ODYSSEY THERAPEUTICS

Corporate Presentation

November 2026



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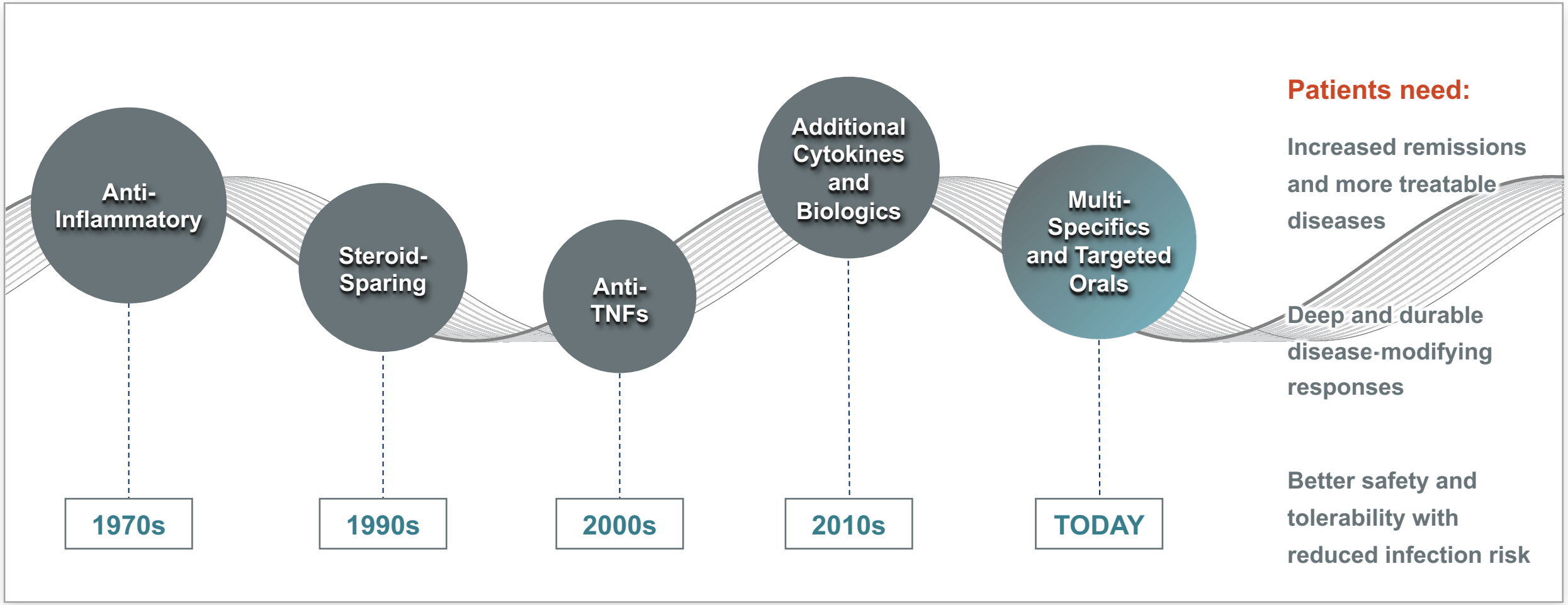
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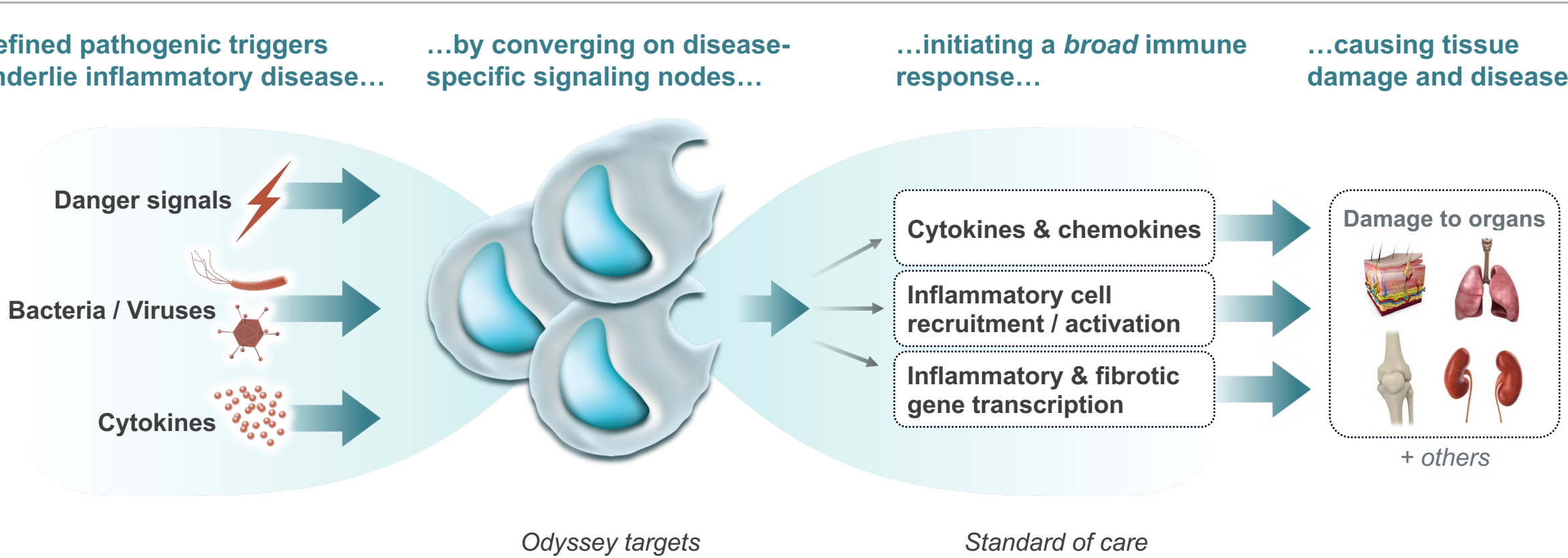
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Few Patients With Autoimmune Disease Have Durable Remission



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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 $387M as of September 30, 2026; expected to fund operations into H2 2028

LEADERSHIP

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Founder, CEO, and Director

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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*						**H1 2027:** Phase 1/2a initiation **H2 2027:** Phase 1 results, including ex vivo stimulation assay
TNFR2, OD-003 *Protein*	*AD, SLE, alopecia areata*						**H1 2027:** IND-enabling study completion
TSLP/IL-33, OD-004 *Protein*	*COPD, asthma*						

$387 million cash, cash equivalents and marketable securities as of September 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

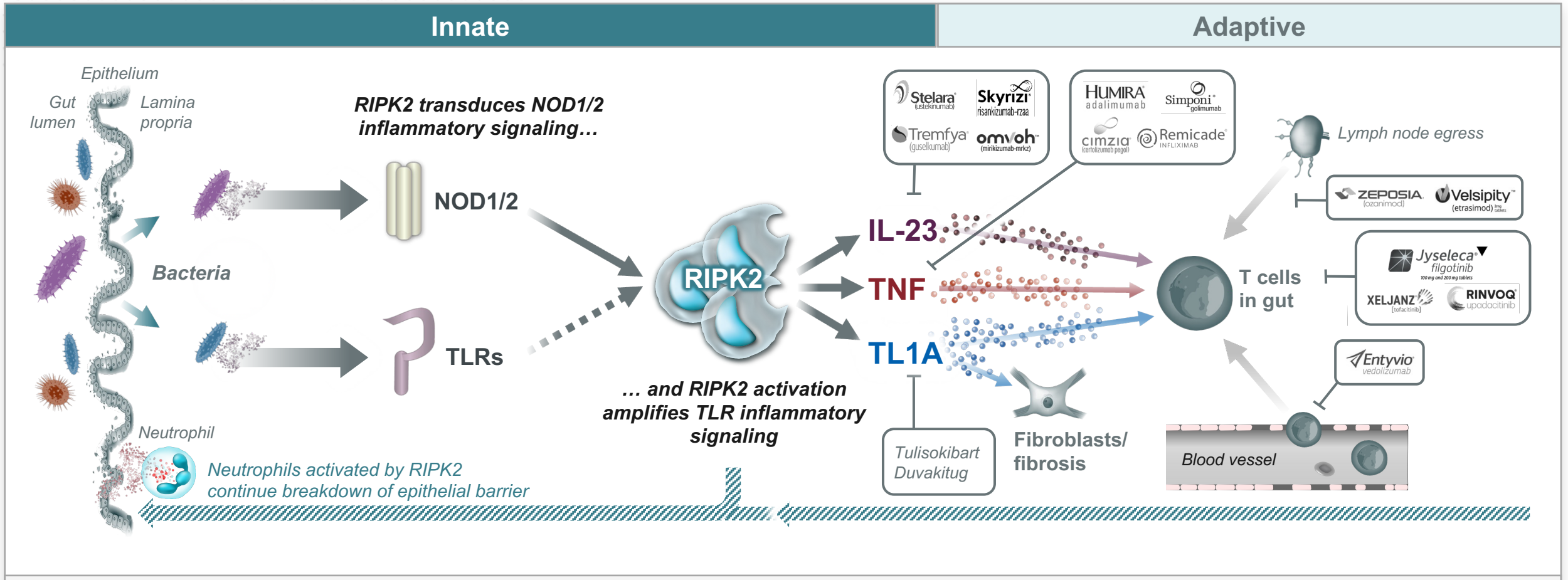
Addressing a Root Cause of Inflammation in IBD Using a Novel Mechanism of Action

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



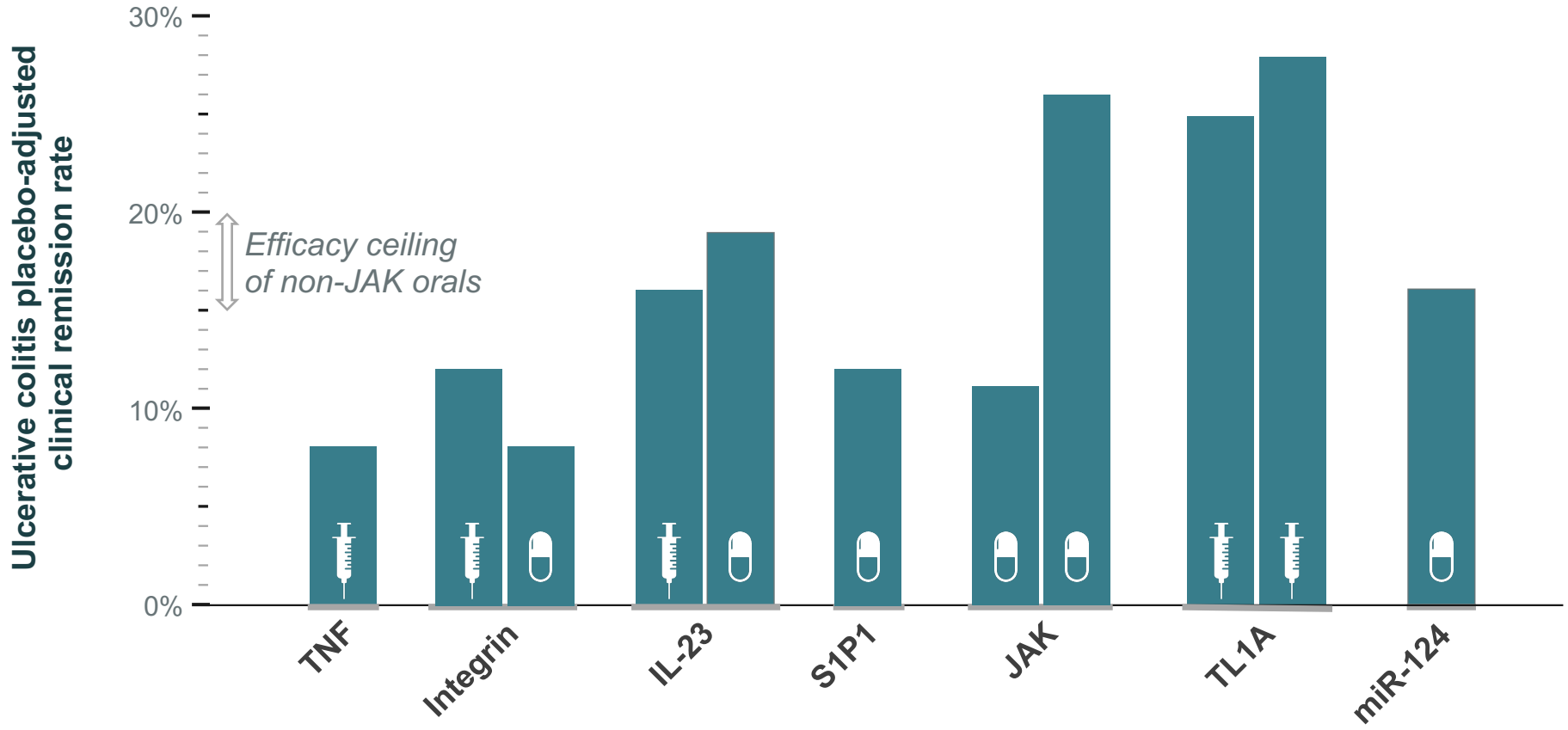
STEP 1:
Loss of epithelial integrity; microbes breach gut barrier

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

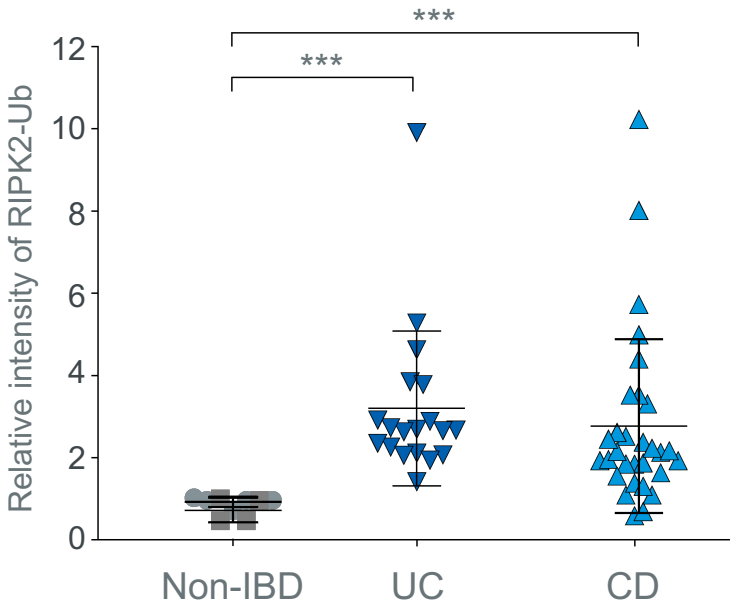
- RIPK2 pathway activation is increased in intestinal tissue from patients with both UC and CD
- RIPK2 ubiquitination is observed across all UC patients, including those with NOD2 LoF variants
- **Genetic validation** of RIPK2*: ATG16L1* and *CARD9* variants are linked to increased IBD risk; protective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

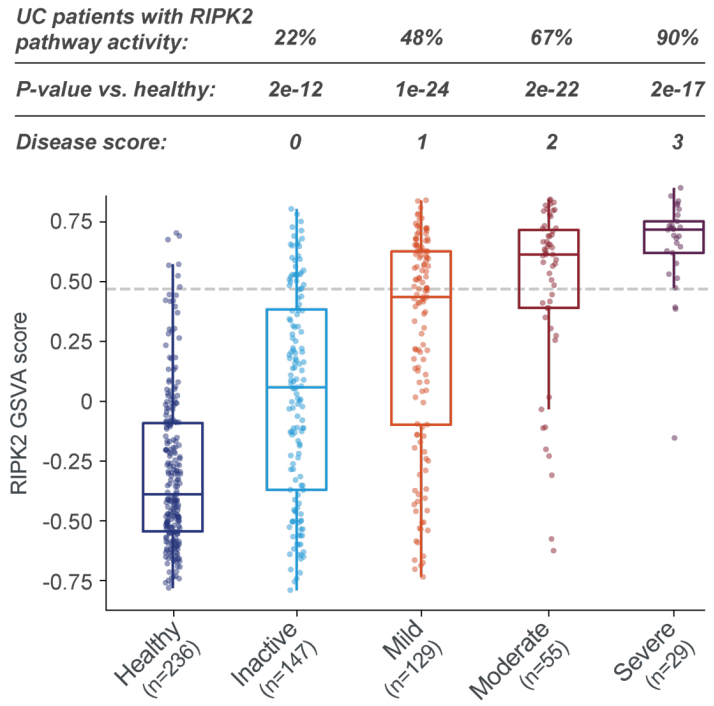
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RIPK2 Is Activated in UC and CD and Correlates With Severity

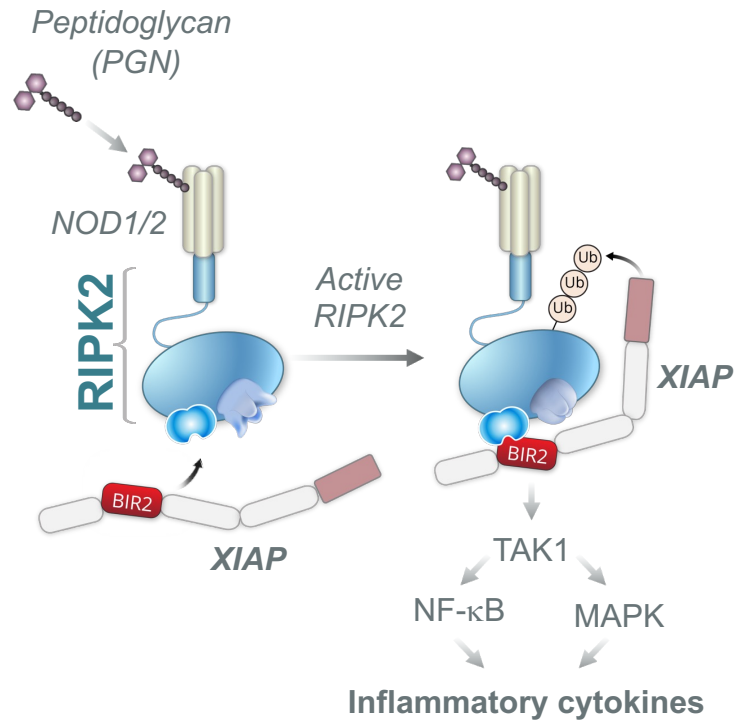
Increased levels of activated (ubiquitinated) RIPK2 in patients with UC and CD



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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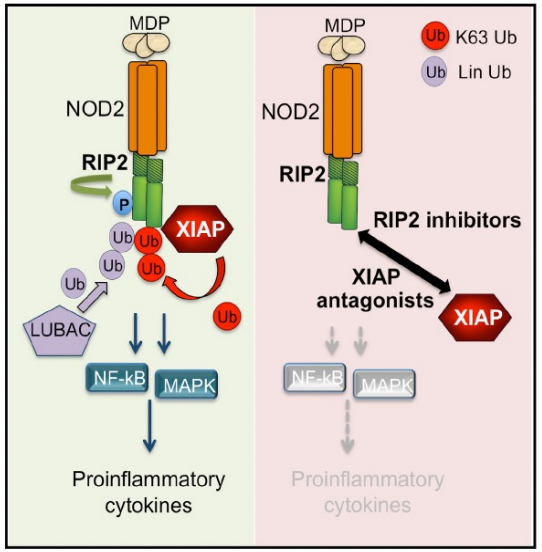
RIPK2 Drugs Must Block Scaffolding to Fully Inhibit NOD2 Signaling

Article

Molecular Cell

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

Graphical Abstract



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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 RIPK2 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[6], 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 NOD1 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

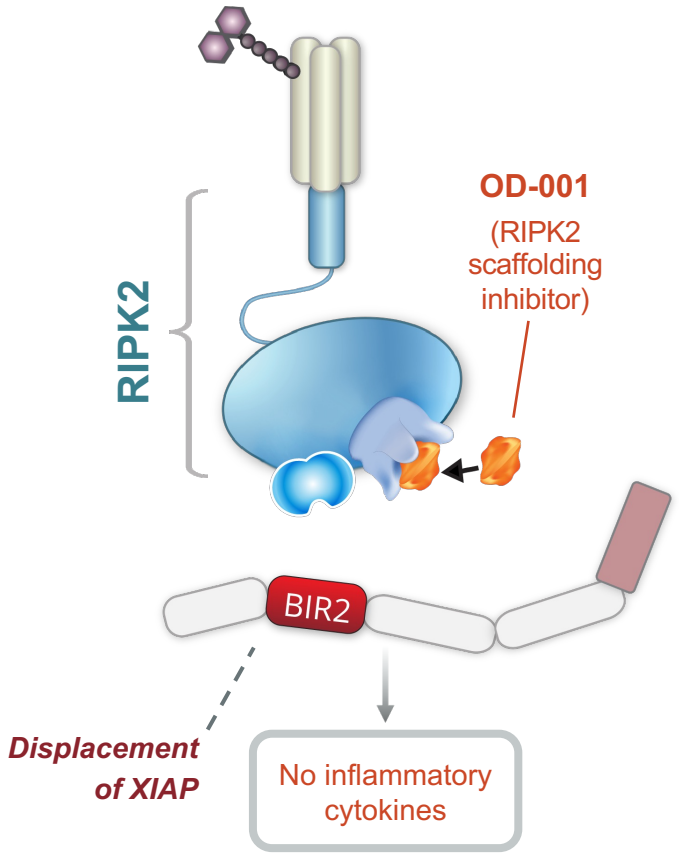
Receptor-interacting kinases (RIPKs) are components ofinnate immune receptor signaling complexes where they become ubiquitinated and contribute to NF- j B-mediated inflammatory signaling and cell death (Hrdinka & Gyrd-Hansen, 2017; Annibaldi & Meier, 2018). The intracellular bacteria-sensing receptors 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

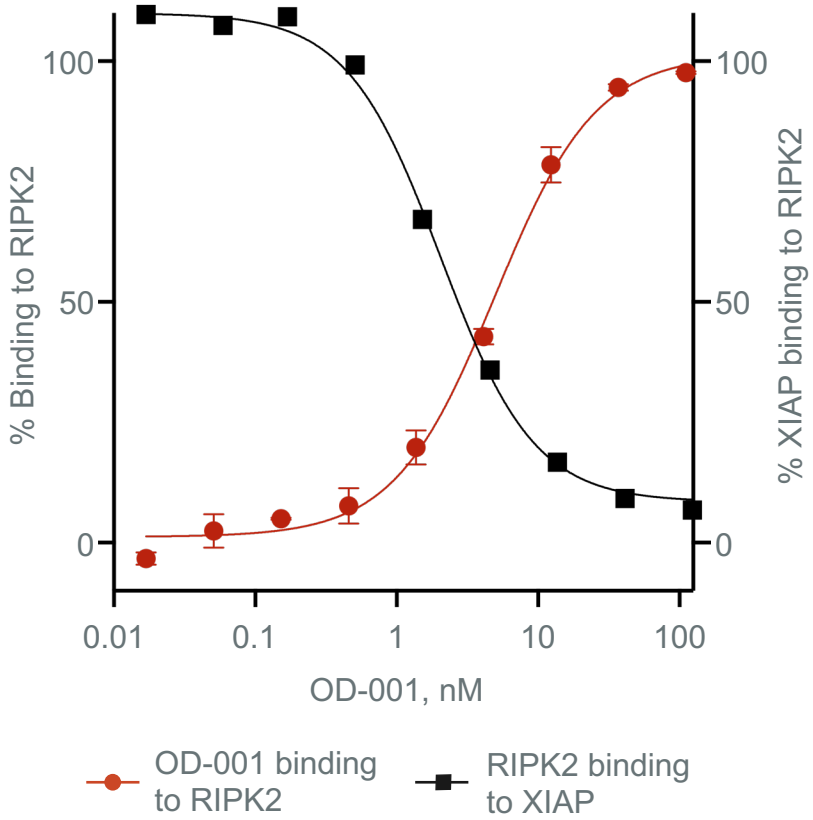
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Blocking RIPK2 Scaffolding With OD-001 Achieves Maximal Inhibition

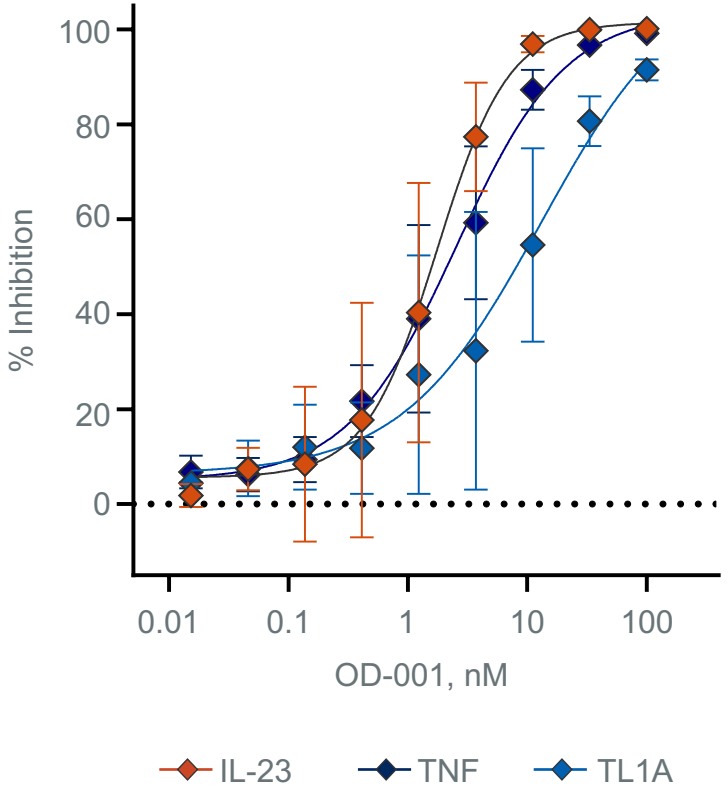
OD-001 is an allosteric scaffolding inhibitor that reduces the affinity for XIAP



Relationship between OD-001 and XIAP binding to RIPK2



OD-001 blocks cytokine production from macrophages stimulated with PGN

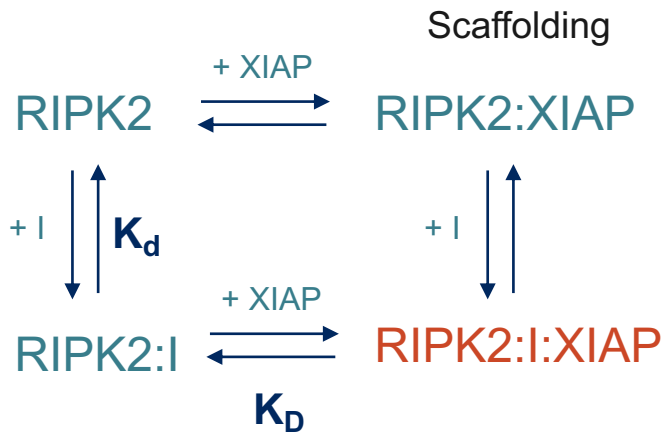


Notes: Binding to RIPK2 was measured by competition with a fluorescent ATP-site tracer in cells. XIAP binding to RIPK2 using recombinant protein was measured by HTRF. Cytokine production was assessed after 24 hours by MSD/ELISA in macrophages treated with compound for 1 hour and then stimulated with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

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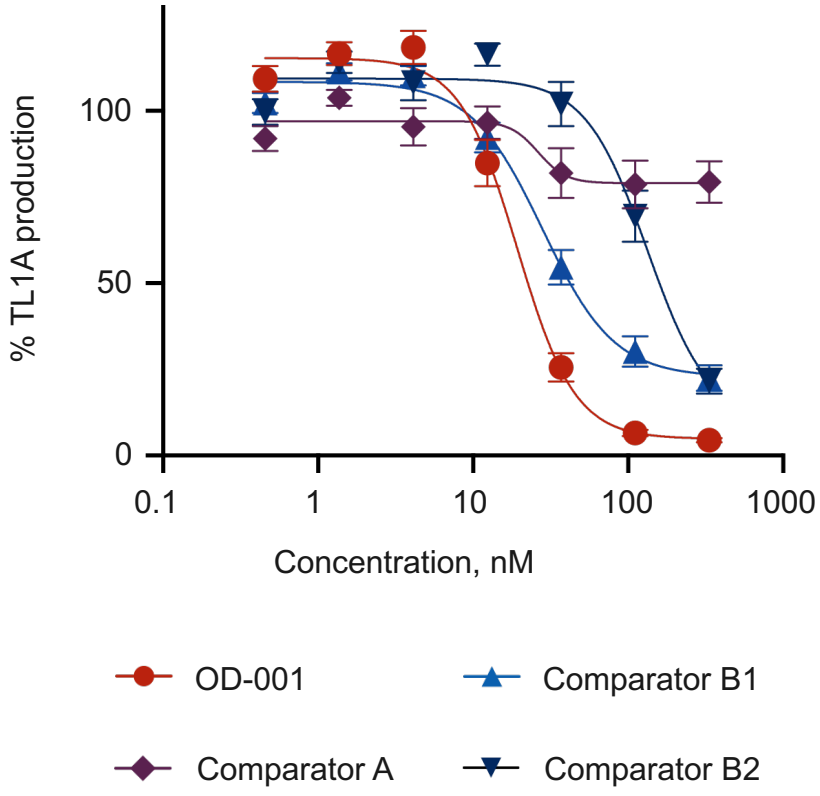
Not All RIPK2 Scaffolding Inhibitors Are the Same

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.

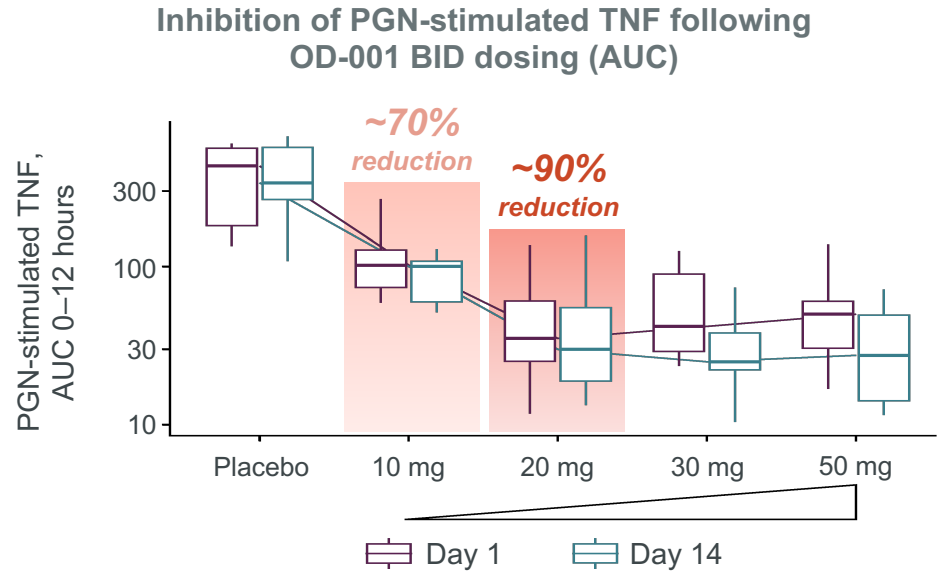
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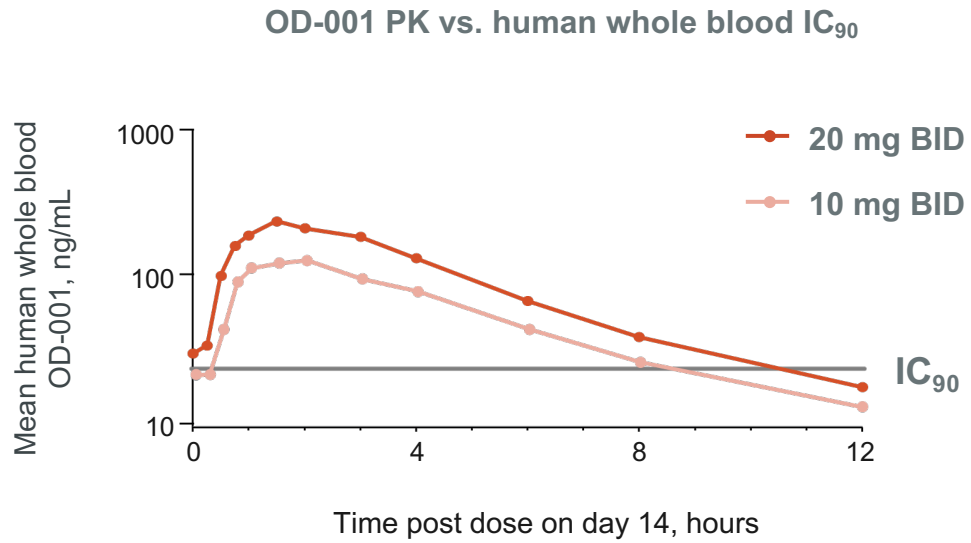
Phase 2a Monotherapy Trial Results

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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



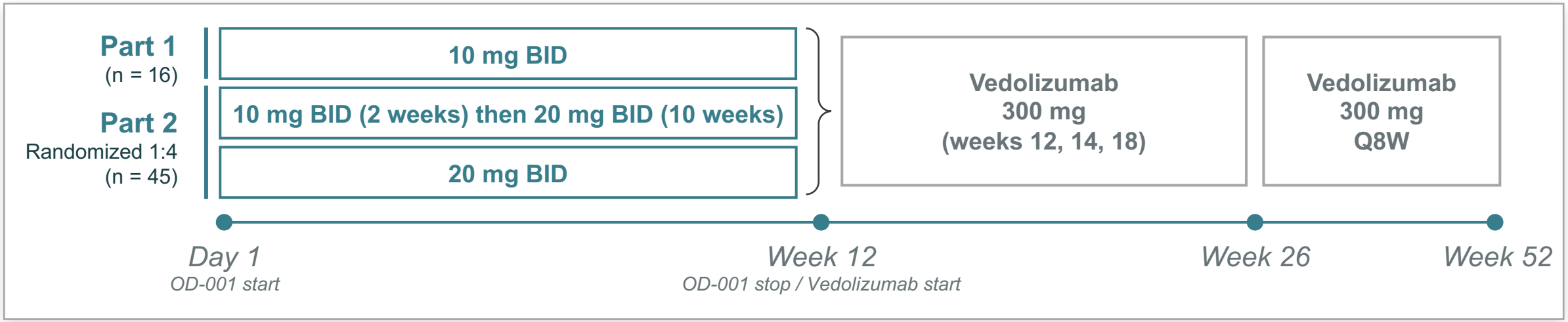


Notes: 3-component modified Mayo Clinical score (mMS) includes endoscopic severity (0–3), stool frequency (0–3), and rectal bleeding (0–3).

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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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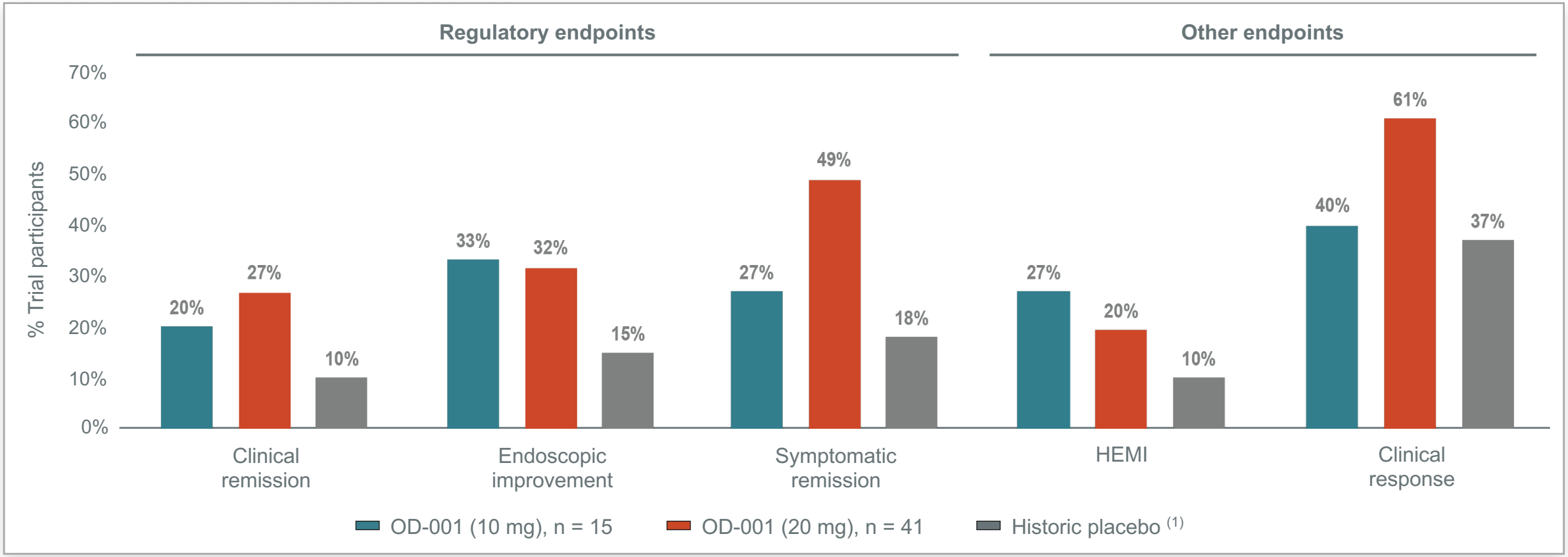
Participant Characteristics Are Similar to Other Contemporary UC Trials

	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 Demonstrates Efficacy Across Key Week 12 Outcomes, 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

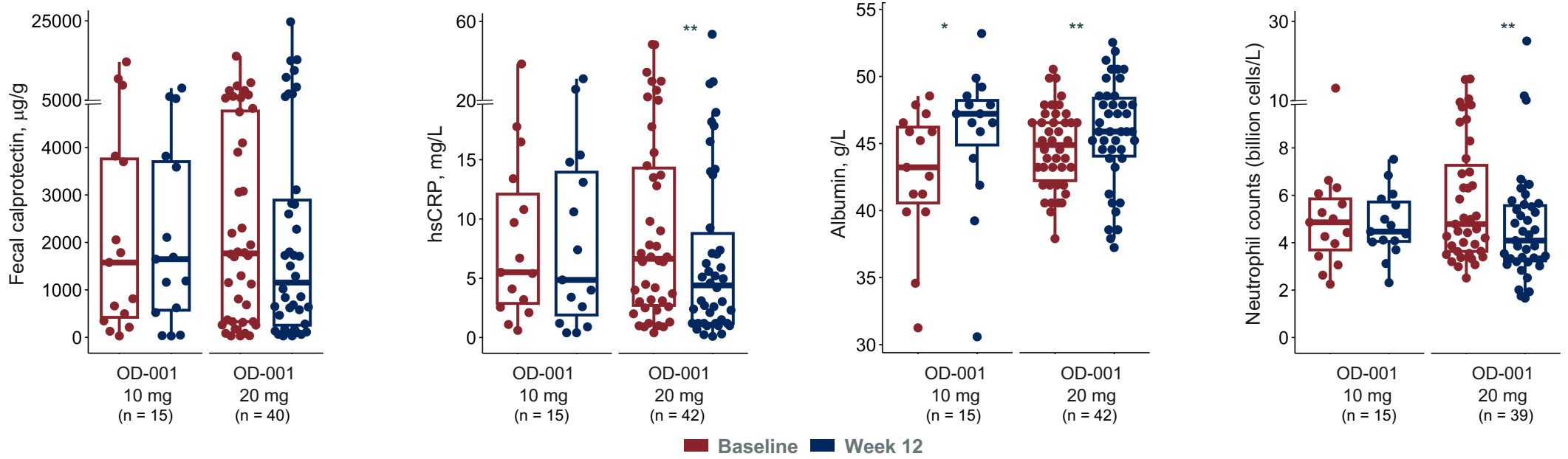
Disclaimer: Data presented are from ongoing studies, which will not be finalized until database lock. Thus, these data are subject to change prior to release of the CSR. Future results may differ from those shown here.
Notes: One participant in Part 1 and three participants in Part 2 discontinued treatment 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



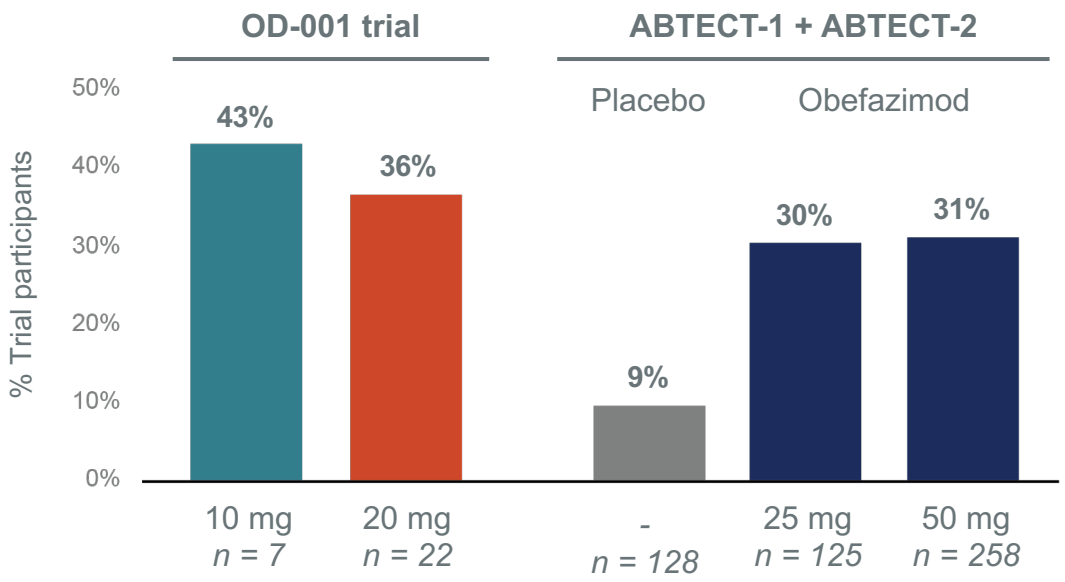
Notes: * $p < 0.05$; ** $p < 0.01$; nominal, unadjusted p values from paired Wilcoxon signed-rank tests comparing baseline and week 12 within each dose group; patients with hsCRP > 5 mg/L: 10 mg, baseline n = 9 and week 12 n = 7; 20 mg, baseline n = 24 and week 12 n = 18. 20 mg n varies by sample availability. 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)

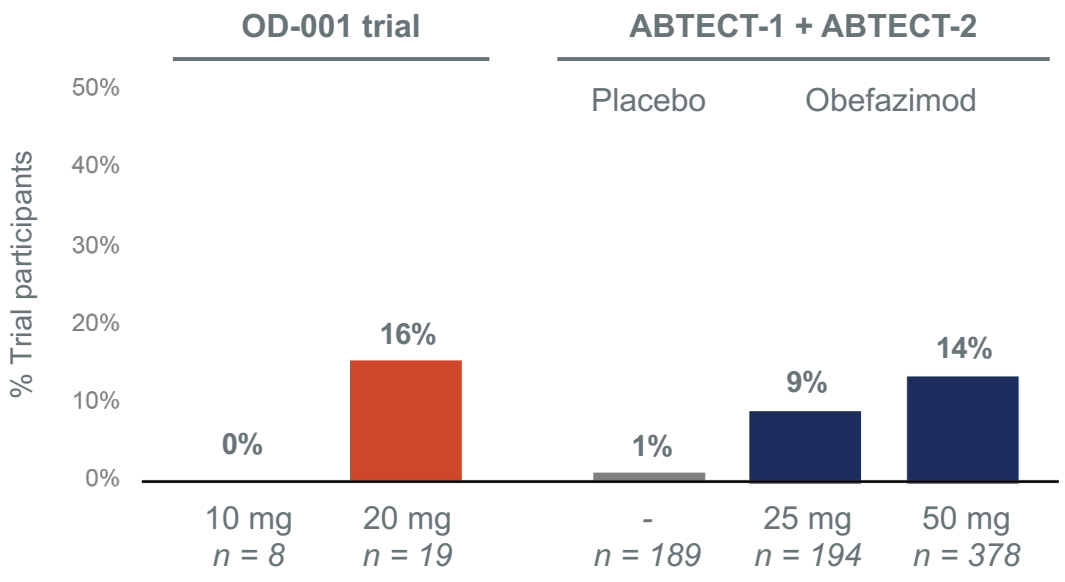
Similar activity between OD-001 doses in patients with moderate disease (MES = 2)

- 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

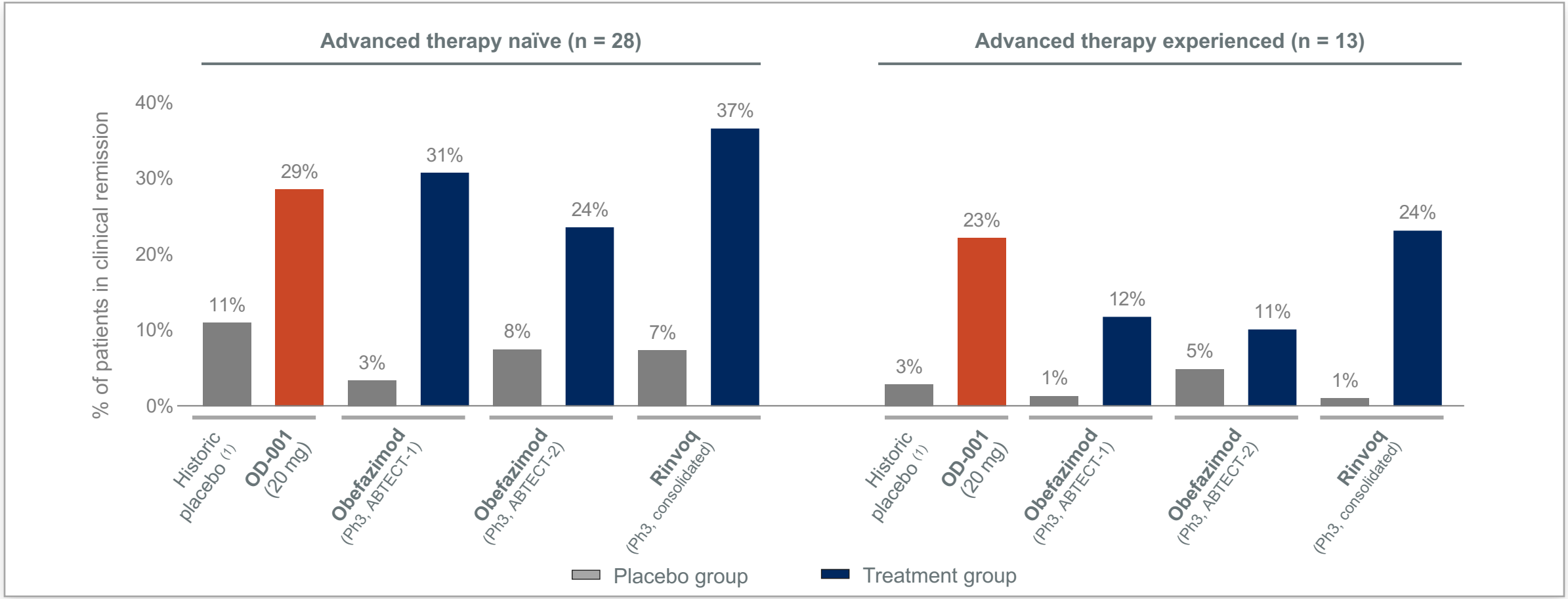


Disclaimer: Cross-trial comparisons are for context only, and no head-to-head study was conducted; differences in study design, patient populations, and other factors should be considered when interpreting results. OD-001 and a comparator have not been approved and may never be approved by any regulatory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

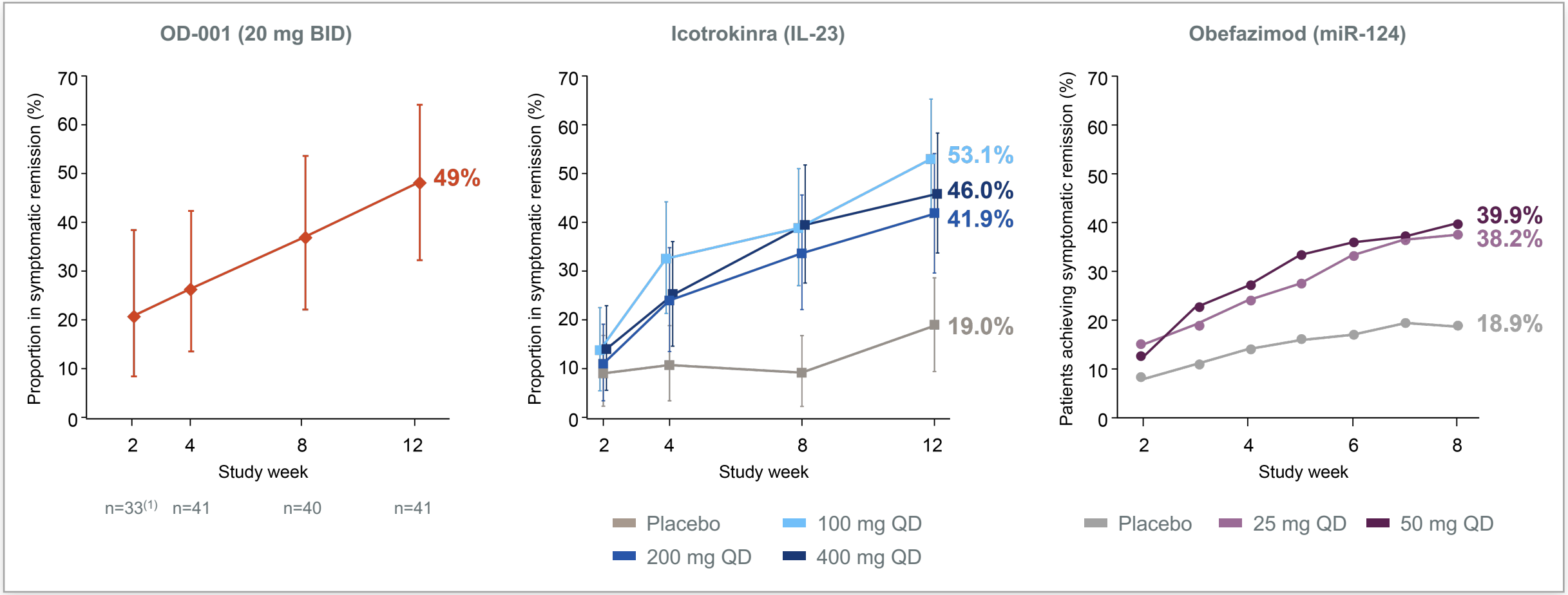


Disclaimer: Cross-trial comparisons are for context only, and no head-to-head study was conducted; differences in study design, patient populations, and other factors should be considered when interpreting results. OD-001 and a comparator have not been approved and may never be approved by any regulatory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

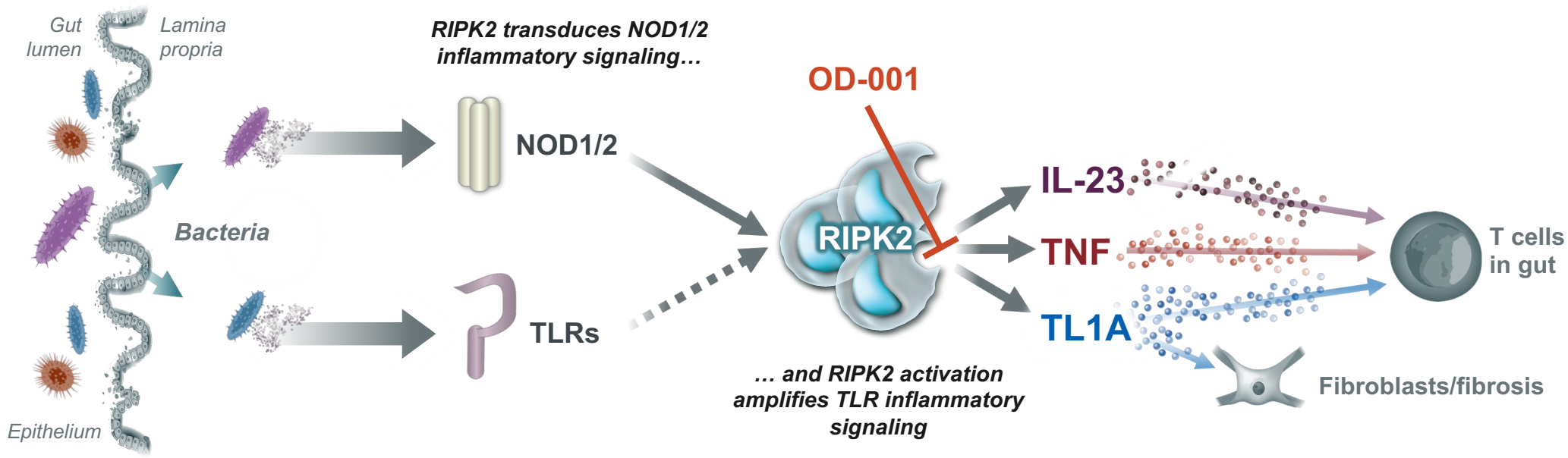


Identification of OD-001-specific effects and comparison to public datasets

- **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

RINVOQ upadacitinib

Velsipity (etrasimod) tablets

Injectables

HUMIRA adalimumab

Entyvio vedolizumab

Skyrizi risankizumab-rzaa

Tulisokibart

Combinations



Odyssey opportunity and development plan

~$3B Monotherapy

OD-001 for conventional patients

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

OD-001 for advanced therapy patients

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

Expansion of addressable population in 2028 and beyond

$4B+ Combination induction

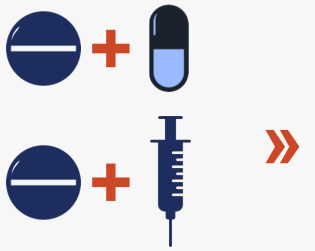
OD-001 combinations + **+ Vedolizumab** *(α4β7)*

+ Rinvoq *(JAK)*

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

Phase 2a vedolizumab combination induction results expected in 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

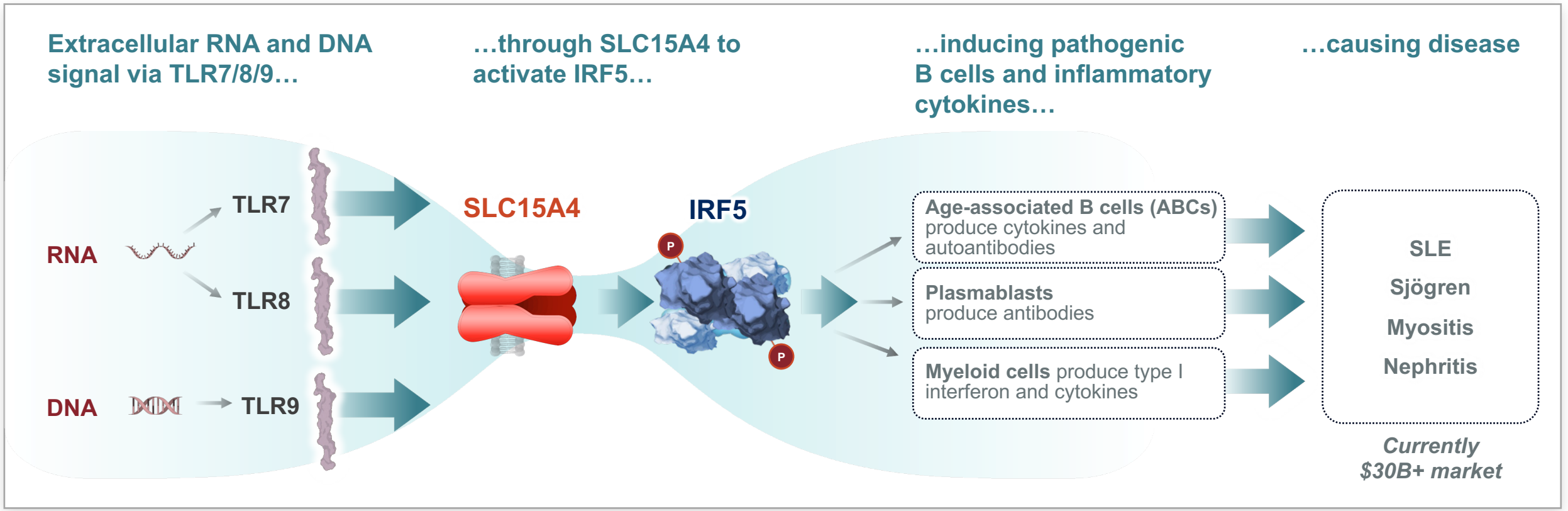
CTA Filing in H2 2026

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



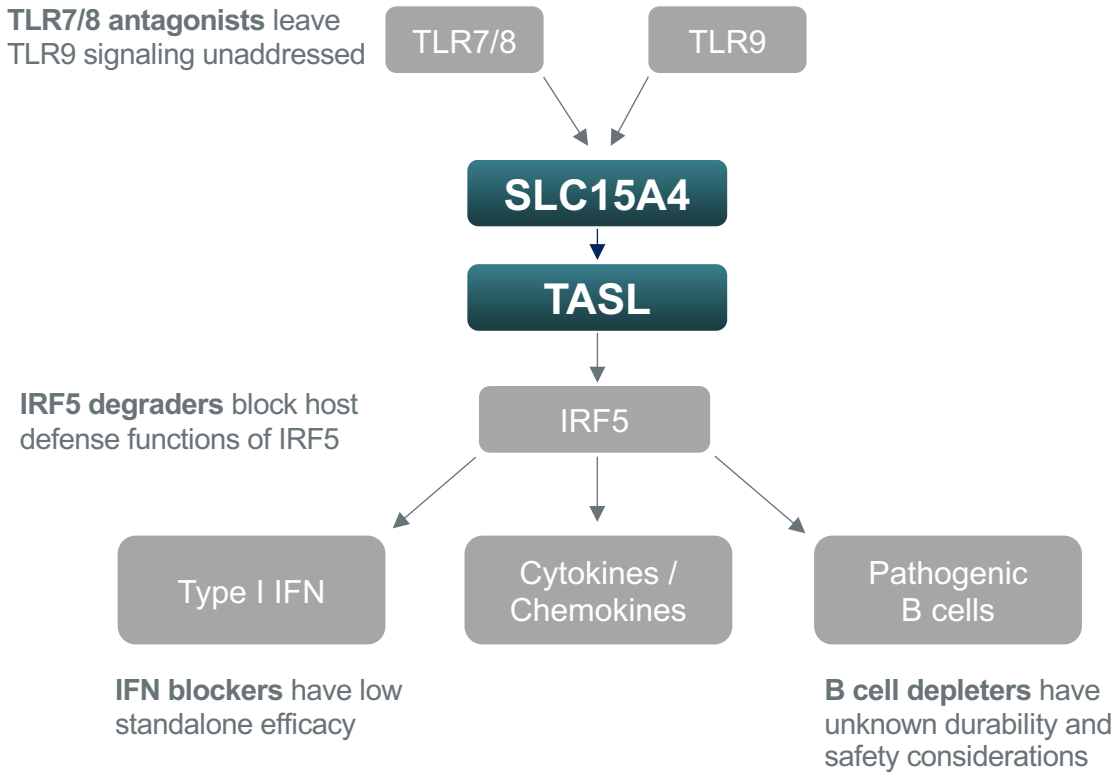
Source: Evaluate Pharma.

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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

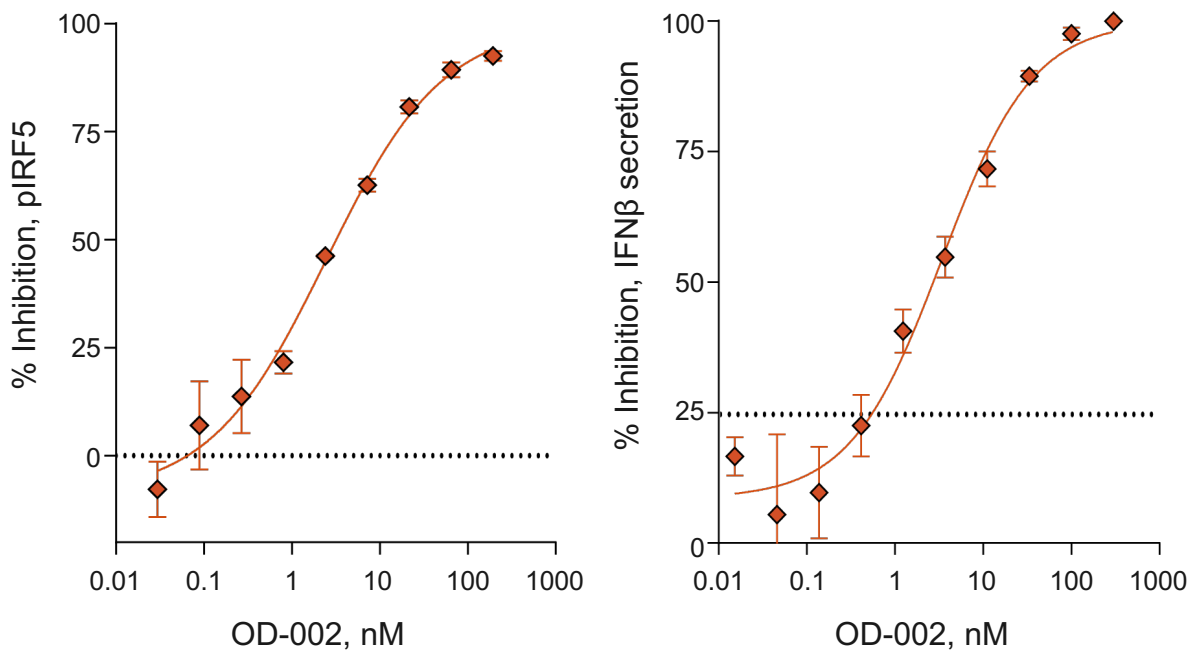


Sources: Immunity 2006;25:429; Nat Immunol 2022;23:1457; PLoS One 2021;16:e0244439; Proc Natl Acad Sci U S A 2013;110:2940; Proc Natl Acad Sci U S A 2022;119:e2200544119; Nat Commun 2025;16:968; Proc Natl Acad Sci U S A 2010;107:10154; PLoS One 2014;9:e103478; J Immunol 2010;184:796.

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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

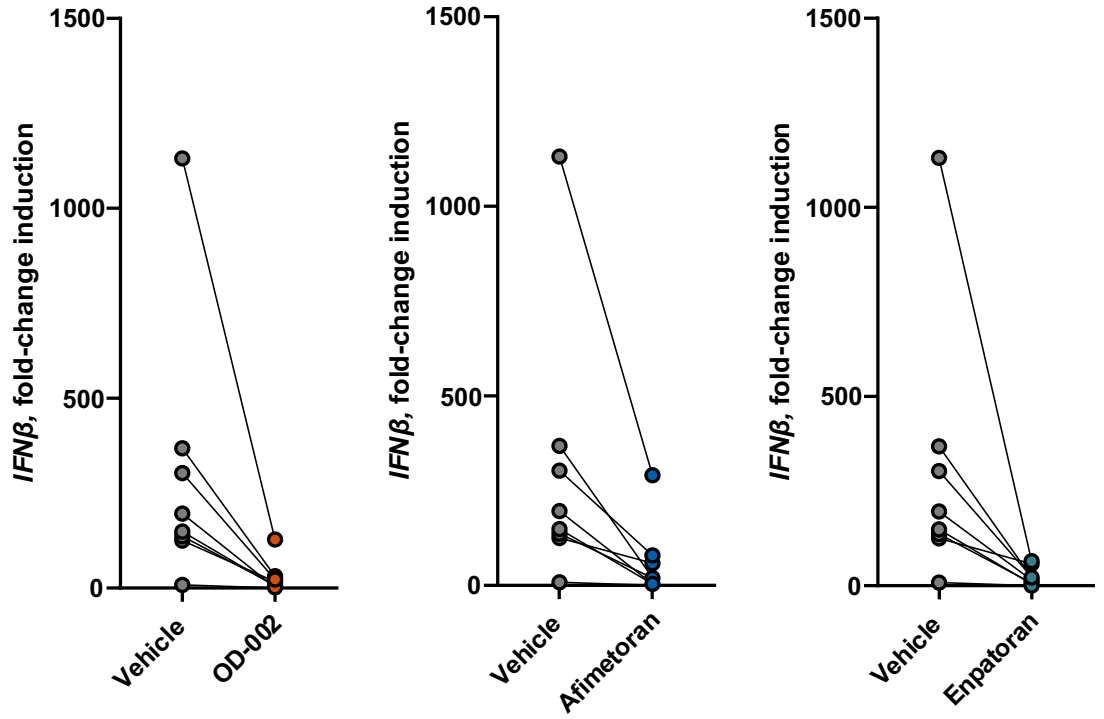
Potency in vitro IC_{50} in human primary cells (e.g., TNF, IFN)	≤ 10 nM
Potency in vivo IFNα inhibition at 3 mg/kg; *phenocopies SLC15A4 knockout*	> 95%
Selectivity TLR3, STING, and SLC15A1/2/3	> 1,000x
Projected once-daily human dose Dose to maintain IC_{90}	18 mg
Preclinical safety and tolerability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

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OD-002 Blocks IFN Signaling From Both RNA and DNA

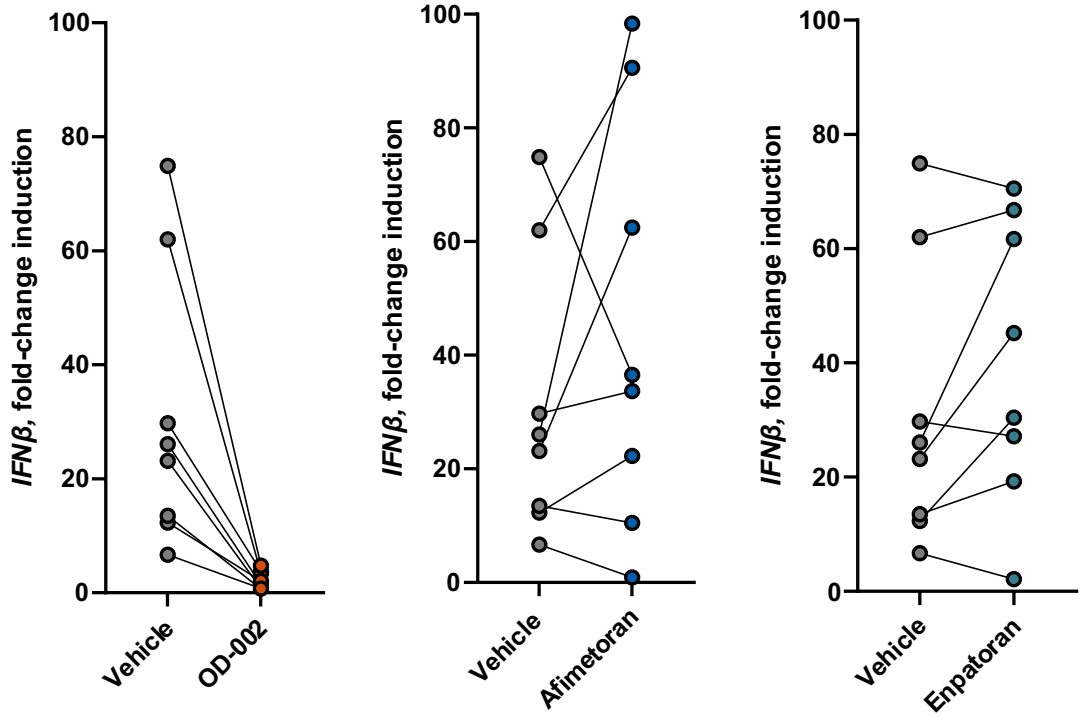
- TLR7/8 antagonists cannot block both arms of the nucleic acid response driving disease, with pathogenic signaling through TLR9 remaining active

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*

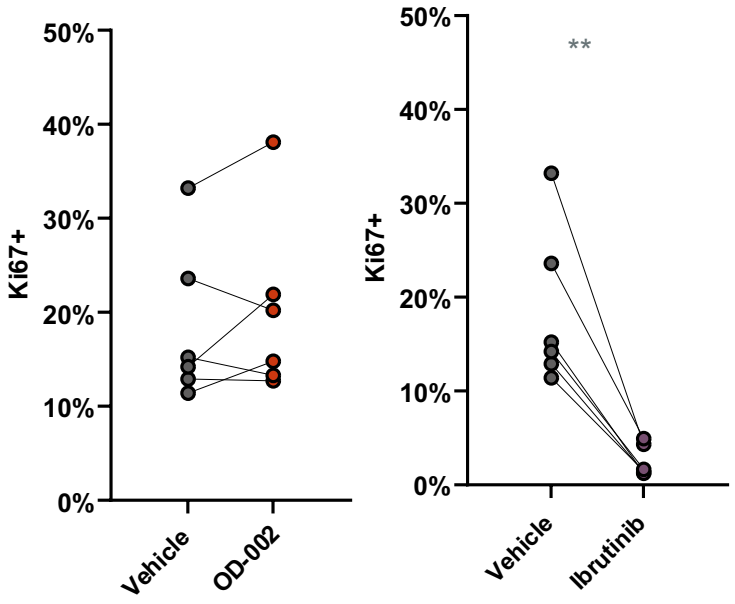


TLR9 stimulation induced by ODN-2395 in PBMCs from healthy donors

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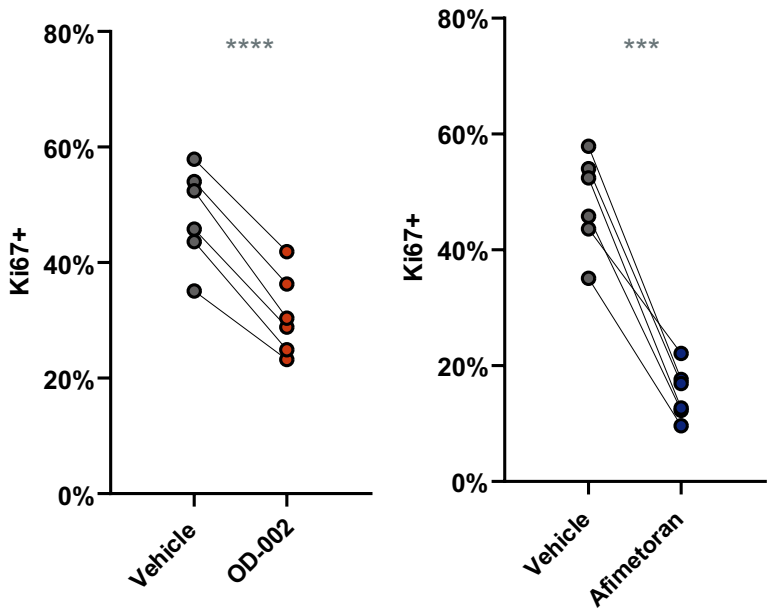
OD-002 Selectively Blocks Human B Cell Proliferation From Pathogenic Stimuli

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



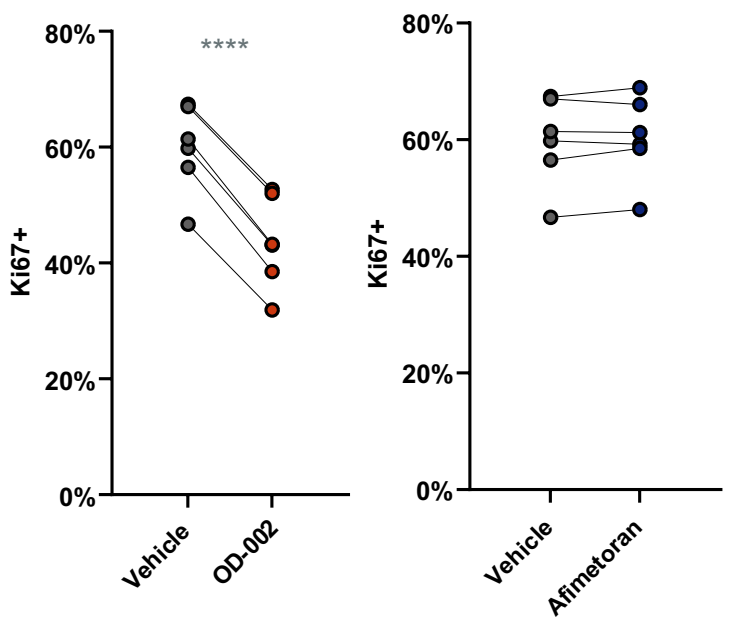
Ibrutinib, a BTK inhibitor, blocks non-pathogenic B cell proliferation

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



Afimetoran does not block TLR9-driven B cell proliferation

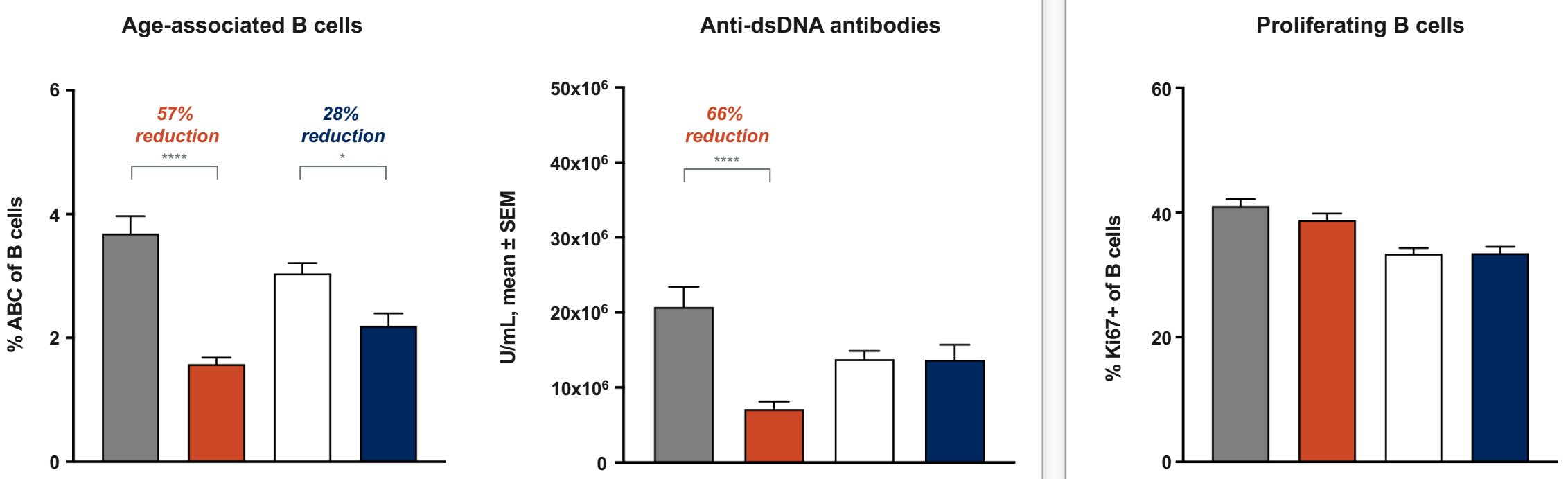
Notes: Isolated memory B cells from healthy donors were pretreated with indicated compound for 24 hours prior to stimuli and % Ki67+ was measured after three days; ** $p < 0.01$; *** $p < 0.001$; **** $p < 0.0001$.

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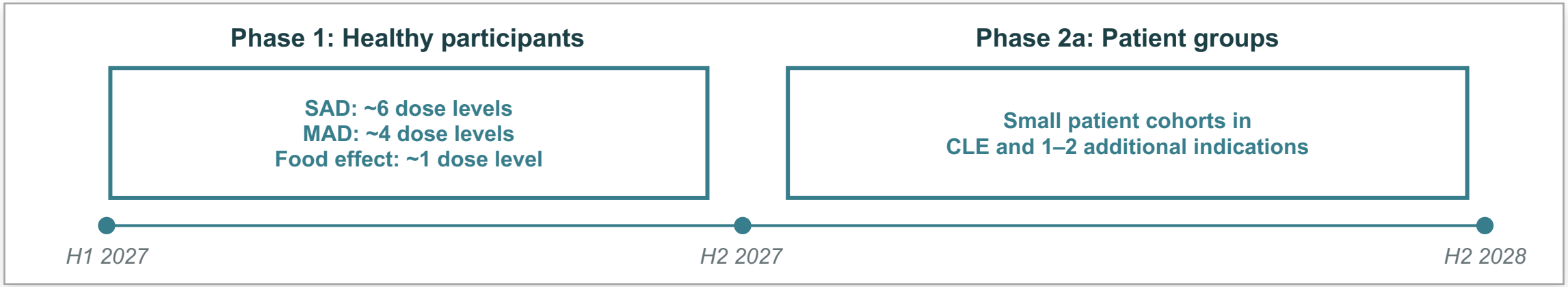
Phase 1/2a Trial Initiation Expected in H1 2027 With Efficacy Readouts Expected in H2 2028

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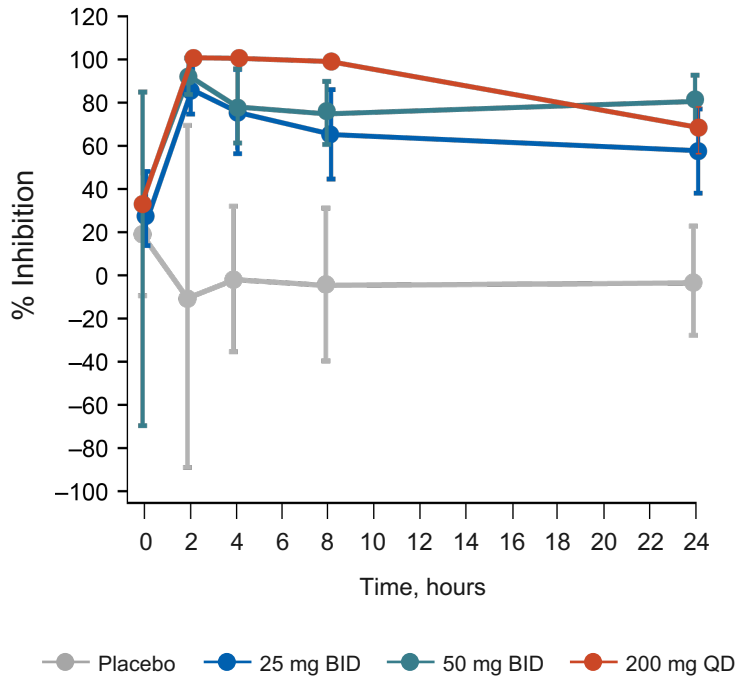
Phase 1 Ex Vivo Stimulation Assays Correlate With Activity in Lupus Patients

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

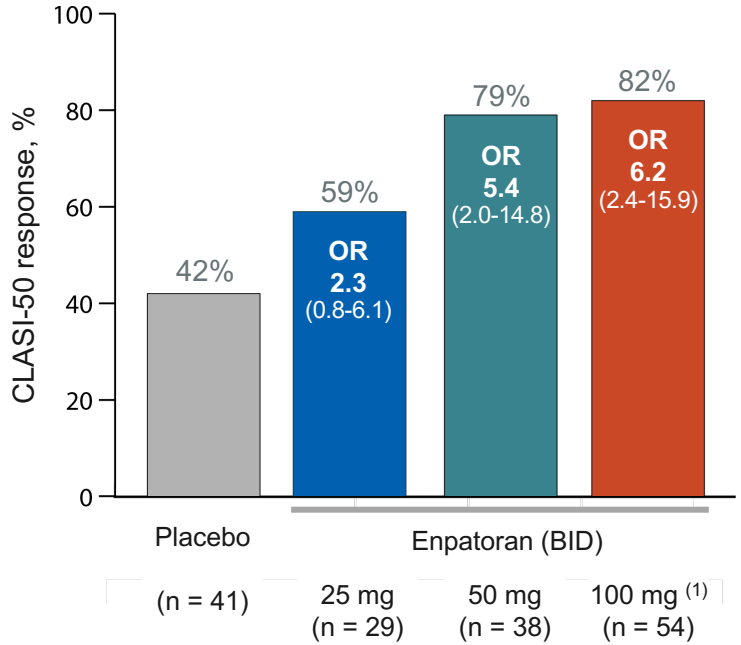
Enpatoran phase 1 ex vivo stimulation assay

- 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

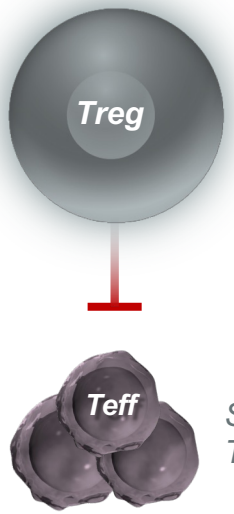
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Treg Therapy Has Broad Therapeutic Potential in Large I&I Diseases

Atopic dermatitis	SLE	Alopecia areata	Vitiligo	Multiple sclerosis	Rheumatoid arthritis	Asthma

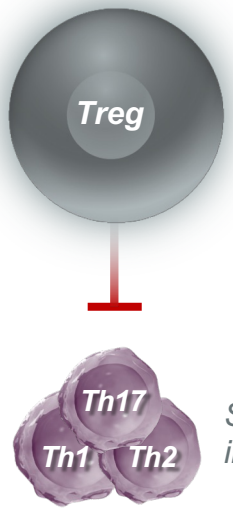
Correcting Treg dysregulation or increasing the number of Treg has the potential to be used across many inflammatory and autoimmune diseases

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



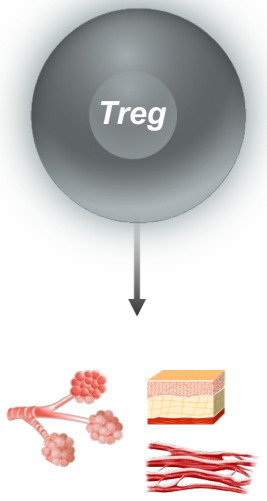
Stops Teff cells

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



Stops immune cells

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



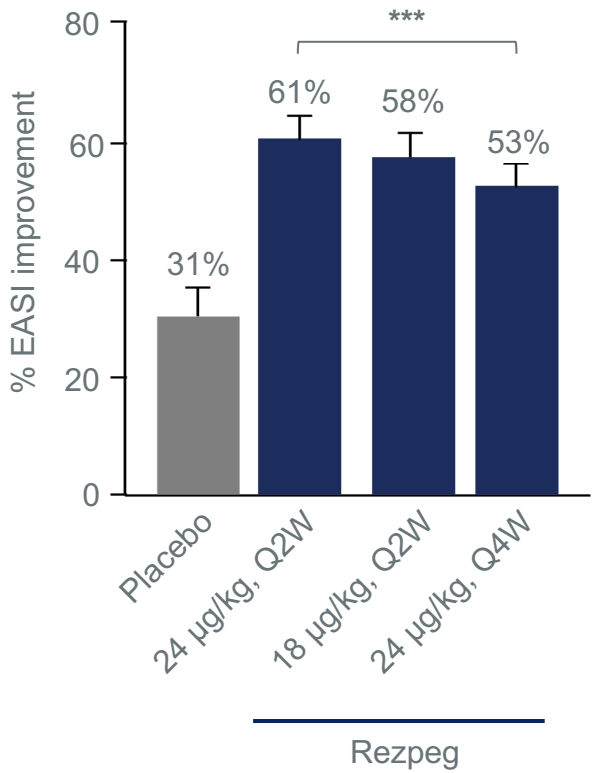
Repairs lung, skin, and muscle cells

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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

Stage of validation

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

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

Source: Evaluate Pharma.

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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 participant 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 Ph1/2a 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!