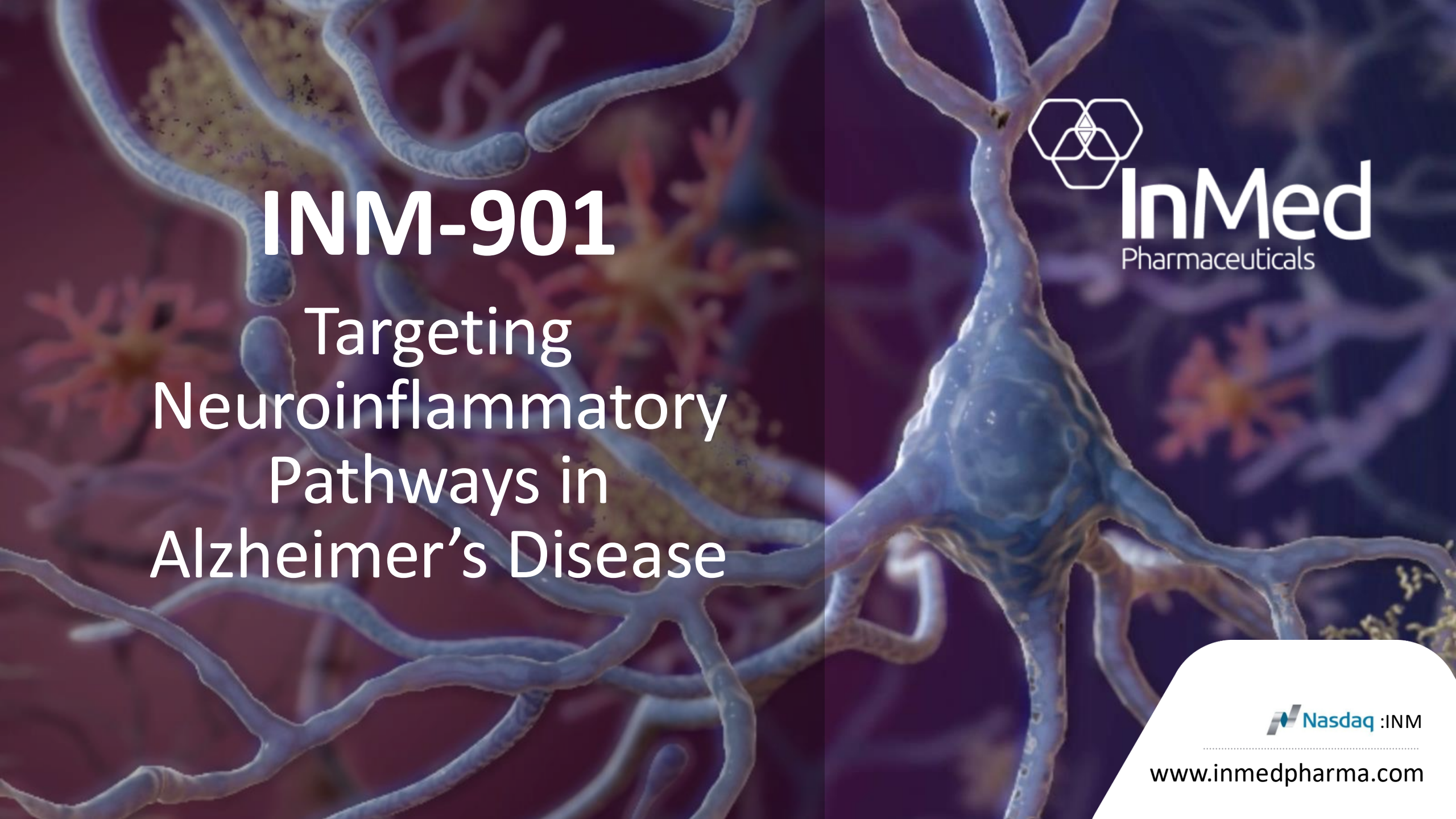


INM-901

Targeting Neuroinflammatory Pathways in Alzheimer's Disease

InMed Pharmaceuticals

Nasdaq :INM

www.inmedpharma.com

Forward Looking Statements

This presentation contains forward-looking statements and forward-looking information within the meaning of applicable securities laws (collectively, "forward-looking statements") including, among others, statements concerning: anticipated development activities, timelines, catalysts, and milestones; the potential benefits of product candidates; anticipated revenue and market opportunities; and the continued availability of key personnel. All statements other than statements of historical fact are statements that could be deemed forward-looking statements.

With respect to the forward-looking information contained in this presentation, the Company has made numerous assumptions regarding, among other things; INM-901 shows improved neuronal function, neuroprotection, as well as an improvement in cognitive function, memory, locomotor and anxiety-based behavior; shows increased neurite outgrowth, signifying potential for enhanced neuronal function; INM-901 demonstrating potential to target several biological pathways associated with Alzheimer's disease; INM-901 is a Proprietary small molecule compound can cross BBB; can be formulated orally; INM-901 shown to have statistical significance in the reduction of neuroinflammation; INM-901 is shown to have a positive effect on neuroprotection, cytotoxicity, neurite outgrowth, neuronal function, locomotion, cognition, memory and inflammation; preferential signaling ligand for CB1 and CB2; ongoing CMC activities for drug substance and drug product; advancing INM-901 in preclinical in vivo studies and CMC preparation.

These statements are based on management's current expectations and beliefs and are subject to a number of risks, uncertainties and other factors that could cause actual results to differ materially from those described in the forward-looking statements. These risks and uncertainties include, among others: the possibility that clinical trials will not be successful, or be completed, or confirm earlier clinical trial results; risks associated with obtaining funding from third parties; risks related to the timing and costs of clinical trials; key personnel may become unable to serve the Company; the need for receipt of regulatory approvals; changes in regulations that are adverse to our business; and economic and market conditions may worsen. This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. Readers are cautioned that the foregoing list is not exhaustive. A more complete discussion of the risks and uncertainties facing InMed's stand-alone business is disclosed in InMed's Annual Report on Form 10-K and other filings with the Security and Exchange Commission on www.sec.gov as well as Company's full financial statements and related MD&A for the fiscal year ended June 30, 2025 and the first quarter of fiscal 2026 ended September 30, 2025, are available at www.sedar.com. The Company undertakes no obligation to update the forward-looking statements contained herein or to reflect events or circumstances occurring after the date hereof, except as required by law.

Alzheimer’s Disease – A Major Medical & Societal Burden

CURRENT TREATMENT OPTIONS DO NOT REVERSE DISEASE EFFECTS

What is Alzheimer’s Disease?

Alzheimer's is a subset of dementia that impacts the part of the brain that controls thought, memory and language and leads to increased morbidity and mortality.

The two most recognized hallmarks of Alzheimer’s disease are the build-up of amyloid-beta plaques and neurofibrillary tangles caused by tau proteins. Emerging research indicates that neuroinflammation is also a factor. Lifestyle and genetics are likely contributors to disease development.

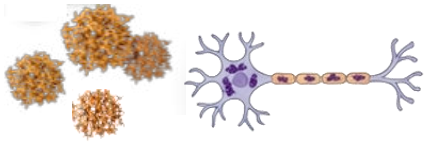
Impact

- 7.2M Americans affected
- 1 in 9 people age 65+ (11%)
- 1 in 5 women, 1 in 10 men
- 6th leading cause of death for 65+
- Alzheimer’s accounts for 60-80% of dementia cases
- U.S. annual financial impact $384B in 2025
  (Alzheimer’s and other dementia)

Source: Alzheimer’s Association (U.S.)

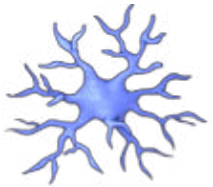
A New Perspective on Inflammation Response in Alzheimer's Disease

OLD VIEW



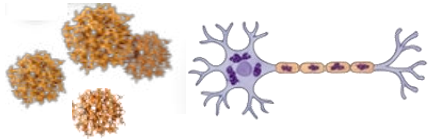
Aβ & Tau

Neuronal Damage

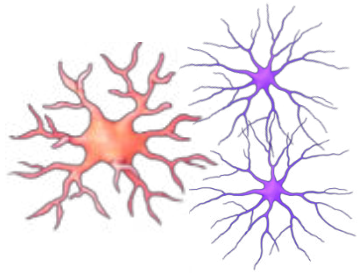


Inflammation
(Cleanup Response)

NEW VIEW



Aβ & Tau



Neuroinflammation

Vicious Cycle

Neuronal Damage

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Snapshot: Alzheimer's Disease Treatments in Development

ANTI-AMYLOID BETA AND ANTI-TAU TARGETS DOMINATE ALZHEIMER'S TREATMENTS IN LATE DEVELOPMENT

Therapeutic Targets

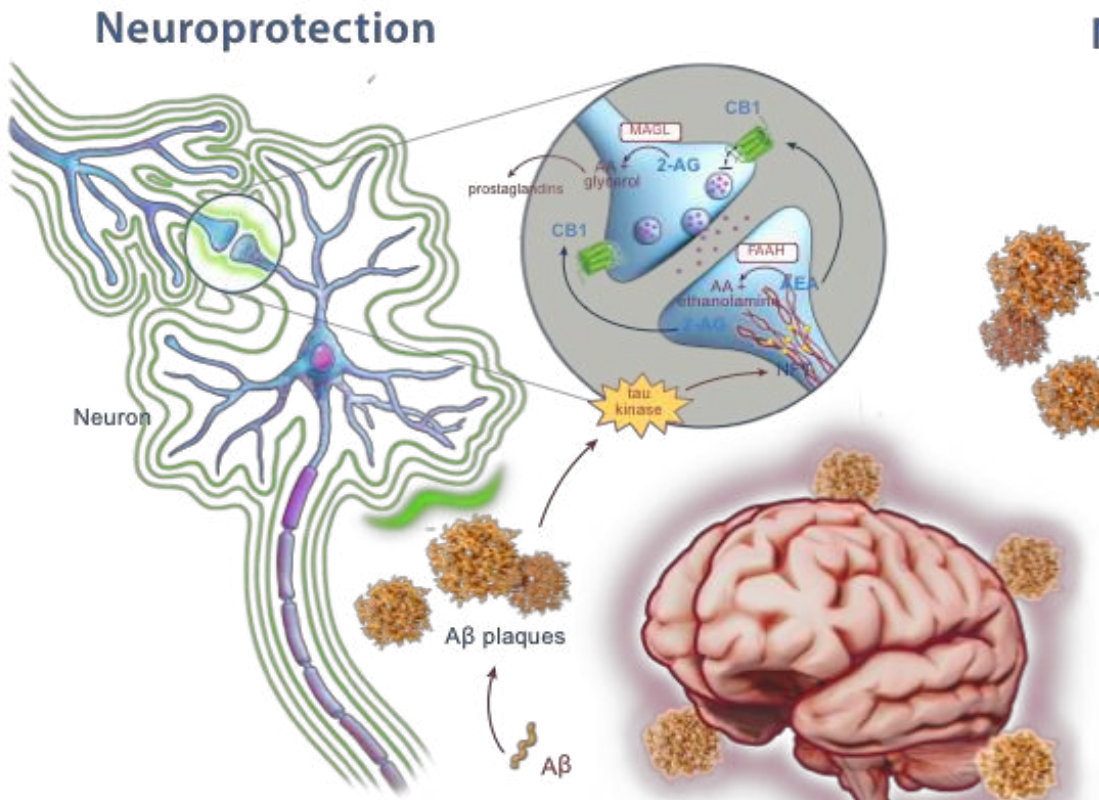
- 30 neurotransmitter receptors (22%)
- 25 amyloid beta (18%)
- **24 neuroinflammation (17%)**
- 15 tau-related processes (11%)
- 9 synaptic plasticity/neuroprotection (6%)
- 8 metabolism and bioenergetics (6%)
- 5 growth factors/hormones (4%)
- 3 multi-target (2%)
- 3 oxidative stress (2%)
- 3 proteostasis/proteinopathy (2%)
- 3 vasculature factors (2%)
- 2 ApoE, lipids, and lipoprotein receptors (1%)
- 2 gut-brain axis (1%)
- 2 neurogenesis (1%)
- 1 circadian rhythm (1%)
- 3 undisclosed (2%)

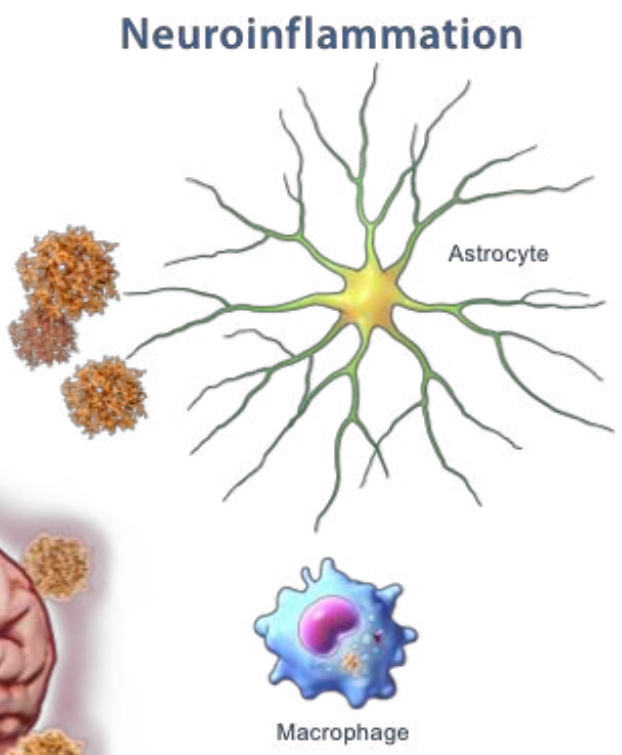
Alzheimer's Therapeutic Development

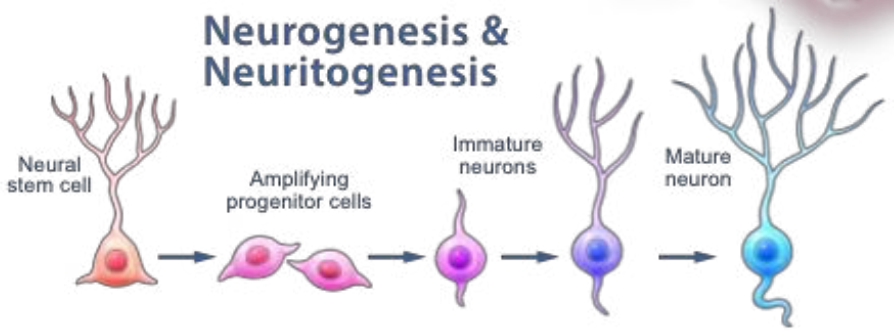
- 2 approved disease-modifying products (large molecule, anti-amyloid beta)
- 138 drugs in 182 clinical trials (31 in Phase 3; 75 in Phase 2; 45 in Phase 1)
- 102 disease-targeted therapies
- 60 small molecules, 42 biologics

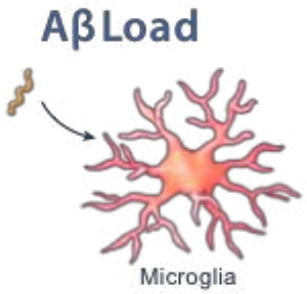
Source: Alzheimer's disease drug development pipeline: 2025 (Cummings et al), April 2025

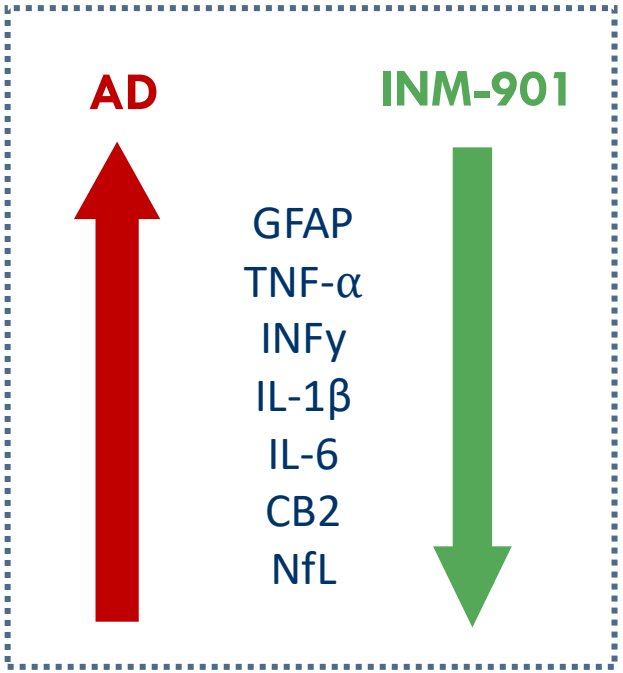
INM-901: May Address Multiple Alzheimer's Pathologies











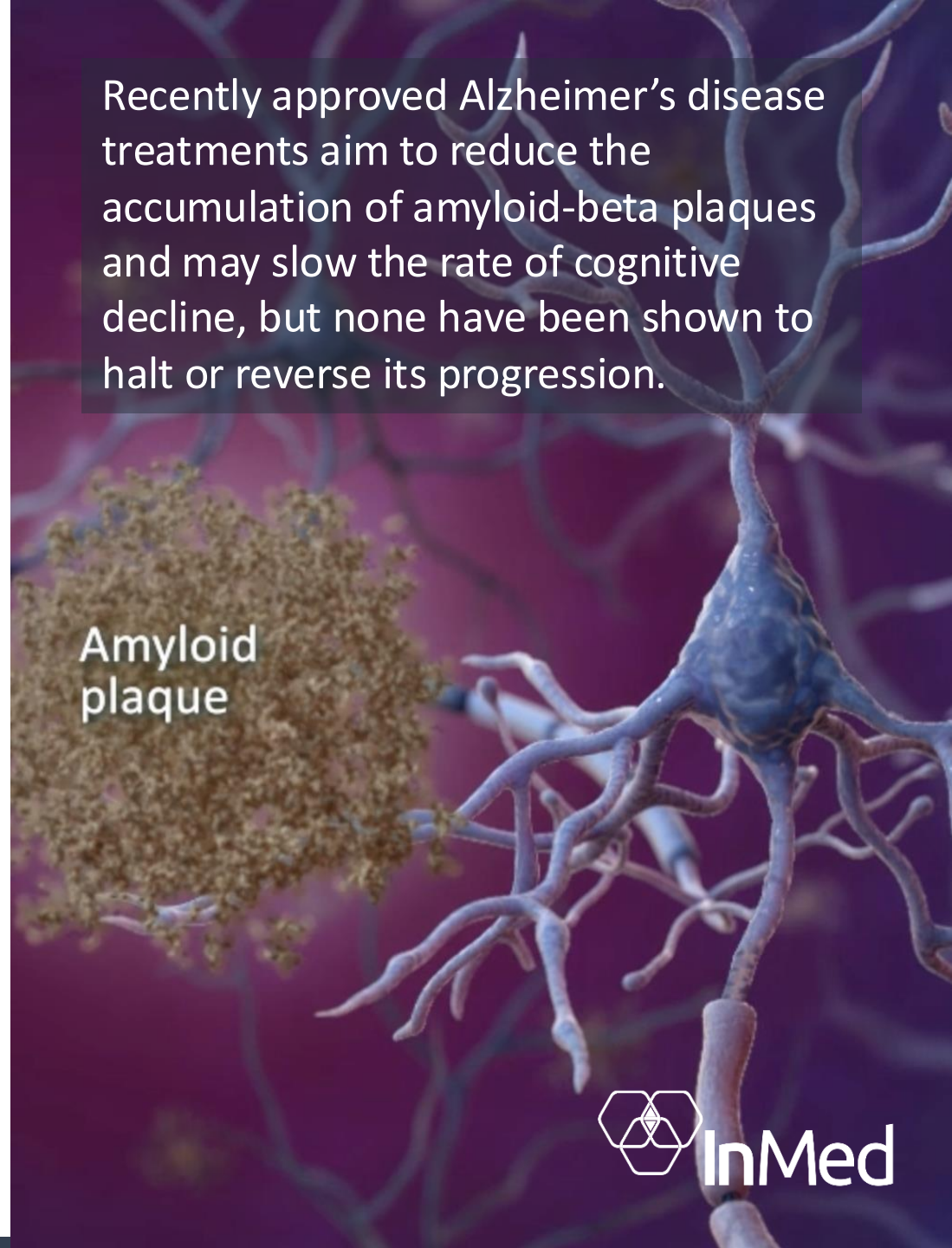
Limitations of Anti-Amyloid Beta Targeting Monoclonal Antibodies

	Aβ Antibody Profile	INM-901 Target Profile*
Limited Therapeutic Effects	Limited evidence of restoring lost cognitive function	Aims to reverse disease effects and restore cognitive function
Side Effects Risk/Benefit Profile	Amyloid-related Imaging Abnormalities (ARIA), brain swelling, brain bleeding	ARIA-type side effects not anticipated
Ongoing Screening Requirement	Requires MRI brain scan 1-2x/yr	No ongoing MRI screening anticipated
Drug Delivery Challenges	Intravenous 45-60 min. infusion every 2-4 weeks with trained medical staff	Oral formulation preferable to IV infusion
Accessibility	Typically available only at specific infusion sites, limiting accessibility	No limitations on accessibility

*Based on non-human *in vivo* data to date

Nasdaq : INM

Recently approved Alzheimer's disease treatments aim to reduce the accumulation of amyloid-beta plaques and may slow the rate of cognitive decline, but none have been shown to halt or reverse its progression.



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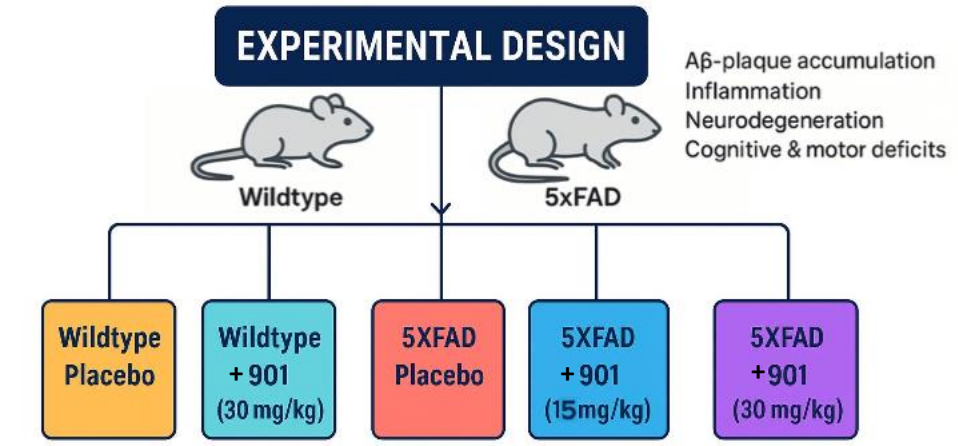
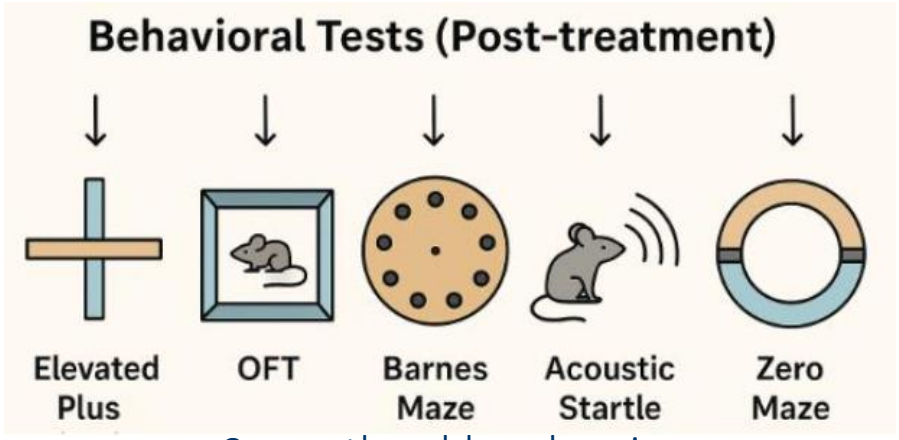
Well-characterized Study Design Using 5xFAD Mouse Model

LONGER TREATMENT DURATION AND ADVANCED DISEASE STAGE

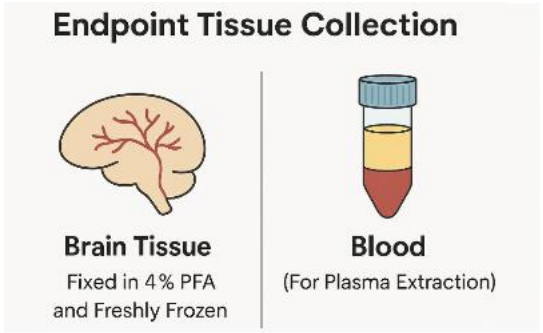


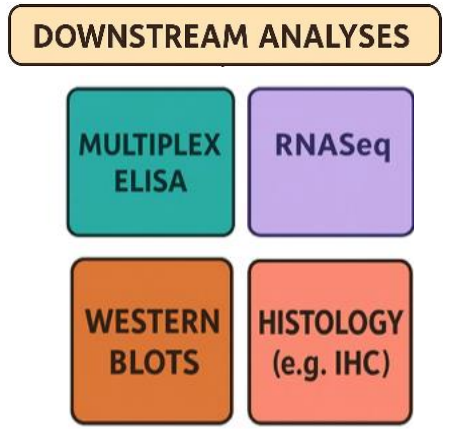
2-month-old male mice were injected twice-weekly for 7 months



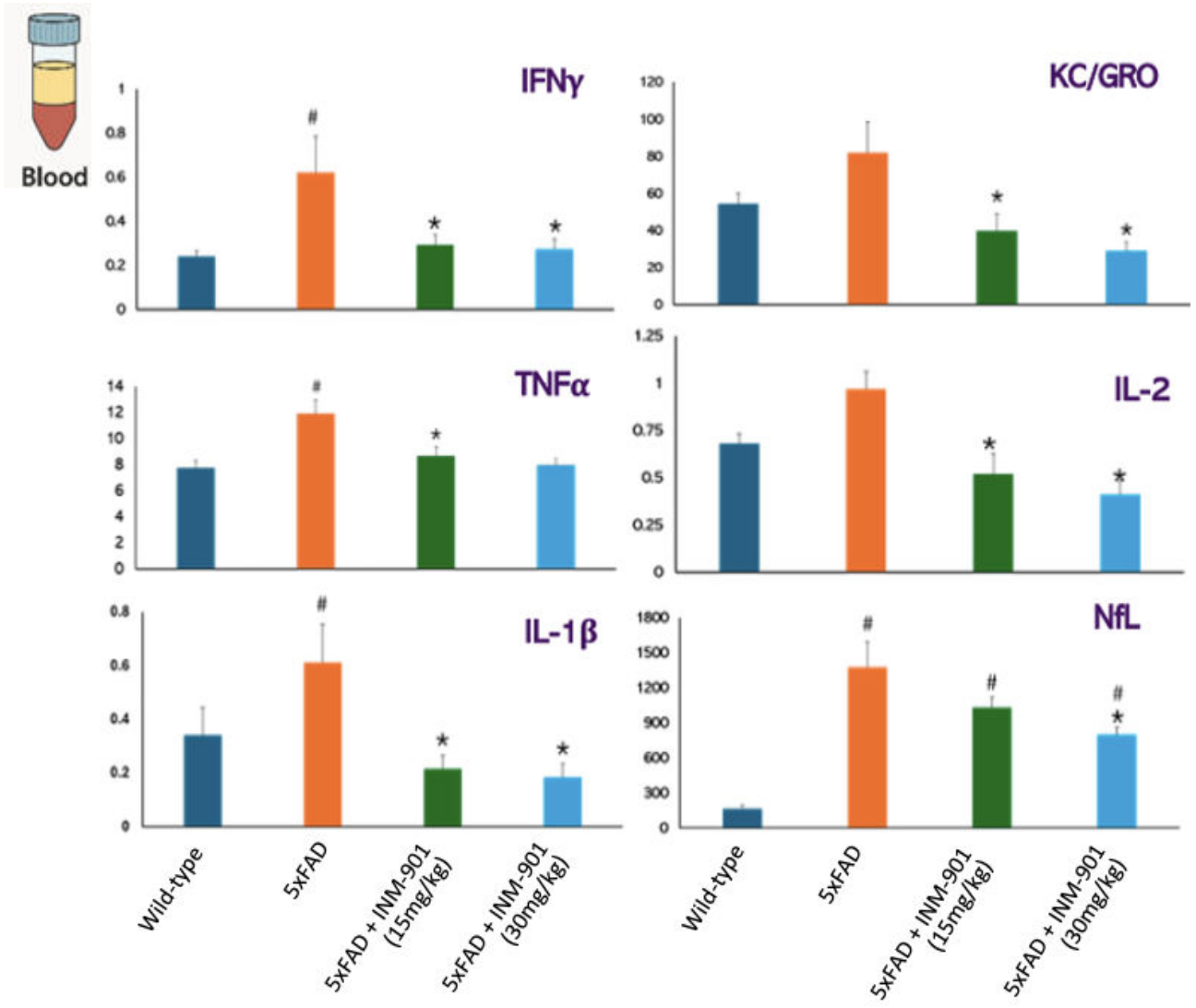
9 months old male mice

This study evaluates INM-901 in the 5xFAD AD mouse model, using a longer treatment duration and a more advanced disease stage to validate and expand upon previous short-term findings.





INM-901 Demonstrates Strong Neuroinflammatory Modulation in 5xFAD Mouse Model



Plasma Inflammatory Markers

5xFAD transgenic mice exhibited significantly elevated levels of these markers compared to Wild-type mice.

Treatment with INM-901 at 15 or 30 mg/kg resulted in a significant reduction in these biomarkers, suggesting a dose-dependent therapeutic effect of INM-901 in the 5xFAD model.

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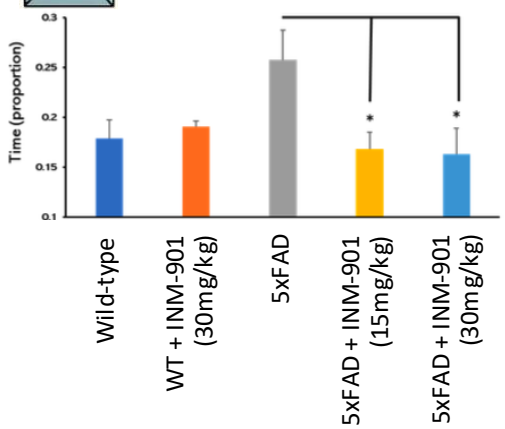
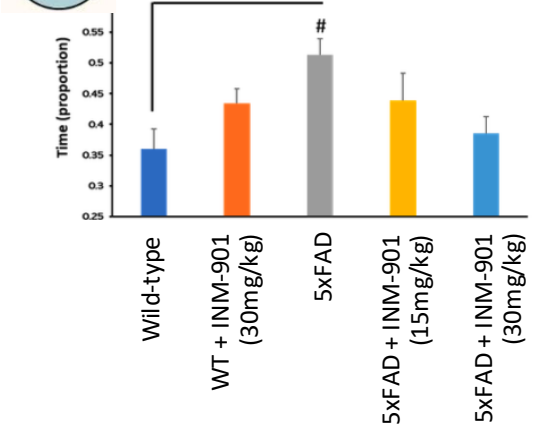
INM-901 Demonstrates Positive Trends in Cognitive Function, Anxiety-Related Behavior and Sensory Responsiveness

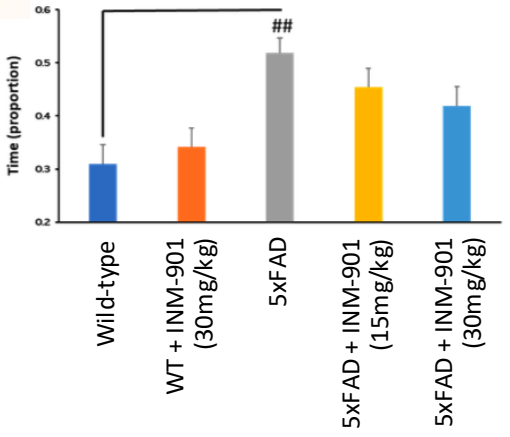
Open Field Test (Centre)



Zero Maze (Open Section)

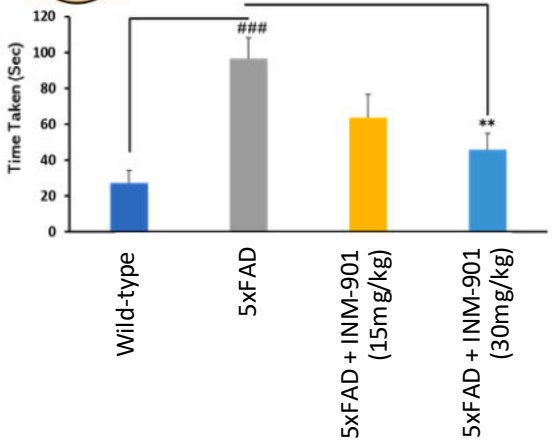


Elevated Plus Maze (Open Arm)



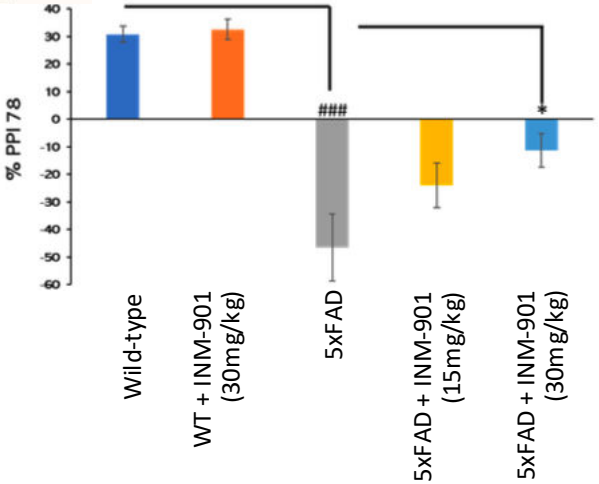
5xFAD mice spent more time in the center zone or open arms, suggesting reduced anxiety-like behavior. INM-901 treatment restored typical anxiety-like behavior similar to wild-type mice.

Barnes Test



5xFAD mice showed impaired special learning and memory, while INM-901 treatment improved performance.

Acoustic Startle Response



5xFAD mice showed reduced acoustic startle response compared to Wild-type mice, indicating sensory dysfunction, which was partially restored by INM-901 treatment.

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Summary: INM-901 Potentially Mitigates AD Pathology

Anti-Inflammatory Action – 5xFAD

Significantly reduced inflammatory biomarkers IFN-γ, TNF-α, IL-1β, KC-GRO, IL-2 & NfL in 5xFAD Alzheimer's model

Anti-Inflammatory Action – LPS-induced

Significantly reduced inflammasome marker activation of NLRP3 and IL-1β, key contributors to neurodegeneration

Direct Impact on Neuroinflammation

Demonstrated anti-inflammatory effects independent of amyloid-beta or tau pathology

Neuroprotection

Significantly reduced amyloid-beta-induced cell death in *in vitro* studies

Robust Bioavailability

Oral formulation achieved anticipated therapeutic levels of systemic exposure

INM-901

Neuronal Regeneration

Promotes neurite outgrowth, indicating its ability to enhance neuronal connectivity and function

Molecular Validation

mRNA data supports observed improvements in cognition, memory and neurogenesis

Behavioral Improvements

Improved behavior and cognitive function in preclinical *in vivo* studies

INM-901 Next Steps

Research & Development

- Dose-ranging studies
- Pre-IND meeting
- GLP studies to follow

Business Development

- Identify co-development partners and strategic investors to accelerate development

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Thank you!

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