Athira PHARMA

Corporate Presentation

SEPTEMBER 2022

ADVANCING NEW THERAPIES FOR NEURONAL HEALTH

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The ACT-AD trial was 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 presentation 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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OUR MISSION

To restore lives by advancing bold therapies for neuronal health, thoughtfully and urgently

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Investment Highlights

Novel small molecule compounds designed to act on naturally occurring mechanism to repair and restore neuronal health

HGF/MET neurotrophic system is critical to normal brain function and plays a role in neurological diseases

- Alzheimer's, Parkinson's, ALS, Depression, Schizophrenia, Neuropathic Pain

Lead program fosgonimeton suggested potential neuroprotection and improvements in cognition and function in Alzheimer's patients

- Exploratory ACT-AD study showed potential improvements in both biomarker (NfL) and psychometric measurements (ADAS-Cog, ADCS-ADL) in Alzheimer's patients
- Adapting LIFT-AD study to focus on the evaluation of fosgonimeton alone
- Interim analysis for LIFT-AD study is intended to further validate and inform program strategy

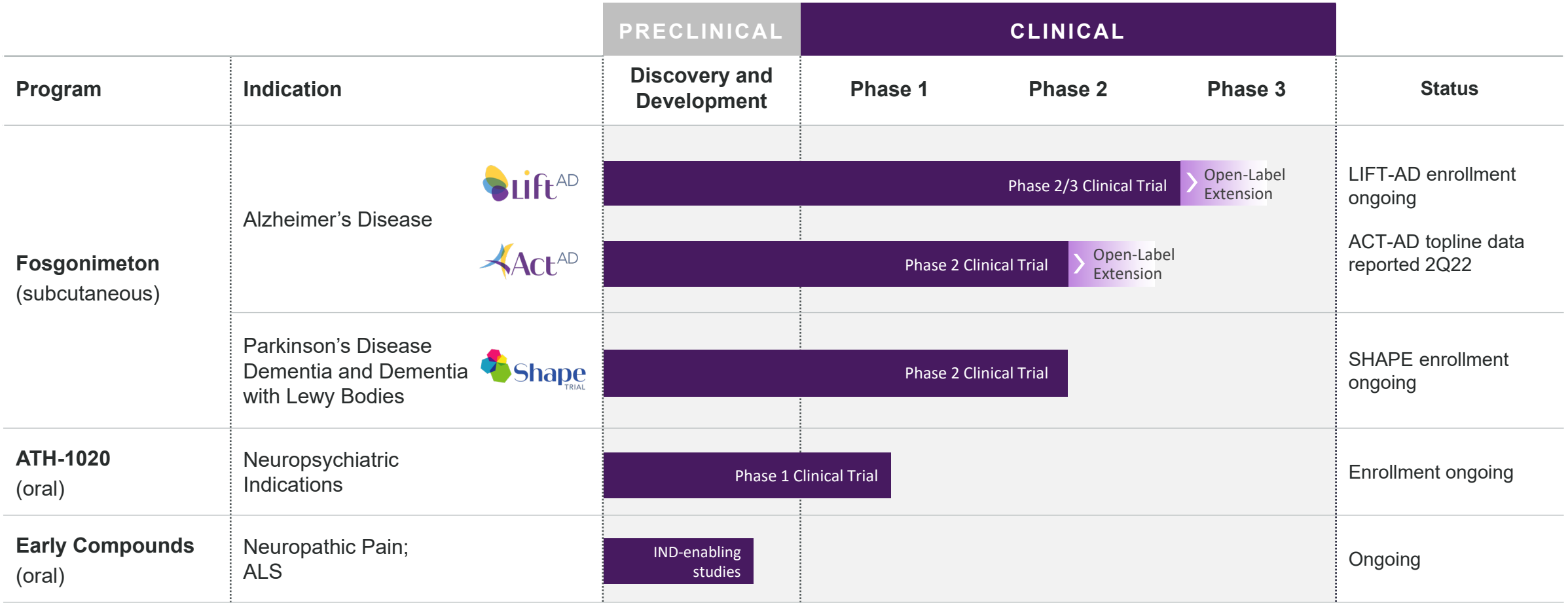
Robust pipeline of HGF/MET small molecules to address a variety of neurological diseases

Strong balance sheet to support clinical programs through key inflection points

Leadership team with significant CNS product development and approval experience

AD, Alzheimer's disease; ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living; ALS, amyotrophic lateral sclerosis; CNS, central nervous system; HGF, hepatocyte growth factor; NfL, neurofilament light chain.

Therapeutic Potential Across a Broad Range of Clinical Applications



AD, Alzheimer's disease; ALS, amyotrophic lateral sclerosis; IND, investigational new drug.

HGF/MET is a Critical Neurotrophic System

MET EXPRESSION IS FOUND IN MULTIPLE CELL TYPES IN THE NERVOUS SYSTEM

HGF (ligand) and MET (receptor) are tightly regulated and conserved across species

HGF/MET activity is critical to neuronal function and survival

CELL TYPE	FUNCTION OF HGF/MET
Hippocampal neurons	Survival, dendritic maturation
Midbrain dopaminergic neurons	Survival, neurite extension, increased TH activity
Cerebral cortical neurons	Survival, neurite extension
Motor neurons	Survival, neurite extension
Oligodendrocyte progenitor cells	Proliferation, migration
Astrocytes	Migration, EAAT2/GLT-1 expression
Schwann cells	Proliferation, migration
SVZ neural stem-like cells	Growth and self-renewal

HGF, hepatocyte growth factor; EAAT2/GLT-1, excitatory amino acid transporter 2/glutamate transporter 1; SVZ, subventricular zone; TH, tyrosine hydroxylase.

Desole et al, *Frontiers in Cell and Dev Bio* 2021; Funakoshi and Nakamura, *Curr Signal Transduc Ther* 2011; Korhonen et al, *Eur J Neurosci* 2000; Akita et al, *Exp Neurol* 2008; Hamanoue et al, *J Neurosci Res* 1996; Sun et al, *Brain Res Mol Brain Res* 2002; Ebens et al, *Neuron* 1996; Yan et al, *J Neurosci Res* 2002; Sun et al, *J Neurosci* 2002; Ko et al, Sci Rep 2018; Nicoleau et al, *Stem Cells* 2009.

Preclinical Evidence Support HGF/MET Mechanism of Action

POSITIVELY MODULATING HGF/MET PATHWAY SHOWS POSITIVE EFFECTS ACROSS MULTIPLE NEUROLOGICAL DISEASES

In vitro models

Mechanism of action pathway
- MET
- ERK
- AKT

Neurotrophic
- Synaptogenesis
- Neurite outgrowth

Neuroprotection against toxic insults
- Inflammation – LPS
- Oxidative stress – H_2O_2
- Excitatory toxicity – glutamate
- Pesticides/herbicides – rotenone, MPP+, 6-OHDA

In vivo models

Alzheimer's disease
- Scopolamine
- LPS

Parkinson's disease
- 6-OHDA

Neuropathic Pain
- Streptozotocin

ALS
- TDP-43

Depression/anxiety
- Chronic unpredictable stress
- Forced-swim test

Schizophrenia
- MMN following MK-801 treatment

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6-OHDA, 6-hydroxydopamine; AKT, protein kinase B; ALS, amyotrophic lateral sclerosis; ERK, extracellular-regulated kinase; H_2O_2, hydrogen peroxide; HGF, hepatocyte growth factor; LPS, lipopolysaccharide; MK-801, dizocilpine hydrogen maleate; MMN, mismatch negativity; MPP^+, 1-methyl-4-phenylpryidinium; TDP-43, TAR DNA-binding protein 43.

Lead Program: Fosgonimeton (ATH-1017)

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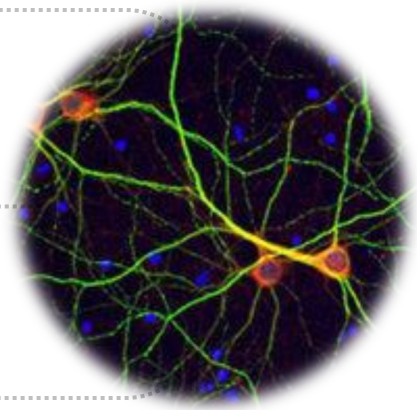
Alzheimer's Disease Pathology

MULTIFACTORIAL COMPLEX PATHOLOGIES LEAD TO NEURODEGENERATION

Alzheimer's disease

- **Nervous System** → Protein misfolding, Oxidative stress, Synaptic dysfunction
- **Vasculature** → Reduced cerebral blood flow
- **Immune System** → Inflammation
- **Neurotransmitters** → Neurotransmitter system dysfunction



Neurodegeneration Synaptic Loss

Loss of network connectivity

Symptom onset

Disease progression

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HGF/MET System is Critical to Normal Brain Function

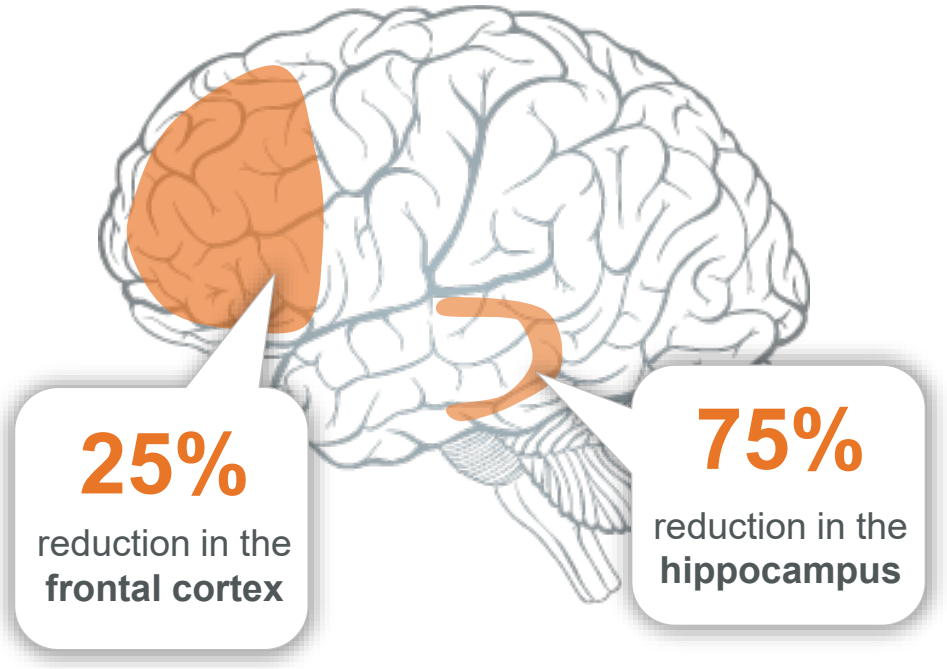
MET is one of the most stably expressed genes in the adult human brain

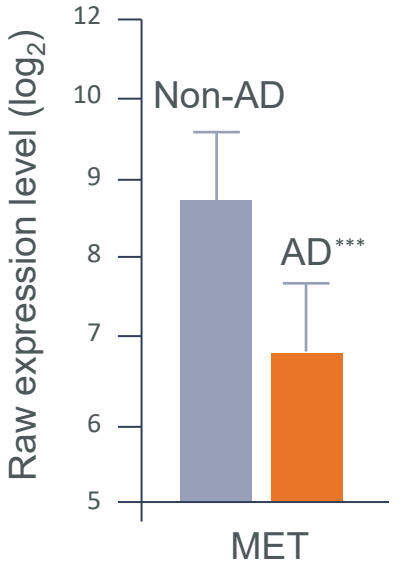
Stable MET expression is a signature of the healthy adult brain[1]

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

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

MET EXPRESSION IN ALZHEIMER'S DISEASE





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

AD, Alzheimer's disease; HGF, hepatocyte growth factor.

[1] Hawrylycz et al, *Nature Neuroscience* 2015.

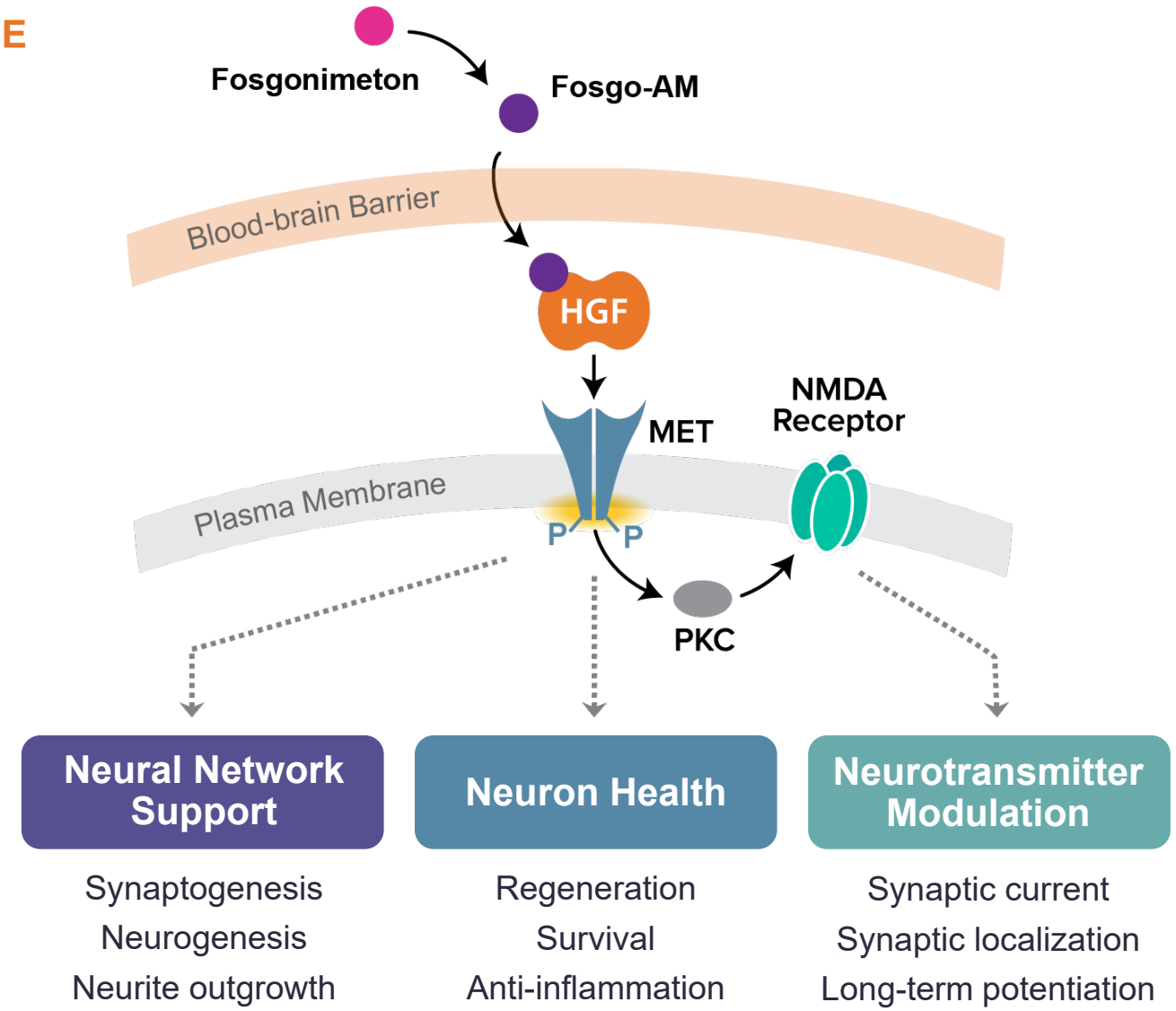
[2] Hamasaki et al, *Neuropathology* 2014.

Fosgonimeton: Positive Modulator of 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



HGF, hepatocyte growth factor; NMDA, *N*-methyl-D-aspartate; P, phosphorylation; PKC, protein kinase C.

Desole et al, *Frontiers in Cell and Dev Bio* 2021.
Funakoshi and Nakamura, *Current Signal Transduction Therapy* 2011.

Neurotrophic Effects of Fosgonimeton

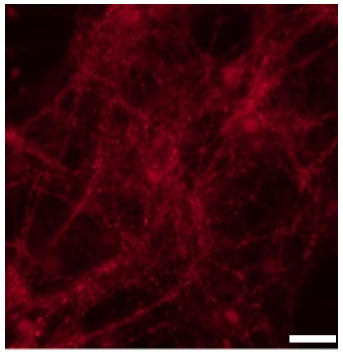
ENHANCED SYNAPTOGENESIS, NEURONAL PLASTICITY, AND NEUROPROTECTION IN VITRO

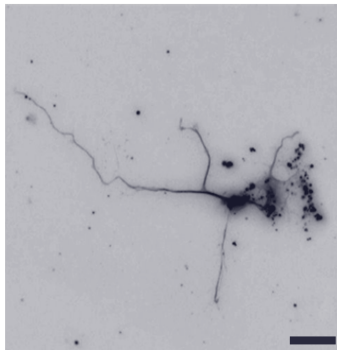
Synaptogenesis
Formation of new synapses

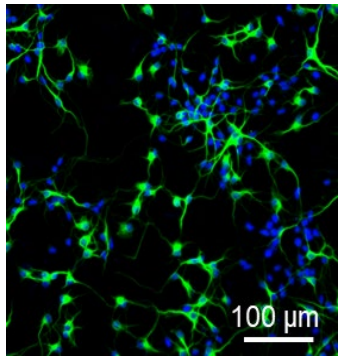
Neurite Outgrowth
Development of neuronal projections

Network Enhancement
Development of new neurons and networks

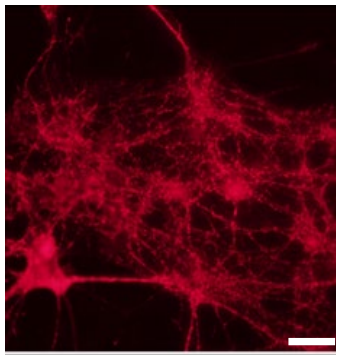
Control

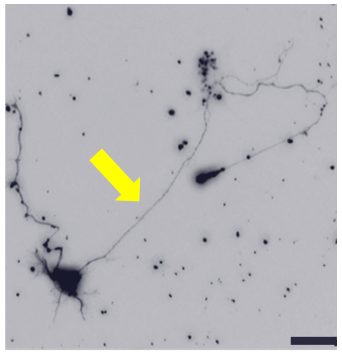


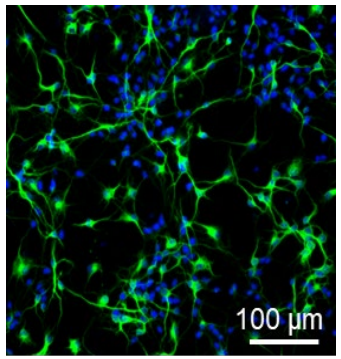




Fosgo-AM







Synaptobrevin-II | **β-III Tubulin** | **MAP2 / Hoechst**

Fosgonimeton also protected cells against various neurotoxic insults

MAP2, microtubule associated protein 2.
Scale bars for synaptogenesis and neurite outgrowth panels = 20 µm.
Reda et al, *Alzheimer's Association International Conference* 2022.
Johnston et al, *ASENT Annual Meeting* 2022.

Initial Focus on Mild-to-Moderate Alzheimer's Disease

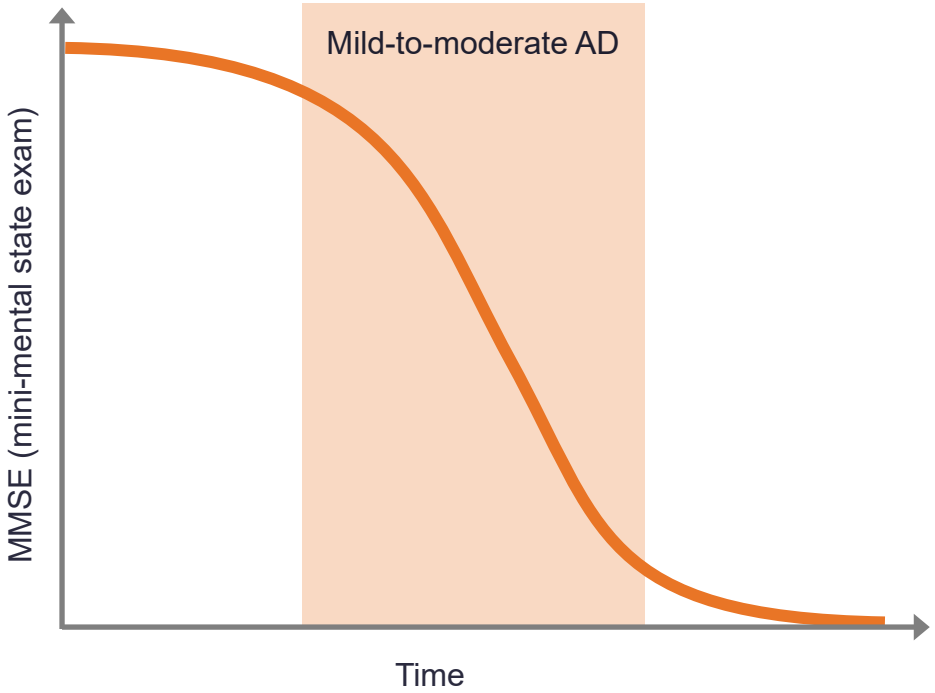
UNIQUE MECHANISM OF ACTION COULD BE APPLICABLE ACROSS ALL DISEASE STAGES

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)

AChEI, acetylcholinesterase inhibitor; AD, Alzheimer's disease.

[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 ACT-AD Trial

EXPLORATORY STUDY TO INFORM LARGER LIFT-AD STUDY IN SIMILAR POPULATION

POPULATION

ACT-AD: N=77
Mild-to-moderate AD subjects

- 55-85 years
- CDR 1 and 2
- MMSE 14-24
- 40% of patients not on background AChEI

PRIMARY ENDPOINT

- Change of ERP P300 latency
- Safety

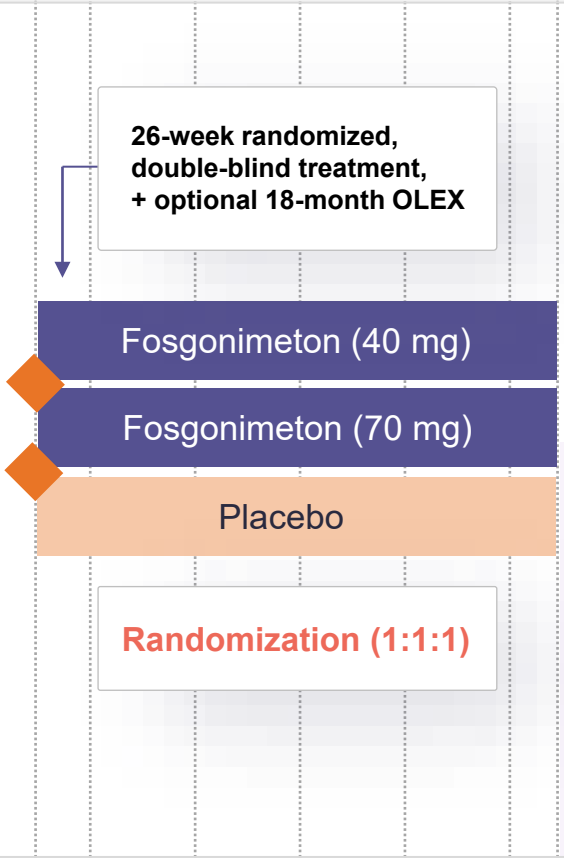
SECONDARY ENDPOINTS

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

EXPLORATORY ENDPOINTS

- Fluid biomarkers (e.g., NfL)

TREATMENT DURATION



RESULTS

Full Study Population:

- Did not achieve statistical significance on primary endpoint of biomarker ERP P300 latency
- Well tolerated with a favorable safety profile
- Showed a numerical improvement in the functional measure of ADCS-ADL23 and plasma levels of NfL, a validated fluid biomarker of neurodegeneration

Fosgonimeton Alone Population:

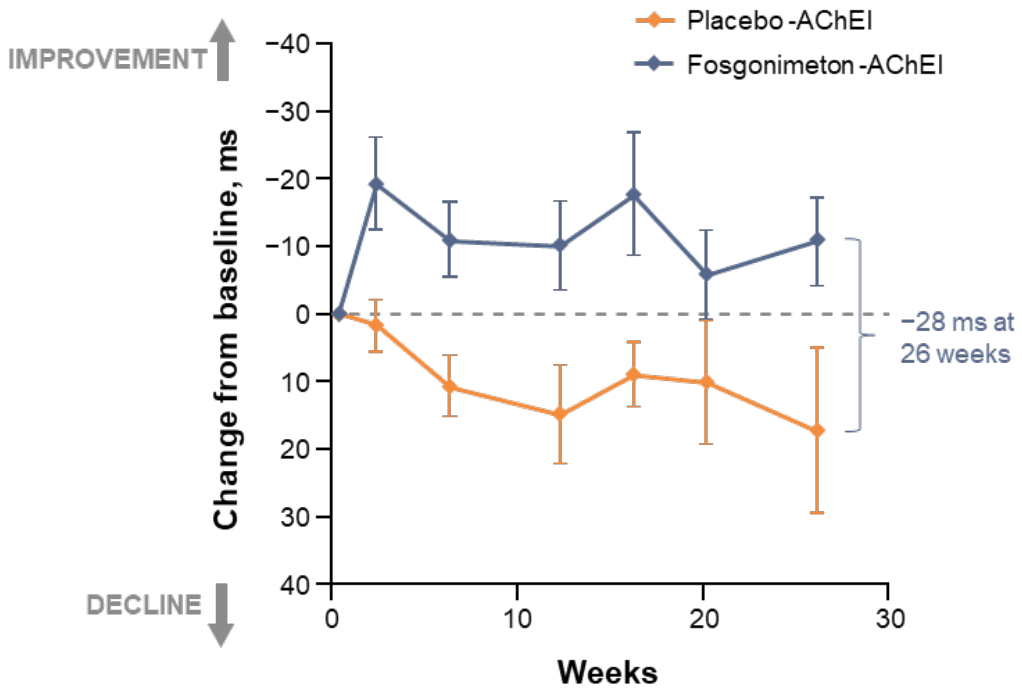
- Showed potentially beneficial change in ERP P300 latency (-28 milliseconds, n.s.)
- Improved cognition as measured by ADAS-Cog11 (-3.3 points, n.s.)
- Achieved a statistically significant improvement in plasma levels of NfL (p=0.018)

AChEI, acetylcholinesterase inhibitor; AD, Alzheimer's disease; ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living; ADCS-CGIC, Alzheimer's Disease Cooperative Study–Clinical Global Impression of Change; CDR, clinical dementia rating; ERP, event related potential; MMSE, mini-mental state examination; NfL, neurofilament light chain; OLEX, open-label extension.

Suggested Improvements in ERP P300 Latency and ADAS-Cog11

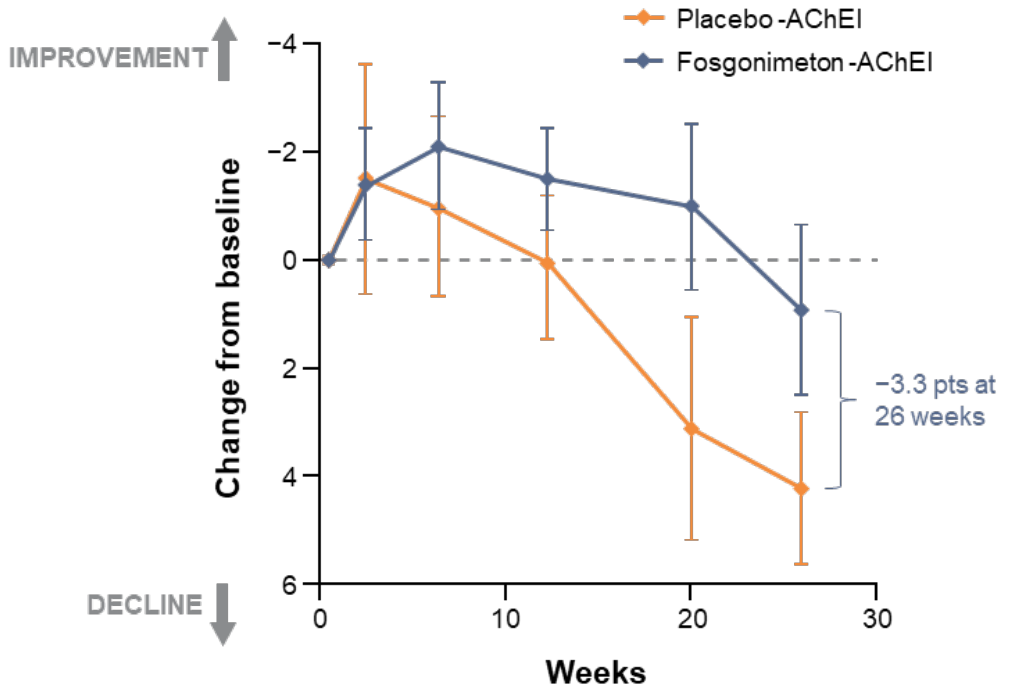
EFFECTS SHOWN IN FOSGONIMETON ALONE (WITHOUT BACKGROUND ACHEIS)

ERP P300 LATENCY



n at each visit	W2	W6	W12	W16	W20	W26
Placebo	8	8	6	6	7	6
Fosgonimeton	20	19	18	15	16	17

ADAS-COG11



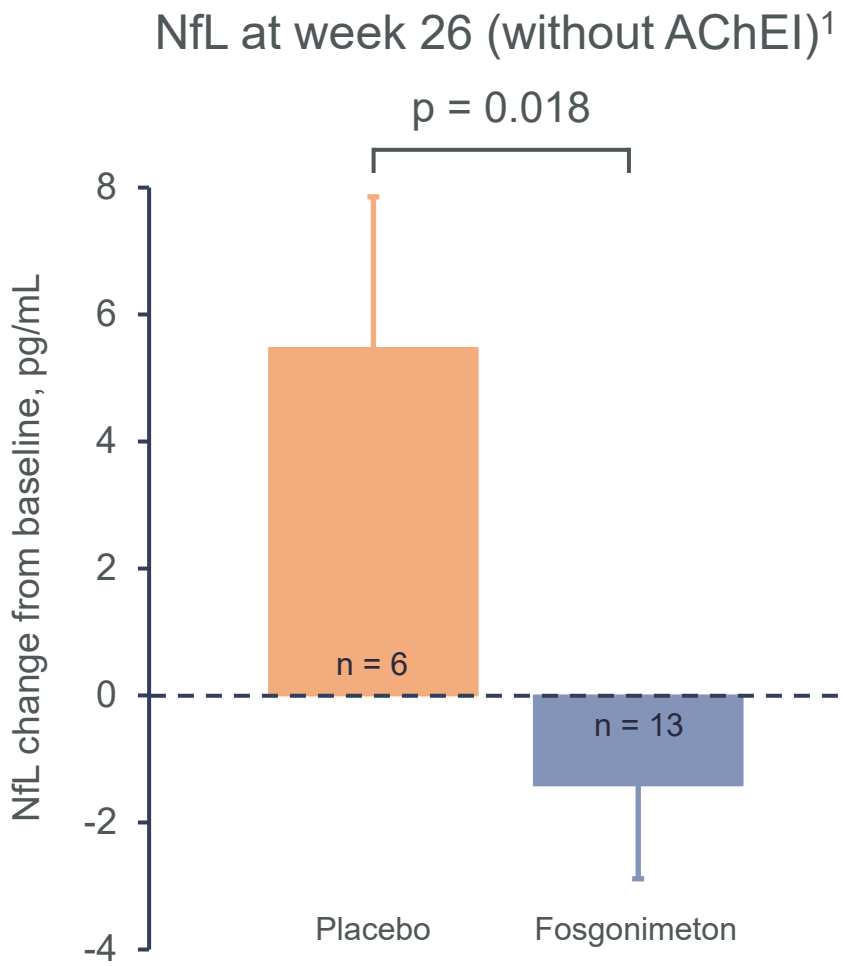
n at each visit	W2	W6	W12	W20	W26
Placebo	8	8	7	7	6
Fosgonimeton	20	20	18	17	18

AChEI, acetylcholinesterase inhibitor; ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ERP, event related potential; W, week.
mITT population. Data presented as unadjusted mean ± SEM.

Neurofilament Light Chain (NfL) Analysis Suggests Neuroprotection

VALIDATED FLUID BIOMARKER OF NEURODEGENERATION (CSF OR PLASMA)

Analysis of plasma NfL levels showed statistically significant improvements with fosgonimeton alone from baseline to week 26



CSF, cerebrospinal fluid; NfL, neurofilament light chain; SE, standard error.
[1] Data presented are least squares mean ± SE.

Fosgonimeton Phase 2/3 LIFT-AD Trial

LEARNINGS FROM ACT-AD INFORM TRIAL DESIGN OPTIMIZATION

POPULATION

LIFT-AD: Target N=TBD, informed by interim analysis

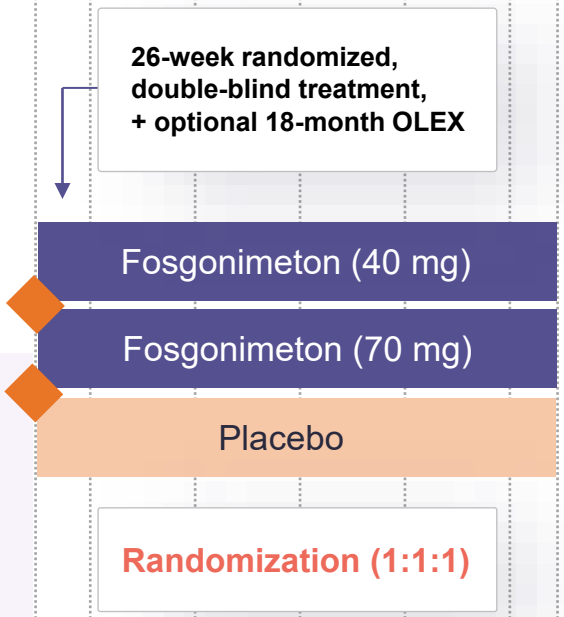
Mild-to-moderate AD subjects

- 55-85 years
- CDR 1 and 2
- MMSE 14-24
- Currently ~40% of patients not on background AChEI

Path Forward:

- Exclusion criterion added for subjects on background AChEIs
- Independent, unblinded interim analysis to inform sample size for primary endpoint

TREATMENT DURATION



RESULTS

PRIMARY ENDPOINTS

- Global Statistical Test (GST) – unbiased composite of data from two key secondary endpoints (ADAS-Cog11 and ADCS-ADL23)
- Safety

SECONDARY ENDPOINTS

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

EXPLORATORY ENDPOINTS

- Fluid biomarkers (e.g., NfL)

TIMELINE

- Enrollment ongoing

AChEI, acetylcholinesterase inhibitor; AD, Alzheimer's disease; ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living; ADCS-CGIC, Alzheimer's Disease Cooperative Study–Clinical Global Impression of Change; CDR, clinical dementia rating; GST, global statistical test; MMSE, mini-mental state examination; NfL, neurofilament light chain; OLEX, open-label extension.

Fosgonimeton – A New Potential Therapy for Alzheimer's Disease

35 million
Estimated Alzheimer's cases worldwide[1]

Multi-Billion $ Market
Despite generic entries

Zero
New marketed products 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[3,4]

Significant opportunity for fosgonimeton

Limited treatment options exist today for those with Alzheimer's disease; novel approaches to improve cognition and function are needed

[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.

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Potential of Fosgonimeton Beyond Alzheimer's Disease

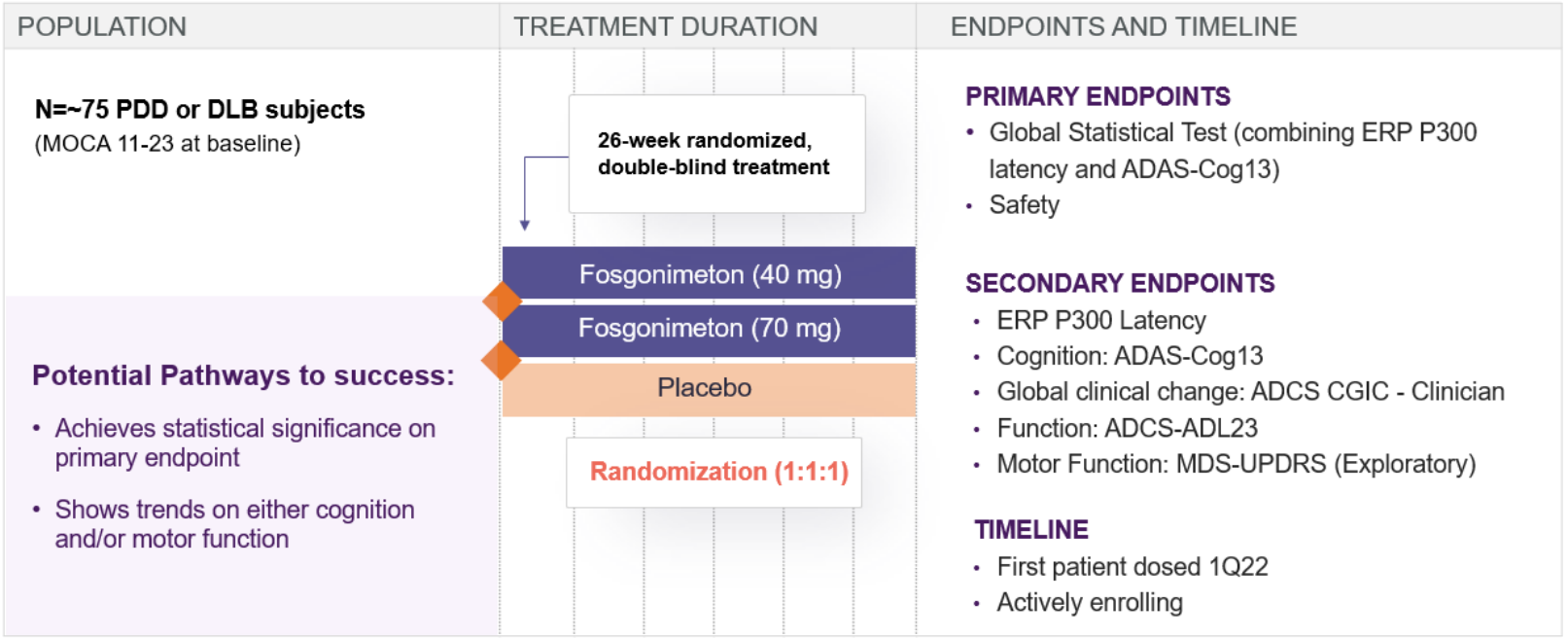
PROOF-OF-CONCEPT TRIAL IN PARKINSON'S DISEASE DEMENTIA AND DEMENTIA WITH LEWY BODIES

Beneficial effects on motor function, behavior and pathology in preclinical Parkinson's disease (PD) models

Data presentations at upcoming scientific conferences

Phase 2 study ongoing

~50% of nearly 1 million PD patients in the US experience dementia[1-3]



ADAS-Cog13, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living; ADCS-CGIC, Alzheimer's Disease Cooperative Study–Clinical Global Impression of Change; DLB, dementia with Lewy bodies; ERP, event related potential; GST, global statistical test; MDS-UPDRS, movement disorder society-unified Parkinson's disease rating scale; MOCA, Montreal cognitive assessment; PD, Parkinson's disease; PDD, Parkinson's disease dementia.

[1] https://www.parkinson.org/Understanding-Parkinsons.

[2] https://www.alz.org/alzheimers-dementia/what-is-dementia/types-of-dementia.

[3] Yang et al, *NPJ Parkinsons Dis* 2020.

Fosgonimeton Program Summary

CHANGING THE TREATMENT PARADIGM TO RESTORE NEURONAL HEALTH

Compelling biologic activity in Alzheimer's patients suggests potential for improved cognition, function and neuronal health

Based on promising science using novel HGF/MET positive modulators to repair and restore neuronal health

LIFT-AD – enrollment ongoing with a focus on fosgonimeton alone (without background AChEIs)

18-month open-label extension study ongoing with majority of patients rolling over

Urgent need for continued innovation and options that improve cognition and function for patients

Opportunity to expand into additional indications

AChEI, acetylcholinesterase inhibitor; AD, Alzheimer's disease; HGF, hepatocyte growth factor.

Neuropsychiatric Indications

ATH-1020

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ATH-1020 Phase 1 Study Ongoing in Healthy Volunteers

PHYSIOLOGICAL CHANGES IN THE BRAIN AFFECT BEHAVIOR AND EMOTION

Focused on restoring neuronal health and function to repair disruptions in neuronal connectivity found in a variety of neuropsychiatric diseases

A brain-penetrant small molecule positive modulator of HGF/MET with convenient once-daily oral dosing

Demonstrated improvements in depression and schizophrenia in preclinical animal models[1]

Independent published data demonstrate enhancing HGF/MET activity has anti-depressant and anxiolytic effects[2,3]

Human clinical trials also show an association between reduced HGF/MET expression levels and depression/anxiety and schizophrenia[4-8]

HGF, hepatocyte growth factor.

[1] Berthiaume et al, *ASENT Annual Meeting* 2022.
[2] Isogawa et al, *Neuropsychobiology* 2005.
[3] Wakatsuki et al, *Neuropeptides* 2007.
[4] Russo, *Biomarker Insights* 2010.
[5] Ciuculete et al, *Epigenetics* 2019.
[6] Ramsey et al, *PLoS ONE* 2016.
[7] Russo, *Proteomic Insights* 2010.
[8] Burdick et al, *AM J Psychiatry* 2010.

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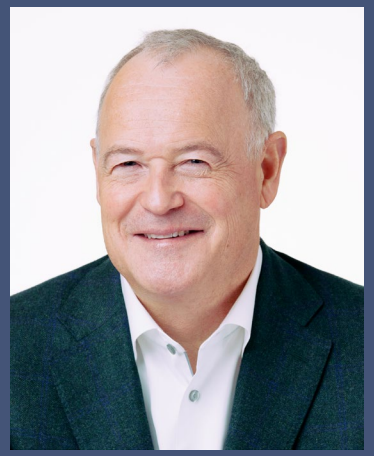
Athira Management Team with Significant CNS Product Development and Approval Experience

EXECUTIVE LEADERSHIP



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Achievements and Upcoming Milestones

RECENT ACHIEVEMENTS

- ✓ Phase 2 ACT-AD topline data reported 2Q22
- ✓ Amending LIFT-AD for focus on fosgonimeton alone
- ✓ Open label extension trial underway for ACT-AD and LIFT-AD
- ✓ First subject dosed in 1Q22 with first oral molecule, ATH-1020, in Phase 1 trial to evaluate safety of potential product candidate for neuropsychiatric indications
- ✓ Continued to strengthen IP portfolio
- ✓ Strong balance sheet – cash and cash equivalents of $282.2M as of June 30, 2022, and no debt

LOOKING AHEAD

- → **Complete LIFT-AD trial** enrollment
- → **Continued transition of patients** to open-label extension study
- → **Complete Phase 1** healthy volunteer study for ATH-1020
- → **Ongoing IND-enabling studies** of new product candidates
- → **Continued publications and presentations** of supportive scientific and clinical data at multiple medical meetings

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AD, Alzheimer's disease; IND, investigational new drug; IP, intellectual property.

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