



Pioneering Innovative Treatments for Fibrotic Diseases

Corporate Overview | Non-Confidential | January 2021

Forward Looking Statements



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Chemomab Highlights

A Clinical Stage Biotech Company



Focus

- Development of innovative therapies for multiple fibrotic diseases

Clinical Differentiation

- CM-101, a first-in-class CCL24 neutralizing mAb with confirmed anti-fibrotic MoA
- Validated CCL24 as critical fibrosis target: clinical findings and experimental models
- Positive Ph1b data including safety, tolerability, PK, PD and biomarker readouts

Near-Term Catalysts

- Initiating staggered phase 2 clinical programs, with first started in 4Q20
- Clinical readouts are expected over the next 12-18 months to drive value inflection

Robust IP Portfolio

- Issued CoM, multiple nationalization stage filings, worldwide patent exclusivity through 2041

Top Tier Investors



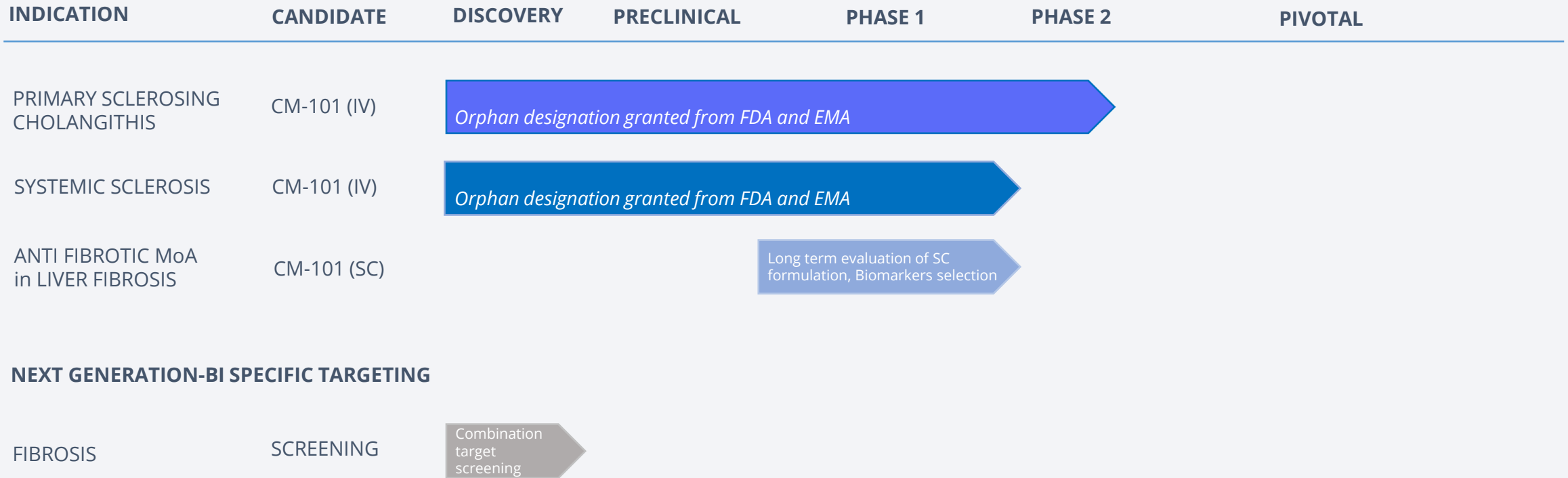
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Chemomab Strategy: Fibrotic Disease Franchise



Addressing Fibrotic Diseases with High Unmet Need



Fibrotic Diseases – Unmet Medical Need

Potentiating High Morbidity and Mortality

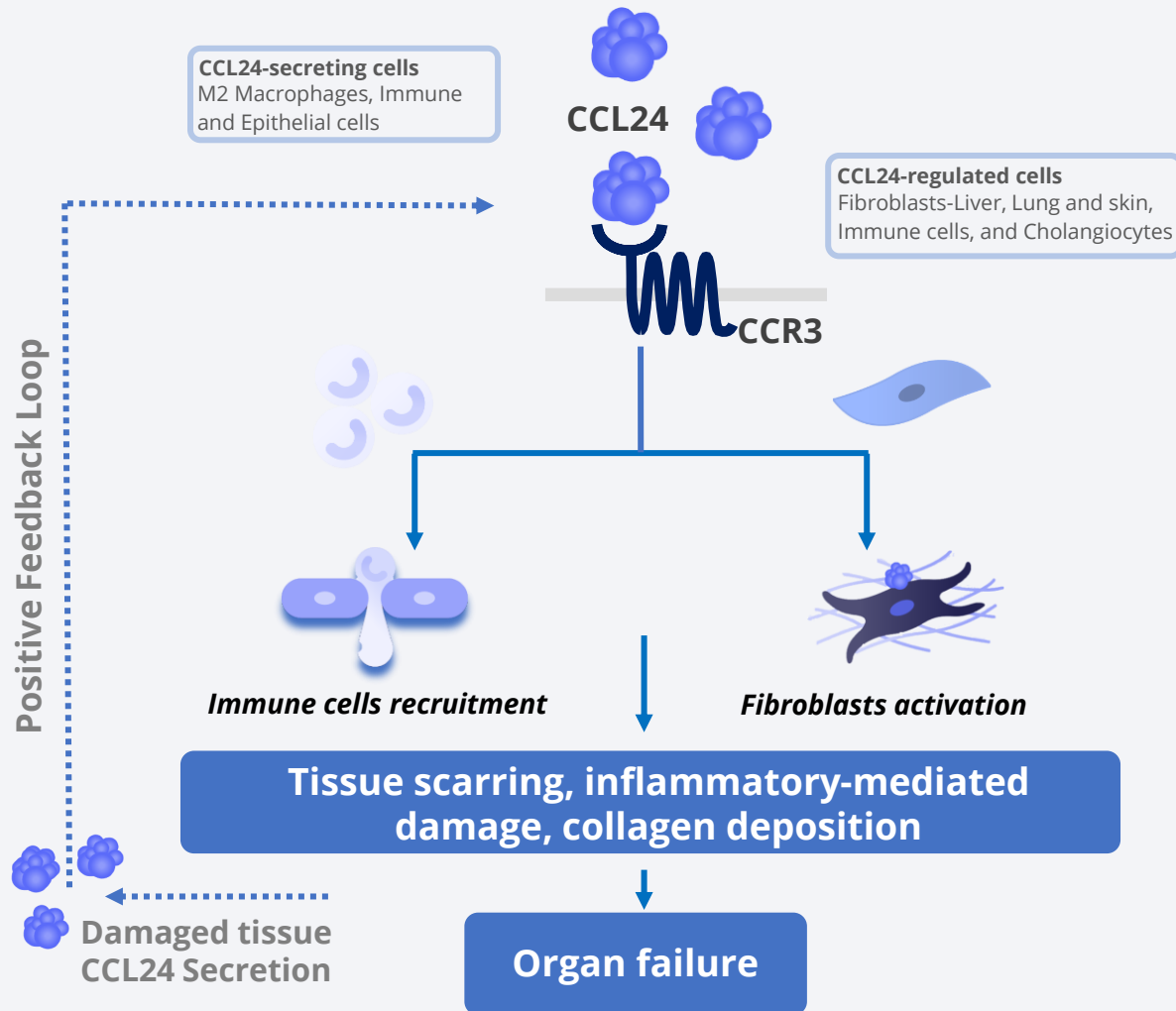


Fibrotic Disease	Orphan Disease	SOC	Sequela	Median Survival	7MM Prevalence	7MM Value
Primary Sclerosing Cholangitis (PSC)	✓	Symptomatic approach, UDCA (EU), Liver Transplant - the only disease modifying option	Bile duct damage, liver fibrosis, cirrhosis, cholangiocarcinoma	10 – 12 yr	70K pts	>1B\$
Systemic Sclerosis (SSc)	✓	Symptomatic approach, Nintedanib approved for ILD. No disease modifying option	Skin fibrosis, vasculopathy multi-organ failure (lung, GI, heart)	10 yr	140K pts	>1B\$

No disease-modifying drugs have been approved for these fibrotic disorders

CCL24 is a Novel Therapeutic Target for Fibrosis

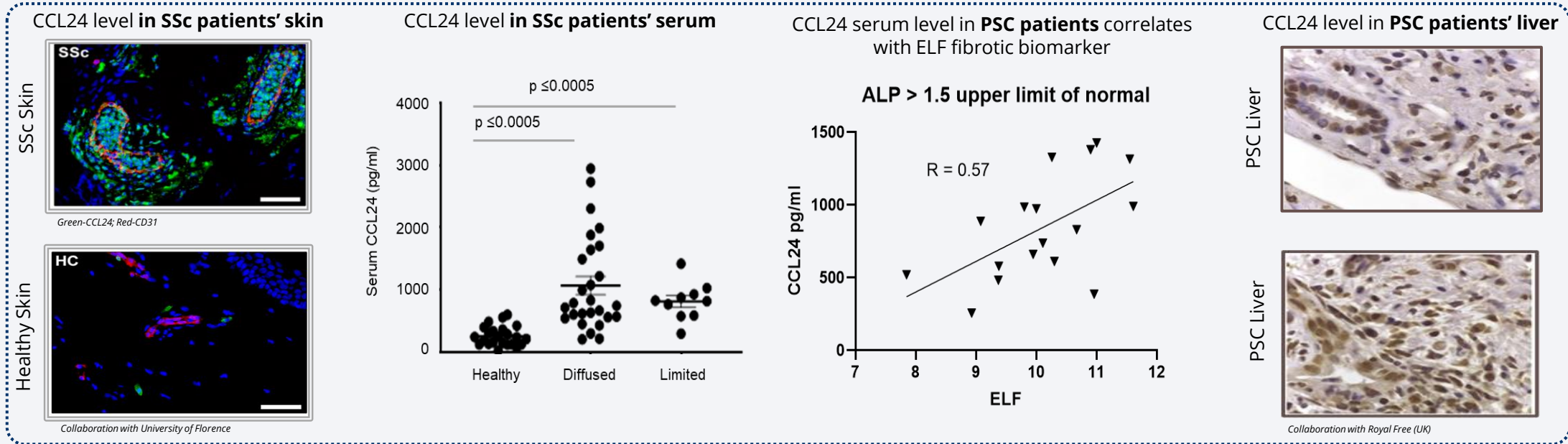
Critical Mediator Promoting Inflammation and Fibrosis



- ✓ **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

CCL24: a Critical Node Potentiating Fibrosis-Related Diseases

Target Validation in PSC and SSc

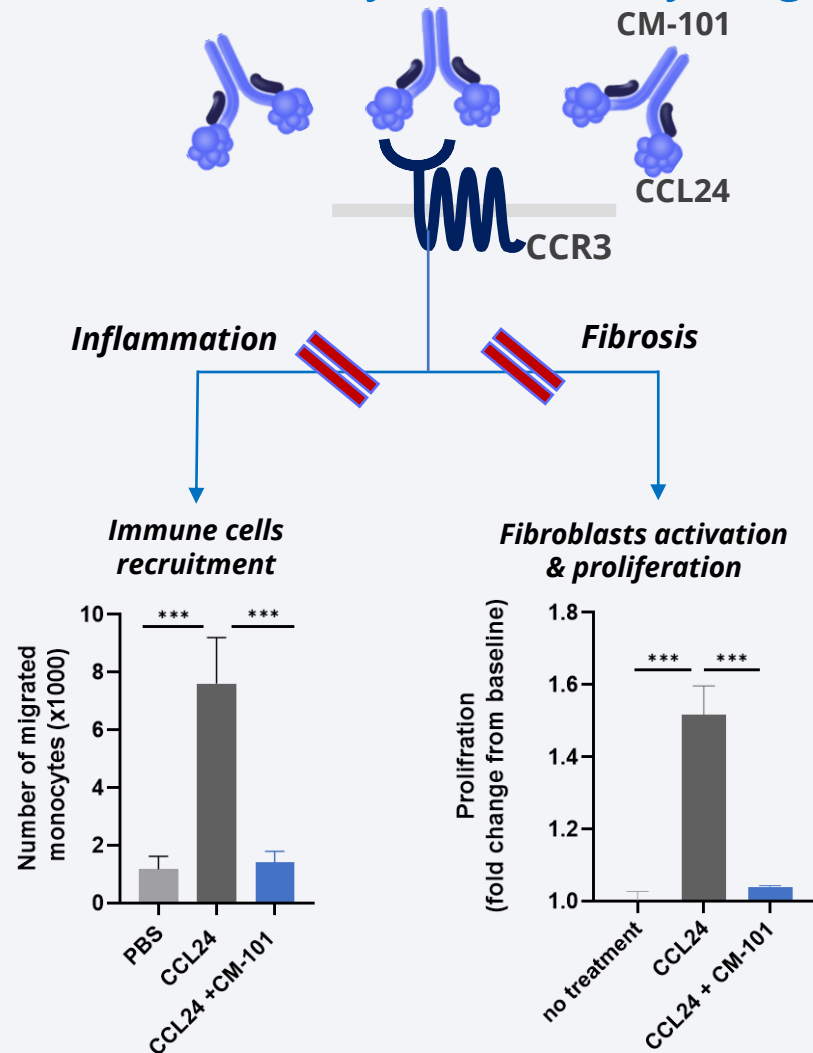


- CCL24 expression are **significantly and selectively elevated** in diseased vs. healthy tissues
- CCL24 levels **correlates with disease progression** and circulating fibrotic biomarkers
- CCL24 functionally **drives critical fibrotic** pathways across several experimental models
- CCL24 blockade using CM-101 demonstrates first evidence of **anti-fibrotic activity in human**

CM-101- A First in Class mAb Blocking CCL24



Dual Mechanism of Action Interfering with the Core Fibrotic Pathways



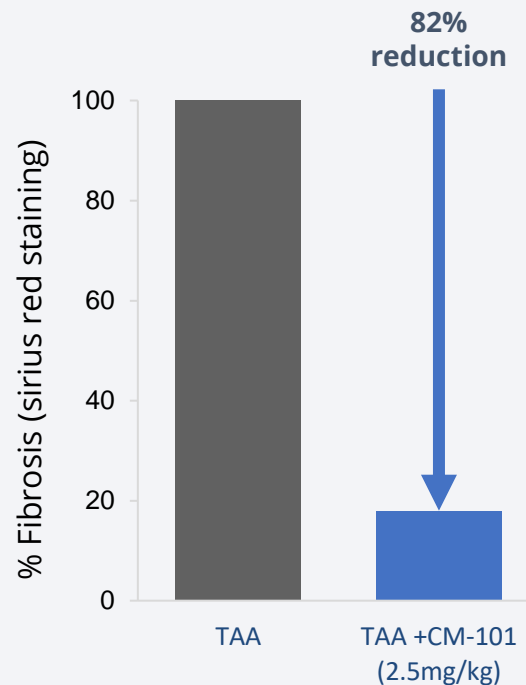
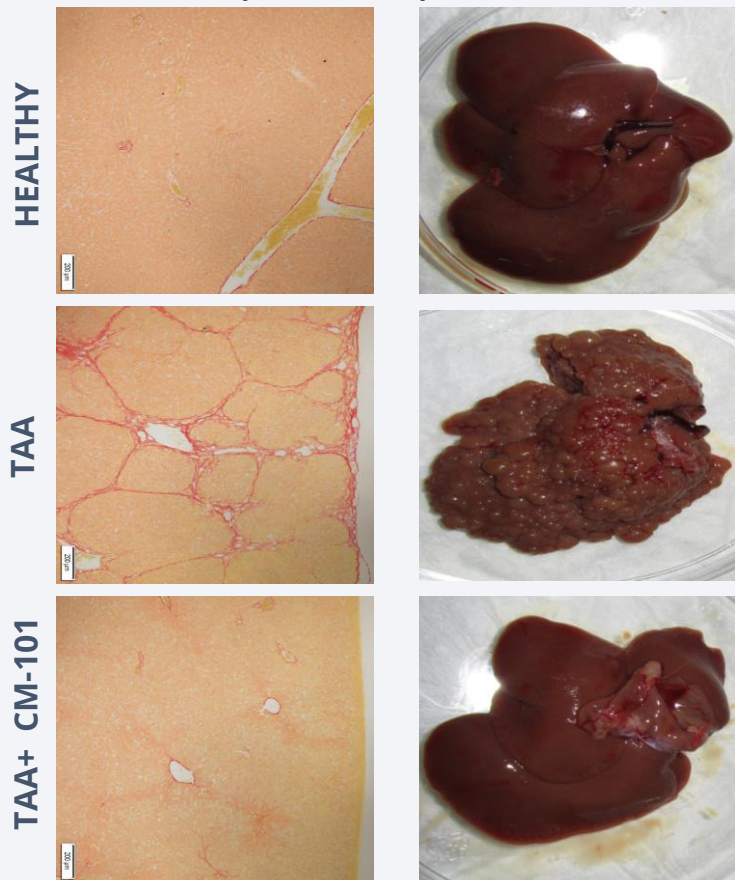
- ✓ Fully humanized antibody, binds and blocks CCL24 with high affinity
- ✓ CM-101's epitope directly interfering with Receptor-Ligand interaction
- ✓ Significant attenuation of fibrosis across several liver, skin and lung fibrosis models
- ✓ Favorable PK profile (IgG1)
- ✓ Two formulations: IV and SC
 - Well defined manufacturing process
- ✓ Strong IP portfolio based on issued CoM patents

CM-101 Reduces Liver Fibrosis by 80%

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



SIRIUS RED (COLLAGEN)



Pro-fibrotic genes

Col1A1
Col3A1
TIMP1
ACTA2
TGF- β

Liver enzymes

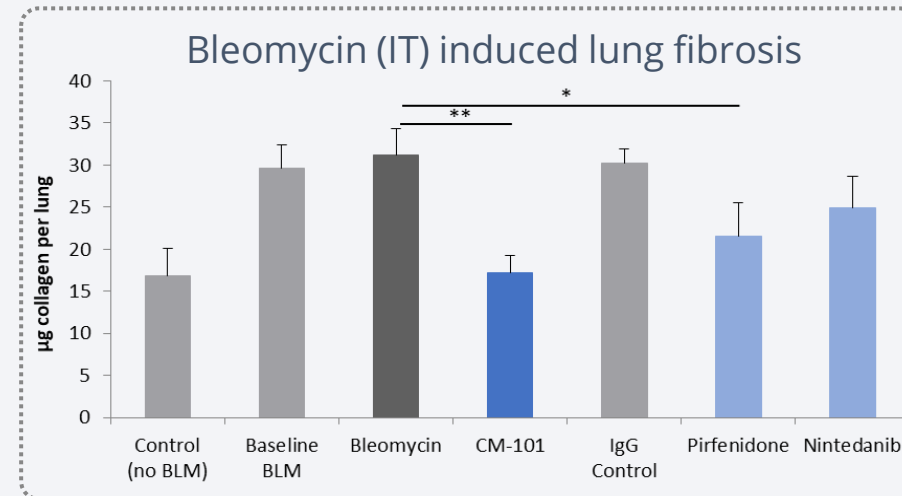
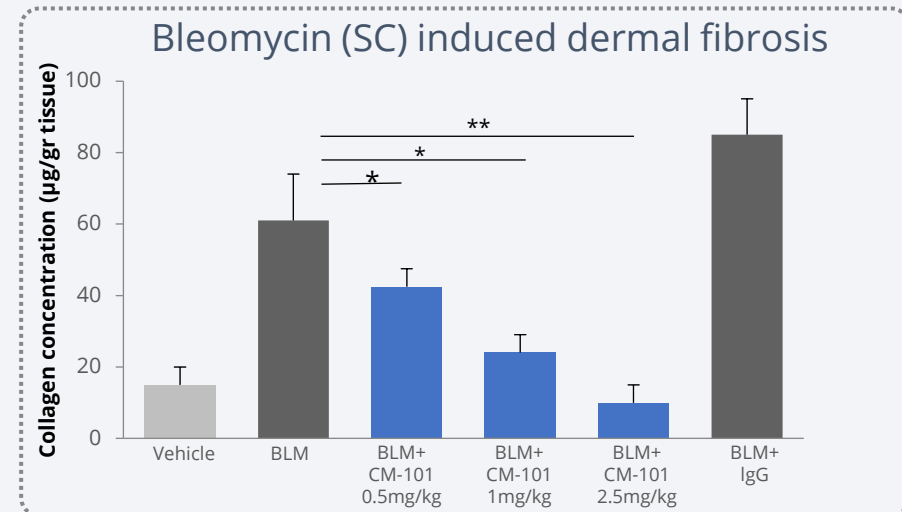
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AST
ALP

CM-101 Profoundly Reduces Skin and Lung Fibrosis in SSc

Skin and Lung Experimental Models Relevant to Systemic Sclerosis Using Therapeutic Design



- **Normalizes skin and lung fibrosis levels** in bleomycin induced models using therapeutic dosing
- Substantially **reduces lung collagen and inflammation as compared to approved drugs** for lung fibrosis



Mor et al, Annals of Rheumatoid Diseases, 2019

CM-101 Holds a Robust Preclinical Package

Significantly Attenuates Fibrosis & Inflammation Across a Wide Range of Models



CCL24 Target Validation

Ex-Vivo (Patient Samples)

PSC

- Biomarkers correlation
- Overexpression of CCL24 and CCR3

Systemic Sclerosis

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

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)

NASH

- CCL24 knock out 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)

Nonalcoholic steatohepatitis

- STAM (mice)
- MCD diet induced NASH (mice)

Atherosclerosis

- ApoE knock out model (mice)

Mechanism of Action

CM-101 effects on fibroblasts 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
- Monocytes recruitment

Toxicology

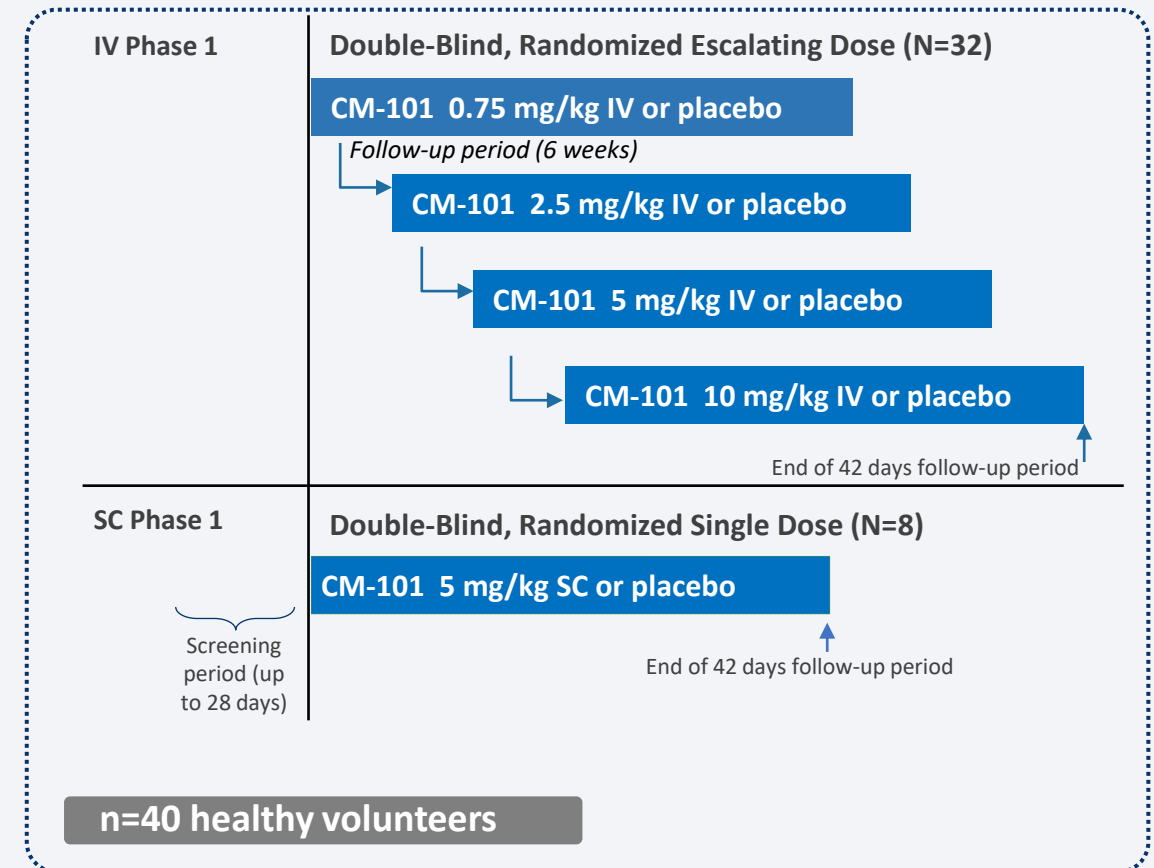
- **Short-term GLP** in rodents
- **Long-term GLP** in Non-human primates
- **Ex-vivo safety:** ADCC, CDC, cytokine secretion
- **Tissue cross reactivity**

CM-101 is Safe & Well Tolerated in Healthy Volunteers

Phase 1a Single Administration study



- CM-101 was **safe and well tolerated** at all tested doses up to 10 mg/kg and for both formulations
- Average $t_{1/2}$ 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

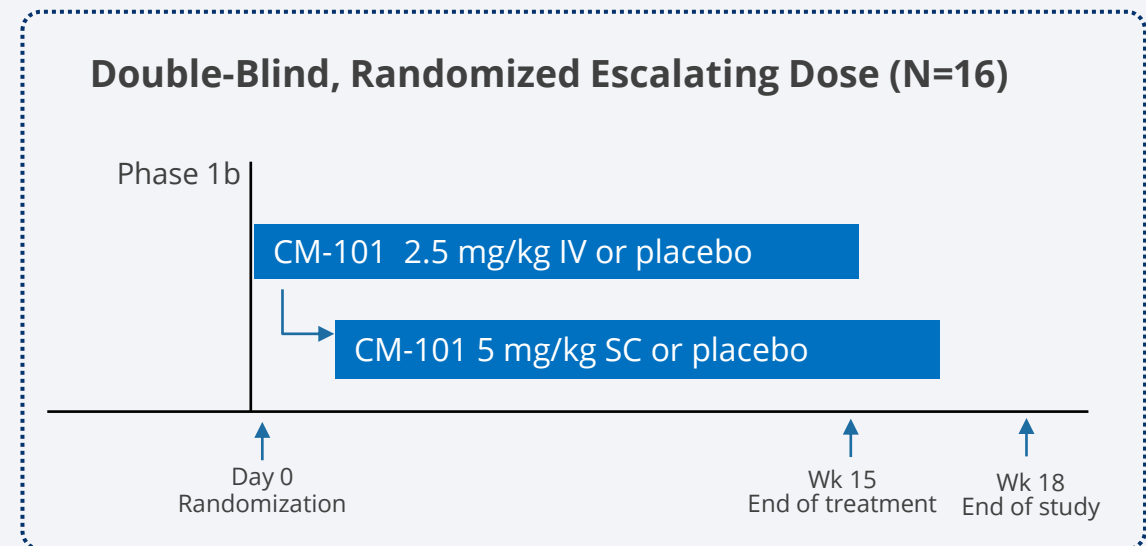


Ph1b Demonstrates Safety & Tolerability Along 15 Weeks Treatment

Phase 1b Multiple Administration Study 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
- Favorable $t_{1/2}$, supports **long dosing interval** (Q2W - Q4W)
- Dose dependent **PK and target engagement**



Study Design

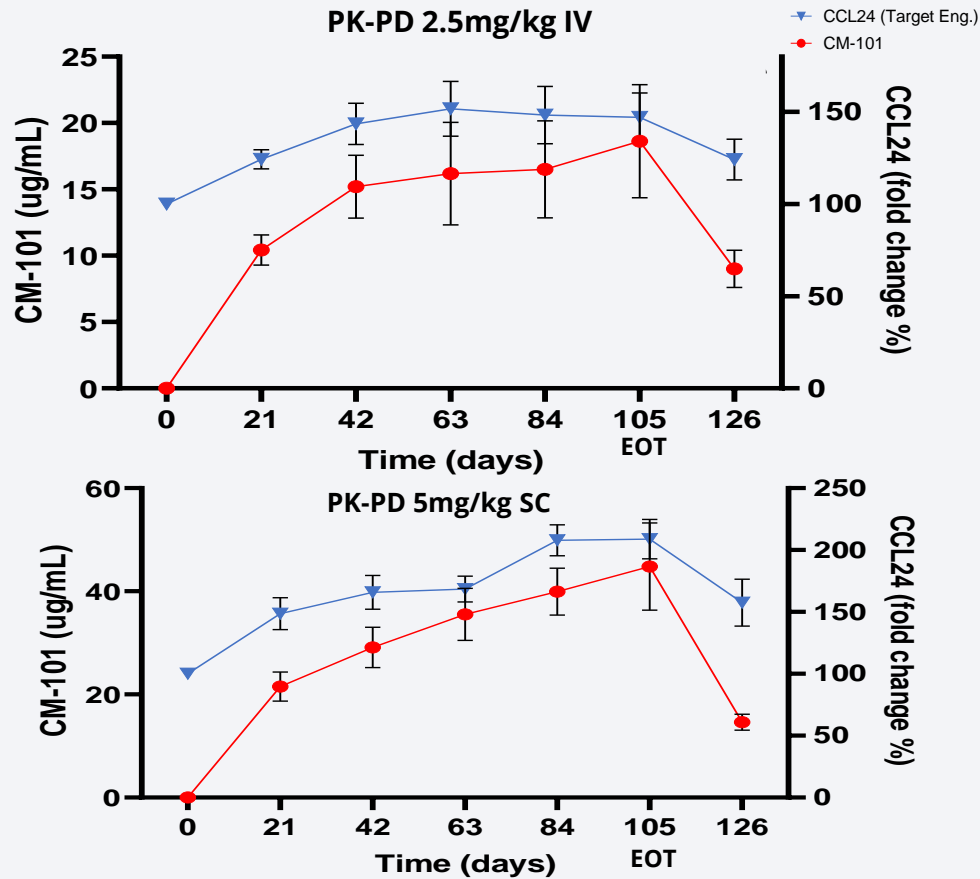
- Tested doses - 2.5 mg/kg IV infusion and 5 mg/kg SC injection
- 5 repeated administrations per patient; Q3W
- Primary endpoint - safety and tolerability

CM-101 Target Engagement & Anti-Fibrotic Mechanism

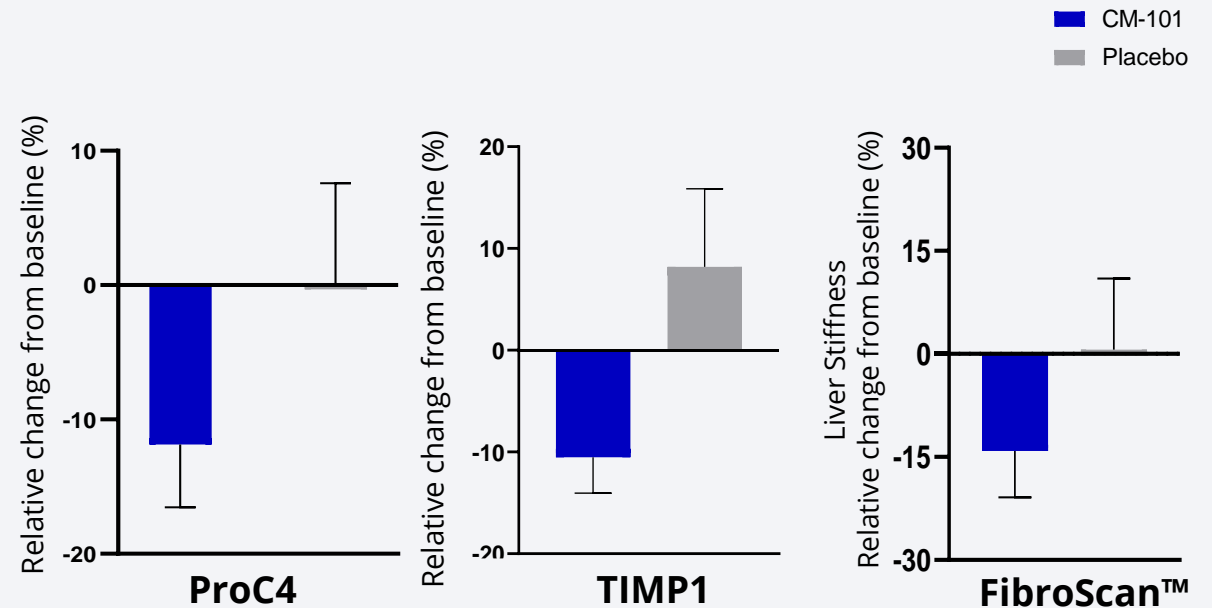


Human Confirmation for CM-101 Anti-Fibrotic Mechanism of Action

PK-PD



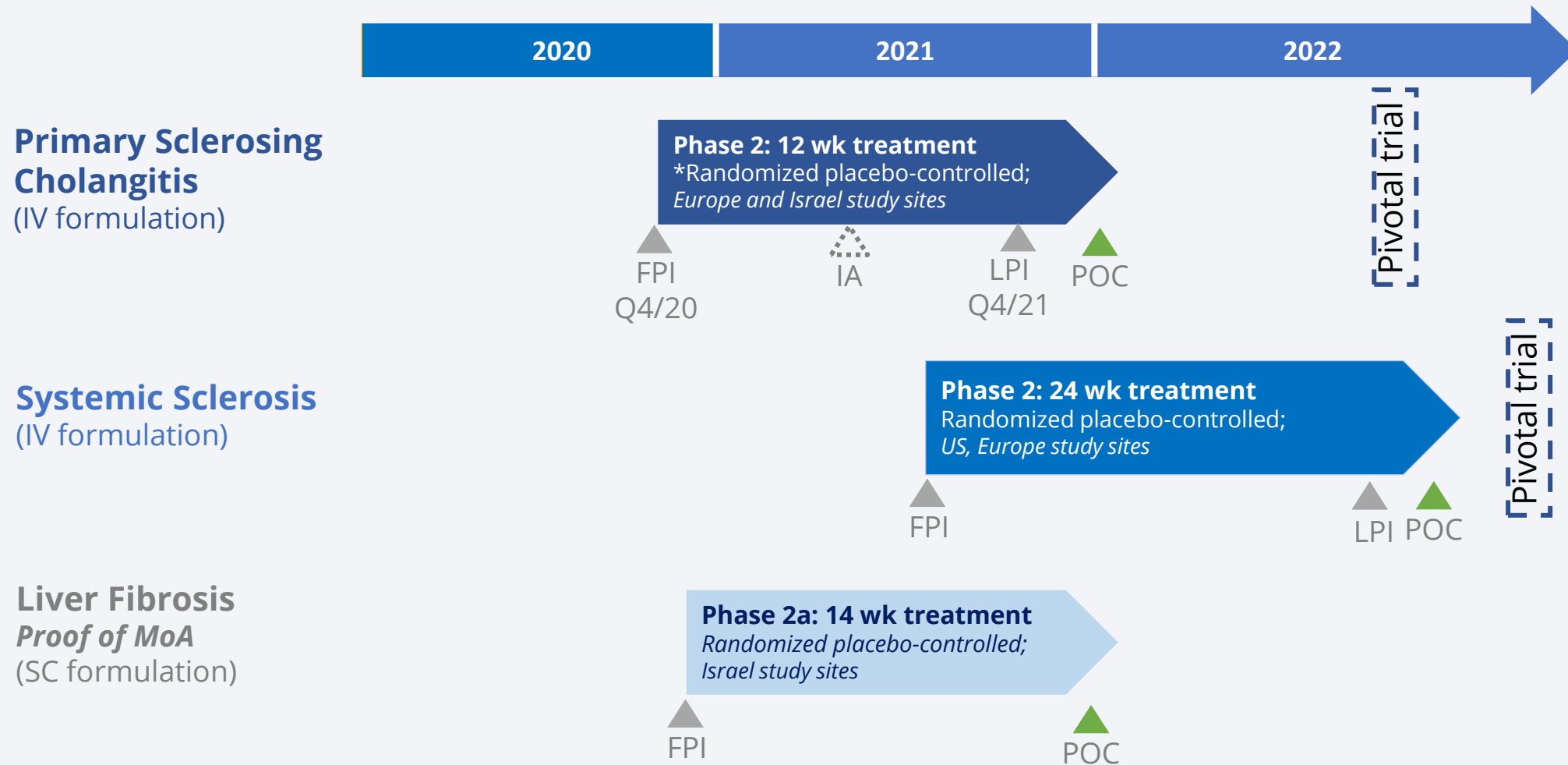
Fibrotic Biomarkers* & Elastography



*Concordant results across 6 relevant fibrotic markers

CM-101 Clinical Development Plan and Key Catalysts

Phase 2 Studies Driving Pivotal Studies



Experienced Leadership



Management



ADI MOR, PhD
Chief Executive Officer, Co-Founder



ARNON AHARON, MD
Chief Medical Officer



SIGAL FATTAL, CPA
Chief Financial Officer



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VP Business Development



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Chemomab, Fighting Fibrosis Across Indications



Opportunity

- **Clinical stage company entering Ph2 trials in multiple fibrotic indications with high unmet need**
- **Substantial value inflection points in 2021-2022**
- **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 Formulation
- 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



Chemomab

THERAPEUTICS