LANDDS BIOPHARMA

COMPANY PRESENTATION | OCTOBER 2021



Statements in this presentation about future expectations, plans and prospects for Landos Biopharma, Inc. (the "Company"), including statements about the Company's strategy, clinical development of the company's therapeutic candidates, the Company's anticipated milestones and future expectations and plans and prospects for the Company and other statements containing the words "subject to", "believe", "anticipate", "plan", "expect", "intend", "estimate", "project", "may", "will", "should", "would", "could", "can", the negatives thereof, variations thereon and similar expressions, or by stated discussions of strategy constitute forward-looking statements. Actual results may differ materially from those indicated by such forward-looking statements as a result of various important factors, including: the uncertainties inherent in the initiation and enrollment of future clinical trials, expectations of expanding ongoing clinical trials, availability and timing of data from ongoing clinical trials, expectations for regulatory approvals, other matters that could affect the availability or commercial potential of the Company's product candidates and other similar risks. In addition, the forward-looking statements included in this presentation represent the Company's views to change. However, while the Company may elect to update these forward-looking elements at some point in the future, the Company specifically disclaims any obligation to do so, except as may be required by law. These forward-looking statements should not be relied upon as representing the Company's views as of any date subsequent to the date hereof.





Pioneering Drug Development Platform

- Proprietary LANCE[®] advanced A.I. platform applied to the discovery and development of new therapeutic targets (LANCL2, NLRX1 and PLXDC2)
- LANCE helped advance 17 product candidates and cleared six INDs cleared in less than four years
- New immunometabolic targets: multimodal pathways at the intersection of immunity and metabolism

Ĩ **Strong IP and Financial Position**

- Composition of matter and method of use IP with long patent life 2035/2041 (at least 58 issued patents and over 64 pending applications)
- Raised \$188M plus a \$3M NIH grant to date, with runway through the end of 2023
- Executed \$218M China/Asia territory deal

High-Impact Clinical Stage Assets

- Omilancor, Phase 3-ready lead product candidate, is an orally active, once-daily, gut-restricted, first-in-class therapeutic for UC, CD and EoE
- NX-13 is an orally active, once-daily, gut-restricted, first-inclass therapeutic candidate in a Phase 1b study for UC
- LABP-104 is an oral, systemically distributed candidate for lupus and rheumatoid arthritis in a Phase 1 study

Innovative Inflammation & Immunology Pipeline

- 17 programs currently under development across 14 autoimmune disease indications
- Franchise of first-in-class oral therapeutics targeting immunometabolic function
- Opportunity to license and partner some programs while advancing our core programs to commercialization





Dr. Josep Bassaganya-Riera

Chairman of the Board, President, and CEO

Biotech entrepreneur and innovator with 25 years of scientific innovation in immunology, drug development, business development and fundraising experience.



Dr. Raquel Hontecillas

Chief Scientific Officer

20 years of translational experience in immunology, drug development, and the biotech industry focusing on infectious, autoimmune, and metabolic diseases.



Jyoti Chauhan, MS, RAC

Executive VP of Operations & Regulatory Affairs

Expertise encompassing strategic regulatory liaising with focus on policy analysis and filings, product development lifecycle & clinical trials management.



Dr. Andrew Leber VP of Discovery & Product Development

Expertise spans immunology and A.I.-based drug development for autoimmune disease with specific focus on CD, UC and lupus.



Marek Ciszewski, JD

VP of Financial Strategy & Investor Relations

25 years of biopharma and financial industry expertise, encompassing developing and managing capital structures for biotech companies.





Dr. Nuria Tubau-Juni

Director of Inflammation & Immunology

Expertise in I&I related to infectious and autoimmune diseases. She leads new target identification, mechanistic and translational studies.

Dr. Simon Lichtiger

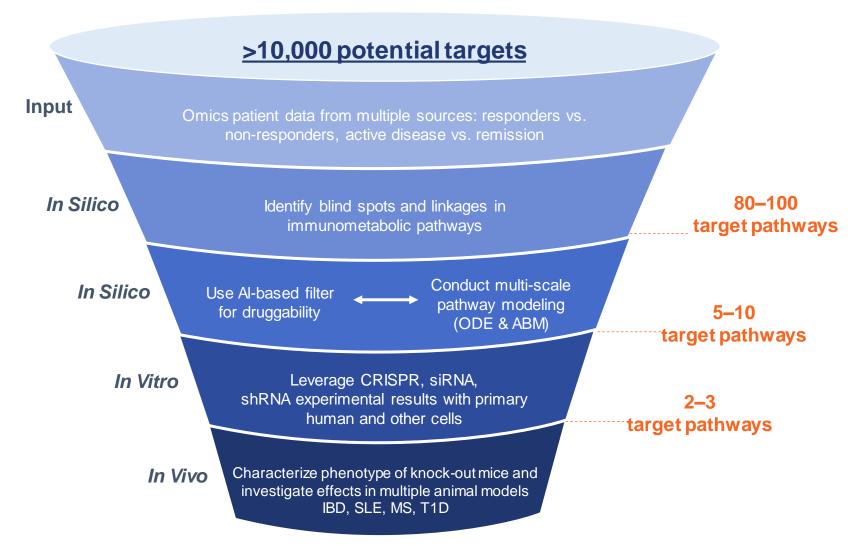
VP of Clinical Development & Medical Affairs

Clinical gastroenterologist and internal medicine expert with over 40 years of experience in CD and UC Phase 2 & 3 clinical trials.



LANCE[®] Advanced A.I. Precision Medicine Platform

<u>Identifies novel therapeutic targets and biomarkers</u> with significant potential to exert immunoregulatory control of patients with autoimmune diseases, <u>prioritizes targeted product candidates for certain indications</u> based on PK and the types of immune responses they elicit, and <u>identifies new precision medicine signatures</u>





Pipeline: Inflammation & Immunology Candidates with Novel Mechanisms

Pathway	Program*	Indication	Discovery	Preclinical	Phase I	Phase II	Phase III	Collaborators
	Omilancor	Ulcerative Colitis *						
		Crohn's Disease *						
		Eosinophilic Esophagitis						
		Psoriasis						
LANCL2		Atopic dermatitis						
		Lupus						
	LABP-104	Rheumatoid Arthritis						
	LABP-111	NASH						
		Type 1 Diabetes						
	NX-13	Ulcerative Colitis *						
		Crohn's Disease *						
NLRX1	LABP-66	Multiple Sclerosis						
NLKAI		Alzheimer's Disease						
	LABP-73	Asthma						
		COPD						
	LABP-69	Diabetic Nephropathy						
PLXDC2		Rheumatoid Arthritis						

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* Development and potential commercialization rights in China licensed in a territory deal.



Partnership with LianBio to Develop and Commercialize Omilancor and NX-13 in Greater China



Territory Deal Terms	•	Received \$18 million upfront from LianBio Additionally, eligible to receive up to \$200 million for development and commercialization milestones, as well as tiered low double-digit royalties
Additional Info	•	LianBio will aid the recruitment efforts for future global Phase 3 trials of omilancor and NX-13 in Greater China and other select Asian markets



<u>3Q'21</u>	<u>4Q'21</u>	<u>1Q'22</u>	<u>2Q'22</u>	<u>2H'22</u>
 ✓ UEGW abstract acceptance 	 ✓ Cleared LABP-104 Lupus IND 	 Omilancor Psoriasis IND 	• LABP-69 RA IND	• LABP-73 Asthma IND
 ✓ 2Q'21 Results ✓ Lead Academic 	 ✓ LABP-104 P1 1st human dosed 	 Omilancor Atopic Dermatitis IND 	• LABP-66 MS IND	 LABP-69 Diabetic nephropathy
R&D Collaborations	• LABP-104 RA IND	 NX-13 P1b UC topline data 	• LABP-66 AD IND	IND
✓ JHU– MS ✓ MSSM - IBD	 3Q'21 Results NX-13 IBS IND 	• 4Q'21 & FY'21 Results	 Omilancor P2 CD topline data 	 2Q'22 Results
✓ \$3M NIH grant for		Results		
omilancor in CD	 Enroll 1st omilancor P3 UC patient 		 LABP-104 P1 topline data 	
 ✓ LANCE[®] Platform Update 			• 1Q'22 Results	



Omilancor Overview



Indications

- Ulcerative Colitis (UC), Crohn's disease (CD) and Eosinophilic Esophagitis (EoE)
- A topical form is also in development for psoriasis and atopic dermatitis



- Activates LANCL2 pathway, a membrane receptor that has been shown to modulate immunological mechanisms



Orally active and gutrestricted, allowing target engagement within the GI tract without systemic distribution

Recent & Upcoming Milestones

- Successful outcome of an End-of-Phase 2 meeting in mild-to-moderate active UC with the U.S. FDA
- Initiated Phase 2 trial in moderate-to-severe CD in May 2021, results expected 1H 2022
- Expects to initiate a Phase 1b trial in EoE in 1H 2022

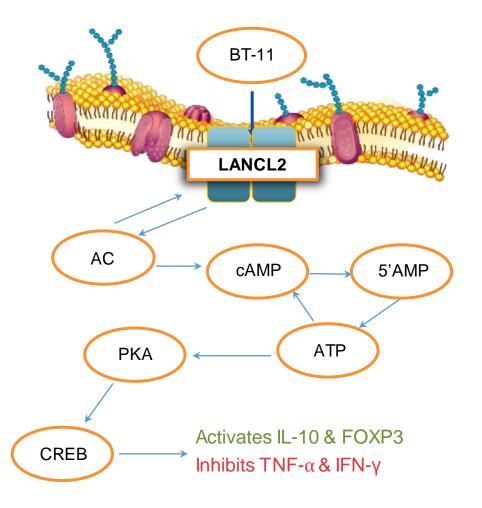


Omilancor Multimodal MoA:

- Decreases the production of inflammatory mediators tied to IBD (TNF α , IFN γ , MCP1, IL-6, and IL-8)
- Increases anti-inflammatory molecules in Tregs that protect from autoimmunity (IL-10, FOXP3)

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

- Decreases proliferation and differentiation of effector CD4+ T cells (Th1 and Th17)
- Supports the reduction of IL-8 and chemokine-dependent neutrophil influx





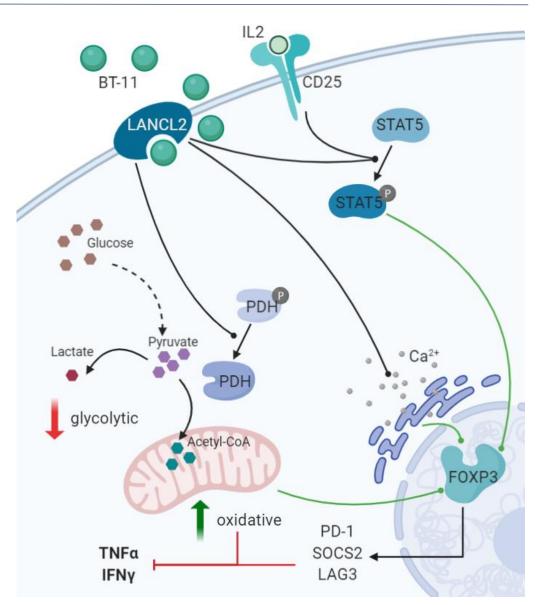
Lanthionine Synthetase C-Like 2 (LANCL2):

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

Anti-Inflammatory Effects of omilancor:

- Enhances CD25/STAT5 signaling to support the stable differentiation of regulatory CD4+ T cells with greater antiinflammatory functionality
- Increases PDH activity, resulting in increased oxidative metabolism supporting FOXP3 stability
- Downregulates glycolytic pathways associated with TNFα production and effector CD4+ T cells, including production of lactate and over-expression of ENO1
- Increases suppressive effects of Tregs due to enhanced immune checkpoint surface markers (LAG3 and PD-1)

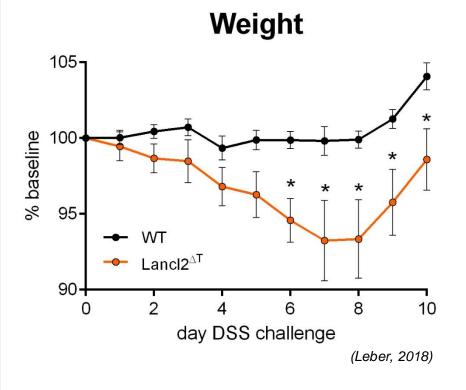
Leber, A., et al. Inflammatory Bowel Diseases. 2018 24:1978-1991. Carbo, A., et al. J Med Chem. 2016 Nov23;59(22):10113-10126. Leber, A., et al. J Immunol, 2019.

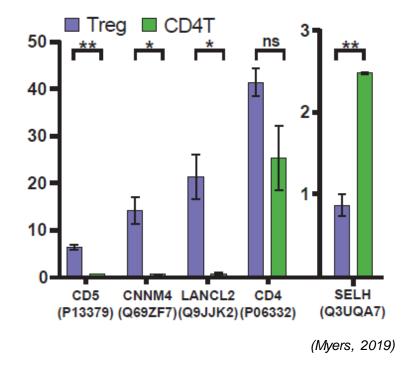




LANCL2 is a novel Treg associated receptor relevant to IBD

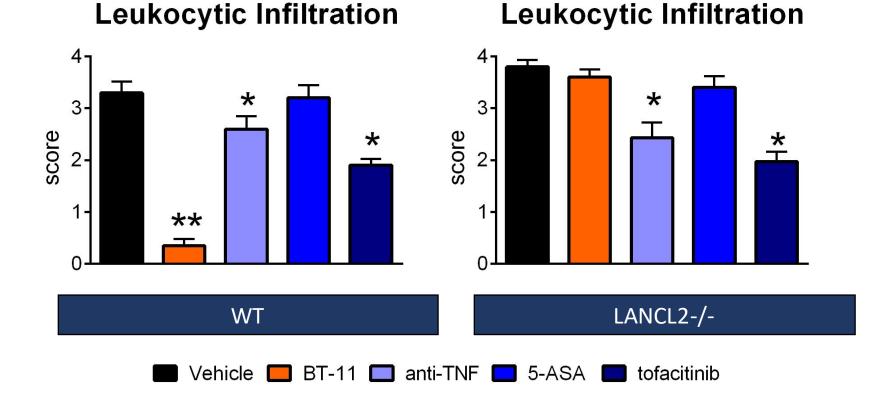
- LANCL2-/- mice have been characterized in multiple autoimmune conditions
- The loss of LANCL2 in CD4+ T cells results in worsening of disease severity and Treg defects in a DSS model of colitis
- LANCL2 was identified to be one of the three most differentially expressed proteins in Tregs relative to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







Efficacy of omilancor is specific to the unique LANCL2 pathway



The efficacy of omilancor in reducing histological disease severity is abrogated in LANCL2-/-mice. Efficacy of comparative therapeutics including anti-TNF and JAK inhibitors is not impaired by the absence of LANCL2.



- ✓ 17% clinical remission rate and 9% placebo-adjusted rate with biologic-like activity
- ✓ Well-tolerated with a clean safety profile
- ✓ Translation of immunological, biomarker results and mechanism of action
- ✓ Conveniently administered via oral once-a-day dosing
- ✓ Gut-Restricted

* The trial was a proof-of-concept study not powered for statistical significance



Positive Clinical Remission Rates

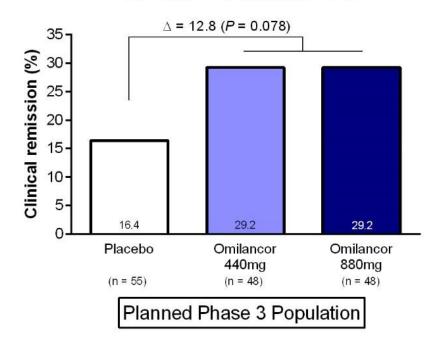
	Placebo (n = 66)	Omilancor 440 mg (n = 66)	Omilancor 880 mg (n = 66)
Clinical remission	22.7%	30.3%	31.8%
P value	-	0.340	0.235

Induced placebo-adjusted clinical remission rates of up to 31.8% at week 12

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	All randomized subjects
Analysis method	Stratified Cochran-Mantel-Haenszel Method
Planned stratifications	Previous biologic usage Baseline Mayo score greater than median value (7) Subjects with SF ≥ 2, RB ≥ 1, MES ≥ 2

Clinical Remission

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





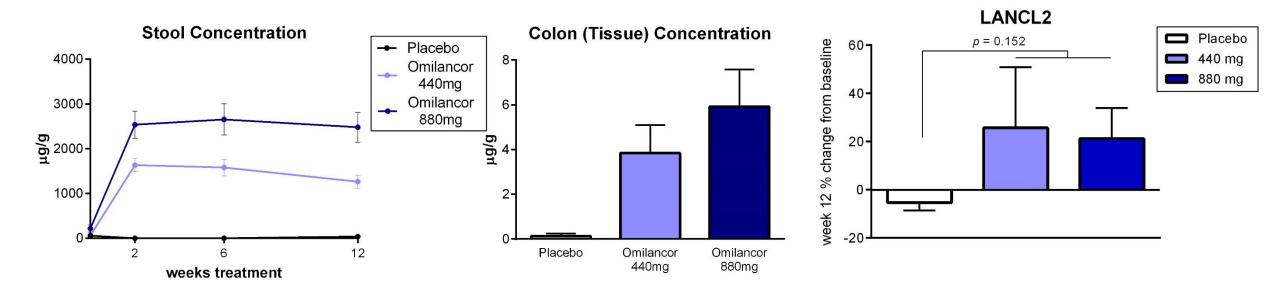
	Trial Name	Remission Rate	Placebo Adjusted Rate	Endpoint	Safety
Omilancor	BT-11-201	31.8 (all subjects)	12.8 (Planned Phase 3)	3-component remission	No identified trends in AE profiles

	Trial Name	Remission Rate	Placebo Adjusted Rate	Endpoint	Safety
Filgotinib	Selection	26.1 (biologic naïve) 11.5 (experienced)	10.8 (biologic naïve) 7.3 (experienced)	3-component remission	Class warnings for thrombosis, Herpes zoster and serious infection. Leukopenia
Ozanimod	True North	18.4	12.4	3-component remission	CV risk, macular edema, LFT elevations
Vedolizumab (ENTYVIO)	Gemini 1	16.9	11.5	Total Mayo score≤2	Slightly increased risk of infection. Severe hepatitis in small numbers of patients
Adalimumab (HUMIRA)	Ultra 1	18.5	9.3	Total Mayo score≤2	Increased risk for cancers and infections.
Tofacitinib (XELJANZ)	Octave 1	18.5	10.3	Total Mayo score≤2	Class warnings for thrombosis, Herpes zoster and serious infection. Leukopenia

Filgotinib (Gilead press release May 2020); Ozanimod (Sandborn, W., et al. 2016); Vedolizumab (Feagan, B., et al. 2013); Adalimumab (Reinisch, W., et al. 2011, Sandborn, W., et al. 2012); Tofacitinib (Sandborn, W., et al. 2017, D'Amico, F., et al. 2019)

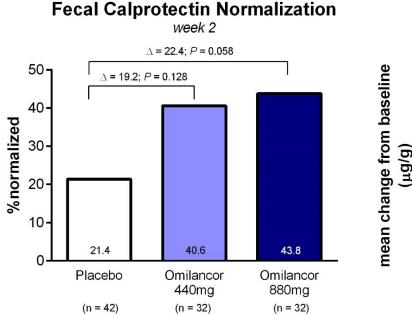


PK/PD results validate sufficient target engagement at both doses

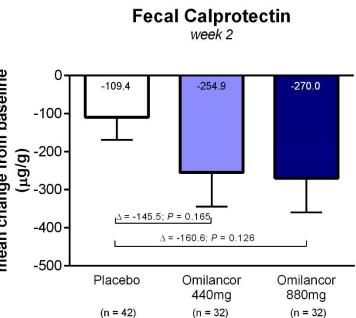


- Omilancor stool concentrations stable between 2 and 12 weeks of dosing
- No significant difference in stool concentrations between UC patients after 12 weeks and healthy volunteers after 7 days
- Stool and tissue concentration scale in a near dose-proportional manner
- 440 and 880 mg doses effectively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





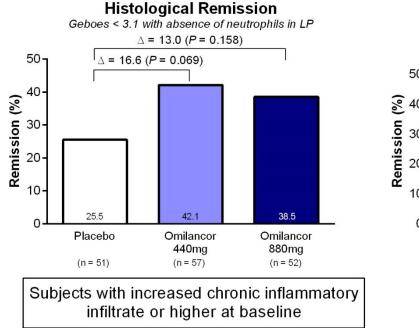
Fecal calprotectin considered normalized at < 250 ug/g Inclusive of subjects with abnormal levels at baseline

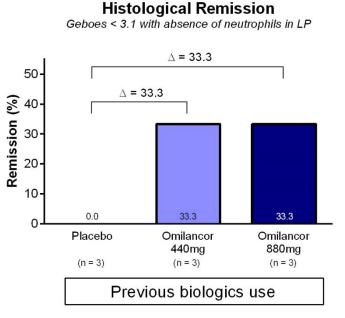


	Rate	Placebo Adjusted
Normalization < 250 u	ıg/g	
Omilancor (440 mg) <i>Week 2</i>	40.6	19.2
Tofacitinib Week 12	29.0	N/A
Ustekinumab Week 8	30.3	8.5
Normalization < 150 u	ıg/g	
Omilancor (440 mg) Week 2	33.3	15.1
Vedolizumab Week 6	29.3	12.5



Improvement in Histological Remission





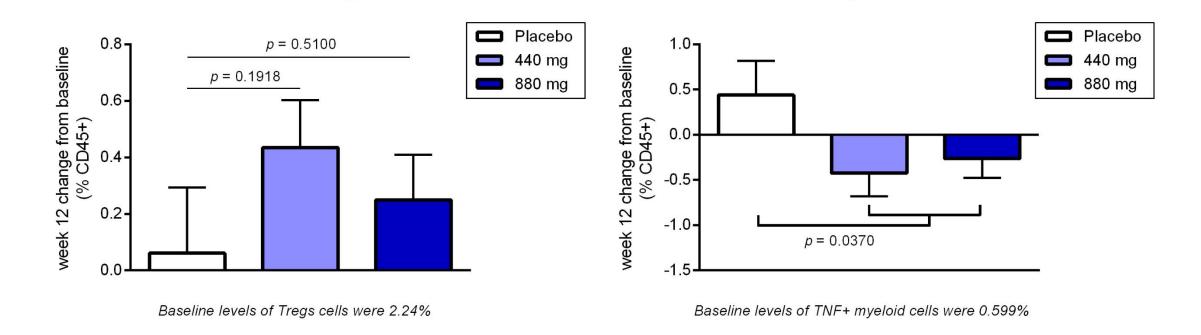
- Geboes score < 3.1 is indicative of:
- No ulceration, erosion, or granulation
- Absence of neutrophils in the epithelium

- No crypt destruction
- No elevation of neutrophils in the lamina propria
- Histological remission has been associated with a >40% increase in patients remaining symptom free, as well as a lower rate of relapse, hospitalization, and colorectal cancer in previous meta-analyses

TNF+ Myeloid Cells

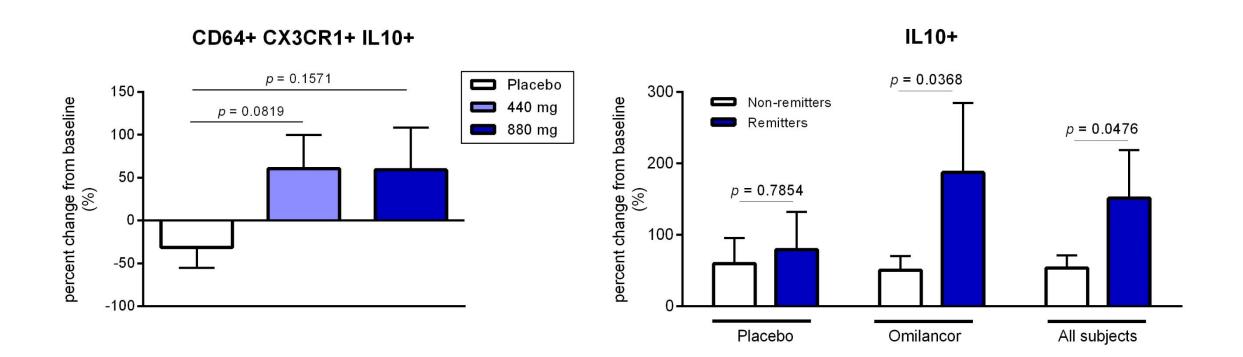
Colonic Tregs, TNF-producing Cells, TNF and IL-6 Concentrations

CD25+ Tregs



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.

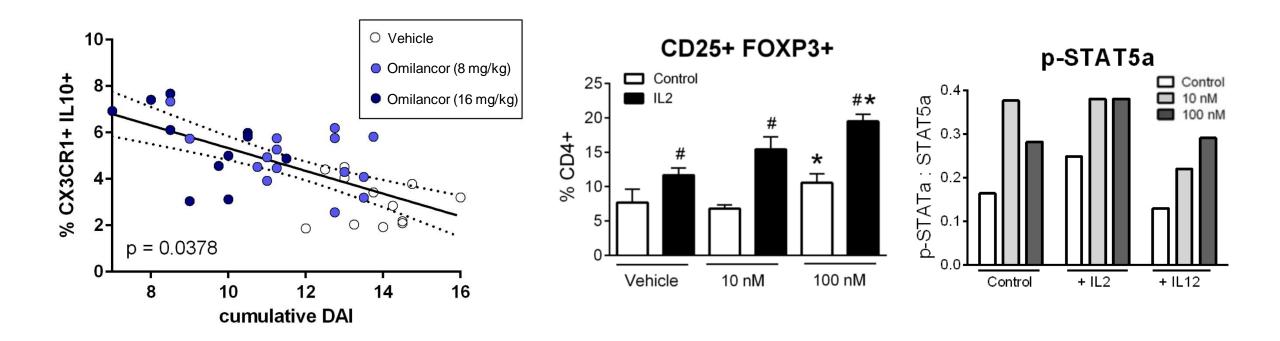




CD64+ and CX3CR1+ regulatory macrophages are key producers of IL10 and were associated with lower disease activity scores preclinically after omilancor treatment (p = 0.0378)



Omilancor treatment impacts CD4+ and myeloid regulatory cell types



Increases of CX3CR1+IL10 producing regulatory macrophages resulting from omilancor treatment are significantly associated with disease activity in mouse models. Omilancor induces differentiation of CD25^{hi} Tregs and the phosphorylation of STAT5 beyond IL-2 treatment.



Omilancor/LANCL2 agonists

- Demonstrated ability to translate mucosal Treg responses to patients with association of increased IL-10 to clinical response in ulcerative colitis
- Strong POC clinical efficacy
- Induction of both increased Tregs and increased suppressive capacity
- Wide efficacy and safety range with stable efficacy shown between 440 and 880 mg/d
- Robust maintenance of Treg phenotype in inflammatory environments
- Omilancor results in more potent phosphorylation of STAT5 than IL-2
- Oral
- No immunogenicity risk
- Able to decrease TNF, IL-6 and other inflammatory cytokines independently of Tregs

IL-2 Based Approaches

- Increase of CD25+ Tregs in blood in lupus patients (NKTR-358) with an unknown relationship to clinical responses
- No clinical efficacy data reported
- Limited data showing increased function independent of Treg population expansion
- Narrow dose titration window due to connection to proliferation of effector cells
- Lack of data showing benefit to Treg in non-Treg biased or inflammatory conditions
- Modified IL-2s result in less potent phosphorylation of STAT5 than native IL-2
- Injectable
- Potential for immunogenicity
- No direct effects on inflammatory pathways



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

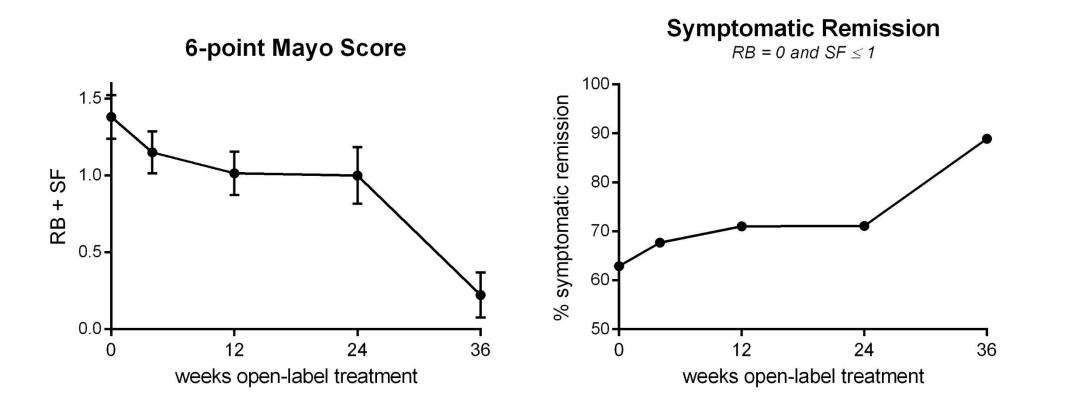
	Placebo (n = 66)	Omilancor 440 mg (n = 66)	Omilancor 880 mg (n = 66)
Subjects reporting ≥ 1 AE – no. (%)	20 (30.3%)	18 (27.3%)	20 (30.3%)
Total AEs – possibly related or higher	10	16	11
Total AEs – definitely related	0	0	0
Infections and Infestations	5 (7.6%)	4 (6.1%)	5 (7.6%)
Lymphopenia	1 (1.5%)	0 (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 Pathwayby Omilancor





- 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.



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

 Landos and the FDA agreed on key elements of pivotal global Phase 3 program that are necessary to prepare for regulatory approval

PACIFY.

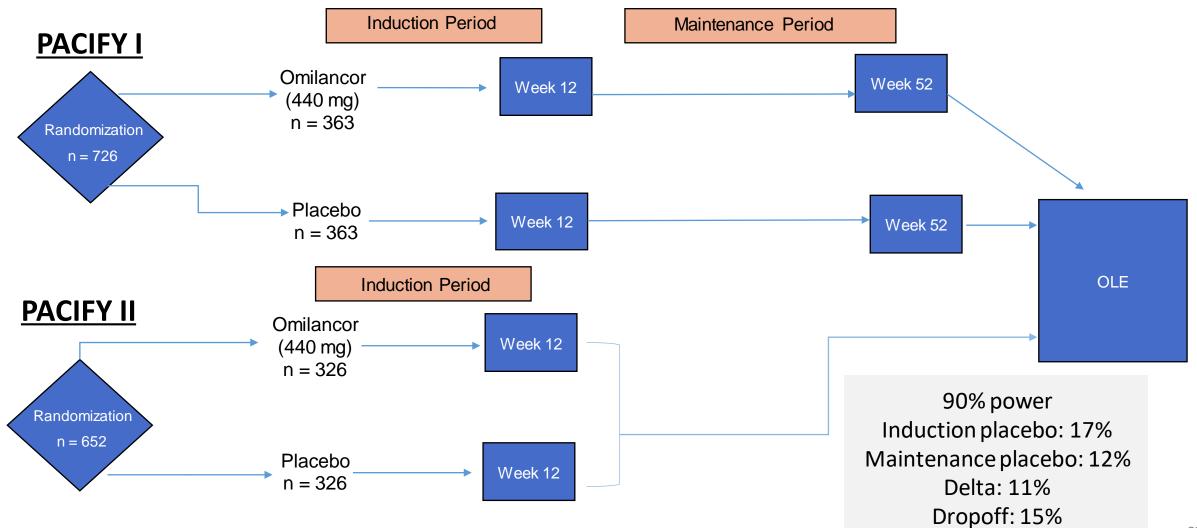
Phase 3 Design

- Total of 1,378 patients with mild-to-moderate 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 (label: mucosal healing)



PACIFY Phase 3 Pivotal Study Design of Omilancor in UC

Aim to enroll a total of 1,378 patients



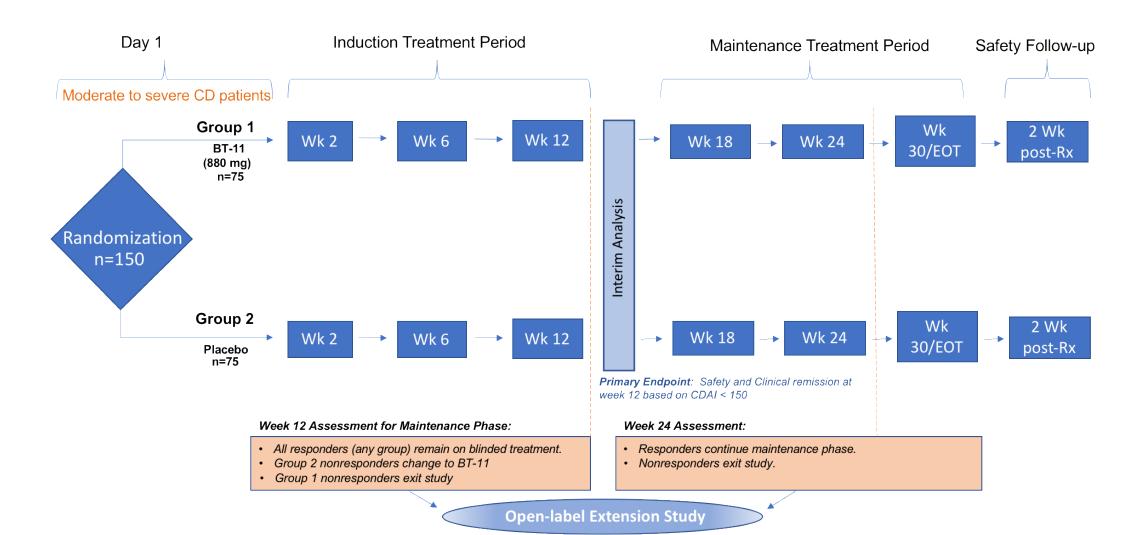
PACIFY Phase 3 Omilancor Study (PACIFY) Enrollment Criteria

- Male and female subjects aged 18 to 75 years, inclusive
- Diagnosis of UC for at least 3 months prior to screening
- Mild to moderate UC, as defined by a modified Mayo Score greater than or equal to 4 at baseline with a MES equal or greater than 2 and a <u>rectal bleeding subscore of at least 1</u>. Offering the broadest possible label (90% of UC patients with active disease).
- If subjects have previously received biologic therapy for UC (i.e., tumor necrosis factor [TNF] antagonists, vedolizumab or ustekinumab), they must have a washout period of 8 weeks prior to randomization, and any previous failure of biologic treatment is limited to only one class of biologic. <u>Controlled stratification to ensure a minimum of 20% of the overall population and a</u> <u>maximum of 40%.</u>
- If subjects are receiving the following UC treatments, they must be on a stable dose for at least 1 month prior to randomization: 5aminosalicylates (5-ASAs) (not exceeding 4.8 g per day), oral corticosteroids (not exceeding prednisone 20 mg, budesonide 9 mg, or equivalent)
- If subjects are receiving bile-salt sequestrant, they must be on a stable dose for at least 3 months prior to randomization

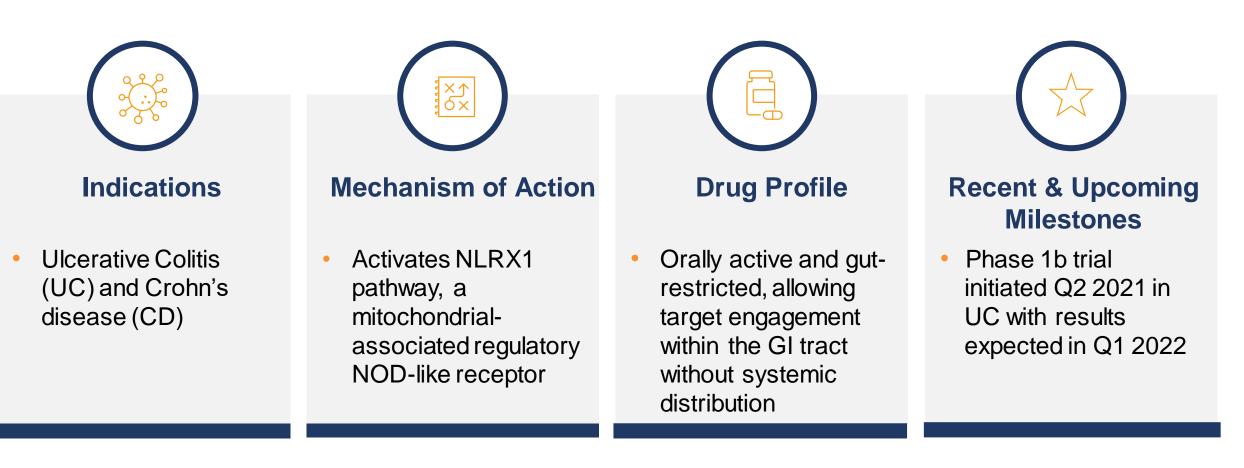


Ongoing Omilancor Phase 2 Study Design in CD

Results expected in 1H 2022







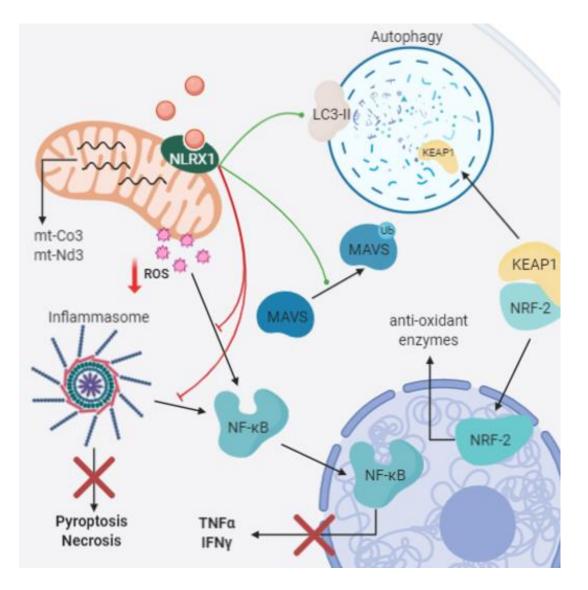


NX-13 multi-modal mechanism of action by activating the NLRX1 pathway

- Decreases cellular reactive oxygen species
- Antagonism of NF-kB activation resulting in downregulation of myeloid cell and T cell derived cytokines like TNF and IFNγ
- Decreases inflammasome formation (NLRP1 and NLRP3)
- Decreases differentiation of effector CD4+T cells

NLRX1 activation in intestinal epithelial cells increases mitochondrial metabolism and prevent oxidative stress

 Favors cell survival, the maintenance of barrier integrity and the expression of tight junction proteins



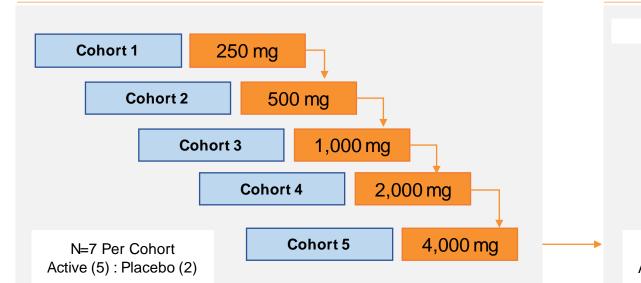


NX-13 Phase 1a Results: MTD 10-fold greater than anticipated therapeutic dose

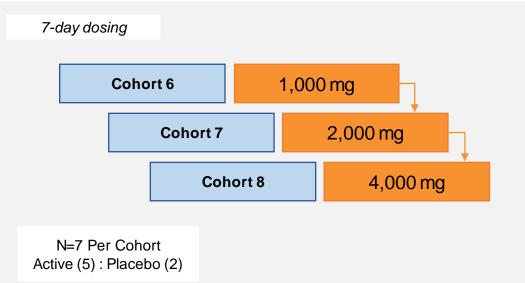
• No serious adverse events

Single Ascending Dose

- GI concentrations >2500-fold peak plasma concentrations
- 56% of subjects dosed with NX-13 brought fecal calprotectin to at or below detection limit after single oral dose
- Mean levels of FCP at all NX-13 tested dose levels on d 2 near that of omilancor 500 mg (12.3 μg/g)
- All primary and secondary endpoints in safety and tolerability were achieved

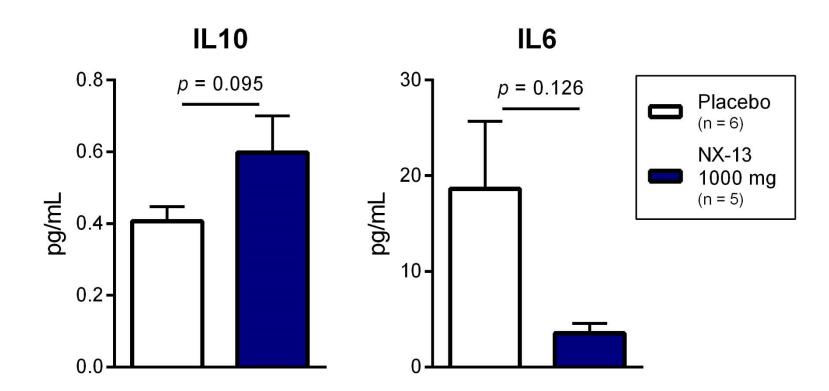


Multiple Ascending Dose





NX-13 Promotes Detectable Changes in Cytokines in NHVs

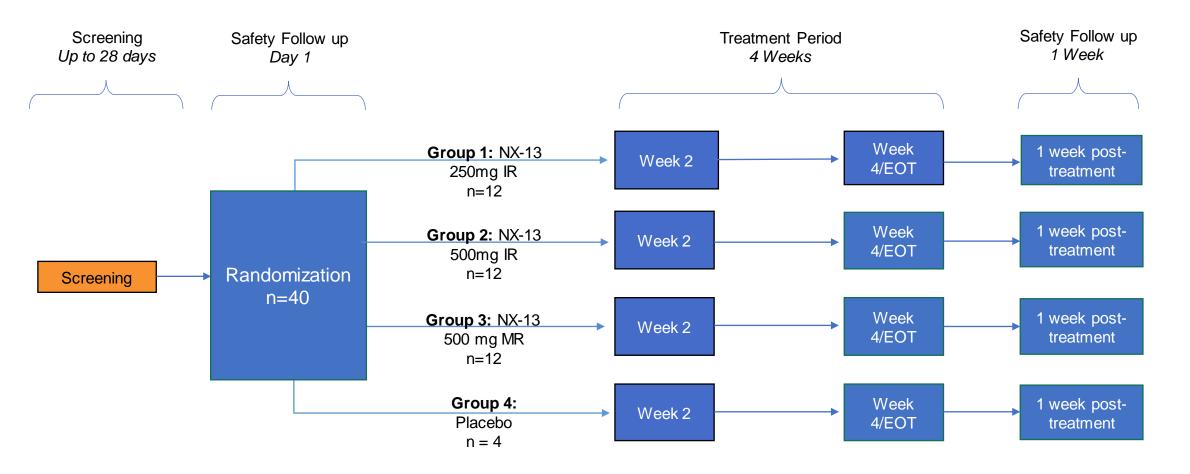


Serum cytokines from normal healthy volunteers dosed with 1000 mg (lowest dose tested the MAD) or placebo for 7 days results in a nearly 50% increase in IL-10 and over 5-fold reduction in IL-6 relative to placebo

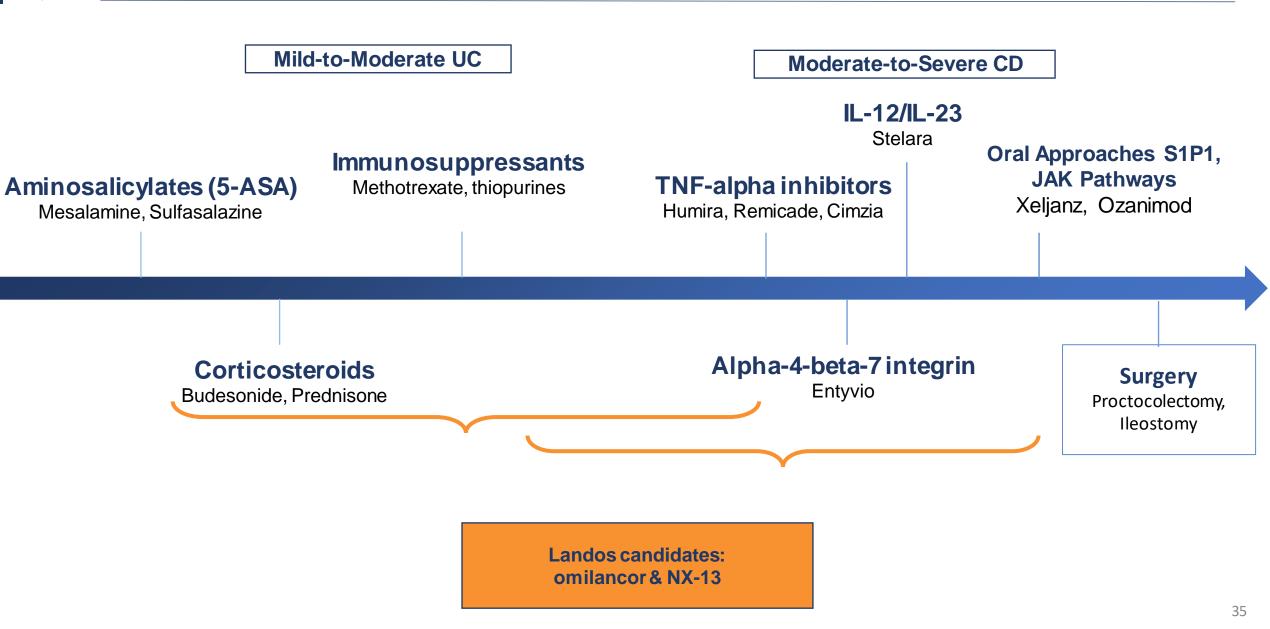


Ongoing NX-13 Phase 1b Trial Design in UC

- **Primary endpoints:** Evaluate safety and pharmacokinetics of multiple dose levels
- **Expected data readout:** Q1 2022











- Systemic Lupus Erythematosus (SLE)
- Rheumatoid Arthritis (RA)
- Other indications may be explored based on clinical and translational findings



- a membrane receptor that has been shown to modulate immunological mechanisms
- Same MoA as omilancor



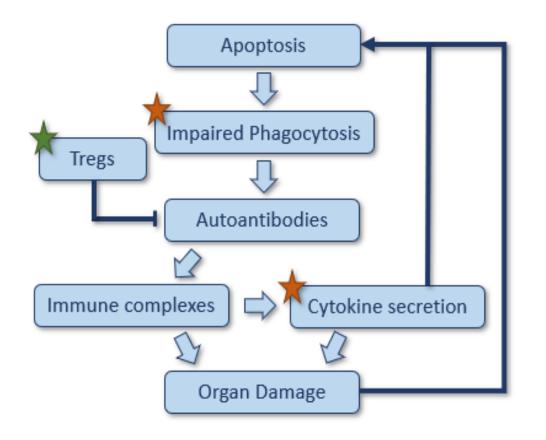
- Orally active with systemic distribution
- Once daily dosing

Recent & Upcoming Milestones

- First dosing in a Phase 1a NHV first in human study initiated in October 2021
- Abstract of preclinical findings accepted for oral presentation at ACR Convergence 2021

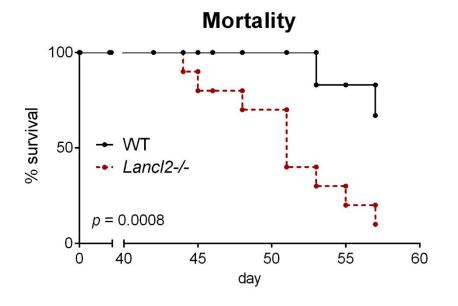


LANCL2 in Lupus



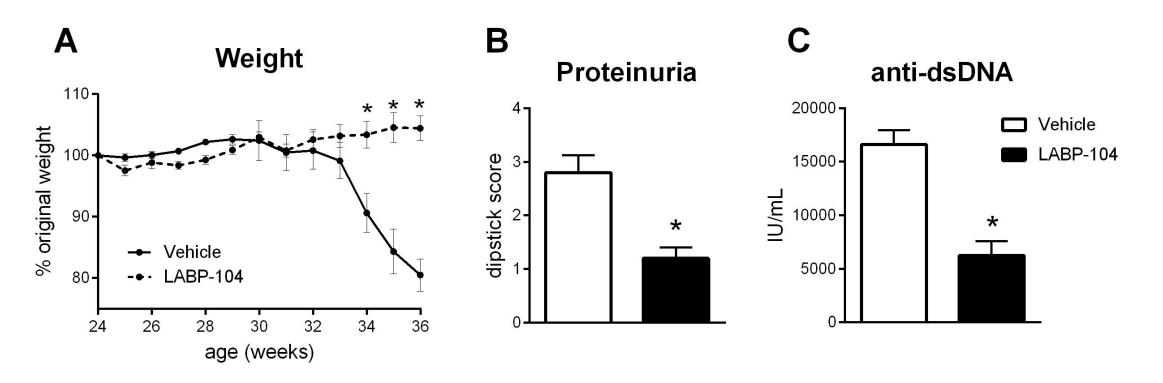
 LANCL2 intersects the pathogenesis at multiple levels including reversing the impaired Treg function and inhibiting cytokine secretion resulting from impaired phagocytosis

- Loss of LANCL2 increases mortality, spleen size and autoantibody production in TLR7-induced model of SLE (presented AAI 2019)
- LANCL2 and markers of downstream metabolic events are downregulated in SLE patient PBMCs



LANCL2-/- mice experience accelerated and increased mortality in an R848 induced model of lupus and TLR activation

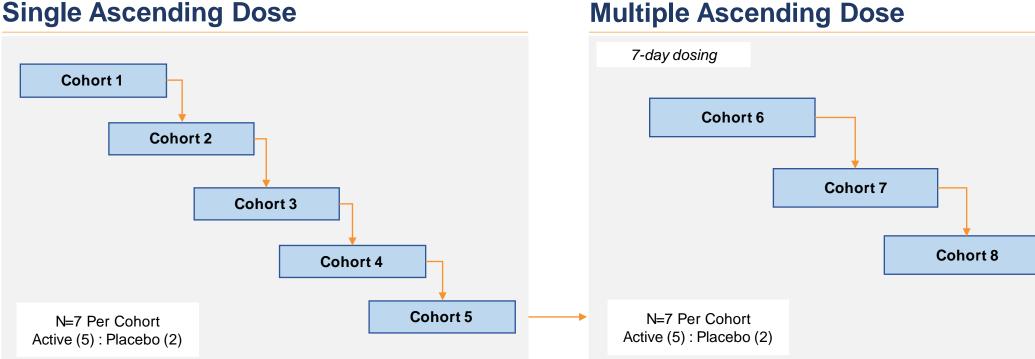
LABP-104 protects against markers of disease in an NZB/W F1 model



24-week-old NZB/W F1 mice with grade 2 or higher proteinuria were treated with vehicle or LABP-104 (20 mg/kg) for 12 weeks (n = 10).

Oral LABP-104 treatment protects against weight loss and reduces serum anti-dsDNA antibodies in an NZB/W F1 model. LABP-104 reduces proteinuria grade and prevents worsening of proteinuria from baseline in 90% of mice.





Single Ascending Dose

• Monitoring of safety laboratory results, vital signs, AEs, and ECG

Assessment of LABP-104 transcriptional response profile in whole blood •





Landos is well-positioned to develop a franchise of new oral, first-in-class therapeutics for autoimmune diseases



Innovative LANCE[®] Advanced A.I. platform yielding novel targets (LANCL2, NLRX1, PLXDC2), biomarkers and lead product candidates with strong intellectual property foundation (>122 patents)



Extensive animal pharmacology, mechanism of action, toxicology, benign safety profile and human translational and clinical data on lead candidates (Phase 3-ready omilancor, NX-13 and LABP-104)



Cleared six INDs since inception and filed four new INDs in 2021 for new product candidates



Committed leadership team with autoimmune disease and biopharma industry experience effectively executing 7 clinical development plans



Strong financial position with cash and equivalents of \$115 million and up to \$200M in milestone payments committed by LianBio in China territory deal

LANDOS BIOPHARMA

THANK YOU | WWW.LANDOSBIOPHARMA.COM jbr@landosbiopharma.com



Omilancor (BT-11) & LANCL2 Publications

Leber, A., Hontecillas, R., Zoccoli-Rodriguez, V., Colombel, J-F., Chauhan, J., Ehrich, M., Farinola, N., Bassaganya-Riera, J. Safety, tolerability and PK of BT-11, an oral, gut-restricted LANCL2 agonist investigational new drug for IBD: A randomized, double-blind, placebo-controlled Phase 1 clinical trial, Inflammatory Bowel Diseases 2019. PMID: 31077582

Leber, A., Hontecillas, R., Zoccoli-Rodriguez, V., Chauhan, J., Bassaganya-Riera, J. Oral treatment with BT-11 ameliorates IBD by enhancing Treg responses in the gut. J Immunol, 2019. PMID: 30760618 DOI: <u>10.4049/jimmunol.1801446</u>

Leber, A., Hontecillas, R., Zoccoli-Rodriguez, V., Ehrich, M., Davis, J., Chauhan, J., Bassaganya-Riera, J. Nonclinical toxicology and toxicokinetic profile of an oral LANCL2 agonist, BT-11. Int J Tox, 2019. PMID: 30791754 DOI: 10.1177/1091581819827509

Leber A., Hontecillas R. Zoccoli-Rodriguez V, Bassaganya-Riera J. Activation of LANCL2 by BT-11 Ameliorates IBD by Supporting Regulatory T Cell Stability through Immunometabolic Mechanisms. Inflammatory Bowel Diseases. 2018 24:1978-1991. PMID: 29718324 PMCID: <u>PMC6241665</u>

Carbo A., Gandour RD, Hontecillas R, Philipson N, Uren A, Bassaganya-Riera J. *An N,N Bis(benzimidazolylpicolinoyl)piperazine (BT-11): A Novel LANCL2-Based Therapeutic for Inflammatory Bowel Disease*. J Med Chem. 2016 Nov 23;59(22):10113- 10126. PubMed PMID: 27933891.

Bissel P., Boes K, Hinckley J, Jortner BS, Magnin-Bissel G, Were SR, Ehrich M, Carbo A, Philipson C, Hontecillas R, Philipson N, Gandour RD, Bassaganya-Riera J. *Exploratory Studies With BT-11: A Proposed Orally Active Therapeutic for Crohn's Disease*. Int J Toxicol. 2016 Sep;35(5):521-9. doi: 10.1177/1091581816646356. PubMed PMID: 27230993; PubMed Central PMCID: <u>PMC5033715</u>.



NX-13 & NLRX1 Publications

Leber, A., Hontecillas, R., Zoccoli-Rodriguez, V., Bienert, C., Chauhan, J. and Bassaganya-Riera, J. Activation of NLRX1 by NX-13 ameliorates IBD through immunometabolic mechanisms in CD4+ T cells. J Immunol, PMID: 31694910

Leber, A., Hontecillas, R., Zoccoli-Rodriguez, V., Ehrich, M., Chauhan, J. and Bassaganya-Riera, J. Exploratory Studies with NX-13: Oral toxicity and pharmacokinetics in rodents of an orally active, gut-restricted first-in-class therapeutic for IBD that targets NLRX1. Drug and Chemical Toxicology, PMID: 31650868

Leber, A., Hontecillas, R., Tubau-Juni, N., Zoccoli-Rodriguez, V., Abedi, V., and Bassaganya-Riera, J. NLRX1 Modulates Immunometabolic Mechanisms Controlling the Host-Gut Microbiota Interactions during Inflammatory Bowel Disease. Front Immunol, 2018. PMID: 29535731

Leber, A., Hontecillas, R., Tubau-Juni, N., Zoccoli-Rodriguez, V., Hulver, M., McMillan, R., Eden, K., Allen, IC., and Bassaganya-Riera, J. NLRX1 regulates effector and metabolic functions of CD4+ T cells. J Immunol, 2017 PMID: 28159898