InMed Pharmaceuticals

Corporate Presentation

April 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; INM-089 showing promise in preserving retinal function in the *in vivo* AMD disease model; INM-089 being a preferential signaling ligand for CB1 and CB2; showing improved photoreceptor function, RPE integrity, thickness of outer nuclear layer; ability to proactively protect the retinal ganglion cells; deliverable through preferred IVT administration; having high yield scalable production methods; INM-755 cream shows enhanced anti-itch activity versus the control cream alone; INM-755 cream demonstrated a favorable safety and tolerability profile; INM-755 cream demonstrated sufficient clinically important anti-itch activity to warrant further development; InMed will now pursue strategic partnership opportunities for INM-755 in EB and other itch related diseases; the advancement of chemistry, manufacturing, and controls (CMC) activities; the planning of GLP-enabling studies and the preparation of an IND submission the further development; planning for a pre-IND meeting in Q3 2026; engaging regulatory / clinical experts to map out topline clinical design for first in human clinical trials for the INM-901; targeting submission of an IND and initiation of a Phase 1 clinical trial in 2027; potential efficacy, and marketability of INM-089 for dry age-related macular degeneration; preparing for a pre-IND meeting with the FDA in Q4 2026 for INM-089; and, identifying and executing on strategic initiatives to build shareholder value.

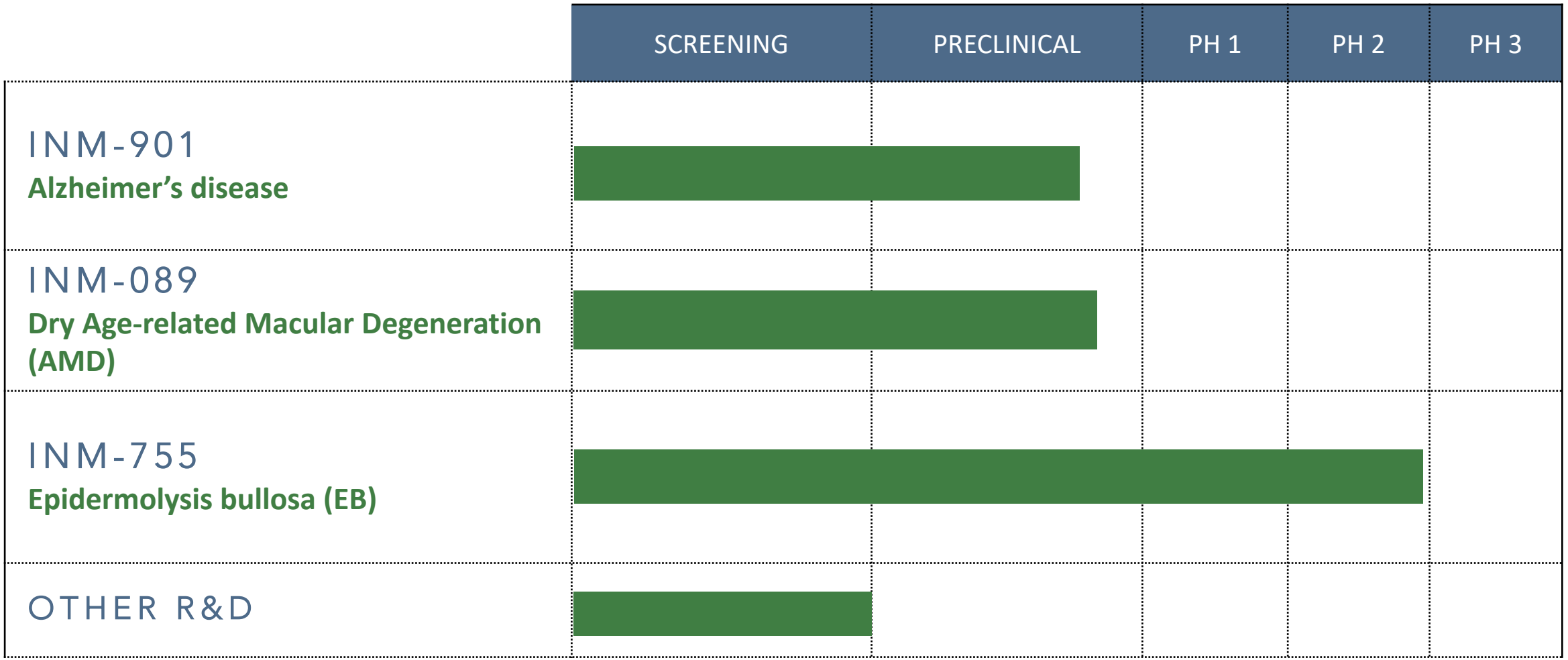
These statements are based on management's current expectations and beliefs and are subject to a number of risks, uncertainties and other factors that could cause actual results to differ materially from those described in the forward-looking statements. These risks and uncertainties include, among others: 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.

Executive Summary & Highlights

InMed's lead drug candidate, INM-901, targets neuroinflammation in Alzheimer's. INM-901 is a proprietary, orally bioavailable, disease-modifying therapeutic that is a preferential CB1/CB2 signaling agonist that can cross the blood-brain barrier.

- Additional R&D pipeline drug candidates:
  - **INM-089 for Ocular Disease** – Functional and pathological improvements for dry AMD
  - **INM-755 for Dermatology** - Phase 2 completed in EB, currently seeking partnerships
- Exceptional leadership team covering all areas of drug discovery
- An optimal capital markets structure and clean balance sheet.

Pharmaceutical Pipeline: Small Molecules Targeting CB1/CB2



Alzheimer’s Disease – A Major Medical & Societal Burden

CURRENT TREATMENT OPTIONS DO NOT REVERSE 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 the associated neuroinflammation is also a factor. Lifestyle and genetics are likely contributors to disease development.

Impact

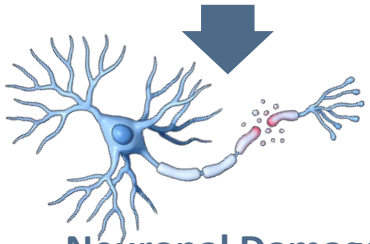
- 7.2M Americans affected
- 1 in 9 people age 65+ (11%)
- 1 in 5 women, 1 in 10 men
- 6th leading cause of death for 65+
- Alzheimer’s accounts for 60-80% of dementia cases
- U.S. annual financial impact $384B in 2025
  (Alzheimer’s and other dementia)

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

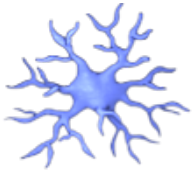
Evolving Perspective in the AD Community: Neuroinflammation Response in Alzheimer's Disease

OLD VIEW

Aβ & Tau

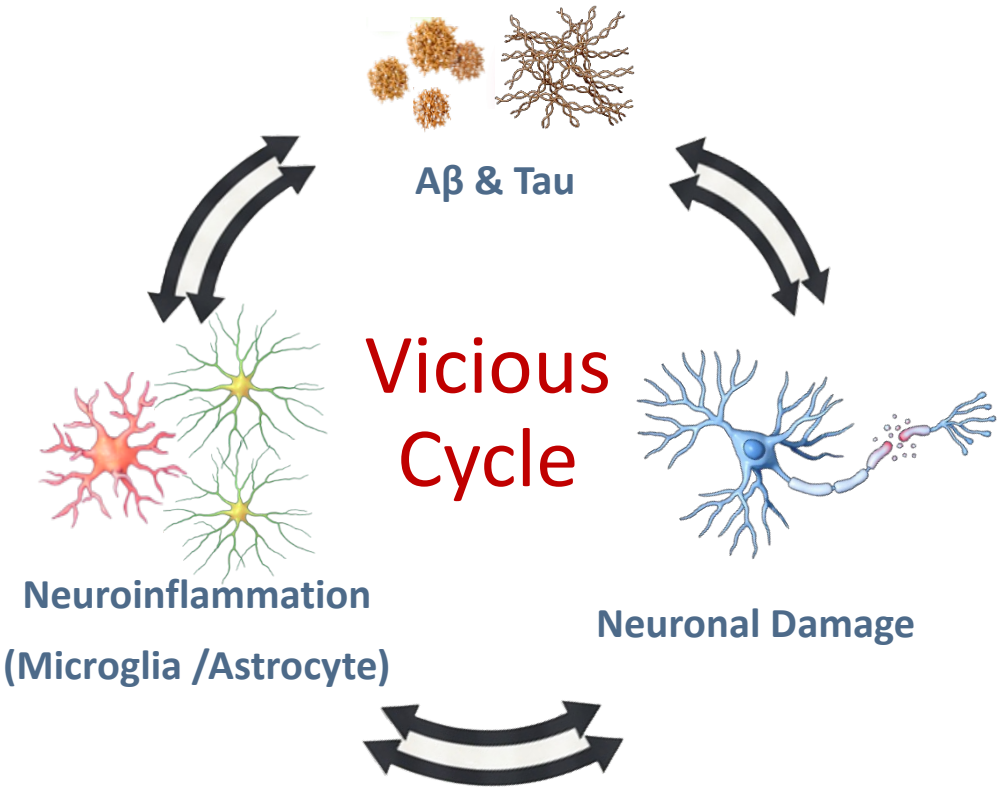


Neuronal Damage



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

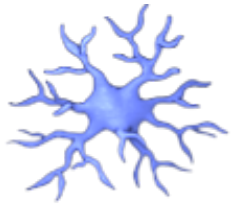
Limitations of Targeted Therapies

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

Emerging Role of Neuroinflammation

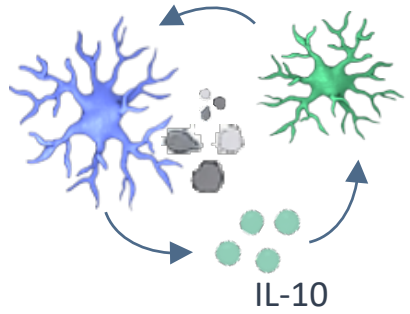
- Activation of microglia and astrocytes contributes to pathology
- Neuroinflammation is not just a "cleanup" response; increasingly recognized as a driver of AD progression

The Key Players in Neuroinflammation: Microglia and Astrocytes

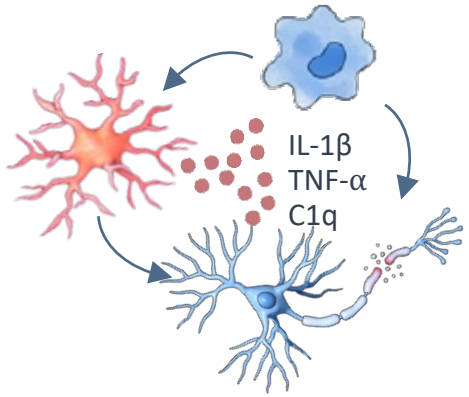


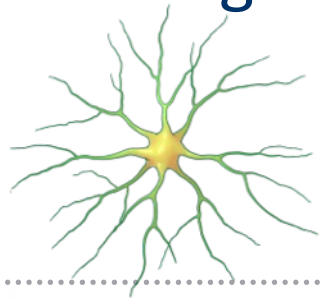
Microglia
(The Sentinels)

Protective (M2)
Responsible for neuroinflammation resolution, phagocytosis of debris, and anti-inflammatory actions via IL-10 release.



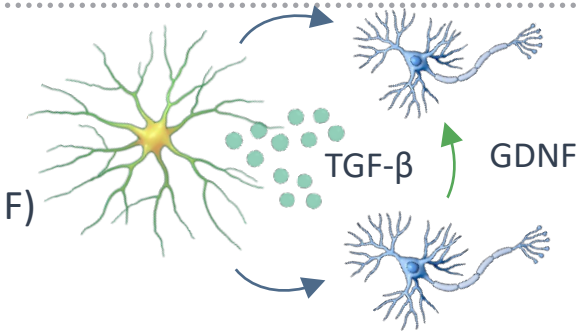
Destructive (M1)
Releases pro-inflammatory cytokines (IL-1β, TNF-α, C1q), initiating a toxic cascade and driving neurodegeneration.



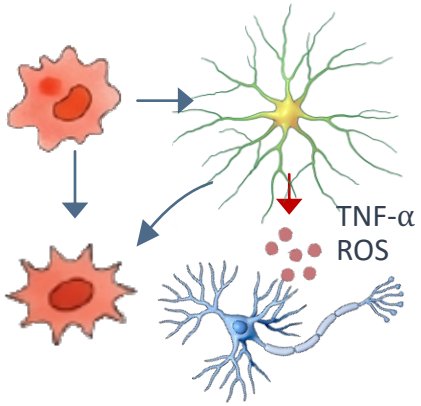


Astrocytes
(The Support System)

Protective (A2)
Releases anti-inflammatory molecules (TGF-β) and neuroprotective factors (GDNF) to support neuronal survival.



Destructive (A1)
Induced by M1 microglia, this phenotype actively secretes neurotoxic factors (TNF-α, ROS), directly damaging neurons and amplifying inflammation.



Inhibiting/reversing the transition to the destructive M1/A1 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 (M2 and A2) state from the reactive (M1 and A1) 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.

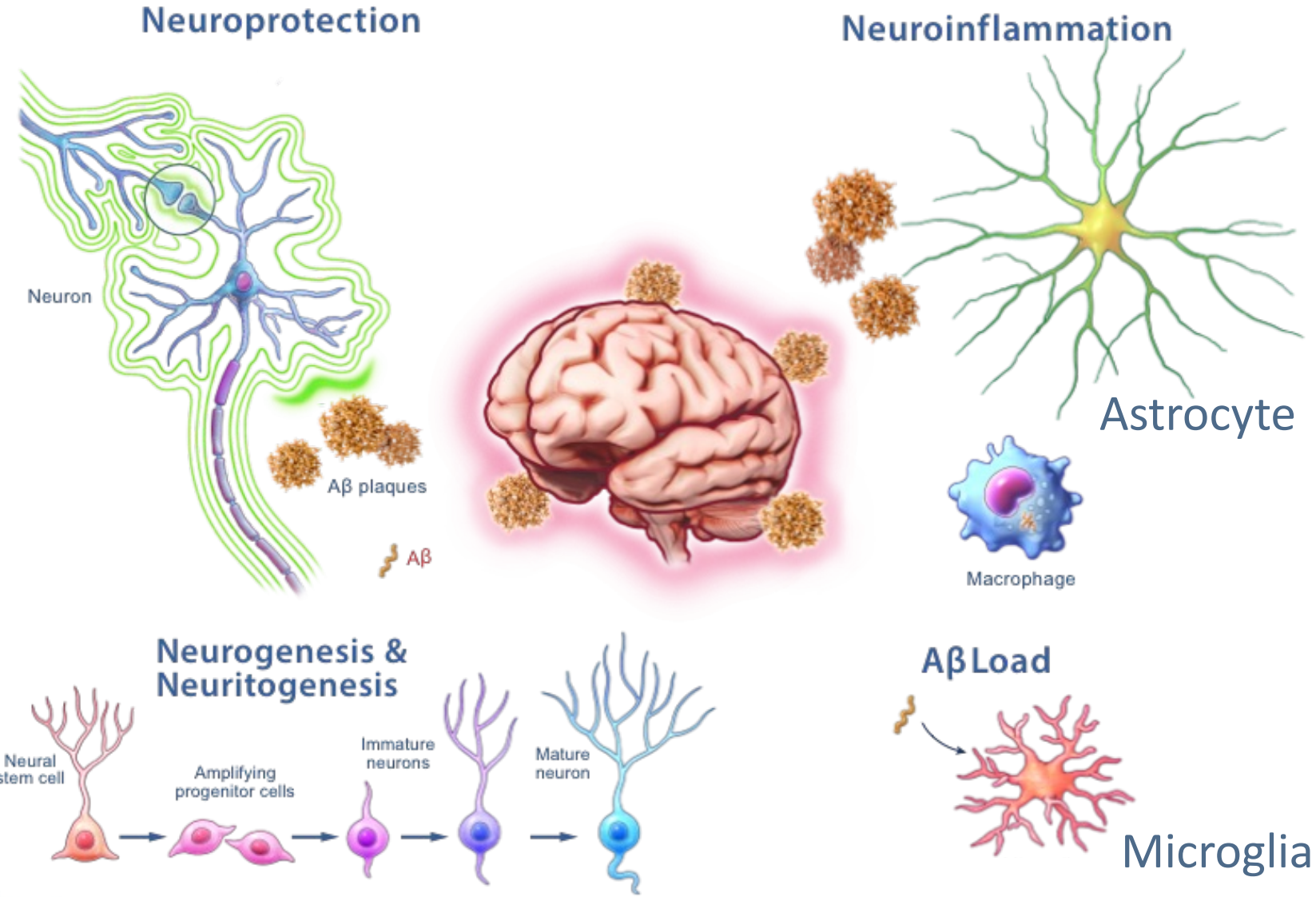
Role of CB1 Receptors in Neuroinflammation – Neuroprotection

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

CB1 and CB2 receptors modulate neuroinflammation through distinct but complementary roles.

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

INM-901 Demonstrates Positive Impacts on AD Pathologies



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

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

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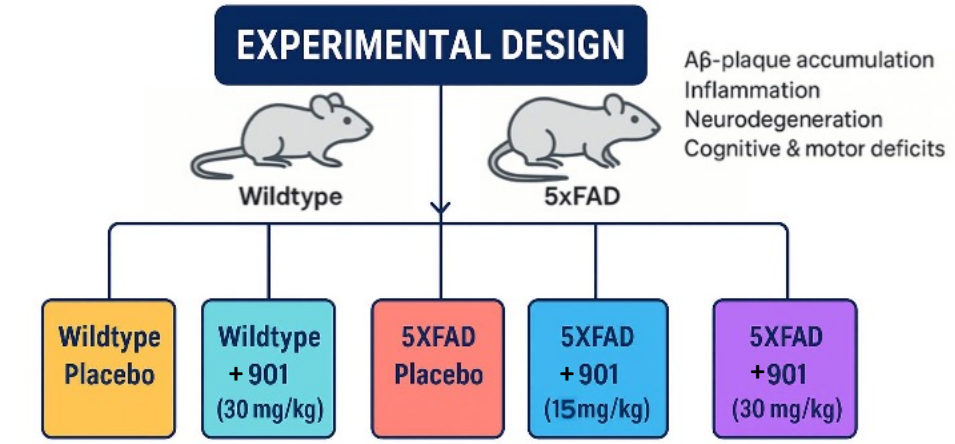
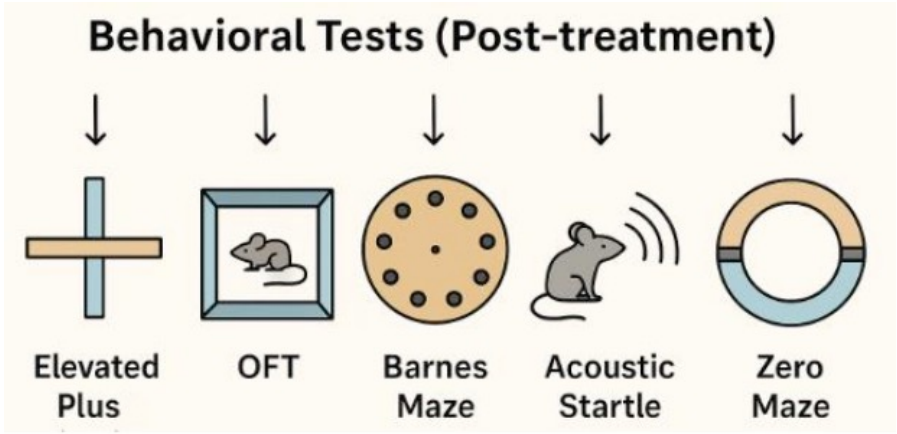
INM-901 Proof of Concept in Alzheimer's Using 5xFAD Mouse Model

LONG TREATMENT DURATION AND ADVANCED DISEASE STAGE

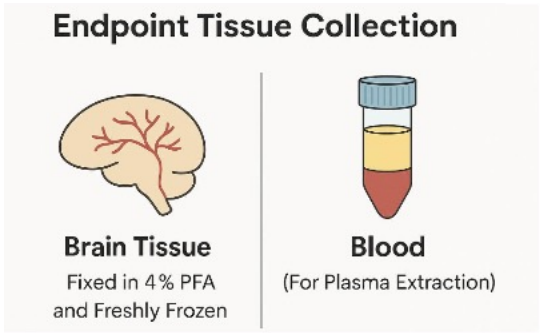


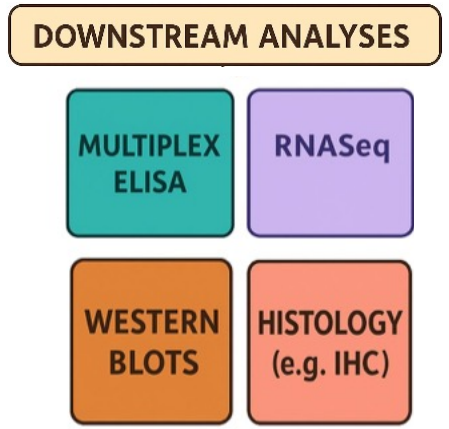
2-month-old male mice were injected twice-weekly for 7 months



9 months old male mice

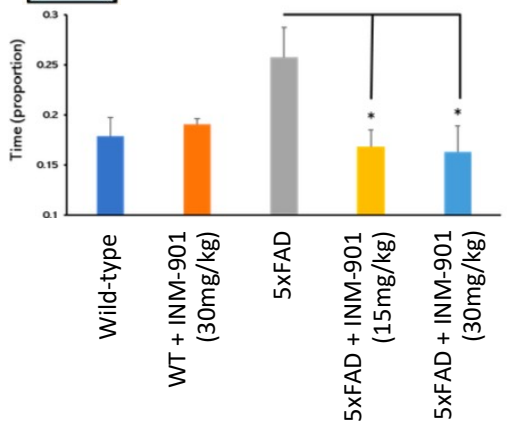
This study evaluates INM-901 in the 5xFAD (amyloidosis) mouse model, using a long treatment duration (7 months) and a more advanced disease stage to validate and expand upon previous short-term model findings.



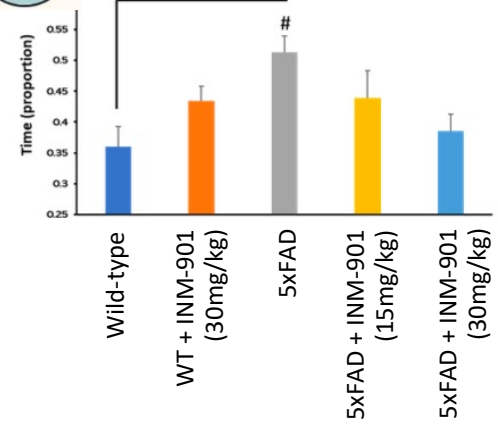


INM-901 Demonstrates Positive Trends Across all Tested Parameters in Cognitive Function, Anxiety-Related Behavior and Sensory Responsiveness

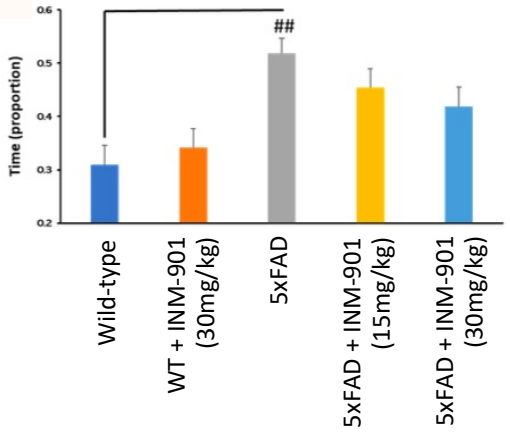
Open Field Test (Centre)



Zero Maze (Open Section)

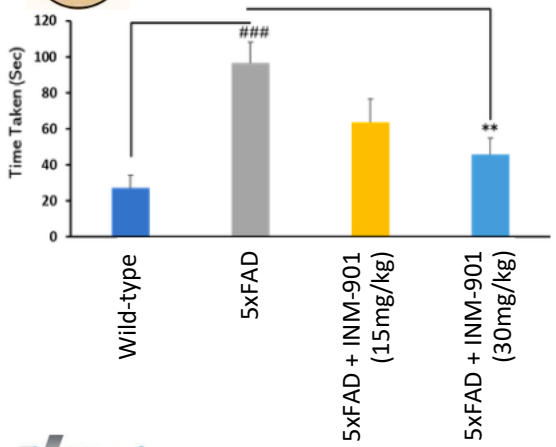


Elevated Plus Maze (Open Arm)



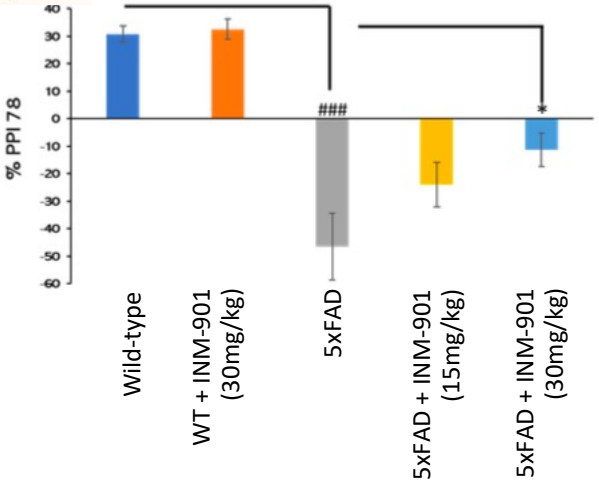
5xFAD mice spent more time in the center zone or open arms, suggesting reduced anxiety-like behavior. **INM-901 treatment restored typical anxiety-like behavior similar to wild-type mice.**

Barnes Test



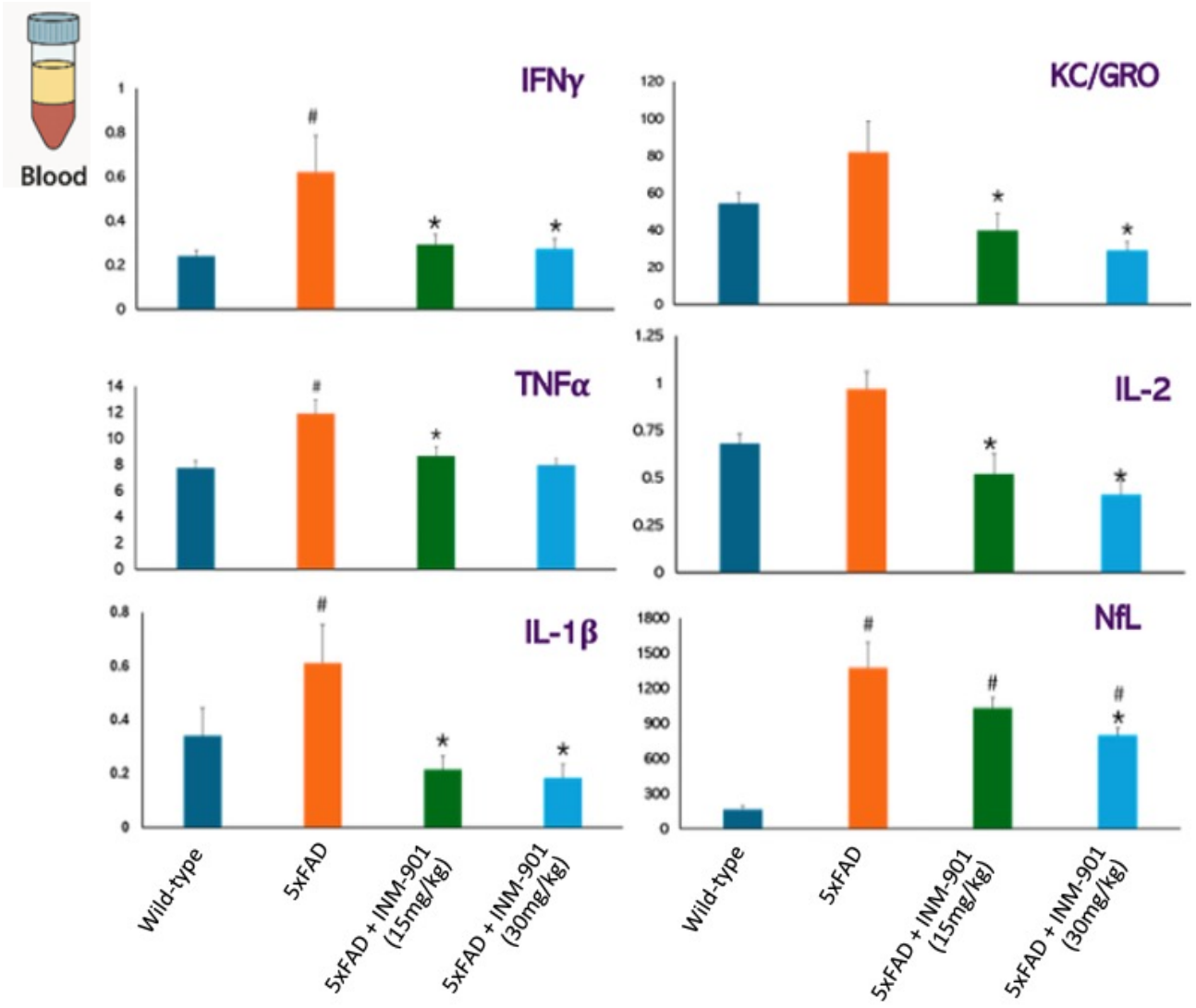
5xFAD mice showed impaired special learning and memory, while **INM-901 treatment improved performance.**

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.**

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.

Summary: INM-901 Impact on 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 mouse model

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

Robust Bioavailability
Oral formulation achieved anticipated therapeutic levels of systemic exposure

Anti-Inflammatory Action – LPS-induced
Significantly reduced inflammasome marker activation of NLRP3 and IL-1β, key contributors to neurodegeneration

Neuronal Regeneration
Promotes neurite outgrowth, indicating an ability to enhance neuronal connectivity and function

Direct Impact on Neuroinflammation
Demonstrated anti-inflammatory effects independent of amyloid-beta or tau pathology

Molecular Validation
mRNA data supports observed improvements in cognition, memory and neurogenesis

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

Impact of Dry Age-related Macular Degeneration (AMD)

LEADING CAUSE OF VISION LOSS

What is AMD?

AMD is an eye disease that can blur your central vision, eventually leading to loss of vision. It happens when aging causes damage to the macula, the part of the eye that controls sharp, straight-ahead vision.

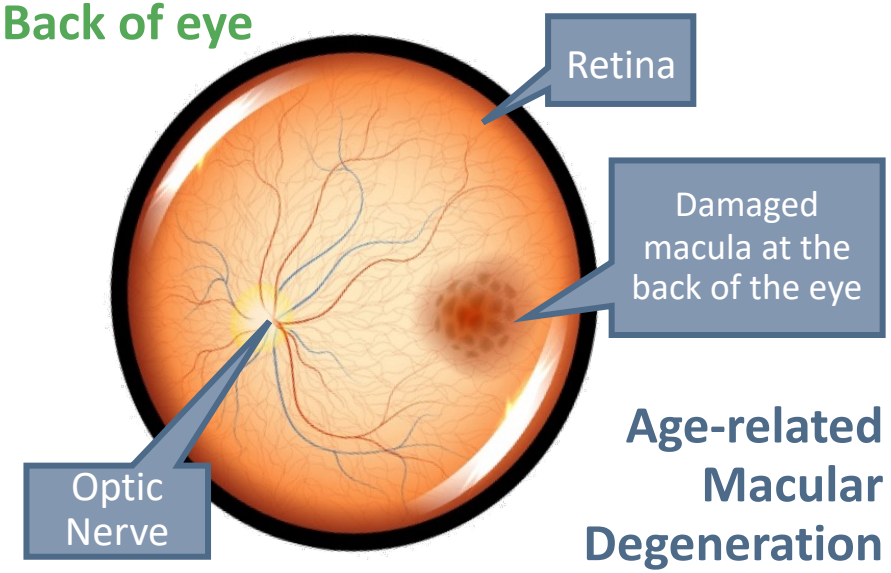
AMD Opportunity

- Affects 19.8M Americans aged 40+
- 12.6% of the U.S. population
- ~200M people worldwide
- Dry AMD = ~80% of cases
- Leading cause of vision loss for aged 65+

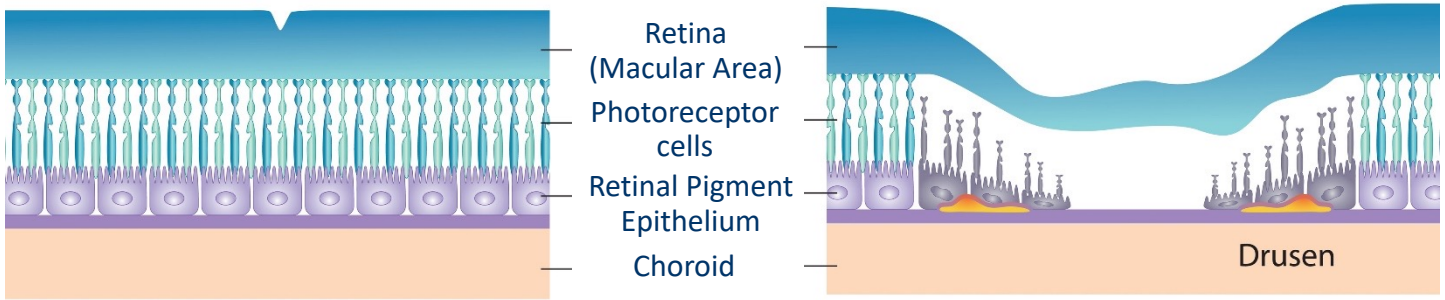
Sources: American Academy of Ophthalmology, U.S. Centers for Disease Control & Prevention, 2019

AMD Occurs When the Macula is Damaged

Back of eye



Age-related Macular Degeneration



Normal Retina

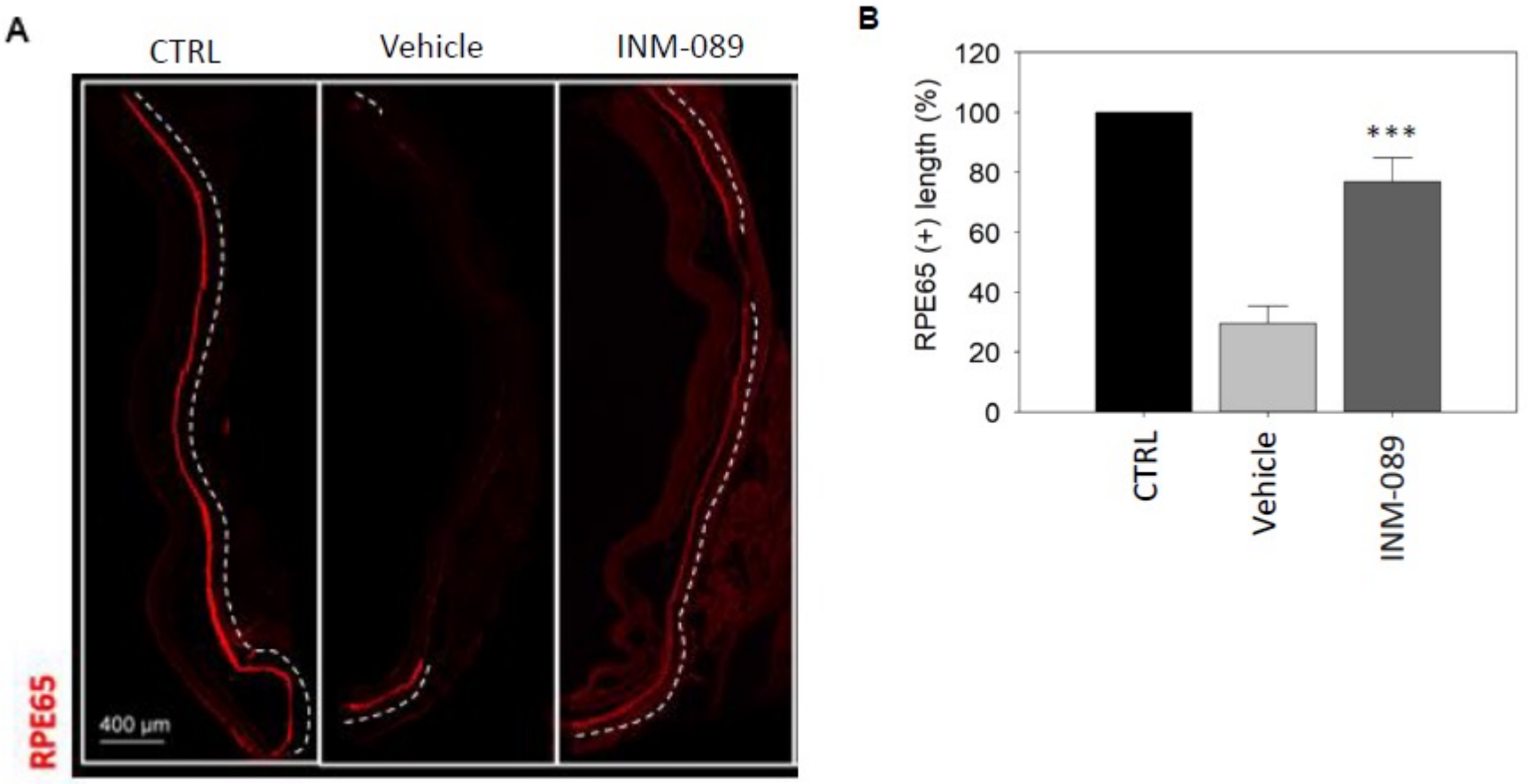
Healthy Photoreceptor cells and Retinal Pigment Epithelium (RPE)

DRY AMD

Photoreceptor cells and RPE are damaged and lost by inflammation

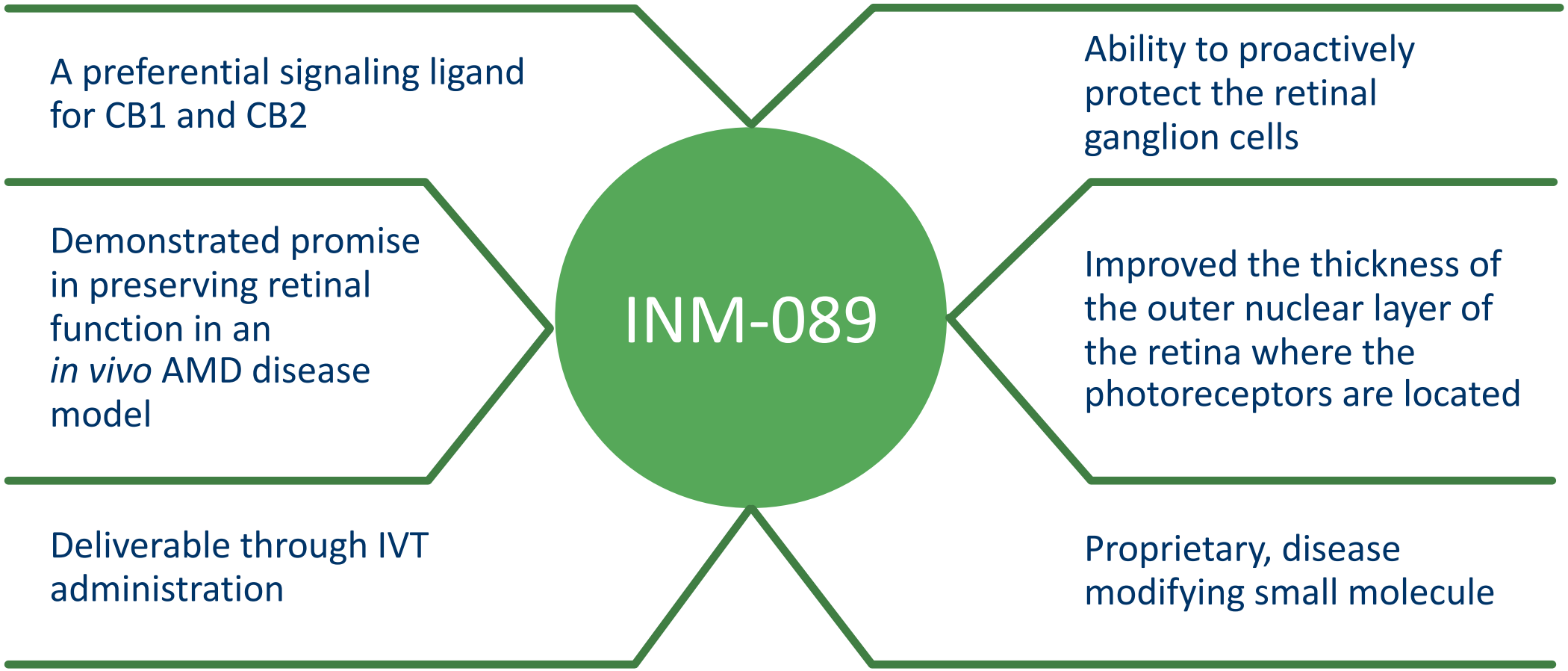
Dry AMD is the most common form of AMD. In the advanced stages of dry AMD, called Geographic Atrophy ("GA"), the retina has atrophied and the macula has wasted away, leading to the loss of central vision.

INM-089 Improved Retinal Pigment Epithelium Integrity



RPE65 analysis. **(A)** Retinal cryosections were immunostained with anti-RPE65 (red). The cryosections crossing the optic nerve were selected for the analysis and the integrity of the RPE was measured at the dorsal retina. The white segmented lines delineate the intact RPE. **(B)** RPE65 (+) length was expressed as mean ± SE. Statistical analysis was Student's *t*-test between vehicle and INM-089 [3uM] (n=7/group). ***p<0,001 *versus* Vehicle.

INM-089: A Differentiated Approach to Treating Dry AMD



INM-755 Cannabinol (CBN) Cream: Phase 2 Results in Itch

Conducted in Epidermolysis bullosa patients – a severe genetic dermatological disease with chronic, severe itch as a primary symptom.

Key Results:

- A positive indication of enhanced anti-itch activity for INM-755 cream versus the control cream alone.
- INM-755 CBN cream demonstrated a favorable safety and tolerability profile.
- Results for non-wound itch were not statistically significant in favor of INM-755 CBN cream due, in part, to the clinically important anti-itch effect of the underlying control cream.

Non-Wound Itch: Data breakdown

Of the 18 participants assessed, chronic itch improved by a clinically meaningful amount in **12 patients (66.7%),** of whom:

- **6 patients (33.3%)** had the same level of itch improvement with INM-755 cream as with control cream;
- **5 patients (27.8%)** treated with INM-755 showed meaningful anti-itch activity beyond that of the control cream; and
- **1 patient (5.6%)** showed better itch reduction with the control cream.

INM-755 CBN cream demonstrated sufficient clinically important anti-itch activity to warrant further development. InMed will now pursue strategic partnership opportunities for INM-755 in chronic itch and other related diseases.

Corporate

Depth in Pharmaceutical R&D

EXTENSIVE EXPERIENCE IN PHARMA DISCOVERY, Drug Development

Eric A. Adams, MIBS

Chief Executive Officer

30+ years of experience in global biopharma leadership in BusDev, S&M with enGene, QLT, Abbott, Fresenius

Neeta Jagpal, CPA

Chief Financial Officer

20+ years of biotech financial leadership: Zymeworks, Angiotech, D-Wave, Ernst & Young.

Michael Woudenberg, PEng

Chief Operating Officer

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

Colin Clancy

VP, IR & Corp Comms

15+ years of experience in finance, investor relations & business development in Pharma, legal cannabis, mining & financial services industries

Eric Hsu, PhD

SVP, Preclinical R&D

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

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.

Distinguished Board of Directors

Andy Hull

Chair, Board of Directors

30+ years of commercial and business development roles with leading pharma companies including Takeda, Immunex, Abbott.

Nicole Lemerond, CFA

Chair, Compensation Committee

25+ years of experience in investment management, private equity and investment banking with companies such as Lehman Bros. and The Carlyle Group.

Neil Klompas, CPA, CA

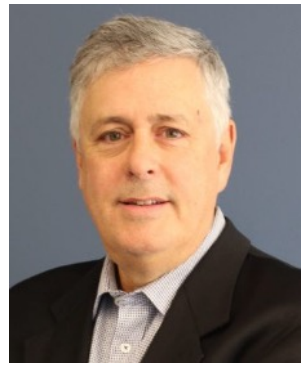
Chair, Audit Committee

30+ years of senior management in biotech, including CEO of Augurex and President/COO and CFO of Zymeworks.

John Bathery

Chair, Governance Committee

30+ years of pharma industry experience, including corporate development and strategic partnerships at Takeda and at TAP Pharmaceuticals.



Eric A. Adams, MIBS

Chief Executive Officer

30+ years of experience in global biopharma leadership in BusDev, S&M with enGene, QLT, Abbott, Fresenius.

Nasdaq : INM

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Financial Snapshot (As of 03/30/2026)

Cash and Short-term Investments	$7M(1)
Shares I/O	4.2 M(2)
Options	197 K
Warrants and Preferred Investment Options	2.6 M(3)
Fully Diluted Shares	7 M
Close	$0.65
52-week High	$7.98
52-week Low	$0.65
Average Daily Volume (Trailing 50 Days)	126 K
Market Cap	$2.7 M(2)

(1) As of December 31, 2025
(2) Includes ~1M unexercised, fully paid 'pre-funded warrants' (PFW's) from June 2025 financing
(3) Includes 1.95M warrants @$2.43, expires December 2026. All with one institutional investor.

Key Value Drivers – 2026 /2027

Advance INM-901 to IND and First-in-Human Trials

- Targeting pre-IND meeting with FDA in Q3 2026
- Complete outstanding IND-enabling pharmacology and toxicology studies
- Submit IND in early 2027
- Initiate Phase I clinical development in 2027, creating a key value inflection point

Progress INM-089 to IND and Clinical Development

- Execute IND-enabling activities, including preclinical validation, formulation optimization, and manufacturing readiness
- Planning for pre-IND meeting with FDA in Q4 2026
- Submit IND and advance into first-in-human studies targeting a differentiated position in the Dry AMD sector

Expand Proprietary Pipeline with High-Impact Drug Candidates

- Leverage discovery platform to identify and prioritize novel, IP-protected small-molecule assets

Execute Strategic Initiatives to Maximize Shareholder Value

- Pursue strategic partnerships, co-development opportunities for pipeline

InMed Pharmaceuticals

Thank you!

Forward Inquiries to, Colin Clancy

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Investor Relations & Corporate Communications

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