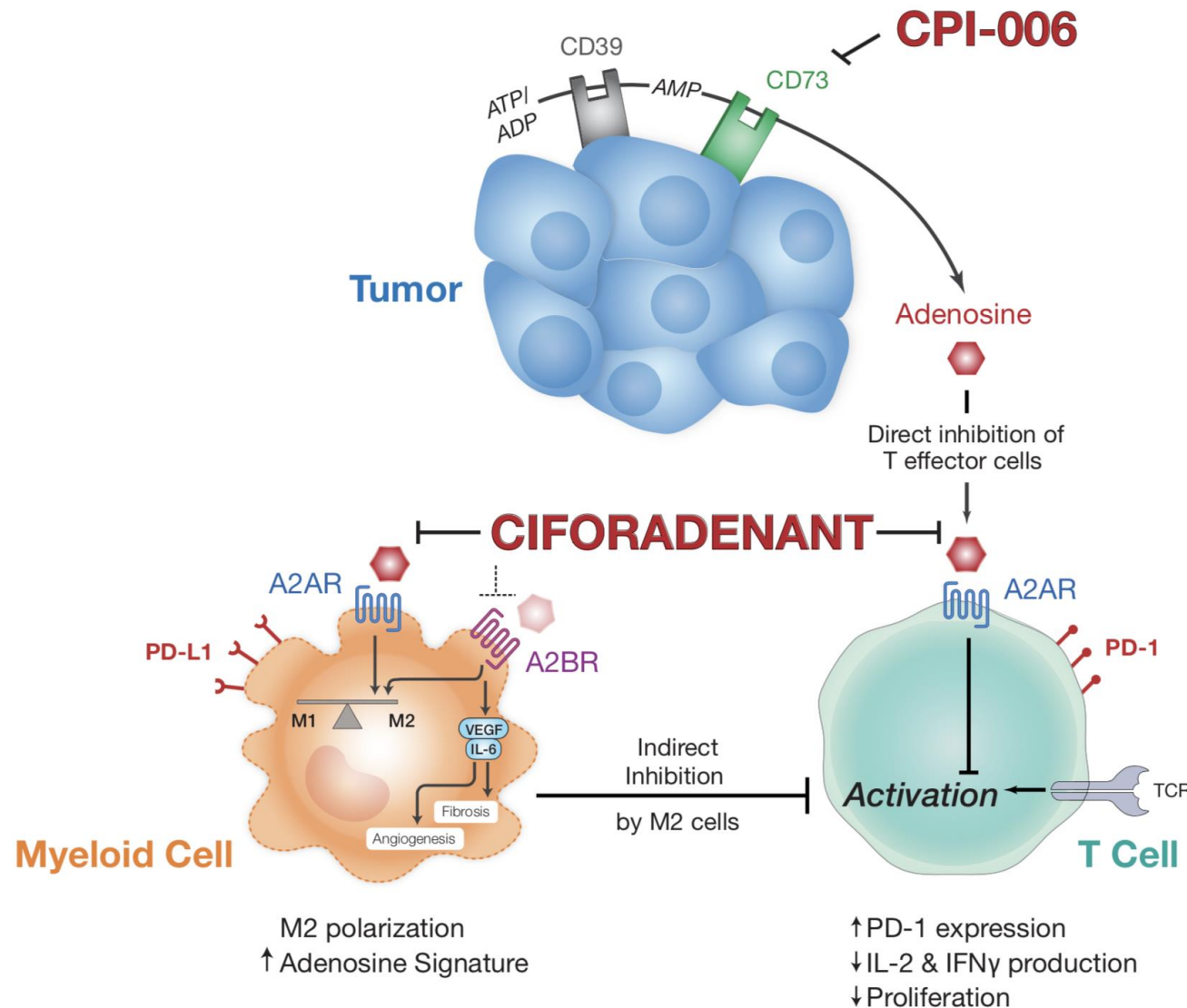
T cell Differentiation

ITK Inhibitor	CPI-818				OPEN Phase 1/1b
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Myeloid Suppression

Undisclosed target					Development candidate selected
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Adenosine in the Tumor Microenvironment



- Tumors make adenosine, which is immunosuppressive
- Adenosine is a potential resistance mechanism to anti-PD-(L)1 therapy. (*Beavis et al, Can Immunol Res 2015*)
- **Ciforadenant (CPI-444)** blocks adenosine A2A receptors on immune cells, restoring their activity
- **CPI-006** targets CD73, blocking adenosine production



SITC 2018

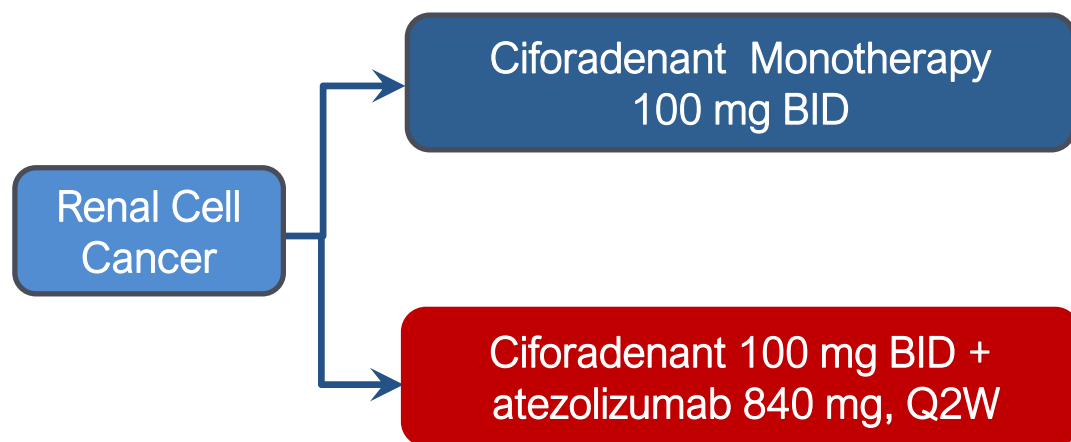
NOVEMBER 7-11
WASHINGTON, D.C.

Walter E. Washington
Convention Center



Society for Immunotherapy of Cancer

TRIAL DESIGN & PATIENT CHARACTERISTICS



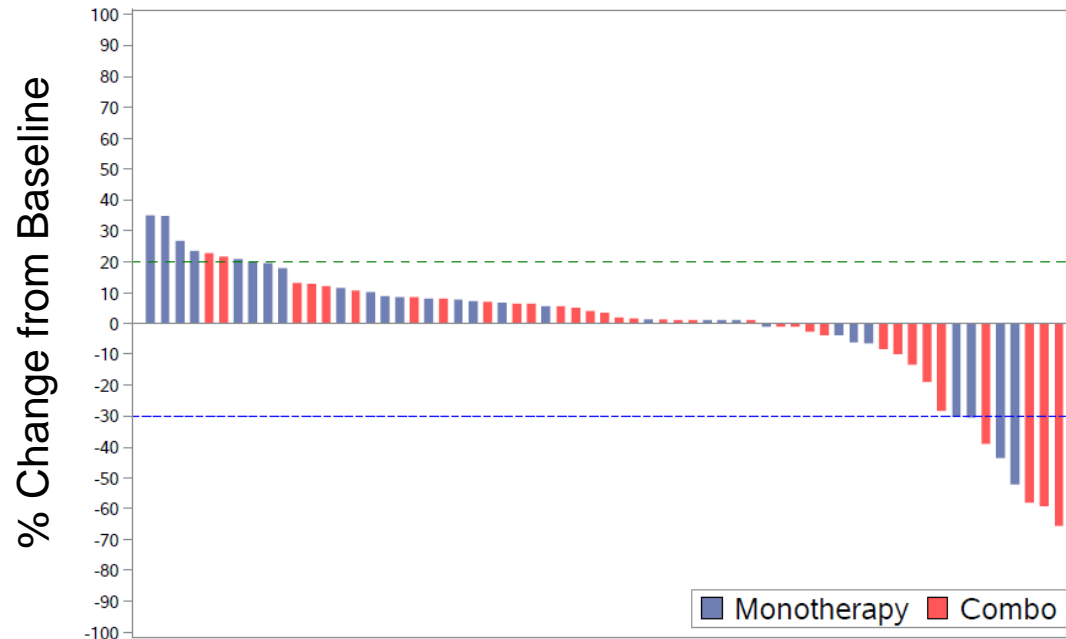
Eligibility

- Prior anti PD-(L)1 allowed
- Progressive disease at time of entry
- No selection for PD-L1 expression

Baseline Demographics	Ciforadenant (n=33)	Ciforadenant + atezolizumab (n=35)
Age (years), median (range)	60 (47, 76)	65 (44, 77)
Gender, male n (%)	25 (75.8)	28 (80.0)
Number of prior therapies median (range)	3 (1, 5)	3 (1, 5)
Prior IO, number of subjects n (%)	24 (72.7)	25 (71.4)
Months since prior IO therapy, median (range)	3.1 (1.2, 70.4)	1.7 (0.9, 23.6)
Prior Anti-Cancer Therapy n (%)		
TKI	27 (81.8)	30 (85.7)
mTor	9 (27.3)	11 (31.4)
anti-PD-1	23 (69.7)	25 (71.4)
anti-VEGF, Bevacizumab	6 (18.2)	4 (11.4)
IL-2	7 (21.2)	9 (25.7)

TUMOR RESPONSE TO TREATMENT

Best Response of All Patients

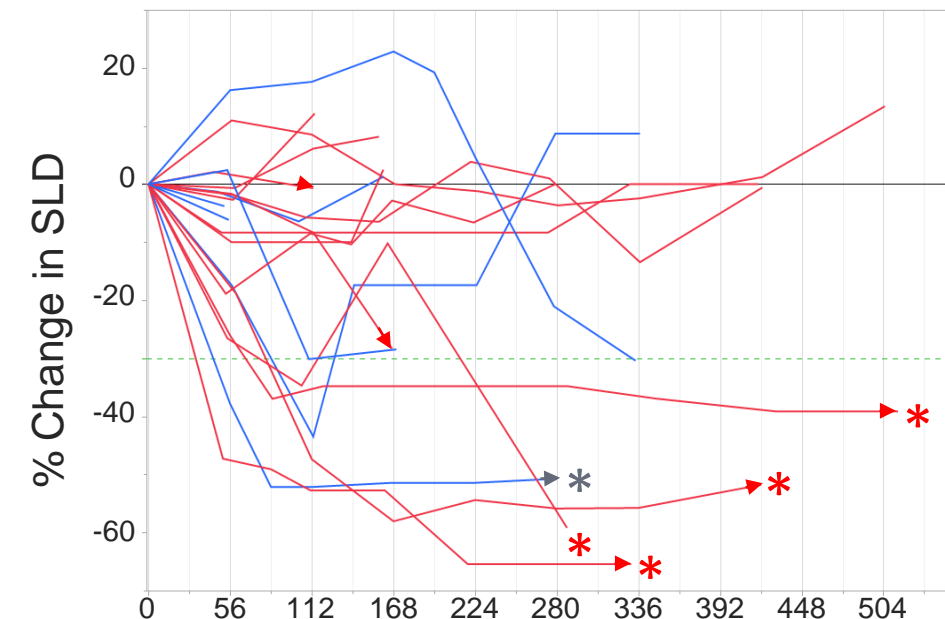


6 Month Disease Control Rate

	Mono*	Combo*
Prior PD(L)-1	25% (5/20)	32% (7/22)
Naive	0% (0/9)	44% (4/9)
Total	17% (5/29)	35% (11/31)

* Disease control % (# Disease control patients/total)

Spider Plot of Patients with Tumor Regression



* = PR by RECIST

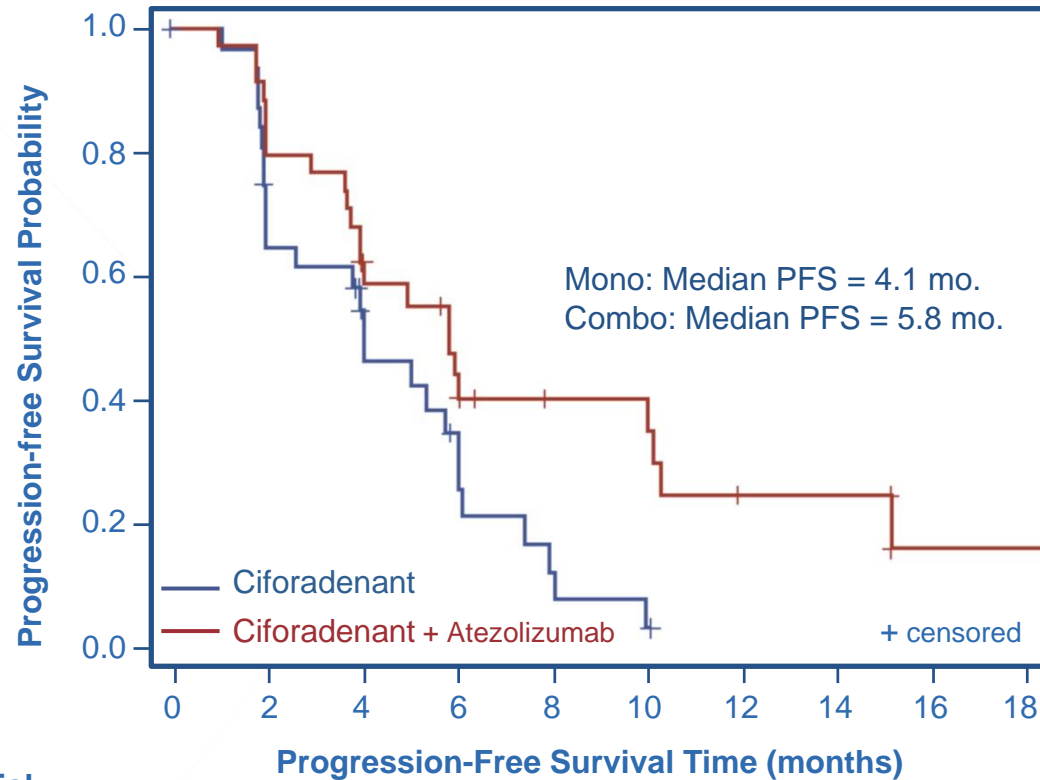
→ = Patients continuing on treatment

Median time to best tumor reduction:

- Monotherapy 3.4 months
- Combination 5.5 months

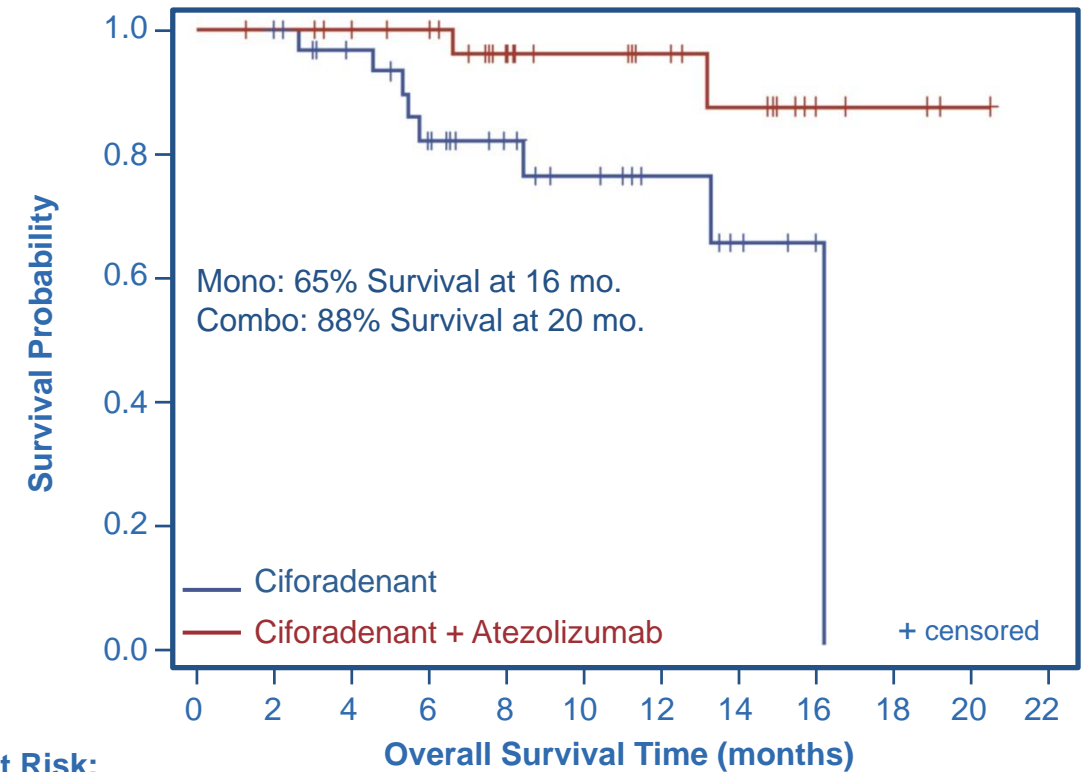
PROGRESSION FREE AND OVERALL SURVIVAL

Median follow-up 8.7 months



N. At Risk:

	33	23	15	8	3	1			
Ciforadenant	33	23	15	8	3	1			
Ciforadenant + Atezolizumab	35	31	23	12	8	7	4	4	1



N. At Risk:

	33	33	27	22	15	11	7	4	2	0
Ciforadenant	33	33	27	22	15	11	7	4	2	0
Ciforadenant+ Atezolizumab	35	34	32	30	22	16	13	10	5	3

TREATMENT-RELATED ADVERSE EVENTS

- Ciforadenant is well tolerated as monotherapy and in combination

Adverse Event	Number (%) of Patients			
	Monotherapy (N=33)		Combination Therapy (N=35)	
	Any Grade	Grade 3 or 4	Any Grade	Grade 3 or 4
Nausea	3(9.1)	0(0.0)	7(20.0)	1(2.9)
Arthralgia	2(6.1)	1(3.0)	4(11.4)	1(2.9)
Hypophosphataemia	2(6.1)	0(0.0)	3(8.6)	1(2.9)
Abdominal pain	1(3.0)	0(0.0)	3(8.6)	1(2.9)
Aspartate aminotransferase increased	1(3.0)	0(0.0)	2(5.7)	1(2.9)
Decreased appetite	4(12.1)	1(3.0)	6(17.1)	0(0.0)
Anaemia	2(6.1)	1(3.0)	4(11.4)	0(0.0)
Oedema peripheral	2(6.1)	1(3.0)	1(2.9)	0(0.0)
Fatigue	13(39.4)	0(0.0)	16(45.7)	0(0.0)
Pruritus	7(21.2)	0(0.0)	9(25.7)	0(0.0)
Diarrhoea	2(6.1)	0(0.0)	5(14.3)	0(0.0)
Vomiting	2(6.1)	0(0.0)	4(11.4)	0(0.0)
Dizziness	4(12.1)	0(0.0)	1(2.9)	0(0.0)
Cough	2(6.1)	0(0.0)	3(8.6)	0(0.0)
Rash	2(6.1)	0(0.0)	3(8.6)	0(0.0)
Influenza like illness	0(0.0)	0(0.0)	3(8.6)	0(0.0)
Pyrexia	3(9.1)	0(0.0)	1(2.9)	0(0.0)
Musculoskeletal chest pain	2(6.1)	0(0.0)	2(5.7)	0(0.0)
Myalgia	2(6.1)	0(0.0)	2(5.7)	0(0.0)
Osteoarthritis	2(6.1)	0(0.0)	2(5.7)	0(0.0)
Blood creatinine increased	1(3.0)	0(0.0)	2(5.7)	0(0.0)
Insomnia	1(3.0)	0(0.0)	2(5.7)	0(0.0)
Dysgeusia	0(0.0)	0(0.0)	2(5.7)	0(0.0)
Musculoskeletal pain	0(0.0)	0(0.0)	2(5.7)	0(0.0)
Neuropathy peripheral	0(0.0)	0(0.0)	2(5.7)	0(0.0)
Paraesthesia	0(0.0)	0(0.0)	2(5.7)	0(0.0)
Rash maculo-papular	0(0.0)	0(0.0)	2(5.7)	0(0.0)
Chills	2(6.1)	0(0.0)	1(2.9)	0(0.0)
Hyperhidrosis	2(6.1)	0(0.0)	1(2.9)	0(0.0)
Epistaxis	2(6.1)	0(0.0)	0(0.0)	0(0.0)

Case Report - Monotherapy

Patient 103035 - 58-year-old male patient with AdenoSig+ disease in lung, pleura, mediastinum, lymph node, chest wall, and bone treated in the past with:

- **MEDI4736 (Durvalumab)**: for 2.3 months and progressed. Stopped treatment 23 months prior to CPI-444.
- Pazopanib: for ~1 month and came off due to toxicity and had progression.
- Axitinib: Received for 5.5 months and achieved an SD. Discontinued due to “other” reason.
- Cabozantinib: Most recent treatment, which he received for 8.5 months, achieved a SD. Stopped due to progression of the diseases. He discontinued 3.4 months before being enrolled on trial.

Maximum decrease in SLD was - 30.1 % on ciforadenant monotherapy and had PR as best response. He remained on therapy for 5.6 months and discontinued due to PD.

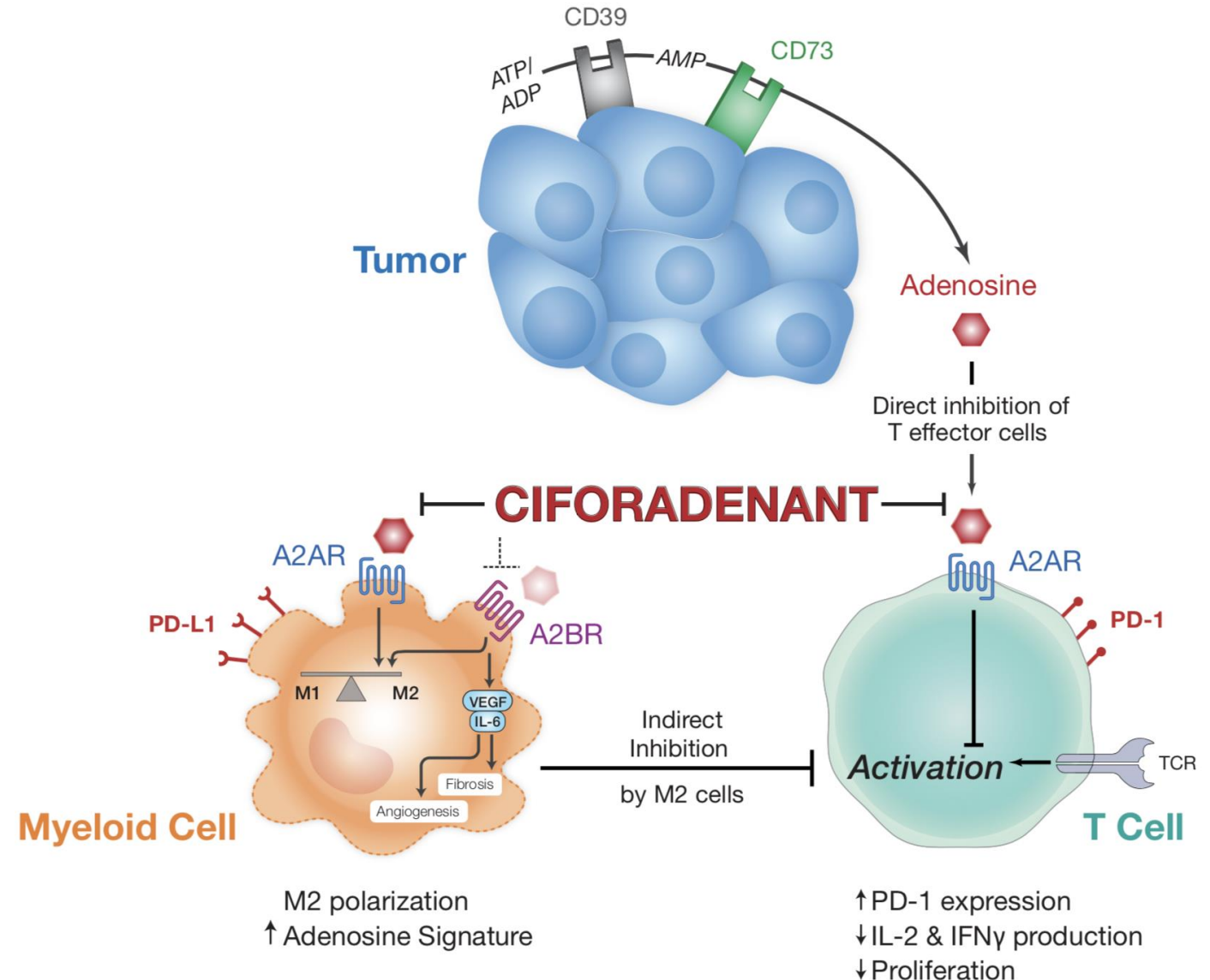
BIOMARKERS TO ASSESS IMMUNE FUNCTION AND CLINICAL ACTIVITY

Intra tumoral adenosine leads to:

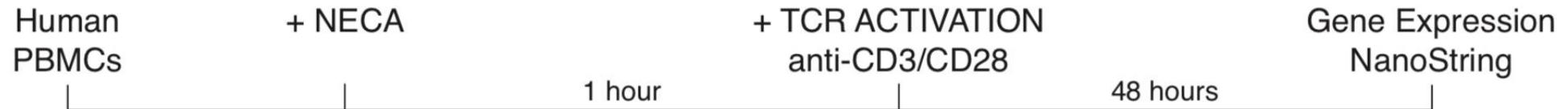
- T cell suppression
- M2 polarization

Hypothesis:

- Ciforadenant treatment will enhance T cell responses
- Patients with M2 skewed tumors may be most sensitive to treatment



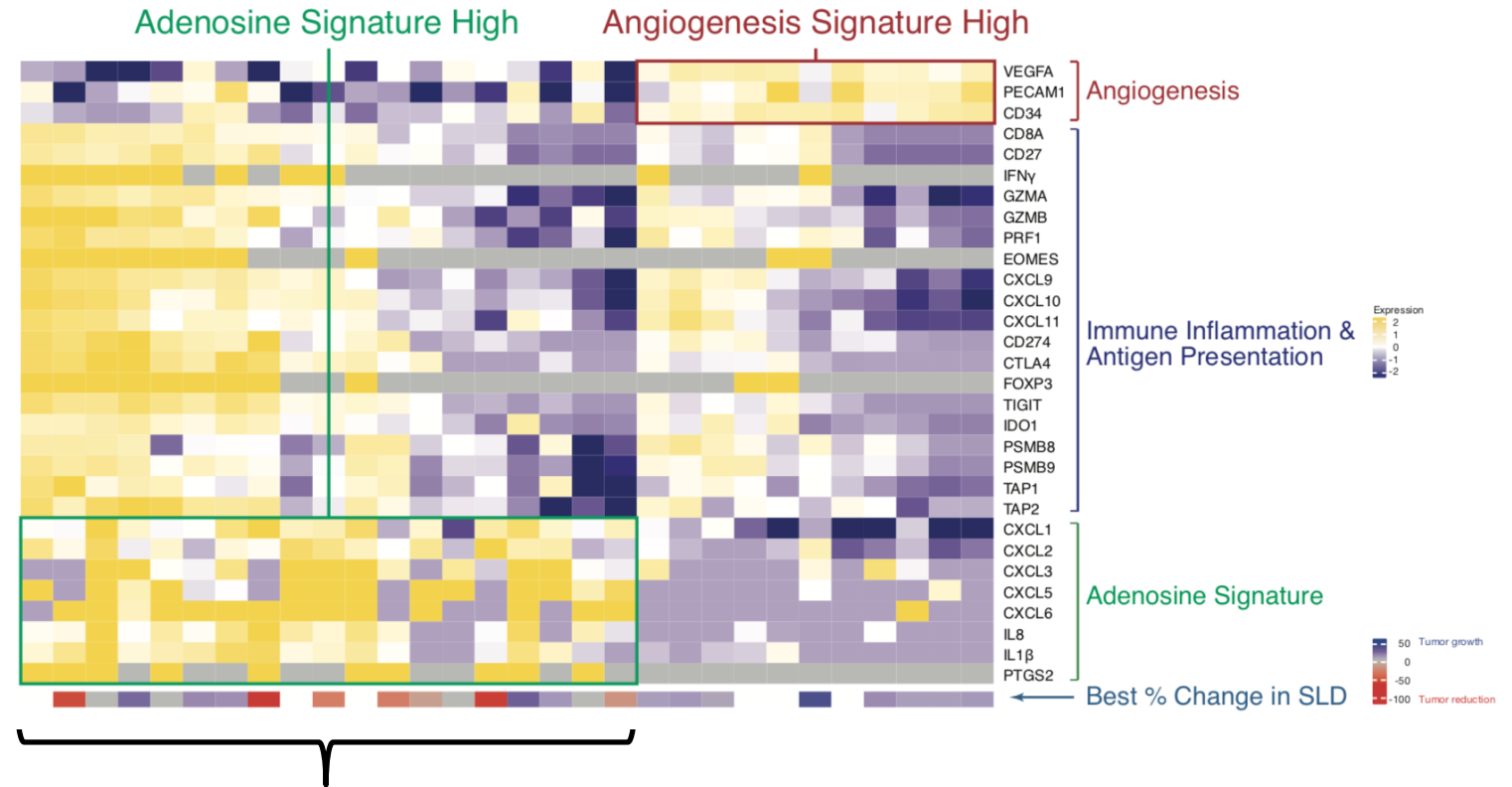
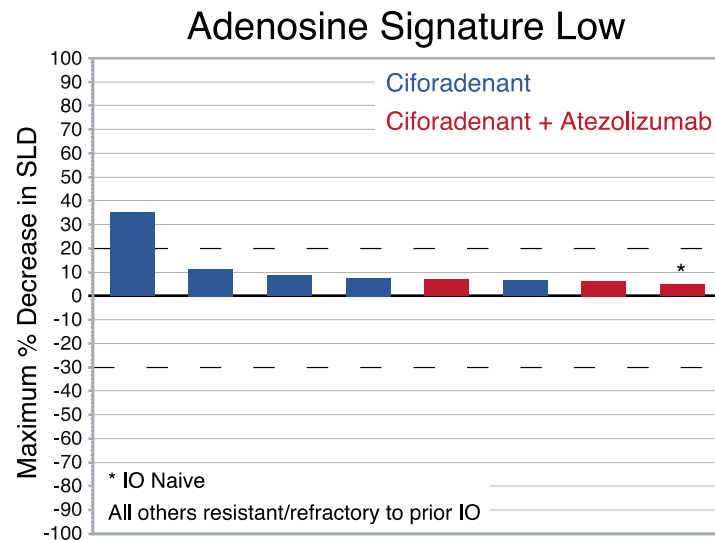
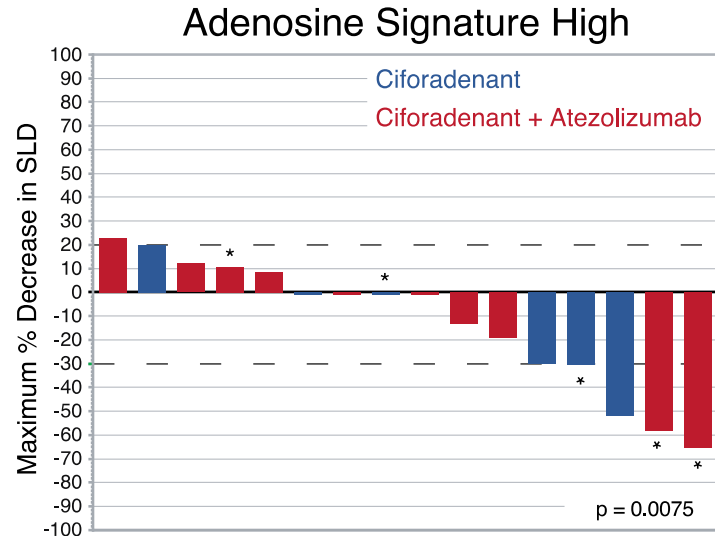
Adenosine Gene Expression Signature



ADENOSINE SIGNATURE - NANOSTRING

	Adjusted p Value	Function
CXCL1	3.92E-03	Neutrophil chemotractant
CXCL2	1.27E-03	MIP2a: macrophage inflammatory protein 2, alpha
CXCL3	1.40E-03	Controls migration and adhesion of monocytes
CXCL5	1.98E-03	Attracts and activates neutrophils
CXCL6	1.40E-03	GCP2: Granulocyte chemotactic protein 2
CXCL8	1.98E-03	IL-8. Neutrophil chemotactic factor
IL-1 β	2.38E-03	Inflammation
PTGS2	2.65E-03	COX-2. Elevated during inflammation and cancer

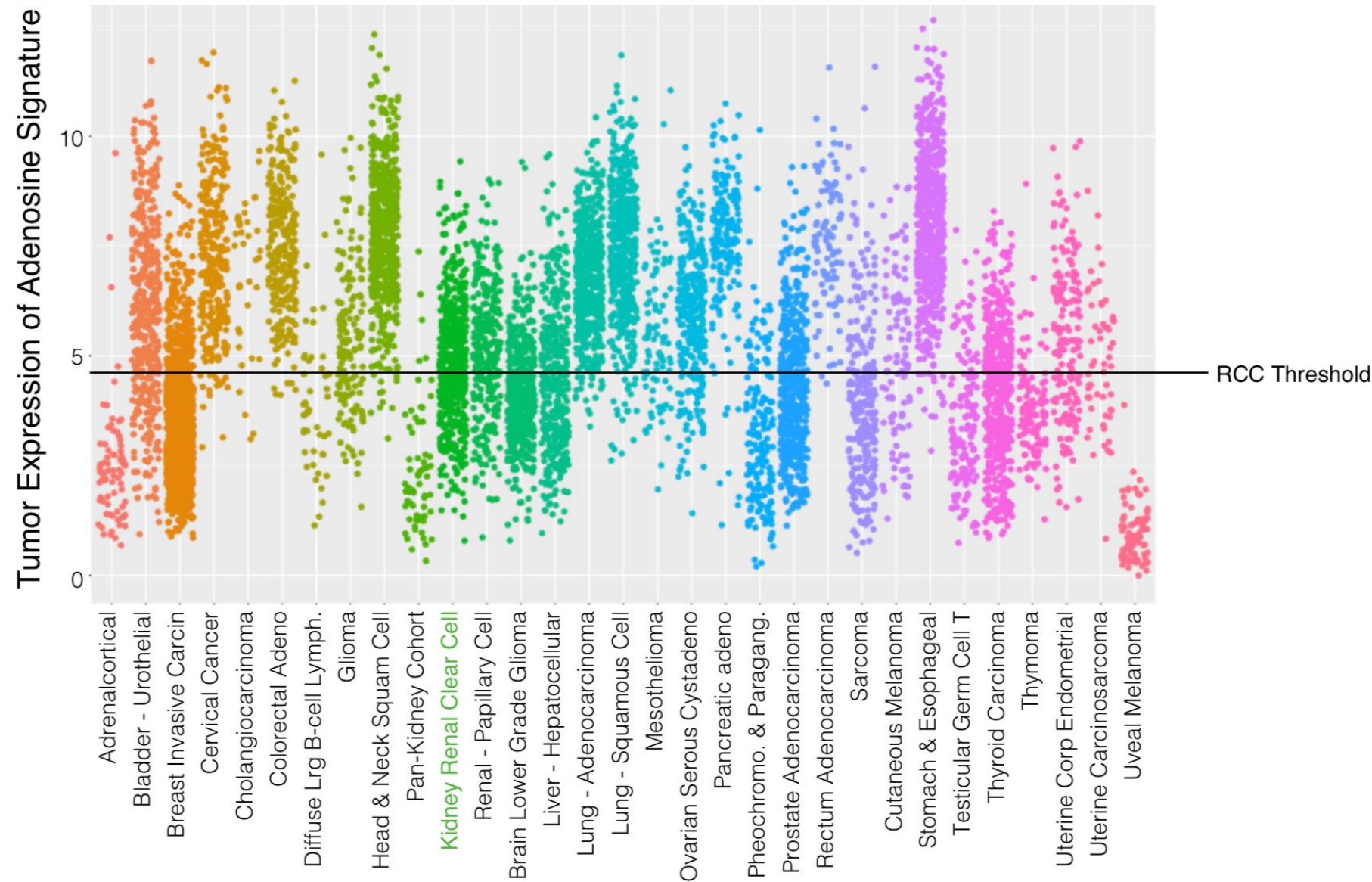
Adenosine Gene Expression Signature Correlates with Tumor Response



- Enriched for ciforadenant response
- Angio^{Low}: Poor PFS with TKI (Sunitinib)¹
- Myeloid^{High}: Poor PFS with single agent atezo¹

Ciforadenant activity to date observed in AdenoSig^{High}, Angio^{Low} tumors

Prevalence of the Adenosine Signature in TCGA



Comparison of RCC Therapies

	Line of Rx	No. Pts	Prior IO	ORR %	PFS (mo) / OS
Ciforadenant/Atezo¹	4th	35	72%	11	5.8 / 88% 20 mo.
Atezo ²	3 rd	70	No	15	5.6 / 28.9 mo. median
Nivo ³	2 nd	410	No	24	4.6 / 25 mo. median
Everolimus ³	2 nd	411	No	5	4.4 / 19.6 mo. median
Nivo/Ipi ⁴	1 st	425	No	42	11.6 / 75% 18 mo.
Sutent ⁴	1 st	422	No	27	8.4 / 26 mo. median
Cabozantinib ⁵	2 nd or 3 rd	223	No	16	~8.3* / 22 mo. median
Cabozantinib ⁶	3 rd **	25	100%	28	4.7 / 40% 12 mo.

*Prior sunitinib 9.1 months; prior pazopanib 7.4 months

** Prior IO+TKI

1. Fong et al, SITC 201

2. McDermott et al, JCO 2016

3. Motzer et al, NEJM 2015

4. Motzer et al, NEJM 2018

5. Powles et al, BJC 2018

6. McGregor ESMO 2018

Current Phase 1b/2 Studies

Earlier stage patients, TKI and anti-PD(L)-1 failures

	Renal Cell Cancer	Non-small cell lung cancer
Status	Enrolling	Ph 1b/2 GNE-Morpheus Enrolling
Eligibility	1 or 2 prior regimens including anti-PD(L)1 and TKI	1 or 2 prior regimens including anti-PD(L)1 and platinum agent
Design	Single arm: atezo + Ciforadenant	Randomized: atezo + Ciforadenant vs. docetaxel
Endpoint	Overall Response Rate	Overall Response Rate
Sample Size	≤50 patients	Up to 65 patients
Biomarker	Adenosine Signature	Adenosine Signature

Proposed Pivotal Clinical Trials In Renal Cell Cancer

Potential routes to approval

Clinical Trial Design	
	2 nd Line/3 rd Line
Eligibility	<ul style="list-style-type: none">• RCC failed IO + TKI
Design	<ul style="list-style-type: none">• Ph 2 in AdenoSig+: Ciforadenant+anti-PD(L)-1• Ph 3: Ciforadenant+anti-PD(L)-1 vs TKI
Endpoints	<ul style="list-style-type: none">• OS, PFS, ORR
Biomarker	<ul style="list-style-type: none">• AdenoSig+• Hierarchical analysis

CPI-006 Ph 1/1b Clinical Trial Design

Single agent and combination with CPI-444, and with anti-PD1

DOSE ESCALATION (3X3 DESIGN)

CPI-006 alone

CPI-006 + CPI-444

CPI-006 +
pembrolizumab

DOSE EXPANSION - STAGE 1 (N=11 PER COHORT)

CPI-006 alone

CPI-006 + CPI-444

CPI-006 +
pembrolizumab

NSCLC

RCC

Others

NSCLC

RCC

Others

NSCLC

RCC

Others

If ≥ 1 response observed in a disease cohort

DOSE EXPANSION - STAGE 2 (N=17 PER COHORT)

CPI-006 Ph 1/1b Clinical Trial Design

Single agent and combination with CPI-444, and with anti-PD1

DOSE ESCALATION (3X3 DESIGN)

CPI-006 alone

CPI-006 + CPI-444

CPI-006 +
pembrolizumab

DOSE EXPANSION - STAGE 1 (N=11 PER COHORT)

CPI-006 alone

CPI-006 + CPI-444

CPI-006 +
pembrolizumab

NSCLC

RCC

Others

NSCLC

RCC

Others

NSCLC

RCC

Others

If ≥ 1 response observed in a disease cohort

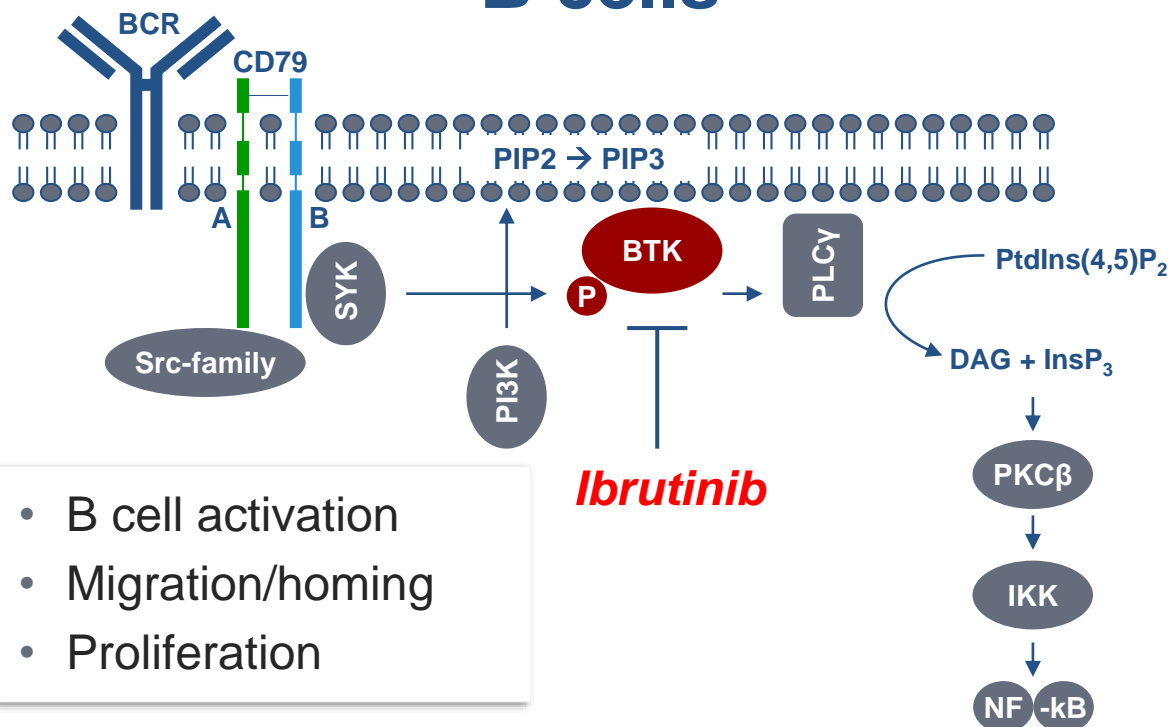
DOSE EXPANSION - STAGE 2 (N=17 PER COHORT)

ITK and BTK are Homologous Kinases

Founding scientists of Corvus pioneered covalent kinase inhibition with Ibrutinib



B cells



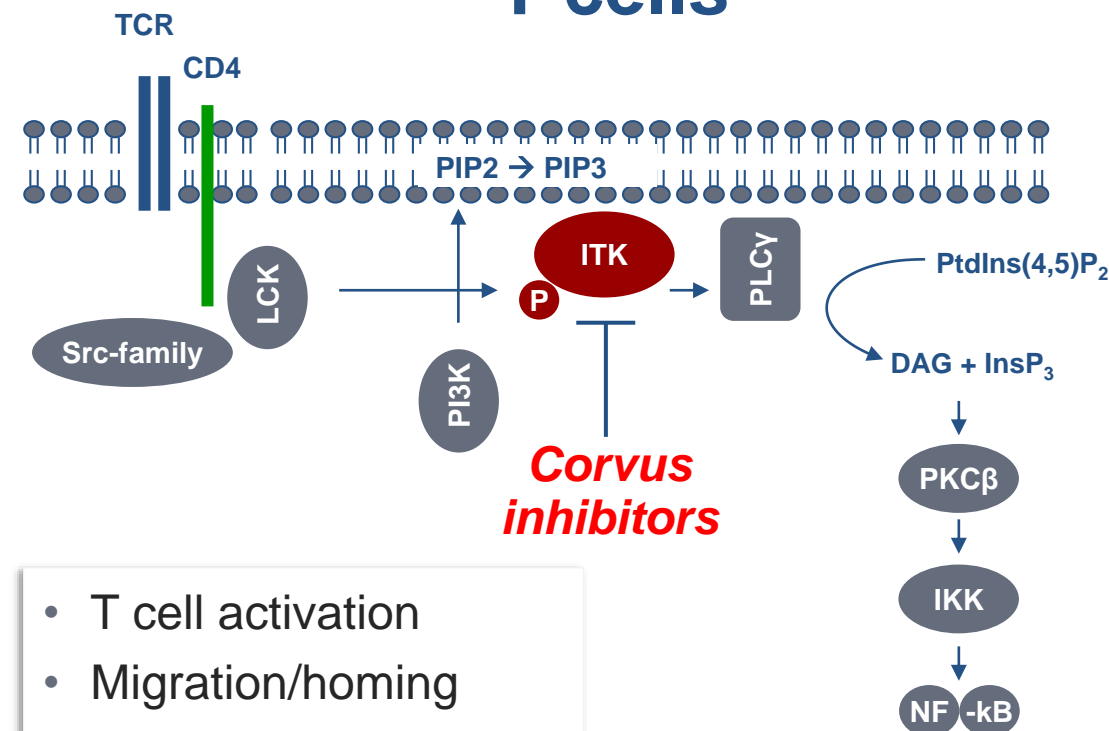
- B cell activation
- Migration/homing
- Proliferation

Ibrutinib

The Bruton tyrosine kinase inhibitor PCI-32765 blocks B-cell activation and is efficacious in models of autoimmune disease and B-cell malignancy *PNAS* 2010

Lee A. Honigberg^{a,1}, Ashley M. Smith^{a,1}, Mint Sirisawad^a, Erik Verner^a, David Loury^a, Betty Chang^a, Shyr Li^{b,c}, Zhengying Pan^{b,d}, Douglas H. Thamm^e, Richard A. Miller^{a,f}, and Joseph J. Buggy^{a,2}

T cells

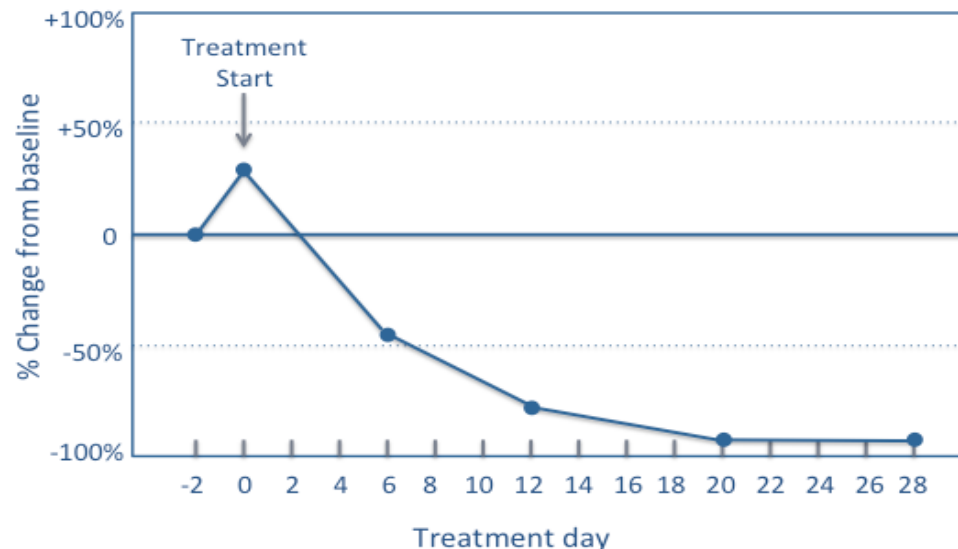


- T cell activation
- Migration/homing
- Proliferation
- Th1 skewing

Corvus inhibitors

Tumor Responses With ITKi in Canine T Cell Lymphoma

Naturally occurring disease in companion dogs



Chloe
7 yo
Boxer
Aggressive PTCL



14 days



Rudy
11 yo
Golden Retriever
CTCL

Complete Response in Dog CTCL

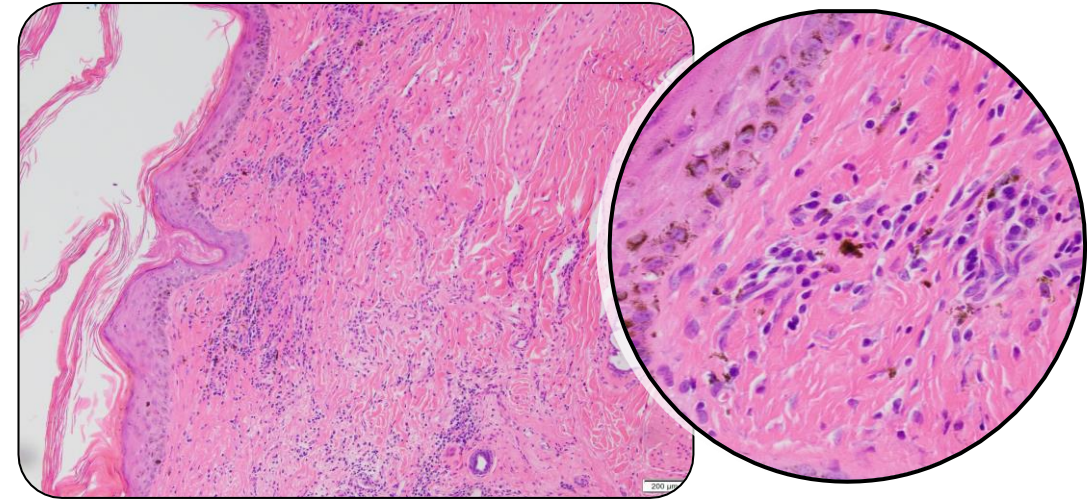
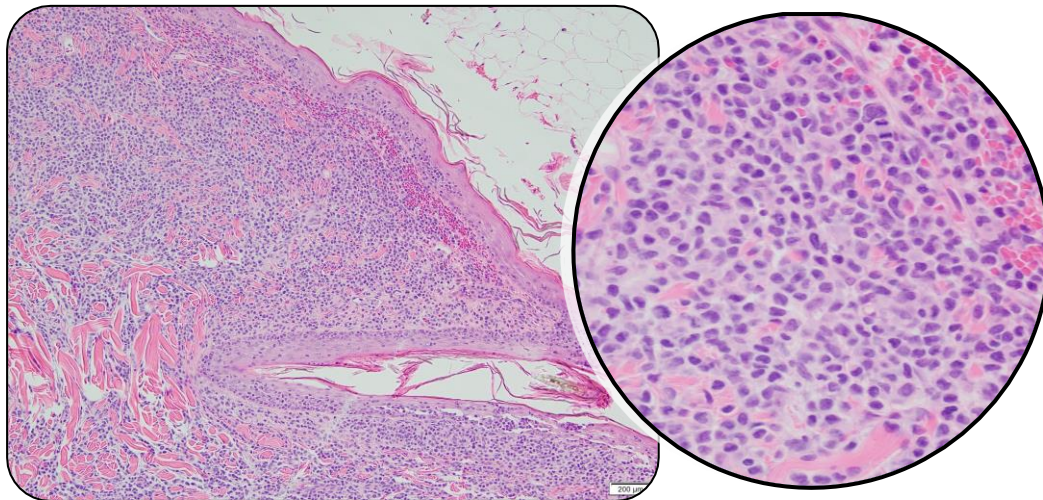
Complete elimination of tumor infiltrates in skin

Copper

11 year old, Male
Golden Retriever



4 months



CPI-818 Ph 1/1b Clinical Trial Design

Relapsed/Refractory T-Cell Lymphomas

DOSE ESCALATION (3X3 DESIGN)

CPI-818

DOSE EXPANSION - STAGE 1 (N=11 PER COHORT)

CPI-818

PTCL

AITL

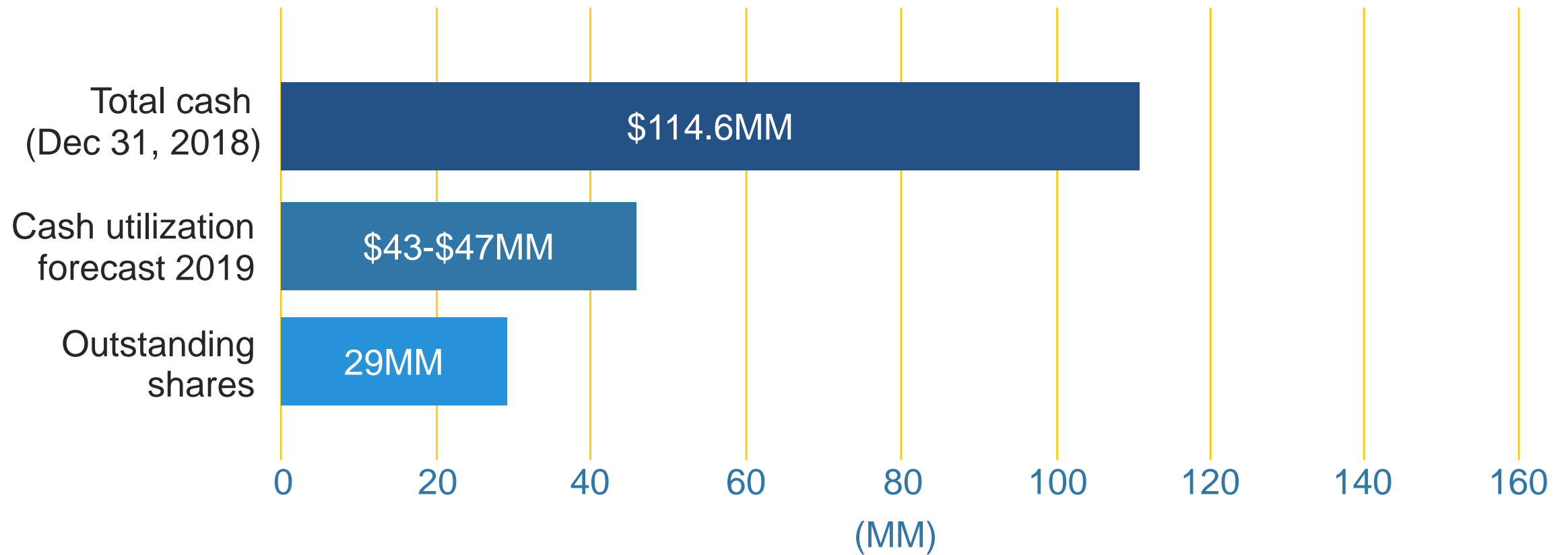
CTCL

Others

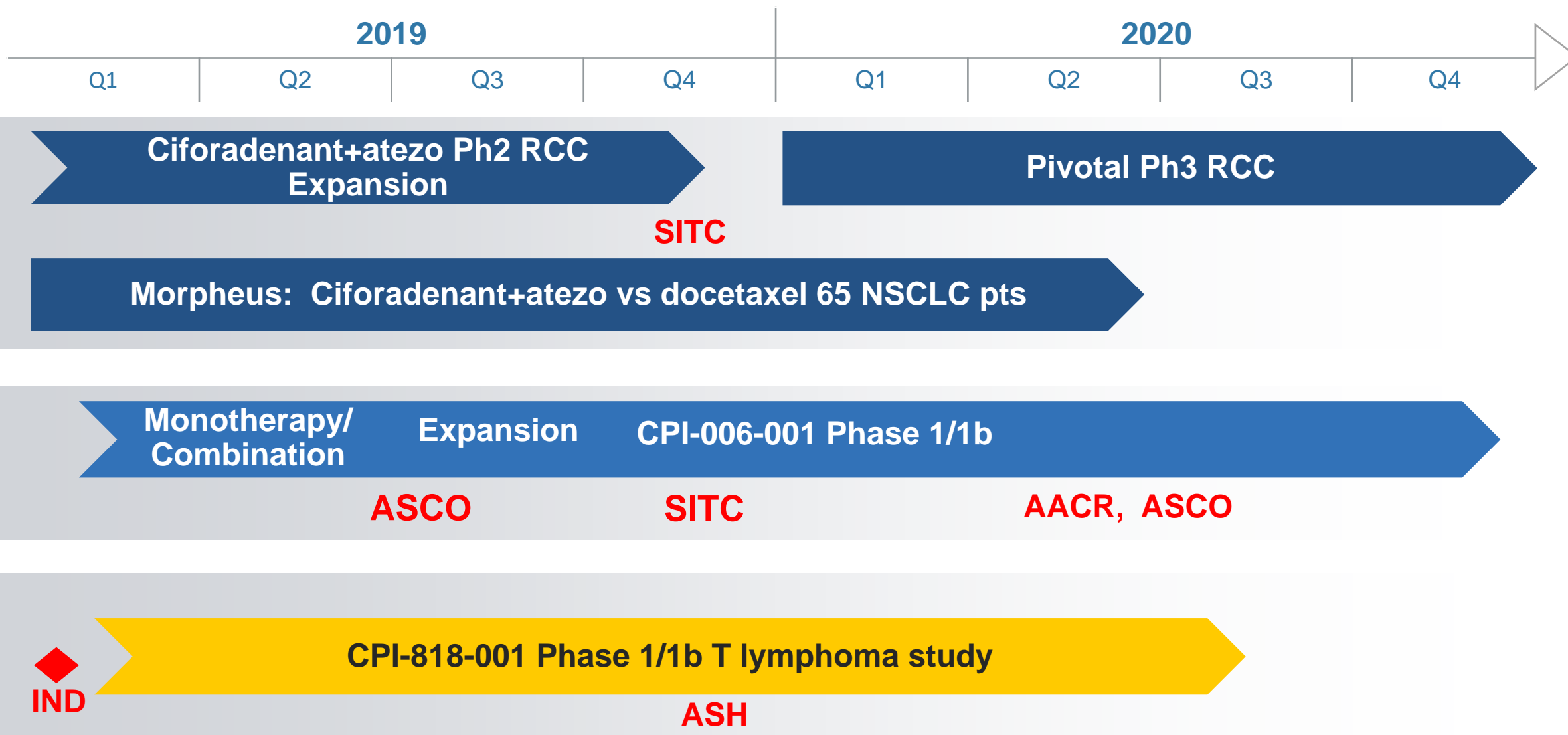
If ≥ 2 responses observed in a disease cohort

DOSE EXPANSION - STAGE 2 (N=17 PER COHORT)

Financials



Near-Term Milestones and Value-Drivers



Corvus Corporate Presentation