SEPTEMBER 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.

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Chemomab
THERAPEUTICS

Clinical stage biotech company discovering and developing **innovative therapeutics** for rare 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 Phase 2 starting 4Q 2022
- First data readout in 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

- $51.8M cash & equivalents (06/30/22)
- 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 and COO

Orchid · Abbott · Bayer · Boehringer Ingelheim · concentric

David Weiner, MD
Interim Chief Medical
Officer

Acadia · Lumos Pharma · PTI Proteostasis · aTyr · EMD Serono

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

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

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

Neil Cohen, MA
Director

Alan Moses, MD
Director

Nissim Darvish, MD, PhD
Director

Claude Nicaise, MD
Director

Jill Quigley, JD
Director

SCIENTIFIC ADVISORS (Selected)

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

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

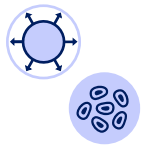
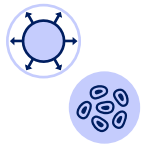
Scott L. Friedman, MD
Dean for Therapeutic Discovery and Chief, Division of Liver Diseases

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

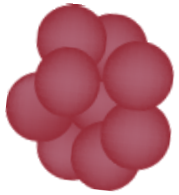
Massimo Pinzani, MD, PhD, FRCP
Sheila Sherlock Chair of Hepatology, Director
UCL Institute for Liver and Digestive Health

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		**Liver Fibrosis Biomarkers & Subcutaneous Formulation**	CCL24	SC Route of Administration and biomarker data				◆ Enrollment 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			◆ P2 Initiates		**Trial Initiates 4Q 2022**
		**Fibro-inflammatory Diseases**	CCL24						
Next-Gen (Novel Targets)		**TBD**	Screening						

Differentiated by Our Dual-acting, Novel Target: CCL24



CCL24
The Target

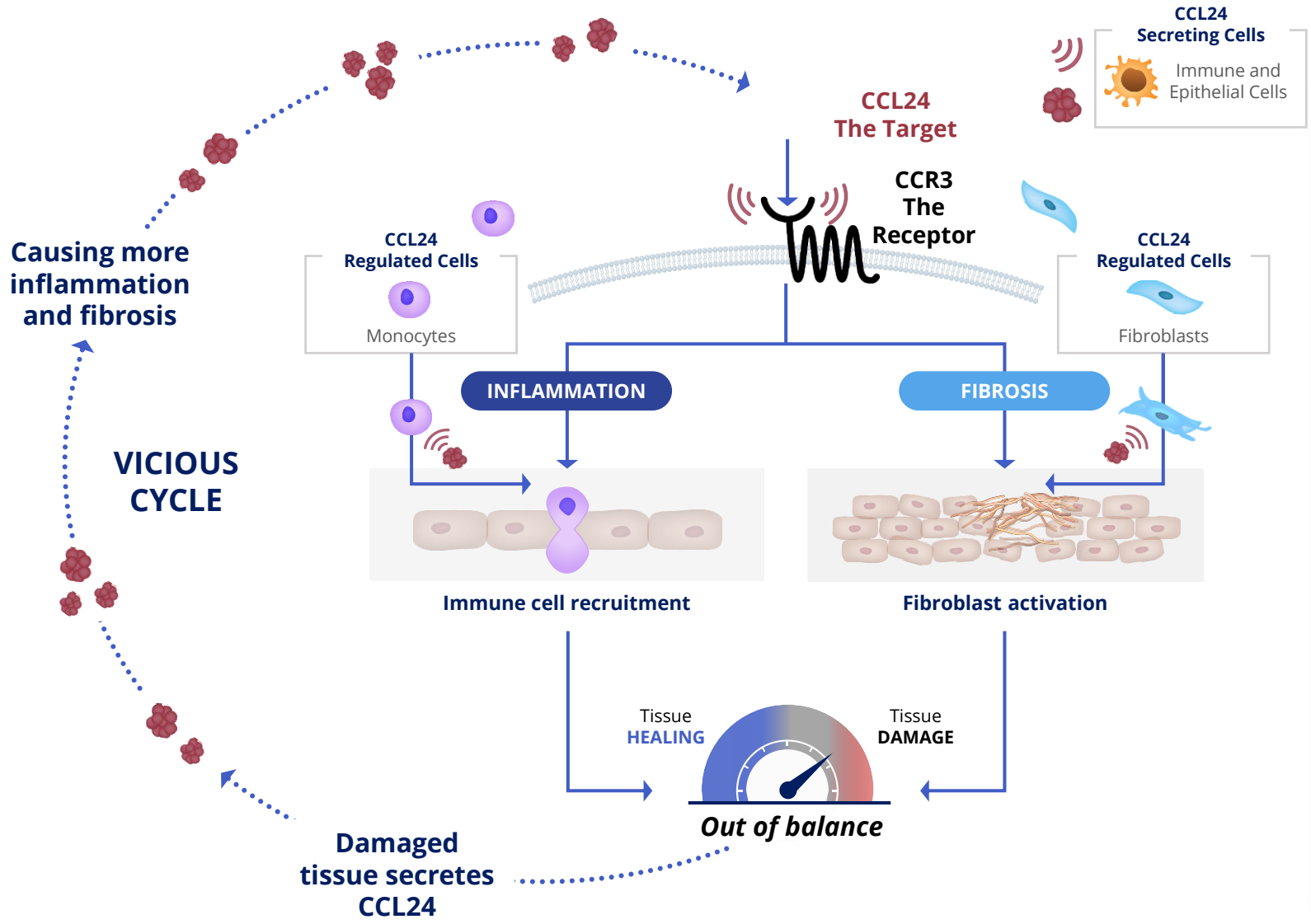
A cytokine-family, soluble protein central to the biology of inflammation and fibrosis

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

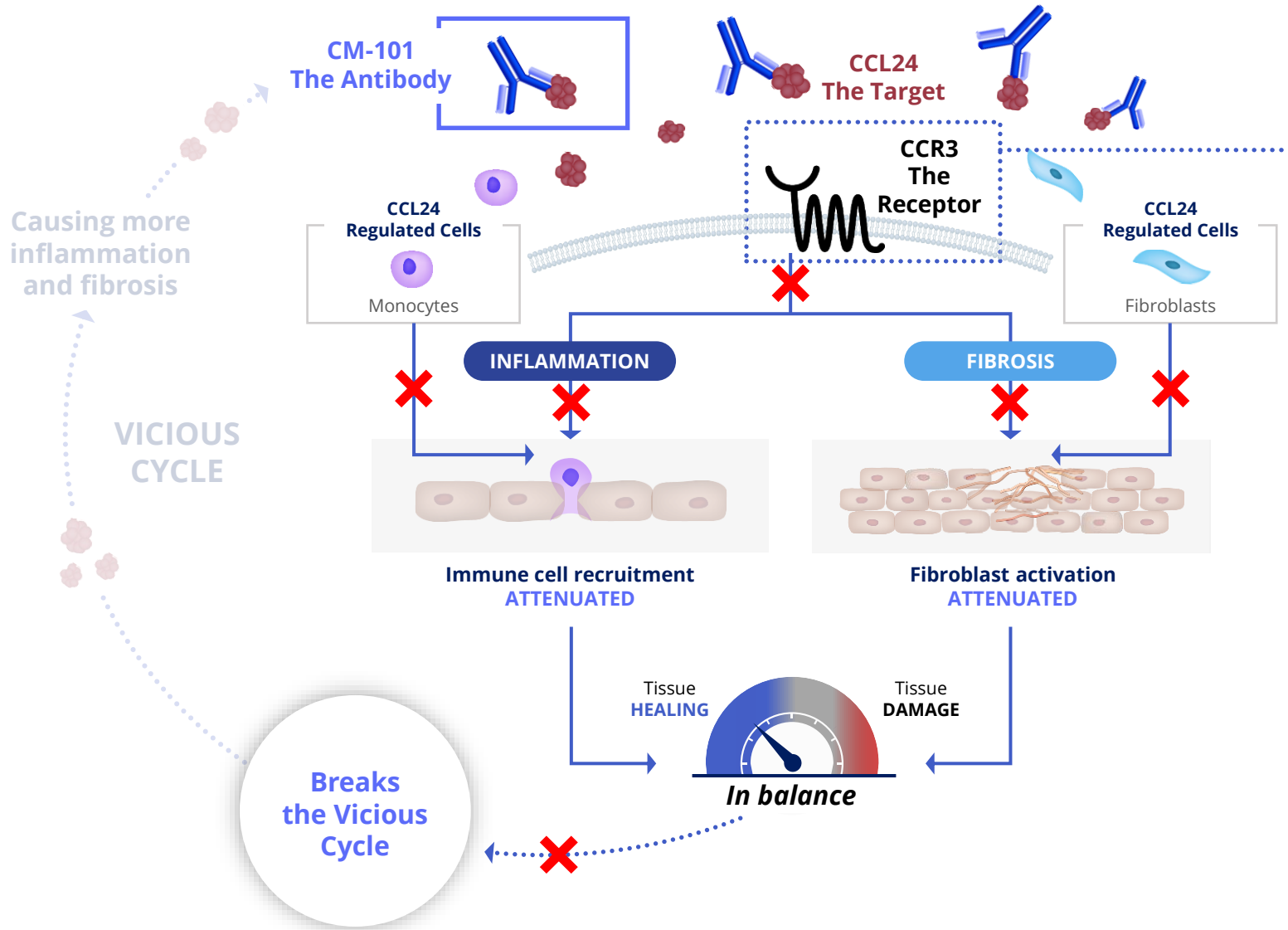
Correlates with fibrotic biomarkers and disease outcome

Low in Healthy Tissue; Elevated in Fibrotic Tissue

Liver, skin, lung, kidney, others

Wide therapeutic margin

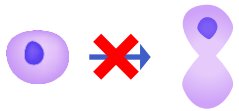
CM-101 Neutralizes CCL24, Breaking Vicious Cycle



Neutralizing CCL24 Has Advantages Over Blocking the Receptor

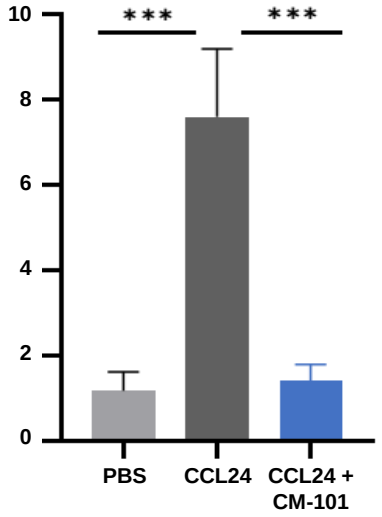
- Avoids context dependence of CCR3 receptor
- Efficacy of direct approach
- Favorable safety profile; retains normal repair functions

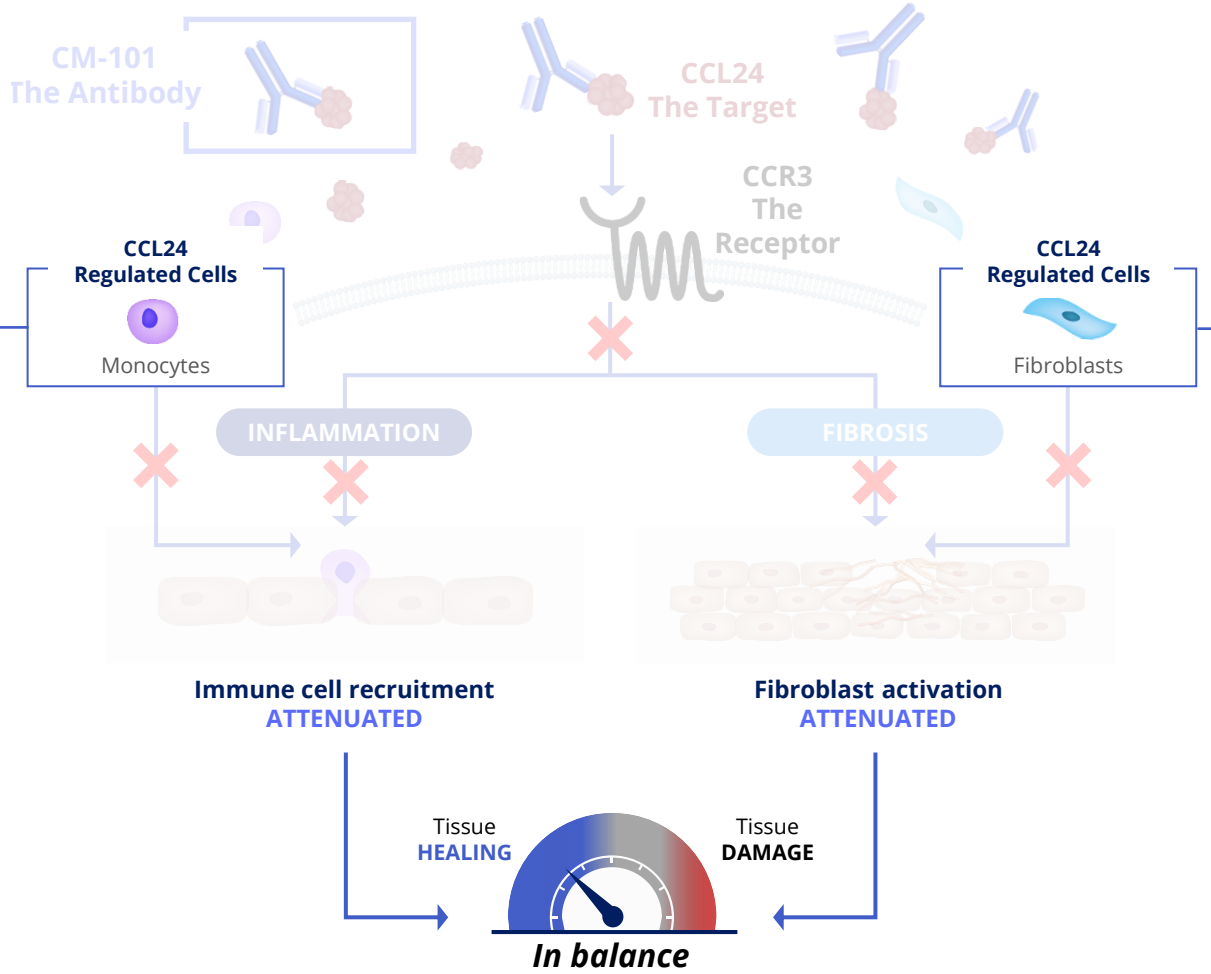
The Proof: First-in-Class CM-101 Neutralizes CCL24



CM-101 Reduces *In vivo* Monocyte Recruitment

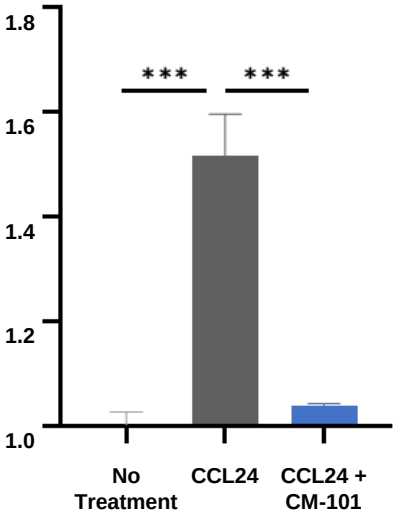
Number of Migrated Monocytes (x1000)





CM-101 Inhibits Primary Fibroblast Activation

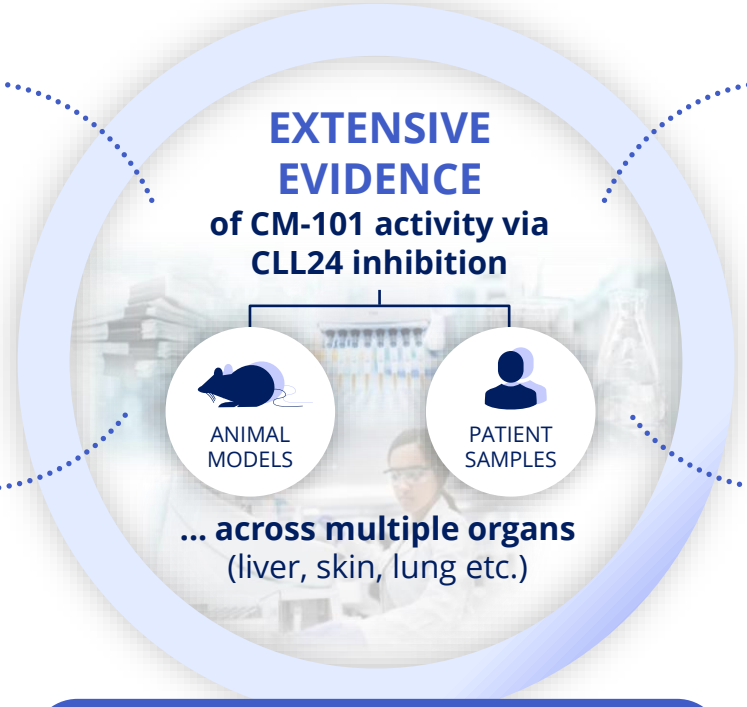
Proliferation: Relative Change from Baseline



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 CLL24 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

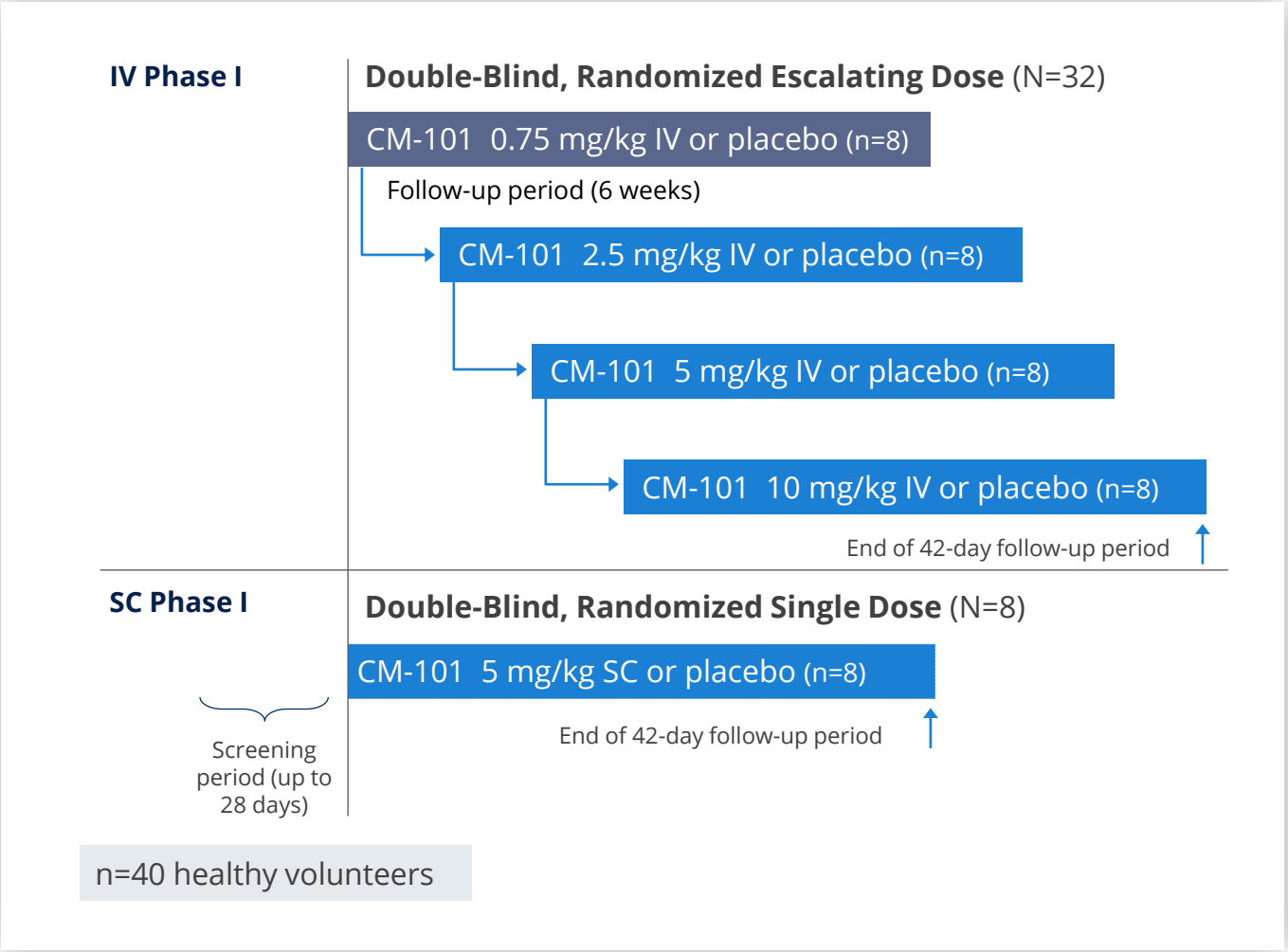
CM-101 was **safe and well tolerated** at all tested doses up to 10 mg/kg and for both IV and SC formulations

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 NAFLD* Patients

PATIENTS

PHASE 1B MULTIPLE ADMINISTRATION STUDY

Study population: **NAFLD patients** with normal liver function

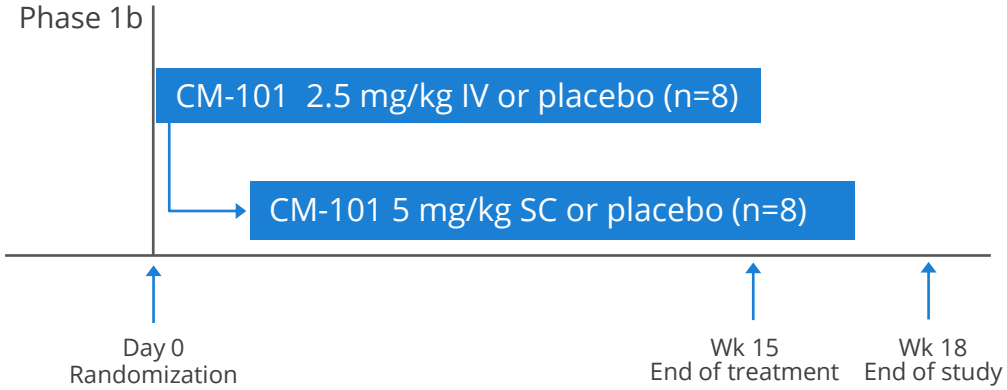
Multiple CM-101 administrations were **safe and well tolerated** using both **IV** and **SC formulations**

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 patient; Q3W
- Primary endpoints - safety and tolerability

* Nonalcoholic fatty liver disease

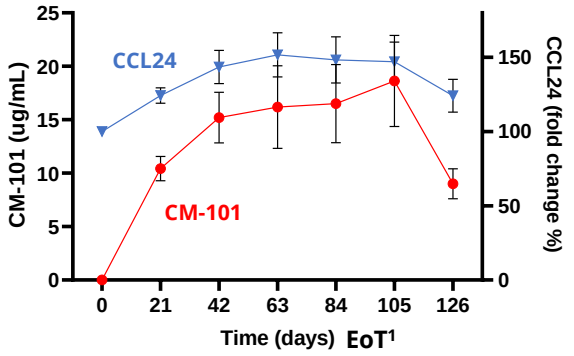
Anti-Fibrotic Mechanisms of CM-101 Confirmed in NAFLD Patients

PATIENTS

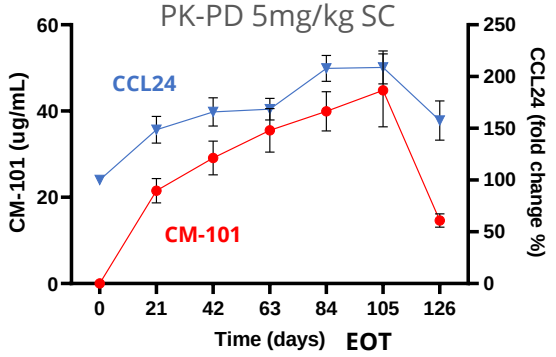
CM-101

PK-TARGET ENGAGEMENT

PK-PD 2.5mg/kg IV



PK-PD 5mg/kg SC



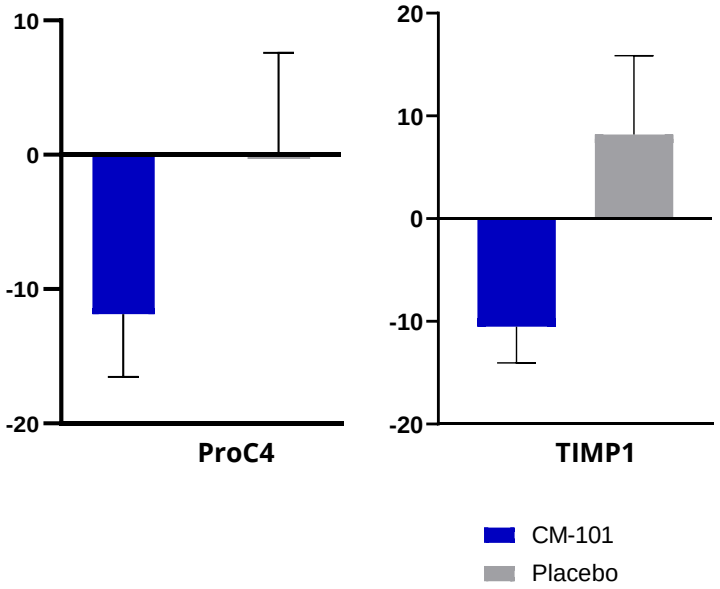
Clear correlation between antibody (red) and target (blue) levels

Dose dependent PK-target engagement

FIBROSIS

Biomarkers*

Relative Change from Baseline (%)



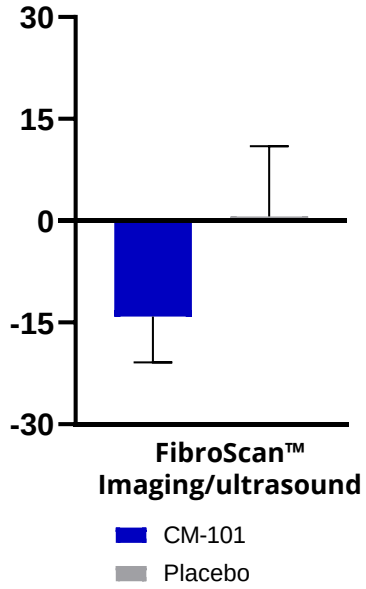
Concordant results across 6 additional fibrotic markers

Improved liver biomarkers

LIVER STIFFNESS

Physiological Measure

Relative change from baseline (%)



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

Decreased liver stiffness

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

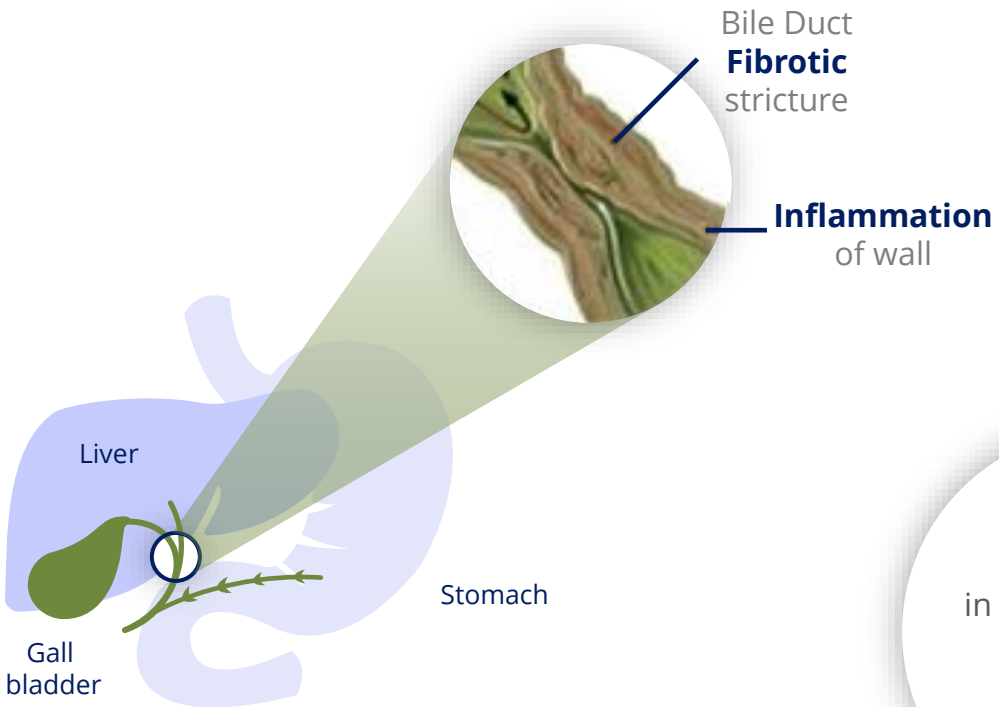
CM-101

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

~77K
patients
in 7 major markets

>$1B
est. commercial opportunity

Liver transplant only therapy with some curative potential

Source: Johns Hopkins Medicine

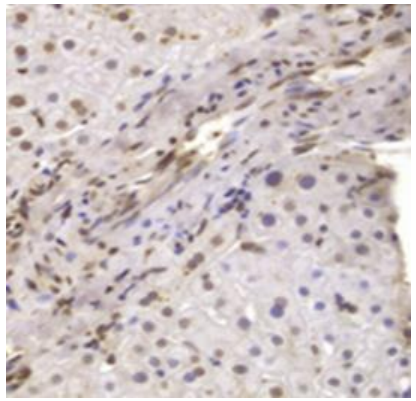
CCL24 Plays a Key Role in PSC

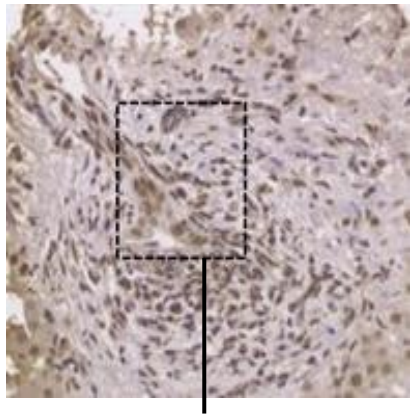
PATIENT SAMPLES

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

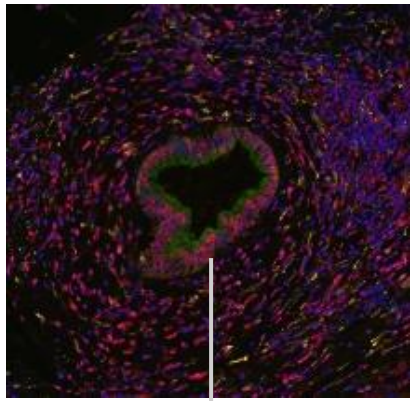
Healthy Liver | **PSC Livers**

Ccl24 Staining





CCL24 (brown) overexpressed in damaged bile duct area

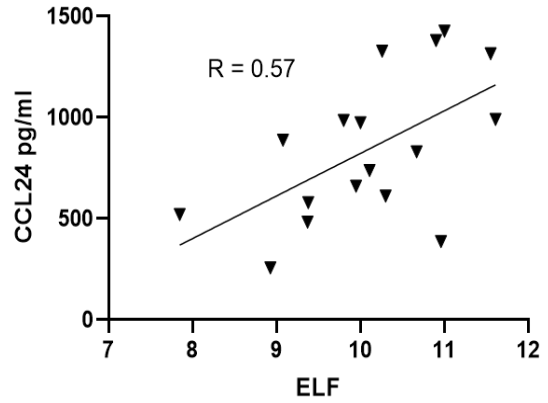


CCL24 (red) overexpressed in bile epithelial cells (green) and immune cells (yellow)

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

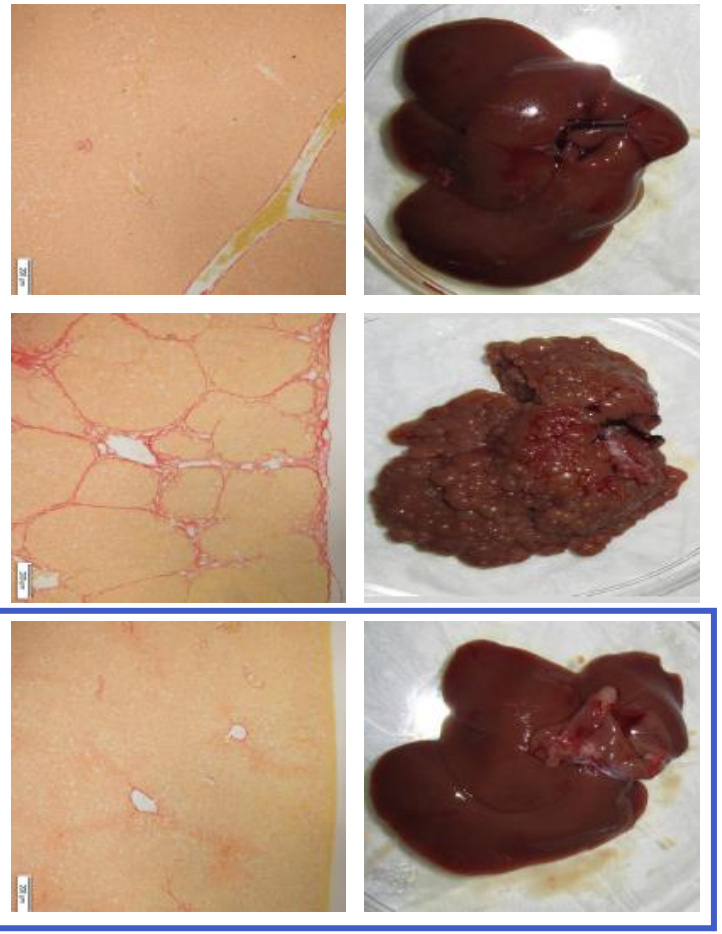
1. Collaboration with RFH, UK
ELF – Enhanced Liver Fibrosis; ALP – Alkaline Phosphatase

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

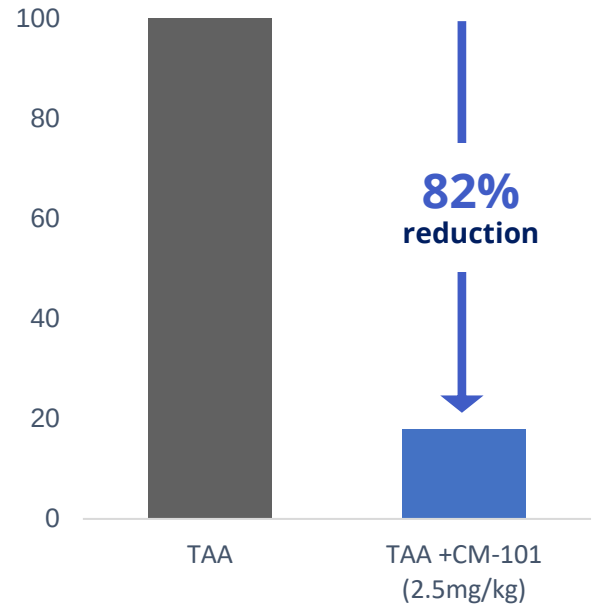
ANIMAL MODELS (TAA RAT_

RAT MODEL LIVERS

SIRIUS RED (COLLAGEN)



% FIBROSIS
(SIRIUS RED STAINING)



Pro-fibrotic Genes
Col1A1
Col3A1
TIMP1
ACTA2
TGF-β

Liver Enzymes
ALT
AST
ALP

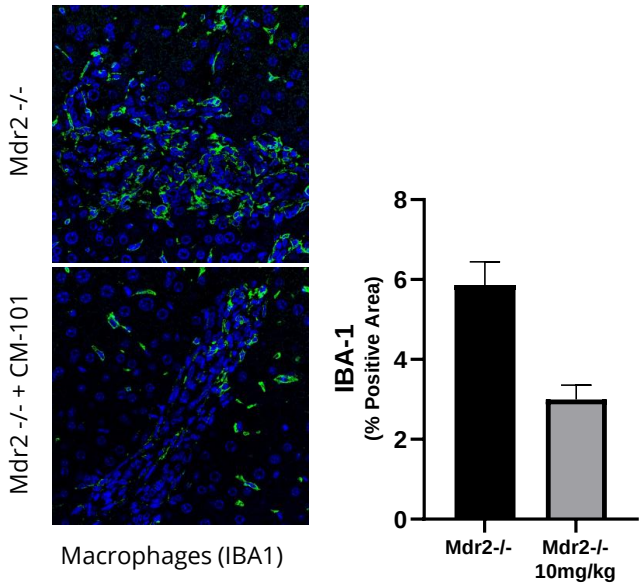
Dramatically reduced liver collagen in TAA liver fibrosis rat model with established liver fibrosis

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

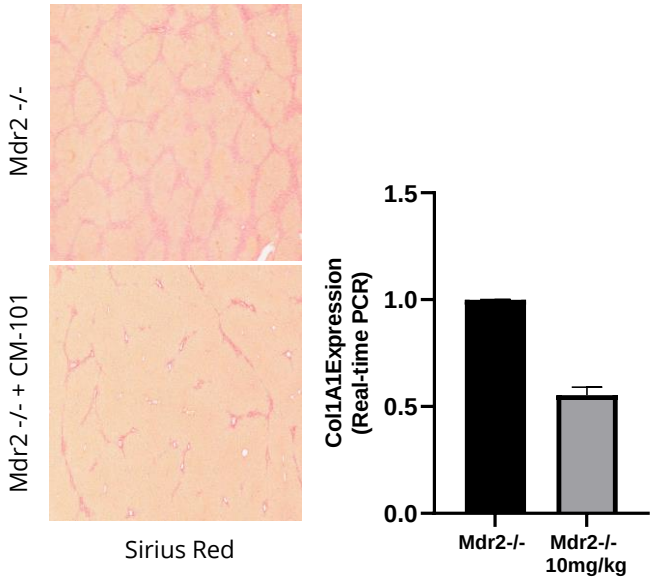
REDUCED MACROPHAGE NUMBER



Macrophages (IBA1)

Reduction in
INFLAMMATION

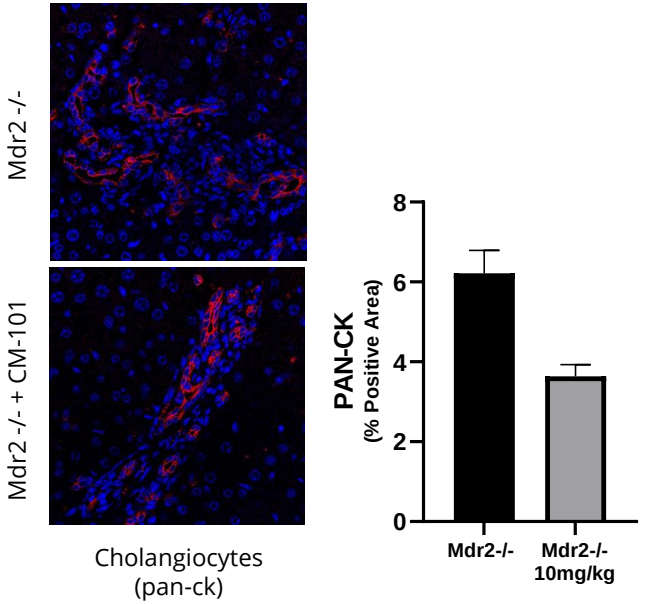
ATTENUATED LIVER COLLAGEN DEPOSITION



Sirius Red

Reduction in
FIBROSIS

REDUCED ABERRANT BILE EPITHELIAL PROLIFERATION



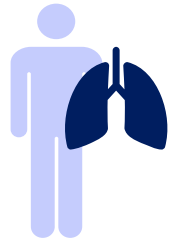
Cholangiocytes
(pan-ck)

Main driver of
DISEASE PATHOLOGY

CM-101 interferes with core pathways that drive PSC

CM-101

for the Treatment of Systemic Sclerosis (SSc)

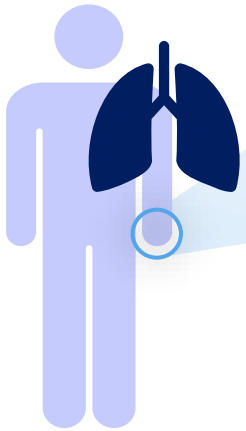


Systemic Sclerosis (SSc): Most Lethal Systemic Rheumatic Disease

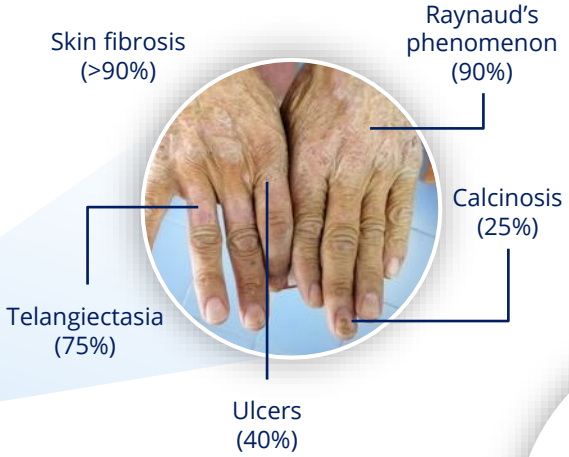
Rare Autoimmune Rheumatic Disease

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

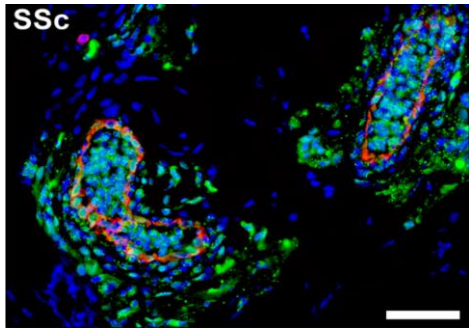
Nintenadib, Tocilizumab only treat symptoms, **NOT** disease modifying

Source: Johns Hopkins Medicine

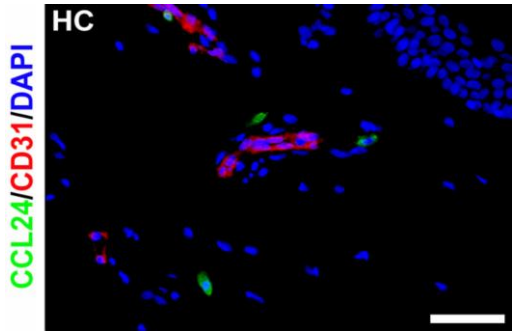
CCL24: A Critical Node Potentiating Systemic Sclerosis

PATIENT SAMPLES

SSc Skin

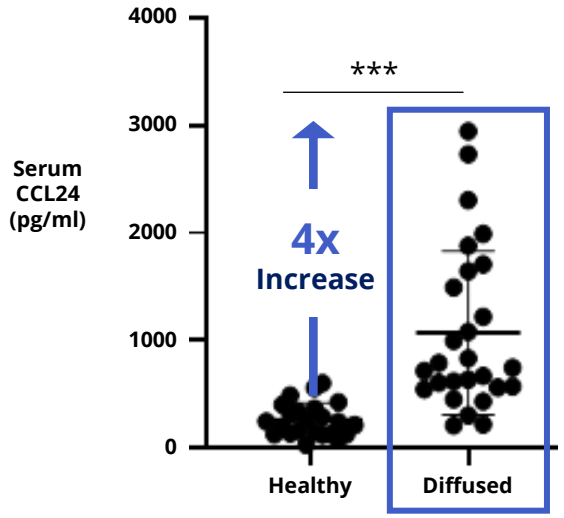


Healthy Skin



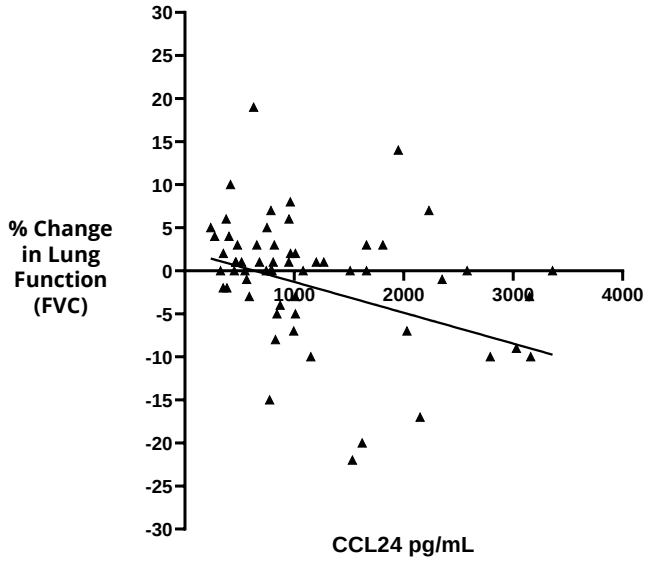
Collaboration with University of Florence

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

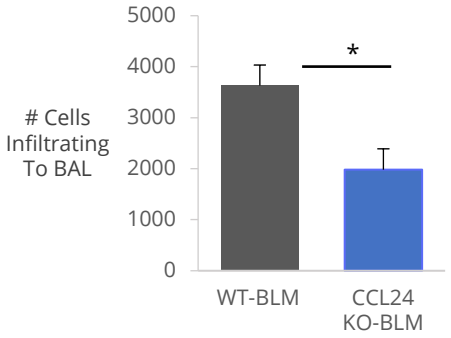
Data from patient samples supports potential therapeutic utility of blocking CCL24 in systemic sclerosis

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

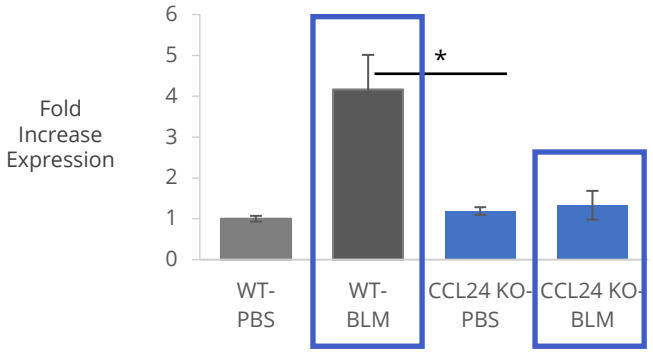
Target Validation: CCL24 Gene Knockout Ameliorates Induced SSc

ANIMAL MODELS
SSc BLEOMYCIN
MOUSE MODEL

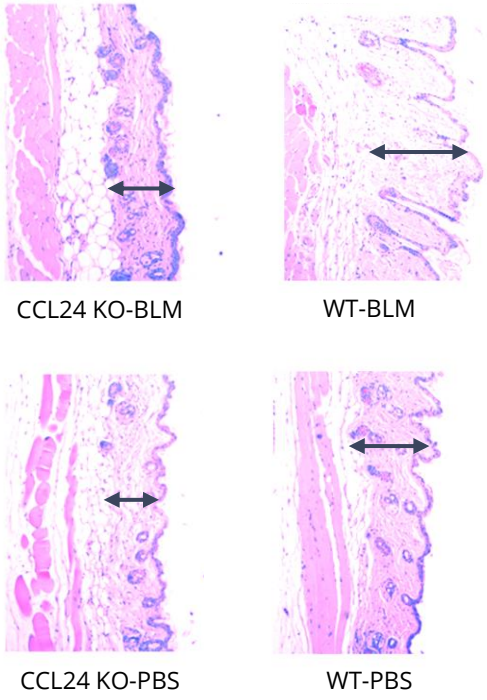
Immune Cell Lung Infiltration

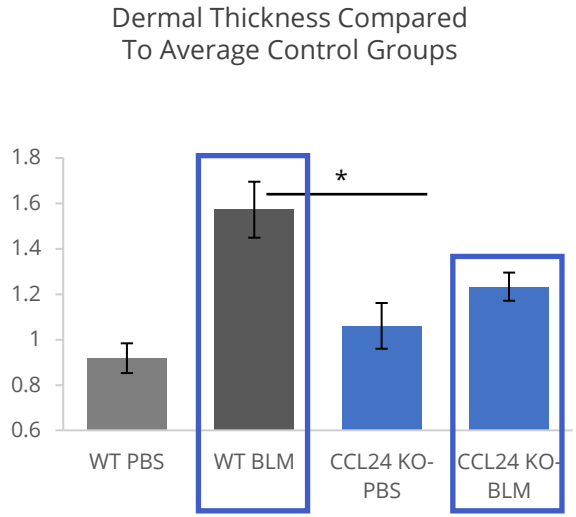


α SMA Expression in Skin Lesions



Dermal Thickness**





CCL24 Knockout attenuates 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

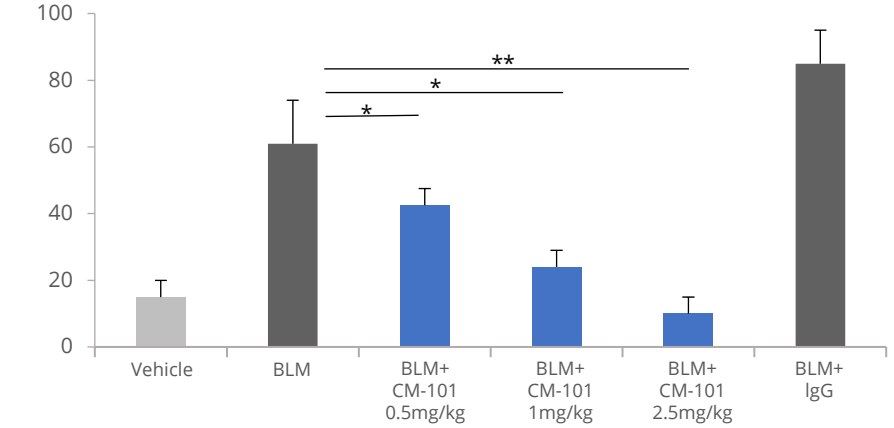
CM-101 Significantly Reduces Skin and Lung Fibrosis

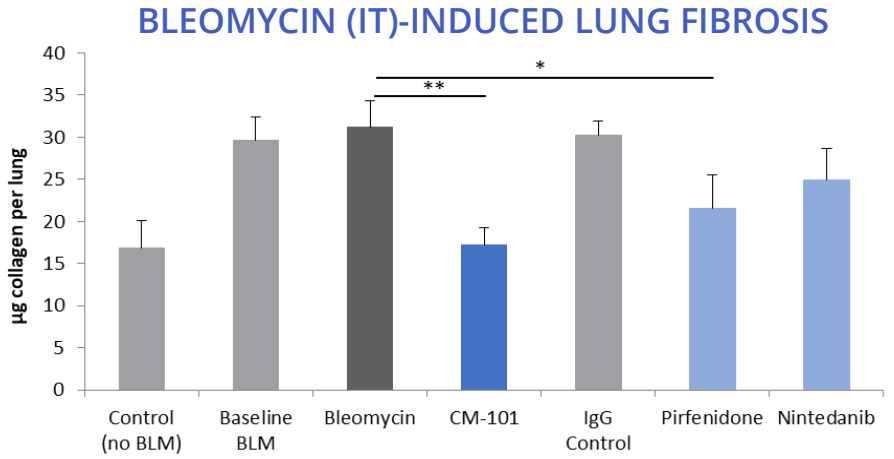
BLEOMYCIN-INDUCED ANIMAL MODEL

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

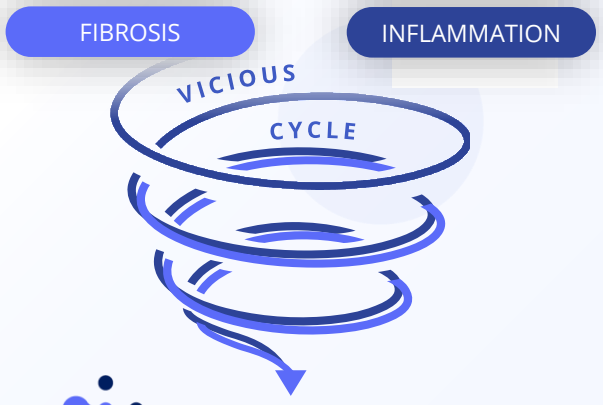




Mor et al, Annals of Rheumatoid Diseases, 2019
**p ≤0.01; *p ≤0.05.
BLM - bleomycin; IT - Intratracheal; SC – Subcutaneous IgG - Immunoglobulin G

CM-101 Clinical Development Plan Overview

	STATUS	2021	2022	2023	
Liver Fibrosis/ Subcutaneous Formulation	**Enrollment Concluded:** Measure safety, PK, biomarkers and assess SC formulation	**Phase 2a: 16-week treatment** Randomized, placebo-controlled Israel study sites			**Topline data expected 4Q 2022**
Primary Sclerosing Cholangitis	**Expanding Enrollment:** Adding sites, dose-ranging cohorts & open label extension	**Phase 2: 15-week treatment** Randomized, placebo-controlled US, Europe and Israel study sites		**Expanding** To estimated 93 total patients	**Topline data expected 2H 2024**
Systemic Sclerosis	**In Planning:** Establish proof-of-biology; assess effects on skin, lung, vascular mechanisms			**Phase 2:** Randomized, placebo-controlled US, Europe and Israel study sites	**Trial initiation expected 4Q 2022**



Chemomab
THERAPEUTICS

Clinical stage biotech company discovering and developing **innovative therapeutics** for rare 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 to estimated end-2023

Opportunities in Other Indications

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

Thank you.

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THERAPEUTICS