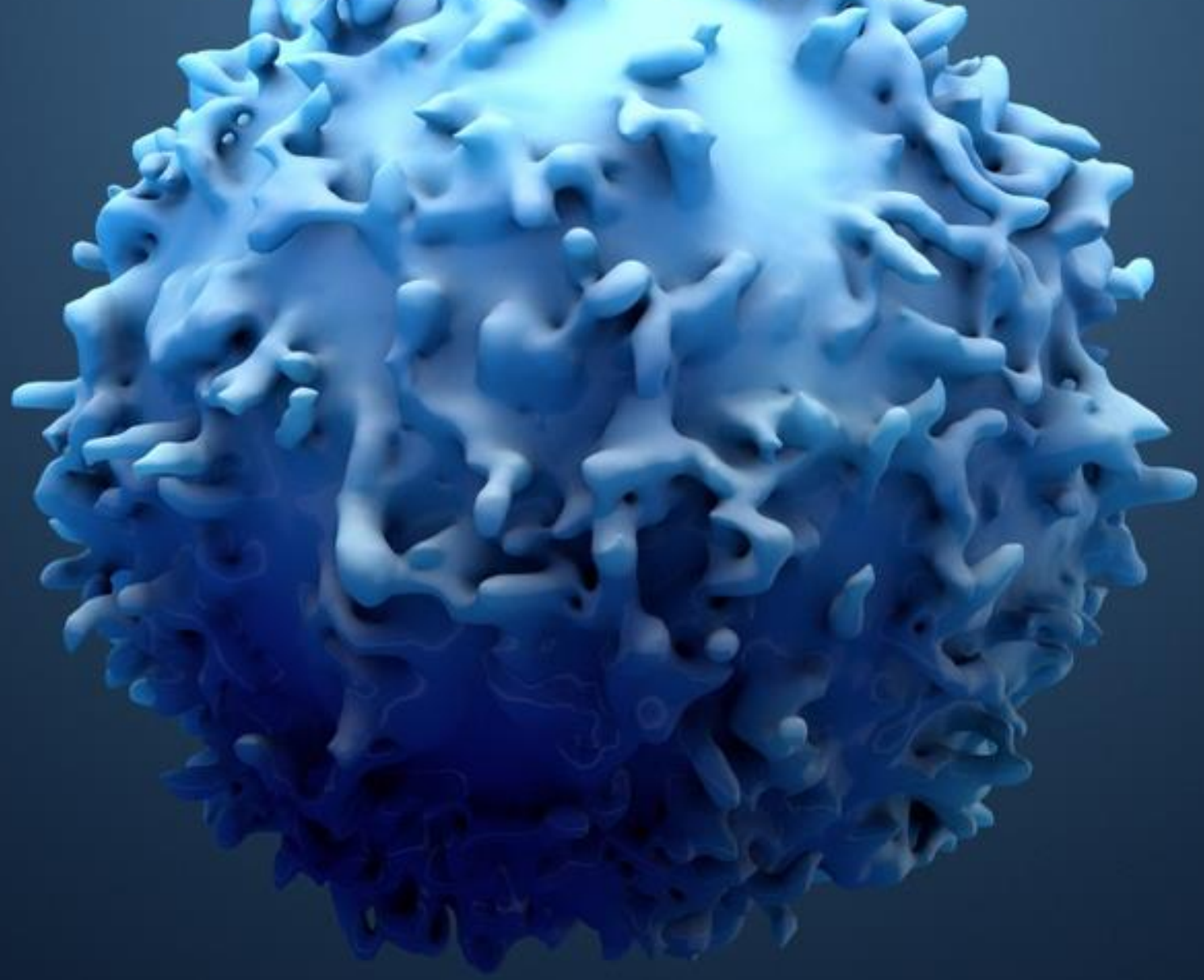
COYA THERAPEUTICS

Unleashing the Power of Regulatory T cells (Tregs)



Investor Presentation

October 29, 2024

NASDAQ: COYA

www.coyatherapeutics.com

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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 Diseases (**AD**)
Recent Business Developments	➢ Executed a potential **$700M+** transaction for COYA 302 in ALS with **Dr. Reddy's Laboratories** ➢ Completed **$5M** strategic investment by **Alzheimer's Drug Discovery Foundation (ADDF)** ➢ 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)

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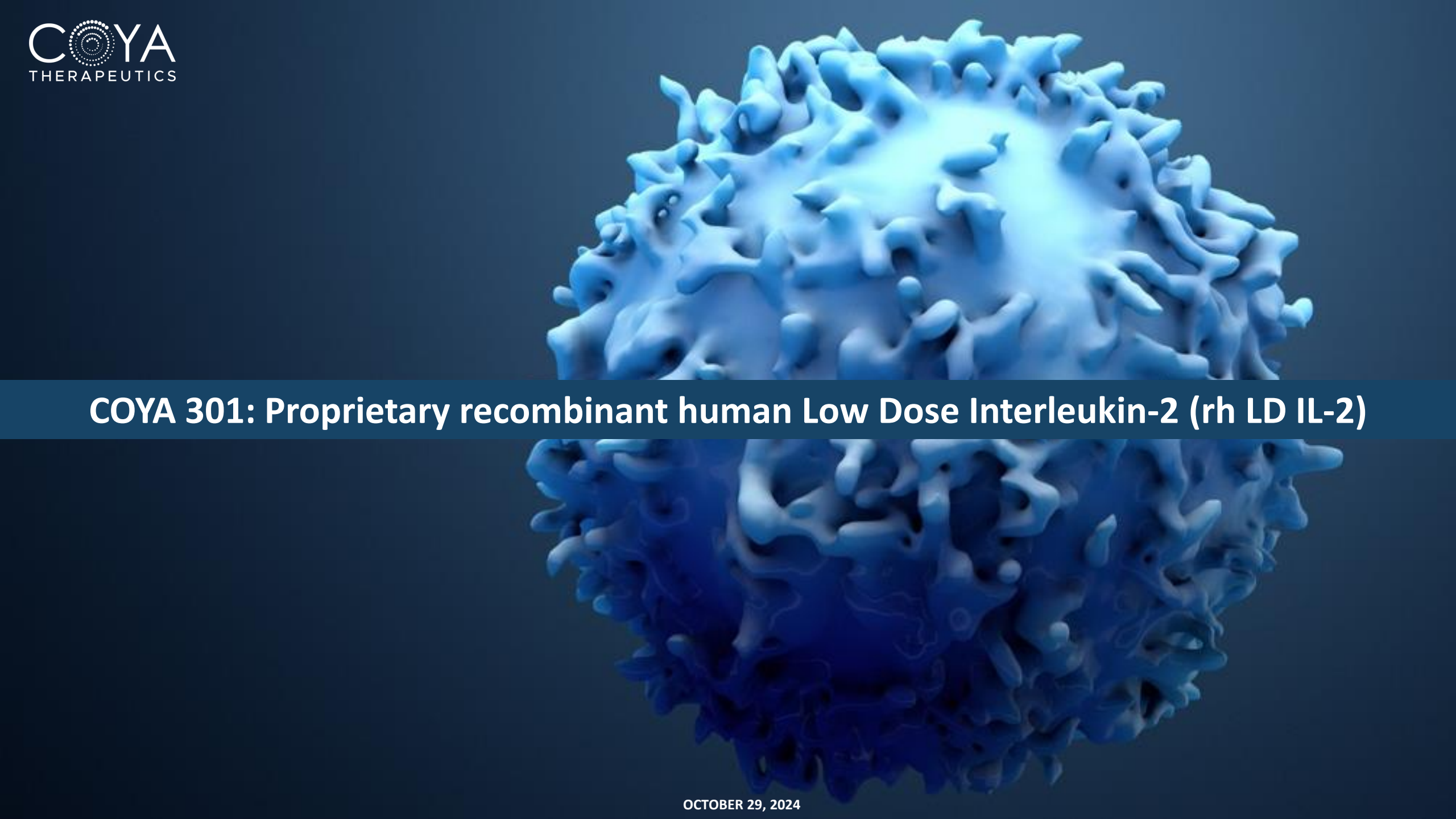

COYA 301: Proprietary recombinant human Low Dose Interleukin-2 (rh LD IL-2)

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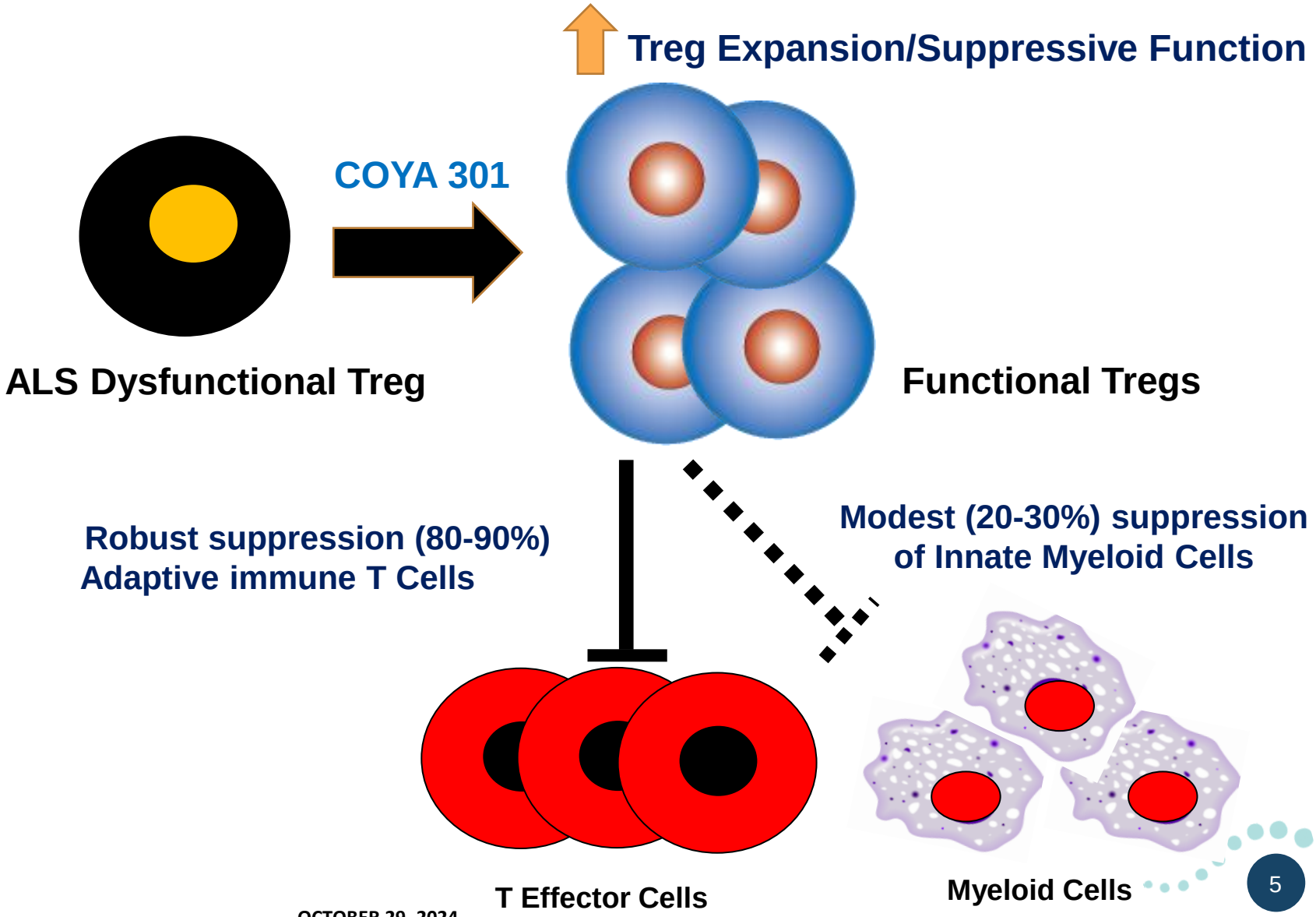
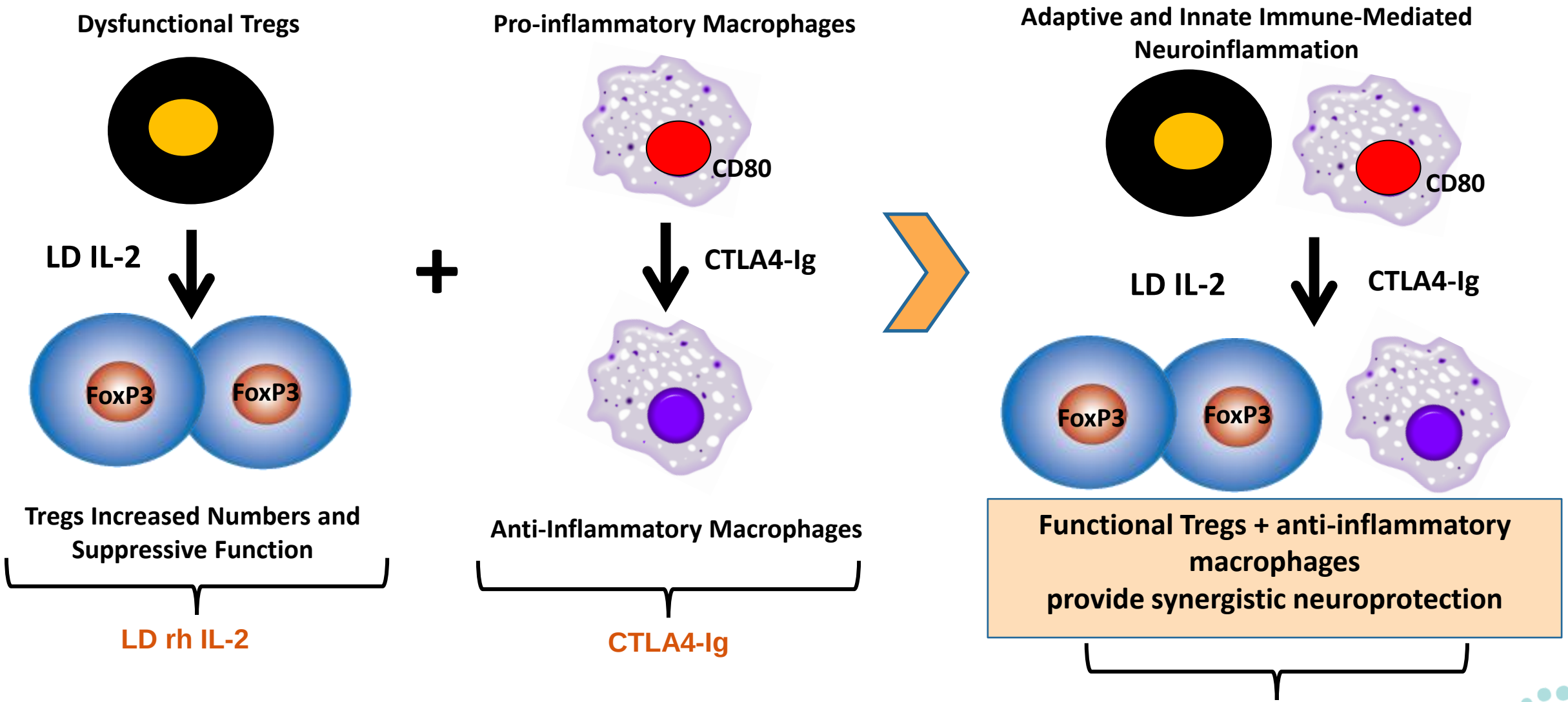
COYA 301: Proprietary Treg Enhancing Low-Dose IL-2 Suppresses Adaptive Immunity Robustly and Innate Immunity Modestly

Dysfunctional Tregs are associated with neuroinflammation promoted by activated T effector cells and activated innate myeloid cells



LD IL-2 Trial Tested Single Component of COYA 302 (proprietary LD rh IL-2 + CTLA4-Ig)



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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

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

✓ Cognition Stabilization (Exploratory Endpoint) Throughout 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 central nervous system (CNS) Amyloid Beta Pathology 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

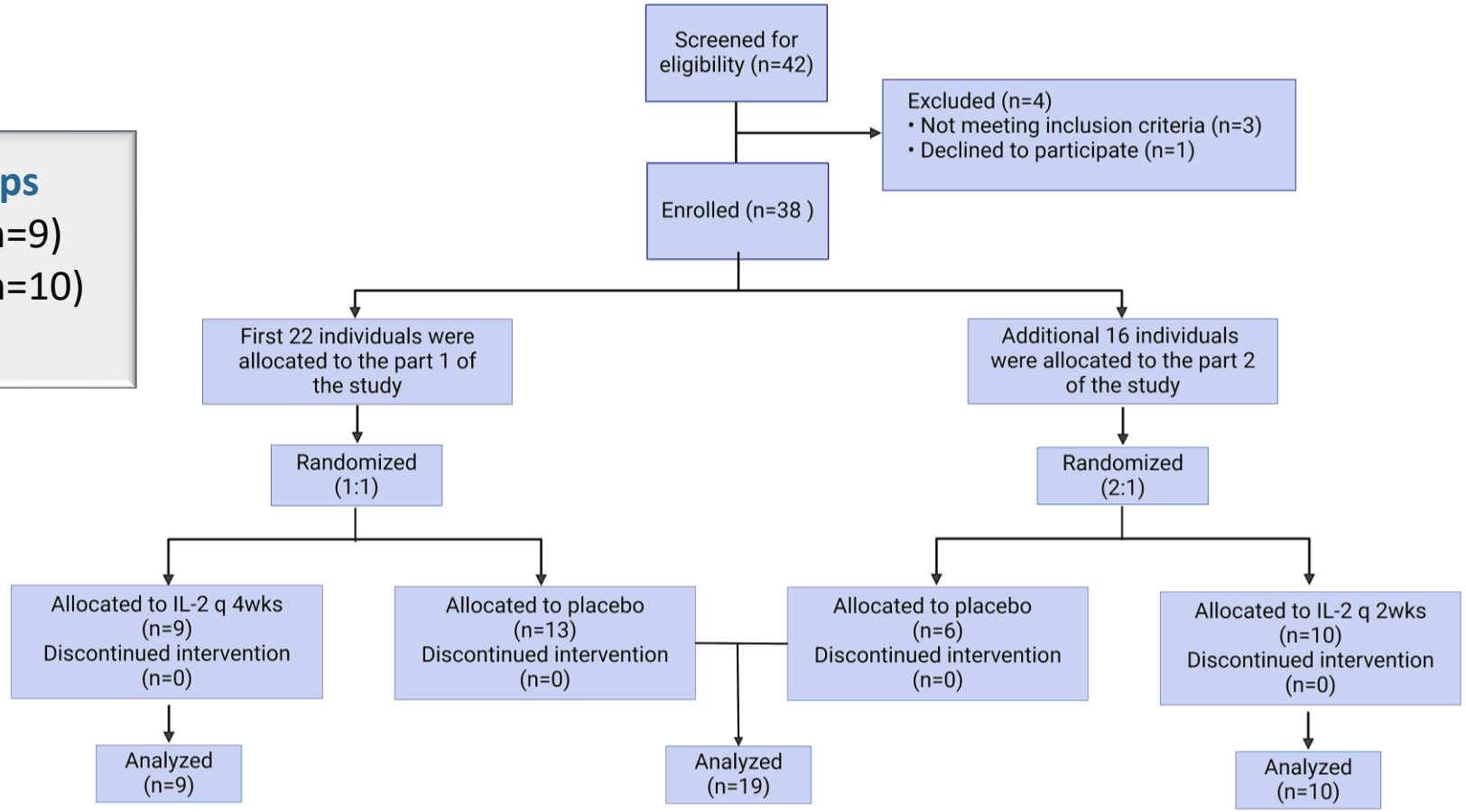
*The Alzheimer's Disease Assessment Scale–Cognitive Subscale (ADAS-Cog) was developed in the 1980s to assess the level of cognitive dysfunction in Alzheimer's disease. Clinical Global Impression of Change (CGIC) scales have been used as primary outcomes in phase 2 and 3 clinical trials for Alzheimer 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.

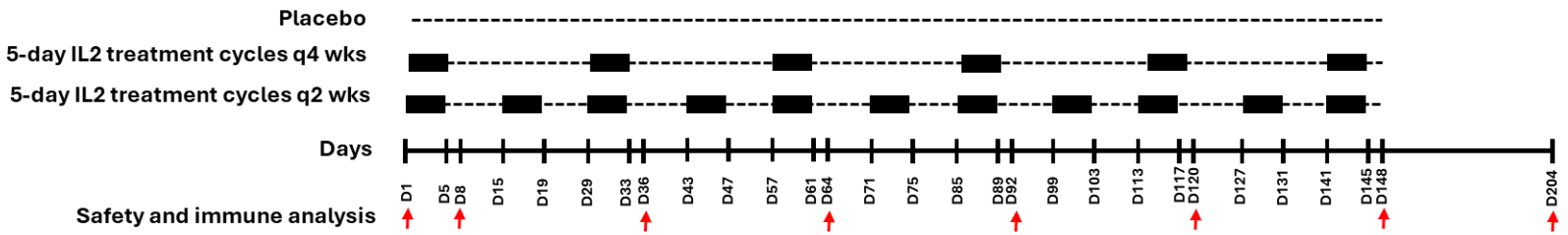
LD IL-2 Overview of Study Design

Randomized, Double-Blind, Placebo-Controlled Trial (N=38)

Treatment Groups

- LD IL-2 Q4wk: (n=9)
- LD IL-2 Q2wk: (n=10)
- Placebo: (n=19)





LD IL-2 Baseline Characteristics

Consistent Across Groups

Characteristics of the participants at baseline			
Characteristics	**IL-2 q 4 wks (n=9)**	**IL-2 q 2 wks (n=10)**	**Placebo (n=19)**
Age-yr (MEAN±SD)	68.67±6.96	75.9±6.54	68.68±7.63
Sex- no. (%)			
Female	6 (66%)	6 (60%)	11 (58%)
Male	3 (33%)	4 (40%)	8 (42%)
Race/ Ethnicity			
White/non-Hispanic	9 (100%)	9 (90%)	16 (85%)
White/ Hispanic	0 (0%)	1 (10%)	2 (10%)
Black	0 (0%)	0 (0%)	1 (5%)
Education			
High school	0 (0%)	2 (20%)	2 (11%)
College	6 (66%)	6 (60%)	12 (63%)
Post-grad	3 (33%)	2 (20%)	5 (26%)
APOE ε4 status — no. (%)			
Noncarrier	3 (33%)	2(22%)	6 (32%)
Carrier-Heterozygotes	4 (44%)	6 (66%)	5 (26%)
Carrier-Homozygotes	2 (22%)	2 (22%)	8 (42%)
Cognitive scales (MEAN±SD)			
MMSE score	18.78±2.95	18.50±2.84	17.37±3.47
CDR-SB score	4.72±3.19	4.30±2.35	4.84±3.04
ADAS-Cog score	31.24±12.84	30.75±8.54	36.04±15.39

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LD IL-2 Primary Endpoint

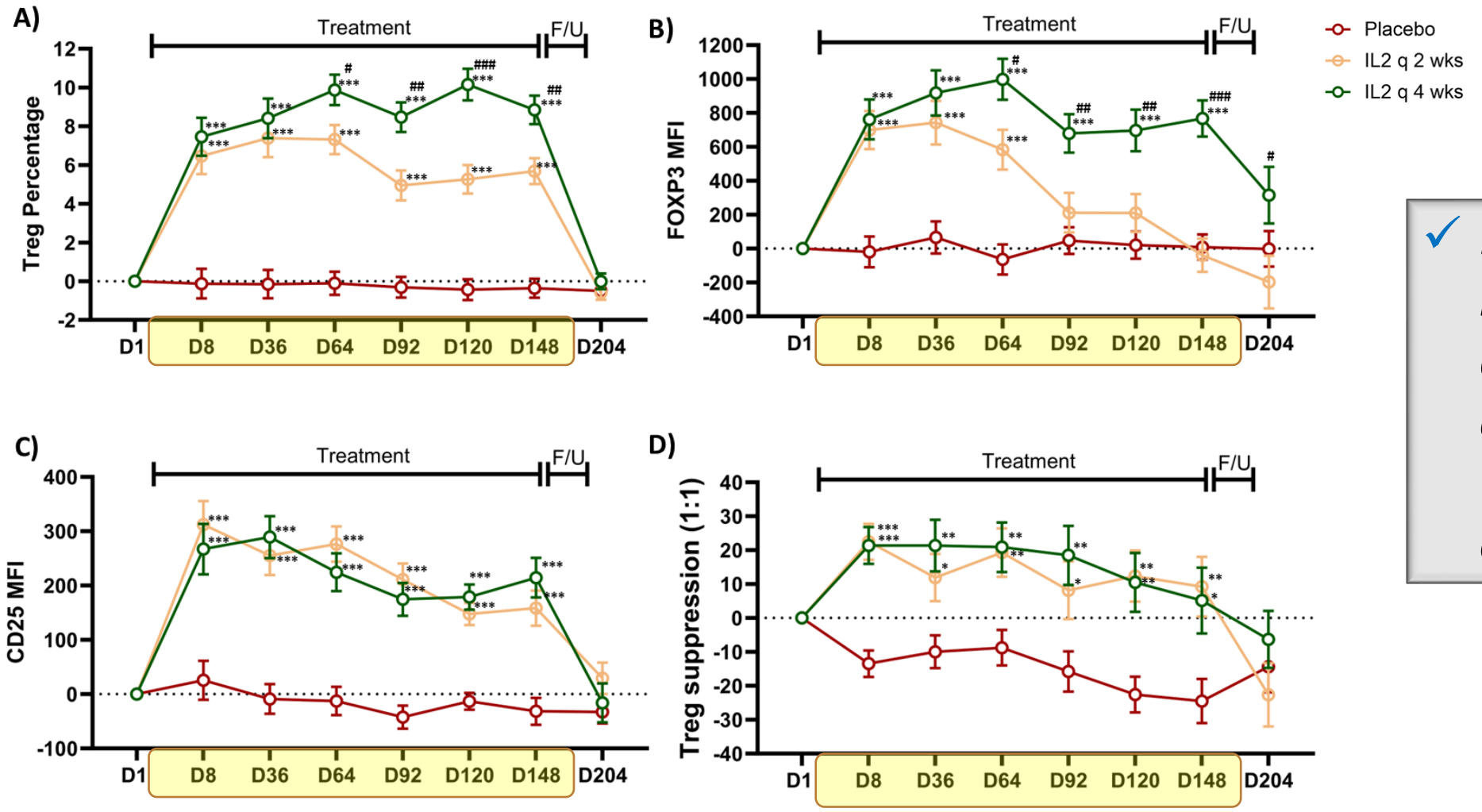
Safety & Tolerability

ADVERSE EVENTS SUMMARY				
Events	**IL-2 q 4 wks (n=9)**	**IL-2 q 2 wks (n=10)**	**Placebo (n=19)**	**Relation to intervention**
Subjects with at least one AE:	6(66%)	8(80%)	14(73%)	N/A
Subjects with related AEs:	3(33%)	5(50%)	3(15.7%)	N/A
Death	0	0	0	-
Serious AEs	0	0	0	-
Non-serious Moderate AEs				
Fall	1(11%)	-	1 (5%)	No
Gallstone	1(11%)	-	-	No
Covid Pneumonia	-	-	1 (5%)	No
Musculoskeletal pain	-	-	1 (5%)	No
Gout relapse	1(11%)	-	-	No
Presyncope	-	-	1 (5%)	No
Non-serious Mild AEs				
Increased eosinophil count	2(22%)	3 (30%)	-	Yes
Erythema at injection site	1(11%)	3 (30%)	-	Yes
Decreased hemoglobin level	1(11%)	1 (10%)	5 (26%)	No
Nausea/Vomiting/Abdominal pain	-	2 (20%)	3 (15.7%)	No
Leukopenia	-	1 (10%)	3 (15.7%)	Yes
Fall	1(22%)	-	1 (5%)	No
Covid pneumonia	-	-	2 (10.5%)	No
Hypokalemia	-	1 (10%)	1 (5%)	No
Presyncope	-	-	1 (5%)	No
Elevated tsh	-	-	1 (5%)	No
Shingles	1(11%)	-	-	No
Cough	-	-	1 (5%)	No
Leg sciatica	-	1	1 (5%)	No
Fatigue	-	-	1 (5%)	No
Chest pain	-	1 (10%)	-	No
Elevated troponin	-	1 (10%)	-	No

- ✓ *All Patients Completed the Trial and the Drug Was Well-Tolerated*
- ✓ *No Serious Adverse Events Were Reported*

Secondary Endpoint

Effective, Sustainable, and Significant Expansion of Treg Populations

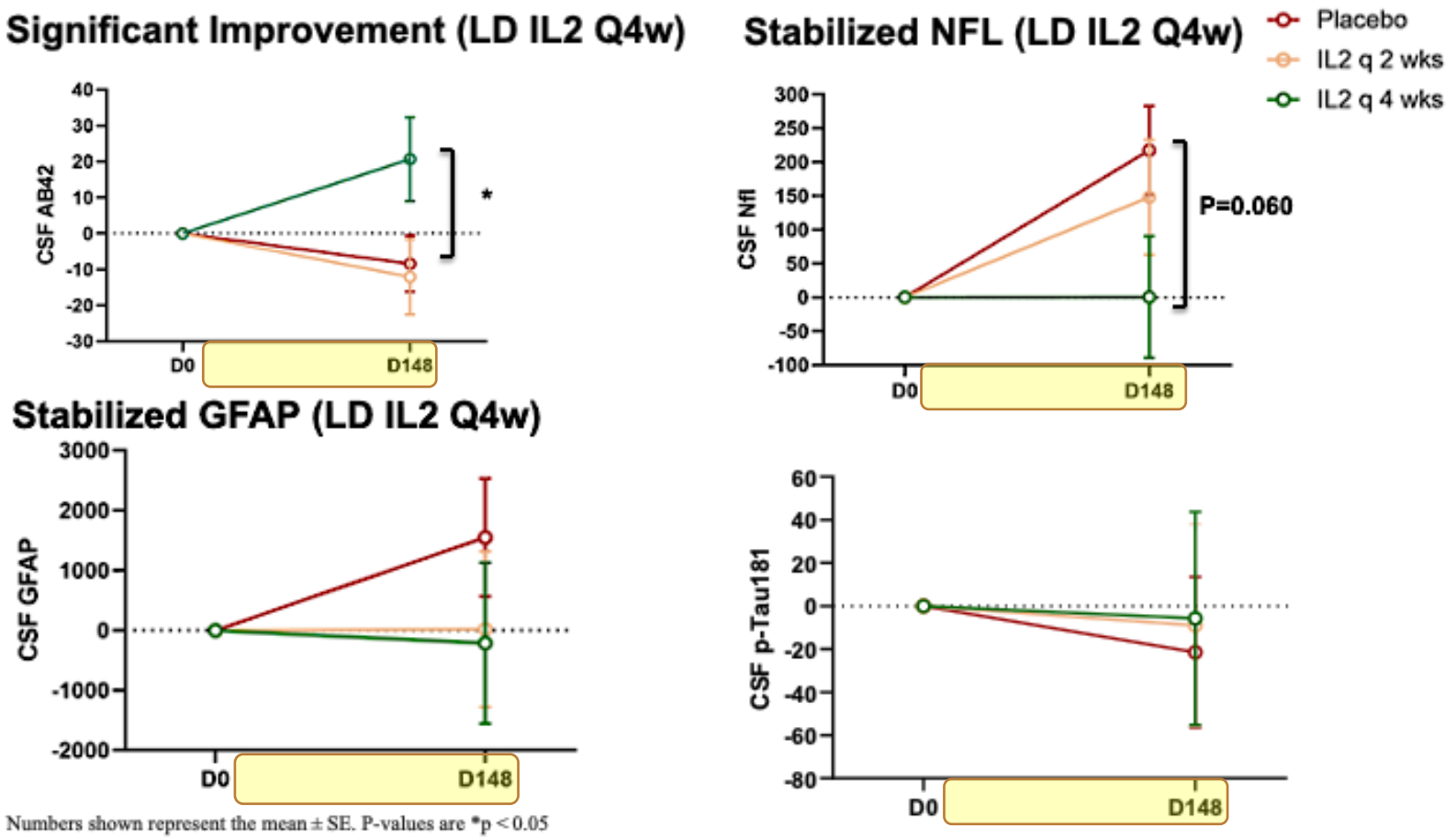


- ✓ *LD IL-2 q4wks resulted in effective and sustainable expansion of Tregs without off-target effects*

: 21 week or 148 day on-treatment period

Exploratory Endpoints

CSF Modifications of Disease Pathology



Numbers shown represent the mean ± SE. P-values are *p < 0.05

LD IL-2 q4wks administration improved CSF soluble Aβ42 levels (an index of amyloid pathology) and stabilized CSF NfL (an index of neuronal injury) and CSF GFAP (an index of neurodegeneration)

- ✓ *LD IL-2 q4wks resulted in stabilized or improved key CSF biomarkers related to AD*

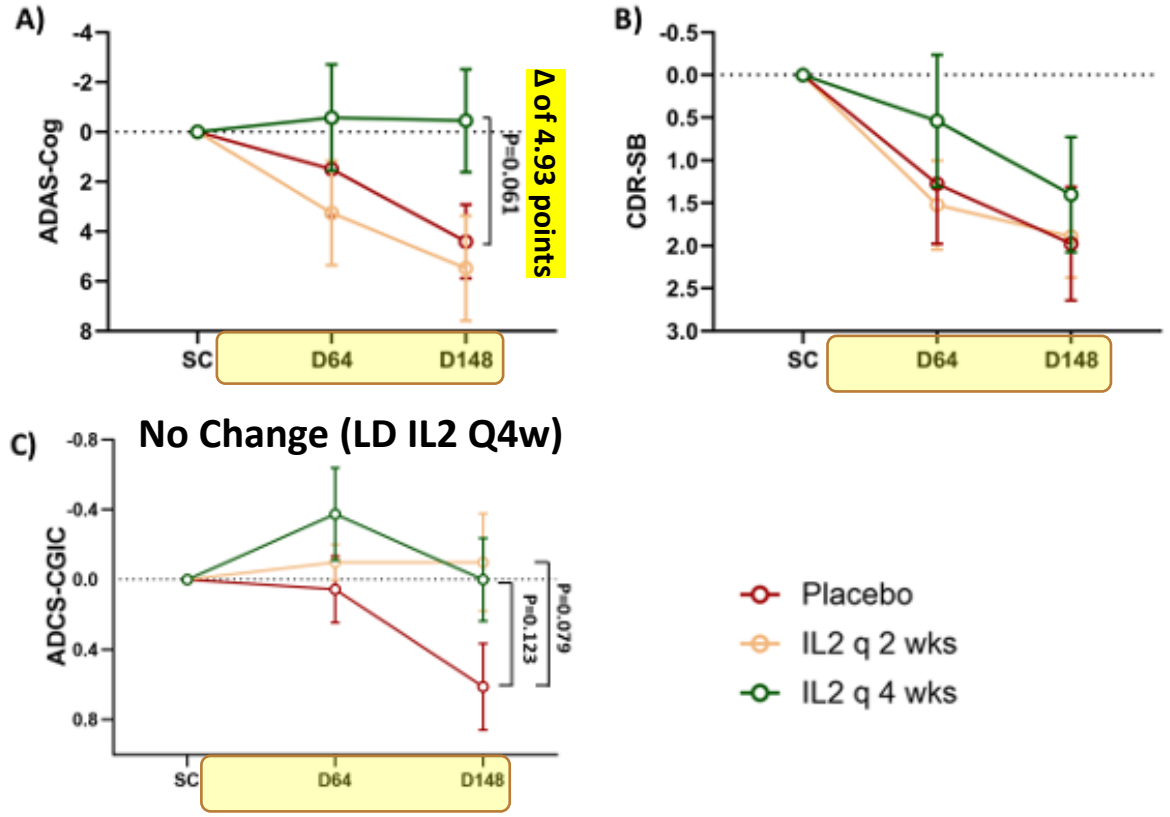
: 21 week or 148 day on-treatment period

Exploratory Endpoint

Clinical Scales Demonstrating Cognitive Stabilization

0.45 Point Improvement (LD IL2 Q4w)

27% Slower Decline (LD IL2 Q4w)



Promising trend in stabilizing cognitive function as monitored by the ADAS Cog and ADCS-CGIC

- ✓ *LD IL-2 q4wks resulted in slight improvement of ADAS-Cog Score, with stabilization or decreased rate of decline in key clinical scales for AD*

: 21 week or 148 day on-treatment period

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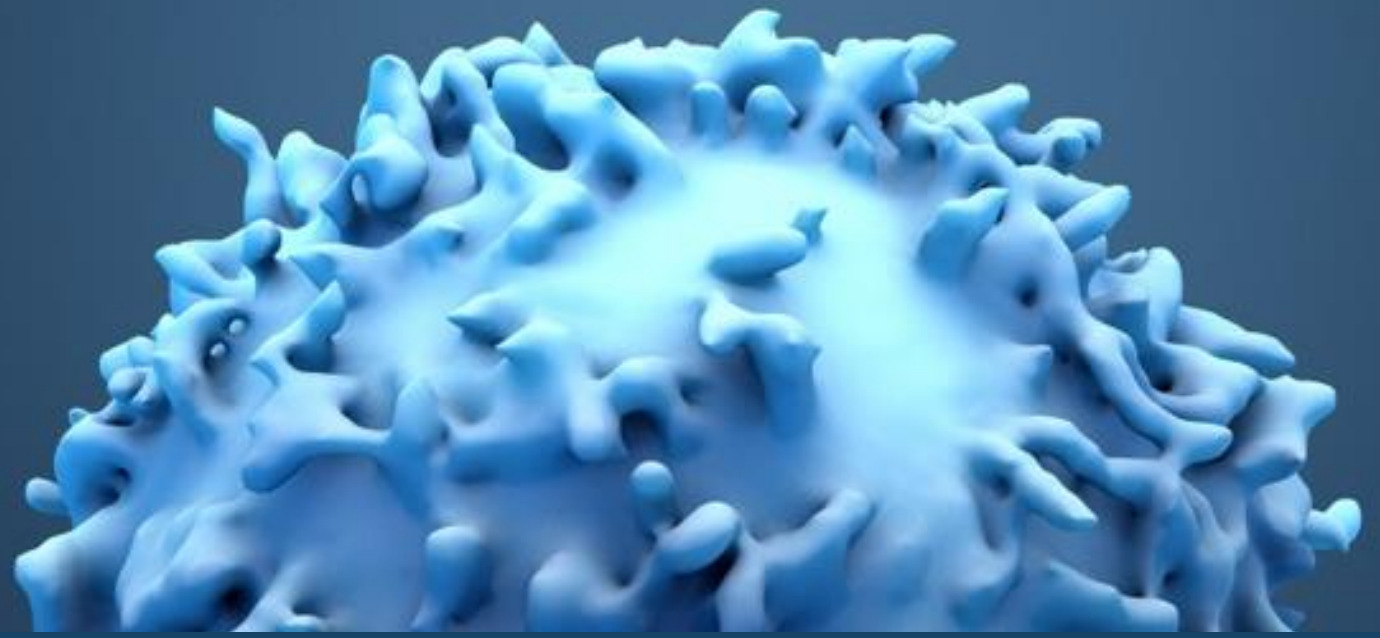

LD IL-2 Study in Support of COYA 301 & 302*

*COYA 301 and 302 are investigational products, not yet approved by the US Food and Drug Administration

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What's Next?

- Reporting of additional data from the LD IL-2 study documenting the effect of LD IL-2 treatment vs. placebo on systemic and CNS inflammatory markers (including peripheral blood cytokines and chemokines, and PET brain imaging)
- Discussions ongoing to advance other combination strategies with COYA 301 in larger cohorts of Alzheimer's Disease patients, as well as for COYA 302
- Planned clinical trials for COYA 302 in ALS and FTD

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

Questions & Answers

Thank you!