COGNITION™ Therapeutics

Disease-modifying medicines for degenerative disorders

January 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 facts or statements that relate to present facts or current conditions, including but not limited to, statements regarding the Company's cash and financial resources and its clinical development plans, are forward-looking statements. These 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, performance, or 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," "anticipate," "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 control, including, among others: our ability to successfully advance our current and future product candidates through development activities, preclinical studies, and clinical trials; the timing, scope and likelihood of regulatory filings and approvals, including final regulatory approval of our product candidates; the impact of the COVID-19 pandemic on our business and operations; the performance of third parties in connection with the development of our product candidates, including third parties conducting our future clinical trials as well as third-party suppliers and manufacturers; our ability to successfully commercialize our product candidates and develop sales and marketing capabilities, if our product candidates are approved; and our ability to maintain and successfully enforce adequate intellectual property protection. These and other risks and uncertainties are described more fully in the "Risk Factors" section of our most recent filings with the Securities and Exchange Commission and are available at www.sec.gov. You should not rely on these forward-looking statements as predictions of future events. The events and 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 dynamic 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 applicable 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, service marks, trade names and copyrights referred to in this presentation may be listed without the TM, SM © or ® symbols, but the Company will assert, to the fullest extent under applicable law, the rights of the applicable owners, if any, to these 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 estimates. Although the Company believes that its third party-sources are reliable, the Company cannot guarantee the accuracy or completeness of its sources. The Company's management estimates are derived from third-party sources, publicly available information, the Company's knowledge of its industry and assumptions based on such information and knowledge. The Company's management estimates have not been verified by any independent source. All of the projections, estimates, 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, estimates and assumptions relating to the Company's and its 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 could cause future performance to differ materially from the Company's 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 funding through key collaborations with the National Institute of Aging and other thought-leading institutions

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

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

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

Addressing Degenerative Disease by Targeting Sigma-2 (σ-2)

Cellular Homeostasis

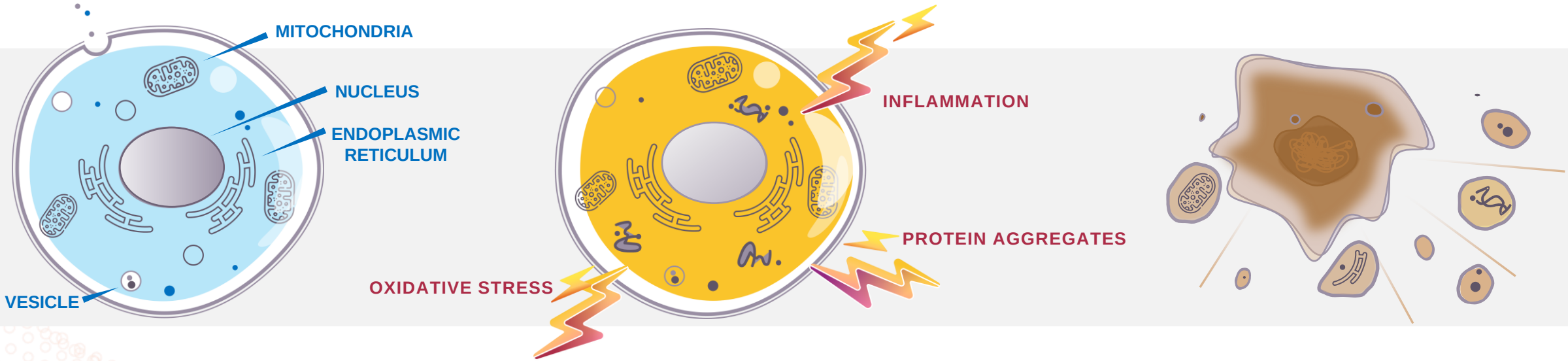
Several key cellular processes (vesicle trafficking and autophagy) required for homeostasis are disrupted in disease

Degeneration

Build-up of age-related stressors (protein aggregates, oxidative stress, inflammation) leads to progressive cellular degeneration in diseases like Alzheimer's, Parkinson's and dry AMD resulting in declines in cognition, vision and other functions

Dysfunction / Death

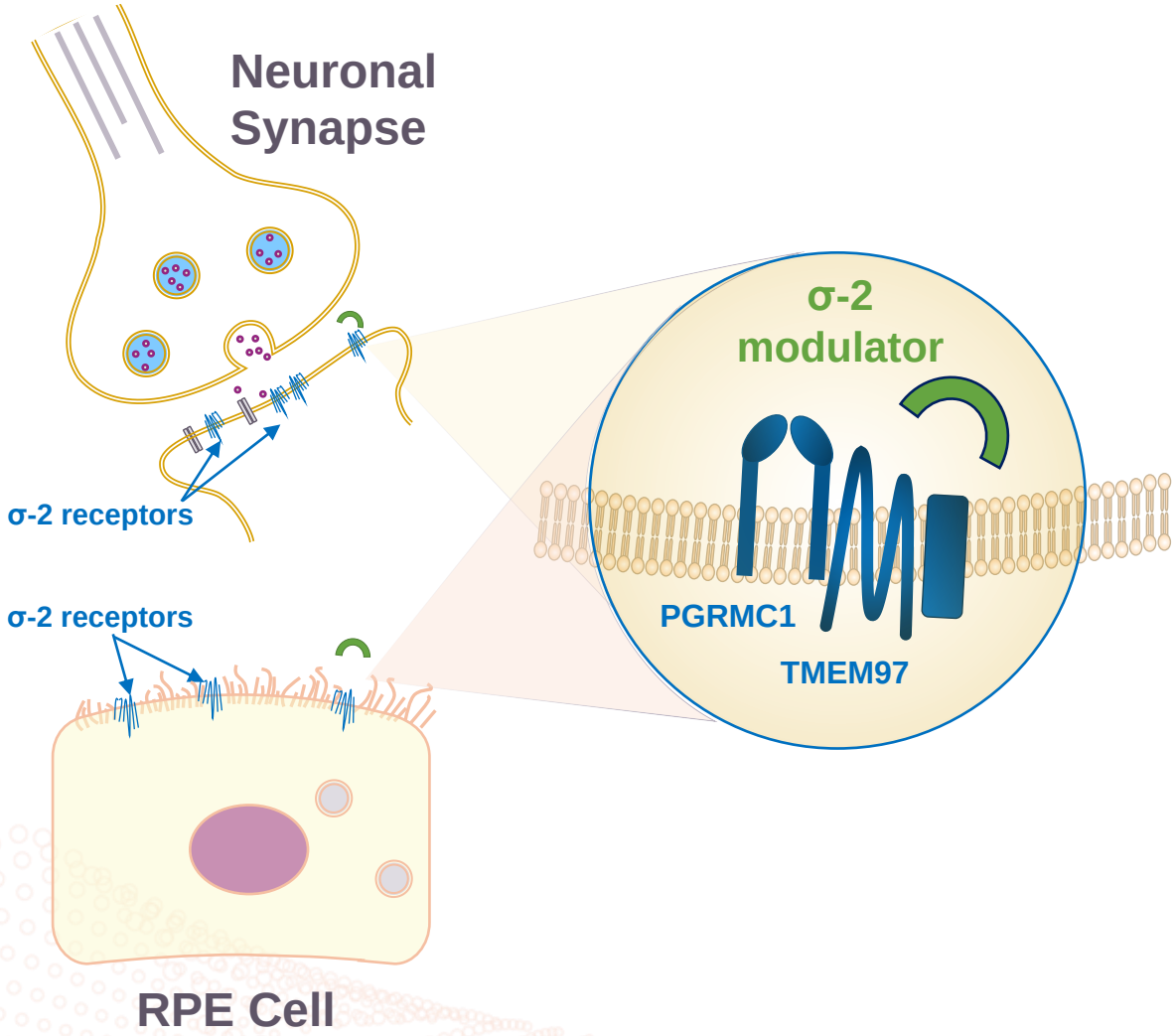
Untreated, degeneration leads to cell death, which in turn results in decreased quality of life and/or shorter life span



σ-2 receptor complex components, PGRMC1 and TMEM97, control or influence these processes

COGNITION™ Therapeutics

Modulating σ-2 Receptor Restores Cellular Dysfunction



- CT1812 is orally available, penetrates the blood brain barrier and binds selectively to the σ-2 receptor
- The σ-2 receptor complex is expressed in:
  - Brain: cortex, hippocampus, substantia nigra, cerebellum
  - Retina: retinal pigment epithelial (RPE) cells, retinal ganglion cells, photoreceptors
- The σ-2 receptor complex, TMEM97 and PGRMC1, regulates key cellular processes disrupted in degenerative diseases, such as:
  - Autophagy
  - Cell survival
  - Trafficking
  - Cell function

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	**ACTC** Early-stage AD					$81M NIH NIA
CT1812	**DLB**					$30M NIH NIA
CT1812	**Dry AMD ***					
CT2168	**Synucleinopathies †**	*IND enabling studies*				MJFF
CT2074	**Dry AMD**	*IND enabling studies*				

** Provided the FDA agrees, we intend to proceed with a Phase 2 study supported by the Phase 1 AD 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

Key Upcoming Milestones

2021

Completed

- ✓ SNAP
- ✓ SPARC
- ✓ SHINE (cohort 1)

2022

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

2023

Completing

- SHINE (cohort 2)

Ongoing Studies

- Phase 2 in DLB
- Phase 2 with ACTC
- Phase 2 in dry AMD

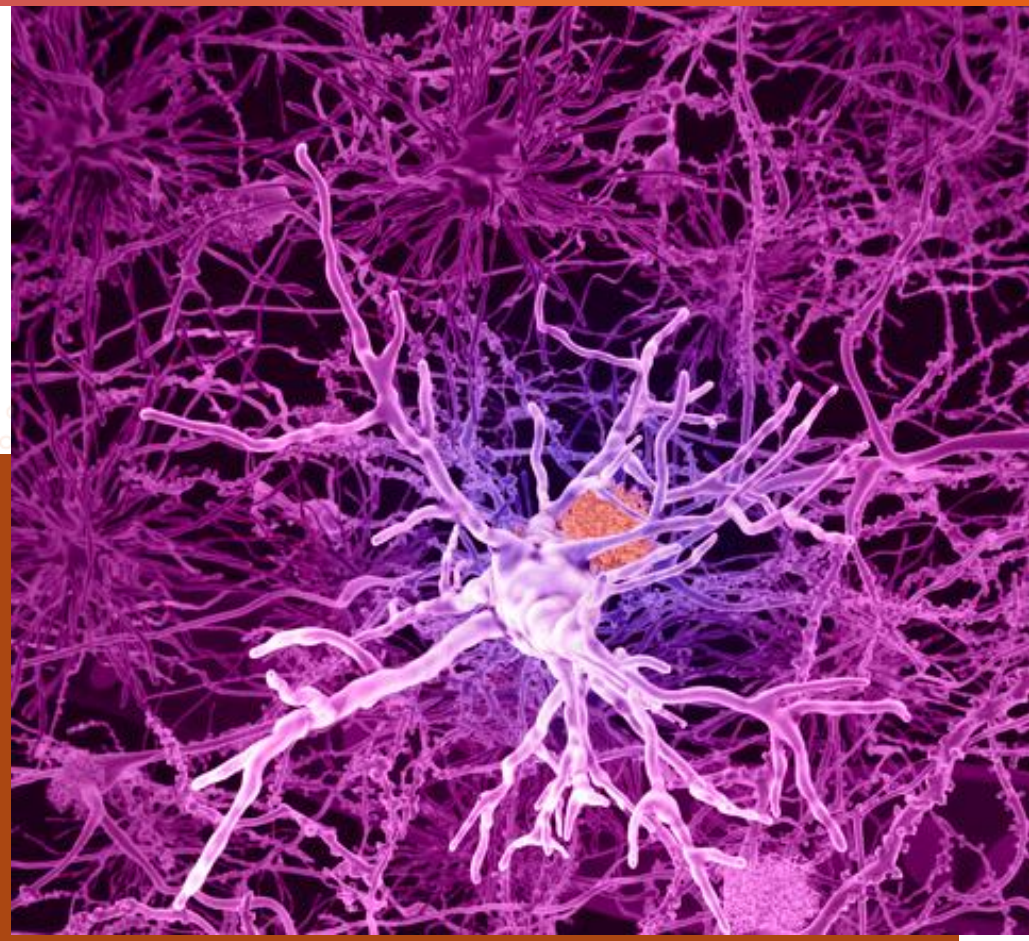
Regulatory Actions

- IND filings for pipeline compounds

** Subject to discussion with FDA*

Cognition Lead Program:

Review of CT1812 for the Treatment of Alzheimer's Disease

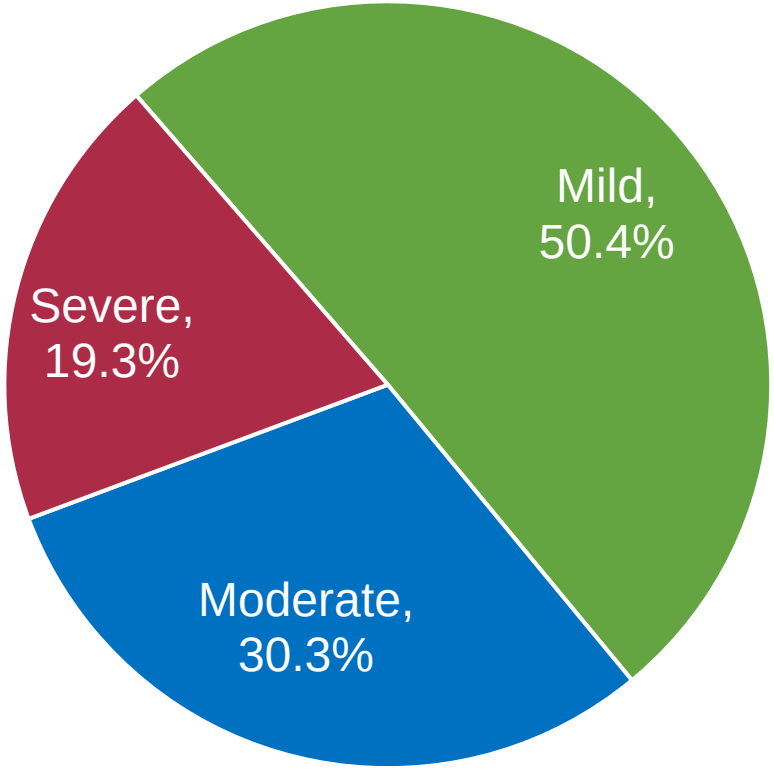


COGNITION™ Therapeutics

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]

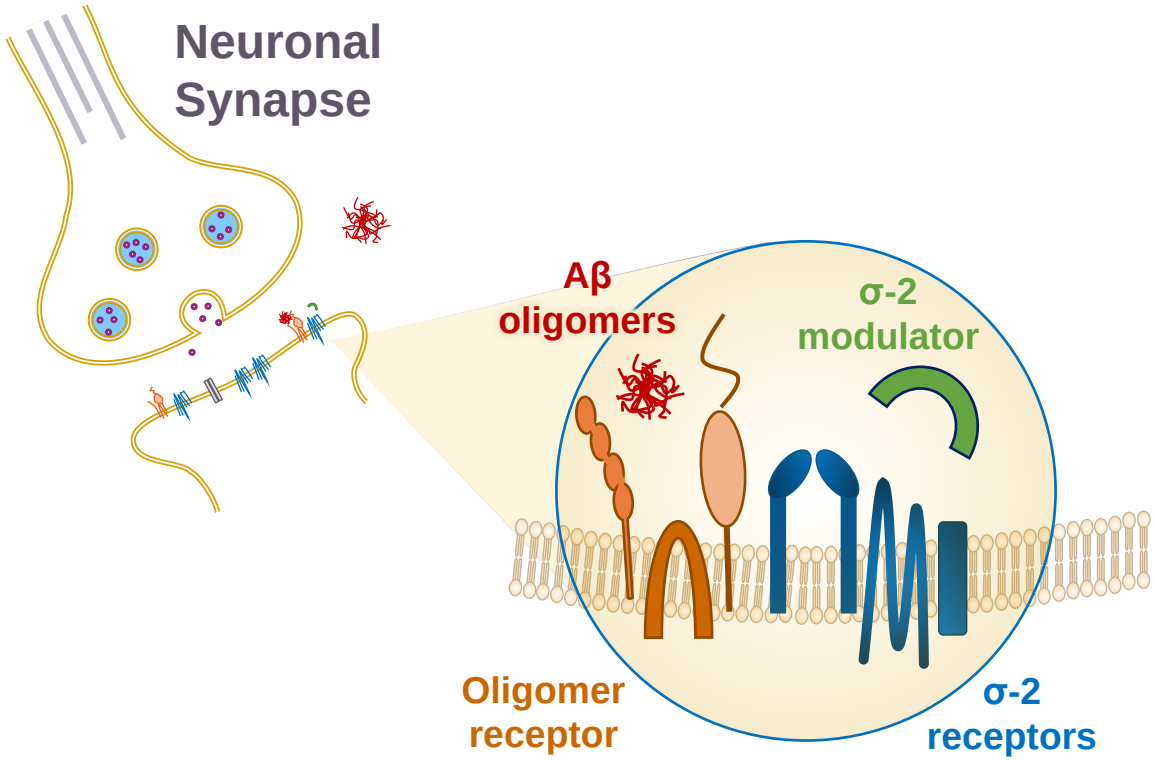
Stages of Disease Afflicting Current Population of Alzheimer's Patients



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

COGNITION™ Therapeutics

CT1812: A Synaptoprotective Approach to Alzheimer's Disease



- CT1812 penetrates the blood brain barrier 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

- *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):e111898*

Alzheimer's Clinical Program Overview

	FIH / Safety		Proof of Concept / Mechanism				Impact on Disease Pathology	
Study	**SAD/MAD** COG0101 (n=93)	**DDI** COG0103 (n=15)	COG0102 (n = 19)	**SNAP** COG0104 (n=3)	**SPARC** COG0105 (n = 23)	**SEQUEL** COG0202 (n = 16)	**SHINE** COG0201 (n = 120)	**ACTC** COG0203 (n=540)
Population	Healthy Volunteers		Mild-Moderate Alzheimer's				Mild-Moderate Alzheimer's	Early Alzheimer's
Status	Completed 2015	Completed 2016	Completed 2018	Completed 1H2021	Enrollment completed	Ongoing	Ongoing Topline 1H2023	Enrollment commencing 2022
Results	Well tolerated	No clinically significant DDI	Well tolerated	Evidence of target engagement	Topline reported	Topline 2023	*Interim*: trend in cognitive improvement	

Unique Protective Effect: *A673T-APP Mutation Supports CT1812 MoA*

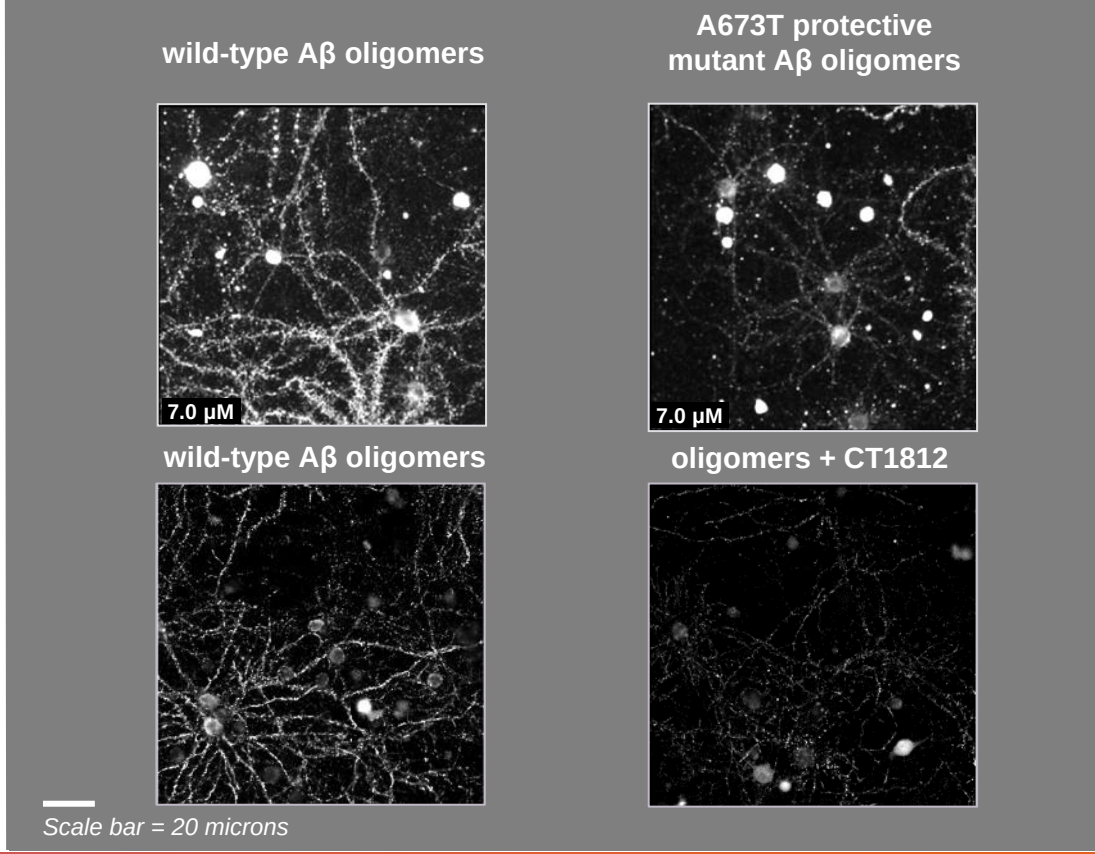
- First variant associated with protection against Alzheimer's disease[1]
- Carriers are four times less likely to get Alzheimer's disease than non-carriers[2]
- Internal data demonstrates that mutant Aβ oligomers bind with four-fold lower affinity to neuronal synapses than normal protein
- To our knowledge, CT1812 is the only drug that mimics the protective effects of this mutation

Journal of Neurochemistry JNC The Official Journal of the International Society for Neurochemistry

Alzheimer's Protection Effect of A673T Mutation May Be Driven by Lower Aβ Oligomer Binding Affinity

Colleen S. Limegrover, Harry LeVine III, Nicholas J. Izzo, Raymond Yurko, Kelsie Mozzoni, Courtney Rehak, Kelsey Sadlek, Hank Safferstein, Susan M. Catalano

Journal of Neurochemistry. doi: https://doi:10.1111/jnc.15212



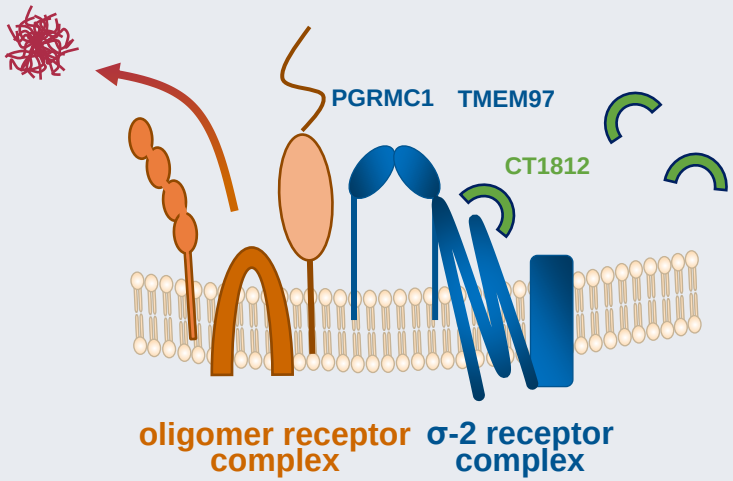
1) *Jonsson, T et al. A mutation in APP protects against Alzheimer's disease and age-related cognitive decline. Nature 488, 96–99 (2012)*
2) *Andrews SJ et al. Protective Variants in Alzheimer's Disease. Curr Genet Med Rep. 2019 March ; 7(1): 1–12*

COGNITION™ Therapeutics

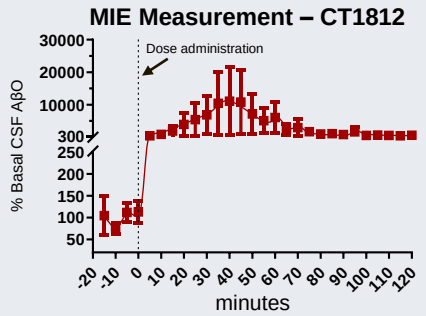
Evidence of Target Engagement: *SNAP (COG0104)*

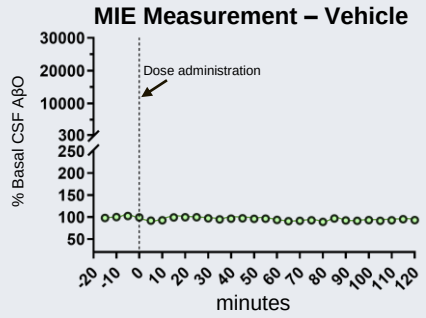
First evidence of target engagement in humans, mirrors preclinical findings, corroborating our mechanism of action

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



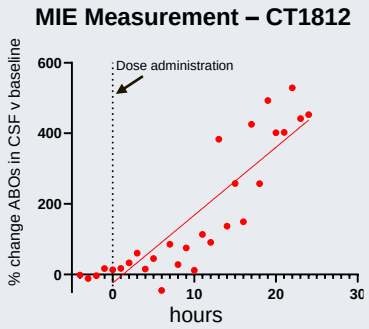
This displacement can be measured in the CSF in Alzheimer's mice treated with CT1812

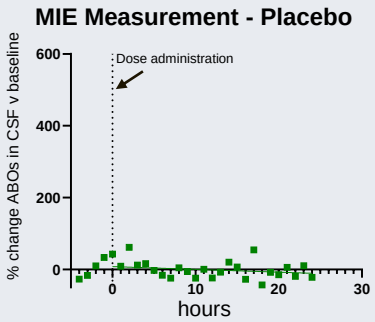




Izzo, NJ et al. Alzheimer's Dement. 2021; 17: 1365-1382. https://doi.org/10.1002/alz.12302

…and now in people with Alzheimer's disease



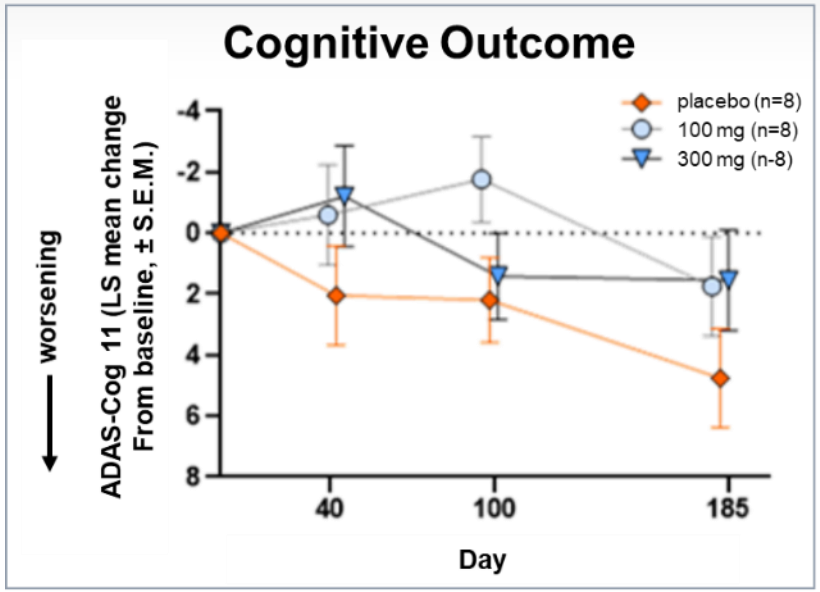


SNAP COG0104 study (NCT03522129) funded by NIA grant 1RF1AG057780

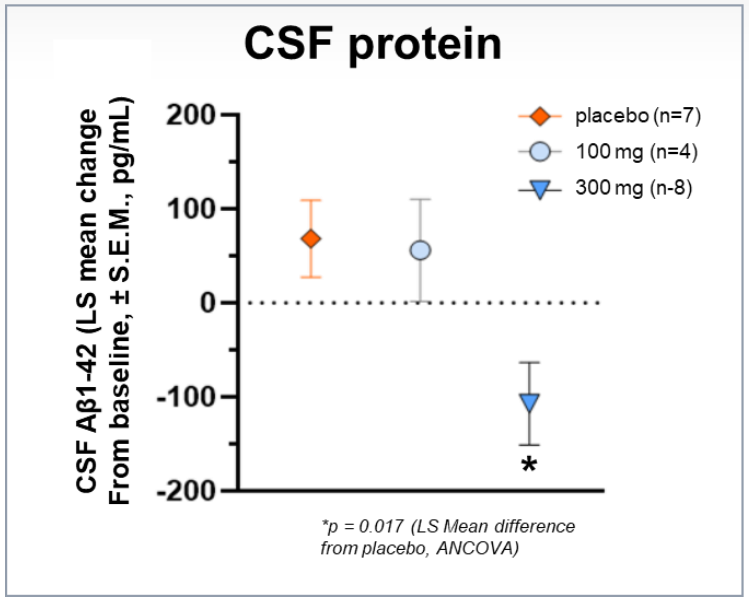
COGNITION™ Therapeutics

Cognitive & Biological Outcomes

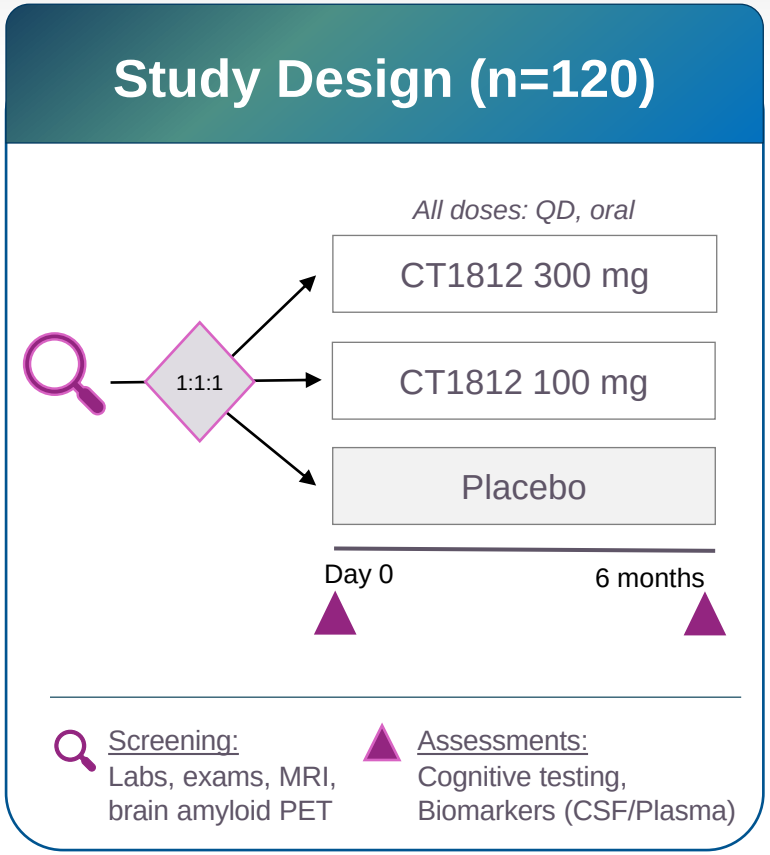
SHINE interim analysis (n=24) yields promising evidence



- 3-point difference (ADAS-COG) between treated and untreated patients at day 185
- Clinically meaningful magnitude of change
- Trend for improved cognitive outcomes



- 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

COGNITION™ Therapeutics

SPARC (COG0105) Results

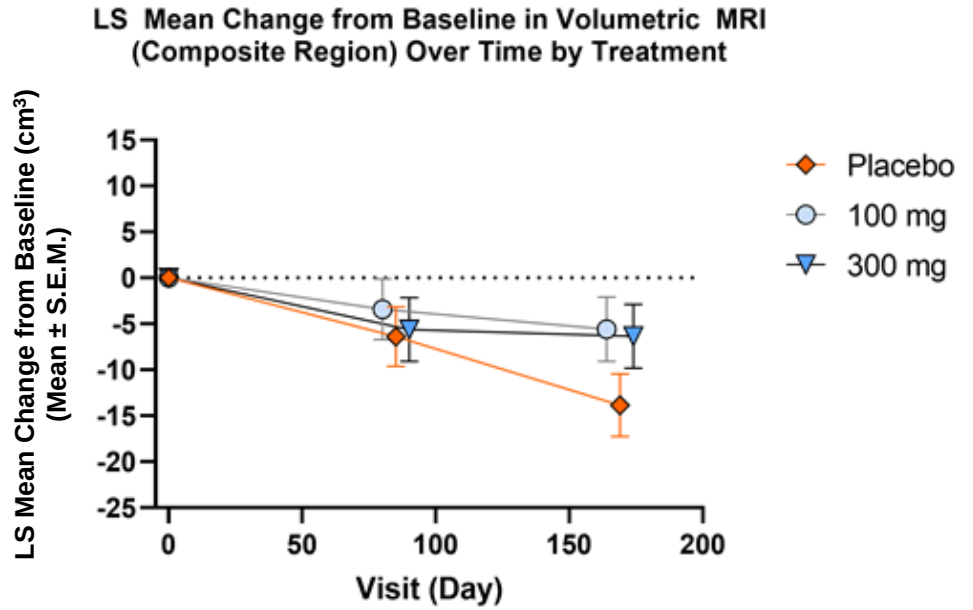
First longitudinal study of [^{11}C]UCB-J tracer in AD patients

Study goals:

1. Primary objective: evaluate safety and tolerability of CT1812
2. Secondary objective: evaluate CT1812 via:
   - SV2A PET, FDG-PET, volumetric MRI and functional MRI
   - CSF biomarkers, cognitive and clinical endpoints

Results:

- Safety profile consistent with prior studies
- Trend towards preservation of brain volume (composite) in patients treated with CT1812 versus placebo (top right)
- Statistically significant ($p<0.05$) improvement in volume in three regions (bottom right)
- No significant treatment differences on other key endpoints including on the ADAS-Cog 11 and on SV2A signal change
- Analyses of biomarker and proteomic data underway; to be presented at 2022 medical meetings



MRI Brain Volume (cm³)
6-mo change from baseline

	CT1812 (Pooled)	Placebo	P-value vs 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

COGNITION™ Therapeutics

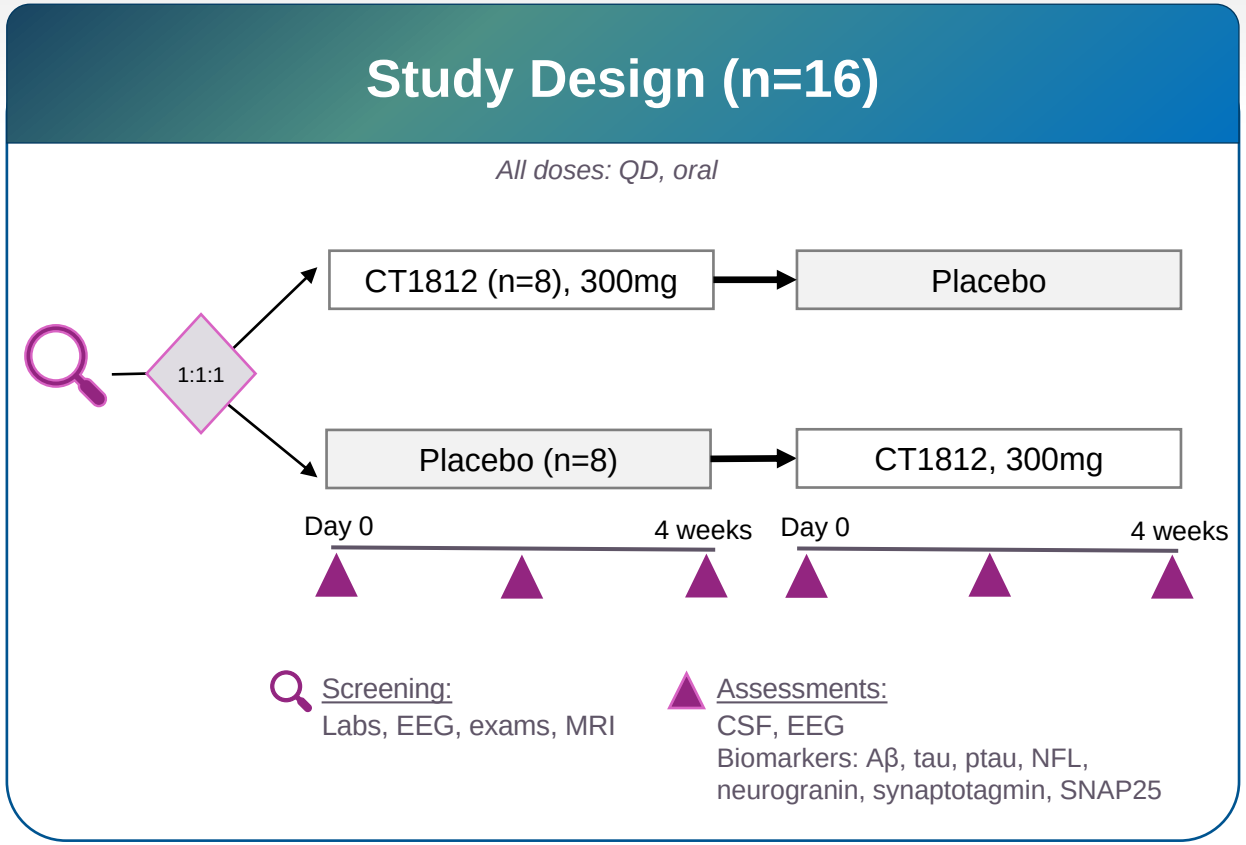
SEQUEL (COG0202)

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 AG058710

COGNITION™ Therapeutics

Expanded Patient Population: Early AD

540-patient trial of CT1812 fully funded by $81M grant

- COG0203: Phase 2 efficacy study, powered to show change in cognition: slowing or halting cognitive decline
- Enrollment: 540 patients with early AD
- Initiate in 2022
- Collaboration with premier NIA-funded Alzheimer's clinical trial group
- ~35 leading academic U.S. sites

ACTC ALZHEIMER'S CLINICAL TRIALS CONSORTIUM

NIH NIA

COG0203 Study funded by NIA grant R01AG065248

Cognition Pipeline:

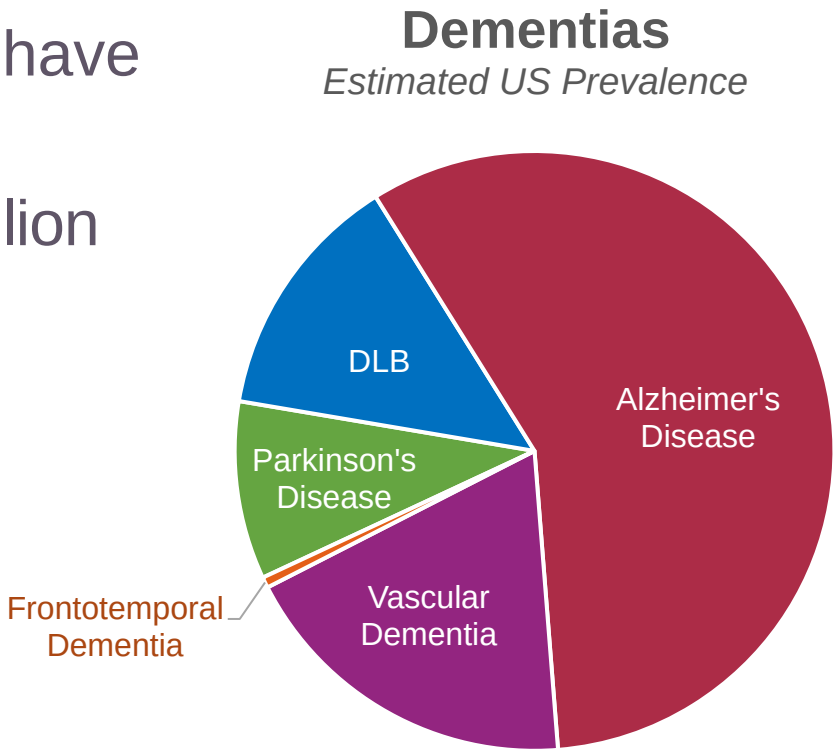
Synucleinopathies

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)*

COGNITION™ Therapeutics

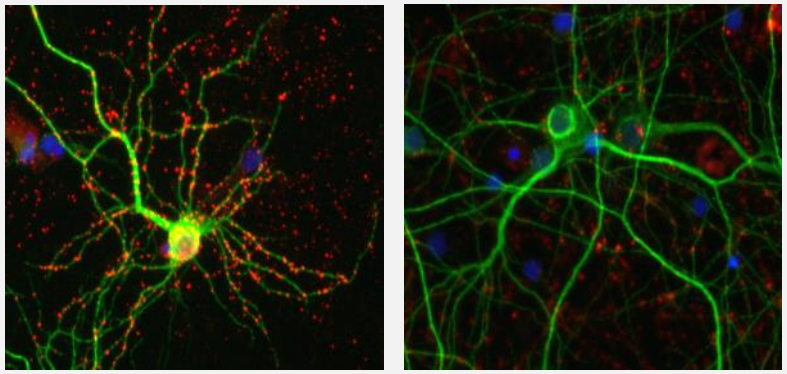
σ-2 Modulators May be Disease Modifying in Synucleinopathies

Cellular evidence that σ-2 modulators have a beneficial impact

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

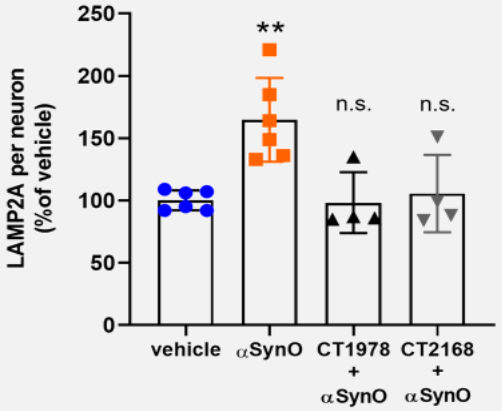
α-synuclein oligomers

α-synuclein oligomers + CT1812

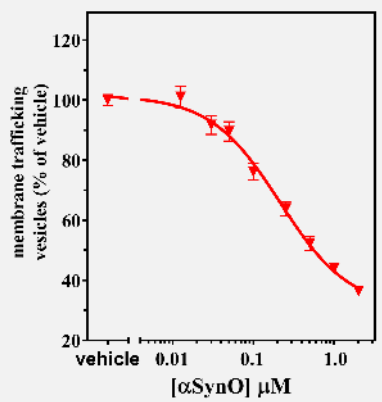


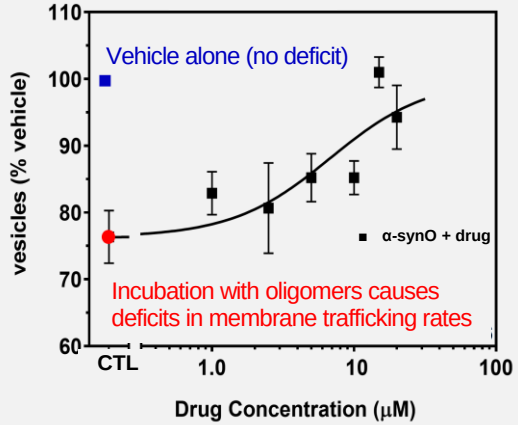
α-Synuclein oligomers in red

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

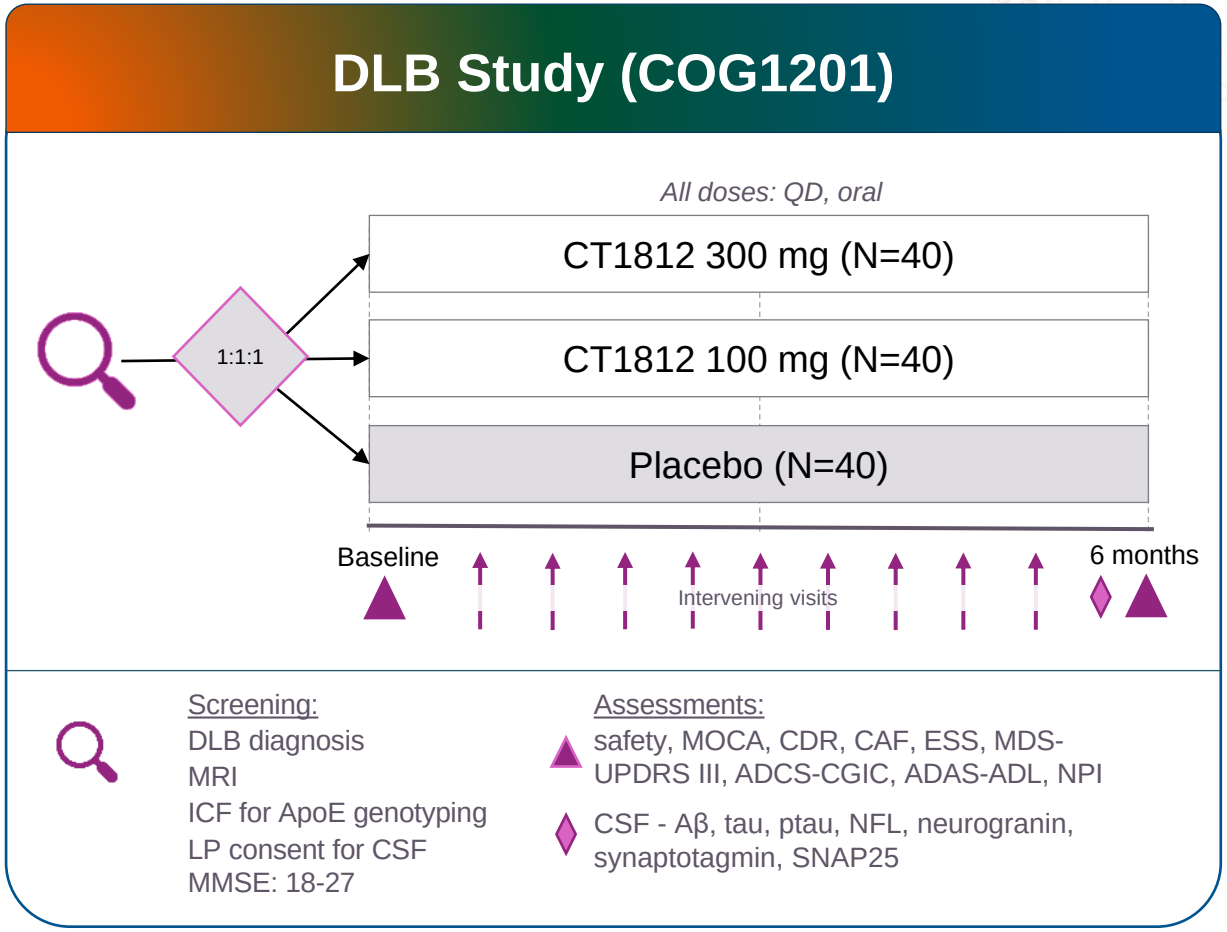
COGNITION™ Therapeutics

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

- Initiate Phase 2 in 1H 2022
- U Miami and 20+ academic sites
- DLB R&D educational symposium: https://ir.cogrx.com



COG1201 study funded by NIA grant R01AG071643

COGNITION™ Therapeutics

MJFF Grant Award Supports Further Study in Parkinson's Disease Models

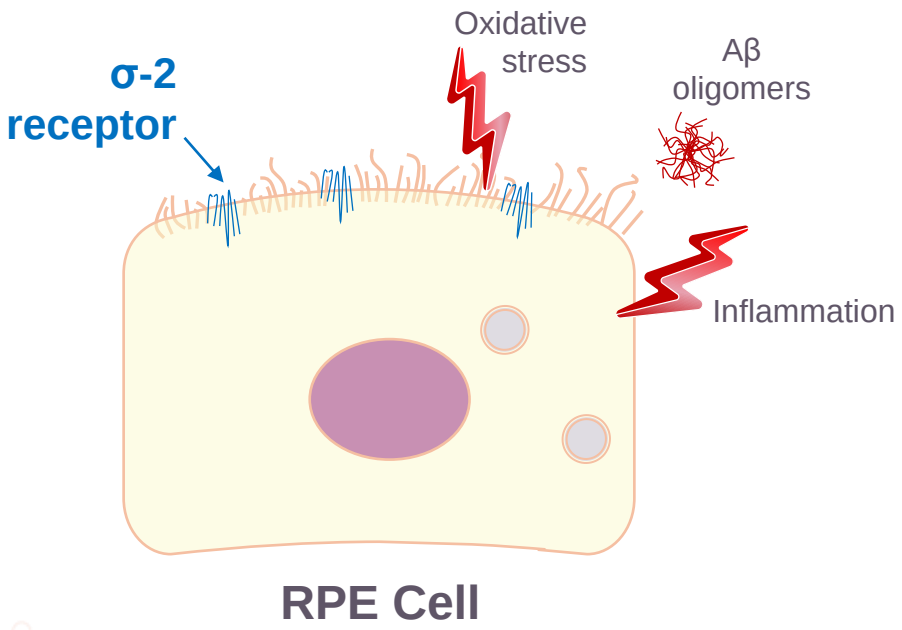
- Jan 2022 cognition awarded a Therapeutic Pipeline Program Grant from The Michael J. Fox Foundation for Parkinson's Research (MJFF)
- Will fund preclinical studies of two chemically distinct σ-2 receptor modulators in animal models of Parkinson's disease
- Successful completion will inform selection of a clinical candidate and dose(s)

Cognition Pipeline:

Dry Age-related Macular Degeneration

COGNITION™ Therapeutics

Role of σ-2 Receptor Complex in Dry AMD



- **Expression:** In retinal pigment epithelial (RPE) cells[1], retinal ganglion cells[1], photoreceptors in retina[2]
- **Biology**: Regulates autophagy, protein trafficking, lipid metabolism[3], which are dysregulated in dry AMD[4]
- **Target validation:** TMEM97 knockdown has been shown to be protective[1]
- **Human genetics:** Link to dry AMD[5]

1. Wang JH et al (2020) *BioRxiv*. doi: 10.1101/2020.07.10.198143
2. Shanmugam AK et al. (2016). *Current Eye Research*, doi: 10.3109/02713683.2015.1085579
3. Izzo, et al. (2020). *eNeuro*. doi: 10.1523/ENEURO.0317-20.2020
4. Keeling E et al (2019). *Mol. Nutr. Food Res*. doi: 10.1002/mnfr.201800951
5. Yan Q et al. Human Molecular Genetics, 2018. doi: 10.1093/hmg/ddy002

COGNITION™ Therapeutics

Human Genetics Points to a Role for TMEM97 in Dry AMD

GWAS identified a SNP in TMEM97-VTN locus that confers decreased risk of dry AMD

Locus identified in several independent and large-scale GWAS

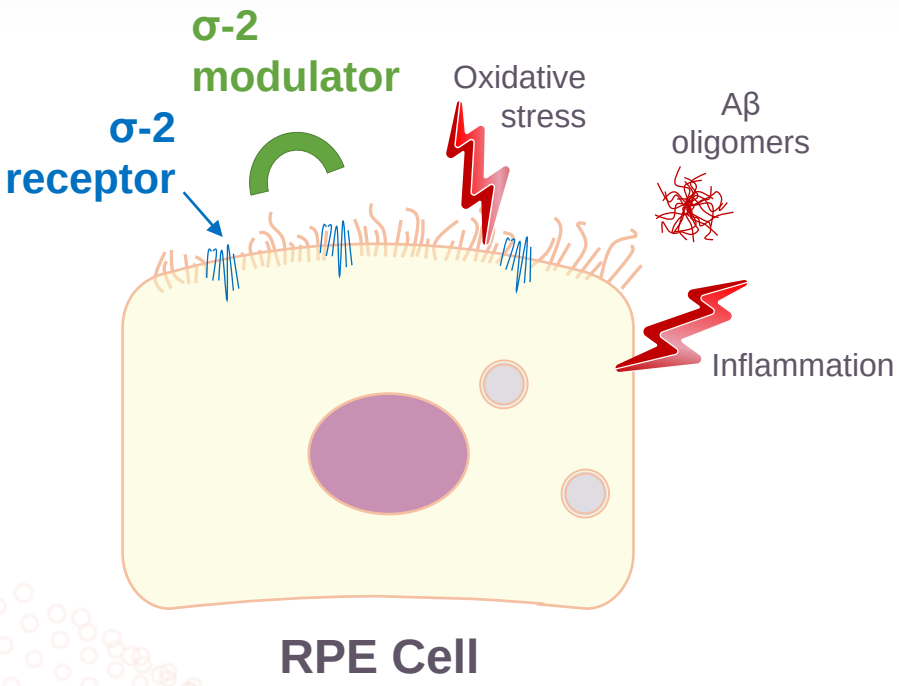
Table 4. Results for 34 AMD risk variants reported in the latest case–control study conducted by the International AMD Genomics Consortium

SNP	Chr	Position	Major/ minor allele	MAF	Gene	With BL severity		Without BL severity		Fritsche *et al.* case–control results	
						HR	*P*-value	HR	*P*-value	OR	*P*-value
Known SNPs identified in consortium case–control studies											
rs11080055	17	26 649 724	C/A	0.48	*TMEM97-VTN*	0.89	0.0267	0.97	0.5847	0.91	1.0×10^{-8}
rs6565597	17	79 526 821	C/T	0.38	*NPLOC4-TSPAN10*	1.03	0.5769	1.06	0.2877	1.13	1.5×10^{-11}
rs67538026	19	1 031 438	C/T	0.45	*CNN2*	0.90	0.0475	0.90	0.0684	0.9	2.6×10^{-8}
rs142450006	20	44 614 991	TTTTC/T	0.13	*MMP9*	0.77	0.0021	0.77	0.0029	0.85	2.4×10^{-10}
rs201459901	20	56 653 724	T/TA	0.06	*C20orf85*	1.07	0.4688	0.96	0.7440	0.76	3.1×10^{-16}

Yan Q et al. *Human Molecular Genetics*, 2018. doi: 10.1093/hmg/ddy002

Clinical and Preclinical Data Support σ-2 Modulators as a Promising Therapeutic Approach for Dry AMD

Goal: protect RPE cells from disease-relevant stressors



- **Clinical support:** Proteomics analyses showed differential movement of proteins involved in dry AMD
- **Preclinical data:** Regulates inflammatory response, ameliorates trafficking deficits and prevents cell death

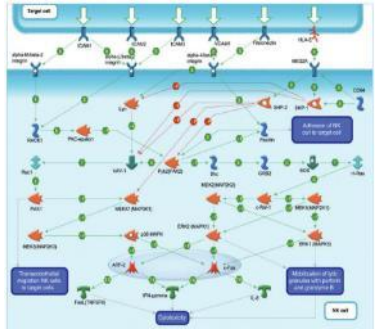
Unbiased Pathway Analysis of Patient Clinical Trial Proteomics Data Implicates CT1812 in Dry AMD

Proteomics datasets from two Phase 2 clinical trials

1. COG0102 – Ph2 Clinical Trial of σ-2 Receptor Modulator, CT1812 (90, 280, 560 mg) or Placebo in Mild-to-Moderate AD Patients
Proteomics analysis of CSF at 28 day (N=15)

2. SHINE-A – Ph2 Clinical Trial of σ-2 Receptor Modulator, CT1812 (100, 300 mg) or Placebo in Mild-to-Moderate AD Patients
Proteomics Analysis of CSF at 6 mo (N=18)

1. Generated list of proteins differentially expressed in CSF from CT1812 vs. placebo-treated patients
2. Performed Metacore pathway analysis of CSF (CT1812 vs placebo) across trials to ascertain which predesignated functional disease ontologies may be significantly affected



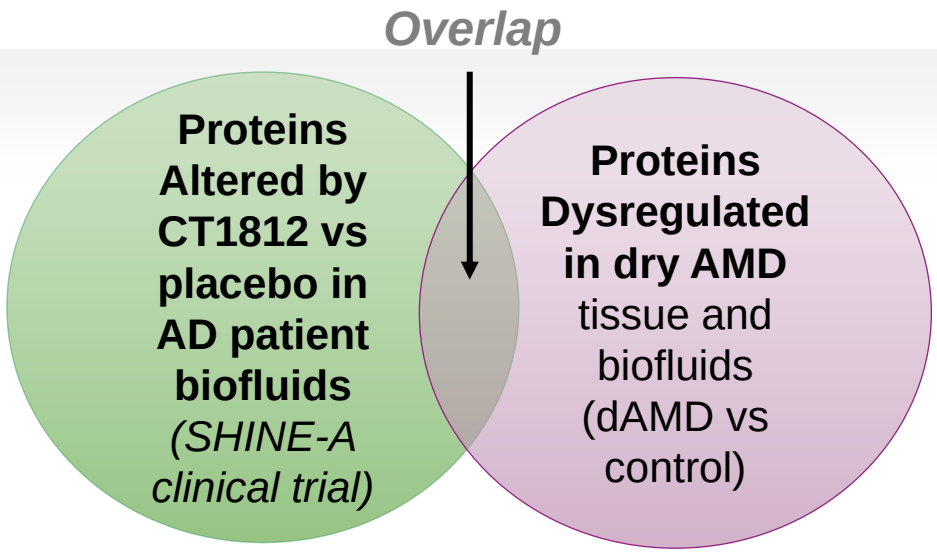
MetaCore+MetaDrug™
Version 20.3 build 70200

Top Disease Ontologies

1. Geographic atrophy
2. Central nervous system diseases
3. Cognition disorders
4. Mental disorders
5. Psychiatry and psychology
6. Macular degeneration
7. Neurocognitive disorders
8. Rett syndrome
9. Dementia
10. Movement disorders
11. Neurodegenerative diseases
12. Brain diseases
13. Basal ganglia diseases
14. Anemia
15. Infections

Analysis of Alzheimer's Disease Patient Clinical Trial Data

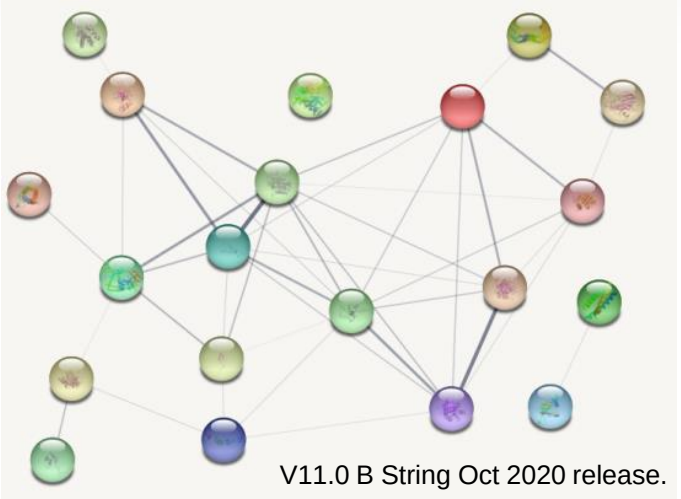
Identification of proteins & pathways in AD patient proteomes affected by CT1812 that are linked to dry AMD



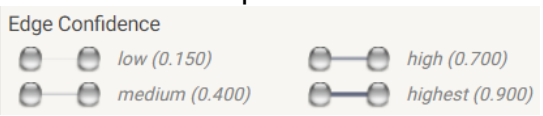
Analysis included proteomics datasets from both plasma (1 & 6mo) and CSF (6 mo)

Data sources: Lynch et al.,2019, Yuan et al., 2010, Lambert et al. 2016

STRING Protein Interaction Network



PPI enrichment p-value: 9.2e-09



Enabled the identification of a subset of proteins and pathways genetically linked to or known to be dysregulated in dry AMD that were regulated in AD patients biofluids given CT1812 vs placebo…

Provides preliminary proof of principle that the σ-2 modulator CT1812 may be capable of altering AMD-relevant proteins and pathways in an aged population

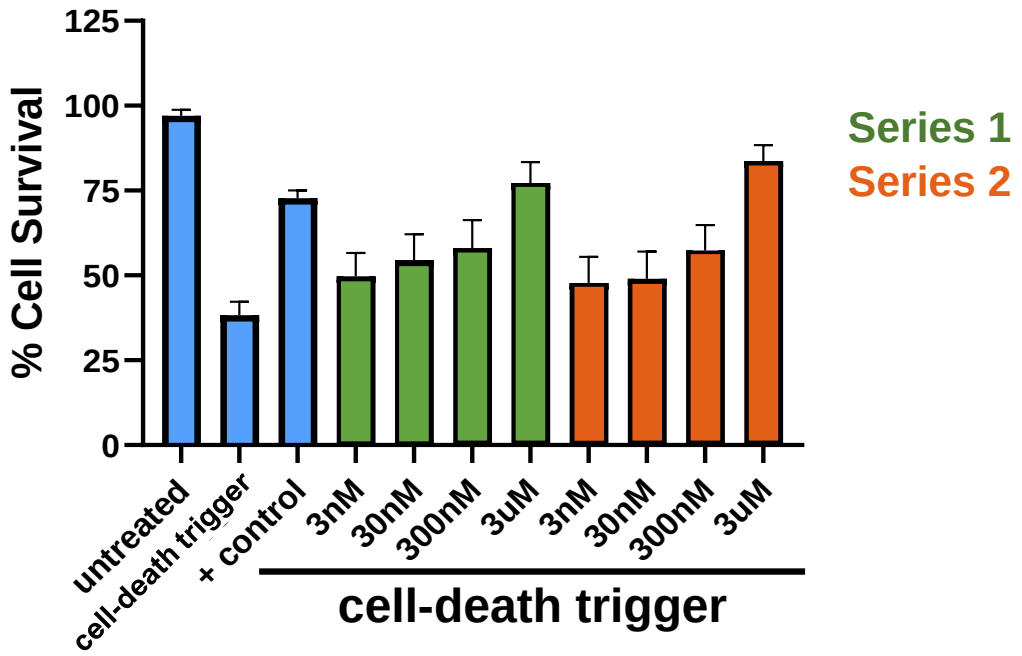
COGNITION™ Therapeutics

Data Indicate σ-2 Modulators Rescue RPE Deficits and May be Protective in Dry AMD

Cell Survival and Inflammatory Pathways are Altered by CT1812 vs Vehicle (p<0.05)

Oxidative stress
Apoptosis and survival APRIL and BAFF signaling
Signaling Transduction Role of MIF as an intracellular mediator
Cell cycle Role of SCF complex in cell cycle regulation
Immune response IFN-alpha/beta signaling via JAK/STAT
Development PEDF signaling
Aβ Oligomer
Immune response BAFF-induced non-canonical NF-kB signaling
Immune response Role of PKR in stress-induced antiviral cell response
Apoptosis and survival APRIL and BAFF signaling
Apoptosis and survival NGF activation of NF-kB
Apoptosis and survival Apoptotic TNF-family pathways
Inflammation
Cytoskeleton remodeling Regulation of actin cytoskeleton nucleation and polymerization by Rho GTPases
Neurophysiological process Activity-dependent synaptic AMPA receptor removal
Cell adhesion Classical cadherin-mediated cell adhesion
Transcription Ligand-dependent activation of the ESR1/SP pathway
Immune response Lysophosphatidic acid signaling via NF-kB

Data Show σ-2 Modulators Prevent Cell Death in Concentration-dependent Manner

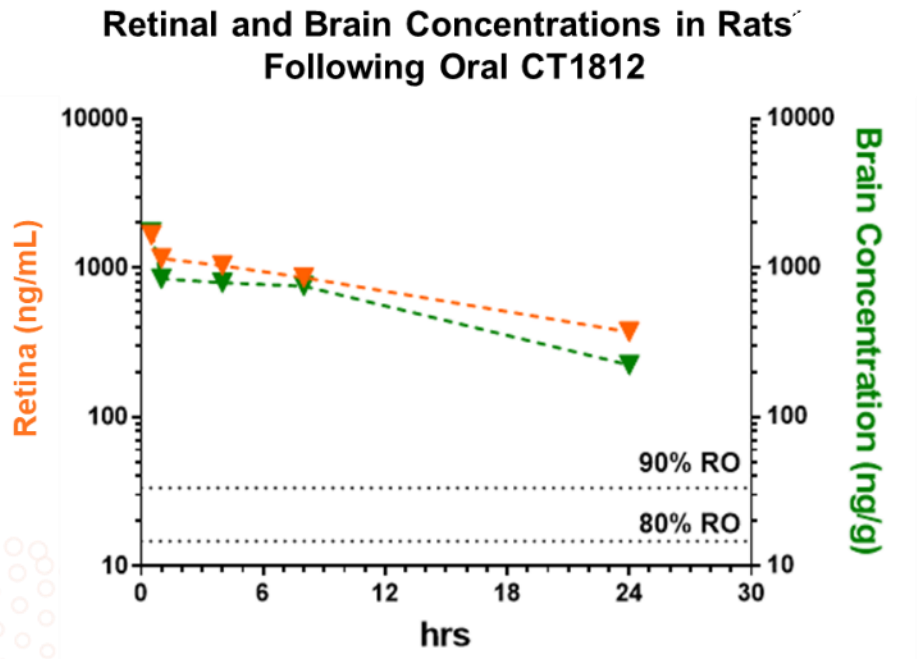


Additional mechanistic and PoC data to be shown at the ARVO conference May 2022

COGNITION™ Therapeutics

In Vivo Pharmacokinetics Support Phase 2 for Dry AMD

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



Planned Phase 2* to enroll patients with diagnosed dry AMD and measurable GA

Screening:

- Age: ≥ 50
- Diagnosis of dry AMD
- BCVA ≥ 24 letters (ETDRS)
- GA lesion ≥ 2.5 and ≤17.5mm^2

Assessments:

- *Primary endpoints*:
  - Change in GA lesion using fundus autofluorescence (FAF)
- *Secondary endpoints*:
  - Low luminance visual acuity (LLVA)
  - Best-corrected visual acuity (BCVA)
  - Optical coherence tomography (OCT)

* Subject to discussion with FDA

Financial Position

Capital Markets

• IPO priced Oct 8	$45.2 million
• Overallotment exercised Nov 15	$6.8 million
- *Total IPO proceeds (gross)*	**$52.0 million**

NIA funding for CT1812 studies

• Preclinical through Phase 2 studies	$168.4 million
• Approximate funding used	($58.0 million)
- *Available NIA funding*	**$110.4 million**

COGNITION™ Therapeutics

Thank You

Lisa Ricciardi
President & CEO
917-658-5789
lricciardi@cogrx.com

James O'Brien
Chief Financial Officer
203-536-1797
jobrien@cogrx.com

COGNITION™ Therapeutics