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Athira
PHARMA
Corporate Presentation
APRIL 2022
ADVANCING NEW THERAPIES FOR NEURONAL HEALTH

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The ACT-AD trial is supported by a grant from the National Institute on Aging of the National Institutes of Health under Award Number R01AG06268. The information presented in this press release is solely the responsibility of Athira and does not necessarily represent the official views of the National Institutes of Health.

This presentation concerns drug candidates that are under clinical investigation and which have not yet been approved for marketing by the U.S. Food and Drug Administration. The drug candidates are currently limited by federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

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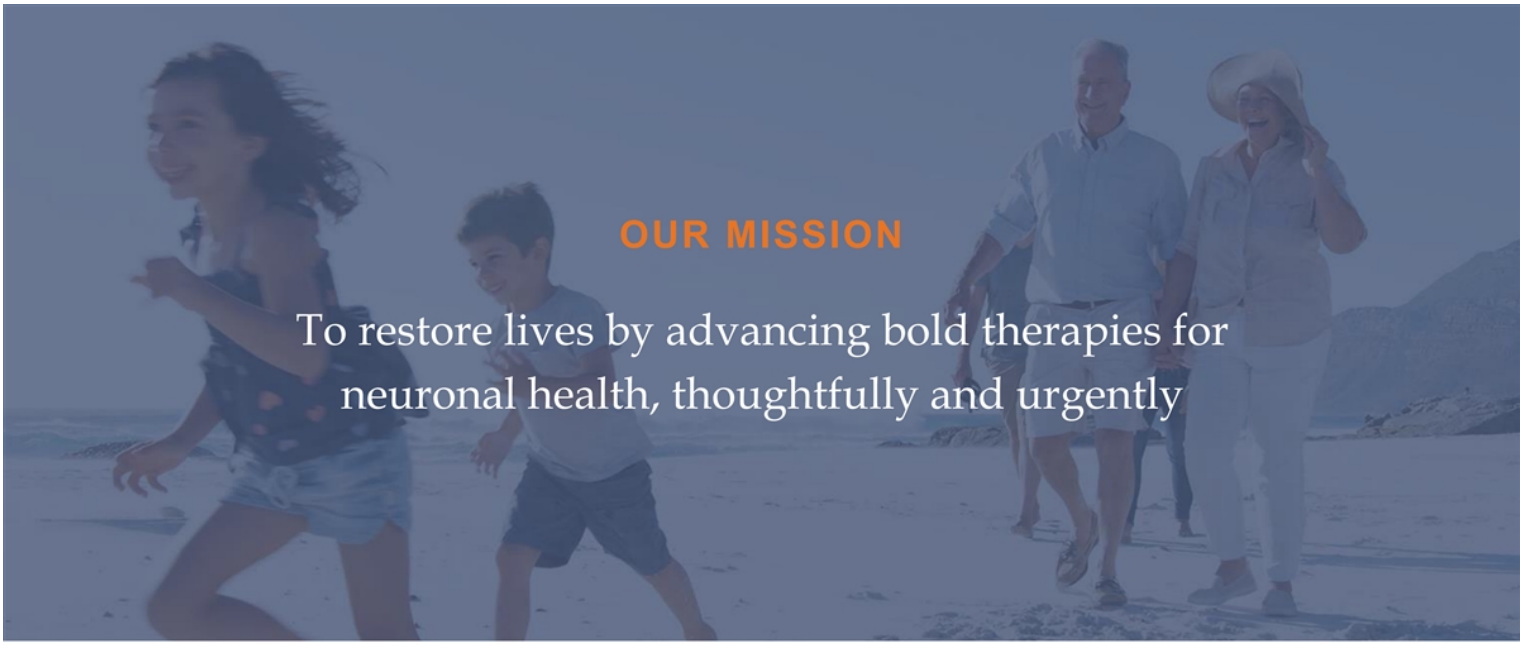

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Investment Highlights

Our novel small molecule compounds are designed to act on a naturally occurring mechanism to repair and restore neuronal health

Potentially pivotal program in Alzheimer's with a growing pipeline to address neurodegenerative and neuropsychiatric indications

Late-stage program fosgonimeton (ATH-1017) designed to enhance Hepatocyte Growth Factor (HGF) and its receptor, MET

- Well established HGF/MET pathway is critical to normal brain function and is compromised in Alzheimer's disease (AD) and other neurological diseases
- Data readout for Phase 2 ACT-AD clinical trial expected by end of 2Q22
- Phase 3 LIFT-AD clinical trial expected to complete enrollment in 3Q22 with data readout expected in 1H23
- Compelling Phase 1 data in AD demonstrated statistically significant improvement (p=0.027) of ERP P300 latency, an objective measure of working memory processing speed
- Cognitive improvement in Alzheimer's disease is a multi-billion dollar market opportunity

Strong balance sheet to support clinical programs through key inflection points

Leadership team with significant CNS product development and approval experience

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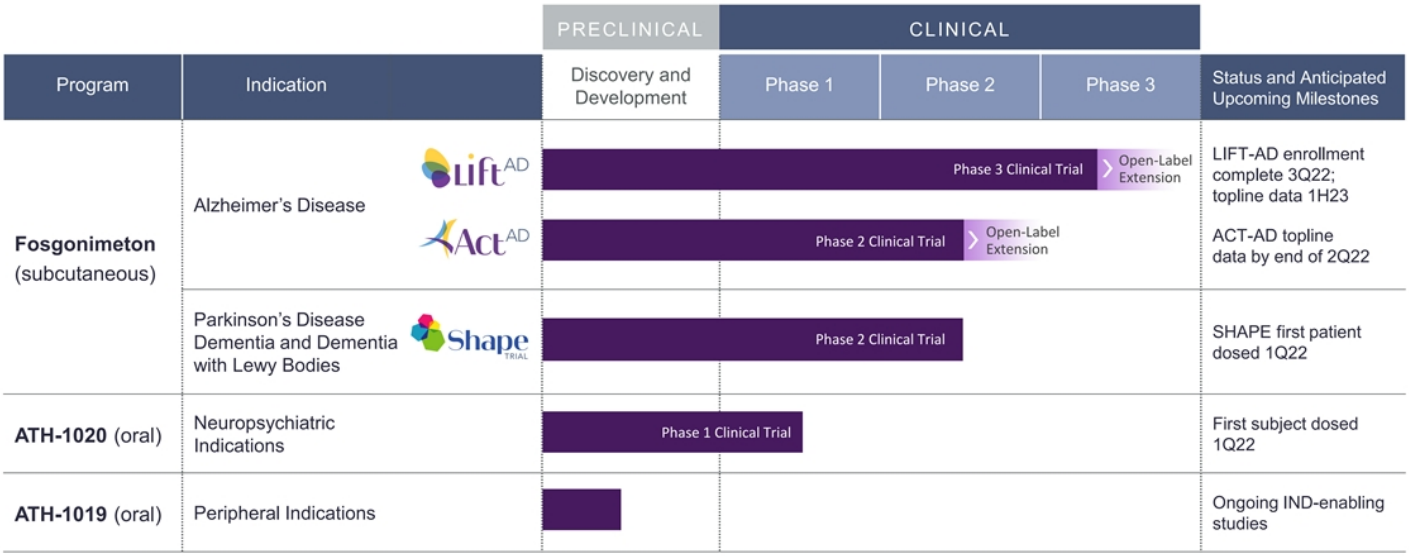
Therapeutic Potential Across a Broad Range of Clinical Applications



Lead Program:
FOSGONIMETON

Alzheimer's Disease (AD)

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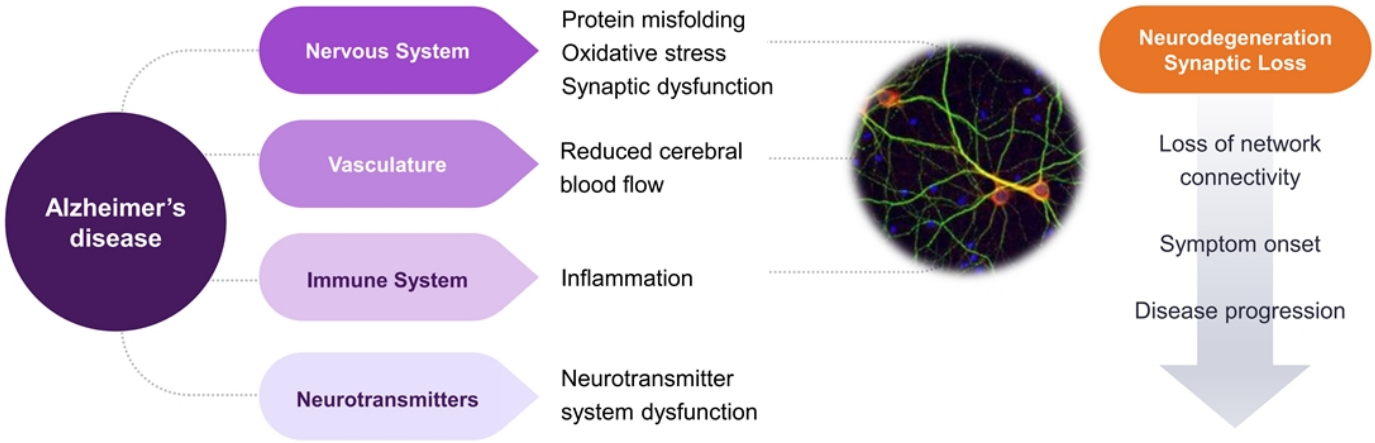
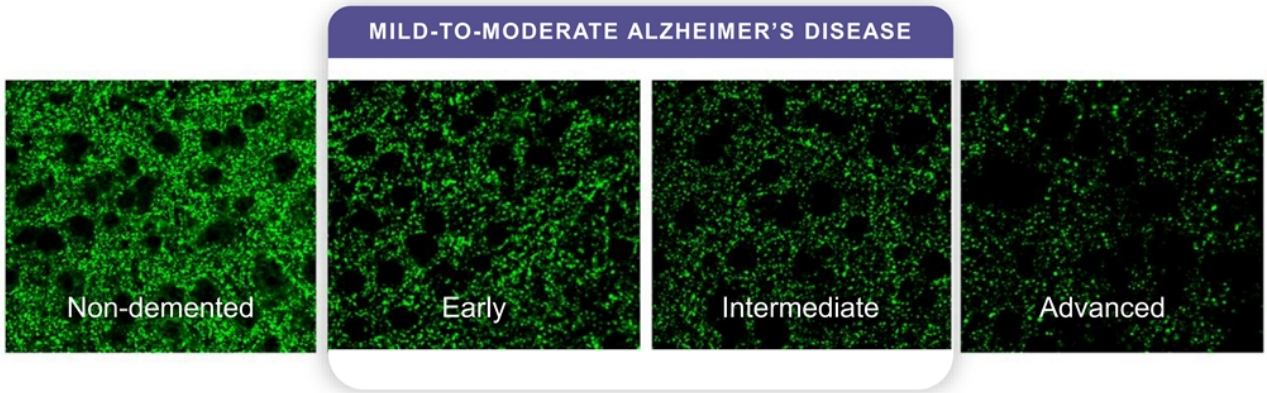
Alzheimer's Disease Pathology

MULTIFACTORIAL AND COMPLEX PATHOLOGIES ULTIMATELY LEAD TO NEURODEGENERATION



Synapse Loss in Alzheimer's Disease

- In AD, **25-36% of synapses are lost**[1]
- **Synapse loss is an early event in disease progression** that impacts several brain regions, including the hippocampus and frontal cortex, important for learning and memory[2]



[1] Davies et al, *J Neuroscience* 1987
[2] Masliah et al, *Neurosci Letters* 1989
Slide adapted from Olichney, as presented by Olichney, in Athira P300 KOL Webinar –November 2021

HGF/MET System is Critical to Normal Brain Function

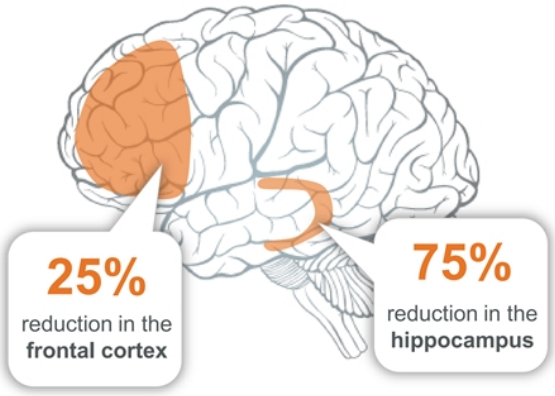
MET is one of the most stably expressed genes in the adult human brain

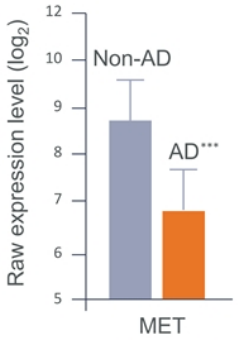
Stable MET expression is a signature of the healthy adult brain[1]

Suggests that dysregulation of HGF/MET could be implicated in brain pathologies

MET expression is reduced in the brains of AD patients[2]

NEURONAL MET EXPRESSION IN ALZHEIMER'S DISEASE





Adapted from Hokama et al, Cerebral Cortex 2014; ***p<0.001

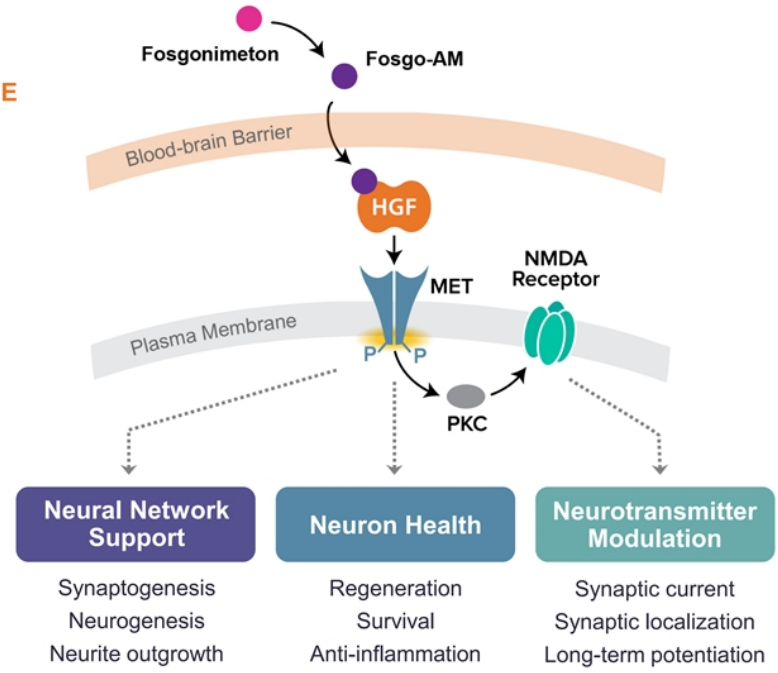
[1] Hawrylycz et al, *Nature Neuroscience* 2015
[2] Hamasaki et al, *Neuropathology* 2014

Fosgonimeton (ATH-1017): A Positive Modulator of the HGF/MET Neurotrophic System

MULTIMODAL, PROTECTIVE AND REGENERATIVE

Fosgonimeton:

- Small molecule prodrug that is rapidly converted to an active metabolite (Fosgo-AM) in plasma
- Crosses the blood-brain barrier
- Positively modulates HGF/MET
- Administered via subcutaneous injection

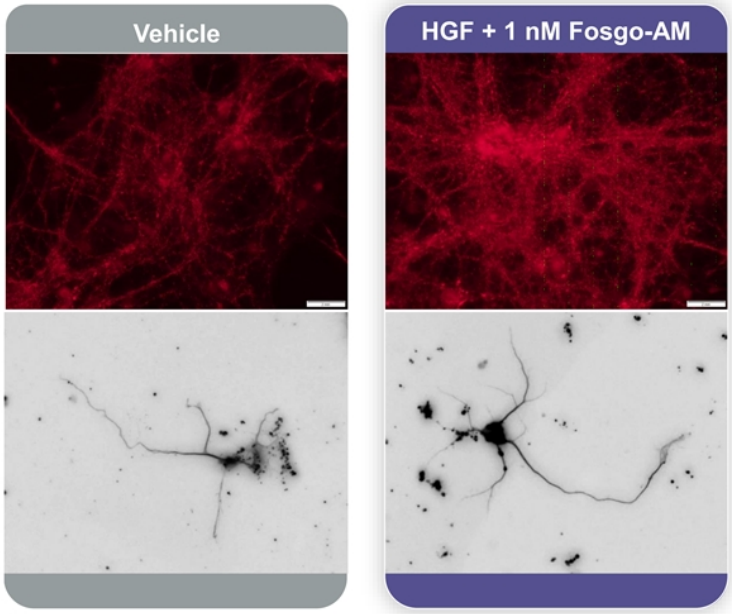


Neural Network Support	Neuron Health	Neurotransmitter Modulation
Synaptogenesis	Regeneration	Synaptic current
Neurogenesis	Survival	Synaptic localization
Neurite outgrowth	Anti-inflammation	Long-term potentiation

HGF/MET signaling and downstream effects described in:
Desole et al, *Frontiers in Cell and Dev Bio* 2021
Funakoshi and Nakamura, *Current Signal Transduction Therapy* 2011

Fosgo-AM Enhances Synaptogenesis and Neurite Outgrowth

PRECLINICAL DATA IN PRIMARY RAT HIPPOCAMPAL NEURONS



Synaptic count (number of synapses) and **synaptic strength** (relative abundance of presynaptic vesicles per synapse) were both significantly enhanced with fosgo-AM

Neurite outgrowth was significantly improved following treatment with fosgo-AM

Scale bars, 2mm
HGF, hepatocyte growth factor
HGF alone was also assessed and did not show statistically significant effects on synaptogenesis and neurite outgrowth

Clinical Development Strategy Includes Measures Strongly Correlated with Cognition

QUANTITATIVE EEG (qEEG)

Translational tool from rodents to humans

PK/PD modeling for dose selection

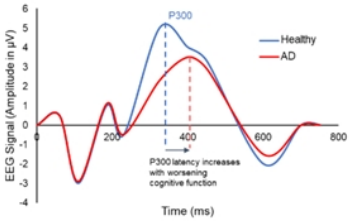
Noninvasive EEG recordings reflect brain activity and function

EEG records brain electrical activity from electrodes placed on the scalp

EVENT RELATED POTENTIALS (ERP): P300 Latency

Functional measurement for working memory access and executive function

Strongly correlated with memory improvement



Pathological changes in P300 latency correlate with cognitive impairment

Approved therapies have demonstrated parallel improvement in P300 latency and cognition

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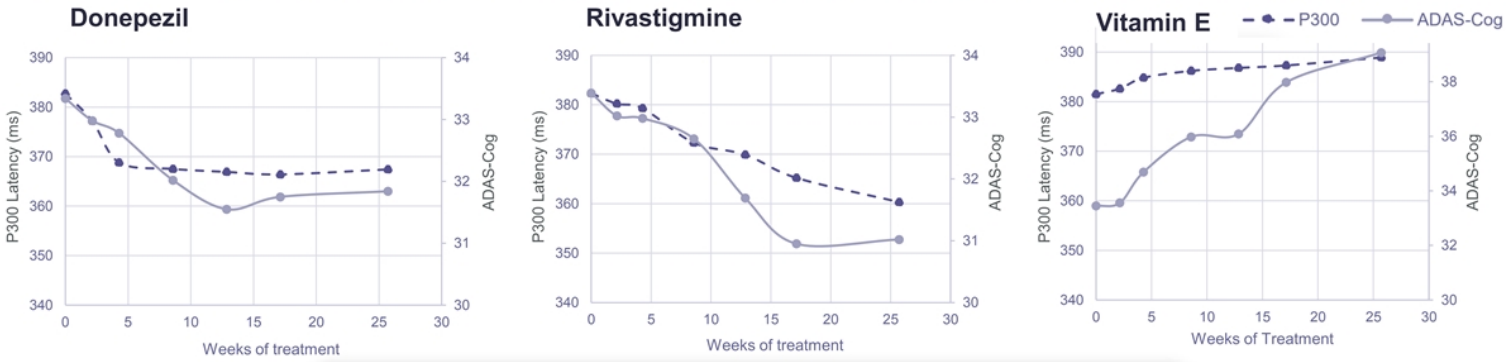
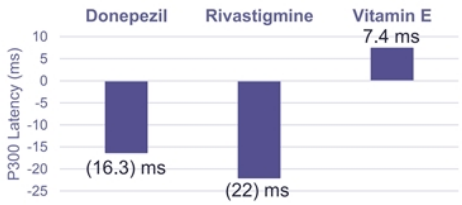
Changes in P300 Latency Correlate with Cognitive Outcomes with Treatment of Approved Therapies in AD Subjects

PREVIOUSLY PUBLISHED RESULTS SUPPORT THE CORRELATION OF P300 LATENCY AND COGNITION



Peak P300 Latency Change

Improvement in cognition (ADAS-Cog ↓) is correlated with reduction in P300 latency



Adapted from Thomas et al, *Clin Neuropharmacology* 2001

Fosgonimeton Treatment Improved P300 Latency in AD Subjects – CTAD 2019

PHASE 1B – AD SUBJECTS

N=11

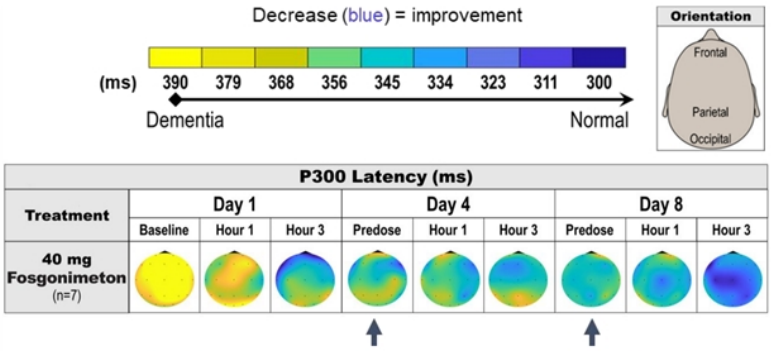
Randomized, Placebo, Fosgonimeton (40 mg)

Subcutaneous, Daily, 8 days

ERP OBSERVATIONS

ERP analysis to-date suggests treatment effects on P300 latency

- Gradual decrease in latency over time in the treated group (N=7)
- Short-term, rapid improvements are indicative of neurotransmitter, NMDA receptor modulation
- Lasting effects may be indicative of connectivity and structural improvements



Decreased latency on pre-dose recordings (arrows) taken 24 hours after the last dose on the previous day may be indicative of sustained improvement

Fosgonimeton Treatment Improved P300 Latency in AD Subjects – CTAD 2019

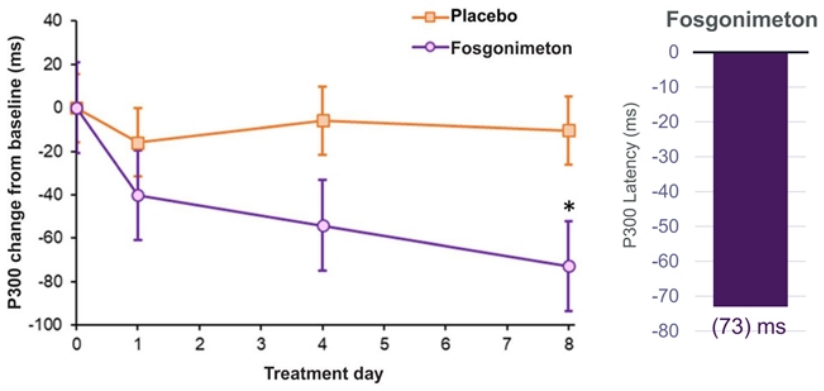
PHASE 1B – AD SUBJECTS

- Group averages of AD subjects receiving fosgonimeton (N=7) demonstrate decreased P300 latency over time

Significant change from baseline observed on Day 8

- AD subjects receiving placebo (N=4) had no consistent P300 latency change from baseline to study end

P300 LATENCY: ACTIVE v PLACEBO TREATED AD SUBJECTS



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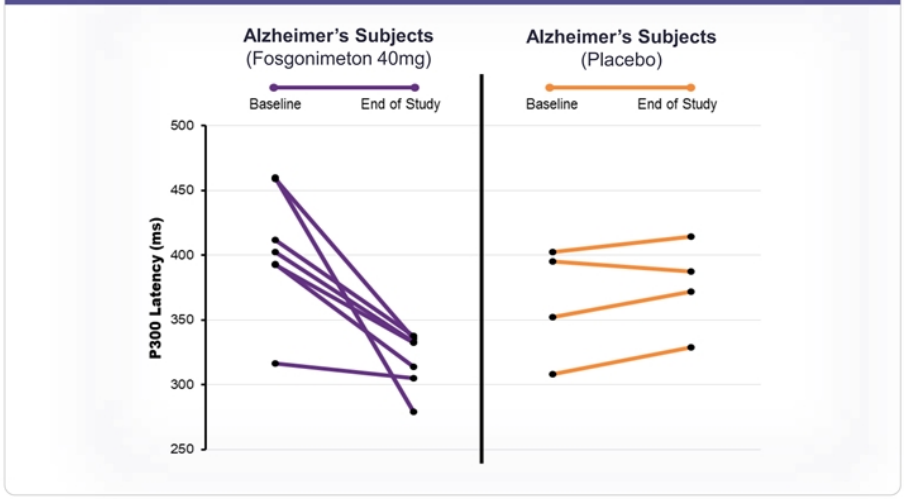
Note: P300 data from FZ, CZ, and PZ electrodes. Data plotted as mean +/- SE. *p=0.027 with MMRM.

Fosgonimeton Treatment Improved P300 Latency in AD Subjects – CTAD 2019

PHASE 1B – AD SUBJECTS

- Every AD subject receiving fosgonimeton had a level of improvement in P300 latency
- AD subjects receiving placebo had no consistent response from baseline to end of study

P300 LATENCY: ACTIVE v PLACEBO TREATED AD SUBJECTS



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Note: P300 data from FZ, CZ, and PZ electrodes.

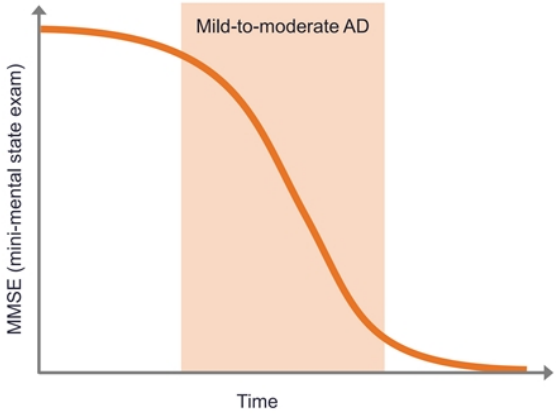
Why Mild-to-Moderate AD Instead of Pre-Dementia?

Medical need:

The point of most accelerated disease progression[1,2]

Currently marketed drugs in mild-to-moderate space have only modest effects[3]

Higher financial burden than pre-dementia[4]



Reduced development risk:

Clinical, syndromal diagnosis is possible[5]

Increased likelihood of tangible placebo decline

Established regulatory path (AChEIs, memantine)

1. Ower et al, *Eur J Epidemiol* 2018
2. Caroli et al, *Neurobiol Aging* 2010
3. Fink et al, *Ann Intern Med* 2020
4. Cerejeira et al, *Front Neurol* 2012
5. de Aquino et al, *Front Neurol* 2021

Fosgonimeton Phase 2 Trial (ACT-AD)

PROOF-OF-CONCEPT TRIAL TO HELP BETTER UNDERSTAND NATURE OF NOVEL INTERVENTION

POPULATION	TREATMENT DURATION	ENDPOINTS AND TIMELINE
ACT-AD: N=77 (recruitment complete) mild-to-moderate AD dementia subjects (55-85 years; CDR 1 and 2; MMSE 14-24 incl.)	**26-week randomized, double-blind treatment, + optional 26-week OLEX** Fosgonimeton (40 mg) Fosgonimeton (70 mg) Placebo **Randomization (1:1:1)**	**PRIMARY ENDPOINT** • Change of P300 latency • Safety **SECONDARY ENDPOINTS** • Cognition: ADAS-Cog11 • Global clinical change: ADCS CGIC - Clinician • Function: ADCS-ADL23 **TIMELINE** • Data readout expected by end of 2Q22

Potential Pathways to success:

- Achieves statistical significance on primary endpoint
- Key secondary endpoints trending
- Functions as interim analysis for LIFT-AD without statistical penalty

Fosgonimeton Phase 3 Trial (LIFT-AD)

TRIAL MAY PROVIDE PIVOTAL EVIDENCE TO SUPPORT PRODUCT REGISTRATION

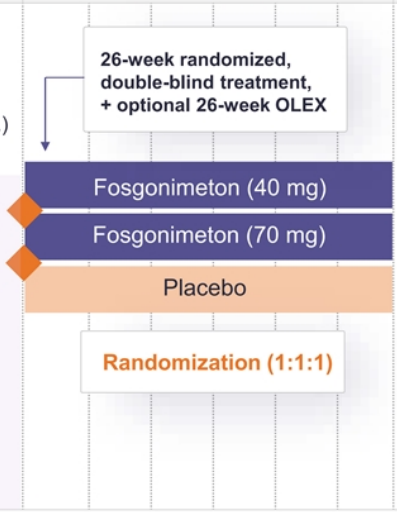
POPULATION

LIFT-AD: Target N=~420
mild-to-moderate AD dementia subjects
(55-85 years; CDR 1 and 2; MMSE 14-24 incl.)

Potential Pathways to success:

- Achieves statistical significance on primary endpoint
- Achieves statistical significance on two key secondary endpoints, which may support approval with a single pivotal study

TREATMENT DURATION



ENDPOINTS AND TIMELINE

PRIMARY ENDPOINT

- Global Statistical Test (GST) – unbiased composite, fed by data from two key secondaries
- Safety

SECONDARY ENDPOINTS

- Cognition: ADAS-Cog11
- Global clinical change: ADCS CGIC - Clinician
- Function: ADCS-ADL23

TIMELINE

- Complete enrollment expected 3Q22
- Data readout expected 1H23

Fosgonimeton – Potential First-Line Therapy to Improve Cognition

35 million
Estimated Alzheimer's cases worldwide[1]

Multi-Billion $ Market
Despite generic entries

Only One
New product (Aduhelm™) launched since 2003

Over 100 million globally by 2050
~900,000 new patients diagnosed annually in the US alone[1,2]

6.2 million treatment eligible patients in the US in 2021 based on prevalence data
Growing at 3% per year[2,3]

Mild to Moderate comprises 81% of all patients with Alzheimer's Disease
78.5% of these patients receive Rx therapies[3,4]

Significant opportunity for fosgonimeton
Market research suggests favorable reaction and receptivity to fosgonimeton base case target product profile as a potential first-line therapy to improve cognition[5]

[1] https://www.who.int/news-room/fact-sheets/detail/dementia
[2] https://www.alz.org/media/documents/alzheimers-facts-and-figures.pdf
[3] GlobalData AD prevalence data access and analysis
[4] https://www.nia.nih.gov/news/half-alzheimers-disease-cases-may-be-mild
[5] ClearView Healthcare Partners Market Research Analysis

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FOSGONIMETON

Parkinson's Disease Dementia (PDD) and Dementia with Lewy Bodies (DLB)

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Phase 2 Trial in PDD and DLB

Shape TRIAL

PROOF-OF-CONCEPT TRIAL TO UNDERSTAND THE POTENTIAL OF FOSGONIMETON BEYOND ALZHEIMER'S DISEASE

POPULATION	TREATMENT DURATION	ENDPOINTS AND TIMELINE
N=~75 mild-to-moderate PDD or DLB subjects (MOCA 11-23 at baseline)	**26-week randomized, double-blind treatment** Fosgonimeton (40 mg) Fosgonimeton (70 mg) Placebo **Randomization (1:1:1)**	**PRIMARY ENDPOINT** • Global Statistical Test (combining P300 and ADAS-Cog13) • Safety **SECONDARY ENDPOINTS** • P300 Latency • Cognition: ADAS-Cog13 • Global clinical change: ADCS CGIC - Clinician • Function: ADCS-ADL23 • Motor Function: MDS-UPDRS (Exploratory) **TIMELINE** • First patient dosed 1Q22 • Actively enrolling

Potential Pathways to success:

- Achieves statistical significance on primary endpoint
- Shows trends on either cognition and/or motor function

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PDD and DLB – Critical Unmet Need and Significant Opportunity

Nearly 1 million
people in the US and more than 10 million people globally are living with Parkinson's disease (PD)[1]

~50%
of PD patients experience dementia symptoms[2,4]

Only One
treatment option for dementia symptoms of PD[1,3]

DLB is the third most common cause of dementia
accounting for 5-15% of all dementia cases globally[2,4]

PDD and DLB are both types of Lewy body disorder,
differentiated by onset of dementia symptoms relative to PD diagnosis[3]

TYPES OF LEWY BODY DISORDER

$51.9B
Economic burden of PD overall in the US, as of 2017[4]

Significant opportunity for fosgonimeton
Initial market research suggests fosgonimeton base case target product profile has the potential to address an overlooked and underserved PDD and DLB patient population[5]

[1] https://www.parkinson.org/Understanding-Parkinsons
[2] https://www.alz.org/alzheimers-dementia/what-is-dementia/types-of-dementia
[3] Galasko, *Neurol Clin* 2017
[4] Yang et al, *NPJ Parkinsons Dis* 2020
[5] ClearView Healthcare Partners Market Research Analysis

Fosgonimeton Program Summary

CHANGING THE TREATMENT PARADIGM TO RESTORE NEURONAL HEALTH

Based on strong science using novel approach to leverage naturally occurring repair mechanism

Compelling data in Alzheimer's patients suggesting improved neuronal connectivity

LIFT-AD – a potentially pivotal program, informed by ACT-AD Phase 2 trial

Opportunity to expand into additional indications including PDD and DLB

SHAPE Phase 2 trial –first patient dosed 1Q22

Significant market potential

- **Over 2.5 million** mild-to-moderate AD patients in the US being treated with therapies with continued unmet need
- PDD, DLB and other dementias represent additional large market opportunities
- Complementary not competitive with current and potentially future therapies

Nearing key anticipated value inflection points:

ACT-AD	LIFT-AD
Data by end of 2Q22	LPI 3Q22 Data 1H23

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ATH-1020

Neuropsychiatric Indications

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ATH-1020 – Addressing Neuronal Connectivity

PHYSIOLOGICAL CHANGES IN THE BRAIN AFFECT BEHAVIOR AND EMOTION

Our novel approach is focused on restoring neuronal health and function to repair disruptions in neuronal connectivity found in a variety of neuropsychiatric diseases

- Preclinical data demonstrate **enhancing HGF/MET activity has anti-depressant and anxiolytic effects**[1,2]
- Human clinical trials also show an association between **reduced HGF/MET expression levels and depression/anxiety and schizophrenia**[3-7]
- **ATH-1020**
  - A brain-penetrant small molecule positive modulator of HGF/MET
  - Demonstrated improvements in depression and schizophrenia in preclinical animal models
  - Convenient once-daily oral dosing

Phase 1 first in-human studies launched, first subject dosed 1Q22

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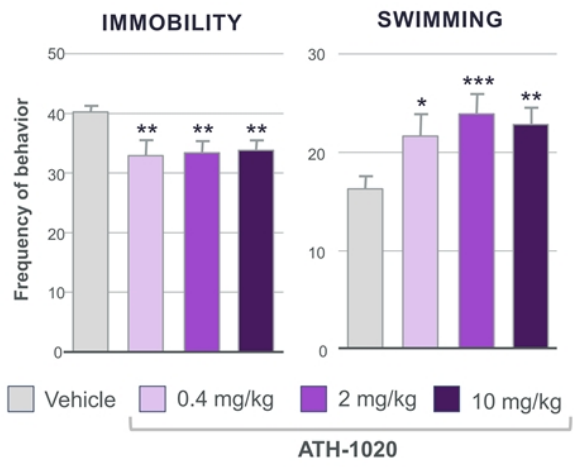
[1] Isogawa et al, *Neuropsychobiology* 2005
[2] Wakatsuki et al, *Neuropeptides* 2007
[3] Russo, *Biomarker Insights* 2010
[4] Ciuculete et al, *Epigenetics* 2019
[5] Ramsey et al, *PLoS ONE* 2016
[6] Russo, *Proteomic Insights* 2010
[7] Burdick et al, *AM J Psychiatry* 2010

Preclinical Studies Demonstrate Improvement in Rodent Models of Depression and Schizophrenia

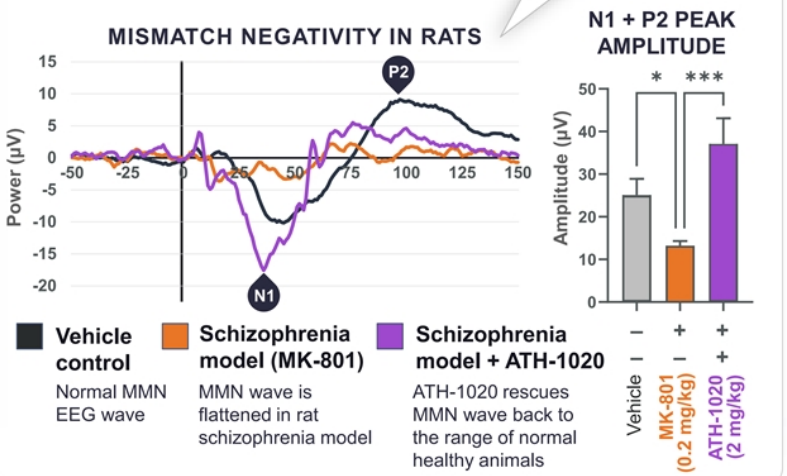
Reduction of Depression-related Behaviors in the Rodent Forced-Swim Test Model



Two-way ANOVA with Dunnett's multiple comparisons vs vehicle were conducted. *P<0.05, **P<0.01, ***P<0.001.

ATH-1020 Rescues the MMN Deficit Seen in the Rat MK-801 Schizophrenia

MMN Response is Translatable from Rodent Models to Human Clinical Schizophrenia



Vehicle control
Normal MMN EEG wave

Schizophrenia model (MK-801)
MMN wave is flattened in rat schizophrenia model

Schizophrenia model + ATH-1020
ATH-1020 rescues MMN wave back to the range of normal healthy animals

One-way ANOVA with Dunnett's multiple comparisons post test vs MK-801 were conducted. *P<0.05, ***P<0.001.

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Market Opportunity: Neuropsychiatric Indications

Depression

3.8% Worldwide population affected by depression[1]

~280 million

people of all ages suffered from depression, globally[1]

13.1 million

U.S. adults aged 18 or older had at least one major depressive episode with severe impairment in the past year[2]

Global Market Size for Depression[3]

2021:	2028 Projected:
$11.9B	**$15.4B**

Global Market Size for Schizophrenia[4]

2018:	2026 Projected:
$6.75B	**$9.48B**

Schizophrenia

~20 million

people across the globe are affected with schizophrenia[1]

~1.2%

of Americans (3.2 million) have the disorder[5]

[1] World Health Organization Data Fact Sheets
[2] https://www.nimh.nih.gov/health/statistics/major-depression
[3] Coherent Market Insights – Depression
[4] Fortune Business Insights – Schizophrenia
[5] https://www.mentalhelp.net/schizophrenia/statistics/

Corporate

Athira PHARMA

Athira Management Team with Significant CNS Product Development and Approval Experience

EXECUTIVE LEADERSHIP

Mark Litton, PhD
President and CEO

Rachel Lenington
COO

Hans Moebius, MD, PhD
CMO

Kevin Church, PhD
Executive Vice President, Research

Glenna Mileson
CFO

Mark Worthington, JD
General Counsel

BOARD OF DIRECTORS

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BlueRipple Capital, LLC

Joseph Edelman
Perceptive Advisors

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Former CFO of Nohla Therapeutics, NanoString Technologies and ZymoGenetics

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Wave Life Sciences

Mark Litton, PhD
President and CEO

John Fluke, Jr.
Fluke Capital Management

Barbara Kosacz
Kronos Bio

Grant Pickering
Vaxcyte, Inc.

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Achievements and Upcoming Milestones

RECENT ACHIEVEMENTS

- ✓ Enrollment completed for Phase 2 ACT-AD trial in Oct 2021
- ✓ Strong enrollment to-date in the Phase 3 LIFT-AD trial
- ✓ Open label extension trial underway for ACT-AD and LIFT-AD
- ✓ First patient dosed in Phase 2 SHAPE trial 1Q22
- ✓ First subject dosed with first oral molecule, ATH-1020, in Phase 1 trial as a potential treatment candidate for neuropsychiatric indications in 1Q22
- ✓ Continued to strengthen IP portfolio including issuance of fosgonimeton (ATH-1017) US patent
- ✓ Strong balance sheet – cash of $319.7M as of 12/31/21 and no debt

LOOKING AHEAD

- → **Phase 2 ACT-AD Trial:** Data readout expected by end of 2Q22
- → **Phase 3 LIFT-AD Trial:** Complete enrollment expected in 3Q22; Data readout expected 1H23
- → **SHAPE** enrollment ongoing
- → Ongoing IND-enabling studies of ATH-1019 in peripheral indications

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