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February 2024.

**NYSE American: IGC**


The matters discussed in this presentation include forward-looking statements about the business prospects of IGC Pharma, Inc. Forward-looking statements are often preceded by words such as believes, expects, anticipates, plans, will, goal, may, intends, assumes, or similar expressions. Forward-looking statements reflect management's current expectations as of the date of this conference call and involve certain risks and uncertainties. The forward-looking statements are based on assumptions that we have made in light of our industry experience and our perceptions of historical trends, current conditions, expected future developments, and other factors that we believe are appropriate under these circumstances.

As with any projection or forecast, they are inherently susceptible to uncertainty and changes in circumstances. IGC Pharma, Inc.'s actual results could differ materially from those anticipated in these forward-looking statements as a result of various factors and the forward-looking statements are not guarantees of performance. Some of the factors that could cause future results to materially differ from recent results or those projected in forward-looking statements are included in our filings with the Securities and Exchange Commission (the “SEC”), such as our Annual Report on form 10-K filed with the SEC on on July 7, 2023, and subsequent filings on Form 10Q. We are under no obligation and expressly disclaim any obligation to update or alter the forward-looking statements, whether as a result of such changes, new information, subsequent events, or otherwise.

# SAFE HARBOR

# COMPANY OVERVIEW

- Our focus is treating **Alzheimer's disease (AD)** through our growing pipeline of **five drug assets**
- **Lead therapeutic candidate IGC-AD1 is currently in a 146-person Phase 2b trial for agitation in dementia due to Alzheimer's.** IGC-AD1 is a CB1r partial agonist that reduces neuroinflammation and restores neurotransmitter imbalance
- TGR-63 and three other candidates have demonstrated in Alzheimer's cell lines the potential to **ameliorate plaques and/or tangles, two hallmarks of Alzheimer's**

# ALZHEIMER'S DISEASE

**1in 9**
**65+ yrs.**

Americans with Alzheimer's in 2023[1]

**13.8 Million**

Americans with Alzheimer's by 2050[1]

**6.7 million**

Americans with Alzheimer's age (65+) in 2023[1]

**$1 Trillion**

Expected total expenses for Alzheimer's and other dementias by 2050[2]

**The global number of persons with AD dementia, prodromal AD, and preclinical AD were estimated at:**

- AD dementia: **32 million.**
- Prodromal AD: **69 million.**
- Preclinical AD: **315 million.**

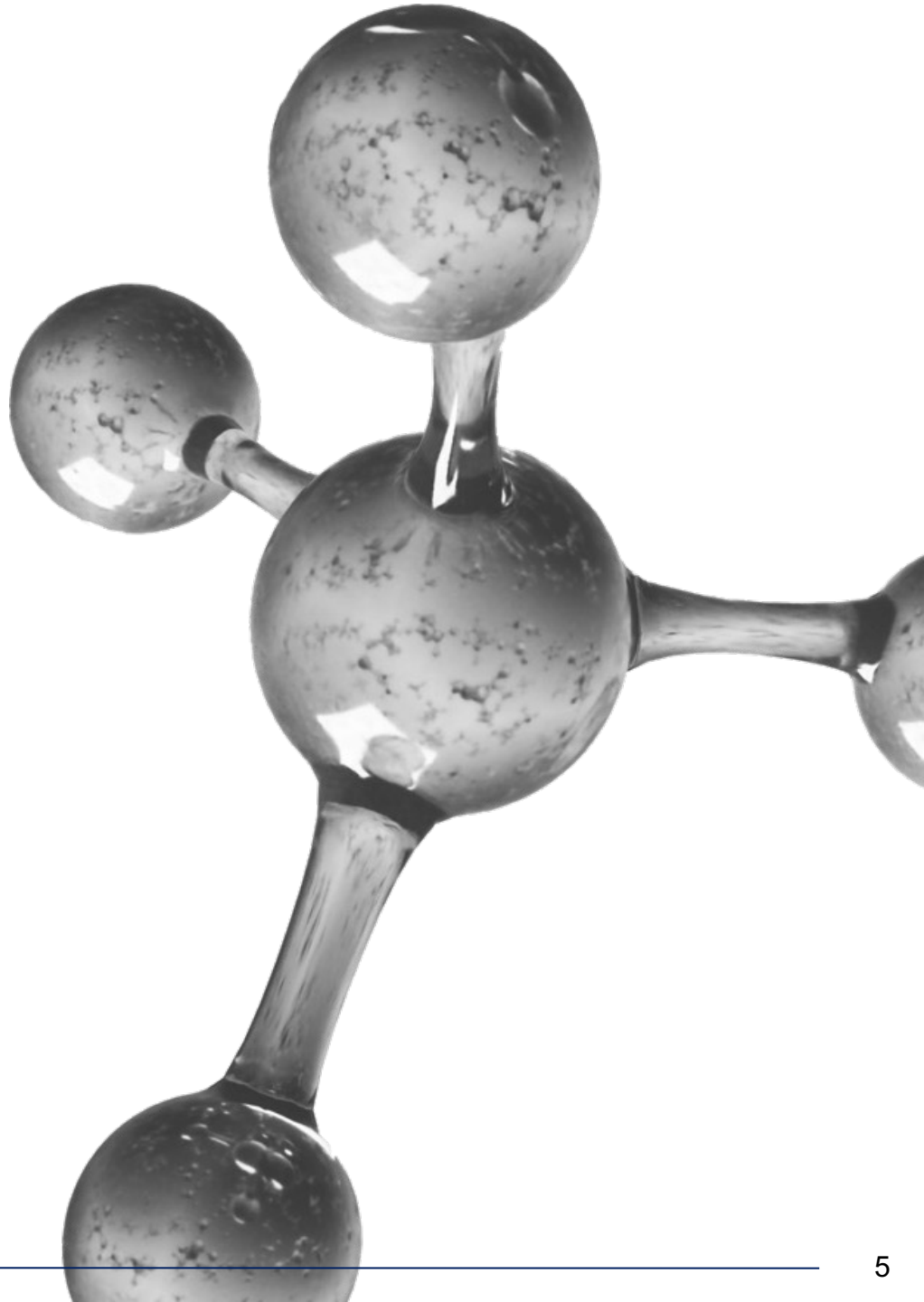
**Together they constitute:**

- **416 million** across the AD continuum
- Or **22%** of all persons aged 50 and above[2]

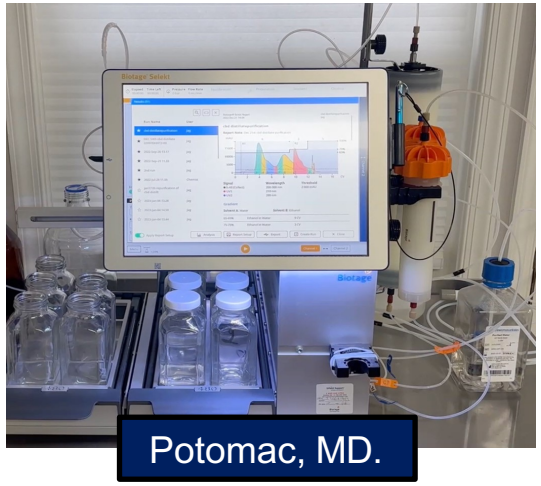
References: (1) Alzheimer's Association 2023 Alzheimer's Disease Facts and Figures. (2) Economic Burden of Alzheimer Disease and Managed Care Considerations, Winston Wong, 2017
References: (2) Alzheimer's Association 2022 Global estimates on the number of persons across the Alzheimer's disease continuum, Gustavsson & Others, 2022, https://alz-journals.onlinelibrary.wiley.com/doi/10.1002/alz.12694

# INVESTMENT HIGHLIGHTS

- Two Patented and three patent pending investigational drug candidates targeting Alzheimer's
- All showing favorable pre-clinical attributes towards Alzheimer's disease pathology
- **IGC-AD1** is currently in **Phase 2B with data milestones expected in mid-2024**
- Leading **artificial intelligence** partnerships
- Manufacturing and production facilities
- **Clean capital structure**
- **Manufacturing and production facilities**
- **CLEAN CAPITAL STRUCTURE**

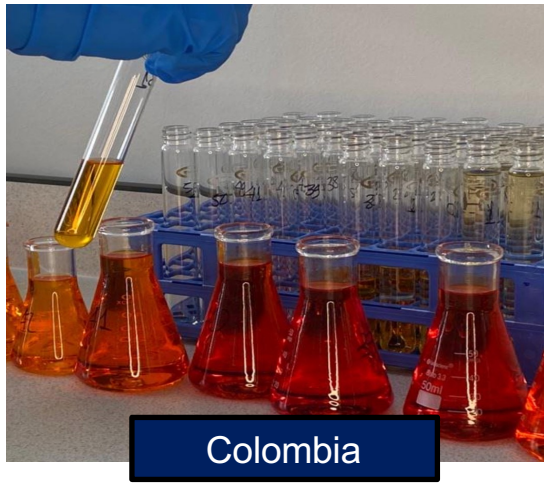


# VERTICALLY INTEGRATED OPERATIONS

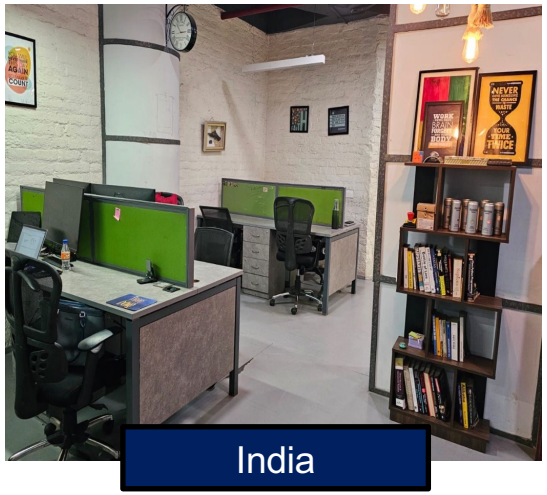



Headquarters


cGMP Manufacturing and Processing facility




Licensed R&D facilities




Analytics

## Constructed Significant Operational Barriers to Entry

- Approved licenses
- Phase 3-ready manufacturing facilities
- Facilities for potential commercialization
- Licensed access to raw APIs
- Licensed access to purification
- State-of-the-art distillation facilities

# AD PIPELINE OVERVIEW

| ASSET | PRECLINICAL | PHASE 1 | PHASE 2 | PHASE 3 | MILESTONE |
|---|---|---|---|---|---|
| IGC-AD1 | TARGETS neuroinflammation, neurotransmitter imbalance, inflammasome-3 and AD pathology | | | | Interim Topline data Second half 2024 |
| TGR-63 | TARGETS Aβ PLAQUES (Early- Moderate Stage Alzheimer's) | | | | Tox study 2024 |
| LMP | TARGETS neuroinflammation, neurotransmitter imbalance, inflammasome-3 and AD pathology | | | | Bio Equivalence to IGC-AD1 2025 |
| IGC-M3 | TARGETS Aβ PLAQUE AGGREGATION (Early Alzheimer's) | | | | Tox study 2024 |
| IGC-1C | TARGETS TAU AND NEUROFIBRILLARY TANGLES | | | | Tox study 2025 |

# AGITATION IN ALZHEIMER'S

**Agitation:** excessive motor activity, verbal aggression, or physical aggression that is severe enough to impair personal relationships, social functioning, and/or daily activities[1].

**Agitation** starts early in AD and increases in severity as the disease progresses[2].

**76% of Alzheimer's patients suffer from agitation[3]**

**Agitation is associated with[4]:**

- Higher admission rate to assisted living facilities
- Higher use of medications
- Long-term hospitalization
- Higher mortality

- In 2023 the **FDA-approved Brex (Brexpiprazole)** to treat agitation in AD dementia - a repurposed atypical anti-psychotic with a black box warning

* References: (1) Cummings J et al., 2014 (2) Rebecca Palm et al., 2021 (3) Van der Mussele et al., 2015 (4) Economic Burden of Alzheimer Disease and Managed Care Considerations, Winston Wong, 2017

# OUR SOLUTION

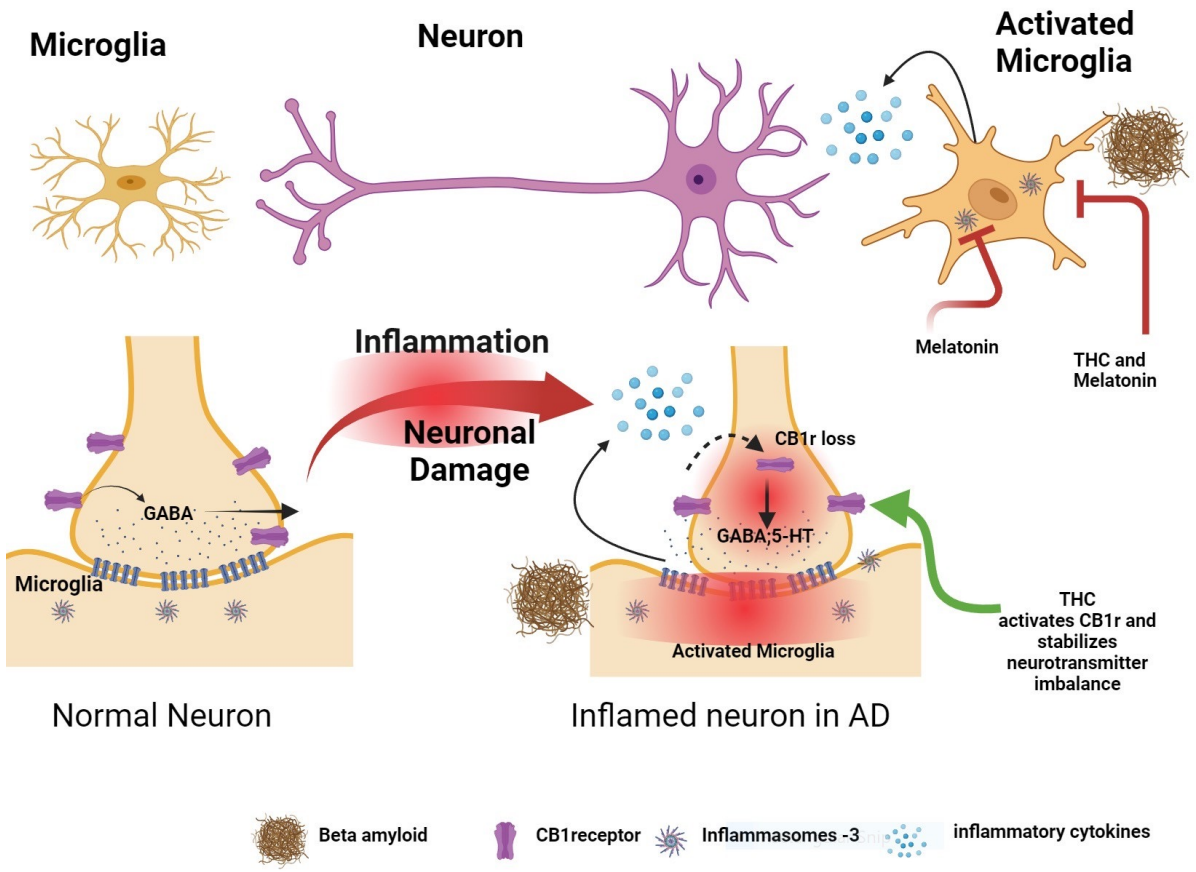
## The Promise of IGC-AD1

Two APIs that target 1) neuroinflammation, 2) neurotransmitter imbalance, 3) CB1r agonism and 4) inflammasome-3, all implicated in agitation in AD

IGC-AD1 can potentially reduce agitation, and act on AD pathology (plaques, tangles) making it a significantly more powerful alternative

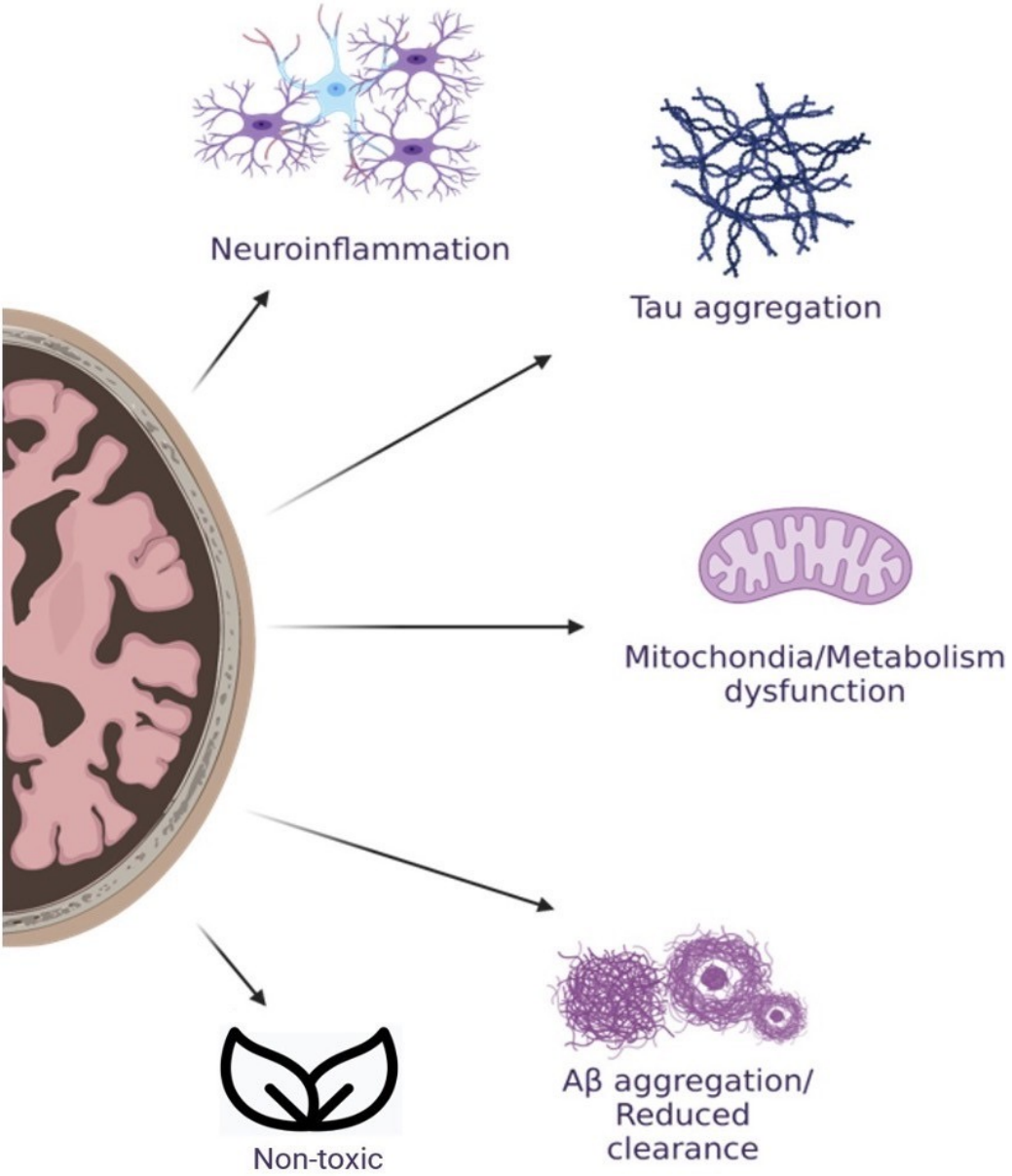
A patent-protected compound in **Phase 2B** trials that targets agitation in Alzheimer's

IGC-AD1 would be a treatment option that is **not an antipsychotic with a black box warning**




**IGC-AD1 contains two APIs that are safer than traditional antipsychotic therapies.**

# DATA FROM PRECLINICAL TRIAL



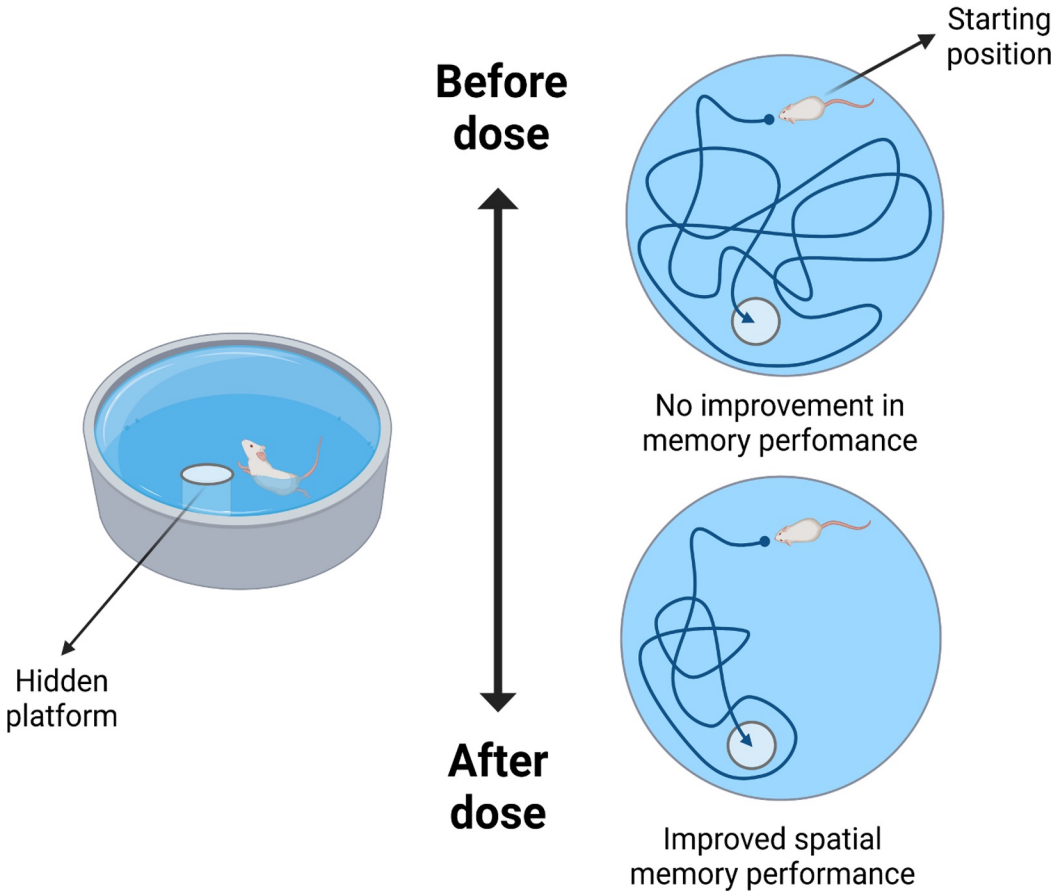

## RESULTS ON ALZHEIMER'S CELL LINES

**Reduces two key hallmarks of Alzheimer's plaques and tangles (pTau):**

- Inhibits the aggregation of amyloid plaque
- **Reduces** phosphorylated tau (pTau)
- Enhances mitochondrial function
- **Non-toxic.** Repeated low dosing over 48 hours is non-toxic

J. Pineal. Res. 2011, 51, 75-86; J. Alzheimer's disease 2014, 42, 973-984; Int. J. Mol. Sci. 2022, 23, 2757; Int. J. Mol. Sci. 2022, 23, 4253

# PRECLINICAL DATA: ANIMAL MODEL




## MEMORY IMPROVED IN AD (APP/PS1) MOUSE MODEL

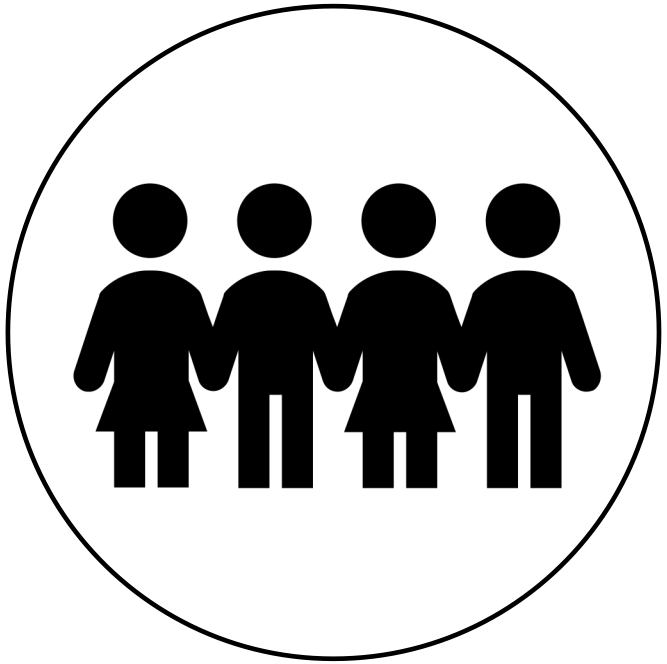
**IGC- AD1** significantly **improved times** and **fewer errors in a Morris Water Maze** test than those in the control group in an Alzheimer's mouse model

- The maze uses spatial cues for mice to navigate a swimming container full of stained water and find a safe platform.[1]
- The memory task is assessed in multiple trials to measure how well the mouse finds the platform.[1]

References: (1) Nature Protocols. 2006; 1: 848-858; Int. J. Mol. Sci. 2022, 23, 2757

# IGC-AD1 PHASE I CLINICAL TRIAL, DATA

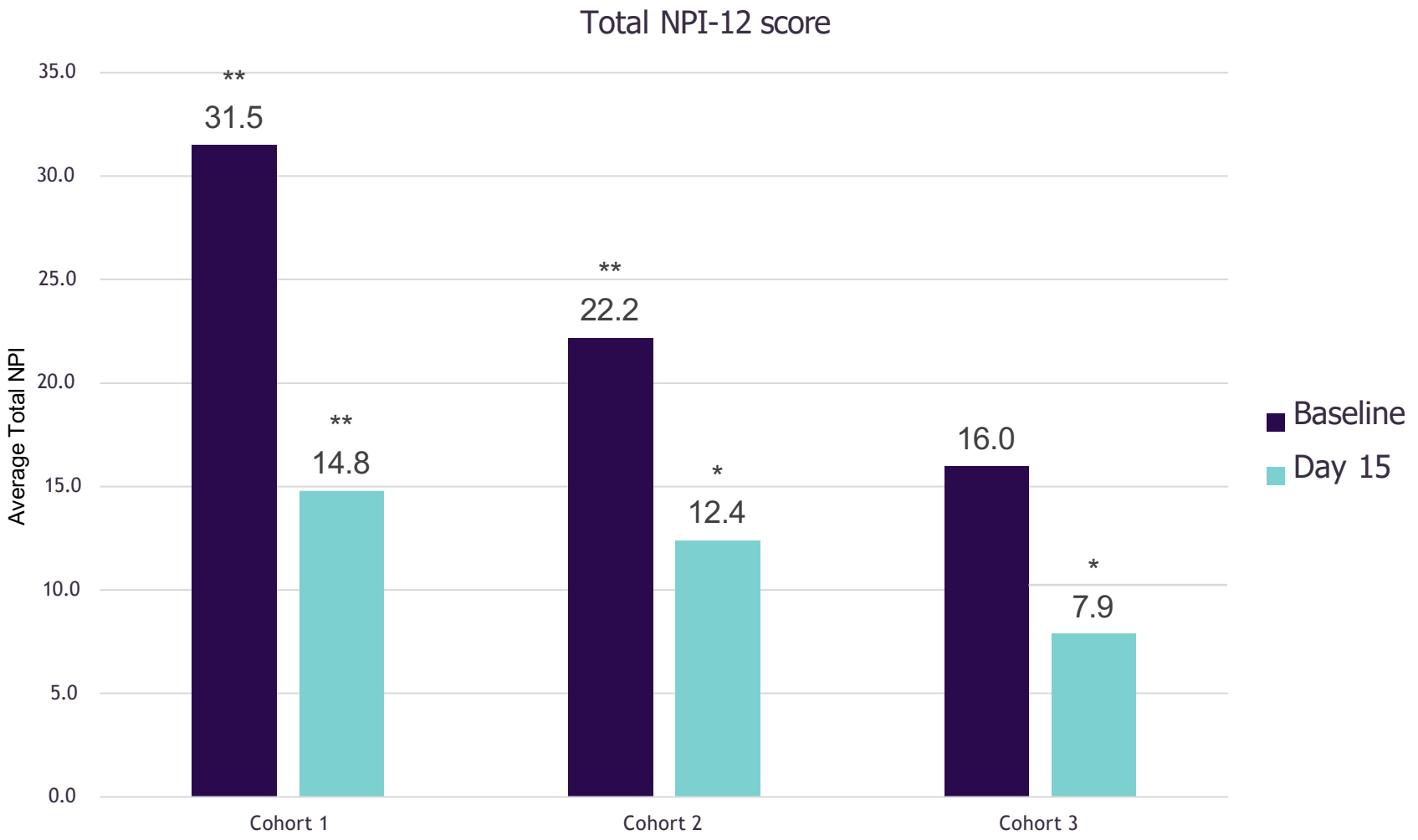
Multiple Ascending Dose (MAD) study to evaluate safety and tolerability of IGC-AD1 in participants with AD & NPS (Neuropsychiatric Symptoms) using the NPI (Neuropsychiatric Inventory)



## *Safety, Tolerability, Agitation, NPS*

- No life-threatening or serious adverse events at any dosing level
- Decrease in agitation by 36% - 66% depending on dosage (NPI agitation scale)
- Decrease in depression by 67% - 75% depending on dosage (NPI depression scale)
- Phase 1findings show IGC-AD1 has potential to treat NPS in AD

# DATA ON NEUROPSYCHIATRIC SYMPTOMS MEASURED BY NPI SCORES

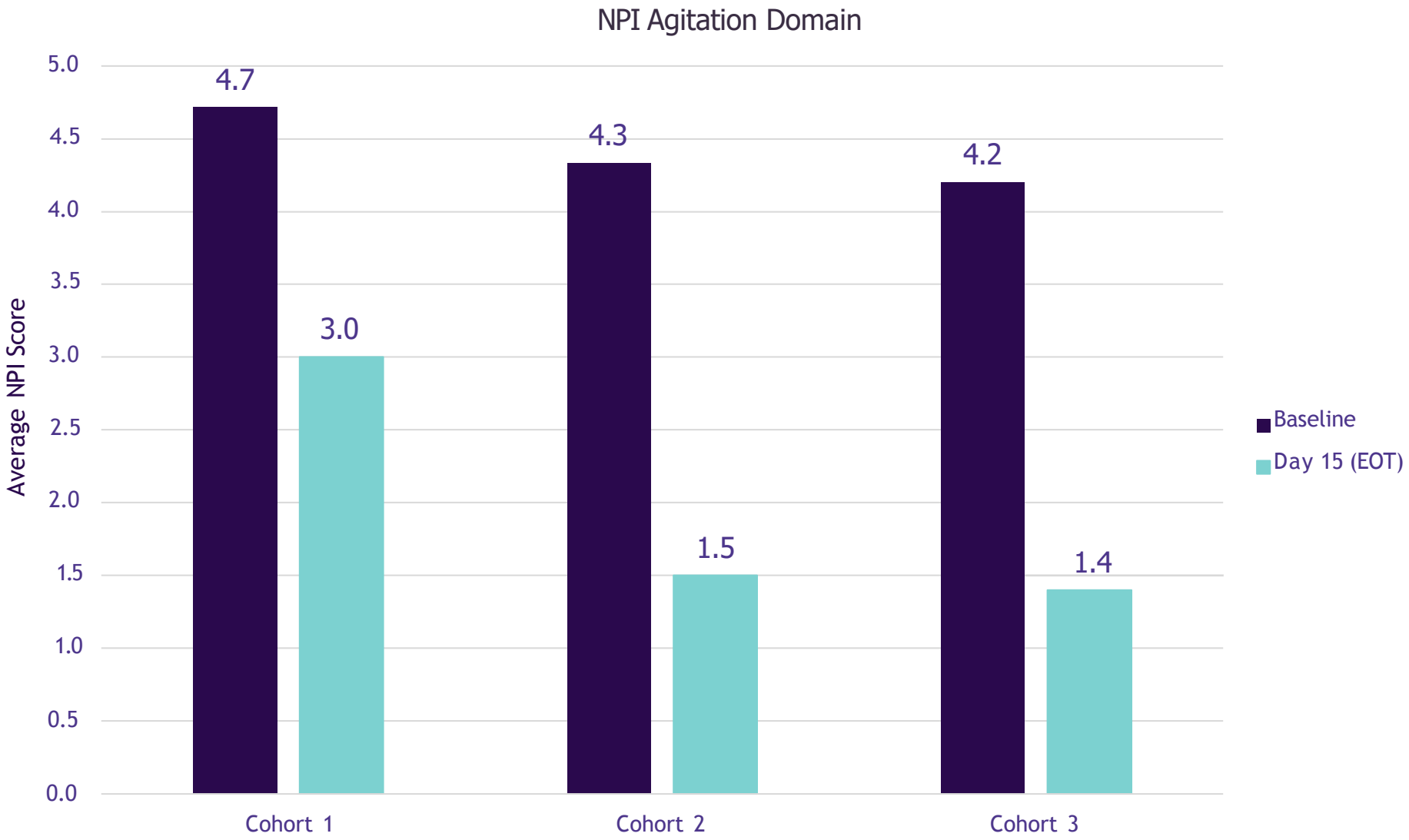



## Results on Neuropsychiatric Symptoms (NPS) Measured by NPI-12

Patients taking IGC-AD1 showed an **overall improvement in NPS in all three cohorts**

Caregiver distress improved as well.

# DATA ON AGITATION




Cohorts 1, 2,3 received IGC-AD1 once a day, twice a day and three times a day respectively

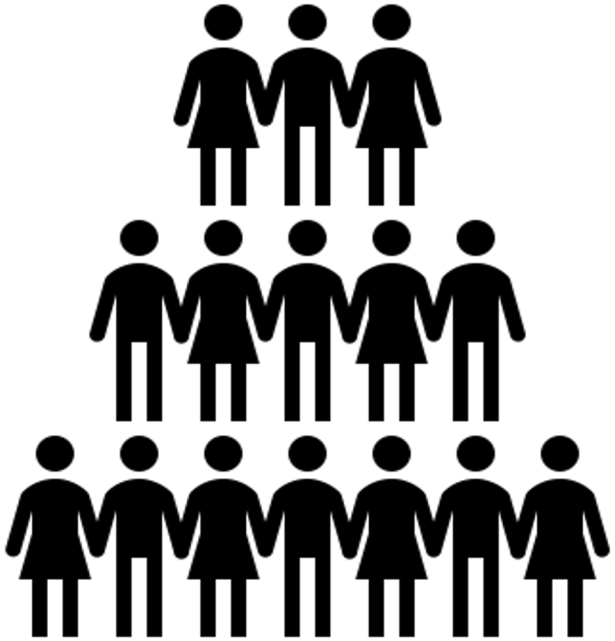
At three different doses, agitation improved both clinically and statistically ($p<.05$)

Cohort 1 baseline 4.7 EOT 3.0
Cohort 2 baseline 4.3 EOT 1.5
Cohort 3 baseline 4.2 EOT 1.4

# ON GOING IGC-AD1 PHASE 2b

## Placebo Controlled, Double Blind, Randomized, Multi Site

Phase 2b protocol seeks to show that IGC-AD1 is effective, compared to placebo, in lowering agitation in participants with Alzheimer's



### Objective

- Evaluate **if IGC-AD1 is superior to placebo in reducing agitation** in a six-week trial

### Key Inclusion Criteria

- Individuals 60 years and above
- Diagnosis of AD with established and persistent agitation

### Sites

- 20-30 trial sites

**TARGET: 146 Participants**

# AI-INTEGRATED TRIAL

IGC is partnered with leading Artificial Intelligence research center **CINFONIA at the University of Los Andes**

Partnership aims to **leverage deep learning algorithms and generative AI** to analyze variations in disease signatures among patients, enabling IGC Pharma to identify individuals more likely to respond to treatment and subsequently **accelerate the delivery of treatments to patients**

Our goals:

- **Optimize clinical trials**
- **Personalized medicine**
- **Personalized medicine.**

  **Early detection of Alzheimer's**

IGCPHARMA

+

CinfonIA
CENTRO DE INVESTIGACIÓN Y FORMACIÓN EN INTELIGENCIA ARTIFICIAL

AD DRUG CANDIDATE 2

# TGR-63 Background

## TGR-63 A PROMISING PATH-BREAKING MOLECULE

- India-based Jawaharlal Nehru Centre for Advanced Scientific Research (JNCASR) created the **TGR-63 molecule**
- IGC acquired exclusive rights to **TGR-63** from JNCASR researchers in 2022
- Pre-clinical testing demonstrates that **TGR-63 holds potential to ameliorate plaque** due to AD
- Behavioral tests with AD (APP/PS1) mice show that **TGR-63 can:**
  - ✓ **Rescue neuronal cells from amyloid toxicity**
  - ✓ **Minimize learning deficiency, memory impairment & cognitive decline**
- **Current status:** Toxicology and other studies leading to Phase 1trials in progress

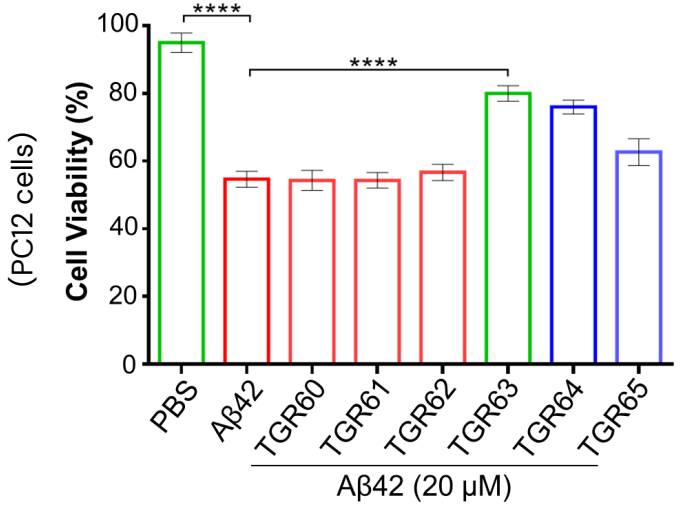
*Subject to FDA trials and approvals

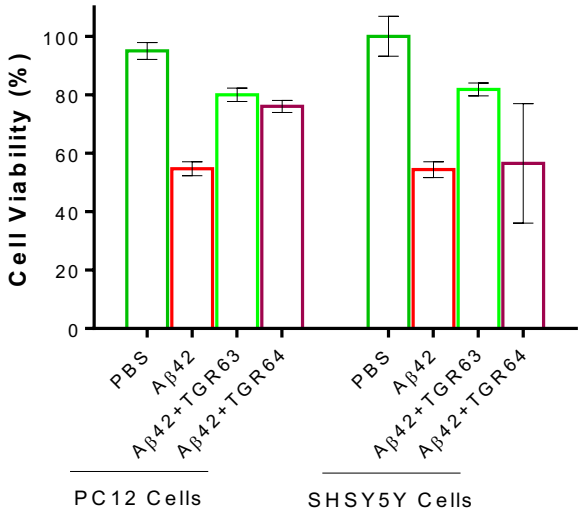
# TGR-63: Mode of action

To study how TGR-63 can treat AD pathology and cognitive and behavioral symptoms, the AD-like environment was mimicked in vitro by exposing cultured PC12 and SHSY5Y cells to Aß42.

This resulted in the generation of cytotoxic aggregation species in the growth media. Aß42 caused mutilation to the cultured neuronal cells, as revealed by the decreased cell viability (54%) compared to the PBS control.

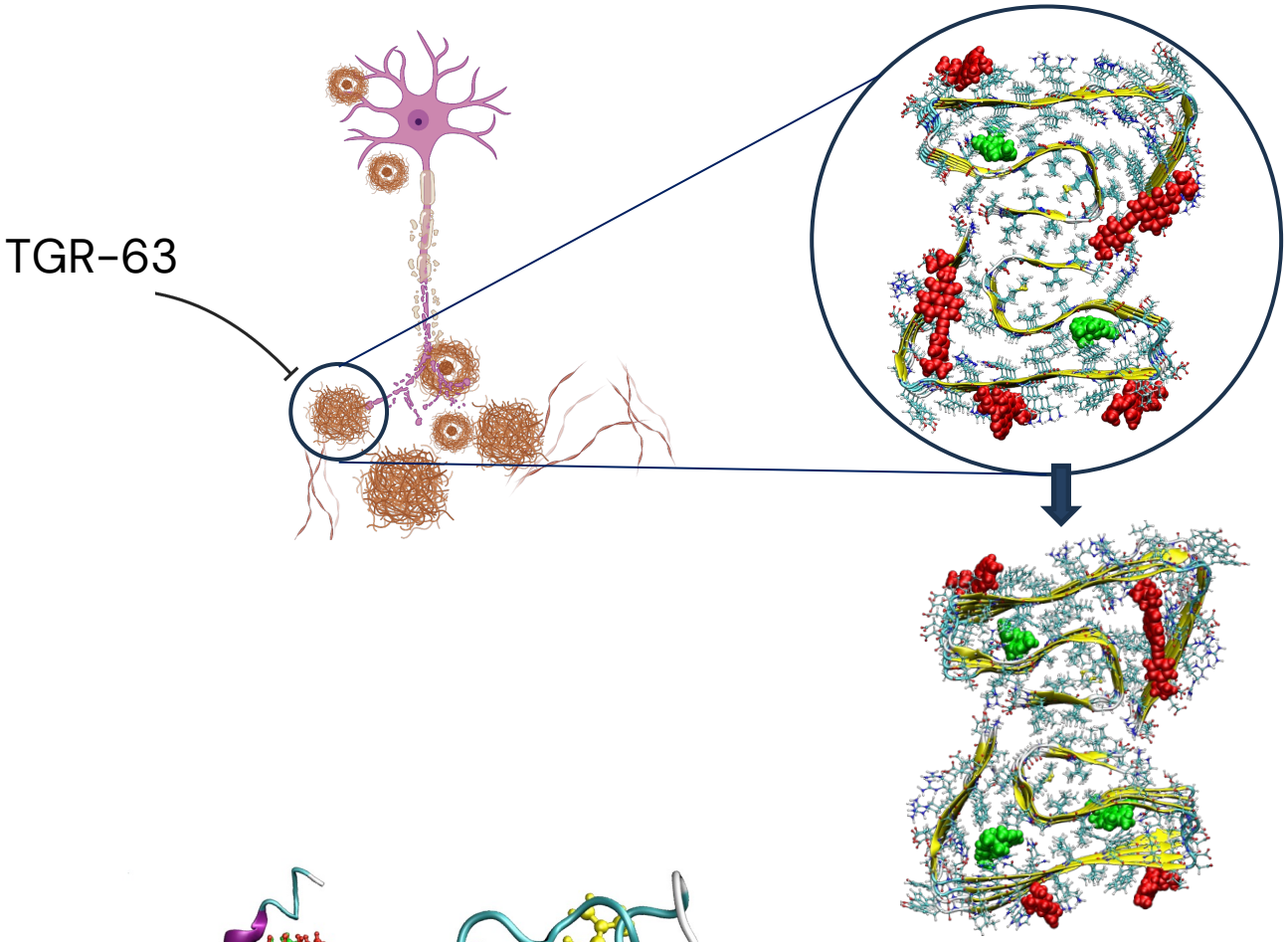
However, TGR-63 treated cells increased their viability to 80%, which means an increase of 26% in neuronal rescue.




*Adv. Therap.* **2021**, *4*, 2000225

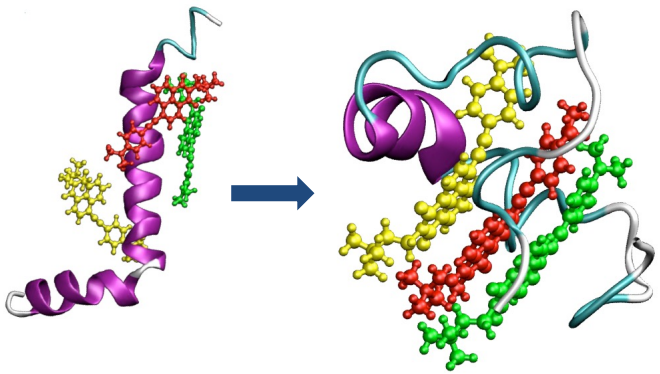
# TGR-63: Mode of action




- To understand the molecular mechanism underlying its ability to decrease plaque aggregates and increase neuronal viability, computational studies like molecular docking and molecular dynamics were performed.
- These suggested that the molecular mode of action of TGR-63 consists of disrupting three different types of bonds within amyloid aggregates:

1. Hydrogen bonds
2. Hydrophobic and π-π interactions
3. Salt bridges.

This destabilizes plaques, allowing it to break down.

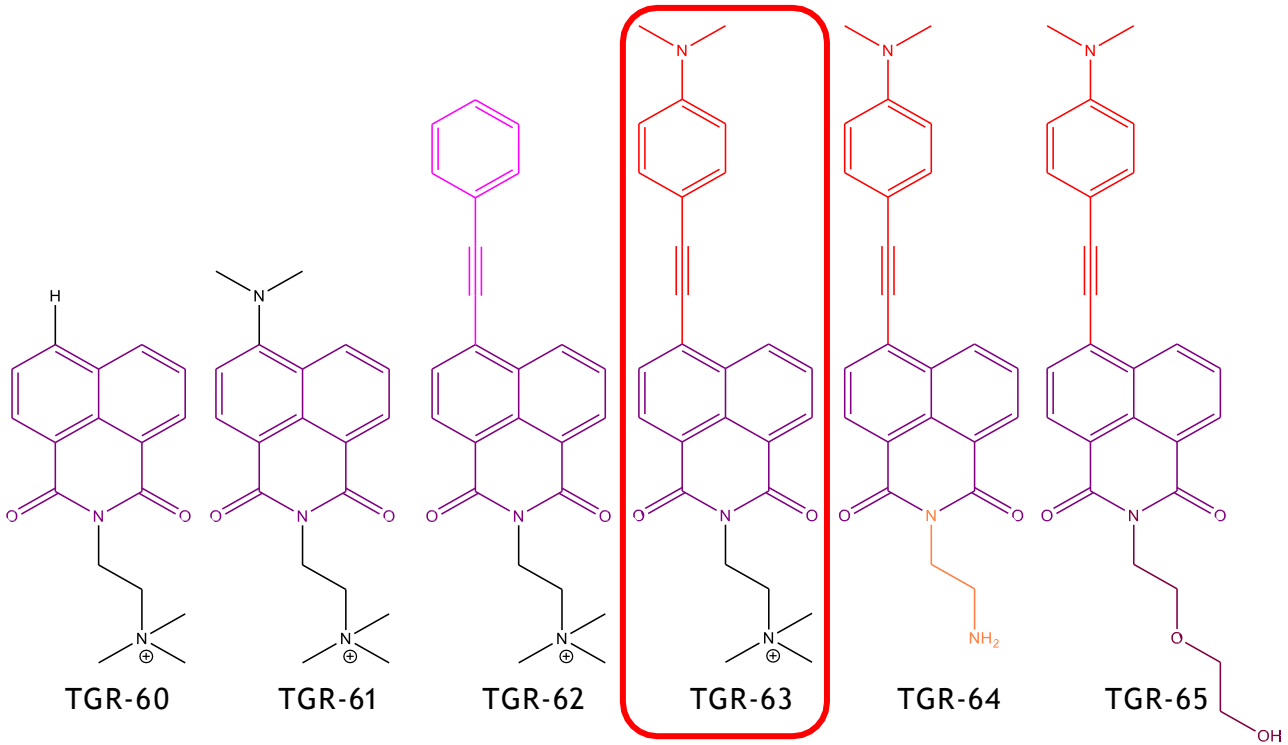


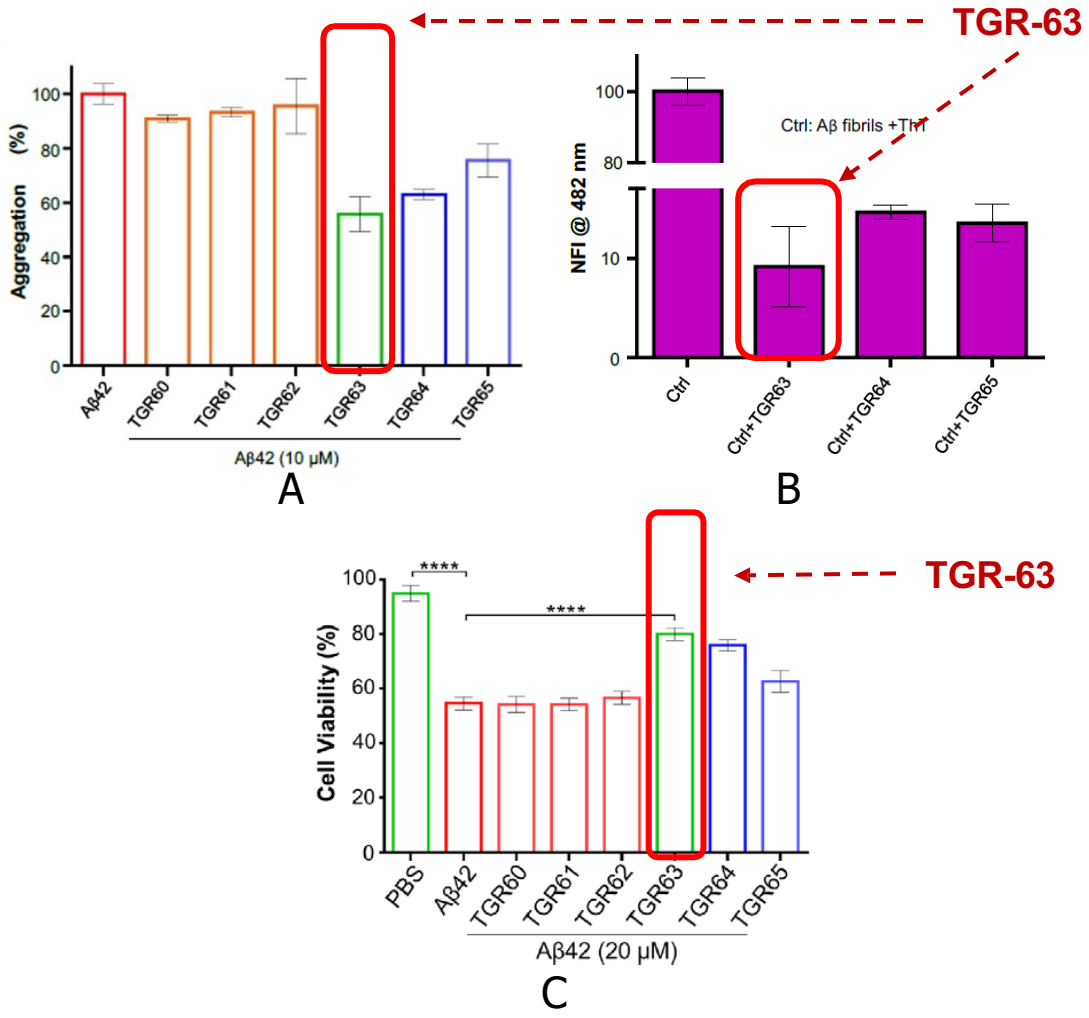
It also has a high affinity to the Aß42 peptide. The interaction of TGR-63 with the peptide compromises its tertiary structure, allowing the formation of globular non-toxic structure that can be metabolized.

# Molecular Structures for TGR

- Molecular structures that are candidates to modulate A$\beta$42 aggregation and associated toxicity.

- TGR-63 demonstrated higher capacity to inhibit A$\beta$42 aggregation (A), higher capacity to dissolve Aβ42 aggregates (B) and increased PC12 cell viability (C) when compared to other structures.

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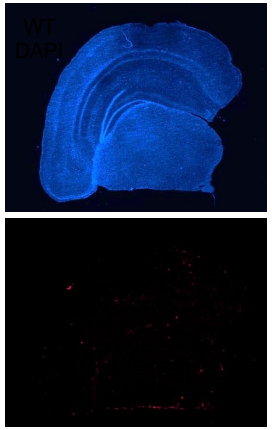
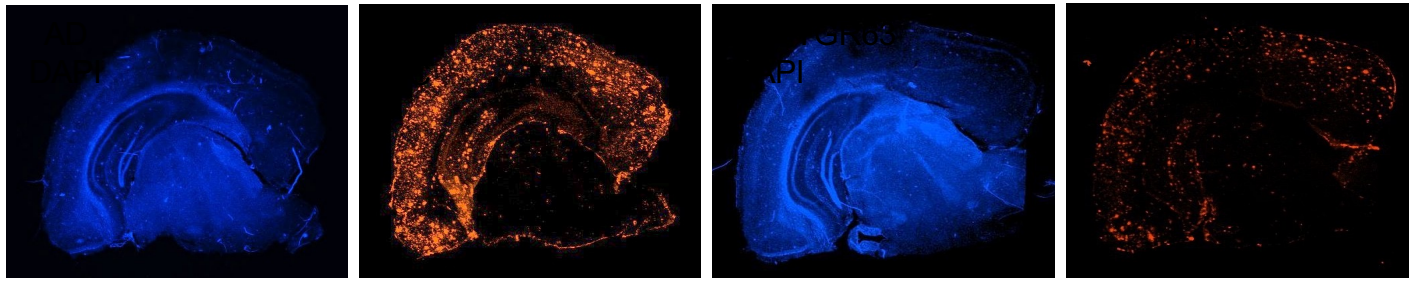
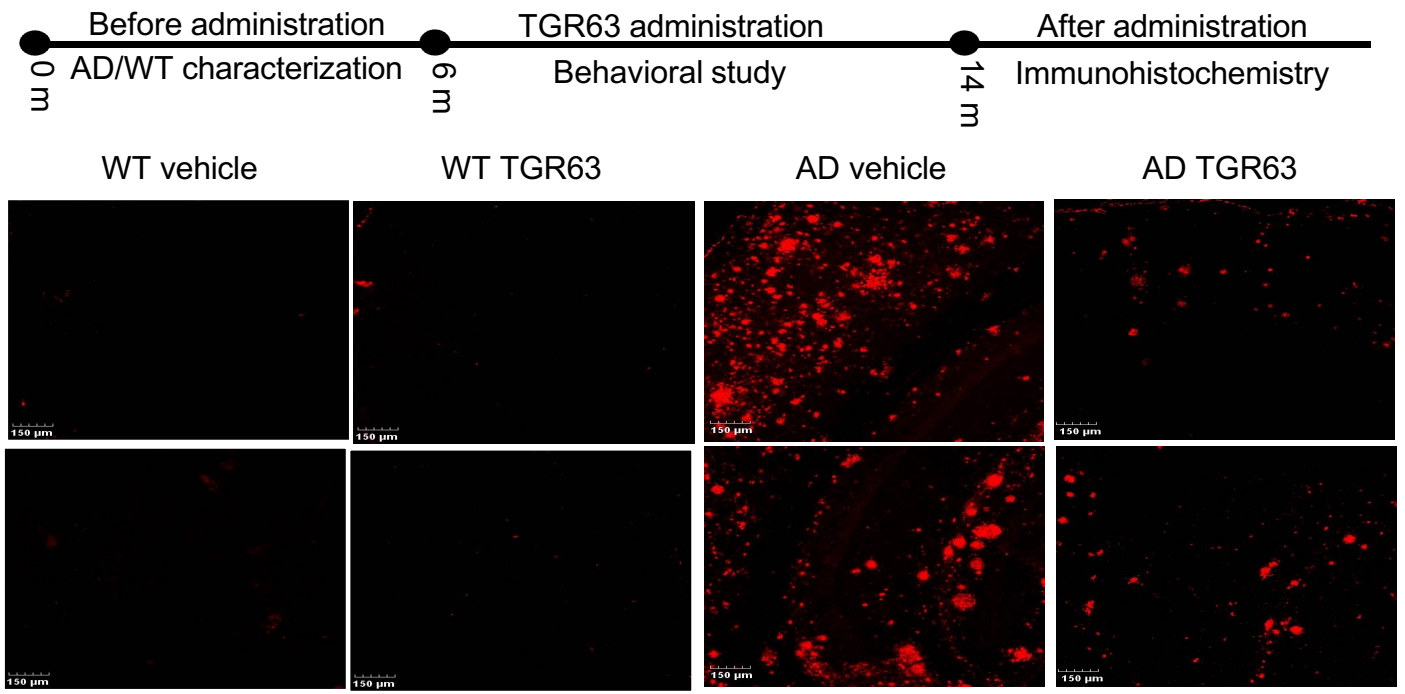
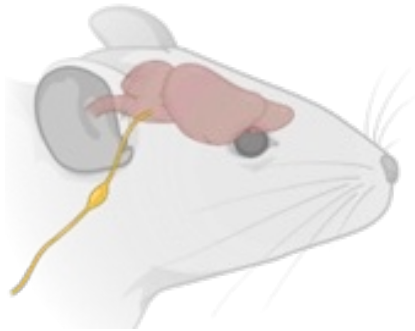
# The Promise of TGR-63: Reduction of Plaque in Mouse Model

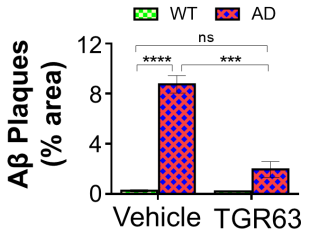
TGR-63 effectively reduced the amyloid burden in the mice cortex and hippocampus.

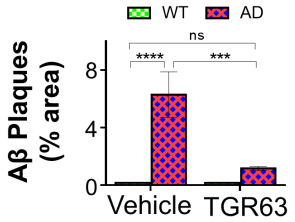






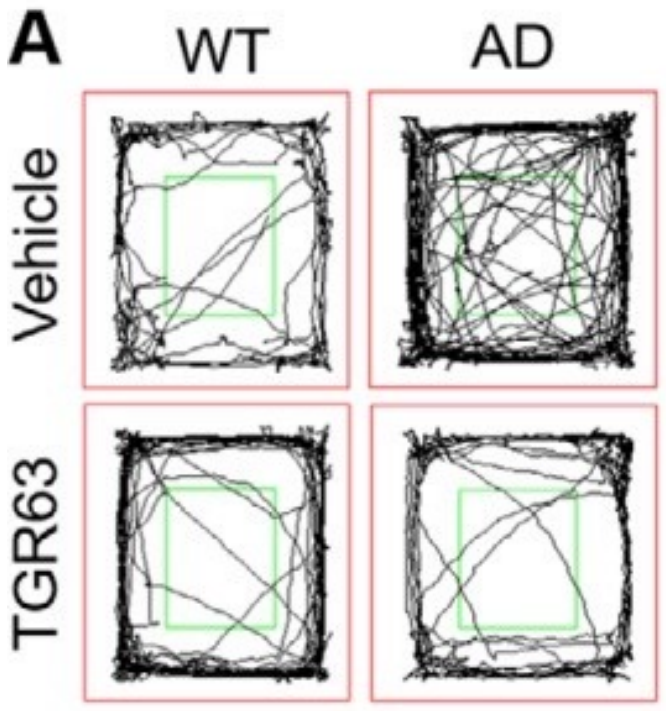

The brain tissue sections were immunostained with amyloid fibrils specific primary antibody (OC) and red fluorescent-labeled secondary antibody.

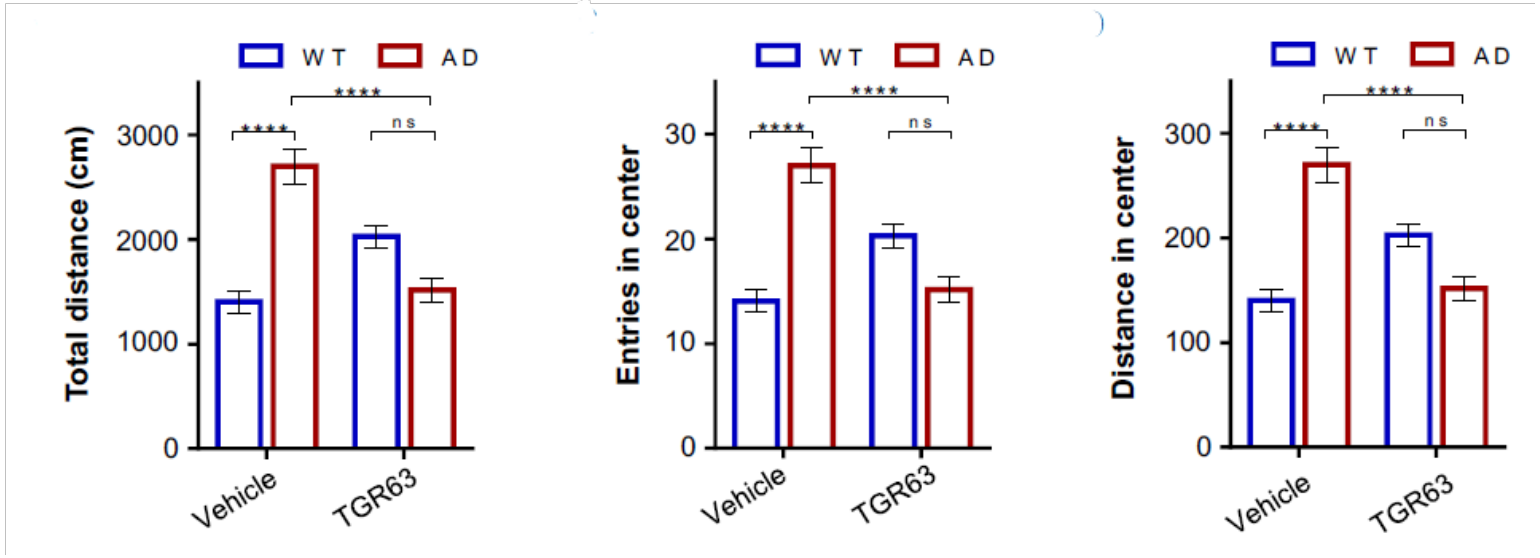
*Adv. Therap.* **2021**, *4*, 2000225

# TGR-63-Improved Behavior In a Mouse Model

TGR-63 improved cognitive performance in APP/PS1 and Wild Type mice. The dose administered was 5 mg/kg body weight.

**Open field test**




TGR-63 treated mice had a lower locomotion in the field, indicating improved behavior.

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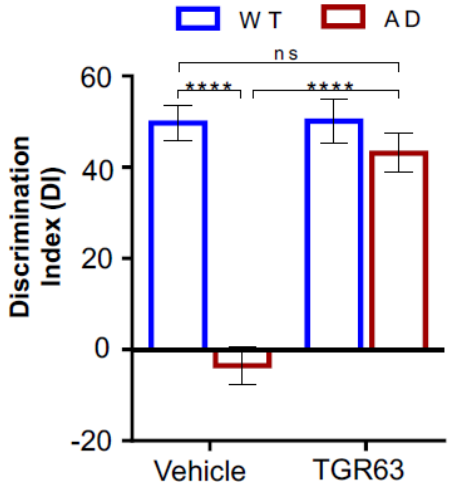
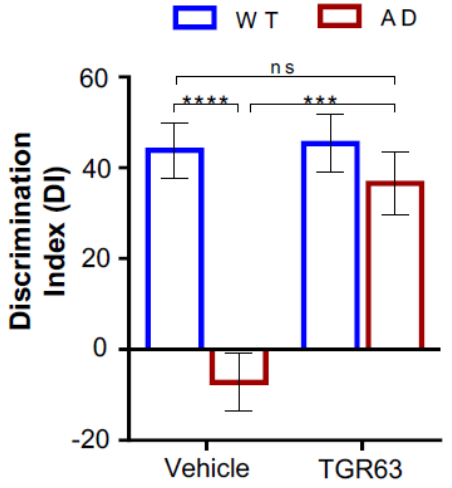
# TGR-63 Improved memory in a Mouse Model

## Novel object identification test

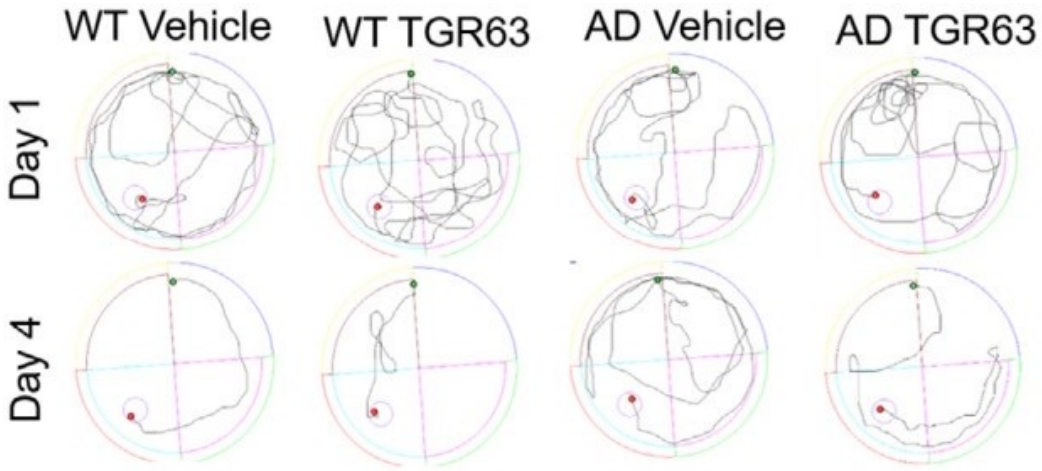
*Adv. Therap.* 2021, *4*, 2000225


TGR-63 treated mice had a higher exploration of the novel object.

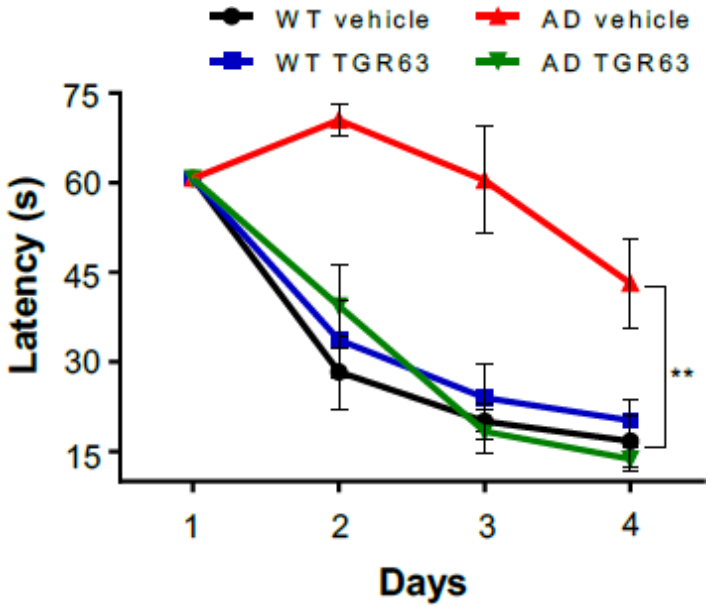


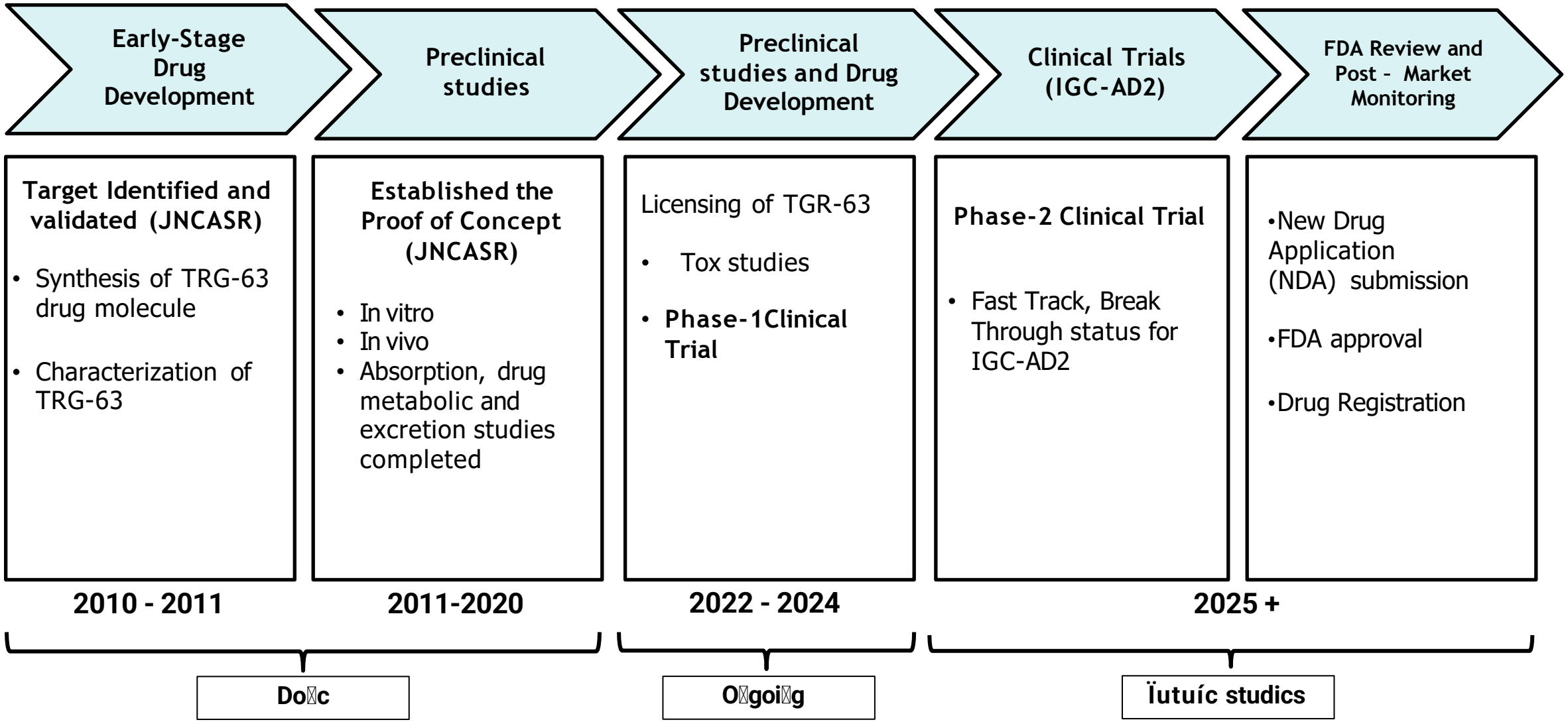

## Morris Water Maze




TGR-63 treated mice learned the location of the target and had less error rate than non treated mice.



# TGR-63 TO "IGC-AD2" TIMELINE



# FIRST MOVER ADVANTAGE WITH INTELLECTUAL PROPERTY

| TARGET | DESCRIPTION | PATENT APPLICATIONS | US-PATENTS GRANTED | FOREIGN PATENTS GRANTED |
|---|---|---|---|---|
| Alzheimer's Disease (IGC-AD1) | Composition & Method for treating CNS Disorders | 7 | 1 | 1 |
| Alzheimer's Disease (IGC-AD1) | Composition & Method for treating CNS Disorders | | 1 | |
| Alzheimer's Disease (TGR-63) | Naphthalene Monoimide Derivatives with ability to impact Aβ protein build-up | 6 | | |
| Alzheimer's Disease (IGC-1C) | Naphthalene Monoimide Derivatives with ability to impact Tau aggregation and neurofibrillary tangle formation | 1 | | |
| Alzheimer's Disease (IGC-M3) | Naphthalene Monoimide Derivatives with ability to impact Aβ plaque buildup and neurofibrillary tangle formation | 1 | | |
| Cancer (Naphthalene Diimdes) | Naphthalene diimide Derivatives with the ability to self-assemble molecular interactions for biological and nonbiological systems | | 1 | 1 |
| Alzheimer's Disease (IGC-LMP) | Composition, Synthesis, & Medical use of Hybrid Cannabinoid | 1 | | |
| Epilepsy | Composition & Method for Treating Seizures in humans & cats/dogs | 2 | 2 | |
| Eating Disorders | Cannabis formulation with Cyproheptadine for treating Cachexia & Eating Disorders | 1 | 1 | |
| Stuttering & Tourette Syndrome | Cannabinoid-Based formulation for Treating Stuttering & Symptoms of Tourette Syndrome | 3 | | |
| Pain | Cannabinoid-Based Formulation combined with Cobalamin and method for Pain Managment | 1 | 2 | 2 |

- In-house patents and applications
- Patents and applications acquired through exclusive license agreements

We have filed 24 patent applications, which are distributed among the US, Canada, Europe, Colombia, India, Mexico, Brazil, and Hong Kong.

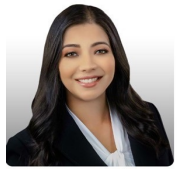
**23** **8** **4**

# SENIOR SCIENTIFIC TRIAL TEAM

## Scientific Management

**Dr. Varduhi Ghazaryan, MD, MPH**
Medical Director

NIH National Institutes of Health
*Turning Discovery Into Health*



**Dr. Saadia Shahnawaz, MD**
Medical Director

HC HOLY CROSS HEALTH

**Dr. Juan Manuel Orjuela, MD**
Neuropsychiatrist

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Scientific Manager

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# SENIOR SCIENTIFIC ADVISORS

## Inventors

**Prof. Chuanhai Cao, PhD**
Professor of Pharmaceutical Science

**Prof. T Govindaraju, PhD, FRSC.**
Professor - Bioorganic Chemistry

**Dr. L. Elliot Hong, MD**
Professor Psychiatrist

## Scientific Advisors

**Dr. James Saunders, PhD**
Ret. Professor, Molecular Biology

**Jeffrey L. Cummings**
MD, ScD, is Chair of the ACTC Neuropsychiatric Symptoms Committee

# POTENTIAL TO DRIVE SIGNIFICANT VALUE

| | NORTH AMERICA AND EUROPE | | |
|---|---|---|---|
| **ITEM** | **Year 1** | **Year 2** | **Year 3** |
| Individuals with AD | 15M | 15M | 15M |
| Agitation in AD (76%) | 11M | 11M | 11M |
| %adoption of drug (IGC-AD1) | 3% | 5% | 10% |
| Annual Price/Patient of drug | $2,400 | $2,400 | $2,400 |
| **Estimated Revenue** | **$0.8B** | **$1.3B** | **$2.6B** |

## Key Market Factors

- No non-antipsychotic FDA approved drugs for the treatment of agitation due to Alzheimer's exist today
- Distribution strategy involves direct to consumer e-commerce accelerating time-to-market
- Accessible at an affordable price

www.alzheimer-europe.org/ - https://www.alz.org/ - https://www.hsph.harvard.edu/news/press-releases/alzheimers-international-survey/ - https://www.alzint.org/about/dementia-facts-figures/dementia-statistics

# ESTIMATED PUBLIC COMPANY VALUATIONS DURING TRIALS

## Assumptions

- Public.
- Year shows year when the company was in the clinical stage that is mentioned.
- Estimated valuation is the valuation at the time the company was closest to Phase entered the Phase that is mentioned.
- Current Market valuation is public valuation currently.

| Script | Symbol | Clinical trial for disease | Name of formulation | Clinical Stage | Year | Estimated Valuation at the time of clinical Stage | Market Valuation in 2024 |
|---|---|---|---|---|---|---|---|
| **IGC PHARMA** | **IGC** | **AD** | **IGC-AD1** | **IIb** | **2023** | **$20M** | **$17M** |
| **Axsome Therapeutics** | AXSM | Pain: complex regional pain syndrome (CRPS) | AXS-02 | II/III | 2015 | $162M | $4.24B |
| **Acumen Pharmaceuticals** | ABOS | Alzheimer's | ACU193 | I | 2022 | $188M | $192M |
| **Acadia Pharmaceuticals Inc.** | ACAD | Schizophrenia | ACP-103 | PC/II | 2006 | $265M | $4.62B |
| **Cassava** | SAVA | Alzheimer's | PTI-125 | II | 2020 | $260M | $1.1B |
| **GW Pharma** | JAZZ | Dravet Syndrome (CBD) | GWP42003-P | I | 2013 | $389M | $7.56B (sold) |



# Initiating Coverage with BUY and $2.75 Target (~ $167m valuation)



https://ascendiant.com/

# THANK YOU




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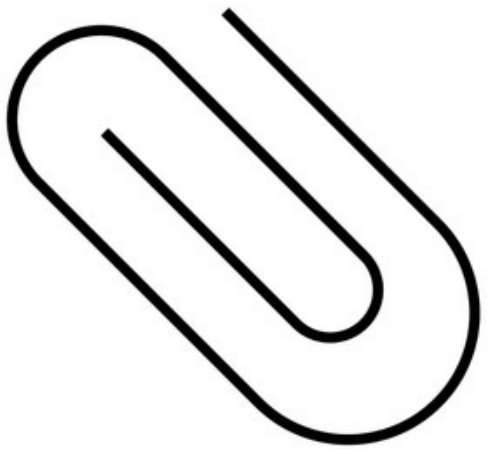
IGCPHARMA



# ADDITIONAL INFORMATION

# Caregiver Testimonial

## From our Phase 1IGC-AD1 study

"I want to tell you a story that brought incredible joy to me.

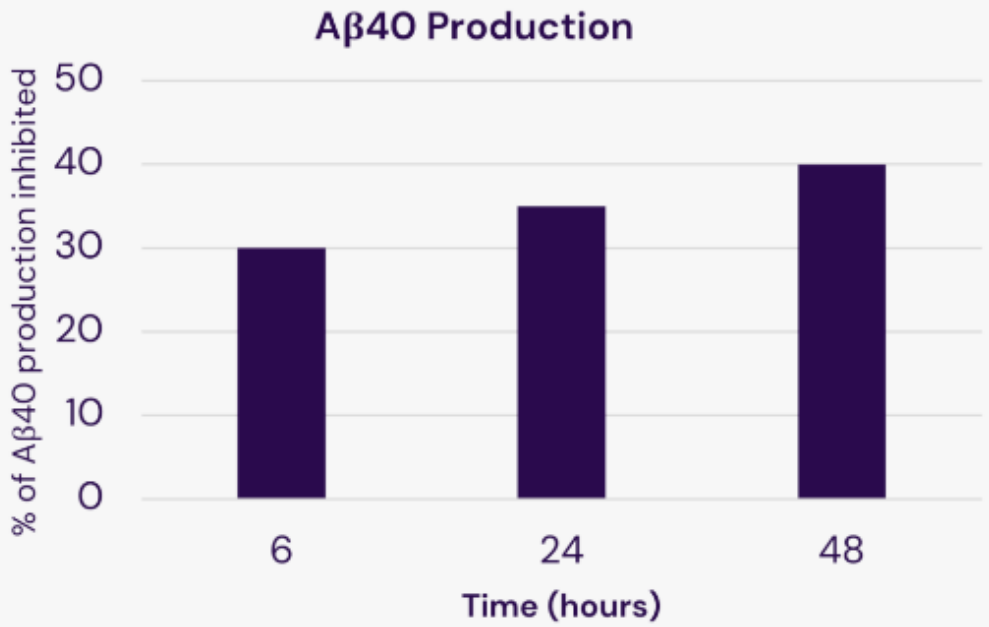
We participated in your Phase 1Clinical trial in Puerto Rico. After about a week of medication, one day I came home to find that she had made dinner and was dancing to music that she enjoys.

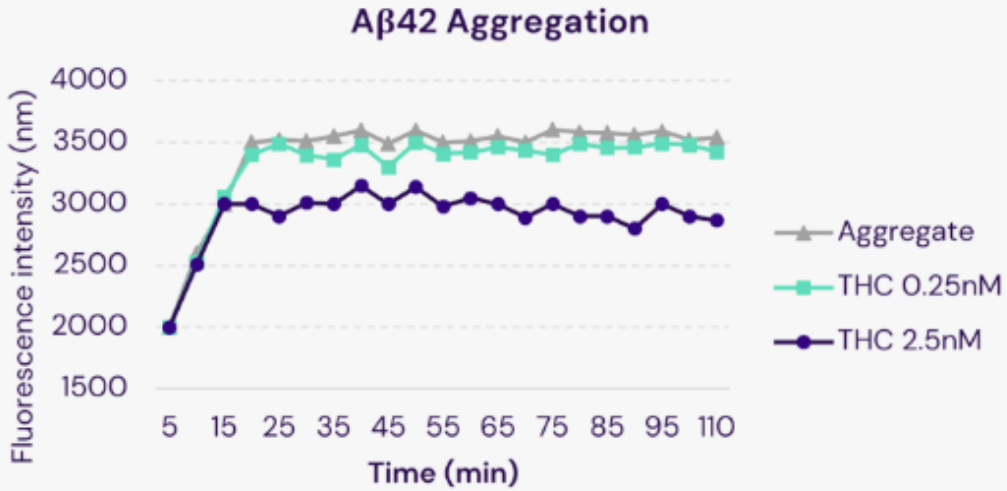
**That night she was coherent, was not agitated, and for a brief period she was almost her normal self.** I also contacted IGC Pharma, because of the good benefits my mother had during her participation in the trial and to see if I could continue to get the medication."

Marcial D.

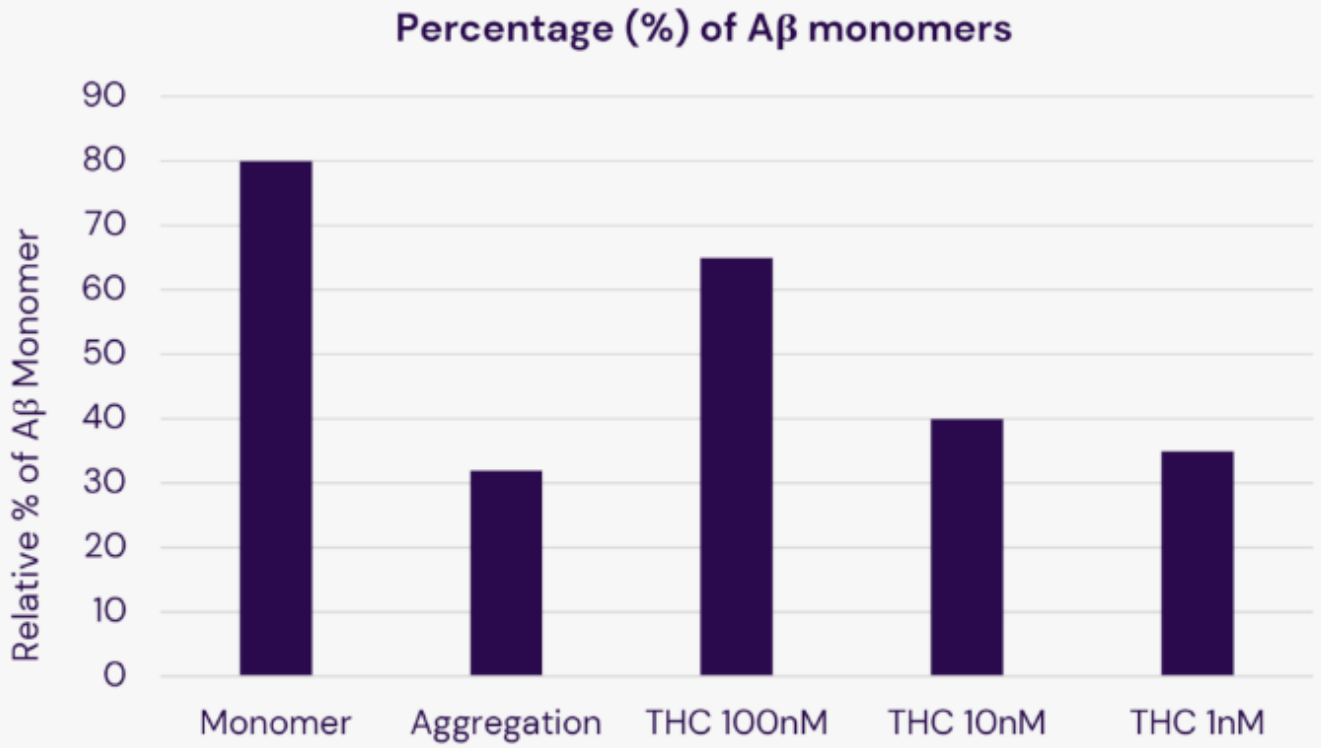
# Pre-clinical studies: Detailed scientific results

The API in IGC-AD1 reduces Aβ40 peptide production and Aβ42 aggregation in Alzheimer's cell lines.



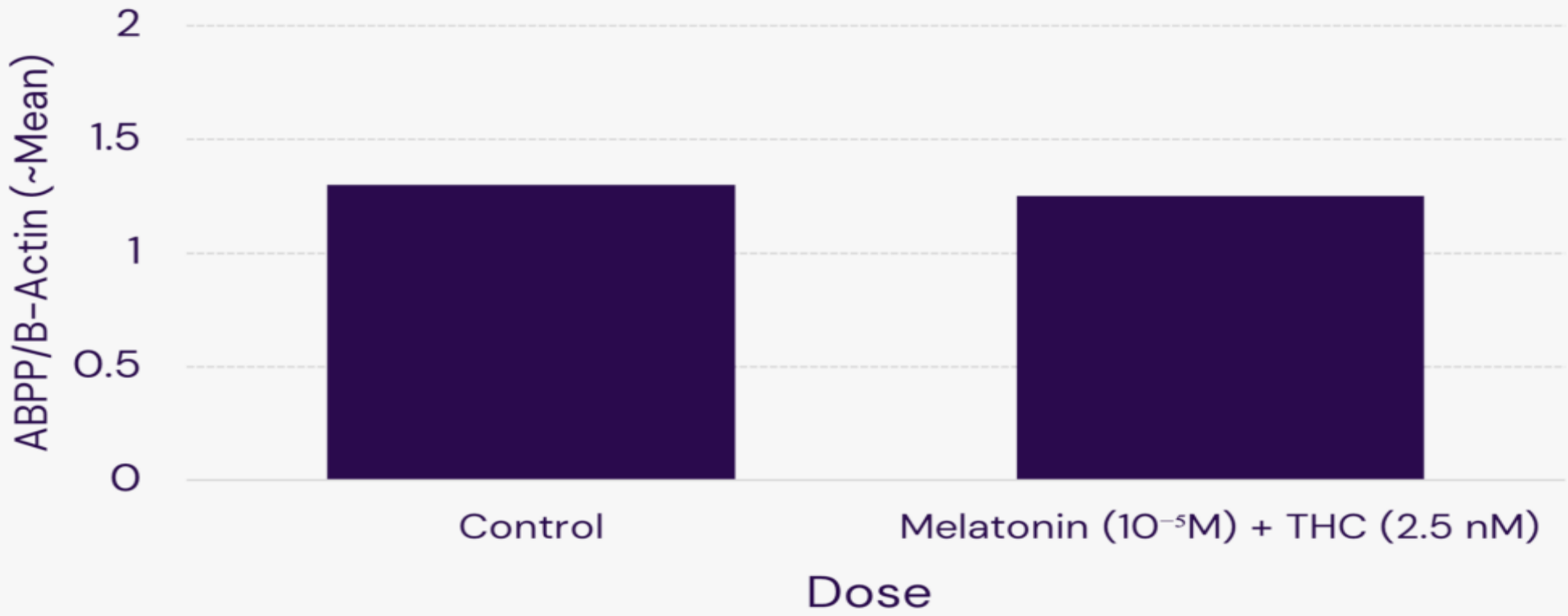



Aβ Production & Aggregation | **Aβ Monomers** | APP Levels | Spatial Memory | Neurotoxicity



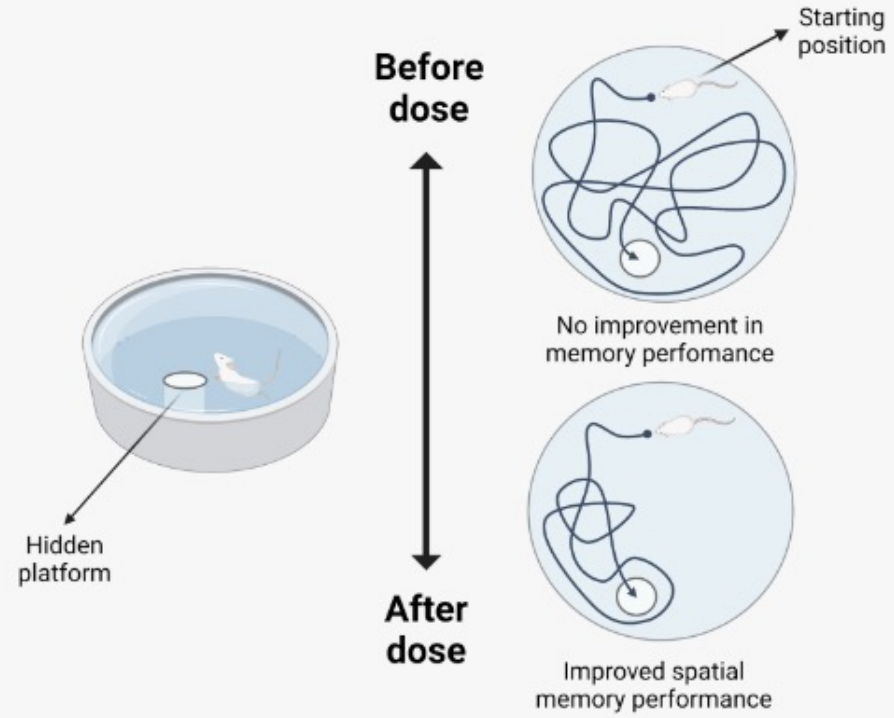

In Alzheimer's cell lines, IGC-AD1 increased Aβ monomers and decreased Aβ aggregation in a dose–dependent manner.

The APIs in IGC-AD1 did not reduce Amyloid Precursor Protein (APP) levels in Alzheimer's cell lines. APP modulates cell growth, motility, and survival; it is cut to create small fragments such as the Aβ peptide that eventually deposit as plaque.




Representation of Cao et al., 2014

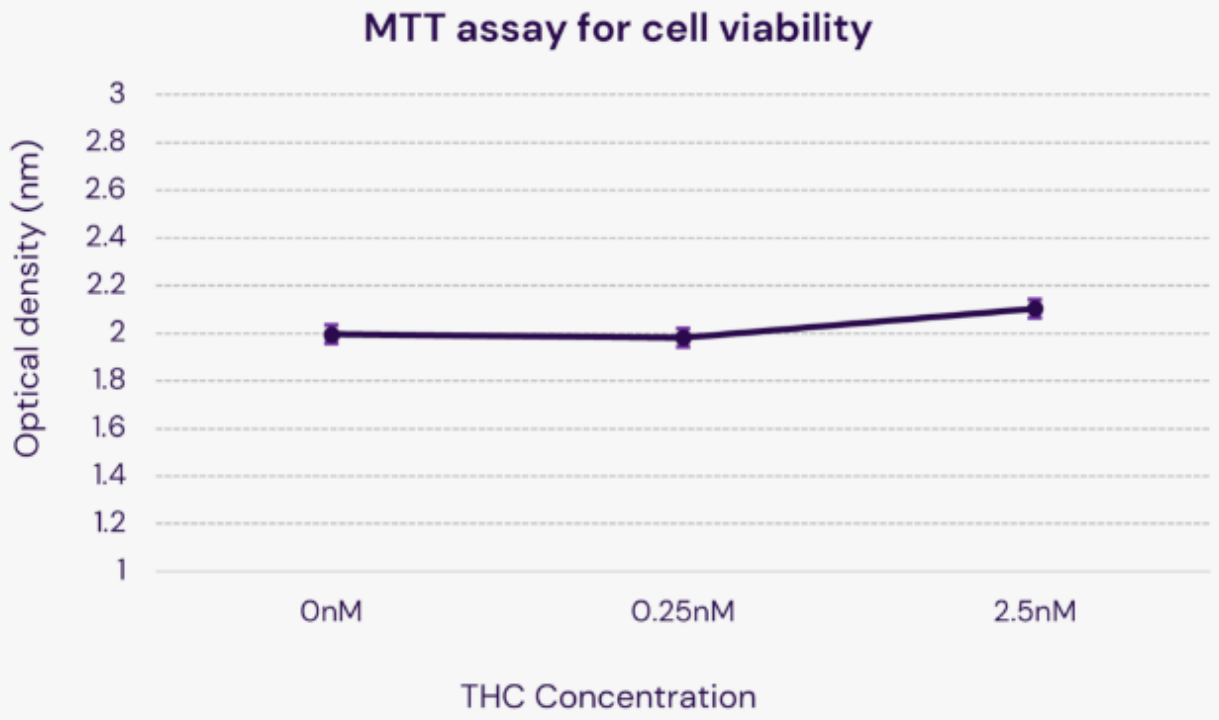
Aβ Production & Aggregation | Aβ Monomers | APP Levels | Spatial Memory | Neurotoxicity




# Memory Improved in Alzheimer's Mice Model

In a Morris Water Maze test, mice dosed with the API in **IGC-AD1** had significantly improved times and less errors than those in the control group demonstrating **that memory improved in transgenic (APP/PS1) mice.**

Nature Protocols. 2006; 1: 848-858; Int. J. Mol. Sci. 2022, 23, 2757




Over 48 hours, repeated low-dose exposure to the API in IGC-AD1 was not toxic to Alzheimer's cells (N2a/AßPPsWe cells).

Representation of Cao et al., 2014

# TWO APIs THAT TARGET MULTIPLE PATHWAYS

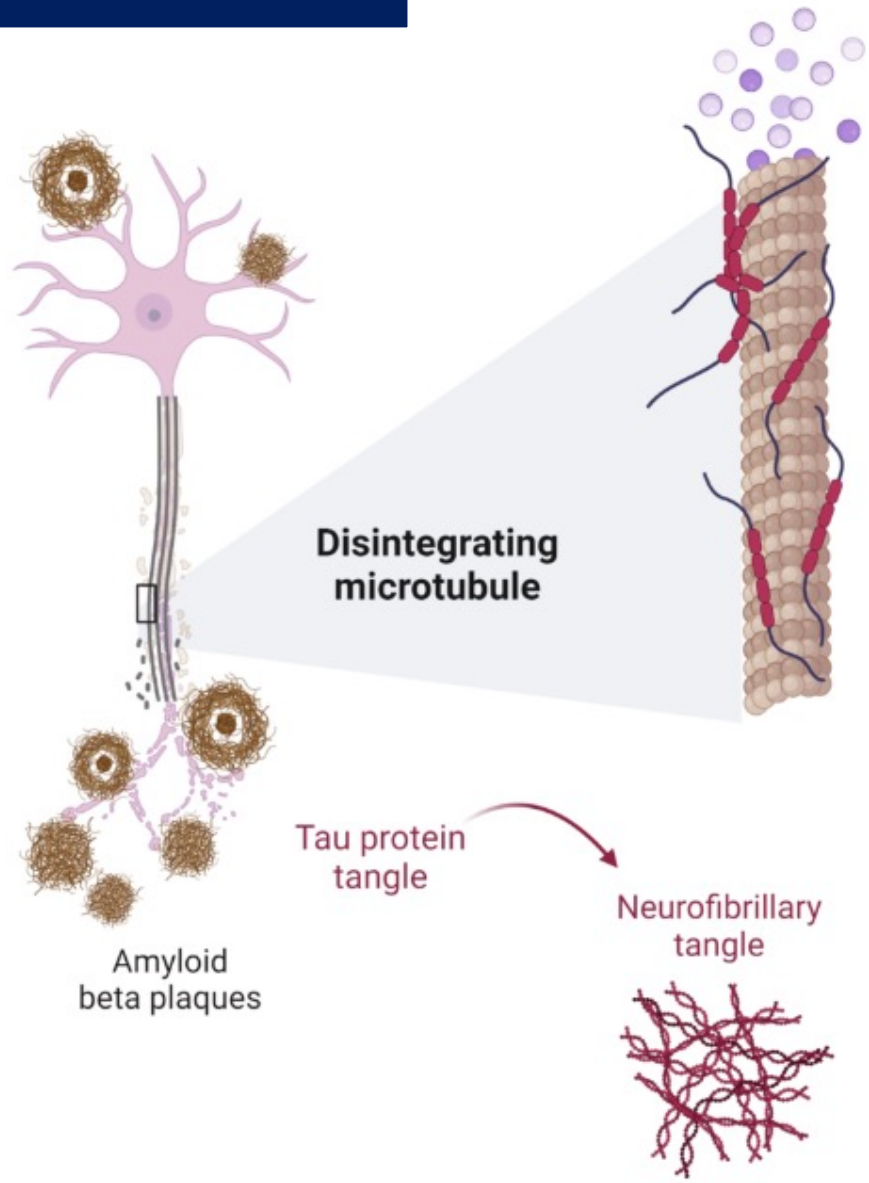
## Focused on relieving pathological alteration due to agitation in AD

| Agitation in AD | | Treatment with IGC-AD1 |
|---|---|---|
| Deficient in CB1 receptor function increases agitation animals[1] ; CB1 activity at earlier AD stages is higher in hippocampal areas and internal layers of frontal cortex Decreases at the advanced stages[2] | → | **Partial agonist targets CB1 receptor function[5]** |
| Inflammation in the medial temporal region as agitation develops[3] | → | **APIs mediate anti-inflammatory actions, including inhibition of inflammasome-3** |
| Neurotransmitter imbalance[4] | → | **APIs target CB1r activation that corrects the release of GABA and glutamate[7]** |

**References**: (1).Marta Rodriguez-Arias et al., 2013. (2).Manuel et al 2014. (3). Yasuno et al., 2023 (4). Liu et al., 2018 (5). Kendalll and Yudowski 2017 (6). Kozela et al., 2009 (7). Hoffman and Luoica 2013

# PRECLINICAL DATA

## Pre-clinical study results




The pre-clinical studies tested the API in IGC-AD1 on Alzheimer's cell lines and Alzheimer's mouse models and found that it had the potential to be a disease modifying drug that could:

- **Inhibit the formation of neurofibrillary tangles**
- **Inhibit the formation of plaques.**
- **Enhance mitochondrial functioning.**
- **Improve spatial memory.**

**For more detailed scientific results, click here >**

https://igcpharma.com/igc-ad1/#results