CONFIDENTIAL

NIMMUNE BIOPHARMA

COMPANY PRESENTATION | March 2025

Precision Immunology Therapeutics for Human Diseases

Best-in-Class, Biomarker-Driven Therapeutics

- Private late-stage precision I&I biopharmaceutical company developing first-in-class therapeutics.
- 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.

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; completed Phase 1 testing.

Highly Experienced Biotech Team

- Experienced team that led one of the best performing NASDAQ listed biopharma, 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 immunology.
- Developed a 17-product pipeline and cleared 8 INDs in <4 years.

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.

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.

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.

Jennifer Collette, MSA, CPA

Chief Accounting Officer & Controller

15 years of experience in accounting and financial systems. Expertise in technical & project accounting, budget planning, controls, and financial reporting. Former Head of Finance 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.

“Bet on the Jockey . . . “ Proven Winning Team

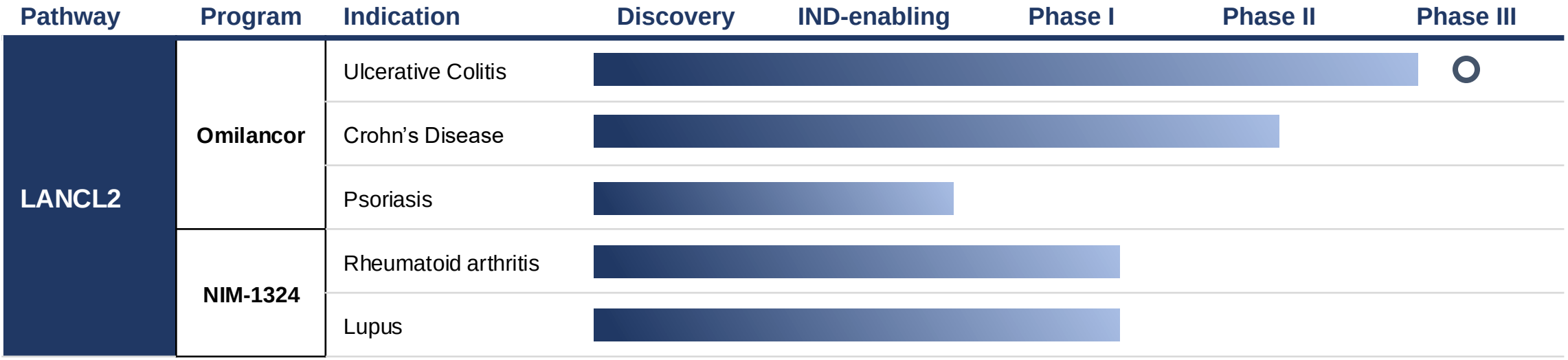
AbbVie to bolster immunity illness drug pipeline with Landos deal

By **Reuters**

March 25, 2024 8:16 AM PDT · Updated 2 days ago

- NImmune team founded Landos, invented and developed all LABP programs.
- TITAN-X, the computational drug discovery platform that discovered and guided development of all LABP programs was invented and is exclusively controlled by NImmune team and discovered NImmune’s LANCL2 portfolio, including omilancor and guides omilancor’s development.
- Acquisition of LABP by ABBV validates TITAN-X and the NImmune team as the “right jockey.”
- Same proven team, same proven platform, same proven therapeutic focus.
- **Proven winning team to build and capture transformative value from a portfolio of first and best in class I&I therapeutics.**

Active Pipeline: Inflammation & Immunology Drug Development Portfolio



Omilancor and the entire LANCL 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 potential with unrivaled safety

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
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
	Omvoh (mirikzumab)	LLY	10/27/23 approved	Mod-to-Sev UC	24.2%	**9.1%**	LUCENT 1 & 2	Infection, arthralgia, rash, headache	IV/SC q2mo
TYK2	Deucravacitinib (sotyktu)	BMY	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)	BMY	$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 and the LANCL2 Pathway in IBD

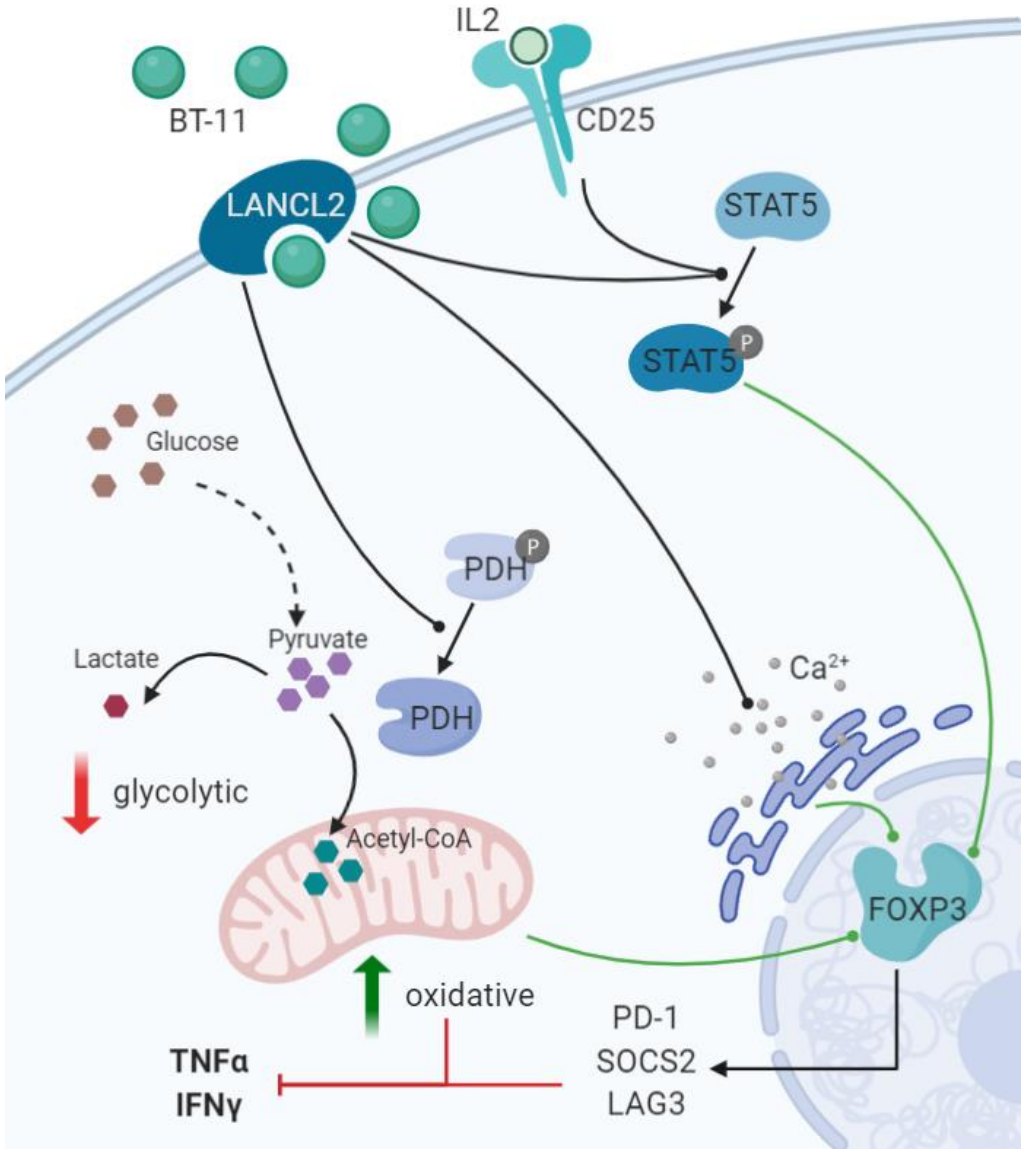
Omilancor Activates Novel Immunoregulatory Target LANCL2

Lanthionine Synthetase C-Like 2 (LANCL2):

- Multipronged mechanism of action targeting known immunological targets downstream tied to autoimmune diseases, including IBD

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

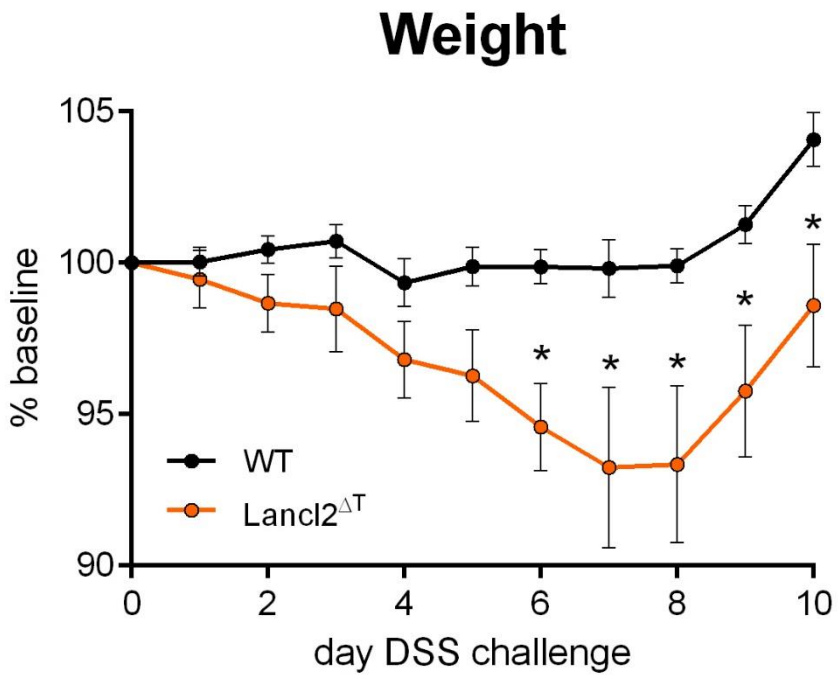
- ✓ Enhances CD25/STAT5 signaling to support the stable differentiation of regulatory CD4+ T cells with greater anti-inflammatory functionality
- ✓ Increases PDH activity, resulting in increased oxidative metabolism supporting FOXP3 stability
- ✓ Increases suppressive effects of Tregs due to enhanced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



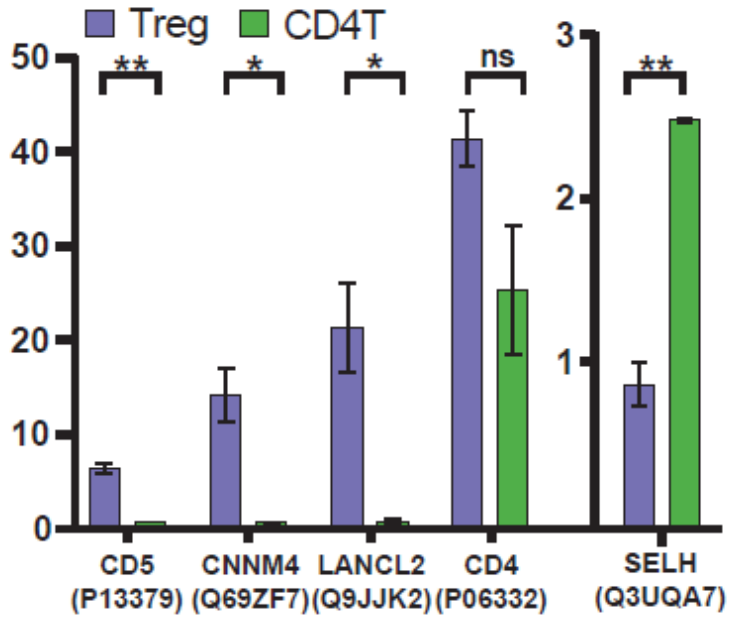
Leber, A., et al. Inflammatory Bowel Diseases. 2018 24:1978-1991.
Carbo, A., et al. J Med Chem. 2016 Nov 23;59(22):10113-10126.
Leber, A., et al. J Immunol, 2019.

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*

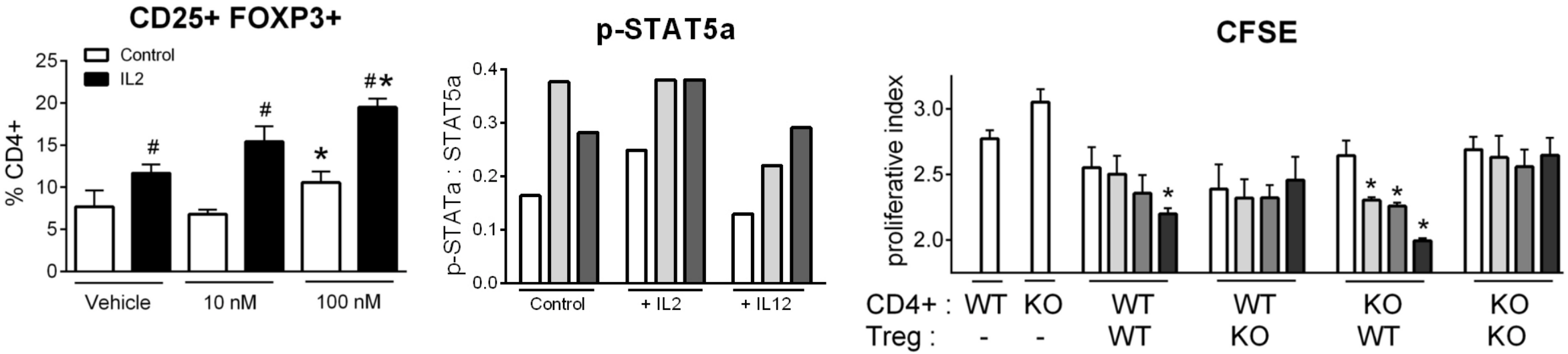


(Leber, 2018)



(Myers, 2019)

LANCL2 in Regulatory CD4+ T cells

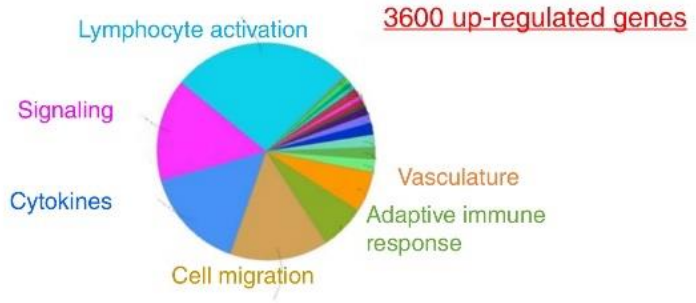


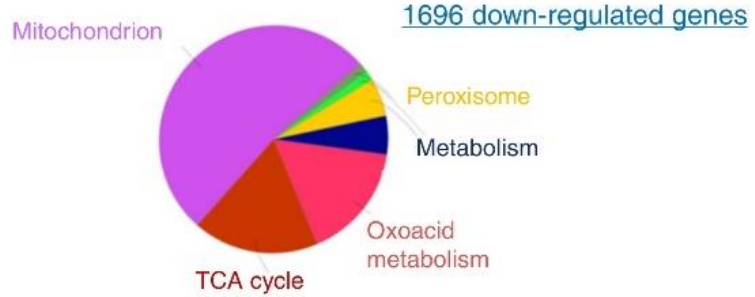
LANCL2 activation supports IL-2 induced CD25+ Treg differentiation and capacity to suppress proliferation

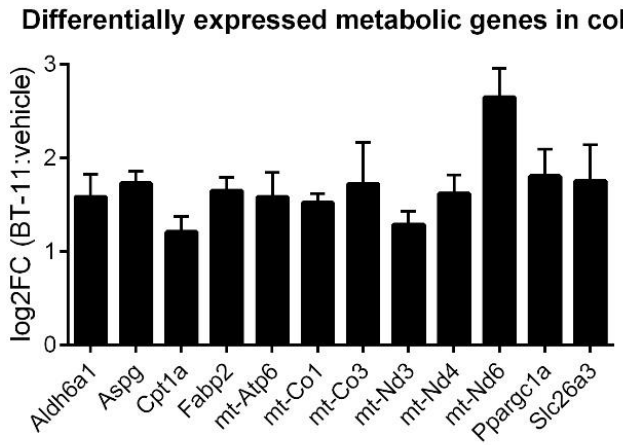
LANCL2 can serve as a target to reverse the diminished CD25+ expression in regulatory CD4+ T cells that occurs during autoimmune disease, rescue compromised IL-2 signaling and restore suppressive capacity

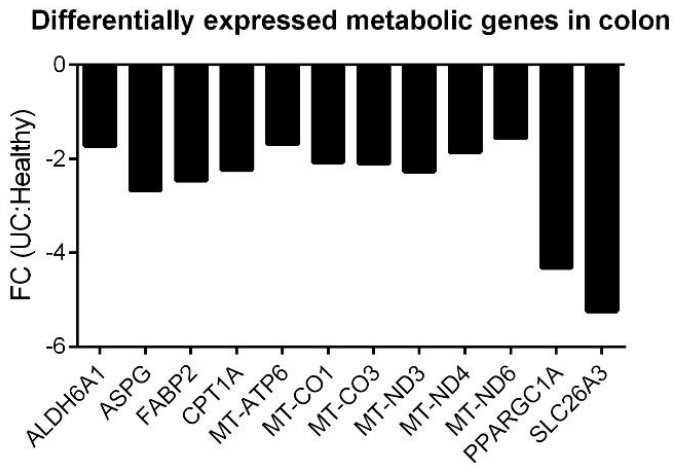
Immunometabolism in IBD

- 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

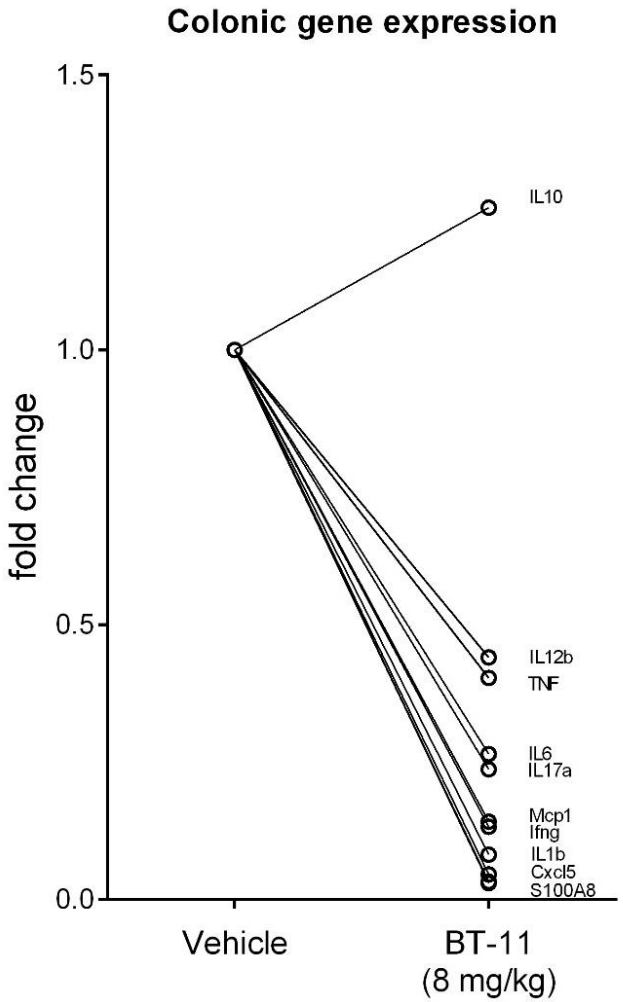


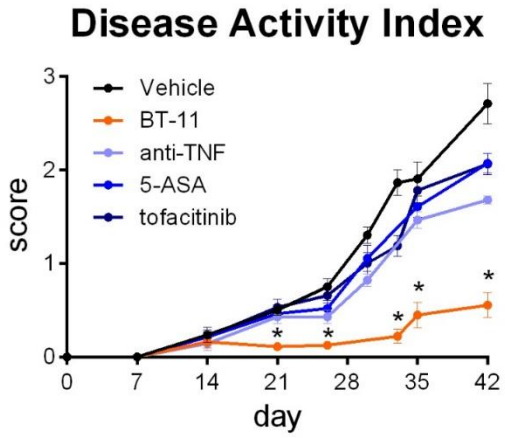


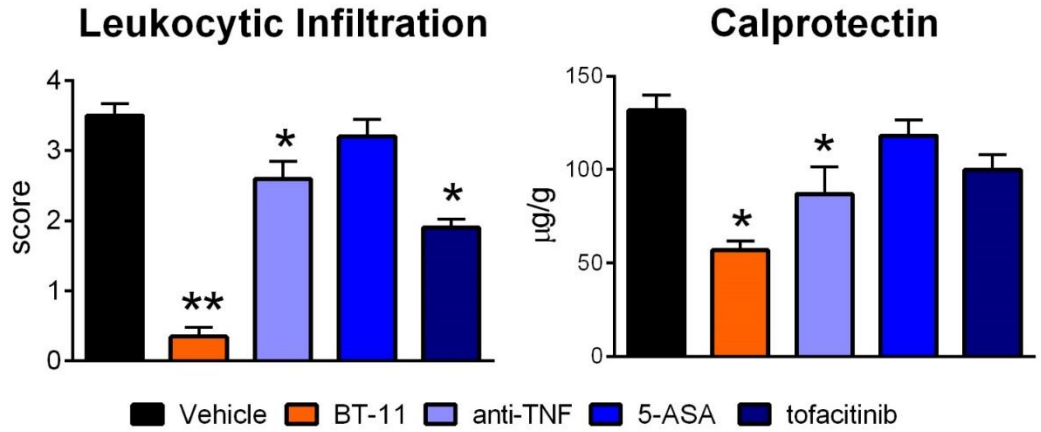




Preclinical Efficacy - Omilancor



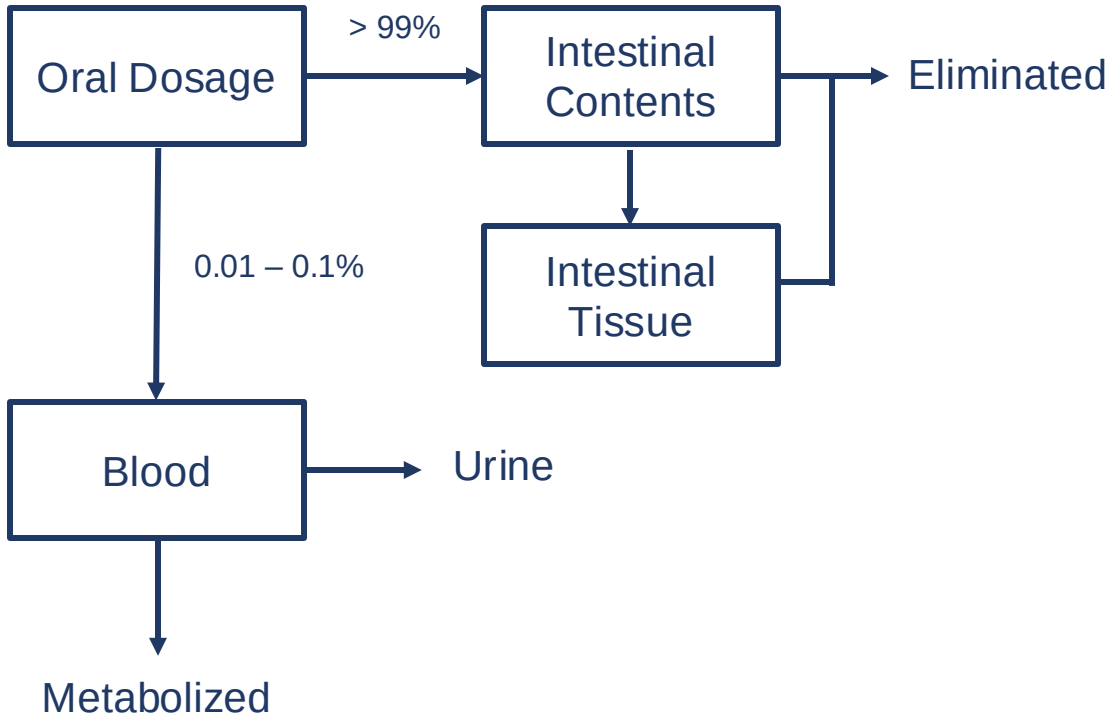




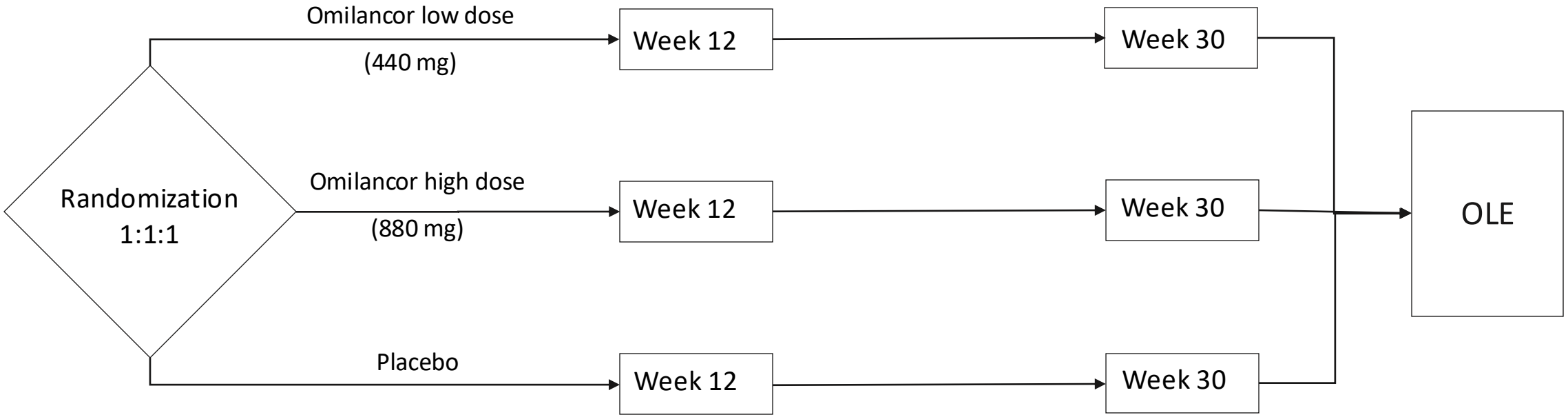
- ✓ Effective in 5 mouse models of IBD and a pig model of IBD
- ✓ Outperforms standard of care and recent approvals in UC including anti-TNF and JAK inhibitors
- ✓ Validated local PK/PD relationships in the colons of mice and pigs without the need for systemic exposure or specialized formulation
- ✓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

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



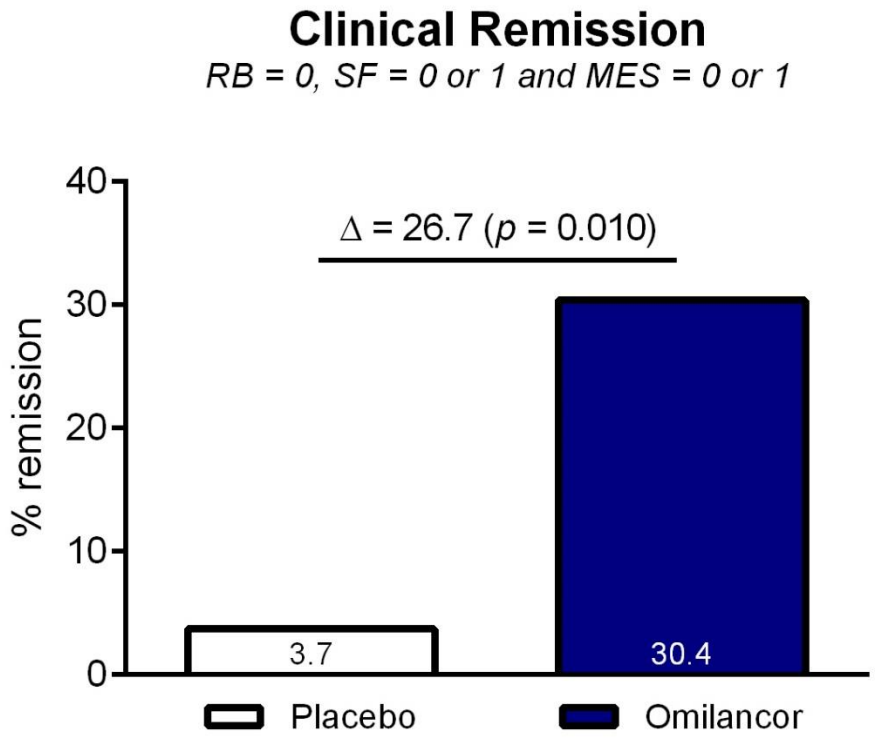
Completed First-in-Patient Study Design of Omilancor in UC



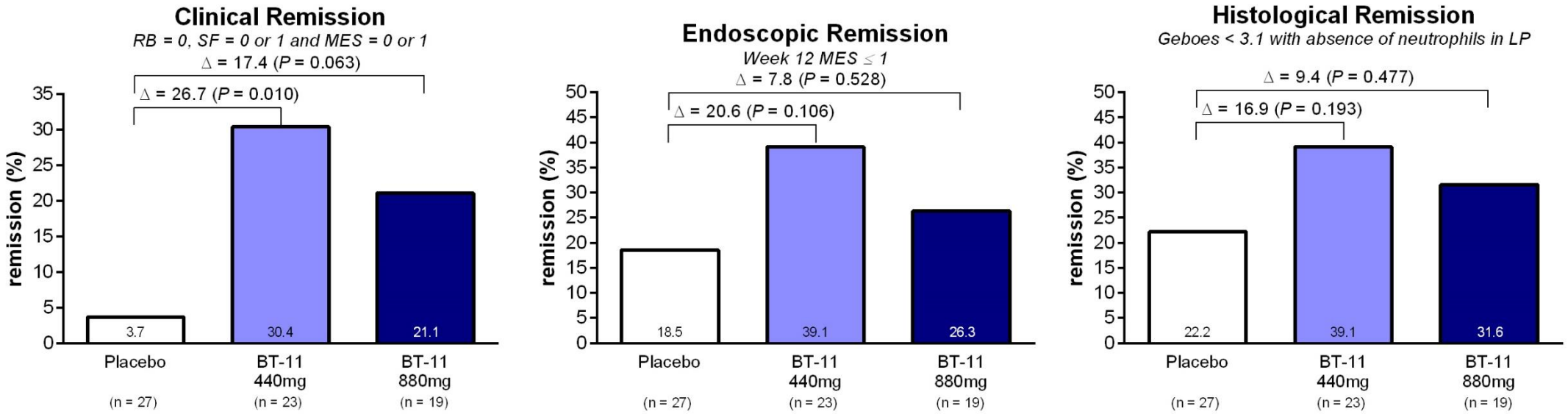
- Primary Objective
- The primary objective of this proof-of-concept study was to establish the efficacy of oral BT-11 in inducing clinical remission at Week 12 in subjects with ulcerative colitis (UC).
- Key Inclusion Criteria
- Male and female subjects with active UC defined by a total Mayo Score of ≥ 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



Statistically Significant Approvable Primary Endpoint



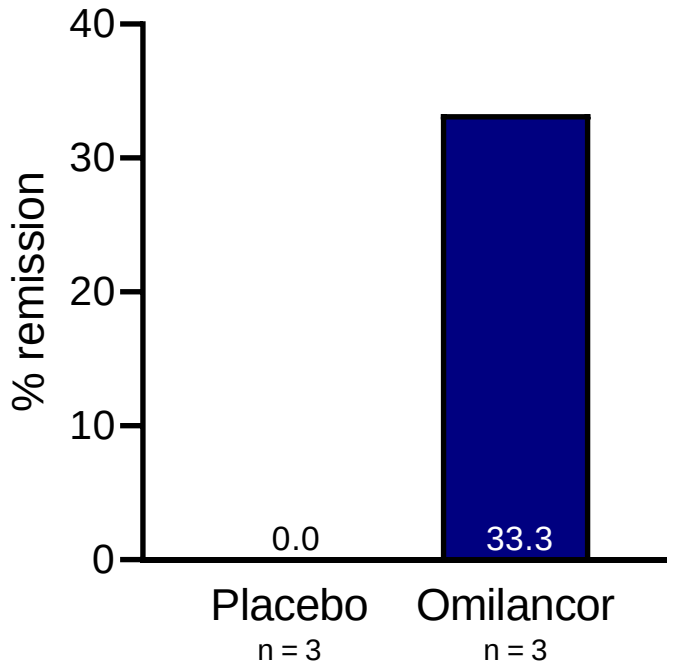
Population excludes patients with rectal bleeding = 0, Geboes histological score of 0, or fecal calprotectin < 250 ug/g at baseline

In patients with clinically, endoscopically and histologically active UC at baseline, 440 mg omilancor outperforms placebo for the induction of clinical remission.

Omilancor provides consistent induction responses in biologic experienced UC patients

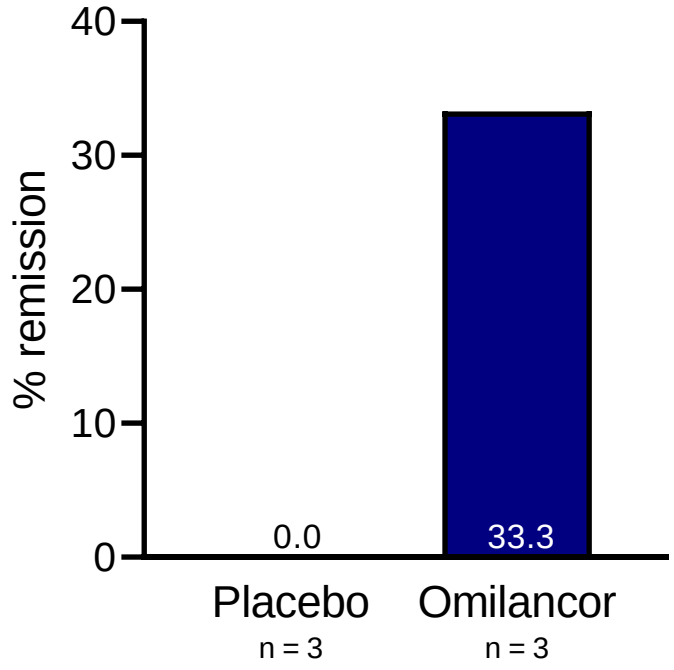
Clinical Remission

RB = 0, SF = 0 or 1 and MES = 0 or 1



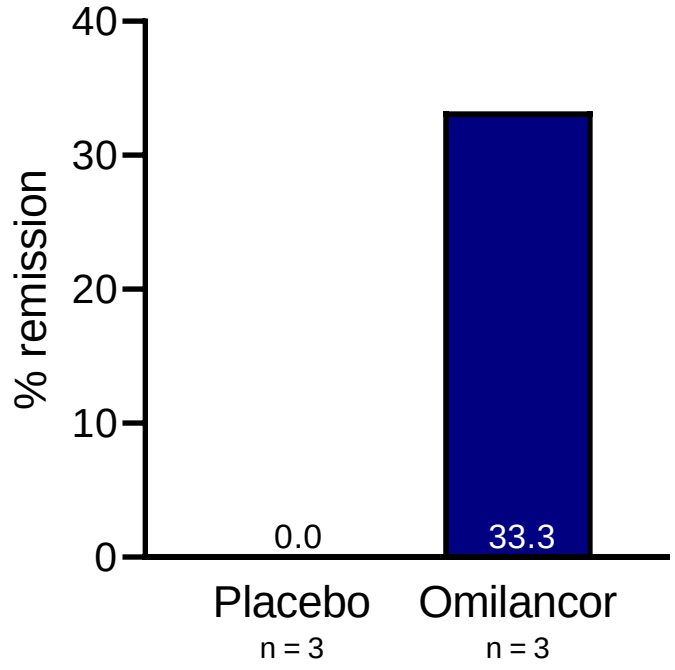
Endoscopic Remission

Week 12 MES ≤ 1

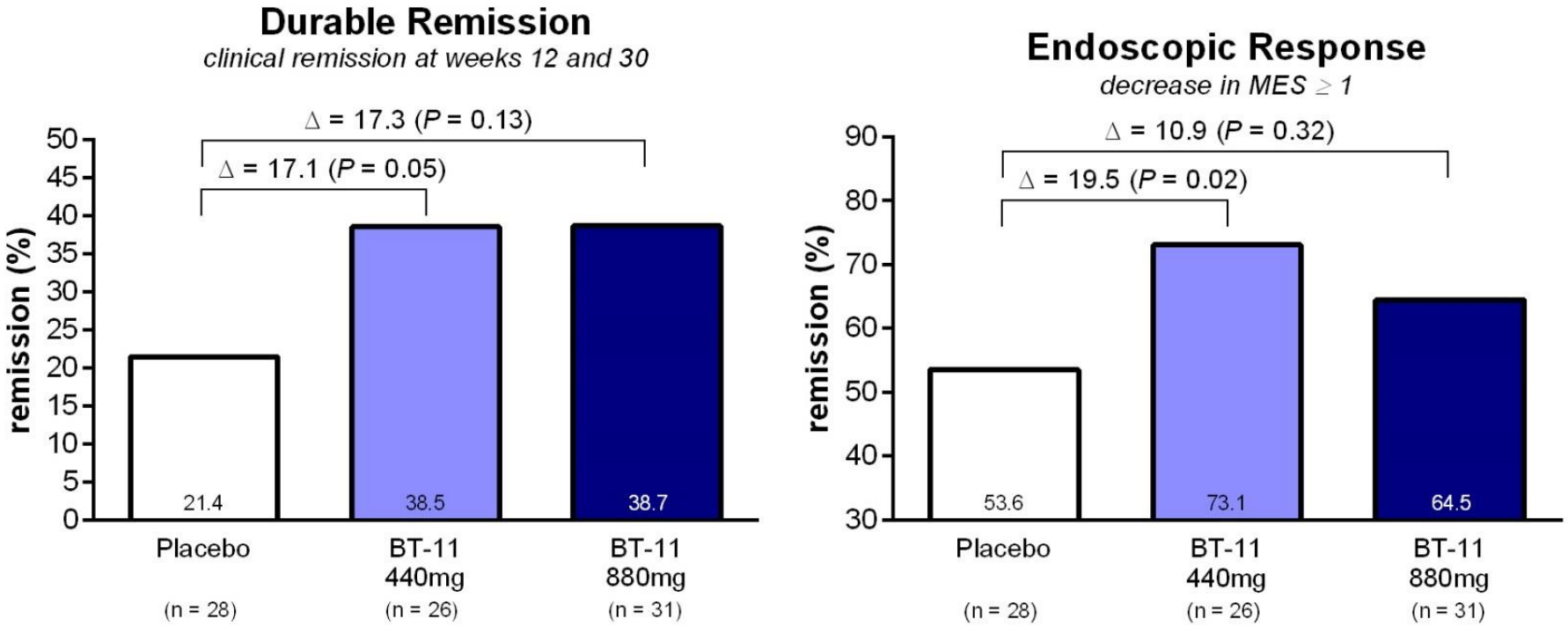


Histological Remission

continuous Geboes < 10



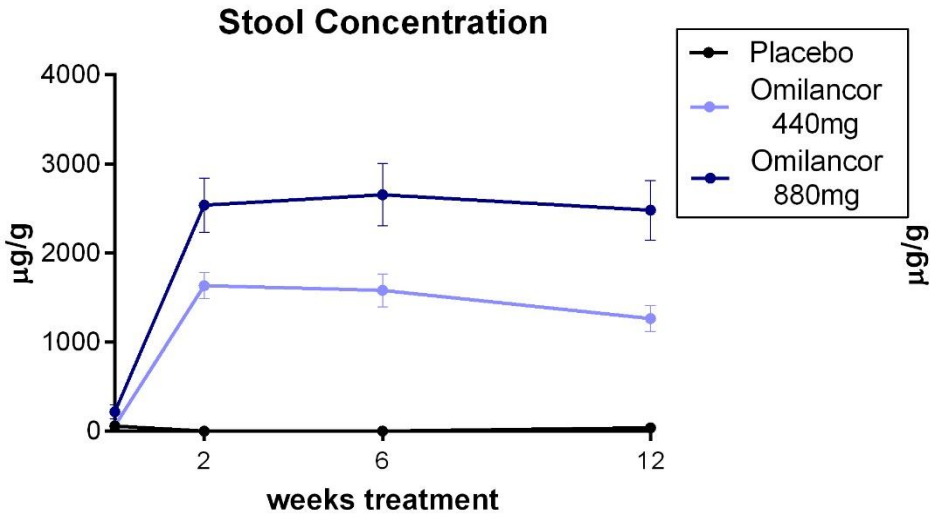
Clinical remission to omilancor is maintained through week 30

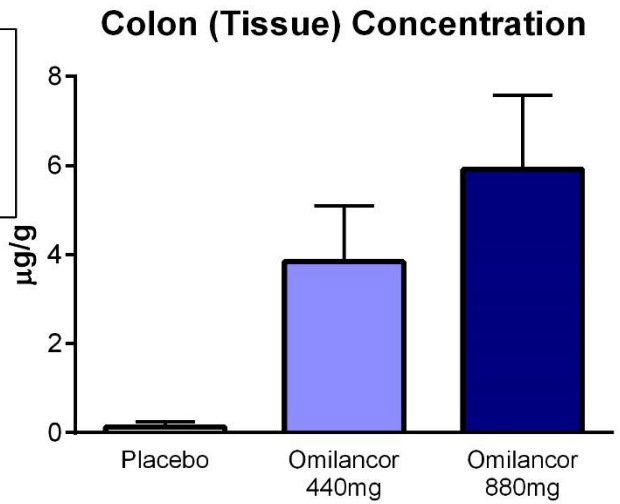


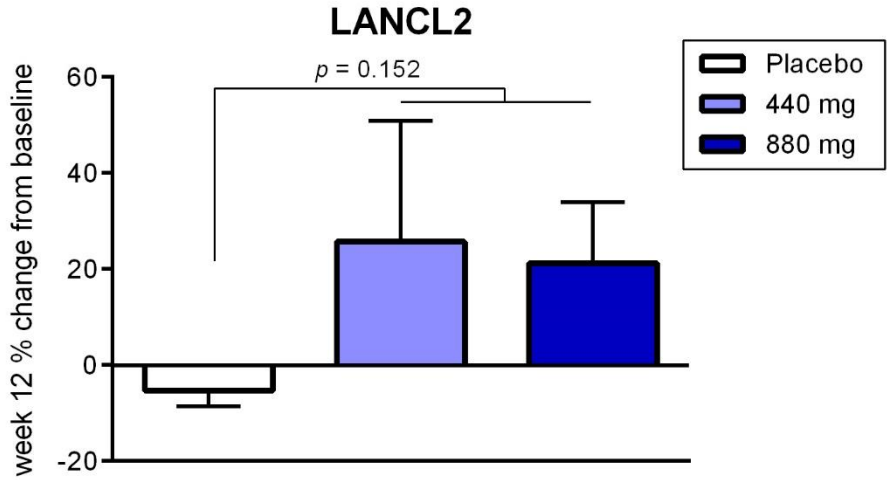
Population is the induction responder set, the primary analysis set for the maintenance period

Responses observed endoscopically and clinically with omilancor are maintained after 30 weeks of treatment.

PK/PD results validate sufficient target engagement at both doses

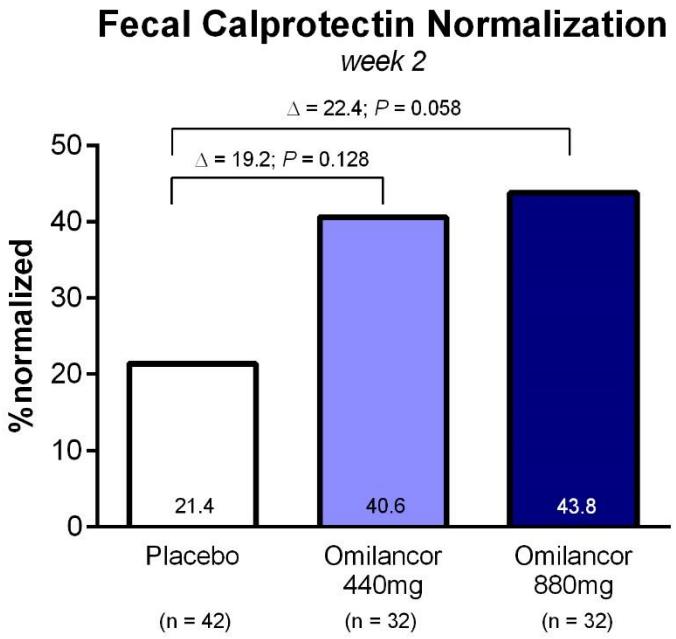


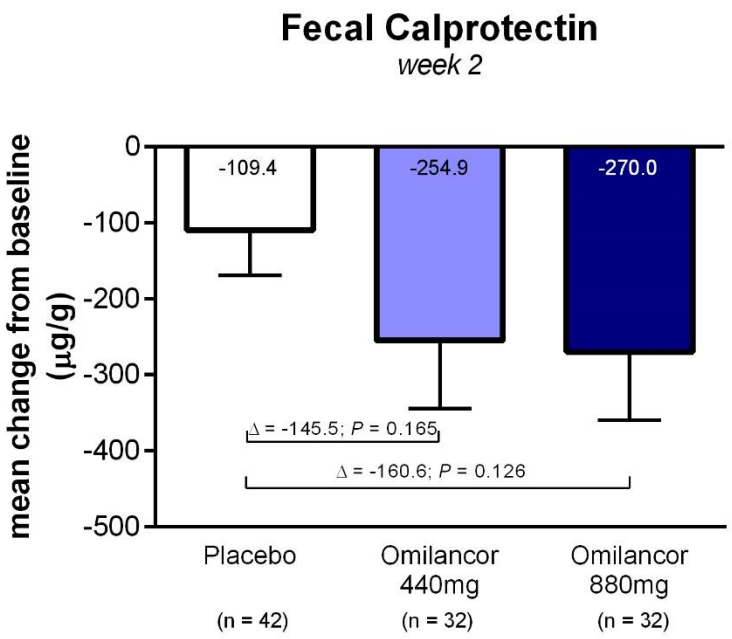




- Omilancor stool concentrations stable between 2 and 12 weeks of dosing
- No significant difference in stool concentrations between UC patients after 12 weeks and healthy volunteers after 7 days
- Stool and tissue concentration scale in a near dose-proportional manner
- 440 and 880 mg doses effectively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

Biomarker Response After 2 Weeks of Treatment

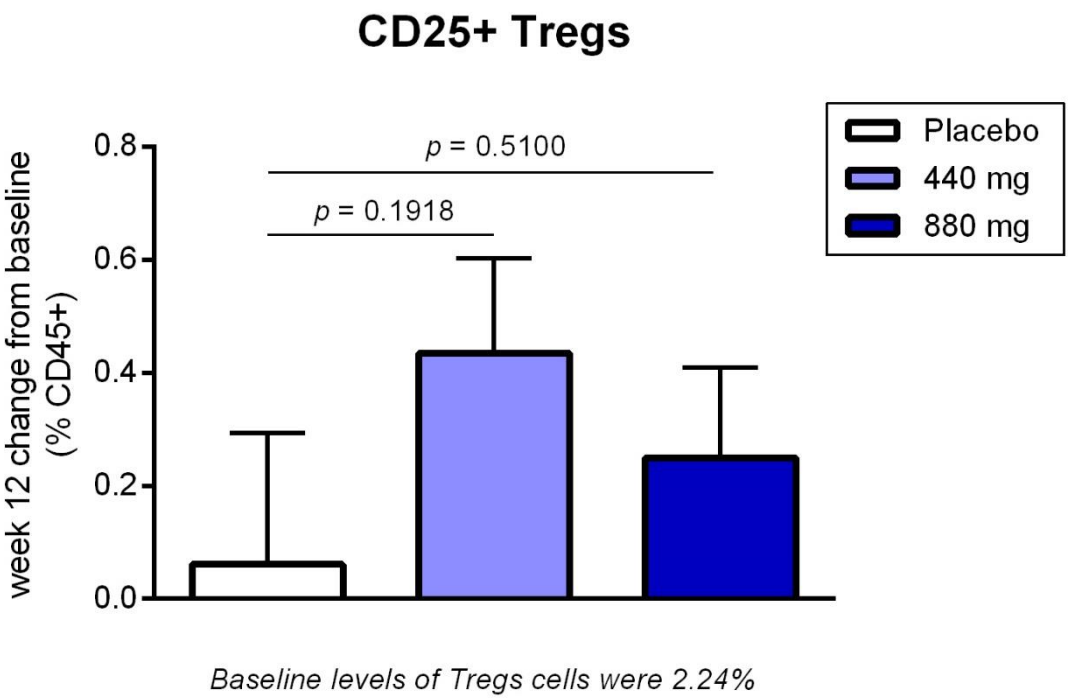




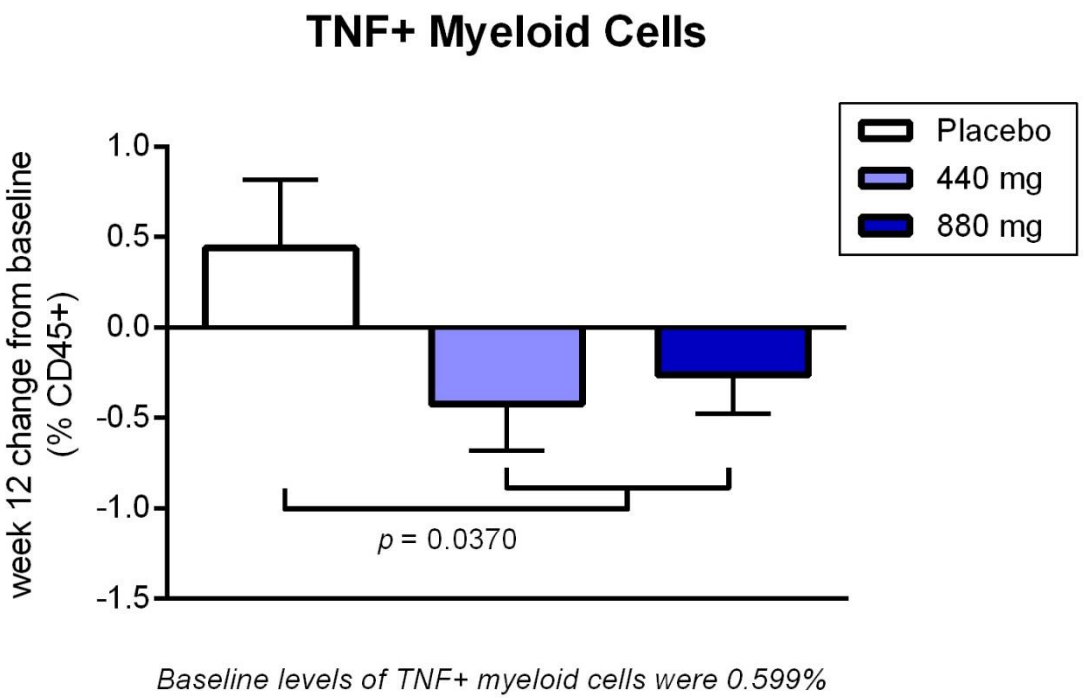
	Rate	Placebo Adjusted
Normalization < 250 ug/g		
Omilancor (440 mg) *Week 2*	40.6	19.2
Tofacitinib *Week 12*	29.0	N/A
Ustekinumab *Week 8*	30.3	8.5
Normalization < 150 ug/g		
Omilancor (440 mg) *Week 2*	33.3	15.1
Vedolizumab *Week 6*	29.3	12.5

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

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



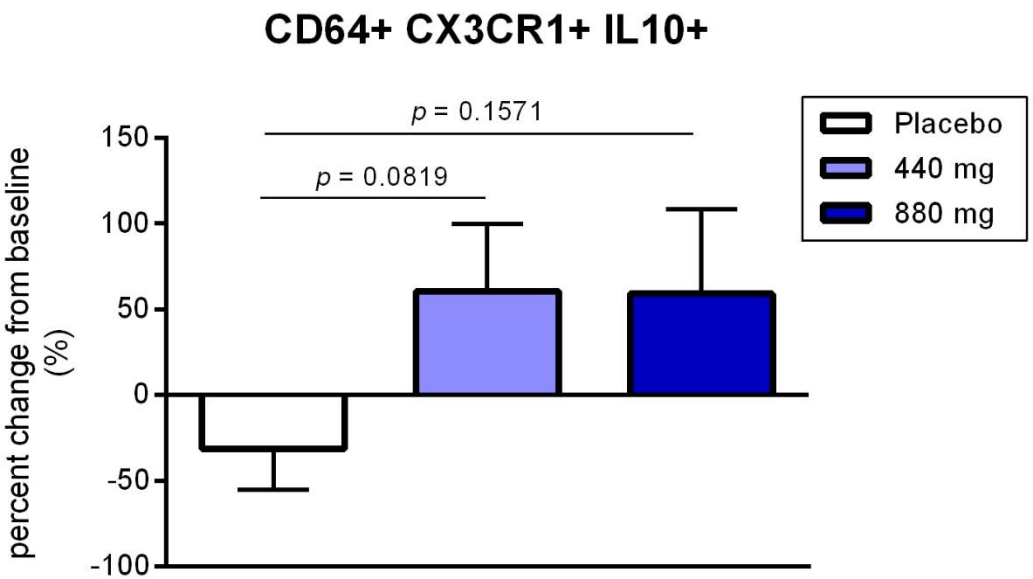
Baseline levels of Tregs cells were 2.24%

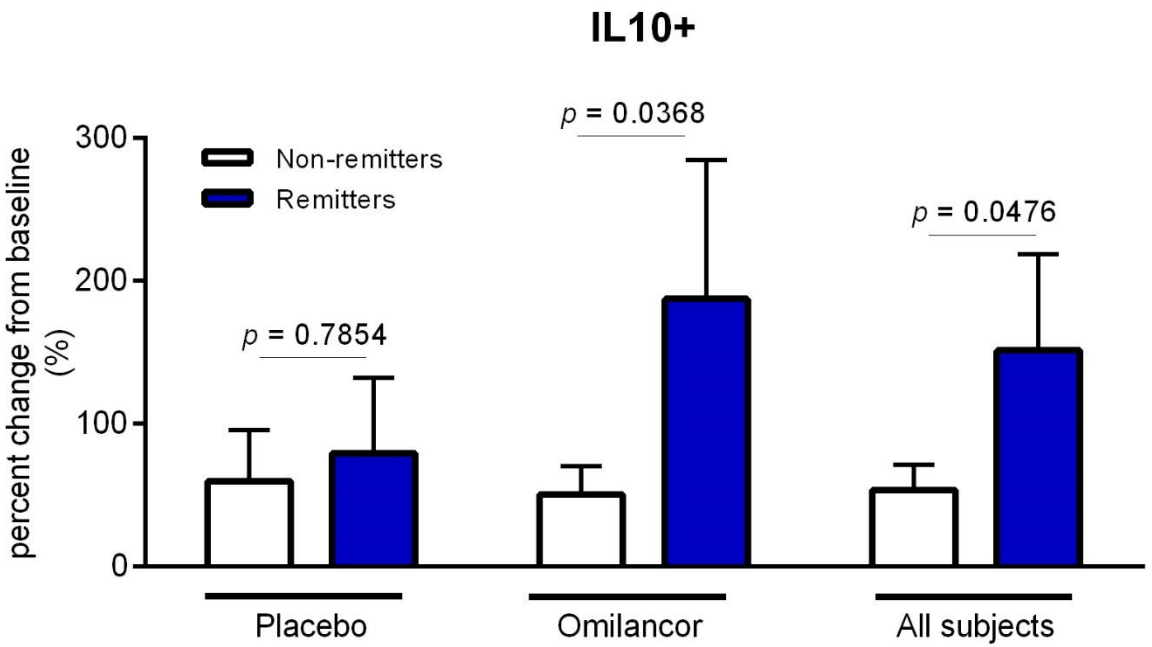


Baseline levels of TNF+ myeloid cells were 0.599%

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.

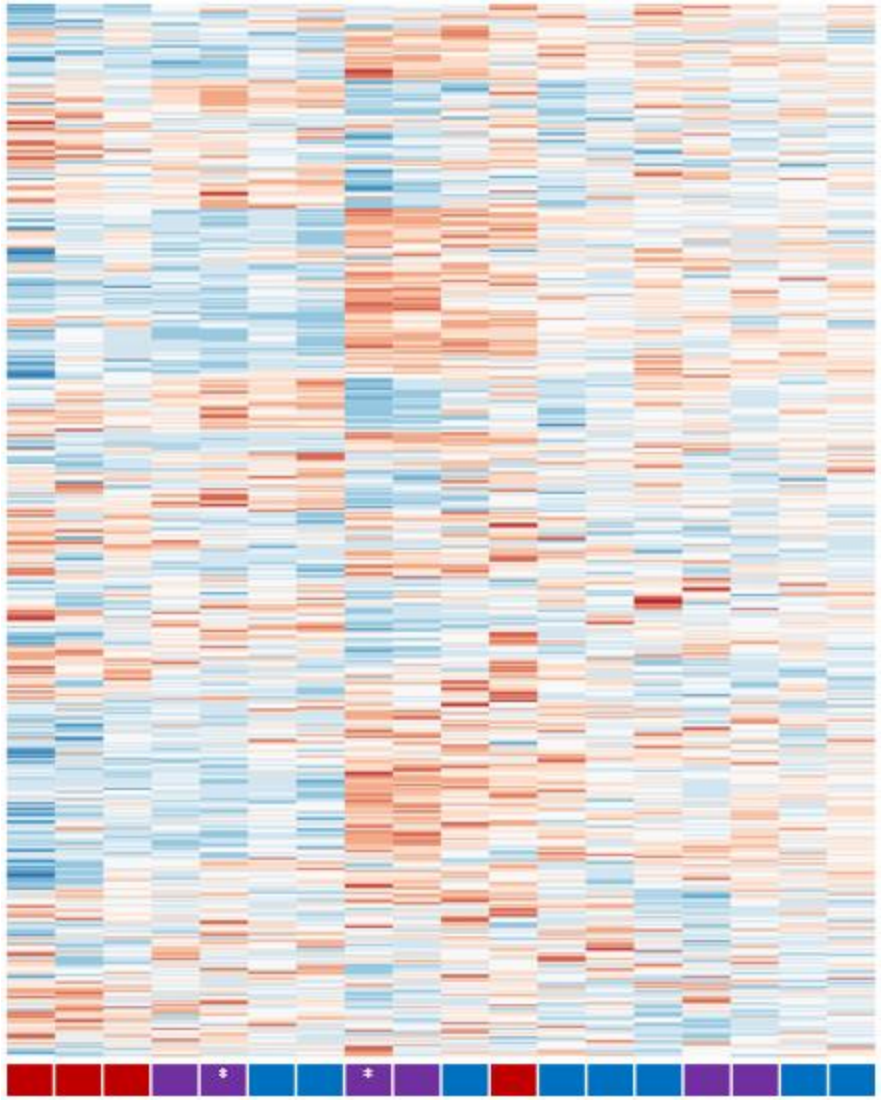
Increase in Colonic IL-10+ Cells Associated with Low Disease Activity



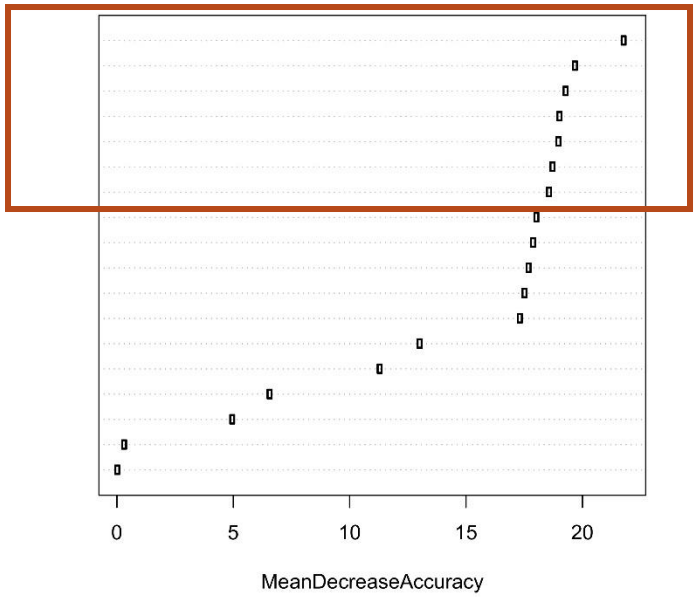


CD64+ and CX3CR1+ regulatory macrophages are key producers of IL10 and were associated with lower disease activity scores preclinically after omilancor treatment (p = 0.0378)

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.

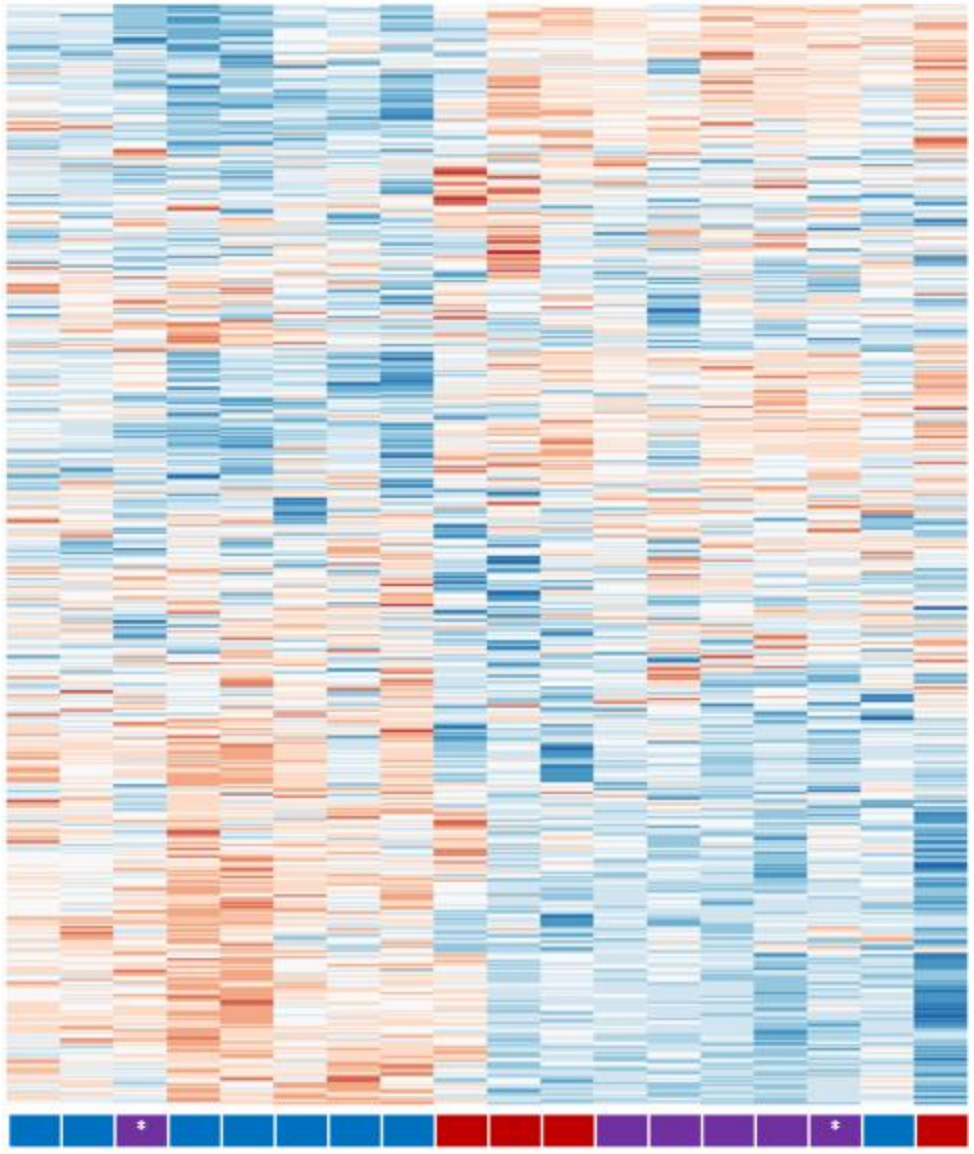


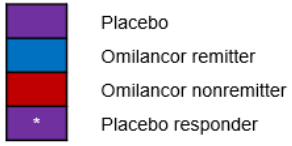
Overall accuracy – 75%

Omilancor remitters	75%
Omilancor nonremitters	75%

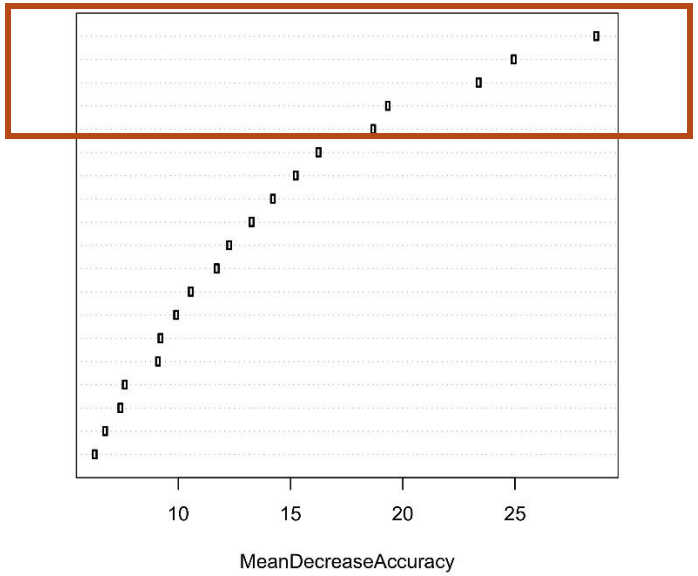
Model predicts response to omilancor would occur in 50% of patients who received placebo

Colonic gene expression pattern associated with clinical response to omilancor treatment





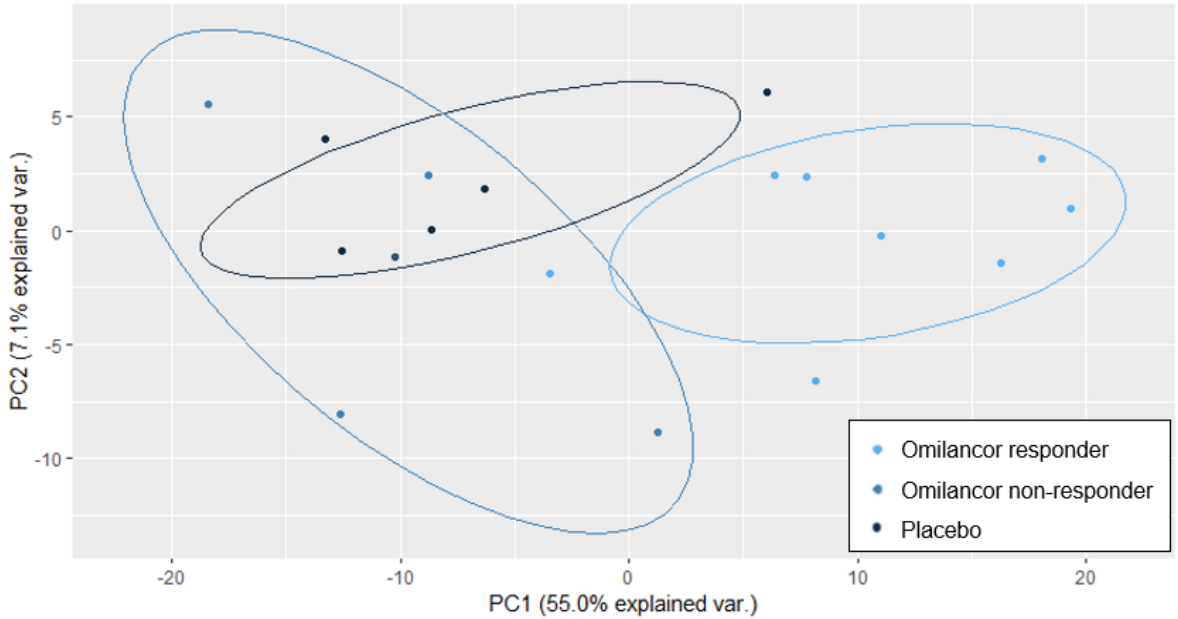
- DEGs upregulated by omilancor relative to baseline and placebo were associated with lipid metabolism and ion balance in addition to known elements of the LANCL2 pathway.
- DEGs downregulated were associated with immune system processes, primarily those in relation to neutrophils and leukocyte trafficking.

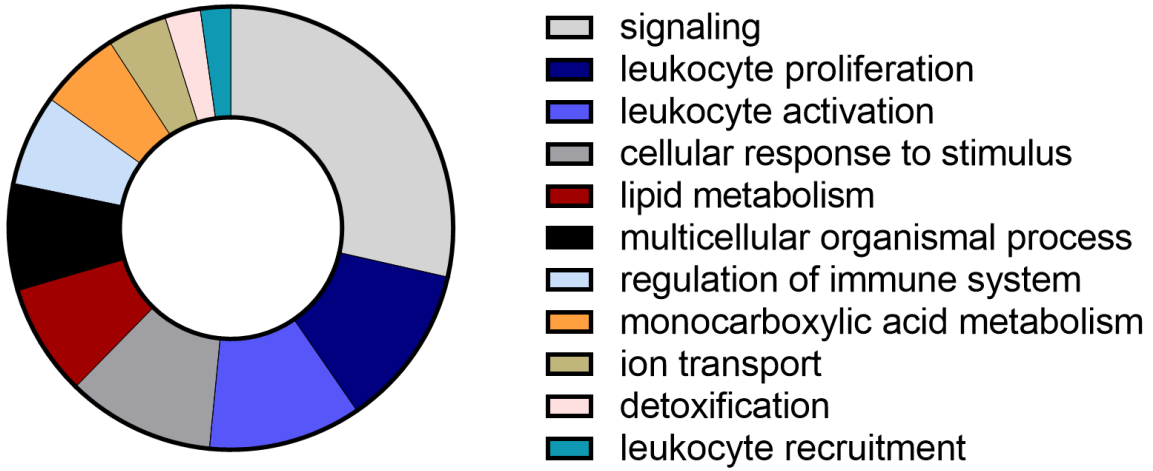


Overall accuracy – 83%

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

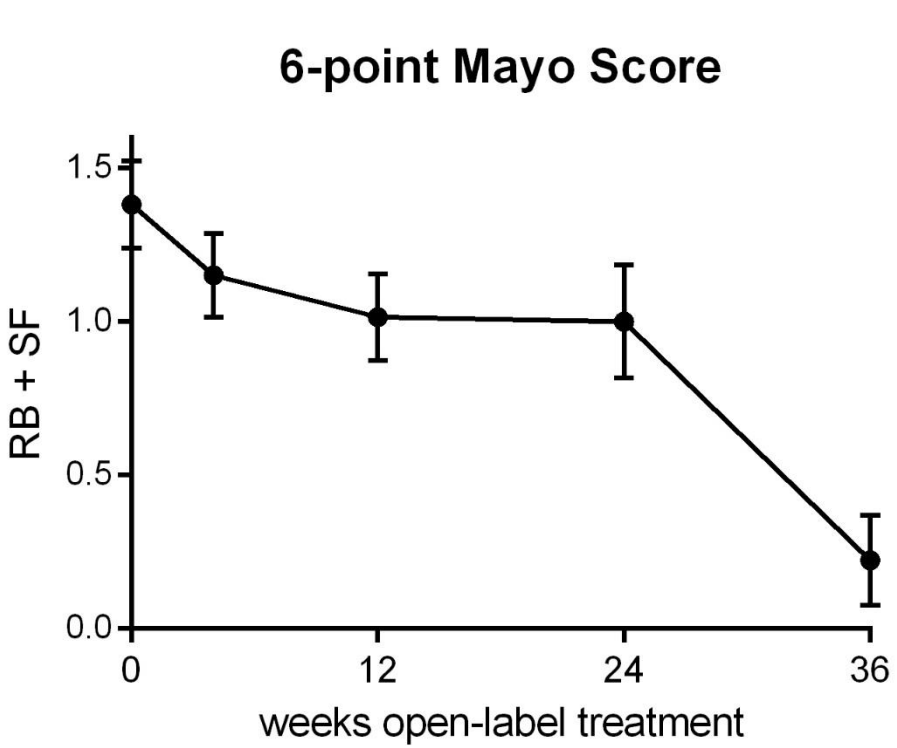
	Placebo	Omilancor 440 mg	Omilancor 880 mg
Subjects reporting ≥ 1 AE – no. (%)	20 (30.3%)	18 (27.3%)	20 (30.3%)
Total AEs – possibly related or higher	10	16	11
Total AEs – definitely related	0	0	0
Infections and Infestations	5 (7.6%)	4 (6.1%)	5 (7.6%)
Lymphopenia	1 (1.5%)	0 (0%)	0 (0%)
AEs experienced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

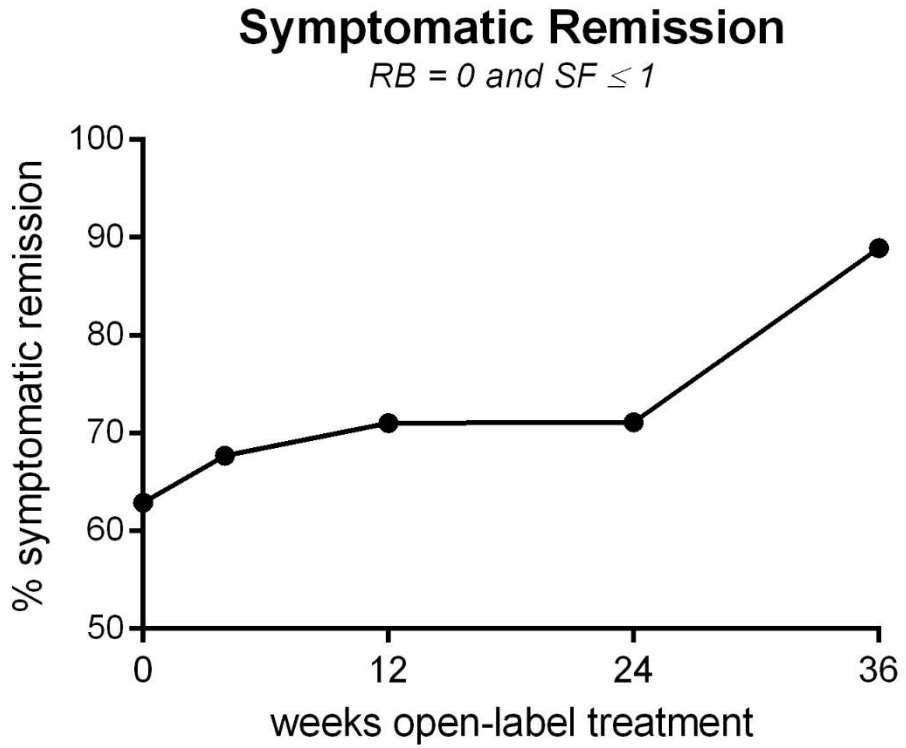
4 SAEs were reported during the induction phase. All were judged to be not related to study treatment:

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

No Inhibition of the JAK Pathway by Omilancor

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% (Δ = 16.7%) of the omilancor 880 mg group and 35.5% (Δ = 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

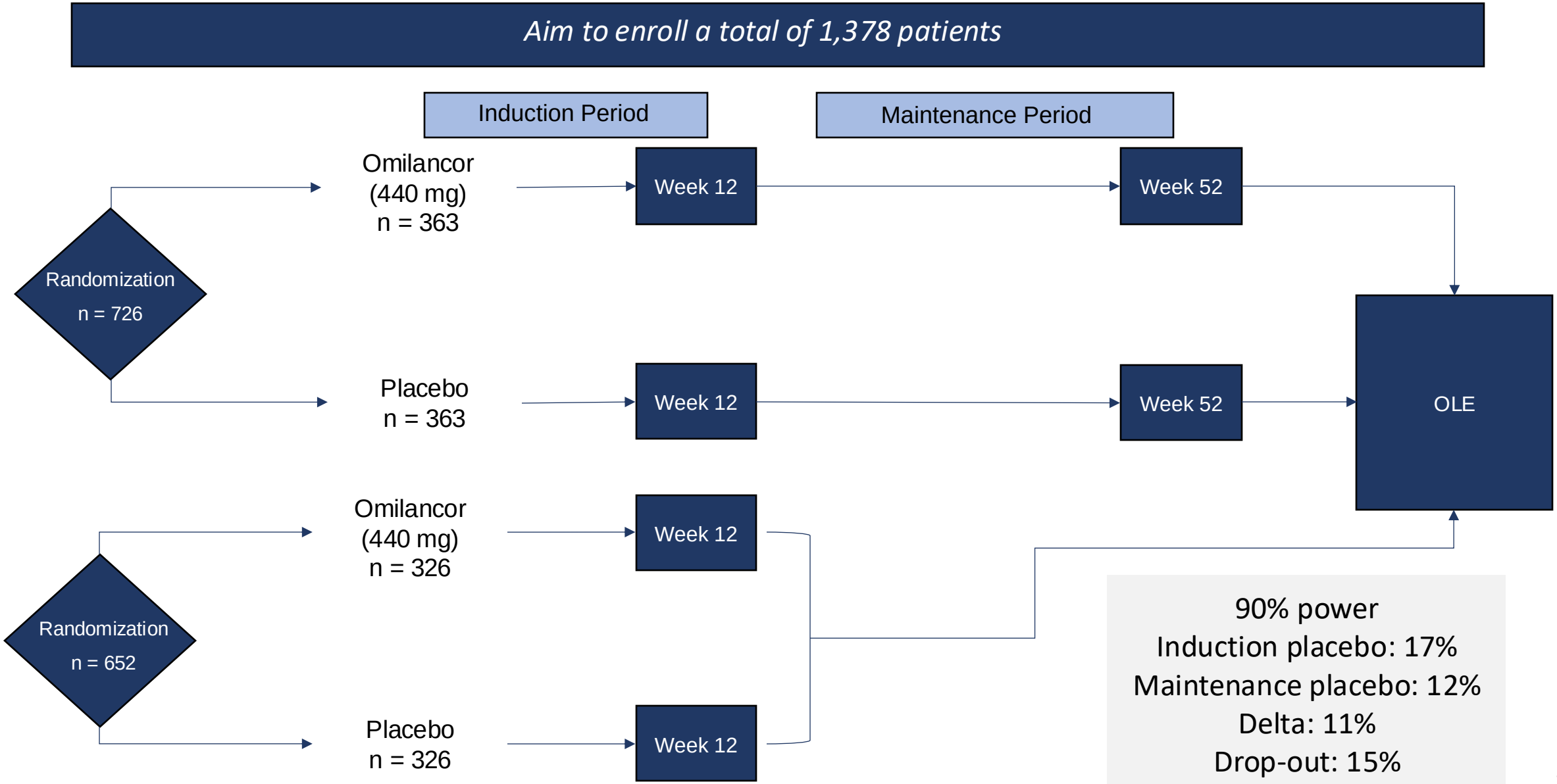
Phase 3 Design

- 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

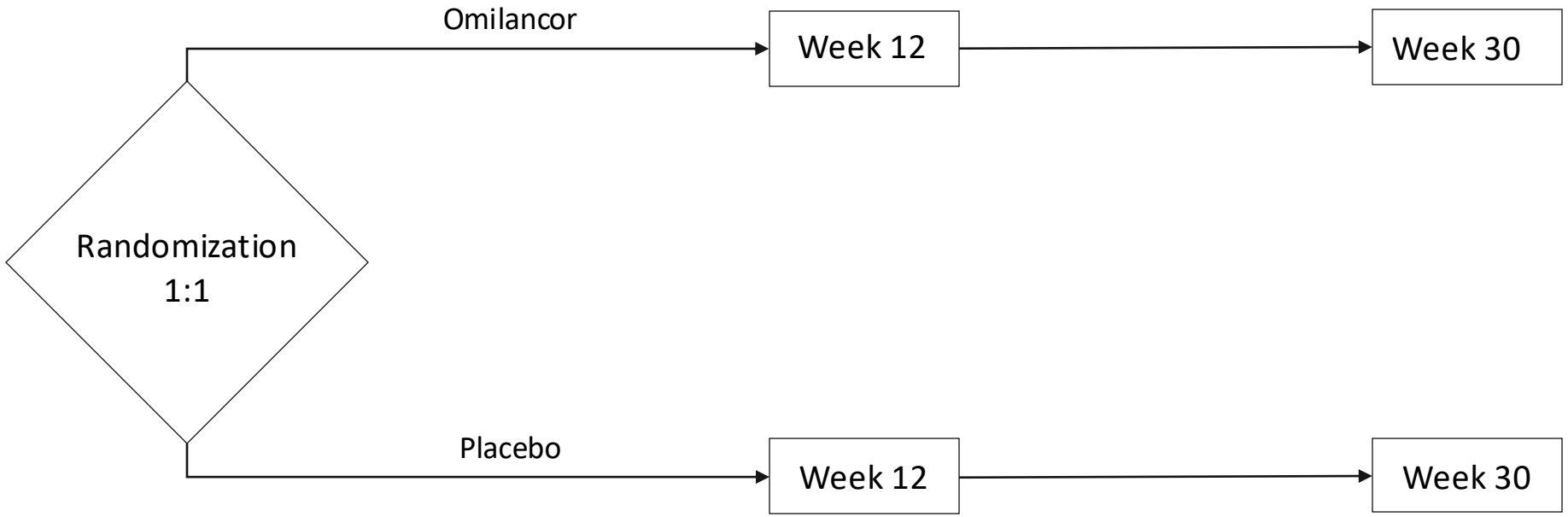
Initiate registrational Phase 3 trial

Phase 3 Pivotal Study Design of Omilancor in UC



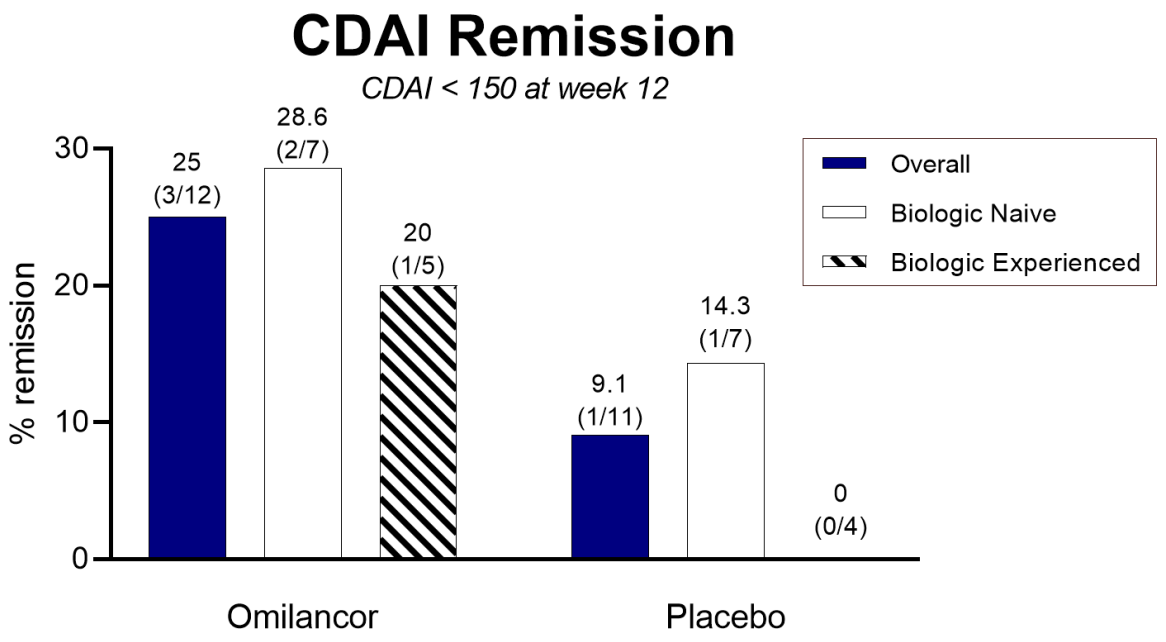
90% power
Induction placebo: 17%
Maintenance placebo: 12%
Delta: 11%
Drop-out: 15%

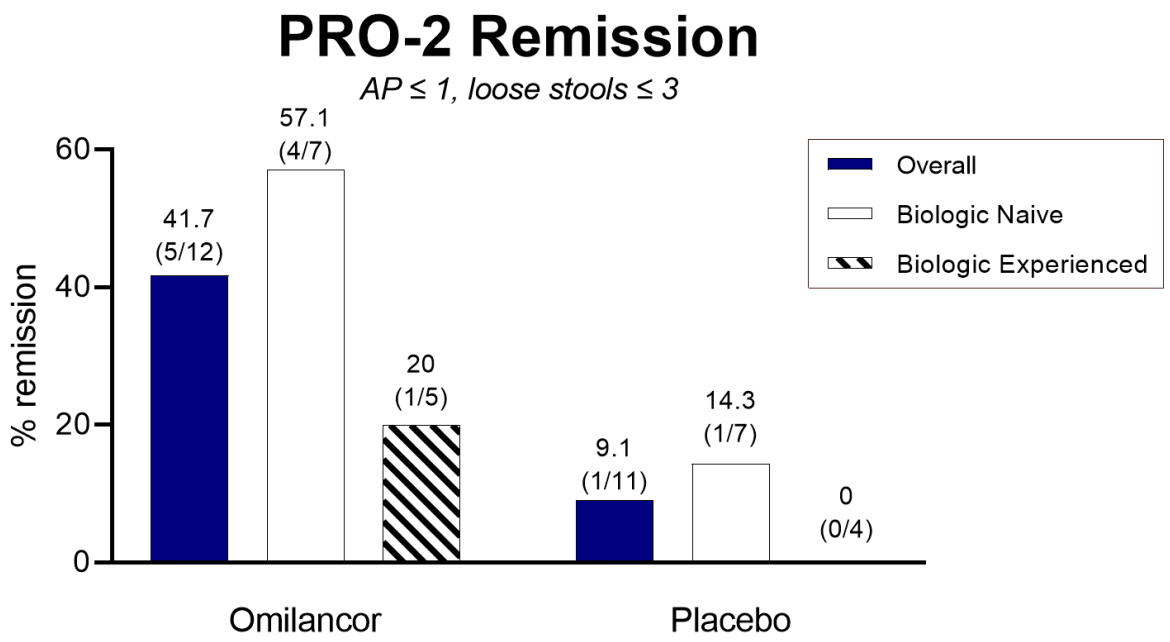
First-in-Indication Study of Omilancor in Moderate to Severe Crohn's Disease



- 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

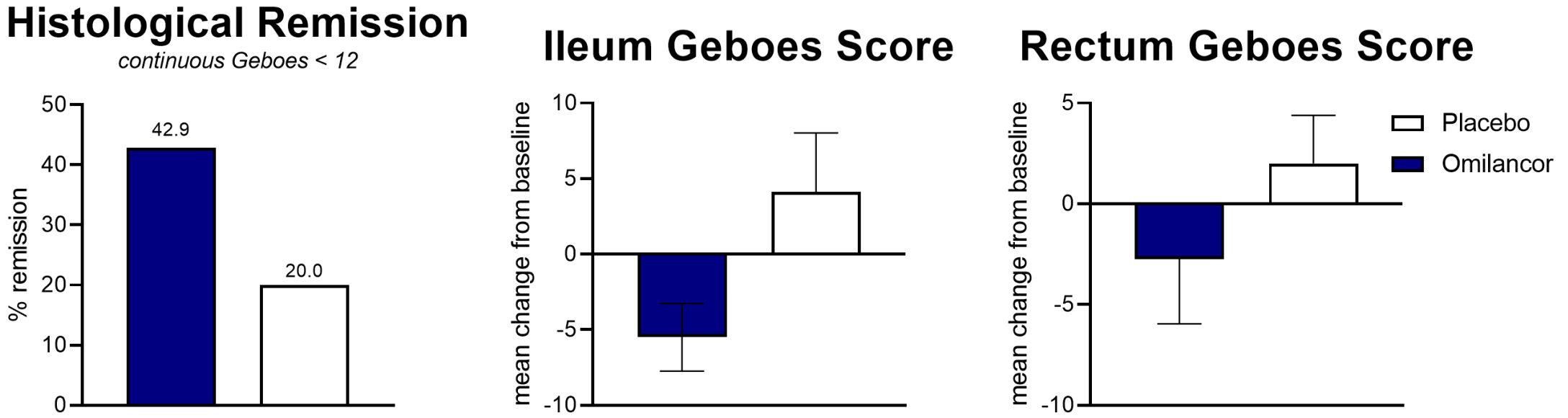




Population includes all subjects that had received study drug and met eligibility criteria

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

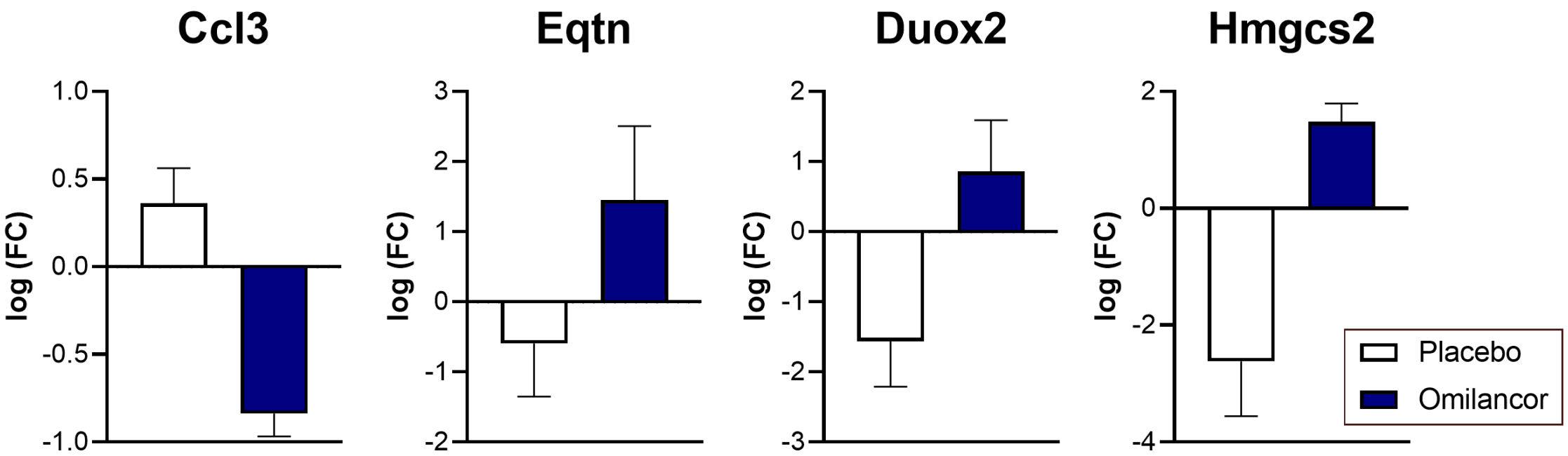
Omilancor treatment increases likelihood of histological remission at Week 12



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.

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

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 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.

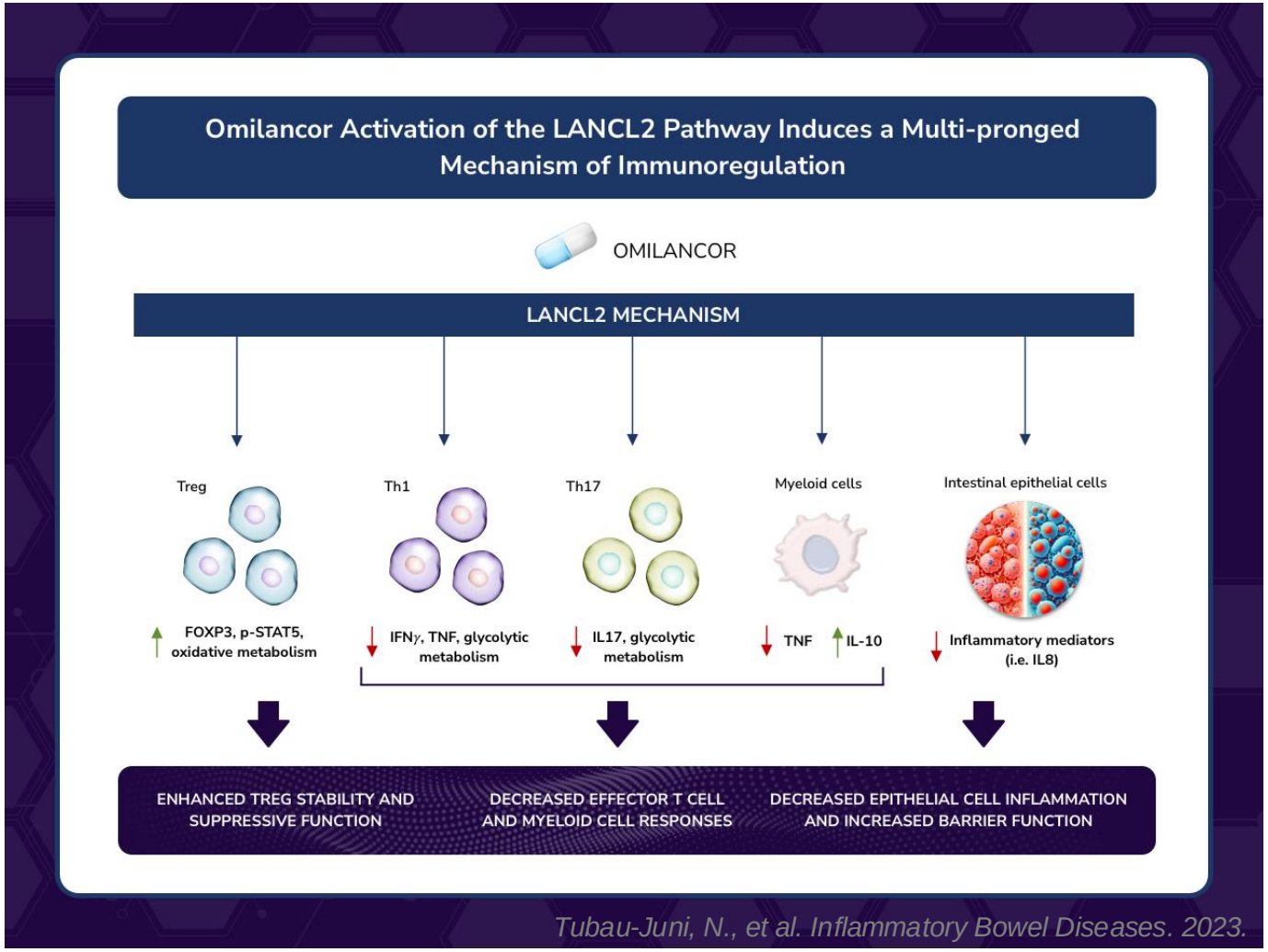
Omilancor offers an ideal mechanistic, safety and DDI profile to serve as a combination therapy with other small molecules or biologics.

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.



Tubau-Juni, N., et al. Inflammatory Bowel Diseases. 2023.

Omilancor has a low risk for negative DDI

Omilancor has limited systemic distribution with observed Cmax of 372 ng/mL and average Cmax 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 µL/min/mg in microsomes, 4.9 µL/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 µg/mL (12.6% inhibition at physiologically relevant concentration) and CYP3A4 at an IC50 of 4.53 µg/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 µg/mL (<1% inhibition at physiologically relevant concentration) and OATP1B3 at 0.234 µg/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

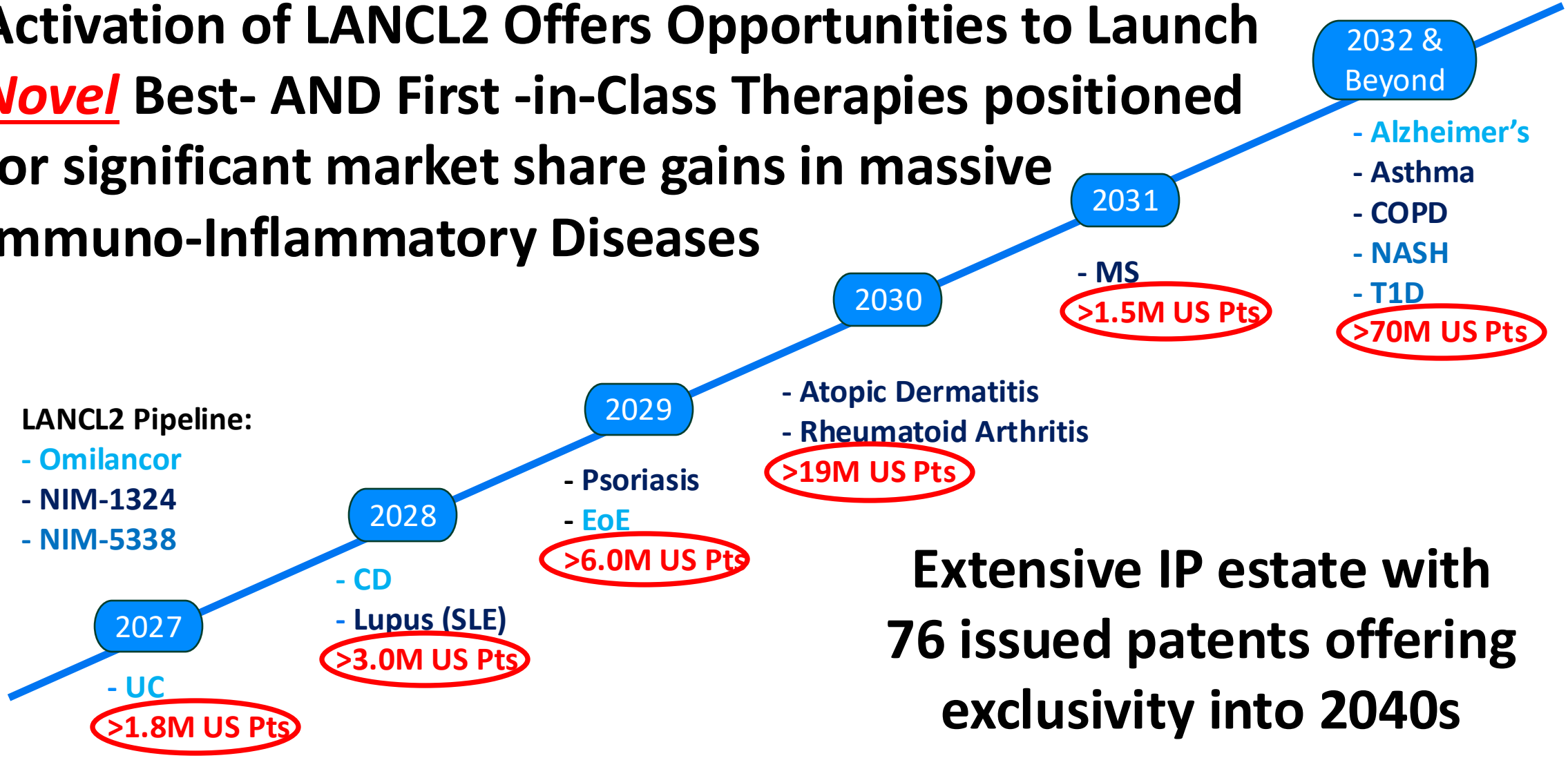
Omilancor CMC

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.

Pipeline in a Drug

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



Extensive IP estate with 76 issued patents offering exclusivity into 2040s

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).
- 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 accretion.