ODYSSEY THERAPEUTICS

Corporate Presentation

June 2026

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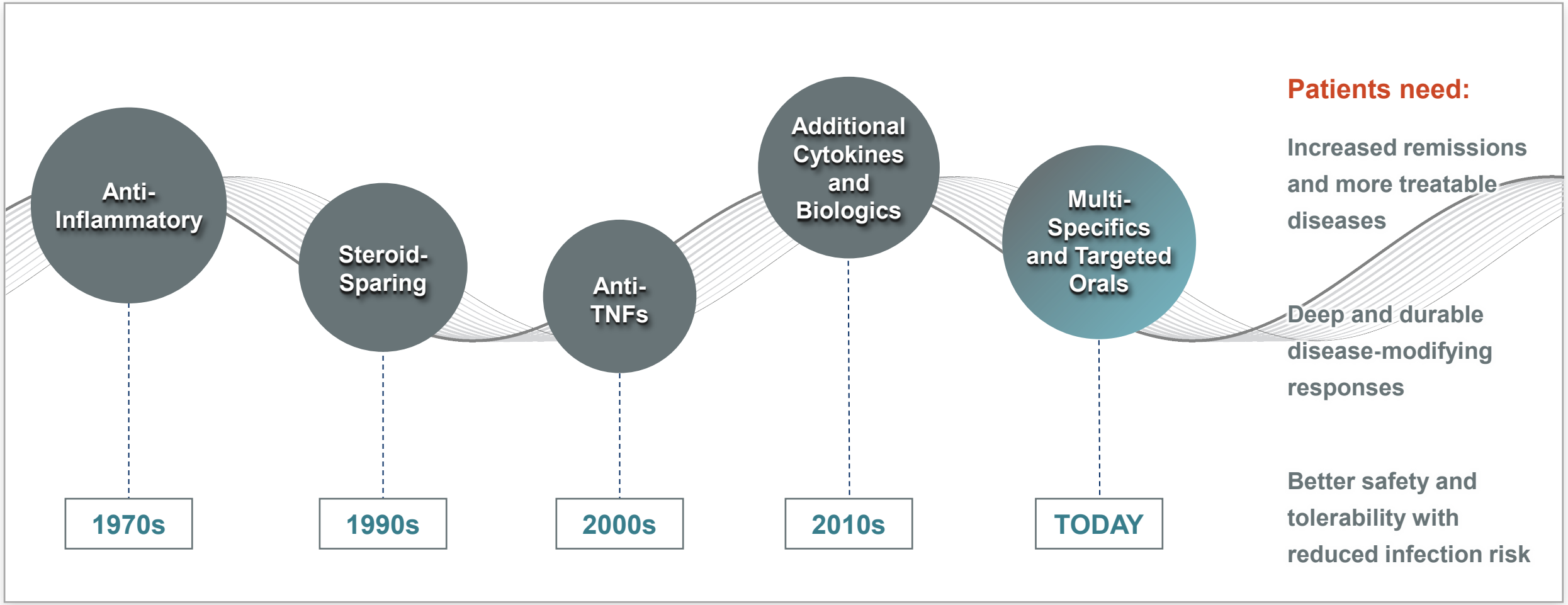
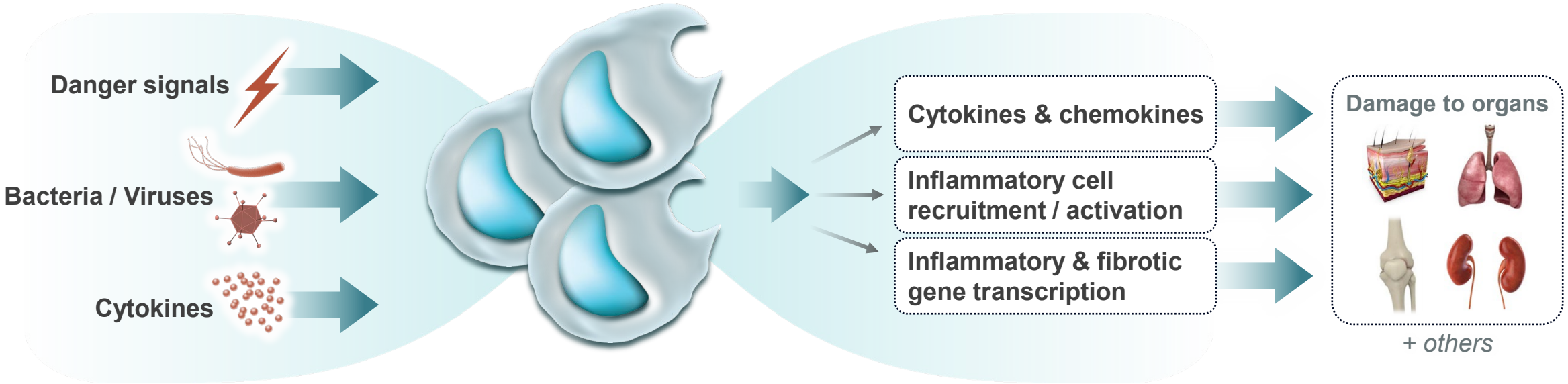
Despite Decades of New Therapies, Few Patients With Autoimmune Disease Have Durable Remission



Unlocking Durable Remissions by Targeting the Upstream Root of Disease

Inflammatory diseases are driven by multiple cytokines so targeting any one of them has limited benefit



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 home-grown 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 $464M as of March 31, 2026 (1)

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(1) Pro-forma for net proceeds from IPO and concurrent private placement.

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

OD-001 *(RIPK2)*

- **+ Vedolizumab** *(α4β7)*
- **+ Rinvoq** *(JAK)*

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

B CELL-MEDIATED DISEASES

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

OD-002 *(SLC15A4)*

- **Pathogenic B cells**
- **Autoantibodies**
- **Interferon**
- **Cytokines/ Chemokines**

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

INFLAMMATORY AND AUTOIMMUNE DISEASES

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

OD-003 *(TNFR2)*

Regulatory T cells (Treg)

⊥

Effector T cells (Teff)

Current market: ***$100B***

Source: Evaluate Pharma.

Broad Therapeutic Pipeline for Diseases With Unmet Need

Target *Modality*	Indications	Discovery	IND-Enabling	Phase 1	Phase 2	Phase 3	Anticipated Upcoming Clinical Milestones
RIPK2, OD-001 *Small molecule*	*UC, CD*						**2H 2026:** Phase 2a monotherapy data update **2H 2027:** Phase 2a combination readout **2H 2027:** Phase 2b monotherapy readout
SLC15A4, OD-002 *Small molecule*	*CLE, nephropathies, and B cell-mediated diseases*						**2H 2026:** CTA filing **1H 2027:** Phase 1/2 initiation
TNFR2, OD-003 *Protein*	*AD, SLE, alopecia areata*						
TSLP/IL-33, OD-004 *Protein*	*Asthma, COPD*						

$464 million cash as of March 31, 2026, pro forma including net proceeds from IPO and concurrent private placement; expected runway into 2H 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.

Data to Build a Potential RIPK2 IBD Franchise Expected by 2028

Monotherapy 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**

Break therapeutic ceiling through innate-adaptive combinations for induction

2027

OD-001 *(RIPK2)*

- **+ Vedolizumab** *(α4β7)**
- **+ Rinvoq** *(JAK)*

Demonstrate potential of RIPK2-only 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 2043

Note: * Trial initiation expected in 2H 2026; IBD = inflammatory bowel disease; AT = advanced therapy.

Oral RIPK2 Scaffolding Inhibitor: OD-001

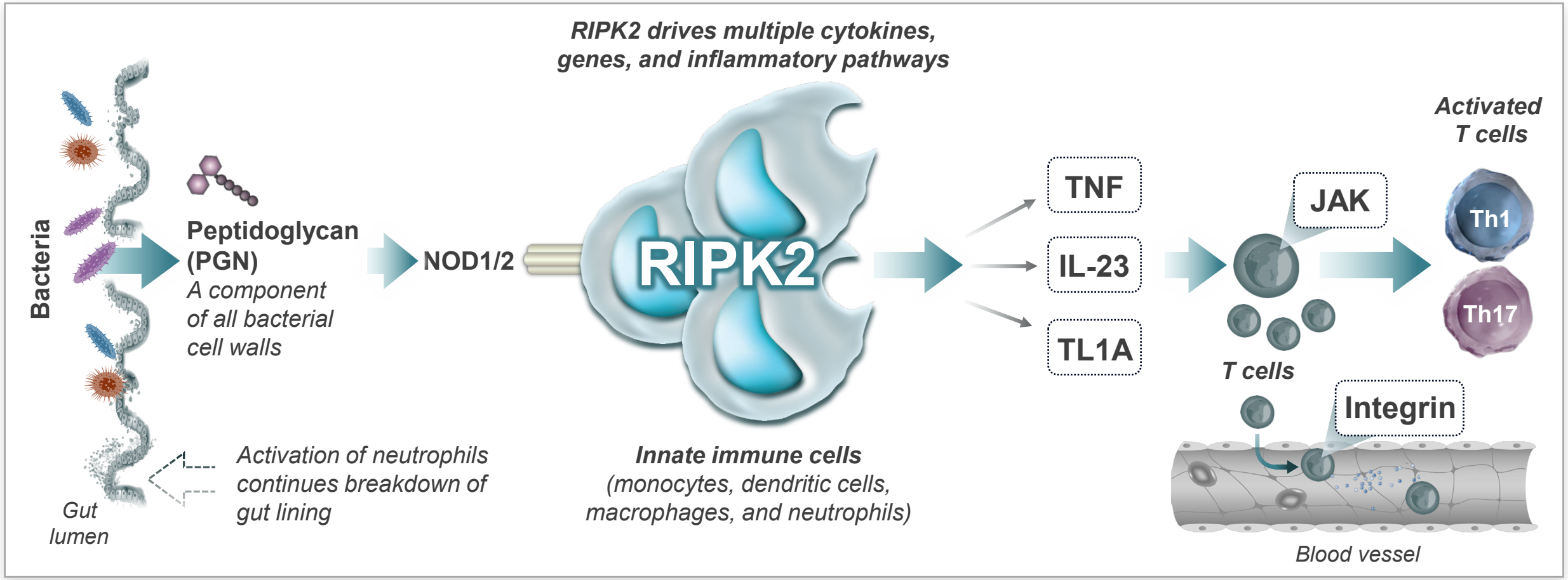
Addressing a Root Cause of Inflammation in IBD Using a Novel Mechanism of Action

Proof-of-Concept Achieved in Ulcerative Colitis

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

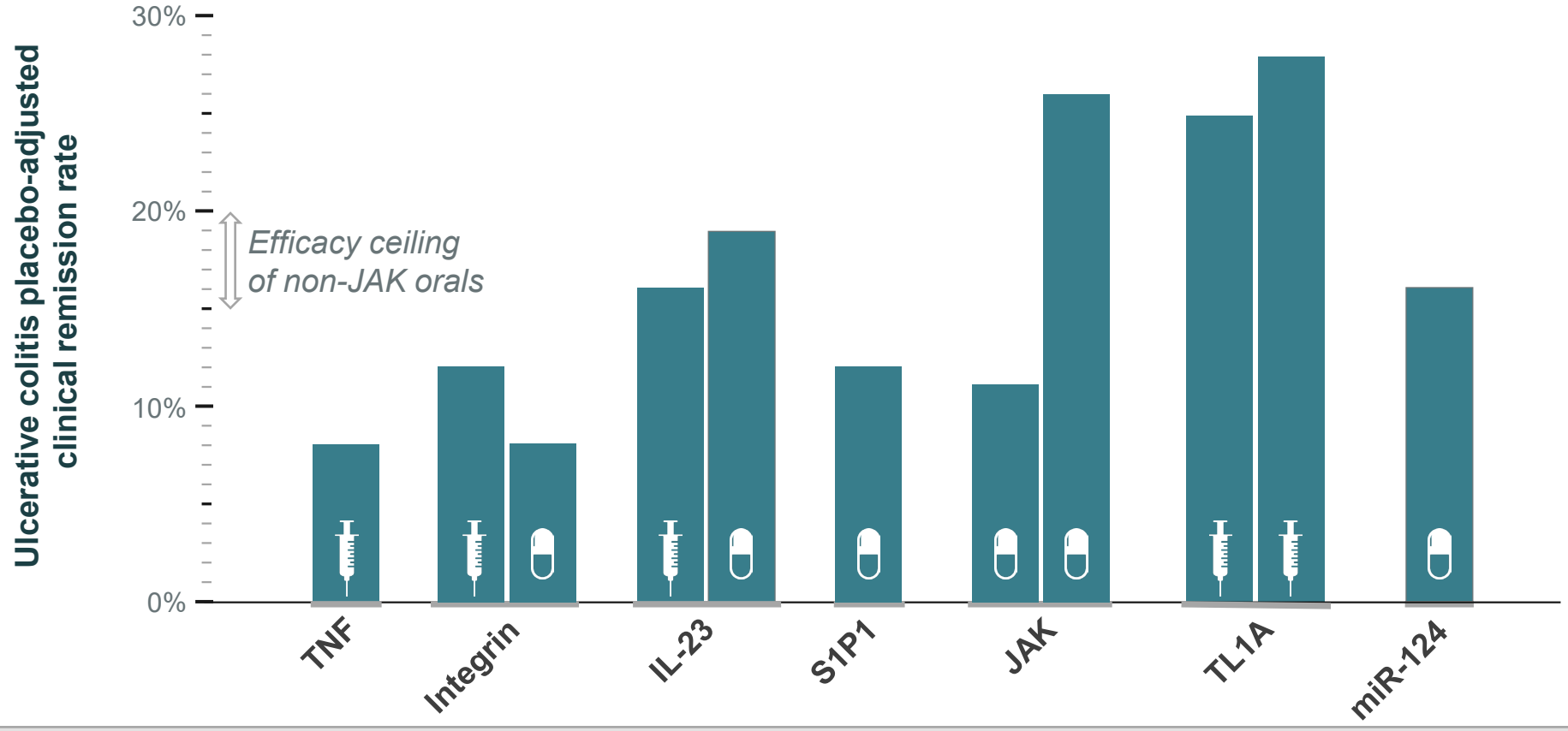
IBD Is Driven by Innate Immune Cells Responding to Gut Bacteria

- Breakdown of the gut lining and exposure of innate immune cells to bacteria are a root cause of IBD



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

Approved therapies targeting the adaptive immune system do not block the inflammatory response to bacteria driving disease and suffer a common resistance mechanism, innate immune activation



~75% of patients fail to achieve clinical remission

Remission rates are ~50% lower in AT-experienced patients

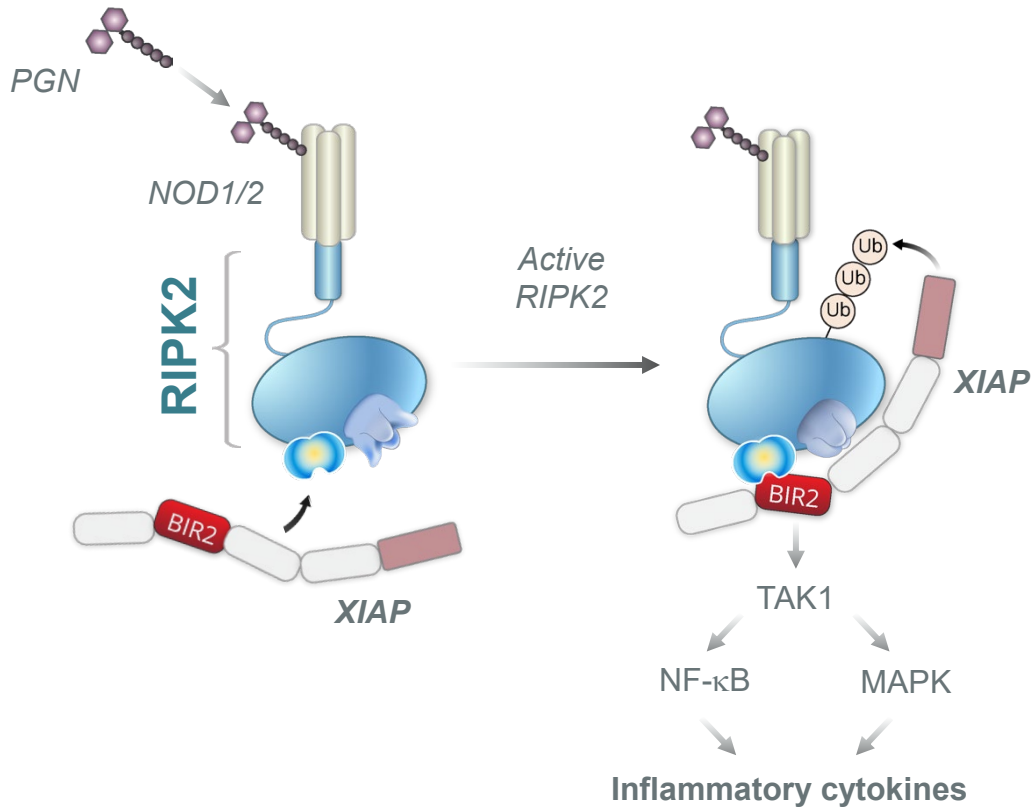
45% of patients who achieve remission progress within five years (1,2)

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 a Validated and 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
- RIPK2 knock-out mice were developmentally normal and protected from IBD
- Inhibiting RIPK2:XIAP scaffolding blocks multiple proinflammatory cytokines

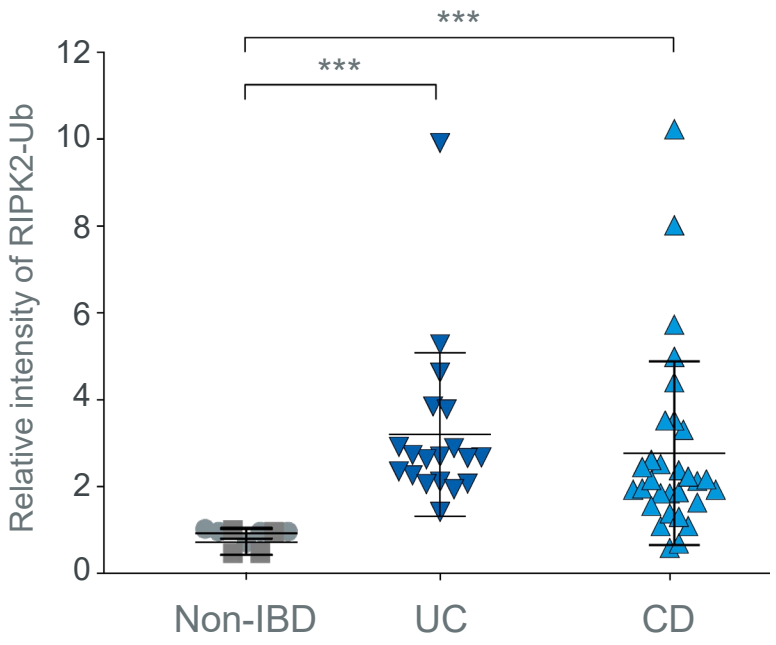
RIPK2 activation leads to scaffolding with XIAP and inflammatory signaling



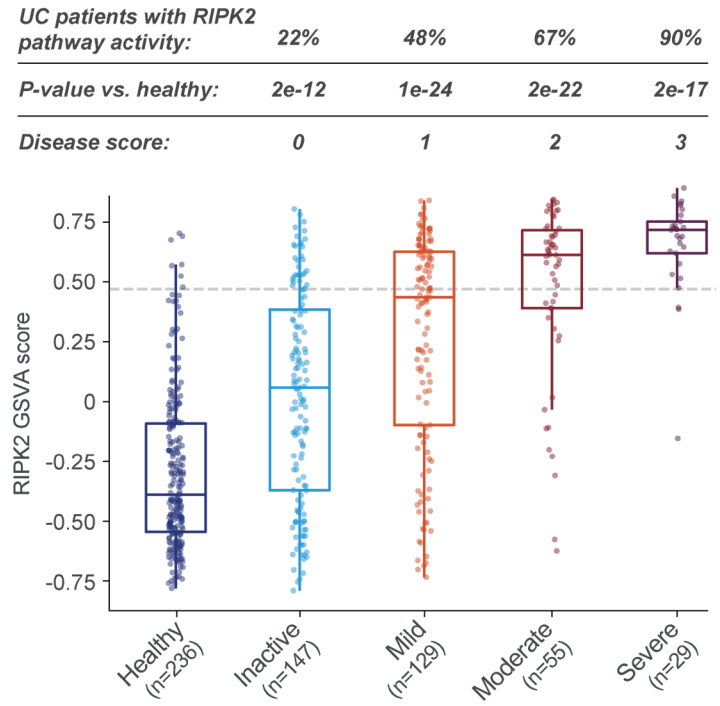
Sources: Inflamm Bowel Dis 2015;21(4):862; J Med Chem 2022;65(13):9312; J Med Chem 2019;62(14):6482; Proc Natl Acad Sci 2014;111(25):E2559; Am J Physiol 2021;321(5):G500; Commun Biol 2020;3(1):140; J Med Chem 2016;59(10):4867; Int Immunol 2019;31(10):669; United Eur Gastroenterol J 2023;12(1):103; Front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

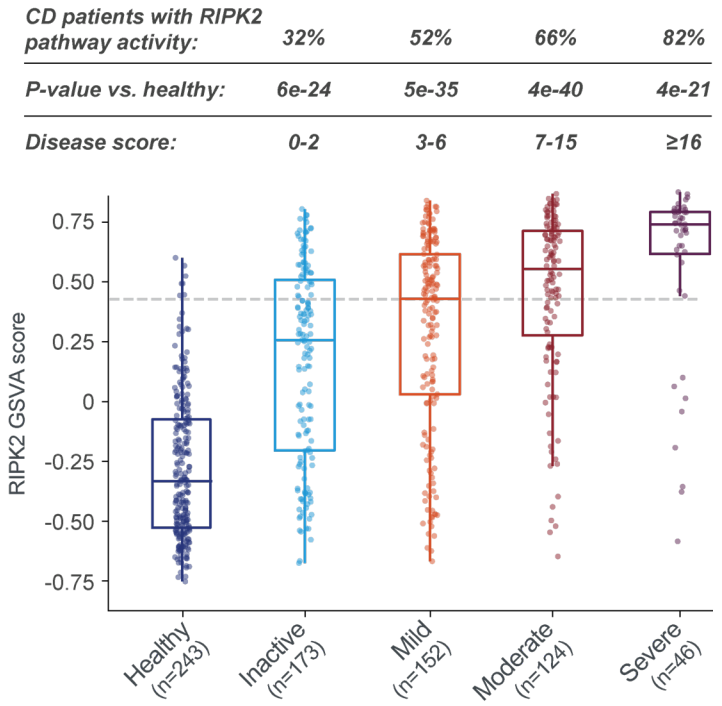
RIPK2 Is Activated in UC and Crohn's Disease and Correlates With Severity

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

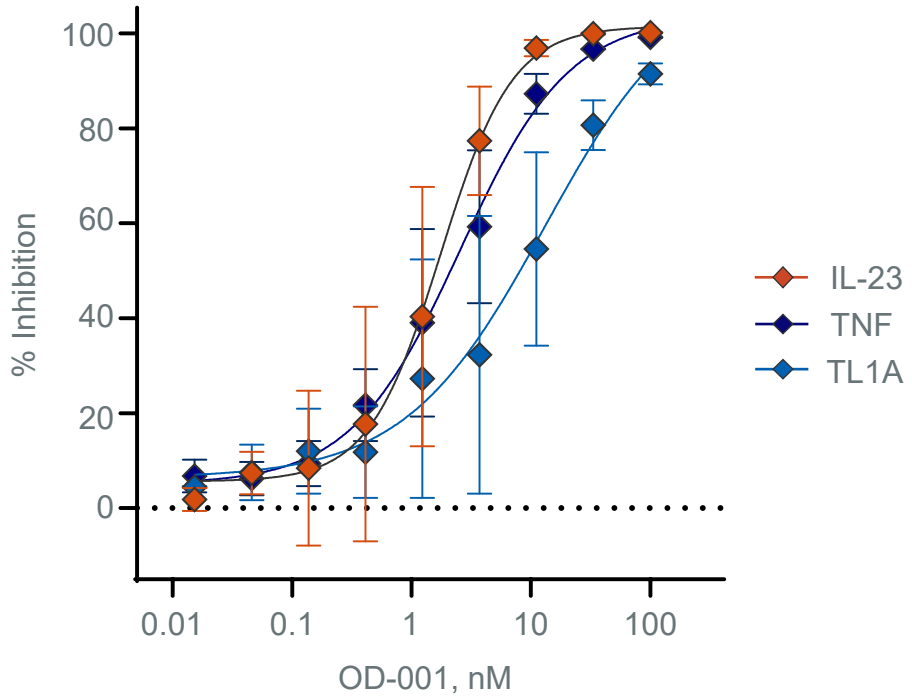




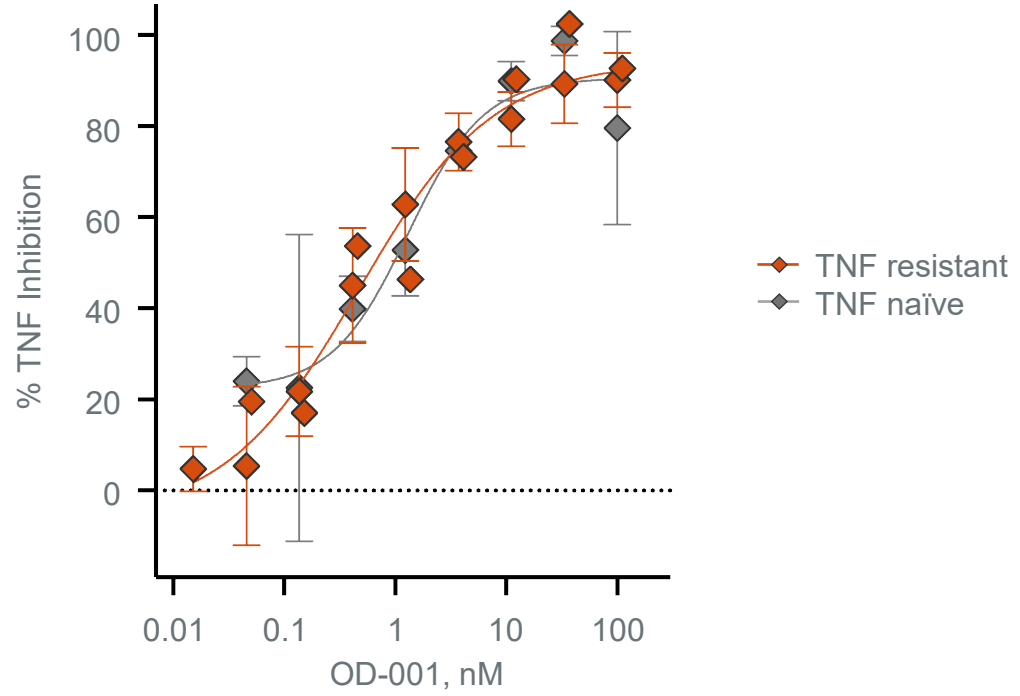
Notes: *** = $p < 0.001$; GSVA = gene set variation analysis; GSVA cut-off for % of UC or CD patients with RIPK2 pathway activity is 0.46 or 0.42, respectively.
Sources: EMBO J 2018;37(17):e99372; Sci Signal 2024;17(819):eabn1101; Gene expression datasets utilized = GSE193677.

OD-001 Blocks Multiple Cytokines That are Validated IBD Drug Targets with Similar Effect in Samples from AT-Experienced or AT-Naïve Patients

Effect on cytokine production from macrophages stimulated with PGN



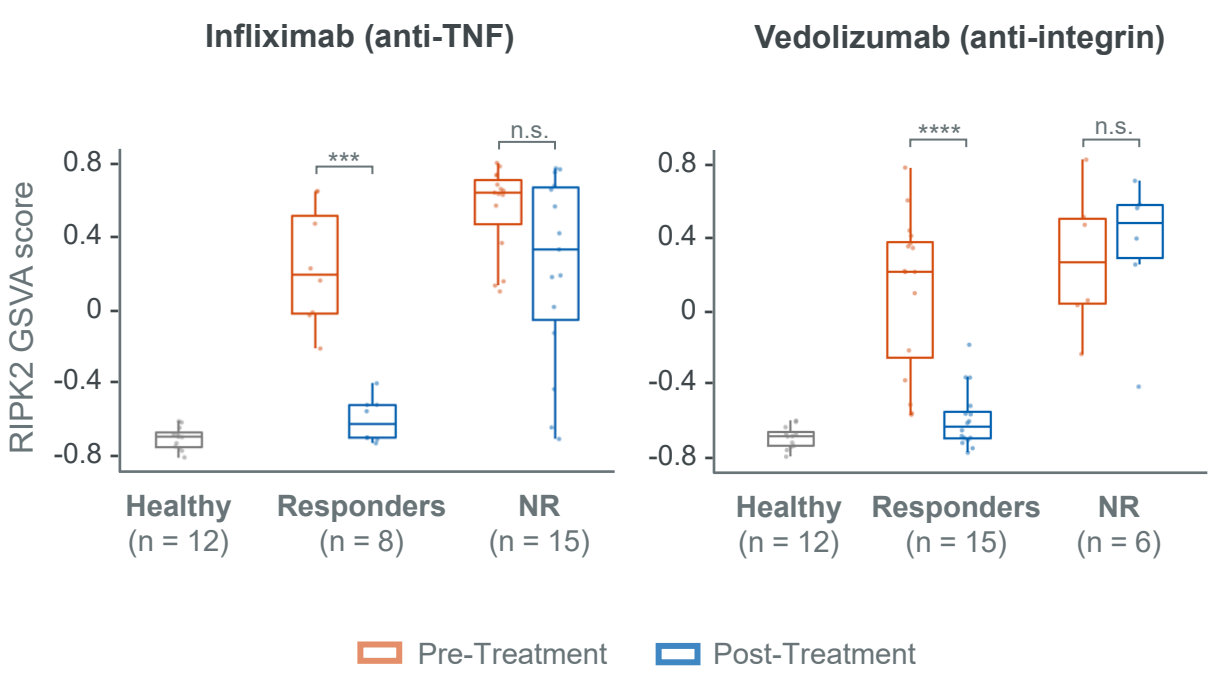
Effect on TNF produced by cells from TNF resistant and naïve patients stimulated with PGN



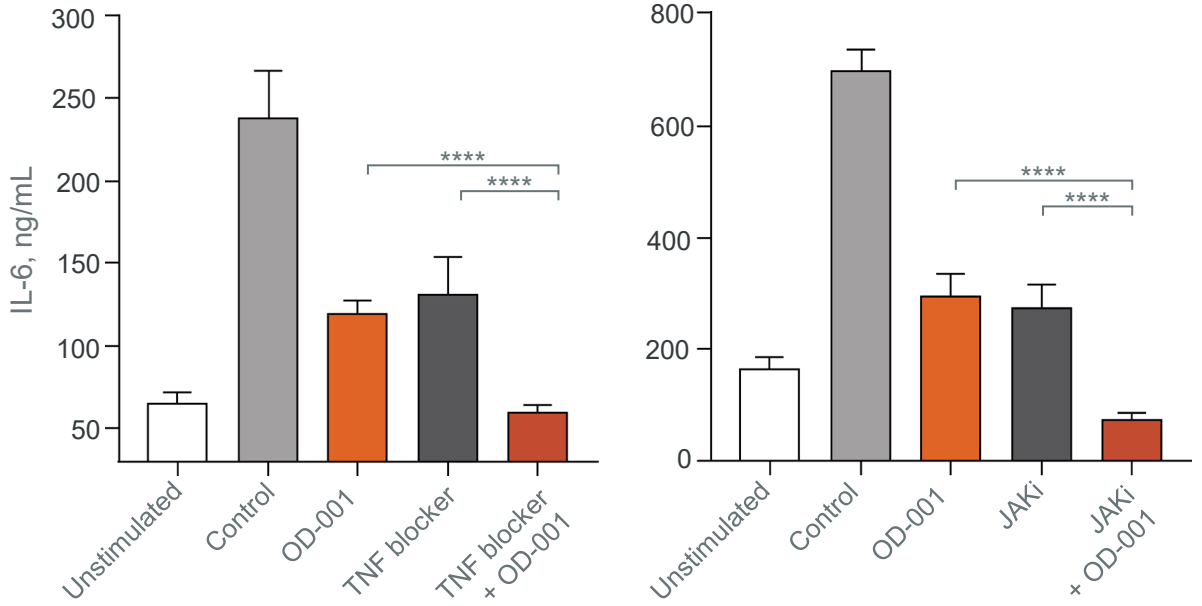
Lamina propria mononuclear cells (LPMCs) were isolated from patient colon tissue

OD-001 Has The Potential To Improve Outcomes By Overcoming Resistance to Standard of Care or Use as a Combination Partner

RIPK2 gene signature remains elevated in non-responders to approved therapies



OD-001 is additive or synergistic when combined with approved therapies and reduced inflammatory cytokines to unstimulated levels



Murine LPMCs from DSS mice activated in vitro with PGN fragment, PMA, and ionomycin

Notes: GSVA = gene set variation analysis; NR = Non-Responder; gene expression datasets utilized = GSE73661; infliximab and vedolizumab datasets used inflamed mucosal tissue; *** = $p < 0.001$; **** = $p < 0.0001$; n.s. = not significant.

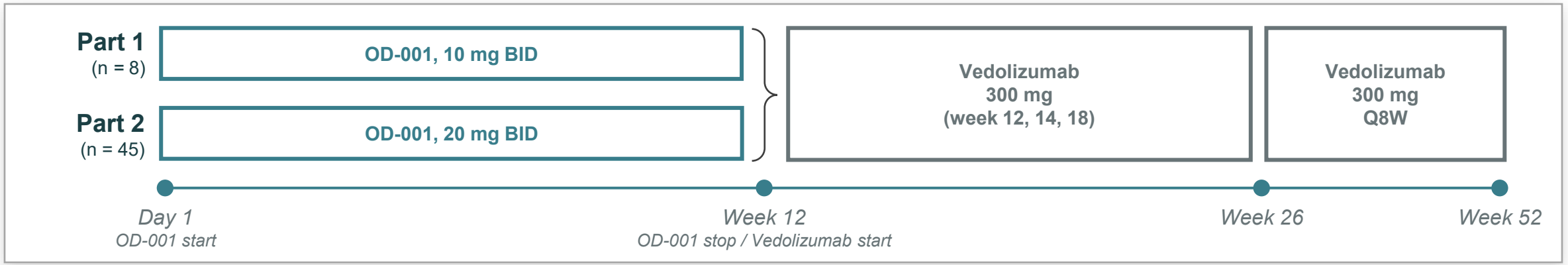
Phase 2a Monotherapy Trial Overview

Trial design (1)

- Enrolled patients with moderate-to-severe UC, modified Mayo Clinical score (MMCS) of 5–9; allowed up to 3 prior advanced therapies
- Both doses expected to be active based on Phase 1 ex vivo TNF release assay
- Features and strategies 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: ≥ 15% placebo-adjusted clinical remission rate at week 12 (comparison to 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 1Q 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		Icotrokinra	Tulisokibart	Duvakitug	Afimkibart
Phase	Phase 2a	Phase 2b	Phase 3	Phase 2a	Phase 2b	Phase 2b	Phase 2	Phase 2b	Phase 2b
Age, mean	40	41	42	39	40	42	41	41	39
Sex, male	51%	59%	-	54%	55%	58%	53%	63%	60%
Advanced therapy experienced (1)	32%	49%	47%	40%	31%	43%	47%	39%	39%
Modified Mayo score (mean)	6.5	7.1	6.9	6.7	6.7	6.6	7.0	6.8	7.0 (median)
MES = 3	51%	71%	60%	49%	50%	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	8	7	8	7
Concomitant corticosteroid use	21%	52%	40%	26%	39%	37%	55%	42%	42%

Lowest concomitant steroid use; minimizes placebo response

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; Morphic UEG Week presentation, October 2025; 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

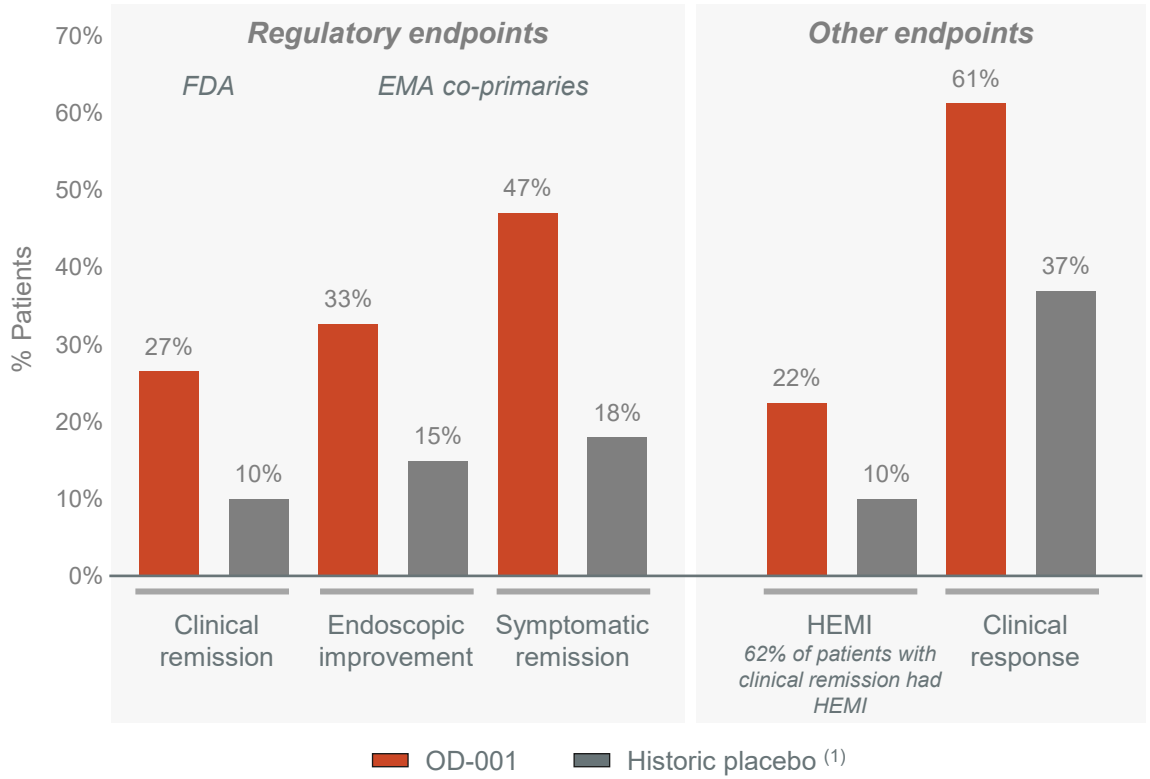
Treatment-Emergent Adverse Events	Part 1: 10 mg BID, n = 8	Part 2: 20 mg BID, n = 45 [1]
Participants with any TEAE (n, %)	**2 (25%)**	**20 (44%)**
Severe (grade ≥ 3) TEAE	0	0
Drug-related TEAE	0	2 (4%)
Liver function abnormality identified as TEAE	0	0
TEAE leading to trial drug discontinuation [1]	0	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.
AE = adverse events; SAE = serious adverse events.

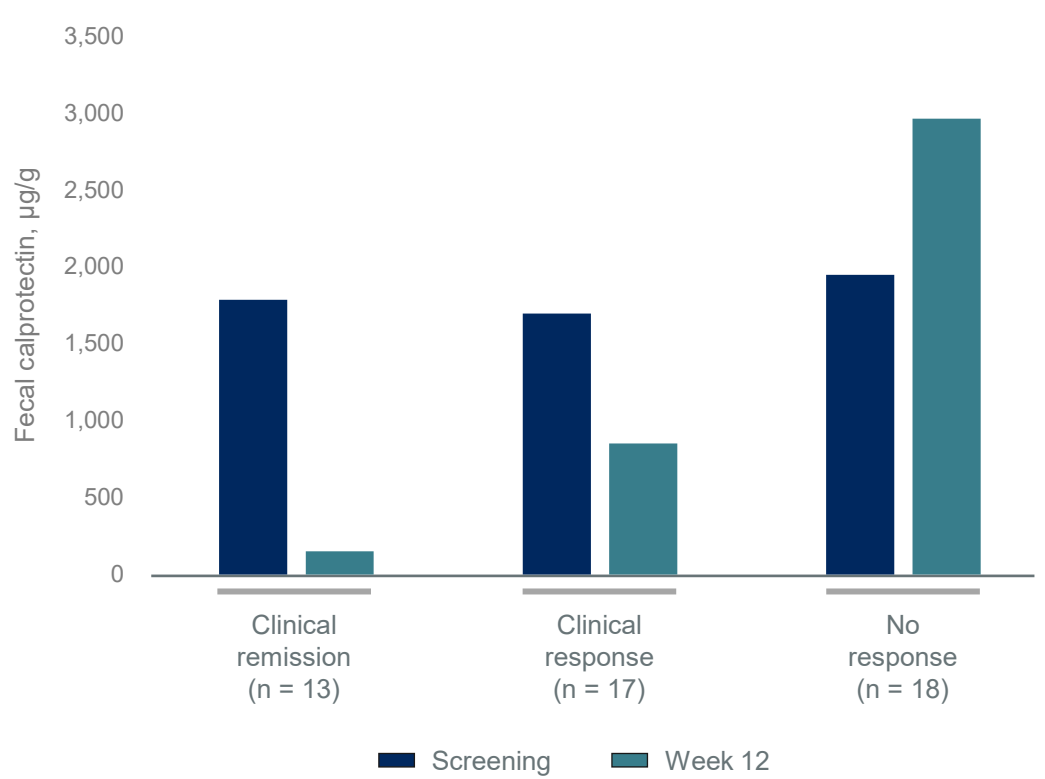
Trial Met Key Efficacy Endpoints, Including Clinical Remission

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)

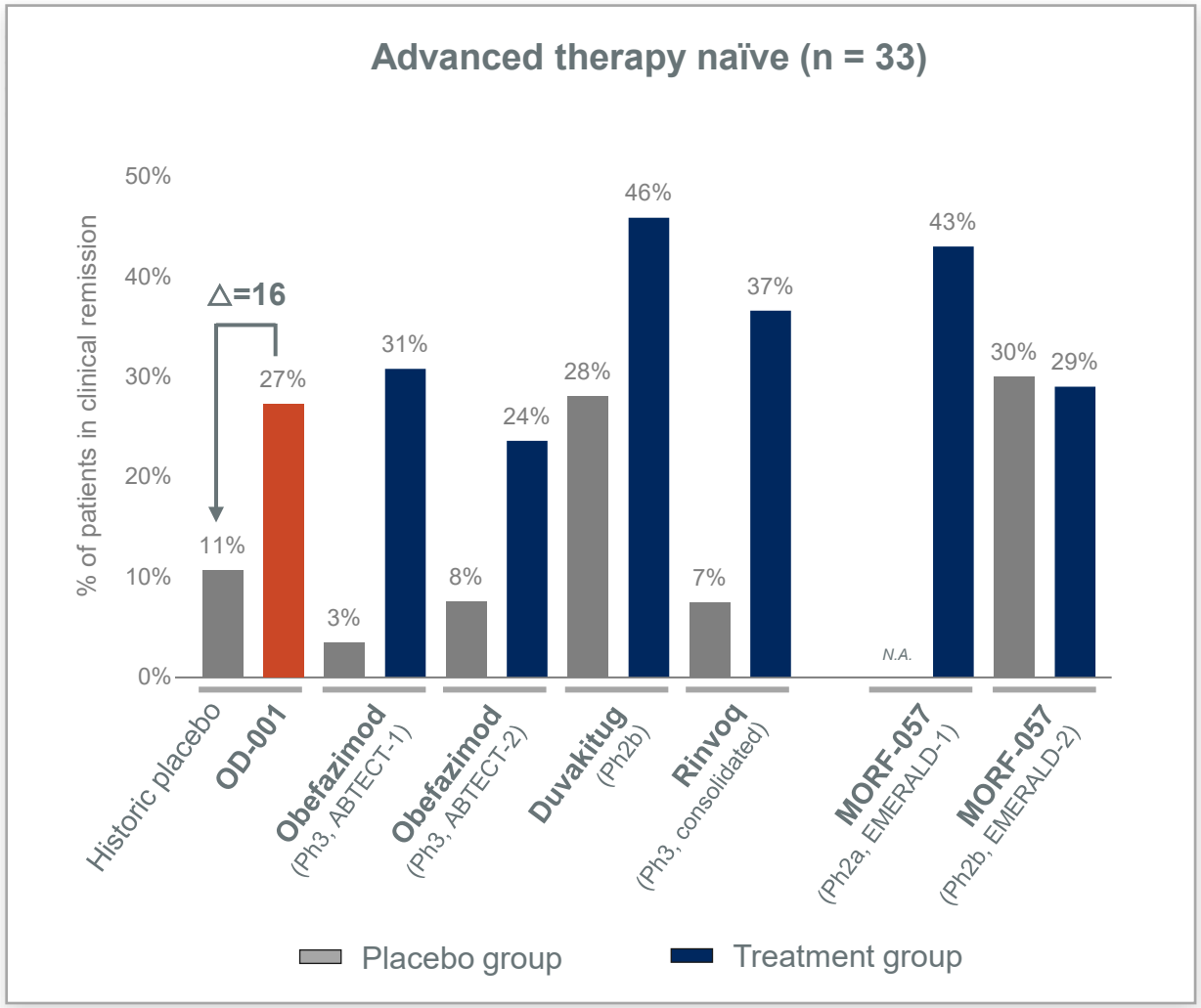


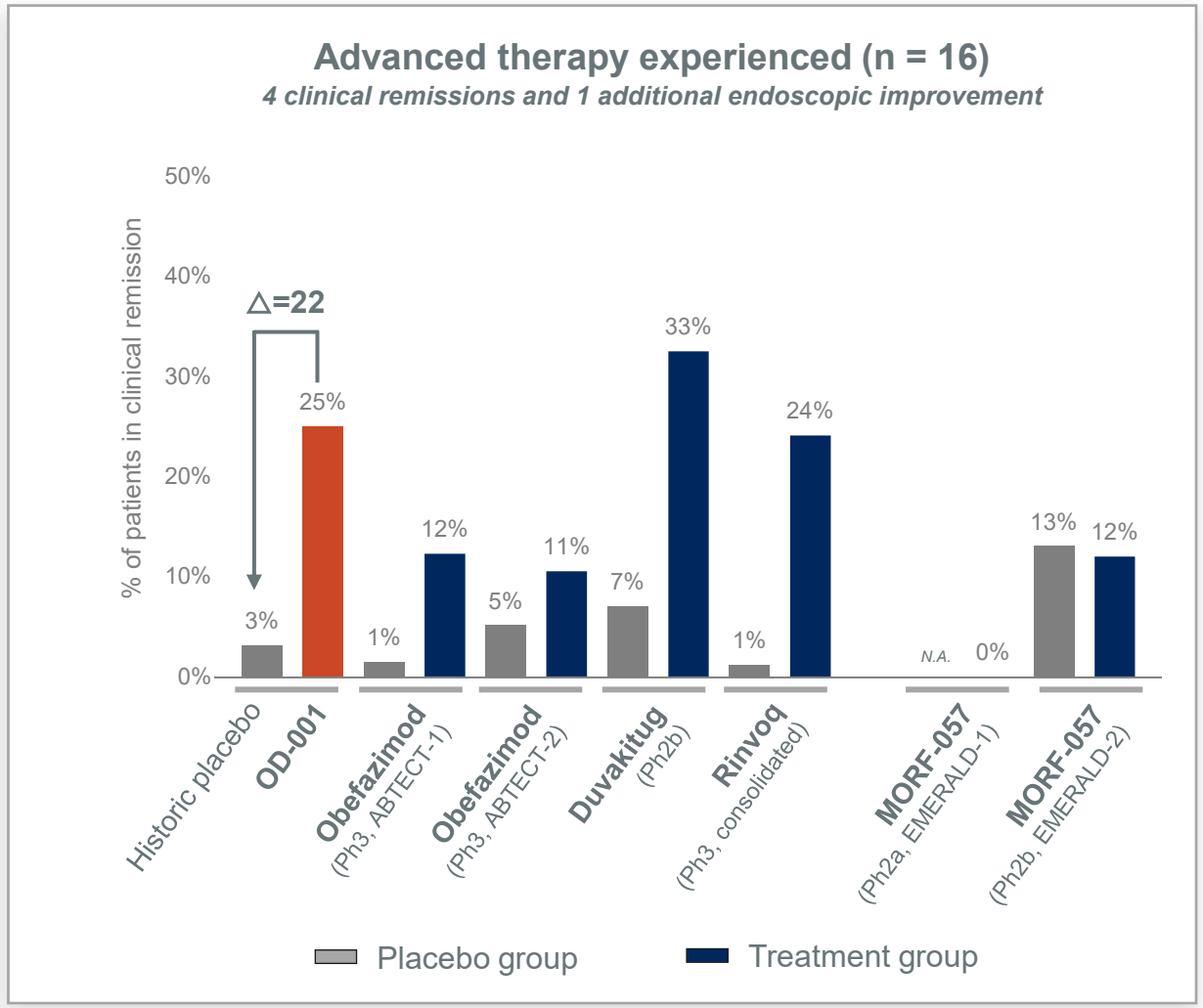
Median fecal calprotectin by week 12 outcome



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).

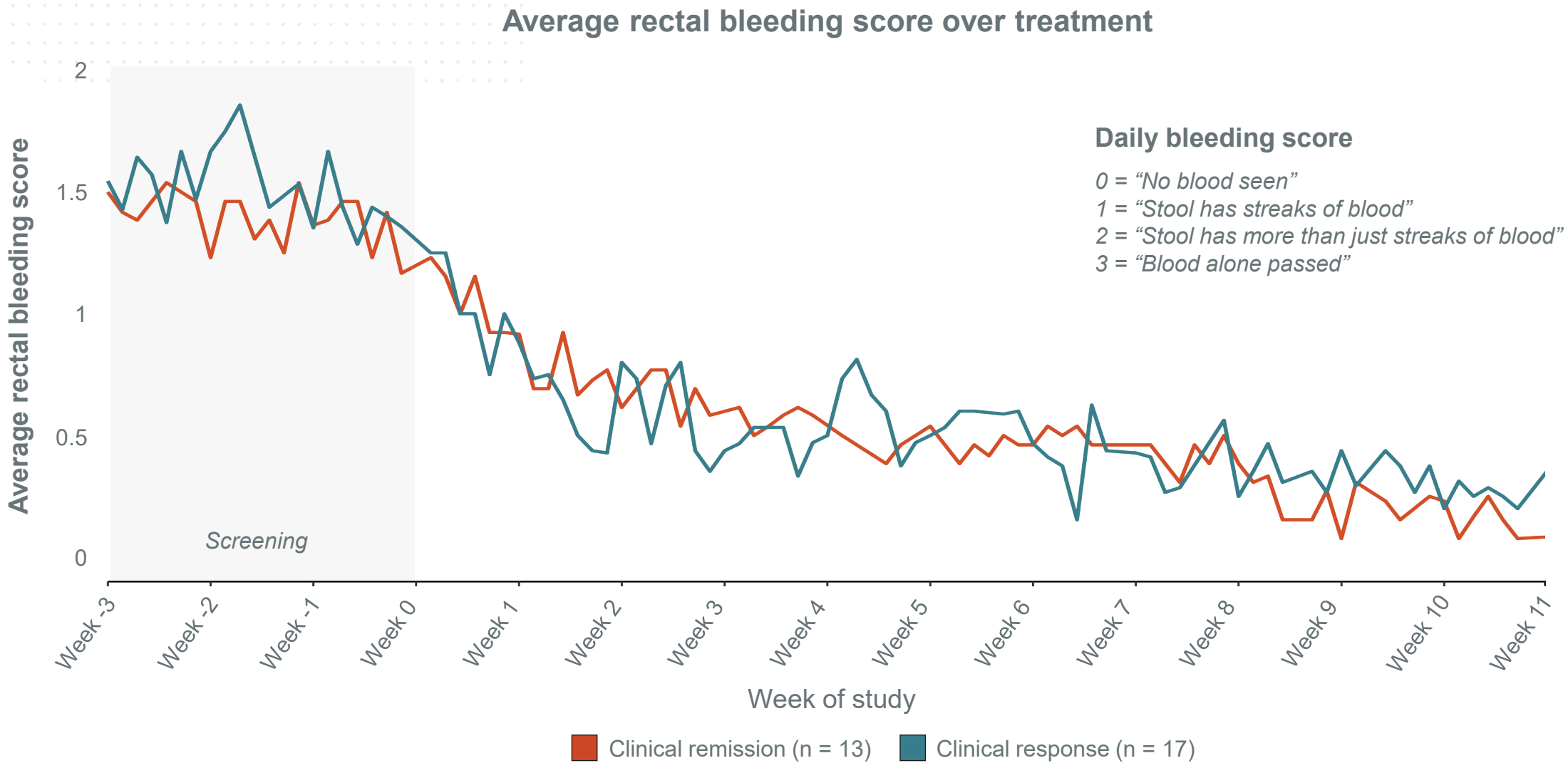
Clinical Remission Rates in AT-Experienced vs. AT-Naïve Patients





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, and historic placebo reflects the weighted average of the placebo rates from the trials indicated. Comparator trial dataset represents 1,458 AT-naïve and 1,254 AT-experienced patients treated with active agent or placebo. The MORF-057 phase 2a study did not include a placebo control. 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. AT = advanced therapy.

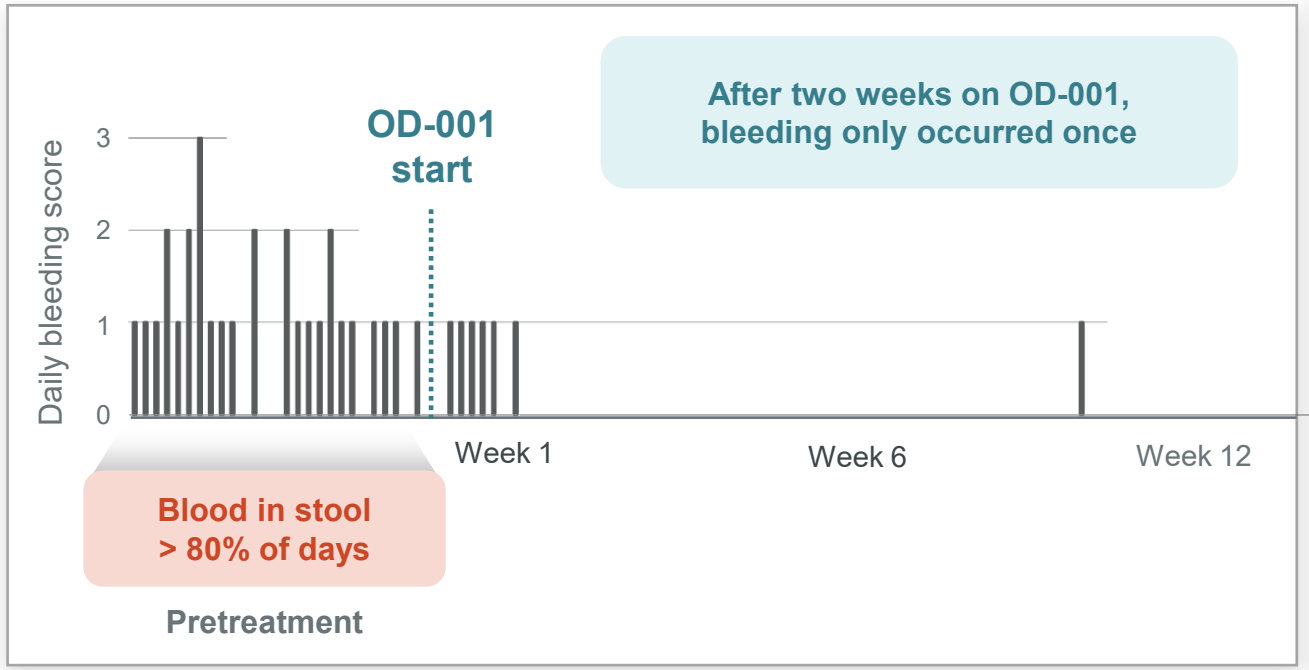
Rapid Onset of Action With Deepening of Response, Consistent with MoA



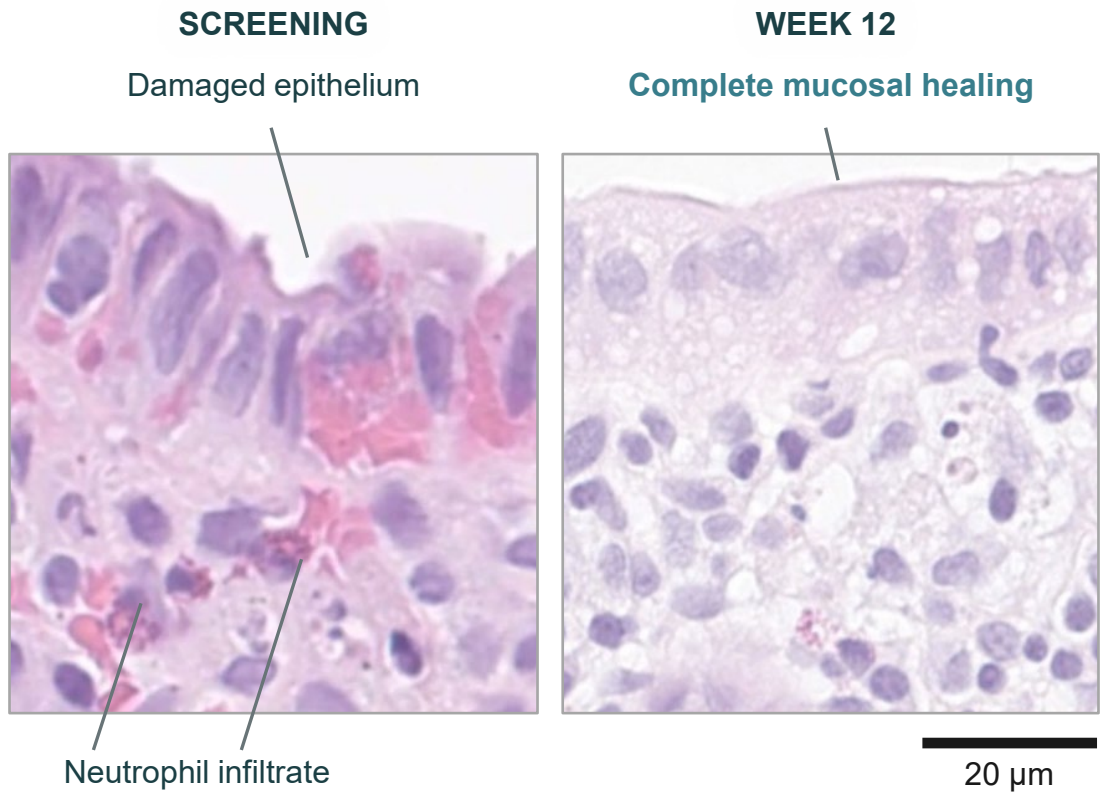
Disclaimer: Data presented are from ongoing studies, which will not be finalized until database lock. Thus, these data are subject to change prior to release of the CSR.
Note: OD-001 results are consolidated from both part 1 and part 2.

Patient Vignette A: Clinical Remission in AT-Experienced Patient

- 44-year-old Australian male with 13-year history of UC
  - Previously treated with anti-TNF therapy
  - No concomitant steroid use during study
- MES of 2 and MMCS of 6 at screening
- **Clinical remission achieved by week 12 with MES of 0** [1]



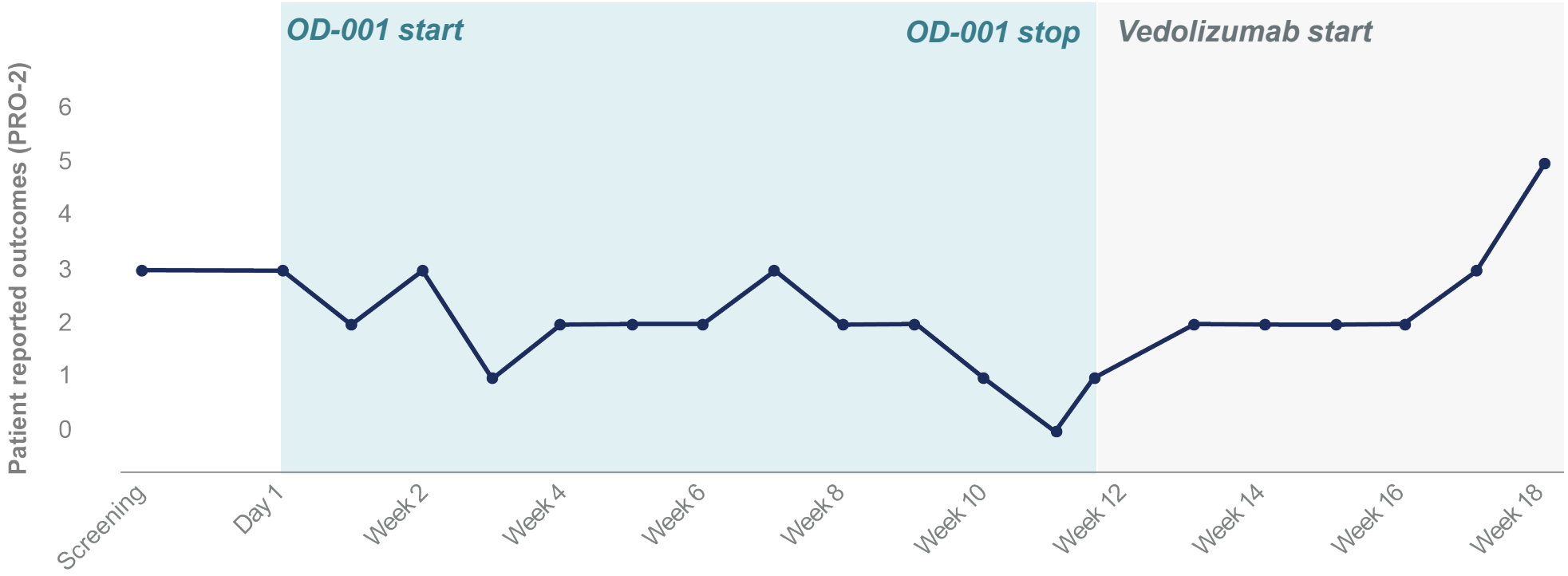
Histopathology showed evidence of mucosal healing



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.
Notes: (Left) Daily bleeding score: "no blood seen" = 0, "stool has streaks of blood" = 1, "stool has more than just streaks of blood" = 2, and "blood alone passed" = 3; (Right) JPG images of colon tissue samples stained with hematoxylin and eosin are non-validated conversions of raw data.

Patient Vignette B: 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)]



Disclaimer: (1) While we believe the observations derived from this patient's results are informative, the results depicted above 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.
Note: PRO-2 score is comprised of a patient-reported rectal bleeding score and stool frequency score.

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
  - ***Patients, regardless of prior treatment history, are eligible for combination treatment with OD-001***

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

RINVOQ upadacitinib

Velsipity (etrasimod) tablets

Injectables

HUMIRA adalimumab

Entyvio vedolizumab

Skyrizi risankizumab-rzaa

Tulisokibart

Combinations

Odyssey opportunity and development plan

~$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

Expansion of addressable population in 2028 and beyond

$4B+ Combination induction

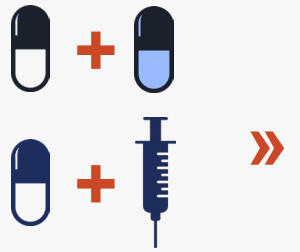
OD-001 combinations +

- **+ Vedolizumab** *(α4β7)*
- **+ Rinvoq** *(JAK)*

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

Phase 2a vedolizumab combination induction results expected in 2H 2027

$7B+ Combination induction with monotherapy maintenance



Combination induction

OD-001 maintenance

Preferred therapy for induction and maintenance

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

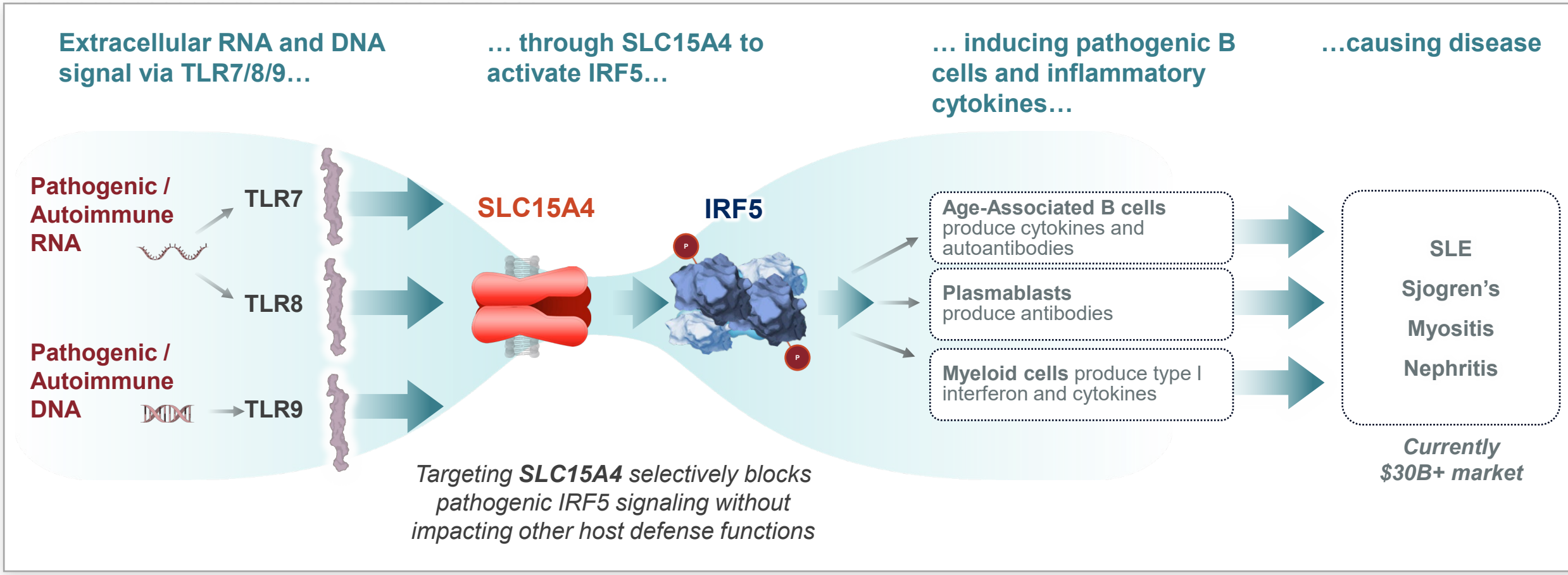
Oral SLC15A4 Inhibitor: OD-002

Modulating B Cell Activity and Interferon Signaling

IND-Enabling Studies Ongoing

Program Inception to GLP Studies: ***15 Months***

RNA and DNA Signaling Pathways Converge on SLC15A4 to Activate IRF5, a Master Regulator of Autoimmune Responses



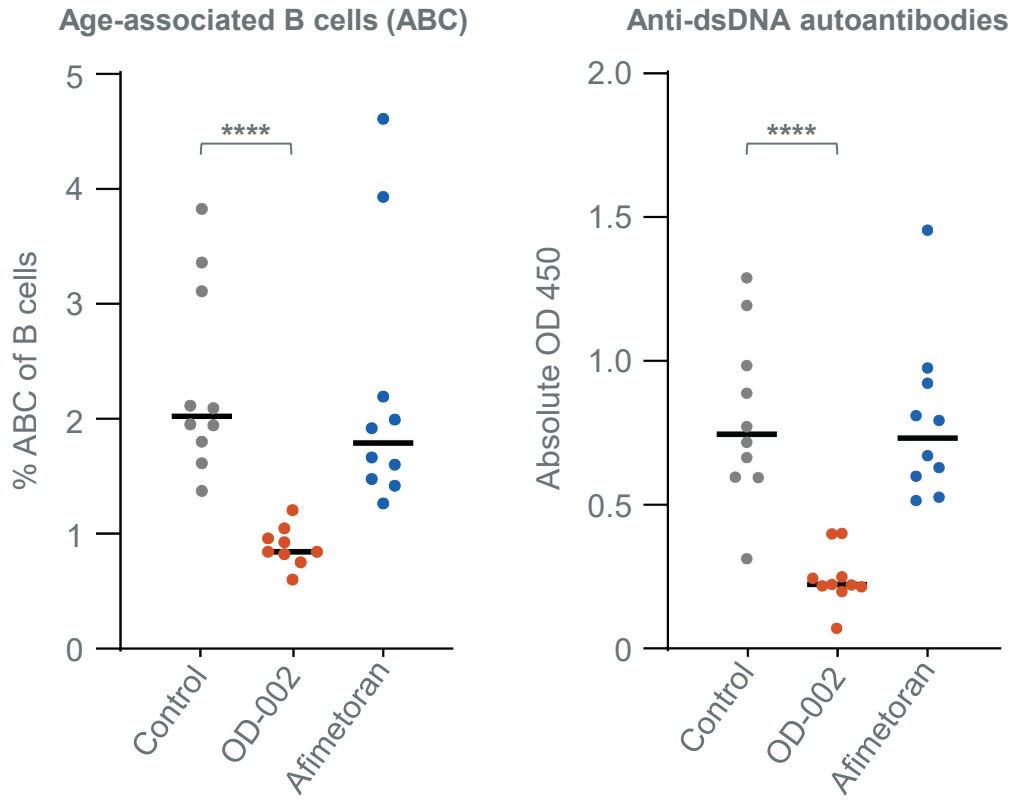
Source: Evaluate Pharma.

SLC15A4 Development Candidate, OD-002, Observed to Address Multiple Aspects of Immune Cell-Mediated Autoimmune Pathogenesis

OD-002 is potent and selective with favorable druglike properties

Potency in vitro IC_{50} in human primary cells (e.g., TNF, IFN)	≤ 10 nM
Potency in vivo IFNα inhibition at 3 mg/kg; phenocopies SLC15A4 knockout	> 95%
Selectivity TLRs, STING, and SLC15A1/2/3	> 1,000x
Projected once-daily human dose Dose to maintain IC_{90}	18 mg
Tolerability margins from 2-week studies	> 60x
Intellectual property Composition of matter	2046

OD-002 reduced pathogenic B cell populations and autoantibody production



7–9-week-old lupus-prone mice were dosed for 3 weeks, and B cells (from spleen) and anti-dsDNA autoantibodies (from plasma) were analyzed; afimetoran is a TLR7/8 inhibitor currently in clinical development

Notes: **** = p < 0.0001.

Potential for CAR-T-Like Modulation of Pathogenic B Cells in a Pill

	CD19 and BCMA-targeted CAR-T therapy	OD-002
TARGET	Broad B cell depletion *Removes healthy and pathogenic B cells*	Targeted to B cells sustained by pathogenic RNA and DNA *Spares non-disease associated B cells*
EFFICACY	Patients with multiple autoimmune diseases have achieved disease remission *(SLE, myositis, MG)*	Potential for deep and durable remission across multiple indications
DURABILITY	B cells return ~110 days post-infusion *Following B cell reconstitution, relapse risk emerges* "Immune reset" hypothesis and re-treatment potential are unproven	Continuous but reversible pathway suppression *Daily oral dosing sustains suppression of pathogenic B cells* Persistent inhibition of expansion and activation by RNA and DNA
ACCESS & BURDEN	Autologous manufacturing • Inpatient *4–8-week lead time • $400–600k+ per cycle • Lymphodepletion required • Risk of CRS and hypogammaglobulinemia* Academic or hospital setting only	Oral small molecule • Outpatient *No manufacturing lead time • No hospitalization* *Small molecule COGS • Potential for improved tolerability and safety* Outpatient setting
POPULATION	< 5% of autoimmune patients *Restricted to most refractory cases* 	Designed for usage across treated patients *Expect few or no eligibility limitations* 

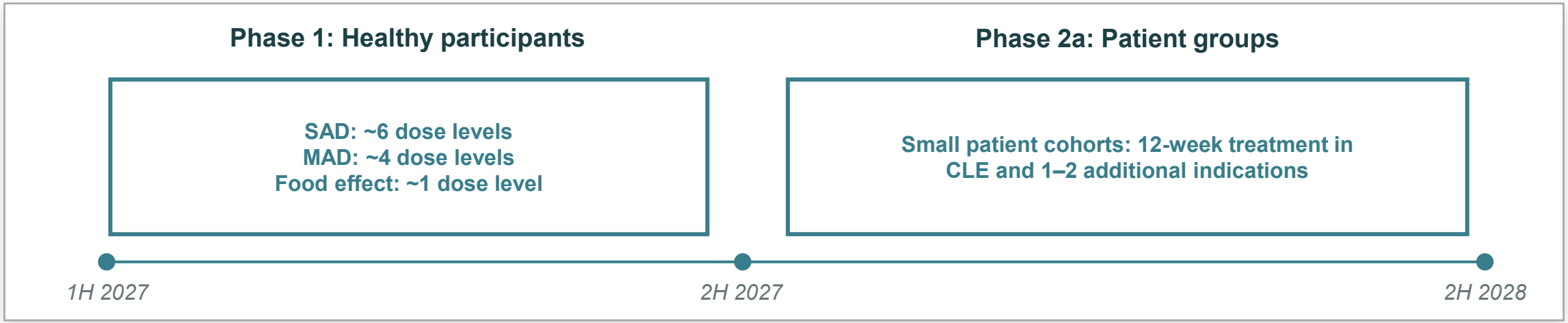
Phase 1/2a Trial Initiation Expected in 1H 2027 With Proof-of-Concept Expected in 2H 2028

Trial overview

- Phase 1: Randomized, double-blind, placebo-controlled SAD, MAD, and food effect cohorts in ~100 healthy participants
- Phase 2a: Basket trial design, planned to enroll cutaneous lupus erythematosus patients along with signal-seeking cohorts in other autoimmune diseases
  - CLE eligibility: Active CLE with CLASI-A score ≥ 8
  - Precedent for establishing PoC in CLE with < 50 patient studies based on experience with TLR7/8 antagonists

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.

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

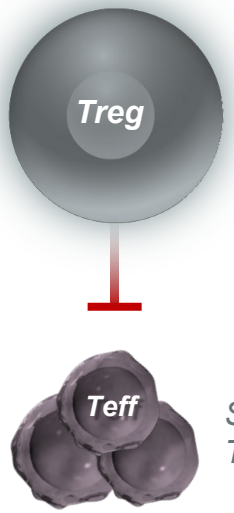
Atopic dermatitis | SLE | Alopecia areata | Vitiligo | Multiple sclerosis | Rheumatoid arthritis | Asthma

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

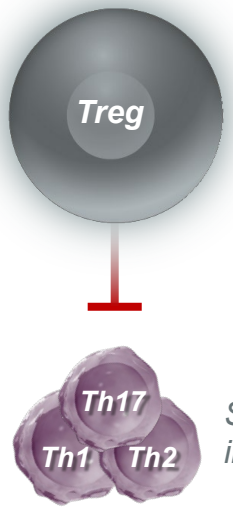


Stops Teff cells

EFFECT 2:

Reduction of immune cell activity

Reduces production of proinflammatory cytokines, including IFNɣ, IL-4, IL-5, and IL-13

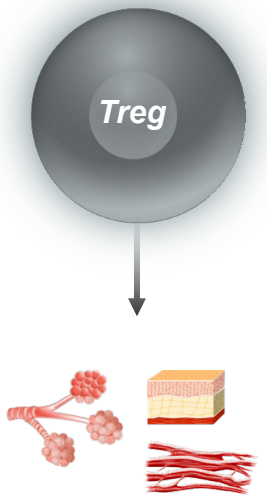


Stops immune cells

EFFECT 3:

Repair of tissue-resident non-immune cells

Induces durable disease-modifying effects

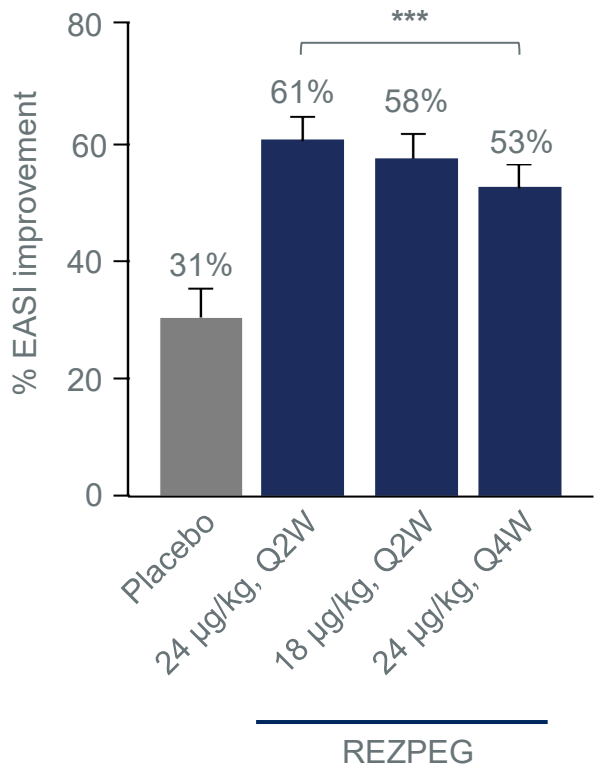


Repairs lung, skin, and muscle cells

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

TRexBio

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

NEKTAR®

NKTR-1065:
IND submission expected in 2027

ODYSSEY THERAPEUTICS

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	CLINICAL SIGNAL	TRANSLATIONAL EVIDENCE	MECHANISTIC EVIDENCE
$30B+ Dermatology *AD, alopecia areata, vitiligo*	**~$25B Respiratory** *Asthma*	**$20B+ Neurology** *Multiple sclerosis*	**~$25B IBD** *UC and CD*
• Rezpeg, IL-2 agonist, demonstrated efficacy in moderate-to-severe AD and alopecia areata	• Rezpeg-treated AD patients with self-reported asthma history showed improvement in asthma control questionnaire (ACQ-5) scores	• TNFR2 agonism reduces disease severity in EAE models (representative of human multiple sclerosis)	• 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.

Final Words

Significant Clinical Milestones Expected Through 2028

2026

- 2H: Initiation of OD-001 phase 2b monotherapy trial and phase 2a combination trial in UC
- 2H: File CTA for OD-002 (SLC15A4) program

2027

- 1H: Initiate OD-002 healthy participant dosing in Ph1/2 trial
- 2H: Complete OD-002 healthy participant dosing in Ph1/2 trial
- 2H: OD-001 phase 2a vedolizumab combination induction readout
- 2H: OD-001 phase 2b monotherapy induction readout

2028

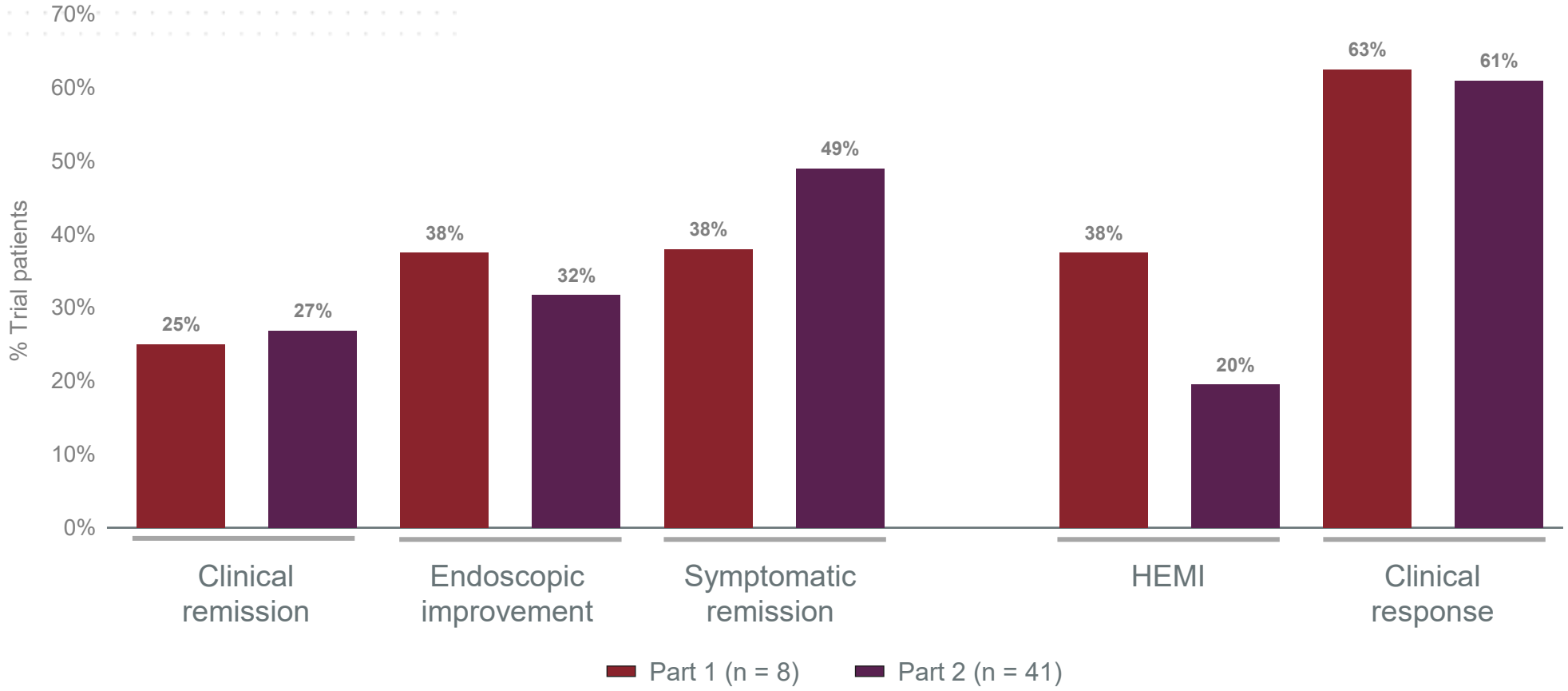
- 2H: OD-002 phase 2a readouts in multiple indications
- 2H: First OD-001 maintenance readout

ODYSSEY THERAPEUTICS

Thank you!

Appendix

Similar Activity Observed Across Efficacy Measures in Part 1 and Part 2 in Phase 2a Monotherapy Trial of OD-001

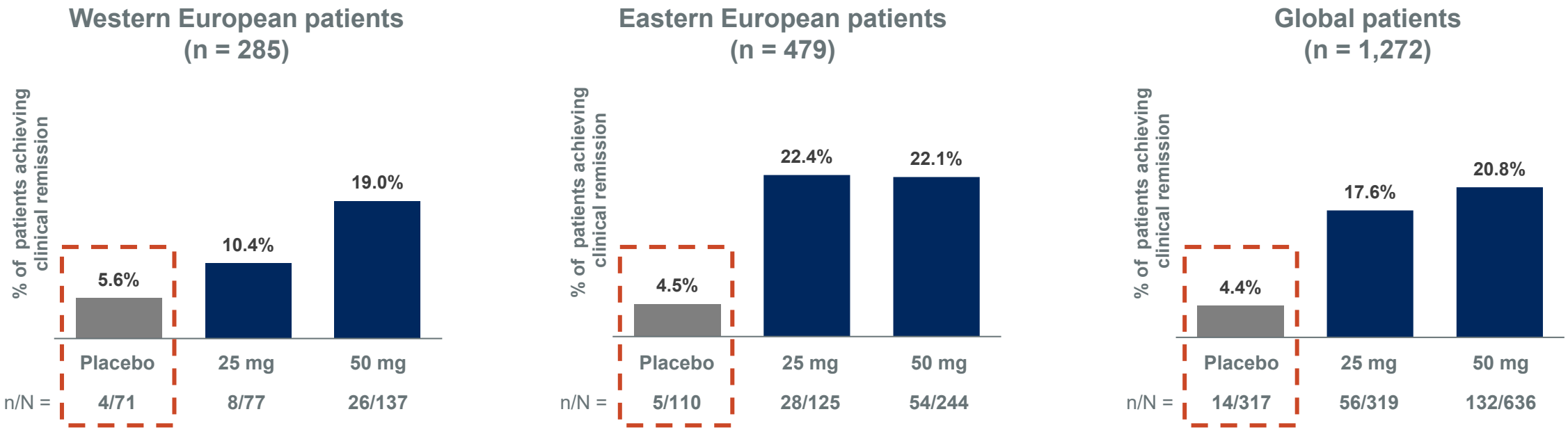


Part 1 and part 2 each met the primary endpoint of change in MMCS from baseline (-3.1, p = 0.004 and -2.5, p < 0.001, respectively)

Disclaimer: Data presented are from ongoing studies, not finalized until database lock and, thus, are subject to change prior to release of the CSR. Future results may differ from those shown here.
Note: Includes all patients who completed 12 weeks of treatment per protocol (n = 49).

Placebo Clinical Remission Rates Are Generally Similar Across Regions

Consolidated results from Abivax's ABTECT-1 and ABTECT-2 phase 3 UC trials



Source: Scaldaferri F, Seidler U, Treton X, et al. ECCO 2026; P1159.