Corvus Corporate Presentation

August 2022

An immunology focused company developing drugs and antibodies that target the most critical elements of the tumor immunity axis

CORVUS PHARMACEUTICALS

Forward-Looking Statements / Safe Harbor

This presentation and the accompanying oral presentation contain “forward-looking” statements, including statements related to the potential safety and efficacy of CPI-818, ciforadenant and mupadolimab; the Company’s ability and Angel Pharmaceutical’s ability to develop and advance product candidates into and successfully complete preclinical studies and clinical trials, including the Company’s plan to initiate a Phase 2 clinical trial with ciforadenant in collaboration with the Kidney Cancer Clinical Trials Consortium, the timing of the availability and announcement of clinical data and certain other product development milestones, including the timing of results in the Phase 1/1b clinical trial of CPI-818, and in the ;planned Phase2 clinical trial of ciforadenant. All statements other than statements of historical fact contained in this press release are forward-looking statements. These statements often include words such as “believe,” “expect,” “anticipate,” “intend,” “plan,” “estimate,” “seek,” “will,” “may” or similar expressions. Forward-looking statements are subject to a number of risks and uncertainties, many of which involve factors or circumstances that are beyond the Company’s control. The Company’s actual results could differ materially from those stated or implied in forward-looking statements due to a number of factors, including but not limited to, risks detailed in the Company’s Quarterly Report on Form 10-Q for the quarter ended June 30, 2022, filed with the Securities and Exchange Commission on or about August 8, 2022, as well as other documents that may be filed by the Company from time to time with the Securities and Exchange Commission. In particular, the following factors, among others, could cause results to differ materially from those expressed or implied by such forward-looking statements: the Company’s ability to demonstrate sufficient evidence of efficacy and safety in its clinical trials of CPI-818, ciforrdadenant and mupadolimab; the accuracy of the Company’s estimates relating to its ability to initiate and/or complete preclinical studies and clinical trials; the results of preclinical studies may not be predictive of future results; the unpredictability of the regulatory process; regulatory developments in the United States, and other foreign countries; regulatory developments in the United States, and other foreign countries; the costs of clinical trials may exceed expectations; the Company’s ability to accurately estimate available cash providing funding into early 2024 and the Company’s ability to raise additional capital. Although the Company believes that the expectations reflected in the forward-looking statements are reasonable, it cannot guarantee that the events and circumstances reflected in the forward-looking statements will be achieved or occur, and the timing of events and circumstances and actual results could differ materially from those projected in the forward-looking statements. Accordingly, you should not place undue reliance on these forward-looking statements. All such statements speak only as of the date made, and the Company undertakes no obligation to update or revise publicly any forward-looking statements, whether as a result of new information, future events or otherwise. The Company’s results for the quarter ended March 31, 2022 are not necessarily indicative of its operating results for any future periods.

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.

Corvus Development Strategy

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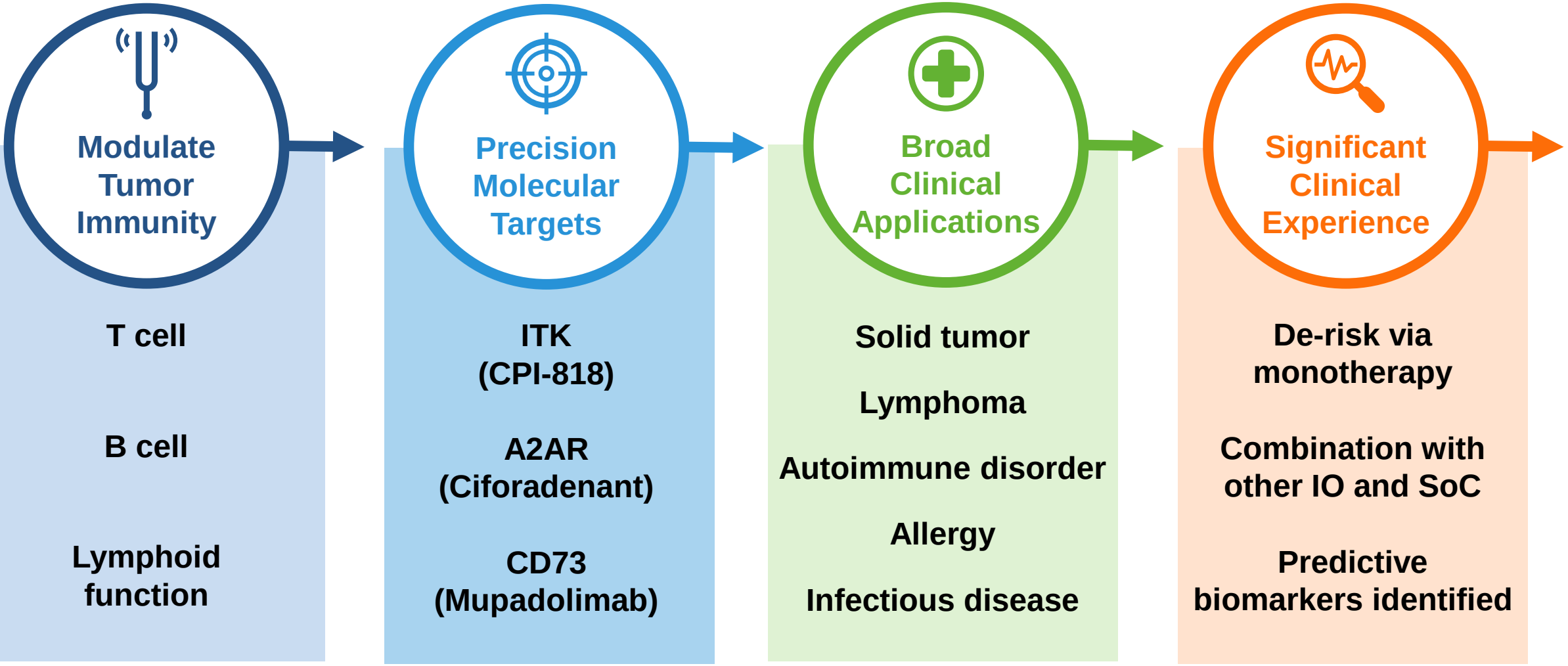

Corvus Precision Immunotherapy

Controlling multiple steps in the tumor immunity axis

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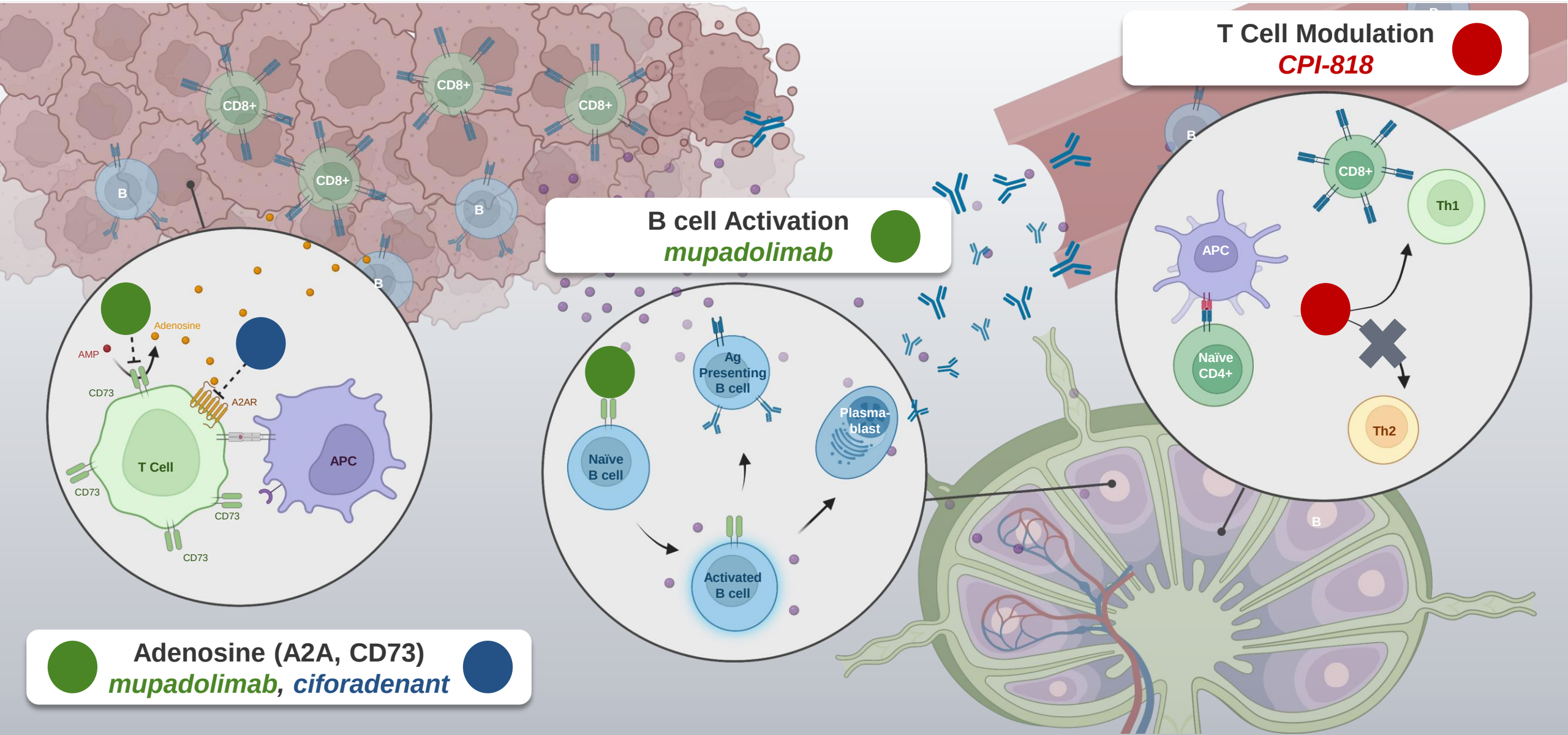


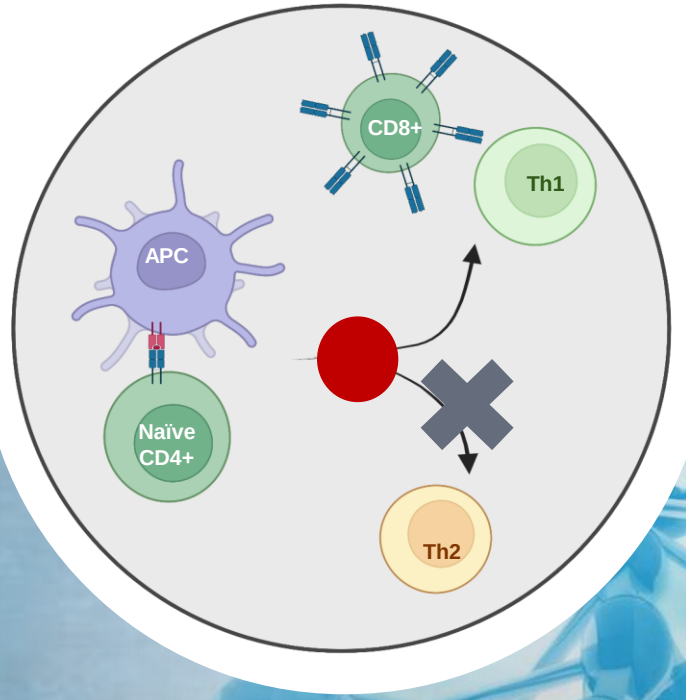
Corvus Pharmaceuticals Overview

Efficiently advancing clinical programs

Target	Program	Indication	IND enabling	Phase 1a	Phase 1b	Phase 2
ITK Inhibitor	CPI-818	T Cell Lymphoma			Data Anticipated in 2H22	
		Autoimmunity / Allergy				
A2A Inhibitor	Ciforadanent	r/r RCC *Mono or in combo with Atezolizumab*				
		Frontline RCC *In combo with Nivo and Ipi*			Plan to Initiate Trial in 3Q22	
Anti-CD73	Mupadolimab	*Frontline Stage IV NSCLC* *Mono or in combo with Pembro + Chemo*				
		r/r Advanced Tumors *Mono or in combo with anti-PD-1*				
		r/r NSCLC and HNSCC *Mono or in combo with anti-PD-1*				
Anti-CXCR2	CPI-182	Multiple Cancers				
		Inflammation				
A2B Inhibitor	CPI-935	Fibrosis				

CPI-818

Novel ITK Inhibitor



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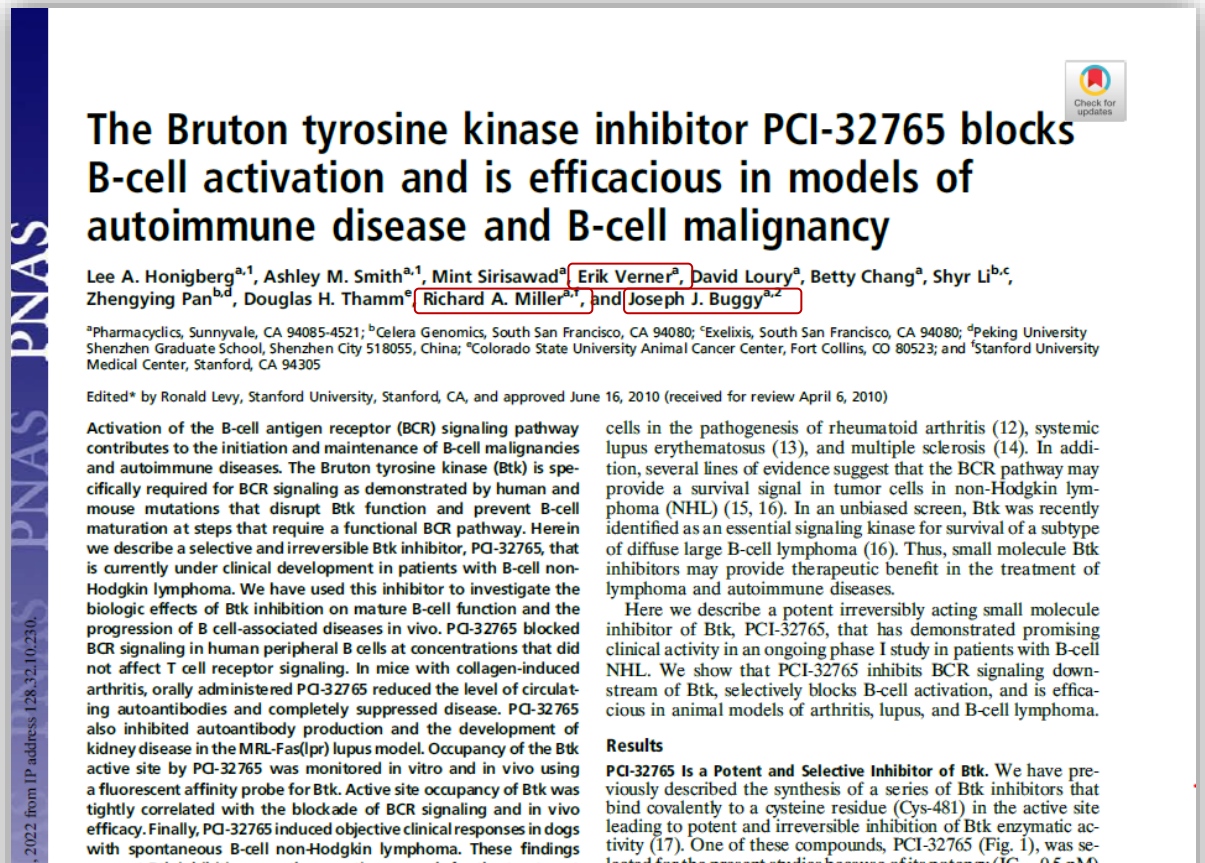
Proven Track Record
Team developed ibrutinib

- Founders of Corvus developed ibrutinib
- Among the top 4 oncology drugs by worldwide sales in 2021



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

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Edited* by Ronald Levy, Stanford University, Stanford, CA, and approved June 16, 2010 (received for review April 6, 2010)

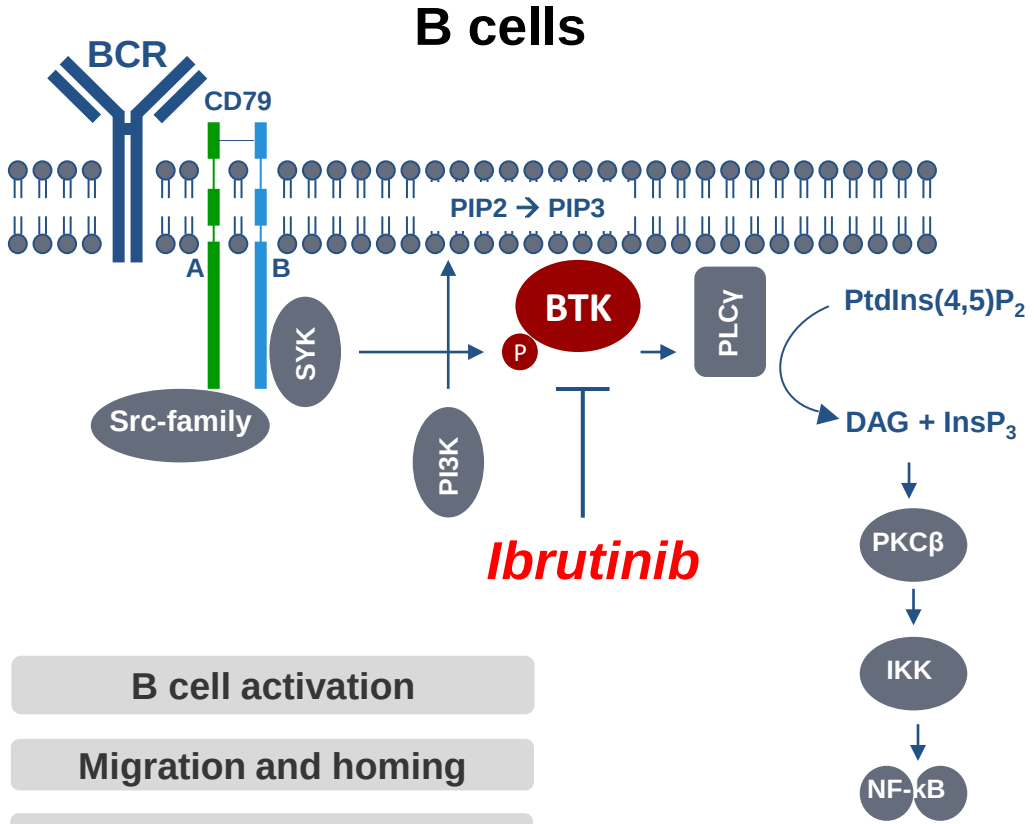
Activation of the B-cell antigen receptor (BCR) signaling pathway contributes to the initiation and maintenance of B-cell malignancies and autoimmune diseases. The Bruton tyrosine kinase (Btk) is specifically required for BCR signaling as demonstrated by human and mouse mutations that disrupt Btk function and prevent B-cell maturation at steps that require a functional BCR pathway. Herein we describe a selective and irreversible Btk inhibitor, PCI-32765, that is currently under clinical development in patients with B-cell non-Hodgkin lymphoma. We have used this inhibitor to investigate the biologic effects of Btk inhibition on mature B-cell function and the progression of B cell-associated diseases in vivo. PCI-32765 blocked BCR signaling in human peripheral B cells at concentrations that did not affect T cell receptor signaling. In mice with collagen-induced arthritis, orally administered PCI-32765 reduced the level of circulating autoantibodies and completely suppressed disease. PCI-32765 also inhibited autoantibody production and the development of kidney disease in the MRL-Fas(lpr) lupus model. Occupancy of the Btk active site by PCI-32765 was monitored in vitro and in vivo using a fluorescent affinity probe for Btk. Active site occupancy of Btk was tightly correlated with the blockade of BCR signaling and in vivo efficacy. Finally, PCI-32765 induced objective clinical responses in dogs with spontaneous B-cell non-Hodgkin lymphoma. These findings

cells in the pathogenesis of rheumatoid arthritis (12), systemic lupus erythematosus (13), and multiple sclerosis (14). In addition, several lines of evidence suggest that the BCR pathway may provide a survival signal in tumor cells in non-Hodgkin lymphoma (NHL) (15, 16). In an unbiased screen, Btk was recently identified as an essential signaling kinase for survival of a subtype of diffuse large B-cell lymphoma (16). Thus, small molecule Btk inhibitors may provide therapeutic benefit in the treatment of lymphoma and autoimmune diseases.

Here we describe a potent irreversibly acting small molecule inhibitor of Btk, PCI-32765, that has demonstrated promising clinical activity in an ongoing phase I study in patients with B-cell NHL. We show that PCI-32765 inhibits BCR signaling downstream of Btk, selectively blocks B-cell activation, and is efficacious in animal models of arthritis, lupus, and B-cell lymphoma.

Results

PCI-32765 Is a Potent and Selective Inhibitor of Btk. We have previously described the synthesis of a series of Btk inhibitors that bind covalently to a cysteine residue (Cys-481) in the active site leading to potent and irreversible inhibition of Btk enzymatic activity (17). One of these compounds, PCI-32765 (Fig. 1), was se-

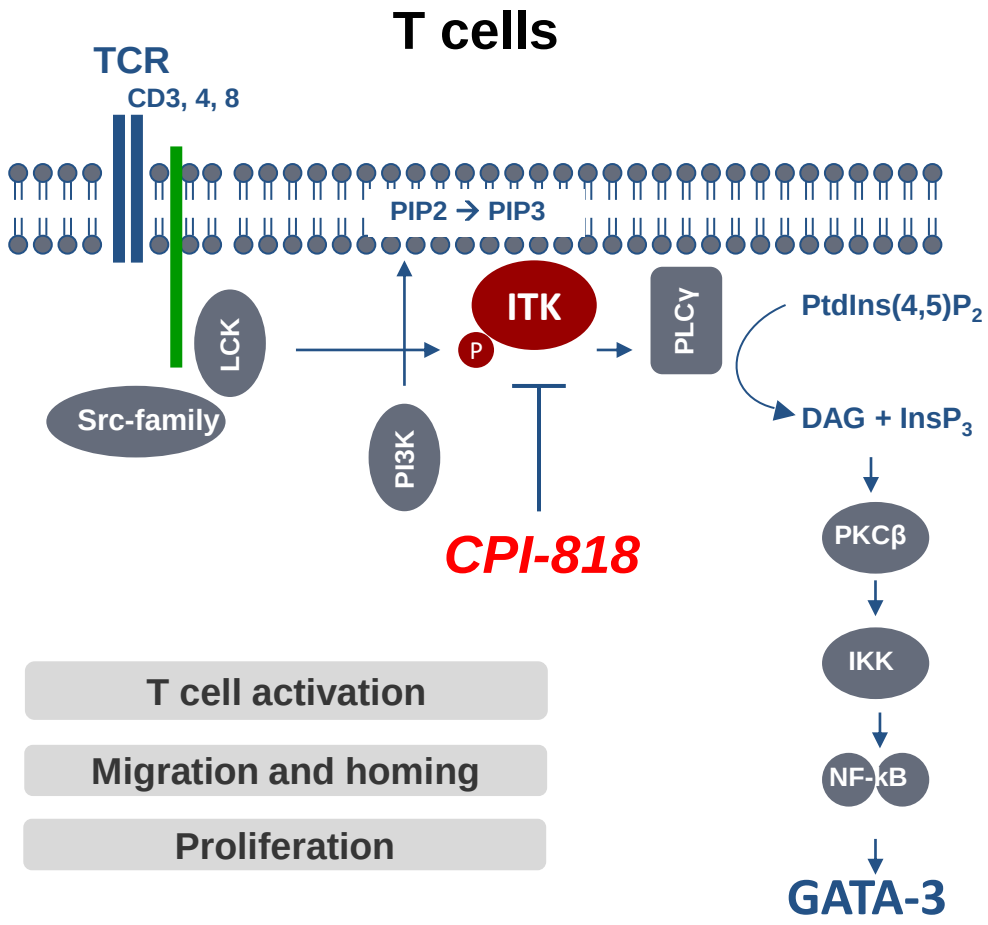


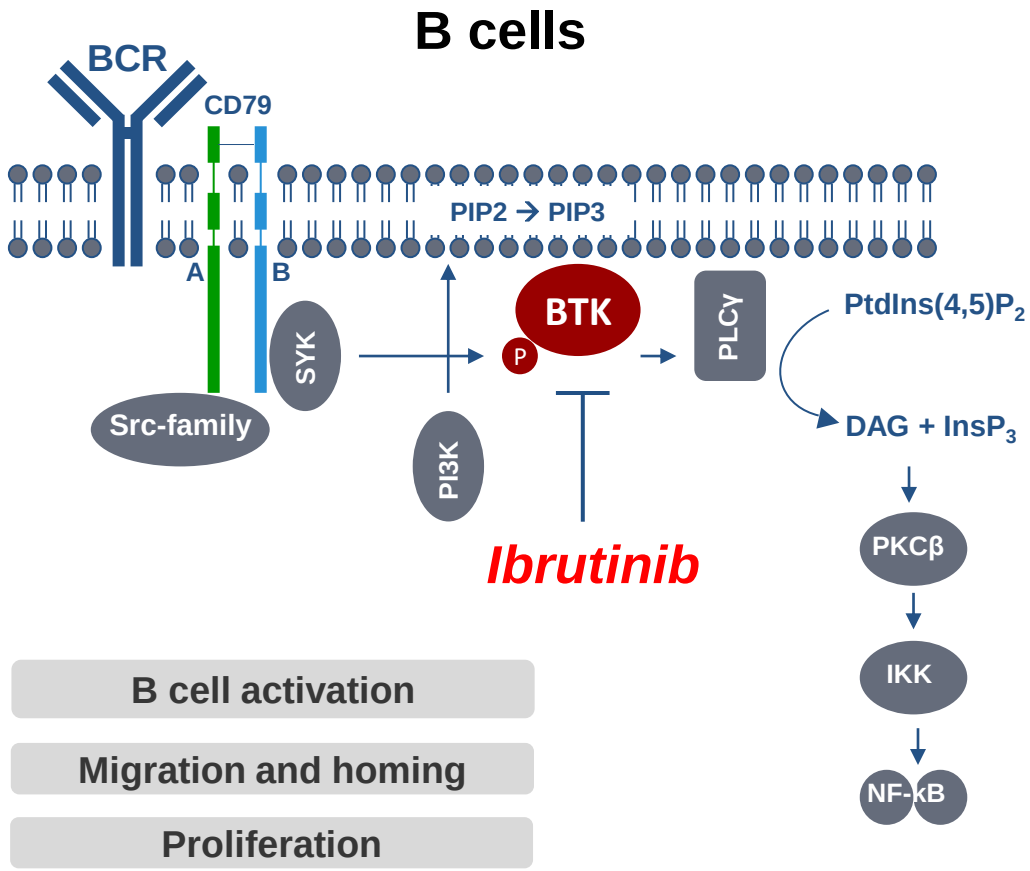
B cell activation

Migration and homing

Proliferation

CPI-818: Novel ITK Inhibitor





ITK Plays Critical Roles in T Cell Mediated Diseases

Selectivity is crucial for immune modulation

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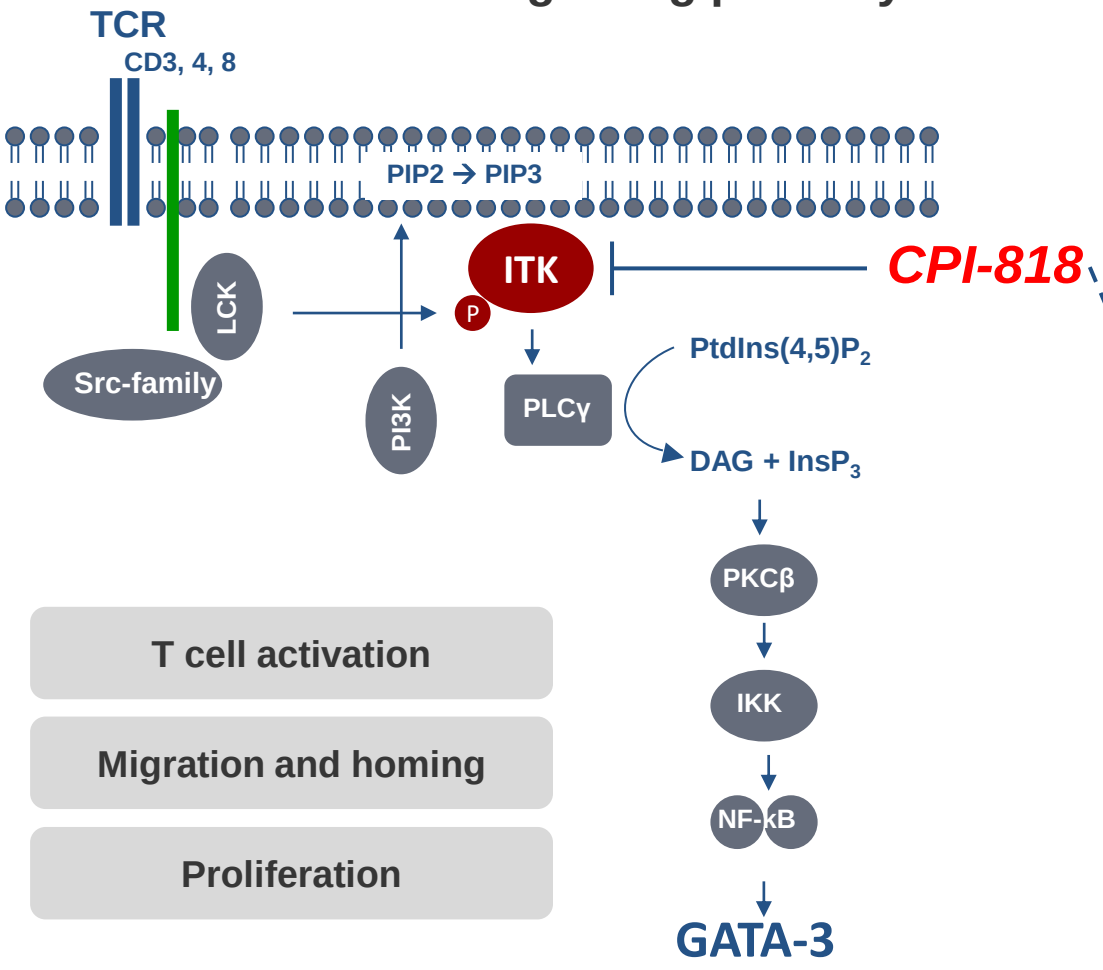
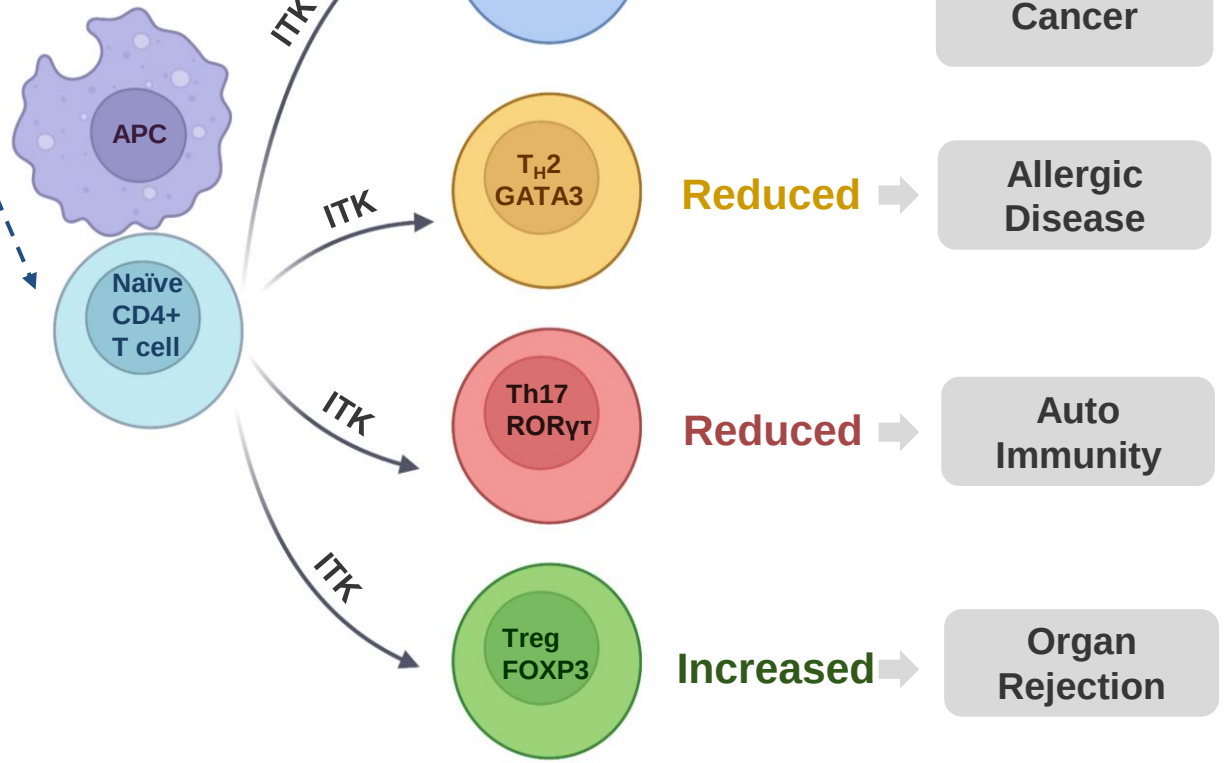
Blocks TCR signaling pathway



CPI-818

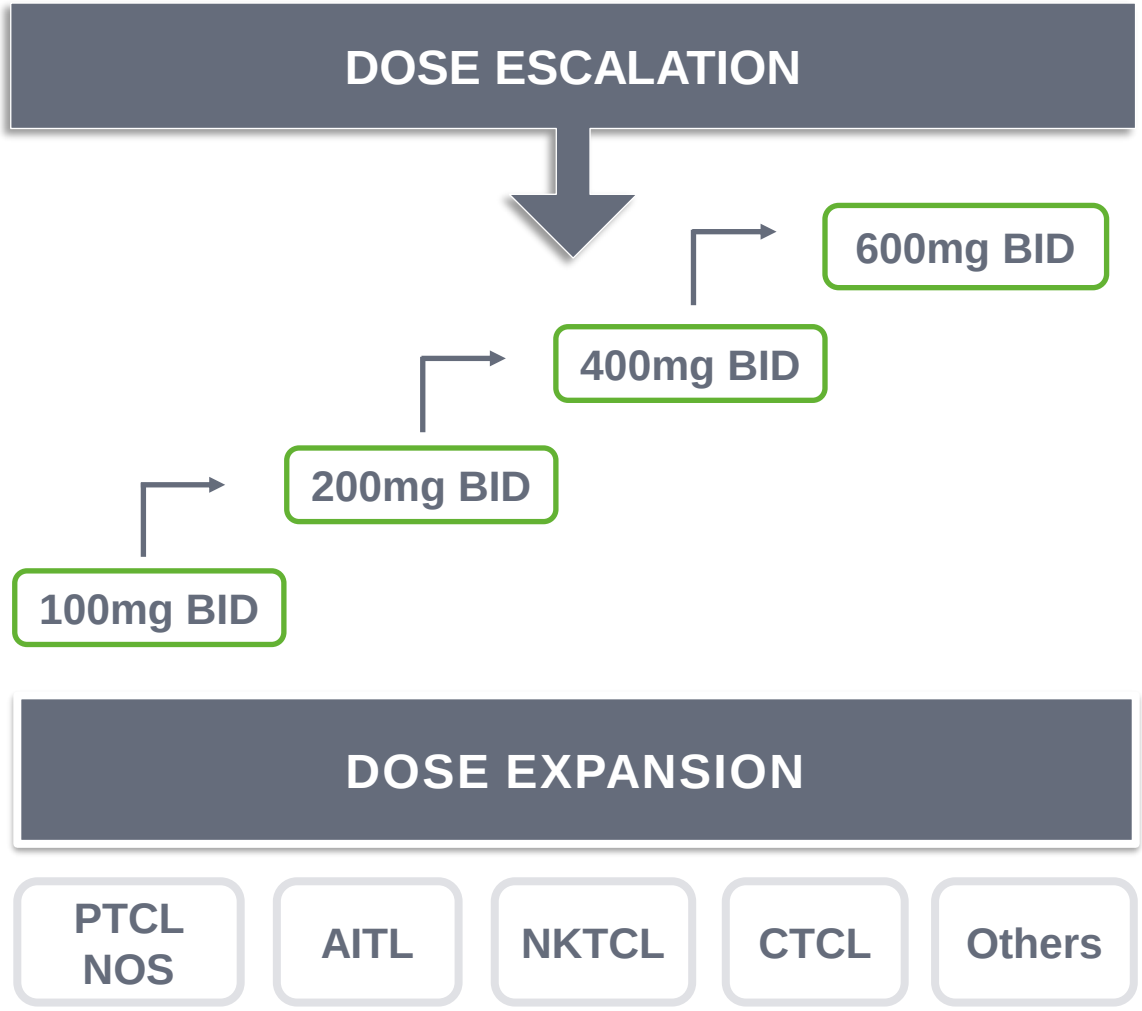
- T cell activation
- Migration and homing
- Proliferation

Modulates T helper cell differentiation



CPI-818 in T cell Lymphomas

Phase 1/1b clinical trial design



Design

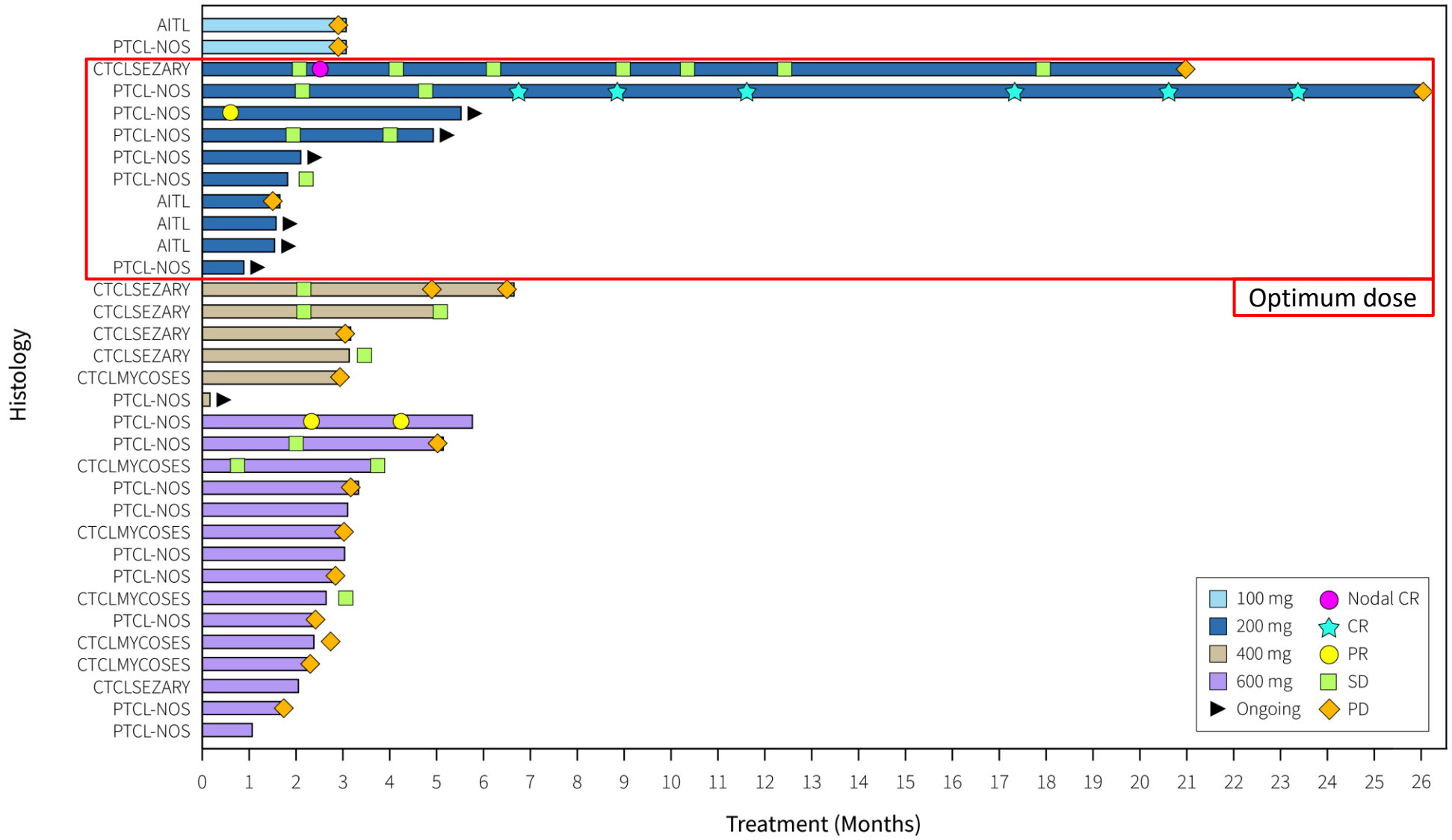
- Dose escalation 3+3 design
- Patients with T cell lymphoma (PTCL and CTCL) who have progressed on, refractory to, relapsed, to standard therapies
- CPI-818 orally BID continuously up to sixteen 21-day cycles, until progression or unacceptable toxicity

Objectives

- Primary: To establish safety / tolerability and determine MTD or MAD, as well as expansion cohort dose
- Secondary: PK/PD, biomarkers and efficacy

Interim Results of Anti-tumor Activity in PTCL & CTCL

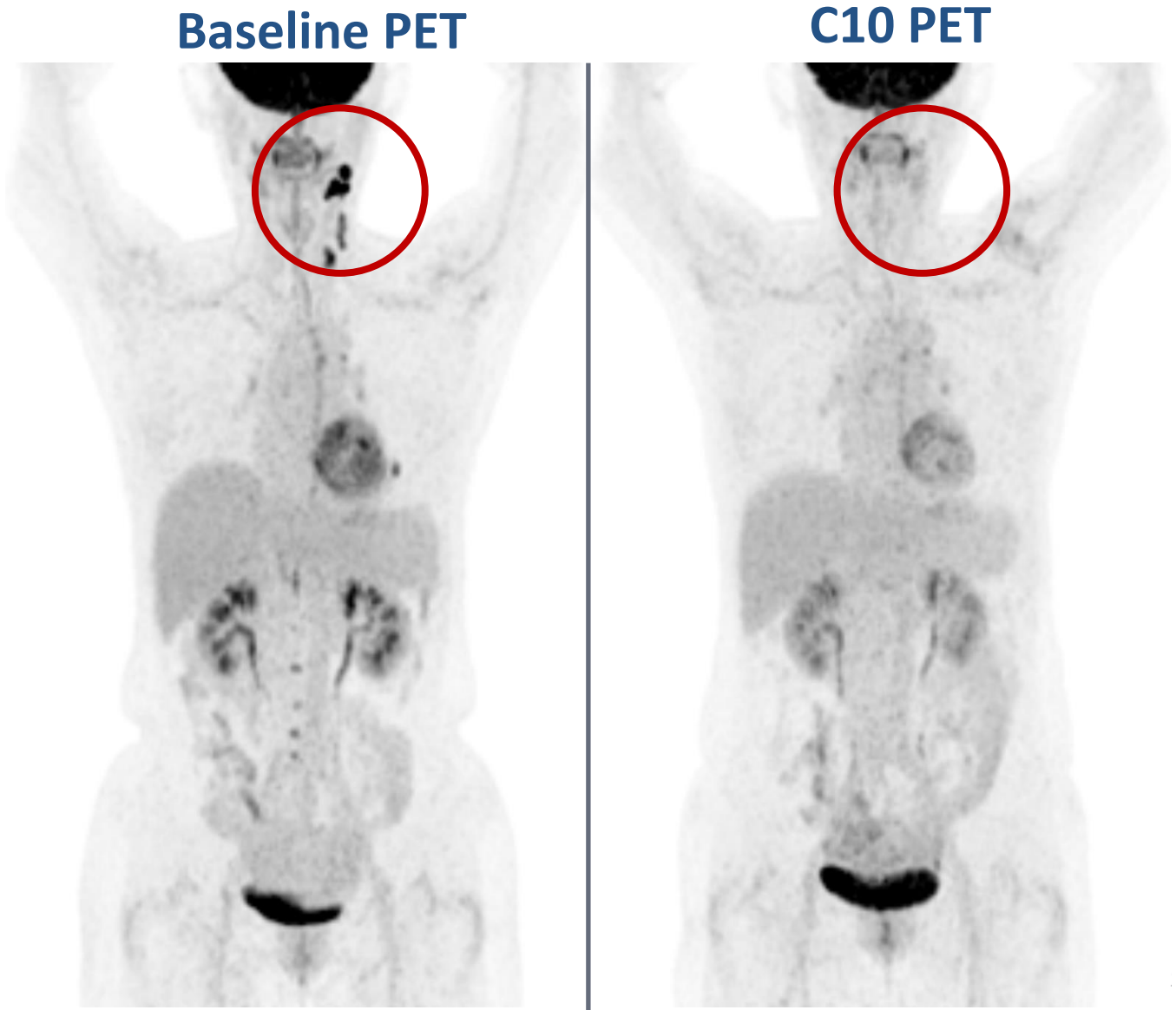
Optimum dose identified



PTCL Patient with Complete Response

Durable response lasting 25 months

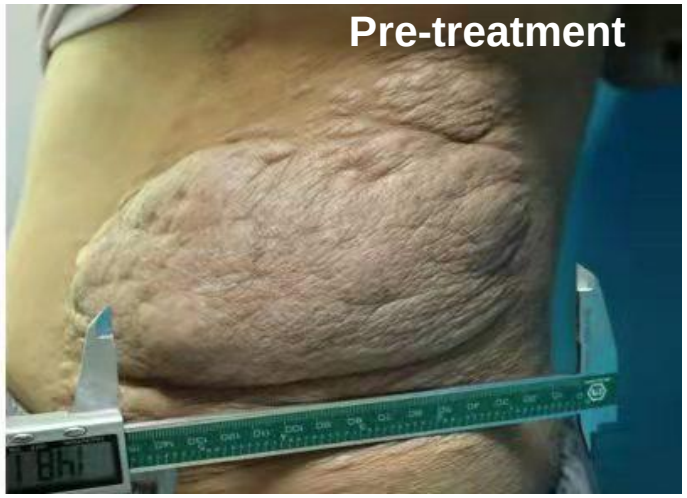
- 57 y/o female with PTCL-NOS
  - Multiple nodes in neck, mediastinum abdomen, pelvis, groin
- CHOP with PR for 5 months
- ASCT for progressive disease
  - Relapse 1 yr
- Started on CPI-818 with disease involving multiple nodal sites
  - CR lasting 25 months

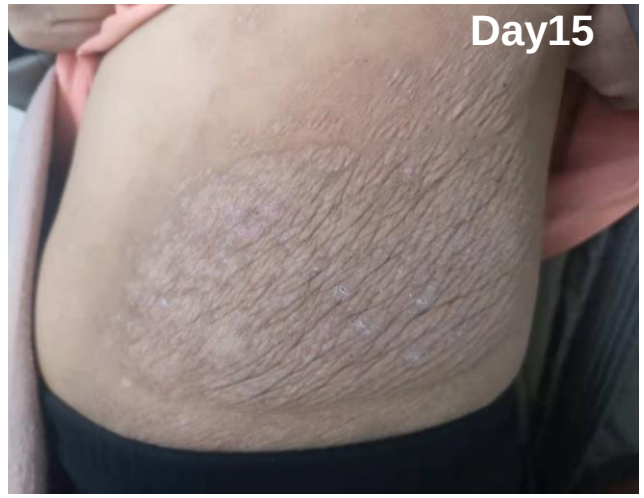


PTCL Patient with Prompt Response

Marked tumor reduction in subcutaneous mass and lymph nodes

- Patient with PTCL NOS
  - CD3-,CD4+, CD20-, TCR clonal, EBV+
- Involvement of LN, skin, blood
- Prior therapies
  - CHOEP x 4, PR;
  - GDP x 2 SD;
  - anti-PD1/HDACi/azacytidine x 4 PD
- CPI-818 monotherapy
  - Dramatic reduction of SQ tumor and improvement in Eos, platelets and LDH
  - Transient lymphocytosis





Lab	Pre-treatment	Day 8	Day 15	Day 21	Day 42	Day 63
White Blood Cells ($x10^9$/L)	27.13	21.92	18.50	16.87	17.87	17.24
Lymphocyte ($x10^9$/L)	6.62	16.17	13.52	13.11	10.22	10.57
Eosinophil count ($x10^9$/L)	17.18	1.6	0.93	1.34	4.21	4.42
Platelets ($x10^9$/L)	105	104	141	145	153	159
LDH (IU/L)	651	378	299	262	286	253

PTCL Patient with Prompt Response

Th1 and T effector cells increase on treatment

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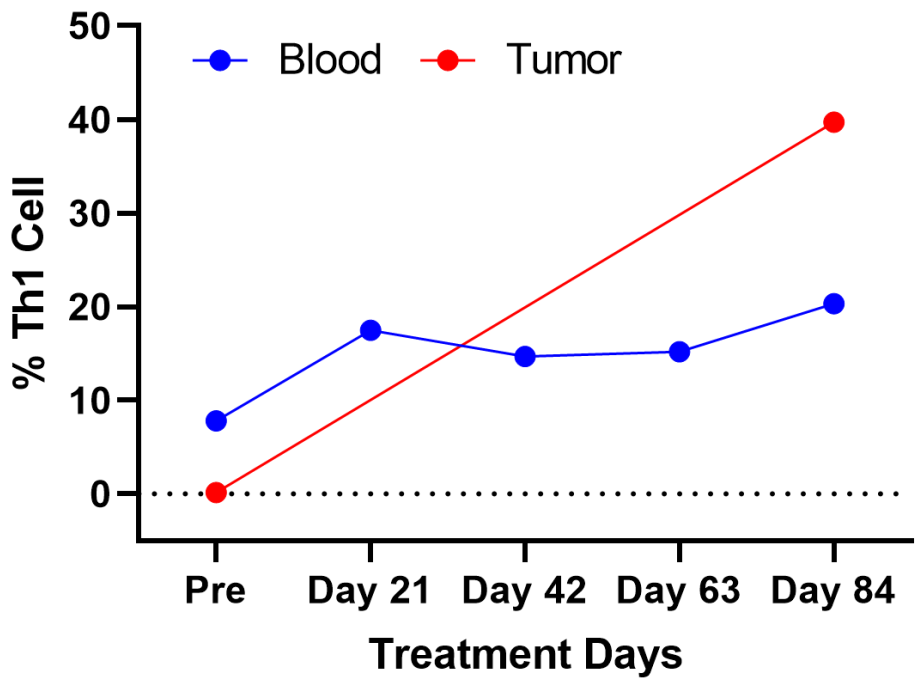
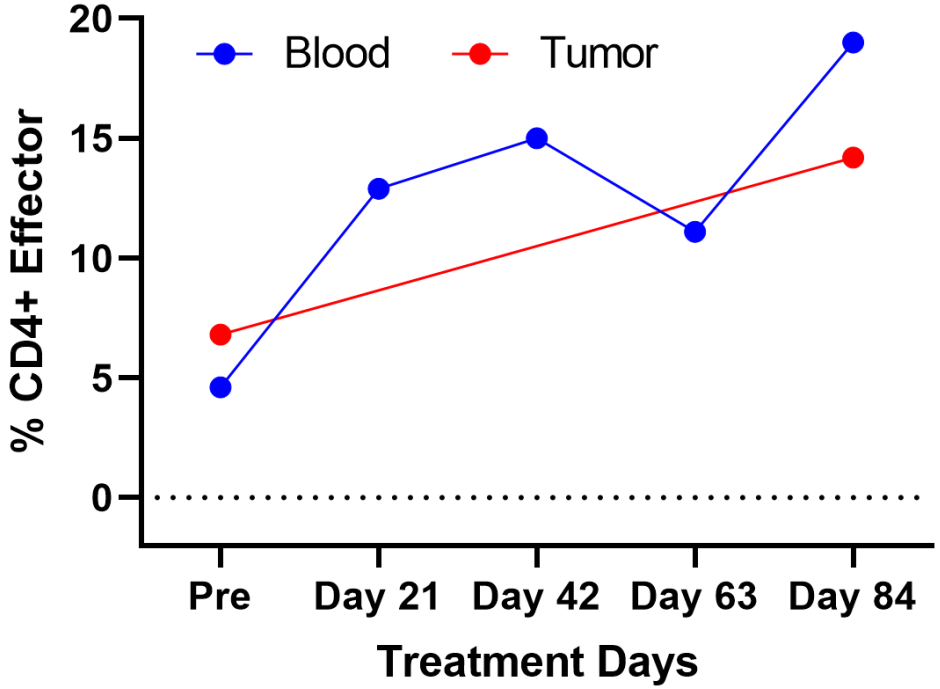
Th1 cells increase in blood and tumor during CPI-818 treatment



CD4+ effector cells increase in blood and tumor during CPI-818 treatment



PTCL Patient with Prompt Response

Activated T cells increase on treatment

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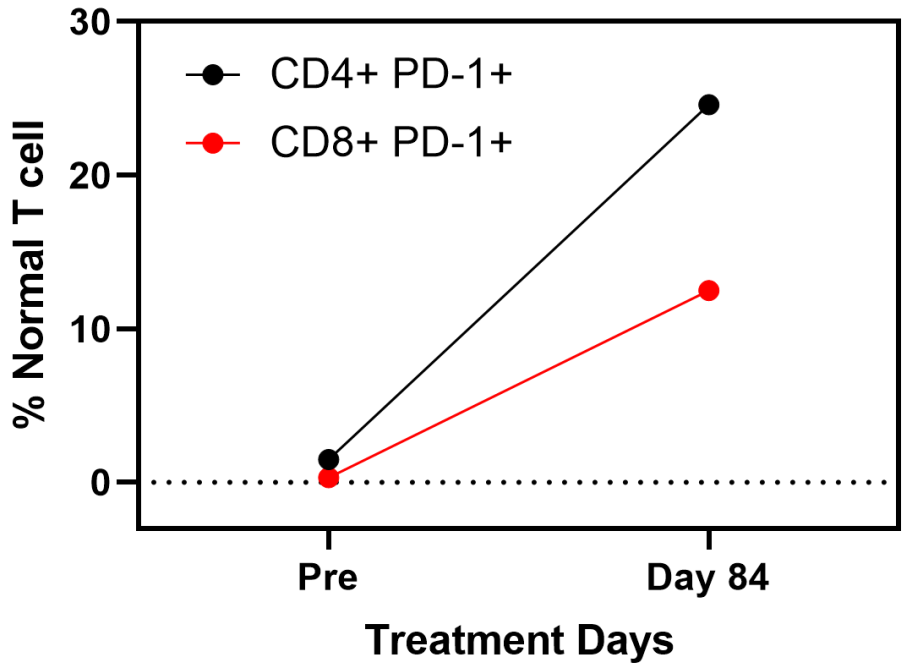
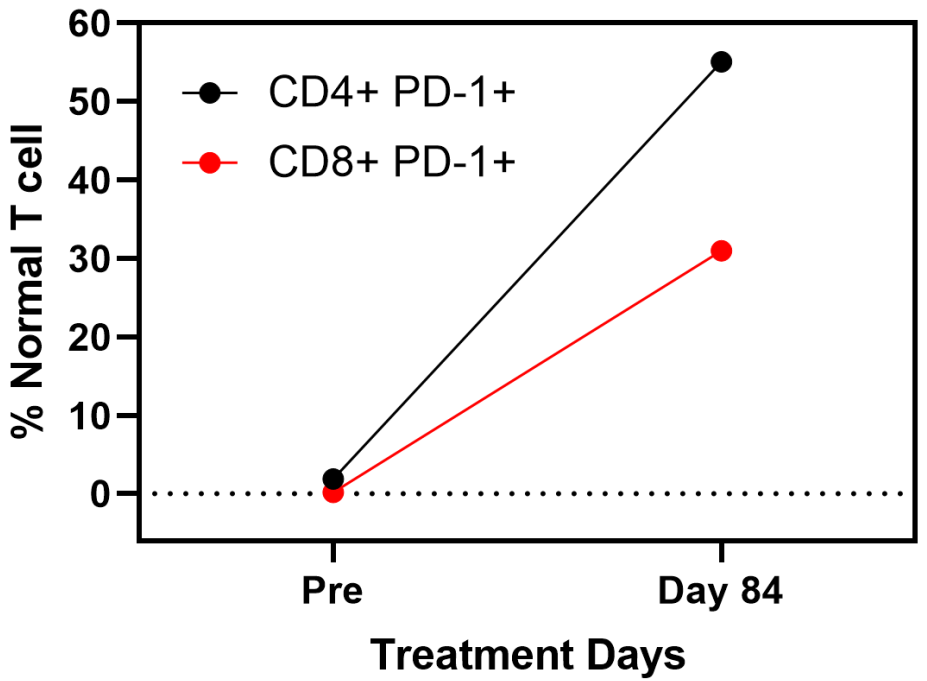
CD4+PD-1+ and CD8+PD-1+ normal T cells increase in *blood* during CPI-818 treatment



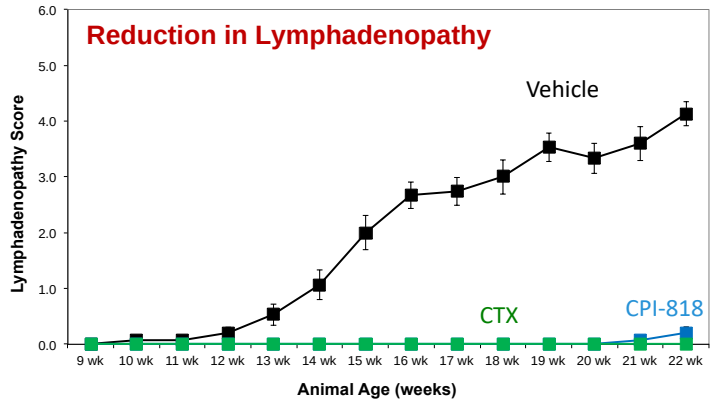
CD4+PD-1+ and CD8+PD-1+ normal T cells increase in *tumor* during CPI-818 treatment

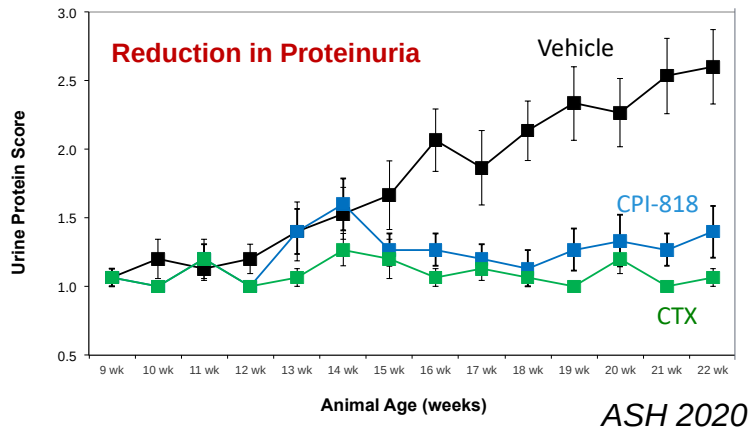


CPI-818 Activity in Autoimmunity

Lupus, Psoriasis and GVHD model

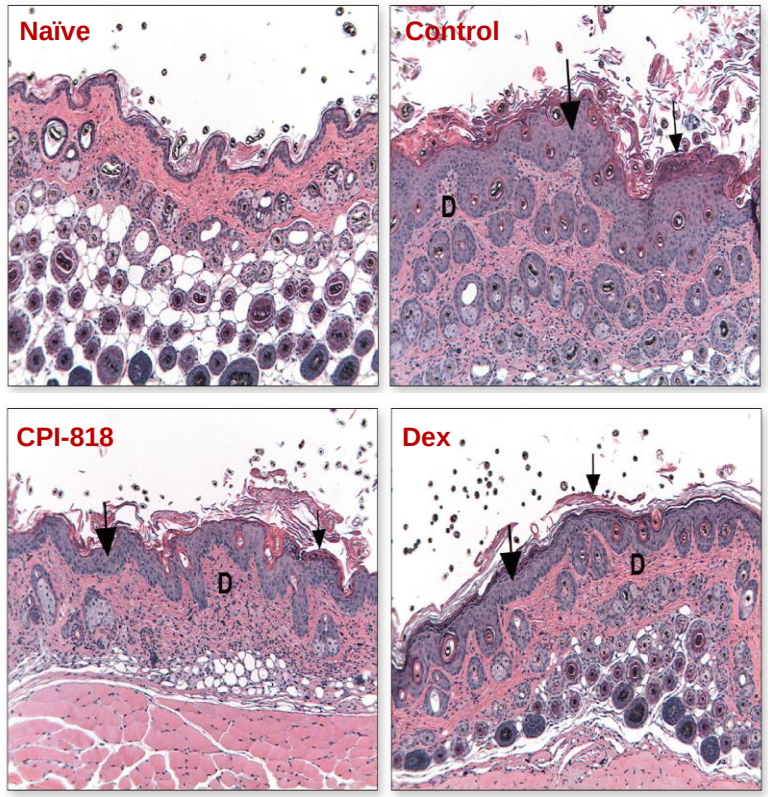
CPI-818 inhibits proteinuria and lymphadenopathy in MRL/lpr$^{-/-}$ Lupus Model



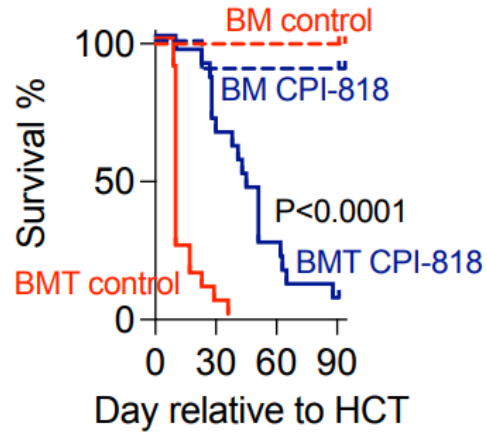


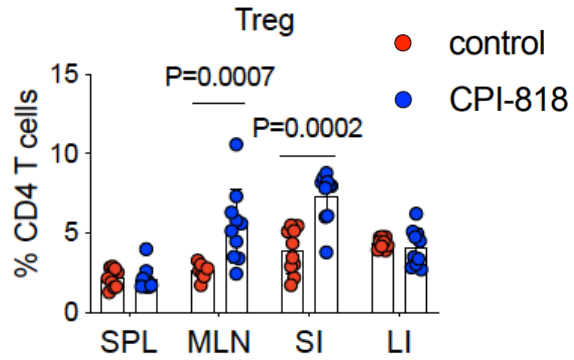
ASH 2020

CPI-818 significantly reduced skin thickening and dermal inflammation in imiquimod-induced psoriasis



CPI-818 reduces GVHD, improves survival and increases Treg





ASH 2021

CPI-818 Summary

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Modulate Tumor Immunity

Induces Th1 skewing

Increases effector cells in the tumor

Evidence of T cell activation in the tumor

Precision Molecular Targets

Oral, selective, covalent inhibitor

Optimal dose identified

Well-tolerated

Broad Clinical Applications

Activity seen in PTCL, CTCL and AITL

Preclinical activity in autoimmunity model

Next Steps

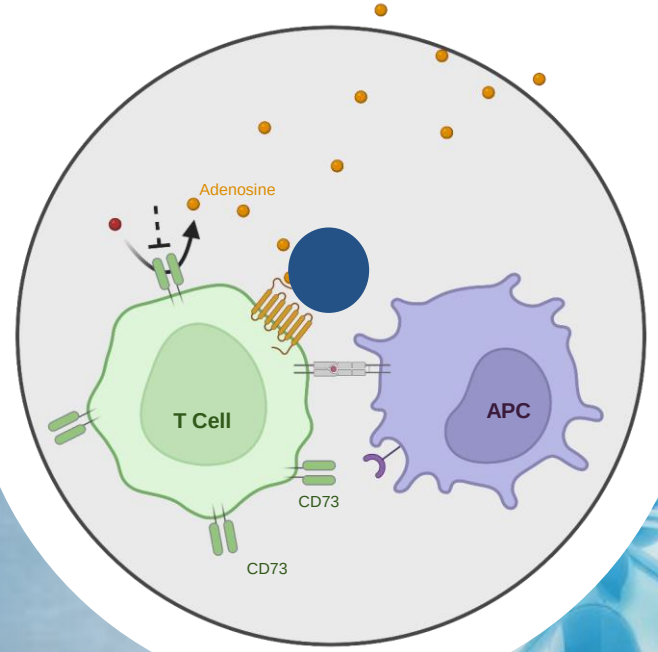
Angel enrolling in China

Enrolling at optimal dose; data expected 2H 2022

Autoimmune Ph 1 trial

Ciforadenant

Adenosine Receptor Inhibition



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Cancer Discovery January 2020

Publication of clinical results in RCC

Published OnlineFirst November 15, 2019; DOI: 10.1158/2159-8290.CD-19-0980

RESEARCH ARTICLE

Adenosine 2A Receptor Blockade as an Immunotherapy for Treatment-Refractory Re

Lawr
Saby
Shiva
Daru
Philip
Brian
Richa

VIEWS

IN THE SPOTLIGHT

Lessons from the A2A Adenosine Receptor Antagonist–Enabled Tumor Regression and Survival in Patients with Treatment-Refractory Renal Cell Cancer

Michail V. Sitkovsky

Summary: In this issue of *Cancer Discovery*, Fong and colleagues describe the encouraging observations of tumor regression, disease control, and survival of patients with otherwise refractory renal cell cancer with progressive disease after treatment with the conceptually novel oral antagonist of the A2A adenosine receptor (A2AR), ciforadenant. A2AR antagonists may represent the until now missing but critically important part of more effective immunotherapies of cancer, because they prevent the inhibition of tumor-reactive T and natural killer cells by blocking the immunosuppressive hypoxia–A2A-adenosinergic signaling, which represents an emerging immunosuppressive hallmark of tumors that are the most resistant to therapies.

See related article by Fong et al., p. 40 (1).

Currently, the majority of patients with cancer are still eventually refractory to any cancer therapy despite a massive and decades-long effort. The hope for the solution to this acute medical problem may come from taking a different and novel therapeutic path, as did Fong and colleagues (1), who, in an "out-of-the-box" approach, treated patients with refractory renal cell cancer (RCC) with a drug that inactivates the biochemical, hypoxia-A2-adenosinergic, immunosuppressive tumor protection (2–8). This powerful mechanism of tumor protection inhibits the antitumor T and natural killer (NK) cells near and within tumors, thereby making them the most resistant to cancer therapies (3, 4, 7), even after the blockade of immunologic negative regulators (4, 6).

THE A2A ADENOSINE RECEPTOR IS A

misguidedly protects the hypoxic and extracellular adenosine-rich cancerous tissues (3, 4, 7). This is why A2AR blockade with synthetic A2AR antagonists has been proposed for a long time (2, 3) as a therapeutic tool to unleash tumor-reactive T and NK cells to enable immunotherapy-mediated tumor regression (3–7). The synthetic A2AR antagonists can also be termed "super-caffeine," because the research and development of these highly selective for A2AR and long-lived *in vivo* drugs was in part prompted by observations of favorable effects of caffeine consumption in patients with Parkinson disease.

Originally, not only the A2AR but also the low-affinity A2B adenosine receptor (A2BR) were considered to be targets to antagonize to improve immunotherapies of cancer (3). However, the subsequent biochemical considerations of the differences between the Gs-coupled A2AR and Gs/Gq-coupled A2BR, as well as the more detailed preclinical tumor

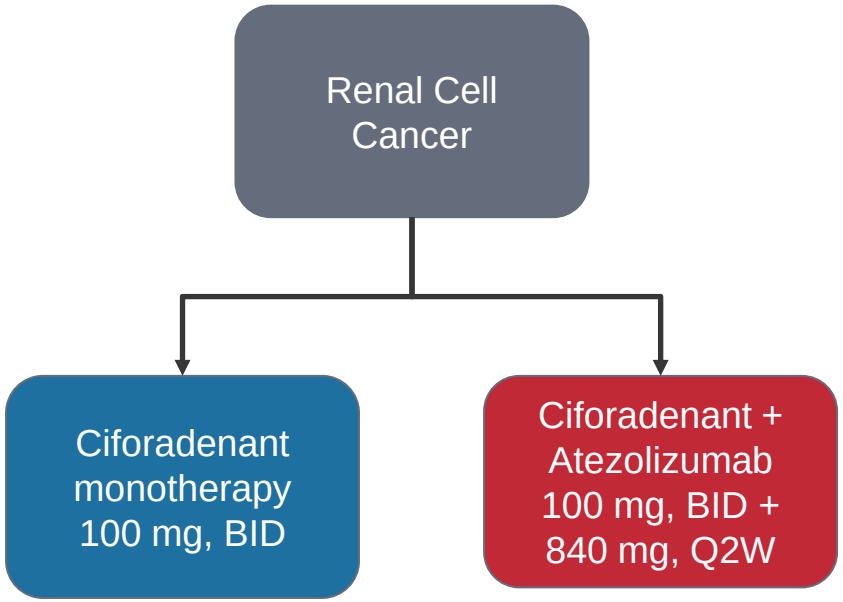
"Fong and colleagues describe… tumor regression, disease control, and survival of patients with otherwise refractory renal cell cancer with progressive disease after treatment with the conceptually novel…. ciforadenant."

"Fong and colleagues are among the first clinical development teams that aimed to block not only the immunologic negative regulators, but also the powerful A2A-adenosinergic negative regulators of antitumor immunity."

Renal Cell Cancer Clinical Results

Patient characteristics

- 68 patients with RCC enrolled
- Median on-treatment time was 5 (1-21.7) months

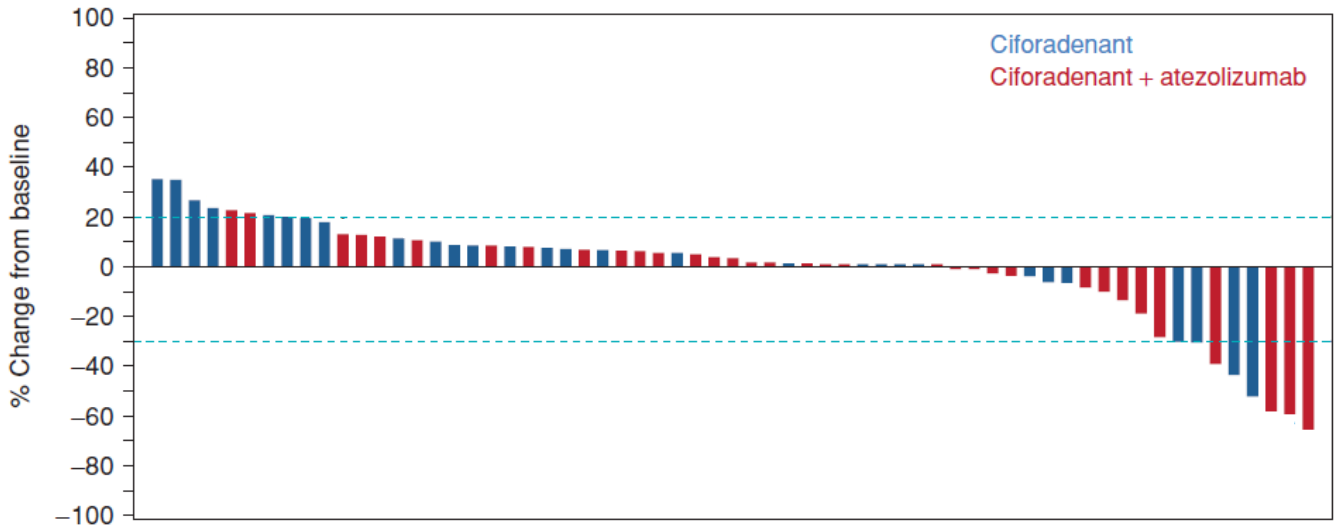


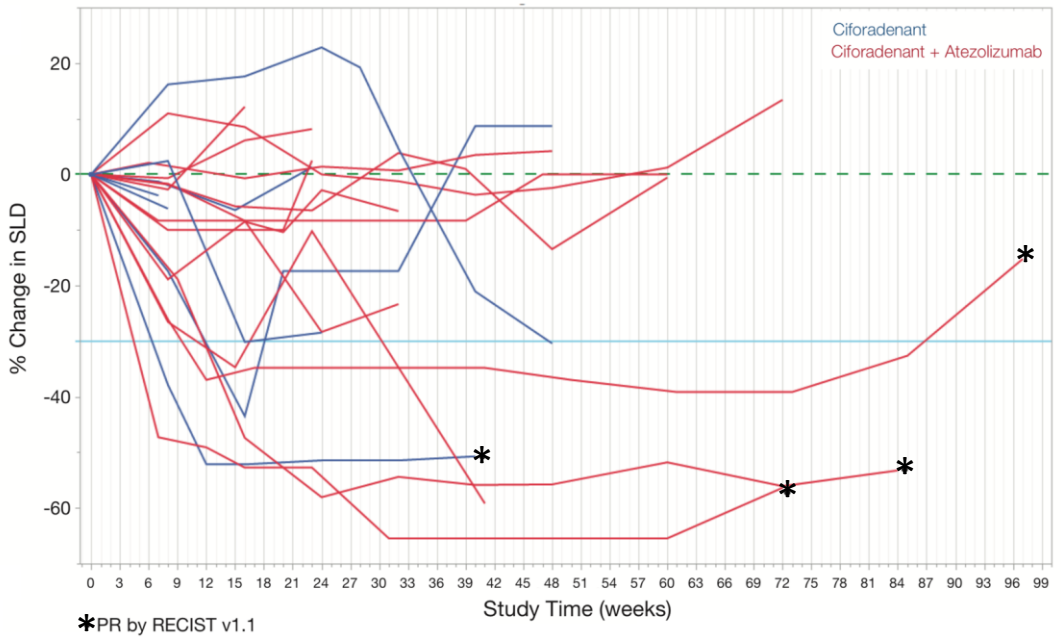
Characteristic	Ciforadenant (n=33)	Ciforadenant + Atezolizumab (n=35)
Median Age (range), years	60 (47, 76)	65 (44, 77)
Gender, male, n (%)	25 (75.8)	28 (80)
No. of prior therapies, median (range)	3 (1, 5)	3 (1,5)
Prior IO, number of subject, n (%)	24 (72.7)	25 (71.4)
Months since prior IO Median (Range)	3.1 (1,2, 70.4)	1.7 (0.9, 23.6)
PD-L1 Negative, n(%)*	25/27 (92.6)	28/31 (90.3)
Prior PD-1 therapy, n (%)	23 (69.7)	25 (71.4)

* PD-L1 status determined using FDA-approved assay (SP142, cutoff = 5%)

Fong et al, Cancer Discovery, 2020

Renal Cell Cancer Response to Treatment



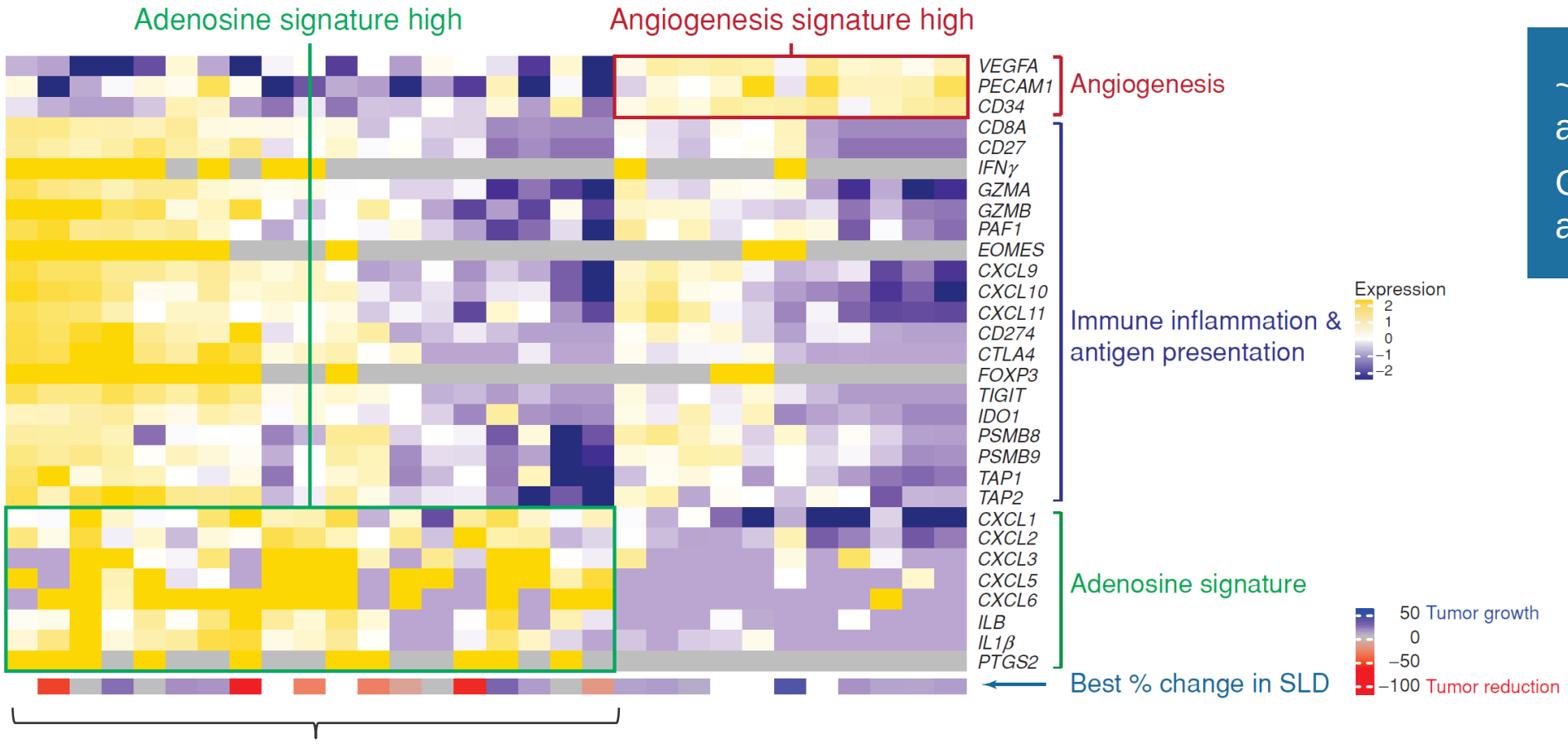


	Ciforadenant (n=29)	Ciforadenant + Atezolizumab (n=33)
6-month Disease Control rate		
Prior anti-PD-(L)1	25% (5/20)	35% (8/23)
Naïve	0% (0/9)	50% (5/10)
Total	17% (5/29)	39% (13/33)
Median time to best tumor response	3.4 months	5.5 months

Fong et al, Cancer Discovery, 2020

Adenosine Signature Correlates with Anti-Tumor Activity

Potential predictive biomarker



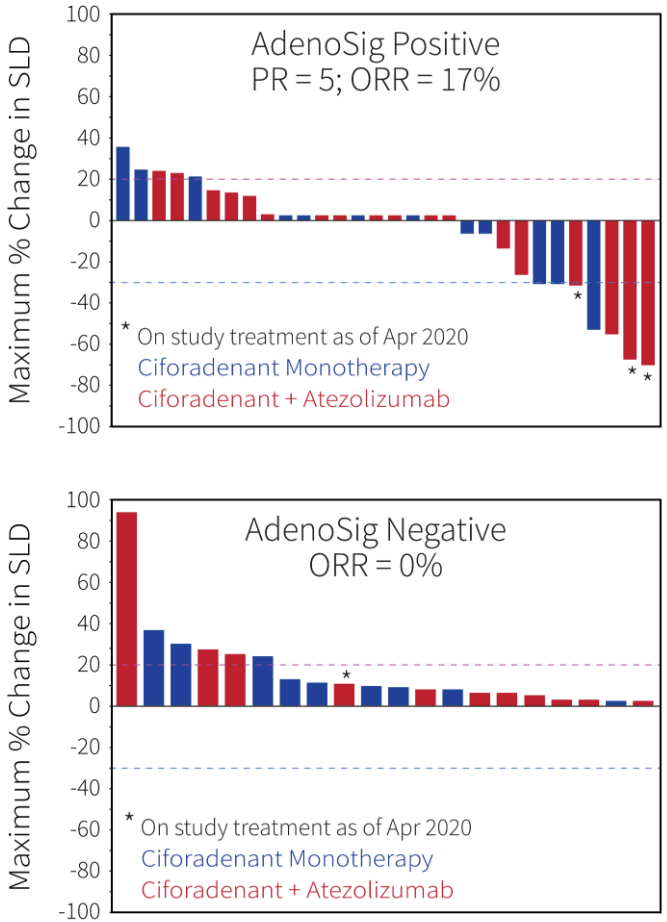
~50 – 60% of RCC pts are positive

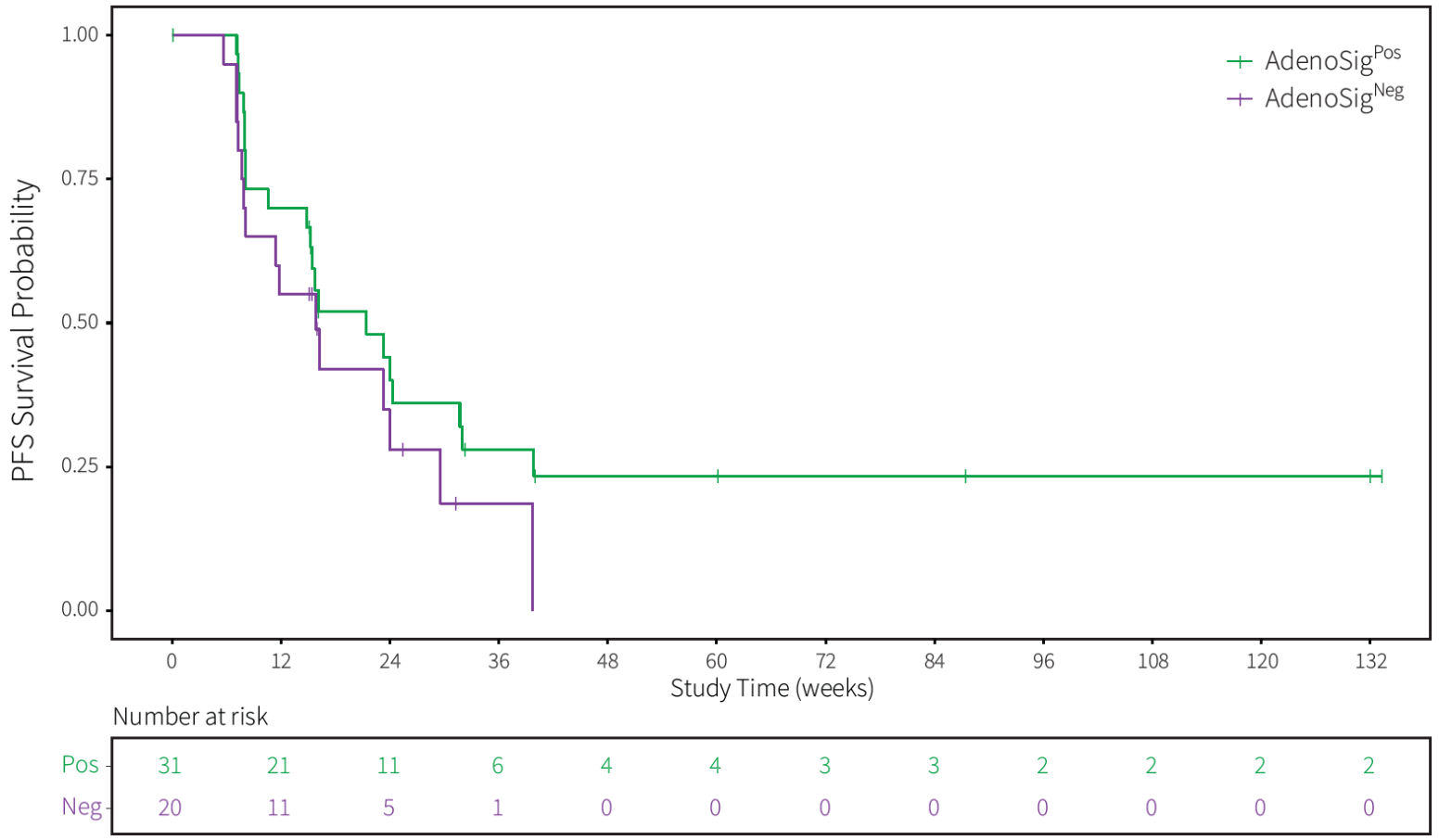
Confirmed by outside academic groups

- **Enriched for ciforadenant response**
- Angio^{Low}: Poor PFS with TKI[1,2]
- Myeloid^{High} : Poor PFS with single agent atezolizumab[1]

1. McDermott et al, Nature Medicine, 2018; 2. Fong et al, Cancer Discovery, 2020

Adenosine Signature Correlates with Anti-Tumor Activity





Fong et al, Cancer Discovery, 2020

Strong Rationale for Frontline Triplet Combination

Supports triplet aimed at increasing durable remissions

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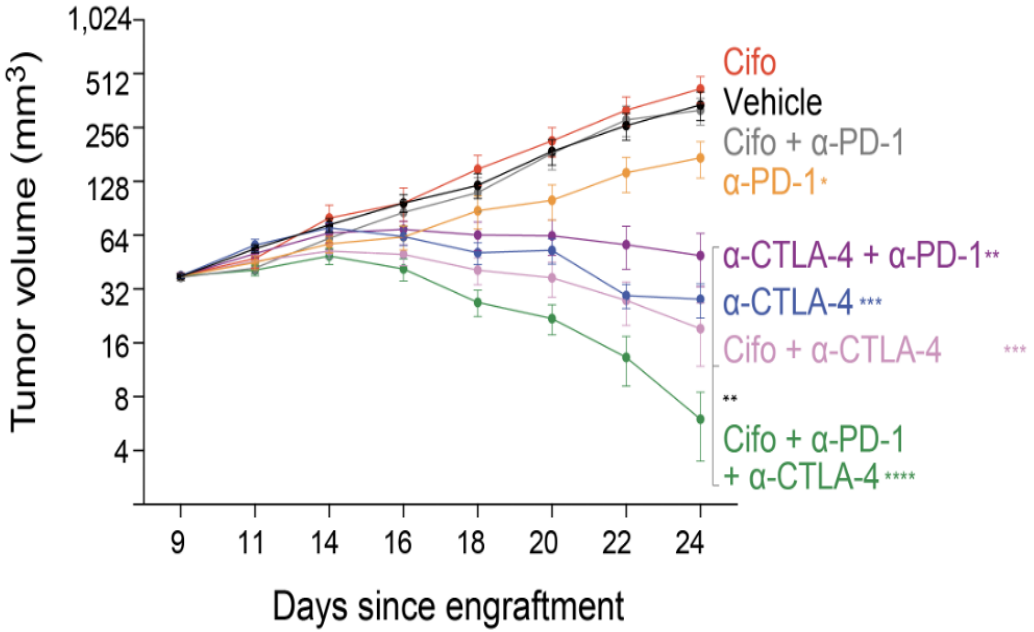
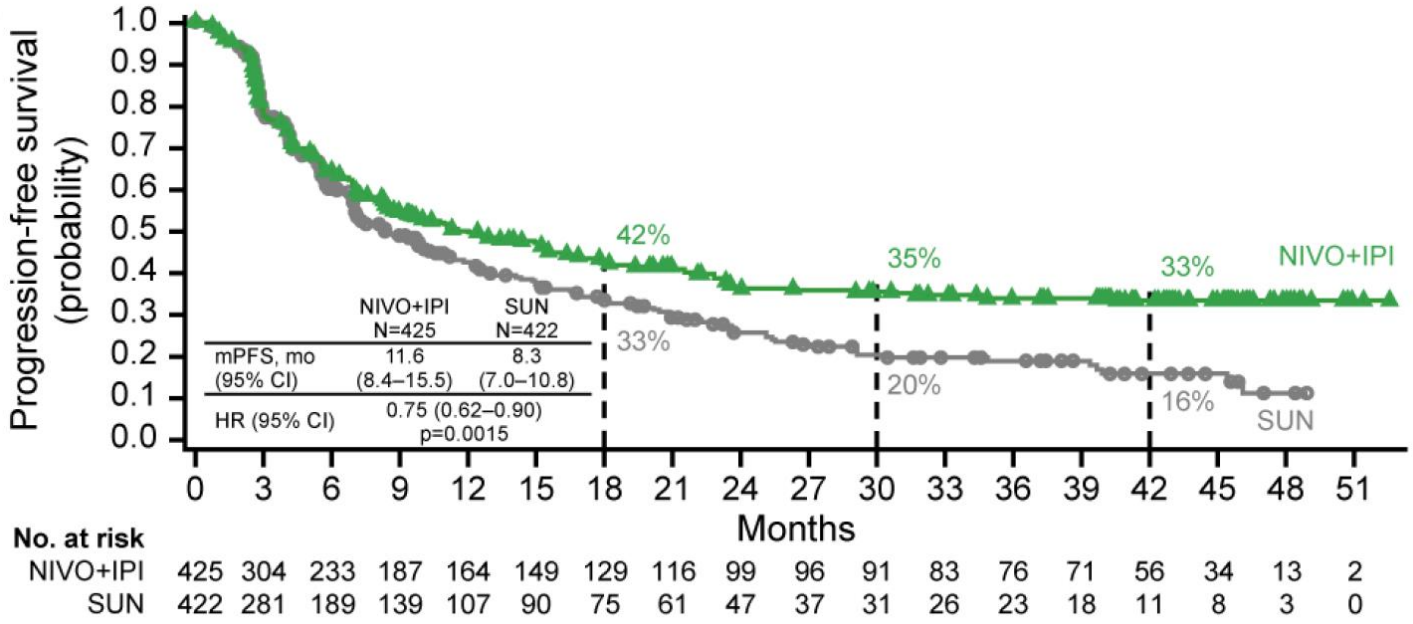
CT26 Preclinical Model Established Tumor
(*Willingham et al, Cancer Imm Res. 2018*)



Triplet Cifo, anti-PD1, anti-CTLA4 cures most animals

CheckMate 214 Trial
(*Motzer et al, J. Immunother. Cancer, 2020*)



4-year follow-up from CheckMate 214 study of IPI/NIVO showing a tail on the curve suggesting potential cures

Phase 1b/2 Trial Design in Frontline RCC

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Eligibility

- Newly diagnosed or recurrent stage IV clear cell RCC
- No prior systemic therapy
- Tumor sample for histologic confirmation & biomarker assessment

N = 8

Phase 1b

Ipilimumab 1 mg/kg
IV q3w x 4
+
Nivolumab 3 mg/kg
IV q3w
+
Ciforadenant 100 mg
PO BID

- Primary endpoint: Safety, tolerability and anti-tumor

N = 51

(Minmax two stage <7/28 stop for futility)

Phase 2

Ipilimumab 1 mg/kg
IV q3w x 4
+
Nivolumab 3 mg/kg
IV q3w
+
Ciforadenant 100 mg
PO BID

- Primary endpoint: percentage who achieve depth of response of >50% tumor reduction from historical control of 34% to 50%
- Secondary endpoint: ORR, PFS, irAE
- Exploratory: gene expression

VANDERBILT UNIVERSITY MEDICAL CENTER

Kidney Cancer RESEARCH CONSORTIUM

THE UNIVERSITY OF TEXAS MD Anderson Cancer Center

Ciforadenant Summary

Modulate Tumor Immunity

Enhances T cell infiltration in tumor

New T cell clones detected in blood

Augments efficacy to anti-PD-(L)1 / CTLA-4

Precision Molecular Targets

Oral, selective

Block A2AR signaling

Treatment response correlates with adenosine signature

Broad Clinical Applications

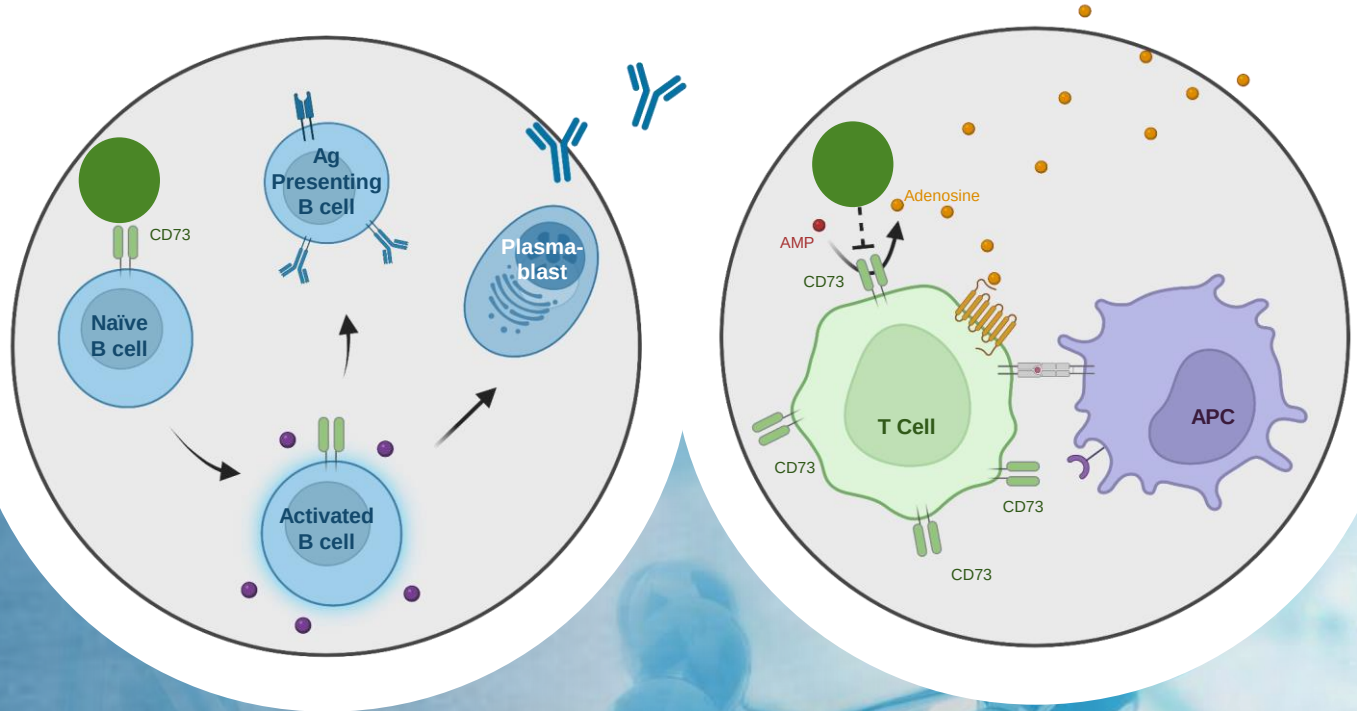
Well tolerated and shows activity in mono and combination therapy of advanced cancer

Next Steps

Kidney Cancer Consortium to conduct Phase 2 trial in frontline RCC patients with a triplet in Q3 2022

Mupadolimab

B cell Activation
And Adenosine Blockade



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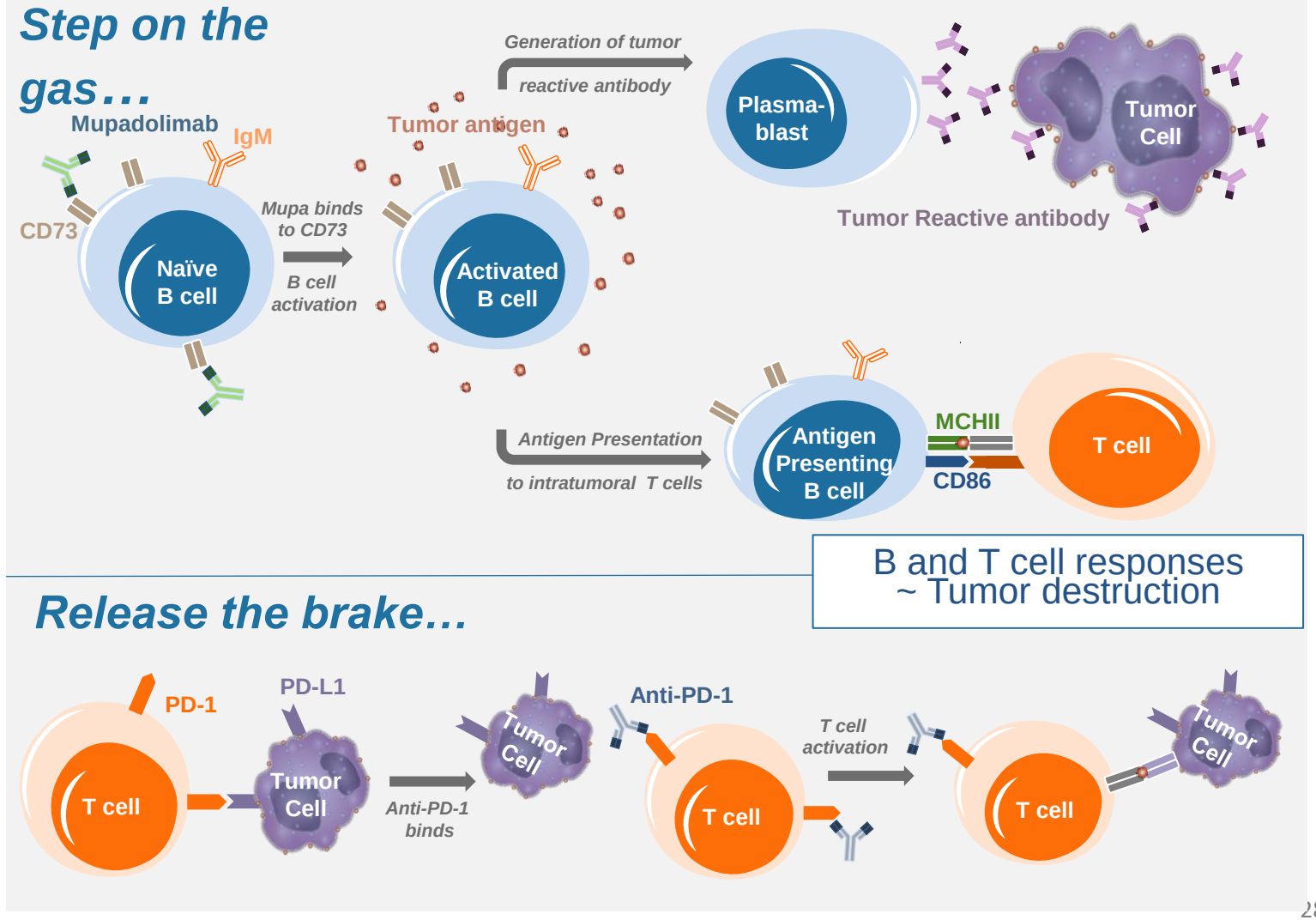
Mupadolimab Background and Strategy

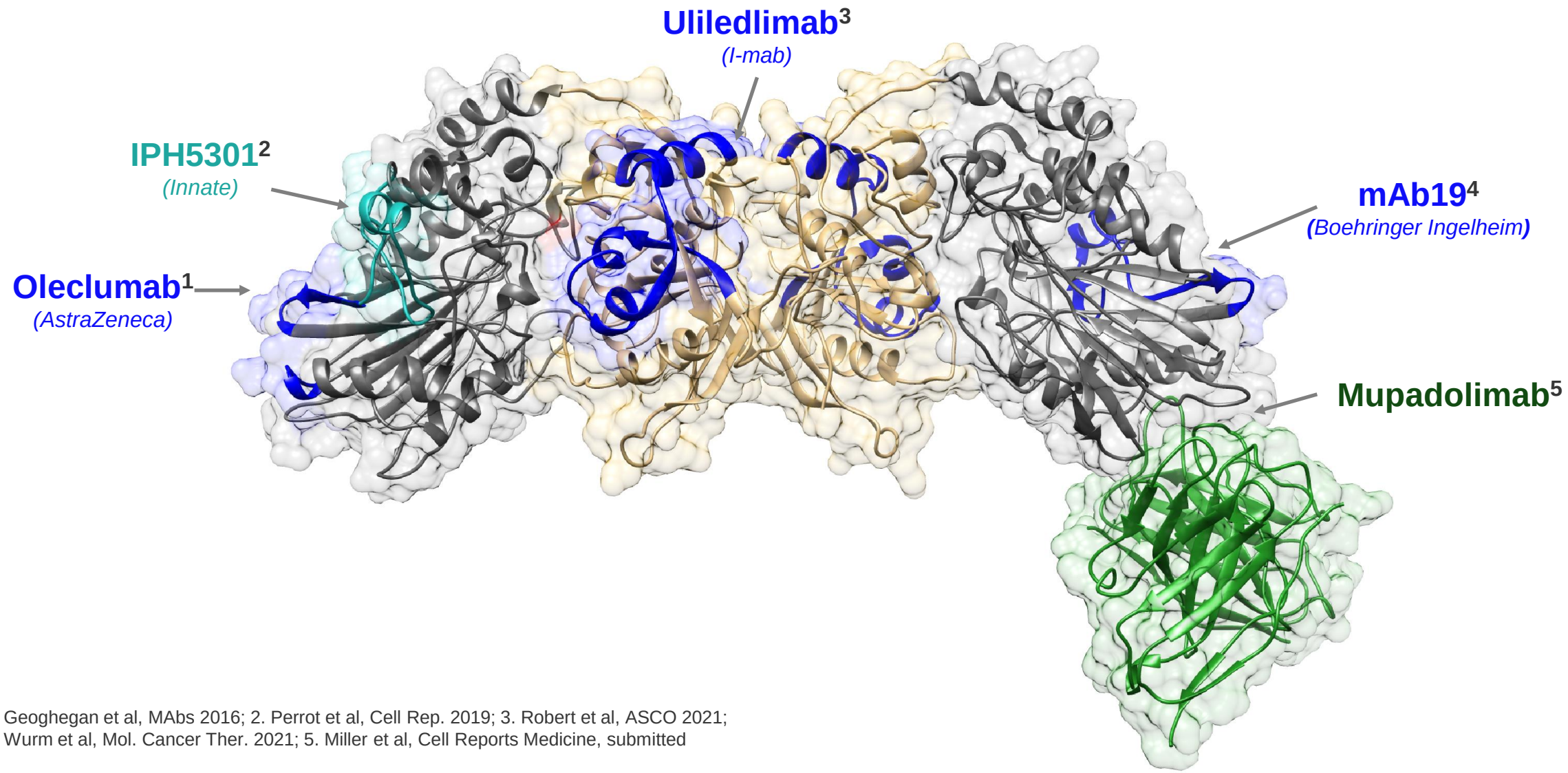
- CD73 is an ectoenzyme present on many tissues including subsets of T (CD4 10%, CD8 50%) and B cells (70%)
  - Catalyzes conversion of AMP into immunosuppressive adenosine
  - Functions in lymphocyte adhesion, migration and activation
- Mupadolimab is a humanized IgG1 Fcγ receptor binding deficient anti-CD73 with unique properties
  - Blocks CD73's catalytic activity
  - Agonistic immunomodulatory activity on CD73 positive B cells and T cells

Targeting B Cells and T Cells: Mupa, anti-PD-1 Combo



Unique Binding Epitope Confirmed by Cryo-EM

Comparison with other CD73 antibodies



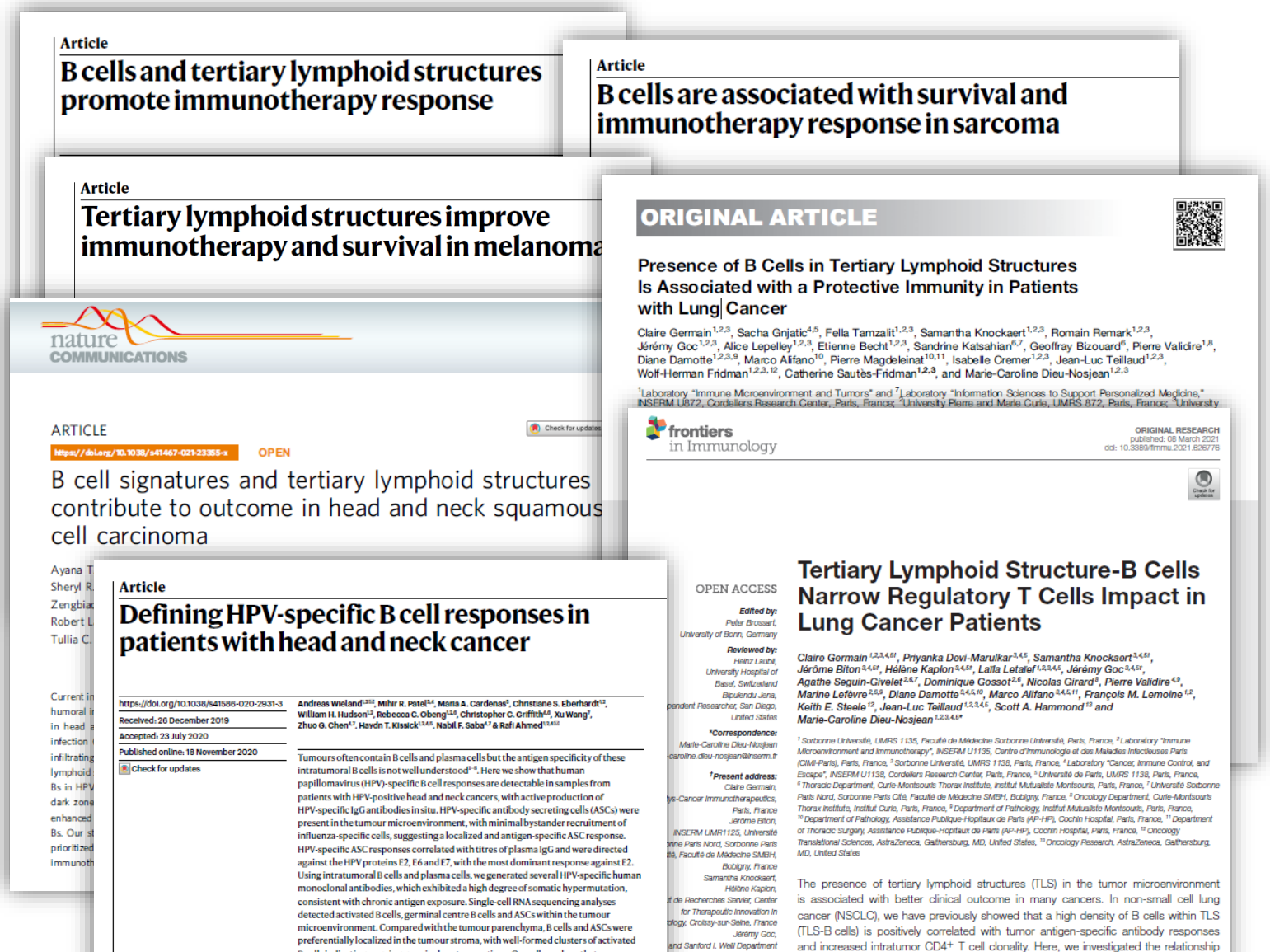
1. Geoghegan et al, MAbs 2016; 2. Perrot et al, Cell Rep. 2019; 3. Robert et al, ASCO 2021;
4. Wurm et al, Mol. Cancer Ther. 2021; 5. Miller et al, Cell Reports Medicine, submitted

Comparison Between Mupadolimab and Oleclumab

Parameters	Mupadolimab	Oleclumab
Isotype	human IgG1κ	human IgG1λ
Fc engineering	Deficient FcγR-binding	Deficient FcγR-binding
Affinity (K_D)[1]	~100-200 picomolar	~100-200 picomolar
Internalization	No	Yes
Hook Effect	No, fully blocking adenosine	Yes, partially blocking adenosine
B cell activation	Strong	Weak
T cell restoration	Effective	Less Effective
Stage of Development	Phase 2	Phase 3
RP2D	1200 mg Q3W	3000 mg Q2W (first 2 cycles, then Q4W)

1. Binding of CD73 antibody to recombinant human CD73-His was measured by Octet

B cells - Important Predictors of IO Response and Prognosis



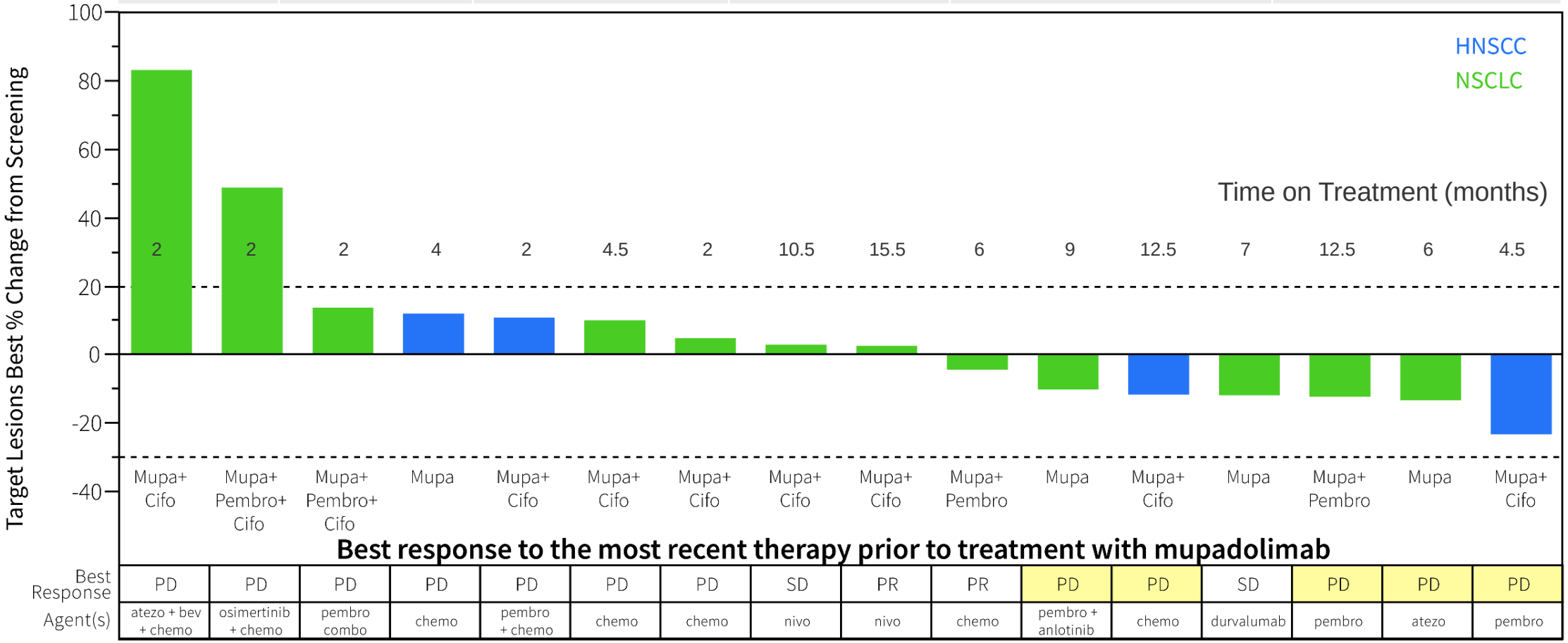
- B cells are found in tumors of responders[1,2,3]
- The B lineage signature in tumors was the dominant parameter for overall survival[2]
- Activated B cells and antibody secreting cells specific for tumor-specific antigens found in the tumor microenvironment in HPV$^+$ head and neck patient samples[4,5]
- High density B cells within tertiary lymphoid structure promote CD4+ T cell response and are associated with superior clinical outcomes in NSCLC patients[6,7]

1. Helmink et al, Nature, 2020; 2. Petitprez et al, Nature 2020; 3. Cabrita et al, Nature 2020; 4. Weiland et al, Nature 2020; 5. Ruffin et al, Nat. Commun. 2021; 6. Germain et al, Am. J. Respir. Crit. Care. Med. 2014; 7. Germain et al, Front Immunol. 2021

Anti-tumor Activity in HNSCC and NSCLC with ≥12 mg/kg

Tumor regression seen in pts with PD as best response to prior Rx

	Subject Counts	Age (yrs) Median (range)	Gender, male N (%)	No. of Prior Therapy Median (range)	Prior PD-(L)1 Therapy N (%)
HNSCC	10	65 (43, 87)	9 (90)	3 (1,5)	10 (100)
NSCLC	15	64 (53, 80)	6 (40)	3 (2,4)	14 (93)



- Cifo = ciforadenent (A2AR antagonist), pembro = pembrolizumab (anti-PD-1), atezo = atezolizumab (anti-PD-L1), bev = bevacizumab (anti-VEGF), chemo = chemotherapy, nivo = nivolumab (anti-PD-1)
- PD = progressive disease; SD = stable disease; PR = partial response

Mupadolimab Summary

Modulate Tumor Immunity

Evidence of B cell activation

B cell redistribution to lymphoid tissues

Evidence of anti-tumor antibodies

Precision Molecular Targets

CD73 novel epitope defined by Cryo-EM

Complete adenosine blockade

Favorable safety in mono and combo therapy

Broad Clinical Applications

Anti-tumor activity seen in advanced cancers

Potential application in infectious disease

Next Steps

Randomized, placebo-controlled Phase 2 trial in frontline NSCLC, (paused)

Angel Phase 1 trial in NSCLC and HNSCC

Efficient Development Strategy

Cash runway into early 2024

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3 Clinical programs with significant anticipated near-term milestones

- CPI-818 Phase 1/1b data in T-cell lymphoma in 2H 2022
- CP-818 Phase 1 trial in Autoimmunity in 1H 2023
- Ciforadenant interim Phase 2 data in front-line RCC in 1H 2023

Unique pipeline focused on the tumor immunity axis

- Precisely defined targets
- Novel ITK inhibitor control T cell differentiation
- Selective A2AR inhibitor augments efficacy to anti-PD-1 and anti-CTLA-4
- First anti-CD73 to demonstrate B cell modulation

Robust pre-clinical and clinical data

- First to show clinical activity of ITK inhibitor in lymphomas and immune diseases
- Experience in a large number of cancer patients with ciforadenant or mupadolimab
- Pioneer in adenosine pathway and kinase inhibitor R&D
- Identified predictive Adenosine Gene Signature biomarker in RCC