INmuneBio
INNATE IMMUNITY

Corporate Presentation

August 2025

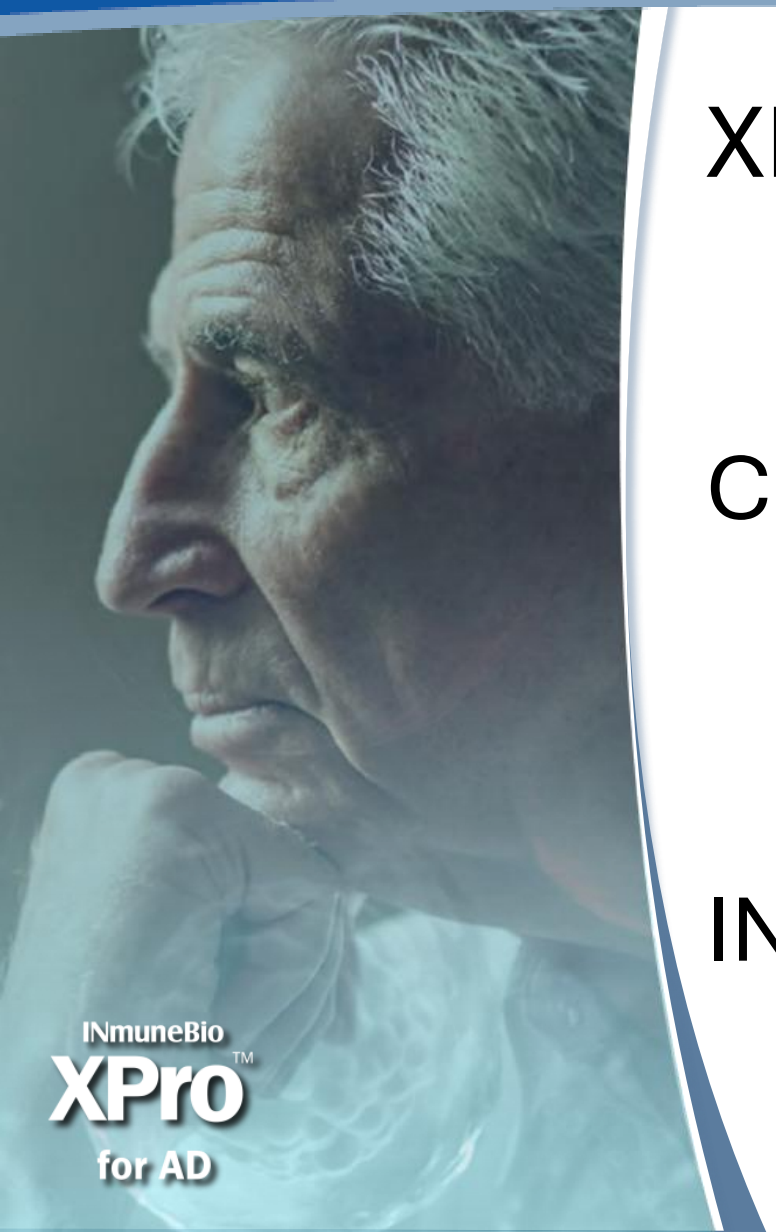
NASDAQ: INMB

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

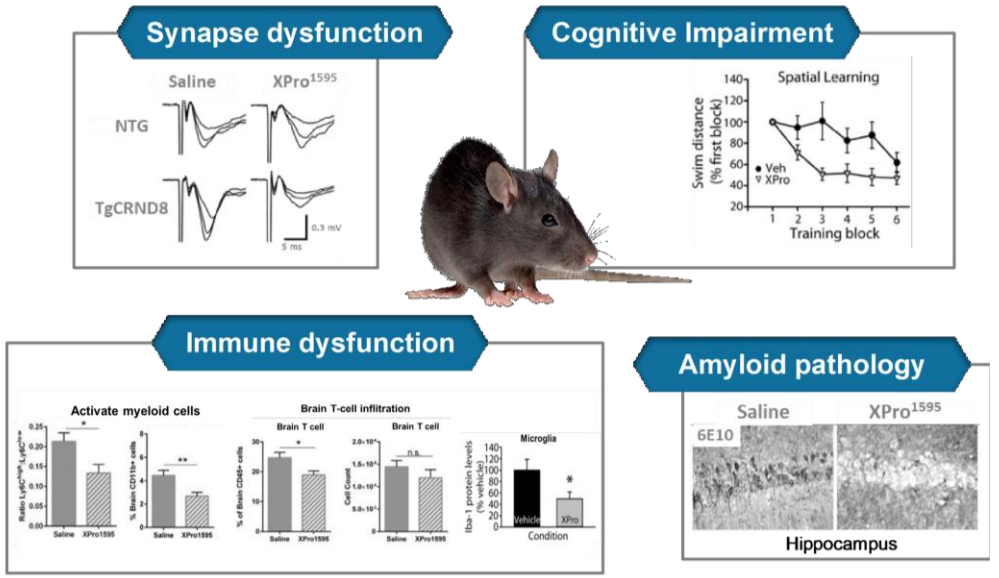
TNF increases amyloid[7,8] and Tau[9-12] TNF causes cell loss and cognitive impairment[13]

TNF inhibitors reduce risk of AD

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

72%

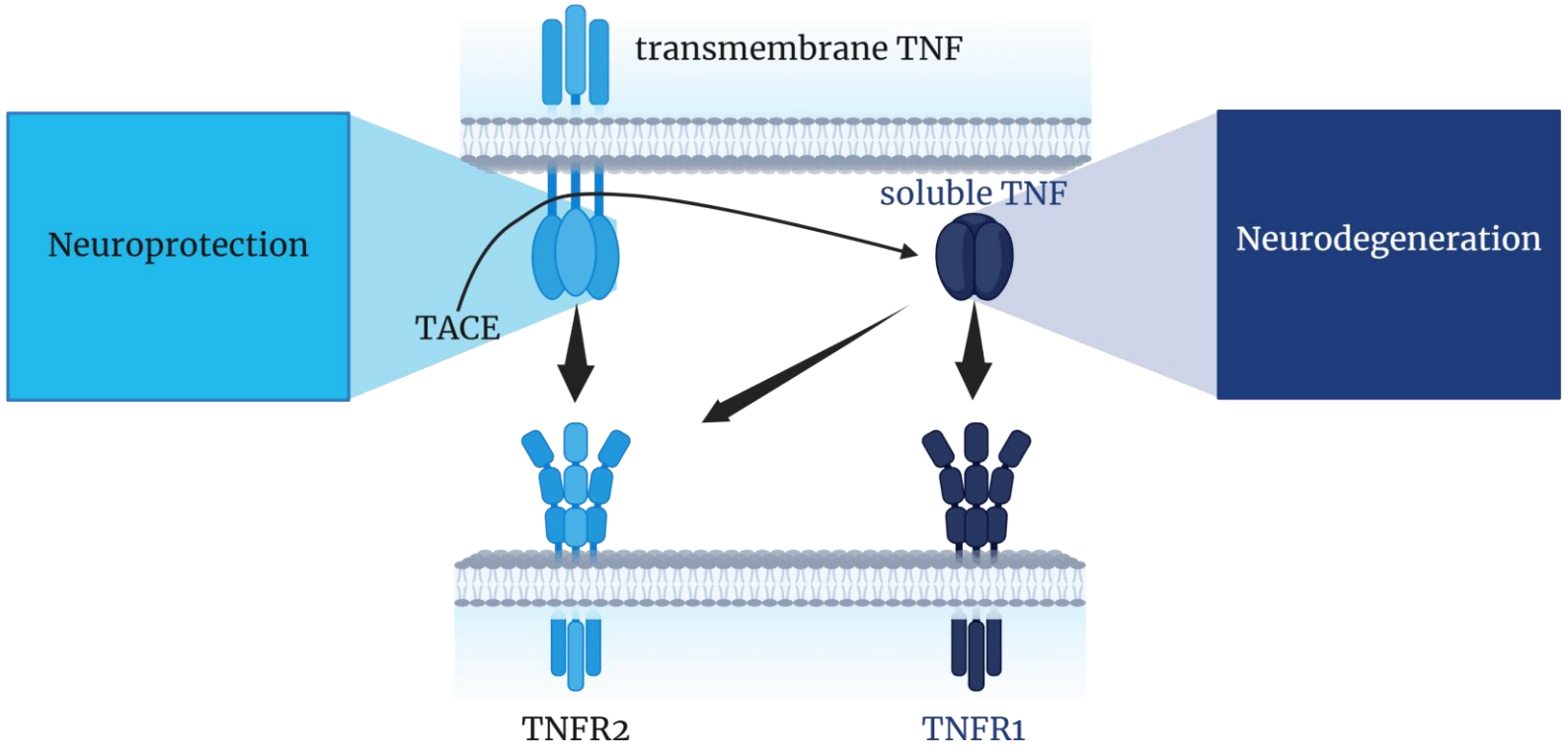
XPro™ efficacy in AD models



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

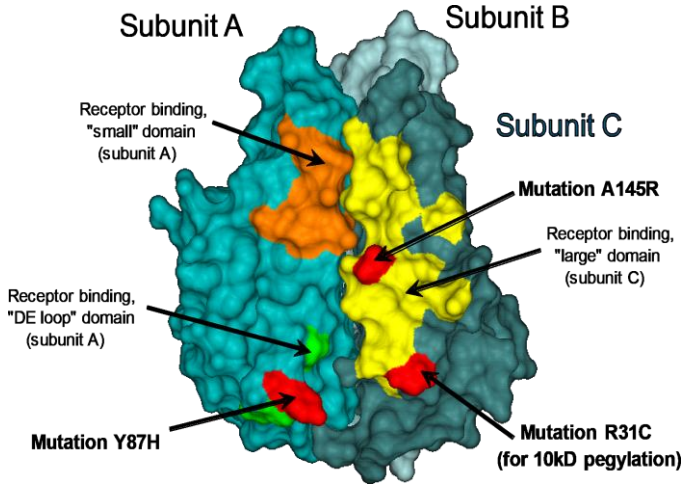
TNF Biology – Two Ligands with Opposite Effects



Created with BioRender.com.

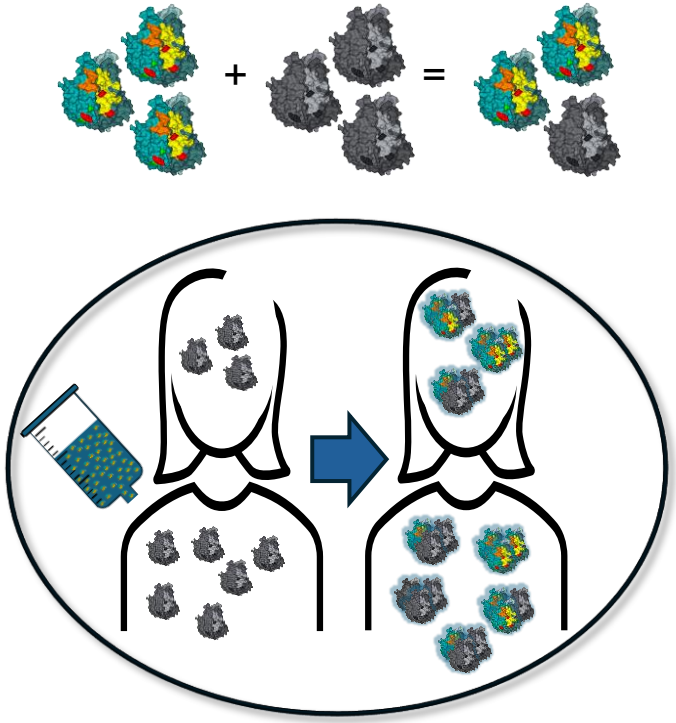
XPro™: A TNF Inhibitor Designed to Treat Neurologic Disease

XPro™

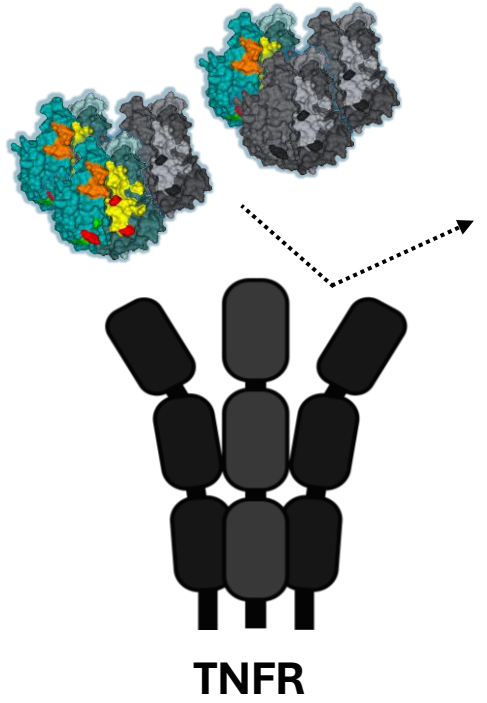


A dominant-negative* protein identical to the human soluble TNF monomer but has mutations in the receptor binding domain that inhibit solTNF from binding to the TNFR

forms inactive heterotrimers



that cannot bind to TNFR

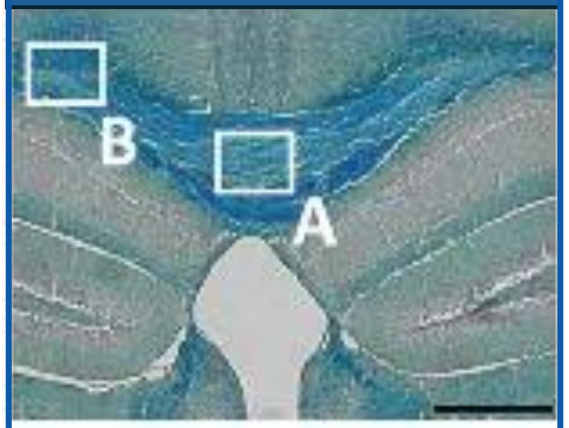


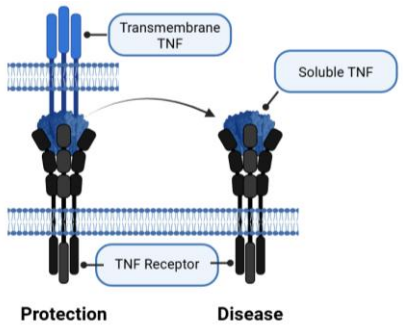
*Dominant-Negative in genetics: A mutation producing a rogue protein that interferes with the function of the native protein.

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

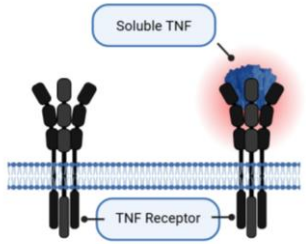
Normal





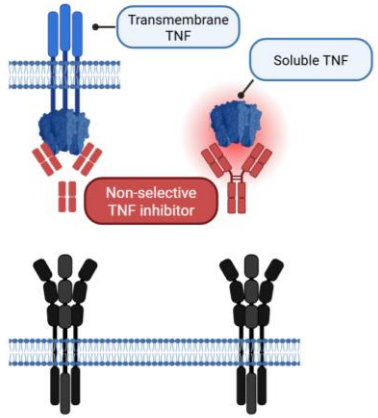
Disease

(Model of Multiple Sclerosis)



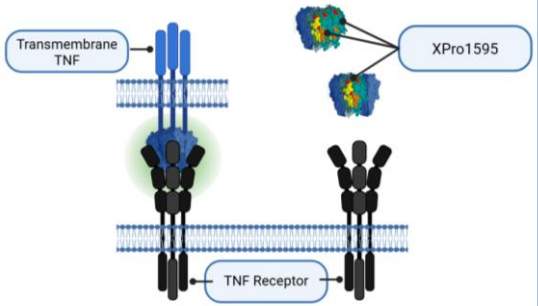
Etanercept

Non-selective TNF inhibition



XPro™

Soluble TNF inhibition



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

Rationale and Background for XPro1595 in Alzheimer's Disease

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]

XPro1595 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 neuroprotection, homeostatasis and repair

XPro1595 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 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.

Six month, Randomized, Placebo-Controlled, Blinded Study of XPro1595 in Early Alzheimer's with Biomarkers of Inflammation

MINDFuL
A study of inflammation in Alzheimer's disease

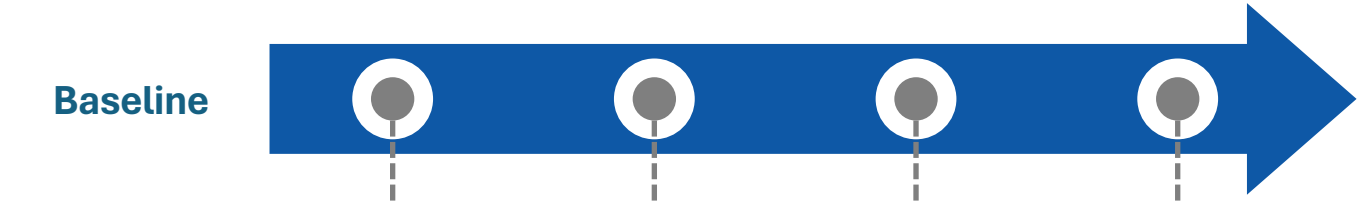
n= 208
2:1
XPro : Placebo

XPro1595
1 mg/kg weekly subQ injection

Placebo

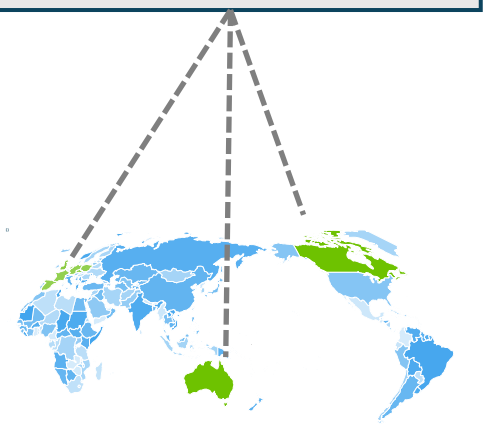
Primary Endpoint
- ❑ EMACC

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



Baseline	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™

Phase 2 Study Results

Baseline Demographics / Disease Characteristics (SAF Population)[a]

Demographic	Placebo (n=67)	XPro1595 (n=139)	Total (n=206)
Age, years	72.1 (6.75)	72.7 (6.40)	72.5 (6.51)
Sex (female), n (%)	35 (52.2%)	70 (50.4%)	105 (51.0%)
Race (white), n (%)	65 (97.0%)	131 (94.2%)	198 (95.1%)
Ethnicity (not Hispanic or Latino), n (%)	64 (95.5%)	134 (96.4%)	192 (96.1%)
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 (screening), mean (SD)	26.0 (2.19)	25.7 (2.40)	25.8 (2.33)
CDR-SB (baseline, mean (SD)	2.67 (1.35)	3.23 (1.58)	2.95 (1.46)
Modified Intention to Treat Population (mITT)			
APOE ε4 genotype, n	66	134	200
Noncarrier, n (%)	19 (28.8%)	26 (19.4%)	45 (22.5%)
Carrier, n (%)	37 (56.1%)	80 (59.7%)	117 (58.5%)
Homozygote, n (%)	4 (6.1%)	11 (8.2%)	15 (7.5%)
Amyloid-beta (Aβ) positive, n (%)	47 (71.2%)	103 (76.9%)	150 (75.0%)
Enrichment biomarkers of inflammation, n (%)			
1 biomarker	28 (42.4%)	47 (35.1%)	75 (37.5%)
2 biomarkers	27 (40.9%)	58 (43.3%)	85 (42.5%)
3 biomarkers	9 (13.6%)	24 (19.9%)	33 (16.5%)
4 biomarkers	0 (0%)	5 (3.7%)	5 (2.5%)

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

Source: INmune Bio, Inc., Protocol: XPro1595-AD-02, Unblinded Tables

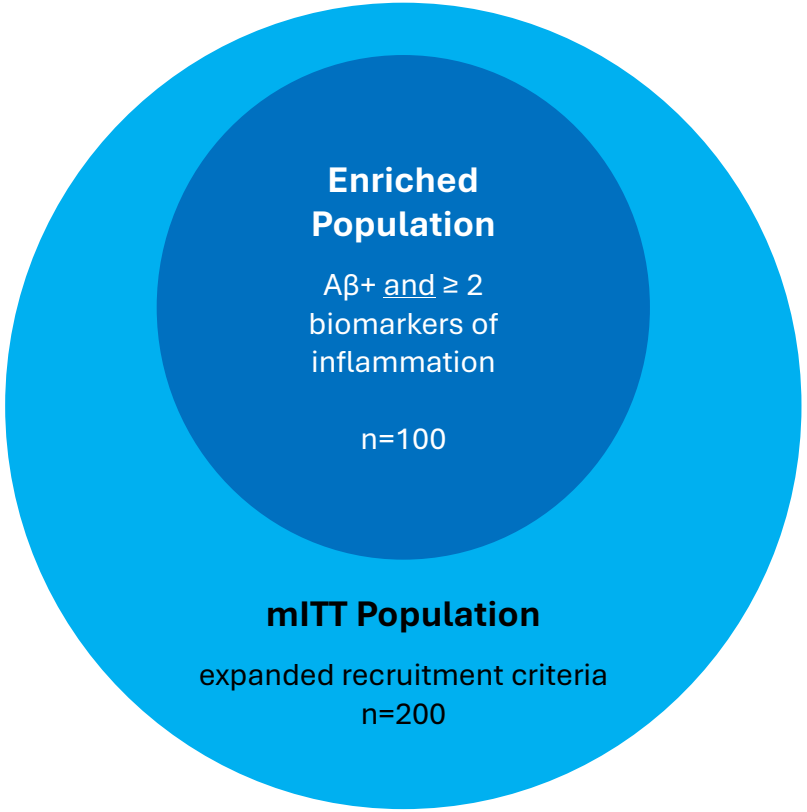
Population Changes Due to Operational Limitations

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

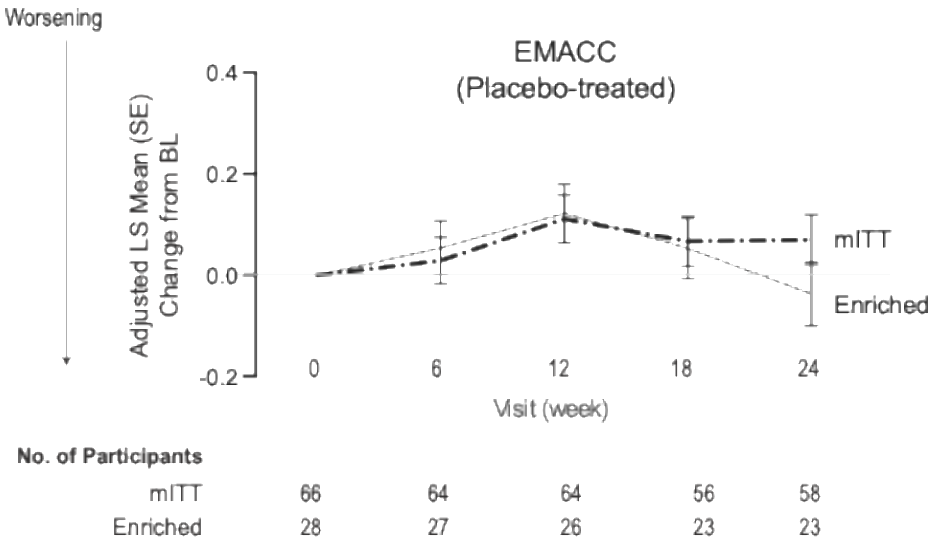
Expanded Inclusion Criteria: Revised Protocol

- 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**



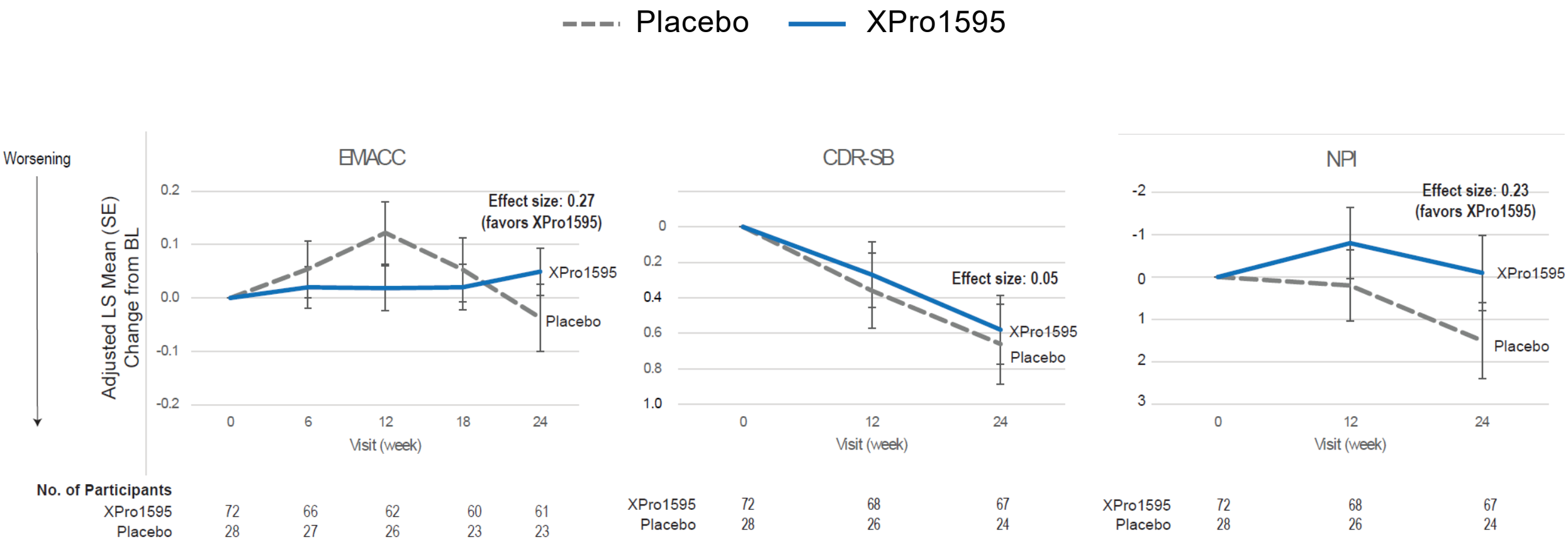
To assess a possible treatment effect of XPro1595 compared to placebo, a decrease in cognition within the placebo-treated group over the study period is necessary.

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.



Enriched Population (n=100): Primary and Key Secondary Endpoints

Change From Baseline



No. of Participants					
XPro1595	72	66	62	60	61
Placebo	28	27	26	23	23

XPro1595	72	68	67
Placebo	28	26	24

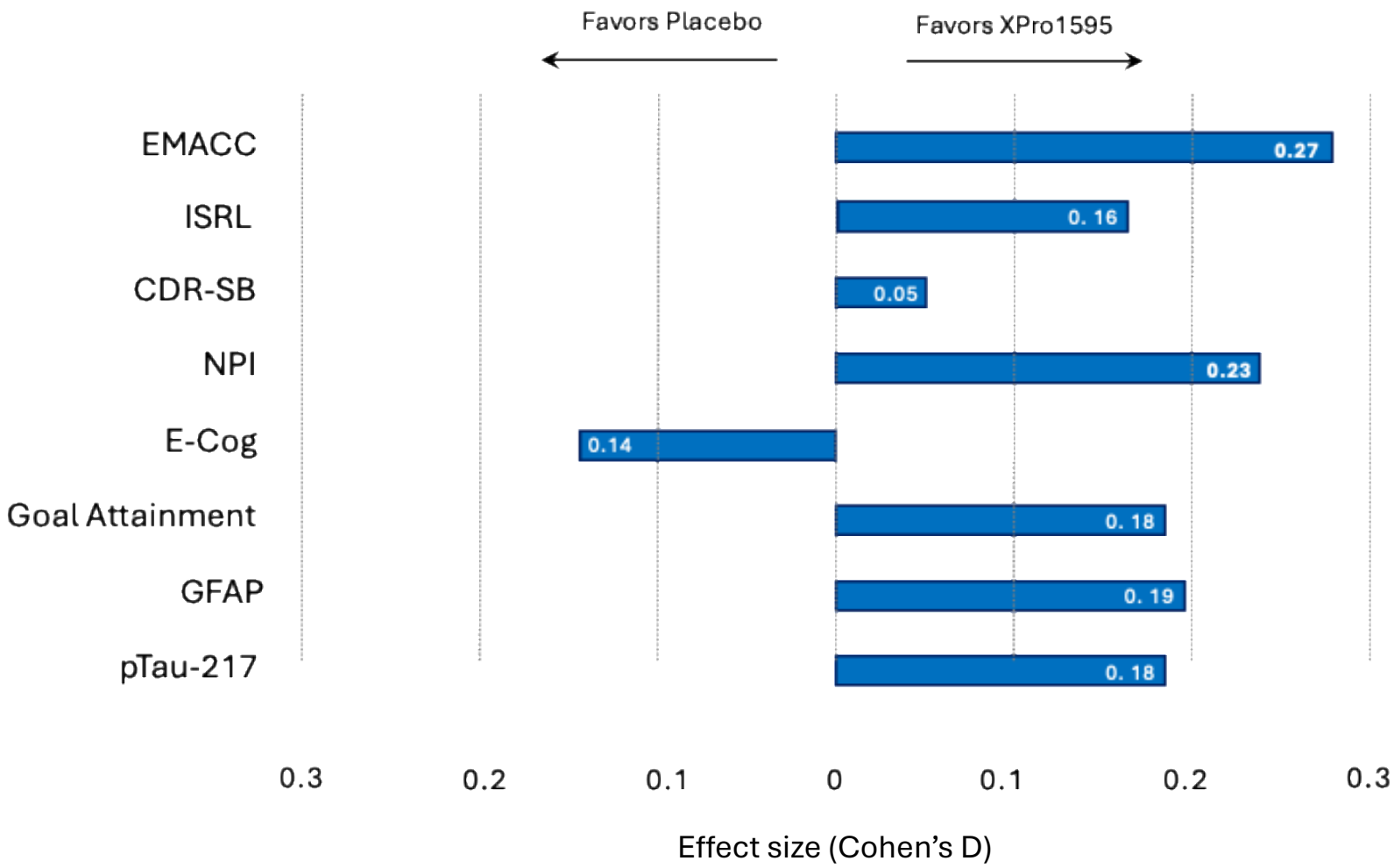
XPro1595	72	68	67
Placebo	28	26	24

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 XPro1595

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

No ARIA

Despite inclusion of patients at risk for ARIA

ARIA Treatment emergent, SAF	Placebo (n=67)	XPro1595 (n=139)	Total (n=206)
ARIA-E	0	0	0
ARIA-H	0	0	0

ARIA risk factors at baseline, SAF	Placebo (n=67)	XPro1595 (n=139)	Total (n=206)
Cerebral microbleeds (<15)	24 (35.8%)	46 (33.1%)	70 (34.0%)
Antithrombotics (e.g., apixaban, clopidogrel, dabigatran, edoxaban, enoxaparin, nadroparin, ticagrelor, warfarin)	19 (28.4%)	37 (26.6%)	56 (27.2%)
APOE4 heterozygotes	37 (55%)	80 (57.5%)	117 (56.8%)
APOE4 homozygotes	8 (11.9%)	18 (12.9%)	26 (12.6%)

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

XPro1595 has a Favorable Safety Profile

Summary of TEAEs

Safety: Treatment Emergent Adverse Events (TEAEs): Safety Analyses Set			
Event, n (%)	**Placebo (n=67)**	**XPro1595 (n=139)**	**Total (n=206)**
Any TEAE	59 (88.1%)	131 (94.2%)	190 (92.2%)
Any TEAE by Maximum Severity			
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 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 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

TEAEs present in ≥10%

System Organ Class & Preferred Term	Placebo (n=67)	XPro1595 (n=139)	Total (n=206)
General disorders; administration site conditions			
Injection site reaction	2 (3.0%)	73 (52.5%)	75 (36.4%)
Injection site erythema	0	49 (35.3%)	49 (23.8%)
Fatigue	9 (13.4%)	17 (12.2%)	26 (12.6%)
Injection site hypersensitivity	0	14 (10.1%)	14 (6.8%)
Injection site pruritus	0	12 (8.6%)	12 (5.8%)
Infections and infestations			
Upper respiratory tract infection	11 (16.4%)	9 (6.5%)	20 (9.7%)
Musculoskeletal and connective tissue disorders			
Arthralgia	4 (6.0%)	16 (11.5%)	20 (9.7%)
Nervous system disorders			
Headache	7 (10.4%)	14 (10.1%)	21 (10.2%)

Summary and Conclusions

mITT population

- ❑ The primary endpoint was not met in the mITT population.
- ❑ The mITT study population (n=200) did not decline in the 6-month period.
- ❑ Decline was observed in the originally intended population (the enriched population) having amyloid and 2 or more biomarkers of inflammation.

Enriched population

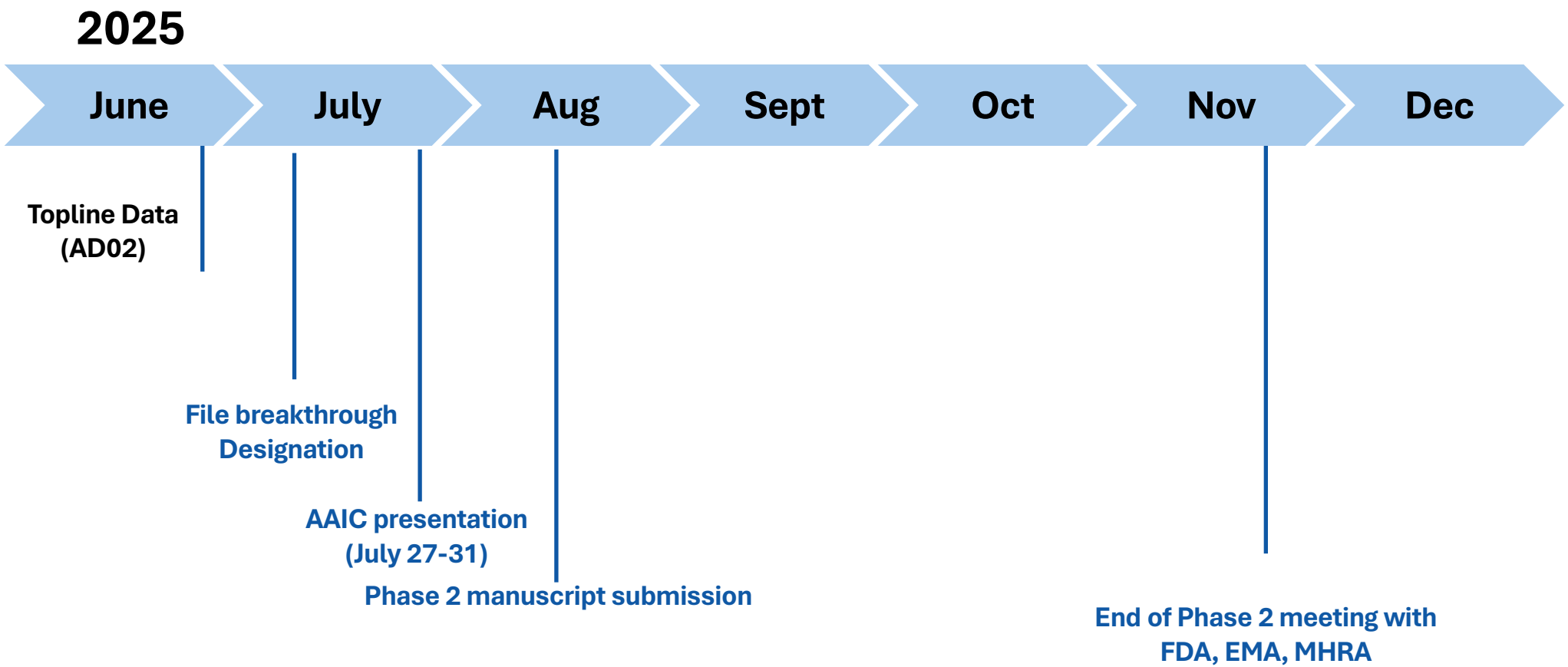
- ❑ XPro1595 showed a beneficial signal, consistent across multiple measures, in the intended study population (n=100).
  - Cognition (EMACC)
  - Memory (ISRL)
  - Neuropsychiatric (NPI)
  - Biomarkers (pTau217, GFAP)
- ❑ XPro1595 did not show benefit on traditional functional measures (CDR, E-Cog).
- ❑ The Goal Attainment Scale (GAS), individualized goals around cognition and function, shows a benefit in the XPro1595 treated group.

Safety

- ❑ XPro is safe.
- ❑ There were no cases of ARIA and no deaths.
- ❑ The most common TEAE was Injection Site Reaction (ISR), most were mild/moderate and resolved without sequelae.
- ❑ SAEs were rare and not clearly related to study drug.

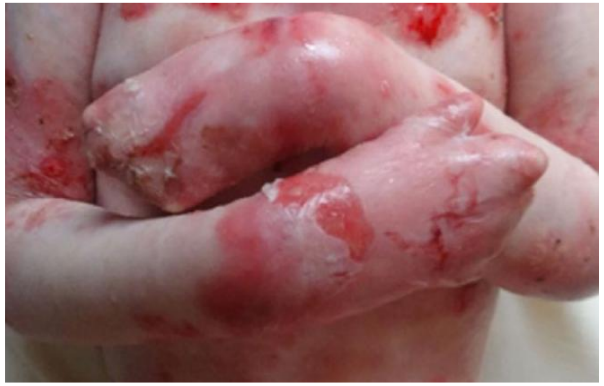
Next steps: validate the enriched population in a fully powered trial.

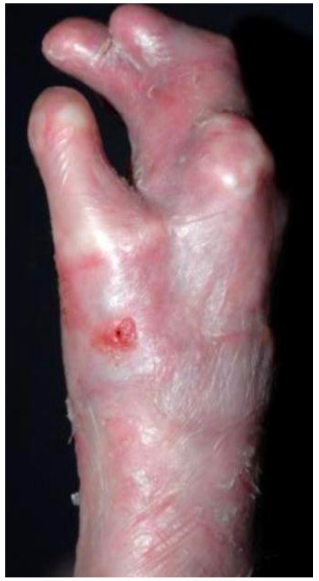
XPro™ Anticipated Next Steps and Timeline

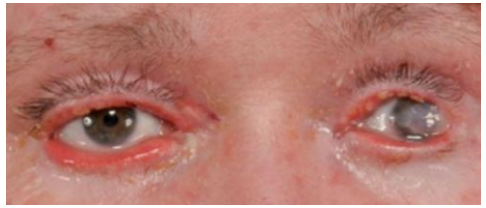


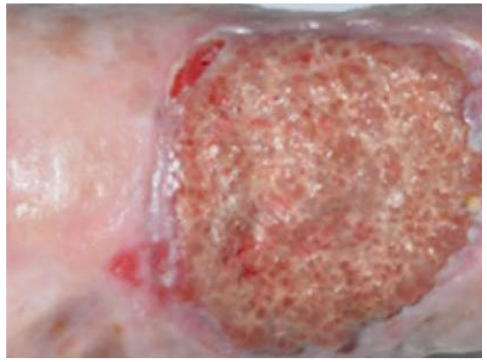

INmuneBio
CORDStrom™
for RDEB

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

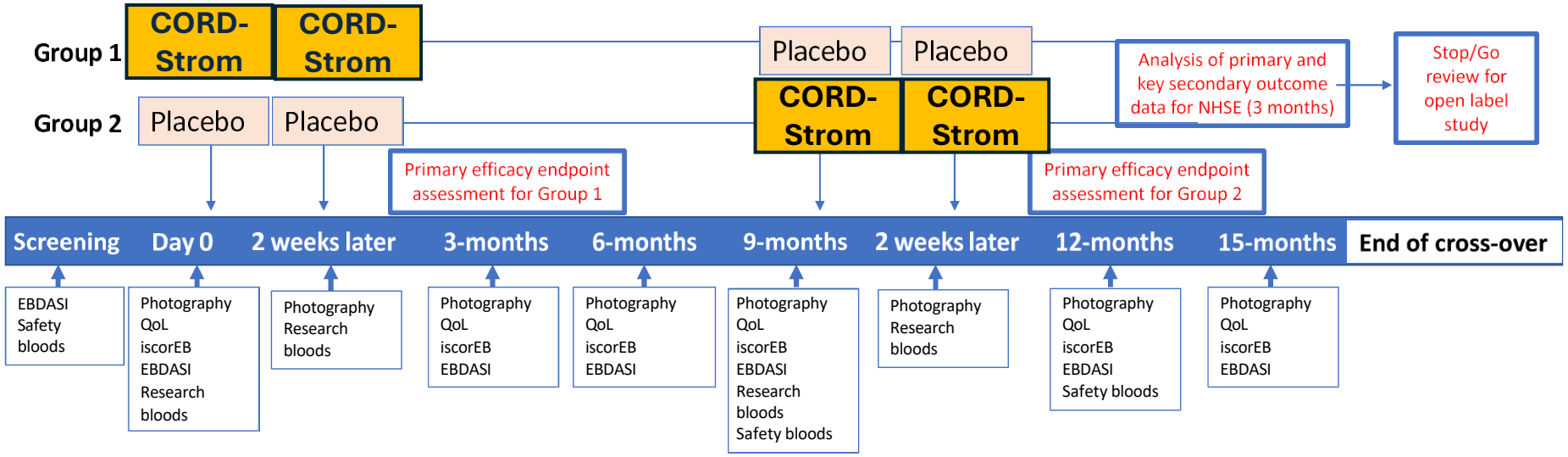
- CORDStrom is an innovative cellular medicine, comprising allogeneic, pooled human umbilical cord-derived mesenchymal stromal cells (hucMSCs) formulated for injection or infusion.
- CORDStrom addresses challenges related to the source, identity, heterogeneity, and manufacturing costs of MSCs, positioning 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



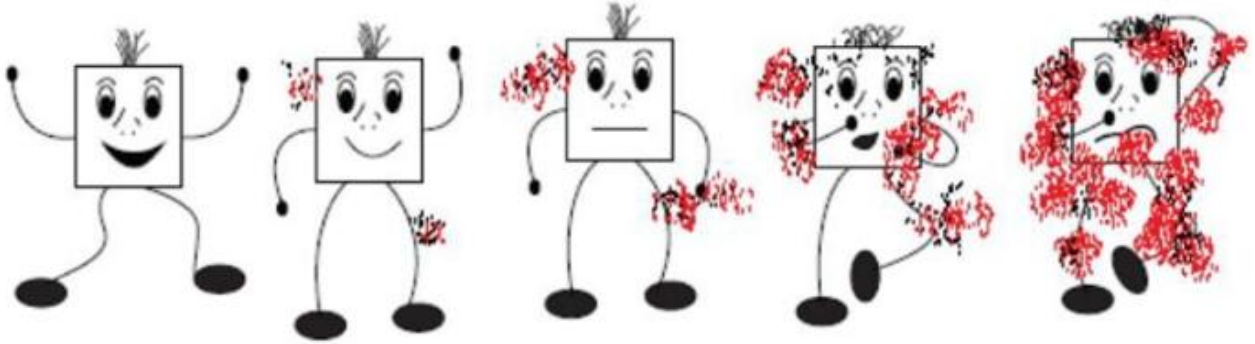
Itch assessment at every visit – Itch Man Scale

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

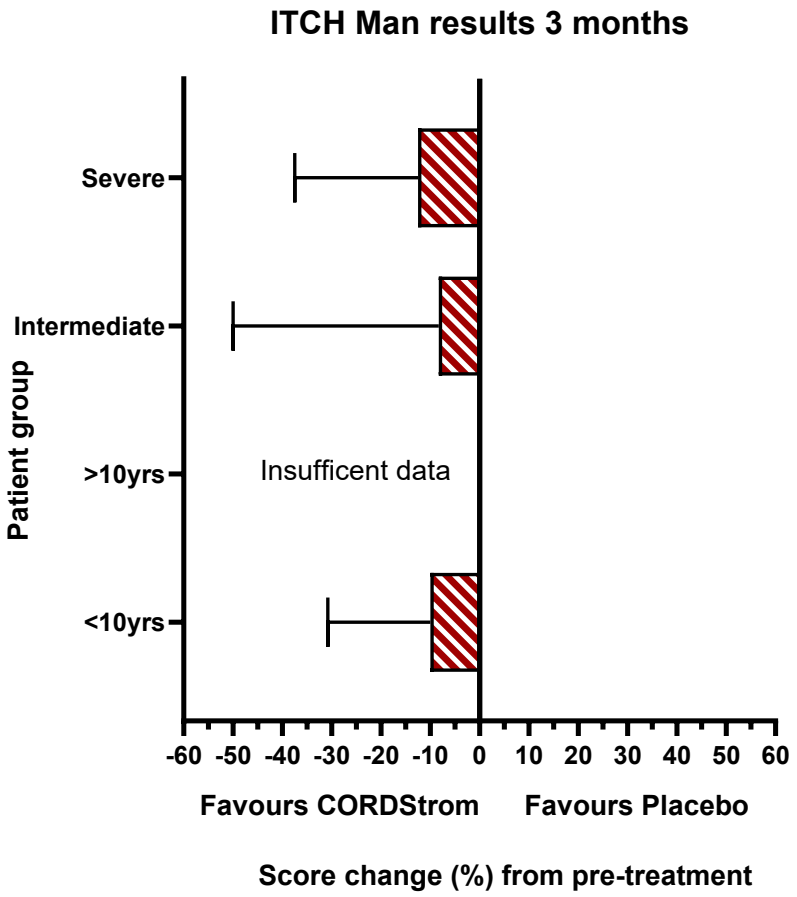
Itch Man Scale

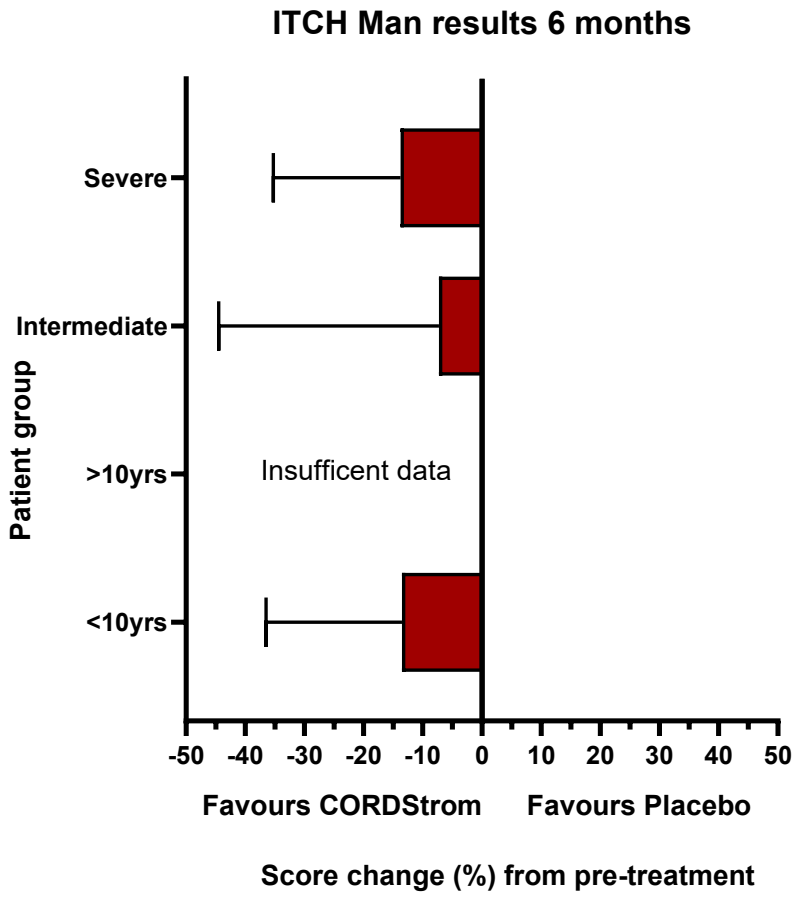


0	1	2	3	4
Comfortable, no itch	Itches a little; does not interfere with activity	Itches more; sometimes interferes with activity	Itches a lot; difficult to be still, concentrate	Itches most terribly; impossible to sit still, concentrate

* 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

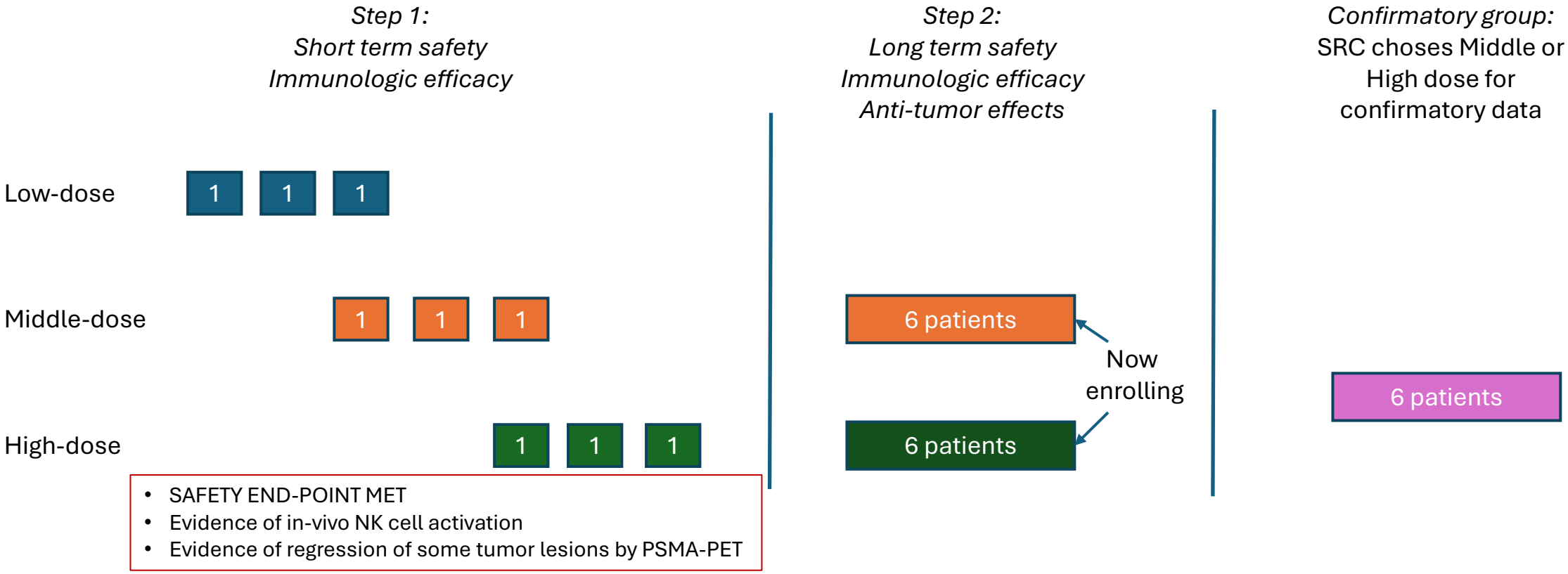
Goals of future open label trial post BLA: i) correlate decrease in itch with improved wound healing; ii) demonstrate systemic benefits on extra-cutaneous manifestations of disease (e.g.: dysphagia, corneal blisters and scaring)

INmuneBio

INKmune™ for Oncology

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

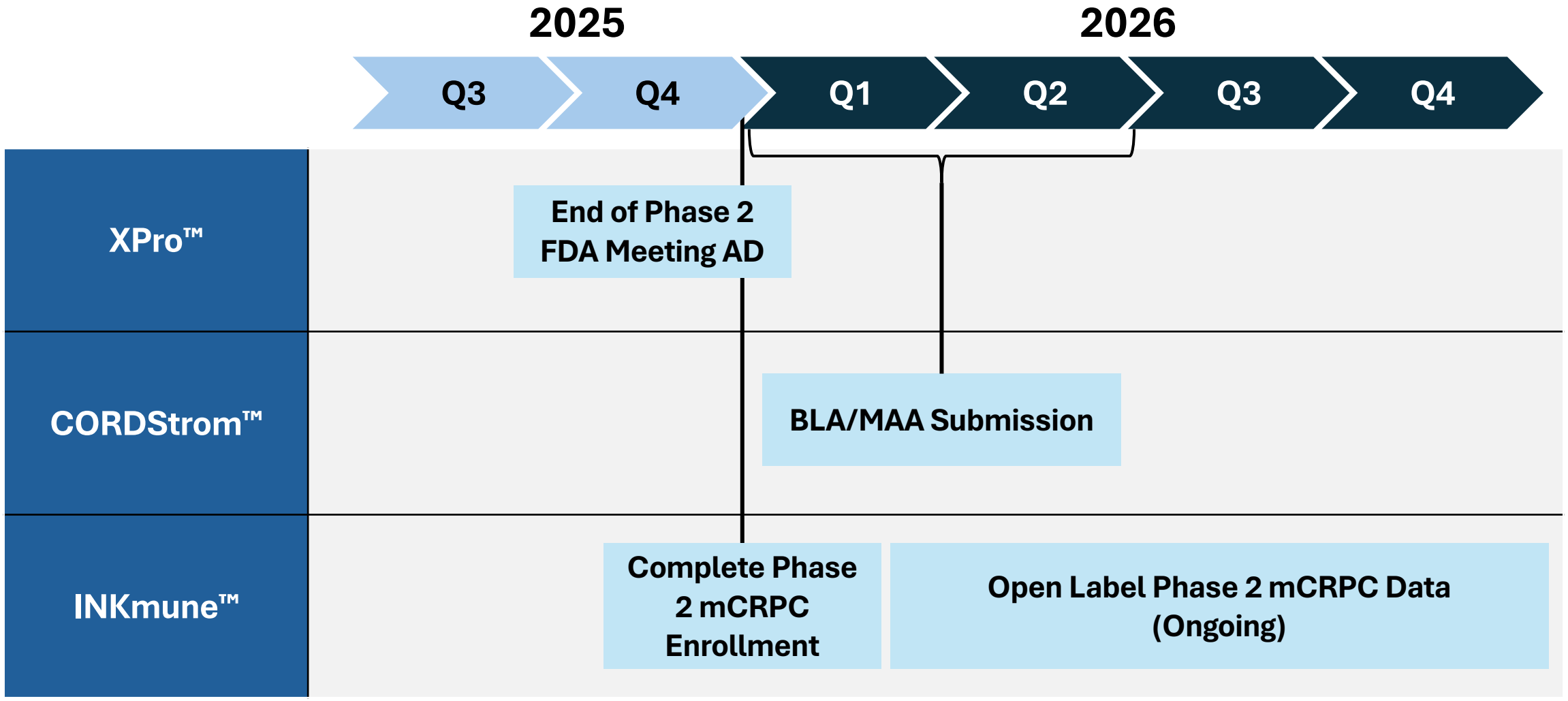
INKmune® mCRPC Phase I/II Trial Design

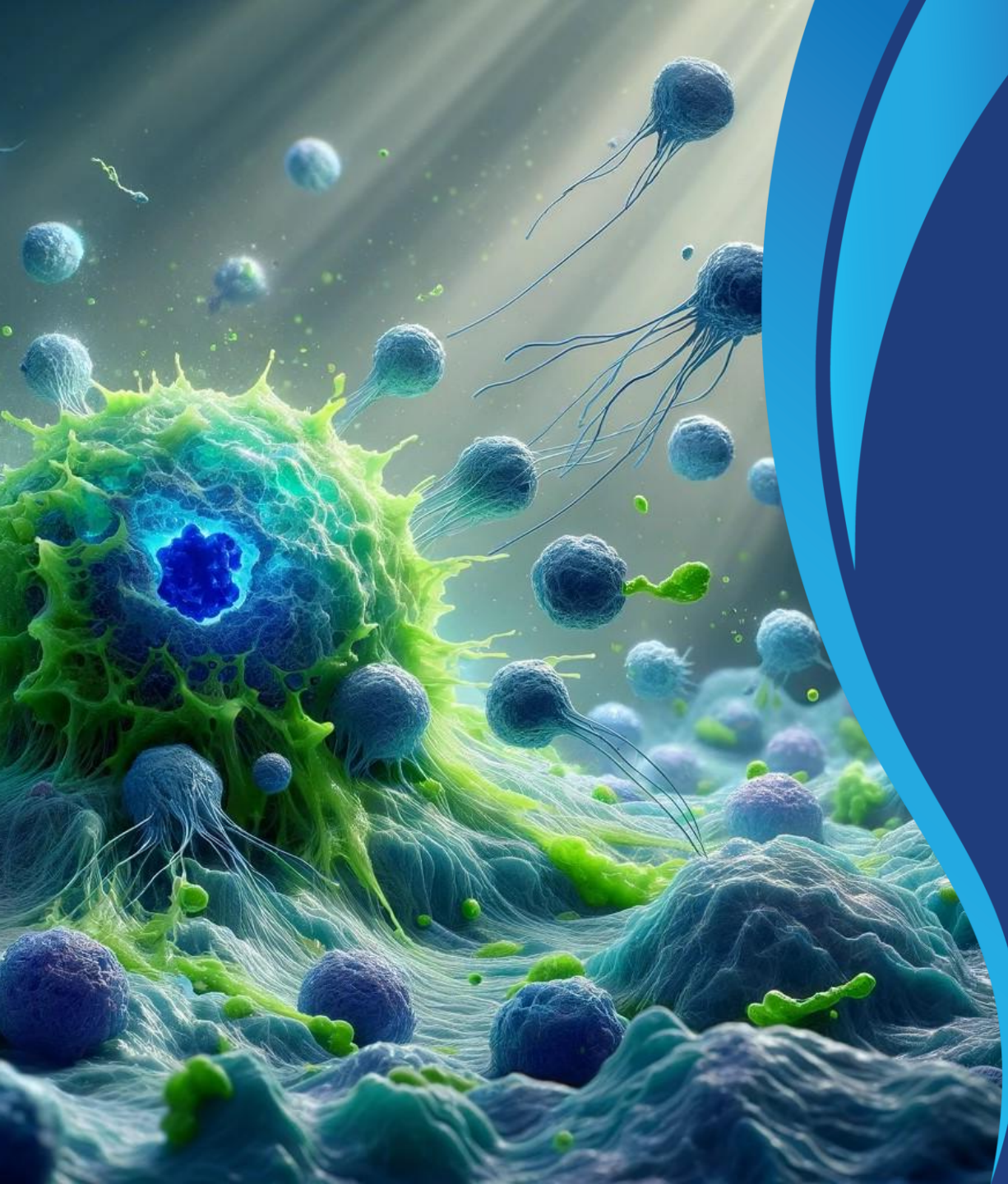


Trial will determine:

- 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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