Athira PHARMA

Restoring Lives by Advancing Bold Therapies

NOVEMBER 2021

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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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Athira PHARMA

Athira Pharma, Inc. (NASDAQ: ATHA) is a late clinical-stage biopharmaceutical company focused on developing small molecules to restore neuronal health and stop neurodegeneration.

Our mission is to restore neuronal health.

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Investment Highlights

Novel approach to rapidly improve cognition

Leveraging a critical repair pathway, HGF/MET, and naturally occurring repair mechanisms that are agnostic to underlying disease pathology

Lead Asset ATH-1017

Lead indication is in potentially pivotal trial

Encouraging Phase 1 clinical data in Alzheimer's disease

- **LIFT-AD trial actively recruiting, topline data by late 2022**
- **ACT-AD trial topline data by first half 2022**

Efficient clinical development strategy

Clinical development strategy to investigate the potential for fast onset with tangible cognitive improvement

Potential follow-on indications with established regulatory pathway and faster timelines

Pipeline of novel, small molecule compounds

Opportunity to explore several neurological indications

- **CNS degenerative, PNS disorders, neuropsychiatric indications**

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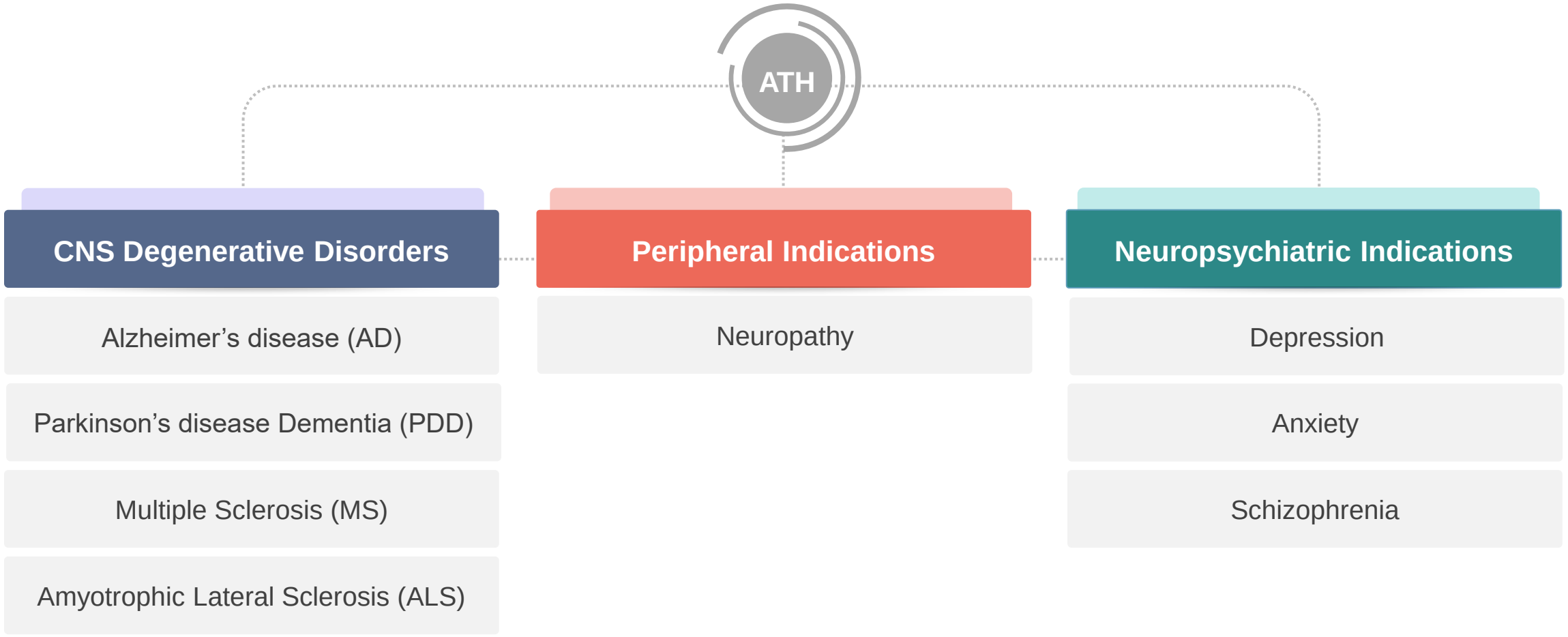
ATH Compounds

Have therapeutic potential in a broad range of clinical applications



Expanding a Pipeline of Novel, Small Molecule Compounds Designed to Improve Neuronal Health

PROGRAM (ROA)[1]	INDICATION	DISCOVERY AND DEVELOPMENT / PHASE 1 / PHASE 2 / PHASE 3	ANTICIPATED UPCOMING MILESTONES
ATH-1017 (SC)	Alzheimer's Disease	LIFT^{AD} Phase 2/3 Clinical Trial[2]	LIFT-AD topline data by end of 2022
		ACT^{AD} Phase 2 Clinical Trial	ACT-AD topline data by first half 2022
		Open-Label Extension for LIFT^{AD} & ACT^{AD}	26-week open label extension initiated June 2021
	Parkinson's Disease Dementia and Dementia with Lewy Bodies	SHAPE TRIAL Phase 2 Clinical Trial	Phase 2 initiation by end of 2021[3]
ATH-1020 (PO)	Neuropsychiatric Indications		IND filing by end of 2021
ATH-1019 (PO)	Peripheral Indications		IND filing by end of 2022

(1) RoA: route of administration; SC: subcutaneous; PO: oral.
(2) ATH-1017 for AD is in a Phase 2/3 clinical trial that may provide pivotal data in support of registration based on discussions with FDA.
(3) PDD clinical development of ATH-1017 will be based on results from Phase1a and 1b trials and include all required regulatory filings.

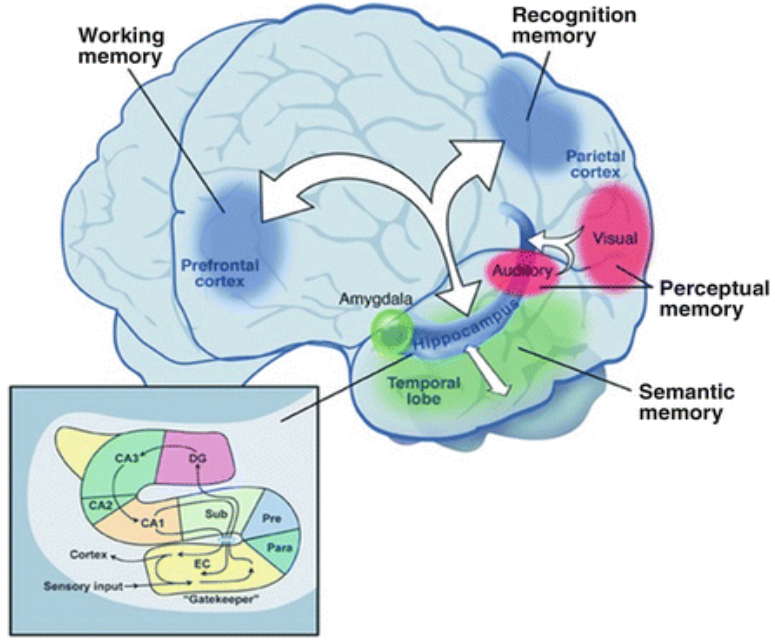
HGF/MET System is Critical to Normal Brain Function

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]



Adapted from *Hokama et al, Cerebral Cortex 2014*

Neuronal MET expression in AD:

- **75%** reduction in the hippocampus
- **25%** reduction in the frontal cortex

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

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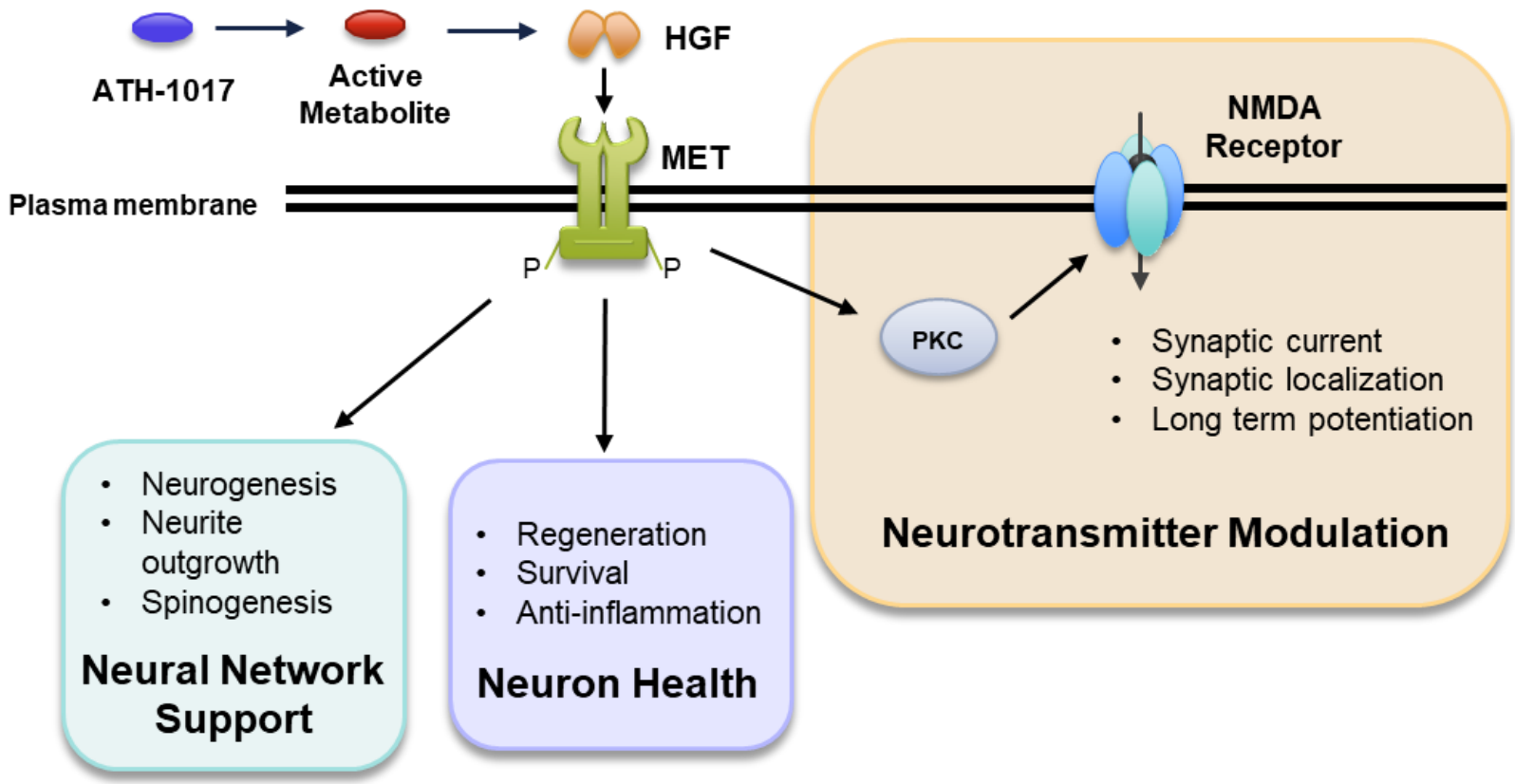
ATH-1017 is a Positive Modulator of the HGF/MET Neurotrophic System

ATH-1017:

- Administered via subcutaneous injection
- Is a small molecule prodrug that is immediately converted to an active metabolite in plasma
- Crosses the blood-brain barrier
- Positively modulates HGF/MET



Multimodal, protective, and regenerative

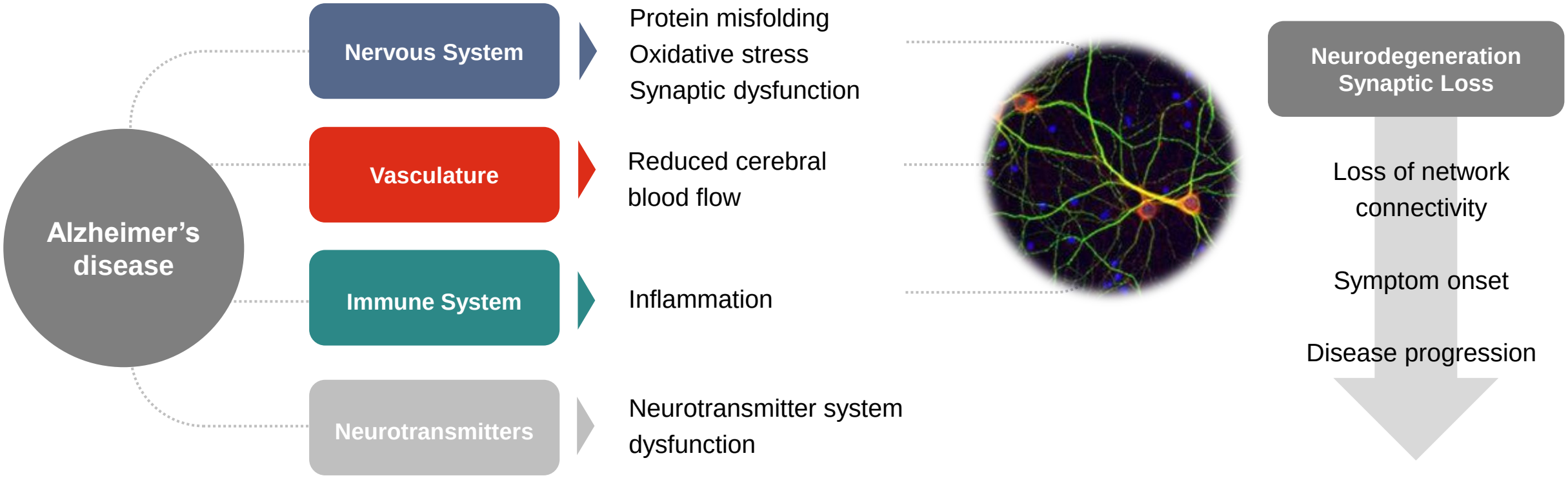
HGF/MET signaling and downstream effects described in:
Desole et al, Frontiers in Cell and Developmental Biology 2021
Funakoshi and Nakamura, Current Signal Transduction Therapy 2011

Lead Program

ATH-1017 – Dementia

Alzheimer’s Disease Pathology

Multifactorial and complex pathologies ultimately lead to neurodegeneration



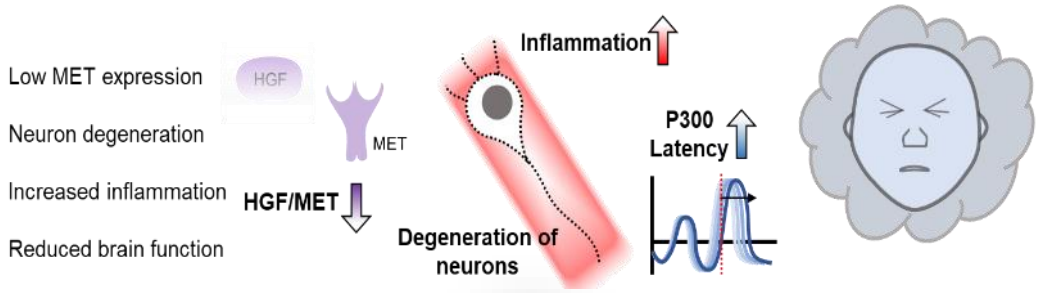
Clinical Trial Design to Assess Effects of Novel MOA

01 The unique MOA of pharmacologically promoting HGF/MET activity is expected to allow for rapid assessment of neurological effects

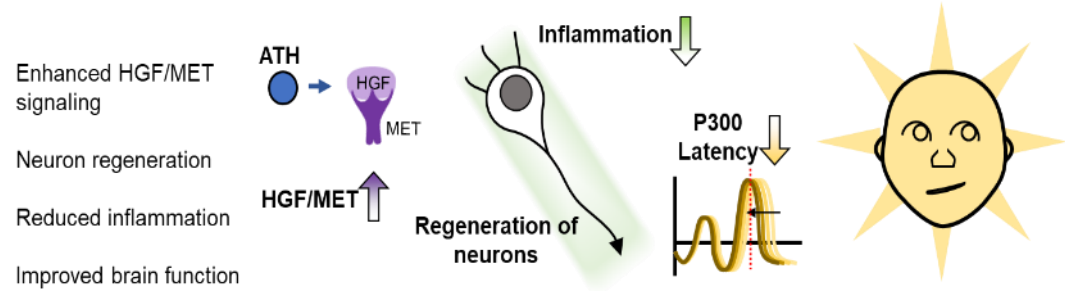
02 qEEG and ERP P300 are non-invasive methods that provide a direct measure of brain activity and are impacted in Alzheimer's disease. P300 is a neurophysiological measurement that refers to a spike in activity following presentation of the target stimulus

03 Including qEEG/ERP methods in early clinical trials supports rational dose finding, suggests CNS penetration early in development, and provides quantitative, functional read-outs of treatment effects

Alzheimer's Disease



ATH-1017 Therapy



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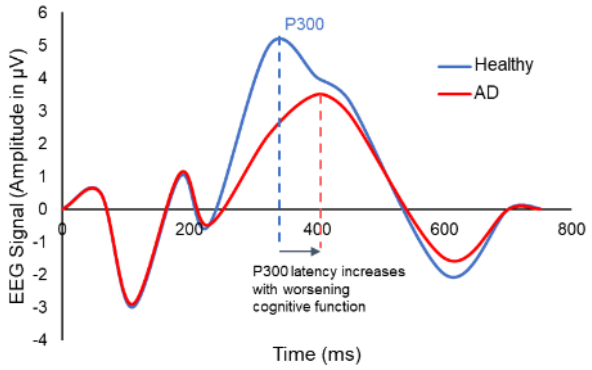
Clinical Development Plan Includes Measures Strongly Correlated with Cognition and Translatable Tools to Guide Dose Selection

EEG measures electrical activity from firing neurons in the brain

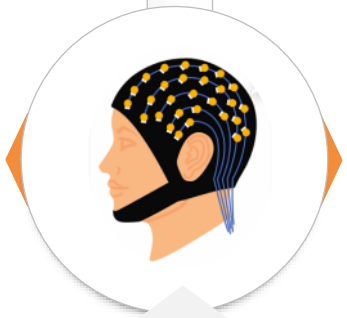
EVENT RELATED POTENTIALS (ERP): P300 Latency

Functional measurement for working memory access and executive function

Strongly correlated with memory improvement



Pathological changes in P300 latency correlate with cognitive impairment



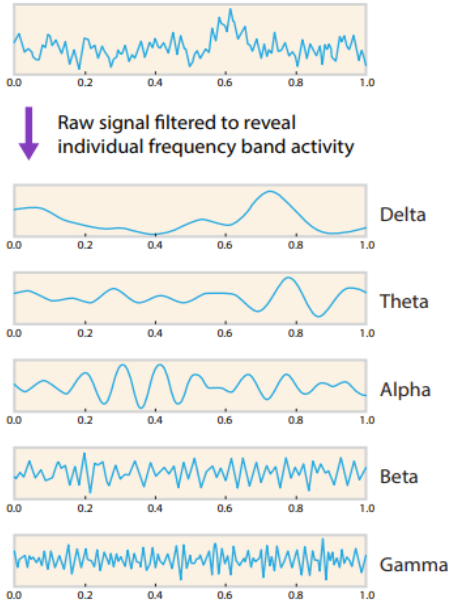
EEG records brain electrical activity from electrodes placed on the scalp

QUANTITATIVE EEG (qEEG)

Translational tool from rodents to humans

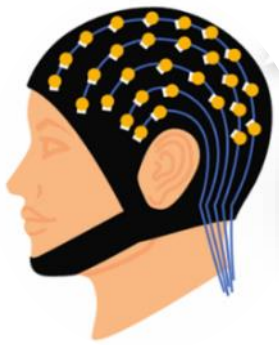
PK/PD modeling for dose selection

Noninvasive EEG recordings reflect brain activity and function

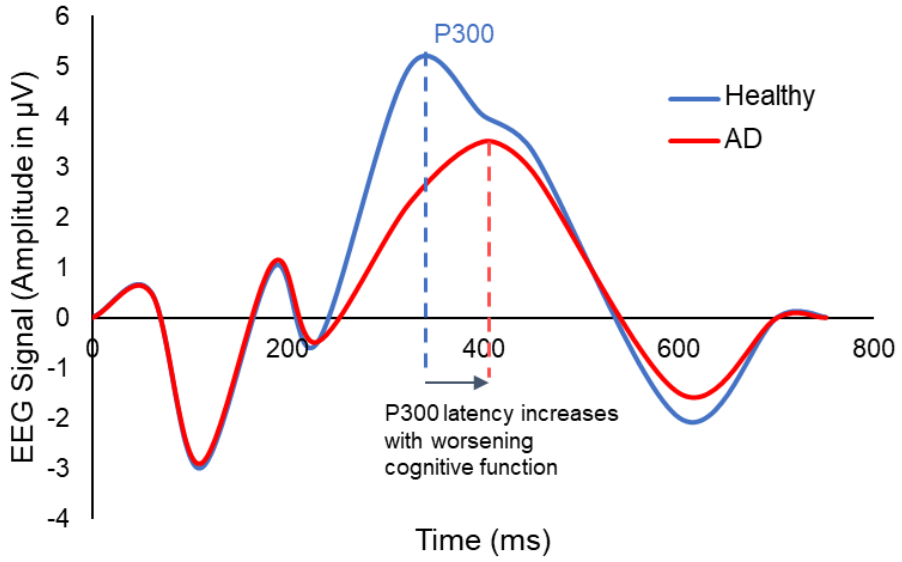


P300 Latency as a Functional Measure of Working Memory Processing Speed that Highly Correlates with Cognition

Pathological changes in P300 latency correlate with cognitive impairment



P300 latency is highly influenced by AD and other dementias



Recording brain activity while a subject is presented with a task reveals neural activity related to cognitive processing

Time to peak positive wave response (following an external stimulus) is ~300 ms

Approved therapies have demonstrated parallel improvement in P300 latency and cognition

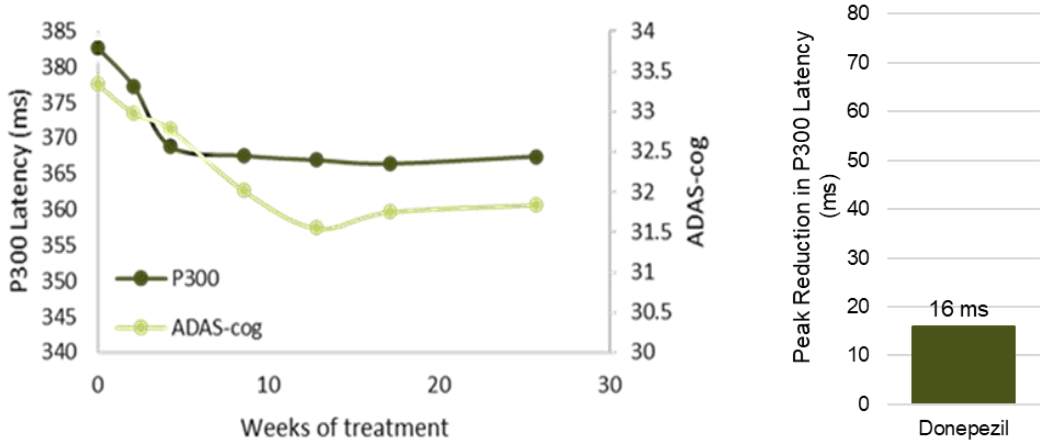
Athira utilized P300 Latency in its ATH-1017 Phase 1 clinical trial as a potentially predictive measure of cognition

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

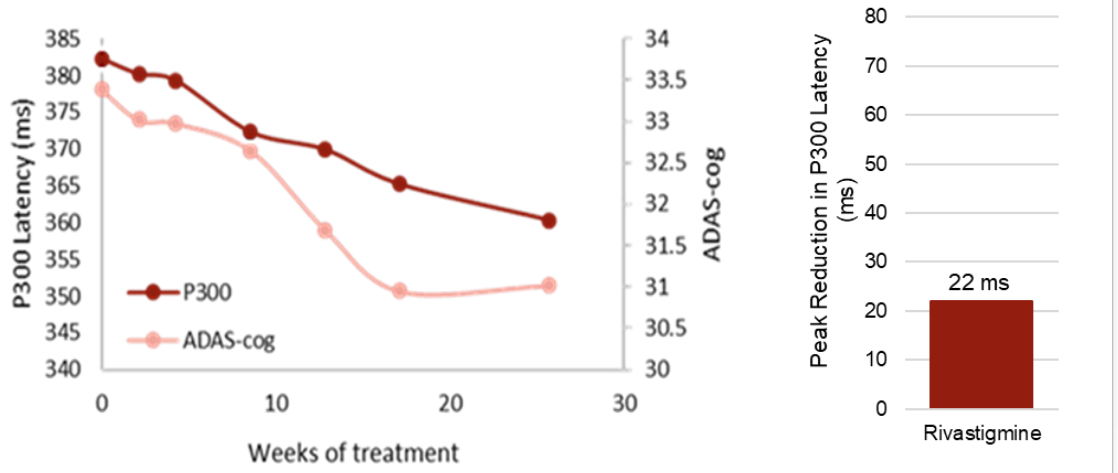
Previously published results support the correlation of P300 latency and cognition in AD subjects

- Donepezil and Rivastigmine
- Improvement in cognition (ADAS-cog ↓) is correlated with reduction in P300 latency

DONEPEZIL



RIVASTIGMINE



Note: Results from donepezil and rivastigmine adapted from *Thomas et al., 2001.*

ATH-1017 Treatment Improved P300 Latency in AD Subjects – CTAD 2019

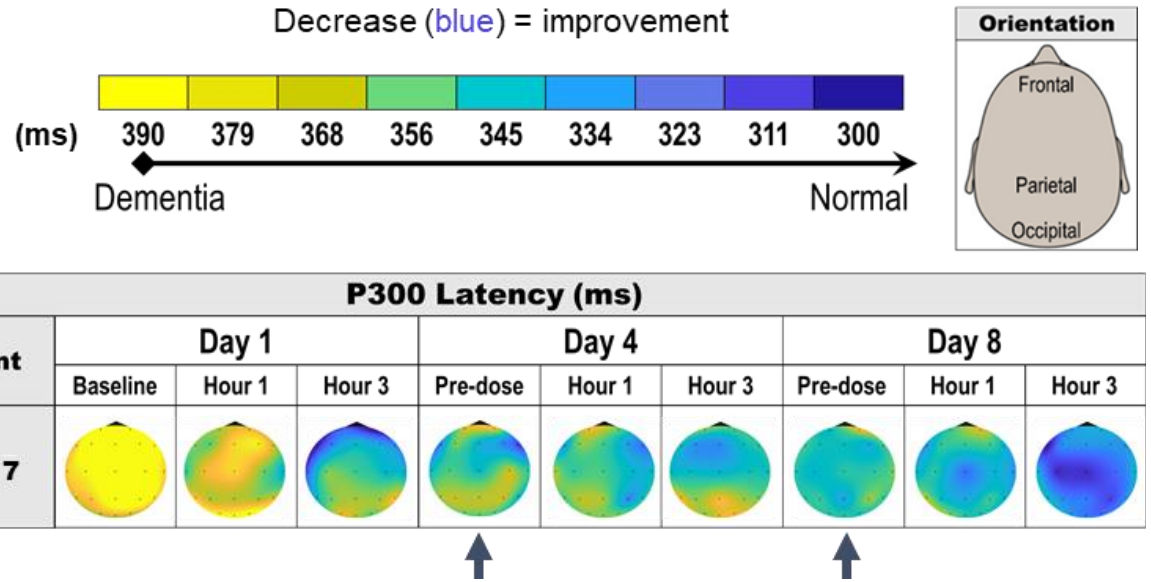
Phase 1b – AD Subjects

- **N=11**
- **Randomized, Placebo, ATH-1017 (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

ATH-1017 Treatment Improved P300 Latency in AD Subjects – CTAD 2019

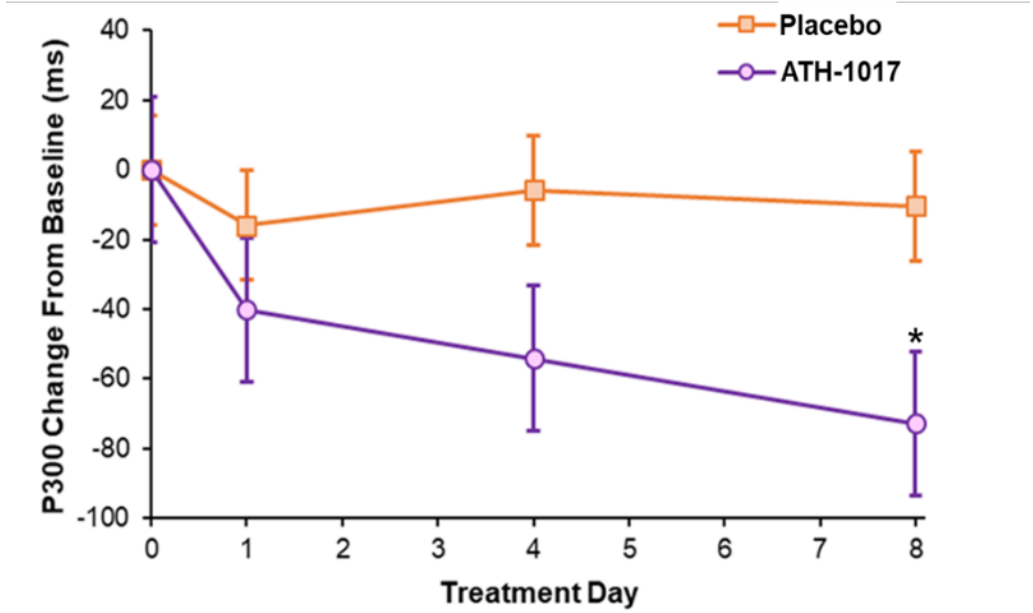
Phase 1b – AD Subjects

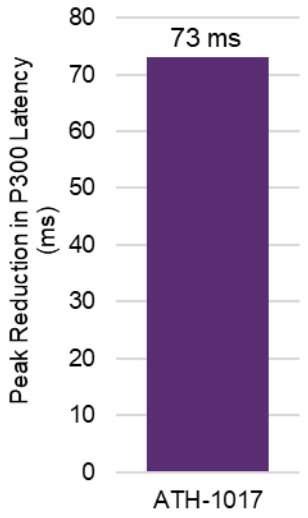
- Group averages of AD subjects receiving ATH-1017 (N=7) demonstrate decreased P300 over time

  Significant change from baseline observed on Day 8

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

P300 LATENCY: ACTIVE v PLACEBO TREATED AD SUBJECTS





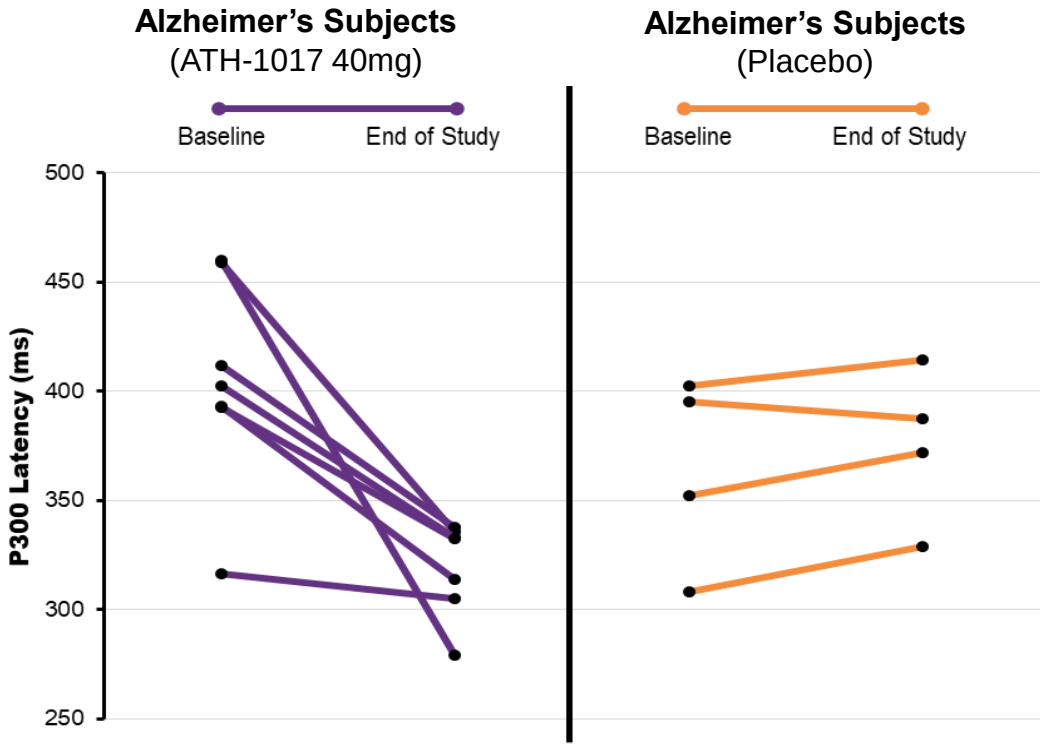
Note: P300 data from FZ, CZ, and PZ electrodes, Data plotted as mean +/- SE.. *p<0.05 with MMRM.

ATH-1017 Treatment Improved P300 Latency in AD Subjects – CTAD 2019

Phase 1b – AD Subjects

- Every AD subject receiving ATH-1017 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



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

Studies Suggest Changes in P300 Latency Have Been Predictive of Changes in Cognition

Treatment	P300 Latency Effect	Change in P300 Latency	Population	Cognitive Effect	Summary
Donepezil	Improved	(16) ms	AD	Improved	P300 latency and cognition both improved in mild-to-moderate AD, though improvements were modest.
Rivastigmine	Improved	(22) ms	AD	Improved	P300 latency and cognition both improved in mild-to-moderate AD, though improvements were modest.
Memantine	Improved	(15) ms	AD	Improved	P300 latency and cognition both improved in moderate to severe AD, though improvements were modest.
Scopolamine	Worsened	50 ms	Healthy	Worsened	Scopolamine offers a counter example, P300 latency increases while cognitive performance is reduced.

Note: Results from donepezil and rivastigmine adapted from *Thomas et al., 2001*; results from memantine adapted from *Sallach et al., 2011*; and results from scopolamine adapted from *Potter et al., 2000.*

ATH-1017 Phase 2/3 Trial (LIFT-AD)

Trial may provide pivotal evidence to support product registration

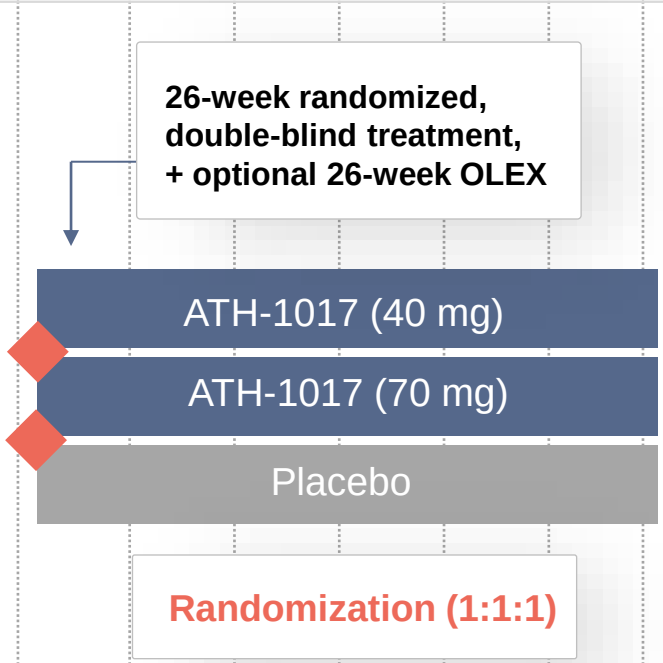
POPULATION

LIFT-AD: Target N=300 mild-to-moderate AD dementia subjects
(55-85 years; CDR 1 and 2; MMSE 14-24 incl.)

Potentially pivotal study design

- If both key secondaries are positive
- If key secondaries are positive GST will also be positive

TREATMENT DURATION



ENDPOINTS

PRIMARY ENDPOINT

- Global Statistical Test (GST, O'Brien 1984)
- Safety

SECONDARY ENDPOINTS WITH HIERARCHY

1. Cognition (key secondary): ADAS-Cog11
2. Global (key secondary): ADCS CGIC - Clinician
3. Function (key secondary for ex-US): Alzheimer's Disease Cooperative Study - Activities of Daily Living (ADCS-ADL23)

INTEGRAL GST PROVIDES PRIMARY READ OUT

GST – unbiased composite, fed by data from two key secondaries

ATH-1017 Phase 2 Trial (ACT-AD)

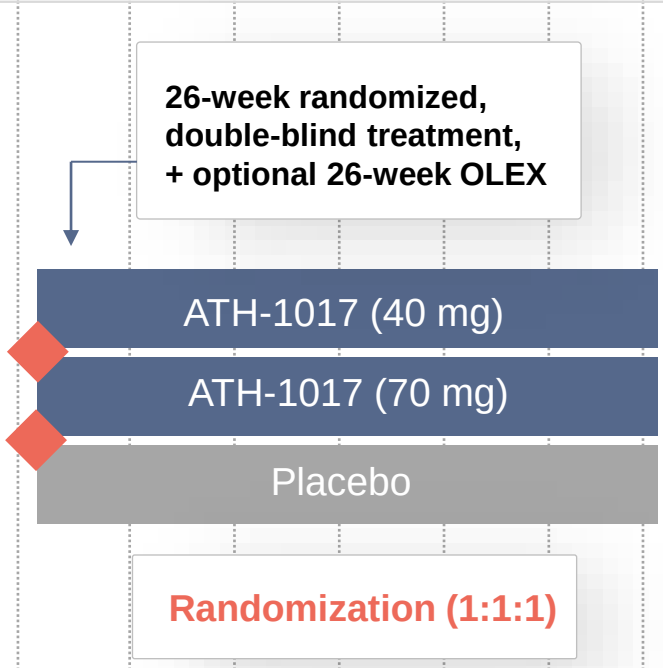
Proof of concept trial to help better understand nature of novel intervention and refine LIFT-AD

POPULATION

ACT-AD: N=77 (rec. completed) mild-to-moderate AD dementia subjects
(55-85 years; CDR 1 and 2; MMSE 14-24 incl.)

- Enables readout 1H2022, ahead of LIFT-AD
- Informs LIFT SAP and enables earlier strategic decisions

TREATMENT DURATION



ENDPOINTS

PRIMARY ENDPOINT

- Change of P300 latency
- Safety

SECONDARY ENDPOINTS WITHOUT HIERARCHY

1. Global Statistical Test (GST, O'Brien 1984)
2. Cognition: ADAS-Cog11
3. Global clinical change: ADCS CGIC - Clinician
4. Function: ADCS-ADL23

ATH-1017 Program Summary

- Initiation of LIFT-AD trial in September 2020 and ACT-AD trial in November 2020
- Completed nonclinical GLP long term toxicology and safety pharmacology studies
- In Phase 1a/b trial (N=88), ATH-1017 was generally well-tolerated with no serious adverse events (SAEs)
- PK/PD modeling defined active dose range for potentially pivotal LIFT-AD trial and ACT-AD trial
- Data showing ERP P300, a functional measure that is highly correlated with cognition, improved with treatment ($p<0.05$)

ATH-1020

Neuropsychiatric Indications

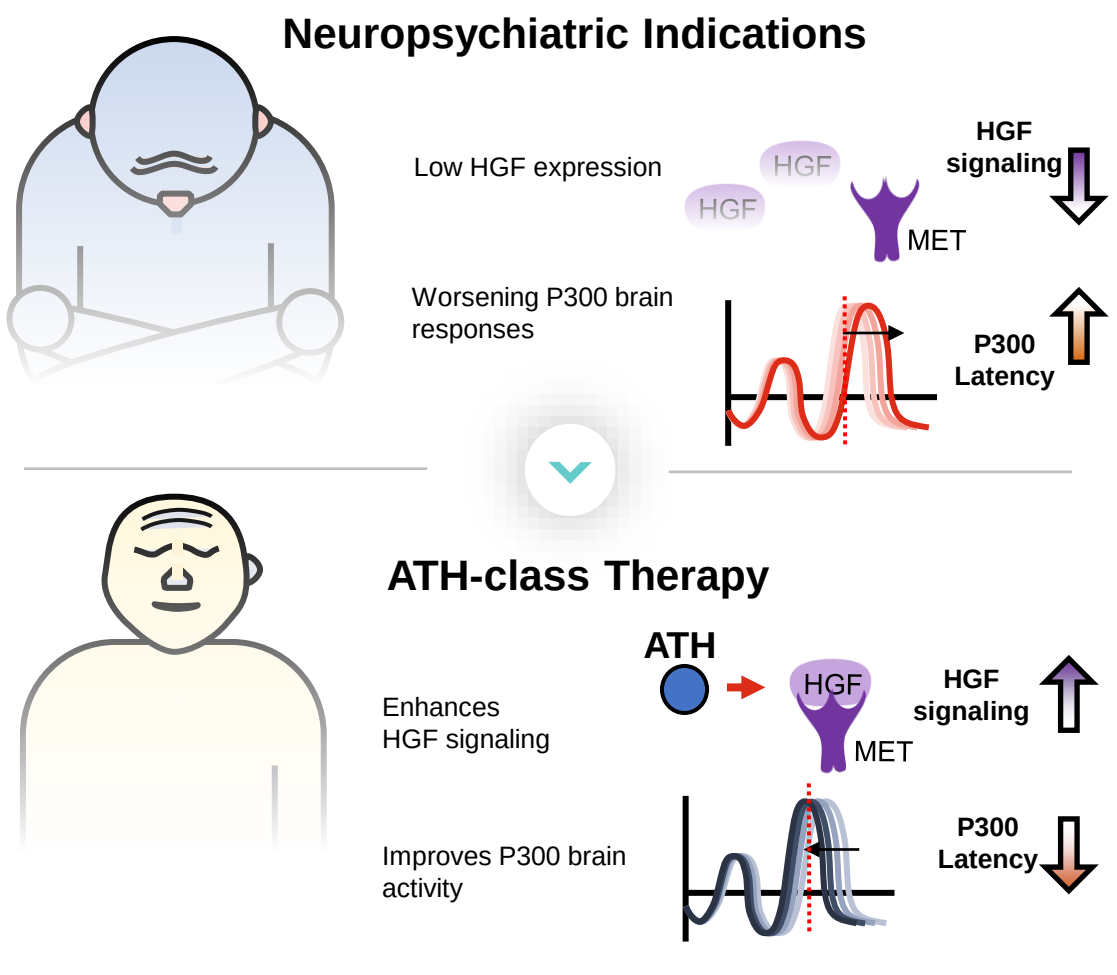
Rationale for Targeting HGF/MET for Neuropsychiatric Indications

01 Preclinical studies demonstrate enhancing HGF/MET activity has anti-depressant and anxiolytic effects in rodents[1]

02 Clinical trials show an association between reduced HGF/MET expression levels and depression/anxiety[2]

03 Neuropsychiatric patients often exhibit worsened P300 latency, a marker of impaired cognitive processing[3]

04 ATH activity has the potential to rescue HGF/MET signaling, promote neuronal health and function, and restore P300 latency and cognitive function



1. Isogawa et al., 2005; Wakatsuki et al., 2007
2. Russo, 2010; Ciuculete et al., 2019; Ramsey et al., 2016
3. Vandoolaeghe et al., 1998

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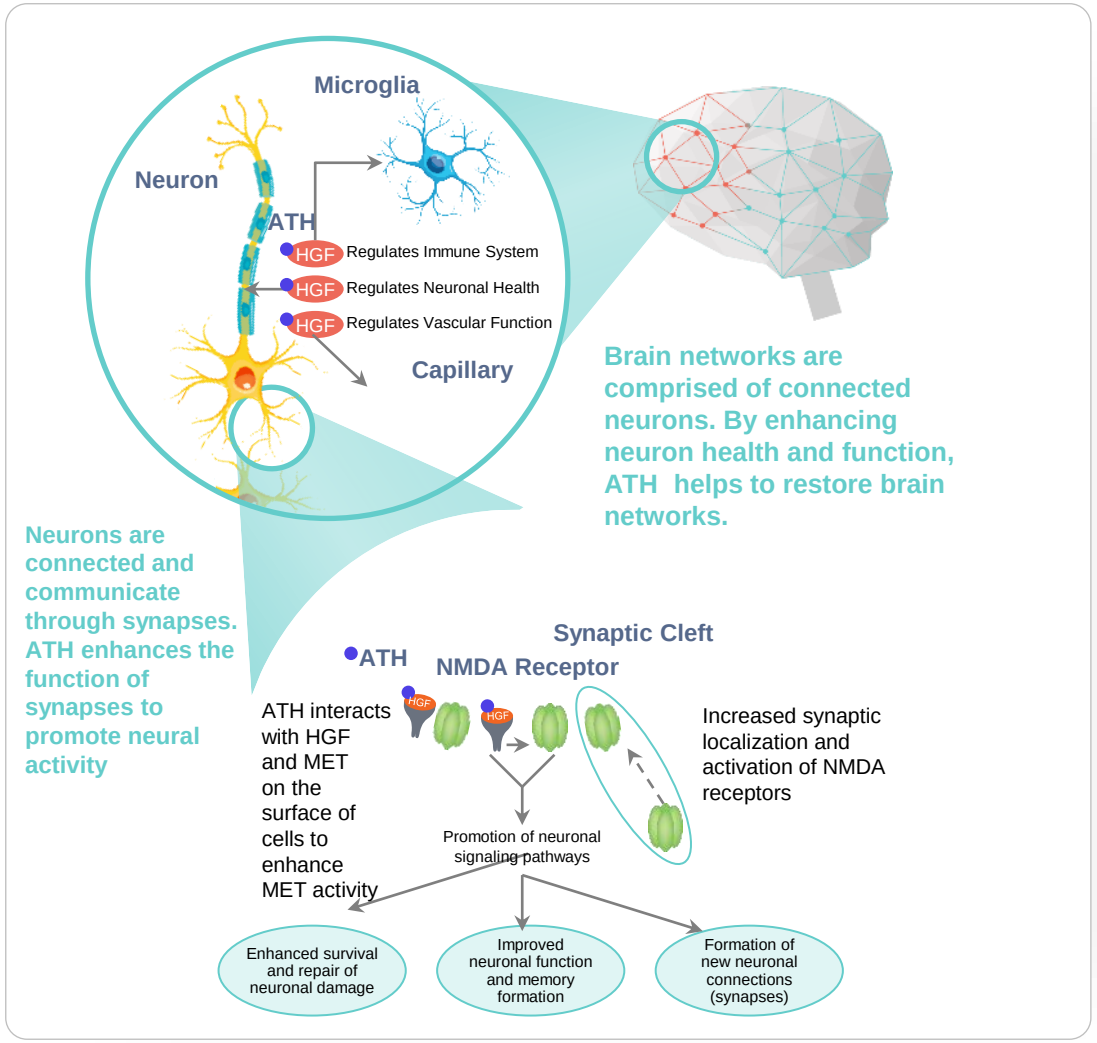
ATH Small Molecules Enhance HGF/MET Activity

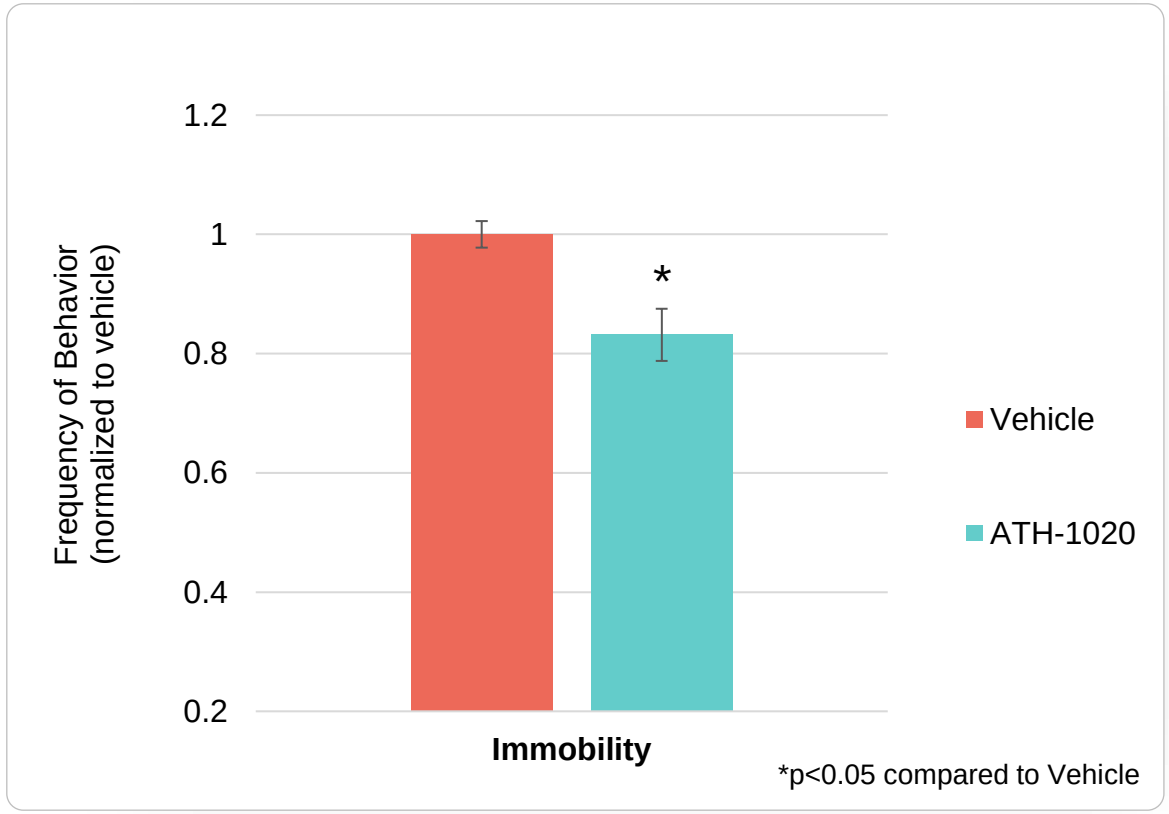
ATH-1020 is in development as a potential candidate for neuropsychiatric indications, including depression, anxiety, and potentially schizophrenia

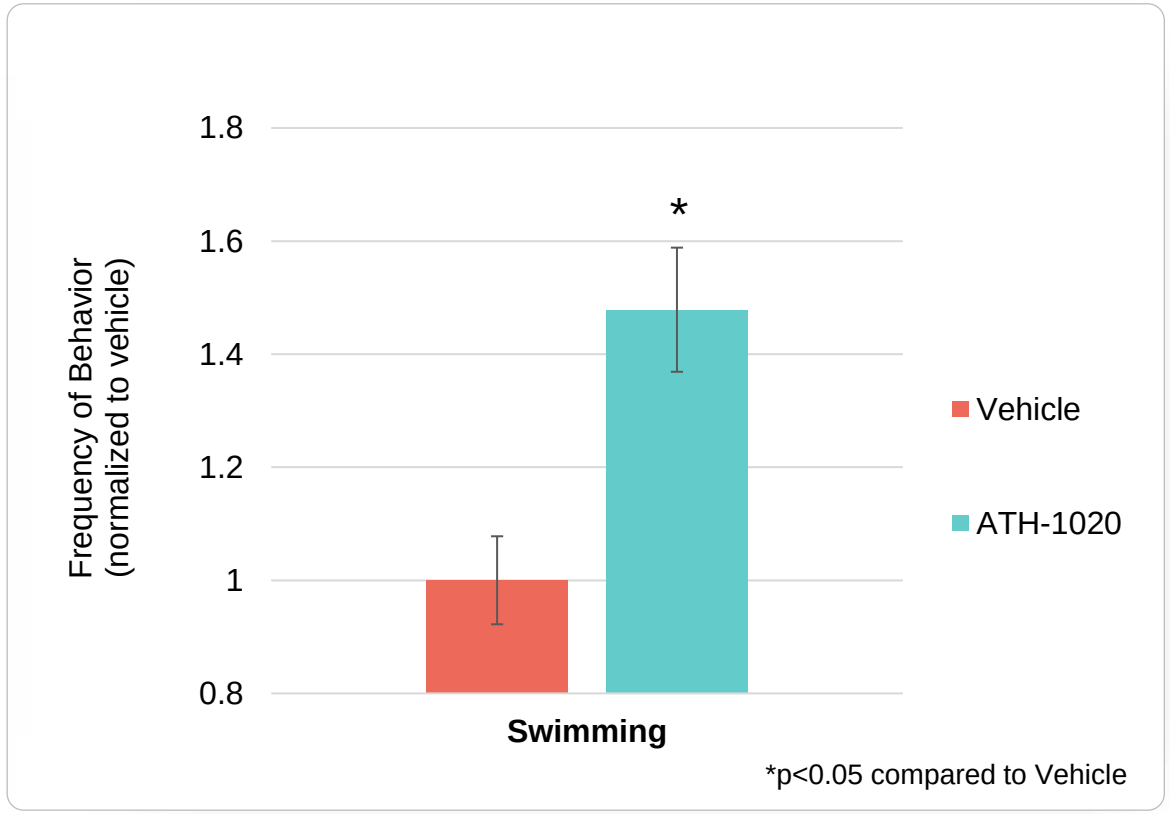
- Positively modulate HGF/MET with the potential to promote nerve cell health in several indications
- Orally bioavailable
- Distribute to the brain
- Neuroactive in animal models
  - Procognitive effects in scopolamine-induced amnesia model of dementia in rats
  - Anti-depressant activity in forced swim test in rats



ATH-1020 Demonstrated Anti-Depressant Effects in Animal Models

Treatment with ATH-1020 (oral delivery) led to significant improvement in depressive-like behaviors in the rat forced swim model of depression.





ATH-1020, First-in-Class for Neuropsychiatric Indications

INDICATIONS

Neuropsychiatric disorders

DELIVERY MODE

Oral

MOA

Small molecule agonist of HGF/MET

REGIMEN

Targeting once per day

Anticipated Upcoming Milestones

- ACT-AD: Top-line data by first half 2022
- LIFT-AD: Top-line data by the end of 2022
- SHAPE for Parkinson's disease dementia and dementia with Lewy bodies: Initiate by end of 2021
- ATH-1020: IND filing by end of 2021

Financial Position Summary

Strong Capital Position

Cash, cash equivalents and marketable securities were $339.4 million as of September 30, 2021 compared to $268.2 million as of December 31, 2020.

Athira Management Team with Significant CNS Product Development and Approval Experience

EXECUTIVE LEADERSHIP

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Kronos Bio

Investment Highlights

Novel approach to rapidly improve cognition

Leveraging a critical repair pathway, HGF/MET, and naturally occurring repair mechanisms that are agnostic to underlying disease pathology

Lead Asset ATH-1017

Lead indication is in potentially pivotal trial

Encouraging Phase 1 clinical data in Alzheimer's disease

- LIFT-AD trial actively recruiting, topline data by late 2022
- ACT-AD trial topline data by first half 2022

Efficient clinical development strategy

Clinical development strategy to investigate the potential for fast onset with tangible cognitive improvement

Potential follow-on indications with established regulatory pathway and faster timelines

Pipeline of novel, small molecule compounds

Opportunity to explore several neurological indications

- CNS degenerative, PNS disorders, neuropsychiatric indications

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