COYA THERAPEUTICS

Addressing Neuro-Inflammation through Therapeutic Intervention

Investor Overview
July 2025

Cautionary Note of Forward-Looking Statements and Disclaimers in this Presentation

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: 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

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 $35.5 million as of 03/31/2025 with runway into 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 ($8.4M anticipated in 2025)

Accomplished Management Team and Board

- Proven track record of execution and value creation

Presentation Overview

- Company Overview
- Neurodegenerative Overview
- The Coya Solution
- Therapeutic Pipeline
- Our Experienced Team
- Product Overviews (Coya 302, 301, 303)

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)

The Coya Solution

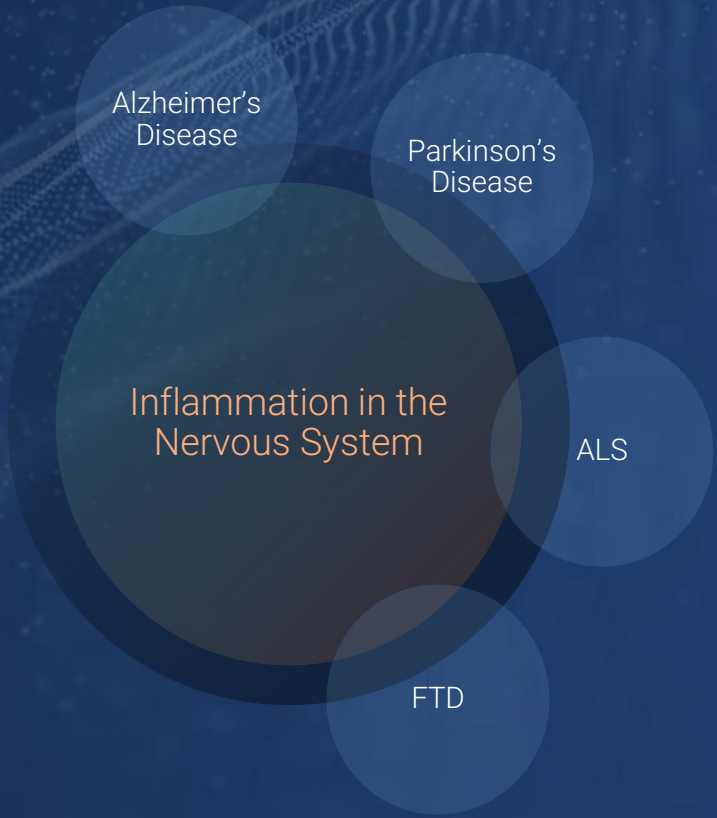
We will address neurodegeneration at its source by developing innovative therapies to meet the needs of patients with these diseases.

Combining Therapeutic Approaches

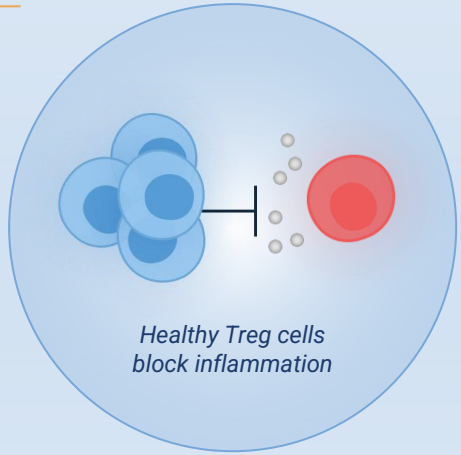
Neurodegenerative disorders impact people in a variety of ways. There are clear therapeutic benefits in focusing on the common underlying characteristics of these diseases, to develop a comprehensive treatment that benefits a range of patients.

Harnessing Tregs

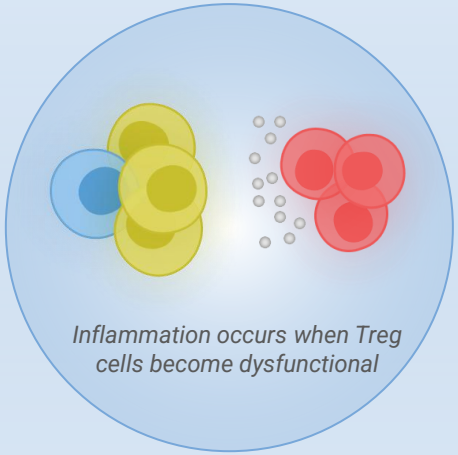
Tregs manage your immune system's response to sickness, and play a key role in helping you feel better. In applying our knowledge of Tregs, we will empower patients' internal first responders with support to tackle these diseases.



Intro to TRegs



Healthy Treg cells block inflammation



Inflammation occurs when Treg cells become dysfunctional

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.

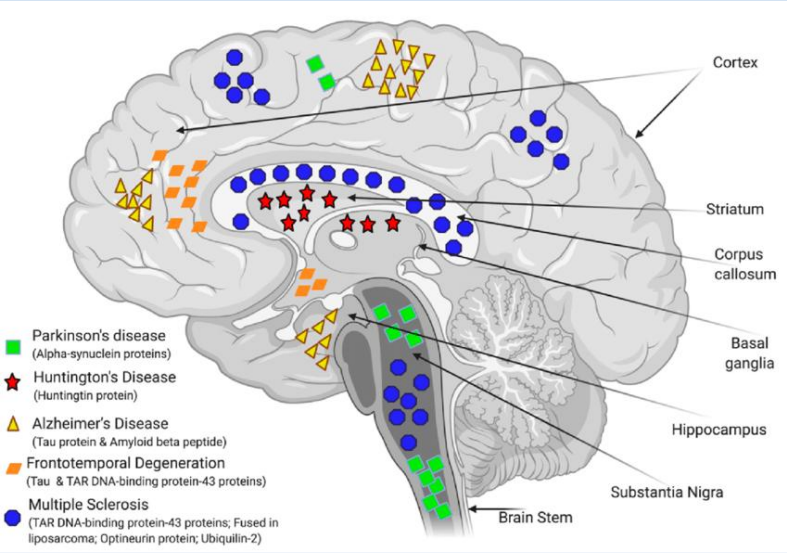
How to treat disease by targeting Tregs?

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

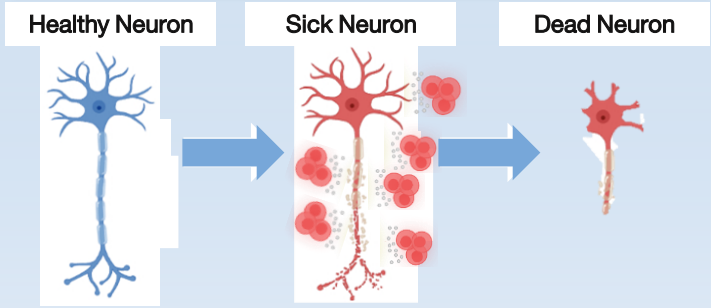
Inflammation - A Critical Role in Neurodegeneration

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

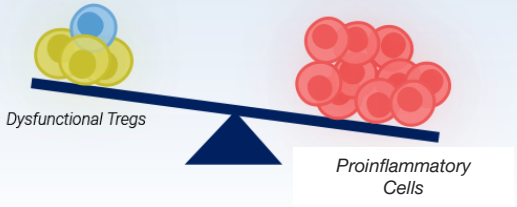
Neurodegeneration:
Loss of Selective Population of Neurons



Inflammation in the Nervous System



Regulatory T Cells (Tregs) *play a central role in lowering inflammation and stemming the progression of neurodegeneration*



Bioengineering 8(2):30
DOI:10.3390/bioengineering8020030

Introducing Coya 302

Our Lead Therapy - Coya 302

Our research indicates that by decreasing inflammation and increasing Treg suppressive function we can unlock a therapeutic pathway that may be more effective than a singular solution

Boost the Good

Strengthen the function of Tregs, giving them a much needed boost.

Combine for Success

These approaches are most effective in combination, working to control inflammation.

Reduce the Bad

Block harmful immune responses to stop inflammation in its tracks.

LD IL-2 + CTLA4-Ig Investigator Initiated Clinical Trial (ALS)

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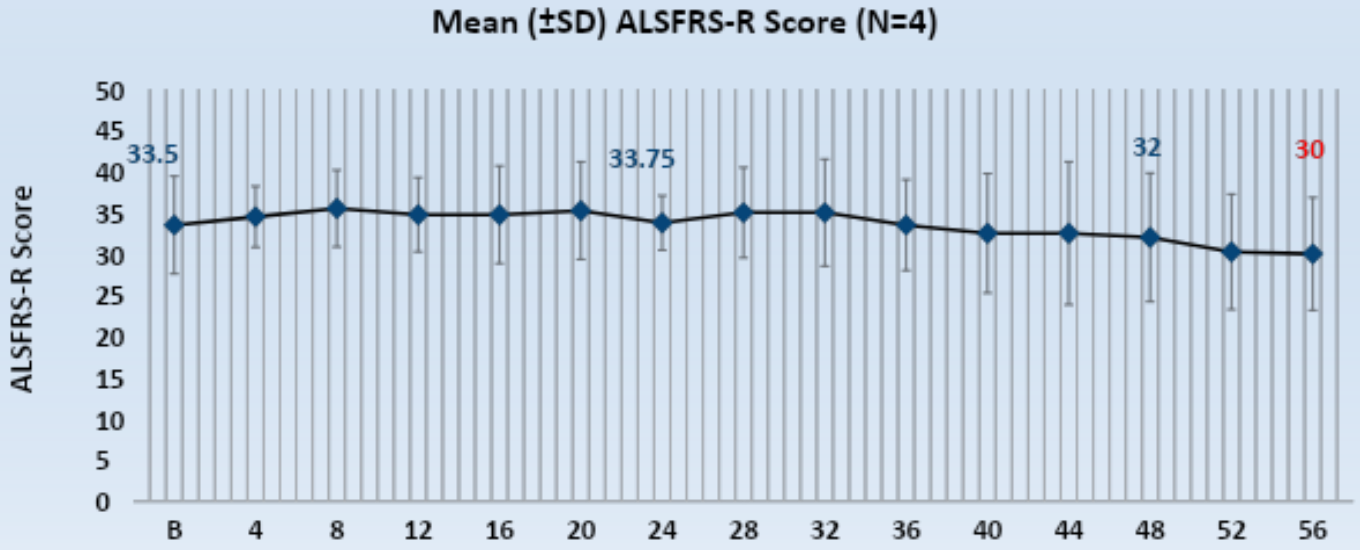
Trial in Patients with ALS, appears to Ameliorate ALS Progression Over 48 Weeks



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.

B: Baseline
n.s.: not significant (paired t test)
Conducted using commercially available products

Interim Results in 5 patients with Frontotemporal Dementia

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Patients treated with low dose IL-2 and CTLA-4 Combination in Investigator Initiated Clinical Trial

Treg numbers and suppressive function increased after the first treatment cycle ($p < 0.01$ and $p < 0.05$, respectively), and remained at higher significant levels throughout the treatment period.

The Mean Scores in FTD patients treated with low-dose IL-2 and CTLA4-Ig combination exhibited minimal to no cognitive decline over the course of the study, compared to pre-treatment baseline values.

LD IL-2 + CTLA4-Ig was well tolerated over 22 weeks; the most common AE was mild injection site reaction. no deaths or serious AEs (SAEs) occurred over the course of the study.

Interim results n=5; enrollment target is 10
Conducted using commercially available products

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

HOUSTON Methodist LEADING MEDICINE

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

COYA THERAPEUTICS

Dr. Reddy's

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

Coya 302 Program Pipeline

Product Type	Discovery	Preclinical	IND-Enabling	Phase 1	Phase 2	Phase 3	Partnerships
ALS *Amyotrophic Lateral Sclerosis*	COYA 302 (Low Dose IL-2 + CTLA4-Ig)						Licensing Transaction on 12/6/23 with Dr. Reddy's Laboratories
FTD *Frontotemporal Dementia*	COYA 302 (Low Dose IL-2 + CTLA4-Ig)						Retained Worldwide Rights
AD *Alzheimer's Disease*	COYA 302 (Low Dose IL-2 + CTLA4-Ig)						Retained Worldwide Rights
PD *Parkinson's Disease*	COYA 302 (Low Dose IL-2 + CTLA4-Ig)						Retained Worldwide Rights

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 ➢ **Upon Acceptance and first patient dosing:** eligible to receive non-dilutive 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

Additional Pipeline Assets

Product Type	Discovery	Preclinical	IND-Enabling	Phase 1	Phase 2	Phase 3	Key Milestones
Coya 301 *Proprietary Low Dose IL-2*	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 *COYA 301 + GLP-1 Agonist*	COYA 303 Undisclosed						**1H 2025:** Additional IP filings Publish preclinical in-vitro data Initiate preclinical translational study Partnership discussions
Coya 201 *Allogeneic Treg Derived Exosomes*	COYA 201 Undisclosed						**2025:** Pre-clinical characterization
Coya 206 *Antigen-Directed Allogeneic Treg-Derived Exosomes*	COYA 206 Undisclosed						**2025:** Pre-clinical characterization

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.

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

Appendix

- Coya 302 Overview
- Coya 302 in ALS
- Coya 302 in FTD
- Coya 301 in AD
- Coya 303 (Coming Soon)

COYA THERAPEUTICS

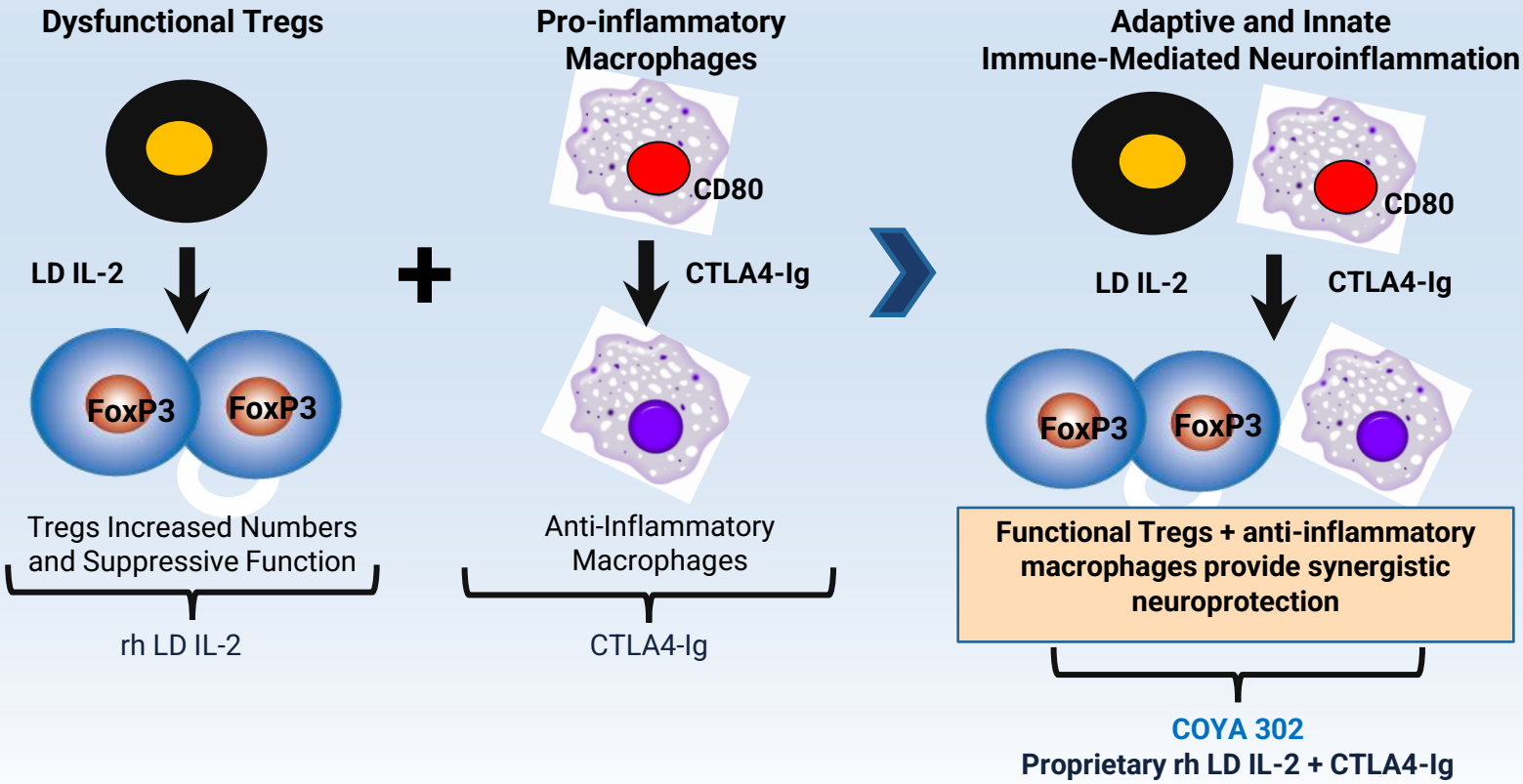
LEAD PRODUCT

COYA 302

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

The Future of Neurodegenerative Disease Therapy

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

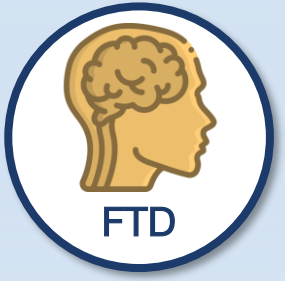


Coya 302 is a Pipeline within a Product with > $10BN Potential

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

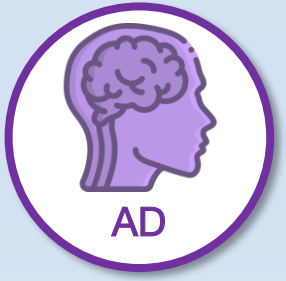
ALS or Amyotrophic Lateral Sclerosis and is an Orphan Indication

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



FTD or Frontotemporal Dementia and is an Orphan Indication

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



AD or Alzheimer's Disease

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

PD or Parkinson's Disease

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

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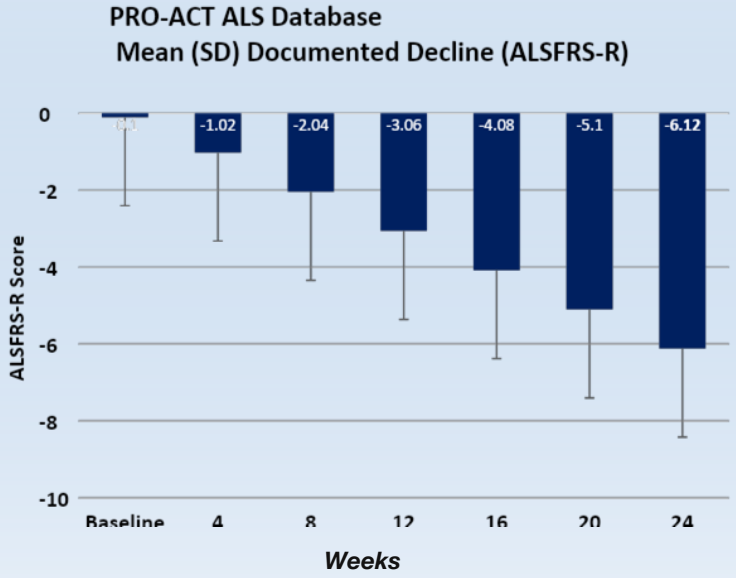
COYA 302 in ALS

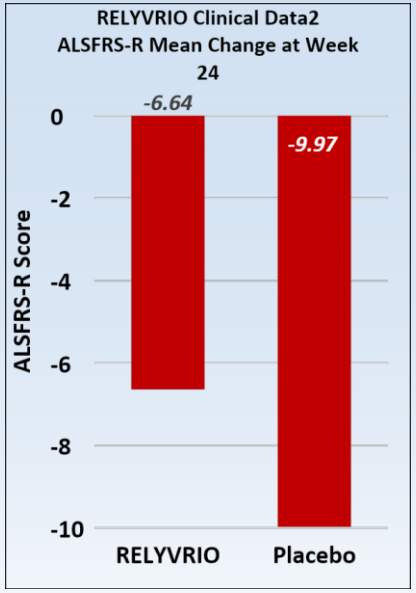
Proprietary, Recombinant Human Low Dose Interleukin-2 (rh LD IL-2) and CTLA4-Ig

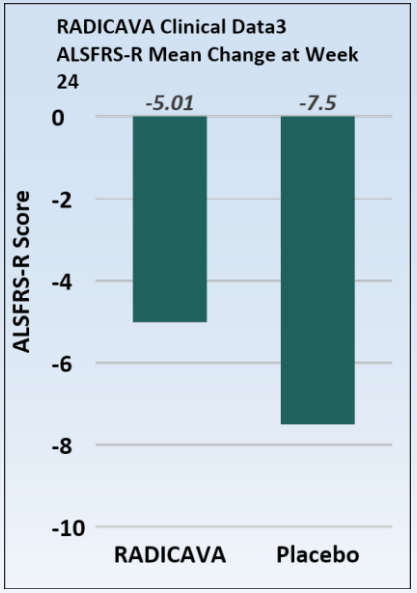
Current Therapies for ALS aim to slow disease progression

Average rate of patient decline is 1.02 points/month in ALSFRS-R score[1]



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	Treatment Period	Follow-Up
20 weeks	***LD IL-2 + CTLA4-Ig** was administered via subcutaneous injection over 48 weeks*	*8 weeks*

Screening Assessments

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

Treatment Period Assessments

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

Post-Treatment Assessments

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

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

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

** Conducted using commercially available products*

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

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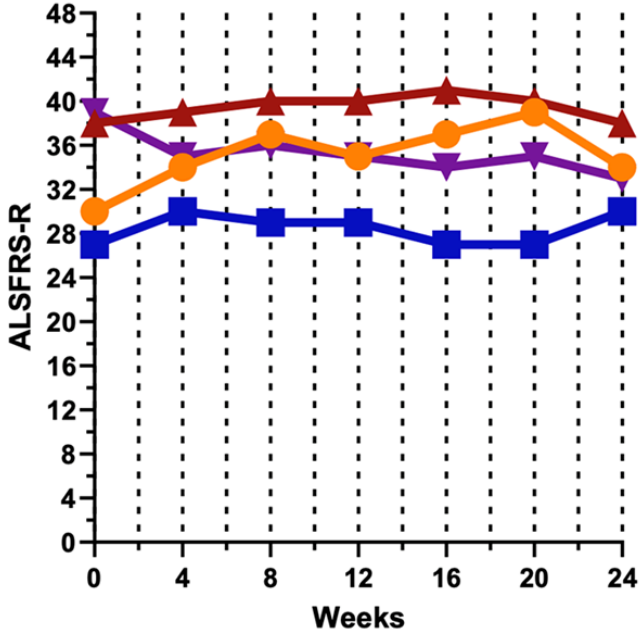
Baseline Characteristics

	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 remained mostly stable or improved



Participant 1: +4 points
Participant 2: +3 points
Participant 3: Stable
Participant 4: -6 points*

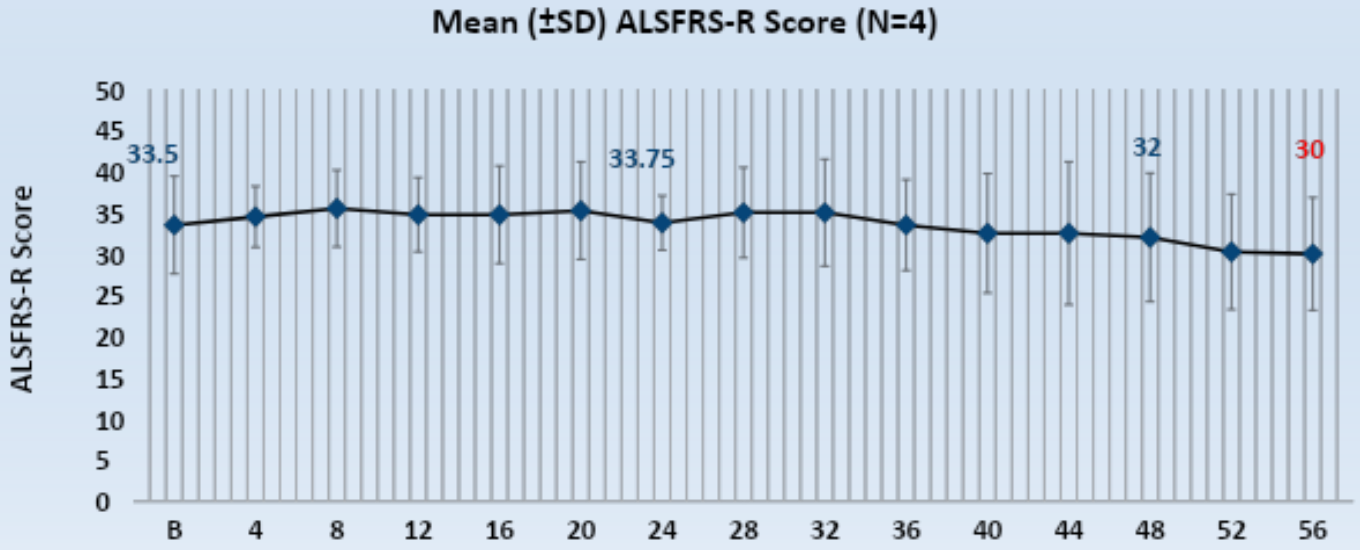
Participant #1
Participant #2
Participant #3
Participant #4

** 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.

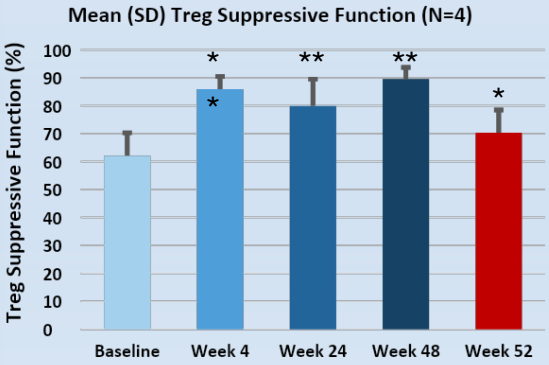
B: Baseline
n.s.: not significant (paired t test)
Conducted using commercially available products

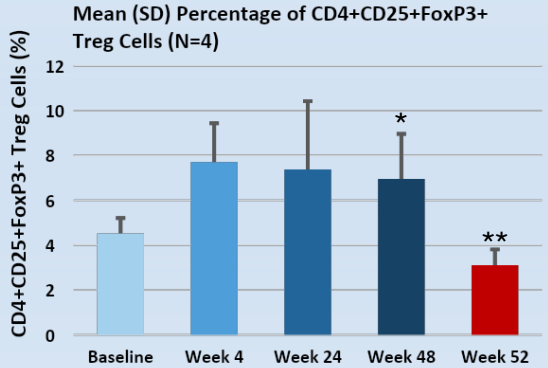
LD IL-2 + CTLA4-Ig Investigator Initiated Clinical Trial

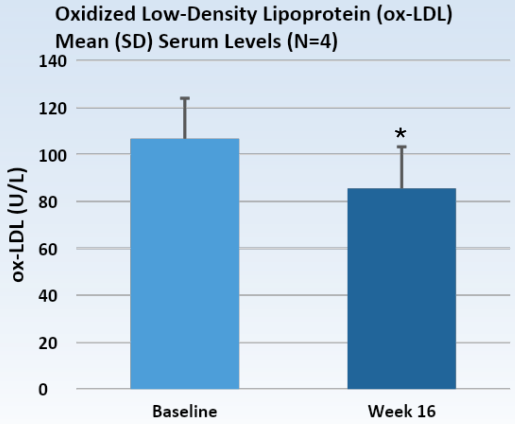
Lowered Oxidative Stress Markers

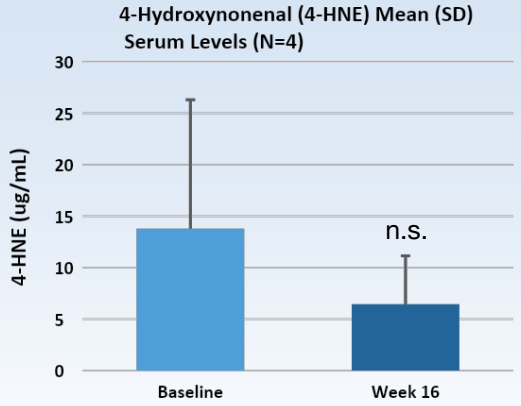
Key Takeaways

- ✓ **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.









n.s.: not significant (paired t test)
**p <0.05*
*** p <0.01 (paired t test)*

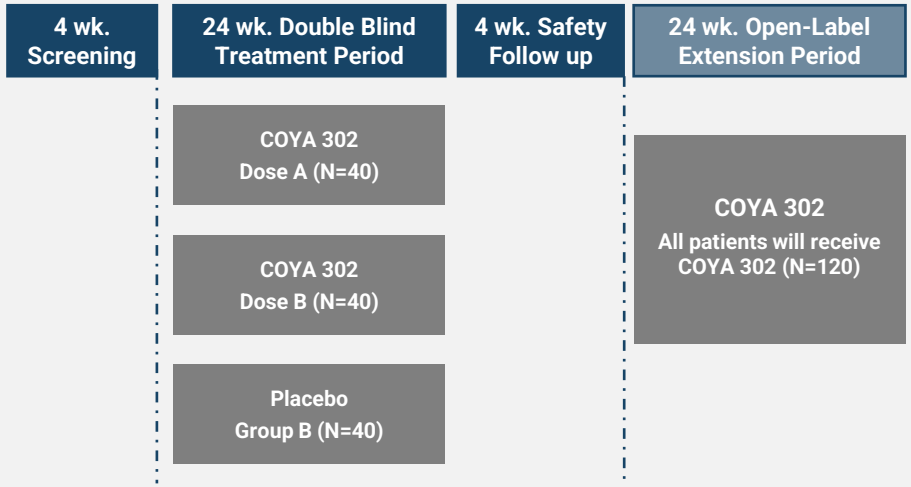
Overview of Phase 2 Study Design in ALS

Trial will be conducted starting later in H2-2025

Key Inclusion Criteria

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

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COYA 302 in FTD

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

Partnership with Alzheimer's Drug Discovery Foundation

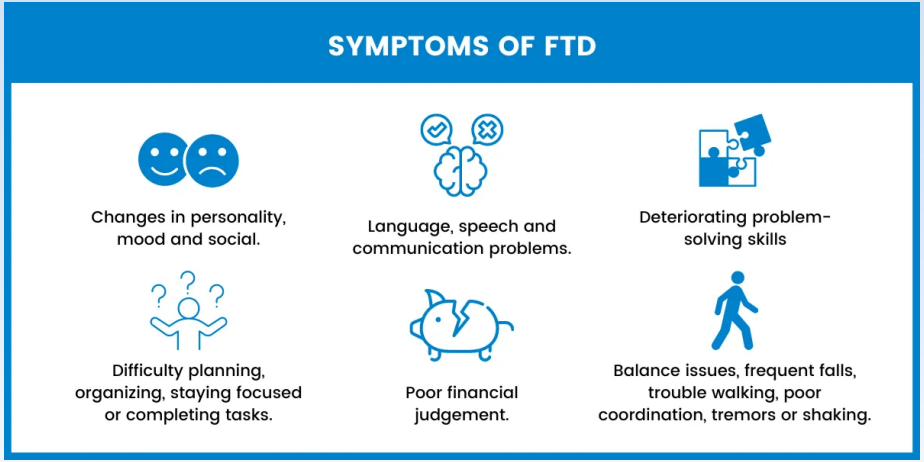
$5M Investment to accelerate Phase 2b trial in FTD

Alzheimer's Drug Discovery Foundation

Study Title

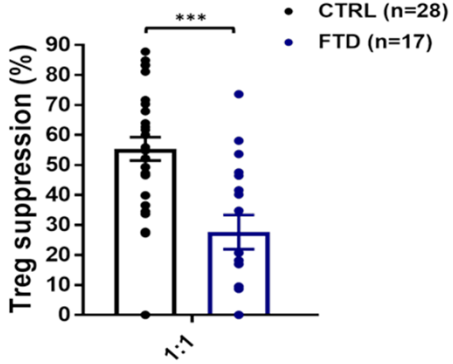
A Phase II, Randomized, Double-Blind, Placebo-Controlled, Multi-Center, 26-Week Study to Evaluate the Safety and Efficacy of COYA 302 for the Treatment of Nonfluent Variant Primary Progressive Aphasia (nfvPPA) Subtype of Frontotemporal Lobar Degeneration (FTLD)

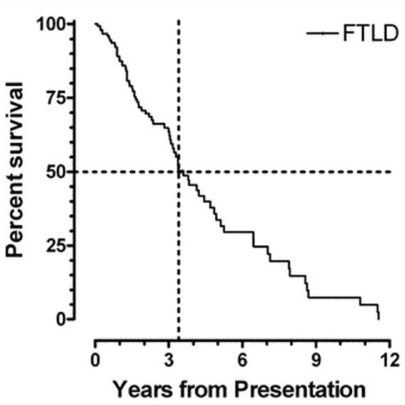
Frontotemporal Disorder (FTD) is a Rare Disease and is one of Most Common Dementias in Younger People. Frontotemporal disorder progresses to death faster than Alzheimer's disease, and, unfortunately, there is no effective treatment

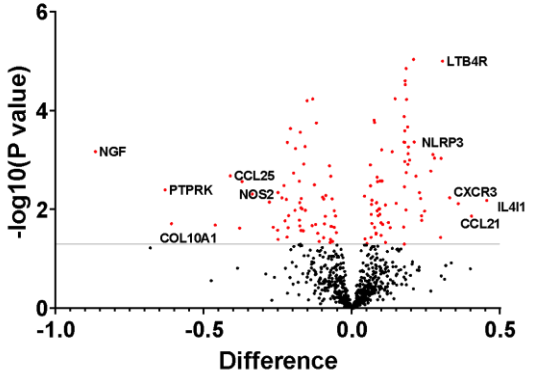


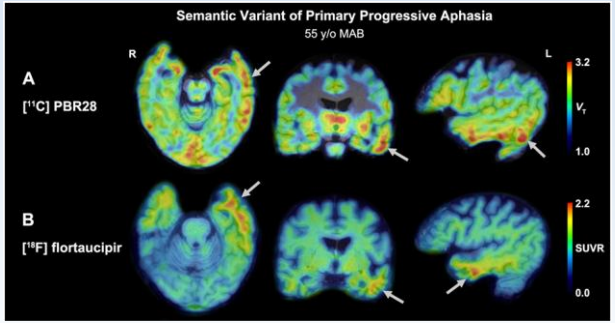
Exacerbated Systemic and Central Inflammatory Responses

FTD is a devastating disease that unfortunately has no effective treatment

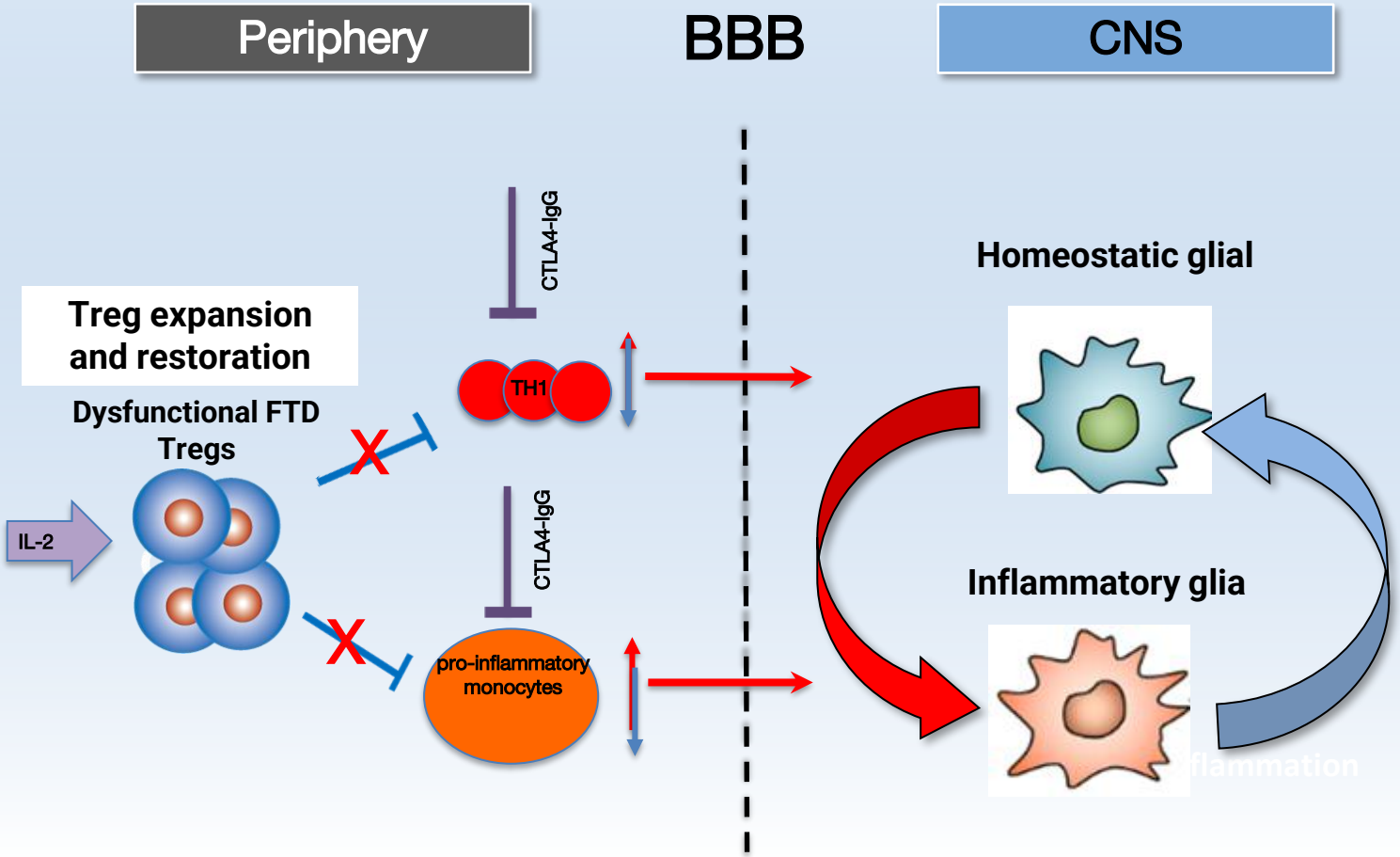






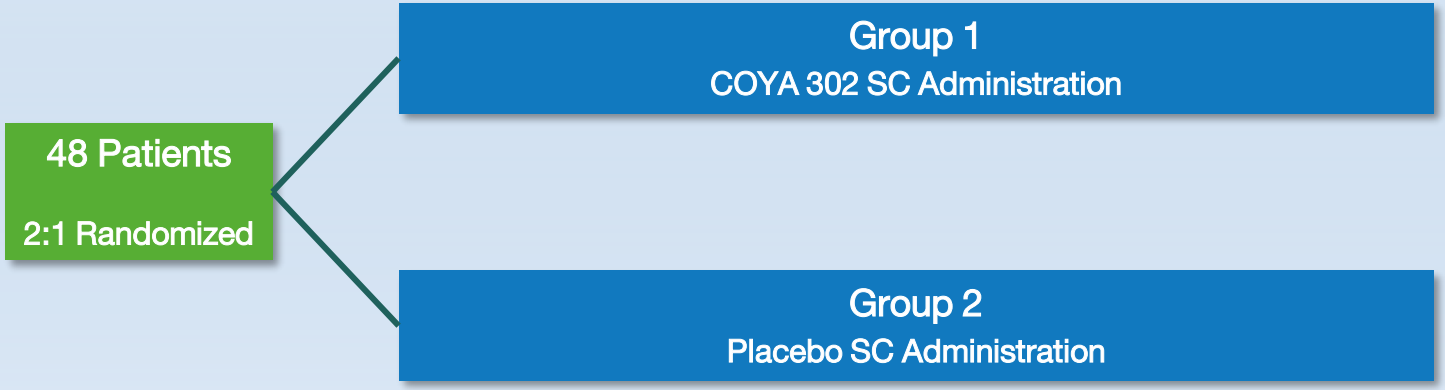


Targeting Inflammation in FTD to Slow Disease Progression



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

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

COYA THERAPEUTICS

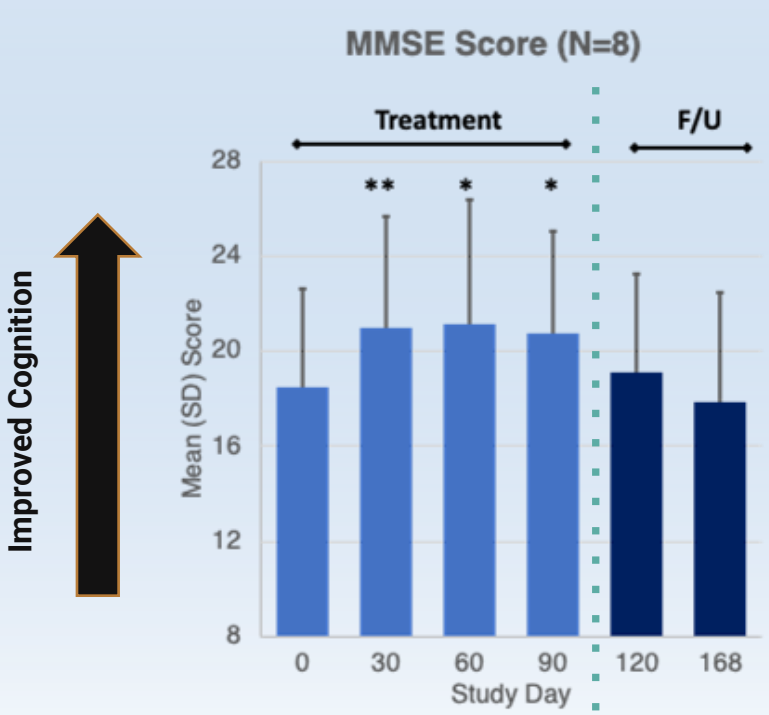
PIPELINE PRODUCT

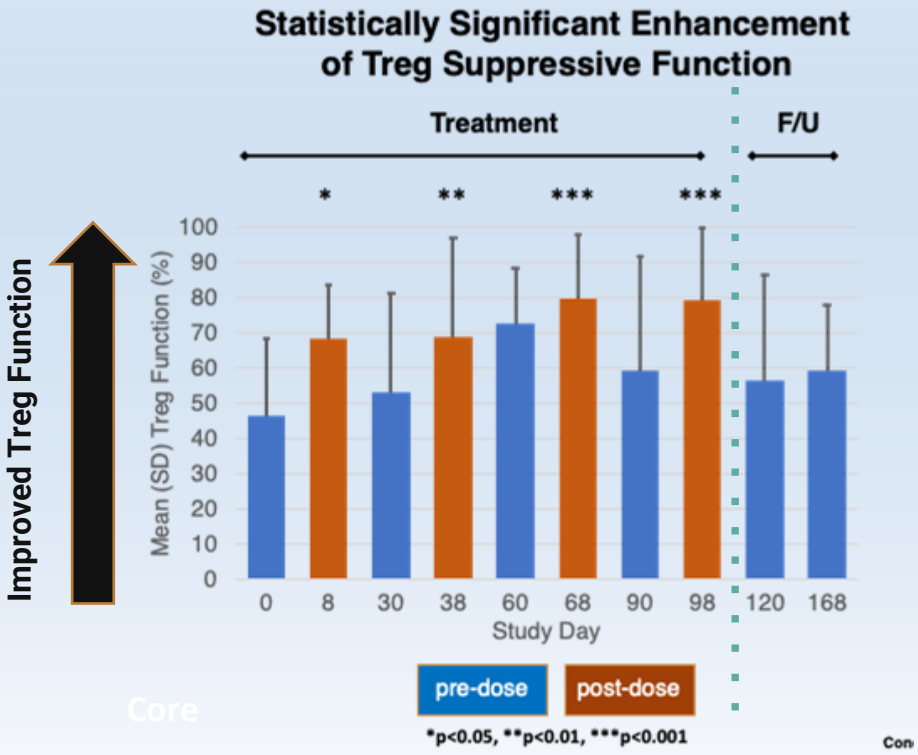
COYA 301

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





Translational Neurodegeneration **volume 12**, Article number: 54 (2023)

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

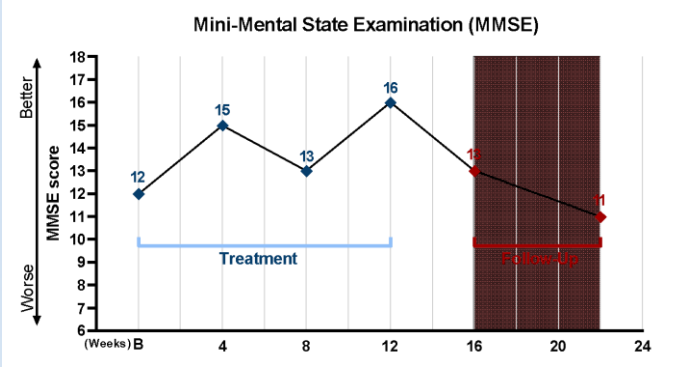
70-Year-Old Male

Baseline MMSE: 12

Positive PIB PET Scan

Positive Neurodegeneration MRI

Other Rx: Donepezil 10mg

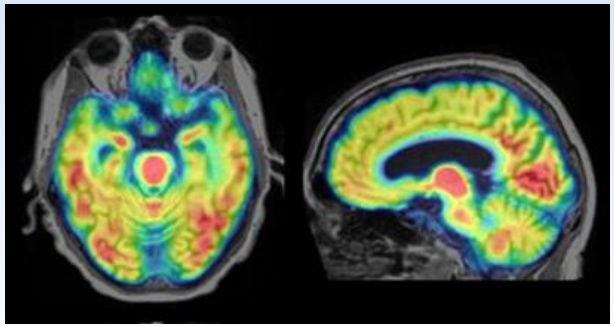


Neuroinflammation (TSPO)
(^{11}C) ER-176 PET Scan at baseline and after 4-monthly cycles of low dose IL-2.

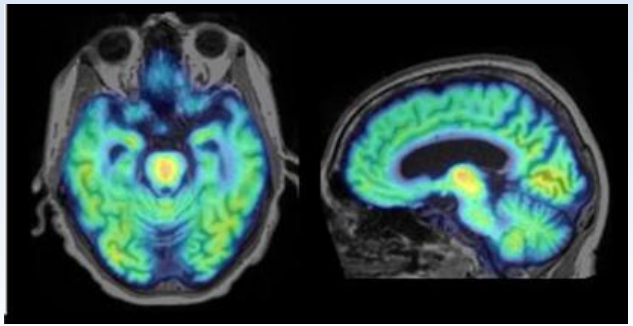
The second PET scan was done 2 weeks after last dose.

PET: Positron Emission Tomography
TSPO: translocator protein

PET Scan (Baseline)

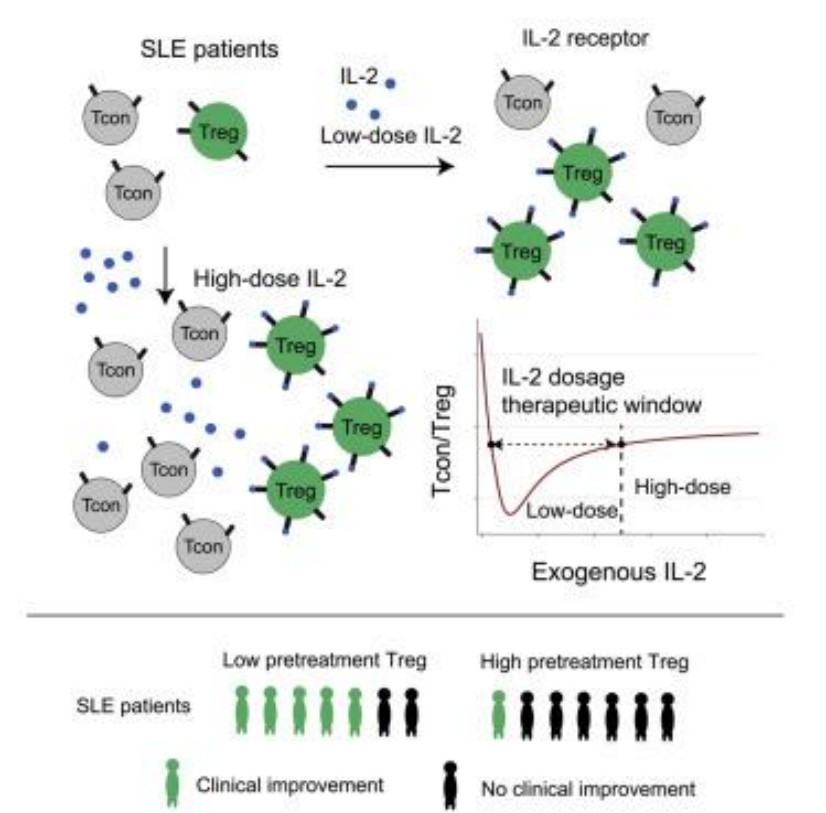


PET Scan (2 weeks after last dose)



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

iScience; Volume 25, Issue 9104911 September 16, 2022

COYA THERAPEUTICS

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

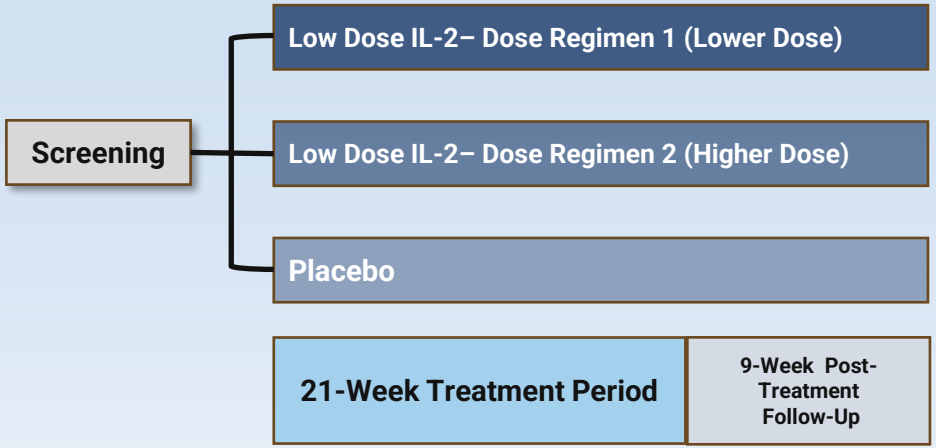
Double-Blind, Placebo-Controlled Study in AD Patients

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**

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

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

COYA THERAPEUTICS

✓ 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 Aβ42 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 AIzheimer 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

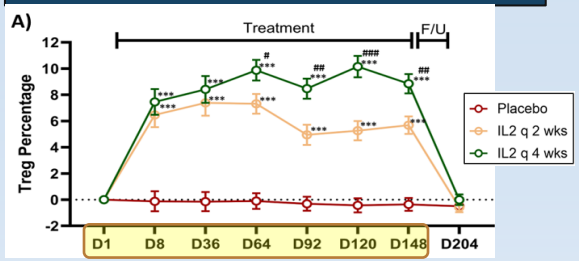
COYA THERAPEUTICS

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

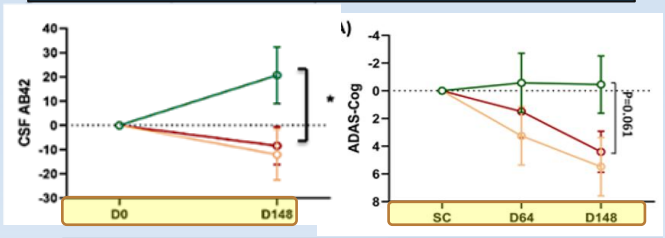


✓ Significant change in Treg population following two different dosing frequency regimens of LD IL-2 treatment, compared to placebo

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 THERAPEUTICS

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 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:

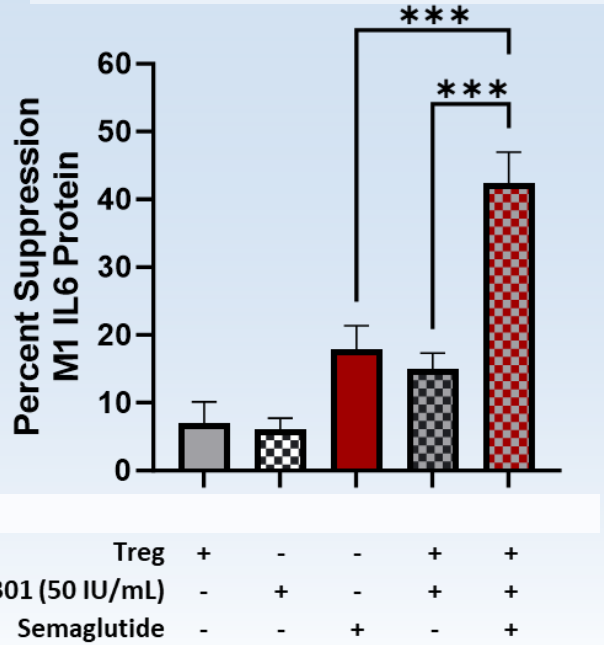
- In vitro suppression assays (activated macrophages and responder T cells)
- Preclinical model (Parkinson's disease)
- Potential exposure study in patients at Houston Methodist Hospital

The combination produced a statistically significantly higher Treg suppressive effect on pro-inflammatory myeloid cells and enhanced Treg survival in in vitro human immune cells, compared to the individual components - LD IL-2 and GLP-1RA

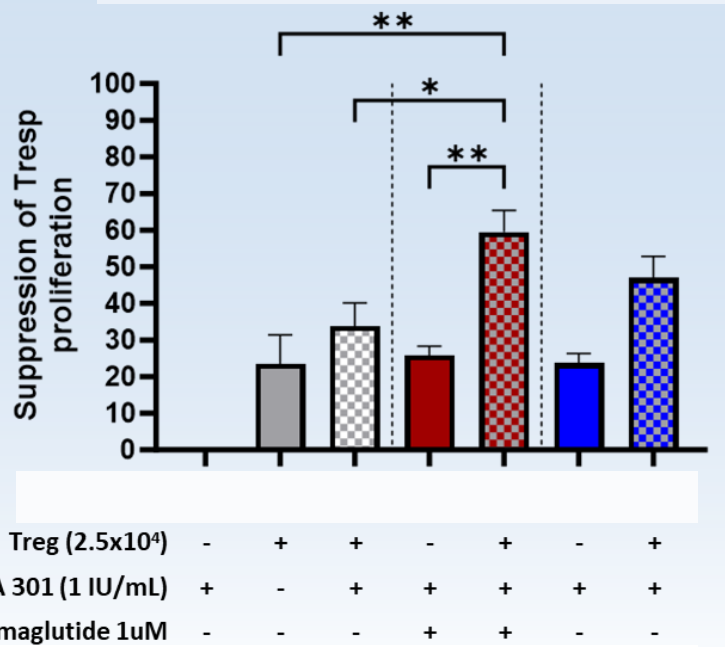
Evaluation of COYA 301 and GLP-1RA combination

COYA 301 and GLP-1RA combination suppresses proinflammatory macrophages and responder T cell proliferation in vitro

Suppression of Activated Macrophages



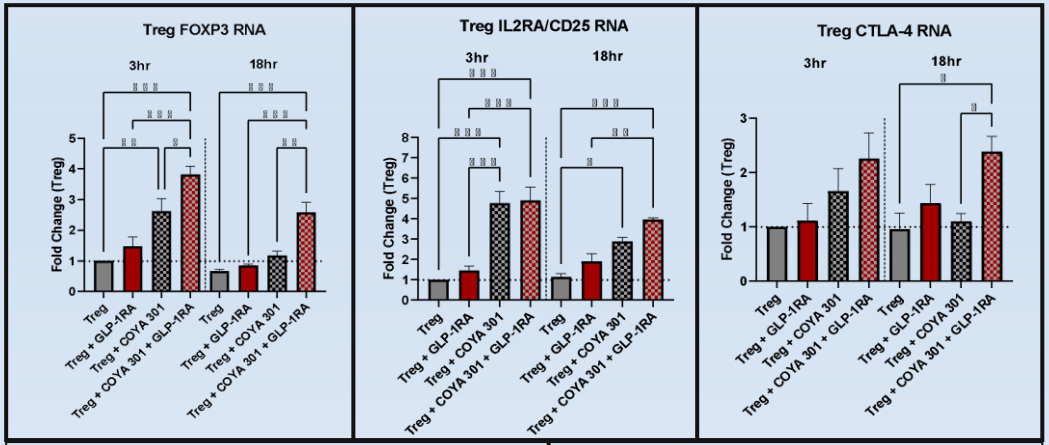
Suppression of Responder T Cell Proliferation



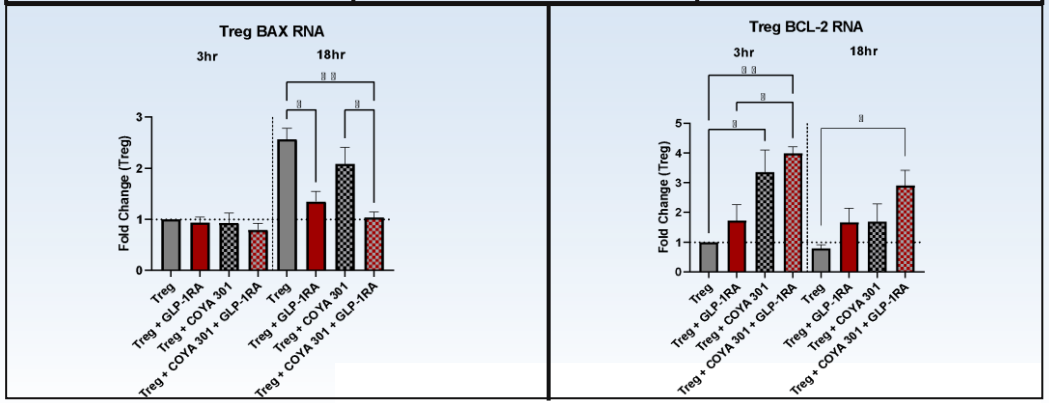
Evaluation of COYA 301 and GLP-1RA combination

COYA 301 and GLP-1RA combination increases mRNA transcripts associated with Treg function and survival

Upregulation of Treg survival and function

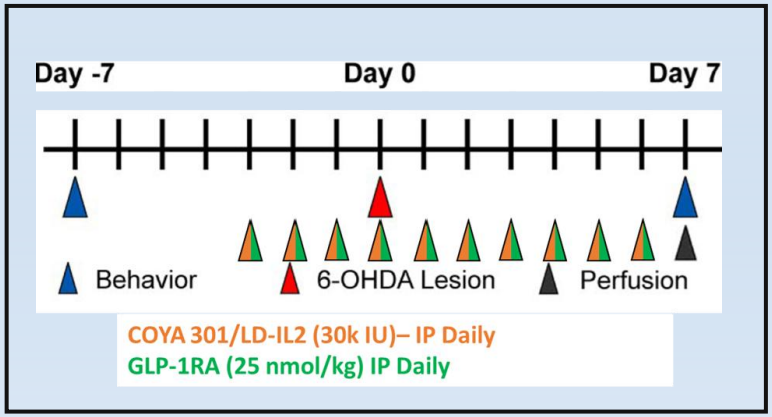


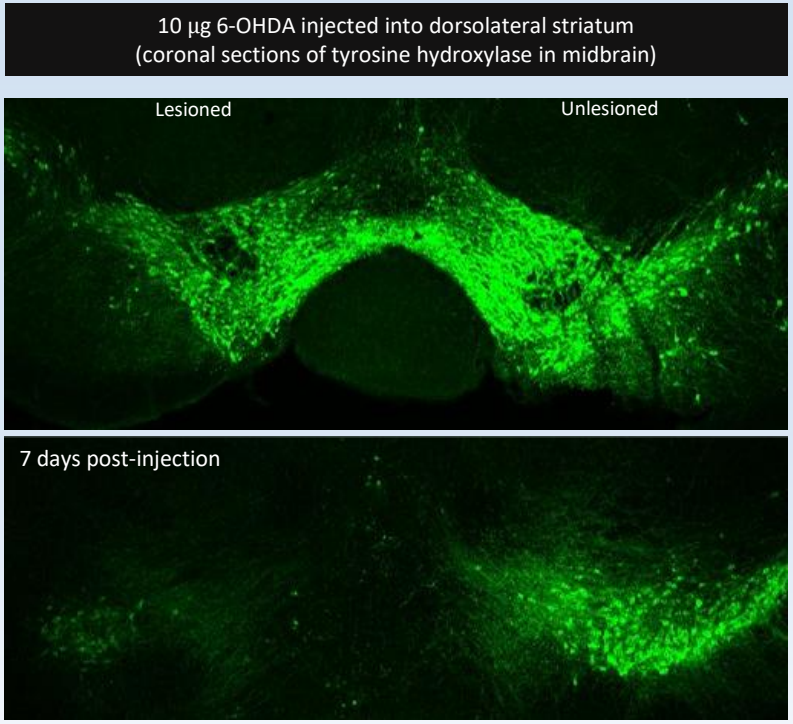
Protection of Tregs from apoptosis



Evaluation of COYA 301 and GLP-1RA combination

COYA 301 and GLP-1RA in a mouse model of Parkinson's disease





6-OHDA model of PD with the following endpoints:

1. Dopaminergic neuronal loss
2. Rescue of motor/behavior phenotype
3. Neuroinflammation (microgliosis/astrogliosis)
4. Peripheral T cell population

Coya Teams

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

Expert Leadership Team

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

David Snyder
Chief Financial Officer &
Chief Operating Officer

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Actinium Pharmaceuticals Inc.

Alteogen Inc.

Johnson-Johnson

Sunovion

Bristol Myers Squibb

Mesoblast

Eli Lilly

Glenmark

DisperSol Technologies

Exicure

Cellular Dynamics International

Roche NimbleGen

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Revance Therapeutics

CytomX

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Coherus BioSciences

Karen King
Senior Vice President of
Program Management
and Clinical Operations

<– Prior Experiences –>

Recursion

MacroGenics

Supernus

Shire

MedImmune

Daniel Barvin
Vice President of Operations
& Patient Advocacy

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

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

Discovered Tregs in 1995

Clive Svendsen, Ph.D.
Director, Cedars-Sinai Regenerative Medicine Institute

Professor in Residence at UCLA

Consulting Professor at Stanford University

Renowned expert on stem cell biology and regenerative therapeutic approaches

Malcolm Brenner, M.D., Ph.D.
Director, Center for Cell and Gene Therapy

Professor, Baylor College of Medicine

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

Stanley Appel, M.D.
Co-Director, Houston Methodist Neurological Institute

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

<– Prior Experiences –>

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COYA THERAPEUTICS

Thank You!