MARCH 2023

Chemomab

THERAPEUTICS

Pioneering Innovative Treatments for 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 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.

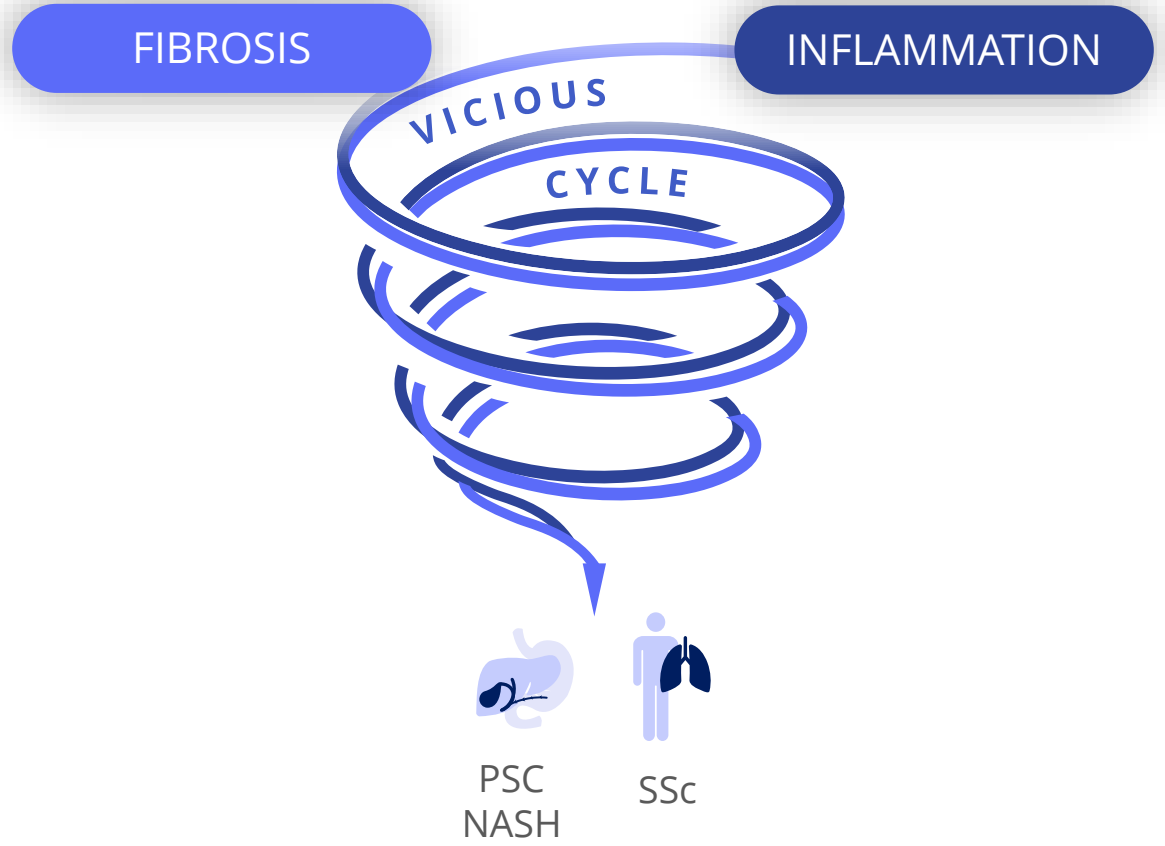
We have filed a registration statement (including a preliminary prospectus) on Form S-1 (File No. 333-269218), as amended, with the U.S. Securities and Exchange Commission (SEC) for the offering to which this presentation and the accompanying oral commentary relate. Before you invest, you should read 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.

Chemomab
THERAPEUTICS

Biotechnology company focused on fibro-inflammatory disease

- Unique target with disease modifying potential
- De-risked—extensive preclinical & early clinical data
- High unmet need—large commercial opportunities
- Two Phase 2 read-outs expected next year



CCL24: target central to biology of inflammation & fibrosis

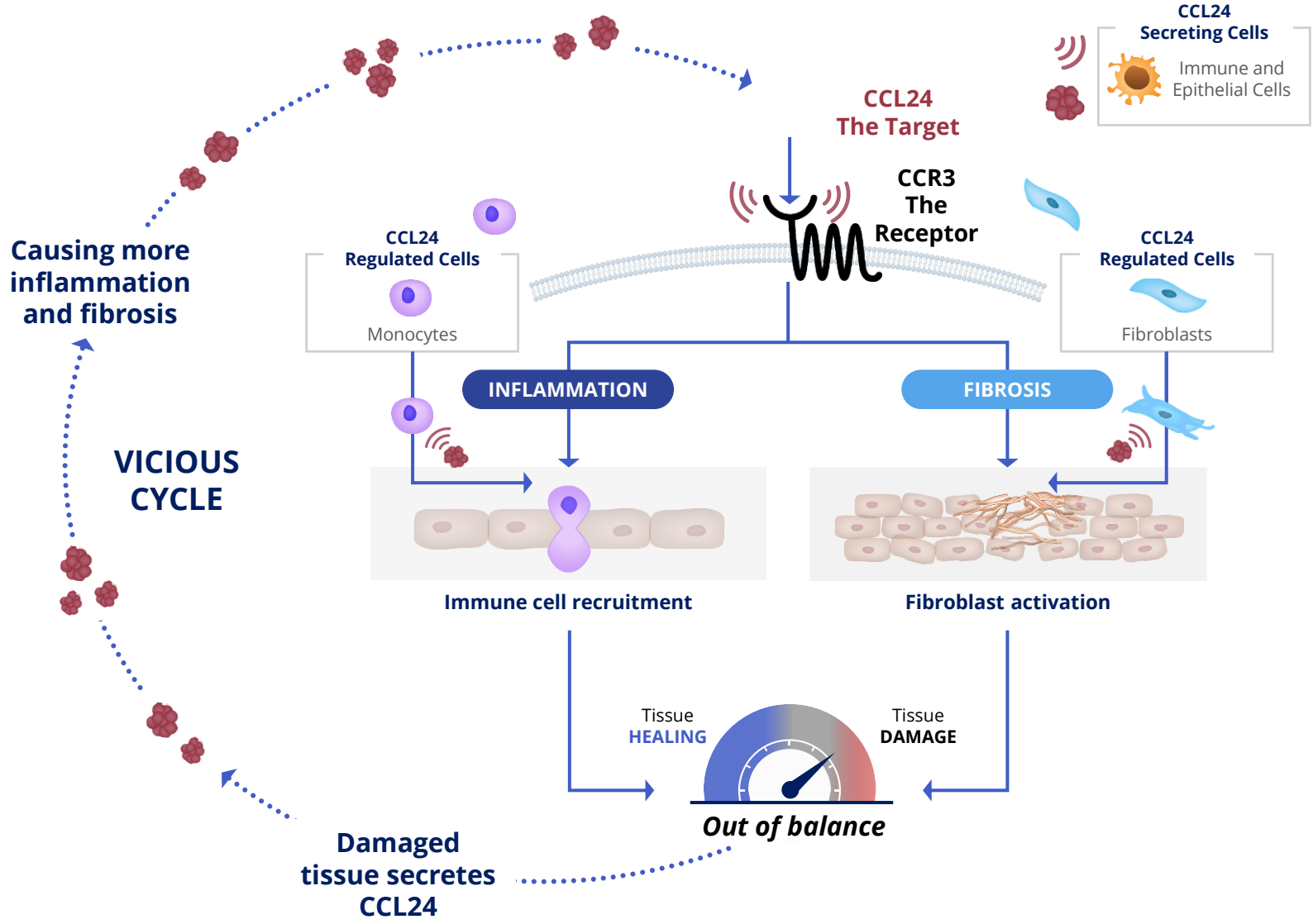
PSC-primary sclerosing cholangitis; NASH-nonalcoholic steatohepatitis; SSc-systemic sclerosis

CM-101: Pipeline-in-a-Product

ADDRESSING FIBRO-INFLAMMATORY DISEASES WITH HIGH UNMET NEED

PROGRAM	INDICATION	TARGET	DISCOVERY / PRECLINICAL / PHASE 1A / 1B / PHASE 2	MILESTONES
CM-101	**Liver Fibrosis Biomarkers** • Ph 1B **NAFLD** (IV/SC) • Ph 2A **NASH** (SC & Liver Fibrosis Biomarkers)	CCL24		Data Reported 2021 Completed—Data Reported 2023
	Primary Sclerosing Cholangitis	CCL24		Top-line Data Expected 2H 2024
	Systemic Sclerosis	CCL24		Expected to Initiate 1H 2023 Top-line Data Expected 2H 2024
	Fibro-inflammatory Diseases	CCL24		Reported Open-Label Lung Injury Study 4Q 2022
Next-Gen (Novel Targets)	**TBD**	Multiple		

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

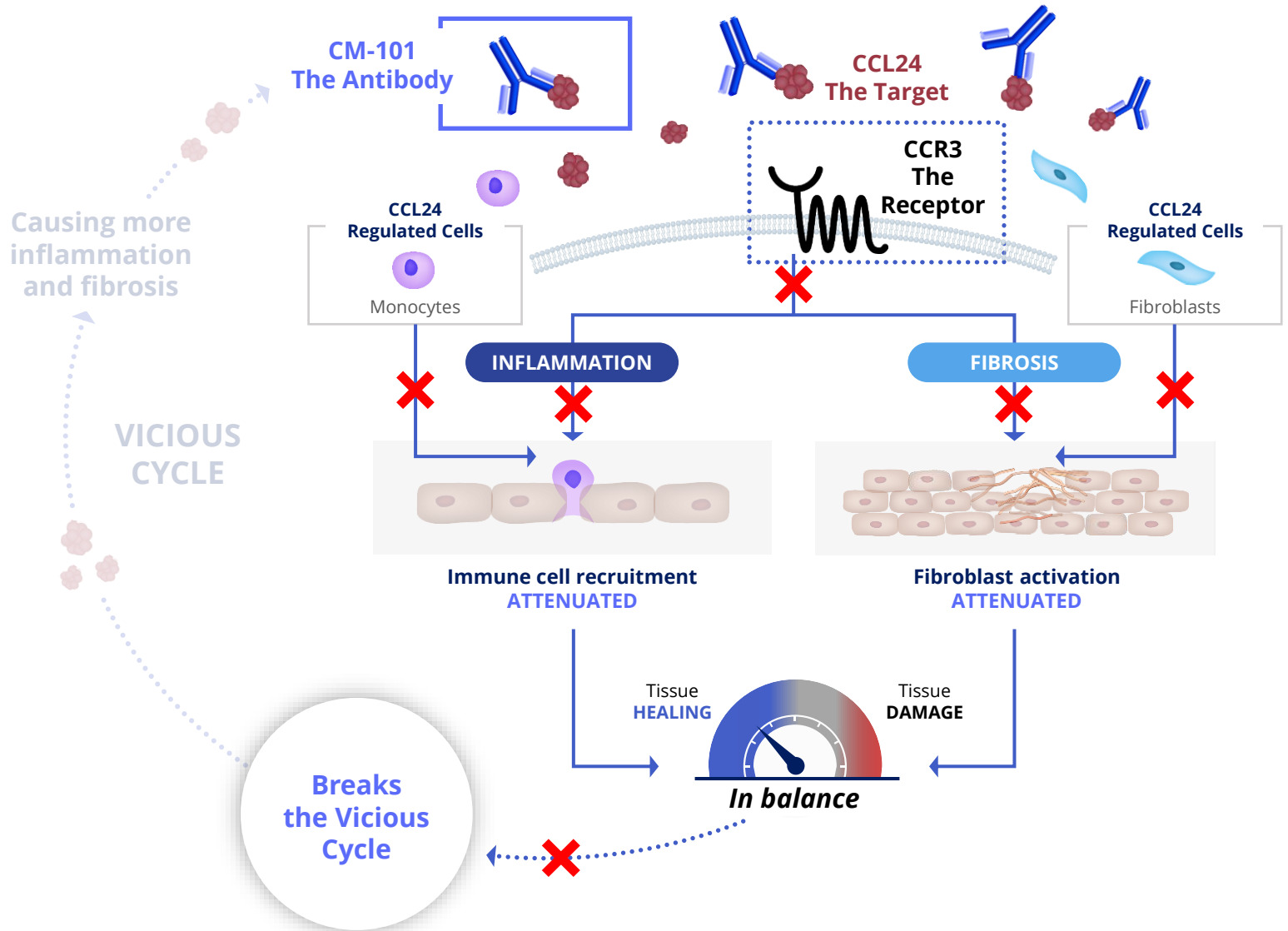


THE POWER OF CCL24

- **Dual Role in Fibrosis & Inflammation**
- **Differentiated Activity**
- **Low in Healthy Tissue; Elevated in Fibrotic Tissue**

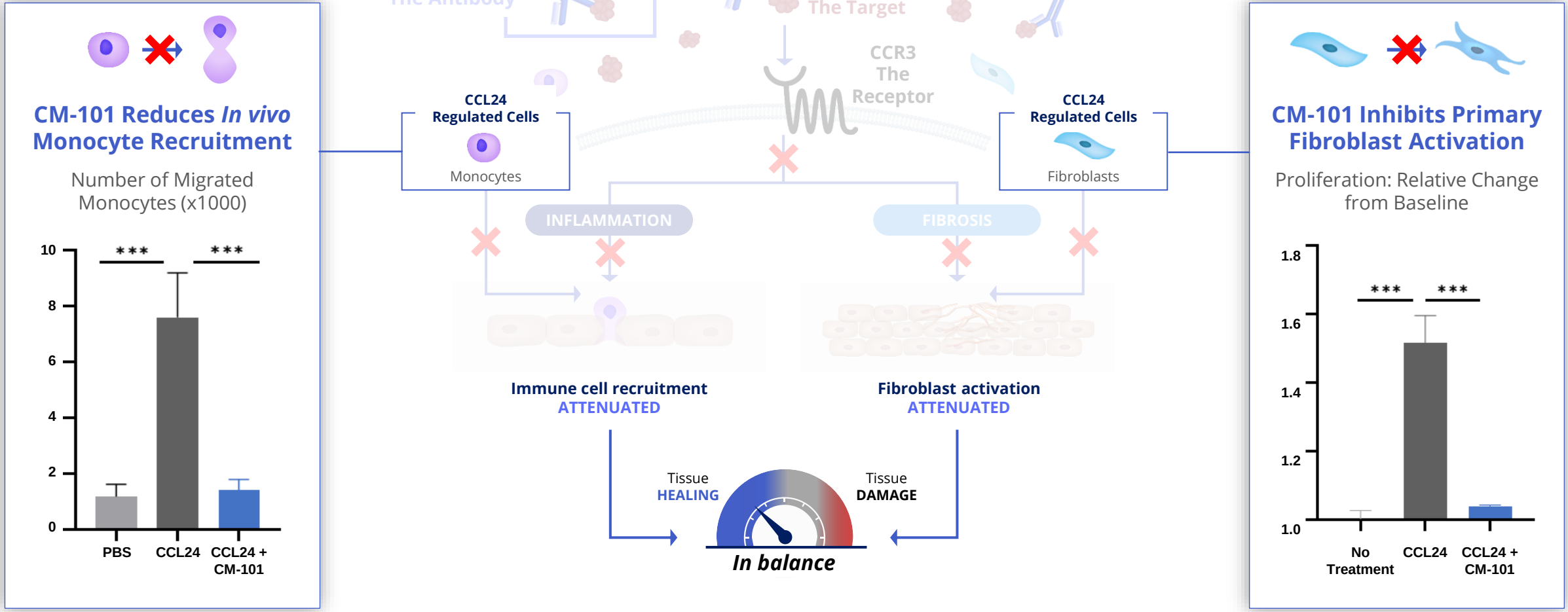
*Based on Chemomab preclinical *in vivo* & *in vitro* data-see Endnote 1

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



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

Preclinical Proof: First-in-Class CM-101 Neutralizes CCL24*



CM-101 Reduces *In vivo* Monocyte Recruitment

Number of Migrated Monocytes (x1000)

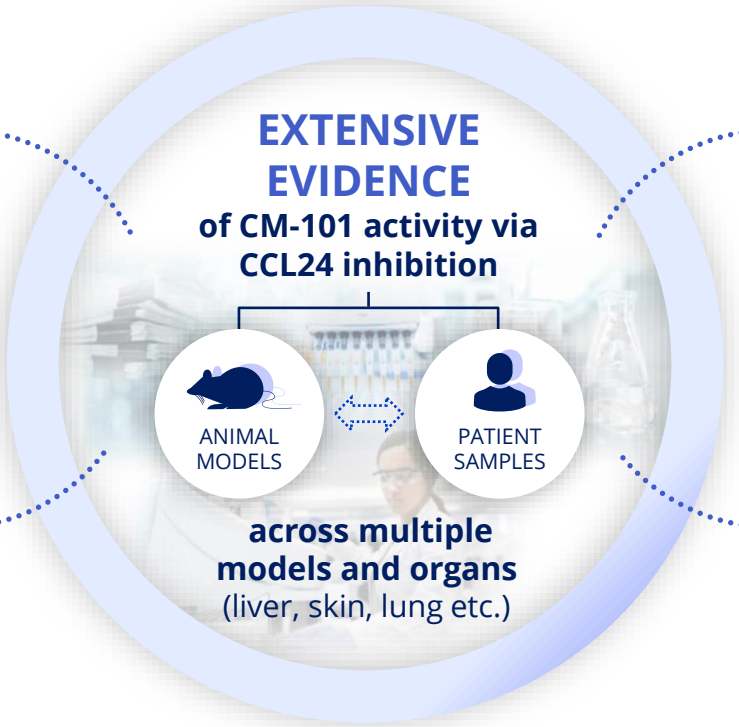
CM-101 Inhibits Primary Fibroblast Activation

Proliferation: Relative Change from Baseline

***p ≤.001 PBS-phosphate buffered saline

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

Broad & Diverse Preclinical Evidence De-risks Clinical Development



- Mechanism of Action
- CCL24 Target Validation In-vivo (Knockout Animal Models)
- CCL24 Target Validation Ex-vivo Patient Samples
- Toxicology & Safety

Proof of Concept: Animal Models Concordant with Patient Data

40-50 Person Years of Work **OVER** **~10** Calendar Years **USING** **~12** Different Animal Disease Models **AND** **300+** Patient Samples

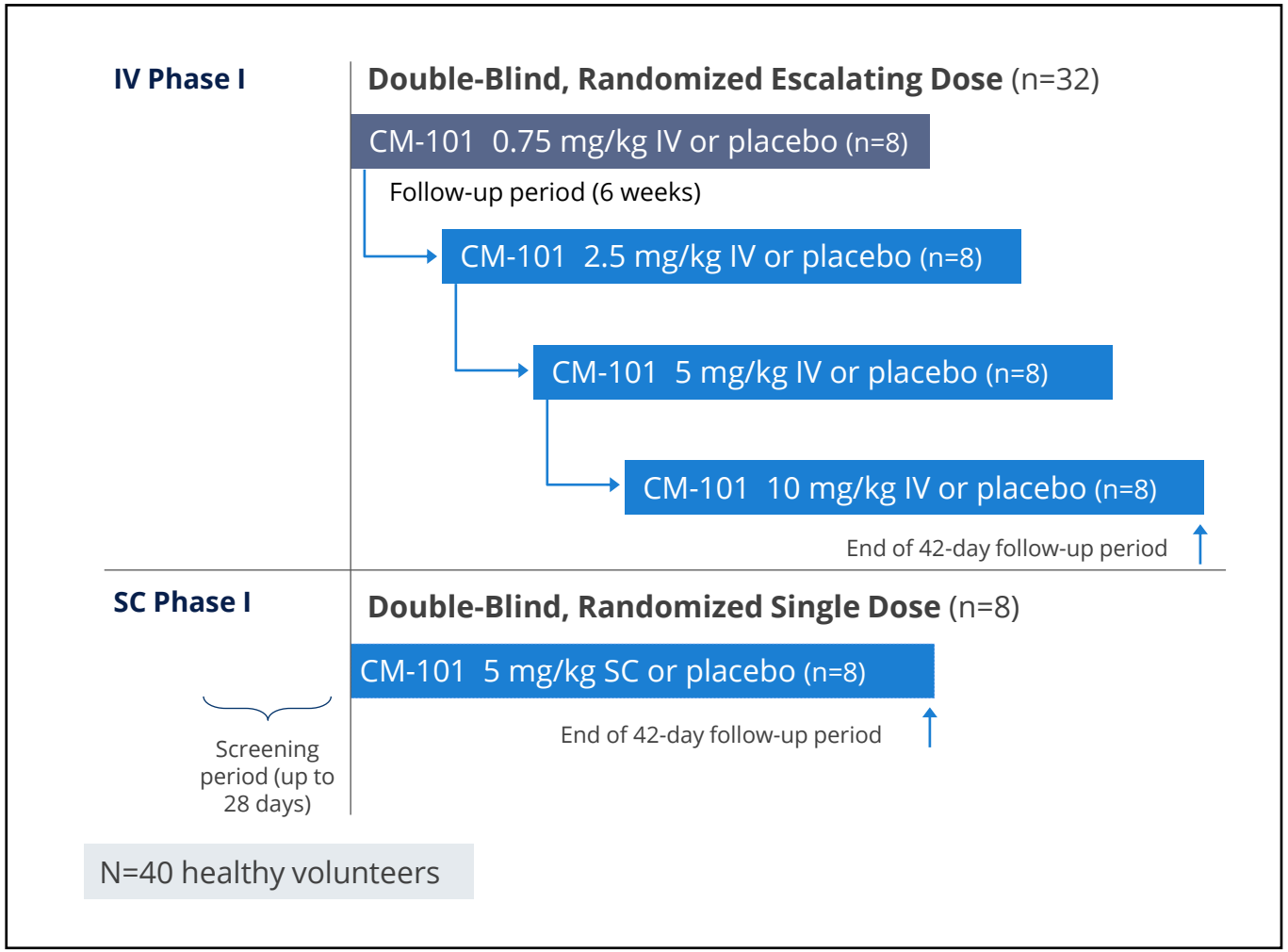
Sources: Chemomab published & unpublished data–see Endnote 2

CM-101 Appeared Safe & Well Tolerated in Phase 1a Trial

HEALTHY VOLUNTEERS

SINGLE ASCENDING DOSE STUDY

- CM-101 appeared safe and well tolerated up to 10 mg/kg
- Drug related AEs were mild with no SAEs
- Half-life supports 2-4-week dosing
- Dose-dependent target engagement*
- SC & IV formulations showed comparable target engagement & PK profiles



*Measured by serum CCL24 levels. AEs-adverse events; SAEs-serious adverse events; SC-subcutaneous; IV-intravenous; PK-pharmacokinetic

CM-101: Pipeline-in-a-Product

ADDRESSING FIBRO-INFLAMMATORY DISEASES WITH HIGH UNMET NEED

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Next-Gen (Novel Targets)	**TBD**	Multiple					

CM-101 Phase 1b Multiple Dose Safety Trial in NAFLD Patients

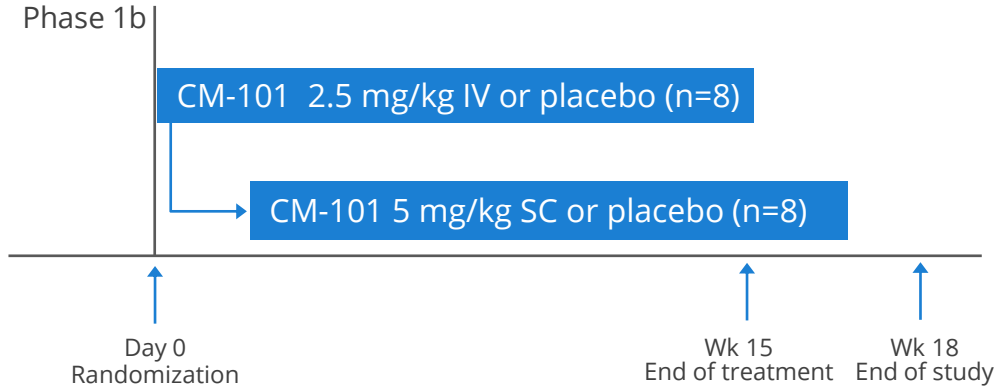
PATIENTS

CM-101 WAS WELL-TOLERATED; DEMONSTRATED FAVORABLE TARGET ENGAGEMENT & PK

- Patients had nonalcoholic fatty liver disease (NAFLD) & normal liver function
- Multiple CM-101 IV & SC administrations appeared safe & well-tolerated
- Most frequently reported AEs were mild with no drug-related SAEs
- Half-life supports 2-4-week dosing

Study Design:

Double-Blind, Randomized Escalating Dose (N=16)



- Doses - 2.5 mg/kg IV & 5 mg/kg SC
- 5 administrations; Q3W
- Primary endpoints - safety & tolerability
- Other parameters – target engagement, PK, changes in biomarkers

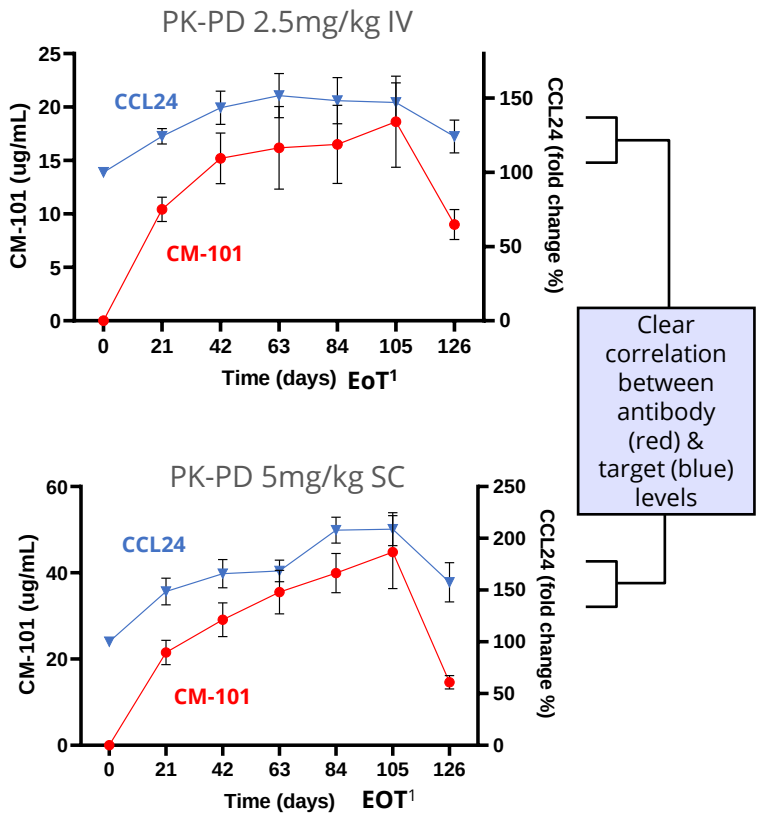
IV-intravenous; SC-subcutaneous; AEs-adverse events; SAEs–serious adverse events; Q3W-dosing once every 3 weeks; PK-pharmacokinetic

Phase 1b Anti-Fibrotic Data Support Proposed Mechanism

NAFLD PATIENT DATA PROVIDED FOUNDATION FOR FURTHER CLINICAL TRIALS

PATIENTS

PK-TARGET ENGAGEMENT



Dose Dependent PK-Target Engagement

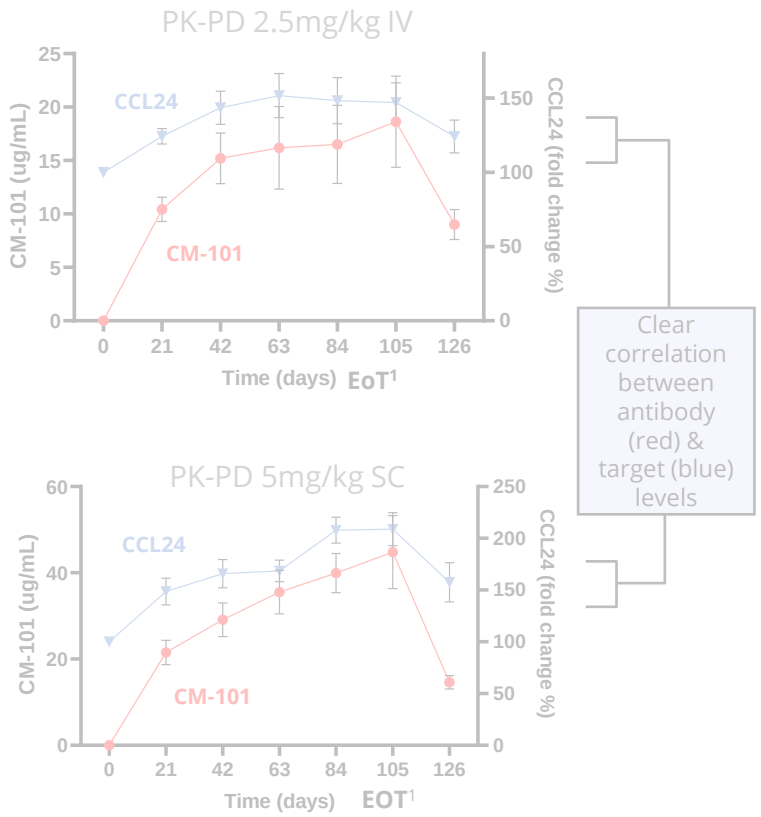
NAFLD-nonalcoholic steatohepatitis; n=12 active; PK-pharmacokinetic; PD-pharmacodynamic. [1] EoT-End of Treatment

Phase 1b Anti-Fibrotic Data Support Proposed Mechanism

NAFLD PATIENT DATA PROVIDED FOUNDATION FOR FURTHER CLINICAL TRIALS

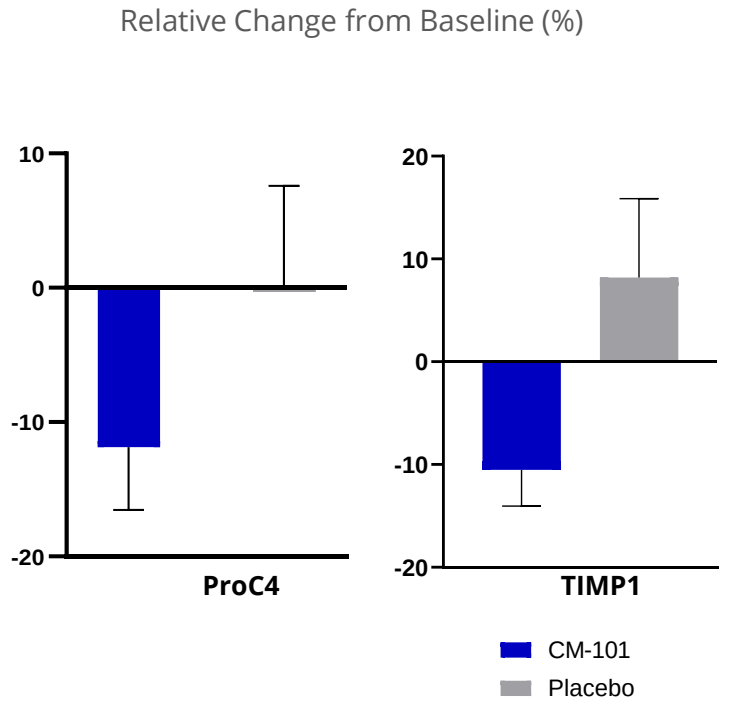
PATIENTS

PK-TARGET ENGAGEMENT



Dose Dependent PK-Target Engagement

FIBROSIS BIOMARKERS*



Concordant results across six additional fibrotic markers

Improved Fibrosis Biomarker Responses

NAFLD-nonalcoholic steatohepatitis; n=12 active; PK-pharmacokinetic; PD-pharmacodynamic. [1] EoT-End of Treatment

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

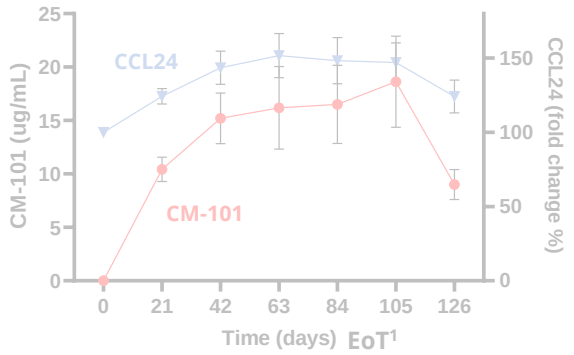
Phase 1b Anti-Fibrotic Data Support Proposed Mechanism

NAFLD PATIENT DATA PROVIDED FOUNDATION FOR FURTHER CLINICAL TRIALS

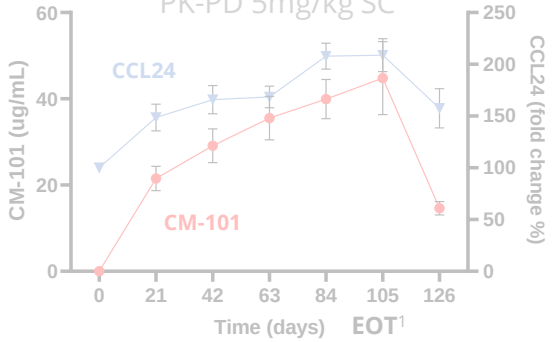
PATIENTS

PK-TARGET ENGAGEMENT

PK-PD 2.5mg/kg IV



PK-PD 5mg/kg SC

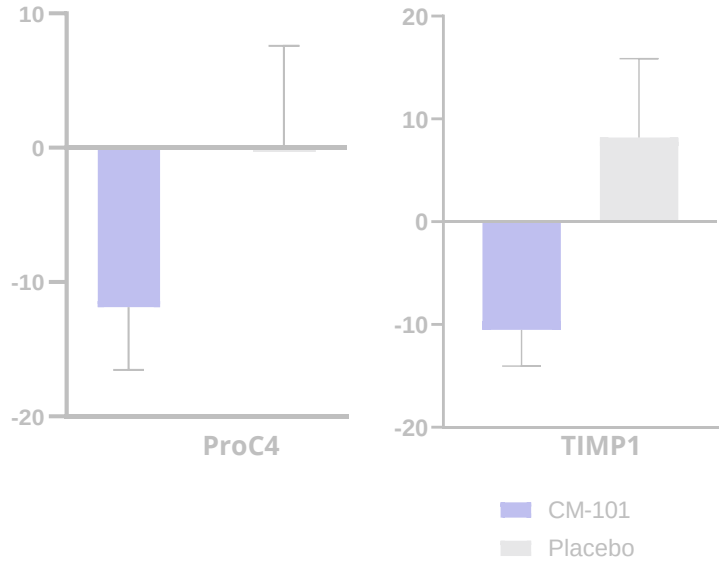


Clear correlation between antibody (red) & target (blue) levels

Dose Dependent PK-Target Engagement

FIBROSIS BIOMARKERS*

Relative Change from Baseline (%)



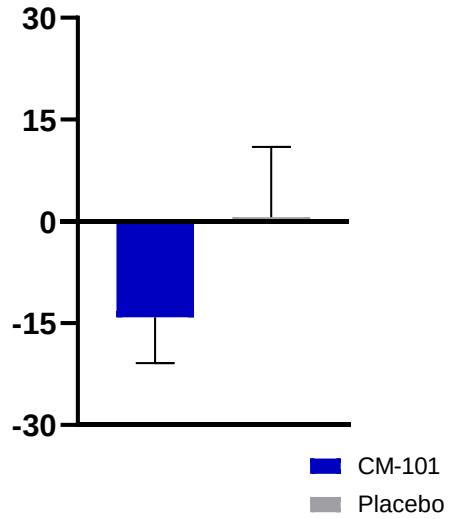
Concordant results across six additional fibrotic markers

Improved Fibrosis Biomarker Responses

LIVER STIFFNESS

Changes in FibroScan® Fibrosis Status+

Relative change from Baseline (%)



Reduced liver stiffness

Improved Physiological Measure

NAFLD-nonalcoholic steatohepatitis; n=12 active; PK-pharmacokinetic; PD-pharmacodynamic. [1] EoT-End of Treatment

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

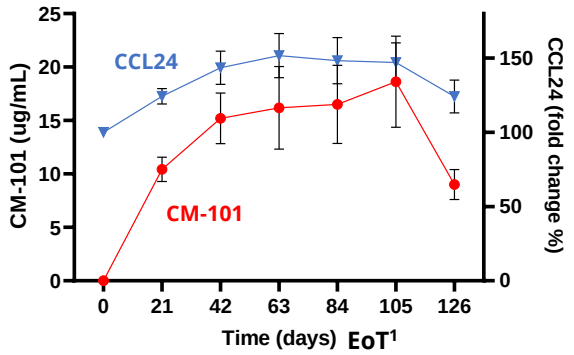
Phase 1b Anti-Fibrotic Data Support Proposed Mechanism

NAFLD PATIENT DATA PROVIDED FOUNDATION FOR FURTHER CLINICAL TRIALS

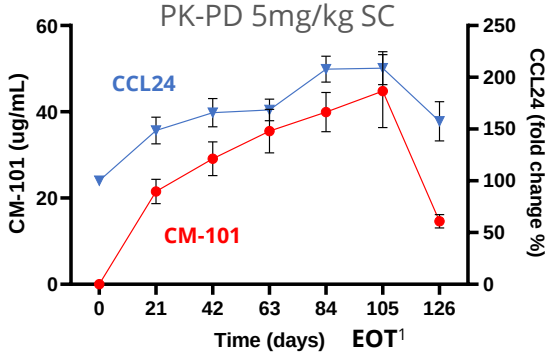
PATIENTS

PK-TARGET ENGAGEMENT

PK-PD 2.5mg/kg IV



PK-PD 5mg/kg SC

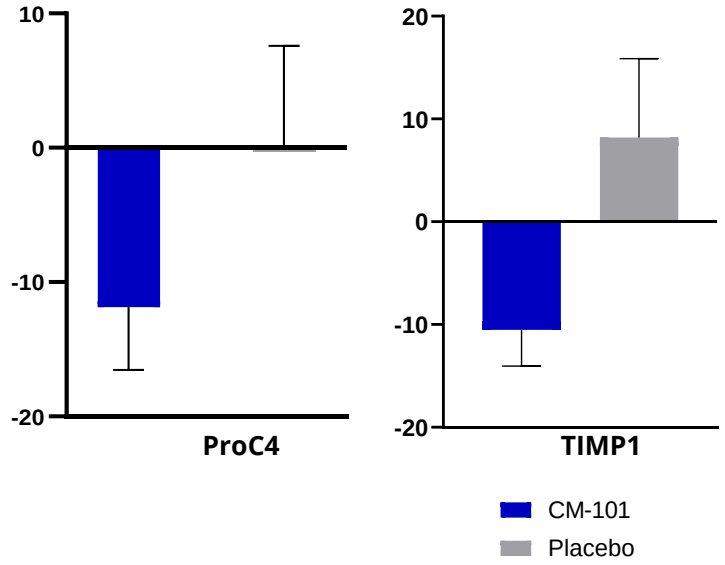


Clear correlation between antibody (red) & target (blue) levels

Dose Dependent PK-Target Engagement

FIBROSIS BIOMARKERS*

Relative Change from Baseline (%)



Concordant results across six additional fibrotic markers

Improved Fibrosis Biomarker Responses

LIVER STIFFNESS

Changes in FibroScan® Fibrosis Status+

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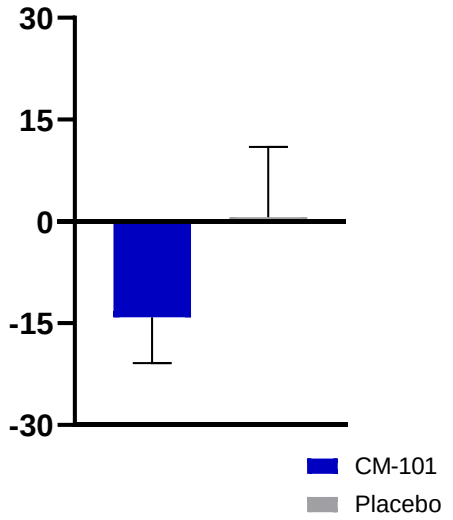

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*ProC4-procollagen 4; TIMP1-tissue inhibitor of metallopeptidase

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

CM-101 Phase 2a Liver Fibrosis Trial in NASH Patients

PATIENTS

APPEARED SAFE & WELL TOLERATED IN NASH PATIENTS WITH F1C-F3 FIBROSIS

Primary objective:

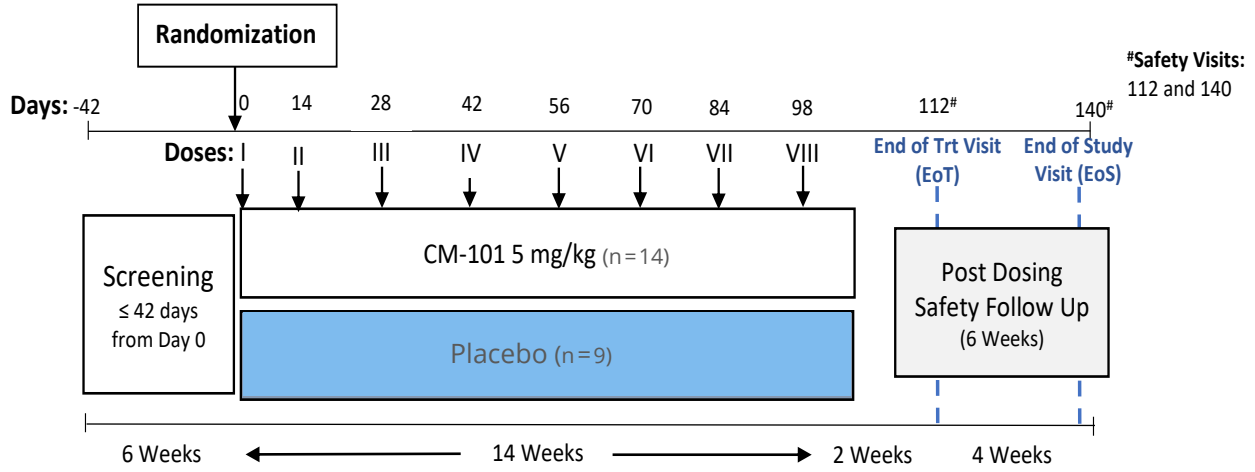
- Determine safety & tolerability of subcutaneous (SC) CM-101 in NASH patients

Secondary objectives:

- Explore PK & PD profiles & drug exposure
- Evaluate CM-101using liver fibrosis biomarkers & imaging
- Monitor development of Anti Drug Antibodies (ADAs)

Safety Results

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



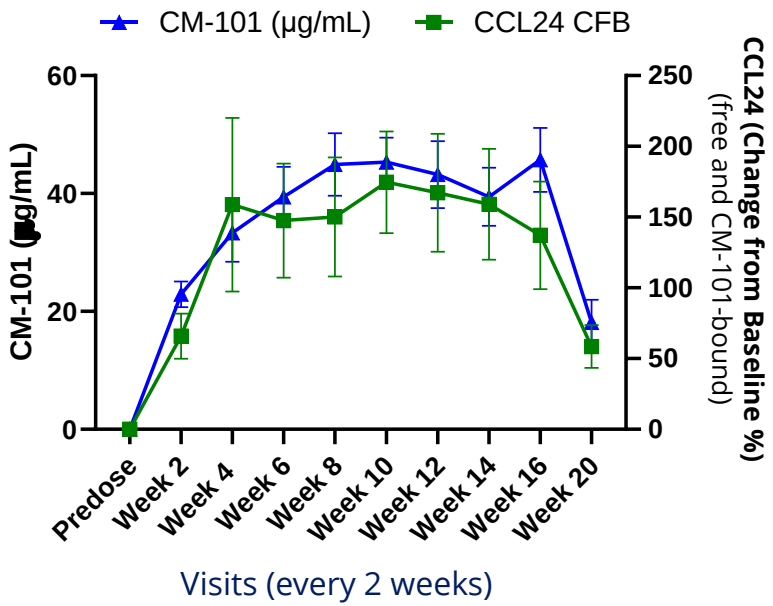
- Double-blinded, randomized placebo-controlled trial in 23 non-cirrhotic NASH patients with biopsy-confirmed F1c-F3 fibrosis
- 5mg/kg SC injection
- 8 administrations per subject; Q2W

NASH–nonalcoholic steatohepatitis; PK-pharmacokinetic; PD-pharmacodynamic; AEs–adverse events; SAEs–serious adverse events; Q2W-dosing once every 2 weeks

CM-101 Demonstrated Favorable PK-Target Engagement Profiles

PATIENTS

PK - TARGET ENGAGEMENT



- Drug exposure aligned with previous clinical studies
- Drug average half-life ~19 days

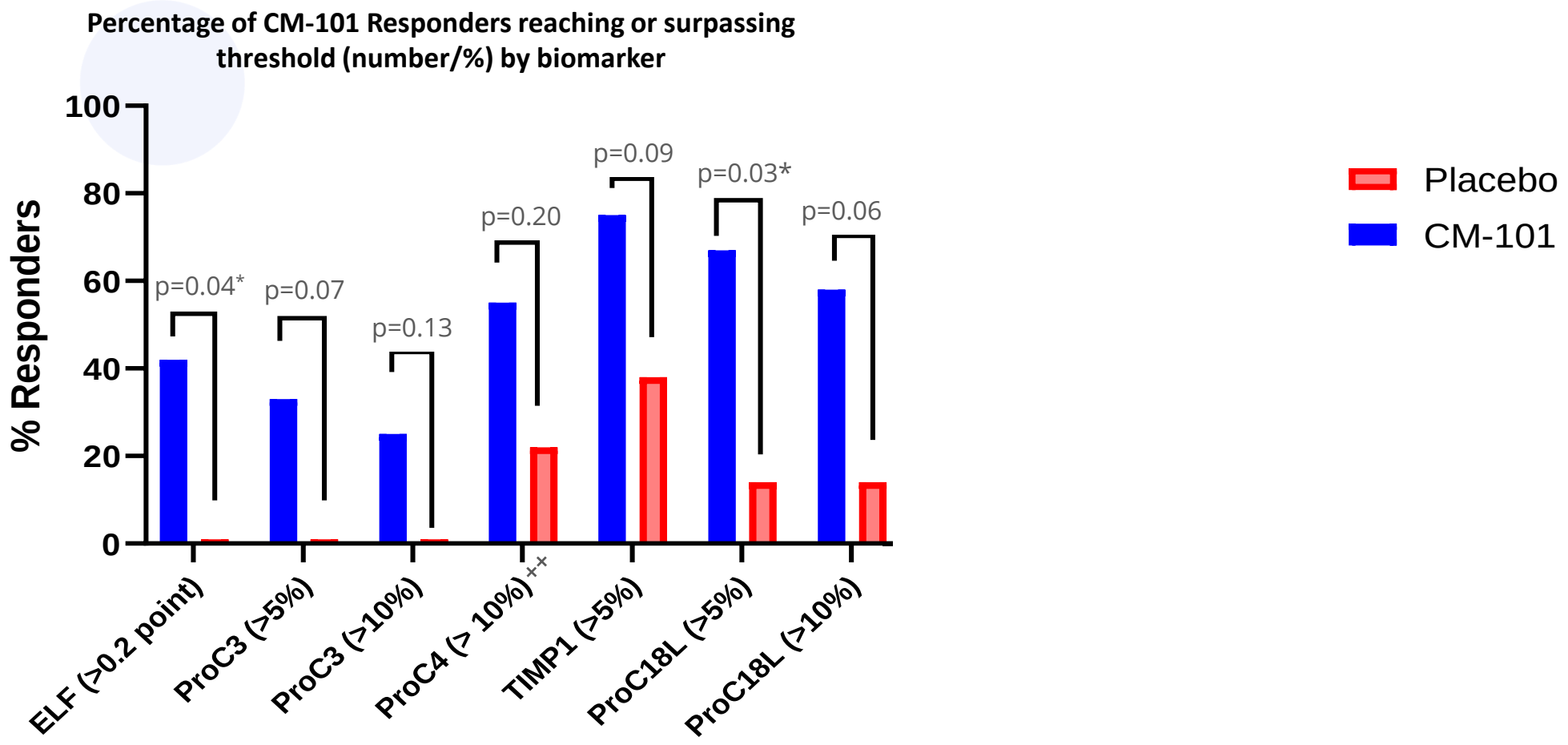
Robust PK - target engagement relationship

n=12 active; PK-pharmacokinetic; CFB-change from baseline

CM-101 Produced More Biomarker Responders

PATIENTS

POSITIVE FIBROSIS BIOMARKER RESPONSES

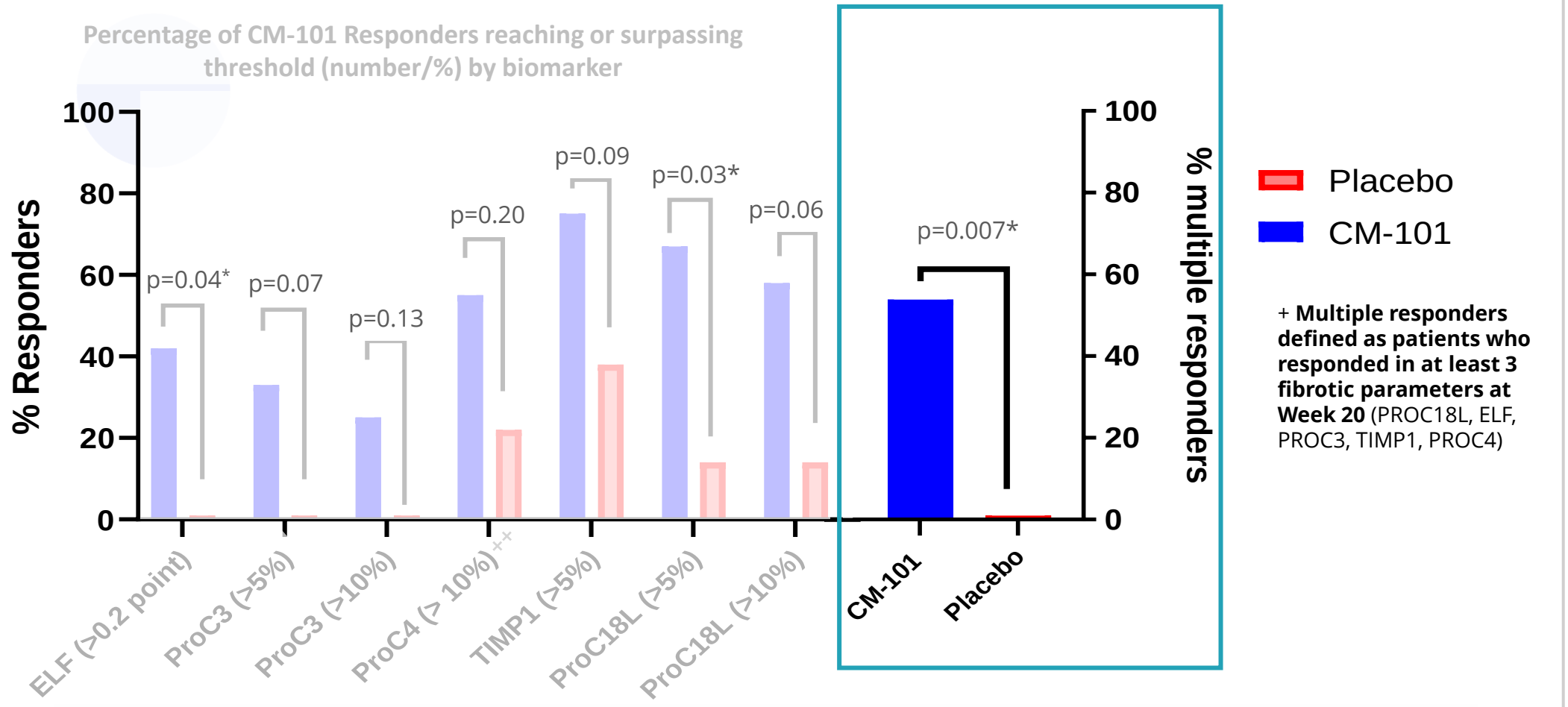


N=12 active, 8 placebo per protocol population analysis; *p ≤0.05; ++ Measured at week 16. The other biomarkers were measured at week 20.
ELF- enhanced liver fibrosis score, consists of TIMP-1, amino-terminal pro-peptide of type III procollagen (PIIINP) & hyaluronic acid; ProC3-procollagen 3; ProC4-procollagen 4; TIMP1-tissue inhibitor of metallopeptidase; ProC18L-procollagen 18L

CM-101 Produced More Multiple Biomarker Responders+

PATIENTS

POSITIVE FIBROSIS BIOMARKER RESPONSES



+ **Multiple responders defined as patients who responded in at least 3 fibrotic parameters at Week 20** (PROC18L, ELF, PROC3, TIMP1, PROC4)

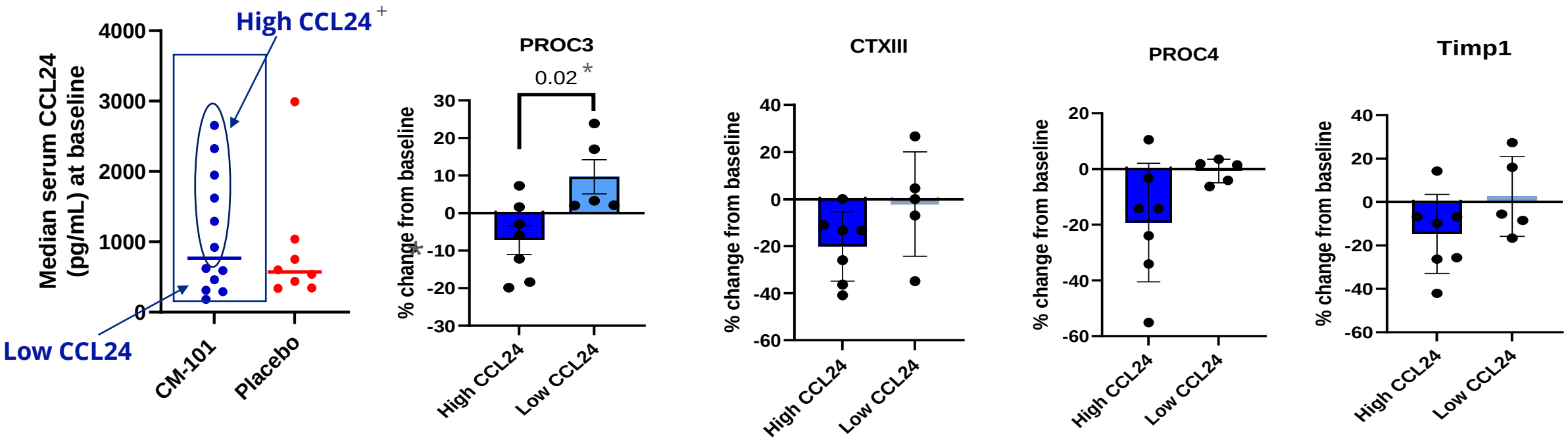
Majority of CM-101 patients were Multiple Responders—60% compared to zero placebo patients

N=12 active, 8 placebo per protocol population analysis; *p ≤0.05

Patients with High CCL24 at Baseline Had Greater Biomarker Responses

PATIENTS

CM-101-TREATED PATIENTS WITH HIGH CCL24 ALSO MORE LIKELY TO BE MULTIPLE RESPONDERS



Threshold defined as CCL24 baseline median of 610 pg/ml

+ High CCL24 defined as >610 pg/mL; Low CCL24 defined as <610 pg/mL

Provides further evidence of role of CCL24 in fibrotic liver disease pathophysiology

N=12 active, 8 placebo per protocol population analysis; *p ≤0.05; ProC3-procollagen 3;
CTXIII-cross linked C-telopeptide of type III collagen; ProC4-procollagen 4; TIMP1-tissue inhibitor of metallopeptidase

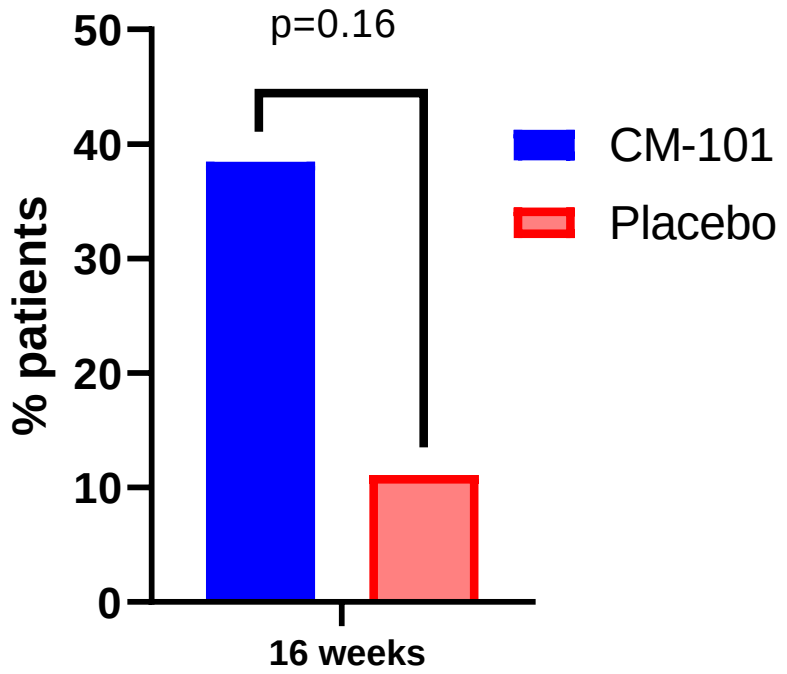
CM-101 Improved Liver Stiffness Score

EVIDENCE OF POSITIVE PHYSIOLOGICAL EFFECTS IN THE LIVER

PATIENTS

CHANGE IN FIBROSCAN®

Fibrosis Grade (reduction of at least 1 grade)



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

Fibrosis Grading by FibroScan (kPa)

N=13 active, 8 placebo per protocol population analysis; kPa-kilopascals

CM-101: Pipeline-in-a-Product

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Next-Gen (Novel Targets)	**TBD**	Multiple		

Anti-Fibrotic Mechanisms Demonstrated in Lung Injury Patients

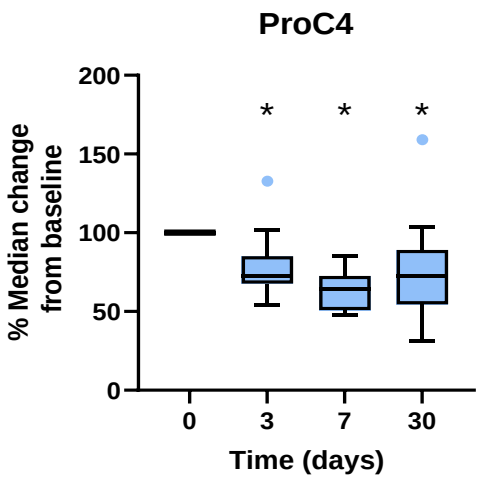
CM-101 REDUCED INFLAMMATION/FIBROGENESIS BIOMARKERS ASSOCIATED WITH SYSTEMIC SCLEROSIS

PATIENTS

Single arm, open label investigator study of CM-101 single 10mg/kg IV dose in 16 COVID patients with severe lung injury[+,++]

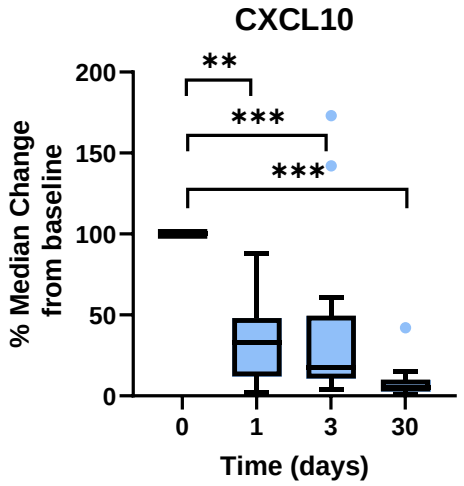
CM-101 Demonstrated Strong & Rapid Reductions in Biomarkers for:

Fibrosis/Fibrogenesis



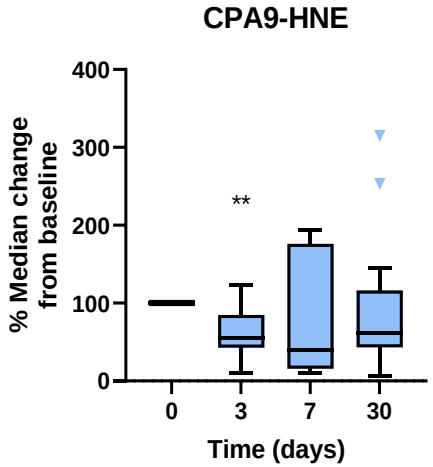
Considered Early Marker of Fibrosis

Lung Inflammation



Elevated in Patients with Systemic Sclerosis

Neutrophil Activity



Elevated in Progressive Liver & Pulmonary Fibrosis

Further supports potential to reduce inflammation & fibrosis in varied organs including lung, liver & skin

[+]Study rationale—SSc expert investigator viewed COVID lung injury as a model for lung involvement in systemic sclerosis

[++]*Treatment with CM-101 Reduced Inflammatory & Fibrotic Biomarkers in Patients with COVID-19-Derived Lung Damage*, A. Mor, Union World Conference on Lung Health 2022

***p ≤0.001; **p ≤0.01; *p ≤0.05 ProC4-procollagen 4; CXCL10-C-X-C motif chemokine ligand 10; CPA9-HNE-human neutrophil elastase-derived calprotectin neo-epitope 9

Anti-Fibrotic Mechanisms Demonstrated in Early Clinical Studies

CM-101 REDUCED FIBRO-INFLAMMATORY BIOMARKERS IN NAFLD, NASH & LUNG INJURY

CM-101 Administered to >70 Subjects in 4 Clinical Trials Appears Safe & Well Tolerated

- Safety demonstrated in healthy volunteers and in 3 studies in patients
- AEs mostly mild; No drug-related SAEs
- Consistent PK & target engagement profiles
- No anti-drug antibodies detected

CM-101 Improved Results in Multiple Readouts in 3 Clinical Trials in Patients

- Reduced fibrogenesis-related biomarkers in fatty liver, liver fibrosis, NASH & severe lung inflammation
- Demonstrated anti-inflammatory effects in NASH & acute lung inflammation
- Reduced physiological endpoint of liver stiffness

Demonstrated CM-101's anti-fibrotic & anti-inflammatory effects in varied organs & diseases

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

CM-101: Pipeline-in-a-Product

ADDRESSING FIBRO-INFLAMMATORY DISEASES WITH HIGH UNMET NEED

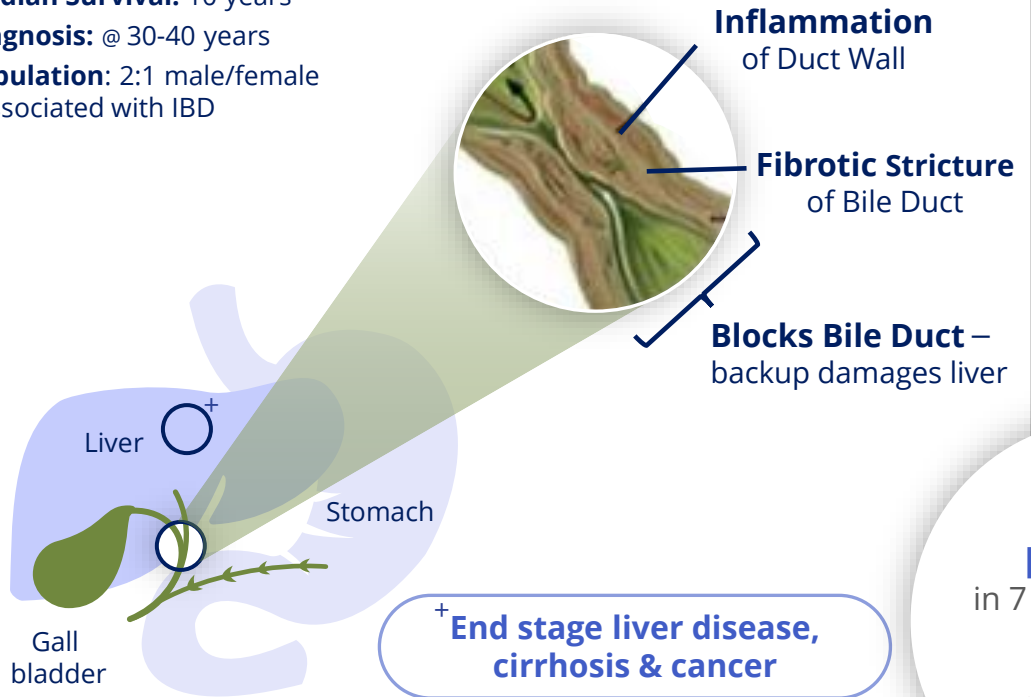
PROGRAM	INDICATION	TARGET	DISCOVERY	PRECLINICAL	PHASE 1A / 1B	PHASE 2	MILESTONES
CM-101	**Liver Fibrosis Biomarkers** • Ph 1B **NAFLD** (IV/SC) • Ph 2A **NASH** (SC & Liver Fibrosis Biomarkers)	CCL24					Data Reported 2021 Completed—Data Reported 2023
	Primary Sclerosing Cholangitis	CCL24					Top-line Data Expected 2H 2024
	Systemic Sclerosis	CCL24					Expected to Initiate 1H 2023 Top-line Data Expected 2H 2024
	Fibro-inflammatory Diseases	CCL24					Reported Open-Label Lung Injury Study 4Q 2022
Next-Gen (Novel Targets)	**TBD**	Multiple					

Primary Sclerosing Cholangitis (PSC): High Morbidity and Mortality

Rare Chronic Liver Disease—No Disease Modifying Therapies

DEVASTATING DISEASE CAUSED BY INFLAMMATION & FIBROSIS

Median Survival: 10 years**
Diagnosis: @ 30-40 years
Population: 2:1 male/female
Associated with IBD



+End stage liver disease, cirrhosis & cancer

~77K patients
in 7 major markets

>$1B
est. commercial opportunity

INTEREST BY MAJOR PHARMA*

Fibro-Inflammatory Liver Diseases

Takeda
abbvie
Genentech
Mitsubishi Tanabe Pharma
Pfizer

Expressed Pharma BD Interest

** With no intervention. Liver transplant only therapy with some curative potential

Source: See Endnote 3. 7 Major Markets – US, EU (UK, Germany, France, Italy, Spain), Japan

*Examples Based on Publicly-Available Information
BD-business development

CCL24 Plays a Key Role in PSC

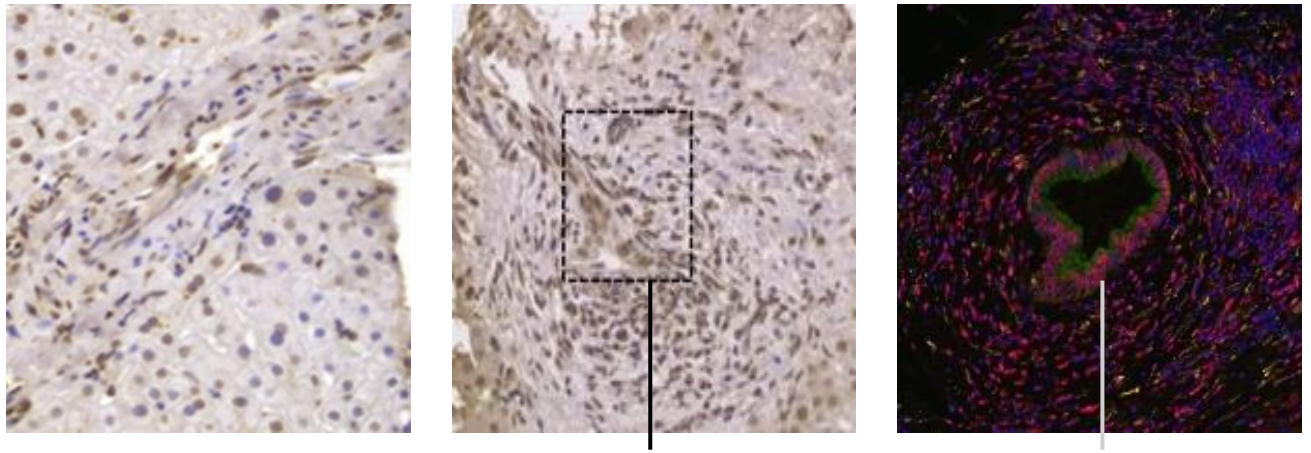
DATA FROM PATIENT SAMPLES SUPPORTS CLINICAL RELEVANCE OF CCL24 IN PSC

PATIENT SAMPLES

CCL24 LEVEL IN HEALTHY VS PSC PATIENT LIVER TISSUE[1]

Healthy Liver | **PSC Livers**

CCL24 Staining



CCL24 (brown) overexpressed in damaged bile duct area

CCL24 (red) overexpressed in bile epithelial cells (green) and immune cells (yellow)

CCL24 expression is significantly & selectively elevated in PSC livers

1. Collaboration with RFH, UK
ELF – Enhanced Liver Fibrosis score; ALP – alkaline phosphatase
Chemomab unpublished data

CCL24 Plays a Key Role in PSC

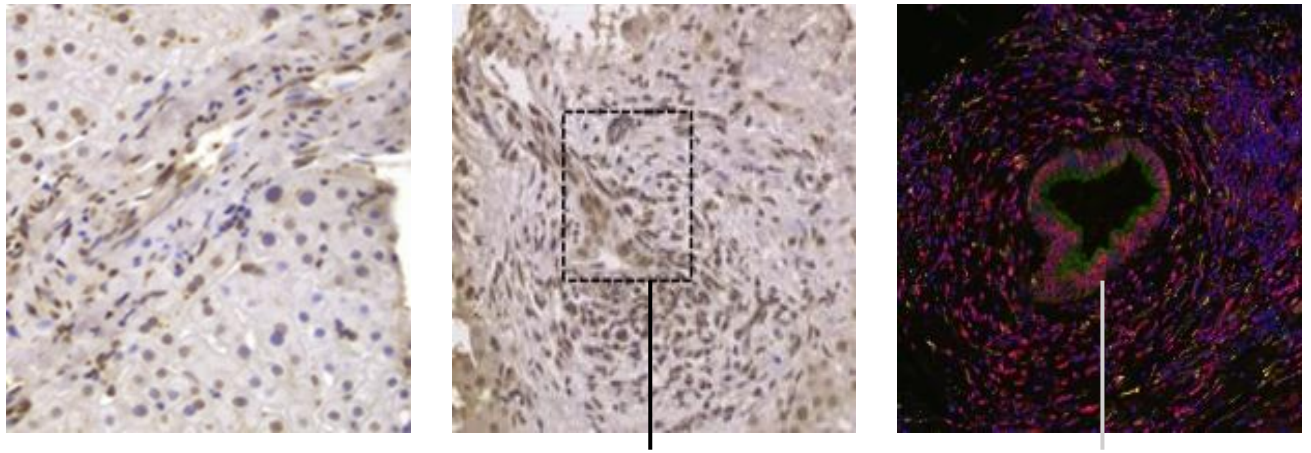
DATA FROM PATIENT SAMPLES SUPPORTS CLINICAL RELEVANCE OF CCL24 IN PSC

PATIENT SAMPLES

CCL24 LEVEL IN HEALTHY VS PSC PATIENT LIVER TISSUE[1]

Healthy Liver | **PSC Livers**

CCL24 Staining



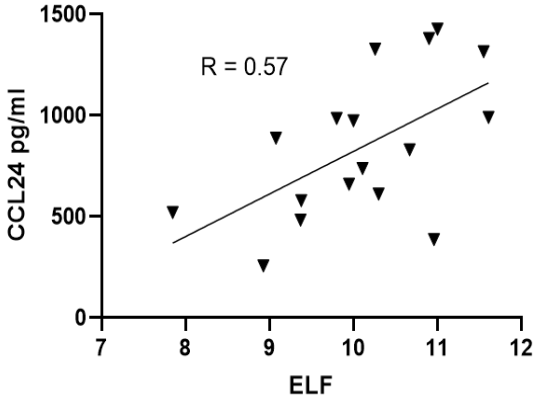
CCL24 (brown) overexpressed in damaged bile duct area

CCL24 (red) overexpressed in bile epithelial cells (green) and immune cells (yellow)

CCL24 expression is significantly & selectively elevated in PSC livers

CCL24 SERUM LEVEL IN PSC PATIENTS

ALP > 1.5 Upper Limit of Normal



Enhanced Liver Fibrosis (ELF) test reflects fibrosis and predicts clinical outcomes in PSC[1]

CCL24 correlates with ELF in PSC patients with elevated ALP

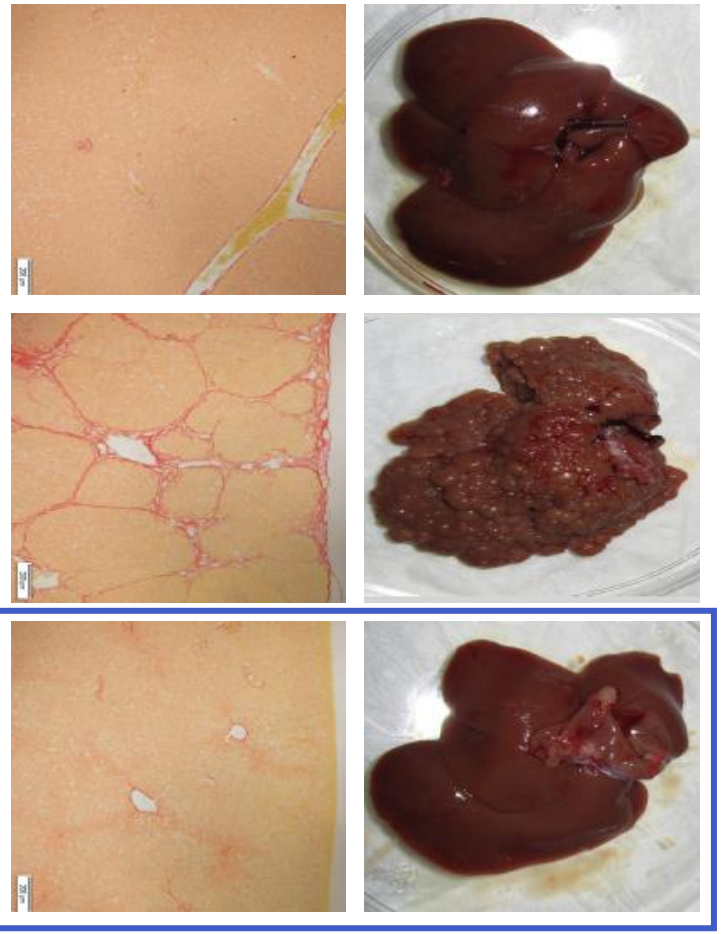
1. Collaboration with RFH, UK
ELF – Enhanced Liver Fibrosis score; ALP – alkaline phosphatase
Chemomab unpublished data

CM-101 Reduces Liver Fibrosis by >80% in TAA Model

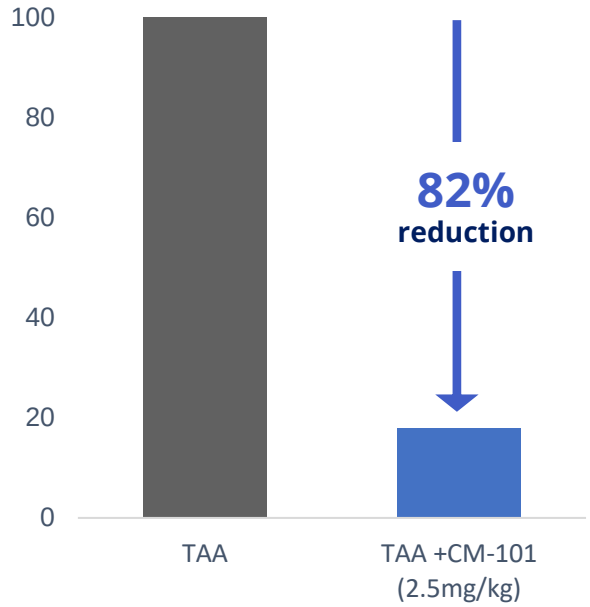
ANIMAL MODELS (TAA RAT)

RAT MODEL LIVERS

SIRIUS RED (COLLAGEN)



% FIBROSIS
(SIRIUS RED STAINING)



Pro-fibrotic Genes
Col1A1
Col3A1
TIMP1
ACTA2
TGF-β

Liver Enzymes
ALT
AST
ALP

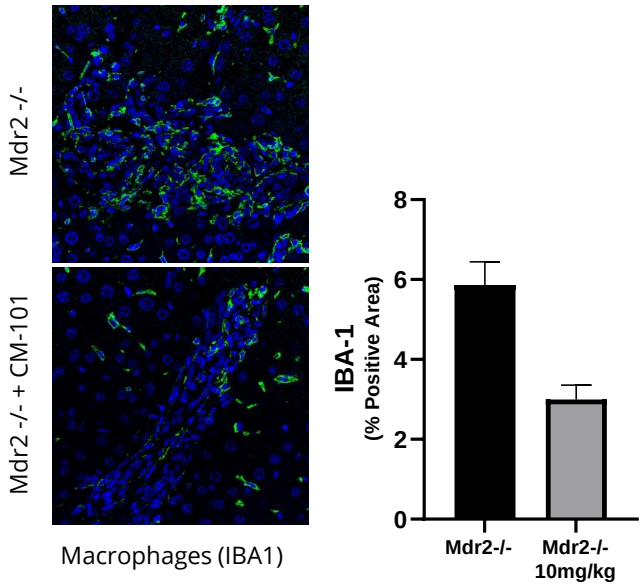
Dramatically reduced liver collagen in rat model with established liver fibrosis

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

CM-101 Reduces Liver Injury and Fibrosis in PSC Animal Model

ANIMAL MODELS
MDR 2 MOUSE
KNOCKOUT

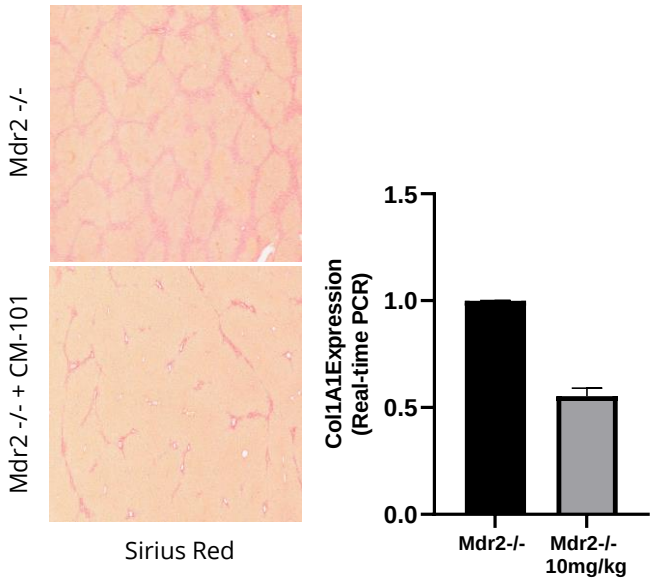
REDUCED MACROPHAGE NUMBER



Macrophages (IBA1)

Reduction in INFLAMMATION

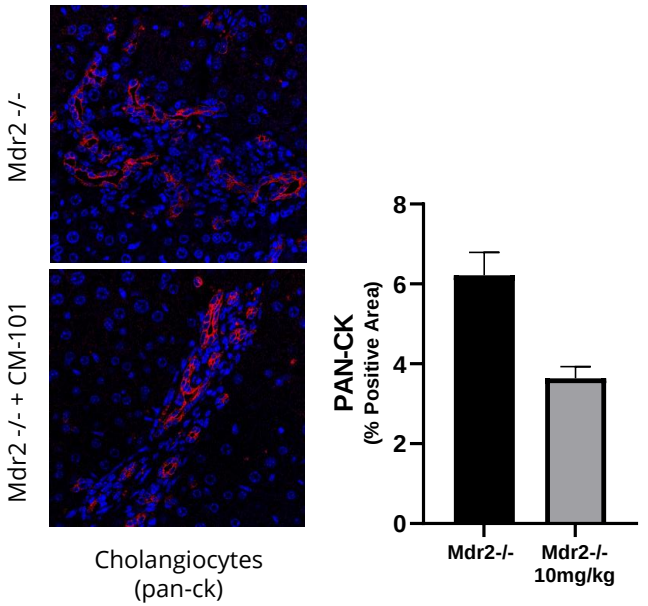
ATTENUATED LIVER COLLAGEN DEPOSITION



Sirius Red

Reduction in FIBROSIS

REDUCED ABERRANT BILE EPITHELIAL PROLIFERATION



Cholangiocytes
(pan-ck)

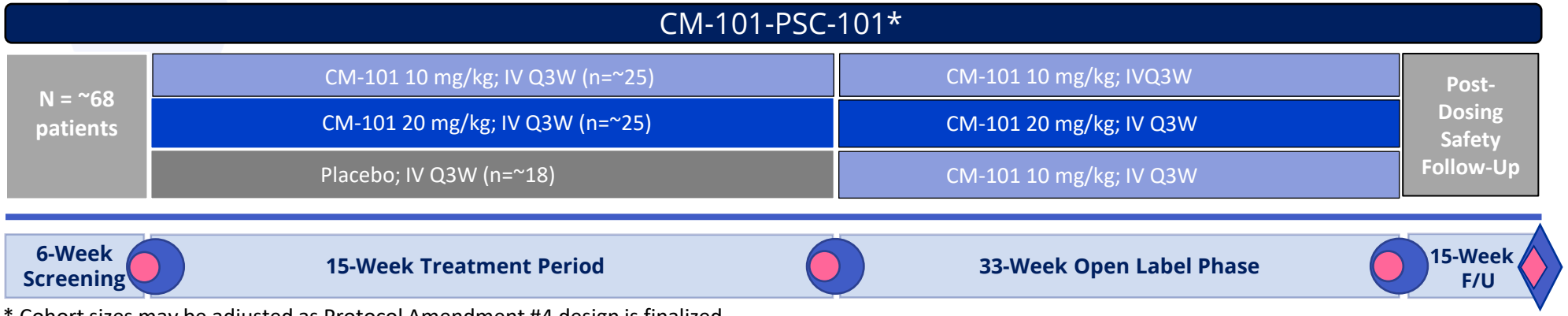
Main driver of DISEASE PATHOLOGY

CM-101 interferes with core pathways that drive PSC

Chemomab unpublished data

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
- Stable UDCA treatment allowed

Outcome Measures

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

Top-line results expected 2H 2024

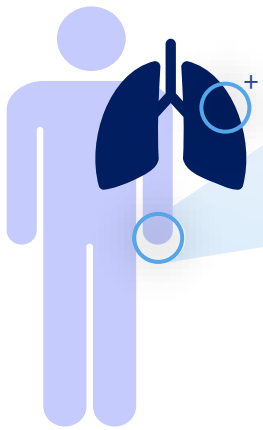
PSC-primary sclerosing cholangitis; Q3W-once every 3 weeks; IV-intravenous; ALP-alkaline phosphatase; IBD-inflammatory bowel disease; UDCA-ursodeoxycholic acid; ELF–enhanced liver fibrosis score; AST-aspartate aminotransferase; Pro-C3-type lll collagen biomarker; Pro-C5-type V collagen biomarker; FibroScan-measure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

Systemic Sclerosis (SSc): Most Lethal Systemic Rheumatic Disease

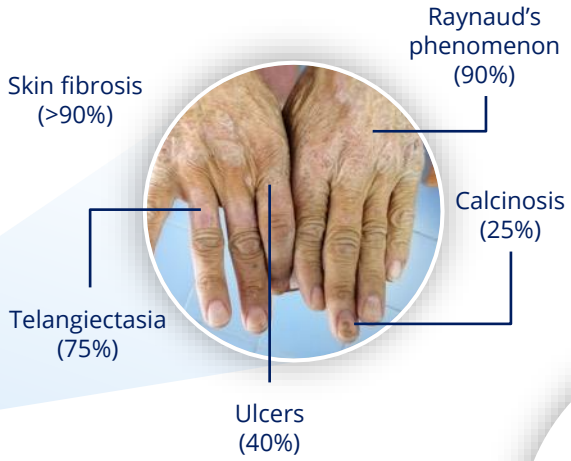
Rare Autoimmune Rheumatic Disease—No Disease Modifying Therapy

DEVASTATING DISEASE CAUSED BY INFLAMMATION, FIBROSIS & VASCULOPATHY

Median Survival: 10 years**
Diagnosis: @ 30-50 years
Population: 3:1 female/male
Current Rx: Nintedanib & tocilizumab only treat pulmonary symptoms; **NOT** disease modifying



SKIN: Most evident early symptom



‡Lung Involvement Leading Cause of Death**

INTEREST BY MAJOR PHARMA*

Fibro-Inflammatory Vascular/Pulmonary Diseases

Boehringer Ingelheim
Roche
AMGEN
sanofi
Mitsubishi Tanabe Pharma

~170K patients
in 7 major markets

>$1.5B
est. commercial opportunity

Expressed Pharma BD Interest

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

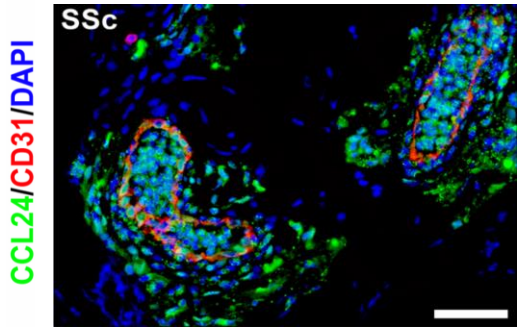
*Examples Based on Publicly-Available Information
BD-business development

CCL24: A Critical Node Potentiating Systemic Sclerosis

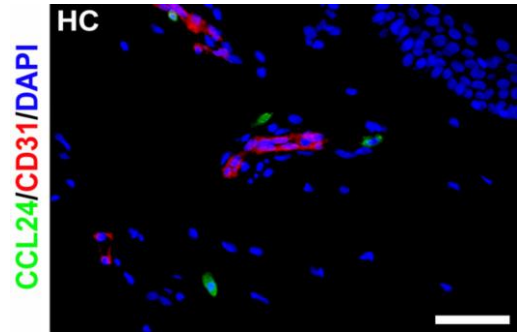
PATIENT SAMPLES

PATIENT SAMPLE DATA SUPPORTS THERAPEUTIC UTILITY OF BLOCKING CCL24 IN SYSTEMIC SCLEROSIS

SSc Skin

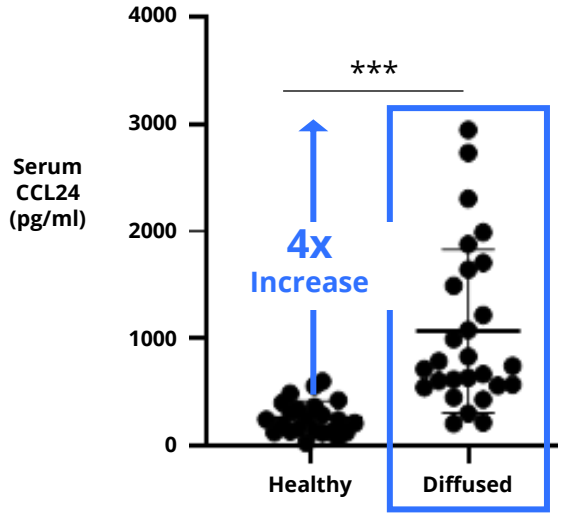


Healthy Skin



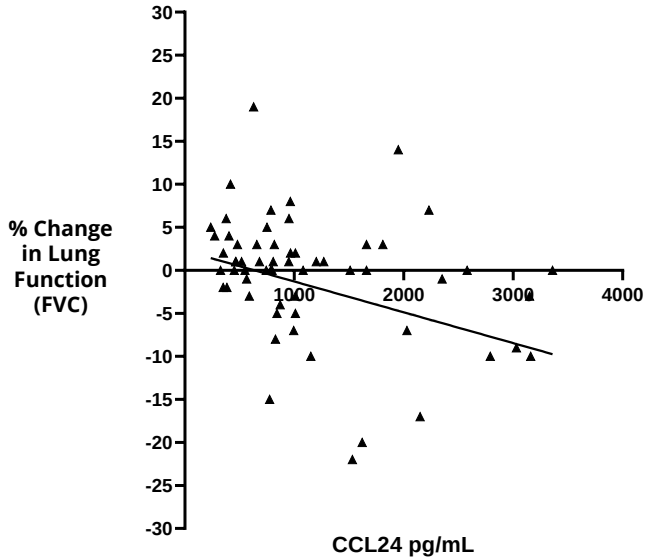
Collaboration with University of Florence

CCL24 SERUM LEVELS



CCL24 overexpressed in serum samples of dSSc patients compared to healthy

CCL24 SERUM LEVEL vs DISEASE PROGRESSION



CCL24 serum level correlates with worsening of lung function

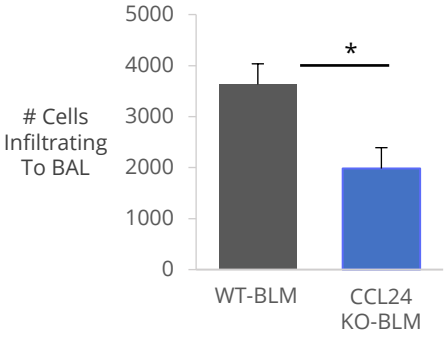
dSSc – diffuse systemic sclerosis; Mor A et al., Annals of Rheumatic Diseases, 2019
*** p ≤0.001

Target Validation: CCL24 Gene Knockout Ameliorates Induced SSc

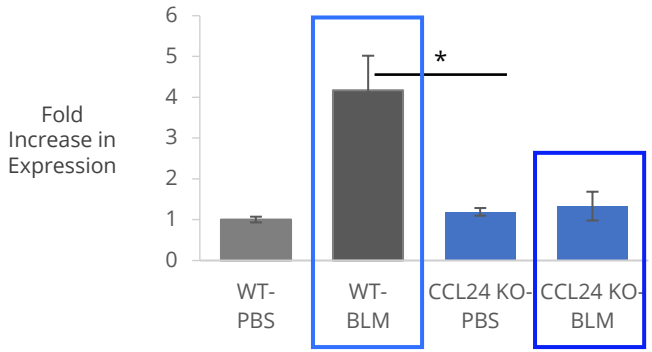
REDUCES INDUCED SSC-LIKE INFLAMMATION & FIBROSIS IN LUNG & SKIN IN MOUSE MODEL

ANIMAL MODELS
SSc BLEOMYCIN
MOUSE MODEL

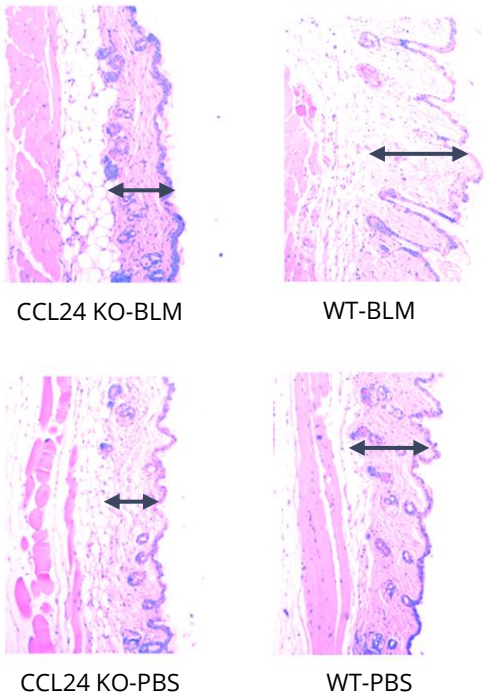
Immune Cell Lung Infiltration

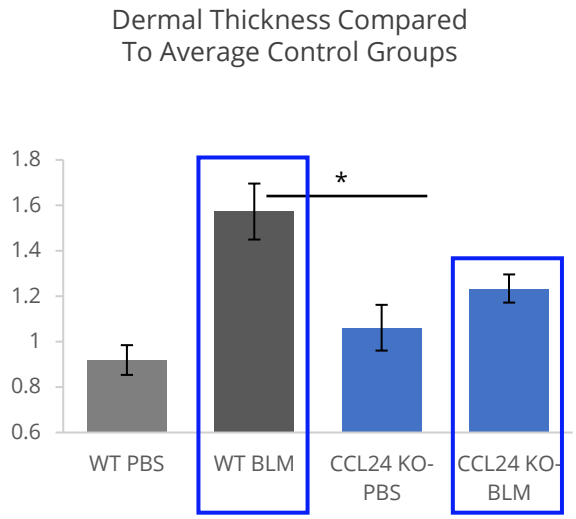


α SMA Expression in Skin Lesions



Dermal Thickness**



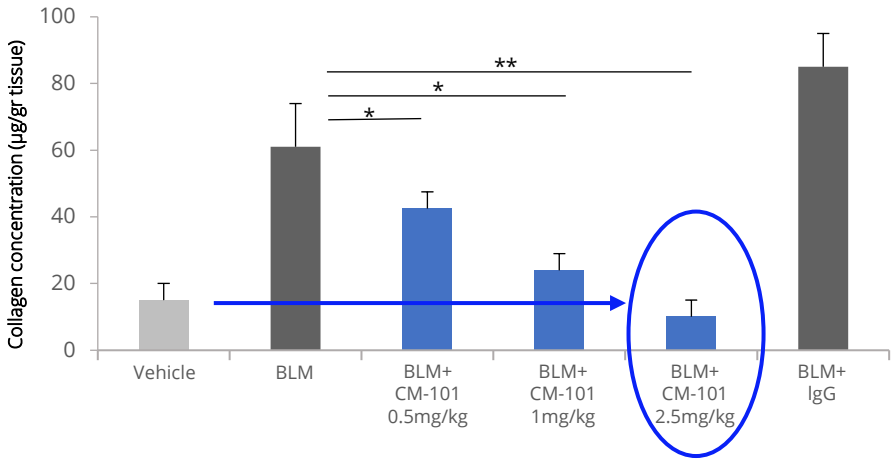


Mor A et al., Annals of Rheumatic Diseases, 2019; BLM – bleomycin; WT - wild type; KO – knock-out, BAL- bronchoalveolar lavage, PBS- phosphate-buffered saline
*p ≤0.05
** Thickness measures taken at multiple locations on samples. Arrows on graphic are for illustrative purposes only

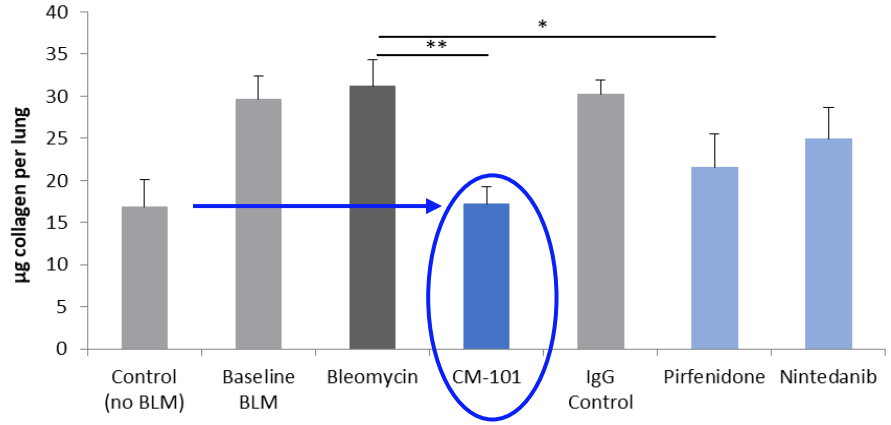
CM-101 Significantly Reduces Skin and Lung Fibrosis in Induced SSc

BLEOMYCIN-INDUCED ANIMAL MODEL

BLEOMYCIN (SC)-INDUCED DERMAL FIBROSIS



BLEOMYCIN (IT)-INDUCED LUNG FIBROSIS

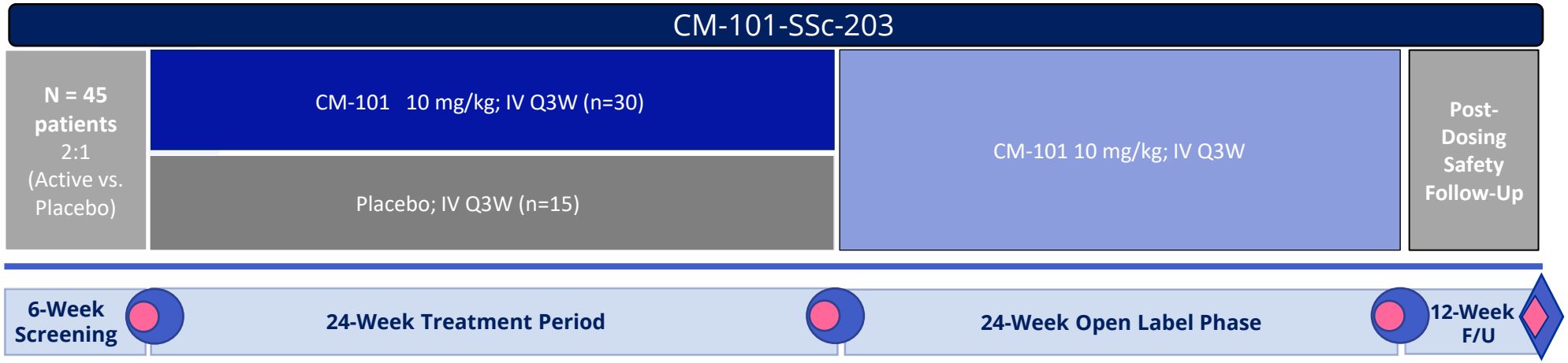


- Reduces skin & lung fibrosis levels in bleomycin-induced animal models
- Demonstrates dose dependent reduction of fibrosis
- Reduces lung collagen & inflammation more than the 2 approved drugs for lung fibrosis

Mor et al, Annals of Rheumatoid Diseases, 2019
**p ≤0.01; *p ≤0.05.
BLM-bleomycin; IT - intratracheal; SC–subcutaneous; IgG - immunoglobulin G

CM-101 Phase 2 Trial in Systemic Sclerosis

RANDOMIZED, DOUBLE-BLIND, PLACEBO-CONTROLLED; PLAN TO INITIATE 1H 2023



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

Key Enrollment Criteria

- SSC patients with active disease (dermatologic, vascular and/or pulmonary) and elevated circulating levels of CCL24
- Half with limited cutaneous SSc; half with diffuse cutaneous SSc

Outcome Measures

Primary: Safety & tolerability

Secondaries:

- Serum biomarkers:
  - Inflammatory cytokines
  - Vascular & growth factor-related
  - Pulmonary-related
  - Fibrogenesis & ECM-related
- Skin biopsy
- Pharmacokinetics & target engagement
- Anti-drug antibodies (ADAs)

Exploratory:

- Clinical & biological outcomes, incl. ACR-CRISS & revised CRISS

Top-line results expected 2H 2024

SSc-systemic sclerosis; Q3W-once every 3 weeks; IV-intravenous; ECM-extracellular matrix ; ACR-American College of Rheumatology; CRISS-Composite Response Index in Systemic Sclerosis

Experienced Management with Extensive Biopharma Experience

Dale Pfost, PhD
Chairman & Chief Executive Officer

Advent Life Sciences · OPKO · SmithKline Beckman Corporation · Orchid

Adi Mor, PhD
Co-founder & Chief Scientific Officer

Tel Aviv University · Tel-Aviv Sourasky Medical Center

Don Marvin
Chief Financial Officer, EVP & COO

Orchid · Abbott · Bayer · Boehringer Ingelheim · concentric

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

Boehringer Ingelheim · Novartis · Reata · FibroGen · Schering-Plough

Jack Lawler
Vice President, Global Clinical Development Operations

teva · Cephalon · Goldfinch Bio · Shire

Ilan Vaknin, PhD
Vice President, Research & Development

compugen · Weizmann Institute of Science

Chris Crater, MD
Vice President, Clinical Development

Atox Bio · parexel · PRAHEALTHSCIENCES · Bristol Myers Squibb

Mitch Jones, MD, PhD
Vice President, Corporate Development & Strategy

FINCH · BIORA Therapeutics · Micropharma

DEEP EXPERIENCE

- Capital efficient company building
- Translational science & global clinical development
- Delivering value to stakeholders

Financial Summary

Ticker symbol: **Nasdaq: CMMB**

No. shares outstanding:* **~229M** Ordinary Shares **= ~11.4M ADS's** (American Depository Shares)

ADS float: **~6-8M ADS's** (Estimated)

Market cap: **~$25 - $37M (**Q4 2022 approximate range)

Average ADS volume: **~420k per day** (Q4 2022 approximate range)

Cash status: **$40.0M** as of December 31, 2022

Cash runway: **at least through end of Q1 2024** (Estimated)

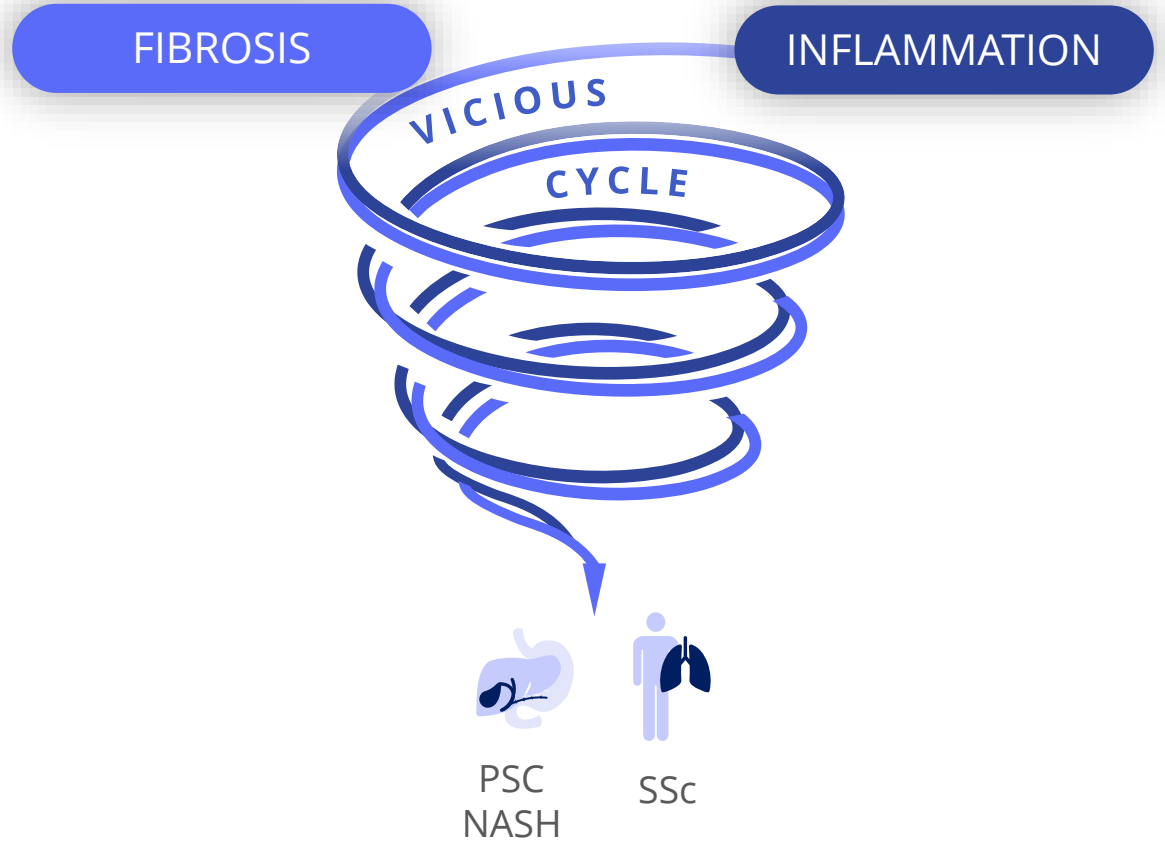
Debt: **None**

* as of September 30, 2022

Chemomab
THERAPEUTICS

Biotechnology company focused on fibro-inflammatory disease

- Unique target with disease modifying potential
- De-risked—extensive preclinical & early clinical data
- High unmet need—large commercial opportunities
- Two Phase 2 read-outs expected next year



CCL24: target central to biology of inflammation & fibrosis

PSC-primary sclerosing cholangitis; NASH-nonalcoholic steatohepatitis; SSc-systemic sclerosis

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

Thank you

Chemomab
THERAPEUTICS