Edesa Biotech®

Vitiligo Program Overview

7th Dermatology Drug Development Europe Summit

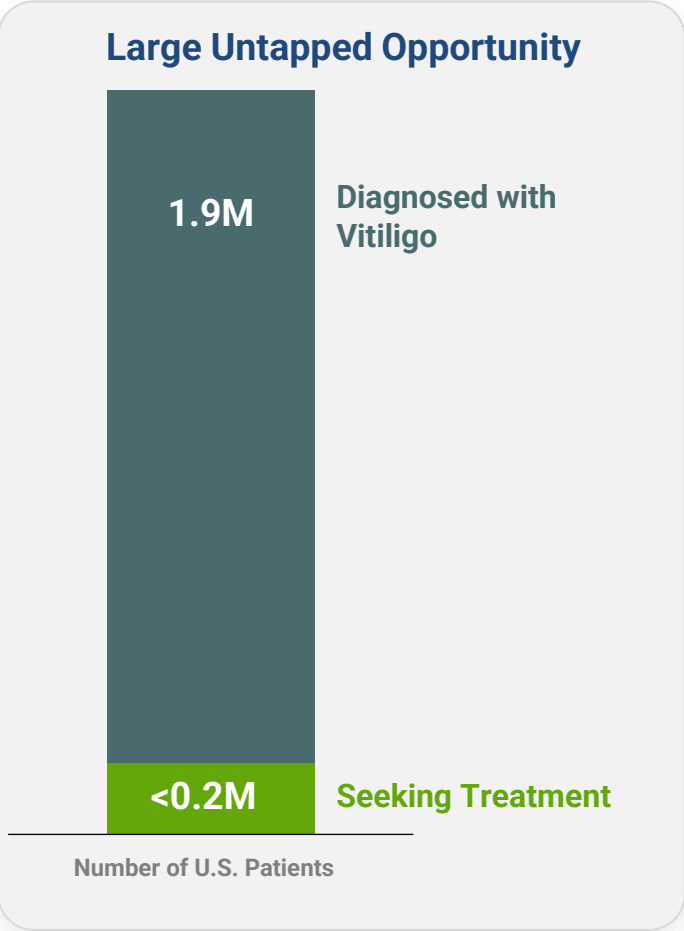
EB06 - Vitiligo

First-in-Class Anti-CXCL10 mAb

Edesa Biotech®

A Significant Unaddressed Market

Latent Market Comprised of Patients Waiting for Better Treatment Options



Large population but low proportion of patients seeking treatment due to **lack of effective and safe treatments**

New therapies likely to drive market growth

- Opzelura is the only approved product.
- Incyte reported that Opzelura **generated $678M***

Need for new options underscored by recent M&A activity

Teva entered into a strategic funding agreement with Royalty Pharma

Teva is developing an anti-IL-15 monoclonal antibody for the treatment of vitiligo

Edesa Biotech

Sources: National Institute of Health and public reports
*Opzelura is marketed for the treatment of nonsegmental vitiligo and mild to moderate atopic dermatitis.

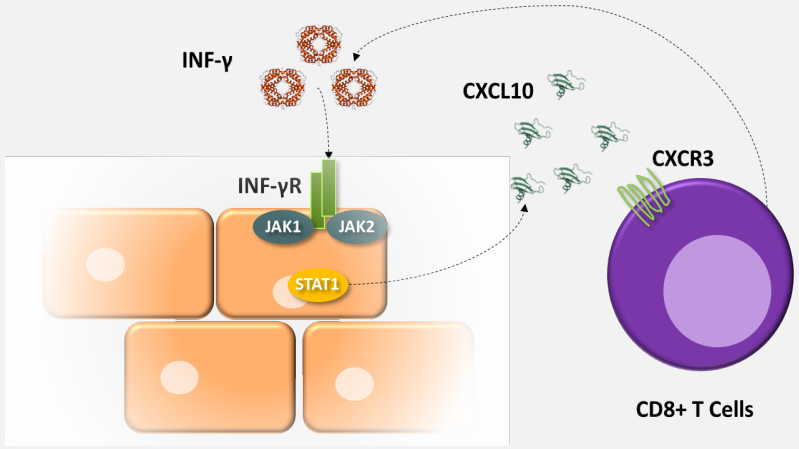
Vitiligo

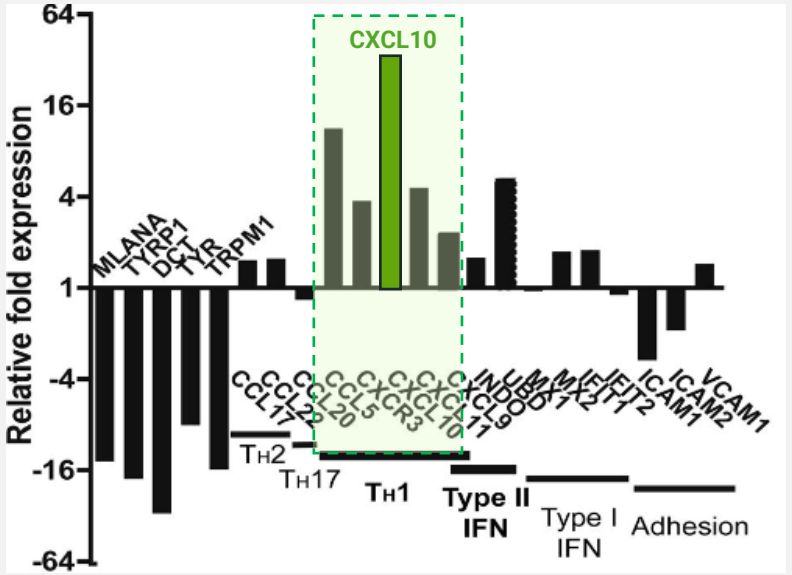
A Life-Altering Autoimmune Disease

- **High Prevalence – 0.5 to 2% Global Population**
  50% Onset Before Age 20; Must be Managed for Decades
  Associated with Type 1 Diabetes and Lupus, among others
- **Severe Quality of Life Impacts**
  Same or Worse than Atopic Dermatitis/Psoriasis
- **Interferon IFNγ-CXCL10-CXCR3 Chemokine Axis**
  CXCL10 is an IFN-γ induced chemokine and is elevated in serum of patients with vitiligo

  Its receptor CXCR3, is upregulated on autoreactive T cells in the blood and skin of patients with vitiligo
- **Therapies for Atopic Derm (Th2) or Psoriasis (Th17) are Largely Ineffective or Can Make Symptoms Worse**
  No Systemic Drugs Approved by FDA to Repigment Skin
  Topical and Phototherapies Limited Effectiveness
  Targeted Immunotherapies are Needed

IFNγ-CXCL10-CXCR3 Chemokine Axis Play a Key Role in the Pathogenesis of Vitiligo





Rashighi M et al. Sci Transl Med. 2014 Feb 12;6(223):223ra23

Vitiligo Treatment Paradigm

Limited Options with Topical Ruxolitinib as the Only Approved Product

TREATMENT

Topicals

Corticosteroids

Calcineurin inhibitors

Ruxolitinib

Phototherapy

Systemic Steroids

Surgery

Skin grafting

Hair follicle transplant

Significant Unmet Need

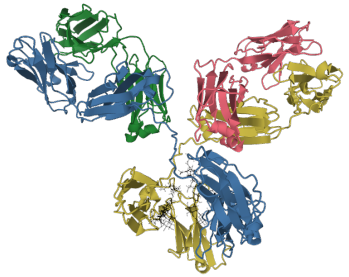
Large unaddressed market due to lack of approved and effective options

Only one approved drug with safety concerns (black box warnings)

Need for safe and effective systemic options, especially for high body surface area

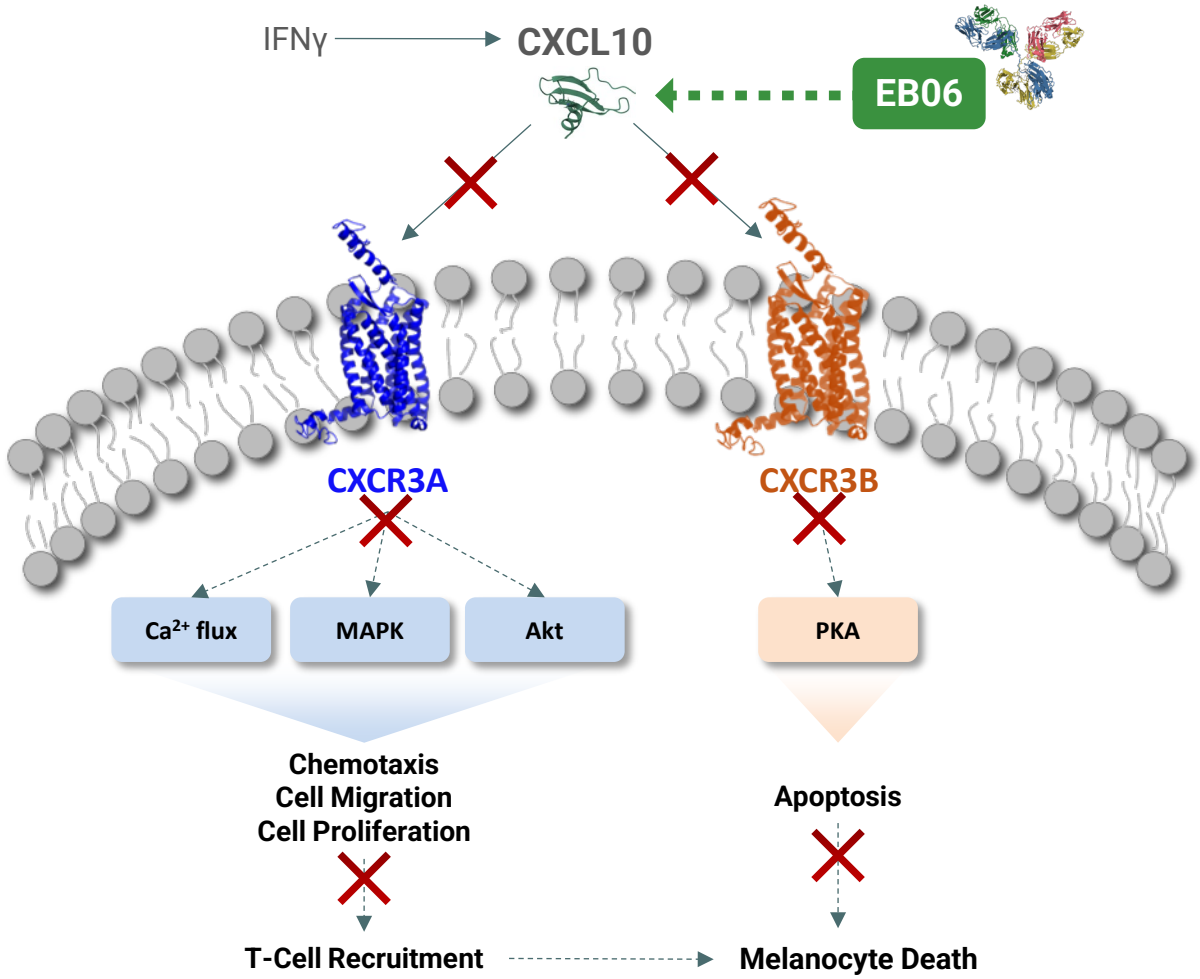
EB06 – Targeting the Chemokine CXCL10

Monoclonal Antibody that Directly Binds CXCL10 with High Affinity and Blocks it from Binding to CXCR3



Drug Profile

- A fully human IgG1k monoclonal antibody
- Binds specifically to CXCL10 with high affinity
- Sequesters and renders CXCL10 inactive
- Multiple manuf. runs by a leading CDMO; IV formulation; future subcutaneous formulation being investigated



EB06 Positioning – Target Product Profile

Addressing Unmet Needs in Vitiligo

	Topical JAK Inhibitors (e.g. Ruxolitinib)	**Oral JAK Inhibitors** (e.g. ritlecitinib, povorcitinib)	**Biologics** (e.g. EB06, auremolimab)
Treats lesional and non-lesional skin	✗	✓	✓
Viable for patients with >10% BSA	✗	✓	✓
No Expected Safety Precaution (Black Box)	✗	✗	✓
No Daily Dosing required	✗	✗	✓

EB06 - Vitiligo

Preclinical Evidence

Edesa Biotech™

Vitiligo Progression - IFNγ-CXCL10-CXCR3 Chemokine Axis

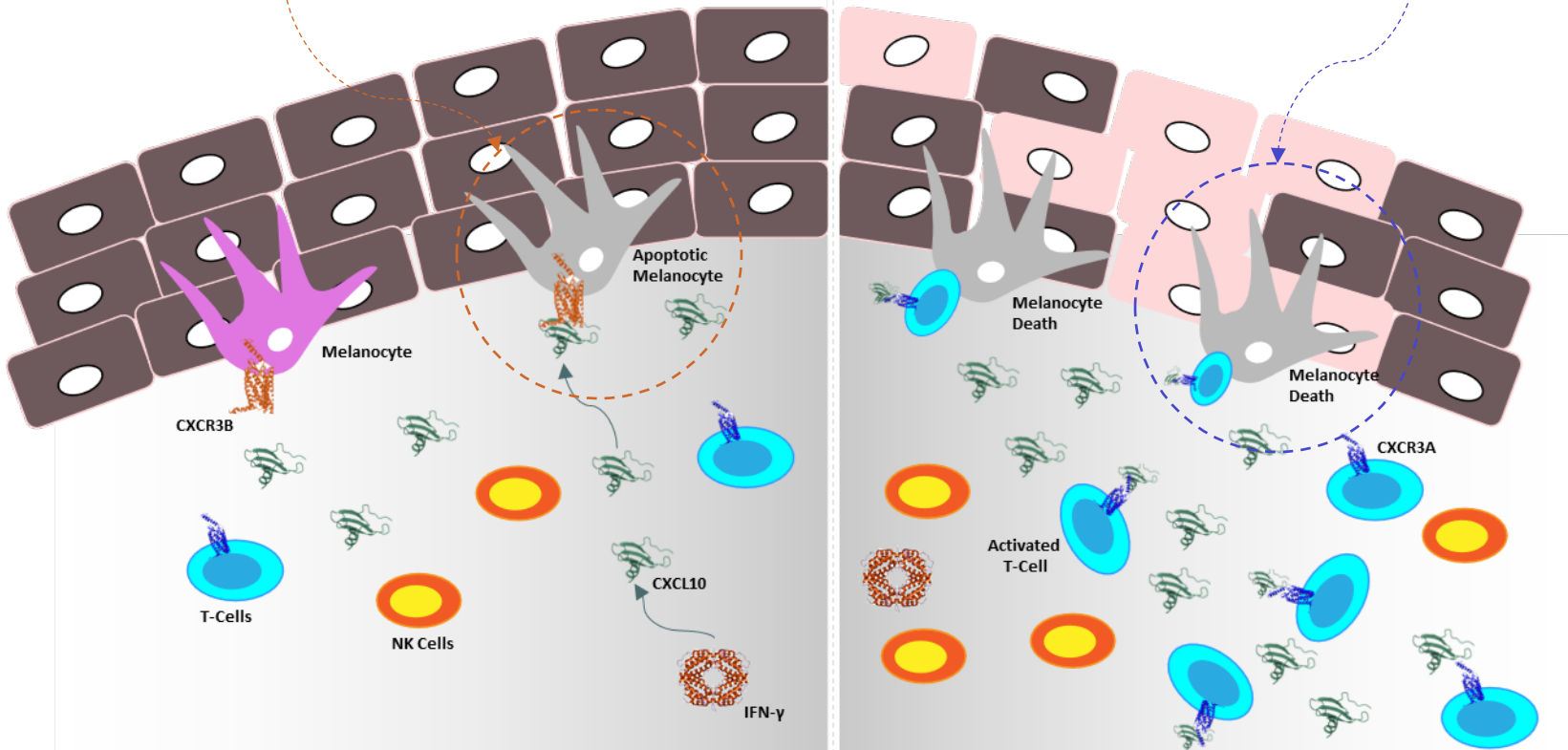
CXCL10 is Crucial in the Multiple Stages of Vitiligo Disease Progression

Non-Lesional (Innate)

- Increased NK cells and CXC3B expression
- INF-γ induced production of CXCL10
- **CXCL10/CXCR3-B mediated melanocyte apoptosis** and antigen presentation

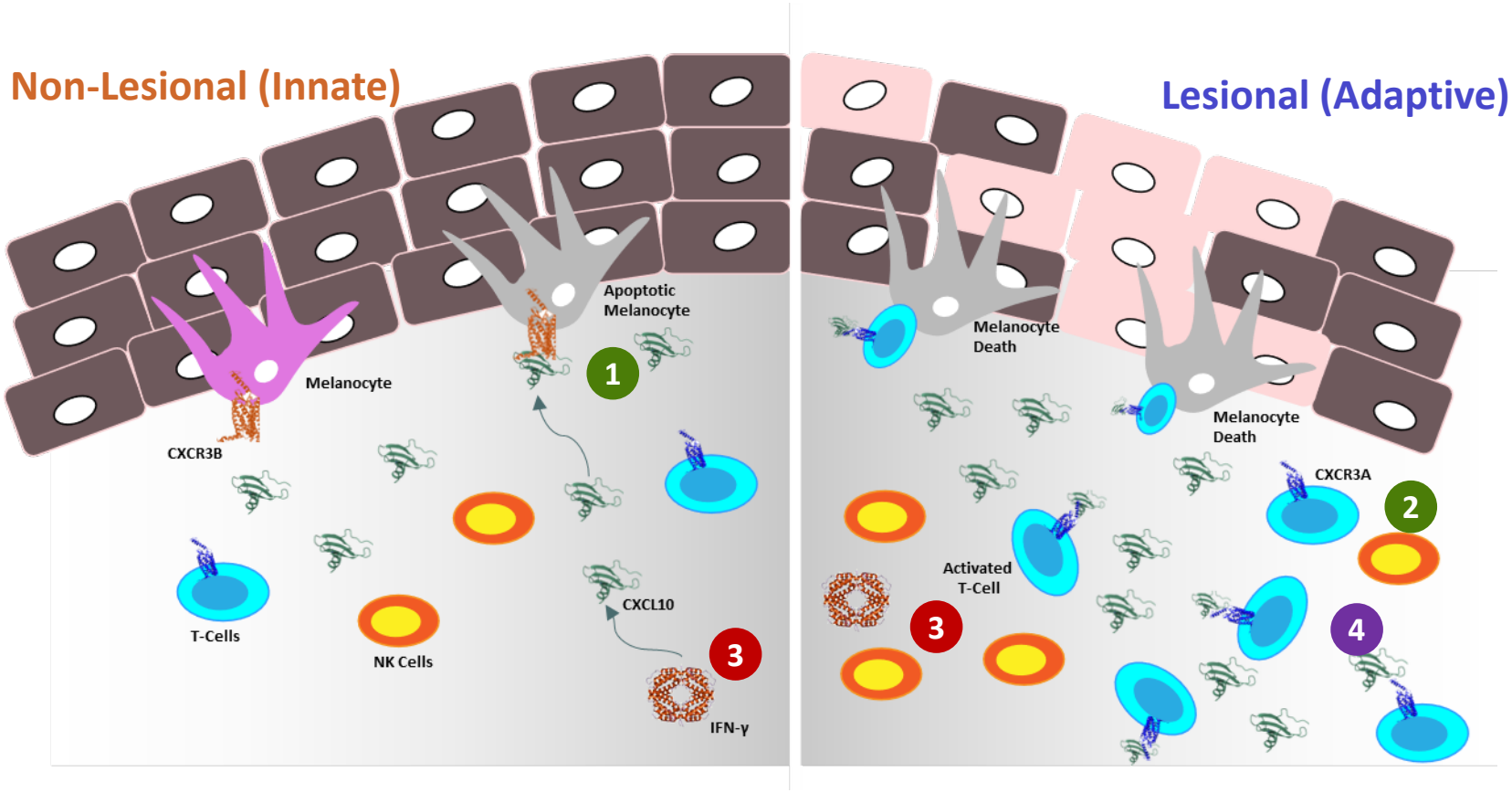
Lesional (Adaptive)

- **CXCL10 creates chemotactic gradient and recruits melanocyte-specific T-Cells through CXCL10-CXCR3A**
- CD8+ T-cell activation results in melanocyte death
- T-cells/chemokines levels prevent re-pigmentation



Targeting the IFNγ-CXCL10-CXCR3 Chemokine Axis

EB06 is an anti-CXCL10 Monoclonal Antibody that Can Act on Different Stages of Vitiligo



EB06 inhibits:

1. CXCL10/CXC3B-mediated melanocyte apoptosis and antigen presentation
2. CXCL10/CXCR3A-mediated trafficking of anti-melanocytic CD8+ T cells to the epidermis

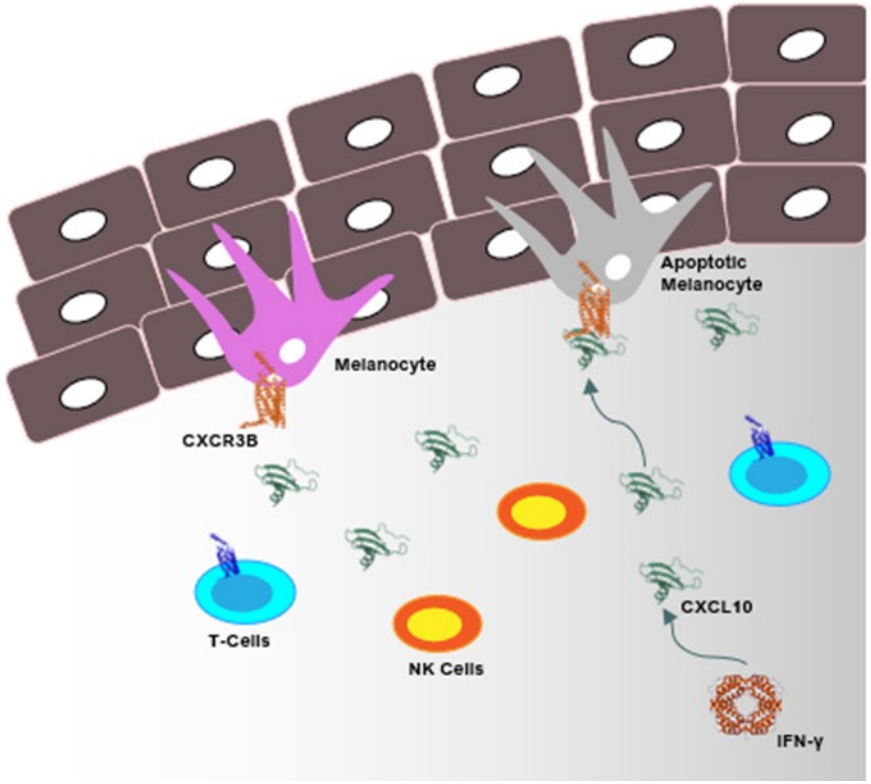
JAK inhibitor (ruxolitinib) interferes with:

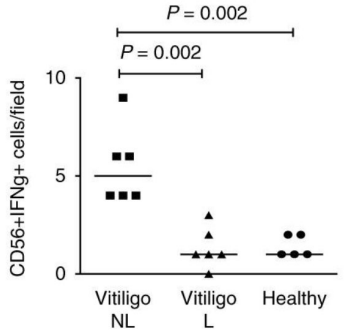
3. the JAK-STAT signaling that leads to production of CXCL9/10.

Vitiligo Progression – In Non-Lesional Skin

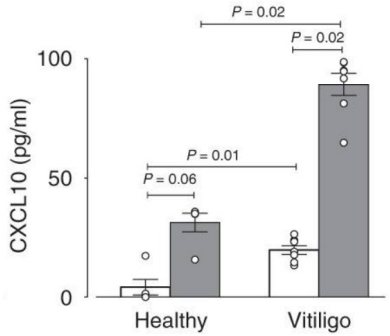
Innate cells are Important in CXCL10 Expression and Melanocyte Apoptosis Occurs via CXCL10-CXCR3B

Non-Lesional (Innate)

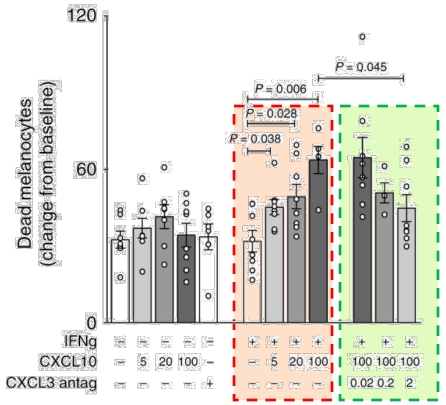




Increased levels of NK cells in non-lesional vitiligo



Increased levels of CXCL10 with and without IFN-γ in vitiligo



Dose dependent **melanocyte death** in response to increasing CXCL10 levels

Dose dependent **melanocyte survival** in response to increasing CXCL10-CXCR3 antagonist

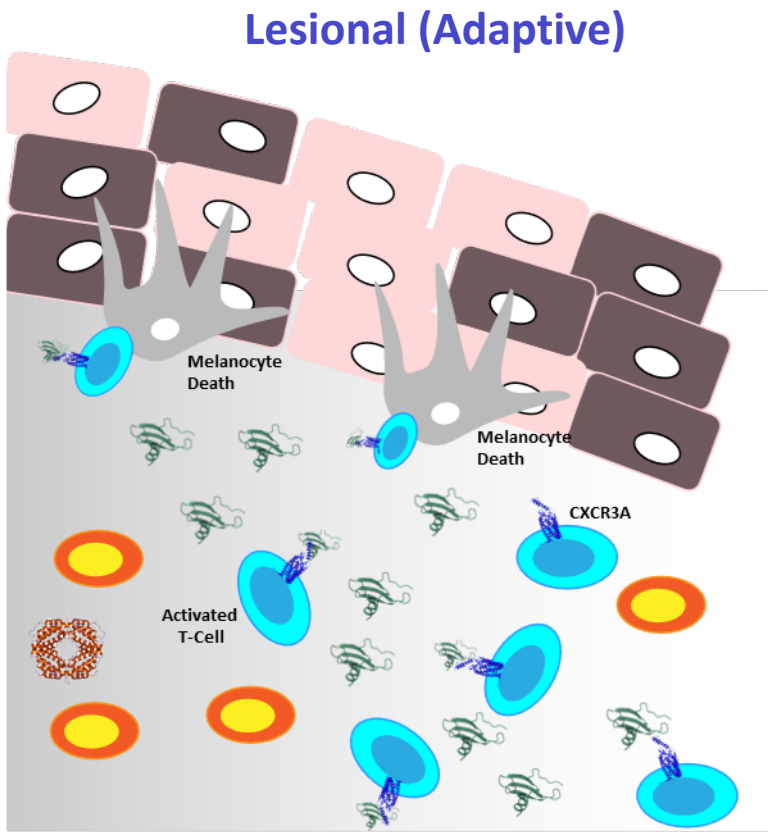
Edesa Biotech

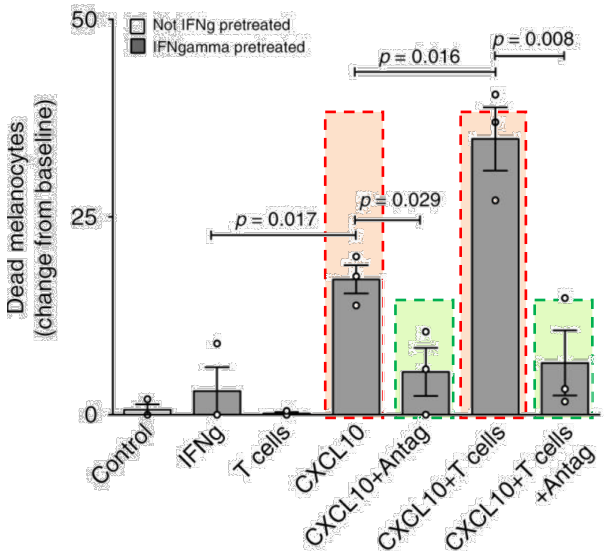
Tulic et al. Nat Commun. 2019 May 16;10(1):2178.

Vitiligo Progression – In Non-Lesional Skin

Innate cells are Important in CXCL10 Expression and Melanocyte Apoptosis Occurs via CXCL10-CXCR3B

Lesional (Adaptive)





Melanocyte death is increased in the presence of CXCL10

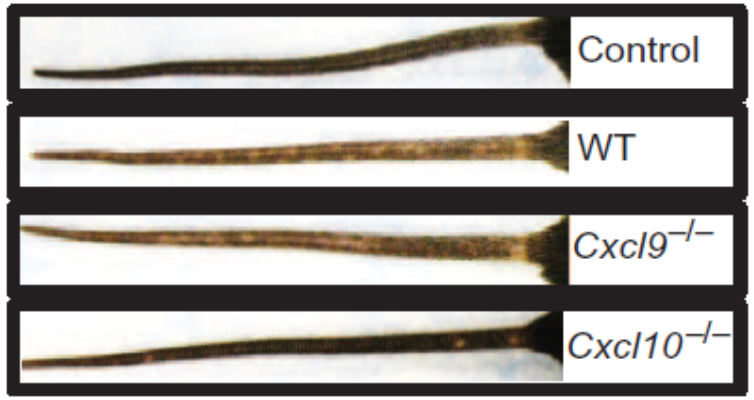
Melanocyte survival increased when CXCL10 / CXCR3 is blocked

Tulic et al. Nat Commun. 2019 May 16;10(1):2178.

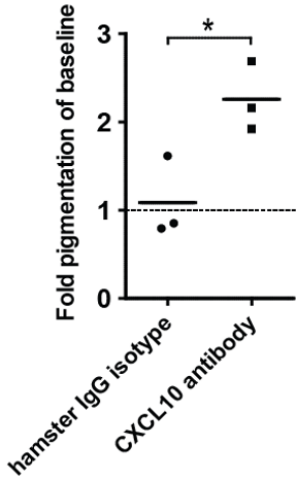
Vitiligo – Blocking IFNγ-CXCL10-CXCR3 Chemokine Axis

Evidence of therapeutic potential of CXCL10 as a target

Mice without CXCL10 do not develop Vitiligo



Anti-CXCL10 Antibodies Result in Re-pigmentation of Mice with Vitiligo

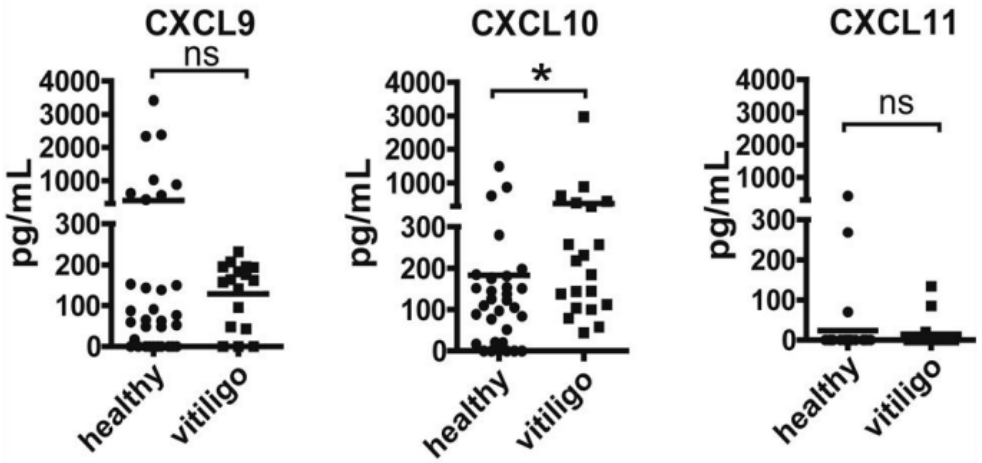


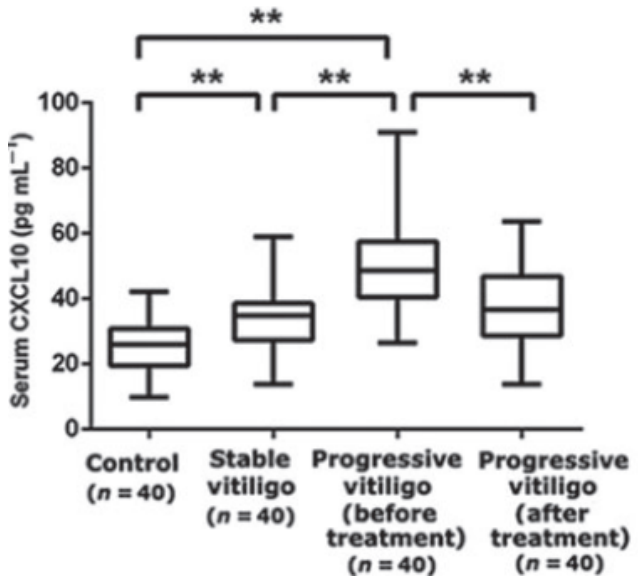
Edesa Biotech®

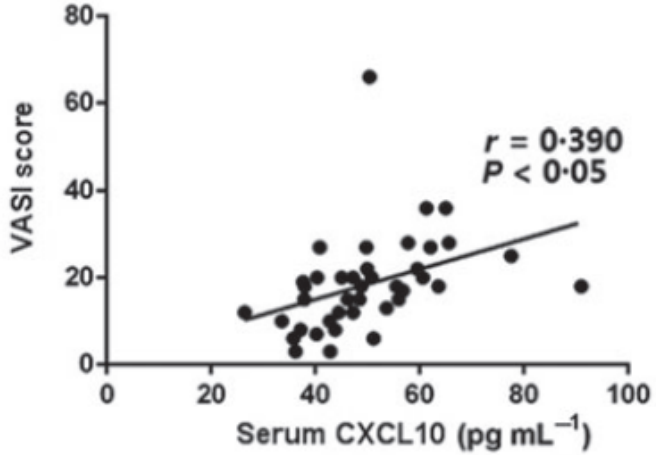
Rashigi *et al*. Science Translational Medicine, 2014

Biomarkers for Vitiligo - CXCL10 indicative of Disease and Severity

Increased levels of CXCL10 in Serum is Characteristic of Vitiligo







CXCL10 in Patient Samples → Predictors of disease progression and severity

Rashigi *et al*. Science Translational Medicine, 2014

Wang et al. British Journal of Medicine, 2016.

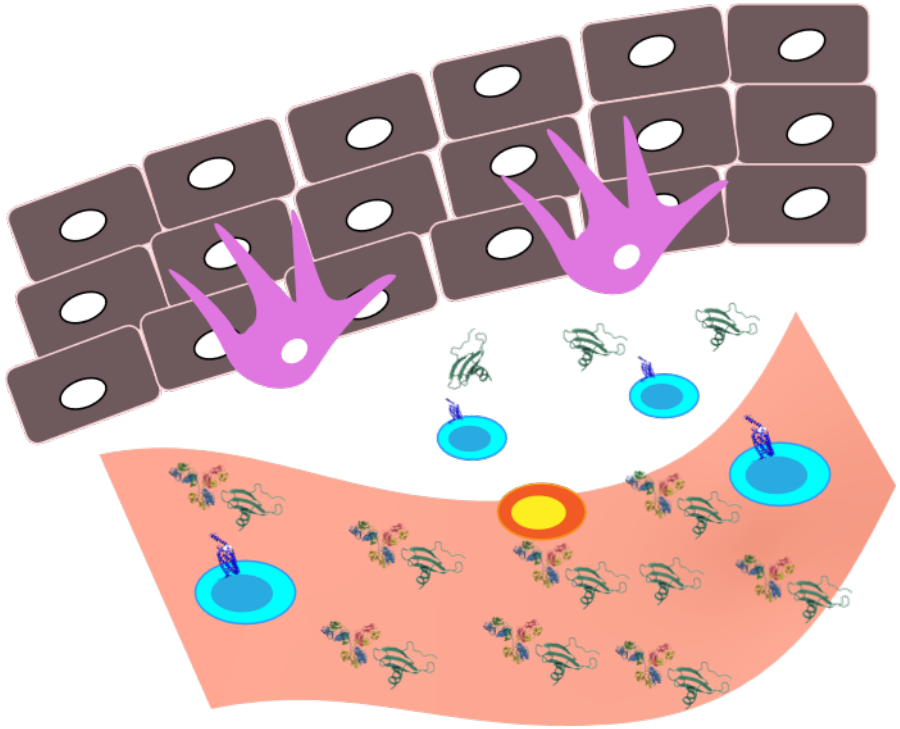
EB06 - Vitiligo

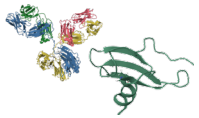
Clinical Evidence

Edesa Biotech™

Evidence supporting EB06's Biological Activity in Humans

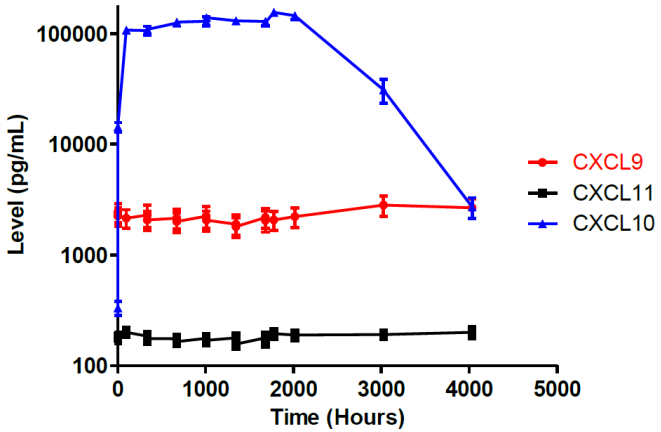
Treatment with EB06 Traps CXCL10 in the Circulatory System and Renders it Biologically Inactive

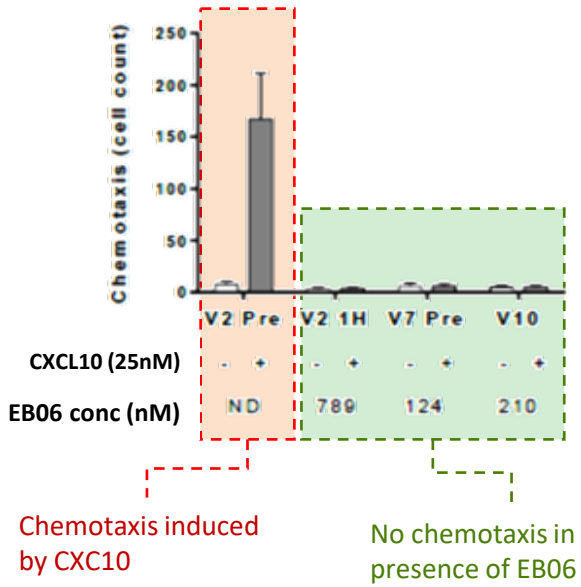




= EB06/CXCL10 Complex

EB06 induces an increase in serum CXCL10 levels while rendering them biologically inactive

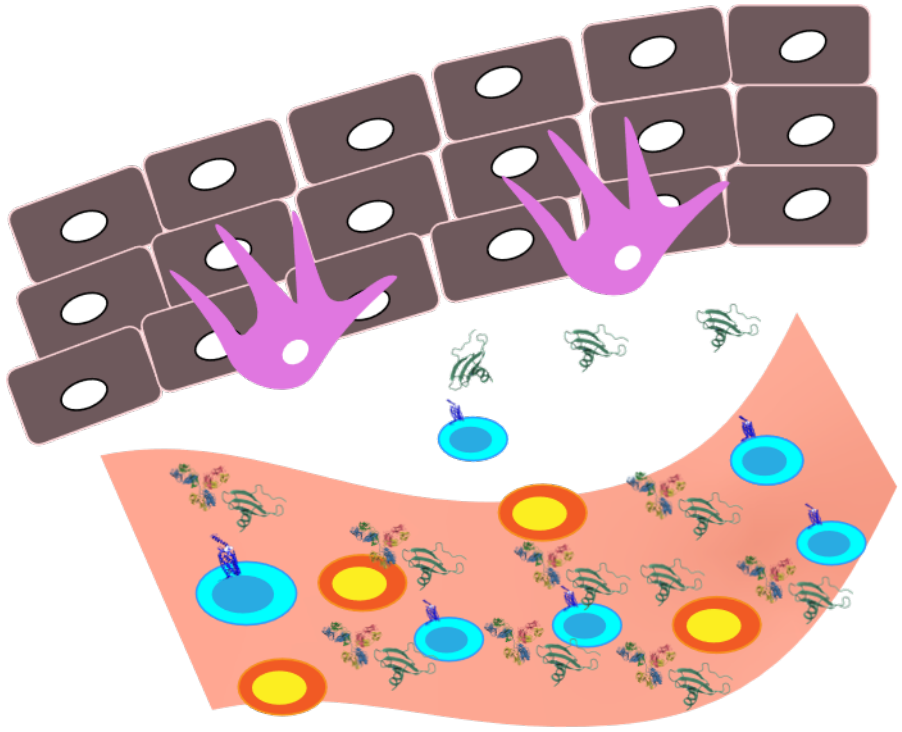




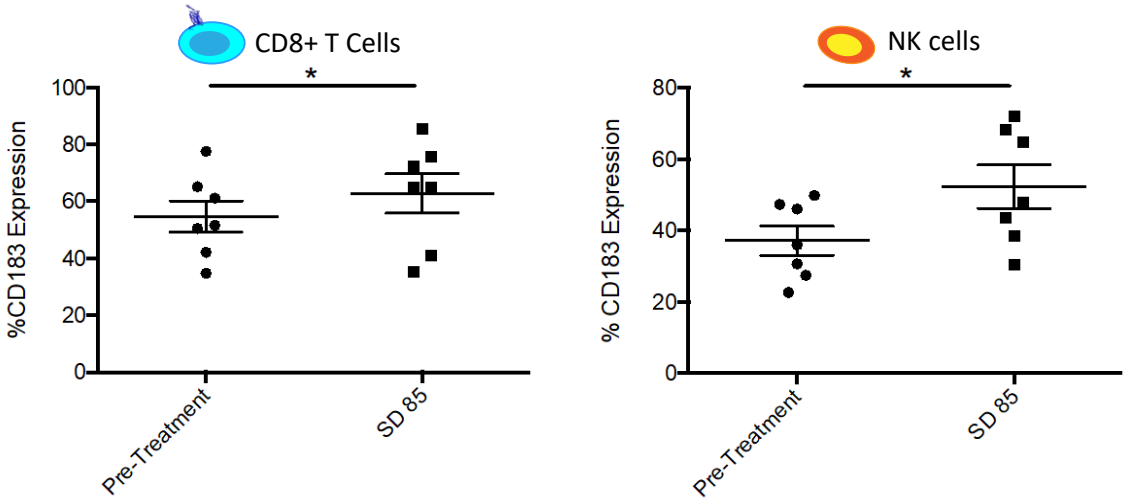
Edesa Biotech

Evidence supporting EB06's Biological Activity in Humans

With Chemotactic Gradient Disrupted by EB06, CXCR3+ leukocytes are Unable to Migrate and Remain in Circulation



EB06 induces an increased proportion of CXCR3+ leukocytes in circulation



Phase 2 Proof of Concept

Moderate to Severe Non-Segmental (Generalized) Vitiligo

Status	CTA approved & IND in progress
Subjects	Total of up to 160 evaluable patients across up to 25 study centers
Treatment Period	EB06 or placebo will be administered via IV every two weeks for up to 24 weeks, followed by a 12 week follow up period.
Primary Endpoint	Proportion of patients achieving F-VASI50 at week 24
Secondary Endpoints	Endpoints based on F-VASI50 and F-VASI75, mean % change in F-VASI, same for T-VASI and others Number of treatment-emergent adverse events and serious adverse events.

F-VASI = Facial Vitiligo Area Scoring Index; T-VASI = Total Vitiligo Area Scoring Index. Final study protocol subject to change

EB06: Anti-CXCL10 Monoclonal Antibody

Summary and Next Steps

Targeted Mechanism of Action
Binds free and bound CXCL10

65 Subjects Dosed
No Significant AEs

Biological Activity
Demonstrated

Phase 2 Ready
CTA Approved

Manufacturing
Leading CDMO

NEXT STEPS

IND in progress

CRO identified and ready to be initiated

Manufacturing campaign activities underway

Edesa Biotech®

www.EdesaBiotech.com
investors@edesabiotech.com

EDSA
Nasdaq
LISTED