NORTH IMMUNOLOGY

Company Overview

SEPTEMBER 2026

Disclaimer and Forward-looking Statements

Disclaimer

This presentation (together with any information communicated orally in connection herewith, this "Presentation") is being provided by North Immunology, Inc. ("North" or the "Company") on a confidential basis solely for informational purposes in connection with a proposed private placement of securities (the "Private Placement") to be conducted concurrently with a proposed business combination between North and Aethlon Medical, Inc. (Nasdaq: AEMD) ("AEMD") pursuant to which a successor entity to North would become a wholly owned subsidiary of AEMD (the "Transaction"). By accepting this Presentation, the recipient agrees to keep its contents confidential, not to reproduce or distribute it in whole or in part, and to return or destroy it upon request. This Presentation does not purport to be all-inclusive or to contain all information a prospective investor may require and is qualified in its entirety by the definitive transaction agreements and the disclosure documents referred to below

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Additional Information and Where to Find It

In connection with the Transaction, AEMD intends to file with the U.S. Securities and Exchange Commission (the "SEC") a registration statement on Form S-4 (the "S-4") that will contain a proxy statement of AEMD and a prospectus of AEMD (the "proxy statement/prospectus"). After the S-4 is declared effective by the SEC, the definitive proxy statement/prospectus will be mailed to AEMD's stockholders. INVESTORS AND SECURITY HOLDERS OF AEMD AND NORTH ARE URGED TO READ THE S-4, THE PROXY STATEMENT/PROSPECTUS AND ANY OTHER RELEVANT DOCUMENTS FILED OR TO BE FILED WITH THE SEC, AS WELL AS ANY AMENDMENTS OR SUPPLEMENTS THERETO, CAREFULLY AND IN THEIR ENTIRETY WHEN THEY BECOME AVAILABLE, BECAUSE THEY WILL CONTAIN IMPORTANT INFORMATION ABOUT AEMD, NORTH, THE TRANSACTION AND RELATED MATTERS. Investors and security holders will be able to obtain free copies of the S-4 and the proxy statement/prospectus (when available), and other documents filed with the SEC by AEMD, through the SEC's website at www.sec.gov and on the investors section of AEMD's website.

Participants in the Solicitation

AEMD, North and their respective directors and executive officers may be deemed to be participants in the solicitation of proxies from AEMD's stockholders in connection with the Transaction. Information about AEMD's directors and executive officers, including a description of their interests in AEMD, is included in AEMD's most recent definitive proxy statement, as filed with the SEC on September 1, 2026, and in AEMD's Annual Report on Form 10-K for the fiscal year ended March 31, 2026. To the extent that holdings of AEMD's securities by AEMD's directors and executive officers have changed since the amounts set forth in AEMD's most recent definitive proxy statement, such changes have been or will be reflected on Statements of Change in Ownership on Forms 3, 4 or 5 filed with the SEC. Additional information regarding the persons who may, under the rules of the SEC, be deemed participants in the solicitation of AEMD's stockholders in connection with the Transaction, including a description of their direct or indirect interests, by security holdings or otherwise, will be included in the S-4 and the proxy statement/prospectus and other relevant materials to be filed with the SEC when they become available.

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Forward-Looking Statements

This Presentation contains "forward-looking statements" within the meaning of the federal securities laws, including for purposes of the safe harbor provisions under the Private Securities Litigation Reform Act of 1995. All statements other than statements of historical fact are forward-looking statements, including, without limitation, statements regarding the proposed Transaction and Private Placement and their expected timing and completion; the combined company's expected cash position andcash runway; NOR-101's and North's other product candidates' target profiles, anticipated benefits, mechanism, dosing, and development plans; the timing and design of preclinical studies and clinical trials and the expected timing of data; market size and opportunity; and the combined company's strategy, prospects and future operations. Words such as "anticipate," "believe," "expect," "intend," "may," "plan," "potential," "project," "target," "will" and similar expressions identify forward-looking statements. These statements are based on current expectations and assumptions and are subject to known and unknown risks and uncertainties (many beyond the parties' control) that could cause actual results to differ materially, including, without limitation: the risk that the Transaction or the Private Placement may not be completed on the anticipated timeline or at all; the failure to satisfy closing conditions, including obtaining required stockholder approvals, the effectiveness of the S-4, receipt of the minimum financing, and stock exchange listing approval; the outcome of preclinical studies and clinical trials; regulatory processes and the possibility that NOR-101 target profiles are not achieved; the fact that NOR-101 is investigational and cross-trial comparisons are not head-to-head; competition; reliance on third parties; intellectual property risks; and the need for substantial additional funding. Additional risks will be described in the S-4 and the proxy statement/prospectus and in AEMD's filings with the SEC. Forward-looking statements speak only as of the date of this Presentation, and the parties undertake no obligation to update them except as required by law. You should not place undue reliance on forward-looking statements.

Investigational Product Candidates

NOR-101 and North's other product candidates are investigational and have not been approved by the U.S. Food and Drug Administration or any other regulatory authority. Their safety and efficacy have not been established, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated. Comparisons to approved products and to other investigational product candidates are based on separate studies with different designs, endpoints, timepoints and patient populations; no head-to-head studies have been conducted, and such comparisons are for illustrative purposes only.

Industry and Market Data

Certain information in this Presentation regarding the market and industry in which North operates, including market size, position and opportunity, is based on the Company's estimates and on third-party sources, internal analyses and assumptions that the Company believes to be reasonable but that have not been independently verified. Such information is inherently uncertain and subject to change.

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NOR-101 is a Half-life Extended Anti-IL-13 x IL-18 Bispecific Antibody

Potential to deliver best-in-disease treatment for atopic dermatitis (AD)by targeting both Th2 and non-Th2 inflammation

NOR-101 Target Product Profile

Efficacy: Improved efficacy vs Th2 inhibitors

- Inhibit both Th2 and non-Th2 inflammation

Safety: In-line with Th2 inhibitors; potentially reduced conjunctivitis

- Potential to reduce IL-13 associated conjunctivitis by co-inhibiting IL-18

Extended Dosing: Potential for Q3-6M dosing

- NHP half-life exceeds best-in-class half-life extended monoclonal antibodies

IL-13 Inhibition:

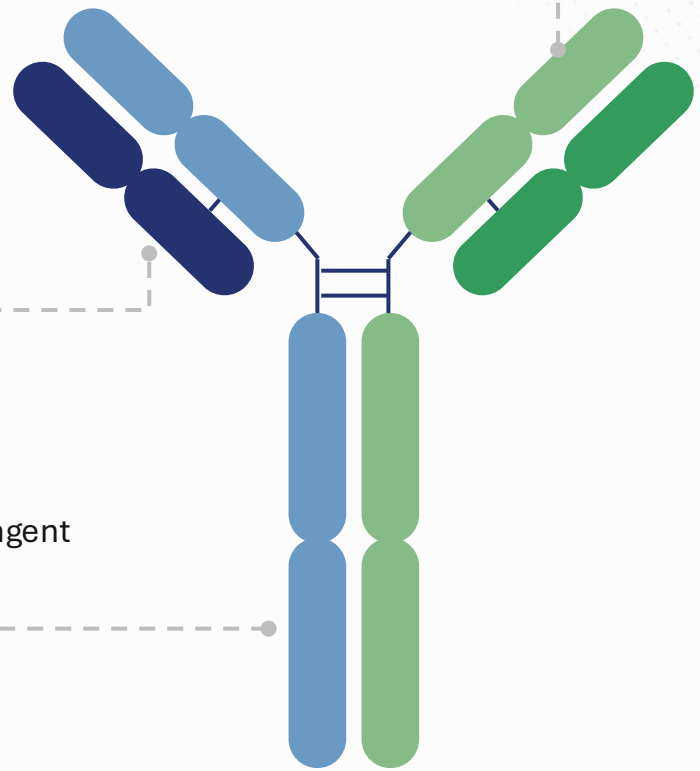
- Target Th2 Inflammation
- Lebrikizumab epitope
- Improved potency and developability vs lebrikizumab

IL-18 Inhibition:

- Target non-Th2 inflammation
- Aletekitug epitope
- Potential to reduce conjunctivitis
- Validated through 4 placebo-controlled studies of single agent IL-18 inhibition in AD

Engineered Fc:

- Half-life extended through validated Fc modification
- Effector Silenced



Target Phase 1a Start: Q1 2027

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Note: No clinical studies of NOR-101 have been conducted and results from clinical trials of other agents are not indicative of results that may be demonstrated in clinical studies of NOR-101

Better Treatments Are Required for Atopic Dermatitis

	Dupixent	Ebglyss	Nemluvio	Rinvoq
Target	IL-4Rα	IL-13	IL-31	JAK1
Pathway	Th2	Th2	Th2	Th2 + non-Th2
Dosing	Q2W	Q4W	Q4W	QD
Safety	✓	✓	✓	X
Efficacy	–	–	–	✓

Th2 therapies are safe, but result in clear skin in <30% of patients

Rinvoq delivers strong efficacy by targeting Th2 + non Th2 inflammation, but has safety liabilities

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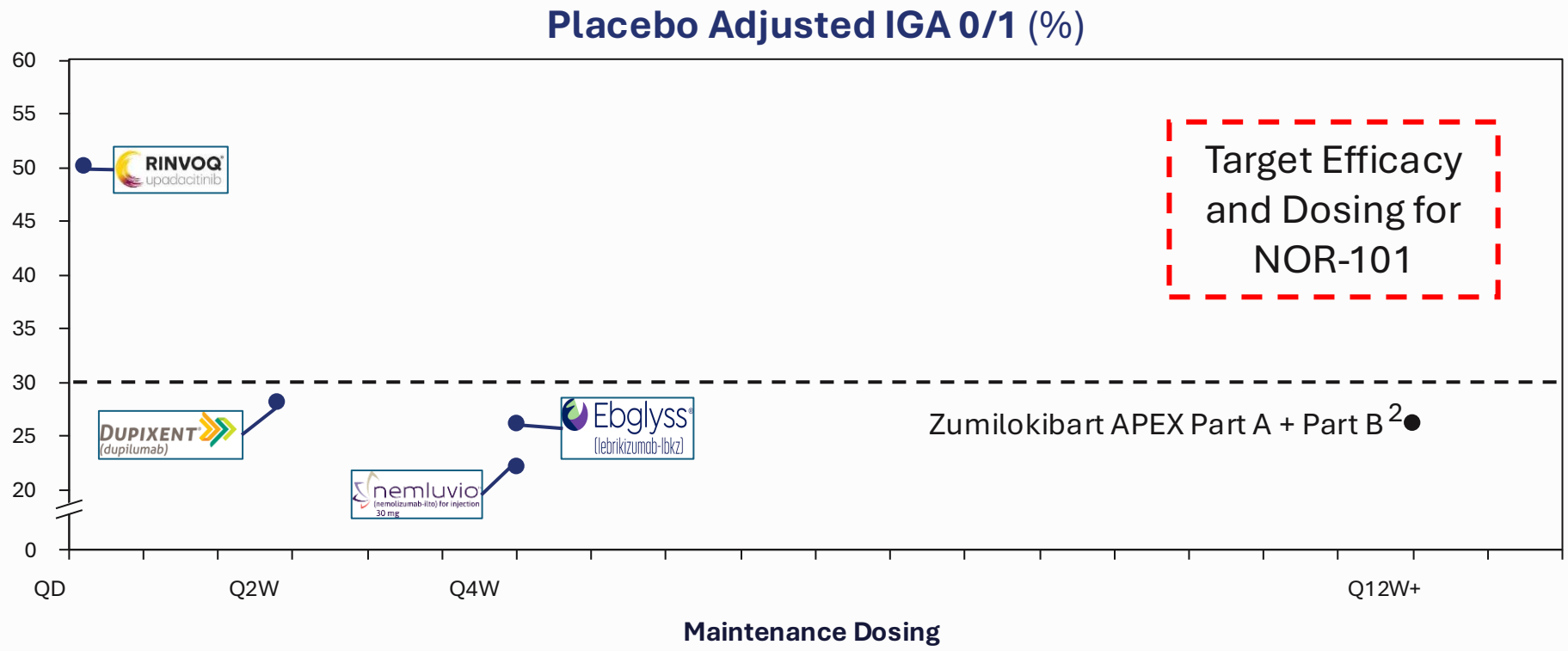
Source: FDA Labels **Note:** This chart is meant to show the treatment landscape for atopic dermatitis and the target therapeutic profile for NOR-101. No clinical studies of NOR-101 have been conducted and results from clinical trials of other agents are not indicative of results that may be demonstrated in clinicalstudies of NOR-101

We Believe NOR-101 has the Potential to Offer a Material Improvement to the Standard of Care

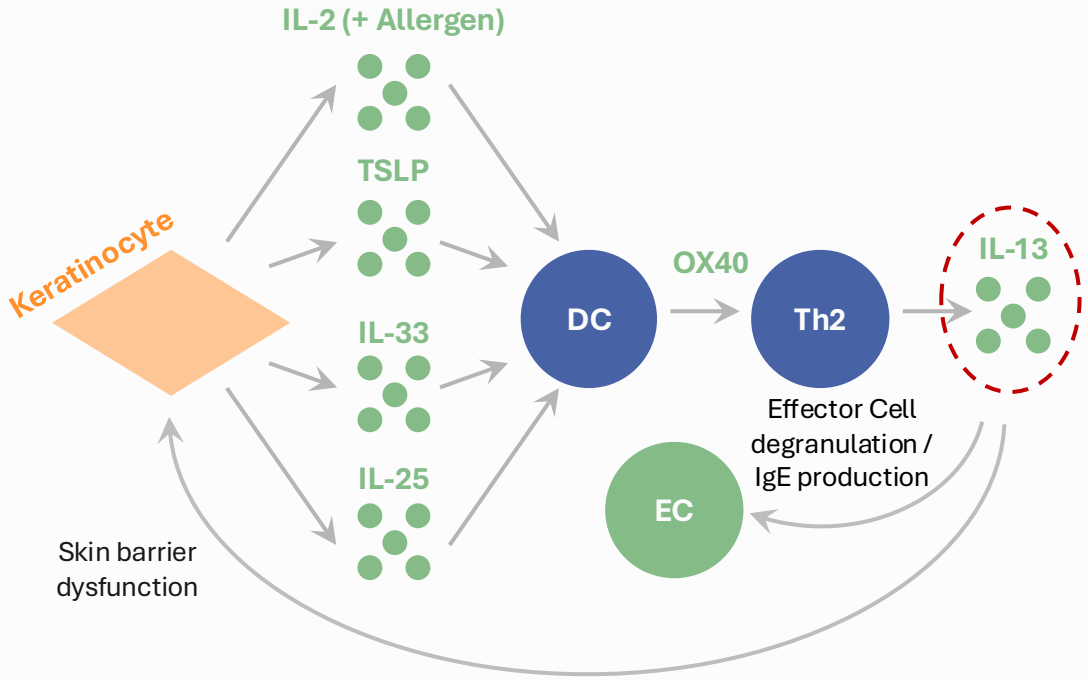


If NOR-101 is able to demonstrate clinical data in line with its target profile, we believe it would be a material improvement to current standard-of-care

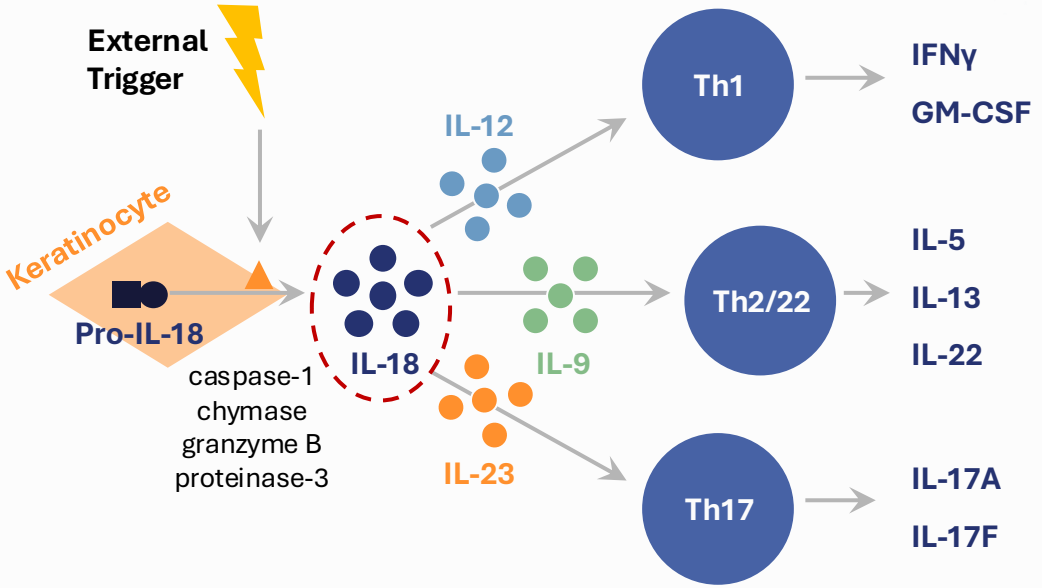
Source: FDA Labels; APGE APEX Part A results presentation; APGE APEX Part B results presentation **Note:** [1]Average of APEX Part A and APEX Part B; No clinical trials have been conducted for NOR-101. This chart is meant to show the treatment landscape for atopic dermatitis and what we believe NOR101 has the potential to demonstrate when tested in clinical trials. No clinical studies of NOR-101 have been conducted and results from clinical trials of other agents are not indicative of results that may be demonstrated in clinical studies of NOR-101

AD is Not Just a Th2 Disease; IL-18 is a Key Driver of Non-Th2 Inflammation

IL-13 mediates Th2 inflammation in AtD[1]



IL-18 drives heterogeneous non-Th2 inflammation[2,3,4]



Combined IL-13 and IL-18 inhibition targets both the Th2 and Non-Th2 inflammatory pathways associated with AD

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Source: [1]Fania et al., *Int J Mol Sci (2022)*. https://doi.org/10.3390/ijms23052684. [2]Lee et al., *PLoS One (2017)*. https://doi.org/10.1371/journal.pone.0186351. [3]Scharli et al., *J Allergy Clin Immunol (2024)*. https://doi.org/10.1016/j.jaci.2024.10.027. [4]Lusty et al., *Mol Immunol (2017)*. https://doi.org/10.1016/j.molimm.2017.06.025; North Immunology Analysis

Robust Evidence Across Genetic, Biomarker, and Clinical Data Implicate IL-18 as a Key Driver of AD

Genetics[1]

European Discovery Dataset

Gene	OR	P value
IL18Rβ	0.91	8.14E-35
IL2Rα	1.10	1.98E-20
OX40L	1.07	1.85E-17
IL7R	0.94	5.97E-16
IL22	1.05	1.46E-25
IL15	1.04	7.30E-09

Major GWAS meta-analysis found IL-18 was strongly linked to AD

Biomarker[2,3]

33x

IL-18 elevation in dermis of chronic AD patients

100x

IL-18 elevation in lesional epidermis

Multi-fold elevations in lesional AD skin

Clinical[4,5,6,7]

GSK	aletekitug
NOVARTIS	CMK389
APOLLO THERAPEUTICS	camoteskimab
evommune	EVO301

Robust efficacy signal and clean safety from 4 placebo-controlled studies

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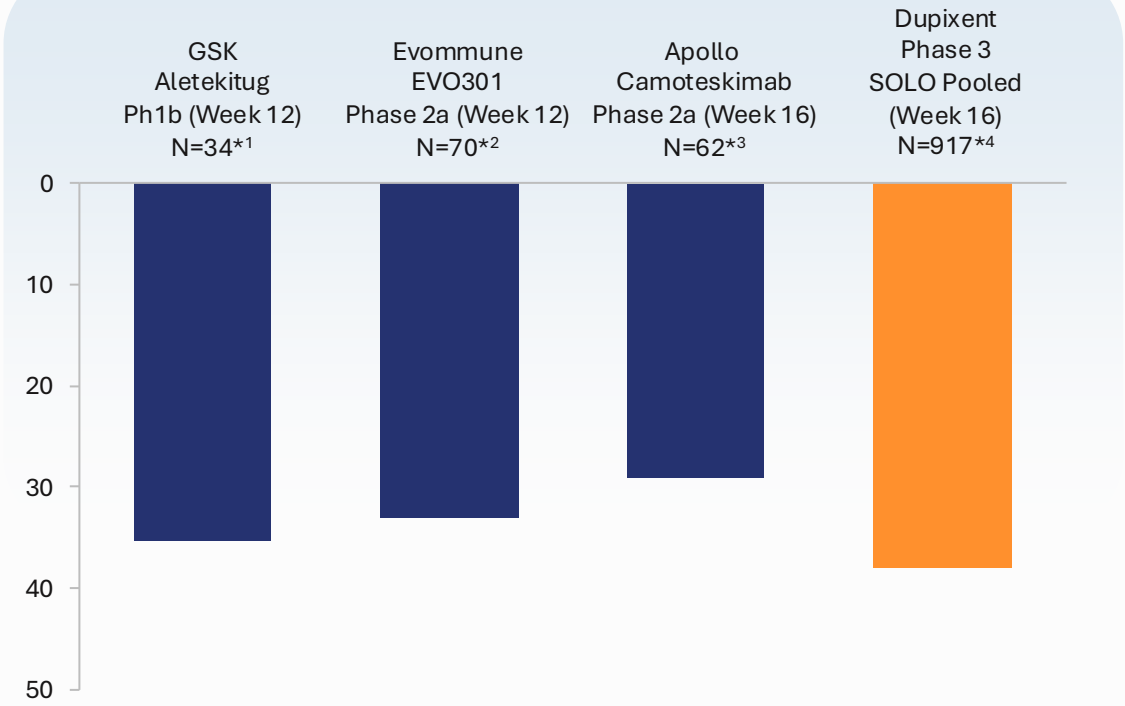
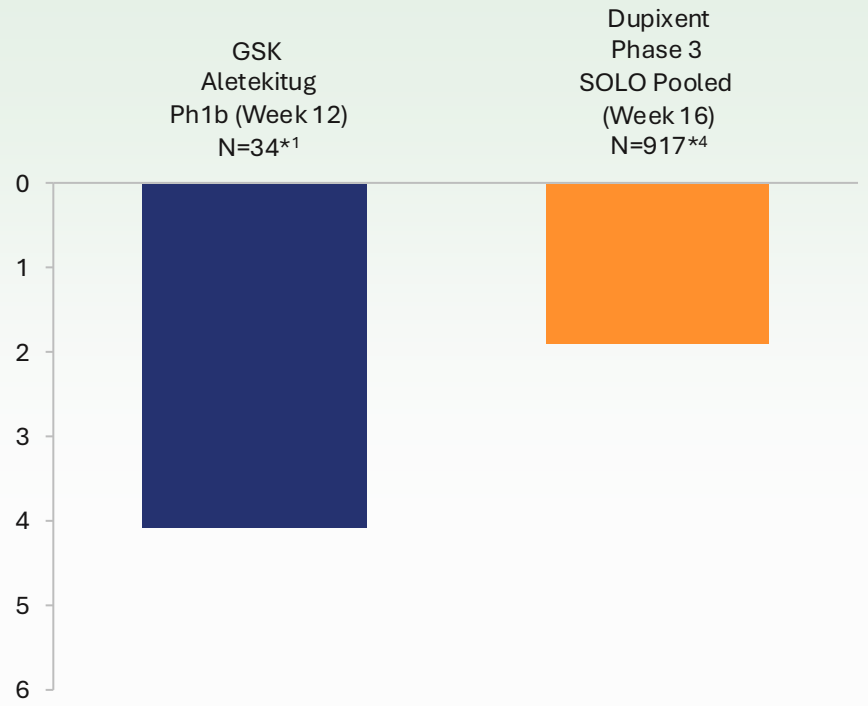
Source: [1]Budu-Aggrey et al., *Nat Commun* (2023). https://doi.org/10.1038/s41467-023-41180-2. [2]Szegedi et al., *J Eur Acad Dermatol Venereol* (2015). https://doi.org/10.1111/jdv.13160. [3]Clausen et al., *Sci Rep* (2020). https://doi.org/10.1038/s41598-020-78943-6. [4]Ellis et al., *Allergy* (2025). https://doi.org/10.1111/all.70172.; [5]Evommune, EVO301 Top-Line Results, 2026; [6]Silverberg et al., SID 2026; [7]Novartis, https://www.novctrd.com/ctrdweb/patientsummary/patientsummaries?patientSummaryId=1800

IL-18 Directed Therapies Have Demonstrated Efficacy across Both Lesions and Itch, with a Favorable Safety Profile, and No Reported Cases of Conjunctivitis

Placebo-Adjusted PCFB EASI (%)



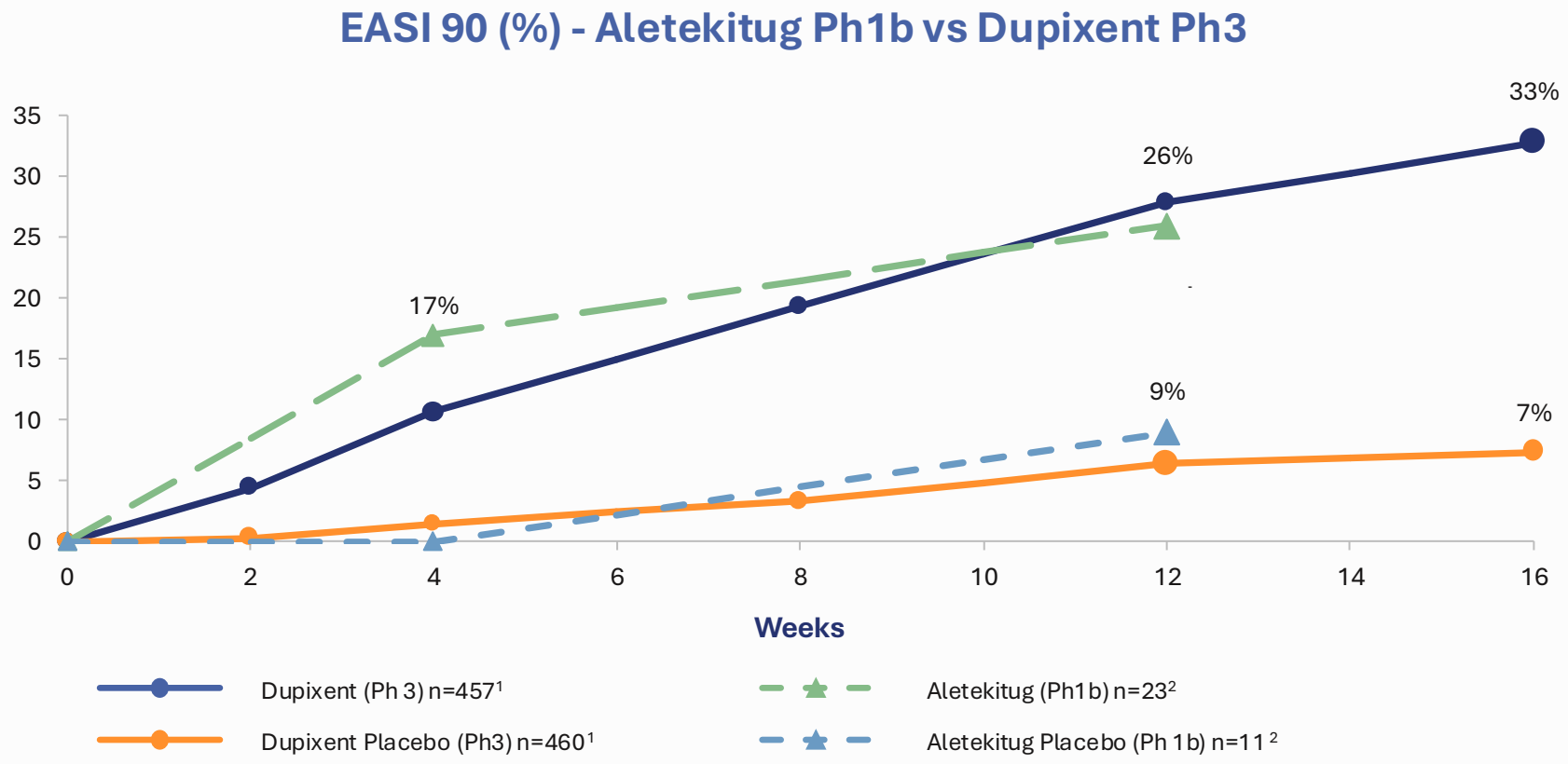
Placebo-Adjusted PP-NRS Reduction (median)



Source: [1]Ellis et al., Allergy (2025). https://doi.org/10.1111/all.70172. [2]Evommune, EVO301 Top-Line Results (2026). [3]Silverberg et al., SID (2026). [4]Cather et al., Dermatol Ther (Heidelb) (2022). https://doi.org/10.1007/s13555-022-00778-y.

Note: *N= total study N. Data are from separate studies and are not from head-to-head trials. Differences in study design, endpoints and timepoints, entry criteria and baseline disease severity, background/concomitant therapy, rescue medication rules, geography, and statistical handling of missing data may preclude direct comparison. No definitive conclusions regarding relative efficacy or safety should be drawn. No clinical studies of NOR-101 have been conducted and results from clinical trials of other agents are not indicative of results that may be demonstrated in clinical studies of NOR-101

IL-18 Inhibition Drives Rapid and Deep Responses



A single 2 mg/kg dose of aletekitug demonstrated EASI90 in line with Dupixent dosed at 300mg Q2W

Source: [1]Cather et al., Dermatol Ther (Heidelb) (2022). https://doi.org/10.1007/s13555-022-00778-y. [2]Ellis et al., Allergy 81:539–551 (2025). https://doi.org/10.1111/all.70172. **Note:** Data are from separate studies and are not from head-to-head trials. Differences in study design, endpoints and timepoints, entry criteria and baseline disease severity, background/concomitant therapy, rescue medication rules, geography, and statistical handling of missing data may preclude direct comparison. No definitive conclusions regarding relative efficacy or safety should be drawn. No clinical studies of NOR-101 have been conducted and results from clinical trials of other agents are not indicative of results that may be demonstrated in clinical studies of NOR-101

IL-18 mAbs Have Demonstrated a Clean Safety Profile and Human Genetic Evidence Suggests That Long-term IL-18 Inhibition is not Deleterious

Clinical Data

- Clinical trials of IL-18 inhibitors have not shown treatment related safety-signals to date
- No cases of conjunctivitis reported across 4 different anti-IL-18 programs[1-4]
- Aletekitug studied in 3 Phase 2 studies up to 5mg/kg[5-7] in addition to phase 1b study in atopic dermatitis
- Compassionate use case for IL-18opathy associated IBD dosed up to 10mg/kg with no long term tolerability issues[8]

Genetics

- No known IL-18 LoF phenotype
- Loss of endogenous negative feedback via IL-18BP or IL-37 LoF associated with fulminant hepatitis[9] and colitis[10] respectively
- Homozygous CASP1 LoF with undetectable IL-18 levels and no change in life expectancy or increased risk of infection[11]

Source: [1]Ellis et al., *Allergy* (2025). https://doi.org/10.1111/all.70172. [2]Evommune, *EVO301 Top-Line Results* (2026). [3]Silverberg et al., SID (2026). [4]Novartis. https://www.novctrd.com/ctrdweb/patientsummary/patientsummaries?patientSummaryId=1800. [5]ClinicalTrials.gov, NCT06447506. https://clinicaltrials.gov/study/NCT06447506. [6]McKie et al., *PLoS One* (2016). https://doi.org/10.1371/journal.pone.0150018. [7]Wlodek et al., *PLoS One* (2021). https://doi.org/10.1371/journal.pone.0247972. [8]Guha et al., *J Clin Med* (2024). https://doi.org/10.3390/jcm13206058. [9]Belkaya et al., *J Exp Med* (2019). https://doi.org/10.1084/jem.20190669. [10]Zhang et al., *Proc Natl Acad Sci U S A* (2021). https://doi.org/10.1073/pnas.2009217118. [11]Dominy et al., *J Allergy Clin Immunol* (2026). https://doi.org/10.1016/j.jaci.2025.09.025.

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Dual IL-13 x IL-18 Inhibition has the Potential to Drive Deeper and Broader Responses than IL-13 Inhibitor Monotherapy

1. **Clinical Data:** Strong responses to IL-18 inhibition in Dupixent refractory patients
2. **Biomarkers:** Dupixent non-responders display a Th1-skewed immune signature; Dupixent responders display a Th2 dominant signature
3. **In Vitro:** IL-13 x IL-18 inhibition demonstrated orthogonality and additivity
4. **Clinical Observation:** Th switching implicated in Dupixent loss of response

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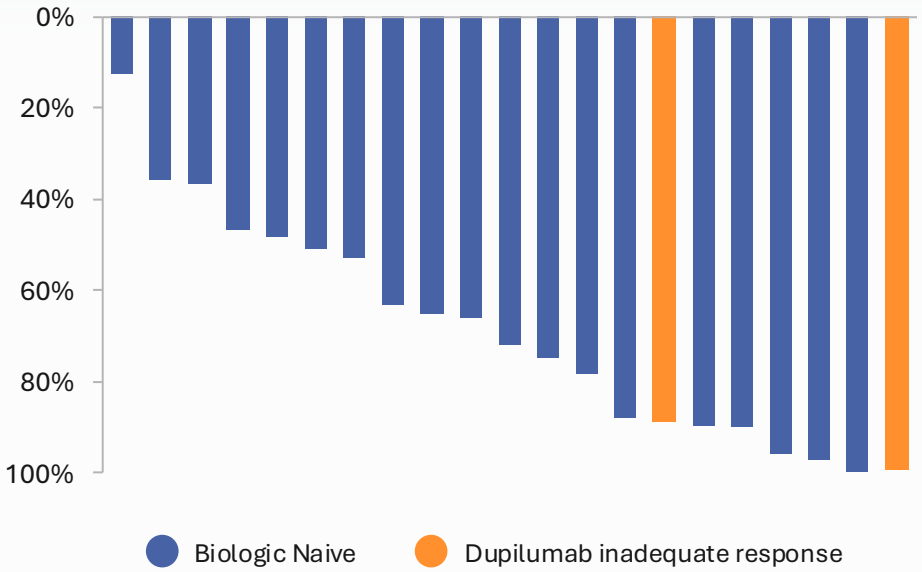
Dual IL-13 x IL-18 Inhibition has the Potential to Drive Deeper and Broader Responses than IL-13 Inhibitor Monotherapy

1 **Clinical Data:** Strong responses to IL-18 inhibition in Dupixent refractory patients

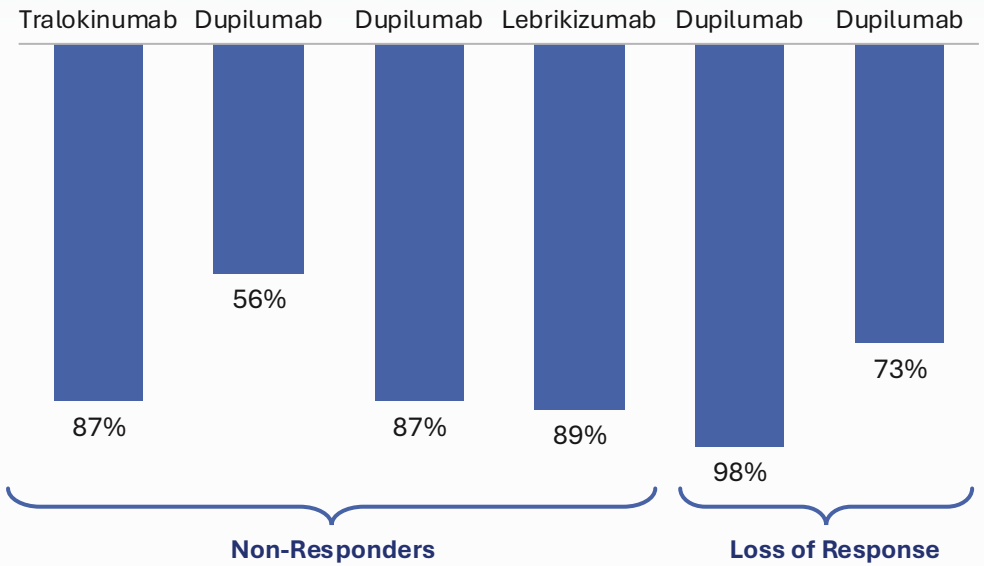
Aletekitug Phase 1b[1]

PCFB EASI at 12 weeks (%)



Camoteskimab Phase 2a SID 2026[2]

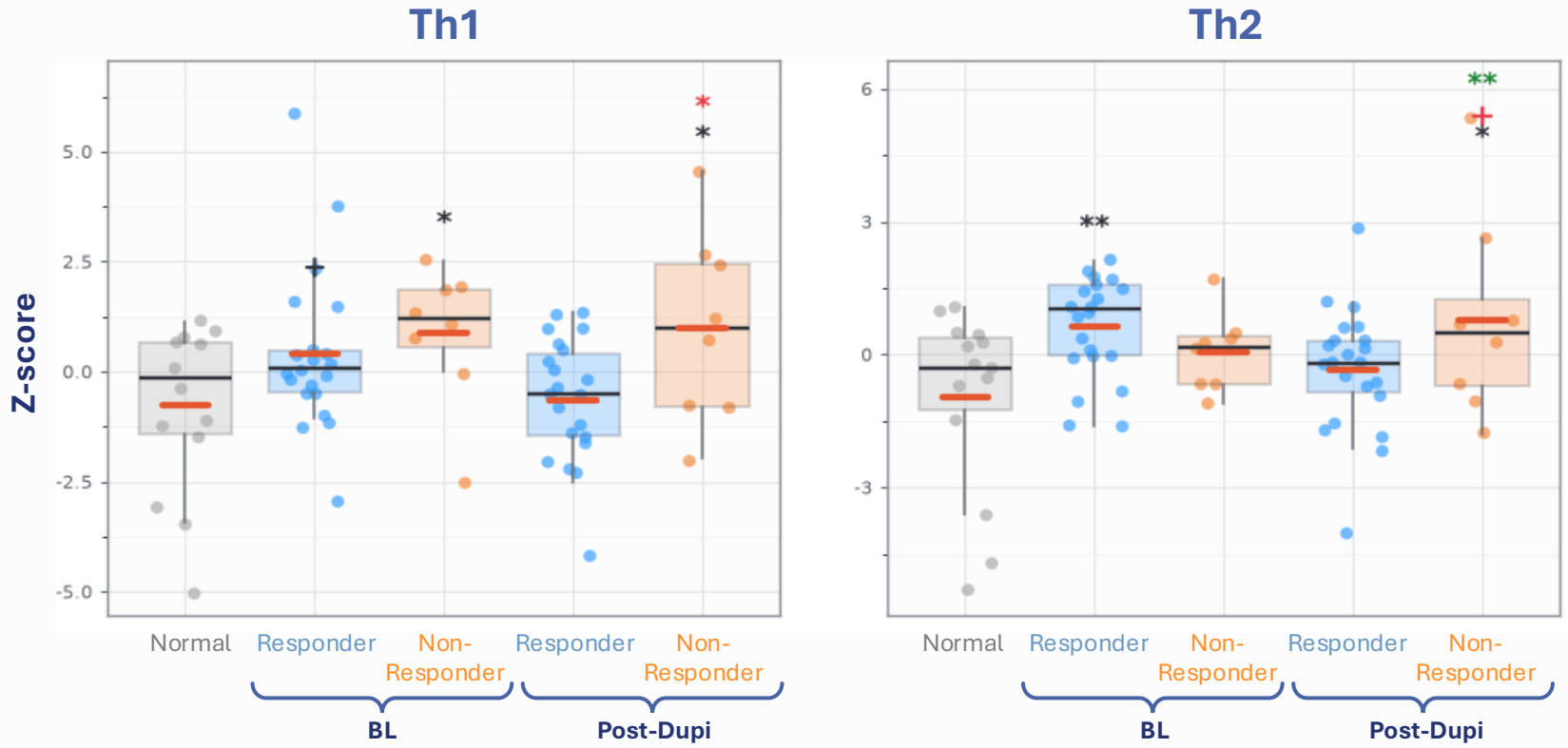
Best PCFB EASI over 32 weeks (%)



Source: [1]Ellis et al., *Allergy* (2025). https://doi.org/10.1111/all.70172. [2]Silverberg et al., SID 2026. **Note:** Results from clinical trials of other agents are not indicative of results that may be demonstrated in clinical studies of NOR-101

Dual IL-13 x IL-18 Inhibition has the Potential to Drive Deeper and Broader Responses than IL-13 Inhibitor Monotherapy

2 **Biomarkers:** Dupixent non-responders display a Th1-skewed immune signature; Dupixent responders display a Th2 dominant signature



Source: Adapted from Del Duca et al, Serum biomarkers accurately differentiate dupilumab responders from nonresponders in atopic dermatitis, SID 2026 **Note:** Results from clinical trials of other agents are not indicative of results that may be demonstrated in clinical studies of NOR-101

Dual IL-13 x IL-18 Inhibition has the Potential to Drive Deeper and Broader Responses than IL-13 Inhibitor Monotherapy

3 **In Vitro:** IL-13 x IL-18 inhibition demonstrated orthogonality and additivity

Promise of Additive Efficacy

- Dual targeting of IL-13 and IL-18 increased Filaggrin expression more than either alone
- IL-18 inhibition synergizes with IL-13 inhibition to decrease CCL26, a key Th2 biomarker

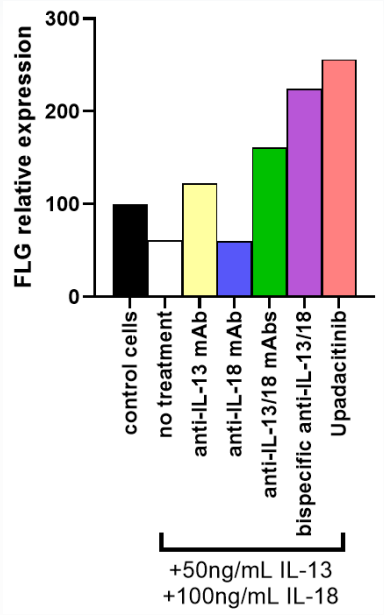
Suggestive of Orthogonal Mechanisms

- Targeting IL-18 alone did not impact CCL26, suggesting that the clinical efficacy seen with IL-18 inhibition is not driven by Th2 activity

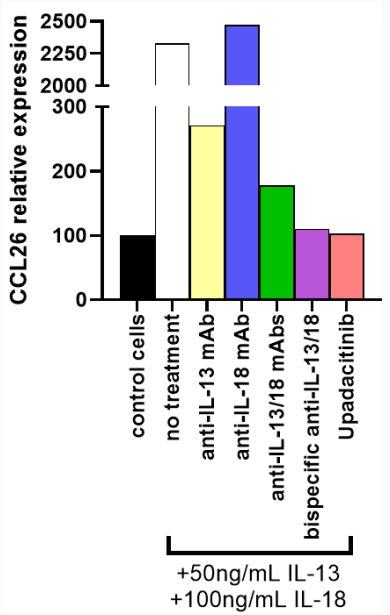
Potential Format Superiority

- Targeting IL-13 and IL-18 via bispecific antibody appears to be superior to combination of monoclonal antibodies

Skin Barrier Function



Th2 Inflammation



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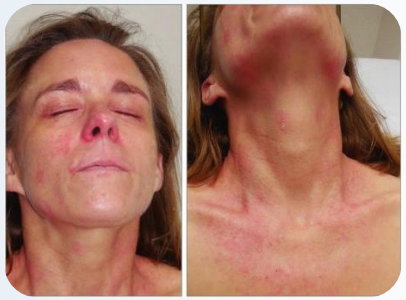
Source: Data on file

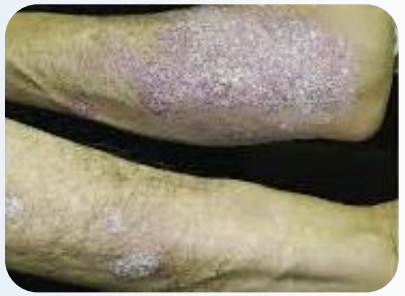
Dual IL-13 x IL-18 Inhibition has the Potential to Drive Deeper and Broader Responses than IL-13 Inhibitor Monotherapy

4 **Clinical Observation:** Th switching implicated in Dupixent loss of response

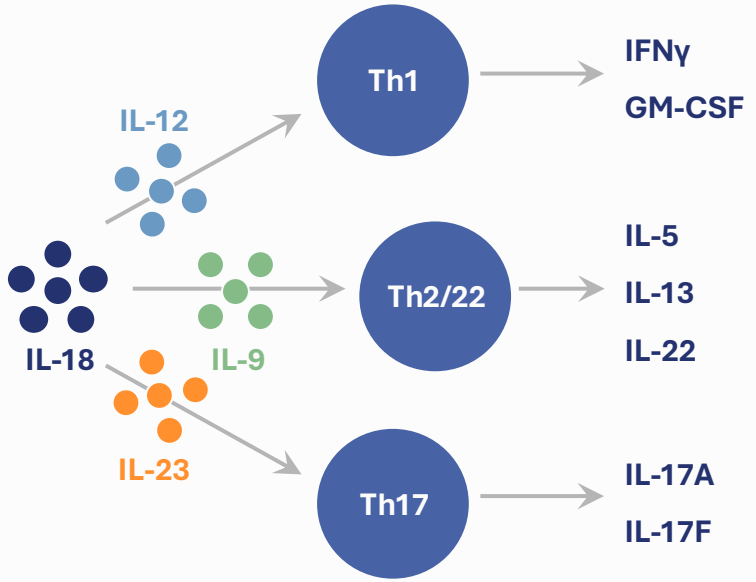
Th22: Head and neck dermatitis[1]



Th1/Th17: Psoriasiform dermatitis[2]



Heterogeneous Th switching likely driven by immune escape via IL-18[3,4,5]



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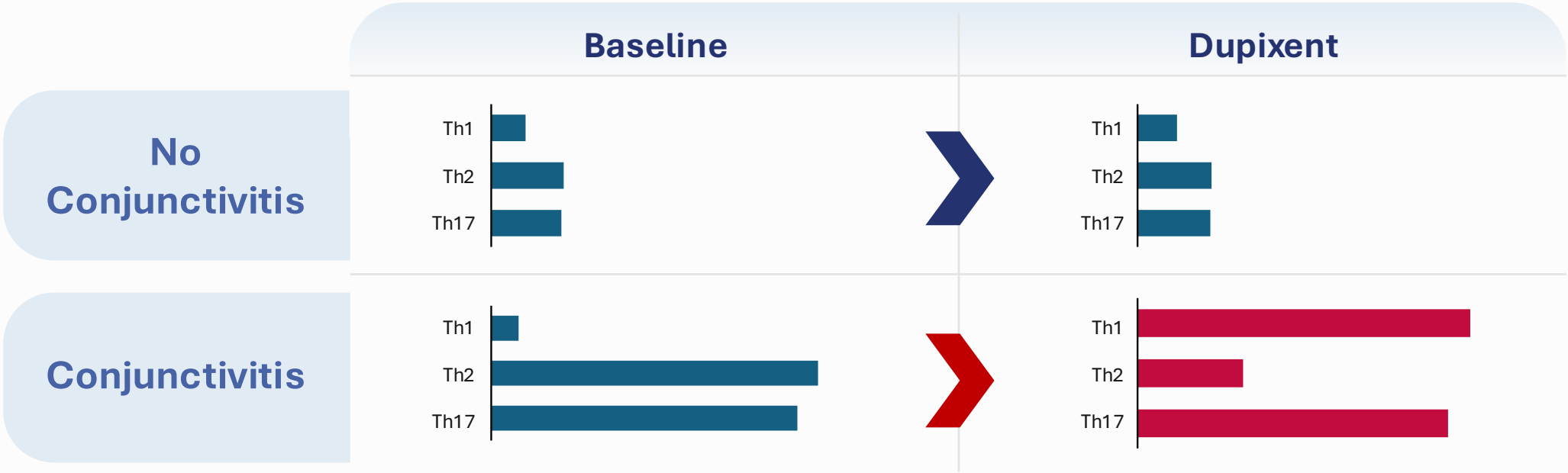
Source: [1]Soria et al., *JAMA Dermatol* (2019). https://doi.org/10.1001/jamadermatol.2019.2613. [2]Varma et al., *JAAD Case Rep* (2020). https://doi.org/10.1016/j.jdcr.2020.01.012. [3]Lee et al., *PLoS One* (2017). https://doi.org/10.1371/journal.pone.0186351. [4]Lusty et al., *Mol Immunol* (2017). https://doi.org/10.1016/j.molimm.2017.06.025. [5]Scharli et al., *J Allergy Clin Immunol* (2024). https://doi.org/10.1016/j.jaci.2024.10.027.

Dual IL-13 x IL-18 Inhibition has the Potential to Ameliorate Conjunctivitis Seen with IL-4Rα/13 Inhibitor Monotherapy

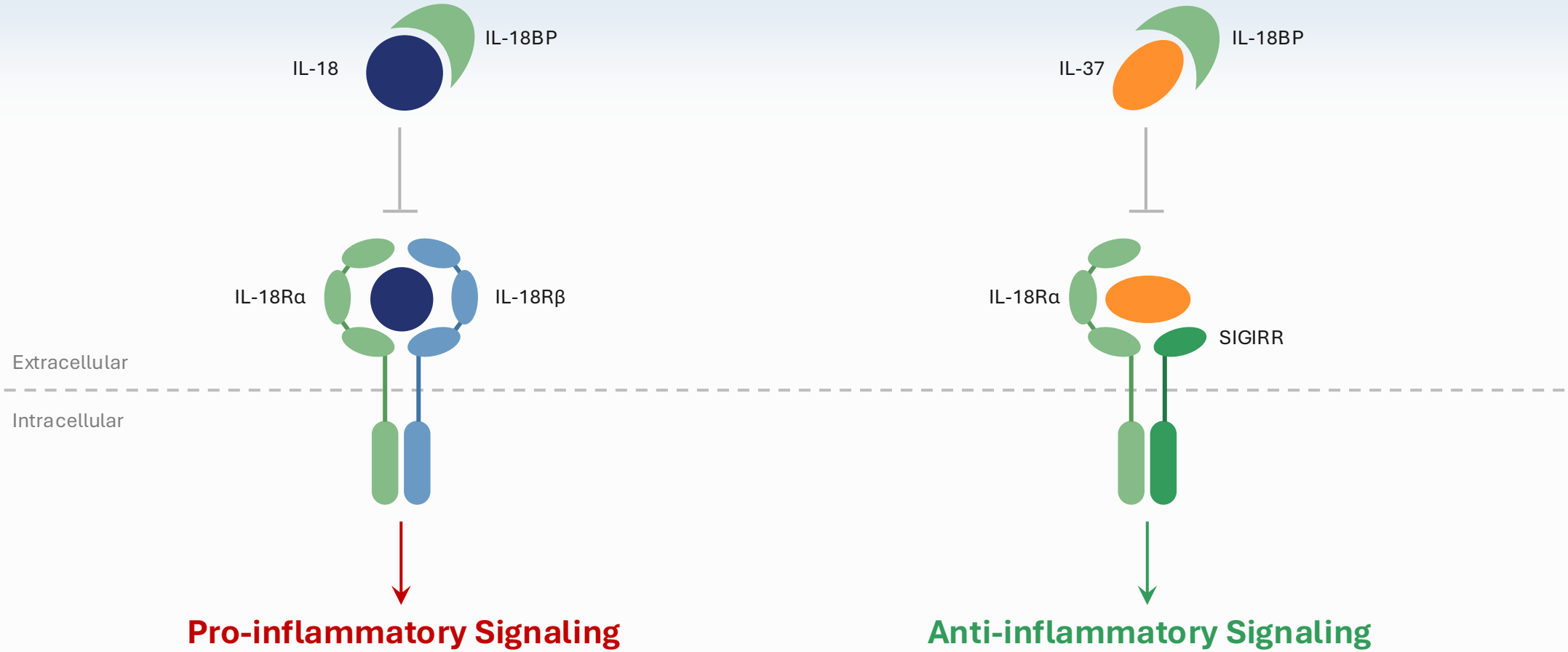
Data from tear immune profiling suggest Th1 switch in patients who develop conjunctivitis



IL-18 inhibition mediates Th1-associated IFNγ production and may reduce conjunctivitis

Source: Thormann et al., *Allergy* (2024). https://doi.org/10.1111/all.16045. North Immunology Analysis. **Note:** Results from clinical trials of other agents are not indicative of results that may be demonstrated in clinical studies of NOR-101.

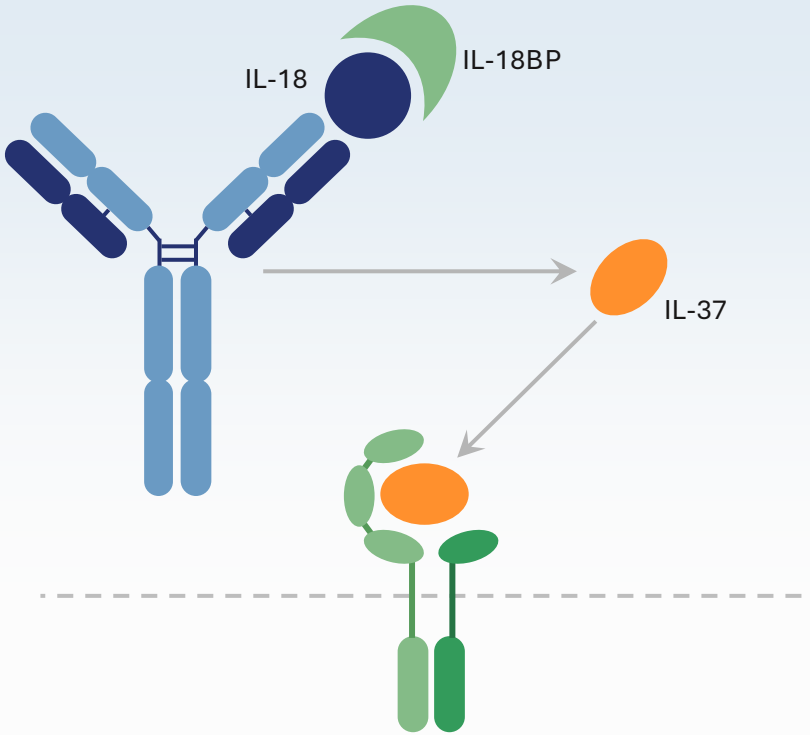
IL-18BP is a Soluble Decoy for Two Opposing Pro- and Anti-inflammatory Cytokines: IL-18 and IL-37



Source: Nold-Petry et al., *Nat Immunol* (2015). https://doi.org/10.1038/ni.3103. Plater-Zyberk et al., *J Clin Invest* (2001). https://doi.org/10.1172/JCI200112097. Dinarello et al., *Front Immunol* (2013). https://doi.org/10.3389/fimmu.2013.00289.

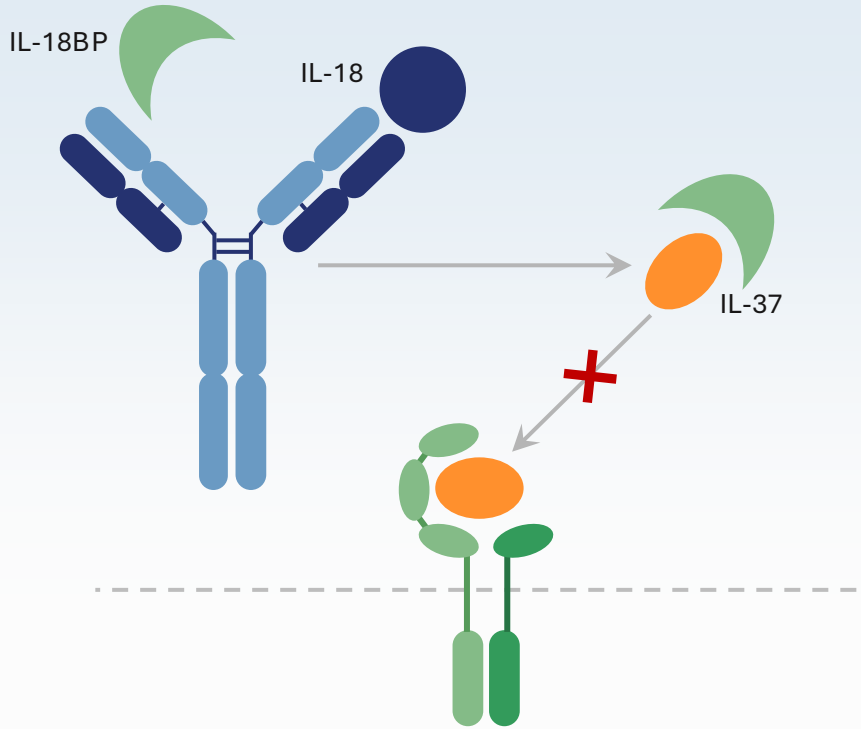
NOR-101 Demonstrates IL-18BP Non-competitive Binding, Sparing IL-37 Anti-inflammatory Signaling; IL-18BP Competitive Binding Decreases IL-37 Signaling

IL-18BP Non-competitive



NOR-101

IL-18BP Competitive



CMK389 (Novartis) | Camoteskimab (Apollo)

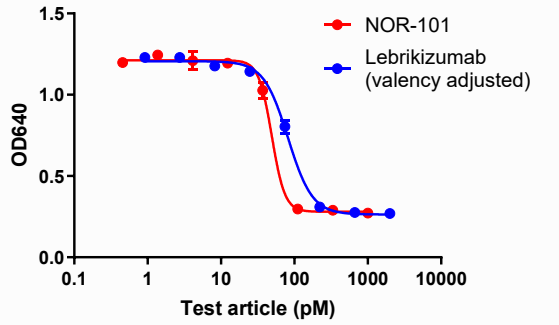
Source: Nold-Petry et al., *Nat Immunol* (2015). https://doi.org/10.1038/ni.3103. Plater-Zyberk et al., *J Clin Invest* (2001). https://doi.org/10.1172/JCI200112097. Dinarello et al., *Front Immunol* (2013). https://doi.org/10.3389/fimmu.2013.00289; internal data

NOR-101 Targets the Same Epitopes as Lebrikizumab (IL-13) and Aletekitug (IL-18); Demonstrates In-line Potency and Strong Developability Profile

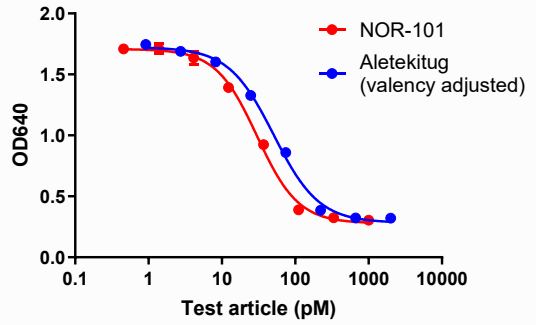
Affinity and Potency

		NOR-101	IL-13 Ref.	IL-18 Ref.
IL-13	Binding affinity, K_D	<6 pM	<5pM[1]	–
	IC_{50}, valency adjusted pSTAT6 RGA	49 pM	65 pM[2]	–
IL-18	Binding affinity, K_D	<3 pM	–	<7 pM[3]
	IC_{50}, valency adjusted NF-kB/AP1 RGA	24 pM	–	32 pM[4]

IL-13 Signaling in pSTAT6 HEK-Blue Reporter cells



IL-18 signaling in NF-κB/AP1 HEK-Blue reporter cells



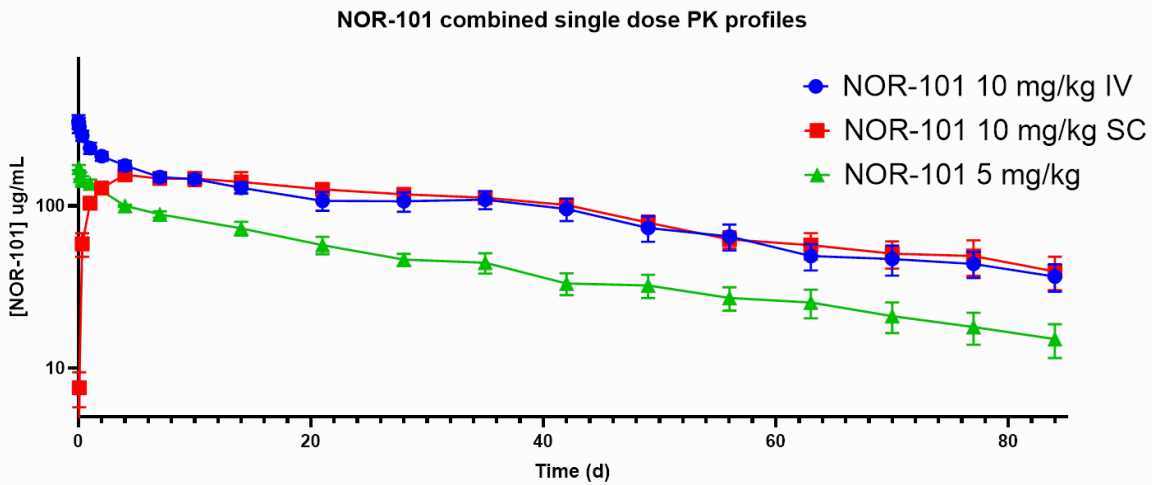
Developability

- High titer expression >6 g/L
- >95% bispecific assembly purity at cell culture
- Demonstrated auto-injector viable viscosity at 200 mg/mL prior to formulation optimization
- SC formulation expected to be ready for Phase 1a

Note: [1]IgG1 monoclonal antibody with lebrikizumab Fvs; [2] IgG1 monoclonal antibody with lebrikizumab Fvs. Valency adjusted to allow for direct comparison to NOR-101; [3] IgG1 monoclonal antibody with aletekitug Fvs; [4] IgG1 monoclonal antibody with aletekitug Fvs. Valency adjusted to allow for direct comparison to NOR-101 **Source:** Internal Data

Potential Best-in-class NHP Half-life Indicates Opportunity for Q3-Q6M Maintenance Dosing; No Adverse Safety Signals in NHP Tox Studies

NHP PK



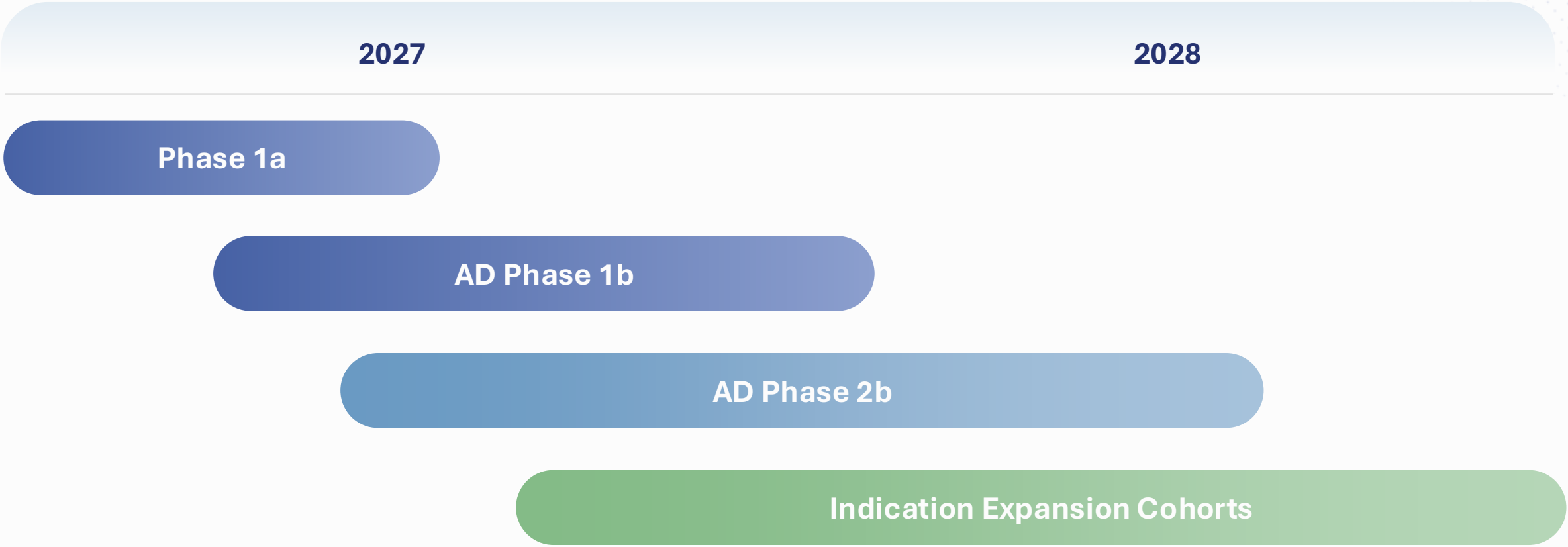
Dose	T1/2 (days)	AUC_{∞} (day • ug/mL)	CL (mL /day/kg)	F (AUC_{last} based)
10 mg/kg SC[2]	**42**	10,310	1.00	~100%
10 mg/kg IV[1]	**37**	9,715	1.07	-
5 mg /kg IV[2]	**35**	4,863	1.04	-

Non-GLP[3] / GLP Tox

- 30 day non-GLP dose range finding study completed in NHPs
  - Administered 5 doses of up to 150 mg/kg IV
  - No safety findings identified
- In-life portion of 30 day GLP-tox study in NHPs completed
  - Administered 5 doses of up to 150 mg/kg IV and SC
  - No clinical observations

Note: [1]n=3; [2] n=2. Excludes one animal in each cohort that developed ADA, 10mg/kg (IV and SC) conducted in separate study vs 5 mg / kg; [3] In-life data only. Necropsy and histopathology not conducted.

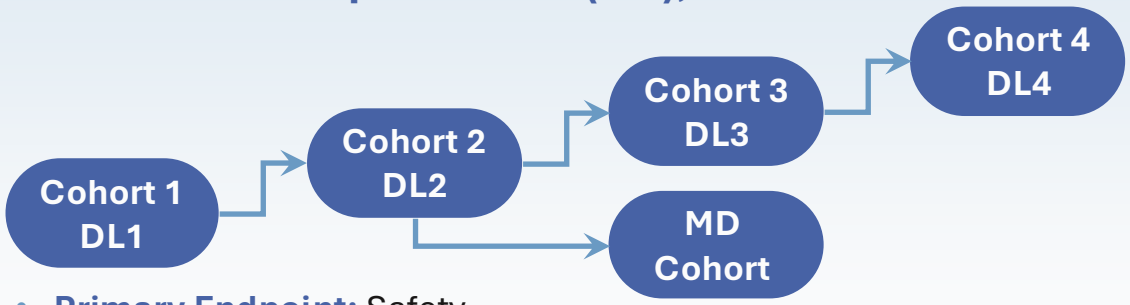
NOR-101 Clinical Plan Designed to Drive Accelerated Path to AD Pivotal Study



Note: Preliminary clinical plan subject to change

Ph1a, Ph1b, and Ph2 Studies in AD Designed to Rapidly Progress to Ph3

Ph 1a HV SAD / MD
N = 8 per cohort (6:2), ROA: SC



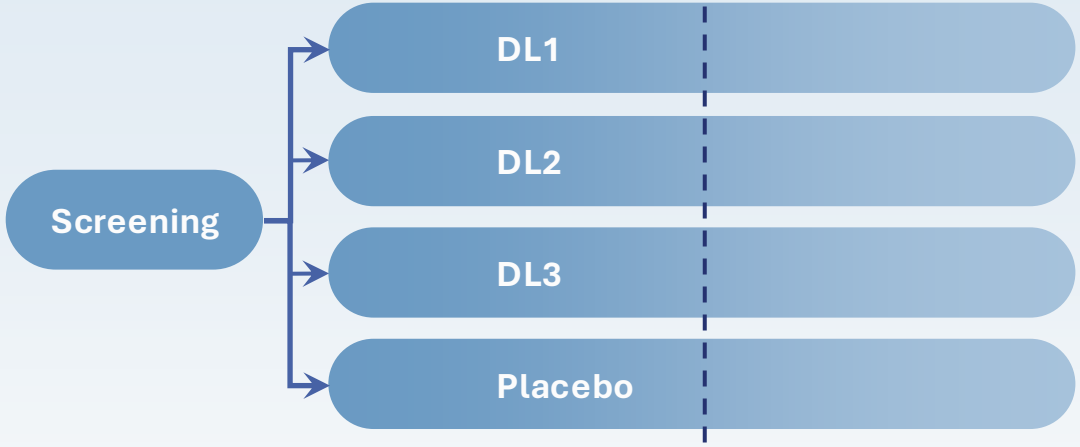
- **Primary Endpoint:** Safety
- **Secondary Endpoints:** PK, ADA
- **Exploratory Biomarkers:** pSTAT6, TARC, non-Th2 biomarkers

Ph 1b Atopic Dermatitis
N=~30-40, ROA: SC

Biologic Naïve and Biologic Inadequate Responder AD Patients

- **Primary Endpoint:** Safety
- **Secondary Endpoints:** PK, ADA, Efficacy
- **Exploratory Biomarkers:** pSTAT6, TARC, non-Th2 biomarkers, skin inflammatory profiling

Ph 2b Atopic Dermatitis
N = 150-200, ROA: SC



Primary Endpoint Week 16

Phase 2 Considerations:

- 3 dose levels vs PBO randomized 1:1:1:1
- Careful attention to data quality: site selection, central review
- Primary Endpoint : % change in EASI at week 16, followed to at least week 32
- Key Secondary Endpoints: % change in itch, EASI75, EASI90, IGA0/1, DLQI
- Exploratory Endpoints: kinetics of itch response, number of flares

NORTH IMMUNOLOGY

Note: Preliminary clinical plan subject to change

Indication Expansion Opportunities

~50M+ potential US patients across eight indications

DERMATOLOGY	
3-5M[1]	Chronic Hand Eczema
1.4M[2]	Nummular Eczema
0.6M[3]	Alopecia Areata
0.2M[4]	Prurigo Nodularis

DERMATOLOGY	
28M[5]	Asthma
20M[6]	Perennial Allergic Rhinitis
10M[7]	Chronic Rhinosinusitis w/ Nasal Polyposis
0.5-1M[8]	Eosinophilic Esophagitis

NORTH IMMUNOLOGY

Source: [1]Chovatiya et al., *SKIN* (2025). https://doi.org/10.25251/x9kr0q89. [2]Loiselle et al., *JID* (2025). https://doi.org/10.1016/j.jid.2025.02.136. [3]Ding et al., *Sci Rep* (2025). https://doi.org/10.1038/s41598-025-07224-x. [4]DelveInsight. [5]CDC, *Most Recent Asthma Data*. https://www.cdc.gov/asthma-data/about/most-recent-asthma-data.html. [6]Long et al., *Management of Allergic and Nonallergic Rhinitis: Summary* (2002). https://www.ncbi.nlm.nih.gov/books/NBK11954/. [7]Asthma and Allergy Foundation of America, *Nasal Polyps Facts and Figures* (2025). https://aafa.org/wp-content/uploads/2025/04/aafa-nasal-polyps-facts-and-figures.pdf. [8]Thel et al., *Clin Gastroenterol Hepatol* (2025). https://doi.org/10.1016/j.cgh.2024.09.031.

Key Anticipated Milestones

	2026	2027	2028
NOR-101 *IL-13xIL-18*	• Q4: Australia HREC and CTN Submission	• Q1: Ph 1a Start • H1: Ph 1b Start • 2027: Ph 2b Start • **Midyear: Ph 1a PK/ Safety Data**	• **Q1: Ph 1b Topline Data** • **2028: Ph 2b Topline Data**
NOR-201 *Undisclosed*		• DC Nomination	• Ph 1a Start

Anticipated $180M PIPE raise provides cash into 2H 2028

IP / License Agreement

Agreement

- IP to be owned by affiliate Central Therapeutics, LLC; NOR-101 composition of matter exclusively licensed to North Immunology
- North to control patent prosecution
- License terms:
  - ***Option fee has been paid***
  - Royalty: mid single digit
  - Milestones: Up to mid-single digit development and approval milestones

IP Status

- Initial IP filed* with coverage anticipated through 2047+

NORTH IMMUNOLOGY

Note: *Patents pending

Pro Forma Capitalization Table

		Shares Outstanding / Issued	Ownership in Pro-Forma Company
Aethlon Medical	**Shares outstanding** (including shares underlying warrants and restricted stock units)	**3,380,423**[(1)]	**4.75%**
North Immunology		**30,719,158**	**43.17%**
Concurrent Financing		**37,067,067**[(2)]	**52.08%**
Total		**71,166,648**	

Estimated post-closing capitalization based on information as of the signing of the proposed transaction and concurrent financing
Aethlon shares outstanding include shares granted in connection with financial advisor fee. (1) Shares inclusive of Parent warrants with a strike price of $3.55 | (2) Financing is inclusive of $34M convertible note

Team Overview

Management Team

Jonathan Barr
CEO

Mohit Gupta
Cofounder and CSO

Sejal Hall
COO

John Kelly
CMO

Li Malmberg
CTO

Ramei Sani-Grosso
SVP, Clin Ops

Yan Zhao
SVP, Finance

Toni Jun
VP, Preclinical

Investors & Board of Directors

Daniel Schneeberger
Cofounder; Portfolio Manager, ADAR1

Mohit Gupta
Cofounder and CSO, North Immunology

Stuart Graham
COO, ADAR1

ADAR1 Capital Management

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IMMUNOLOGY

Thank You

Company Overview

SEPTEMBER 2026