UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): November 10, 2026

Acrivon Therapeutics, Inc.

(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-41551**	**82-5125532**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

480 Arsenal Way Suite 100 Watertown, Massachusetts	**02472**
(Address of Principal Executive Offices)	**(Zip Code)**

Registrant's Telephone Number, Including Area Code: (617) 207-8979

(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, par value $0.001 per share	ACRV	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

On November 10, 2026, Acrivon Therapeutics, Inc. (the "Company") hosted a webcast corporate R&D event with Eosinophilic esophagitis (EoE) Key Opinion Leaders to discuss Acrivon's new immunology and inflammation (I&I) programs, acquired using its AI-driven AP3 platform. The webcast took place from approximately 4:00 p.m. to 5:00 p.m. ET.

The agenda featured a presentation by the Company's leadership team introducing the ACR-525 and PKCθ inhibitor preclinical programs and initial development plans in EoE, followed by an overview of EoE epidemiology and unmet need by Dr. Arjan Bredenoord (principal investigator and professor, Gastroenterology and Hepatology, Amsterdam University Medical Center) , a review of EoE standard of care by Dr. Evan Dellon (professor, Department of Medicine, Division of Gastroenterology and Hepatology and Director, Center for Esophageal Diseases and Swallowing, School of Medicine, University of North Carolina at Chapel Hill), and an interactive Q&A session and discussion.

In connection with this event, the Company posted to the "Investors & Media" section of the Company's website at ir.acrivon.com, the presentation used in the event (the "Presentation").

A copy of the Presentation is attached hereto as Exhibit 99.1 and is incorporated by reference into this Item 7.01 of this Current Report on Form 8-K.

The information in this Item 7.01, including Exhibit 99.1 attached hereto, shall not be deemed "filed" for purposes of Section 18 of the Exchange Act, or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits.

Exhibit Number	Description
99.1	Acrivon Therapeutics, Inc., Presentation
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Acrivon Therapeutics, Inc.

Date: November 10, 2026

By: /s/ Peter Blume-Jensen, M.D., Ph.D.
Name: Peter Blume-Jensen, M.D., Ph.D.
Title: Chief Executive Officer and President

Exhibit 99.1

Acrivon Therapeutics

CORPORATE R&D AND KOL EVENT

ACRIVON'S NEW I&I PROGRAMS

DIFFERENTIATED ASSETS IDENTIFIED WITH PROPRIETARY AP3 PLATFORM

NOVEMBER 10, 2026

FORWARD-LOOKING STATEMENTS

Certain information contained in this presentation includes forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995 regarding our future results of operations or financial condition, business strategy and plans and objectives of management for future operations. In some cases, you can identify forward-looking statements because they contain words such as "anticipate," "believe," "contemplate," "continue," "could," "estimate," "expect," "intend," "may," "plan," "potential," "predict," "project," "should," "target," "will," or "would" or the negative of these words or other similar terms or expressions. Our forward-looking statements are based primarily on our current expectations and projections about future events and trends that we believe may affect our business, financial condition and results of operations. The outcome of the events described in the forward-looking statements is subject to risks and uncertainties, including the factors described in our filings with the U.S. Securities and Exchange Commission. New risks and uncertainties emerge from time to time, and it is not possible for us to predict all risks and uncertainties that could have an impact on the forward-looking statements contained in this presentation. The results, events, and circumstances reflected in the forward-looking statements may not be achieved or occur, and actual results, events, or circumstances could differ materially from those described in the forward-looking statements.

You are cautioned not to place undue reliance on these forward-looking statements, which are made only as of the date of this presentation. We undertake no obligation to update any forward-looking statements or to reflect new information or the occurrence of unanticipated events, except as required by law.

Note: Acrivon intends to use its website as a means of disclosing material non-public information and for complying with its disclosure obligations under Regulation FD. For more information, please visit www.acrivon.com and follow the company on LinkedIn.

TODAY'S AGENDA

Welcome	Adam Levy, PhD, MBA, *CFO*
Introduction and Overview	Peter Blume-Jensen, MD, PhD, *CEO*
EoE epidemiology and unmet need	Arjan Bredenoord, MD, PhD
EoE SOC (ACG Guidelines)	Evan Dellon, MD, MPH
Q&A and Discussion	Mansoor Mirza, MD, *CMO*
Adjourn	Peter Blume-Jensen, MD, PhD, *CEO*

TODAY'S PARTICIPANTS



Arjan Bredenoord, M.D., Ph.D.

Professor, Gastroenterology and Hepatology, Amsterdam University Medical Center; Former President of the European Society of Eosinophilic Esophagitis EUREOS, a member of the UEG Scientific Committee



Evan Dellon, M.D., M.P.H.

Professor, Department of Medicine, Division of Gastroenterology and Hepatology; Director, Center for Esophageal Diseases and Swallowing, School of Medicine, University of North Carolina at Chapel Hill



Peter Blume-Jensen, M.D., Ph.D.

Chief Executive Officer, President and Co-Founder, Acrivon Therapeutics; Formerly Executive at Serono, Merck & Co., Daiichi Sankyo, Metamark Genetics; Inventor of the AP3 Platform



Mansoor Raza Mirza, M.D.

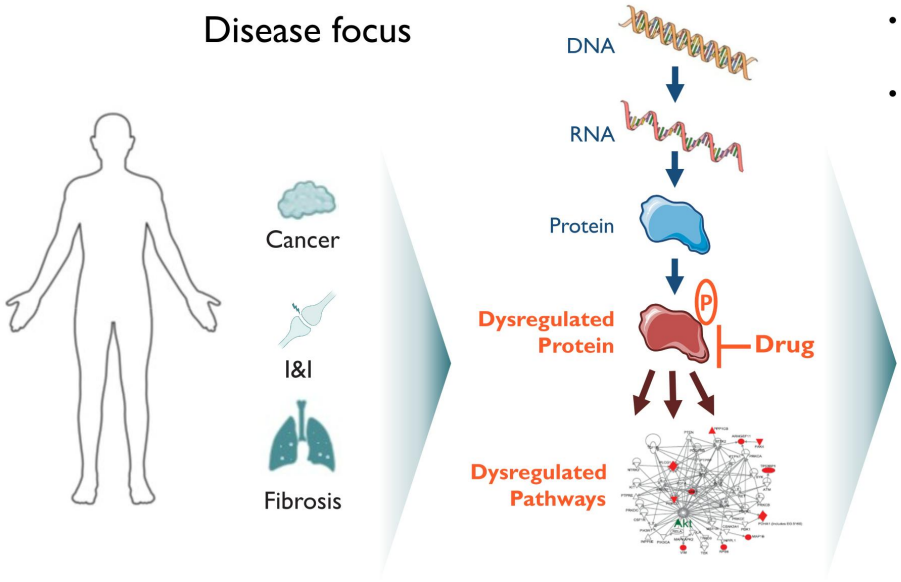
Chief Medical Officer, Acrivon Therapeutics; Formerly Chief Oncologist at Copenhagen University Medical School; Lead PI for numerous guideline-changing regulatory approvals



Erick Gamelin, M.D., Ph.D.

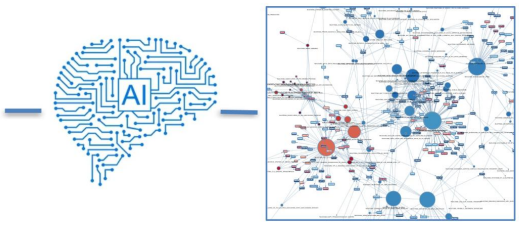
Chief Development Officer, Acrivon Therapeutics; Formerly CEO for national cancer center; Executive at Pfizer, AMGEN, Dynavax, Macrogenics; Chevalier in the National Order of Merit by the French Minister of Health

NEXT-GENERATION PRECISION MEDICINE POWERED BY AP3



Acrivon Predictive Precision Proteomics (AP3)

- Generative AI-driven, proteomics-based rational drug design and informed clinical development
- AP3 enables an exact match between the disease-driving, dysregulated pathways with drug mechanism of action (Acrivon meaning ≈ exact, accurate)



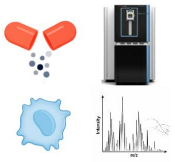
Proprietary tools
- Data portal
- Interactome
- Protein activity predictor

AP3 GENERATIVE AI PHOSPHOPROTEOMICS PLATFORM

MATCHING GLOBAL DRUG EFFECTS WITH DISEASE DRIVING MECHANISMS

Comprehensive Profiling of Drug Effects

Global MS Phosphoproteomic Profiling



Biological Multimodal Data

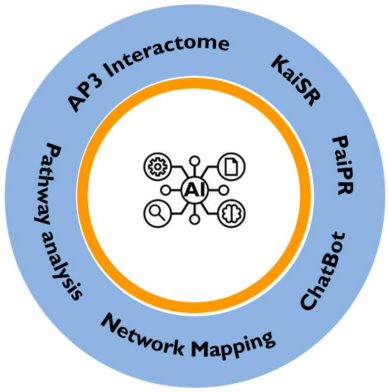


AP3 Data Portal



- Structured
- AI-enabled

Acrivon Proprietary Generative AI-driven tools

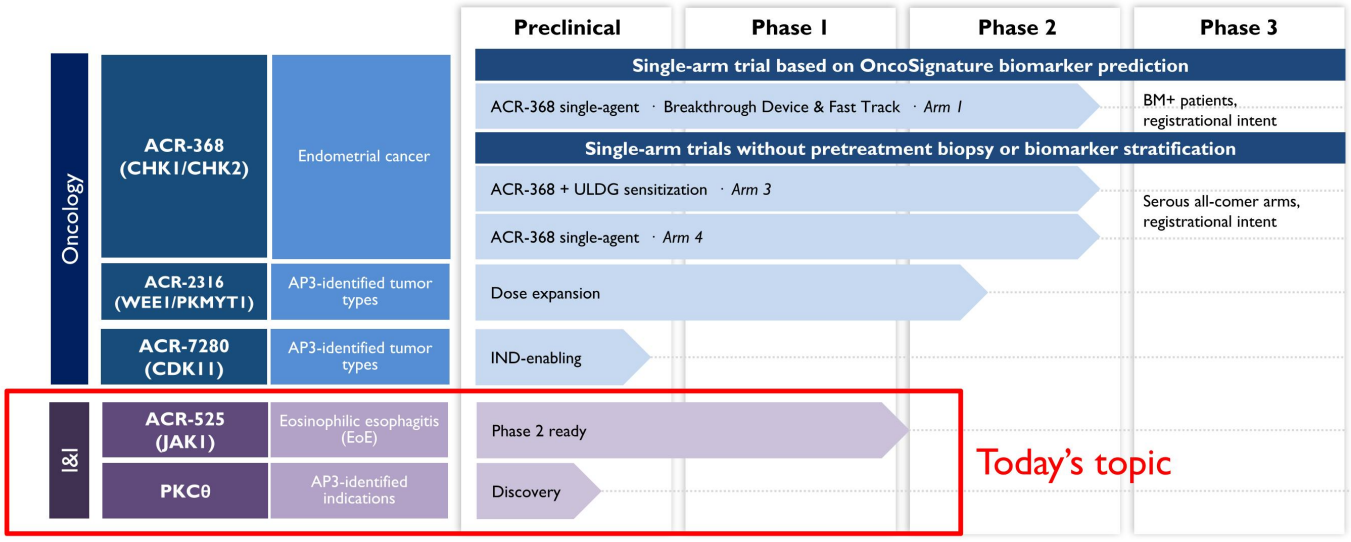


AP3 Deliverables

Streamlined Clinical Development

- Patient responder prediction
- **Indication Finding**
- **Drug combinations**
- Target ID
- Drug Resistance
- **Global PD markers**
- **Asset prioritization**

OUR PIPELINE



FIRST AP3-BASED I&I PROGRAM: PHASE 2-READY, ORAL, SELECTIVE JAK1 INHIBITOR, ACR-525 (ALSO KNOWN AS AQ280)

AP3 profiling

Extensive AP3 profiling against approved JAK inhibitors and dupilumab

Desirable global phospho-proteomic pathway effects

Differentiated profile

Highly JAK1-selective, oral

Desirable suppression of TGFβ signaling (anti-fibrotic)

No obvious pro-apoptotic BAD activation (potential safety advantage)

Broader anti-inflammatory effects beyond IL-4 and IL-13 signaling than dupilumab

Phase 1 clinical data

Clinical PD/PK data support once daily oral dosing based on preclinical PD/PK/efficacy studies

Favorable initial safety and dose-dependent PD (CXCL10) decrease

Development path

Phase 2-ready

Initial development in eosinophilic esophagitis (EoE), but broader I&I opportunity

Strong combo rationale with IL-23i, TNFi, PKC- Θi

Wholly-owned, no milestone or royalty obligations
CoM protection: 2038 (with extension opportunities)

ACR-525 COMPARES FAVORABLY TO CLINICAL BENCHMARKS

Clinical comparators

ACR-525

Upadacitinib
Filgotinib
Abrocitinib
Tofacitinib
Brepocitinib
Londamocitinib

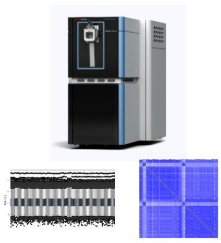
Dupilumab
IL-4Rα antibody

Disease-relevant cellular models



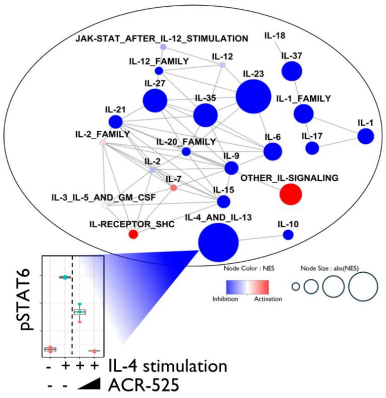
- IFNα-stimulated
- Th2-polarized (TCR, IL-4)

AP3 phosphoproteomics



Tight, robust, structured data

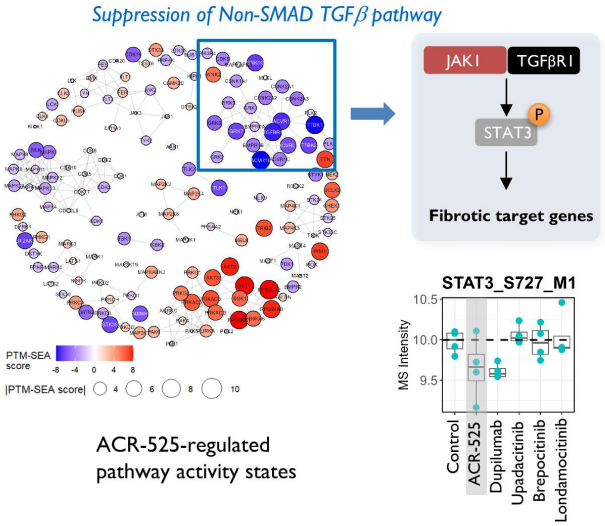
Generative AI-enabled pathway insights



ACR-525 potently reduces inflammatory cytokine signaling activity

AP3 pathway mapping shows differentiated, high selectivity against JAK2, JAK3, and TYK2 signaling, suggesting potential for improved efficacy and safety over existing JAK inhibitors

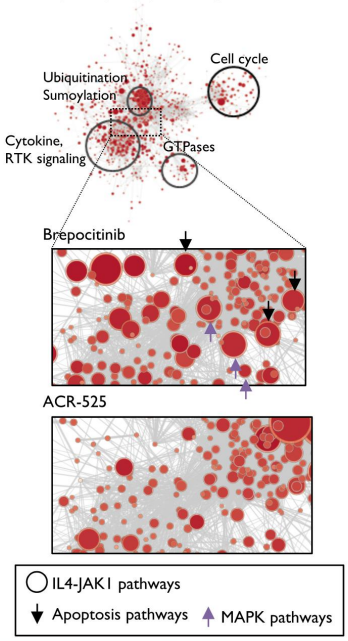
GENERATIVE AI-DRIVEN AP3 ANALYSES UNCOVER TGF β PATHWAY SUPPRESSION BY ACR-525, A POTENTIAL ANTIFIBROTIC MECHANISM



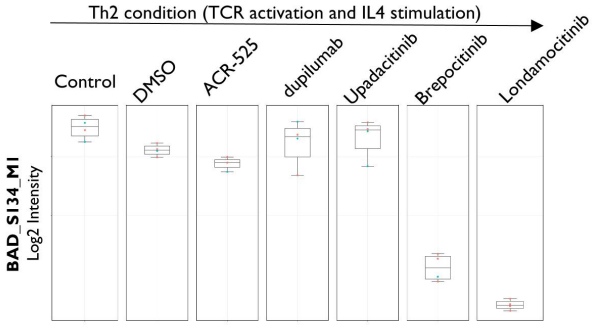
- AP3 reveals ACR-525-induced inhibition of non-SMAD TGF β signaling activity
- JAK1-regulated non-SMAD TGF β signaling is reported to drive fibrotic processes
- ACR-525 (and dupilumab), unlike other JAK inhibitors, inhibit STAT3 pS727, a marker of non-canonical TGF β pathway activity

ACR-525 DOES NOT ACTIVATE PRO-APOPTOTIC BAD SIGNALING, SUGGESTING A POTENTIAL SAFETY ADVANTAGE

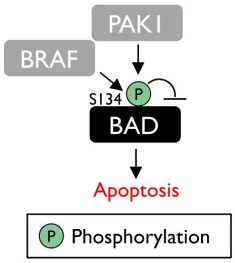
JAK1 inhibitor engaged phospho-pathway network



Differential JAK1 inhibitor regulation of BAD S134 phosphorylation



Decreased BAD-S134 phosphorylation can lead to pro-apoptotic cell death



- AP3 pathway reconstitution shows differential engagement of apoptosis pathways by brepocitinib and londamocitinib compared to ACR-525
- Brepocitinib and londamocitinib uniquely suppress phosphorylation of BAD S134, releasing its pro-apoptotic function, a potential rhabdomyolytic liability

ACR-525 IS POTENT AND HIGHLY SELECTIVE FOR JAK1

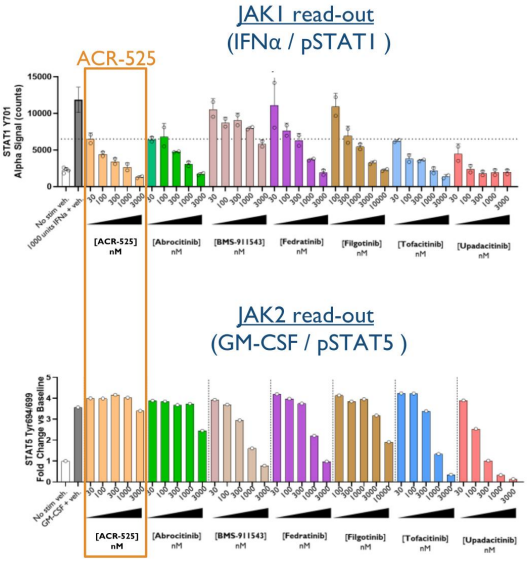
COMPARING FAVORABLY TO OTHER JAK INHIBITORS

Molecule	Relative potency (biochemical IC_{50}) JAK1/JAK2	Relative potency (cellular IC_{50}) JAK1/JAK2 (right panel)
ACR-525	49× JAK1: 3.5 JAK2: 171	>120× IFNα ~24 GM-CSF >3,000
Abrocitinib	28× JAK1: 29.2 JAK2: 803	>90× IFNα ~34 GM-CSF >3,000
BMS-911543	0.01× JAK1: 75 JAK2: 1.1	0.40× IFNα ~1,300 GM-CSF ~540
Fedratinib	0.03× JAK1: 105 JAK2: 3	6× IFNα ~170 GM-CSF ~980
Filgotinib	~1× JAK1: 10–53 JAK2: 28–29	19× IFNα ~400 GM-CSF ~7,500
Tofacitinib	1.3× JAK1: 3.2 JAK2: 4.1	30× IFNα ~20 GM-CSF ~610
Upadacitinib	2.8× JAK1: 43 JAK2: 120	>4× IFNα <30 GM-CSF ~110
Brepocitinib*	4.6× JAK1: 16.8 JAK2: 76.6	24× IFNα 85 GM-CSF 1,995

Grey values in nM. Ratios >1 favor JAK1-selective inhibition.

*Brepocitinib: Public data only

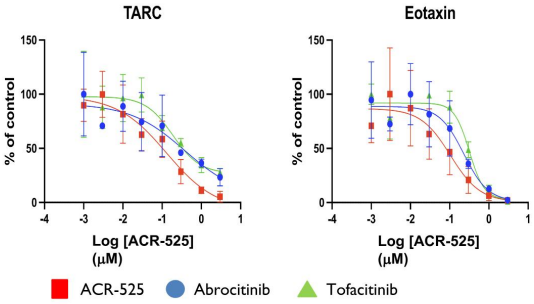
Cellular pathway engagement



ACR-525 DOSE-DEPENDENTLY REDUCES TH2-RELEVANT DISEASE MARKERS IN PRECLINICAL AND CLINICAL STUDIES

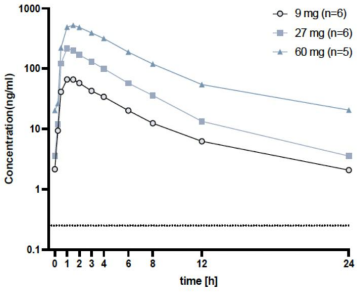
Preclinical

- ACR-525 reduces pSTAT3 in Th2 allergic inflammation in vivo mouse model
- ACR-525 reduces EoE Th2 inflammation markers (*ex vivo* stimulated human donor whole blood)

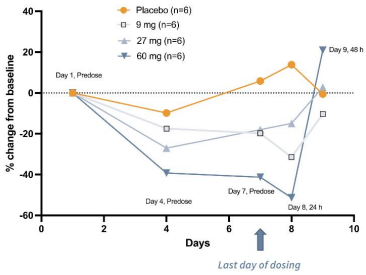


Clinical

- Dose-proportional PK (Phase 1)



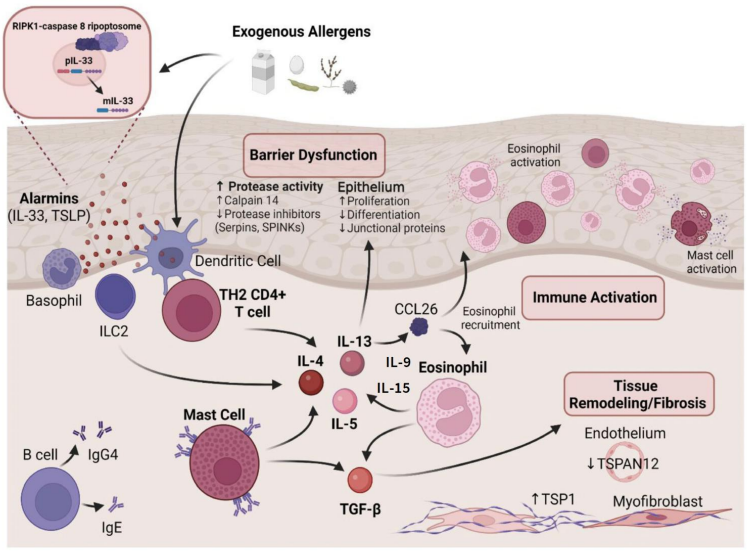
- ACR-525 reduces the inflammation marker CXCL10 (Phase 1 MAD)



Summary of Phase 1 healthy volunteer studies

- PD biomarker changes consistent with JAK1-selective inhibition
- Limited effect on reticulocytes (consistent with minimal JAK2 inhibition)
- Dose-proportional, predictable PK (T1/2 ~12h)
- Safe and tolerable – no SAEs

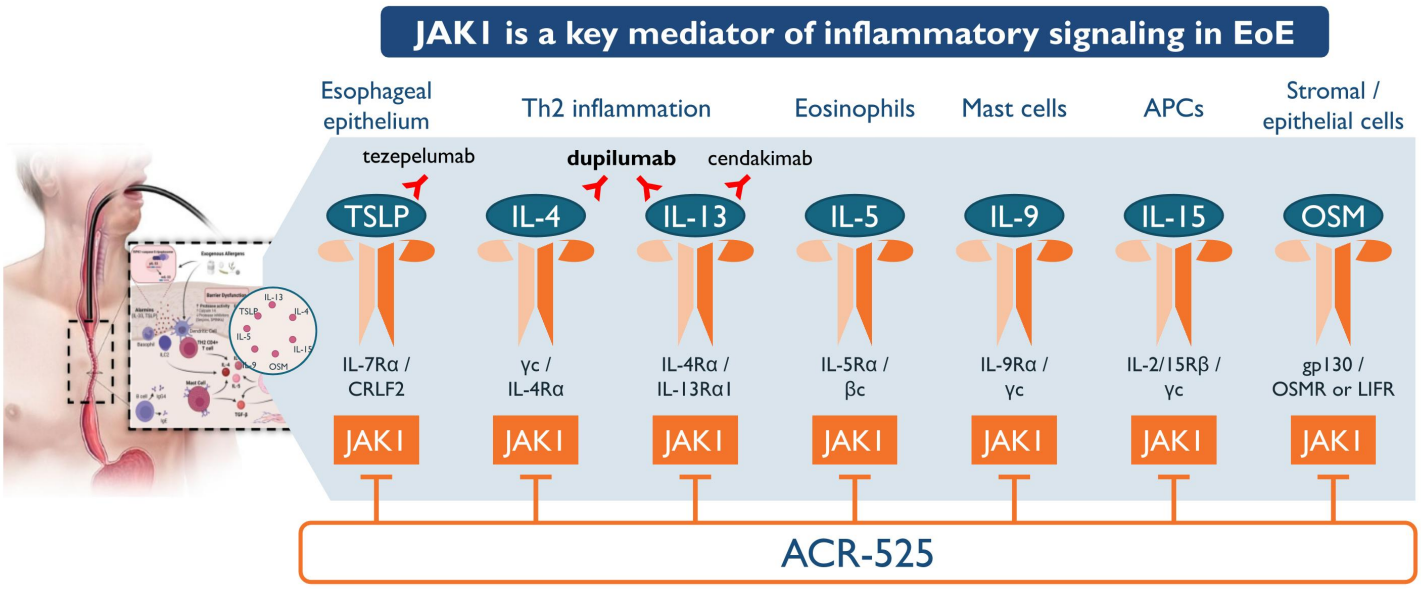
JAK1 PLAYS A CENTRAL ROLE IN E oE



Modified from: B.Underwood et.al, Annals of Allergy, Asthma & Immunology, January 2023

- Key cytokines IL-4 and IL-13 signal via JAK1-STAT6
- Several critical Single Nucleotide Polymorphisms (SNPs) within the JAK-STAT signaling pathway in EoE
- JAK inhibitors are effective for atopic dermatitis
- Case reports show efficacy of JAK inhibition in EoE

EoE, AN ATTRACTIVE FIRST INDICATION FOR ACR-525



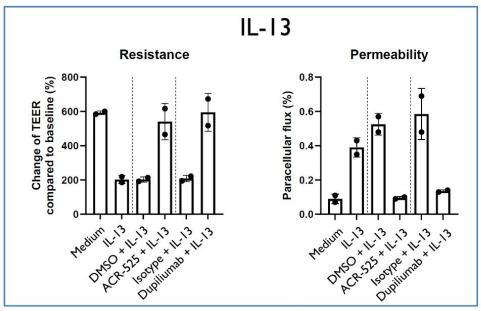
- Dupilumab (approved) targets only IL-4 and IL-13
- Tezepelumab and cendakimab (Phase 3) target only TSLP and IL-13, respectively

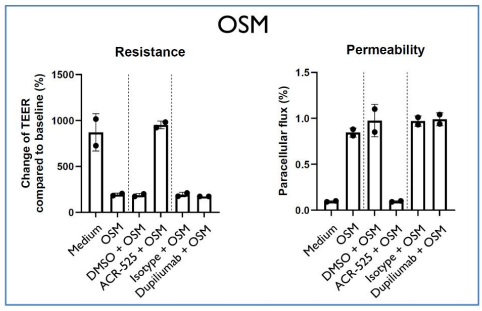
ACR-525 PREVENTS IL-13 AND OSM-INDUCED EPITHELIAL BARRIER DAMAGE

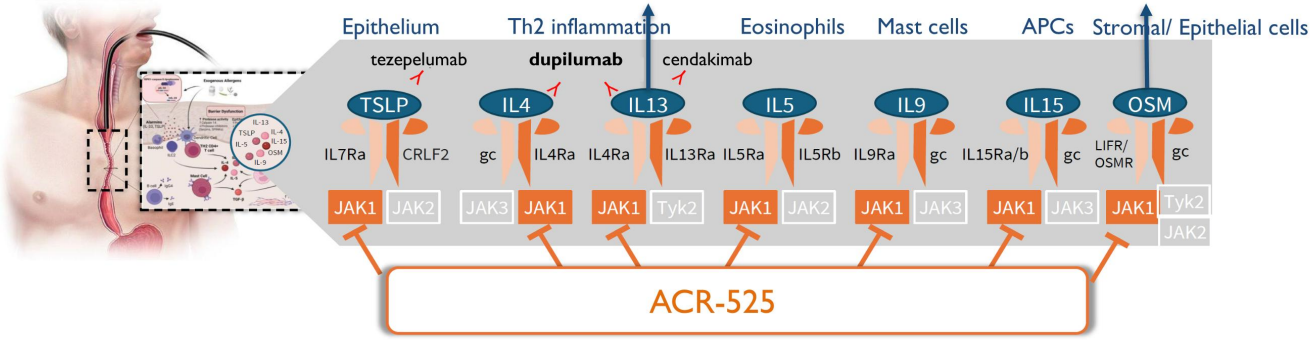
BROADER ACTIVITY THAN DUPILUMAB, WHICH FAILS TO PREVENT OSM DAMAGE

EoE model

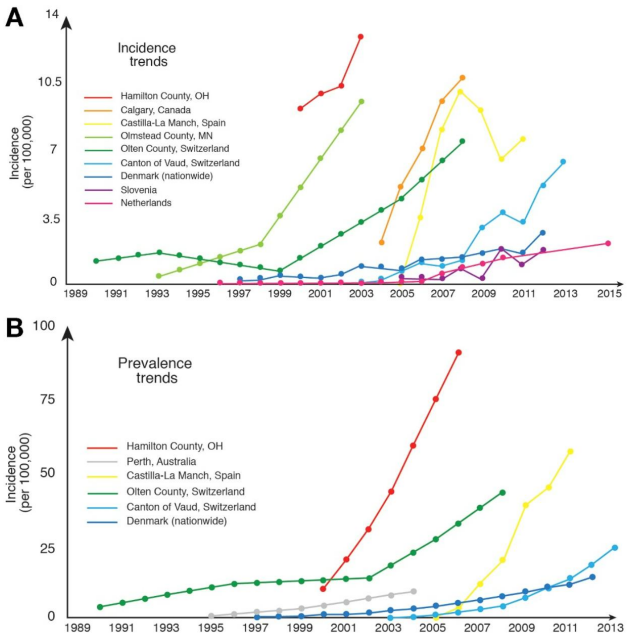
Kleuskens et al.
Digestive Diseases Week 2026

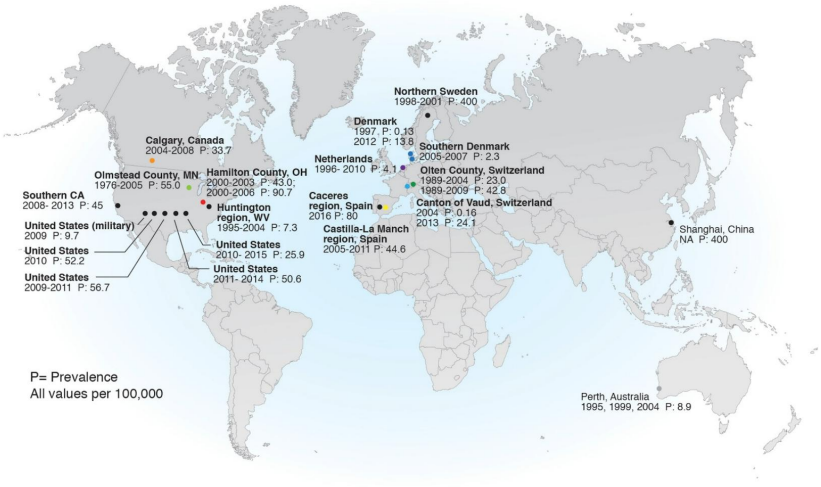






HUGE INCREASE IN EoE OVER THE PAST 25 YEARS





Dellon and Hirano, Gastroenterology 2018

EoE – A RAPIDLY GROWING PATIENT POPULATION OF UNMET NEED

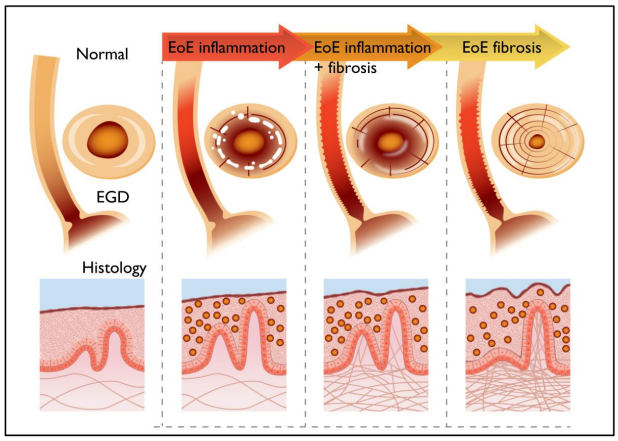
- **Annual Incidence (US): ~120,000**
- **Current prevalence (US): >500,000**

Growth Metric	US Estimate
Prevalence in 2009	28.3/100,000
Prevalence in 2022	163.1/100,000
Total increase over 13 years	***+477%***
Average annual growth	***~14.4%***
Equivalent growth every 5 years	***~96%***
Multiplication over 13 years	5.8×

- *Greenhawt M, Bansal P, Goodwin B, et al. (2025) Journal of Allergy and Clinical Immunology*
- *Thel HL, Anderson C, Xue AZ, Jensen ET, Dellon ES. (2025)Clinical Gastroenterology and Hepatology.*

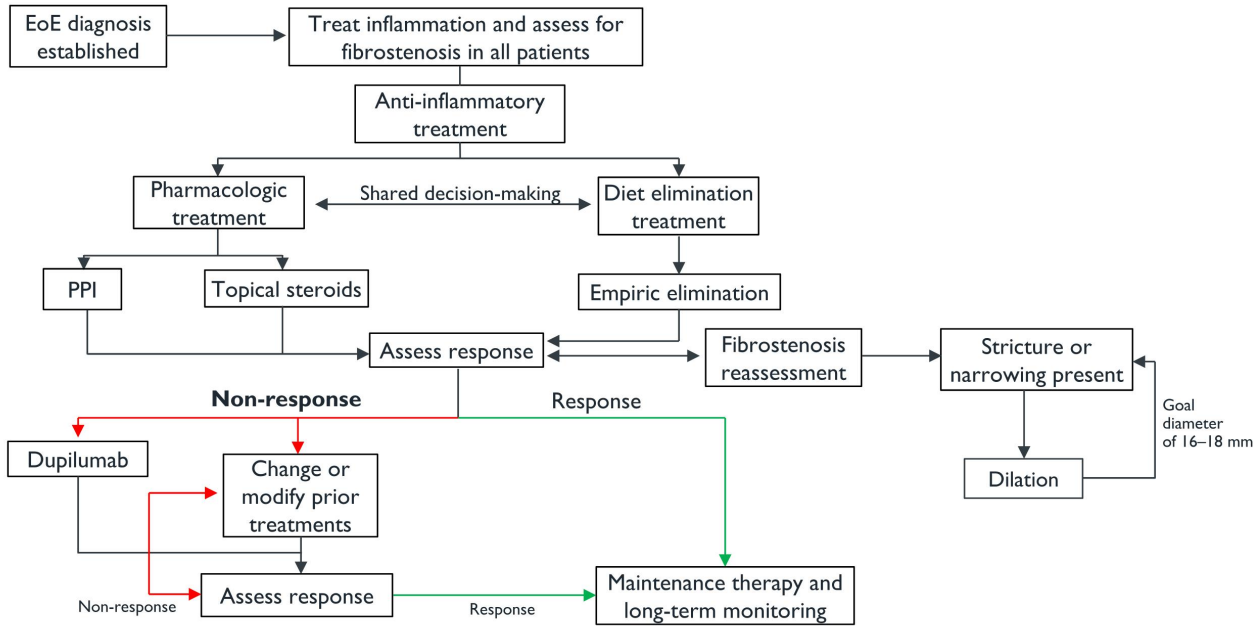
EOE BURDEN: SYMPTOMS AND CHRONIC THERAPY

- EoE leads to esophageal obstruction: inability to pass solid foods
- EoE leads to a profound loss in health-related quality of life
- EoE is chronic and leads to fibrostenosis
- Requires long-term treatment for symptom control and prevention of esophageal scarring



Dellon & Hirano Gastroenterology 2018;
Bredenoord et al. Am J Gastroenterol 2022

EOE TREATMENT ALGORITHM – ACG GUIDELINES 2025



Dellon et al; AJG; 2025

UNMET NEED IN EOE – NEW TREATMENTS ARE STILL NEEDED

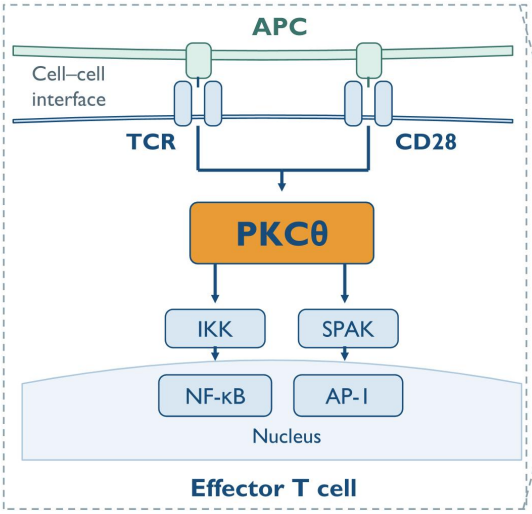
- First line therapies:
  - Suboptimal responses (>50% may be non-responders)
  - Adherence difficulties (twice daily dosing; restrictive diets)
  - Concern for side effects
  - Loss of response is frequently observed
- Biologics
  - Currently position as "step-up" and second line agents
  - Require frequent injections
- There are no advanced small molecule orally-available medications at either the first or second line for EoE treatments

A highly specific, oral JAK1 inhibitor, if proven to be safe and effective, would fill an important treatment gap and unmet need

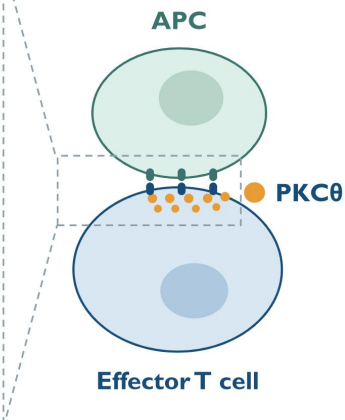
PRECLINICAL PKCΘ PROGRAM

PKCΘ PLAYS CRITICAL ROLE IN T CELL ACTIVATION

T cell signaling



APC - T cell synapse



PKCθ recruited to immune synapse to mediate T cell activation

Key role in T-cell activation
Induces NF-κB and AP-1 activation

Strong evidence based on extensive preclinical studies
Mouse colitis, allergic airway and human RA studies uncover key role of PKCθ in T cell-mediated inflammation

ACR-525+PKCθi combination rationale
JAK/STAT pathways converge with PKC θ for Th2/Th17 activation

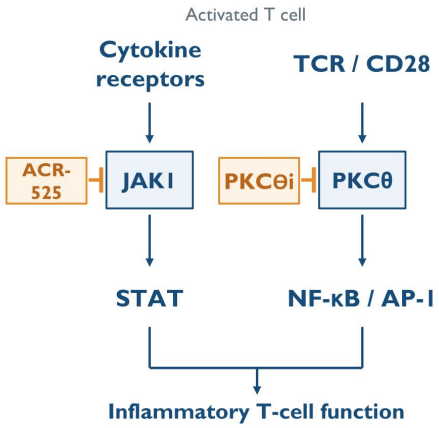
Selected primary studies: Sun, Nature 2000. Marsland, J Exp Med 2004. Nagahama, Gastroenterology 2008. Zanin-Zhorov, Science 2010. Kong, Nat Immunol 2011.

PRECLINICAL PKCΘ PROGRAM
PKCΘ PLAYS CRITICAL ROLE IN T CELL ACTIVATION

Promising PKCθ inhibitor lead series

- ✓ Potent PKCθ inhibition
- ✓ Potent activity in human immune cells
- ✓ High selectivity over PKCδ
- ✓ Low clearance in human hepatocytes

ACR-525 + PKC-θi rational combination



Broad potential:
UC, RA, psoriasis, atopic dermatitis, allergic asthma, SLE

Selected primary studies: Sun, Nature 2000. Marsland, J Exp Med 2004. Nagahama, Gastroenterology 2008. Zanin-Zhorov, Science 2010. Kong, Nat Immunol 2011.



ACR-525 profiled H:H against
6 approved JAK inhibitors and dupilumab

Broader indication potential

JAK1-selectivity + distinct TGFβ pathway suppression

Benchmark differentiation

Selectivity, oral availability, and desirable pathway potency

Safety-related profiling

No pro-apoptotic BAD phosphorylation

EoE targeted as a first indication for ACR-525

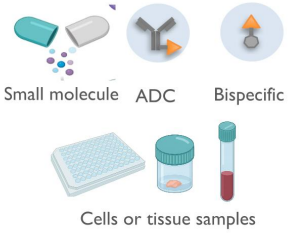
Q&A AND DISCUSSION

APPENDIX

AP3 GENERATIVE AI-DRIVEN PHOSPHOPROTEOMICS PROFILING

GLOBAL, CELLULAR DRUG PATHWAY EFFECTS AND COMPOUND DIFFERENTIATION

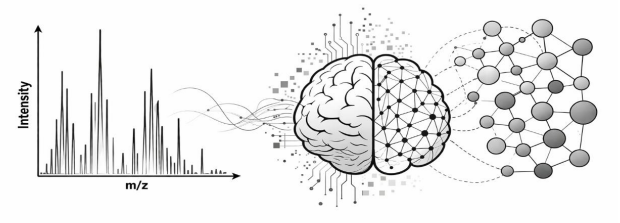
Drug treatment



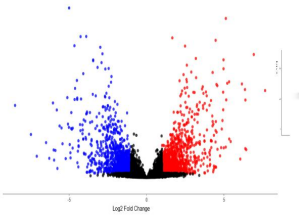
LC-MS/MS profiling



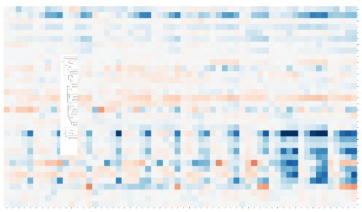
AP3 Generative AI-driven phosphoproteomics analytics



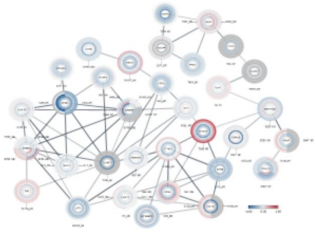
Drug-regulated phosphosites



Drug-regulated kinase activity



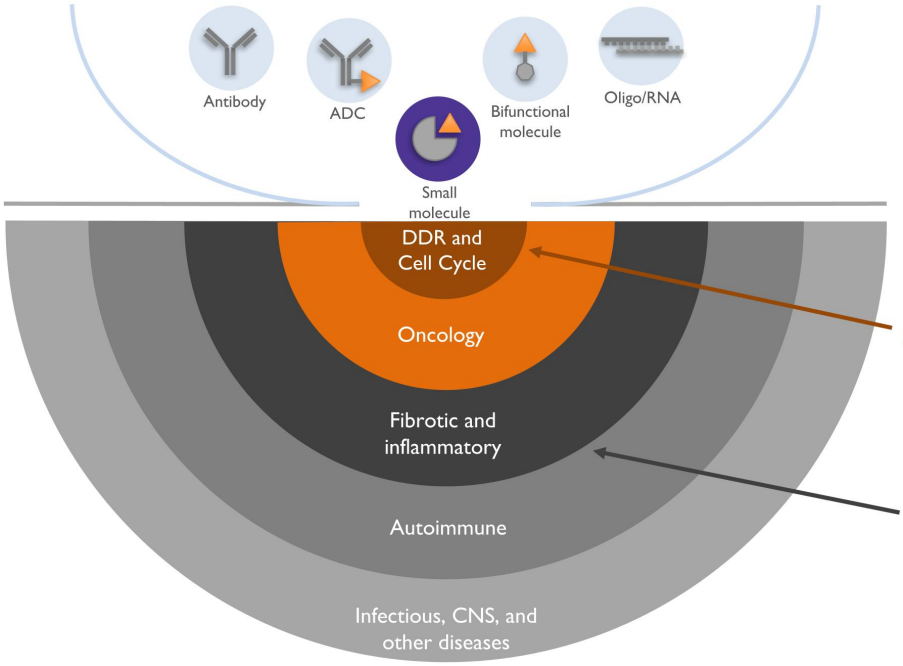
Drug-regulated pathway perturbation



THE AP3 APPROACH IS MODALITY AND DISEASE AGNOSTIC

Therapeutic modalities

Therapeutic areas



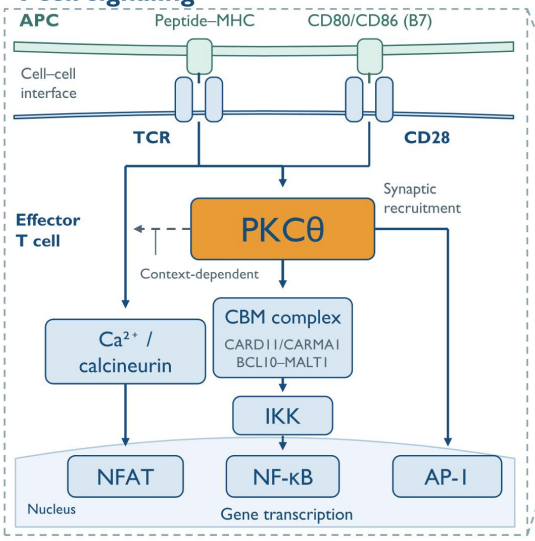
AP3 Oncology programs

AP3 I&I programs

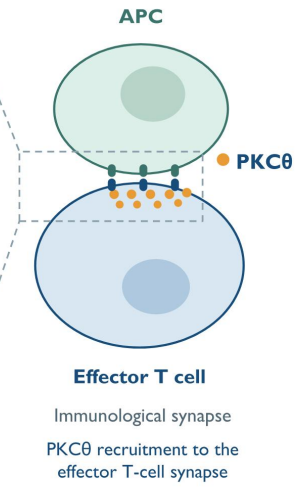
PRECLINICAL PKCΘ PROGRAM

PKCΘ PLAYS CRITICAL ROLE IN T CELL ACTIVATION

T-cell signaling



Synapse localization



Immunological synapse

PKCθ recruitment to the effector T-cell synapse

Critical role in T-cell activation

Supports NF-κB and AP-1 activation and cooperates with calcium/NFAT signaling.

Effector–regulatory balance

Convergence with JAK/STAT mediated Th2/Th17 pathways

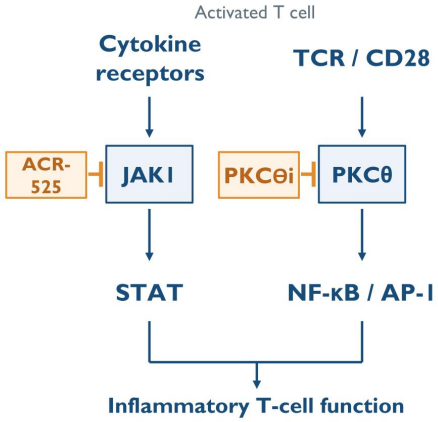
Strong rationale based on extensive preclinical studies

Mouse colitis, allergic airway and human RA Treg studies suggest PKCθ exterts a critical role in T cell-mediated inflammation

Selected primary studies: Sun, Nature 2000. Marsland, J Exp Med 2004. Nagahama, Gastroenterology 2008. Zanin-Zhorov, Science 2010. Kong, Nat Immunol 2011.

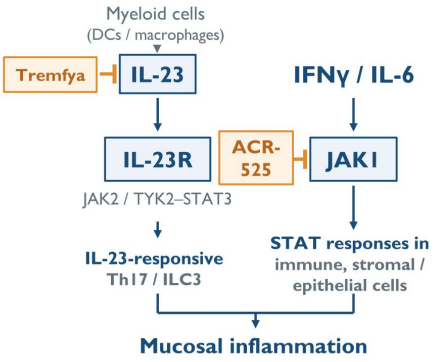
DEVELOPMENT OPPORTUNITIES: AP3-DEFINED RATIONAL COMBINATIONS WITH ACR-525

ACR-525 + PKC-θi



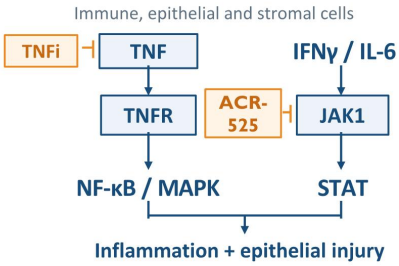
Indication potential:
UC, RA, psoriasis, atopic dermatitis, allergic asthma, SLE

ACR-525 + IL-23i



Indication potential:
Crohn's, UC, psoriatic arthritis, plaque psoriasis, uveitis

ACR-525 + TNFi



Indication potential:
Candidate indications: IBD, RA, AS

High selectivity and oral availability of ACR-525 provides for broad combination potential