NOVEMBER 2022

Chemomab

THERAPEUTICS

Pioneering Innovative Treatments for Fibro-inflammatory Diseases

NON-CONFIDENTIAL CORPORATE OVERVIEW
NASDAQ: CMMB

Forward Looking Statements

This presentation contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act. These forward-looking statements include, among other things, statements regarding the clinical development pathway for CM-101; the future operations of Chemomab and its ability to successfully initiate and complete clinical trials and achieve regulatory milestones; the nature, strategy and focus of Chemomab; the development and commercial potential and potential benefits of any product candidates of Chemomab; and that the product candidates have the potential to address high unmet needs of patients with serious fibrosis-related diseases and conditions. Any statements contained in this communication that are not statements of historical fact may be deemed to be forward-looking statements. These forward-looking statements are based upon Chemomab's current expectations. Forward-looking statements involve risks and uncertainties.

Because such statements deal with future events and are based on Chemomab's current expectations, they are subject to various risks and uncertainties and actual results, performance or achievements of Chemomab could differ materially from those described in or implied by the statements in this presentation, including: the uncertain and time-consuming regulatory approval process; risks related to Chemomab's ability to correctly manage its operating expenses and its expenses; Chemomab's plans to develop and commercialize its product candidates, including CM-101; the timing of initiation of Chemomab's planned clinical trials; the timing of the availability of data from Chemomab's clinical trials; the timing of any planned investigational new drug application or new drug application; Chemomab's plans to research, develop and commercialize its current and future product candidates; the clinical utility, potential benefits and market acceptance of Chemomab's product candidates; Chemomab's commercialization, marketing and manufacturing capabilities and strategy; Chemomab's ability to protect its intellectual property position; and the requirement for additional capital to continue to advance these product candidates, which may not be available on favorable terms or at all. Additional risks and uncertainties relating to Chemomab's and its business can be found under the caption "Risk Factors" and elsewhere in Chemomab's filings and reports with the SEC. Chemomab expressly disclaims any obligation or undertaking to release publicly any updates or revisions to any forward-looking statements contained herein to reflect any change in Chemomab's expectations with regard thereto or any change in events, conditions or circumstances on which any such statements are based.

This presentation ("Presentation") is for informational purposes only and does not constitute an offer to sell, solicitation of an offer to buy, or a recommendation to purchase any equity, debt or other financial instruments of Chemomab. The data contained herein is derived from various internal and external sources. No representation is made as to the reasonableness of the assumptions made within or the accuracy or completeness of any other information contained herein. All levels, prices and spreads are historical and do not represent current market levels, prices or spreads, some or all of which may have changed since the issuance of this document. Any data on past performance, modeling contained herein is not an indication as to future performance. Chemomab assume no obligation to update the information in this Presentation. Chemomab does not accept any liability whatsoever for any losses arising from the use of this Presentation or reliance on the information contained herein. Nothing herein shall be deemed to constitute investment, legal, tax, financial, accounting or other advice. This Presentation is being provided for use only by the intended recipient.

Chemomab
THERAPEUTICS

Clinical stage biotech company discovering and developing **innovative therapeutics** for fibro-inflammatory diseases with high unmet need

Our Focus

FIBROSIS

INFLAMMATION

Fibro-Inflammatory Diseases
at the confluence of
Fibrosis & **Inflammation**

Chemomab Highlights

A PIPELINE IN A PRODUCT

The Drug

CM-101
First-in-class monoclonal antibody with disease modifying potential

The Target

CCL24
Novel, broadly applicable, dual-acting target

The Science

De-risked--extensive & rigorous preclinical package & early clinical validation of:

- CCL24 as driver of fibrosis-inflammation
- CM-101's ability to attenuate fibrotic-inflammatory pathways to impact disease

Disease Focus

Fibrotic-inflammatory with high unmet need & strong commercial potential

- Primary Sclerosing Cholangitis
- Systemic Sclerosis
- Other fibro-inflammatory diseases

Est. Commercial Potential $2.5B+

SOLID FOUNDATION

High Value Clinical Programs

- Two Phase 2 trials underway; 3rd starting 4Q 2022/1Q 2023
- Liver biomarker Phase 2 data readout expected 4Q 2022

Robust IP Portfolio

- Global patent exclusivity thru 2038 with possible extensions
- COM in major markets + use patents; others pending

Experienced Management

- Value-creation focused leadership with record of success

Top Tier Investors

OrbiMed Healthcare Fund Management | Cormorant Asset Management | THIEL

Solid Balance Sheet

- $46.5M cash & equivalents (9/30/22)
- Estimated runway through end-2023

Experienced Management with Extensive Biopharma Experience

Dale Pfost, PhD
Chairman & Chief Executive Officer

OPKO · Advent Life Sciences · 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

Strong Board and Scientific Advisors

BOARD OF DIRECTORS

Dale Pfost, PhD
Chairman
Chief Executive Officer
Chemomab Therapeutics

Adi Mor, PhD
Director
Co-founder & Chief Scientific Officer
Chemomab Therapeutics

Neil Cohen, MA
Director
Anchiano therapeutics

Alan Moses, MD
Director
novo nordisk

Nissim Darvish, MD, PhD
Director
eOHR Ventures

Claude Nicaise, MD
Director
Alexion; Bristol Myers Squibb

Jill Quigley, JD
Director
PassageBio; NPS Pharma

SCIENTIFIC ADVISORS (Selected)

Marco Matucci-Cerinic, MD, PhD
Director of the Division of Rheumatology
University of Florence, Italy

Francesco Del Galdo, MD, PhD
Head of the Scleroderma Program at NIHR
University of Leeds

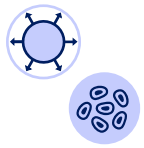
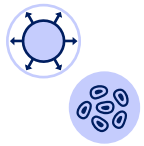
Scott L. Friedman, MD
Dean for Therapeutic Discovery and Chief, Division of Liver Diseases
Mount Sinai

Dinesh Khanna MD, MBBS, MSc
Director of the Scleroderma Program
University of Michigan

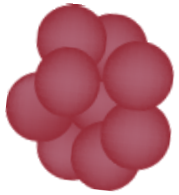
Massimo Pinzani, MD, PhD, FRCP
Sheila Sherlock Chair of Hepatology, Director UCL Institute for Liver and Digestive Health
Royal Free London NHS Foundation Trust

CM-101: Pipeline in a Product

ADDRESSING FIBRO-INFLAMMATORY DISEASES WITH HIGH UNMET NEED

PROGRAM		INDICATION	TARGET	DISCOVERY	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	UPCOMING MILESTONES
CM-101		Subcutaneous Formulation & Liver Fibrosis Biomarkers	CCL24	SC Route of Administration & biomarker data in NASH patients				Treatment Concluded	Top-line Data 4Q 2022
		Primary Sclerosing Cholangitis	CCL24	Orphan designation granted by FDA and EMA				Enrolling Patients	Top-line Data 2H 2024
		Systemic Sclerosis	CCL24	Orphan designation granted by FDA and EMA			Phase 2 Initiates		Trial Initiates 4Q 2022/1Q 2023 Top-line Data 2H 2024
		Fibro-inflammatory Diseases	CCL24						
Next-Gen (Novel Targets)		TBD	Screening						

Differentiated by Our Dual-acting, Novel Target: CCL24



CCL24
The Target

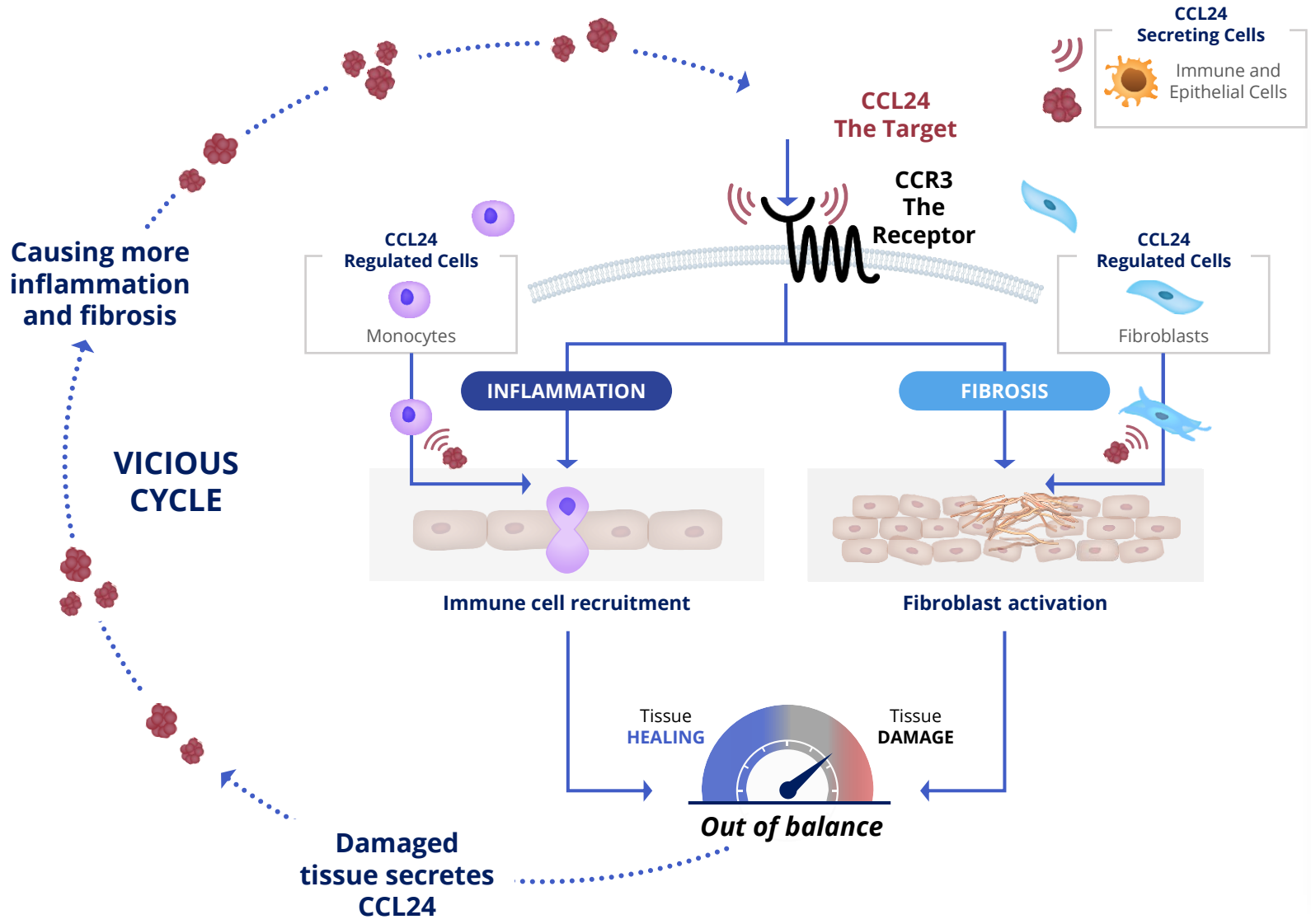
A cytokine-family, soluble protein central to the biology of inflammation and fibrosis

Our Advantages

Leadership in Addressing High Potential, Unique Target

De-risked via Extensive Preclinical Validation

CCL24's Role in The Vicious Cycle of Inflammation and Fibrosis



THE POWER OF CCL24

Dual Role in Promoting Fibrosis and Inflammation

Directly activates fibroblasts

Enhances local immune cell recruitment

Differentiated Activity

Data shows **unique role** vs. other CCLs

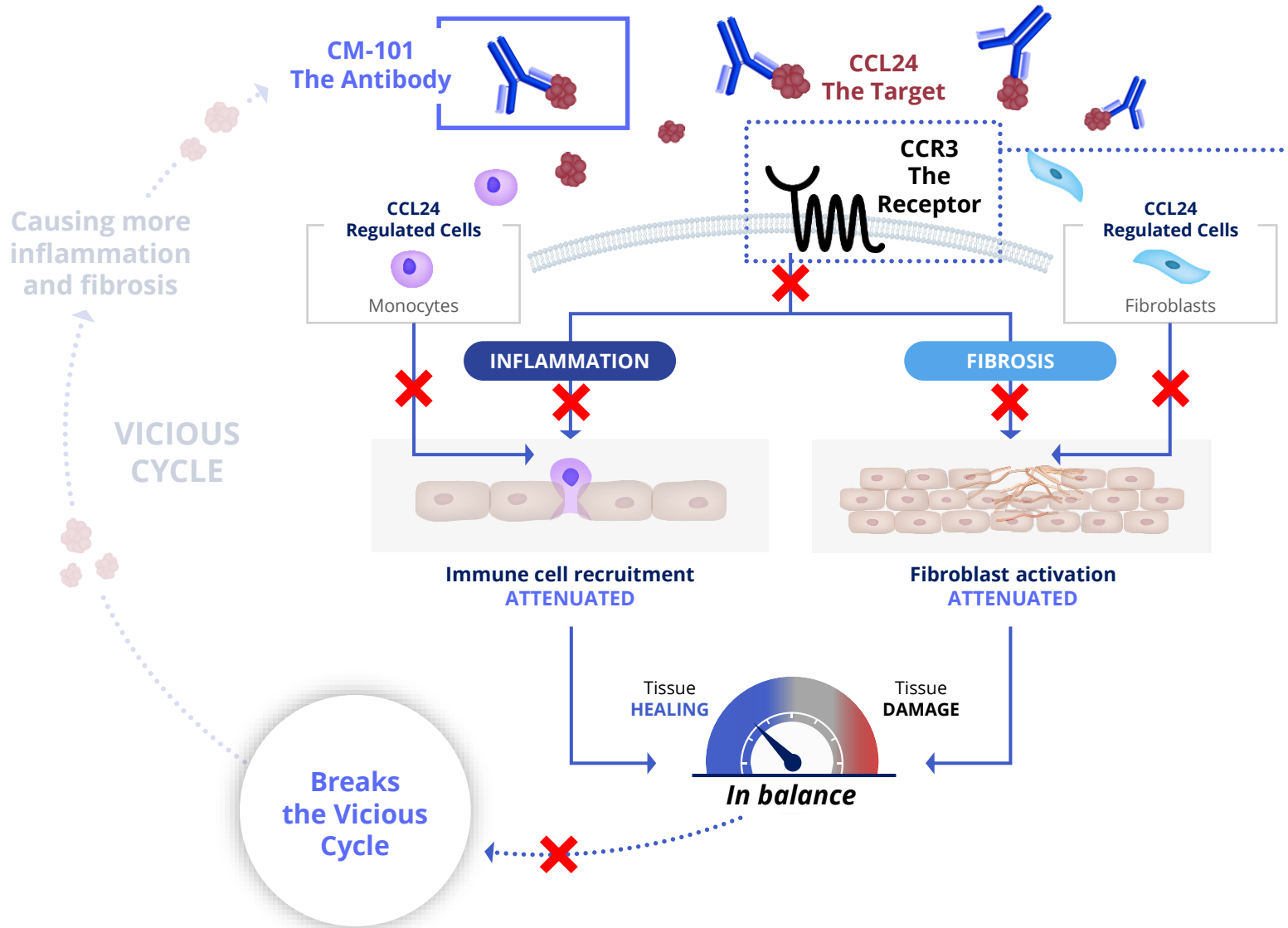
Correlates with fibrotic biomarkers and disease outcome

Low in Healthy Tissue; Elevated in Fibrotic Tissue

Liver, skin, lung, kidney, others

Wide therapeutic margin

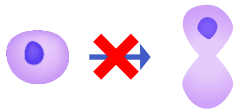
CM-101 Neutralizes CCL24, Breaking Vicious Cycle



Neutralizing CCL24 Has Advantages Over Blocking the Receptor

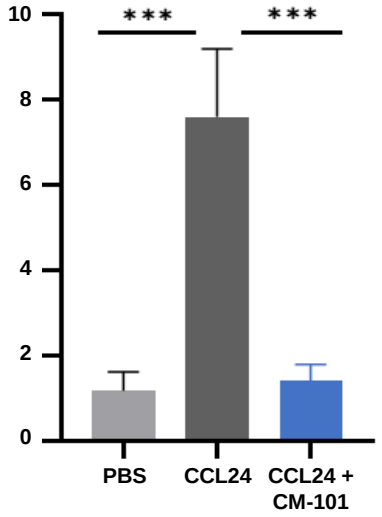
- Avoids context dependence of CCR3 receptor
- Efficacy of direct approach
- Favorable safety profile; retains normal repair functions

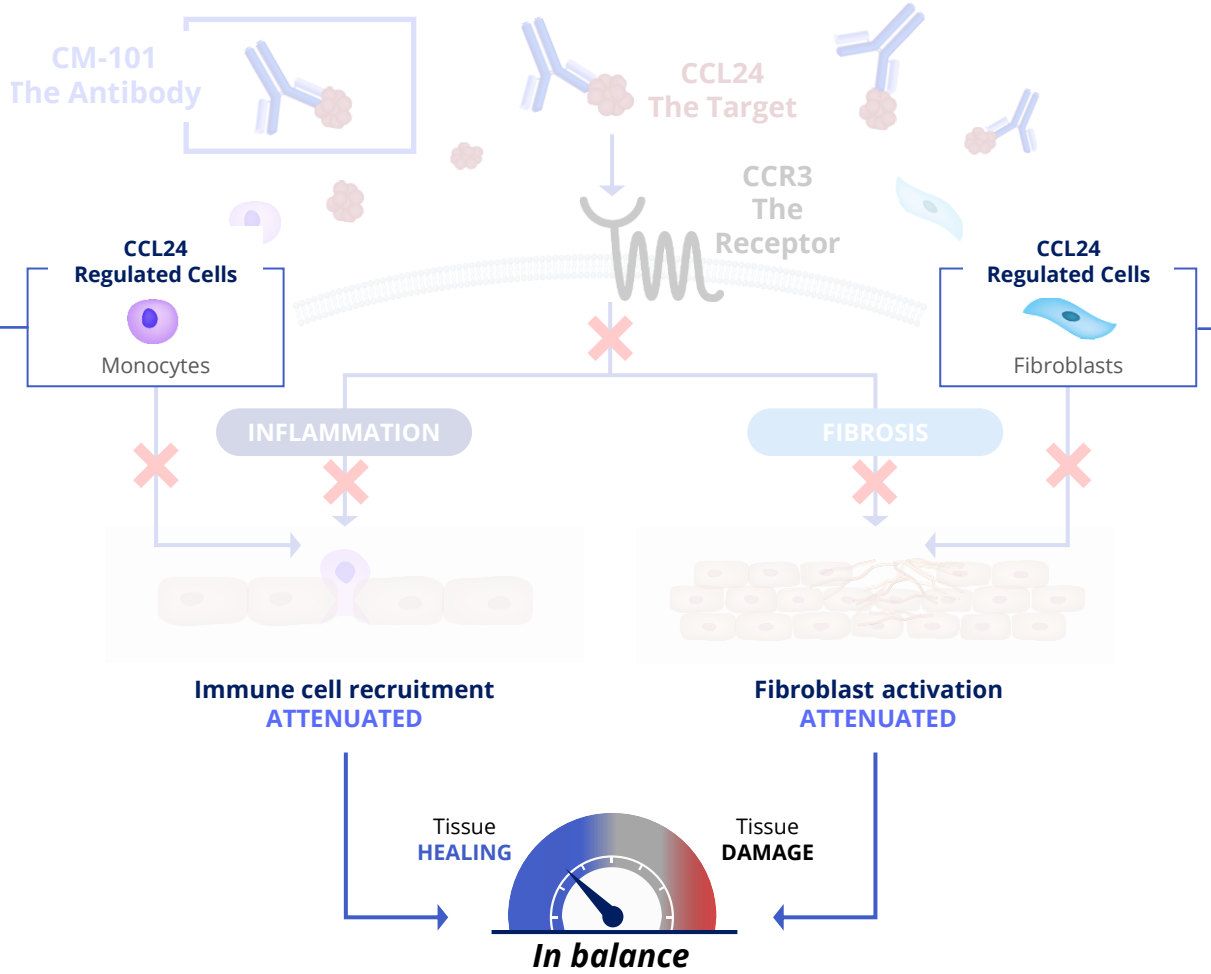
The Proof: First-in-Class CM-101 Neutralizes CCL24



CM-101 Reduces *In vivo* Monocyte Recruitment

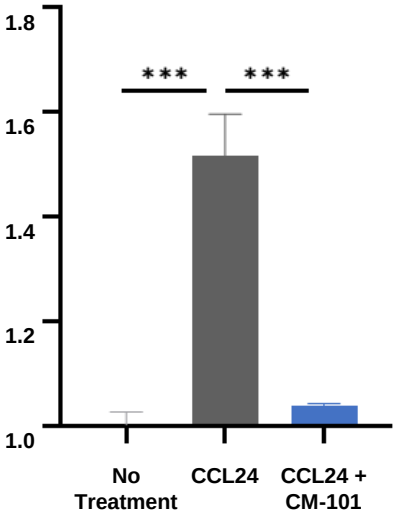
Number of Migrated Monocytes (x1000)





CM-101 Inhibits Primary Fibroblast Activation

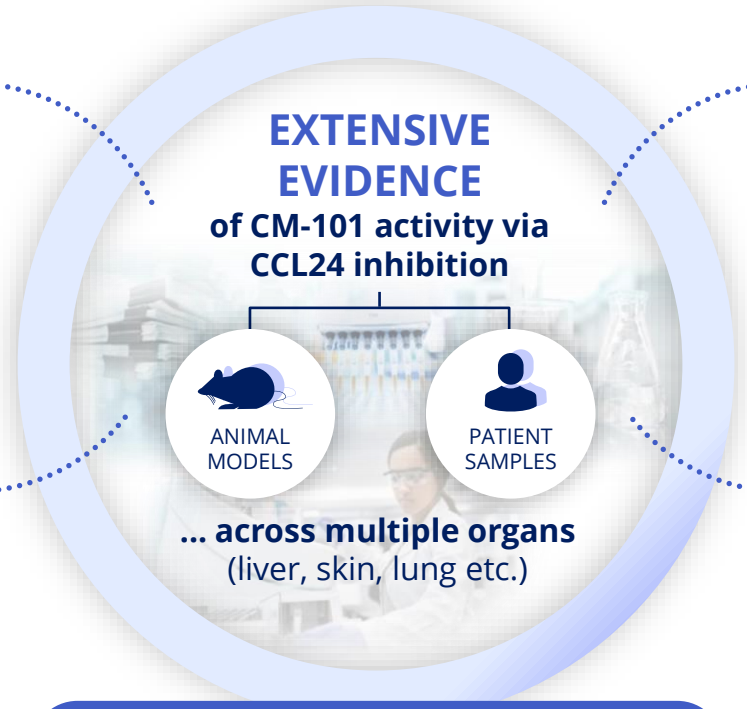
Proliferation: Relative Change from Baseline



Broad, Diverse Preclinical Evidence De-risks Clinical Development

CCL24 Target Validation
Ex-vivo Patient Samples

CCL24 Target Validation
In-vivo (Knockout Animal Models)



Mechanism of Action

Toxicology

Proof of Concept / Animal Models

Broad, Diverse Preclinical Evidence De-risks Clinical Development

CCL24 Target Validation Ex-vivo Patient Samples

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

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

Systemic sclerosis
- Fibrotic biomarker correlation
- Disease deterioration correlation
- Overexpression 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)

EXTENSIVE EVIDENCE

of CM-101 activity via CCL24 inhibition

ANIMAL MODELS

PATIENT SAMPLES

... across multiple organs
(liver, skin, lung etc.)

Mechanism of Action

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

- **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

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)

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

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

HEALTHY VOLUNTEERS

PHASE 1A STUDY – SINGLE ASCENDING DOSE STUDY

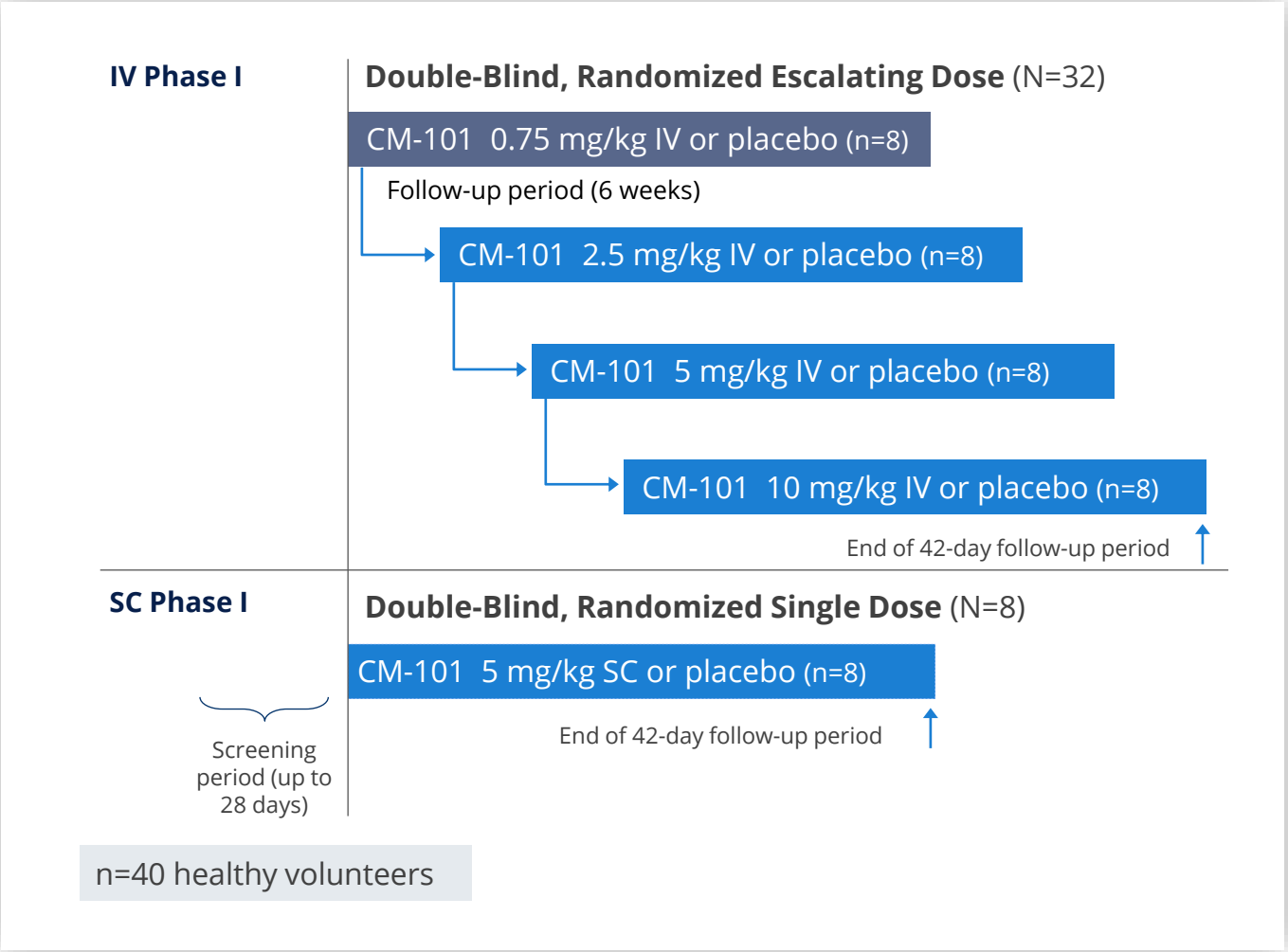
CM-101 was **safe and well tolerated** at all tested doses up to 10 mg/kg and for both IV and SC formulations

All drug related AEs were classified as **mild with no SAEs reported**

Average half-life of 19-21 days supports long interval **administration once every 2-4 weeks**

Achieved dose-dependent target engagement measured by serum CCL24 levels

SC and IV formulations showed **comparable target engagement** and **PK profiles**



CM-101 Safe & Well Tolerated in Subjects with NAFLD*

PATIENTS

PHASE 1B MULTIPLE ADMINISTRATION STUDY

Study population: individuals with **NAFLD** and normal liver function

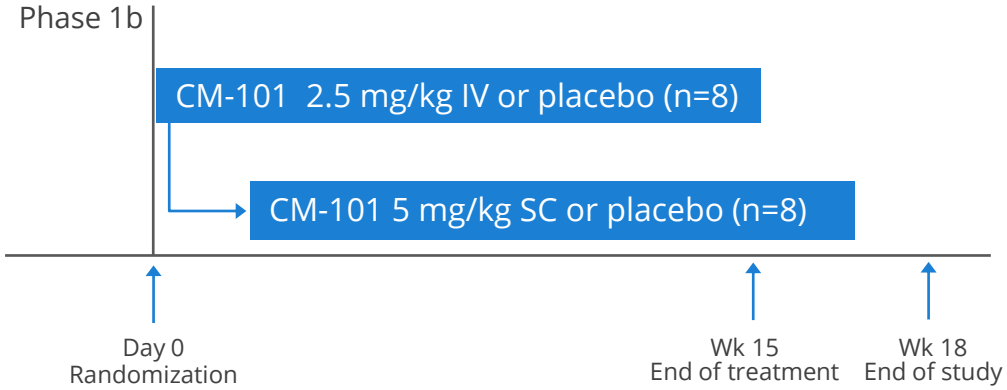
Multiple CM-101 administrations were **safe and well tolerated** using both **IV** and **SC formulations**

Most frequently reported AEs were **mild with no drug-related SAEs reported**

Favorable half-life supports **long dosing interval** (Q2W - Q4W)

Study Design

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



- Tested doses - 2.5 mg/kg IV infusion and 5 mg/kg SC injection
- 5 repeated administrations per subject; Q3W
- Primary endpoints - safety and tolerability

* Nonalcoholic fatty liver disease

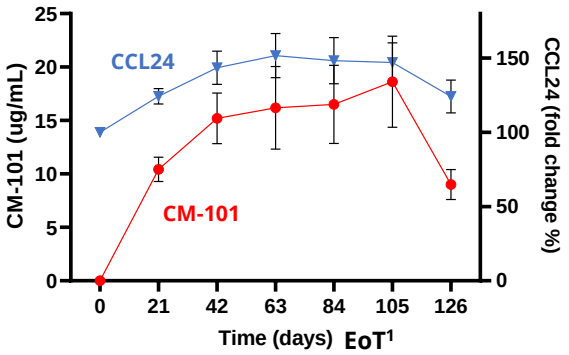
Anti-Fibrotic Mechanisms of CM-101 Confirmed in NAFLD Subjects

PATIENTS

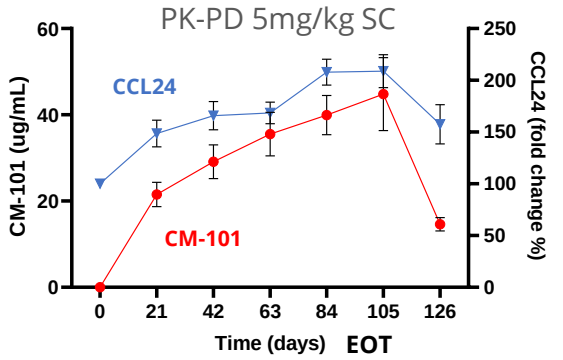
CM-101

PK-TARGET ENGAGEMENT

PK-PD 2.5mg/kg IV



PK-PD 5mg/kg SC



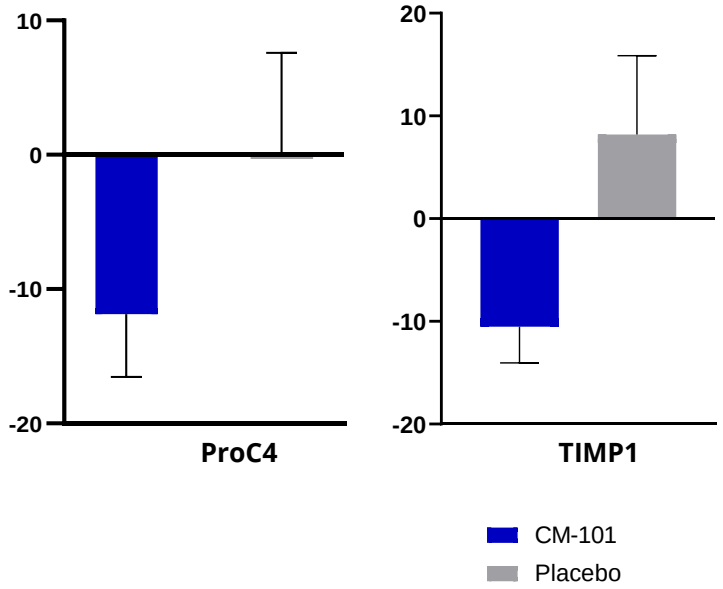
Clear correlation between antibody (red) and target (blue) levels

Dose dependent PK-target engagement

FIBROSIS

Biomarkers*

Relative Change from Baseline (%)



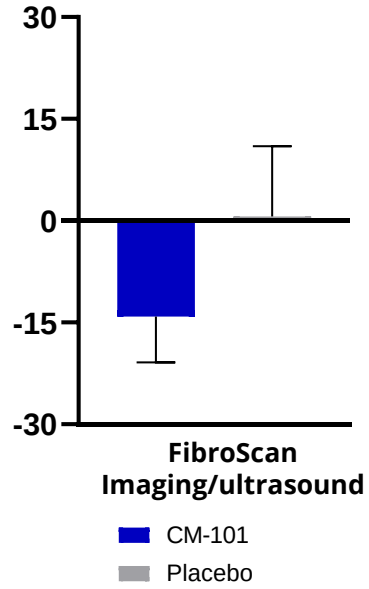
Concordant results across 6 additional fibrotic markers

Improved liver biomarkers

LIVER STIFFNESS

Physiological Measure

Relative change from baseline (%)



N=10 active, 3 placebo
All doses; all subjects KPA > 4

Decreased liver stiffness

*ProC4-Procollagen 4; TIMP1-Tissue inhibitor of metallopeptidase 1. EoT-End of Treatment

CM-101 Reduced Fibro-Inflammatory Biomarkers

INVESTIGATOR OPEN LABEL, SINGLE ARM STUDY EVALUATED SAFETY & ACTIVITY IN COVID-19 PATIENTS WITH SEVERE LUNG DAMAGE*

PATIENTS

KEY ENROLLMENT CRITERIA

- Enrolled 16 hospitalized Covid-19 patients with severe lung injury
- Received single I.V. administration of 10 mg/kg of CM-101
- Standard of Care treatment allowed -- corticosteroids, anti-coagulants & antivirals

PRIMARY OBJECTIVE:

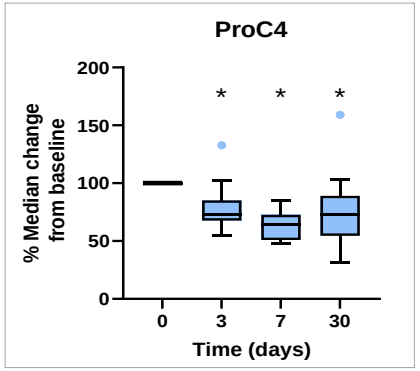
- Assess safety & activity of CM-101 in patients with Covid-19-related severe pneumonia

SECONDARY OBJECTIVE:

- Determine association between CM-101 treatment & chemokine/cytokine profile in patients

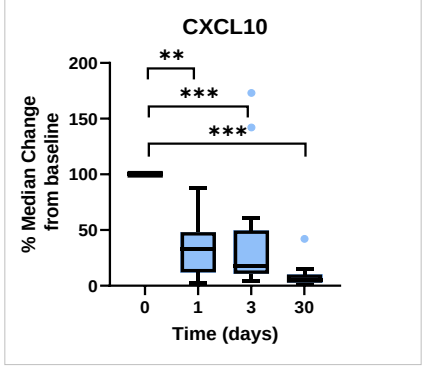
CM-101 Demonstrated Strong & Rapid Reductions in Biomarkers Associated With:

FIBROGENESIS



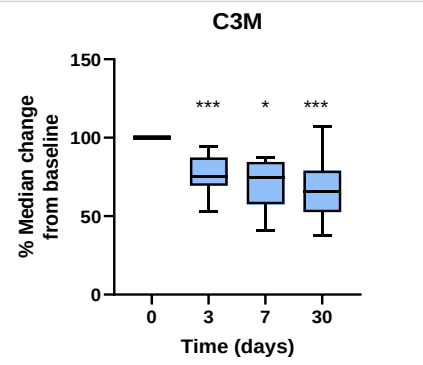
Considered early marker of fibrosis

LUNG INFLAMMATION



Elevated in patients with systemic sclerosis

NEUTROPHIL ACTIVITY



Elevated in progressive liver & pulmonary fibrosis

RESULTS

- CM-101 was safe & well-tolerated
- CM-101 demonstrated **strong & rapid reductions in biomarkers** associated with:
  - ✓ Lung inflammation
  - ✓ Fibrogenesis
  - ✓ Neutrophil activity
- Results are consistent with previous CM-101 clinical and preclinical data
- Further confirm potential of CM-101 **to attenuate inflammation & fibrosis in varied organs** including lung, liver & skin

CM-101 reduced lung inflammation & fibrogenesis biomarkers known to be associated with 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

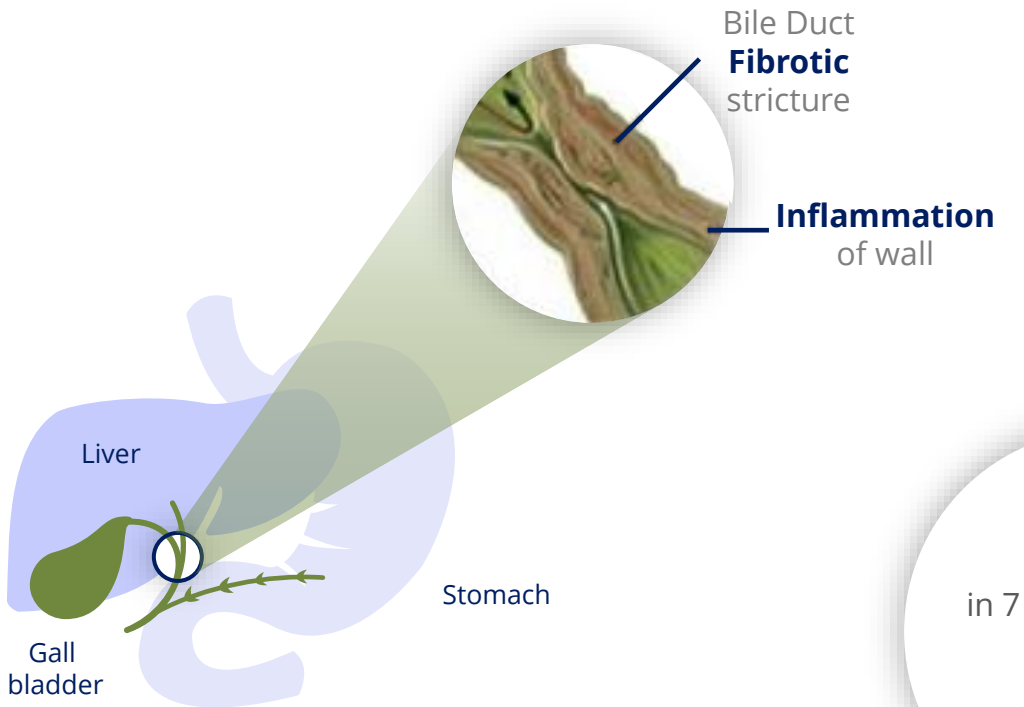
CM-101

for the Treatment of Primary Sclerosing Cholangitis (PSC)

Primary Sclerosing Cholangitis (PSC): High Morbidity and Mortality

Rare, Chronic Liver Disease

CHARACTERIZED BY INFLAMMATION AND FIBROSIS



DEVASTATING DISEASE WITH NO APPROVED THERAPIES

Affects:
Mostly Men (2:1 Male/Female); Associated with IBD

Diagnosis:
Age ~30-40

Fibrosis shuts down bile duct
and acid backs up in liver

MEDIAN SURVIVAL
10 years with no intervention

End stage liver disease, cirrhosis and cancer

Liver transplant only therapy with some curative potential

~77K
patients
in 7 major markets

>$1B
est. commercial opportunity

Source: Johns Hopkins Medicine

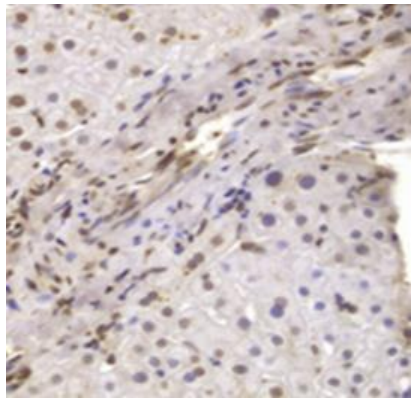
CCL24 Plays a Key Role in PSC

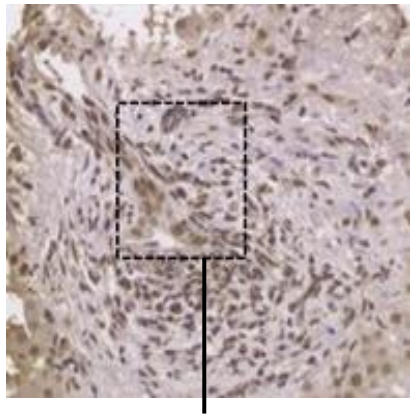
PATIENT SAMPLES

CCL24 LEVEL IN HEALTHY VS PSC PATIENTS' LIVER TISSUE[1]

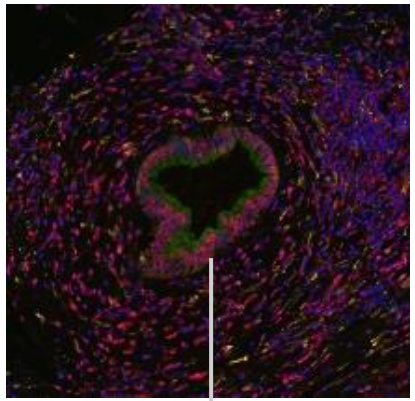
Healthy Liver | **PSC Livers**

CCL24 Staining





CCL24 (brown) overexpressed in damaged bile duct area

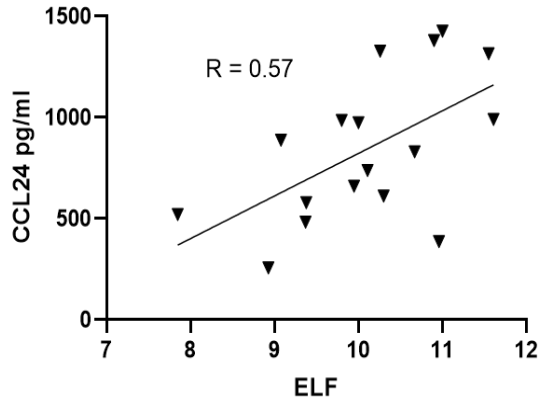


CCL24 (red) overexpressed in bile epithelial cells (green) and immune cells (yellow)

CCL24 expression is significantly and 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

Data from patient samples supports clinical relevance of CCL24 in PSC

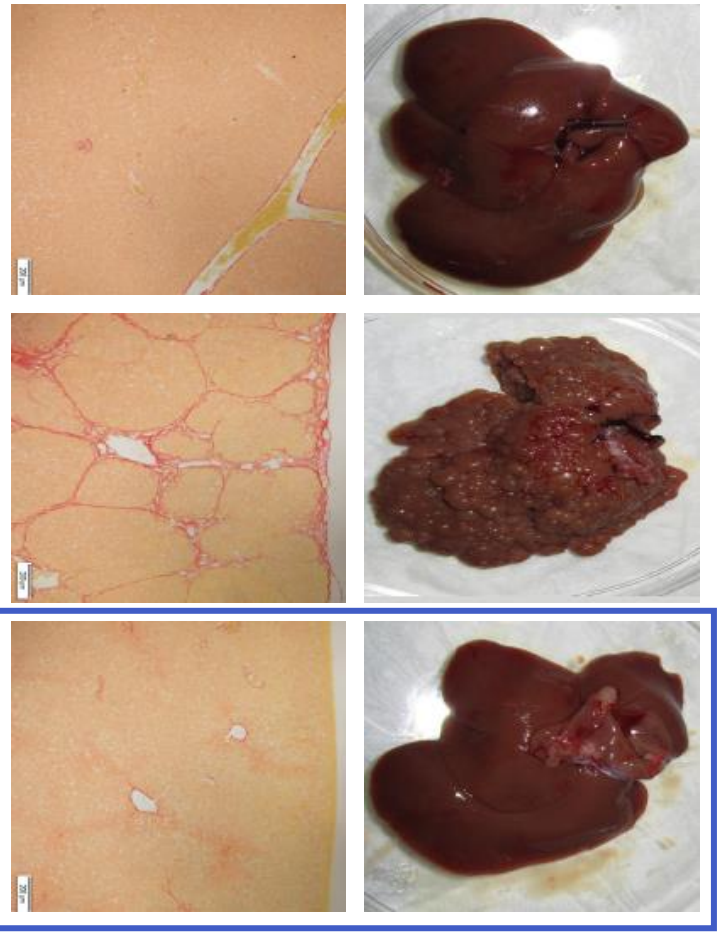
1. Collaboration with RFH, UK
ELF – Enhanced Liver Fibrosis; ALP – Alkaline Phosphatase

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

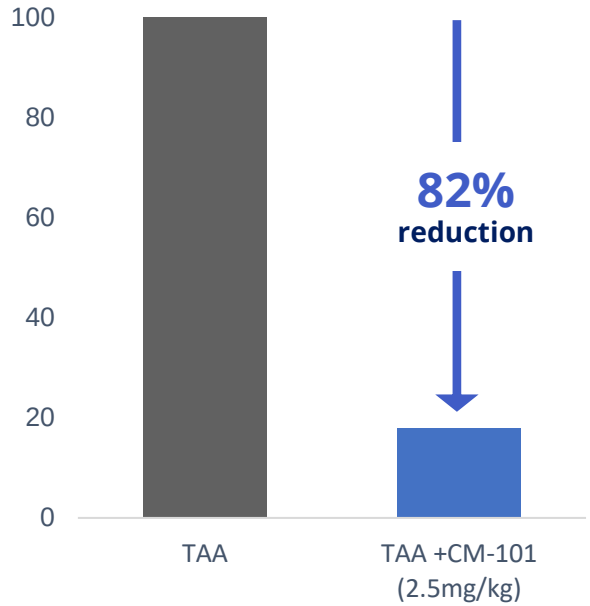
ANIMAL MODELS (TAA RAT)

RAT MODEL LIVERS

SIRIUS RED (COLLAGEN)



% FIBROSIS
(SIRIUS RED STAINING)



Pro-fibrotic Genes
Col1A1
Col3A1
TIMP1
ACTA2
TGF-β

Liver Enzymes
ALT
AST
ALP

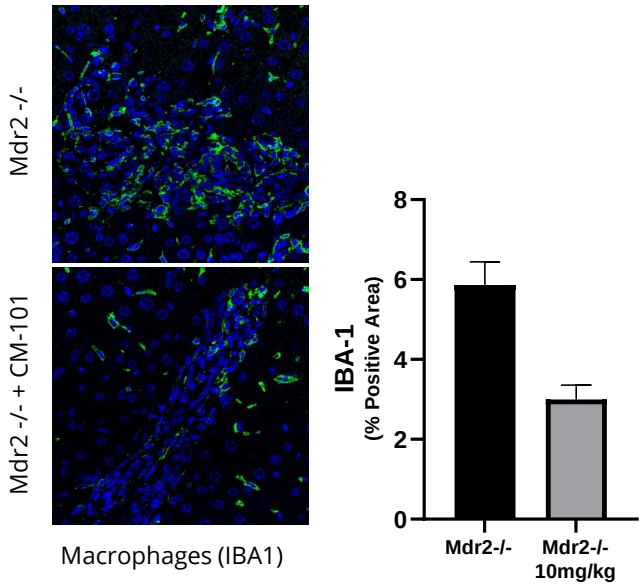
Dramatically reduced liver collagen in TAA liver fibrosis rat model with established liver fibrosis

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

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

ANIMAL MODELS
MDR 2 MOUSE
KNOCKOUT

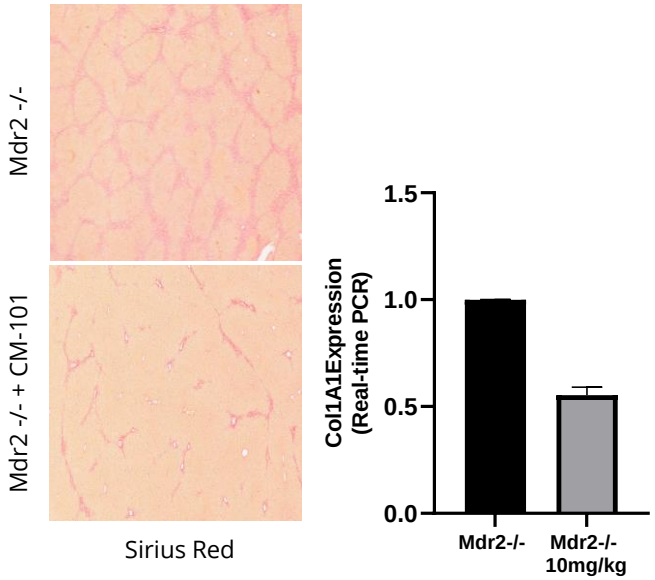
REDUCED MACROPHAGE NUMBER



Macrophages (IBA1)

Reduction in
INFLAMMATION

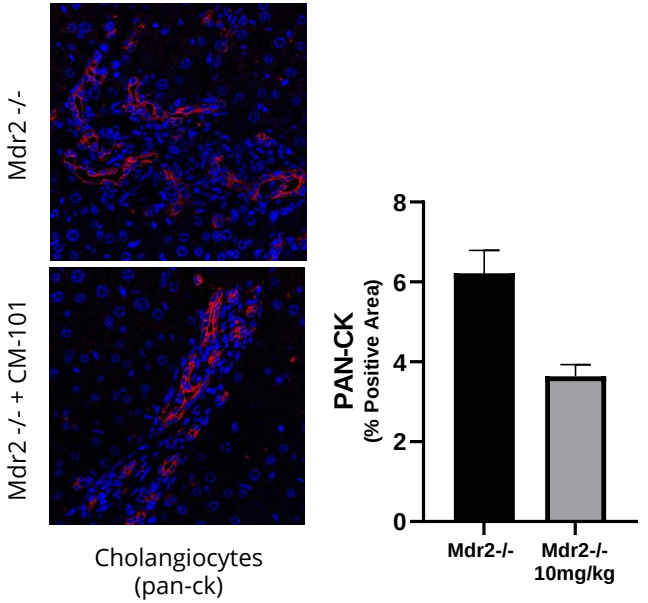
ATTENUATED LIVER COLLAGEN DEPOSITION



Sirius Red

Reduction in
FIBROSIS

REDUCED ABERRANT BILE EPITHELIAL PROLIFERATION



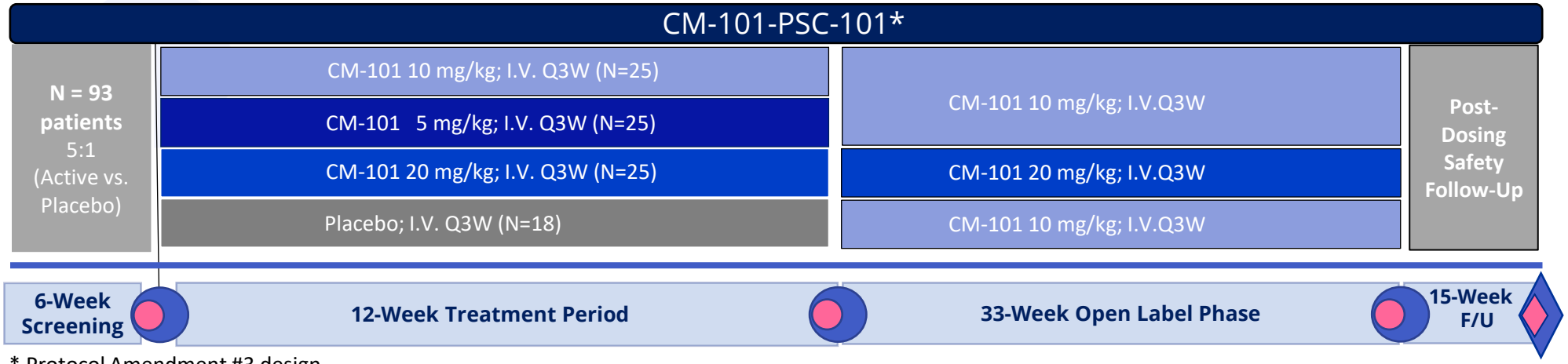
Cholangiocytes
(pan-ck)

Main driver of
DISEASE PATHOLOGY

CM-101 interferes with core pathways that drive PSC

CM-101 Phase 2 Trial in Primary Sclerosing Cholangitis

RANDOMIZED, DOUBLE-BLIND, PLACEBO-CONTROLLED



* Protocol Amendment #3 design

- **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
- Anti-Drug Antibody
- Pharmacodynamic parameters

Top-line results expected 2H 2024

PSC-primary sclerosing cholangitis; **Q3W**-once every 3 weeks; **I.V.**-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

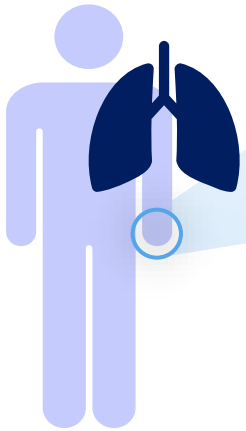
for the Treatment of Systemic Sclerosis (SSc)

Systemic Sclerosis (SSc): Most Lethal Systemic Rheumatic Disease

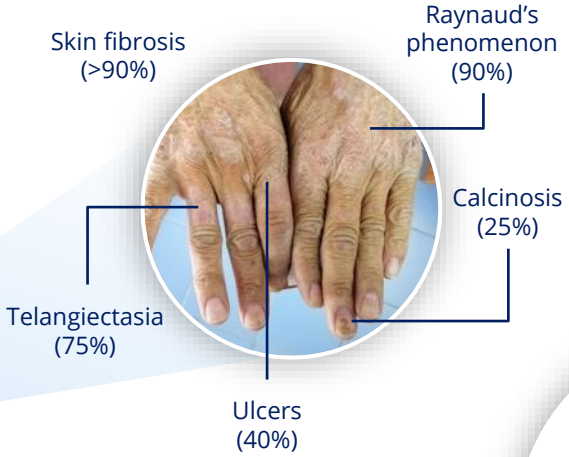
Rare Autoimmune Rheumatic Disease

CHARACTERIZED BY INFLAMMATION, FIBROSIS AND VASCULOPATHY

LUNG INVOLVEMENT:
Leading cause of death



SKIN:
Most evident symptom



~170K
patients
in 7 major markets

>$1.5B
est. commercial opportunity

DEVASTATING DISEASE WITH NO APPROVED DISEASE MODIFYING THERAPY

Affects:
Predominantly Women (3:1 Female/Male)

Diagnosis:
Age ~30-50

MEDIAN SURVIVAL
10 years

Pulmonary Involvement leading cause of death

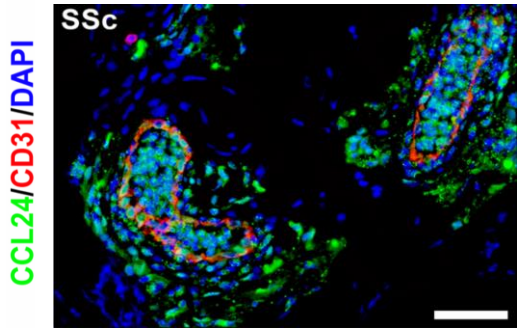
Nintedanib, Tocilizumab only treat symptoms, **NOT** disease modifying

Source: Johns Hopkins Medicine

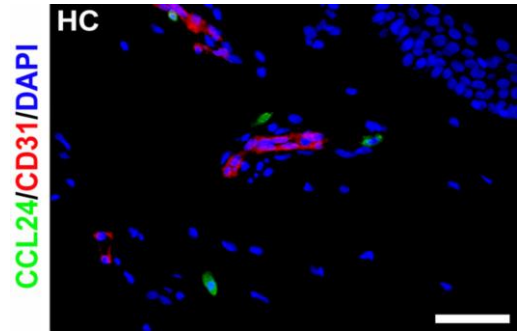
CCL24: A Critical Node Potentiating Systemic Sclerosis

PATIENT SAMPLES

SSc Skin

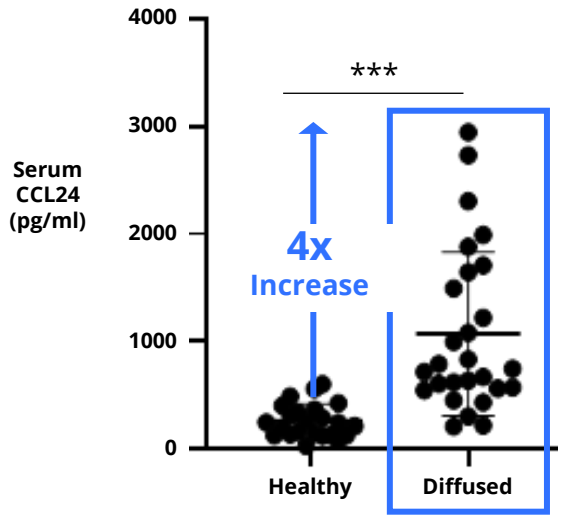


Healthy Skin



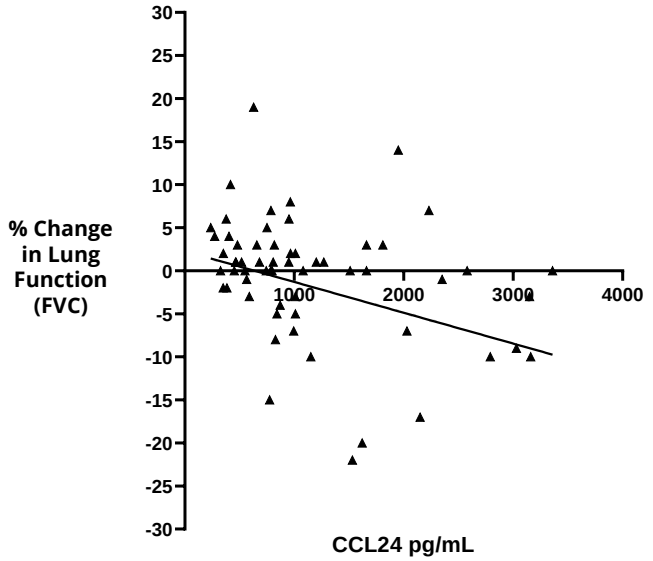
Collaboration with University of Florence

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

Data from patient samples supports potential therapeutic utility of blocking CCL24 in systemic sclerosis

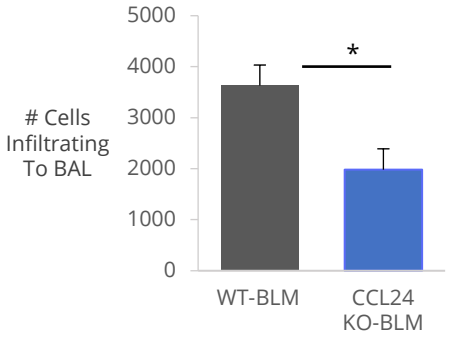
dSSc – diffuse systemic sclerosis; Mor A et al., Annals of Rheumatic Diseases, 2019
***p ≤0.001

Target Validation: CCL24 Gene Knockout Ameliorates Induced SSc

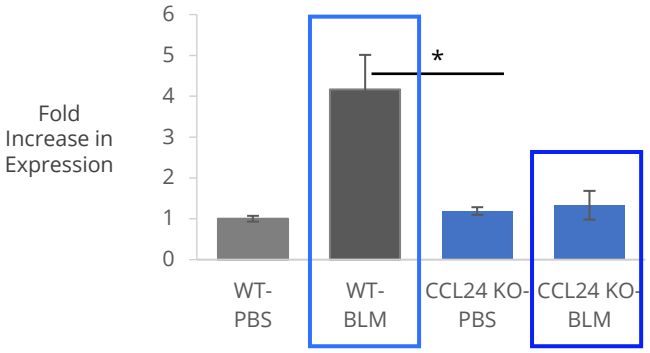
ANIMAL MODELS
SSc BLEOMYCIN
MOUSE MODEL

BLEOMYCIN-INDUCED SSc-LIKE CONDITION IN MICE

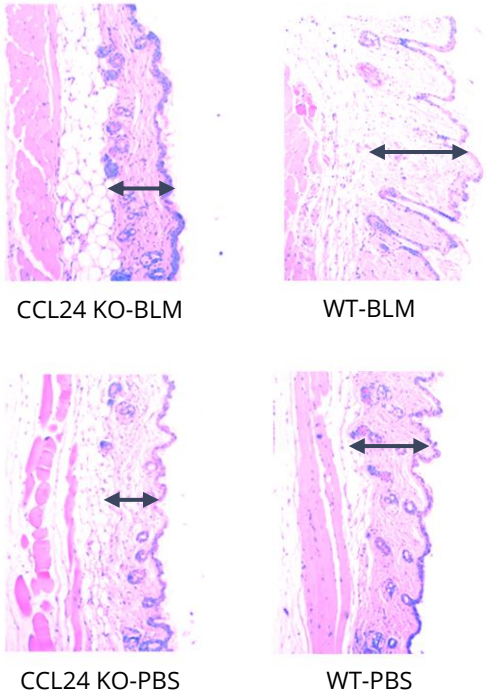
Immune Cell Lung Infiltration

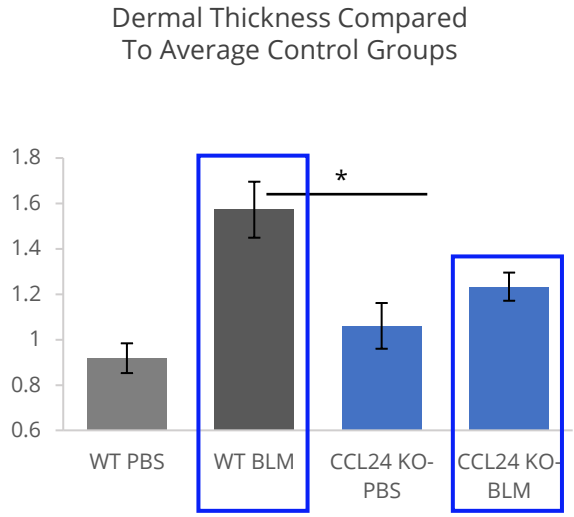


α SMA Expression in Skin Lesions



Dermal Thickness**





CCL24 Knockout attenuates induced SSc-like inflammation and fibrosis in lung and skin

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

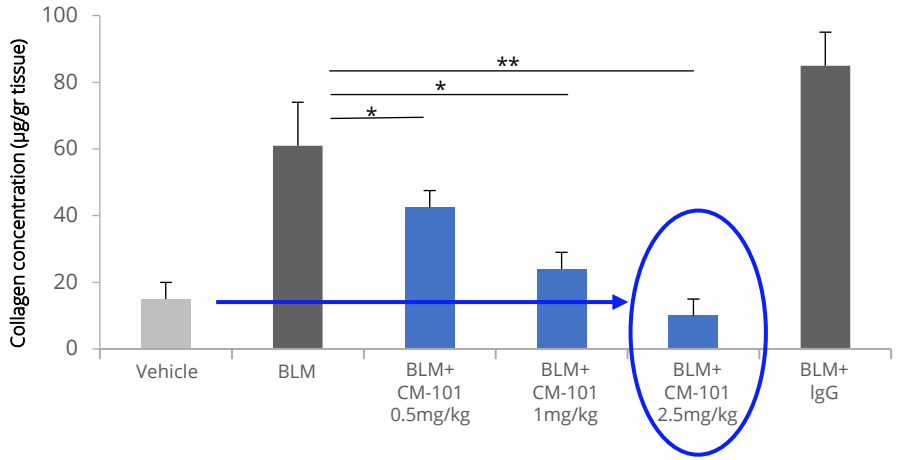
CM-101 Significantly Reduces Skin and Lung Fibrosis in Induced SSc

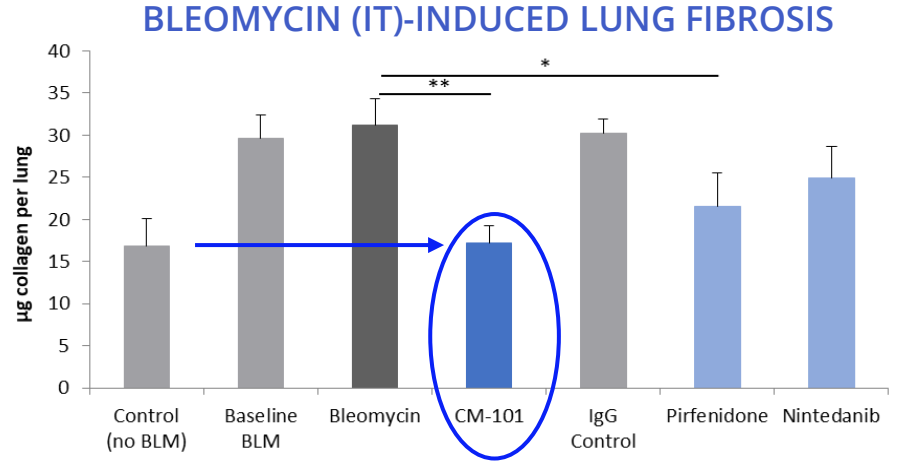
BLEOMYCIN-INDUCED ANIMAL MODEL

Attenuates skin and lung fibrosis levels
in bleomycin-induced animal models

Demonstrates **dose dependent attenuation of fibrosis**

Reduces lung collagen and inflammation compared to 2 approved drugs
for lung fibrosis

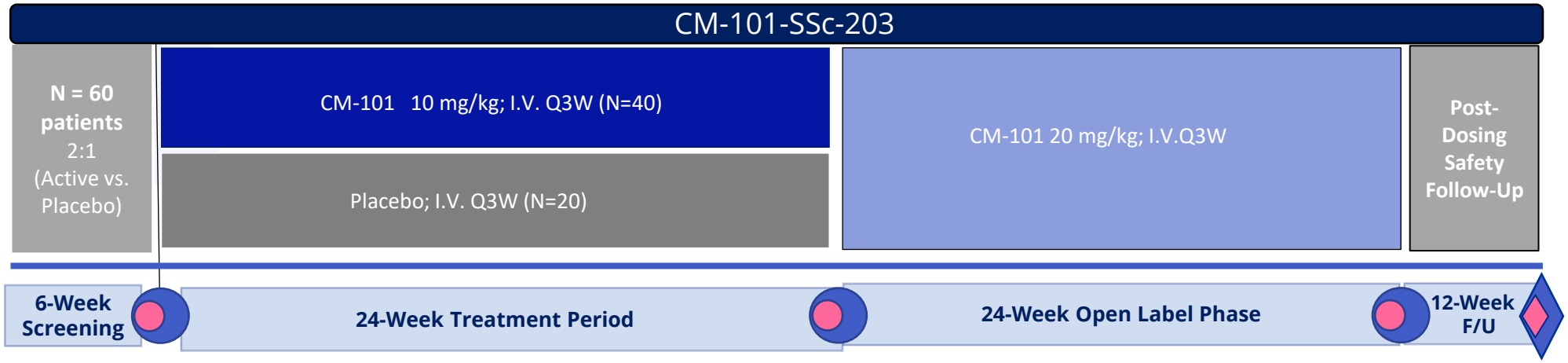




Mor et al, Annals of Rheumatoid Diseases, 2019
**p ≤0.01; *p ≤0.05.
BLM - bleomycin; IT - Intratracheal; SC – Subcutaneous IgG - Immunoglobulin G

CM-101 Phase 2 Trial in Systemic Sclerosis

RANDOMIZED, DOUBLE-BLIND, PLACEBO-CONTROLLED



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

Key Enrollment Criteria

- SSC patients with active disease (dermatologic or pulmonary) & high circulating levels of CCL24
- Half with limited 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

Exploratory:

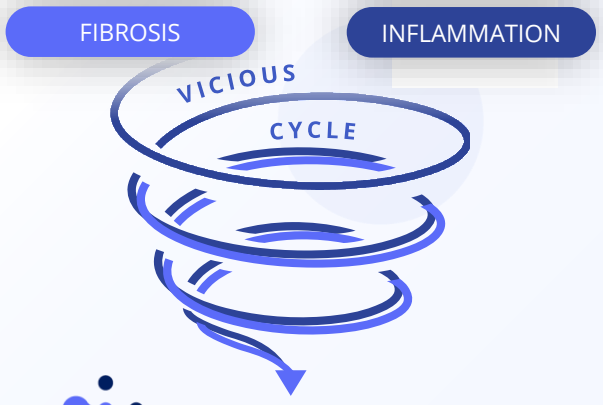
- Clinical & biological outcomes

Top-line results expected 4Q 2024

SSc-Systemic Sclerosis; **Q3W**-once every 3 weeks; **I.V.**-intravenous; **ECM**-extracellular matrix

CM-101 Clinical Development Plan Overview

	STATUS	2021	2022	2023	2024	
Liver Fibrosis/ Subcutaneous Formulation	**Treatment Concluded:** Measure SC formulation safety, PK & exploratory biomarkers in NASH	**Phase 2a: 16-week treatment** Randomized, placebo-controlled Israel study sites				**Top-line data expected 4Q 2022**
Primary Sclerosing Cholangitis	**Expanded Enrollment:** Adding sites, dose-ranging cohorts & open label extension	**Phase 2: 15-week treatment** Randomized, placebo-controlled US, Europe and Israel study sites		**Expansion underway**		**Top-line data expected 2H 2024**
Systemic Sclerosis	**Initiating Trial:** Establish proof-of-biology; assess effects on skin, lung, vascular mechanisms			**Phase 2**: Randomized, placebo-controlled US, Europe and Israel study sites		**Trial initiation 4Q 2022/1Q 2023** **Top-line data expected 2H 2024**



Chemomab
THERAPEUTICS

Clinical stage biotech company discovering and developing **innovative therapeutics** for fibro-inflammatory diseases with high unmet need

Summary

CM-101: Addresses CCL24 - Unique and Differentiated Target

- Novel, **broadly applicable** fibro-inflammatory disease target

Disease-Modifying Pipeline in a Product

- **De-risked** by extensive animal, patient sample & early human data

Focus on High Value Rare Diseases

- With **high unmet need** & strong **commercial potential**

Clinical Trials Designed to:

- Reduce risk—**increase probability of success**
- Position CM-101 for potential registrational studies
- Preserve capital—cash runway through estimated end-2023

Opportunities in Other Indications

- That share **similar biologic pathways** & pathology

Thank you.

Chemomab
THERAPEUTICS