



NIMMUNE
B I O P H A R M A

COMPANY PRESENTATION | December 2025



Precision Immunology Therapeutics for Human Diseases



Best-in-Class, Biomarker-Driven Therapeutics



- Only private biotech company with a late-stage therapeutic asset for IBD
- Novel, first in class validated anti-inflammatory and immunological targets with gastrointestinal, rheumatic and cutaneous autoimmune indications.
- Clinically de-risked lead programs, designed and developed to maximize therapeutic effect, safety and cost-efficiency with an accelerated path to market.



Highly Experienced Biotech Team

- NASDAQ IPO-ready team with a successful M&A exit, \$170M in equity financing, including Ser. A, B, IPO, \$218m BD deals and >\$20m in non-dilutive rounds.
- IP track record of >200 global patents and >300 publications in I&I.
- Developed a 17-product pipeline and cleared 8 INDs in <4 years.

Late-Stage & High-Impact Therapeutic Assets

- Omilancor and the entire LANCL portfolio utilize a novel mechanism of action compared to other UC and CD treatments.
- Omilancor, Phase 3 LANCL2 agonist, orally active, once-daily, gut-restricted, first-in-class therapeutic for UC. First NDA anticipated in 2027.
- NIM-1324, an orally active, once-daily, systemically distributed drug targeting LANCL2 in lupus, IBD and psoriasis; completed Phase 1 testing and it is Phase 2-ready.



Transformative Drug Development Pipeline

- Portfolio of validated first-in-class oral small-molecule therapeutics targeting large global commercial opportunities.
- Pipeline-in-a-franchise, starting with UC in 2027 and NDAs annually thereafter in multiple I&I indications and formulations.
- Strong global IP estate into 2040s, including composition of matter, method of use, formulation and other patent and intellectual property protections.



Nimmune Leadership Team



Dr. Josep Bassaganya-Riera

Executive Chairman of the Board, President, and CEO

Biotech entrepreneur and innovator with 30+ years of scientific innovation in inflammation and immunology, drug development for inflammatory and autoimmune diseases, business development, biotech fundraising and executive experience. Founded LABP, acquired by Abbvie.



Dr. Raquel Hontecillas

Chief Scientific Officer

25 years of translational experience in immunology, drug development, and the biotech industry focusing on infectious, autoimmune, and metabolic diseases. Former CSO at Landos, acquired by Abbvie.



Marek Ciszewski, JD

Chief Financial Officer

Biopharma and financial industry executive with 30+ years of building and managing financial operations and capital structures for biopharma companies. Former VP of Financial Strategy and Investor Relations at Landos, acquired by Abbvie.



Dr. Andrew Leber

Chief Development Officer

Expertise spans immunology and A.I.-based drug development for autoimmune disease with specific focus on CD, UC, RA, and lupus. Former VP of Scientific & Product Development at Landos, acquired by Abbvie.



Dr. Nuria Tubau-Juni

VP of Inflammation & Immunology

Expertise in I&I related to infectious and autoimmune diseases. She leads new target identification, mechanistic, translational and clinical studies. Former Director of I&I at Landos, acquired by Abbvie.



Active Pipeline: Inflammation & Immunology Drug Development Portfolio

Pathway	Program	Indication	Discovery	IND-enabling	Phase I	Phase II	Phase III	
LANCL2	Omilancor	Ulcerative Colitis						○
		Crohn's Disease						
		Psoriasis						
	NIM-1324	Rheumatoid arthritis						
		Lupus						

Omilancor and the entire LANCL2 portfolio utilize a novel mechanism of action compared to other UC and CD treatments; chemically distinct and utilizing a different pathway altogether than existing pools of follow-on drugs across the market.

Omilancor: Best in Class Therapeutic with Unrivaed Safety

1 in 3 patients respond to omilancor vs 1 in 5 of competitor drugs

MoA	Candidate	Company	2023 Global Sales	Patient Pop.	Induction Remission Rate	Induction Pbo-Adj. Rate	Trial	Safety	Formulation
LANCL2	Omilancor	Nimmune	NA	Mild-to-Sev UC (active disease)	30.4% - 33.3%	26.7% - 33.3%	BT11 UC	No identified trends in AE profiles	Oral QD
Anti-TNFs	Humira (adalimumab)	ABBV	\$14.4B (8 approved uses)	Mod-to-Sev UC	16.5%	7.2%	ULTRA 1 & 2	Infections, lymphoma, heart failure	SC q2wk
	Remicade (infliximab)	JNJ	\$1.8B (6 approved uses)	Mod-to-Sev UC	34%-39% (low dose.hi dose)	19-24%	UC Study 1, 2	Infections, lymphoma, heart failure	IV q8wk
	Simponi (golimumab)		\$2.2B (4 approved uses)		18%	12%	PURSUIT	TB/infections, skin cancer, MS, HF (typically used in TNF naïve pts only)	SC q4wk
Integrin	Entyvio (vedolizumab)	TAK	Peak: \$7.5-\$9.0	Mod-to-Sev UC	16.9%	11.5%	GEMINI 1	TB/infections, PML, liver damage	IV
miR-124	ABX464 (obefazimod)	ABVX	NA	Mod-to-Sev UC	11.3%-23.8%	5.1%-21.4%	ABTECT -1 & -2	1 pt cancer, 2 pts grade >3 infections	Oral QD
IL-7	Lusvertikimab	FRA: OSE	NA	Mod-to-Sev UC	13% high dose 22% low dose	8.6% 17.6%	CoTikiS	No drug related SAEs reported (mAb antagonist of IL-7 receptor)	IV q4wk
IL-12/23	Stelara (ustekinumab)	JNJ	\$10.9B (7 approved uses)	Mod-to-Sev UC (FDA 10/21/19)	19.0%	12.0%	UNIT 1 & 2	Cancers and potentially fatal lung and brain inflammation ("PRES")	IV/SC q2mo
IL-23	Skyrizi (risankizumab)	ABBV	\$7.8B (4 approved uses)	Mod-to-Sev UC	20.3%	14.1%	INSPIRE	Infections, liver damage	IV q4wk
	Tremfya (guselkumab)	JNJ	\$3.2B (3 approved uses)	Mod-to-Sev UC	22.6% (low dose>hi dose)	14.9%	QUASAR	"AEs reported by 49% of pts in both groups."	IV q4w
	Icotrokinra (JNJ-2113)	JNJ	NA	Mod-to-Sev UC	30.2%	19.1%	ANTHEM-UC	Safety data to be presented.	Oral QD
TYK2	Omvo (mirikzumab)	LLY	10/27/23 approved	Mod-to-Sev UC	24.2%	9.1%	LUCENT 1 & 2	Infection, arthralgia, rash, headache	IV/SC q2mo
	Deucravacitinib (sotyktu)	BMJ	Approved for Pso	Mod-to-Sev UC	2 failed P2s; discontinued Oct '23		LATTICE-UC	Not reported	Oral QD
JAK	Rinvoq (upadacitinib)	ABBV	\$4.0B (6 approved uses)	Mod-to-Sev UC	26%-33%	21%-29%	U-Achieve U-Accomplish	FDA/EMU class wide warnings: - "Serious heart-related events, cancer, blood clots, and death for JAK inhibitors to treat certain chronic inflammatory conditions."	Oral QD
	Xeljanz (tofacitinib)	PFE	\$1.7B (5 approved uses)	Mod-to-Sev UC	18.0%	12.0%	Octave 1 & 2		Oral BID
	Jyseleca (filgotinib)	GILD/GLPG	(xUS only) €87.6	Mod-to-Sev UC	26.1%	10.8%	SELECTION		Oral QD
S1P antagonist	Zeposia (ozanimod)	BMJ	\$0.434B (MS & UC)	Mod-to-Sev UC	18%	12%	UC Study 1	PML, low HR, HBP, macular edema	Oral QD
	Velsipity (etrasimod)	PFE	10/13/23 approved	Mod-to-Sev UC	24.8%	20.0%	ELEVATE	Atrial fib, colonic tears, pancreatitis	Oral QD
	Amiselimod	BHC	NA	Mild-to-Mod UC	32.4%	14.6%	NA	Full data to be published	Oral QD
Anti-TL1A	Tulisokibart (MK-7420)	MRK/RXDX	Ph 3 ongoing	Mod-to-Sev UC	26.5%	24.5%	ARTEMIS-UC	NA (infection, infusion reaction)	IV
	Duvakitug (TEV-48574)	SNY/TEVA	Ph3 start in 2025	Mod-to-Sev UC	36.2%-47.8% (lo/hi)	15.7%-27.4%	RELIEVE UCCD	Well tolerated, no AE trends	IV q2wk
	RVT-3101	Roche	Ph 2/3 planned to start in 2025	Mod-to-Sev UC	32% (37% biomarker+)	20% (27%)	TUSCANY	Well tolerated; no identifiable trends in AE profiles	SC monthly

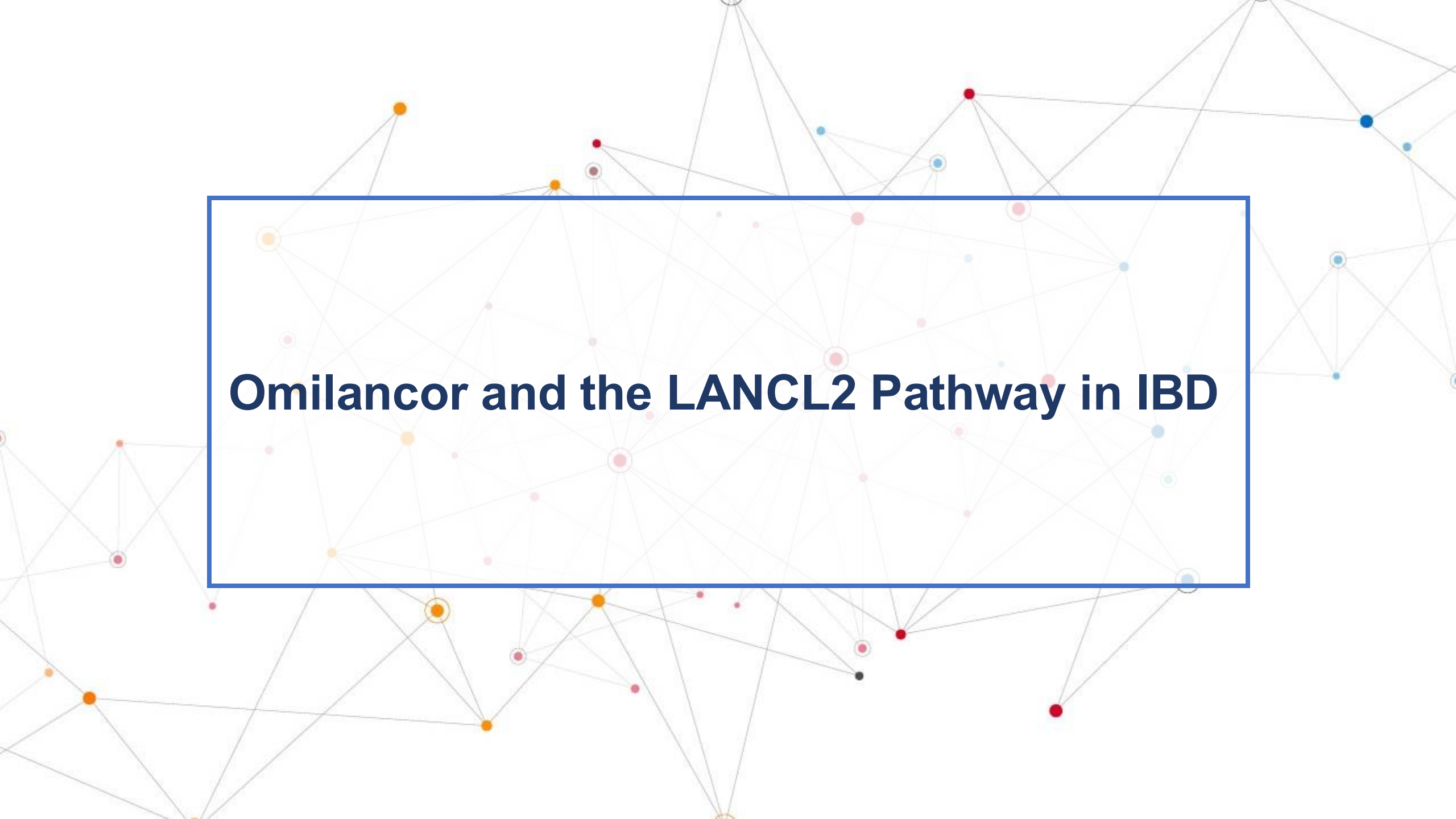


Omilancor vs. Top Anti-TL1As

	anti-TL1a (Merck, Sanofi, Roche)	Omilancor Ph3 dose (NImmune Bio)
Clinical remission	26.5 – 47.8 (Δ = 15.7 – 27.4)	30.4 – 33.3 (Δ = 26.7 – 33.3)
Endoscopic Response	36.0 – 36.8	60.9
Proportion with MES = 3 (at baseline)	66 – 68%	78%
Route of administration	IV or SQ, Q2wk	Oral, once daily
Participants w/ AEs (at P3 dose)	41 – 54%	27%
Immunogenicity	12 – 46% ADA post- induction	None

Data representative of publicly disclosed data:

1. <https://www.ecco-ibd.eu/publications/congress-abstracts/item/op40-pra023-demonstrated-efficacy-and-favorable-safety-as-induction-therapy-for-moderately-to-severely-active-uc-phase-2-artemis-uc-study-results.html>
2. [Press Release: Duvakitug positive phase 2b results demonstrate best-in-class potential in ulcerative colitis and Crohn's disease](#)
3. <https://investor.roivant.com/static-files/7eabab02-5d9f-471d-8aaa-4da26c486bcf>

A complex network diagram with numerous nodes of various colors (orange, red, blue, pink, yellow) connected by thin grey lines, forming a dense web. A central rectangular box with a blue border contains the title text.

Omilancor and the LANCL2 Pathway in IBD



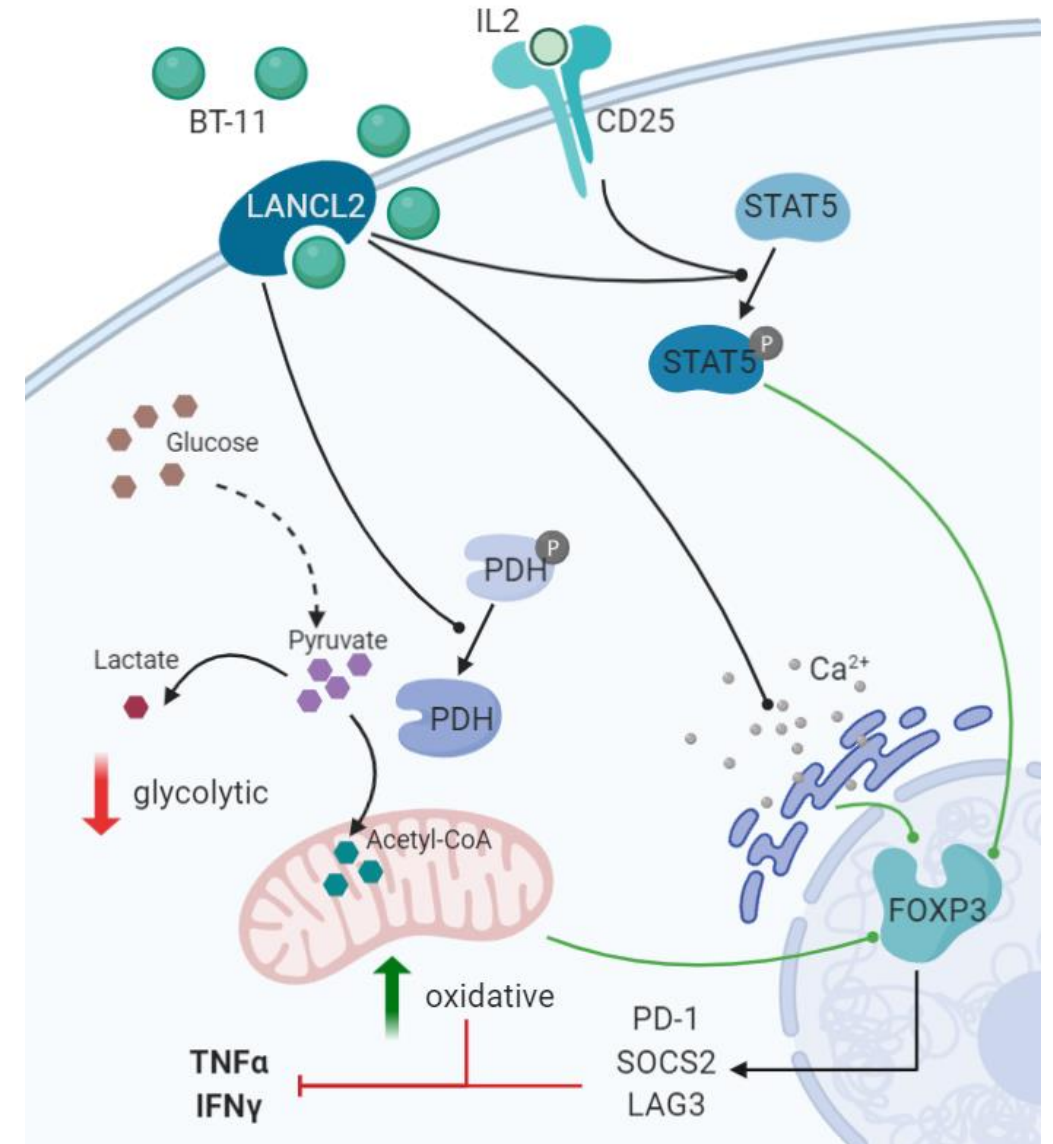
Omilancor Activates Novel Immunoregulatory Target LANCL2

Lanthionine Synthetase C-Like 2 (LANCL2):

- Multipronged mechanism of action targeting known immunological targets downstream tied to inflammatory and autoimmune diseases, including IBD
- LANCL2 controls how the immune system is kept in check

Omilancor generates suppressive regulatory CD4⁺ T cells (Tregs) that restore and maintain immune tolerance in the GI tract:

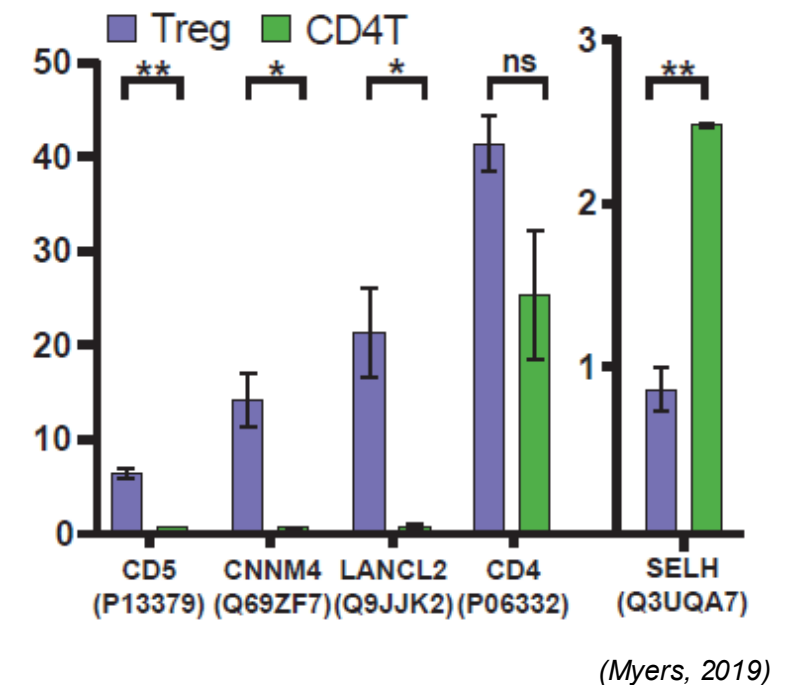
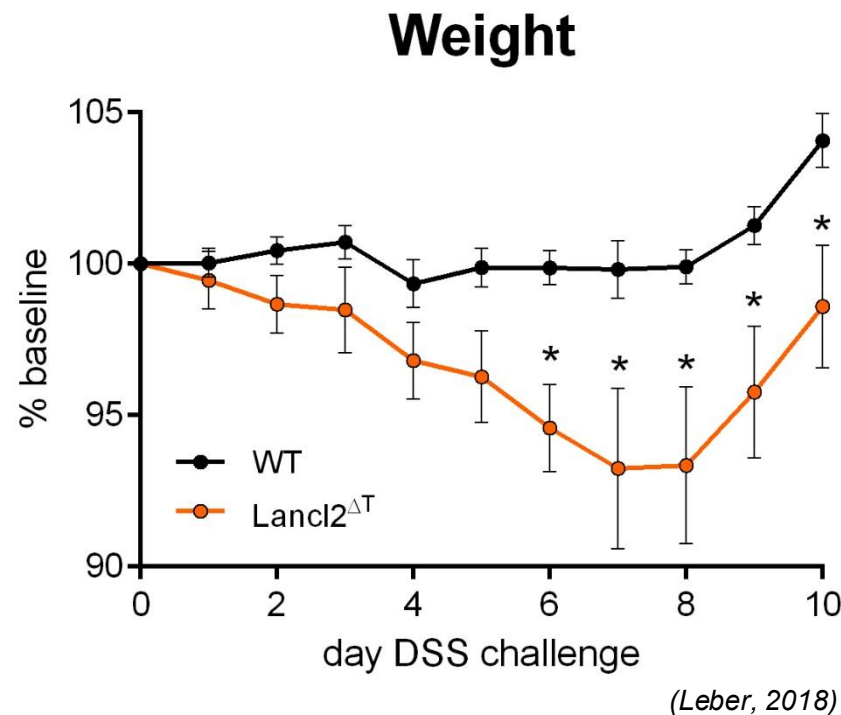
- ✓ Enhances CD25/STAT5 signaling to support the stable differentiation of regulatory CD4⁺ T cells with greater anti-inflammatory functionality
- ✓ Increases PDH activity, resulting in increased oxidative metabolism supporting FOXP3 stability
- ✓ Increases suppressive effects of Tregs due to enhanced immune checkpoint surface markers (LAG3 and PD-1)





LANCL2 is a novel Treg associated receptor relevant to IBD

- *LANCL2^{-/-} mice have been characterized in multiple autoimmune conditions*
- *The loss of LANCL2 in CD4⁺ T cells results in worsening of disease severity and Treg defects in a DSS model of colitis*
- *LANCL2 was identified to be one of the three most differentially expressed proteins in Tregs relative to CD4⁺ T cells as a whole*



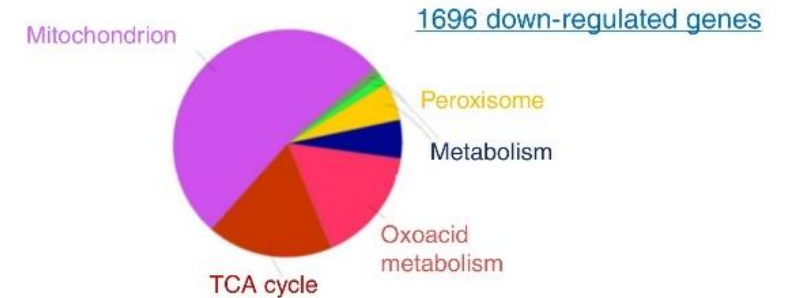
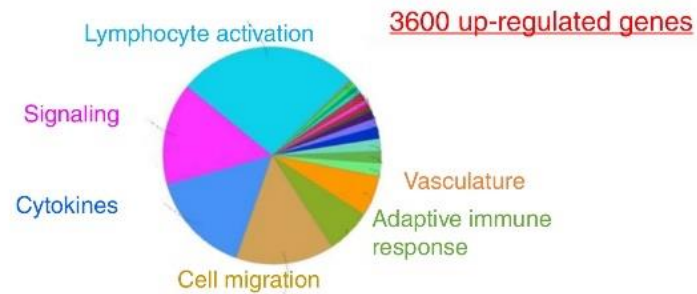


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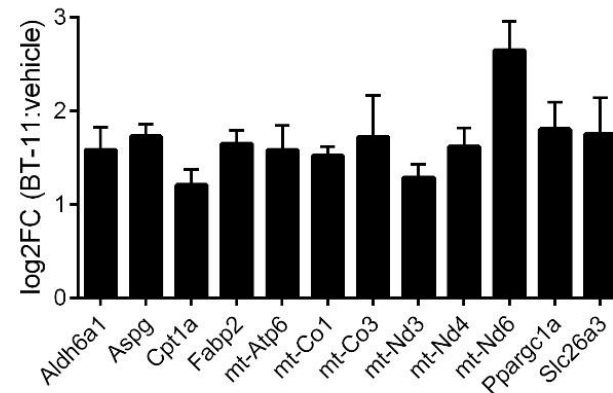


Immunometabolism in IBD

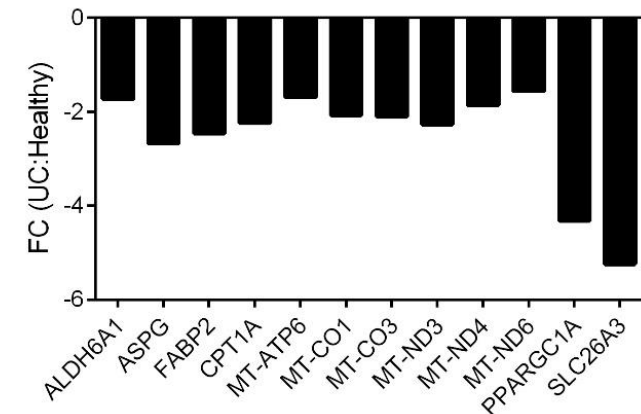
- The majority of upregulated genes in IBD are associated with immune activation or immune cell recruitment
- 90% of downregulated genes in active IBD are associated with metabolism, in particular mitochondrial metabolism (Haberman, 2019)
- Activation of LANCL2 through omilancor reverses the down-regulation of key mitochondrial metabolism genes



Differentially expressed metabolic genes in colon

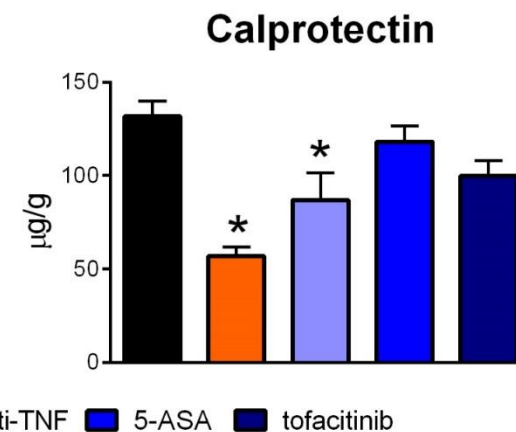
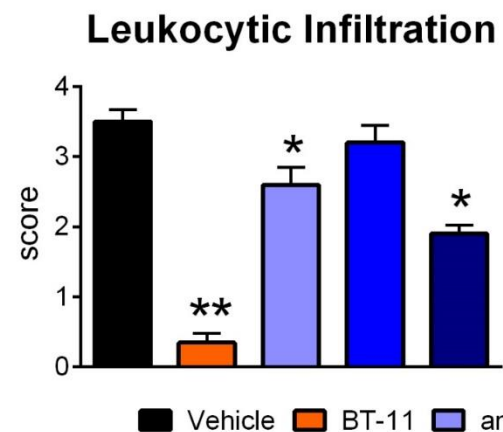
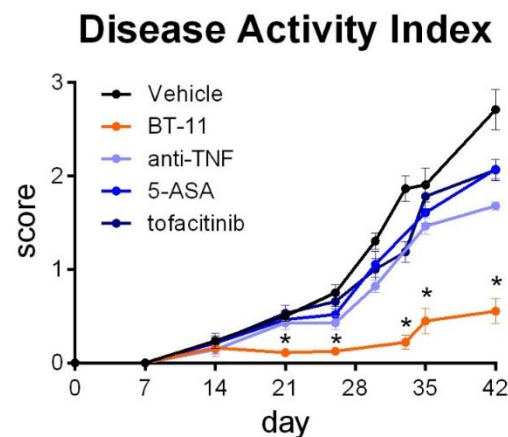
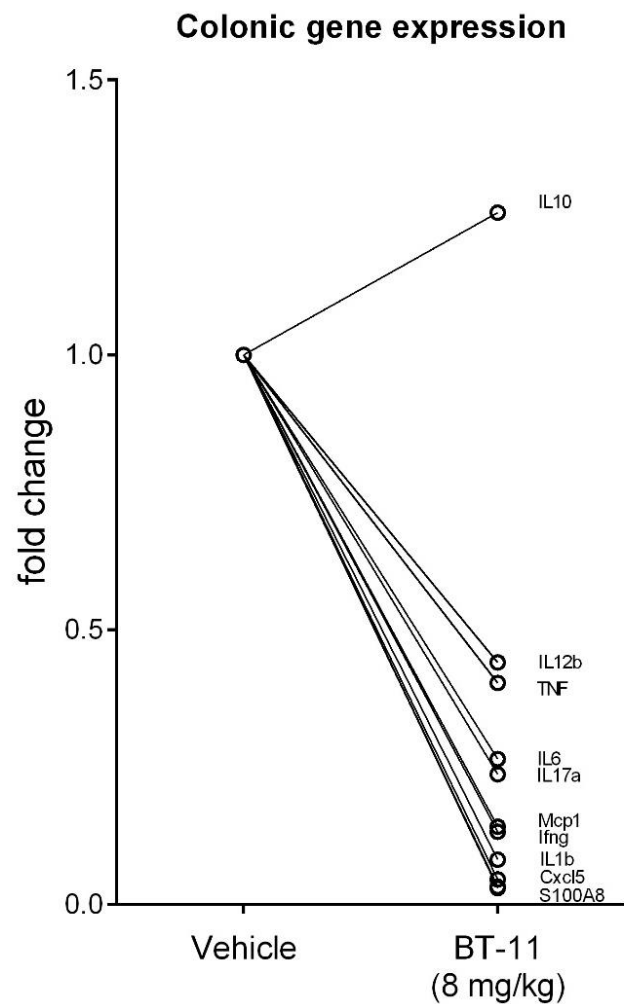


Differentially expressed metabolic genes in colon



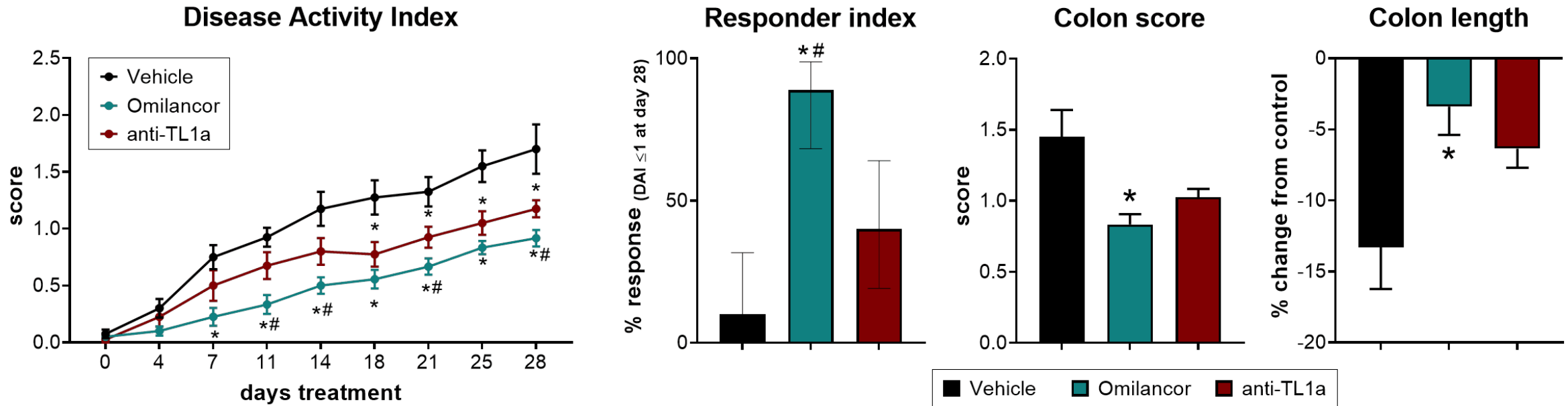


Preclinical Efficacy - Omilancor



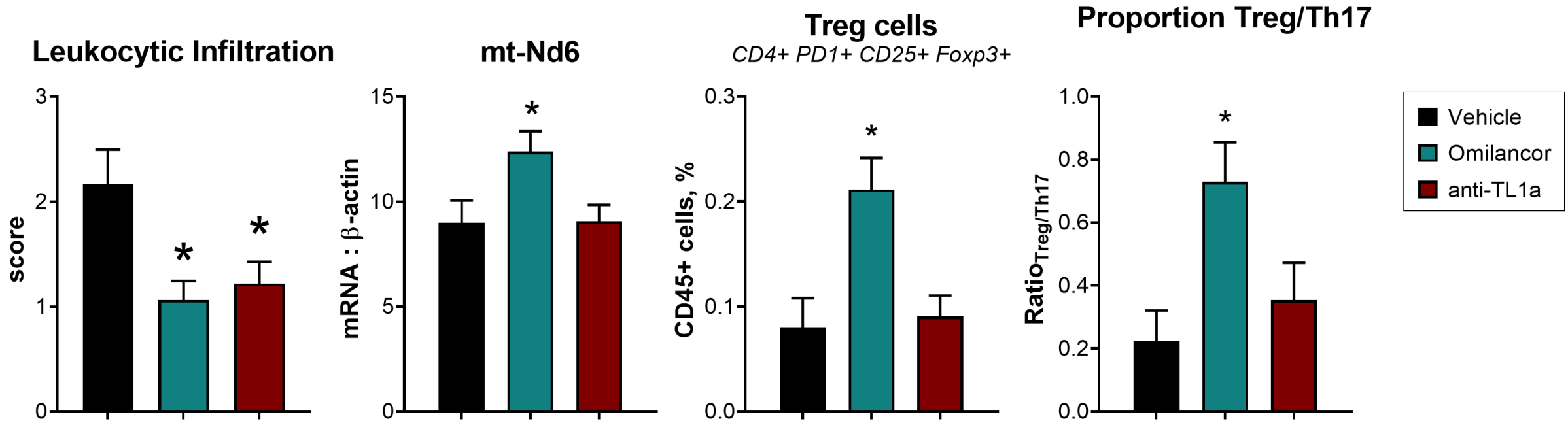
- ✓ Effective in 5 mouse models of IBD and a pig model of IBD
- ✓ Outperforms standard of care and recent approvals in UC including anti-TNF and JAK inhibitors
- ✓ Validated local PK/PD relationships in the colons of mice and pigs without the need for systemic exposure or specialized formulation
- ✓ Identified 8 mg/kg (440 mg human equivalent) as optimal dose

Comparative efficacy of omilancor and anti-TL1a in an Mdr1a-/- model



Omilancor decreases disease activity and macroscopic scores of colonic appearance and length while increasing the proportion of responders relative to vehicle and TL1a neutralization.

Comparative efficacy of omilancor and anti-TL1a in an Mdr1a-/- model

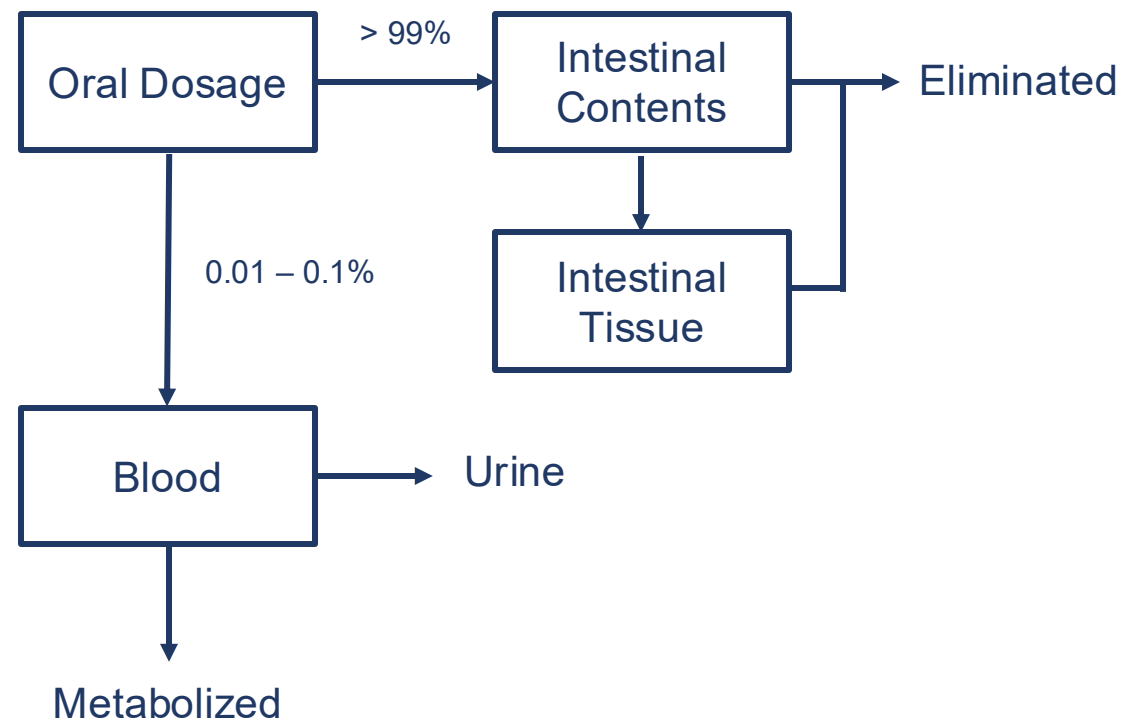


Omilancor and TL1a neutralization effectively reduce histological disease severity. The omilancor group presented with an advantage in all parameters evaluated relative to TL1a neutralization.



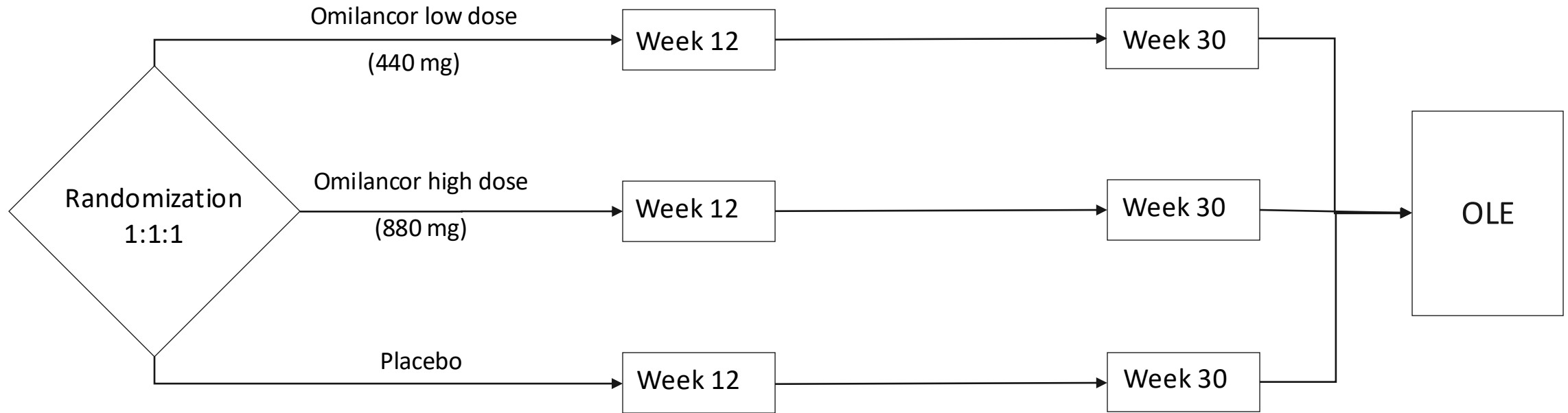
Safety and Pharmacokinetics - Omilancor

- ✓ No adverse event trends or AEs of special interest at doses of up to 6600 mg for 7 days or 880 mg for over 1 year in humans
- ✓ NOAEL > 1000 mg/kg/d in pivotal 6-month rat and 9-month dog GLP toxicology studies
- ✓ No effects on carcinogenicity, genotoxicity or reproductive, cardiovascular, central nervous, and respiratory systems in targeted nonclinical studies
- ✓ No restrictions on ADME-based drug-drug interactions





Completed First-in-Patient Study Design of Omilancor in UC

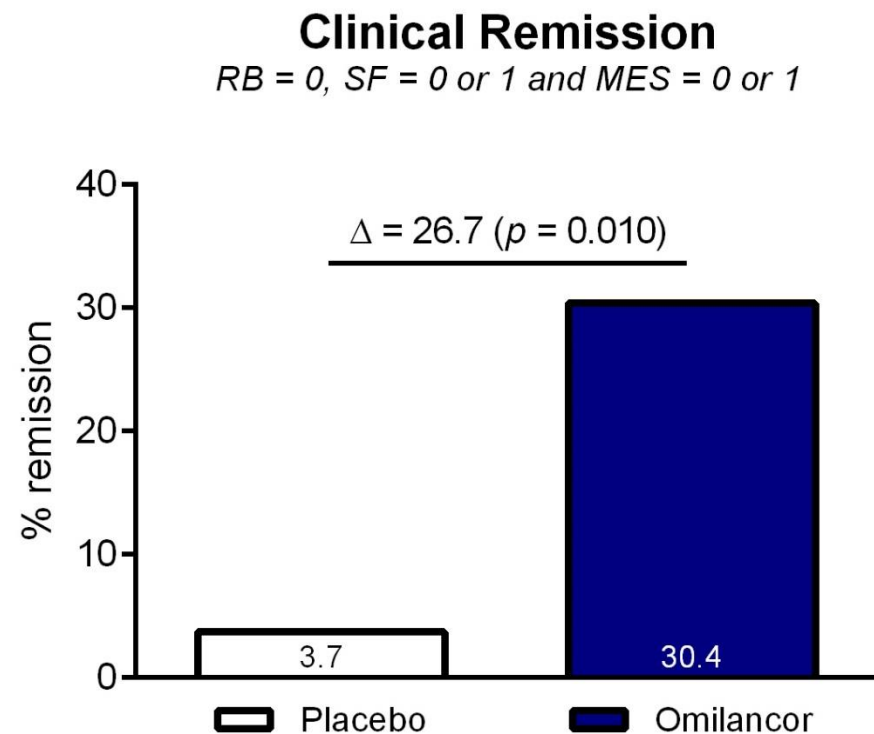


- Primary Objective
- The primary objective of this proof-of-concept study was to establish the efficacy of oral BT-11 in inducing clinical remission at Week 12 in subjects with ulcerative colitis (UC).
- Key Inclusion Criteria
- Male and female subjects with active UC defined by a total Mayo Score of ≥ 4 with MES ≥ 2 (confirmed by central reader); 5-aminosalicylates (max 4.8 g/day) and oral corticosteroids (max 20 mg/day prednisone or equivalent) must be stable for the 12-week induction period.



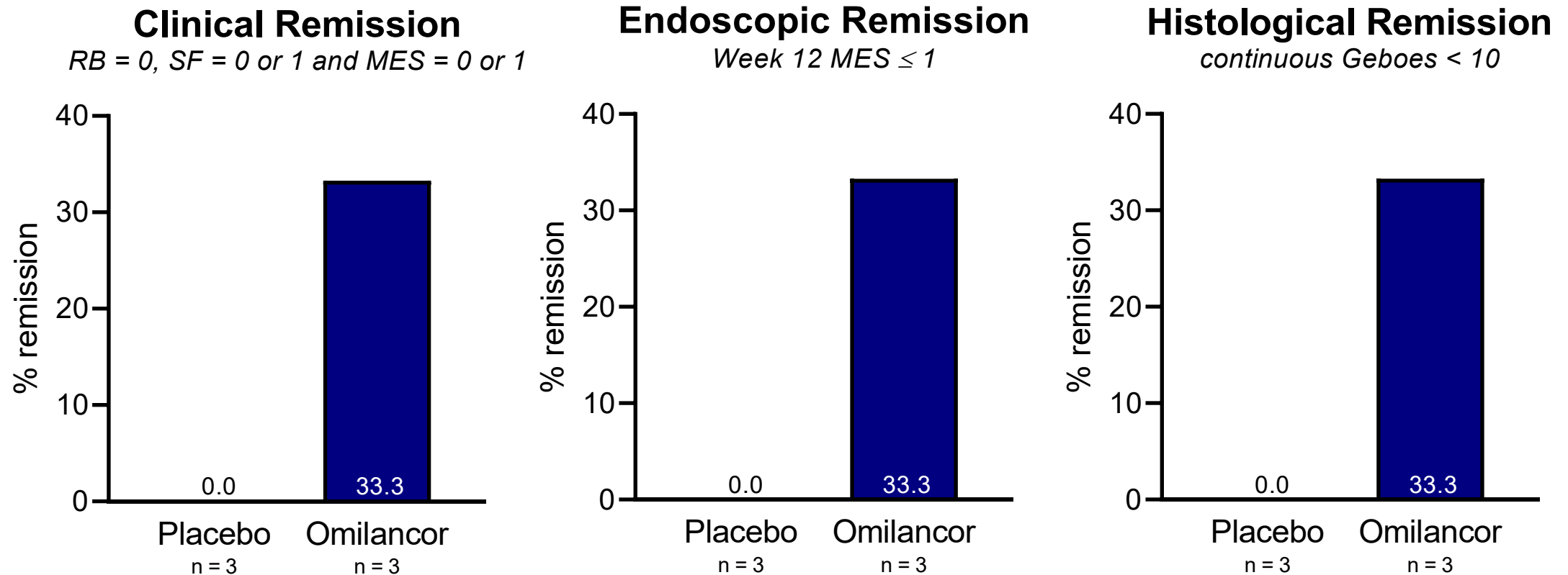
Positive Clinical Remission Rates

Primary endpoint definition	Clinical remission at Week 12 as defined by stool frequency of 0 or 1, rectal bleeding of 0 and Mayo endoscopic subscore of 0 or 1
Analysis population	Subjects with rectal bleeding greater than 0, histological activity and elevated fecal calprotectin at baseline
Analysis method	Stratified Cochran-Mantel-Haenszel Method
Stratifications	Previous biologic usage Corticosteroid use at baseline





Omilancor provides consistent induction responses in biologic experienced UC patients



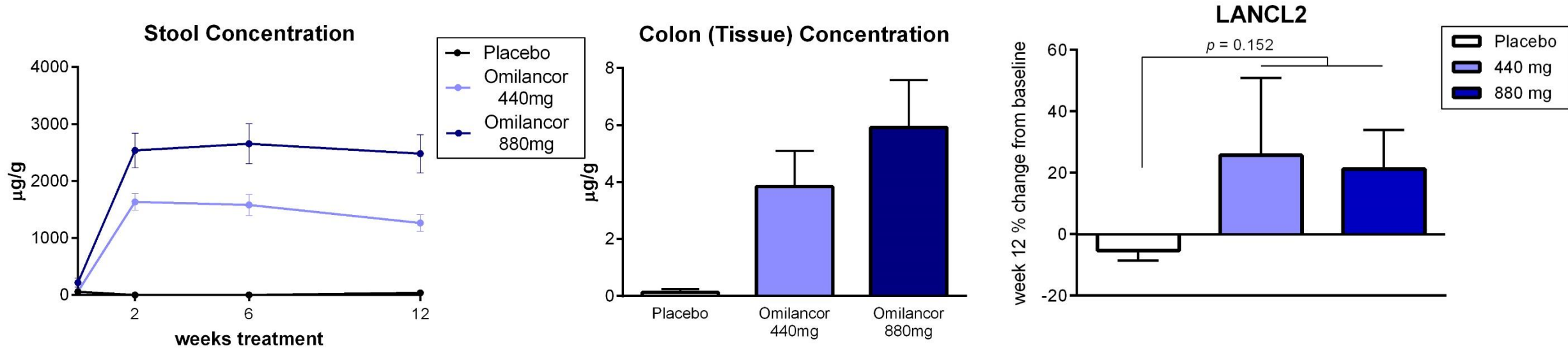


Statistically Significant Approvable Primary Endpoint

	Clinical Remission	Durable Remission
Omilancor (n = 23)	30.4	38.5
Placebo (n = 27)	3.7	21.4
Δ (P value)	26.7 (0.01)	17.1 (0.05)



PK/PD results validate sufficient target engagement at both doses

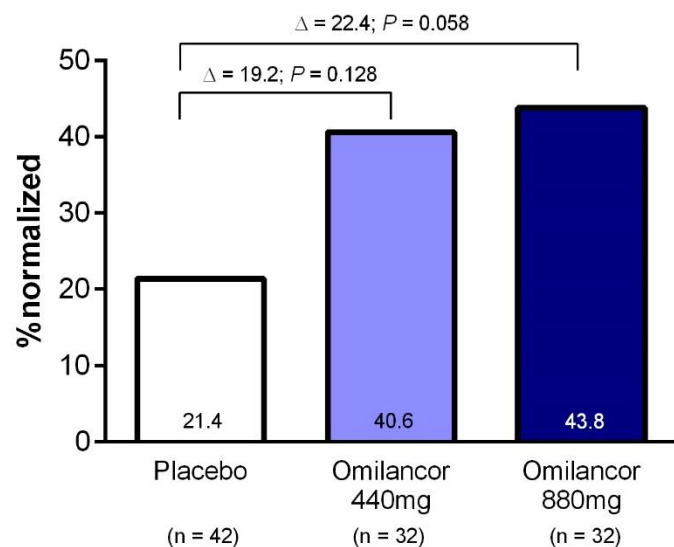


- Omilancor stool concentrations stable between 2 and 12 weeks of dosing
- No significant difference in stool concentrations between UC patients after 12 weeks and healthy volunteers after 7 days
- Stool and tissue concentration scale in a near dose-proportional manner
- 440 and 880 mg doses effectively clear therapeutic threshold, engage LANCL2, and increase LANCL2 expression in the colon

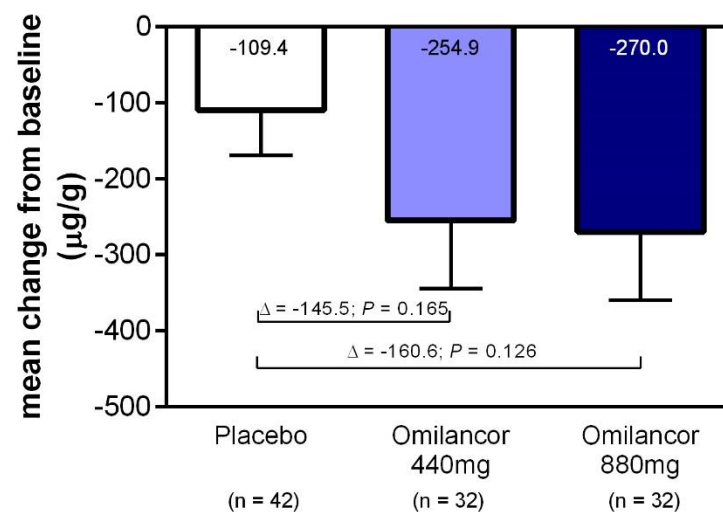


Biomarker Response After 2 Weeks of Treatment

Fecal Calprotectin Normalization
week 2



Fecal Calprotectin
week 2

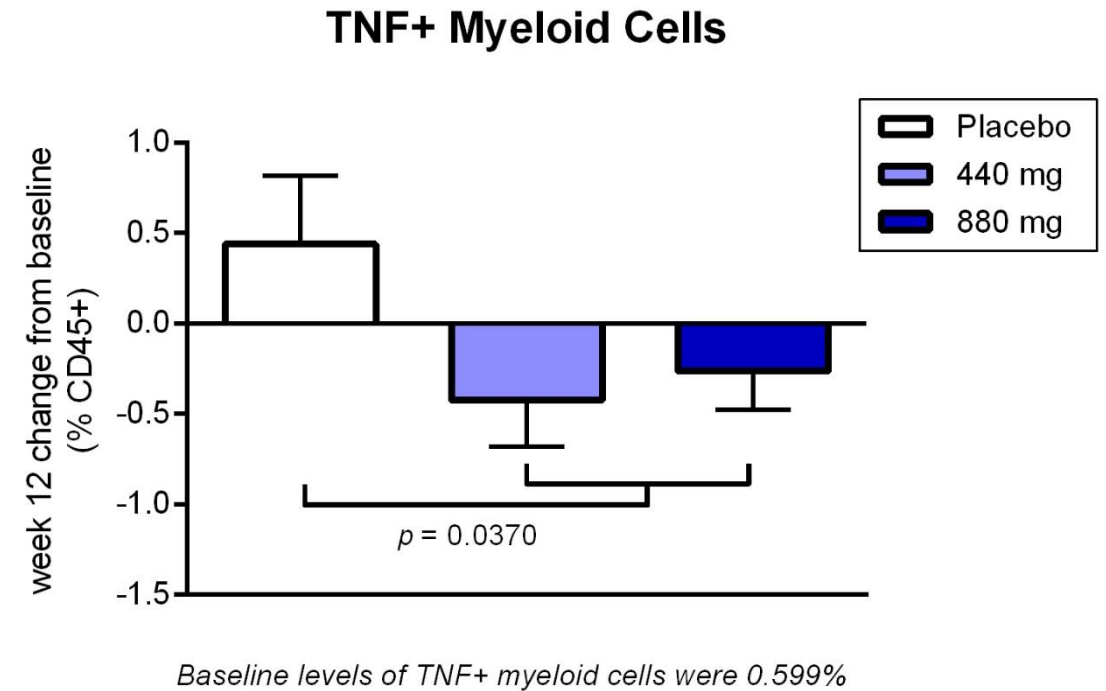
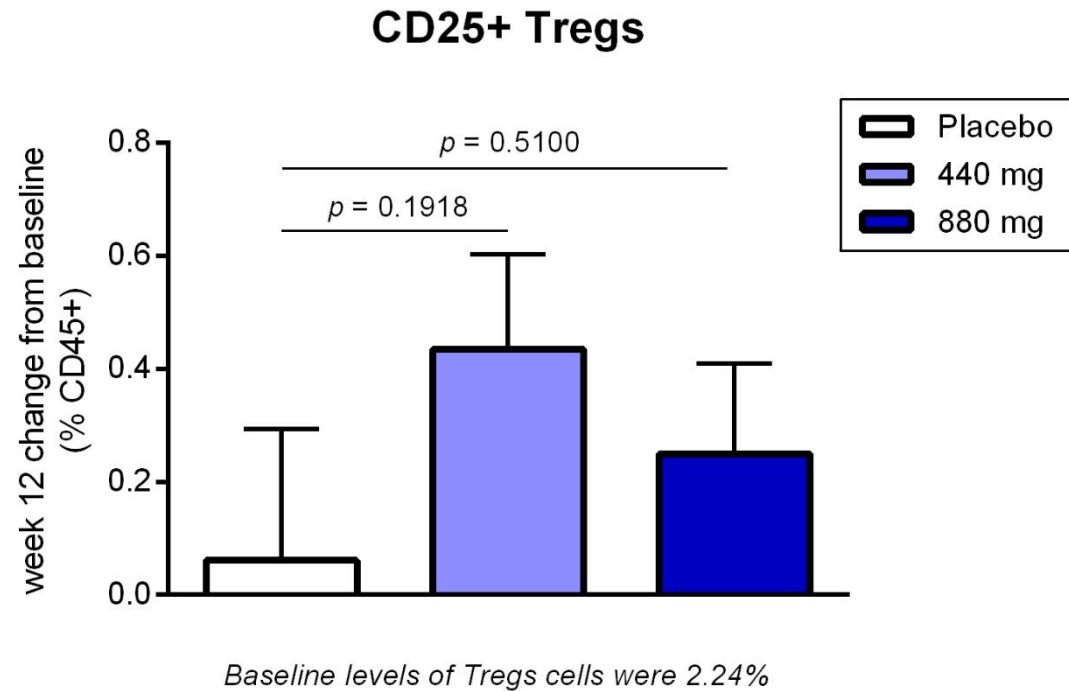


*Fecal calprotectin considered normalized at < 250 ug/g
Inclusive of subjects with abnormal levels at baseline*

	Rate	Placebo Adjusted
Normalization < 250 ug/g		
Omilancor (440 mg) Week 2	40.6	19.2
Tofacitinib Week 12	29.0	N/A
Ustekinumab Week 8	30.3	8.5
Normalization < 150 ug/g		
Omilancor (440 mg) Week 2	33.3	15.1
Vedolizumab Week 6	29.3	12.5



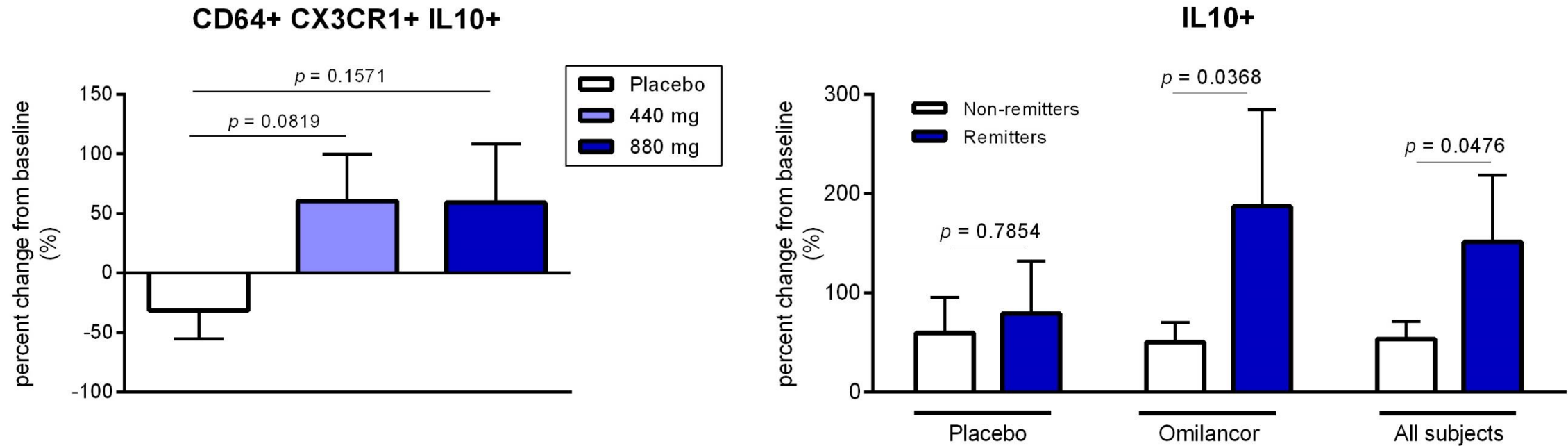
Colonic Tregs, TNF-producing Cells, TNF and IL-6 Concentrations



Omilancor induced increased levels of regulatory CD4+ T cells and myeloid cells, increased IL-10 expression in remitters, decreased TNF expressing myeloid cells, decreased IL-6 colonic concentrations by 55% and TNF concentrations by 44% relative to patients receiving placebo.



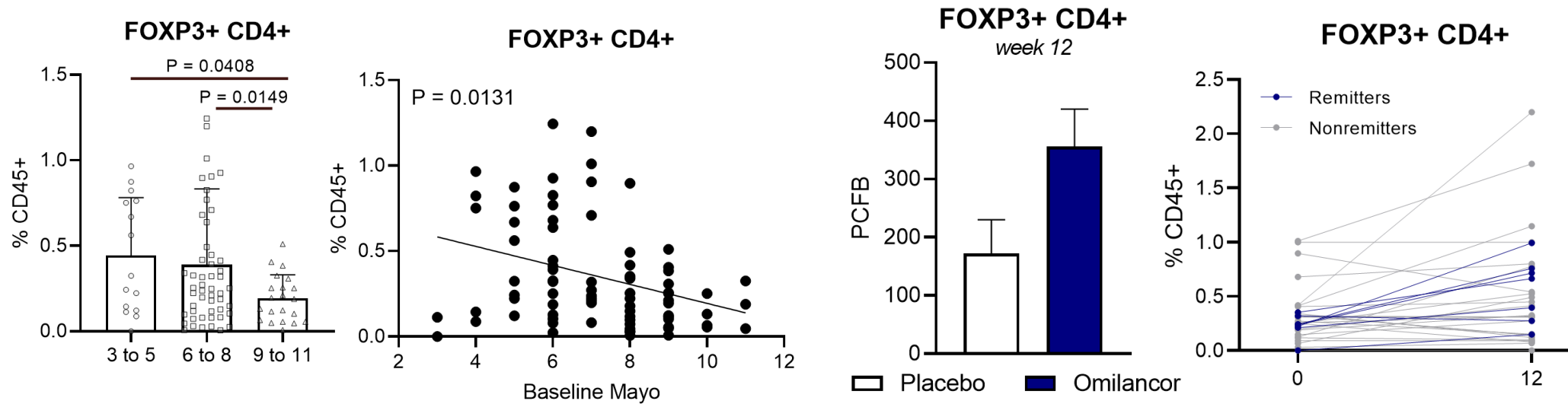
Increase in Colonic IL-10+ Cells Associated with Low Disease Activity



CD64+ and CX3CR1+ regulatory macrophages are key producers of IL 10 and were associated with lower disease activity scores preclinically after omilancor treatment ($p = 0.0378$)



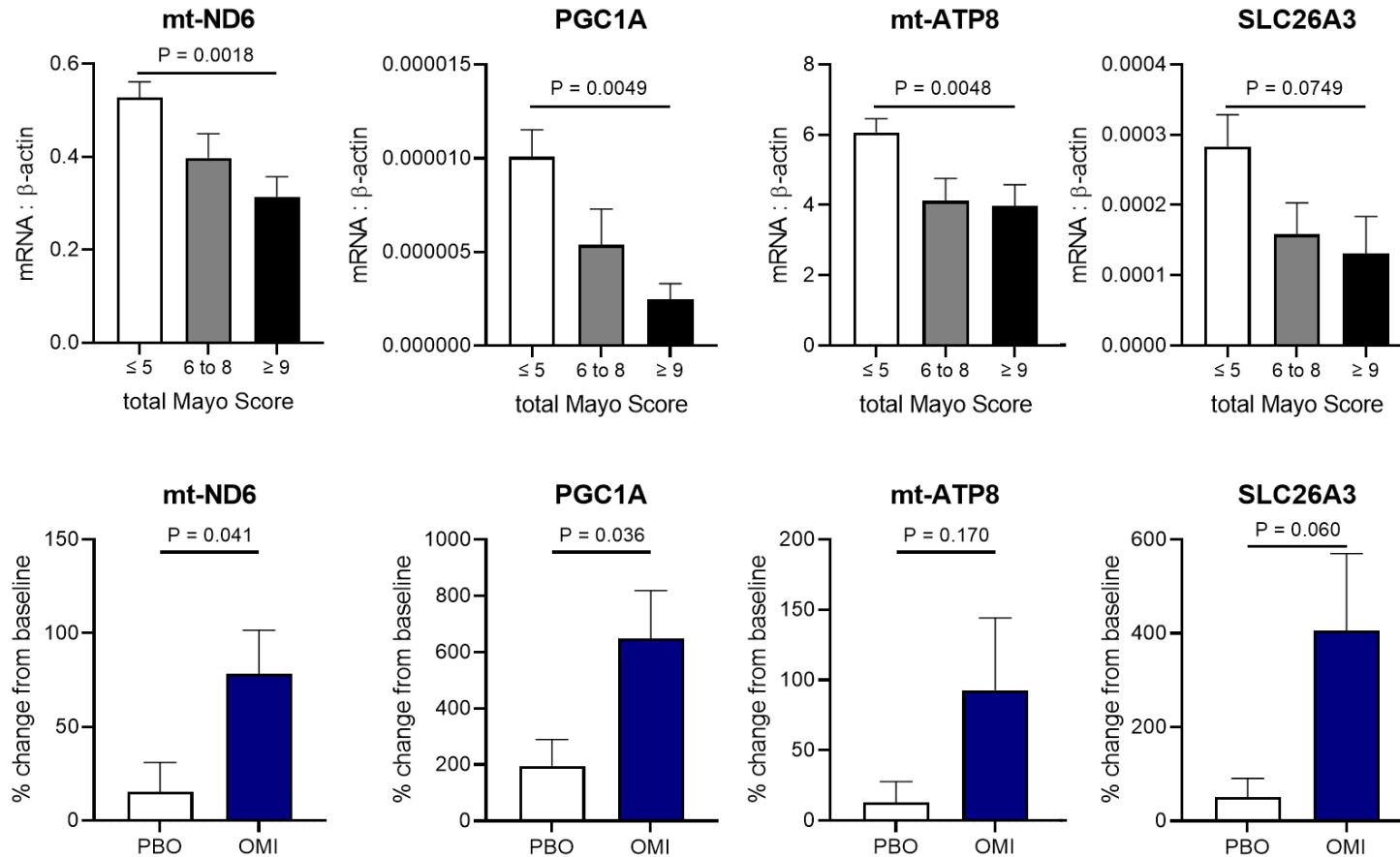
Omilancor Reverses Depletion of Tregs in Severe UC patients



85% of patients in clinical remission after omilancor treatment experienced an increase in Treg abundance from baseline.



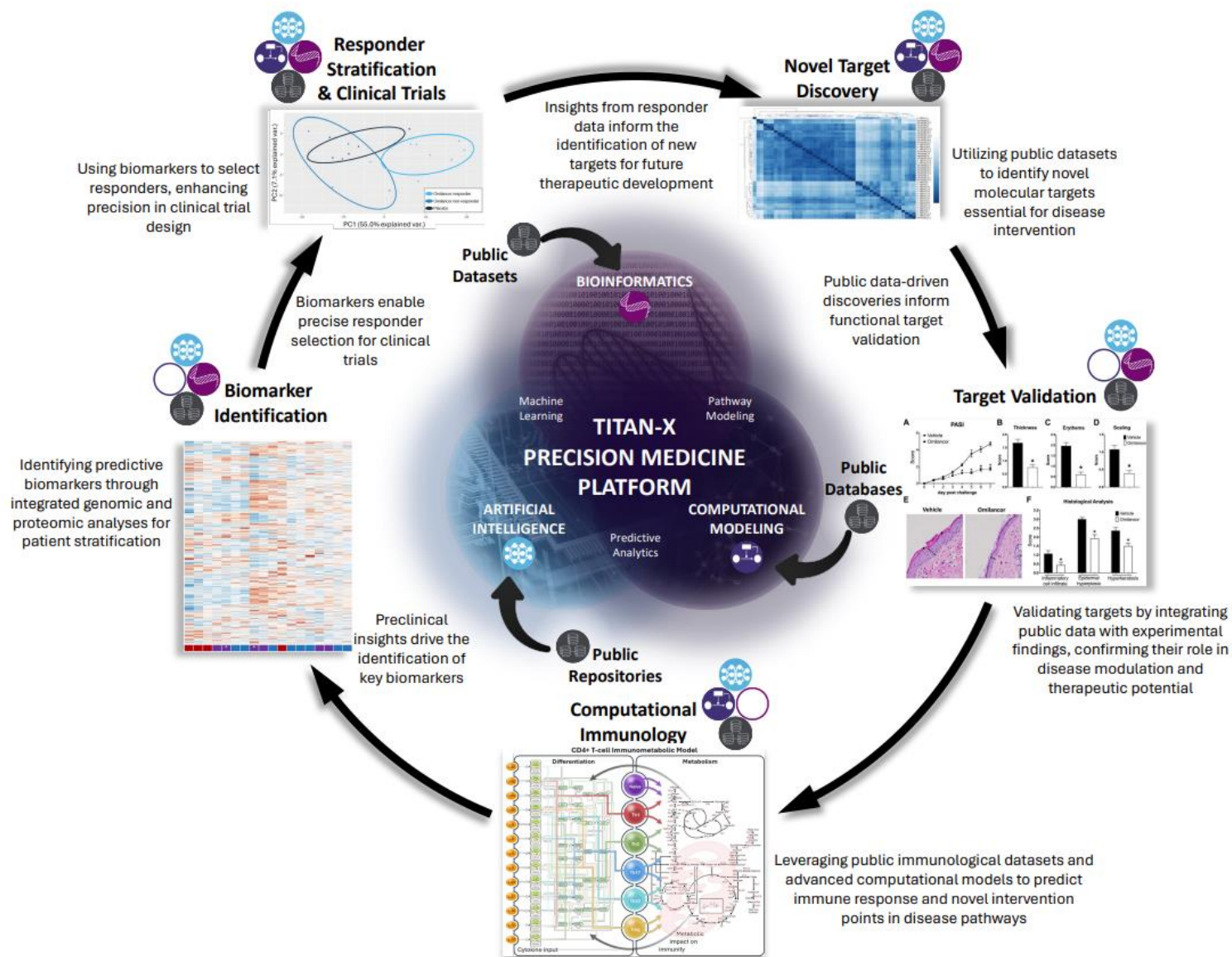
Omilancor Rescues Dysfunctional Mitochondrial Metabolism in Severe UC



Genes are associated with mitochondrial metabolism and are critical to the support of epithelial barrier integrity.

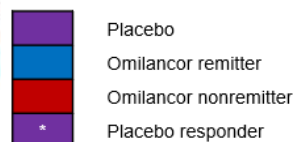
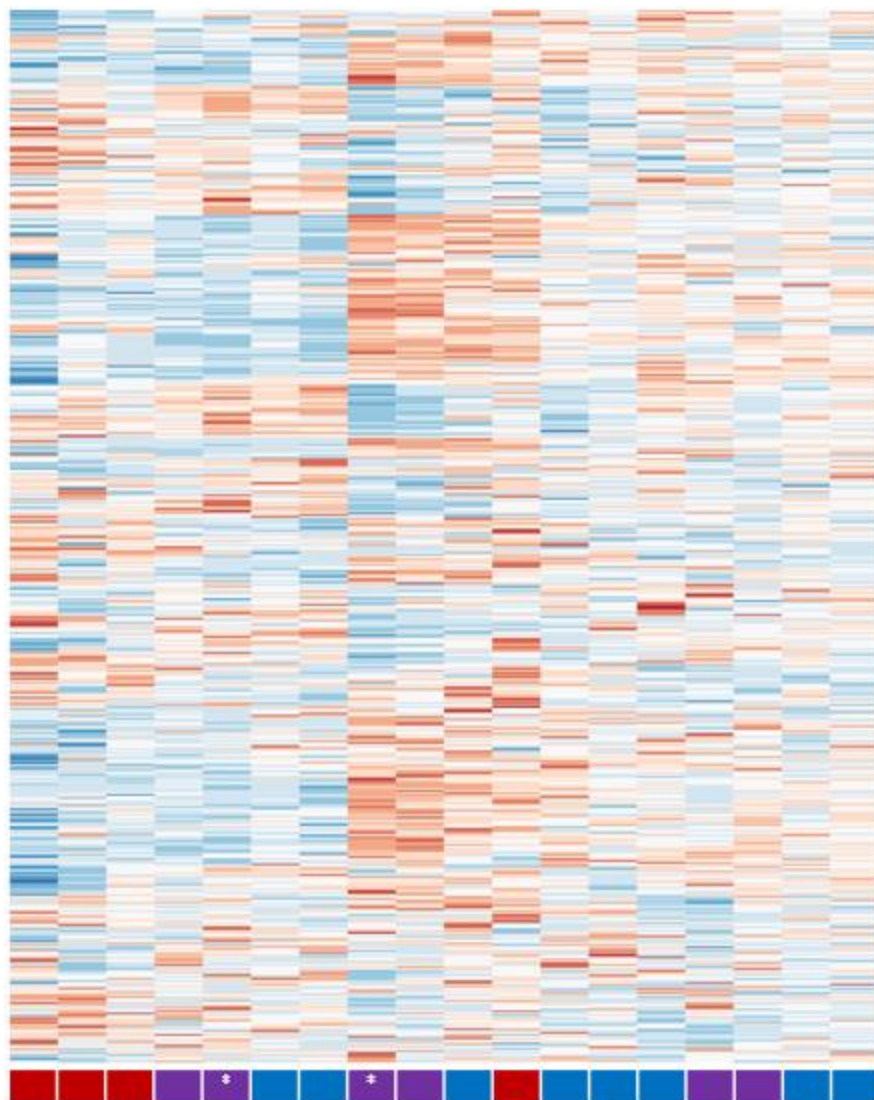


TITAN-X Precision Medicine Platform

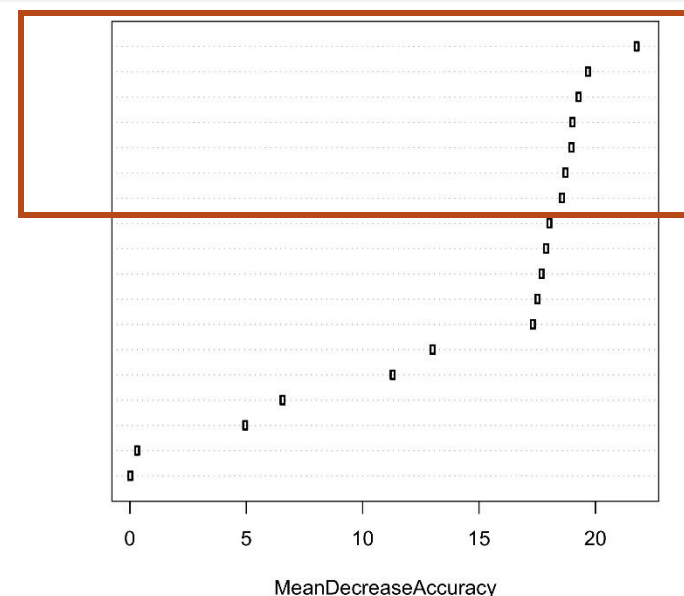




Colonic gene expression pattern associated with clinical response to omilancor treatment



Gene expression analysis of colonic biopsies of ulcerative colitis patients treated with omilancor results in clear pattern separating responders from non-responders. In particular, 7 individual genes comprise a predictive model with accuracy of 75% or higher.



Overall accuracy – 75%

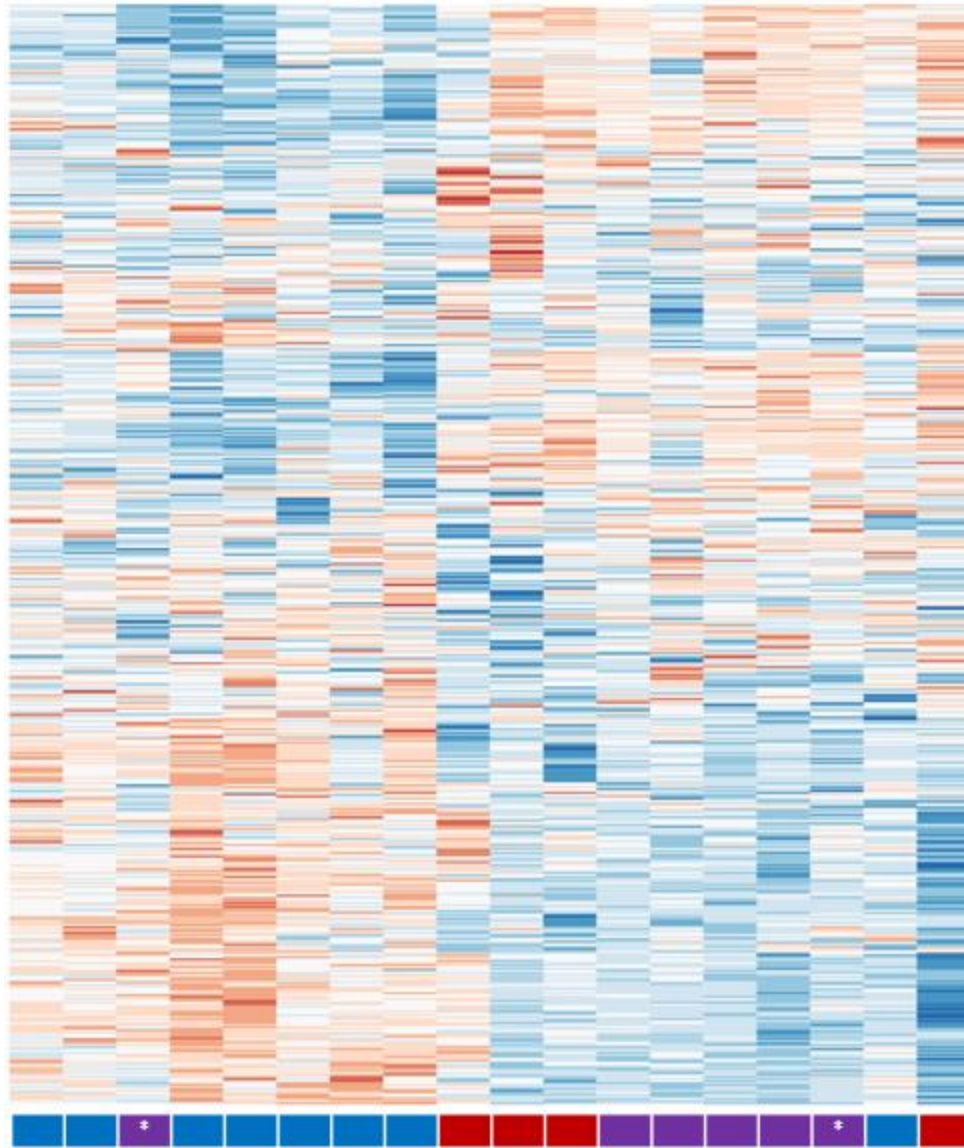
Omilancor remitters 75%

Omilancor nonremitters 75%

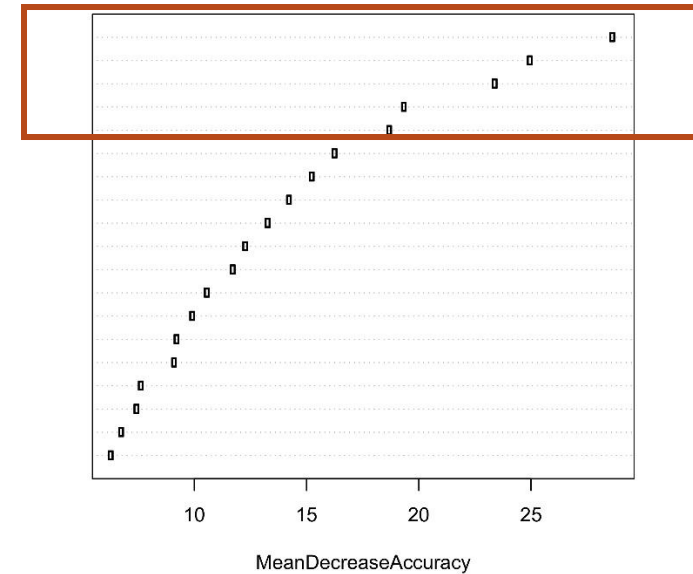
Model predicts response to omilancor would occur in 50% of patients who received placebo



Colonic gene expression pattern associated with clinical response to omilancor treatment



- DEGs upregulated by omilancor relative to baseline and placebo were associated with lipid metabolism and ion balance in addition to known elements of the LANCL2 pathway.
- DEGs downregulated were associated with immune system processes, primarily those in relation to neutrophils and leukocyte trafficking.

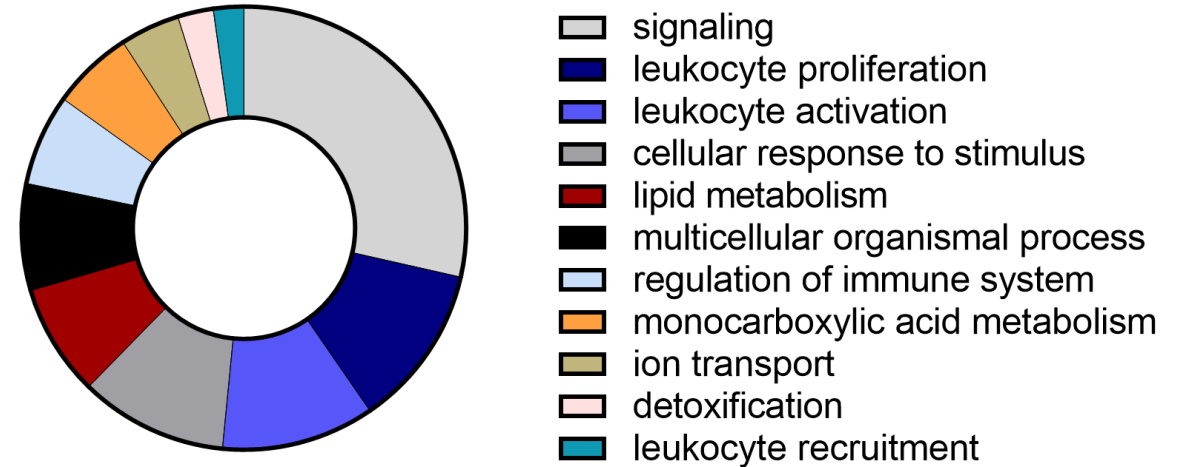
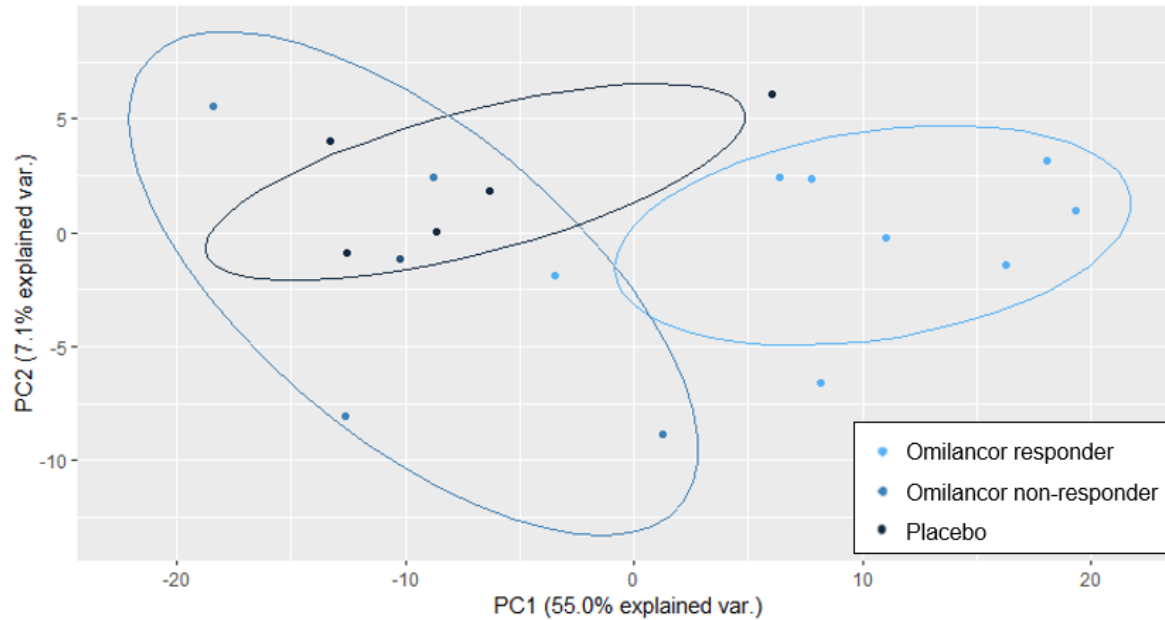


Overall accuracy – 83%

Omilancor remitters	100%
Placebo	83%
Omilancor nonremitters	50%



Colonic biopsies have mechanistic and predictive value in omilancor response



The ability of our Week 12 gene signature was validated by PCA, wherein responders were clearly separated from the other groups. Functionally, the differentially expressed genes at Week 12 were associated with key immune system functions including activation, proliferation and recruitment in addition to metabolic and ion transport pathways.



No Emergent Trends in AE Profiles in UC Patients Relative to Placebo

	Placebo	Omilancor 440 mg	Omilancor 880 mg
Subjects reporting ≥ 1 AE – no. (%)	20 (30.3%)	18 (27.3%)	20 (30.3%)
Total AEs – possibly related or higher	10	16	11
Total AEs – definitely related	0	0	0
Infections and Infestations	5 (7.6%)	4 (6.1%)	5 (7.6%)
Lymphopenia	1 (1.5%)	0 (0%)	0 (0%)
AEs experienced in ≥ 5% of subjects			
<i>Ulcerative colitis worsening</i>	5 (7.6%)	7 (10.6%)	7 (10.6%)

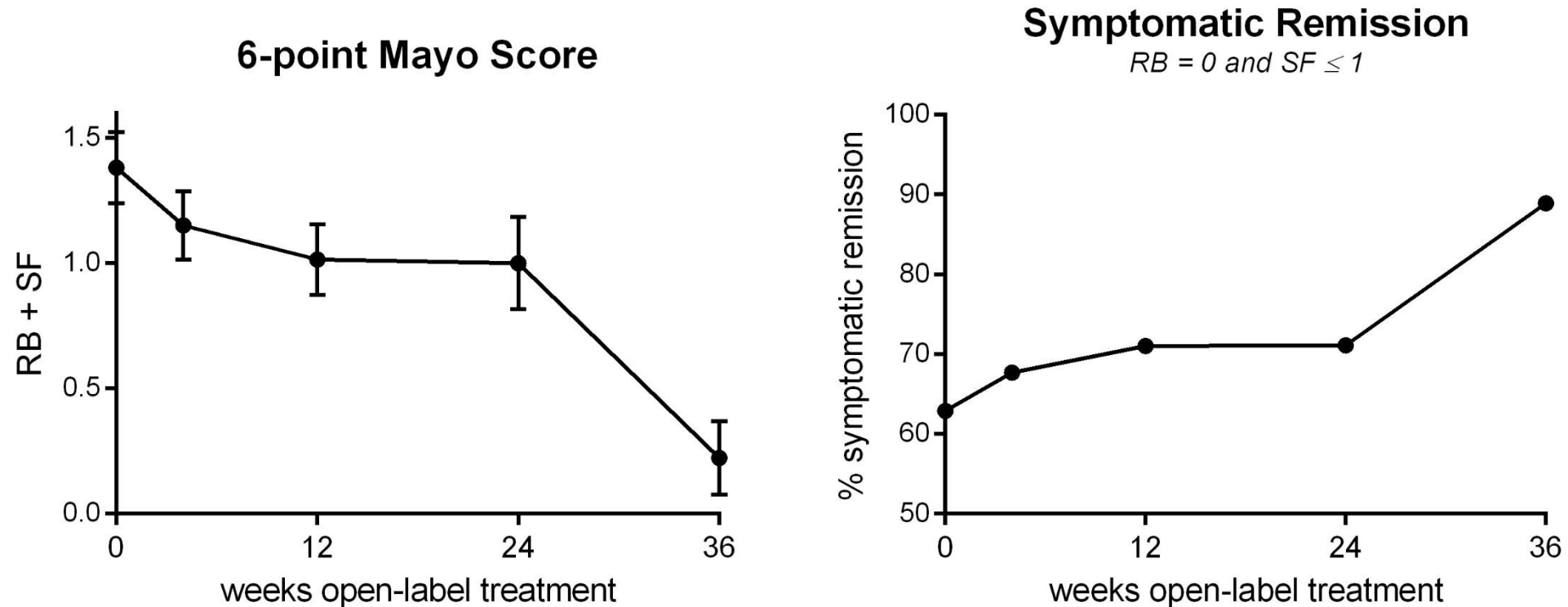
4 SAEs were reported during the induction phase. All were judged to be not related to study treatment:

- Worsening of UC (2)
- Calcaneus fracture
- Amoebiasis

Placebo-like safety and tolerability profile with no inhibition of the JAK pathway and no systemic side effects



Patients Treated with Omilancor Maintain low Mayo Scores and UC Symptoms beyond 1 year of Treatment



- Nearly 90% of patients achieving remission thresholds in stool frequency and rectal bleeding after 36 weeks of open-label treatment.
- Clinical remission (based on 3-component Mayo) was observed in 36.1% ($\Delta = 16.7\%$) of the omilancor 880 mg group and 35.5% ($\Delta = 16.1\%$) of the omilancor 440 mg group during the blinded maintenance phase.



Omilancor Phase 3 Trial Design offers flexibility for NDA

Omilancor is uniquely positioned to address the full range of disease severity in UC with the capacity to pursue labels in either moderate-to-severe or mild-to-moderate UC.

- In Phase 2, the efficacy of omilancor treatment did not lessen at higher disease severity or previous biologic use status

Moderate-to-Severe UC

- Definition: Modified Mayo Score of 5 to 9 with endoscopic subscore of at least 2, with balanced representation of biologic naïve and failure patients
- Phase 3 design: >90% of randomized patients expected in the 5 to 9 range, 40% biologic failure. The trial is sufficiently powered to provide statistical significance in this subpopulation.

Mild-to-Moderate UC

- Definition: Modified Mayo Score of at least 4 with endoscopic subscore of at least 2 and rectal bleeding of at least 1.
- Phase 3 design: Modified Mayo Score of at least 4 with endoscopic subscore of at least 2 and rectal bleeding of at least 1. 100% of patients

The recent approval of etrasimod sets a precedent for approval in moderate-to-severe UC from a subset of patients in an active UC trial.



Clinical Plans for Omilancor in UC

Positive Outcome from End-of-Phase 2 meeting with FDA

- Gained FDA agreement on an approvable population of active UC patients for the upcoming Phase 3 program
- Analysis of Phase 2 data using Phase 3 population demonstrated statistically significant approvable primary endpoint for clinical remission and correlated with changes in predictive biomarkers of response to treatment such as FCP as early as 2 weeks from dosing

Phase 3 Design

- Total of 1,378 patients with active UC across two trials
- Trials will evaluate one dose (440 mg) versus placebo
- Primary endpoints include:
 - Clinical remission at Week 12
 - Clinical remission at Week 52
- Mucosal healing rate at Week 12 defined by endoscopic subscore of 0 or 1 with Geboes histologic index < 3.1

✓ Initial Phase 2 data announced

✓ Provided follow-up Phase 2 data

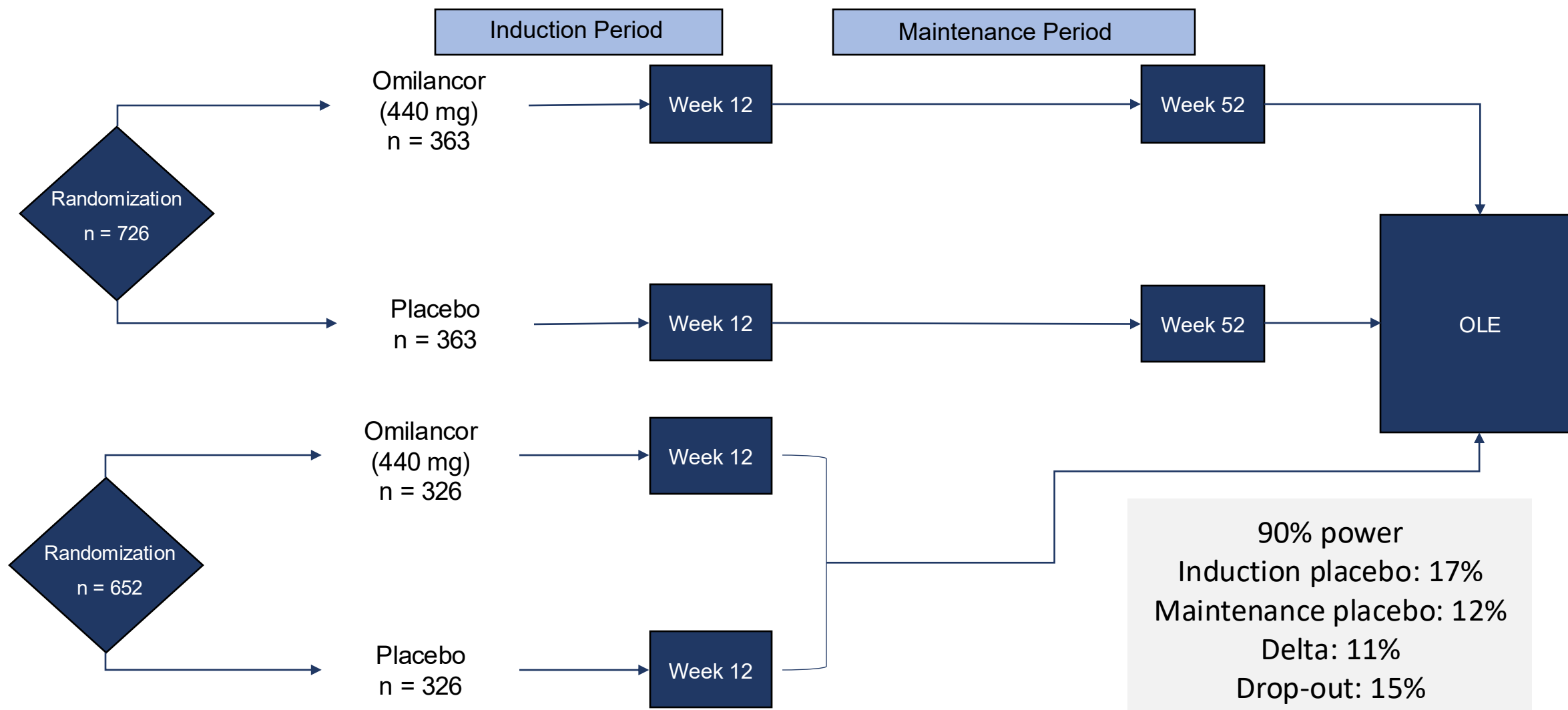
✓ EoP2 meeting with FDA

Initiate registrational Phase 3 trial



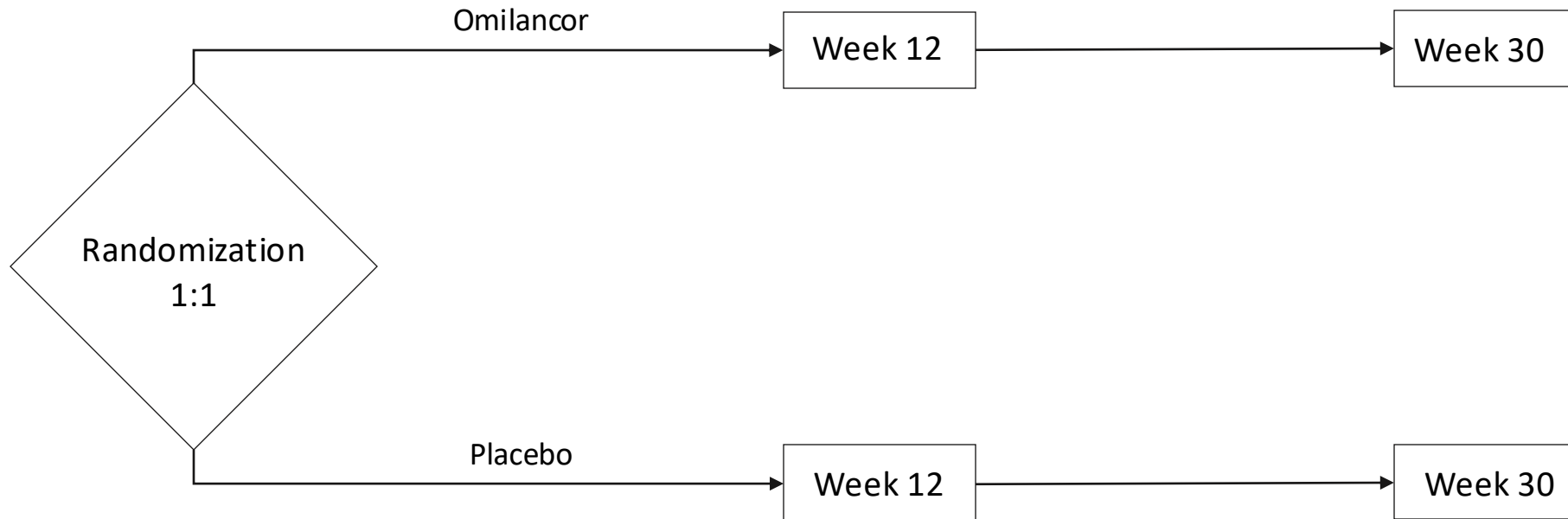
Phase 3 Pivotal Study Design of Omilancor in UC

Aim to enroll a total of 1,378 patients





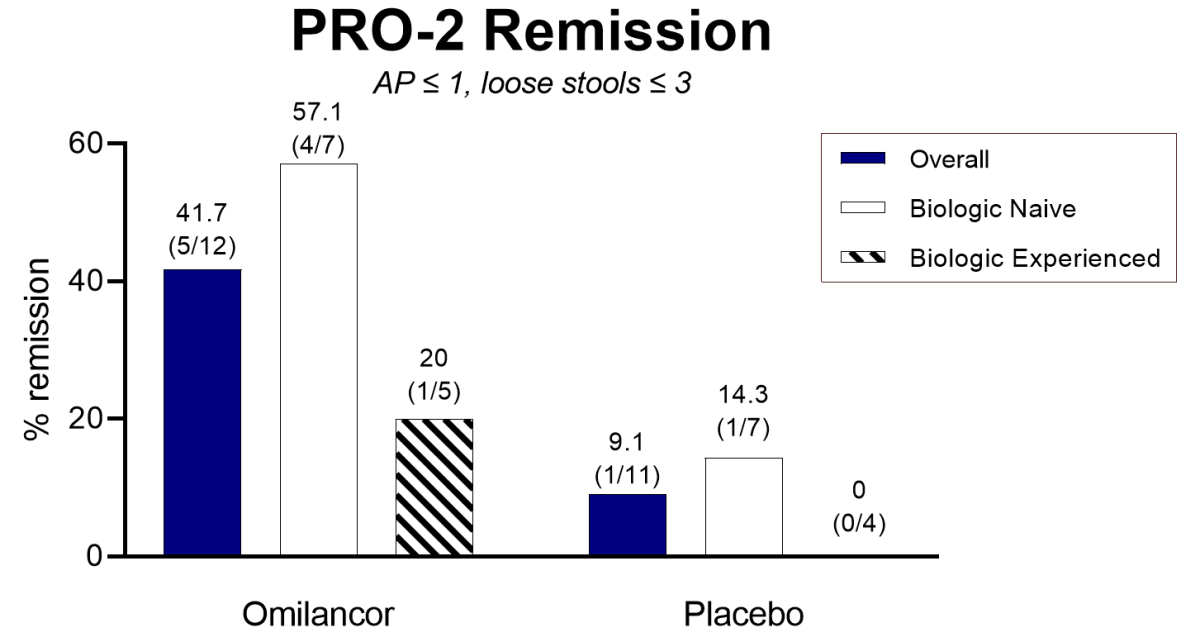
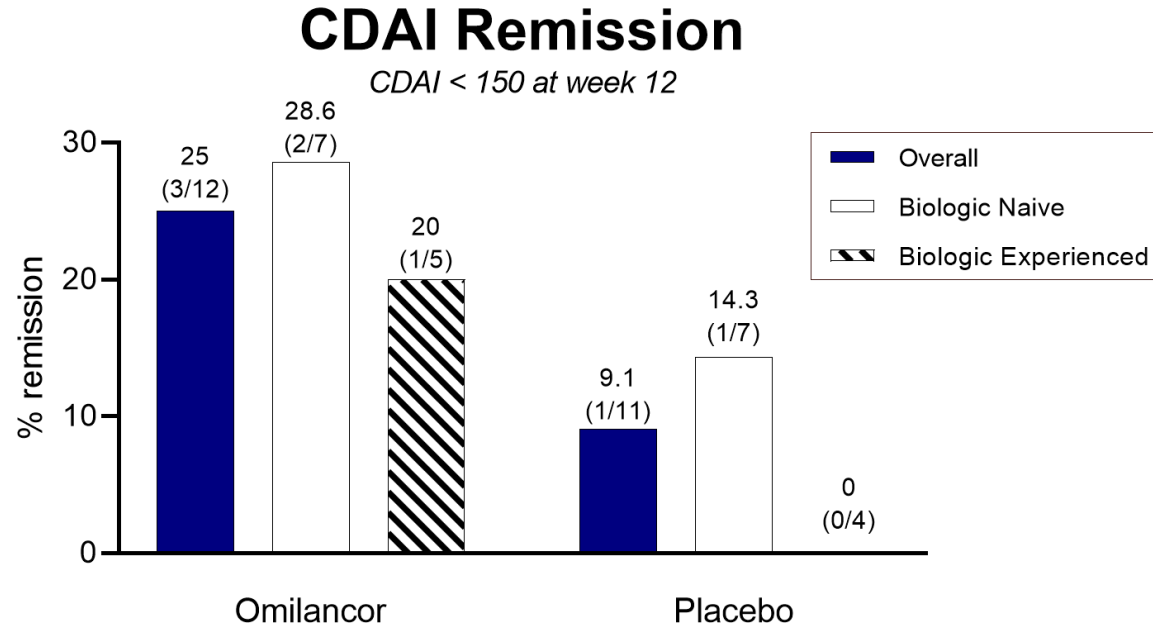
First-in-Indication Study of Omilancor in Moderate to Severe Crohn's Disease



- Primary Objective
- The primary objective of this proof-of-concept study was to establish the efficacy of oral BT-11 in inducing clinical remission at Week 12 in subjects with moderate to severe Crohn's disease (CD).
- Key Inclusion Criteria
- Male and female subjects with moderate to severe CD defined by a CDAI between 220 and 450 with SES ≥ 6 (≥ 4 for isolated ileitis); 5-aminosalicylates (max 4.8 g/day) and oral corticosteroids (max 20 mg/day prednisone or equivalent) must be stable for the 12-week induction period.



Promising signal in primary endpoint in both biologic naïve and exposed patients



Population includes all subjects that had received study drug and met eligibility criteria

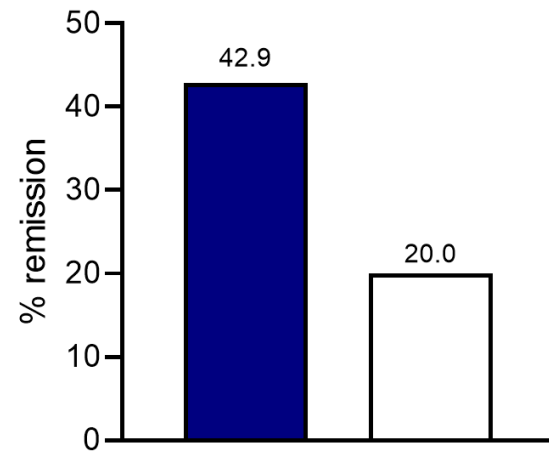
Based on a $\Delta = 15.9$ and placebo rate of 9.1, the needed sample size to observe a significance was 68.



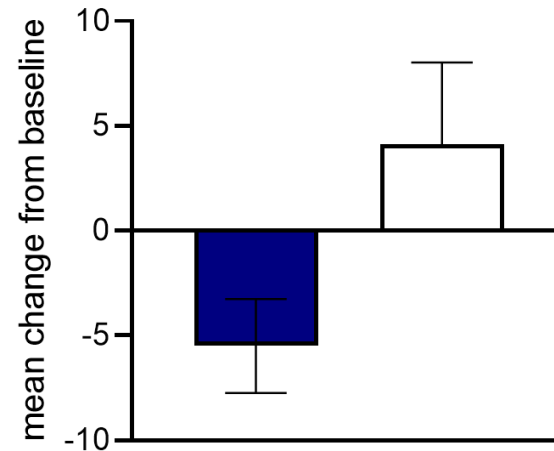
Omilancor treatment increases likelihood of histological remission at Week 12

Histological Remission

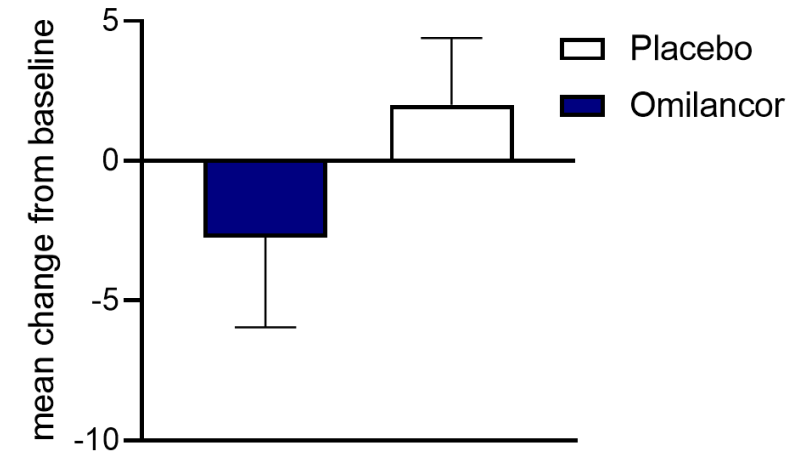
continuous Geboes < 12



Ileum Geboes Score



Rectum Geboes Score

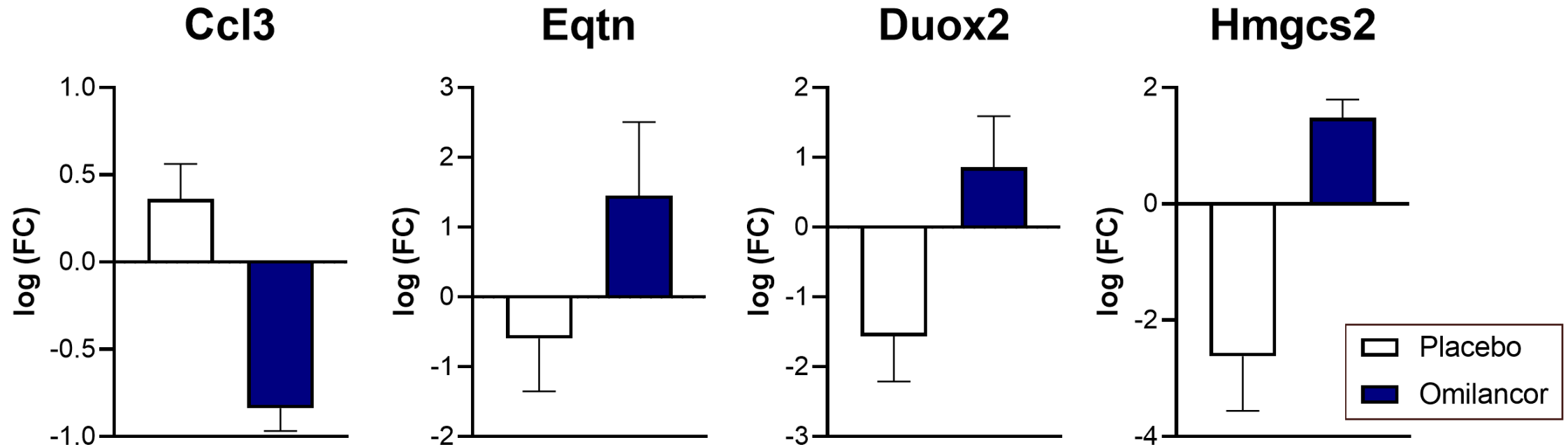


Population includes all subjects that had received study drug and met eligibility criteria

In patients with histological activity (cGeboes > 12) in at least one segment at baseline, omilancor induces remission in all segments in 42.9% of patients relative to 20.0% in the placebo group. Omilancor treatment provided a mean decrease in Geboes score in both the ileum and rectum after 12 weeks.



Omilancor treatment increases LANCL2-linked genes in ileal biopsies



Population includes all subjects that had received study drug and met eligibility criteria

Omilancor treatment resulted in reduction of multiple cytokines in the ileum relative to baseline, including Ccl3. Notable genes upregulated by omilancor treatment including those associated with endosomal, metabolic, and antimicrobial defense pathways.



Combination therapy in IBD

- Approximately **5-10%** of patients remain refractory to all available treatment classes.
- Time to flare is estimated to be **6 months** even after achievement of clinical remission in moderate to severe patients, often leading to repeated steroid regimens or change of primary therapy.
- Efficacy for induction of clinical remission has plateaued at around **one-third** of patients across multiple classes (JAK inhibitors, S1P antagonists, anti-TL1a biologics).
- Evaluation of combination therapy has lagged behind the recent innovation in the IBD therapeutic market:
 - Anti-TNFs with azathioprine/6-MPs evaluated pre-2010
 - Anti-TNF and anti-IL23 evaluated in 2023
 - Sporadic case studies
- Overall use of combination therapy remains low (9% in UC, 21% in CD) with the primary combination being anti-TNF with azathioprine or similar immunosuppressant.

Omilancor offers an ideal mechanistic, safety and DDI profile to serve as a backbone combination therapy with other small molecules or biologics.

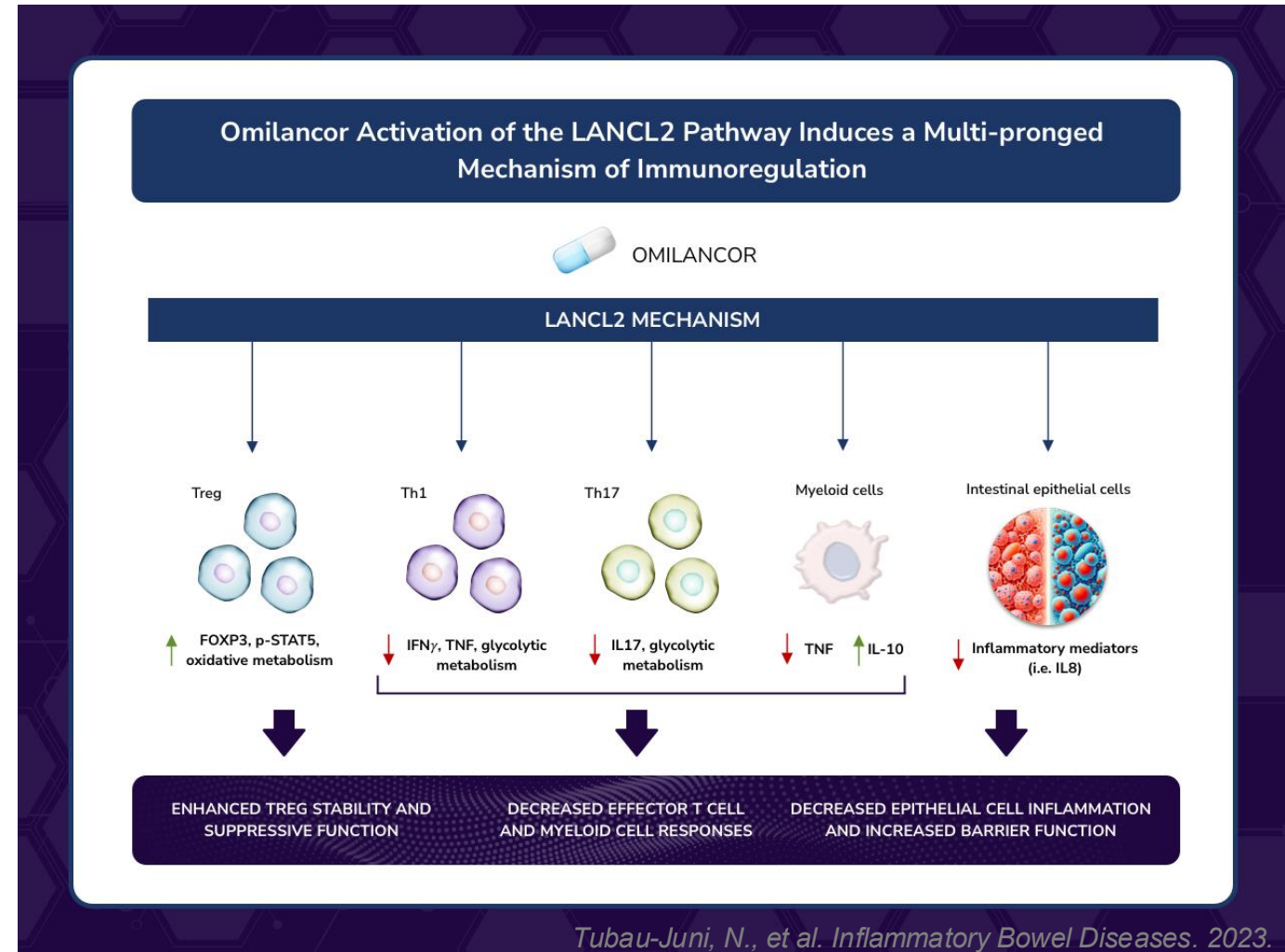


LANCL2 Activation is a Clinically Validated Novel Mechanism of Action

Most therapies marketed or in development for IBD have immunosuppressive MoAs directly inhibiting receptors, kinases, or cytokines tied to inflammation. In contrast, omilancor:

- Supports the differentiation and functionality of Tregs.
- Improves tolerogenic responses in macrophages and other phagocytes.
- Restores barrier function to the intestinal epithelial layer.
- Induces clinical remission in severe patients with highly dysregulated immunometabolic profiles

Omilancor can expand the addressable target population and can enhance mechanisms tied to long-term maintenance of clinical remission to prevent therapy switching.





Omilancor has a low risk for negative DDI

Omilancor has limited systemic distribution with observed C_{max} of 372 ng/mL and average C_{max} of 140 ng/mL in Phase 2 UC patients and no target organ class toxicities, limiting the risk for amplification of systemic side effects. Chronic omilancor dosing does not result in immunogenicity. Metabolism, transport, and ADR profiles provide a strong potential for combination

Metabolism

- NADPH intrinsic clearance of 42.1 $\mu\text{L}/\text{min}/\text{mg}$ in microsomes, 4.9 $\mu\text{L}/\text{min}/10^6$ cells in hepatocytes
- Primary metabolizing enzyme was CYP3A4 with half-life of 71.6 min

CYP Inhibition and induction

- No inhibition of CYP2B6, CYP2C8, CYP2C9, CYP2C19, or CYP2D6.
- No induction of common isoforms.
- Inhibition of CYP1A2 at an IC₅₀ of 5.75 $\mu\text{g}/\text{mL}$ (12.6% inhibition at physiologically relevant concentration) and CYP3A4 at an IC₅₀ of 4.53 $\mu\text{g}/\text{mL}$ (14.7% inhibition at physiologically relevant concentration).

Transporters

- Not a substrate of any common transporter. Transport is observed to be passive.
- No inhibition of Oct2, ASBT, BCRP, MRP1, MRP2, MRP3, NTCP, OAT1, OAT3, OCT1, or P-gp.
- Inhibition of OATP1B1 at IC₅₀ of 1.56 $\mu\text{g}/\text{mL}$ (<1% inhibition at physiologically relevant concentration) and OATP1B3 at 0.234 $\mu\text{g}/\text{mL}$ (44.6% inhibition at physiologically relevant concentration).

ADR

- No inhibition of 44 common ADR receptors, ion channels, enzymes and transporters at physiologically relevant concentrations

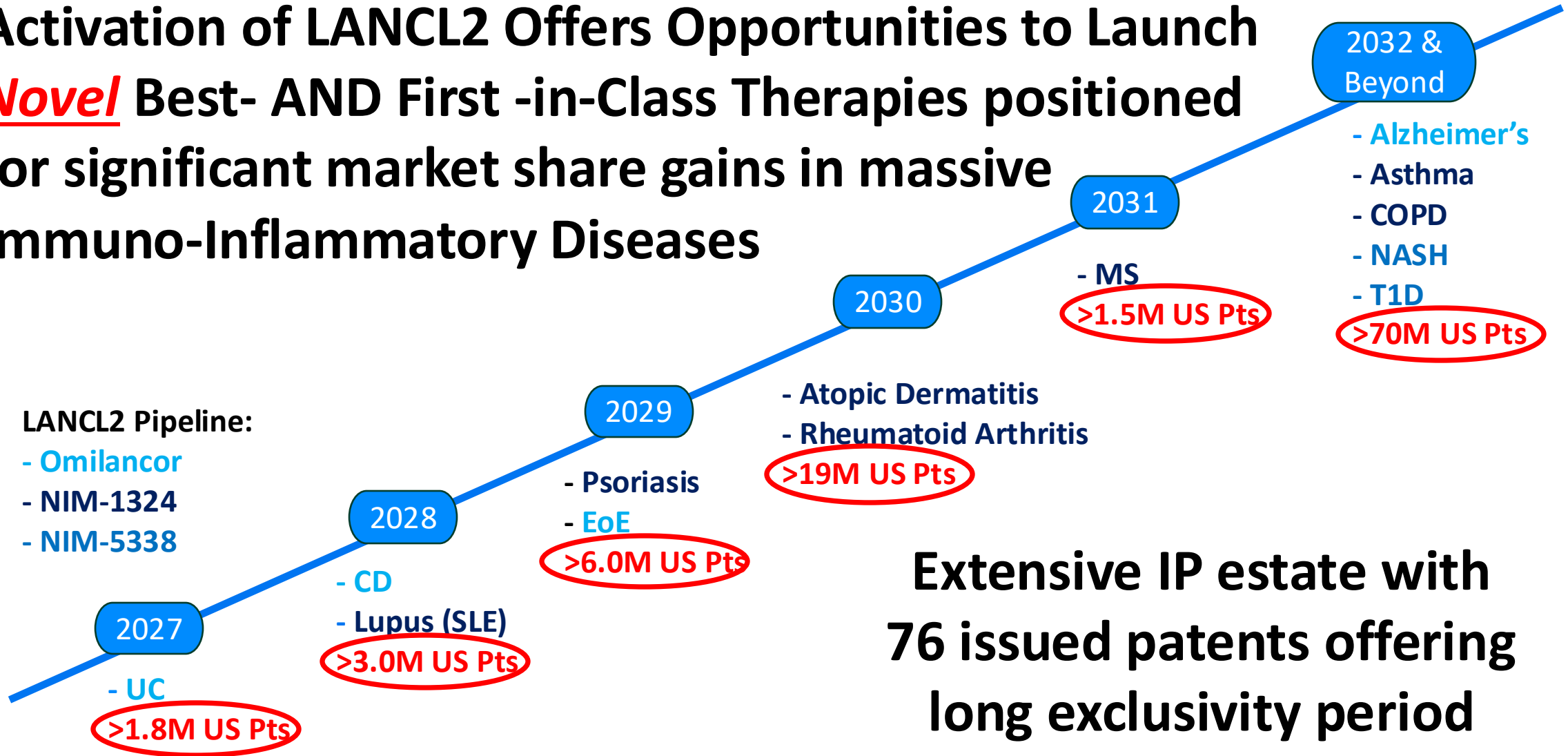


Oral Omilancor is an immediate release tablet

- Current tablet size for IBD development is 440 mg.
 - Tablets of 440 mg to 880 mg strength have been used across nonclinical and clinical development
 - The current 440 mg strength is equivalent to that used in Phase 2 (named 500 mg at the time). The difference is only a change in naming convention of base weight versus salt weight.
- All excipients in the IR tablet are sourced from the inactive ingredients database and within the listed daily amounts.
 - Note: omilancor is compatible with all excipients used in the current formulation.
- The IR tablets are film coated for consistent appearance and do not use TiO_2 .
- No scaling concerns have been noted during scale-up.
- All analytical methods, including discriminatory dissolution, have been developed and validated.
- Registrational batches on the 200 – 300 kg scale are currently in progress.



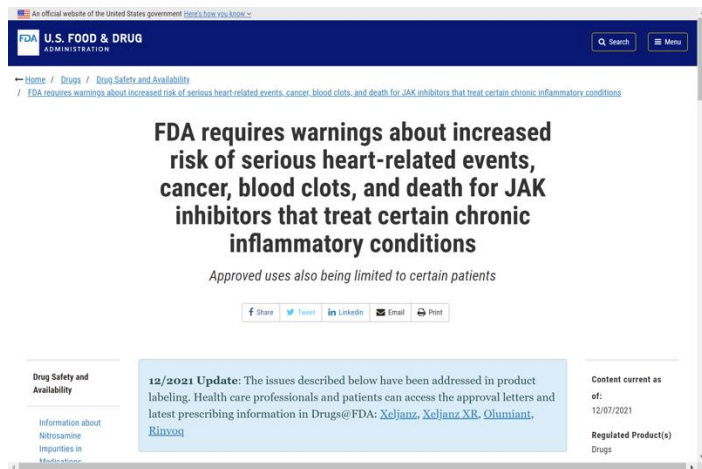
Activation of LANCL2 Offers Opportunities to Launch **Novel** Best- AND First -in-Class Therapies positioned for significant market share gains in massive Immuno-Inflammatory Diseases





Ripe Opportunity for NImmune/Omilancor

Black Box Warnings on Existing Therapies



And yet . . .



ABBV Rinvoq FY'23 US
Rev. \$4.0 Billion



PFE Xeljanz FY'23 US
Rev. \$1.7 Billion

Shows the
high unmet
need

Failures abound . . . (sample of headlines)

June 3, 2022 11:01 AM EDT | KGW

**Immunix shares cut in half as biotech
dumps ulcerative colitis after PhII fail**

Connect Seeks Partners to Advance
UC Asset after Phase II Fail

Published: May 04, 2022 | By Vanessa

**Phase 2 deja vu for
gossamer bio after
ulcerative colitis med flops**

By Max Bayer • Apr 25, 2022 08:23am



**Omilancor has succeeded where most have failed,
offering a first and best in class oral, 1/day therapy**



Summary and Conclusions



Nimmune is Phase 3 science-driven company well-positioned to develop a franchise of new first-in-class immunoregulatory therapeutics for GI and rheumatology.



Innovative Precision Immunology-focused drug discovery and development platform yielding novel targets, biomarkers, therapeutics and biomarker-driven drug development.



Extensive animal pharmacology, mechanism of action, toxicology, benign safety profile and clinical data on lead candidates (Phase 3 omilancor in UC; Phase 2-ready NIM-1324 for lupus and IBD).



First NDA for omilancor in UC planned for 2027 with multiple I&I indications thereafter.



Experienced leadership team with immunology and public/private biotech company experience with a successful track record of operational excellence and demonstrated ability to execute.



Track record of significant shareholder value capture and accretion.