APRIL 2024

Chemomab

THERAPEUTICS

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
2 Upcoming Milestones with Major Catalyst Midyear 2024	PSC Phase 2 topline data Midyear 2024; Phase 2 Open Label data in late 2024/early 2025 Cash runway through end Q1 2025 Long-term holders include well-known investors: Orbimed, Thiel, Apeiron No debt; clean capital structure

Pipeline in a Drug: Targeting Rare Diseases

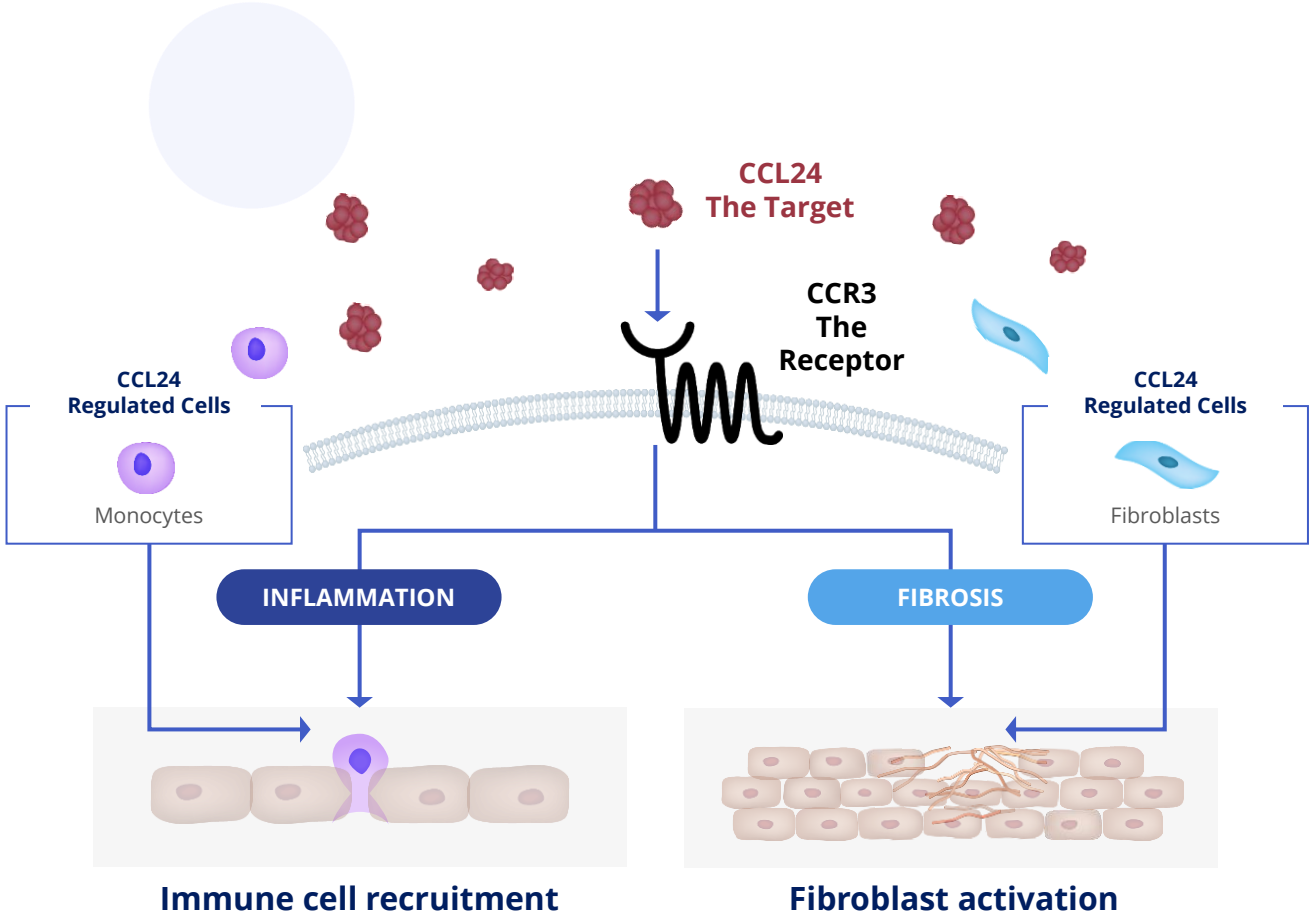
Agent	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated Milestones	Global Rights
CM-101 Anti-CCL24 mAb	Primary Sclerosing Cholangitis (PSC)	Orphan designation from FDA & EMA; FDA Fast Track designation; Patient enrollment completed				Topline 15-Week Data Midyear 2024 Topline Open Label Data Late 2024/ Early 2025	Chemomab THERAPEUTICS
CM-101 Anti-CCL24 mAb	Systemic Sclerosis (SSc)	Orphan designation from FDA & EMA				Phase 2-Ready Open US IND	Chemomab THERAPEUTICS

Four Completed Clinical Studies:

- Phase 2a Liver Fibrosis in NASH patients--Safety, PK & positive biomarker response (2023)
- Lung injury investigator-initiated study--Safety, PK & positive biomarker response (2022)
- Phase 1b in NAFLD patients--Safety, PK & positive biomarker response (2021)
- Phase 1a safety study in healthy volunteers-–Safety, PK (2018, 2019)

NAFLD-nonalcoholic fatty liver disease; NASH-nonalcoholic steatohepatitis; SC-subcutaneous

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

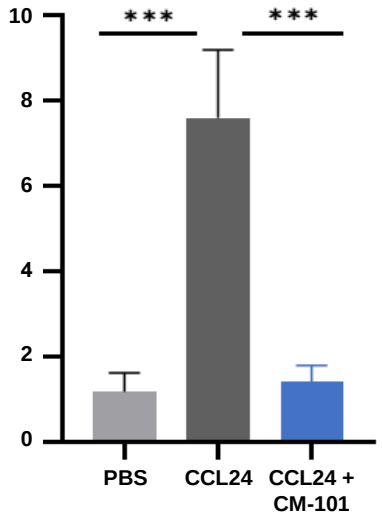
Liver, skin, lung, kidney, others

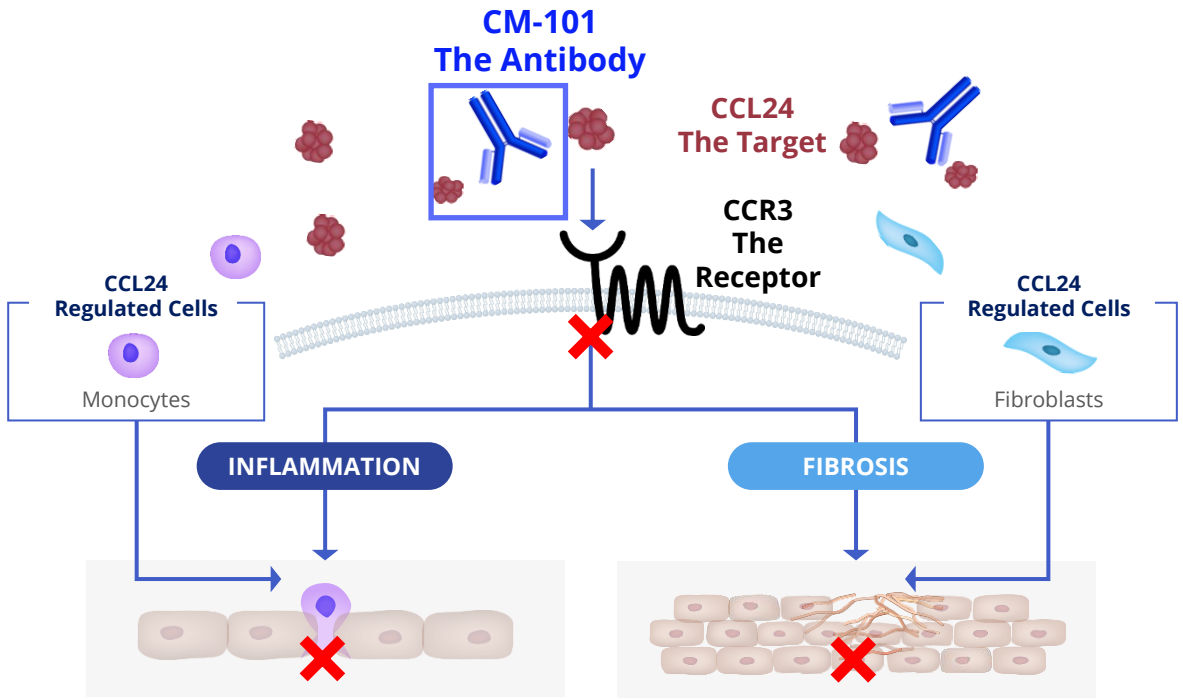
Wide therapeutic margin

CM-101 Reduces Inflammation and Fibrosis by Neutralizing CCL24

CM-101 Reduces In Vivo Monocyte Recruitment

Number of Migrated Monocytes (x1000)





Immune cell recruitment
ATTENUATED

Fibroblast activation
ATTENUATED

Neutralizing CCL24 Has Advantages Over Blocking the Receptor

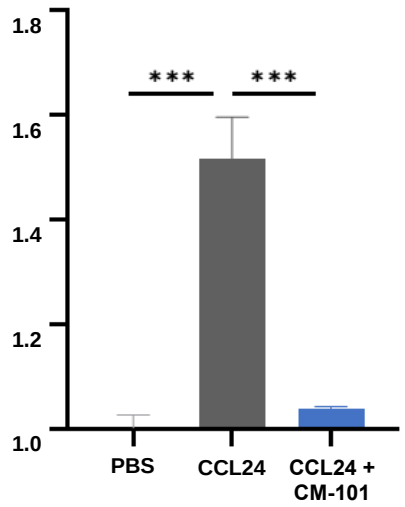
Avoids context dependence of CCR3 receptor

Efficacy of direct approach

Favorable safety profile; retains normal repair functions

CM-101 Inhibits Primary Fibroblast Activation

Proliferation: Relative Change from Baseline



PBS-phosphate buffered saline used as placebo; *** p ≤0.001

Broad & Diverse Preclinical Data De-risks Clinical Development

Mechanism of Action

Primary sclerosing cholangitis
- Biomarker correlations
- Expression of CCL24 and CCR3

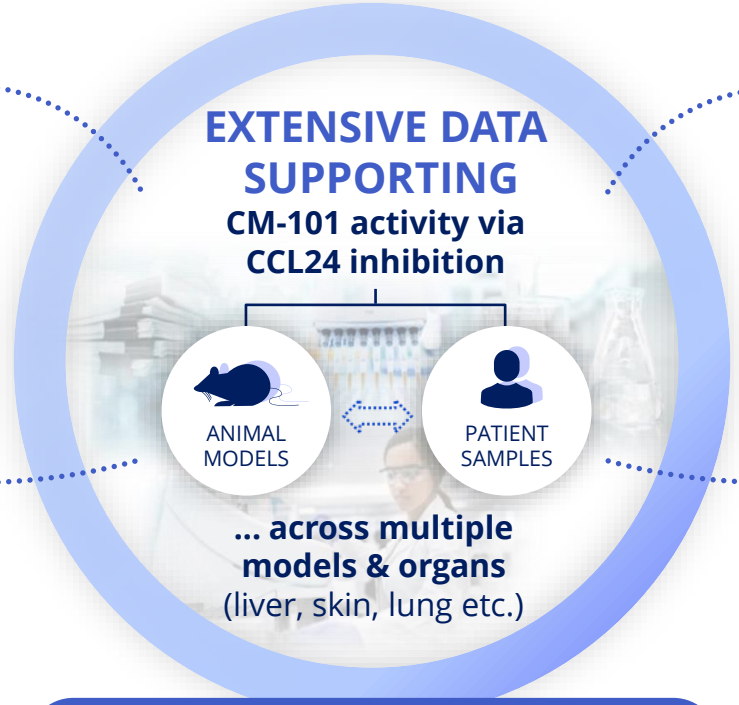
Liver fibrosis (NASH)
- Disease severity correlation
- Expression of CCL24 and CCR3

Systemic sclerosis
- Fibrotic biomarker correlation
- Disease deterioration correlation
- Expression of CCL24 and CCR3

CCL24 Target Validation In-vivo (Knockout Animal Models)

Systemic sclerosis
- CCL24 knock-out vs. WT (wild type) in Bleomycin-induced skin fibrosis model (mice)

Liver metabolism and inflammation
- CCL24 knock-out vs. WT in MCD-induced NASH (mice)



CCL24 Target Validation Ex-vivo Patient Samples

CM-101 effects on fibroblast activation
- Dermal, hepatic and lung fibroblast activation
- Dermal and liver fibroblast transition to myofibroblast
- Hepatic fibroblast motility

CM-101 effects on immune cell migration and recruitment
- Dermal fibroblast migration
- Monocyte polarization
- Monocyte recruitment

Toxicology & Safety

- **Short-term GLP** in rodents
- **Long-term GLP** in non-human primates
- **Ex vivo safety**: ADCC, CDC, cytokine secretion
- **Tissue cross reactivity**

Proof of Concept: Animal Models Concordant with Patient Data

Primary sclerosing cholangitis
- ANIT-induced cholestasis--chronic and acute (mice)
- Bile duct ligation (rat)
- MDR2 knock-out (mice)

Systemic sclerosis
- Bleomycin-induced skin fibrosis (mice)
- Bleomycin-induced lung fibrosis (mice)

Liver fibrosis
- TAA-induced liver fibrosis (rat and mice)

Liver metabolism and inflammation
- STAM (mice)
- MCD diet-induced NASH (mice)

Atherosclerosis
- ApoE knock-out model (mice)

~12 Different Animal Disease Models

AND

300+ Patient Samples

Anti-Fibrotic Mechanisms Demonstrated Across Clinical Trials

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

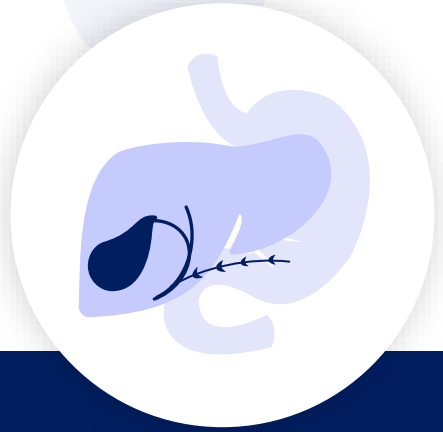
CM-101 IMPROVED MULTIPLE BIOMARKERS IN 3 CLINICAL TRIALS IN PATIENTS

- Reduced fibrosis-related biomarkers in fatty liver, liver fibrosis, NASH & acute lung inflammation[1]
- Demonstrated anti-inflammatory effects in NASH & acute lung inflammation[1]
- 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

1-*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

NAFLD-nonalcoholic fatty liver disease; NASH-nonalcoholic steatohepatitis; PSC-primary sclerosing cholangitis; AE-adverse event; SAE-serious adverse event; PK-pharmacokinetic



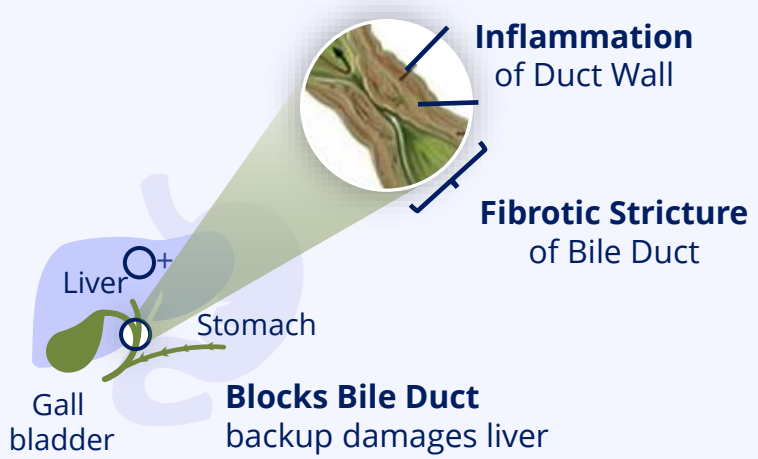
CM-101: Potential Treatment for Primary Sclerosing Cholangitis (PSC)

PSC Has High Unmeet 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

(1) Rupp C (2018) United European Gastroenterology Journal. 6(2): 255–262. (2) Palmela C (2018) Gut Liver. 12(1): 17-29. (3) Emek E (2019) Transplantation Proceedings 51(7): 2439-2441. (4) Bakhshi Z (2020). Journal of gastroenterology, 55(5), 523–532. Figure adapted from John Hopkins.

CM-101's Unique Dual Activity Has Disease-Modifying Potential

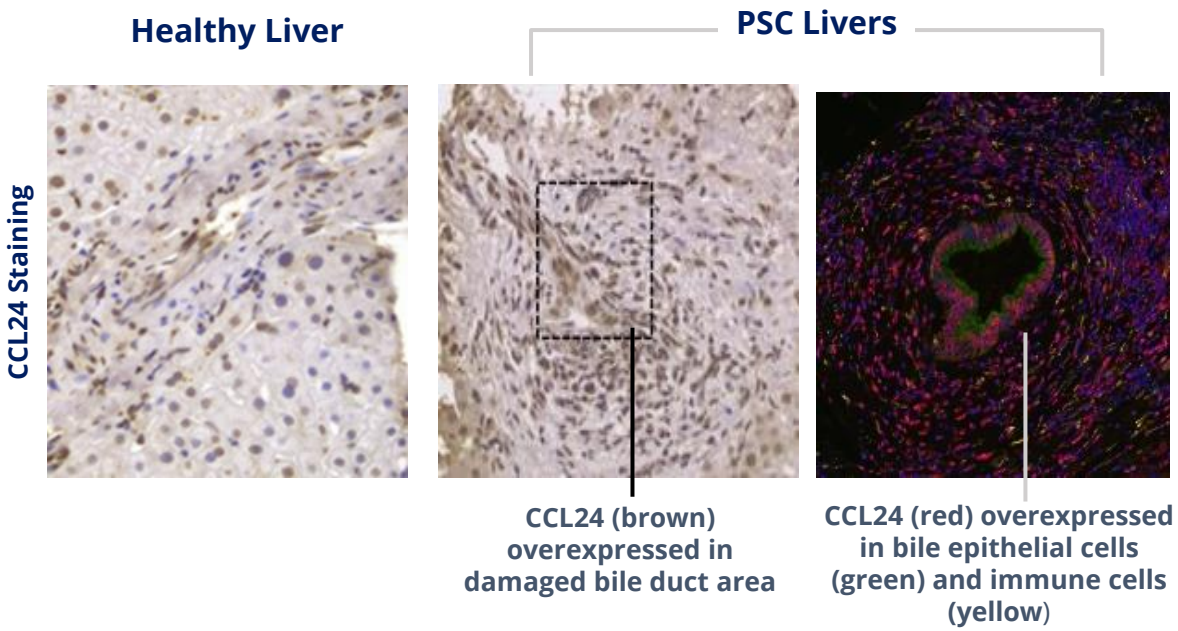
CM-101 is the only therapeutic candidate targeting both inflammation and fibrosis

Company	Candidate	Mechanism	Disease Modifying Potential	Stage
Chemomab	CM-101	Anti-CCL24; Inhibits key inflammatory AND fibrotic processes	√√	**Phase 2** *Recruitment completed. Midyear 2024 readout*
Pliant	PLN-74809 Bexotegrast	Fibrosis-focused selective integrin inhibitor	√	**Phase 2** *Safety; activity; No dose response. High dose 24-wk data mid-2024*
NGM Bio	Aldafermin	FGF19 analog-regulates bile acid synthesis & metabolic components	√	**Phase 2** *Completed. Safety & dose-dependent response in ELF. In discussions with FDA for Phase 3 design*
Dr Falk	norUDCA	UDCA homologue--metabolic bile acid mechanism	–	**Phase 3** *Enrollment completed. 2-yr trial with no interim data. Only in Europe*
Mirum	Volixibat	Ileal bile acid inhibitor Targeting pruritis	–	**Phase 2** *Interim readout 1H2024*
Ipsen/(Albireo)	Ritivixibat	Ileal bile acid inhibitor Targeting pruritis	–	**Phase 2** *Recruiting. open-label*
Ipsen/Genfit	Elafibranor	PPAR dual agonist Metabolic focus	–	**Phase 2** *Recruiting. Topline data end 2024*

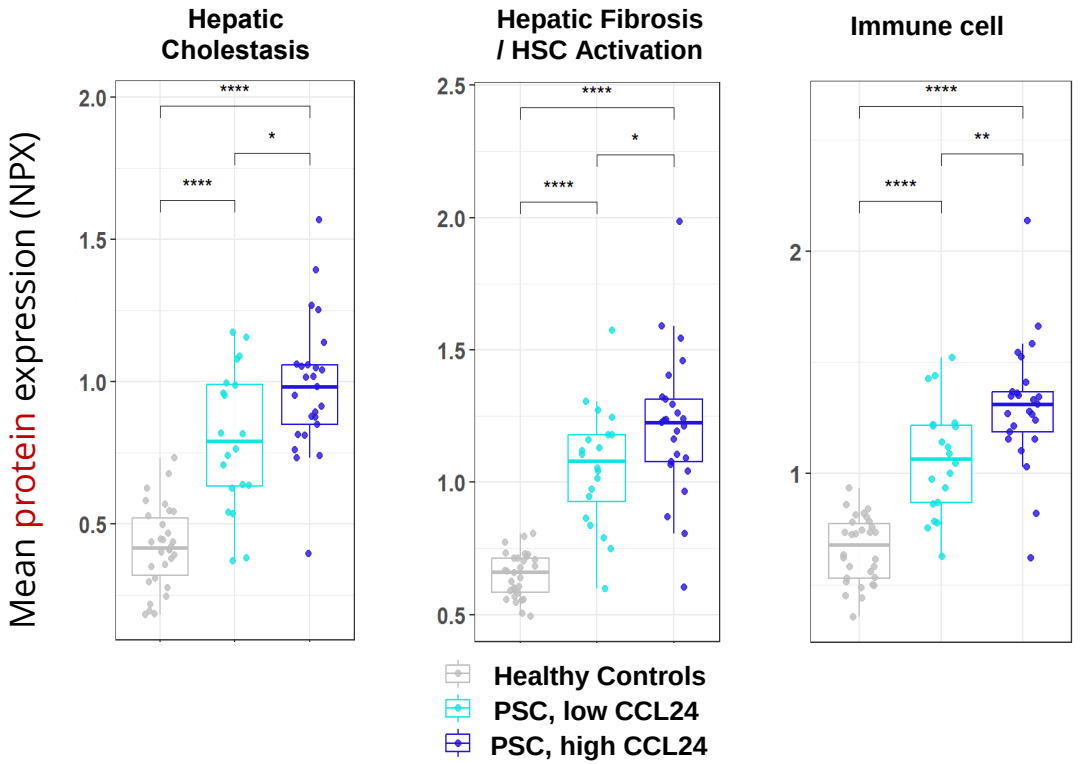
CCL24 Levels Are Elevated in PSC Patients and Associated with Protein Expression in Key PSC Pathways

PATIENTS

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

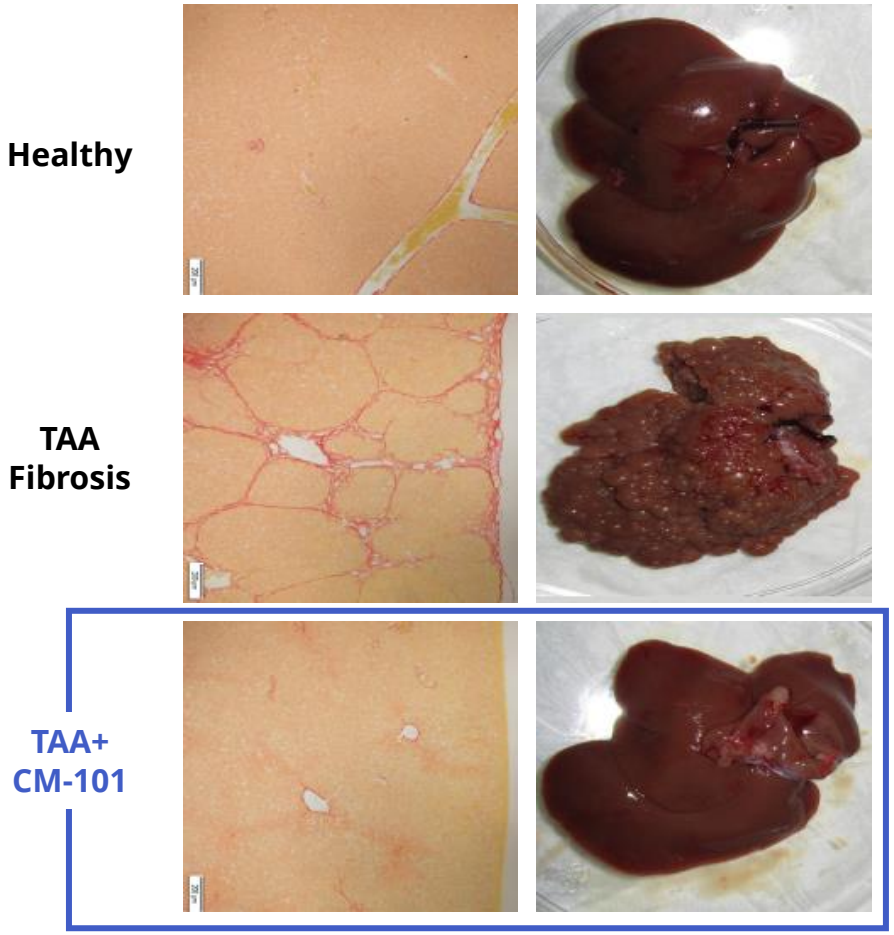
Greenman et al JCI insight 2023; "*Serum proteomics reveals association of CCL24 with key aspects of primary sclerosing cholangitis*", EASL 2023. abstract 2178
Low CCL24=levels below median; high CCL24=levels above median; HSC-hepatic stellate cells; NPX-or Normalized Protein eXpression, is a way of measuring how much of a protein is present in a sample

CM-101 Reduces Liver Fibrosis by >80% Preclinically

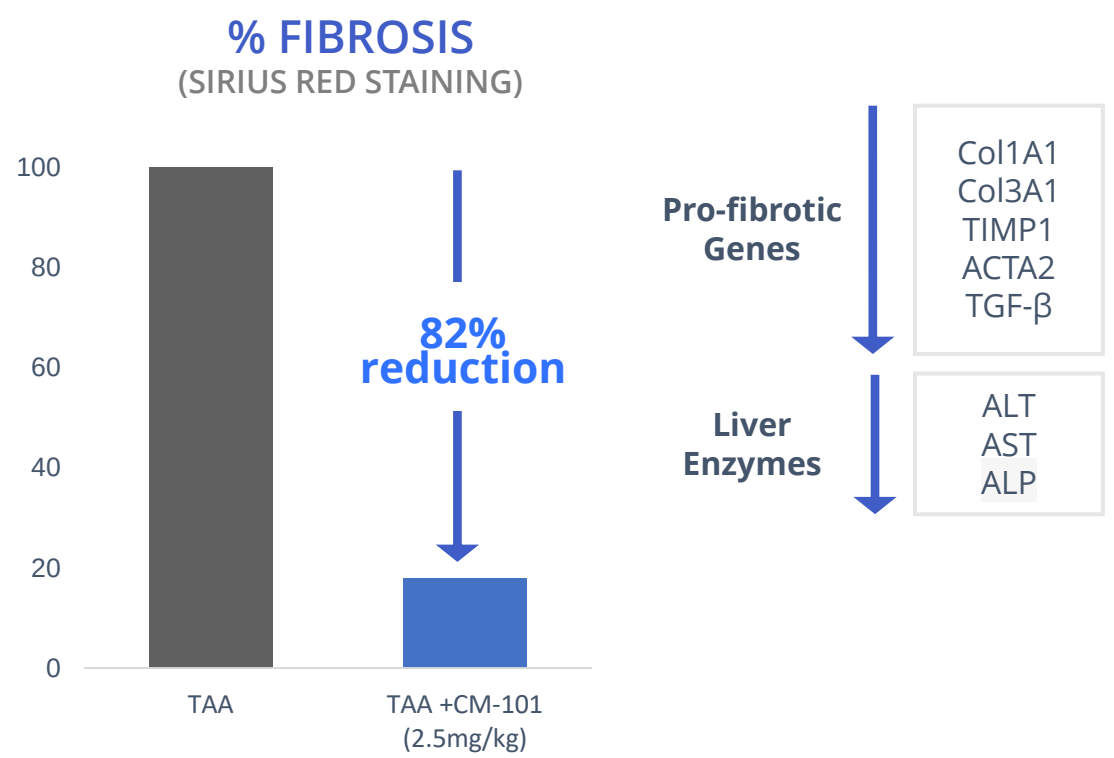
ANIMAL MODELS
RAT TAA
MODEL

RAT MODEL LIVERS

SIRIUS RED (COLLAGEN)



REDUCED LIVER COLLAGEN DEPOSITION



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

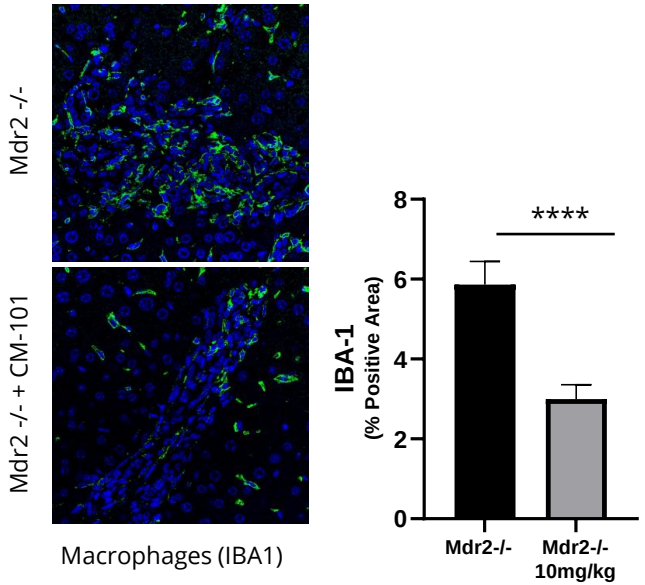
Segal-Salto et al, JHEP reports 2020
TAA- thioacetamide

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

MDR 2 MOUSE KNOCKOUT MODEL

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

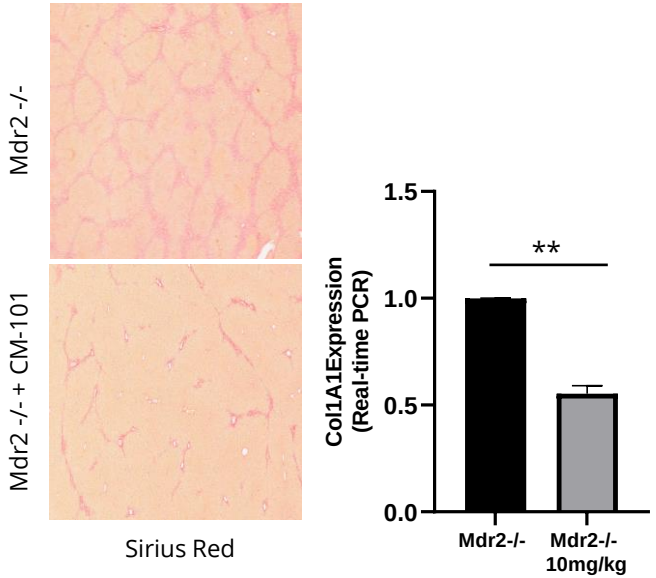
REDUCED MACROPHAGES



Macrophages (IBA1)

Reduction in **INFLAMMATION**

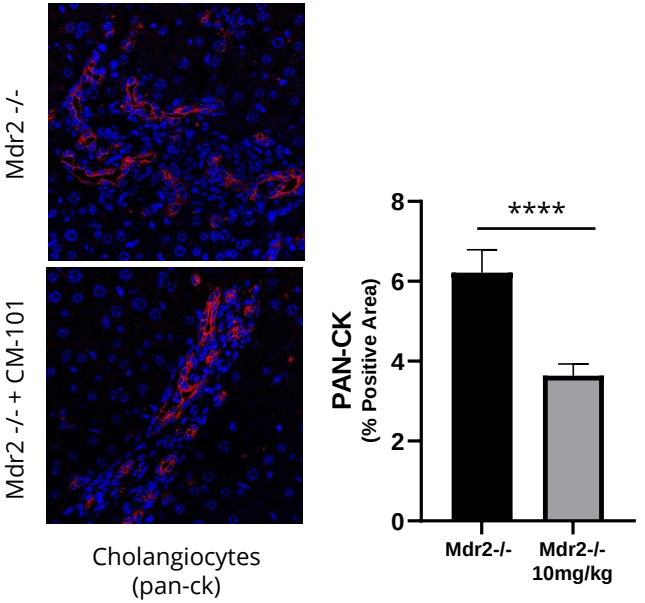
ATTENUATED LIVER COLLAGEN DEPOSITION



Sirius Red

Reduction in **FIBROSIS**

REDUCED BILE EPITHELIAL PROLIFERATION



Cholangiocytes (pan-ck)

Reduction in main driver of **DISEASE PATHOLOGY**

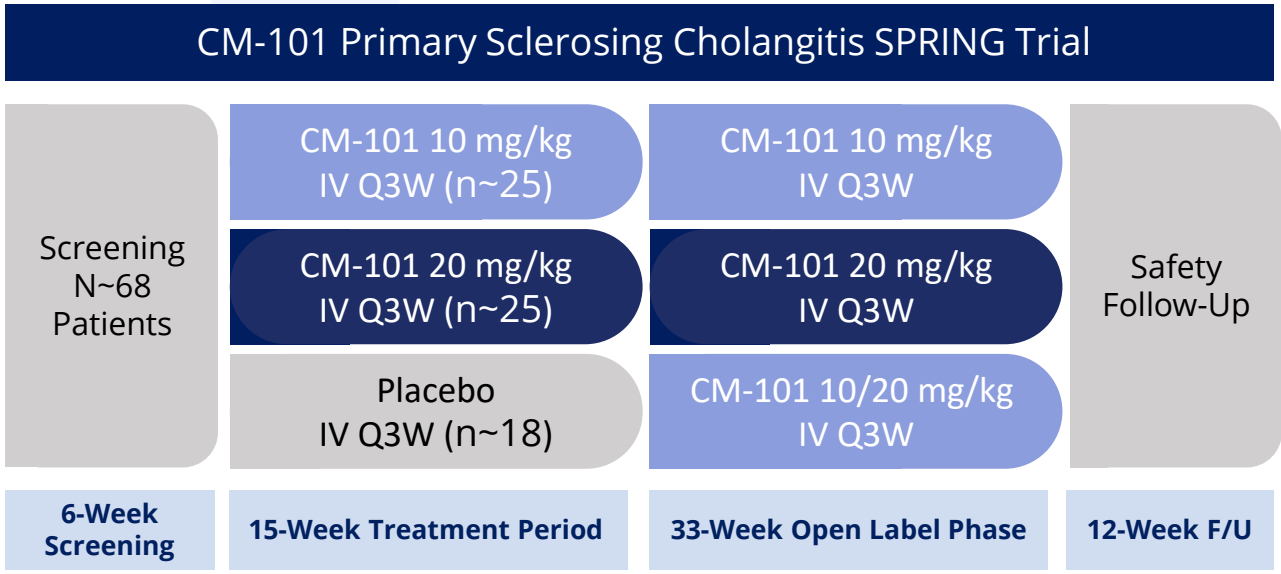
CM-101 interferes with the 3 major pathways that drive PSC pathology

Greenman et al JCI insight 2023; * In MDR2-/- Mouse model

Patient Enrollment Completed in CM-101 Phase 2 PSC Trial

PATIENTS

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

Targeting 15-Week topline data readout Midyear 2024 & Open Label topline data readout late 2024/early 2025

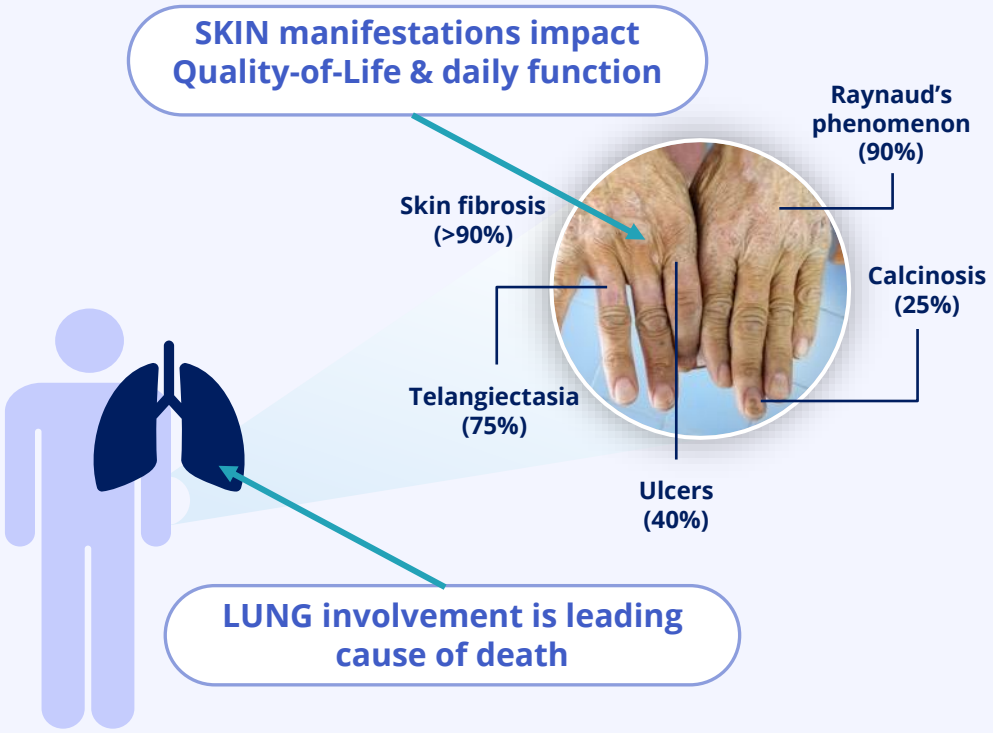
PSC-primary sclerosing cholangitis; Q3W-once every 3 weeks; IV-intravenous; ALP-alkaline phosphatase; IBD-inflammatory bowel disease; UDCA-ursodeoxycholic acid; ELF–enhanced liver fibrosis score; AST-aspartate aminotransferase; Pro-C3-type lll collagen biomarker; Pro-C5-type V collagen biomarker; FibroScan-measure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

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

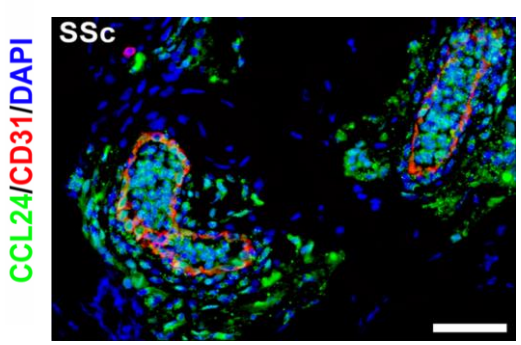
Source: See Endnote 4
7 Major Markets – USA, 5EU (UK, Germany, France, Italy, Spain), Japan

CCL24: A Critical Target in Systemic Sclerosis

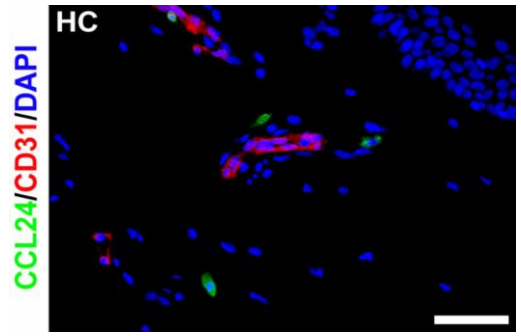
PATIENT SAMPLES

CCL24 Elevated in SSc Skin

SSc Skin

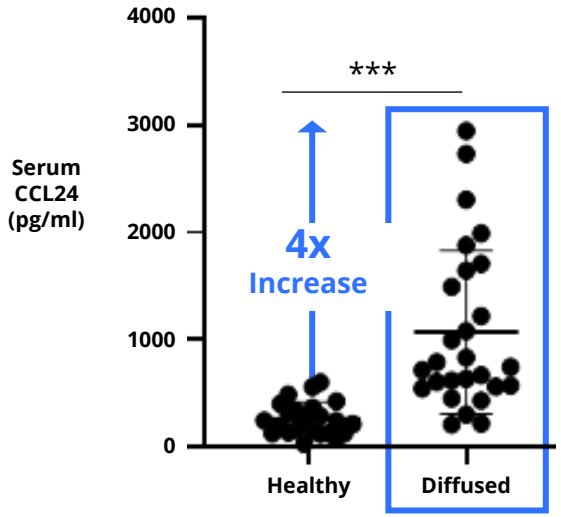


Healthy Skin



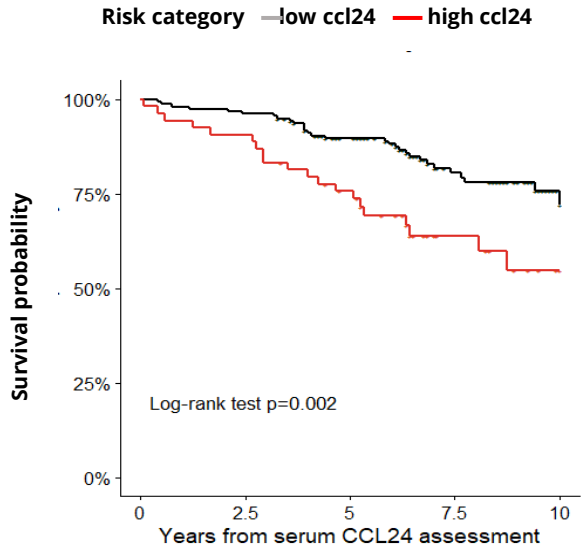
Collaboration with University of Florence

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

dSSc – diffuse systemic sclerosis; Mor A et al., Annals of Rheumatic Diseases, 2019
*** p ≤0.001

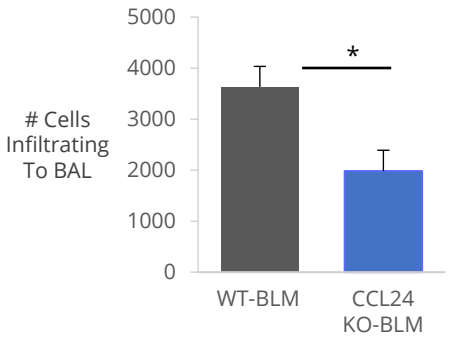
CCL24 Blockade or Knockout Ameliorates SSc in Mouse Model

SSc BLEOMYCIN MOUSE MODEL

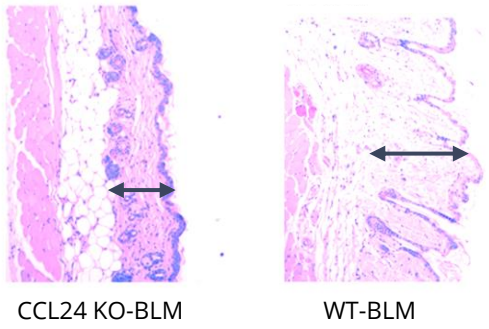
CCL24 BLOCKADE REDUCES SSc-LIKE INFLAMMATION & FIBROSIS IN LUNG & SKIN

CCL24 knockout mice

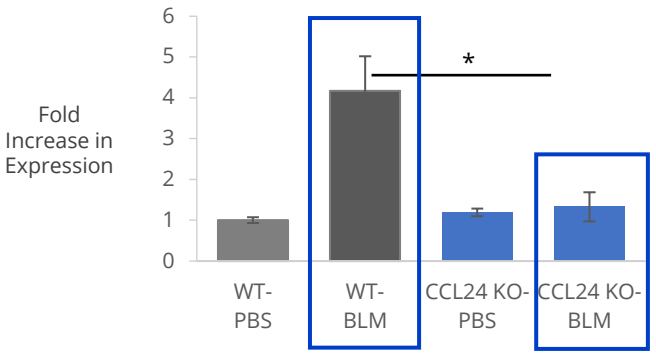
Immune Cell Lung Infiltration

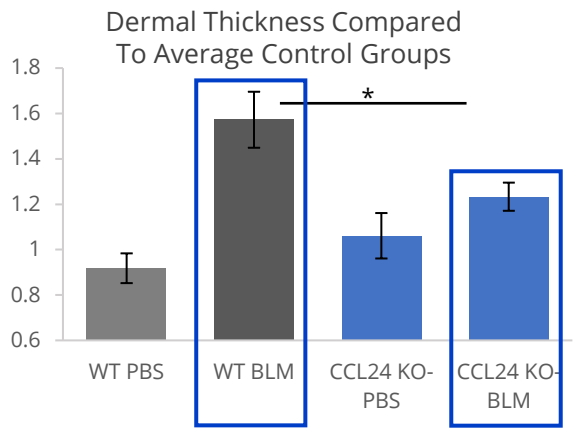


Dermal Thickness++



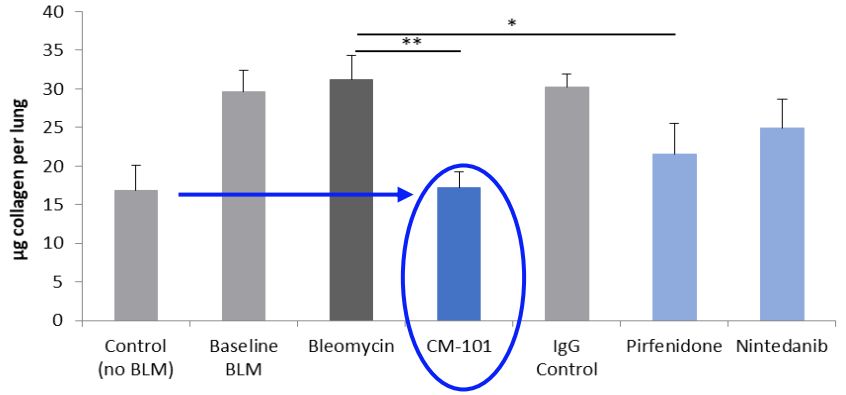
α SMA Expression in Skin Lesions



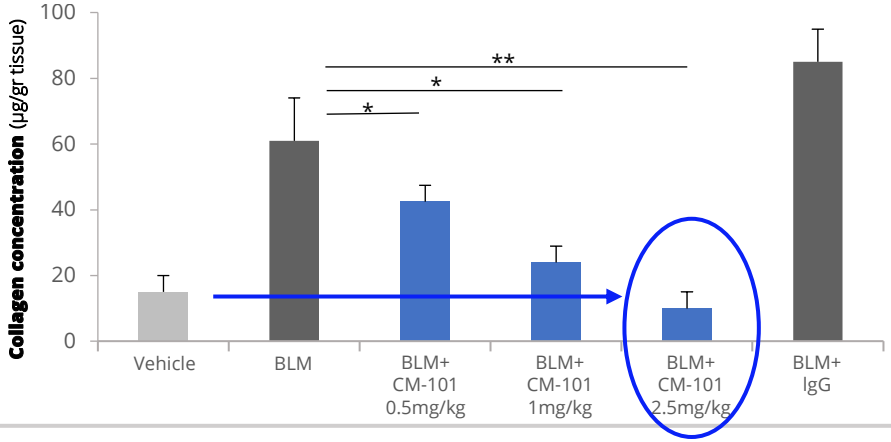


CM-101 treated mice

BLEOMYCIN (IT)-INDUCED LUNG FIBROSIS



BLEOMYCIN (SC)-INDUCED DERMAL FIBROSIS

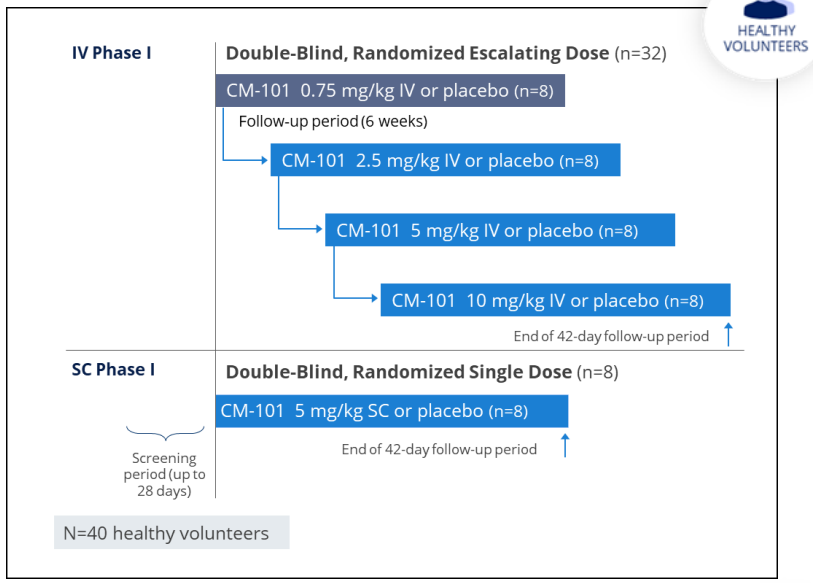


Mor A et al., Annals of Rheumatic Diseases, 2019; BLM-bleomycin; WT-wild type; KO-knock-out, BAL-bronchoalveolar lavage, PBS-phosphate-buffered saline; IT-intrathecal; SC-subcutaneous
*p ≤0.05; **p ≤0.01
++ Thickness measures taken at multiple locations on samples. Arrows on graphic are for illustrative purposes only

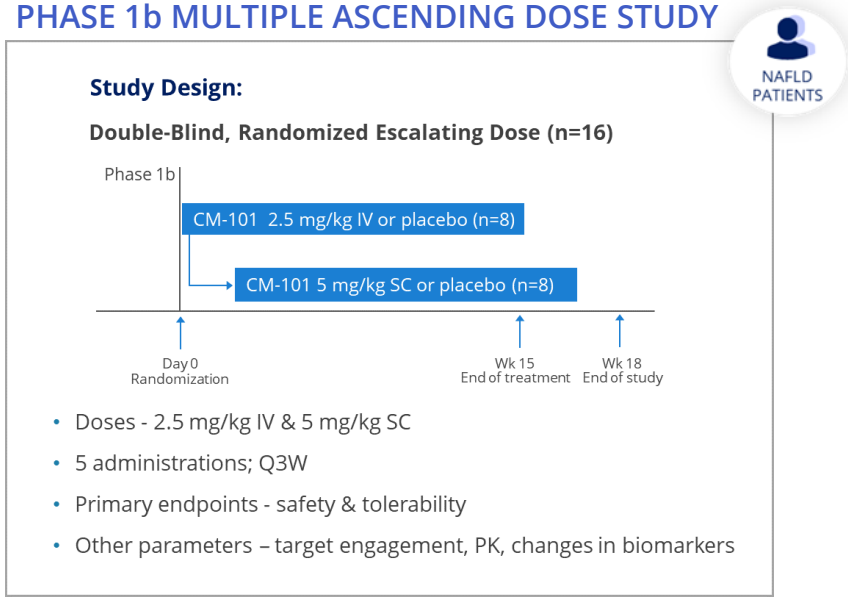
CM-101: Clinical Trial Data

CM-101 Appeared Safe & Active in Phase 1 Trials

PHASE 1a SINGLE ASCENDING DOSE STUDY



PHASE 1b MULTIPLE ASCENDING DOSE STUDY



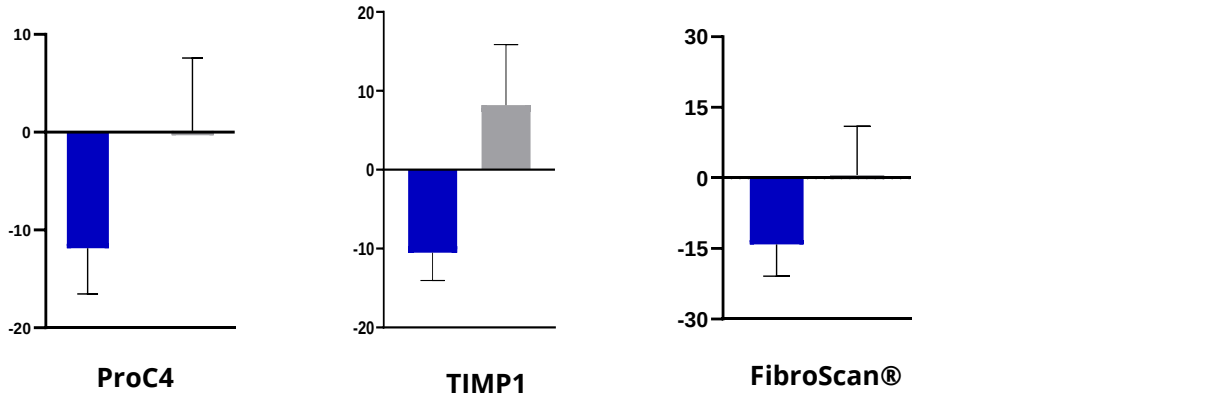
PHASE 1b STUDY IN NAFLD PATIENTS

NAFLD PATIENTS

- 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

IMPROVED FIBROSIS BIOMARKERS & LIVER STIFFNESS IN NAFLD PATIENTS

Relative Change from Baseline (%)



Concordant results across six additional fibrotic markers

Reduced liver stiffness

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

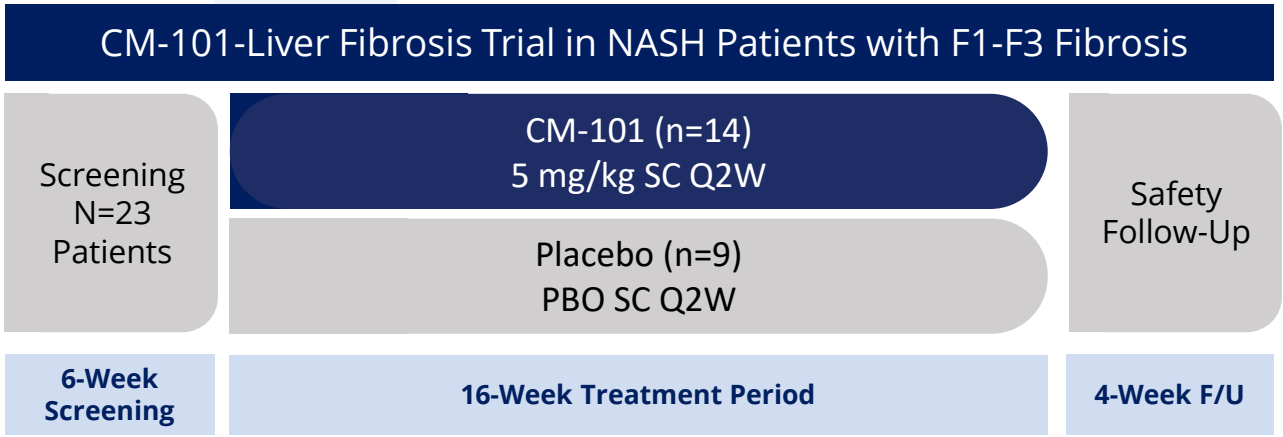
*Subjects with baseline transient elastography ≥ 4 kPa
N=10 active, 3 placebo. Pooled from both dosing cohorts

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

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

PATIENTS

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

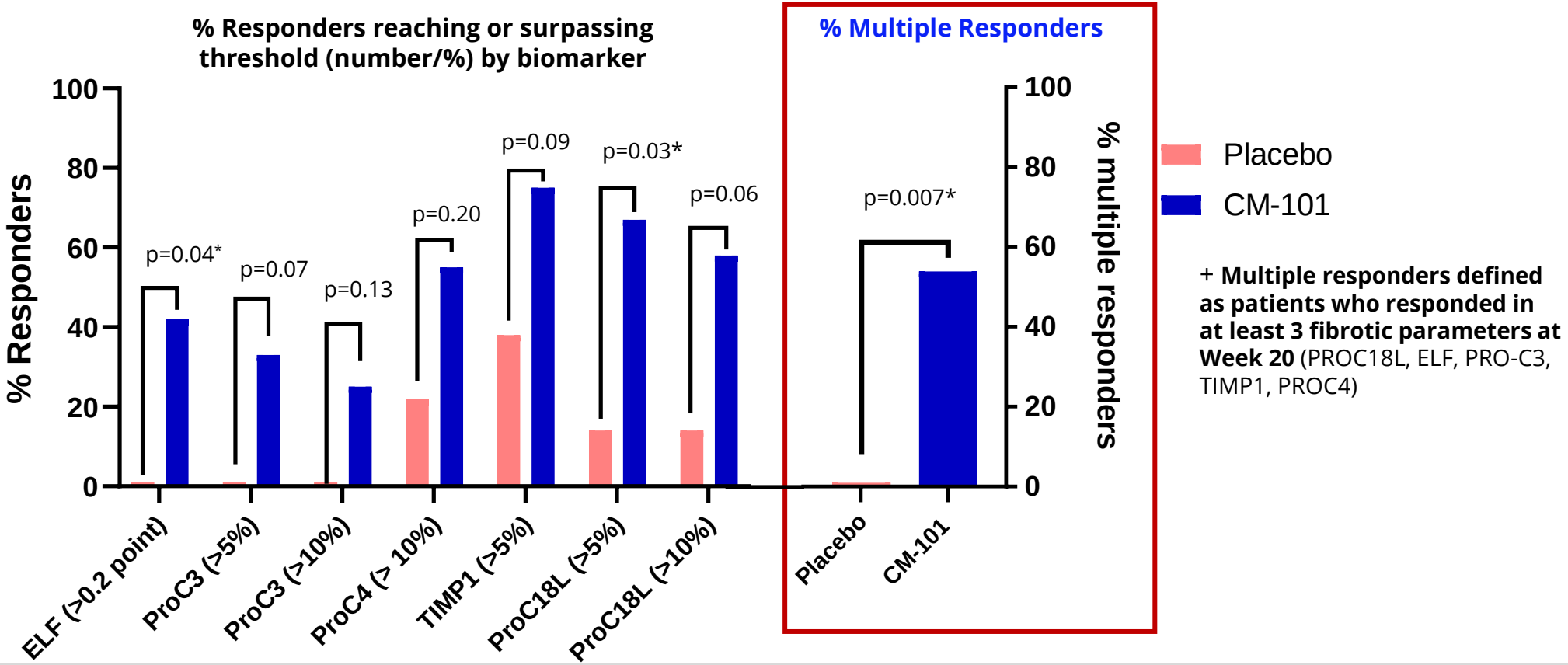
CM-101-treated patients demonstrated improvements across a range of biomarkers associated with inflammation and fibrogenesis

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+

PATIENTS

CM-101 NASH PATIENTS DEMONSTRATED A GREATER RESPONSE ACROSS INFLAMMATION & FIBROSIS BIOMARKERS COMPARED TO PLACEBO; ~60% WERE MULTIPLE RESPONDERS COMPARED TO 0% IN PLACEBO GROUP



+ **Multiple responders defined as patients who responded in at least 3 fibrotic parameters at Week 20** (PROC18L, ELF, PRO-C3, TIMP1, PROC4)

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

N=12 active, 8 placebo per protocol population analysis; p=p value (T test)

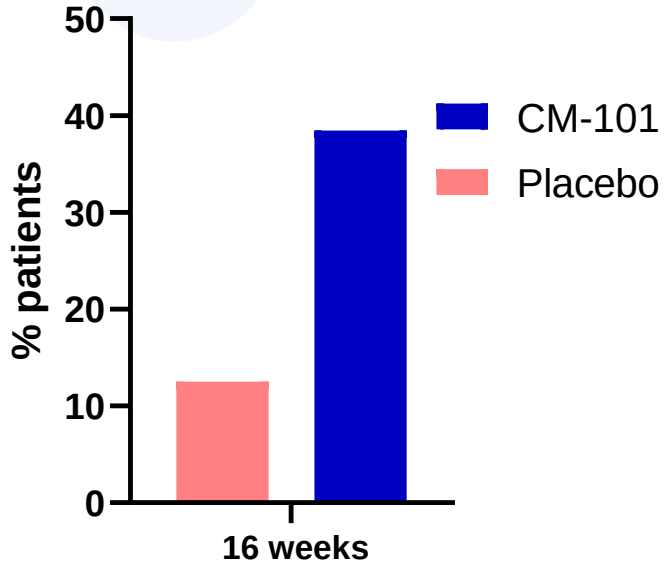
Liver Stiffness & Fibrogenesis Improved in CM-101-treated Patients

PATIENTS

CM-101 PATIENTS HAD REDUCED LIVER STIFFNESS & PROC-3 LEVELS vs PLACEBO

Liver Stiffness Reduction (FibroScan)

(≥ 1 Grade--% patients showing reduction of at least 1 grade in FibroScan)



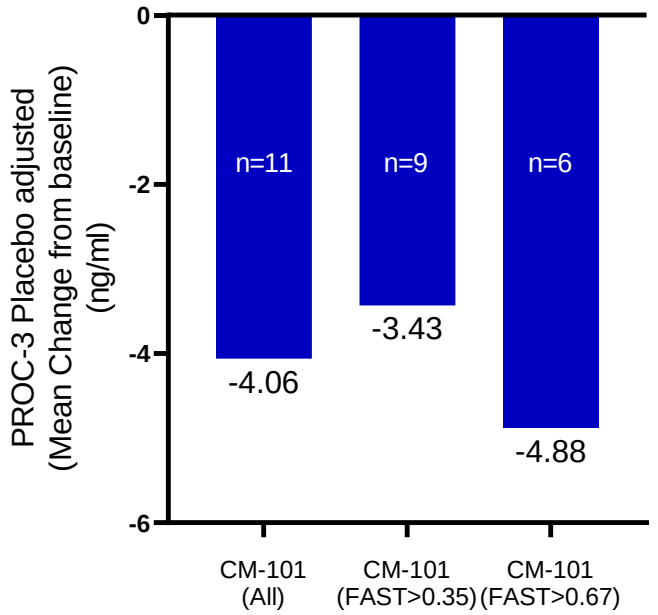
F0-F1	F2	F3	F4
2 - 8.5	8.5 - 9.5	9.5 - 13.5	>13.5

Fibrosis Grading by FibroScan (kPa)

Placebo n=8; CM-101 n=13

Fibrogenesis Improvement (Pro-C3)

(≥ 0.67 FAST score represents patients with high risk of progression)



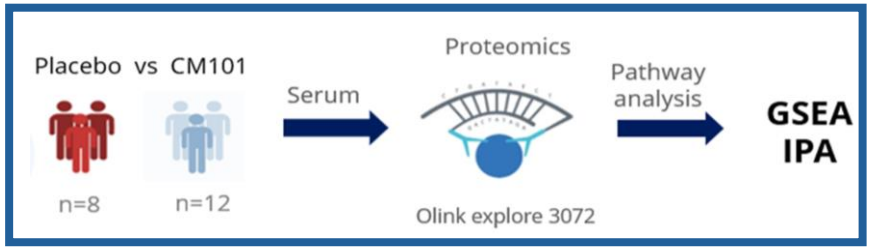
Higher proportion of CM-101-treated patients showed improvement in Liver Stiffness

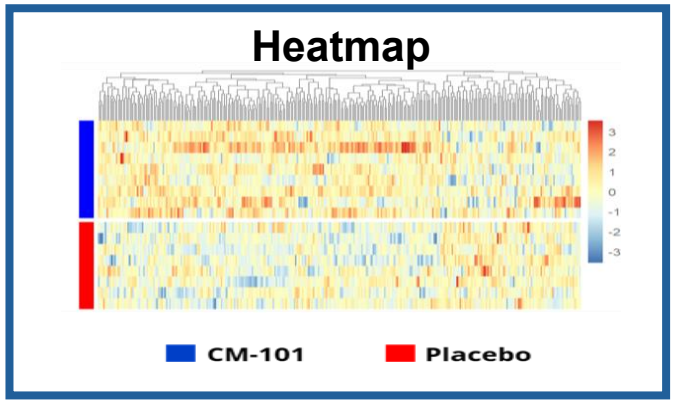
CM-101-treated patients with higher FAST* scores showed the greatest improvements in fibro-inflammatory biomarkers

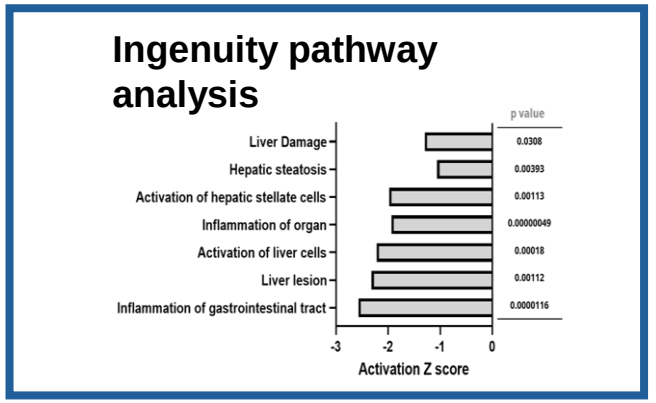
*FAST Combines FibroScan (LSM by VCTE™ & CAP™) & AST levels; Low-risk (FAST ≤ 0.35); Indeterminate risk (0.35 < FAST score < 0.67); High-risk (FAST ≥ 0.67); FAST is a well-validated non-invasive score for assessing NAFLD/NASH status & categorizing the risk of progression

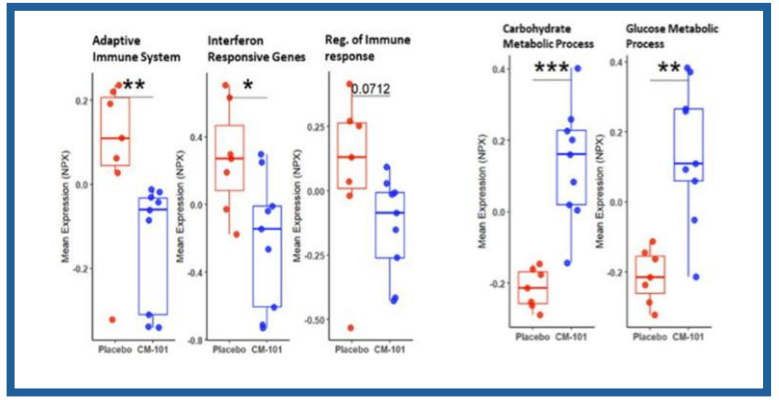
Proteomic Analysis of CM-101 Phase 2 Liver Fibrosis Study Showed Improvements in Fibrotic, Inflammatory and Metabolic Pathways

Proteome Profiling Analysis of 3000 Proteins Differentiates between CM-101 and Placebo Groups in NASH Patients with Liver Fibrosis









CM-101 attenuates multiple fibrotic and inflammatory pathways while improving metabolic pathways – all are essential in mitigating the pathology associated with progressive fibrotic liver diseases

*Heatmap of significant (p<0.05, by linear mixed model) proteins altered in the treatment group compared to placebo. Values are centered and scaled.
Boxplots of key pathways showing mean fold change (NPX values) across all proteins in a given pathway, for each treatment group (p<0.05: *, <0.01: **, < 0.001: *).
***Ingenuity pathway analysis (IPA)- Diseases & Bio Functions and Toxicity Functions, filtered for liver & hepatic related pathways & their corresponding activation z-score & p values.

Experienced Management with Extensive Biopharma Experience

Adi Mor, PhD
Co-founder, Chief Executive Officer & Chief Scientific Officer

TEL AVIV UNIVERSITY אוניברסיטת תל אביב
Tel-Aviv Sourasky Medical Center

Sigal Fattal, CPA
Chief Financial Officer

BiomX
evogene

Jack Lawler
Senior Vice President, Global Clinical Development Operations

teva
Cephalon
Goldfinch Bio the kidney company
Shire

Matthew Frankel, MD
Chief Medical Officer & Vice President, Drug Development

Boehringer Ingelheim
NOVARTIS
REATA PHARMACEUTICALS
FibroGen
Schering-Plough

Ilan Vaknin, PhD
Vice President, Research & Development

compugen
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Revital Aricha, PhD
Vice President, Translational Science

brainstorm cell therapeutics
WEIZMANN INSTITUTE OF SCIENCE

DEEP EXPERIENCE

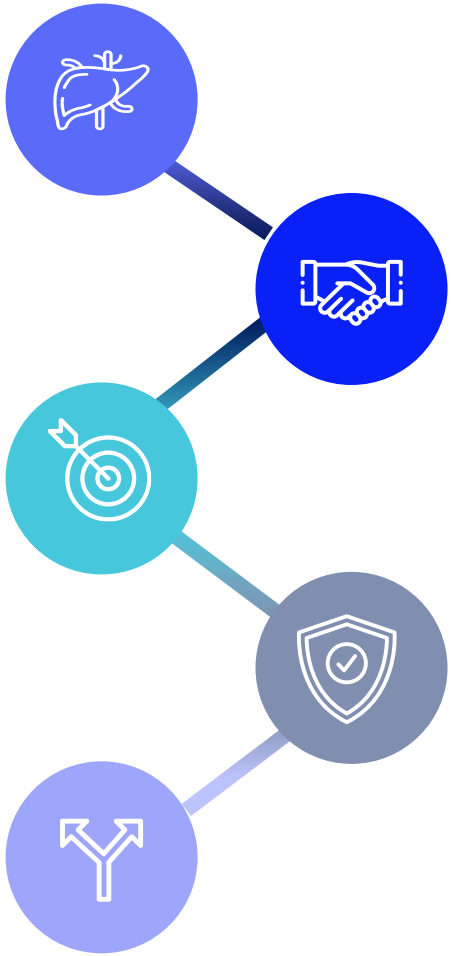
- Capital efficient
- Translational science & global clinical development
- Focused on delivering value to stakeholders

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

UPCOMING CATALYSTS–Phase 2 PSC topline data Midyear 2024 & PSC Open Label data Late 2024/Early 2025, with Cash Runway through End Q1 2025

Chemomab
THERAPEUTICS

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

Thank you!