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CORPORATE PRESENTATION

March 2026 | BIO Investment and Growth Summit

Precision dermatology powered by synthetic biology.

DISCLAIMER AND SPECIAL NOTE REGARDING FORWARD-LOOKING STATEMENTS

This document has been prepared and is made for informational purposes only to familiarize yourself with Azitra, Inc. ("Azitra", the "Company," "we," "us," and "our"). This document is neither an offer to sell or purchase, nor a solicitation of an offer to sell, buy or subscribe for any securities in any jurisdiction. Nothing contained in this document is, or should be construed as, a recommendation, promise, or representation by the Company or any officer, director, employee, agent, affiliate, representative, or advisor of the Company. You should not construe the contents of this document as legal, tax, accounting or investment advice or a recommendation.

This document contains forward-looking statements concerning Azitra. The words "believe," "may," "will," "potentially," "estimate," "continue," "anticipate," "intend," "could," "would," "project," "plan," "expect" and similar expressions that convey uncertainty of future events or outcomes are intended to identify forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. These forward-looking statements include, but are not limited to, statements concerning the following: (i) our future financial and operating results; (ii) our intentions, expectations and beliefs regarding anticipated growth, market penetration and trends in our business; (iii) the timing and success of our plan of commercialization; and (iv) our ability to successfully develop and clinically test our product candidates.

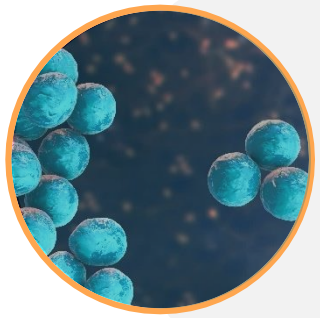
These forward-looking statements are subject to a number of risks, uncertainties and assumptions, including (i) we are an early-stage clinical biopharmaceutical company with limited operating history, (ii) there are no drug products to date that incorporate our microbial library and genetic engineering platform and the clinical and commercial utility of our microbial library and genetic engineering platform is uncertain and may never be realized; (iii) we have only recently commenced Phase 1 clinical studies of our initial product candidates and our product candidates will require extensive additional preclinical and clinical testing; (iv) we expect we will need additional financing to execute our business plan and fund operations, which additional financing may not be available on reasonable terms or at all; and (v) those other risks described or incorporated by reference in the "Risk Factors" section in our Form 10-K filed by Azitra with the Securities and Exchange Commission ("SEC") on February 24, 2025 and in any subsequently filed quarterly reports on Form 10-Q.

In light of these risks, uncertainties and assumptions, the forward-looking events and circumstances discussed in this document may not occur and actual results could differ materially and adversely from those anticipated or implied in our forward-looking statements. You should not rely upon forward-looking statements as predictions of future events. Although we believe that the expectations reflected in our forward-looking statements are reasonable, we cannot guarantee that the future results, levels of activity, performance or events and circumstances described in the forward-looking statements will be achieved or occur. Azitra does not undertake and specifically disclaims any obligation to update or revise our forward-looking statements to reflect new circumstances or unanticipated events as they occur, except as required by law.

This document contains only basic information concerning Azitra. Because it is a summary it does not contain all of the information you should consider with regard to Azitra. You should read our Form 10-K filed with the SEC on February 24, 2025 , as supplemented and/or amended from time to time, and our other filings with the SEC for more complete information about Azitra.

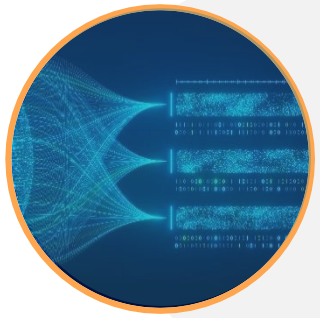
Three foundational platforms for precision dermatology

POWERED BY SYNTHETIC BIOLOGY AND THE METAGENOME



Bacterial Cell Library

- Proprietary, robust library of **~1,500 microbial strains**
- Engineered and non-engineered bacterial chasses
- Over 60 species in house, mostly *Staphylococcus epidermidis*



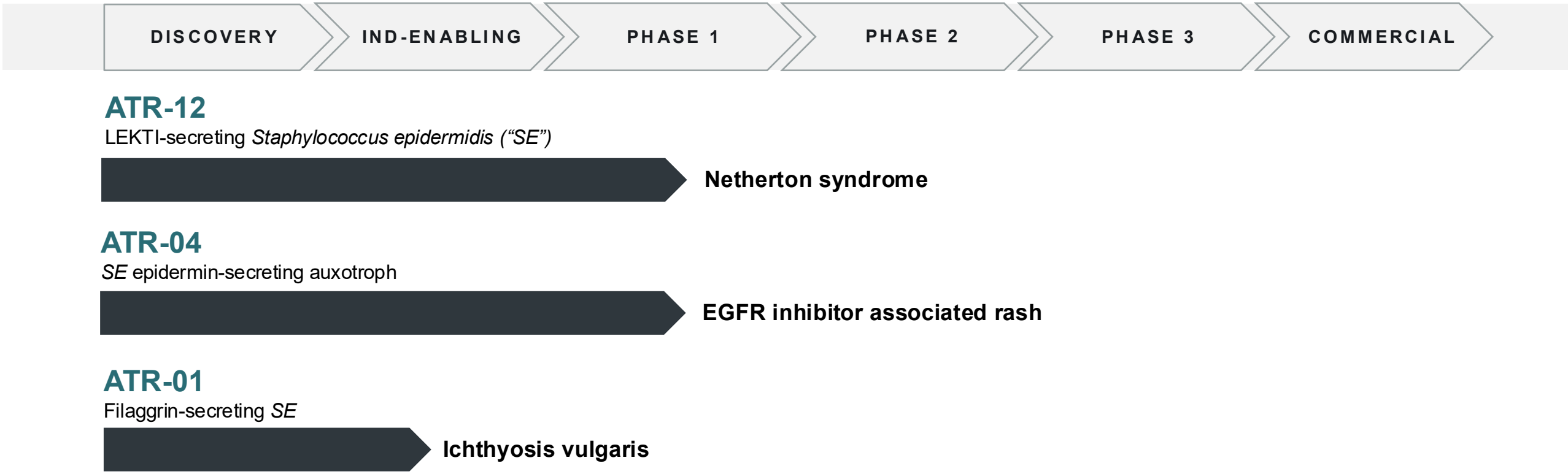
Artificial Intelligence / Machine Learning Discovery

- Predictive algorithms for **novel microbial-derived proteins, peptides & small molecules**
  - Exclusive agreement covering specific strains with team from Carnegie Mellon
  - Based on genetic sequences and biosynthetic gene clusters

Microbial Genetic Engineering Platform

- Demonstrated ability to make **novel transformations** to overcome challenge of thick cell walls and restriction modification systems
  - Exclusive worldwide license with Fred Hutchinson Cancer Center

Azitra's pipeline features multiple internally developed programs



ATR-12 Program

Netherton Syndrome

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ATR-12: LEKTI-Secreting *Staphylococcus epidermidis* for Netherton syndrome

ATR-12 Summary

- **Netherton syndrome** is a rare, orphan autosomal recessive disease with no current FDA-approved treatment option
- Characterized by severe inflammation, pruritus, scaling, red, and dehydrated skin
  - Caused by mutations in the *SPINK5* gene, which encodes the serine protease inhibitor, **LEKTI** (lympho-epithelial Kazal-type related inhibitor)
  - Results in overactive proteases causing desquamation, skin barrier defects, and activation of inflammation
  - ~10% mortality rate in infants[1]
- **Mechanism of action:** Auxotrophic ATR-12 inhibits the overactive proteases through LEKTI fragment secretion
- **Pediatric Rare Disease Designation** received from FDA

ATR-12 Key Facts

Primary Mechanism:
Topical protein replacement and kallikrein Inhibition

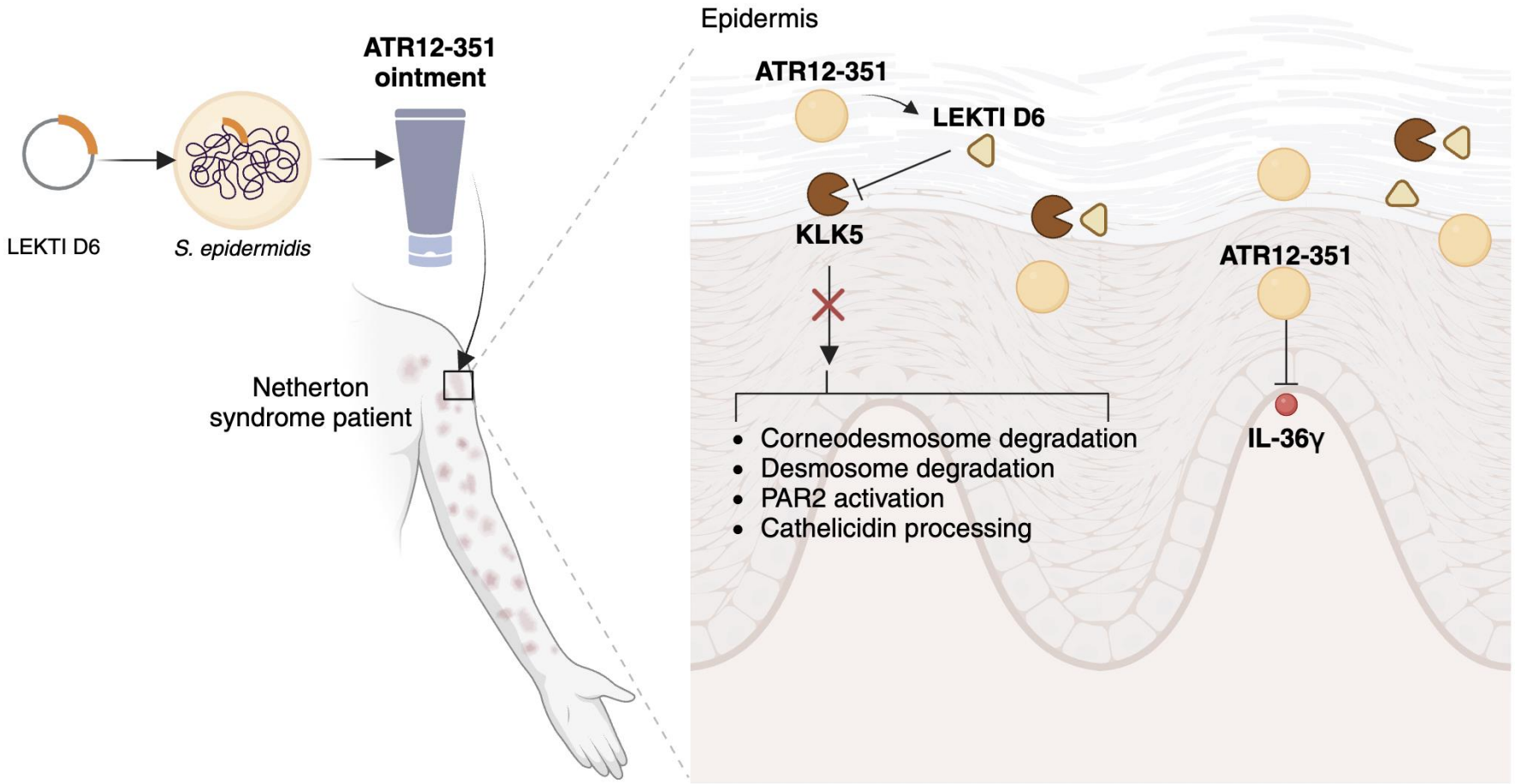
Clinical Status:
Phase 1b

Global Prevalence:
~20K+ Patients[2]

Potential Peak Sales Opportunity:
~$250M[3]

[1] Bellon N et al. Br J Dermatol. 2021.
[2] Barbati F, et al. Front Pediatr. 2021.
[3] Company estimates of 2,500 patients x $100,000 annually.

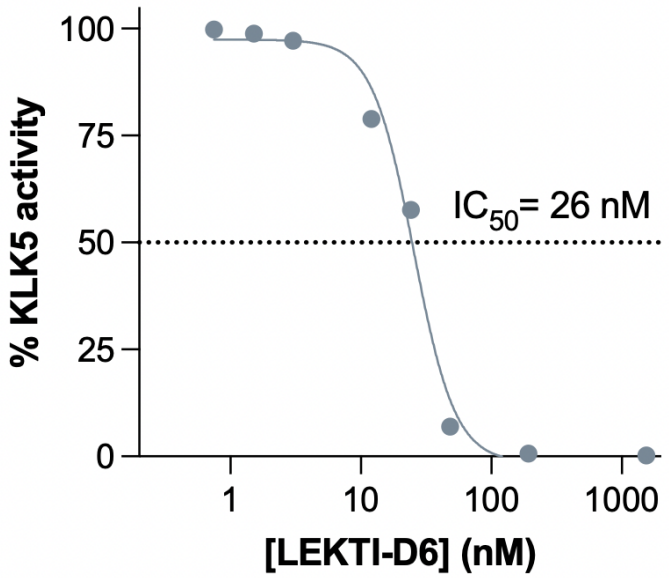
Mechanism of action of ATR-12



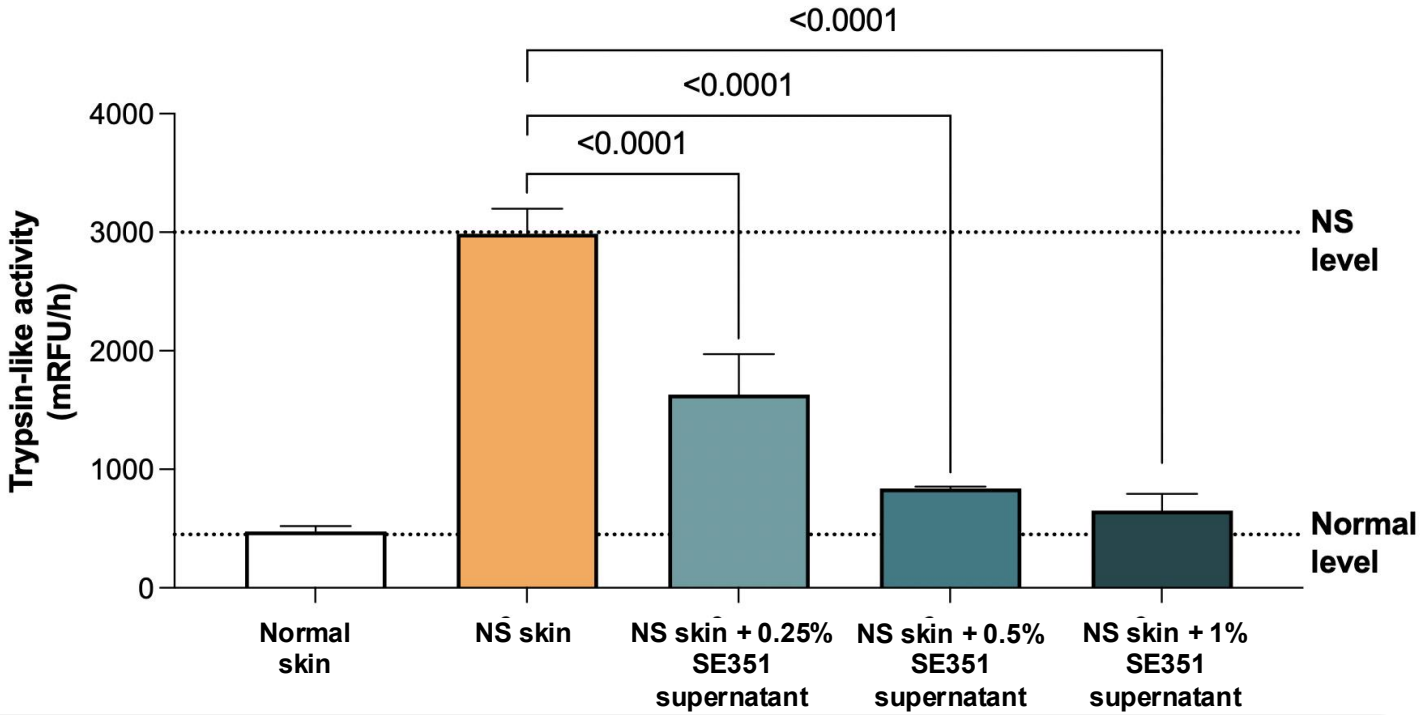
Collectively, ATR-12 addresses Netherton syndrome by LEKTI replacement and IL-36γ inhibition

ATR-12 shows potent reduction in protease activity

Nanomolar IC50 of KLK5 from SE351 *in vitro*



***S. epidermidis* strain SE351 (LEKTI-secreting) spent broth inhibition of human skin tape stripped extracts**



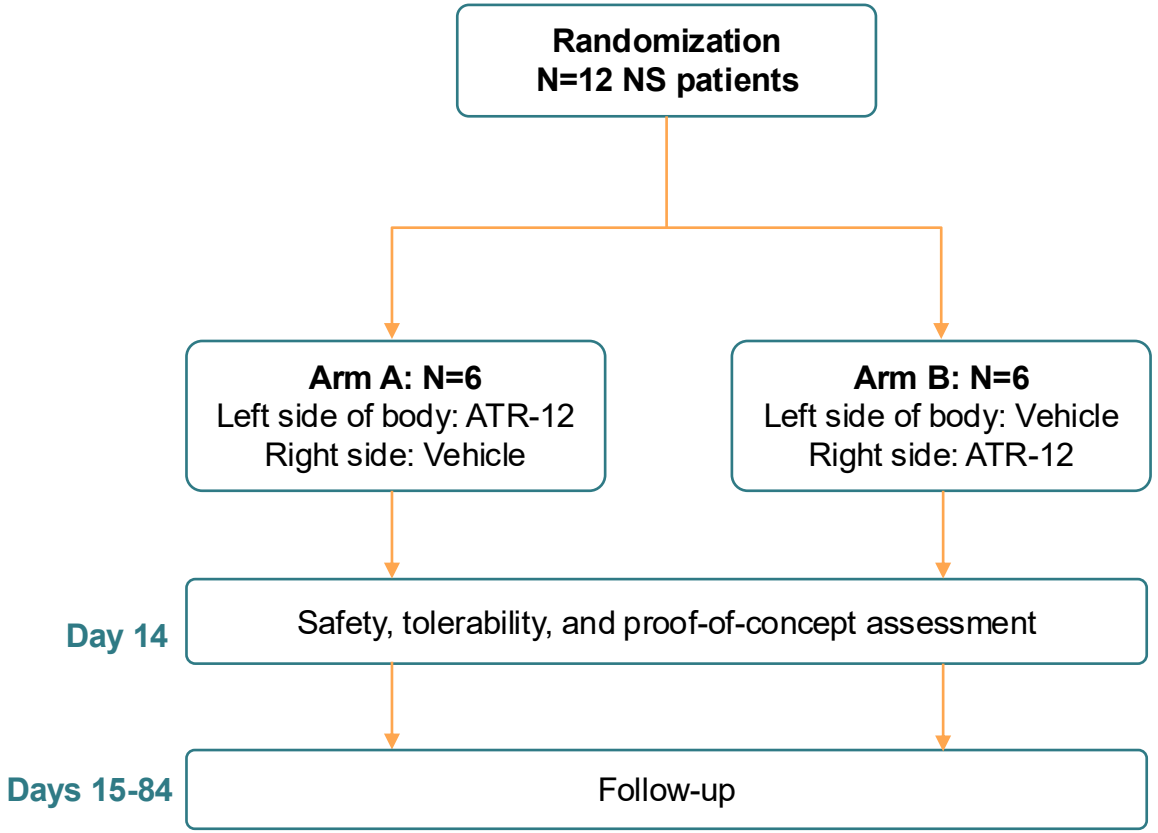
- ✓ Nanomolar inhibition of KLK5
- ✓ Trypsin-like activity (key measure of protease activity in NS patients) decreased after addition of spent broth from LEKTI-secreting strain SE351 in ATR-12
- ✓ Dose-dependent response seen across concentrations of supernatant

Phase 1 clinical trial design

Study overview

- Multicenter, randomized, double-blind, vehicle-controlled study in adults (n=12) with Netherton syndrome
  - Dose level: 10^9 CFU / g ATR-12
  - N=12 patients dosed twice daily for 14 days
- Primary objective: to assess the safety and tolerability of topical application of ATR-12
- Secondary objectives:
  - Evaluate efficacy signals (investigator and patient global assessments, NS-modified SCORAD)
  - Evaluate the skin pharmacokinetics of rhLEKTI-D6
- Exploratory objectives:
  - Evaluate pharmacodynamic parameters, including anti-rhLEKTI response, cytokine responses, biomarkers such as KLK5, KLK7, IL-36γ, TARC/CCL17, trypsin-like activity, and chymotrypsin-like activity

Design



Initial safety data

Safety overview

- ✓ Generally safe and well tolerated
- ✓ 6 patients dosed to date with safety data (N=5) as of June 13, 2025
- ✓ No reports of serious adverse events. All adverse events were mild (Grade 1) except for one transient moderate (Grade 2) event that resolved
- ✓ Local reactions at the application sites have typically been transient and self-resolving and have included mild (Grade 1) localized itch, redness, and a burning sensation with application
- ✓ Such reactions have been observed bilaterally (i.e. on both active and vehicle sides), suggesting this is not specifically an active drug related effect.

ATR-04 Program

EGFR inhibitor-associated rash

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ATR-04: auxotrophic *S. epidermidis* for EGFR inhibitor-associated rash

ATR-04 Summary

- Chemotherapy agents such as EGFR inhibitors and immunotherapies such as early BTK inhibitors lead to an aggressive and debilitating rash on most patients
- Severity of the rash is linked to IL-36γ signaling as well as correlations to *S. aureus* increases
- EGFR inhibitors produce the most prevalent and most predictable affliction
- **ATR-04** is topically administered and inhibits IL-36γ and *S. aureus*
- Fast Track designation from the FDA

ATR-04 Key Facts

Primary Mechanism:
Topical IL36γ inhibition, *S. aureus* control

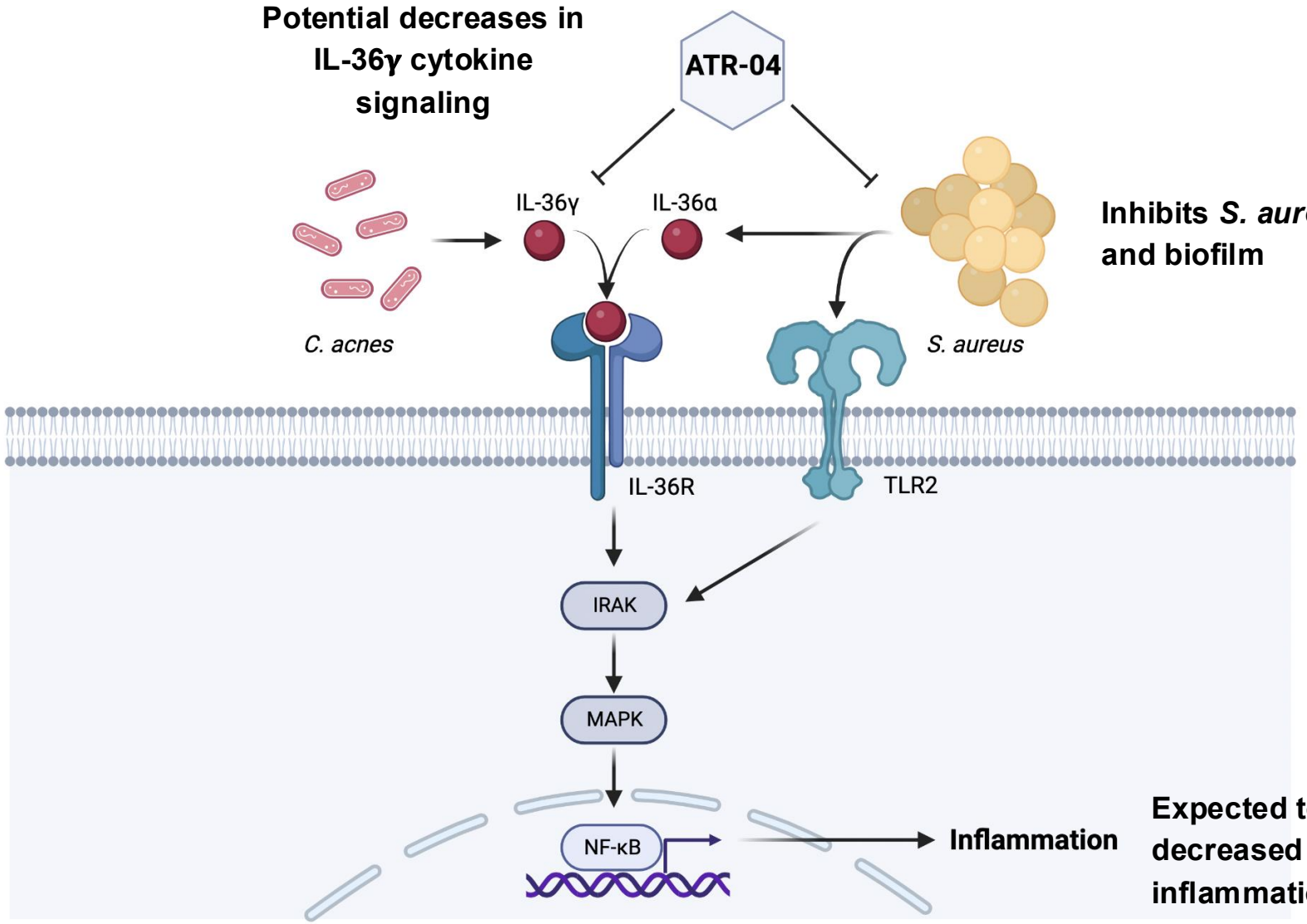
Clinical Status:
Phase 1/2 ready

US Prevalence:
~150,000 patients[1]

Potential Peak Sales Opportunity:
>$1B[2]

[1] Bloomberg/Symphony drug prescription data and FDA labels
[2] Company estimates of 150,000 patients x $10,000/year.

Mechanism of action of ATR-04



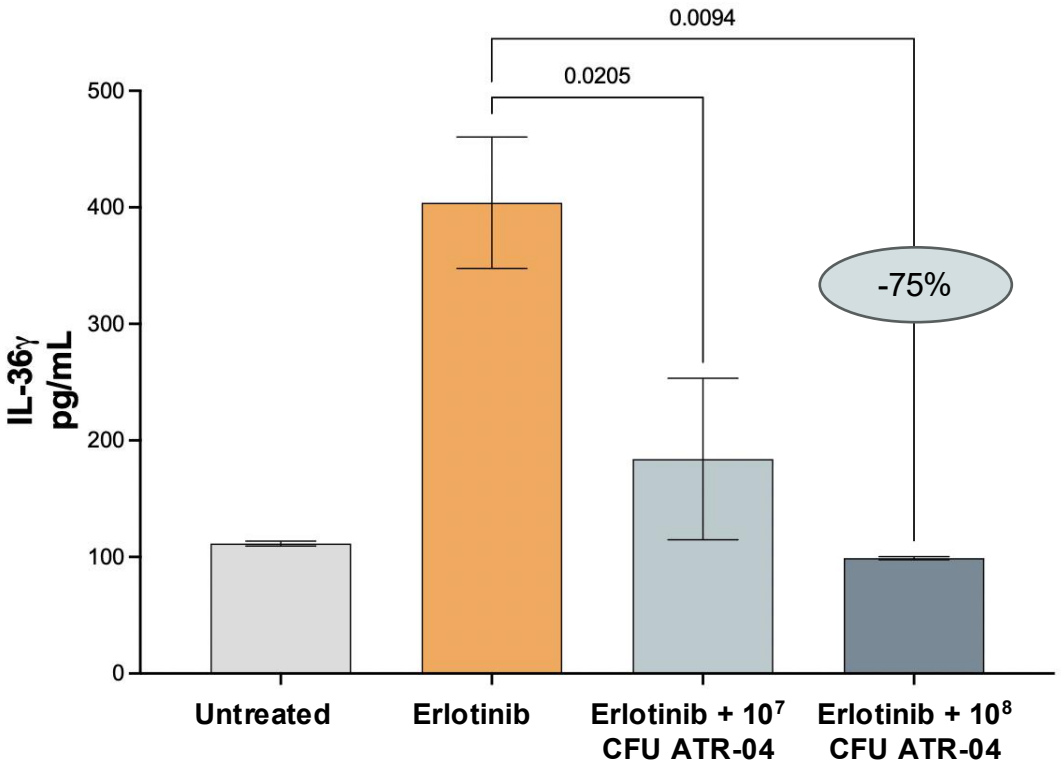
Inhibits *S. aureus* growth and biofilm

Expected to lead to decreased inflammation

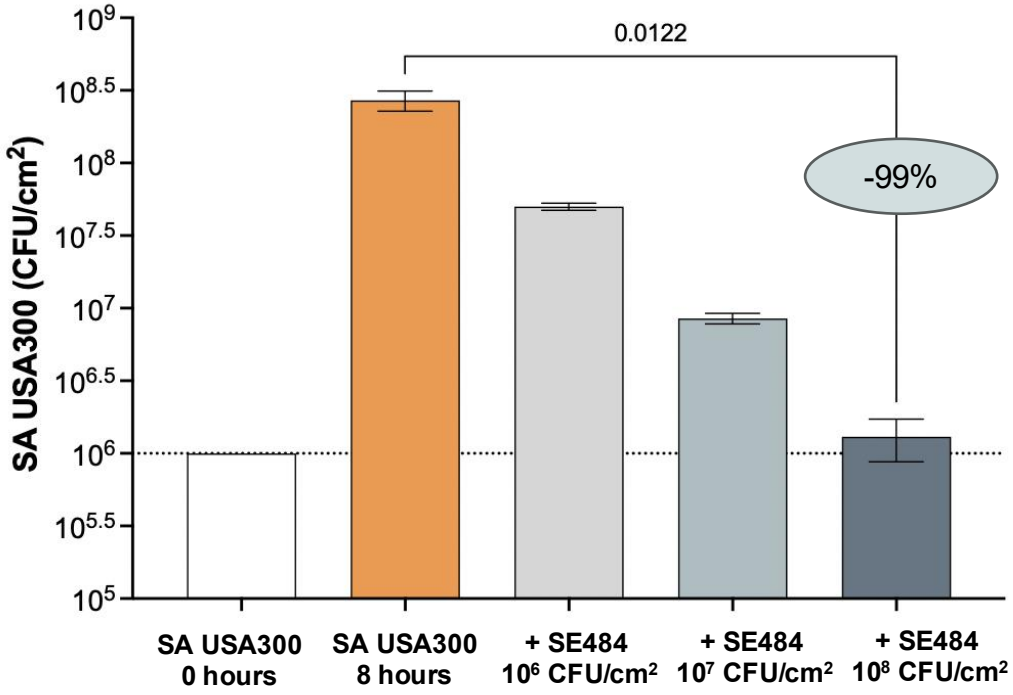
Collectively, ATR-04 addresses rash severity driven by EGFR inhibition

In vitro data show ATR-04 reduces erlotinib-induced IL-36γ and *S. aureus*

IL-36γ reduction in reconstructed human epidermis



***S. aureus* reduction on *ex vivo* pig skin**



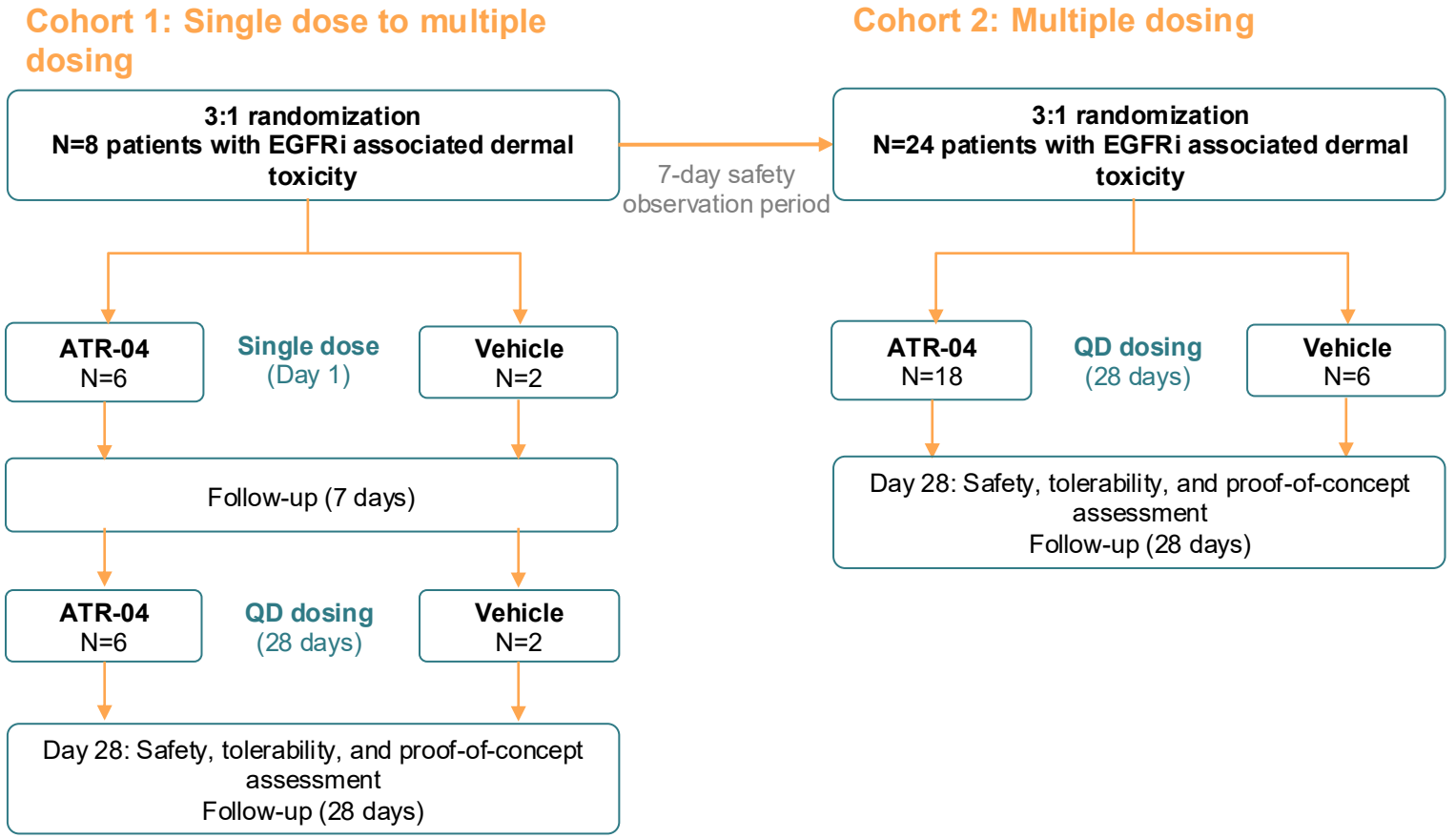
- ✓ IL-36γ is elevated in reconstructed human epidermis following erlotinib exposure
- ✓ ATR-04 reduces IL-36γ induced by erlotinib and reduces *S. aureus*
- ✓ Dose-dependent effect observed

Phase 1/2 clinical trial design: IND cleared August 2024

Study overview

- Multicenter, randomized, double-blind, vehicle-controlled study in adults (n=32) with EGFRi associated dermal toxicity
  - Dose level: 10^9 CFU / g ATR-04
  - Cohort 1 (n=8): single dose leading to multiple dose for 28 days
  - Cohort 2 (n=24): multiple dose cohort for 28 days
- Primary objective: to assess the safety and tolerability of topical application of ATR-04
- Secondary objectives:
  - Evaluate efficacy signals (modified CTCAE, pruritus, and pain)
  - Bioavailability of ATR-04
- Exploratory objectives:
  - Evaluate PD parameters, including IL-36γ
  - Quality of life questionnaire

Design



ATR-01 Program

Ichthyosis vulgaris

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ATR-01: topical filaggrin protein for ichthyosis vulgaris

ATR-01 Summary

- **Ichthyosis vulgaris** is a common autosomal dominant disease with no current FDA-approved treatment option
  - Characterized by dry, rough, and scaly skin
  - Caused by loss-of-function mutations in the *filaggrin* gene
- **Mechanism of action:** ATR-01 delivers missing filaggrin protein to result in restoration of the skin barrier and skin hydration
  - A human filaggrin subunit is attached to a peptide (RMR) that facilitates transdermal delivery

ATR-01 Key Facts

Primary Mechanism:
Topical protein replacement

Clinical Status:
IND-enabling studies

US Prevalence[1]:
>1M patients

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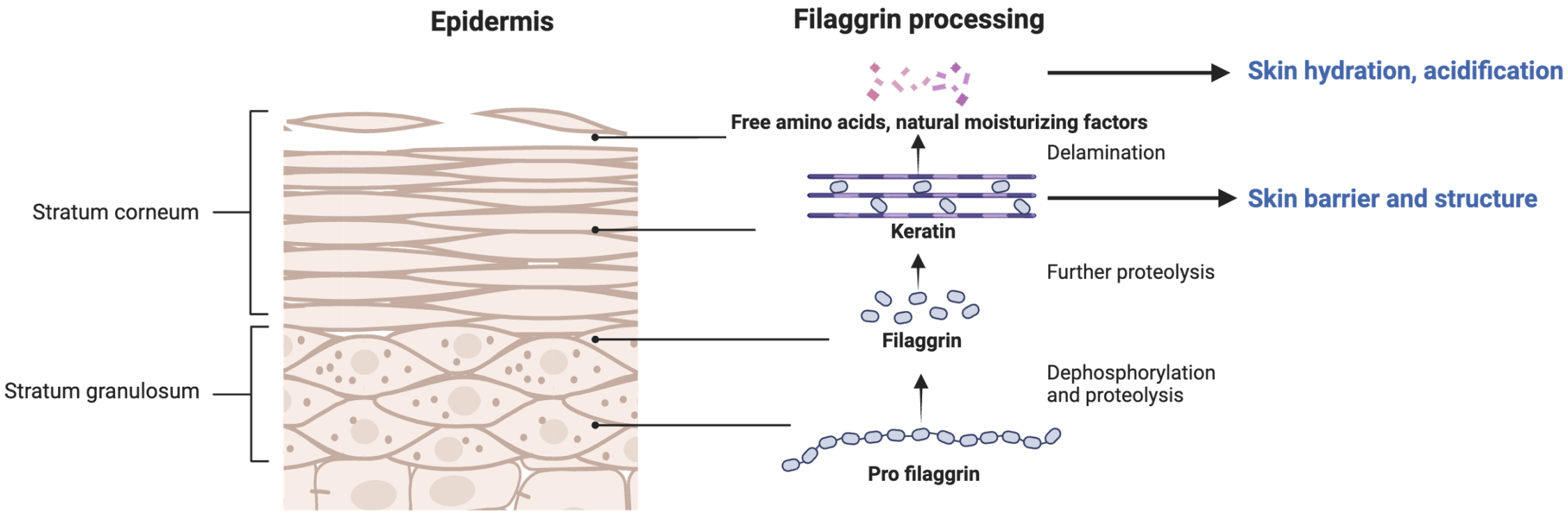
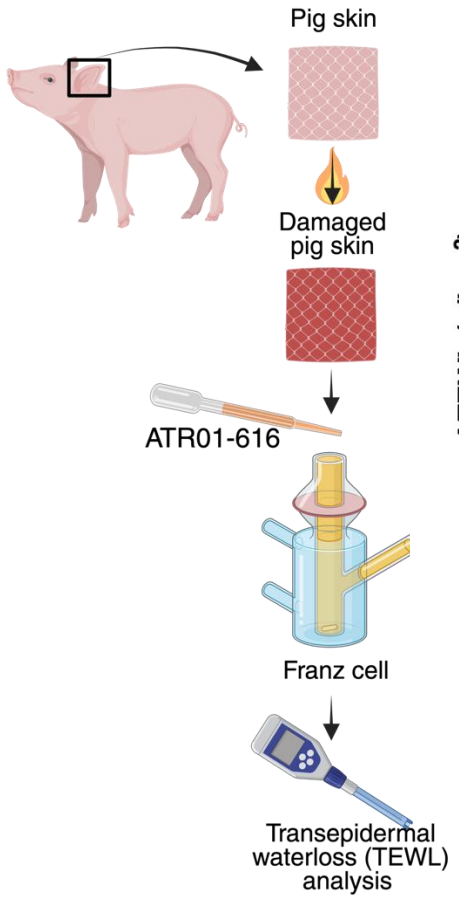
[1] Süßmuth K, et al, J Dtsch Dermatol Ges. 2020.

Restoring filaggrin function in the skin

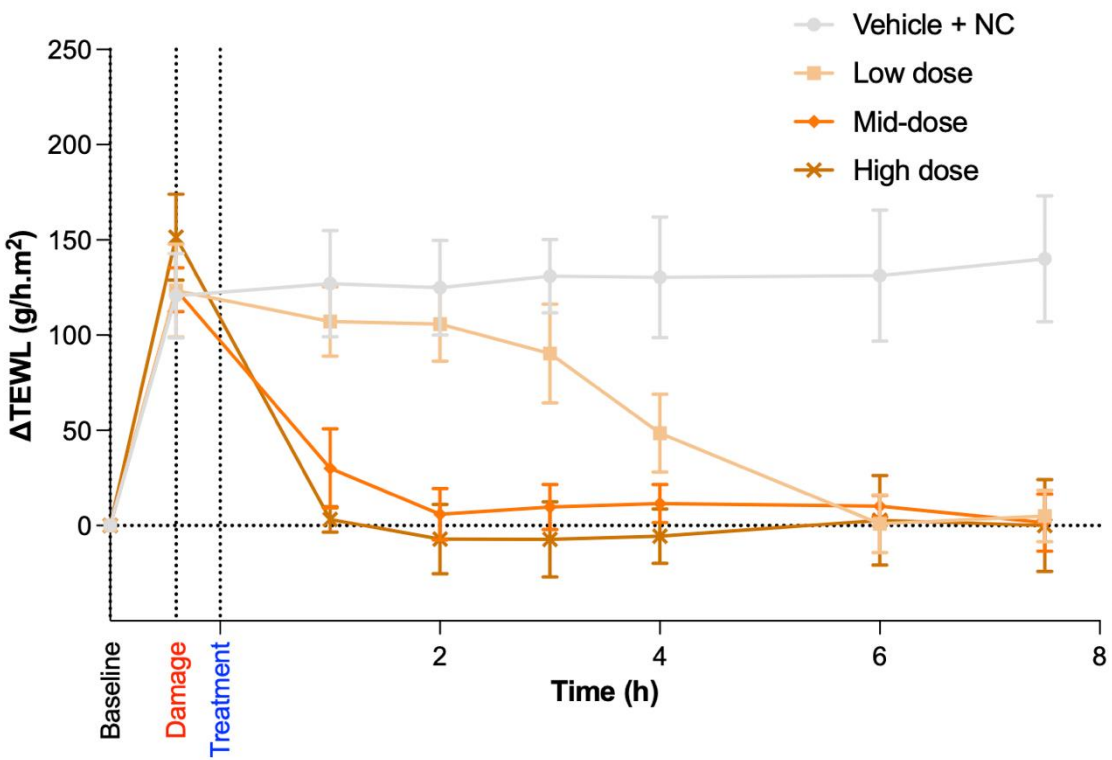


ATR-01 (SE616) decreases transepidermal water loss (TEWL) on *ex vivo* damaged skin

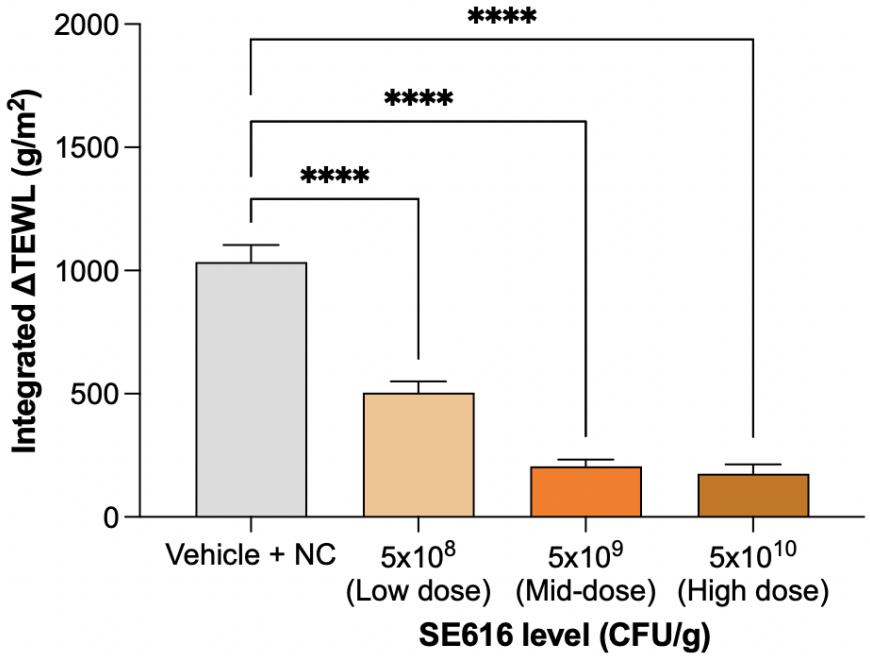
Ex vivo model



Change in TEWL over time



Total change in TEWL



Future Directions

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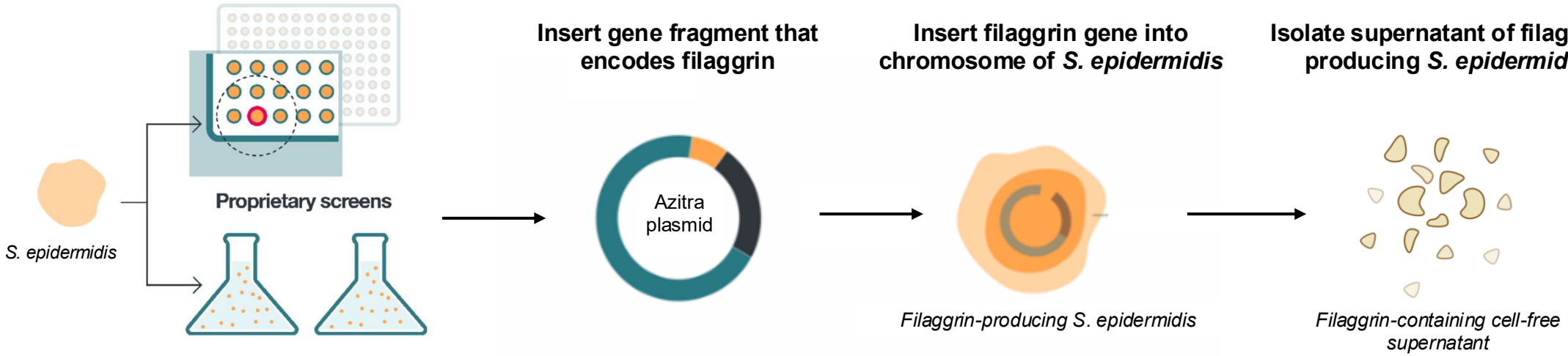
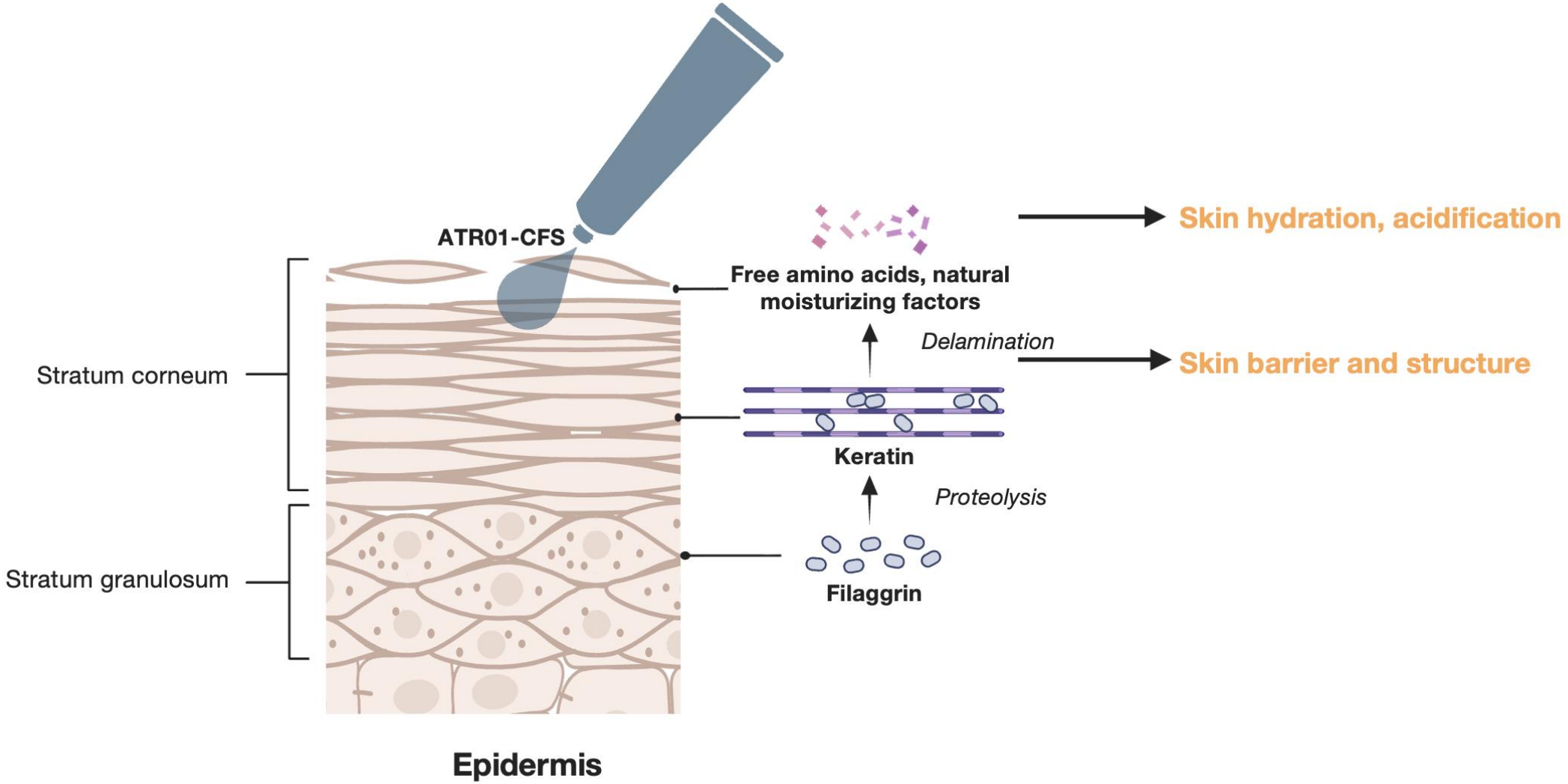
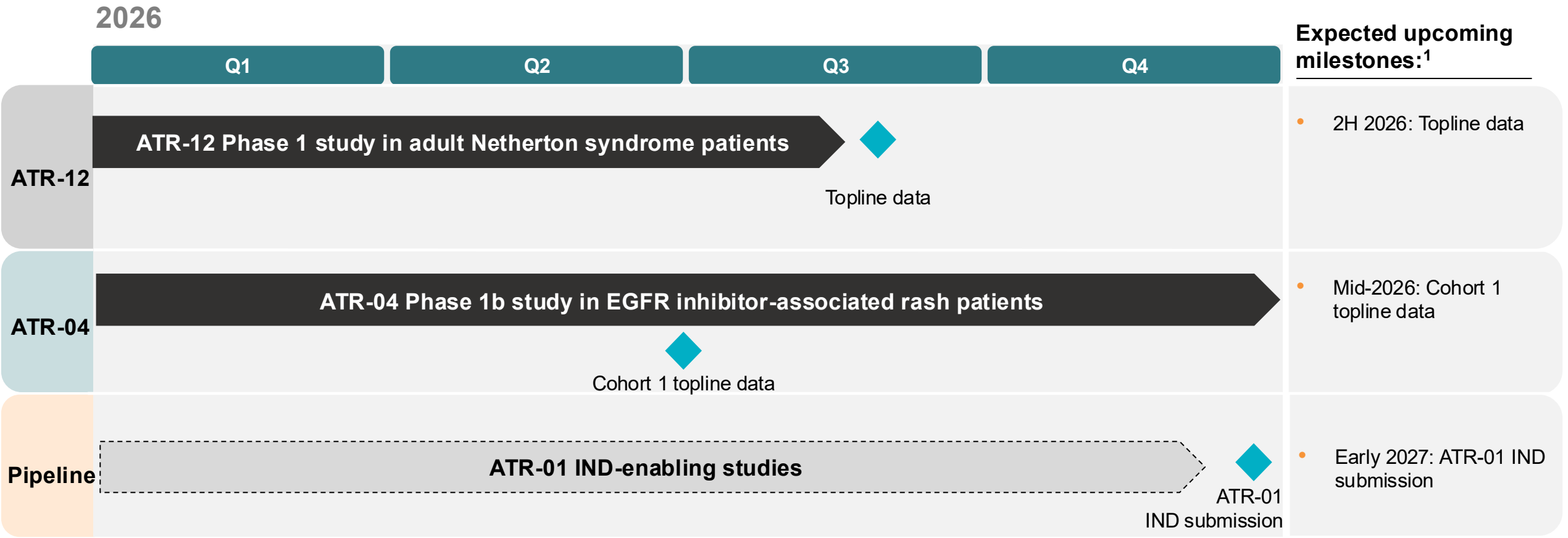
Topical filaggrin + bacterial lysate for cosmeceutical indications



Mechanism of action for addressing the appearance of fine lines and wrinkles



ATR-12 and ATR-04 bring value-creating anticipated milestones in 2026[1]



[1] Upcoming milestones are estimates.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THANK YOU
Precision dermatology powered by synthetic biology.

APPENDIX

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ATR-01 summary

- ✓ ATR01-616 delivers human filaggrin in a **dose-dependent manner**, and delivers filaggrin to the stratum granulosum in human skin
- ✓ Filaggrin delivered from ATR01-616 is **functional**, measured by keratin collapse assays and shown to colocalize with keratin in fluorescent imaging
- ✓ ATR01-616 leads to reduced transepidermal water loss (TEWL) in a heat-damaged pig skin model, **demonstrating initial proof-of-concept**
- ✓ Additional IND-enabling studies are ongoing with an **IND submission in 2026**

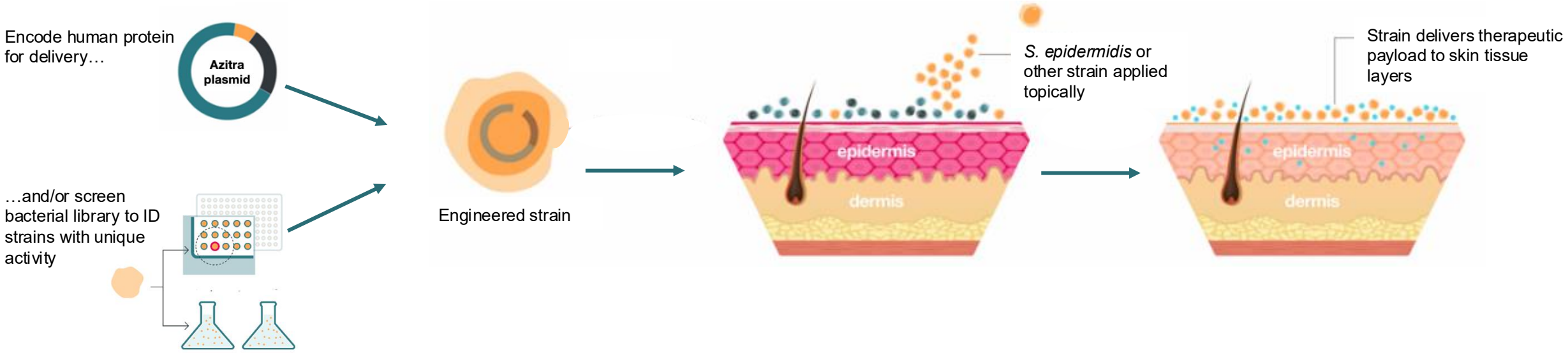
Using synthetic biology and *S. epidermidis* biology for skin therapeutics

1 **Identify human or bacterial protein or molecule of interest**

2 **Engineer auxotrophy to control strain growth**

3 **Colonize affected skin with engineered strain**

4 **Secrete active molecule throughout layers of the epidermis for therapeutic treatment**



Azitra is led by world-class management team

Francisco Salva, MSc.
President and CEO

- Prior Co-Founder and VP of Operations at Acerta Pharma – Sold for $6.3 billion
- Formerly Senior Director –Corporate Development at Pharmacyclics
- 25+ years experience in life science venture capital, investment banking and operating roles

Travis Whitfill, M.P.H.
Co-Founder and COO

- Prior Partner at Bios Partners, a venture capital fund with $350M+ assets under management
- Assistant Professor Adjunct in the Department of Pediatrics at Yale University
- Named one of Forbes' 30 Under 30 in healthcare in 2018

Norman Staskey, CPA
CFO

- Currently Acting CFO via Danforth Advisors
- Previously, Managing Director at E&Y
- 20+ years accounting experience, including multiple IPO, SPAC and M&A transactions

Mary Spellman, M.D.
Acting CMO

- Prior CMO of Revance Therapeutics, Menlo Therapeutics, and Castle Creek Biosciences
- Previously Scientific Director at Biogen and Novartis
- 30+ years of dermatology and broad industry experience, including 10+ NDAs

Leonard Milstone, M.D.
Professor Emeritus of Dermatology
Yale School of Medicine
Azitra Scientific Advisory Board

- Led the group that first demonstrated gene editing in the epidermis
- Discovered the unique proteoglycan Epican as well as keratins 4 and 13
- Former Chair, Medical and Scientific Advisory Board, Foundation for Ichthyosis and Related Skin Types

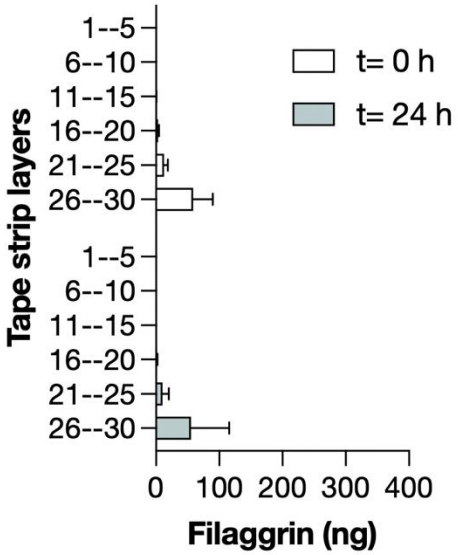
Differentiated approach in an attractive space

Azitra's differentiated approach to precision dermatology

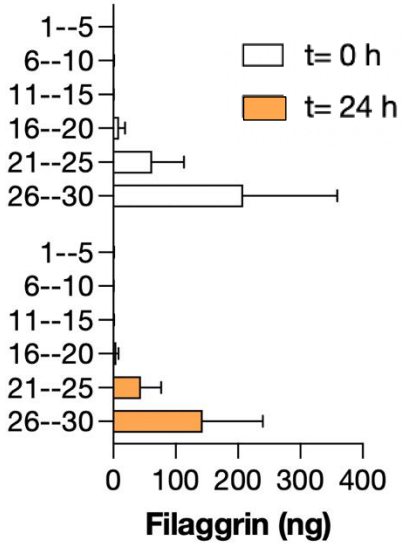
	Penetrates or delivers to skin	Disease modifying	Safe	Low cost	Ease of use
Oral small molecules	−	✗	−	✓	✓
Topical small molecules	✓	✗	−	✓	✓
Injectable antibodies	−	−	−	✗	✗
Topical gene therapy	−	✓	✓	✗	✓
Other gene therapies	✓	✓	−	✗	✗
azitra™	✓	✓	✓	✓	✓

Penetration of filaggrin into *ex vivo* human skin using ATR01-616

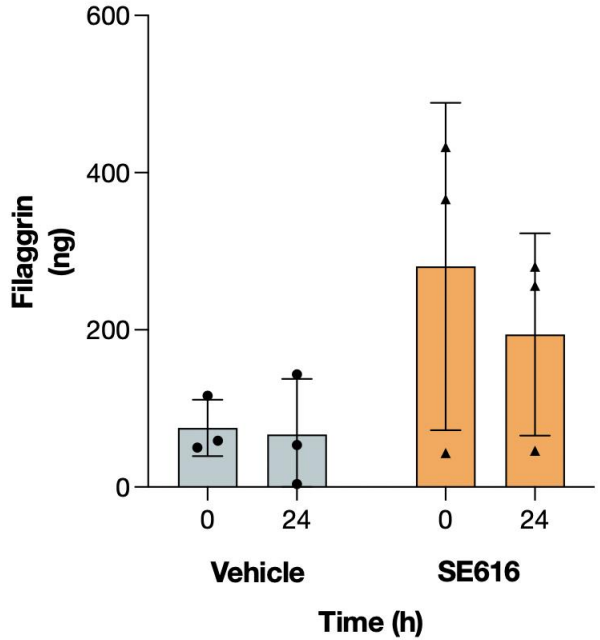
Vehicle



ATR01-616



Total filaggrin



- ✓ Filaggrin delivery from ATR01-616 treatment is significantly higher after 24 hours compared to T0 in all layers following ATR01-616 application
- ✓ Filaggrin penetrates to at least 30 layers deep in substantial amounts of protein delivered after 24 hours