InMed Pharmaceuticals

INM-901
Alzheimer's Disease

Acquisition Opportunity

May 2026

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 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; preparing for a pre-IND meeting with the FDA in Q3 2026 and InMed's recent merger announcement and subsequent plans to divest the INM-901 asset.

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 subsequent quarterly filings 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 EFFECTS

What is Alzheimer’s Disease?

Alzheimer's is the most common form 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 common pathological hallmarks of Alzheimer’s disease are the build-up of amyloid-beta plaques and neurofibrillary tangles. Emerging research indicates that the associated neuroinflammation is also a key factor. Lifestyle and genetics are likely contributors to disease development.

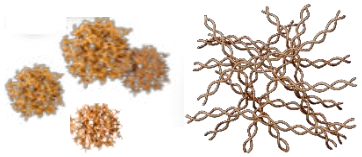
Impact

- 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)

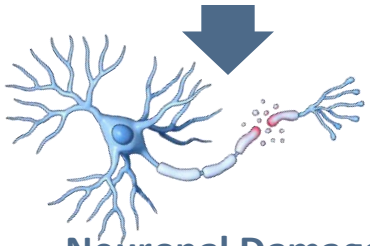
Source: Alzheimer’s Association (U.S.)

Evolving Perspective in the AD Community: The Role of Neuroinflammation in Alzheimer's Disease

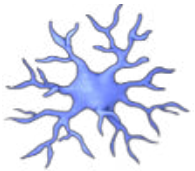
OLD VIEW



Aβ & Tau

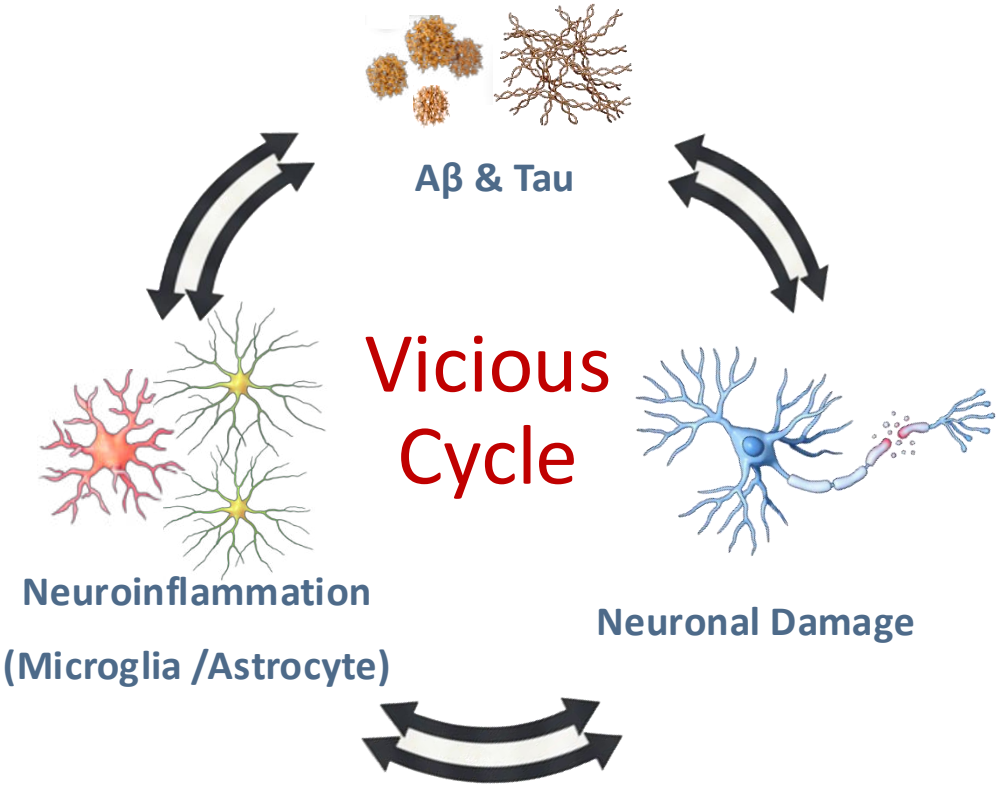


Neuronal Damage



Inflammation
(Cleanup Response)

NEW VIEW



Historical View

- Aβ (amyloid-beta) and tau considered the primary causes of AD

New Perspective

- Aβ and tau are now viewed as part of a more complex disease process
- AD recognized as multifactorial
- Many genetic risk factors for AD are genes linked to inflammation.

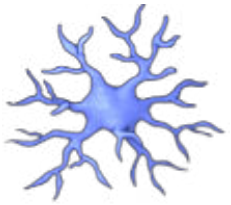
Limitations of Targeted Therapies

- Aβ- and tau-targeted treatments show modest slowing of disease progression
- No reversal of established disease

Emerging Role of Neuroinflammation

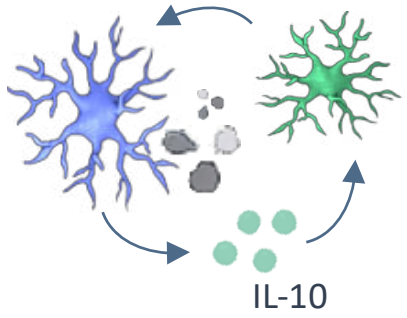
- Activation of microglia and astrocytes contributes to pathology
- Neuroinflammation is not just a "cleanup" response; it is also a contributory driver of AD progression

The Key Players in Neuroinflammation: Microglia and Astrocytes

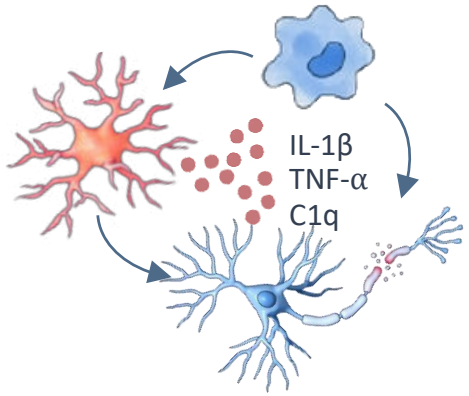


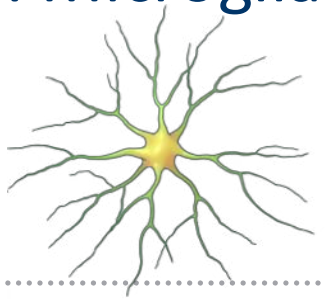
Microglia
(The Sentinels)

Protective
Responsible for neuroinflammation resolution, phagocytosis of debris, and anti-inflammatory actions via IL-10 release.



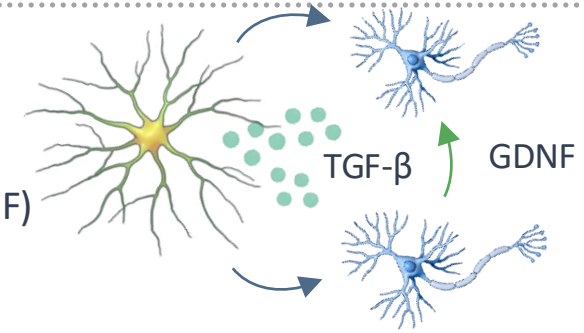
Destructive
Releases pro-inflammatory cytokines (IL-1β, TNF-α, C1q), initiating a toxic cascade and driving neurodegeneration.



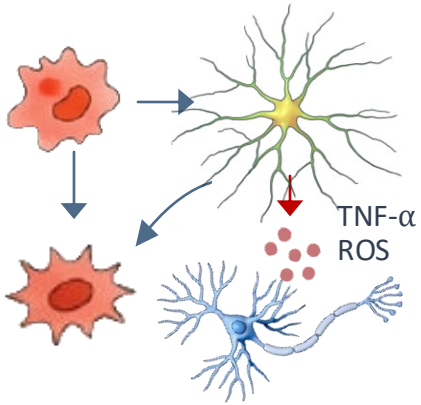


Astrocytes
(The Support System)

Protective
Releases anti-inflammatory molecules (TGF-β) and neuroprotective factors (GDNF) to support neuronal survival.



Destructive
*Induced by destructive microglia. This phenotype actively secretes neurotoxic factors (TNF-α, ROS), directly damaging neurons and amplifying inflammation.



Inhibiting/reversing the transition to the destructive states may play an important role in AD reversal.

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Role of CB1/CB2 Receptors in Targeting Microglia and Astrocytes

Role of CB2 Receptors in Neuroinflammation – Anti-inflammatory

- **Immune Regulation:** Highly expressed on microglia and astrocytes during inflammation, CB2 receptors are upregulated in response to injury or disease (e.g., MS, Alzheimer's)
- **Microglial and Astrocyte Modulation:** Activation of CB2 receptors promotes their transition toward a reparative, anti-inflammatory (Protective) state from the reactive (Destructive) state.
- **Immune Cell Migration:** CB2 receptor activation, via microglia/astrocyte cross-talk, restricts the movement of peripheral immune cells (like T-lymphocytes) into the CNS, limiting further inflammation.

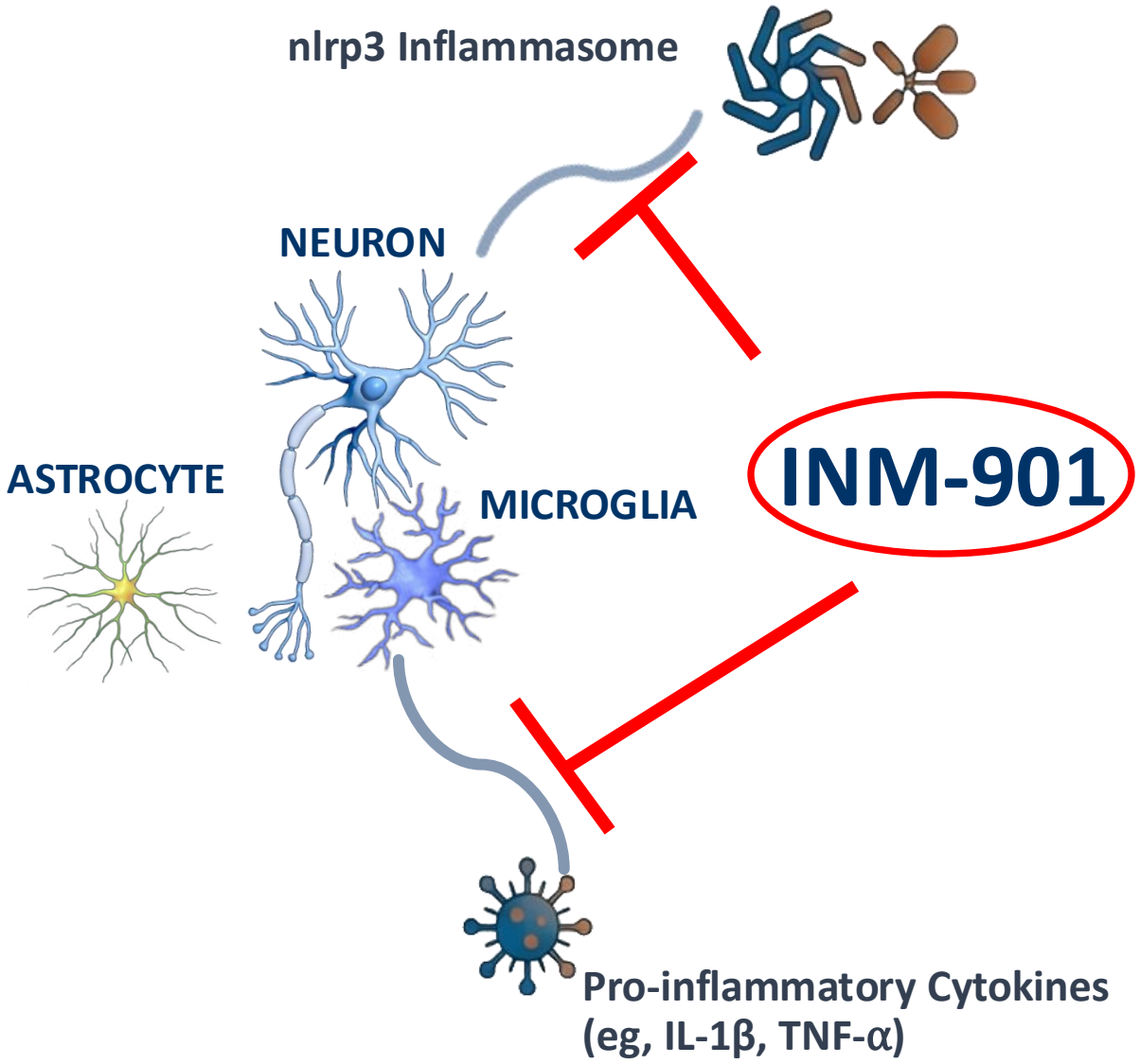
Role of CB1 Receptors in Neuroinflammation – Neuroprotection

- **Neuroprotection:** CB1 receptors help protect neurons against excitotoxicity-induced death during neuroinflammation.
- **Interaction with CB2:** CB1 receptors work alongside CB2 to mitigate inflammation and may form heteromers to modulate intracellular signaling, such as MAPK/ERK and PI3K/Akt pathways, that control cell survival and inflammation.

While INM-901 is primarily a CB2 agonist, both CB1 and CB2 receptors modulate neuroinflammation via distinct but complementary pathways.

Together, they regulate immune responses, reduce oxidative stress and, when activated, may mitigate neurodegenerative disease progression.

INM-901 Mechanism of Action on Neuroinflammation



Rationale:

Since inflammation is a core driver of AD, modulating inflammatory immune response is a promising therapeutic strategy.

INM-901 data, via the CB2 pathway, demonstrates down-regulation of both inflammasome activation and pro-inflammatory cytokines.

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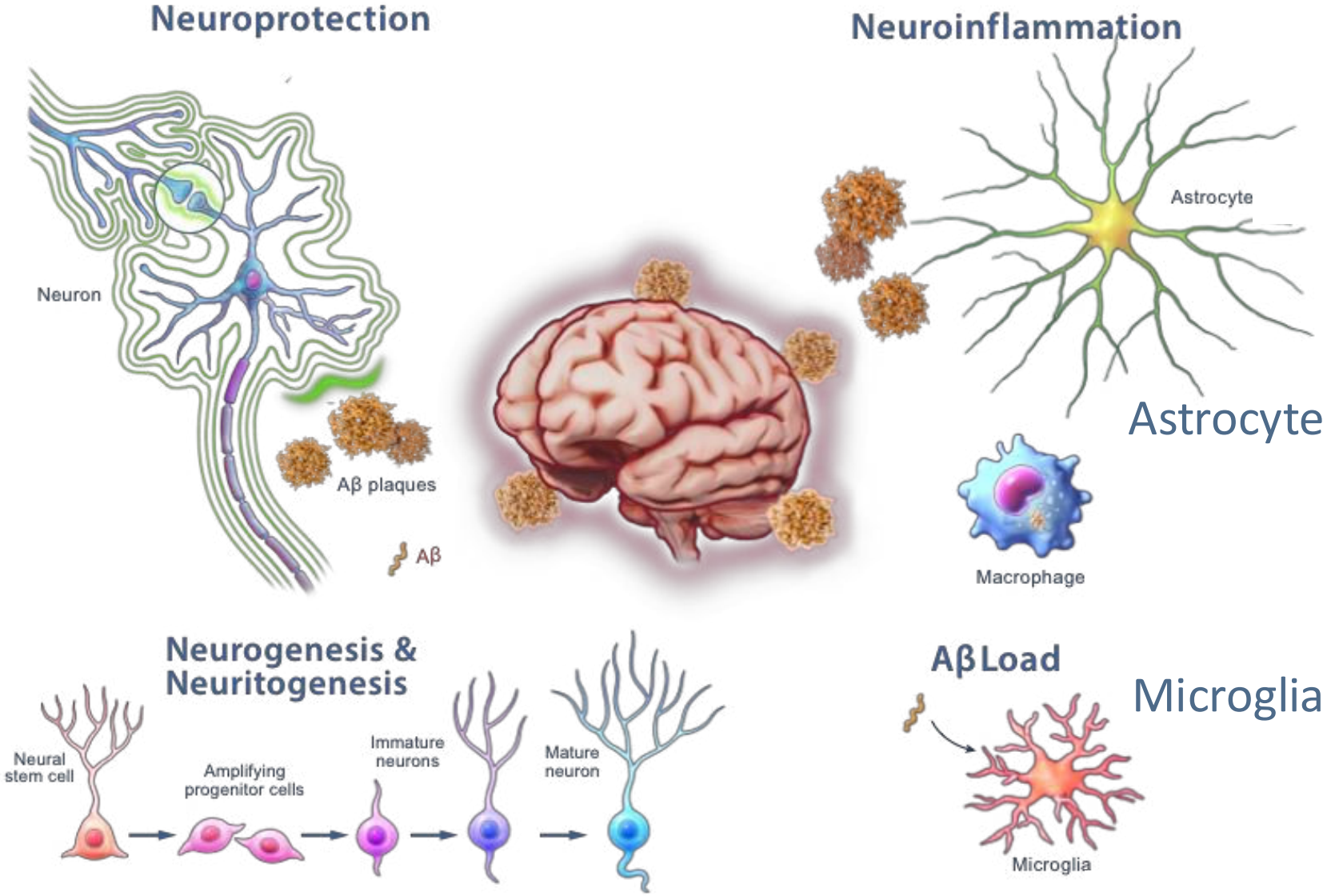
INM-901 Demonstrates Positive Impacts on AD Pathologies



Multiple pro-inflammatory and neurodegenerative proteins are elevated in AD; INM-901 demonstrated meaningful decreases in these proteins.

AD	INM-901
GFAP	↓
TNF-α	↓
IFNγ	↓
IL-1β	↓
IL-6	↓
CB2	↓
NfL	↓

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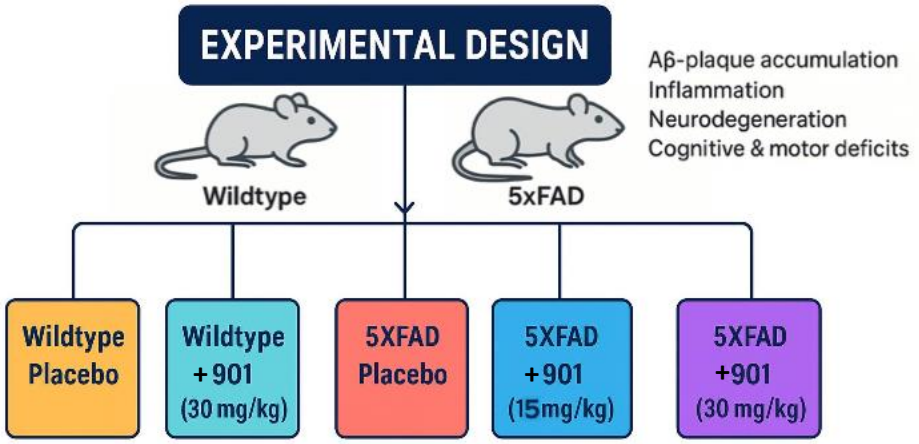
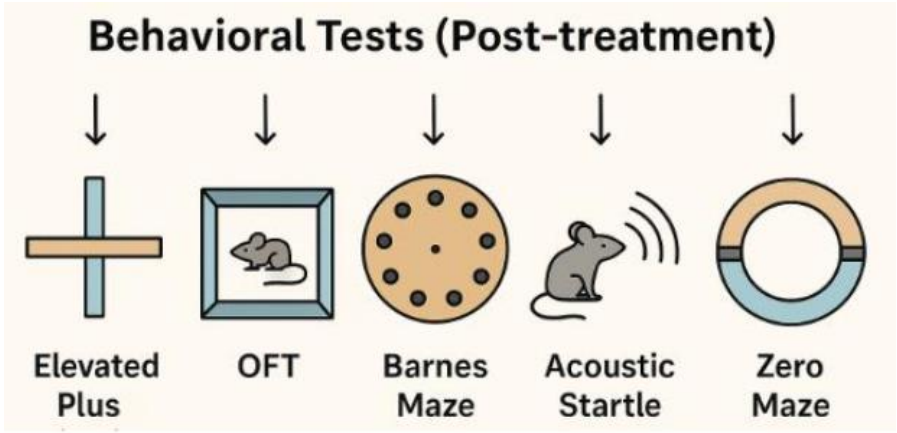
INM-901 Proof of Concept in Alzheimer's Using 5xFAD Mouse Model

LONG TREATMENT DURATION AND ADVANCED DISEASE STAGE

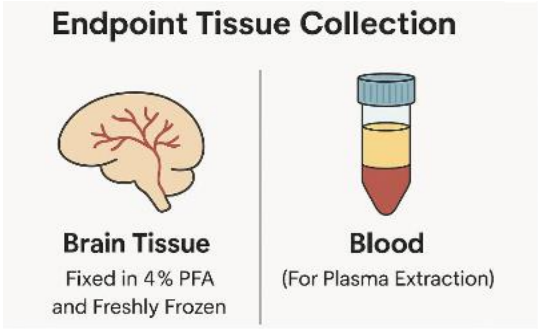


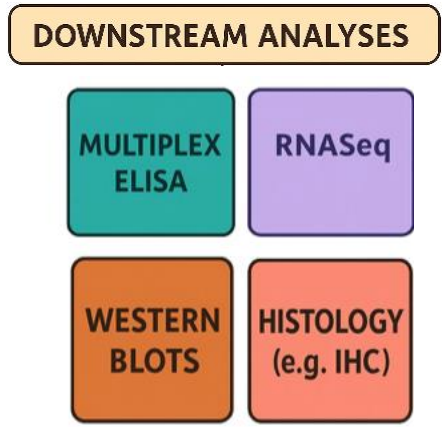
2-month-old male mice were injected twice-weekly for 7 months



9-month-old male mice

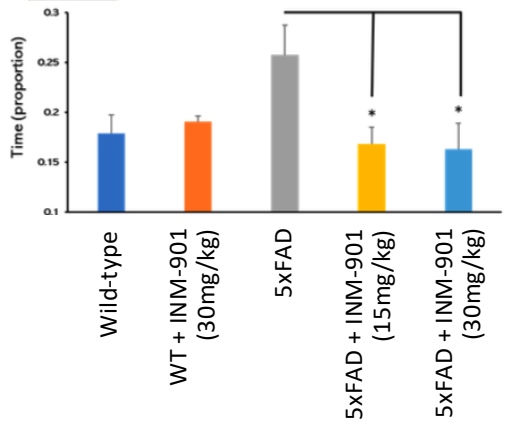
This study evaluates INM-901 in the 5xFAD (amyloidosis) mouse model, using a long treatment duration (7 months) and a more advanced disease stage to validate and expand upon previous short-term model findings.



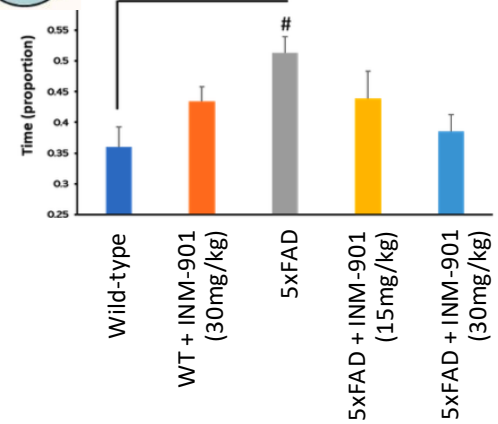


INM-901 Demonstrates Positive Trends Across all Tested Parameters in Cognitive Function, Anxiety-Related Behavior and Sensory Responsiveness

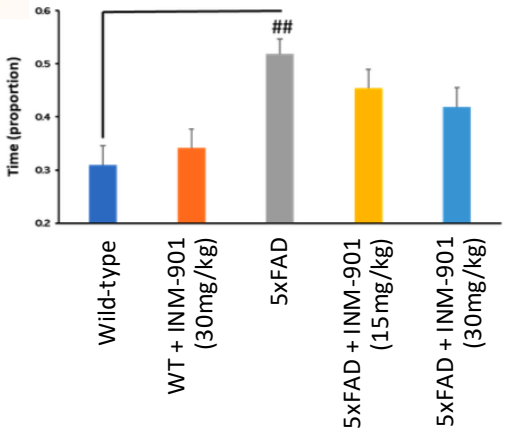
Open Field Test (Centre)



Zero Maze (Open Section)

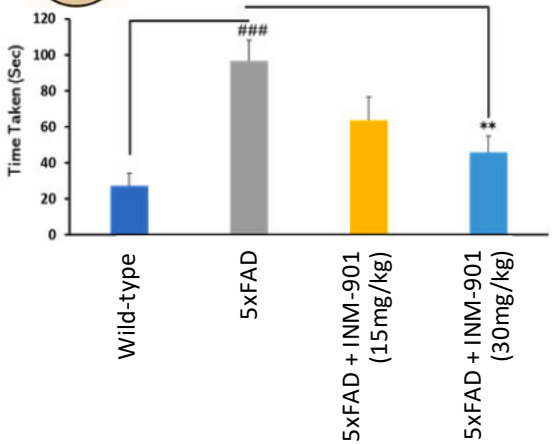


Elevated Plus Maze (Open Arm)



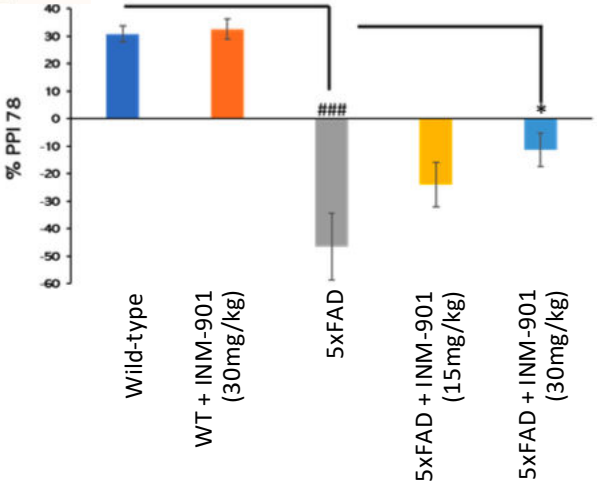
5xFAD mice spent more time in the center zone or open arms, suggesting reduced anxiety-like behavior. **INM-901 treatment restored typical anxiety-like behavior similar to wild-type mice.**

Barnes Test



5xFAD mice showed impaired spatial 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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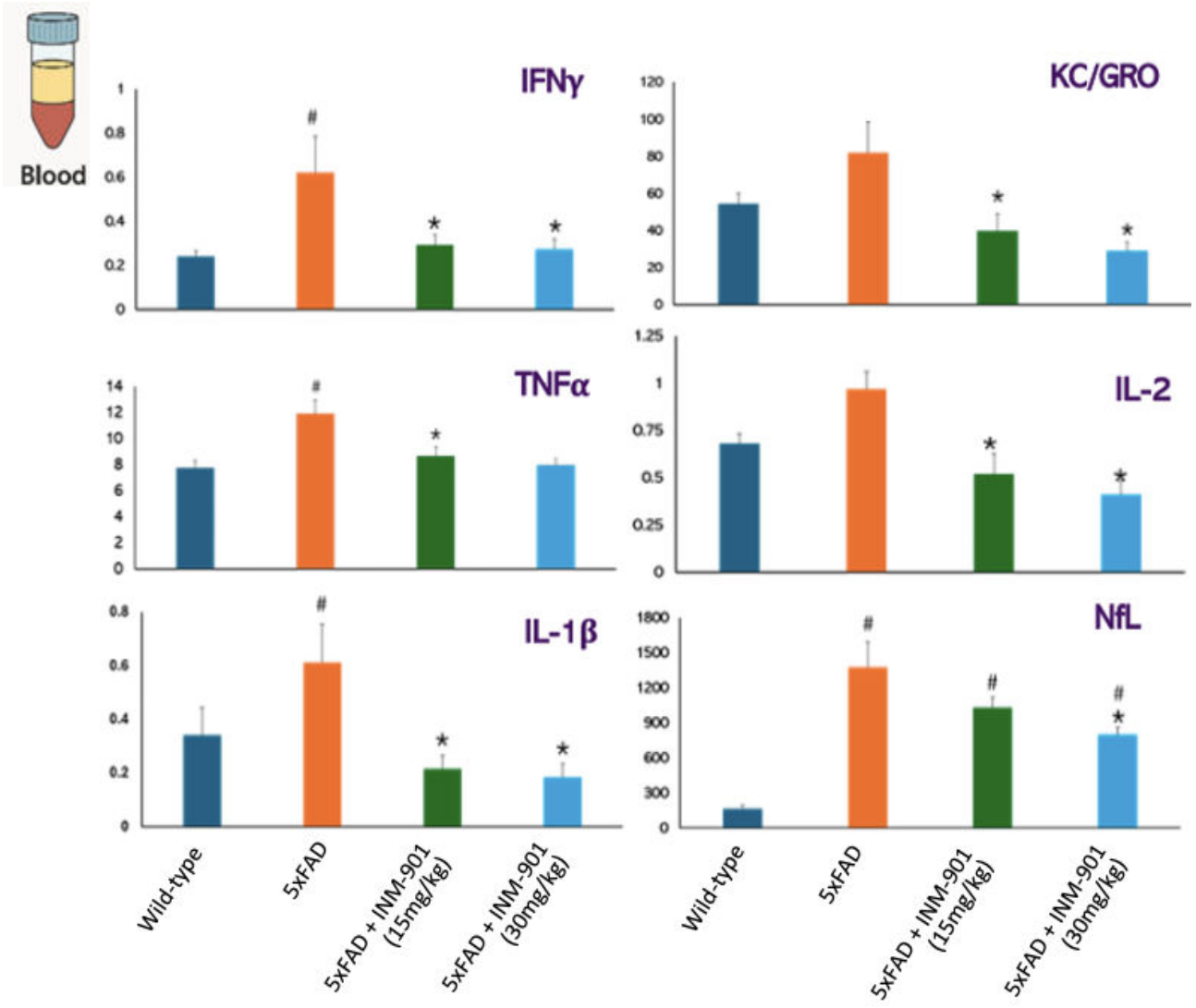
INM-901 Demonstrates Strong Neuroinflammatory Modulation in 5xFAD Mouse Model



Plasma Inflammatory Biomarkers

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, demonstrating **a dose-dependent therapeutic effect of INM-901**.

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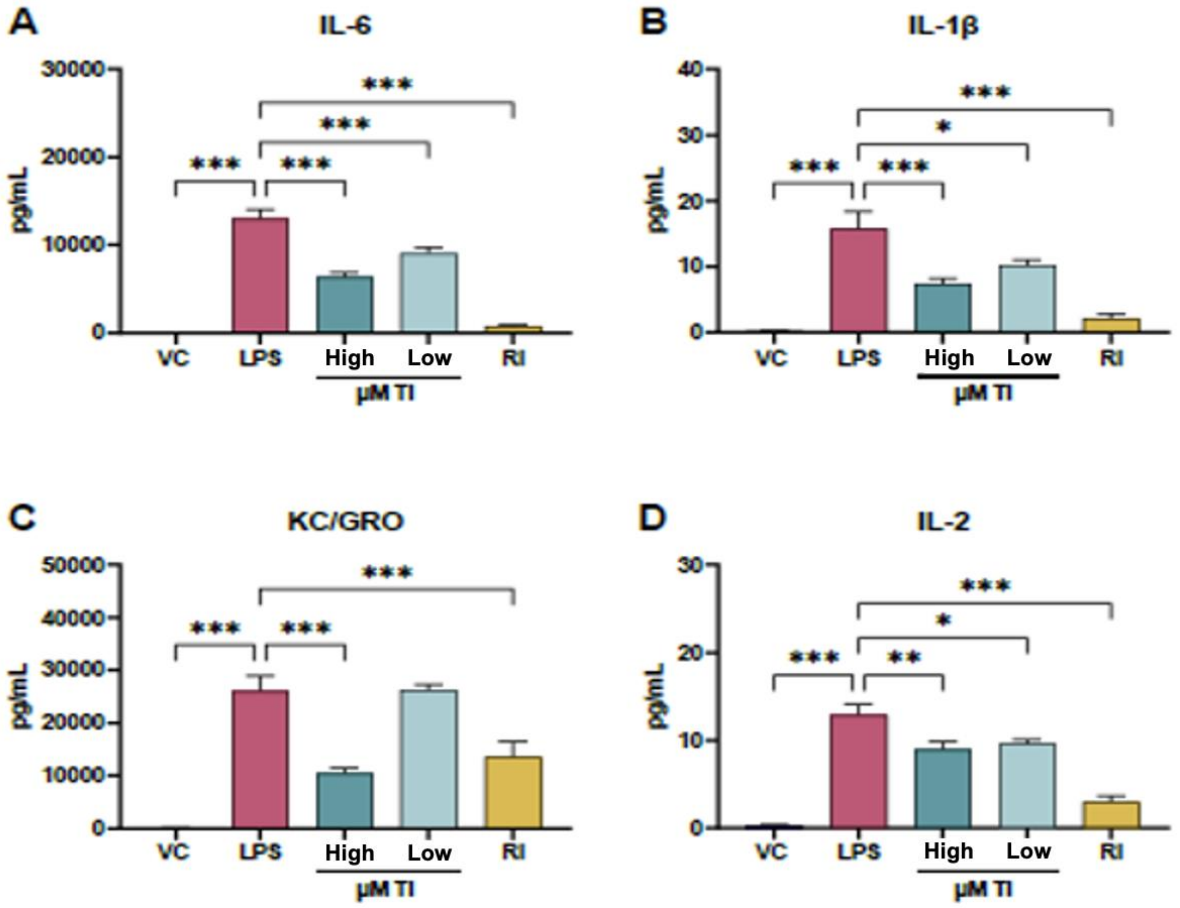
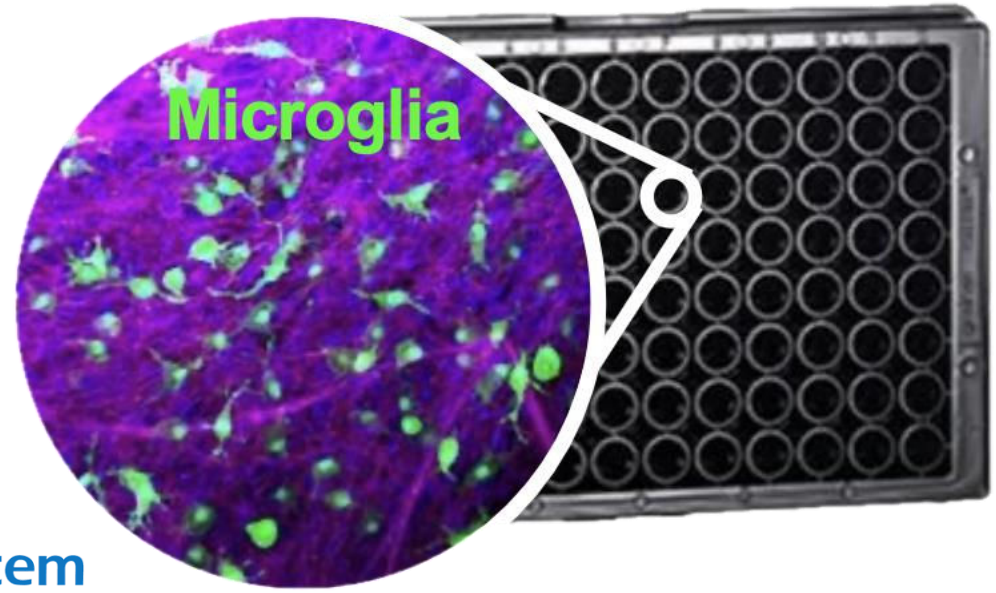
INM-901 Reduces Inflammatory Markers in the Brain Tissues of *Ex Vivo* LPS Model

- Pro-inflammatory markers increased following LPS exposure
- Cytokines/Chemokine in the INM-901 treated groups showed a reduction in inflammation compared to the LPS group
- INM-901 can exert a direct anti-inflammatory effect independent of the amyloid or tau pathology



INM-901 Anti-inflammatory Effects in 3D Human Brain Organoid Model

HUMAN ORGANOID DATA CONSISTENT WITH *IN VIVO* AND *EX VIVO* RESULTS



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3D Human Brain Organoid Model

- **Closest approximations to human brain tissues**
- **Complex Cellular Environment:** neurons, astrocytes, vascular cells and microglia
- **Two neuroinflammation models:** neuroinflammation induced with LPS and IFN-γ and proprietary Alzheimer's specific neuroinflammation model

INM-901 Effects in Brain Organoid Model

- Significant reduction in neuroinflammation in Stem Pharm's LPS-induced and Alzheimer's disease models
- A dose-dependent reduction of key pro-inflammatory markers such as IL-6 and IL-8
- Effects align with prior findings from an *in vivo* Alzheimer's model and an *ex vivo* LPS-induced neuroinflammation model
- Provides supportive evidence of mechanistic translation from animal models to human tissue systems

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Summary: INM-901 Impact on Alzheimer's Pathology

Robust Bioavailability

Oral formulation achieved anticipated therapeutic levels of systemic exposure

Behavioral Improvements

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

Anti-Inflammatory Action – 5xFAD

Significantly reduced inflammatory biomarkers IFNγ, TNF-α, IL-1β, KC-GRO, IL-2 & NfL in 5xFAD mouse model

Anti-Inflammatory Action – LPS-induced

Significantly reduced inflammasome marker activation of NLRP3 and IL-1β, key contributors to neurodegeneration independent of amyloid-beta or tau pathology

INM-901

Neuronal Regeneration

Promotes neurite outgrowth, indicating an ability to enhance neuronal connectivity and function in *in vitro* assays

Impact on human Neuroinflammation

Reduced IL-6 and IL-8 in two different human brain organoid models

Molecular Validation

Additional mRNA data (not shown) provide supportive evidence for improvements in cognition, memory and neurogenesis

Neuroprotection

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

Pipeline Next Steps

Research & Development

InMed activities:

- Pre-IND meeting with the U.S. Food and Drug Administration planned for summer 2026
- Top-line clinical plan developed

On hold pending acquisition:

- Completion of remaining IND-enabling pharmacology and toxicology studies
- Finalization of CMC and manufacturing activities to support IND submission

Business Development

- Following the recent merger announcement, InMed is planning to divest INM-901 asset.
- InMed is open to all potential divestiture structures related to INM-901.

Additional Information available under CDA

- Neurological assessment in dogs
- No negative impact on general attitude, behavior, motor function.
- PK studies in mice, rats and dog
- Dose Ranging Studies in rats and dogs
- DMPK studies
- CMC scale-up activities
- Regulatory planning activities towards IND

Key Patent Summary

All key patents in place to support the INM-901 program

Subject Matter	Scope	Ownership/ Origin	Filing Status / Filing Date	Patent Reference Number – Patent Nos.	Earliest Potential/ Patent Expiry[2]	Jurisdictions - Status
Cannabinoid analogs and methods for their preparation	Composition, Manufacturing Process (INM-089, INM-901)	Assigned to InMed(CA) from BM	PCT Application filed 10/31/2019	WO2020/092823 AU 2019371379 CN ZL201980087003.4 IL 282822 IN 570335 JP 7817830 MX 417531 US (patent no.TBD)	2039	Granted: AU, CN, IN, JP, MX Pending: AU, CA, CN, EP, IL (allowed), IL, IN, JP, US, US (allowed)
Compositions and methods for treating neuronal disorders with cannabinoids	Use (INM-901 fence)	InMed(CA)	PCT Application filed 10/21/2022	WO 2022/082313	2041	Pending: AU, CA, CN, EP, IL, JP, MX, US
Cannabinoids Compounds and Methods for Treatment of Neuronal Disorders	Composition, Use (INM-901)	InMed(CA)	PCT Application filed 10/23/2024	WO 2025/090587	2043	Pending: PCT (National stage application filings due April 2026)

Current Pharmaceutical R&D Team

Technical and scientific expertise may be available post-acquisition

Michael Woudenberg, PEng
Chief Operating Officer

20+ years of engineering, scale-up and GMP manufacturing experience: Phyton Biotech, Arbutus Biopharma, 3M and Cardiome Pharma

Eric Hsu, PhD
SVP, Preclinical R&D

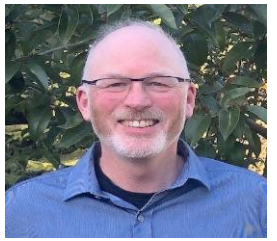
20+ years of scientific leadership experience in gene transfer technologies, formulation and process development: enGene Inc.

Sapna Padania
Scientist III

Scientist with over a decade of experience in pharma and biotechnology in management of early discovery and development projects

Charles Marlowe, PhD
VP, Chemistry

30+ years R&D discovery-to-FDA approval: Millennium Pharma, COR, Chiron, Takeda, Dow Chemical, Exelixis.



Jim Kealy, PhD
VP, Synthetic Biology

25+ years in synthetic biology and tech development at Amyris, Intrexon and Kosan Biosciences.

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Thank you!

Forward Inquiries to

Colin Clancy

Cclancy@inmedpharma.com

+1-778-855-2408

Nasdaq :INM

www.inmedpharma.com