INmuneBio
INNATE IMMUNITY

Corporate Presentation

July 2025

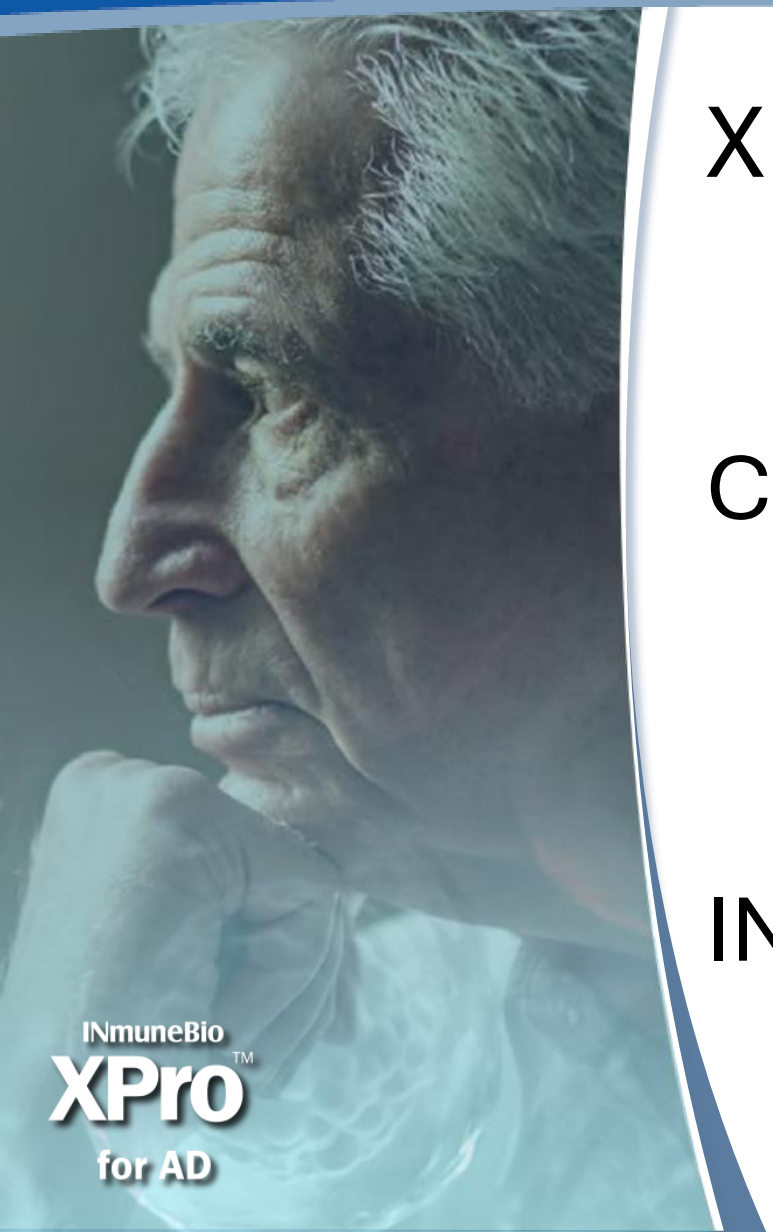
NASDAQ: INMB

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 risks contained in the section of this prospectus entitled “Risk Factors”) 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 in this press release 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 of this release.

Three Platforms Modulating the Innate Immune System



XPro™

- TNF is the master regulator of the innate immune system and causes TNF dysfunction in many neurologic disease.
- XPro™ is designed to reestablish normal TNF function to treat disease.

CORDStrom™

- Mesenchymal Stromal cells (MSC) are recognized for their immunomodulatory, anti-inflammatory and wound healing properties with potential to treat a diverse set of diseases.
- CORDStrom™ technology is designed to solve the limitations of previous MSC therapies and is currently in development for a rare pediatric disease and other indications.

INKmune™

- Natural Killer cells are responsible for detecting and eliminating cancer cells and become dysfunctional with age.
- INKmune™ works within the body to activate the patients own NK cells against multiple forms of cancer.

XPro™

Approaching Alzheimer's as an Immunologic Disease

Strong Evidence for XPro™ to Treat Alzheimer's Disease

Humans and Animals

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

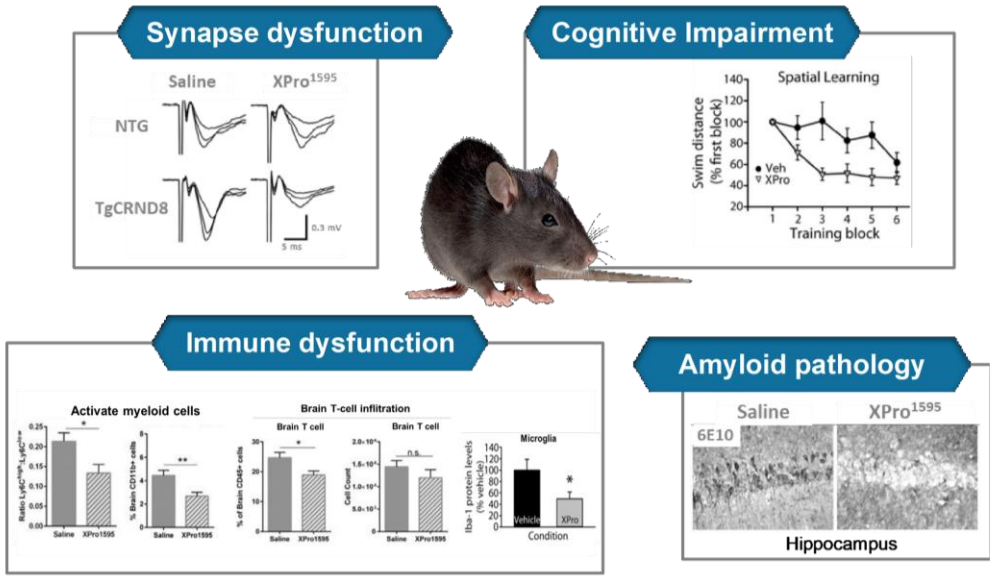
TNF increases amyloid[7,8] and Tau[9-12] TNF causes cell loss and cognitive impairment[13]

TNF inhibitors reduce risk of AD

Anti-TNF therapies[1] reduce the risk of AD in humans by up to:

72%

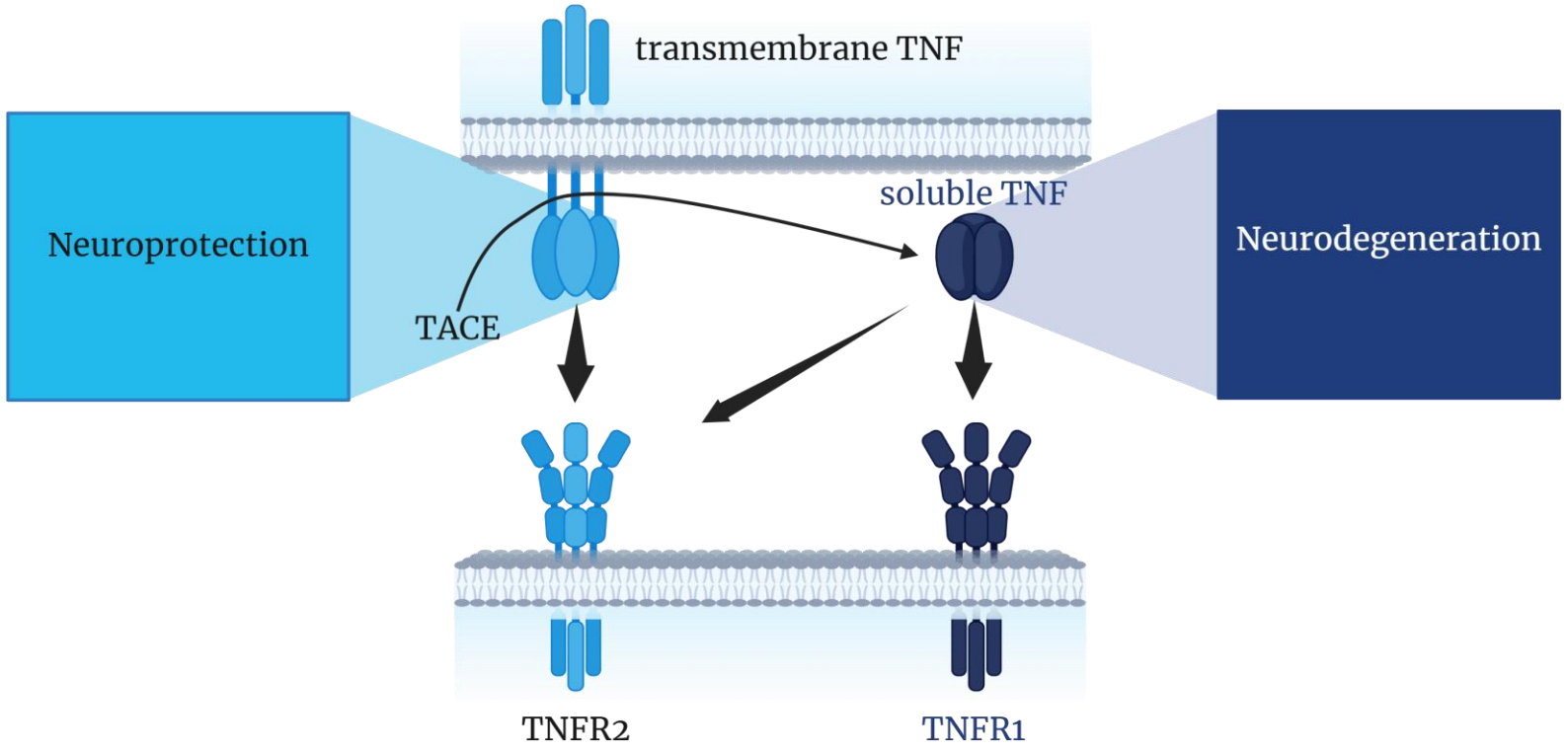
XPro™ efficacy in AD models



Efficacy has been shown in 3xTgAD, 5xFAD, TgCRND8 and aged mice

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

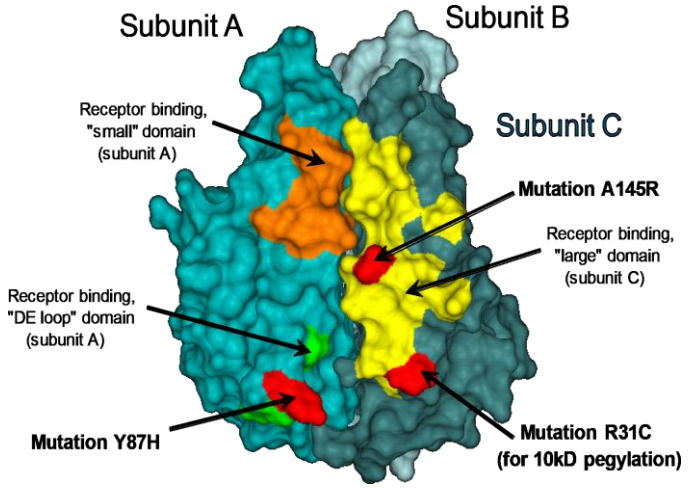
TNF Biology – Two Ligands with Opposite Effects



Created with BioRender.com.

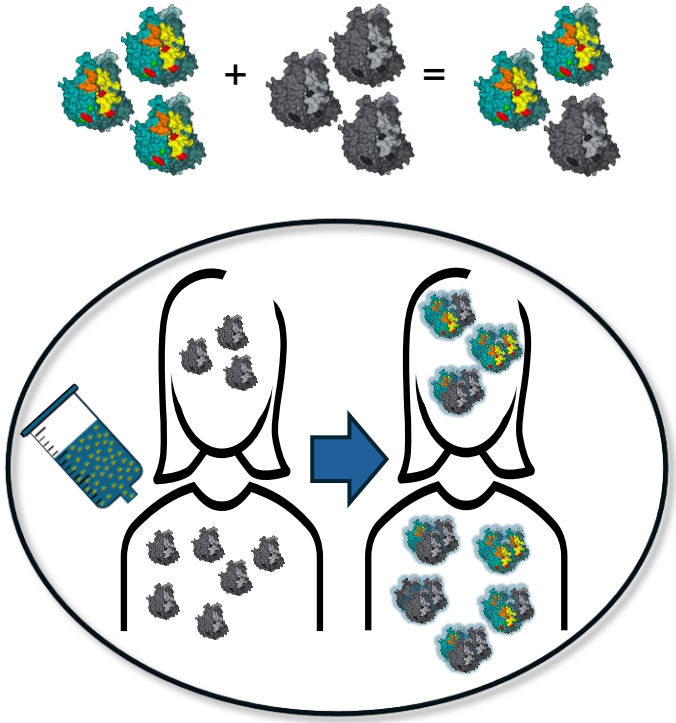
XPro™: A TNF Inhibitor Designed to Treat Neurologic Disease

XPro™

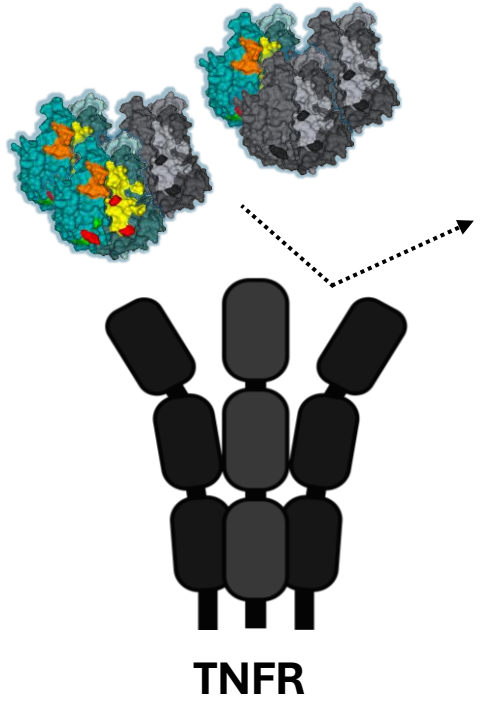


A dominant-negative* protein identical to the human soluble TNF monomer but has mutations in the receptor binding domain that inhibit solTNF from binding to the TNFR

forms inactive heterotrimers



that cannot bind to TNFR

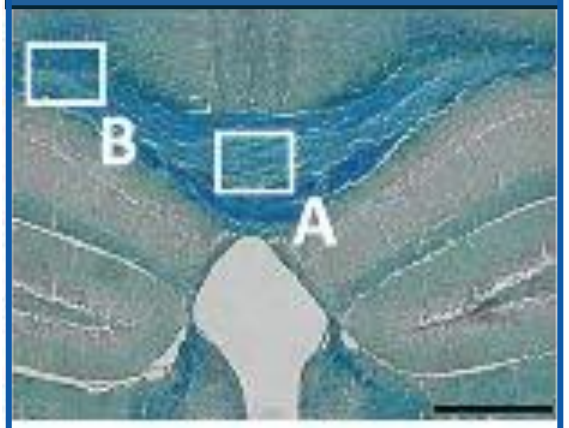


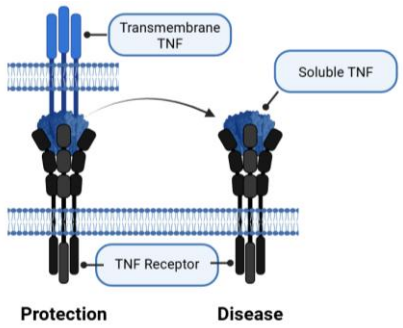
*Dominant-Negative in genetics: A mutation producing a rogue protein that interferes with the function of the native protein.

Steed, *Science* 2003; Zalevsky, JI 2007; McCoy, JNeuro 2006; Vujanovic, Blood 2010; Brambilla, Brain 2011; Khera, Clin Exp Immunol 2012

Selective Inhibition of Soluble TNF is Necessary to Treat AD

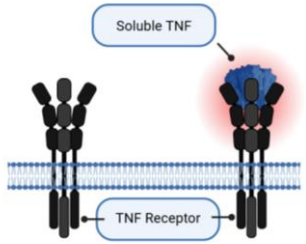
Normal





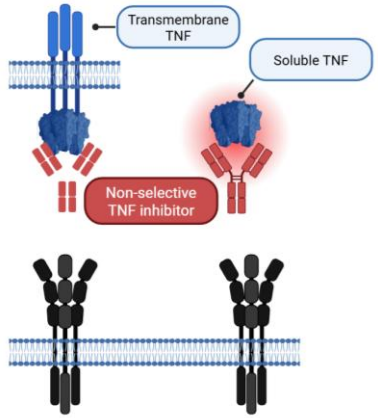
Disease

(Model of Multiple Sclerosis)



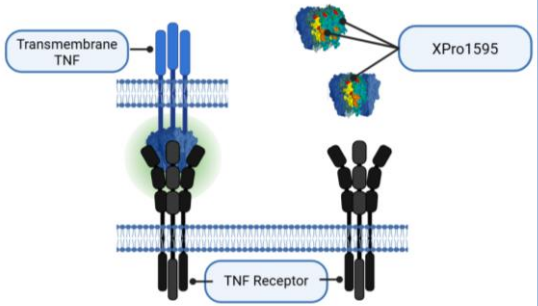
Etanercept

Non-selective TNF inhibition



XPro™

Soluble TNF inhibition



Adapted from: Karamita et al., 2017. Illustrations generated with bioRender.

Phase 1b Trial in Alzheimer's Patients with Biomarkers of Inflammation

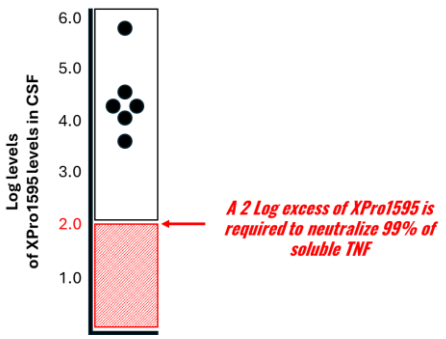
Study Design
- ❑ Open label
- ❑ Three dose (0.3, 0.6, 1.0 mg/kg)
- ❑ 12-week

Key Enrollment Criteria
- ❑ AD Diagnosis
- ❑ Biomarker of inflammation

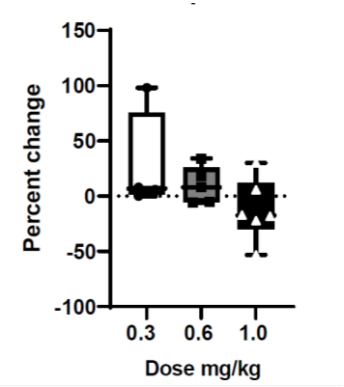
Goals
- ❑ Safety
- ❑ Dose Identification
- ❑ Proof of Biology

Key Results

XPro1595 gets into the brain

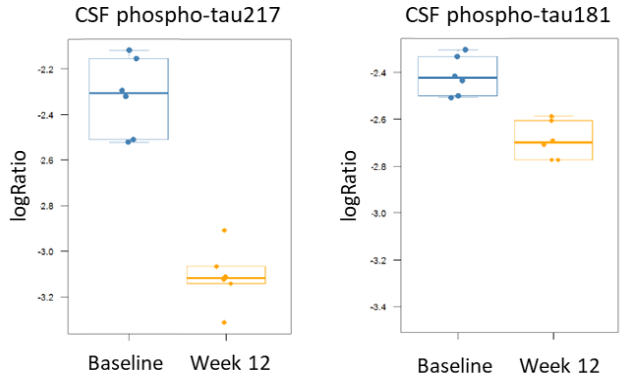


XPro1595 dose-dependently reduced neuroinflammation



15% reduction in Neuroinflammation

XPro1595 reduced biomarkers of AD pathology



43% reduction in pTau-217
2% reduction in pTau181

6 month, Randomized, Placebo-Controlled, Double-Blind Study of XPro1595 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

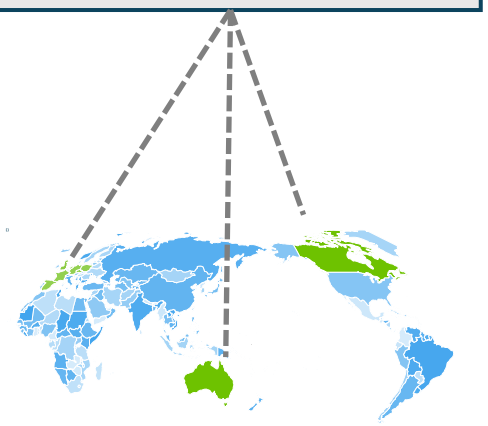
Primary Endpoint
- ❑ EMACC

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+



EMACC and CDR: Primary End-point for Early AD Clinical Trials

	CDR	EMACC
Clinically derived to stage AD	+	
Empirically derived to measure cognitive change in Early AD		+
Clinically validated measurements	+	+
No floor or ceiling effects		+
Lower variance and shorter retest intervals provides smoother measure of cognitive change		+
Greater dynamic range allows measure of stable, worsening or improved cognition		+
Allows for shorter and smaller clinical trials		+

MINDFuL
A study of inflammation in Alzheimer's disease

The Early/ Mild Alzheimer's Cognitive Composite (EMACC)

EMACC provides an accurate cognitive assessment for early Alzheimer's disease

EMACC measures cognitive changes in early AD

Developed in 2017 to measure cognitive changes that occur in early Alzheimer's disease

Quantitative/Objective/Sensitive

EMACC objectively measures cognitive performance and is sensitive to the effects of disease progression and treatments

Comprised of 6 validated tests

Word list learning, digit span, fluency, trail making, coding

EMACC improves signal detection in early AD which allows clinical trials to enroll fewer patients for a shorter duration thereby reducing trial costs and clinical development timelines

Sources: ADNI (https://adni.loni.usc.edu/)

XPro™

Phase 2 Study Results

XPro™ for Early AD: 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]
- Implicated in neurotoxic gliosis and tau-related neurodegeneration downstream of amyloid-beta (Aβ) in AD[3]

XPro™ Mechanism of Action

- 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
  - Does not interfere with immune homeostasis

XPro™ in AD: Phase 1

- Demonstrated safety in Phase 1b study in AD (n=20)
- Dose dependent reduction in inflammatory cytokines in cerebrospinal fluid (CSF)
- Dose dependent modulation of synaptic proteins differentially affected in AD[5]
- Signal for target engagement in gray matter cortices most frequently impacted by AD pathology[6]

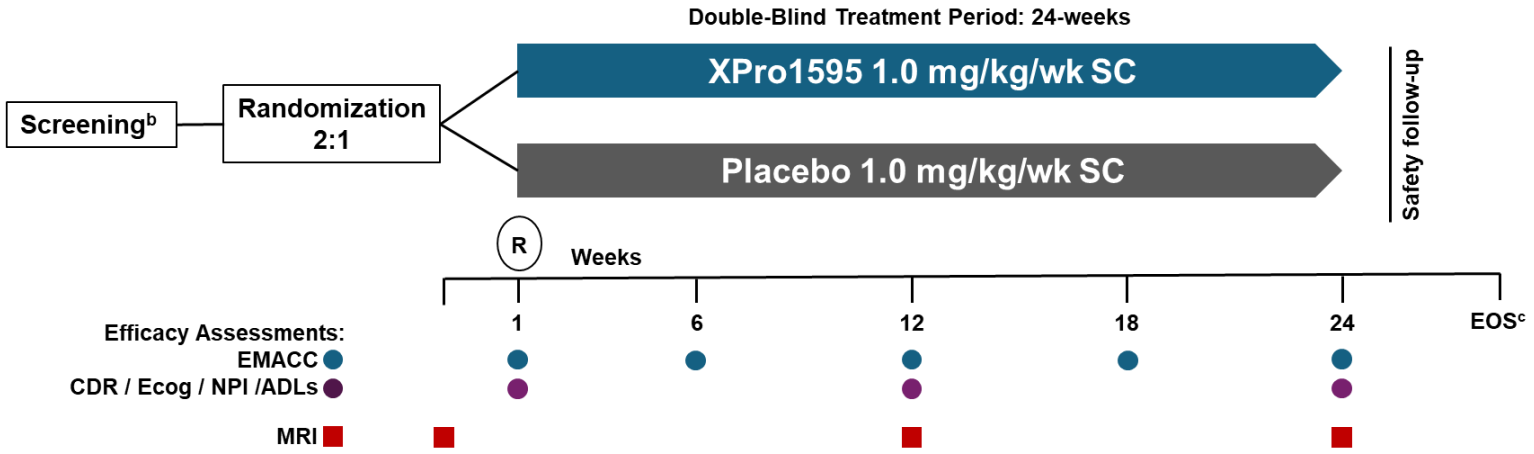
[1]Jack CR Jr, et al. Alzheimers Dement. 2024. [2]Taddei RN, et al. JAMA Neurol. 2023. [3]Sánchez-Juan P, et al. Brain. 2024. [5]Pope P, et al. Alzheimer's & Dementia, 2024. 20(S8): p. e095343. [6]Pope, P., et al. Alzheimer's Dement., 19: e083229.

XPro™ for Early AD: Phase 2 study Design

MINDFuL: A Phase 2, Multicenter, Randomized, Double-Blind, Placebo-Controlled Trial in Participants with Early Alzheimer's Disease

Key Eligibility Criteria

- Age (50-85 years)
- Diagnosis of MCI or mAD[1]
- Amyloid-beta (Aβ) positive
- MMSE ≥ 22
- CDR global rating: 0.5 or 1.0
- ≥ 1 blood biomarker of inflammation/immune dysfunction[a]
- MRI: No evidence of ARIA, SVD, diffuse WM disease or non-AD neurodegenerative disease



[1]Jack CR Jr, et al. Alzheimers Dement. 2018

[a]Blood biomarkers: hsCRP>1.5 mg/L or erythrocyte sedimentation rate (ESR) >10mm/hr or HbA1C >6.0% or APOE ε4 (≥1 allele). [b]Screening (up to 45 days). [c]EOS safety follow-up visit (28-days after last dose).

E-Cog=Everyday Cognition Scale; ADL=Alzheimer's Disease Cooperative Study – Activities of Daily Living (ADCS-ADL and ADCS-MCI-ADL); ARIA=Amyloid-related Imaging Abnormalities; CDR=Clinical Dementia Rating Scale; EMACC=Early and Mild Alzheimer's Cognitive Composite; mAD=Mild Alzheimer's Disease; MCI=Mild Cognitive Impairment; MRI=Magnetic Resonance Imaging; NPI=Neuropsychiatric Inventory-12; R=Randomization; SVD=Small Vessel Disease; WM=white matter

XPro™ for Early AD: Phase 2 study Design

Trial Endpoints	Change from Baseline (CFB) to Week-24
Primary Endpoint	• **Early and Mild Alzheimer's Cognitive Composite (EMACC)**
Key Secondary Endpoints	• Clinical Dementia Rating Scale – Sum of Boxes (CDR-SB)
	• Everyday Cognition Scale (E-Cog)
	• Neuropsychiatric Inventory (NPI-12) study partner items
Secondary Endpoints	• Alzheimer's Disease Cooperative Study – Activities of Daily Living (ADCS-ADL and ADCS-MCI-ADL)
	• Blood Biomarkers of AD pathology (pTau-217) and neuroinflammation (GFAP)
	• MRI Brain volumetrics

XPro™ for Early AD: Phase 2 Study Results

Baseline Demographics (SAF Population)[a]

Demographic	Placebo (n=67)	XPro™ (n=139)	Total (N=206)
Age, years	72.1 (6.75)	72.7 (6.40)	72.5 (6.51)
Sex , n (%)			
Female	35 (52.2)	70 (50.4)	105 (51.0)
Race, n (%)			
White	65 (97.0)	131 (94.2)	198 (95.1)
Asian	0	5 (3.6)	5 (2.4)
Not Reported	1 (1.5)	0	4 (1.9)
Other	1 (1.5)	3 (2.2)	1 (0.5)
Ethnicity, n (%)			
Hispanic or Latino	0	2 (1.4)	2 (1.0)
Not Hispanic or Latino	64 (95.5)	134 (96.4)	192 (96.1)
Not Reported	3 (4.5)	3 (3.6)	6 (2.9)
APOE ε4 genotype, n (%)*			
n	66	134	200
Noncarrier	19 (28.8)	26 (19.4)	45 (22.5)
Carrier	37 (56.1)	80 (59.7)	117 (58.5)
Heterozygote	33 (50.0)	69 (51.5)	102 (51.0)
Homozygote	4 (6.1)	11 (8.2)	15 (7.5)
Unknown	10 (15.2)	28 (20.9)	38 (19.0)

[a]SAF=Safety Analysis Set (all patients who have received any amount of XPro1595 or Placebo)
*APOE genotype information from Modified Intention to Treat (mITT) population

XPro™ for Early AD: Phase 2 Study Results

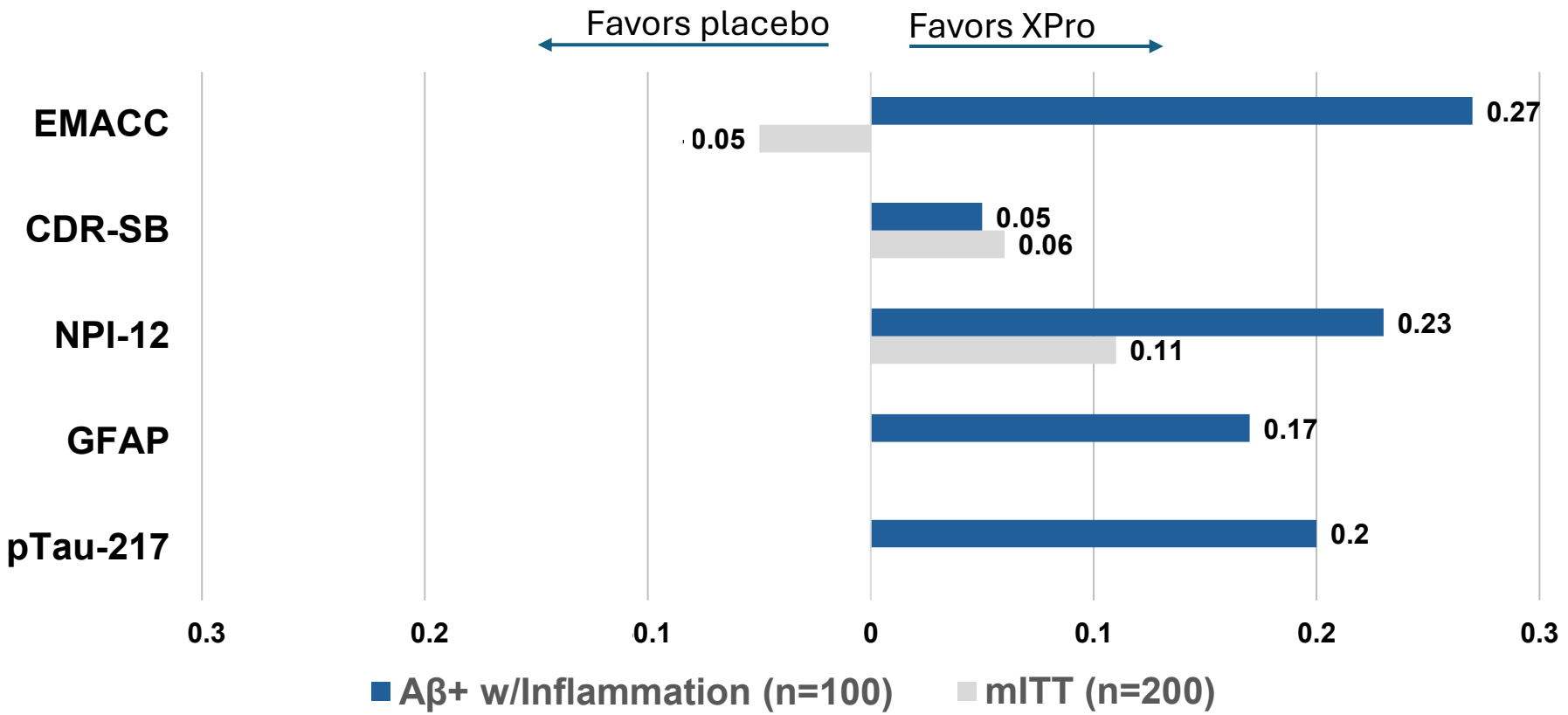
Baseline Disease Characteristics (SAF Population)[a]

Characteristic	Placebo (n=67)	XPro™ (n=139)	Total (N=206)
Time (yrs) since AD diagnosis, n	64	135	199
Mean (SD)	1.05 (1.42)	1.20 (1.52)	1.15 (1.49)
Diagnosis (randomization), n (%)			
MCI	31 (46.3)	62 (44.6)	93 (45.1)
mAD	36 (53.7)	77 (55.4)	113 (54.9)
Concomitant AD medication, n (%)			
Acetylcholinesterase inhibitors	20 (29.9)	43 (31.6)	63 (30.6)
Memantine	6 (9.0)	10 (7.4)	16 (7.8)
MMSE, SAF (screening)	26.0 (2.19)	25.7 (2.40)	25.8 (2.33)
Modified Intention to Treat (mITT) Population	**Placebo (n=66)**	**XPro™ (n=139)**	**Total (n=200)**
Clinical Scores, mean (SD)			
EMACC (day-1)	0.011 (0.6918)	-0.011 (0.7432)	NA
CDR-SB (day-1)	2.99 (1.61)	3.08 (1.49)	3.03 (1.55)
E-Cog	2.18 (0.67)	2.17 (0.67)	2.28 (0.73)
Enrichment Biomarker Positivity, n (%)			
hsCRP > 1.5 mg/L	12 (18.2)	39 (29.1)	51 (25.5)
ESR > 10 mm/hr	42 (63.6)	89 (66.4)	131 (65.5)
HbA1c > 6.0 DCCT%	16 (24.2)	34 (25.4)	50 (25.0)
APOE ε4 Carriers	38 (57.6)	80 (59.7)	118 (59.0)

[a]SAF=Safety Analysis Set (all patients who have received any amount of XPro1595 or Placebo)

XPro™ for Early AD: Phase 2 study Results

Key Endpoint Effect Sizes (Cohen's d)*



Aβ+ w/Inflammation = Amyloid-beta positive with ≥2 enrichment biomarkers of inflammation in blood

* Absolute effect sizes displayed in the direction of favored treatment

XPro™ for Early AD: Phase 2 Study Results - Safety

Safety: Treatment Emergent Adverse Events (TEAEs): Safety Analyses Set[a]

Event, n (%)	Placebo (n=67)	XPro™ (n=139)	Total (N=206)
Any TEAE [1]	59 (88.1)	131 (94.2)	190 (92.2)
Any TEAE by Maximum Severity [2]			
Mild	34 (50.7)	73 (52.5)	107 (51.9)
Moderate	22 (32.8)	56 (40.3)	78 (37.9)
Severe	3 (4.5)	2 (1.4)	5 (2.4)
Any TEAE Related to Study Treatment	19 (28.4)	118 (84.9)	137 (66.5)
Any Serious TEAE	5 (7.5)	8 (5.8)	13 (6.3)
Any Treatment-Related Serious TEAE	0	2 (1.4)	2 (1.0)
Any TEAE of Special Interest (Hypersensitivity / Injection Site Reaction	7 (10.4)	112 (80.6)	119 (57.8)
Any TEAE of Hypersensitivity	3 (4.5)	43 (30.9)	46 (22.3)
Any TEAE of Injection Site Reaction	4 (6.0)	111 (79.9)	115 (55.8)
Any TEAE Leading to Treatment Discontinuation	2 (3.0)	12 (8.6)	14 (6.8)
Any TEAE Leading to Study Withdrawal	2 (3.0)	12 (8.6)	14 (6.8)
Any TEAE with Fatal Outcome	0	0	0

XPro™ for Early AD: Phase 2 Study Results - Safety

Safety: Injection Site Reactions (ISRs): Safety Analyses Set[a]			
Event, n (%)	**Placebo (n=67)**	**XPro™ (n=139)**	**Total (N=206)**
Hypersensitivity / Injection Site Reaction	7 (10.4)	112 (80.6)	119 (57.8)
Hypersensitivity	3 (4.5)	43 (30.9)	46 (22.3)
Injection Site Reaction	4 (6.0)	111 (79.9)	115 (55.8)

XPro™ for Early AD: Phase 2 Study Results - ARIA

Safety: Amyloid-Related Imaging Abnormalities (ARIA)

Safety Endpoints: Imaging

- Comprehensive imaging protocol and review criteria focused on detection and grading of safety events in the brain

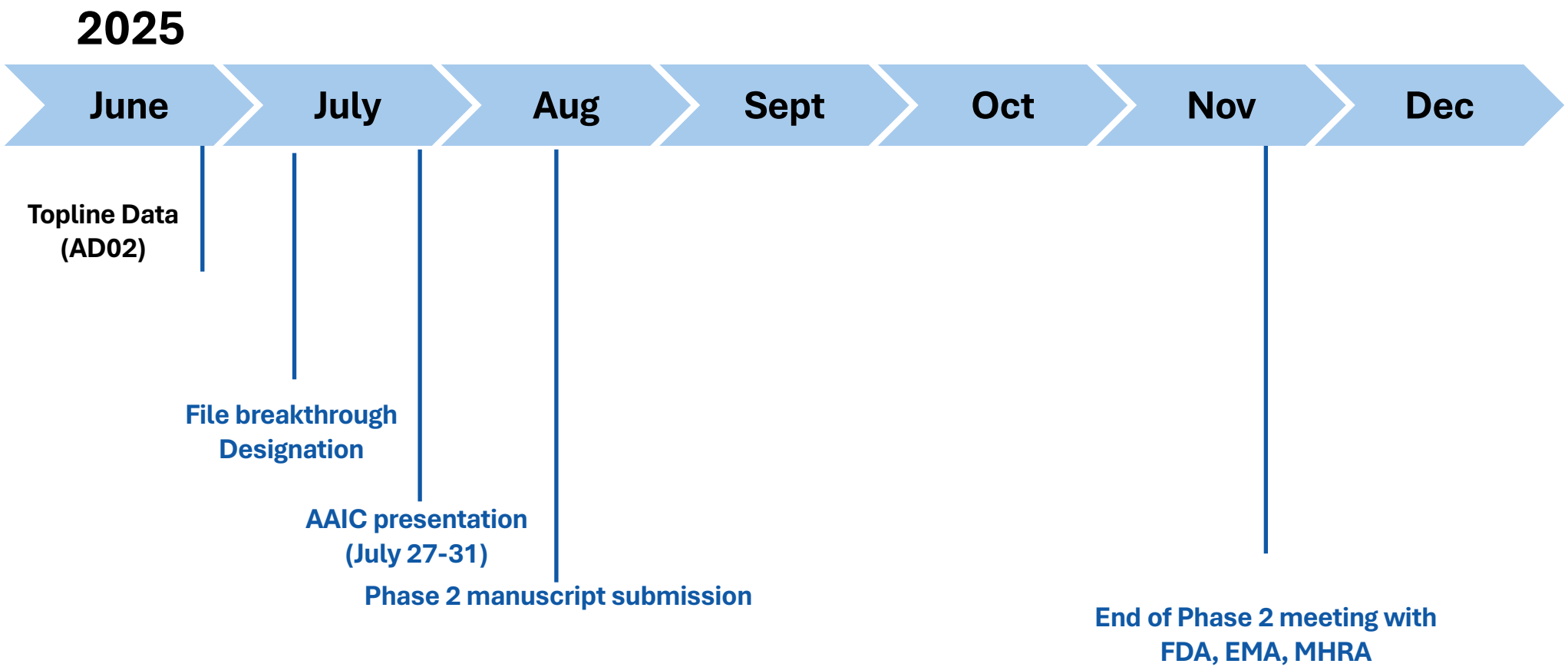
	Placebo (n=67)		XPro™ (n=139)	
	WK12	WK24	WK12	WK24
ARIA-E	0	0	0	0
ARIA-H	0	0	0	0
New Cerebral Microbleed Count	2	3	2	3
New Cortical Superficial Siderosis	0	0	0	0
New Focal Subarachnoid Hemorrhage	0	0	0	0
Intracerebral Hemorrhage Presence	0	0	0	0

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

XPro™ for Early AD: Summary and Conclusions

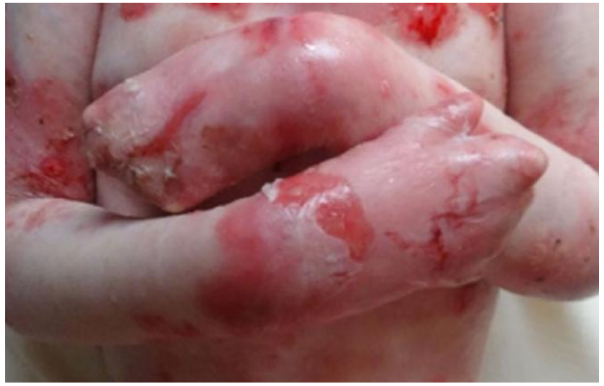
- MINDFuL was a well designed and well executed clinical trial in Early AD
  - ✓ Successful enrollment of a well-balanced cohort of participants with MCI or mAD
  - ✓ Objective measure of cognitive performance with improved sensitivity for detection of discrete changes as the primary outcome
- The primary endpoint was not met
- Study results:
  - ✓ Consistent absolute effect sizes indicative of positive effects attributable to treatment with XPro™
  - ✓ Identified the population of AD patients most likely to respond to treatment with XPro™ (i.e., Aβ+ patients with biomarker evidence of elevated inflammation)
- Safety outcomes indicated good safety and tolerability profile in participants with Early AD
  - ✓ Predominantly mild to moderate adverse events (AEs) across both study groups
  - ✓ No ARIA or neuroimaging AEs
  - ✓ No deaths

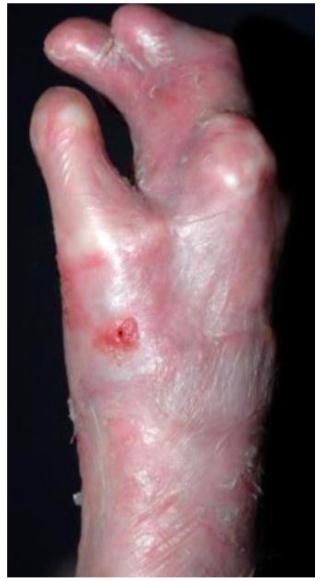
XPro™ Anticipated Next Steps and Timeline

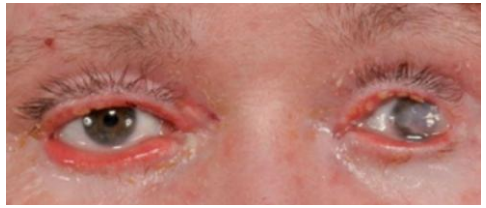


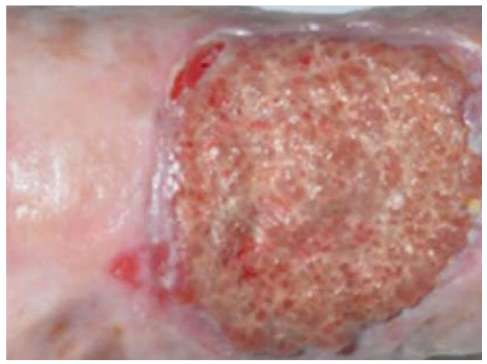

INmuneBio
CORDStrom™
for RDEB

RDEB – An Ultra-Rare Genetic Disease with Significant Unmet Need









- RDEB is a severe form of epidermolysis bullosa (EB), a rare disease that causes severe skin fragility, itch and chronic pain
- RDEB is caused by mutations in the *COL7A1* gene that makes type VII collagen, a protein that holds the layers of skin together
- Children with RDEB have skin that is damaged by even the smallest amount of friction which causes severe blistering, deep wounds, and scars
- There are limited options available for treatment, none that adequately meet the needs of patients, and the condition gets worse over time, with most children reliant on a wheelchair as they move into their teenage years
- Many of those with RDEB will also go on to develop aggressive life-threatening skin cancer in adulthood caused by the accumulated damage to their skin
- Krystal Biotech's VYJUVEK launch in DEB is off to an impressive start (~$84M net revenue in Q3 '24); CORDStrom is potentially the first systemic therapy, with itch benefit as a key differentiating factor, potential for use as an adjunctive therapy
- It is estimated that more than 4000 people suffer from RDEB in the US, UK and EU, representing a > $1B peak sales opportunity

*Recessive Dystrophic Epidermolysis Bullosa (RDEB)

CORDStrom Platform Overview

Investigational disease-modifying treatment for recessive dystrophic epidermolysis bullosa (RDEB)

CORDStrom Overview

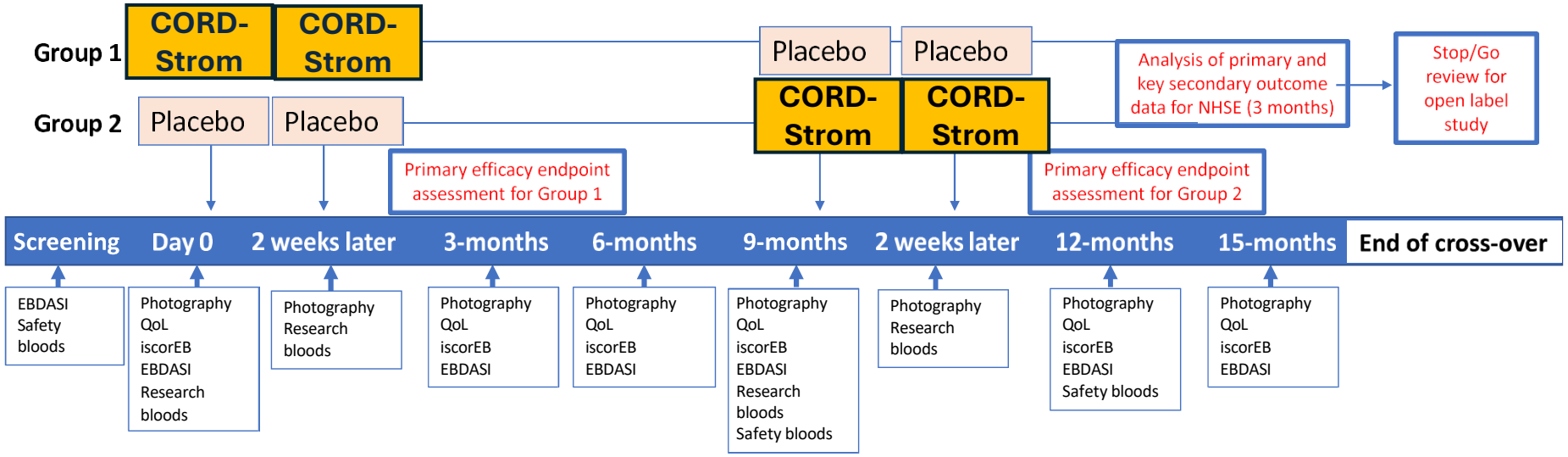
- CORDStrom is an innovative cellular medicine, comprising allogeneic, pooled human umbilical cord-derived mesenchymal stromal cells (hucMSCs) formulated for injection or infusion.
- CORDStrom addresses challenges related to the source, identity, heterogeneity, and manufacturing costs of MSCs, positioning them as a viable drug platform.
- The FDA has awarded CORDStrom both the Rare Pediatric Disease and Orphan Drug Designation, qualifying it for a Priority Review Voucher post-FDA approval.

Mission EB Phase 2 Trial

- Phase 2 Trial completed by investigators at Great Ormond Street Hospital for Children & Birmingham Children's Hospital in the UK and primarily funded by grant from NIHR (National Institute of Health and Care Research).
- Double-blind, randomized, placebo-controlled, cross-over Phase 2 trial to evaluate the safety and efficacy of CORDStrom in 30 pediatric patients in the UK with intermediate and severe RDEB .
- Patients received two intravenous infusions of placebo or CORDStrom two weeks apart and then followed for nine months; each child then crossed over to the other arm and received two doses of the alternate arm two weeks apart with a further nine-month follow-up
- Topline results showed CORDStrom was easily administered, well tolerated and there were beneficial effects with respect to Itch Man Scale, iscorEB clinician score and skin score and QOL
- Safety profile - no CORDStrom-related serious adverse events were reported

FDA = Food and Drug Administration; GOSH = Great Ormond Street Hospital; NIHR = National Institute for Health and Care Research; ODD = Orphan Drug Designation; PRV = Priority Review Voucher; RPDD = Rare Pediatric Disease Designation

Mission EB Trial Design:
Double-Blind Randomized Crossover Trial in Children with RDEB



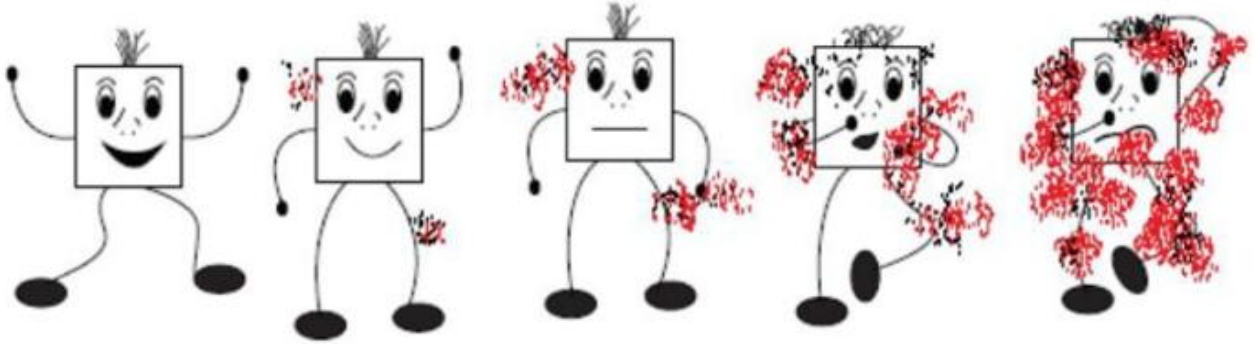
Itch assessment at every visit – Itch Man Scale

30 pediatric patients (age≤16 years) with RDEB confirmed by C7 testing were treated in a blinded, randomized placebo controlled cross-over design clinical trial at two university centers in the UK under MHRA authorization. All patients received all four doses of therapy (two each of CORDStrom or placebo) and completed the trial. Safety and efficacy data was collected. No drug related SAEs were reported. Disease related SAE and AE were equally balanced between treatment groups. Patient and caregiver interviews were performed in a subset of trial participants.

Itch: Clinically Meaningful Endpoint

- 100% of kids have itch as an important clinical problem
- FDA guidance highlights itch as a clinically important end-point* for RDEB
  - Itch Man Scale is a validated scales used in pediatric patients
  - Itch is as an endpoint used to approve drugs (eg: atopic dermatitis)
- Itch has negative impact on QOL
- Itch-scratch cycle may worsen wounds and complicate wound management

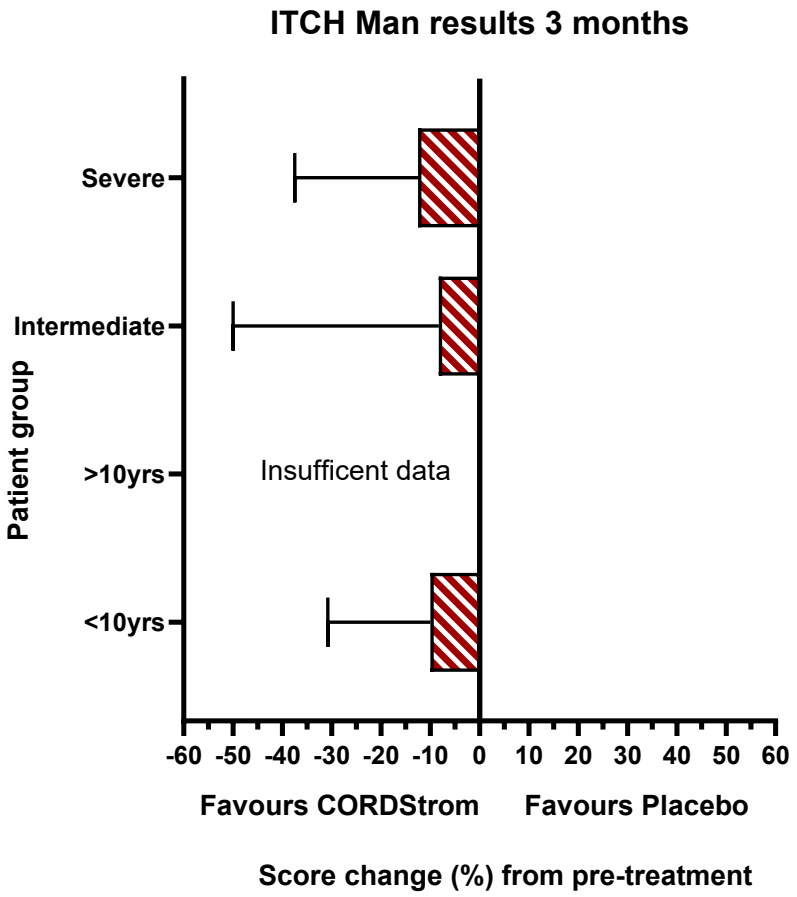
Itch Man Scale

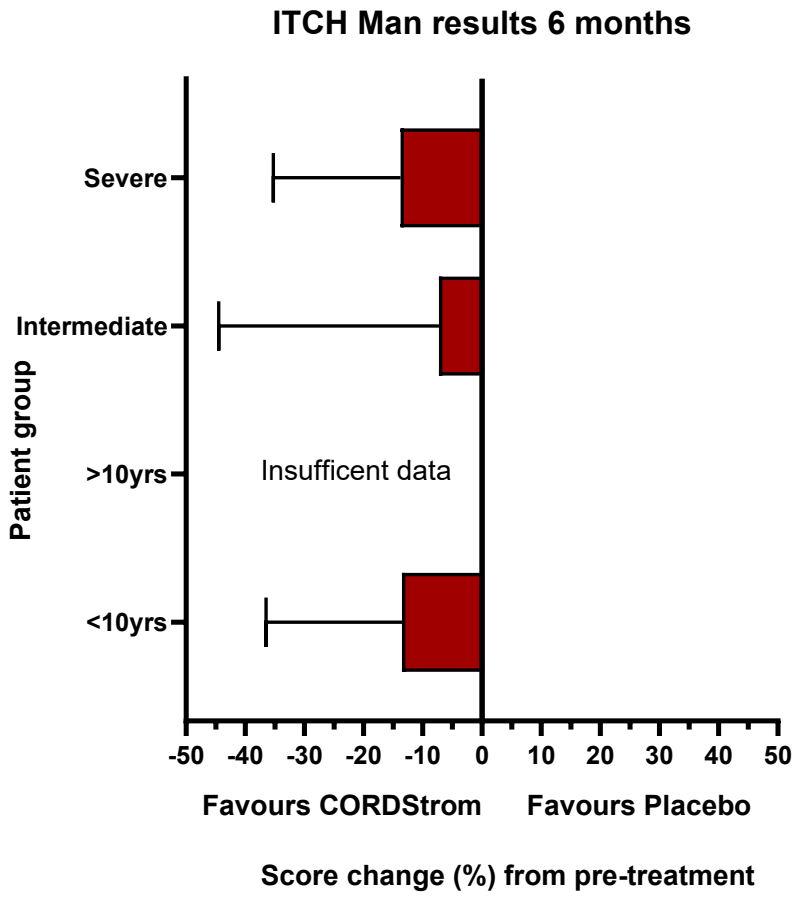


0	1	2	3	4
Comfortable, no itch	Itches a little; does not interfere with activity	Itches more; sometimes interferes with activity	Itches a lot; difficult to be still, concentrate	Itches most terribly; impossible to sit still, concentrate

* https://www.fda.gov/regulatory-information/search-fda-guidance-documents/epidermolysis-bullosa-developing-drugs-treatment-cutaneous-manifestations-guidance-industry

Itch: Clinically Meaningful Endpoint





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

CORDStrom for RDEB: Clinical and Qualitative Summary

Clinical Benefits

- Improvement in itch in all patient groups – the most common and complained of symptom in RDEB
- In some patient groups
  - Less pain
  - Better iSCOREB wound score
- Durable benefit of CORDStrom therapy for 6 months

Qualitative Benefits

- 10 of 13 respondents confirm benefit of therapy on clinical problems of itch, wound care and quality of life
- All patient/caregivers want to remain on therapy
- Favoravble safety profile and a formulation which fits conventional drug delivery makes treatment easy

NEXT STEPS: Compile and file BLA in US & MAA in UK/EU in 1H 2026

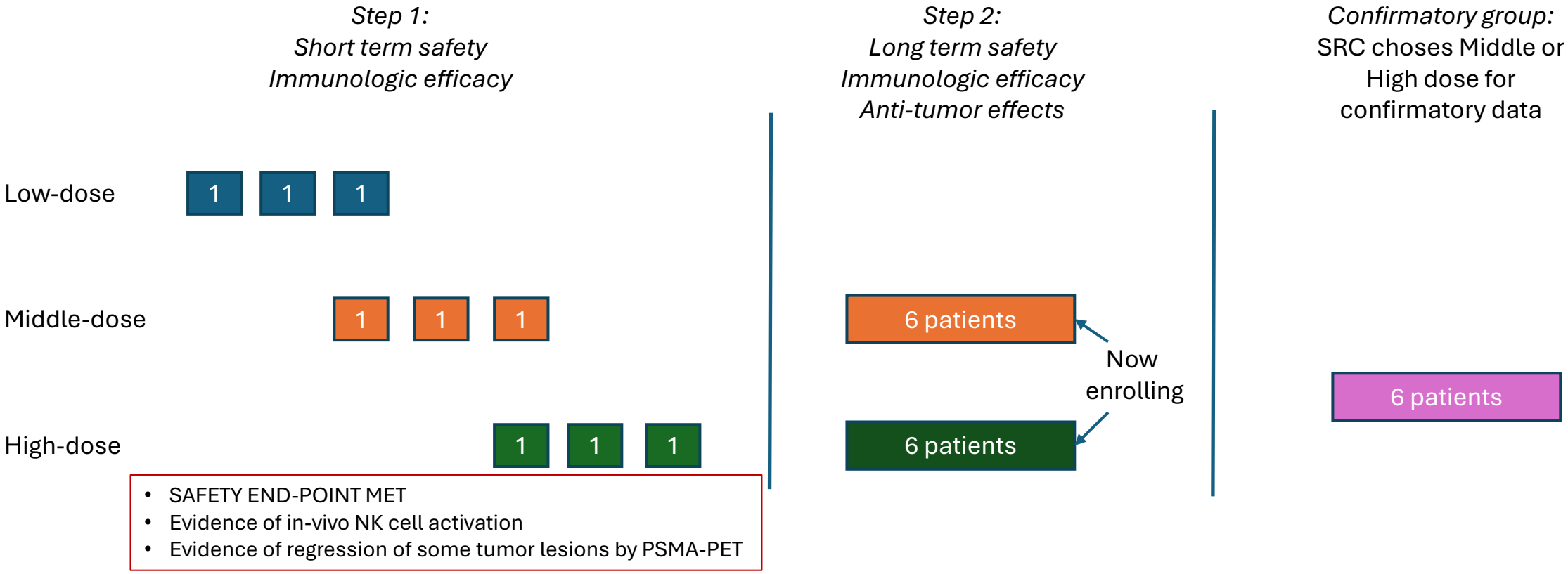
Goals of future open label trial post BLA: i) correlate decrease in itch with improved wound healing; ii) demonstrate systemic benefits on extra-cutaneous manifestations of disease (e.g.: dysphagia, corneal blisters and scaring)

INmuneBio

INKmune™ for Oncology

Off-the-Shelf NK Therapic Candidate Converts Patient's Resting NK cells into Cancer Killing Memory like NK cells

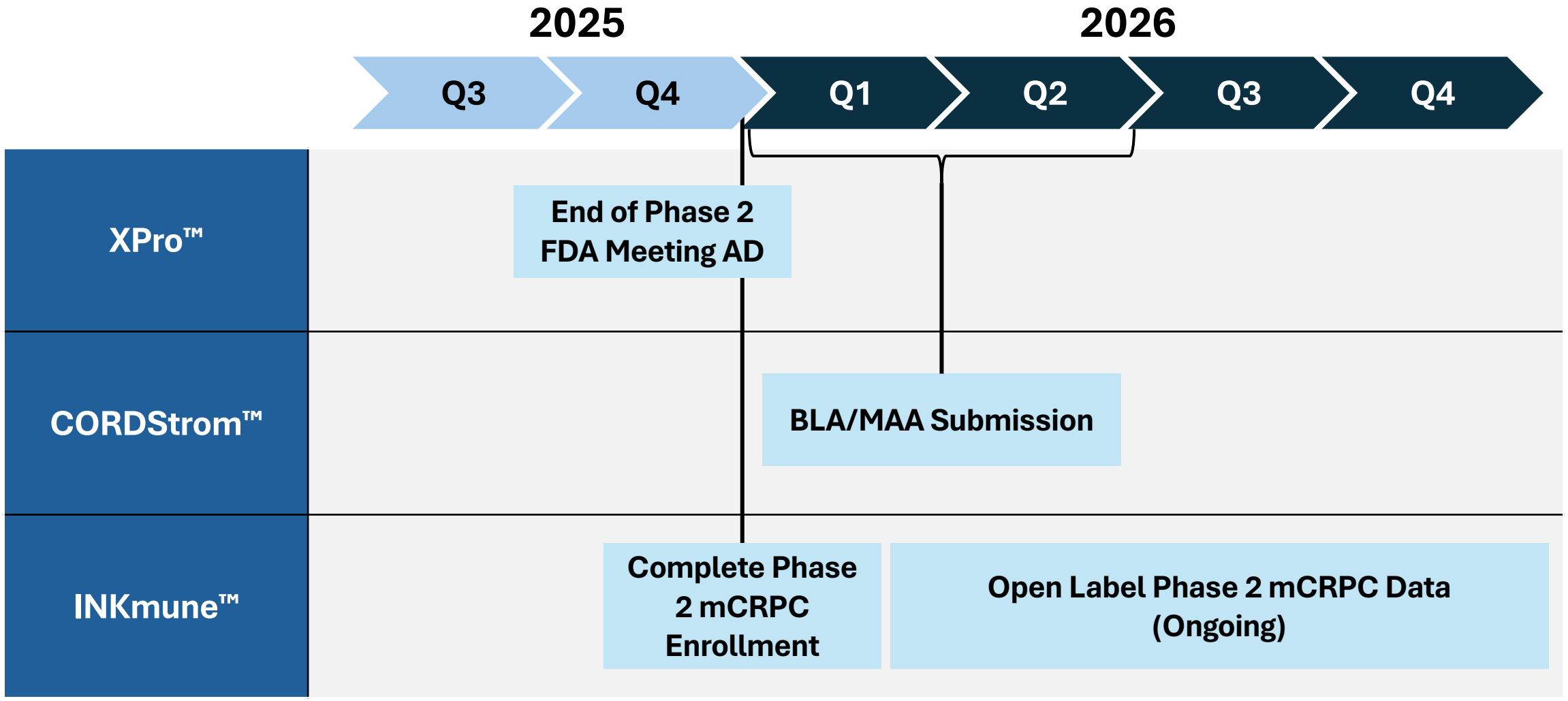
INKmune® mCRPC Phase I/II Trial Design

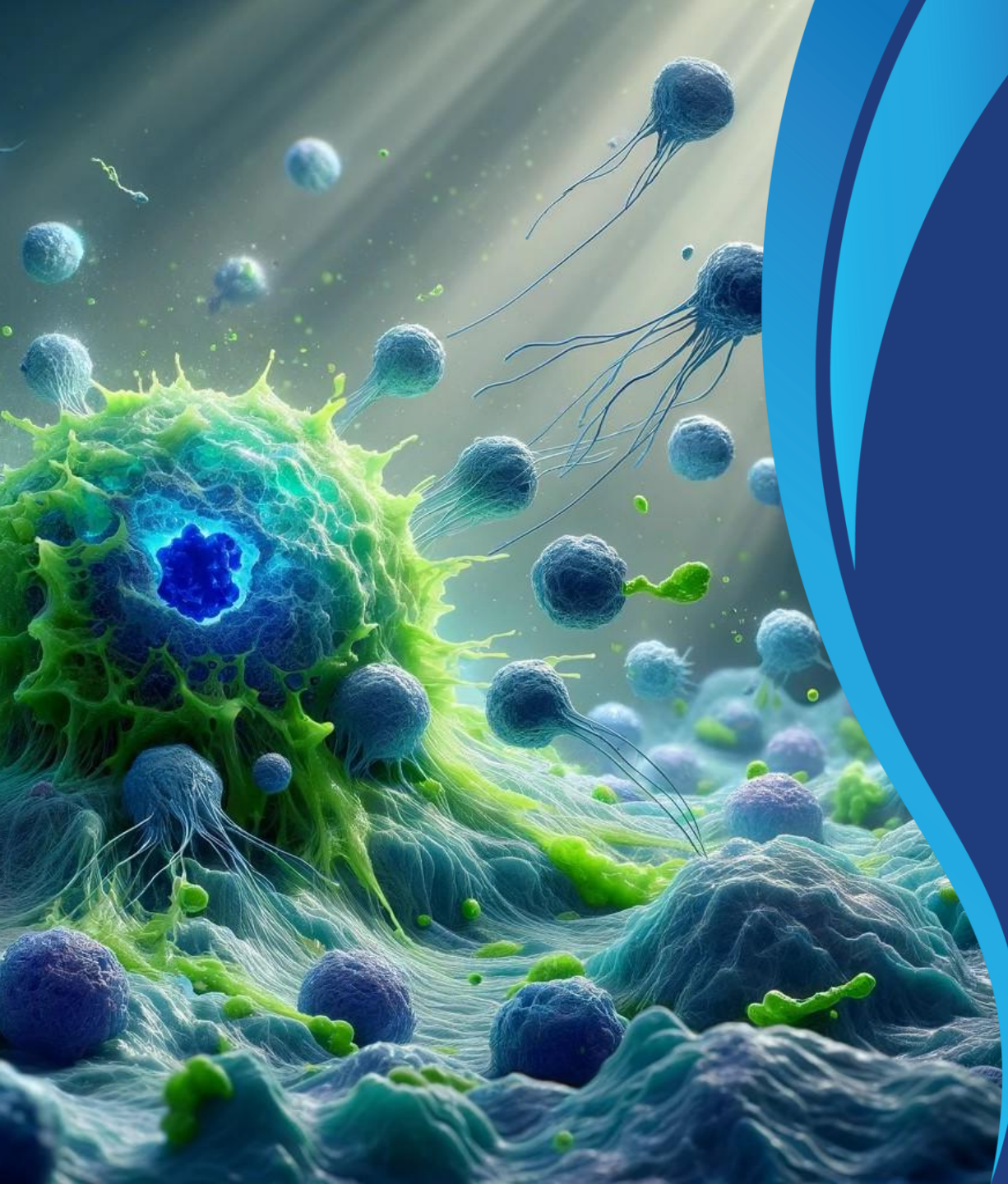


Trial will determine:

- Effective dose: safe with evidence of tumor effects
- Short and long-term safety – no drug related serious adverse effects
- Immunologic efficacy – converts patient's NK cells to mlNK cells that kill tumor cells (ex vivo assay) with long-term persistence of mlNK cells in patient's circulation
- Anti-tumor effects – evidence of control of tumor burden by PSMA-PET and/or ctDNA

Anticipated Milestones in 2025 and 2026





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