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 Highlights

**A Clinical Stage Biotech Company**

### Focus
- Discovery and development of innovative therapeutics for rare 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
- Advancing three phase 2 clinical programs in parallel
- Clinical readouts are expected during 2022 to drive multiple value inflections

### Robust IP Portfolio
- Issued CoM, multiple nationalization stage filings, worldwide patent exclusivity through 2041

### Top Tier Investors
- [OrbiMed](https://www.orbimed.com/), Cormorant Asset Management, [THIEL](https://www.thielgroup.com/)

### Solid Balance Sheet
- $55 million cash following recent $45 million PIPE, with cash runway through H2 2023 and all key Phase 2 trial read-outs

CoM- Composition of matter
# Experienced Leadership

| Management |
|------------|---|
| **ADI MOR, PhD**  
Chief Executive Officer, Co-Founder |
| **ARNON AHARON, MD**  
Chief Medical Officer |
| **SIGAL FATTAL, CPA**  
Chief Financial Officer |
| **SHARON ELKOBI, MSc, MBA**  
VP Business Development |
| **MICHAL SEGAL-SALTO, PhD**  
VP Research and Development |
| **SHARON HASHMUELI, PhD**  
Head of CMC and Regulatory Affairs |

<table>
<thead>
<tr>
<th>Board of Directors</th>
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| **Stephen Squinto, PhD**  
Chairman of the Board |
| **Adi Mor, PhD**  
Chief Executive Officer & Co-Founder |
| **Alan Moses, MD**  
Director |
| **Claude Nicaise, MD**  
Director |
| **Joel Maryles, CFA, MBA**  
Director |
| **Neil Cohen, MA**  
Director |
| **Nissim Darvish, MD, PhD**  
Director |

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<th>Scientific Advisory Board</th>
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| **Prof. Marco Matucci, Cerinic, MD, PhD**  
Director of the Division of Rheumatology, University of Florence, Italy |
| **Scott L. Friedman, MD**  
The Dean for Therapeutic Discovery and Chief, Division of Liver Diseases, Mount Sinai, NY, USA |
| **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 |
| **Prof. Francesco Del Galdo, MD, PhD**  
Head of the Scleroderma Program at NIHR, University of Leeds, UK |
| **Gideon Hirschfield, MA MB PhD**  
Lily and Terry Horner Chair in Autoimmune Liver Disease, University of Toronto, Toronto General Hospital, Canada |
Chemomab Strategy: Fibrotic Disease Franchise

Addressing Fibrotic Diseases with High Unmet Need

<table>
<thead>
<tr>
<th>PROGRAM</th>
<th>INDICATION</th>
<th>TARGET</th>
<th>DISCOVERY</th>
<th>PRECLINICAL</th>
<th>PHASE 1</th>
<th>PHASE 2</th>
<th>PHASE 3</th>
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<tbody>
<tr>
<td>CM-101 (IV)</td>
<td>PRIMARY SCLEROSING CHOLANGITIS</td>
<td>CCL24</td>
<td>Orphan designation granted from FDA and EMA</td>
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<td>CM-101 (IV)</td>
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</tr>
<tr>
<td>CM-101 (SC)</td>
<td>ANTI FIBROTIC MoA in LIVER FIBROSIS</td>
<td>CCL24</td>
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<tr>
<td>NEXT-GEN</td>
<td>FIBROSIS</td>
<td>SCREENING</td>
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IV- Intravenous; SC- Subcutaneous, 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 attenuates inflammation and fibrosis by inhibiting fibroblast activation and immune cell recruitment
Primary Sclerosing Cholangitis (PSC)

Potentiating High Morbidity and Mortality

- PSC is a rare, chronic **bile duct inflammatory and fibrotic disease** leading to end-stage liver disease and cirrhosis
- 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; +$1B market potential

*Orphan Drug Designation granted for CM-101 in EU and US*
CCL24 Plays a Key Role in PSC Pathology

Target Validation in PSC

CCL24 expression is significantly and selectively elevated in PSC livers

Strong correlation between CCL24 and fibrotic biomarkers

CCL24 level in healthy vs PSC patients' livers tissues

Collaboration with RFH, UK

CCL24 serum level in PSC patients

Chemomab's Internal report
ELF – Enhanced Liver Fibrosis; ALP – Alkaline Phosphatase
CM-101 Reduces Liver Fibrosis by 80%

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

**SIRIUS RED (COLLAGEN)**

<table>
<thead>
<tr>
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<th>HEALTHY</th>
<th>TAA</th>
<th>TAA + CM-101</th>
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<td><img src="taa.png" alt="Image" /></td>
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</table>

**% Fibrosis (sirius red staining)**

- TAA: 100%
- TAA + CM-101: 18%

82% reduction

**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 Reduces Liver Injury and Fibrosis in PSC

Cholestasis, Inflammation and fibrosis are reduced in the MDR2 Knockout Model in Mice

**CM-101 attenuates liver collagen levels and fibrotic biomarkers**

![Graph showing expression levels of TIMP1 and Col1a1 with or without CM-101 treatment.]

**CM-101 reduces cholangiocytes proliferation and monocytes recruitment**

![Images showing Sirius Red staining, macrophages (IBA1), and cholangiocytes (panck).]

CM-101 interferes with the core pathways that drive PSC
Systemic Sclerosis (SSc)  
*Most Lethal Among Systemic Rheumatic Diseases*

- **Systemic Sclerosis (SSc)** is a rare autoimmune rheumatic disease characterized by inflammation and fibrosis of the skin and internal organs.

- 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**

- ~140K patients in 7 major markets; +$2B market potential.

*Orphan Drug Designation granted for CM-101 in EU and US*
CCL24: A Critical Node Potentiating Systemic Sclerosis (SSc)

Target Validation in SSc

CCL24 Levels in Skin Tissues of SSc Patients

- **Significantly overexpressed in skin** of SSc patients vs. healthy

CCL24 Levels in Serum samples of SSc patients

- **Strongly elevated** in diffused SSc serum
- **Correlates with fibrotic biomarkers and disease progression**

Collaboration with University of Florence

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Mor A et al., Annals of Rheumatic Diseases, 2019

***p ≤0.001

BLM - Bleomycin; WT - Wild type; KO - Knock out
CCL24 Blockade is Singularly Sufficient to Attenuate Fibrosis

Knocking out the CCL24 gene attenuates experimental SSc model (bleomycin)

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

---

**Bleomycin (SC) induced dermal fibrosis**

**Bleomycin (IT) induced 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 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

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**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.

**IV Phase 1**

- Double-Blind, Randomized Escalating Dose (N=32)
  - CM-101 0.75 mg/kg IV or placebo
  - CM-101 2.5 mg/kg IV or placebo
  - CM-101 5 mg/kg IV or placebo
  - CM-101 10 mg/kg IV or placebo

**Follow-up period (6 weeks)**

**SC Phase 1**

- Double-Blind, Randomized Single Dose (N=8)
  - CM-101 5 mg/kg SC or placebo

**Screening period (up to 28 days)**

**End of 42 days follow-up period**

**n=40 healthy volunteers**

**IV - Intravenous; SC - Subcutaneous**
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 t₁/₂, 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

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

- CM-101 2.5 mg/kg IV or placebo
- CM-101 5 mg/kg SC or placebo

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

IV - Intravenous; SC – Subcutaneous; Wk - week
CM-101 Target Engagement & Anti-Fibrotic Mechanism

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

PK-PD

Fibrotic Biomarkers*

Elastography

First evidence for an anti fibrotic effect in human supported by dose dependent PK-PD

*Concordant results across 6 relevant fibrotic markers

Nordic Biosciences, Denmark

ProC4-Procollagen 4
TIMP1- metallopeptidase inhibitor 1
EOT-End of Treatment
## CM-101 Clinical Development Plan and Key Catalysts

### Phase 2 Studies Driving Pivotal Studies

<table>
<thead>
<tr>
<th>Condition</th>
<th>Formulation</th>
<th>2020</th>
<th>2021</th>
<th>2022</th>
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<tbody>
<tr>
<td><strong>Primary Sclerosing Cholangitis</strong></td>
<td>(IV)</td>
<td>FPI</td>
<td>IA LPI POC</td>
<td>POC FPI</td>
</tr>
<tr>
<td><strong>Systemic Sclerosis</strong></td>
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<td><strong>Liver Fibrosis</strong></td>
<td>(SC)</td>
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<td><strong>Proof of MoA</strong></td>
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**Phase 2: 12 wk treatment**
- Randomized placebo-controlled;
- Europe and Israel study sites

**Phase 2: 48 wk treatment**
- Randomized placebo-controlled;
- US, Europe study sites

**Phase 2a: 14 wk treatment**
- Randomized placebo-controlled;
- Israel study sites

FPI - First patient in, LPI - Last patient in, IA - Interim assessment, POC - Proof of Concept, IV - Intravenous, SC - Subcutaneous, MoA - Mechanism of Action

* NCT04595825
Chemomab, Fighting Fibrosis Across Indications

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

**Chemomab**
- Clinical stage company entering Ph2 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 Formulation; Contract manufactured via reputable & established CMO
- 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
Thank You