

**UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549**

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): June 23, 2026

ODYSSEY THERAPEUTICS, INC.

(Exact name of Registrant as Specified in Its Charter)

Delaware
(State or Other Jurisdiction
of Incorporation)

001-43270
(Commission File Number)

86-3384382
(IRS Employer
Identification No.)

51 Sleeper Street, Suite 800
Boston, Massachusetts
(Address of Principal Executive Offices)

02210
(Zip Code)

Registrant's Telephone Number, Including Area Code: (617) 865-9628

(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

- Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
- Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
- Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
- Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, par value \$0.001 per share	ODTX	The Nasdaq Stock Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act.

Item 7.01 Regulation FD Disclosure.

On June 23, 2026, Odyssey Therapeutics, Inc. (the “**Company**”) posted a corporate presentation on its website at <https://investors.odysseytx.com/news-events/presentations>. A copy of the presentation is furnished herewith as Exhibit 99.1 to this Current Report on Form 8-K. The information contained in Item 7.01 of this Current Report on Form 8-K and Exhibit 99.1 attached hereto is intended to be furnished and shall not be deemed “filed” for purposes of Section 18 of the Exchange Act, or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act or the Exchange Act, except as expressly set forth by specific reference in such a filing. The furnishing of this information hereby shall not be deemed an admission as to the materiality of any such information.

Item 9.01 Financial Statements and Exhibits.

Exhibit Number	Description
99.1	Corporate Presentation, dated June 2026
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Odyssey Therapeutics, Inc.

Date: June 23, 2026

By: /s/ Jason Haas
Jason Haas
Chief Financial Officer



Corporate Presentation

June 2026



Disclaimers

Confidential information, forward-looking statements, and third-party information

This presentation (including any discussion that accompanies this presentation) contains highly confidential information regarding Odyssey Therapeutics, Inc. (the "Company," "Odyssey," "we," or "us") and its subsidiaries and may not be disclosed, copied, or distributed to any person other than the intended recipient, directly or indirectly, in whole or in part, without the Company's prior written consent.

This presentation contains forward-looking statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995. All statements other than statements of historical facts are forward-looking statements, including, but not limited to, statements regarding our business strategy; the future clinical development, efficacy, safety, and therapeutic potential of our product candidates; expectations regarding the initiation, design, and reporting of data from our preclinical studies and clinical trials; future financial results; anticipated manufacturing plans; expectations regarding regulatory approval of any of our product candidates; and plans for future operations or other characterizations of future events. In some cases, you can identify forward-looking statements through the use of words such as "may," "will," "should," "expects," "plans," "anticipates," "could," "intends," "target," "projects," "contemplates," "believes," "estimates," "predicts," "potential," or "continue," or the negatives of these words or other similar terms or expressions that concern our expectations, strategy, plans, or intentions. These statements are not guarantees of future performance and involve risks and uncertainties, some of which are beyond our control, and you are cautioned not to place undue reliance on any forward-looking statements. Among the factors that could cause actual results to differ materially from those suggested by forward-looking statements are: our current and future research and development activities and expenses; sufficiency of our capital resources; our anticipated regulatory activities and our ability to obtain regulatory approval for any of our product candidates; our ability to commercialize future products, if approved; our ability to enroll patients in clinical trials; possible safety and efficacy concerns; the timing and results of our preclinical studies and clinical trials; and risks facing our operations and intellectual property. Although the Company believes the information contained in this presentation related to the Company is accurate in all material respects, neither the Company nor any other person assumes responsibility for the accuracy and completeness of the information contained in this presentation except to the extent required by law. The information contained in this presentation is provided for discussion purposes only as of the date hereof, and the Company undertakes no obligation to update or revise such information, including any forward-looking statements, whether as a result of new information, future events or circumstances, or otherwise, except to the extent required by law.

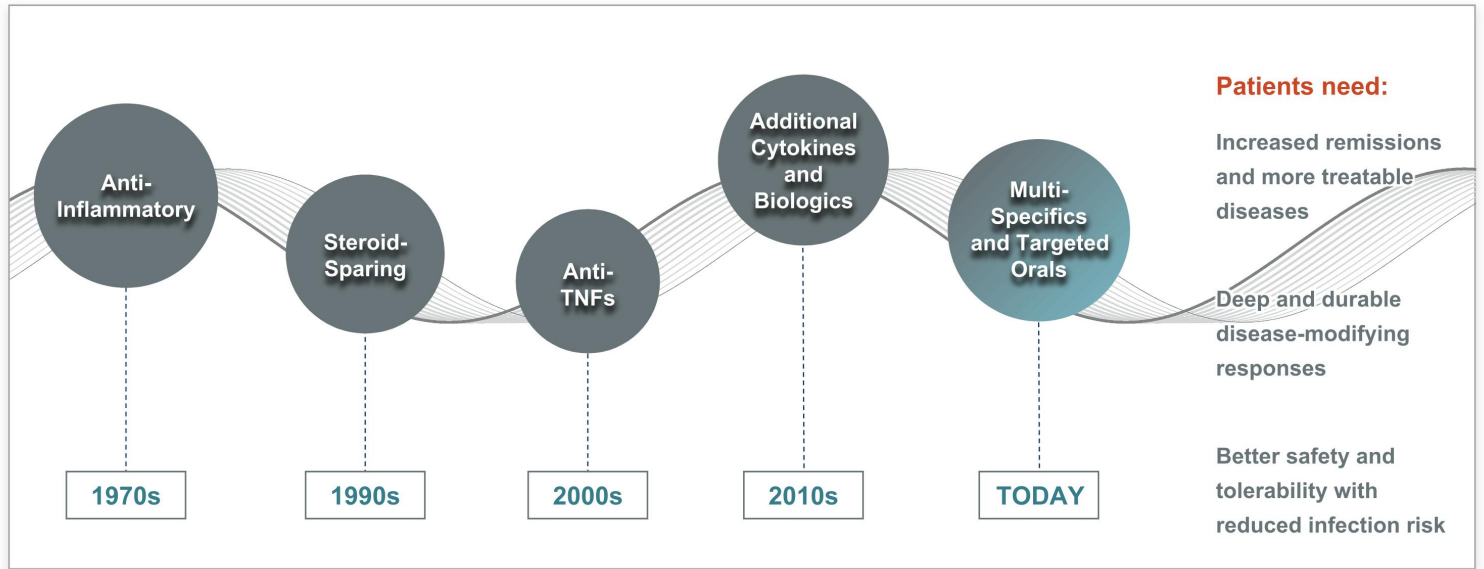
Certain information contained in this presentation and statements made orally during this presentation may relate to or be based on studies, publications, estimates, projections, and other data obtained from third-party sources as well as our own internal estimates and research. While we believe these third-party sources to be reliable as of the date of this presentation, we have not independently verified, and make no representation as to the adequacy, fairness, accuracy, or completeness of any information obtained from third-party sources.

Intellectual property

This presentation contains references to our trademarks and service marks and to those belonging to other entities. Solely for convenience, trademarks and trade names referred to in this presentation, including logos, artwork and other visual displays, may appear without the ® or ™ symbols, but such references are not intended to indicate in any way that we will not assert, to the fullest extent under applicable law, our rights or the rights of the applicable licensor to these trademarks and trade names. This presentation may also contain trademarks, service marks, and trade names of third parties, which are the property of their respective owners. We do not intend our use or display of other entities' trade names, trademarks, or service marks to imply a relationship with, or endorsement or sponsorship of us by, any other entity.

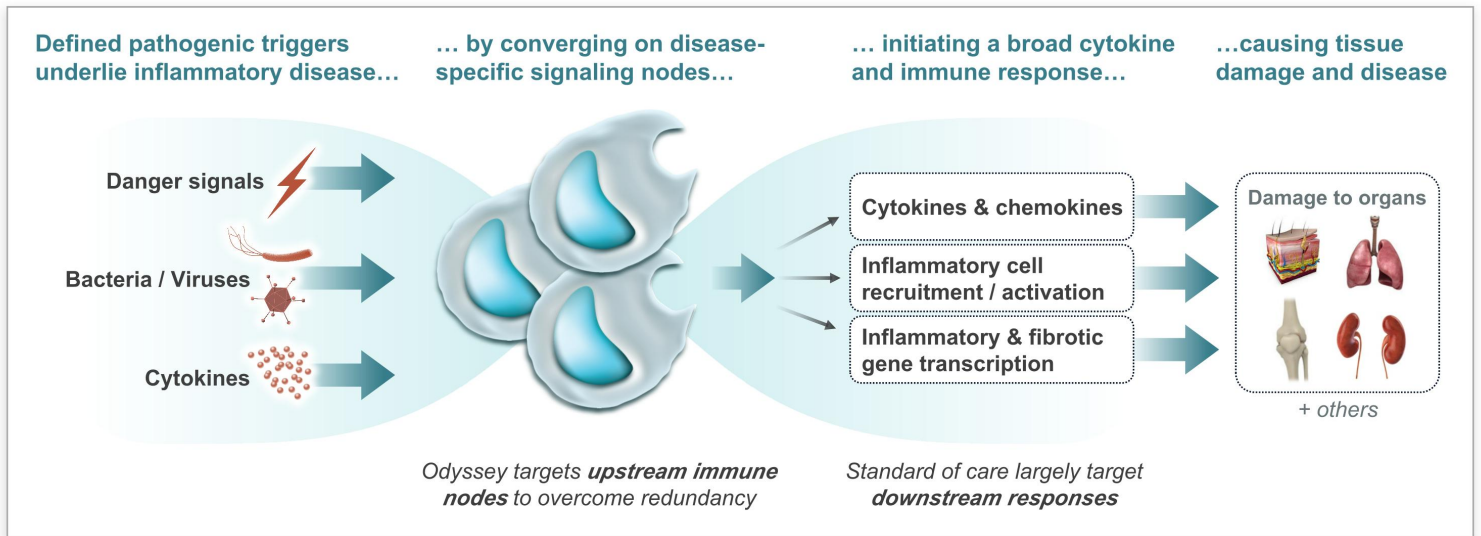


Despite Decades of New Therapies, Few Patients With Autoimmune Disease Have Durable Remission



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⁾

R&D LEADERSHIP

Gary Glick, Ph.D.
Founder and CEO



Steve Soisson, Ph.D.
Protein Therapeutics

Tony Opirari, M.D., Ph.D.
Interim Chief Medical Officer

Bill Roush, Ph.D.
Small Molecule Research

Joe McDonald, Ph.D.
Chief Data Officer

Jeff Hermes, Ph.D.
Enabling Sciences

Natalie Dales, Ph.D.
Research Operations

Luigi Franchi, M.D., Ph.D.
Immunology

BOARD OF DIRECTORS

Jeff M. Leiden, M.D., Ph.D.

Shelley Chu, M.D., Ph.D.

Nia Tatsis, Ph.D.

Paulina Hill, Ph.D.

Valerie Odegard, Ph.D.

Tim Walbert

Nan Li

Ian F. Smith

Ksenija Pavletic

Carolyn Ng, Ph.D.



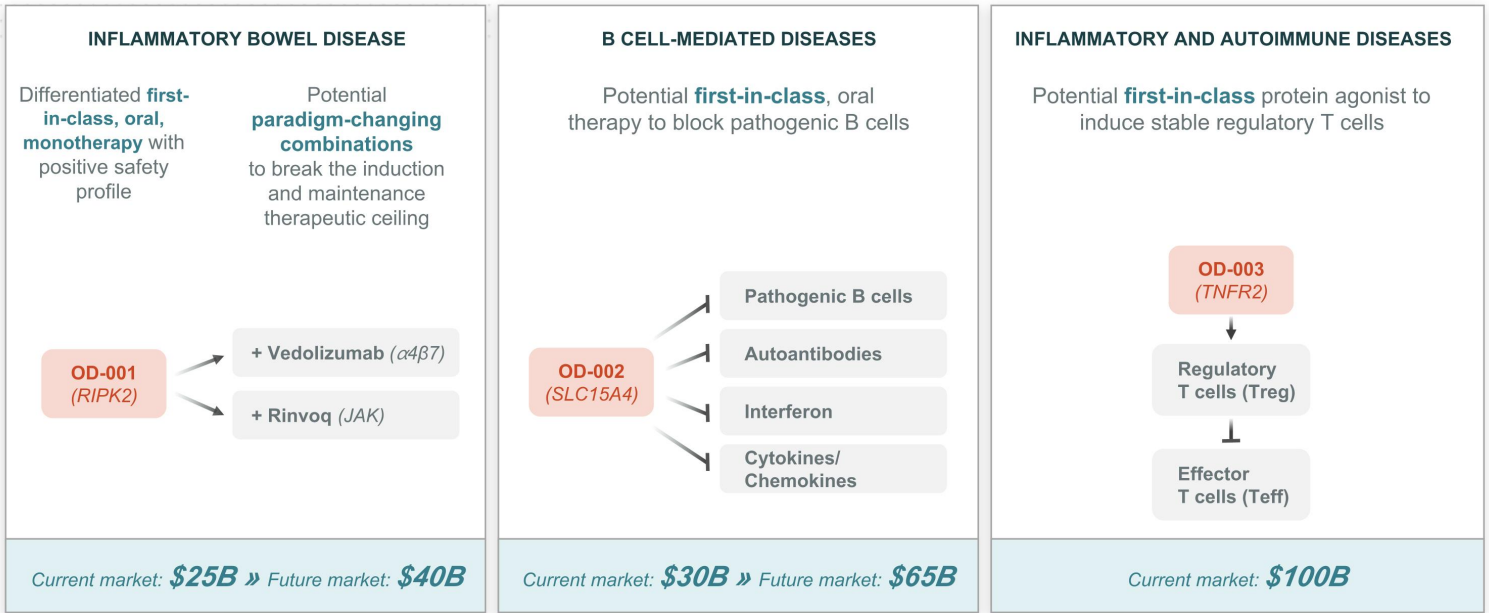
⁽¹⁾ Pro-forma for net proceeds from IPO and concurrent private placement.

Copyright 2026 | Odyssey Therapeutics | All Rights Reserved

In search of extraordinary for patients

5

Pioneering Next Generation I&I Therapies to Redefine Standard of Care



Source: Evaluate Pharma.

Copyright 2026 | Odyssey Therapeutics | All Rights Reserved

In search of extraordinary for patients

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	[Progress bar spanning Discovery, IND-Enabling, Phase 1, and Phase 2]						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	[Progress bar spanning Discovery and IND-Enabling]						2H 2026: CTA filing 1H 2027: Phase 1/2 initiation
TNFR2, OD-003 Protein	AD, SLE, alopecia areata	[Progress bar spanning Discovery and IND-Enabling]						
TSLP/IL-33, OD-004 Protein	Asthma, COPD	[Progress bar spanning Discovery]						

\$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.

Copyright 2026 | Odyssey Therapeutics | All Rights Reserved

In search of extraordinary for patients

7

Data to Build a Potential RIPK2 IBD Franchise Expected by 2028

Monotherapy induction efficacy



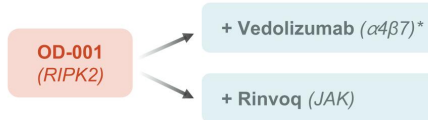
March 2026

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



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.

Copyright 2026 | Odyssey Therapeutics | All Rights Reserved

In search of extraordinary for patients

8

Oral RIPK2 Scaffolding Inhibitor: OD-001

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

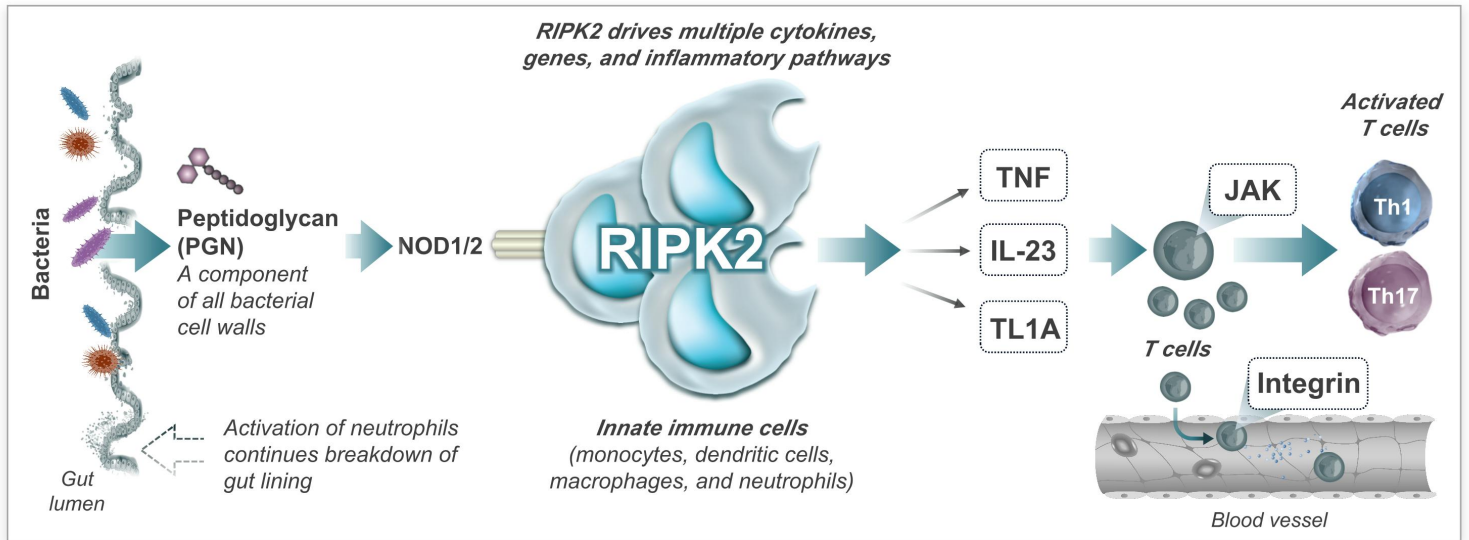
Proof-of-Concept Achieved in Ulcerative Colitis

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



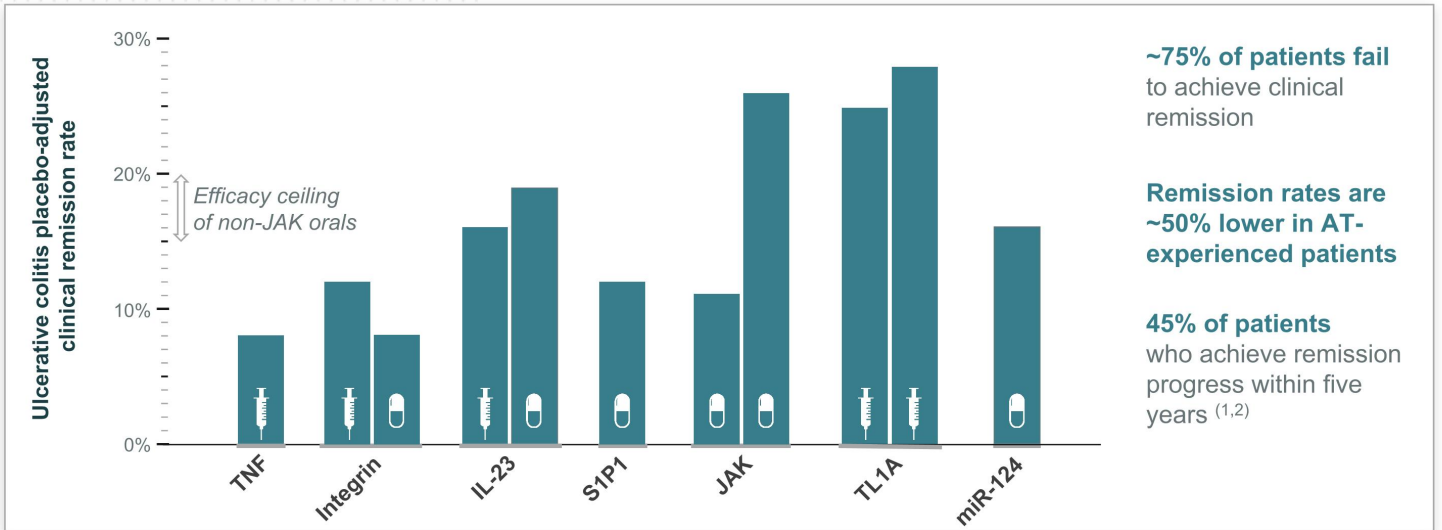
IBD Is Driven by Innate Immune Cells Responding to Gut Bacteria

- 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



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 UCDD, ECCO 2025; ABTECT-1 and ABTECT-2, UEG 2025.

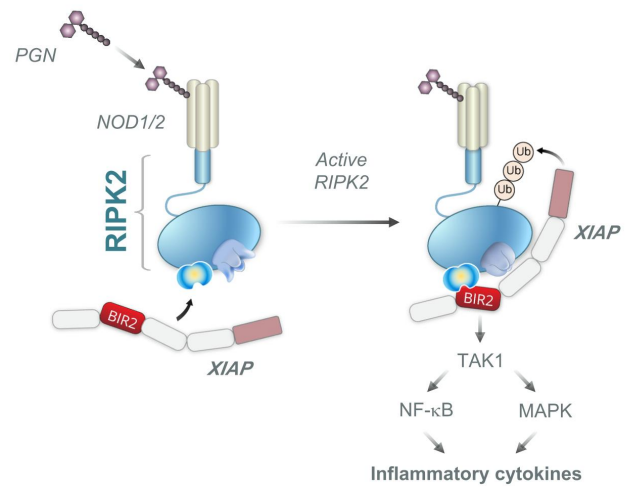
Copyright 2026 | Odyssey Therapeutics | All Rights Reserved

In search of extraordinary for patients

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
- ✓ Inhibiting RIPK2:XIAP scaffolding blocks multiple proinflammatory cytokines
- ✓ RIPK2 involvement in IBD is recapitulated in animal models
- ✓ RIPK2 knock-out mice were developmentally normal and protected from IBD

RIPK2 activation leads to scaffolding with XIAP and inflammatory signaling



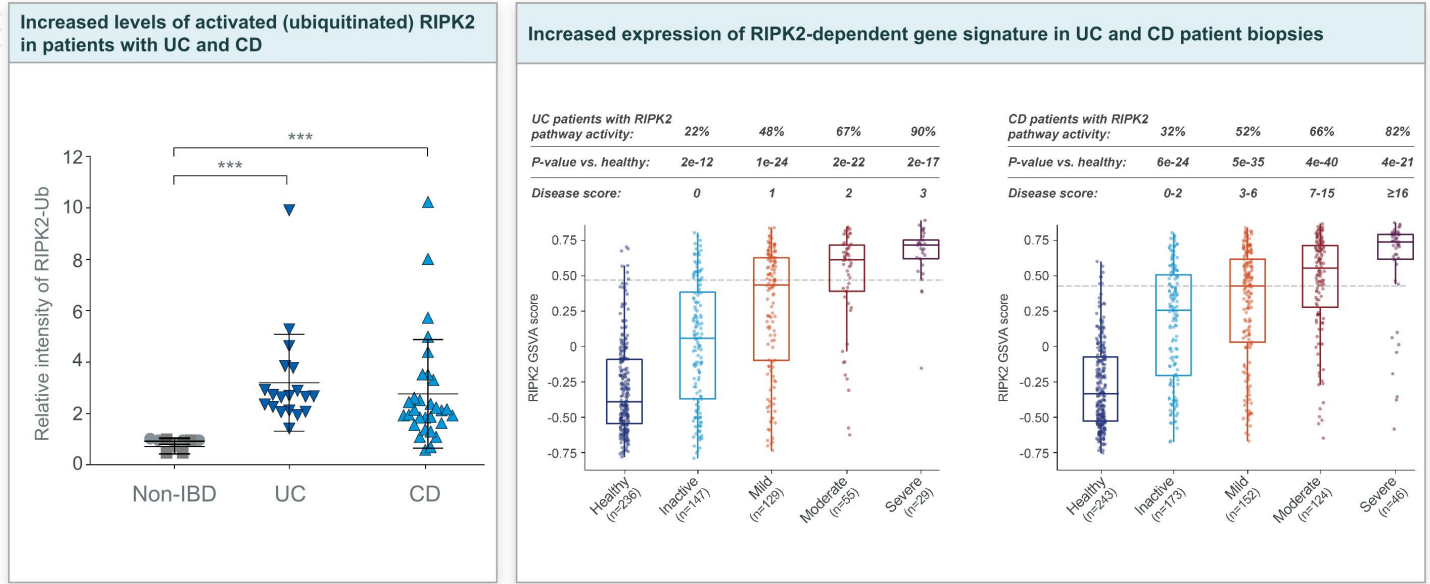
Sources: Inflamm Bowel Dis 2015;21(4):862; J Med Chem 2022;65(13):9312; J Med Chem 2019;62(14):6482; Proc Natl Acad Sci 2014;111(25):E2559; Am J Physiol 2021;321(5):G500; Commun Biol 2020;3(1):140; J Med Chem 2016;59(10):4867; Int Immunol 2019;31(10):669; United Eur Gastroenterol J 2023;12(1):103; Front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

Copyright 2026 | Odyssey Therapeutics | All Rights Reserved

In search of extraordinary for patients

12

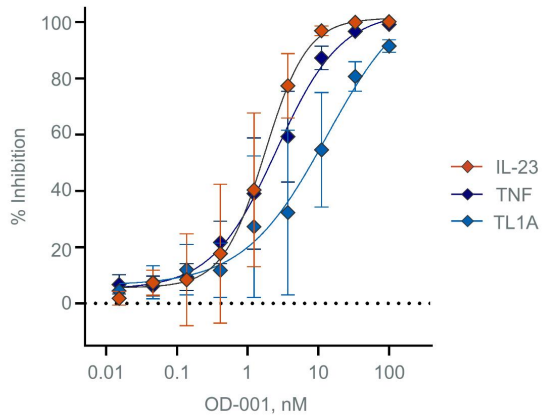
RIPK2 Is Activated in UC and Crohn's Disease and Correlates With Severity



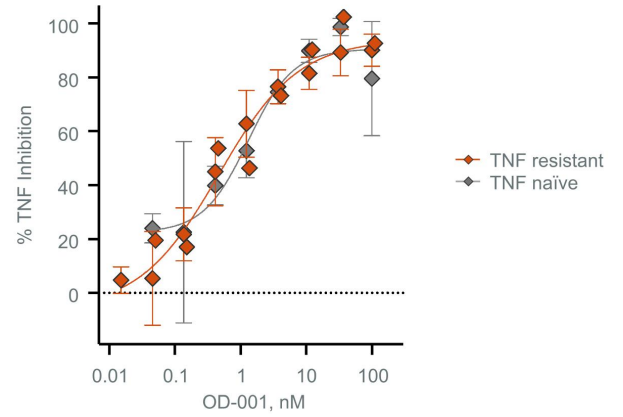
Notes: *** = $p \leq 0.0001$; GSVAscore = gene set variation analysis; GSVAscore cut-off for % of UC or CD patients with RIPK2 pathway activity is 0.46 or 0.42, respectively. Sources: EMBO J 2018;37(17):e99372; Sci Signal 2024;17(819):eabn1101; Gene expression datasets utilized = GSE193677.

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

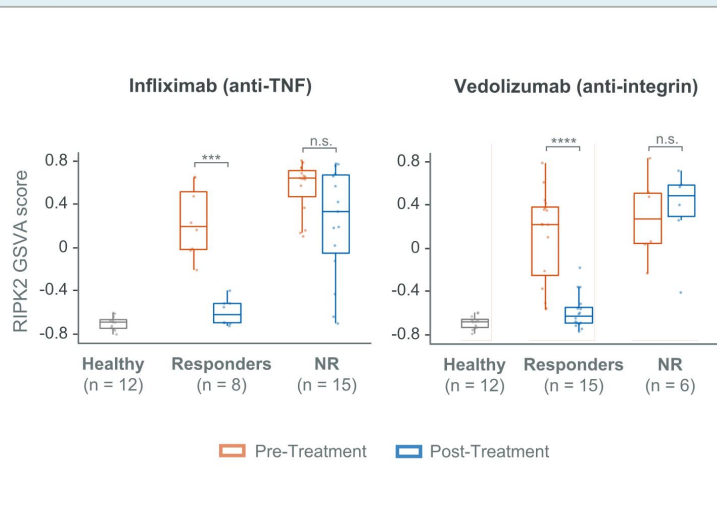


Lamina propria mononuclear cells (LPMCs) were isolated from patient colon tissue

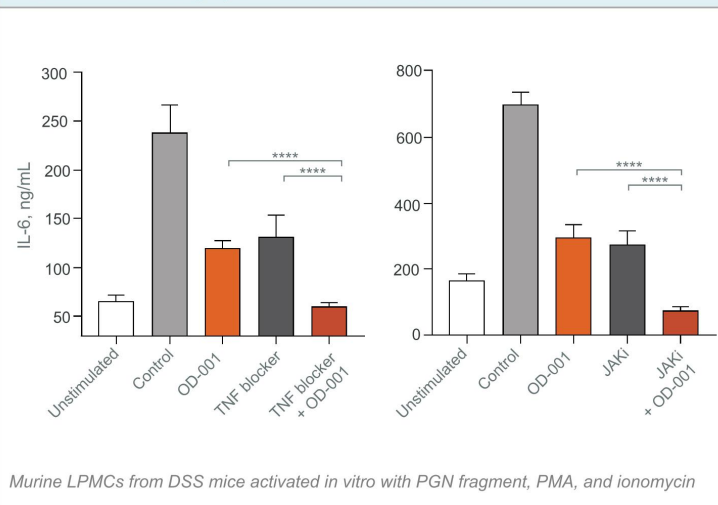


OD-001 Has The Potential To Improve Outcomes By Overcoming Resistance To Standard of Care or Use as a Combination Partner

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



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

Copyright 2026 | Odyssey Therapeutics | All Rights Reserved

In search of extraordinary for patients

15

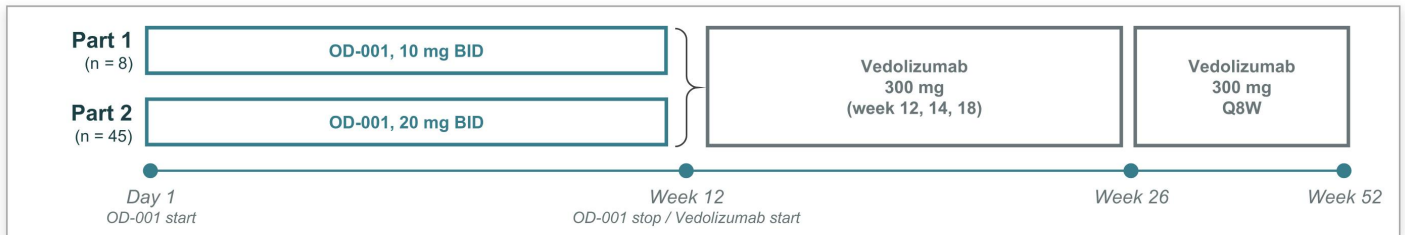
Phase 2a Monotherapy Trial Overview

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: $\geq 15\%$ 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%)

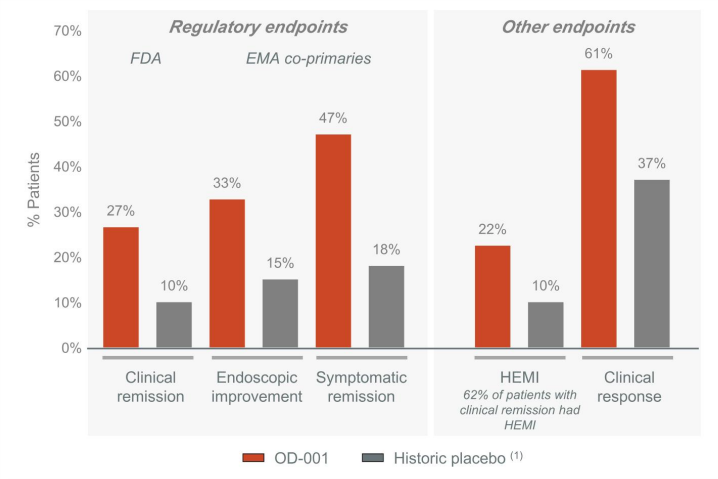
Disclaimer: Data presented are from ongoing studies, which will not be finalized until database lock. Thus, these data are subject to change prior to release of the clinical study report ("CSR").
 Notes: (1) Three participants in part 2 discontinued treatment early (between weeks 1 and 3) for reasons 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.



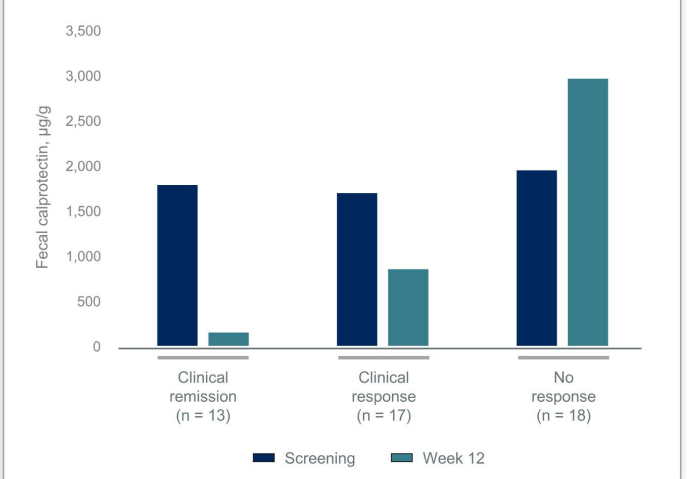
Trial Met Key Efficacy Endpoints, Including Clinical Remission

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)



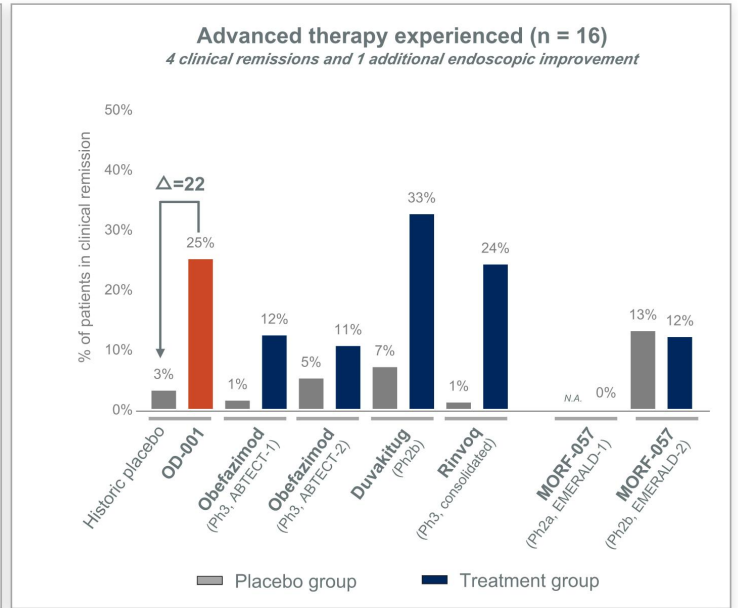
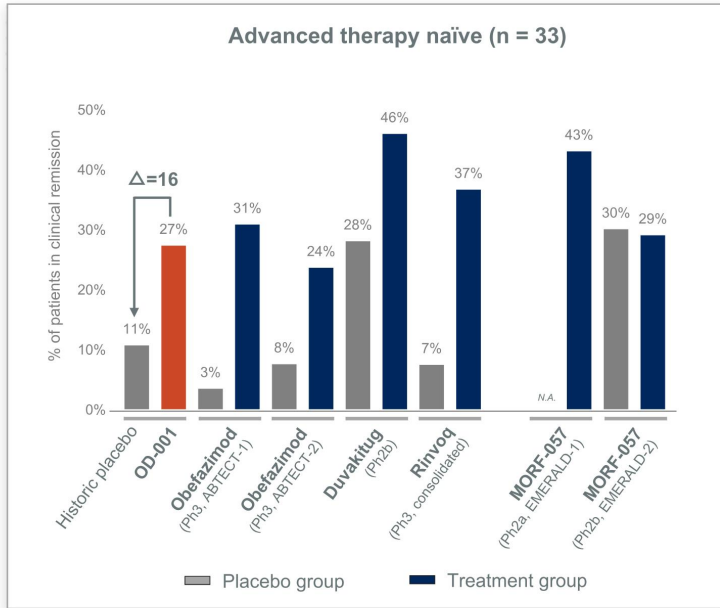
Median fecal calprotectin by week 12 outcome



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

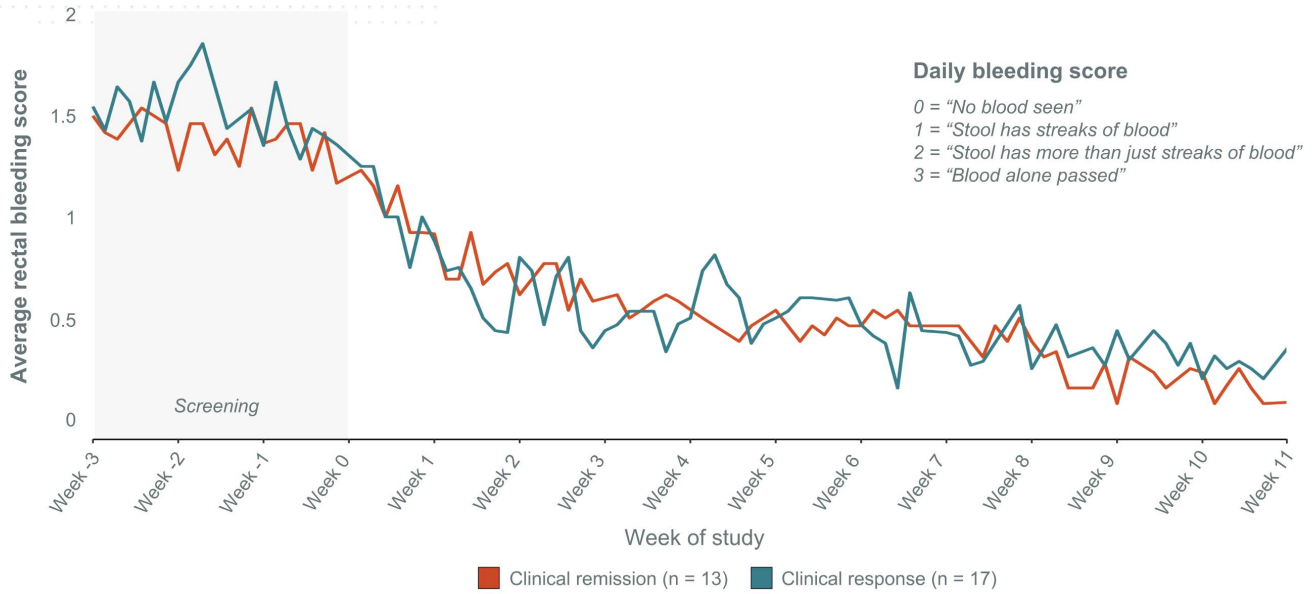


Disclaimer: Data presented are from ongoing trial, which will not be finalized until database lock. Thus, these data are subject to change prior to release of the CSR. Future results may differ from those shown here.
 Notes: OD-001 results are consolidated from both part 1 and part 2. In trials where 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

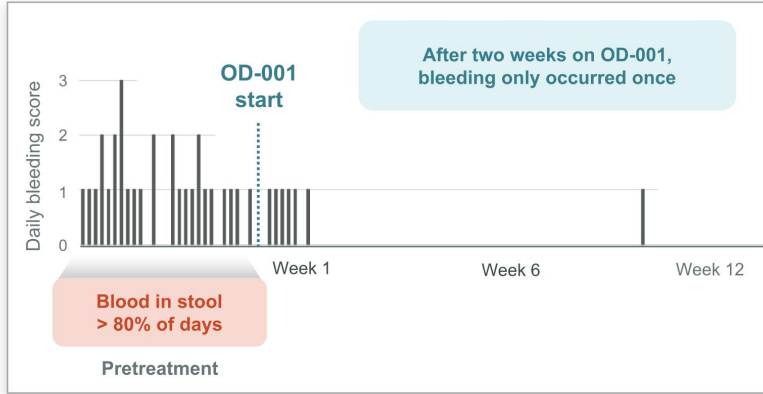
Average rectal bleeding score over treatment



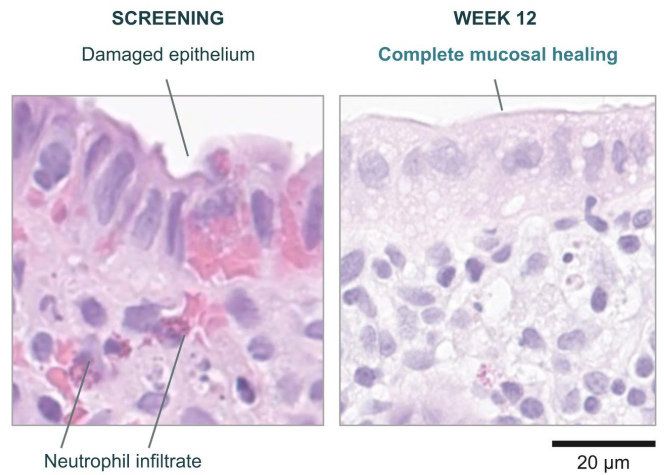
Disclaimer: Data presented are from ongoing studies, which will not be finalized until database lock. Thus, these data are subject to change prior to release of the CSR.
 Note: OD-001 results are consolidated from both part 1 and part 2.

Patient Vignette 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



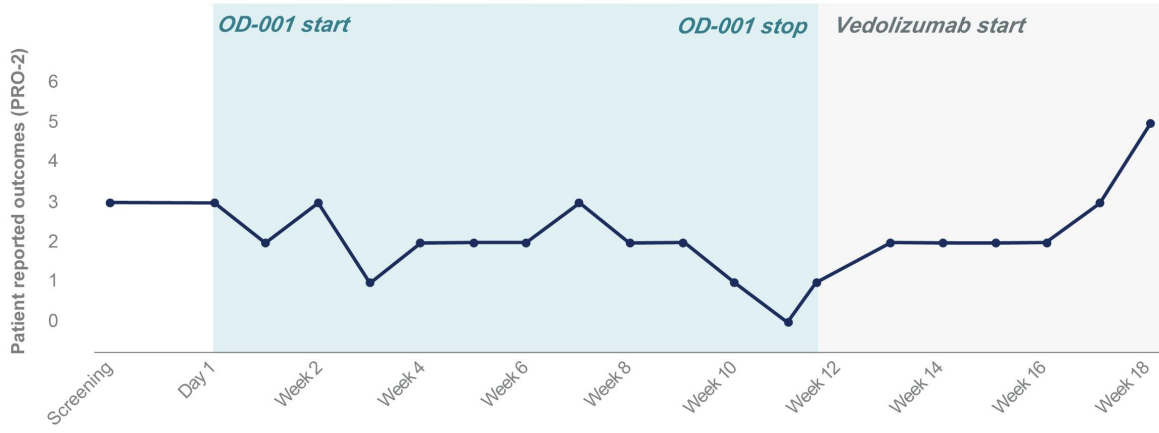
Disclaimer: (1) While we believe the observations derived from this patient's results are informative, the results depicted are preliminary results from a single patient and may not be replicated in other patients or predictive of outcomes in clinical trials for OD-001.

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.

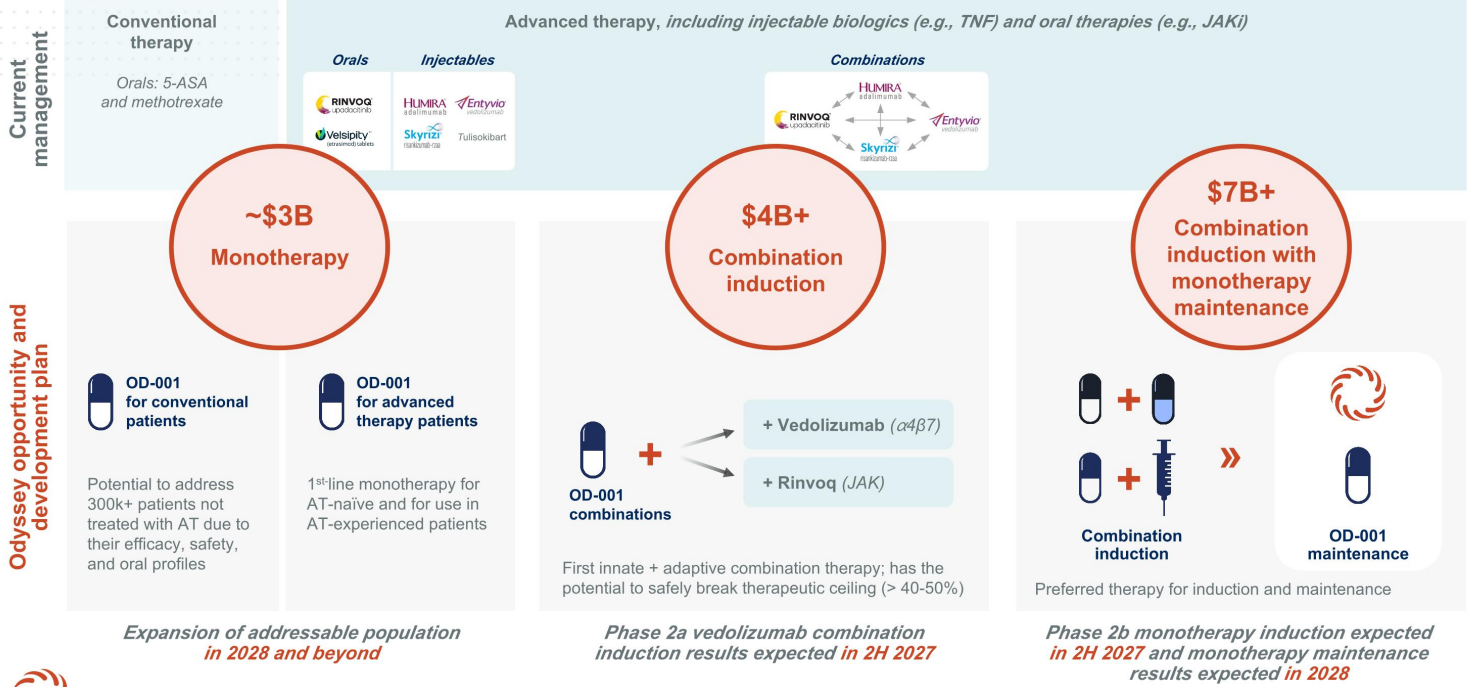


OD-001 Is the First Innate Immune-Targeted Therapy for IBD

- 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



Oral SLC15A4 Inhibitor: OD-002

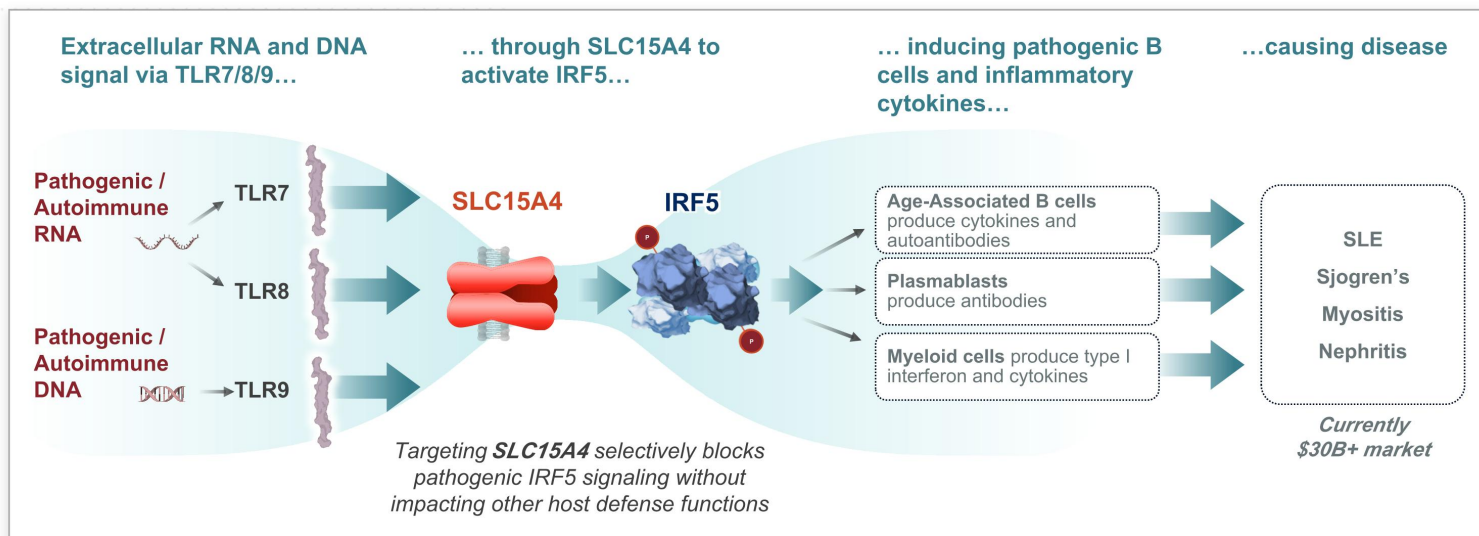
*Modulating B Cell Activity
and Interferon Signaling*

*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.

Copyright 2026 | Odyssey Therapeutics | All Rights Reserved

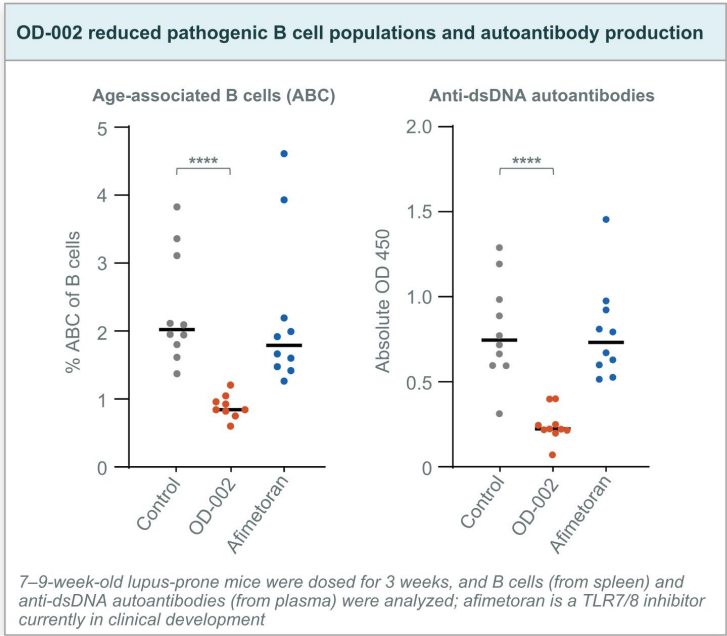
In search of extraordinary for patients

27

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



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



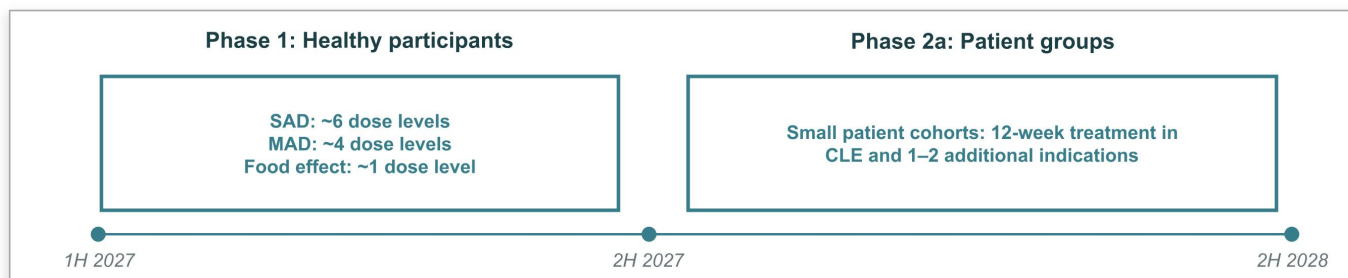
Phase 1/2a Trial Initiation Expected in 1H 2027 With Proof-of-Concept Expected in 2H 2028

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.

Copyright 2026 | Odyssey Therapeutics | All Rights Reserved

In search of extraordinary for patients

30

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

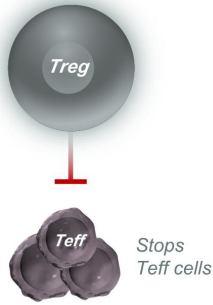
Rheumatoid arthritis

Asthma

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

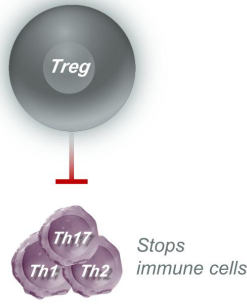
EFFECT 1:

Reduction in Teff cell proliferation and activity
Reduces direct tissue damage and immune activation



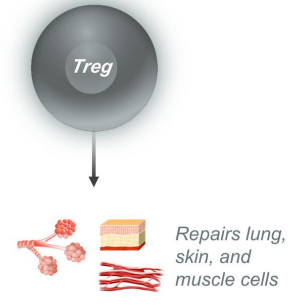
EFFECT 2:

Reduction of immune cell activity
Reduces production of proinflammatory cytokines, including IFN γ , IL-4, IL-5, and IL-13



EFFECT 3:

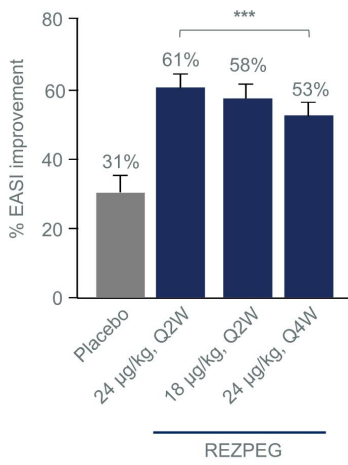
Repair of tissue-resident non-immune cells
Induces durable disease-modifying effects



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 1H 2027



NKTR-1065:
IND submission expected in 2027



OD-003:
IND-enabling studies ongoing

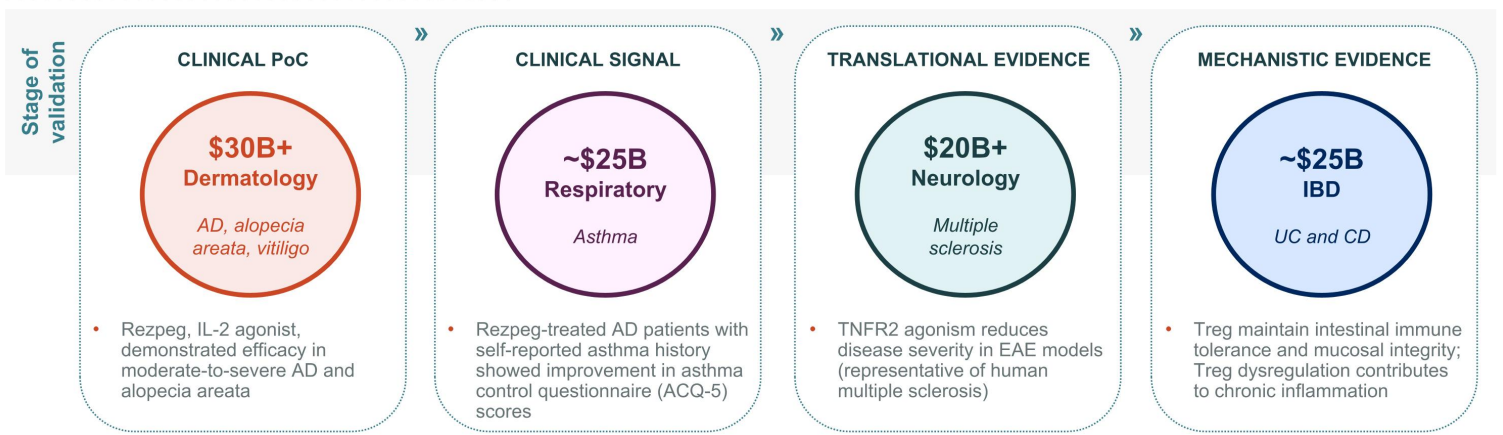


Note: *** p < 0.001.
Sources: Nektar corporate presentation, January 2026; TRexBio press releases, April 2026 and January 2026; clinicaltrials.gov.

Copyright 2026 | Odyssey Therapeutics | All Rights Reserved

In search of extraordinary for patients

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



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



Source: Evaluate Pharma.

Copyright 2026 | Odyssey Therapeutics | All Rights Reserved

In search of extraordinary for patients

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



Thank you!

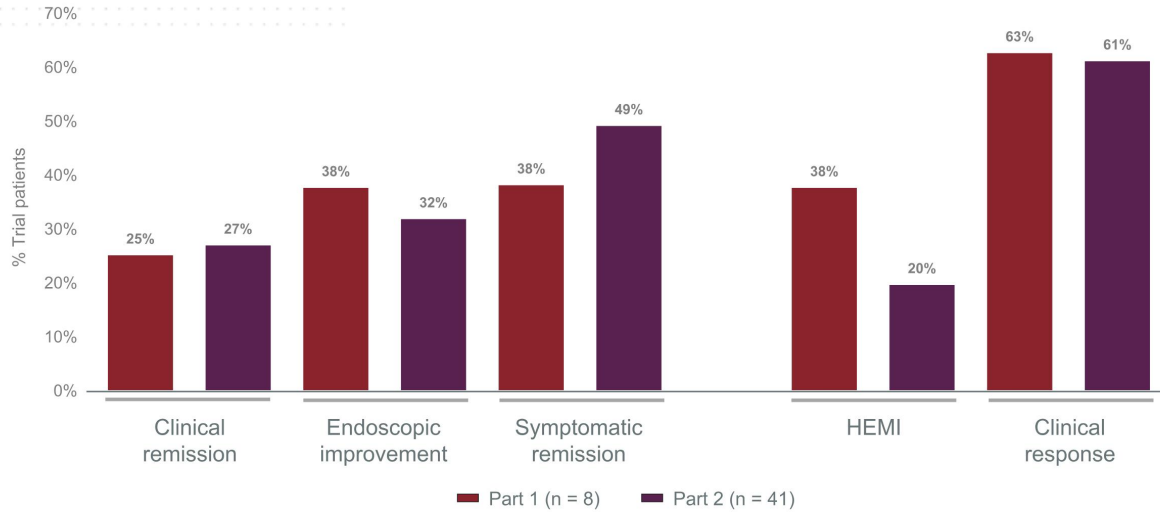
SLEEPER ST

SEAPORT

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)



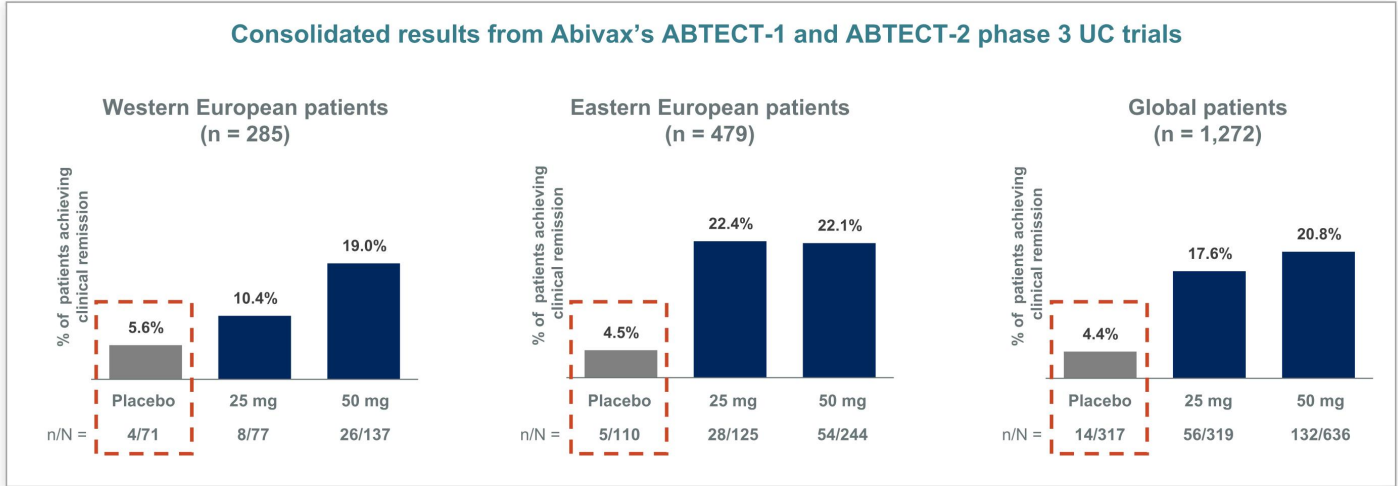
Disclaimer: Data presented are from ongoing studies, not finalized until database lock and, thus, are subject to change prior to release of the CSR. Future results may differ from those shown here.
Note: Includes all patients who completed 12 weeks of treatment per protocol (n = 49).

Copyright 2026 | Odyssey Therapeutics | All Rights Reserved

In search of extraordinary for patients

39

Placebo Clinical Remission Rates Are Generally Similar Across Regions



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

Copyright 2026 | Odyssey Therapeutics | All Rights Reserved

In search of extraordinary for patients