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): October 20, 2026

ODYSSEY THERAPEUTICS, INC.

(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-43270**	**86-3384382**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(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 October 20, 2026, Odyssey Therapeutics, Inc. (the "**Company**") issued a press release titled "Odyssey Therapeutics Presents Completed Phase 2a OD-001 Induction Results Strengthening Clinical Proof of Concept at UEG Week 2026" and posted a corporate presentation on its website at https://investors.odysseytx.com/news-events/presentations. A copy of each of the press release and presentation is furnished herewith as Exhibit 99.1 and Exhibit 99.2, respectively, 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 and Exhibit 99.2 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	Press Release issued October 20, 2026
99.2	Corporate Presentation, dated October 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: October 20, 2026

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

Odyssey Therapeutics Presents Completed Phase 2a OD-001 Induction Results Strengthening Clinical Proof of Concept at UEG Week 2026

Correlation between clinical efficacy and changes in inflammatory biomarkers, mucosal transcriptomic signatures, and histologic disease activity strengthens the previously shown clinical proof of concept for OD-001

Favorable tolerability profile and oral administration underscore the potential of OD-001 as a monotherapy and backbone for combination regimens in inflammatory bowel disease

Company to host conference call and webcast at 8:00 a.m. EDT

BOSTON, October 20, 2026 (GLOBE NEWSWIRE) — Odyssey Therapeutics, Inc. (Nasdaq: ODTX) ("Odyssey" or the "Company"), a clinical-stage biopharmaceutical company seeking to transform the standard of care for patients suffering from autoimmune and inflammatory diseases by developing medicines that precisely target disease pathology, today announced results from the completed induction period of its Phase 2a clinical trial evaluating OD-001, an oral, selective RIPK2 scaffolding inhibitor, in patients with moderate-to-severe ulcerative colitis (UC). The results were presented earlier today at United European Gastroenterology (UEG) Week in Barcelona, Spain, and will be discussed alongside additional analyses during a conference call and webcast to be hosted by Odyssey.

The completed OD-001 induction analysis comprises all 56 efficacy-evaluable patients. The high correlation between treatment outcomes and changes in inflammatory biomarkers, mucosal transcriptomic signatures, and histologic disease activity strengthens the previously established clinical proof of concept for OD-001 and provides biological evidence consistent with RIPK2 scaffolding inhibition. Combined with Phase 1 pharmacology data, these findings support 20 mg twice daily (BID) as the projected lowest maximally efficacious dose.

“These new analyses provide compelling evidence of pharmacologic activity at 20 mg BID, including clinical efficacy in patients with severe endoscopic disease and improvements across markers of intestinal and systemic inflammation,” said Gary D. Glick, Ph.D., founder and chief executive officer of Odyssey Therapeutics. “Transcriptomic findings that align with clinical activity and are consistent with RIPK2 scaffolding inhibition reinforce the mechanistic rationale for this approach in inflammatory bowel disease, strengthen our confidence in the treatment activity of OD-001, and support its continued clinical development.”

Key highlights from the induction period data

- **Induction (Week 12) efficacy:** OD-001 met the trial’s primary endpoint of change from baseline in modified Mayo score at the end of induction at Week 12 (mean reduction of 2.4 points across the evaluable population; $p<0.001$). At this time point, clinical remission, clinical response, and symptomatic remission were 27%, 61%, and 49%, respectively, in the pooled 20 mg BID group (n=41) and 20%, 40%, and 27%, respectively, at 10 mg BID (n=15).
- **Improvement in validated biomarkers of intestinal and systemic inflammation:** In patients treated with 20 mg BID, fecal calprotectin trended lower, hsCRP and circulating neutrophils were significantly decreased, and serum albumin significantly increased—all changes consistent with reduced intestinal and systemic inflammation. The reduction in neutrophils, a RIPK2-expressing cell type implicated in IBD pathogenesis, provides additional mechanistic support.
- **Rapid onset and deepening symptomatic benefit:** Symptomatic remission at 20 mg BID reached 21% at Week 2, increasing throughout the induction period with no plateau observed through Week 12.
- **Comparable efficacy across prior treatment exposure:** After 12 weeks of treatment with OD-001, clinical remission at 20 mg BID was 29% in advanced therapy-naïve (n=28) and 23% in advanced therapy-experienced (n=13) patients.
- **Clinical activity in patients with most severe endoscopic disease:** Clinical remission at Week 12 for patients taking OD-001 at 20 mg BID was 16% in patients with a Mayo Endoscopic Score (MES) of 3 (n=19).

Transcriptomic analyses reinforce OD-001 treatment activity

- **On-target activity indicative of RIPK2 inhibition:** Exploratory analyses of paired colonic biopsies from baseline and Week 12 showed reductions in RIPK2-responsive and downstream inflammatory gene signatures, including IL-23, TNF/NF-κB, TL1A, AP-1 and T-cell-associated pathways, supporting the proposed mechanism of action.
- **Modulation of fibrosis-associated and epithelial gene signatures:** Reductions in fibrosis-associated signatures and increased expression of genes linked to epithelial homeostasis and mucosal healing aligned with clinical outcomes.
- **Distinct pharmacology relative to approved mechanisms:** Exploratory patient-level comparisons with published datasets of approved therapies suggest OD-001 modulates RIPK2-associated pathways beyond general mucosal improvement achieved with other agents, supporting a differentiated mechanism of action and pharmacodynamic profile.

Safety and tolerability

OD-001 was generally well tolerated during the 12-week induction period, with no serious adverse events, serious infections, treatment-related discontinuations, or treatment-emergent liver function abnormalities. As previously reported, three adverse events in the 20 mg BID group were considered drug-related (two fevers and one headache). One Grade 3 anemia event in the 10 mg BID group occurred in a patient with pre-existing anemia, was considered unrelated to treatment, and resolved with iron supplementation without discontinuing OD-001.

Phase 2a clinical trial overview

The Phase 2a trial (NCT06850727) evaluated the efficacy, safety and tolerability of oral OD-001 in patients with moderate-to-severe ulcerative colitis using an open-label, dose-ranging design. The 12-week induction period included 10 mg and 20 mg BID dosing, with change from baseline in the three-component modified Mayo score as the primary endpoint. This open-label trial had no concurrent placebo control, and the data remain subject to change until database lock.

OD-001 advancing into Phase 2b monotherapy and Phase 2a combination trials

Odyssey plans to advance OD-001 into a randomized, placebo-controlled Phase 2b monotherapy trial and a randomized, active-controlled Phase 2a trial evaluating OD-001 plus vedolizumab versus vedolizumab plus placebo. Both studies will be double-blind, with topline induction results expected in the second half of 2027.

Conference call and webcast

Odyssey will host a conference call and webcast today at 8:00 a.m. EDT to discuss the completed Phase 2a induction results, including additional analyses beyond those presented at UEG. The live webcast and accompanying presentation will be available through the Investors section of Odyssey Therapeutics' website at www.odysseytx.com. A replay will be available following the event.

About Odyssey Therapeutics

Odyssey Therapeutics is a clinical-stage biopharmaceutical company seeking to transform the standard of care for patients suffering from autoimmune and inflammatory diseases by developing medicines that are designed to precisely target disease pathology. Since its founding in 2021, Odyssey has built a portfolio of internally discovered and developed medicines with its first program advancing through multiple clinical milestones. Odyssey's portfolio leverages the scientific expertise of its team of experienced drug hunters and a comprehensive suite of tools to efficiently advance product candidates that the Company believes have the potential to induce deep and durable remission for patients across several inflammatory diseases with unmet need.

For more information, please visit http://www.odysseytx.com and follow Odyssey on LinkedIn or X.

Forward-looking statements

This press release includes certain disclosures that contain "forward-looking statements" within the meaning of the safe harbor provisions of the Private Securities Litigation Reform Act of 1995, including, without limitation, statements regarding the interpretation and potential implications of OD-001's Phase 2a clinical, safety and exploratory transcriptomic results;

evidence of treatment activity and RIPK2 inhibition; selection of 20 mg BID for further development; OD-001's potential efficacy, safety and use as an oral monotherapy, in combination with vedolizumab or for maintenance treatment; and the design, timing and anticipated results of planned Phase 2b monotherapy and Phase 2a combination trials, including expected topline induction results in the second half of 2027.

Forward-looking statements may be identified by words such as "anticipate," "believe," "could," "expect," "intend," "goal," "may," "plan," "potential," "will," "would" and variations of these words or similar expressions, although not all forward-looking statements contain these identifying words. Forward-looking statements reflect Odyssey's current expectations and involve risks and uncertainties that could cause actual results to differ materially. These include the inherent limitations of an open-label trial without a concurrent placebo control; potential uncertainty about exploratory transcriptomic findings; the possibility of different outcomes in future trials; clinical, regulatory, competitive and third-party risks; and other factors described under "Risk Factors" in Odyssey's most recent Form 10-Q and other SEC filings. Forward-looking statements contained in this press release are made as of this date, and Odyssey undertakes no obligation to update these statements except as required by law.

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For media
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ODYSSEY THERAPEUTICS

Corporate Presentation

October 2026



Disclaimers

Forward-looking statements and third-party information

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.

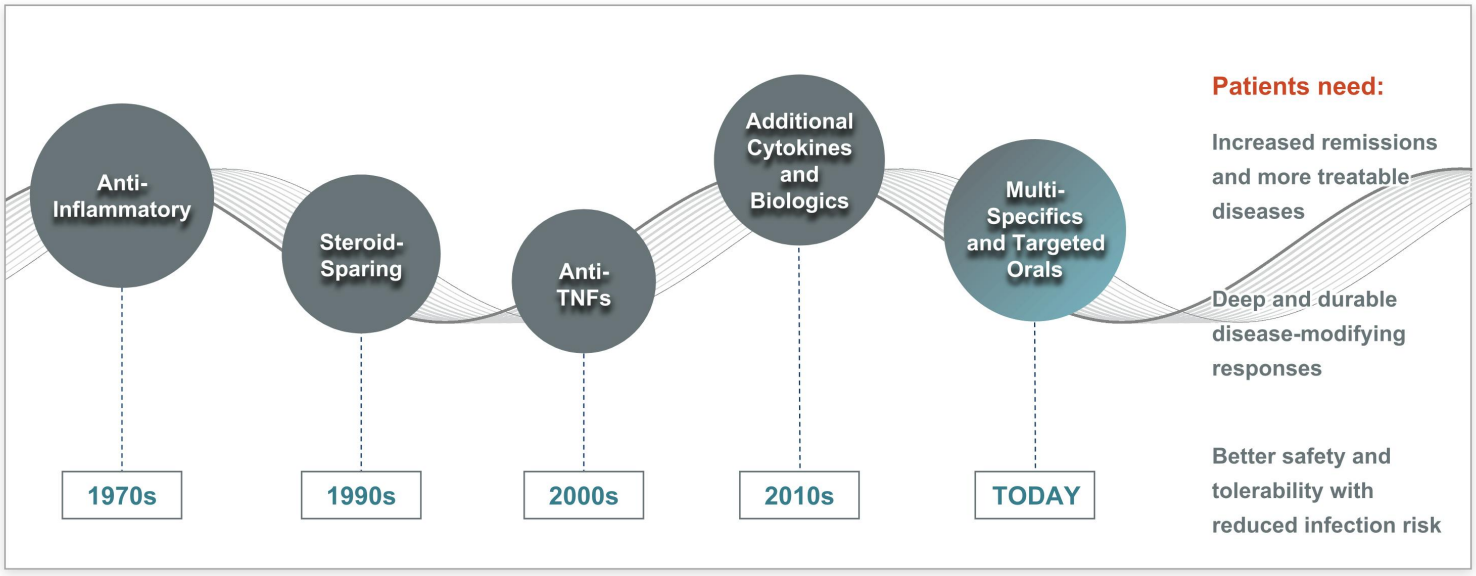
Certain information contained in this presentation and statements made orally during this presentation may relate to or be based on studies, publications, estimates, projections, and other data obtained from third-party sources as well as our own internal estimates and research. While we believe these 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.

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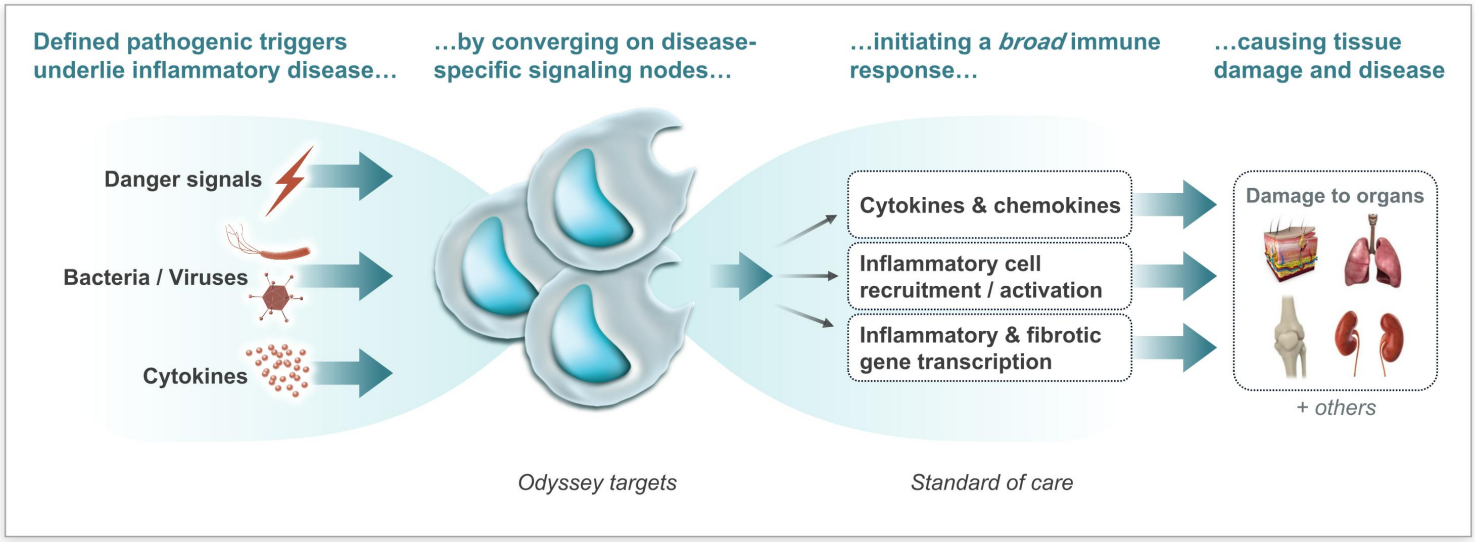
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Few Patients With Autoimmune Disease Have Durable Remission



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Targeting Upstream Signaling Has Potential to Unlock Durable Remissions



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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, CEO, and Director

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

Joe McDonald, Ph.D.
Chief Data Officer

Jason Haas
Chief Financial Officer

Natalie Dales, Ph.D.
Research Operations

Jolie Siegel
EVP, General Counsel, and Secretary

Collin Todd
SVP, Strategy and Business Development

BOARD OF DIRECTORS

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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.

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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

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)

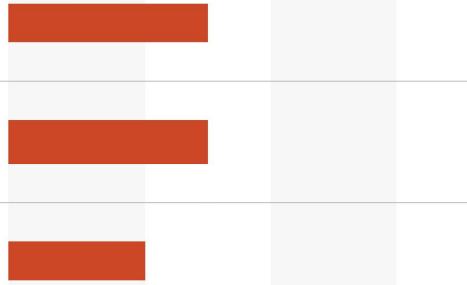
Current market: ***$100B***

Source: Evaluate Pharma.

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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*						**H2 2027:** Phase 2a combination readout **H2 2027:** Phase 2b monotherapy readout
SLC15A4, OD-002 *Small molecule*	*CLE, nephropathies, and B cell-mediated diseases*						**H2 2026:** CTA filing **H1 2027:** Phase 1/2 initiation
TNFR2, OD-003 *Protein*	*AD, SLE, alopecia areata*						**H1 2027:** IND-enabling study completion
TSLP/IL-33, OD-004 *Protein*	*COPD, asthma*						



$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

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Oral RIPK2 Scaffolding Inhibitor: OD-001

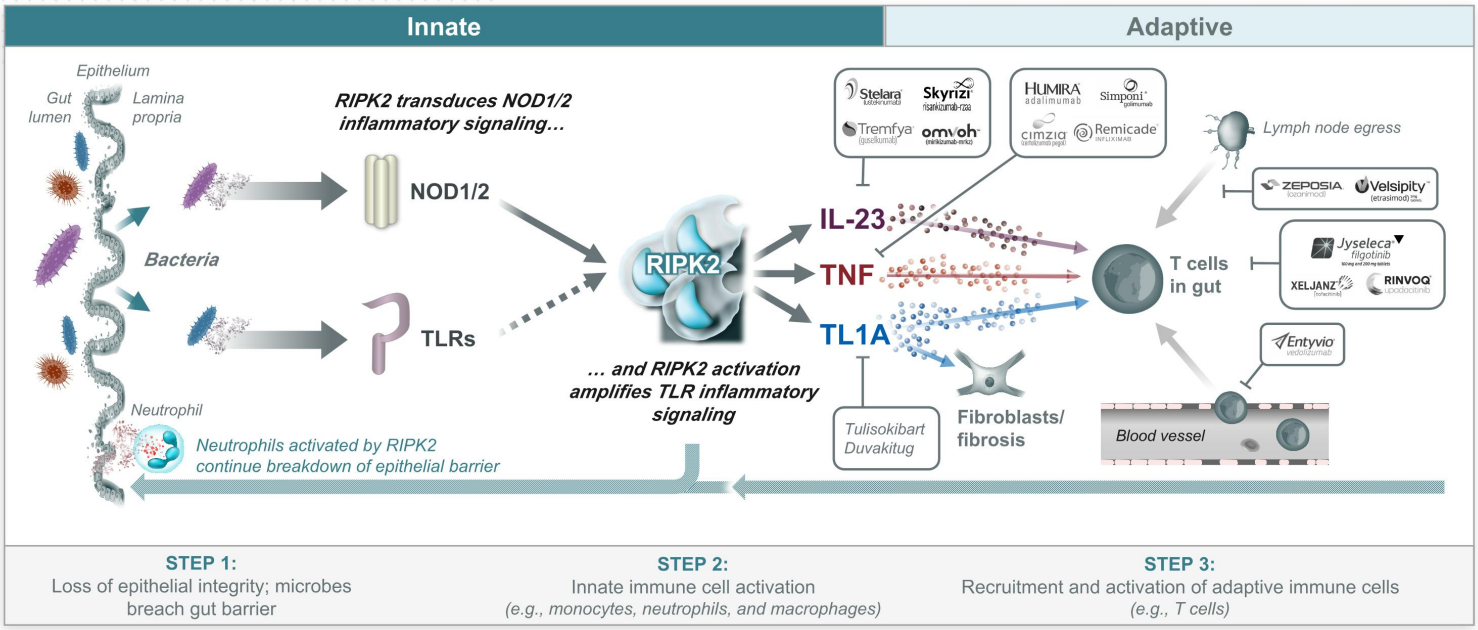
Addressing a Root Cause of Inflammation in IBD Using a Novel Mechanism of Action

Proof-of-Concept Achieved in Ulcerative Colitis

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

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



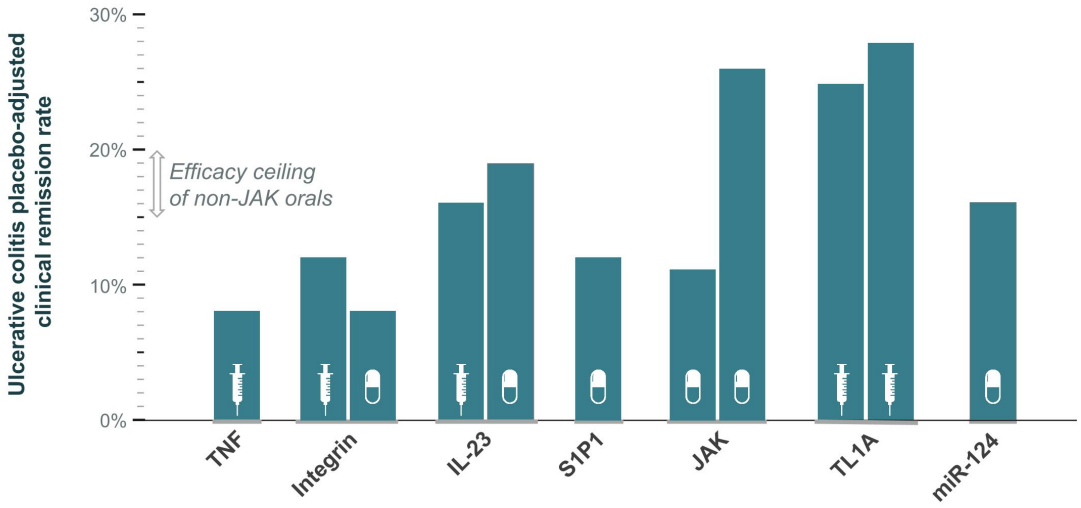
STEP 1:
Loss of epithelial integrity; microbes breach gut barrier

STEP 2:
Innate immune cell activation
(e.g., monocytes, neutrophils, and macrophages)

STEP 3:
Recruitment and activation of adaptive immune cells
(e.g., T cells)

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



~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.

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RIPK2 Is a Validated Target to Treat IBD

Animal data

- Microbes are required for spontaneous colitis in rodent models
- RIPK2 knockout reduces inflammatory cytokines and improves intestinal histology in animal models of IBD
- Blocking RIPK2 signaling reduces inflammatory cytokines and intestinal disease in mouse models
- RIPK2 knockout mice are developmentally normal

Human data

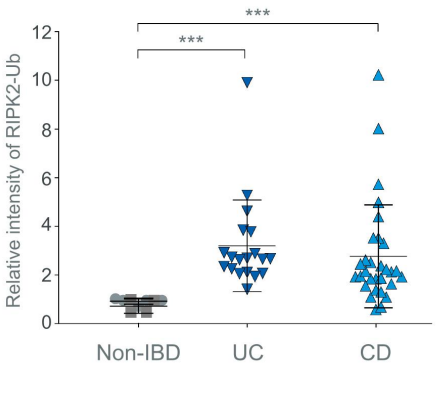
- RIPK2 pathway activation is increased in intestinal tissue from patients with both UC and CD
- RIPK2 ubiquitination is observed across all UC patients, including those with NOD2 LoF variants
- **Genetic validation** of RIPK2*: ATG16L1* and *CARD9* variants are linked to increased IBD risk; protective *CARD9* variants to reduced risk
- **Pharmacologic validation** of RIPK2: Clinical efficacy with OD-001 provides target validation in UC

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; Mucosal Immunol 2017;10(4):971; J Biol Chem 2014;289(43):29651; Commun Biol 2020;3(1):140; J Med Chem 2016;59(10):4867; Int Immunol 2019;31(10):669; Sci Signal 2024;17(819):eabn1101; Immunity 2013;39(5):858; Gut 2011;60(9):1229; Nat Immunol 2007;8(2):198; Nature 2012;491(7422):119; United Eur Gastroenterol J 2023;12(1):103; Front Immunol 2019;10:419.

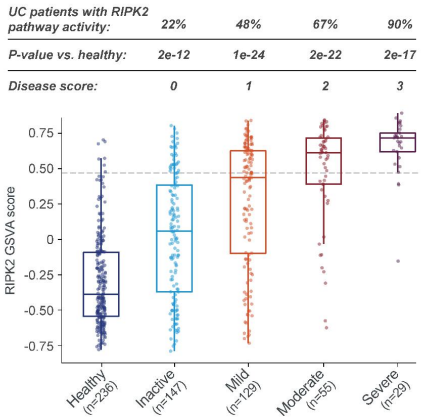
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RIPK2 Is Activated in UC and CD and Correlates With Severity

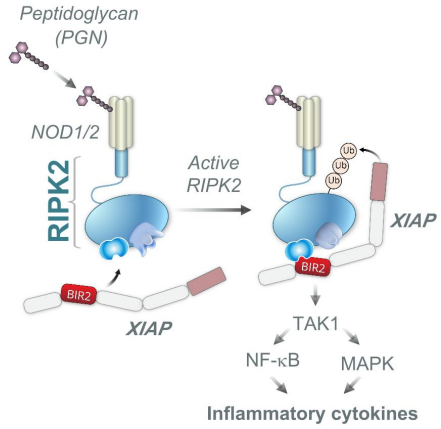
Increased levels of activated (ubiquitinated) RIPK2 in patients with UC and CD



Increased expression of RIPK2-responsive gene signature in IBD patient biopsies



RIPK2 scaffolding with XIAP is necessary for activation and inflammatory signaling



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

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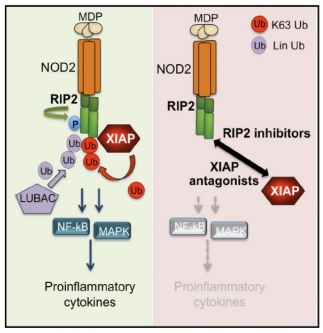
RIPK2 Drugs Must Block Scaffolding to Fully Inhibit NOD2 Signaling

Article

Molecular Cell

Disruption of XIAP-RIP2 Association Blocks NOD2-Mediated Inflammatory Signaling

Graphical Abstract



Authors

Tatiana Goncharov, Stefanie Hedayati, Melinda M. Mulvihill, ..., Wayne J. Fairbrother, Kurt Deshayes, Domagoj Vucic

Correspondence

domagoj@gene.com

In Brief

Goncharov et al. demonstrate that the kinase activity of RIP2 is dispensable for NOD2 signaling. Instead, effective RIP2 kinase inhibitors block NOD2 signaling by disrupting RIP2-XIAP interaction in a similar fashion as XIAP antagonists. These findings provide a novel therapeutic strategy for targeting the NOD2 pathway.

Mol Cell 2018;69(4):551–565

Article

SOURCE DATA | TRANSPARENT PROCESS | OPEN ACCESS

THE EMBO JOURNAL

Small molecule inhibitors reveal an indispensable scaffolding role of RIPK 2 in NOD2 signaling

Matous Hrdinka[1,†,‡], Lisa Schlicher[1,†], Bing Dai[2], Daniel M Pinkas[3], Joshua C Bufton[3,§], Sarah Picaud[3], Jennifer A Ward[3,4], Catherine Rogers[3,4], Chalada Suebsuwong[5,¶], Sameer Nikhar[6], Gregory D Cuny[5], Kilian VM Huber[3,4], Panagis Filippakopoulos[3], Alex N Bullock[3], Alexei Degterev[2,*] & Mads Gyrd-Hansen[1,**]

Abstract

RIPK2 mediates inflammatory signaling by the bacteria-sensing receptors NOD 1 and NOD2. Kinase inhibitors targeting RIPK 2 are a proposed strategy to ameliorate NOD-mediated pathologies. Here, we reveal that RIPK 2 kinase activity is dispensable for NOD 2 inflammatory signaling and show that RIPK 2 inhibitors function instead by antagonizing XIAP-binding and XIAP-mediated ubiquitination of RIPK 2. We map the XIAP binding site on RIPK 2 to the loop between b2 and b3 of the N-lobe of the kinase, which is in close proximity to the ATP-binding pocket. Through characterization of a new series of ATP pocket-binding RIPK 2 inhibitors, we identify the molecular features that determine their inhibition of both the RIPK2-XIAP interaction, and of cellular and in vivo NOD2 signaling. Our study exemplifies how targeting of the ATP-binding pocket in RIPK2 can be exploited to interfere with the RIPK 2-XIAP interaction for modulation of NOD signaling.

Introduction

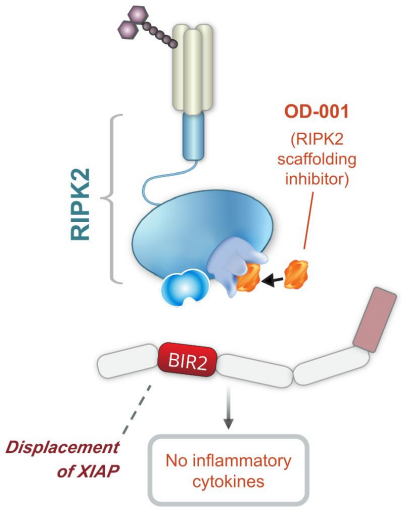
Receptor-interacting kinases (RIPKs) are components ofinnate immune receptor signaling complexes where they become ubiquitinated and contribute to NF-j B-mediated inflammatory signaling and cell death (Hrdinka & Gyrd-Hansen, 2017; Annibaldi & Meier, 2018). The intracellular bacteria-sensing receptors NOD1 and NOD2 (nucleotide-oligomerization domain-containing proteins 1 and 2) stimulate inflammatory signaling by promoting RIPK2 ubiquitination in response to binding ofiE-DAP (D-glutamyl-meso-diaminopimelic acid) and MDP (muramyl dipeptide) constituents of bacterial peptidoglycan, respectively (Girardin et al, 2003). NOD1/2 signaling contributes to gastro-intestinal immunity (Philpott et al, 2014), and genetic variants in NOD2 are the strongest susceptibility factors to Crohn's disease —one of the two major inflammatory bowel diseases afflicting millions in Europe and North America alone (Hugot et al, 2001; Ogura et al, 2001; Ananthakrishnan, 2015). Mutations of

EMBO J 2018;37(17):e99372

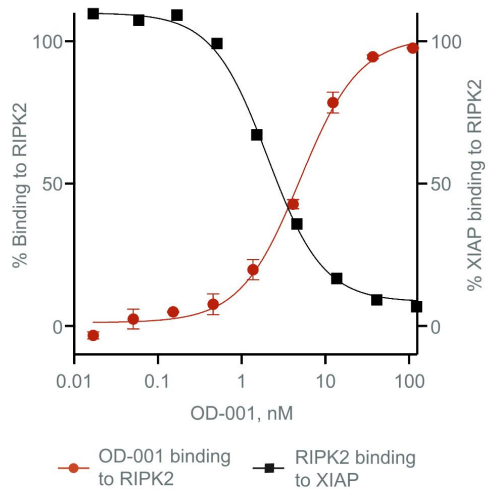
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Blocking RIPK2 Scaffolding With OD-001 Achieves Maximal Inhibition

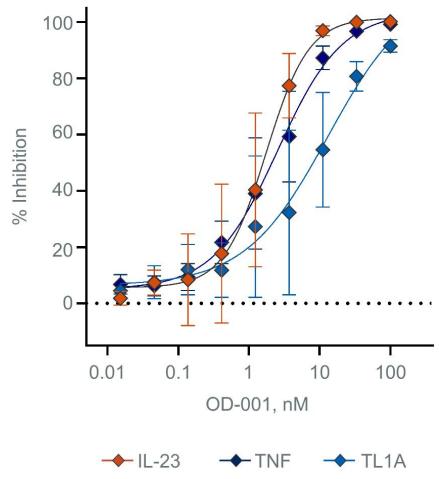
OD-001 is an allosteric scaffolding inhibitor that reduces the affinity for XIAP



Relationship between OD-001 and XIAP binding to RIPK2



OD-001 blocks cytokine production from macrophages stimulated with PGN

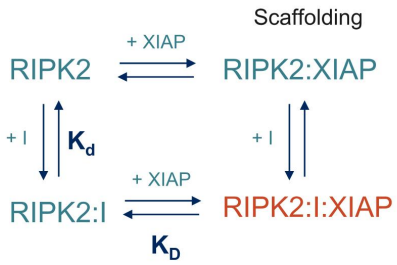


Notes: Binding to RIPK2 was measured by competition with a fluorescent ATP-site tracer in cells. XIAP binding toRIPK2 using recombinant protein was measured by HTRF. Cytokine production was assessed after 24 hours by MSD/ELISA in macrophages treated with compound for 1 hour and then stimulated with PGN.
Sources: EMBO J 2018;37(17):e99372; Mol Cell 2018;69(4):551–565.

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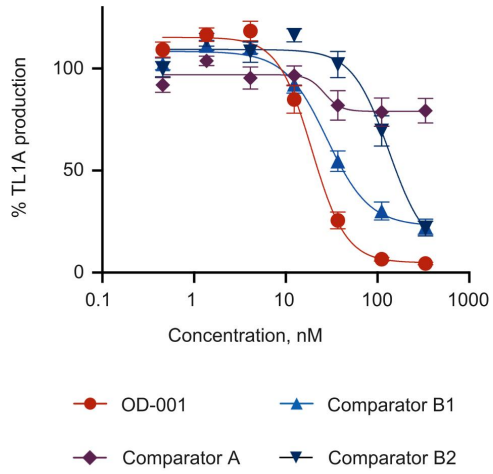
Not All RIPK2 Scaffolding Inhibitors Are the Same

Pharmacology of noncompetitive allosteric inhibitors



- Inhibitor and XIAP can bind simultaneously to RIPK2 to form a ternary complex
- Inhibitor binding to RIPK2 potentially alters the affinity for XIAP
- Extent of scaffolding inhibition depends on K_D, *which can vary depending on XIAP concentration*

OD-001 blocks TL1A production in response to microbial stimulation (PGN) in human macrophages



Notes: 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 Example A6; Comparator B1 WO2024254539 Example 253; Comparator B2 WO2024254539 Example 52.

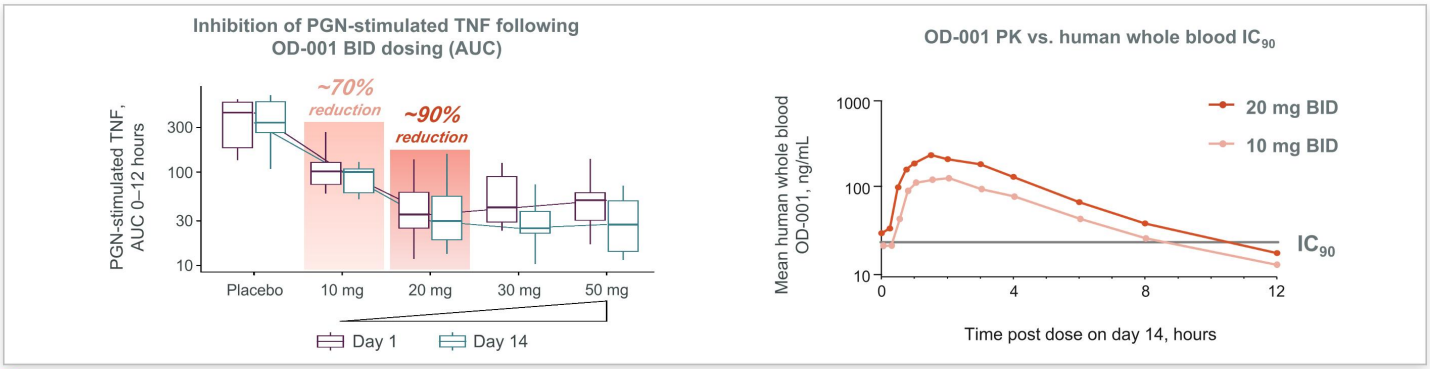
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Phase 2a Monotherapy Trial Results

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Rationale and Dose Selection for Proof-of-Concept Phase 2a Trial

- OD-001 is the first *innate immune-targeted therapy* and the first *RIPK2 scaffolding inhibitor* to enter UC clinical development
- Trial was designed to assess safety and efficacy of OD-001 and determine whether to advance the program
  - Partnered with Alimentiv, a leading IBD-specialized CRO with deep investigator experience
  - Primary endpoint: Change from baseline in 3-component mMS at week 12 (90% power at a two-sided 5% significance to detect a 2-point change)
  - Secondary endpoint: Remission rate (≥ 15% placebo-adjusted compared with a historic placebo rate of 10%; 82% power at a two-sided 10% significance)
- Doses selected based on an ex vivo blood assay in the phase 1 trial; 20 mg BID is the lowest dose projected to be maximally effective
  - Level of inhibition with 20 mg BID approximates that observed with RIPK2 knockout cells

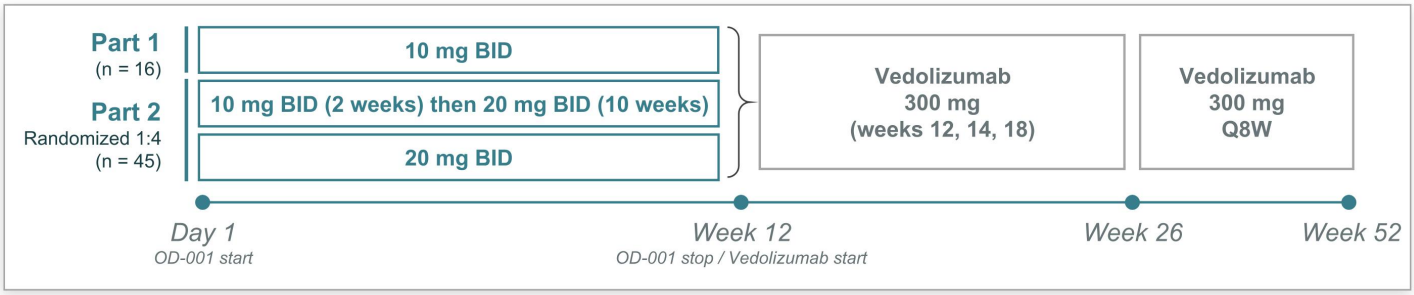


Notes: 3-component modified Mayo Clinical score (mMS) includes endoscopic severity (0–3), stool frequency (0–3), and rectal bleeding (0–3).

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Phase 2a Monotherapy Trial Overview

- Patients with moderately to severely active UC and an mMS of 5–9 were enrolled from 18 sites across seven countries; up to three prior advanced therapies were permitted
- Dosing strategy:
  - Part 1: Initial evaluation at 10 mg BID to ensure safety in UC patients with submaximal RIPK2 inhibition before testing a higher dose with maximal inhibition
  - Part 2: Evaluated 20 mg BID to assess safety and efficacy of maximal RIPK2 inhibition; included a subgroup with step-up dosing from 10 to 20 mg BID
- Design features to minimize placebo effect:
  - Required Mayo endoscopic subscore (MES) ≥ 2 and rectal bleeding subscore ≥ 1
  - Centralized and blinded endoscopy reads
  - Limited concomitant steroid dose to maximum of 20 mg prednisone



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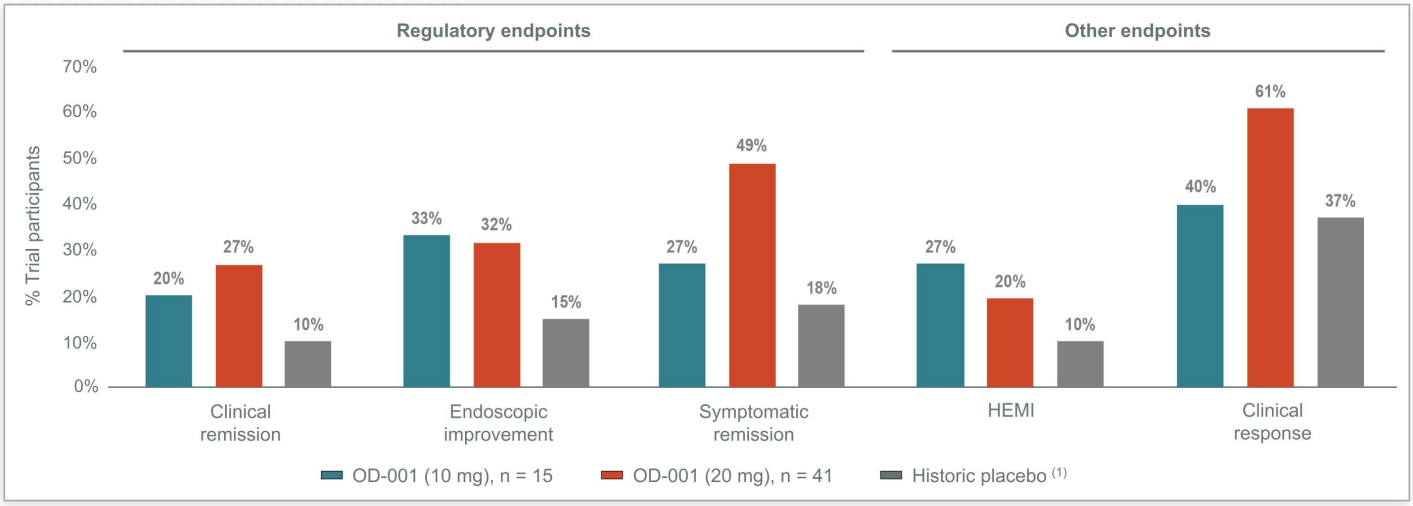
Participant Characteristics Are Similar to Other Contemporary UC Trials

	OD-001 *RIPK2*	Obefazimod *miR-124*		SPY001 *α4β7*	Icotrokinra *IL-23*	Tulisokibart *TL1A*	Duvakitug *TL1A*	Afimkibart *TL1A*
Phase	Phase 2a	Phase 2b	Phase 3	Phase 2, Part A	Phase 2b	Phase 2	Phase 2b	Phase 2b
Age, mean	40	41	42	44	42	41	41	39
Sex, male	49%	59%	-	72%	58%	53%	63%	60%
Advanced therapy-experienced (1)	30%	49%	47%	19%	43%	47%	39%	39%
Modified Mayo score, mean	6.5	7.1	6.9	6.8	6.6	7.0	6.8	7.0 (median)
MES = 3	51%	71%	60%	56%	59%	73%	56%	52%
Median fecal calprotectin, µg/g	1,752	1,647	1,666	-	1,523	1,307	-	1,511
Duration of disease, mean, years	7	8	8	5	8	7	8	7
Concomitant corticosteroid use	23%	52%	40%	42%	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; 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.

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OD-001 Efficacy Across Key Week 12 Outcomes by Dose, With Stronger Activity at 20 mg BID



Trial met primary endpoint of change in mMS from baseline in both parts and on a consolidated basis (-2.4, p < 0.001)

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 represent all patients who completed 12 weeks of treatment per protocol (n = 56); histologic-endoscopic mucosal improvement (HEMI) is defined as MES of 0 or 1 and Geboes index score ≤ 3.1. (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 total; 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 Crohns Colitis 2025;19(10):jjaf191).

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OD-001 Is Safe and Well Tolerated at 10 mg BID and 20 mg BID

Treatment-emergent adverse events	Part 1: 10 mg BID, n = 16	Part 2: 20 mg BID, n = 45
Total number of TEAEs	**11**	**40**
Severe (grade ≥ 3) TEAE	1 (anemia) [1]	0
Drug-related TEAE	0	3
Liver function abnormality identified as TEAE	0	0
Drug-related TEAEs leading to drug discontinuation	0	0
SAE	0	0
TEAEs of special interest		
Elevated body temperature that meets specific criteria	0	0
Elevated hsCRP that meets specific criteria	0	0
Serious infection	0	0
Drug-related TEAEs		
Fever	0	2
Headache	0	1

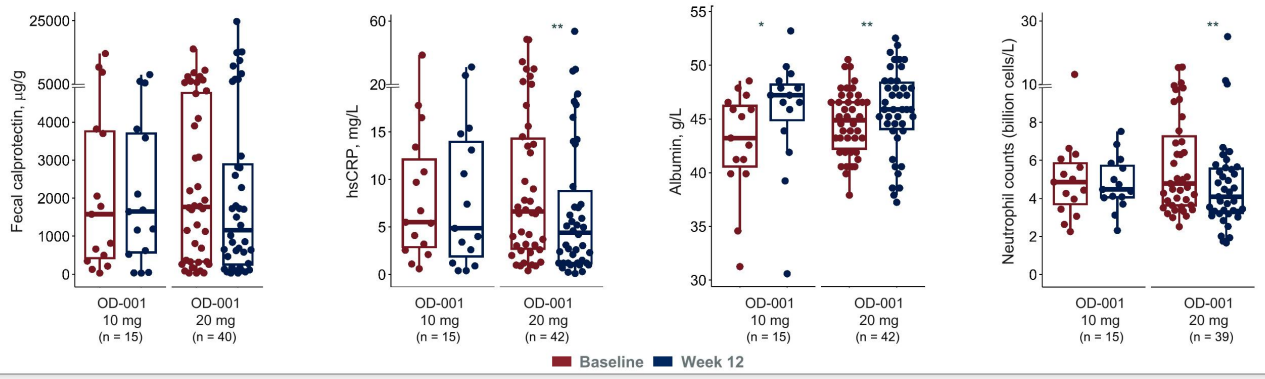
Disclaimer: Data presented are from ongoing studies, which will not be finalized until database lock. Thus, these data are subject to change prior to release of the CSR. Future results may differ from those shown here.
Notes: One participant in Part 1 and three participants in Part 2 discontinued treatment for reasons unrelated to the trial drug. One participant in Part 2 was not evaluable for efficacy. Total of 56 patients between Part 1 and Part 2 completed 12 weeks of induction treatment per protocol. (1) Patient had medical history of anemia with mild anemia at time of enrollment; OD-001 treatment was not discontinued and grade 3 anemia resolved with iron supplementation; TEAE was deemed not related to OD-001 treatment. TEAE = treatment-emergent adverse event; SAE = serious adverse event; hsCRP = high-sensitivity C-reactive protein.

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Markers of Systemic and Local Inflammation Are Reduced After OD-001 Treatment

- 20 mg BID dosing results in **reduced fecal calprotectin** across treated patients
- 20 mg BID results in **reduced serum C-reactive protein**
- 10 mg and 20 mg BID result in **increased serum albumin**
  - Serum albumin may increase due to increased hepatic synthesis as systemic inflammation declines *and* reduced rectal bleeding as the colonic mucosa heals
- 20 mg BID **reduces neutrophils**, a cell type that expresses RIPK2 and is involved in epithelial barrier breakdown in IBD

Markers of systemic and local inflammation suggest 20 mg BID inhibits colonic and systemic inflammation



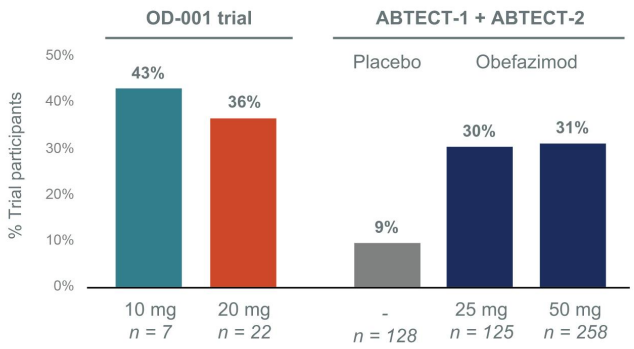
Notes: * $p < 0.05$; ** $p < 0.01$; nominal, unadjusted p values from paired Wilcoxon signed-rank tests comparing baseline and week 12 within each dose group; patients with hsCRP > 5 mg/L: 10 mg, baseline n = 9 and week 12 n = 7; 20 mg, baseline n = 24 and week 12 n = 18. 20 mg n varies by sample availability. Analyses across fecal calprotectin, hsCRP, albumin, and neutrophils include one patient who was not efficacy-evaluable at week 12.

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Higher Remission Rate With 20 mg BID Dose in Severe Patients (MES = 3)

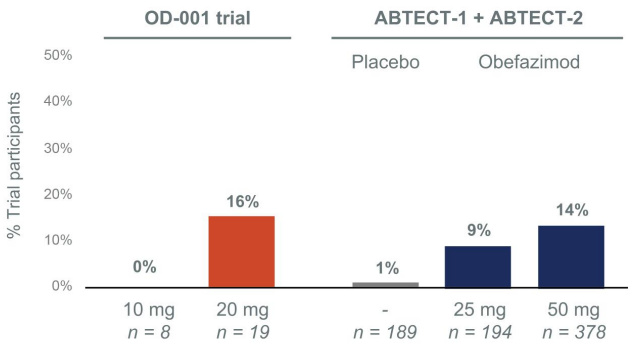
Similar activity between OD-001 doses in patients with moderate disease (MES = 2)

- Clinical remission rates in patients with moderate endoscopic disease are comparable at both doses



Dose-dependent activity in patients with severe disease (MES = 3)

- Greater barrier disruption may increase microbial exposure, requiring greater RIPK2 inhibition for efficacy

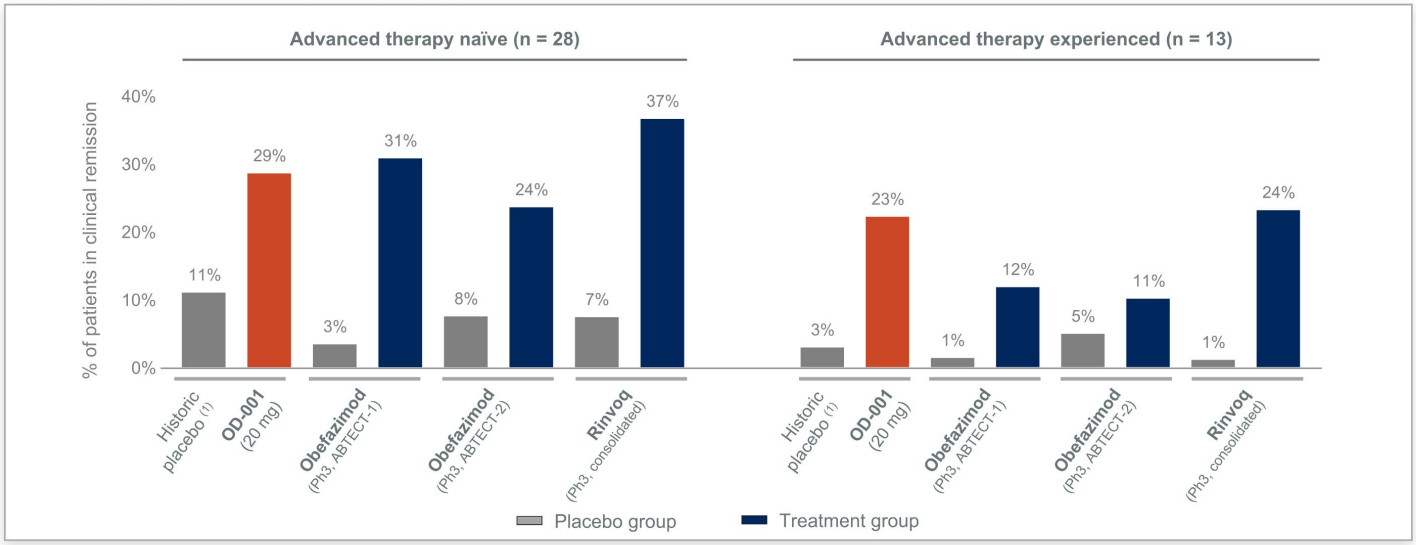


Disclaimer: Cross-trial comparisons are for context only, and no head-to-head study was conducted; differences in study design, patient populations, and other factors should be considered when interpreting results. OD-001 and a comparator have not been approved and may never be approved by any regulatory authority.
Source: Obefazimod ECCO 2026 presentation (P0952).

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Clinical Remission Rate at 20 mg BID Is Similar in AT-Naïve and AT-Experienced Patients

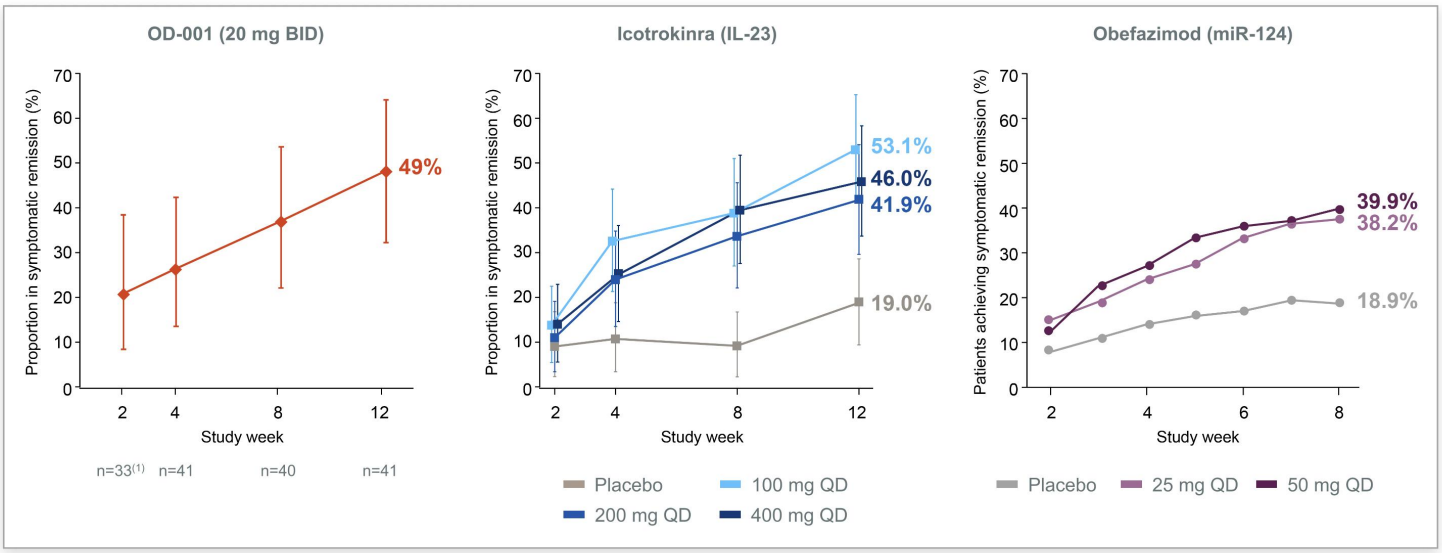
- Existing therapies often show reduced activity in AT-experienced patients, whereas OD-001 shows similar activity in both populations



Disclaimer: Cross-trial comparisons are for context only, and no head-to-head study was conducted; differences in study design, patient populations, and other factors should be considered when interpreting results. OD-001 and a comparator have not been approved and may never be approved by any regulatory authority.
Notes: (1) 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.

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OD-001 Is Competitive With Other Oral Agents on Early Symptomatic Remission, With Increases Through Week 12



Notes: (1) Part 2 patients were randomized 1:4 to either 10 mg BID for the first two weeks followed by escalation to 20 mg BID for weeks 3–12, or 20 mg BID from week 1 through week 12. This staggered dosing design accounts for the difference in n at the week 2 time point.
Sources: Obefazimod ECCO 2026 presentation (P0923); Icotrokinra UEG Week 2025 presentation (OP206).

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Transcriptomic Assessments to Support Clinical and Biomarker Assessments

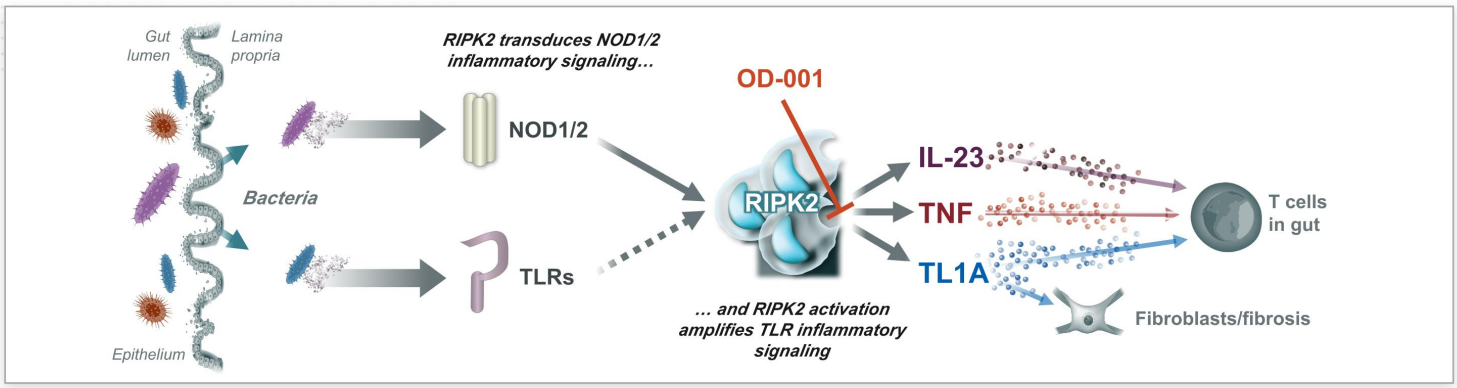
- Transcriptomic analysis on mucosal biopsies from patients at baseline and after induction therapy to detect differentially expressed genes

ASSESSMENTS SUPPORT OD-001 THERAPEUTIC ACTIVITY

Unbiased assessment of ~20,000 genes across the whole transcriptome	• Transcriptional response identified modulation of RIPK2-responsive genes, genes encoding inflammatory cytokines or T cell subsets, and fibrotic or mucosal healing genes
Correlation between efficacy, biomarkers, histology, and transcriptomics	• Patients with clinical remission or response show concordant improvement across biomarkers and reductions in inflammatory and fibrotic transcriptional profiles
Identification of OD-001-specific effects and comparison to public datasets	• Transcriptional responses to OD-001 overlap with response to anti-IL-23 antibodies • OD-001 also modulates inflammatory responses not addressed by existing therapeutic mechanisms

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OD-001 Reduces Inflammatory and Fibrotic Gene Expression in IBD



OD-001 reduces RIPK2 and microbial signatures

Gene set	Effect	p_{adj}
RIPK2 signaling	⬇	9E-12
Innate host defense response	⬇	9E-5
AP-1	⬇	6E-4

OD-001 reduces cytokine expression and signaling

Gene set	Effect	p_{adj}
IL-23 signaling	⬇	1E-7
TNF/NF-κB signaling	⬇	1E-8
TL1A signaling	⬇	7E-4

OD-001 reduces T cell activity

Gene set	Effect	p_{adj}
Th17 signaling	⬇	6E-26
Th1 signaling (1)	⬇	7E-9

OD-001 reduces fibrotic signaling and increases epithelial homeostasis signaling

Gene set	Effect	p_{adj}
Inflammatory fibroblasts	⬇	2E-4
Penetrating fibrosis (1)	⬇	7E-3
Epithelial homeostasis	⬆	2E-10

Notes: Week 12–baseline change in the 20 mg cohort (n = 40 paired mucosal samples); two additional samples failed RNA-seq QC. Pathway enrichment used hypergeometric testing with FDR adjustment; (1) p values reflect week 12 vs. baseline for only responder/remitter patients (n = 25); all other comparisons are week 12 vs. baseline for the full cohort, including one patient not evaluable for efficacy (n = 40); gene sets from MSigDB: Reactome Antimicrobial Peptides, PID AP-1 pathway, PID IL-23 pathway, Hallmark TNF/NF-κB; Internal: RIPK2 Pathway, cited studies: TL1A, Th17, Th1, Inflammatory Fibroblasts, Penetrating Fibrosis, Epithelial Homeostasis.
Sources: Cell Syst 2015;1(6):417–425; Am J Gastroenterol 2011;106(7):1272–1280; J Leukoc Biol 2017; 101(3):727-737; Nat Genet 2025;57(7):1742–1753; Gut 2022;71(7):1289–1301; Sci Rep 2021;11(1):9010.

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

- Phase 2a trial provides clinical proof of concept for a first-in-class oral therapy targeting RIPK2 with a compelling efficacy and safety profile
  - Efficacy, biomarker, and transcriptomic data support 20 mg BID as projected therapeutic dose
  - Similar clinical remission rates irrespective of AT experience with 20 mg BID
  - Symptomatic remission as early as week 2 with increased remission rate through week 12 of induction therapy
  - Colonic and systemic biomarkers and transcriptomics link RIPK2 inhibitor therapeutic effects with the clinical efficacy observed

- Safety is potentially differentiating: no SAEs, no serious infections, and no discontinuations due to TEAEs
  - Profile could position OD-001 as a backbone for combinations and long-term oral maintenance therapy
  - No RIPK2-targeting therapy is currently approved; therefore, use of OD-001 is not limited by patients' failure to other agents in the class

- OD-001 is being advanced toward late-stage monotherapy and combination trials concurrently, with topline results expected H2 2027
  - Phase 2a combination trial with vedolizumab for induction (OD-001 + vedolizumab vs. vedolizumab + placebo), followed by OD-001 monotherapy(1)
  - Phase 2b monotherapy trial testing multiple doses of OD-001 vs. placebo

Notes: (1) Phase 2a combination trial overview presented at ACG 2026; poster 5698 is available on corporate website HERE).

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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., anti-TNF) and oral therapies (e.g., JAKi)***

Orals ***Injectables*** ***Combinations***

RINVOQ upadacitinib · Velsipity (etrasimod) tablets · HUMIRA adalimumab · Entyvio vedolizumab · Skyrizi risankizumab-rzaa · Tulisokibart

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 H2 2027

$7B+ Combination induction with monotherapy maintenance

Combination induction » **OD-001 maintenance**

Preferred therapy for induction and maintenance

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

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Oral SLC15A4 Inhibitor: OD-002

Modulating B Cell Activity and Interferon Signaling

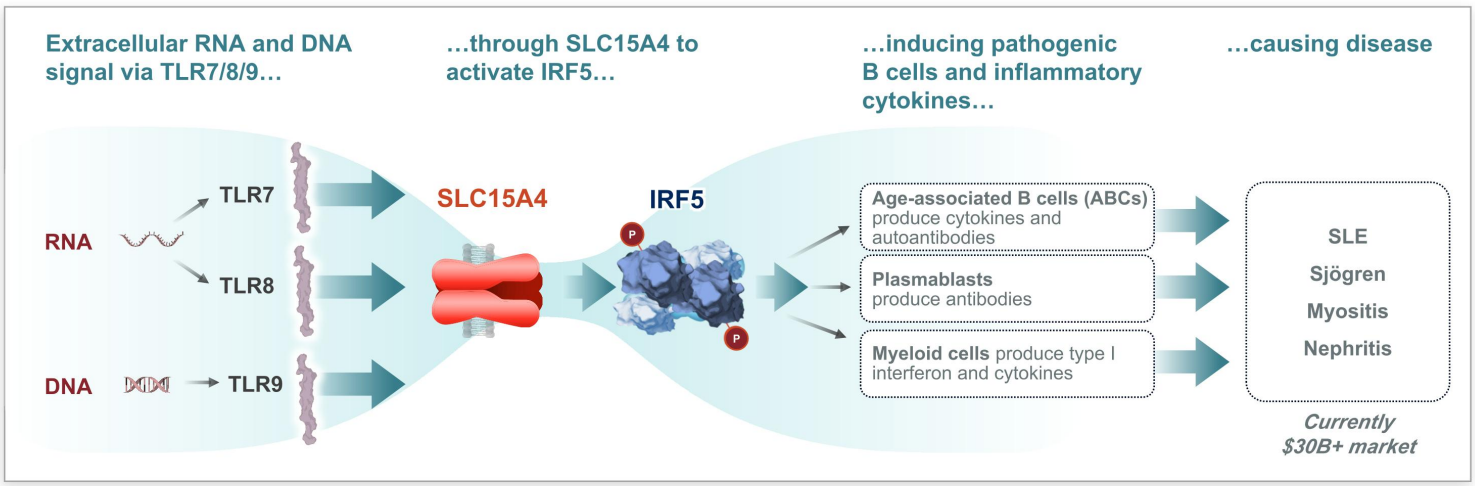
CTA Filing in H2 2026

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

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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



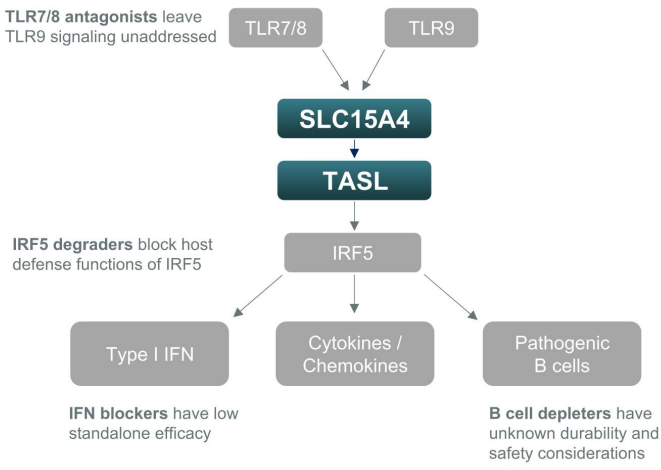
Source: Evaluate Pharma.

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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

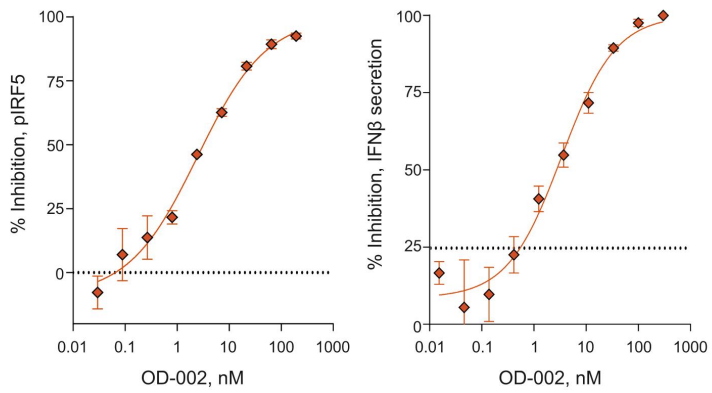


Sources: Immunity 2006;25:429; Nat Immunol 2022;23:1457; PLoS One 2021;16:e0244439;Proc Natl Acad Sci U S A 2013;110:2940; Proc Natl Acad Sci U S A 2022;119:e2200544119; Nat Commun 2025;16:968;Proc Natl Acad Sci U S A 2010;107:10154; PLoS One 2014;9:e103478; J Immunol 2010;184:796.

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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

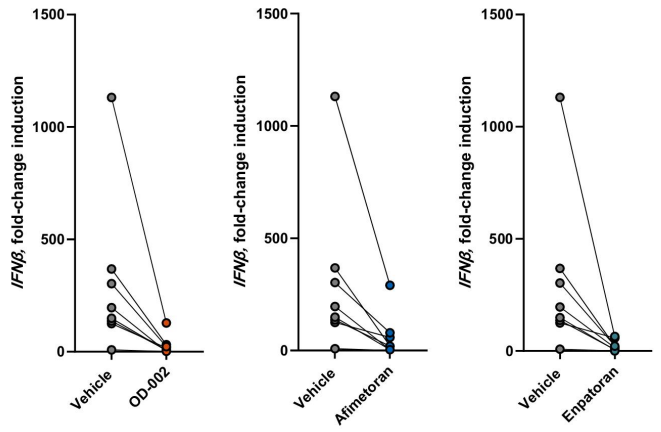
Potency in vitro IC_{50} in human primary cells (e.g., TNF, IFN)	≤ 10 nM
Potency in vivo IFNα inhibition at 3 mg/kg; *phenocopies SLC15A4 knockout*	> 95%
Selectivity TLR3, STING, and SLC15A1/2/3	> 1,000x
Projected once-daily human dose Dose to maintain IC_{90}	18 mg
Preclinical safety and tolerability	High
Intellectual property Composition of matter	2046

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OD-002 Blocks IFN Signaling From Both RNA and DNA

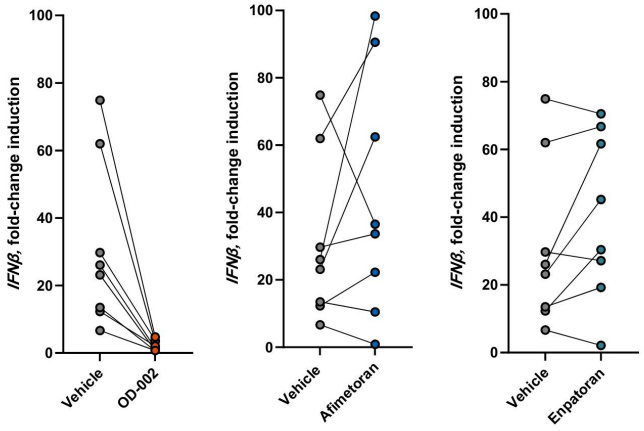
- TLR7/8 antagonists cannot block both arms of the nucleic acid response driving disease, with pathogenic signaling through TLR9 remaining active

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



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

SLC15A4 inhibition also blocks cytokine production *initiated by DNA*

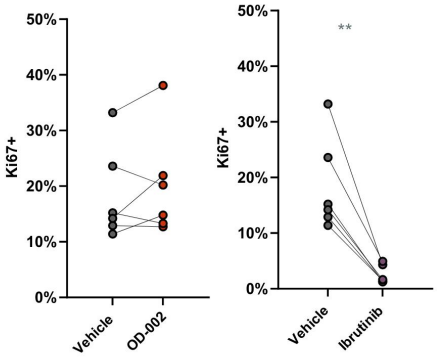


TLR9 stimulation induced by ODN-2395 in PBMCs from healthy donors

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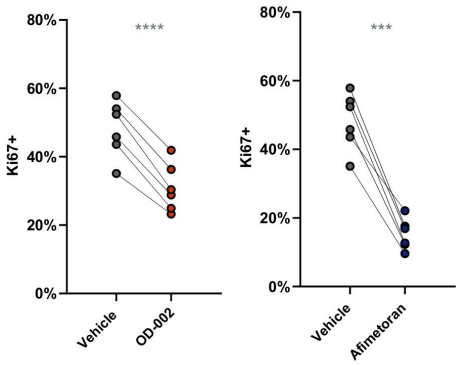
OD-002 Selectively Blocks Human B Cell Proliferation From Pathogenic Stimuli

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



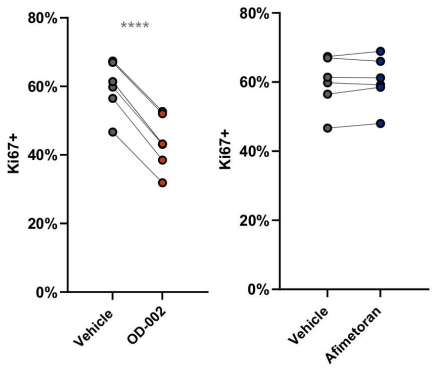
Ibrutinib, a BTK inhibitor, blocks non-pathogenic B cell proliferation

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 <u>also</u> blocks B cell proliferation induced by CpG, a TLR9 agonist that mimics pathogenic stimuli



Afimetoran does not block TLR9-driven B cell proliferation

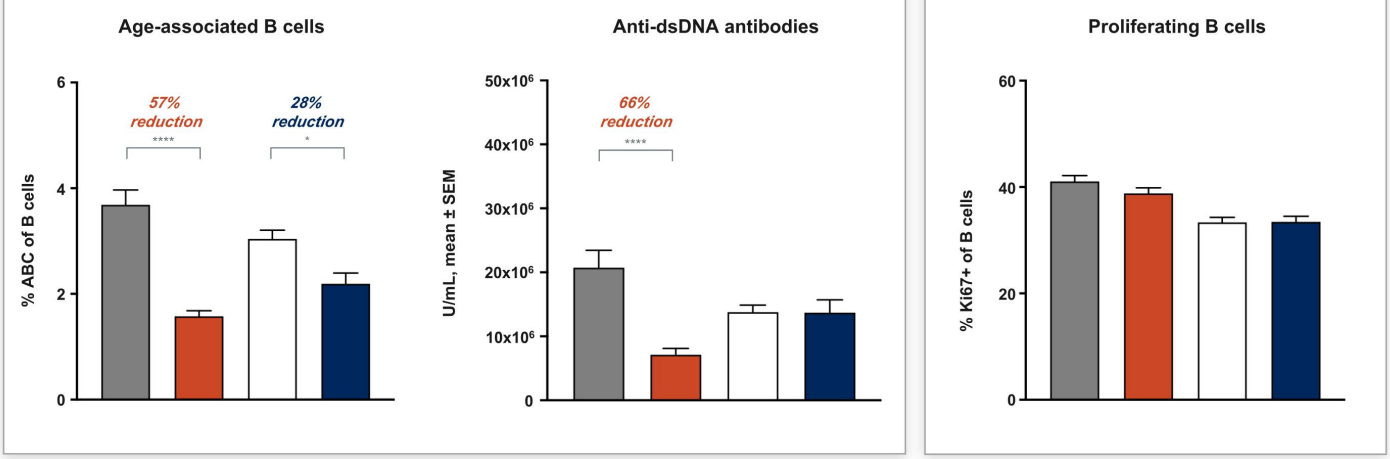
Notes: Isolated memory B cells from healthy donors were pretreated with indicated compound for 24 hours prior to stimuli and % Ki67+ was measured after three days; ** p < 0.01; *** p < 0.001; **** p < 0.0001.

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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; afimetoran is a TLR7/8 antagonist currently in clinical development

HPMC vehicle OD-002 PBS/DMSO vehicle Afimetoran

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

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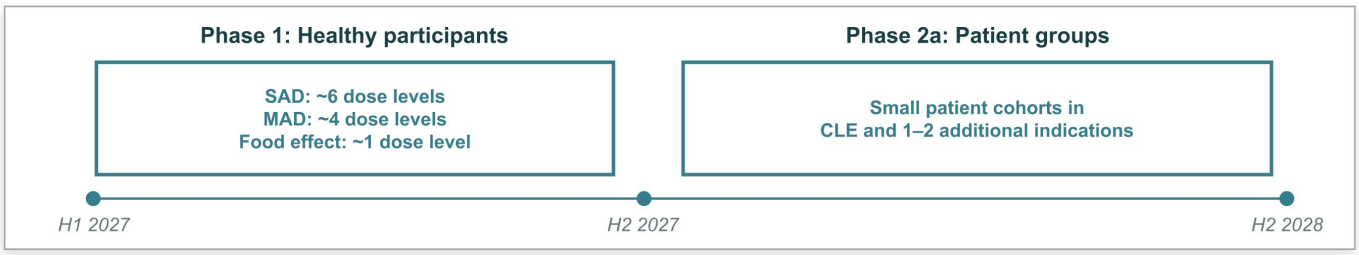
Phase 1/2a Trial Initiation Expected in H1 2027 With Efficacy Readouts Expected in H2 2028

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.

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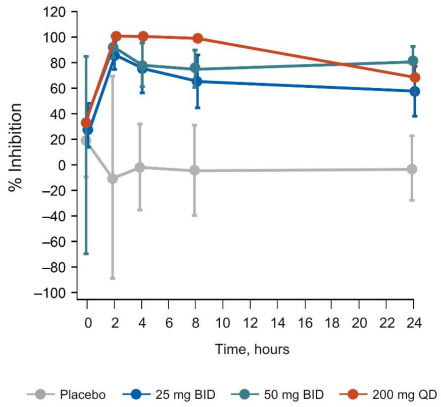
Phase 1 Ex Vivo Stimulation Assays Correlate With Activity in Lupus Patients

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?
MERCK *Enpatoran*	Ph3 initiated March/April 2026	✓	✓
beeline MEDICINES *Afimetoran*	Ph2 completed June 2026	✓	✓
Eisai *E6742*	Ph2 initiated March 2026	✓	✓
NOVARTIS *MHV370*	Discontinued for strategic reasons	✓	N/A

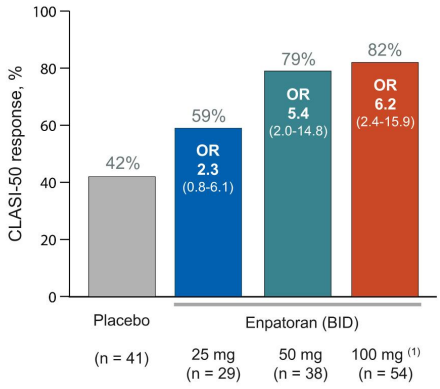
Enpatoran phase 1 ex vivo stimulation assay

- Doses advanced into phase 2 trials achieved > 60% IFNα 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



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

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TNFR2 Agonist: OD-003

Modulating Treg to Treat Autoimmune and Inflammatory Disease

IND-Enabling Studies Ongoing

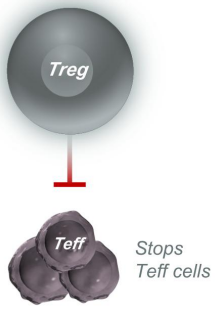
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Treg Therapy Has Broad Therapeutic Potential in Large I&I Diseases

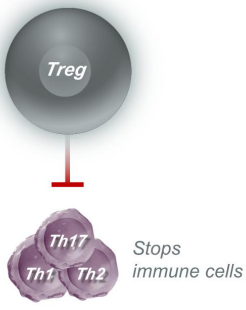
Atopic dermatitis	SLE	Alopecia areata	Vitiligo	Multiple sclerosis	Rheumatoid arthritis	Asthma

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

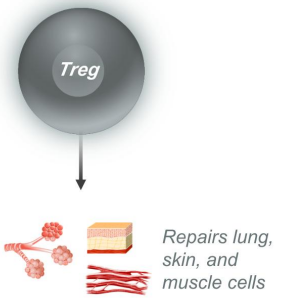
EFFECT 1:
Reduction in Teff cell proliferation and activity
Reduces direct tissue damage and immune activation



EFFECT 2:
Reduction of immune cell activity
Reduces production of pro-inflammatory cytokines, including IFNɣ, IL-4, IL-5, and IL-13



EFFECT 3:
Repair of tissue-resident non-immune cells
Induces durable disease-modifying effects

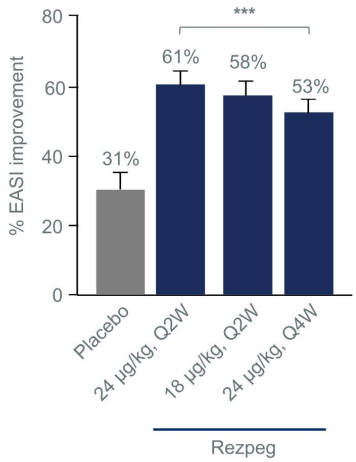


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TNFR2 Agonism Is the Next Evolution of Treg-Targeted Therapy

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 mid-2027

NEKTAR®

NKTR-1065: IND submission expected in 2027

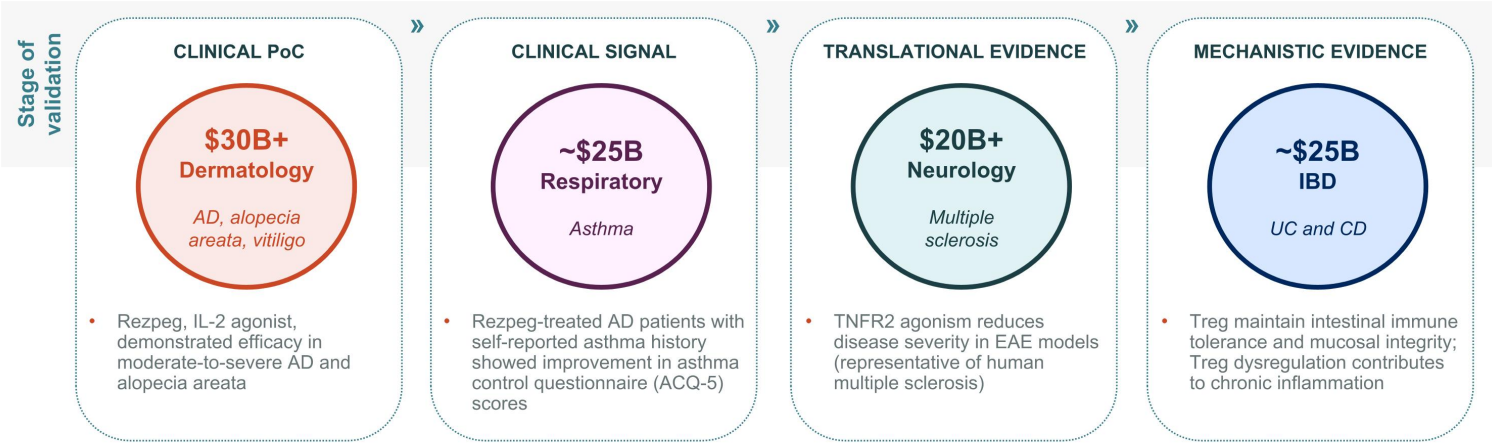
ODYSSEY THERAPEUTICS

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.

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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.

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Final Words

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Upcoming Milestones for Lead Programs Through 2028

- Topline induction results from phase 2b monotherapy and phase 2a combination trials are expected in H2 2027
  - Phase 2b monotherapy trial is randomized, double-blind, and placebo-controlled
  - Phase 2a combination trial is randomized, double-blind, and active-controlled (OD-001 and vedolizumab vs. vedolizumab and placebo)
- Odyssey expects to file a CTA for OD-002, an SLC15A4 inhibitor, in H2 2026, with phase 1/2a trial initiation expected in H1 2027
  - Phase 1 healthy-volunteer trial will use a validated ex vivo stimulation assay that has translated to efficacy with TLR7/8 antagonists
  - Phase 2 trial will enroll patients with multiple inflammatory diseases, including one cohort of cutaneous lupus patients
- OD-003, a TNFR2 agonist, is currently in IND-enabling studies, with projected completion in Q1 2027

2026	2027		2028	
H2	H1	H2	H1	H2
OD-001 Ph2b monotherapy trial initiation	**OD-002** Initiate healthy participant dosing in Ph1/2 trial	**OD-001** Ph2b monotherapy induction readout		**OD-001** First maintenance readout
OD-001 Ph2a vedolizumab combo trial initiation	**OD-003** IND-enabling study completion	**OD-001** Ph2a vedolizumab combo induction readout		**OD-002** Ph2a readouts in multiple indications
OD-002 File CTA		**OD-002** Ph1 results, including ex vivo stimulation assay		

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Thank you!
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