Chemomab
THERAPEUTICS

Pioneering Innovative Treatments for Fibrotic Diseases

Corporate Overview | Non-Confidential | September 2021

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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Investment Highlights

Clinical Stage Biotech Company Focused on Inflammatory-Fibrotic Diseases

Focus	• Discovery and development of innovative therapeutics for rare inflammatory and fibrotic diseases with high unmet need

CM-101: A Pipeline in a Product	• CM-101, a first-in-class CCL24 neutralizing monoclonal antibody targeting:
	  ○ Primary Sclerosing Cholangitis (PSC)
	  ○ Systemic Sclerosis (SSc)
	  ○ Liver fibrosis (MoA*)

Near-Term Catalysts	• Conducting 2 Phase II clinical trials with 3rd Phase II trial expected to initiate in 1Q22 • First clinical readouts targeted for 2022 expected to drive multiple value inflections
Robust IP Portfolio	• Global patent exclusivity through 2041. Granted composition of matter patent with multiple additional patents pending.
Top Tier Investors	OrbiMed Healthcare Fund Management; **Cormorant Asset Management**; THIEL
Solid Balance Sheet	• Cash and equivalents of $67M as of June 30, 2021 enabling runway through mid-2023

*MoA – Mechanism of Action

Experienced Management

ADI MOR, PhD

Founding Chief Executive Officer &
Chief Scientific Officer

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Tel-Aviv Sourasky Medical Center

DALE PFOST, PhD*

Incoming Chief Executive Officer

OPKO

Advent Life Sciences

SmithKline Beckman CORPORATION

ORCHID

SIGAL FATTAL, CPA

Chief Financial Officer

BiomX

evogene DECODING BIOLOGY

ARNON AHARON, MD

Chief Medical Officer

BIOLINERX

THROMBOTECH

PHARMOS

SHARON ELKOBI, MSc, MBA

VP Business Development

teva

MEDISON Delivering Innovative Healthcare

Neurim PHARMACEUTICALS

MICHAL SEGAL-SALTO, PhD

VP Research and Development

TEL AVIV UNIVERSITY אוניברסיטת תל אביב

מכון ויצמן למדע WEIZMANN INSTITUTE OF SCIENCE

*Post shareholder vote

Experienced Leadership

Board of Directors

Stephen Squinto, PhD
Chairman of the Board

Adi Mor, PhD
Founding Chief Executive Officer & Chief Scientific Officer

Alan Moses, MD
Director

Claude Nicaise, MD
Director

Dale Pfost, PhD*
Incoming Chief Executive Officer

Joel Maryles, CFA, MBA
Director

Neil Cohen, MA
Director

Nissim Darvish, MD, PhD
Director

*Post shareholder vote

Scientific Advisory Board (partial)

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

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

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

Gideon Hirschfield, MA MB PhD
Lily and Terry Horner Chair in Autoimmune Liver Disease, University of Toronto, Toronto General Hospital, Canada

Prof. Dinesh Khanna MD, MBBS, MSc
Director of the Scleroderma Program, University of Michigan, Ann Arbor, Michigan, USA

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

CM-101: Pipeline in a Product

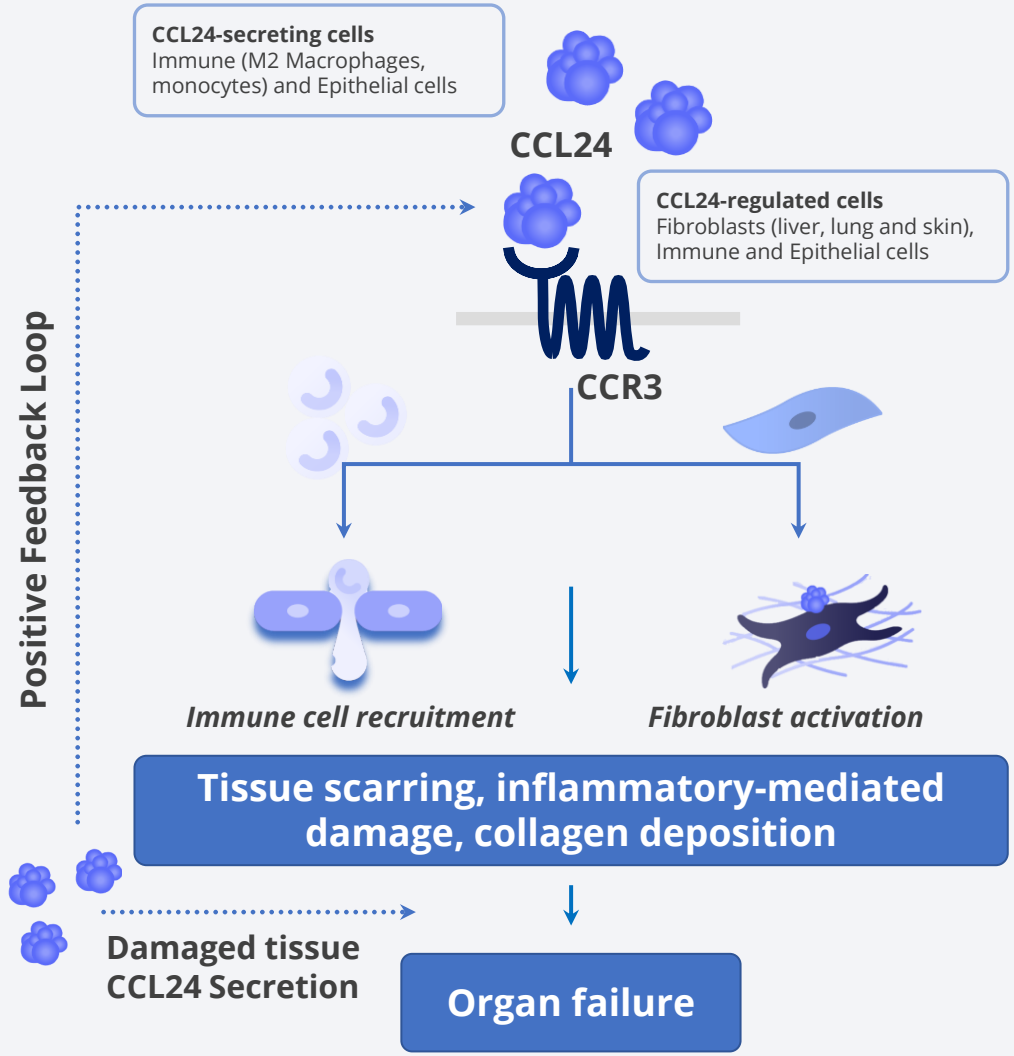
Addressing Fibrotic Diseases with High Unmet Need

Program	Indication	Target	Discovery	Preclinical	Phase 1	Phase 2	Status
CM-101	Primary Sclerosing Cholangitis	CCL24	*Orphan designation granted from FDA and EMA*				Enrolling Patients Top Line Data 2H 2022
CM-101	Systemic Sclerosis	CCL24	*Orphan designation granted from FDA and EMA*				Initiation Expected In 1Q 2022
CM-101	Liver Fibrosis (MoA*)	CCL24	*Mechanism of Action & SC Route of Administration (in NASH patients)*				Enrolling Patients Top Line Data 1H 2022
Next-gen	Fibrosis	Screening					In Progress

*MoA – Mechanism of Action

CCL24 is a Novel Therapeutic Target for Fibrosis

Critical Mediator Promoting Inflammation and Fibrosis

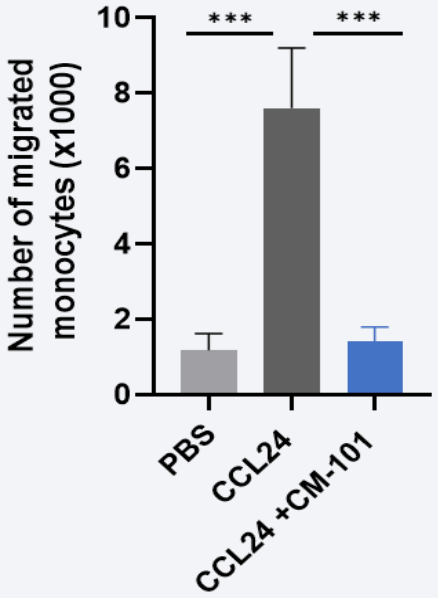


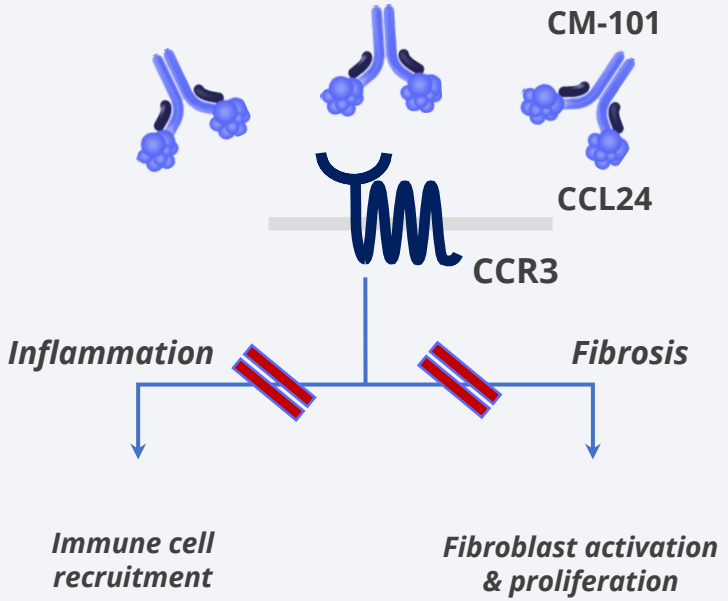
- ✓ **Dual role in promoting fibrosis**
  - directly activates fibroblasts
  - enhances local immune cell recruitment
- ✓ **Unique and differentiated activity**
  - ex vivo and in vivo data confirms unique role vs other CCLs
  - correlates with disease outcome and fibrotic biomarkers
- ✓ **Minor expression in healthy tissue**
  - significantly elevated in liver, skin, lung fibrotic tissue
  - wide therapeutic margin
- ✓ **Positive feedback loop potentiates tissue damage**
  - responsible for initiation and perpetuation of fibrosis

CM-101: A First-in-Class mAb Blocking CCL24

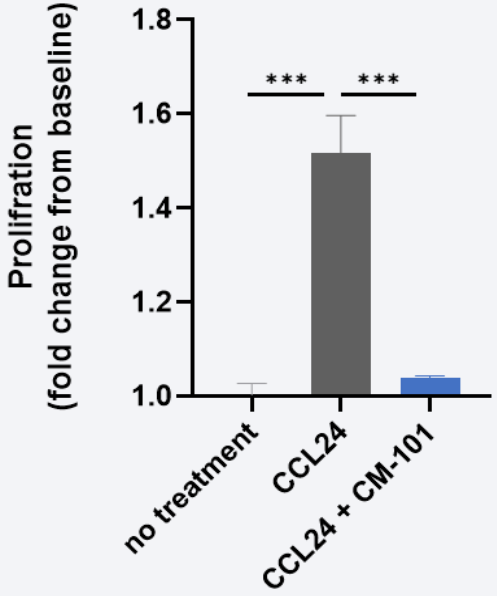
Dual Mechanism of Action Interfering with the Core Fibrotic Pathways

CM-101 Reduces In-vivo Monocyte Recruitment





CM-101 Inhibits Primary Hepatic Fibroblast Activation



CM-101 attenuates inflammation and fibrosis by inhibiting fibroblast activation and immune cell recruitment

Strong Preclinical Evidence of CM-101 via CCL24 Inhibition

Significant Attenuation of Fibrosis & Inflammation Across a Wide Range of Models

CCL24 Target Validation

Ex-Vivo (Patient Samples)

Primary sclerosing cholangitis

- Biomarker correlations
- Overexpression of CCL24 and CCR3

Systemic Sclerosis

- Fibrotic biomarkers correlation
- Disease deterioration correlation
- Overexpression of CCL24 and CCR3

Liver Fibrosis (NASH)

- Disease severity correlation
- Overexpression of CCL24 and CCR3

In-Vivo (Knockout Animal Models)

Systemic Sclerosis

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

Liver metabolism and inflammation

- CCL24 knockout vs. WT in MCD-induced NASH (mice)

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)

Mechanism of Action

CM-101 effects on fibroblast activation

- Dermal, Hepatic and Lung fibroblast activation
- Dermal and liver fibroblast transition to myofibroblasts
- Hepatic fibroblast motility

CM-101 effects on immune cells migration and recruitment

- Dermal fibroblast migration
- Monocyte polarization
- Monocyte recruitment

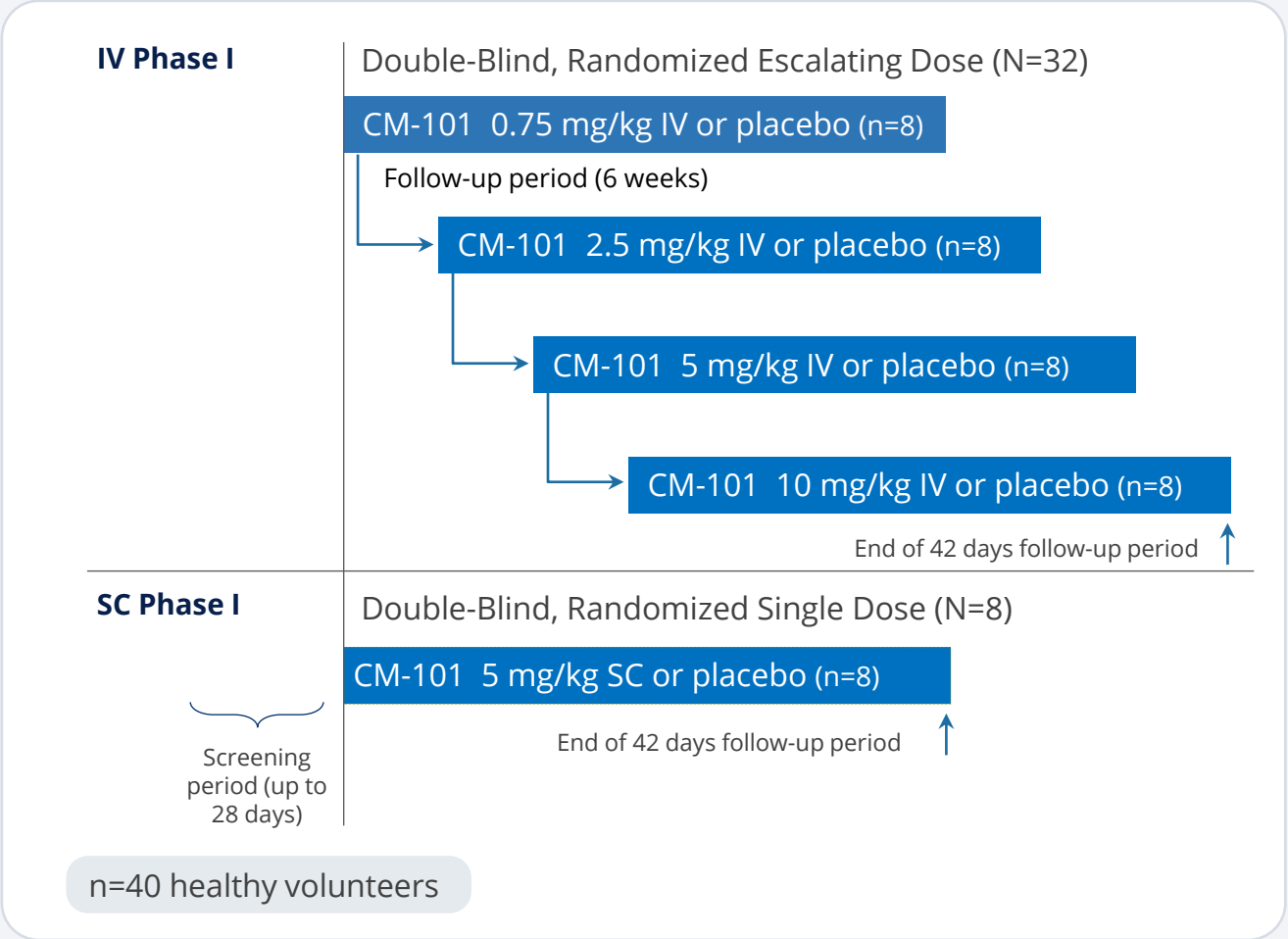
Toxicology

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

Phase Ia Results

Single Ascending Dose Study Shows CM-101 to be Safe and Well Tolerated in Healthy Volunteers

- CM-101 was **safe and well tolerated** at all tested doses up to 10 mg/kg and for both formulations
- All drug related AEs were classified as **mild with no SAEs reported**
- Average half-life of 19-21 days (for IV and SC), supports long interval **administration once every 2-4 weeks**
- **Dose-dependent target engagement** measured by serum CCL24 levels
- **Comparable target engagement & PK profiles** for the SC and IV formulations



IV - Intravenous; SC - Subcutaneous

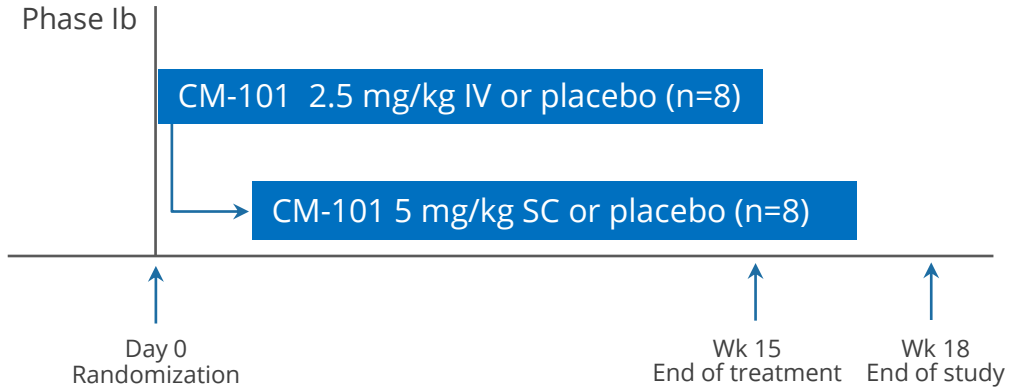
Phase Ib Results

Multiple Administration Study Showed CM-101 to be Safe & Well Tolerated in NAFLD Patients

- 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 $t_{1/2}$, supports **long dosing interval** (Q2W - Q4W)

Study Design

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



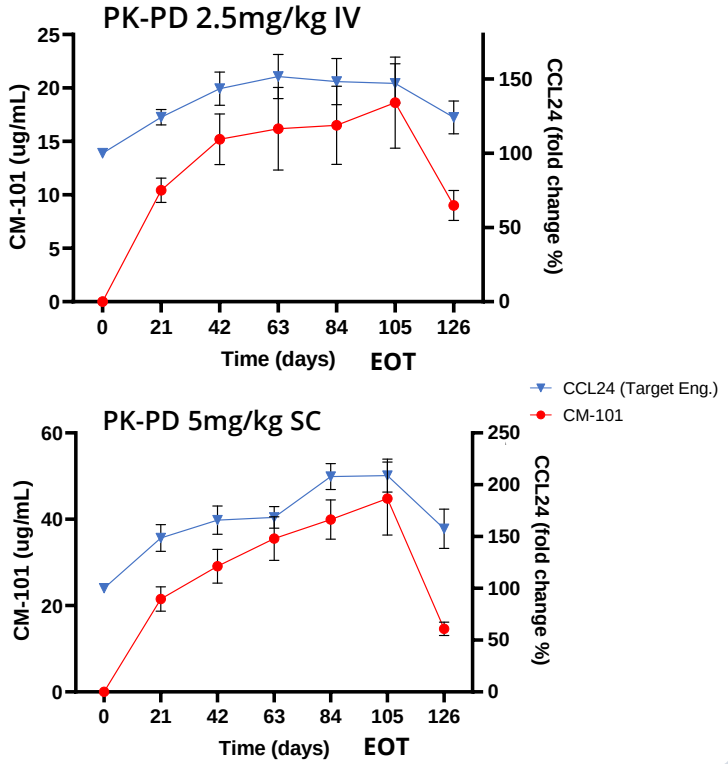
- Tested doses - 2.5 mg/kg IV infusion and 5 mg/kg SC injection
- 5 repeated administrations per patient; Q3W
- Primary endpoint - safety and tolerability

IV - Intravenous; SC – Subcutaneous; Wk - week

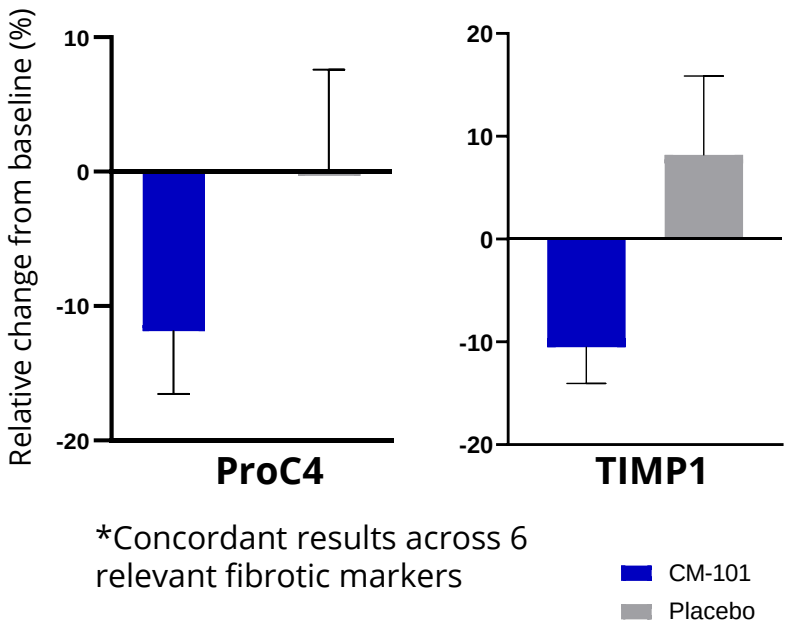
Phase Ib: Confirms CM-101 Anti-Fibrotic Mechanism in Humans

Demonstrated Biomarker Improvement with Dose-Dependent PK-Target Engagement

PK-Target Engagement

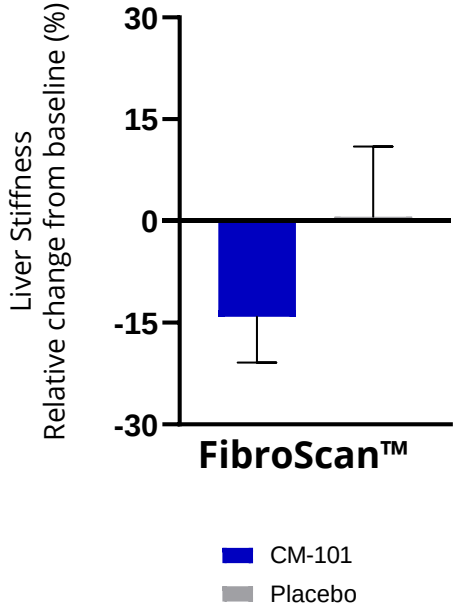


Fibrotic Biomarkers Improved*



Nordic Biosciences , Denmark

Elastography Decreased



ProC4-Procollagen 4
TIMP1- metallopeptidase inhibitor 1
EOT-End of Treatment

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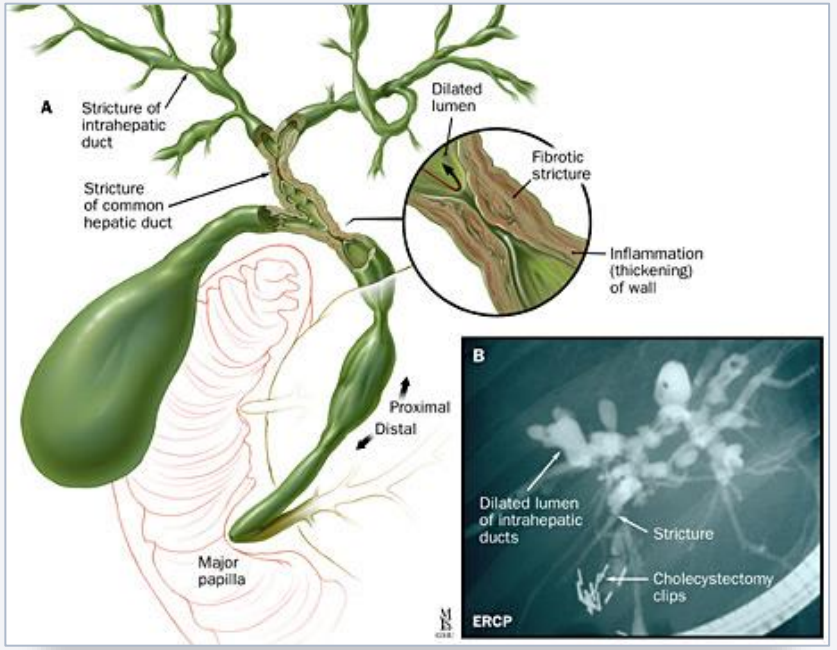
CM-101 for the Treatment of Primary Sclerosing Cholangitis

Primary Sclerosing Cholangitis (PSC)

Characterized by High Morbidity and Mortality

- **PSC is a rare, chronic liver disease** characterized by progressive course of cholestasis **with inflammation and fibrosis** of the intrahepatic and extrahepatic bile ducts **leading to end-stage liver disease and cirrhosis**
- Diagnosis is most often between the **age of 30 and 40 years**; Predominantly affect men (2:1 male:female ratio)
- **No FDA approved drug;** Liver transplant is the only therapy with curative potential
- **Median Survival of 10-12 years** with no intervention
- ~77K patients in 7 major markets



Source: Johns Hopkins Medicine

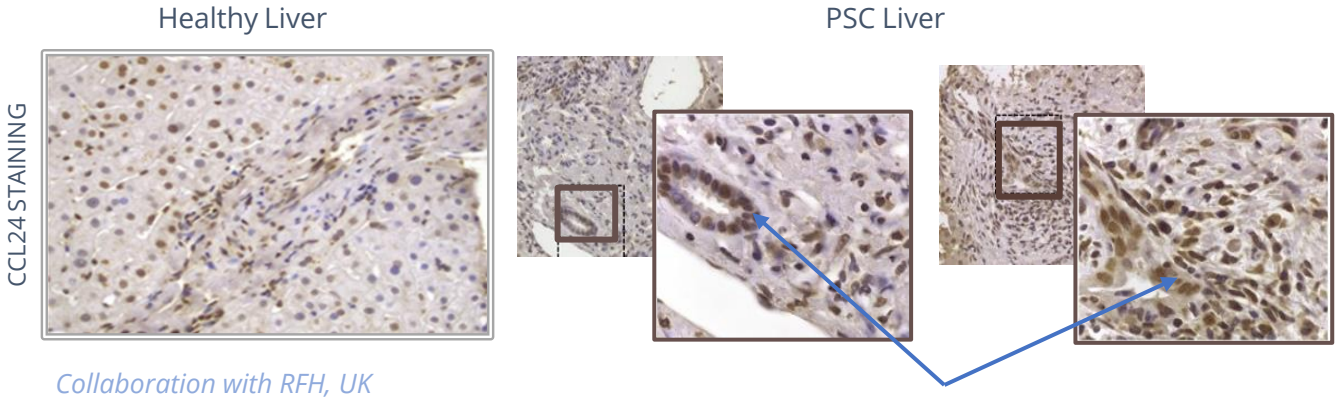
Lazaridis et al. Primary Sclerosing Cholangitis., N Engl J Med. 2016
James H et al.Primary Sclerosing Cholangitis, Part 1: Epidemiology, Etiopathogenesis, Clinical Features, and Treatment Gastroenterology & Hepatology 2018
7 Major Markets – USA, 5EU (UK, Germany, France, Italy, Spain), Japan

CCL24 Plays a Key Role in PSC

Clinical Relevance of CCL24 in PSC Pathophysiology is Supported by Patient Samples

CCL24 expression is significantly and selectively elevated in PSC livers

CCL24 level in healthy vs PSC patients' livers tissues

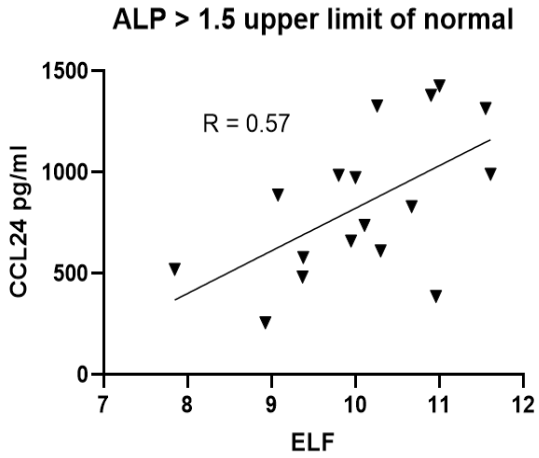


Collaboration with RFH, UK

CCL24 overexpressed in bile epithelial cells

CCL24 correlates with ELF in PSC patients with elevated ALP

CCL24 serum level in PSC patients



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

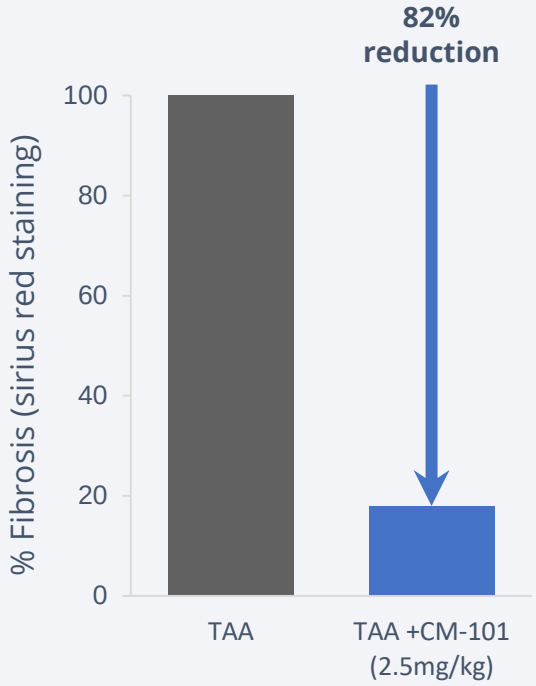
CM-101 MoA guided the selection of ELF and ALP as primary endpoints in Phase II

ELF – Enhanced Liver Fibrosis; ALP – Alkaline Phosphatase

1. M. Vesterhus et al. Hepatology, 62 (1) (2015), pp. 188-197

CM-101 Reduces Liver Fibrosis by 82%

Reduced Liver Collagen in TAA Liver Fibrosis Rat Model Using Therapeutic Design



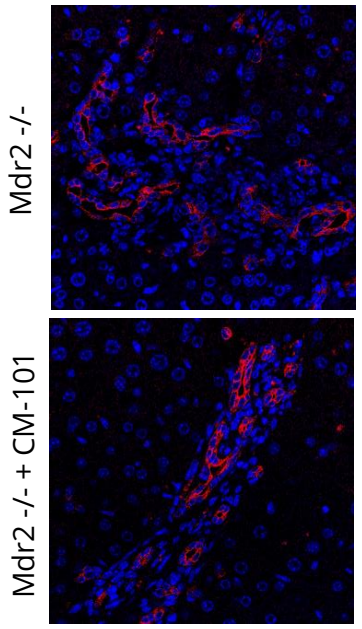
Pro-fibrotic genes
- Col1A1
- Col3A1
- TIMP1
- ACTA2
- TGF-β

Liver enzymes
- ALT
- AST
- ALP

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

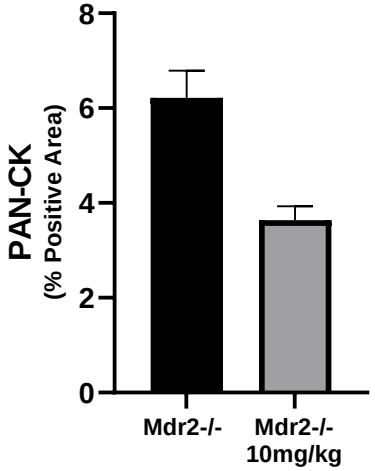
CM-101 Ameliorates Liver Injury and Fibrosis in PSC

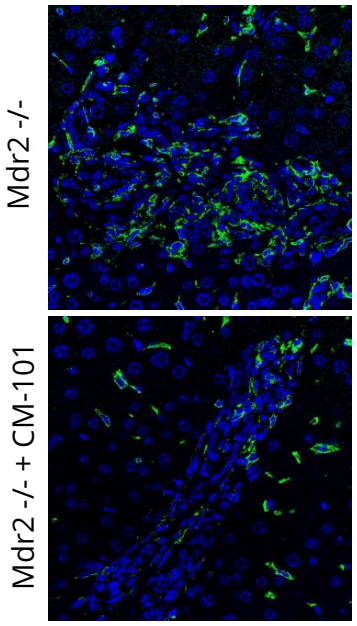
Cholestasis, Inflammation and Fibrosis are Reduced in the MDR2 Knockout Model in Mice



Cholangiocytes
(panck)

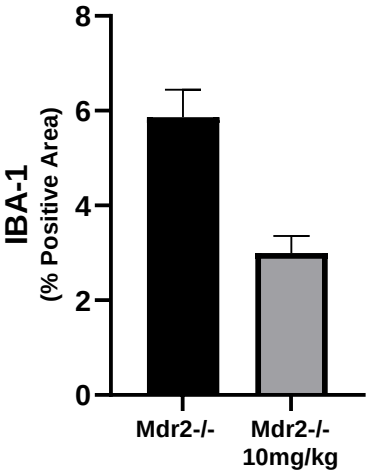
Decreased Bile Epithelial Cell Proliferation

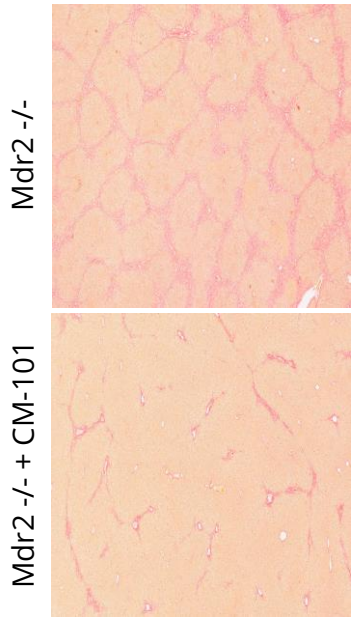




Macrophages (IBA1)

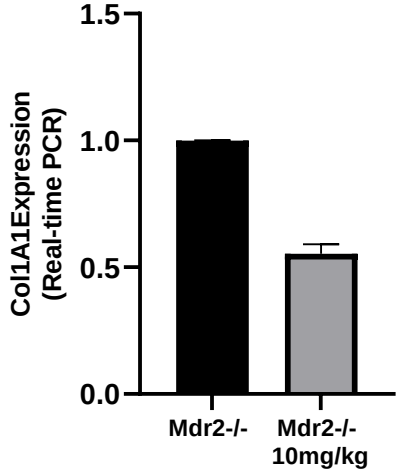
Reduced Macrophage Number





Sirius Red

Attenuated Liver Collagen Deposition



CM-101 interferes with the core pathways that drive PSC

Phase II PSC Study Enrolling Patients

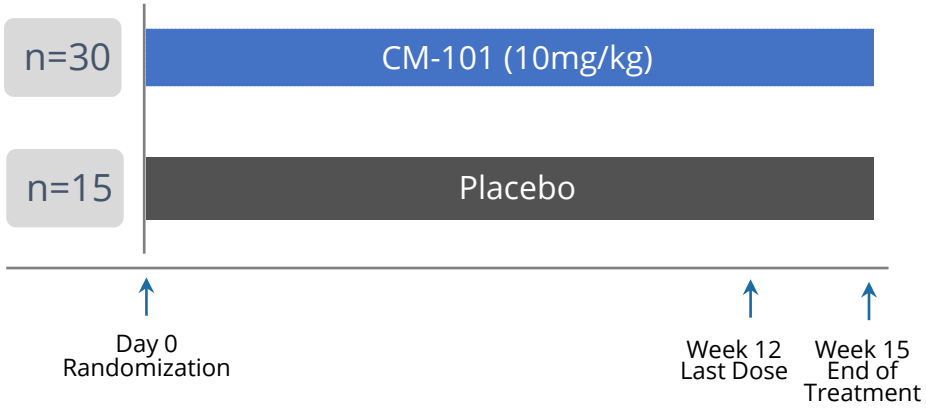
Orphan Drug Designation Granted by FDA and EMA

Trial Design

Key inclusion/exclusion criteria

- **Adults with large duct PSC** of more than 24 weeks duration, diagnosed via MRCP/ERCP
- **ALP > 1.5 ULN**
- Stable IBD is allowed
- Stable Ursodeoxycholic Acid (UDCA) treatment is allowed

IV Administrations of CM-101 vs. Placebo; Q3W



Primary endpoints:

- **Serum alkaline phosphatase** Change from baseline to W15
- **ELF score** Change from baseline to W15

Secondary endpoints:

- **Fibrotic biomarkers and liver enzymes (e.g. AST, ALT, Pro-C3, Pro-C5)** Change from baseline to W15
- PK, Anti Drug Antibody
- PD parameters

Topline data expected in 2H2022

IV - Intravenous

Clinicaltrials.gov: NCT04595825

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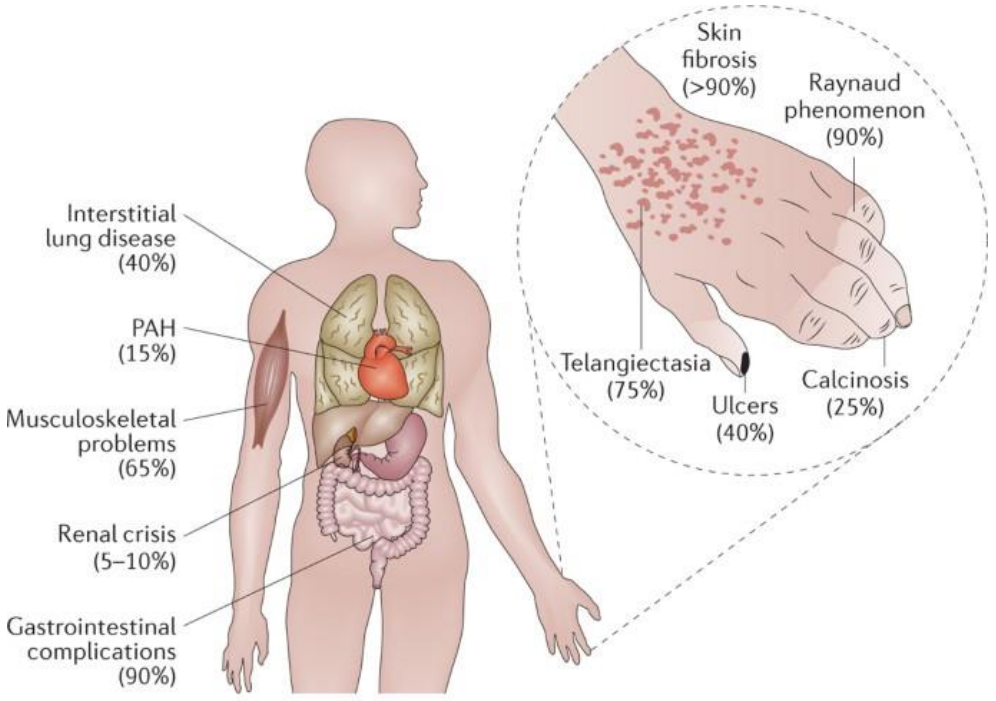
CM-101 for the Treatment of Systemic Sclerosis

Systemic Sclerosis (SSc)

Most Lethal Among Systemic Rheumatic Diseases

- **Rare** autoimmune rheumatic disease characterized by **inflammation and fibrosis** of the internal organs as well as vasculopathy
- **Age of diagnosis 30-50 years**, Predominantly affects women (3:1 female:male ratio)
- There is **no approved disease modifying drug;** Treatments focus on managing disease symptoms (Nintedanib, Tocilizumab)
- Median Survival of 10 years - the **highest mortality rate among the systemic rheumatic diseases**
- Pulmonary involvement is the leading cause of death
- ~170K patients in 7 major markets



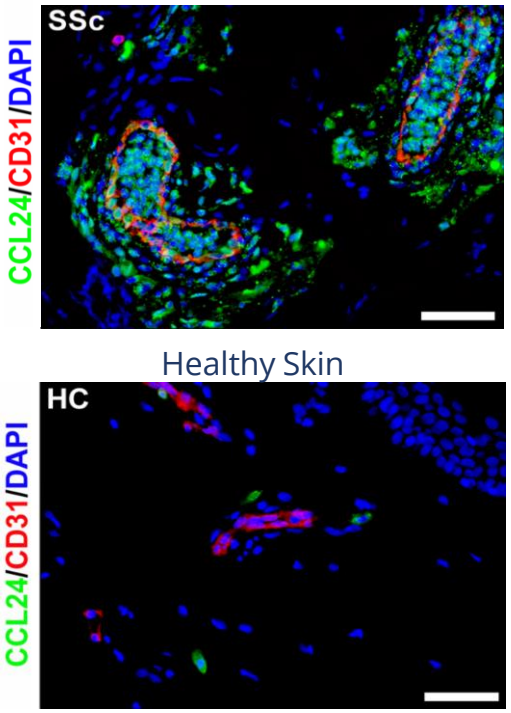
Source: Nature Reviews | Disease Primers

Global data; Bergamasco et al., Clin Epidemiol. 2019; 11: 257–273
7 Major Markets – USA, 5EU (UK, Germany, France, Italy, Spain), Japan

CCL24: A Critical Node Potentiating Systemic Sclerosis (SSc)

Clinical Relevance of CCL24 in SSc Pathophysiology is Supported by Patient Samples

SSc Skin



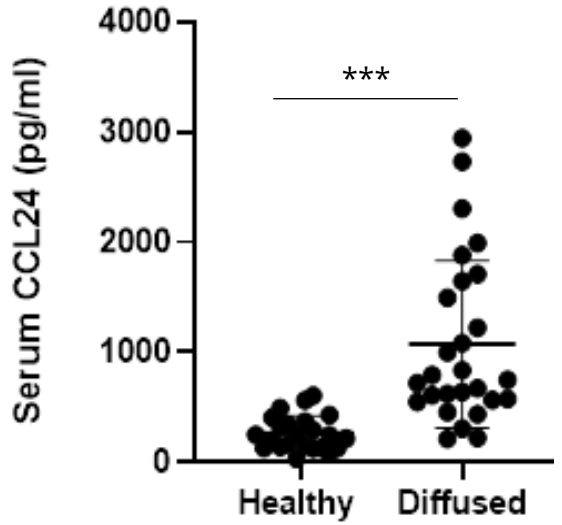
Healthy Skin

Collaboration with University of Florence

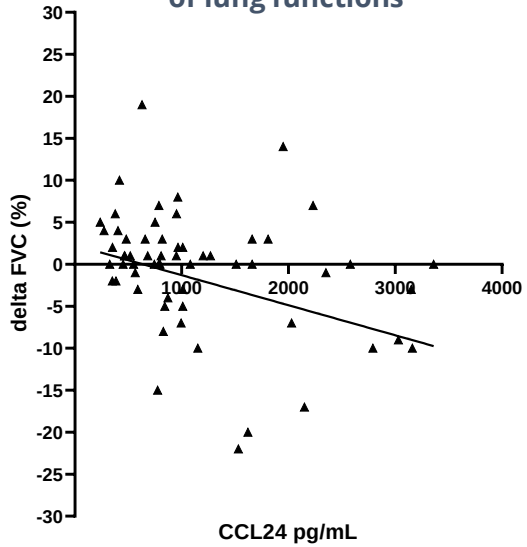
CCL24 overexpressed in skin and serum samples of dSSc Patients compared to healthy

CCL24 correlates with disease progression and fibrotic biomarkers

CCL24 is highly elevated in dSSc population



CCL24 serum level correlates with worsening of lung functions

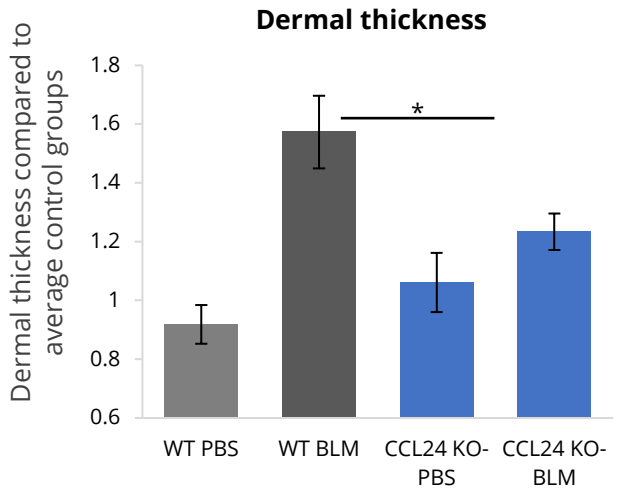


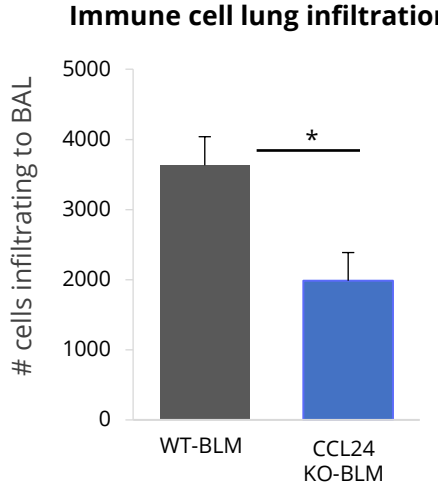
Patient samples support the targeted patient population and clinical trial endpoints

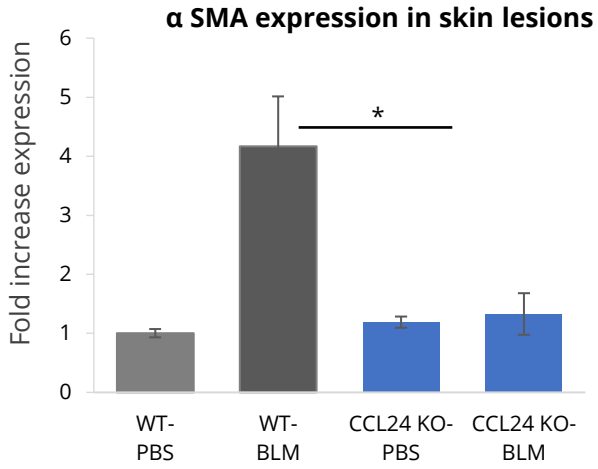
dSSc – diffuse systemic sclerosis
Mor A et al., Annals of Rheumatic Diseases, 2019
***p ≤0.001

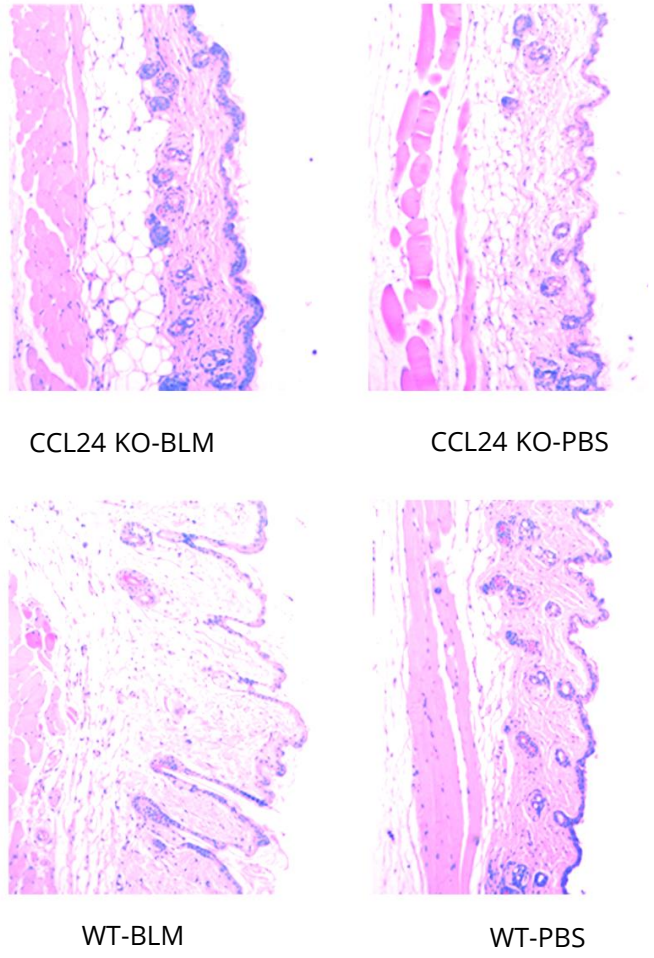
CCL24 Blockade is Singularly Sufficient to Attenuate Fibrosis

Knocking Out the CCL24 Gene Attenuates Inflammation and Fibrosis in SSc Bleomycin Experimental Model









Mor A et al., Annals of Rheumatic Diseases, 2019
BLM – Bleomycin; WT - Wild type; KO – Knockout, BAL- Bronchoalveolar lavage, PBS- Phosphate-buffered saline
*p ≤0.05

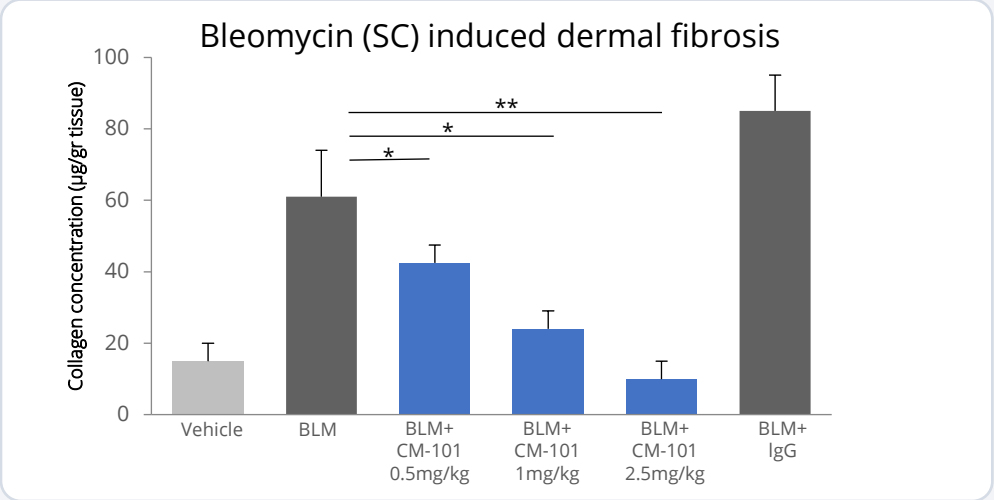
CM-101 Profoundly Reduces Skin and Lung Fibrosis in SSc

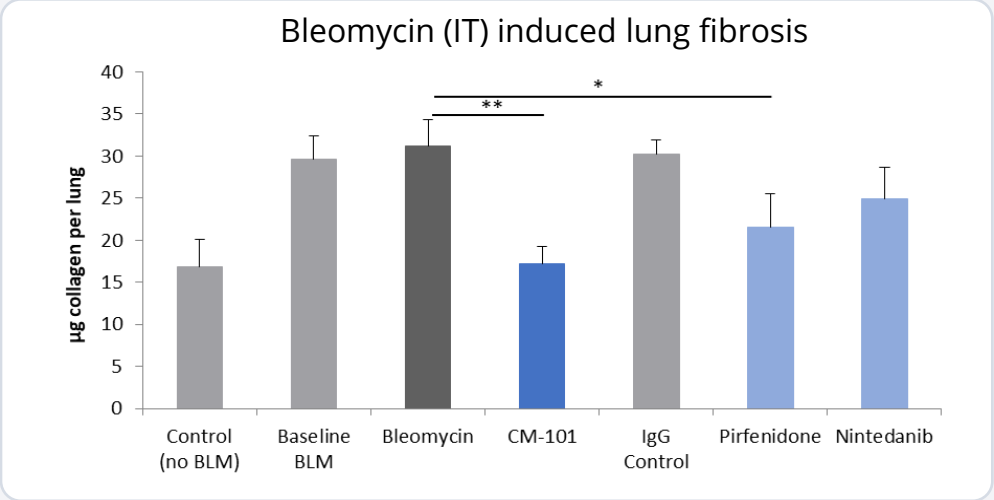
Experimental Models Relevant to Systemic Sclerosis Using Prevention and Therapeutic Designs

Attenuates skin and lung fibrosis levels in bleomycin induced models using treatment mode

Demonstrates a **dose dependent attenuation of fibrosis**

Reduces lung collagen and inflammation as compared to 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 and Key Catalysts

Will be Advancing in Parallel Three Phase II Clinical Programs

	Status	2021	2022	2023
Primary Sclerosing Cholangitis	**Enrolling** Top line data expected in 2H2022	**Phase II: 15wk treatment** *Randomized placebo-controlled; *Europe and Israel study sites*		
Systemic Sclerosis	Initiation expected in 1Q2022		**Phase II: 24-48 wk treatment** Randomized placebo-controlled; *US & Europe study sites*	
Liver Fibrosis Proof of MoA using SC formulation	**Enrolling** Top line data expected in 1H2022	**Phase IIa: 14 wk treatment** Randomized placebo-controlled; *Israel study sites*		

SC – Subcutaneous, MoA- Mechanism of Action

*NCT04595825

Investment Summary

Pioneering Innovative Treatment for Fibrosis-Related Diseases with High Unmet Need

Chemomab

- **Clinical stage company advancing PhII trials in multiple fibrotic indications with high unmet need**
- **Substantial near term value inflection points**
- **Strong leadership with proven track record**

CM-101

- First-in-class mAb blocking CCL24
- Novel and differentiated dual anti-fibrotic and anti-inflammatory MoA
- SC and IV formulations available; well established manufacturing process
- Strong IP protection

Efficacy

- First anti-fibrotic evidence in patients
- Significant anti-fibrotic effects across multiple in vivo, ex vivo and in vitro models

Safety

- Favorable safety and tolerability that support chronic treatment based on toxicology, Phase Ia and Phase Ib clinical trials

PK & Mode of Administration

- Optimal PK for both SC and IV formulations
- Comparable exposure levels and target engagement using both formulations

IV – Intravenous; SC – Subcutaneous
MoA – Mechanism of Action

Thank You

Chemomab
THERAPEUTICS