MORPHIC

Delivering A New Generation Of Integrin Medicines

March 2024

Forward-Looking Statements

This presentation contains "forward-looking" statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995, including, but not limited to statements regarding the timing and success of Morphic's ongoing clinical trials and related data, updates and results from Morphic's clinical trials and the potential therapeutic benefits of MORF-057.

Certain data in this presentation are based on cross-study comparisons and are not based on any head-to-head clinical trials. Cross-study comparisons are inherently limited and may suggest misleading similarities and differences. The values shown in the cross-study comparisons are directional and may not be directly comparable.

Statements including words such as "believe," "plan," "continue," "expect," "will," "develop," "signal," "potential," or "ongoing" and statements in the future tense are forward-looking statements. These forward-looking statements involve risks and uncertainties, as well as assumptions, which, if they do not fully materialize or prove incorrect, could cause our results to differ materially from those expressed or implied by such forward-looking statements.

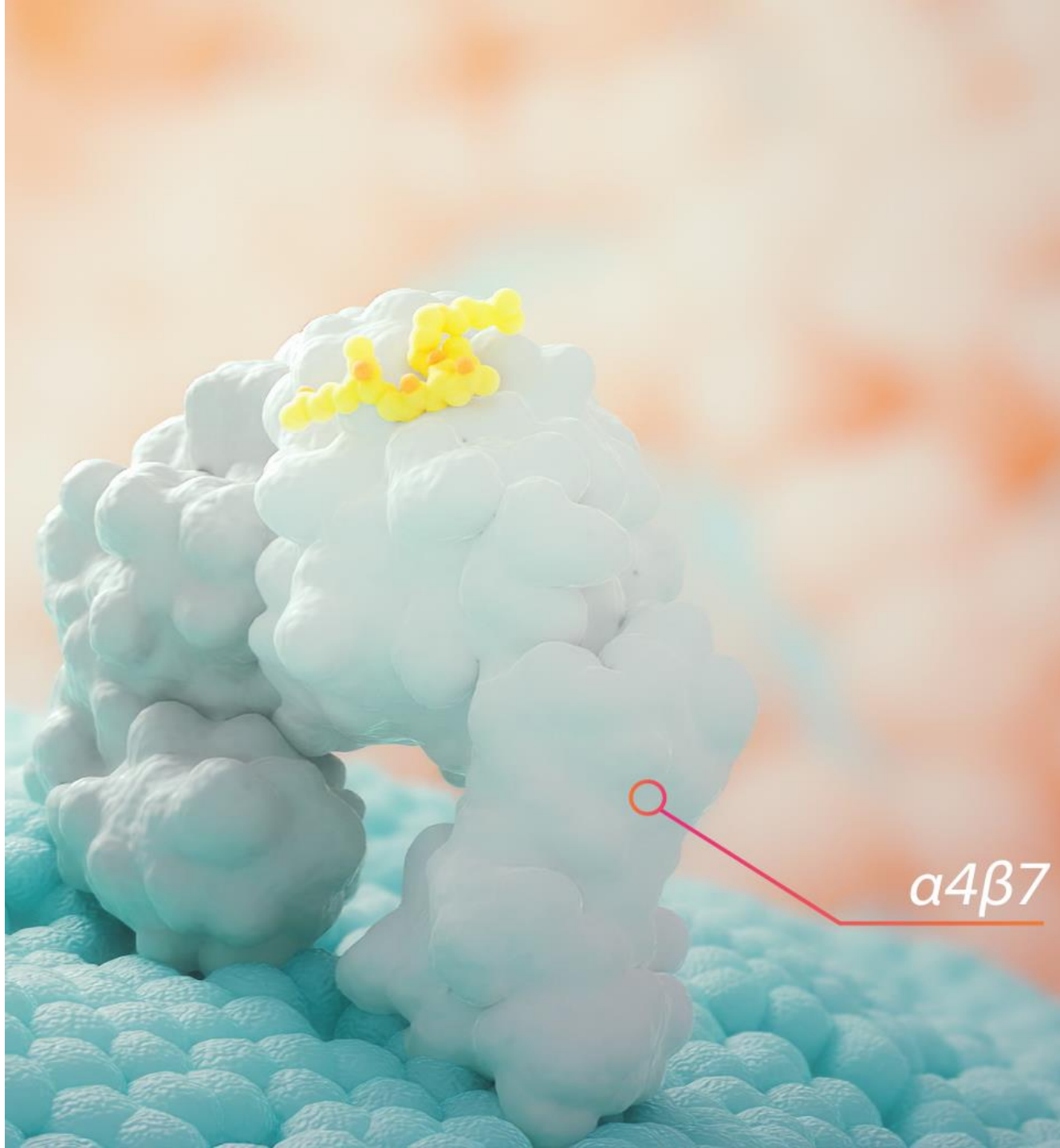
Forward-looking statements are subject to risks and uncertainties that may cause Morphic's actual activities or results to differ significantly from those expressed in or implied by any forward-looking statement, including risks and uncertainties related to the forward-looking statements in this presentation and other risks set forth in our filings with the Securities and Exchange Commission (SEC), including the Annual Report on Form 10-K for the fiscal year ended December 31, 2023 filed with the SEC on February 22, 2024, and the Quarterly Report on Form 10-Q for the fiscal quarter ended September 30, 2023 filed with the SEC on November 3, 2023. These forward-looking statements speak only as of the date hereof and Morphic specifically disclaims any obligation to update these forward-looking statements or reasons why actual results might differ, whether as a result of new information, future events or otherwise, except as required by law.

Note regarding trademarks: all third-party trademarks, including names, logos and brands, referenced by in this presentation are the property of their respective owners. All references to third-party trademarks are for identification purposes only and shall be considered nominative fair use under trademark law.

Unique Receptors: Unique Therapeutic Potential

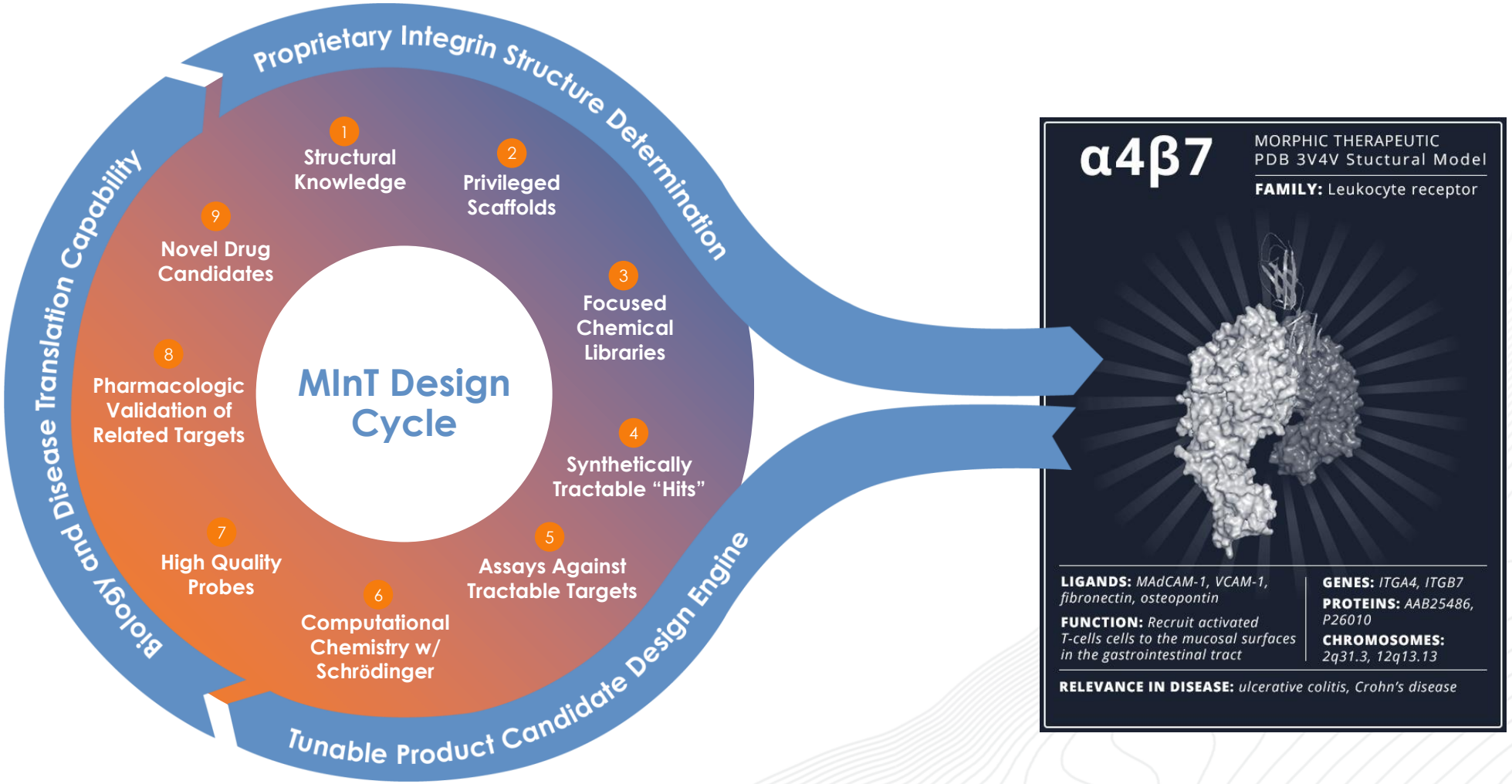
What are integrins?

- Only receptor to signal bidirectionally, giving them central biologic roles in complex diseases: autoimmune, fibrotic, cardio-metabolic and oncologic
- Expensive, complex biologics have shown clinically meaningful efficacy by targeting integrins



MORPHIC

MInT Platform: Morphic's Solution to the Oral Integrin Challenge



MORPHIC

Proprietary Pipeline

Candidate	Target (Program)	Indication	Discovery	IND-enabling	Phase 1	Phase 2	Phase 3
MORF-057	**$\alpha_4\beta_7$**	Ulcerative Colitis					
		Crohn's disease[1]					
Next-generation	**$\alpha_4\beta_7$**	GI Disorders					
MORF SMI[2]	**IL23, TL1A, etc**	Immune and Inflammatory Diseases					
MORF SMI	**$\alpha_5\beta_1$**	Pulmonary Hypertensive Diseases					
MORF-088	**$\alpha_v\beta_8$**	Myelofibrosis Solid Tumors					
MORF SMI/mAbs	**Undisclosed**	Multiple Indications					

MORPHIC

1 Crohn's disease phase 2 study anticipated to begin 1H24
2 SMI: oral small molecule inhibitor

MORPHIC

MORF-057

Small molecule inhibitor of $\alpha_4\beta_7$: a well-validated mechanism to treat IBD

EMERALD-2 Phase 2b study ongoing

IBD: Ideal Future Treatment Paradigm

WELL TOLERATED

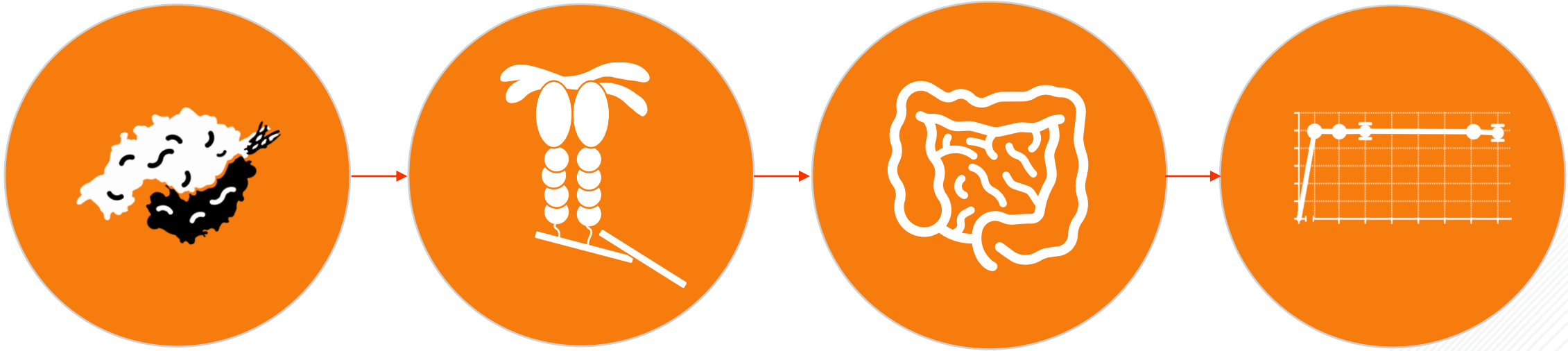
ORAL

EFFICACIOUS

COMBINABLE

MORPHIC

First-In-Class Oral Integrin Drug for IBD



MORF-057

Highly selective orally available small molecule inhibitor of $a_4\beta_7$, well validated mechanism for the treatment of IBD through approved monoclonal antibody vedolizumab

Mechanism

Occluding $a_4\beta_7$ blocks intestinal homing of lymphocytes, which in turn reduces pathologic inflammation in IBD

Indications

Inflammatory bowel disease with initial focus on ulcerative colitis

Approximately 1.6 million Americans currently have irritable bowel disease [1]

Clinical Data

Clinically meaningful and consistent activity data across multiple validated efficacy measures in Phase 2a study

Well tolerated to date across multiple clinical trials

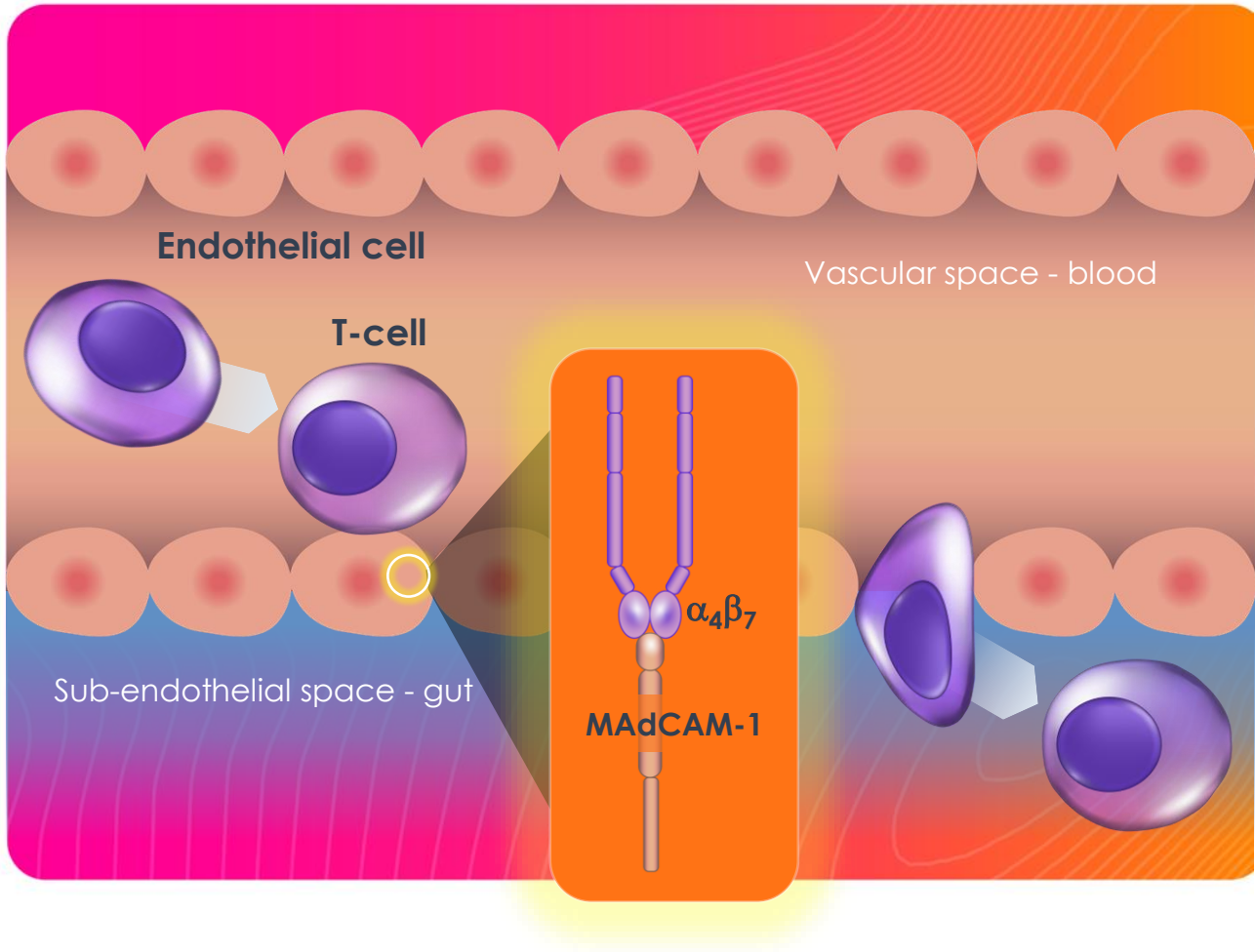
Phase 2b ongoing in UC, Crohn's disease to begin 1H24

MORPHIC

[1] *Chrohn's and Colitis Foundation of America*

$a_4\beta_7$ Inhibition is a Proven Mechanism to Treat IBD

- **Approved antibody Entyvio® (vedolizumab)**
- Vedolizumab, an anti-$a_4\beta_7$ antibody, inhibits T-cell trafficking via well validated mechanism to treat UC and Crohn's disease
- Since approval, over 265,000 patients have received vedolizumab[1]
- Vedolizumab generated $5.2B sales in FY2022[2]



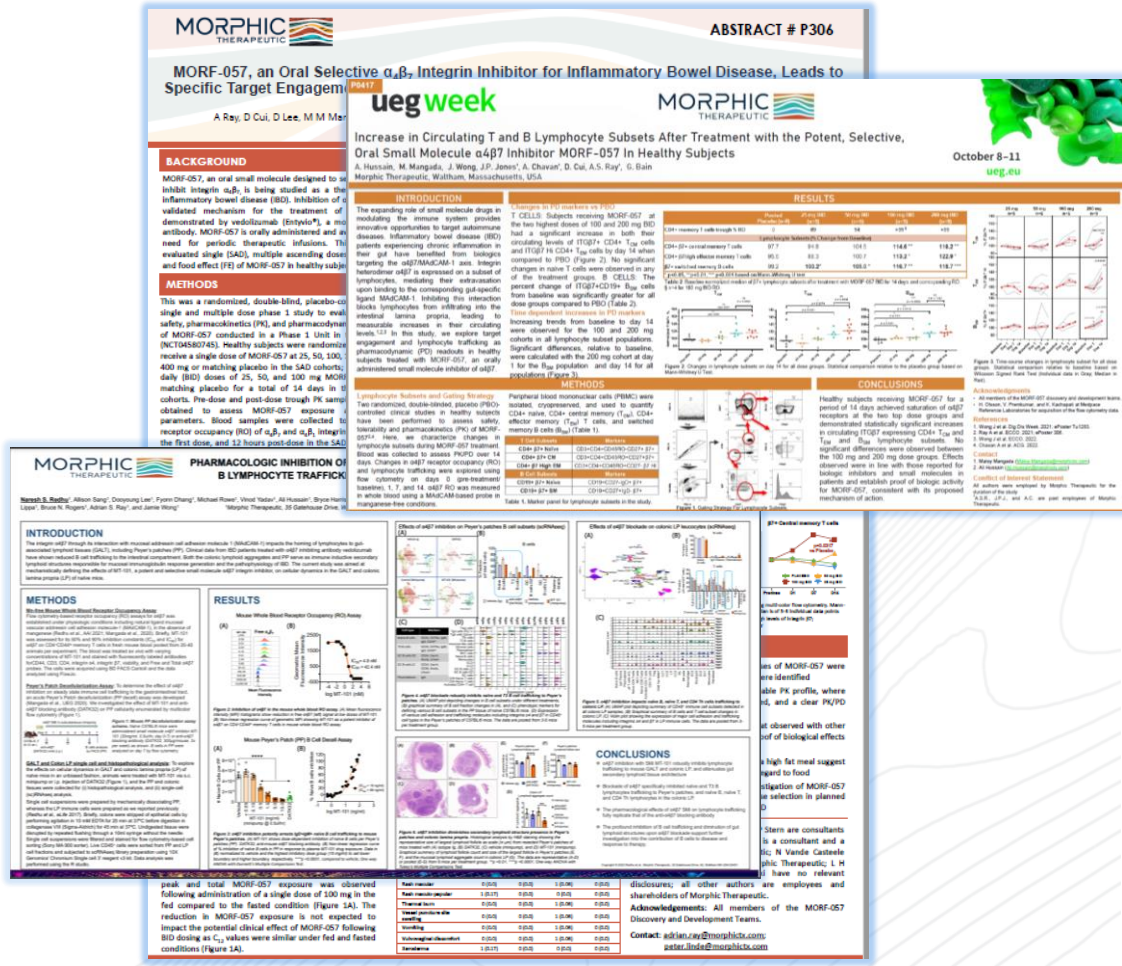
MORPHIC

[1]Takeda press release
[2]Global Data
ENTYVIO® is a registered trademark of Millennium Pharmaceuticals, Inc.

MORF-057 has Consistently Delivered on Expectations for an Oral $\alpha_4\beta_7$ Inhibitor in IBD

	MORF-057		
	PRECLINICAL	PHASE 1	PHASE 2a
Meaningful Clinical Effects			✓
30-50% ↑ in Key Lymphocytes		✓	✓
α4β7 Saturation (serum)	✓	✓	✓
Favorable Tolerability Profile	✓	✓	✓
Oral Route of Administration	✓	✓	✓

Please click on links in row headings above for underlying data



All scientific posters and presentations available at https://investor.morphictx.com

MORPHIC

EMERALD-1

Phase 2a Study of MORF-057

MORF-057 Phase 2a: EMERALD-1Study in Moderate to Severe UC

EMERALD

Phase 2a open-label single-arm study of MORF-057 (100mg BID) in patients with moderately to severely active ulcerative colitis (n=35 main cohort)

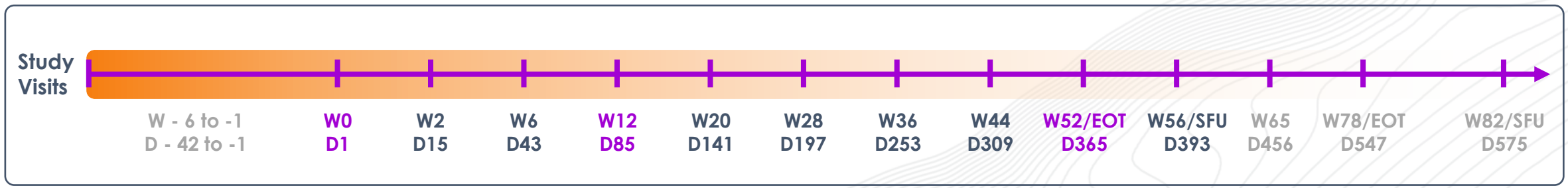
Phase 2a

- Primary endpoint: Change in RHI measured at 12 weeks
- Secondary endpoints: mMCS change from baseline, safety
- Pre-specified exploratory endpoints:
  - RHI remission
  - mMCS remission
  - mMCS response
  - Multiple PK/PD parameters
  - Relevant biomarkers

Open-Label Treatment Period
MORF-057 (100mg B.I.D. P.O.)
for 52 weeks
Primary endpoint at week 12

Long-Term Extension
Direct rollover to extension
MORF-057 (100mg B.I.D. P.O.)
for 26 weeks

Safety Follow-Up Period
Safety Follow-up Visit
to occur 4 weeks
after last dose of study drug



MORPHIC

Data from the 52-week readout of the EMERALD-1 phase 2a study of MORF-057 in ulcerative colitis, including the 40-week maintenance phase of the main cohort and from the 12-week induction phase of the exploratory cohort of four patients of secondary non-responders to vedolizumab, have been collected and analyzed. No safety signals have been identified in either cohort. Morphic believes the 52-week readout, including safety, clinical efficacy and pharmacokinetic/pharmacodynamic measures, are substantially consistent with data trends from the 12-week induction phase and the 44-week readout that we reported in October 2023 for EMERALD-1. The Company is preparing a manuscript for submission and intends to publish the EMERALD-1 data set in an appropriate medical journal or forum as soon as practicable, pending review and acceptance of these data.

Baseline Patient Demographics: a Moderately-to-Severely Active UC population with High Disease Burden

Category		Patients, N=35
Age, mean ± SD	Years	39.2 ± 14.1
Sex, n (%)	Female	16 (45.7)
Geography, n (%)	Poland United States	28 (80.0) 7 (20.0)
Duration of disease, mean ± SD	Years	7.5 ± 8.0
Extent of disease, n (%)	Proctosigmoiditis L-sided colitis Pancolitis	12 (34.3) 10 (28.6) 10 (28.6)
RHI Score, mean ± SD	Points	22.7 ± 7.3
mMCS, mean ± SD	Points	6.7 ± 1.1
MES, n (%)	2 3	18 (51.4) 17 (48.6)
Corticosteroid use, n (%)	No Yes	26 (74.3) 9 (25.7)
Previous use of AT*, n (%)	Naïve Experienced	21 (60.0) 14 (40.0)

AT, advanced therapy; MES, Mayo endoscopic score; mMCS, modified Mayo Clinic score; RHI, Robarts histopathology index; SD, standard deviation

*The number of AT-experienced patients was updated from n=13/35 to n=14/35 during re-review of data for presentation at a medical conference. During this re-review, It was determined that one patient had received an investigational agent deemed to be an advanced therapy before the MORF-057-201 trial. This change does not impact any of the clinical efficacy data presented from the EMERALD-1 study.

MORPHIC

EMERALD-1

12-week Induction Phase

Data as of 4/25/23

Generally Well-Tolerated in EMERALD-1
No Safety Signal Observed

Adverse Event (AE) profile consistent with underlying disease state

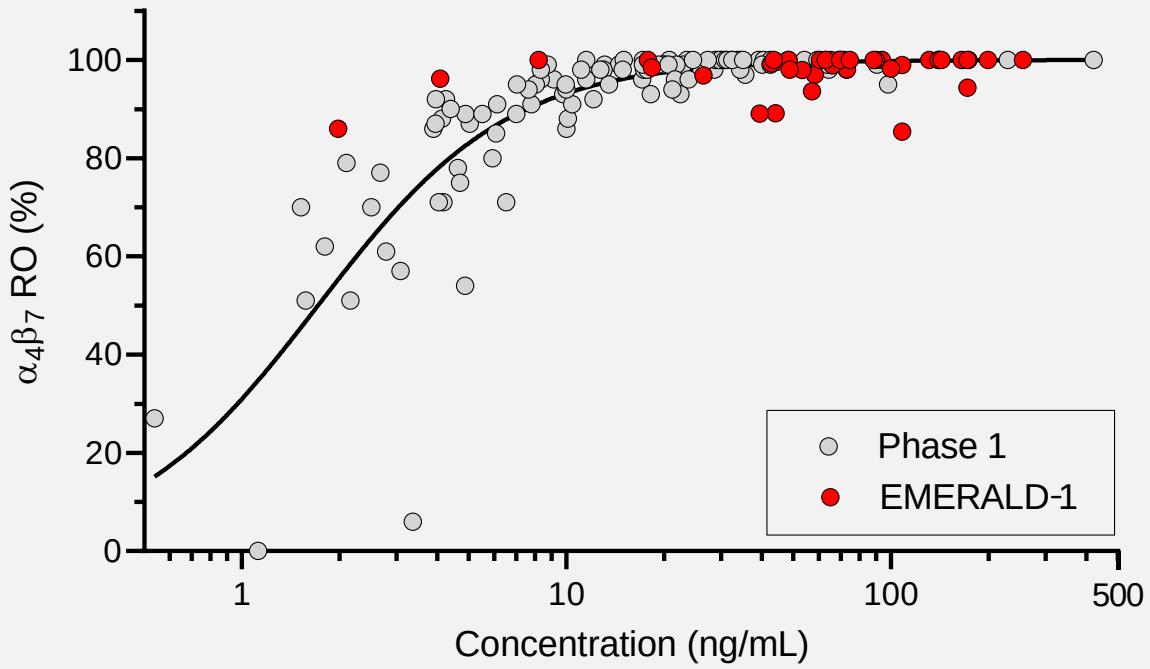
Patients with at least one AE	**12 (34.3%)**
Patients with any serious AE	**0**
Patients with AE leading to death	**0**
Patients with any grade 3 AE	**2 (5.7%)[1]**
Patients with treatment-related AE	**2 (5.7%)**
Common (>5%) AEs Exacerbation of UC Anemia	 **4 (11.4%)** **3 (8.6%)[2]**

MORPHIC

1. Both UC exacerbations, one led to early discontinuation
2. All anemic at baseline and continued on study with iron supplements

*Safety data as of 4/25/23 induction presentation. As of 3/12/24, patients have been on EMERALD-1 study beyond the 52-week maintenance phase and no safety signals have been reported.

Patient α4β7 Receptor Occupancy (RO) Consistent with Healthy Volunteer RO



α4β7 selectivity over α4β1 consistent with Phase 1 results

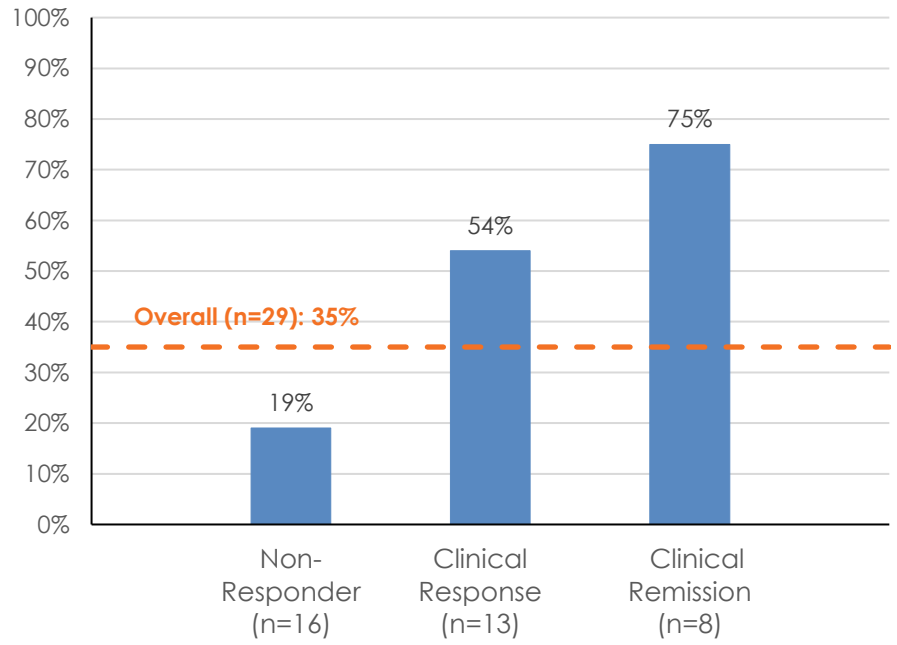
RO at 12 weeks		
	α4β7	α4β1
Mean	**>98%**	BLQ
Median	**>99%**	BLQ

- α4β7 RO achieved early and sustained saturating levels
- α4β1 RO remained at low levels
- No lymphocytosis or changes to circulating naïve T-cells were observed
- α4β1 projected RO was below the limit of quantitation with mean trough value estimated to be <15%

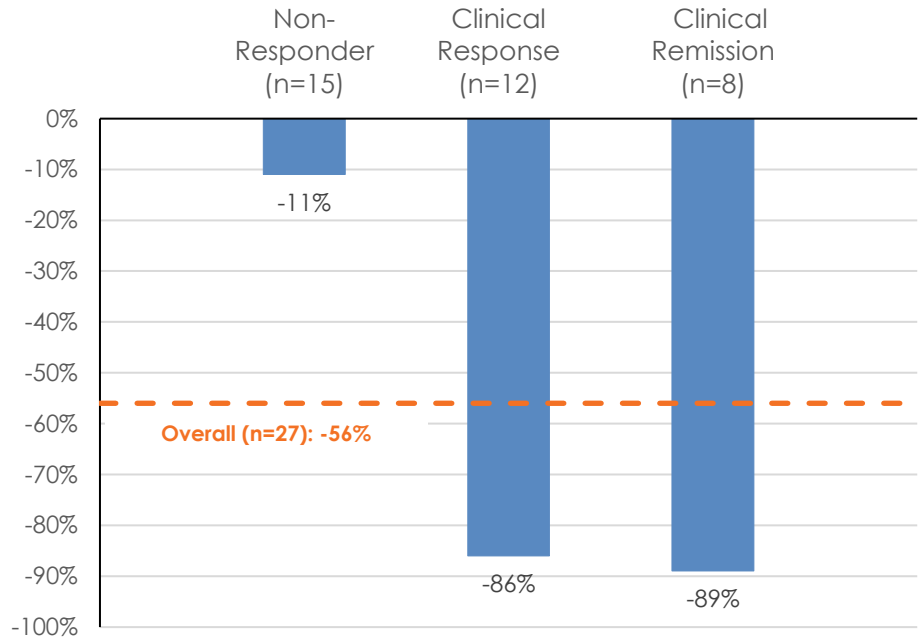
MORPHIC

Fecal Calprotectin Decreases Correlated with Disease Improvement

Proportion of Patients with Fecal Cal < 250 mg/kg at Week 12 (Baseline > 250 mg/kg), n=29



Percentage Reduction From Baseline in Fecal Cal at Week 12 (Baseline > 250 mg/kg & Week 12 data available), n=27[a]

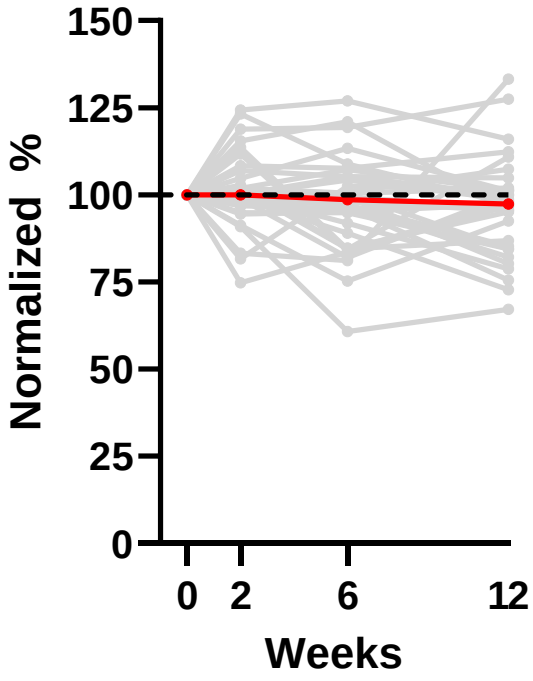


n = Patients with baseline FC > 250 mg/kg. No inclusion/exclusion criteria for FC levels
Patients experiencing clinical remission also included in clinical response
a. Data unavailable for 2 patients at week 12

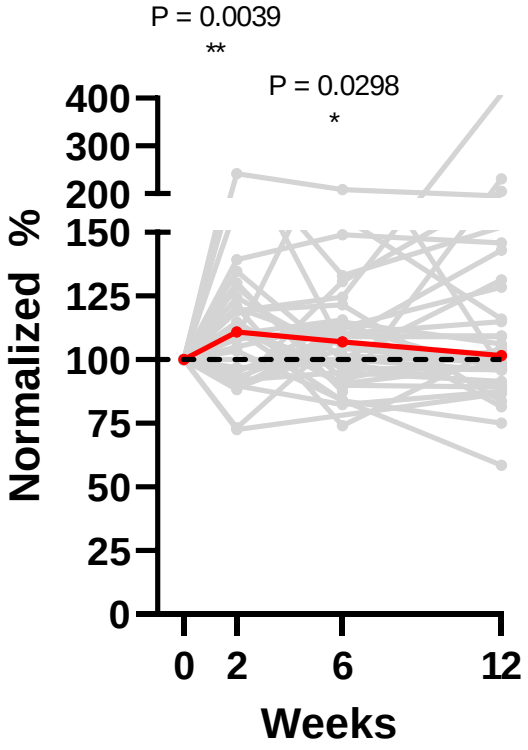
Substantial Lymphocyte Subset Changes Observed, Consistent With Engagement Of a4β7

CD4 Expressing T Cells

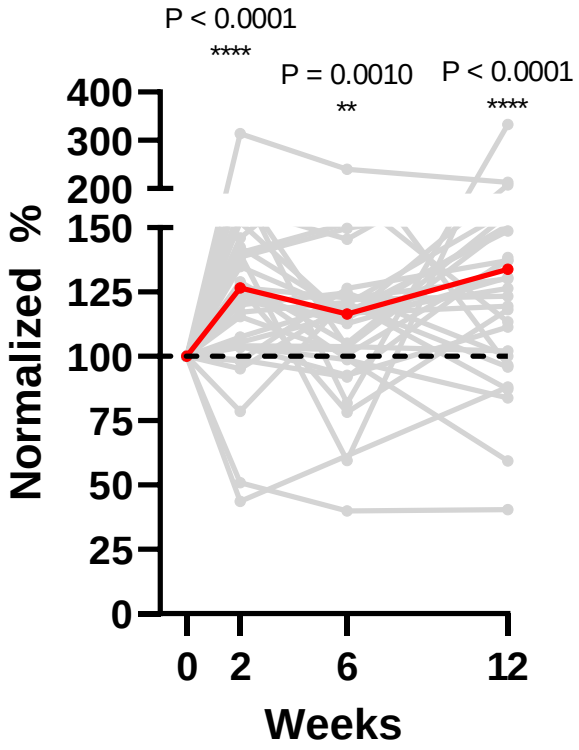
Naïve (Neg. Control)



Central Memory (ITGB7+)

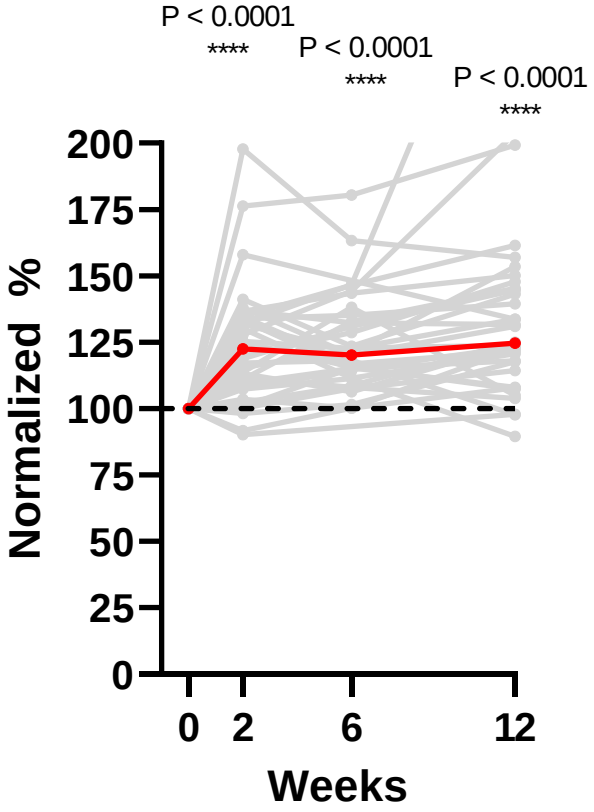


Effector Memory (ITGb7 Hi)



B Cells

Switched Memory (ITGb7+)



Median of all patients
Individual patients

MORPHIC

MORPHIC

EMERALD-1
Induction Phase

Clinical Efficacy Results

Primary Endpoint Met with Statistical Significance

Consistent Effects Observed Among All Exploratory Measures

Endpoint @ Week 12	Overall (N=35)
Change in RHI, Mean (SD)	**-6.4 (11.18)** *p=0.0019*
RHI remission, n (%)	8 (22.9%)
Clinical response (mMCS)[1], n (%)	16 (45.7%)
Clinical remission (mMCS)[2], n (%)	9 (25.7%)
Endoscopic Response/Improvement[3], n (%)	9 (25.7%)
Change from baseline to Week 12 in the Modified MCS, Mean (SD)	-2.3 (2.14)

1. Clinical response (mMCS): decrease from baseline in the mMCS ≥2 points and ≥30% from baseline, plus a decrease in rectal bleeding subscore ≥1 or an absolute rectal bleeding subscore ≤1
2. Clinical remission (mMCS): rectal bleeding subscore of 0; a stool frequency subscore of ≤1; and an MES of ≤1 without friability
3. Endoscopic response / improvement: MES ≤1

MORPHIC

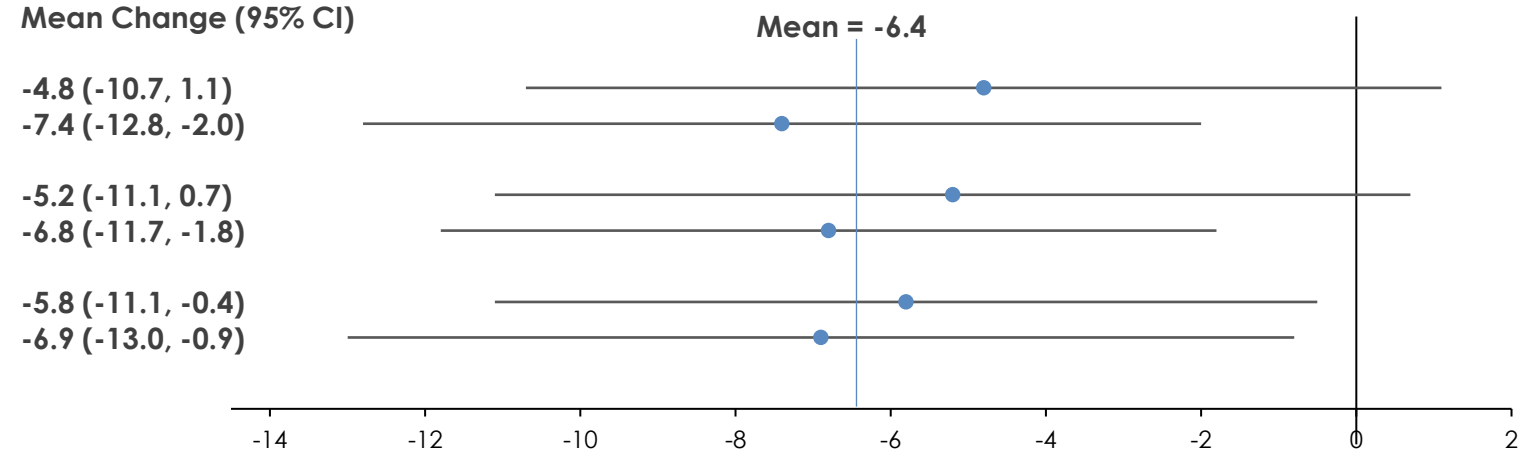
EMERALD-1 Efficacy Results by AT Status and MES

Endpoint @ Week 12	Overall N=35	AT-naïve n=21	AT-experienced n=14	MES =2 n=18	MES =3 n= 17
Change in RHI, mean ± SD	**-6.4 ± 11.2**	**-7.4 ± 11.9**	**-4.8 ± 10.3**	**-6.9 ± 12.1**	**-5.8 ± 10.4**
RHI change ≥ 7 points, n (%)	17 (48.6)	12 (57.1)	5 (35.7)	10 (55.6)	7 (41.2)
RHI remission[1], n (%)	8 (22.9)	6 (28.6)	2 (14.3)	6 (33.3)	2 (11.8)
RHI reduction ≥ 50%, n (%)	12 (34.3)	9 (42.9)	3 (21.4)	9 (50.0)	3 (17.6)
Change in mMCS, mean ± SD	-2.3 ± 2.1	-2.9 ± 2.4	-1.6 ± 1.5	-2.7 ± 2.3	-1.9 ± 1.9
Clinical response (mMCS)[2], n (%)	16 (45.7)	11 (52.4)	5 (35.7)	9 (50)	7 (41.2)
Clinical remission (mMCS)[3], n (%)	9 (25.7)	9 (42.9)	0	6 (33.3)	3 (17.6)
Symptomatic remission[4], n (%)	11 (31.4)	10 (47.6)	1 (7.1)	7 (38.9)	4 (23.5)
Endoscopic response / improvement[5], n (%)	9 (25.7)	9 (42.9)	0	6 (33.3)	3 (17.6)
Change in SF, mean ± SD	-0.8 ± 1.1	-1.0 ± 1.2	-0.5 ± 0.7	-0.9 ± 1.3	-0.6 ± 0.8
Change in RB, mean ± SD	-1.1 ± 0.8	-1.1 ± 0.9	-0.9 ± 0.8	-1.4 ± 0.8	-0.7 ± 0.7

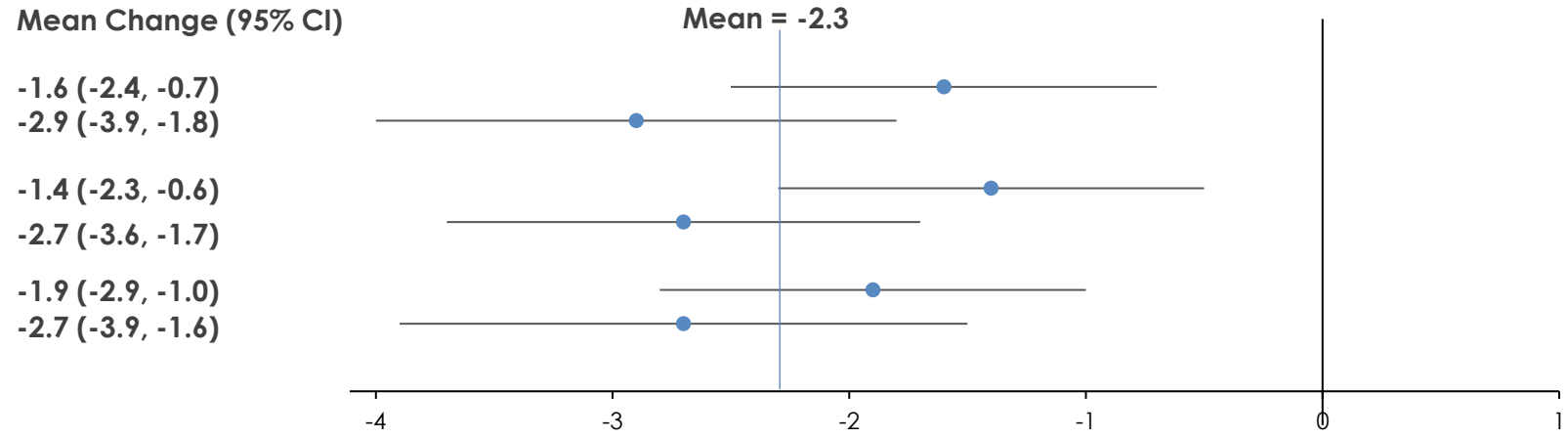
AT, advanced therapy; MCS, Mayo Clinic Score; mMCS, modified MCS; RHI, Robarts histopathology index; SF, Stool Frequency; RB, Rectal Bleeding; SD, standard deviation
1. RHI Remission: RHI ≤ 2
2. Clinical response (mMCS): decrease from baseline in the mMCS ≥2 points and ≥30% from baseline, plus a decrease in rectal bleeding subscore ≥1 or an absolute rectal bleeding subscore ≤1
3. Clinical remission (mMCS): rectal bleeding subscore of 0; a stool frequency subscore of ≤1; and an MES of ≤1 without friability
4. Symptomatic remission: SFS = 0 (or = 1 with ≥ 1 point decrease from baseline) and RBS = 0
5. Endoscopic response/improvement: MES ≤1

Consistent "Across-the-Board" Efficacy Signals Observed

Robarts Histopathology Index (RHI)

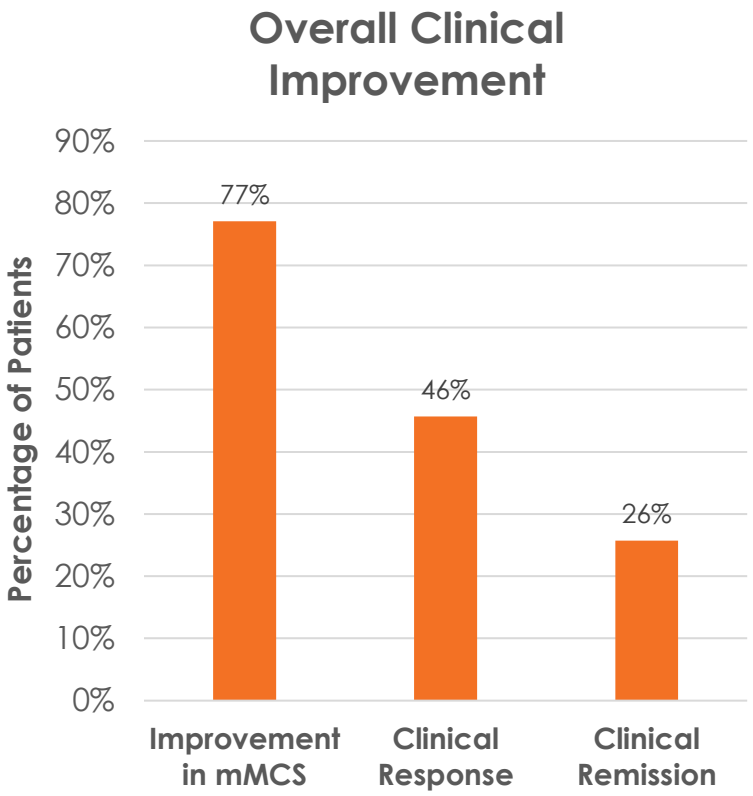


Modified Mayo Clinical Score (mMCS)

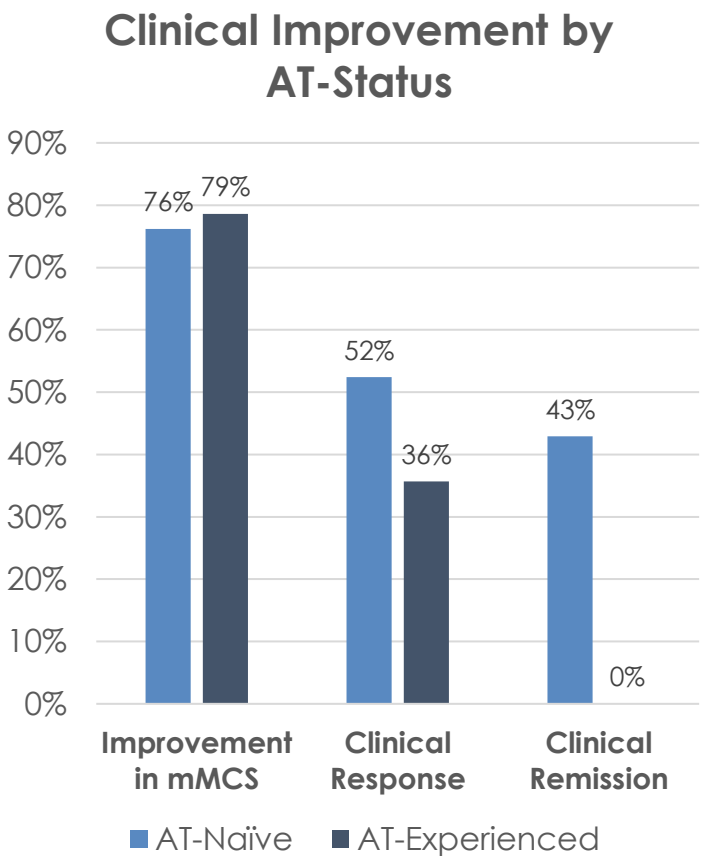


MORPHIC

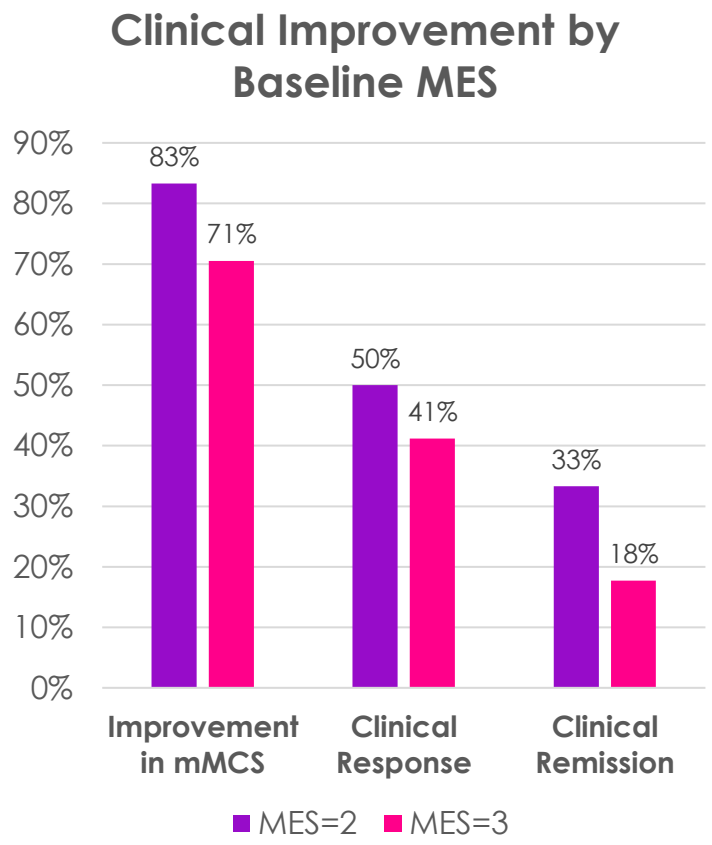
Clinical Improvement in >75% of All Patients, Regardless of Prior Therapy and Baseline MES



N=35



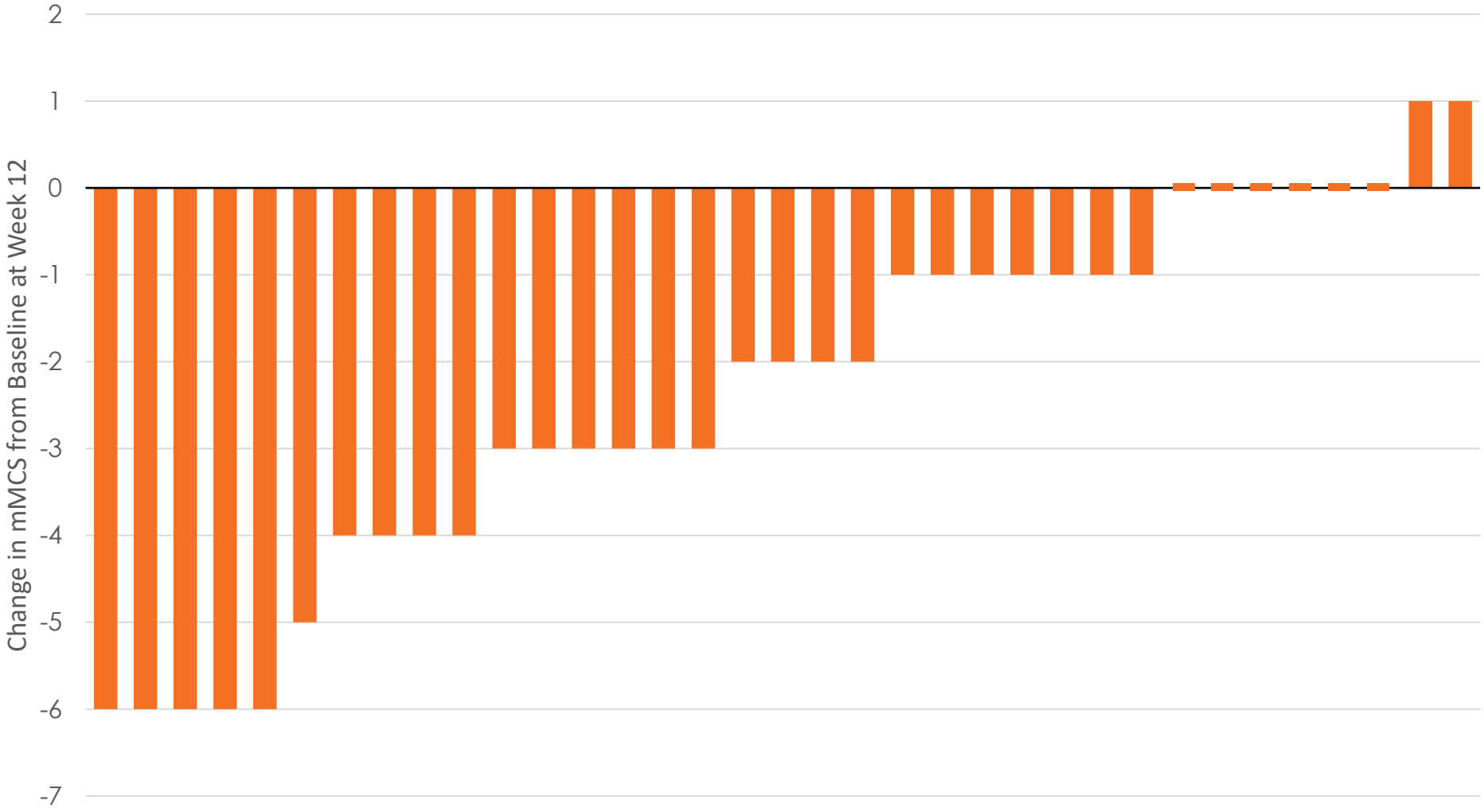
AT-Naïve: n=21; AT-Experienced: n=14



Baseline MES=2: n=18; Baseline MES=3: n=17

MORPHIC

Change in Central mMCS By Patient from Baseline at Week 12



MORPHIC

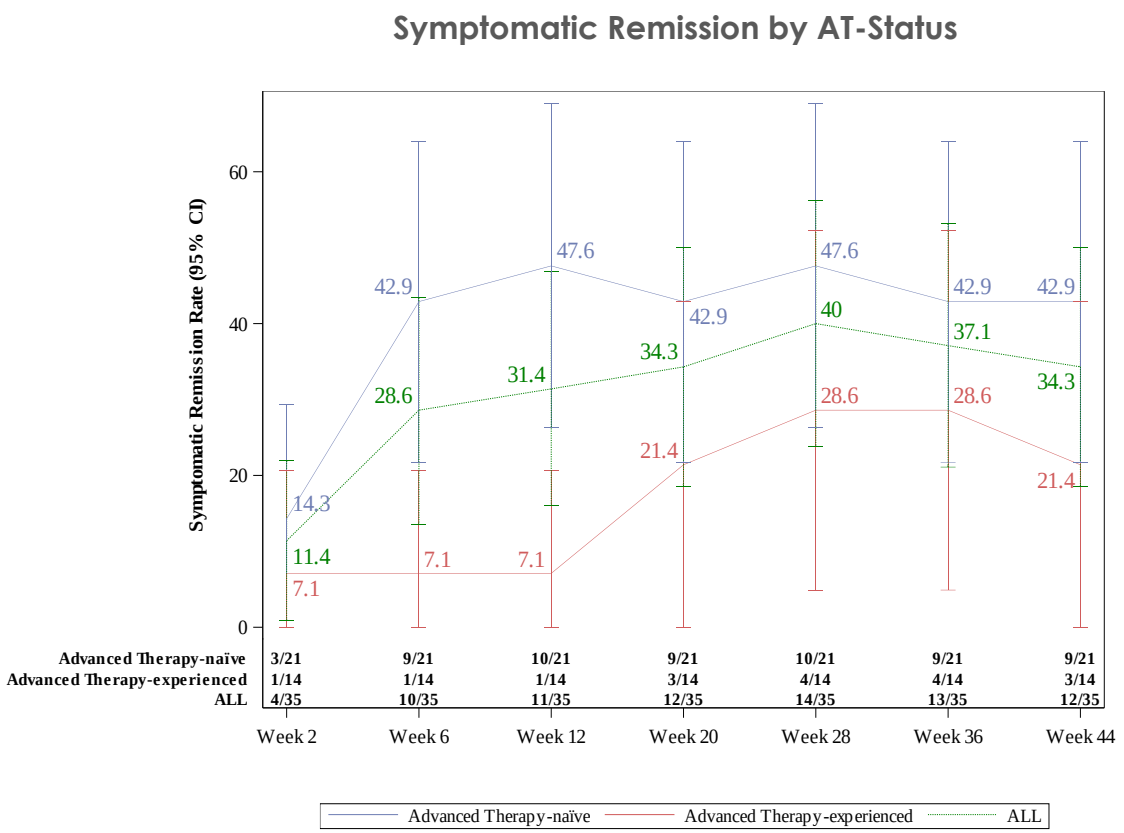
MORPHIC

EMERALD-1

Data Beyond 12 Weeks

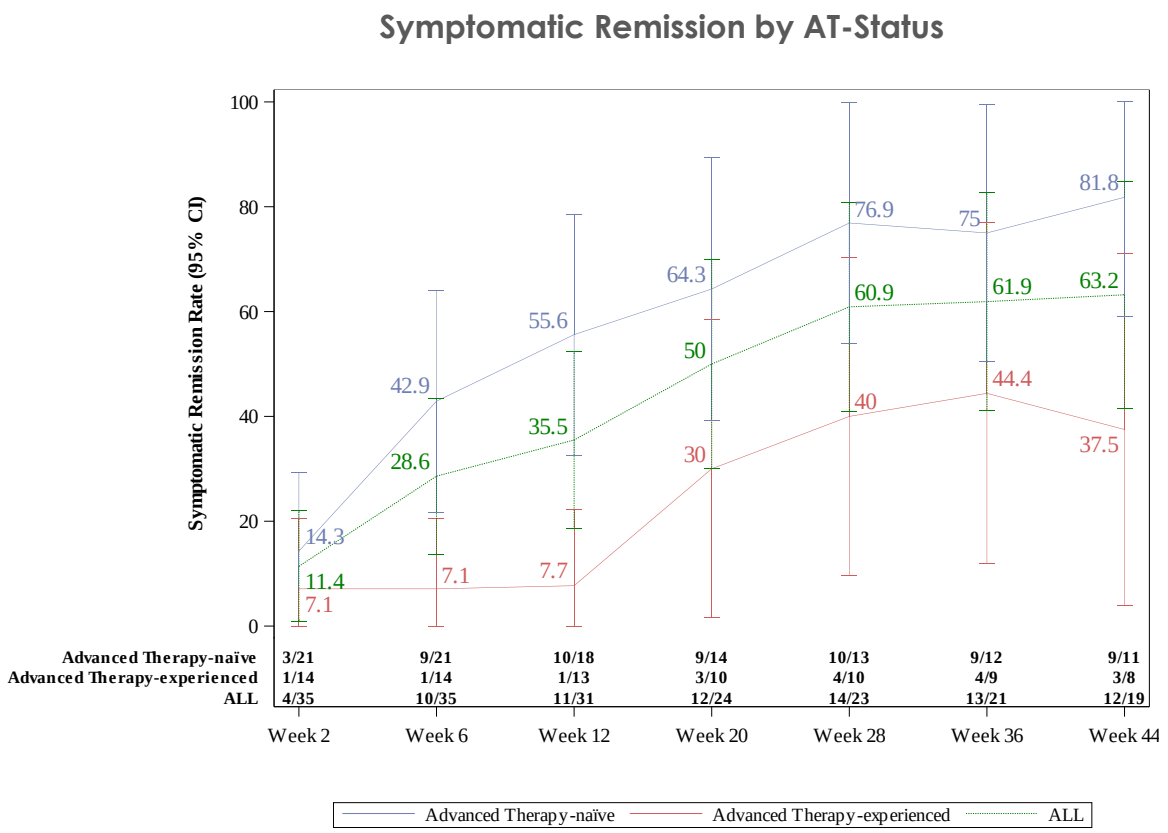
Symptomatic Remission By AT-Status: Week 44

Intent to Treat (ITT): Denominator includes all enrolled patients (N=35)



Symptomatic remission is defined as an stool frequency subscore=0 (or =1 with a >=1-point decrease from baseline) and rectal bleeding subscore=0

As observed: Denominator includes only patients who completed the visit



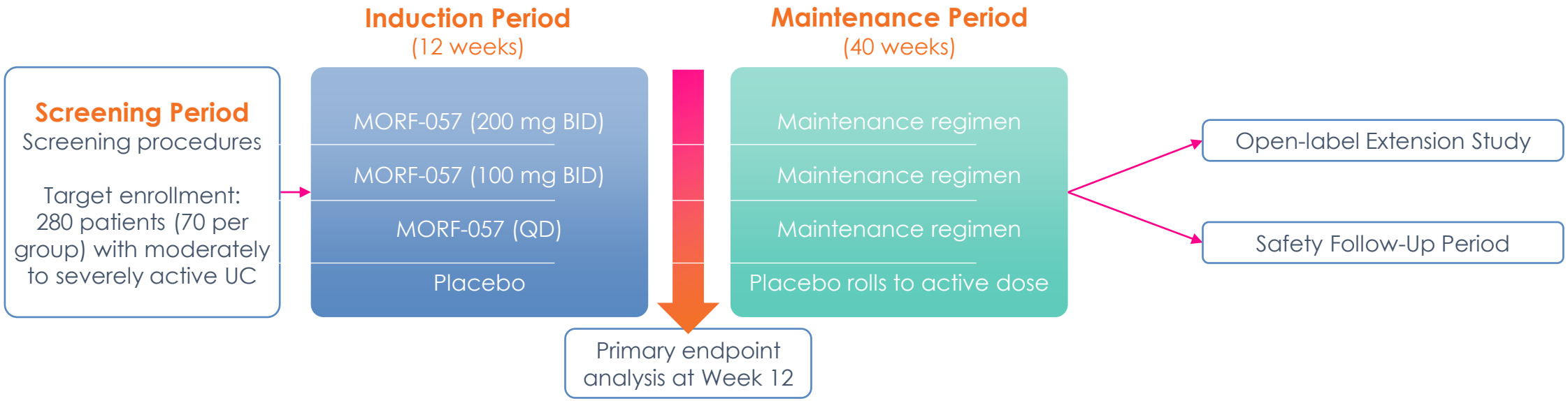
Symptomatic remission is defined as an stool frequency subscore=0 (or =1 with a >=1-point decrease from baseline) and rectal bleeding subscore=0

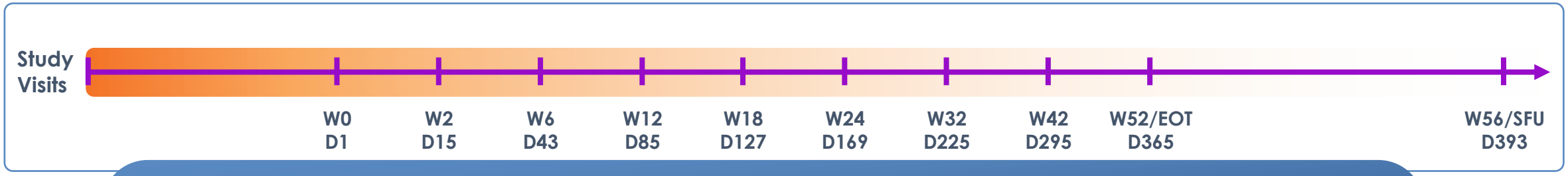
MORPHIC

Note: These are ad hoc analyses and are subject to change during the quality control and trial completion processes

MORF-057 Phase 2b: EMERALD-2 Study in Moderate to Severe UC

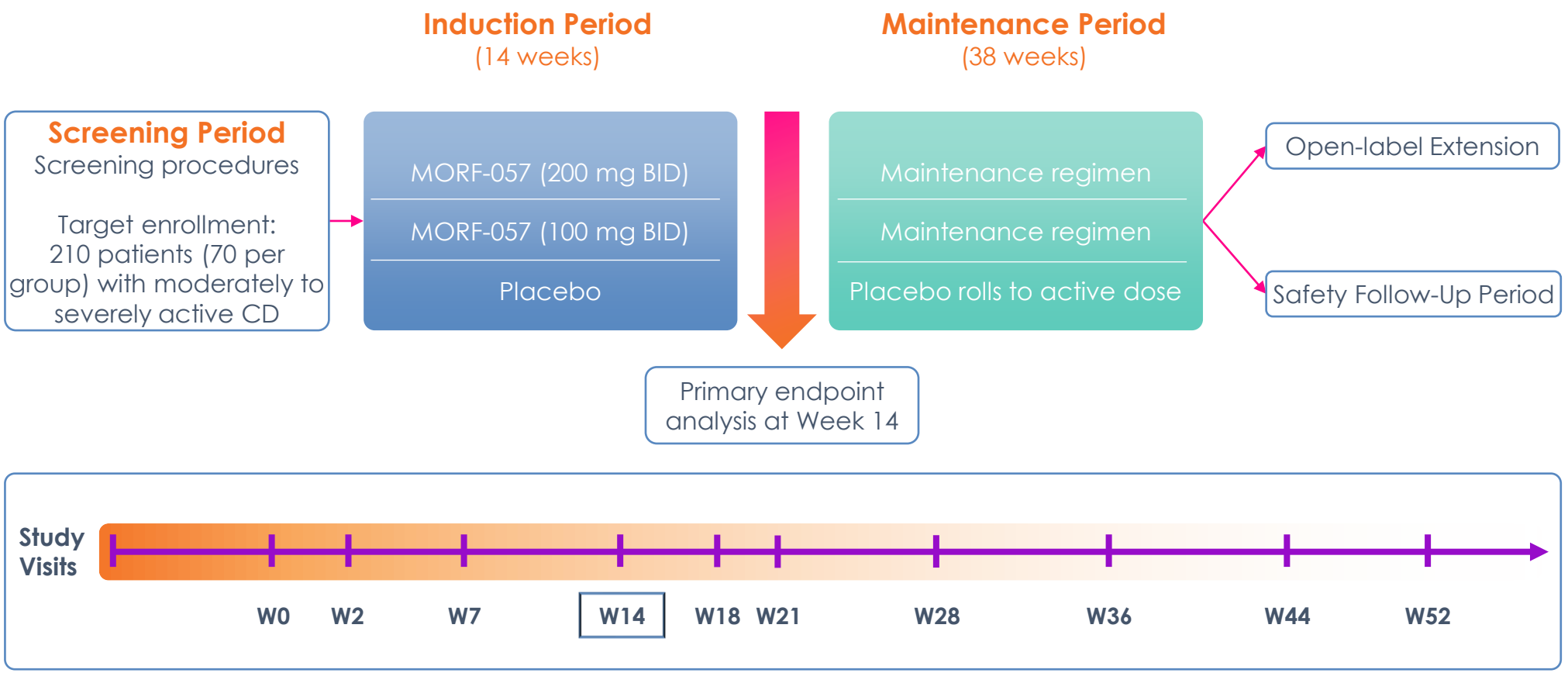
EMERALD





Primary Endpoint: **proportion of participants in clinical remission at Week 12 as determined using the Modified Mayo Clinic Score (mMCS)**

GARNET Phase 2 Study of MORF-057 in Moderate to Severe Crohn's Disease



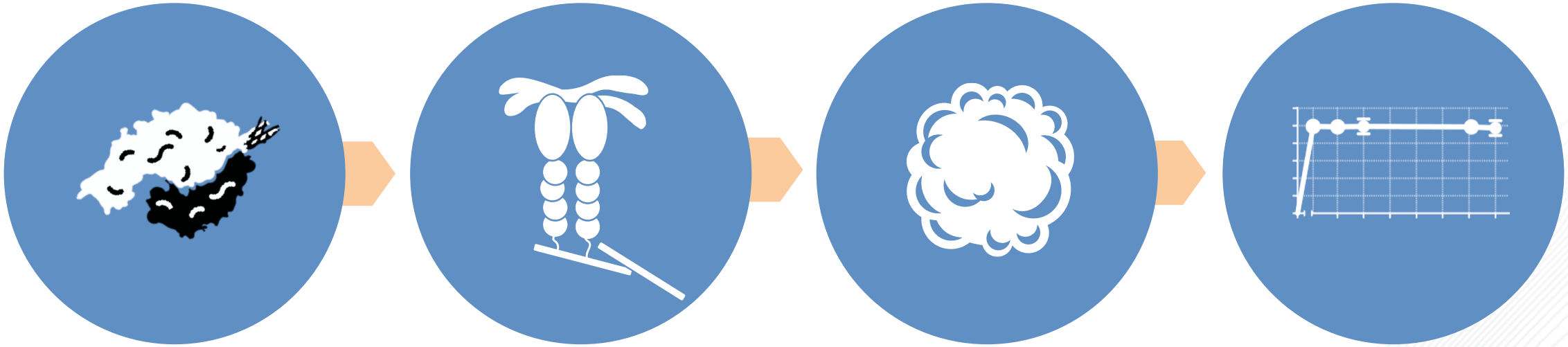
***Primary Endpoint*: proportion of patients with endoscopic response (>=50% reduction) at week 14 as determined using SES-CD**

MORPHIC

EMERGING PIPELINE

Creating the next generation of proprietary integrin inhibitor candidates

$\alpha_5\beta_1$: Small Molecule Integrin Inhibitor for Pulmonary Hypertensive Diseases



Program

Small molecule inhibitors of fibronectin integrins in preclinical development

Mechanism

Fibronectin integrin inhibition suppresses pulmonary arterial smooth muscle cell proliferation

Indications

Multiple pulmonary hypertensive diseases

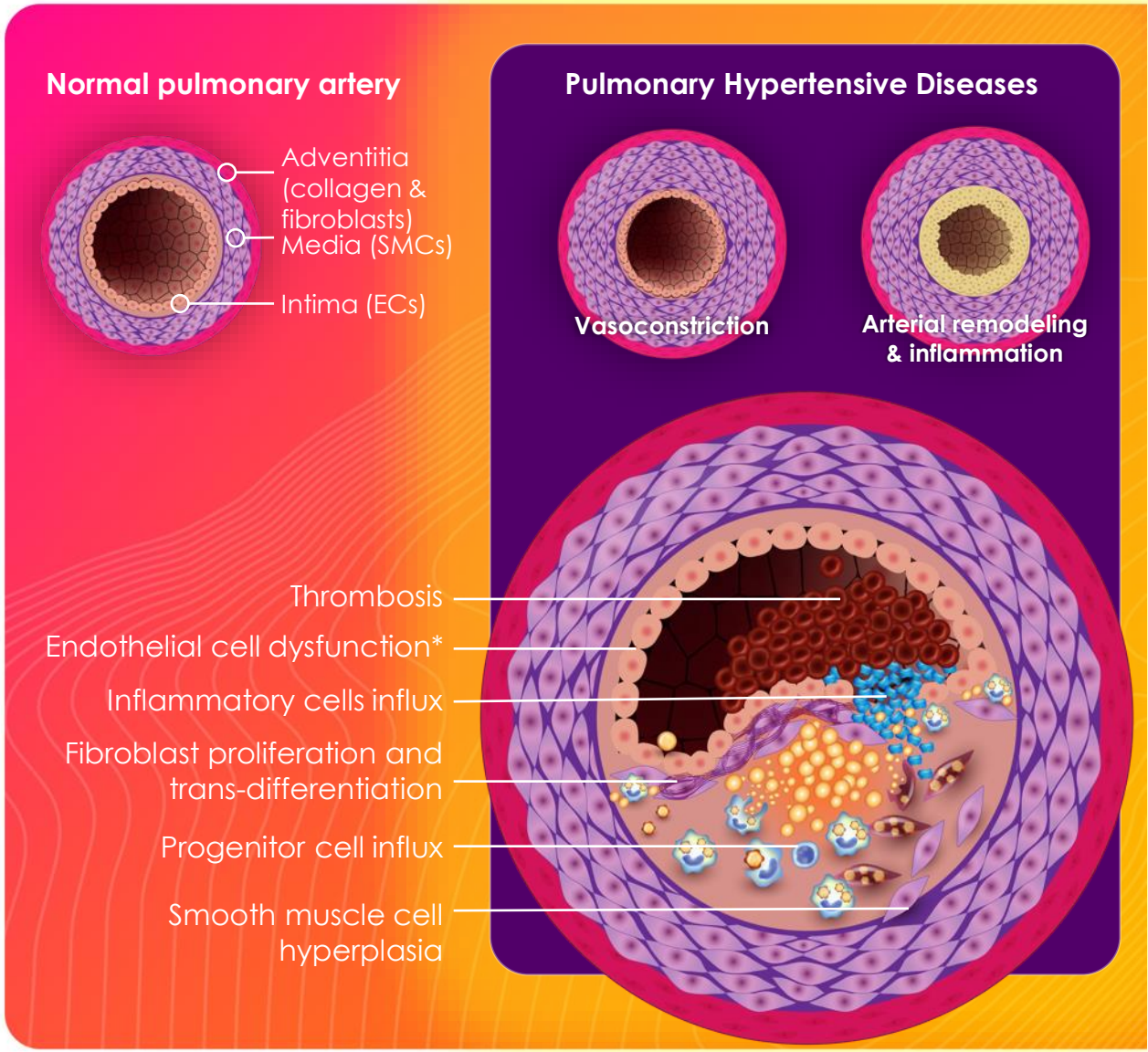
Data

Preclinical data demonstrating improved cardiac output and reversal of vascular remodeling

MORPHIC

$\alpha_5\beta_1$ Integrin Inhibition for Pulmonary Hypertensive Diseases

- Potential applications in severely underserved pulmonary hypertensive diseases
- In preclinical studies, $A_5\beta_1$ inhibition may drive multiple independent processes:
  - Reverses remodeling in pulmonary vasculature
  - Directly prevents right ventricle fibrosis
  - Improves cardiomyocyte efficiency
- $A_5\beta_1$ inhibition holds potential for true disease-modifying activity



*FDA approved drugs (Vasodilators)

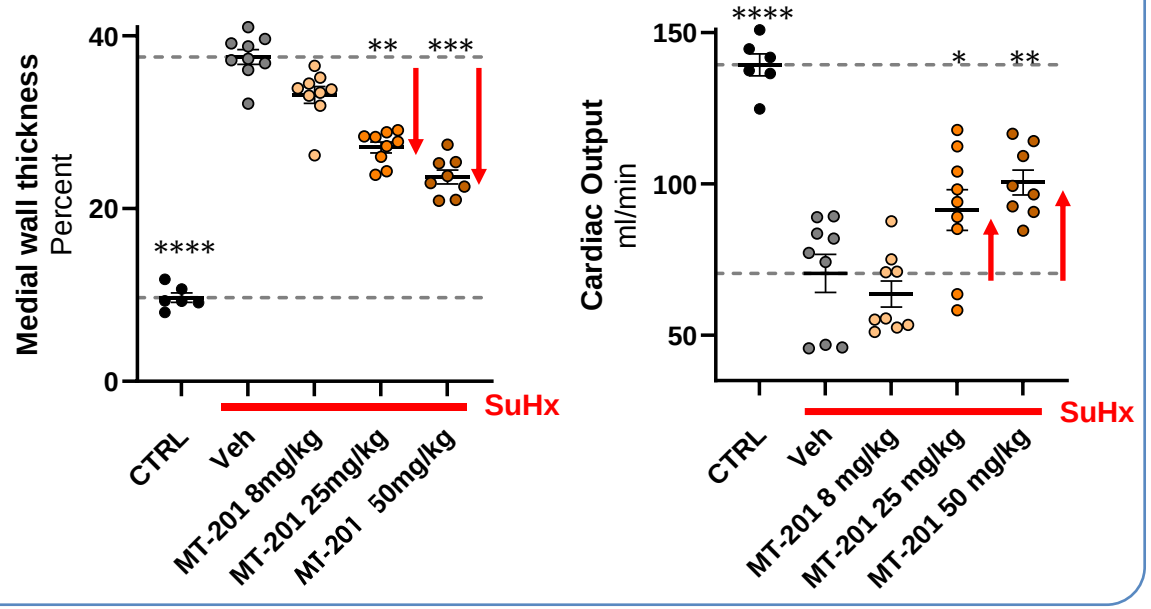
MORPHIC

Modified from Pharmacol Ther. 2013 Jun;138(3):409-17

α5b1 Inhibition Improves Pulmonary Artery Remodeling and Cardiac Function

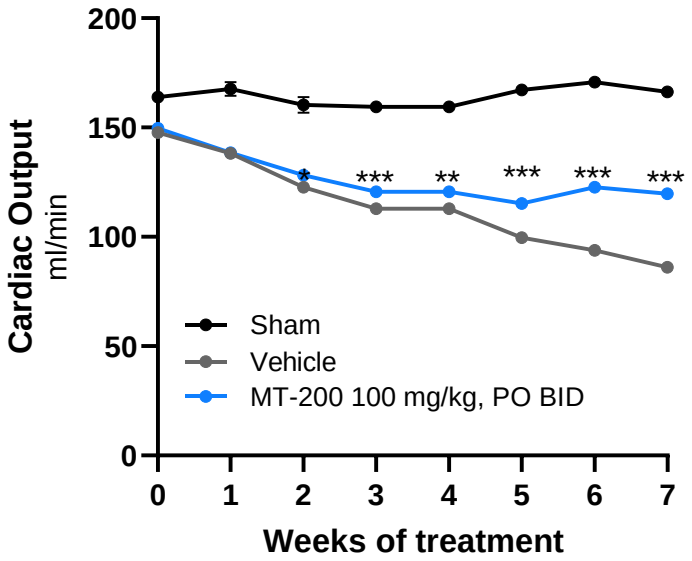
Rat Sugen/hypoxia (SuHx) Model

Pulmonary Artery Remodeling and Injury



Rat Pulmonary Arterial Banding Model

Right Ventricular Remodeling and Dysfunction



α5β1 inhibition Improves Pulmonary Artery Remodeling and prevents right ventricle failure in preclinical models

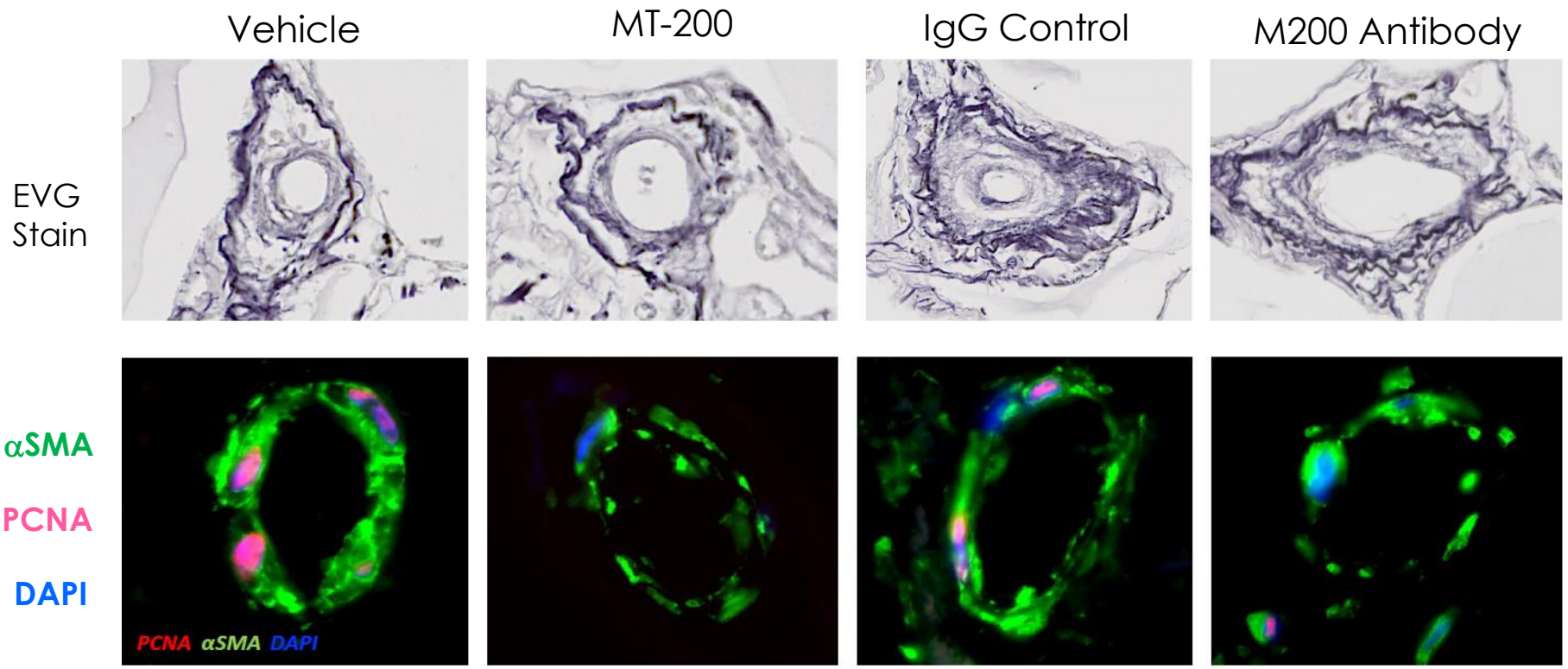
Potential differentiation from TGF-β family inhibitors, which did not show improvement in cardiac output in patients

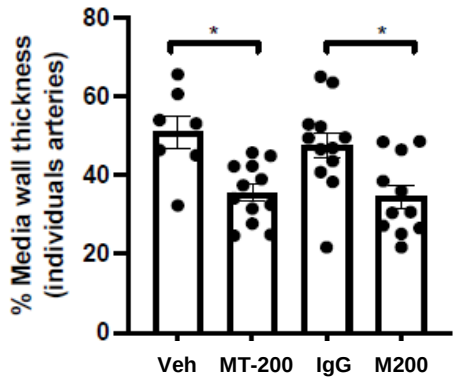
MORPHIC

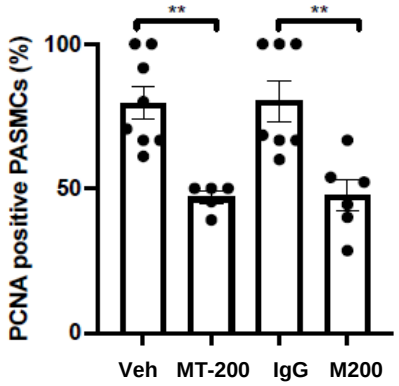
MT-200 = α5β1/αv small molecule inhibitor; (100 mg/kg, PO BID). SOC = Macitentan (Endothelin receptor antagonist, 1 mg/kg, PO BID), Tadalafil (phosphodiesterase type 5 inhibitor , 10 mg/kg, PO BID). MT-201 = α5β1 small molecule inhibitor (PO BID)

Data generated by Sebastien Bonnet, Laval University
Mean ± SEM. *p<0.05; **p<0.01; ***p<0.001
One-way ANOVA followed by Dunnett's test vs. Vehicle

$\alpha_5\beta_1$ Inhibition Blocks Pulmonary Artery Smooth Muscle Cell Proliferation in Human PAH Lung Slices







EVG = elastic stain; αSMA = smooth muscle actin; PCNA = cell proliferation marker; DAPI = nuclear stain

Study assessed the use of precision cut lung slices (PCLS) from human PAH patients to assess vascular remodeling *ex vivo*

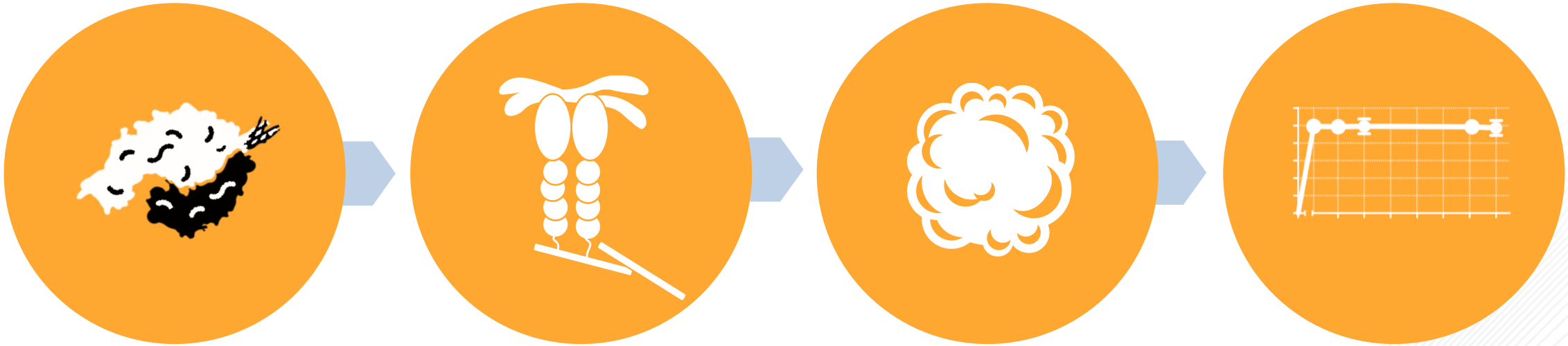
Impressive inhibition of pulmonary artery remodeling achieved in this human system

MORPHIC

MT-200 = α5β1 small molecule inhibitor; M200 = Volociximab, α5β1-specific antibody

Data generated by Sebastien Bonnet, Laval University

$\alpha_v\beta_8$ Small Molecule Integrin Inhibitor Program for Myelofibrosis and Immuno-oncology



αvβ8 Program

Small molecule inhibitors of the $a_v\beta_8$ integrin in preclinical development

Mechanism

$a_v\beta_8$ inhibition suppresses activation of TGFβ isoforms 1 and 3

Indications

Myelofibrosis;
Combination therapy for solid tumors

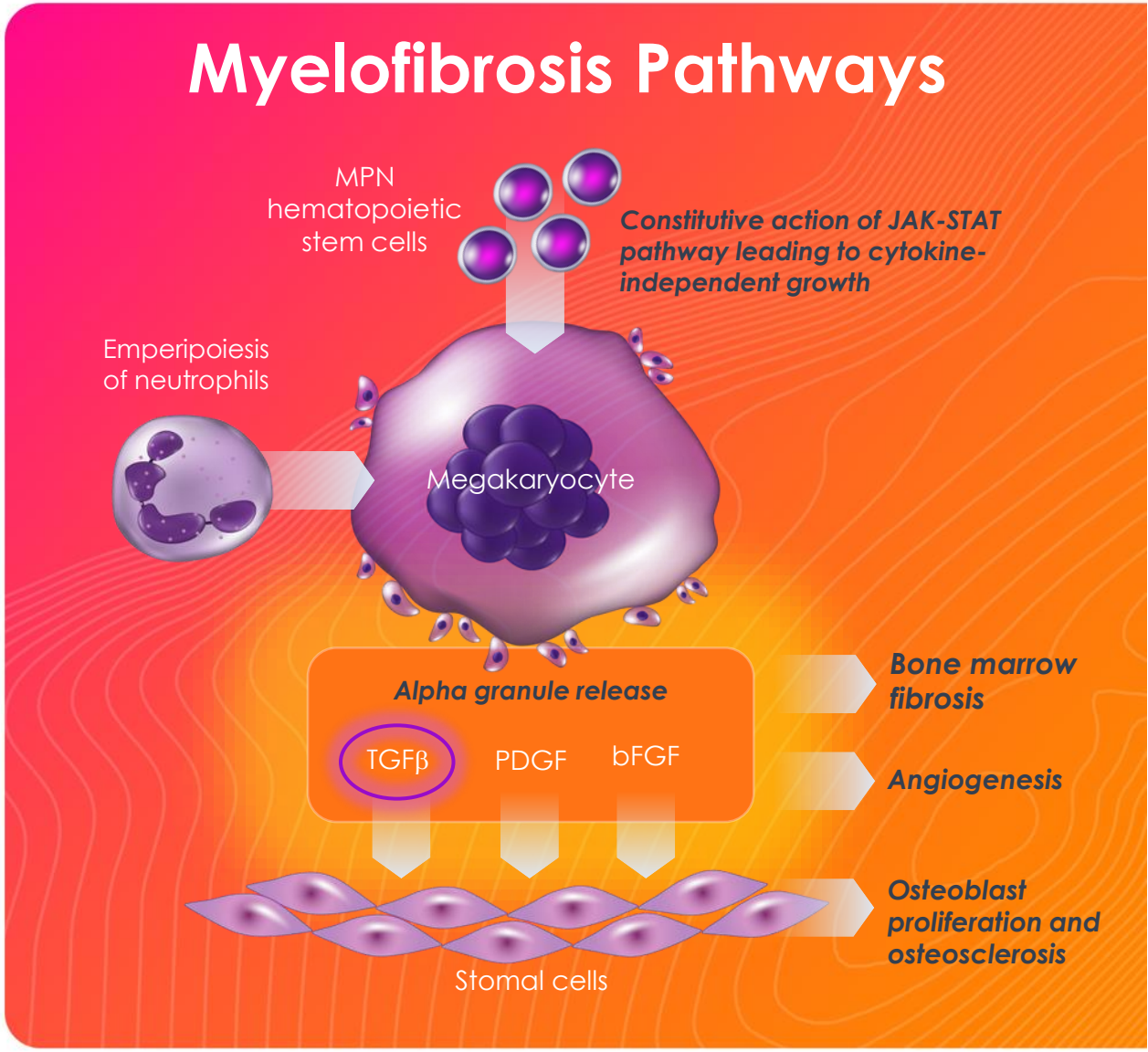
Data

Oral $a_v\beta_8$ inhibitor, in combination with anti-PD-1, drives efficacy across mouse models of treatment-resistant breast cancer;
Myelofibrosis: $a_v\beta_8$ inhibition drives increase in platelet production in published literature

MORPHIC

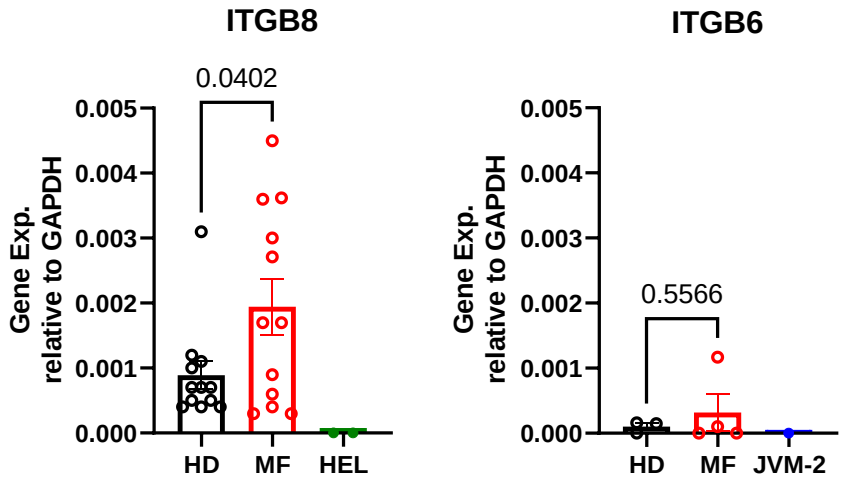
MORF-088: $a_v\beta_8$ Inhibitor for Myelofibrosis (MF)

- MF: multi-mechanistic etiology including TGF-B
- Blockbuster rare disease indication
  - Jakafi $1 billion MF sales alone
- No disease modifying Tx except allogeneic hematopoietic stem cell transplant
- Current SoC has multiple deficiencies
  - Toxicity: anemia and thrombocytopenia
  - Intolerance or resistance to therapy develops over time
  - Not disease modifying
- $a_v\beta_8$ Smi offers potential to increase platelet counts



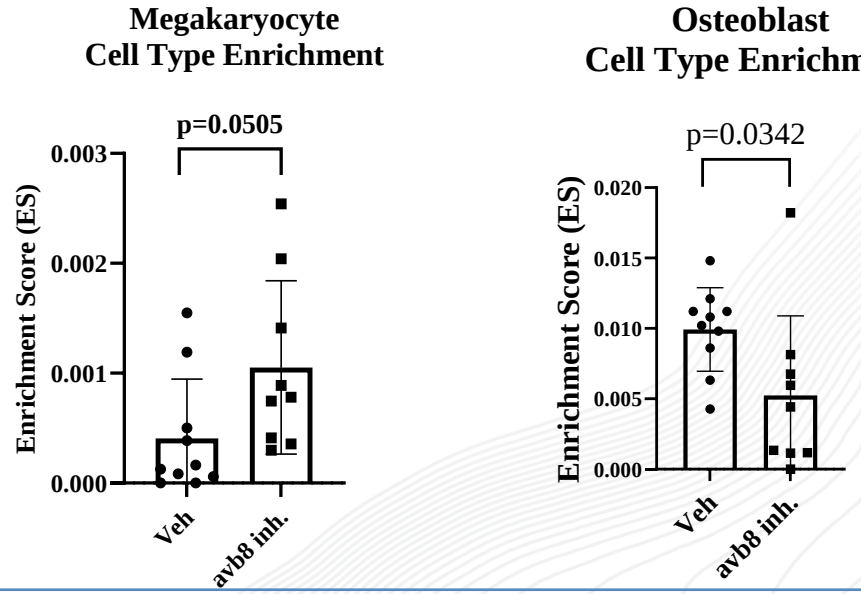
$\alpha_v\beta_8$ Inhibition: Central Role in TGF-β Modulation

$\alpha_v\beta_8$ is the dominant TGF-β forming integrin in human bone marrow



Healthy donor (HD) and myelofibrosis (MF) CD34+ HSC[1]

$\alpha_v\beta_8$ inhibition *in vivo* leads to enrichment of megakaryocytes and decreased osteoblasts, suggesting a healthier bone marrow niche



[1]The HEL 92.1.7 (HEL) and JVM-2 cell lines were used as a negative controls for ITGB8 and ITGB6 expression, respectively.

Deep Specialist Expertise Across Management, and Board of Directors

Executive Team

Praveen Tipirneni, MD
Chief Executive Officer

Bruce Rogers, PhD
President

Marc Schegerin, MD
Chief Financial Officer
Chief Operating Officer

William DeVaul
General Counsel
and Secretary

Blaise Lippa, PhD
Chief Scientific Officer

Board of Directors

Gustav Christensen, MBA Chairman Morphic Board, former CEO, Dyax

Timothy Springer, PhD Founder, Morphic Therapeutic, Member of Morphic Scientific Advisory Board; Latham Family Professor, Professor of Biological Chemistry and Molecular Pharmacology; Professor of Medicine, Harvard Medical School

Norbert Bischofberger, PhD President & CEO, Kronos; EVP R&D, CSO, Gilead

Martin Edwards, MD Former Chairman, Kalvista; Senior Partner, Novo Holdings

Nisha Nanda, PhD Group VP, External Innovations at Eli Lilly and Company

Amir Nashat, PhD Managing Partner, Polaris Partners

Susannah Gray, PhD Former CFO, Royalty Pharma

Joseph P. Slattery, CPA Former CFO, Transenterix, Baxano, Digene

Praveen Tipirneni, MD CEO Morphic Therapeutic

MORPHIC

MORPHIC

THANK YOU