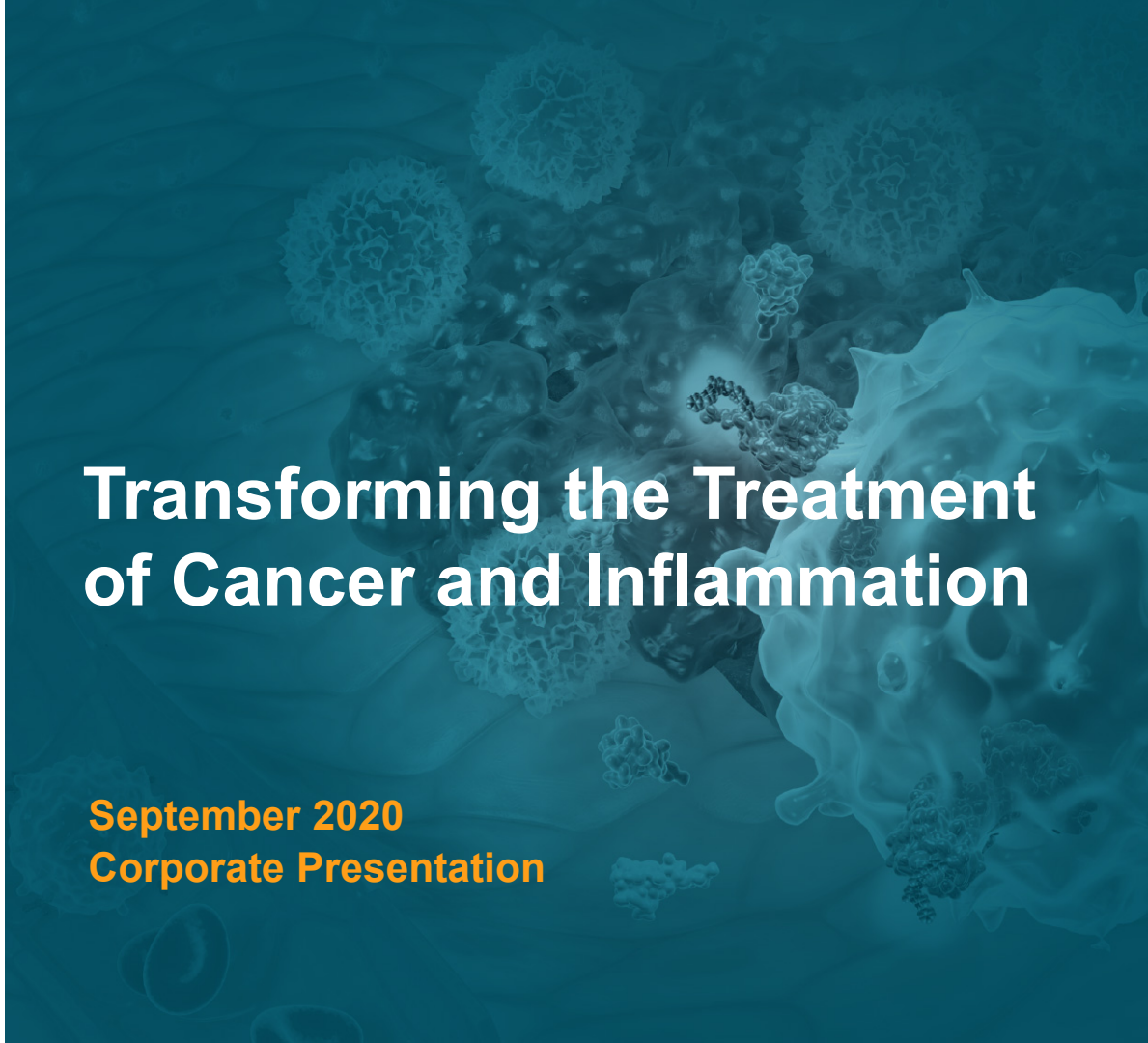
RAPT THERAPEUTICS

# Transforming the Treatment of Cancer and Inflammation



**September 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-Q filed with the Securities and Exchange Commission on August 13, 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

- Proprietary discovery engine
- Diversified pipeline
- Large market opportunities
- Multiple near-term clinical readouts
- Strategic collaborations

**CLINICAL**

**FLX475** (Oncology):

- Selectively targets immunosuppressive tumor $T_{reg}$
- Encouraging clinical activity in Phase 1 study
- **Phase 2 PoC study ongoing – data readout 2H 2020**

**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 – data readout by YE 2020**

**DISCOVERY**

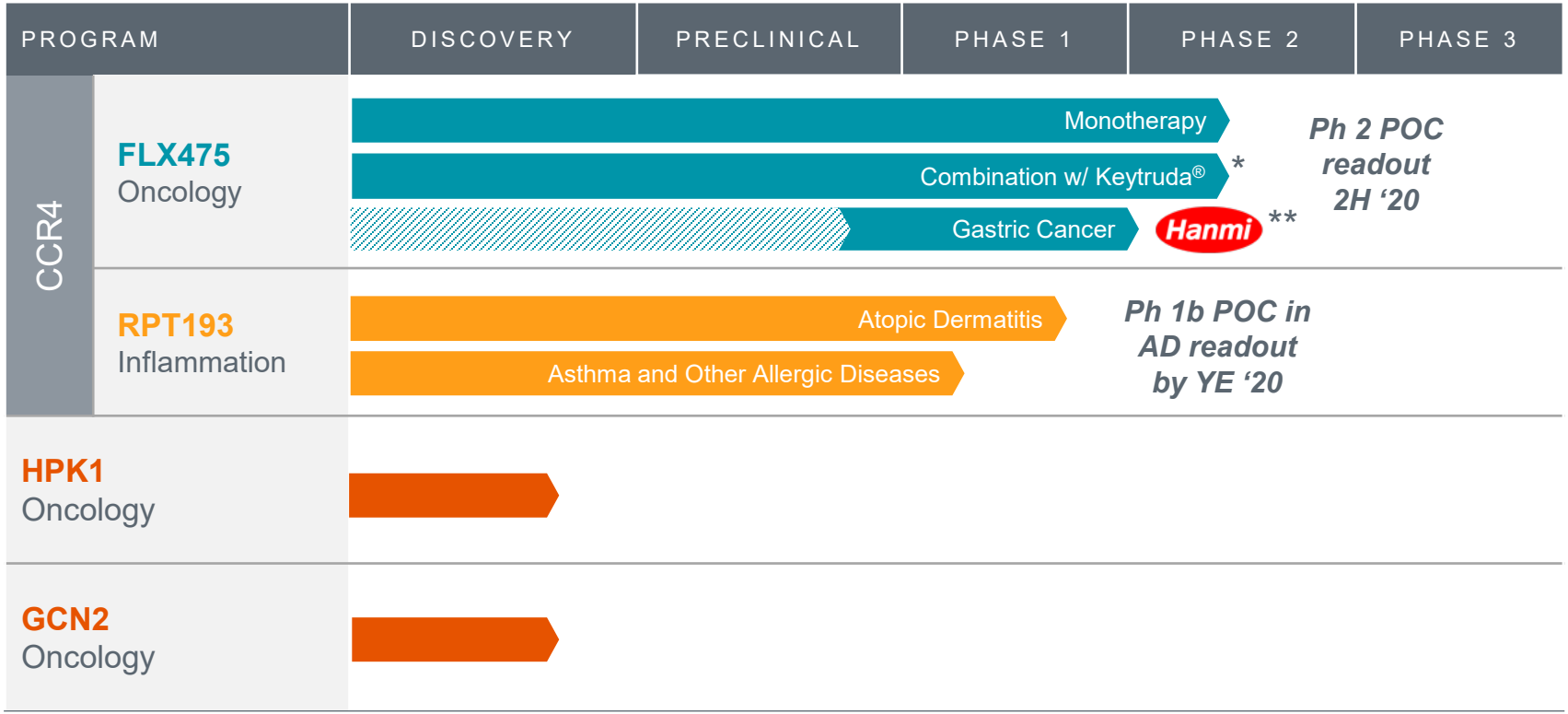
**HPK1** (Oncology):

- 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)

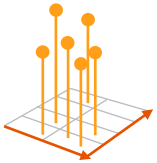
# Proprietary Drug Discovery and Development Engine

**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



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# Experienced Leadership Team and Scientific Advisory Board

## Leadership

**Brian Wong, MD, PhD**
Chief Executive Officer

**Dirk Brockstedt, PhD**
Chief Scientific Officer

**Phyllis Gray**
Vice President, Human Resources

**William Ho, MD, PhD**
Chief Medical Officer

**Paul Kassner, PhD**
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**Sylvia Wheeler**
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**Peter Svennilson**
Managing Partner and Founder, The Column Group

**Brian Wong, MD, PhD**
CEO, RAPT Therapeutics

## Scientific and Clinical Advisors

### Oncology

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

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Professor, Medicine, Hematology/Oncology & Director, UCLA

**Scott J. Antonia, MD, PhD**
Instructor, 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**
Managing Partner, The Column Group

### Allergy / Immunology

**Emma Guttman-Yassky, MD, PhD**
Professor and Vice Chair for Research, Department of Dermatology; Director, Center for Excellence in Eczema; and Director, Laboratory of Inflammatory Skin Diseases, 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



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# Summary Financial Information

- Cash (at 6/30/20): $133.0M
- Q2 2020 net loss: $12.4M
- LTM net loss: $48.7M

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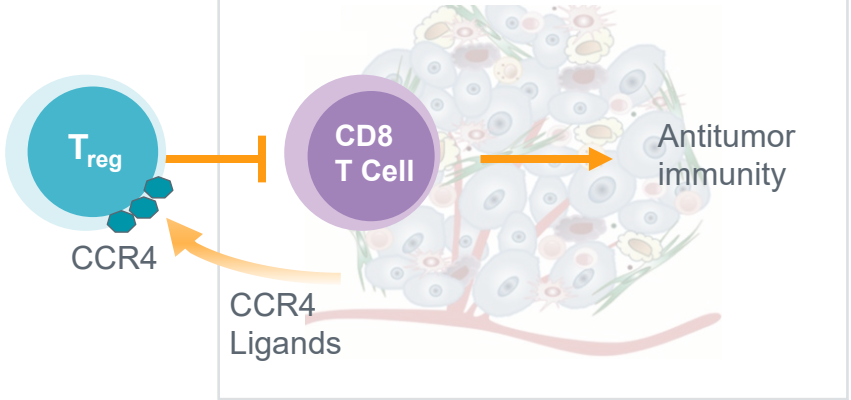
- Shares outstanding: 24.5M
- Options/RSUs outstanding: 1.6M
- FD shares outstanding: 26.1M

# Our CCR4 Program

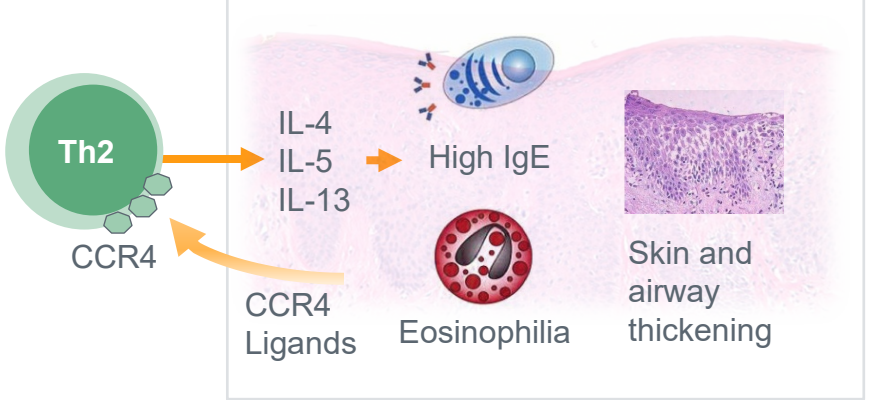
# CCR4 Drives Tumor Progression and Allergic Inflammation

## Cancer




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

## Atopic Dermatitis, Asthma

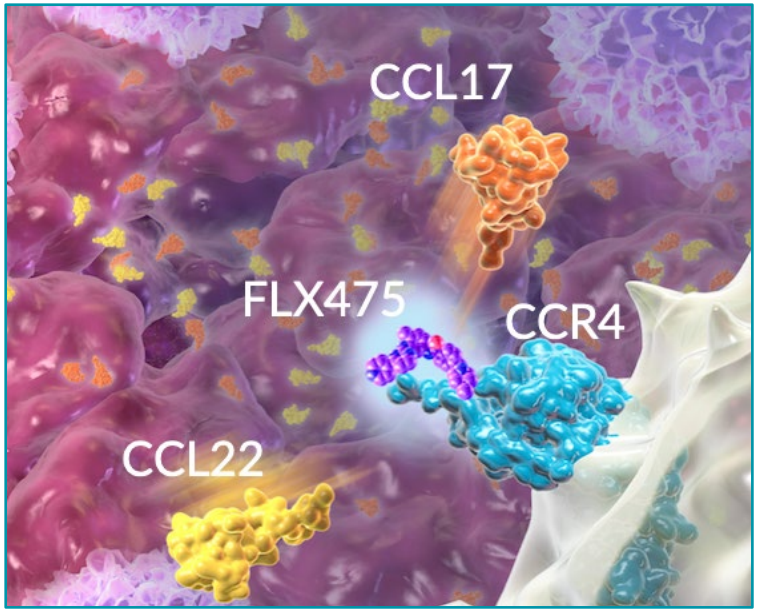



Th2 cells are recruited via CCR4 into tissues and **drive** allergic inflammation

# FLX475: CCR4 Antagonist for Oncology

# FLX475: Oral CCR4 Antagonist in Phase 2

- Highly potent and selective CCR4 small molecule antagonist
- Non-depleting mechanism designed to selectively block tumor $T_{reg}$ while sparing normal tissues and beneficial immune cells
- Potential for superior safety and efficacy compared to depleting antibodies
- 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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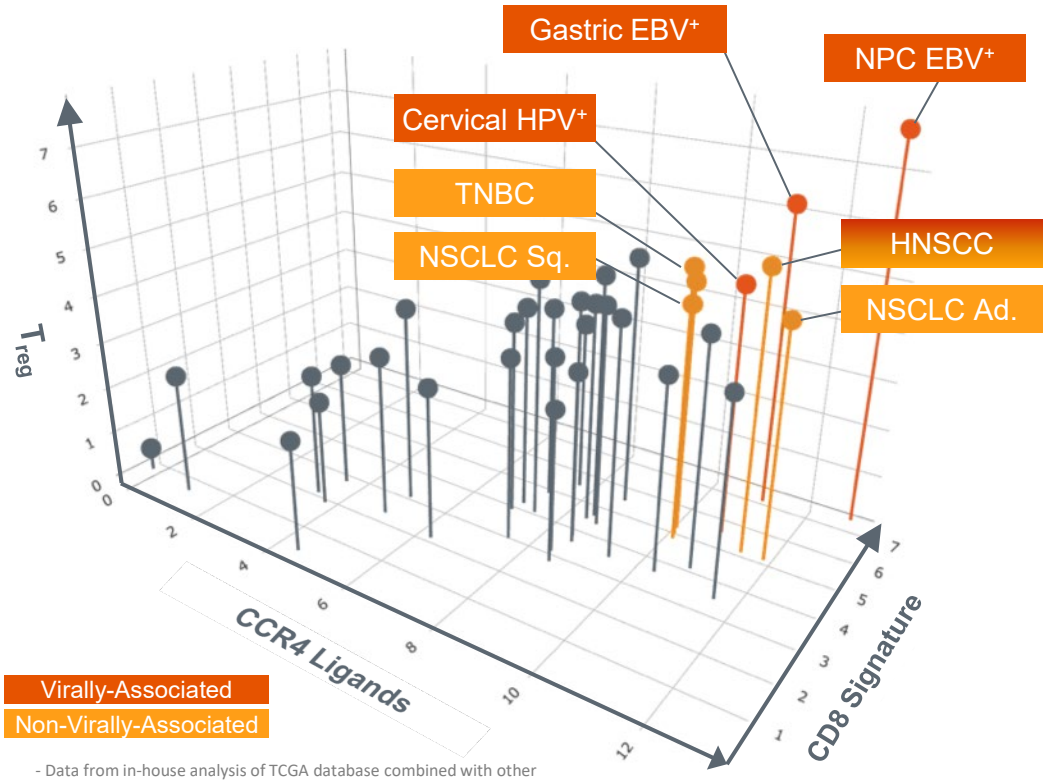
# Identification and Characterization of “Charged” Tumors




- 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

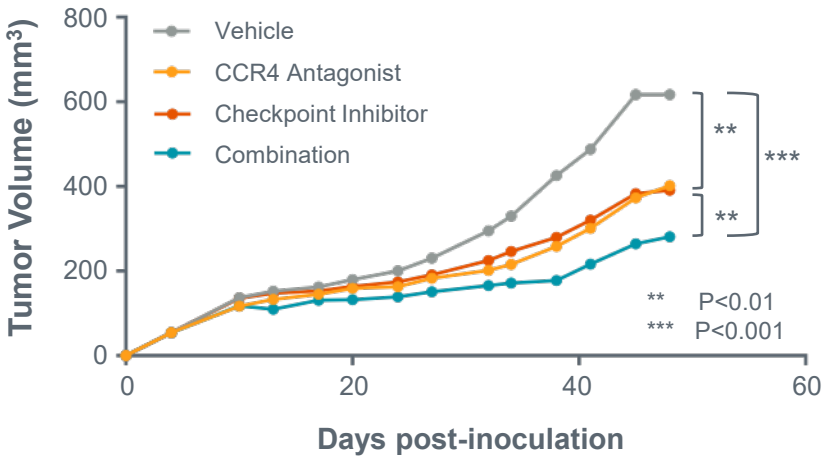
# A Large Proportion of Multiple Tumor Types are “Charged”

| 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 | |

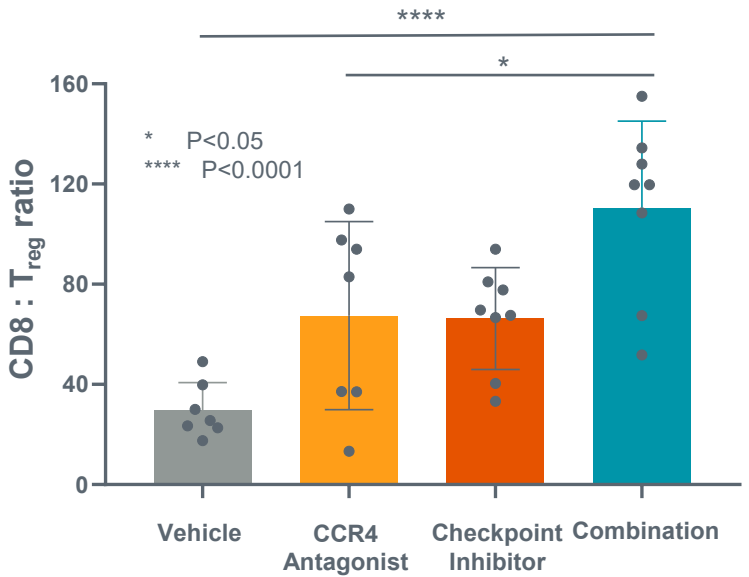
* Based on 2012 Globocan registries 5-year prevalence (2008-2012 estimates)
** Data from in-house analysis
*** World-wide prevalence
**** Based on 2018 Globocan registries 5-year prevalence (2013-2018 estimates)

# CCR4 Antagonist: Single Agent Activity in a Mouse Model of a "Charged" Tumor

## Single Agent Efficacy




## CD8 : $T_{reg}$ Ratio




Pan02 "Charged" Tumor



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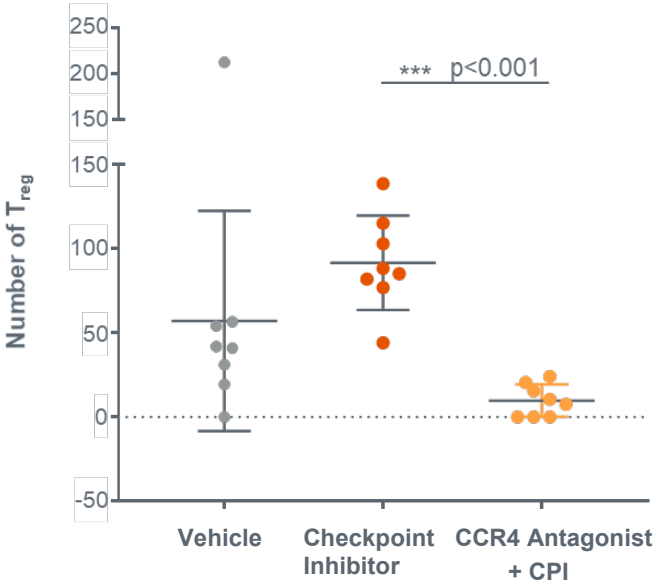
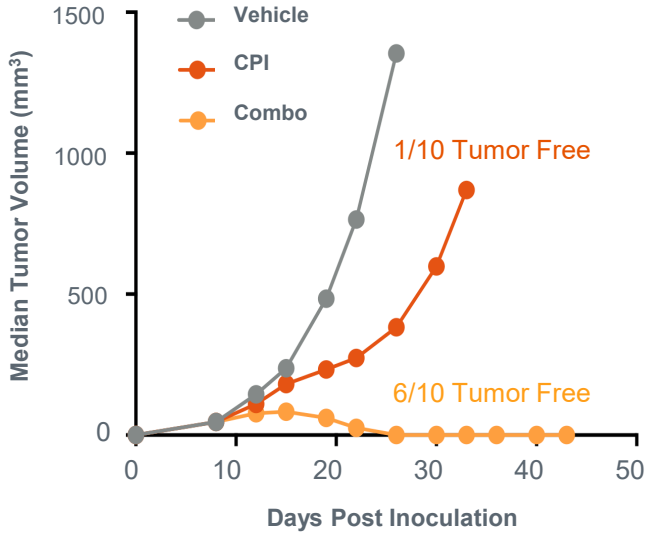
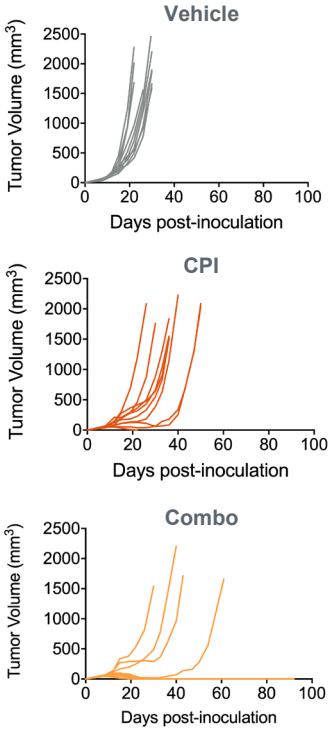
# CCR4 Antagonist Synergizes with Checkpoint Inhibitor Blockade

## Inhibition of $T_{reg}$ Migration




## Combination Efficacy




CT26 Tumor Model



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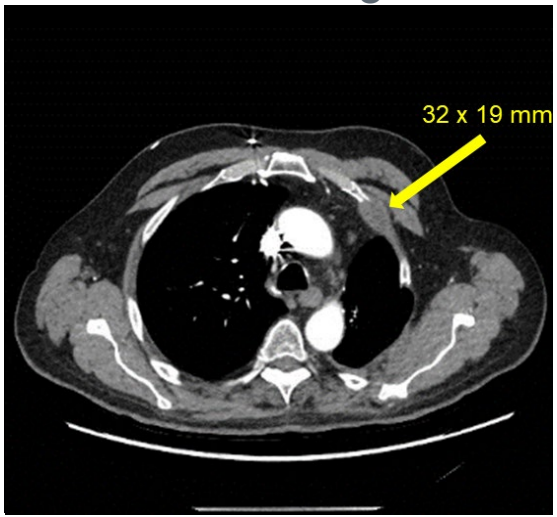
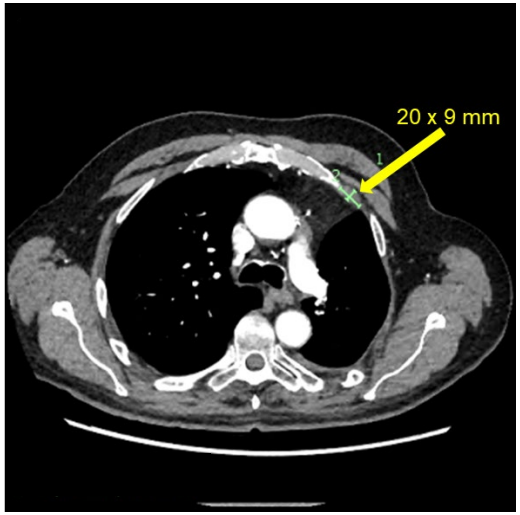
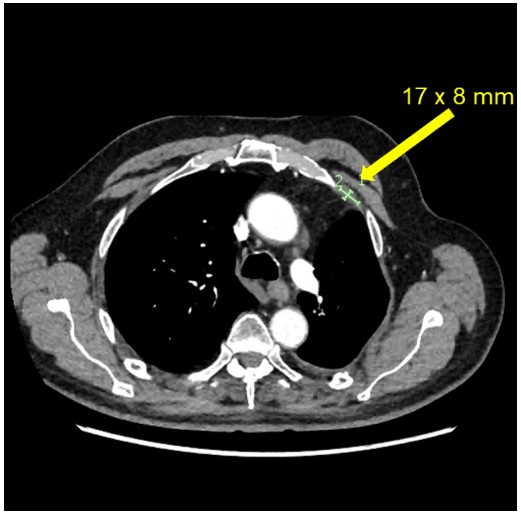
# Phase 1 Summary

- **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 and in response.

| 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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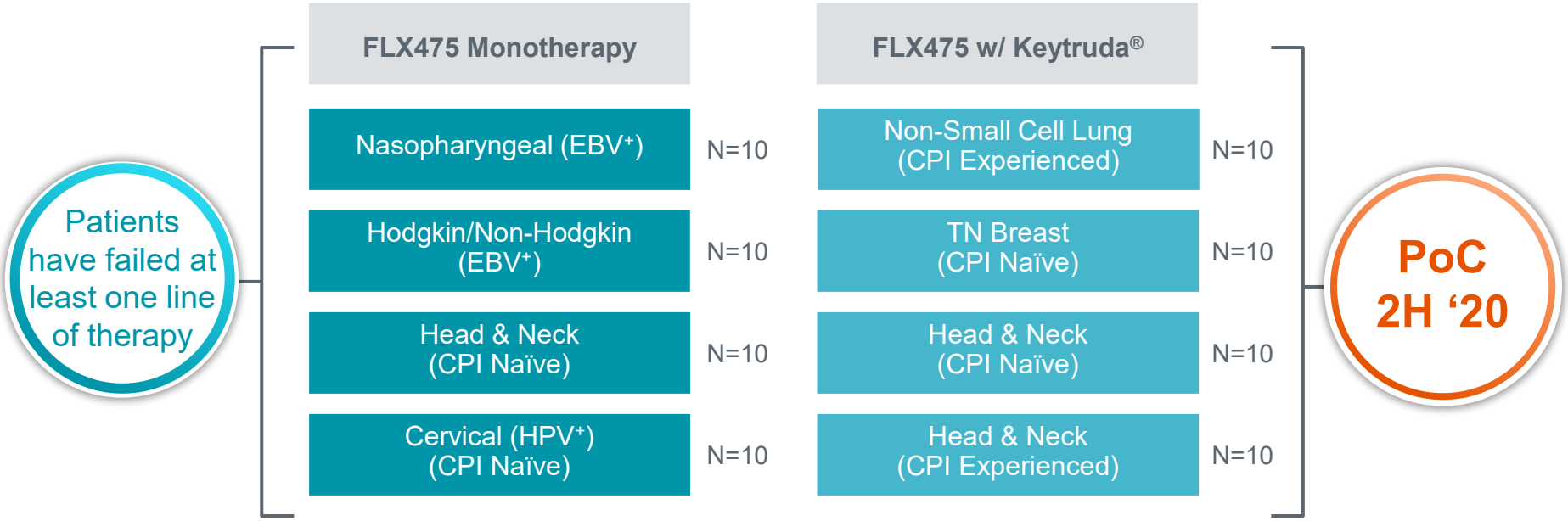
# FLX475 Phase 2 Trial: PoC Readout




**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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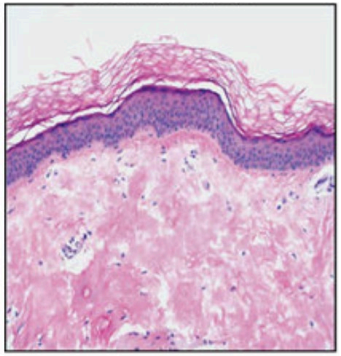
# RPT193: CCR4 Antagonist for Allergic Diseases

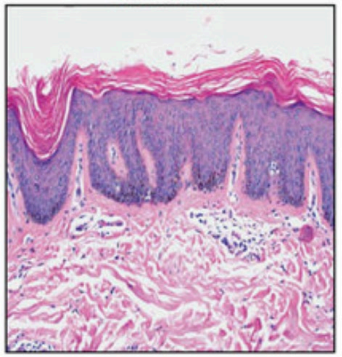
# RPT193: Oral CCR4 Antagonist for Allergic Diseases

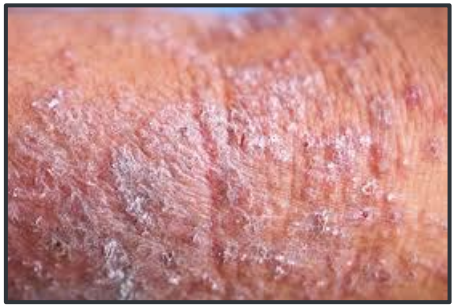
- Targeting atopic dermatitis, asthma, others
- Oral convenience could provide substantial competitive advantage to injectables and topical agents
- Preclinical studies and healthy volunteer data suggest an excellent safety profile
- Phase 1b trial ongoing in atopic dermatitis patients with PoC readout by YE 2020

Normal Human Skin

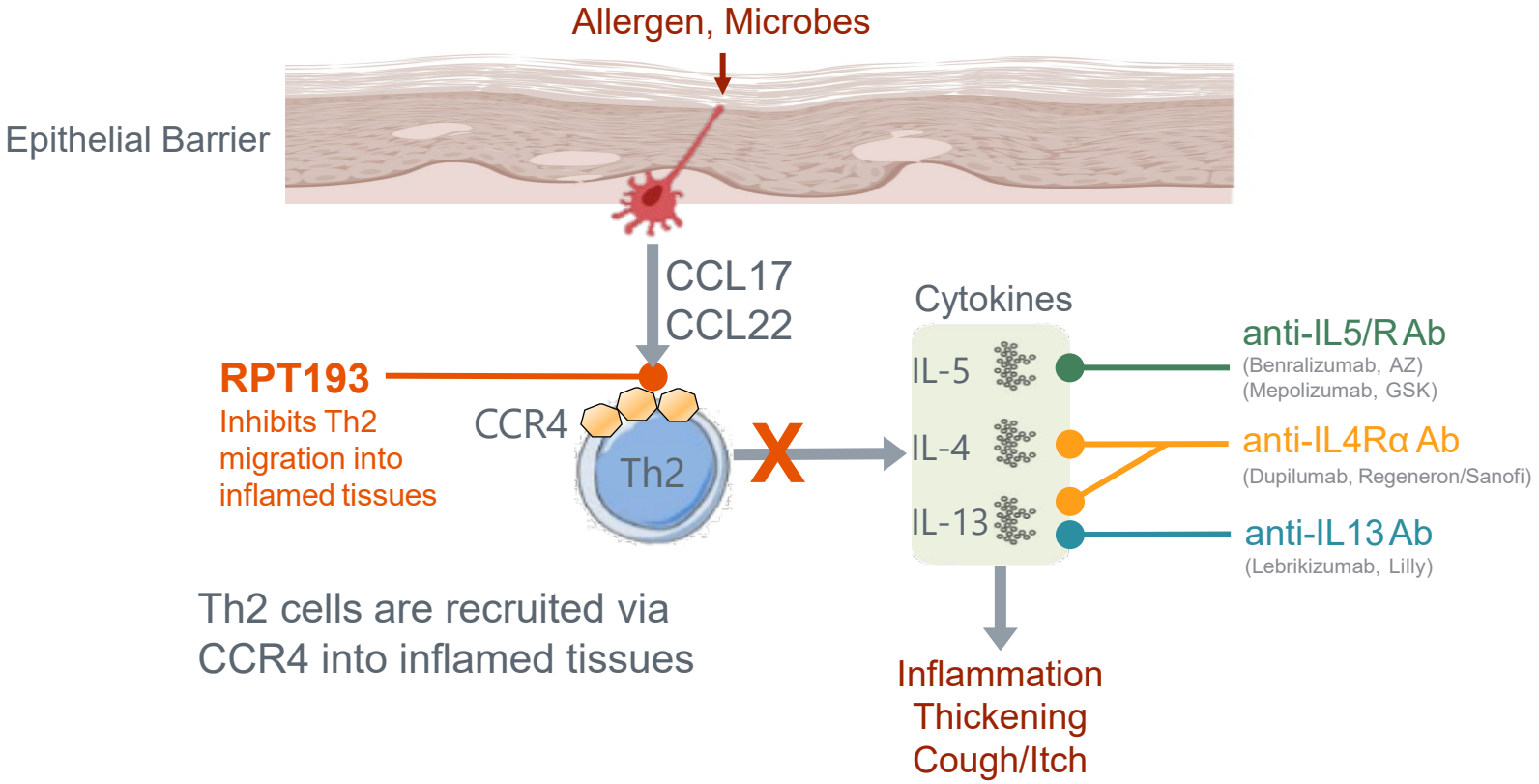


AD Lesional Skin





# RPT193 Acts on the Well Validated Th2 Pathway in Allergic Inflammation



# RPT193 Potential Advantages

| | RPT193 | Dupilumab* | JAK inhibitors |
|---|---|---|---|
| **Safety** | ● Preclinical and healthy volunteer data show an excellent safety profile | ● Generally safe and well tolerated<br>● Conjunctivitis | ● Immunosuppressive<br>● 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<br>● Activity in AD and asthma | ● Similar to dupilumab* |

* DUPIXENT®

● Favorable Characteristic
● Unfavorable Characteristic



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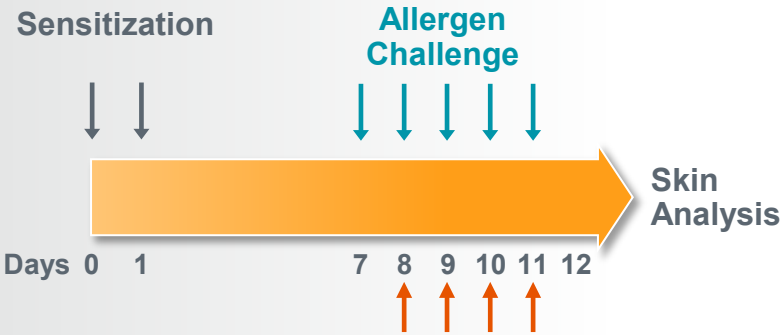
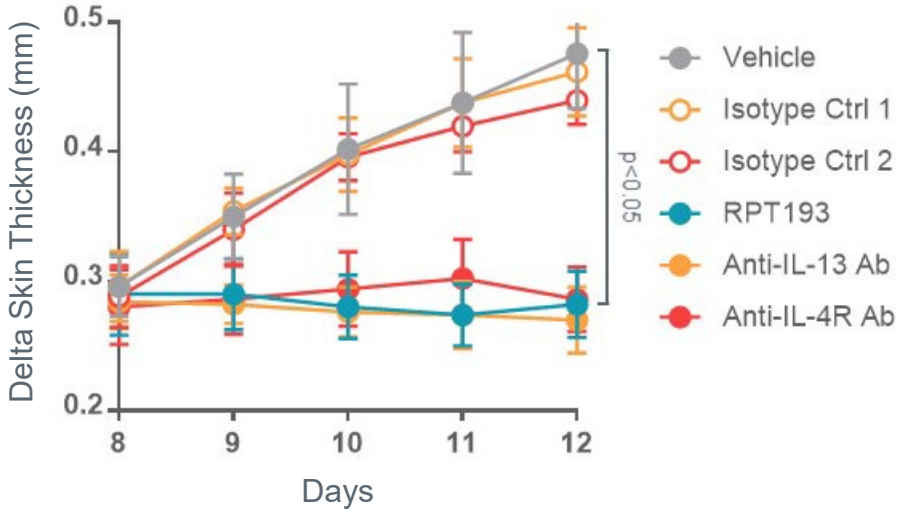
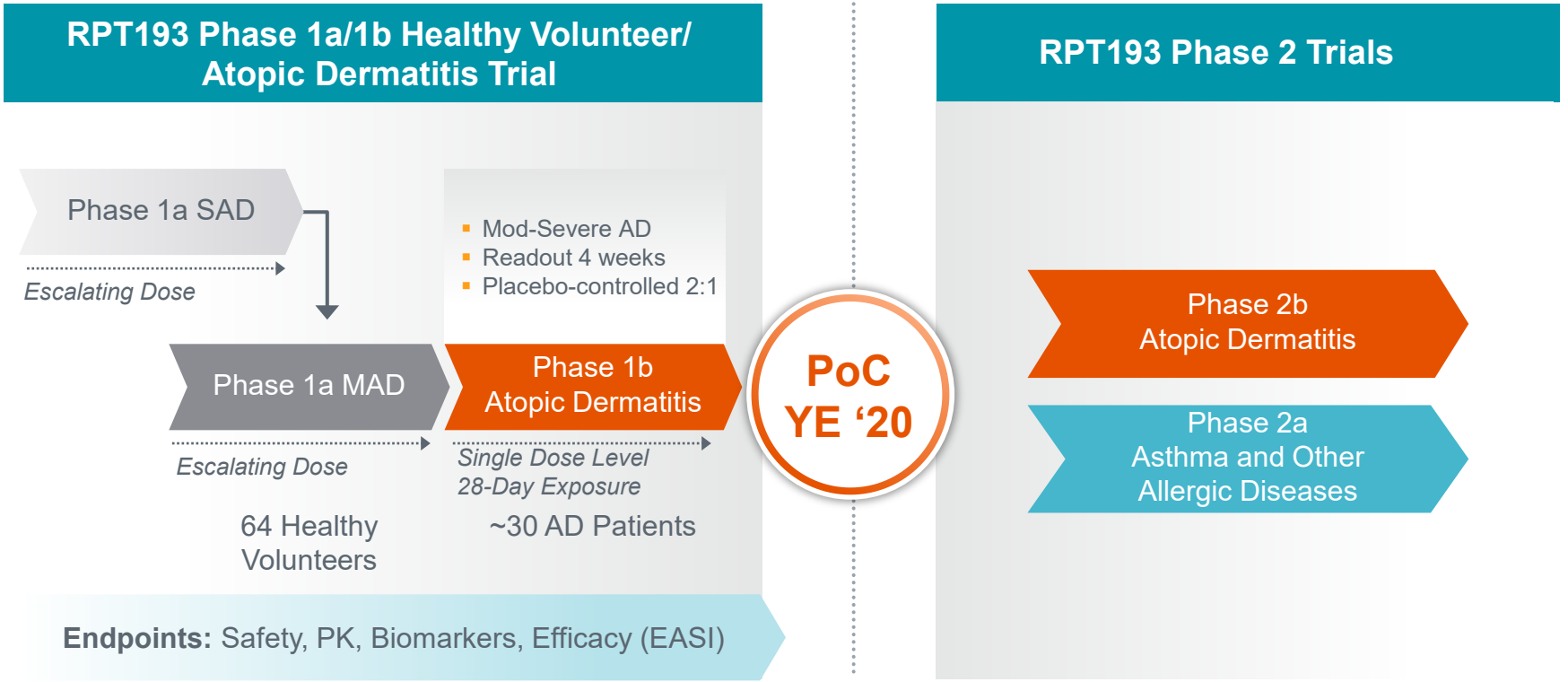
# RPT193 Reduces Skin Inflammation in a Therapeutic Th2-Driven Atopic Dermatitis Model



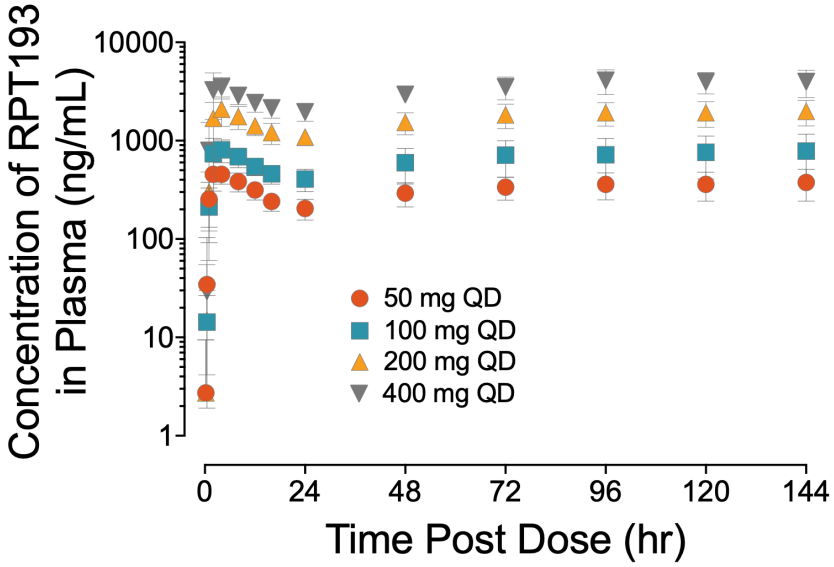



# RPT193: Seamless Clinical Trial Design to PoC and Beyond



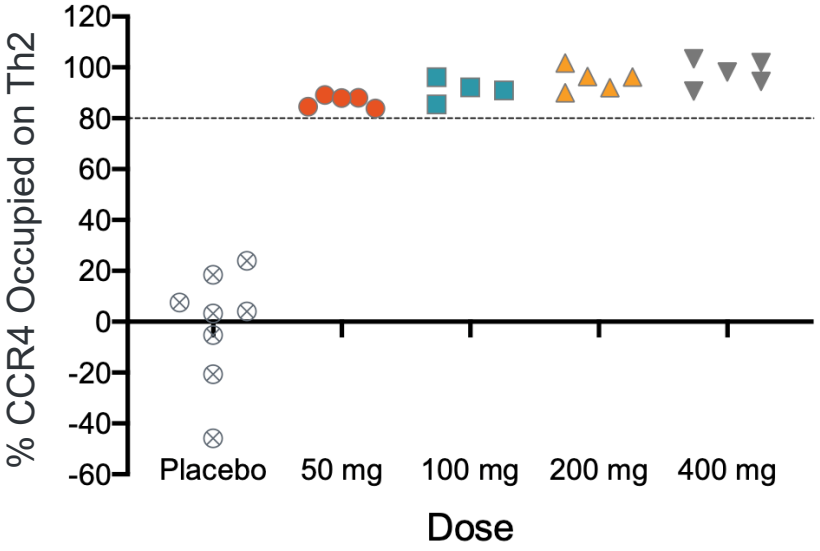
# Phase 1a HV Data Supports Once-Daily Dose

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



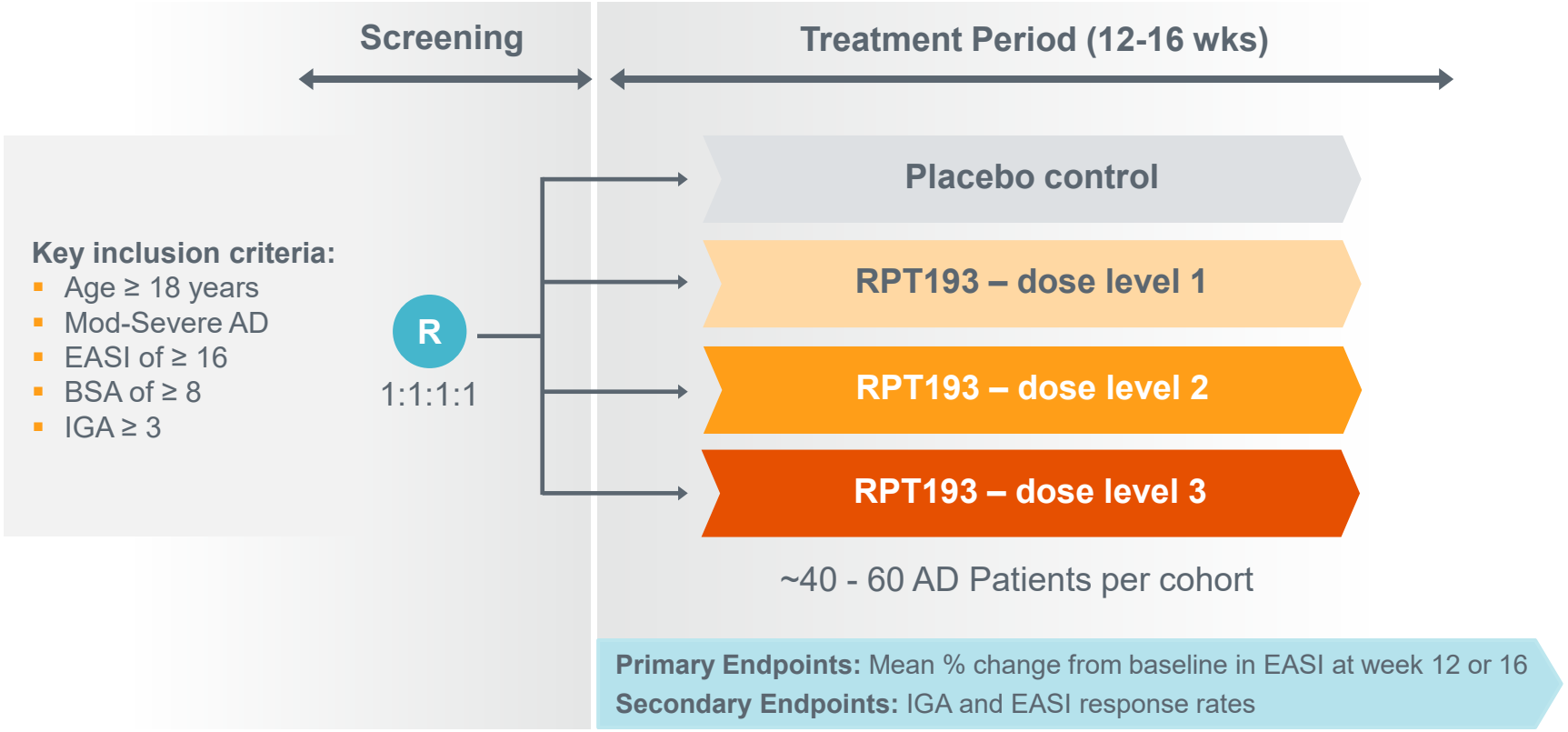

## Targeted Level of CCR4 Inhibition Exceeded

Day 8 trough after last dose

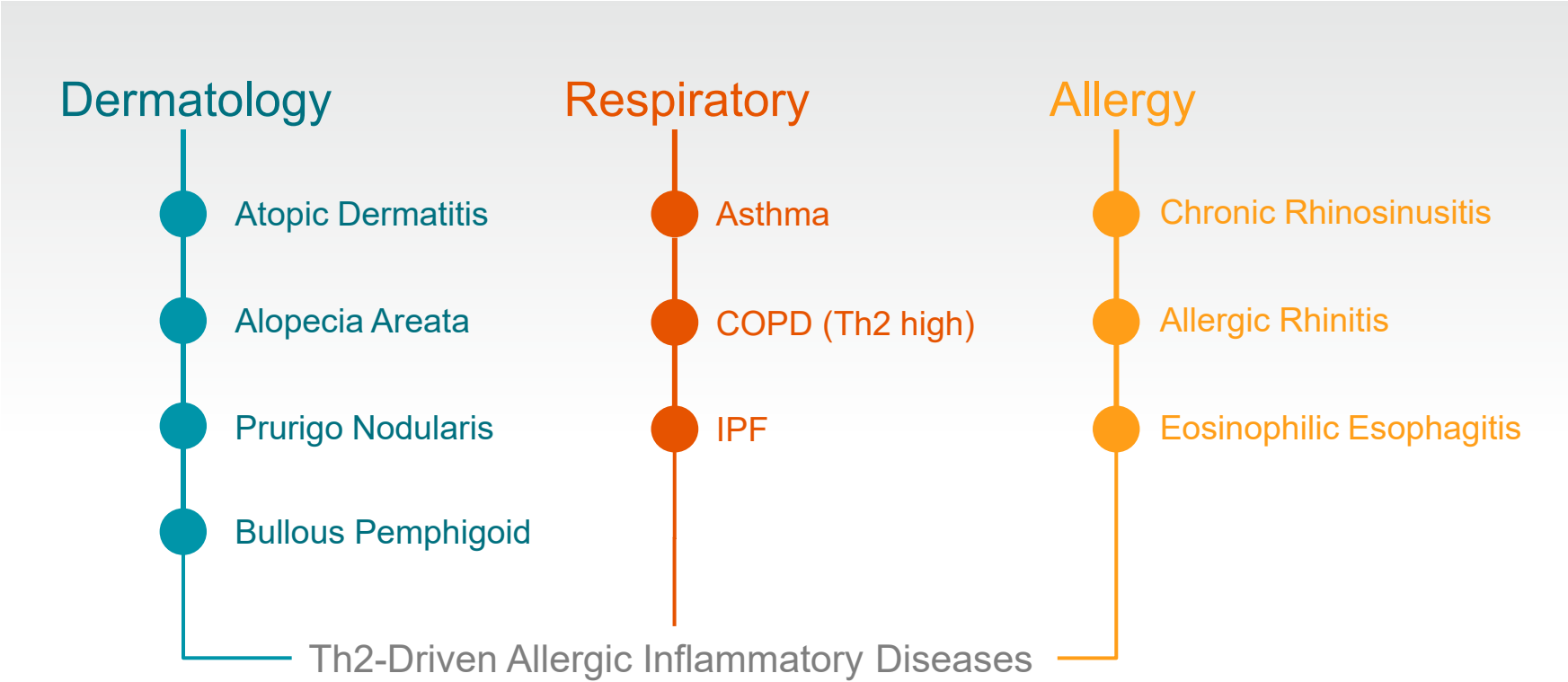



- Excellent safety and tolerability profile (blinded)

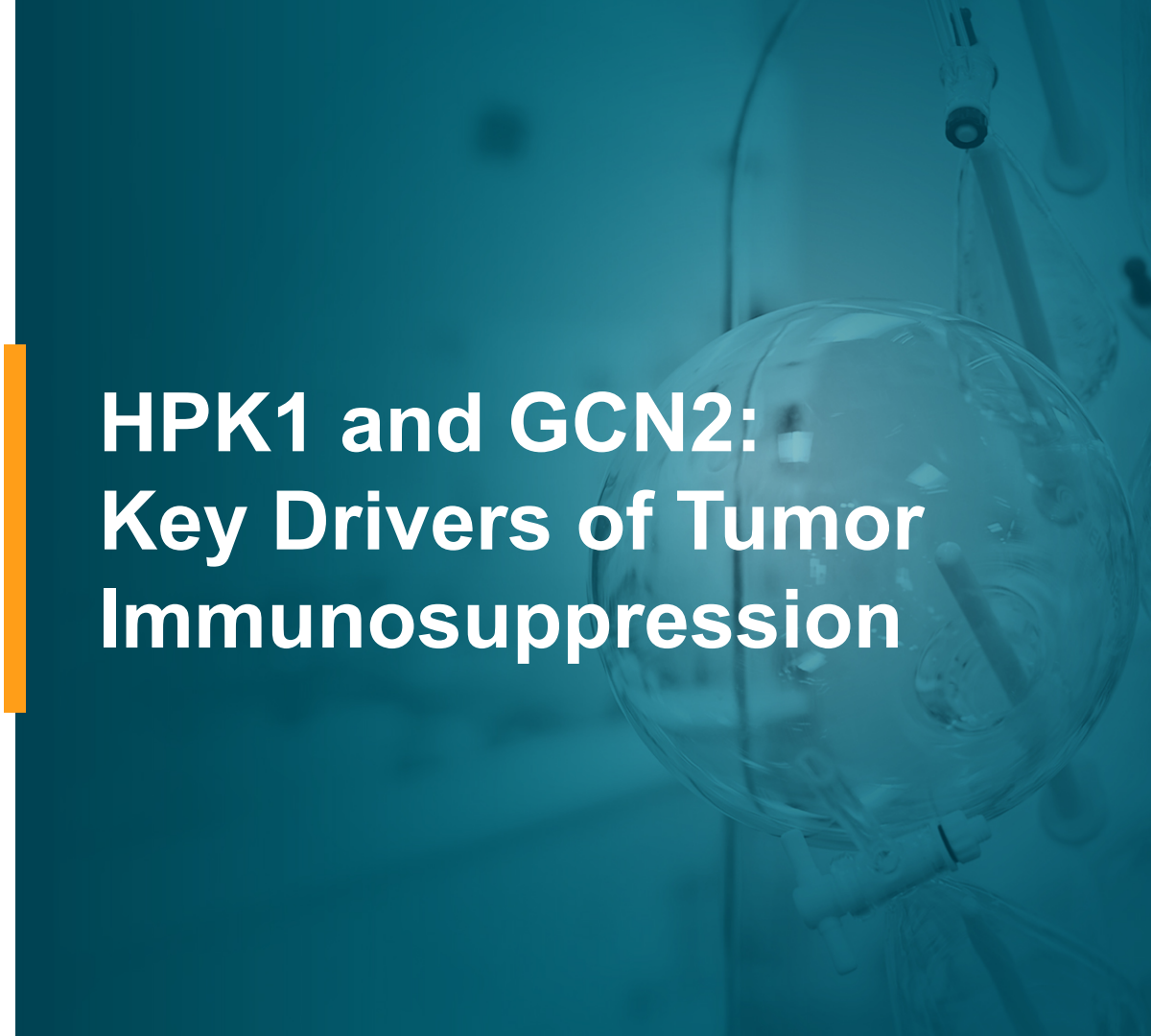
# Proposed Phase 2b Double Blind, Placebo-Controlled Trial



# Potential “Pipeline in a Product”

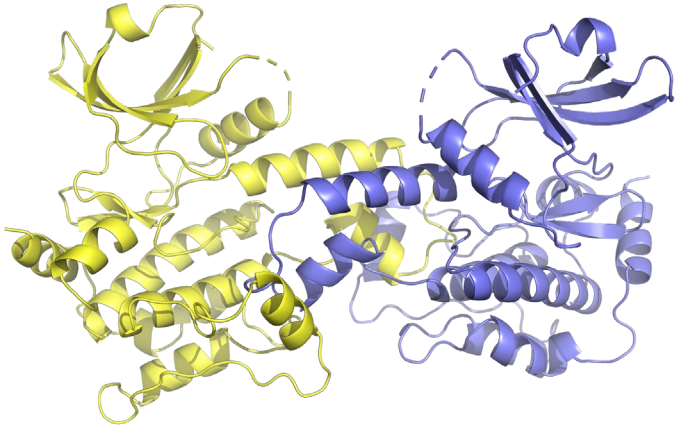


# HPK1 and GCN2: Key Drivers of Tumor Immunosuppression



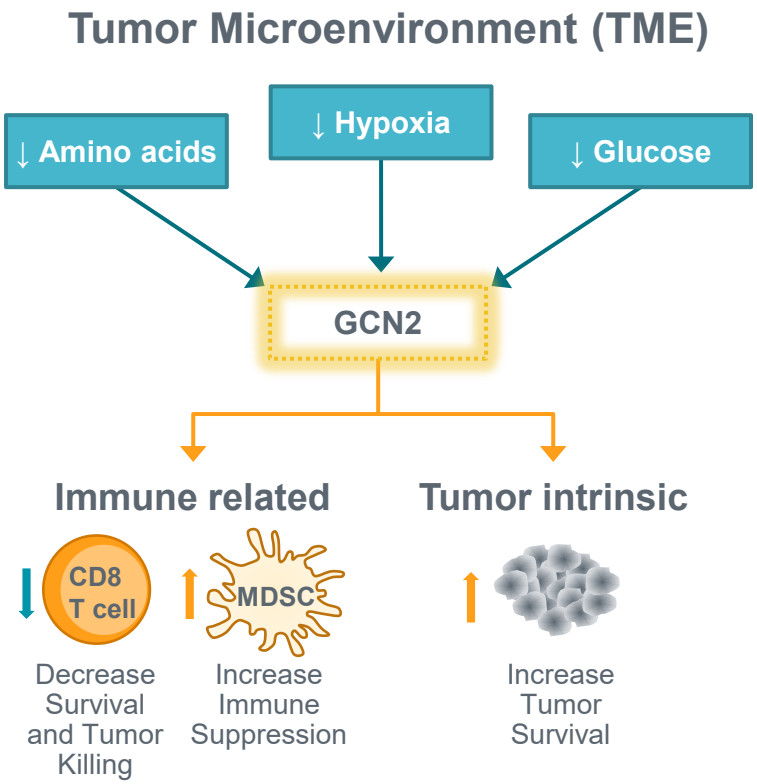
# HPK1 Program: Unlocking Antitumor T Cells

- 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

# Focused on Oral Drugs Targeting Critical Immune Drivers of Disease

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**DISCOVERY**

**HPK1** (Oncology):

- Unlocks T cell activation to tumor antigens

**GCN2** (Oncology):

- Turns on an antitumor metabolic switch in TME



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# Thank You