



# Advancing First-in-Class Therapy with Disease-Modifying Potential in Fibro-Inflammatory Diseases

CORPORATE OVERVIEW  
NASDAQ: CMMB

# 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 raise additional funds; and the intensity and duration of the current war in Israel, and its impact on our operations in Israel. 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.

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# Chemomab Highlights: Major Catalyst Expected Midyear 2024



## Novel Mechanism with Disease-Modifying Potential

Unique target central to biology of inflammation & fibrosis  
CM-101: disease-modifying potential in multiple fibro-inflammatory diseases

## Targeting Diseases with Large Commercial Potential

Primary Sclerosing Cholangitis (PSC) is deadly disease with no approved therapies  
Orphan & Fast Track designations; PSC represents more than \$1 billion market opportunity  
Interest from potential partners

## De-risked with Extensive Preclinical & Early Clinical Data




Extensive preclinical validation of mechanism & activity  
CM-101 Phase 2 study confirmed anti-fibrogenic & anti-inflammatory activity  
4 completed clinical trials show safety & consistent positive biomarker responses

## Have Runway to Achieve Major Catalyst Expected Midyear 2024

Cash sufficient to achieve major catalyst--PSC Phase 2 read-out expected Midyear 2024  
Long-term holders include well-known investors: Orbimed, Thiel, Aperia  
No debt; clean capital structure



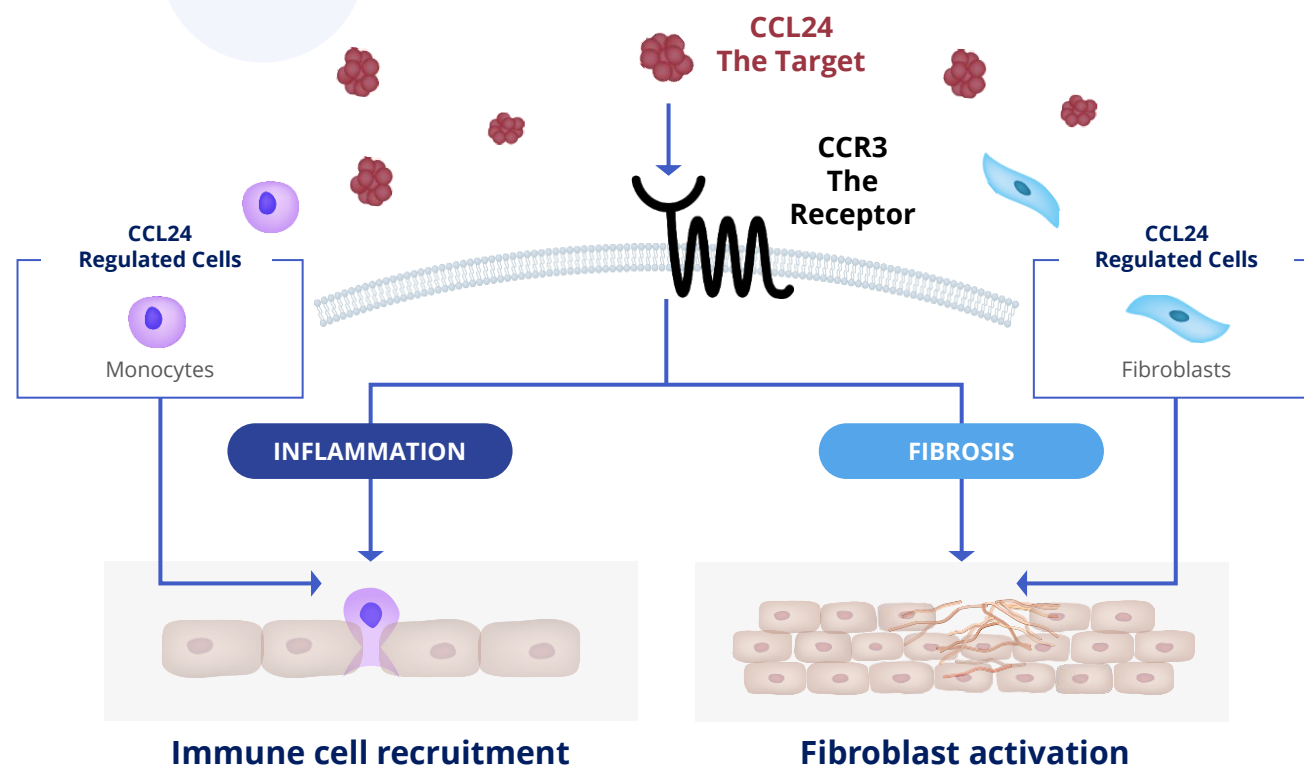
# Pipeline in a Drug: Current Focus on PSC

Agent	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated Milestone	Global Rights
<b>CM-101</b> Anti-CCL24 mAb	Primary Sclerosing Cholangitis (PSC)	Orphan designation from FDA & EMA; FDA Fast Track designation; Patient enrollment completed				Topline Data Expected Midyear 2024	 Chemomab THERAPEUTICS
<b>CM-101</b> Anti-CCL24 mAb	Systemic Sclerosis (SSc)	Orphan designation from FDA & EMA				Phase 2-Ready Open US IND	 Chemomab THERAPEUTICS
<b>CM-101</b> Anti-CCL24 mAb	Liver Fibrosis (in NASH patients)	Proof-of-Concept: Safety & biomarker changes; SC formulation				Completed. Data Reported Q1 2023	 Chemomab THERAPEUTICS

## Four Completed Clinical Studies:

- Phase 1a safety study in healthy volunteers reported in 2018
- 2 Liver fibrosis biomarker trials – Phase 1b in NAFLD and Phase 2a in NASH patients, reported in 2021 and 2023 respectively
- Lung injury investigator-initiated biomarker study in COVID patients reported in 2022

# CCL24's Dual Role in Inflammation and Fibrosis-related Pathology



## THE POWER OF CCL24

### Dual Role in Promoting Fibrosis & Inflammation

Directly activates fibroblasts  
Enhances local immune cell recruitment

### Differentiated Activity

Data shows **unique role** vs. other chemokines  
Correlates **with** fibrotic biomarkers and disease outcome

### Low in Healthy Tissue; Elevated in Fibrotic Tissue

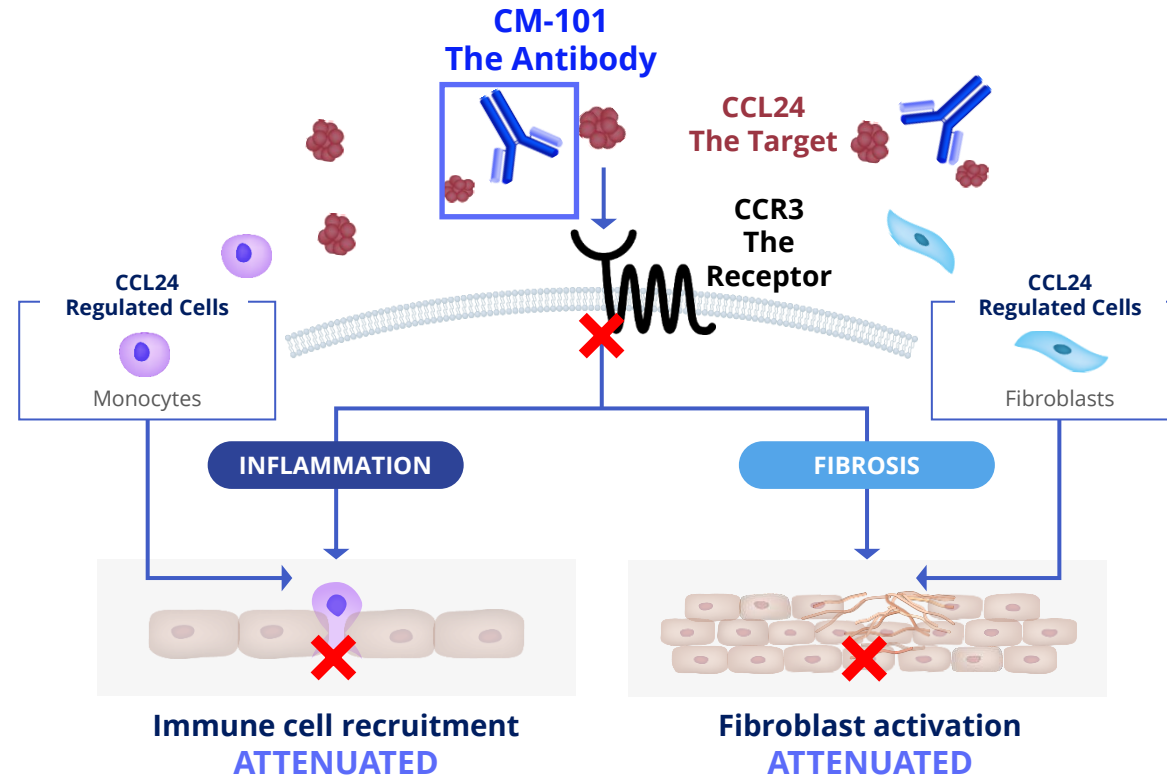
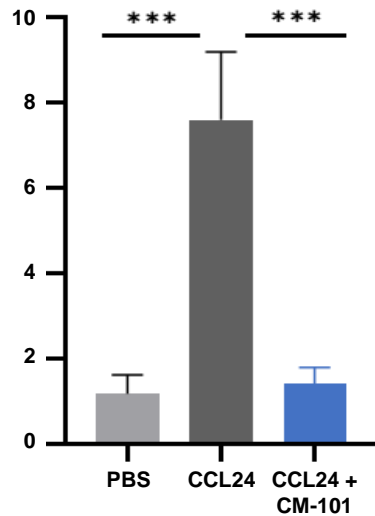
Liver, skin, lung, kidney, others  
Wide therapeutic margin

# CM-101 Reduces Inflammation and Fibrosis by Neutralizing CCL24



## CM-101 Reduces In Vivo Monocyte Recruitment

Number of Migrated Monocytes (x1000)

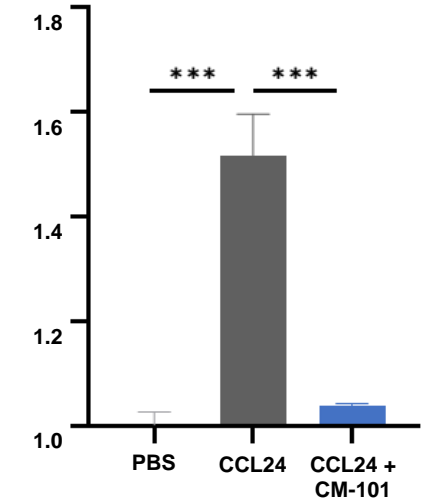


## Neutralizing CCL24 Has Advantages Over Blocking the Receptor

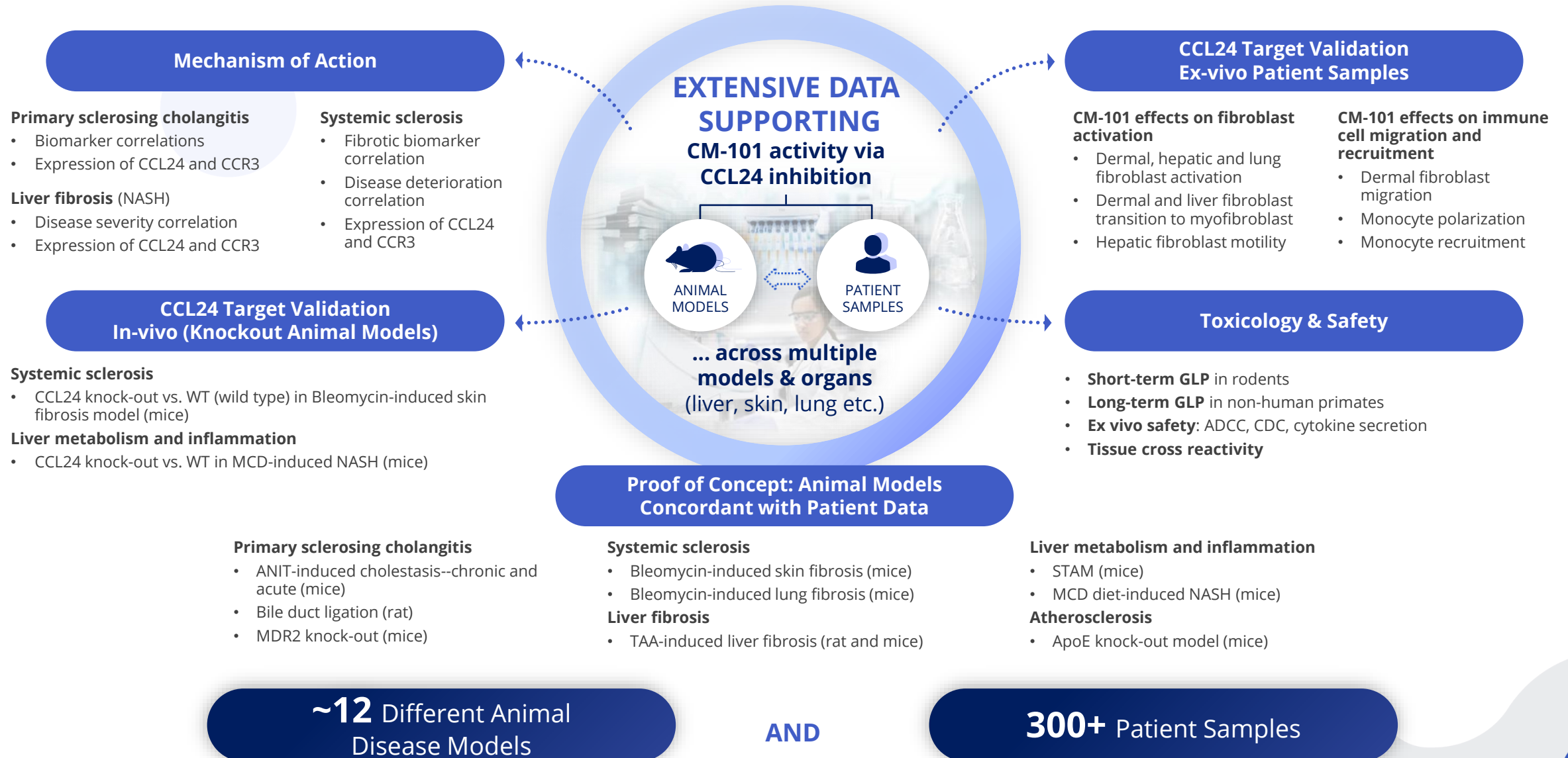
Avoids context dependence of CCR3 receptor  
Efficacy of direct approach  
Favorable safety profile; retains normal repair functions

## CM-101 Inhibits Primary Fibroblast Activation

Proliferation: Relative Change from Baseline



# Broad & Diverse Preclinical Data De-risks Clinical Development



# Anti-Fibrotic Mechanisms Demonstrated Across Clinical Trials



PATIENTS

## CM-101 APPEARED SAFE IN 4 CLINICAL TRIALS IN HEALTHY VOLUNTEERS & IN PATIENTS

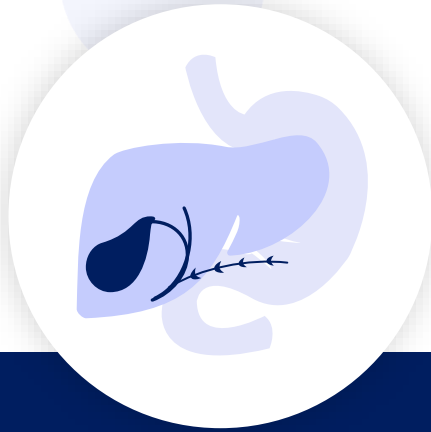
- Safe & well-tolerated in 4 clinical trials
- Adverse Events mostly mild; NO drug-related SAEs
- Consistent PK & target engagement profiles
- No anti-drug antibodies detected

## CM-101 IMPROVED MULTIPLE BIOMARKERS IN 3 CLINICAL TRIALS IN PATIENTS

- Reduced fibrosis-related biomarkers in fatty liver, liver fibrosis, NASH & acute lung inflammation<sup>1</sup>
- Demonstrated anti-inflammatory effects in NASH & acute lung inflammation<sup>1</sup>
- Produced greater response in NASH patients who had greater risk of disease progression
- Improved biomarkers associated with PSC

Clinical trials to date have demonstrated CM-101's safety & its anti-fibrotic & anti-inflammatory effects in varied organs and diseases

<sup>1</sup>-[Treatment with CM-101 Reduced Inflammatory & Fibrotic Biomarkers in Patients with COVID-19-Derived Lung Damage](#), Dr. Adi Mor, Union World Conference on Lung Health, Nov. 9, 2022



## CM-101: Potential Treatment for Primary Sclerosing Cholangitis (PSC)

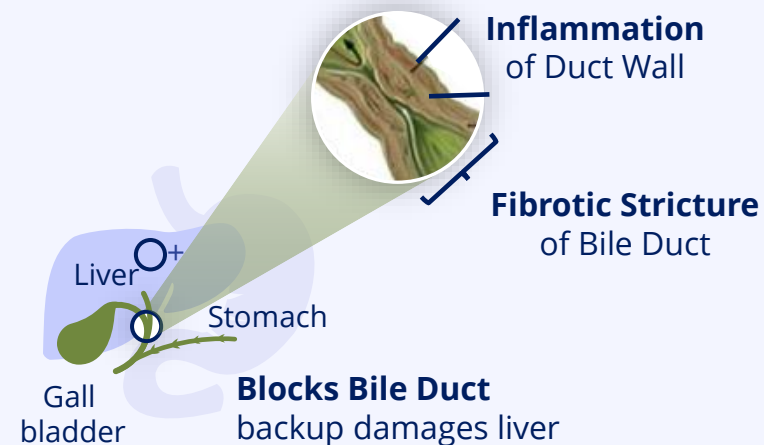
# PSC Has High Unmet Need & Large Commercial Potential



## Debilitating orphan liver disorder with no FDA-approved therapies

- Primarily affects men in their 40's
- Symptoms include fatigue, pruritis, abdominal pain and jaundice
- Diagnosed via serum liver enzyme abnormalities, cholangiography
- Unknown cause; associated with IBD in ~70% of patients
- **50% of patients require liver transplantation;** PSC re-occurs in ~20% of recipients
- **Leads to end-stage liver disease and cancer, which causes half of all deaths**
- Median transplant-free survival is 10-20 years

## Progressive disease characterized by inflammation and fibrosis



## Sizeable Market Opportunity, Orphan & Fast Track Incentives

- ~80,000 PSC patients in 7 major markets: U.S., Europe and Japan
- **Commercial opportunity worldwide estimated at ~\$1 billion**

# Unlike Most PSC Competitors, CM-101's Unique Dual Activity Has Disease-Modifying Potential



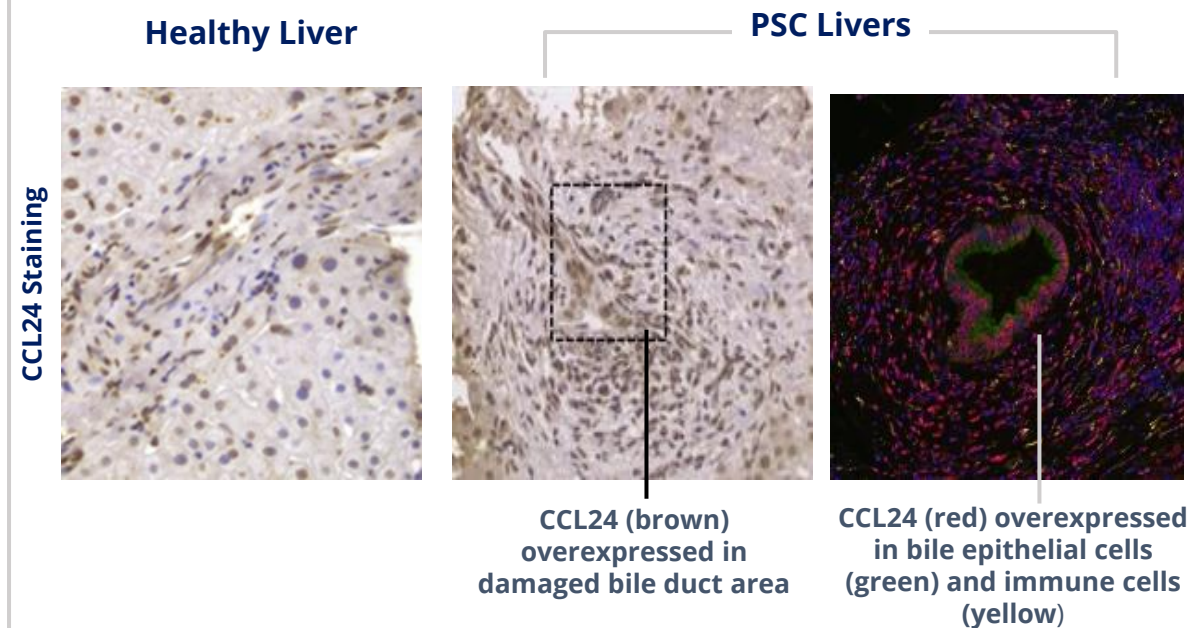
CM-101 is the only therapeutic candidate targeting both inflammation and fibrosis

Company	Candidate	Mechanism	Disease Modifying Potential	Stage
Chemomab	CM-101	Anti-CCL24; Inhibits key inflammatory AND fibrotic processes	✓✓	<b>Phase 2</b> <i>Recruitment completed Midyear 2024 readout</i>
Pliant	PLN-74809 Bexotegrast	Fibrosis-focused selective Integrin inhibitor	✓	<b>Phase 2</b> <i>- Positive initial readout High dose readout 1Q24</i>
Dr Falk	norUDCA	UDCA homologue--metabolic bile acid mechanism	—	<b>Phase 3</b> <i>- Recruiting</i>
Mirum	Volixibat	Ileal bile acid inhibitor--targeting pruritis	—	<b>Phase 2</b> <i>- Interim readout 2H23</i>
Ipsen/Albireo	IPN60250	Ileal bile acid inhibitor--targeting pruritis	—	<b>Phase 2</b> <i>- Recruiting</i>
Ipsen	Elafibranor	PPAR dual agonist—metabolic focus	—	<b>Phase 2</b> <i>-Recruiting</i>

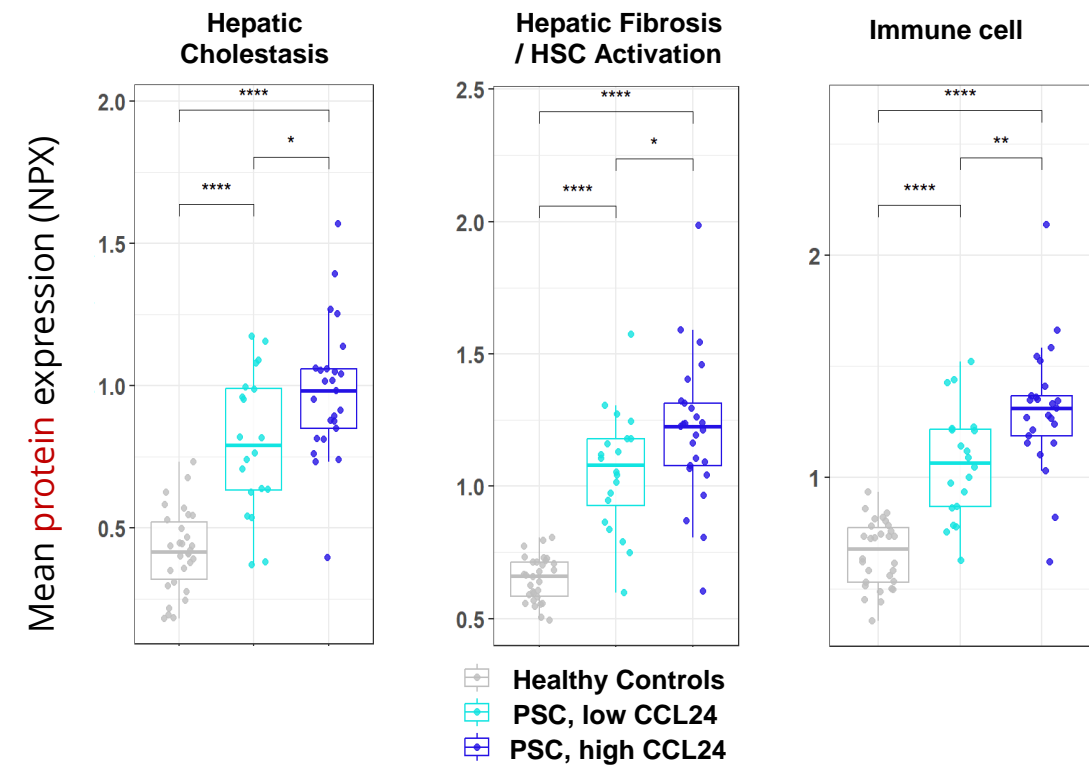
# CCL24 Levels Are Elevated in PSC Patients and Associated with Protein Expression in Key PSC Pathways



## CCL24 LEVELS IN HEALTHY VS PSC PATIENT LIVER TISSUE



## CCL24 LEVELS ARE ASSOCIATED WITH KEY PSC PATHWAYS



CCL24 expression is significantly & selectively elevated in PSC livers  
Elevated serum CCL24 levels in patients are associated with key PSC pathways

# CM-101 Reduces Liver Fibrosis by >80% Preclinically

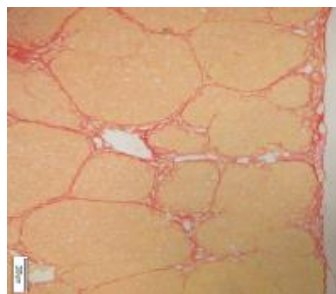
## RAT MODEL LIVERS

### SIRIUS RED (COLLAGEN)

Healthy



TAA  
Fibrosis

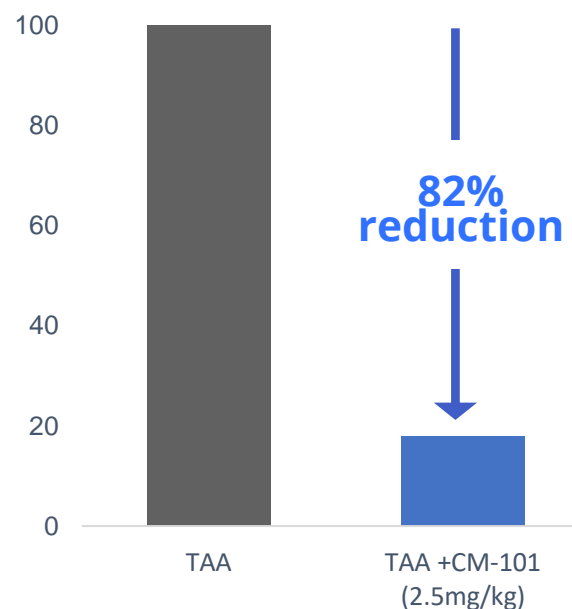


TAA+  
CM-101



## REDUCED LIVER COLLAGEN DEPOSITION

### % FIBROSIS (SIRIUS RED STAINING)



Pro-fibrotic  
Genes

Col1A1  
Col3A1  
TIMP1  
ACTA2  
TGF- $\beta$

Liver  
Enzymes

ALT  
AST  
ALP

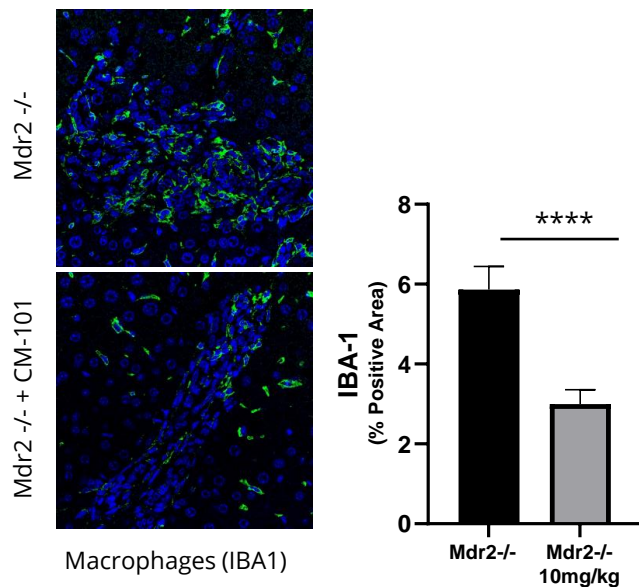
Dramatically reduced liver collagen using therapeutic design  
(treatment starts after fibrosis is established)

# CM-101 Reduces Liver Injury & Fibrosis in Multiple PSC Models



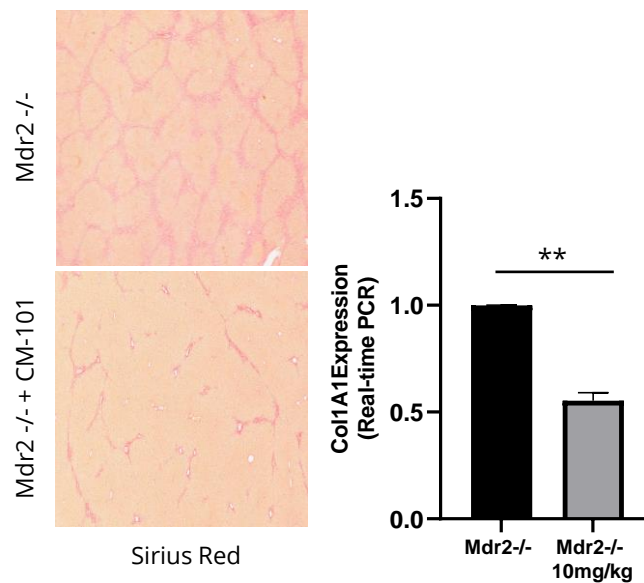
CM-101 DEMONSTRATED SIZEABLE REDUCTIONS IN PSC-RELATED PATHOLOGY\*

## REDUCED MACROPHAGES



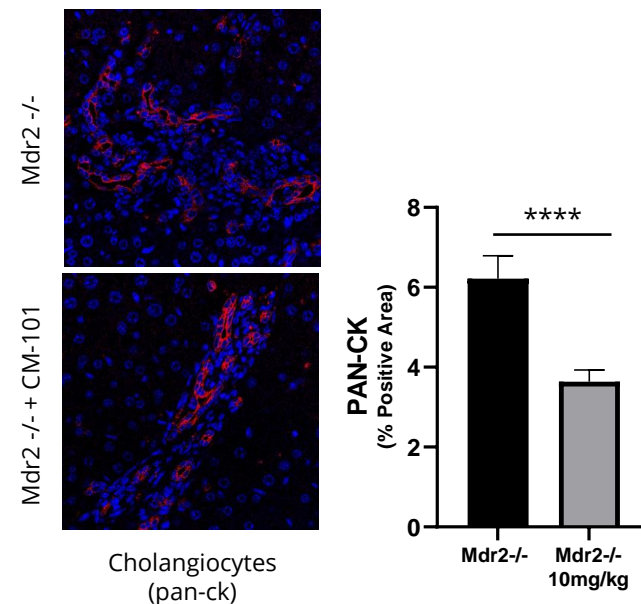
Reduction in  
INFLAMMATION

## ATTENUATED LIVER COLLAGEN DEPOSITION



Reduction in  
FIBROSIS

## REDUCED BILE EPITHELIAL PROLIFERATION



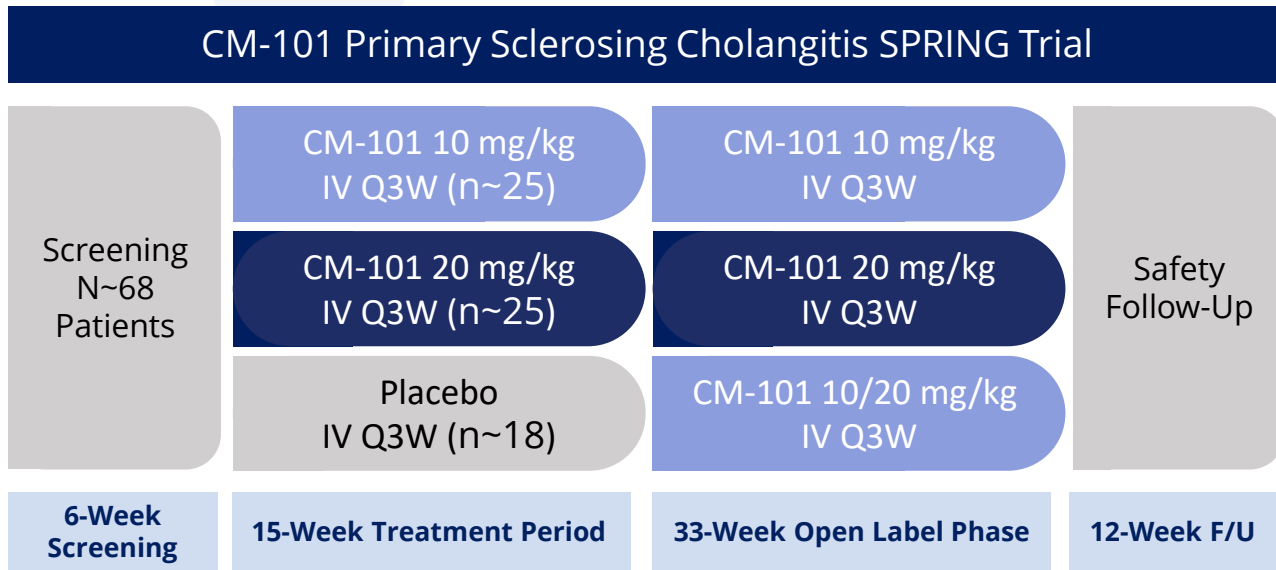
Reduction in main driver of  
DISEASE PATHOLOGY

CM-101 interferes with the 3 major pathways that drive PSC pathology



# Patient Enrollment Completed in CM-101 Phase 2 PSC Trial

RANDOMIZED, DOUBLE-BLIND, PLACEBO-CONTROLLED--INCLUDES DOSE-FINDING & OPEN LABEL EXTENSION



### Key Enrollment Criteria

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

### Outcome Measures

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

- **Territories:** US, UK, Germany, Spain, Israel
- Orphan Drug designations in US & EU
- Fast Track designation in US

Early completion of patient enrollment—Topline data expected Midyear 2024

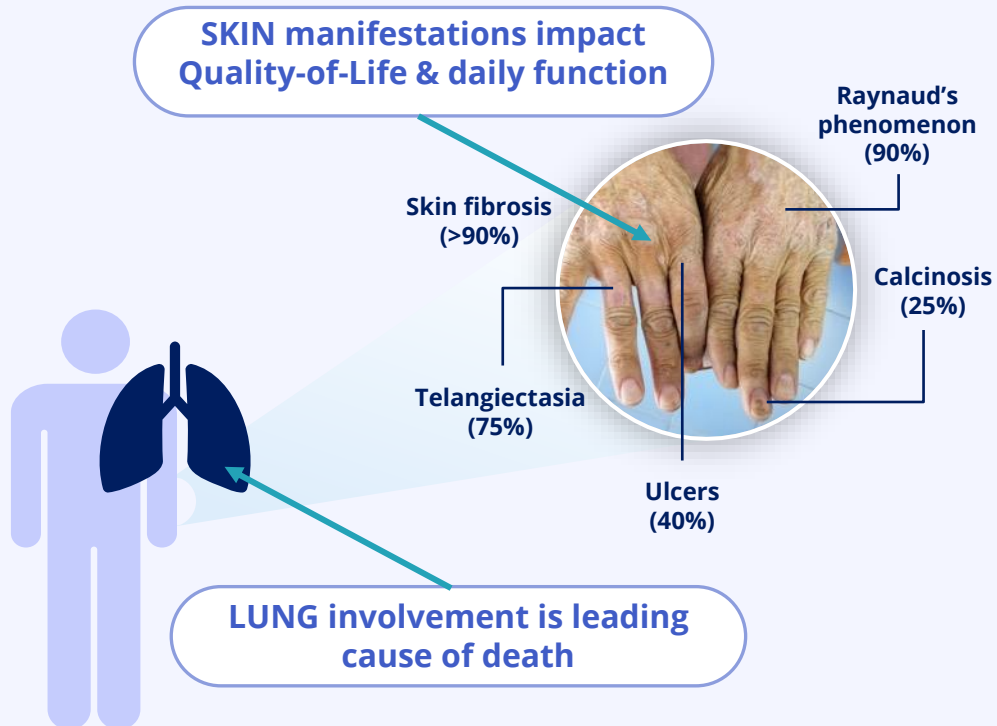


## CM-101: Potential Treatment for Systemic Sclerosis (SSc) *Phase 2-Ready*

# Systemic Sclerosis (SSc): Most Lethal Systemic Rheumatic Disease



DEVASTATING ORPHAN DISEASE CAUSED BY  
INFLAMMATION, FIBROSIS & VASCULOPATHY



RARE AUTOIMMUNE RHEUMATIC DISEASE  
NO DISEASE MODIFYING THERAPY

Median Survival: 10 years

Diagnosis: between 30-50 years

Population: 3:1 female/male

Current Rx: nintedanib & tocilizumab FDA-  
approved but only treat pulmonary symptoms;  
**NOT** disease modifying

**~170K**  
**patients**  
in 7 major markets  
**>\$1.5B**  
est. commercial  
opportunity

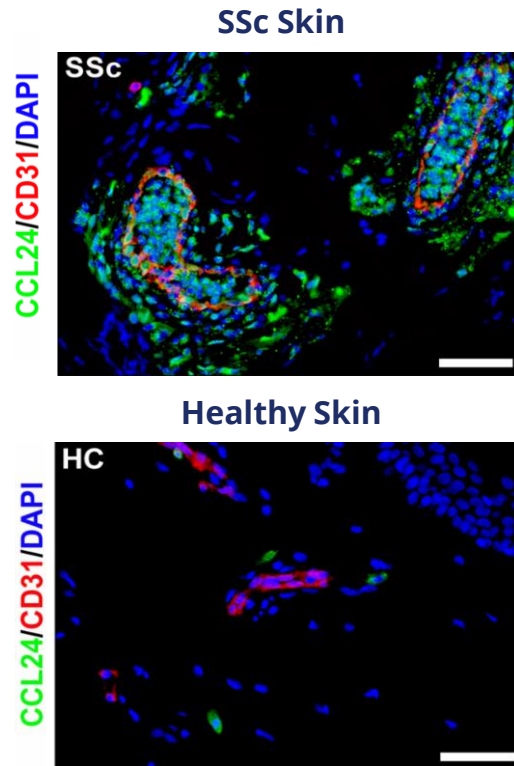
CM-101 SSc program is Phase 2-ready, with an open US IND



PATIENT  
SAMPLES

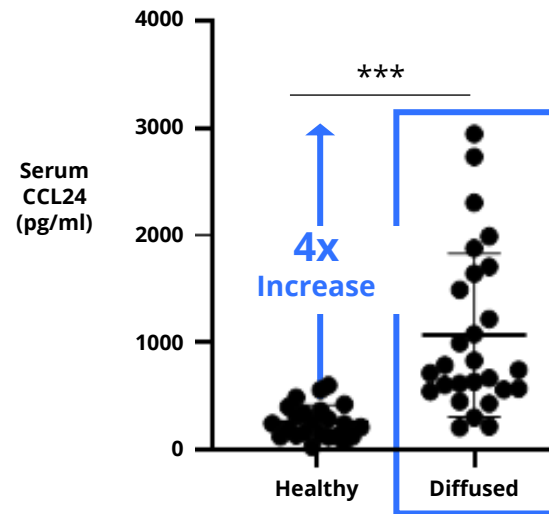
# CCL24: A Critical Target in Systemic Sclerosis

## CCL24 Elevated in SSc Skin



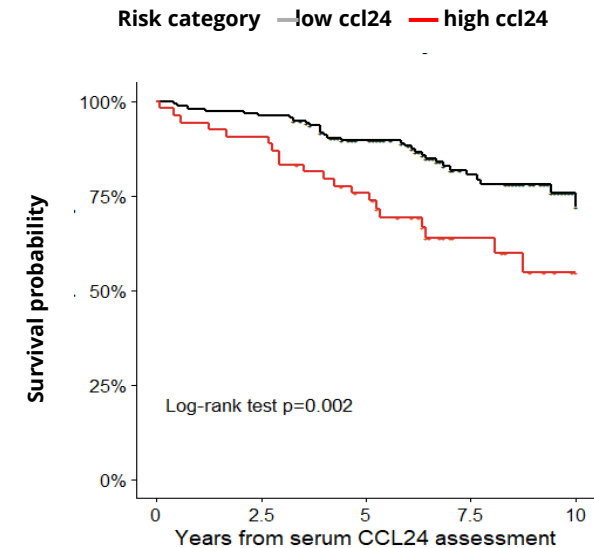
Collaboration with University of Florence

## CCL24 Overexpressed in SSc Serum



CCL24 overexpressed in serum of dSSc patients compared to healthy

## High CCL24 in SSc Serum Predicts Mortality



High CCL24 serum levels predict 5-year SSc-related mortality

Extensive animal model & patient sample data support potential therapeutic utility of CM-101 in Systemic Sclerosis

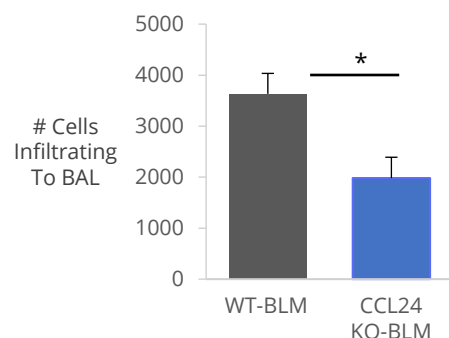
# CCL24 Blockade or Knockout Ameliorates SSc in Mouse Model

## CCL24 BLOCKADE REDUCES SSc-LIKE INFLAMMATION & FIBROSIS IN LUNG & SKIN

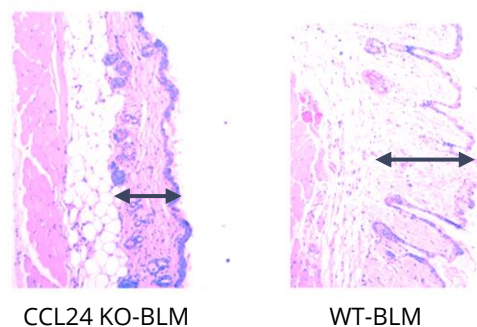


### CCL24 knockout mice

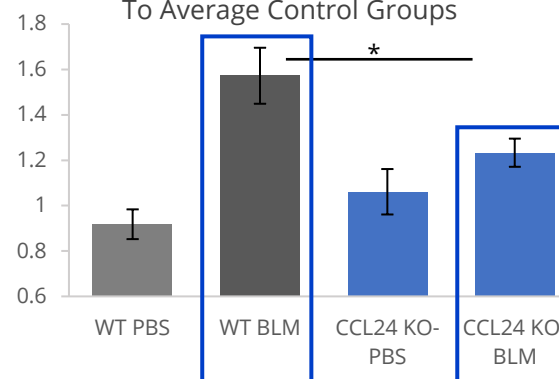
#### Immune Cell Lung Infiltration



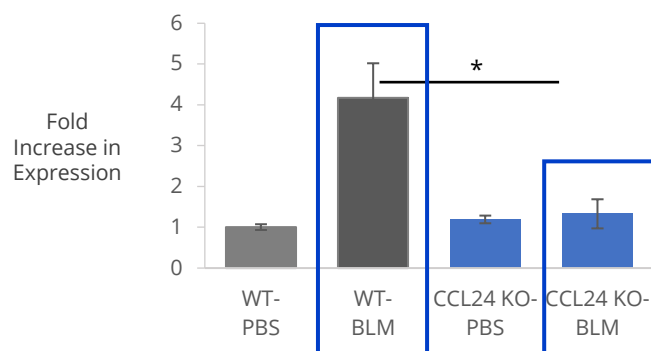
#### Dermal Thickness<sup>++</sup>



#### Dermal Thickness Compared To Average Control Groups

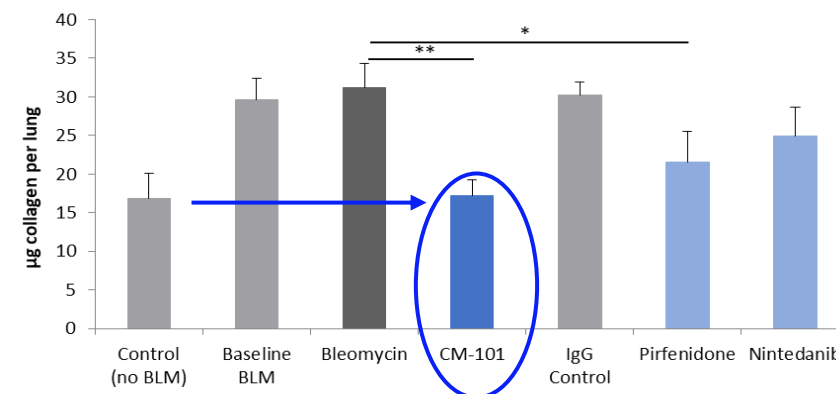


#### α SMA Expression in Skin Lesions

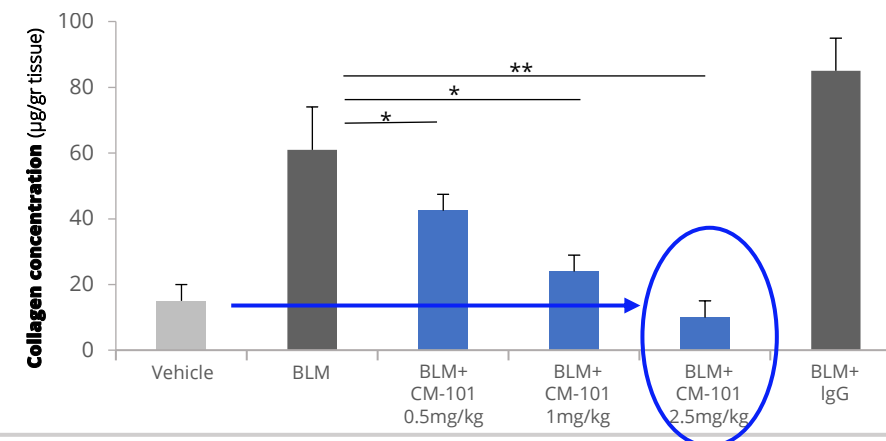


### CM-101 treated mice

#### BLEOMYCIN (IT)-INDUCED LUNG FIBROSIS



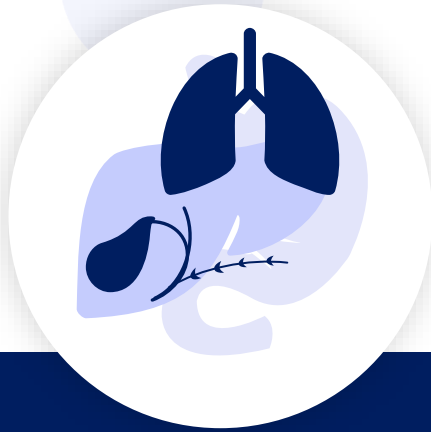
#### BLEOMYCIN (SC)-INDUCED DERMAL FIBROSIS



Mor A et al., Annals of Rheumatic Diseases, 2019; BLM-bleomycin; WT-wild type; KO-knock-out, BAL-bronchoalveolar lavage, PBS-phosphate-buffered saline; IT-intrathecal; SC-subcutaneous

\*p ≤ 0.05; \*\*p ≤ 0.01

++ Thickness measures taken at multiple locations on samples. Arrows on graphic are for illustrative purposes only

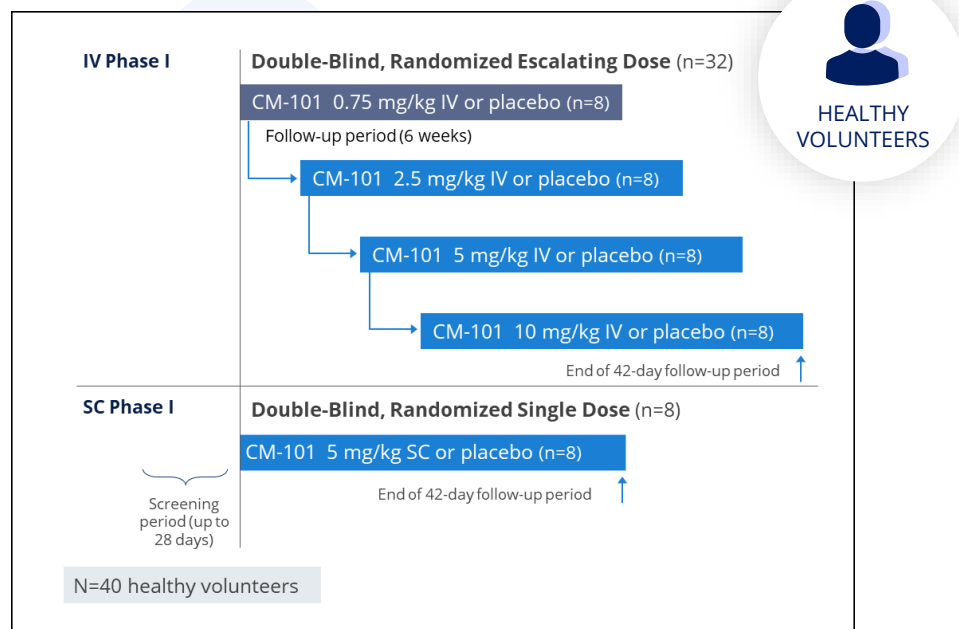


## CM-101: Clinical Trial Data

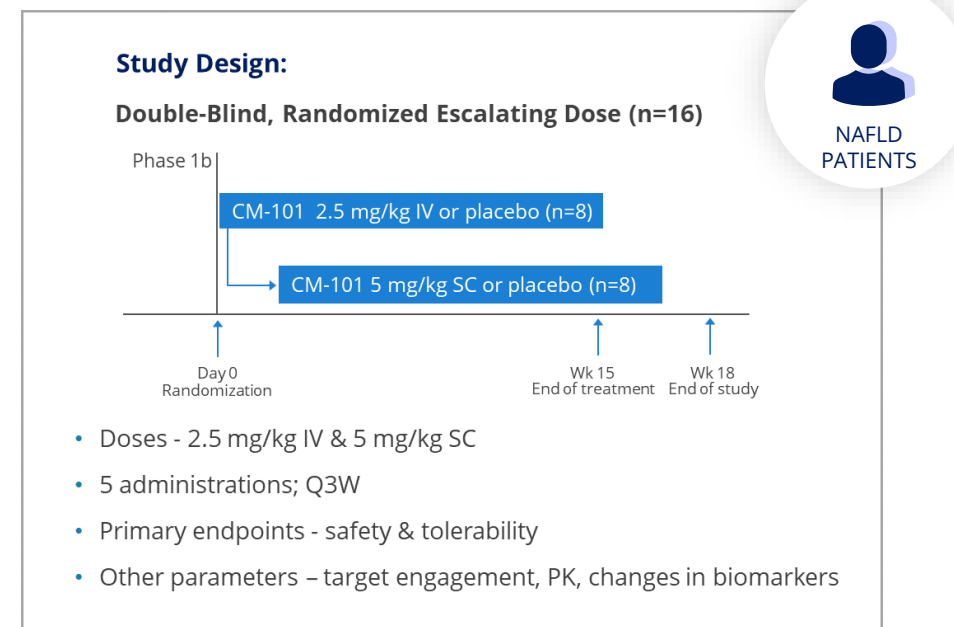
# CM-101 Appeared Safe in Phase 1 Safety Trials



## PHASE 1a SINGLE ASCENDING DOSE STUDY



## PHASE 1b MULTIPLE ASCENDING DOSE STUDY



- Multiple ascending doses (IV and SC) appeared safe and well tolerated
- Adverse Events were mild with no drug-related SAEs
- Dose-dependent target engagement for SC and IV formulations
- PK and half-life support 2-4-week dosing

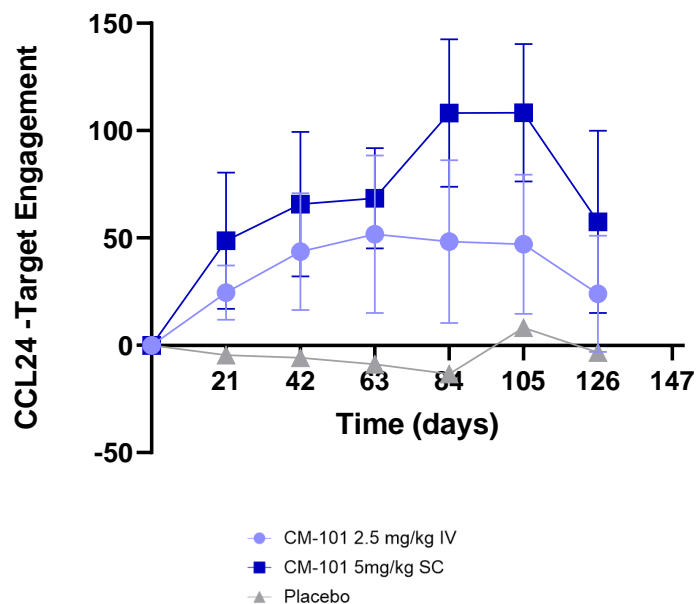
**CM-101 appeared safe and well-tolerated with favorable target engagement and pharmacokinetics in healthy volunteers and NAFLD patients**

# CM-101 Phase 1b Anti-Fibrotic Data Supports Potential Clinical Utility



NAFLD DATA PROVIDED FOUNDATION FOR FURTHER CLINICAL TRIALS

## TARGET ENGAGEMENT

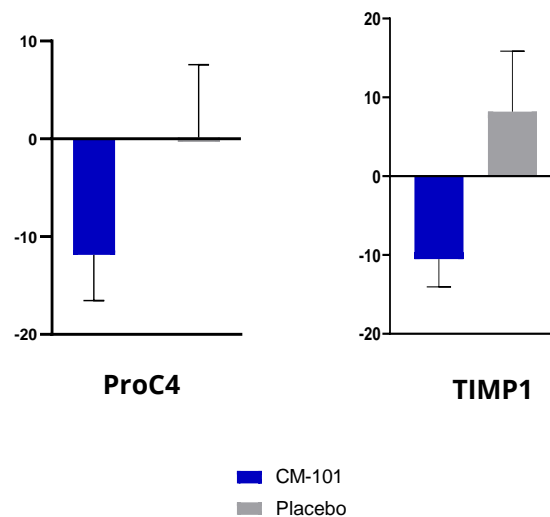


**Dose Dependent  
Target Engagement**

NAFLD-nonalcoholic fatty liver disease; IV-intravenous; SC-subcutaneous

## FIBROSIS BIOMARKERS\*

Relative Change from Baseline (%)



**Concordant results across six  
additional fibrotic markers**

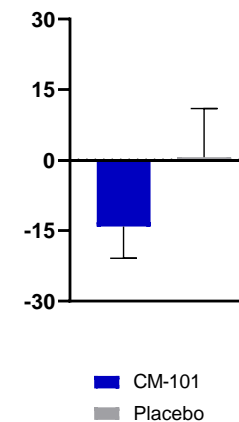
**Improved Fibrosis  
Biomarker Responses**

N=10 active, 3 placebo. Pooled from both dosing cohorts  
\*ProC4-procollagen 4; TIMP1-tissue inhibitor of metalloproteinase

## LIVER STIFFNESS

Changes in FibroScan®  
Fibrosis Status<sup>+</sup>

Relative Change from Baseline (%)



**Reduced liver stiffness**

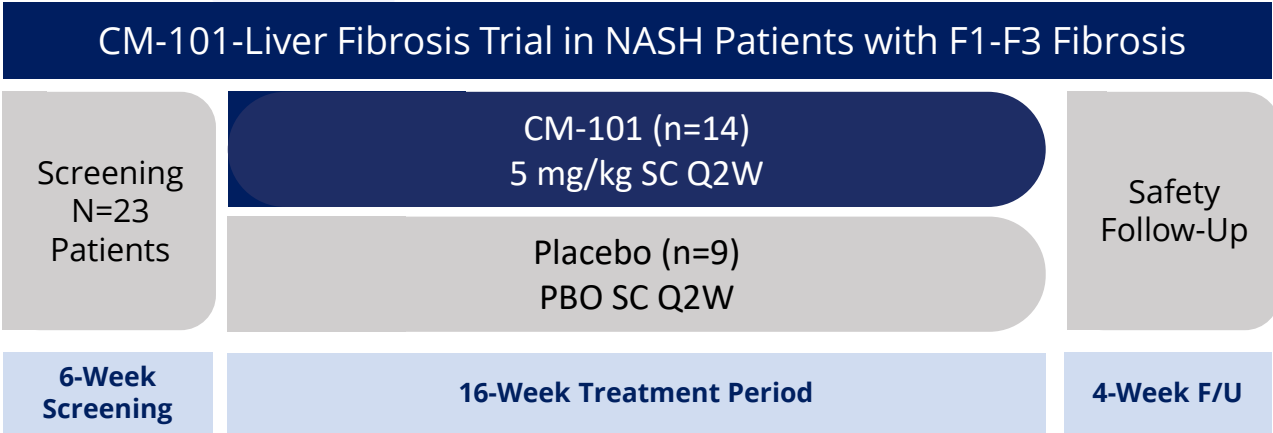
**Improved Physiological  
Measure**

\*Subjects with baseline transient elastography  $\geq 4$  kPa  
N=10 active, 3 placebo. Pooled from both dosing cohorts



# Phase 2a Liver Fibrosis Trial Supports CM-101 Mechanism of Action

## POSITIVE SAFETY AND TOLERABILITY PROFILE & IMPROVED BIOMARKERS IN NASH PATIENTS



- Double-blinded, randomized placebo-controlled trial in 23 non-cirrhotic NASH patients with biopsy-confirmed F1c-F3 fibrosis
- 5mg/kg SC injection
- 8 administrations per subject; Q2W

**Primary objective:**

- Determine safety & tolerability of subcutaneous CM-101 in NASH patients

**Secondary objectives:**

- Assess PK & PD profiles & drug exposure
- Measure liver fibrosis biomarkers & imaging
- Monitor Anti-Drug Antibodies (ADAs)

**Safety Results:**

- Multiple doses appeared safe
- Most AEs were mild with one unrelated SAE
- No significant injection site reactions
- No ADAs

CM-101-treated patients demonstrated improvements across a range of biomarkers associated with inflammation and fibrogenesis

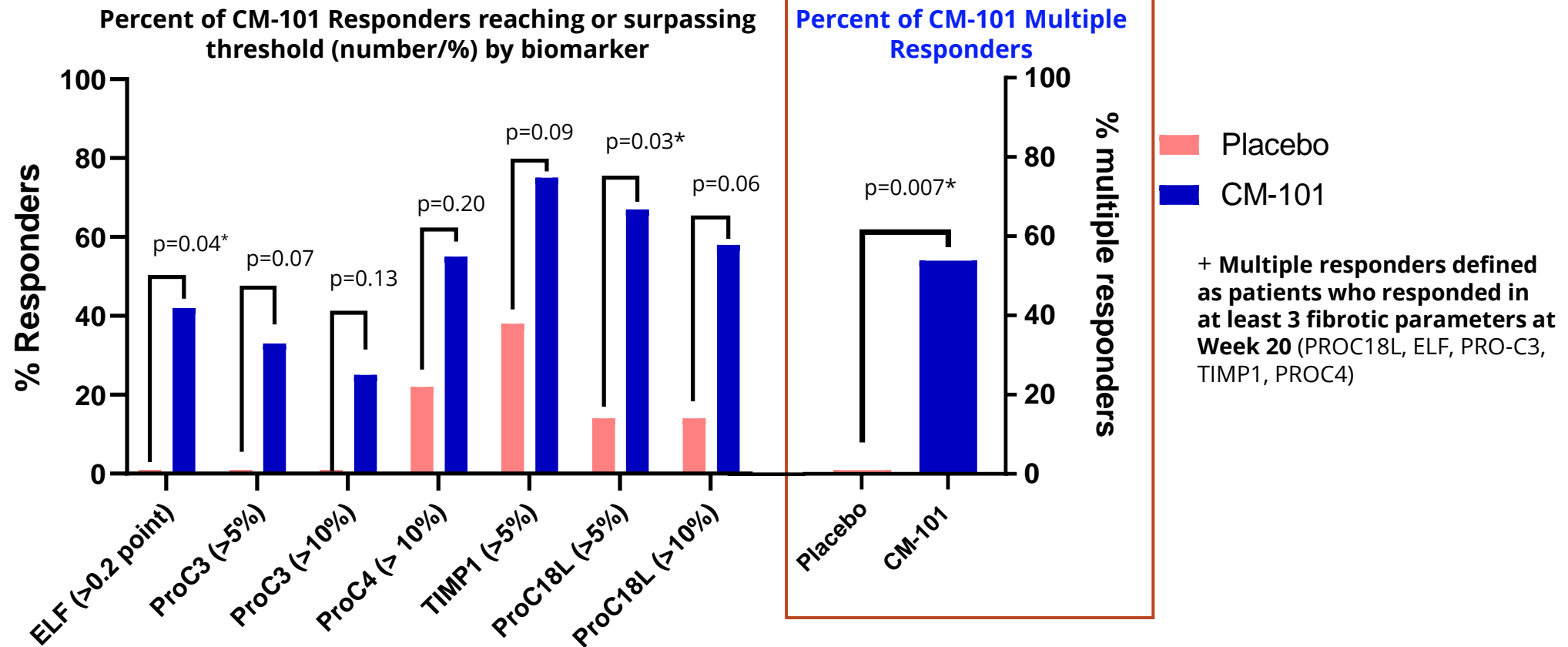
NASH–nonalcoholic steatohepatitis; SC–subcutaneous; Q2W–dosing once every 2 weeks; PK–pharmacokinetic; PD–pharmacodynamic; AEs–adverse events; SAE–serious adverse event; PBO–placebo

# CM-101 Produced More Biomarker Responders & Multiple Responders<sup>+</sup>



PATIENTS

CM-101 NASH PATIENTS DEMONSTRATED A GREATER RESPONSE ACROSS INFLAMMATION & FIBROSIS BIOMARKERS COMPARED TO PLACEBO; ~60% WERE MULTIPLE RESPONDERS COMPARED TO NONE IN PLACEBO GROUP



CM-101-treated patients demonstrated consistent pattern of positive responses across biomarkers

# Liver Stiffness Improved in CM-101-treated Patients

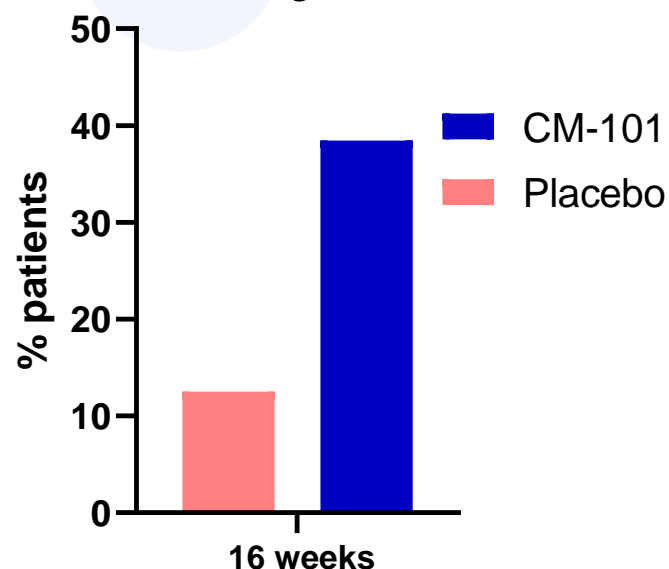
CM-101 PATIENTS HAD REDUCED LIVER STIFFNESS VS PLACEBO

CM-101 PATIENTS WITH HIGHER FAST SCORES HAD GREATER IMPROVEMENTS IN KEY BIOMARKERS



## Fibrosis Reduction

( $\geq 1$  Grade--% patients showing reduction of at least 1 grade in FibroScan)

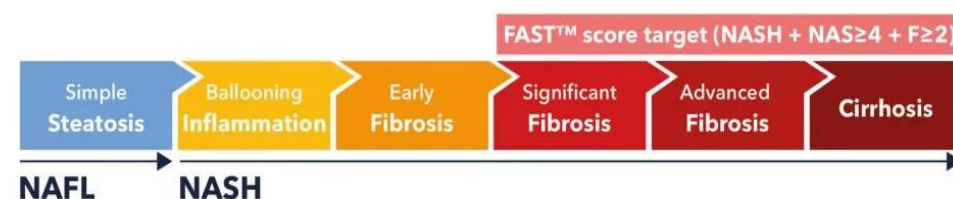


F0-F1	F2	F3	F4	Fibrosis Grading by FibroScan (kPa)
2 - 8.5	8.5 - 9.5	9.5 - 13.5	>13.5	

Placebo n=8; CM-101 n=13

## FibroScan-AST (FAST) Score\*

- FAST is a well-validated<sup>1</sup> non-invasive score for assessing NAFLD/NASH status & categorizing the risk of progression\*\*
- Patients with higher FAST scores are at greater risk of disease progression
- CM-101-treated patients had improved FAST scores vs placebo†
- CM-101-treated patients with higher FAST scores also showed the greatest improvements in fibro-inflammatory biomarkers



Higher proportion of CM-101-treated patients showed improvement in Liver Stiffness and in FAST-related scores

<sup>1</sup> Newsome et al. 2020;

\*Combines FibroScan (LSM by VCTE™ & CAP™) & AST levels; \*\* Low-risk (FAST  $\leq$  0.35); Indeterminate risk (0.35 < FAST score < 0.67); High-risk (FAST  $\geq$  0.67);

† Data not shown

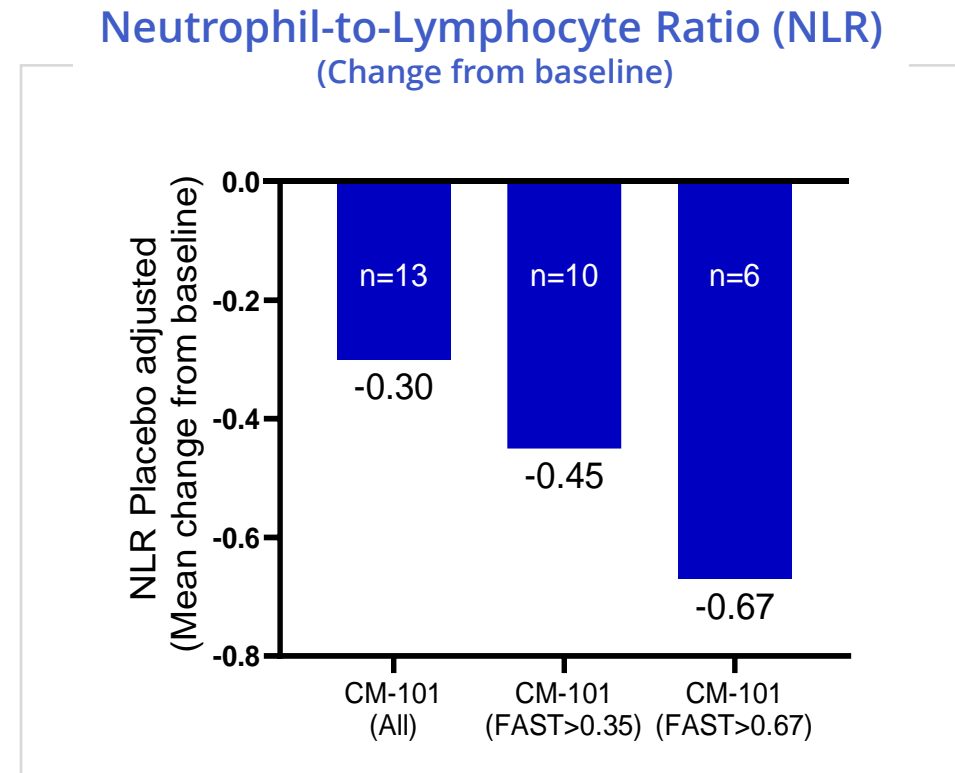
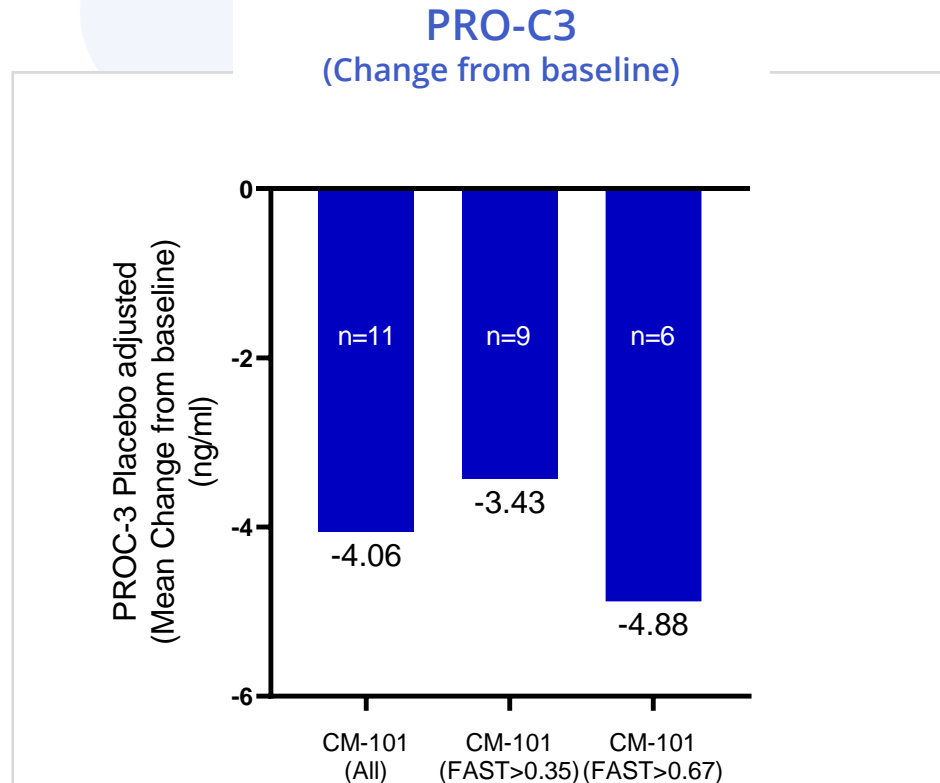
# CM-101 Improves Fibrosis and Inflammatory Biomarkers in High-Risk Patients

PRO-C3 IS A PLASMA BIOMARKER ASSOCIATED WITH ACTIVE LIVER FIBROGENESIS IN NASH AND PSC

NEUTROPHIL-TO-LYMPHOCYTE RATIO (NLR) IS AN INDICATOR OF INFLAMMATION



PATIENTS



Improvements in CM-101 high FAST score in NASH patients at greater risk for disease progression may signal the potential for positive response in PSC

# Experienced Management with Extensive Biopharma Experience



**Adi Mor, PhD**

Co-founder, Chief  
Executive Officer & Chief  
Scientific Officer



**Matthew Frankel, MD**

Chief Medical Officer &  
Vice President, Drug  
Development



**Sigal Fattal, CPA**

Chief Financial Officer



**Ilan Vaknin, PhD**

Vice President,  
Research & Development



**Jack Lawler**

Vice President,  
Global Clinical  
Development Operations



**Revital Aricha, PhD**

Vice President,  
Translational Science



## DEEP EXPERIENCE

Capital efficient

Translational science & global  
clinical development

Focused on delivering value  
to stakeholders

# CM-101 is Potential Breakthrough in Fibro-Inflammatory Diseases



## DUAL MECHANISM OF ACTION

Disease-modifying potential  
with unique target



## PRECISION TARGETING

Selective mAb enhances  
potential safety & efficacy



## INDICATION EXPANSION

Into other diseases



## FOCUSED ON HIGH UNMET NEED, LARGE POTENTIAL RARE DISEASES

With substantial partnering interest



## SAFE AND WELL-TOLERATED

In multiple clinical studies to date



MAJOR CATALYST–Phase 2 PSC Topline Readout Expected Midyear 2024 with Cash Runway  
through End-of-Year 2024



# End Notes

1. Mor A et al., Annals of Rheumatic Diseases, 2019; Segal Salto et al, JHEP, 2020; Isgro et al, Mucosal Immunology, 2013; Kohan et al, Ann Allergy Asthma Immunol. 2010; Braga et al, Frontiers in Immunology, 2015; Foster et al, J. Proteome Res. 2015; Lescoat et al, 2021 & Chemomab unpublished data
2. Mor A et al., Annals of Rheumatic Diseases, 2019; Segal Salto et al, JHEP, 2020; Ablin et al, Clinical and Experimental Immunology, 2010; Mor et al, World Journal of Cardiovascular Diseases, 2013 & Chemomab unpublished data
3. John Hopkins Medicine; Lazaridis et al. Primary Sclerosing Cholangitis, N Engl J Med. 2016, James H et al. Primary Sclerosing Cholangitis, Part 1: Epidemiology, Etiopathogenesis, Clinical Features, and Treatment Gastroenterology & Hepatology 2018
4. Johns Hopkins Medicine; Global data; Bergamasco et al., Clin Epidemiol. 2019; 11: 257-273



Advancing CM-101: Novel Target with Disease-  
Modifying Potential for Fibro-Inflammatory Diseases

Thank you!