JUNE 2023

Chemomab

THERAPEUTICS

Targeting a Novel Approach to Fibro-inflammatory Diseases

Matthew Frankel, MD
CMO

Forward Looking Statements

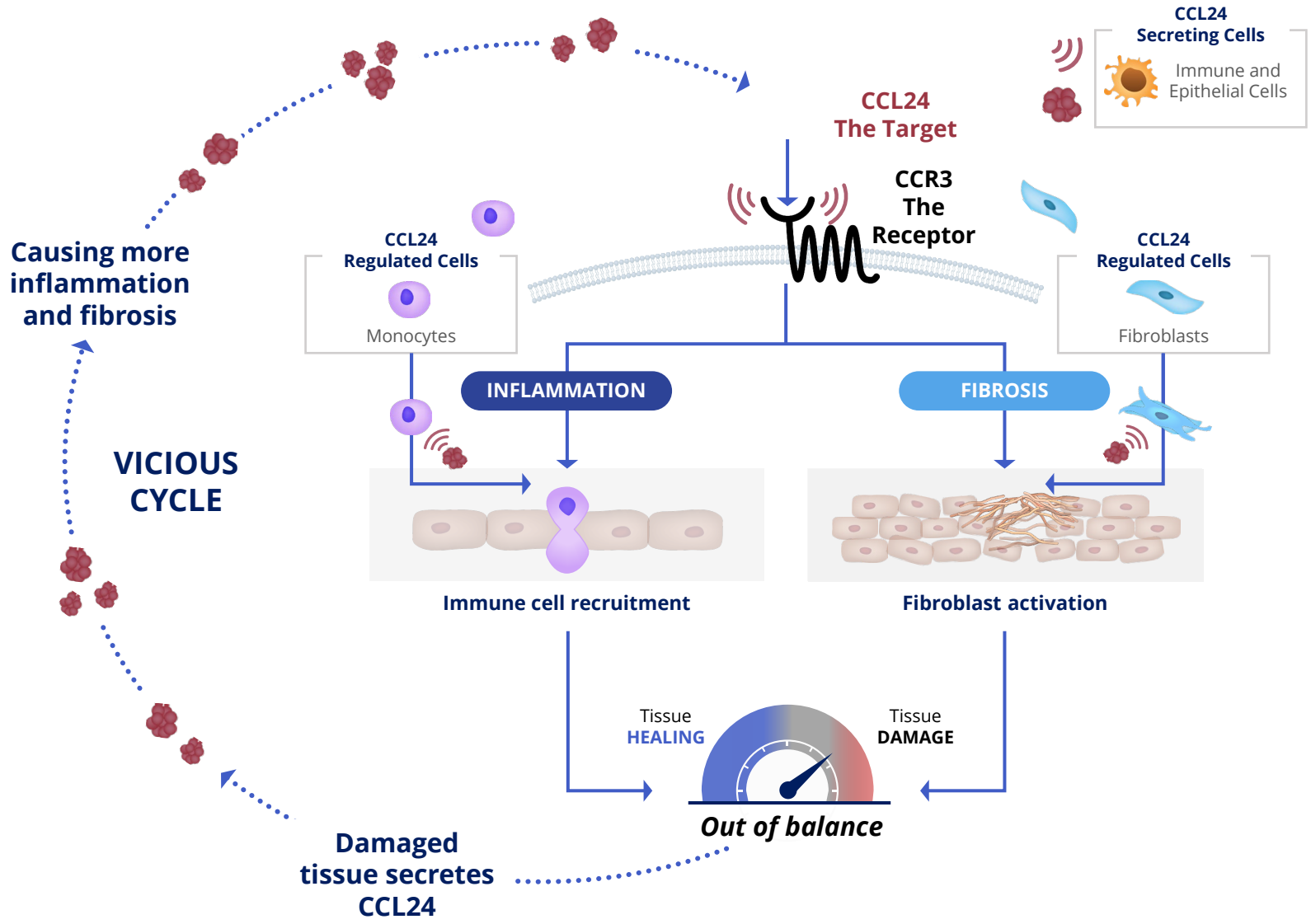
This presentation contains forward-looking statements that involve substantial risks and uncertainties. All statements other than statements of historical facts contained in this presentation, including statements regarding our future financial condition, results of operations, business strategy and plans, and objectives of management for future operations, as well as statements regarding industry trends, are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as “estimate,” “intend,” “may,” “plan,” “potentially” “will” or the negative of these terms or other similar expressions.

We have based these forward-looking statements largely on our current expectations and projections about future events and trends that we believe may affect our financial condition, results of operations, business strategy and financial needs. These forward-looking statements are subject to a number of risks, uncertainties and assumptions, including, among other things: the clinical development pathway for CM-101; our future operations and our ability to successfully initiate and complete clinical trials and achieve regulatory milestones; the potential benefits of any of our product candidates; the market for our product candidates; our expectations regarding our gross margins, operating income and expenses; our ability to complete the proposed offering on the anticipated terms, or at all; and the anticipated use of the net proceeds from the proposed offering. These risks are not exhaustive. New risk factors emerge from time to time, and it is not possible for our management to predict all risk factors, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in, or implied by, any forward-looking statements. You should not rely upon forward-looking statements as predictions of future events. Although we believe that the expectations reflected in the forward-looking statements are reasonable, we cannot guarantee future results, levels of activity, performance or achievements. Except as required by law, we undertake no obligation to update publicly any forward-looking statements for any reason after the date of this presentation.

We have filed a registration statement (including a preliminary prospectus) on Form S-1 (File No. 333-269218), as amended, with the U.S. Securities and Exchange Commission (SEC) for the offering to which this presentation and the accompanying oral commentary relate. Before you invest, you should read the preliminary prospectus and the other documents we have filed and will file with the SEC for more complete information about us and this offering. You may get these documents for free by visiting EDGAR on the SEC website at www.sec.gov. Alternatively, the issuer, any underwriter or any dealer participating in the offering will arrange to send you the preliminary prospectus if you request it by contacting Oppenheimer & Co. Inc., Attention: Syndicate Prospectus Department, 85 Broad St., 26th Floor, New York, NY 10004, by telephone at (212) 667-8055 or by email at EquityProspectus@opco.com.

This presentation shall not constitute an offer to sell or the solicitation of an offer to buy these securities, nor shall there be any sale of these securities in any state or jurisdiction in which such offer, solicitation, or sale would be unlawful prior to registration or qualification under the securities law of any such state or jurisdiction.

CCL24's Role in the Vicious Cycle of Inflammation and Fibrosis*



THE POWER OF CCL24

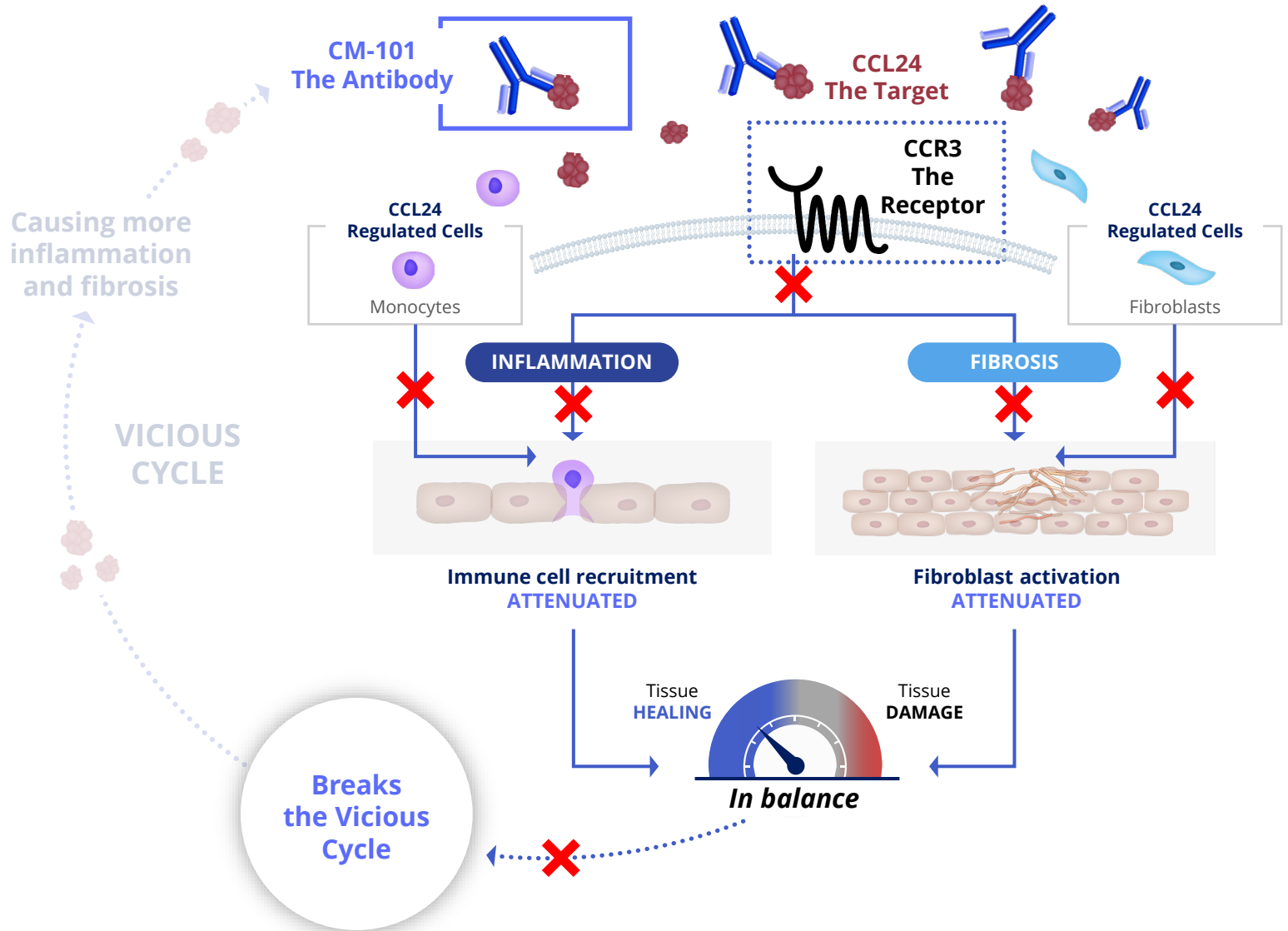
Dual Role in Driving Fibrosis & Inflammation

Differentiated Activity

Low in Healthy Tissue; Elevated in Fibrotic Tissue

*Based on Chemomab preclinical *in vivo* & *in vitro* data-see Endnote 1

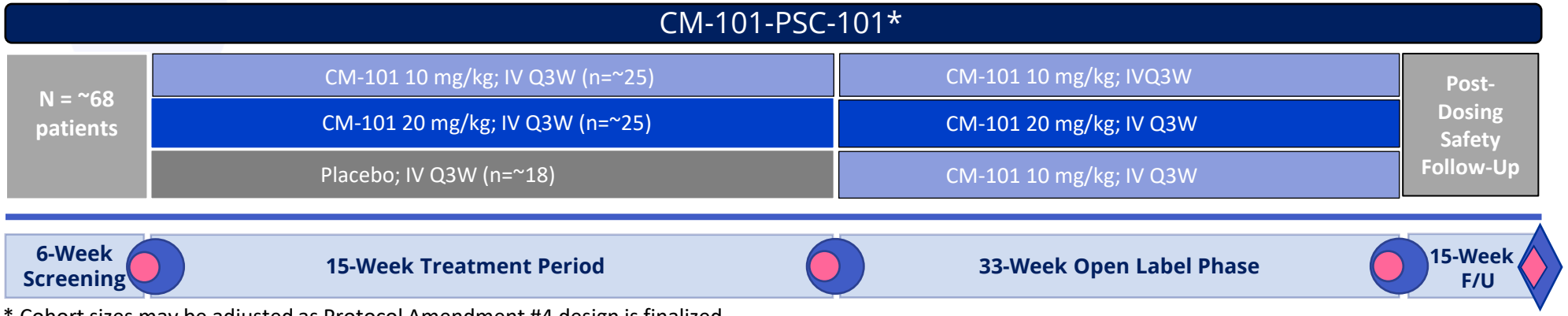
CM-101 Neutralizes CCL24–Aiming to Break Vicious Cycle*



*Based on Chemomab preclinical *in vivo* & *in vitro* data-see Endnote 1

CM-101 Phase 2 Trial in Primary Sclerosing Cholangitis

RANDOMIZED, DOUBLE-BLIND, PLACEBO-CONTROLLED
ENHANCED BY ADDING PATIENTS, SITES, DOSE-FINDING, OPEN LABEL EXTENSION



* Cohort sizes may be adjusted as Protocol Amendment #4 design is finalized

- **Territories**: US, UK, Germany, Spain, Israel
- Orphan Drug Designation in US & EU

Key Enrollment Criteria

PSC patients with large duct disease of >24 weeks duration
- ALP > 1.5 ULN
- Stable IBD allowed including biologic therapy
- Stable UDCA treatment allowed

Outcome Measures

Primary - Safety
Secondary - Change from baseline to Week15 in:
- Serum alkaline phosphatase
- ELF score
- Transient elastography
- Fibrotic biomarkers/liver enzymes (e.g., AST, ALT, Pro-C3, Pro-C5), FibroScan
- Pharmacokinetics
- Pharmacodynamic parameters

Top-line data expected 2H 2024

PSC-primary sclerosing cholangitis; Q3W-once every 3 weeks; IV-intravenous; ALP-alkaline phosphatase; IBD-inflammatory bowel disease; UDCA-ursodeoxycholic acid; ELF–enhanced liver fibrosis score; AST-aspartate aminotransferase; Pro-C3-type lll collagen biomarker; Pro-C5-type V collagen biomarker; FibroScan-measure of liver stiffness/fibrosis

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THERAPEUTICS

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This presentation may include forward-looking statements regarding Pliant's proprietary drug candidate, the timing of the start and conclusion of one or more ongoing or planned clinical trials, including the timing of, and our ability to achieve, anticipated milestones, the sufficiency of our cash, cash equivalents and short-term investments, the timing and outcome of regulatory decisions, future availability of clinical trial data, our collaborations for our product candidates and the maintenance of those collaborations; business and results from operations; and other matters. Actual results could differ materially from those contained in any forward-looking statements as a result of various factors, including without limitation: that Pliant's drug candidate does not advance in development or result in approved products on a timely or cost effective basis or at all; the cost, timing and results of clinical trials; our ability to manage and mitigate the impact of the ongoing COVID-19 pandemic; that many drug candidates that have completed early-stage trials do not become approved drugs on a timely or cost effective basis or at all; the ability to enroll patients in clinical trials; possible safety and efficacy concerns; regulatory developments; the ability of Pliant to protect its intellectual property rights, and unexpected costs, charges or expenses that reduce cash runway. Pliant's pipeline programs are in various stages of pre-clinical and clinical development, and the process by which such pre-clinical or clinical therapeutic candidates could potentially lead to an approved therapeutic is long and subject to significant risks and uncertainties. Pliant undertakes no obligation to update forward-looking statements as a result of new information or otherwise. For a discussion of these and other risks and uncertainties, and other important factors, any of which could cause our actual results to differ from those contained in the forward-looking statements, see the section entitled "Risk Factors" and elsewhere in the Company's most recent Annual Report on Form 10-K and Quarterly Report on Form 10-Q on file with the Securities and Exchange Commission (the "SEC") and our other filings with the SEC.

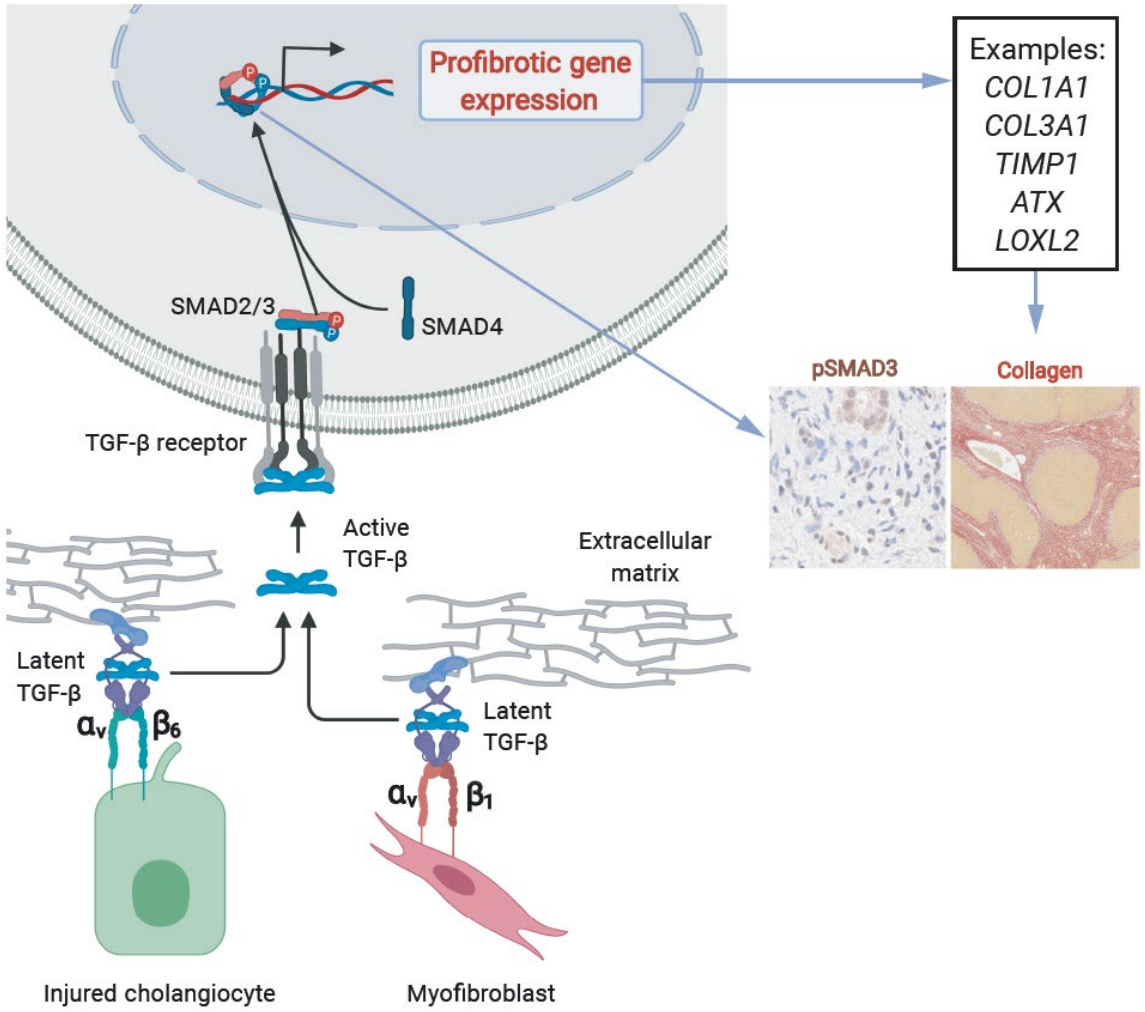
This presentation concerns a drug that is under clinical investigation and which has not yet been approved for marketing by the U.S. Food and Drug Administration (the "FDA"). It is currently limited by Federal law to investigational use, and no representation is made as to its safety or effectiveness for the purposes for which it is being investigated.

Disclosures

- Eric Lefebvre and Richard Pencek are employees and shareholders of Pliant Therapeutics, Inc.

Roles of αvβ6 and αvβ1 integrins in biliary fibrosis

- Transforming growth factor-beta (TGF-β) signaling activated by αv integrins is a key driver of fibrosis in the liver
- In PSC, integrins overexpressed on injured cholangiocytes (αvβ6) and myofibroblasts (αvβ1) regulate TGF-β activity, and are present at elevated levels in liver tissue with biliary fibrosis
- Protein expression of integrins αvβ6 and αvβ1 was higher in explants from patients with PSC compared with control human liver tissue
- Localized TGF-β inhibition in the fibrotic liver, achieved by targeting αvβ6 and αvβ1 integrins, may provide a novel approach to treating PSC, without affecting systemic TGF-β signaling
- Bexotegrast (PLN-74809) is an oral, once-daily, dual-selective inhibitor of integrins αvβ6 and αvβ1 currently in clinical development for the treatment of PSC and idiopathic pulmonary fibrosis



Henderson NC, Sheppard D. *Biochim Biophys Acta* 2013;1832(7):891–896
Turner S, et al. *Hepatology* 2019;70(S1):794A–795A. Abstract 1308
Popov Y, et al. *J Hepatol* 2008;48(3):453–464
Wang B, et al. *Hepatology* 2007;46(5):1404–1412

Bexotegrast INTEGRIS-PSC – Phase 2a Global Safety-PK-Fibrosis and Cholestasis Biomarker Trial in PSC

Enrollment Complete; 12-Week Data Expected in Third Quarter 2023

KEY INCLUSION/EXCLUSION CRITERIA

- Adults with large duct PSC
- Pre-cirrhotic/no hepatic impairment
- Stratified for UDCA use

Randomization 3:1
(bexotegrast : placebo)

- Placebo (n=21)
- bexotegrast 40 mg (n=21)
- bexotegrast 80 mg (n=21)
- bexotegrast 160 mg (n=21)

PRIMARY AND SECONDARY ENDPOINTS

- Safety, tolerability, PK

EXPLORATORY ENDPOINTS

- Effect on fibrosis biomarkers (e.g., Pro-C3, ELF)
- Change in ALP over 12 weeks
- Imaging

PLIANT

Bexotegrast Phase 2a 320 mg Dose Global Safety-PK-Exploratory Efficacy Trial in PSC

Enrollment Open

Randomization 3:1
(bexotegrast : placebo)

Placebo (n=7)

bexotegrast 320 mg (n=21)

KEY INCLUSION/EXCLUSION CRITERIA

- Adults with large duct PSC
- Pre-cirrhotic
- Stable IBD, if present
- Stratified for UDCA use

PRIMARY AND SECONDARY ENDPOINTS

- Safety, tolerability, PK

EXPLORATORY ENDPOINTS

- Effect on fibrosis biomarkers (e.g., Pro-C3, ELF) at Wks 12 and 24
- Change in ALP at Wks 12 and 24

*Trial will complete once the last trial participant enrolled reaches 24 weeks of treatment

PSC Forum

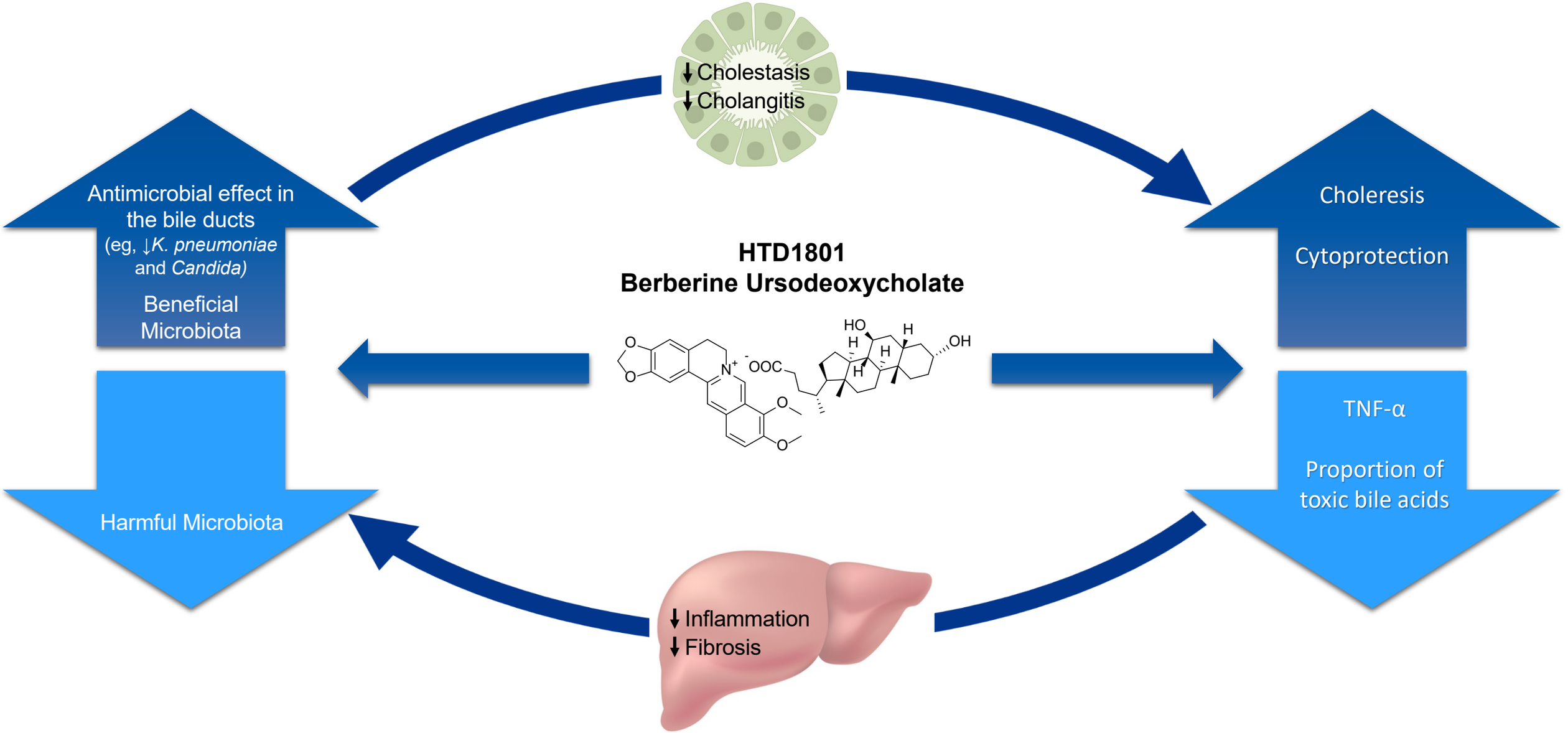
June 20, 2023

HIGHTIDE

Financial Disclosures

- Employment: Hightide Therapeutics

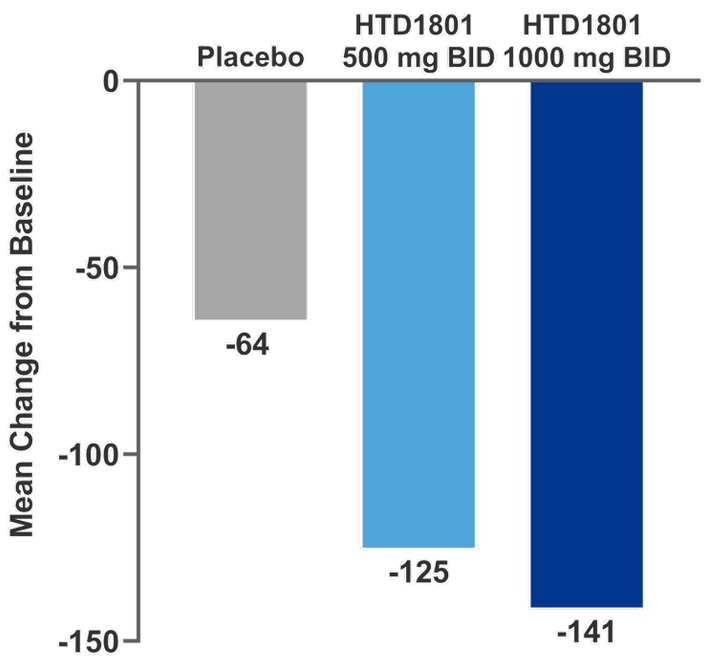
HTD1801 Mechanism of Action in PSC



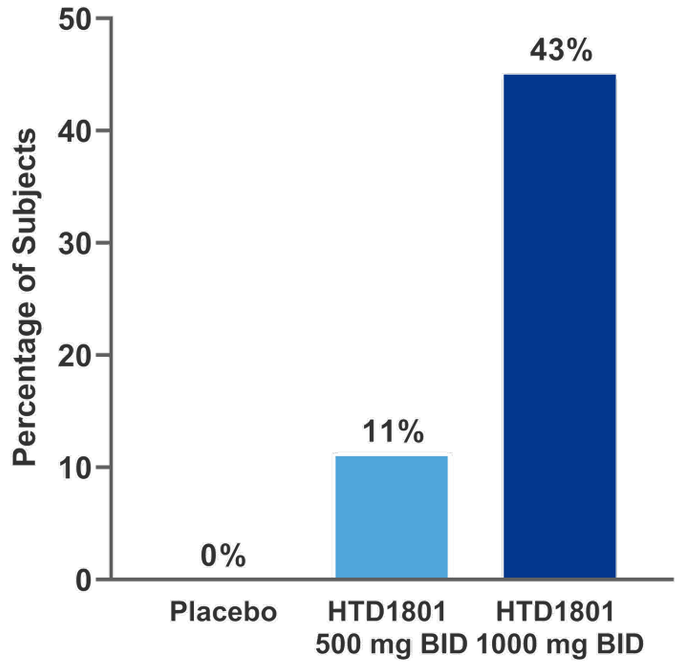
Abbreviations: PSC, Primary Sclerosing Cholangitis; TNF-α, Tumor Necrosis Factor Alpha

Robust Improvements in Key Markers of Cholestasis and Liver Injury in Population Planned for Pivotal Study

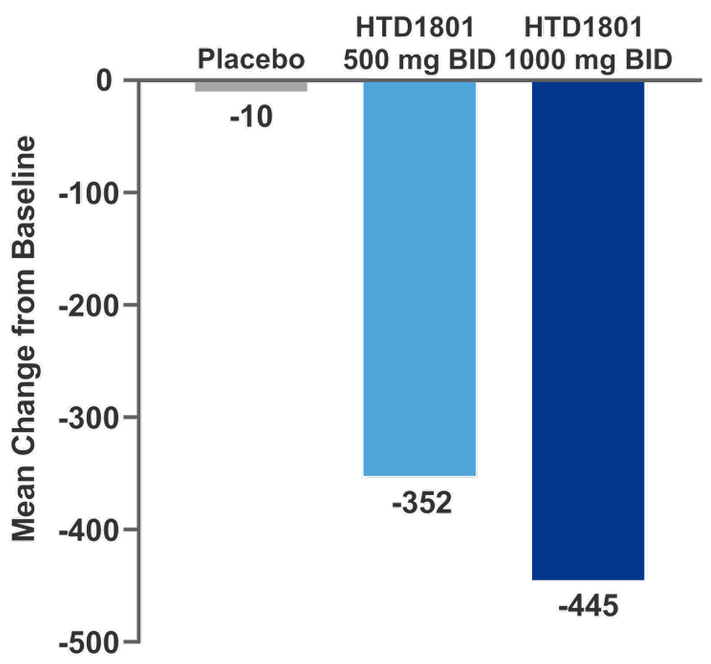
ALP (U/L)



ALP <1.5 xULN



GGT (U/L)



- **Patients that had no prior UDCA in the Medical History**
- **Future studies target population would include UDCA washout or UDCA Naïve patients**

Abbreviations: ALP, Alkaline Phosphatase; ALT, Alanine Aminotransferase; AST, Aspartate Aminotransferase; BID, twice daily dosing; GGT, Gamma-glutamyl Transferase; ULN, upper limit of normal

Clinical Endpoints of Interest for HTD1801 for Later Stage Development

Surrogate Endpoints

- **Biochemistry**
  - Alkaline Phosphatase
  - Enhanced Liver Fibrosis Score
- **Imaging**
  - Transient Elastography
  - MR Elastography
  - MRCP+
- **Liver Function**
  - HepQuant

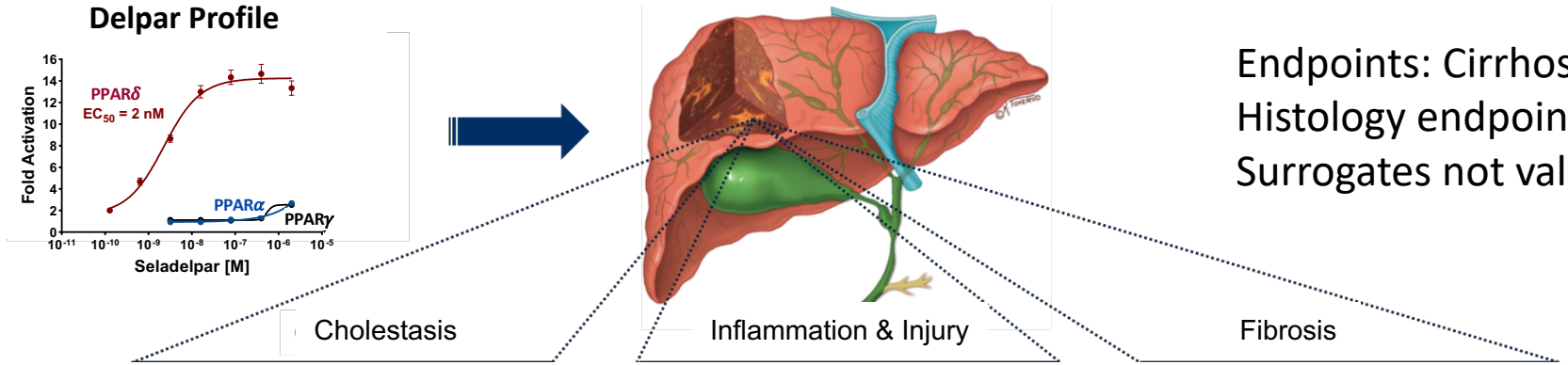
Earlier Stage Clinical Outcomes

- **Biliary Outcomes**
  - Ascending Cholangitis
- **Histologic Outcomes**
  - Progression to Cirrhosis
- **Quality of Life**
  - Pruritus

Later Stage Clinical Outcomes

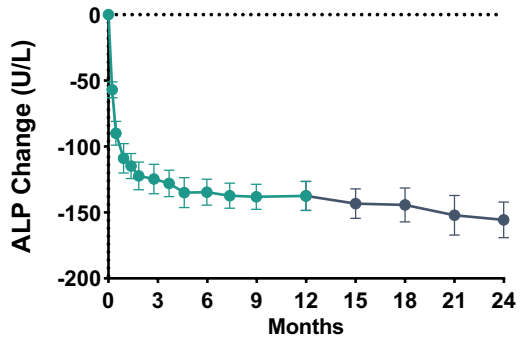
- **Progression to Portal Htn**
- **Complications Associated with Cirrhosis**
- **Progression to Decompensated Cirrhosis**
- **Liver Transplant and Death**

Seladelpar Rationale for PSC: Clues from Action on Cholestasis, Inflammation and Injury in PBC and Fibrosis in NASH

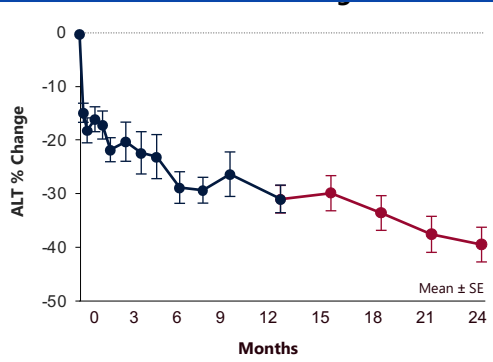


Endpoints: Cirrhosis progression
Histology endpoints are challenging
Surrogates not validated

PBC: ALP Change Over 2 Years

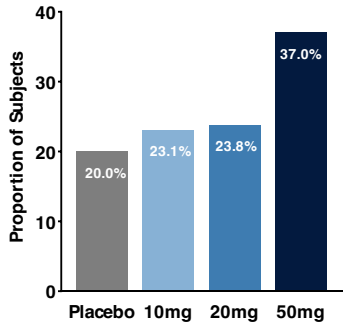


PBC: ALT Change Over 2 Years

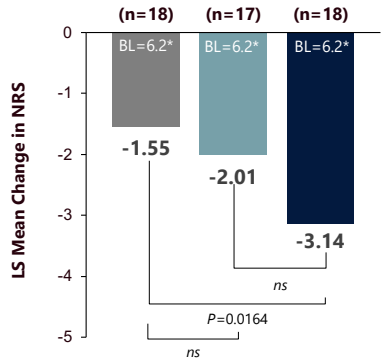


	M0	M3	M6	M9	M12	M15	M18	M21	M24
N	103	101	102	102	102	99	79	65	53

NASH: Fibrosis Improvement at Week 52/ET



PBC: Change in Pruritus Numerical Rating Scale (3 Months)



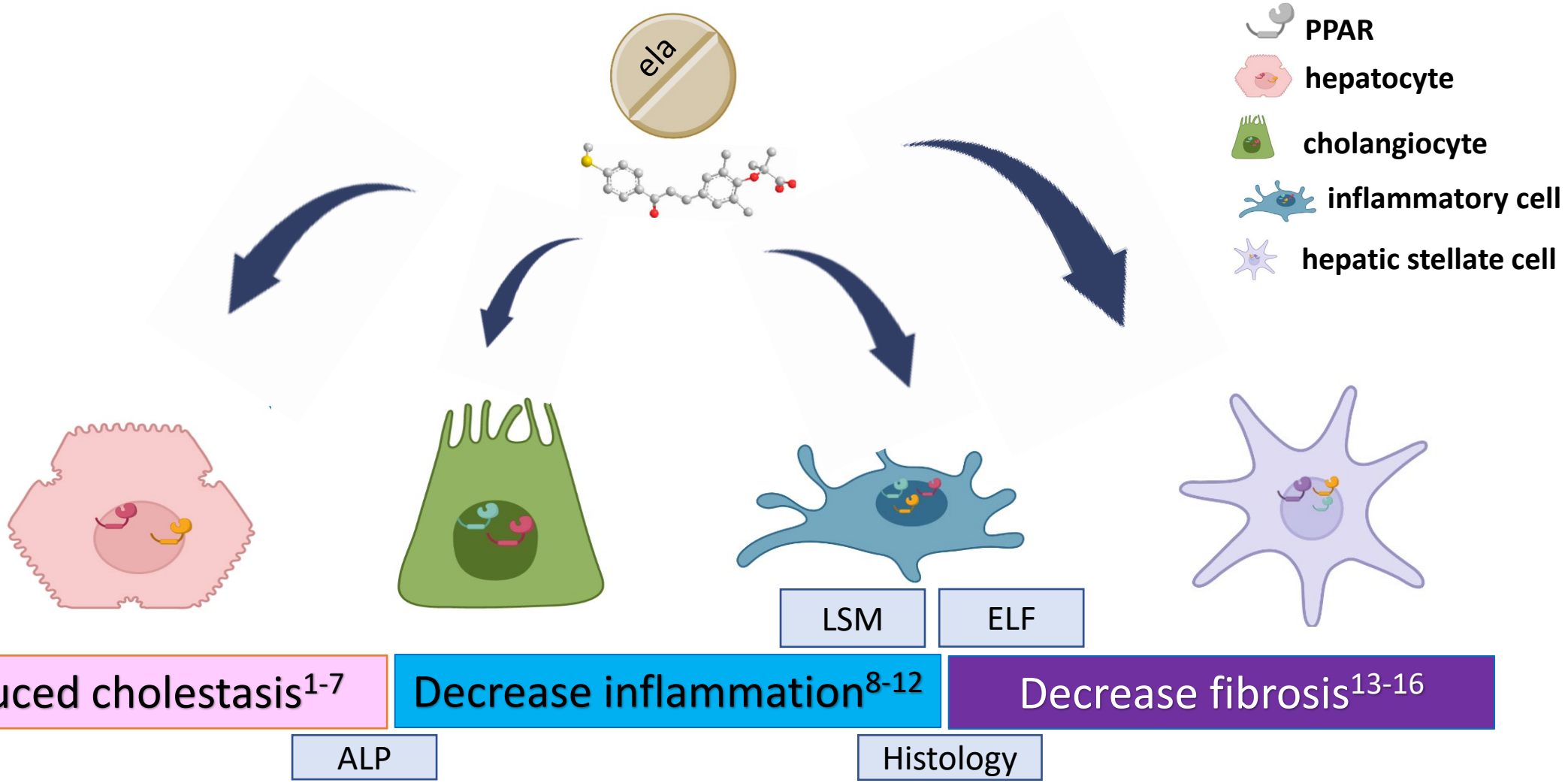
Sources: Mayo M. Presented at AASLD; Nov 12-15, 2021 Harrison S.. Presented at AASLD 2020 Digital
N=103; Baseline ALP Mean (SD) 320U/L (164.7) Hirschfield Heapatology Pre-press 2023

IPSEN
Innovation for patient care

Elafibranor and IPN60250 (formerly A3907) in PSC

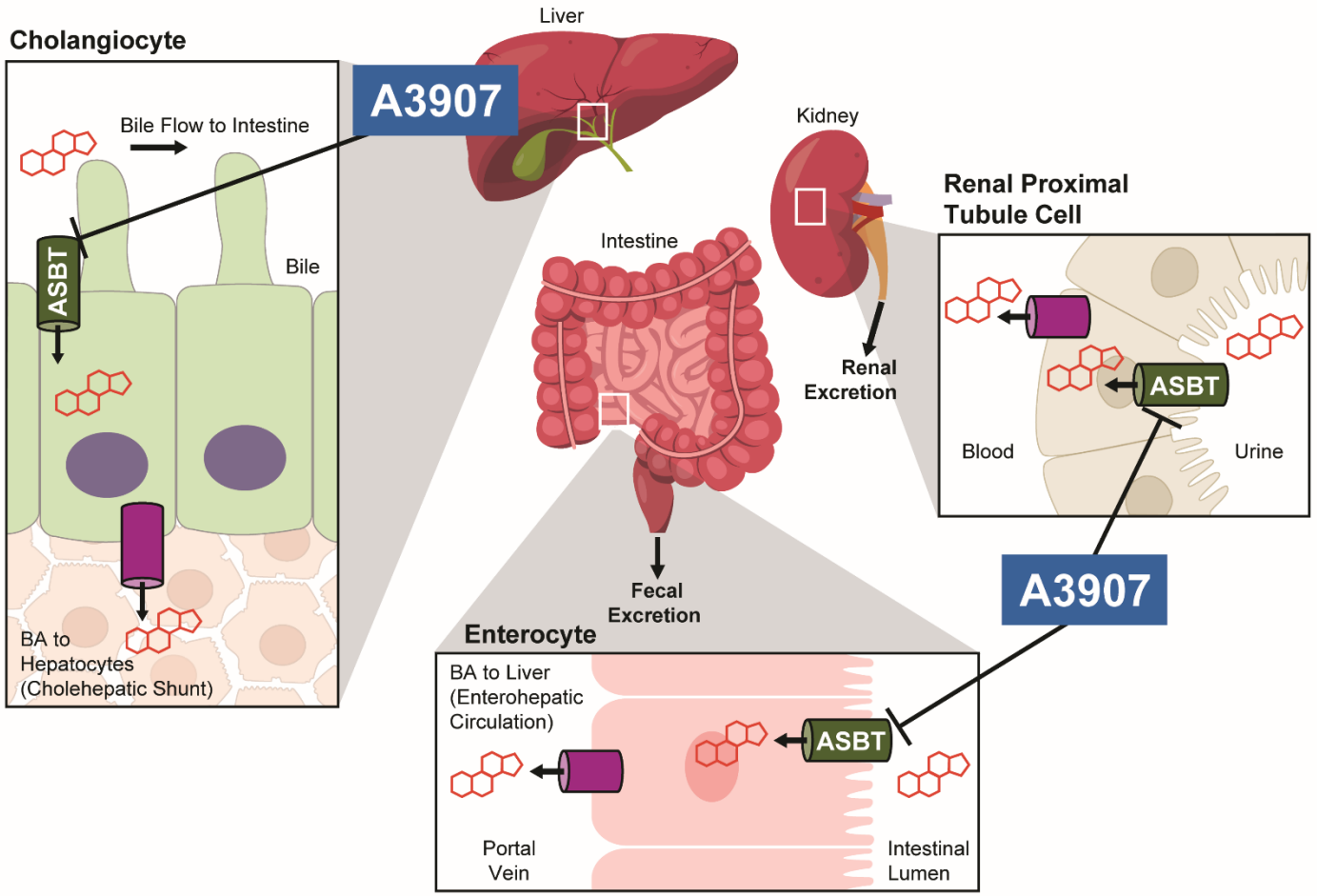
Benjamin Miller, PharmD
VP, Elafibranor Asset Lead

Elafibranor is a dual PPARα,δ agonist being investigated in PSC, a multifactorial disease involving multiple cell types



1. Post SM, et al. Arterioscler Thromb Vasc Biol. 2001;21:1840-45. 2. Ghonem NS, et al. Hepatology. 2015;62:635-43. 3. Xie C, et al. Biochim Biophys Acta Mol Cell Biol Lipids. 2019;1864(10):1396-1411. 4. Zhang Y, et al. Toxicol Sci. 2017;160(2):351-60. 5. Ye X, et al. Front Pharmacol. 2022;13:916866. 6. Jones D, et al. Lancet Gastroenterol Hepatol. 2017;2(10):716-26. 7. Vrins CL, et al. J Lipid Res. 2009;50:2046-54. 8. Delerive P, et al. J Biol Chem. 1999;274(45):32048-54. 9. Chen L, et al. Oncotarget. 2018;9:7204-18. 10. Ricote M, Glass CK. Biochim Biophys Acta. 2007;1771(8):926-35. 11. Delerive P, et al. J Biol Chem. 2000;275(47):36703-7. 12. Coll T, et al. Curr Mol Pharmacol. 2009;2(1):46-55. 13. Leclercq_2014_760A - Poster #1155 - AASLD 2014; 14. Pawlak M, et al. Hepatology. 2014;60(5):1593-606. 15. Guo YC, et al. Toxicol Sci. 2008;105(2):418-28. 16. Gerussi A, et al. Frontiers in Medicine. 2020;7:117.

IPN60250 (formerly A3907) is an oral, systemic inhibitor of bile acid transport being investigated in PSC



- IPN60250 directly targets
  - Cholangiocyte ASBT
  - Intestinal ASBT
  - Renal ASBT
- Expected effect on bile duct inflammation and fibrosis due to direct MoA
- Efficacious in models of biliary duct obstruction (potential for PSC & PBC)
- Potential for efficacy without dose-limiting diarrhea that may be treatment limiting with some gut-restricted IBAT inhibitors

(1) Caballero-Camino FJ, et al. Hepatology. 2023; Apr 3. doi: 10.1097/HEP.0000000000000376. Epub ahead of print.

Thoughts on Biomarkers and Endpoints in PSC

Elafibranor, IPN60250 (formerly A3907)

Biomarkers of Interest
Alkaline phosphatase (ALP)
Serum biomarkers of fibrosis • ELF[4, 5] • ProC3[7]
Liver stiffness measurement with TE [5, 6]
Histology

Potential Phase 3 Clinical Trial Endpoints
Combination of improvement of ALP + no progression (or stability) of fibrosis on liver biopsy[1]
Combination of non-invasive biomarkers: • Improvement of ALP + no progression of fibrosis (for example, based on TE) [2, 3] • Improvement of ALP + no evidence of progression of fibrosis based on ELF
Combination of above with additional novel biomarkers
Patient-reported outcomes
Novel score with biomarkers + patient-reported outcomes
Composite of clinical outcomes: All-cause mortality, liver transplant, decompensation, bacterial cholangitis

(1) EMA, 2018, Reflection paper on regulatory requirements for the development of medicinal products for chronic non-infectious liver diseases (PBC, PSC, NASH; (2) Ponsioen E, Lindor KD, Mehta R, Dimick-Santos L. et al. Design and enpoints for clinical trials in PSC, Hepatology 2018;68:1174-1188; (3) EASL Clinical Practice Guidelines on sclerosing cholangitis. J Hepatology 2022;77:761-806; (4) Trivedi et al. Clin Gastroenterology and Hepatology 2021; 19:1248-1257 (5) Muir et al. Hepatology 2019;69(2):684-698; (6) Corpechot et al. Gastroenterology 2014;146:970-979; (6) EASL Guidelines. J Hepatology 2021;75;659-689; (7) Vesterhus M, et al. JHEP Rep. 2020;3(1):100178.

Volixibat in PSC:

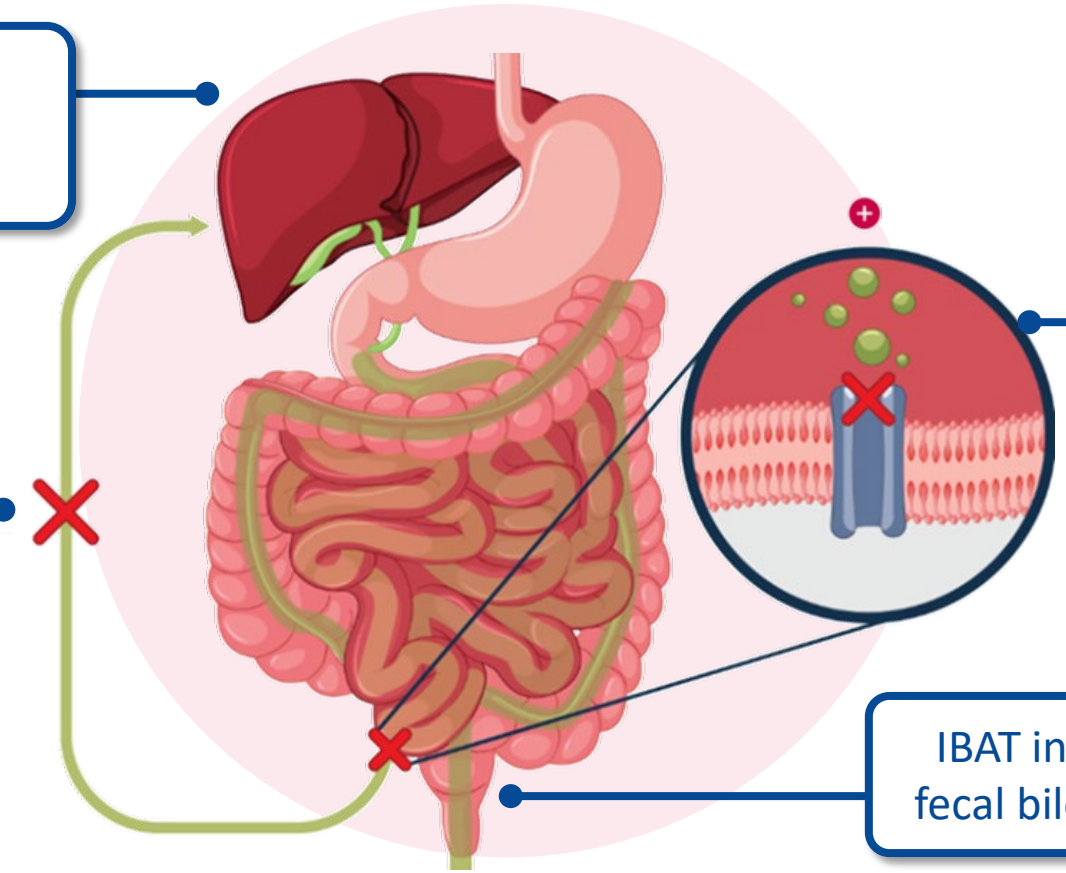
Mechanism of action, biomarker and endpoints of interest

Mirum Pharmaceuticals

Rationale for Volixibat in PSC: an IBAT inhibitor

IBAT inhibitors reduce the amount of bile acid recycled to the liver[1–4]

Interrupting the enterohepatic circulation reduces manifestations of cholestasis[3,5,6]



Use of an IBAT inhibitor has been shown to decrease serum bile acids[5]

IBAT inhibitors increase fecal bile acid excretion[5,6]

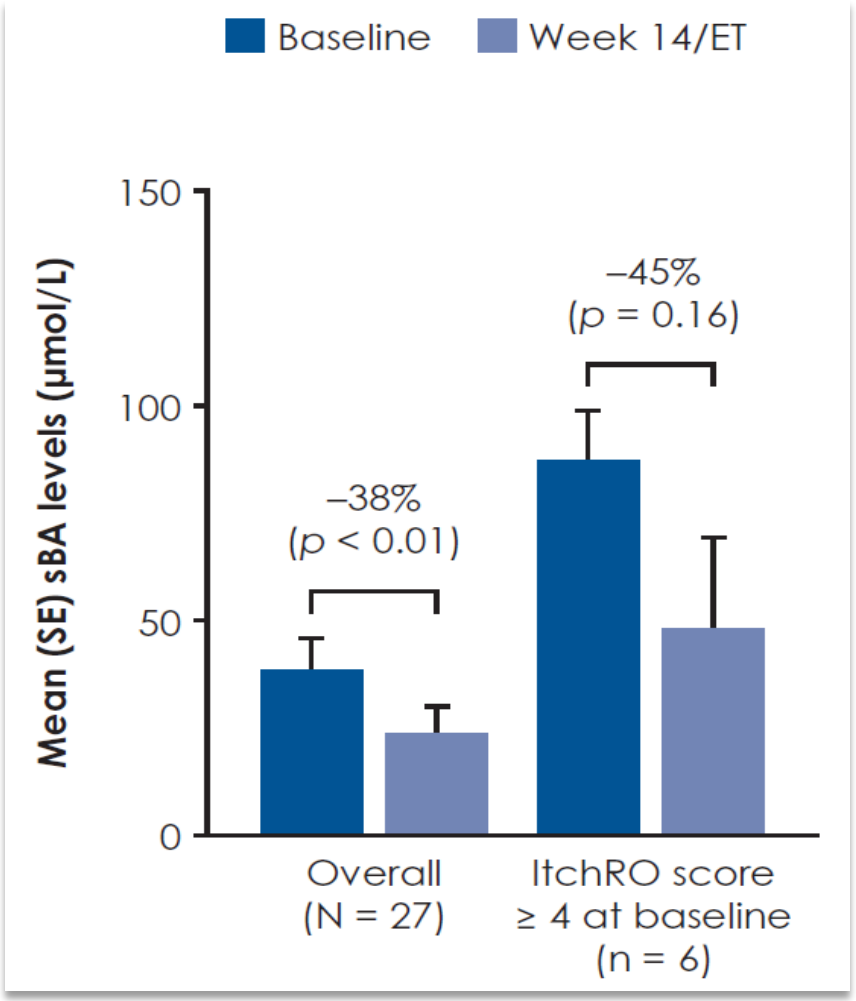
By reducing bile acids, IBAT inhibitors have been shown to improve manifestations of cholestasis in several indications leading to longer event-free survival

IBAT, ileal bile acid transporter; QoL, quality of life.

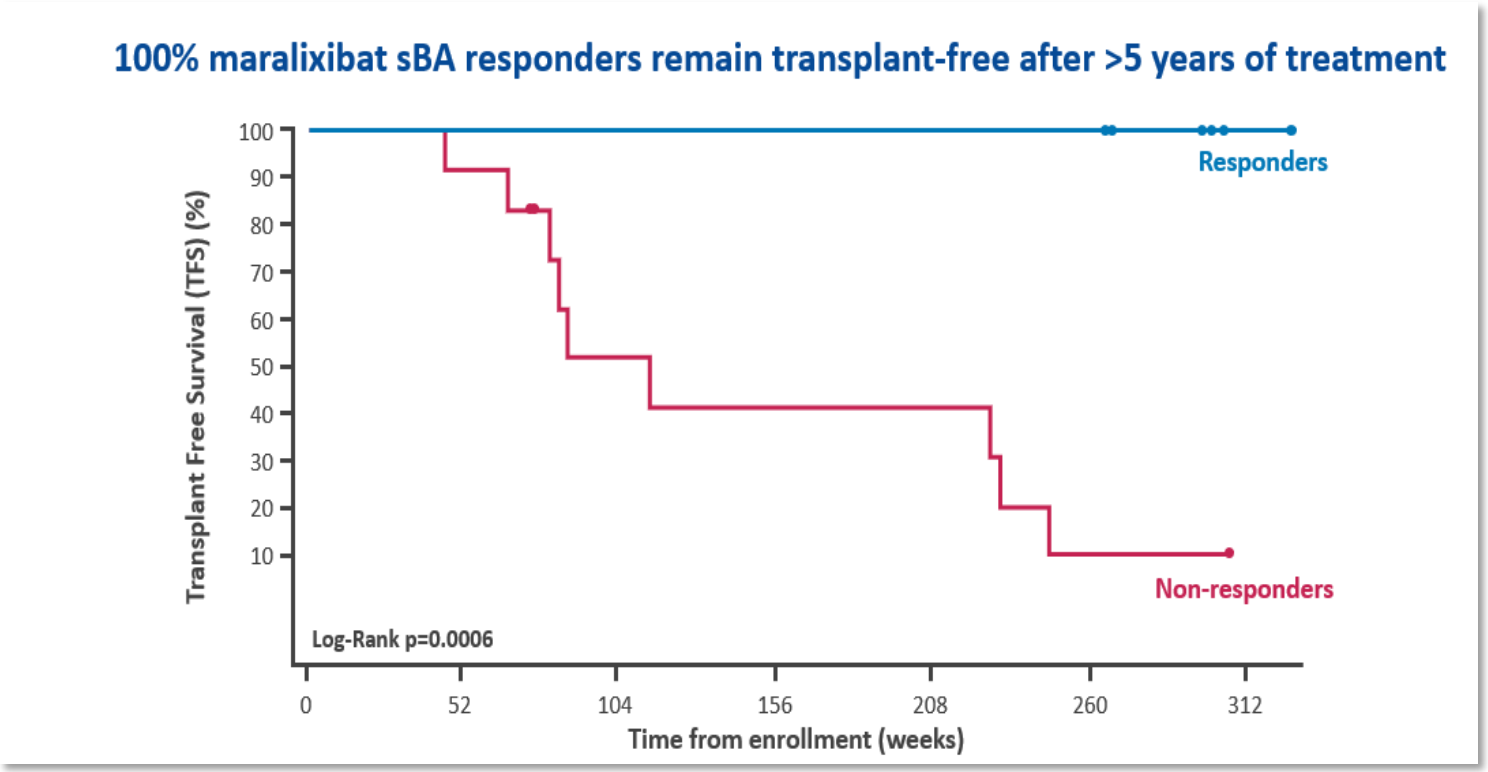
1. Dawson PA. *Handb Exp Pharmacol* 2011; 201:169–203; 2. Miethke AG, *et al. Hepatology* 2016; 63:512–523; 3. Kamath BM, *et al. Liver Int* 2020; 40:1812–1822; 4. Tiessen RG, *et al. BMC Gastroenterol* 2018; 18:3; 5. Hegade VS, *et al. BMC Gastroenterol* 2016; 16:71; 6. Hegade VS, *et al. Therap Adv Gastroenterol* 2016; 9:376–391.

Biomarkers related to IBAT inhibition: sBA is a key efficacy and pharmacodynamic biomarker

IBAT inhibition leads to serum bile acid reduction in PSC patients



IBAT inhibition (response) in PFIC leads to improved TFS



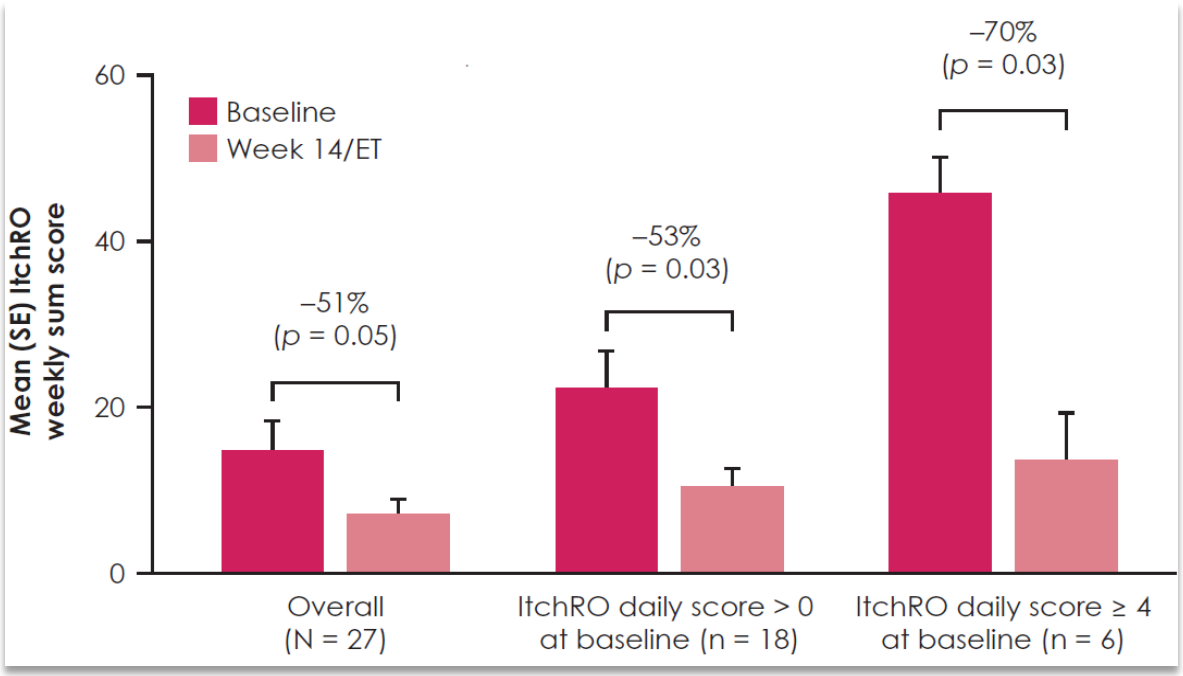
Other biomarkers of IBAT inhibition: sBA subspecies profile, 7aC4 and FGF-19

IBAT, ileal bile acid transporter; PSC, primary sclerosing cholangitis; sBA, serum bile acid; SE, standard error.

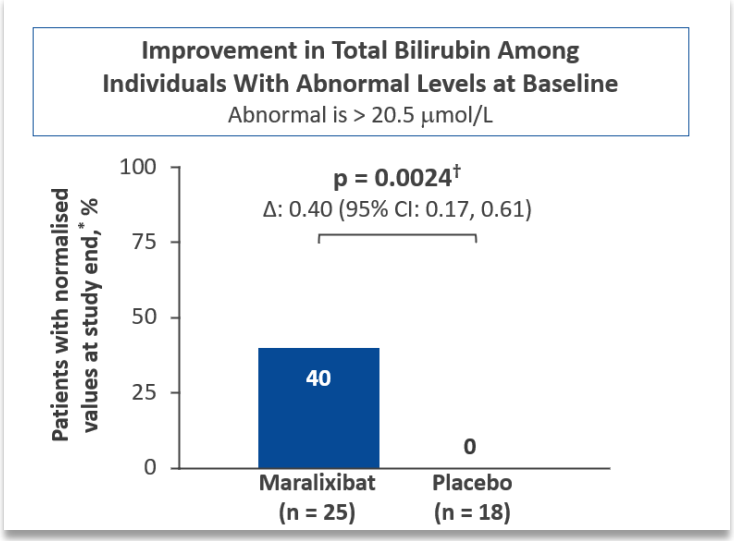
Bowlus, et al. Safety and efficacy of maralixibat in patients with primary sclerosing cholangitis: An open-label proof-of-concept study, The Liver Meeting, AASLD 2019, Boston, MA (Abstract #1262); Hepatology Communications. 2023;7:e0153.

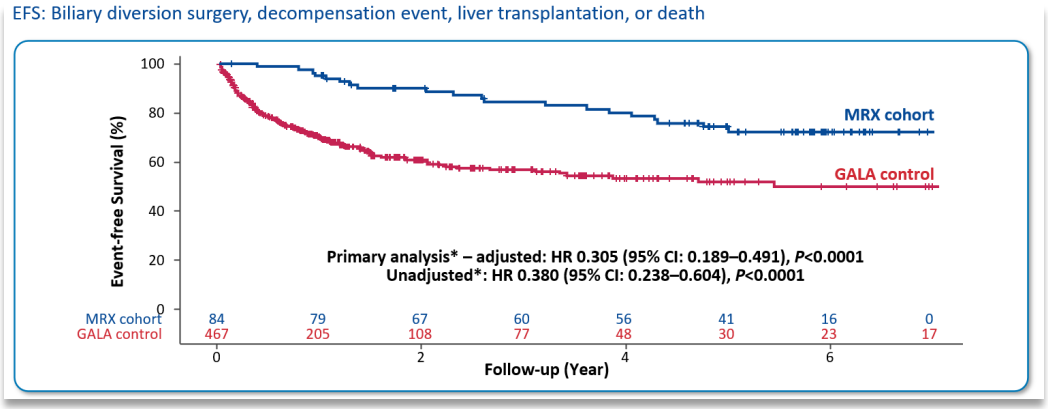
Endpoints related to IBAT inhibition in PSC: pruritus, liver chemistry and event-free survival

IBAT inhibition reduced pruritus in PSC patients



IBAT inhibition normalized bilirubin (PFIC) and led to improvements in EFS (ALGS)



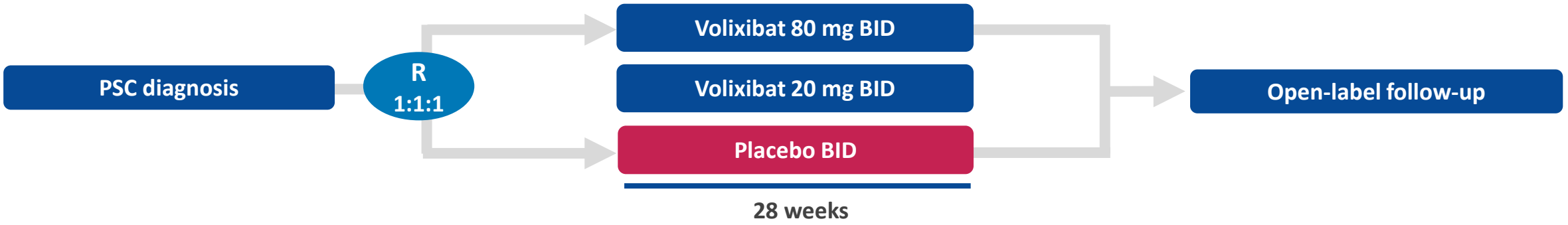


IBAT, ileal bile acid transporter; ItchRO, Adult Itch Reported Outcome (0-10 scale); PSC, primary sclerosing cholangitis; sBA, serum bile acid; SE, standard error.

Bowlus, et al. Safety and efficacy of maralixibat in patients with primary sclerosing cholangitis: An open-label proof-of-concept study, The Liver Meeting, AASLD 2019, Boston, MA (Abstract #1262); Hepatology Communications. 2023;7:e0153.

VISTAS: Phase 2b Clinical Trial of Volixibat in PSC

vistas
EFFICACY & SAFETY OF VOLIXIBAT IN THE TREATMENT OF CHOLESTATIC PRURITUS IN PATIENTS WITH PSC



Primary outcome measure:
Mean change in itch scores

Secondary outcome measures:
- Proportion of participants with itch response
- Changes in fasting serum bile acid levels
- Changes in bilirubin levels
- Changes in alanine transaminase, aspartate transaminase, and alkaline phosphatase
- Change in PSC-specific patient-reported outcome
- Change in Patient-Reported Outcomes Measurement Information System (PROMIS®) fatigue and sleep disturbance questionnaires

BID, twice daily; ItchRO, Itch Reported Outcome; PSC, primary sclerosing cholangitis; R, randomization.