Edesa Biotech™

**Dermatology Drug Development Summit**

November 1, 2023

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# Clinical-Stage Assets with Major Market Potential

## Targeting Unmet Inflammatory/Immunology Disease

### Multiple Late Stage, Validated Therapies

Inflammation + Immunology focus

Potential first-in-class therapies

Addressing unmet, large market opportunities

### Several Near-Term Catalysts & Milestones

Awardee of C$23M funding from Canada's Strategic Innovation Fund

Positive topline data

Upcoming completion of a late-stage trial and regulatory filings

### Leadership with Established Track Record

Entrepreneur CEO with multiple successful start-ups and exits

Multiple acquisitions of first-in-class compounds with human PoC

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# Technology Acquisition and Targeting

## Identify overlooked clinical assets with promising growth potential

### Well Understood Pathway

- CXCL10 pathway in vitiligo identified by KOLs
- Characterization of TLR4 signaling pathway won Nobel Prize in 2011
- Arachidonic acid pathway

### Unique Targets on Pathway

- CXCL10 dual roles in disease initiation and progression
- Key modulator of immune signaling
- Top of the inflammation cascade

### Novel Method of Action

- Dual MOA that disrupts free and bound CXCL10 activity
- Monoclonal antibody that broadly blocks signaling cascade
- Generates intellectual property

### Data-Driven Indication Priority

- Favorable safety data established in humans
- Strong scientific rationale
- Synergistic with currently available products or treatments

# First-in-Class Development Pipeline

## Significant Add-On Opportunities

| Technology | Drug Candidate | Disease Indication | Pre-Clinical | Phase 1 | Phase 2 | Phase 3 | Status | Comments/Milestones |
|---|---|---|---|---|---|---|---|---|
| Paridiprubart (mAb) Anti-TLR4 | **EB05** | ARDS - Covid-19 | | | | Lead Asset | Fast Track Designation | Recruiting; C$23 million funding from federal Strategic Innovation Fund |
| Paridiprubart (mAb) Anti-TLR4 | **EB07** | Systemic Sclerosis | | | | | Prov. Patent Filed | Preparing Investigational New Drug (IND) application |
| Daniluromer sPLA2 Inhibitor | **EB01** | Allergic Contact Dermatitis (ACD) | | | | Lead Asset | Positive topline data | Ex-N.A. licensing opportunities being explored |
| Daniluromer sPLA2 Inhibitor | **EB02** | Hemorrhoids Disease | | | | | CTA Approved | Proof of Concept study pending |
| Monoclonal Antibody Anti-CXCL10 | **EB06** | Vitiligo | | | | | CTA Approved | Partnering and out-licensing opportunities being explored |

ARDS: Acute Respiratory Distress Syndrome

# EB06

## First-in-Class Anti-CXCL10 mAb

Lead Indication:  Vitiligo

Status: CTA Approved

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# EB06

## First-in-Class Monoclonal Antibody for Treatment of Moderate to Severe Non-Segmental (Generalized) Vitiligo

**Product Profile**


**Anti-CXCL10 mAb**

Biweekly IV Infusion Anticipated

Targets Progression and Maintenance of Depigmentation

Establish efficacy and subsequently develop subcutaneous formulation

**Unmet Need**

**Lack of effective drug treatments**

Autoimmune disease with significant impact on quality of life

Lack of effective treatments, no systemic therapies

**Target Market**

**Vitiligo Affects 0.5 to 2% of the Global Population**

Lifelong, chronic condition, 50% onset by age 20

Affects all skin types

**Validation**

**Phase 2-Ready Clinical Asset**

Clinical Trial Application approved

Pharmacodynamic data has established biological activity

Favorable safety profile in over 60 patients

**CXCL10 Pathway of High Interest Among Vitiligo Researchers**

CXCL10 highly expressed in vitiligo patients

Neutralization prevented and reversed pigmentation in mice models

# Vitiligo

## A Life-Altering Autoimmune Disease

**IFN-γ dependent production of CXCL10 appears to drive vitiligo pathogenesis via dual mechanisms of action**

**1. Anti melanocyte T-cell trafficking**

- CXCL10 and CXCR3 play a key role in the trafficking of anti-melanocytic T-cells to the epidermis
- In a mouse model of vitiligo, administration of an anti-CXCL10 antibody was able to both prevent and reverse depigmentation

**2. Melanocyte apoptosis**

- CXCL10 induces apoptosis of melanocytes via activation of CXCR3B in vitiligo

**Therapies for Atopic Derm (Th2) or Psoriasis (Th17) are Largely Ineffective or Can Make Symptoms Worse**
Targeted Immunotherapies are Needed

### CXCL10/CXCR3 Play a Key Role in the Pathogenesis of Vitiligo

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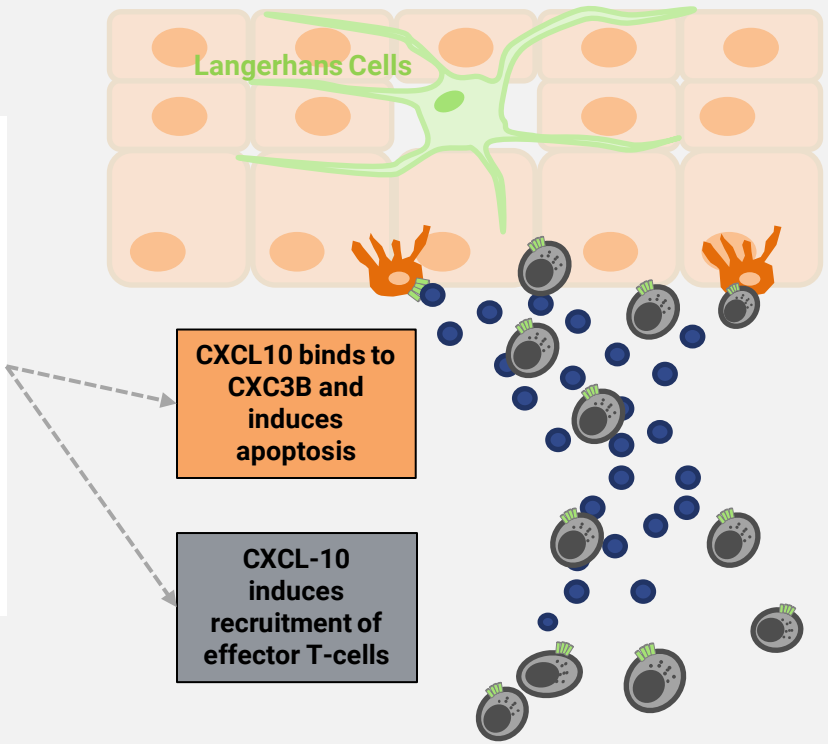
Edesa's EB06 is **Phase 2 asset mAb** that blocks CXCL10, preventing apoptosis and recruitment of effector T-cells.



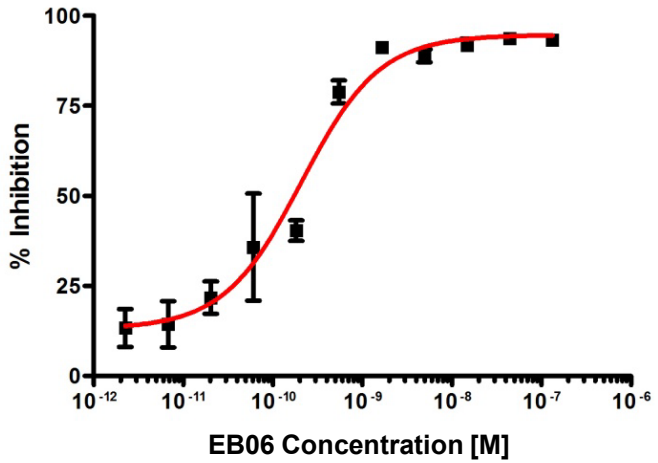

Melanocyte  CD8+ T Cells  CXCL10  CXCR3

# EB06 Monoclonal Antibody Candidate

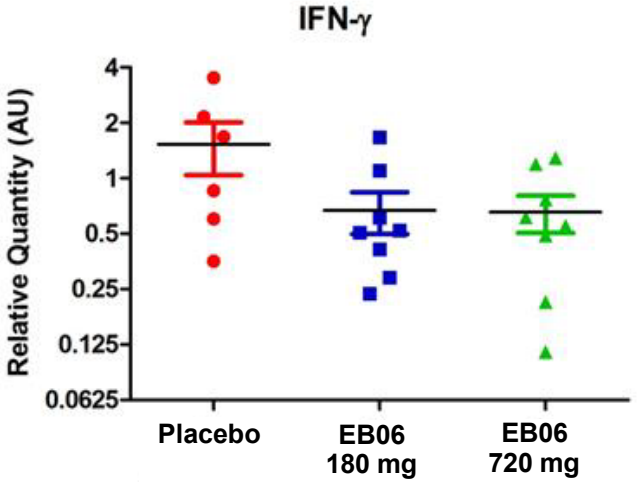
## A First-in-Class, Anti-CXCL10 mAb for Treatment of Vitiligo

### EB06 Blocks CXCL10-induced Cells Chemotaxis

Treatment with EB06 inhibits migration of cells in vitro




### Activity on Targeted Pathway




EB06 treatment reduced expression of pro-inflammatory markers, including IFN-y, in the skin

**Targeted Mechanism of Action**
Binds free and bound CXCL10

**65 Subjects**
No Significant AEs

**Biological Activity**
Demonstrated

**Phase 2 Ready**
CTA Approved

# Regulatory

## Steps to Create Regulatory Filing

- ✓ In vitro characterization demonstrated the requisite binding and inhibition of target
- ✓ Complete preclinical toxicology package
- ✓ Previous clinical studies establishing safety as well as generating PK/PD data enabling dose determination for vitiligo
- ✓ Established CMC package with prior GMP production
- ✓ Strong rationale for target validated by multiple independent groups

## Approved Phase 2 Protocol Design

| | |
|---|---|
| **Status** | Clinical Trial Application Approved |
| **Subjects** | Approximately 120 Adult Subjects<br>Up to 25 Study Centers |
| **Administration** | EB06 or placebo will be administered via IV during the treatment period, followed by a follow-up period. |
| **Primary Endpoint** | Improvement from baseline on the Face Vitiligo Area Scoring Index (F-VASI) |

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