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): January 12, 2026

Aclaris Therapeutics, Inc.
(Exact name of registrant as specified in its charter)

Delaware	**001-37581**	**46-0571712**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

701 Lee Road, Suite 103
Wayne, PA 19087
(Address of principal executive offices, including zip code)

(484) 324-7933
(Registrant's telephone number, including area code)

N/A
(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, $0.00001 par value	ACRS	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 January 12, 2026, Aclaris Therapeutics, Inc. (the "***Company***") updated its corporate overview presentation, a copy of which is furnished as Exhibit 99.1 to this Current Report on Form 8-K. The updated corporate overview presentation is also available on the Company's website.

In accordance with General Instruction B.2. of Form 8-K, the information in this Item 7.01 and Exhibit 99.1 hereto shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "***Exchange Act***"), or otherwise subject to the liability of that section, nor shall it be deemed incorporated by reference in any of the Company's filings under the Securities Act of 1933, as amended, or under the Exchange Act, whether made before or after the date hereof, regardless of any incorporation language in such a filing, except as expressly set forth by specific reference in such a filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	Exhibit Description
99.1	Company Presentation.
104	The cover page from Aclaris Therapeutics, Inc.'s Form 8-K filed on January 12, 2026, formatted in Inline XBRL.

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.

ACLARIS THERAPEUTICS, INC.

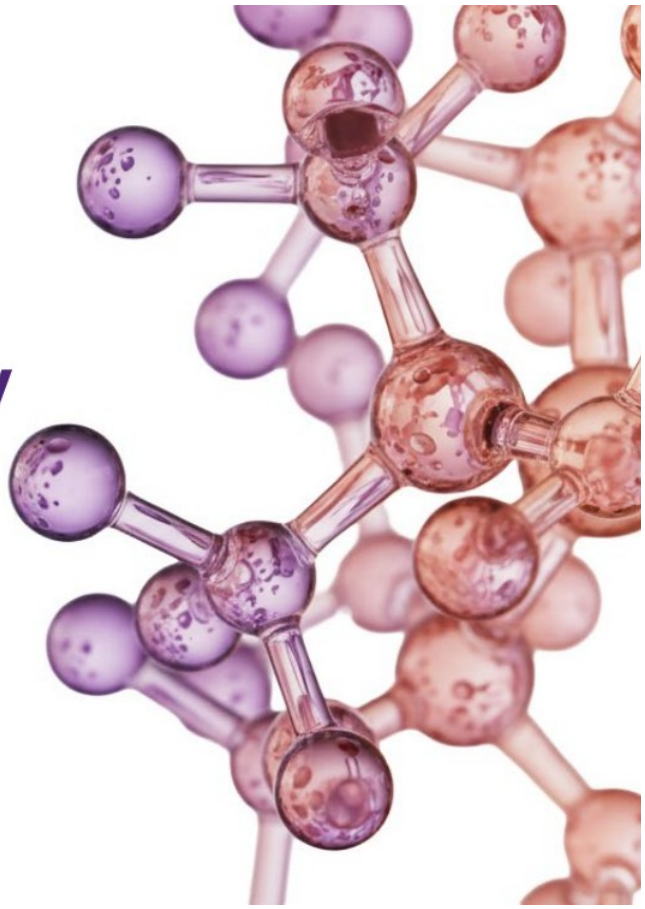
By: /s/ Kevin Balthaser
Kevin Balthaser
Chief Financial Officer

Date: January 12, 2026

aclaris THERAPEUTICS

Corporate Overview

January 2026



EMPOWERING PATIENTS THROUGH

THERAPEUTIC INNOVATION

Disclaimer and Cautionary Note Regarding Forward-Looking Statements

Any statements contained in this presentation that do not describe historical facts may constitute forward-looking statements as that term is defined in the Priv Securities Litigation Reform Act of 1995. These statements may be identified by words such as "anticipate," "believe," "expect," "intend," "may," "plan," "potential," "will," and similar expressions, and are based on Aclaris' current beliefs and expectations. These forward-looking statements include expectations regarding the therapeutic potential of Aclaris' product candidates, including bosakitug (ATI-045), ATI-052, ATI-2138, next generation ITK selective inhibitors, and generation bispecific and multispecific antibodies, including the potential for such product candidates to be best-in-class and have best-in-class attributes, the potential to increase the efficacy ceiling and show superior activity compared to other therapies, and the potential for bosakitug to have extended dosing an 052 to have up to 3-month dosing, the development of such product candidates, including the potential targets and indications Aclaris may pursue, the timing number of regulatory filings, the design of future clinical trials, the timing for the initiation of clinical trials, and the availability and timing of data from clinical tria and Aclaris' cash runway, including potential to extend the cash runway through non-dilutive opportunities. These statements involve risks and uncertainties tha could cause actual results to differ materially from those reflected in such statements. Risks and uncertainties that may cause actual results to differ materially include uncertainties inherent in the conduct of clinical trials, potential changes to interim, topline and preliminary data as more subject data become availab Aclaris' reliance on third parties over which it may not always have full control, Aclaris' ability to enter into strategic partnerships on commercially reasonable te the uncertainty regarding the macroeconomic environment and other risks and uncertainties that are described in the "Risk Factors" section of Aclaris' Annual Report on Form 10-K for the year ended December 31, 2024, and other filings Aclaris makes with the U.S. Securities and Exchange Commission from time to time These documents are available under the "SEC Filings" page of the "Investors" section of Aclaris' website at www.aclaristx.com. Any forward-looking statemen speak only as of the date of this presentation and are based on information available to Aclaris as of the date of this presentation, and Aclaris assumes no obligation to, and does not intend to, update any forward-looking statements, whether as a result of new information, future events or otherwise.

This presentation also contains estimates and other statistical data made by independent parties and by Aclaris relating to market size and other data about Aclaris' industry. These data involve a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions and estimates of the future performance of the markets in which Aclaris operates are necessarily subject to a high degree of uncertai and risk.

Tradenames, trademarks and service marks of other companies appearing in this presentation are the property of their respective owners.

All future development, clinical, and regulatory timelines are expectations, are based on current beliefs and assumptions, and are subject to change based or variety of factors.

Aclaris Therapeutics

- **Multiple anticipated inflection points in 2026**
- **Four anticipated clinical programs**
- **Validated targets**; positioned in the oral and antibody space
- Bispecific and ITK oral programs have the potential to be **paradigm changing in multiple indications**
- **Prudent capital management**
- Underpinned by **state-of-the-art scientific platform and world class scientific acumen**

Advancing potential industry-leading inhibitors designed to address validated, therapeutically-relevant immune targets

Over 1B People Live with Addressable I&I Disease

		U.S. Patients	Global Patients
Dermatology	Atopic dermatitis	26M	204M
	Alopecia areata	6.7M	160M
	Vitiligo	2-3M	70M
	Lichen planus	0.6M	12M
Respiratory	Asthma	25M	340M
	COPD	16M	390M
GI	IBD	2.4M	7M
	EOE	0.5M	3.3M

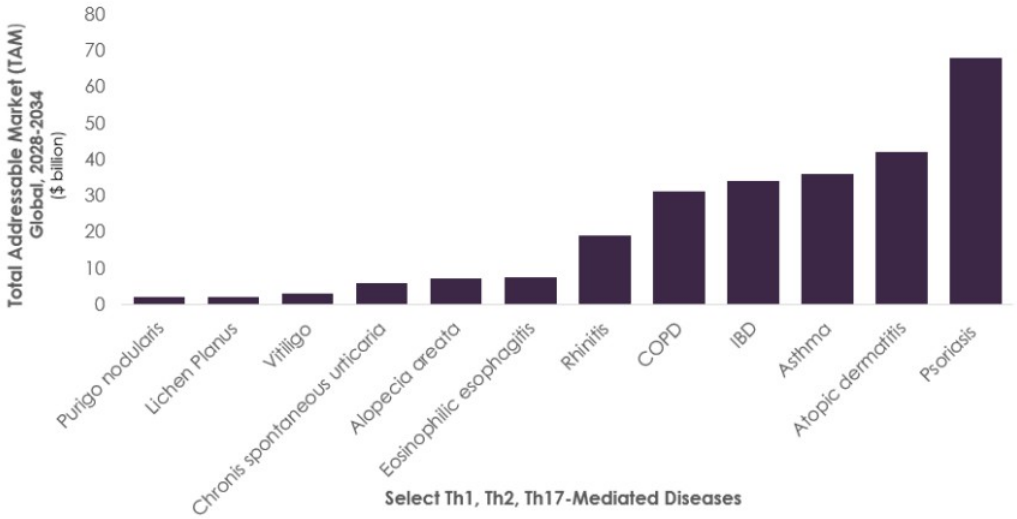
Th1, Th2, and Th17 drive dermatological, respiratory, and gastrointestinal (GI) diseases that impact millions of patients in the U.S. alone

COPD = Chronic obstructive pulmonary disease; IBD = Irritable bowel syndrome; EOE = Eosinophilic esophagitis

Sources: Eczema stats: National Eczema Association (accessed 07/31/25); National Alopecia Areata Foundation (Accessed 07/31/25); Vitiligo Facts: Global Vitiligo Foundation (accessed 07/31/25); Precedence Research; Forbes Business Insights; American Medical Association; American Lung Association; Global Initiative for Asthma; World Health Organization; Lancet Respir Med. 2022 May;10(5):447–458. doi: 10.1016/S2213-2600(21)00511-7; The Centers for Disease Control and Prevention (CDC); Cowen Categories Outlook 2024; Journal of the American Academy of Dermatology, vol 87, DOI: 10.1016/j.jaad.2021.12.013; Journal of the Academy of Dermatology, DOI: 10.1016/j.jaad.2025.05.852. Lancet Gastroenterol.Hepatol.2020,5, 17–30.

Significant Future Value of Addressing I&I Disease

Addressing Th1, Th2 and Th17-Mediated Disorders



Significant opportun for new innovative therapeutics for Th1 Th2, and Th17-mediated dermatological an respiratory disease including potent an well tolerated biologics and oral inhibitors

I&I = Inflammation and Immunology

Sources: Eczema stats: National Eczema Association (accessed 07/31/25); National Alopecia Areata Foundation (Accessed 07/31/25); Vitiligo Facts: Global Vitiligo Foundation (accessed 07/31/25); Precedence Research; Forbes Business Insights; American Medical Association; American Lung Association; Global Initiative for Asthma; World Health Organization; The Centers for Disease Control and Prevention (CDC); Business Research Company; peer research; Delveinsight; HBSC Global Investments Research 2026 Pharma Catalysts; Cowen Categories Outlook 2024

Substantial Opportunities in I&I for Innovative Drug

Potential to Address Significant Gaps in Unsatisfied I&I Indications

Opportunities for Orals

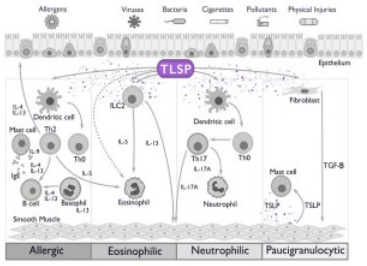
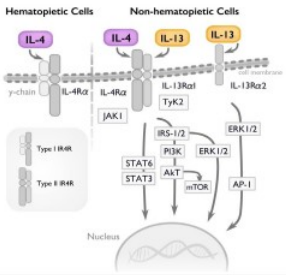
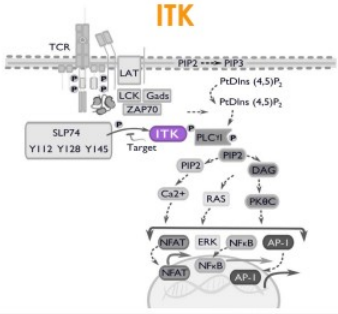
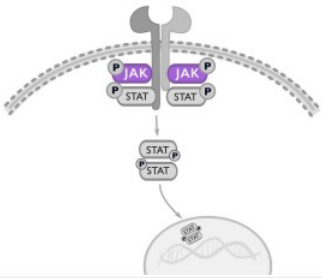
- Faster onset, durable, consistent effect
- Broader efficacy across heterogenous populations
- JAK-like efficacy with no black box warning
- Improved symptom control: Anti-itch effect, FEV1
- Anti-fibrotic effect
- Optimal convenience
- Improved tolerability profile

Opportunities for Antibodies

- Higher efficacy ceiling
  - Faster onset, durable, deeper, and more consistent effect
- Improved symptom control: Anti-itch effect
- Improved tolerability
- Improved convenience and practical dosing schedule

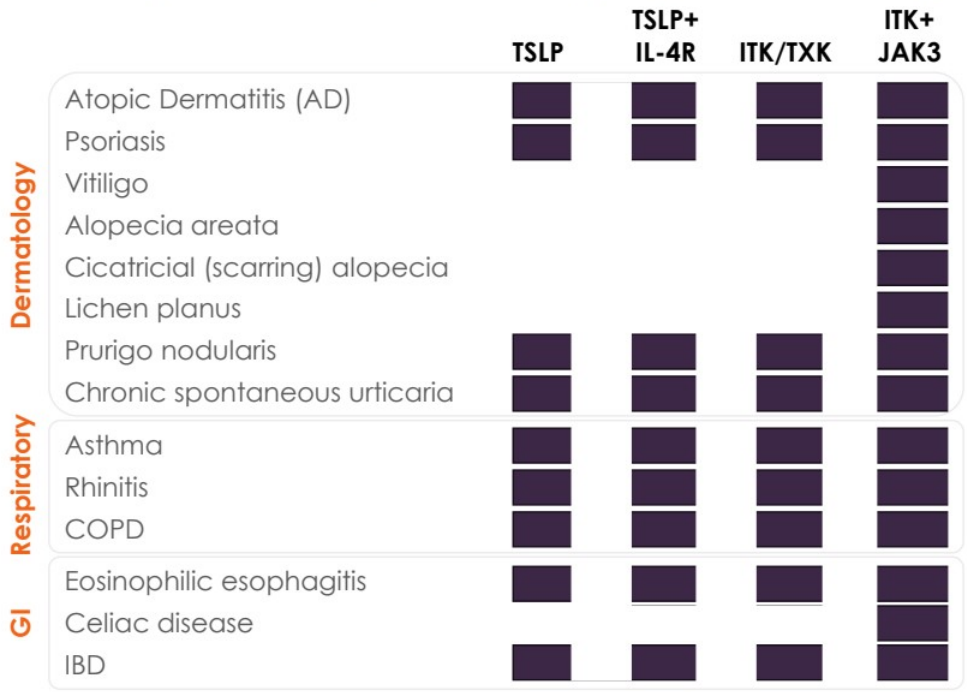
Addressing Validated Targets

With Innovative, Potent and Specific Biologics and Oral Kinase Inhibitors

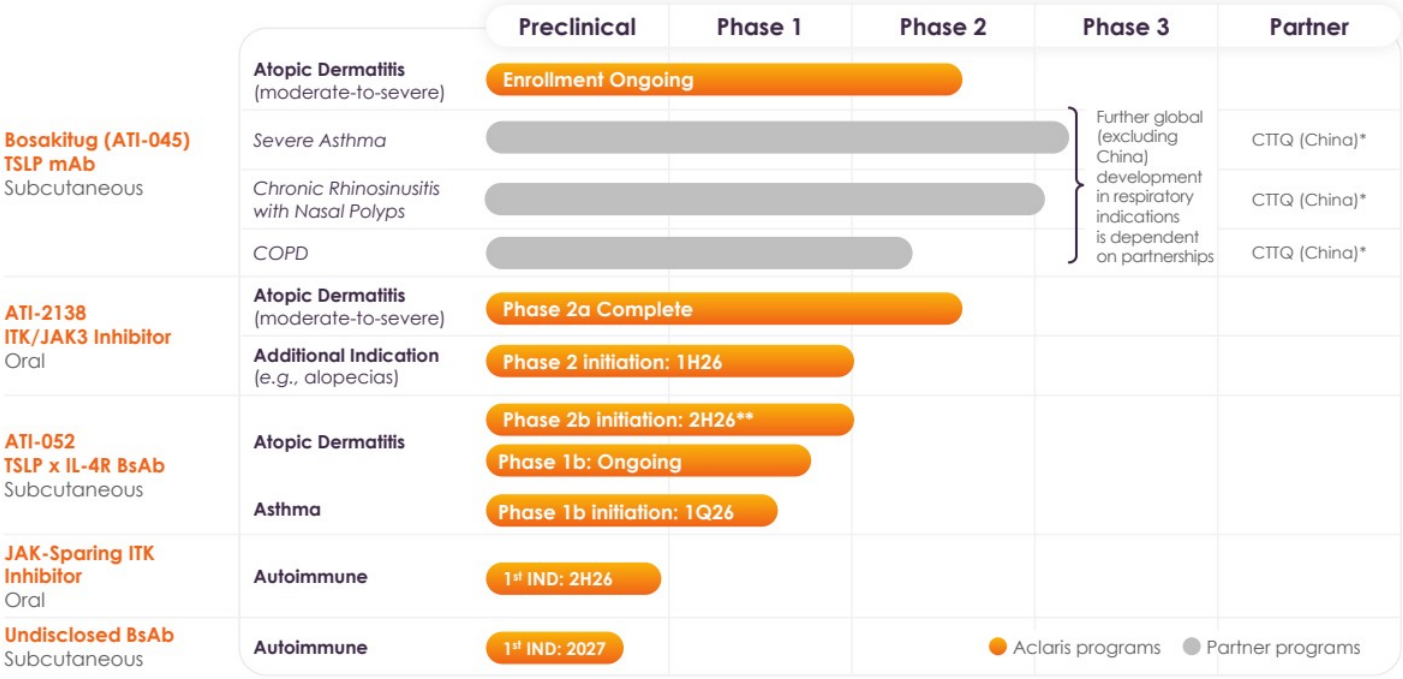
	TSLP	IL-4R	ITK	JAK3
				
Biology	Master activator of innate & adaptive immune responses at epithelial surfaces	Binds to cytokines IL-4 and IL-13, orchestrating allergic responses and immune regulation	Critical for T lymphocyte differentiation, proliferation and activation	Regulates growth, maturation and activation of many immune cells
Implication	Elevated in individuals with respiratory and skin disease	Elevated in individuals with allergic, respiratory, and skin disease	Active in individuals with respiratory and skin disease	Active in individuals with allergic, autoimmune and inflammatory disease
Utility	Th1, Th2 and Th17-driven diseases	Th2-driven diseases	Th1, Th2, and Th17-driven diseases	Th1, Th2, and Th17-driven diseases
	Validated, high conviction target	**BsAb advancing to Proof-of-Concept trials**	**Large opportunity across I&I disease**	**Proven target, ample whitespace**

Opportunity to Impact Major I&I Indications

Assets in Development May Be Broadly Applicable to Significant Diseases



Broad I&I Development Pipeline



*This trial is sponsored and conducted by Chia Tia Tianquing Pharmaceuticals Group, Co., Ltd. ("CTTQ") or its affiliates; Aclaris will not develop bosakitug in this indication on its own.
**In planning
All future development, clinical, and regulatory timelines are expectations, are based on current beliefs and assumptions, and are subject to change based on a variety of factors

aclaris THERAPEUTICS

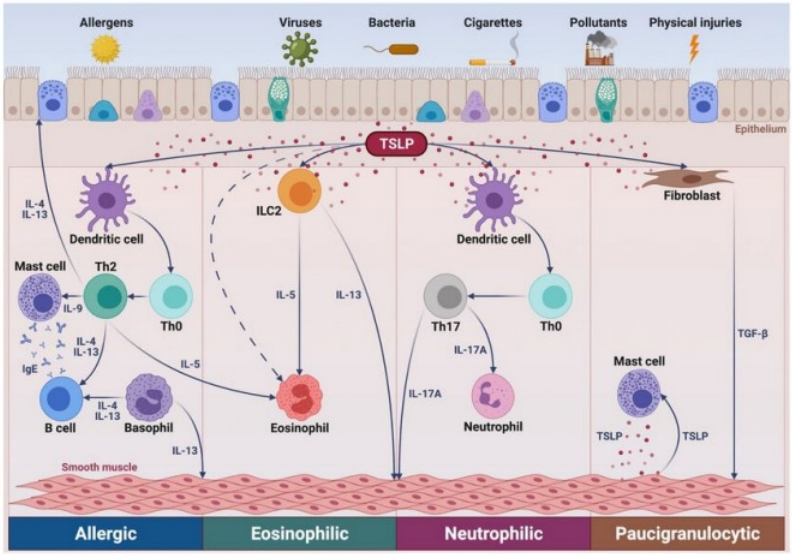
Bosakitug (ATI-045): Highly Differentiated Anti-TSLP Antibody

Investigational Product Candidate with Best-in-Class Potential

Targeting Thymic Stromal Lymphopoietin (TSLP)

Therapeutically Relevant Immune Target

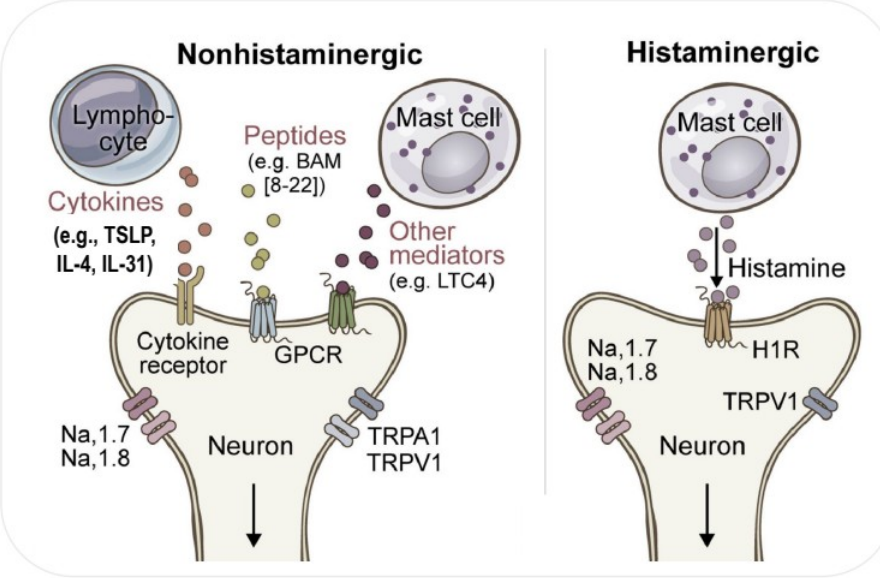
- Pleiotropic and broad activity
  - **Master regulator of type 2 (Th2) immune responses** at the barrier surfaces of skin and the respiratory/ gastrointestinal tract
  - **Drives eosinophilic and neutrophilic inflammation** and acts on a wide variety of adaptive, innate, and structural cells
  - **Broad activity:** Involved in induction phase and effector phase as well as non-Th2 processes
  - **Proven biology:** The expression of TSLP is elevated in individuals with respiratory and skin disease



Adapted from Pelia *et al.*, Int J Mol Sci. 2021 Apr 22;22(9):4369

Targeting TSLP May Impact Nonhistaminergic Itch

- Peripheral pruritus (itch) is mediated by histamine-dependent or histamine-independent mechanisms
- Nonhistaminergic itch is a significant concern in a variety of diseases including AD, CSU, PN, and others
  - Lack of response to traditional oral/topical antihistamines
- TSLP drives chronic nonhistaminergic itch, acting directly on sensory nerves and inflammatory cells

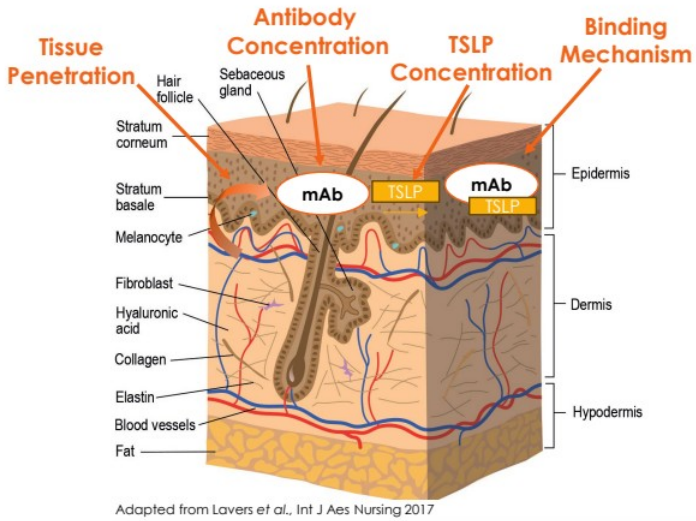


Adapted from Auyeung K, Kim B *et al.,* Annals of Allergy, Asthma, and Immunology. 2023. DOI: 10.1016/j.anai.2023.08.008

 CSU=Chronic spontaneous urticaria; PN=Prurigo nodularis

High Potency Therapeutics are Key to Effectivene

Driving High Efficacy in Dermatological Disease

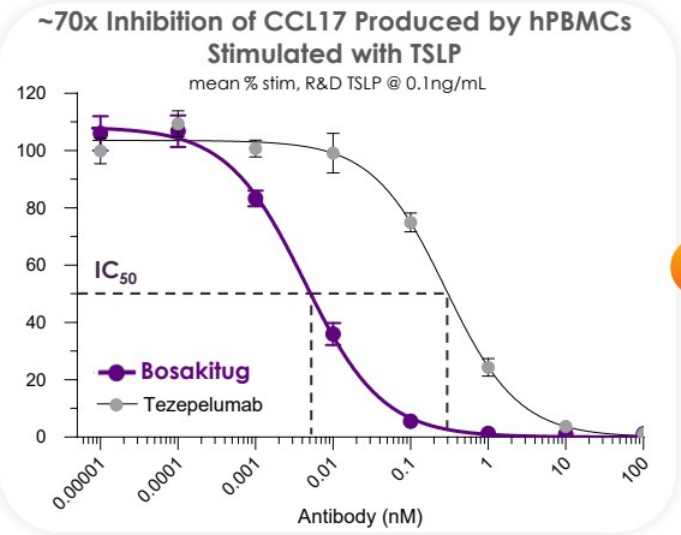


Adapted from Lavers *et al.*, Int J Aes Nursing 2017

- Abs must engage ligands at the site of action
- Key variables related to efficacy
  - TSLP concentration at site of lesion
  - Antibody concentration at site of lesion
    - Concentration of mAb in general circulation
    - Skin penetration of mAb
    - Dose
    - Potency
  - Binding Mechanism of mAb to TSLP
    - Binding affinity
    - Residence Time
  - Degree of TSLP reduction needed at site of lesion

Only 15% of mAb serum levels reach skin/site of lesion: Potency is Key to Efficacy

Bosakitug: High Affinity, Low Dissociation Rate



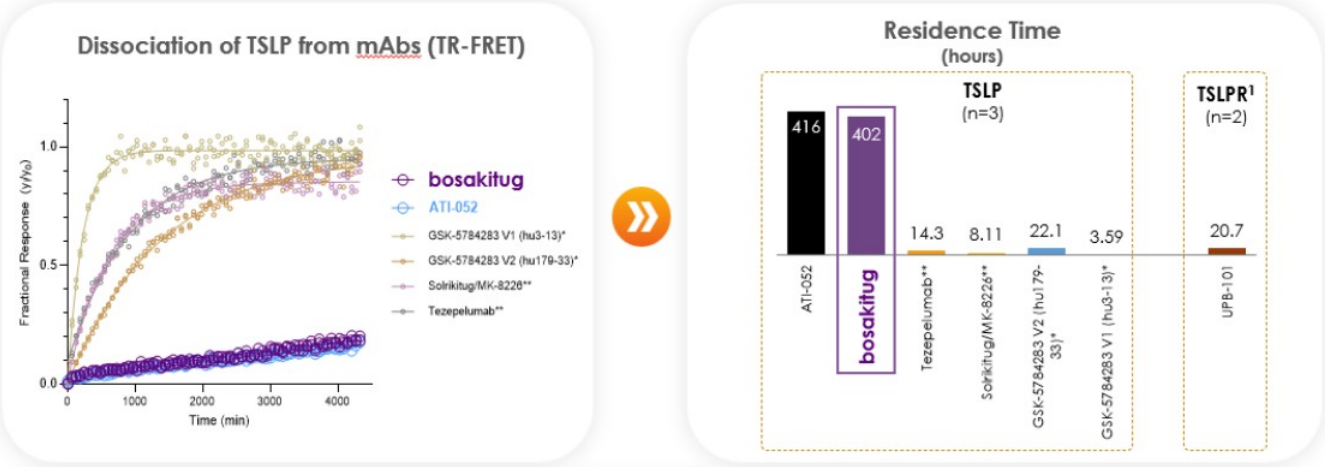
- Very high affinity to TSLP
- Extremely low dissociation rate from TSLP* leading to long residence time and enhanced neutralization activity
- Very high potency
- Unique binding characteristics to TSLP
- ~23-day half-life that can potentially support an extended dosing interval of up to 3 months

Bosakitug is ~70x More Potent than Tezepelumab, the Only Marketed Anti-TSLP mAb

* Quantification of dissociation rate limited by the surface plasmon resonance instrument sensitivity

Bosakitug: Long Residence Time

Lower Dissociation Rate = Potential Best-in-Class Residence Time



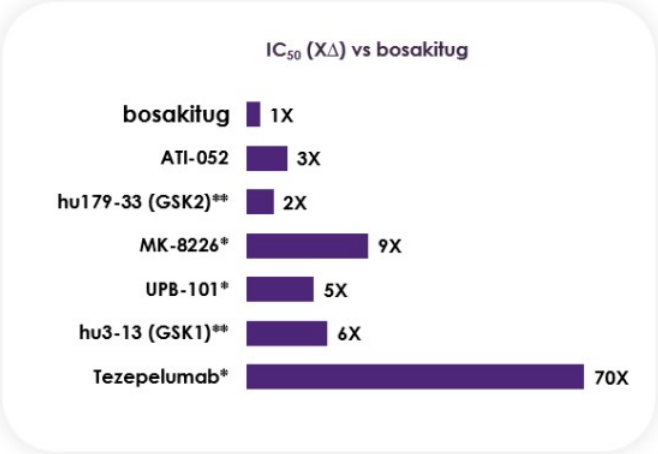
Bosakitug (and ATI-052) demonstrates very slow dissociation kinetics from TSLP

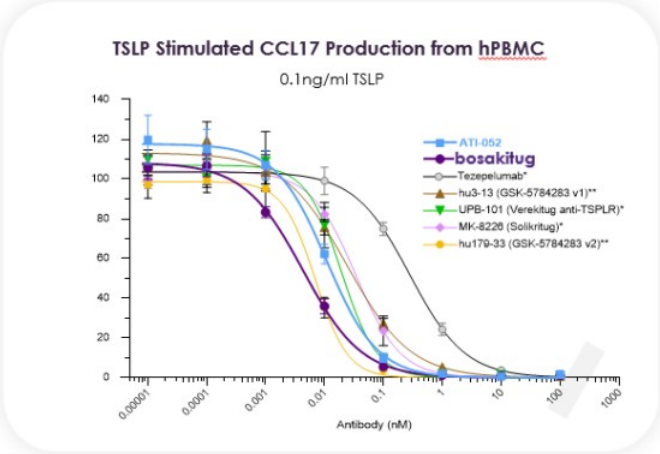
Residence time for Bosakitug (and ATI-052) is ~20-100x longer than comparator antibodies

1. SPR: Residence Time based on apparent dissociation constant (Kd) using standard TSLPR immobilization density and bivalent fit; *Analog mAb; **Biosimilar mAb

Bosakitug: Potential Best-in-Class Potency

Greater Potency Than Other TSLP/TSLPR Antibodies





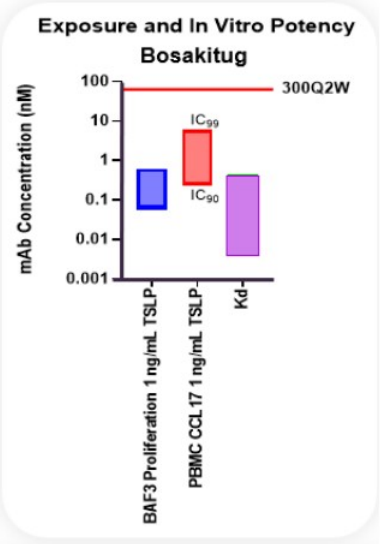
Bosakitug is the most potent of the TSLP/TSLPR antibodies evaluated in blocking CCL17 production

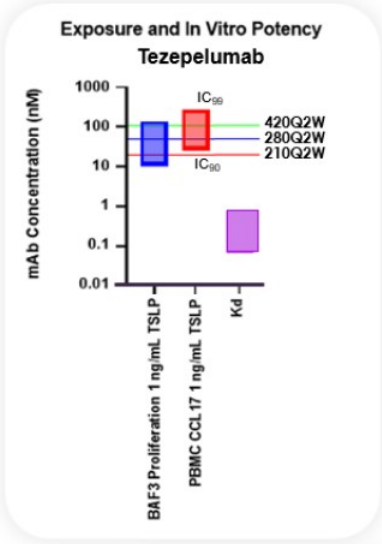
ATI-052 retains much of the potency for TSLP functional blockade

 *Biosimilar; **Analog

Potency Advantage of Bosakitug vs Tezepelumab

Relationship of Potency and Exposure to Extent of TSLP Inhibition





- Bosakitug is expected to cover multiples over the concentration needed for 99% inhibition of TSLP at the site of action based on its in vitro potency
- The highest dose of Tezepelumab may not cover the IC99 for TSLP at the site of action based on in vitro potency

Bosakitug Potency May Allow for Substantial Exposure Above 99% Inhibition of TSLP

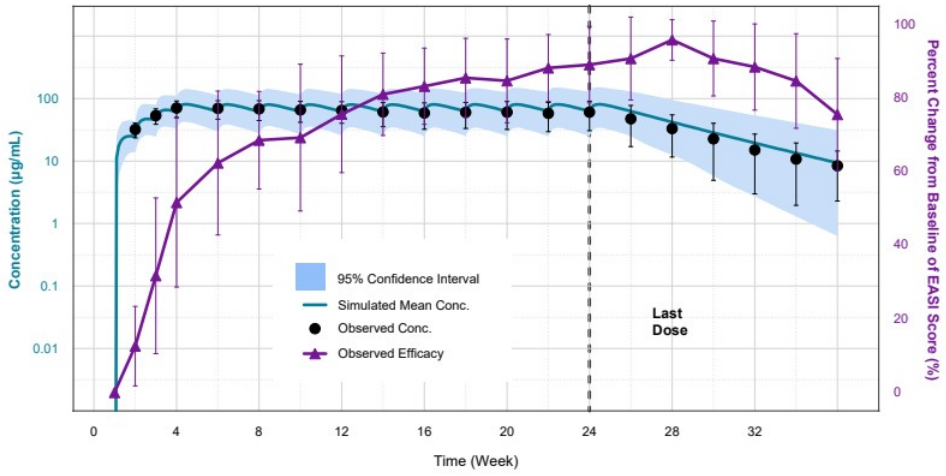
Clinical Translation: Positive Clinical Results

Phase 2a (US-Based) POC Monotherapy Trial

Eligibility	Diagnosis of AD (present for at least 6 months); EASI ≥12; IGA ≥3; total AD BSA ≥10%
Baseline Characteristics	Mean EASI of 17.6, Mean PP-NRS of 6.5; majority had prior medication prior to screening
Primary Objective (Week 24)	To evaluate the efficacy, safety and tolerability of bosakitug as monotherapy in subjects with moderate to severe AD
Secondary Objectives (Week 24)	To evaluate the pharmacokinetics, immunogenicity and pharmacodynamic biomarkers of ATI-045 in subjects with moderate to severe AD

Bosakitug Exposure and Efficacy Time Profile

Demonstrated Sustained Clinical Response After the Last Dose

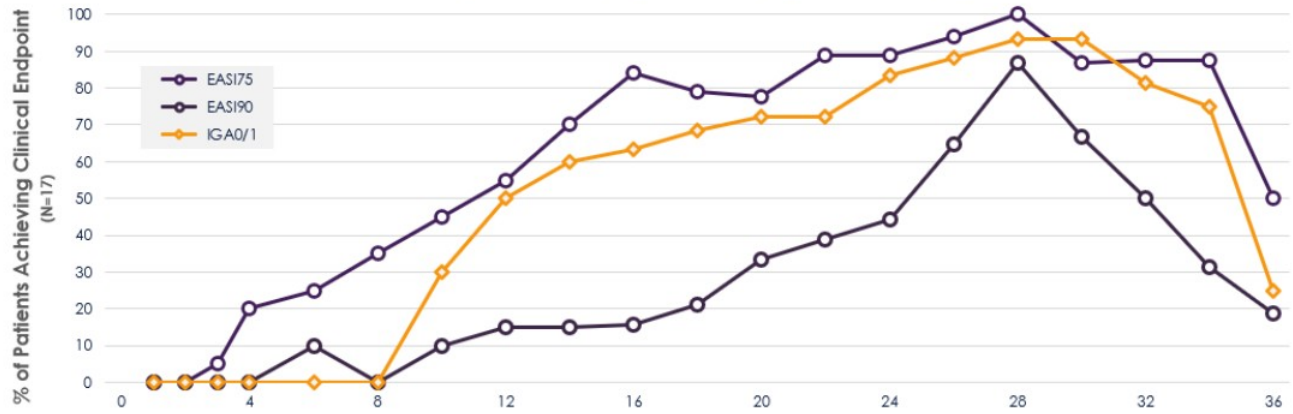


- A time lag in efficacy response relative to exposure was observed both while the drug was onboard and after the last dose
- EASI-75 sustained response after the last dose supports the possibility of longer dosing intervals
- Favorable safety and immunogenicity profile

Phase 2a (US-Based) POC Monotherapy Trial

Bosakitug Demonstrated Improvement in Efficacy Measures

EASI 75 % @ Week 26	EASI 90 % @ Week 26	EASI 100 % @ Week 26	IGA 0/1 @ Week 26
94%	65%	24%	88%



Phase 2 Monotherapy Trial Ongoing

Enrollment Proceeding to Plan

Primary Objective (Week 24)	To evaluate the efficacy of Bosakitug compared to placebo, as measured by the change in Eczema Area and Severity Index (EASI) score in patients with moderate-to-severe AD
Secondary Objectives (Week 24)	To evaluate the safety, tolerability & treatment effect of Bosakitug compared to placebo, on additional clinical outcome measures • EASI response (EASI-50, EASI-75, EASI-90) • Validated Investigator Global Assessment (IGA) response • Body Surface Area (BSA) response • Peak Pruritus Numerical Rating Scale (PP-NRS) score

Bosakitug: Next Steps

Competitively Positioned as Potential Best-in-Class TSLP mAb

Ongoing / Next Steps

- Two-arm placebo-controlled Phase 2 trial in moderate-to-severe AD ongoing; Top line results expected in 2H 2026
- Aclaris is seeking partners to develop bosakitug in respiratory indications; further global (excluding China) development in these indications is dependent on entering into potential partnerships

aclaris THERAPEUTICS

ATI-052: Anti-TSLP x IL-4Rα First Generation Bispecific Antibody Program

Highly Potent and Bioactive Investigational Product Candidate

ATI-052: Potential Best-in-Class Bispecific mAb

Effective Dual Binding of TSLP and IL-4Rα

Anti-IL4Rα scFV

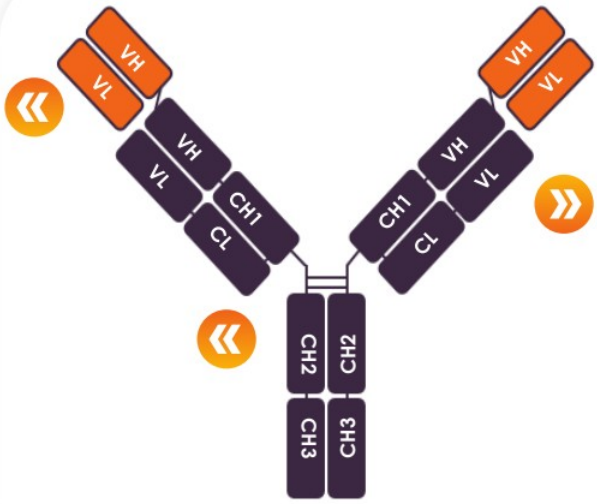
Designed to inhibit immune cells downstream of the Th2 cascade

YTE Mutation

Fc engineered to bind more tightly to FcRn, potentially extending half-life

AQQ Mutation

Fc mutation limits effector functionality, potentially reducing off-target binding and potential toxicity



ATI-052

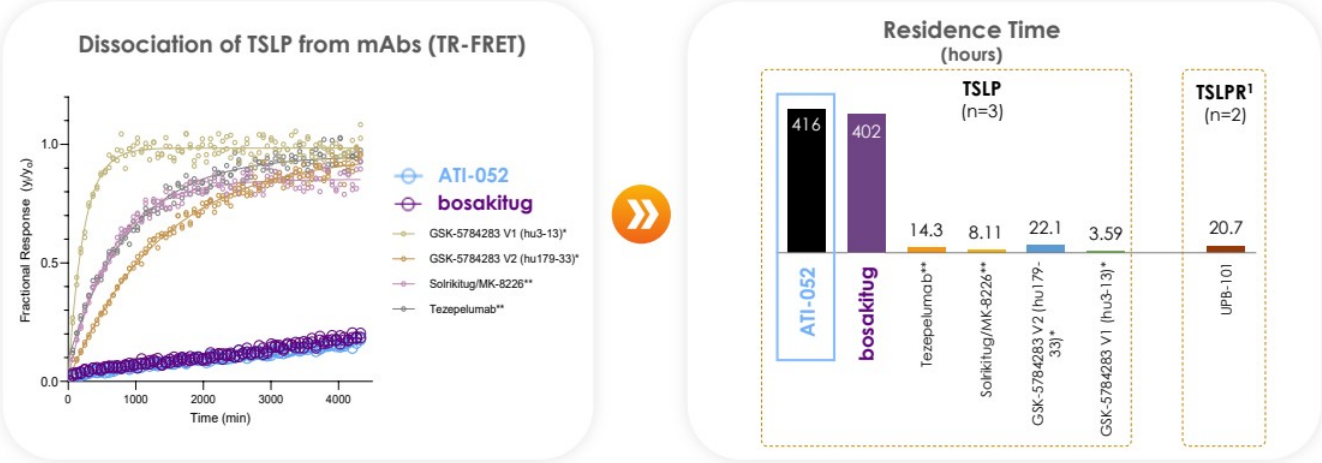
Anti-TSLP Fab

Same anti-TSLP antibody binding regions of Bosakitug, **designed to inhibit TSLP upstream of the Th2 cascade**

- Retains dissociation kinetics, residence time, and potency advantages of bosakitug over comparator antibodies

ATI-052: Longest Residence Time on TSLP

Lower Dissociation Rate Drives Longer Residence Time



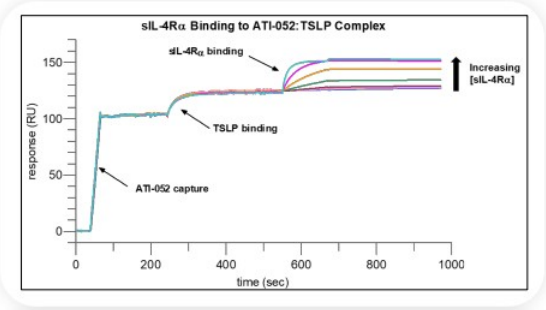
ATI-052 demonstrates very slow dissociation kinetics from TSLP

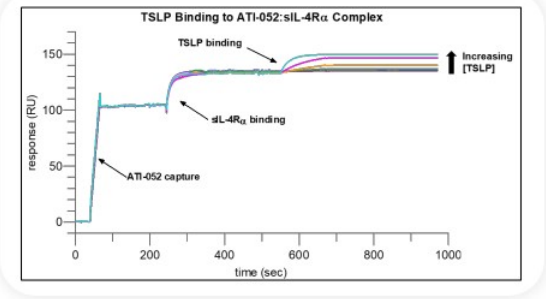
Residence time for ATI-052 is ~30-116x longer than comparator antibodies

1. SPR: Residence Time based on apparent kd (dissociation constant) using standard TSLPR immobilization density and bivalent fit; *Analog mAb; **Biosimilar mAb

Concurrent Binding of TSLP and sIL-4Rα to ATI-052

High Affinity to Both TSLP and IL-4Rα





Comparison of Affinity for sIL-4Rα Binding to ATI-052 or ATI-052:TSLP Complex

Parameter	ATI-052	ATI-052:TSLP
K_D (pM)	348	215

Comparison of Affinity for TSLP Binding to ATI-052 or ATI-052:sIL-4Rα Complex

Parameter	ATI-052	ATI-052:sIL-4Rα
K_D (pM)	41.2	33.9

ATI-052 Binds Both Targets Effectively

High affinity to either target is not altered by the binding to the other

Concurrent Binding of TSLP and sIL4Rα to ATI-052

Simultaneous Binding of TSLP and IL-4Rα

Binding Sequence	TSLP:ATI-052 Stoichiometry*	sIL-4Ra:ATI-052 Stoichiometry*
ATI-052 capture / sIL-4Ra dose-response	n/a	2.25
ATI-052 capture / TSLP load / sIL-4Ra dose-response	1.82	2.10
ATI-052 capture / TSLP dose-response	2.04	n/a
ATI-052 capture / sIL-4Ra load / TSLP dose-response	1.83	1.97

** determined using molecular weights based on amino acid sequence, does not account for glycosylated species*

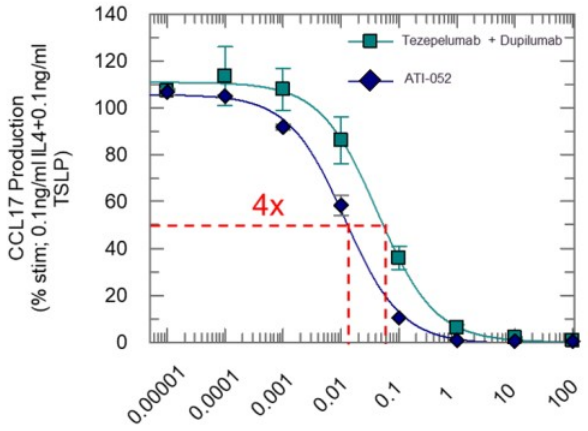
- ~2 molecules of sIL-4Rα bound to ATI-052 in the absence (2.25:1) and presence (2.10:1) of TSLP
- ~2 molecules of TSLP bound to ATI-052 in the absence (2.04:1) and presence (1.82:1) of sIL-4Rα

ATI-052 Demonstrated High Affinity to Both Targets Simultaneously:

ATI-052 binds ~two molecules of TSLP and sIL-4Rα with the potential to saturate all 4 binding sites at the same time

Comparison of ATI-052 vs Dupilumab + Tezepelumal

ATI-052 Demonstrates Greater Potency than the mAb Combination

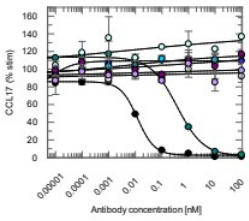


mAb Concentration	
Antibody	**IC50 (nM)**
ATI-052	0.016
Dupilumab + Tezepelumab	0.069
Fold change	4.3

ATI-052 is Substantially More Potent than the Combination of Dupilumab and Tezepelumab

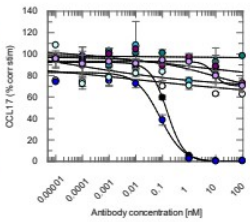
Comparison of IL-4/IL-13 Monoclonal Antibodies

TSLP-induced CCL17 release



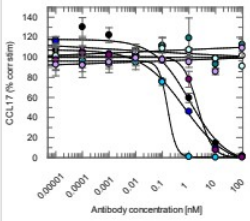
Antibody	IC50 [nM]
ATI-052	0.0077
Dupilumab	>100
Lebrikizumab	>100
Tezepelumab	0.4083
Tralokinumab	>100

IL-4-induced CCL17 release



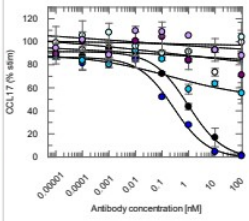
Antibody	IC50 [nM]
ATI-052	0.18
Dupilumab	0.09
Lebrikizumab	>100
Tezepelumab	>100
Tralokinumab	>100

IL-13-induced CCL17 release



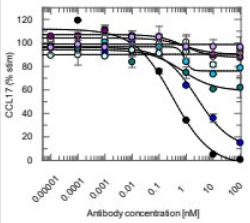
Antibody	IC50 [nM]
ATI-052	0.74
Dupilumab	0.57
Lebrikizumab	0.55
Tezepelumab	>100
Tralokinumab	1.83

IL-4+IL-13-induced CCL17 release



Antibody	IC50 [nM]
ATI-052	0.97
Dupilumab	0.27
Lebrikizumab	>100
Tezepelumab	N/A
Tralokinumab	>100

TSLP + IL-4 + IL-13-induced CCL17 release



Antibody	IC50 [nM]
ATI-052	0.3625
Dupilumab	2.0217
Lebrikizumab	>100
Tezepelumab	>100
Tralokinumab	>100

ATI-052
Dupilumab
Tezepelumab
Tralokinumab
Lebrikizumab
IgG1
IgG4k
IgG4l

ATI-052 Exhibits the Broadest Activity Among the Biologics Tested

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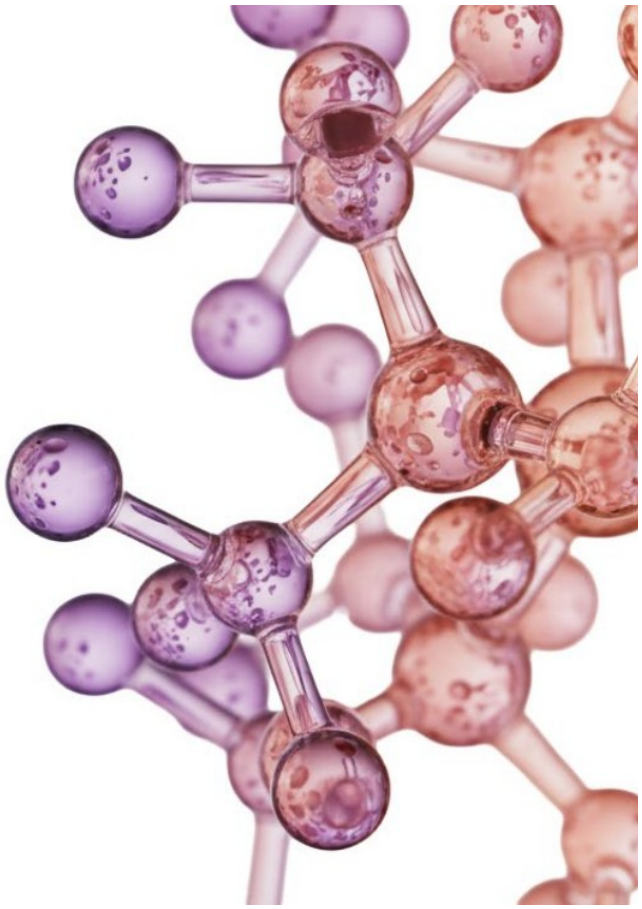


Positive Interim Data: ATI-052 Healthy Volunteer Phase 1a SAD and MAD Trial

Interim results as of December 31, 2025

Patient *Focused Innovation*

Interim ATI-052 Results Exceeded Expectations

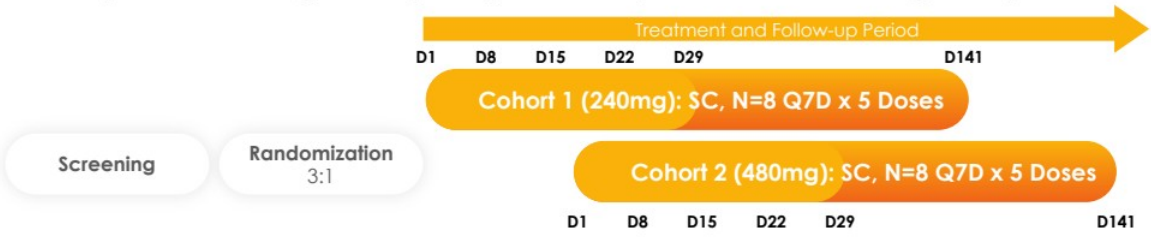
Goal	Goal	Goal	Goal
Confirm **Favorable Tolerability and Safety Profile**	Confirm Pharmacokinetic Profile to Support **~Once a Month Dosing**	Confirm **Strong Pharmacodynamic Response**	Confirm **Efficient Inhibition of Both TSLP and IL-4Rα**
Result ATI-052 was found to be **well tolerated with a favorable safety profile across all SAD and MAD cohorts**, with doses of up to 720 mg **No conjunctivitis** observed in any cohort	**Result** Results support potential for **up to every 3-month dosing interval** **Dose proportional PK** observed across pharmacologic dose range	**Result** Robust target engagement + **near complete/complete target occupancy at low doses** Inhibition of TSLP stim CCL17/TARC observed **at least six weeks after administration (360 mg)**	**Result** **Complete and sustained inhibition** of ex vivo IL-4 or TSLP stimulated CCL17/TARC
Achieved	*Exceeded*	*Exceeded*	*Exceeded*

ATI-052 Placebo Controlled Phase 1a Program

Part A Single Ascending Dose (SAD) in Healthy Volunteers (HV): Dosing Complete

Interim result strongly support advancemer into POC

Part B Multiple Ascending Dose (MAD) in Healthy Volunteers: Dosing Complete



Baseline Characteristics As Expected

Baseline Demographics & Characteristics Typical of HV Patient Population

	Single ascending dose (SAD)					Multiple ascending dose (MAD)			
	ATI-052 Cohort 1 (30 mg) N=6	**ATI-052** Cohort 2 (120 mg) N=6	**ATI-052** Cohort 3 (360 mg) N=6	**ATI-052** Cohort 4 (720 mg) N=6	**Placebo** N=8	**ATI-052** Cohort 1 (240 mg) N=6	**ATI-052** Cohort 2 (480 mg) N=6	**Placebo** N=4	**Total** N=48
Age (yrs), mean (SD)	40.0 (9.6)	34.3 (10.7)	34.7 (7.5)	34.7 (8.6)	34.4 (9.9)	37.2 (12.1)	35.3 (8.0)	30.8 (2.4)	35.3 (8.9)
Female	50%	33.3%	33.3%	16.7%	75%	66.7%	50%	50%	47.9%
Caucasian	66.7%	83.3%	16.7%	16.7%	75%	50%	50%	75%	54.2%
Weight (kg), mean (SD)	74.5 (13.9)	75.8 (16.8)	79.4 (20.5)	86.4 (11.6)	70.8 (9.2)	73.1 (7.2)	81.3 (7.3)	71.5 (8.5)	76.6 (12.8)

CLINICAL RESULTS

ATI-052 Well Tolerated with a Favorable Safety Profile Across Cohorts

	Single ascending dose					Multiple ascending dose			Overall trial
	ATI-052 Cohort 1 (30 mg) N=6	**ATI-052** Cohort 2 (120 mg) N=6	**ATI-052** Cohort 3 (360 mg) N=6	**ATI-052** Cohort 4 (720 mg) N=6	**Placebo** N=8	**ATI-052** Cohort 1 (240 mg) N=6	**ATI-052** Cohort 2 (480 mg) N=6	**Placebo** N=4	**Total** N=48
≥1 TEAE (n, %)	4 (66.7)	1 (16.7)	4 (66.7)	4 (66.7)	3 (37.5)	4 (66.7)	4 (66.7)	2 (50.0)	26 (54.2)
≥1 Serious TEAE (SAEs)	0	0	0	0	0	0	0	0	0
≥1 Grade 3 or higher TEAE	0	0	0	0	0	1 (16.7)	0	0	1 (2.1)
≥1 Conjunctivitis TEAE	0	0	0	0	0	0	0	0	0
≥1 Drug-related TEAE	0	1 (16.7)	0	2 (33.3)	0	3 (50.0)	3 (50.0)	2 (50.0)	11 (22.9)
Discontinued study due to TEAE	0	0	0	0	0	0	0	0	0

- Drug-related TEAEs were predominantly self-resolving Grade 1 injection site erythema (redness)
- One participant in MAD Cohort 1 (240mg) experienced two Grade 3 or higher TEAEs related to elevations in AST and CPK which were determined to be not related to study drug

TEAE=Treatment emergent adverse events

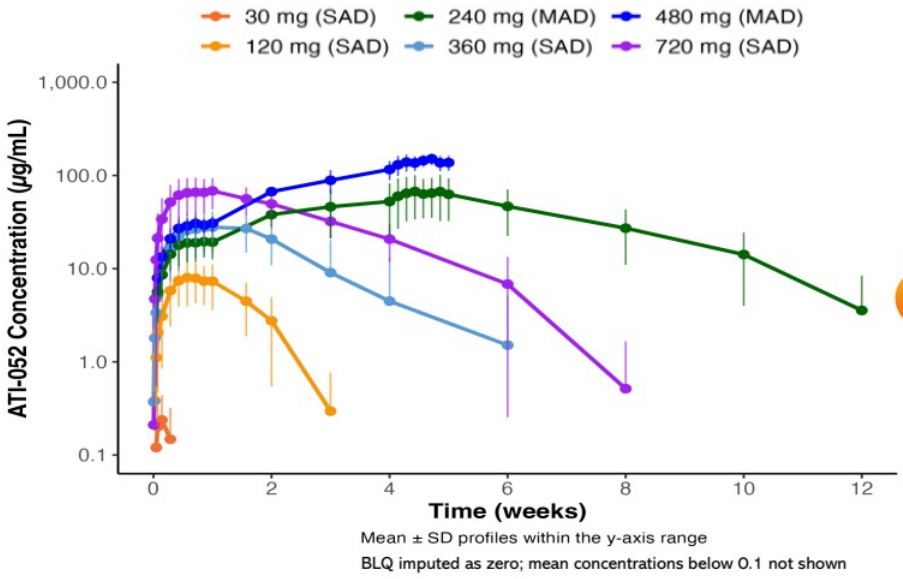
Favorable Tolerability and Safety Profile of ATI-052

Provides Confidence in Continued Development

- No SAEs; no adverse events led to study discontinuation
- Low rate of adverse events (AEs); Predominantly Grade 1
- The most common AE: Injection site redness; self-resolving and generally mild (Grade 1)
- No Grade 3 drug-related TEAEs
- No conjunctivitis observed in any cohort

Favorable tolerability and safety profile demonstrated across all ATI-052 SAD and MAD cohorts, with doses of up to 720 mg/kg

Strong Pharmacokinetic Profile



Mean ± SD profiles within the y-axis range

BLQ imputed as zero; mean concentrations below 0.1 not shown

Dose proportional PK observed across pharmacologic dose range

Strong PK Profile Support Potential Extended Dosir

Dose Proportional PK Observed Across Pharmacologic Dose Range

- Dose proportional PK was observed; dose proportional increases in Cmax and AUC observed
- PK results provide an effective half-life of at least 26 days

Potential best-in-class PK profile

Ex-Vivo Stimulated PD Assay

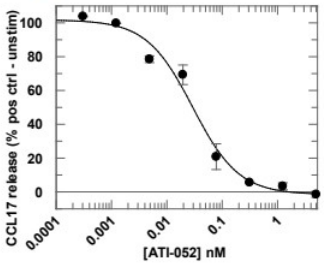
Bridging to Human Whole Blood (HWB)

- TSLP and IL-4R both modulated CCL17 release in PBMC
- For the SAD/MAD study, this assay was adapted to human whole blood to assess the following:
  - TSLP stimulated CCL17 in whole blood
  - IL-4 stimulated CCL17 in whole blood

Robust PD activity ex vivo HWB more closely reflects the real environment in patients with minimal manipulation by maintaining the complex composition of fluids and cells as they are present in circulation

hWB Assays

0.5 ng/mL TSLP stimulation—48 hours

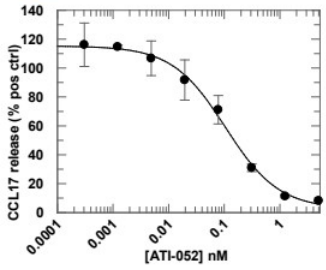


IC50 (nM) ± SEM	0.025 ±0.0042
n	5
S/N	4

5 ng/ml*

*IC50 for the inhibition of TSLP-stimulated CCL17 in whole blood was lower than the Lower Limit Of Quantitation (LLOQ) for the PK analysis of ATI-052 (LLOQ is 25 ng/ml)

2 ng/mL IL-4 stimulation—48 hours



IC50 (nM) ± SEM	0.203 ±0.039
n	6
S/N	15

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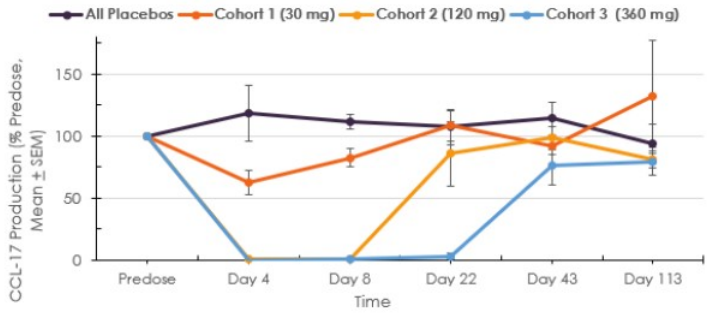
CLINICAL RESULTS

Strong and Sustained Effect of ATI-052

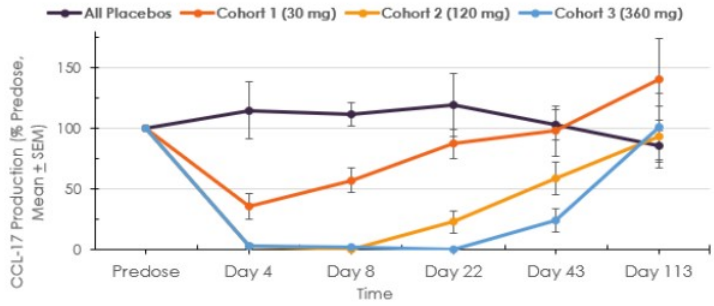
IL-4 Stimulated CCL17/TARC:
SAD Cohorts

Sustained, complete / near complete inhibition of ex-vivo stimulated responses observed for at least 3 weeks at 360 mg dose



TSLP Stimulated CCL17/TARC:
SAD Cohorts

Sustained, complete / near complete inhibition of ex-vivo stimulated responses observed for at least 6 weeks at 360 mg dose; suggests potential best-in-class residence time and potency



ATI-052 binds both targets effectively with complete inhibiti at pharmacological relevant doses for a extended time beyo the PK profile

The combination of duration and the strong and sustaine PD effect support th potential for **up to every three-month dosing**

*SAD 720 mg cohort and MAD 240/480 mg cohort data to be presented at a future medical meeting once complete

Strong Pharmacodynamic Response

Robust Target Engagement + Near Complete Occupancy at Low Doses

ATI-052 exhibited a **potential best-in-class PD profile**:

- Dose and concentration dependent inhibition of IL-4 and TSLP-stimulated CCL17 release observed
- Cohort 1 (30 mg), 2 (120 mg) and 3 (360 mg) data indicate that the time points at which there are detectable levels of ATI-052 present in blood, ATI-052 is active and engaging its target when stimulated ex-vivo
- Cohort 3 results demonstrated complete and sustained inhibition of ex vivo IL-4 or TSLP stimulated CCL17/TARC through week three
- Near complete inhibition of TSLP stimulated CCL17/TARC was observed at least six weeks after administration for Cohort 3

Observed results further validate the potency of ATI-052

Unique Binding Attributes ➡ Clinical Potential

Binding Attributes

Effective Binding of TSLP and IL-4Rα
ATI-052 binds both targets effectively; high affinity of either targets is not altered by the binding of the other

Simultaneous Binding of TSLP and IL-4Rα
High affinity to both targets simultaneously: ATI-052 binds two molecules of TSLP and sIL-4Rα

Higher Potency vs Comparator Antibodies
ATI-052 exhibits greater cellular bioactivity on CCL17 release than the combination of Tezepelumab and Dupilumab

Effective Blockade of IL-4 and IL-13
ATI-052 antagonism of IL-4Rα blocks signaling of both IL-4 and IL-13

Phase 1a Interim Clinical Results

Favorable Tolerability and Safety Profile
Favorable tolerability and safety profile observed across all SAD and MAD cohorts, with doses of up to 720 mg

Robust PK Package
Dose proportional PK observed across pharmacologic dose range

Support potential for up to 3-month dosing

Strong Pharmacodynamic Response
Robust target engagement + near complete target occupancy, even at very low doses

Efficient Inhibition of Both TSLP and IL-4R
Dose and concentration dependent inhibition of IL-4 and TSLP-stimulated CCL17 release

ATI-052: Next Steps

Positive Interim Results Validate ATI-052; Clinical Program Rapidly Advanci

Ongoing / Next Steps

- Phase 1b AD POC trial ongoing
- Initiate Phase 1b asthma POC trial: 1Q 2026
- Phase 1b top line POC results: 2H 2026
- Initiate Phase 2b AD trial: 2H 2026
- Complete assessments of additional Phase 2 indications

All future development, clinical, and regulatory timelines are expectations, are based on current beliefs and assumptions, and are subject to change based on a variety of factors

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Next-Generation Multispecific Antibodies

Next Generation Bispecific Antibodies

Progressing Toward IND

- Multispecific approach enables synergistic target binding and addresses shortcomings of multi-drug administration
- Initial assessment of bispecific targets completed (αTSLP + undisclosed)
- Campaigns progressing: Hit optimization toward lead candidate selection ongoing
- Additional bispecific and trispecific modalities under initial consideration

Targeting first IND from bispecific antibody development efforts in 2027

Opportunities for Aclaris Next Generation BsAbs

Multispecific Antibodies Can Expand Therapeutic Optionality

Pruritis (Itch)	• TSLP combinations with itch mediators may have a positive impact on itch and QoL in AD and other dermatological diseases
Alarmin Combinations	• Could impact initiation of allergic response and associated downstream inflammation and enhance anti-viral immunity during respiratory virus infections
Synergistic Effect with TSLP	• May amplify immune responses, particularly the development of Type 2 inflammation, which is central to allergic diseases like asthma, AD, and others
Eosinophil-Driven Diseases	• Allergic disorders, skin conditions, fungal infections, autoimmune diseases, others • Causes multiple disorders including eosinophilic cystitis, fasciitis, pneumonia, gastrointestinal disorders, granulomatosis with polyangiitis, hypereosinophilic syndron

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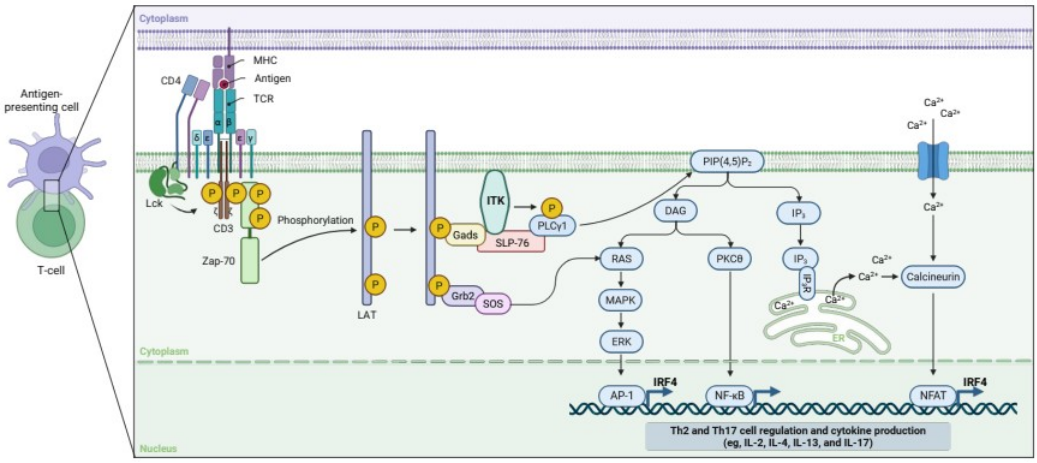
ATI-2138: A First Generation Novel ITK/JAK3 Inhibitor for T Cell-Mediated Diseases

Potent and Selective Investigational Product Candidate with Strong Tolerability Profile

Critical T Cell Receptor (TCR) Pathway

Interleukin-2-Inducible T-cell Kinase (ITK): Key Kinase Involved in TCR Signaling



- TCR activation is critical fo T lymphocyte differentiati proliferation and activatio
- TCR signaling proceeds through a complex intracellular pathway resu in the activation of key transcription factors and production of cytokines
- Central to TCR signaling is kinase ITK

Inhibiting/downregulating ITK shuts down TCR signaling under inflamed/allergic conditions and impacts disease

ITK Inhibition: Broadly Applicable in I&I Pathways

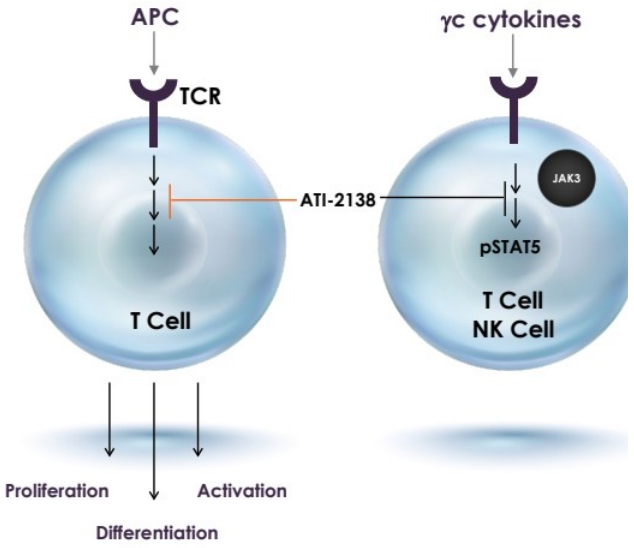
Comparative Impact of ITK Inhibition

	Th2				Th17			ILC2		Th1	
	IL4	IL5	IL13	IL31	IL17	IL21	IL22	IL5	IL13	IFN γ	IL2
ITK Inhibitor	✓	✓	✓	✓	✓	✓	✓	✓	✓		
ITK/TXK Inhibitor	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
αIL-4R	✓		✓						✓		
αIL-13			✓						✓		
αIL-17					✓						
αIL-31R				✓							
JAK1	✓	✓	✓	✓		✓		✓	✓	✓	✓
JAK3	✓					✓					✓
STAT6 Inhibitor	✓		✓	✓					✓		

Potent Inhibition of ITK and JAK3 with ATI-2138

Selective Oral Small Molecule Covalent ITK & JAK3 Inhibitor for I&I Disease

- Investigational oral compound which interrupts T cell receptor (TCR) signaling by inhibiting ITK and JAK3 signaling of common γ chain cytokines in lymphocytes (including IL-2 & IL-15)
- Highly potent for both ITK and JAK3 (IC_{50}: 0.2nM ITK; 0.5nM JAK3)
- Highly selective against other JAK isoforms
- Unique dual pharmacology; best-in-class potential
- Clinical data thus far demonstrate strong safety and PK characteristics; positive readout in a phase 2a AD study



Unique Dual Pharmacology of ATI-2138

Unique Dual Pharmacology of ATI-2138 Provides Best-in-Class Potential

High potency for inhibiting both ITK and JAK3

Regulation of T cell development and function both upstream (ITK) and downstream (JAK3)

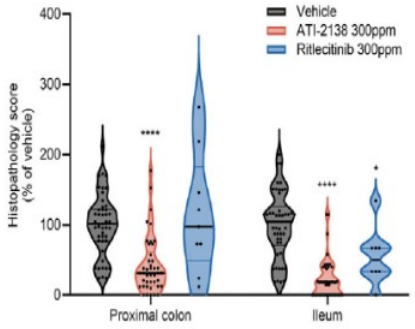
Inhibiting both pathways may provide a **more potent and complete anti-inflammatory response**

As both targets are restricted in expression to immune cells, inhibitors have the **potential for a favorable safety profile**

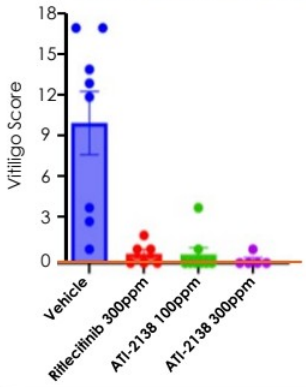
ATI-2138: Anti-Inflammatory Activity

Strong Anti-Inflammatory Activity Shown in Various Murine Models

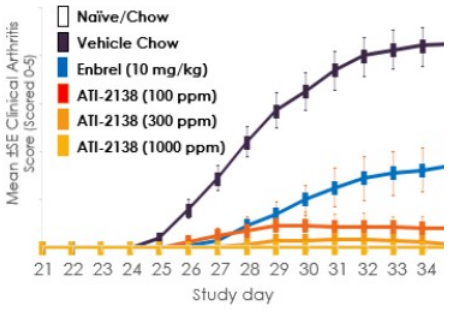
IBD
Mouse T Cell Transfer



Vitiligo
Mouse T Cell Transfer



Rheumatoid Arthritis
Mouse CIA Model

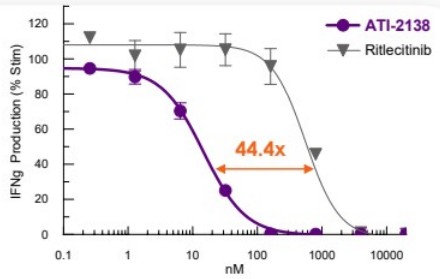


ATI-2138 has demonstrated robust anti-inflammatory activity in mouse models of disease:
Inflammatory Bowel Disease, Vitiligo, and Rheumatoid Arthritis

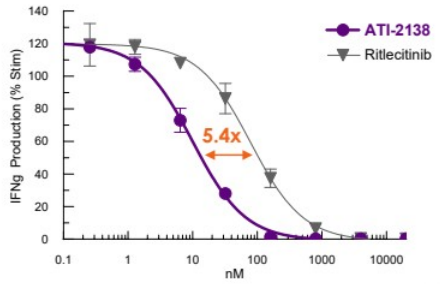
ATI-2138: Best-in-Class Potential

Unique Dual Pharmacology Creates Best-in-Class Potential

ITK: HWB αCD3 Stimulated IFNγ Release



JAK3: HWB IL2 Stimulated IFNγ Release



- **ATI-2138 is 44.4x more potent than ritlecitinib** for inhibiting anti-CD3 induced IFNγ production (ITK) and **5.4x more potent** for inhibiting JAK3 dependent IL-2 induced IFNγ production in human whole blood
- At the FDA recommended 50 mg QD dose for alopecia areata, ritlecitinib plasma levels may not impact ITK (anti-CD3 /IFNγ) for any appreciable time

ATI-2138: Validating the Potency Advantage

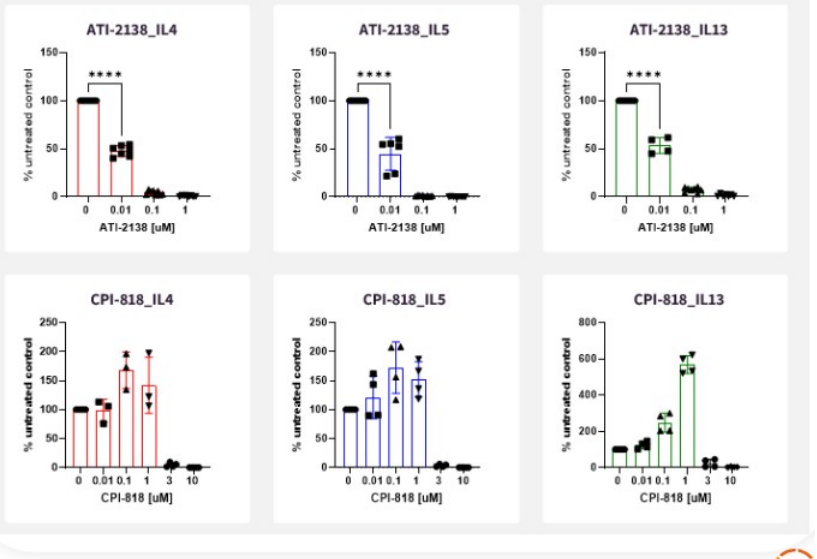
ATI-2138 Compared to CPI-818 (Soquelitinib)

ITK Biochemical Enzyme Potency

	ITK, IC50, nM	Kinact/Ki (uM-1s-1)
ATI-2138	0.25	0.34
CPI-818	9.5	0.022
Potency Ratio	**38x**	**15x**

- **ATI-2138 is 15-38x more potent than CPI-818** in inhibiting the ITK enzyme activity
- ATI-2138 is significantly more potent than CPI-818 in blocking the Th2 derived cytokines, IL-4, IL-5 and IL-13 (30-100x)

Anti-CD3/CD28-Induced Cytokines from Human Th2 Cells



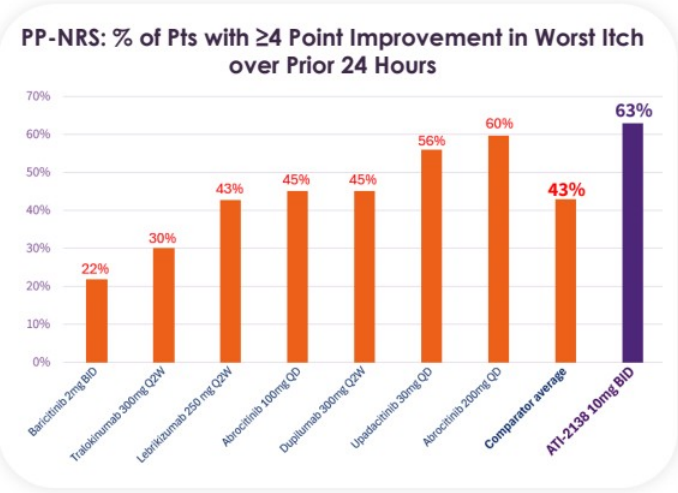
Phase 2a Trial in Atopic Dermatitis (AD): Complete

Results:

- Favorable safety profile
- Efficacy across multiple measures comparable to drugs approved for AD
- Exposure similar to or slightly higher than predicted from MAD study
- PD results validate therapeutic potential of targeting ITK through:
  - Near complete and sustained inhibition and occupancy of ITK
  - Downregulation of multiple ITK-dependent immune pathways in the skin

Phase 2a Trial in Atopic Dermatitis: Efficacy Result

A ≥4-point improvement in PP-NRS score is considered a clinically meaningful result



At week 12 (end of treatment), **63%** of patients receiving a low dose (10mg BID) of ATI-2138 experienced a ≥4-point improvement worst itch in the past 24 hours

At week 12: **60.5% mean (median = 76.8%) improvement in EASI** score in patients (n=10) receiving 10mg BID of ATI-2138

Rapid response: After four weeks of treatmen

- **BSA** decreased by **63.9%** (p<0.001)
- **EASI** scores dropped by **77.3%** (p<0.001)
- **PP-NRS** decreased by **44.7%** (p<0.01)
- These changes were statistically significant and sustained through study treatment (W12)

*Molecular and Clinical Effects of oral ATI-2138, an ITK/JAK3 inhibitor, in Moderate-to-Severe Atopic Dermatitis: Sub-study of a Phase 2a Open-Label, Single-Arm Trial. Beaziz-Tordjman, Jessica *et al.* European Academy of Dermatology and Venereology. September 17, 2026.

Efficacy Results Demonstrated Strong + Consistent Response to ATI-2138

Significant Itch Relief + Improvements in Disease Severity and Extent

Each other compound efficacy is the average percent improvement from multiple studies, at week 12 or 16 data from Phase 2 and Phase 3 published sources; head-to-head clinical studies have not been conducted. Differences exist between trial designs and subject characteristics, and caution should be exercised when comparing data across studies. Data collected on 8 of 10 PP patients.

Phase 2a Trial in Atopic Dermatitis: PD Assessmen

Pharmacodynamic Assessments of ATI-2138

Conducted to Assess **Target, Pathway, and Disease Markers** to Support Mechanism of Action

Understanding PK/PD

- **ITK Assay**
  - αCD3/aCD28 ex vivo stim mRNA (IL-2 and IFN γ) production
- **ITK Target Occupancy**
- **JAK3 Assay**
  - IL-15 ex vivo stim IFNg protein production
- **Immunophenotyping**

Relating PD to Efficacy

- **Punch Biopsy Analysis**
  - Immunohistochemistry
  - RNAseq Analysis (>16,000 genes)
- **Tape Strip Analysis**
  - RNAseq Analysis (>16,000 genes)
  - Olink Proteomics (300+ analytes)
- **Endogenous Biomarkers in Plasma**
  - OLink Proteomics (300+ analytes)

ATI-2138 is Mechanistically Unique; PD Supported Observed Clinical Efficacy

Phase 2a Trial in Atopic Dermatitis: PD Assessmen

ITK Pathway Mediated Anti-Inflammatory Activity in Skin and Plasma

- Marked and sustained target occupancy and functional ITK inhibition across dosing interval
- ATI-2138 significantly downregulated multiple immune pathways in skin and plasma, with reduction of inflammation
- Strong downregulation of key ITK dependent pathway markers such as:
  - Th2 (e.g., CCL17, CCL24, IL13, TSLP)
  - Th17 (e.g., CXCL1, IL17A, IL6R)
  - TCR (ITK) Pathway (e.g., ITK, IL-13, CD3, ZAP70, LCK, PLCg1)
  - Th1 (e.g., CXCL11, CXCL9, IL2RA, TNF)
  - Fibrosis related markers (e.g., MMP9, TNFRSF9)
- Safety profile and expected incremental increase in PD with greater exposure may support higher dosing in subsequent studies

ATI-2138: Next Steps

Oral Small Molecule Covalent ITK & JAK3 Inhibitor for I&I Disease

Phase 1
HV: SAD/MAD
Complete

Phase 2a
Atopic dermatitis
Complete

Phase 2
Additional indication:
e.g., alopecias
Planned Initiation: 1H 2026

Potential targets
Alopecia areata
Lichen planus
Prurigo nodularis
Vitiligo
others

Potential applicability in a variety of I&I indications based on dual pharmacology / MOA

Ongoing / Next Steps

- Finalize trial design, assessment, TPP of next targets
- Planned Phase 2 in additional indication in 1H 2026

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Next Generation JAK-Sparing ITK Inhibitors

Novel Selective Inhibitors Designed to Limit JAK Inhibitory Activity

Next Generation JAK-Sparing ITK Inhibitor Prograr

Progressing Toward IND

- Developing highly potent JAK-sparing compounds; significantly more efficient at inactivating ITK than CPI-818
- Extended half life compared to comparator molecules
- Potent blockade of Th2 function and differential modulation of Th1 activation
- Opportunity for total ITK occupancy at low doses without impacting JAK3 function

Targeting first IND from JAK-sparing ITK inhibitor program in 2026

Significant Market Available for Next Gen ITKi

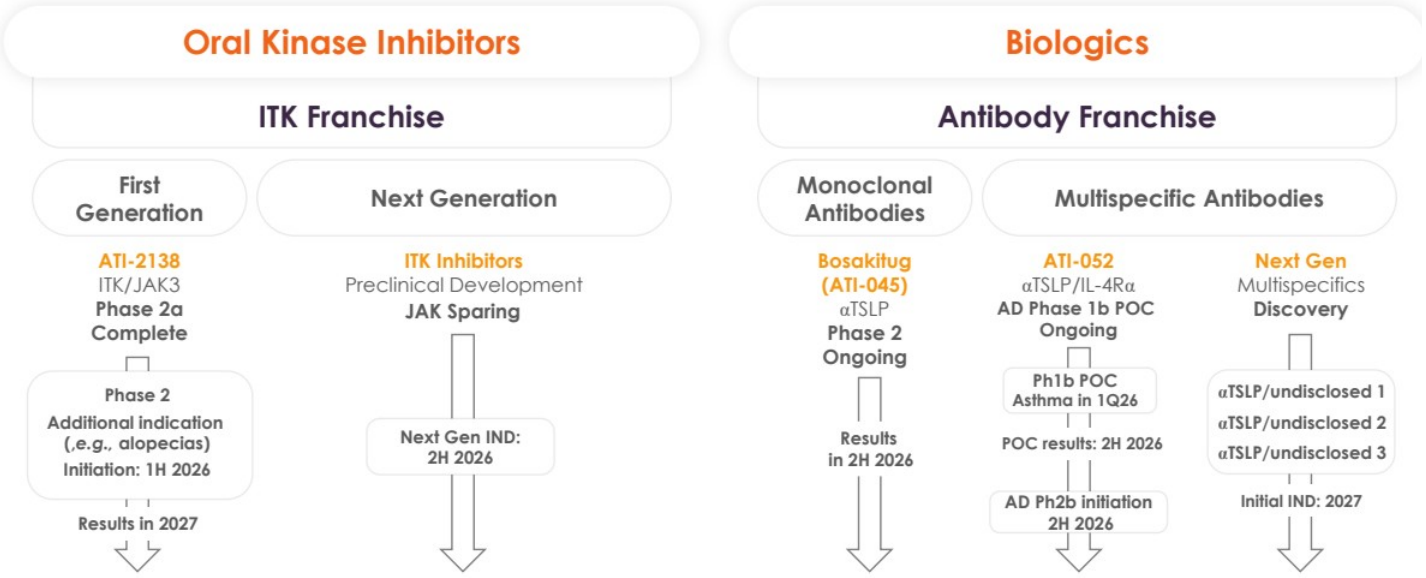
	Potential Indications	Approved Inhibitors	Select TAMs*
ITK/TXK Potentially beneficial in Th1, Th2, and Th17-driven diseases	• Psoriasis • Celiac disease • IBD • Alopecia • SO Transplant Rejection • Eosinophilic esophagitis • Vitiligo • Prurigo nodularis	IL-4R: Dupixent® (dupilumab) IL-13: Ebglyss® (lebrikcizumab) Adbry® (tralokinumab) IL-31R: Nemluvio® (nemolizumab) IL-17A: Cosentyx® (secukinumab) Taltz® (ixekizumab) Bimzelx® (bimekizumab) Siliq® (brodalumab) JAK1: Oluminant® (baricitinib) Renvoq® (upadacitinib) Cibinqo® (abrocitinib) JAK1/2: Opzelura® (ruxolitinib) JAK3: Litfulo® (ritlecitinib)	Psoriasis: $60B Asthma: $36B COPD: $31B Atopic dermatitis: $31B Rhinitis: $19B Alopecia areata: $7B CSU: $6B EoE: $5B Vitiligo: $3B Prurigo nodularis: $2B
ITK Potentially beneficial in Th2-driven atopic and allergic diseases	• Asthma • COPD • Atopic Dermatitis • Rhinitis • CSU • Others		

*TAM=Total Addressable Markets: Estimates, 2028-2034

Sources: Eczema stats: National Eczema Association (accessed 07/31/25); National Alopecia Areata Foundation (Accessed 07/31/25); Vitiligo Facts: Global Vitiligo Foundation (accessed 07/31/25); Precedence Research; Forbes Business Insights; American Medical Association; American Lung Association; Global Initiative for Asthma; World Health Organization; The Centers for Disease Control and Prevention (CDC); Business Research Company; peer research; Delveinsight; Cowen Categories Outlook 2024

Broad Clinical and Preclinical I&I Pipeline

Developing Potential Best-in-Class Inhibitors of Immunoinflammatory Cascad



 All future development, clinical, and regulatory timelines are expectations, are based on current beliefs and assumptions, and are subject to change based on a variety of factors

Continued Clinical Momentum in 2026 and 2027

Four Expected Clinical Programs in 2026

Innovation Driven

Patient Focused

ATI-052

AD POC ongoing; Asthma POC: 1Q 2026

Top line POC results in AD and Asthma in 2H 2026

Start AD Phase 2b: 2H 2026

Next generation Therapeutics

First IND from JAK-sparing ITK inhibitor program in 2H 2026

First IND from multispecific antibody program in 2027

ATI-2138

Start Phase 2 in additional indication *e.g.*, alopecias in 1H 2026

Top line results in 2027

Bosakitug (ATI-045)

Phase 2 in AD ongoing

Top line results in 2H 2026

Advancing potentially industry-leading inhibitor franchises designed to address validated, therapeutically-relevant immune targets

All future development, clinical, and regulatory timelines are expectations, are based on current beliefs and assumptions, and are subject to change based on a variety of factors

Company Summary

Commitment to Patients

Focus on addressing the needs of patients with immuno-inflammatory diseases who lack satisfactory treatment options

State-of-the-Art Discovery Platform

Integrated approach to small and large molecule discovery enables targeted design of novel product candidates from concept through lead optimization

Diversified Pipeline

Executing on multiple therapeutic programs from discovery to clinical development

Multiple milestones expected in 2026 and beyond

Executive Team

Proven track record of R&D, business development, and scientific leadership in immuno-inflammatory diseases

Intellectual Property

Global IP estate

Financial Strength

Cash, cash eq., and marketable securities as of 3Q25 of $167M

Cash runway expected into the second half of 2028*

Potential to extend runway further through non-dilutive opportunities

* Without giving effect to additional business development transactions, financing activities, and trial execution costs associated with trials in our pipeline notated as in planning

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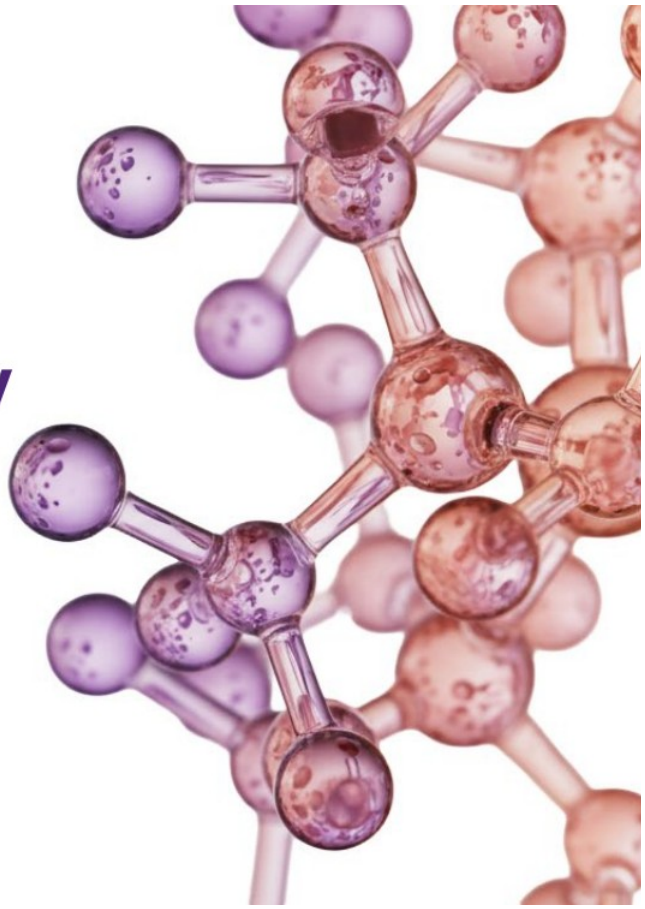
Corporate Overview

January 2026



EMPOWERING PATIENTS THROUGH

THERAPEUTIC INNOVATION