NEWS RELEASE

Coya Therapeutics Issues Letter to Stockholders Highlighting Expansion of COYA 302 into Alzheimer's Disease and Coya's Pathway to a "Pipeline in a Product"

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- Combination biologics with LD IL-2 as a backbone may represent next-generation approaches that target complex immune pathways in neurodegenerative diseases
- Expansion of COYA 302 into Alzheimer's Disease leverages synergistic mode of action and intellectual property position of the biologic combination of the Company's proprietary low-dose interleukin-2 formulation (LD IL-2) with its proprietary CTLA4-Ig that is intended to enhance the anti-inflammatory function of regulatory T cells (Tregs), suppress pro-inflammatory cells, and reduce oxidative stress
- Updated pipeline may be viewed here

HOUSTON--(BUSINESS WIRE)-- **Coya Therapeutics, Inc.** (NASDAQ: COYA) ("Coya" or the "Company"), a clinical-stage biotechnology company developing multiple therapeutic programs intended to enhance regulatory T cell (Treg) function, releases the following letter to stockholders from its Chief Executive Officer and Chairman, Dr. Howard Berman.

Dear Fellow Stockholders,

Just over two months ago we announced our partnership with Dr. Reddy's Laboratories for COYA 302 in Amyotrophic Lateral Sclerosis (ALS), our lead product candidate in our lead indication. This pivotal collaboration is worth up to $733 million ($677.25 million in non-dilutive sales and milestone payments, $40 million in development and regulatory milestones, and two tranche payments) plus low-to-mid teens product royalties to

us. We have already received $7.5 million as an upfront payment tranche from that transaction and expect to receive an additional $8.4 million tranche in 2024 following the IND filing and first patient dosed in the Ph. 2 ALS trial.

Our team continues to see strong value in COYA 302, as exhibited by our announcement in January 2024 that we were expanding the potential indications for that product candidate to include Frontotemporal Dementia (FTD) and Parkinson's disease (PD) as additional indications. FTD and PD are two neurodegenerative diseases that share common inflammatory disease pathways with ALS, and we believe COYA 302 can target these multiple pathways by restoring dysfunctional regulatory T cells (Tregs), reducing oxidative stress, and inhibiting toxic neuroinflammation.

COYA 302 – "Pipeline in a Product" with Expansion into Alzheimer's Disease

Today, we announce that we are further expanding the **pipeline** for COYA 302, adding Alzheimer's disease (AD) to its growing list of indications expected to be validated in the clinic. We recognize the historical challenges faced by pharma companies looking to address the massive problem that AD is posing to patients, families, and the healthcare system. Yet, researchers have shown that ALS, FTD, PD, and AD share common features, including neuroinflammation and catastrophic neuronal loss that leads to cognitive or motor dysfunction through the complex interplay of the body's immune system and dysfunctional anti-inflammatory Tregs. We believe the traditional "one disease – one target - one drug" approach might not be the most appropriate framework for neurodegenerative disorders, and may be partly responsible for the lack of available and truly effective treatments

Based on the results to date from our studies of COYA 302, which is the combination of our proprietary low dose interleukin-2 (COYA 301, or LD IL-2) and the immunomodulatory drug CTLA4-Ig, we believe this combination has the potential to provide a sustained and durable effect on these devastating neurodegenerative disorders through the targeting of multiple pathways. Our research and clinical efforts lead us to believe that combination biologics using our LD IL-2 as a backbone modality could be the best way to treat neurodegenerative conditions that are inherently driven by a complexity of pathways. We also believe that our growing intellectual property portfolio across such combination therapies positions us well to navigate this evolving landscape, with COYA 302 representing the most clinically advanced of what we hope will be a family of combination therapies that all feature our proprietary LD IL-2.

Moreover, given its growing list of indications, we can now refer to COYA 302 as a "Pipeline in a Product." It is not just potentially viable in its lead indication of ALS. Rather, due to its multi-modal mechanism of action, we believe it may be efficacious across the spectrum of neurodegenerative diseases that share a similar mechanistic cascade underlying pathophysiology, such as FTD, PD, and AD, and therefore may have utility for more patients beyond the ~20,000 patients suffering from ALS in the U.S.

Ongoing Double-Blind Trial for LD IL-2 in AD: A Proof-of-Concept Study to Support COYA 302 in AD

A double-blind, placebo-controlled, proof-of-concept study with LD IL-2 in patients with AD is ongoing (and still blinded) at the Houston Methodist Hospital, with top-line results anticipated in mid-2024. While the primary objectives in this study are the customary safety endpoints, a secondary endpoint to be measured is the change in Treg percentage out of the total number of CD4 cells. This study is intended to serve as a proof-of-concept to support the development of COYA 302 as a treatment for AD, including a potential partner strategy.

Since COYA 302 targets both the adaptive and innate immune systems with the addition of CTLA4-Ig to our LD IL-2, we expect a more sustained and durable effect than from just LD IL-2 alone. This synergistic combination effect has, in fact, been clinically documented by sustaining Treg anti-inflammatory suppressive function and increasing Treg count over a 1-year period when given to ALS patients. Further, this combination enhanced suppression of macrophage mediated oxidative stress and proinflammatory cytokine biomarkers in this population, which is critical since inflammation and oxidative stress are hallmarks of neurodegenerative diseases. We believe that this synergistic mechanism leads to the re-establishment of immune balance and amelioration of inflammation in a sustained and durable manner that may not be achieved by either low-dose IL-2 or CTLA4-Ig alone. In AD, there is an additional complex interaction between inflammation and protein aggregation, including amyloid plaques and tau, and both may serve as triggers for the other. Therefore, focusing solely on protein aggregation as some therapies do without addressing inflammation may not be adequate. Our combination therapy from COYA 302 aims to address both pathways.

Additional Clinical Development Updates for COYA 302:

- ALS: Following the encouraging results of an open-label academic study in patients with ALS that was led by Dr. Appel and Dr. Thonhoff, we are planning a controlled, statistically-powered clinical study to demonstrate the safety and efficacy of COYA 302 for the treatment of ALS. The study is expected to commence upon the filing of the IND in the first half of 2024. This study represents our lead indication.
- FTD: We intend to file an IND for the treatment of FTD by Q4 2024.
- PD: We are performing ongoing mechanistic validation studies in animals and intend to present data by Q3 2024.

Expanding Combination Therapies Beyond COYA 302

Beyond COYA 302, our therapeutic platform includes additional drug product combinations using COYA 301 (our proprietary LD IL-2) as their backbone. These combination candidates may have therapeutic effects in diseases driven by inflammation and immune system dysfunction and could enable potential strategic collaborations.

We recently announced a licensing agreement with University of Nebraska Medical Center covering multiple LD IL-2 combinations, including LD IL-2 + Granulocyte-Macrophage Colony Stimulating Factor (GM-CSF) in inflammatory disorders. This license expands our multi-pathway approach in identifying and combining COYA 301 with immunomodulatory drugs that are synergistic in simultaneously enhancing Treg function and lowering inflammation.

Conclusion

With our current cash balance at the end of 2023, along with the initial $7.5 million upfront payment received from Dr. Reddy's in early January 2024, we believe we have at least a two-year cash runway to support our operations. We anticipate milestones in 2024 that could deliver additional shareholder value, including, but are not limited to, 1) the publication of additional data from the COYA 302 investigator initiated trial in ALS patients and biomarker data that is positively correlated to survival and rate of decline in ALS patients, 2) the filing of INDs for COYA 302 in ALS and FTD, and 3) topline data from the Ph. 2 investigator-initiated trial with COYA 301 in AD that we expect will support the development of COYA 302 in that same indication.

We are committed to developing our "Pipeline in a Product" in COYA 302 with the ultimate goal of delivering safe and effective therapies for patients affected by devastating neurodegenerative diseases that collectively affect millions and millions of patients in the U.S. alone. Based on the unveiling of Tregs and the role of multiple pro-inflammatory mechanisms in the immune system by Dr. Sakaguchi and Dr. Appel, members of our scientific advisory board, we believe our desire to treat ALS, FTD, PD, and AD with COYA 302 combination therapy can offer meaningfully sustainable benefits to a large and growing patient base across numerous indications. The traditional "one disease – one target - one drug" approach might not be the most appropriate framework for neurodegenerative disorders and may be partly responsible for the lack of available and truly effective treatments in the indications we are pursuing. At Coya, we aim to bridge this gap in the neurodegenerative world, and given the success combination therapies are having in cancer, we see our approach as a natural scientific progression.

I look forward to providing investors with additional periodic updates on our research, clinical, corporate, and commercial progress.

Sincerely,

Howard Berman

Chairman and CEO

About COYA 302

COYA 302 is an investigational and proprietary biologic combination therapy with a dual immunomodulatory mechanism of action intended to enhance the anti-inflammatory function of regulatory T cells (Tregs) and suppress the inflammation produced by activated monocytes and macrophages. COYA 302 is comprised of proprietary low-dose interleukin-2 (LD IL-2) and CTLA4-Ig and is being developed for subcutaneous administration for the treatment of patients with ALS. These mechanisms may have additive or synergistic effects.

In February of 2023, Coya announced results from a proof-of-concept, open-label clinical study evaluating LD IL-2 and CTLA4-Ig in a small cohort of patients with ALS conducted at the Houston Methodist Research Institute (Houston, Texas) by Stanley Appel, M.D., Jason Thonhoff, M.D., Ph.D., and David Beers, Ph.D. This study was the first-of-its-kind evaluating this dual-mechanism immunotherapy for the treatment of ALS. Patients in the study received investigational treatment for 48 consecutive weeks and were evaluated for safety and tolerability, Treg function, serum biomarkers of oxidative stress and inflammation, and clinical functioning as measured by the ALSFRS-R scale.

During the 48-week treatment period, the therapy was well tolerated. The most common adverse event was mild injection-site reactions. No patient discontinued the study, and no deaths or other serious adverse events were reported.

Patients' disease progression was measured using the ALSFRS-R scale, a validated rating tool for monitoring the progression of disability in patients with ALS. The mean (±SD) ALSFRS-R scores at week 24 (33.75 ±3.3) and week 48 (32 ±7.8) after initiation of treatment were not statistically different compared to the ALSFRS-R score at baseline (33.5 ±5.9), suggesting significant amelioration in the progression of the disease over the 48-week treatment period.

Treg suppressive function, expressed as percentage of inhibition of proinflammatory T cell proliferation, showed a statistically significant increase over the course of the treatment period and was significantly reduced at the end of the 8-week washout post-treatment period. Treg suppressive function at 24 weeks (79.9 ±9.6) and 48 weeks (89.5 ±4.1) were significantly higher compared to baseline (62.1 ±8.1) ($p<0.01$), suggesting enhanced and durable Treg suppressive function over the course of treatment. In contrast, Treg suppressive function (mean ±SD) was significantly decreased at the end of the 8-week washout period compared to end-of-treatment at week 48 (70.3 ±8.1 vs. 89.5 ±4.1, $p <0.05$).

The study also evaluated serum biomarkers of inflammation, oxidative stress, and lipid peroxides. The available data up to 16 weeks after initiation of treatment suggest a decrease in these biomarker levels, which is consistent

with the observed enhancement of Treg function. The evaluation of the full biomarker data is ongoing.

COYA 302 is an investigational product not yet approved by the FDA or any other regulatory agency.

About Amyotrophic Lateral Sclerosis

Amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig's Disease, is a rare neurological disease that affects motor neurons, the nerve cells in the brain and spinal cord that control voluntary muscle movement. About 20,000 people live with ALS in the United States and approximately 5,000 new cases are diagnosed every year. The disease is progressive, meaning the symptoms get worse over time. The functional status of ALS patients declines about 1 point per month on average, as measured by the Revised ALS Function Rating Scale1, or ALSFRS-R, a validated tool to monitor the progression of the disease.

ALS has no cure, and the currently approved drug treatments provide limited benefit to patients. ALS is a type of motor neuron disease. As motor neurons degenerate and die, they stop sending messages to the muscles, which causes the muscles to weaken, start to twitch (fasciculations), and waste away (atrophy). Eventually, the brain loses its ability to initiate and control voluntary movements. Most people with ALS die from respiratory failure, usually within three to five years from when the symptoms first appear.2

About Frontotemporal Dementia

Frontotemporal dementia (FTD) is the result of damage to neurons in the frontal and temporal lobes of the brain. Many possible symptoms can result, including unusual behaviors, emotional problems, trouble communicating, difficulty with work, or difficulty with walking. FTD is rare and tends to occur at a younger age than other forms of dementia. About 60% of people with FTD are 45 to 64 years old. FTD is progressive, meaning symptoms get worse over time. In the early stages, people may have just one symptom. As the disease progresses, other symptoms appear as more parts of the brain are affected. It is difficult to predict how long someone with FTD will live. Some people live more than 10 years after diagnosis, while others live less than two years after they are diagnosed. There is no cure for FTD, and no treatments slow or stop the progression of the disease.3

About Parkinson's Disease

Parkinson's disease (PD) is a progressive brain disorder that causes unintended or uncontrollable movements, such as shaking, stiffness, and difficulty with balance and coordination. The most prominent manifestations of PD occur when nerve cells in the basal ganglia, an area of the brain that controls movement, become impaired or die. As the disease progresses, people may have difficulty walking and talking. They may also have mental and behavioral changes, sleep problems, depression, memory difficulties, and fatigue. Most people with PD first develop the

disease after age 60, but about 10% experience onset before the age of 50. There is no cure for PD and currently available treatments are intended to relieve some symptoms.4

About Alzheimer's Disease

Alzheimer's disease is the most common cause of dementia, a general term for memory loss and other cognitive abilities serious enough to interfere with daily life. Alzheimer's disease accounts for up to 80% of dementia cases, affecting an estimated 5.7 million Americans. In more than 90% of people with Alzheimer's, symptoms do not appear until after age 60. The incidence of the disease increases with age and doubles every 5 years beyond age 65. Alzheimer's is a progressive disease, where dementia symptoms gradually worsen over a number of years. In its early stages, memory loss is mild, but with late-stage Alzheimer's, individuals lose the ability to carry on a conversation and respond to their environment. It is the sixth leading cause of death among all adults and the fifth leading cause for those aged 65 or older. On average, a person with Alzheimer's lives 4 to 8 years after diagnosis but can live as long as 20 years, depending on other factors. 5, 6

References

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5. Alzheimer's Association (**www.alz.org**).
6. Centers for Disease Control and Prevention (**www.cdc.gov**).

About Coya Therapeutics, Inc.

Headquartered in Houston, TX, Coya Therapeutics, Inc. (Nasdaq: COYA) is a clinical-stage biotechnology company developing proprietary treatments focused on the biology and potential therapeutic advantages of regulatory T cells ("Tregs") to target systemic inflammation and neuroinflammation. Dysfunctional Tregs underlie numerous conditions, including neurodegenerative, metabolic, and autoimmune diseases, and this cellular dysfunction may lead to sustained inflammation and oxidative stress resulting in lack of homeostasis of the immune system.

Coya's investigational product candidate pipeline leverages multiple therapeutic modalities aimed at restoring the anti-inflammatory and immunomodulatory functions of Tregs. Coya's therapeutic platforms include Treg-enhancing biologics, Treg-derived exosomes, and autologous Treg cell therapy.

COYA 302 – the Company's lead investigational product – is a proprietary combination of COYA 301 (Coya's

proprietary LD IL-2) and CTLA4-Ig for subcutaneous administration with a unique dual mechanism of action that is now being developed for the treatment of ALS, FTD, PD, and AD. Its multi-targeted approach enhances the number and anti-inflammatory function of Tregs and simultaneously lowers the expression of activated microglia and the secretion of pro-inflammatory mediators. This synergistic mechanism may lead to the re-establishment of immune balance and amelioration of inflammation in a sustained and durable manner that may not be achieved by either low-dose IL-2 or CTLA4-Ig alone.

For more information about Coya, please visit **www.coyatherapeutics.com**

Forward-Looking Statements

This press release contains “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 impact of public health emergencies, including COVID-19; 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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