

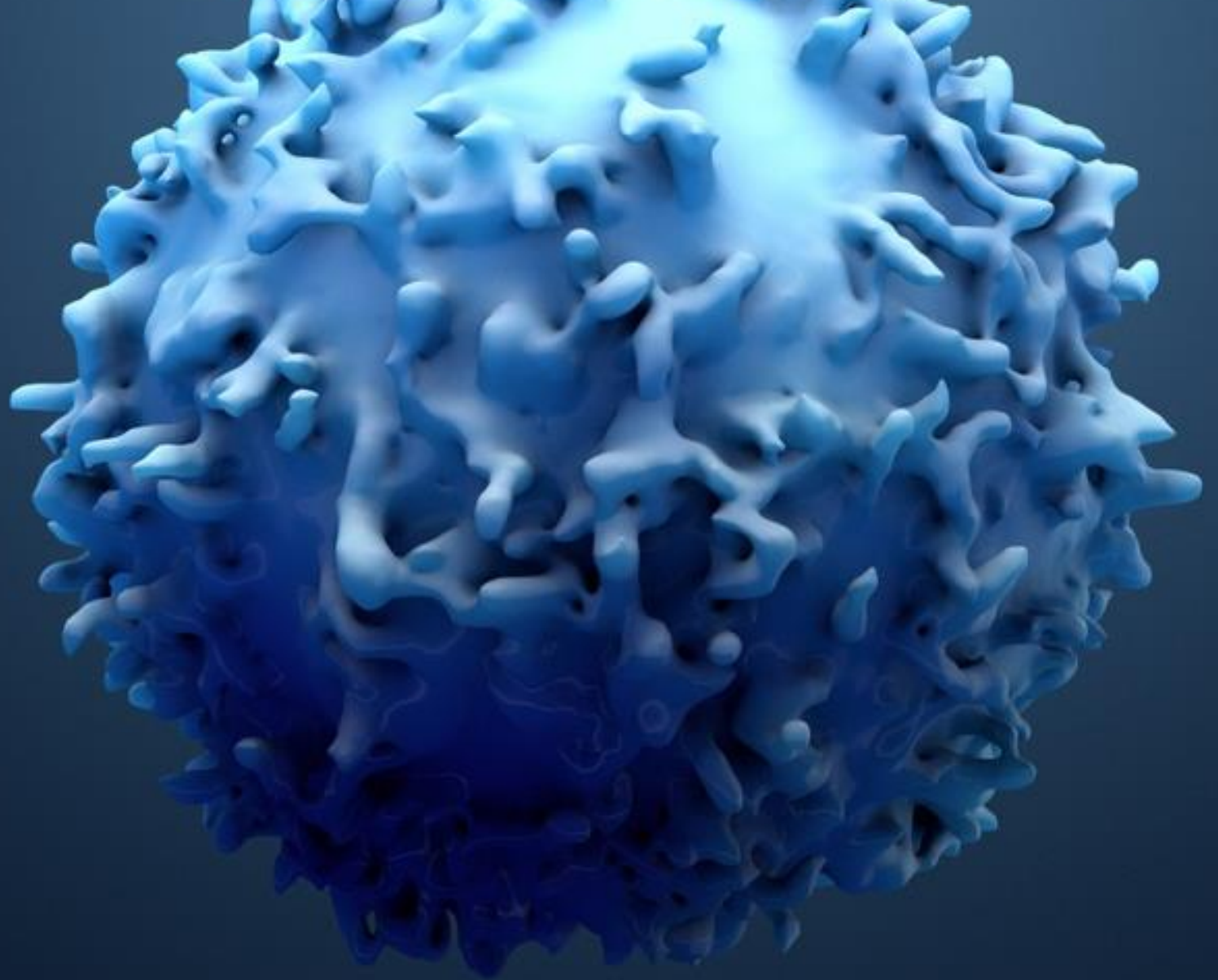


*Unleashing the Power
of Regulatory T cells
(Tregs)*

Investor Presentation

January 2025

NASDAQ: COYA



Cautionary Note of Forward-Looking Statements and Disclaimers

This presentation and the accompanying oral presentation contain “forward-looking” statements that are based on our management’s beliefs and assumptions and on information currently available to management. Forward-looking statements include all statements other than statements of historical fact contained in this presentation, including information concerning our current and future financial performance, business plans and objectives, current and future clinical and preclinical development activities, timing and success of our ongoing and planned clinical trials and related data, the timing of announcements, updates and results of our clinical trials and related data, our ability to obtain and maintain regulatory approval, the potential therapeutic benefits and economic value of our product candidates, competitive position, industry environment and potential market opportunities. The words “believe,” “may,” “will,” “estimate,” “continue,” “anticipate,” “intend,” “expect,” and similar expressions are intended to identify forward-looking statements.

Forward-looking statements are subject to known and unknown risks, uncertainties, assumptions and other factors including, but not limited to, those related to risks associated with the success, cost and timing of our product candidate development activities and ongoing and planned clinical trials; our plans to develop and commercialize targeted therapeutics; the progress of patient enrollment and dosing in our preclinical or clinical trials; the ability of our product candidates to achieve applicable endpoints in the clinical trials; the safety profile of our product candidates; the potential for data from our clinical trials to support a marketing application, as well as the timing of these events; our ability to obtain funding for our operations; development and commercialization of our product candidates; the timing of and our ability to obtain and maintain regulatory approvals; the rate and degree of market acceptance and clinical utility of our product candidates; the size and growth potential of the markets for our product candidates, and our ability to serve those markets; our commercialization, marketing and manufacturing capabilities and strategy; future agreements with third parties in connection with the commercialization of our product candidates; our expectations regarding our ability to obtain and maintain intellectual property protection; our dependence on third party manufacturers; the success of competing therapies or products that are or may become available; our ability to attract and retain key scientific or management personnel; our ability to identify additional product candidates with significant commercial potential consistent with our commercial objectives; and our estimates regarding expenses, future revenue, capital requirements and needs for additional financing.

We have based these forward-looking statements largely on our current expectations and projections about future events and trends that we believe may affect our financial condition, results of operations, business strategy, short-term and long-term business operations and objectives, and financial needs. Moreover, we operate in a very competitive and rapidly changing environment, and new risks may emerge from time to time. It is not possible for our management to predict all risks, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in any forward-looking statements we may make. In light of these risks, uncertainties and assumptions, the forward-looking events and circumstances discussed herein may not occur and actual results could differ materially and adversely from those anticipated or implied in the forward-looking statements. Although our management believes that the expectations reflected in our forward-looking statements are reasonable, we cannot guarantee that the future results, levels of activity, performance or events and circumstances described in the forward-looking statements will be achieved or will occur. We undertake no obligation to publicly update any forward-looking statements, whether written or oral, that may be made from time to time, whether as a result of new information, future developments or otherwise.

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Coya Therapeutics: Overview

Our Mission	➤ Develop innovative therapies to address the significant unmet medical needs of patients with serious neurodegenerative and autoimmune diseases
Targeting Regulatory T Cell (Treg) Biology	➤ Tregs modulate the immune system's response and ameliorate inflammatory mechanisms and inhibit pro-inflammatory cytokines. They play a key role in maintaining immune tolerance and homeostasis and preventing neurodegenerative and autoimmune disease
Lead Assets (for subcutaneous and/or intravenous administration)	➤ COYA 301: Proprietary recombinant human Low Dose Interleukin-2 (rh LD IL-2) ➤ COYA 302: Proprietary biologic combination rh LD IL-2 & cytotoxic T lymphocyte associated antigen-4 immunoglobulin fusion protein (CTLA4-Ig)
Combination Targeting Strategy	➤ Targeting multiple immune pathways to restore the anti-inflammatory functions of Tregs while inhibiting harmful pro-inflammatory pathways ➤ Enhance Treg function while depleting T effector function and activated macrophages (mechanisms that may be synergistic in suppressing inflammation)
Neurodegenerative Targets	➤ Amyotrophic Lateral Sclerosis (ALS), Frontotemporal Dementia (FTD), and Alzheimer's Disease (AD)
Recent Business Developments	➤ Executed a potential \$700M+ transaction for COYA 302 in ALS with Dr. Reddy's Laboratories (12/6/23) ➤ Completed \$5M strategic investment by Alzheimer's Drug Discovery Foundation (ADDF) (5/20/24) ➤ Completed \$10M private placement of common equity (10/23/24) ➤ \$31M in cash & cash equivalents as of 9/30/24 (not including proceeds from 10/23/24 private placement)

Key Catalysts and Milestones - 2025

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

Receipt: Non-Dilutive Milestone Payment

- Upon Acceptance and first patient dosing, eligible to receive milestone payments of \$8.4M from strategic partner, Dr. Reddy's Laboratories (DRL)

ALS Biomarker Data Release

- **2Q 2025:** ALS Biomarker data publication of longitudinal data on Neurofilament Light Chain (NfL) and oxidative stress markers in ALS Patients

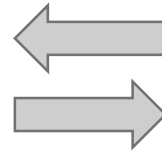
FTD Clinical Data Release and IND

- **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

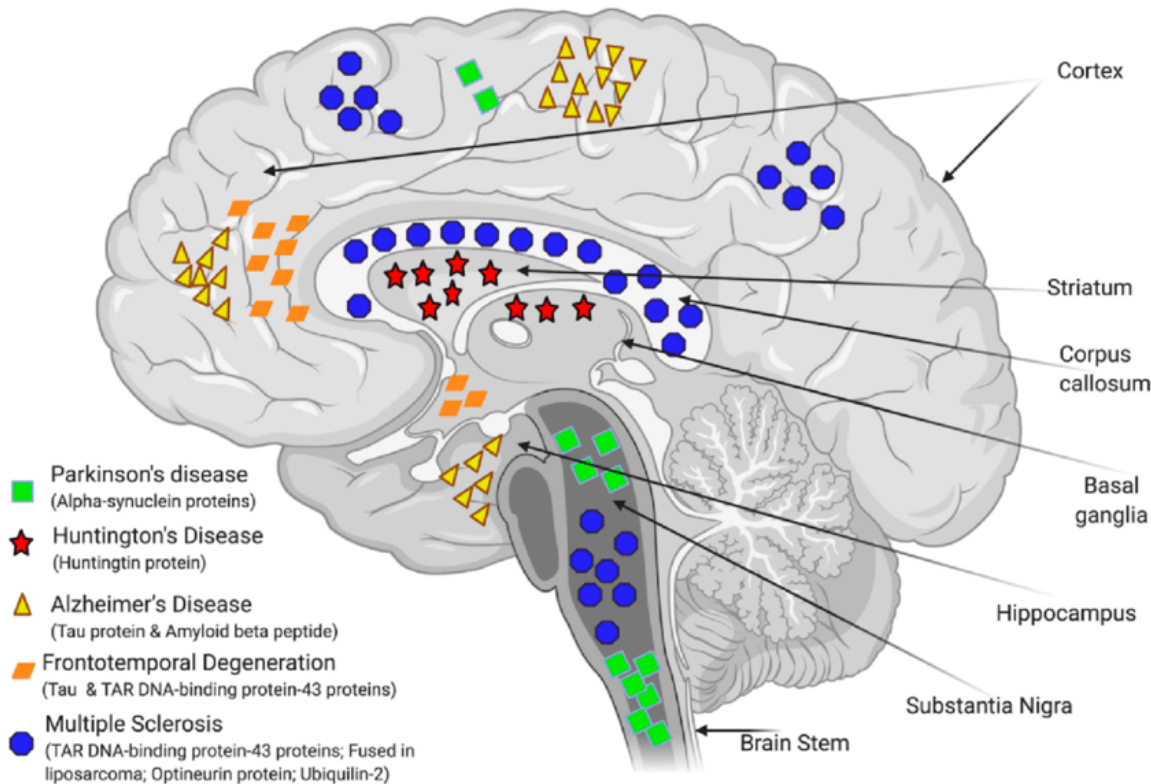
Inflammation Plays a Critical Role in Neurodegeneration:

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

Neurodegeneration: Loss of Selective Population of Neurons



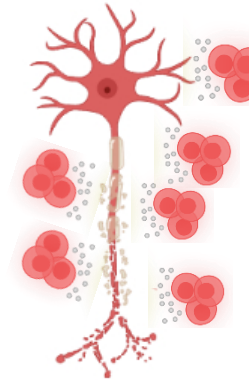
Inflammation in the Nervous System



Healthy Neuron



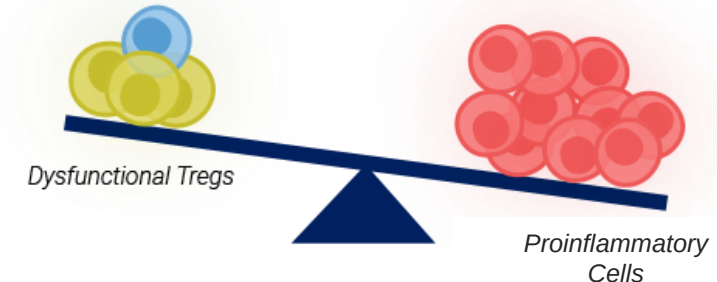
Sick Neuron



Dead Neuron



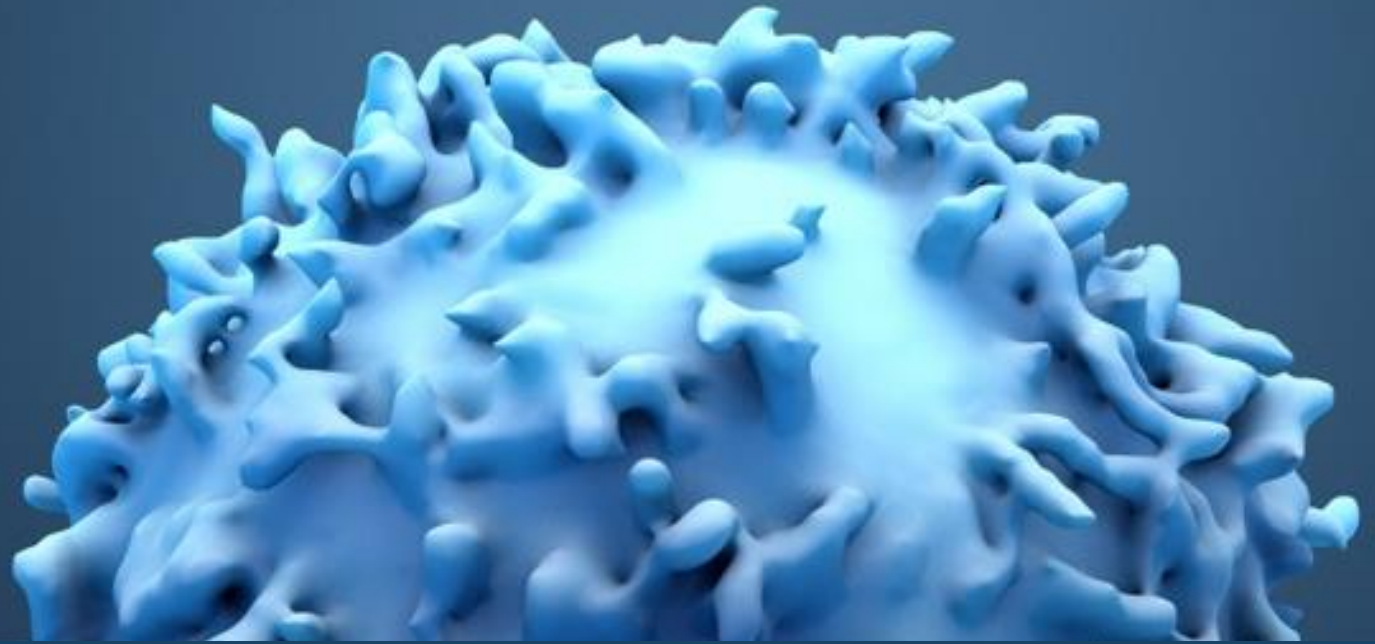
Regulatory T Cells (Tregs) “The Good Guys” play a central role in lowering inflammation and stemming the progression of neurodegeneration



Coya Therapeutics: Combination Therapies Are Integral for Complex Neurodegenerative Diseases

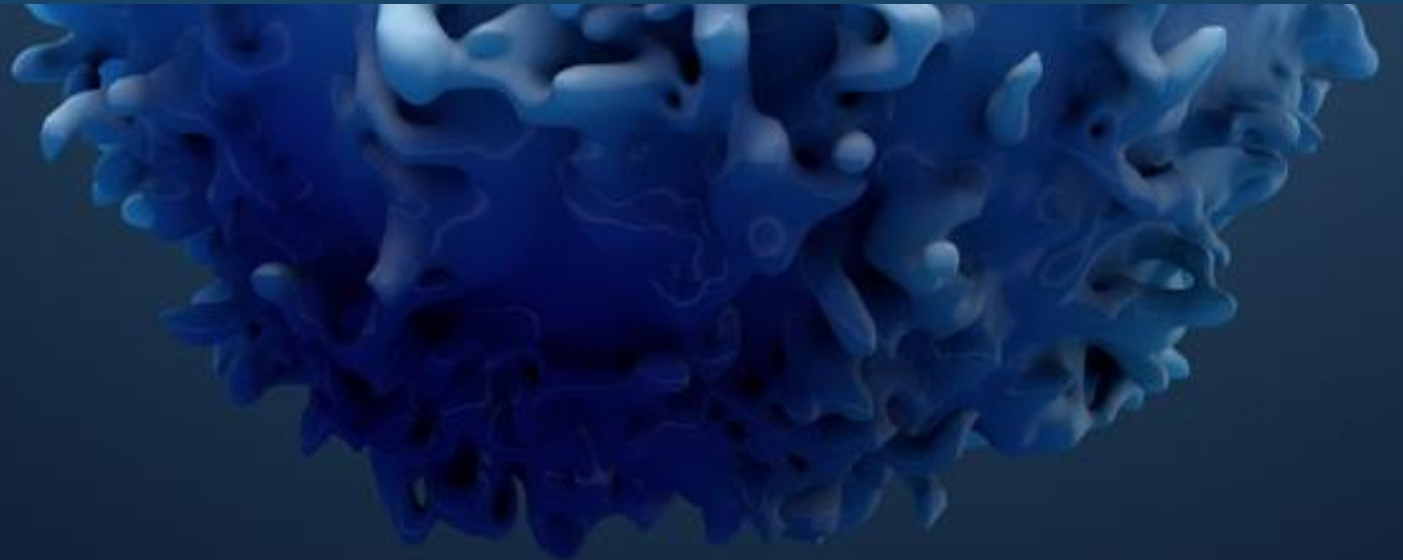
“ Inflammation has emerged as a promising novel pathway for chronic neurological diseases like FTD. A combination drug, like COYA 302, is an innovative approach being developed to suppress neuroinflammation by targeting multiple inflammatory pathways. Combination therapy will be integral to slowing – 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 of the Alzheimer’s Drug Discovery Foundation
Press Release: May 20th, 2024 following announcement of a \$5M strategic investment in Coya



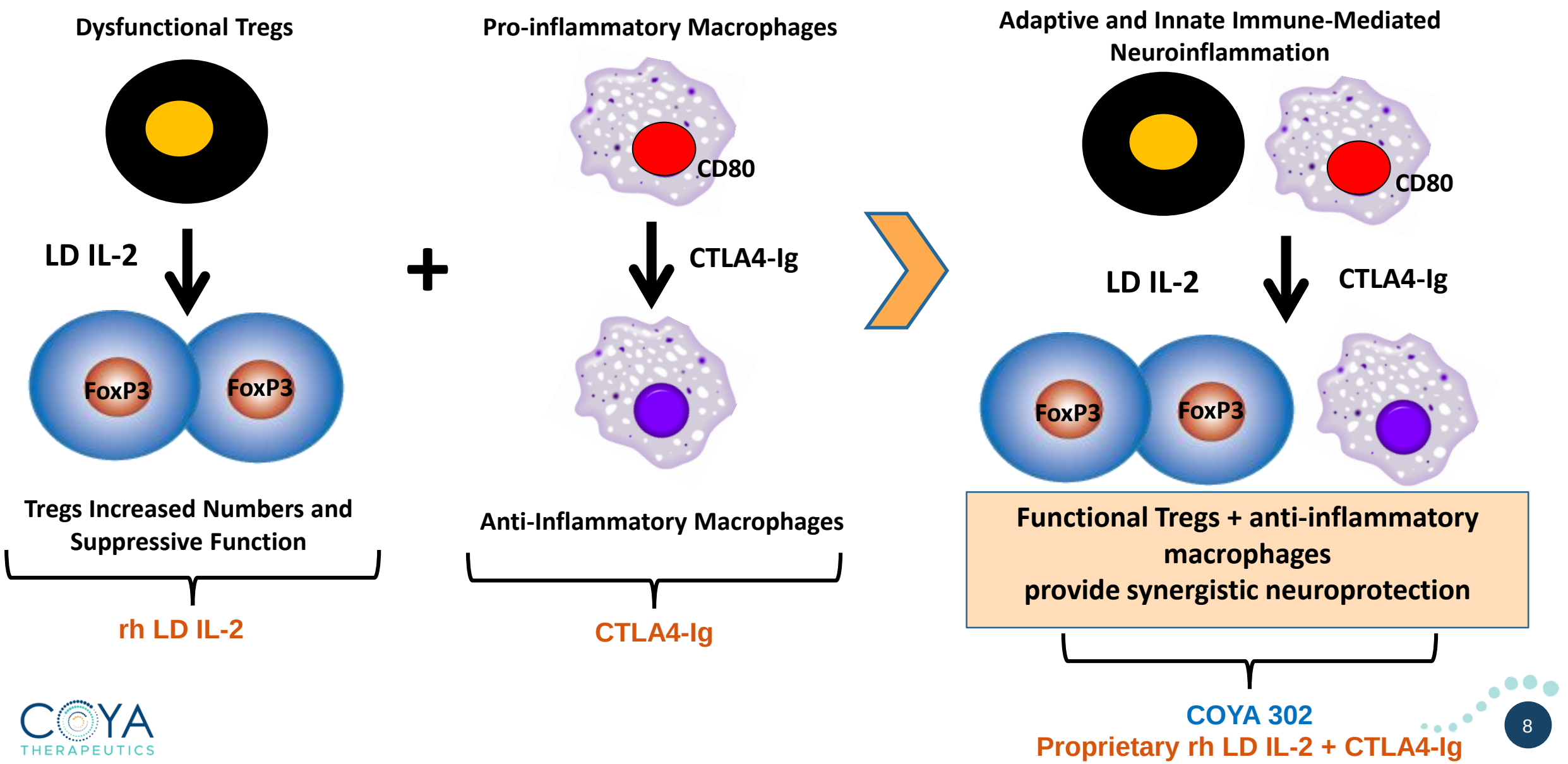
COYA 302

Proprietary recombinant human Low Dose Interleukin-2 (rh LD IL-2) and CTLA4-Ig



Combination Therapies are the Future of Neurodegenerative Disease Therapy

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



COYA-302: A Pipeline Within a Product With > \$10B Potential

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

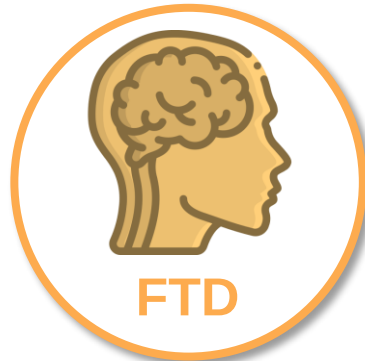


ALS

- High Unmet Need
- Regulatory Flexibility
- >\$1B Sales Potential
- Fast Path to Approval if Effective

ALS = Amyotrophic
Lateral Sclerosis

Orphan Indication

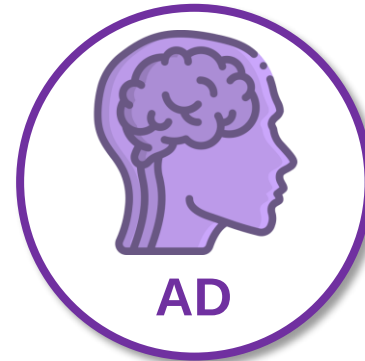


FTD

- No Approved Therapies
- Regulatory Flexibility
- >\$1B Sales Potential
- Fast Path to Approval if Effective

FTD = Frontotemporal
Dementia

Orphan Indication



AD

- High Unmet Need
- Most common
Neurodegenerative Disease
- >\$5B Sales Potential

AD = Alzheimer's Disease

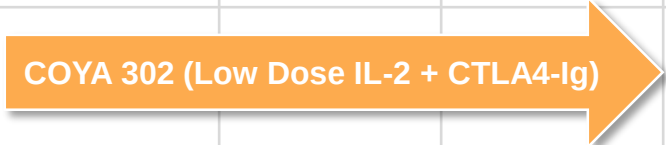
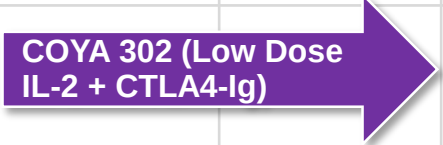
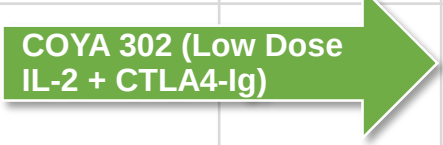


PD

- High Unmet Need
- Second most common
Neurodegenerative Disease
- >\$5B Sales Potential

PD = Parkinson's Disease

COYA 302 Program

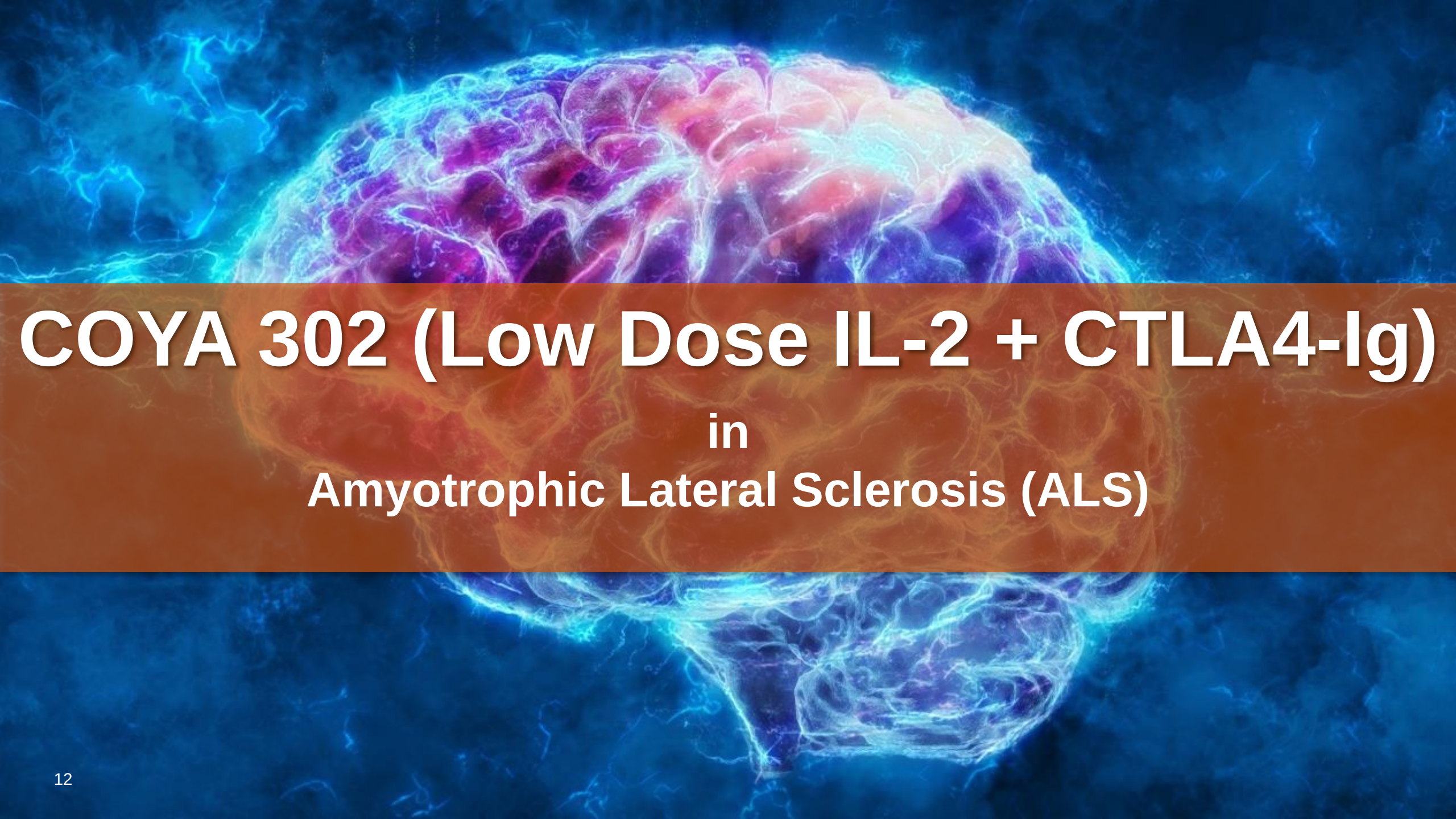
Product Type	Discovery	Preclinical	IND-Enabling	Phase 1	Phase 2	Phase 3	Partnerships / Collaborations	2025 Milestones
COYA 302 Treg-Enhancing / T Effector & Macrophage Depleting Biologics	 COYA 302 (Low Dose IL-2 + CTLA4-Ig) <i>Amyotrophic Lateral Sclerosis (ALS)</i> Completed POC IIT open label study in patients with ALS*						Licensing Transaction on 12/6/23: Dr. Reddy's Laboratories	2Q 2025: IND data submission and Phase 2b Trial initiation upon acceptance 2Q 2025: ALS biomarker data release
COYA 302 Treg-Enhancing / T Effector & Macrophage Depleting Biologics	 COYA 302 (Low Dose IL-2 + CTLA4-Ig) <i>Frontotemporal Dementia (FTD)</i>						Retained Worldwide Rights	2H 2025: Top-line IIT clinical data release: Open label FTD study 2H 2025: File Phase 2 FTD IND
COYA 302 Treg-Enhancing / T Effector & Macrophage Depleting Biologics	 COYA 302 (Low Dose IL-2 + CTLA4-Ig) <i>Alzheimer's Disease (AD)</i>						Retained Worldwide Rights	Q1 2025: New clinical data release from LD IL-2 IIT (post-CTAD data)
COYA 302 Treg-Enhancing / T Effector & Macrophage Depleting Biologics	 COYA 302 (Low Dose IL-2 + CTLA4-Ig) <i>Parkinson's Disease (PD)</i>						Retained Worldwide Rights	1H 2025: Advance pre-clinical

Additional Pipeline Assets

Product Type	Discovery	Preclinical	IND-Enabling	Phase 1	Phase 2	Phase 3	2025 Milestones
COYA 301 Proprietary Low Dose IL-2	 COYA 301 (Low Dose IL-2) <i>Alzheimer's Disease (AD)</i> Presented randomized double-blind placebo controlled IIT of LD-IL2 at CTAD in October, 2024*						1H 2025: Additional biomarker and blood panel data from the academic IIT double-blind Ph2 data presented at CTAD in October 2024
COYA 303 COYA 301 + GLP-1 Agonist	 COYA 303 <i>Undisclosed</i>						1H 2025: - Additional IP filings - Publish pre-clinical in-vitro data - Initiate pre-clinical translational study - Partnership discussions
COYA 201 Allogeneic Treg Derived Exosomes	 COYA 201 <i>Undisclosed</i>						2025: Pre-clinical characterization
COYA 206 Antigen-Directed Allogeneic Treg-Derived Exosomes	 COYA 206 <i>Undisclosed</i>						2025: Pre-clinical characterization

POC- Proof of Concept; IIT- Investigator Initiated Trial

* Conducted using commercially available products

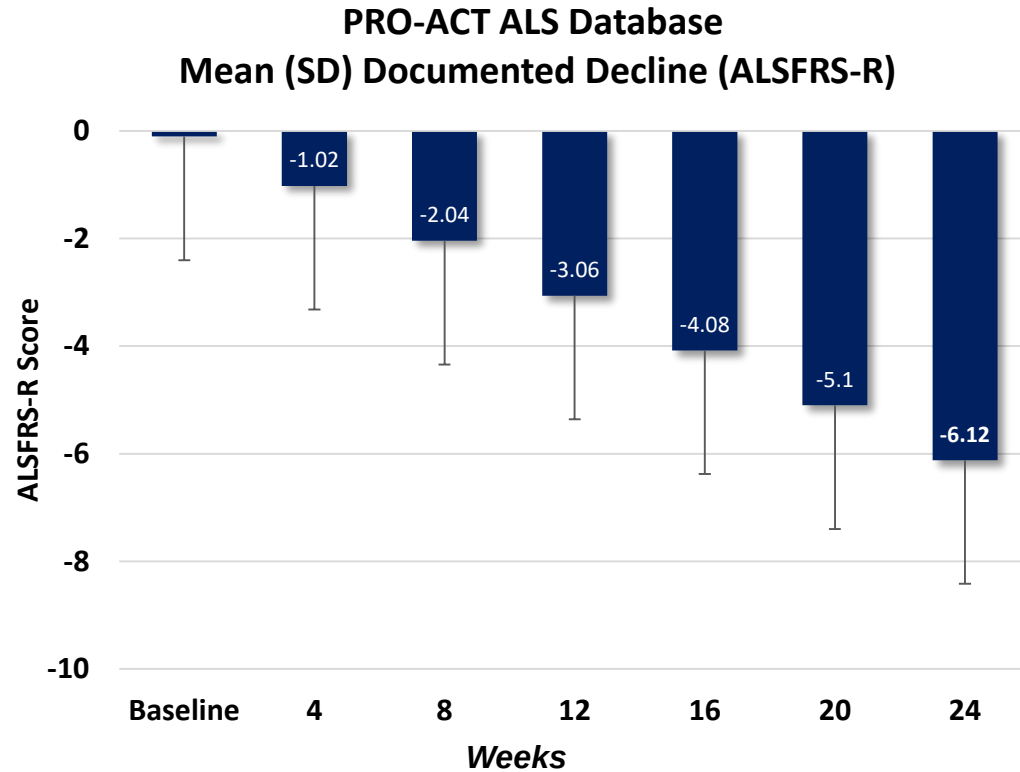


COYA 302 (Low Dose IL-2 + CTLA4-Ig)

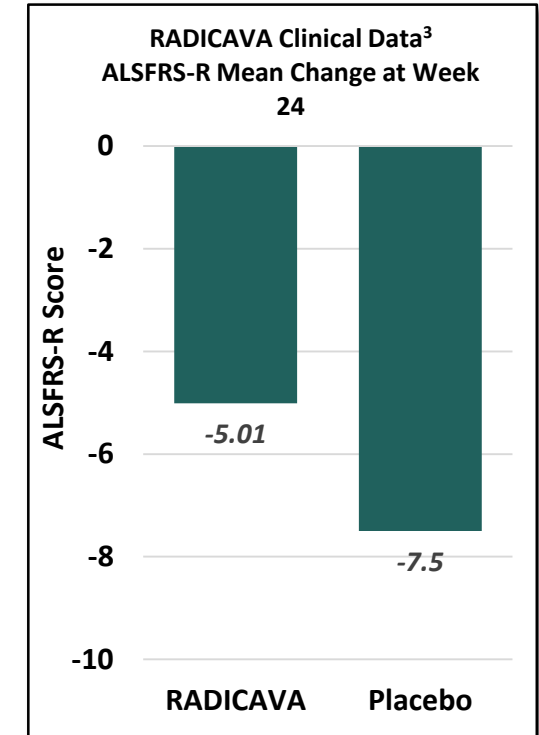
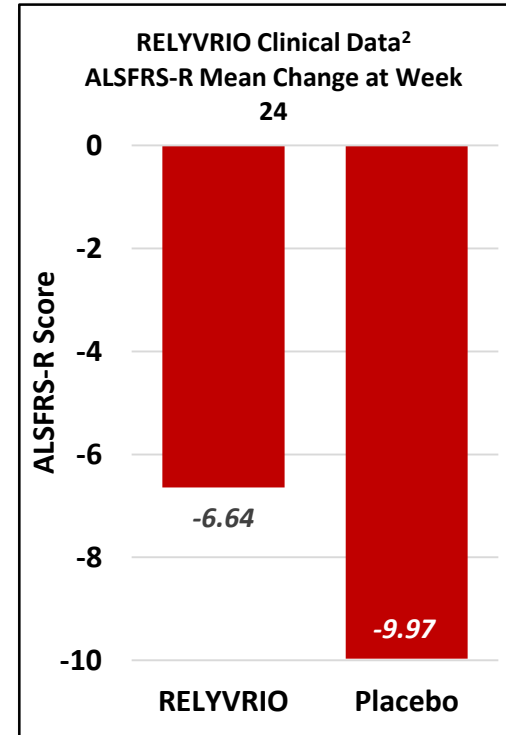
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Amyotrophic Lateral Sclerosis (ALS)

Current Therapies for ALS Aim to Slow Disease Progression



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



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

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

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

Screening

20 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

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

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

Follow-Up

8 weeks

Post-Treatment Assessments

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

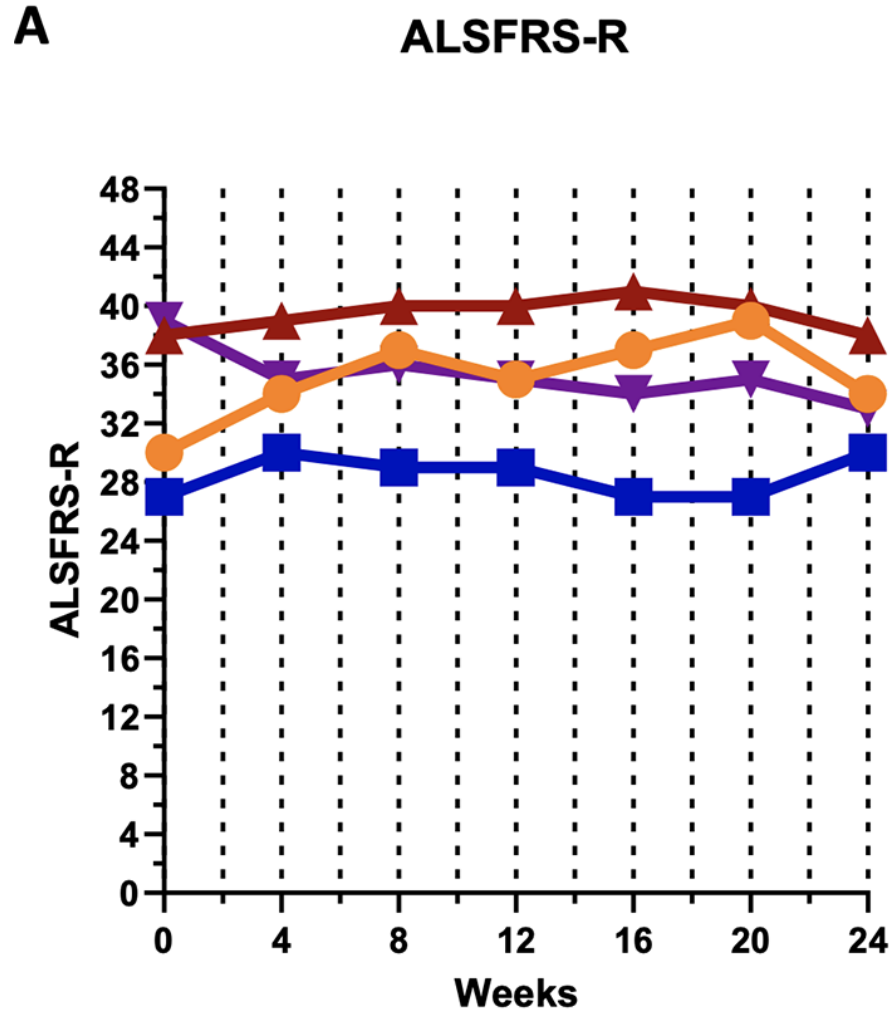
LD IL-2 + CTLA4-Ig Investigator Initiated Clinical Trial

Baseline Characteristics

	Age (years)	Sex	Type	Onset	ALS Progression Prior to Baseline (ALSFRS-R score)	Respiratory Status	Respiratory Support
Patient 1	47	Female	Familial	Limb	-1.6 points / month	No Respiratory Insufficiency	None
Patient 2	54	Male	Sporadic	Limb	-1 points / month	Respiratory Insufficiency	Non-invasive Ventilation
Patient 3	57	Female	Sporadic	Bulbar	-1 point / month	Respiratory Insufficiency	Non-invasive Ventilation
Patient 4	84	Female	Sporadic	Bulbar	-0.7 points / month	Respiratory Insufficiency	None

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

ALSFRS-R Individual Scores

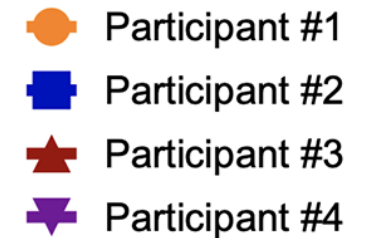


Participant 1: +4 points

Participant 2: +3 points

Participant 3: Stable

Participant 4: -6 points*

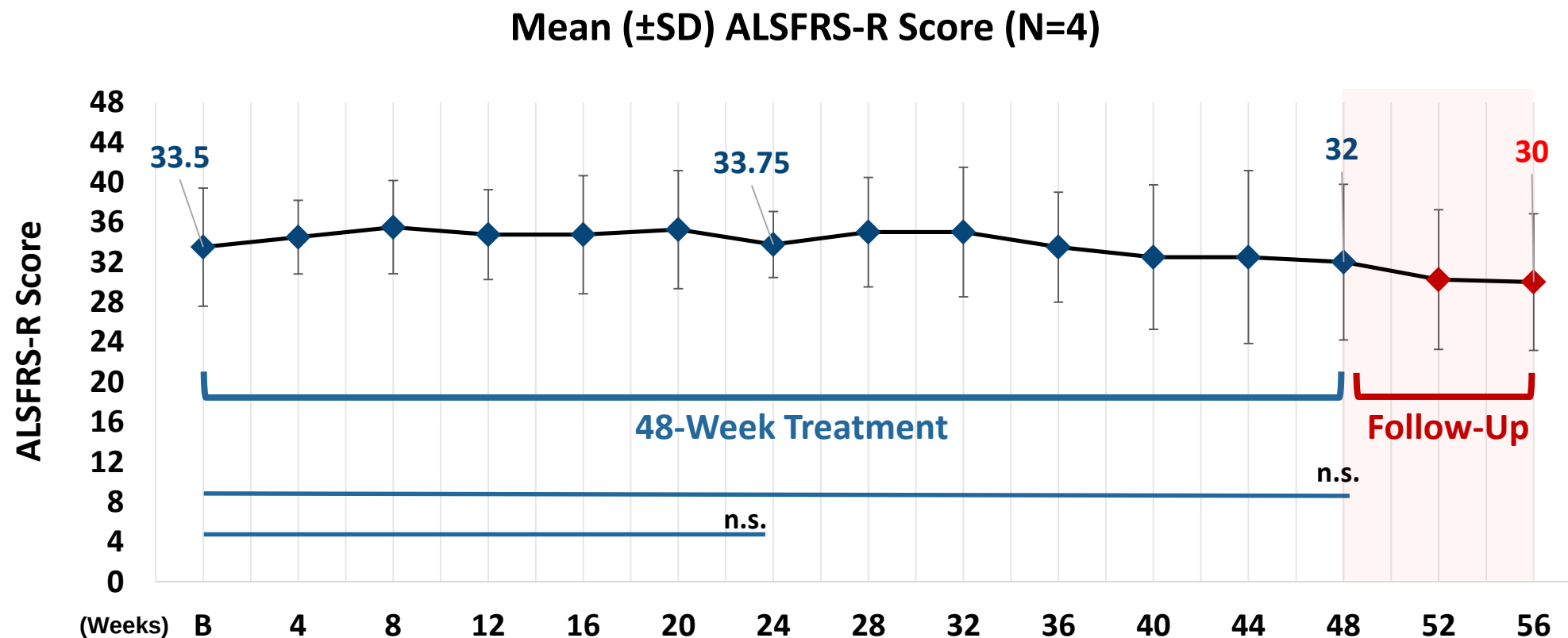


*Declined at month 1 and remained stable thereafter

Thonhoff JR, Beers DR, Zhao W, et al. A Phase 1 Proof-of-Concept Study Evaluating Safety, Tolerability, and Biological Marker Responses with combination therapy of CTLA4-Ig and Interleukin-2 in Amyotrophic Lateral Sclerosis. Frontiers in Neurology, 2024. In press.

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

Appears to Ameliorate ALS Progression Over 48 Weeks



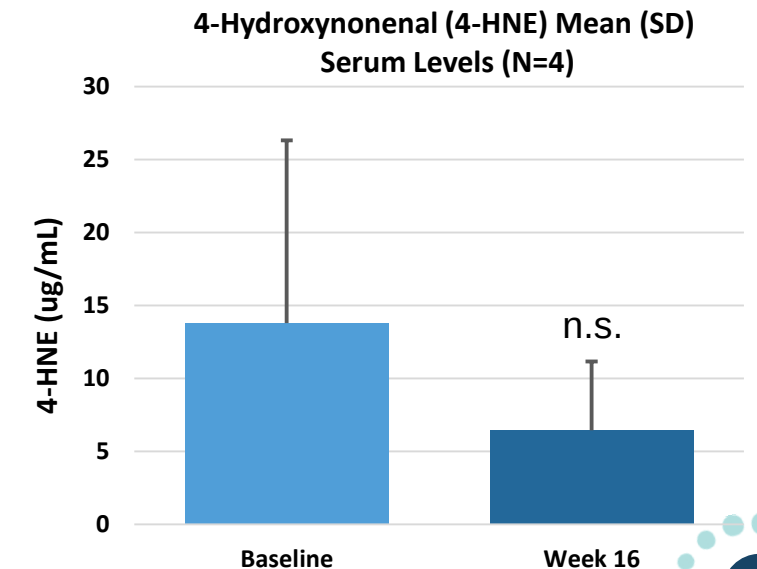
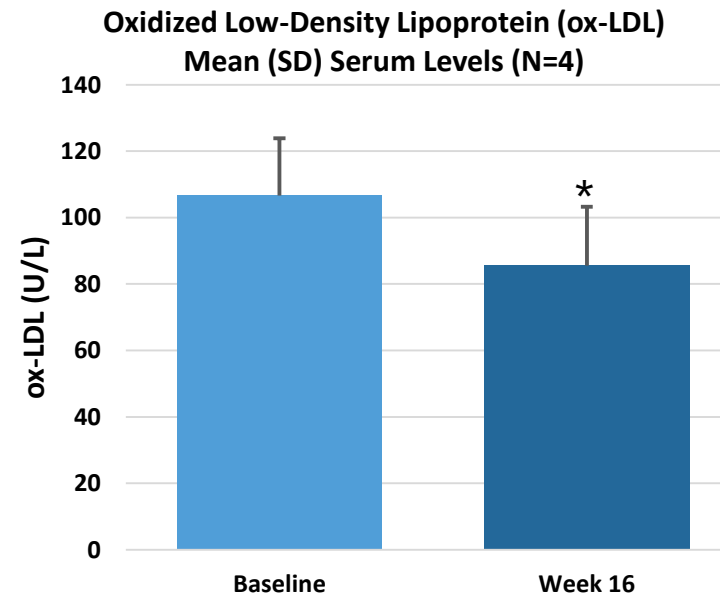
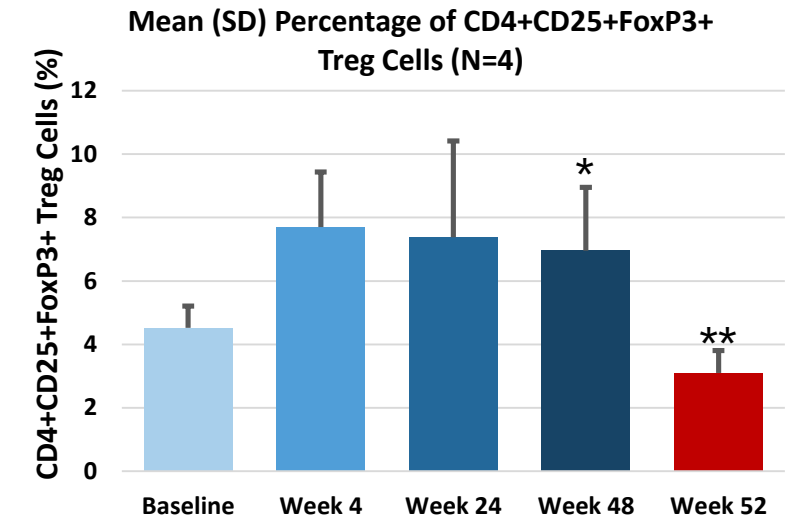
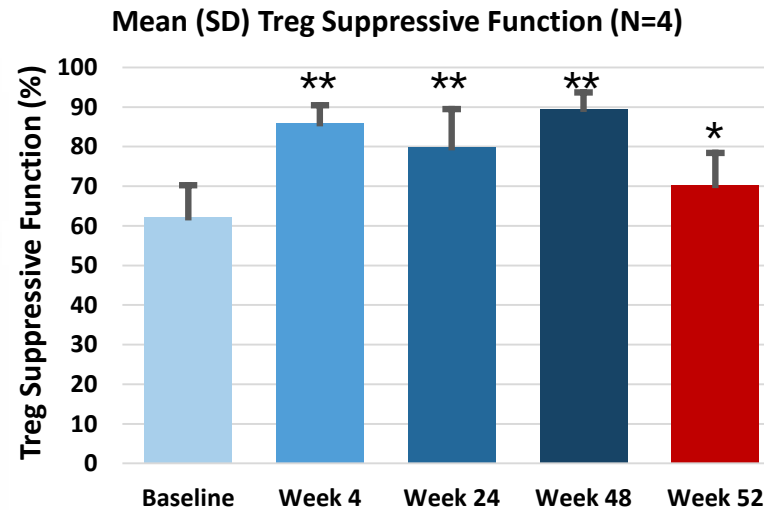
LD IL-2 + CTLA4-Ig was well tolerated over 48 weeks; the most common AE was mild injection site reaction. All patients completed the study; no deaths or serious AEs (SAEs) occurred over the course of the study.

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

Lowered Oxidative Stress Markers

Key Takeaways

- ✓ **LD IL-2 + CTLA4-Ig** significantly expanded Treg suppressive function as early as 4 weeks after initiation of treatment and maintained a significantly increased Treg function.
- ✓ **LD IL-2 + CTLA4-Ig** increased Treg numbers as early as 4 weeks after initiation of treatment and maintained a higher number over the course of treatment.
- ✓ **LD IL-2 + CTLA4-Ig** enhanced suppression of macrophage-mediated oxidative stress and proinflammatory cytokine biomarkers over 48 weeks



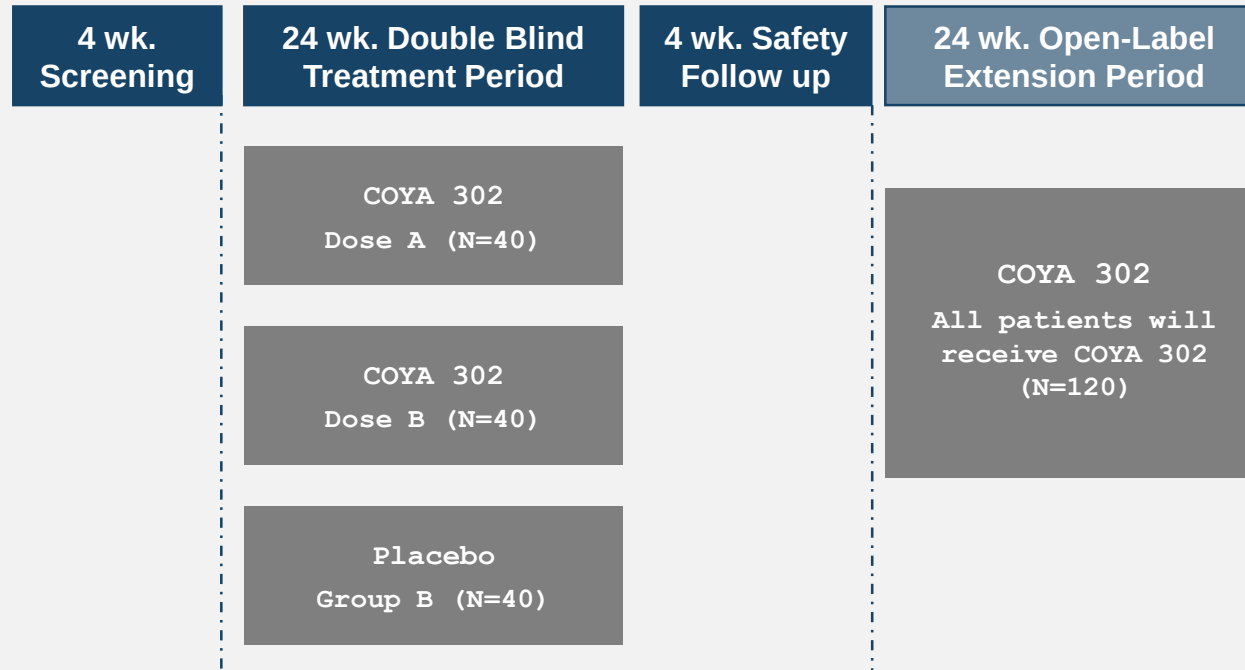
COYA 302:

Overview of Phase 2 Study Design in ALS

Key Inclusion Criteria

- Diagnosis of sporadic or familial ALS
- Time since onset of ALS symptoms $\leq$ 24 months from Screening
- ALSFRS-R score $\geq$ 35 at Screening
- A score of at least 2 points in each ALSFRS-R item
- Forced vital capacity (FVC) $\geq$ 70% of predicted capacity for age, height, and gender at Screening
- Documented disease progression (by ALSFRS-R score) for at least 16 weeks prior to Screening

Randomized, Double-Blind, Placebo-Controlled Phase 2 with Open-Label Extension

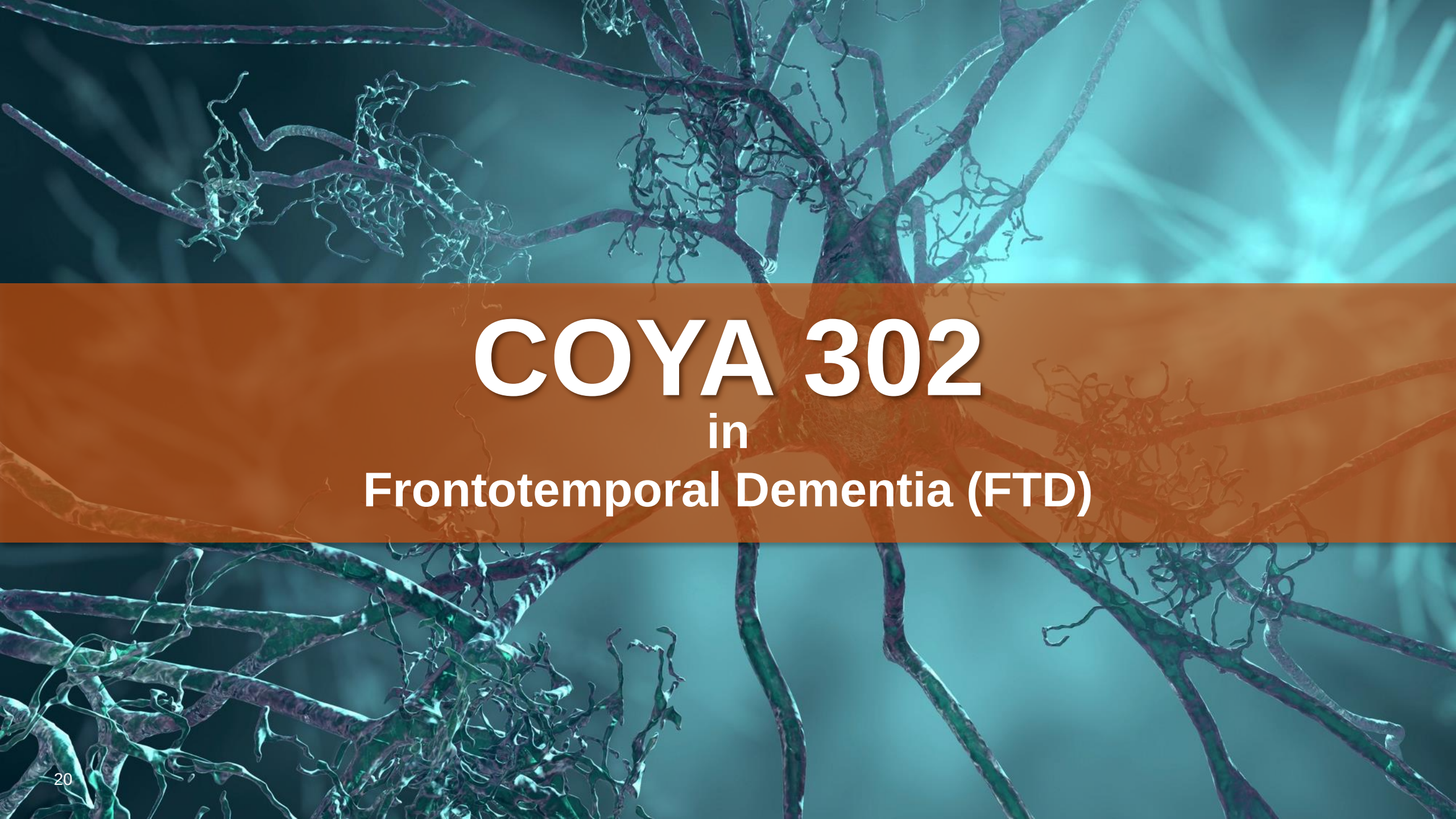


Primary Endpoint

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



COYA 302

in

Frontotemporal Dementia (FTD)

Alzheimer's Drug Discovery Foundation \$5M Investment

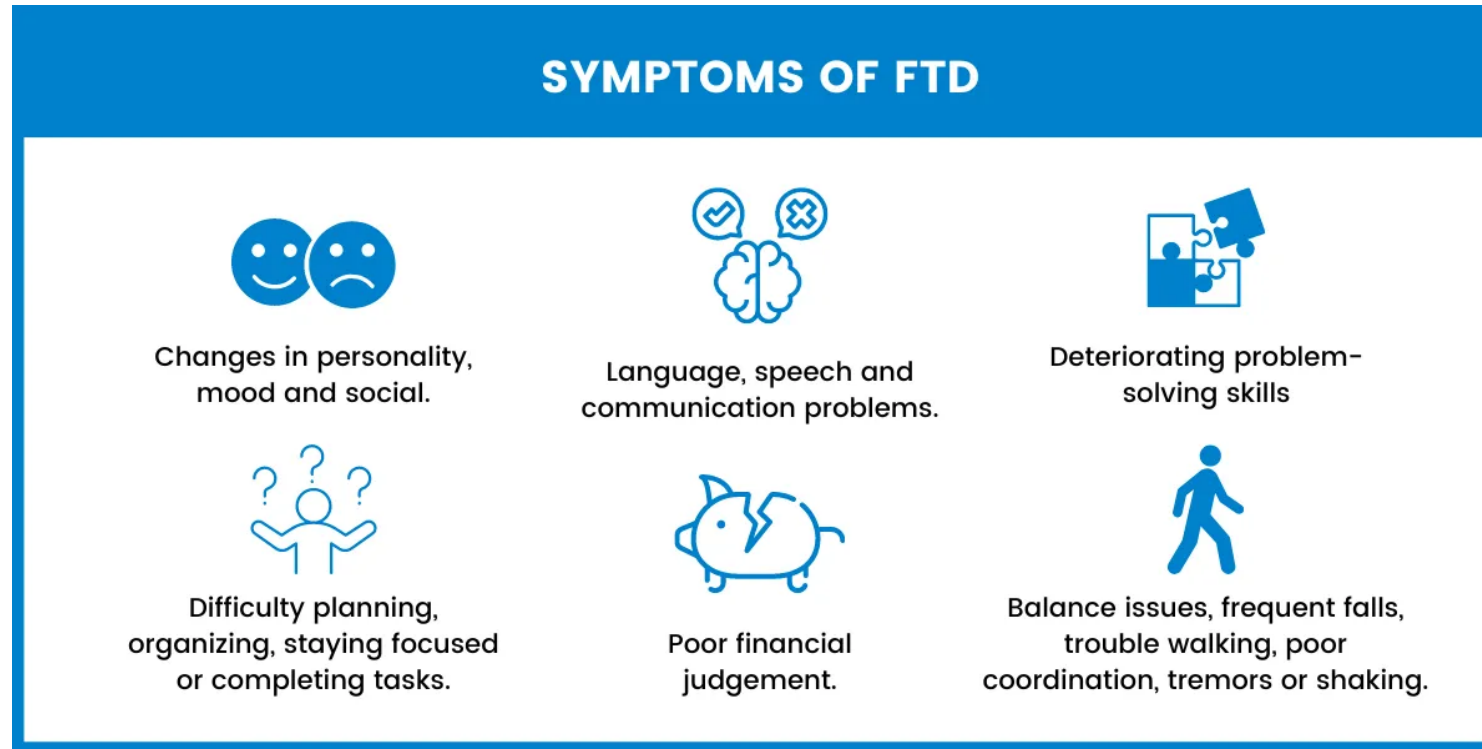
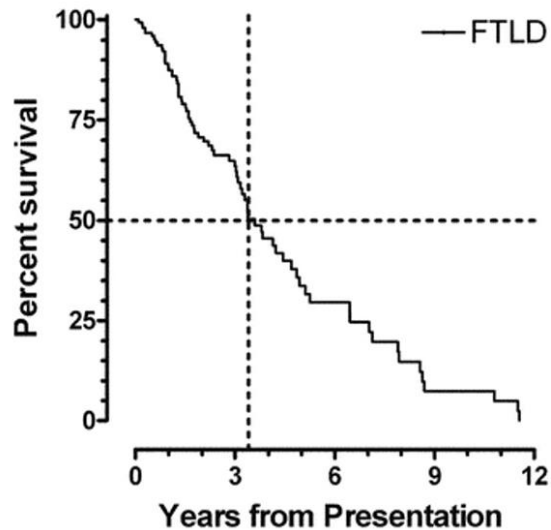
- \$5M investment for funding of Phase 2 FTD study



Alzheimer's
Drug Discovery
Foundation

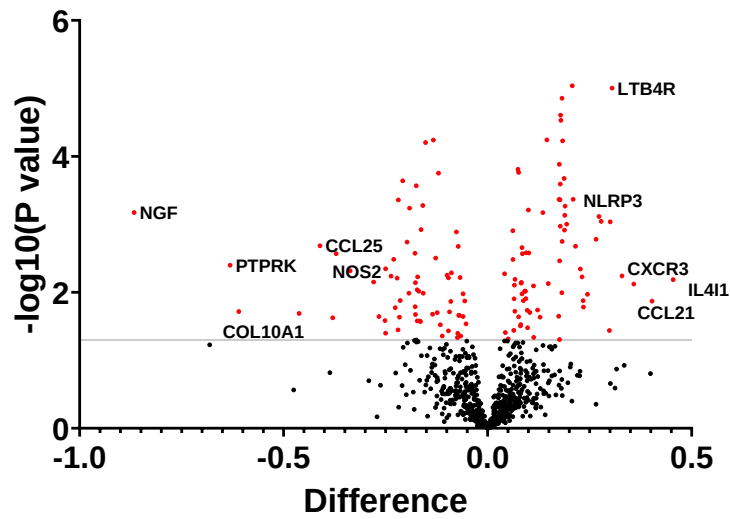
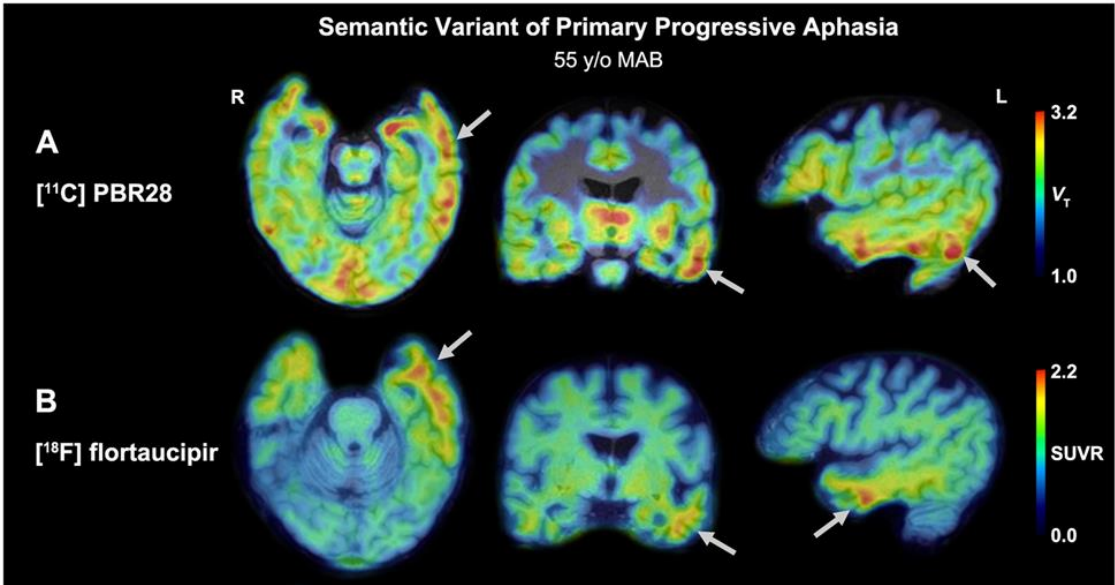
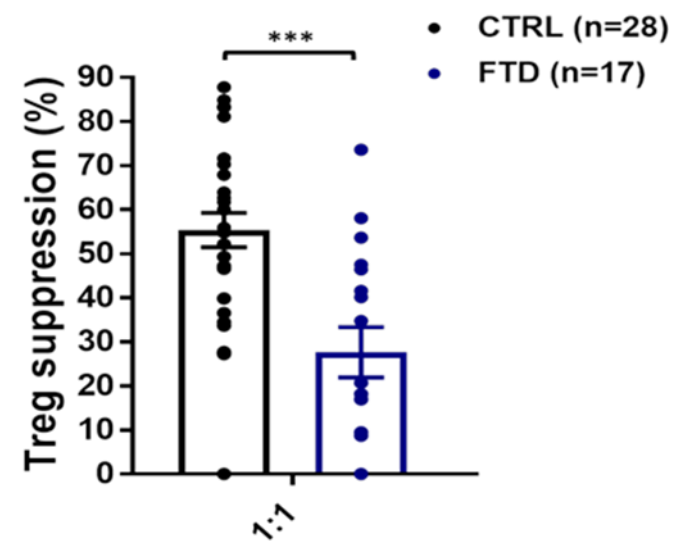
Title: A Phase II, Randomized, Double-Blind, Placebo-Controlled, Multi-Center, 26-Week Study to Evaluate the Safety and Efficacy of COYA 302 for the Treatment of Nonfluent Variant Primary Progressive Aphasia (nfvPPA) Subtype of Frontotemporal Lobar Degeneration (FTLD)

Frontotemporal Disorder (FTD) is a Rare Disease but is One of Most Common Dementias in Younger People

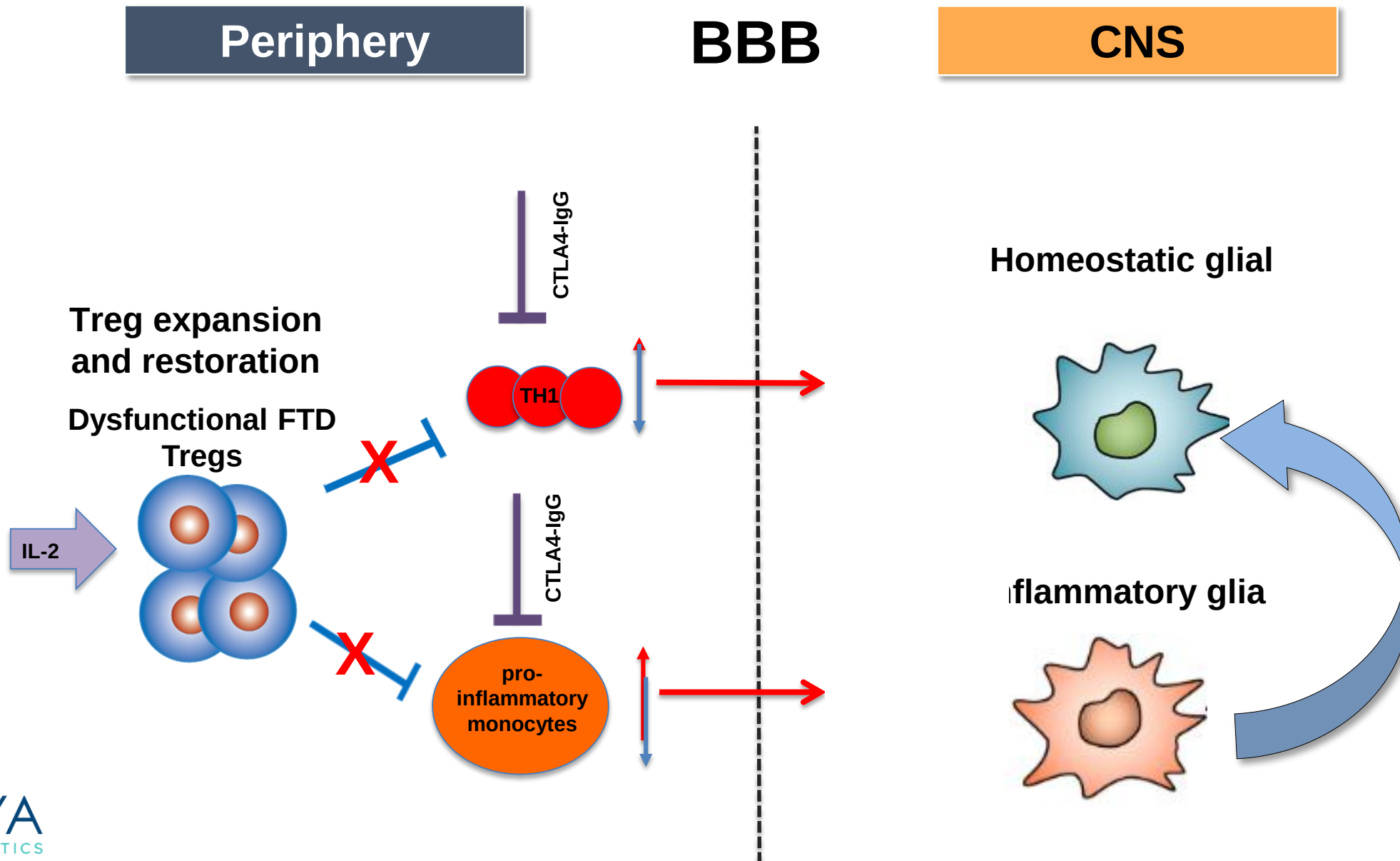


Frontotemporal disorder progresses to death faster than Alzheimer's disease, and, unfortunately, there is no effective treatment

Exacerbated Systemic and Central Inflammatory Responses in FTD

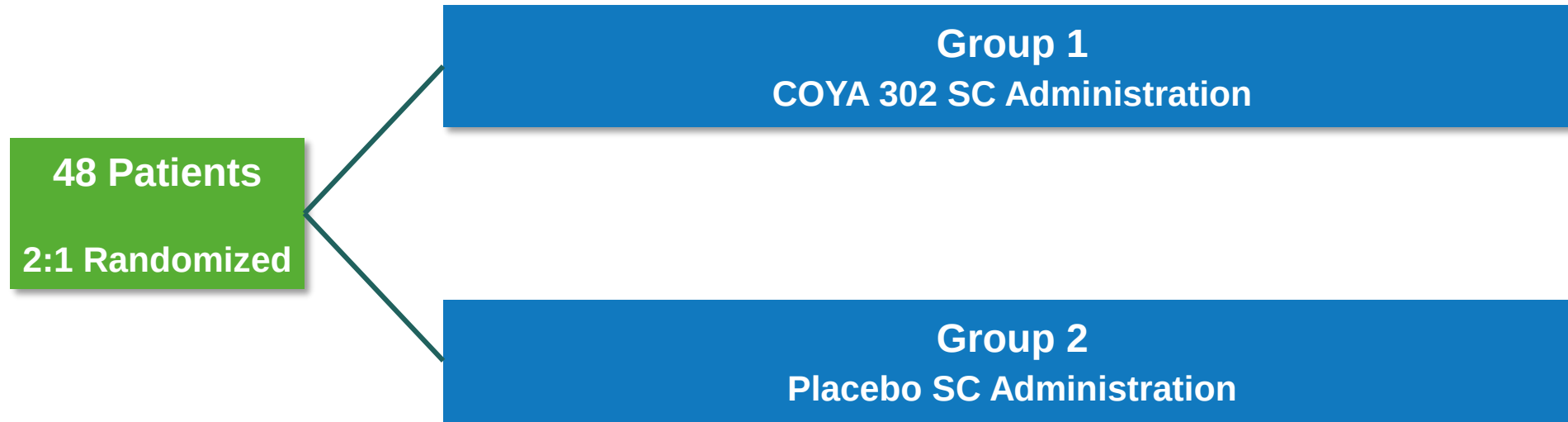


Targeting Inflammation in FTD to Slow Disease Progression



COYA 302 FTD Phase 2a Study

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



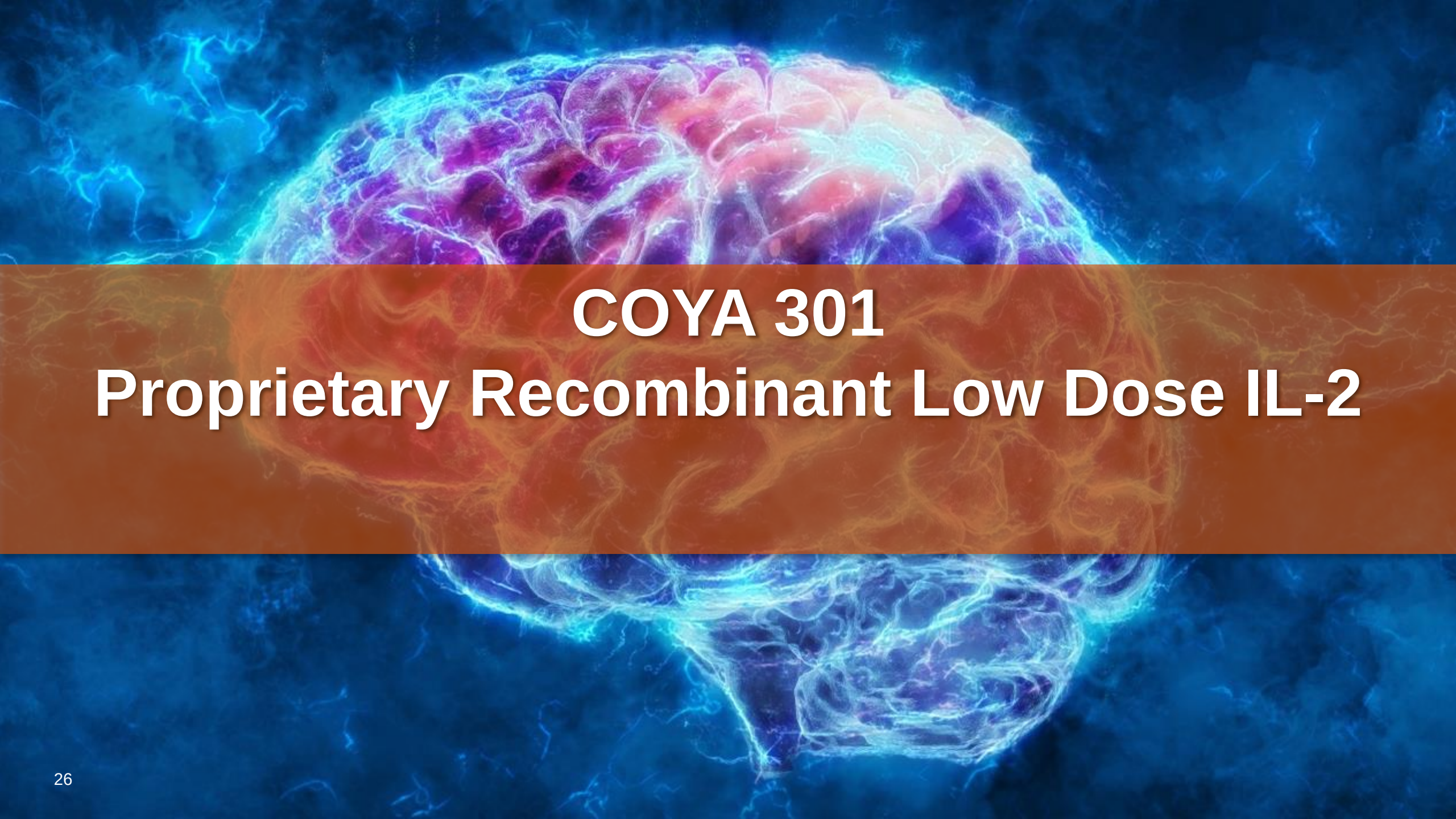
Eligibility Criteria

- 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

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



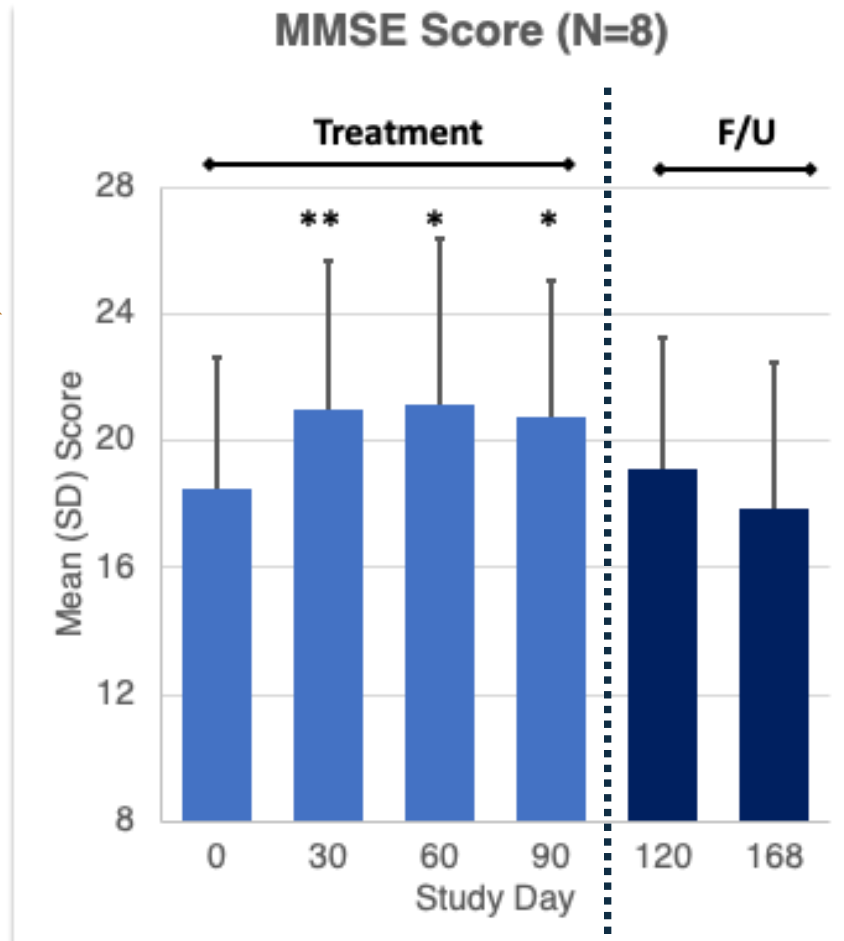
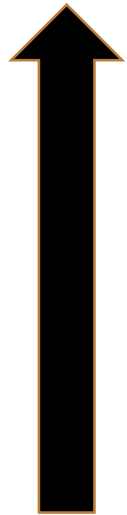
COYA 301

Proprietary Recombinant Low Dose IL-2

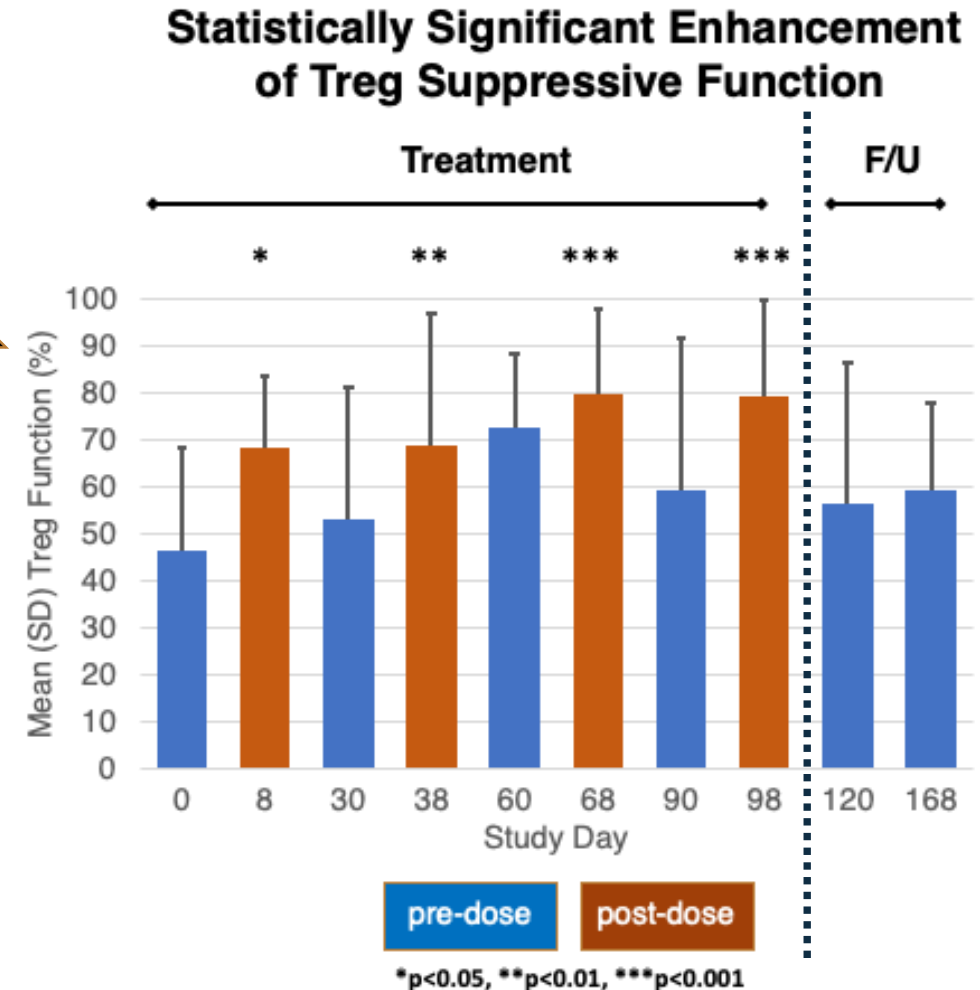
Phase 1 Proof of Concept Open-Label Trial with LD IL-2 in AD

Enhanced Treg Function and halted Cognitive decline

Improved Cognition

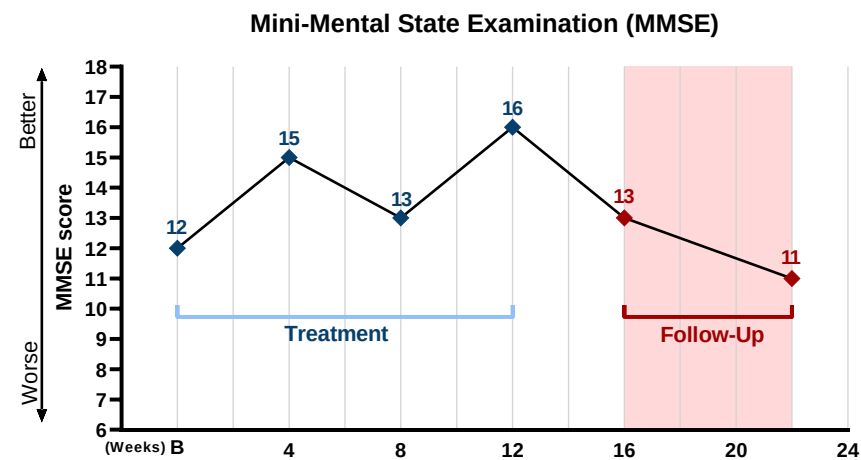


Improved Treg Function



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

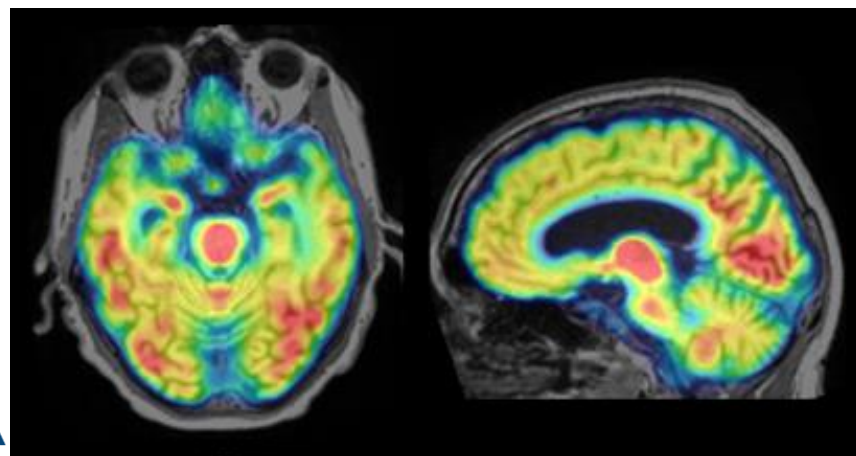


Neuroinflammation (TSPO)
(¹¹C) ER-176 PET Scan at
baseline and after 4-monthly
cycles of low dose IL-2.

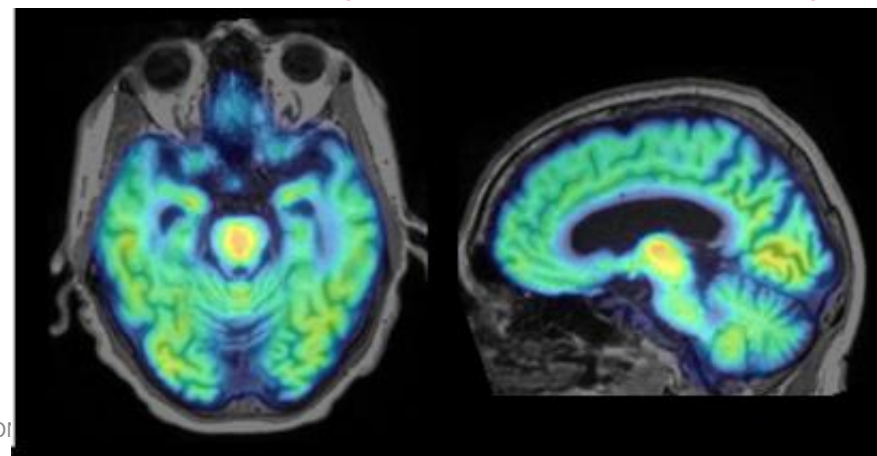
The second PET scan was done
2 weeks after last dose.

PET: Positron Emission Tomography
TSPO: translocator protein

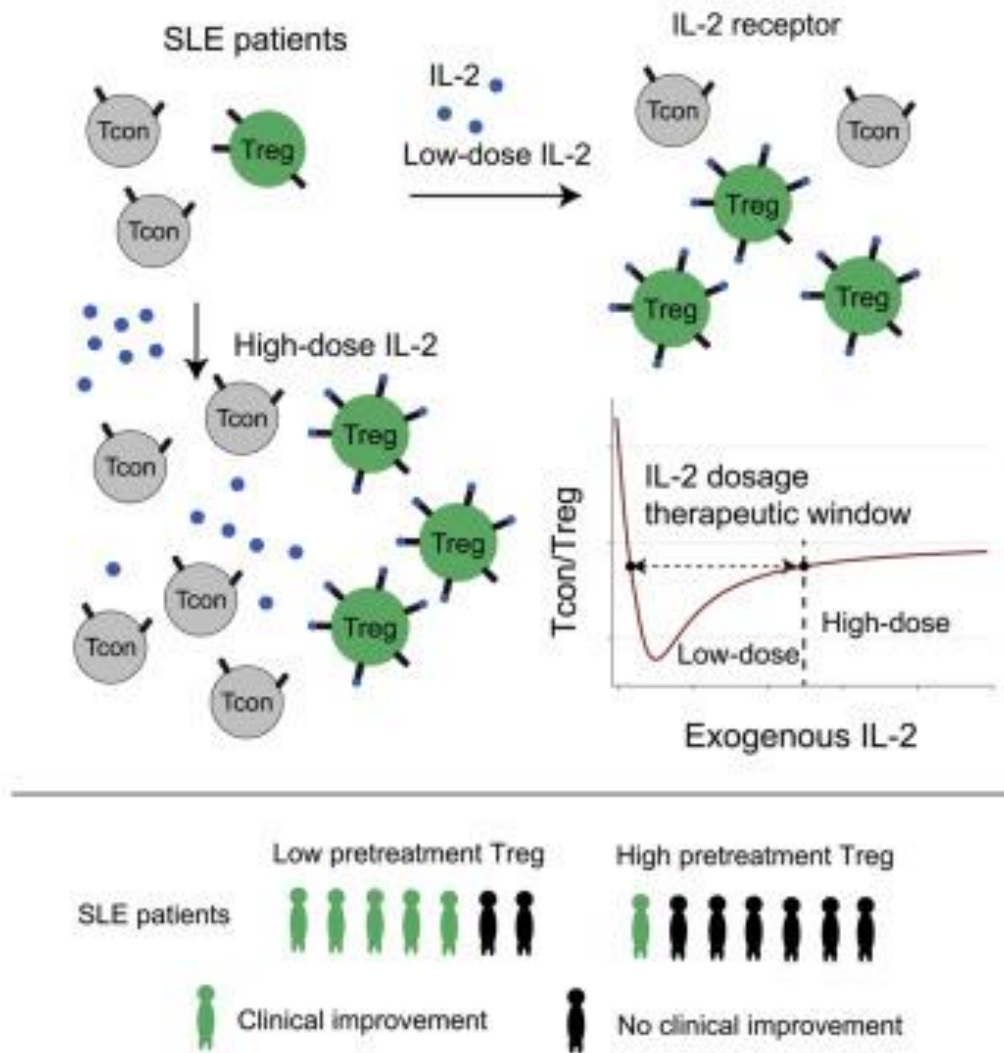
PET Scan (Baseline)



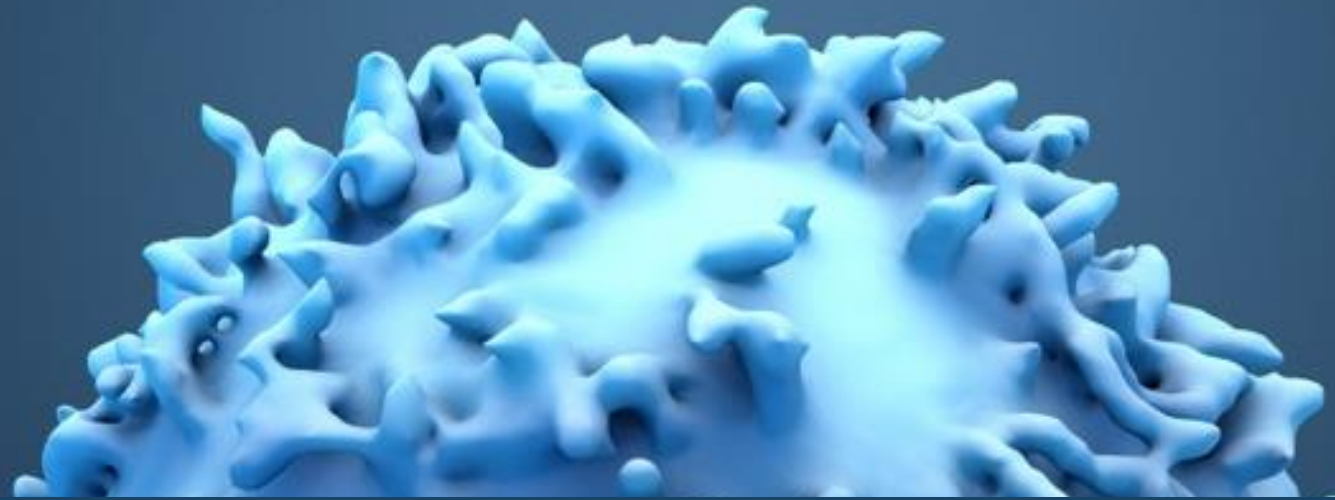
PET Scan (2 weeks after last dose)



A Primer on IL-2 Dosing and Treg Function: Lower IL-2 Dose is Better for Tregs



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



Phase 2 Low Dose Interleukin-2 (LD IL-2) in Patients with Mild to Moderate Alzheimer's Disease: A Randomized, Double-Blind, Placebo-Controlled Clinical Trial

An Investigator Initiated Trial Led by Dr. Alireza Faridar at Houston Methodist Hospital*



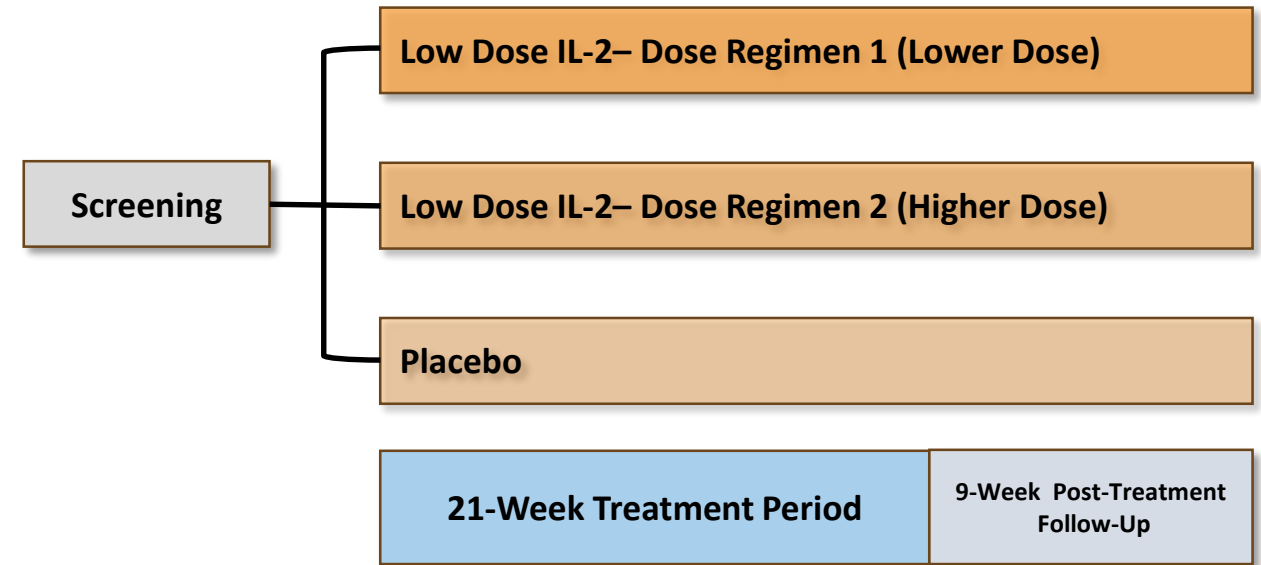
***Trial funded by Alzheimer's Association, Gates Foundation, NIA with support by Coya Therapeutics
IIT study using commercially available product**

Double-Blind, Placebo-Controlled Study in AD Patients Completed

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**

**Study funded by the Gates Foundation
and Alzheimer's Association**

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.

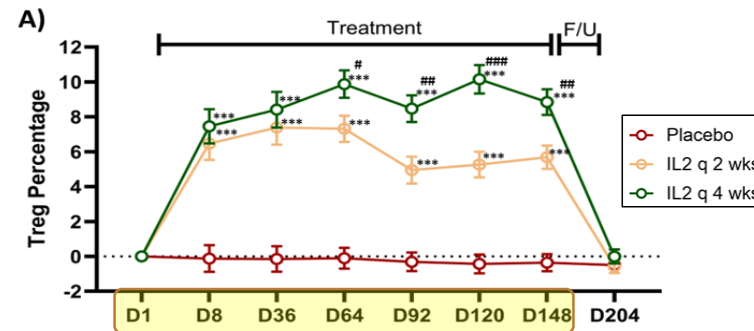
LD IL-2 in Mild to Moderate Alzheimer's Disease: Validated Lower Dose IL-2 is Better

The trial was designed to detect significant differences in primary and secondary endpoints:

Primary Endpoint: Safety and Tolerability

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

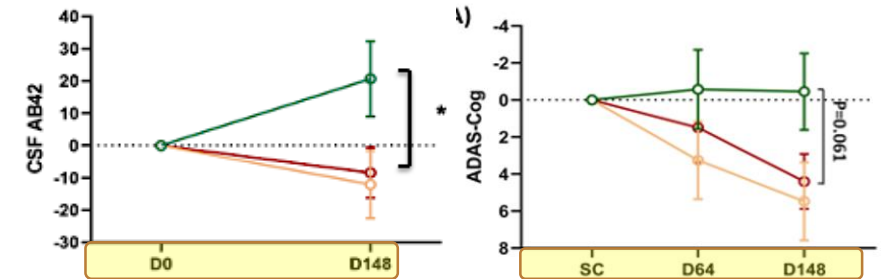
Secondary Endpoint: Treg Cell Populations



- 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
- Clinical scales, including the ADAS-Cog Score
- Assessment of peripheral/central inflammatory responses will be reported in the near future)

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

Key Catalysts and Milestones - 2025

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

Receipt: Non-Dilutive Milestone Payment

- Upon Acceptance and first patient dosing, eligible to receive milestone payments of \$8.4M from strategic partner, Dr. Reddy's Laboratories (DRL)

ALS Biomarker Data Release

- **2Q 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

Thank you!

Coya's Leadership Has Demonstrated Deep Expertise in Biotech

Management Team



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



Arun Swaminathan, Ph.D.
Chief Executive Officer



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



David Snyder
Chief Financial Officer & Chief Operating Officer



Michelle Frazier
Senior Vice President of Regulatory Affairs

Prior Experience*



Board of Directors



Wilbur Ross
Former U.S. Secretary of Commerce



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



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

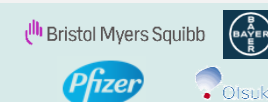


Dieter Weinand
Former Chairman of Board and CEO, Bayer Pharma, AG



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

Prior Experience*



SAB Comprised of Seminal Leadership in Tregs



Following the discoveries of Dr. Sakaguchi and Dr. Appel, Coya is developing multiple product modalities to enhance the therapeutic potential of Tregs for the treatment of disorders of high unmet need



Shimon Sakaguchi, MD, PhD

Distinguished Professor at the World Premier International Research Initiative (WPI)-Immunology Frontier Research Center (IFReC) at Osaka University

**Discovered Tregs
in 1995**



Stanley Appel, MD

*Former Chair of the Stanley H. Appel Department of Neurology
Director of the Ann Kimball & John W. Johnson Center for Cellular Therapeutics
Houston Methodist Hospital*

**Pioneered Role of
Tregs in
Neurodegeneration**