INmuneBio
INNATE IMMUNITY

Modulating the Innate Immune System to Transform Inflammation-Driven Disease

Corporate Presentation

November 2025

NASDAQ: INMB

FORWARD-LOOKING STATEMENTS

This presentation contains “forward-looking statements” Forward-looking statements reflect our current view about future events. When used in this presentation, the words “anticipate,” “believe,” “estimate,” “expect,” “future,” “intend,” “plan,” or the negative of these terms and similar expressions, as they relate to us or our management, identify forward-looking statements. Such statements, include, but are not limited to, statements contained in this presentation relating to our business strategy, our future operating results and liquidity and capital resources outlook. Forward-looking statements are based on our current expectations and assumptions regarding our business, the economy and other future conditions. Because forward–looking statements relate to the future, they are subject to inherent uncertainties, risks and changes in circumstances that are difficult to predict. Our actual results may differ materially from those contemplated by the forward-looking statements. They are neither statements of historical fact nor guarantees of assurance of future performance. We caution you therefore against relying on any of these forward-looking statements. Important factors that could cause actual results to differ materially from those in the forward-looking statements include, without limitation, our ability to raise capital to fund continuing operations; our ability to protect our intellectual property rights; the impact of any infringement actions or other litigation brought against us; competition from other providers and products; our ability to develop and commercialize products and services; changes in government regulation; our ability to complete capital raising transactions; and other factors (including the “Risk Factors” contained in our most recent Annual Report on Form 10-K and any other filings that we have made or may make with the SEC in the future), such as those relating to our industry, our operations and results of operations. Actual results may differ significantly from those anticipated, believed, estimated, expected, intended or planned. Factors or events that could cause our actual results to differ may emerge from time to time, and it is not possible for us to predict all of them. We cannot guarantee future results, levels of activity, performance or achievements. Except as required by applicable law, including the securities laws of the United States, we do not intend to update any of the forward-looking statements to conform these statements to actual results.

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.

INmune Bio – Company Snapshot

Overview

Clinical-stage biotechnology company developing next-generation immunotherapies targeting innate immune dysfunction in rare pediatric inflammatory disease and inflammatory neurologic disease.

Key Programs

Program	Indication	Stage	Differentiation
CORDStrom™	RDEB (ultra-rare pediatric)	Phase 2 complete → MAA/BLA filings in 2026	First systemic therapy for RDEB; defined MSC biologic
XPro™	Early Alzheimer's	Phase 2 complete → Phase 3 start 2027	Selective soluble-TNF blockade; ↓pTau217, ↓GFAP; **zero ARIA**
INKmune®	Cancer (NK activation)	Phase 1/2 complete	Primes NK cells to attack resistant tumor cells

NASDAQ: INMB | Boca Raton, FL | London, UK	
Share Price (a/o 10/14/25)	$1.87
52-week Trading Range	$1.71 - $11.64
FY End	Dec. 31
Shares Outstanding	~26.5M
Market Capitalization (Basic) (a/o 10/14/25)	$50M
Cash (a/o 10/14/25)	$30mm

2026–2027 Catalysts

Q1 2026: XPro End-of-Phase-2 FDA meeting*
Mid-2026: CORDStrom MAA (UK)*
Late-2026: CORDStrom BLA (US)*
2027: XPro Phase 3 initiation*

Regulatory Designations

CORDStrom: RPDD, ODD
Potential PRV upon approval

Model

Capital-efficient clinical execution
GMP scaling via Catapult UK
Focus on translational efficiency + regulatory alignment

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

Late-Stage Pipeline Across Inflammation & Immunology

Multiple near-term regulatory and pivotal-trial milestones

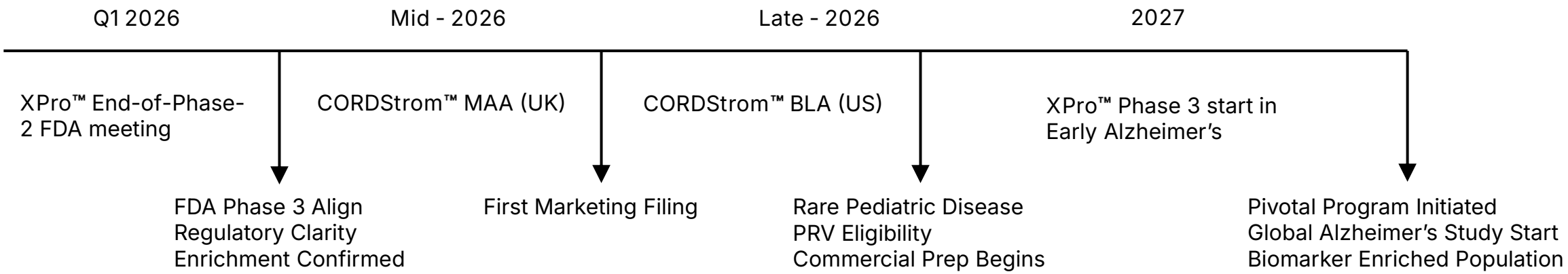
Program/Platform	Indication / Patient Pop (US&EU)	Pre-clinical	Phase 1	Phase 2	Filing / Phase 3
CORDStrom™	RDEB (pediatric, ultra-rare) (4K)	→	→	→	RPDD + ODD Received MAA 2026 → BLA 2026
XPro™	Early Alzheimer's Disease with Infl. (9M)	→	→	→	EOP2 Q1 2026 → Phase 3 2027
INKmune®	Residual Disease in Solid Tumors	→	→		Expansion planning

Two late-stage assets entering regulatory and pivotal-trial phase with established safety, biomarker validation, and pediatric rare disease positioning.

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

Milestones in 2026–2027*

Clear regulatory pathway and late-stage execution across rare disease and Alzheimer's

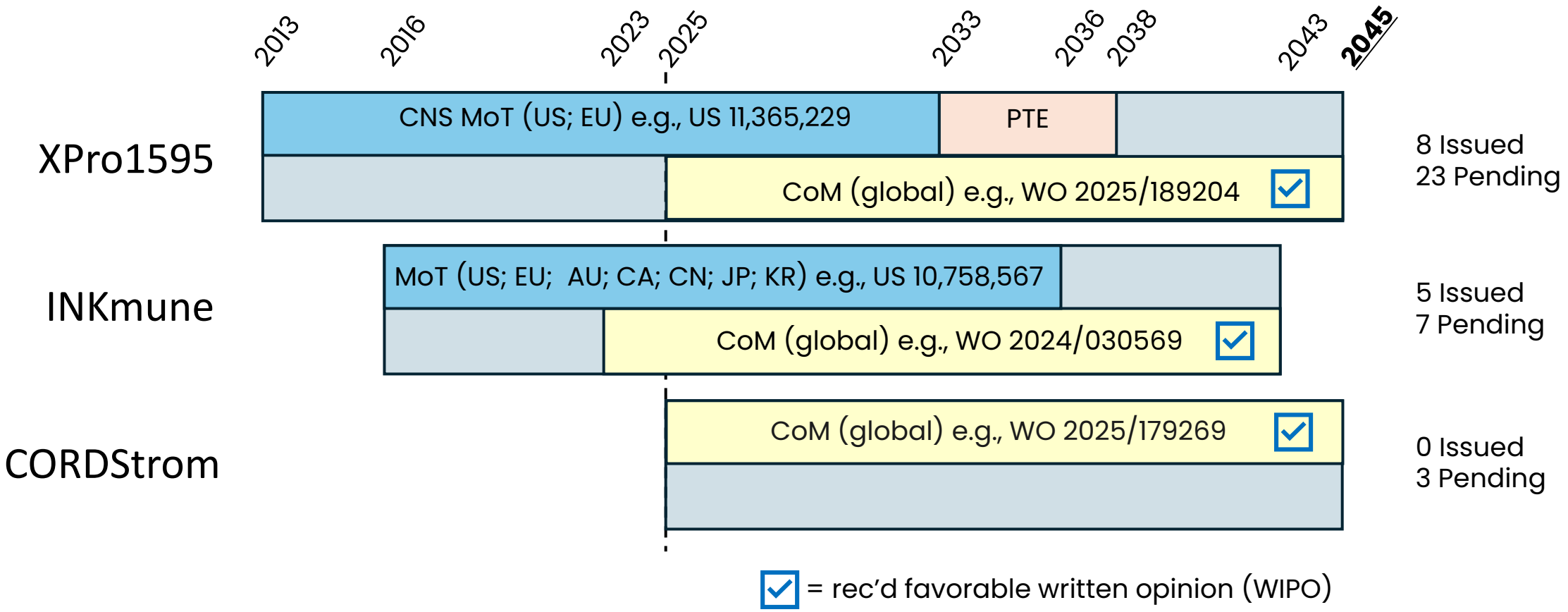


What this means:

- De-risked pipeline entering pivotal phase
- Proprietary innate immunology platform across two major indications
- Rare pediatric regulatory acceleration
- Alzheimer's program aligned for a precision-enriched Phase 3
- Clear path to PRV monetization and capital efficiency

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

Robust and Growing Intellectual Property Estate



Composition of Matter (CoM)
Method of Treatment (MoT)
Central Nervous System (CNS)
Patent term extension (PTE)

*Information as of October 8, 2025, dates and IP are subject to change. See "Forward-Looking Statements"

INmuneBio

CORDStrom™

for EB

Off-the-shelf allogeneic, pooled mesenchymal stromal cell (MSC) platform

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

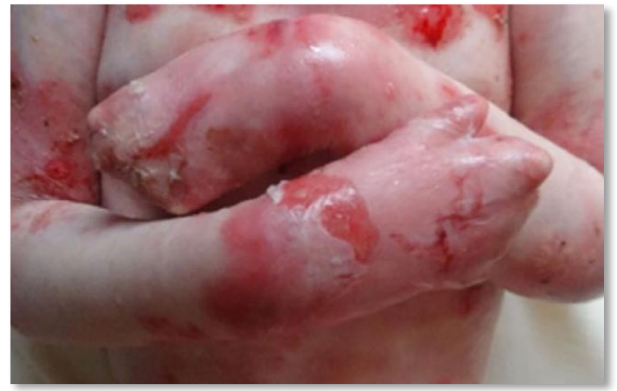
- Suppression of inflammation: RDEB/EB, OA, trauma, etc
- Immunomodulation: Autoimmune diseases etc
- Enhanced wound healing: RDEB/EB, post surgery, SLE, etc
- Suppression of fibrosis: OA, cardiac repair, RDEB/EB, etc

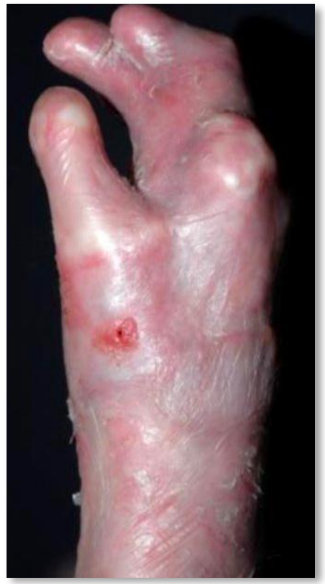
Why CORDStrom Succeeds: Always Developed as a Drug

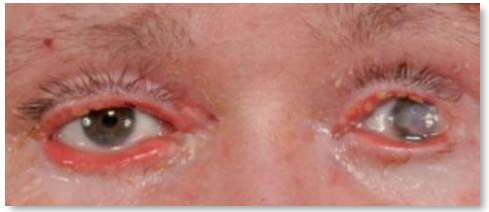
Historical Failures of MSC Drug Development	CORDStrom Solves Historical MSC Failures
• **Many MSC Sources:** Bone marrow, adipose tissue, UC, placenta, etc.	• **CORDStrom Source:** Umbilical Cord Tissues from multiple donors.
• **MSC Identity:** CD73/90/105, lots of subsets with unknown potency, lack of MoA.	• **CORDStrom Identity**: CD73/90/105 plus CD10/29/44/142/146/166/200/271 and MSCA-1. MoA for each indication.
• **MSC Heterogeneity:** donor-to-donor, source-to-source, Cell Population Doubling number causes change in potency, senescence.	• **CORDStrom Homogeneity**: Pooled donors create reproducible products with greater expansion potential. Individual donor selection can tune specific products.
• **Clinically Naïve:** "All MSCs are the same", no need for isolation, adipose SVF is a "stem cell."	• **Clinical Development**: Proven indications from randomized controlled trials and/or meta-analyses

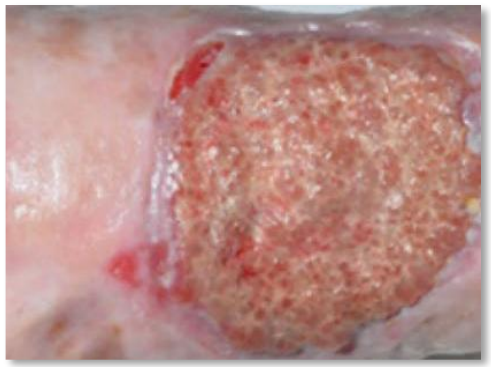
RDEB – An Ultra-Rare Systemic Disease with No Approved Systemic Treatments

- RDEB is a severe form of epidermolysis bullosa (EB), a rare disease that causes severe skin fragility, itch and chronic pain caused by mutations in the COL7A1 gene
- Itch-Scratch-Wound cycle impairs wound healing by separating skin layers and forming new blisters. Intense itch causes poor QOL and over time, most RDEB patients progress to develop squamous cell carcinoma caused by the accumulated damage to their skin
- RDEB systemic nature stems from widespread tissue damage affecting nearly every organ system. There are limited options available for treatment, none which address the systemic tissue damage
- CORDStrom™ is potentially the first systemic therapy, with itch and pain relief benefits as a key differentiating factors in quality of life and potential use as an adjunctive therapy









*Recessive Dystrophic Epidermolysis Bullosa (RDEB); Quality of Life (QOL)

RDEB Disease Progression

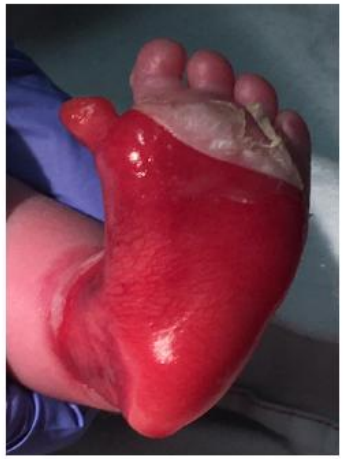
Journal of
Dermatology and Venereology

ScienceAssist International

Prospective | Open Access

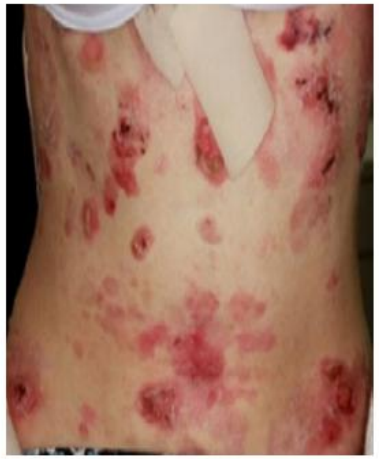
The Natural History of Severe Recessive Dystrophic Epidermolysis Bullosa – 4 Phases Which May Help Determine Different Therapeutic Approaches

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

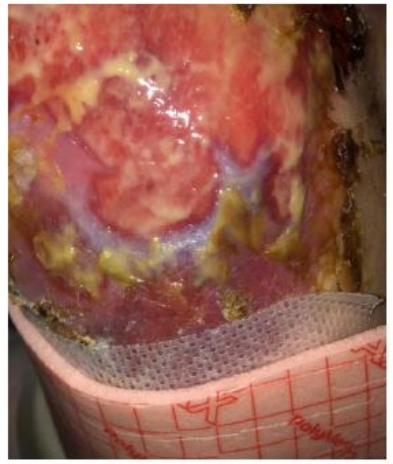


0-18 months

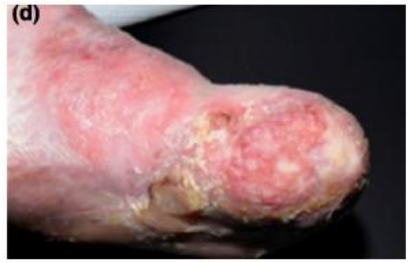
18 months-10 years



10-20 years



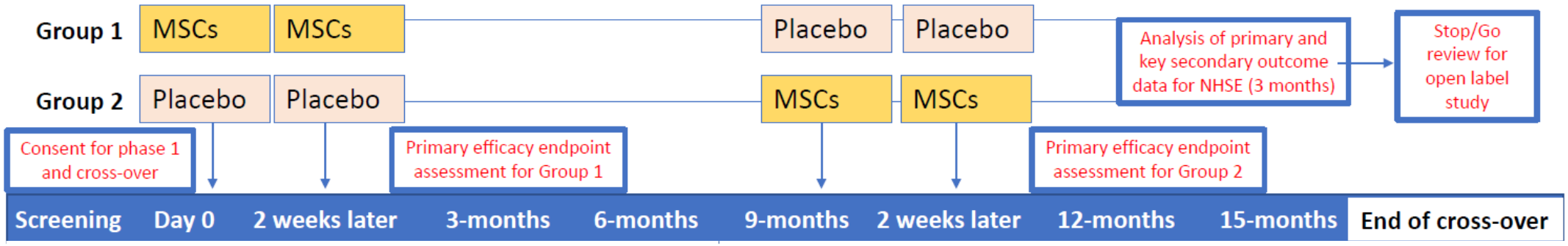
Over 20 years



Squamous cell carcinoma

Phase 2 Mission EB Trial

Double blinded placebo control study of **M**esenchymal **I**ntravenous **S**tromal cell Infu**sion**s in children with recessive dystrophic Epidermolysis Bullosa (MissionEB)



MissionEB

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

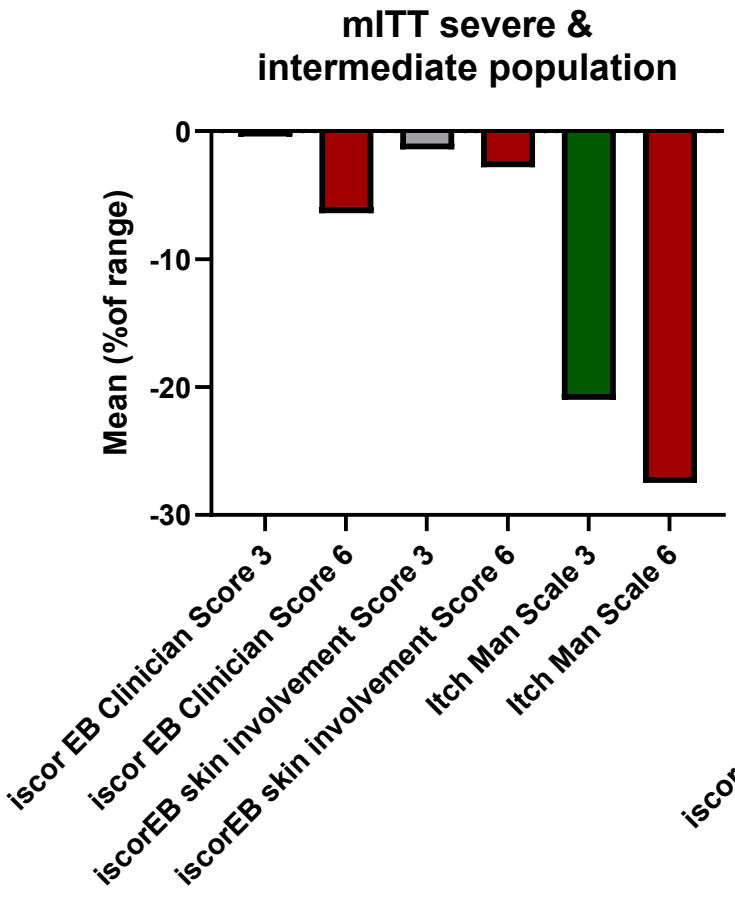
Primary objective

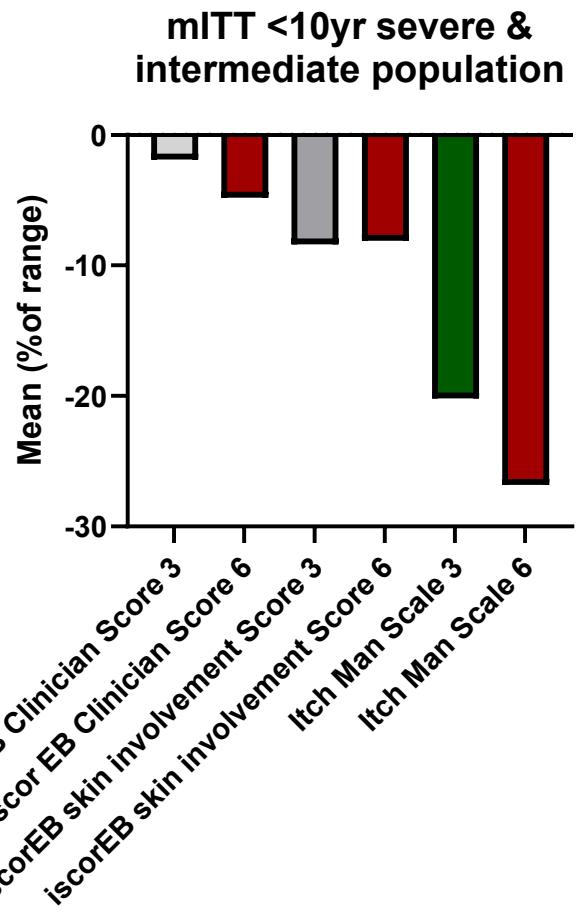
- Internal phase 1 dose de-escalation study: assess safety

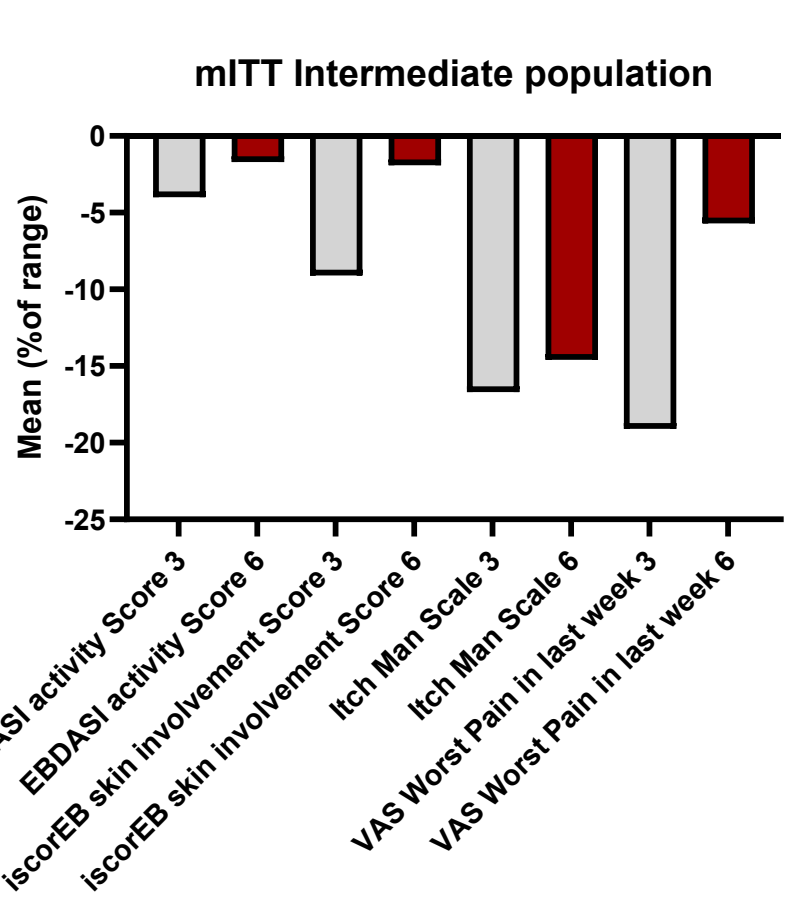
Secondary objectives

- assess the safety & efficacy of repeated UC-MSCs
- undertake a health economic analysis
- explore patients & parents views to treatment effectiveness & acceptability

Quantitative Results – Phase II Preliminary Analysis



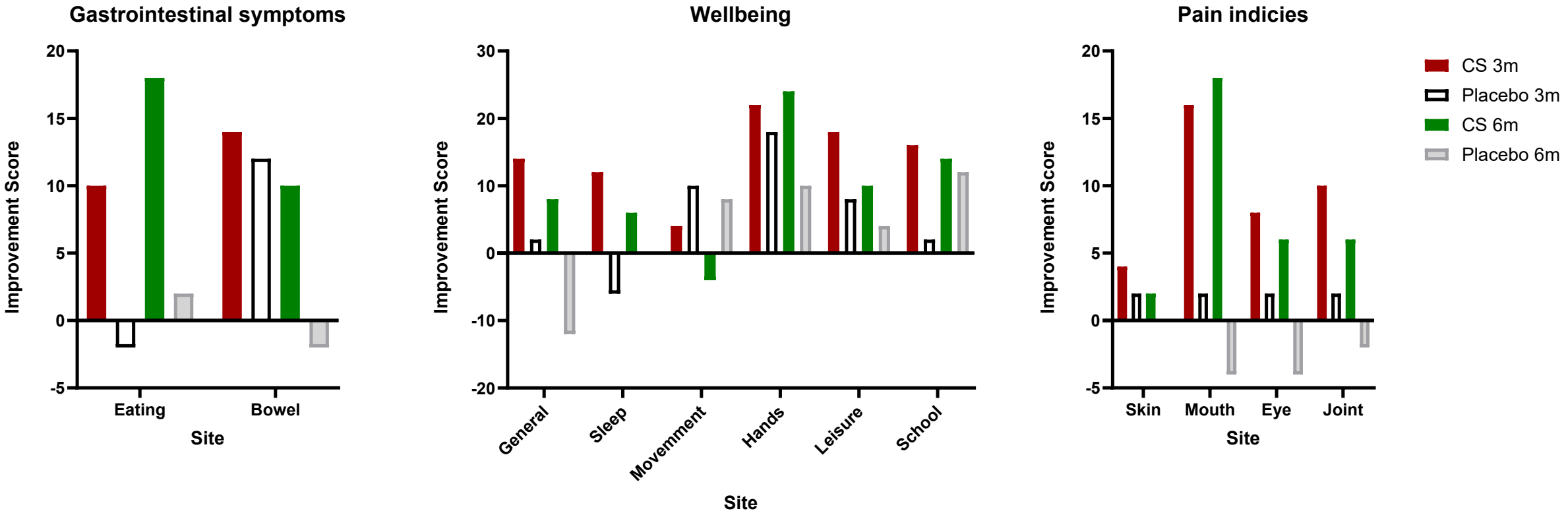




Itch improved at 3 months and remained stable at 6 months.

ISCOREB Improvement Scores – Patient reported data

Group 1 at 3 and 6 months



EB is a *systemic* disease and CORDStrom improves systemic symptoms.

Qualitative Results – Phase II

Interviewee	RDEB subtype	Participant code	Some improvement		Sequence	Favours treatment
			Treatment	Placebo		
Parent	Intermediate	G2	Yes	Yes	UC-MSc/Placebo	7/8 parents
		G4	Yes	No	UC-MSc/Placebo	
		B4	Yes	No	Placebo/UC-MSCs	
		G3	Yes	No	UC-MSc/Placebo	
		B1	Yes	No	Placebo/UC-MSCs	
	Severe	G5	Yes	No	Placebo/UC-MSCs	
		B3	Yes	No	Placebo/UC-MSCs	
		B5	Yes	No	Placebo/UC-MSCs	
Children	Intermediate	B4	Yes	Yes	Placebo/UC-MSCs	3/5 children
		G3	Yes	No	UC-MSc/Placebo	
		B1	Yes	No	Placebo/UC-MSCs	
		B3	Yes	No	Placebo/UC-MSCs	
	Severe	G1	Yes	Yes	UC-MSc/Placebo	

- 13 participants (8 parents & 5 children) completed interviews at both time-points
- 10 out of 13 had a clear difference in their responses in favour of UC-MSCs
- 3 were less clear
  1. (G1) Severe over 10 yrs patient who reported no or minimal changes
  2. (G2) Parent of a child under 1 yr who reported positive effect of the first infusion (UC-MSCs) which continued after the second infusion (placebo)
  3. (B4) Intermediate patient under 10 yrs who reported benefits in both periods. Their parent felt strongly in favour of UC-MSCs

Conclusion

- With itch reduction over time **IscorEB scores** have also **improved in both RDEB severe cohort** and **intermediate**
- There is **large improvement** in **iscorEB scores** in **RDEB-S** cohort at month-6 with improvement in overall score, patient & clinician scores and skin score
- The observed **skin improvement** and **wound closure** is likely to be **disease modifying, improving pain and quality of life** and **reducing** the long-term **risk of squamous cell carcinoma**

BBC

Trial gives hope to children suffering from painful skin condition

15 August 2025

Share

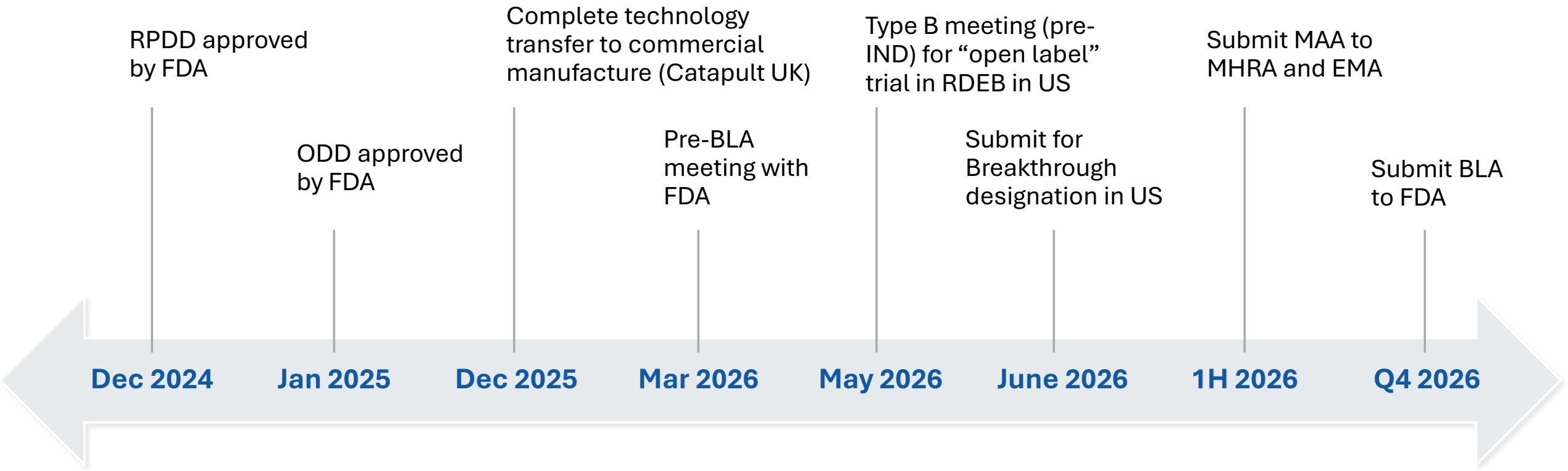
Watch Sharmila Collins, Dr Anna Martinez and the Freir family discuss the results of the Mission EB trial on BBC Breakfast on 15th August 2025.

Mum Jolita continued, “Gabrielius did really well on the trial. His wounds healed quicker, and his skin was less red and inflamed. His skin was also less itchy which also reduced him scratching. His sleep was also less disturbed as he didn’t need to have his special wound dressings changed as often, so this was a great benefit.”

Final Statements

- CORDStrom has proven beneficial treatment with no safety signals in patients with RDEB from age 6 months
- Intermediate and younger patients under the age of 10 saw the greater effect with improvement of their skin, pain and itch
- Severe and older patients over 10 did not show changes in their skin in the timeframe given however itch and pain improved greatly
- Results of the blinded qualitative analysis and clinical photographs were also consistent with these findings
- Early treatment with CORDStrom infusion in RDEB patients is disease-modifying particularly showing benefits in younger patients with milder disease
- Severe patients saw a clinically significant improvement in itch. This reduction in itch over time will likely lead to an improvement in wound closure and therefore reduce risk of squamous cell carcinoma later in life

CORDStrom for RDEB: Path To Market



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"

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 reimbursement per treatment:

- CORDStrom as *the **only systemic** treatment estimated* ~$700,000 per year
  - Potential TAM $USD:
    - 1000 RDEB cases - $0.7b
    - 3500 DEB cases - $2.4b
    - 12500 EB cases - $8.7b

Competitive Positioning

- VYJUVEK topical skin cream $750,000 per year
- 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.

CORDStrom Platform - Future Expanding Pipeline

- Selection of PCS for disease-specific potencies
  - OA: TGF-b / IGF-1 / FGF-2 / HGF / PGE2 — Partnership indication
  - EB: TSG6 / Wound healing / T cell suppression — Orphan
  - SLE: B cell suppression / T cell suppression / Wound healing — Orphan
  - Sjogren's & R-I xerostomia: GDNF / WNT1 / R-spondin 1 — Orphan
  - Intra-partum hypoxia: IDO / TGF-b — Orphan

- Selection of CPD (Cell Population Doublings)
  - CPD 15-20: wound healing
  - CPD 25-30: cytokine secretion

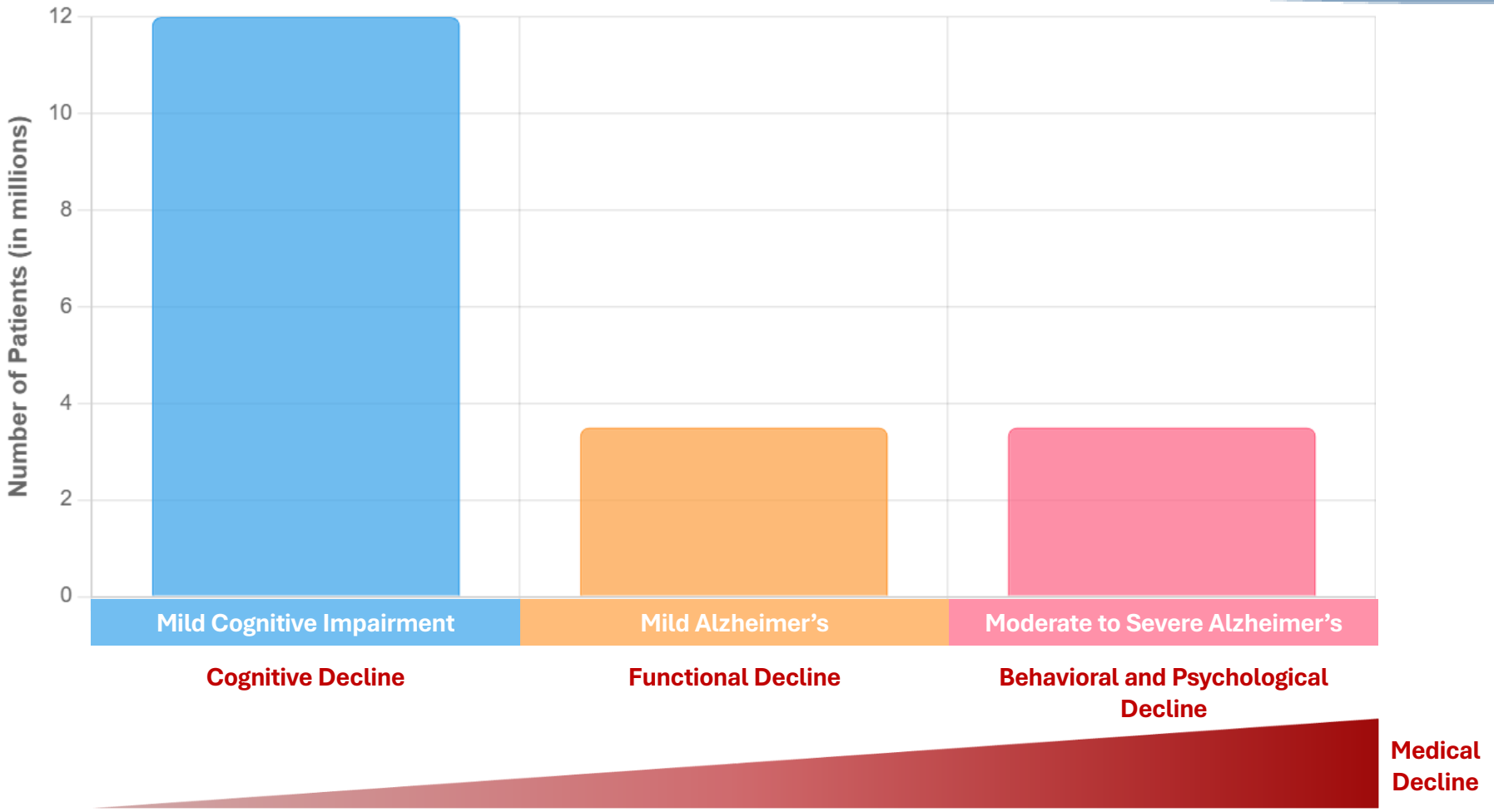
- Formulation
  - DMSO-free: Intranasal delivery route

- In Development
  - CORDStrom-Coll7a in RDEB – in development
  - CORDStrom-TRAIL in cancer – phase I/II complete and submitted for publication

XPro™

Approaching Alzheimer's as an Immunologic Disease

Millions of Patients in the US Need Better Solutions

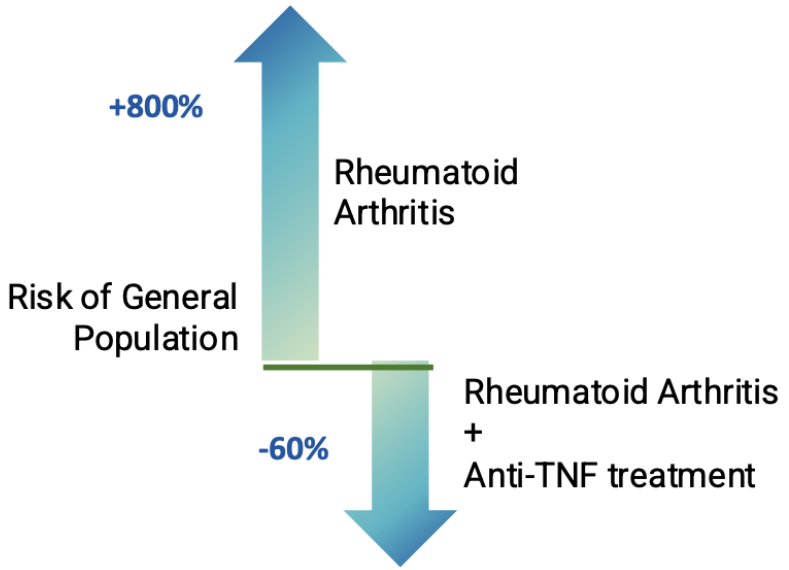


Note: These figures are approximate and based on a synthesis of recent data from various sources including the Alzheimer's Association, National Institute on Aging (NIA), and peer-reviewed studies (circa 2020-2025).

- **MCI (Mild Cognitive Impairment):** ~12 million (primarily all-cause MCI in adults 65+).
- **Mild AD (Alzheimer's Disease):** ~3.5 million (representing about 50% of total Alzheimer's cases).
- **Moderate & Severe AD:** ~3.5 million (representing about 50% of total Alzheimer's cases).

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

XPro™ in Alzheimer's Disease: Rationale and Background

Neuroinflammation in AD

- Recognized contributor to disease progression in AD[1]
- Associated with synaptic dysfunction/loss and cognitive impairment across the AD continuum[2]
- Contributes to brain inflammation and damage in Alzheimer's, worsening disease progression[3]

XPro™: Mechanism of Action

- Anti-inflammatory, next-generation TNF inhibitor
- Selective, brain-penetrant neutralizer of the soluble and proinflammatory form of tumor necrosis factor (solTNF)
- Safely and selectively inhibits inflammatory signaling
  - Does not cause immunosuppression

XPro™ in Alzheimer's

- Stopped Cognitive Decline in early AD patients
- Demonstrated safety in multiple studies in AD with zero cases of ARIA
- Dose dependent reduction in inflammatory cytokines in cerebrospinal fluid (CSF)
- Reduced key AD biomarkers of pTau217 and GFAP

[1]Jack CR Jr, et al. Alzheimers Dement. 2024.
[2]Taddei RN, et al. JAMA Neurol. 2023.
[3]Sánchez-Juan P, et al. Brain. 2024.

Six month, Randomized, Placebo-Controlled, Blinded Study of XPro™ in Early Alzheimer's with Biomarkers of Inflammation

MINDFuL
A study of inflammation in Alzheimer's disease

n= 208
2:1
XPro : Placebo

XPro1595
1 mg/kg weekly subQ injection

Placebo

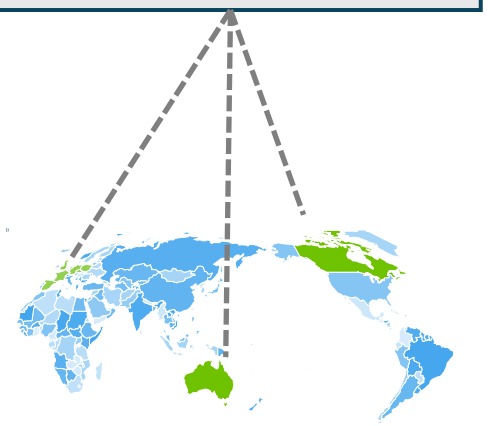
Primary Endpoint
- ❑ EMACC

Secondary Endpoints
- ❑ CDR
- ❑ Ecog
- ❑ ADL, NPI
- ❑ Blood
- ❑ MRI
- ❑ Safety

Baseline

Baseline	Week 6	Week 12	Week 18	Week 24
❑ EMACC	❑ EMACC	❑ EMACC	❑ EMACC	❑ EMACC
❑ Cognition		❑ Cognition		❑ Cognition
❑ Function		❑ Function		❑ Function
❑ Blood		❑ Blood		❑ Blood
❑ MRI		❑ MRI		❑ MRI

Key Enrollment Criterion
- ❑ Early AD (50-85 yrs)
- ❑ Amyloid positive
- ❑ CDR (0.5 or 1)
- ❑ MMSE ≥ 22
- ❑ Ecog >1.5
- ❑ One Inflammatory Biomarker:
  - ❑ hsCRP (1.5 mg/L)
  - ❑ ESR (10 mmg/hr)
  - ❑ HbA1c (6%)
  - ❑ APOE4+

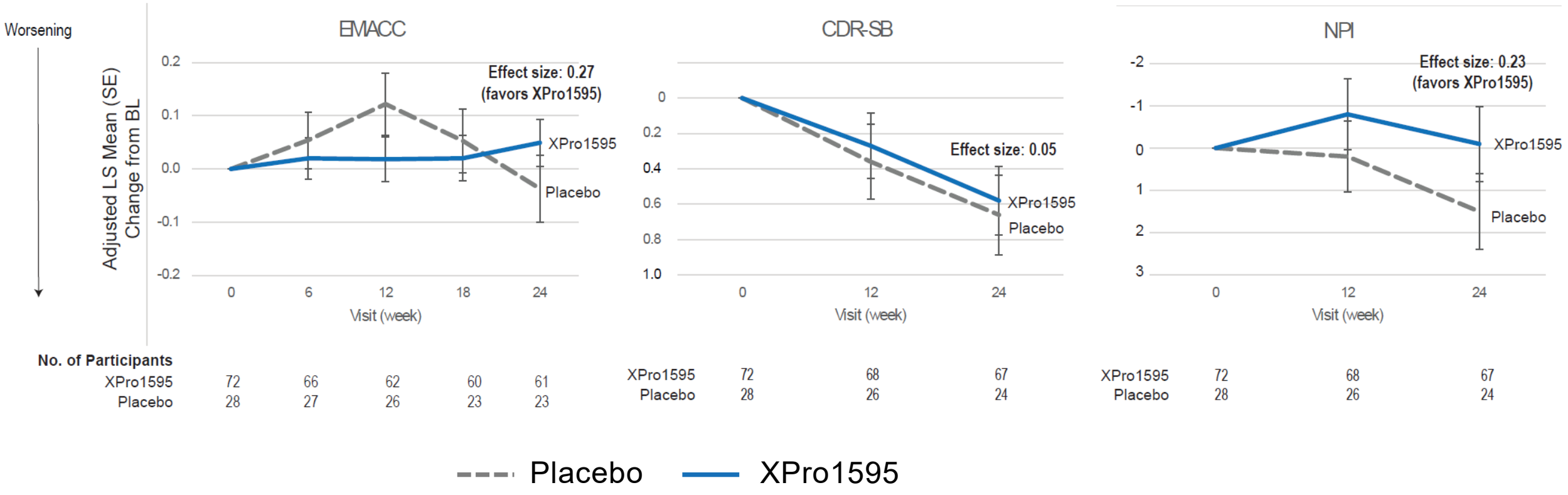


XPro™ Halted Cognitive Decline* in Highly Inflamed Alzheimer's Patients

Change From Baseline

medRxiv

* The complete analysis of the MINDFuL trial is available at MedRxiv (https://www.medrxiv.org/content/10.1101/2025.09.24.25336496v1)

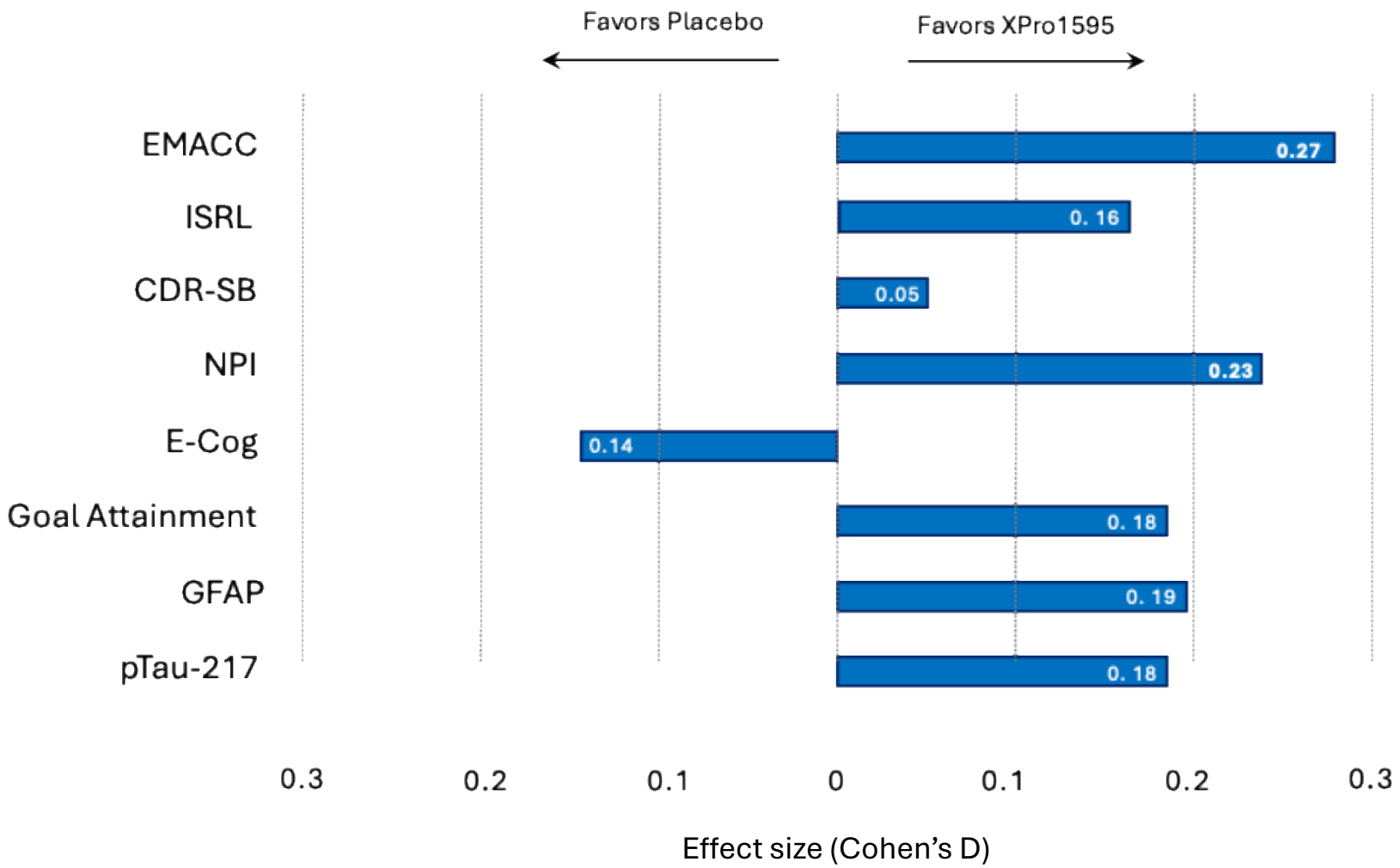


EMACC: LS Mean Diff (SE): 0.086 (0.0603), **90% CI:** -0.0146, 0.1857**, p-value:** 0.1594. **CDR-SB: LS Mean Diff (SE):** -0.08 (0.307), **90% CI:** -0.593, 0.426**, p-value:** 0.7859.

NPI: LS Mean Diff (SE): -1.6 (1.25), **90% CI:** -3.71, 0.47**, p-value:** 0.2003

Enriched Population: Most Endpoints Favor Treatment with XPro™

Depicted as absolute effect sizes (Cohen's D)



EMACC: Early and Mild Alzheimer's Cognitive Composite. ISRL: International Shopping List Delayed Recall. CDR-SB: Clinical Dementia Rating – Sum of Boxes. NPI: Neuropsychiatric Inventory. E-Cog: Everyday Cognition. GFAP: glial fibrillary acidic protein

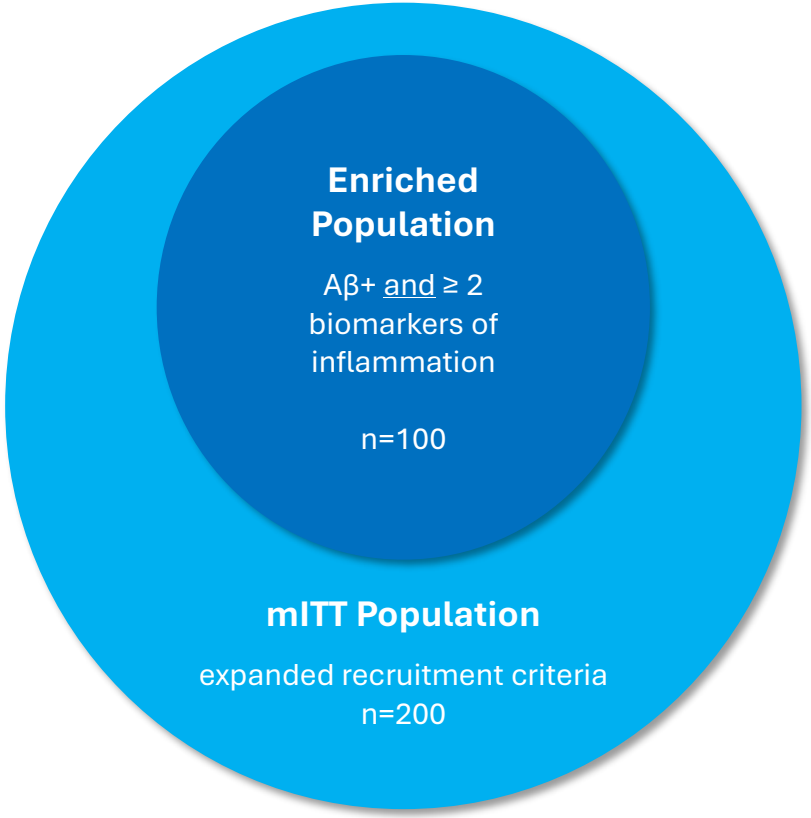
Why Did We Miss? Population Changes Due to Operational Limitations

Original Key Inclusion Criteria: Enriched Protocol

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

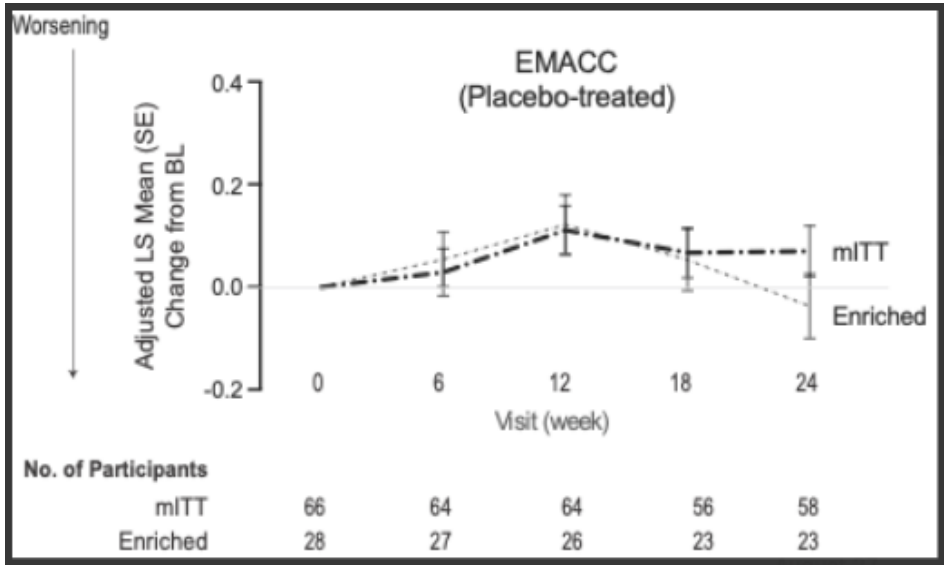
Expanded Inclusion Criteria: Revised Protocol

- Age (50 - 85 years)
- Diagnosis of MCI or mild AD (NIA-AA stages 3-4)[1]
- ~~Amyloid-beta positive (Aβ+)~~
- 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.



XPro™: Safe, Effective, and Poised for Targeted Phase 3 Success

ARIA-E **Safety:**

- *Zero cases of ARIA* (a major issue with amyloid drugs) despite high-risk patients
- No serious treatment-related adverse events

Efficacy:

- Phase 2 showed **preserved cognition** and improved biomarkers in patients with **neuroinflammatory markers**
- Broader population analysis diluted results due to **low placebo decline**

Path Forward:

- Phase 3 will focus on **biomarker-confirmed, inflammation-enriched Alzheimer's patients** — where XPro™ delivers its strongest benefit.

Next steps: End of Phase 2 meeting with FDA for regulatory alignment to validate the enriched population in a fully powered trial.

ARIA-E = amyloid-related imaging abnormalities-edema/effusions; ARIA-H=amyloid-related imaging abnormalities-microhemorrhages/hemosiderin deposits; SAF = Safety Analysis Set

Execution Roadmap: Regulatory & Clinical Milestones

CORDStrom™ (RDEB, rare pediatric)

- ✓ Phase 2 Pediatric Trial Completed
- ✓ RPDD / ODD Granted
- → MAA Submission (UK) – Mid 2026
- → BLA Submission (US) – Late 2026
- → PRV Eligibility & Launch Readiness – 2027

XPro™ (Early Alzheimer's, biomarker-enriched)

- ✓ Phase 2 Study Completed
- ✓ Biomarker Validation Achieved (pTau217, GFAP)
- ✓ Patient Population Defined
- → End-of-Phase-2 Meeting – Q1 2026
- → CMC Phase 3 Optimization
- → Phase 3 Initiation – 2027

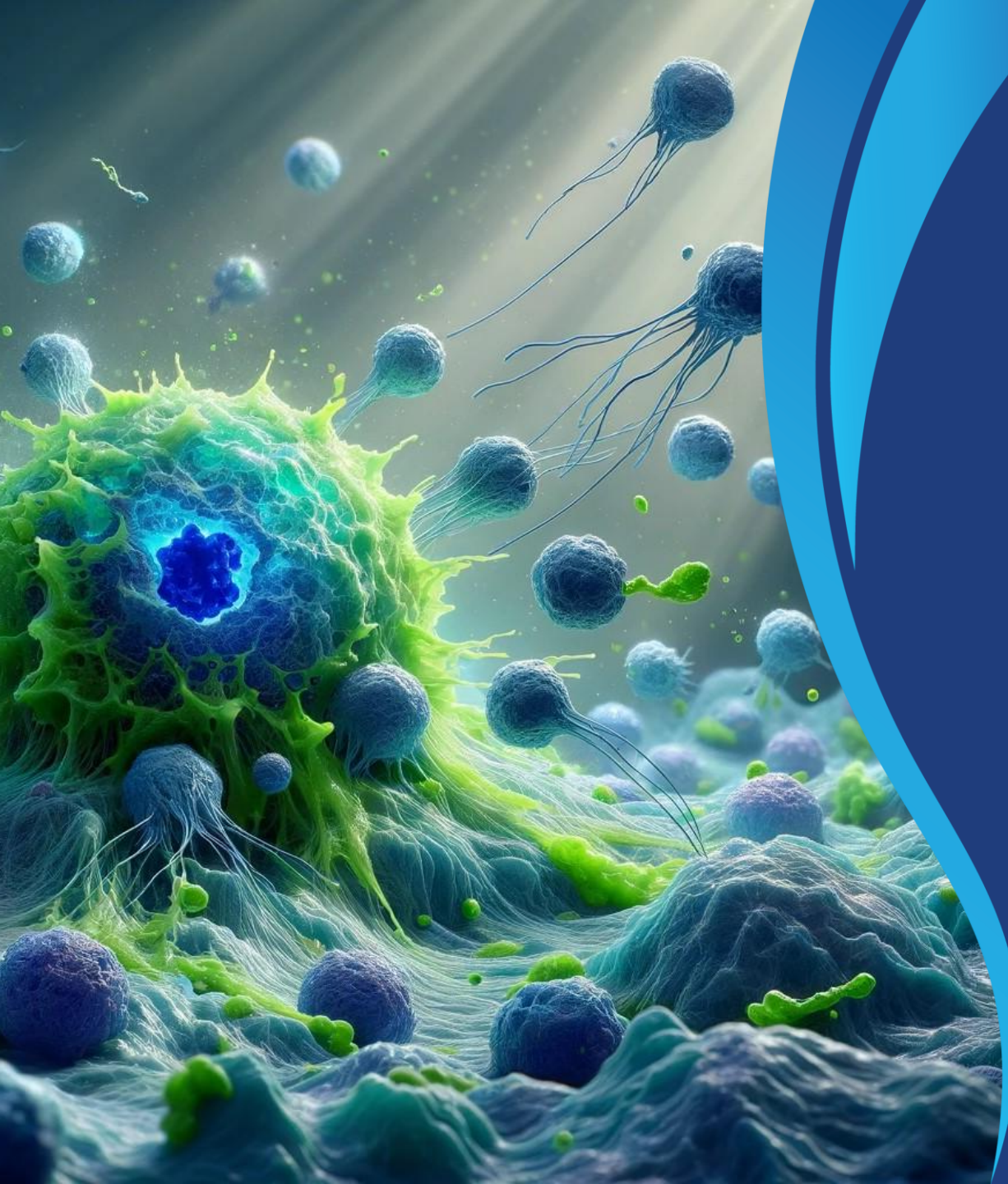
Manufacturing & CMC

- Commercial tech transfer nearly complete (Catapult UK)
- Scalable GMP infrastructure in place

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

Investment Highlights

- ✓ Two late-stage programs with strong safety and efficacy signals
- ✓ First-in-class systemic therapy for RDEB and novel Alzheimer's mechanism
- ✓ Multiple near-term regulatory milestones driving value
- ✓ Capital-efficient development model with strong partnerships



For More Information:

INmune Bio Inc.
225 NE Mizner Blvd. Ste. 640
Boca Raton, FL 33432
(561) 710-0512
info@inmunebio.com

INMB (Nasdaq)