



Addressing Neuro-Inflammation through Therapeutic Intervention

Investor Overview
October 2025



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Coya Therapeutics: Investment Highlights



Novel Therapies, High Unmet Need

- Differentiated approach targeting Treg dysfunction through combination therapies
- Targeting high unmet diseases ALS, FTD, Alzheimer's Disease
- De-Risked approach targeting diseases (ALS, FTD) with regulatory flexibility
- **Phase 2 study in patients with ALS- First Patient Dosing in 4Q 2025**

High Commercial and Value Creating Potential

- COYA-302: "A Pipeline Within a Product" with > \$10B Potential
- COYA-303- Potential to create value for existing GLP-1 Agonists

Strong Cash Runway, Clean Cap Table

- Cash ~\$29.8 million as of 06/30/2025 with runway into 2H 2026 , no debt, 16.7M Shares Outstanding
- Strategic partnership with Dr. Reddy's (RDY) with potential ~\$700M deal value - Steady line of sight to non-dilutive cash (\$4.2 M received in August upon IND acceptance, additional \$4.2M anticipated in 4Q 2025)

Accomplished Management Team and Board

- Proven track record of execution and value creation

A Large and Global Problem

Neurodegeneration is **affecting millions** of people **without clear therapeutic pathways**.
1 in 3 people will be affected by neurological conditions in their lifetime.

50 Million+
People Impacted
Worldwide

\$9 Trillion
Global Cost of
Dementia by 2050

3-5
Years
Life Expectancy
After ALS Diagnosis

What are Neurodegenerative Diseases?

Neurodegenerative diseases are progressive conditions that impair bodily function - impacting your movement, memory, or thinking. They include Amyotrophic Lateral Sclerosis (ALS), Alzheimer's Disease, Parkinson's Disease, and Frontotemporal Dementia (FTD)

Our Pipeline is > \$10Bn Opportunity

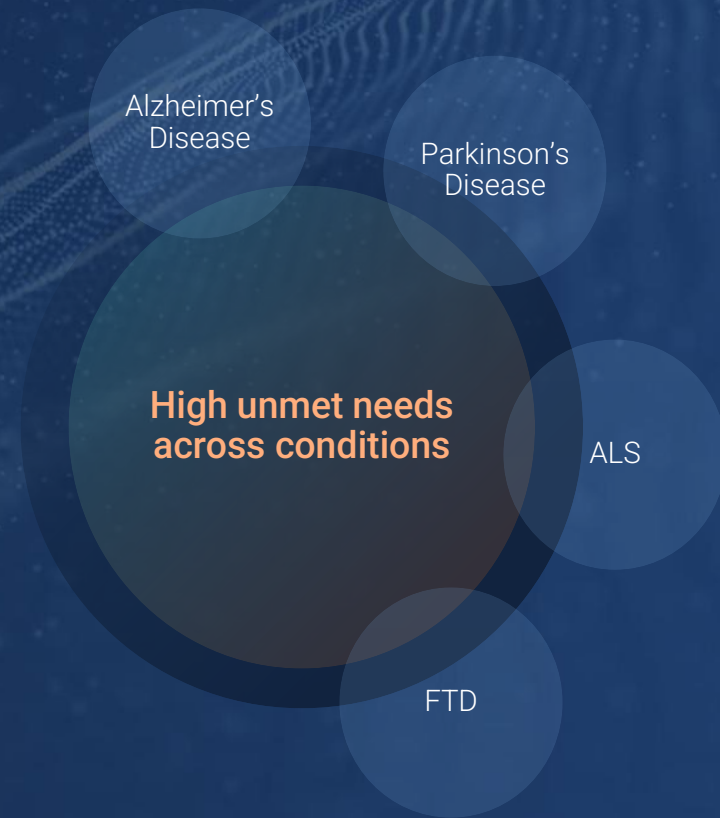
Coya is prioritizing therapies for ALS, FTD, AD, and PD as key initial neurodegenerative diseases

ALS and Frontotemporal Dementia

ALS and FTD both have high unmet need, are designated as orphan indications, and have flexible and fast paths to regulatory approval if shown to be effective.

Alzheimer's and Parkinson's

AD and PD represent the first and second most common neurodegenerative conditions and have high unmet need. Both conditions present a > \$5Bn sales potential each.



The Coya Solution

We will address neurodegeneration at its source by developing innovative therapies to meet the needs of patients with these diseases.



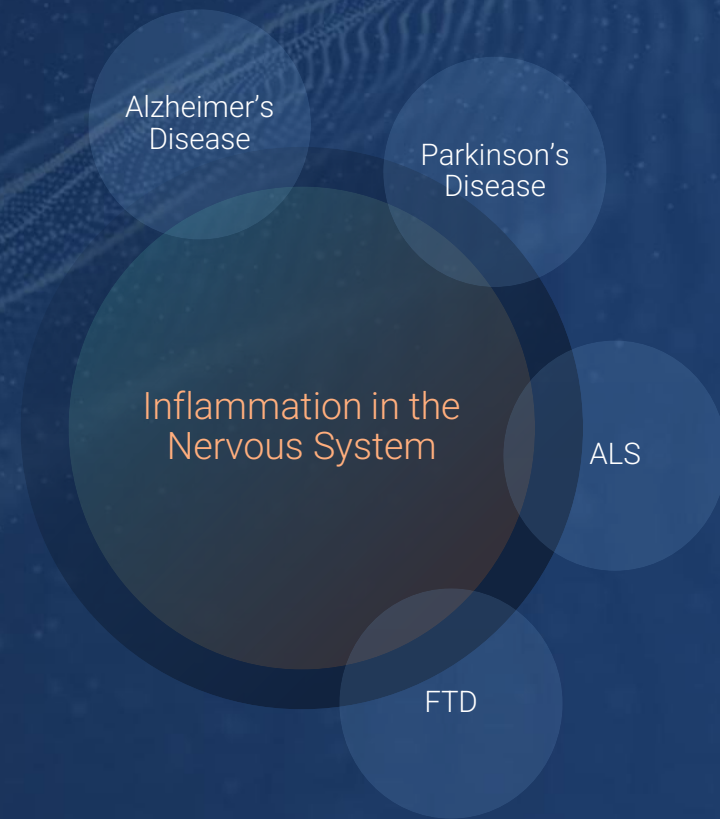
Combining Therapeutic Approaches

Neurodegenerative disorders impact people in a variety of ways. There are clear therapeutic benefits in focusing on the common underlying characteristics of these diseases, to develop a comprehensive treatment that benefits a range of patients.

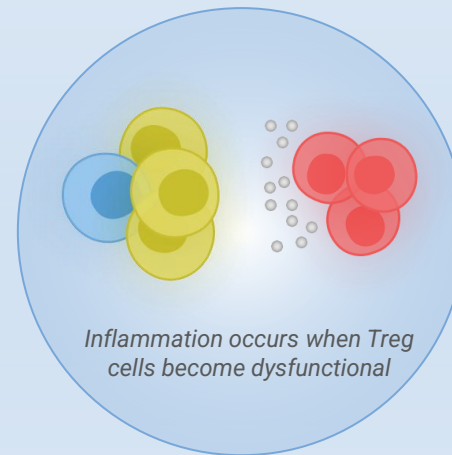
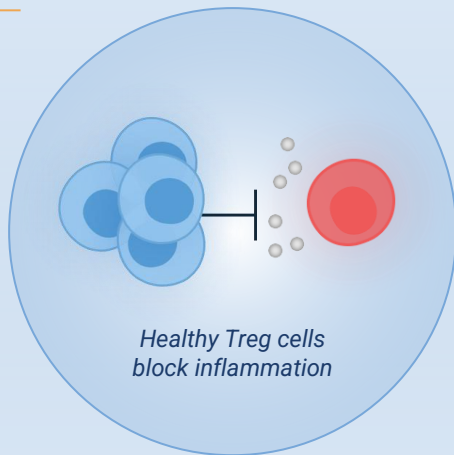


Harnessing Tregs

Tregs manage your immune system's response to sickness, and play a key role in helping you feel better. In applying our knowledge of Tregs, we will empower patients' internal first responders with support to tackle these diseases.



Intro to TRegs



What are Tregs?

Tregs are cells within the body that keep the immune system in check. When functioning properly, they allow the immune system to function appropriately.

How do Tregs influence disease?

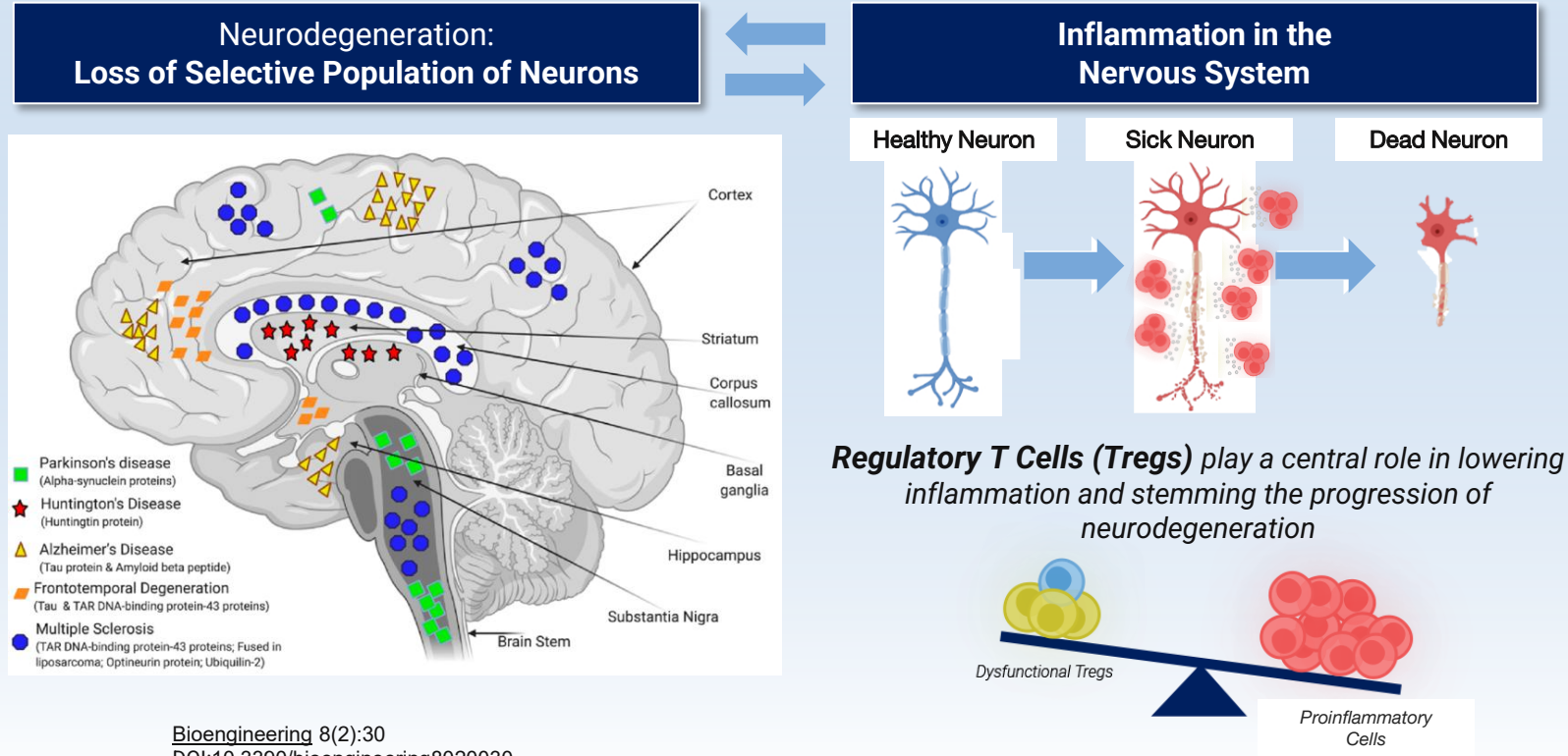
When Tregs do not function properly, autoimmune and neurodegenerative diseases can develop and progress.

How to treat disease by targeting Tregs?

Coya is focusing on restoring Tregs to their normal functional state using combination therapies to potentially treat incurable neurodegenerative diseases.

Inflammation - A Critical Role in Neurodegeneration

Regulatory T cells (Tregs) are Dysfunctional; COYA aims to repair these cells



2025 Key Catalysts and Milestones

Additional Clinical Data Release: Phase 2 IIT AD Trial	➤ 1Q 2025: Phase 2 LD IL-2 investigator initiated trial in patients with AD additional clinical data release: Publication and release of additional and comprehensive systemic immune panel and inflammatory cerebrospinal fluid (CSF) biomarkers comparing LD IL-2 arms to placebo arm
COYA 303 Data Release	➤ 1Q/2Q 2025: COYA 303 combination mechanistic data publication and additional IP filings
COYA 302 Phase 2 ALS Trial Initiation	➤ 2Q 2025: Submission of the additional data package to support the start of the COYA-302 Phase 2 trial in patients with ALS ➤ IND Accepted (August 2025): Triggered \$4.2 M non-dilutive milestone payment from strategic partner, Dr. Reddy's Laboratories (DRL) ➤ Upon first patient dosing: eligible to receive non-dilutive milestone payments of \$4.2M from strategic partner, Dr. Reddy's Laboratories (DRL)
ALS Biomarker Data Release	➤ 2H 2025: ALS Biomarker data publication of longitudinal data on Neurofilament Light Chain (NfL) and oxidative stress markers in ALS Patients
FTD Clinical Data Release and IND	➤ 2H 2025: Top Line Clinical Data of investigator-initiated trial combining LD IL-2 + CTLA4-Ig in FTD Patients ➤ 2H 2025: File COYA-302 IND for Phase 2 FTD Trial



LEAD PRODUCT

COYA 302

Proprietary, Recombinant Human
Low Dose Interleukin-2 (LD rhIL-2)
and CTLA4-Ig



COYA 302 IND accepted to support the initiation of a Phase 2 multicenter, double-blind, placebo-controlled clinical study to evaluate the efficacy and safety of COYA 302 in patients with ALS

Trial Initiation

**Anticipated First
Patient Dosing: 4Q
2025**

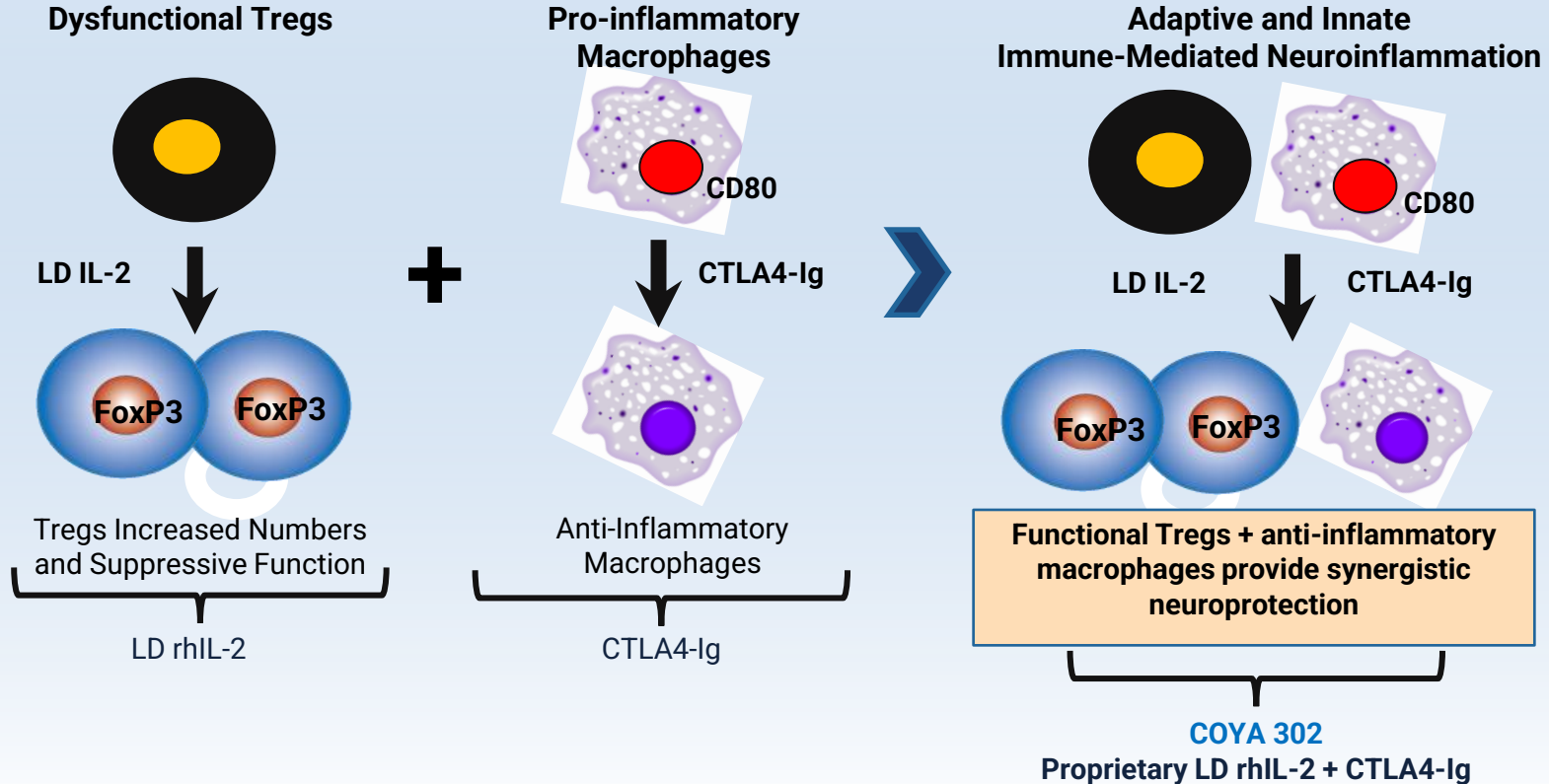
NEALS-affiliated Study

NEALS, the world's largest ALS research consortium, has selected the COYA 302 trial and it is now affiliated with NEALS as part of its mission to accelerate the development of new treatments for people living with ALS;

The COYA 302 study was presented during the NEALS Educational Webinar on September 29, 2025.

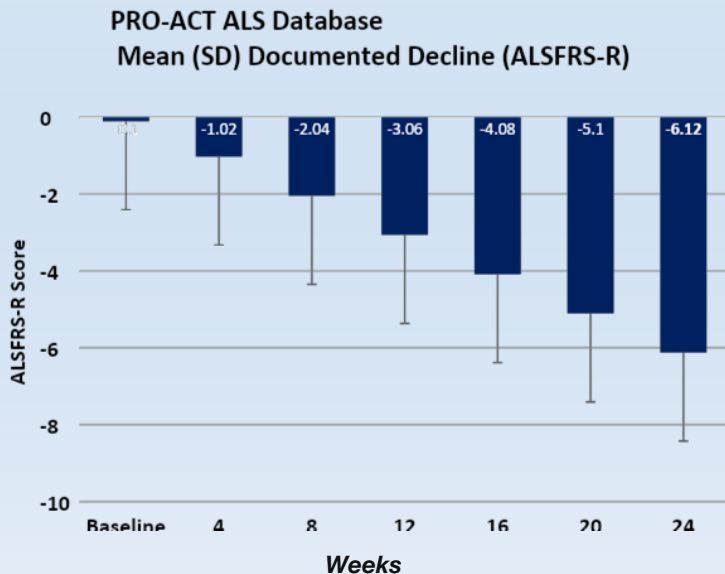
The Future of Neurodegenerative Disease Therapy

COYA 302 (proprietary LD rhIL-2 + CTLA4-Ig Combination Immunotherapy)

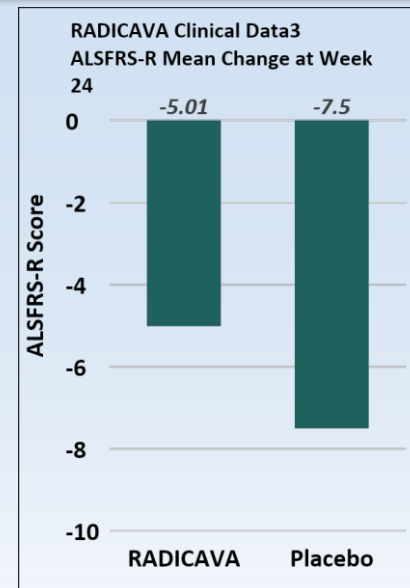
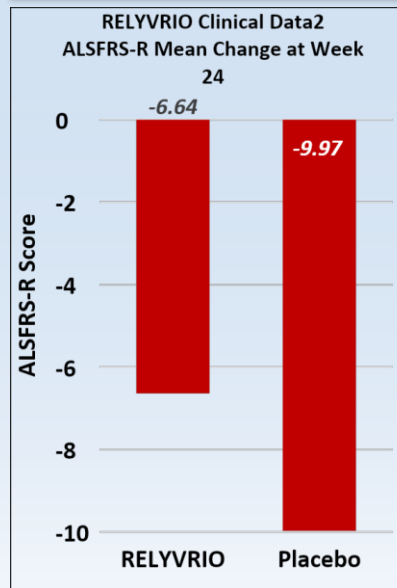


Current Therapies for ALS aim to slow disease progression

Average rate of patient decline is 1.02 points/month in ALSFRS-R score¹



Many companies have garnered significant value by demonstrating a *limited* benefit of slowing the rate of ALS progression



1. The PRO-ACT database is the largest ALS data repository (Atassi et al, 2014)

2. Relyvrio US Prescribing Information (9/2022)

3. Radicava US Prescribing Information (5/2022)

LD IL-2 + CTLA4-Ig Investigator Initiated Clinical Trial

Open-Label, Single-Arm PoC Clinical Study in ALS Patients (N=4)

Screening

20 weeks

Treatment Period

LD IL-2 + CTLA4-Ig was administered via subcutaneous injection over 48 weeks

Follow-Up

8 weeks

Screening Assessments

- ✓ Clinical Labs
- ✓ ALSFRS-R Score
- ✓ Electrocardiogram (ECG)
- ✓ Physical & Neurological Exam

Study patients had well-documented disease progression prior to treatment (-1.1 points/month prior to treatment with COYA 302)

Treatment Period Assessments

- ✓ Safety and Tolerability
- ✓ Treg Function and Numbers
- ✓ Serum Biomarkers
- ✓ ALSFRS-R Score

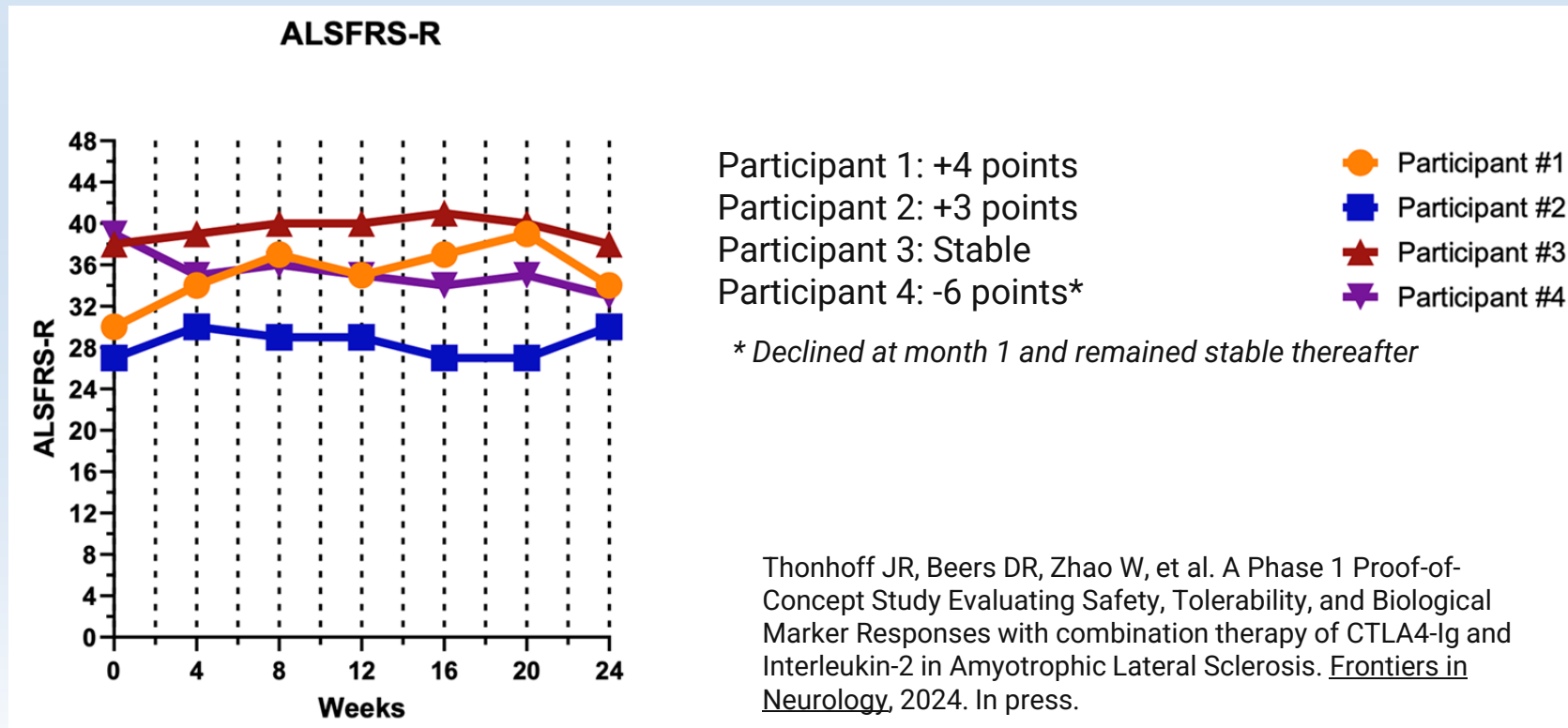
Safety and tolerability assessments included reported adverse events, periodic physical and neurological exams, clinical labs, and ECGs

Post-Treatment Assessments

- ✓ Safety and Tolerability
- ✓ Treg Function & Numbers
- ✓ Serum Biomarkers
- ✓ ALSFRS-R Score

LD IL-2 + CTLA4-Ig Investigator Initiated Clinical Trial

ALSFRS-R Individual Scores remained mostly stable or improved



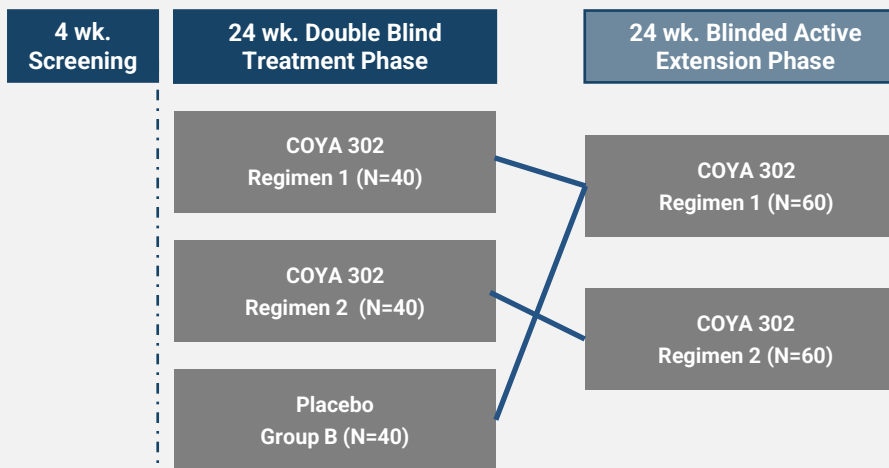
Overview of Phase 2 Study Design in ALS

Trial is active and currently enrolling participants

Key Inclusion Criteria

- Diagnosis of sporadic or familial ALS
- Time since onset of ALS symptoms $\leq$ 24 months from Screening
- ALSFR-R score $\geq$ 35 at Screening
- A score of at least 2 points in each ALSFRS-R item
- Slow vital capacity (SVC) $\geq$ 70% of predicted capacity for age, height, and gender at Screening
- Documented disease progression between -0.5 and -1.5 points per month on ALSFR-R total score

Randomized, Double-Blind, Placebo-Controlled Phase 2 Study with Blinded Active Extension



Primary Endpoint

Change in disease severity over time measured by ALSFRS-R total score from baseline to Week 24 vs. placebo

Study Objectives

1. Efficacy
2. Safety and Tolerability
3. Biological Activity
4. Biomarker levels

Accelerating Research to Commercial

Commercial Growth enabled by Strategic Partnerships

With an already solidified partnership with Dr. Reddy's for Coya 302 for ALS, partnerships will be catalysts for business growth, regulatory approvals and clinical adoption



Research agreements in place to ensure preclinical data is well defined and ready for trial



Leveraging DRL's global footprint to accelerate regulatory and expand global commercial footprint



COYA 302 in FTD

Proprietary, Low Dose Recombinant
Human Interleukin-2 (LD rhIL-2)
and CTLA4-Ig

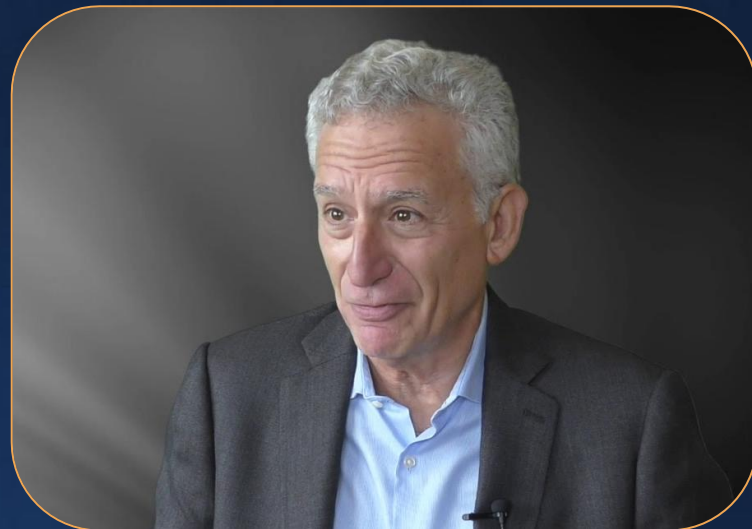


Investment by ADDF

“Inflammation has emerged as a promising novel pathway for chronic neurological diseases like FTD. A combination drug, like COYA 302, is an innovative approach being developed to suppress neuroinflammation by targeting multiple inflammatory pathways. Combination therapy will be integral to slowing – and eventually halting – cognitive decline for a disease as complex as FTD, and exploring combined therapeutic modalities is an important advancement in the development of future care regimens.”

Howard Fillit, MD

Co-Founder and Chief Science Officer
Alzheimer's Drug Discovery Foundation



Press Release: May 20th, 2024 following announcement of a \$5M strategic investment in Coya Therapeutics

Partnership with Alzheimer's Drug Discovery Foundation

\$5M Investment to accelerate Phase 2 trial in FTD



Alzheimer's
Drug Discovery
Foundation

Study Title

A Phase II, Randomized, Double-Blind,
Placebo-Controlled, Multi-Center, 26-Week
Study to Evaluate the Safety and Efficacy of
COYA 302 for the Treatment of Nonfluent
Variant Primary Progressive Aphasia
(nfvPPA) Subtype of Frontotemporal Lobar
Degeneration (FTLD)

Frontotemporal Disorder (FTD) is a Rare Disease and is one of Most Common Dementias in Younger People. Frontotemporal disorder progresses to death faster than Alzheimer's disease, and, unfortunately, there is no effective treatment

SYMPTOMS OF FTD



Changes in personality,
mood and social.



Language, speech and
communication problems.



Deteriorating problem-
solving skills



Difficulty planning,
organizing, staying focused
or completing tasks.



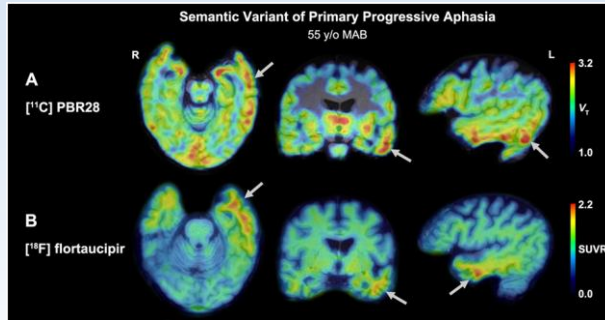
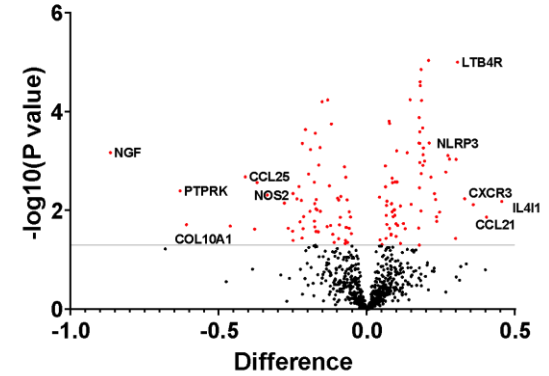
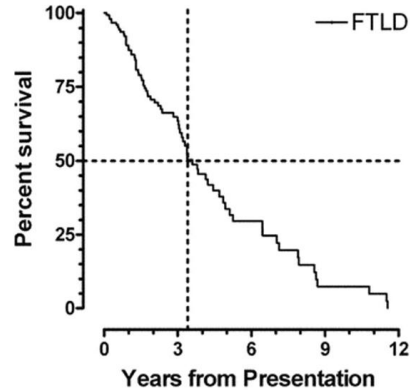
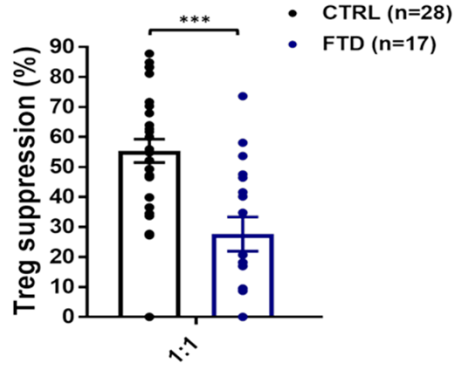
Poor financial
judgement.



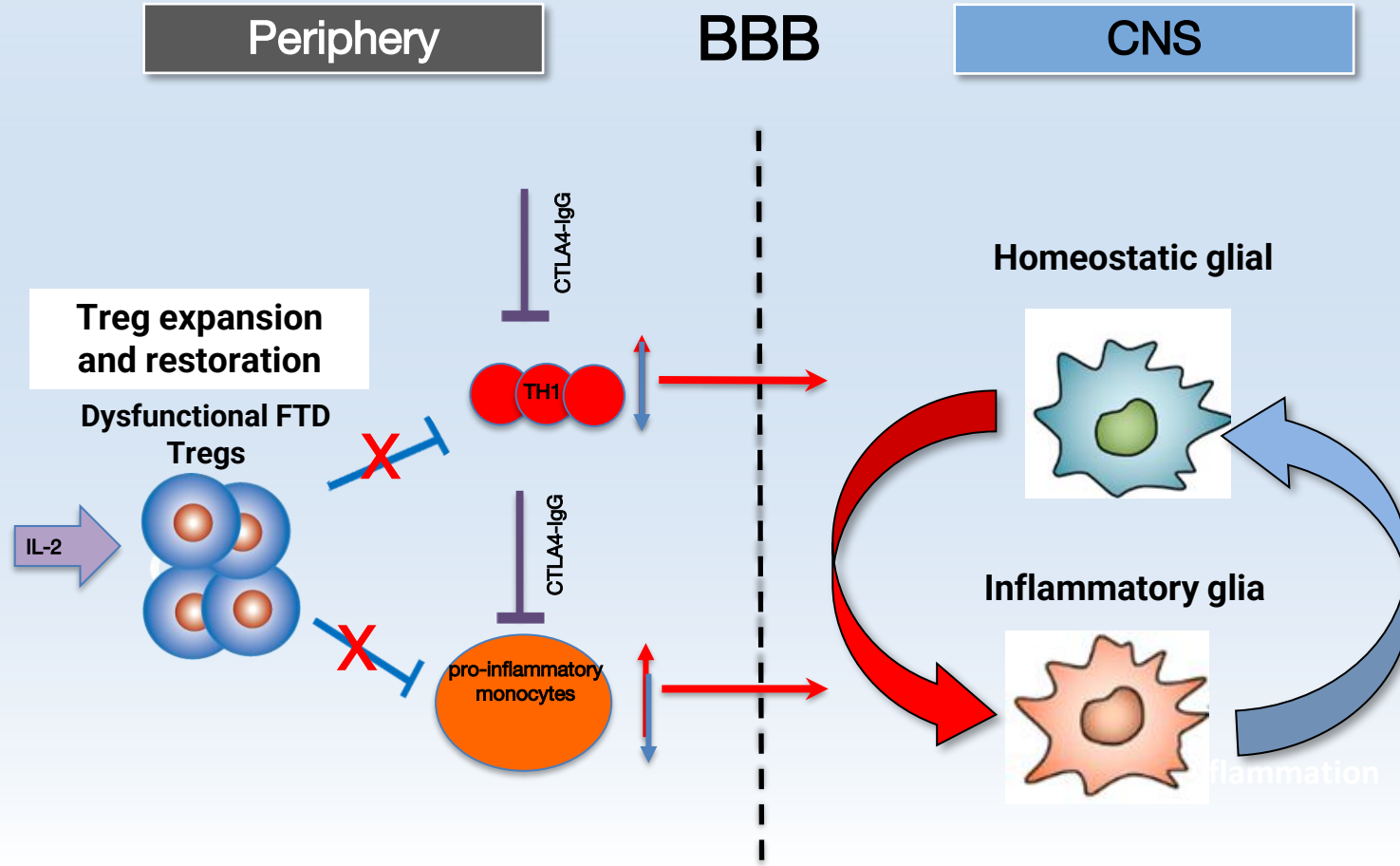
Balance issues, frequent falls,
trouble walking, poor
coordination, tremors or shaking.

Exacerbated Systemic and Central Inflammatory Responses

FTD is a devastating disease that unfortunately has no effective treatment



Targeting Inflammation in FTD to Slow Disease Progression



COYA 302 FTD Phase 2a Study

Houston Methodist Center is a CoE in ALLFTD - a single infrastructure clinical data platform in FTD with collaborative decision making among 5 leading centers around the USA



Eligibility Criteria

- Non-Fluent Primary Progressive Aphasia Subtype
- Global Clinical Dementia Rating - Frontotemporal Lobar Degeneration (CDR® plus NACC FTLD) score of 0.5 or 1

Primary Endpoints

- CDR+NACC-FTLD-SB*
- Neuropsychological tests
- NfL
- MRI Volume

*Draft Design to be finalized

* CDR® Dementia Staging Instrument PLUS National Alzheimer's Coordinating Center (NACC) Behavior and Language Domains for FTLD - sum of boxes

Additional Pipeline Assets

Product Type	Discovery	Preclinical	IND-Enabling	Phase 1	Phase 2	Phase 3	Key Milestones
Coya 301 <i>Proprietary Low Dose IL-2</i>	COYA 301 in AD						1H 2025: Additional biomarker and blood panel data from the academic IIT double-blind Ph2 data that was presented at CTAD in October 2024
Coya 303 <i>COYA 301 + GLP-1 Agonist</i>	COYA 303 Undisclosed						1H 2025: Additional IP filings Publish preclinical in-vitro data Initiate preclinical translational study Partnership discussions
Coya 201 <i>Allogeneic Treg Derived Exosomes</i>	COYA 201 Undisclosed						2025: Pre-clinical characterization
Coya 206 <i>Antigen-Directed Allogeneic Treg-Derived Exosomes</i>	COYA 206 Undisclosed						2025: Pre-clinical characterization



PIPELINE PRODUCT
COYA 301

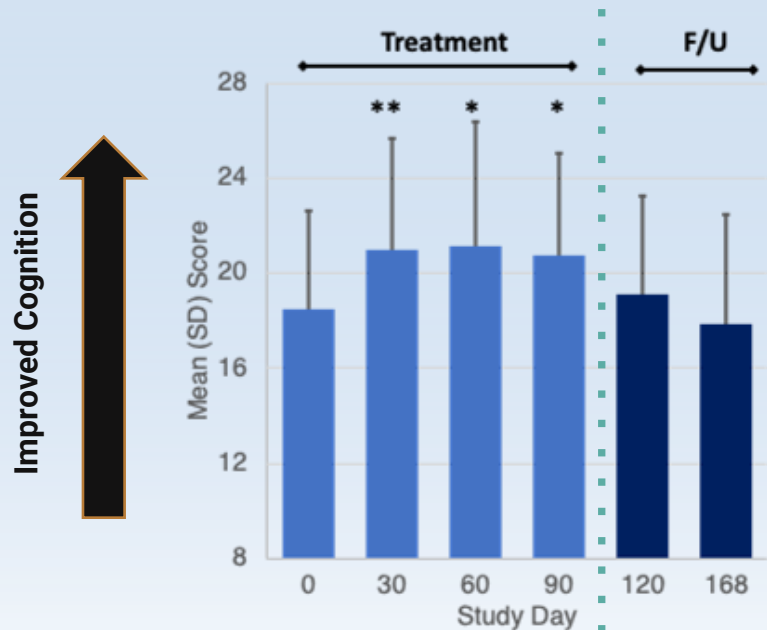
Proprietary Low Dose
Recombinant Human IL-2



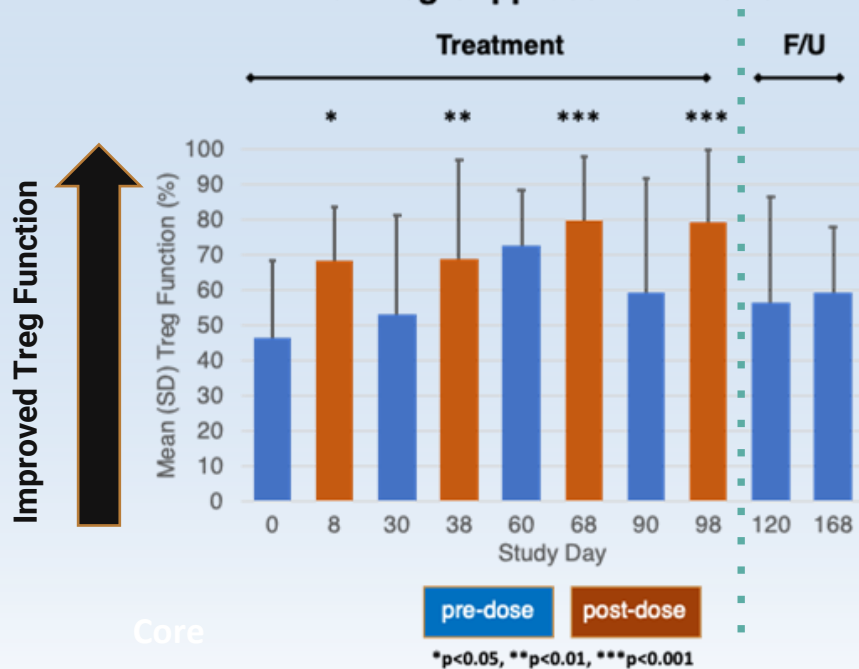
Phase 1 Proof of Concept Open-Label Trial with LD IL-2 in AD

Enhanced Treg Function and halted Cognitive decline

MMSE Score (N=8)



Statistically Significant Enhancement of Treg Suppressive Function



Case Study of Brain Imaging of AD Patient Before and After LD IL-2 Treatment

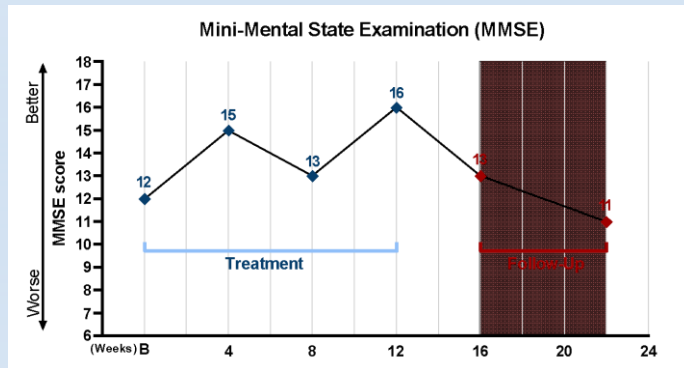
70-Year-Old Male

Baseline MMSE: 12

Positive PIB PET Scan

Positive Neurodegeneration MRI

Other Rx: Donepezil 10mg



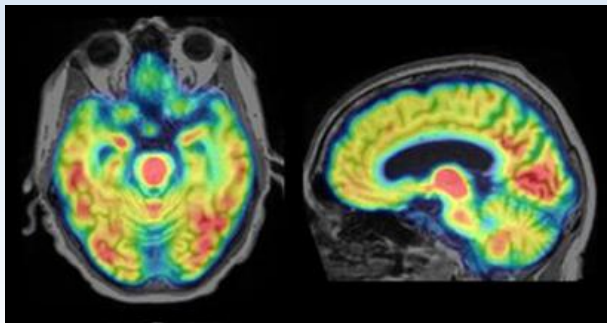
Neuroinflammation (TSPO)

(¹¹C) ER-176 PET Scan at baseline and after 4-monthly cycles of low dose IL-2.

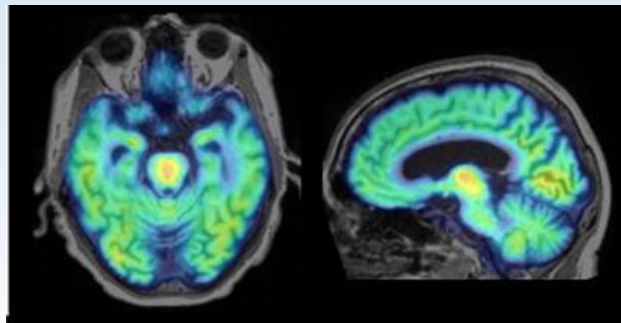
The second PET scan was done 2 weeks after last dose.

PET: Positron Emission Tomography
TSPO: translocator protein

PET Scan (Baseline)

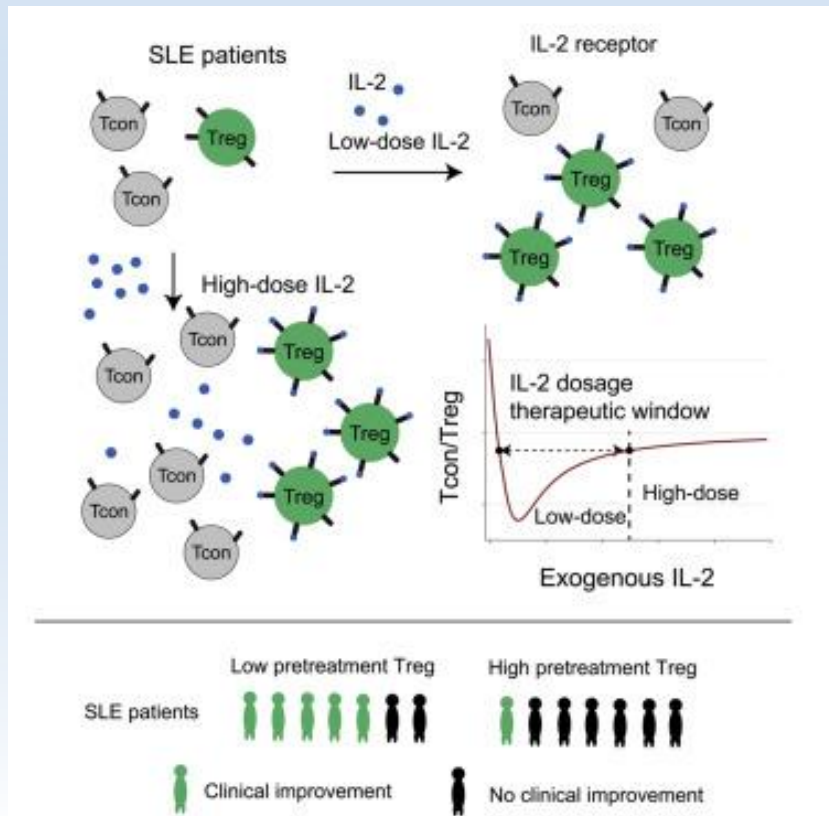


PET Scan (2 weeks after last dose)



A Primer on IL-2 Dosing and Treg Function

Lower IL-2 Dose is Better for Tregs



Study Summary

Previous studies have shown that lower IL-2 doses selectively enhance and increase Treg function reducing pro-inflammatory T Cells while higher IL-2 doses reduce Treg function and enhance pro-inflammatory cells



COYA 301 in AD

Phase 2 Low Dose Interleukin-2 (LD IL-2) in Patients
with Mild to Moderate Alzheimer's Disease

An Investigator Initiated, Randomized, Double-Blind,
Placebo-Controlled Clinical Trial led by Dr. Alireza
Faridar at Houston Methodist Hospital



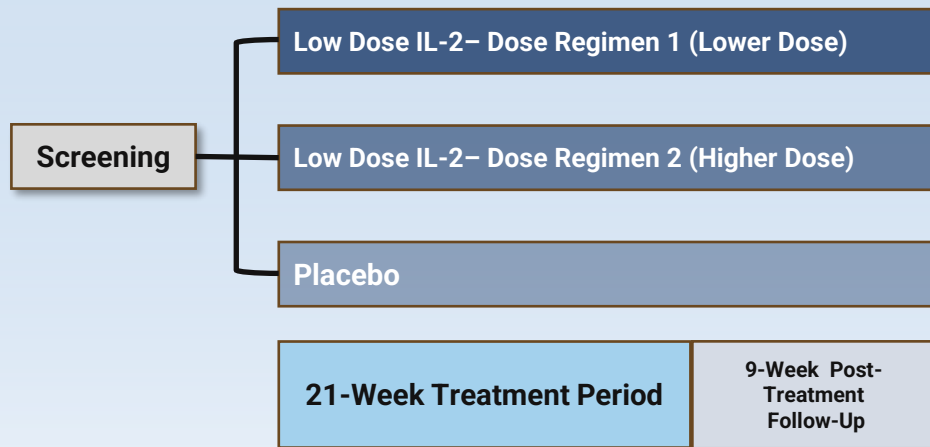
Double-Blind, Placebo-Controlled Study in AD Patients

Study funded by the Gates Foundation and Alzheimer's Association

The encouraging results of the open-label, proof-of-concept study in AD (N=8) supported conducting the ongoing randomized, double-blind, controlled Phase 2 study in 38 patients with mild-to-moderate AD.

Study Objectives

- Safety & tolerability
- Biological activity (Treg function)
- Blood and CSF biomarkers
- Cognitive function



Dose Regimen 1: 1 million IU administered SC daily for 5 days, **every 4 weeks**
Dose Regimen 2: 1 million IU administered SC daily for 5 days, **every 2 weeks**

LD IL-2 Meets Primary and Secondary Endpoints for Potential Treatment of Alzheimer's Disease (AD)

✓ **Cognition Stabilization (Exploratory Endpoint) for Lower IL-2 Dose Through Drug Treatment vs. Placebo:**

- 2 out of 3 scales showed cognitive stabilization (ADAS-Cog14 and ADCS-CGIC)* for the Administering 5-day LD IL-2 cycles every 4 weeks (LD IL-2 q4wk) regimen.
- On day 148, ADAS-Cog14 scores indicated a slight improvement of -0.450 points from baseline for the LD IL-2 administered every four weeks, compared to a worsening of 4.480 points from baseline in the placebo group. The 4.93 point Δ suggests a clinically meaningful treatment effect.
- For patients with mild AD, a change of +3 on the ADAS-Cog has been described as clinically meaningful to assess worsening (Muir et al., Alzheimer's Dement. 2024;20:3352–3363).

✓ **Safety (Primary Endpoint) and Regulatory T cell (Treg) Enhancement (Secondary Endpoint) Validated:**

- Both dosing frequencies regimens of LD IL-2 studied (q4wk & q2wk) were safe and well-tolerated.
- Administering LD IL-2 q4wk effectively, sustainably, and significantly expanded Treg numbers and function vs. placebo without off-target effects on other peripheral lymphocytes.

✓ **Significant Improvement in Amyloid Beta Pathology for Lower IL-2 Dose vs. Placebo:**

- LD IL-2 q4wk significantly improved CSF Soluble A β 42 levels (an index of amyloid pathology) vs. placebo.
- LD IL-2 among the first double-blind human trials to document subcutaneous Treg-enhancing therapy that modifies beta-amyloid.

✓ **Cerebral Spinal Fluid (CSF) Biomarker and Cognitive Effects Were Associated with Boosted Treg Function and Numbers vs. Placebo**

*The Alzheimer's Disease Assessment Scale–Cognitive Subscale (ADAS-Cog) was developed in the 1980s to assess the level of cognitive dysfunction in Alzheimer's disease. Clinical Global Impression of Change (CGIC) scales have been used as primary outcomes in phase 2 and 3 clinical trials for Alzheimer disease, mild cognitive impairment, and for cognitive enhancers. A CGIC score is intended to be used as a measure of clinically meaningful change, as distinct from an instrument's ability to assess any change.

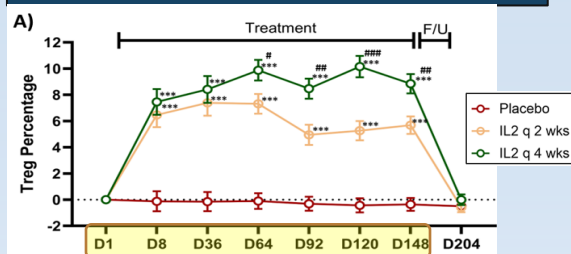
LD IL-2 in Mild to Moderate Alzheimer's Disease

The trial was designed to detect significant differences in primary and secondary endpoints, and validated that Lower Dose IL-2 is Better

Primary Endpoint: Safety and Tolerability

- ✓ No Serious Treatment Related Adverse Events Reported
- ✓ All Patients Completed Trial
- ✓ Drug was Well Tolerated

Secondary Endpoint: Treg Cell Populations

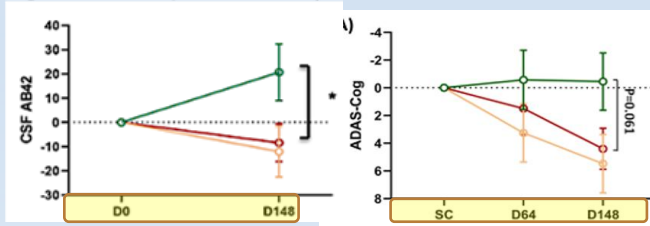


- ✓ Significant change in Treg population following two different dosing frequency regimens of LD IL-2 treatment, compared to placebo

Lower Dose IL-2 Level
Higher Dose IL-2 Level

: 21 week or 148 day on-treatment period

Exploratory Endpoints: CSF Biomarkers and Clinical Scales



- ✓ Significant improvement in cerebrospinal (CSF) AD-related biomarker Soluble AB42

- ✓ Assessment of peripheral/central inflammatory responses will be reported in the near future)

- ✓ Clinical scales, including the ADAS-Cog Score

Key Takeaways of the Trial

Trial validated that lower dose IL-2 is better

Key Takeaways:

- We believe LD IL-2 study results further validate the hypothesis that restoring the numbers and function of Tregs with systemic LD IL-2 alone targets neuroinflammation
- We believe LD IL-2 study results indicate that systemically administered LD IL-2 directly mediates significant disease-modifying pathology in the CNS
- LD IL-2 study results increase our confidence in COYA 302 outcomes in neurodegenerative diseases (ALS and FTD)
- LD IL-2 study results increase our confidence in the opportunity to combine COYA 301 with other complementary modalities targeting AD (Amyloid, Tau targeting agents)
- LD IL-2 study results increase our confidence to pursue additional value-creating strategic partnership opportunities for COYA 301



COYA 303

An investigational biologic combination of COYA 301, Coya's low-dose interleukin 2 (LD IL-2) and a GLP-1 receptor agonist (GLP-1RA), designed to deliver a multi-targeted immunomodulatory therapeutic in autoimmune and neurodegenerative diseases



Evaluation of COYA 303 (COYA 301 and GLP-1RA combination)



Glucagon-like peptide-1 receptor agonists (GLP-1RA) demonstrate promising immunomodulatory effects in preclinical models and clinical trials. To determine whether there are potential additive beneficial effects of GLP-1RA in combination with COYA 301, we conducted the following:

- In vitro suppression assays (activated macrophages and responder T cells)
- Preclinical model (inflammatory model)- Cohort 1 data shows
- Potential exposure study in patients at Houston Methodist Hospital

COYA 303 Shows Synergistic Potential

- COYA 303 demonstrated significant attenuation of neuroinflammation in cortex and hippocampus brain regions and upregulation of anti-inflammatory markers, and significant improvement of systemic Treg function and pro-inflammatory cytokines;
- Results from the first cohort of this in-vivo animal study confirm the previously reported positive findings from in vitro human immune cell systems demonstrating that COYA 303 significantly enhances Treg suppressive function and survival in highly inflammatory microenvironments.

Coya Teams

- 🛡 Leadership Team
- 🛡 Operational Team
- 🛡 Scientific Advisory Team
- 🛡 Board of Directors

Expert Leadership Team



Arun Swaminathan, Ph.D.
Chief Executive Officer



Fred Grossman, D.O., FAPA
President & Chief
Medical Officer



David Snyder
Chief Financial Officer &
Chief Operating Officer

<— Prior Experiences —>

Bristol Myers Squibb

Actinium
Pharmaceuticals Inc.

Alteogen Inc.

Johnson-Johnson

Sunovion

Bristol Myers Squibb

Mesoblast

Eli Lilly

Glenmark

DisperSol
Technologies

Excure

Cellular Dynamics
International

Roche NimbleGen

Experienced Development Team



Michelle Frazier, Ph.D.
Senior Vice President of
Regulatory Affairs



Mark Gordon, MD, FAAN
Vice President of Clinical
Development



Karen King, MS
Senior Vice President of
Program Management
and Clinical Operations



Daniel Barvin, M.B.A
Vice President of Operations
& Patient Advocacy

Revanche Therapeutics

CytomX

FDA

Coherus BioSciences

Long Island Jewish
Medical Center

Boehringer Ingelheim

Teva Pharmaceuticals

Recursion
MacroGenics

Supernus

Shire

MedImmune

End The Legacy:
Genetic ALS & FTD

I AM ALS

Morgan Stanley

World Class Scientific Advisors



Shimon Sakaguchi, M.D., Ph.D.

Member of the National
Academy of Sciences



Clive Svendsen, Ph.D.

Director, Cedars-Sinai
Regenerative Medicine Institute



**Malcolm Brenner, M.D.,
Ph.D.**

Director, Center for Cell and
Gene Therapy



Stanley Appel, M.D.

Co-Director, Houston Methodist
Neurological Institute

<- Prior Experiences ->

Distinguished Professor at
the World Premier
International Research
Institute - Immunology
Frontier Research Center at
Osaka University

Discovered Tregs in 1995

Professor in Residence
at UCLA

Consulting Professor at
Stanford University

Renowned expert on stem cell
biology and regenerative
therapeutic approaches

Professor, Baylor
College of Medicine

Dedicated career to the field
of stem cell transplantation
through the therapeutic use of
T cell immunologic
approaches

For more than 50 years,
Dr. Appel has devoted his
life to finding solutions
for people living with ALS

Pioneered role of Tregs in
Neurodegeneration

Distinguished Board of Directors



Howard Berman, Ph.D.
Executive Chairman
of the Board



Ann Lee, Ph.D.
Chief Technical Officer
of Prime Medicine



Dov Goldstein, M.D., MBA
Chief Financial Officer
of BioAge Labs



Wilbur Ross
Former U.S. Secretary of
Commerce



Anabella Villalobos, Ph.D.
Head of Biotherapeutics and
Medicinal Sciences of Biogen



Dieter Weinand
Former Chairman of Board
and CEO, Bayer Pharma, AG



Thank You!

