



Corporate Presentation

September 2026



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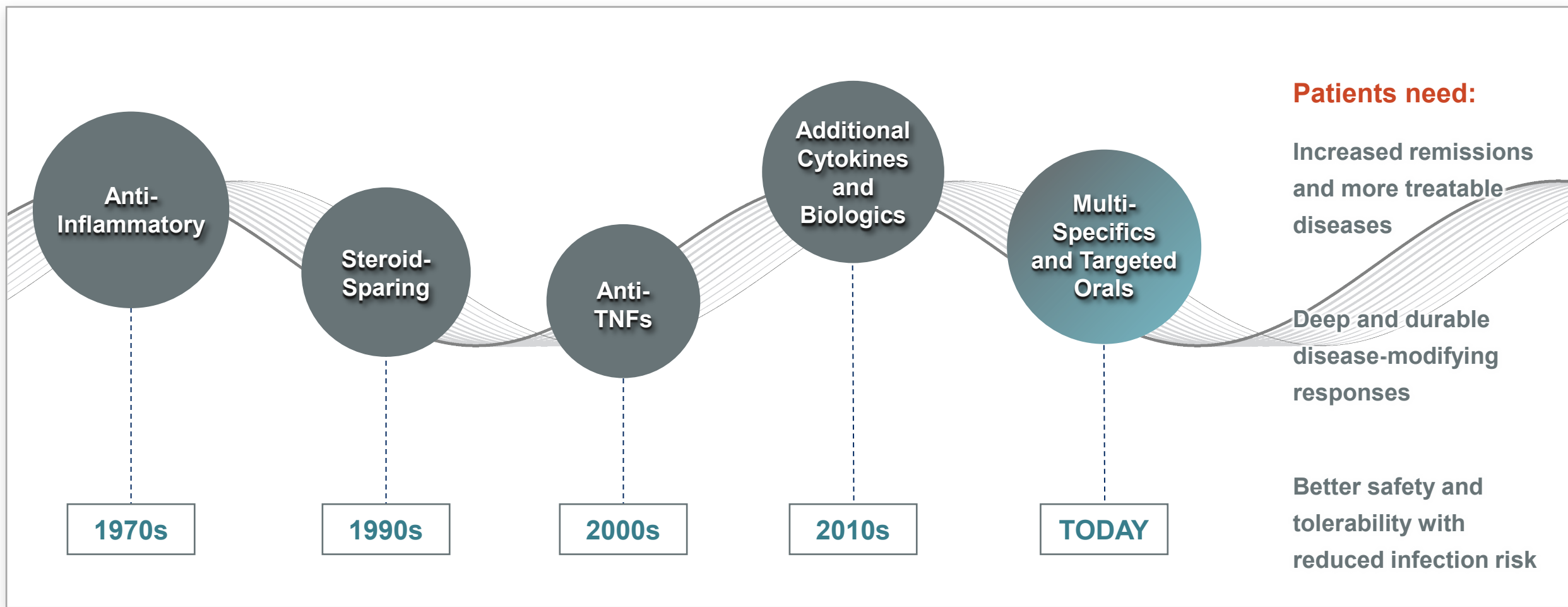
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Few Patients With Autoimmune Disease Have Durable Remission



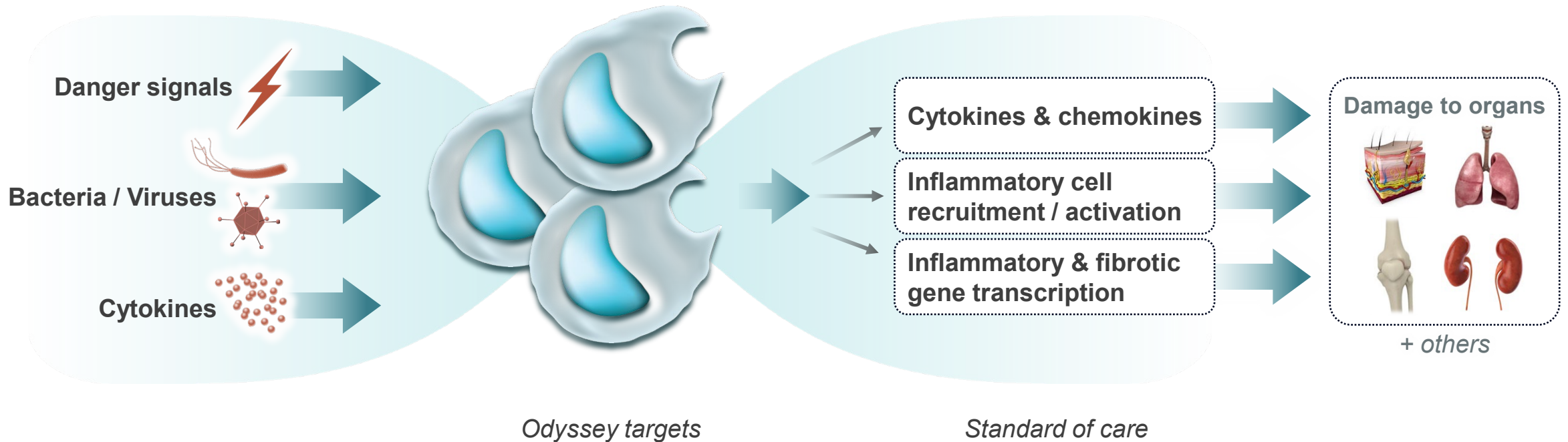
Targeting Upstream Signaling Has Potential to Unlock Durable Remissions

Defined pathogenic triggers underlie inflammatory disease...

...by converging on disease-specific signaling nodes...

...initiating a *broad* immune response...

...causing tissue damage and disease



Experienced Team and Organization to Maximize Success

Strategy

- First-in-class targets
- Validated pathways and mechanisms of action
- Modality agnostic

Portfolio

- Targeting large-market I&I indications
- Multiple programs at development candidate stage or later

People

- Experienced board and senior management
- Proven drug hunters
- Company builders successful from discovery through commercialization

Execution

- Lean and agile structure built for speed
- Phase 2 completion on homegrown program within 4.5 years
- Average time from program initiation to DC: 1–1.5 years

Capital

- Efficient capital allocation with disciplined program prioritization
- Balance sheet of \$433M as of June 30, 2026; expected to fund operations into H2 2028

LEADERSHIP

Gary Glick, Ph.D.
Founder and CEO



Tony Oipari, M.D., Ph.D.
Chief Medical Officer

Natalie Dales, Ph.D.
Research Operations

Joe McDonald, Ph.D.
Chief Data Officer

Jolie Siegel
*EVP, General Counsel,
and Secretary*

Jason Haas
Chief Financial Officer

Collin Todd
*SVP, Strategy and
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Pioneering Next-Generation I&I Therapies to Redefine Standard of Care

INFLAMMATORY BOWEL DISEASE

Differentiated **first-in-class, oral monotherapy** with positive safety profile

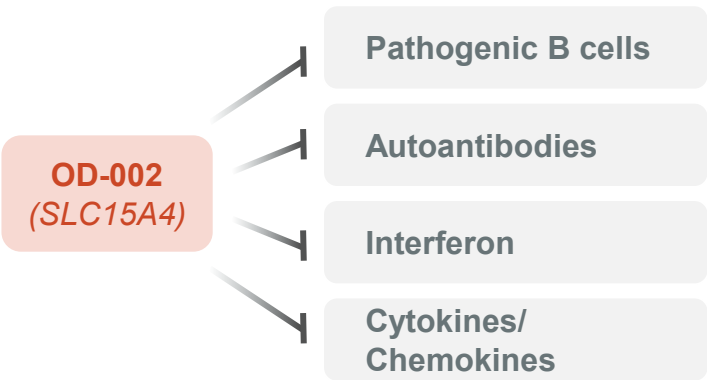
Potential **paradigm-changing combinations** to break the induction and maintenance therapeutic ceiling



Current market: **\$25B** » Future market: **\$40B**

B CELL-MEDIATED DISEASES

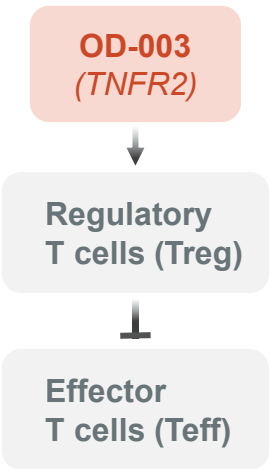
Potential **first-in-class**, oral therapy to block pathogenic B cells



Current market: **\$30B** » Future market: **\$65B**

INFLAMMATORY AND AUTOIMMUNE DISEASES

Potential **first-in-class** protein agonist to induce stable regulatory T cells



Current market: **\$100B**



Source: Evaluate Pharma.

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In search of extraordinary for patients

Broad Therapeutic Pipeline for Diseases With Unmet Need

Target <i>Modality</i>	Indications	Discovery	IND- Enabling	Phase 1	Phase 2	Phase 3	Anticipated upcoming clinical milestones
RIPK2, OD-001 <i>Small molecule</i>	<i>UC, CD</i>						Oct 2026: Updated monotherapy data at UEG H2 2027: Phase 2a combination readout H2 2027: Phase 2b monotherapy readout
SLC15A4, OD-002 <i>Small molecule</i>	<i>CLE, nephropathies, and B cell-mediated diseases</i>						H2 2026: CTA filing H1 2027: Phase 1/2 initiation
TNFR2, OD-003 <i>Protein</i>	<i>AD, SLE, alopecia areata</i>						
TSLP/IL-33, OD-004 <i>Protein</i>	<i>COPD, asthma</i>						

\$433 million cash as of June 30, 2026 with expected runway into H2 2028



Notes: UC = ulcerative colitis; CD = Crohn's disease; CLE = cutaneous lupus erythematosus; AD = atopic dermatitis; SLE = systemic lupus erythematosus; COPD = chronic obstructive pulmonary disease; UEG = United European Gastroenterology Week.

Data to Build a Potential RIPK2 IBD Franchise Expected in 2027 and 2028

**Monotherapy topline
induction efficacy**



March 2026

**Primary and secondary
endpoints met:**
27% clinical remission rate
in moderate-to-severe UC
patients with **similar
activity in AT-experienced
patients**

**Full induction efficacy and
exploratory assessments**



October 2026

Presentation will include data
on **additional patients,
subgroup analyses, and
exploratory biomarkers**

**Break therapeutic ceiling
through innate-adaptive
combinations for induction**



2027

**Goal: Deepen induction
efficacy** with OD-001 +
vedolizumab by targeting
innate inflammation and
lymphocyte trafficking

**Demonstrate potential of
RIPK2 monotherapy as next
SoC maintenance therapy**



2028

**Goal: Safe, well-tolerated,
and efficacious oral therapy**
that blocks the initial step of
inflammation in IBD with
composition of matter IP to at
least 2043



Note: IBD = inflammatory bowel disease; AT = advanced therapy.

Oral RIPK2 Scaffolding Inhibitor: OD-001

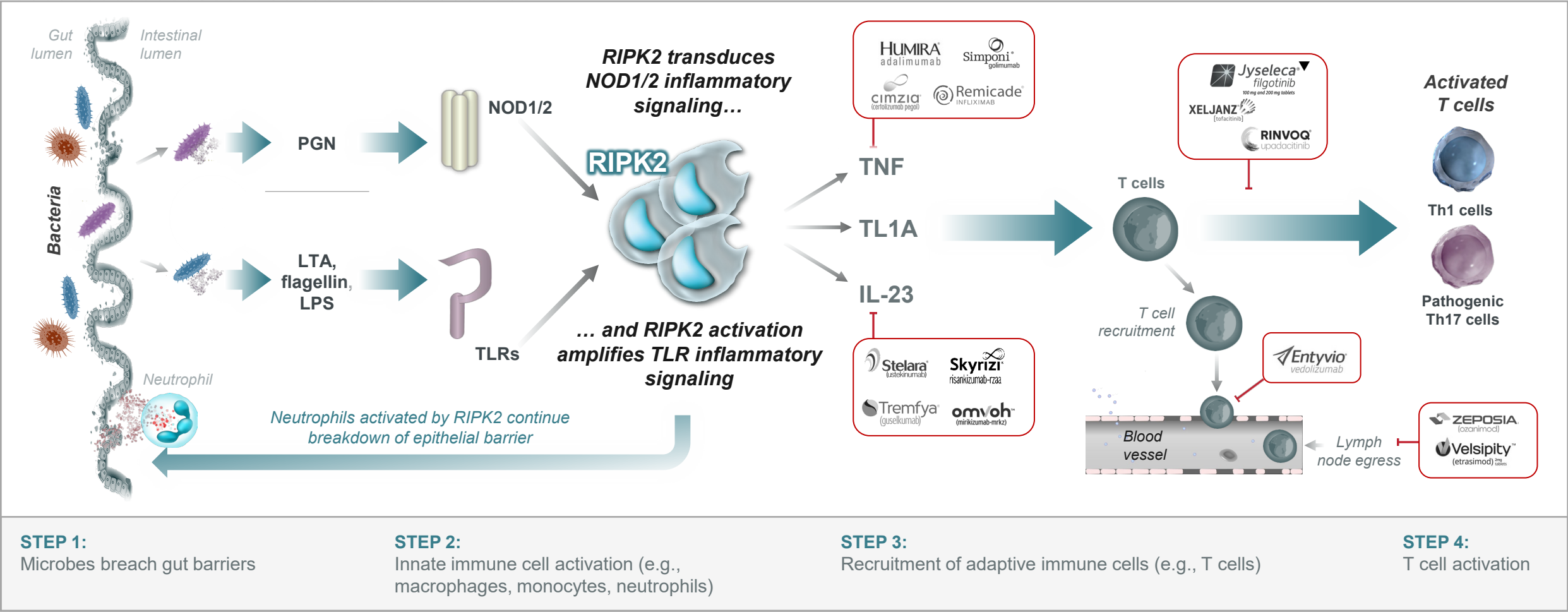
*Addressing a Root Cause of
Inflammation in IBD Using a Novel
Mechanism of Action*

*Proof-of-Concept
Achieved in
Ulcerative Colitis*

Program Inception to Phase 2 Proof-of-Concept: 4.5 Years

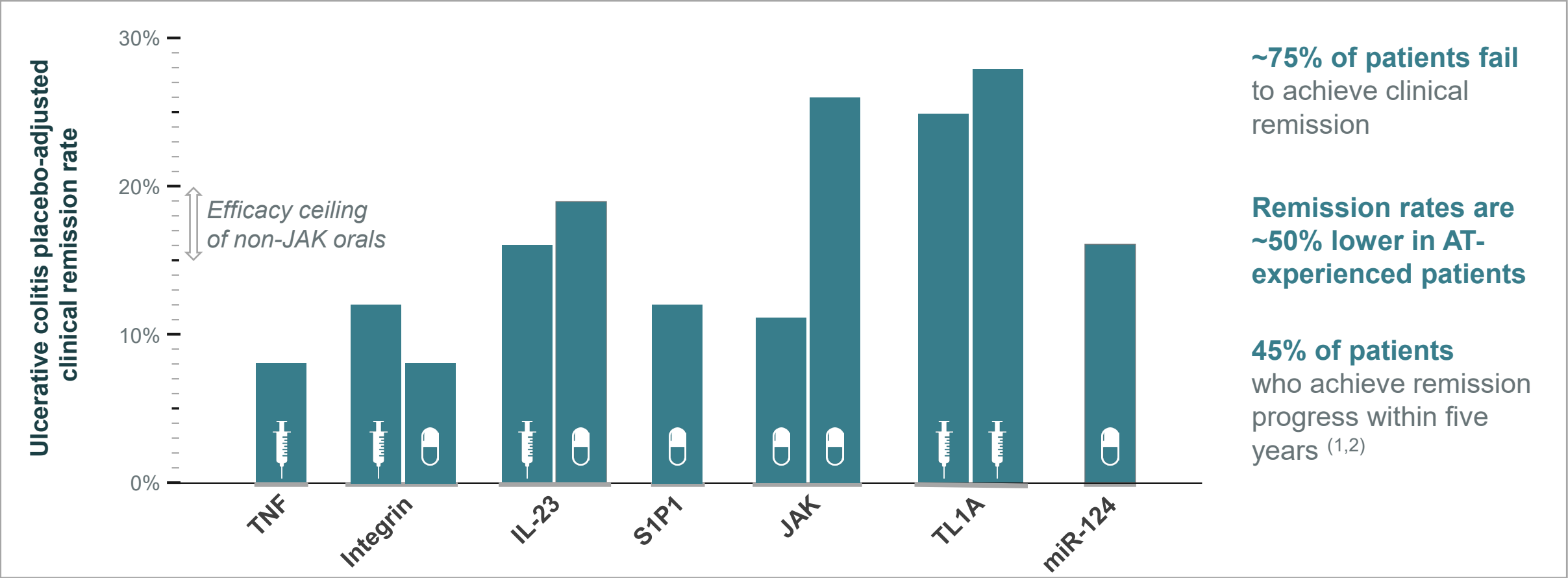


Innate Immune Responses to Gut Microbes is the Root Cause of IBD



Notes: PGN = peptidoglycan; LTA = lipoteichoic acid; LPS = lipopolysaccharide.

Even With Advanced Therapies, Durable Remission Rates for IBD Remain Low

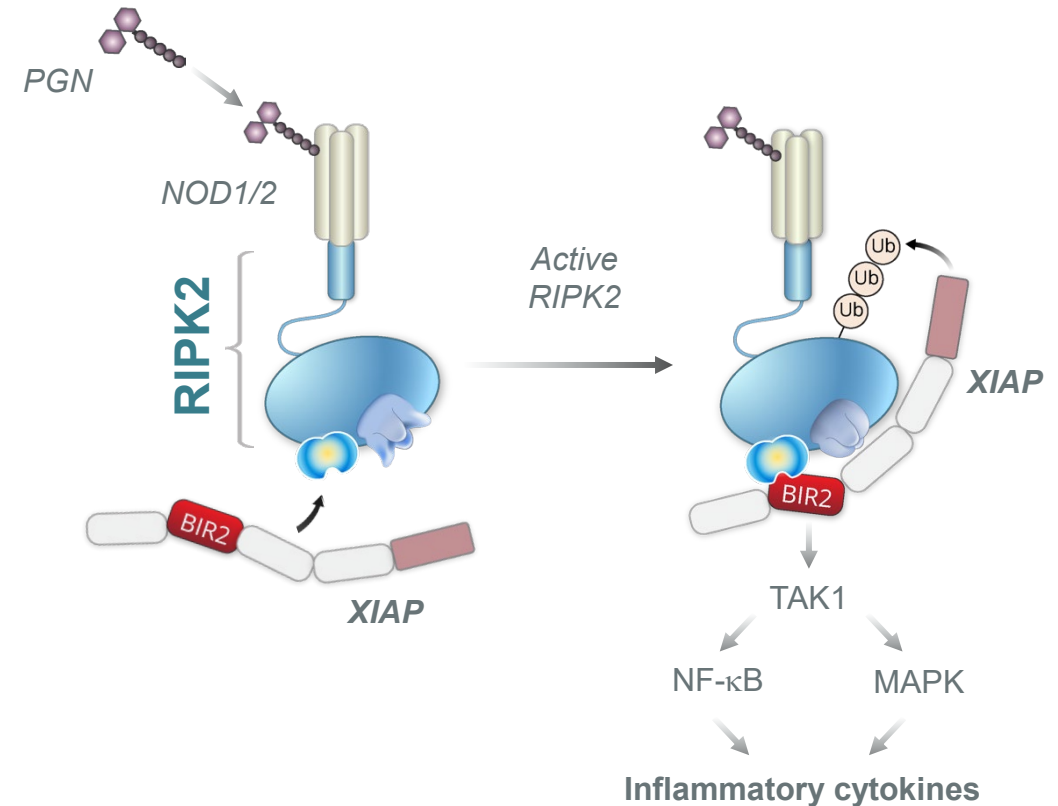


Sources: (1) Clin Gastroenterol Hepatol 2015;13(3):531; (2) World J Clin Cases 2024;12(10):1718; FDA drug labels; clinicaltrials.gov; EMERALD-2, UEG 2025; ANTHEM-UC, UEG 2025; RELIEVE UCCD, ECCO 2025; ABTECT-1 and ABTECT-2, UEG 2025.

We Believe RIPK2 Is an Ideal Target for Treating IBD

- ✓ RIPK2 has low homology to other RIPK proteins and a distinct function in inflammatory signaling
- ✓ RIPK2 activity correlates with disease severity
- ✓ Gene polymorphisms that increase RIPK2 activation predispose individuals to IBD
- ✓ RIPK2 involvement in IBD is recapitulated in animal models
- ✓ Inhibiting RIPK2:XIAP scaffolding blocks multiple pro-inflammatory cytokines
- ✓ RIPK2 knockout mice were developmentally normal and protected from IBD

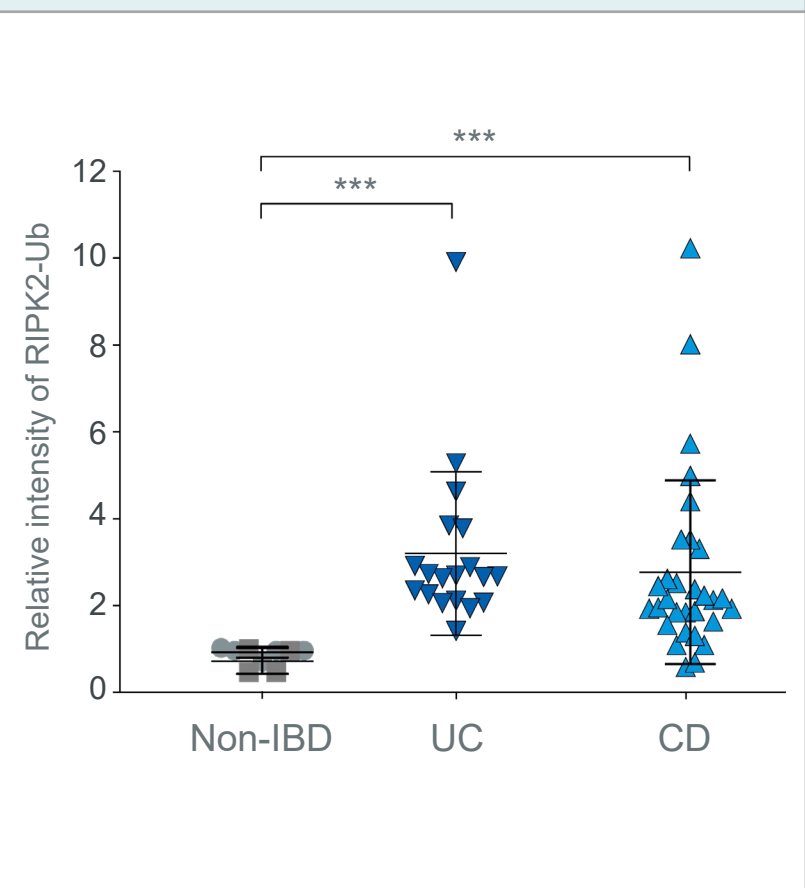
RIPK2 activation leads to scaffolding with XIAP and inflammatory signaling



Sources: Inflamm Bowel Dis 2015;21(4):862; J Med Chem 2022;65(13):9312; J Med Chem 2019;62(14):6482; Proc Natl Acad Sci U S A 2014;111(25):E2559; Am J Physiol Gastrointest Liver Physiol 2021;321(5):G500; Commun Biol 2020;3(1):140; J Med Chem 2016;59(10):4867; Int Immunol 2019;31(10):669; United European Gastroenterol J 2023;12(1):103; Front Immunol 2019;10:419; EMBO J 2018;37(17):e99372; Mol Cell 2018;69(4):551; Cell Rep 2018;22(6):1496.

RIPK2 Is Activated in UC and CD and Correlates With Severity

Increased levels of activated (ubiquitinated) RIPK2 in patients with UC and CD

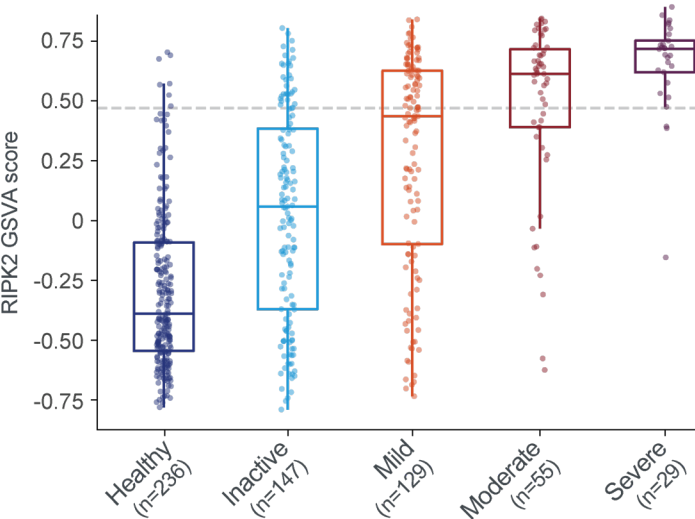


Increased expression of RIPK2-dependent gene signature in UC and CD patient biopsies

UC patients with RIPK2 pathway activity: 22% 48% 67% 90%

P-value vs. healthy: 2e-12 1e-24 2e-22 2e-17

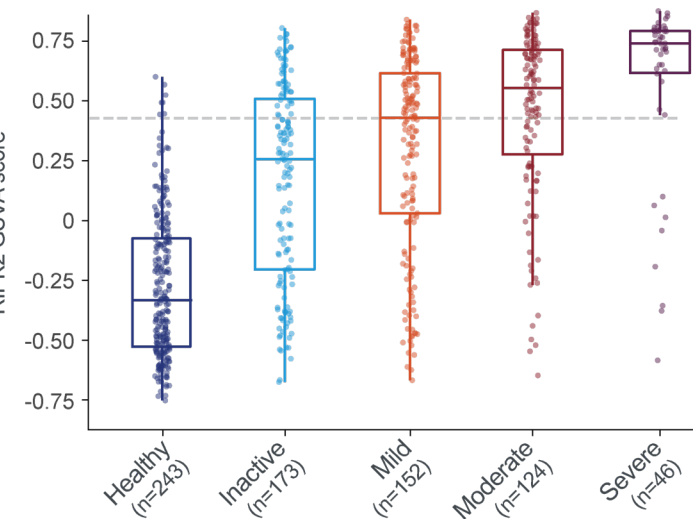
Disease score: 0 1 2 3



CD patients with RIPK2 pathway activity: 32% 52% 66% 82%

P-value vs. healthy: 6e-24 5e-35 4e-40 4e-21

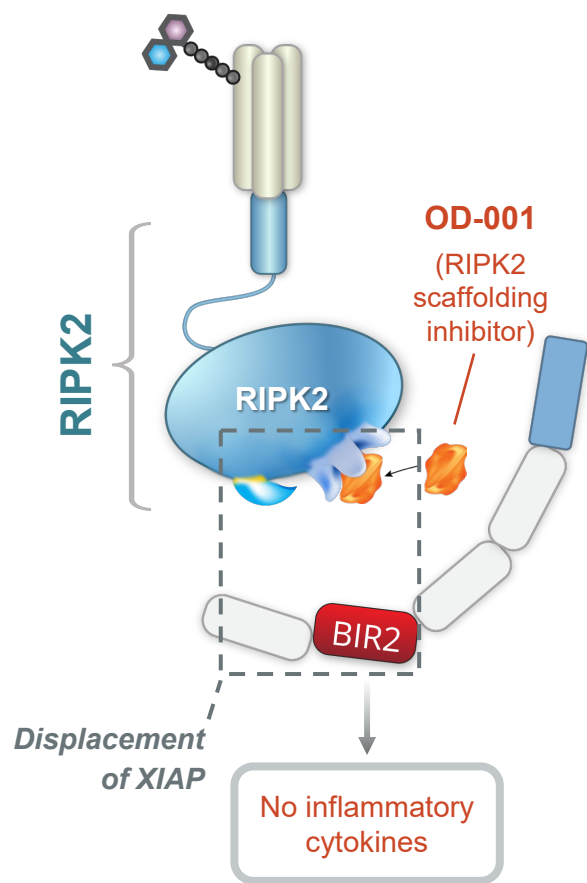
Disease score: 0-2 3-6 7-15 ≥16



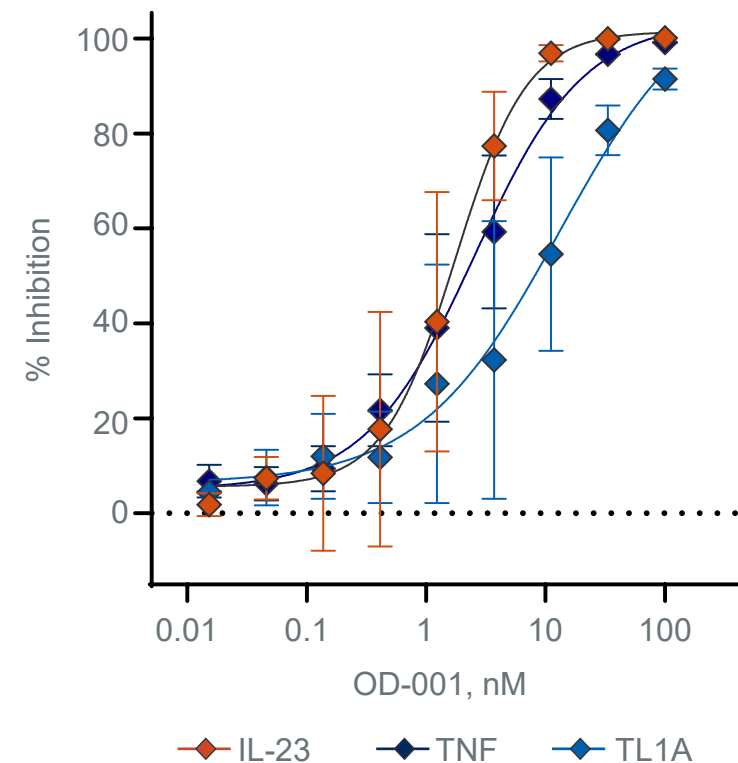
Notes: *** $p < 0.001$; GSVA = gene set variation analysis; GSVA cutoff for % of UC or CD patients with RIPK2 pathway activity is 0.46 or 0.42, respectively.
Sources: EMBO J 2018;37(17):e99372; Sci Signal 2024;17(819):eabn1101; gene expression datasets utilized = GSE193677.

OD-001 Achieves Selective Blockade of RIPK2 Scaffolding With XIAP

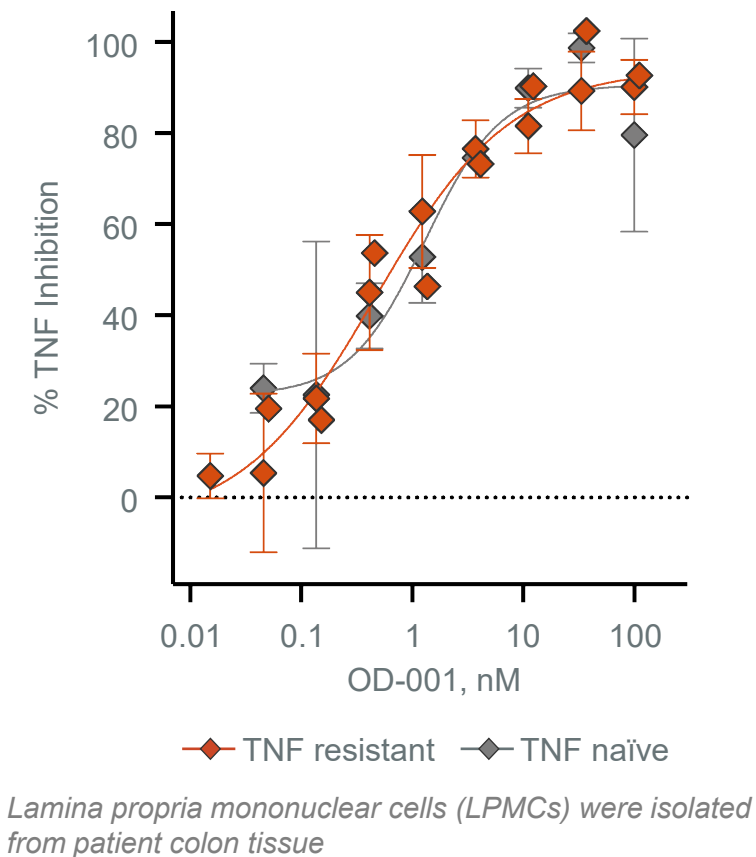
OD-001 binds to RIPK2 and holds the protein in a conformation that reduces the affinity for XIAP



OD-001 blocks cytokine production from macrophages stimulated with PGN



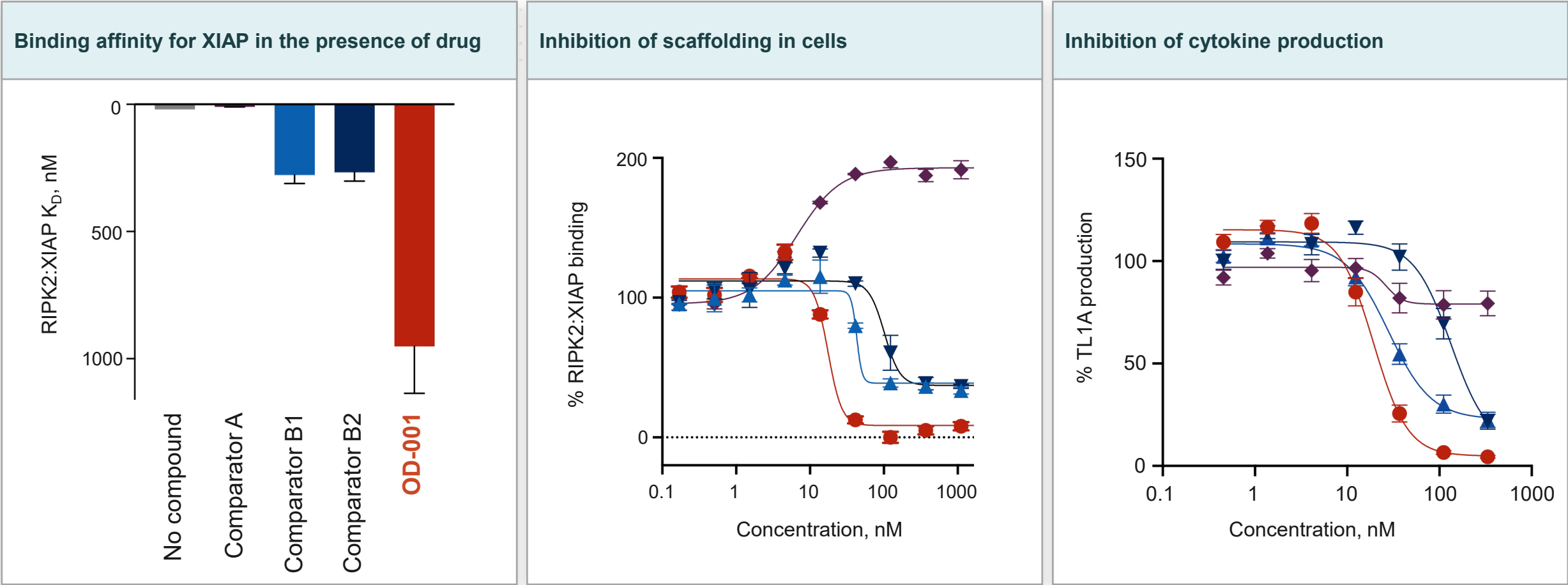
OD-001 activity is not reduced by TNF resistance



Notes: Indicated cell types were treated with compound for 1 hour and then stimulated with PGN. Cytokine production was assessed after 24 hours by MSD/ELISA.
Sources: EMBO J 2018;37(17):e99372; Mol Cell 2018;69(4):551.



Only OD-001 Fully Inhibits RIPK2 Scaffolding in Cells and Completely Blocks Inflammatory Cytokine Production



◆ Comparator A ▲ Comparator B1 ▼ Comparator B2 ● OD-001

Notes: (Left) The K_D for XIAP BIR2 dissociation from RIPK2 kinase domain in the presence of indicated compound calculated with data from a TR-FRET assay with purified proteins; (Middle) Full length XIAP binding to full length RIPK2 in HEK293 in the presence of indicated compound for 4 hours was measured by NanoBRET; (Right) human macrophages were treated with compounds for 1 hour and then stimulated with PGN. Cytokine production was assessed after 24 hours by ELISA. Sources: Comparator A WO2022192760 ex A6; Comparator B1 WO2024254539 ex 253; Comparator B2 WO2024254539 ex 52.



Phase 2a Monotherapy Trial Results



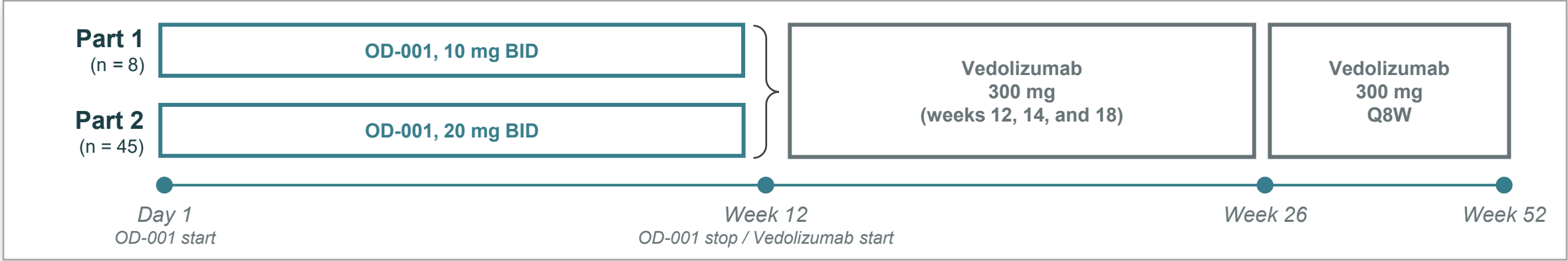
Phase 2a Monotherapy Trial Overview

Trial design ⁽¹⁾

- Enrolled patients with moderate-to-severe UC, modified Mayo Clinical score (MMCS) of 5–9; allowed up to three prior advanced therapies
- Both doses expected to be active based on phase 1 ex vivo TNF release assay
- Approach to minimize placebo effect:
 - Required endoscopic subscore (Mayo endoscopic score, MES) ≥ 2 and rectal bleeding subscore of ≥ 1
 - Centralized and blinded endoscopy reads
 - Limited concomitant steroid dose to maximum of 20 mg prednisone
 - Site and investigator selection; Odyssey team involved in all site initiations

Efficacy endpoints and powering

- Primary: 2-point change in 3-component MMCS at week 12
 - 90% power at the two-sided 5% significance level
- Secondary: $\geq 15\%$ placebo-adjusted clinical remission rate at week 12 (comparison with historic placebo data of 10%)
 - 82% power at 10% significance level



Notes: Based on results observed in part 1, an additional seven patients were enrolled and evaluated for efficacy in a 10 mg BID expansion group in Q1 2026 and results from this cohort will be disclosed at UEG 2026; (1) additional detail on the phase 1 or phase 2a trial is included in the final prospectus filed with the SEC in connection with our May 2026 initial public offering starting on page 120, which you can access [here](#).



Enrolled Participant Characteristics Are Similar to Contemporary UC Trials

	OD-001	Obefazimod		MORF-057		Spyre (α4β7)	Spyre (TL1A)	Icotrokinra	Tulisokibart	Duvakitug	Afimkibart
Phase	Phase 2a	Phase 2b	Phase 3	Phase 2a	Phase 2b	Phase 2	Phase 2	Phase 2b	Phase 2	Phase 2b	Phase 2b
Age, mean	40	41	42	39	40	44	45	42	41	41	39
Sex, male	51%	59%	-	54%	55%	72%	58%	58%	53%	63%	60%
Advanced therapy experienced ⁽¹⁾	32%	49%	47%	40%	31%	19%	35%	43%	47%	39%	39%
Modified Mayo score (mean)	6.5	7.1	6.9	6.7	6.7	6.8	6.9	6.6	7.0	6.8	7.0 (median)
MES = 3	51%	71%	60%	49%	50%	56%	56%	59%	73%	56%	52%
Median fecal calprotectin, µg/g	1,785	1,647	1,666	2,043 (mean)	-	-	-	1,523	1,307	-	1,511
Mean duration of disease (years)	8	8	8	-	7	5	7	8	7	8	7
Concomitant corticosteroid use	21%	52%	40%	26%	39%	42%	40%	37%	55%	42%	42%

Notes: (1) Includes both approved advanced therapies and investigational agents; includes golimumab, etrasimod, ustekinumab, rosnilimab, MORF-057, PN-943, infliximab, icotrokinra, TYK2 inhibitor, adalimumab, and etrolizumab.
Sources: Lancet Gastroenterol Hepatol 2022;7(11):1024-1035; Abivax corporate presentation, March 2023; Abivax phase 3 topline readout, July 2025; Obefazimod UEG Week presentation, October 2025; Clin Gastroenterol Hepatol 2026;24(2):525-534; Spyre SKYLINE Part A induction results, April 2026; Spyre corporate presentation, June 2026; Icotrokinra UEG Week presentation, October 2025; Prometheus Biosciences corporate presentation, March 2023; Duvakitug phase 2 investor call, February 2025; Lancet Gastroenterol Hepatol 2025;10(10):882-895.



OD-001 Was Well Tolerated at 10 mg BID and 20 mg BID With a Promising Safety Profile Observed

Treatment-emergent adverse events	Part 1: 10 mg BID, n = 8	Part 2: 20 mg BID, n = 45 ⁽¹⁾
Participants with any TEAE (n, %)	2 (25%)	20 (44%)
Severe (grade ≥ 3) TEAE	0	0
Drug-related TEAE	0	2 (4%)
Liver function abnormality identified as TEAE	0	0
TEAE leading to trial drug discontinuation ⁽¹⁾	0	0
SAE	0	0
Participants with any TEAE of special interest:		
Elevated body temperature that meets specific criteria	0	0
Elevated hsCRP that meets specific criteria	0	0
Serious infection	0	0
Participants with most common AEs:		
Fever	0	5 (11%)
Headache	0	4 (9%)

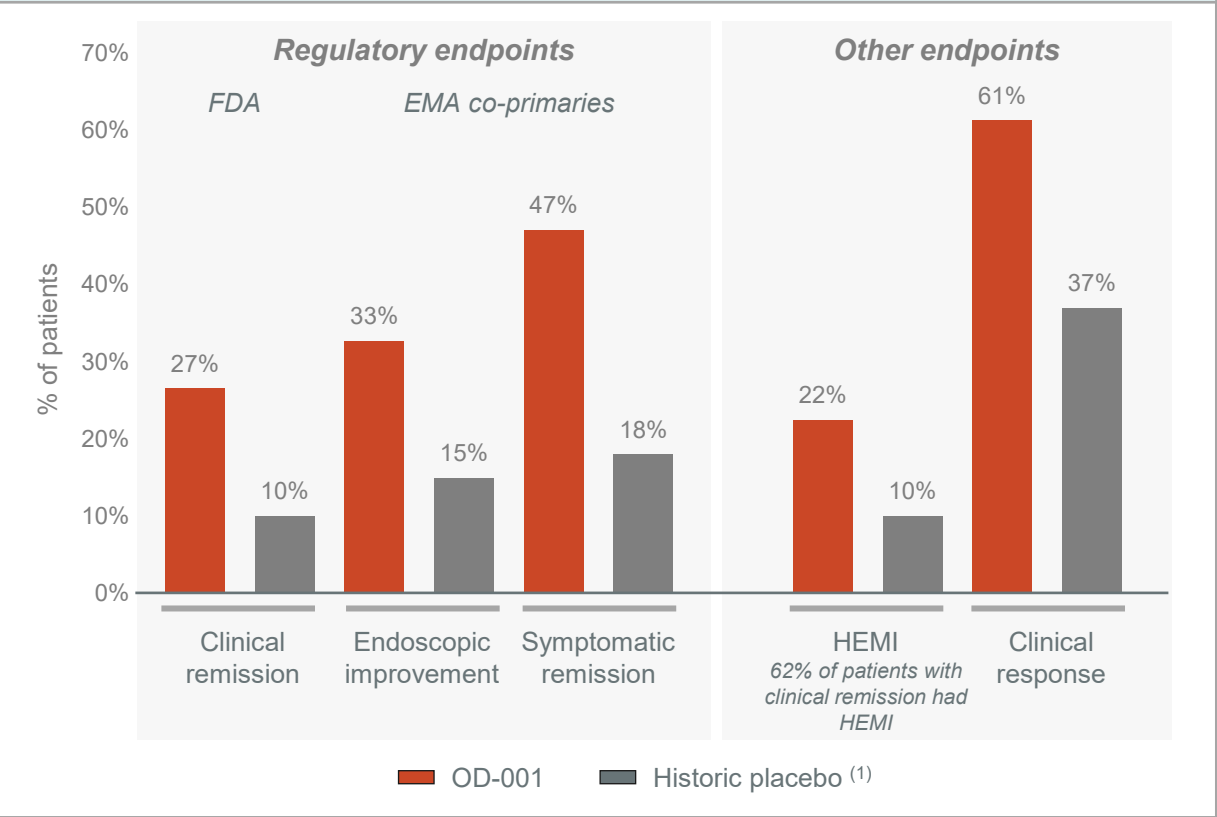
Disclaimer: Data presented are from ongoing studies, which will not be finalized until database lock. Thus, these data are subject to change prior to release of the clinical study report ("CSR").
Notes: (1) Three participants in part 2 discontinued treatment early (between weeks 1 and 3) for reasons unrelated to the trial drug. Total of 49 patients between part 1 and part 2 completed 12 weeks of induction treatment per protocol.
TEAE = treatment emergent adverse events; AE = adverse events; SAE = serious adverse events; hsCRP = high-sensitivity C-reactive protein.



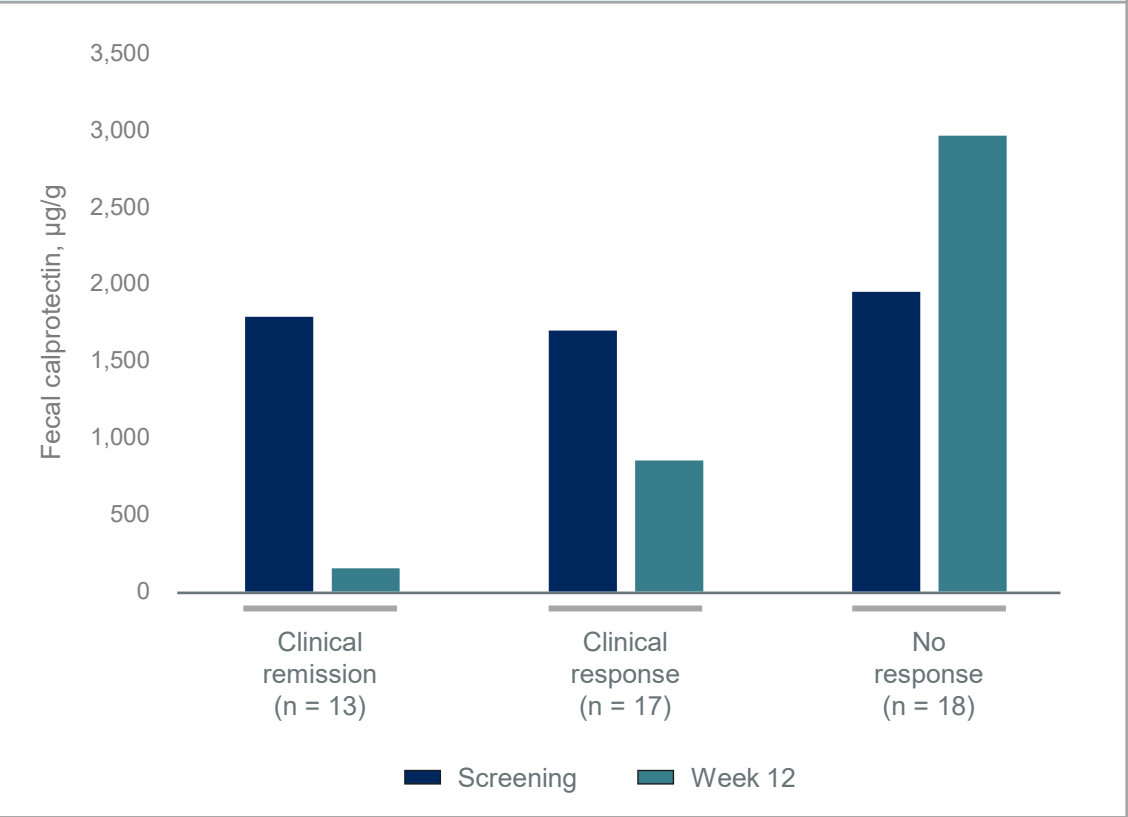
Trial Met Key Efficacy Endpoints, Including Clinical Remission

Met primary endpoint of change in MMCS from baseline in both parts and on a consolidated basis (-2.6, $p < 0.001$)

Efficacy across endpoints at week 12 (part 1 and 2 combined, n = 49)



Median fecal calprotectin by week 12 outcome

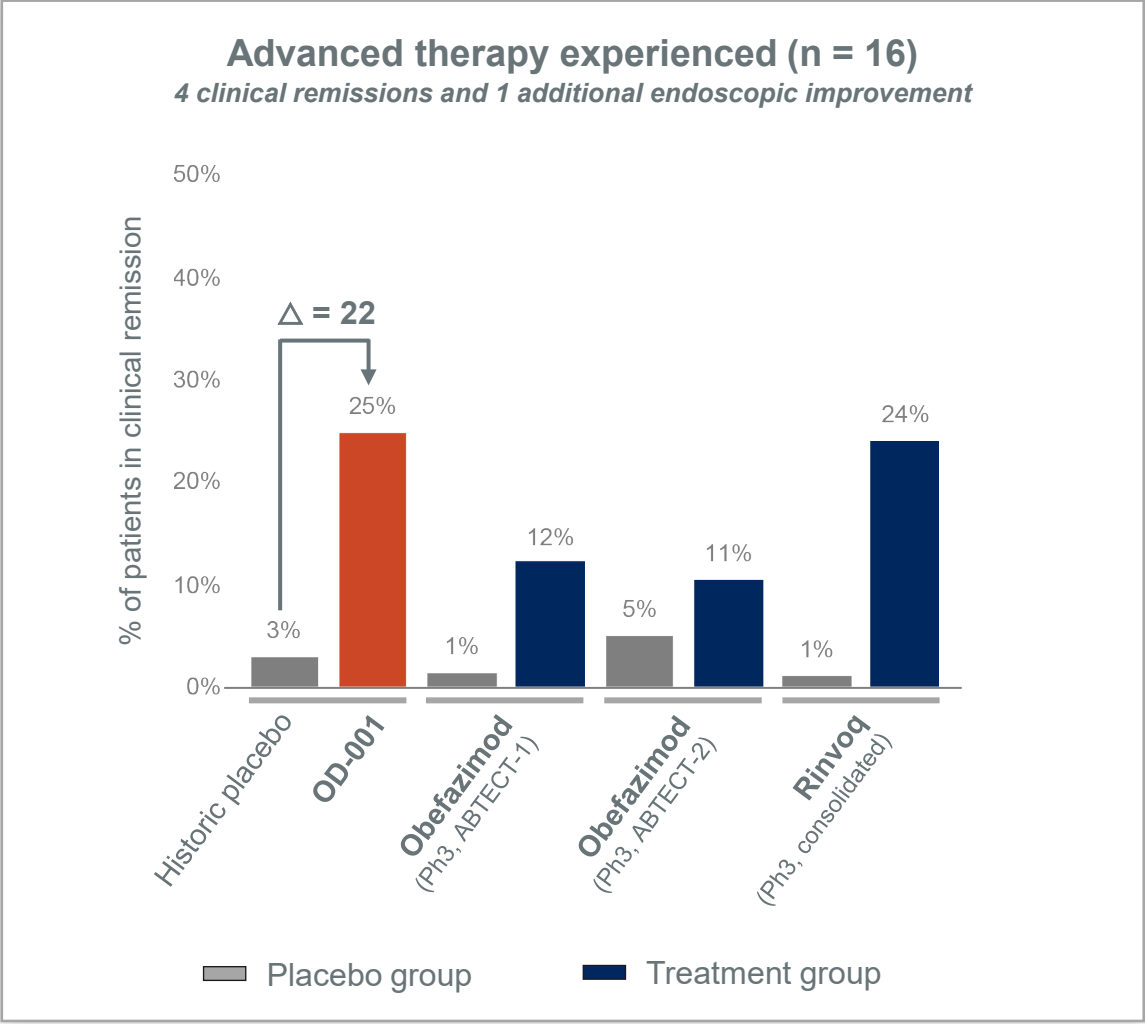
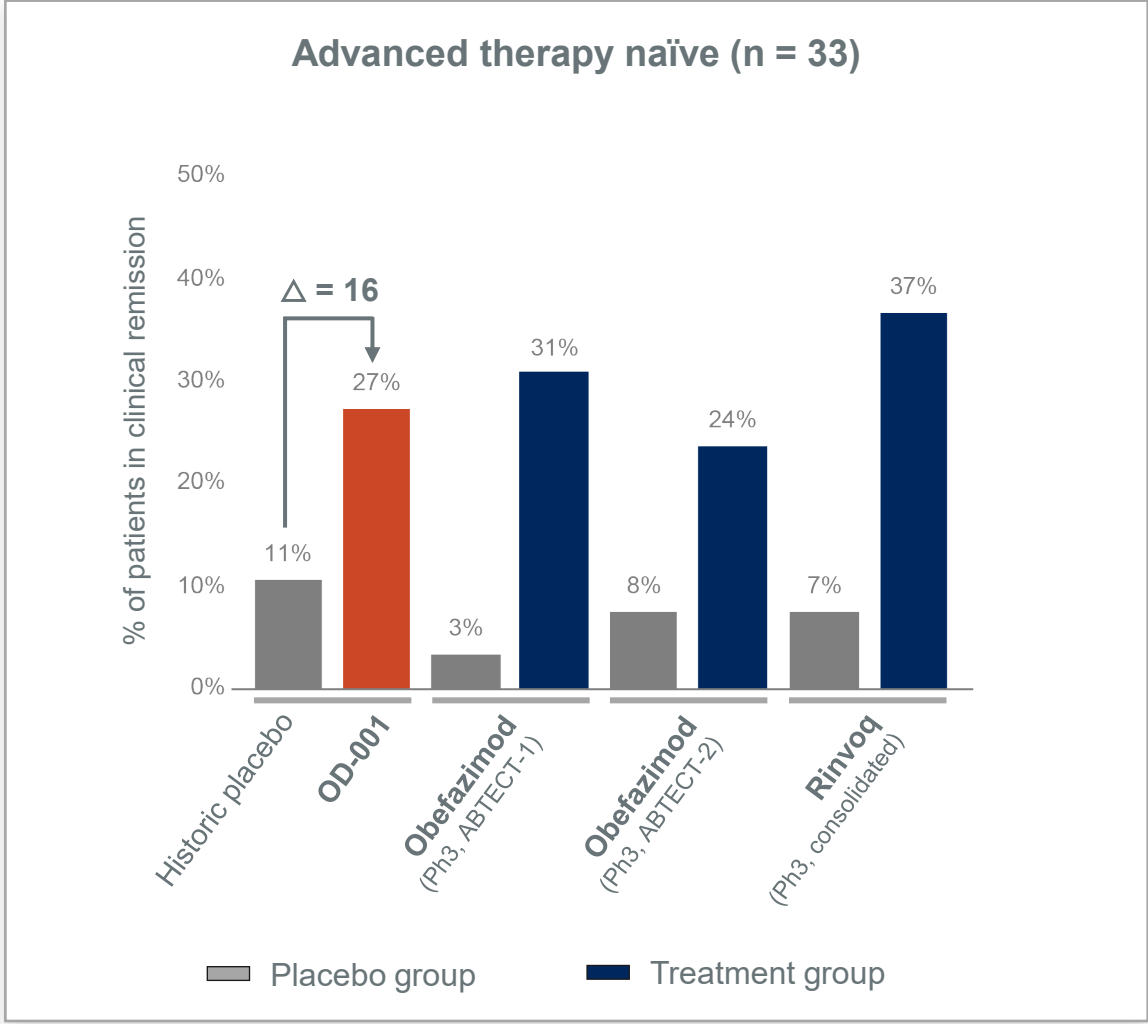


Disclaimer: Data presented are from ongoing studies, which will not be finalized until database lock. Thus, these data are subject to change prior to release of the CSR. Future results may differ from those shown here.

Notes: OD-001 results are consolidated from both part 1 and part 2 and represent all patients who completed 12 weeks of treatment per protocol (n = 49). HEMI = MES of 0 or 1 and Geboes index score ≤ 3.1 ; MMCS statistical assessment includes all 49 patients who completed treatment with required patient-reported outcomes. (1) Historic placebo benchmarks are derived from contemporary post-PoC UC studies (2020–2025); benchmark rates and supporting datasets are as follows: clinical remission 10% (average from 11 trials; n = 3,012 total; 820 placebo-treated patients); endoscopic improvement 15% (average from 10 trials; n = 2,920 total; 789 placebo-treated patients); symptomatic remission 18% (average from 5 trials; n = 1,872; 517 placebo-treated patients); HEMI 10% (average from 8 trials; n = 2,565 total; 676 placebo-treated patients); clinical response 37% (average from 9 trials; n = 2,633 total; 726 placebo-treated patients). Internal assessment of historic placebo rate is supported by third-party publications assessing placebo rates using individual patient data (J Crohn's Colitis 2025;19(10):jjaf191).



Clinical Remission Rates in AT-Experienced vs. AT-Naïve Patients

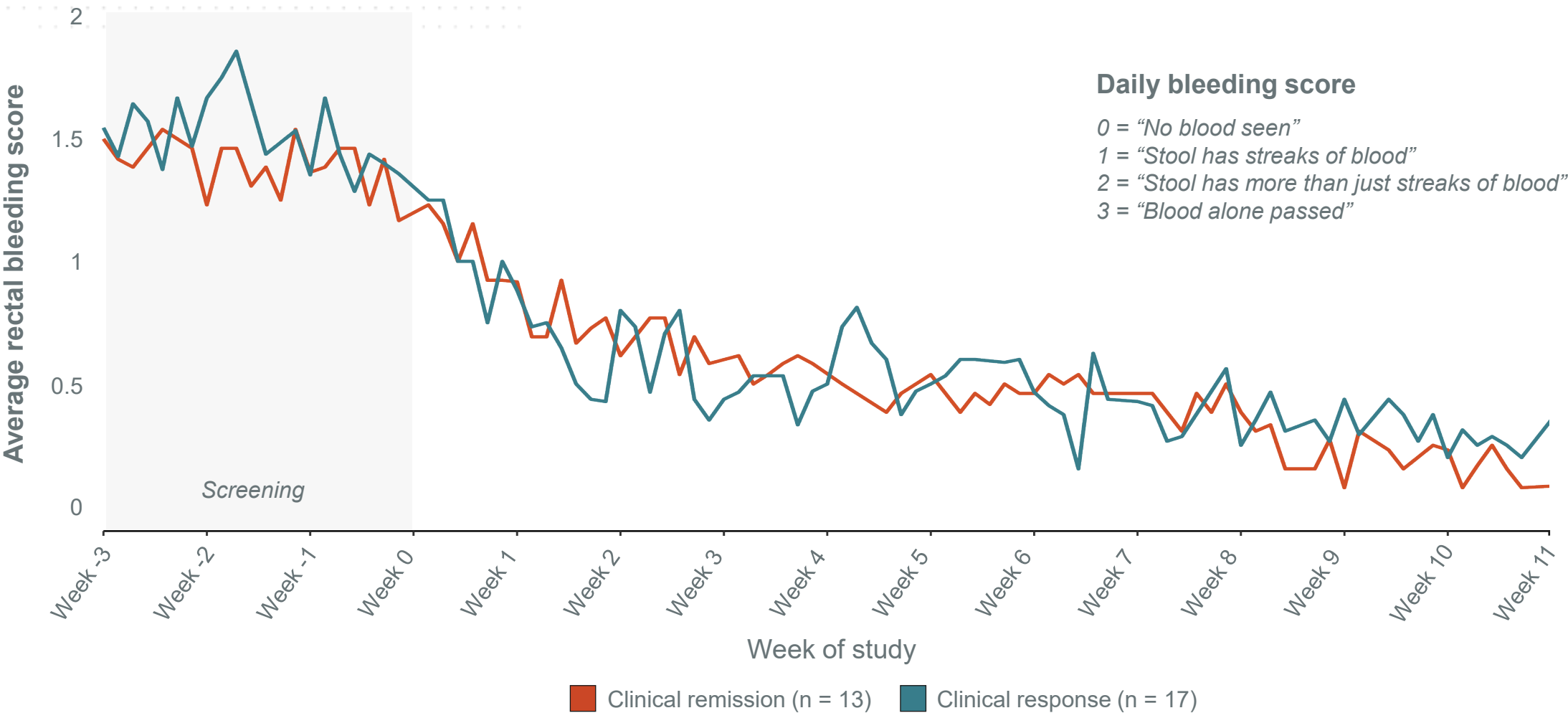


Disclaimer: Data presented are from ongoing trial, which will not be finalized until database lock. Thus, these data are subject to change prior to release of the CSR. Future results may differ from those shown here.
Notes: OD-001 results are consolidated from both part 1 and part 2. In trials where multiple doses were tested, results reflect a weighted average. Historic placebo benchmarks are derived from contemporary post-PoC UC studies (2020–2025) and Rinvoq phase 3 studies; dataset represents 1,819 AT-naïve and 1,475 AT-experienced patients treated with active agent or placebo. The results above do not represent head-to-head comparisons. OD-001 and a number of the comparators have not been approved and may never be approved by any regulatory authority.



Rapid Onset of Action With Deepening of Response, Consistent With MOA

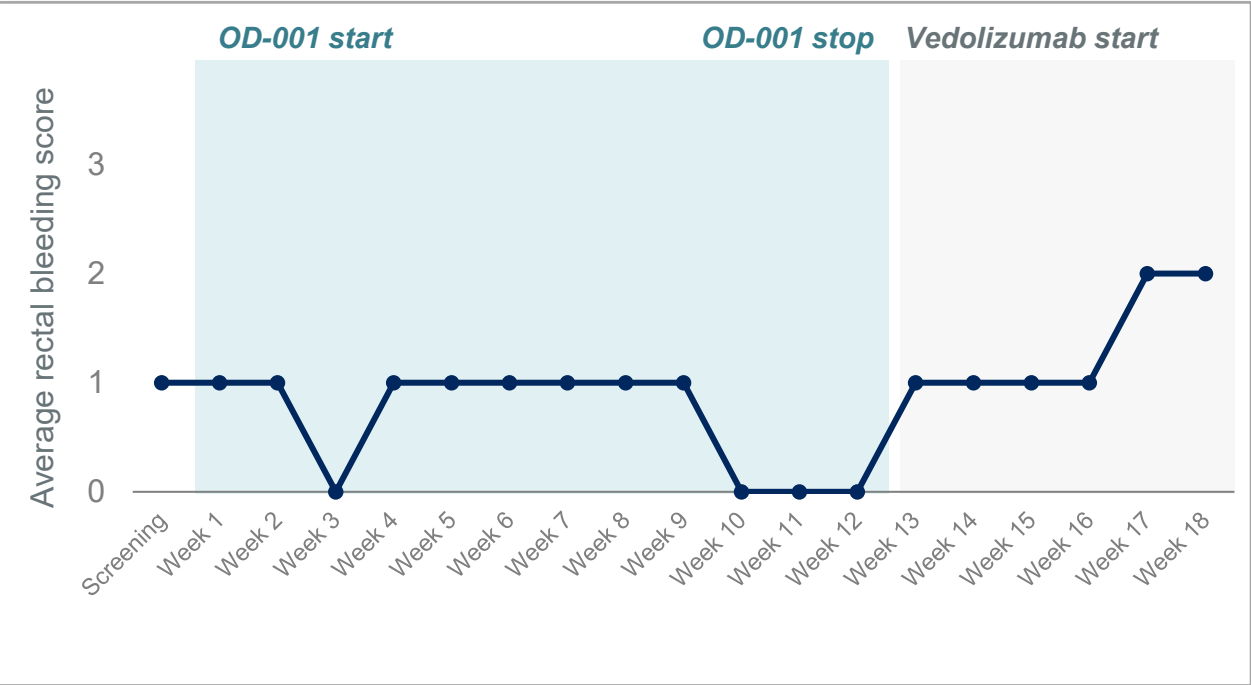
Average rectal bleeding score over treatment



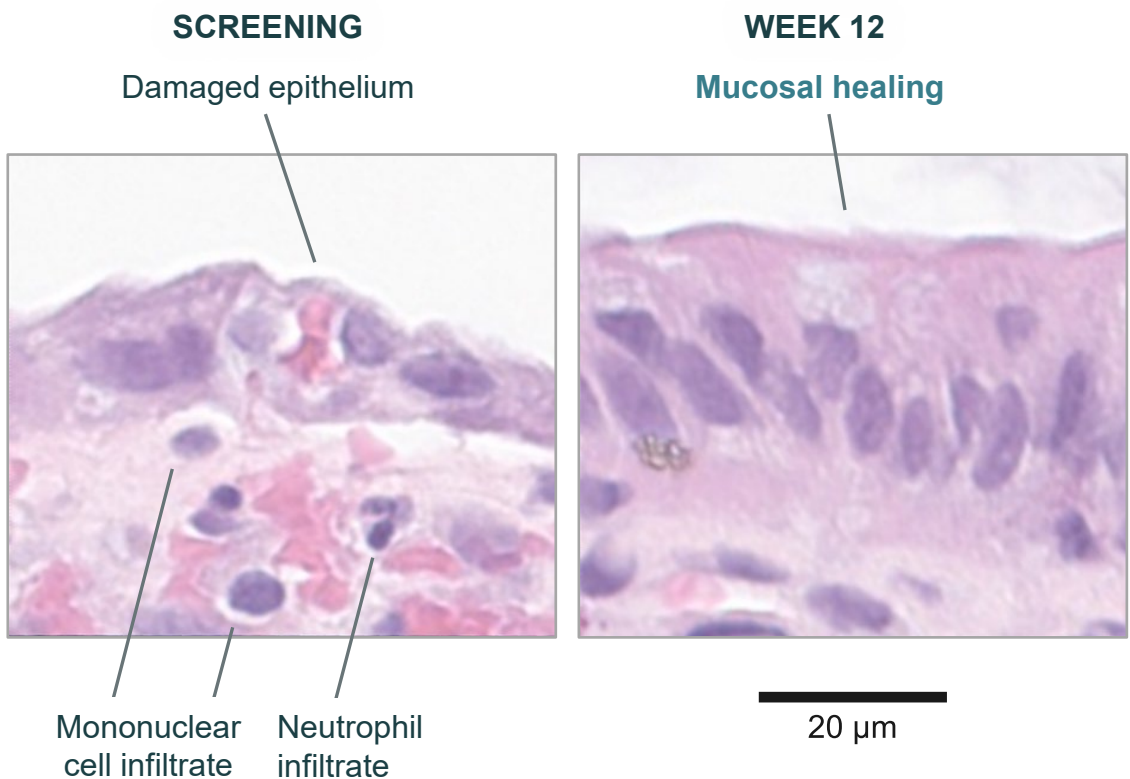
Disclaimer: Data presented are from ongoing studies, which will not be finalized until database lock. Thus, these data are subject to change prior to release of the CSR.
Note: OD-001 results are consolidated from both part 1 and part 2.

Patient Vignette: Clinical Remission With OD-001 but Relapse on Vedolizumab

- 55-year-old Canadian female with 5-year history of UC
 - Patient was previously treated with anti-IL-23 and S1P1
- MES of 2 and MMCS of 5 at screening
- **Achieved clinical remission and histologic endoscopic mucosal improvement (HEMI) at week 12; worsening after OD-001 discontinuation with vedolizumab discontinued at week 18 and Rinvoq started ⁽¹⁾**



Histopathology showed evidence of mucosal healing



Disclaimer: (1) While we believe the observations derived from this patient's results are informative, the results depicted are preliminary results from a single patient and may not be replicated in other patients or predictive of outcomes in clinical trials for OD-001. Average rectal bleeding score scale: 0 = "No blood seen", 1 = "Stool has streaks of blood", 2 = "Stool has more than just streaks of blood", 3 = "Blood alone passed."

OD-001 Is the First Innate Immune-Targeted Therapy for IBD

- Efficacy is competitive with leading approved or investigational therapies
 - Concordant efficacy measures across all endpoints, ***consistent with the mechanism of action for RIPK2***
 - Strong efficacy in AT-experienced patients
 - Innate immune cells (e.g., inflammatory monocytes) drive resistance to SoC therapies and these cells are targeted by OD-001
- Trial utilized contemporary best practices to minimize placebo effects
- Potentially indication leading safety profile
- Potential for broad combination use based on orthogonal mechanism of action to existing SoC
 - ***No RIPK2 is currently approved; therefore, use of OD-001 is not limited by patient's failure to other agents in the class***



Multiple Opportunities for OD-001 to Establish a Leading IBD Franchise

Current management

Conventional therapy

Orals: 5-ASA and methotrexate

Advanced therapy, including injectable biologics (e.g., TNF) and oral therapies (e.g., JAKi)

Orals



Injectables



Combinations



~\$3B
Monotherapy



OD-001 for conventional patients

Potential to address 300k+ patients not treated with AT due to their efficacy, safety, and oral profiles



OD-001 for advanced therapy patients

1st-line monotherapy for AT-naïve and for use in AT-experienced patients

\$4B+
Combination induction



OD-001 combinations



+ Vedolizumab ($\alpha 4\beta 7$)

+ Rinvoq (JAK)

First innate + adaptive combination therapy; has the potential to safely break therapeutic ceiling (> 40-50%)

\$7B+

Combination induction with monotherapy maintenance



Combination induction



OD-001 maintenance

Preferred therapy for induction and maintenance

Expansion of addressable population
in 2028 and beyond

Phase 2a vedolizumab combination
induction results expected in H2 2027

Phase 2b monotherapy induction expected
in H2 2027 and monotherapy maintenance
results expected in 2028



Oral SLC15A4 Inhibitor: OD-002

*Modulating B Cell Activity
and Interferon Signaling*

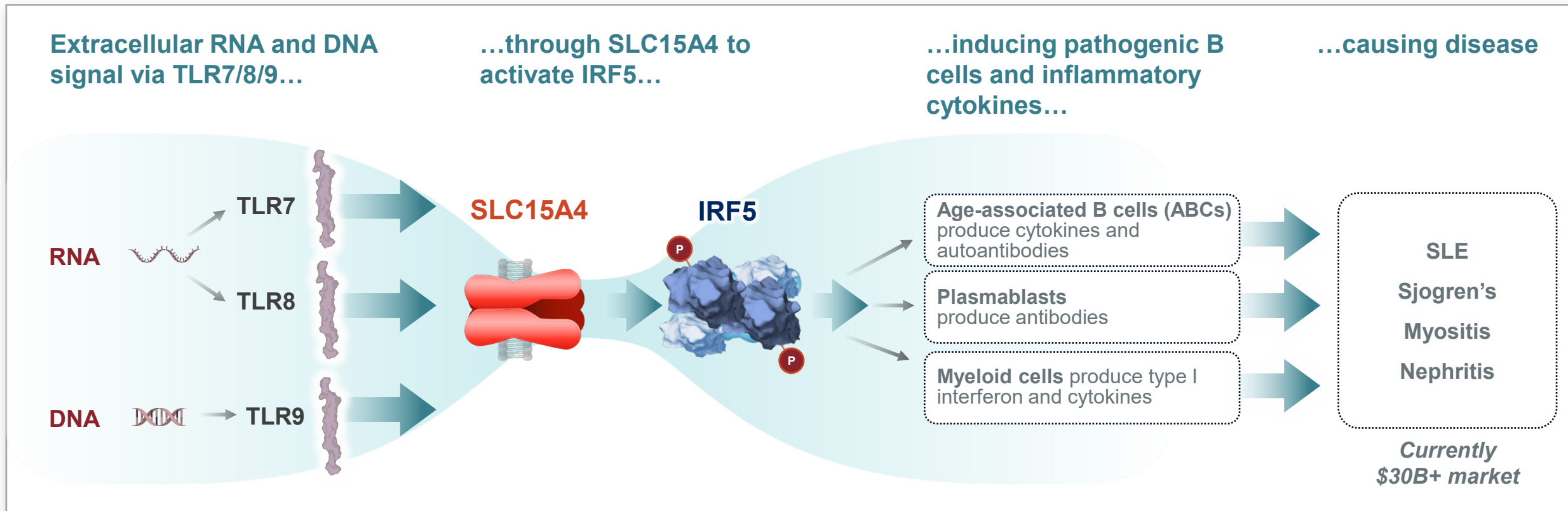
*CTA Filing in
H2 2026*

AI Platform Drove Program from Inception to GLP Study Initiation in 15 Months



Pathogenic Signaling Following TLR Activation is Mediated by SLC15A4

- Inflammatory responses to RNA or DNA are a root cause of many autoimmune diseases and a driver of pathogenic B cell expansion and autoantibody production



Source: Evaluate Pharma.

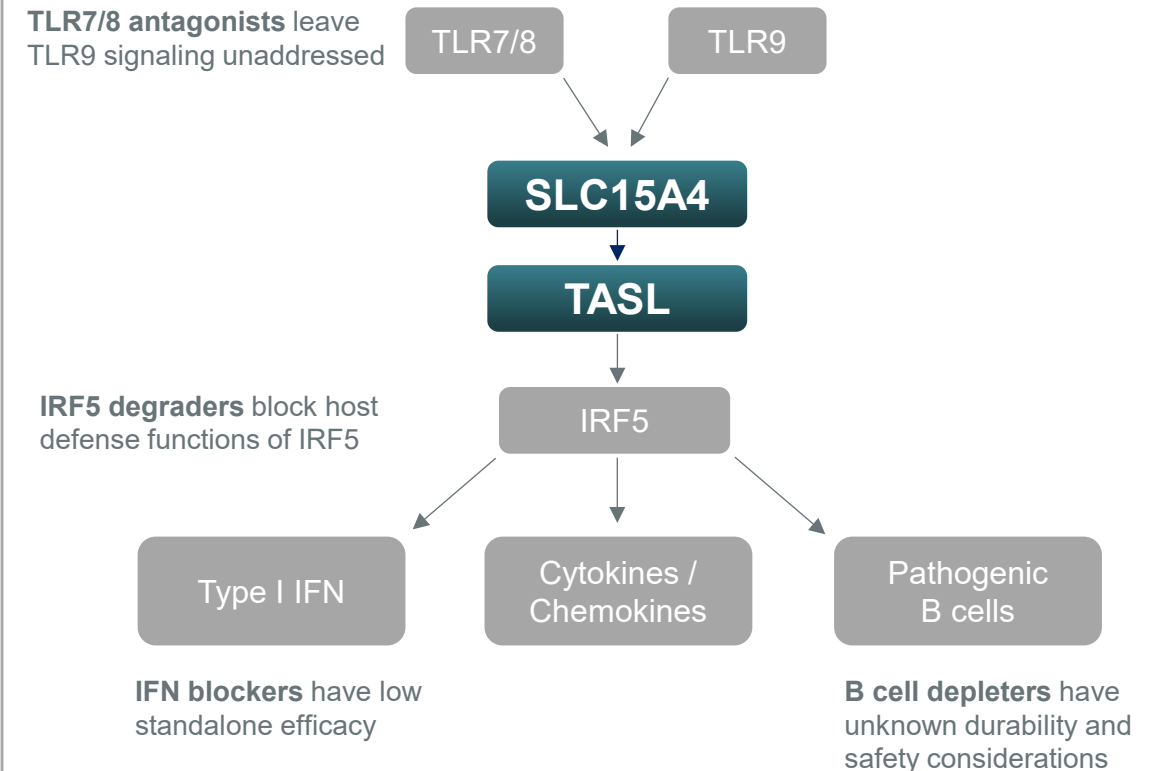
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In search of extraordinary for patients

We Believe SLC15A4 Is an Ideal Target in a Validated Pathway to Treat Diseases Driven by B Cells or Pathogenic RNA and DNA

- ✓ Commercial and clinical validation for targeting upstream or downstream components of the pathway
- ✓ Genetic polymorphisms in SLC15A4, and throughout the pathway, predispose to lupus and interferonopathies
- ✓ Targeting SLC15A4 or TASL is the only way to address TLR7/8/9 signaling while sparing IRF5 host defense
- ✓ SLC15A4 knockout mice are developmentally normal and protected across multiple lupus models
- ✓ SLC15A4 inhibition results in selective depletion of pathogenic B cells (e.g., ABCs, plasmablasts)
- ✓ SLC15A4 inhibition spares healthy B cells

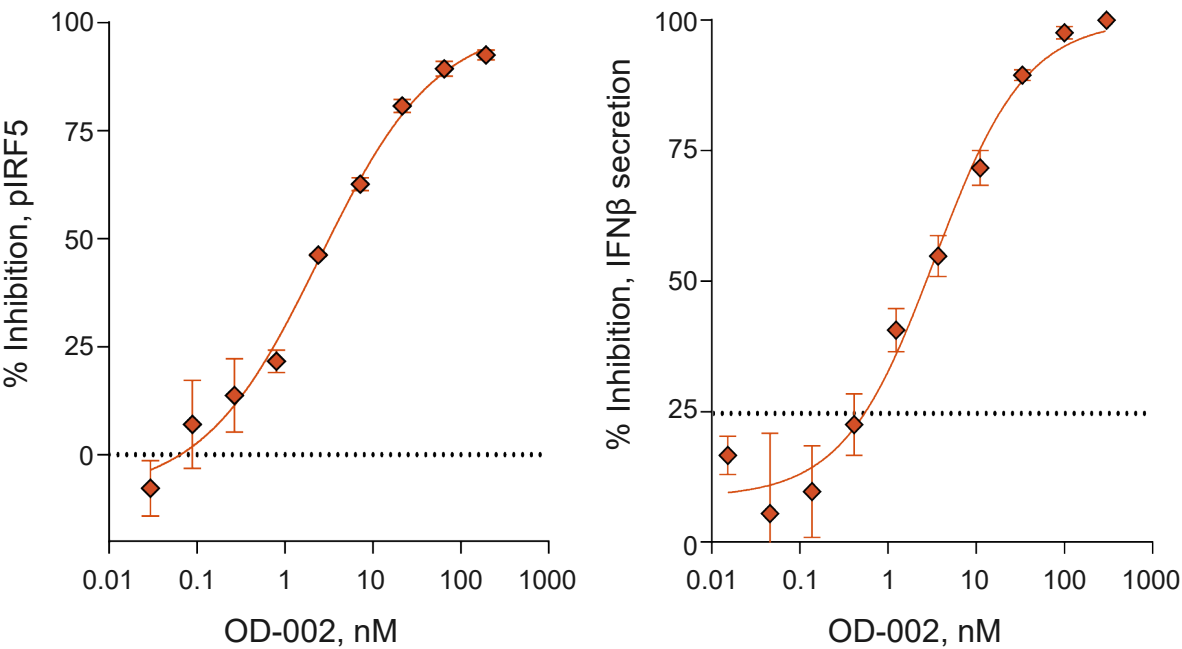
SLC15A4:TASL is the optimal node to maximize efficacy and tolerability



Sources: JEM 2025;13:2995; Immun 2006;25:429; Nat Immunol 2022;23:1457; PLOS ONE 2021;16:e0244439; PNAS 2013;11:2940; PNAS 2022;119:e2200544119; Nat Commun 2024;16:968; PNAS 2010;107:10154; PLOS One 2014;9:e103478; J Immunol 2010;184:796.

OD-002 Is a High-Quality SLC15A4 Development Candidate

OD-002 fully inhibits TLR7/8-dependent IRF5 phosphorylation and type I IFN production



IRF5 phosphorylation was measured in PBMC lysates by immunoassay following stimulation by TLR7/8 agonist, R848; IFNβ secretion was measured from human whole blood by immunoassay following stimulation by R848. In both experiments, cells from healthy human donors were pretreated with compound for 24 hours prior to stimulation.

OD-002 is potent and selective with favorable druglike properties

Potency in vitro

IC₅₀ in human primary cells (e.g., TNF, IFN)

≤ 10 nM

Potency in vivo

IFNα inhibition at 3 mg/kg; *phenocopies* SLC15A4 knockout

> 95%

Selectivity

TLR3, STING, and SLC15A1/2/3

> 1,000x

Projected once-daily human dose

Dose to maintain IC₉₀

18 mg

Preclinical safety and tolerability

High

Intellectual property

Composition of matter

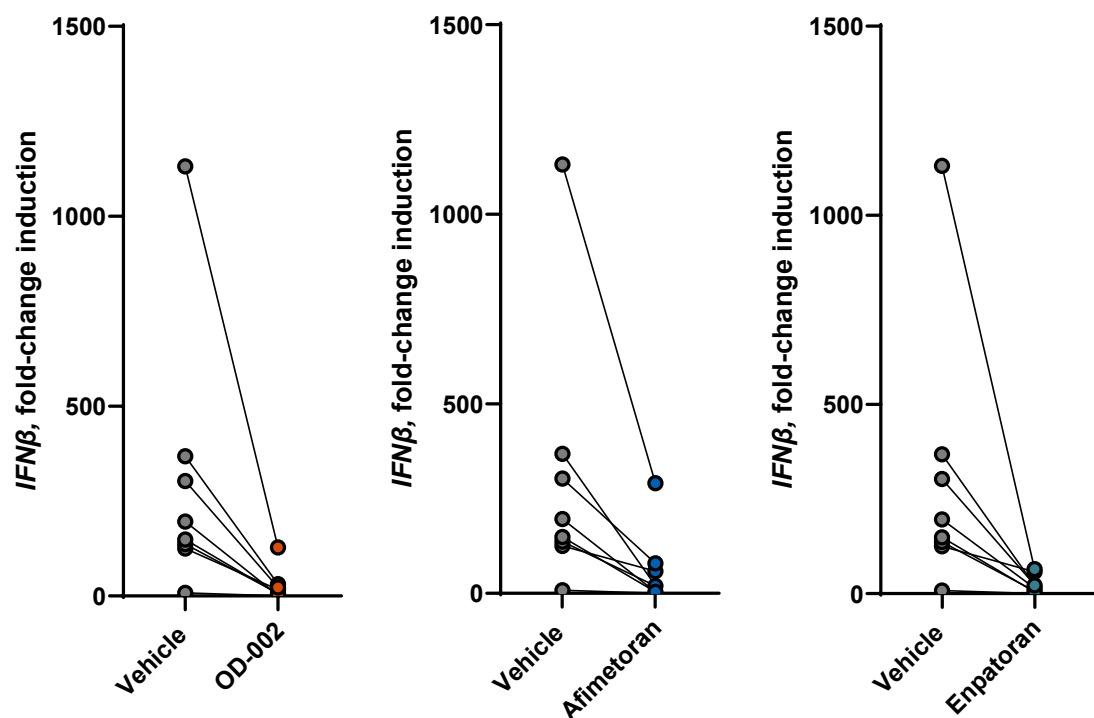
2046



OD-002 Blocks IFN Signaling From Both RNA and DNA

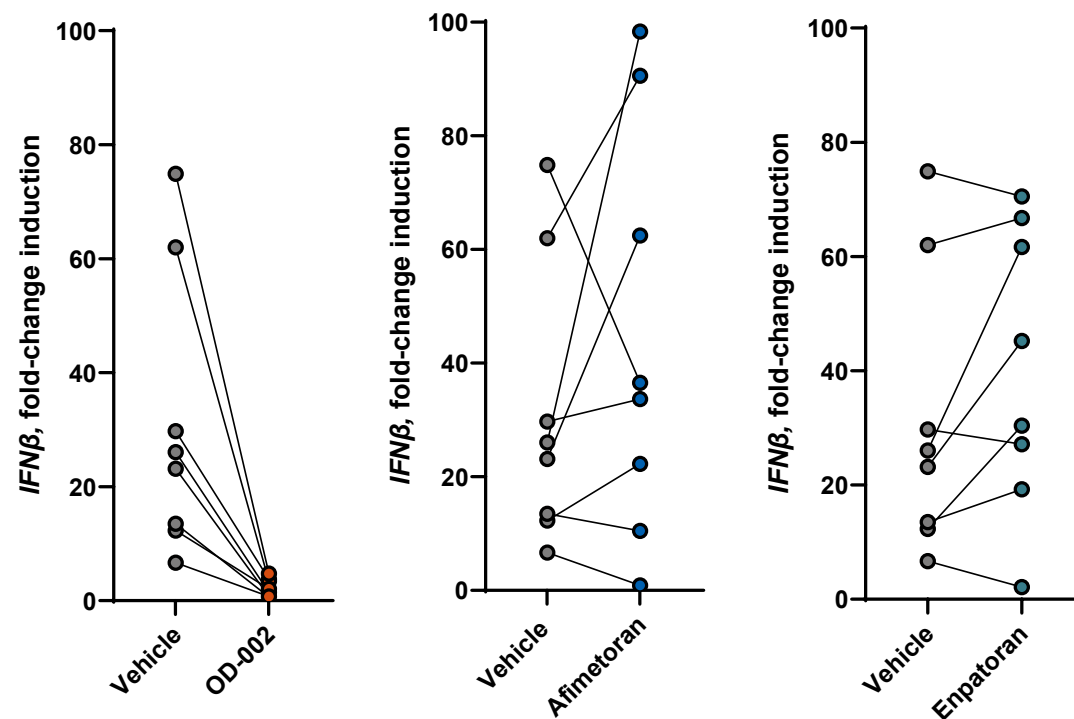
- TLR7/8 antagonists cannot block both arms of the nucleic acid response driving disease; with pathogenic signaling through TLR9 remaining active

SLC15A4 or TLR7/8 inhibitors block cytokines *initiated by extracellular RNA*



TLR7/8 stimulation induced by R848 in PBMC from healthy donors

SLC15A4 inhibition also blocks cytokine production *initiated by DNA*

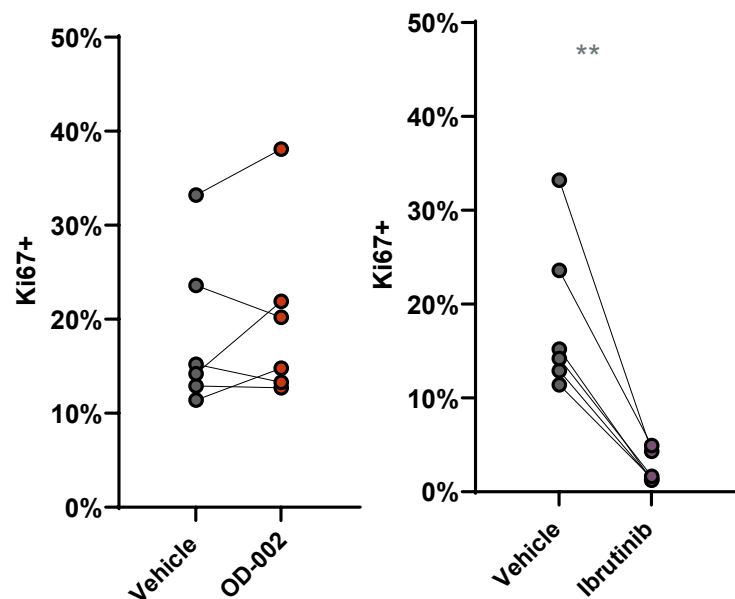


TLR9 stimulation induced by ODN-2395 in PBMC from healthy donors



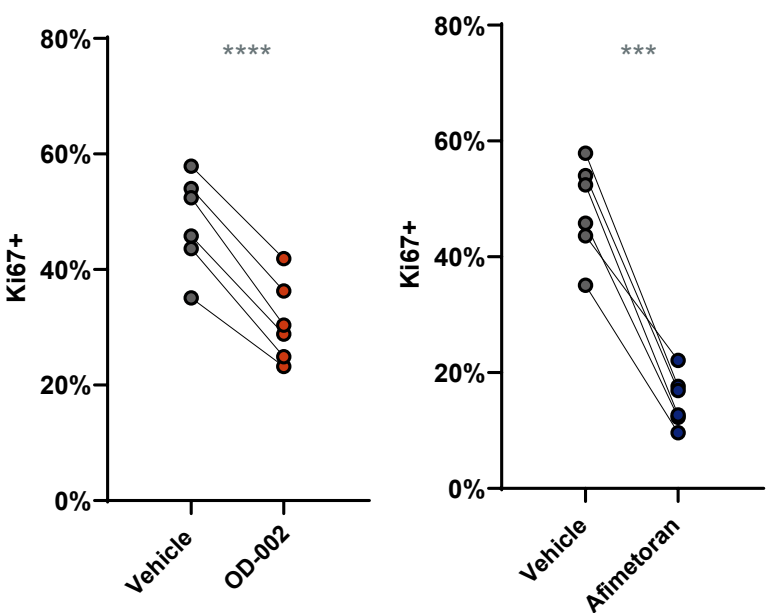
OD-002 Selectively Blocks Human B Cell Proliferation From Pathogenic Stimuli

SLC15A4 inhibition does not alter normal B cell proliferation by activation with CD40L



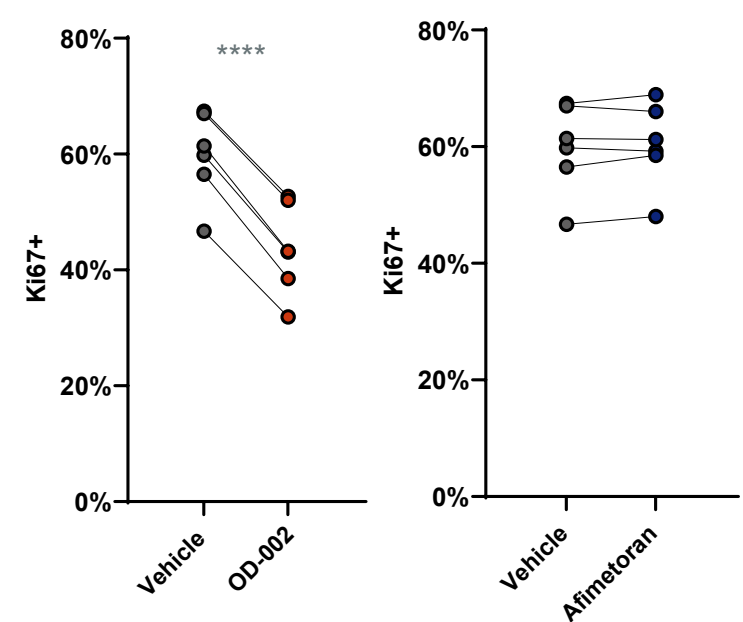
Ibrutinib, a BTK inhibitor, blocks non-pathogenic B cell proliferation

SLC15A4 inhibition blocks B cell proliferation induced by TLR7/8 activation



Afimetoran blocks TLR7/8-driven NF-κB signaling, in addition to IRF5

SLC15A4 inhibition also blocks B cell proliferation induced by CpG, a TLR9 agonist that mimics pathogenic stimuli



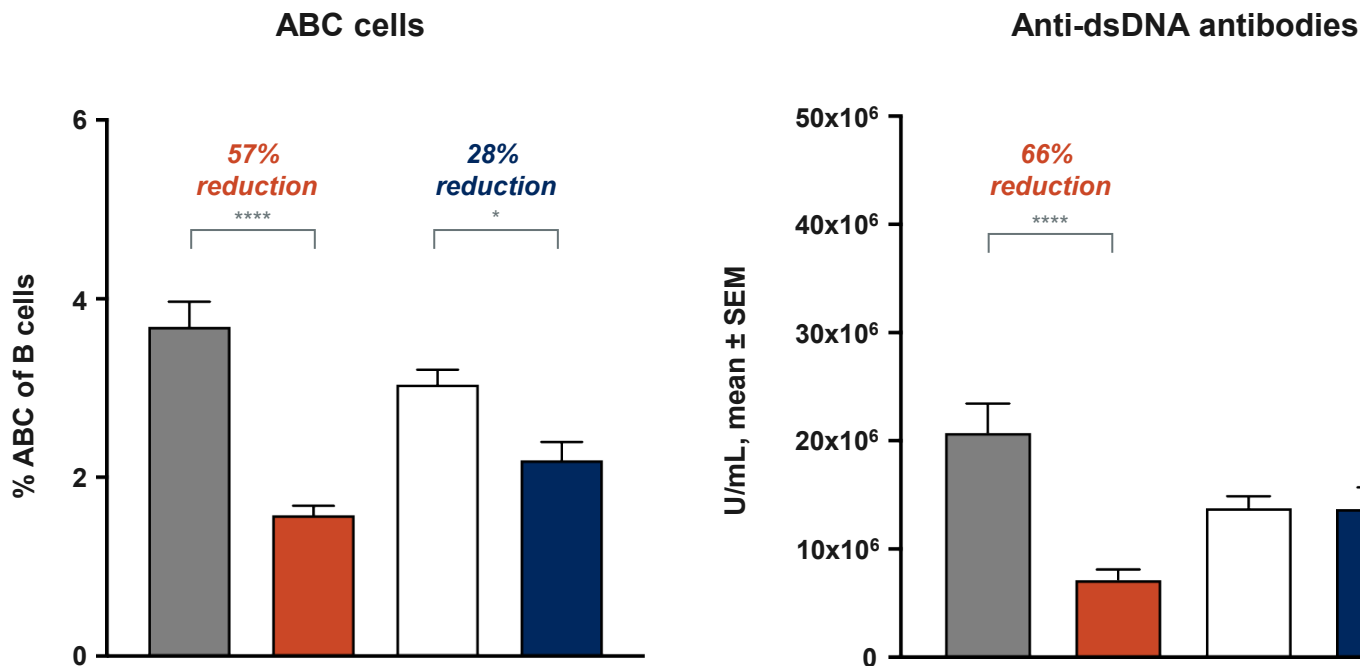
Afimetoran does not block TLR9-driven B cell proliferation



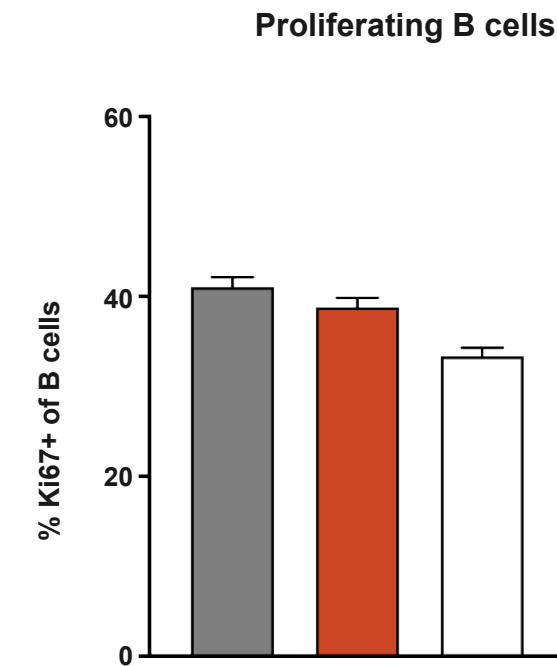
Note: Isolated memory B cells from healthy donors were pretreated with indicated compound for 24 hours prior to stimuli and % Ki67+ was measured after three days; ** p < 0.01; *** p < 0.001; **** p < 0.0001.

Blocking Signaling From Both RNA and DNA Is Necessary to Modulate Pathogenic B Cells *and* Autoantibody Production In Vivo

OD-002 reduced pathogenic B cells and autoantibody production in lupus-prone mice...



...while sparing B cells in healthy mice



8–9 week-old lupus-prone MRL/lpr mice (left) or C57BL/6 mice (right) were dosed for 4 weeks QD, and B cells (from spleen) and anti-dsDNA autoantibodies (from plasma) were analyzed; afimetroan is a TLR7/8 inhibitor currently in clinical development

● HPMC vehicle ● OD-002 ○ PBS/DMSO vehicle ● Afimetroan



Notes: * p < 0.05; **** p < 0.0001; HPMC = hydroxypropyl methylcellulose.

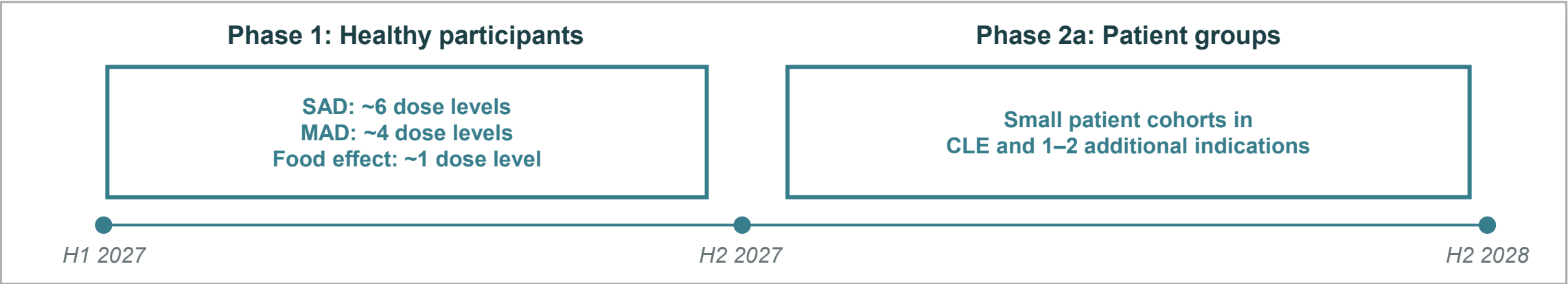
Phase 1/2a Trial Initiation Expected in H1 2027 With Efficacy Readouts Expected in H2 2028

Trial overview

- Phase 1: Randomized, double-blind, placebo-controlled SAD, MAD, and food effect cohorts in ~100 healthy participants
 - Ex vivo stimulation assay using R848 will be used to measure PK/PD relationship of type I IFN and pro-inflammatory cytokine inhibition
- Phase 2a: Plan to enroll cutaneous lupus erythematosus patients along with signal-seeking cohorts in other autoimmune diseases
 - CLE eligibility: Active CLE with CLASI-A score ≥ 8

Endpoints

- Phase 1: Healthy participants
 - Safety, tolerability, and pharmacokinetics
- Phase 2a: Basket trial in patient cohorts
 - Change from baseline in CLASI-A scores; additional endpoints to be defined with indication selection
 - Pharmacodynamic readouts may include cytokines, immune cell phenotyping, and gene expression



Note: CLASI-A = Cutaneous Lupus Erythematosus Disease Area and Severity Index score.

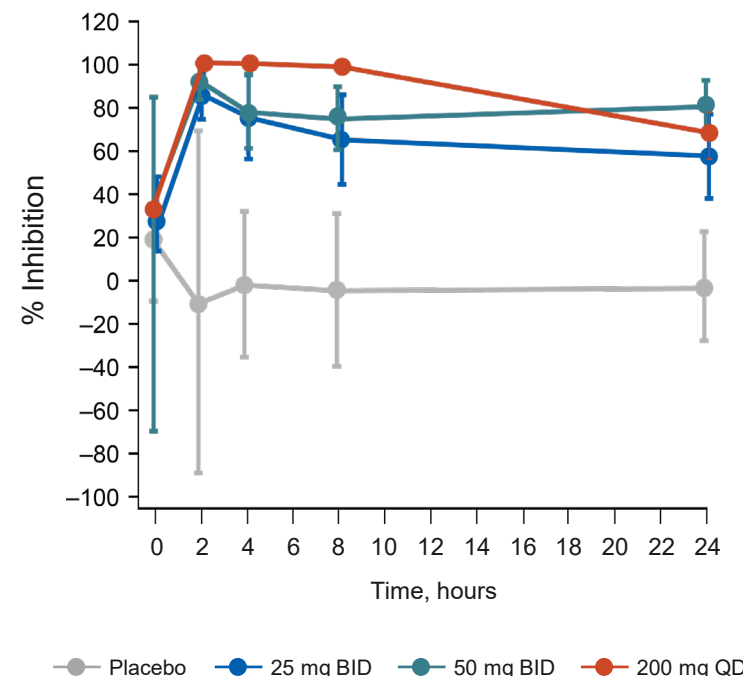
Phase 1 Ex Vivo Stimulation Assays Correlate With Activity in Lupus Patients

TLR7/8 antagonists have validated ex vivo assays for predicting activity in lupus patients

Sponsor, program	Status	Completed ex-vivo stim assay?	Activity in SLE or CLE patients?
 Enpatoran	Ph3 initiated March/April 2026	✓	✓
 Afimedoran	Ph2 completed June 2026	✓	✓
 E6742	Ph2 initiated March 2026	✓	✓
 MHV370	Discontinued for strategic reasons	✓	N/A

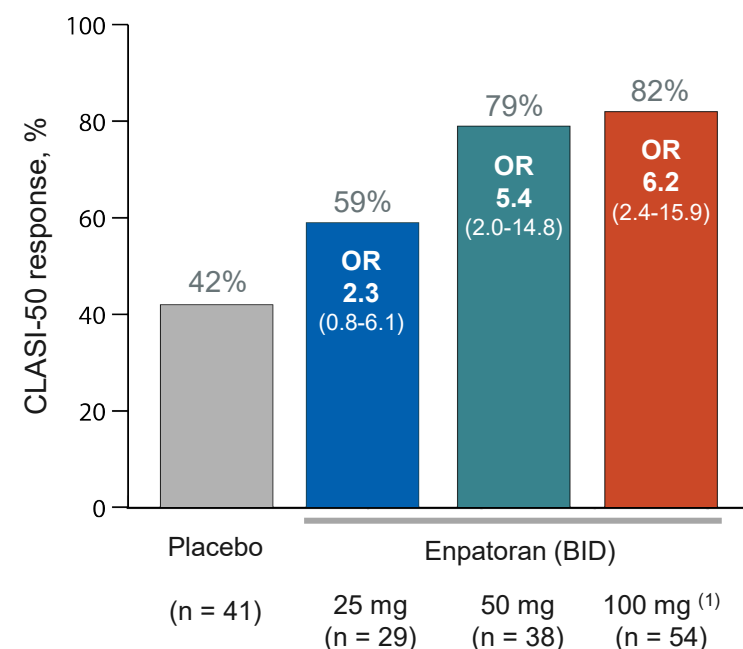
Enpatoran phase 1 ex vivo stimulation assay

- Doses advanced into phase 2 trials achieved > 60% IFN α inhibition



Efficacy across enpatoran doses in phase 2

- Positive phase 2 results based on CLASI-50
- Two phase 3 trials initiated in March/April 2026



Sources: Pharmacol Res Perspect 2021;9:e00842; Lancet 2026;407:1809; ACR Open Rheumatol 2025;7(7):e70059; RMD Open 2024;10:e004701.
Note: CLASI = cutaneous lupus erythematosus disease area and severity index; (1) phase 1 evaluated 200 mg QD and phase 2 used 100 mg BID.

TNFR2 Agonist: OD-003

*Modulating Treg to Treat Autoimmune
and Inflammatory Disease*

*IND-Enabling
Studies Ongoing*



Treg Therapy Has Broad Therapeutic Potential in Large I&I Diseases

Atopic dermatitis

SLE

Alopecia areata

Vitiligo

Multiple sclerosis

Rheumatoid arthritis

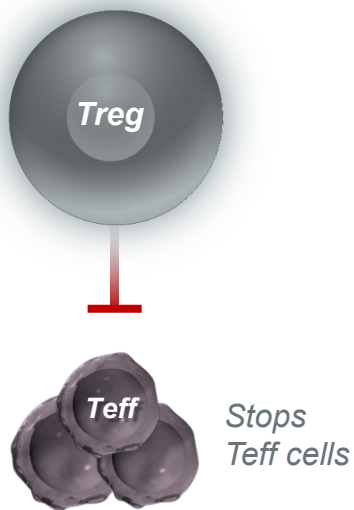
Asthma

Correcting Treg dysregulation or increasing the number of Treg has the potential to be used across many inflammatory and autoimmune diseases

EFFECT 1:

Reduction in Teff cell proliferation and activity

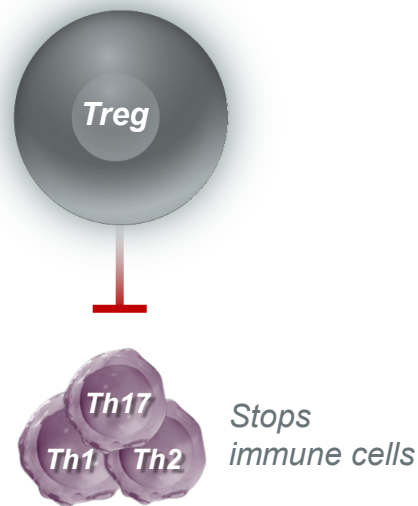
Reduces direct tissue damage and immune activation



EFFECT 2:

Reduction of immune cell activity

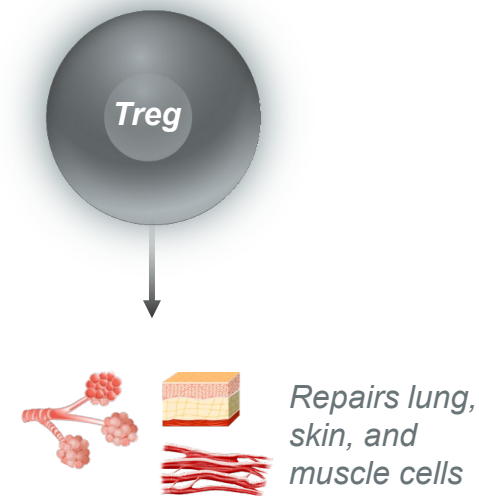
Reduces production of pro-inflammatory cytokines, including IFN γ , IL-4, IL-5, and IL-13



EFFECT 3:

Repair of tissue-resident non-immune cells

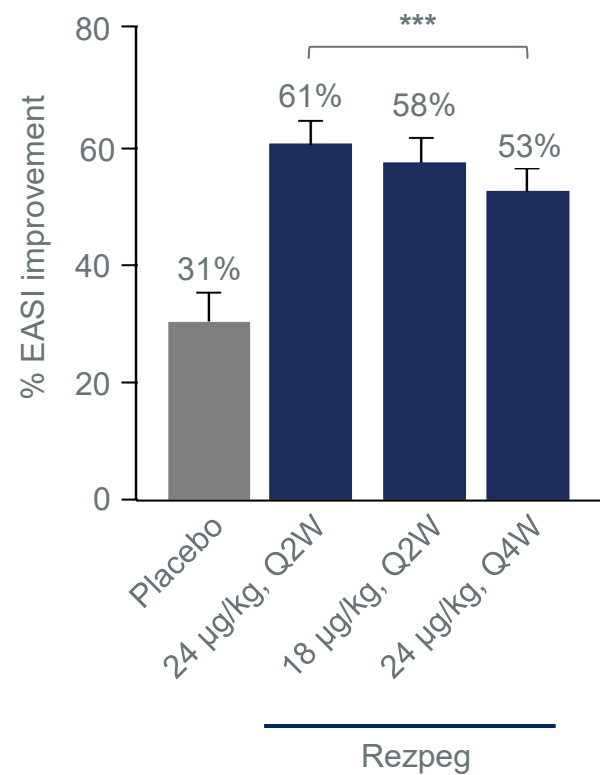
Induces durable disease-modifying effects



TNFR2 Agonism Is Next Evolution of Treg-Targeted Therapy

Expanding Treg number observed to be effective in multiple diseases

- Efficacy observed in atopic dermatitis (shown below), alopecia areata, and asthma



TNFR2 agonism can potentially address the shortcomings of IL-2

Criteria	IL-2	TNFR2
Expand Treg number	✓	✓
Enhance Treg immunosuppressive function and homing markers	✗	✓
Induce stable Treg phenotype	✗	✓
Initiate tissue repair program	✗	✓

TNFR2 agonism clinical PoC upcoming to unlock next stage of Treg therapy development



TRB-061:
Phase 1b AD efficacy data expected in mid 2027



NKTR-1065:
IND submission expected in 2027



OD-003:
IND-enabling studies ongoing



Note: *** $p < 0.001$.
Sources: Nektar corporate presentation, January 2026; TRexBio press releases, April 2026 and January 2026; clinicaltrials.gov.

Treg Therapy Has Potential to Transform Some of the Largest I&I Indications

Stage of
validation

CLINICAL PoC

\$30B+
Dermatology

*AD, alopecia
areata, vitiligo*

- Rezpeg, IL-2 agonist, demonstrated efficacy in moderate-to-severe AD and alopecia areata

»

CLINICAL SIGNAL

~\$25B
Respiratory

Asthma

- Rezpeg-treated AD patients with self-reported asthma history showed improvement in asthma control questionnaire (ACQ-5) scores

»

TRANSLATIONAL EVIDENCE

\$20B+
Neurology

*Multiple
sclerosis*

- TNFR2 agonism reduces disease severity in EAE models (representative of human multiple sclerosis)

»

MECHANISTIC EVIDENCE

~\$25B
IBD

UC and CD

- Treg maintain intestinal immune tolerance and mucosal integrity; Treg dysregulation contributes to chronic inflammation

> \$100B addressable opportunity today across autoimmune and inflammatory diseases linked to Treg dysfunction



Source: Evaluate Pharma.

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Final Words



Significant Clinical Milestones Expected Through 2028

2026

- October: Oral presentation of full OD-001 Phase 2a induction data
- H2: Initiation of OD-001 phase 2b monotherapy trial and phase 2a vedolizumab combination trial in UC
- H2: File CTA for OD-002 (SLC15A4)

2027

- H1: Initiate OD-002 healthy participant dosing in Ph1/2 trial
- H2: Complete OD-002 healthy participant dosing in Ph1/2 trial
- H2: OD-001 phase 2a vedolizumab combination induction readout
- H2: OD-001 phase 2b monotherapy induction readout

2028

- H2: OD-002 phase 2a readouts in multiple indications
- H2: First OD-001 maintenance readout





ODYSSEY
THERAPEUTICS

Thank you!

SLEEPER ST

SEAPORT