COGNITION™ Therapeutics

Targeting Amyloid Beta Oligomers:

A Disruptive Approach to the Treatment of Alzheimer's Disease

August 2024

Forward-looking Statements

FORWARD-LOOKING STATEMENTS

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Overview

THE CHALLENGE IN ALZHEIMER'S

- Only **2** approved disease-modifying therapies
- 35+ years of research mainly focused on beta amyloid (Aβ) & plaques
- Major unmet need remains
- Rapidly growing population of people with Alzheimer's disease

THE OPPORTUNITY

Strong scientific evidence supports the Amyloid Oligomer Hypothesis:

Oligomers — not plaques — are the **MOST NEUROTOXIC** form of Aβ in AD

Oligomers of both α-synuclein & Aβ drive DLB

OUR SOLUTION

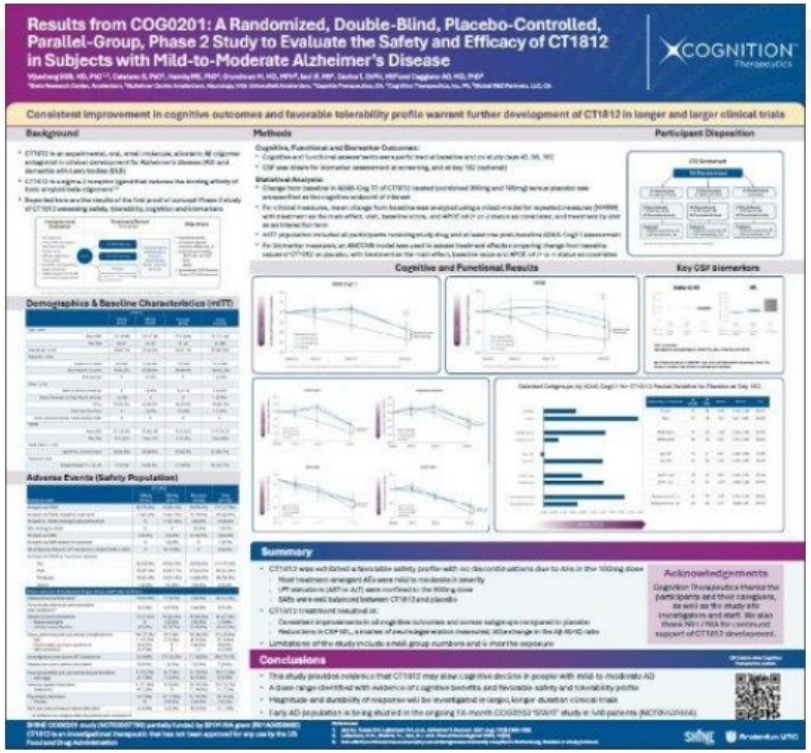
CT1812: An orally delivered small molecule that potently antagonizes amyloid oligomers via a unique mechanism of action

- ✓ **PHASE 2 PoC** data demonstrated ~40% mean improvement in cognitive measures vs placebo in mild-moderate AD
- ✓ **PHASE 2 PoC** in early-mild AD currently enrolling with partners at ACTC
- ✓ In clinical trials for two additional indications
- ✓ Clinical studies to date show favorable tolerability

Breaking News – SHINE Proof-of-Concept Phase 2 Study

CT1812 is Among Few Oral Candidates Showing Cognitive Impact in *Moderate* Patients

- Pooled (100 and 300mg arms) CT1812 treatment **slowed cognitive decline by 39%** on ADAS-Cog 11 vs placebo
- All key cognitive and functional outcome measures trending in favor of CT1812
- Efficacious dose with good safety profile
- Well-designed and executed study
- Supports advancing clinical development

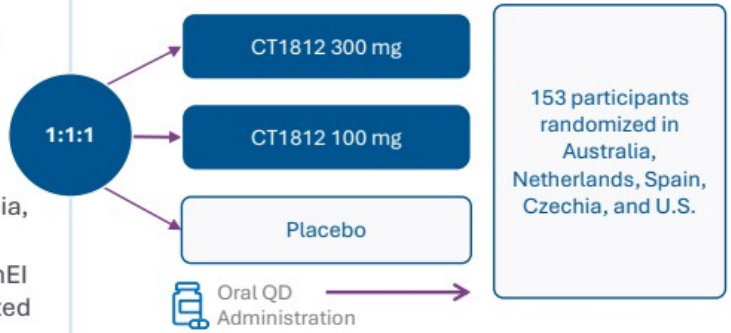


SHINE Phase 2 Safety and Efficacy Study in Adults with Mild-to-Moderate Alzheimer's Disease

Inclusion and Exclusion

- AD diagnosis
- PET or CSF biomarker definition of AD
- MMSE 18-26
- MRI w/o significant abnormality
- No MDD, schizophrenia, bipolar disorder
- Stable regimen of AChEI or memantine permitted

Treatment Period
6 months



Objectives

- Safety/tolerability
- Principal cognitive measure: ADAS-Cog 11
- Exploratory including:
  - ADAS-Cog 13
  - ADCS-ADL & CGIC
  - NTB
  - MMSE
- Biomarkers (CSF/Plasma)
- Plasma CT1812 exposure

SHINE COG0201 study (NCT03507790) partially funded by $31M NIA grant R01AG058660

SHINE COG0201

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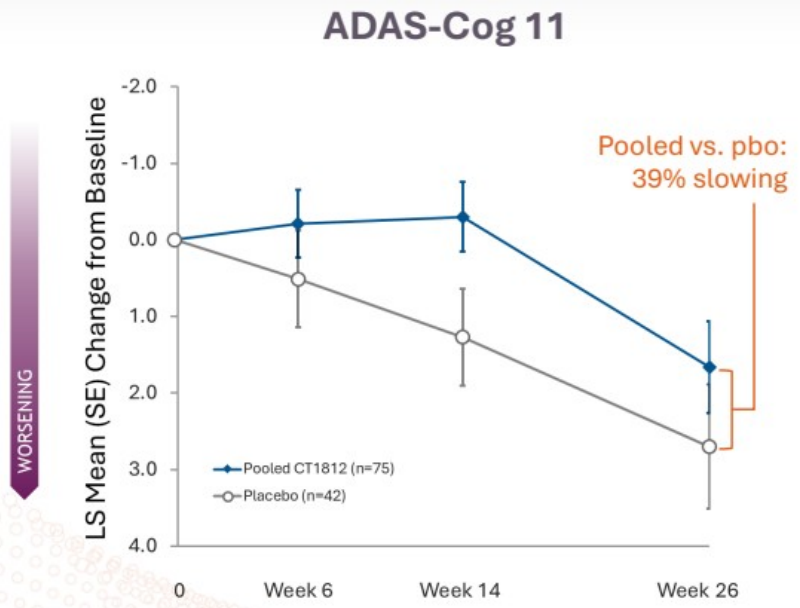
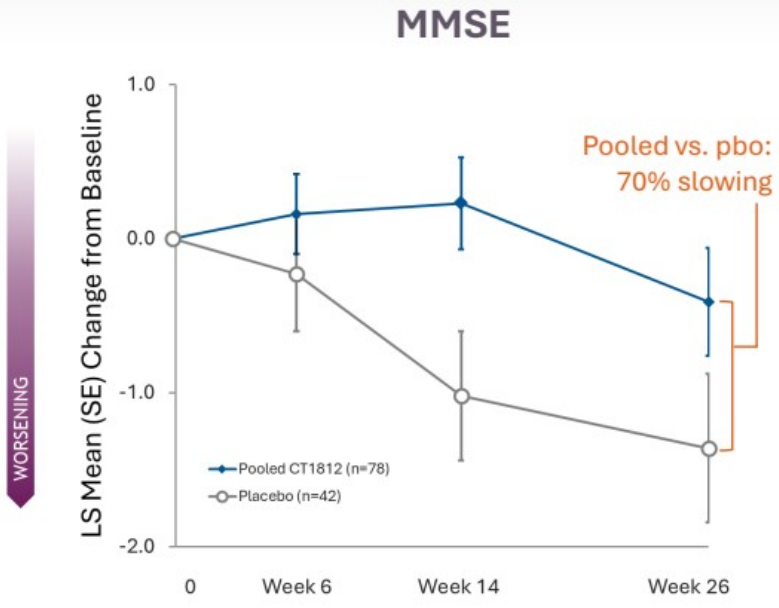
SHINE Patient Population

- PET- or biomarker-confirmed Alzheimer's disease
- Majority of participants were female (60%), Caucasian (96%), approximately 72 years of age
- Mean MMSE score upon entry: 21.37
- ~60% of patients were carriers of the ApoE4 gene
- Characteristics well-balanced between all 3 arms

SHINE Cognitive Endpoints: ADAS-Cog 11 and MMSE

Magnitude of ADAS-Cog 11 decline at 6 months similar to approved MAbs





Note: n's are at Day 182

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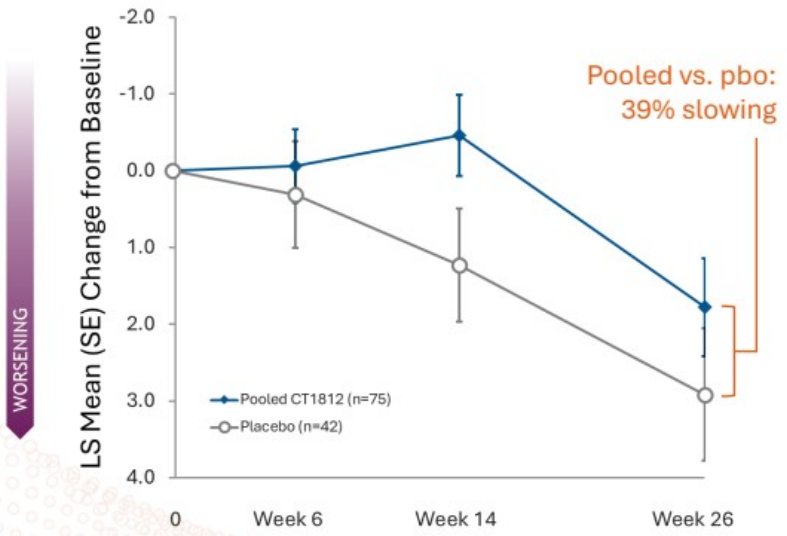
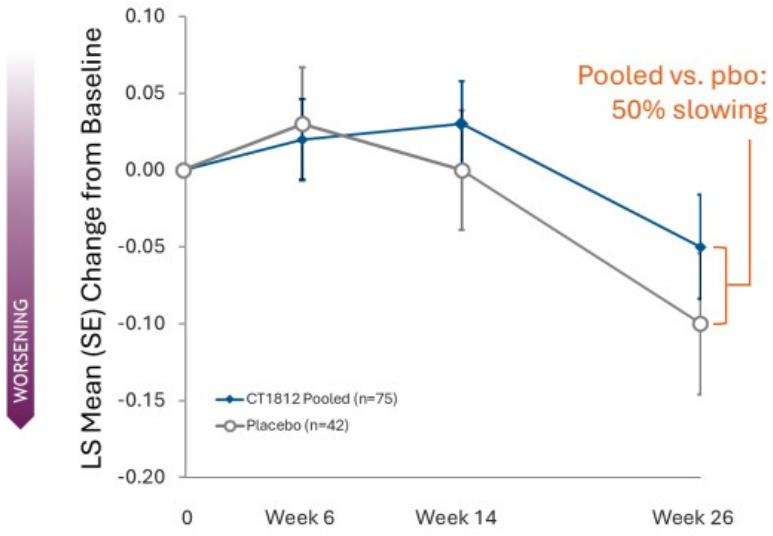
SHINE Cognitive Endpoints: ADAS-Cog 13, Cognitive Composite

Consistent results across multiple cognitive endpoints

ADAS-Cog 13



Cognitive Composite



Note: n's are at Day 182

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Summary of SHINE Safety and Tolerability findings

- CT1812 demonstrated a favorable safety and tolerability profile
- Most TEAEs were mild or moderate in severity
- Similar percentages of adverse events in treated (76.5%) and placebo (78%) groups
- No discontinuations due to AEs in the 100mg dose group
- Most discontinuations were in 300mg dose group and all the reportable liver enzyme elevations were in 300mg dose group

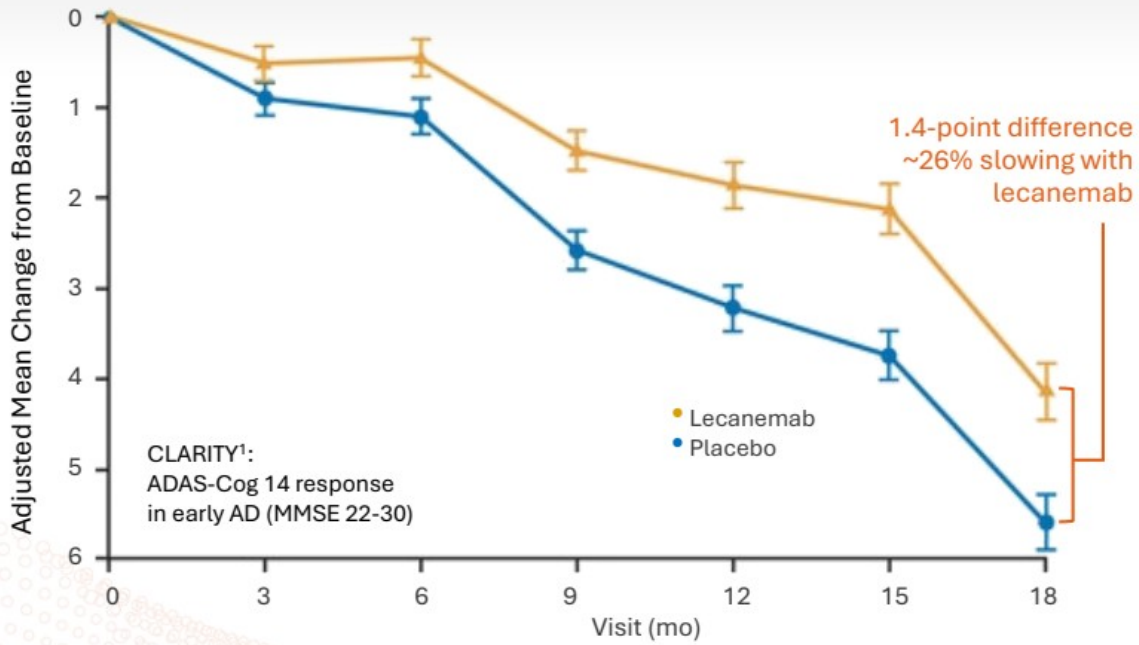
Adverse Events	
CT1812	Placebo
76.5%	78%

Serious AEs	
CT1812	Placebo
4.9%	10%

Deaths	
CT1812	Placebo
0	1 (cancer)

SHINE Response at 6 months Comparable to Approved MAbs

Once-daily pill • no ARIA • 39% slowing at 6 months vs Leqembi's 26% at 18 months



SHINE Results
1.1 pt difference at 6 months
39% slowing pooled CT1812 vs. placebo
Placebo decline in SHINE steeper w moderate AD

1. van Dyck C et al. Lecanemab in Early Alzheimer's Disease (2023) *NEJM* 388:9-21

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SHINE: Summary Exploratory Outcomes - Percent Slowing Day 182

Effect as large or larger than approved MAbs

	ADAS-Cog 11	ADAS-Cog 13	MMSE	Cognitive Composite
CT1812 Pooled[1]	39%	39%	70%	50%
Lecanemab[2] (at 18mo)	--	26% (ADAS-Cog 14)	--	24% (ADCOMS)
Donanemab[3] (at 18 mo)	--	20% (ADAS-Cog 13)	16% (MMSE)	22% (iADRS)

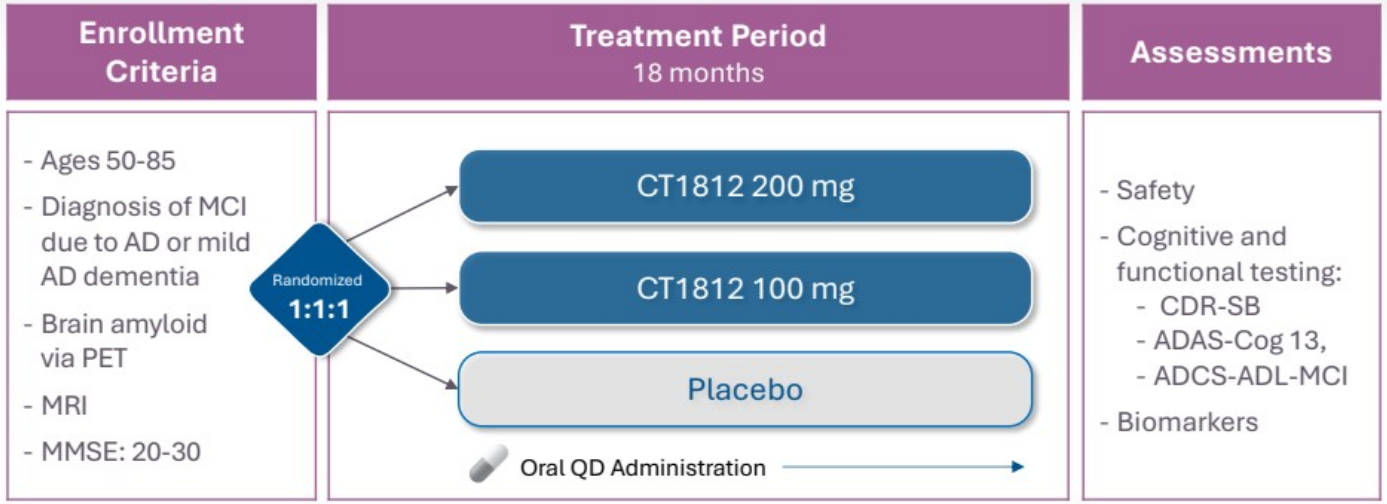
Note: data shown for benchmarking only; no head-to-head studies have been conducted

1. Percentages reflect mean changes from baseline compared to placebo
2. van Dyck C et al. Lecanemab in Early Alzheimer's Disease (2023) *NEJM* 388:9-21
3. Sims JR et al. Donanemab in Early Symptomatic Alzheimer Disease (2023) JAMA. 2023;330(6):512-527

START: Proof-of-Concept 540-Person Study in Early AD

Funded through $81M NIA Grant

- N = 540 with early Alzheimer's
- Supported by $81 M NIA grant awards (R01AG065248)
- First study to allow lecanemab as background therapy in combination with CT1812
- Conducted in collaboration with ACTC (U24AG057437)
- START COG0203 study (NCT05531656)



START COG0203

MMSE, Mini-Mental State Examination; MRI, magnetic resonance imaging; QD, daily; NIA, National Institute on Aging; ACTC, Alzheimer's Clinical Trials Consortium

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CT1812 is Also Being Investigated in two Additional Indications

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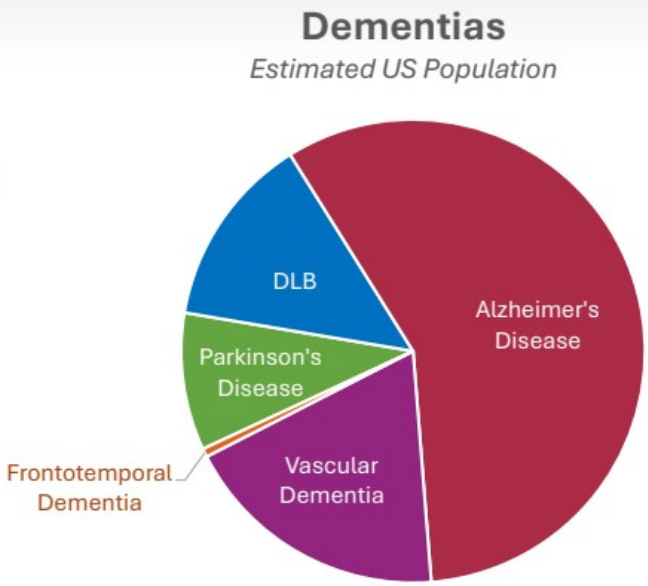
α-Syn Dementias are Second only to AD in Prevalence

- In the U.S. an estimated 1.4 million people[1] have dementia with Lewy bodies (DLB)
- It is estimated that 50-80% of patients with DLB have Aβ as well as α-synuclein[2] pathology
- Core symptoms of DLB include:
  - Progressive cognitive decline
  - Fluctuating cognition with variations in attention
  - Impaired visuospatial perception
  - Recurrent visual hallucinations
  - REM sleep disorder

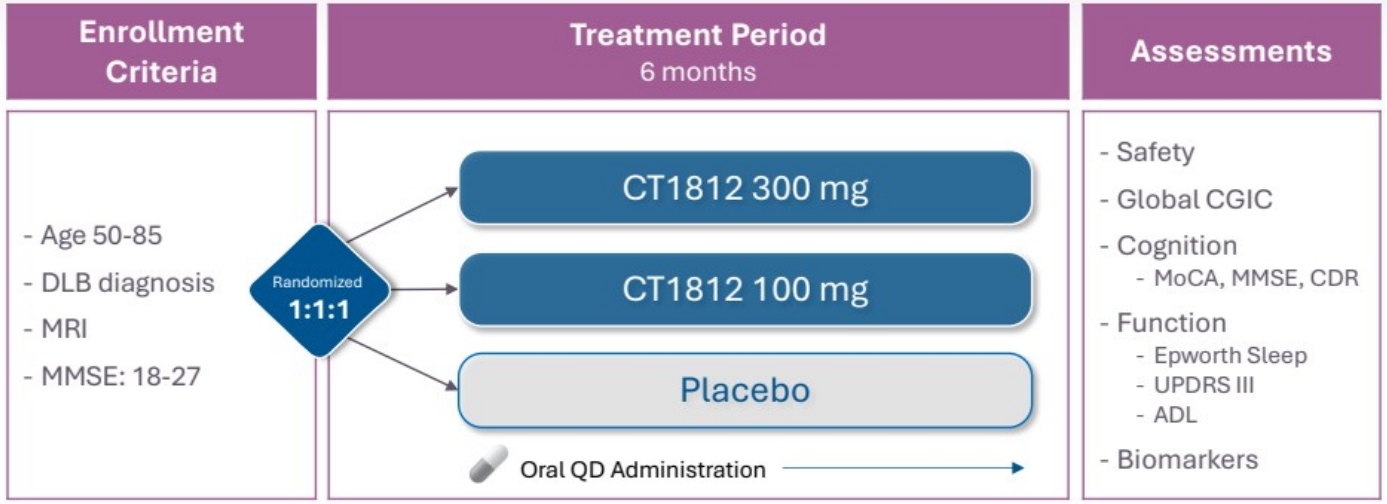


1) Lewy Body Dementia Association. About LBD. Available from: https://www.lbda.org/about-lbd
2) Int J Mol Sci. 2023 Jun; 24(12): 10215. doi: 10.3390/ijms241210215

SHIMMER: Proof-of-Concept Trial Investigating CT1812 in DLB

Sponsored by $30 M NIA grant

- Randomization to oral CT1812 (100 or 300mg) or placebo
- Enrolled 130 adults with mild-to-moderate DLB
- Not powered for statistical significance
- **Topline results YE 2024**
- Collaboration with LBDA and University of Miami



DLB, Dementia with Lewy Bodies; MMSE, Mini-Mental State Examination; MRI, magnetic resonance imaging; QD, daily; NIA, National Institute on Aging; LBD, Lewy Body Dementia; EEG, electroencephalogram

SHIMMER COG1201

Rationale for CT1812 in Dry AMD/Geographic Atrophy

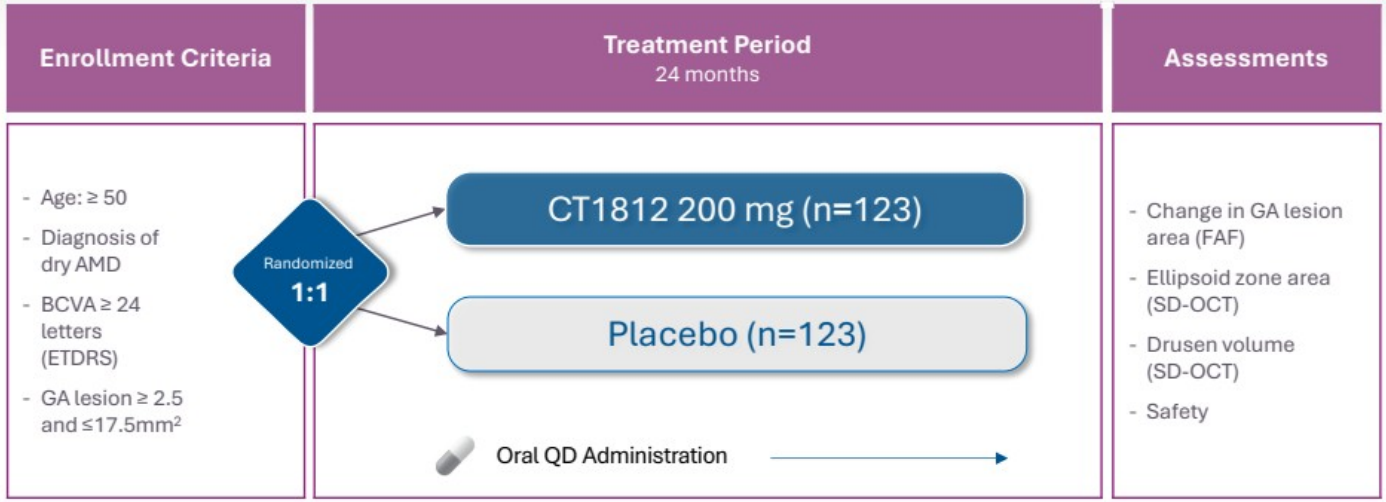
Opportunity: crosses blood-retinal barrier to reach retina without an injection

What is dAMD/GA	Geographic atrophy (GA), the most advanced form of dry AMD, effects ~5M people WW and is associated with significant vision loss
Unmet Need	Current standard of care is primarily drug injections directly into eye(s)
Pathophysiology	Death of retinal pigment epithelium (RPE) cells due to failure to clear degradation products (poor trafficking) Sigma-2 receptor is involved in multiple processes underlying RPE death
MOA of CT1812	Targeting of sigma-2 receptors rescues trafficking function in RPE cells

MAGNIFY: Phase 2 Proof-of-Concept Trial in dAMD/GA

Unique MoA targeting retinal pigment epithelium

- Randomization to placebo or daily oral doses of CT1812
- N = 246 adults with GA secondary to dry AMD
- Potential first oral for GA



Magnify

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Completed Studies Support Potential in Mild-to-Moderate AD

Ongoing Trials Expand CT1812 into New Indications

COG0203 - START

Early-to-mild Alzheimer's disease
Actively recruiting

COG1201 - SHIMMER

Mild-to-moderate DLB
Topline data YE2024

COG2201 - MAGNIFY

GA secondary to dry AMD
Actively recruiting

SHINE

- 153 participants
- Consistent slowing cognitive decline (ADAS-Cog 11 and 13, MMSE, Cog Composite)
- Trends in functional benefit

SEQUEL[1]

- 16 participants
- Normalization of brain waves across EEG measures
- Significant improvement in AEC-c and relative theta in central region

SPARC[2]

- 23 participants
- Preservation of brain atrophy via volumetric MRI
- No change in SV2A treated or pbo

SNAP[3]

- 3 participants
- Rapid displacement of Aβ oligomers via CSF
- Replication of preclinical findings via MEI

1. Vijverberg E et al. J Prev Alzheimers Dis (2024)
2. van Dyck, CH et al. Alz Res Therapy 16, 20 (2024)
3. LaBarbera, KM et al. Transl Neurodegener 2023, 12(24)

The Promise of CT1812

- **First-in-class** Aβ oligomer antagonism via sigma-2 receptor
- **Consistent efficacy** in Alzheimer's disease studies
  - ARIA unlikely to occur based on MoA
- **Potential first-to-market for DLB**
- **Potential first oral for dAMD/GA**
- **Well tolerated safety** profile anticipated
- **Oral** administration
  - No need for IV therapy, a key limitation of immunotherapeutics
  - No surveillance imaging required
  - Greater convenience and access

Current Financial Position

As of June 30, 2024

Cash and cash equivalents	$28.5 M
Expected cash runway into 2Q 2025	
Grant funding for CT1812 studies	
Preclinical through Phase 2	~$171 M
Approximate funding used	($113.7 M)
Remaining grant funding	$57.3M

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Thank You

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