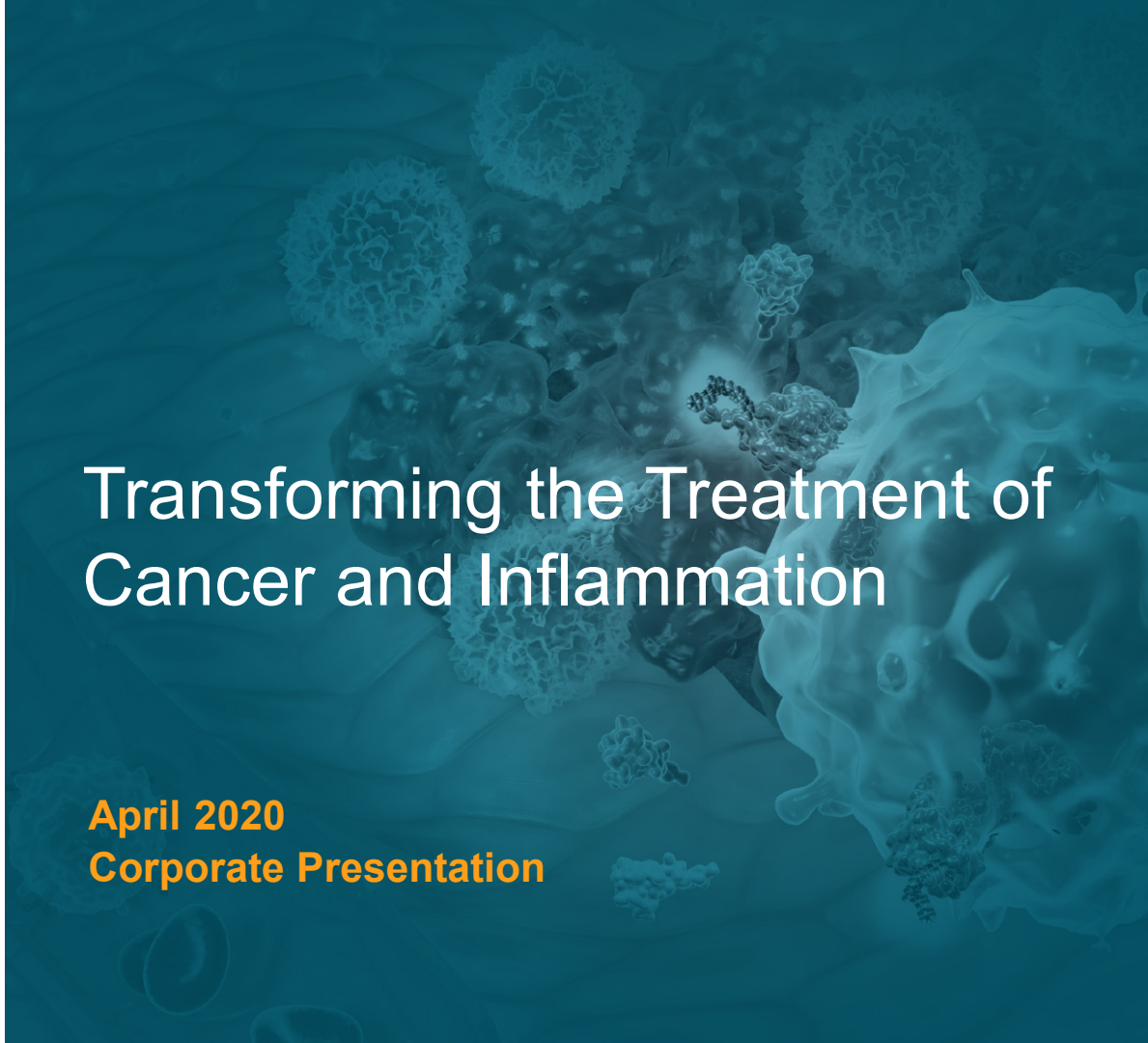
RAPT THERAPEUTICS

Transforming the Treatment of Cancer and Inflammation



April 2020
Corporate Presentation

Legal Disclaimers

Statements in this Presentation that are not statements of historical fact are forward-looking statements. Such forward-looking statements include, without limitation, statements regarding RAPT Therapeutics, Inc.'s (the "Company," "we," or "us") research and clinical development plans; current and future drug candidates; business strategy and plans; regulatory pathways; and our ability to complete certain milestones. Words such as "believe," "anticipate," "plan," "expect," "will," "may," "upcoming," "milestone," "potential," "target" or the negative of these terms or similar expressions are intended to identify forward-looking statements, though not all forward-looking statements necessarily contain these identifying words. These forward-looking statements are based on the current beliefs of the Company's management with respect to future events and trends and are subject to known and unknown risks and uncertainties, including those described in the "Risk Factors" section of our Form 10-K filed with the Securities and Exchange Commission on March 30, 2020, that may cause our actual performance or achievements to be materially different from any future performance or achievements expressed or implied by the forward-looking statements in this Presentation. These forward-looking statements should not be taken as forecasts or promises nor should they be taken as implying any indication, assurance or guarantee that any assumptions on which such forward-looking statements have been made are correct or exhaustive or, in the case of such assumptions, fully stated in the Presentation. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date this Presentation is given. Although we believe that the beliefs and assumptions reflected in the forward-looking statements are reasonable, we cannot guarantee future performance or achievements. Except as required by law, we undertake no obligation to update publicly any forward-looking statements for any reason after the date of this Presentation.

This Presentation discusses drug candidates that are under clinical study and which have not yet been approved for marketing by the U.S. Food and Drug Administration. No representation is made as to the safety or effectiveness of any drug candidates for any use for which such drug candidates are being studied.

Focused on Oral Drugs Targeting Critical Immune Drivers of Disease

- **Diversified pipeline**
- **Large market opportunities**
- **Strategic collaborations**
- **Proprietary discovery engine**

CLINICAL

FLX475 (Oncology):

- Selectively targets immunosuppressive tumor T_{reg}
- Encouraging clinical activity in Phase 1 study
- **Phase 2 PoC study ongoing**

RPT193 (Allergic Disease):

- Oral agent targets inflammatory Th2 cells
- Robust PK/PD with excellent safety in Ph1 study
- **Phase 1b PoC in atopic dermatitis ongoing**

DISCOVERY

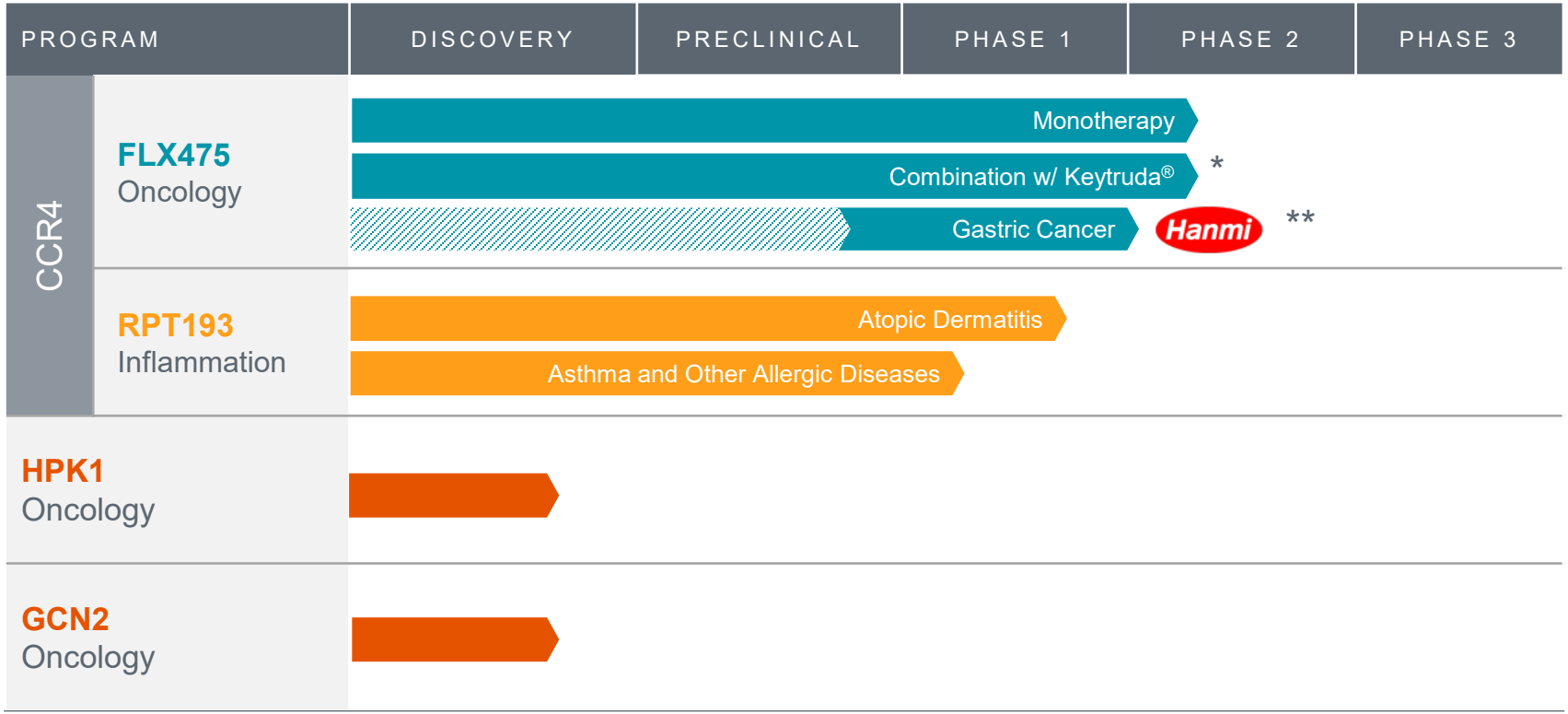
HPK1 (Oncology):

- Unlocks T cell activation to tumor antigens

GCN2 (Oncology):

- Turns on an antitumor metabolic switch in TME

RAPT Therapeutics Diversified Pipeline



* Clinical collaboration with Merck

** Regional collaboration and license with Hanmi in Korea, Taiwan and China (including Hong Kong and Macau)

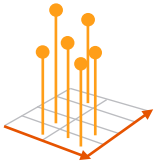
Proprietary Drug Discovery and Development Engine

R **Rapid**

- Drug discovery
- Clinical development to POC

A **Analytics**

- Interrogating clinically-relevant big datasets to identify targets and biomarkers



P **Patient selection**

- Driven by data to improve chances of clinical success

T **Targeting**

- Critical immune drivers of cancer and inflammation

RAPT THERAPEUTICS

Experienced Leadership Team and Scientific Advisory Board

Leadership

Brian Wong, MD, PhD
Chief Executive Officer

Dirk Brockstedt, PhD
Chief Scientific Officer

William Ho, MD, PhD
Chief Medical Officer

Paul Kassner, PhD
Senior Vice President, Quantitative and Computational Biology

Karen Lam
Vice President, Finance and Controller

Lisa Moore, PhD
Vice President, Business Development and Strategy

David Wustrow, PhD
Senior Vice President, Drug Discovery and Preclinical Development

Sylvia Wheeler
Wheelhouse Life Sciences Advisors, Investor Relations

Rodney Young
Chief Financial Officer

Board of Directors

David V. Goeddel, PhD
Managing Partner, The Column Group

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President, Gray Strategic Advisors, LLC

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President and CEO, Blade Therapeutics Inc.

Brian Wong, MD, PhD
CEO, RAPT Therapeutics

Scientific and Clinical Advisors

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Chairman, RAPT Scientific Advisory Board, Chairman, Immunology Program, Sloan-Kettering Institute

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Instructor in the Department of Medicine, Duke University School of Medicine

Drew Pardoll, MD, PhD
Professor, Johns Hopkins University

Philip Greenberg, MD
Professor, Medicine (Oncology) & Immunology, University of Washington

Robert Zamboni, PhD
Adjunct Professor of Chemistry, McGill University

David V. Goeddel, PhD
Founder & CEO Tularik; Founder & Partner The Column Group

Allergy / Immunology

Emma Guttman-Yassky, MD, PhD
Professor and Vice Chair for Research at the Department of Dermatology, Director of the Center for Excellence in Eczema, and Director of the Laboratory of Inflammatory Skin Diseases at the Icahn School of Medicine at Mount Sinai Medical Center

Jasmina Jankicevic, MD
Consulting Dermatologist, Premier Research

Thomas Bieber, MD
Professor of Dermatology and Allergy, University of Bonn, Germany

Andrew Blauvelt, MD, MBA
Dermatologist and President of Oregon Medical Research Center

Summary Financial Information

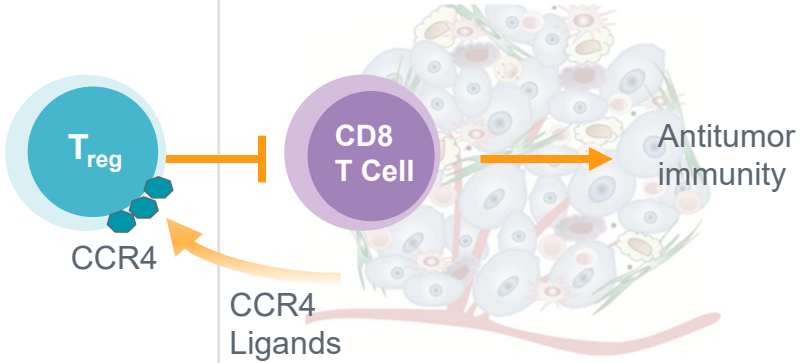
- *Pro forma* cash (at 12/31/19): $147.1M
- 2019 net loss: $43.0M

- Shares outstanding: 24.3M
- Options/RSUs outstanding: 1.6M
- FD shares outstanding: 25.9M

Our CCR4 Program

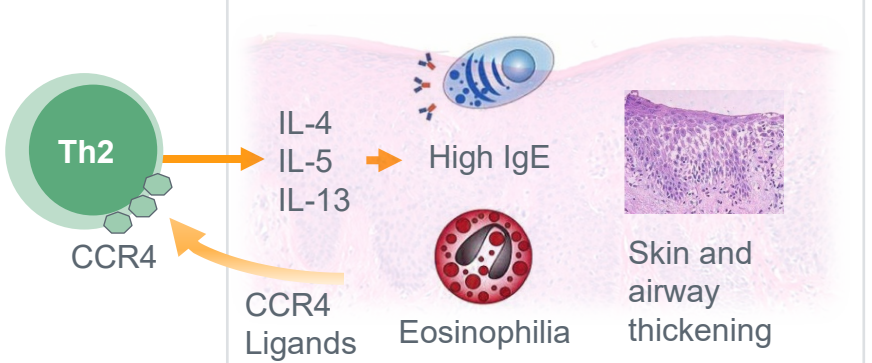
CCR4 Drives Tumor Progression and Allergic Inflammation

Cancer



Regulatory T cells (T_{reg}) are recruited into the tumor via CCR4 and **block** antitumor immunity

Atopic Dermatitis, Asthma

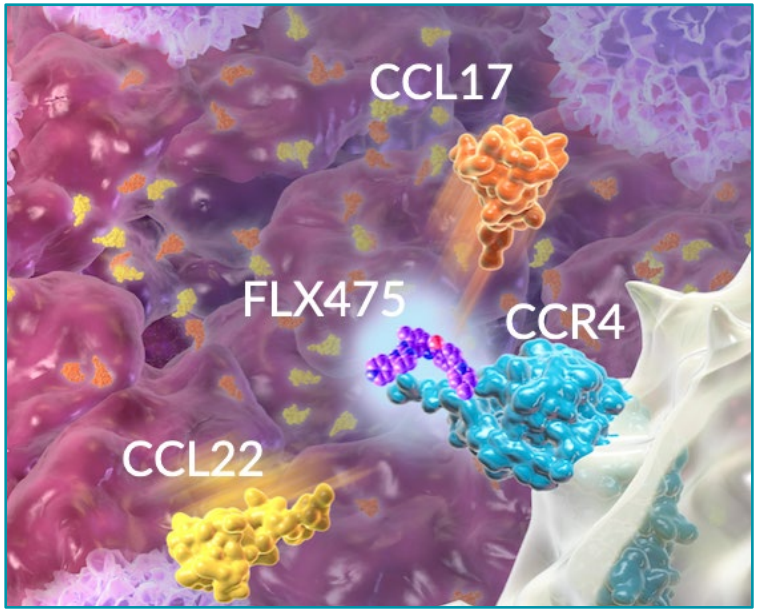


Th2 cells are recruited via CCR4 into tissues and **drive** allergic inflammation

FLX475: CCR4 Antagonist for Oncology

FLX475: Oral CCR4 Antagonist in Phase 2

- Designed to selectively block tumor T_{reg} while sparing normal tissues and beneficial immune cells
- Phase 1/2 study ongoing with PoC readout
- Demonstrated preliminary evidence of clinical activity
- Collaborations with Merck and Hanmi to enable global development program
- Issued U.S. composition of matter patent with coverage through 2037



Blocks interaction with CCR4 ligands CCL22 and CCL17 on T_{reg}

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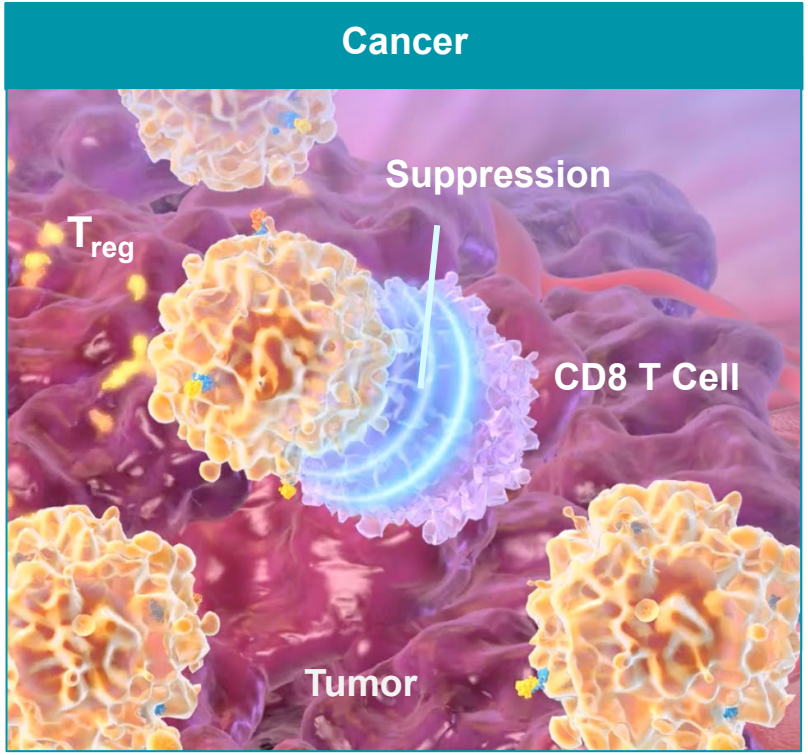
T_{reg} Allows Tumors to Evade the Immune System



- T_{reg} are a major barrier to effective antitumor immunity
  - T_{reg} numbers correlate with poor clinical outcome across most tumor types
- Act as powerful suppressors of the immune response
  - Patients with genetic defects in T_{reg} exhibit severe autoimmunity
- CCR4 pathway specifically drives T_{reg} accumulation in the tumor but not in healthy tissues

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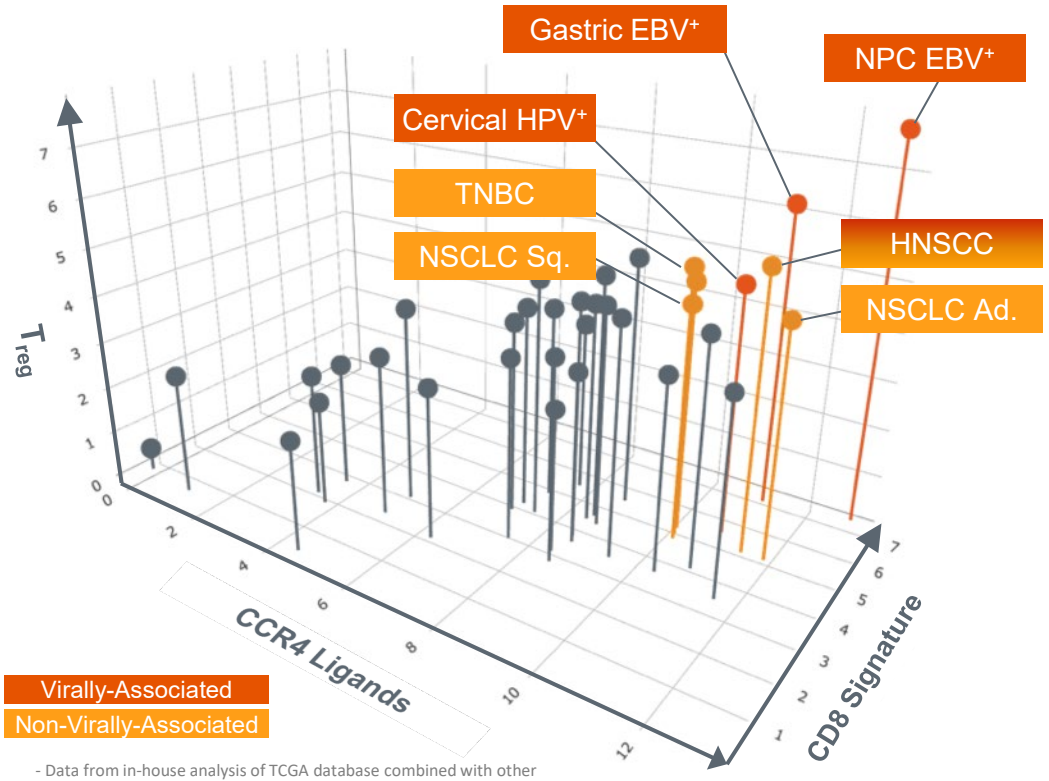
Identification and Characterization of “Charged” Tumors



- Data from in-house analysis of TCGA database combined with other data sets; Confirmed in > 400 tumor microarrays
- The graph above reflects a logarithmic scale on each axis

- “Charged” tumors: express high levels of CCR4 ligands, T_{reg} and CD8 cells
  - Non-Small Cell Lung Cancer
  - Triple Negative Breast Cancer
  - Head and Neck Cancer
  - Virally-Associated Cancers
- “Charged” tumors tend to be “hot” with high levels of T_{reg} likely holding back antitumor immune response
- Potential for tissue-agnostic accelerated approval in virally-associated tumors

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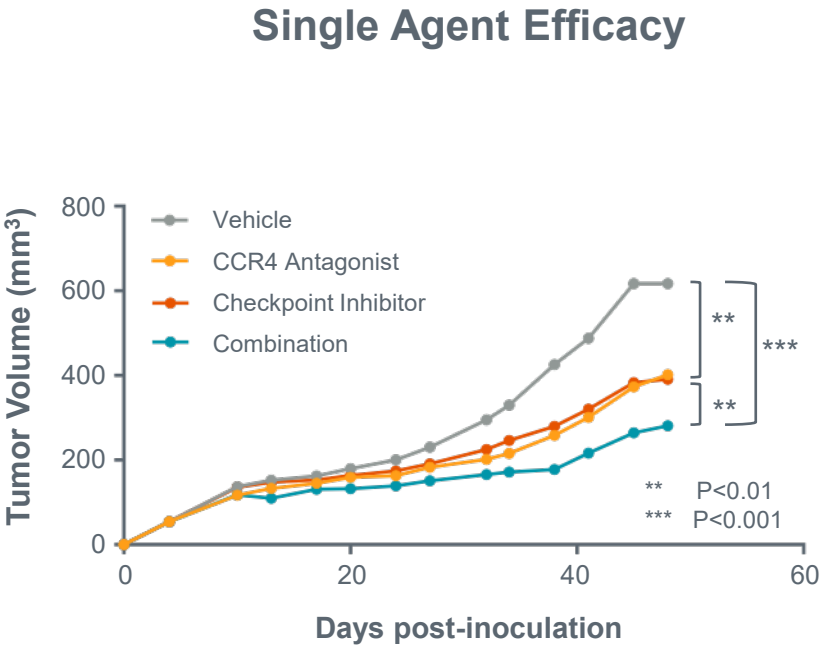
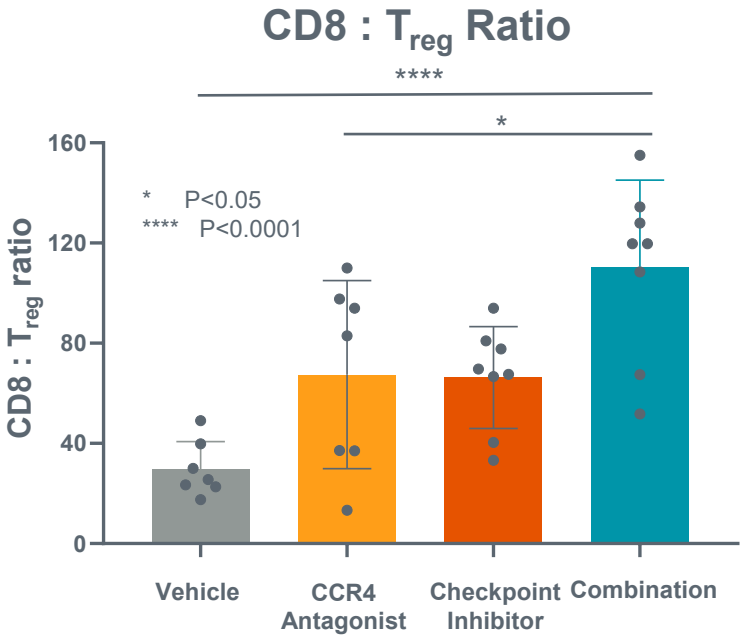
A Large Proportion of Multiple Tumor Types are “Charged”

Tumor Type	Prevalence* (U.S.)	Virally Associated	Percent Viral	Estimated Percent “Charged”**
Non-Small Cell Lung Cancer	268,600	N/A	N/A	60-80%
Triple Negative Breast Cancer	145,500	N/A	N/A	
Head and Neck Squamous Cell Carcinoma	143,000	✓	25%-60%	
Nasopharyngeal Cancer	105,000***	✓	>95%	>90% of virally associated tumors
Hodgkin Lymphoma	28,500	✓	30%-50%	
Cervical Cancer	46,800	✓	>95%	
Non-Hodgkin Lymphoma	225,000****	✓	Widely variable among subtypes	

* Based on 2012 Globocan registries 5-year prevalence (2008-2012 estimates)
** Data from in-house analysis
*** World-wide prevalence
**** Based on 2018 Globocan registries 5-year prevalence (2013-2018 estimates)

CCR4 Antagonist: Single Agent Activity in a Mouse Model of a “Charged” Tumor

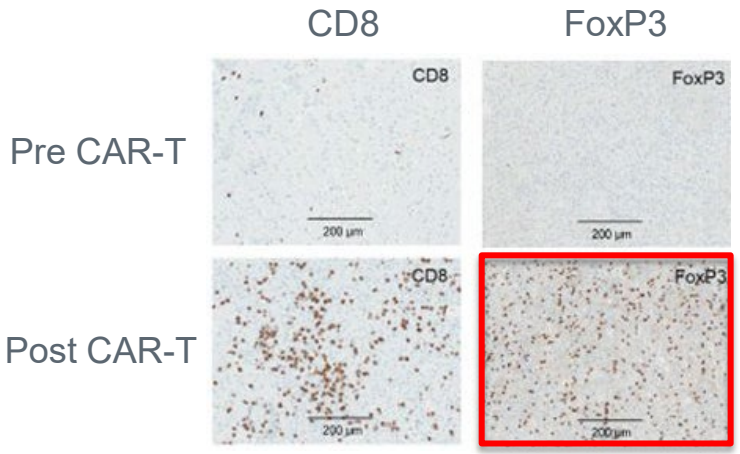




Pan02 “Charged” Tumor

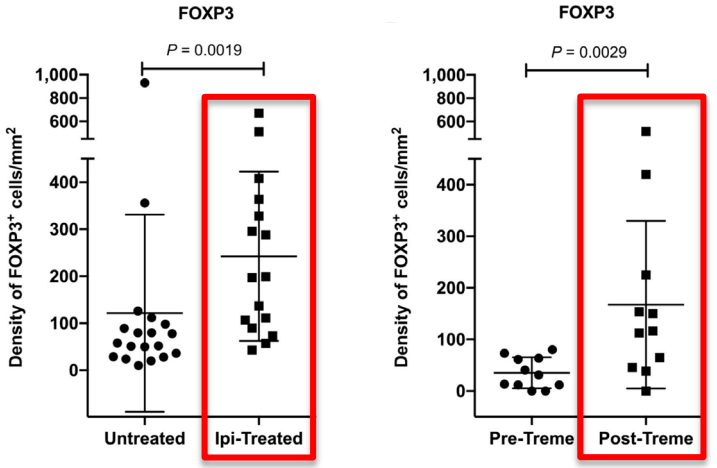
Accumulation of T_{reg} in the TME is a General Adaptive Immune Resistance Mechanism to Treatment

CAR-T Cell Therapy



O'Rourke et al. *Science Trans. Med.* (2017)

Anti-CTLA-4 Therapy



Sharma et al. *Clinical Cancer Research* (2019)

- Accumulation of T_{reg} has also been observed in both post anti-PD-1 and after conventional therapies such as radiation or chemotherapy

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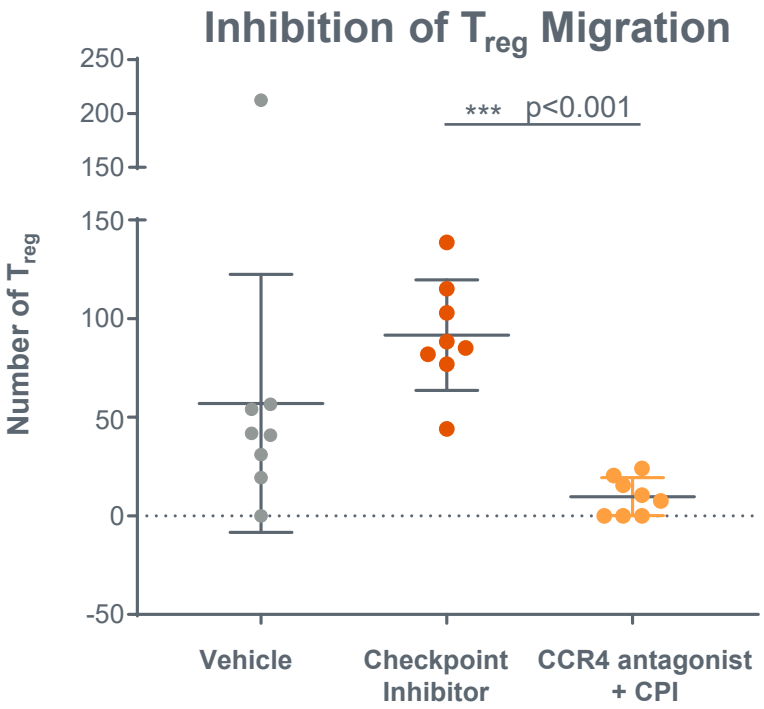
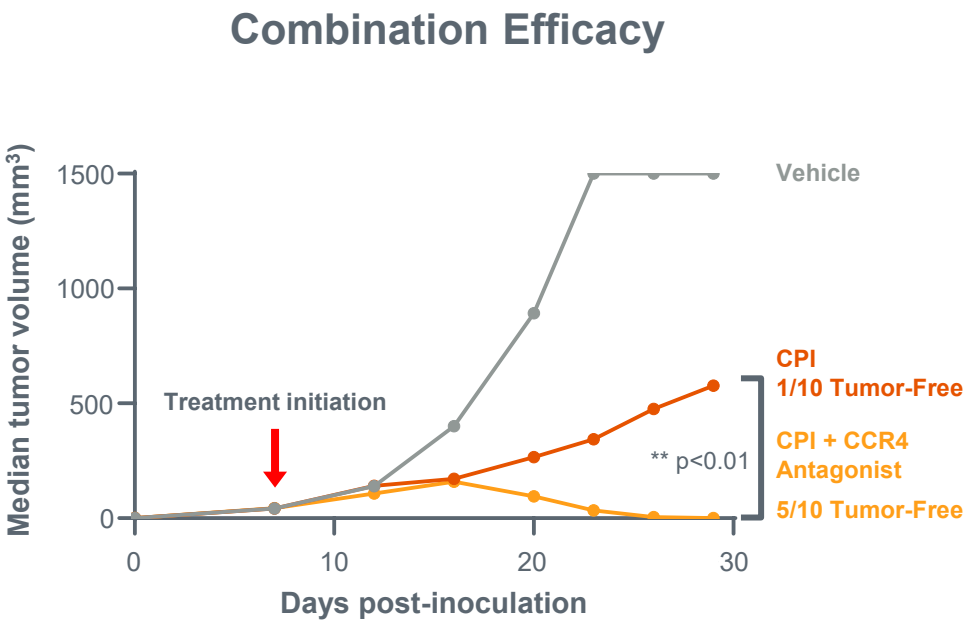
CCR4 Antagonist Synergizes with Checkpoint Inhibitors





CT26 tumor model

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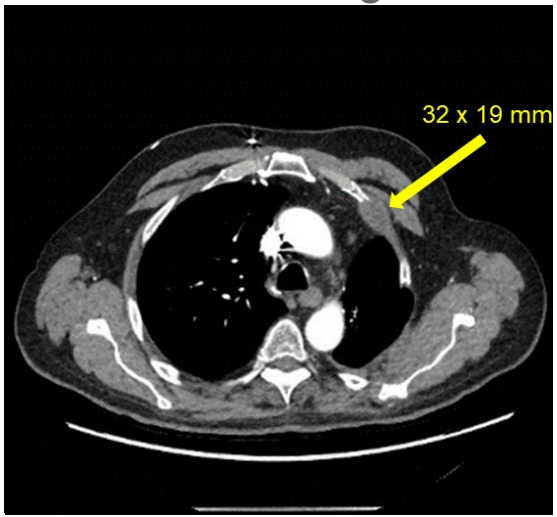
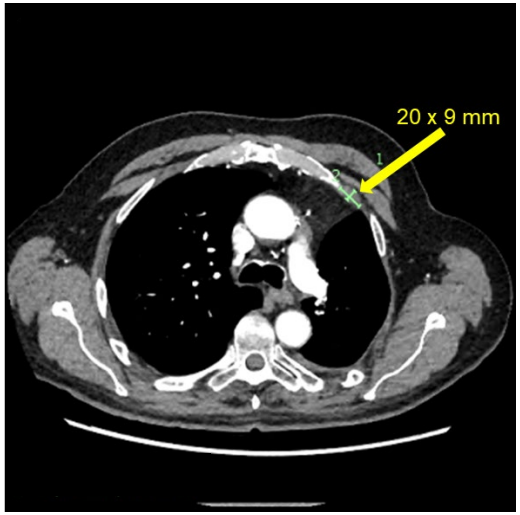
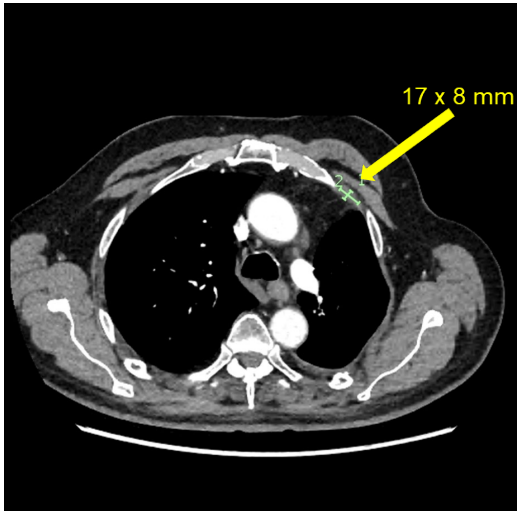
Phase 1 Summary

- **Completed Healthy Volunteer Study**
  - 104 healthy human volunteers
  - Target engagement achieved in majority of subjects at 75 mg QD
  - Excellent safety and tolerability at targeted exposures

- **Completed Phase 1 Portion in Cancer Patients**
  - Standard 3+3 dose escalation study as Monotherapy and Combination in cancer patients with mixed tumors
  - No safety issues; MTD not achieved
  - Recommended Phase 2 Dose (Mono and Combo): 100 mg QD
  - Encouraging evidence of clinical activity

Confirmed Partial Response in a Checkpoint Inhibitor-Refractory NSCLC Patient Treated with 50 mg FLX475+Keytruda*

- 4L NSCLC patient that progressed on prior atezolizumab therapy
- Confirmed partial response (PR) by RECIST 1.1 criteria. Patient remains on study.

Screening	Week 8	Week 14
		
Baseline	-37.5% (PR)	-47% (PR)

*Based on radiological analysis conducted at the clinical investigator site

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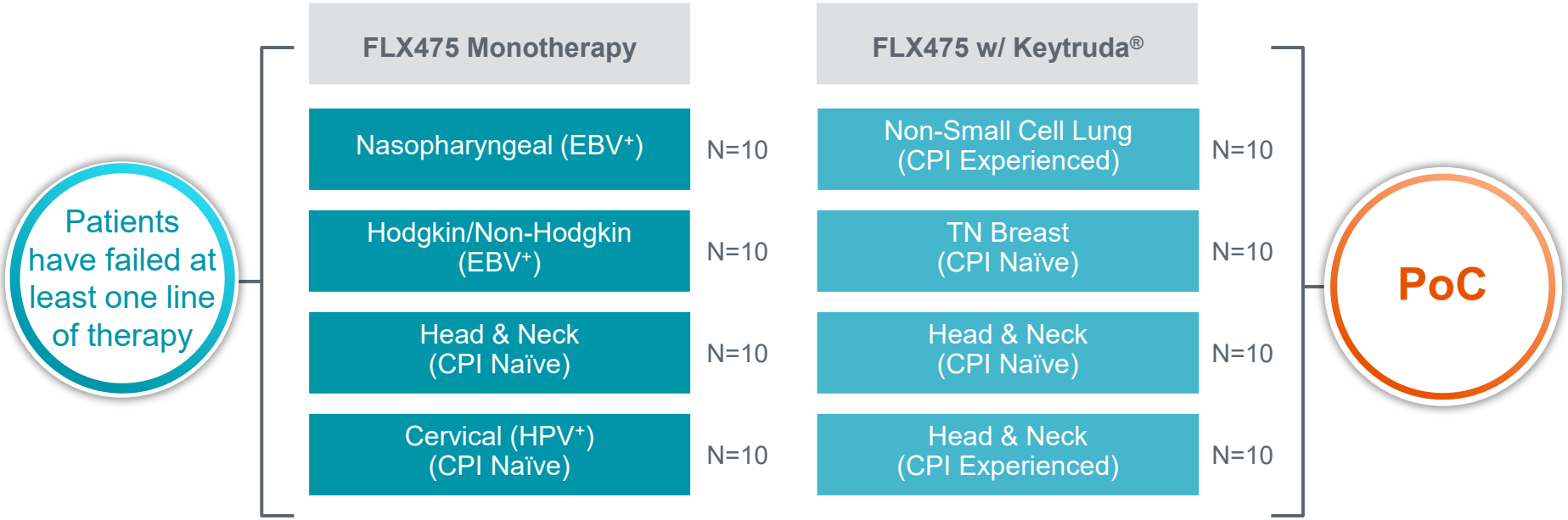
FLX475 Phase 2 Trial: PoC Readout



Endpoints: Safety, PK, Biomarkers, Objective Response Rate

- Gated 2-stage design: if positive ORR in a cohort, enroll additional 19 patients

CPI = Checkpoint Inhibitor

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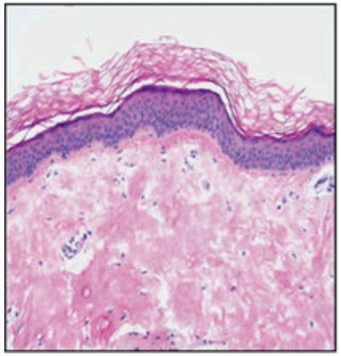
RPT193: CCR4 Antagonist for Allergic Diseases

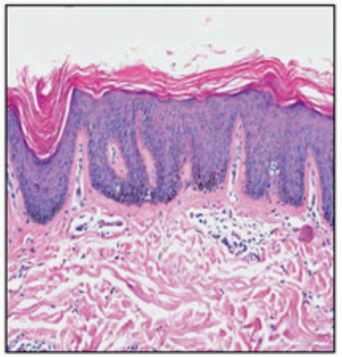
RPT193: Oral CCR4 Antagonist for Allergic Diseases

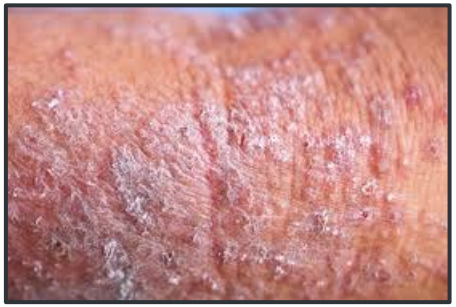
- Targeting atopic dermatitis, asthma, others
- Oral convenience could provide substantial competitive advantage to injectables and topical agents
- Preclinical studies and healthy volunteer data suggest an excellent safety profile
- Phase 1b trial ongoing in atopic dermatitis patients with PoC readout

Normal Human Skin



AD Lesional Skin





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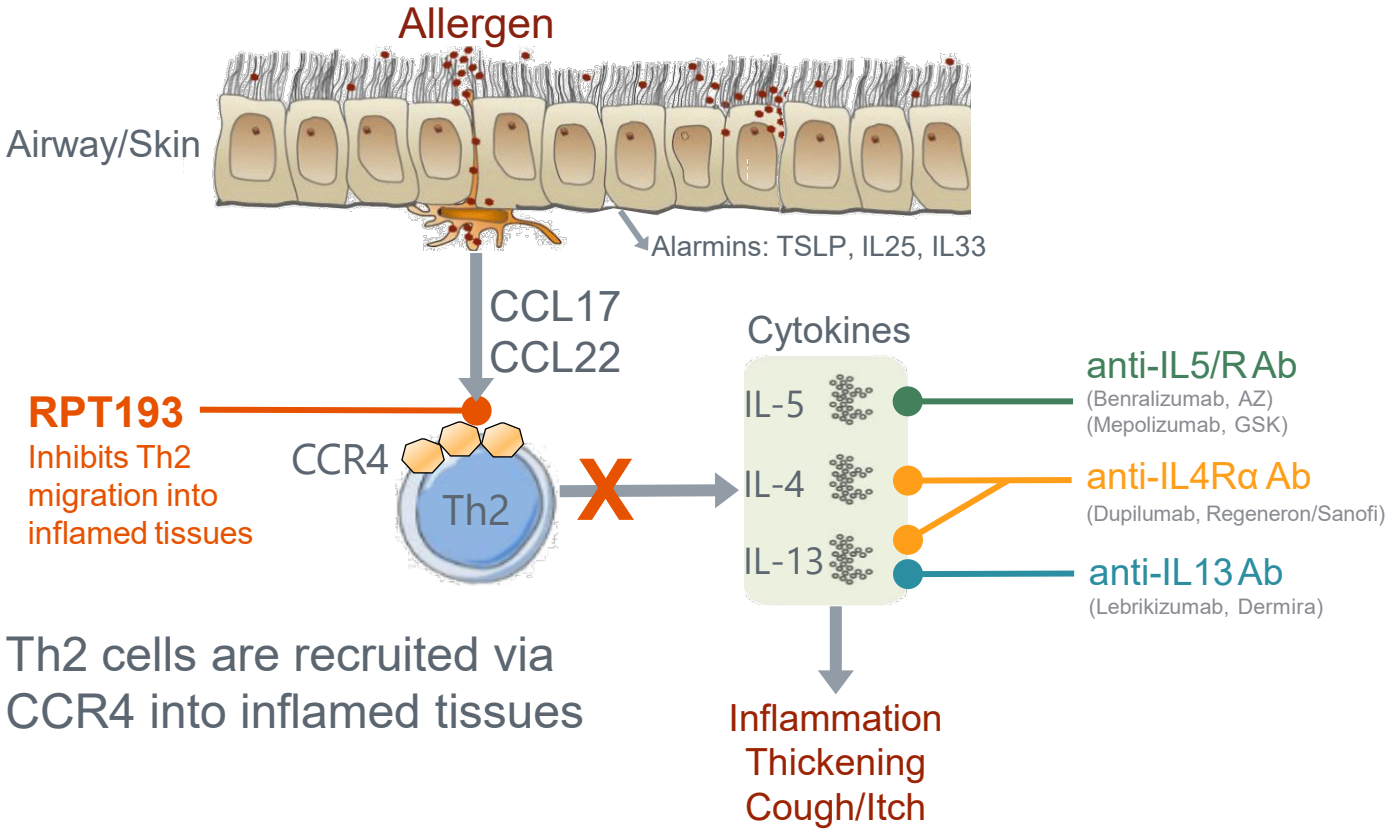
RPT193 Acts on a Well Validated Pathway in Asthma and Atopic Dermatitis (AD)



RPT193 Potential Advantages

	RPT193	Dupilumab*	JAK inhibitors
Safety	● Preclinical and healthy volunteer data show an excellent safety profile	● Generally safe and well tolerated ● Conjunctivitis	● Immunosuppressive ● Potential black box warning for infections, malignancies and thromboembolic events
Route of Administration	● Oral, daily dosing	● Injectable	● Oral
Efficacy	● Preclinical data suggest efficacy similar to dupilumab*	● Durable clinical efficacy ● Activity in AD and asthma	● Similar to dupilumab*

* DUPIXENT®

● Favorable Characteristic
● Unfavorable Characteristic

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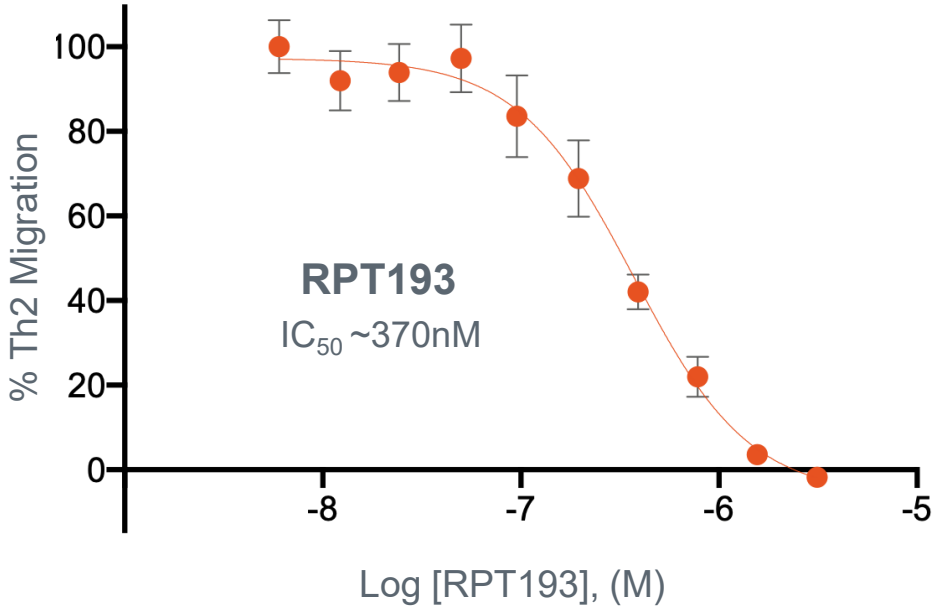
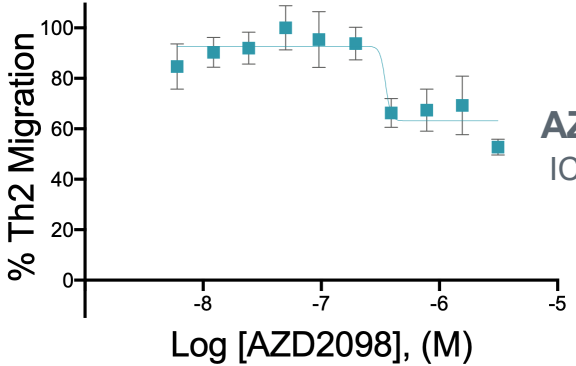
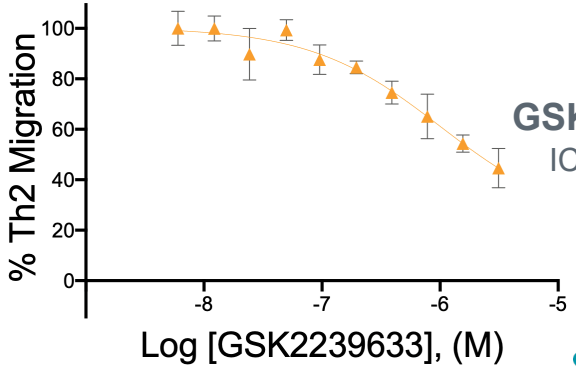
Potency of CCR4 Inhibitors in an In Vitro Th2 Chemotaxis Assay

Head-to-Head Comparison







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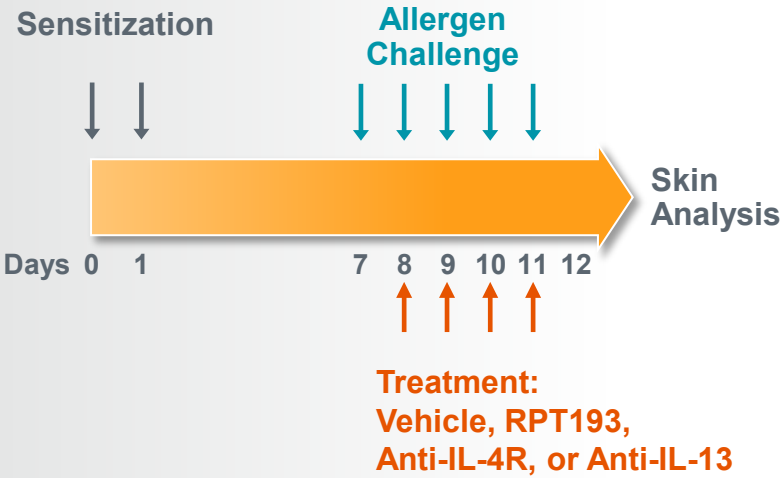
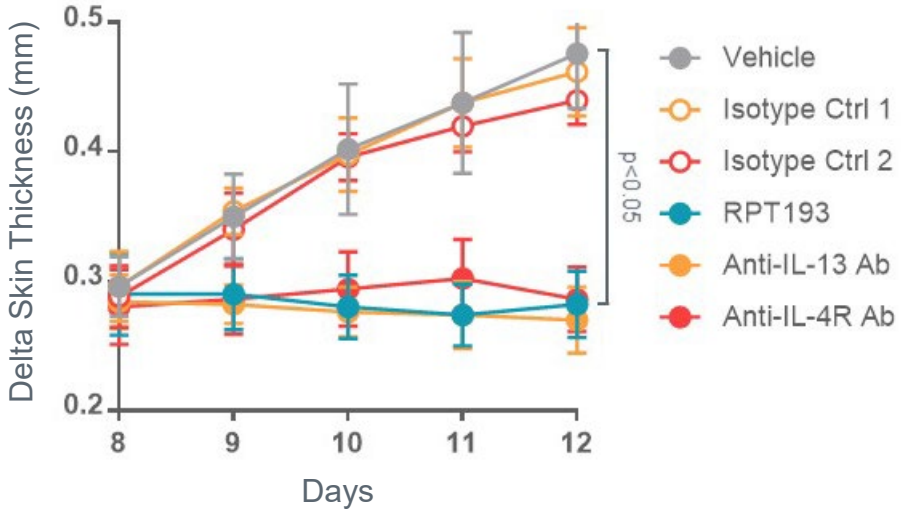
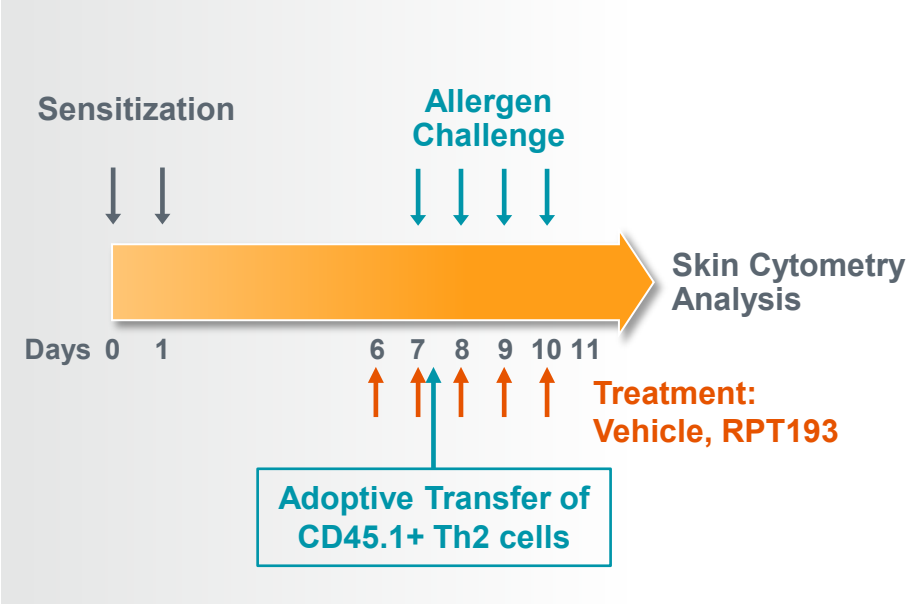
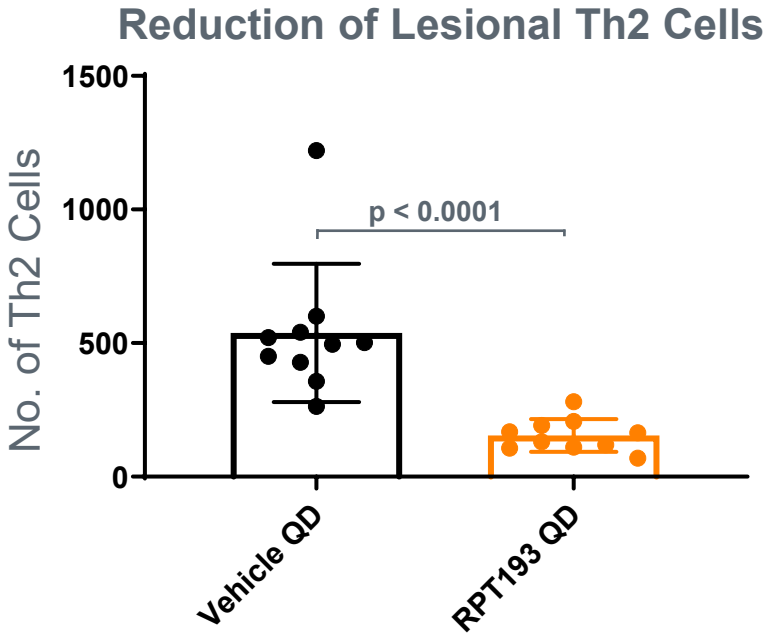
RPT193 Reduces Skin Inflammation in a Therapeutic Th2-Driven Atopic Dermatitis Model



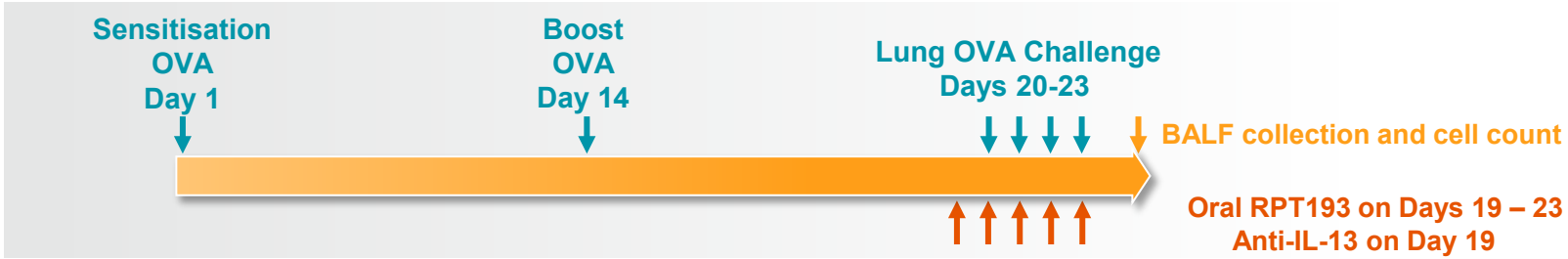


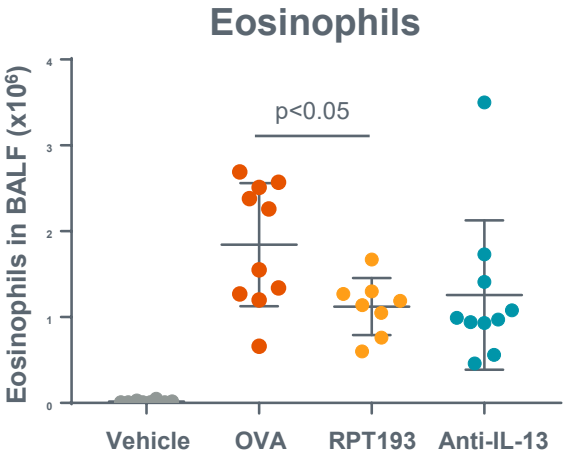
Significant Reduction of Th2 Cell Migration Into Inflamed Skin with RPT193 Assessed in a Mouse AD Model

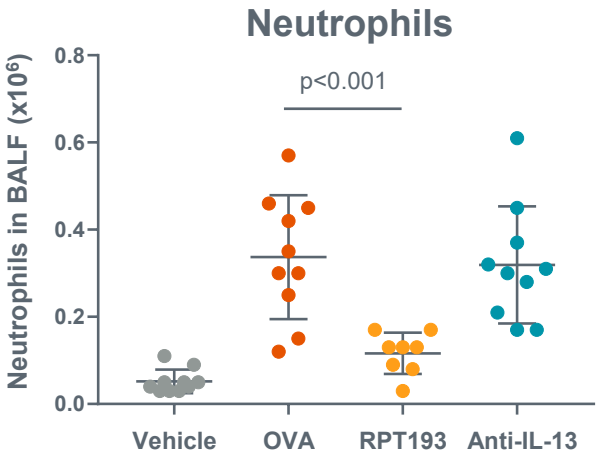


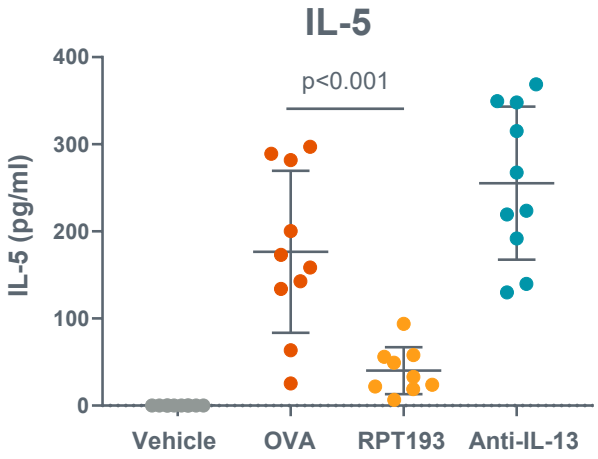


RPT193 Demonstrates Broader Activity than Anti-IL-13 in an Allergic Asthma Model

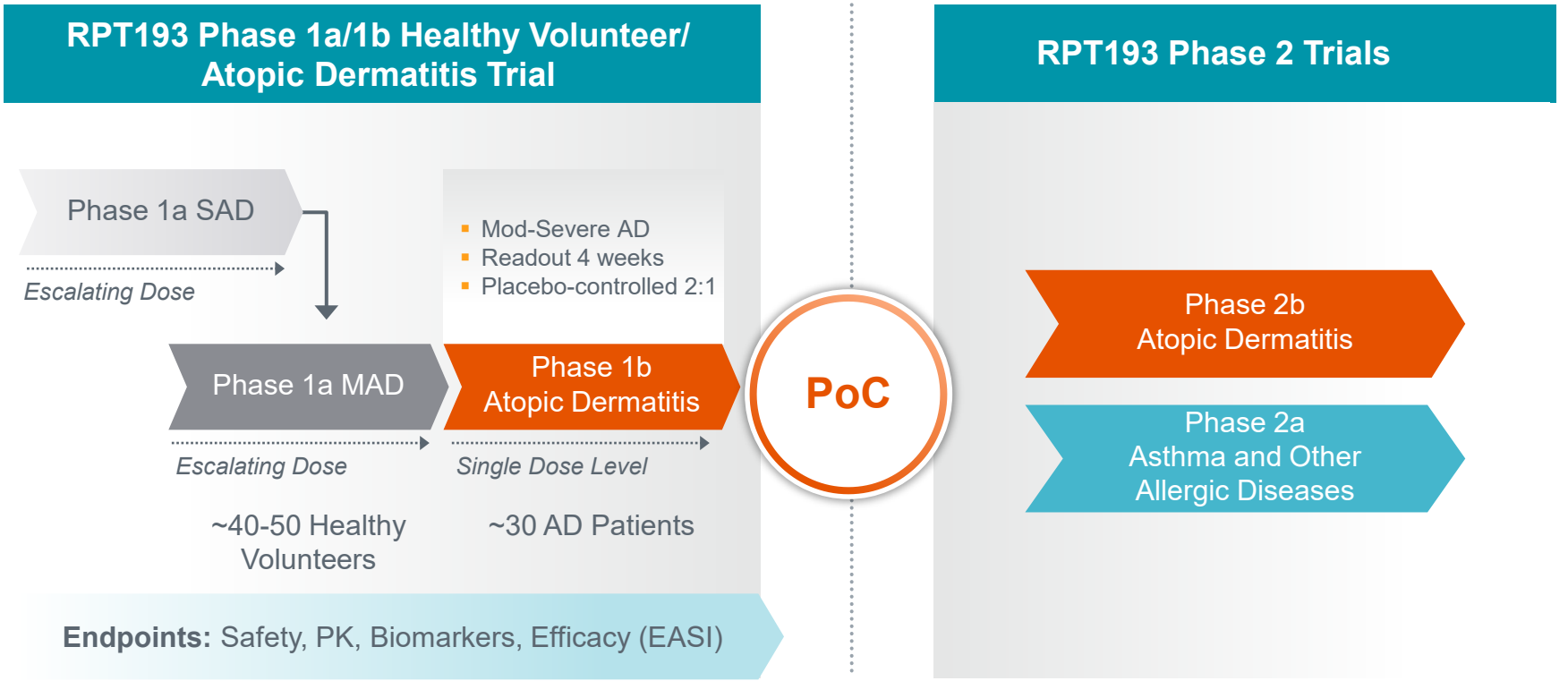






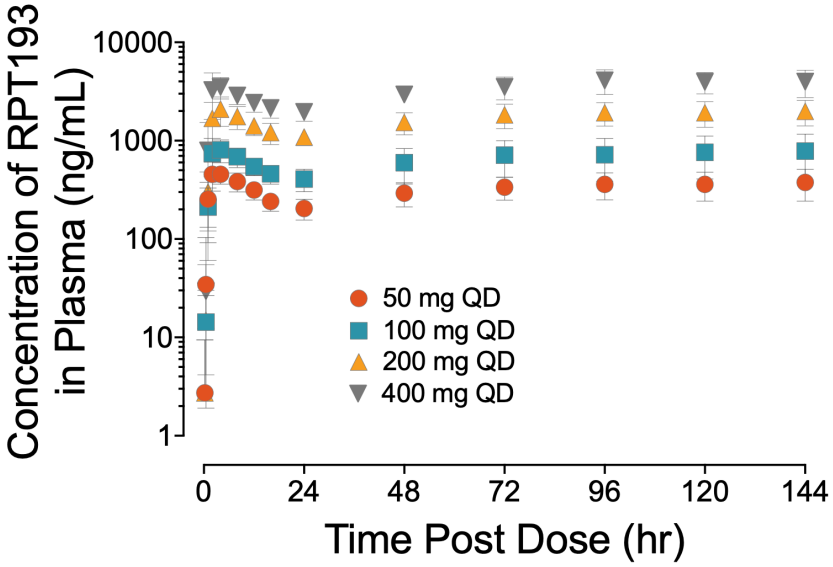


RPT193: Seamless Clinical Trial Design to PoC and Beyond

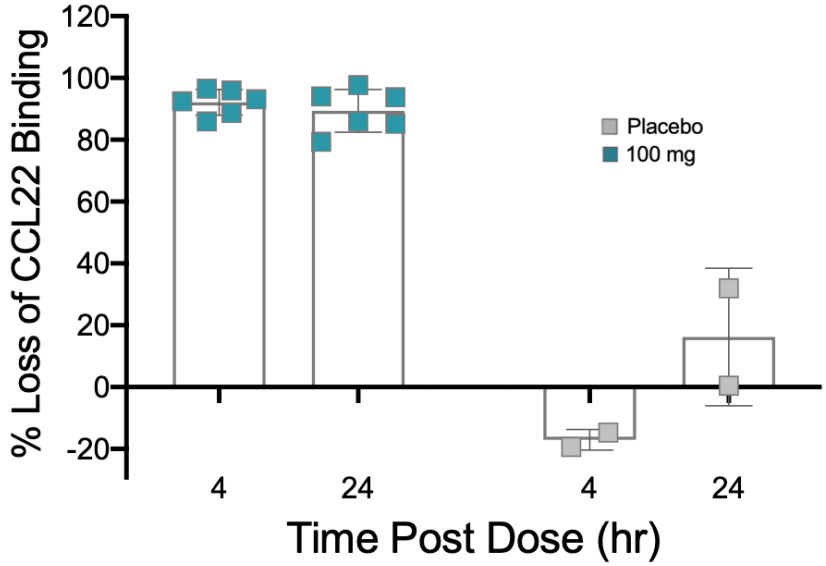


Phase 1a HV Data Supports Once-Daily Dose

Dose-Proportional Oral PK with ~24 Hr. Half-Life



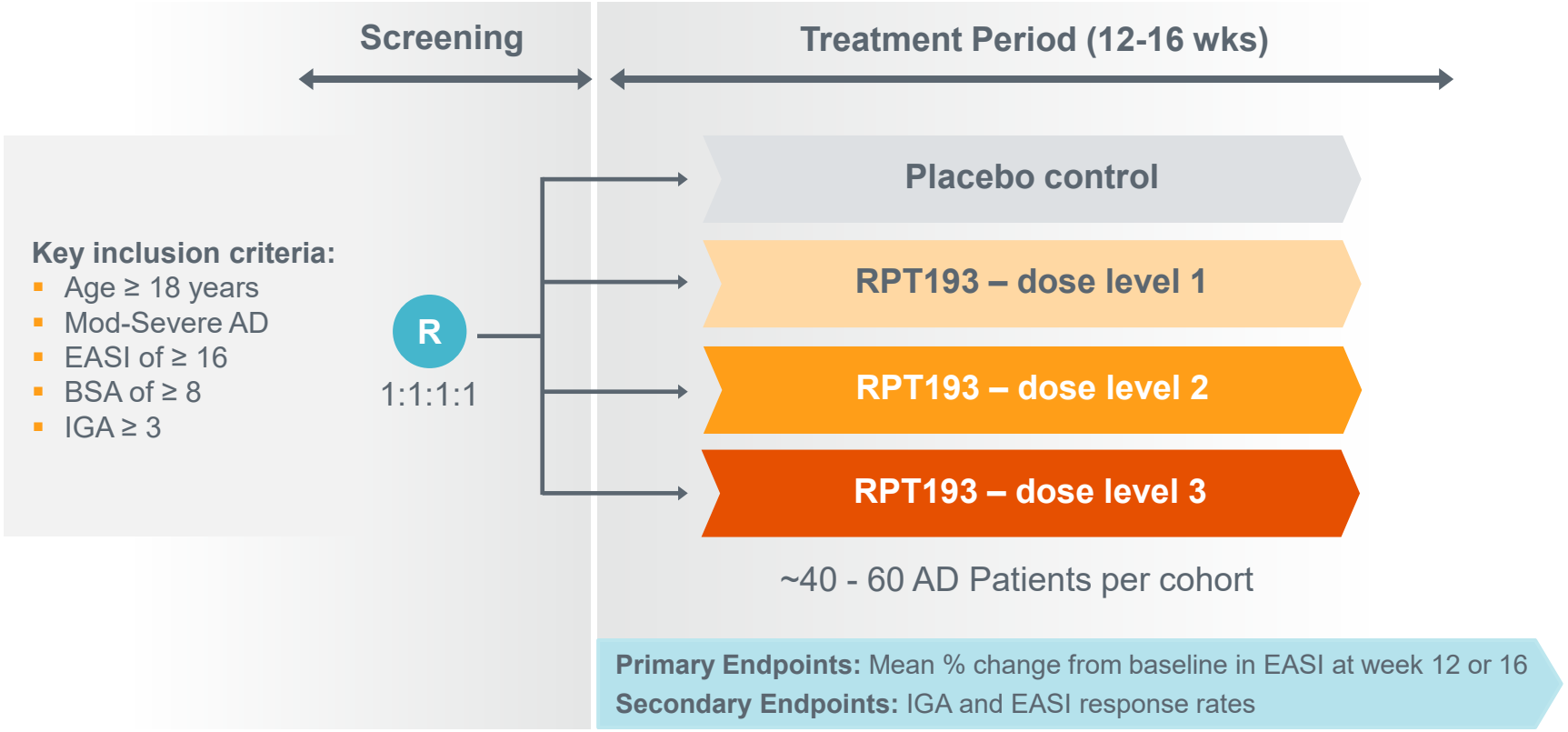
Targeted Level of CCR4 Inhibition Exceeded at 100 mg



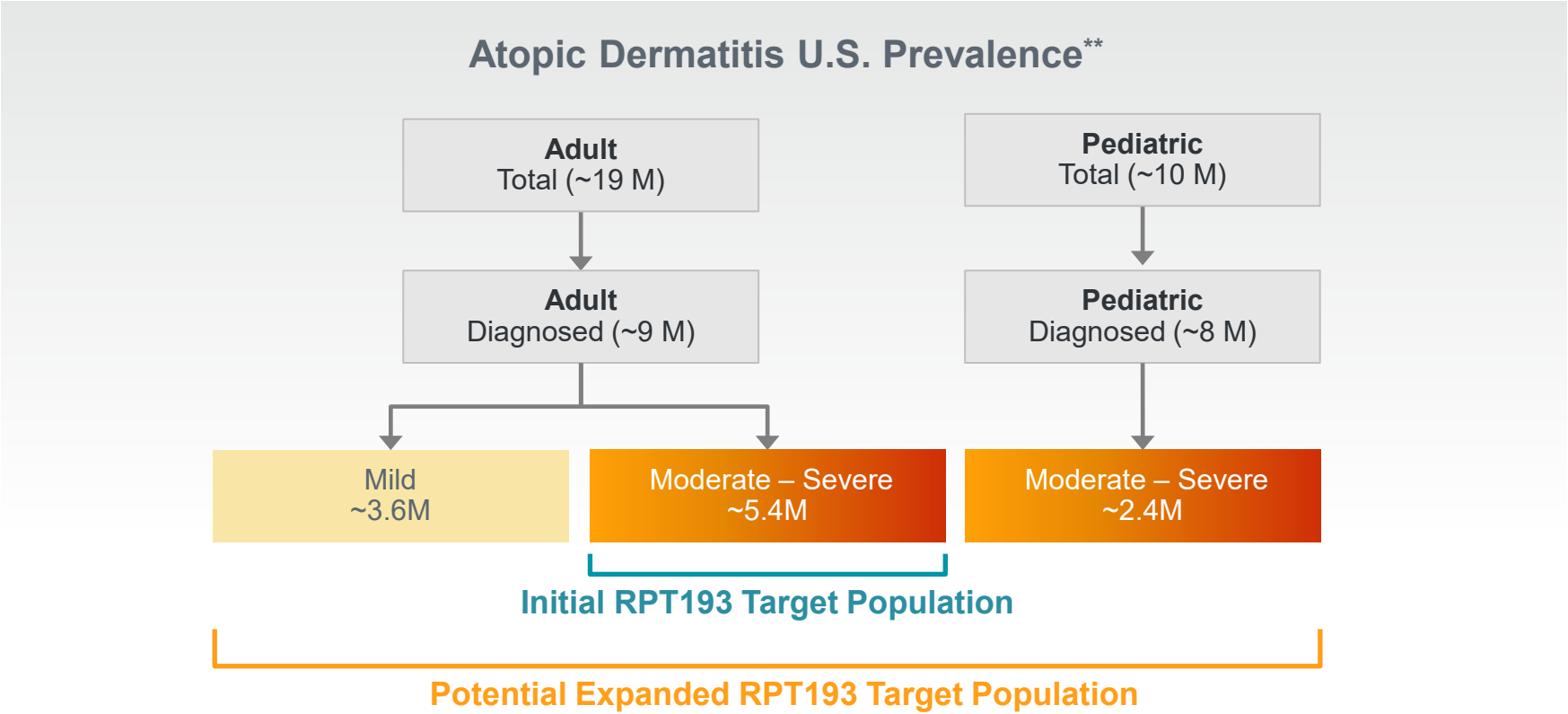
- 100 mg once-daily dose exceeds target exposures and pharmacodynamic effect
- Excellent safety profile to date (n=56)

RAPT THERAPEUTICS

Proposed Phase 2b Double Blind, Placebo-Controlled Trial

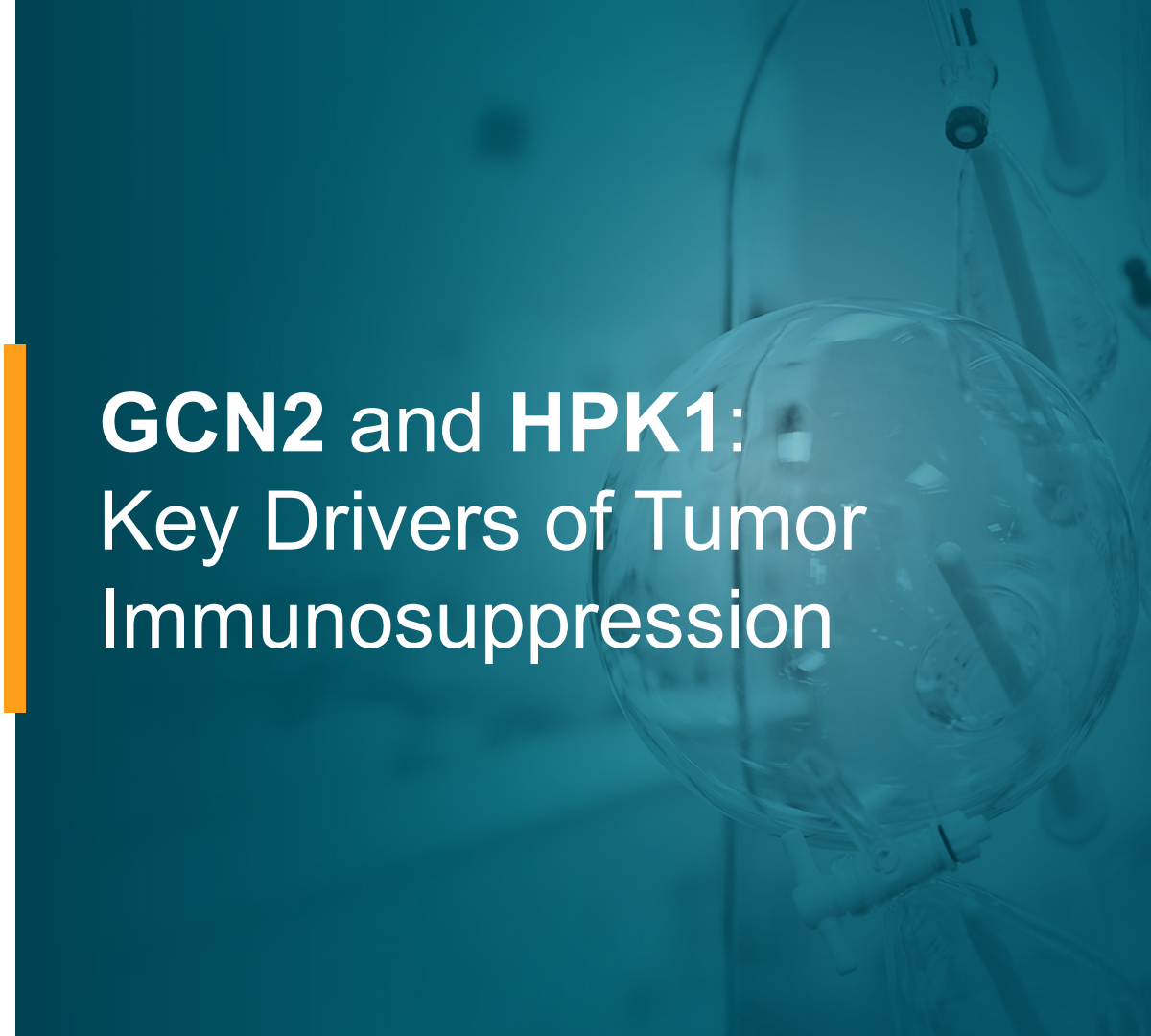


RPT193: Potentially Disruptive Convenience and Safety Profile



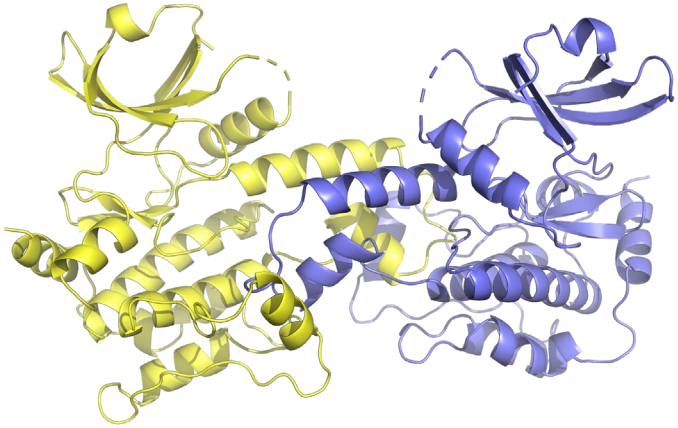
**2018 Data, Decision Resources, nationaleczema.org, Shaw et al., 2011

GCN2 and **HPK1**: Key Drivers of Tumor Immunosuppression



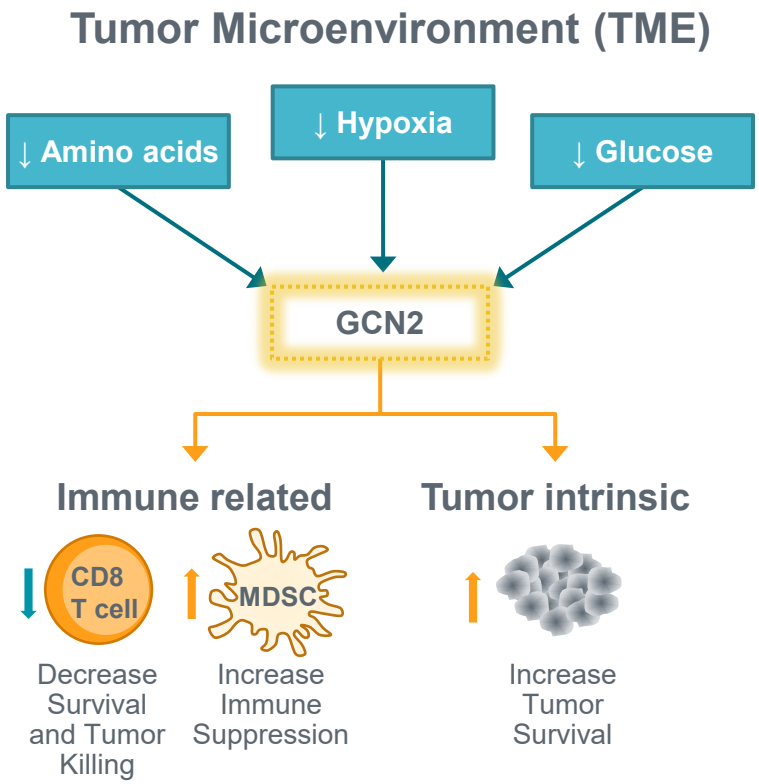
HPK1 Program: Unlocking Antitumor T Cells

- HPK1 is a negative regulator of T cell receptor activation
- Identified from a RAPT computational screen along with PD-1 and CCR4
- High resolution crystal structures and docking models have enabled the discovery of potent and selective HPK1 inhibitors with good PK
- HPK1 inhibition increases tumor-specific T cell activation leading to robust efficacy in tumor models
- Program in Lead Optimization



RAPT HPK1 Crystal Structure

GCN2 Program: Reversing Tumor Progression Caused by Metabolic Stress



- TME harbors significant metabolic stress
- GCN2 inhibitors have potential to:
  - Reactivate the immune response
  - Increase tumor cell death
  - Act specifically in the TME resulting in better therapeutic index
- RAPT GCN2 inhibitor demonstrated enhanced immune function in vitro and single agent antitumor activity in vivo

Mellor and Munn, 2008; Ye et al, 2010; Wang et al, 2013

RAPT THERAPEUTICS

Focused on Oral Drugs Targeting Critical Immune Drivers of Disease

- **Diversified pipeline**
- **Large market opportunities**
- **Strategic collaborations**
- **Proprietary discovery engine**

CLINICAL

FLX475 (Oncology):

- Selectively targets immunosuppressive tumor T_{reg}
- Encouraging clinical activity in Phase 1 study
- **Phase 2 PoC study ongoing**

RPT193 (Allergic Disease):

- Oral agent targets inflammatory Th2 cells
- Robust PK/PD with excellent safety in Ph1 study
- **Phase 1b PoC in atopic dermatitis ongoing**

DISCOVERY

HPK1 (Oncology):

- Unlocks T cell activation to tumor antigens

GCN2 (Oncology):

- Turns on an antitumor metabolic switch in TME

Thank You