COYA THERAPEUTICS

INVESTOR OVERVIEW I November 2026

Combating Neurodegeneration Via Treg-Directed Combination Therapy

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, without limitation, 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, and our financial position, including cash runway. The words "believe," "may," "will," "estimate," "continue," "anticipate," "intend," "expect," and similar expressions are intended to identify forward-looking statements, though not all forward-looking statements necessarily contain these identifying words.

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 does not proceed on estimated timelines; 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. The actual results may vary from the anticipated results, and the variations may be material. Other factors that may cause the Company's actual results to differ from current expectations are discussed in the Company's filings with the U.S. Securities and Exchange Commission, including the section titled "Risk Factors" in the Company's Annual Report on Form 10-K for the year ended December 31, 2025. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date this presentation is given. Except as required by applicable law, 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.

This presentation discusses product candidates that are under clinical study and have not yet been approved for marketing by the U.S. Food and Drug Administration. No representation is made as to the safety or effectiveness of these product candidates for the use for which such product candidates are being studied.

Coya Therapeutics, Inc. (COYA): Investment Highlights

Focused Anti-Neurodegenerative Pipeline

COYA 302 (Low Dose IL-2 + CTLA4-Ig): A pipeline in a product across ALS, FTD, AD, Parkinson's Disease. COYA 303 (Low Dose IL-2 + GLP-1 RA): Alzheimer's Disease

Near-Term COYA-302 ALS Data Readout Early Q2 2027

ALSTARS Phase 2/3 enrollment completed in October 2026, OLE initiation expected November 2026, topline data expected early Q2 2027

Established Clinical Signal in Two IIT Studies of Low Dose IL-2 + CTLA4-Ig

Data show ALSFRS-R stabilization over 48 weeks in ALS and cognitive stability at 22 weeks in FTD

Nobel-Recognized Scientific Foundation

SAB member Dr. Shimon Sakaguchi was among the recipients of the 2025 Nobel Prize in Physiology or Medicine for Treg biology

Dr. Reddy's Partnership Provides Strategic Support and Validation

Up to ~$700M in potential milestones plus mid-teen royalties for global ALS rights (excluding Japan and LATAM) and a $10M equity investment (closed in January 2026)

Cash Runway Through Upcoming Key Milestones

~$35.8M as of September 30, 2026, funding operations into 2H 2027, past expected ALS data readout

LD IL-2 Combination Therapy: 3 Pillars For Success

The neurodegeneration field increasingly recognizes combination-based therapeutic strategies such as Coya's as a viable path forward for diseases driven by complex, multifactorial biology

Validated Science

Nobel Prize–recognized for discovery & role of Treg Biology[1]

Well-established association between Treg dysfunction and ALS/FTD[2]

Independent scientific support of Coya's approach in ALS[3]

Differentiated Combination-based Therapeutic Approach

Combination therapy potentially increases likelihood of success

Endorsed by external thought leaders (KOLs, ADDF, CTAD)

Well-established therapeutics (low-dose IL-2 & Abatacept)

Low-dose IL-2 used at ~50x lower doses vs standard IL-2 dosing

CTLA4-Ig's safety and efficacy established in millions of patients in RA and other inflammatory and autoimmune conditions

Early Clinical Validation

Combination therapy restores and maintains Treg function

Signals of disease stabilization in ALS & FTD

Potential for combo therapies to alter disease trajectory

ALS IIT: no ALSFRS-R decline at 6 months, minimal decline at 1 year (n=4)

FTD IIT: cognitive stability at 6 months (n=9)

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1. *The 2025 Nobel Prize in Physiology or Medicine awarded to Dr. Shimon Sakaguchi, member of Coya SAB*
2. *Appel et al, ALS patients' regulatory T lymphocytes are dysfunctional, and correlate with disease progression rate and severity, JCI Insight, 2017*
3. *Sette et al, Autoimmune response to C9orf 72 protein in amyotrophic lateral sclerosis, Nature, November 2025*

COYA Targets T-Regs Role in Inflammation

Tregs are specialized immune cells that help maintain a balanced immune system

1

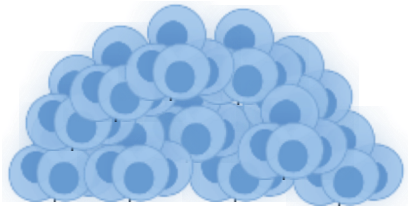
T-reg dysfunction leads to immune activation and chronic inflammation

2

Inflammation drives autoimmune pathology and neuro-degenerative disease progression

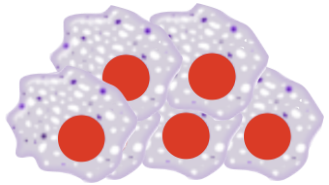


Dysfunctional Tregs

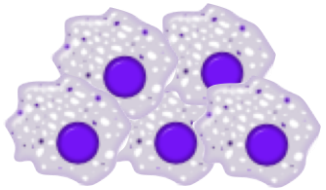


Functional Tregs

COYA: Low Dose IL-2 + CTLA-4Ig / GLP-1 RA



CD80 pro-inflammatory macrophages



Anti-inflammatory macrophages

3

Low dose IL-2 expands T-reg numbers and enhances their suppressive function

4

CTLA-4Ig promotes a shift of myeloid cells toward an anti-inflammatory phenotype

The result is re-balanced immune homeostasis, which leads to neuro-protection against neuro-degenerative disease

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ALS and FTD Market Opportunity

Two orphan diseases with short life expectancy from diagnosis and significant loss of quality of life

Amyotrophic Lateral Sclerosis

ALS

Patient Population & Unmet Need

~33,000 patients in U.S. (NIH)

Partner & Deal Terms

Dr. Reddy's Laboratories: global rights, excluding Japan and LatAm

Up to ~$700M in R&D, regulatory, and sales milestones; mid-teen royalties

$10M strategic investment (Jan. 2026)

Clinical & Regulatory Status

Fast Track Designation granted to COYA 302 in ALS

Phase 2/3 ALSTARS study ongoing, topline expected early Q2'27

Frontotemporal Dementia

FTD

Patient Population & Unmet Need

50,000–60,000 patients in U.S. (Alzheimer's Association)

Development and Commercial Rights

COYA retains 100% development and commercialization rights to COYA 302 in FTD and all other indications, excluding ALS

Third-Party Support

Alzheimer's Drug Discovery Foundation (ADDF): $5M investment (May 2024) to support Phase 2 trial in FTD

Clinical & Regulatory Status

IND accepted Q1 2026

Study initiation expected 2027

COYA 302 in ALS

ALS: An Urgent Need for New Treatment Options

ALS is a progressive disease that attacks the motor neurons in the brain and spinal cord that control voluntary muscles — including those needed to speak, swallow, and breathe[1]

20–30%

die within two years of diagnosis, despite approved therapies[2]



90%

develop “sporadic” ALS with no family history[3] and therefore no known cause

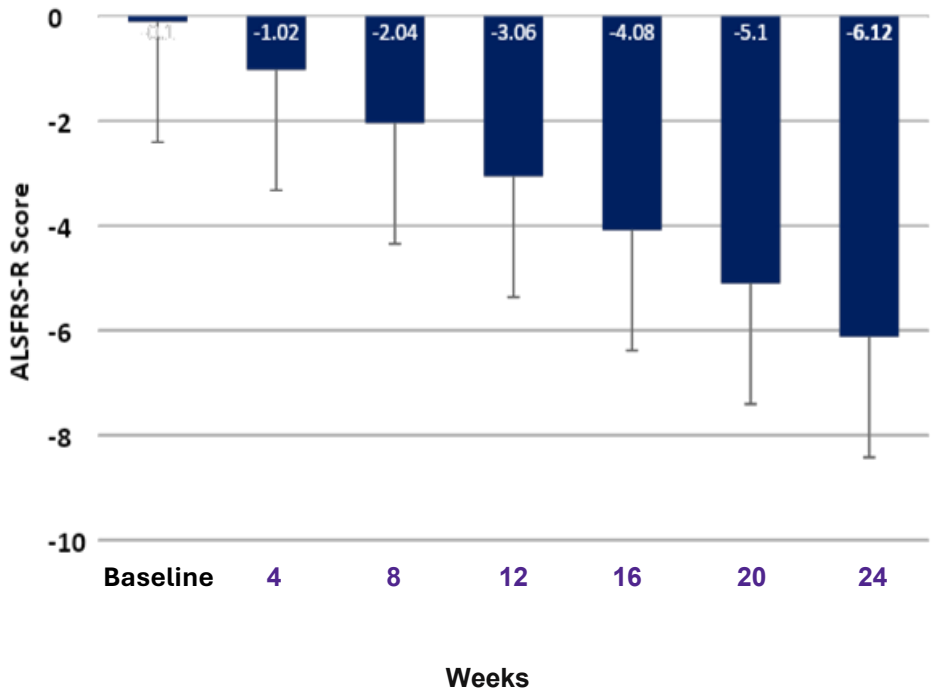


1 https://www.als.org/understanding-als/stages; 2 Brown RH, Al-Chalabi A. Amyotrophic lateral sclerosis. The Lancet Neurology (2017); 3 Masrori P, Van Damme P. Amyotrophic lateral sclerosis: a clinical review. Eur J Neurol. 2020;27(10):1918–1929. doi:10.1111/ene.14393

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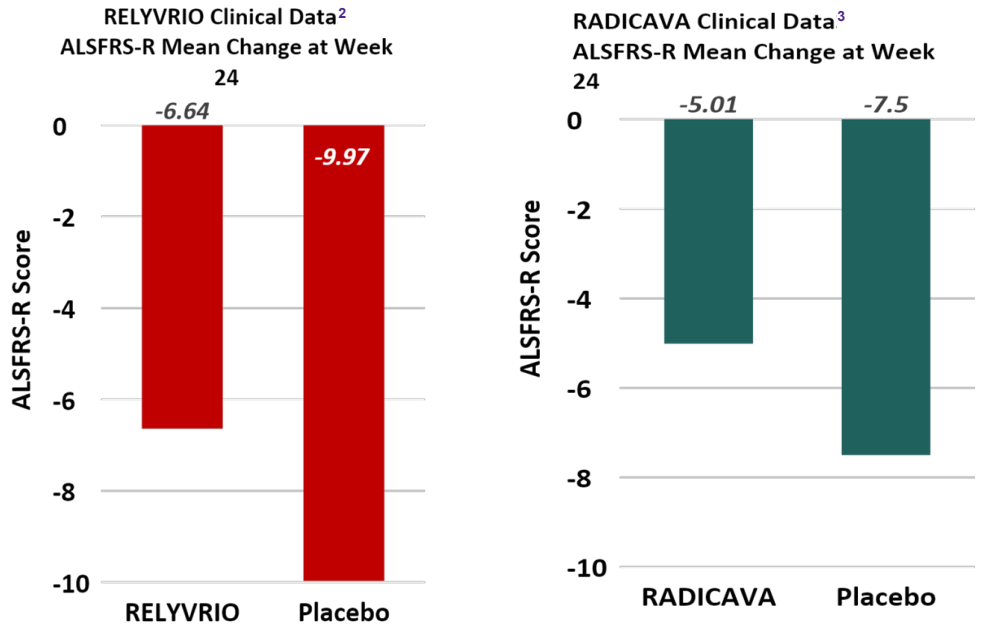
Approved Therapies Show Only a Modest Treatment Effect

PRO-ACT ALS Database Mean (SD) Documented Decline (ALSFRS-R)



In the natural history of ALS, patients average a ~1 point/month rate of decline in ALSFRS-R score[1]

RELYVRIO and RADICAVA Mean Change vs. Placebo on ALSFRS-R at Week 24



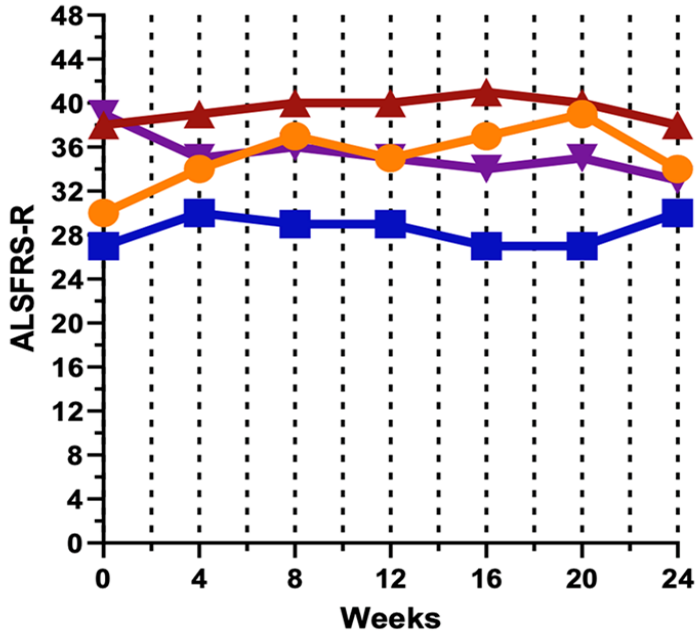
Both approved therapies slowed, but did not stop, disease progression and functional decline. *RELYVRIO withdrawn from US market in April 2024 following failure of the Phase 3 PHOENIX trial*

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)

COYA-302 has not been evaluated head–to-head against either molecule

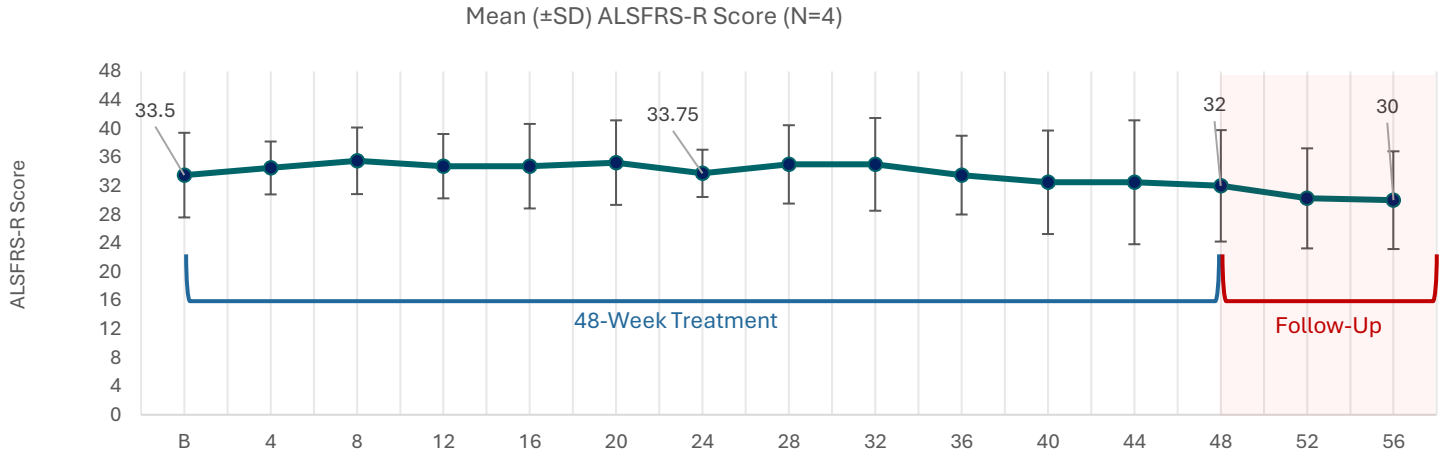
LD IL-2 + CTLA4-Ig IIT: Compelling Signal in Mild-to-Moderate ALS[1]

ALSFRS-R improved or stabilized over 6 months



- Participant 1: +4 points
- Participant 2: +3 points
- Participant 3: Stable
- Participant 4: -6 points (declined at month 1 and remained stable thereafter)

LD IL-2 + CTLA-4 Ig ameliorates progression in a 48-week IST in ALS Patients



- 4 mild-to-moderate ALS patients
- Low-dose IL-2 + CTLA4 Ig led to stable or improved ALSFRS-R Scores
- Well tolerated over 48 weeks (the most common AE mild was ISR)
- All patients completed the study
- No deaths or serious AEs (SAEs)

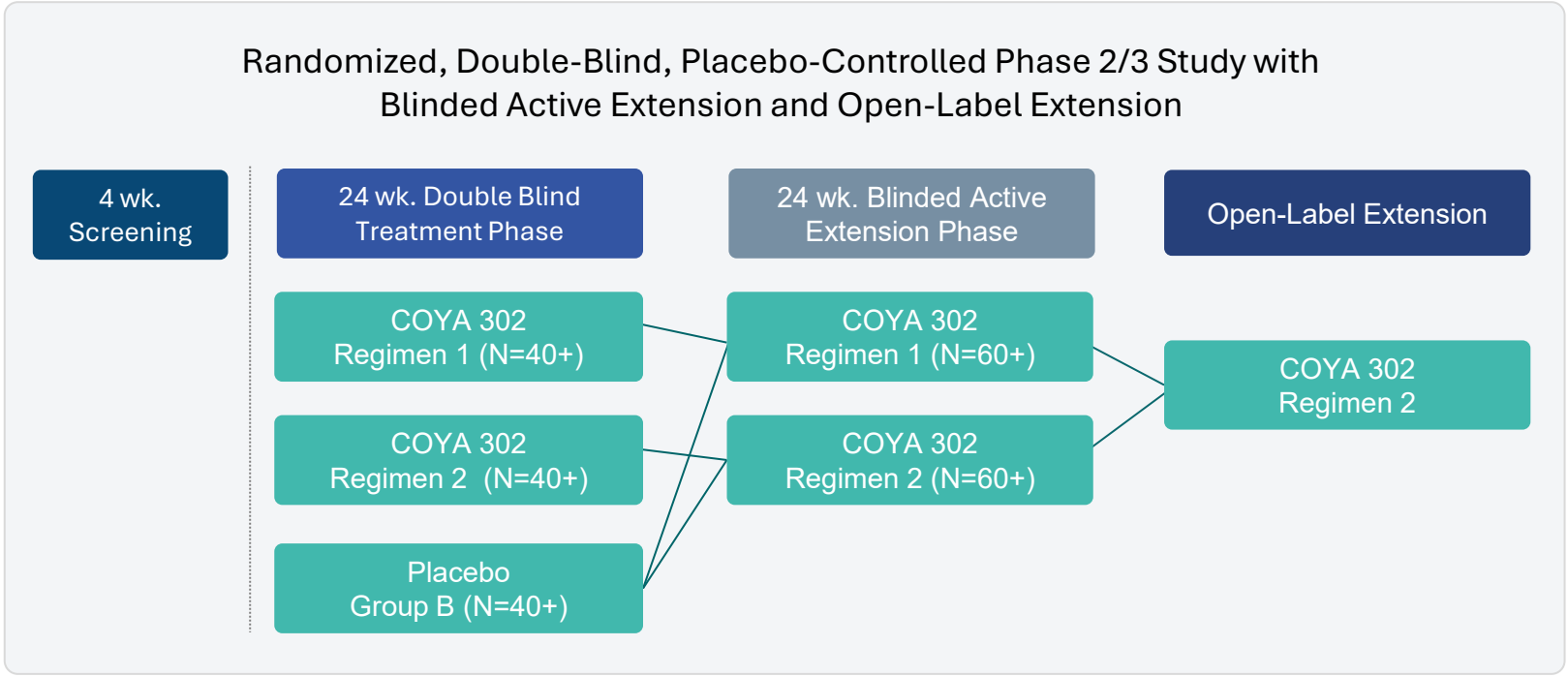
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[1] Thonhoff et al. A Phase 1 Proof-of-Concept Study Evaluating Safety, Tolerability, and Biological Marker Responses with combination therapy of CTLA4-Ig and Interleukin-2 in Amyotrophic Lateral Sclerosis. Frontiers in Neurology, 2024. In press.

COYA 302: ALSTARS Phase 2/3 Study in ALS Ongoing

Key Inclusion Criteria

- Diagnosis of sporadic or familial ALS
- ALSFRS-R score ≥ 35 at Screening
- Slow vital capacity (SVC) ≥ 60% of predicted capacity for age, height, and gender at Screening
- Documented disease progression between -0.5 and -1.5 points per month on ALSFRS-R total score



Trial fully enrolled (N=137) | Topline data expected early Q2 2027 | Fast Track Designation granted to COYA 302 in ALS

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

Note: **Regimen 1** = low dose; **Regimen 2** = high dose

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ALS Summary

The Disease & Unmet Need

Rapidly fatal — ~1-point/month ALSFRS-R decline; 20–30% die within two years of diagnosis

~90% of cases are sporadic, with no family history

Approved therapies only modestly effective; RELYVRIO withdrawn in 2024

Early Clinical Signal

48-week investigator-initiated study in mild-to-moderate ALS

3 of 4 patients stable or improved ALSFRS-R (+4, +3, stable)

Well tolerated — no deaths or SAEs

COYA 302: Coya's Approach

Low-dose IL-2 + CTLA4-Ig combination therapy

Restores and maintains regulatory T-cell (Treg) function to reduce neuroinflammation

Built for the complex, multifactorial biology of ALS

Market Opportunity

Orphan indications with high unmet need

Short life expectancy and major loss of quality of life

Combination therapy seen as a viable path forward

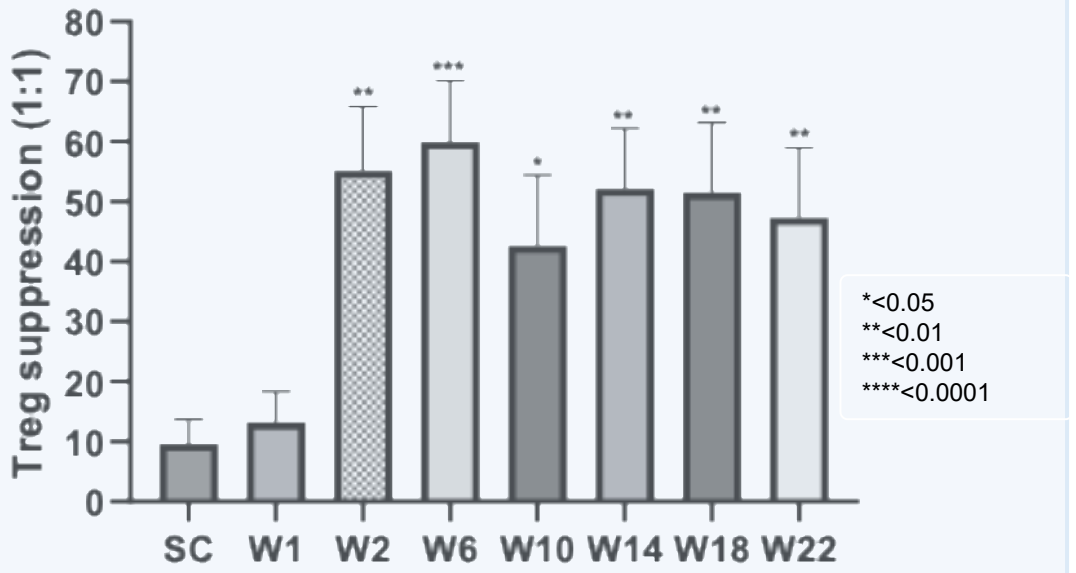
COYA 302 in FTD

LD IL-2 & CTLA-4 Ig: Signal in Mild-to-Moderate FTD

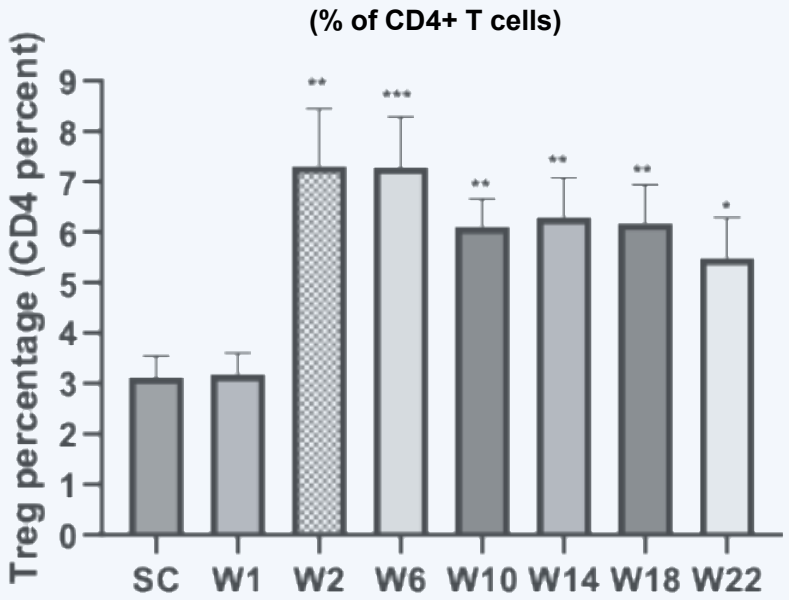
Encouraging clinical data in FTD in Investigator Sponsored Study (n=9)

Treg suppressive function and percentage: (1) Increased after first treatment (2) Remained elevated across 22-week treatment period

Increased Treg Suppressive Function



Expansion of Treg Percentage



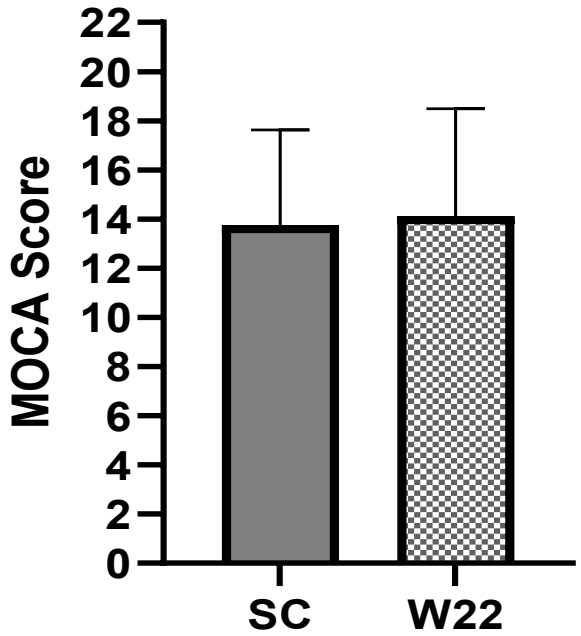
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LD IL-2 & CTLA-4 Ig: Signal in FTD – cont.

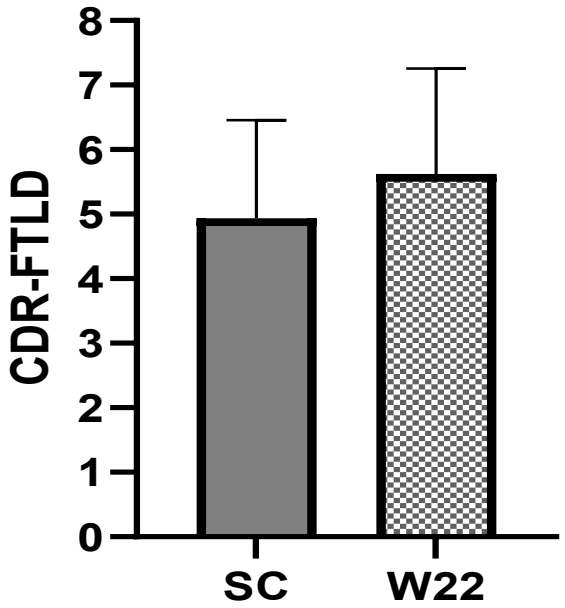
Cognitive Stability in 22-Week Investigator Sponsored Study

CDR-FTLD, MoCA, PASS cognitive tests exhibited no cognitive decline at end of study compared with baseline

Moca Scores Remain Unchanged Compared to Baseline



CDR-FTLD Scores Remains Unchanged Compared to Baseline

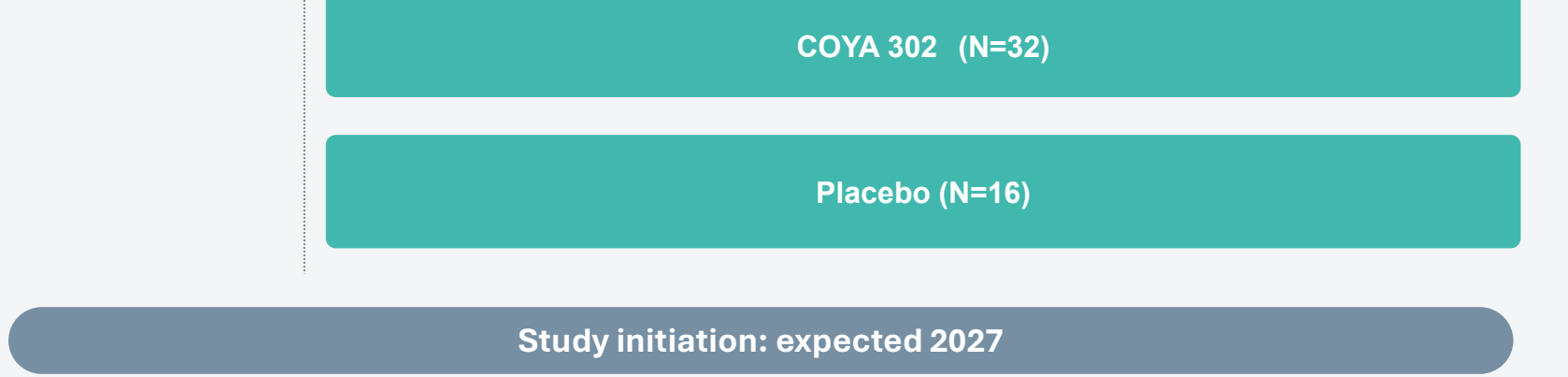


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COYA-302 in FTD: Phase 2 Expected 2027 Start

Key Inclusion Criteria

- Non-Fluent Primary Progressive Aphasia Subtype
- Global Clinical Dementia Rating - Frontotemporal Lobar Degeneration (CDR® plus NACC FTLD) score of 0.5, 1 or 2



Primary Endpoint

Safety and tolerability

Clinical Efficacy Endpoints

CDR+NACC-FTLD-SB*
Neuropsychological tests

Biomarkers

Plasma Glial Fibrillary Acidic Protein (GFAP)
Inflammatory cytokines NfL

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

FTD Summary

The Disease & Unmet Need

Frontotemporal dementia (FTD) — progressive neurodegeneration affecting behavior, language and cognition

A leading cause of dementia in people under 60

No approved disease-modifying therapies — high unmet need

Early Clinical Signal

22-week investigator-sponsored study in mild-to-moderate FTD (n=9)

Treg suppressive function rose after first dose and stayed elevated

No cognitive decline vs baseline on CDR-FTLD, MoCA and PASS

COYA 302: Coya's Approach

Low-dose IL-2 + CTLA4-Ig combination therapy

Restores and maintains regulatory T-cell (Treg) function to reduce neuroinflammation

Same immune-modulating approach advancing in ALS

Market Opportunity

Orphan indication with high unmet need

Short life expectancy and major loss of quality of life

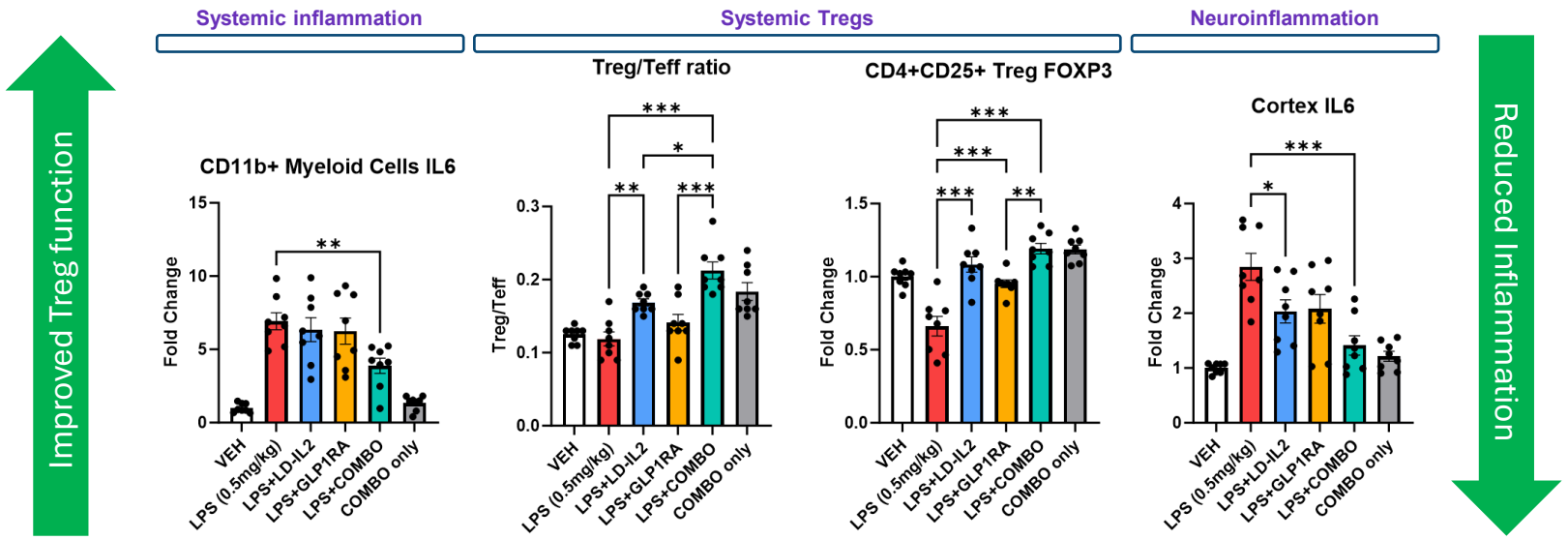
Combination therapy seen as a viable path forward

COYA 303 in AD

COYA 303 in Alzheimer's: Compelling Preclinical Signal

Encouraging in-vitro data and in LPS mouse model

- Lipopolysaccharide (LPS) mouse model is an established murine model of systemic and neuro-inflammation
- COYA 303 decreased both peripheral inflammation and in neuro-inflammation
- Mechanistic synergy: complementary effects on Treg enhancement and myeloid-driven inflammation



Clinical development of COYA 303 contingent upon securing non-dilutive funding or a partnership

COYA-303:

Reduced peripheral pro-inflammatory cell expansion

Enhanced Treg function

Attenuated CNS inflammation

Shifted macrophages to an anti-inflammatory phenotype

Decreased neuroinflammation

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Financial Summary and Dr. Reddy's Collaboration

Financial Summary
As of September 30, 2026

Cash: $35.8 million

Greenlight Capital: 9.9% ownership*

Dr. Reddy's: 9.7% ownership*

No debt

Cash runway:

Into H2 2027, past the ALSTARS Phase 2/3 ALS data readout (anticipated in early Q2 2027)

Shares outstanding:

~23.5 million shares of common stock outstanding as of 11/10/2026

*Based on publicly available ownership schedules filed with the Securities and Exchange Commission

Dr. Reddy's Collaboration
Announced December 2023

Dr. Reddy's gets:

An exclusive license to commercialize COYA 302 for ALS in the US, Canada, the EU and UK, if approved

COYA gets:

$7.5m upfront (received December 2023);

$4.2m upon IND acceptance (received August 2025);

$4.2m upon dosing of the first patient in the Phase 2/3 study (received December 2025)

Up to $40m in potential development and regulatory milestones;

Approximately $677m in potential sales-based milestones, and;

Low-to-mid teens royalties on net sales

COYA responsible for ongoing development of 302 and for obtaining US regulatory approval

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Multi-Layered IP Protection

Formulation

Granted patent on low-dose IL-2 aqueous formulation — enables at-home administration via prefilled syringe (vs. Proleukin, which is lyophilized and IV-only)

Combination

Patent on combinations of any IL-2 (including long-acting) with any CTLA-4 Ig

Biomarker

Biomarker IP for 302 in ALS provides an additional layer of protection

Regulatory

ALS and FTD are orphan diseases — potential 7 years of market exclusivity if 302 receives Orphan Drug Designation from the FDA

Earliest patent expiry: 2042

Key Catalysts and Milestones

COYA 302 in ALS: ALSTARS (Phase 2/3)	Full enrollment: completed October 2026 (N=137); OLE initiation expected November 2026 Topline data: expected early Q2 2027
COYA 302 in FTD:	IND accepted in 1Q 2026 Phase 2 study initiation: expected 2027 Announce biomarker data from the completed investigator-initiated FTD study (Q1 2027)
Translational Datasets	Report additional mechanistic data and single-cell proteomics data from the completed ALS and AD investigator-initiated trials, subject to publication (Q4 2026/Q1 2027) Further elucidate role of Tregs in neuroinflammation and neurodegeneration Provide additional insight into target engagement, pathway modulation, and patient-level biology:
COYA 303 in Alzheimer's	Continue to evaluate path forward Clinical advancement contingent upon securing non-dilutive funding or a partnership New published preclinical data[1] show complementary immunomodulatory effects of COYA 303 (a combination of proprietary LD-IL2 and GLP-1RA) compared to either LD-IL2 or GLP-1RA as monotherapy

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[1] https://www.mdpi.com/1422-0067/27/17/7855

Management and Board



Arun Swaminathan, Ph.D.

Chief Executive Officer

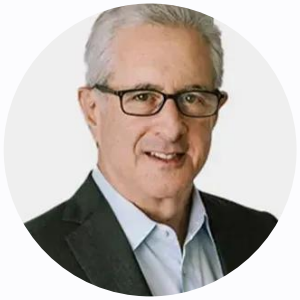
Bristol Myers Squibb

Actinium

Covance

Alteogen

Lynkogen



Fred Grossman, D.O., FAPA

President & Chief Medical Officer

Johnson-Johnson

Sunovion

Bristol Myers Squibb

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DisperSol

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Cellular Dynamics

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Dieter Weinand
Former Chairman and CEO
Bayer Pharma, AG

Scientific Advisory Board



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

Member of the National Academy of Sciences

Nobel Prize for discovery & role of Treg Biology

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



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

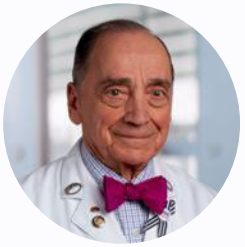


Malcolm Brenner, M.D., Ph.D.

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

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Thank You

INVESTOR OVERVIEW I November 2026