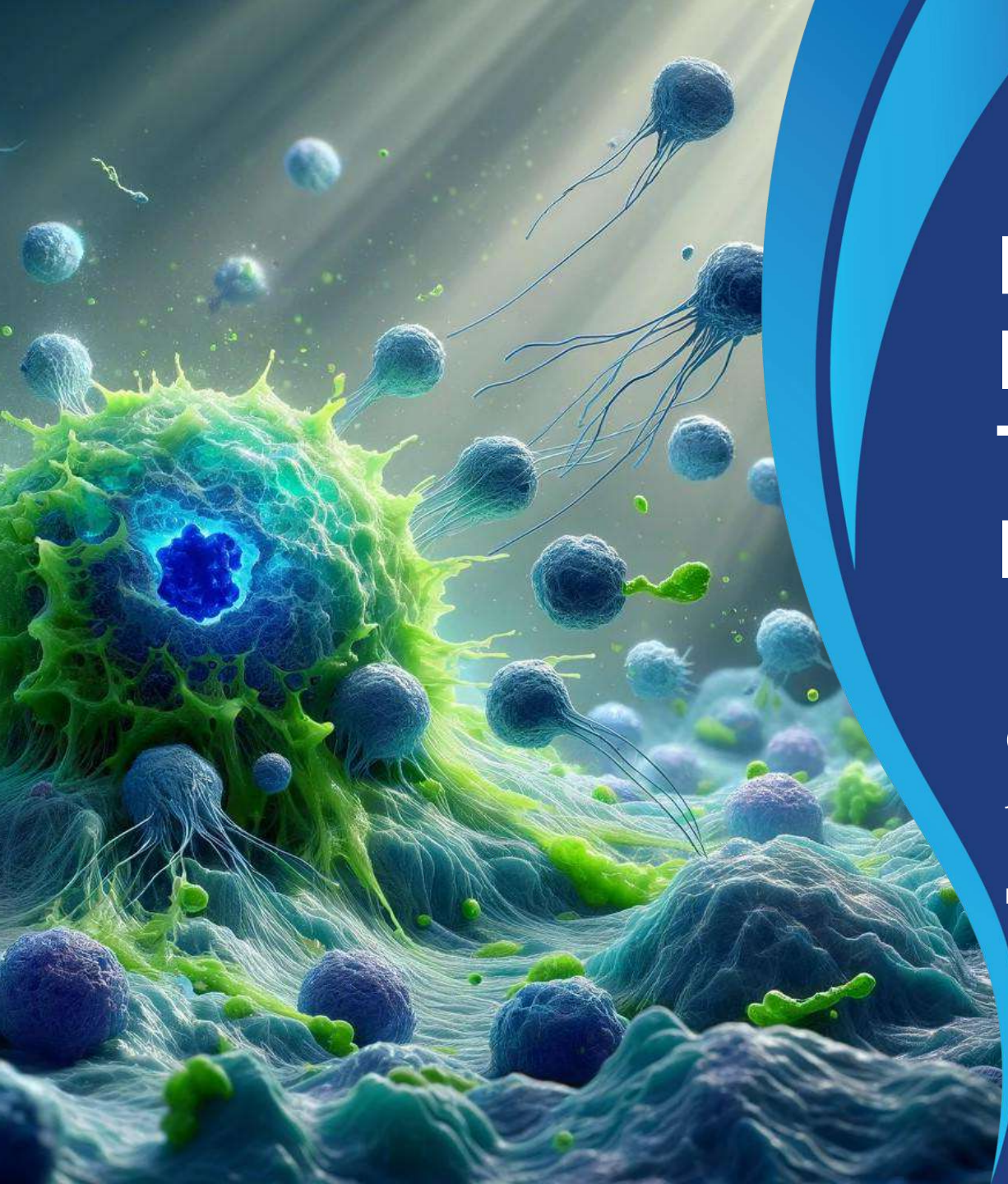

Modulating the Innate Immune System to Transform Inflammation-Driven Disease

Corporate Presentation

June 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.

Late-Stage Pipeline Across Inflammation & Immunology

- **Two Late-Stage Assets:** Leading with CORDStrom™ (RDEB) and XPro1595™ (Alzheimer's), both entering regulatory/pivotal phases.
- **Near-Term Commercialization:** CORDStrom™ targeting MAA filing (UK/EU) in Q3 &Q4 -2026 and BLA (US) in late 2026.*
- **Regulatory-De-Risking:** RPDD + ODD received (RDEB). FDA alignment on XPro Phase 2b/3 design confirmed.
- **Capital Efficiency:** Clear path to Priority Review Voucher (PRV) monetization to fund operations.
- **Upside:** Proprietary innate immunology platforms, CORDStrom™ + XPro™, addressing multi-billion-dollar markets (Rare Disease + Neurodegeneration).

CORDStrom™

XPro1595™

Program	Indication / Patient Pop (US&EU)	Pre-clinical	Phase 1	Phase 2	Phase 2b/3
XPro™ / Neuro	Early Alzheimer's Disease with Infl. (9M)	→	→	→	FDA Alignment on Phase 2b/3 Clinic Ready in 2027

*Estimated dates subject to change – See "Forward-Looking Statements"

Key Patents & Exclusivity Position

CORDStrom™

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International Bureau

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WIPO | PCT

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(74) Agent: **SCHOONOVER, Joshua**; 225 NE Mizner Blvd., STE 640, Boca Raton, Florida 33432 (US).

(81) Designated States *(unless otherwise indicated, for every kind of national protection available)*: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BN, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CV, CZ, DE, DJ, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IQ, IR, IS, IT, JM, JO, JP, KE, KG, KH, KN, KP, KR, KW, KZ, LA, LC, LK, LR, LS, LU, LY, MA, MD, MG, MK, MN, MU, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PA, PE, PG, PH, PL, PT, QA, RO, RS, RU, RW, SA, SC, SD, SE, SG, SK, SL, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UY, UZ, VC, VN, WS, ZA, ZM, ZW.

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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 (hucMSCs) 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

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

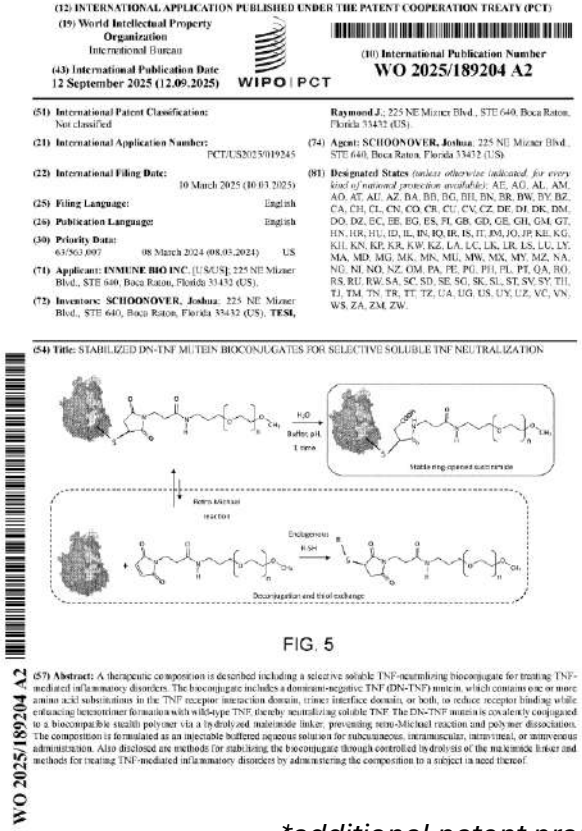
Nominal Patent Coverage thru 2045

Eligible for PTE up to
+5years on approval

**additional patent properties pending*

Eligible for Ref. Biol. Product Exclusivity (~12 years)
ODD Exclusivity (RDEB) ~7years

XPro1595™



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(74) Agent: **SCHOONOVER, Joshua**; 225 NE Mizner Blvd., STE 640, Boca Raton, Florida 33432 (US).

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

(57) Abstract: A therapeutic composition is described including a selective soluble TNF-neutralizing bioconjugate for treating TNF-mediated inflammatory disorders. The bioconjugate includes a dominant-negative TNF (DN-TNF) mutein, 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 heterotrimer formation with wild-type TNF, thereby neutralizing soluble TNF. The DN-TNF mutein is covalently conjugated to a bioconjugable stealth polymer via a hydrolyzed maleimide linker, preventing retro-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 maleimide linker and methods for treating TNF-mediated inflammatory disorders by administering the composition to a subject in need thereof.

FIG. 5

WO 2025/189204 A2

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

**additional patent properties issued/pending*

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

INmuneBio

CORDStrom™ (*pobistrocel*)

for EB

Beyond Topical Care: Disease-Modifying Systemic Therapy

The Progressive Nature of RDEB

18 Months to 10 Years: The Critical Therapeutic Window

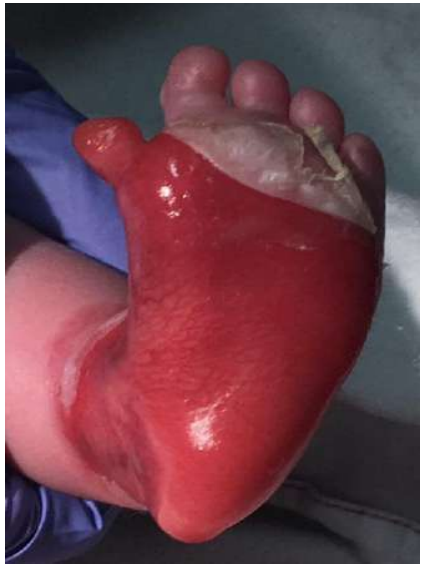
Journal of
Dermatology and Venereology

ScienceAssist
International

Prospective | Open Access

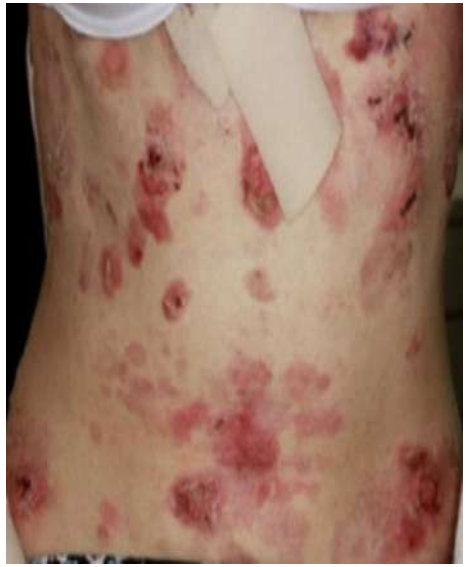
The Natural History of Severe Recessive Dystrophic Epidermolysis Bullosa – 4 Phases Which May Help Determine Different Therapeutic Approaches

Bageta ML[1*], Yerlett N[2], McGrath JA[3], Mellerio JE[4], Petrof G[1], Martinez AE[1]

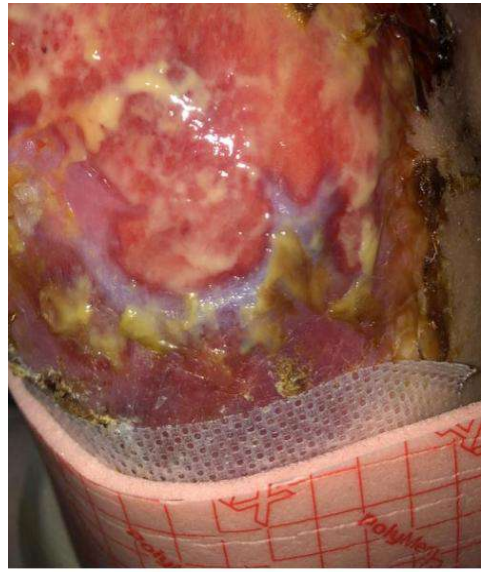


0-18 months

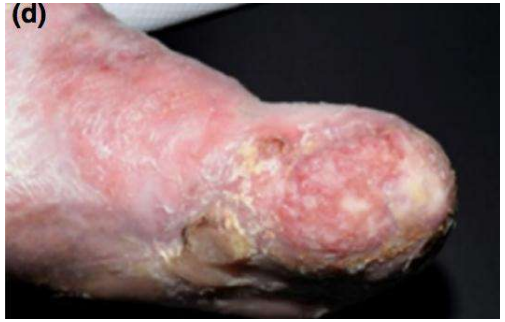
18 months-10 years



10-20 years

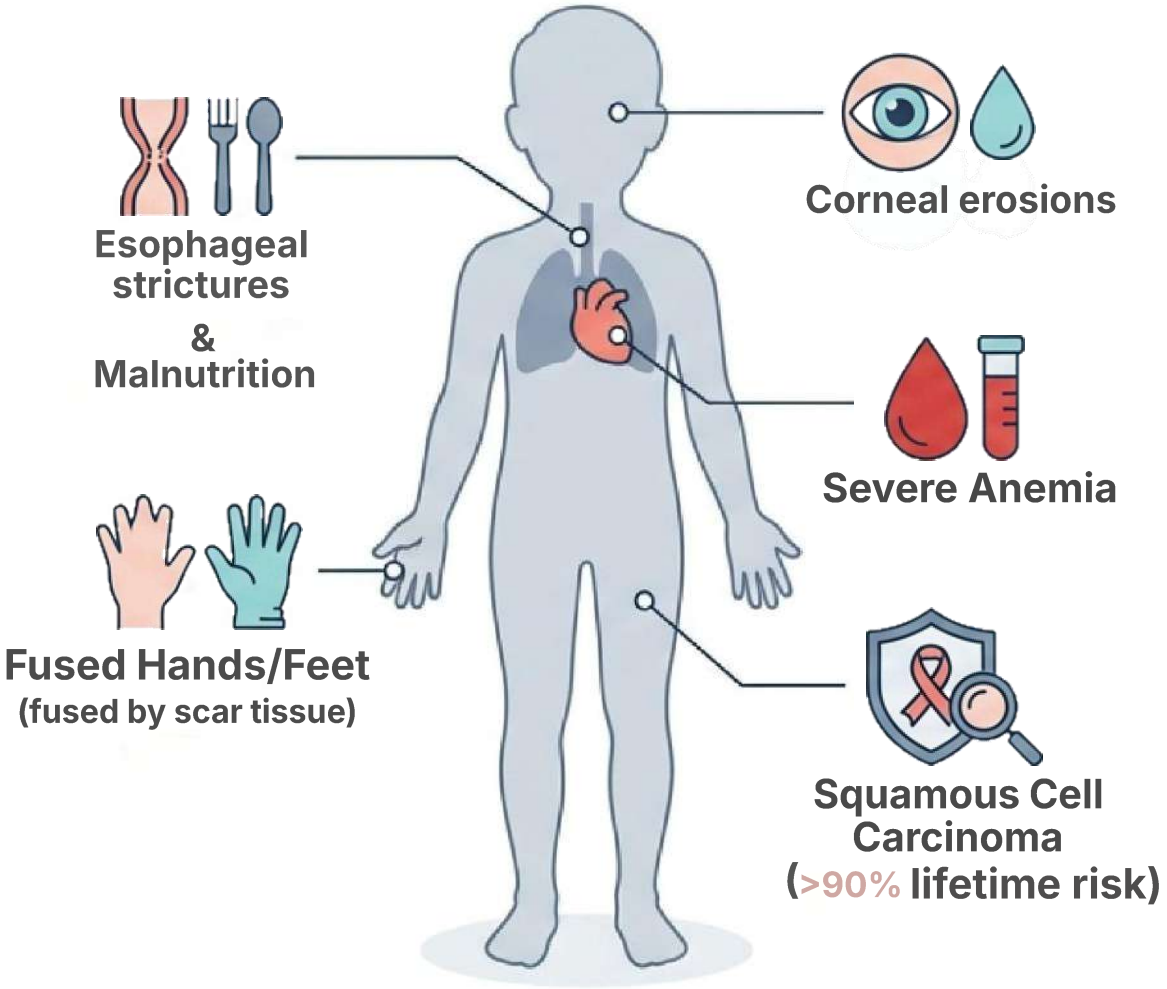


Over 20 years



Squamous cell carcinoma

The Burden of RDEB is Systemic and Life-Limiting



Squamous Cell Carcinoma (SCC) is the leading cause of death (84% mortality by age 40).

Aligning with the FDA's Patient-Focused Guidance

Regulatory Insight

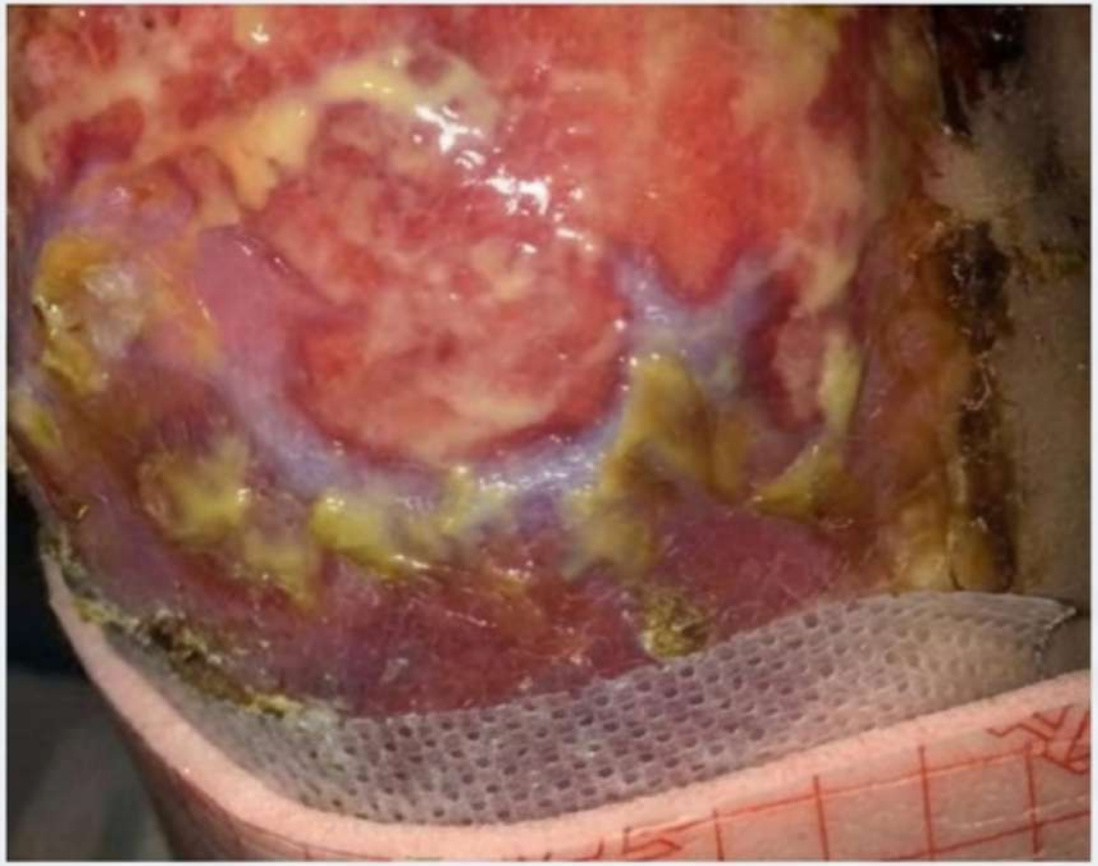
The FDA acknowledges that reduction in Itch and Pain are valid clinical endpoints for RDEB, moving away from 'wound surface area' as the sole metric. Our Phase III data focus is optimized for this patient-centric regulatory reality.

https://www.youtube.com/live/Ids1WAe5Czo

https://academic.oup.com/ced/article/50/6/1125/7985666

CORDStrom™: A Systemic Disease Requires a Systemic Solution

Clinical Manifestation of RDEB



Severe, non-healing wounds characterize RDEB.

- **The Disease: Recessive Dystrophic Epidermolysis Bullosa (RDEB).** A devastating, ultra-rare pediatric disease.
- **The Reality:** RDEB is not just skin blisters. It is a systemic failure.
- **Systemic Symptoms:**
  - Chronic internal wounds;
  - Mucosal involvement (Esophagus, Eyes);
  - Fusion of fingers and toes;
  - Severe chronic pain and itch
- **The Gap:** Current standard of care (creams/bandages) and competitors only treat accessible skin. They fail to address the internal systemic pathology that drives mortality.

(Source: FDA PFDD Meeting)
https://www.youtube.com/live/Ids1WAe5Czo

The Unmet Need: The “Itch-Pain” Cycle Drives Disease Progression

Patients identify Pruritus (Itch) as their #1 or #2 concern, driving disease progression.

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

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

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

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

(Source: FDA PFDD Meeting)
https://www.youtube.com/live/Ids1WAe5Czo

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

Global EB Priority Setting Partnership

identifies research priorities for the four main types of epidermolysis bullosa

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

Study Details

- Design: Randomized, double-blind, placebo-controlled.

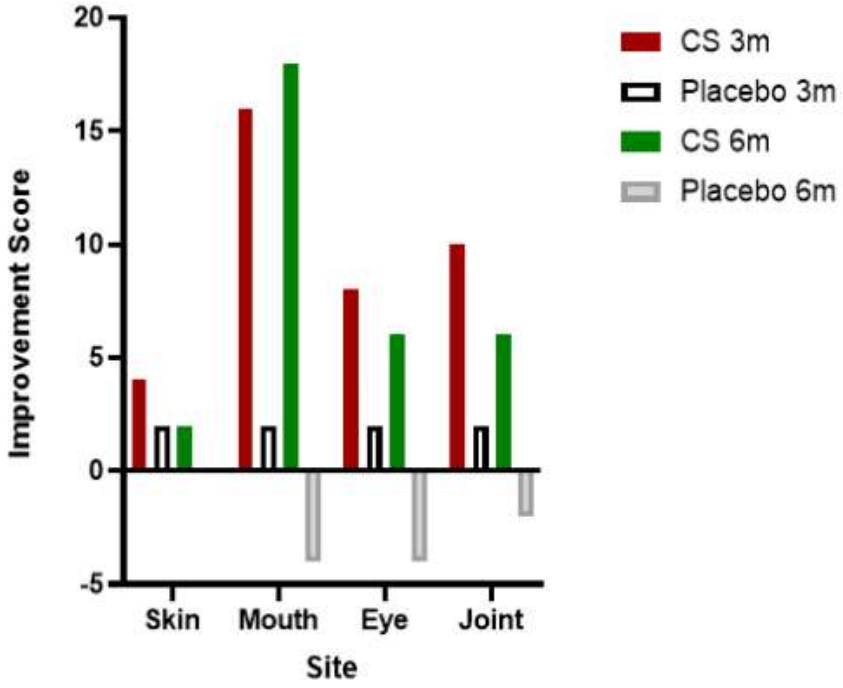
- Population: Children with Recessive Dystrophic Epidermolysis Bullosa.

- Dosing: Intravenous Stromal cell Infusions (Systemic delivery).

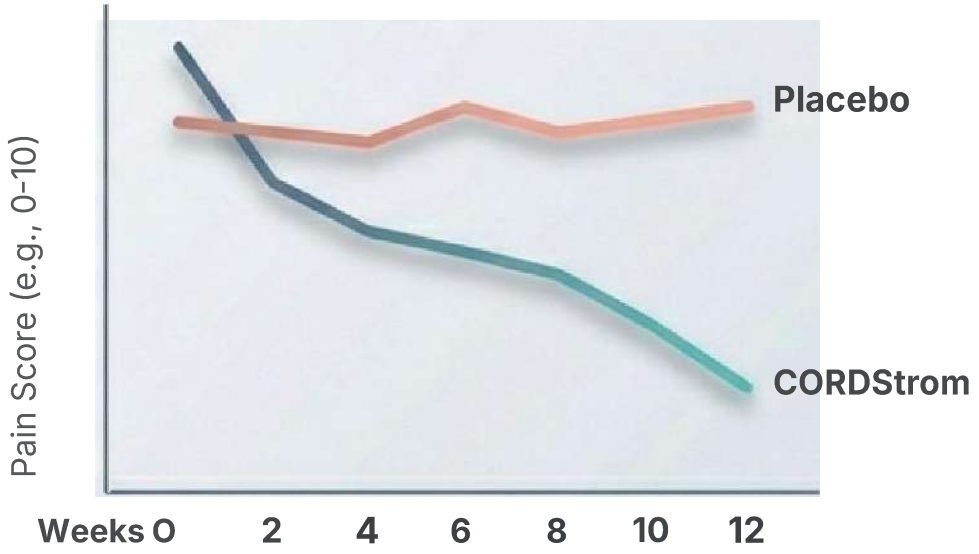
Comprehensive Organ System Improvements*

ISCOREB Improvement Scores*

Pain Indicies



Pain Scores



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

* Period 1 at 3 and 6 months

Comprehensive Organ System Improvements

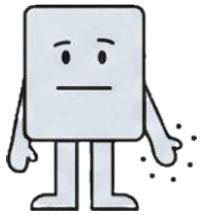
Itch Man

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

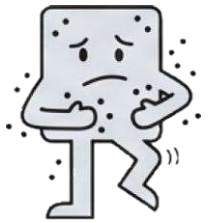
I just wait for the sun to come up so I can start the cycle of bandaging the damage I did in my sleep."

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

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



Level 2: Itches more; sometimes interferes with activity

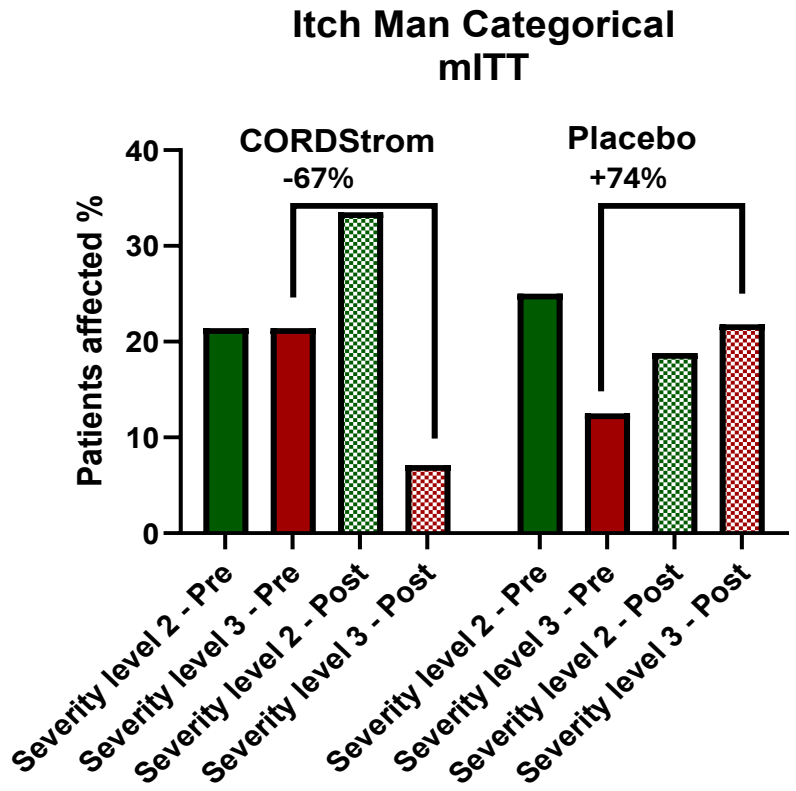


Level 3: Itches a lot; difficult to be still, concentrate

Month 3 - Itch Man Scale improved 17%

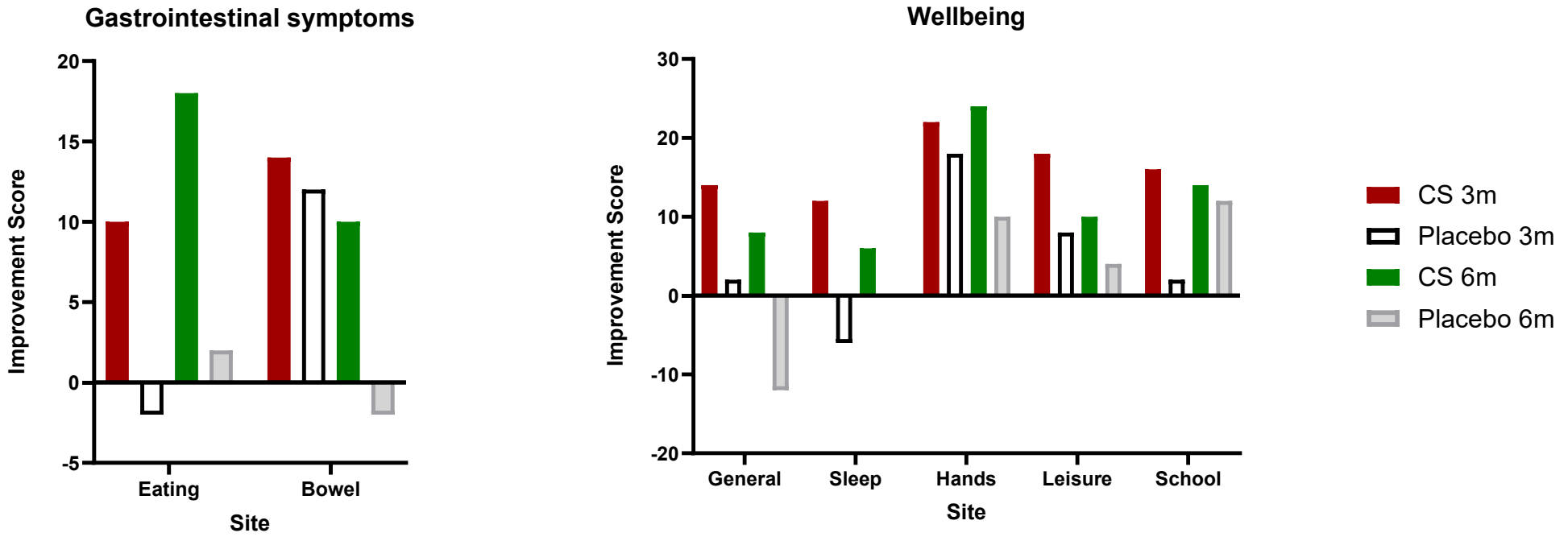
Month 6 - Itch Man Scale improved **27.5%**

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



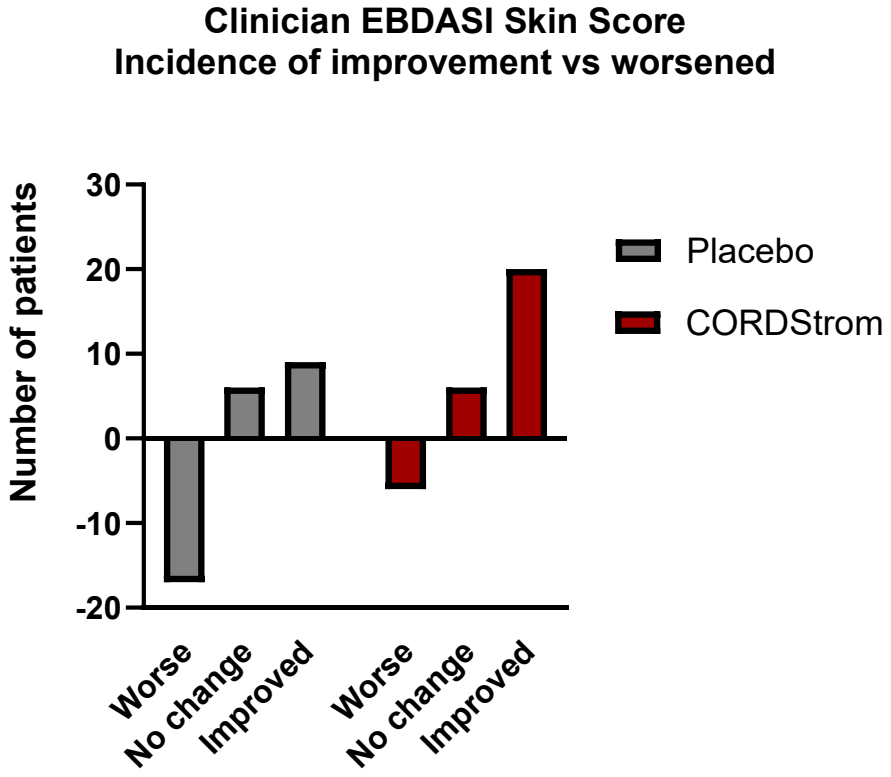
Comprehensive Organ System Improvements

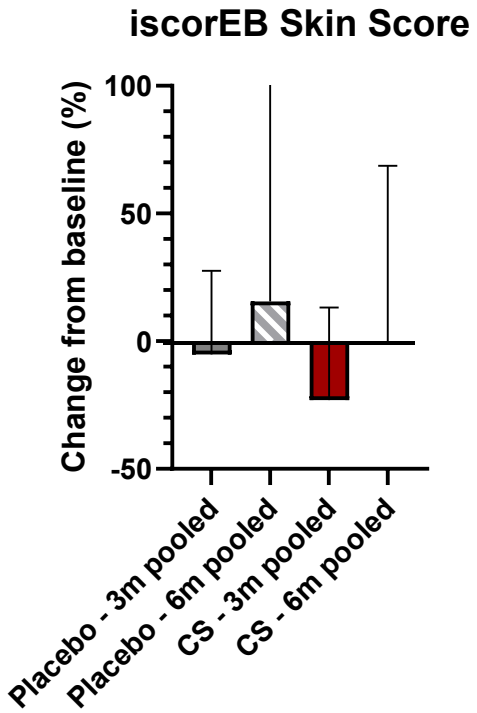
iscorEB Improvement Scores

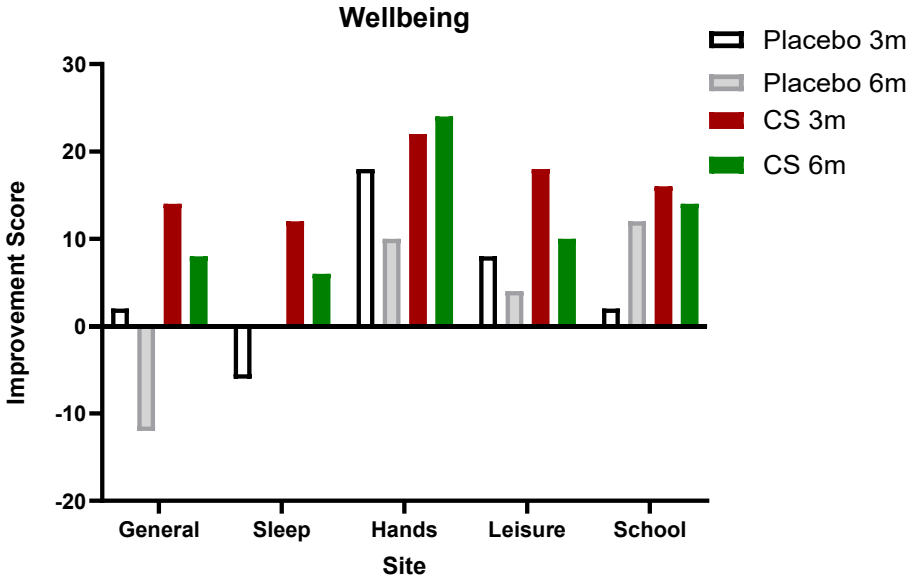


Comprehensive Skin Quality Improvements*

iscoreEB & EBDASI Improvement Scores*







<u>CORDStrom is potentially the first systemic therapy with itch and pain relief benefits as key differentiation factors in quality of life.</u>

* Period 1 at 3 and 6 months

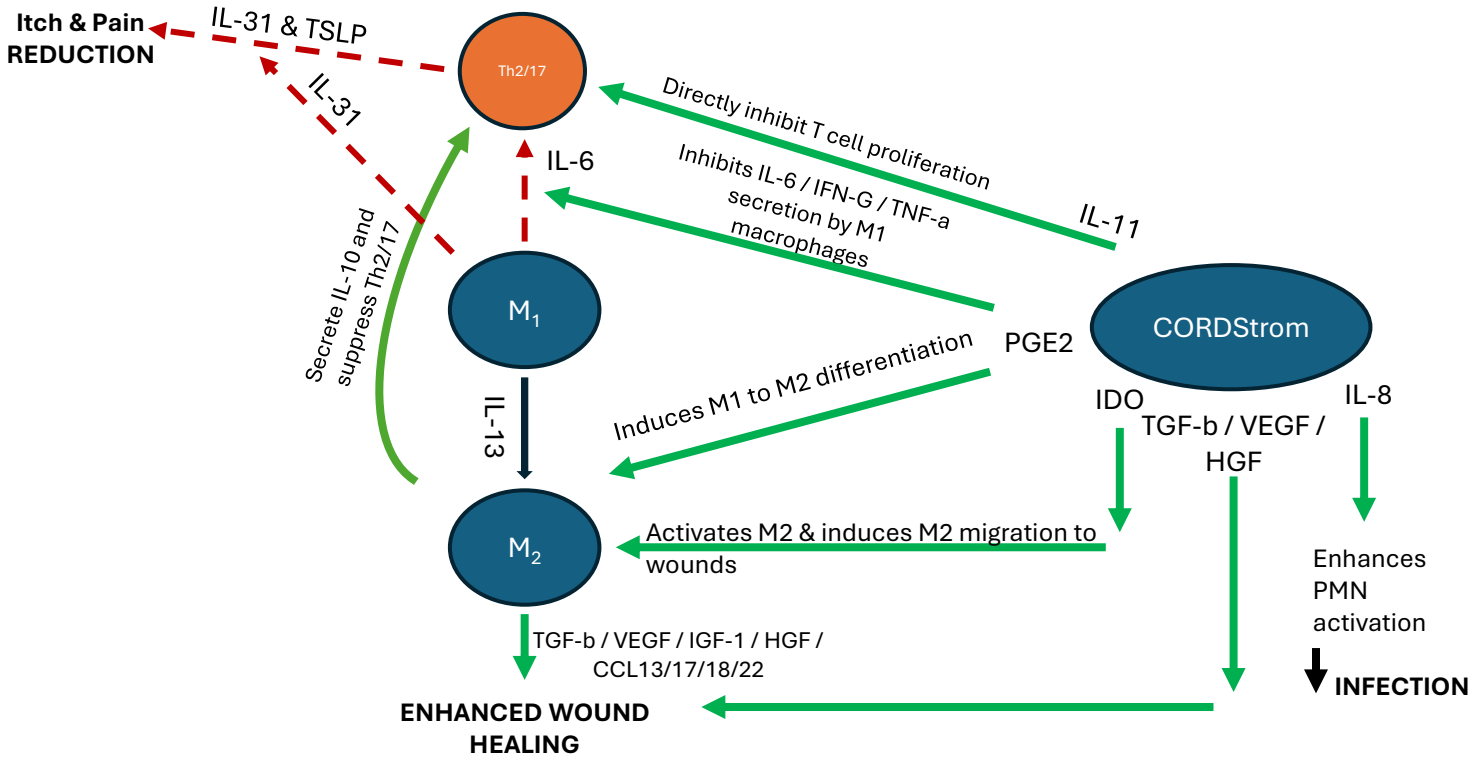
Overall Summary

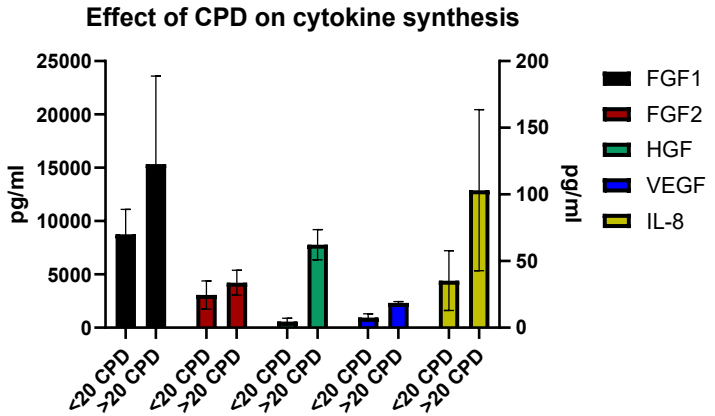
- CORDSTROM has proven to be beneficial treatment with **no safety signals** in children with RDEB from the age of 6 months
- **Intermediate and younger patients** under the age of 10 years saw earlier effects with **improvement of their skin, pain and itch**
- Although the **most severe and older patients** did not show changes in their skin at 3 months, by 6 months their **itch and skin scores had improved greatly**
- ***"Treatment with CORDSTROM 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

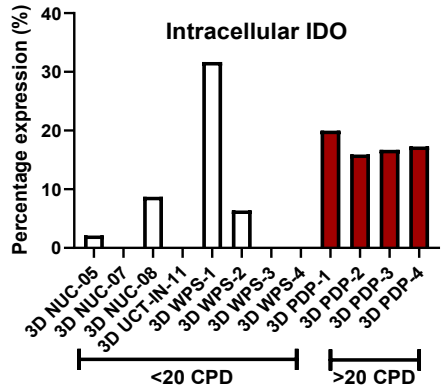
Standards for Conditional Drug Approval

- **Core Efficacy Requirements (MHRA, EMA & Similar Frameworks)**
  - **Positive Benefit-Risk Balance:** Data must indicate that the ***drug's benefits outweigh its risks***, even if the data are less comprehensive than required for standard marketing authorisation.
  - **Proof of Concept/Partial Data:** Evidence must demonstrate a "reasonable prospect" of efficacy and safety, ***often based on early Phase II or Phase III trial results***.
  - **Unmet Medical Need:** The medicine must address a condition where ***no satisfactory treatment***, prevention, or diagnosis exists, or offer a "major therapeutic advantage" over existing methods.
  - **Substantial Evidence Expected:** Although data are ***"less" comprehensive, it must still be robust enough to support a positive benefit-risk assessment***.

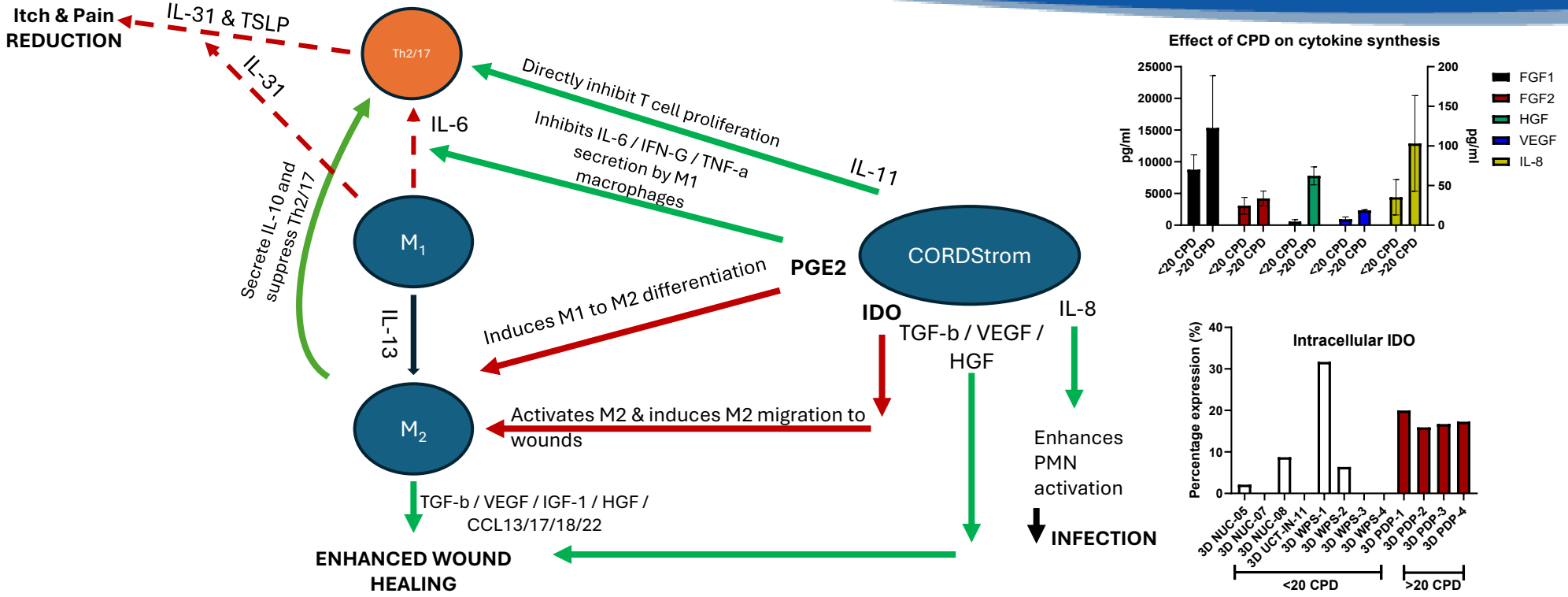
CORDStrom™ Mechanisms of Action – IDO/PGE2 to M1 to M2 Differentiation

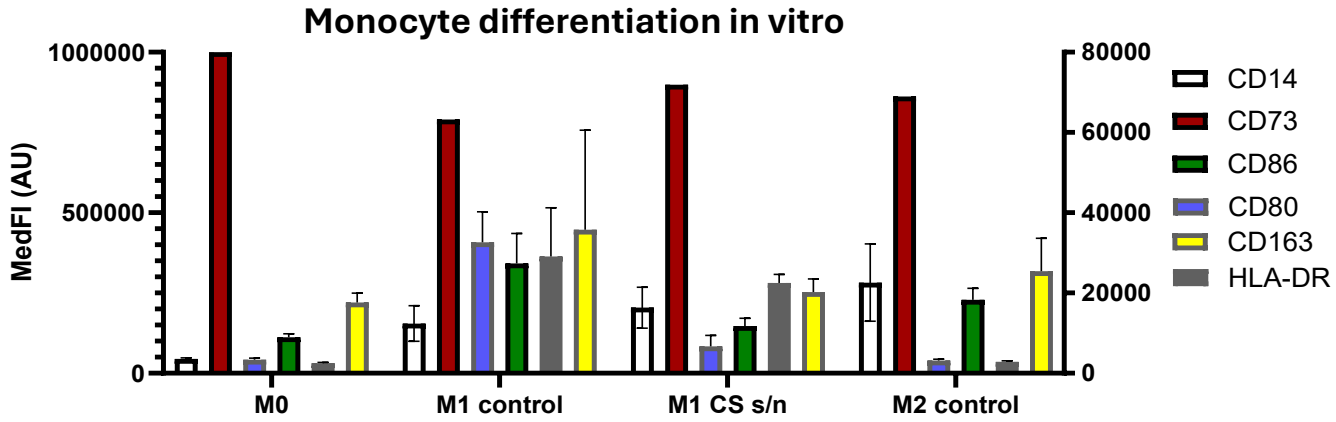






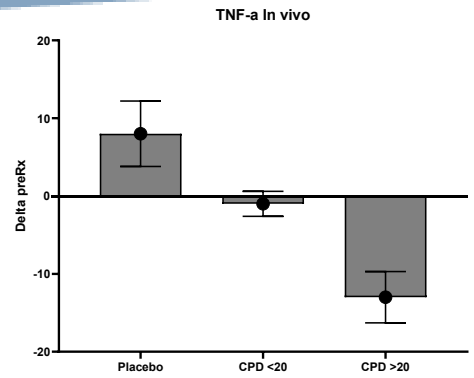
CORDStrom Mechanisms of Action – IL-31 Reduction

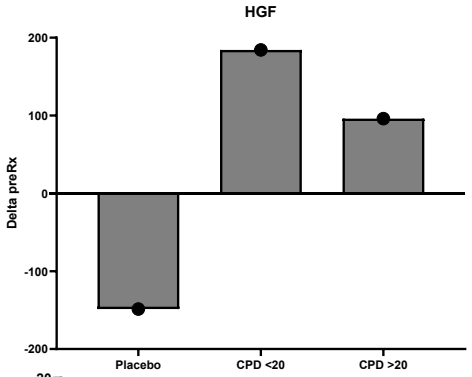


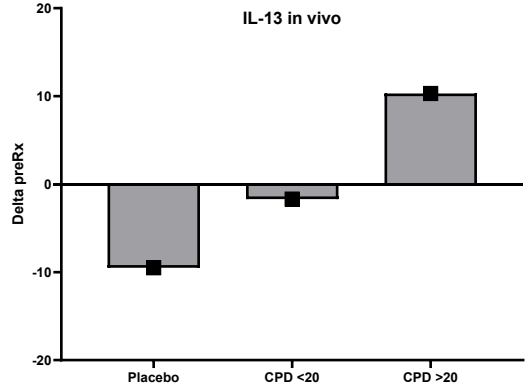


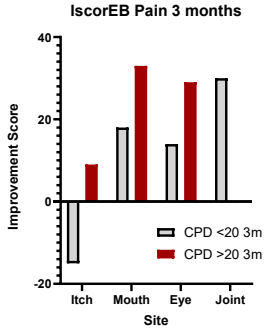
CORDStrom Mechanisms of Action

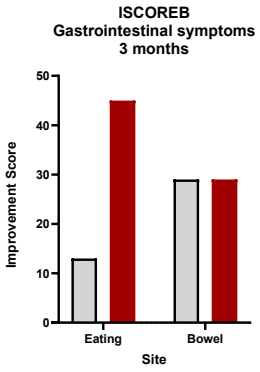
– IL-31 & TNF reduction – Itch and Pain Improved

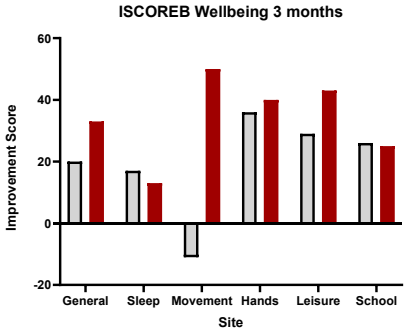


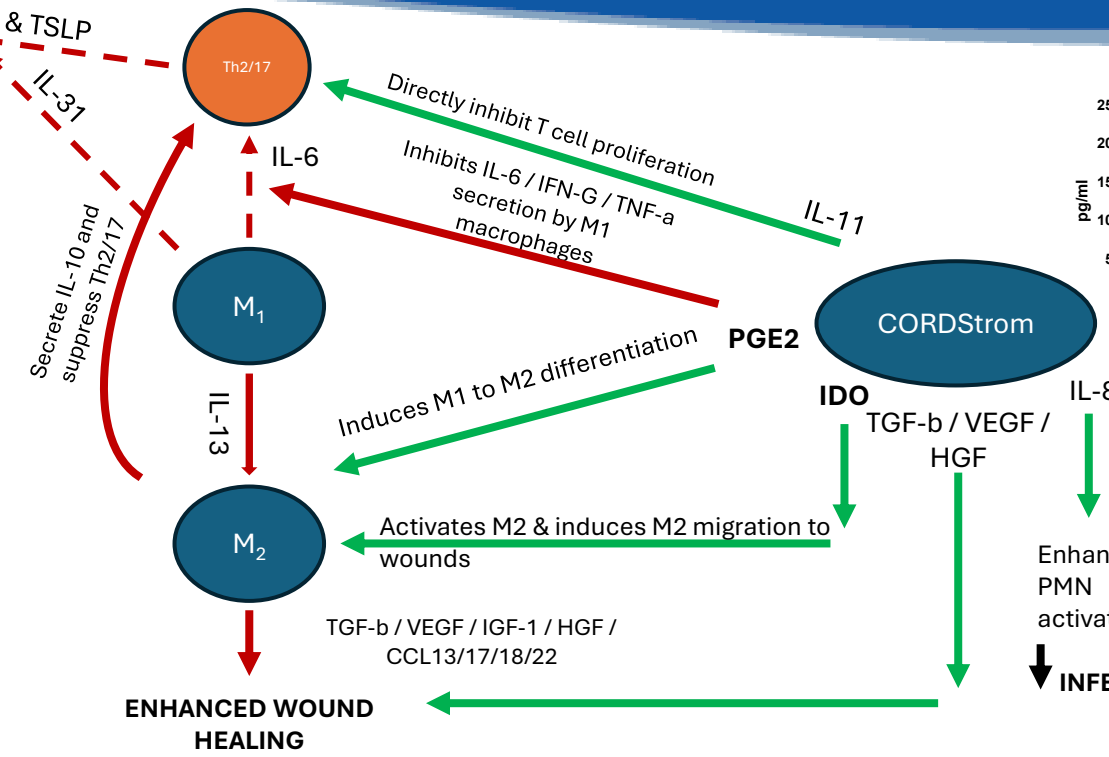


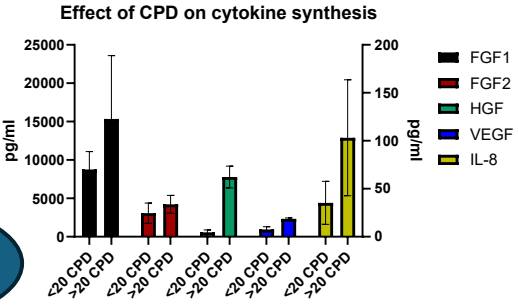


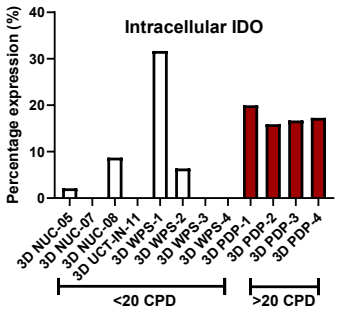


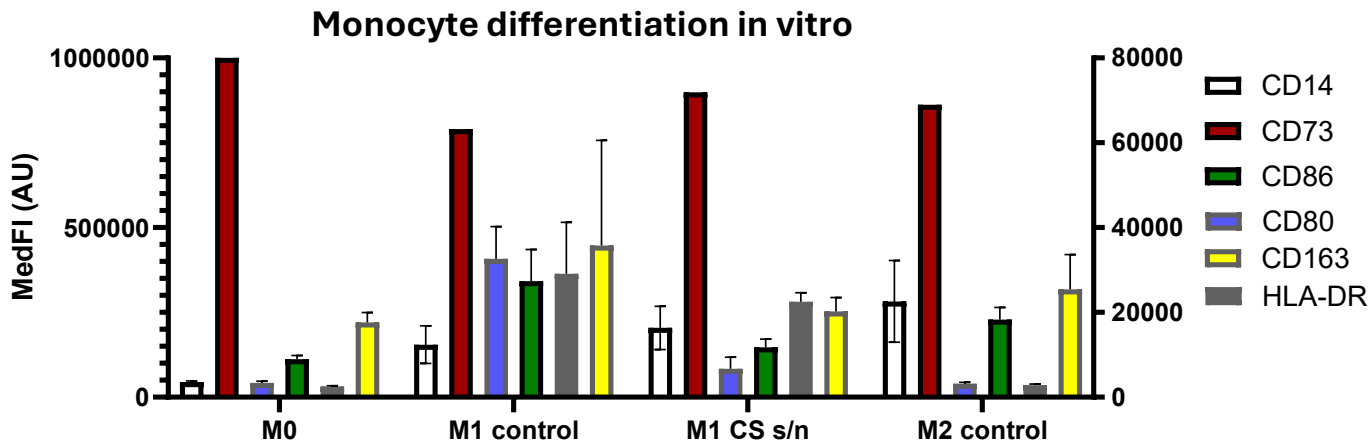






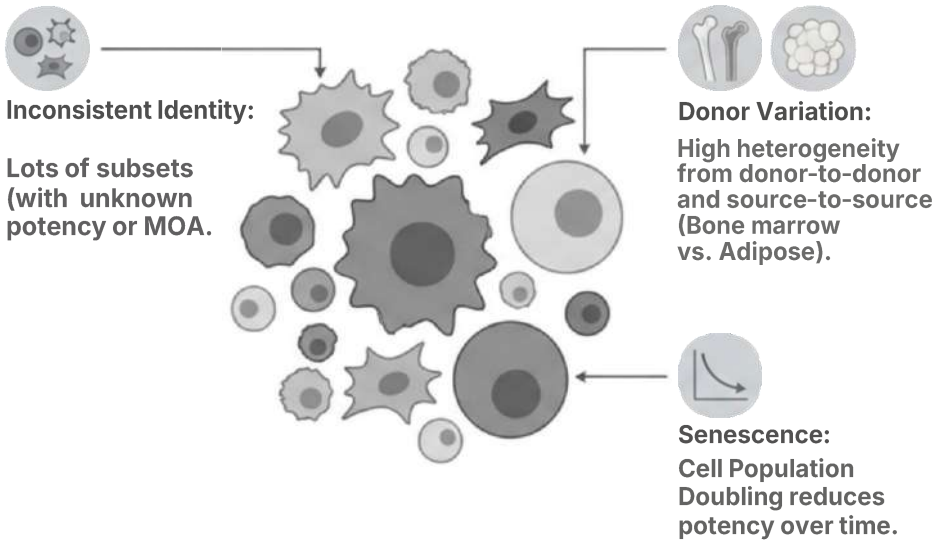






Why Historical MSC Development Failed: The Problem of Heterogeneity

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 CORDStrom™ Solution: Engineered Homogeneity Through Identity Selection and Pooling

Minimum
4 *"selected"* UC Donors

Proprietary
Pooling Process

High Potency
High Reproducibility
Master Cell Stock

Source

Umbilical Cord Tissues
(Superior expansion potential and lower heterogeneity vs. adult tissue).

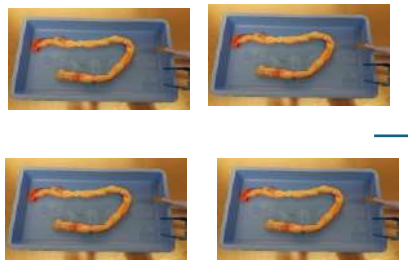
Identity

Characterized by standard markers (CD73/90/105) PLUS potency-specific markers (CD10, CD29, CD44,MSCA-1, and others).

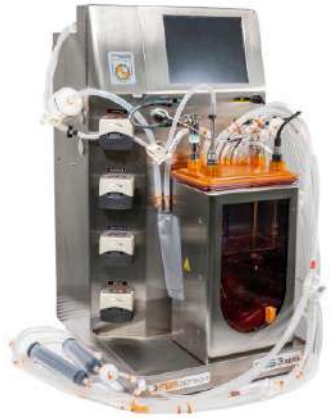
Advantage

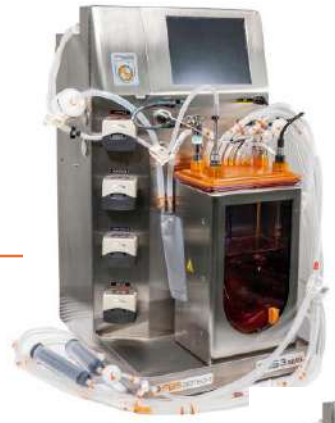
Pooling minimizes donor variation, creates a highly reproducible product, and enables massive scalability.

CMC Process in Place



Test/select/pool

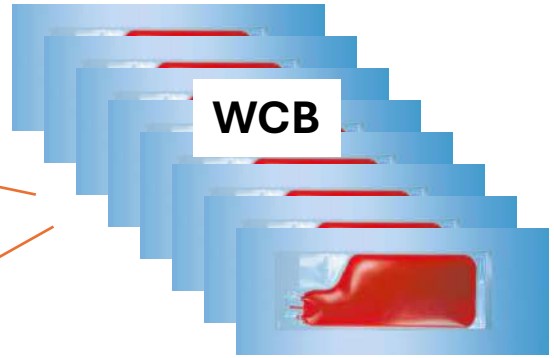




Scale up

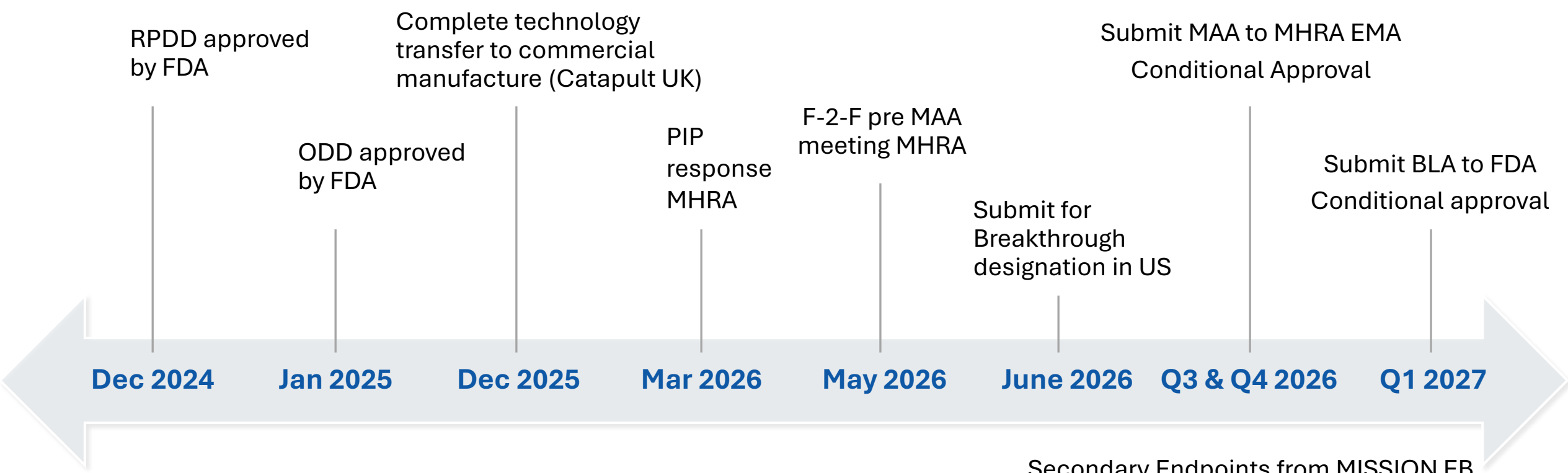
CORDStrom DP
~ 200 Doses
(30 patients' annual treatment)

Scale out



~ 40 x 200M WCB Bags

CORDStrom for RDEB: Path To Market



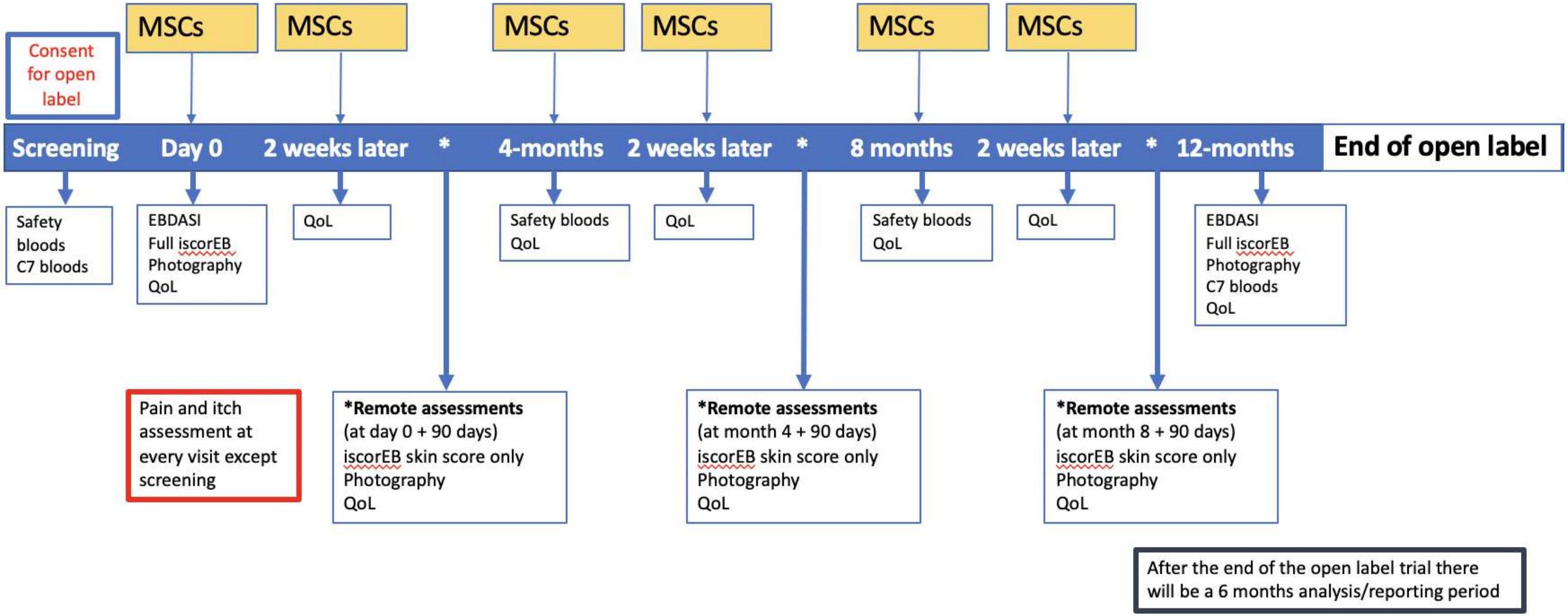
Secondary Endpoints from MISSION EB for conditional MAA submission: IscorEB, Itch, CHU-9D, Clinician Skin Score, patient interviews

positive benefit-risk ratio

FDA = Food and Drug Administration; MHRA, EMA = regulatory agencies in UK and EU; ODD = Orphan Drug Designation; MAA = Market Authorization Application; BLA = Biologics Licensing Application; RPDD = Rare Pediatric Disease Designation

*Estimated dates subject to change – See "Forward-Looking Statements"

Open Label Trial – 18 Months
MISSION EB-DELIVER



CORDStrom™: Rare Disease Therapy with Systemic Treatment Needs

Anticipated treatment schedule

- One treatment (2 doses over 14 days) every 4 months: 3 Rx (6 doses)/yr
- Anticipated Annual COG per treatment: $120 -180K (scale dependent)

Anticipated reimbursement per treatment:

- CORDStrom as *the **only systemic** treatment estimated* ~$700,000 per year
  - Potential TAM $USD:
    - 2000- 4000 RDEB cases - >$1.4bn annual turnover

Competitive Positioning

- VYJUVEK topical skin cream $750,000 per year = **~$400m turn over 2025** Krystal Biotech ~$8b market cap
- ZEVASKYN Coll7a gene-modified topical skin graft >$3m per treatment / per lesion
- FILSUVEZ topical cream for skin lesions $27,000 per 15 tubes – 1 tube per lesion/Rx

TAM = Total addressable market; 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"

CORDStrom™ RDEB Summary

- **Beyond topical care**: First-in-class systemic therapy addressing multi-organ pathology of RDEB
- **CORDStrom i.v. infusions** significantly improve multiple SYSTEMIC symptoms of RDEB and improve clinical skin scores in a randomised, double blind, placebo-controlled trial
  - EBDASI & iscorEB skin scores
  - Itch
  - Multiple pain indices
  - Quality of life
- **Excellent safety profile**:
  - all age groups
  - All patient disease severity groups
- **Commercial readiness**:
  - scale-out and scale-up options
  - Clinician-friendly packaging and supply chain delivery
  - CMC locked
  - Potency assays validated
- **Regulatory engagement underway and on time**

INmune Bio is moving from a clinical development company to a commercial provider

Parent Quotes

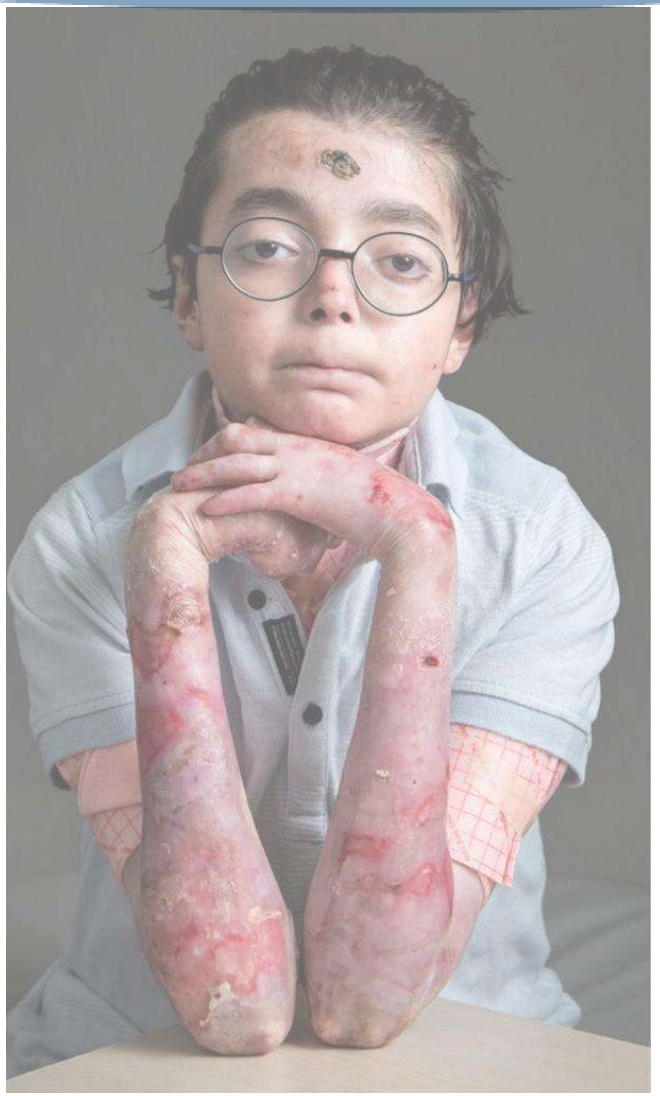
When she has been getting ill she has just been recovering in the same time as her brother, normally illness affects her much more because it causes inflammation. We have just been able to cope with everything so much better and have lots of energy and I suppose ***life has felt normal***

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"*

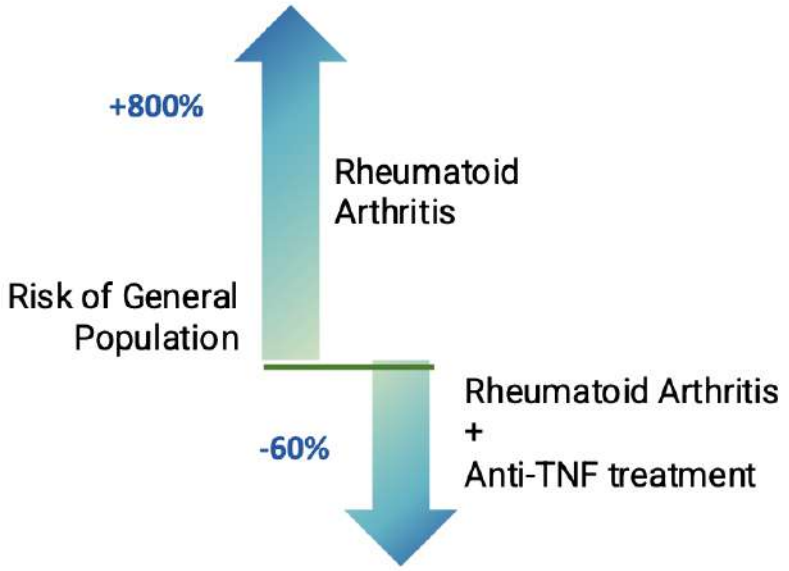
MISSION EB Team 2024

XPro™

Approaching Alzheimer's as an Immunologic Disease

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 age[6]
TNF increased in AD Patients	Plasma and CSF TNF levels increased in AD patients[2,3] TNF co-localizes with amyloid plaques[4] TNF levels correlate with disease progression[5]
TNF causes AD pathology in animals	TNF increases amyloid[7,8] and Tau[9-12] TNF causes cell loss and cognitive impairment[13]
TNF inhibitors reduce risk of AD	Anti-TNF therapies[1] 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. Paganeli 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. Janelsins 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/IKKβ) → glycemic load

Integrates metabolic consequences of chronic TNF signaling

APOE ε4 — 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.

ADi = Alzheimer's Disease with Inflammation. Cutoffs: hsCRP >1.5 mg/L, ESR >10 mm/hr, HbA1c >6%, APOE ≥1 ε4 allele. Enrichment requires ≥2 of 4.

References available upon request

MINDFuL Phase 2: Consistent Signal in ADi

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 ε4 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 ≥ 0.2 = small, d ≥ 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 ≈ 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

Placebo infection rates

2× higher

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

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.

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

End-of-Phase 2 Meeting Completed Q1 2026

Study Population
Patients with Early Alzheimer's disease and Inflammation (ADI).

Study size *
Phase 2b will target ~300
Phase 3 will target ~1000

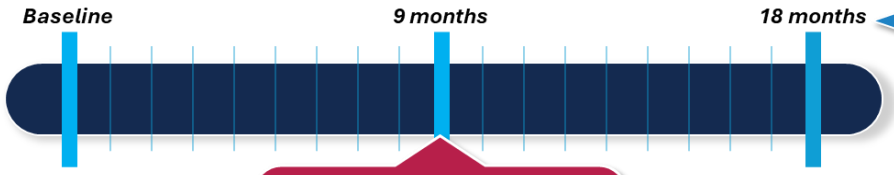
Enrichment Biomarkers
ADi is defined by ≥ 2 (of 4) of the following biomarkers: hsCRP, ESR, HbA1C, APOE4

Timeline & Treatment Arms

1:1 Randomization

XPro1595 Arm
Weekly subcutaneous injections of 1.0 mg/kg XPro1595

Placebo Arm
Matching subcutaneous placebo injection



Go/No-Go Decision Gate
Requires success on EMACC or pTau217; includes Phase 3 sample size re-estimation

Phase 2b Decision Point
Assessment of co-primary endpoints: Cognitive Performance (**EMACC**) and plasma biomarker (**pTau-217**).

Phase 3
(Registration Endpoint)
Cognition and function measured by **CDR-SB**

Secondary Metrics include additional **Clinical** (cognitive, function, Patient reported outcomes) and **Biomarker** assessments (blood and imaging)

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.

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:

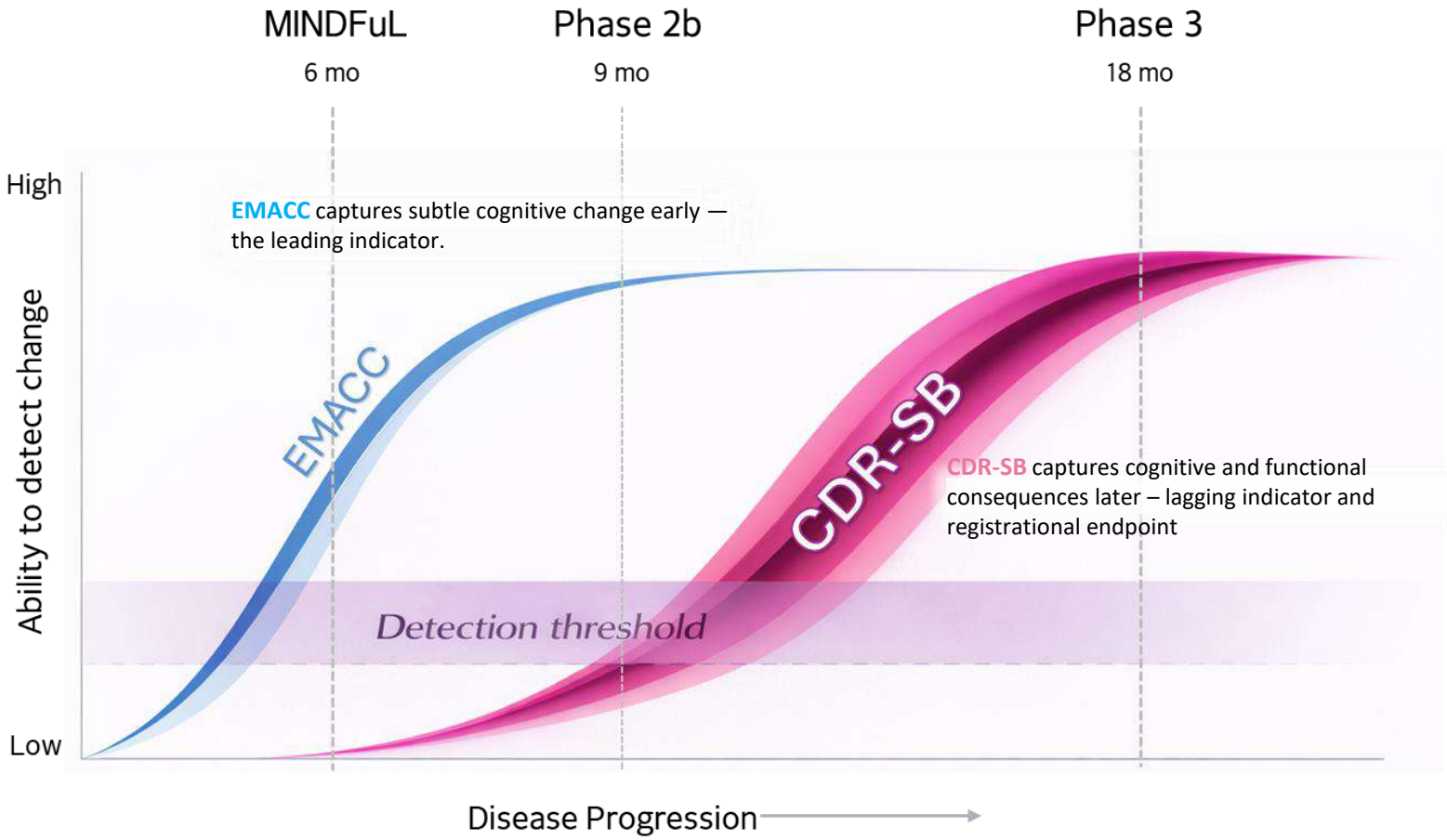
r = 0.41 (early) → r = 0.65 (18 months).

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

- 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.

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

Conceptual Framework — Not Derived from Trial Data



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.

XPro™ Summary: A Targeted Approach to Early AD and Inflammation

Phase 2 MINDFuL Trial Outcomes

Consistent Efficacy Across All Modalities
-Cognitive
- Neuropsychiatric
-Biomarkers

Absolute Cohen's d effect sizes favored XPro across endpoints.

Biomarker Signal in pTau-217 and GFAP

pTau-217 GFAP

Exploratory endpoints showed XPro slowed the accumulation of critical neurodegenerative and inflammatory biomarkers.

Superior Safety Profile with Zero ARIA

No amyloid-related imaging abnormalitles (ARIA-E or ARIA-H) were detected in 206 participants.

Strategic Roadmap: Phase 2b/3 Design

FDA Alignment on Target Population

Early AD

Regulatory agreement focuses on patients with early Alzheimer's and at least two inflammatory biomarkers.

Dual-Phase Efficacy Endpoints

Phase 2b (Month 9)

9 EMACC plasma p-Tau217

Phase 3 (Month 18)

18 CDR-SB

De-risked Staged Investment Model

GO/ NO-GO

Decision milestone before full expenditure

XPro1595 Path to Execution

- **Staged Investment:** The Phase 2b/3 design stages the capital commitment. The Phase 2b serves as a de-risking milestone with a built-in go/no-go decision before full Phase 3 expenditure.
- **Partnership Strategy:** With all this data in hand, we will run a structured global process to identify a strategic partner for the registration program. This will likely include assistance from a third-party advisory firm.
- **Capital Markets:** As catalysts mature across our pipeline, including CORDStrom, we expect improved access to capital markets on favorable terms.
- **CORDStrom Optionality:** A successful CORDStrom program generates both revenue potential and a Priority Review Voucher, either of which strengthens our financial position for XPro development.
- **Advancing Now:** Protocol development is underway. CMC scale-up planning has been initiated. Site feasibility assessments are in process. Our intent is to be clinic-ready in 2027.

INmune Bio Strategy

The Anchor

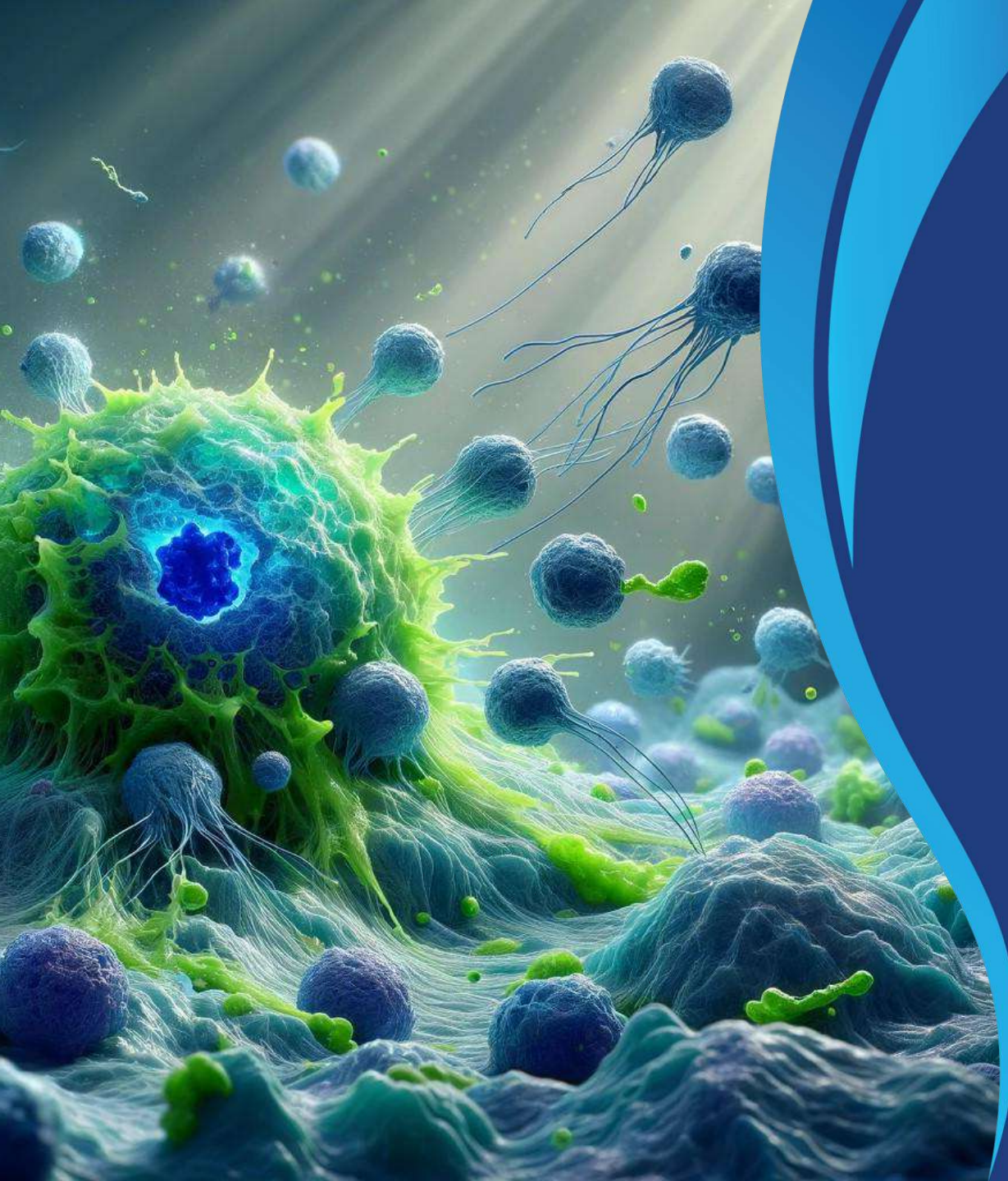
CORDStrom Approvals Expected (2027)

- US, UK, & EU Markets
- Transition to Revenue Generation
- PRV Asset Realization

The Upside

XPro Phase 3 Readiness

- De-risked Protocol
- Biomarker-Driven Precision
- Massive Alzheimer's Opportunity



Thank You!

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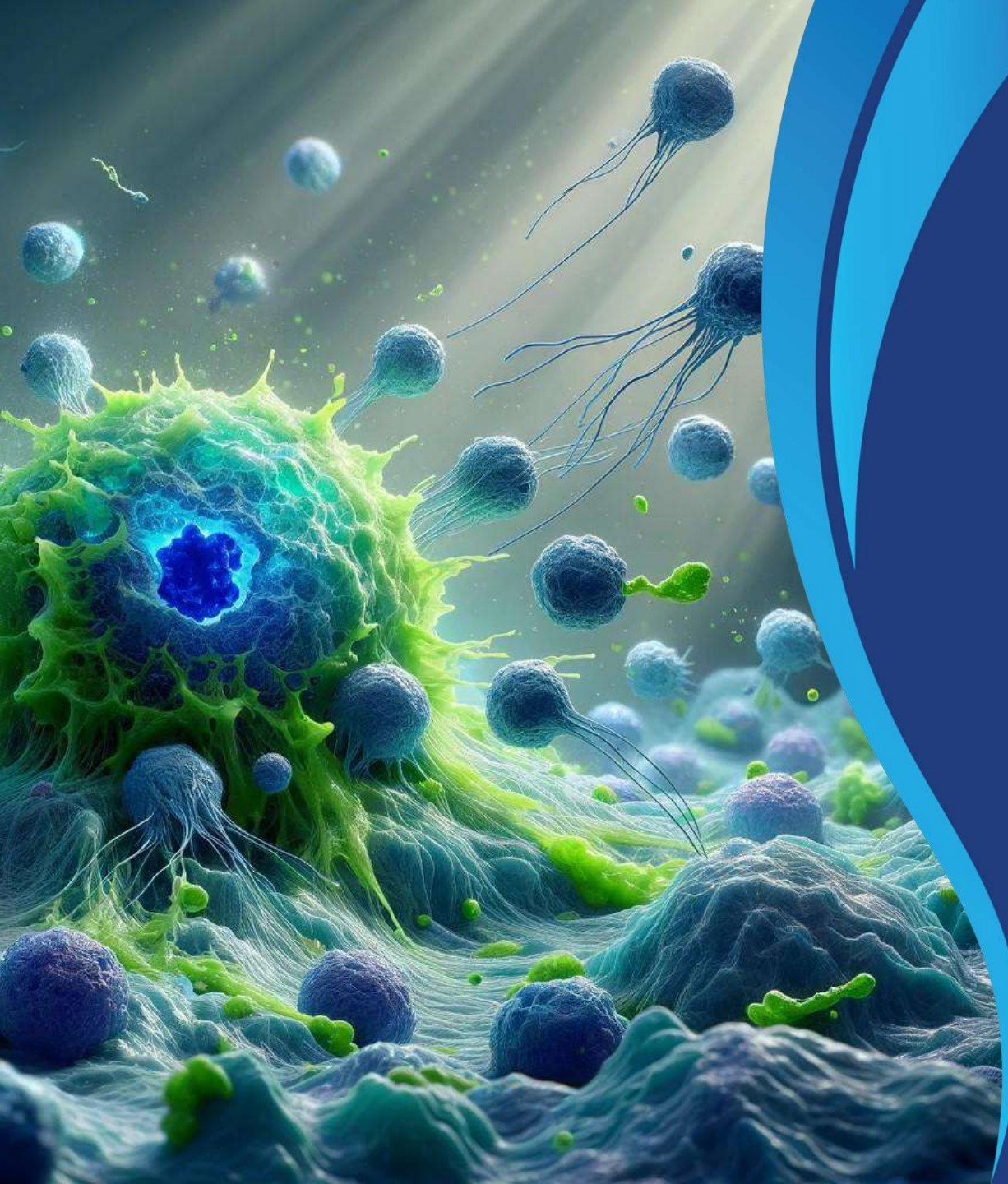
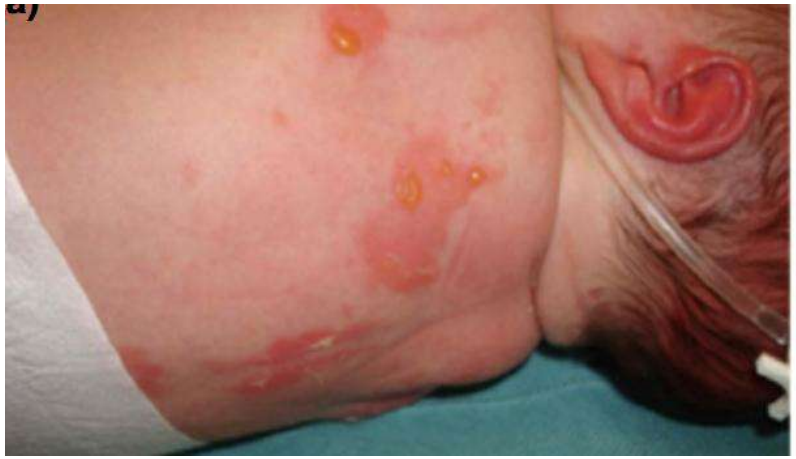
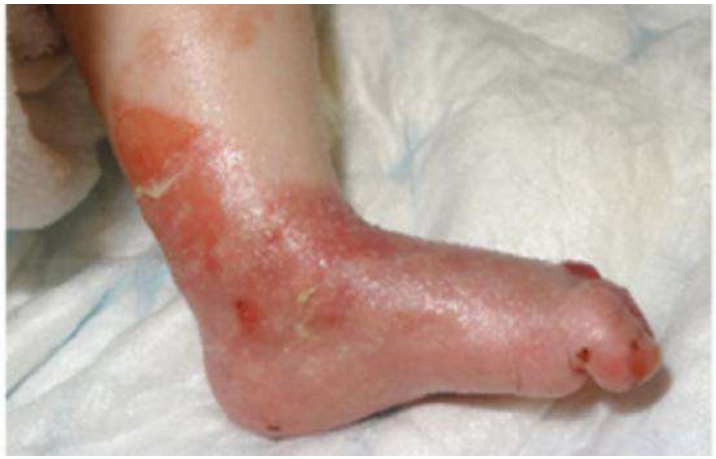
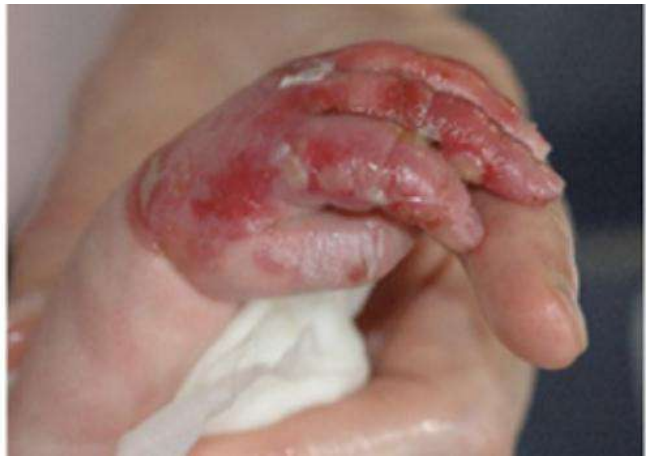


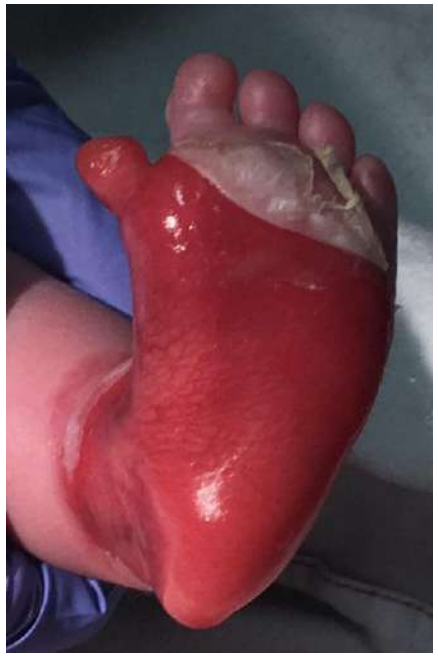
Appendix

Phase 1: 0 to 18 months









- Cutis aplasia-months to heal
- Wounds heal
- Weight gain satisfactory
- Relatively stable period

Consensus **reclassification** of inherited epidermolysis bullosa and other disorders with skin fragility.
Has C, Bauer JW, Bodemer C, Bolling MC, Bruckner-Tuderman L, Diem A, Fine JD, Heagerty A, Hovnanian A, Marinkovich MP, Martinez AE, McGrath JA, Moss C, Murrell DF, Palisson F, Schwieger-Briel A, Sprecher E, Tamai K, Uitto J, Woodley DT, Zambruno G, Mellerio JE.
Br J Dermatol. 2020 Feb 4. doi: 10.1111/bjd.18921. Online ahead of print.
PMID: 32017015 Review.

Phase 2: 18 months to 10 years

- Wound burden & skin colonization advances
- Wounds heal within 21 days
- They are recurrent & reoccurring wounds but not chronic

- First signs of internal involvement
    - inflammatory markers elevated
    - weight faltering
    - the onset of anemia
    - first signs of esophageal involvement and scaring

Phase 1 and 2

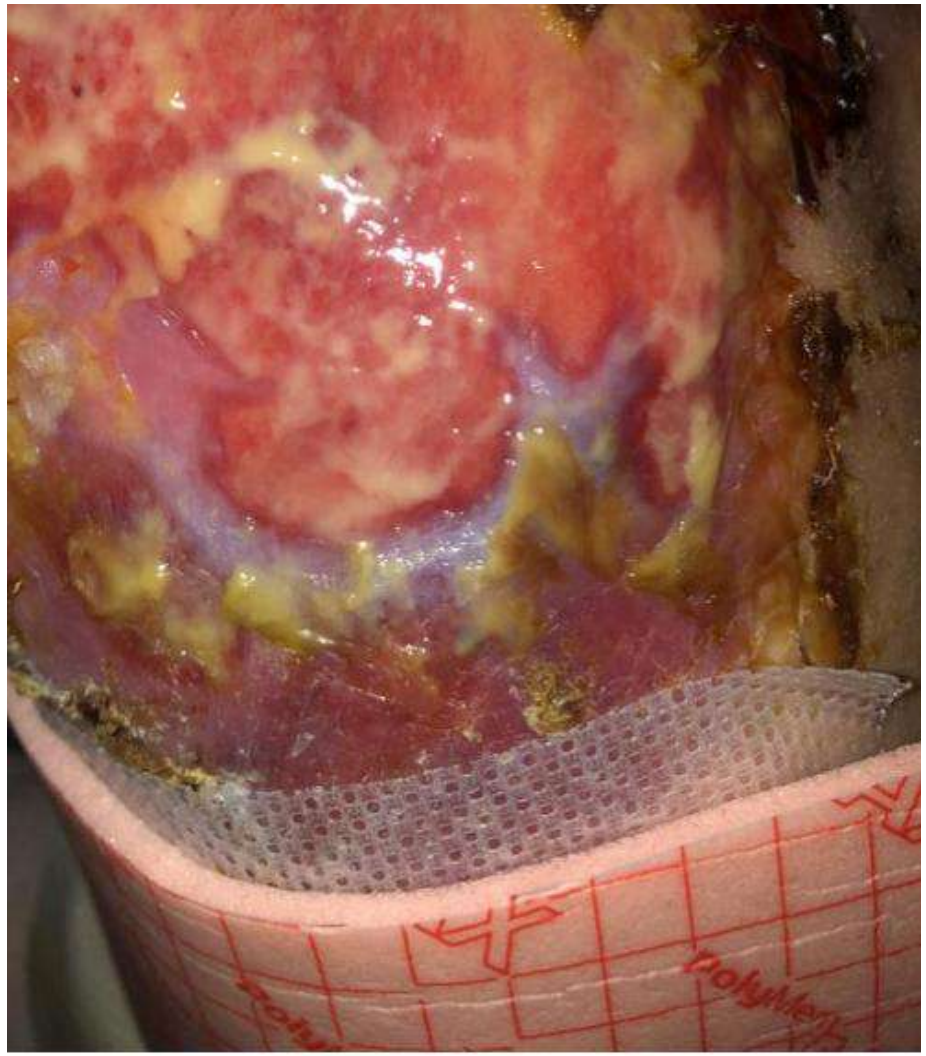
- Window of opportunity for anti-inflammatory therapy such as CORDStrom for systemic disease pathology

Therapeutic Requirements:
Anti-inflammatory cytokines
Reprogramming pro-inflammatory macrophages (M1) to anti-inflammatory (M2)

Phase 3: 10 to 20 years

- Skin involvement continues to increase
  - chronic wounds over 21 days
  - bacterial colonisation
  - multiple co morbidities
- Dramatic reduction in the ability to heal

Therapeutic Requirements:
Wound healing cytokines
Increased innate immune activity against bacteria/fungi

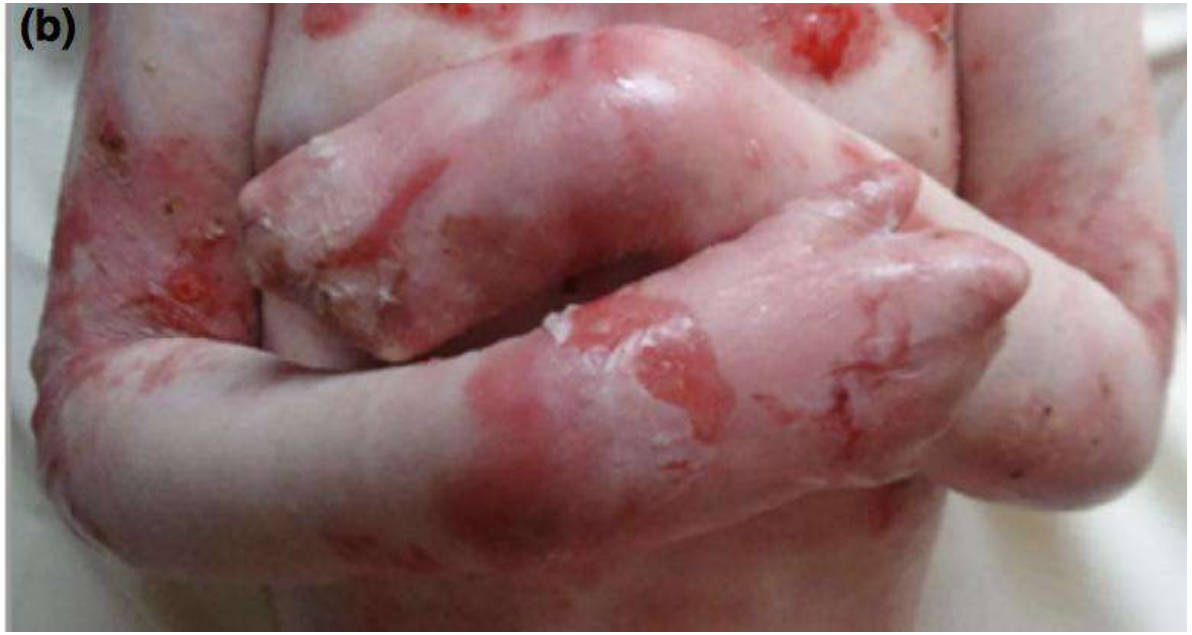


Phase 3: 10 to 20 years

- Massive levels of chronic inflammation
  - affects 4 major pathways

- Hypothalamic-pit-gonadal axis → pubertal delay
- GH/IGF-1 axis → growth delay
- Bone regulatory pathway → osteoporosis & fractures
- Iron metabolism → anaemia

- **Anti-inflammatory therapy much less effective**

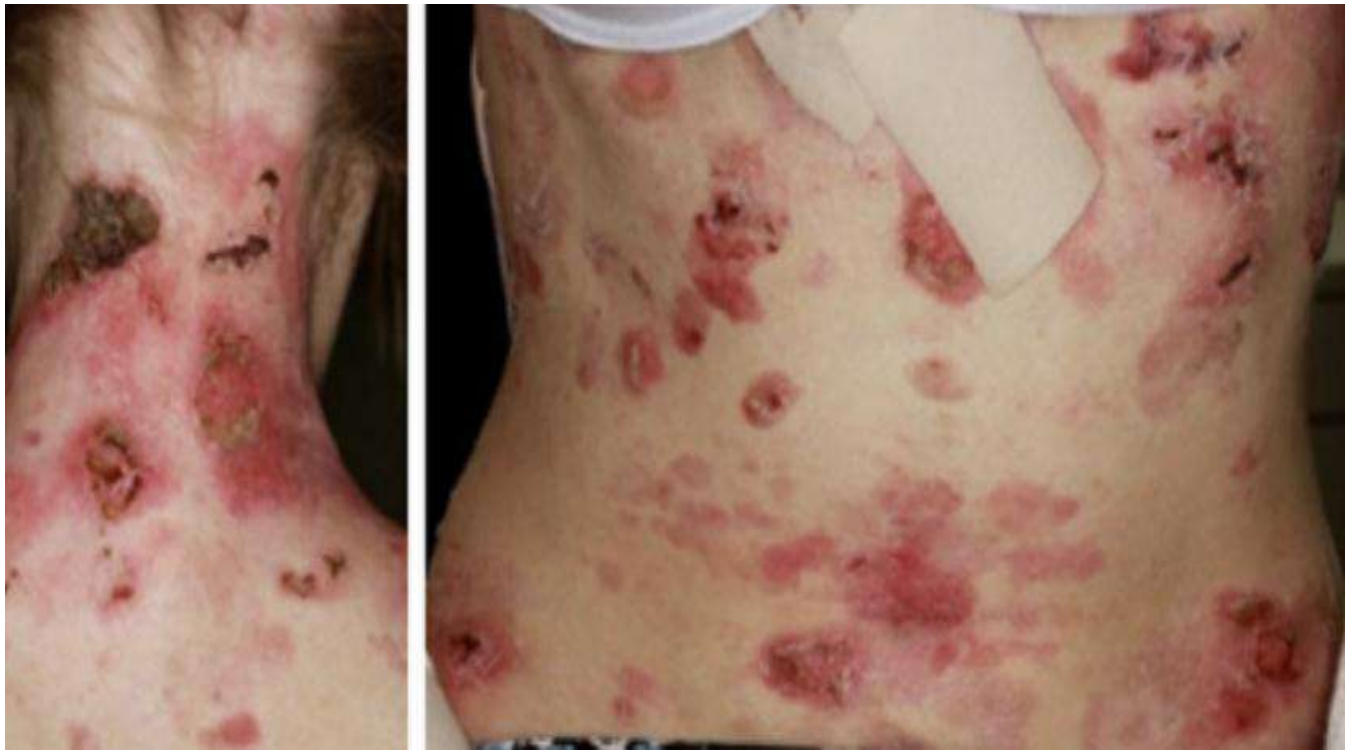


Therapeutic Requirements:
Wound healing cytokines
Increased innate immune activity against bacteria/fungi

Phase 4: From 20 years onwards

- Steady progression of the severity
- Chronic wounds very rarely heal

Therapeutic Requirements:
Symptom control



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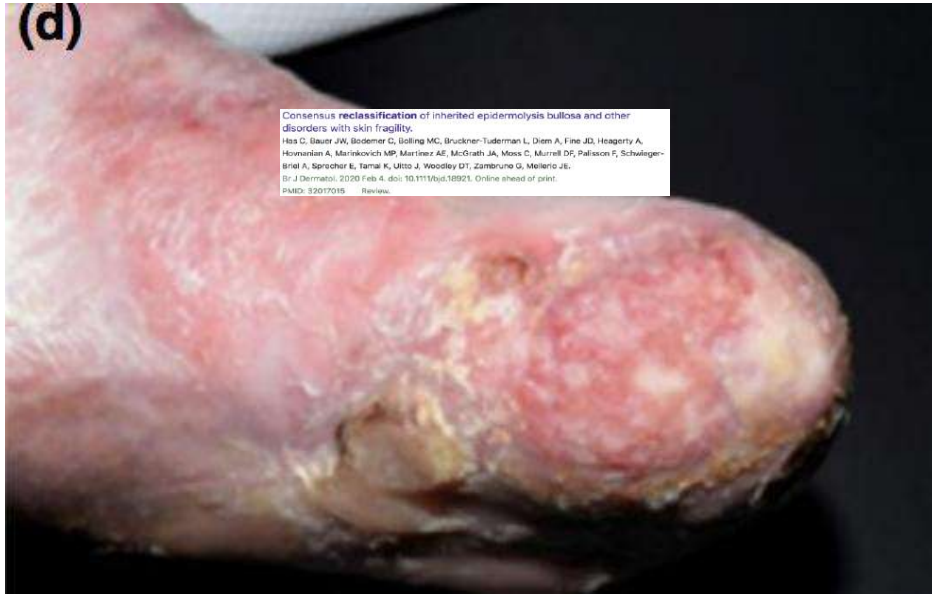
Br J Dermatol. 2020 Feb 4. doi: 10.1111/bjd.18921. Online ahead of print.
PMID: 32017015 Review.

Phase 4: From 20 years onwards

- Patients are at high risk for developing squamous cell carcinoma, a life-threatening complication of severe RDEB
- Tumors are usually well differentiated and rapidly-growing, often on the extremities with a median survival of 2.4 years for this EB subtype
- Anti-inflammatory and antifibrotic therapies are now unlikely to help much

Therapeutic Requirements:
Symptom control



Cutaneous Squamous Cell Carcinoma in Epidermolysis Bullosa: a 28-year Retrospective Study

Susan J. ROBERTSON[1], Elizabeth ORRIN[1], Manpreet K. LAKHAN[1], Gavin O'SULLIVAN[1], Jessie FELTON[2], Alistair ROBSON[3], Danielle T. GREENBLATT[1], Catina BERNARDIS[4], John A. MCGRATH[1,5], Anna E. MARTINEZ[6] and Jemima E. MELLERIO[1,5]
[1]St John's Institute of Dermatology, Guy's and St Thomas' NHS Foundation Trust, London, [2]Department of Dermatology, Brighton and Sussex University Hospitals NHS Trust, Brighton, UK, [3]Department of Pathology, Instituto Português de Oncologia de Lisboa, Lisboa, Portugal, [4]Department of Plastic Surgery, Guy's and St Thomas' NHS Foundation Trust, [5]Genetic Skin Disease Group, King's College London and [6]Department of Dermatology, Great Ormond Street Hospital for Children NHS Foundation Trust, London, UK

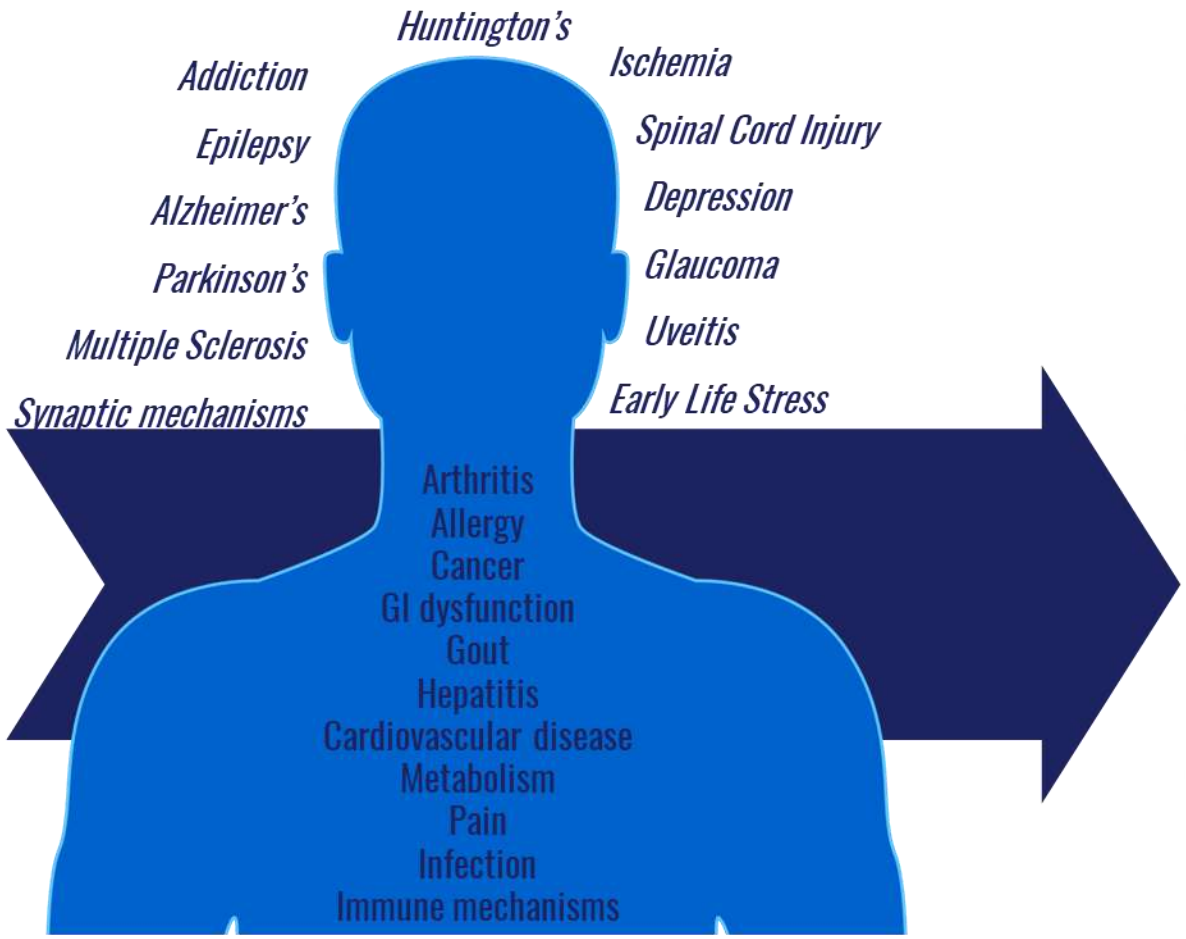
Accepted Jul 6, 2021; Epub ahead of print Jul 7, 2021

Acta Derm Venereol 2021; 101: adv00523.

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)

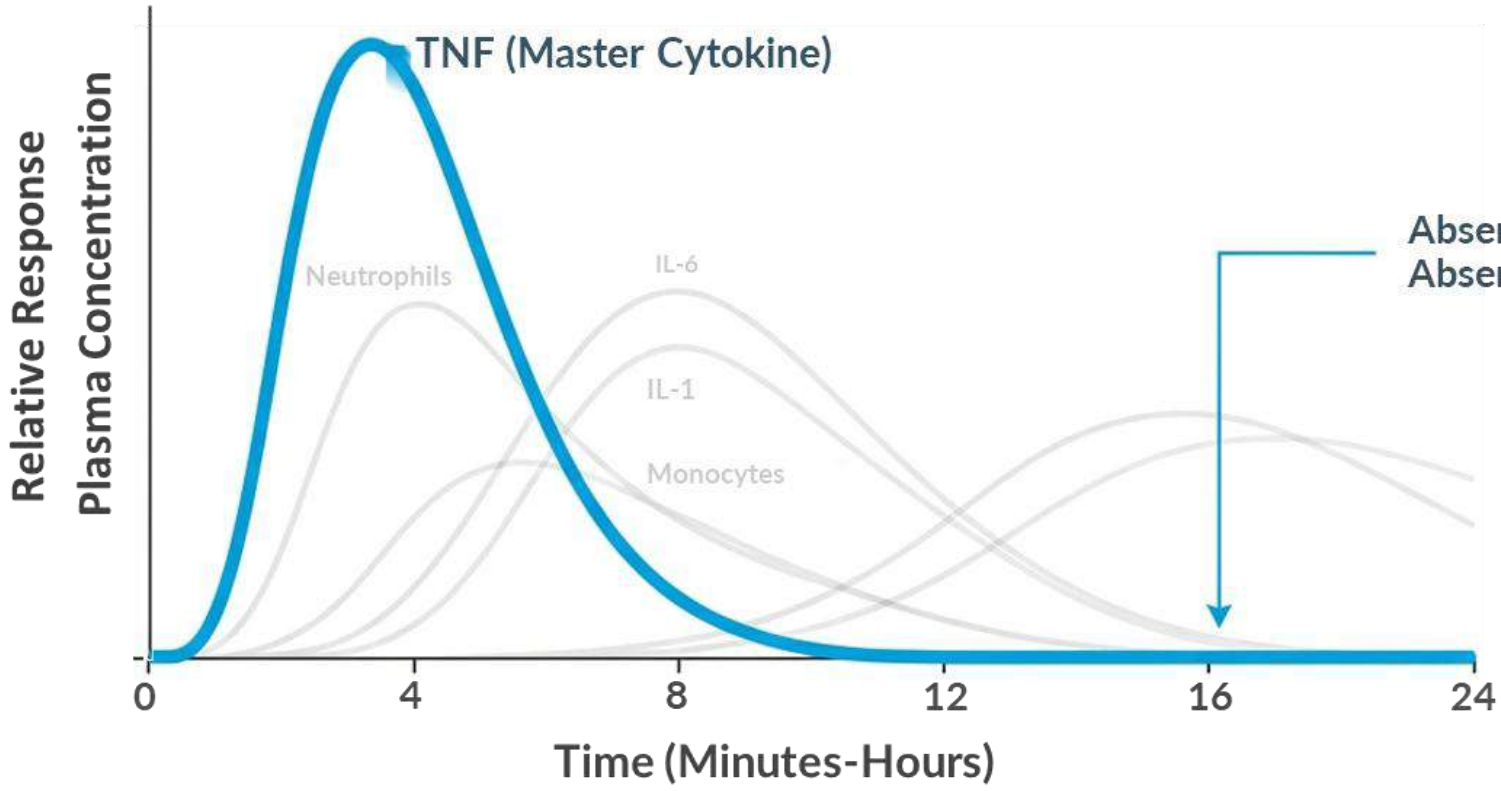


>90 Publications

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)

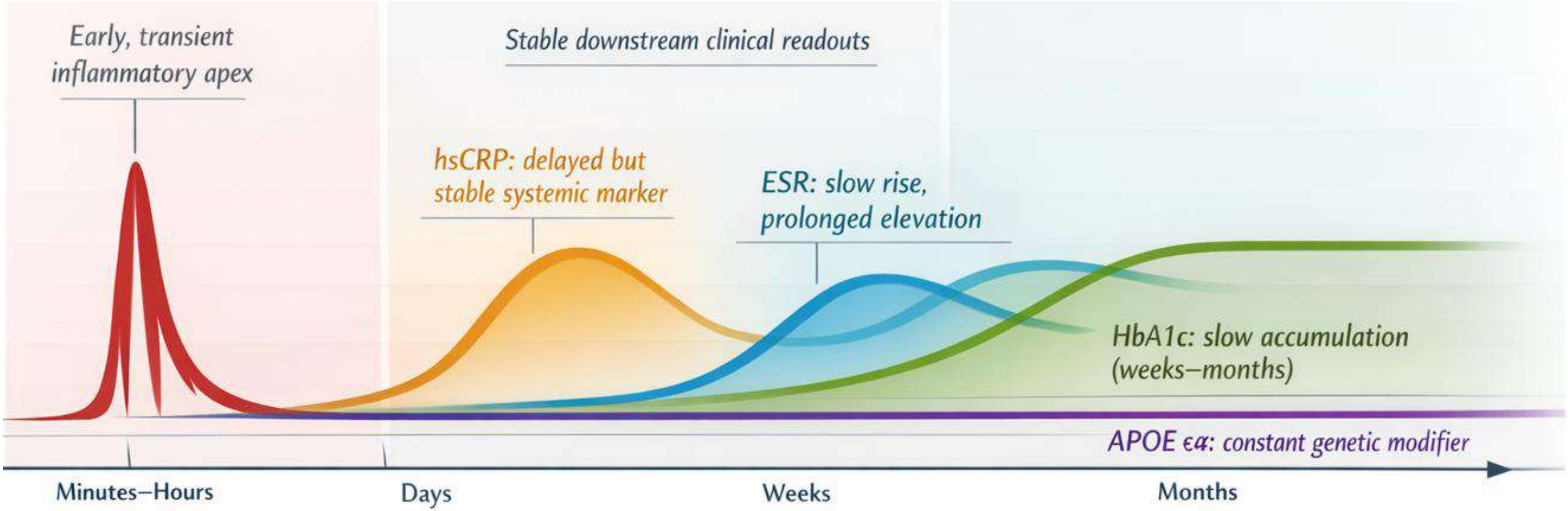


Absence of TNF in plasma ≠
Absence of TNF-driven disease.

Direct measurement of cytokines fails to capture the ongoing pathology due to transient expression windows.

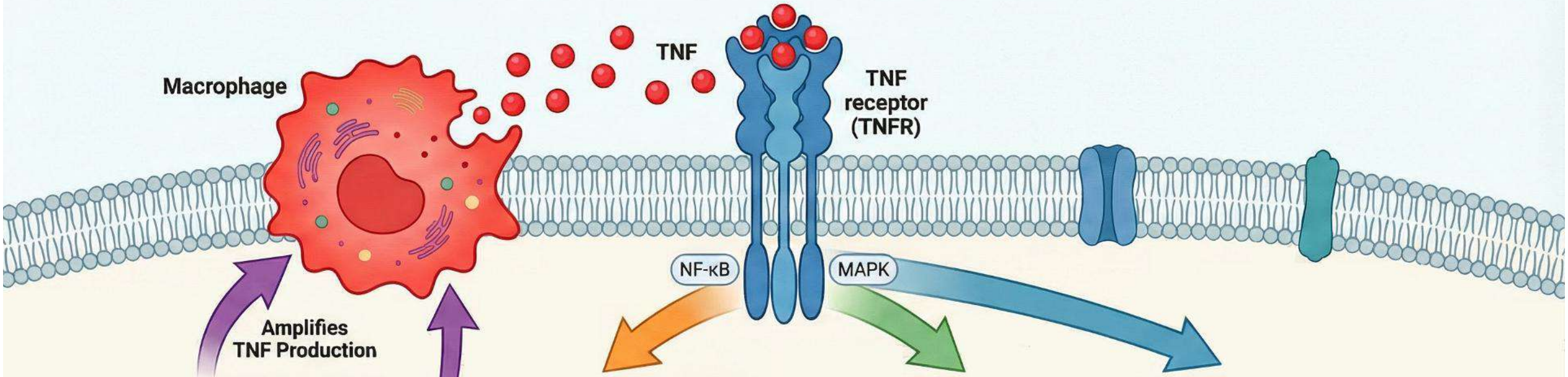
Adapted from PMID: 27994591 and 18673219

Capturing Stability: from Minutes to Lifelong Predisposition

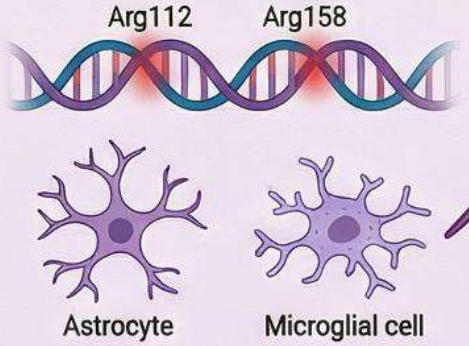


Xu et al., 2020; Resi et al., 2021

Mechanistic Links Between TNF and Downstream Biomarkers

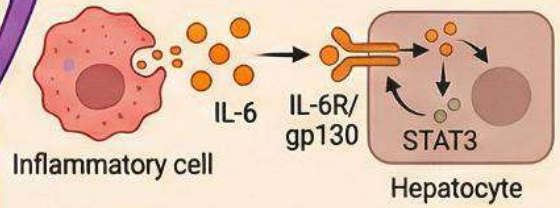


APOE ε4 Constitutional Amplification



Genetic variants Arg112/Arg158 trigger lifelong glial inflammation, increasing TNF production in microglia and astrocytes.

IL-6 to CRP Acute-Phase Cascade

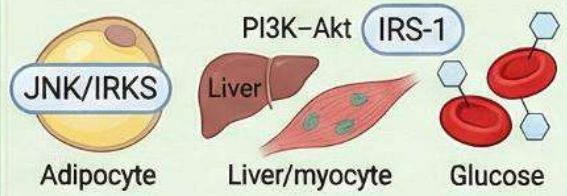


TNF-activated hepatocytes utilize STAT3 signaling to produce C-reactive protein over days to weeks.

C-reactive protein (CRP)

Typical Time Scale: Days to Weeks

Insulin Resistance and HbA1c Elevation

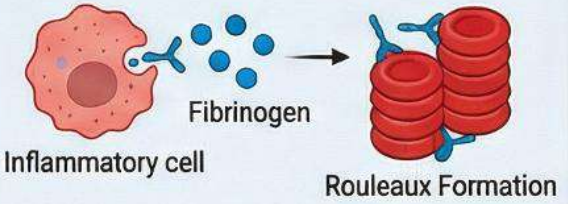


TNF-driven inflammation impairs PI3K–Akt signaling in liver/myocytes, increasing chronic glycemic load over months.

HbA1c

Typical Time Scale: Weeks to Months

Chronic Inflammation and ESR Readout



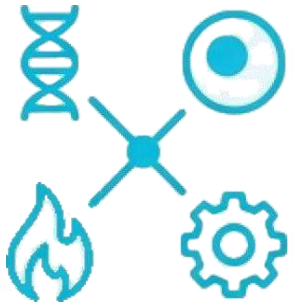
Increased fibrinogen production causes red blood cells to form rouleaux, elevating the Erythrocyte Sedimentation Rate.

Erythrocyte Sedimentation Rate (ESR)

Typical Time Scale: Weeks to Months

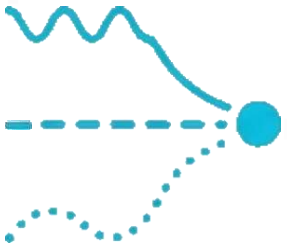
The "≥2 of 4" Composite Design: High-Confidence Identification of a TNF-Driven Phenotype

Requiring positivity on at least two independent biomarkers substantially increases specificity for TNF-driven biology and reduces misclassification arising from single-marker noise or non-TNF-related inflammatory processes.



Captures Multidimensionality

TNF signaling exerts pleiotropic effects across multiple biological domains, including genetic susceptibility, metabolic state, acute inflammatory activity, and chronic inflammatory burden. The composite framework integrates signals across these axes rather than relying on a single biological dimension.



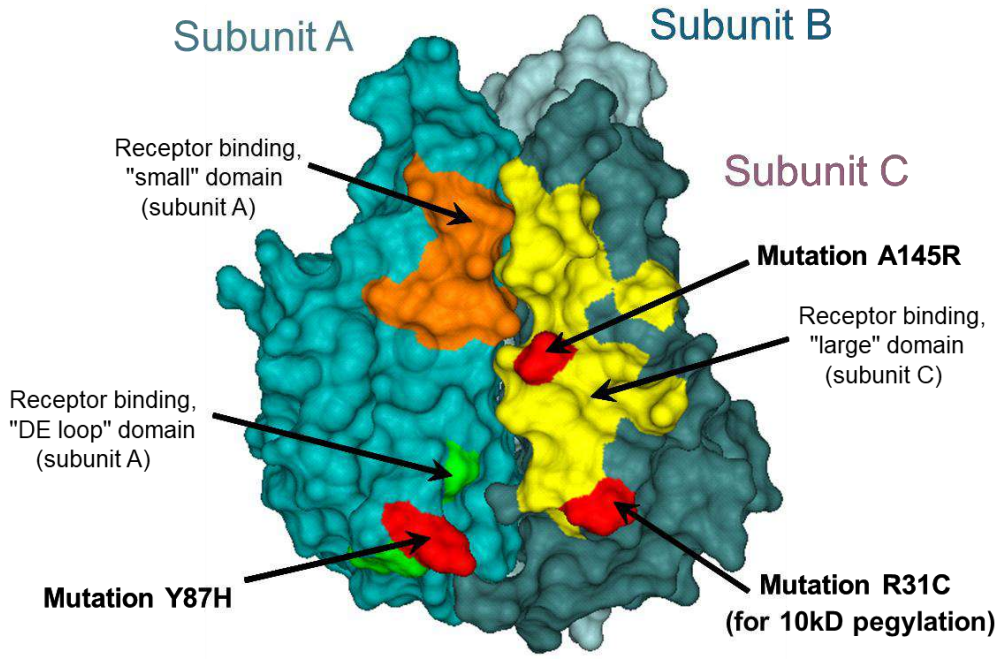
Accommodates Biological Heterogeneity

The design identifies patients with TNF-driven disease regardless of the specific biological manifestation. Patients may qualify through different combinations of genetic, metabolic, acute, and chronic biomarkers, allowing for 11 distinct qualifying biomarker combinations.

Reduces Misclassification Risk

Built-in redundancy ensures that failure of a single assay or transient suppression of an individual biomarker is less likely to incorrectly exclude a truly TNF-responsive patient.

XPro1595: Dominant-negative proteins



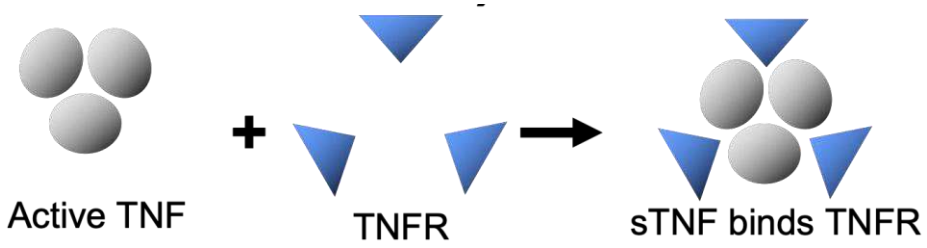
XPro1595 is identical to the human soluble TNF monomer with the exception of mutations in the receptor binding domain and another for pegylation.

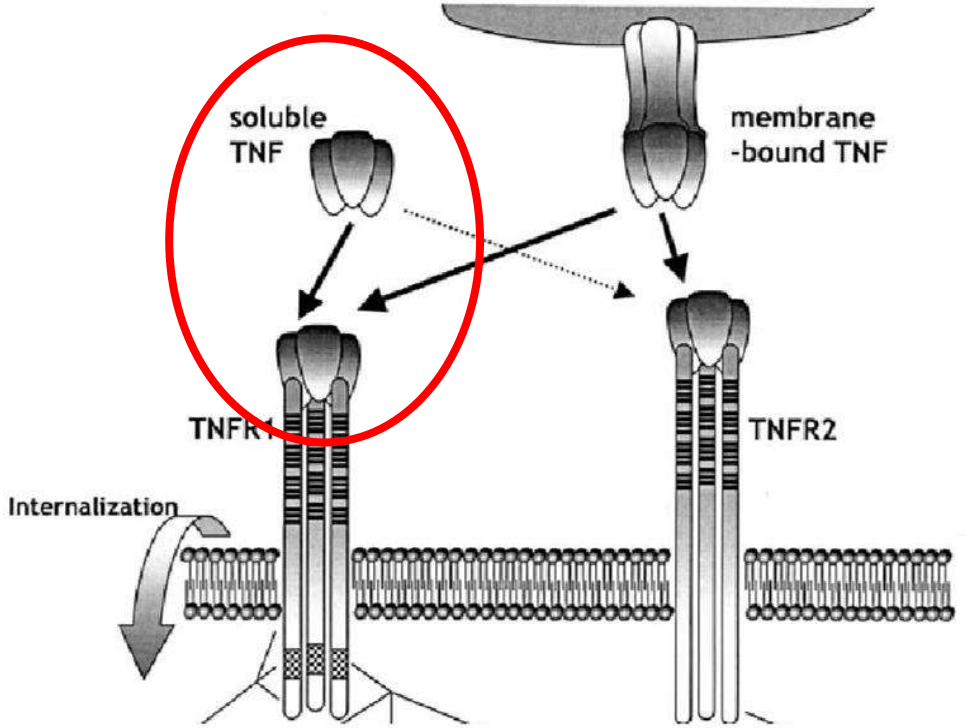
Dominant-Negative in genetics:
"A mutation producing a rogue protein that interferes with the function of the native protein."

see Steed et al., *Science*, 301, 2003

Normal Soluble TNF activity

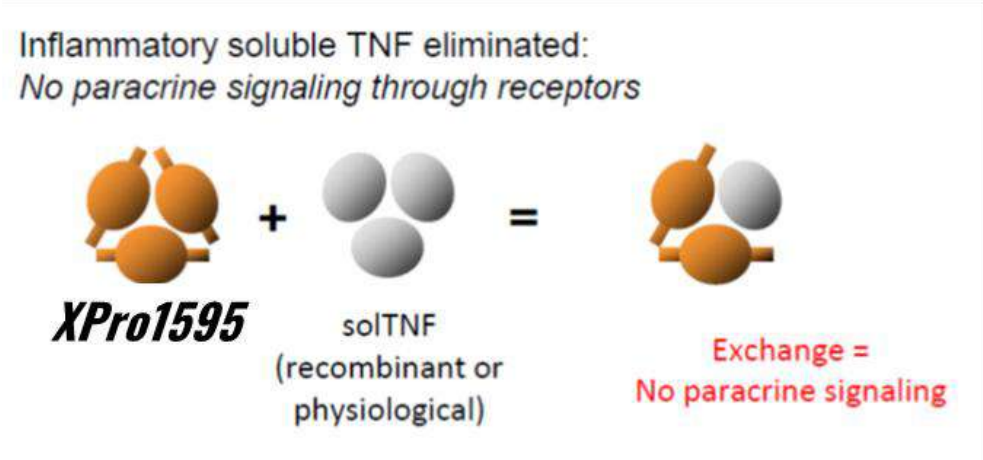
solTNF monomers form active heterotrimers that bind to TNF receptor. Each monomer in the trimer contacts one TNFR subunit



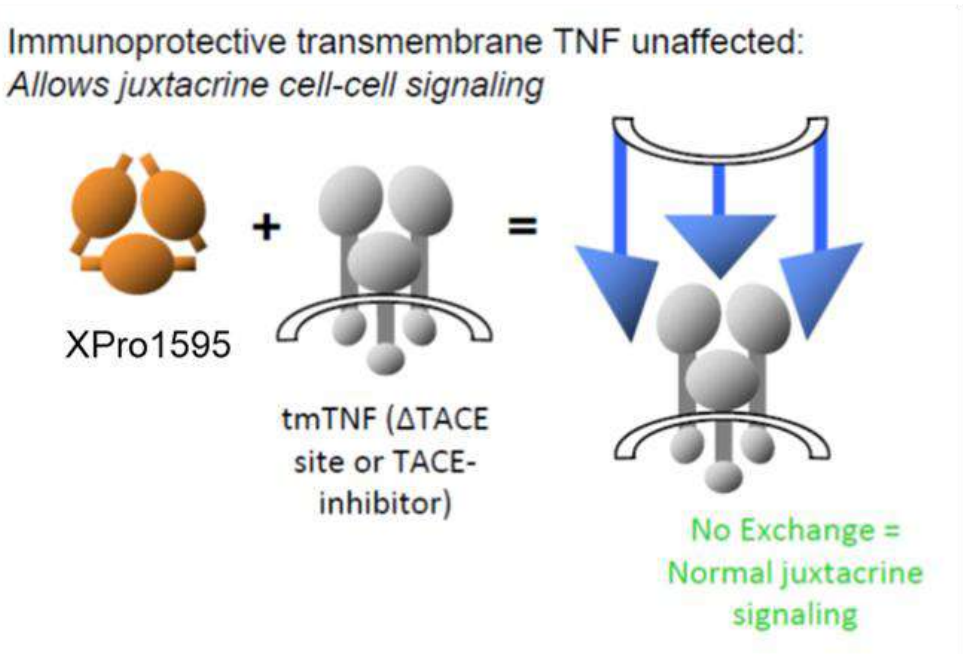


XPro1595 MOA

Xpro1595 freely exchanges with solTNF monomers to form inactive heterotrimers

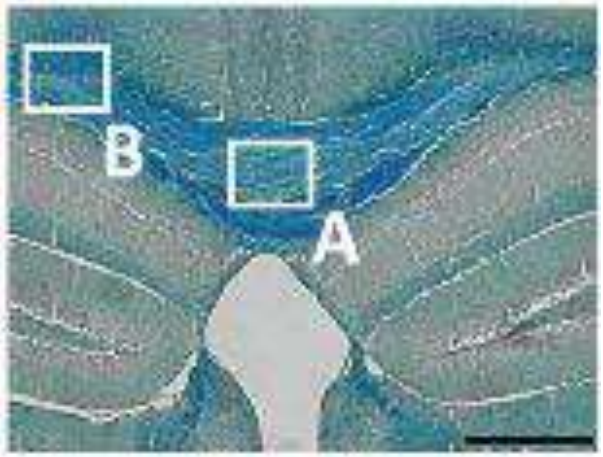


tmTNF homotrimers are anchored to the cell membrane, *XPro1595* cannot exchange



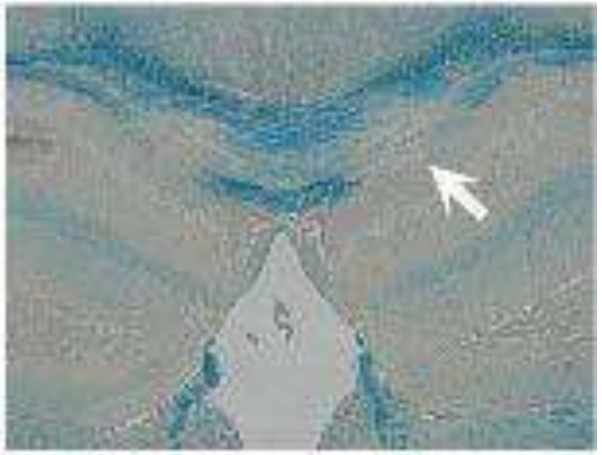
XPro1595 is neuroprotective

Normal



Cuprizone

(Model of Multiple Sclerosis)



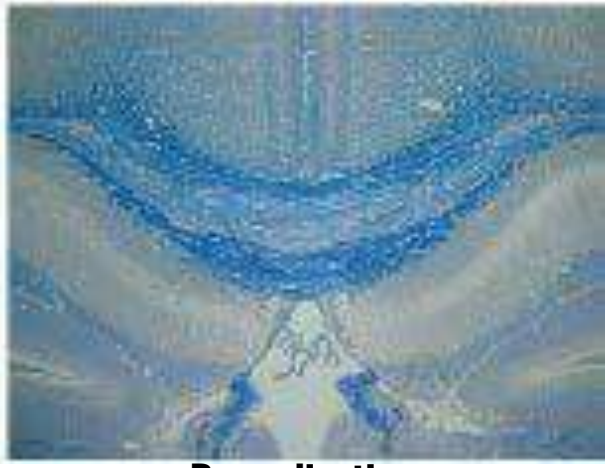
Etanercept

Anti-inflammatory AND immunosuppressive

Exacerbated demyelination

XPro1595

Anti-inflammatory NOT immunosuppressive



Remyelination

Adapted from: Karamita et al., 2017