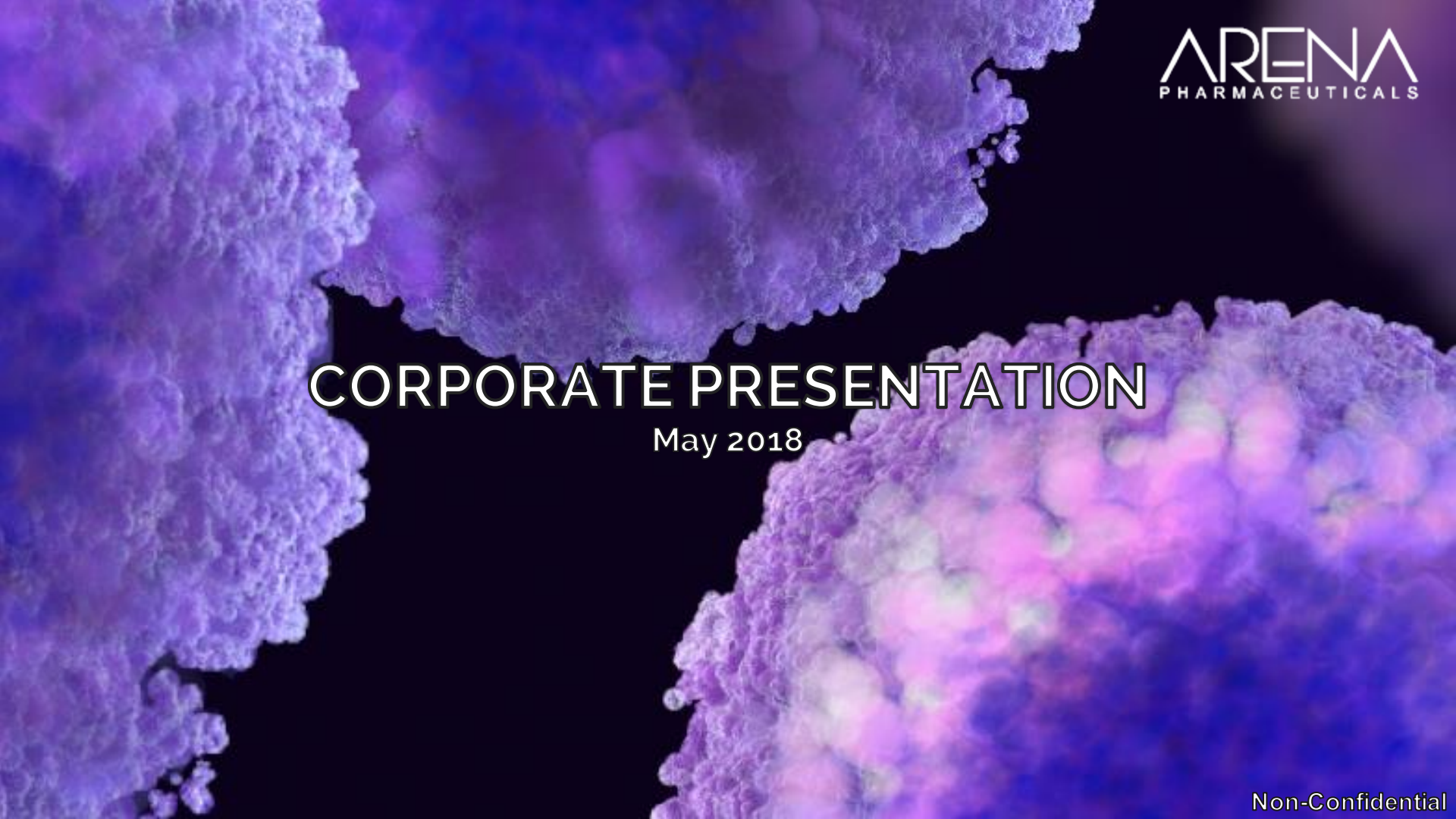


CORPORATE PRESENTATION

May 2018

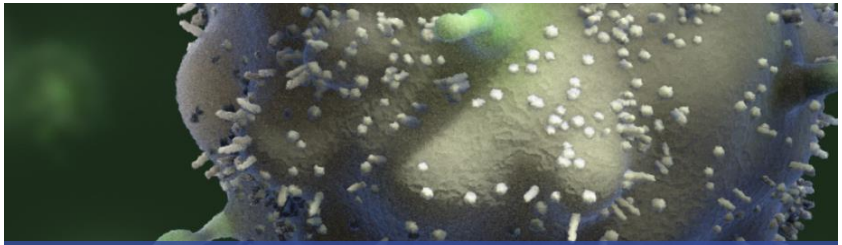
ARENA PHARMACEUTICALS

Non-Confidential

Forward-Looking Statements

This presentation includes forward-looking statements that involve a number of risks and uncertainties, including statements about the Arena investment thesis, catalysts, value, our investigative stage drug candidates, including with respect to their potential (including to become first or best-in-class), safety, efficacy, indications, significance of data, development plans, differentiation, the market and unmet needs and commercialization, expected data readouts and initiation of new clinical trials; our focus, goals, strategy, plans, timelines and guidance; our partnered programs; financial and other guidance; and other statements that are not historical facts, including statements that may include words such as “may,” “will,” “intend,” “plan,” “expect,” “potential” or other similar words. For such statements, we claim the protection of the Private Securities Litigation Reform Act of 1995. Actual events or results may differ materially from expectations, and you are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the time they were made. Factors that could cause actual results to differ materially from such statements include, without limitation: topline data may not accurately reflect the complete results of a particular study or trial; results of clinical trials and other studies are subject to different interpretations and may not be predictive of future results; clinical and nonclinical data is voluminous and detailed, and regulatory agencies may interpret or weigh the importance of data differently and reach different conclusions than Arena or others, request additional information, have additional recommendations or change their guidance or requirements; the timing and outcome of research, development and regulatory review is uncertain; unexpected or unfavorable new data; our drug candidates may not advance in development or be approved for marketing; clinical trials and other studies may not proceed at the time or in the manner expected or at all; data and information related to our programs may not meet regulatory requirements or otherwise be sufficient for further development, regulatory review, partnering or approval; other risks related to developing, seeking regulatory approval of and commercializing drugs, including regulatory, manufacturing, supply and marketing issues and drug availability; we expect to need additional funds to advance all of our programs, and you and others may not agree with the manner in which we allocate our resources; Arena's and third parties' intellectual property rights; competition; reimbursement and pricing decisions; risks related to relying on partners and other third parties; and satisfactory resolution of litigation or other disagreements. Additional factors that could cause actual results to differ materially from those stated or implied by our forward-looking statements are disclosed in our Securities and Exchange Commission (SEC) filings, including under the heading “Risk Factors” in our Annual Report on Form 10-K for the year ended December 31, 2017, and subsequent filings. We disclaim any intent or obligation to update these forward-looking statements, other than as may be required under applicable law.

Arena Investment Thesis



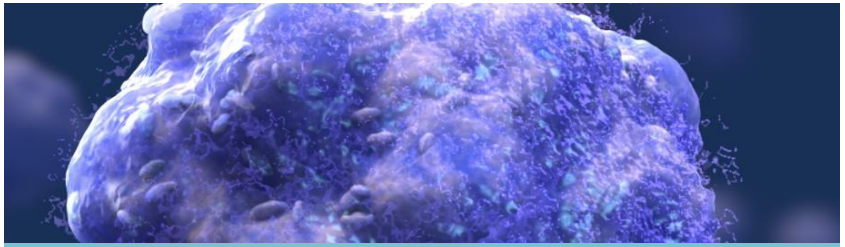
Two potential best-in-class, internally developed drug candidates to Ph 3

Targeting over $30 billion total market opportunity*

Supported by Ph 2 data with clear and meaningful competitive advantages

Retain rights to all major markets and strong intellectual property position

Strong cash balance - Cash and cash equivalents totaled $629.1 M at 3/31/18



Significant catalysts in 2018 and 2019

Olorinab data readout in Crohn's pain

Ralinepag Ph 3 program initiation in PAH

Etrasimod Ph 3 initiation in UC

Etrasimod program initiation in CD

Etrasimod Ph 2 data readout in PBC

Cardiopulmonary pipeline expansion

Additional data readouts across pipeline

*Leerink Analysis & Decision Resources 2016

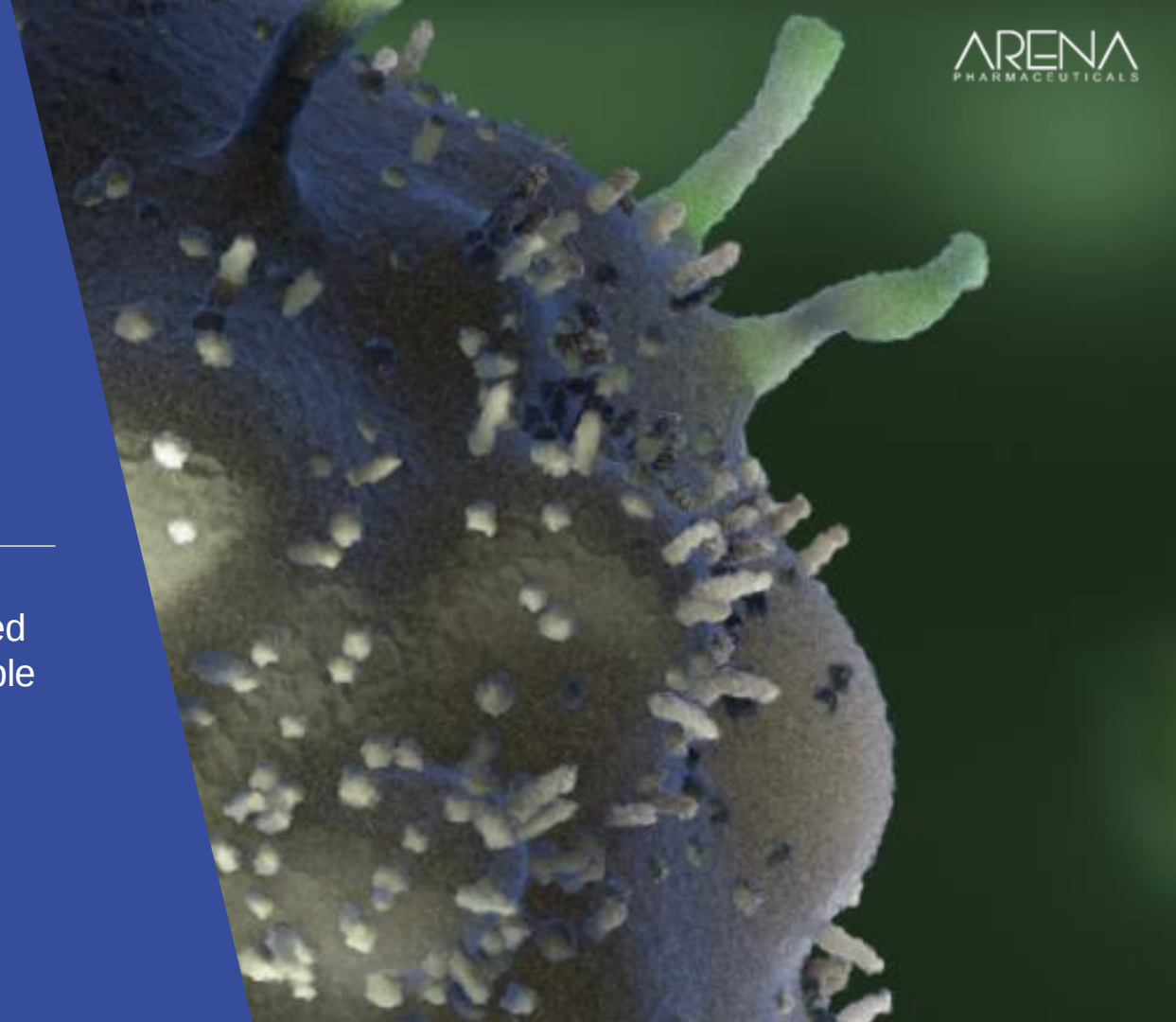
Focus on Pipeline

Differentiated Assets with High-Value Potential

Program	Therapeutic Area/Indication	PC	Ph 1	Ph 2	Ph 3	Status
Ralinepag *Potent IP Receptor Agonist*	PAH - Exercise Capacity					Ph 3 2H:18
	PAH - Time to Clinical Events					Ph 3 2H:18
	PAH - Differentiation					2H:18
Etrasimod *Optimized Activity S1P Receptor Modulator*	Ulcerative Colitis (UC) 1					Ph 3 planning
	Ulcerative Colitis (UC) 2					Ph 3 planning
	Crohn's Disease (CD)					Program planning
	Primary Biliary Cholangitis					Ph 2
Olorinab *Highly Selective Full Agonist* CB_2	Pain Associated with CD					Ph 2 Data Q3:18

Etrasimod

Oral, Next Generation, S1P Receptor Modulator with Optimized Activity Being Evaluated for Multiple Immune-Inflammatory Diseases



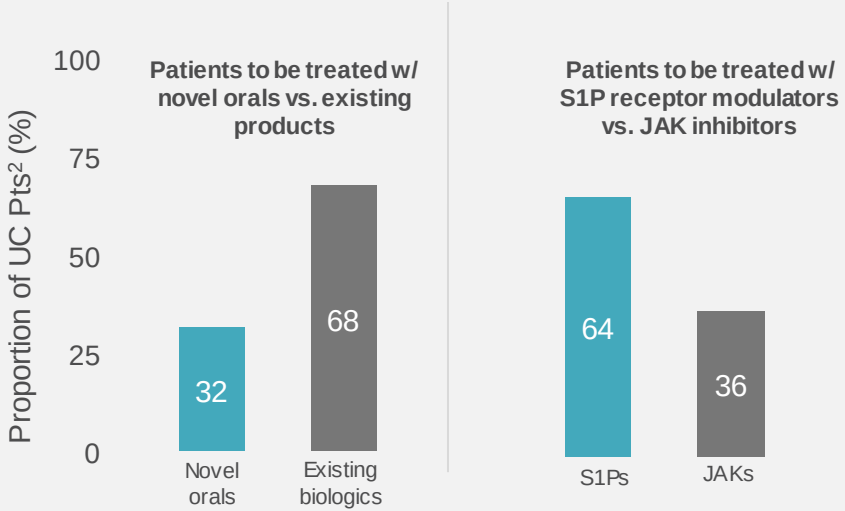
Phase 3 Ready Etrasimod

Potential to Redefine Oral QD Treatment in IBD

$18-23B IBD Market Globally[1]

Physicians project replacing one third of current biologic use with novel orals[2]

Strong preference for S1P receptor modulators over JAK inhibitors



Ph 2 Efficacy

Dose dependent lymphocyte reduction

Strong remission data – 33% of 2 mg patients achieved remission with heavy pretreatment with biologics

Strong and consistent mucosal healing

Fast onset - important in flare state

Ph 2 Safety

Well-characterized

Strong receptor selectivity for S1PR1,4,5 – no off-target activity

Fast offset – 95% lymphocyte recovery within 1 week

No evidence of elevated LFTs, abnormal PFTs, or macular edema

Minimal HR effects and AV conduction issues – no related discontinuations

No sinoatrial arrest cases

Source: 1. Leerink Analysis. 2. Arena Quantitative Market Research n=100, Mar. 2018

Etrasimod's Highly Selective

S1P Receptor Modulation May Avoid Off-Target Activity

Receptor Binding

Etrasimod

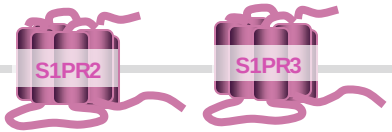
T-Lymphocytes | Dendritic Cells | Oligodendrocytes

Reduces Immune Cell Migration, Differentiation & Proliferation

Decreased Inflammation & Improvement in Immune-Inflammatory Disorders

Positive Outcome

No Activity



Increased Vasoconstriction, Fibrosis & Proliferation

Associated with Cardiac, Pulmonary & Possible Tumor-related Risks

Negative Outcome

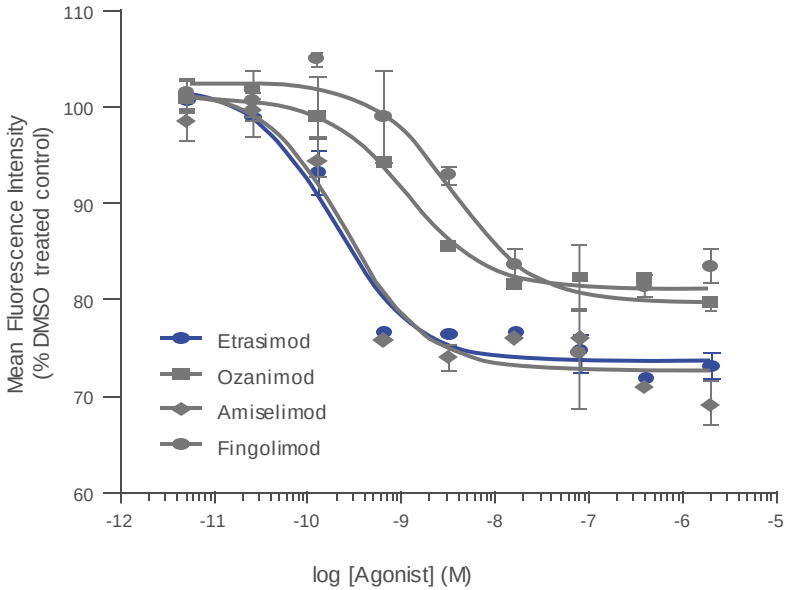
S1P = sphingosine-1-phosphate; UC = ulcerative colitis. *Data generated at Arena or under sponsored research by Arena

In Contrast with Other S1P Receptor Modulators

Etrasimod Does Not Induce S1PR2 Internalization (In Vitro Studies)

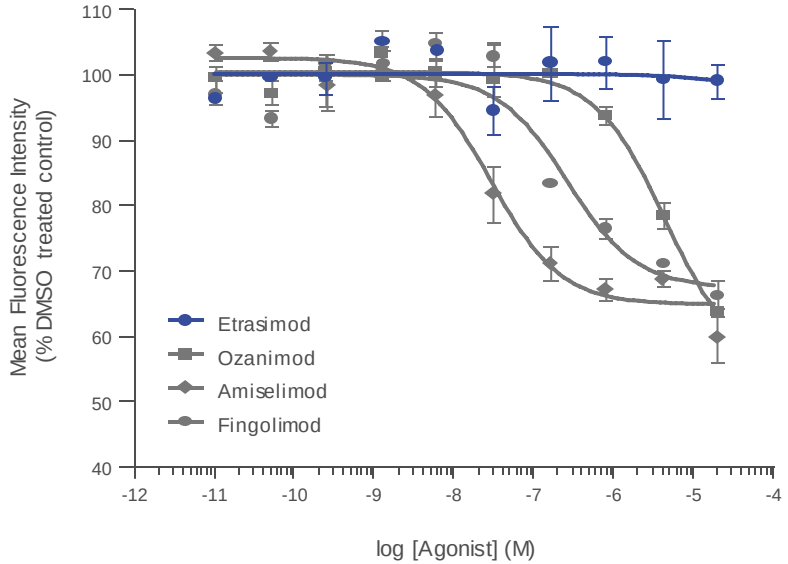
S1P Agonists Induce S1PR1 Internalization

(S1PR1 Internalization Assay)



Etrasimod Does Not Induce S1PR2 Internalization

(S1PR2 Internalization Assay)

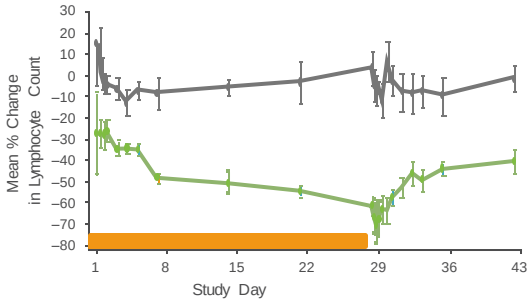


Arena Data presented at ASPET 2017 at Experimental Biology (EB); DMSO = dimethylsulfoxide; S1P = sphingosine-1-phosphate

Etrasimod Has a Quick Offset of Action

Phase 1 Data

Ozanimod[1]

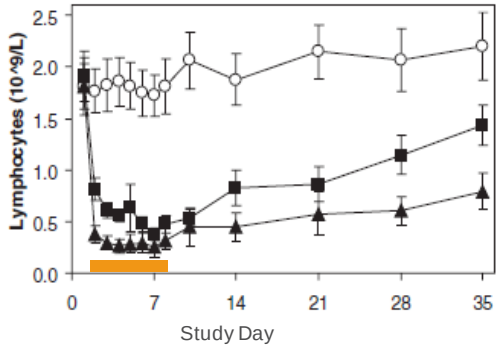


Placebo
Ozanimod, 1 mg (Fixed dose)

At 2 weeks after last dose of **ozanimod**, lymphocyte levels still ~60% recovered to baseline

Half Life ~10-13 Days*

Gilenya[2]

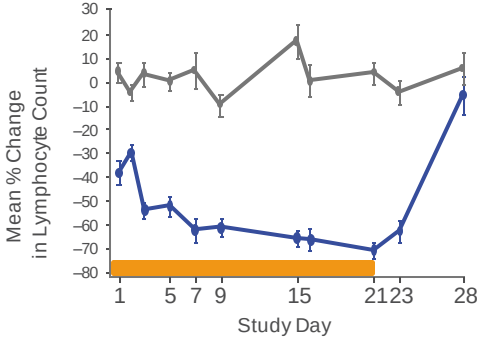


Placebo
Gilenya, 1.25 mg (Fixed dose)
Gilenya, 5 mg (Fixed dose)

At 2 weeks after last dose of **Gilenya®**, lymphocyte levels still ~70% recovered to baseline

Half Life ~6-9 Days

Etrasimod[3]



Placebo
Etrasimod, 2 mg (Fixed Dose)

Within 1 week after last dose of **etrasimod**, lymphocytes levels ~95% recovered to baseline

Half Life ~1-1.5 Days

Note – Data Collected from Separate Studies – Cannot be Directly Compared

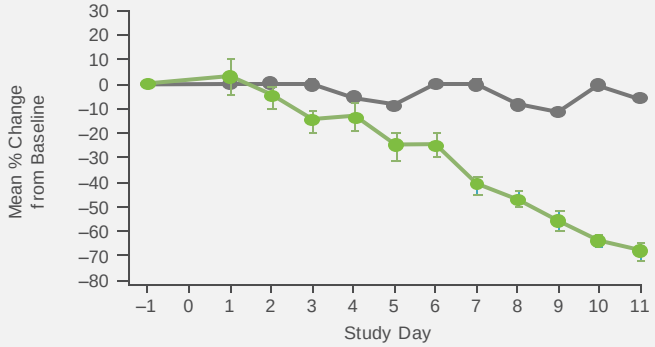
Sources: 1-Tran JQ et al, J Clin Pharmacol. 2017;57:988-996. 2-Kovarik JM et al, J Clin Pharmacol. 2004;44:535-537. 3- ADP334 P1 Data on File; *Active metabolite

Treatment days

Etrasimod Has a Quick Onset of Action

Phase 1 Data

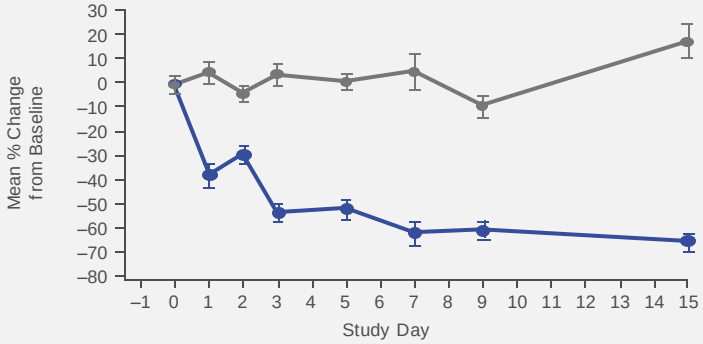
Ozanimod[1]



At Day 3, **ozanimod** showed ~15% lymphocyte reduction

- Placebo
- Ozanimod, 2 mg (Dose Escalation*)

Etrasimod[2]



At Day 3, **etrasimod** showed ~53% lymphocyte reduction

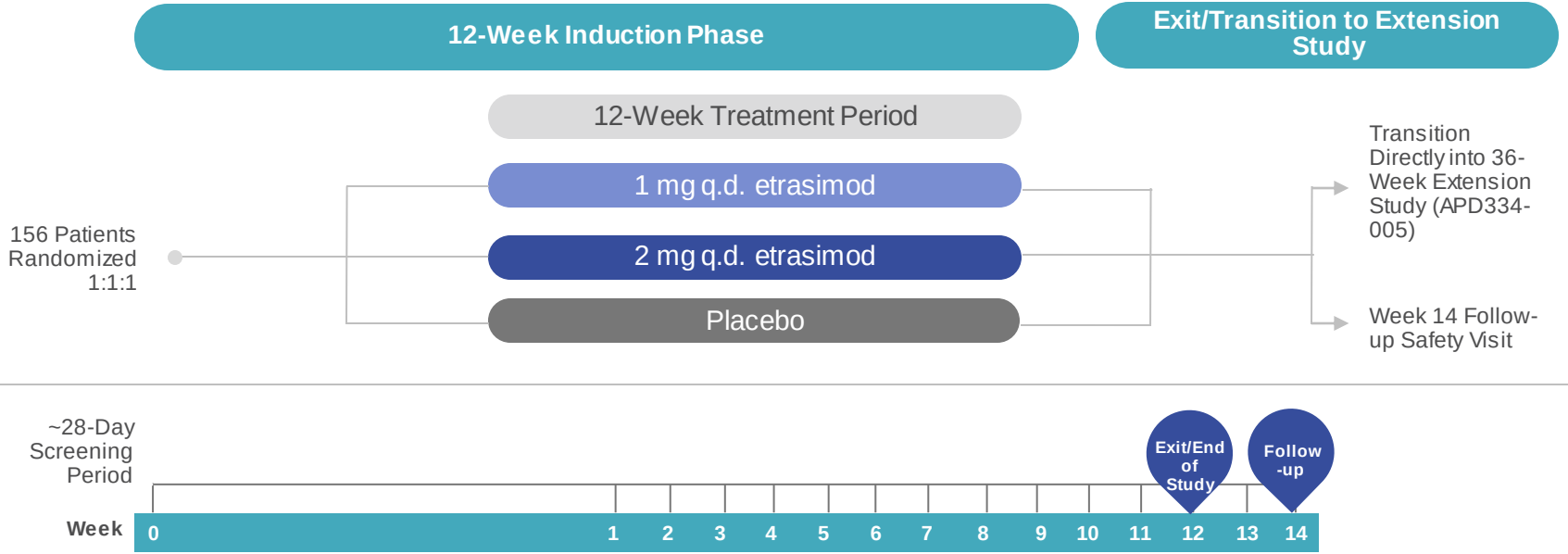
- Placebo
- Etrasimod, 2 mg (Fixed Dose)

Note – Data Collected from Separate Studies – Cannot be Directly Compared

*After dose escalation of ozanimod (0.3 mg/d on days 1–3; 0.6 mg/d on days 4 and 5; 1 mg/d on days 6 and 7; and 3 mg/d on days 8–10. Data truncated at day 11 (ozanimod) and 15 (etrasimod) to show on-treatment onset effects Sources: 1-Tran JQ et al, J Clin Pharmacol. 2017;57:988-996. 2- ADP334 P1 Data on File

Etrasimod Phase 2 Trial in Ulcerative Colitis (OASIS)

Randomized, double-blind, placebo-controlled, parallel-group, dose-ranging study



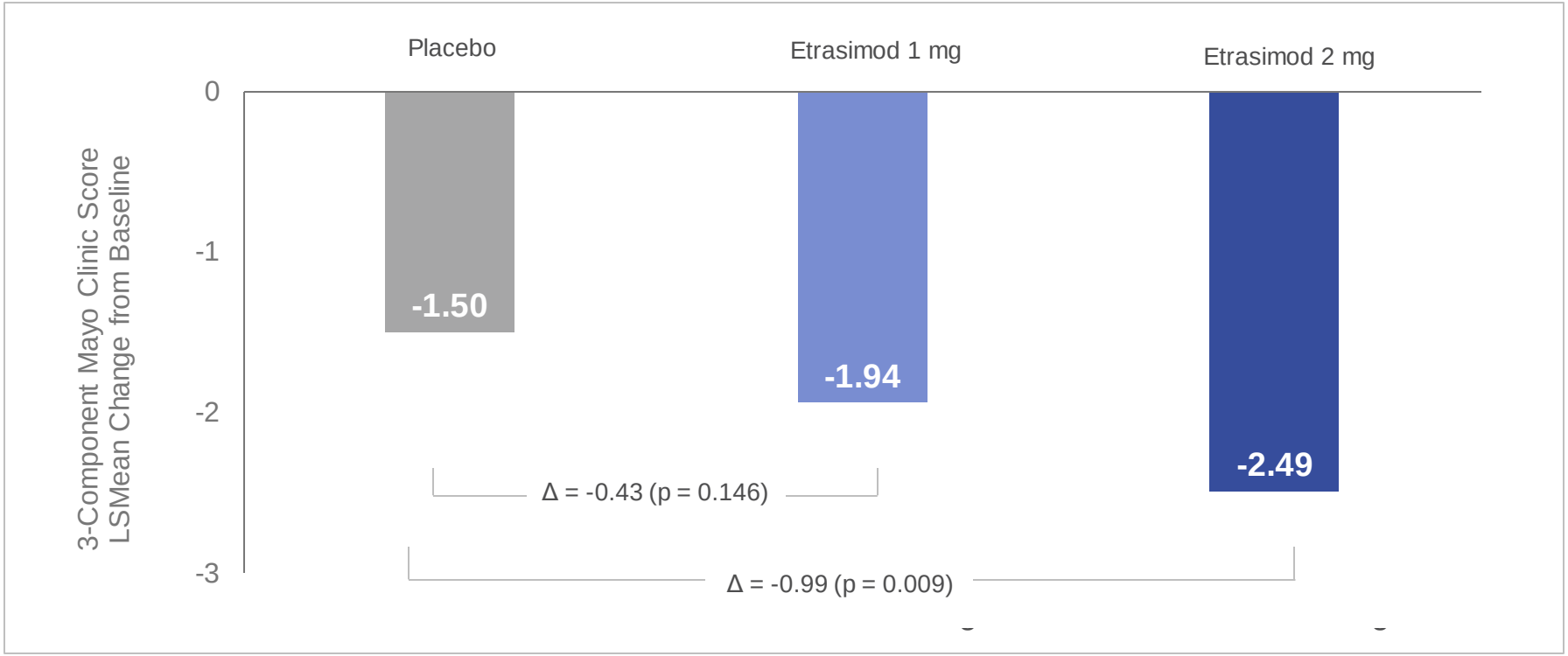
Key Measurements

Endoscopy & Physician's Global Assessment performed during screening and at week 12

Stool Frequency and Rectal Bleeding PROs collected daily using electronic patient diaries

Phase 2 Primary Endpoint: 3-Domain

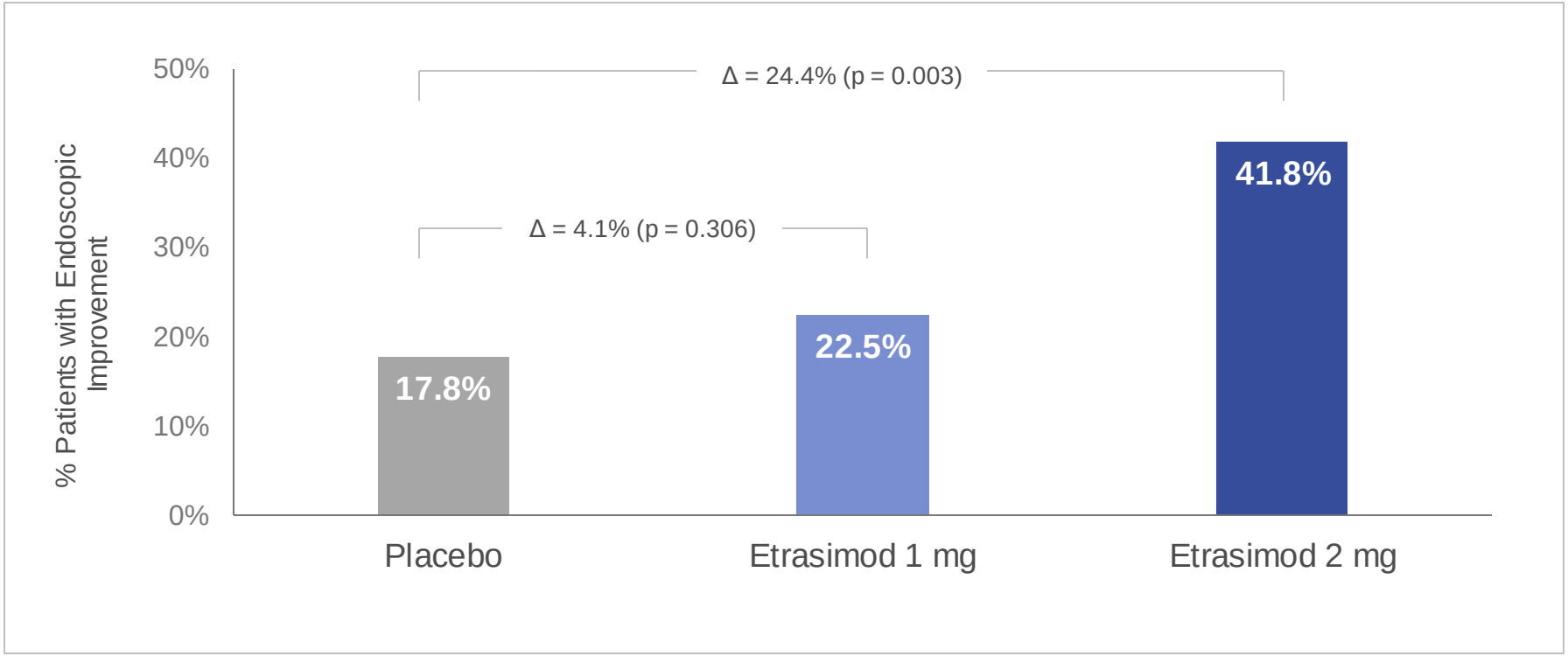
Change on 9-Point Scale Including Rectal Bleeding, Stool Frequency, Endoscopy



LS=least square; Δ LS mean difference from placebo. LS mean was estimated using an ANCOVA model that includes current oral corticosteroid therapy at baseline and previous exposure to TNFα antagonists, baseline measure, and treatment group. MCS = Mayo Clinic Score

Phase 2 Secondary Endpoint: Endoscopic Improvement

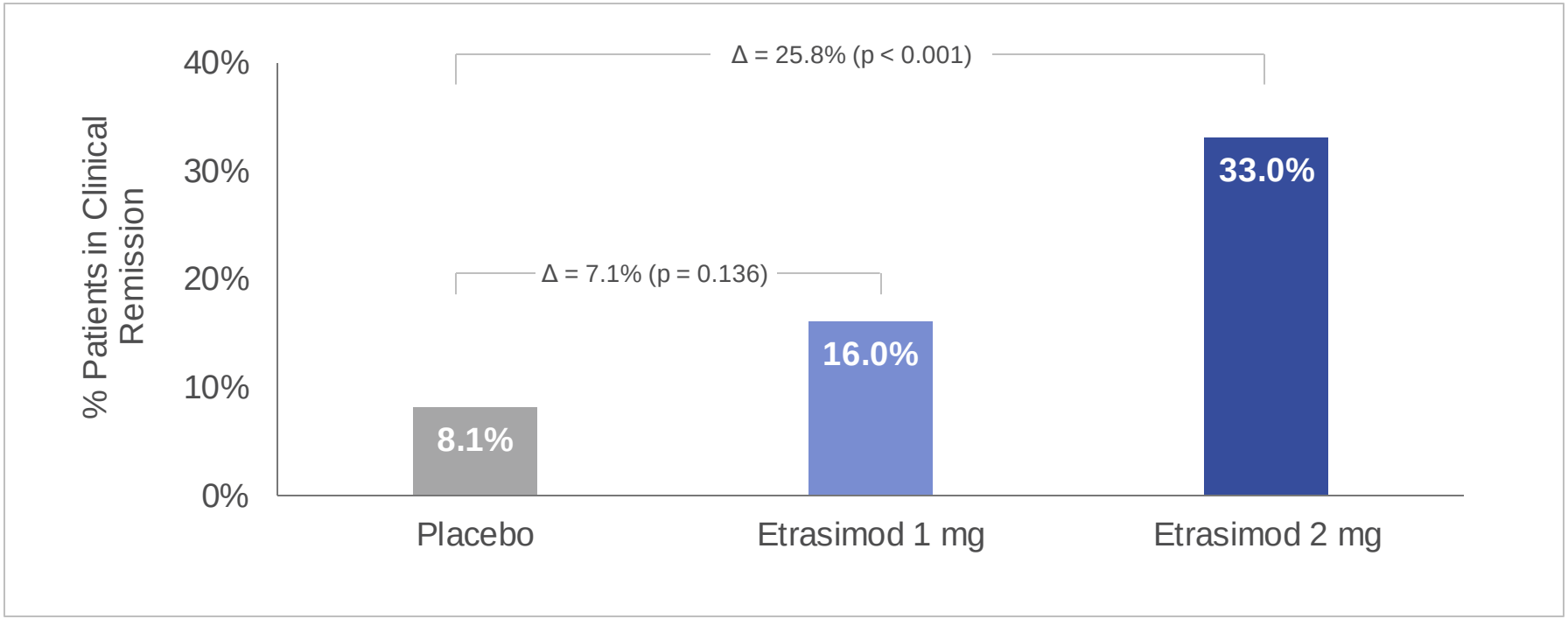
Proportion of Patients with Mayo Clinic Subscore of 0 or 1 points



Δ=% difference from placebo estimated using Mantel-Haenszel method adjusted with current oral corticosteroid use and prior exposure to TNFα antagonists.

Phase 2 Clinical Remission (3-Domain)

Proportion of Patients with Endoscopy, Rectal Bleeding and Stool Frequency Mayo Clinic Subscore of ≤ 1 point and Stool Frequency Improvement of ≥ 1 point

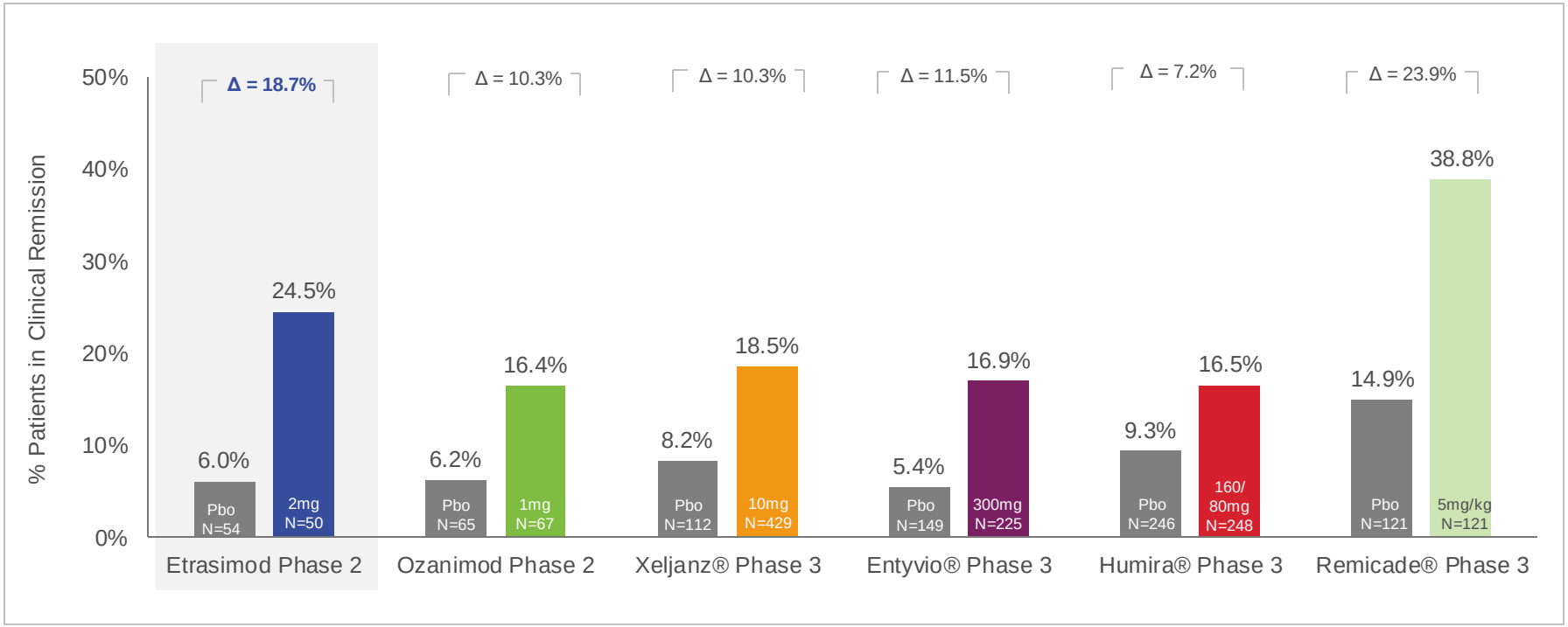


Δ=% difference from placebo estimated using Mantel-Haenszel method adjusted with current oral corticosteroid use and prior exposure to TNFα antagonists.

Phase 2 Clinical Remission (4-Domain) in UC

Proportion of Patients with TMCS ≤ 2 points and No Subscore > 1 point

Note: No direct head-to-head data available - Caution advised when comparing data across clinical studies

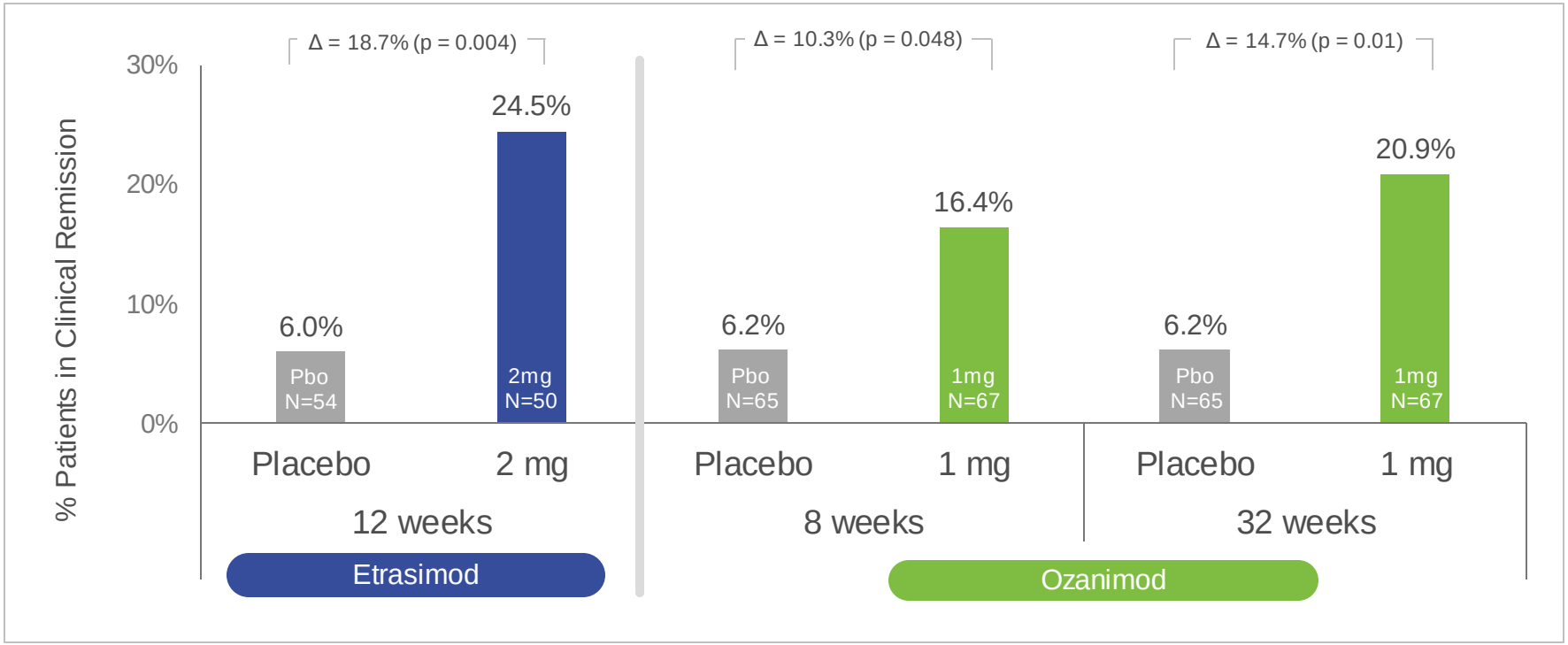


Etrasimod: Post-hoc analysis. Δ=% difference from placebo estimated using Mantel-Haenszel method adjusted with current oral corticosteroid use and prior exposure to TNFα antagonists..
Sources: Ozanimod: Sandborn, et al. NEJM 2016; Xeljanz: Sandborn, et al. NEJM 2017; Entyvio: Feagan, et al. NEJM 2013; Humira : Sandborn, et al. Gastroenterology 2012; Remicade: Rutgeerts, et al. NEJM 2005. TMCS = Total Mayo Clinic Score

Clinical Remission (4-Domain) in UC Studies of S1P Modulators

Proportion of Patients with Total Mayo Clinic Score ≤ 2 points and No Subscore > 1 point

Note: No direct head-to-head data available - Caution advised when comparing data across clinical studies



Total MCS = Mayo Clinic Score. Etrasimod: Post-hoc analysis. Δ=% difference from placebo estimated using Mantel-Haenszel method adjusted with current oral corticosteroid use and prior exposure to TNFα antagonists. Ozanimod: Sandborn, et al. NEJM 2016

Phase 2 Safety Profile

Etrasimod was generally safe and well tolerated

Adverse events were predominantly mild to moderate

No serious adverse events (SAEs) at the 2 mg dose

Impact on HR and AV conduction was low throughout the study with no discontinuations related to bradycardia or AV block. No SAEs related to HR changes or AV block

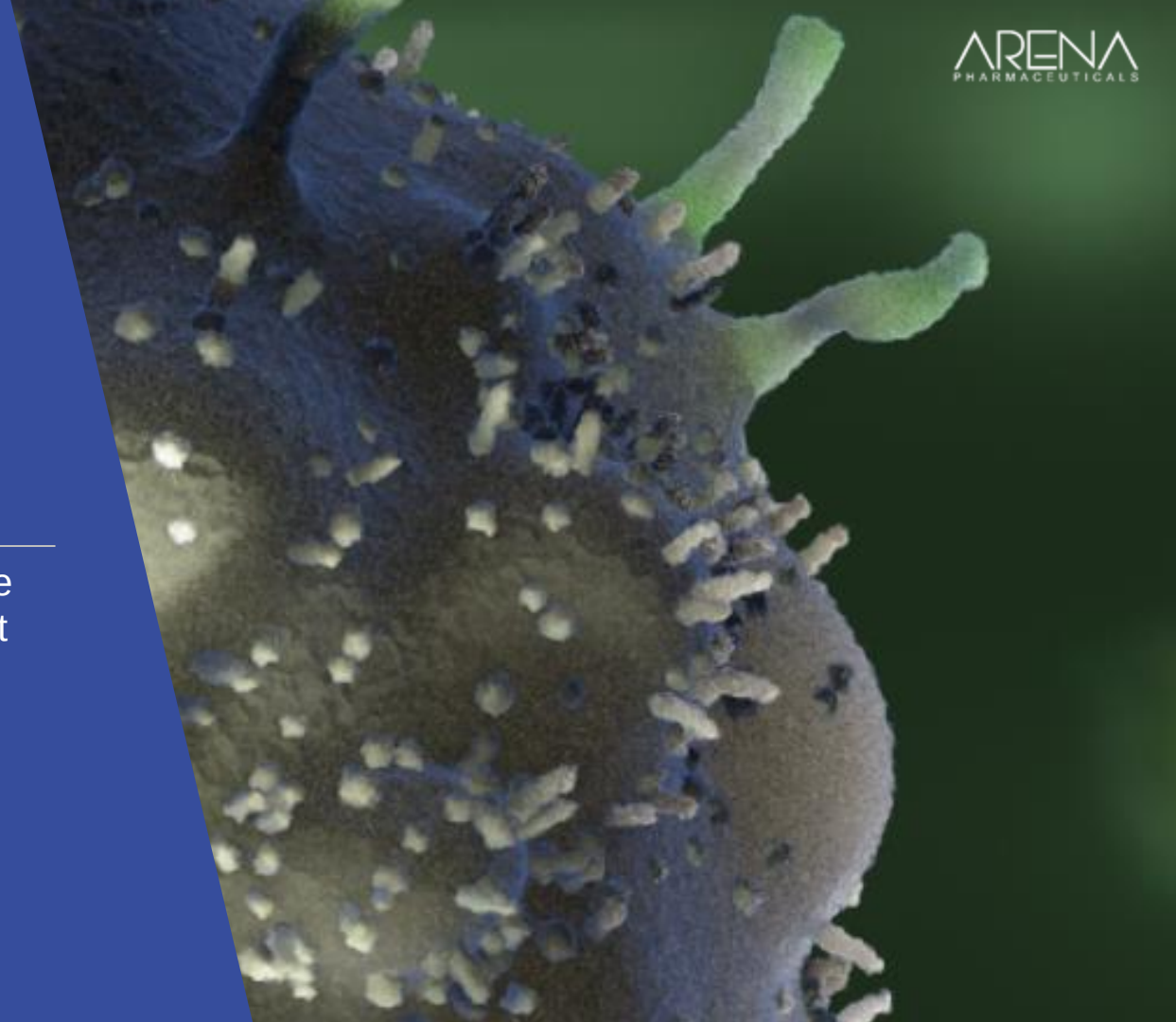
No increases in liver function tests compared to placebo

No reports of macular edema

No reports of abnormal pulmonary function tests

Ralinepag

A Next-Generation, Oral, Selective Prostacyclin Receptor (IP) Agonist for the Treatment of Pulmonary Arterial Hypertension



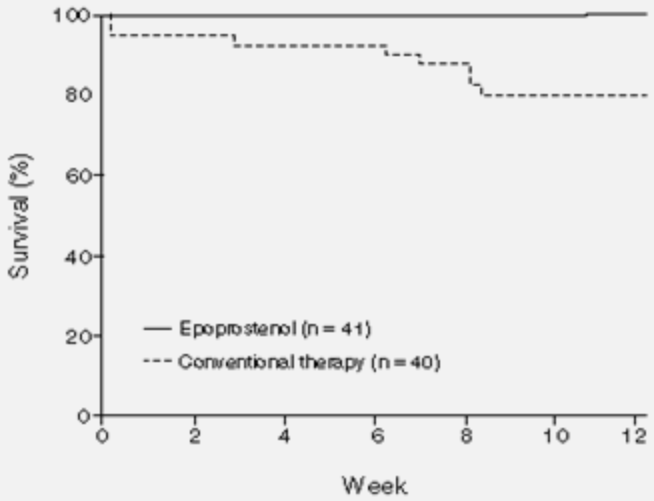
Phase 3 Ralinepag

Potential to Redefine Treatment Paradigm in PAH

Unmet Need in $6.7B[1] PAH Market

1996 Barst – Unremitting 24h coverage with IV prostacyclin improves survival[2]

Difficulty of administration and maintenance limits IV to last line



Oral QD Ralinepag

6.5-10x improved potency compared to selexipag

Long half-life (>24 h), IV-like unremitting receptor engagement

Unprecedented 20.1%* PVR improvement from baseline in patients primarily on dual background therapy

Ph 3 Objectives

Optimize speed to market

Generate data set to establish ralinepag as cornerstone therapy

Potential for best-in-class label in incident and prevalent populations

Generate differentiation data vs. currently approved oral prostacyclins

1. Decision Resources 2016 2. Barst RJ N Engl J Med 1996; 334:296-301

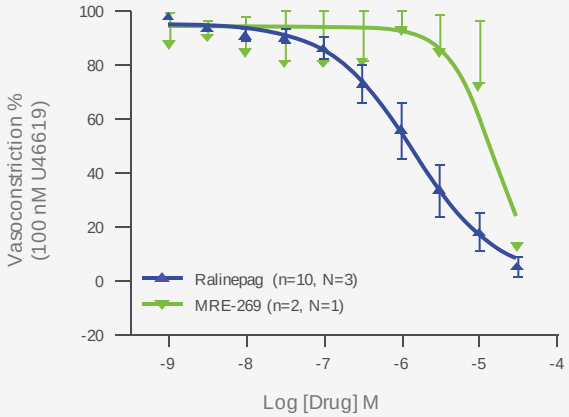
*Least squares mean change from baseline; non parametric testing to account for non-normal distribution

Ralinepag's More Potent IP Agonism

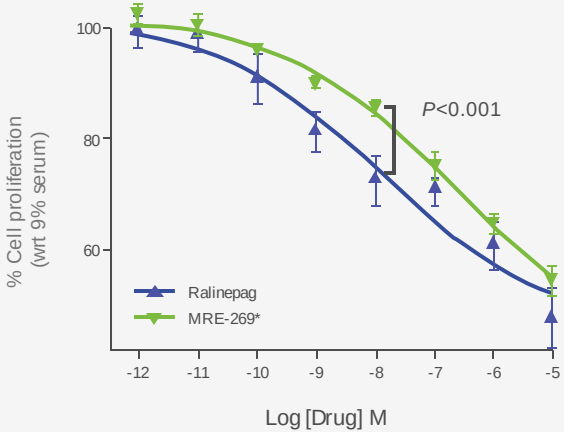
Translates to Improved Tissue Responses vs. Selexipag* in Vitro

IP Receptor Binding → **cAMP Activation 6.5x vs. MRE-269*** →
1. Vasorelaxation
2. Inhibition of SMC Proliferation
3. Inhibition of Platelet Aggregation

Vasodilation[1] (Human PAH Tissue)

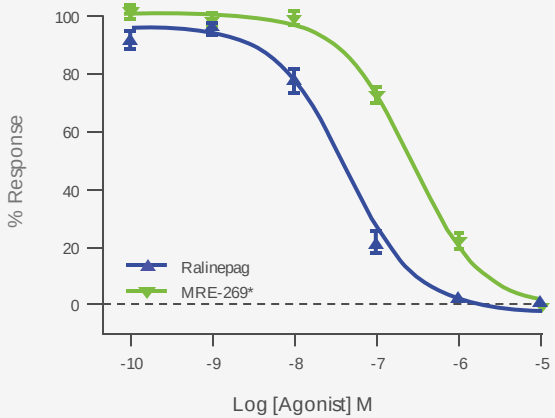


Inhibition of SMC Proliferation[2] (Human PAH Tissue)



Ralinepag potency: **10x vs. selexipag**

Platelet Inhibition[3] (Normal Human Cells)



Ralinepag potency: **7.5x vs. selexipag**

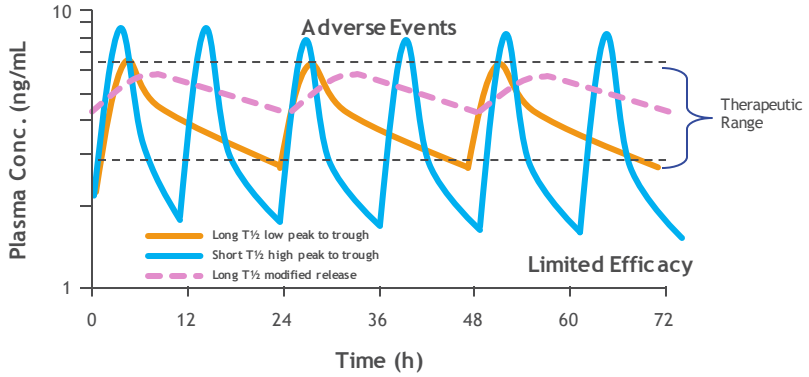
More potent vasodilation, inhibition of vascular remodeling and platelet aggregation suggests that ralinepag may improve on selexipag in the clinical management of PAH

*MRE-269 is the active metabolite of selexipag. cAMP, cyclic adenosine monophosphate; PAH, pulmonary arterial hypertension; SMC, smooth muscle cell [1]Shen et al ERS 2017 (submitted); [2]Shen et al AHA 2016 [3]Tran et al J Med Chem 2016

Achieving Continuous Therapeutic Range Exposure, Mimicking IV Infusion, Enhances Oral Efficacy

- Oral IP agonists with interrupted therapeutic coverage (e.g., beraprost, treprostinil) have shown disappointing efficacy
- Low peak-to-trough ratio is key to minimizing C_{max} related tolerability issues and limiting C_{min} related efficacy issues of oral agents

Potential to Maintain Consistent Exposure and Minimize C_{max} and C_{min} Related Issues



Ralinepag Has a Prolonged Half-Life

Compound	$t_{½}$ (Hr)
Ralinepag	~20-26 hrs[3]
Selexipag	8 hrs[2]
Treprostinil	4.5 hrs[1]

Ralinepag's unique PK profile is expected to provide a low peak-to-trough ratio, approximating continuous IV infusion

Conc, concentration; $C_{max/min}$, maximal/minimum concentration; IV, intravenous; PK, pharmacokinetic; $T_{½}$, half-life
[1]Tapson VF, et al. *Am J Respir Crit Care Med.* 2009;179:A1040; [2]Skoro-Sajer N, et al. *Expert Opin Pharmacother.* 2014;15(3):429-436; [3]Arena Pharmaceuticals data on file

Ralinepag Significantly Improved

Pulmonary Vascular Resistance (PVR); ITT Population

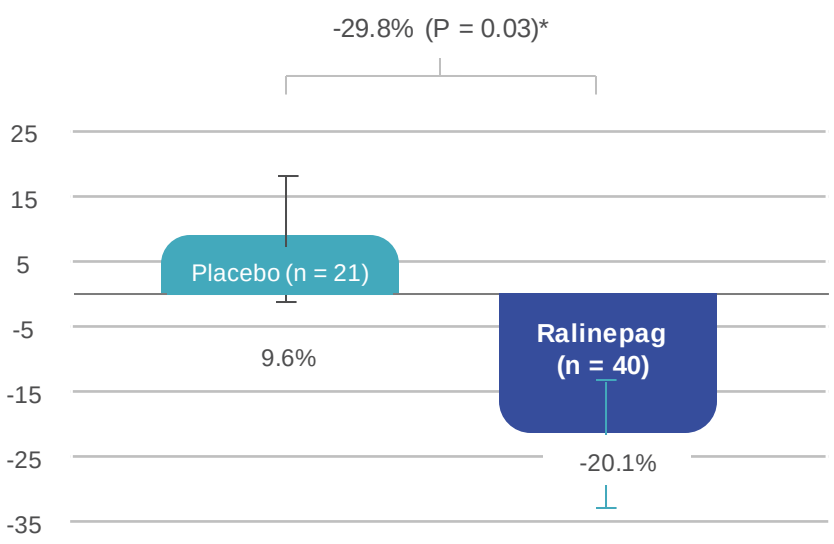
Unprecedented Ph 2 Efficacy in Contemporary Patient Population

Primary Endpoint

Change in PVR Geometric Mean Ratio (GMR)
[Expressed as Percent Change]

Treatment Effect Comparison	-25.8% over placebo (p=0.02)

Secondary Endpoint

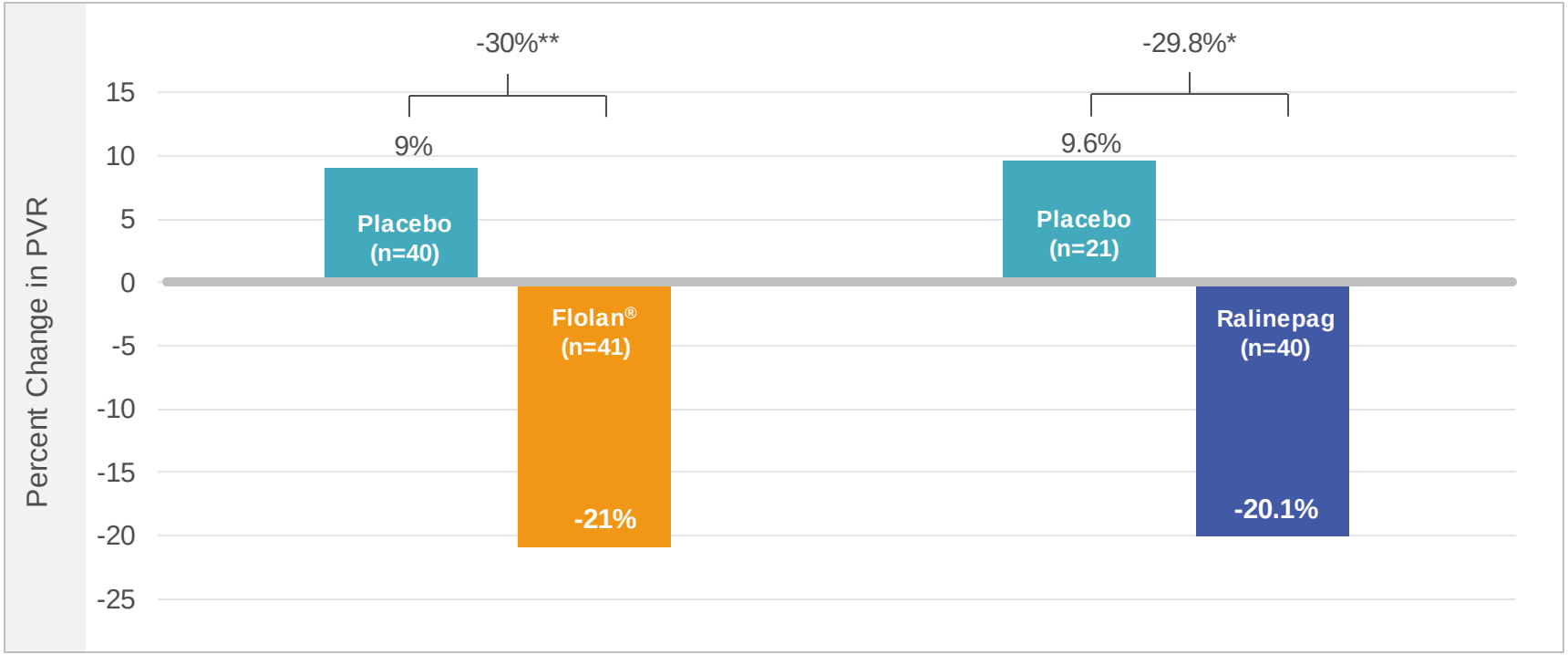


Mean absolute change from baseline: placebo 25.5, ralinepag -175.7 dyn.s.cm-5

* Least squares mean change from placebo; non-parametric test to account for non-normal distribution

PVR Changes With Ralinepag and Historical Flolan®

Note: No direct head-to-head data available - Caution advised when comparing data across clinical studies

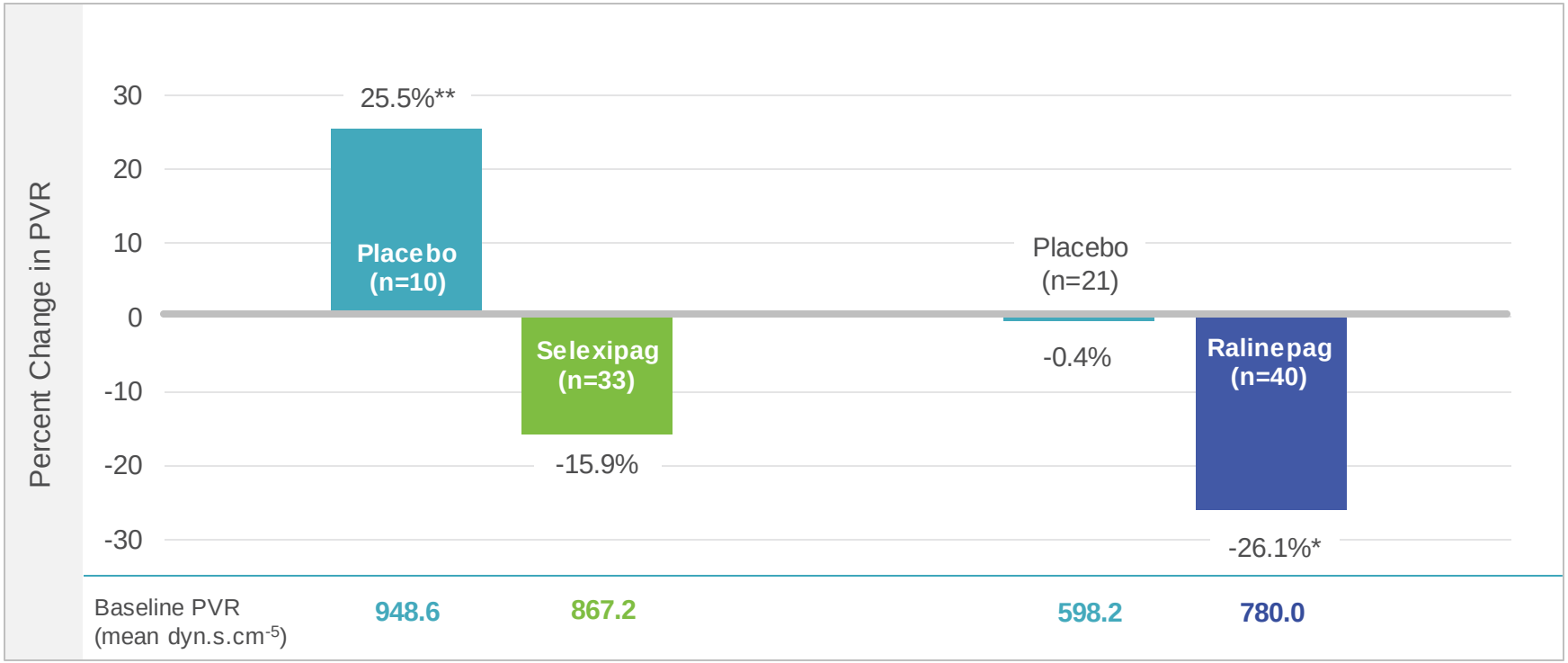


*PVR, change in least squares mean from placebo; **Flolan: Barst et al NEJM 1996 Feb 1;334(5): 296-301

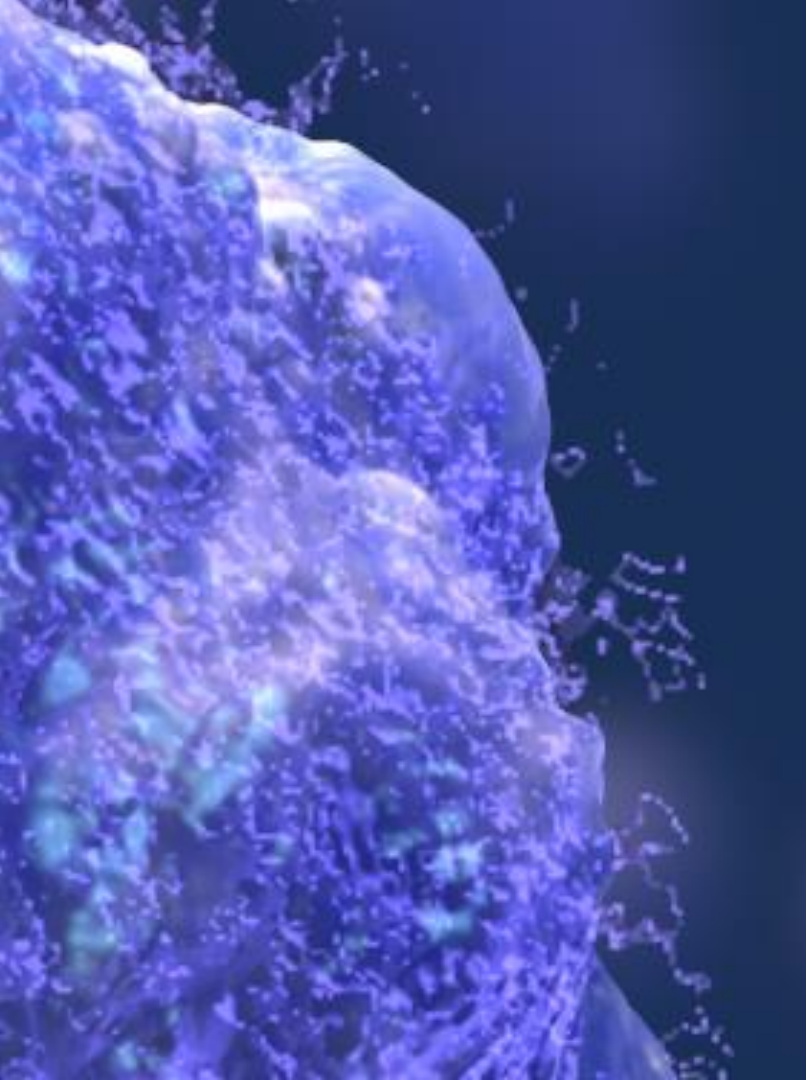
PVR Changes With Ralinepag and Historical Selexipag

ITT Study Populations

Note: No direct head-to-head data available - Caution advised when comparing data across clinical studies



*PVR, change in geometric mean expressed as a percentage of the baseline value; **Selexipag data from P2 study: Medical Review CDER Application: 207947Orig1s000



Phase 2 Safety and Tolerability

The overall safety and tolerability profile consistent with the known profile of the prostacyclin therapy class

Adverse events that occurred most frequently on ralinepag were similar to the on-target effects expected for prostacyclin therapies:

- Headache, nausea, diarrhea, jaw pain, flushing
- Events were more frequent during dose titration; consistent reduction in adverse event frequency during maintenance period

During 25 week safety assessment period 12.5% of patients discontinued ralinepag and 10.0% patients discontinued placebo due to adverse events

Serious adverse events occurred in 4 (10%) patients taking ralinepag and 6 (28.6%) patients taking placebo

- 2 deaths occurred among placebo patients; 0 deaths on ralinepag

Ralinepag is a Potential Best-in-Disease Asset

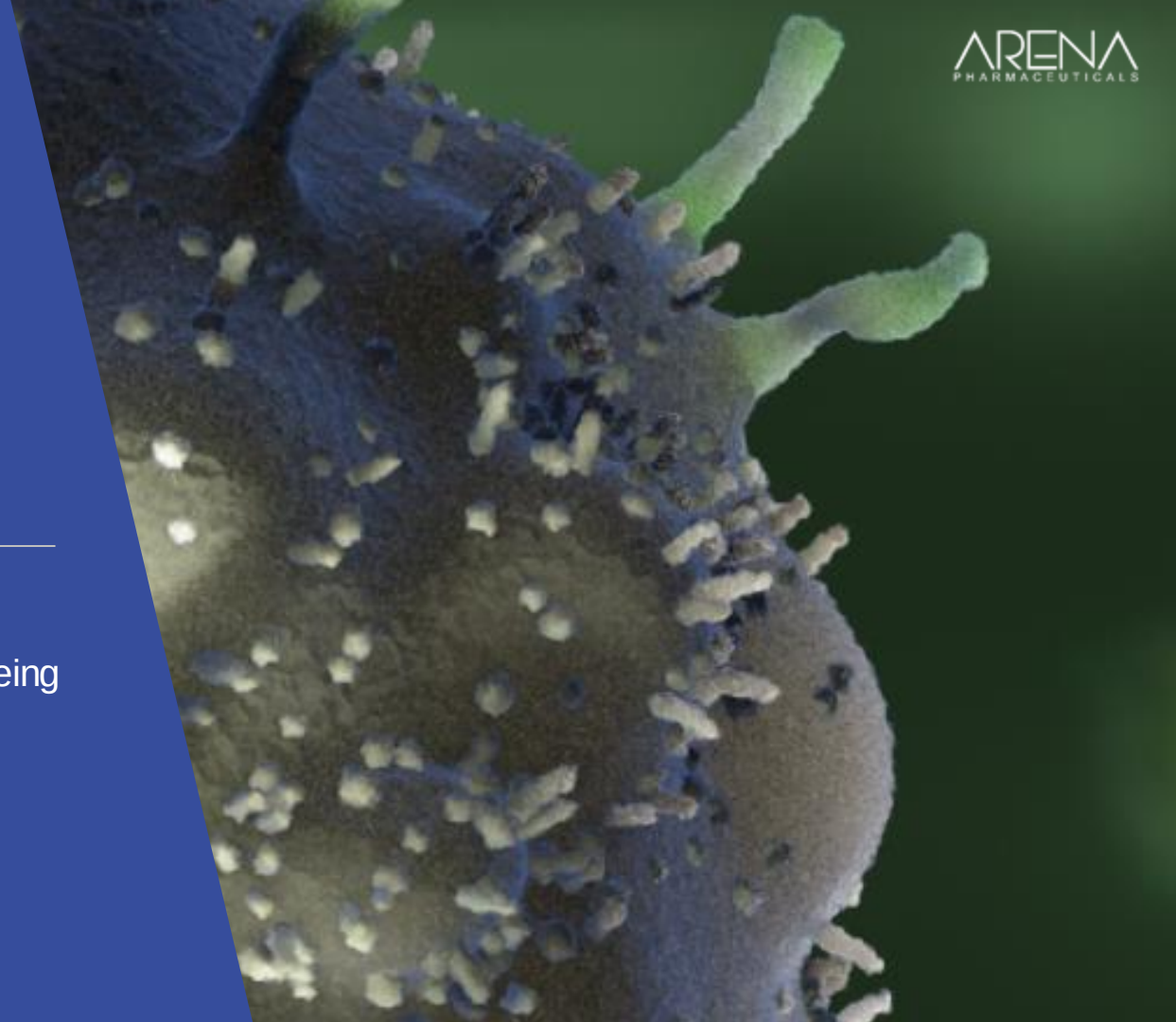
Phase 3 Program Comprehensive Approach

Ralinepag, an oral with IV-like PK/PD, may benefit patients across the treatment continuum

	Naïve / Incident ⟵ ⟶ On Treatment / Prevalent
Exercise Capacity Study *(Registrational)*	**Ralinepag** vs **Placebo** **+ Dual Therapy (PDE5 + ERA)** • First Line / Upfront in ~250 Patients • Endpoints: pVO2, VE/VC02 (CPET), 6MWD • Fixed Treatment Duration
Time to Clinical Events Study *(Registrational)*	**Ralinepag** vs **Placebo** **+ Current Treatment** **(Dual = ERA+PDE5i or Mono=ERA or PDE5i)** • On Treatment / Progressing Disease in ~700 Patients • Endpoints: Clinical Events, Outcomes, Disease Worsening • Event-Driven Duration
Differentiation Studies *(Supportive)*	Not yet disclosed for competitive reasons

Olorinab

Peripherally Restricted, Highly–Selective, Full Agonist to Cannabinoid 2 Receptor (CB2) Being Evaluated for Treatment of Pain Associated with Crohn's Disease



Promise of Olorinab

Unmet Need in Non-Opioid Pain Management

Unmet Need in Pain

Opioid epidemic is a public health emergency

No drugs developed for the treatment of visceral pain including:

- Inflammatory bowel disease
- Interstitial cystitis
- Pancreatitis
- Endometriosis
- Chronic prostatitis

1/8 IBD patients on chronic opioids

Olorinab

Designed to be peripherally restricted

1,000 fold more selective for CB2 vs. CB1

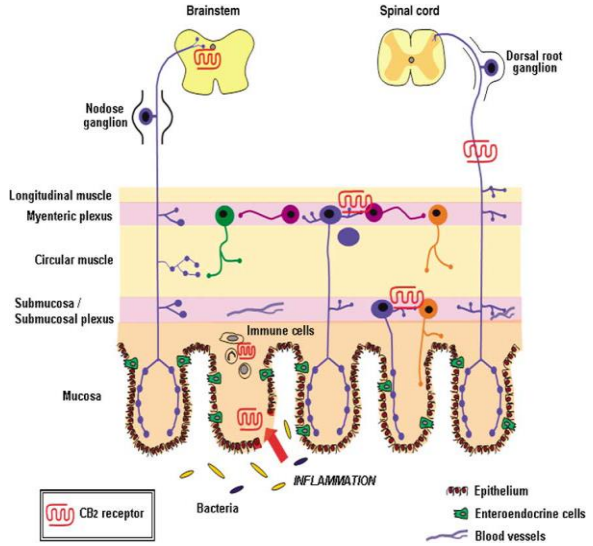
Avoids psychotropic activity

Full agonism avoids tachyphylaxis

Status

Ph 2 ongoing for the treatment of pain associated with Crohn's disease, data Q3'18

CB2 Receptors Are Expressed in the Enteric Nervous System and Immune Cells Within GI Tract



CB2 receptors represent a pathophysiological mechanism in GI tract to regulate abnormal motility, modulate intestinal inflammation & limit visceral sensitivity/pain

CB2 Receptor Expression in the GI Tract

CB2 receptors located in target tissue, neurons & local immune cells in GI tract

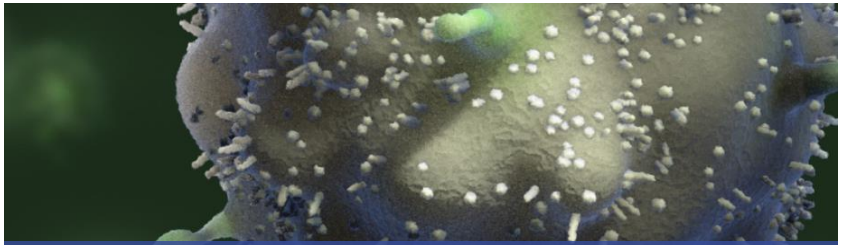
CB2 receptor also expressed on visceral afferent nerves

During inflammation, enhanced expression on epithelial cells at the ulcer margins (red) & infiltrating immune cells

Supporting rationale for inflammatory & non-inflammatory pain signaling

The anti-hyperalgesic actions of the cannabinoid anandamide and the putative CB2 receptor agonist palmitoylethanolamide in visceral and somatic inflammatory pain Sia^n I. Jaggar, Fauzia S. Hasnie, Senthy Sellaturay, Andrew S.C. Rice* ; *Pain* accepted 11 February 1998. Activation of the cannabinoid 2 (CB2) receptor inhibits murine mesenteric afferent nerve activity, Hillsely, Neurogastro Motil (2007) 19, 769–777. Pharmacological Evaluation of a Novel Cannabinoid 2 (CB2) Ligand, PF-03550096, In Vitro and In Vivo by Using a Rat Model of Visceral Hypersensitivity Akira Kikuchi, Pharmacol Sci 106, 219 – 224 (2008)2 Journal of Pharmacological Sciences ©2008 The Japanese Pharmacological Society

Arena Investment Thesis



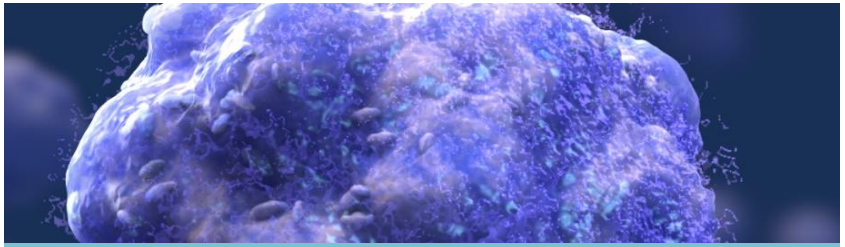
Two potential best-in-class, internally developed drug candidates to Ph 3

Targeting over $30 billion total market opportunity*

Supported by Ph 2 data with clear and meaningful competitive advantages

Retain rights to all major markets and strong intellectual property position

Strong cash balance - Cash and cash equivalents totaled $629.1 M at 3/31/18



Significant catalysts in 2018 and 2019

Olorinab data readout in Crohn's pain

Ralinepag Ph 3 program initiation in PAH

Etrasimod Ph 3 initiation in UC

Etrasimod program initiation in CD

Etrasimod Ph 2 data readout in PBC

Cardiopulmonary pipeline expansion

Additional data readouts across pipeline

*Leerink Analysis & Decision Resources 2016