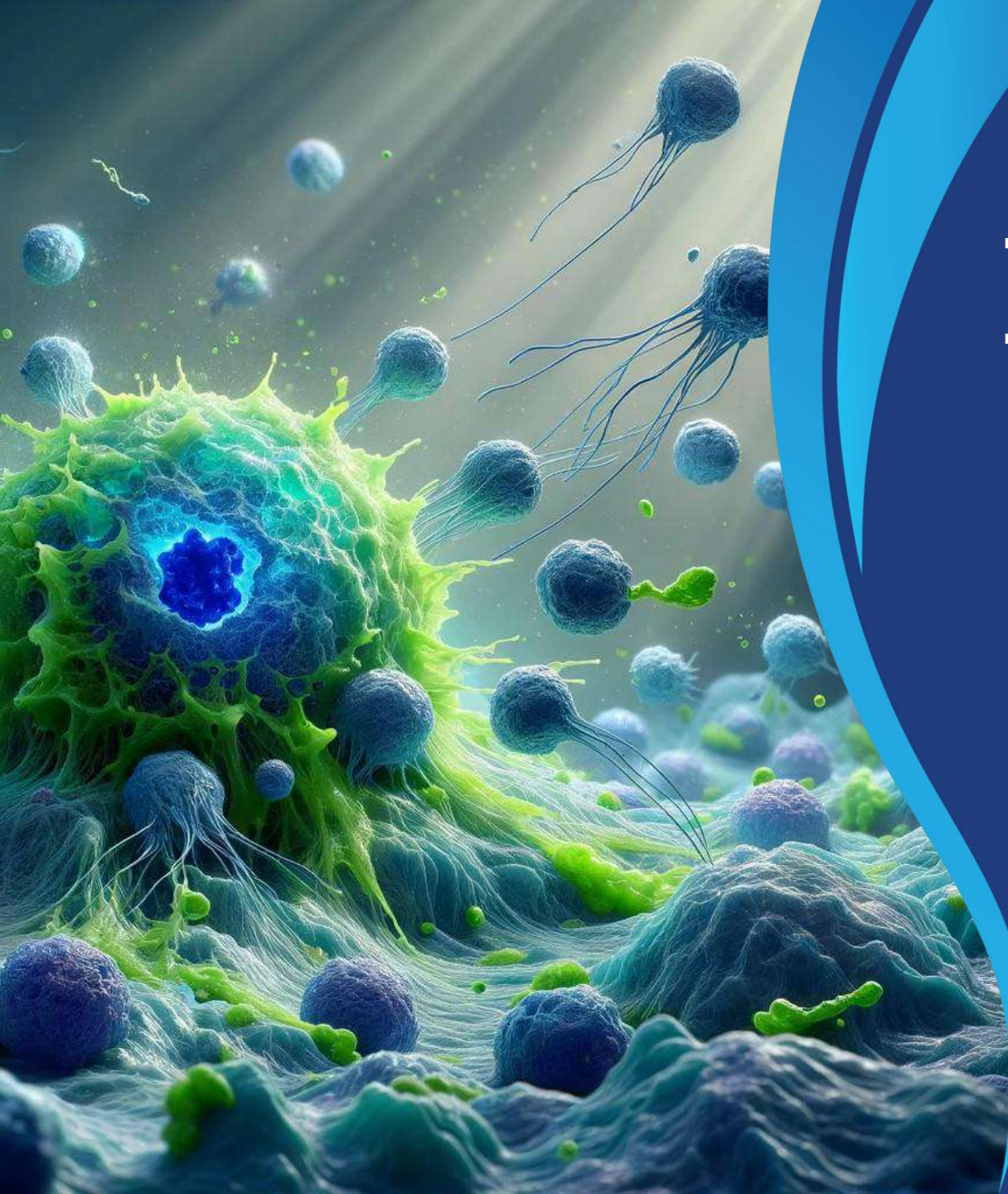




Two Late-Stage Platforms Targeting Inflammation- Driven Disease

Corporate Presentation

October 2026

NASDAQ: INMB



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Clinical trials are in early stages and there is no assurance that any specific outcome will be achieved. Any statements contained herein related to the development or commercialization of product candidates and other business and financial matters, including without limitation, trial results and data, including the results of the Phase 2 MINDFuL trial, the timing of key milestones, future plans or expectations for the treatment of XPro™, and the prospects for receiving regulatory approval or commercializing or selling any product or drug candidates may constitute forward-looking statements as that term is defined in the Private Securities Litigation Reform Act of 1995. Any forward-looking statements contained herein are based on current expectations but are subject to several risks and uncertainties. Actual results and the timing of certain events and circumstances may differ materially from those described by the forward-looking statements because of these risks and uncertainties. CORDstrom™, XPro1595 (XPro™, pegipanermin), and INKmune®™ have either finished clinical trials, are still in clinical trials or are preparing to start clinical trials and have not been approved by the US Food and Drug Administration (FDA) or any regulatory body and there cannot be any assurance that they will be approved by the FDA or any regulatory body or that any specific results will be achieved. The factors that could cause actual future results to differ materially from current expectations include, but are not limited to, risks and uncertainties relating to the Company’s ability to produce more drug for clinical trials; the availability of substantial additional funding for the Company to continue its operations and to conduct research and development, clinical studies and future product commercialization; and, the Company’s business, research, product development, regulatory approval, marketing and distribution plans and strategies. These and other factors are identified and described in more detail in the Company’s filings with the Securities and Exchange Commission, including the Company’s Annual Report on Form 10-K, the Company’s Quarterly Reports on Form 10-Q and the Company’s Current Reports on Form 8-K. The Company assumes no obligation to update any forward-looking statements to reflect any event or circumstance that may arise after the date hereof.



Two Late-Stage Platforms Across Inflammation & Immunology

Ebstrocel™ / CORDStrom™

RDEB regulatory submissions

UK pre-MAA package submitted
Full UK MAA targeted for February 2027
Potential UK approval in mid-2027

U.S. BLA planned for Q2 2027, subject to FDA feedback.

Uniphar agreement supports planned named-patient access in selected European and MENA markets.

XPro1595™

Selective soluble TNF inhibitor

Designed to reduce harmful inflammation while preserving transmembrane TNF signaling that supports immune function and tissue repair.

Rare disease expansion

Selected TNF-driven diseases could offer smaller, shorter trials and faster development than Alzheimer's.

Potential additional PRV opportunity through an eligible pediatric rare disease approval.*

*PRV eligibility and FDA award are not assured. Current program sunsets September 30, 2029. Development speed depends on indication, endpoints and recruitment. Ebstrocel timelines remain subject to regulatory review. Named-patient access requires applicable local permissions and implementation agreements.



Key Patents & Exclusivity Position

CORDStrom™

International PCT Stage
USPTO (ISA)
Favorable Written
Opinion on Patentability
(all claims)
-CoM
-Formulation
-MoT

**Nominal Patent
Coverage thru 2045**

Eligible for PTE up to
+5years on approval

**additional patent properties pending*

Eligible for Ref. Biol. Product Exclusivity (~12 years)

ODD Exclusivity (RDEB) ~7years

(12) INTERNATIONAL APPLICATION PUBLISHED UNDER THE PATENT COOPERATION TREATY (PCT)
(19) World Intellectual Property Organization
(43) International Publication Date: 28 August 2025 (28.08.2025)
(10) International Publication Number: WO 2025/179269 A1

(51) International Patent Classification: A61K 35/17 (2015.01), A61K 35/28 (2006.01), C12N 5/077 (2010.01)
(21) International Application Number: PCT/US2025/017038
(22) International Filing Date: 24 February 2025 (24.02.2025)
(25) Filing Language: English
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(30) Priority Data: 63/556,999 (23 February 2024 (23.02.2024)), 63/742,382 (06 January 2025 (06.01.2025)), 63/756,066 (10 February 2025 (10.02.2025))
(71) Applicant: IMMUNE BIO INC. (US), 225 NE Mizner Blvd., Ste 640, Boca Raton, Florida 33432 (US)
(72) Inventors: WEIL, Ben (London (GB)), LOWELL, Mark (London (GB)), SCHOONOVER, Joshua (Boca Raton, Florida (US))
(74) Agent: SCHOONOVER, Joshua, 225 NE Mizner Blvd., STE 640, Boca Raton, Florida 33432 (US)
Published: with international search report (Art. 21(3))

(54) Title: THERAPEUTIC COMPOSITIONS COMPRISING POOLED, CULTURE-EXPANDED HUMAN UMBILICAL CORD DERIVED MESENCHYMAL STROMAL CELLS

(57) Abstract: The disclosure provides a therapeutic composition comprising pooled, culture-expanded human umbilical cord-derived mesenchymal stromal cells (hUC-MSCs) derived from at least four and up to twelve donors, which can be reproduced with batch to batch consistency. These cells exhibit consistent marker expression profiles, enhanced and consistent secretion of therapeutic factors, and potent immunomodulatory activities after at least ten to thirty population doublings. The composition, formulated with a pharmaceutically acceptable carrier, is designed for clinical applications addressing inflammatory, immune, and degenerative conditions.

FIG. 15

WO 2025/179269 A1

XPro1595™

International PCT Stage
USPTO (ISA)
Favorable Written
Opinion on Patentability
(all claims)
-CoM
-Formulation
-MoT

**Nominal Patent
Coverage thru 2045**

Eligible for PTE up to
+5years on approval

**US 11,365,229 B2 Expires 2033
-MoT (Neurologic Diseases)**

(12) INTERNATIONAL APPLICATION PUBLISHED UNDER THE PATENT COOPERATION TREATY (PCT)
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(71) Applicant: IMMUNE BIO INC. (US), 225 NE Mizner Blvd., STE 640, Boca Raton, Florida 33432 (US)
(72) Inventor: SCHOONOVER, Joshua, 225 NE Mizner Blvd., STE 640, Boca Raton, Florida 33432 (US), TESL, Raymond J., 225 NE Mizner Blvd., STE 640, Boca Raton, Florida 33432 (US)
(74) Agent: SCHOONOVER, Joshua, 225 NE Mizner Blvd., STE 640, Boca Raton, Florida 33432 (US)

(54) Title: STABILIZED DN-TNF MUTEN BIOCONJUGATES FOR SELECTIVE SOLUBLE TNF NEUTRALIZATION

FIG. 5

(57) Abstract: A therapeutic composition is described including a selective soluble TNF-sequestering bioconjugate for treating TNF-mediated inflammatory disorders. The bioconjugate includes a dominant-negative TNF (DN-TNF) muten, which contains one or more amino acid substitutions in the TNF receptor interaction domain, trimer interface domain, or both, to reduce receptor binding while enhancing hexamer formation with wild-type TNF, thereby sequestering soluble TNF. The DN-TNF muten is covalently conjugated to a biocompatible stealth polymer via a hydrolyzed malimide linker, preventing non-Michael reaction and polymer dissociation. The composition is formulated as an injectable buffered aqueous solution for subcutaneous, intramuscular, intravitreal, or intravenous administration. Also disclosed are methods for stabilizing the bioconjugate through controlled hydrolysis of the malimide linker and methods for treating TNF-mediated inflammatory disorders by administering the composition to a subject in need thereof.

WO 2025/189204 A2

**additional patent properties issued/pending*

Eligible for Ref. Biol. Product Exclusivity (~12 years)

INmuneBio

CORDStrom™ (*Ebstrocel*) for EB

Beyond Topical Care: Disease-Modifying Systemic Therapy



CORDStrom™ – a Mesenchymal Stromal Cell (MSC) Platform

What is CORDStrom™?

- Pooled, allogeneic donor, umbilical cord-derived Mesenchymal Stromal/Stem Cell (MSC) product (minimum four donors)
- Highly reproducible, off-the-shelf
- Global IP Filed
- Large Volume Manufacturing
- Cost Effective
- Easily genetically modified to deliver payloads

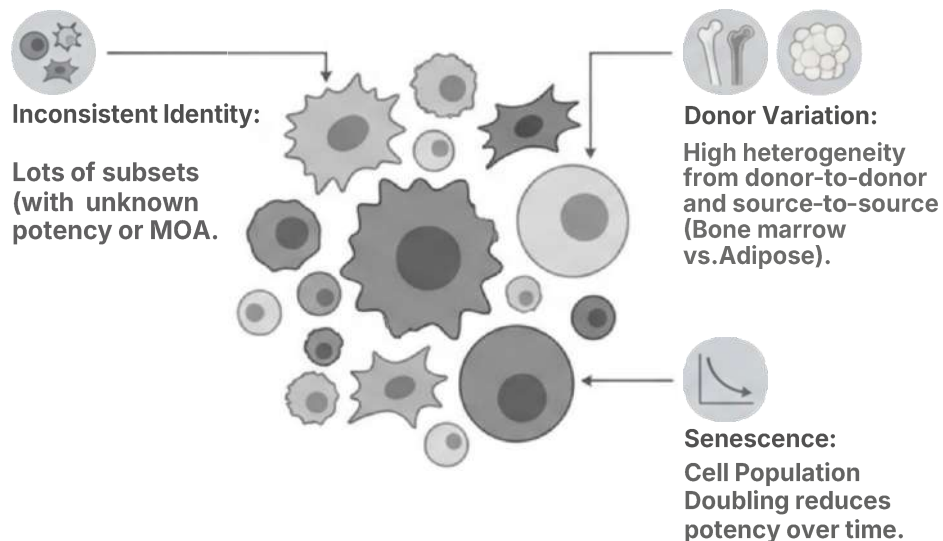
Mechanisms Of Action

- Suppression of inflammation: **RDEB**/EB, OA, trauma, etc
- Immunomodulation: Autoimmune diseases etc
- Enhanced wound healing: **RDEB**/EB, post surgery, SLE, etc
- Suppression of fibrosis: OA, cardiac repair, **RDEB**/EB, etc



Why Historical MSC Development Failed: The Problem of Heterogeneity – AND THE SOLUTION

The Heterogeneity issue



The Consequence

Clinically Naive Assumptions.

The assumption that "all MSCs are the same" led to few approvals. Past failures were not due to a lack of therapeutic potential, but a lack of product consistency.

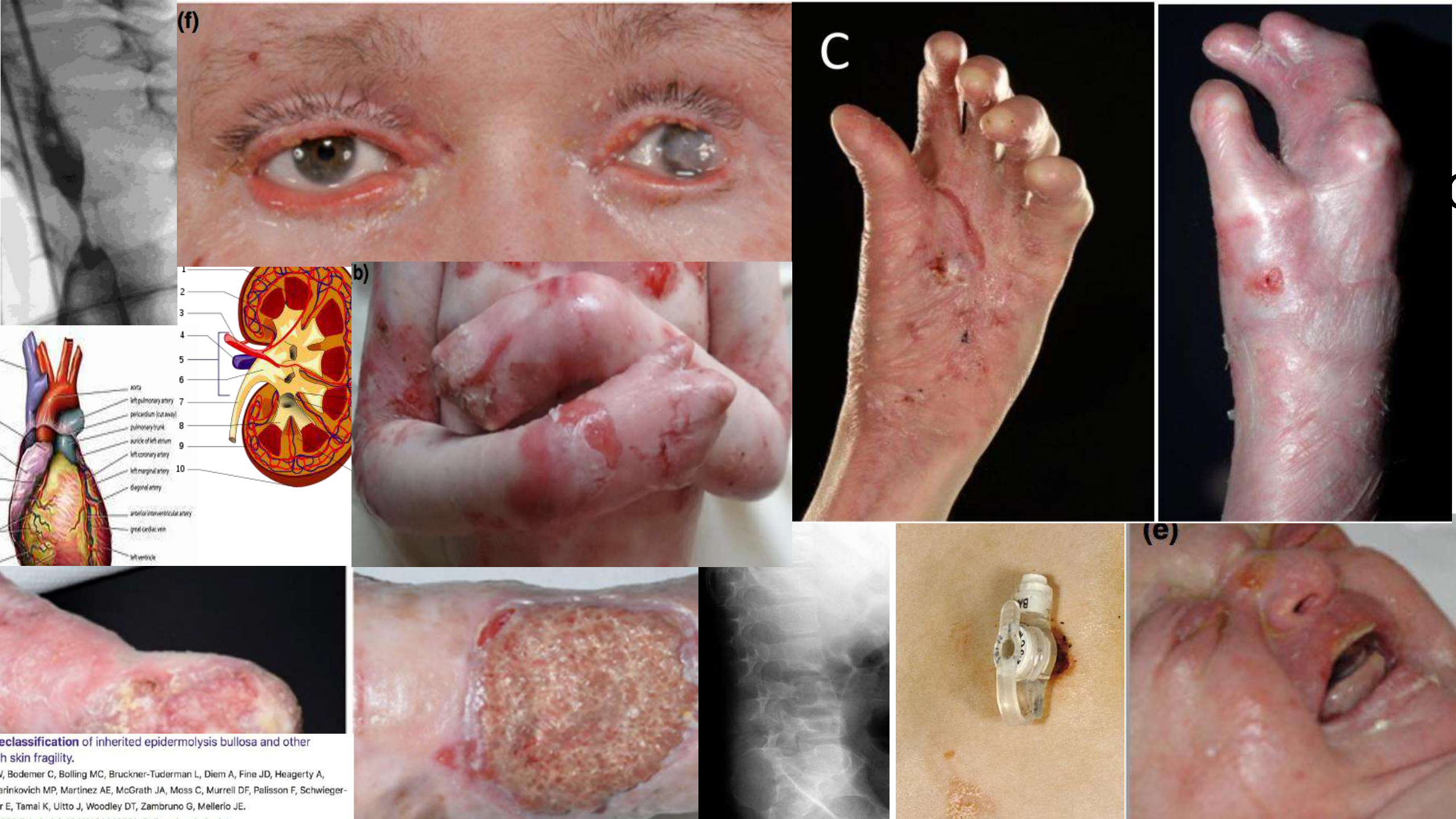
The Answer

CORDStrom

Umbilical Cord derived MSC
Pooled donors – homogeneity
Product defined on potency.



Recessive Dystrophic EB



Reclassification of inherited epidermolysis bullosa and other skin fragility.

W, Bodemer C, Bolling MC, Bruckner-Tuderman L, Diem A, Fine JD, Heagerty A, Marinkovich MP, Martinez AE, McGrath JA, Moss C, Murrell DF, Palissot F, Schwieger E, Tamai K, Utito J, Woodley DT, Zambruno G, Mellerio JE.



The Progressive Nature of Recessive Dystrophic EB (RDEB)

18 Months to 10 Years: The Critical Therapeutic Window

Journal of
Dermatology and Venereology



Prospective | Open Access

The Natural History of Severe Recessive Dystrophic Epidermolysis Bullosa – 4 Phases Which May Help Determine Different Therapeutic Approaches

Bageta ML^{1*}, Yerlett N², McGrath JA³, Mellerio JE⁴, Petrof G¹, Martinez AE¹



0-18
months



18 months-10
years



10-20
years



Over 20
years



Squamous cell
carcinoma (SCC)



What Treatments do People Living with DEB Want Right Now?

Prioritisation survey
865 members EB
global community
2024-Top 5 issues

1. Pain relief

2. Itch relief

3. Mental health children/YP

4. Impact of inflammation

5. GI issues e.g., dysphagia

Source: FDA Patient-Focused Drug Development Meeting
<https://www.youtube.com/live/lds1WAe5Czo>



Global EB Priority Setting Partnership

identifies research priorities for the
four main types of epidermolysis bullosa



Pruritus is a Clinically Meaningful Endpoint in RDEB

Evidence from Danial et al. (2015) and the PEBLES Prospective Registry (2023)

Danial et al. (2015)

- 146 EB patients
- Itch ranked the most bothersome complication
- Healing wounds produced greatest itch
- Major impact on sleep and quality of life
- Defined key itch triggers

PEBLES Registry (2023)

- 50 RDEB patients
- 243 longitudinal assessments over 7 years
- Itch present in 93% of assessments
- Severe RDEB had highest itch burden
- Supports itch as endpoint in future trials

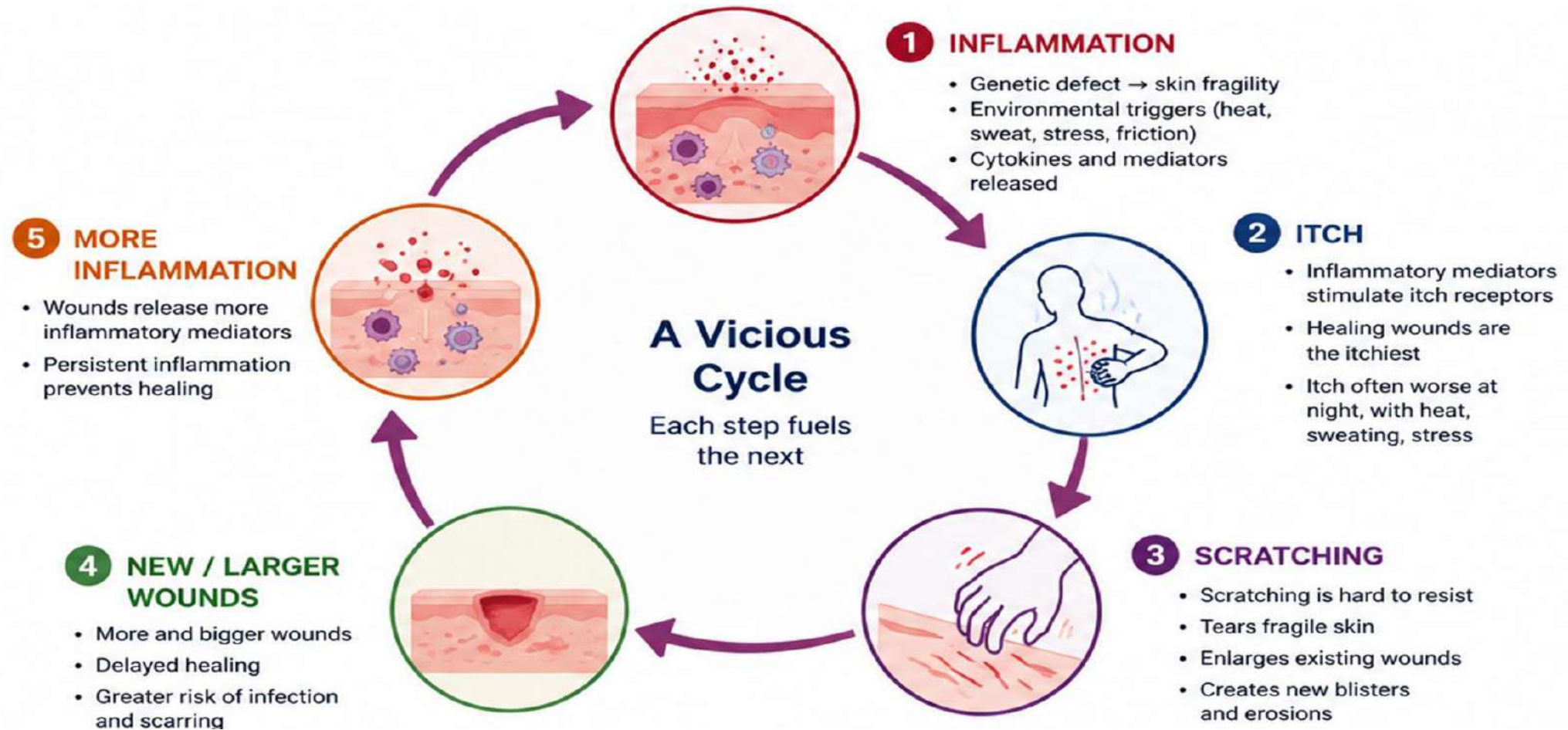
Key Clinical Findings

- ✓ Itch is nearly universal in RDEB (93% prevalence) and in wider EB (~85% prevalence)
- ✓ Patients describe itch as deep, painful and difficult to control
- ✓ Severity increases with disease severity
- ✓ Patients rank itch among the most burdensome symptoms
- ✓ Itch drives the itch-scratch-wound-inflammation cycle
- ✓ Healing wounds produce the greatest itch
- ✓ **Current therapies provide inadequate relief**
- ✓ Night-time itch causes significant sleep disruption
- ✓ Heat, sweating, stress and dry skin worsen symptoms
- ✓ Global disease severity scores do not adequately capture itch
- ✓ **Both studies support itch as a clinically meaningful endpoint in RDEB and in wider EB**



The Itch-Scratch-Wound-Inflammation Cycle in RDEB

A self-perpetuating cycle that drives diseases disease burden





Why Reducing Pruritus May Reduce Wounds and Long-Term SCC Risk in RDEB



Evidence

- Scratching tears fragile skin, enlarges wounds and creates new blisters.
- Healing wounds are the itchiest wounds.
- Larger wounds are associated with greater itch burden.
- MSC and calcipotriol studies showed wound improvement together with itch reduction.
- Current evidence strongly supports: ↓ itch → ↓ scratching → ↓ wound burden.
- ***Ebstrocel reduced systemic inflammatory cytokines which drive itch leading to clinically meaningful reduction***

Implications

- Chronic non-healing wounds are the principal substrate for SCC in RDEB.
- Persistent inflammation drives repeated tissue injury and malignant transformation.
- No study has yet proven that reducing itch lowers SCC incidence.
- However, the biological rationale is strong: interrupting the itch-scratch cycle may reduce chronic wounds and, over time, could reduce SCC risk.

“Treatment with Ebstrocel infusions in RDEB patients is likely to be disease-modifying, improving itch, skin healing, quality of life and reducing the risk of squamous cell carcinoma later in life” –

Professor Anna Martinez, UK clinical lead for paediatric RDEB.

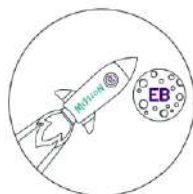


Mission EB Phase III: Randomized, Placebo-Controlled Crossover Trial

2012 - Cure EB Charity funded **EBSTEM** an open label study 10 children with RDEB from Great Ormond Street treated with 3 infusions of bone derived stem cells with promising results lasting up to 6 months

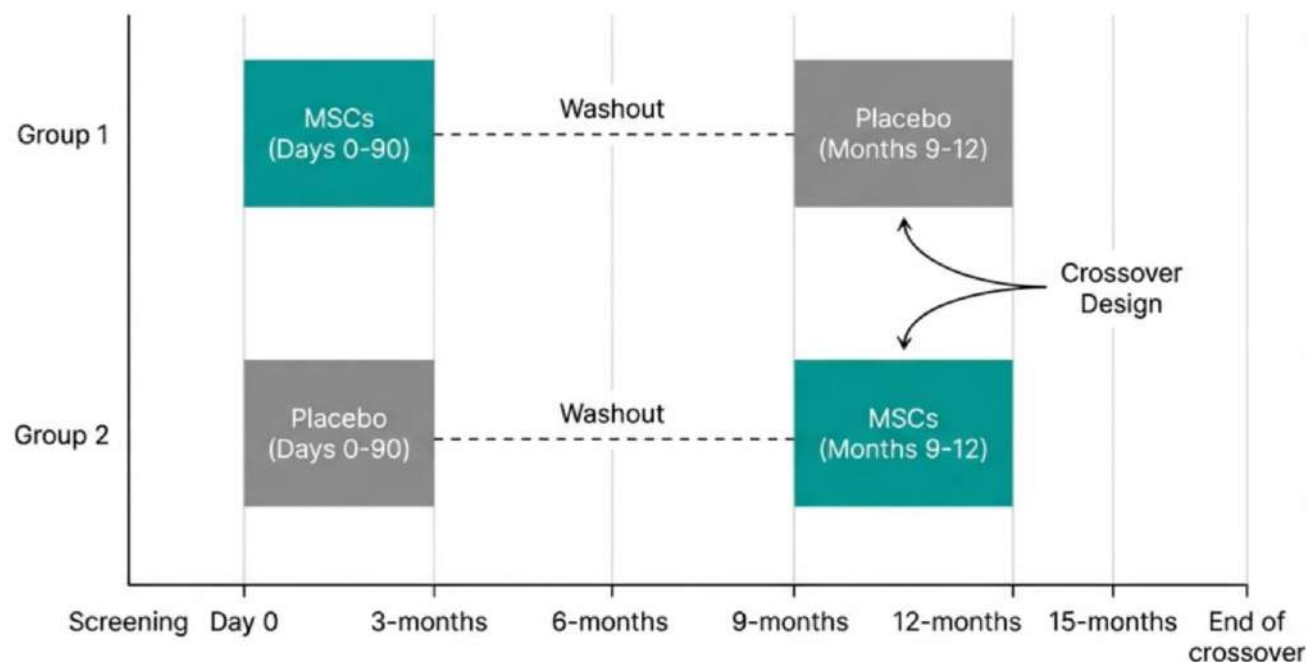
2019 – NIHR / NHS England / CureEB funded a **Double blind, placebo controlled, crossover** trial at GOSH and BCH for children with RDEB

MissionEB



Mesenchymal Intravenous Stromal cell Infusions in children with recessive dystrophic Epidermolysis Bullosa

Both UK National centres, 33 enrolled, 31 evaluable –
all crossed over from drug to placebo or placebo to drug
all remained double blind through treatment and follow-up





Month-3 Data

- **RDEB-Severe** patients across all ages (9/30)
 - No improvement in their skin scores
 - **25% reduction in ITCH**

Month-6 Data

- Itch reduction **maintained** 6 months post cell infusions across **all** of the itch-man scale cohort
- The largest itch reduction was seen in the **RDEB-S** group at month 6 post cell infusion of **27.5%**
- **IscorEB scores** improved in all **RDEB patients**
- There is large improvement in **all iscorEB** scores in RDEB-S cohort **not seen** at month 3
 - overall score 9.3%
 - patient score 9.6%
 - clinician score 6.4%
 - skin score 2.8%



Conclusion From Month-6 Data

- There is **large improvement** in **all iscorEB scores** in **RDEB-S** cohort not seen at month-3
- The observed **skin improvement** and **wound closure** is likely to be **disease modifying**, improving **pain** and **quality of life** and **reduce** the long-term **risk of squamous cell carcinoma**



Qualitative Results

- 13 participants (reporting on 9 cases)
 - 8 parents
 - 5 children
- Completed blinded interview at two time points
 - 3 months post UC-MSCs
 - 3 months post placebo
- 12/13 reported benefits on Ebstrocel
- 3/13 reported benefits on placebo
 - 2 post Ebstrocel
 - 1 treatment naive
- **10/13 correctly identified order of treatment**

ID	Patient details	Role	UC-MSCs	Placebo
A	Under 6 years* RDEB intermediate Order of infusions: UC-MSCs/Placebo	Parent	• Parent described that wound healing was quicker and had changed in form and this reduced the frequency of dressing changes. • It was difficult to monitor whether the treatment had made a difference to some symptoms.	• Positive changes such as fewer blisters on the feet, fewer open wounds and as a result, less dressing changes. • Seemed to be talking about improvement since the start of the trial, rather than since the most recent infusion.
B	Under 6 years* RDEB intermediate Order of infusions: UC-MSCs/Placebo	Parent	• Reported a huge improvement all round: improvements in energy, eating, wound healing and recovery time from illness was quicker.	• Reported no change this time, compared to 1st infusion where there was a great improvement.
C	Over 10 years RDEB intermediate Order of infusions: UC-MSCs/Placebo	Patient Parent	• Reported a significant decrease of wounds and blisters, less pain and that new wounds healed faster. Improved sleep and general activities have become easier to do. • Felt that there was significant positive change since the infusion, skin was less sensitive, wound healing was quicker and less itchiness and pain.	• Said there was no impact of this infusion, and compared to the first infusion where they thought it had helped. • Reported eye pain and itchiness was worse after the 2nd infusion. • Compared this time to last time by saying that the 1st infusion was much better.
D	Over 10 years RDEB severe Order of infusions: UC-MSCs/Placebo	Patient Parent	• Reported having one positive experience where their skin had improved on holiday but that this returned to normal when he came back to the UK. • Nothing else had changed in relation to his EB symptoms.	• Reported a small positive change in pain and itchiness, wounds on their legs had decreased but when they returned, this was more difficult to deal with. • No impact on dressings, bathing or sleep.
E	Under 10 years RDEB intermediate Order of infusions: Placebo/UC-MSCs	Patient Parent	• Reported positive changes such as fewer blisters, quicker wound healing, less pain during bathing and EB activities took less time due to the decrease in pain. • Positive impact on quality of life e.g., able to socialise more with peers.	• Noticed improvement in wound healing; these improvements lasted two months and their legs were 'clear' for three weeks. • These changes had improved their social life as they were able to walk more and being abroad was easier due to fewer complications.
		Parent	• Described less pain and that wounds had healed quicker. This made bath times easier and impacted positively on their mental wellbeing. • They felt that the participant was more positive now because of the improvement and more willing to be engaged in activities.	• Reported no change in any symptoms or quality of life.
F	Over 10 years RDEB intermediate Order of infusions: Placebo/UC-MSCs	Patient Parent	• A bit less pain and itchiness since the infusion. Did not last long. Improved school and sleep. • Reported improvements in itchiness, pain and activities at school and sleep.	• Reported no change in any symptoms or quality of life. • Reported no change in any symptoms or quality of life.
G	Over 10 years RDEB intermediate Order of infusions: Placebo/UC-MSCs	Patient Parent	Withdrawn Withdrawn	• Improvement in skin, fewer cuts and they have healed quicker, less itchiness and were able to write longer at school because of the positive changes. • Highlighted positive improvement in wound healing and formation of blisters.
H	Under 6 years* RDEB severe Order of infusions: Placebo/UC-MSCs	Parent	• Reported improved sleep, energy, and mood. • No impact on skin and wounds.	• No change, stated everything is still the same.
I	Under 6 years* RDEB severe Order of infusions: Placebo/UC-MSCs	Parent	• Unclear but thinks there may have been some improvement in the healing of wounds. Noticed less scratching but thought this could just be them growing up. • Childminder thought the skin had improved.	• Reported no change in any symptoms or quality of life, some more itching reported.
J	Under 10 years RDEB severe Order of infusions: Placebo/UC-MSCs	Patient Parent	• Reported that pain reduced a little, itchiness improved slightly and they had played with their sibling more since treatment. • No impact on sleep, their eyes or dressings. • Wounds are healing quicker. • Reported less waking but not certain if this is due to infusion or getting used to it.	• Initially said pain and itch had improved a little but later in the interview said it had not improved, no impact on activities. • Reported no change in any symptoms or quality of life, except more itching.

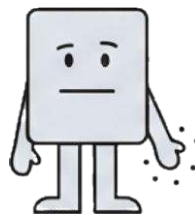
*Patients under 6 years were too young to be interviewed.

Table 3: Summary of qualitative interviews.



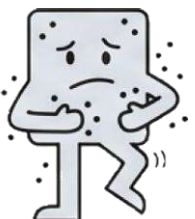
Comprehensive Organ System Improvements; Itch Man Scale

"I don't sleep because of the itch.
I just wait for the sun to come up."



Level 2: Itches more;
sometimes interferes
with activity

"The itch is just a different
form of pain."



Level 3: Itches a lot;
difficult to be still,
concentrate

"The itch is a monster... an itch that
feels like it's inside my bones."

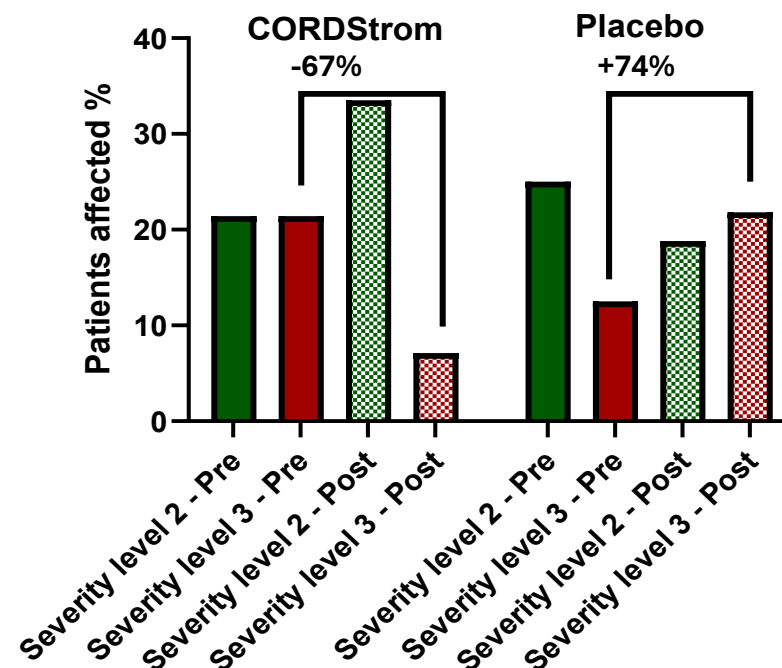
"I just wait for the sun to come up so
I can start the cycle of bandaging
the damage I did in my sleep."

Month 3 - Itch Man Scale
improved 17%

Month 6 - Itch Man
Scale improved **27.5%**

CS switches 2/3 of pts with grade 3 to grade 2
1/4 of pts on placebo who were grade 2
progress to grade 3

Itch Man Categorical
mITT

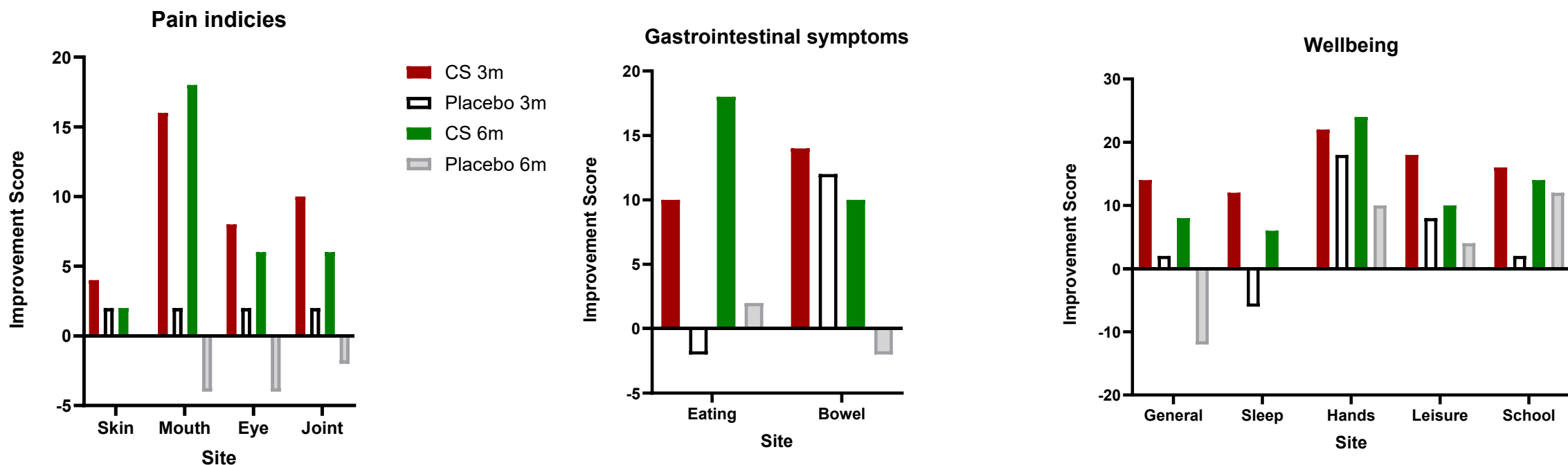




Comprehensive Organ System Improvements; ISCOREB Improvement Scores

Period 1 at 3 and 6 months

Ebstrocel is potentially the first systemic therapy with itch and pain relief benefits as key differentiation factors in quality of life.





Overall Summary

- Ebstrocel has proven to be beneficial treatment with **no safety signals** in children with RDEB from the age of 6 months
- **Intermediate and younger patients** under the age of 10 years saw earlier effects with **improvement of their skin, pain and itch**
- Although the **most severe and older patients** did not show changes in their skin at 3 months, by 6 months their **itch and skin scores had improved greatly**
- ***“Treatment with Ebstrocel infusions in RDEB patients is likely to be disease-modifying improving itch, skin healing, quality of life and reduce the risk of squamous cell carcinoma later in life”*** – Professor Anna Martinez, UK clinical lead for paediatric RDEB



Uniphar: International Access and Commercialization

Agreement signed with Uniphar Durbin Ireland Limited

A scalable framework for international access, distribution and future commercialization of Ebstrocel.

Initial access markets

Selected European countries and the Middle East and North Africa (MENA).

Physician-requested supply for individual eligible patients before local marketing approval, where permitted.

Capital-efficient commercial model

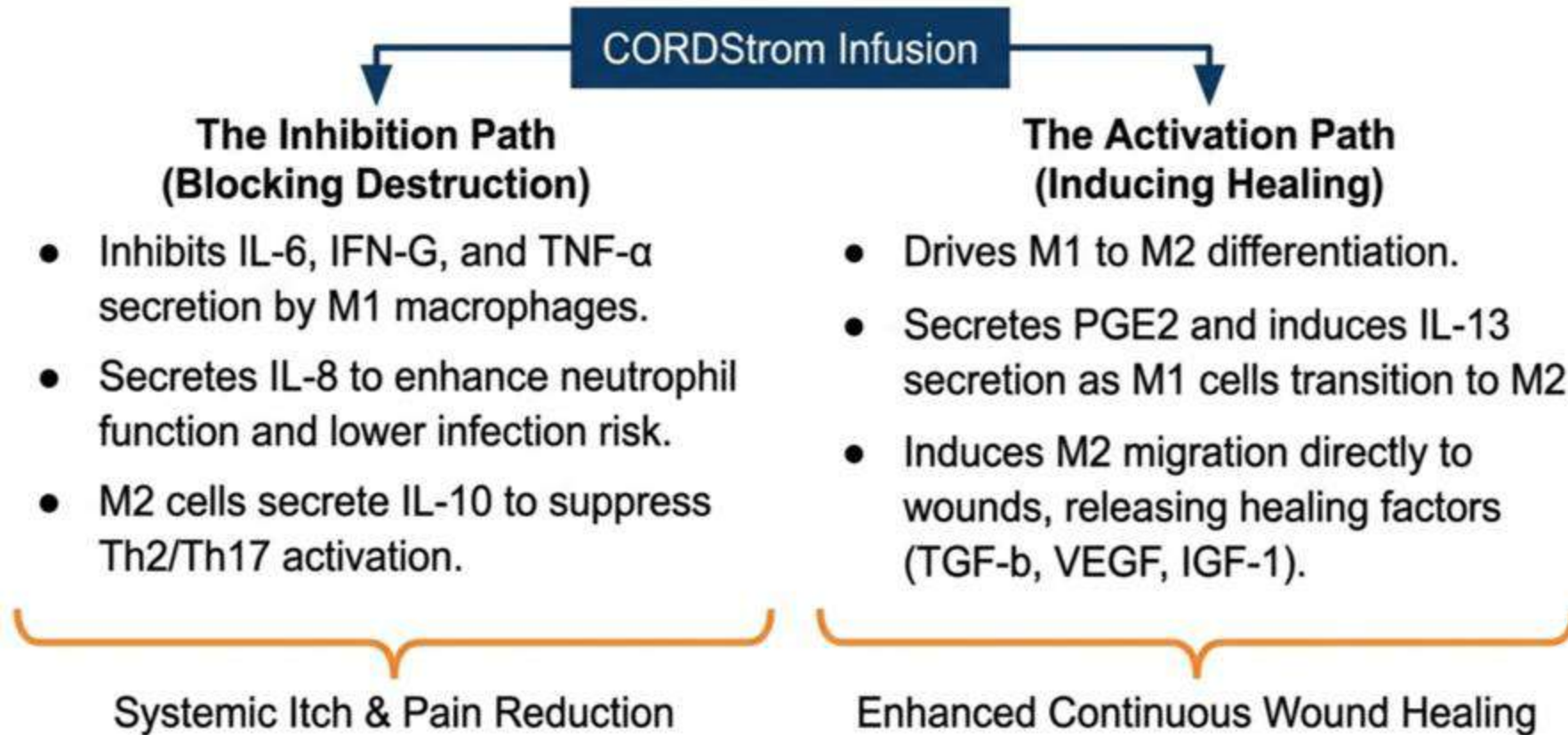
Use Uniphar's established infrastructure for market access, storage, distribution and order-to-cash services.

Potential expansion as approvals, pricing and reimbursement are secured.

Implementation depends on applicable law and country-specific statements of work. Additional territories and services require mutual agreement. Ebstrocel remains investigational.



Complex Mechanism of Systemic Resolution: M1 to M2 Shift

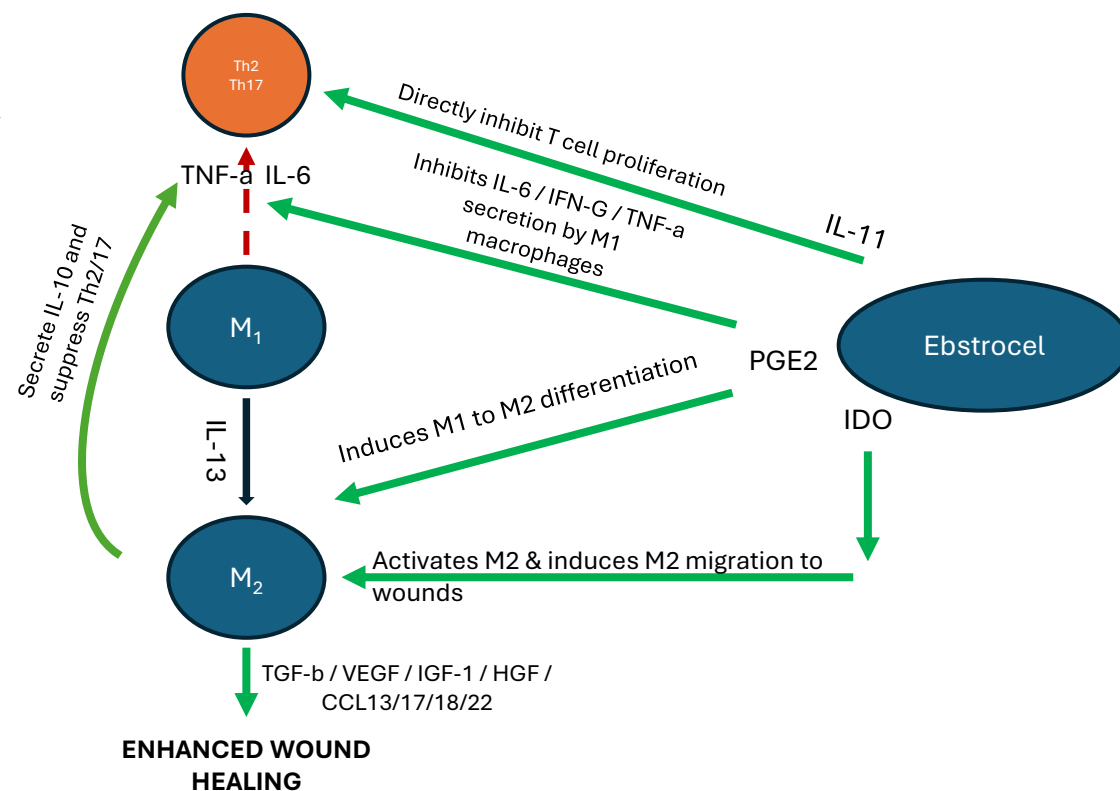
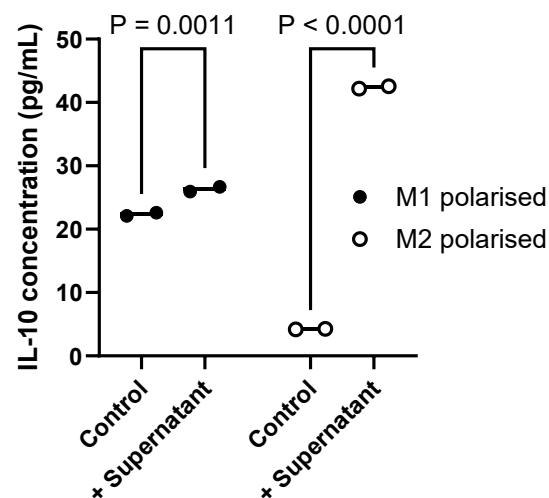
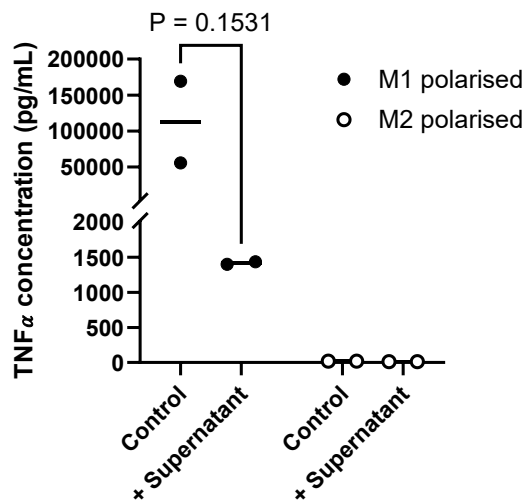
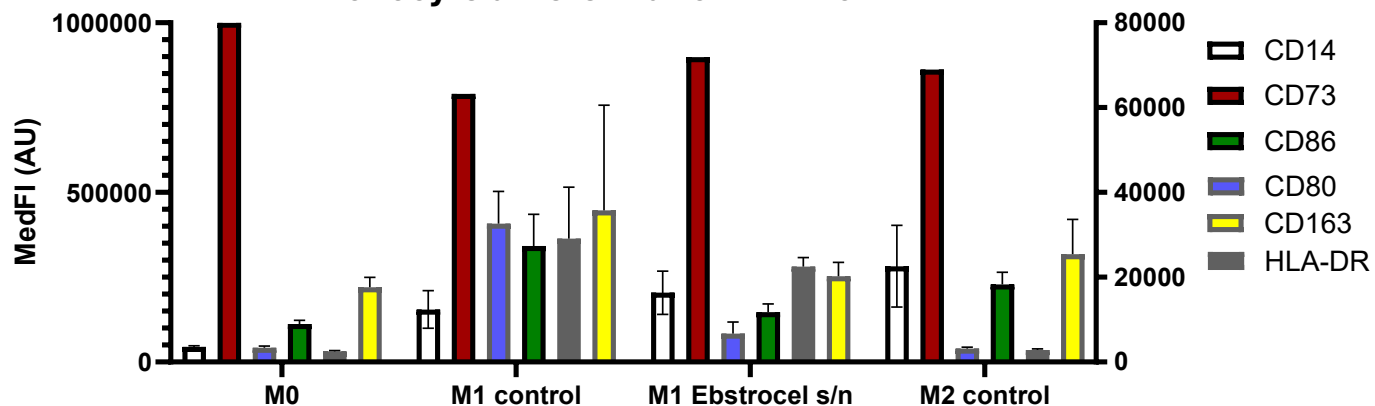




Ebstrocel Mechanisms of Action - Details

M1 to M2 differentiation *in vitro*

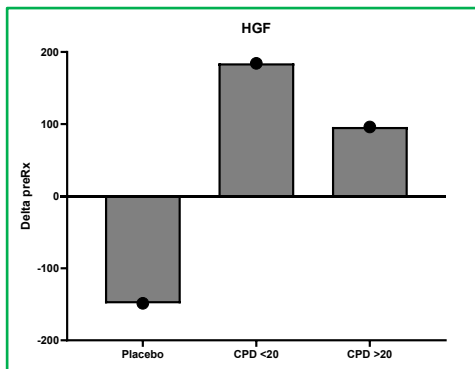
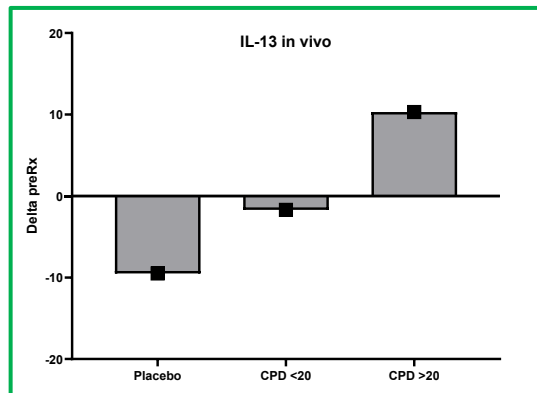
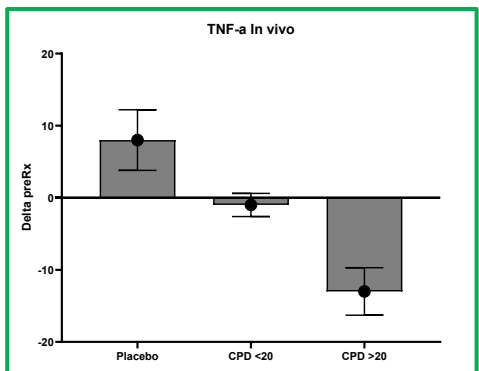
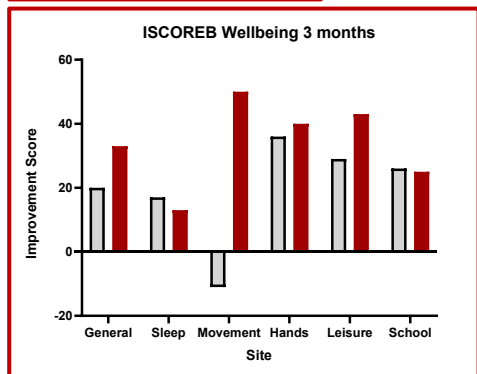
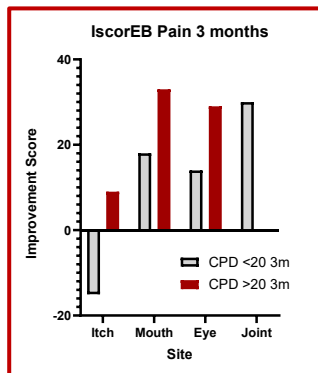
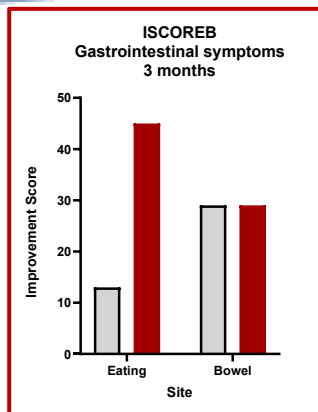
Monocyte differentiation in vitro





Ebstrocel Mechanisms of Action

Proof of MoA *in vivo*



Itch & Pain
REDUCTION

IL-31 & TSLP

IL-31

Secrete IL-10 and
suppress Th2/17

Th2
Th17

TNF- α

IL-6

M₁

M₂

ENHANCED WOUND
HEALING

Directly inhibit T cell proliferation

Inhibits IL-6 / IFN- γ / TNF- α
secretion by M1
macrophages

Induces M1 to M2 differentiation

Activates M2 & induces M2 migration to
wounds

TGF- β / VEGF / IGF-1 / HGF /
CCL13/17/18/22

IL-11

PGE2

Ebstrocel

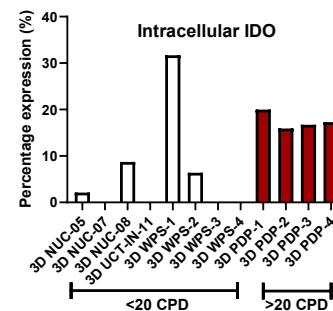
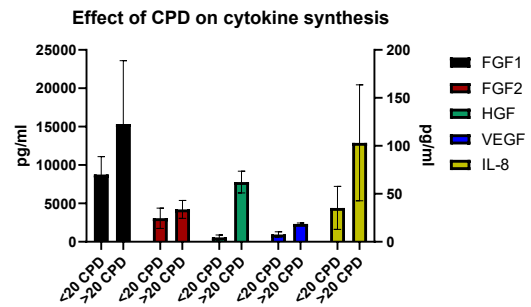
IDO

TGF- β / VEGF /
HGF

IL-8

Enhances
PMN
activation

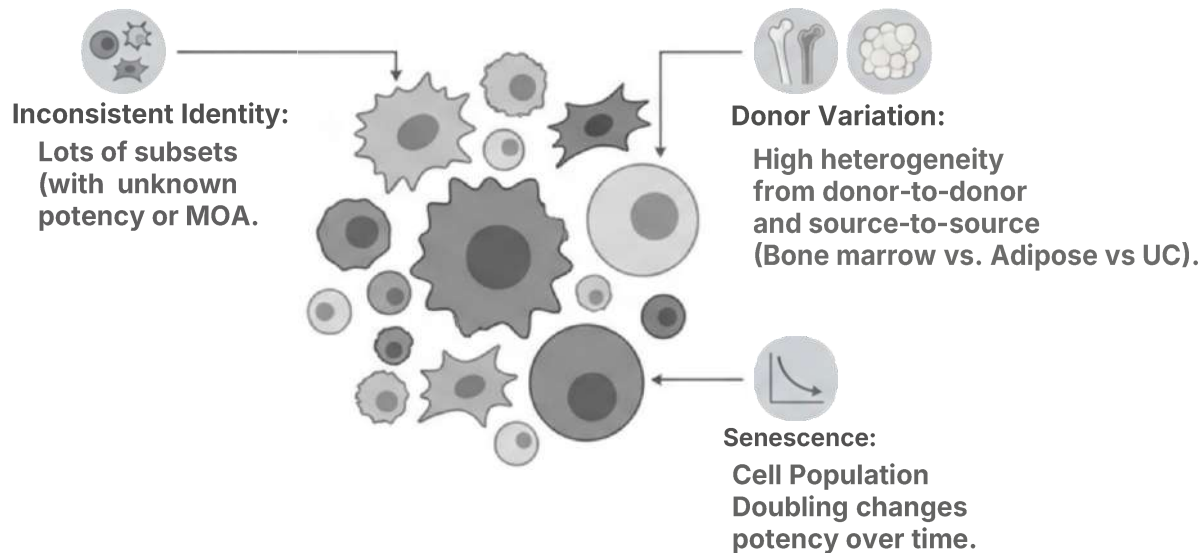
INFECTION





Why Historical MSC Development Failed: The Solutions to Heterogeneity

The Heterogeneity issue



The Solution

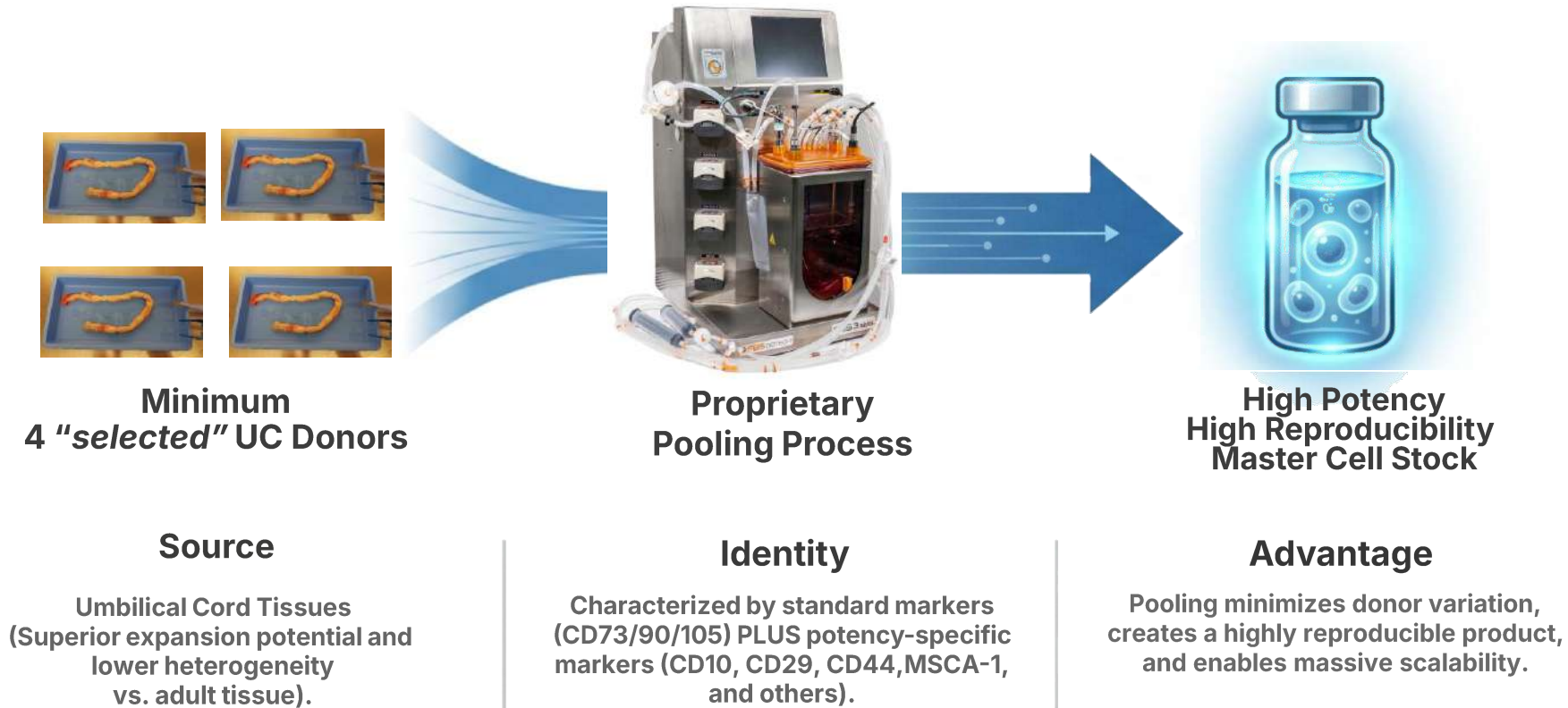
Remove age – UC derived MSC
Reduce donor-to-donor differences – POOL donors
Overcome senescence – UC derived MSC

Reduce cost – close and semi-automate manufacture

UNDERSTAND MECHANISMS OF ACTION



The CORDStrom™ Solution: Engineered Homogeneity Through Identity Selection and Pooling





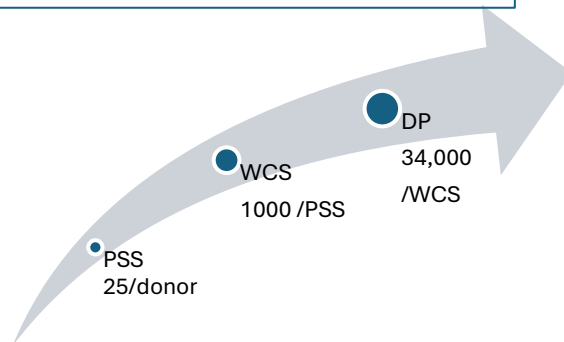
Commercial-scale manufacture complete and future-proofed

Supply Reliability

Available = 80L

1. Phase-dependent, scalable

~100 patients in UK
4 doses/year
45 years from same 4xdonor
stock



Current scale = 3L

In development = 15L



2. Closed Bioprocessing

Closed from MCB to
Final Drug Product



0.5L to 80L



0.2L to 2000L



Automated formulation &
bag filling x21 per
manifold



High-capacity
(75) controlled
rate freezers



Stable long-term
storage



Ebstrocel™: Updated Regulatory Milestones

Completed	Pre-MAA package submitted to MHRA
February 2027	Full UK MAA targeted after MHRA feedback
Mid-2027	Potential UK approval following agency review
Q2 2027	U.S. BLA planned, subject to Type B meeting feedback
European Union	EMA filing prioritized after the FDA application

Company targets, not agency commitments. MHRA response expected 3–6 months after submission, subject to validation, review and requests for additional information. Approval is not assured.



MISSION EB Phase II trial - endpoints

Primary

Safety - MET

Change in EBDASI total score at 3 months – **FAILED**

Secondary

Improvement in Total iscorEB – MET (Intermediate Group)

Improvement in Pain (FACES score) - MET

Improvement in Itch – MET (Itch Man)

Reduction in analgesia - MET

**Improvement in QoL (CHU-9D score) – FAILED @ 3 months
MET @6 months**

Analgesics	Treatment Group	
	Ebstrocel	Placebo
Reduced	13.30%	0%
Remained the same	80%	100%
Increased	6.70%	0%

Qualitative Results

- 13 participants (reporting on 9 cases)
 - 8 parents
 - 5 children
- Completed blinded interview at two time points
 - 3 months post UC-MSCs
 - 3 months post placebo
- 12/13 reported benefits on Ebstrocel
- 3/13 reported benefits on placebo
 - 2 post Ebstrocel
 - 1 treatment naive
- **10/13 correctly identified order of treatment**



UK: complete application replaces rolling submission

Faster CMC progress supports a full MAA in early February 2027.

The revised plan could advance potential UK approval by approximately six weeks.

U.S.: FDA application ahead of EMA

Rare disease regulatory advisors engaged for a Type B meeting request.

Q2 2027 BLA target, seeking traditional or accelerated approval as appropriate to FDA feedback.

“With potential UK and U.S. approvals in 2027, we are approaching a pivotal year. Approval in either or both markets would mark a major milestone for INmune Bio and bring Ebstrocel closer to patients and families living with RDEB.”

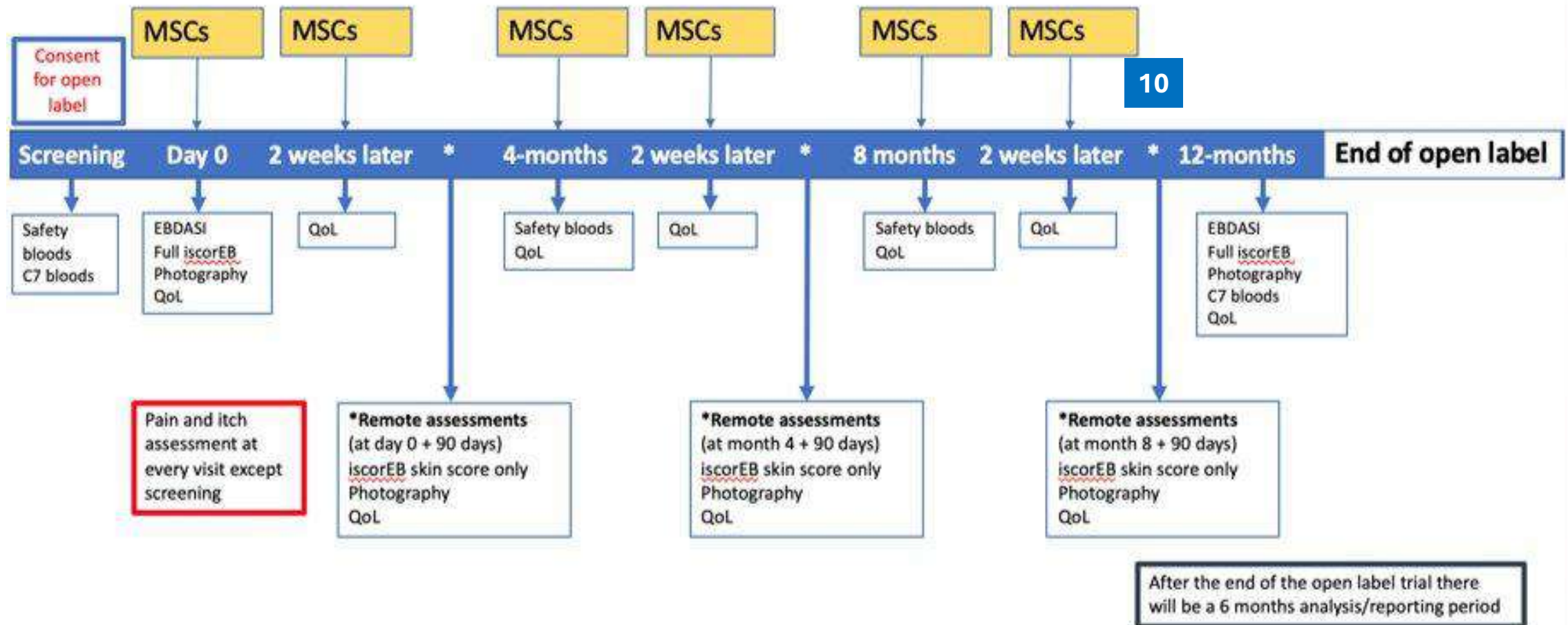
David Moss, Chief Executive Officer

Ebstrocel holds U.S. Orphan Drug and Rare Pediatric Disease designations. Approval pathways and timing remain subject to agency feedback and review.



Phase 3 Open Label Trial – 18 Months

MISSION EB-DELIVER





Market Landscape: Comparison of Therapeutic Approaches

	Vyjuvek (Krystal Biotech, ~\$8B Cap)	Zevaskyn (Abeona)	Filsuvez (Chiesi)	CORDStrom
Mechanism of Action	Gene therapy, topical application	Autologous gene-modified epidermal sheets	Topical gel	Allogeneic mesenchymal stromal cells, intravenous infusion
Route of Administration	Localized topical application to wounds	Surgical grafting to specified wounds	Localized topical application to wounds	Systemic intravenous infusion
Systemic Efficacy	None (Localized effect)	None (Localized effect)	None (Localized effect)	Significant systemic reduction in pruritus and pain
Estimated Cost	~\$750k annually	>\$3M (One-time treatment)	~\$27k (Per treatment course, variable)	~\$700k annually (Estimated)

Vyjuvek = Product of Krystal Biotech, Inc.; Filsuvez = Product of Chiesi Farmaceutici S.p.A.; Zevaskyn = Product of Abeona Therapeutics Inc.

The TAM is an estimate based on internal assumptions and third-party sources, is a forward-looking statement and does not guarantee any future performance. Actual TAM may differ materially. See "Disclaimer" and "Forward-Looking Statements"



Parent Quotes

*When she has been getting ill she has just been recovering in the same time as her brother, normally illness affects her much more because it causes inflammation. We have just been able to cope with everything so much better and have lots of energy and I suppose **life has felt normal***

*I think it's just been lovely to see him having energy and having an appetite , I suppose actually that's the one thing we've noticed as well, **she has put on weight***





Children's Quotes

*It was less painful, when I itch it's painful so obviously that's not comfortable, but **I had less pain, my skin was less itchy**, my hands were less hot and when they are hot I itch. That was not happening that much.*

Normally it would hurt when I have a bath but lately it hasn't been hurting





Quote from a parent:

“If it makes only one per cent difference, then I’m happy to come whenever they call us for the trial because that one per cent isn’t a lot I know, maybe it’s nothing for somebody, but **it’s a lot for him, one per cent less pain for him is one per cent more real life—that’s a lot**”

MISSION EB Team 2024



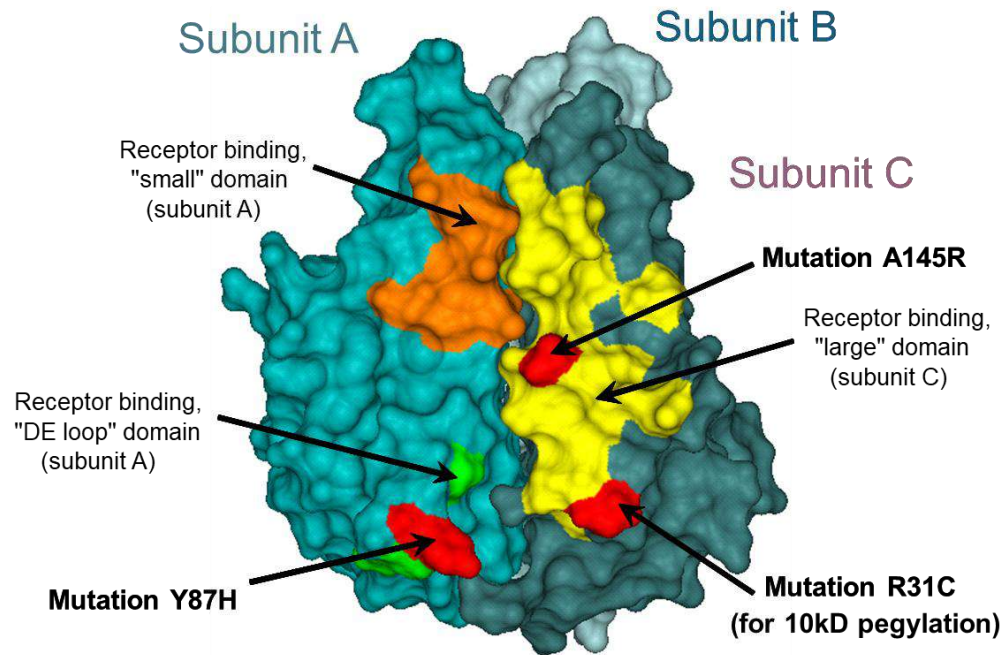
XProTM

Approaching Alzheimer's as an Immunologic Disease





XPro1595 (XPro): Neutralize TNF while Preserving Immune Function



XPro1595 is identical to the human soluble TNF monomer with the exception of mutations in the receptor binding domain and another for pegylation.

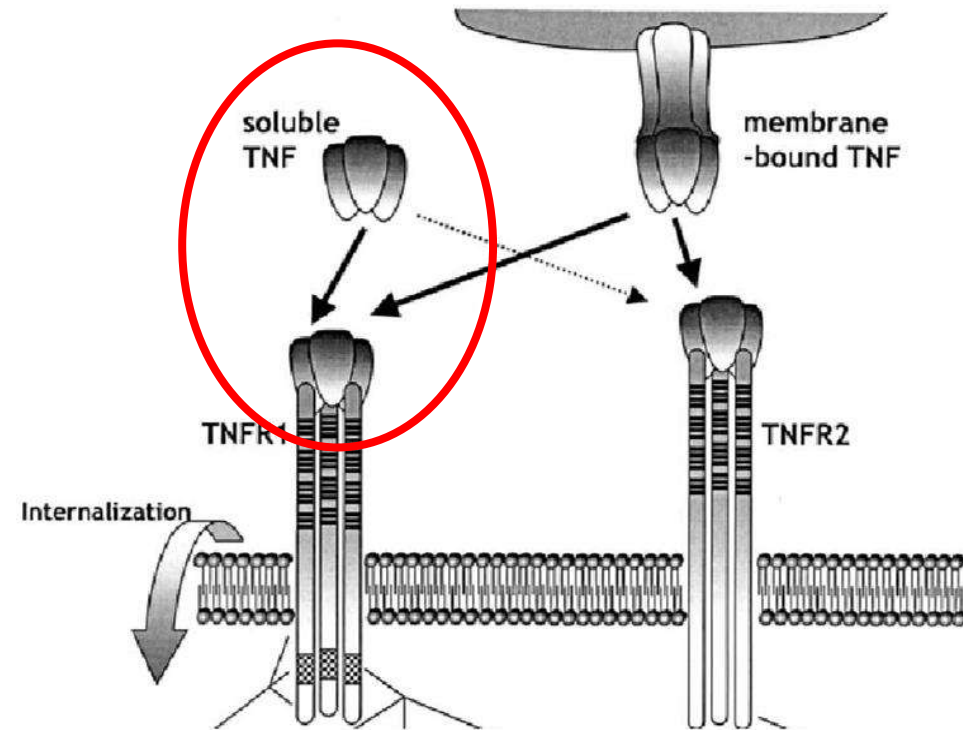
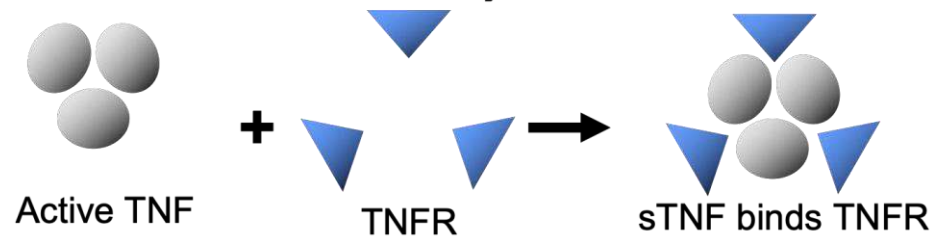
Dominant-Negative in genetics:

"A mutation producing a rogue protein that interferes with the function of the native protein."



Normal Soluble TNF activity

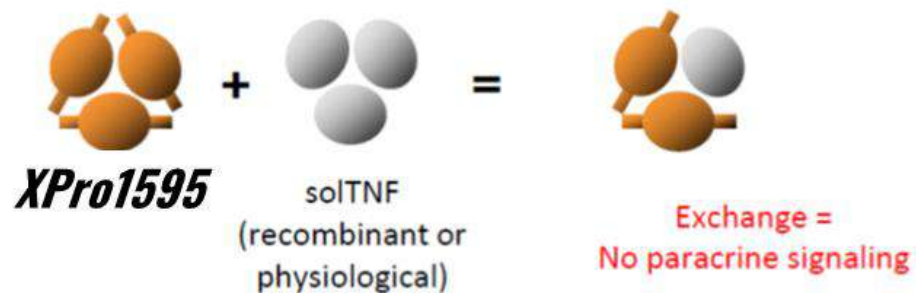
solTNF monomers form active heterotrimers that bind to TNF receptor. Each monomer in the trimer contacts one TNFR subunit





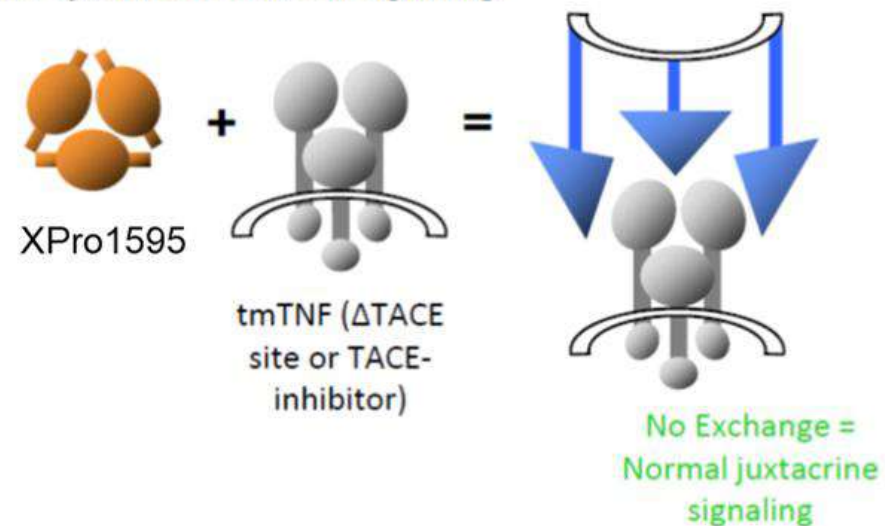
***Xpro1595* freely exchanges with solTNF monomers to form inactive heterotrimers**

Inflammatory soluble TNF eliminated:
No paracrine signaling through receptors



tmTNF homotrimers are anchored to the cell membrane, *XPro1595* cannot exchange

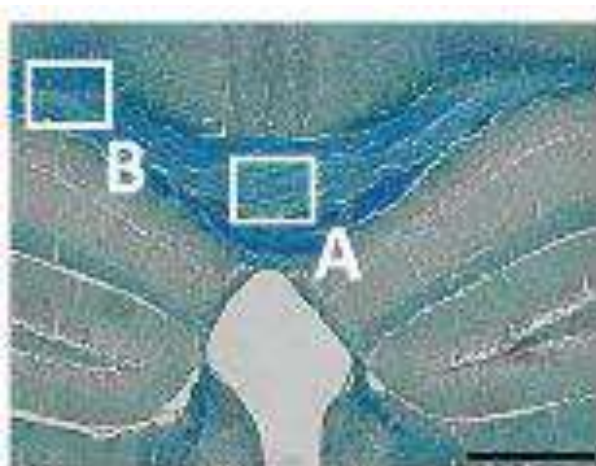
Immunoprotective transmembrane TNF unaffected:
Allows juxtacrine cell-cell signaling



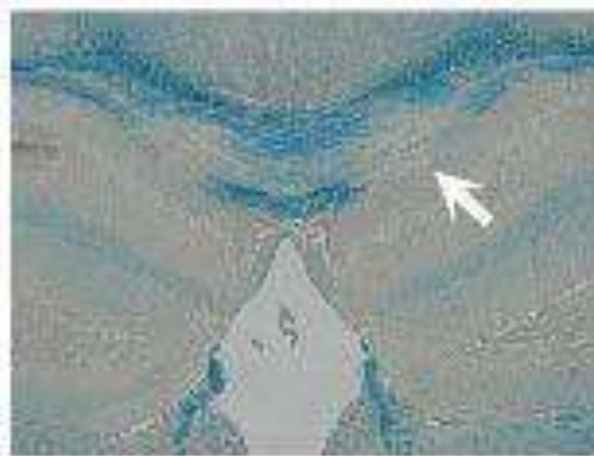


XPro1595 is Neuroprotective

Normal



Cuprizone (Model of Multiple Sclerosis)



Etanercept Anti-inflammatory **AND** immunosuppressive



Exacerbated demyelination

XPro1595 Anti-inflammatory **NOT** immunosuppressive



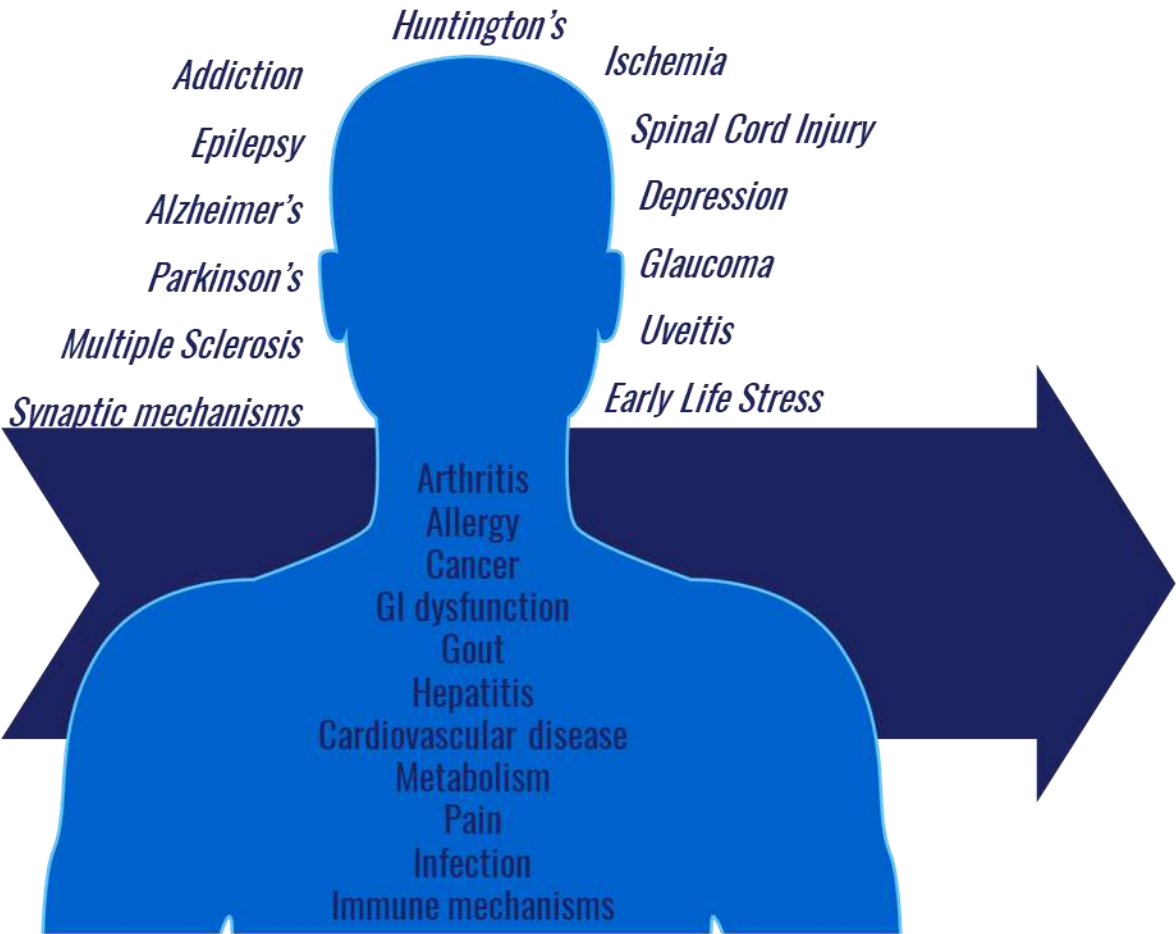
Remyelination

Adapted from: Karamita et al., 2017

XPro1595 Peer-Reviewed Publications: Breadth of Scientific Validation

More Than A Decade of Research Excellence

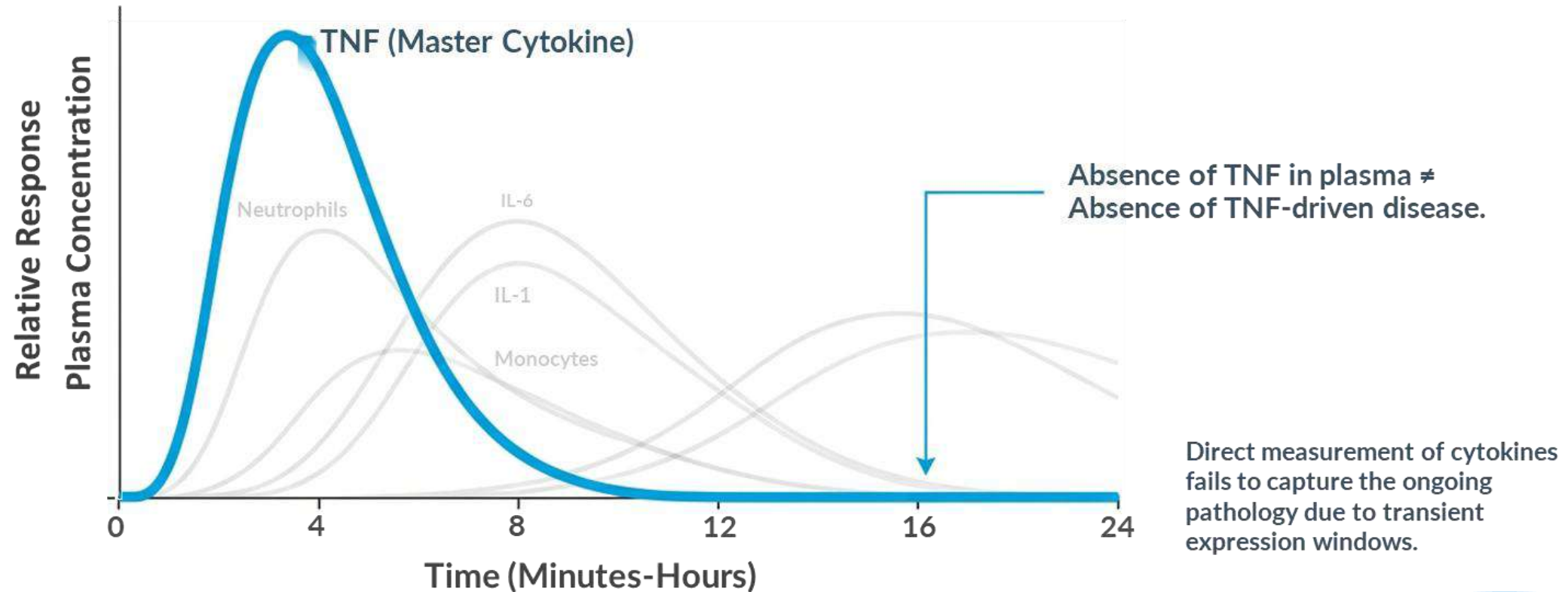
XPro1595's therapeutic potential has been rigorously validated across more than 90 peer-reviewed publications spanning 24 therapeutic areas, 2 dozen independent laboratories, and 3 continents. This extensive body of evidence demonstrates consistent neuroprotective, anti-inflammatory, and disease-modifying effects across diverse pathologies. (See appendix)



>90 Publications
24 therapeutic areas
2 dozen laboratories
3 continents

	Non-selective TNF inhibitors	XPro1595
Decreases inflammation	yes	yes
Immunosuppression	yes	No
Demyelination	yes	No
Neuroprotective	no	yes
Enhances neuroplasticity	no	yes

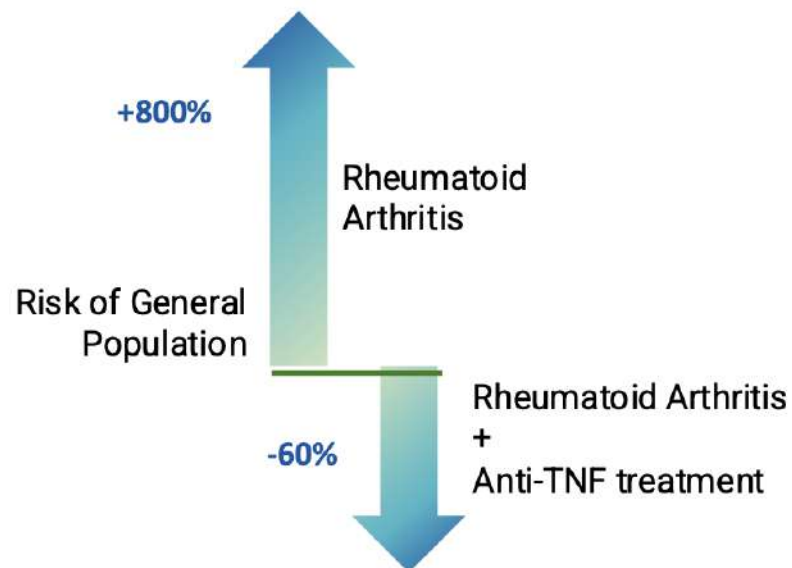
Cytokines are not informative biomarkers of chronic inflammation (Immune Dysfunction)





Strong Evidence for anti-TNF to Treat Alzheimer's Disease

TNF Inhibitors Reduce Risk of Developing AD



Epidemiological studies including a meta-analysis of more than 60 million cases linking **TNF Blocking Agents** to reduced risk of AD

Adapted from PMID: 27470609, 33016914

Evidence linking TNF to AD



TNF increases with Age

TNF levels increase beginning the 3rd or 4th decade of life and correlate with age⁶



TNF increased in AD Patients

Plasma and CSF TNF levels increased in AD patients^{2,3} TNF co-localizes with amyloid plaques⁴ TNF levels correlate with disease progression⁵



TNF causes AD pathology in animals

TNF increases amyloid^{7,8} and Tau⁹⁻¹² TNF causes cell loss and cognitive impairment¹³



TNF inhibitors reduce risk of AD

Anti-TNF therapies¹ reduce the risk of AD in humans by up to:

60%

1. Torres-Acosta N, et al. *J Alzheimer's Dis.* 2020;78:619–626
2. Fillit H, et al. *Neurosci Letters.* 1991;129:318-320
3. Tarkowski E, et al. *J Clin Immunol.* 1999, 19(4):223-230
4. Dickson DW. *J Neuropathol Exp. Neurol.* 1997;56:321-339
5. Paganelli R, et al. *Experimental Gerontology.* 2002;37:257-263
6. Parker et al. *The Journals of Gerontology* (2019) 74(3):283
7. Lahiri et al. *J Alzheimer's Dis.* 2003;5(2): 81-90
8. Blasko et al. *FASEB Journal.* 1999, 13(1):63-68
9. Gorlovoy et al. *FASEB Journal.* 2009, 23(8):2502-2513
10. Montgomery et al. *Am Journal Pathology.* 2013, 182(6):2285-2297
11. Janelins et al. *Am Journal Pathology.* 2008, 173(6):1768-1782
12. Lee et al. *Molecular Med Rep.* 2014, 10(4):1869-1874
13. He et al. *J Cell Biol.* 2007, 178(5):829-841



Biomarker Enrichment Rationale

Matching drug biology to patient · Predictive enrichment using downstream indicators of TNF-driven biology

The Challenge

TNF is the therapeutic target
but cannot serve as the enrichment biomarker.

Ultra-short half-life

TNF peaks in minutes–hours and resolves rapidly. Circulating levels don't reflect tissue or CNS inflammation.

Solution

Measure downstream biomarkers that integrate TNF-associated biology over stable timeframes.



hsCRP

Days to Weeks

TNF → IL-6 → hepatic CRP production

Delayed but stable systemic marker of TNF-driven acute-phase response



ESR

Weeks to Months

TNF → fibrinogen → rouleaux formation

Slow rise, prolonged elevation reflecting chronic inflammatory burden



HbA1c

Weeks to Months

TNF → insulin resistance (JNK/IKK β) → glycemic load

Integrates metabolic consequences of chronic TNF signaling



APOE ϵ 4

Constant

Amplifies TNF production in microglia and astrocytes

Genetic variants (Arg112/Arg158) drive lifelong glial inflammatory priming



≥2 of 4 Composite Design

Captures Multidimensionality · Accommodates Biological Heterogeneity · Reduces Misclassification

11 distinct qualifying biomarker combinations — built-in redundancy against single-assay failure

Enrichment is predictive — identifying patients with TNF-driven disease biology most likely to benefit from XPro1595.
hypothesis-driven precision medicine.



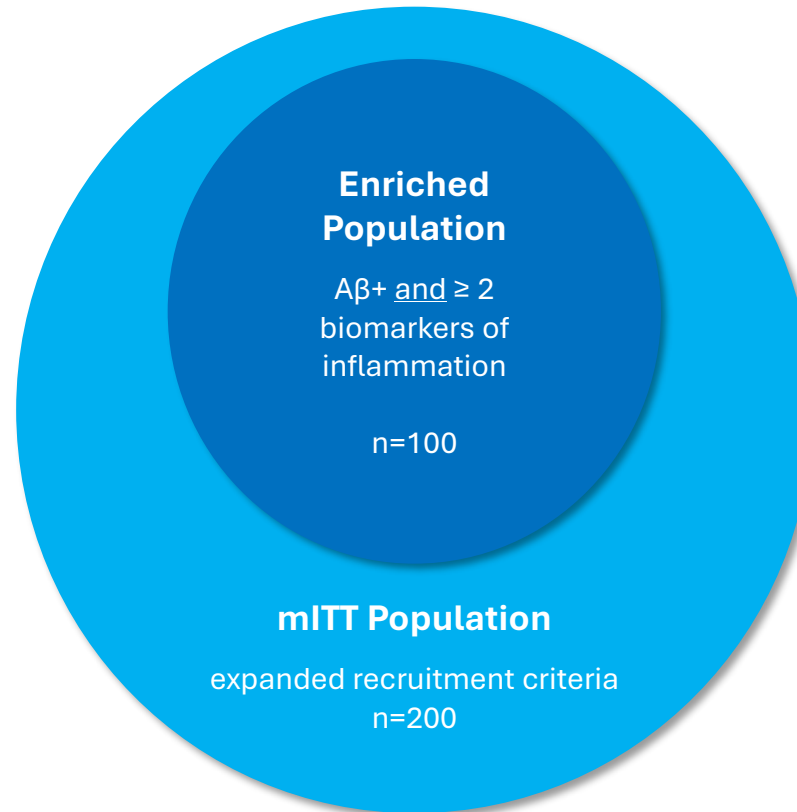
Phase 2: Why Did We Miss P-Value in Cognition (EMACC)? No Placebo Decline in mITT

Original Key Inclusion Criteria: Enriched Protocol

- Age (60 - 85 years)
- Diagnosis of mild AD (NIA-AA stage-4)¹
- Amyloid-beta positive (A β +)
- MMSE ≥ 22
- ≥ 2 blood biomarkers of inflammation

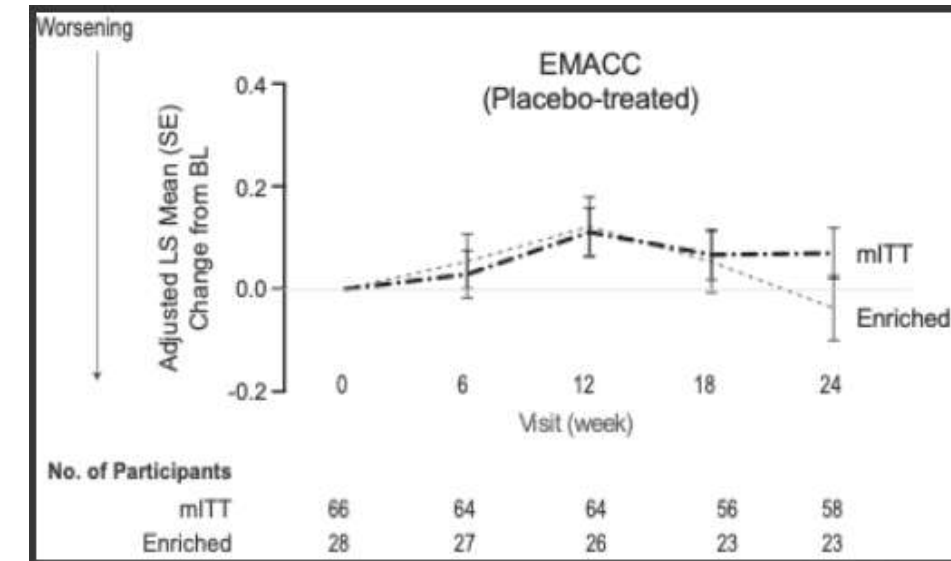
Expanded Inclusion Criteria: Revised Protocol

- Age (50 - 85 years)
- Diagnosis of MCI or mild AD (NIA-AA stages 3-4)¹
- ~~Amyloid-beta positive (A β +)~~
- MMSE ≥ 22
- ≥ 1 blood biomarker of inflammation



To assess a possible treatment effect of XPro1595 compared to placebo, a decrease in cognition within the placebo-treated group over the study period is necessary.

No such decline is observed in the placebo group within the mITT population, but a decline is present in the placebo group among the Enriched population.





Phase 2: Consistent Signal in AD Inflammation Enriched Population

6-month RCT · N = 206 (mITT: XPro n=139, placebo n=67) · ADi enrichment: ≥ 2 of 4 biomarkers AND amyloid positive (n=100)

Overall Population (mITT)

72 yrs median age · 51% female · 45% MCI

68.5% APOE4 carriers — highest ARIA risk population

75% amyloid positive — confirmed AD pathology

62.5% ADi population (≥ 2 inflammatory biomarkers)

ADi = Alzheimer's Disease with Inflammation. Requires ≥ 2 of 4 biomarkers: hsCRP >1.5 mg/L, ESR >10 mm/hr, HbA1C $>6\%$, or ≥ 1 APOE $\epsilon 4$ allele, in addition to confirmed amyloid positivity

Key Findings: ADi Enriched Population

Endpoint	Effect Size	Signal
EMACC (primary) Cognitive composite	d = 0.27	Clinically meaningful
*Chi-separation myelin (imaging)	d = 0.59	Clinically meaningful
Cortical Disarray (PerpPD+) Gray matter (imaging)	d = 0.32	Clinically meaningful
NPI Total Score Neuropsychiatric symptoms	d = 0.23	Clinically meaningful
GFAP (plasma) Astrocyte reactivity	d = 0.19	Directionally supportive
pTau-217 (plasma) Tau accumulation	d = 0.18	Directionally supportive
Goal Attainment Scale Patient-reported outcome	d = 0.18	Directionally supportive
ISRL Delayed recall / memory	d = 0.16	Directionally supportive

*** Statistically significant ($p=0.0098$).** Positive Cohen's d favors XPro1595. $d \geq 0.2$ = small, $d \geq 0.5$ = med-large; but clinically meaningful signal threshold; $d < 0.2$ = directionally supportive. ADi population (amyloid positive AND ≥ 2 of 4 inflammatory biomarkers, $n \approx 100$).

Dose-compliant analyses show increased effect sizes for NPI, pTau-217, and GFAP, Chi-separation — effects attributable to greater XPro1595 exposure



Differentiated Safety Profile

No ARIA · Immune Function Preserved · SAEs Lower on Drug

0

ARIA Events

Despite a high-risk population:
68.5% APOE4+ · 34% with microbleeds
27% on antithrombotic

Serious & Severe AEs Lower on Drug

Both serious and severe events were less frequent on XPro1595 than placebo. Severe events 3× more frequent on placebo. Zero fatal events across 206 patients.

Placebo infection rates

2× higher

XPro1595 preserves systemic immune function
— selectively inhibiting inflammatory TNF (sTNF)
while preserving protective TNF (tmTNF)

ISR & Blinding Considerations

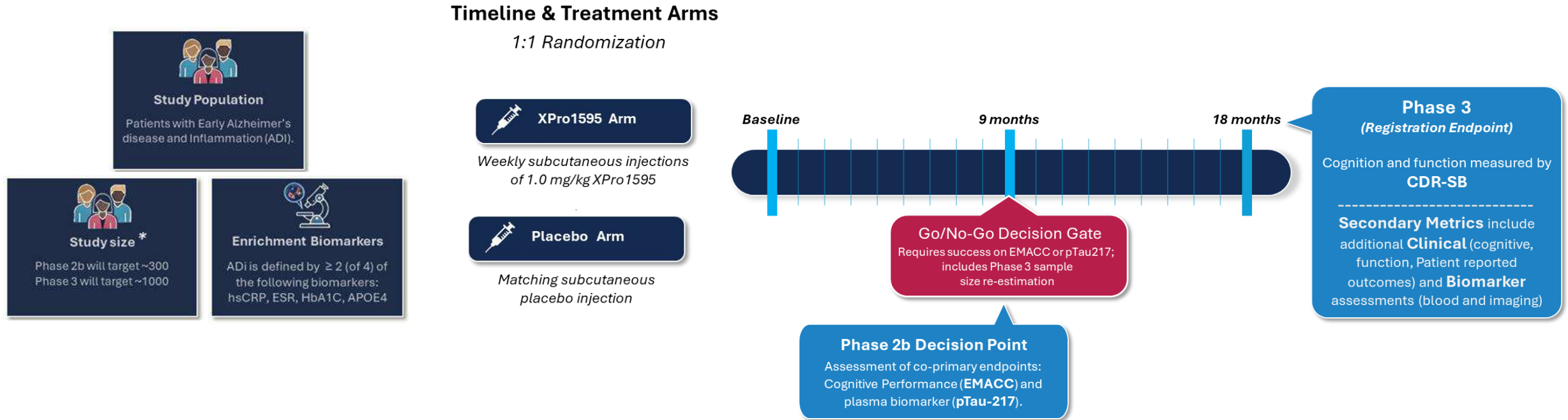
ISR rates higher on drug (79%) — majority were mild and transient, characterized by an independent safety board as "more nuisance than safety." Blinding strategy addressed in Phase 3 design.

Safety profile supports Phase 3 advancement — predictable, manageable, with no unexpected signals



Phase 2b/3 Adaptive Trial Design — FDA Aligned

FDA End-of-Phase 2 Meeting Completed Q1 2026; Fast Track Granted Q2 2026



FDA Alignment

✓ Enrichment

ADI biomarker strategy endorsed

✓ Design

Adaptive 2b/3 with decision gate accepted

✓ Endpoint

CDR-SB as sole Phase 3 primary

✓ Exploratory

Non-enriched cohort recommended

Summary represents sponsor interpretation of FDA feedback.



CDR-SB Confidence: Why 18 Months Will Work

EMACC detects subtle cognitive changes- CDR-SB measures the consequences.

- ✓ Phase 2b: 3 more months + 3× sample
→ more time for placebo decline
- ✓ Anti-amyloid precedent: CDR-SB separation emerged at 18 months
- ✓ We have the early signal.
Now we need the time.



EMACC — Cognitive Sensitivity

What it measures

Measures subtle cognitive changes — word recall, fluent speech, processing speed — too subtle for conventional clinical scales

Why it detects change early

Captures early changes such as slower performance or fewer words recalled in individuals that remain independent - A “*Stress Test for the brain*”

MINDFuL Phase 2 (ADi, 6 months)

d = 0.27



CDR-SB — Functional Impact

What it measures

Clinician and caregiver-rated assessment of whether cognitive deficits interfere with daily activities — appointments, finances, conversations

Why it requires more time

Broad scoring categories (none, slight, mild) don't reflect small changes. Requires observable change in day-to-day activities.

Phase 3 Registration (ADi, 18 months)

Primary endpoint for FDA approval

The Confidence Bridge: In external AD cohorts, correlation between cognitive composites and CDR-SB strengthens over time:

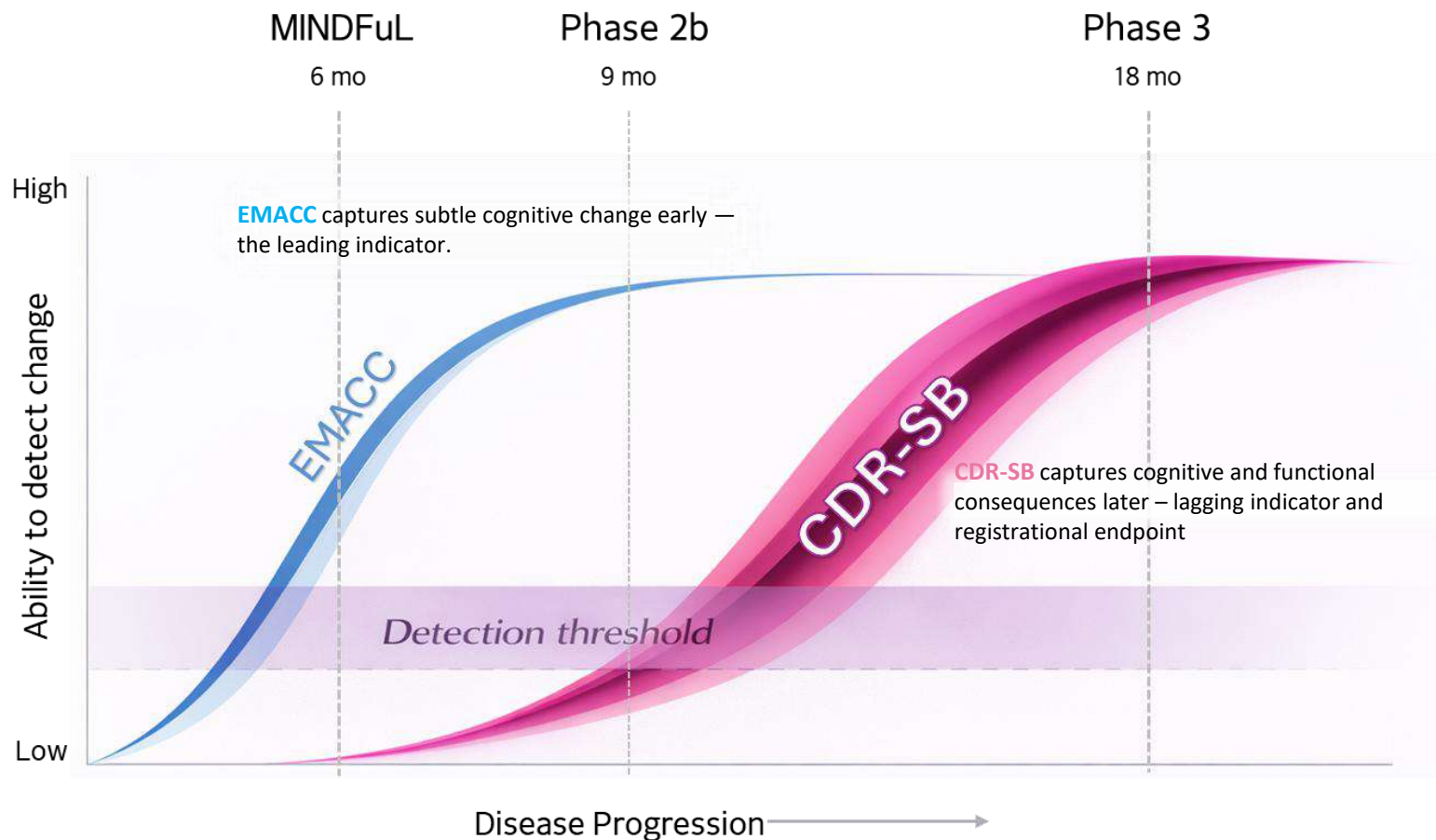
r = 0.41 (early) → r = 0.65 (18 months).

Cognition precedes function — the same temporal pattern seen in lecanemab and donanemab.



Why CDR-SB Requires More Time: Evidence from External AD Cohorts

Enough Cognitive Change Must Occur to Measure Functional Change



How to Read This Chart

Y-axis: Ability to reliably separate drug from placebo.

X-axis: Time / disease progression.

Horizontal band: Detection threshold — where enough signal accumulates to measure a difference.

EMACC (blue curve)

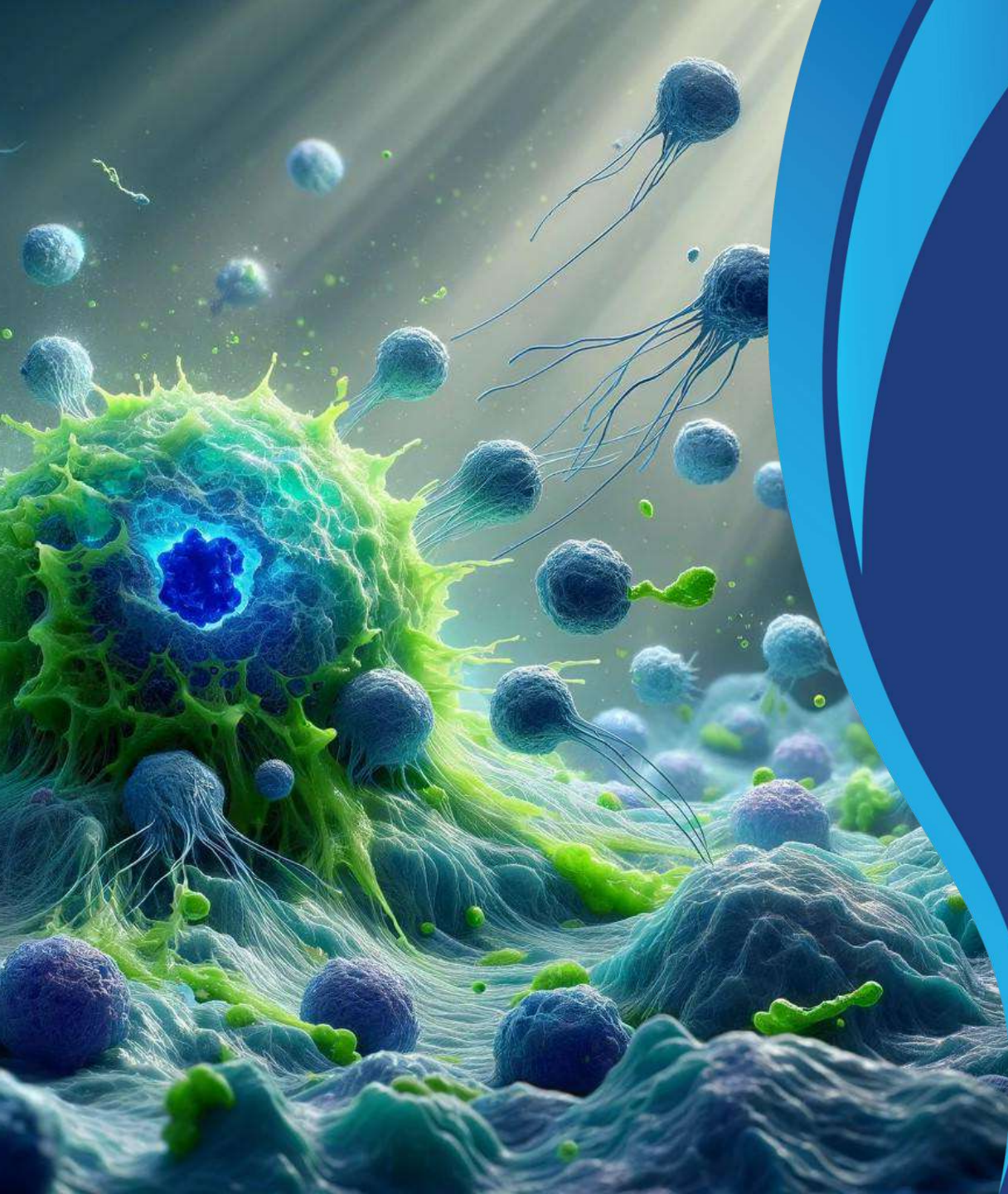
Cognitive composite measured on a continuous scale. Crosses threshold early — detected treatment signal at 6 months ($d = 0.27$).

CDR-SB (magenta curve)

Broad scoring categories need larger functional change to register. Crosses threshold much later when daily activities are impacted.

Key: Same pattern as lecanemab and donanemab — cognitive separation first, functional later.

Conceptual framework. Curves are illustrative — not derived from trial data.



Thank You!

INmune Bio Inc.

225 NE Mizner Blvd. Ste. 640

Boca Raton, FL 33432

+1 561 710 0512

info@inmunebio.com

Investor Relations:

Daniel Carlson

+1 415 509 4590

dcarlson@inmunebio.com