INmuneBio
INNATE IMMUNITY

Inflammation and Immunology Repair

Two Platforms in the Clinic: XPro™ and INKmune™

INMB
Nasdaq

August 2024

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 relating to our industry, our operations and results of operations. There is no guarantee that any specific outcome will be achieved. Investment results are speculative and there is a risk of loss, potentially all loss of investments. 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.

Two Novel Platforms with Near Term Data

- Two platforms targeting CNS and Oncology
- Treating Alzheimer's as an Immunologic Disease without immunosuppression
- Creating memory-like NK cells for solid tumors
- Phase 2 Alzheimer's top-line expected in early 2025; Phase 1 metastatic prostate cancer read-out late 2024
- Clean balance sheet with strong insider participation and ownership

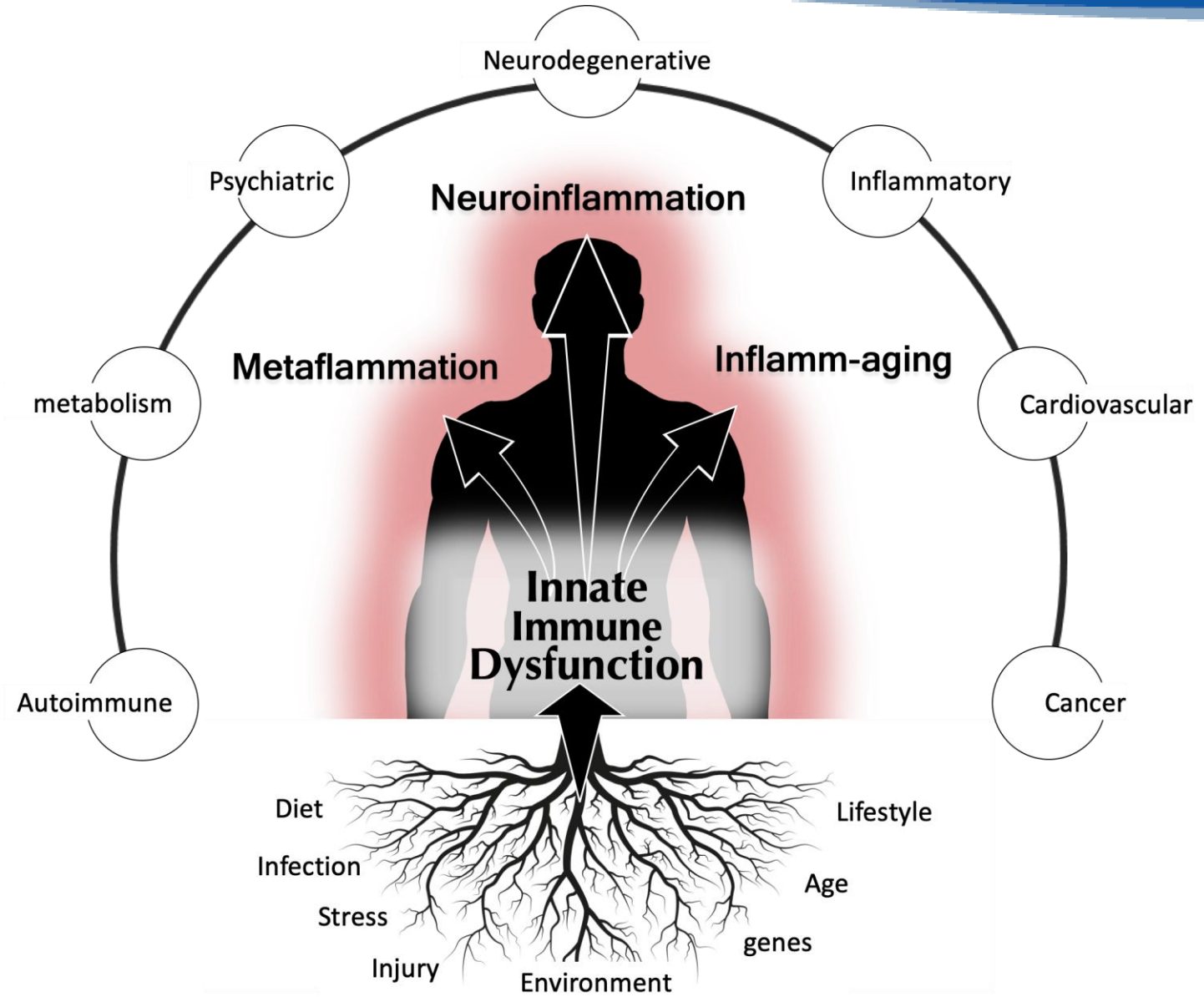
Development Timeline

DN-TNF PLATFORM	DESEASE FIELD	PRE-CLINICAL	PHASE 1	PHASE II (POC)	PIVOTAL	EST.NEXT MILESTONE
XPro™	Early Alzheimer's Disease	→				Full Enrollment Q3-2024 Topline Data ~ 6m Later
XPro™	Treatment Resistant Depression		→			P2 Start 2024

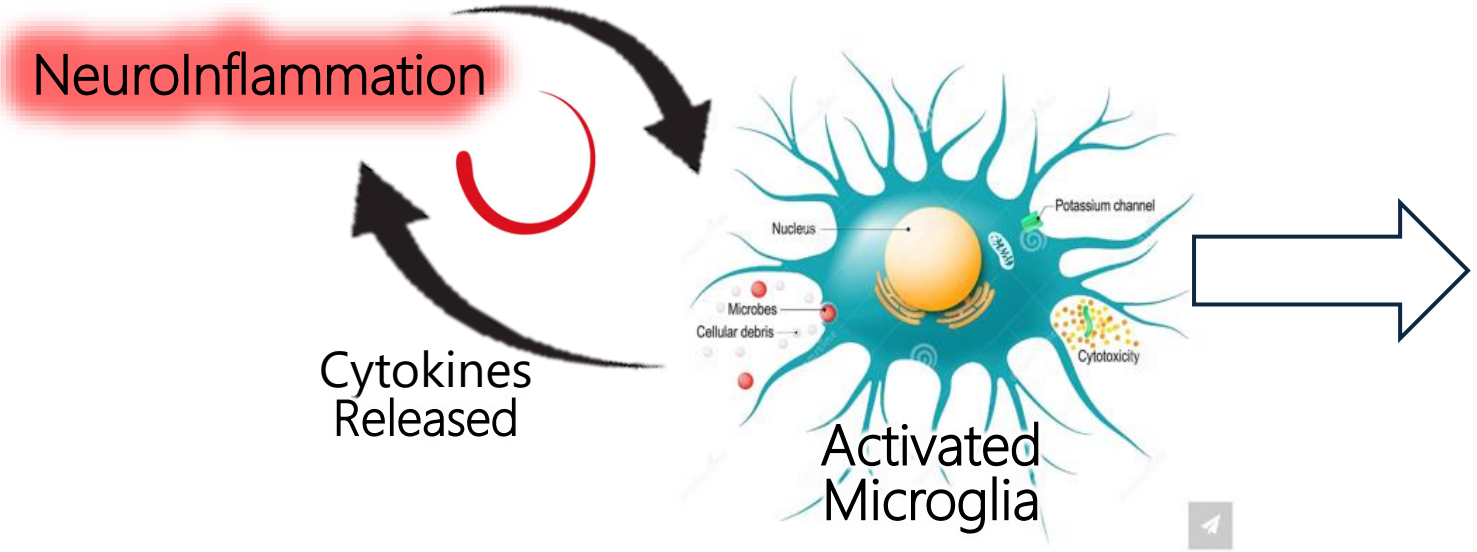
NK PRIMING PLATFORM						
INKmune™	Metastatic Castrate Resistant Prostate Cancer		→			Open Label Trial Data 2024
INKmune™	Other Solid Tumors			→		Open Label 2025


INmuneBio
XPro™
for AD
Treating Alzheimer's as an Immunologic Disease
...not a Neurologic Disease

Chronic Inflammation: the Match that Lights the Fire



The *"Doom Loop"* of Neuroinflammation and Cognitive Decline



Essential Pathologies of Cognitive Decline

- Synaptic Dysfunction
- Demyelination
- Nerve Cell Death

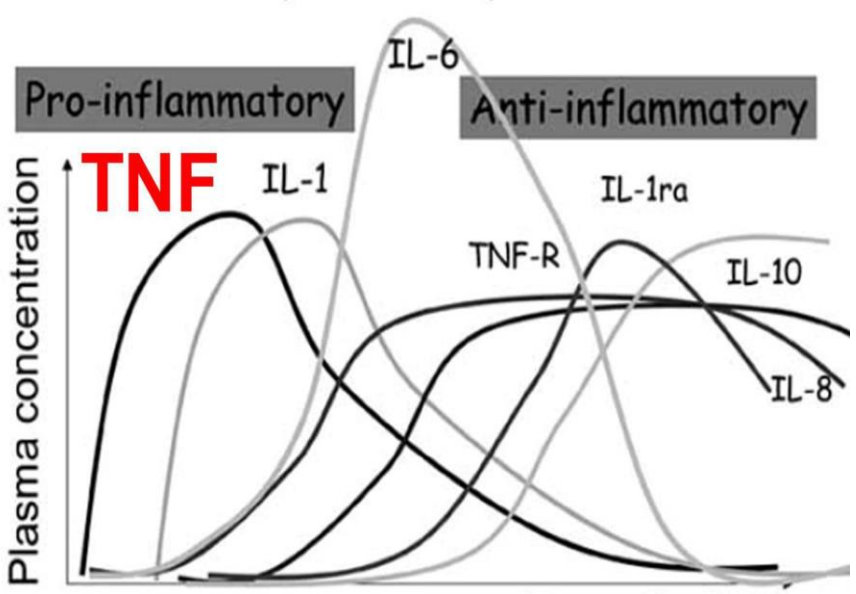
Targeting neuroinflammation with XPRO should stop cognitive decline to allow remodeling and repair

1. Chang R, et al. *J Cent Nerv Syst Dis*. 2017;9: 1-5
2. Shamim D, et al. *J Cent Nerv Syst Dis* .2017 ;9:1-10
3. Hulshof LA, et al. *Front Cell Neurosci*. 2022;16:1-17
4. Planas-Fontánez TM, et al. *Brain Res*. 2021;1764:147464
5. Marzan DE, et al. *Glia*. 202;69(6):1583–1604

Targeting the Root Cause of Neuroinflammation

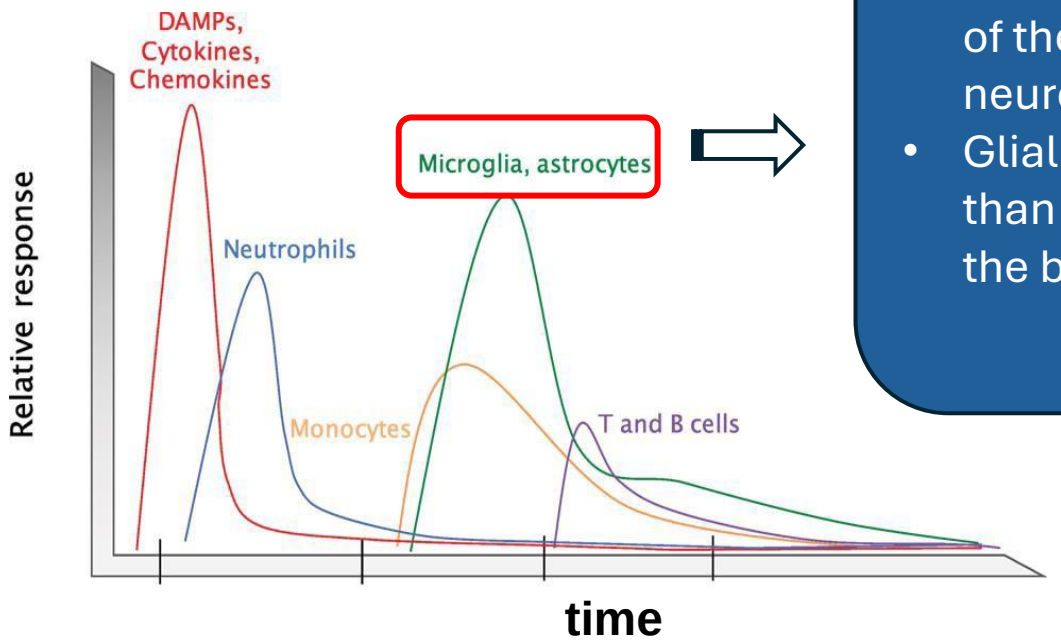
Why sTNF? sTNF is the "Master Cytokine" that drives neuroinflammation

Soluble TNF (sTNF) drives neuroinflammation



PMID: 18673219

Inflammatory cytokines drive immune dysfunction



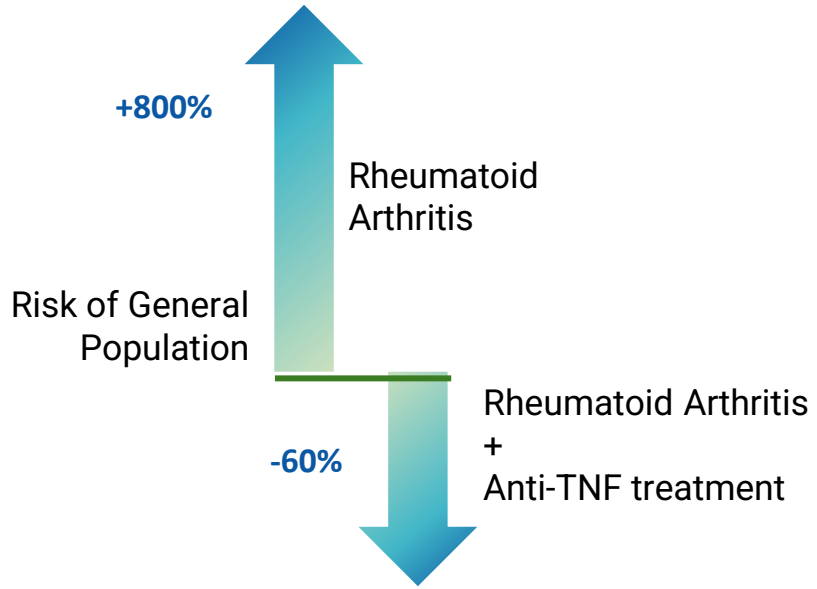
Aadapted from PMID: 27994591

- Glial cells, the immune cells of the brain, cause neuroinflammation
- Glial cells are more than 50% of cells in the brain

Targeting sTNF in Man Makes a Difference

Prevention of chronic inflammation with anti-TNF therapy lowers risk of AD

TNF Inhibitors Reduce Risk of Developing AD



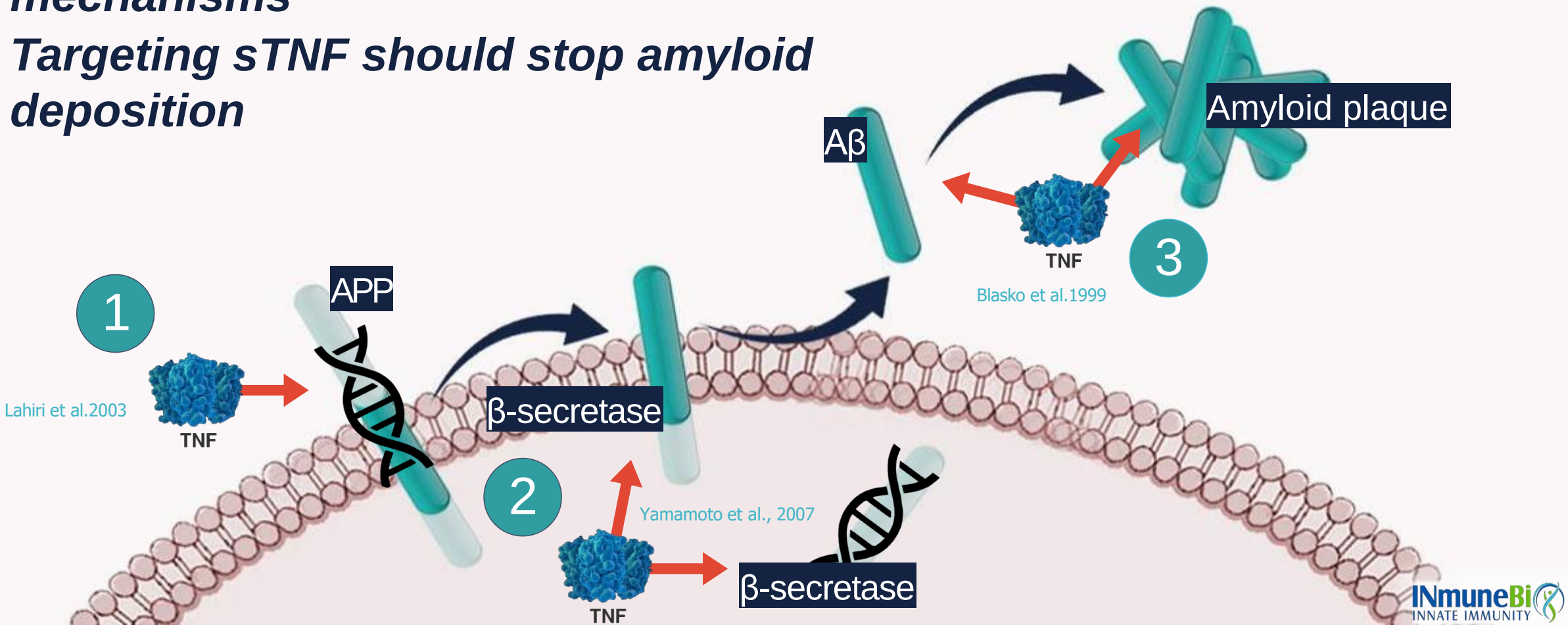
Epidemiological studies including a meta-analysis of more than 60 million cases linking **TNF Blocking Agents** to reduced risk of AD

Adapted from PMID: 27470609, 33016914

Soluble TNF Promotes Deposition of the Biomarkers of AD

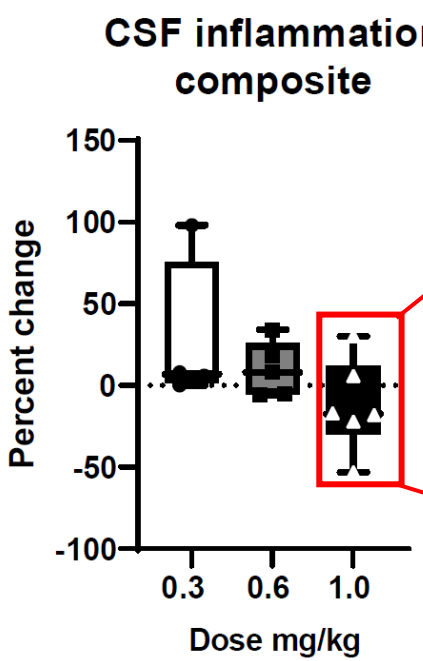
sTNF drives neuroinflammation that promotes amyloid plaque deposition

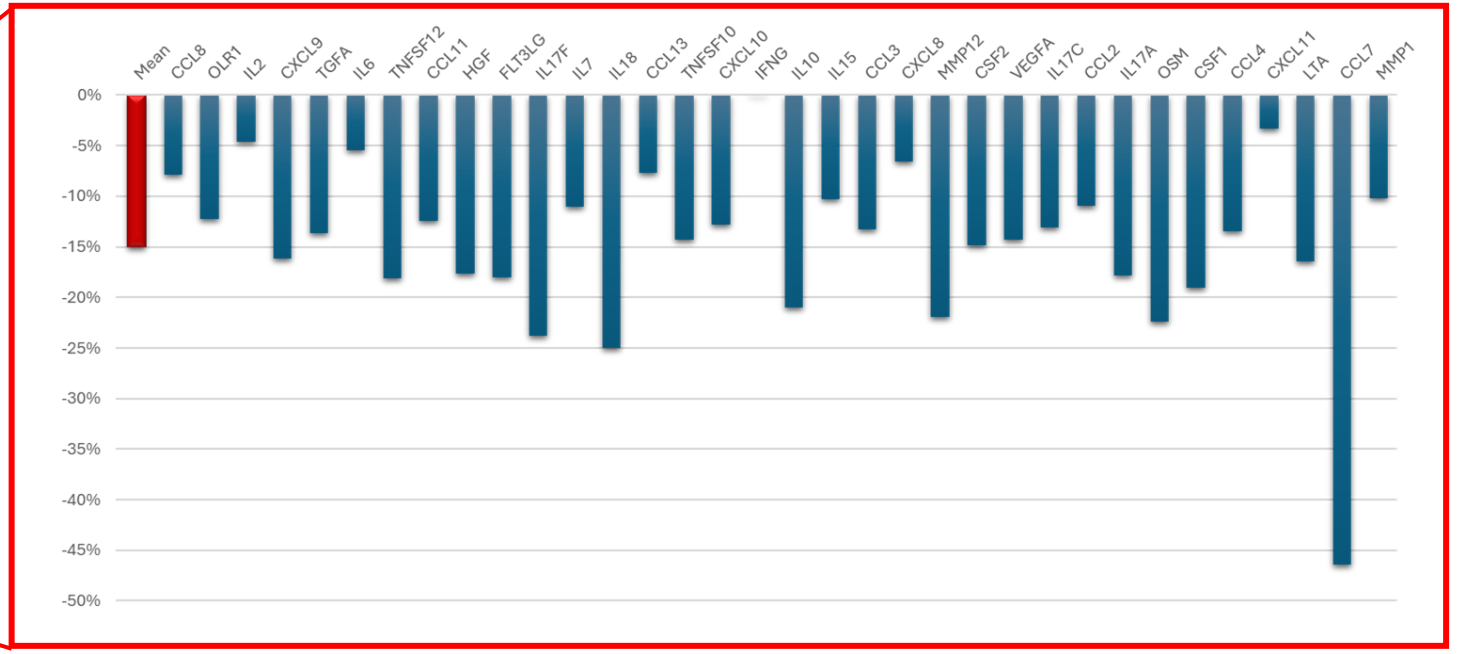
- ***TNF potently induces the expression and accumulation of Ab via a number of mechanisms***
- ***Targeting sTNF should stop amyloid deposition***



Phase 1 Results: Neutralizing sTNF with XPro™ Decreases Neuroinflammation

Dose-dependent reduction of CSF biomarkers of neuroinflammation





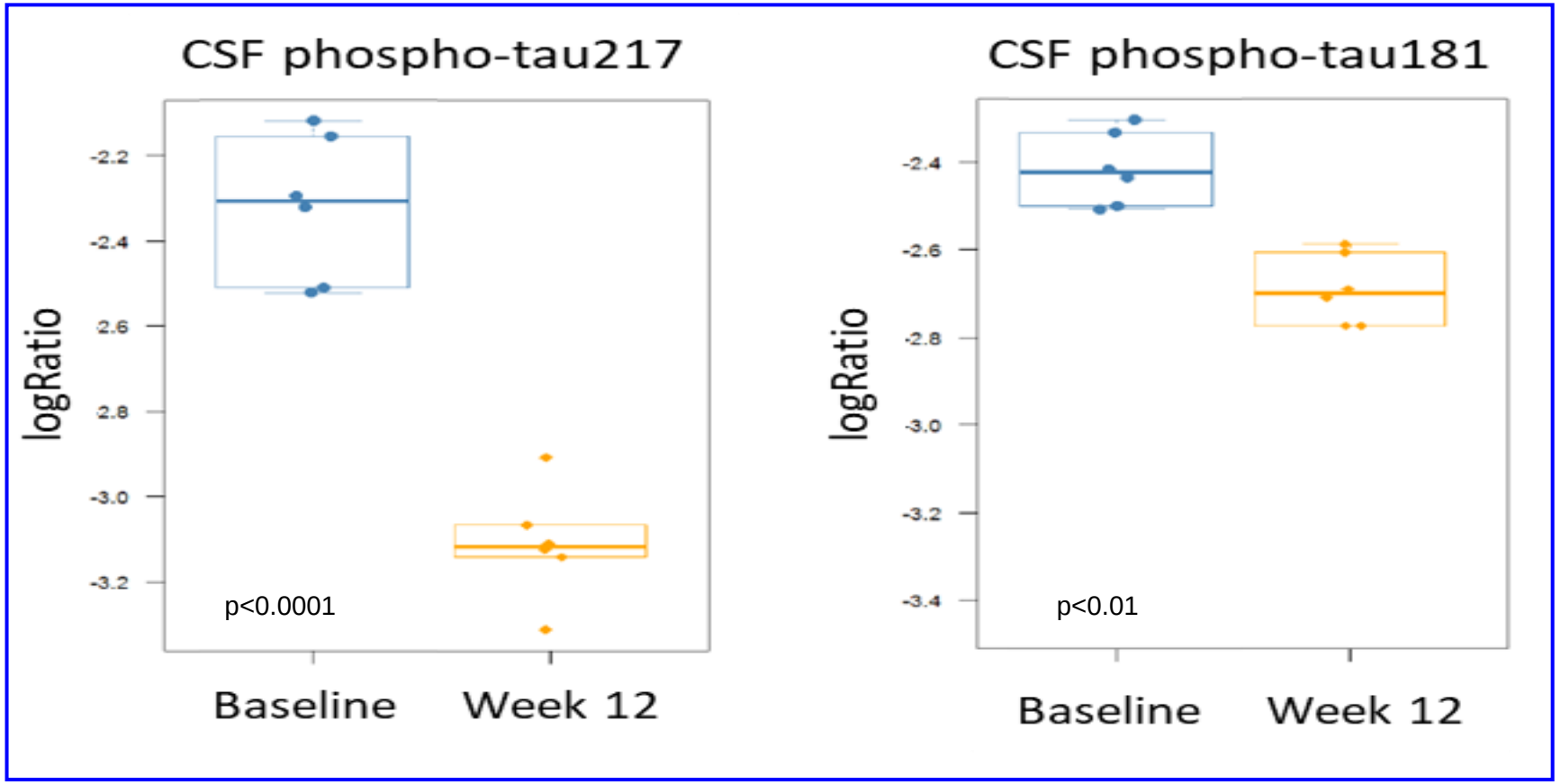
**1 mg/kg group (N=6)*

Olink® Target 48 Cytokine

Phase 1 Results: XPro™ Decreases Neurodegeneration

pTau217 is best biomarker for neurodegeneration in patients with AD*

Phase I data: XPro™ 1mg/kg subQ once a week for 12 weeks decrease pTau is CSF in patients with AD



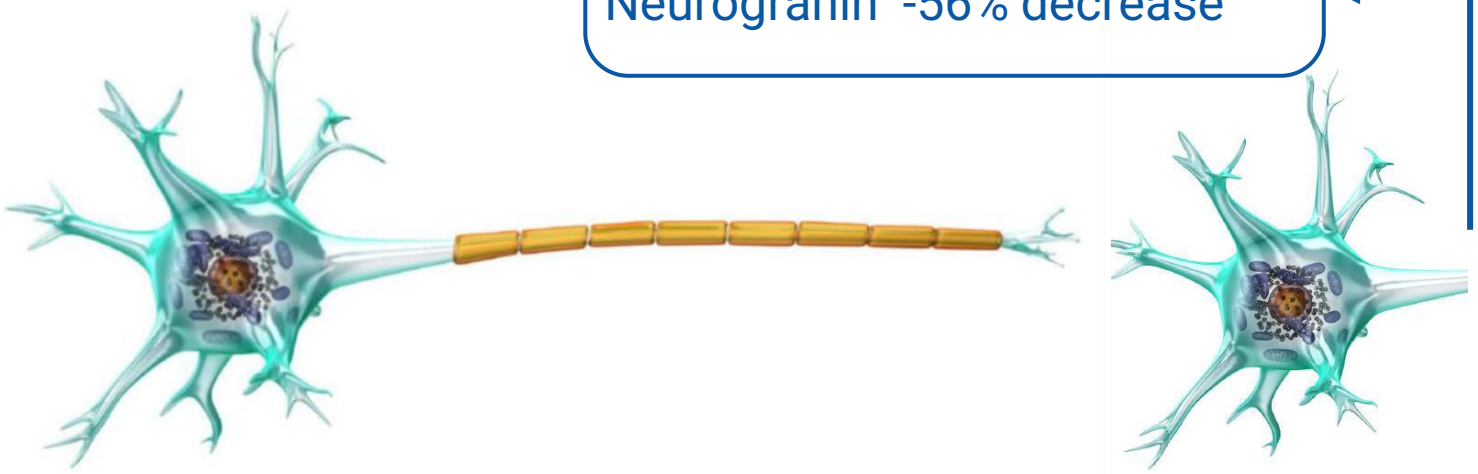
proteome sciences

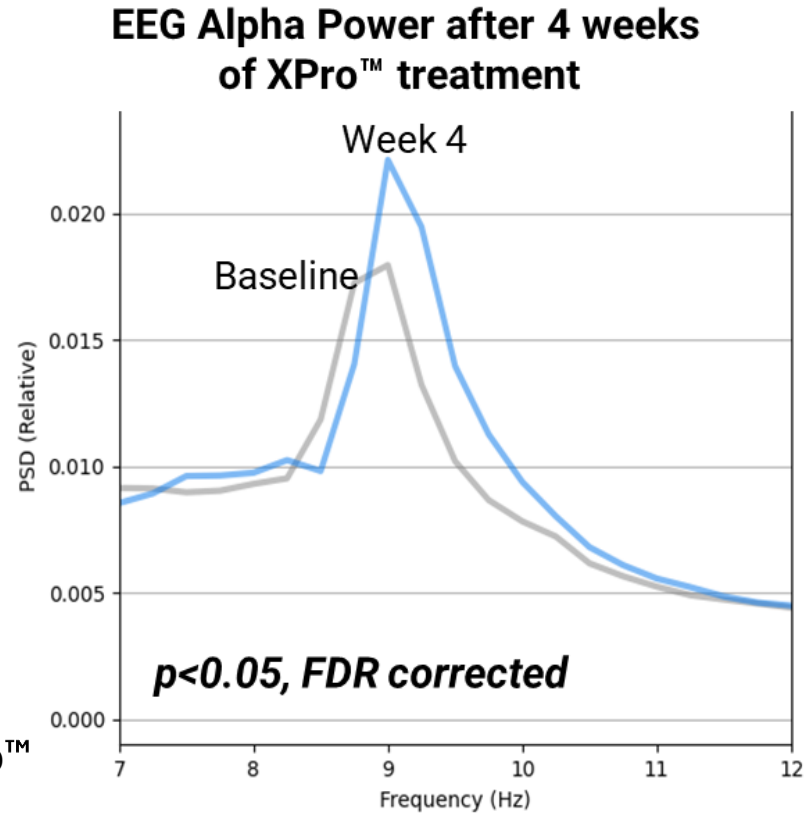
*https://jamanetwork.com/journals/jamaneurology/fullarticle/2813751

Phase 1 Results: XPro™ Improves Synaptic Function

Studies have demonstrated both changes in synaptic proteins and improvements in synaptic function as measured by EEG Alpha waves

Synaptic Proteins
Contactin-2 +222% increase
Neurogranin -56% decrease



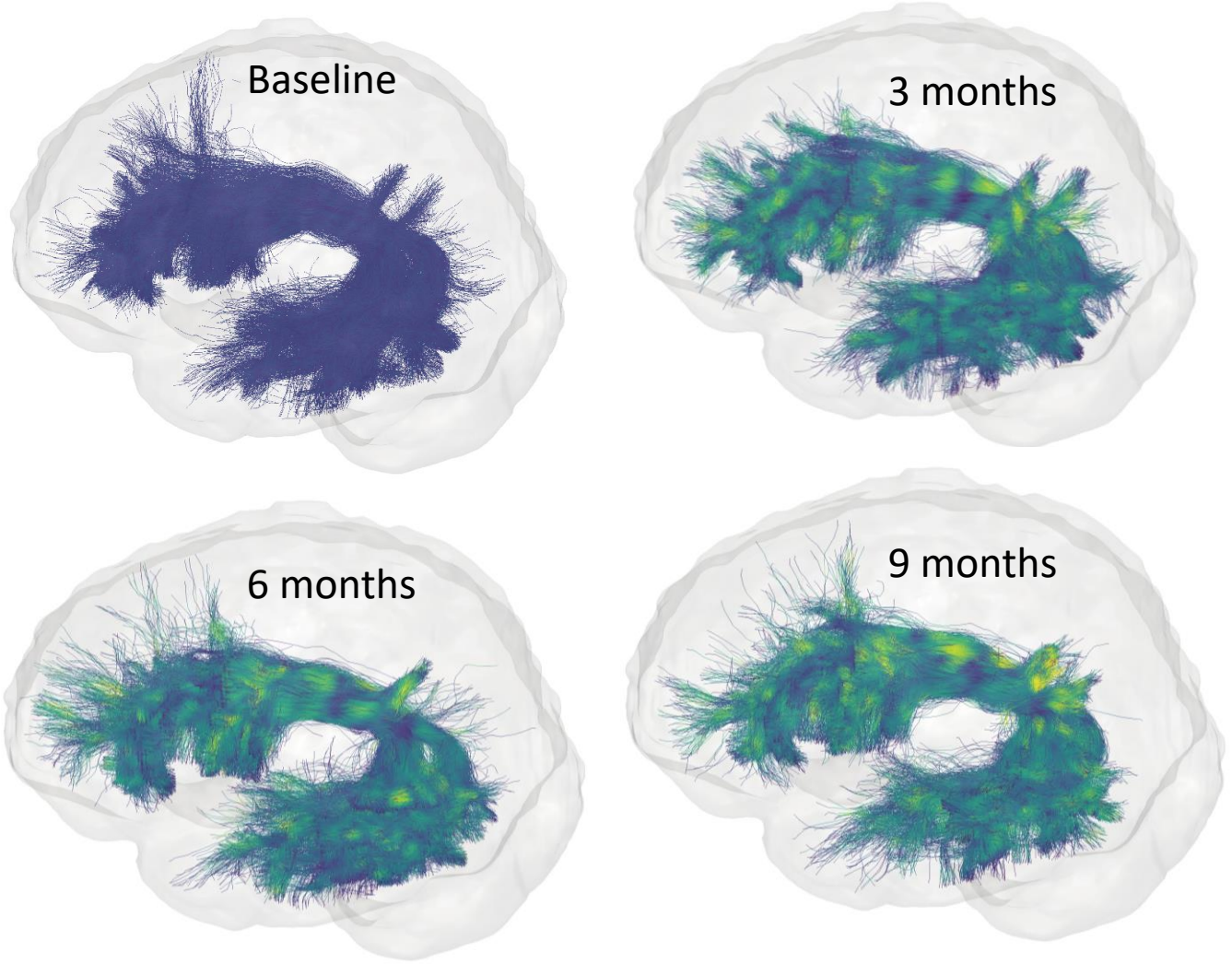


Above: CSF synaptic proteins improved after treatment with 12 weeks of XPro™
Right: Alpha Power EEG improves after 4 weeks of XPro™

Both are data from Phase I trials in AD patients with XPro 1mg/kg once a week by subQ injection

Remodeling of White Matter Tracts After XPro™

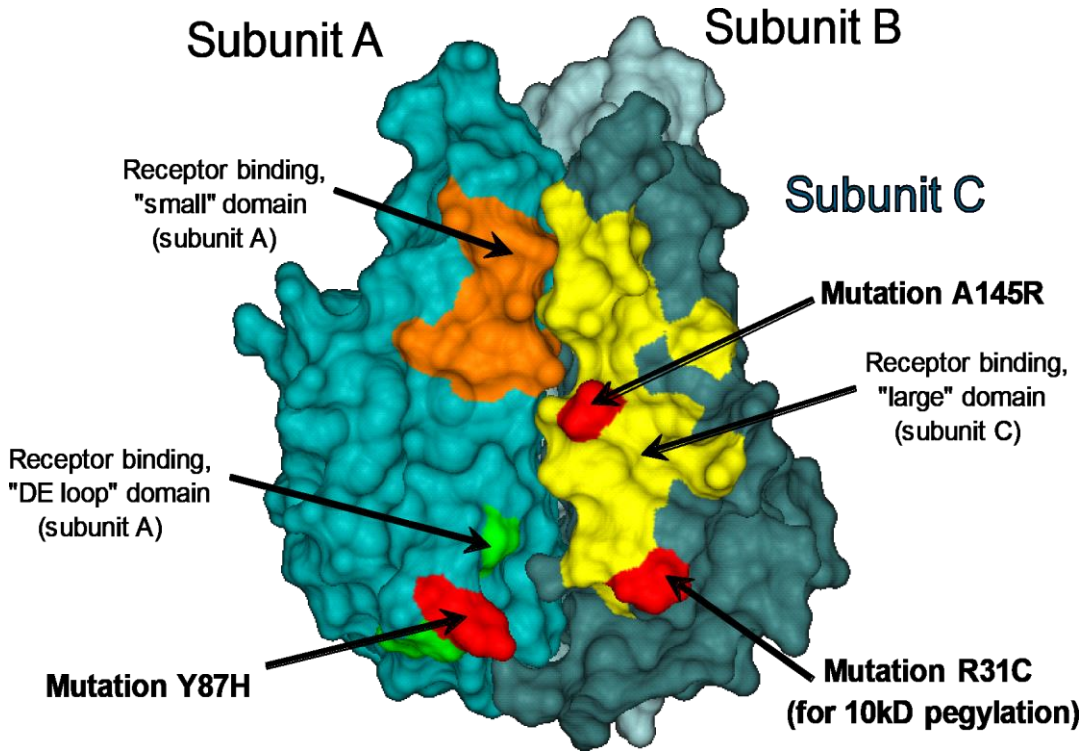
Phase 1b Data: CHANGES IN AFD IN AD WHITE MATTER TRACTS – CASE STUDY



- 65-year-old white male retired due to AD
- Returned to work after 6 months of XPro therapy
- Increasing green/blue shows improvement in axonal quality

A TNF Inhibitor Designed to Treat Neurologic Disease

XPro™: a dominant-Negative selective inhibitor of *ONLY* soluble TNF



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

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

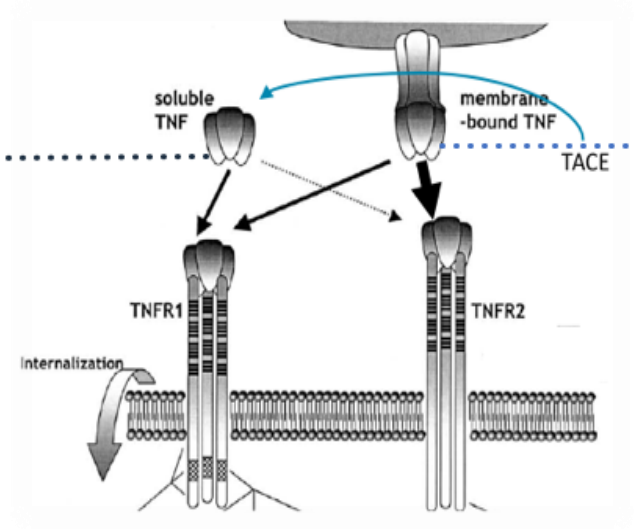
TNF Biology: Two Ligands with Opposite Effects

Soluble TNF

- Pro-inflammatory
- Demyelination
- Neurodegeneration

Neurodegenerative

TNF Biology



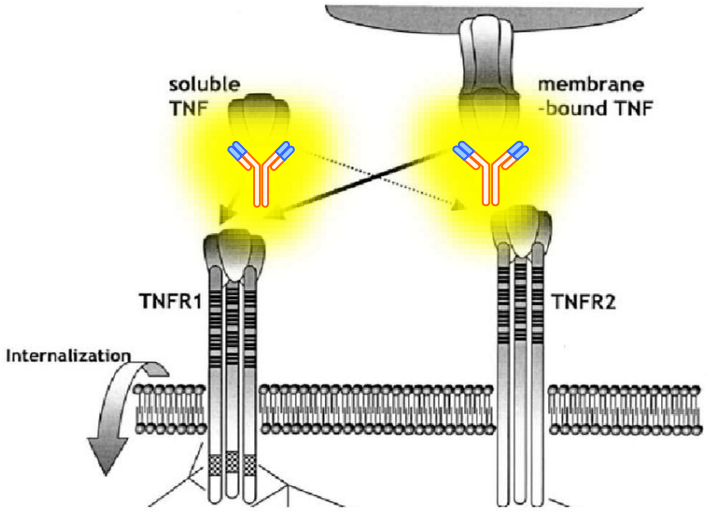
Transmembrane TNF

- Lymphoid organ development
- Myelination
- Immunity to infection
- Neuroprotection

Neuroprotective

Adapted from (MacEwan et al. 2002)

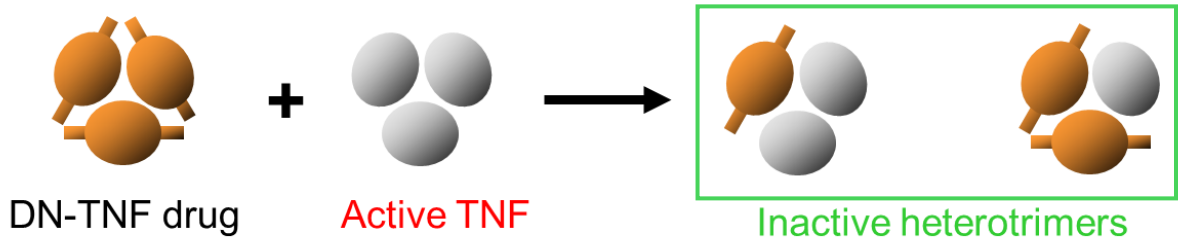
Current TNF inhibitors block both TNF ligands



XPro™ Mechanism of Action

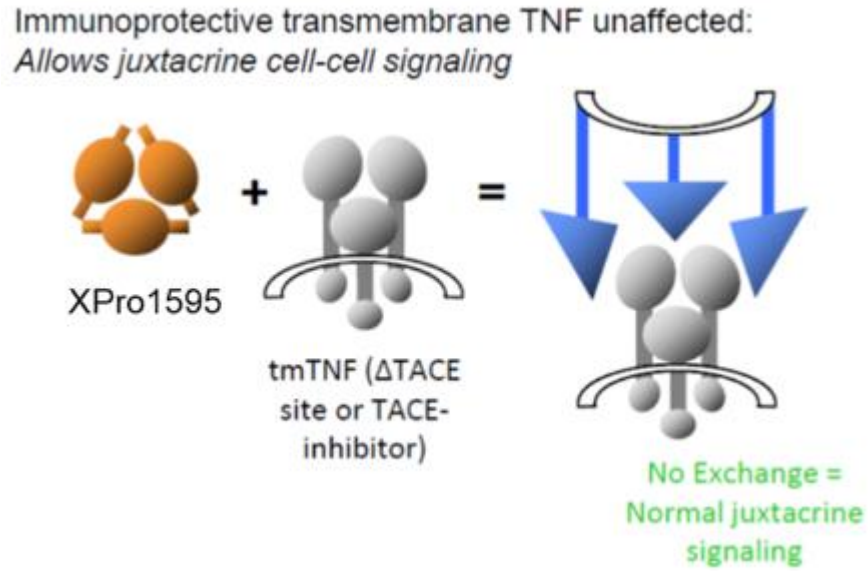
XPro™ freely exchanges with solTNF monomers to form inactive heterotrimers

Inflammatory soluble TNF eliminated:
No paracrine signaling through receptors



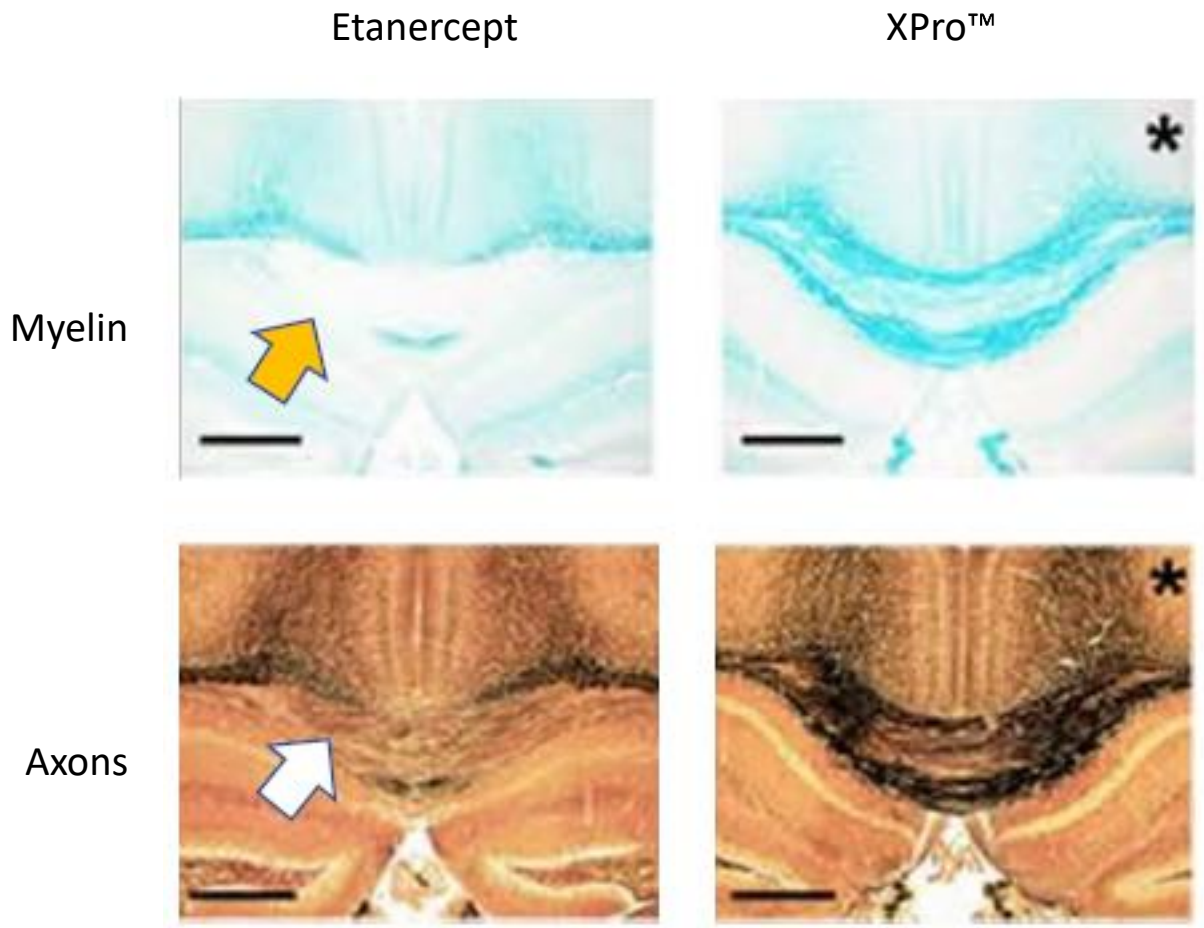
Dominant-Negative in genetics:
"A mutation producing a rogue protein that interferes with the function of the native protein."

TmTNF homotrimers are anchored to the cell membrane; XPro™ cannot exchange



XPro™ is the only TNF inhibitor that is safe for the Brain

- Currently approved TNF inhibitors are contraindicated in treatment of neurologic disease such as AD
  - promote demyelination (yellow arrow)
  - promote axon degeneration (white arrows)
- XPro™ promotes remyelination and axonal regeneration.



Karamita; Therapeutic inhibition of soluble brain TNF promotes remyelination by increasing myelin phagocytosis by microglia. https://doi.org/10.1172/jci.insight.87455

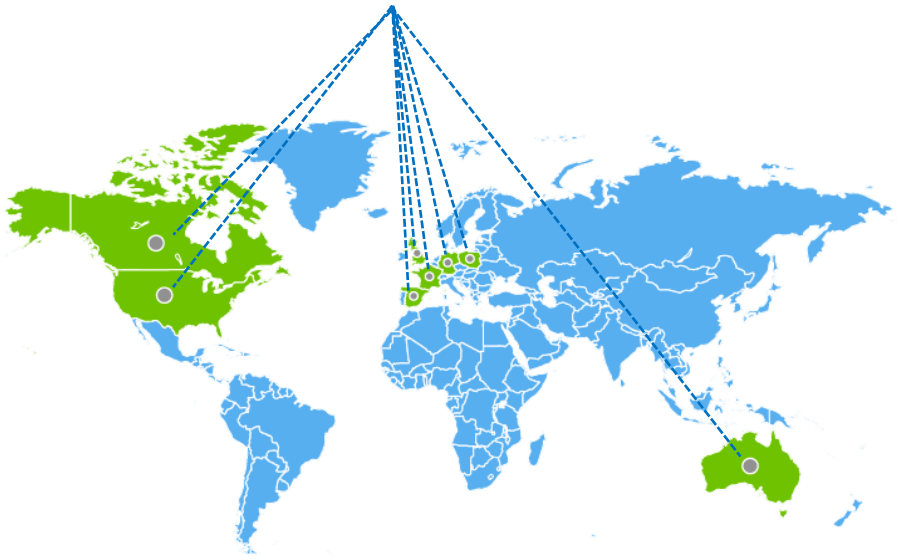
Benefits of Selective Versus Non-Selective TNF Inhibition

	Non-Selective TNF Inhibitor	XPro™
Decreases Inflammation	yes	yes
Immunosuppression	yes	No
Demyelination	yes	No
Neuroprotective	no	yes
Enhances Neuroplasticity	no	yes

Phase 2 Trial of XPro™ in Patients with Early Alzheimer's Disease

Top-Line Data Expected in Early 2025

8 Countries
35 Sites



- 6-Month Trial Powered Off CDR
- 201 Patients
- 2:1 XPro / Placebo
- Primary Endpoint: EMACC
- Secondary Endpoint: CDR
- Patient Pool Enriched for Biomarkers of Inflammation

Clinically Proven Cognitive Endpoints of EMACC and CDR

Study designed around these primary and key secondary endpoints

EMACC

- EMACC was empirically derived by pharma to measure change in Early AD
- Clinically validated measurements
- OBJECTIVE
- 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

CDR

- Approvable endpoint for AD in registration studies
- Used to power the phase 2 study

MINDFuL
A study of inflammation in Alzheimer's disease

Phase 2 Trial Summary

- **Enriching for patients that have AD with elevated neuroinflammation (ADi)**
  - ADi patients have faster progressing disease with less variance which allows for smaller trial size and shorter trial duration

- **Cognitive and functional measures that are meaningful and relevant for Early AD patients**
  - EMACC has greater dynamic range to detect change in the appropriate cognitive symptoms
  - GAS allows us to assess cognitive functional change important to each patient.
  - E-Cog allows us to assess clinically meaningful functional changes

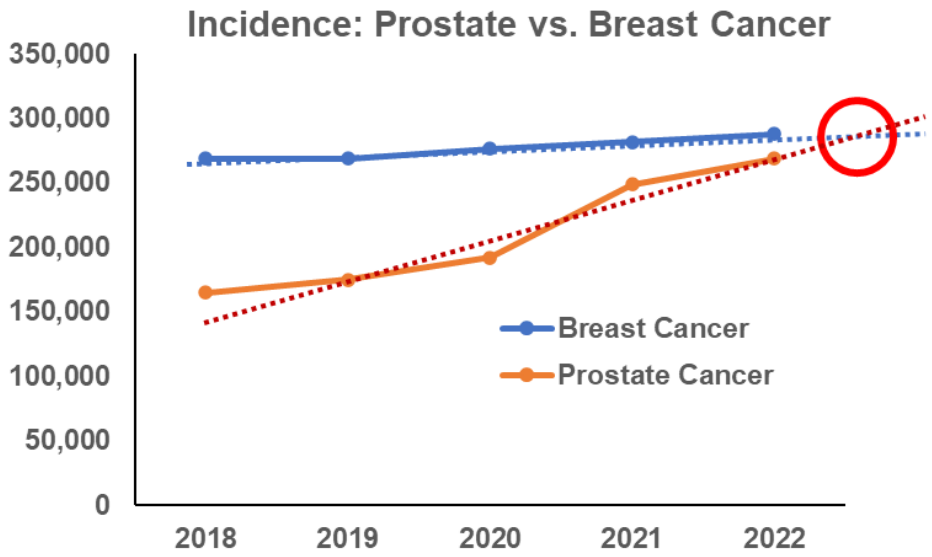
- **Novel approach to evaluate placebo response**
  - Multidimensional Psychological Questionnaire

- **Patient friendly design**
  - Short duration, 2:1 randomization, guaranteed access to drug in extension study


INmuneBio
INKmune™
for Oncology
Off-the-Shelf NK Therapy Converts Patient's Resting NK cells into Cancer Killing memory like NK cells

Problem: New Treatments for Metastatic Castration Resistant Prostate Cancer (mCRPC) Have Little Impact on Survival

- Incidence of prostate cancer increasing
- New therapies for mCRPC give <6 month survival benefit
- Immune check-point inhibitors have failed in mCPRC

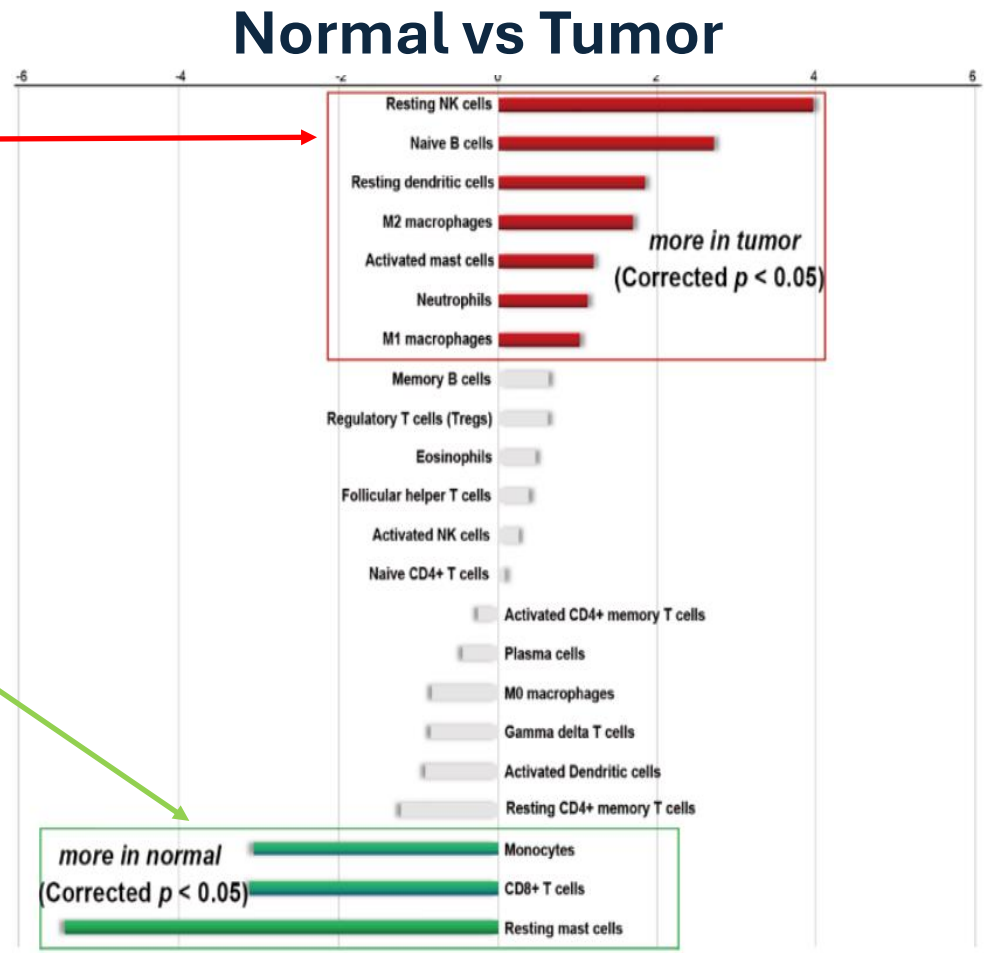


Agent	Sipuleucel-T	Abiraterone	Enzalutamide	Docetaxel	Cabazitaxel	Radium-223	PSMA RLT	Olaparib
Median OS benefit (months)	4.1	Post-doc: 4.6 Pre-doc: 4.4	Post-doc: 4.8 Pre-doc: 4.0	2.4	2.4	3.6	5.3	2.3

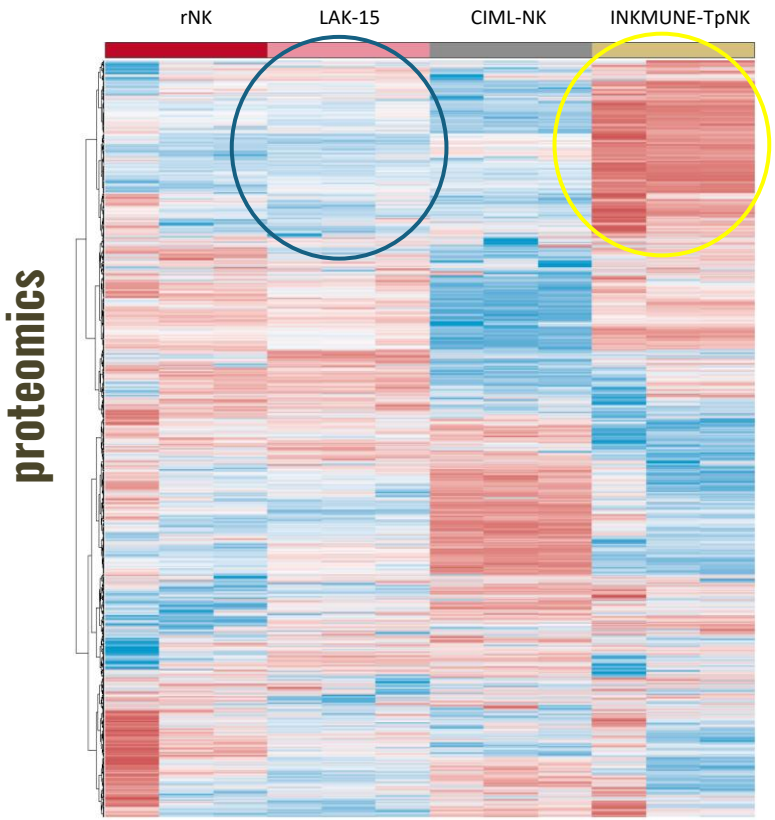
Solution: Use INKmune™ to Target NK Cells in mCRPC

in vitro* - INKmune™ induced changes needed to promote NK function in Tumor MicroEnvironment (TME)

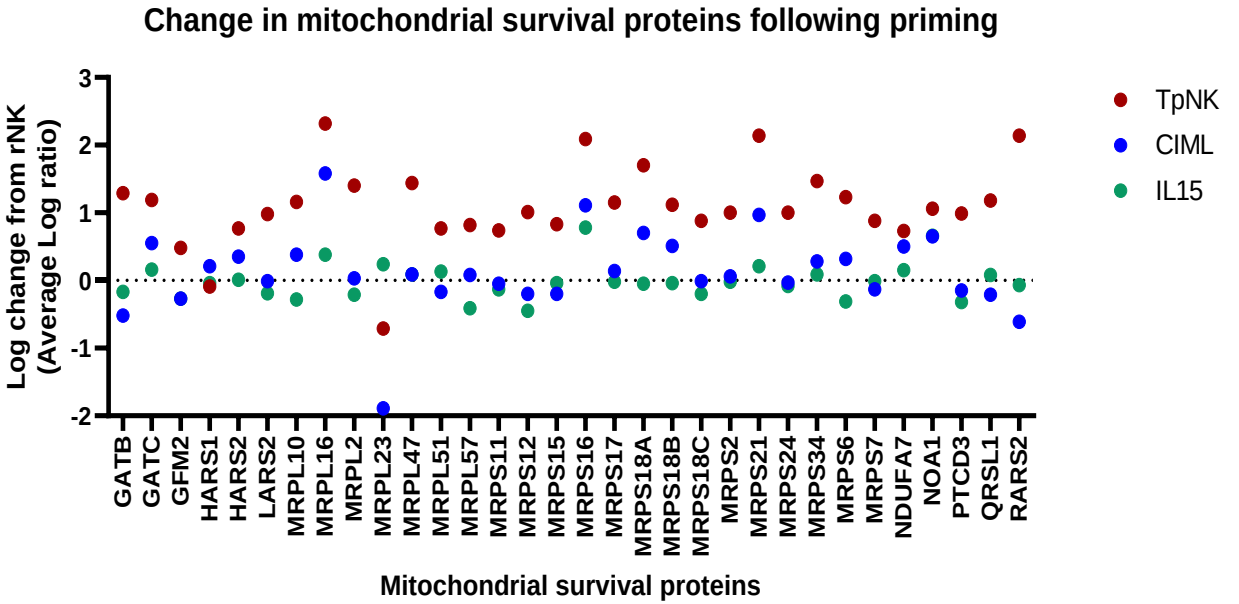
- Prostate cancer immune infiltrate cells are resting NK cells ***NOT*** T cells
- Is lack of T cell infiltrate why PDL1 and TIGIT fail in mCRPC?
- NK cells in mCRPC are resting NK cells that do not kill tumor
- INKmune goal: convert resting NK cells to cancer killing memory like NK cells

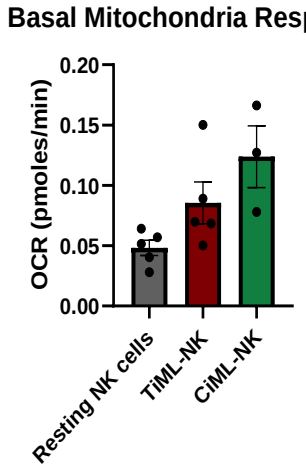


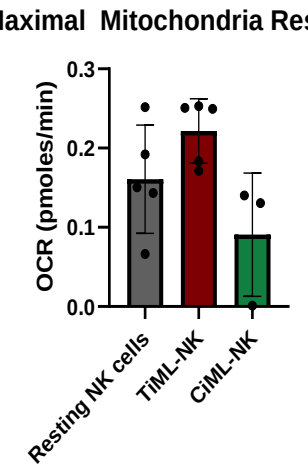
INKmune™ Primed NK Cells "Fitter" Than Cytokine Primed NK Cells

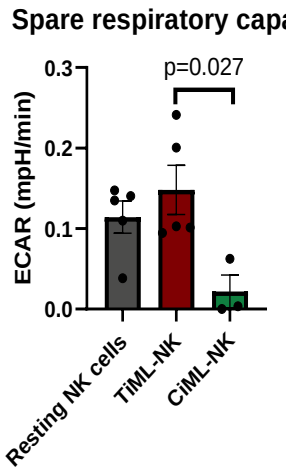


*studies of human NK cells targeting human prostate cancer cells







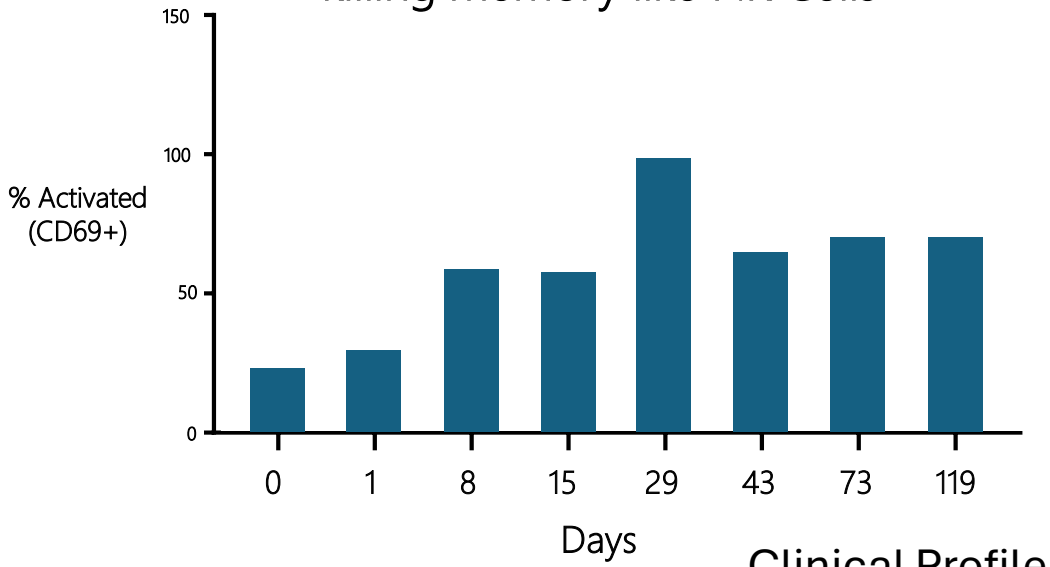


INKmune Primed NK Cells Present in Circulation Longer Than Cytokine Primed NK Cells

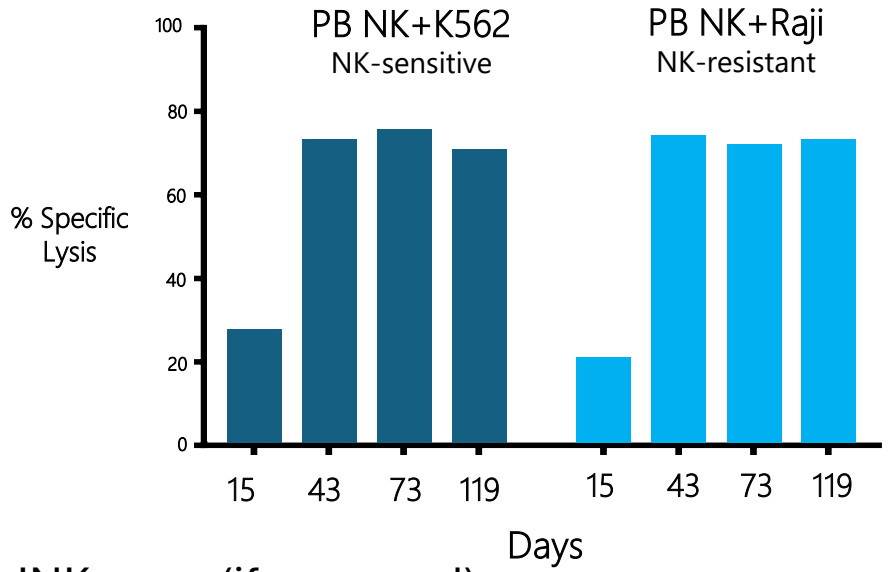
Most important elements of INKmune therapy:

- INKmune primed NK cells seen in patient blood for months
- INKmune primed NK cells kill NK resistant targets *in vitro*

INKmune activated tumor-killing memory like NK Cells



INKmune induces memory-like NK cells with enhanced lysis of NK-sensitive and NK-resistant cells



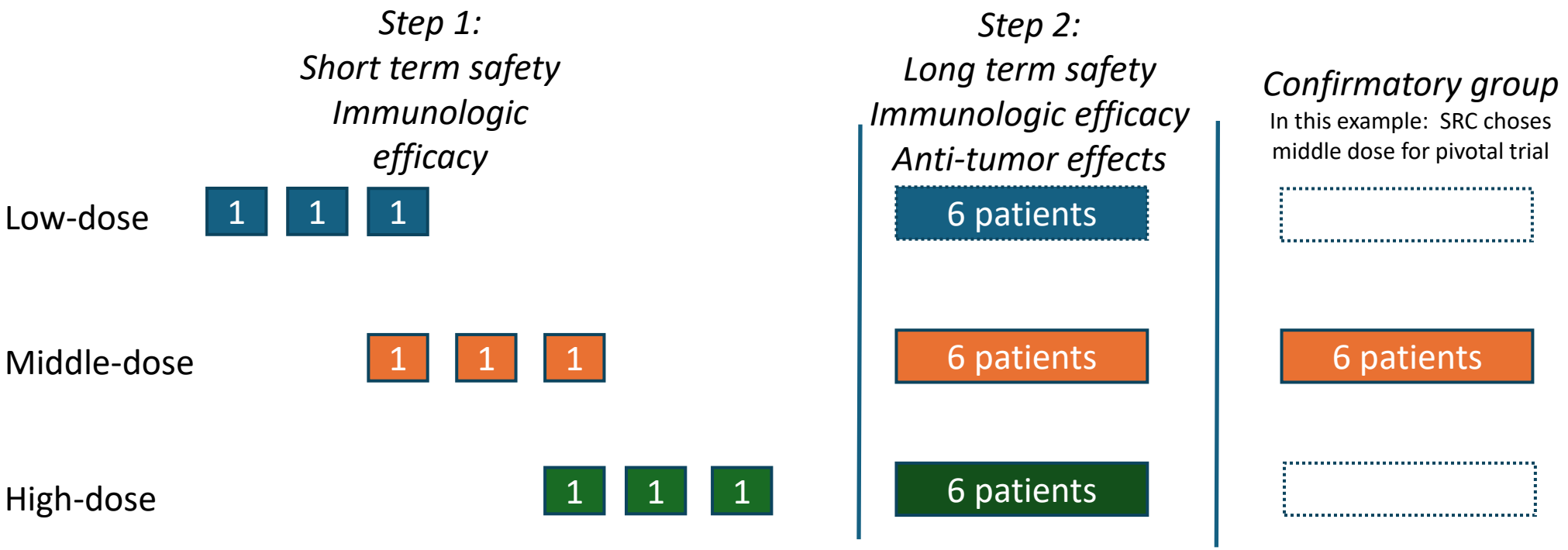
<u>Clinical Profile Goals for INKmune (if approved):</u>

Safe and well-tolerated as an out-patient

Controls disease with excellent QOL

Bridge to transplant in AML

INKmune™ mBION12 mCRPC Trial Design



Definitions:

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

Anticipated Milestones in 2024 and 2025

Key Upcoming Clinical & Regulatory Milestones

	EVENT	EXPECTED TIMING
XPro™	Complete Phase 2 AD Enrollment	Q3 2024
	Topline Phase 2 AD Data	~6m From Last Patient Enrolled
	End of Phase 2 FDA Meeting AD	Mid 2025
	Pre-clinical Anti-AB and XPro Data	2H 2024
	Initiate Phase 2 TRD Trial	2H 2024
INKmune™	Complete Phase 2 mCRPC Enrollment	1H 2025
	Topline Phase 2 mCRPC Data	2H 2025
	End of Phase 2 FDA Meeting mCRPC	4Q 2025 or 1Q 2026

Contact Us:

Inflammation and Immunology Repair

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