UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, DC 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): **August 29, 2022**

Cognition Therapeutics, Inc.
(Exact name of registrant as specified in its charter)

Delaware	**001-40886**	**13-4365359**
(State or other jurisdiction of incorporation or organization)	(Commission File Number)	(I.R.S. Employer Identification No.)

2500 Westchester Avenue **Purchase, NY**	**10577**
(Address of principal executive offices)	(Zip Code)

Registrant's telephone number, including area code: **(412) 481-2210**

Not Applicable
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of Each Class	**Trading Symbol**	**Name of Exchange on Which Registered**
Common Stock, par value $0.001 per share	CGTX	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

Attached as Exhibit 99.1 and furnished for purposes of Regulation FD is a presentation that Cognition Therapeutics, Inc. may use from time to time in presentations or discussions with investors, analysts, and other parties.

The information in this Item 7.01 (including Exhibit 99.1) is being furnished solely to satisfy the requirements of Regulation FD and shall not be deemed to be "filed" for the purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that Section, nor shall it be deemed to be incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

The following exhibit is being furnished herewith:

Exhibit No.	Document
99.1	Investor presentation of Cognition Therapeutics, Inc.
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

COGNITION THERAPEUTICS, INC.

By: */s/ Lisa Ricciardi*
Name: Lisa Ricciardi
Title: President and Chief Executive Officer

Date: August 29, 2022

COGNITION™ Therapeutics

Developing disease-modifying medicines for degenerative disorders

August 2022

Forward-looking Statement

This presentation contains forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995. All statements contained in this presentation, other than statements of historical fact statements that relate to present facts or current conditions, including but not limited to, statements regarding our cash and financial resources and our clinical development plans, are forward-looking statements. Th statements involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance, or achievements to be materially different from any future results, performanc achievements expressed or implied by the forward-looking statements. In some cases, you can identify forward-looking statements by terms such as "may," "might," "will," "should," "expect," "plan," "aim," "seek," "an "could," "intend," "target," "project," "contemplate," "believe," "estimate," "predict," "forecast," "potential" or "continue" or the negative of these terms or other similar expressions. We have based these forward-looking statements largely on our current expectations and projections about future events and financial trends that we believe may affect our business, financial condition, and results of operations. These forward-looking statements speak only as of the date of this presentation and are subject to a number of risks, uncertainties and assumptions, some of which cannot be predicted or quantified and some of which are beyond our co Factors that may cause actual results to differ materially from current expectations include, but are not limited to: competition, our ability to secure new (and retain existing) grant funding, our ability to grow and mana growth, maintain relationships with suppliers and retain our management and key employees; our ability to successfully advance our current and future product candidates through development activities, preclinical and clinical trials and costs related thereto; the timing, scope and likelihood of regulatory filings and approvals, including regulatory approval of our product candidates; changes in applicable laws or regulations; the p that the we may be adversely affected by other economic, business or competitive factors; our estimates of expenses and profitability; the evolution of the markets in which we compete; our ability to implement our s initiatives and continue to innovate our existing products; our ability to defend our intellectual property; the impact of the COVID-19 pandemic on our business, supply chain and labor force; and the risks and uncerta described in the "Risk Factors" section of our annual and quarterly reports filed the Securities Exchange Commission. You should not rely on these forward-looking statements as predictions of future events. The ev circumstances reflected in our forward-looking statements may not be achieved or occur, and actual results could differ materially from those projected in the forward-looking statements. Moreover, we operate in a c industry and economy. New risk factors and uncertainties may emerge from time to time, and it is not possible for management to predict all risk factors and uncertainties that we may face. Except as required by ap law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise.

TRADEMARKS

This presentation may contain trademarks, service marks, trade names and copyrights of other companies, which are the property of their respective owners. Solely for convenience, some of the trademarks, servic trade names and copyrights referred to in this presentation may be listed without the TM, SM © or ® symbols, but we will assert, to the fullest extent under applicable law, the rights of the applicable owners, if any, to trademarks, service marks, trade names and copyrights.

MARKET & INDUSTRY DATA

Projections, estimates, industry data and information contained in this presentation, including the size of and growth in key end markets, are based on information from third-party sources and management estimate Although we believe that these third party-sources are reliable, we cannot guarantee the accuracy or completeness of these sources. Our management's estimates are derived from third-party sources, publicly avai information, our knowledge of our industry and assumptions based on such information and knowledge. Our management's estimates have not been verified by any independent source. All of the projections, estim market data and industry information used in this presentation involve a number of assumptions and limitations, and you are cautioned not to give undue weight to such information. In addition, projections, estimate assumptions relating to us and our industry's future performance are necessarily subject to a high degree of uncertainty and risk due to a variety of factors, including, but not limited to, those described above, that c cause future performance to differ materially from our expressed projections, estimates and assumptions or those provided by third parties.

Who We Are

Targeting unmet needs in age-related degenerative disorders of the central nervous system and retina such as Alzheimer's disease, dementia with Lewy bodies (DLB), dry age-related macular degeneration (dry AMD), and Parkinson's disease

Functionally distinct therapeutic approach focused on the sigma-2 (σ-2) receptor complex, backed by decades of expertise in the biology of synaptic function and plasticity

Received over $160 million in non-dilutive grant funding through key collaborations with the National Institute of Aging and other thought-leading institutions

- *Approximately 50% of ongoing R&D expenses covered by grants*

Multiple Phase 2 programs expected to provide significant value-creating milestones and data readout over the next 12 to 24 months

Expanding pipeline through our proprietary NICE screening platform, strategic alliances and acquisiti

Seasoned management team with broad scientific, drug development and commercial expertise; experienced board and advisors

Pipeline

Product Candidate	Target Indication	Preclinical	Phase 1	Phase 2	Phase 3	Funding
CT1812	**SEQUEL** Mild-moderate AD					$3.3M NIH NIA
CT1812	**SHINE** Mild-moderate AD					$30M NIH NIA
CT1812	**SHIMMER** DLB					$30M NIH NIA
CT1812	**START** Early-stage AD					$81M NIH NIA
CT1812	**Dry AMD**					
CT2168	**Synucleinopathies** †	*IND enabling studies*				THE MICHAEL J. FOX FOUNDATION FOR PARKINSON'S RESEARCH
CT2074	**Dry AMD**	*IND enabling studies*				

† *including Parkinson's disease and DLB*

Please note: CT1812 and other pipeline candidates are not approved for use in the US or other jurisdictions

Grants Continue to Provide Funding

Approximately 50% of R&D Funded by Grants

Clinical Study	Total Awarded	Remaining (yet to be drawn down)
START (early AD)	$ 81 Million	$ 65.1 Million
SHINE (mild/mod AD)	$ 30.5 Million	$ 11 Million
SHIMMER (DLB)	$ 29.5 Million	$ 20.5 Million
SEQUEL (qEEG in AD)	$ 3.3 Million	$ 0.4 Million
	$ 144.3 Million	**$ 97 Million**

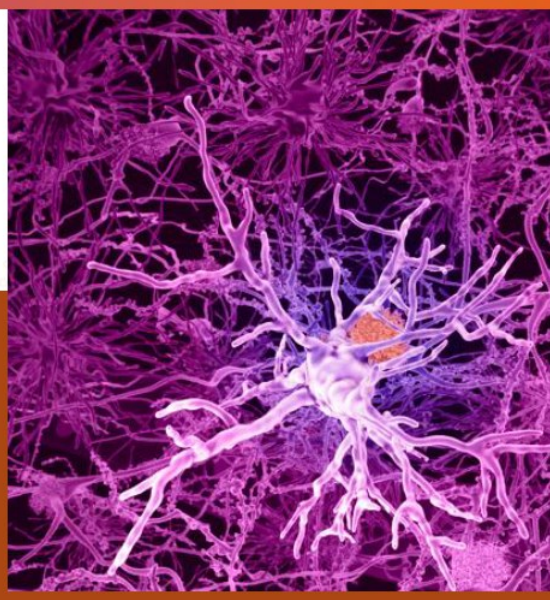
NOTE: figures are approximate dollar amounts as June 30, 2022

Key Upcoming Milestones & Expected Timing

2021	2022	2023
Completed ✓ SNAP ✓ SPARC ✓ SHINE (cohort 1)	**Completing** • PK • Human AME • SEQUEL **Conducting** • SHINE (cohort 2) • Phase 2 in DLB • Phase 2 with ACTC • Phase 2 in dry AMD • IND-enabling studies for research compounds	**Completing** • SHINE (cohort 2) **Ongoing Studies** • Phase 2 in DLB • Phase 2 with ACTC • Phase 2 in dry AMD **Regulatory Actions** • IND filings for pipeline compounds

Cognition Lead Program:

Review of CT1812 for the Treatment of Alzheimer's Disease

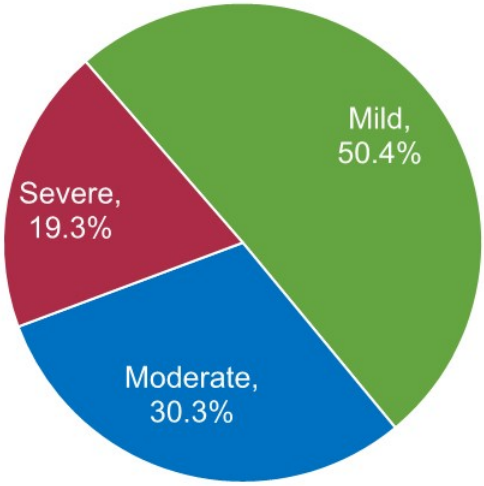


COGNITION Therapeutics

Alzheimer's Disease Market Overview

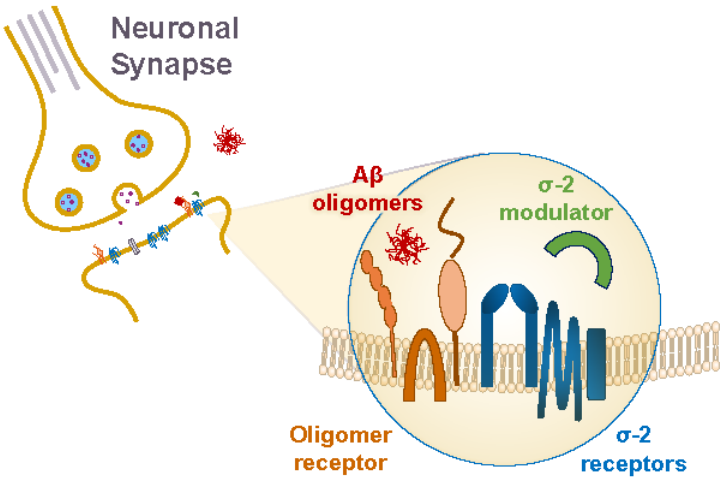
- Approx. 6.2 million individuals in United States are afflicted with Alzheimer's disease[1]
  - Approximately 35 million people worldwide[2]
  - Prevalence expected to double by 2050[1]
- Direct healthcare costs for patients with Alzheimer's and other dementias is estimated to exceed $300 billion in the United States alone[1]
  - Projected to increase to $1+ trillion by 2050[1]

Stages of Disease Afflicting Current Population of Alzheimer's Patients



1) Economic Burden of Alzheimer Disease and Managed Care Considerations. Am J Manag Care. 2020;26:S177-S183
2) World Health Organization Key Facts: Dementia

CT1812: A Synaptoprotective Approach to Alzheimer's Disea



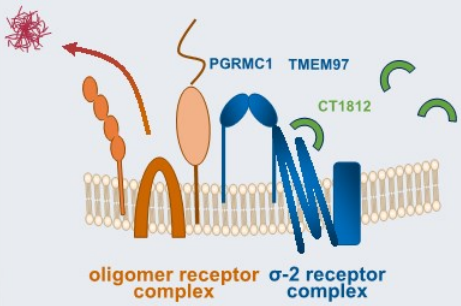
- CT1812 penetrates the blood-brain barrier (BBB) and binds selectively to the σ-2 receptor
- By modulating σ-2, CT1812 displaces and prevents oligomers from binding to neurons and clears them into CSF
- Prevents synaptotoxicity and facilitates restoration of neuronal function
- CT1812 mimics the protective effects of the A673T-APP "Icelandic" mutation

- *Izzo NJ, et al. Preclinical and clinical biomarker studies of CT1812: A novel approach to Alzheimer's disease modification. Alzheimer's Dement. 2021;1–18*
- *Izzo NJ, et al. Alzheimer's therapeutics targeting amyloid beta 1-42 oligomers II: Sigma-2/PGRMC1 receptors mediate Abeta 42 oligomer binding and synaptotoxicity PLoS One. 2014 Nov 12; 9(11):e111899*
- *Izzo NJ, et al. Alzheimer's therapeutics targeting amyloid beta 1-42 oligomers I: Abeta 42 oligomer binding to specific neuronal receptors is displaced by drug candidates that improve cognitive deficits PLoS One. 2014 Nov 12; 9(11):e1118*
- *Limegrover, CS, et al. Alzheimer's Protection Effect of A673T Mutation May Be Driven by Lower Aβ Oligomer Binding Affinity. J Neurochem. 2020; 00: 1– 15. doi:10.1111/jnc.15212*

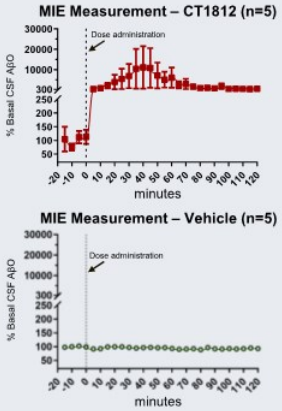
Evidence of Target Engagement: *SNAP (COG0104)*

First evidence of target engagement in humans, mirrors preclinical findings, corroborating our mechanism of act

CT1812 mechanism of action: By binding to the σ-2 receptor, CT1812 displaces Aβ oligomers from synapses, thus preventing synaptic toxicity

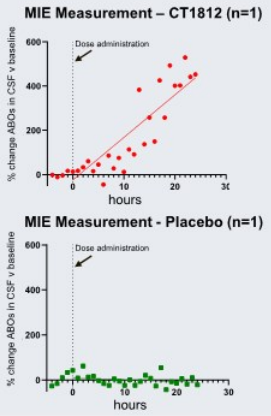


This displacement can be measured in the CSF in Alzheimer's mice treated with CT1812



Izzo, NJ et al. Alzheimer's Dement. 2021; 17: 1365-1382.
https://doi.org/10.1002/alz.12302

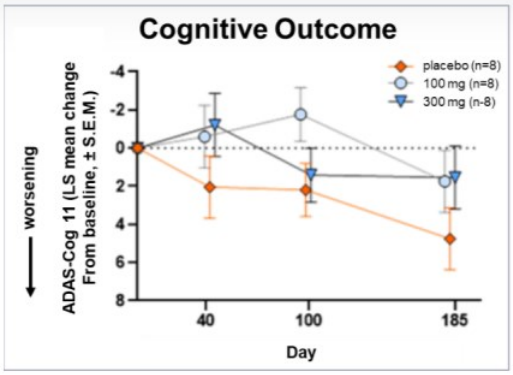
…and now in people with Alzheime disease



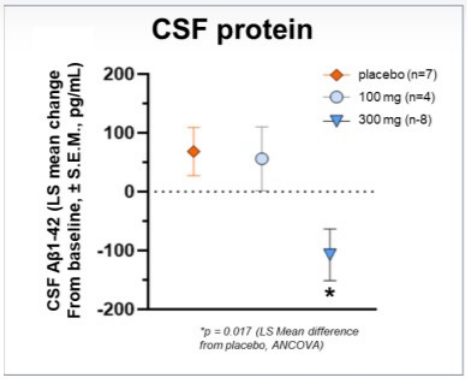
SNAP COG0104 study (NCT03522129)
funded by NIA grant 1RF1AG057780

Cognitive & Biological Outcomes

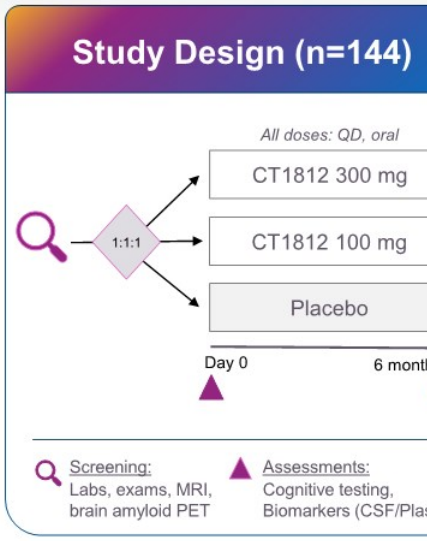
SHINE interim analysis (n=24) yields promising evidence



- Trend towards slower cognitive decline in CT1812-treated vs placebo-treated participants as measured by ADAS-Cog 11



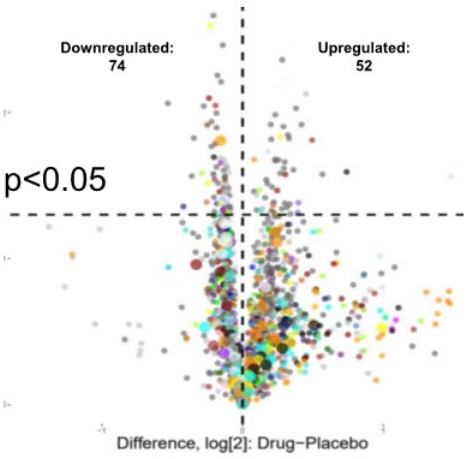
- Statistically significantly lower Aβ protein (p=0.017) in treated vs placebo patients
- Additional analyses on p-tau, synaptic and AD-related proteins ongoing



SHINE COG0201 study (NCT03507790)
funded by NIA grant R01AG058660

Biomarkers Associated with Alzheimer's Pathology Normalized by CT1812

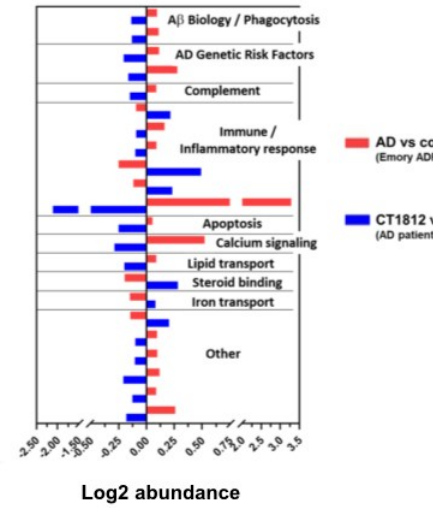
Pharmacodynamic Biomarkers of CT1812 Discovered



Most Highly Represented Pathways

- M4 Synapse/Neuron
- M3 Oligo/Myelination
- M1 Synapse/Neuron
- M21 MHC Complex/Immune
- M42 Matrisome
- M17 Transcription
- M11 Cell-ECM Interaction
- M29 Glycosylation/ER
- M7 MAPK/Metabolism
- M13 RNA Splicing
- M2 Mitochondria
- M16 RNA Binding

Biomarkers normalized by CT1812



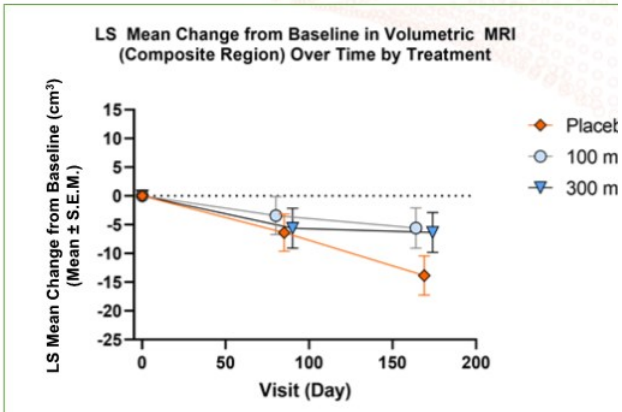
SPARC (COG0105) Results

1° Endpoints:

- Impact of CT1812 on synaptic density as measured by SV2A; safety and tolerability

Results:

- No difference in synaptic density in CT1812- or placebo-treated vs baseline
- Trend towards preservation of brain volume (composite) in CT1812- vs placebo-treated
  - Statistically significant ($p<0.05$) improvement in volume in three regions (bottom right)
- Adverse event profile consistent with prior studies
  - Elevated liver enzymes resolved upon discontinuation of study drug
  - No serious TEAEs were reported



MRI Brain Volume (cm^3)
6-mo change from baseline

	CT1812 (Pooled)	Placebo	P-value v placebo
Hippocampus	-0.09 (0.08)	-0.40 (0.11)	0.0412
Prefrontal Cortex	-0.41 (0.95)	-4.80 (1.37)	0.0125
Pericentral Cortex	0.07 (0.53)	-2.90 (0.77)	0.0032

SPARC COG0105 study (NCT03493282) funded by NIA grant RF1AG057553

Proteomic Data from SPARC Shows Effect of CT1812 on Disrupted Alzheimer's Disease Processes

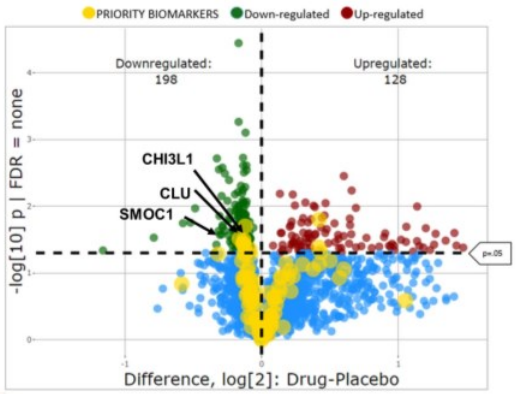
- 37 proteins in CSF were significantly ($p<0.05$) normalized towards control with CT1812 compared to placebo
- These proteins are involved in key pathways disrupted in Alzheimer's: autophagy, inflammation, synaptic function
  - Include previously well-characterized biomarkers of Alzheimer's disease, such as YKL-40 and genetic risk factors for Alzheimer's disease such as clusterin
- Analyses support targeting σ-2 receptor with CT1812

NOTE: proteomic analyses conducted on 18 SPARC participants for whom there were CSF samples at baseline and at end of study

Biomarkers of CT1812 Identified, Several of Which May Reflect Disease Modification

NEW

Pharmacodynamic Biomarkers of CT1812 Discovered

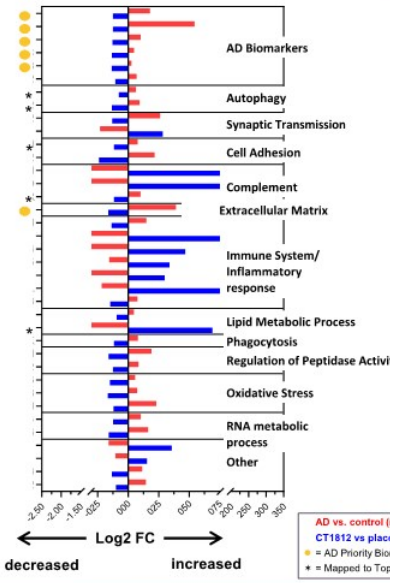


CHI3L1 (YKL-40) = inflammatory protein ↑ in Alzheimer's disease
SMOC1 = Aβ plaque-associated protein ↑ in Alzheimer's disease
CLU = Alzheimer's disease genetic risk factor (clusterin; APOJ)

Most Highly Represented Pathways

M3	Oligo / Myelination
M26	Complement / Acute Phase
M7	MAPK / Metabolism
M4	Synapse / Neuron
M1	Synapse / Neuron
M14	Protein Folding
M21	MHC Complex / Immune
M27	Extracellular Matrix

Biomarkers normalized by CT1812



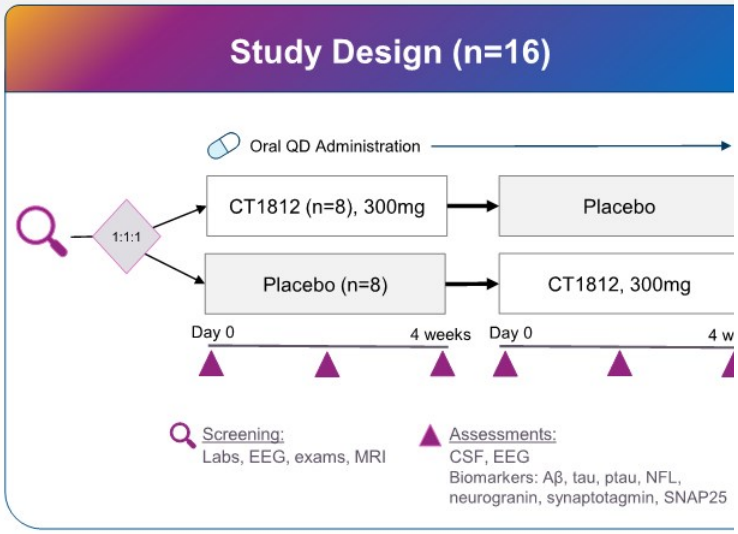
NOTE: proteomic analyses conducted on 18 SPARC participants for whom there were CSF samples at baseline and at end of study

SEQUEL (COG0202)

Assessment of brain wave activity via quantitative EEG

- Principal investigator: E. Vijverberg, MD, PhD; neurologist at Vumc Alzheimer's Center

  Amsterdam UMC
  Universitair Medische Centra
- Single-site quantitative EEG study in patients with mild-to-moderate AD
- Two group cross over design
- Objective: evaluate the efficacy of CT1812 in restoring synaptic function through quantitative EEG, as reflected by relative theta power

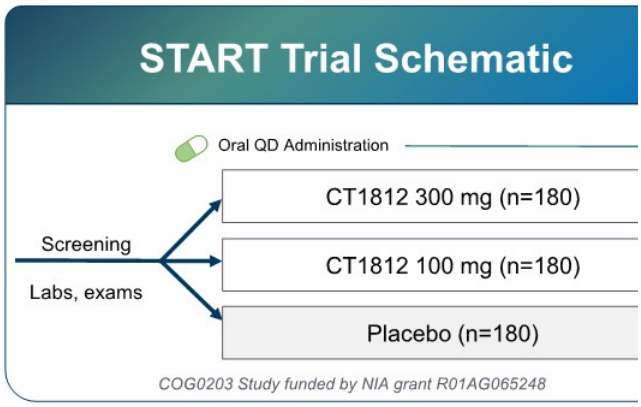


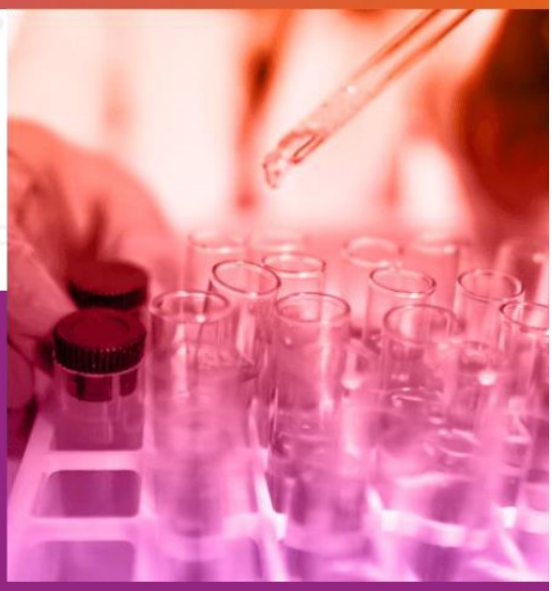
SEQUEL COG0202 study (NCT04735536) funded by NIA grant RF1AG058710

Patient Population: Early AD

Substantial grant award of $81M funds program

- Phase 2 efficacy study, powered to show change in cognition: slowing or halting cognitive decline
- Enrollment expected to commence 2H 2022
- Enrolling 540 participants with early Alzheimer's disease at 50-60 U.S. sites
- Supported by $81M Grant from NIA
  - Grant awarded in collaboration with ACTC: premier Alzheimer's clinical trial group





Cognition Pipeline:

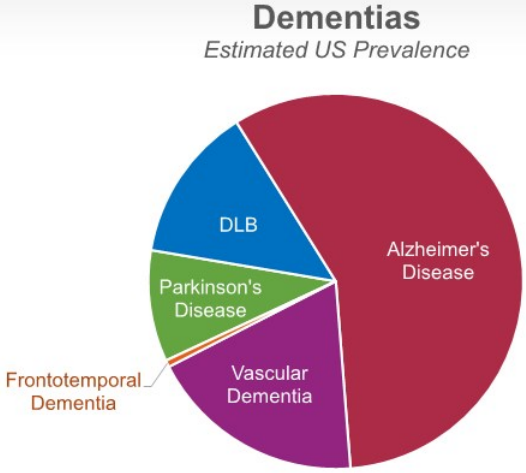
Synucleinopathies

Disorders such as DLB and Parkinson's disease that are characterized by deposits of α-synuclein aggregates (called Lewy bodies) that disrupt key cellular processes

COGNITION Therapeutics

Synucleinopathies are 2nd only to AD in Prevalence

- In the U.S., approximately 13 million people have a form of dementia[1]
  - Dementia with Lewy bodies (DLB): 1.4 million
  - PD: 1 million
- Direct healthcare costs:
  - DLB: $31.5 billion[2]
  - PD: $25 billion[3]



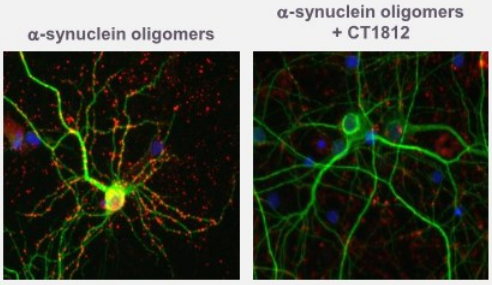
α-synuclein oligomers, which disrupt key cellular processes, are a hallmark of both disorders

1) Milken Institute report: (2019) Reducing the Cost and Risk of Dementia: Recommendations to Improve Brain Health and Decrease Disparities.
2) LBDA (extrapolated): LBD is the Most Expensive Dementia in America and Yingjia Chen et al Alzheimers Dement. 2019
3) MJFF and The Lewin Group: Economic Burden and Future Impact of Parkinson's Disease (2019)

σ-2 Modulators May be Disease Modifying in Synucleinopathies

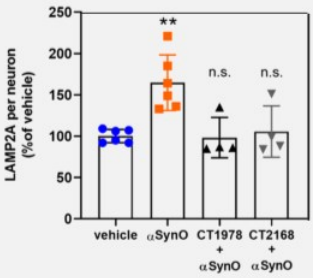
Cellular evidence that σ-2 modulators have a beneficial impact

Cognition antagonists block the binding / internalization of α-synuclein oligomers at synapses

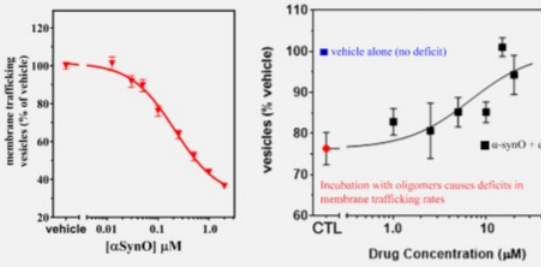


α-Synuclein oligomers in red

Cognition compounds (black and gray) reverse the effect of α-synuclein oligomers on LAMP2a (orange)



α-synuclein oligomer-induced trafficking deficits (red) are reversed by Cognition drug (black)



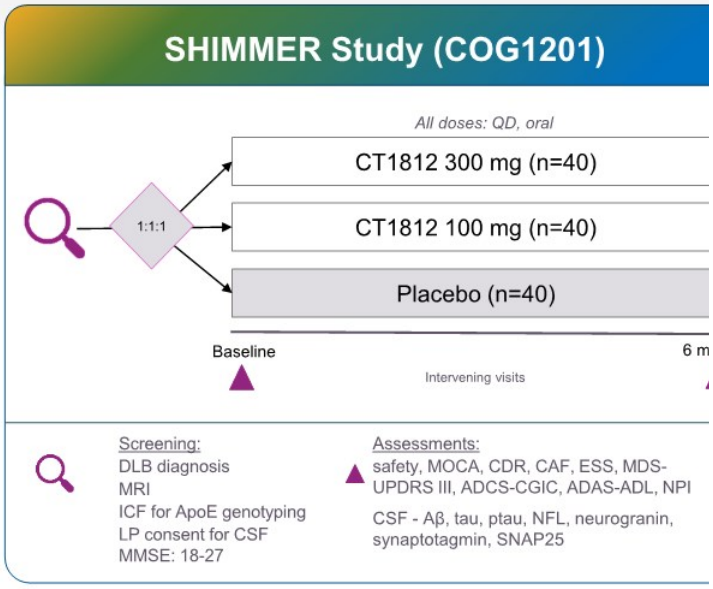
Limegrover CS, et al. *J Neurosci Res*. 2021. doi: 10.1002/jnr.24782

DLB Phase 2 Funded with ~$30M NIA Grant

- Principal investigator: James E. Galvin, MD, MPH, professor of neurology and director of the Comprehensive Center for Brain Health

  MIAMI MILLER SCHOOL of MEDICINE

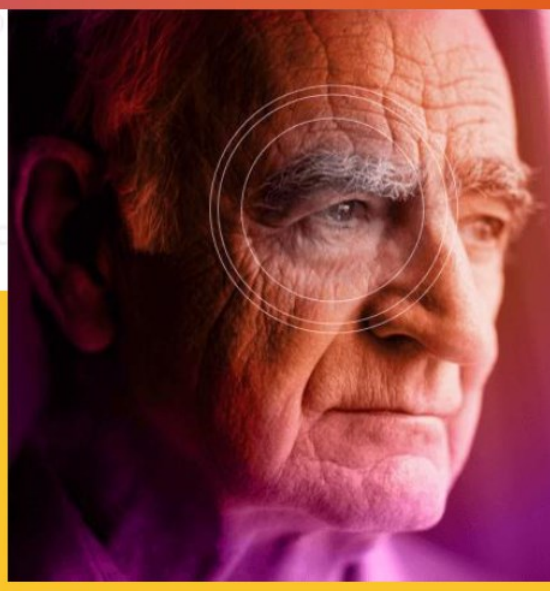
- Phase 2 SHIMMER trial ongoing
  - Participant dosing underway
- U Miami and 20+ academic sites
- *Archived DLB R&D educational symposium available: https://ir.cogrx.com*



COG1201 study partially funded by NIA grant R01AG071643

Cognition Pipeline:

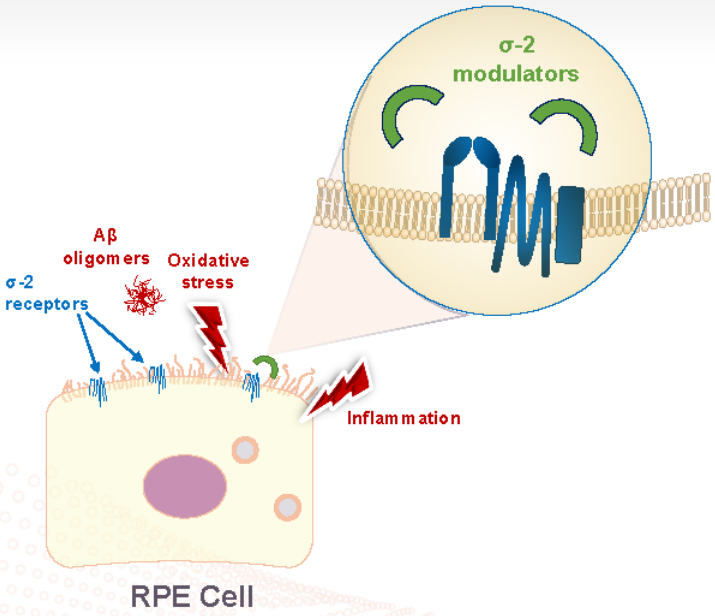
Dry Age-related Macular Degeneration



COGNITION Therapeutics

Rationale for σ-2 Modulators in Dry AMD

Goal: Protect RPE Cells From Disease-relevant Stressors



σ-2 receptors

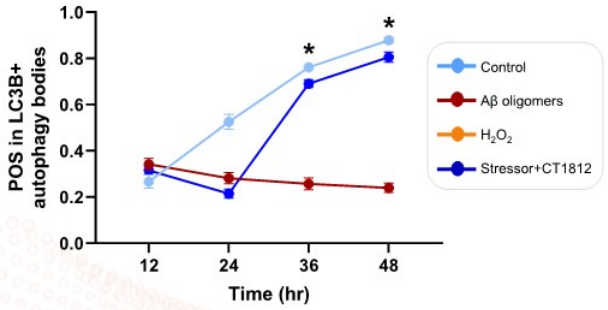
- **Expression:** in RPE cells, retinal ganglion cells, photoreceptors in retina
- **Biology**: Regulates autophagy, protein trafficking, lipid metabolism dysregulated in AMD
- **Target validation:** TMEM97 knockdown is protective
- **Human genetics:** Linked to dry AMD

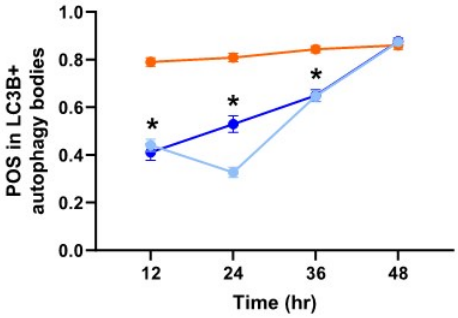
σ-2 receptor modulators

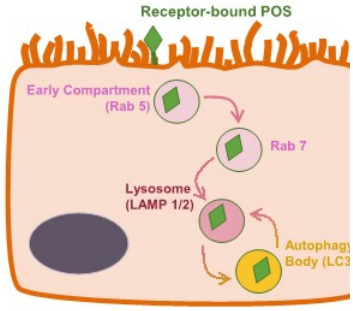
- **Non-invasive oral small molecule approach to reach ret**
- **Clinical biomarker support**: Proteomics analyses showed differential movement of proteins involved in dry AMD
- **Preclinical data:** regulates cell survival and inflammatory pathways, ameliorate trafficking deficits

Results: *σ-2 Receptor Modulators Prevent Deficits in Trafficking / Degradation of POS from 2 Insults*

- Aβ oligomers and oxidative stress cause lysosomal deficits in capacity to traffic & degrade photoreceptor outer segments (POS) cargos in RPE cells
- σ-2 receptor modulators from 3 chemically distinct series rescued these deficits

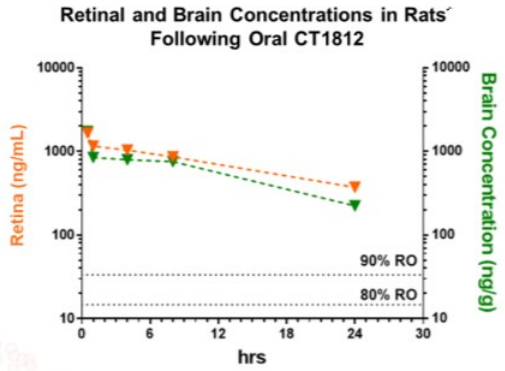




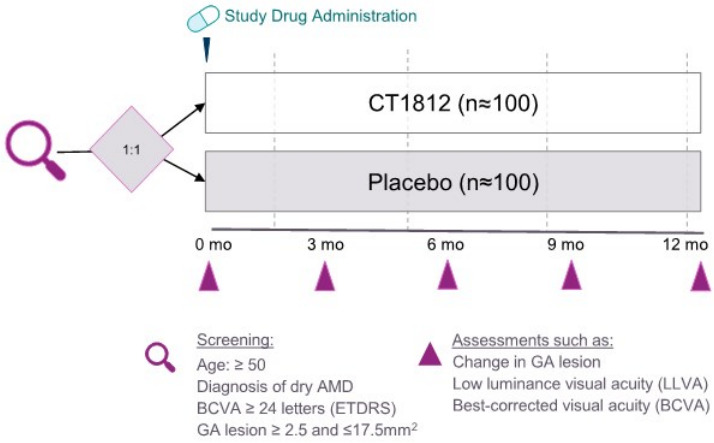


Genetic, Clinical Biomarker and Preclinical Data Support Moving Forward with CT1812 for dAMD

Orally administered CT1812 reaches the target tissue at >80% RO



Planned Phase 2 will assess CT1812 in patients with diagnosed dry AMD and measurable GA



Screening:
Age: ≥ 50
Diagnosis of dry AMD
BCVA ≥ 24 letters (ETDRS)
GA lesion ≥ 2.5 and ≤17.5mm²

Assessments such as:
Change in GA lesion
Low luminance visual acuity (LLVA)
Best-corrected visual acuity (BCVA)

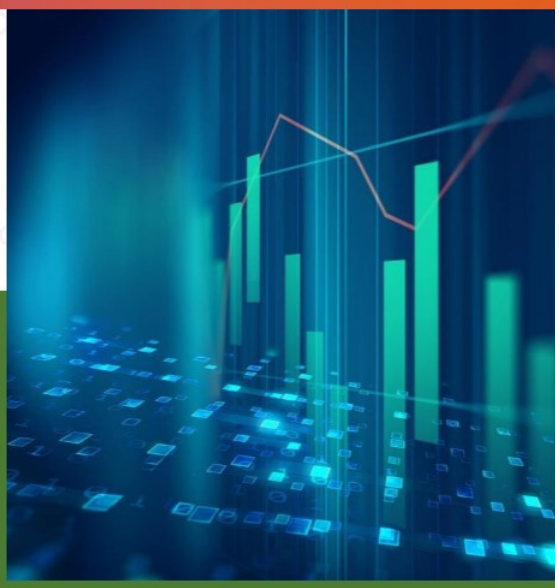
Financial Position

Financials as of June 30, 2022

- Cash and Cash Equivalents: $45.8 million
- Proceeds raised from IPO: $52.0 million
- Expected cash runway into 2H 2023

NIA funding for CT1812 studies as of June 30, 2022

- Preclinical through Phase 2 studies $168.4 million
- Approximate funding used ($70.4 million)
  - *Remaining NIA funding* **$98.0 million**



COGNITION Therapeutics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