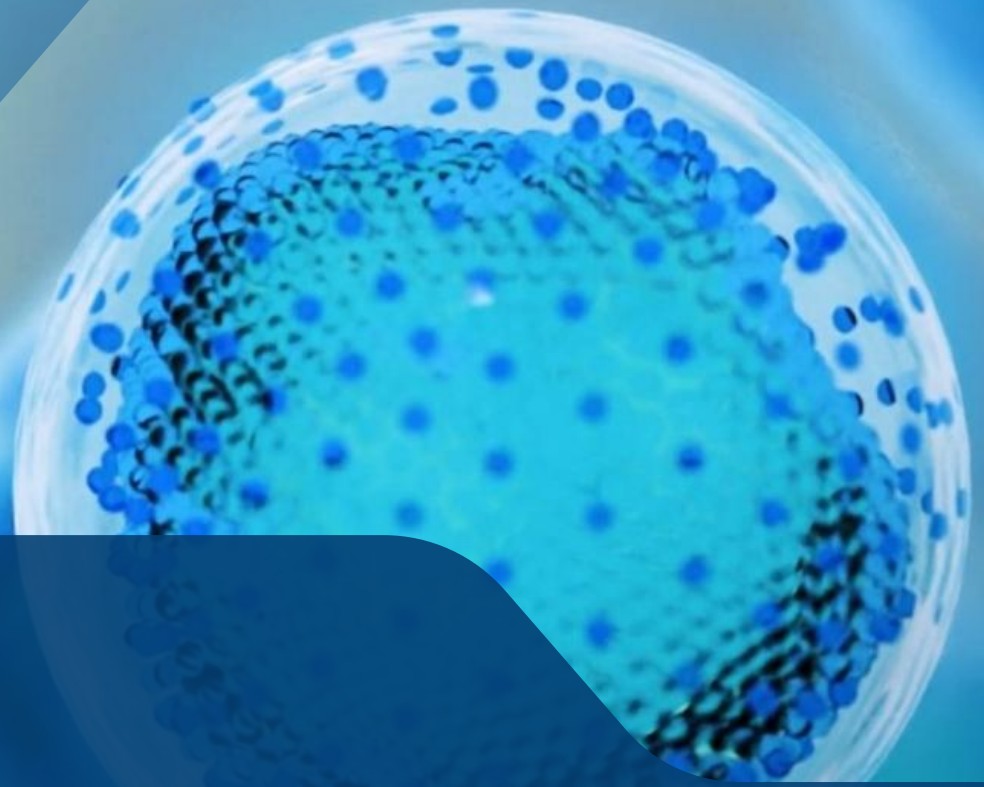
ADVANCING NON-SYSTEMIC MEDICINES FOR INFLAMMATORY SKIN DISEASES

TURN THERAPEUTICS



July 2026

Corporate Presentation

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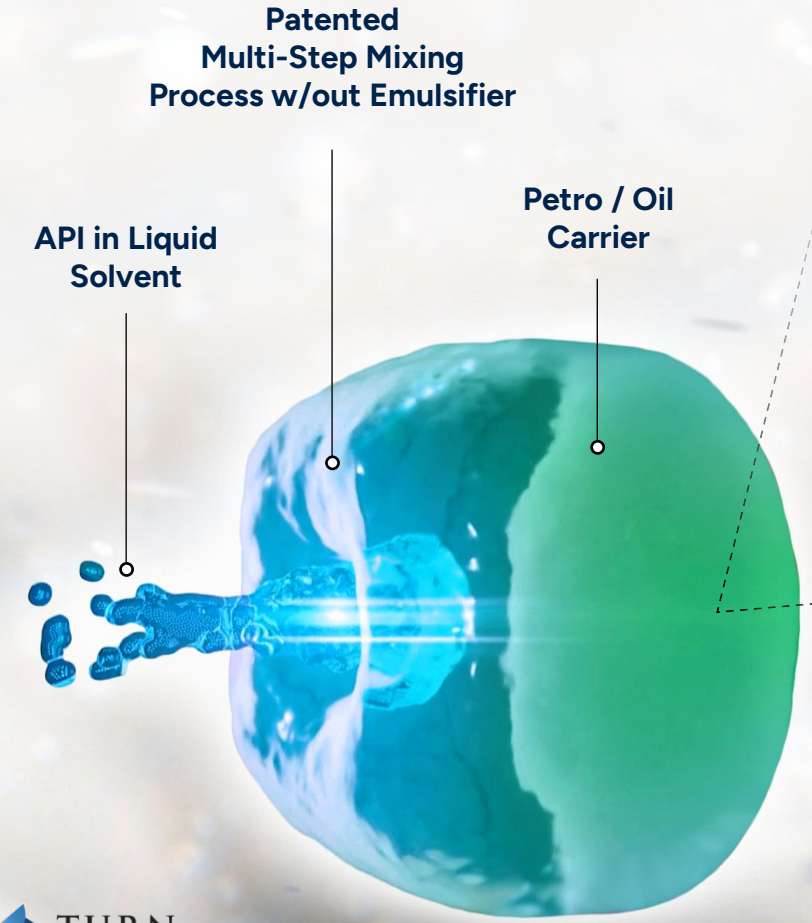
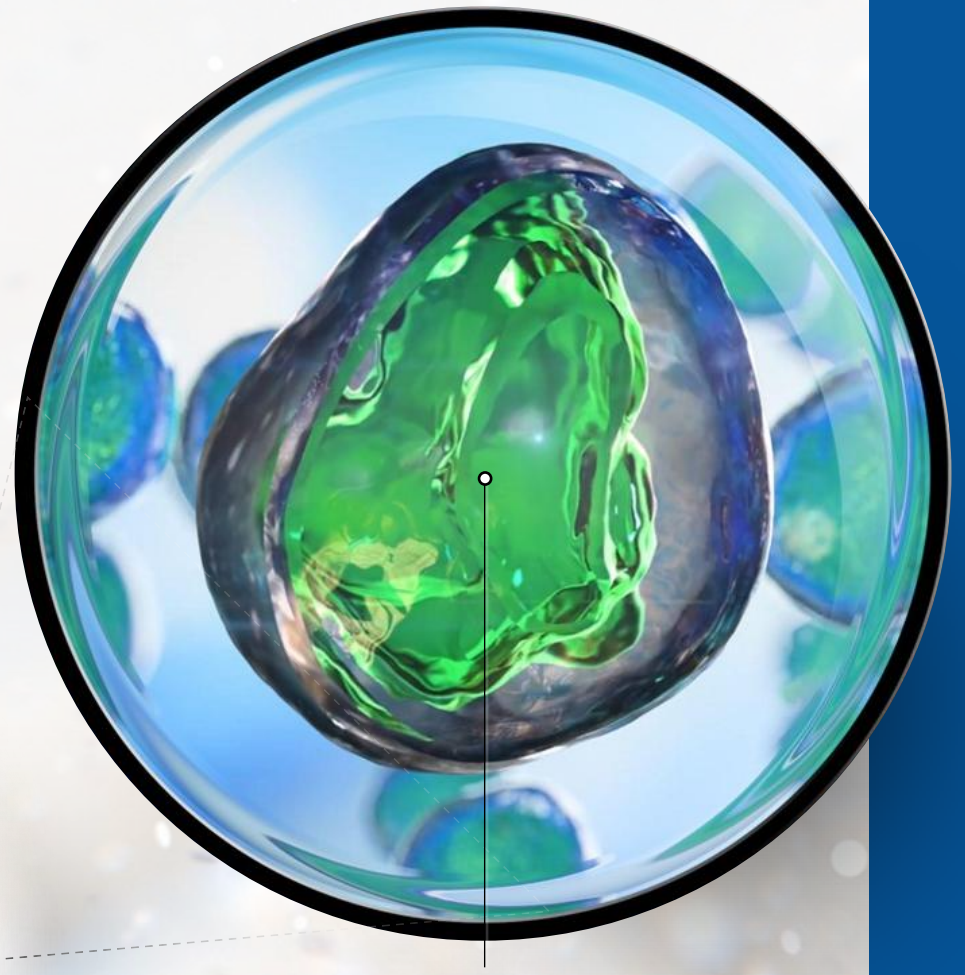
PermaFusion: API-Agnostic Delivery Platform





1.3 Micron

Active ingredient nanodroplets suspended within petrolatum matrix

A transformative delivery platform enabling stable, emulsifier-free dispersion of active ingredients in oil-based carriers for superior penetration by APIs

- Multi-patented, proprietary process
- API-agnostic drug delivery platform designed for continued innovation
- Suspended, non-diluted nanodroplets embedded within oil-based carrier deliver active ingredients through skin, nails, and mucous membranes
- Compatible with any liquid or liquid-soluble API, including live payloads (i.e., viruses/vectors)

TURN THERAPEUTICS

Executive Summary

Turn Therapeutics is a biotechnology company developing GX-03, a first-in-class, precision, non-systemic immunomodulation therapy that targets IL-36 and key downstream cytokines to address high-unmet-need inflammatory diseases, with an initial focus on moderate to severe AD.

IL-36/IL-31 Inhibitor – GX-03

Novel MOA

employs precision immunomodulation to prevent unnecessary immune activation and systemic uptake

200,000+

patients have received GX-03 with zero reported adverse events[1]

Robust IP

including issued composition and method patents, supporting durable commercial exclusivity

Phase 2 RCT trial

underway for moderate to severe AD, topline results expected Q4 2026

FDA clearances

with non-cytotoxic, non-sensitizing and non-irritating claims

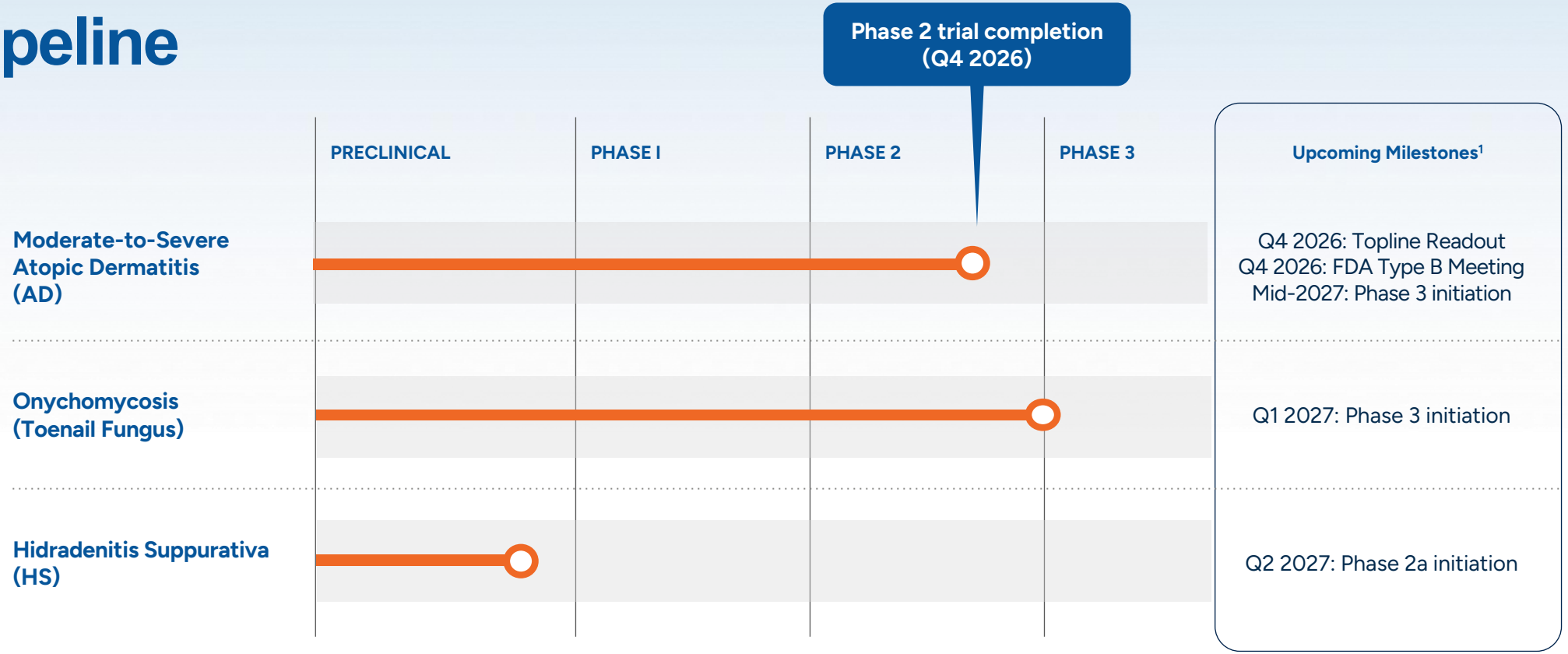
1. In other FDA cleared indications (or as a management tool for chronic wound care)

Turn Tx Pipeline

GX-03

IL-36α, IL-36γ, IL-31 and IL-4 inhibitor

Non-systemic and non-steroid potentially best-in-class topical



Medical Devices

FDA-cleared antimicrobial surgical gauze out-licensed

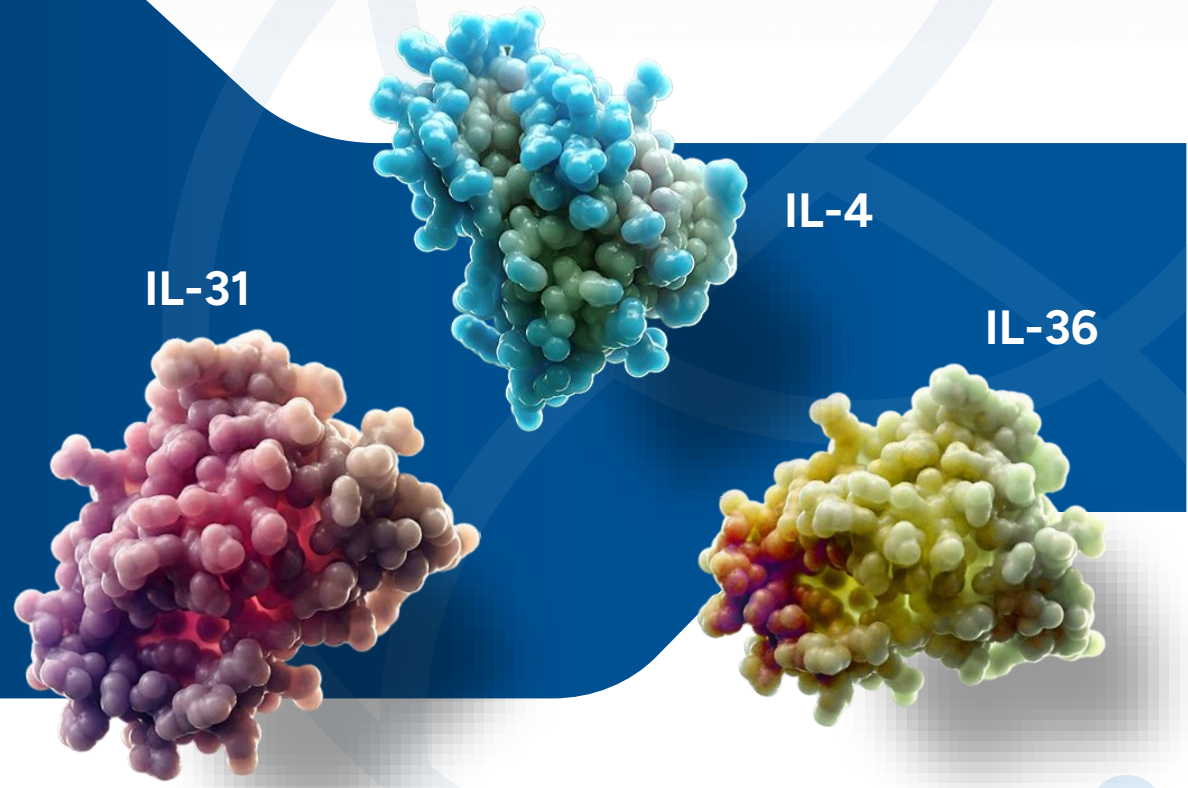
Sterile collagen powder out-licensed for $70M+ milestones

1. Subject to successful completion of each phase and capital availability.

TURN THERAPEUTICS

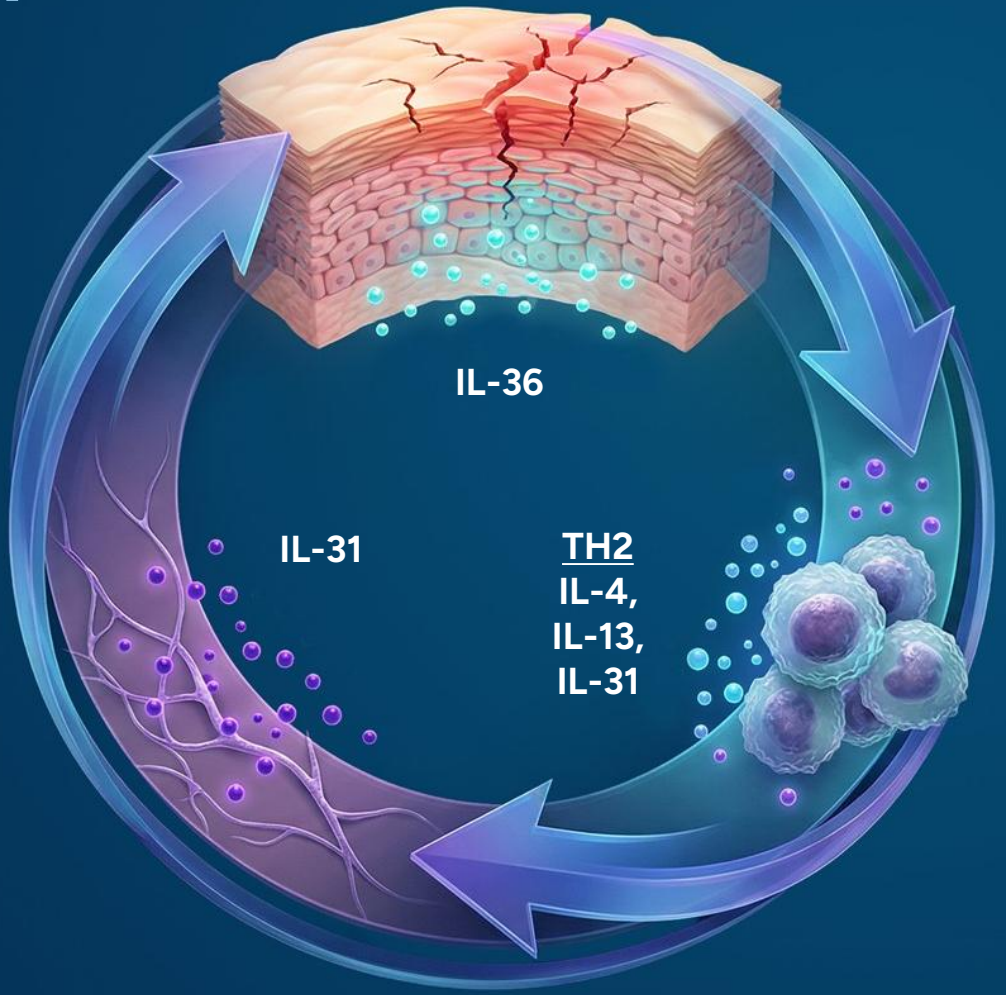
GX-03 for AD

Phase 2 Ongoing



The Chronic Inflammatory ‘Loop’

Skin Barrier Disruption
Skin barrier disruption results in release of IL-36



IL-31 Drives Itch & Scratch

IL-31 release drives itch which leads to further disruption of the skin barrier and restarts loop

Th2 Immune Response

IL-36 initiates TH2 signaling with IL-4, IL-13, and IL-31 release

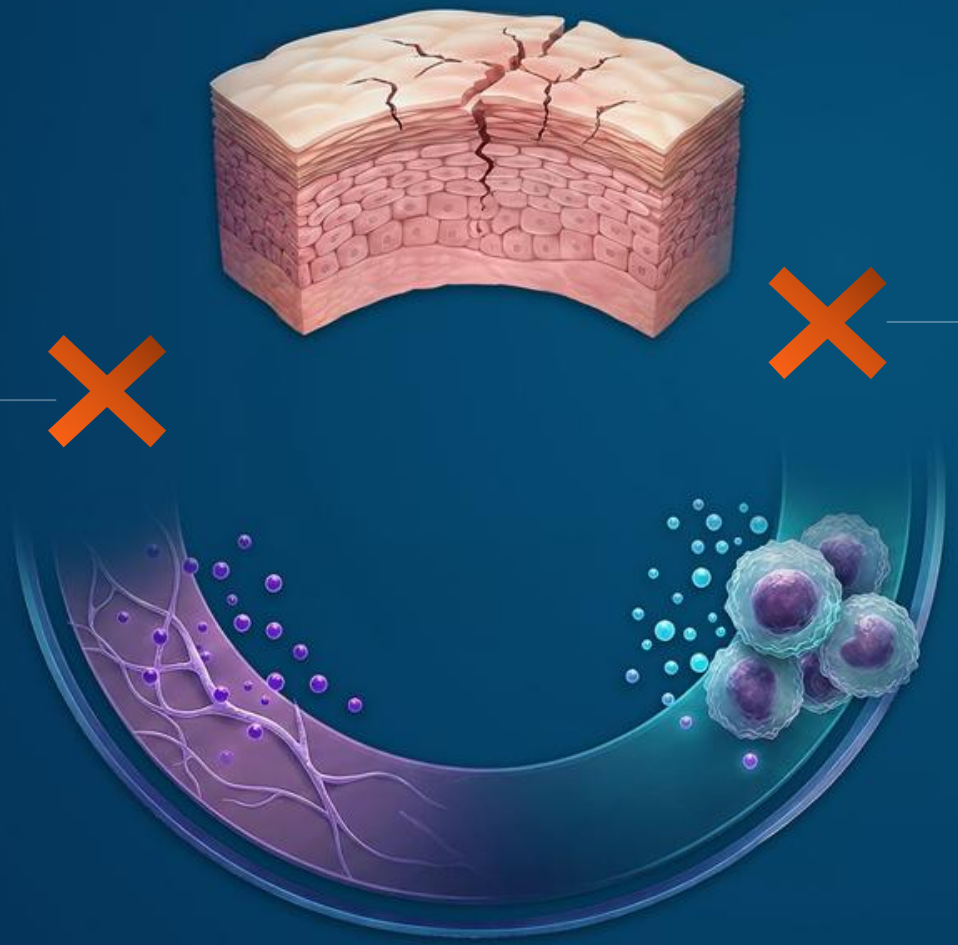
Chronic AD Cycle

Stop the ‘Loop’

IL-36 and IL-31 Inhibition

GX-03 targets key upstream and downstream cytokines to stop the chronic inflammatory ‘loop’

GX-03 suppresses IL-31 signaling, blunting itch/scratch signal and continued barrier disruption



GX-03 suppresses IL-36 release via targeted modulation of the epithelial microenvironment, blunting Th2 response

GX-03 Inhibition

Ongoing Phase 2 for GX-03 in AD

Q4 2026
Topline Results Expected

June 2026
Stage 1 Complete

N = 50
Adaptive design

Ongoing Stage 2 Enrollment

2026 | 2027

Stage 2 Optimized Design

Population:

- N = ~ 120-135 (*Powered to ≥ 99.8%*)
- EASI ≥ 1.1, IGA ≥ 3 and PP-NRS ≥ 7
- Age: 18 – 80
- No concomitant therapy

Design:

- Double-blind
- 1:1 Randomized w/ Prospective 1:1 Stratification
- Vehicle Controlled
- BID Topical
- Week 4 and Week 8 Endpoints
- IDMC Governed Adaptive Design

Selected Endpoints:

- **Week 4 – vIGA-AD of 0/1 with ≥ 2 grade improvement**
- **Week 4 – EASI-75**
- **Week 8 – EASI-90**
- **Week 8 – EASI-100**

Endpoints will be evaluated using a single prespecified Hochberg multiple-testing method across the four endpoints.

How is Atopic Dermatitis Severity Measured?

Lesion Severity vs Inflammatory Burden

Lesion Severity Measured through vIGA-AD

vIGA = 4
EASI≈1.1

- **Validated Investigator Global Assessment (vIGA-AD)**:

A 5 point scale (0-4) rating overall disease severity based on the worst lesion characteristics – erythema, papulation, and lichenification.

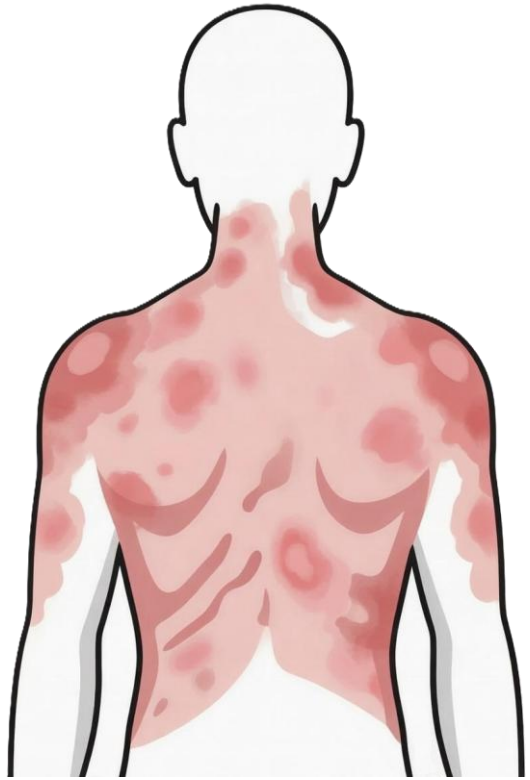
- **Eczema Area Severity Index (EASI)**:

A weighted score (0-72) that accounts for the extent (% body surface area) and intensity of inflammation across 4 body regions.

- **Key Difference**:

vIGA reflects the severity of the single worst area; EASI captures total inflammatory burden across the whole body.

Inflammatory Burden Measured through EASI



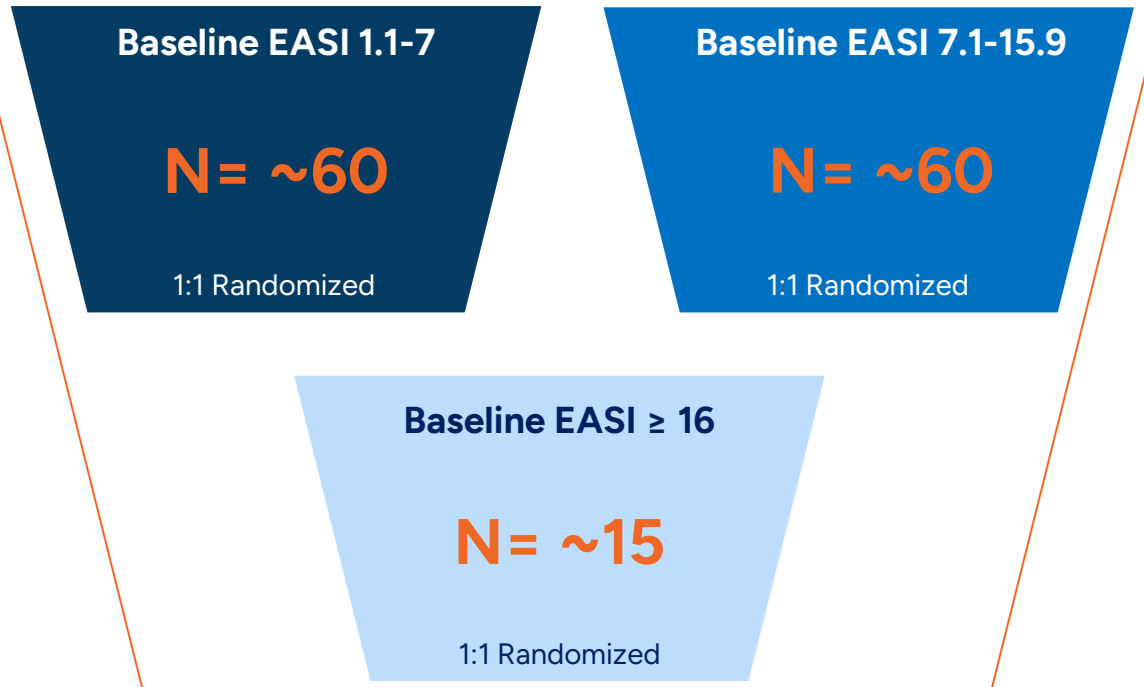
vIGA = 4
EASI ≈ 17

Stage 2 Design – Prospective Stratification by EASI

Three Targeted Enrollment Groups

Stratified by Baseline EASI

Balanced by **Biased Coin Randomization**



Enrollment continued during Stage 1 Analysis and Stage 2 Optimization

Enrollment Criteria & Stratification

1. Baseline PP-NRS

Baseline ≥ 7 – Mandatory Inclusion Criteria

2. Baseline EASI

Stratified by baseline EASI score

- Separate strata(s) prevents imbalanced disease severity among GX-03 and Vehicle
- Stratified patient population represents full disease spectrum and EASI≥16 groups represent potential biological therapy candidates
- Intentional weighting among disease groups to generate robust treatment separation

3. Baseline vIGA-AD

Baseline ≥ 3 – Mandatory Inclusion Criteria

Stage 2 Design – Selected Endpoints

Endpoints and timepoints optimized from observed separation and effect in Stage 1

Selected Endpoints Family

To be evaluated using Hochberg Method

- **Week 4 – vIGA-AD Success**
  (vIGA of 0/1 with ≥ 2 grade improvement)
- **Week 4 – EASI-75**
- **Week 8 – EASI-90**
- **Week 8 – EASI-100**

Clinically meaningful signals emerged at Week 4 with responses deepening through Week 8

Primary Statistical Analysis

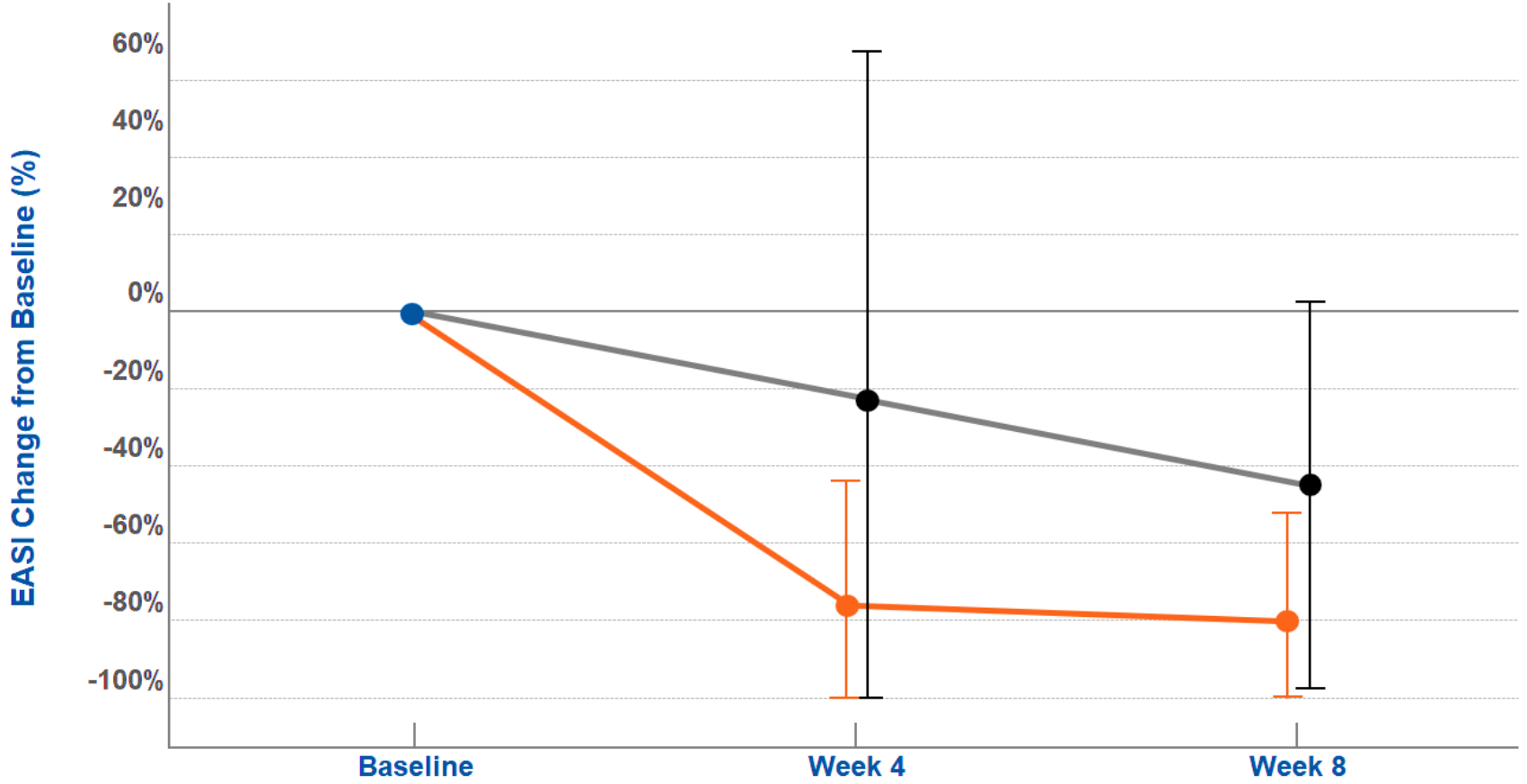
The entire enriched efficacy population will be evaluated using a single prespecified Hochberg multiple-testing method across the four selected endpoints.

Hochberg Method Characteristics:

- One primary population
- One analysis set
- No predetermined alpha (α) allocation
- Full alpha (α) directed to a single multiplicity-controlled analysis

Stage 2 Design – Representative Subgroup from Interim Analysis

Baseline EASI ≥ 1.1 and Baseline PP-NRS ≥ 7



Baseline Characteristics	GX-03 (n=13)	Vehicle (n=12)
Mean EASI (SD)	12.1 (13.3)	7.2 (5.7)
Mean vIGA (SD)	3.15 (0.36)	3.08 (0.49)

Baseline characteristics favor Vehicle

Stage 2 Design – Representative Subgroup from Interim Analysis

Baseline EASI ≥ 1.1 and Baseline PP-NRS ≥ 7

Endpoint	GX-03 (n=13)	Vehicle (n=12)	Treatment Difference	Two-Sided p-value*	Stage 2 Statistical Power at n≈135
Week 4 vIGA-AD Success vIGA of 0/1 with ≥ 2 grade improvement	**61.5%** (8/13)	8.3% (1/12)	**Δ 53.2%**	0.0100	~99.99%
Week 4 EASI-75	**69.2%** (9/13)	25.0% (3/12)	**Δ 44.2%**	0.0407	~99.99%
Week 8 EASI-90	**53.8%** (7/13)	16.7% (2/12)	**Δ 37.1%**	0.0914	~99.83%
Week 8 EASI-100	**46.2%** (6/13)	8.3% (1/12)	**Δ 37.9%**	0.0704	~99.97%

**Fisher's Exact Test*

Baseline Characteristics	GX-03 n=13	Vehicle n=12
Mean EASI (SD)	12.1 (13.3)	7.2 (5.7)
Mean vIGA (SD)	3.15 (0.36)	3.08 (0.49)

Baseline characteristics favor Vehicle

Encouraging treatment separation across all four endpoints selected for Stage 2 design

Powered with high confidence intervals to obtain maximum developmental value

TURN THERAPEUTICS

Stage 1 – Safety Summary

	GX-03 N = 27	Vehicle N = 23
Subjects with AE, n	1*	1**
Severe (Grade ≥ 2)	0	0
Serious AEs	0	0
AEs leading to study drug discontinuation	0	0

**Subject reported a mild warming sensation that was described as pleasant.*

***Subject reported a common cold which was determined by PI to not be treatment related.*

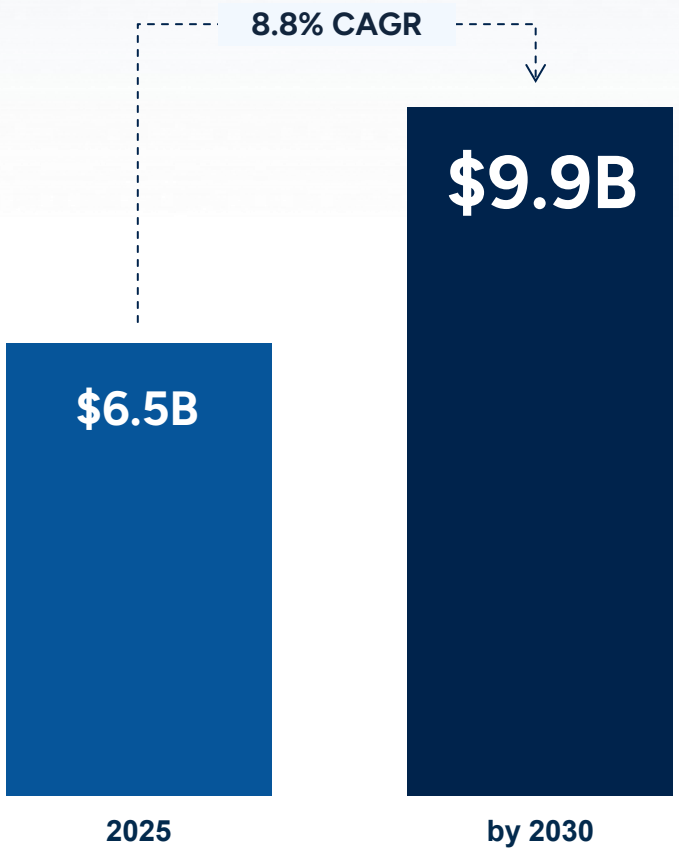
AD TAM & Prevalence

16.5M
US patients suffer from AD

6.6M
patients in moderate-severe category[1]

AD is the largest and fastest growing I&I market in the U.S.[2]

US Market Size[3]



High Unmet Needs

- Safe therapies
- Non-systemic therapies
- Rapid Action
- Needle-free options

GX-03 Path to Market[4]

2026
Phase 2 Readout

2027
Phase 3 Initiation

2029
Potential Commercial Launch

1. The National Eczema Association (NEA)
2. Largest by patient population, fastest based on projected market size (EvaluatePharma)
3. Grand View Research - AD Outlook
4. Subject to successful completion of each phase and capital availability.

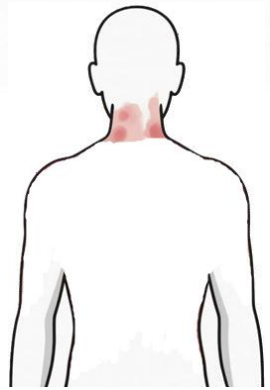
GX-03: For Patients Across The Full Moderate-Severe AD Spectrum

Lower EASI (1.1 – 7)

Patients with clinically moderate-severe lesions per vIGA are not all eligible for the same treatments based upon current guidelines

Higher EASI (>20)

Moderate, Limited
IGA 3/4 - EASI 1.1 - 3



Typical Therapy Used

- *Corticosteroids*
- *Calcineurin Inhibitors*
- *Ruxolitinib (Opzelura®)*
- *Tapinarof (Vtama®)*

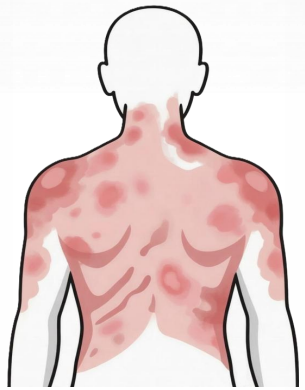
Moderate, Scattered
IGA 3/4 - EASI 3.1 – 7

Typical Therapy Used

- *Corticosteroids*
- *Calcineurin Inhibitors*
- *Ruxolitinib (Opzelura®)*
- *Tapinarof (Vtama®)*

Currently Treated with Topicals (PDE4, AhR, JAK)

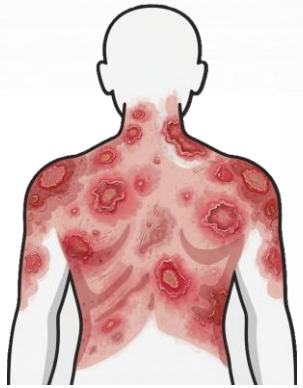
Moderate, Extensive
IGA 3 - EASI 14-16



Typical Therapy Used

- *High potency steroids*
- *Dupilumab (Dupixent®)*
- *Upadacitinib (Rinvoq®)*

Severe, Extensive
IGA 4 - EASI > 20



Typical Therapy Used

- *Dupilumab (Dupixent®)*
- *Upadacitinib (Rinvoq®)*
- *Tralokinumab (Adbry®)*
- *Abrocitinib (Cibinqo®)*

Currently Treated with Biologics

Patients with EASI<16 and IGA 3/4 are oftentimes not eligible for biologics despite clinically significant, localized disease.

GX-03 for Onychomycosis

Phase 3 Ready

GX-03 – Human Data in Onychomycosis

"Novel Approach to Polymicrobial Nail Infection"

R. Dan Davis, DPM
Former President of American Podiatric Medical Association

Study Design (n≈100)

- GX-03 applied once daily or BID application
- No debridement or occlusive dressings required

Study Results

- Visible evidence of Beau's line development, **clear nail plate**
- **70% complete cure**[1] (approx.) with once-daily application
- **85% complete cure**[1] (approx.) with BID application
- **No adverse events** reported during the study

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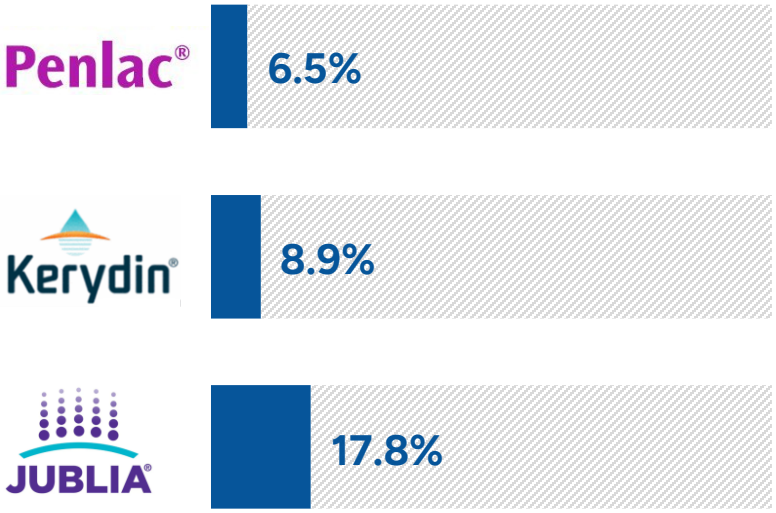
1. Complete cure is defined as 0% involvement of the target toenail (no clinical evidence of onychomycosis of the target toenail).

Current Topicals for Onychomycosis

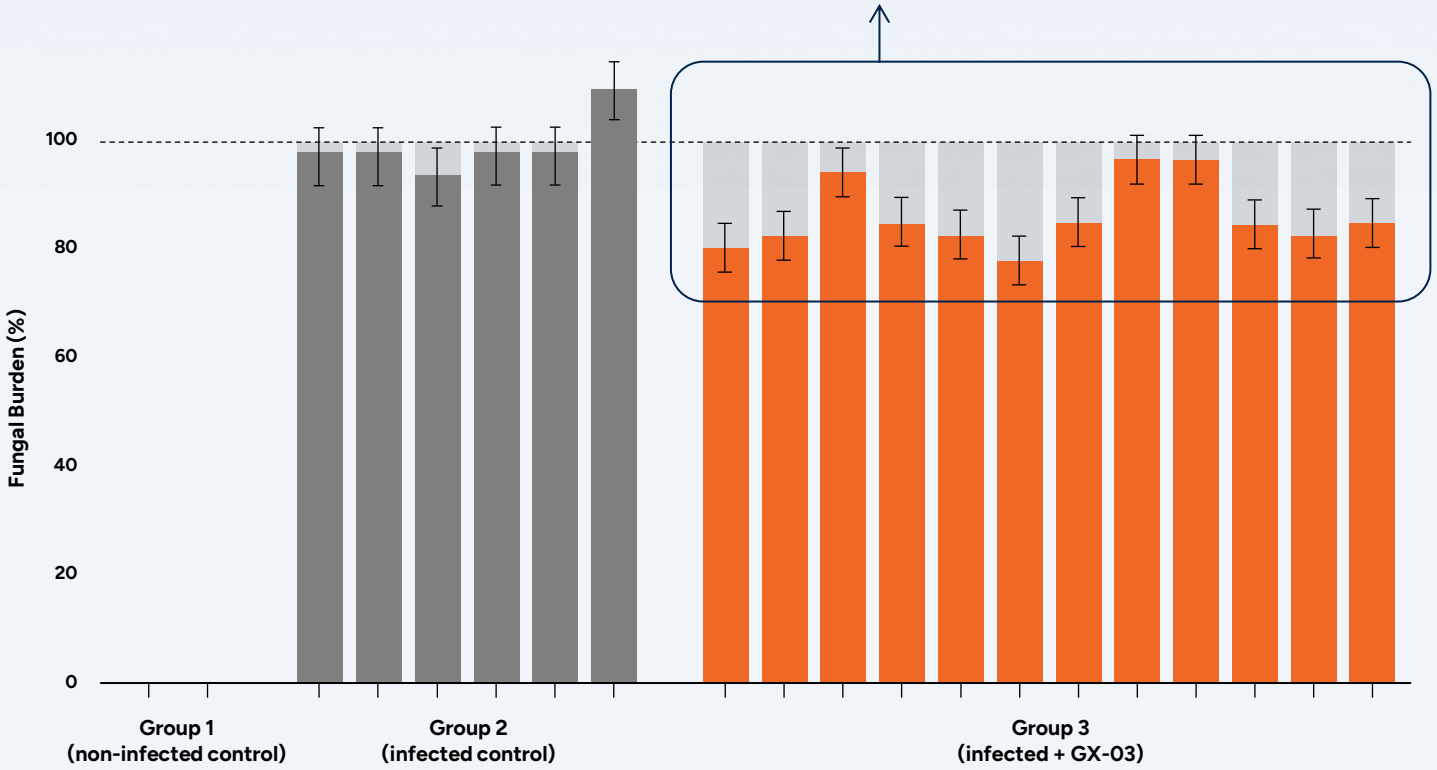
Currently approved topical onychomycosis products have **failed to penetrate the nail** and eliminate fungal pathogens leading to lower complete cure rates[2,3]



1. https://pubmed.ncbi.nlm.nih.gov/21555762/
2. Complete cure is defined as 0% involvement of the target toenail (no clinical evidence of onychomycosis of the target toenail).
3. Not studied in head-to-head trials with GX-03

In the same in-vivo model, GX-03 successfully penetrated the nail, REDUCING FUNGAL BURDEN BY 12% - 18% with just two weeks of application.[1]

Lipid-based delivery enables passive diffusion of API through lipid bilayers.



GX-03 successfully penetrated nails and eliminated fungal pathogens in the standard model

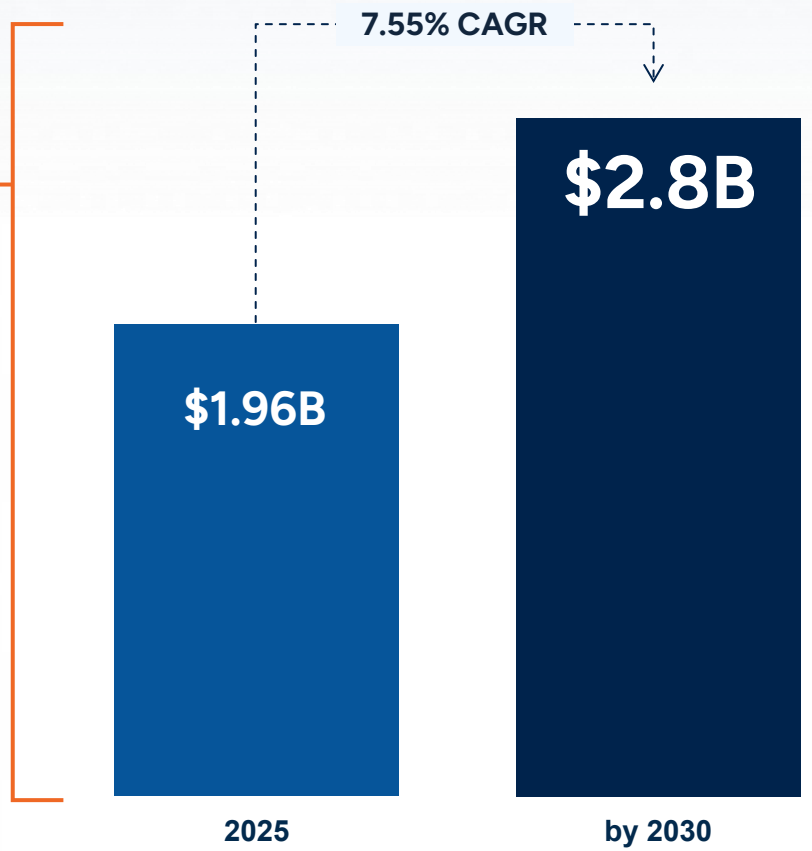
Onychomycosis TAM & Prevalence

Only 15%
of the affected population seeks treatment due to lack of awareness, limited efficacy and hepatotoxicity of available therapeutics

47.6M
patients suffer from onychomycosis in U.S.

1 in 7
people globally suffers from onychomycosis[1]

US Market Size[2]



High Unmet Needs

- Effective therapies as current topical treatments are only 6-18% effective
- Patent cliff for current onychomycosis treatments in 2026

GX-03 Path to Market[3]

2026
Phase 3 Ready

2027
Phase 3 Initiation

2029
Potential Commercial Launch

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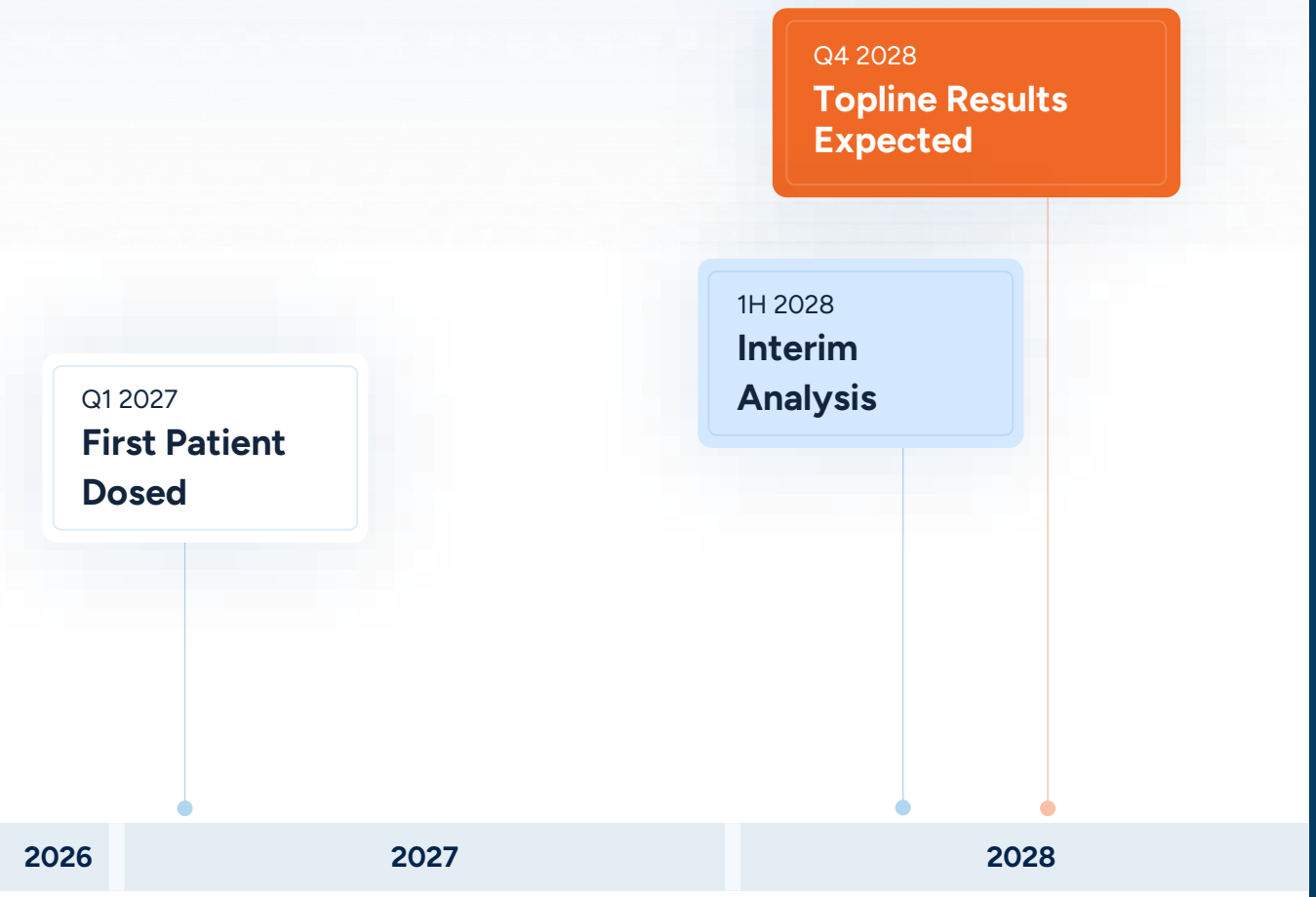
1. www.cdc.gov
2. Precedence Research - Onycho Treatment market.com
3. Subject to successful completion of each phase and capital availability.

Planned Phase 3 Pivotal Trial



Population:

- N ≈ 400
- Age: 18 – 80
- No concomitant therapy

Design:

- Double-blind
- 1:1 Randomized
- Vehicle Controlled
- BID Topical Application
- Week 52 Endpoints
- Week 26 Interim Analysis

Endpoints:

- **Primary: Complete Cure at Week 52**
- Key Secondary Endpoints:
  - Mycological Cure at Week 52
  - ≤ 5% Clinical Involvement at Week 52
  - ≤ 5% Mycological Involvement at Week 52

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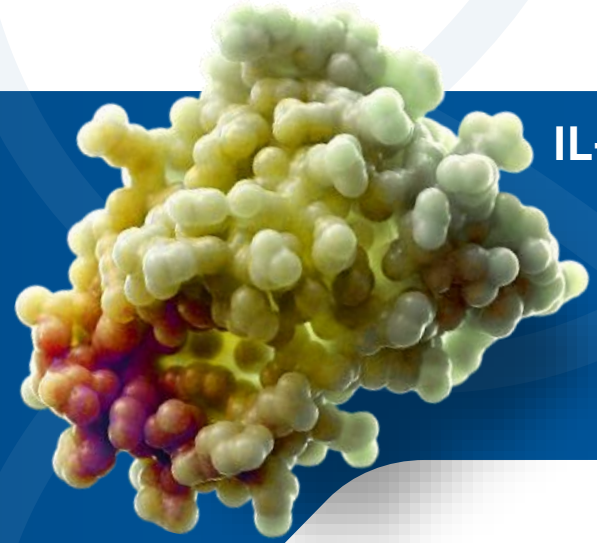
GX-03 for
Hidradenitis Suppurativa (HS)

Preclinical Stage



HS – Major I&I Challenge with Significant Unmet Need

Inflammatory Burden
HS is an IL-36 driven chronic, inflammatory, recurrent skin disease that causes follicular occlusion and rupture leading to painful inflammatory nodules and abscesses

GX-03's IL-36 Pathway
GX-03 inhibits IL-36 signaling at site of disease, which may help downregulate key inflammatory mediators (IL-1β, IL-17A and TNFα)

Skin Dysbiosis
Inflamed, occluded follicles trap anaerobic bacteria, resulting in tunnels, draining, and intensifying disease

GX-03 Manages Dysbiosis
GX-03 manages skin dysbiosis by selectively eliminating trapped anaerobes without cytotoxicity or sensitization

Systemic Options
Current HS treatments include steroids, biologics, and antibiotics, increasing the risk of treatment-related side effects

Non-Systemic Topical
GX-03's non-systemic topical application potentially provides safe, needle-free treatment of both IL-36 driven inflammation and bioburden

HS Progression

Early Nodule

Inflammatory Nodule

Abscess

Draining Tunnels

TURN THERAPEUTICS

GX-03 – Planned Phase 2a Trial in HS

Q1 2028
Topline Readout Expected

Q2 2027
First Patient Dosed

2026 | 2027 | 2028

Trial Design

Population

- 30 GX-03 : 30 Vehicle (1:1 Randomization)
- Hurley Stage I–III; active inflammatory nodules/abscesses (± tunnels)
- Topical GX-03 applied BID or TID to affected areas for 12 weeks

Study Evaluations

- Safety and tolerability
- Efficacy on four endpoints
- Adaptive expansion available based on Phase 2a observed response
- IDMC Governed Adaptive Design

Four-Endpoint Efficacy Family (Week 12)

- **Week 12 – HiSCR75** (≥75% reduction in abscesses)
- **Week 12 – HiSCR50** (≥50% reduction in abscesses)
- **Week 12 – IHS4-55 Response** (≥55% reduction in IHS4 total score)
- **Week 12 – Skin Pain Response (NRS)** (≥30% reduction in average skin pain NRS)

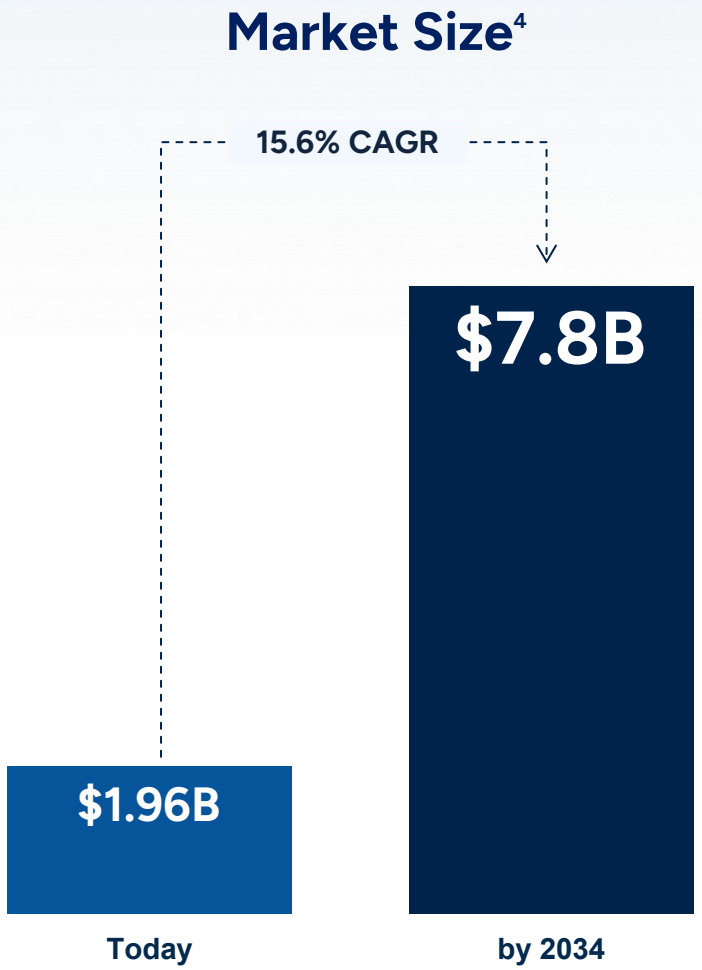
All four endpoints will be evaluated using a single prespecified Hochberg multiple-testing procedure.

HS – TAM & Prevalence

~2.9M
adults suffer from HS[1]

+300K
new patients diagnosed with HS every quarter[2]

...US HS market has a +$10B potential commercial opportunity for multiple drugs[3]



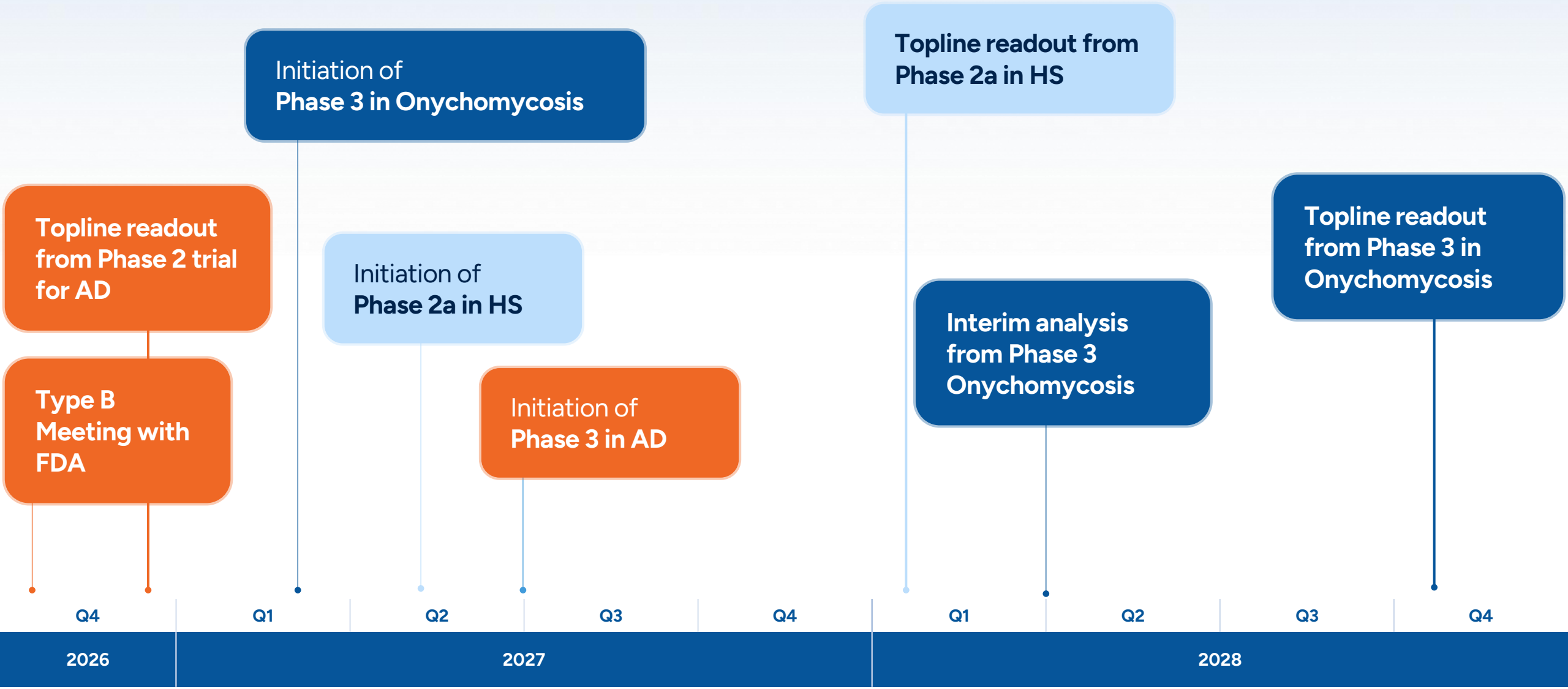
High Unmet Need

- Safe, non-systemic therapies
- Lower cost treatment options
- Limited approved treatment options for children
- Tachyphylaxis and treatment durability

1. Patients ≥18 years with a HS diagnosis in US
2. Net new diagnosed HS patients
3. Research analyst reports from TD Cowen, Leerink and Oppenheimer
4. HS market forecast to reach $7.8bn across 7MM by 2034

TURN THERAPEUTICS

GX-03 – Value-Driving Milestones[1]



1. Subject to successful completion of each phase and capital availability.

Financial & IP Highlights

~$250K/mo.
Current G&A burn as a public co.

~$29M
Total money raised since inception (2015)

~$21M
Cash burned since inception[1]

$11.2M Cash Balance
As of March 31, 2026

Cash Runway into Q3 2027
Additional capital available under existing facilities

~29.8M Common Shares Outstanding
As of May 8, 2026

Extensive Patent Families
Various patent families for composition and/or methods around API

17 Issued Patents
Various additional applications pending

Coverage Through 2040
Various applications pending to extend coverage

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1. $23.4M accumulated deficit as of March 31, 2026 includes stock-based compensation and non cash expenses.

Management & Board

Board Members

Arthur Golden, JD
Senior Counsel, Davis Polk & Wardwell

Andrew Gengos
Former CFO Terns Pharmaceuticals

Kent Kester, MD
Executive Director, Vaccine Research and Development at CEPI

Martin Dewhurst
McKinsey Veteran
Senior Advisor at PJT Partners

Key Management

Bradley Burnam
CEO,
Chairman & Founder

Dr. Neil Ghodadra, MD
Chief Medical Officer

Dr. Stephen Hahn, MD
Clinical and Regulatory Lead

Sasha Damouni Ellis
Corporate Communications & IR

Zuraiz Chaudhary
Chief Accounting Officer

Advisors & KOLs

Dr. Robert Redfield
Former Director, CDC

Dr. Redfield is a nationally recognized virologist and public health leader who served as Director of the CDC. As Senior Advisor of Health Policy and Regulatory Affairs at Turn.

Dr. R. Daniel Davis, DPM
Former President, CPMA & APMA Board of Trustee

Dr. Davis is board Certified Foot and Ankle Surgeon and has been in practice for over 30 years. Dr. Dan Davis is an advisor and KOL for Turn's onychomycosis program.

Stephen Bresnick, MD
Board-Certified Plastic Surgeon

Dr. Stephen Bresnick is a board-certified surgeon in skin health and immunology-related fields, has two doctorate degrees, and is an esteemed research publisher. Dr. Bresnick is an advisor and KOL for Turn's Atopic Dermatitis program.

ADVANCING NON-SYSTEMIC MEDICINES FOR INFLAMMATORY SKIN DISEASES

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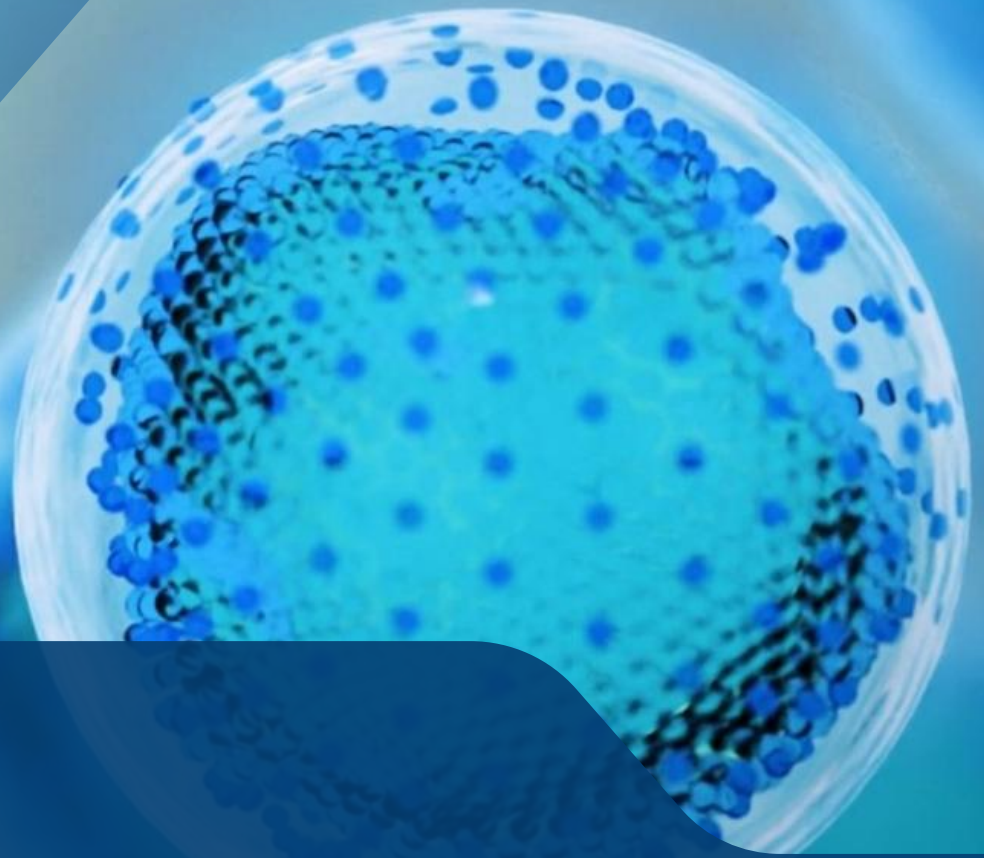

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Thank You!

Appendix

In-vivo and Safety Study

57% Reduction in Disease Severity in an IL-36 Environment[1]

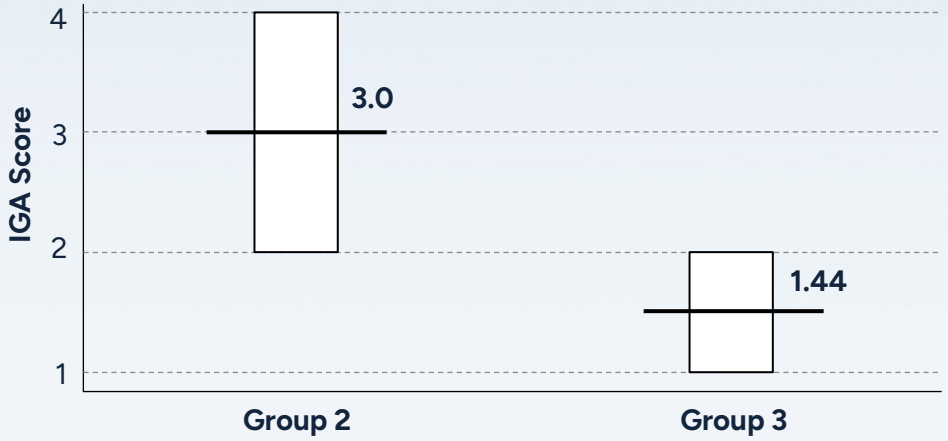
N = 30

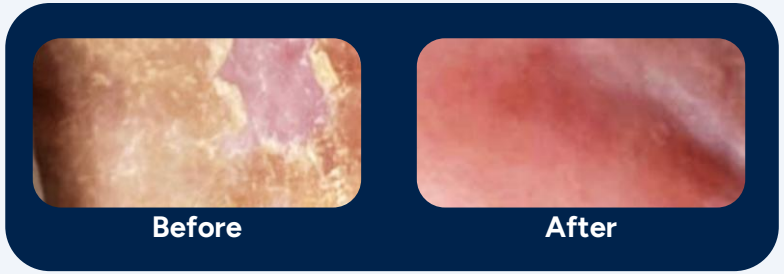
Inflammation induced using a standardized epicutaneous *Staphylococcus aureus* exposure protocol

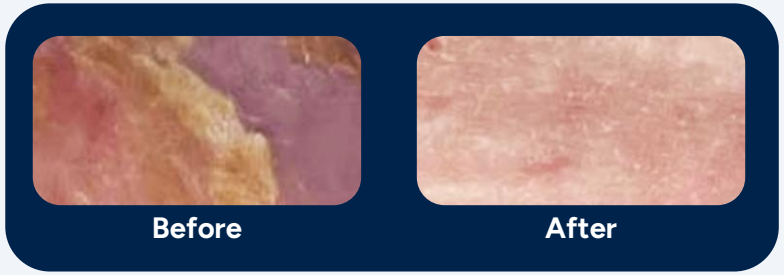
Group 1 N = 10	Group 2 N = 10	Group 3 N = 10
Pre-treatment Control	**No Treatment Control**	**GX-03 Treatment**
3.33 ~ Mean IGA after 7 days of *S.aureus* inducement	3.33 ~ Mean IGA after 7 days of *S.aureus* inducement	3.33 ~ Mean IGA after 7 days of *S.aureus* inducement
	3.00 ~ Mean IGA after 7 days of no treatment	1.44 ~ Mean IGA after 7 days of GX-03 treatment
IL-36 expression observed following microbial challenge	**No significant reduction in IGA**	**57% reduction in IGA over 7 days of treatment**

Group 2 and Group 3 at 14 days

(p-value ~ 0.0003)

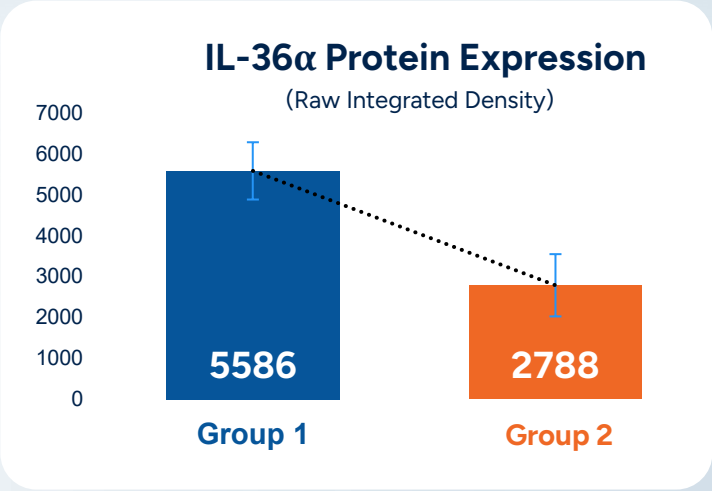


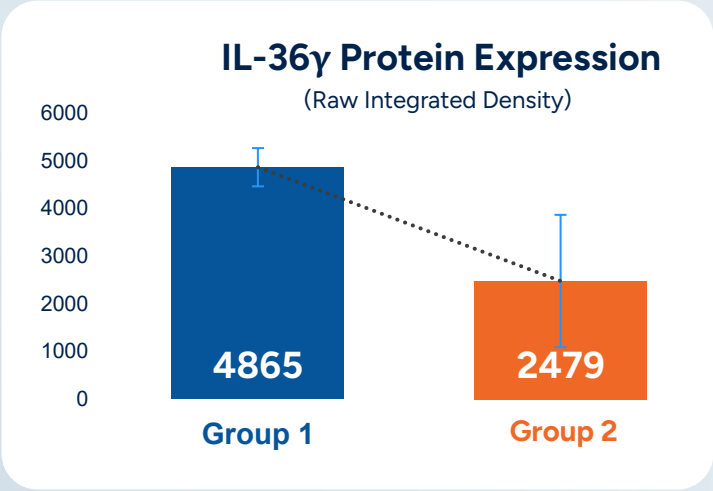


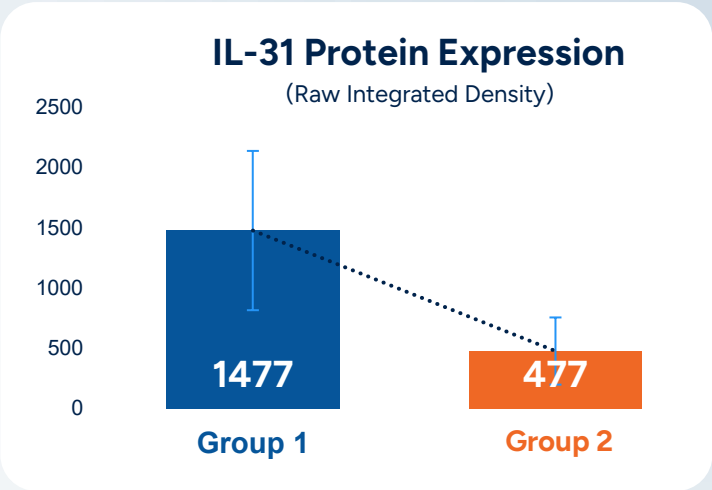


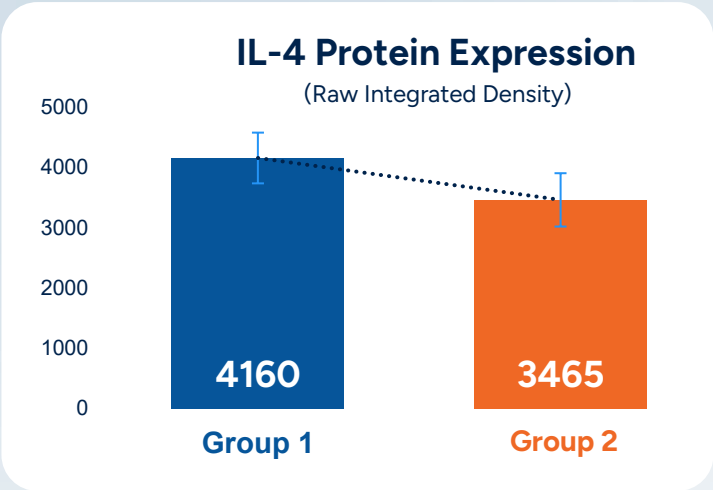
1. Utilizing a validated in-vivo AD model, www.cell.com

Demonstrated Inhibition of Key Upstream and Downstream Cytokines









Objective: To ascertain the immunological inhibitory effects of GX-03 for upstream and downstream cytokines

N = 36
Group 1: no treatment (N = 18)
Group 2: GX-03 treatment (N = 18)

Inflammation induced using a standardized epicutaneous *Staphylococcus aureus* exposure protocol

Cytokine Inhibition Demonstrated:

Cytokine	% Inhibition	p-value
IL-36α	50.08%	0.0000000000
IL-36γ	49.05%	0.0000000435
IL-31	67.70%	0.0000011200
IL-4	16.71%	0.00002870

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Phase 1-Level Safety Data:
53 Patients RIPT Study

Trial Design

- First application: 48 hours of continuous exposure
- 2nd through 9th application: 24 hours of continuous exposure three times a week for three consecutive weeks

Endpoints:

Adverse reaction of erythema and edema scoring:

0 = no reaction
1 = erythema throughout at least ¾ of site
2 = erythema and induration throughout at least ¾ of site
3 = erythema, induration & vesicles
4 = erythema, induration & bullae

Trial Results:

No adverse reactions of any kind
were reported during the study

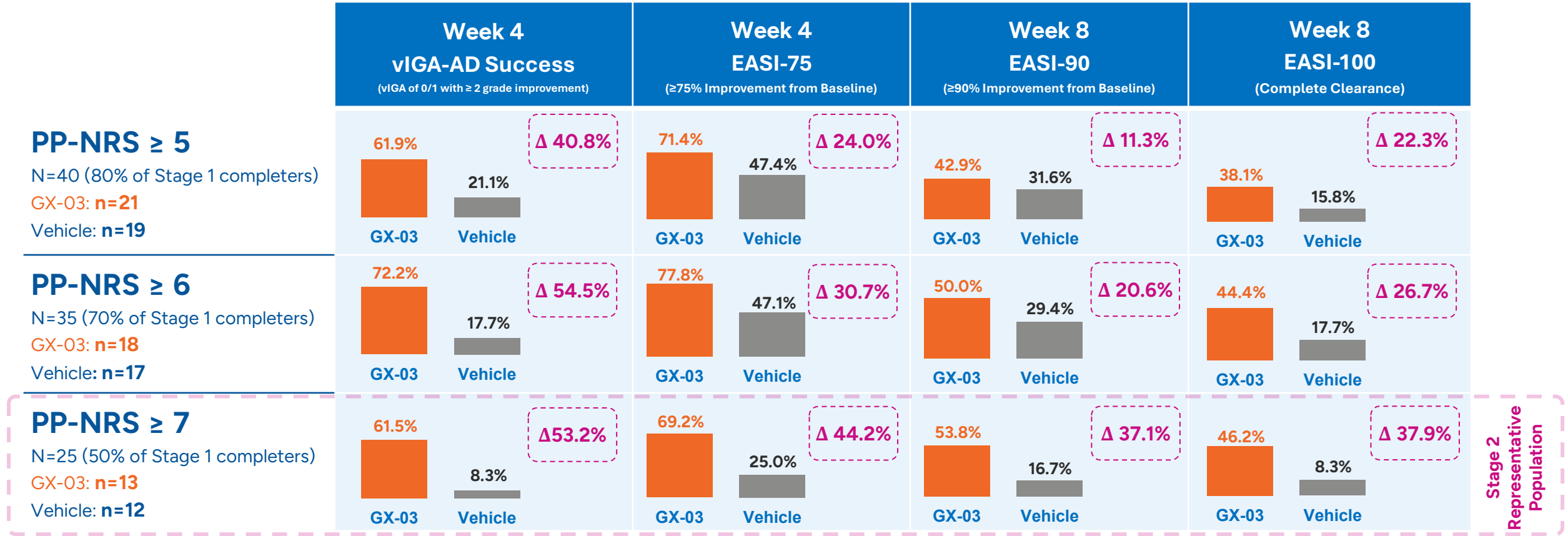
(580+ applications across all subjects)

GX-03 has been FDA cleared as **non-cytotoxic**, **non-sensitizing** and **non-irritating**

Appendix

Stage 1 Subgroup Analysis

Pruritus as Potential Predictive Biomarker for AD Success



Higher baseline pruritus severity is consistently associated with stronger responses across all four efficacy endpoints

Enriching for PP-NRS ≥ 7 increases event rates and maximizes Stage 2 efficiency

Why Enrichment Improves Stage 2 Efficiency

- *Greater treatment separation vs. vehicle observed at every endpoint*
- *Higher event rates requires smaller sample size*
- *More efficient path to achieving statistically significant results*

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Moderate-Severe AD: Assessing for Stage 2 EASI Enrichment

IGA 3/4 & Baseline EASI ≥ 10

Endpoint	GX-03 (n=9)	Vehicle (n=4)	Treatment Difference
Week 4 vIGA-AD Success vIGA of 0/1 with ≥ 2 grade improvement	**55.6%** (5/9)	25.0% (1/4)	**Δ 30.6%**
Week 4 EASI-75	**88.9%** (8/9)	50.0% (2/4)	**Δ 38.9%**
Week 8 EASI-90	**77.8%** (7/9)	50.0% (2/4)	**Δ 27.8%**
Week 8 EASI-100	**44.4%** (4/9)	50.0% (2/4)	**Δ -5.6%**

Baseline Characteristics	GX-03 n=9	Vehicle n=4
Mean EASI (SD)	22.1 (10.8)	14.8 (3.51)
Mean vIGA (SD)	3.44 (0.49)	3.25 (0.43)

Baseline characteristics favor Vehicle

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Interim Analysis – EASI 1.1 – 7 Subgroup

Presents as Meaningful on Key Disease Clearance Endpoints

Endpoints	GX-03 (n=14)	Vehicle (n=18)	Treatment Difference
Week 4 vIGA-AD Success vIGA of 0/1 with ≥ 2 grade improvement	**71.4%** (10/14)	33.3% (6/18)	**Δ 38.1%**
Week 4 EASI-100	**28.6%** (4/14)	5.6% (1/18)	**Δ 23.1%**
Week 8 EASI-100	**35.7%** (5/14)	11.1% (2/18)	**Δ 24.6%**

Baseline Characteristics	GX-03 n=14	Vehicle n=18
Mean EASI (SD)	3.59 (1.71)	3.99 (1.70)
Mean vIGA (SD)	3 (0.69)	3 (0.79)

Balanced Population

Baseline disease severity was well balanced between treatment groups, supporting interpretation of the observed treatment differences.

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