Athira PHARMA

Corporate Presentation

MAY 2023

ADVANCING NEW THERAPIES FOR NEURONAL HEALTH

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The ACT-AD trial and the related open-label extension for ACT-AD participants 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 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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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 a naturally occurring mechanism to protect and repair neuronal networks

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

- Therapeutic potential in Alzheimer's, Parkinson's, ALS, neuropathic pain, etc.

Late-stage clinical development ongoing with fosgonimeton without concomitant AChEI in Alzheimer's disease

- Exploratory ACT-AD trial showed congruent improvements in biomarkers of neurodegeneration, inflammation and Alzheimer's protein pathologies as well as measures of cognition and function
- Mitigated development risk of lead program fosgonimeton for Alzheimer's following independent unblinded interim analysis of Phase 2/3 LIFT-AD trial
- Well-tolerated with a favorable safety profile

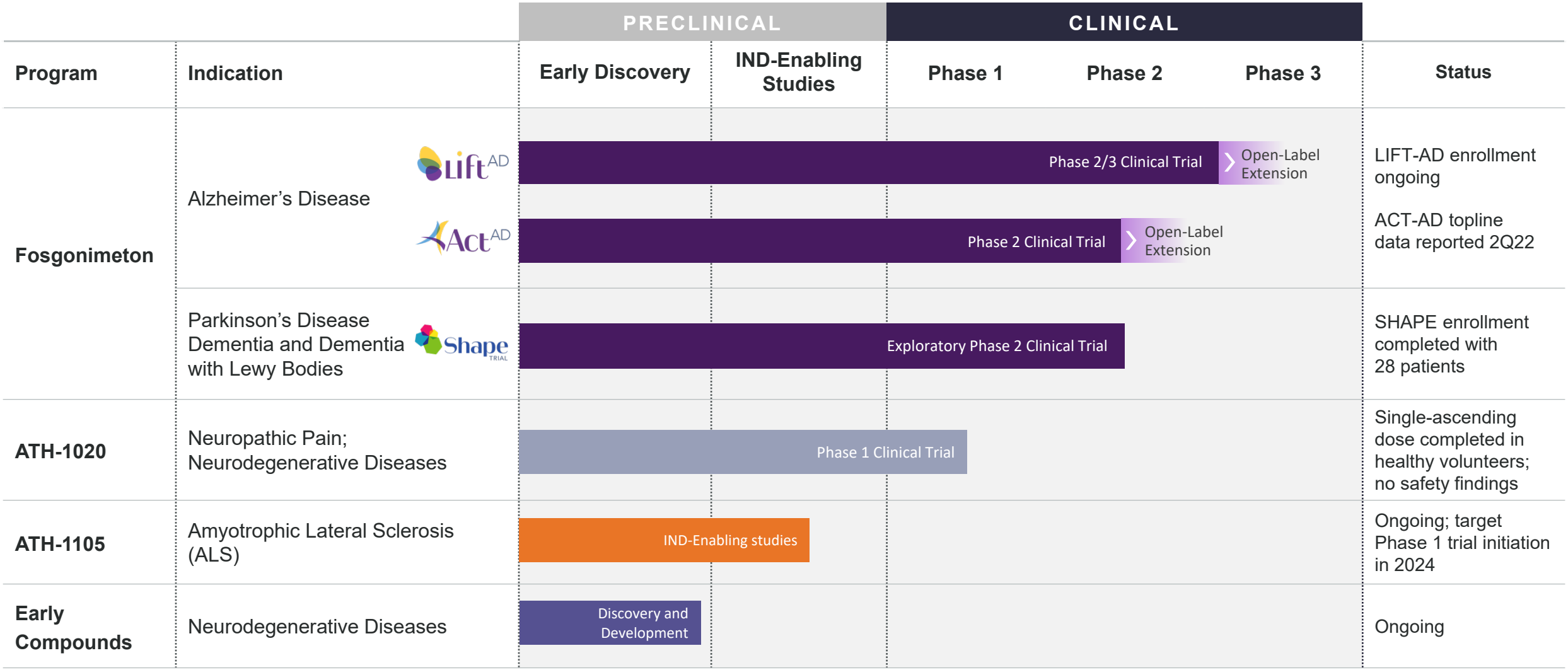
Robust pipeline of proprietary small molecules targeting HGF/MET

Strong balance sheet to support clinical programs through key inflection points

Leadership team with significant CNS product development and approval experience

AChEI, acetylcholinesterase inhibitor; AD, Alzheimer's disease; ALS, amyotrophic lateral sclerosis; CNS, central nervous system; DMC, data monitoring committee; HGF, hepatocyte growth factor.

Therapeutic potential across a broad range of clinical indications



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

Multifactorial complex pathologies lead to neurodegeneration

CAUSES OF NEURODEGENERATIVE DISEASES

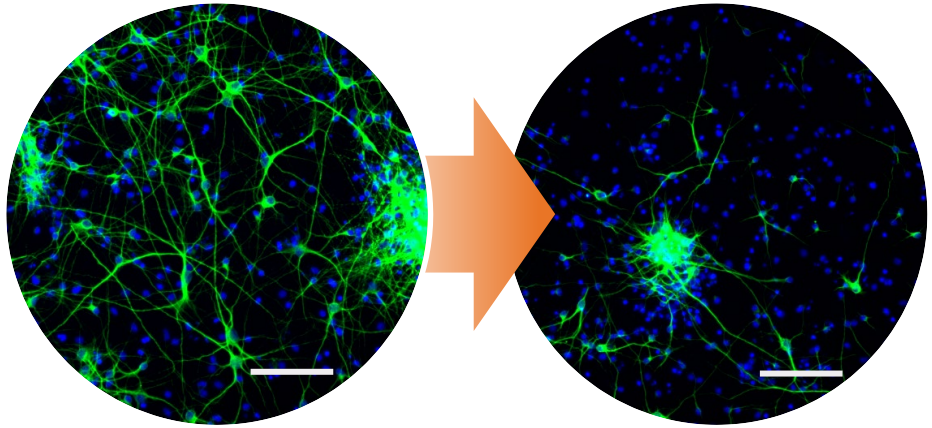
Inflammation

Oxidative stress

Protein pathology

Excitotoxicity

Synaptic dysfunction



HEALTHY **DISEASED**

= Neuron Marker

HALLMARKS OF NEURODEGENERATION

Neuronal damage

Loss of network connectivity

Loss of function

Disease progression

Images are of primary cortical neuron cultures, showing healthy (untreated) cultures on the left, and cultures treated with glutamate (20 µM) on the right, demonstrating glutamate-mediated neuronal death and neurite degeneration.
Scale bar: 100 µm.
Neuron Marker=MAP2, microtubule associated protein 2

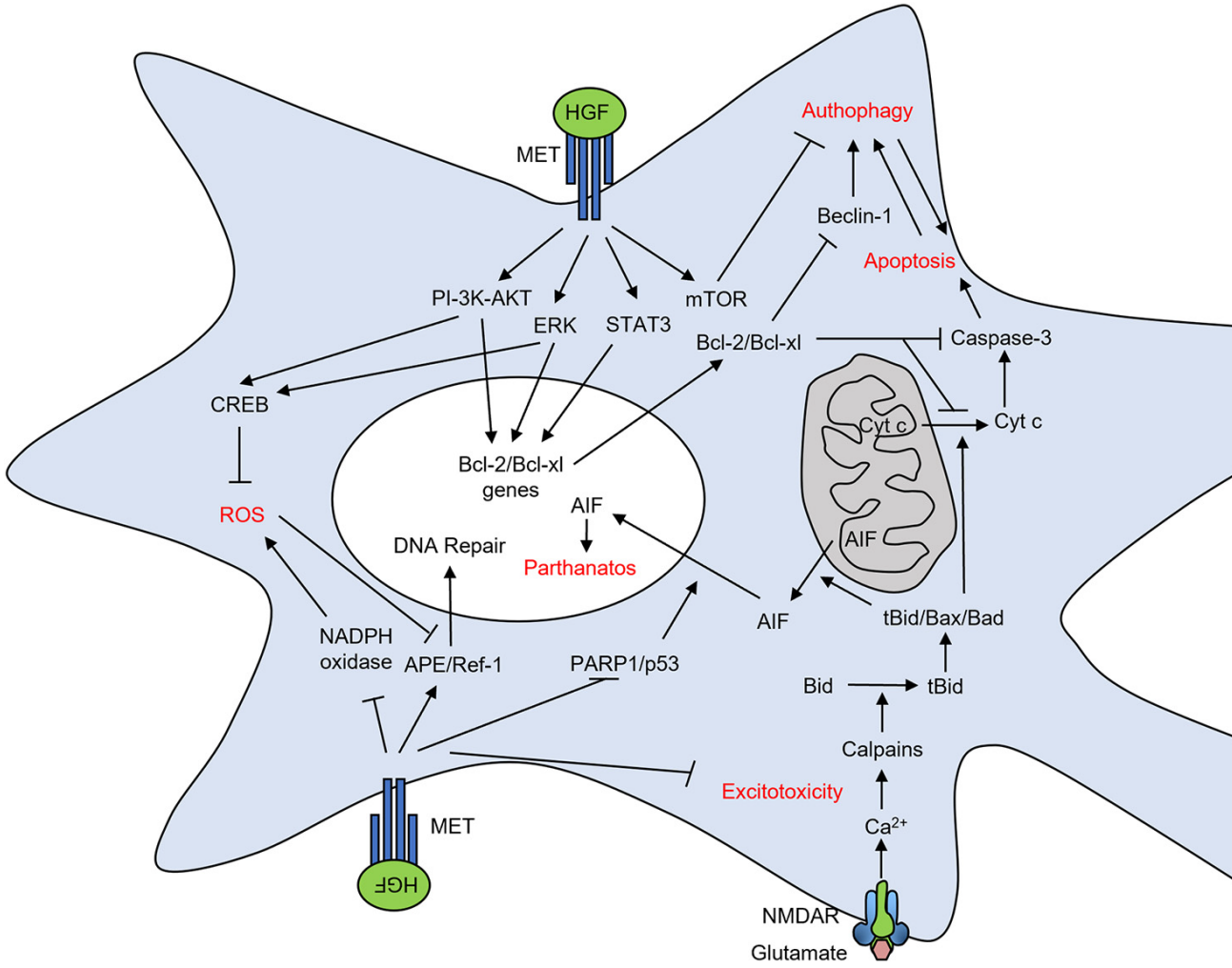
HGF/MET is a critical neuroprotective system

THE ROLE OF HGF/MET IN THE NERVOUS SYSTEM HAS BEEN EXPLORED THROUGH 30 YEARS OF RESEARCH

HGF/MET activates signaling pathways to protect neurons from:

- Oxidative stress
- Excitotoxicity
- Apoptosis

Adapted from Desole et al, 2021



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Desole et al., *Frontiers in Cell and Developmental Biology* 2021.
HGF, hepatocyte growth factor.

Positive modulators of the HGF/MET neurotrophic system

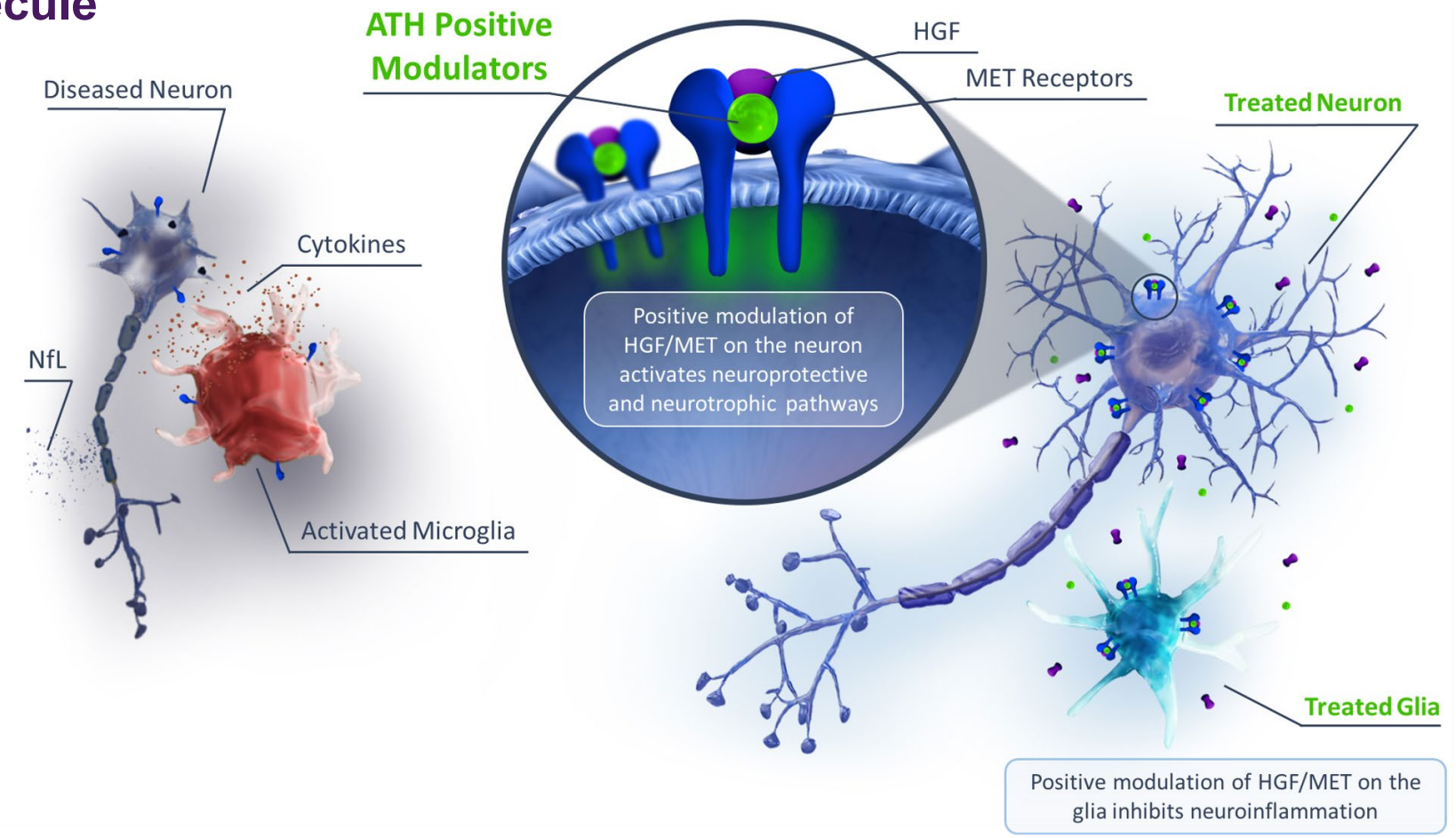
MULTIMODAL, PROTECTIVE, REGENERATIVE, DISEASE MODIFYING

Potential first-in-class small molecule drug candidates

- Able to cross the blood-brain barrier
- Positively modulate HGF/MET

Mechanism of action may

- Reduce inflammation
- Promote regeneration
- Provide neuroprotection
- Modify the course of disease



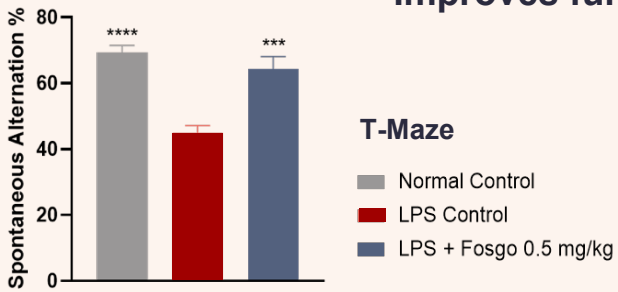
HGF, hepatocyte growth factor.

Fosgonimeton protects and repairs neuronal networks in preclinical models

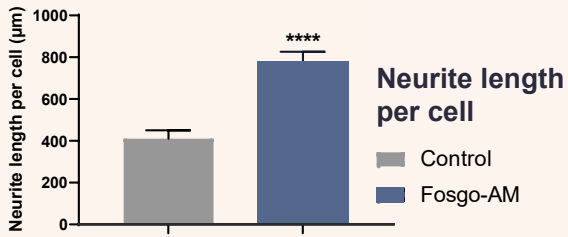
MULTIMODAL APPROACH FOR MULTIMODAL DISEASES WITH POTENTIAL FOR DISEASE MODIFICATION

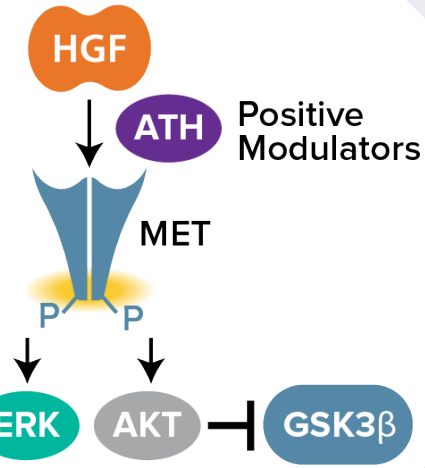
REPAIR | **PROTECT**

Improves function

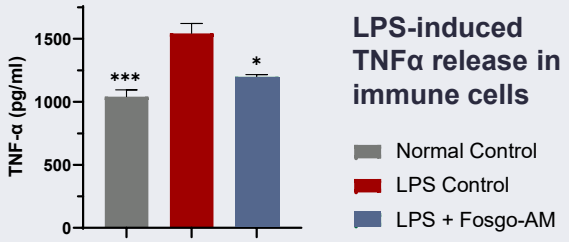


Neurotrophic

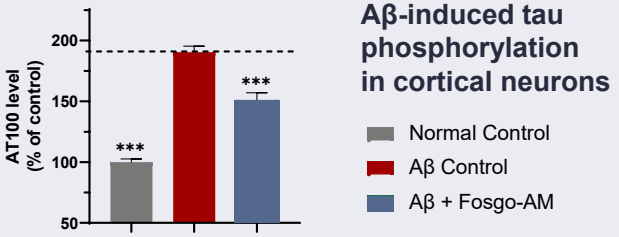




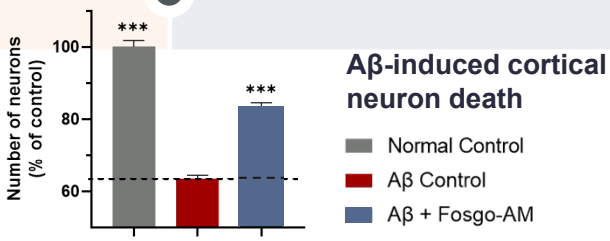
Anti-inflammatory



Reduces protein pathology



Neuroprotective



[1]Johnston et al., *Neurotherapeutics* 2022, Berthiaume, et al., *MNDA* 2022, Reda et al., *AAIC* 2022, Setti et al., *SfN* 2022.

Aβ, amyloid beta; AKT, protein kinase B; ERK, extracellular-signal regulated kinase; fosgo-AM, fosgonimeton active metabolite; GSK3b, glycogen synthase kinase-3 beta; HGF, hepatocyte growth factor; LPS, lipopolysaccharide; TNF-α, tumor necrosis factor alpha.

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Fosgonimeton exploratory phase 2 trial in mild-to-moderate Alzheimer's disease

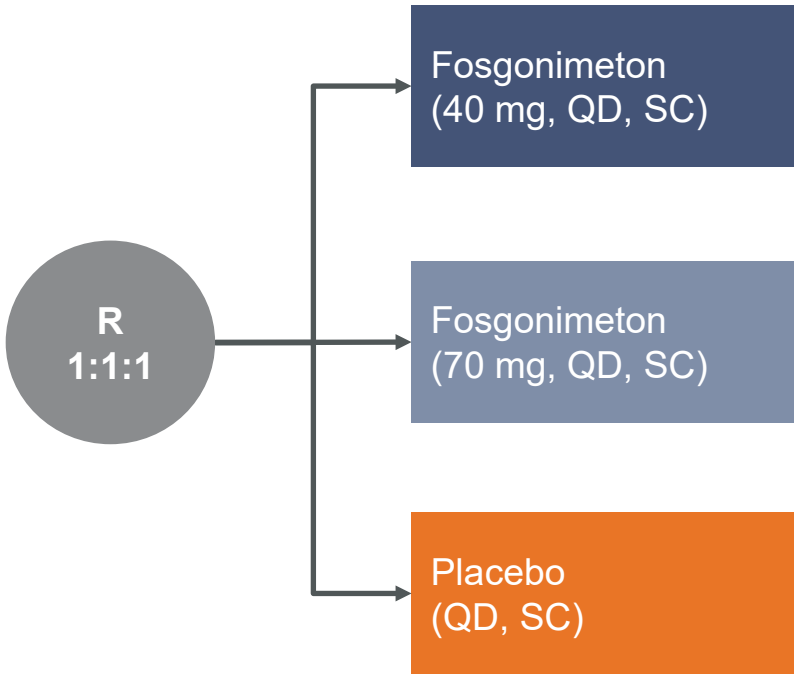
RANDOMIZED PLACEBO-CONTROLLED SIX-MONTH TRIAL (N=77)

Key Inclusion Criteria

- 55–85 years
- MMSE score: 14-24
- CDR global score 1 or 2
- Clinical diagnosis of dementia, probably due to AD[1]
- Could receive prior or concomitant AChEI
- Reliable and capable support person/caregiver

Patient Population
Mean Baseline Characteristics

Age, years:	71.4
MMSE	19.3
Concomitant AChEI, %:	61.0
APOE4 carrier status	
Heterozygous, %:	36.4
Homozygous, %:	19.5
Plasma biomarkers	
Aβ 42/40 ratio:	0.06
p-Tau181, pg/mL:	3.83
NfL, pg/mL:	20.92



Endpoints
Primary: Change in ERP P300 latency (not met), safety and tolerability
Secondary: ADAS-Cog11, ADCS-CGIC, ADCS-ADL23
Exploratory: Plasma biomarkers

Key Learnings from ACT-AD

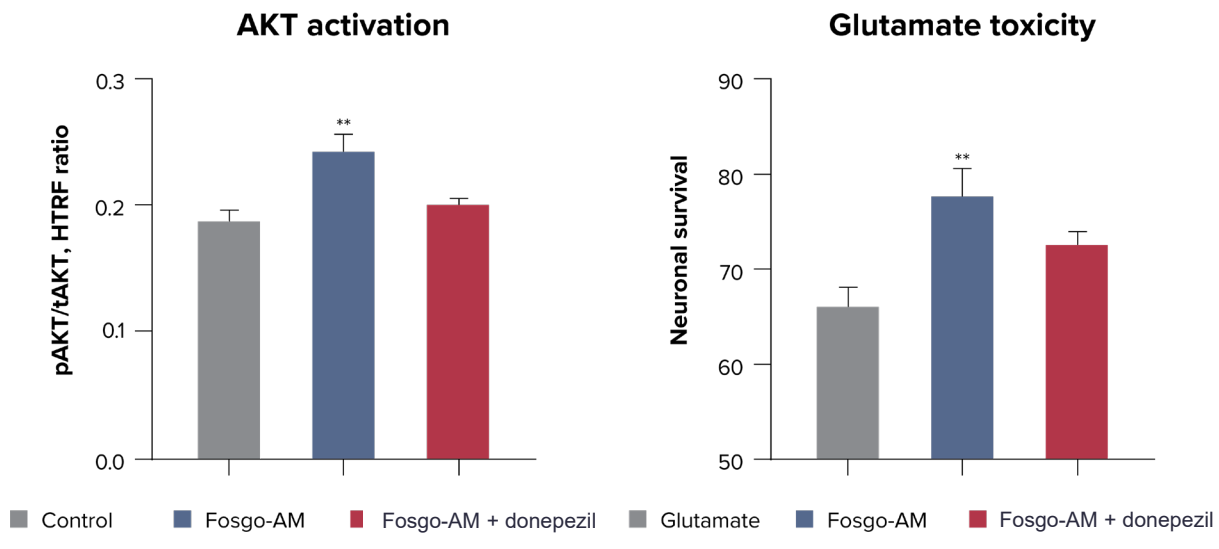
- Fosgonimeton was well-tolerated with a favorable safety profile
- Unexpected potential pharmacodynamic interaction with AChEIs
- Greater than 85% enrollment into OLEX from both ACT-AD and LIFT-AD studies
- Congruent clinical effects and biological signals

[1]McKhann GM et al., *Alzheimers Dementia* 2011.
Aβ, amyloid beta; 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; APOE, apolipoprotein E; CDR, clinical dementia rating; ERP, event-related potential; MMSE, mini-mental state examination; NfL, neurofilament light chain; OLEX, open-label extension; p-Tau181, tau phosphorylated at threonine-181; QD, once daily; R, randomization; SC, subcutaneous; SD, standard deviation.

Neuroprotective effects of fosgonimeton are reduced with exposure to AChEIs

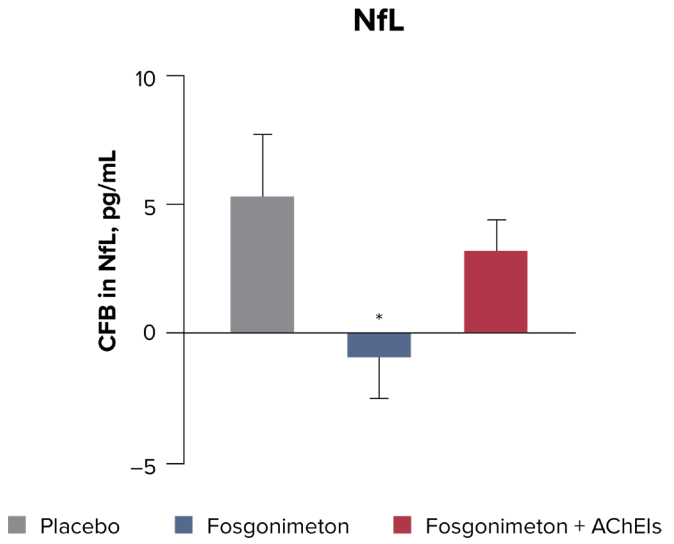
Preclinical

- Combination of fosgo-AM with donepezil interferes with fosgo-AM-induced AKT activation
- Neuroprotective effects of fosgo-AM are reduced when combined with donepezil
  - Likely the result of observed decrease in fosgo-AM-induced AKT activation



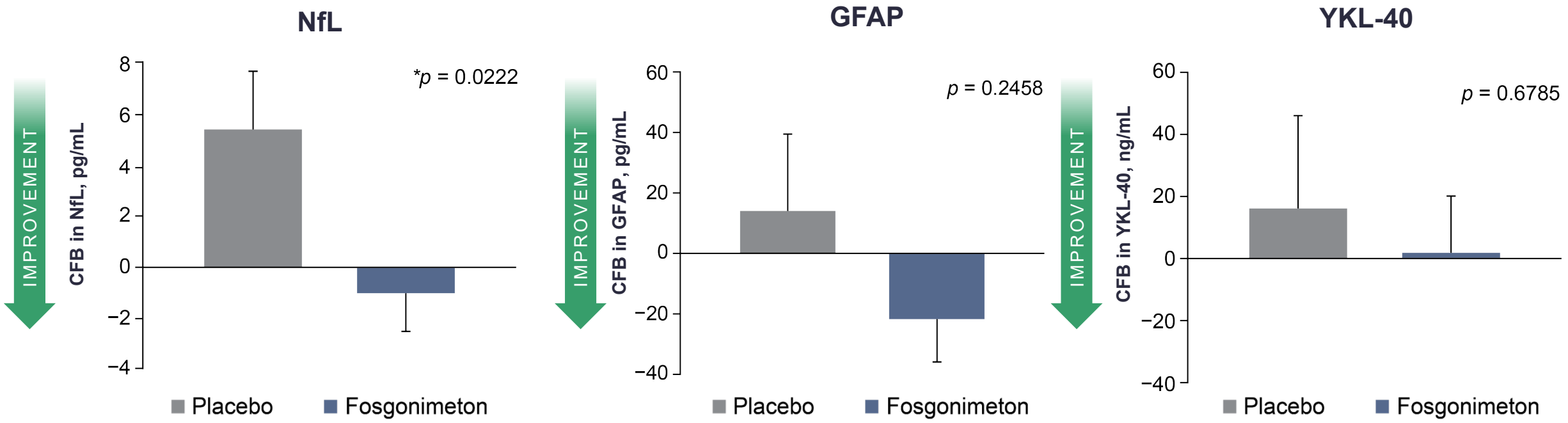
Clinical

- Fosgonimeton significantly reduced NfL, a biomarker of neurodegeneration, in the ACT-AD study
- When combined with AChEIs, NfL increased, suggesting a loss of the neuroprotective effect
- This result is consistent with preclinical findings of reduced neuroprotection in combination with AChEIs



AChEIs, acetylcholinesterase inhibitors; AKT, protein kinase B; ANOVA, analysis of variance; CFB, change from baseline; NfL, neurofilament light chain; SEM, standard error of the mean.
For in vitro assays, fosgonimeton was administered as the active metabolite, fosgo-AM. AKT assay: one-way ANOVA with Dunnett's post-test; **p<0.01 vs. control; n = 6 (control), 5 (fosgo-AM), 3 (fosgo-AM + AChEIs), mean + SEM. Glutamate assay: one-way ANOVA with Dunnett's post-test; **p<0.01 vs. glutamate; n = 6 (glutamate), 6 (fosgo-AM), 5 (fosgo-AM + AChEIs); mean + SEM.
For clinical data, NfL data are least squares means from an ANOVA model with CFB as the dependent variable with the following in the model: treatment group, AChEI use, baseline biomarker value, and the interaction of treatment and AChEI use. Error bars are ± SE. n = 5 (placebo); n= 12 (fosgonimeton − AChEIs), n = 22 (fosgonimeton + AChEI).

Neuroprotective and anti-inflammatory: Fosgonimeton reduces markers of neurodegeneration and inflammation



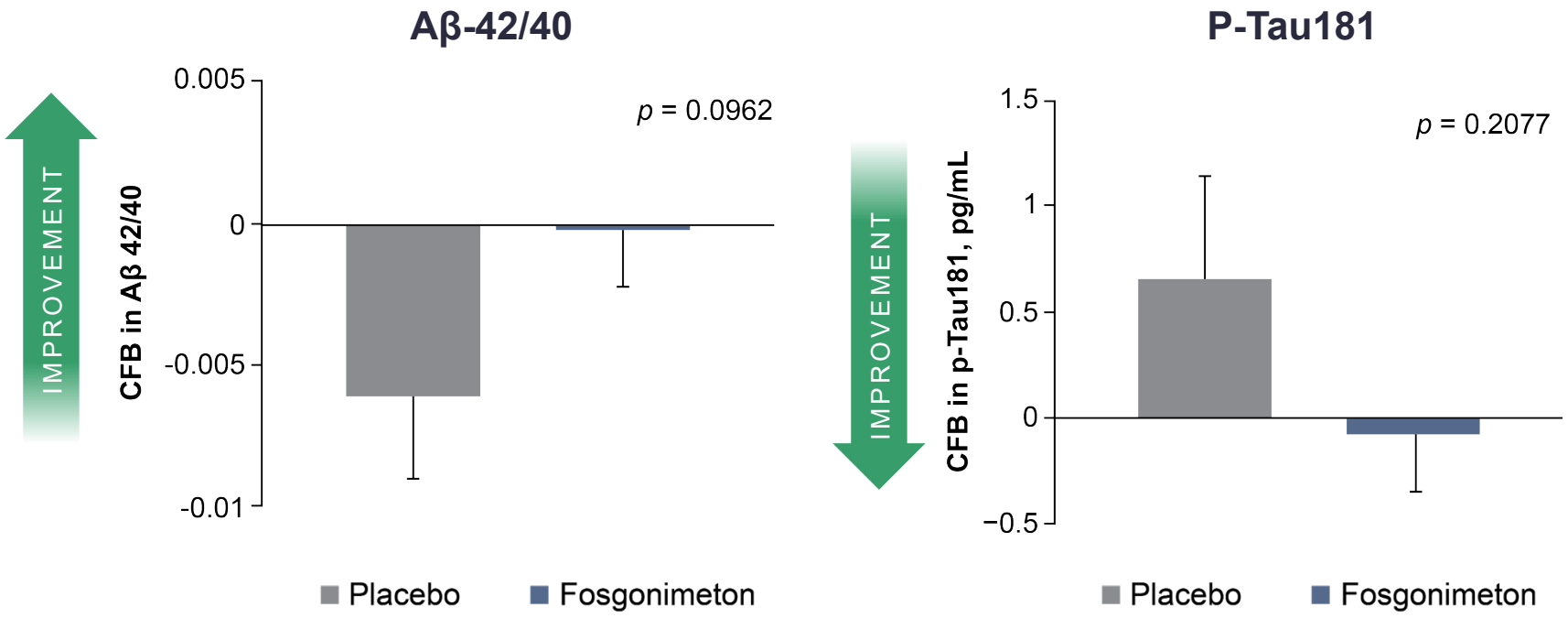
- Neurofilament light (NfL) is an established, objective biomarker of neurodegeneration
- Decrease of NfL to below baseline levels suggestive of repair in this continuously progressive disease
- GFAP and YKL-40 are markers of neuroinflammation
- Magnitude of decrease is encouraging in this continuously progressive condition

Data are least square means from an ANOVA model with change from baselines as the dependent variable with the following in the model: treatment group, AChEI use, baseline biomarker value and the interaction of treatment and AChEI use. Error bars are ± SE. N=5 for placebo treatment; N=12 for fosgonimeton treatment without concomitant AChEI.

CFB, change from baseline; GFAP, glial fibrillary acidic protein; NfL, neurofilament light chain; SE, standard error; YKL-40, chitinase-3–like protein 1.

Protein pathology: Fosgonimeton induces directional improvements in hallmarks of Alzheimer's disease

- Decreased Aβ 42/40 ratio and increased absolute p-Tau values are hallmarks of Alzheimer's disease
- Changes support relevance of the HGF/MET pathway also to Alzheimer's-specific protein pathology
- Supports disease modifying potential of fosgonimeton



Data are least square means from an ANOVA model with change from baselines as the dependent variable with the following in the model: treatment group, AChEI use, baseline biomarker value and the interaction of treatment and AChEI use. Error bars are ± SE. N=5 for placebo treatment; N=11 or 12 for fosgonimeton treatment without concomitant AChEI.

Aβ, amyloid beta; CFB, change from baseline; HGF, hepatocyte growth factor; p-Tau181, tau phosphorylated at threonine-181; SE, standard error.

Functional improvements: Potential benefits in cognition and function from fosgonimeton treatment

SUPPORTS POTENTIAL TO BE A SAFE AND DIFFERENTIATED FUTURE THERAPY

74% | **-3.0** points n.s.

IMPROVED COGNITION

Improvement over placebo over 6 months as measured by ADAS-Cog11 in patients without background therapy

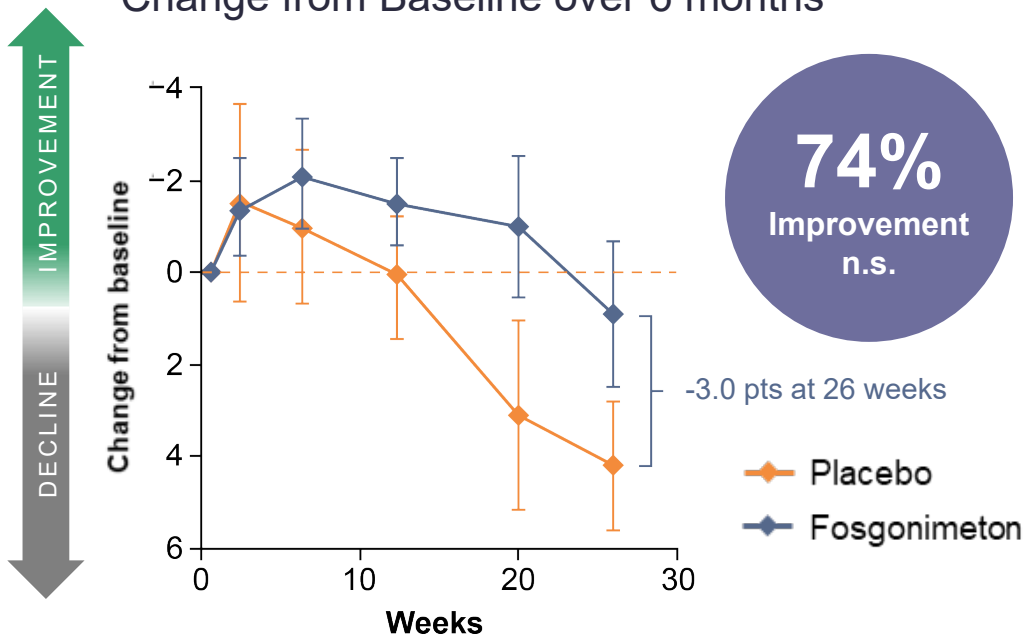
41% | **+1.7** points n.s.

IMPROVED FUNCTION

Improvement over placebo over 6 months as measured by ADCS-ADL23 in full study population

Favorable safety and tolerability profile, injection site reactions are most frequent AE

ADAS-COG11 (Procognitive Effect)
Change from Baseline over 6 months

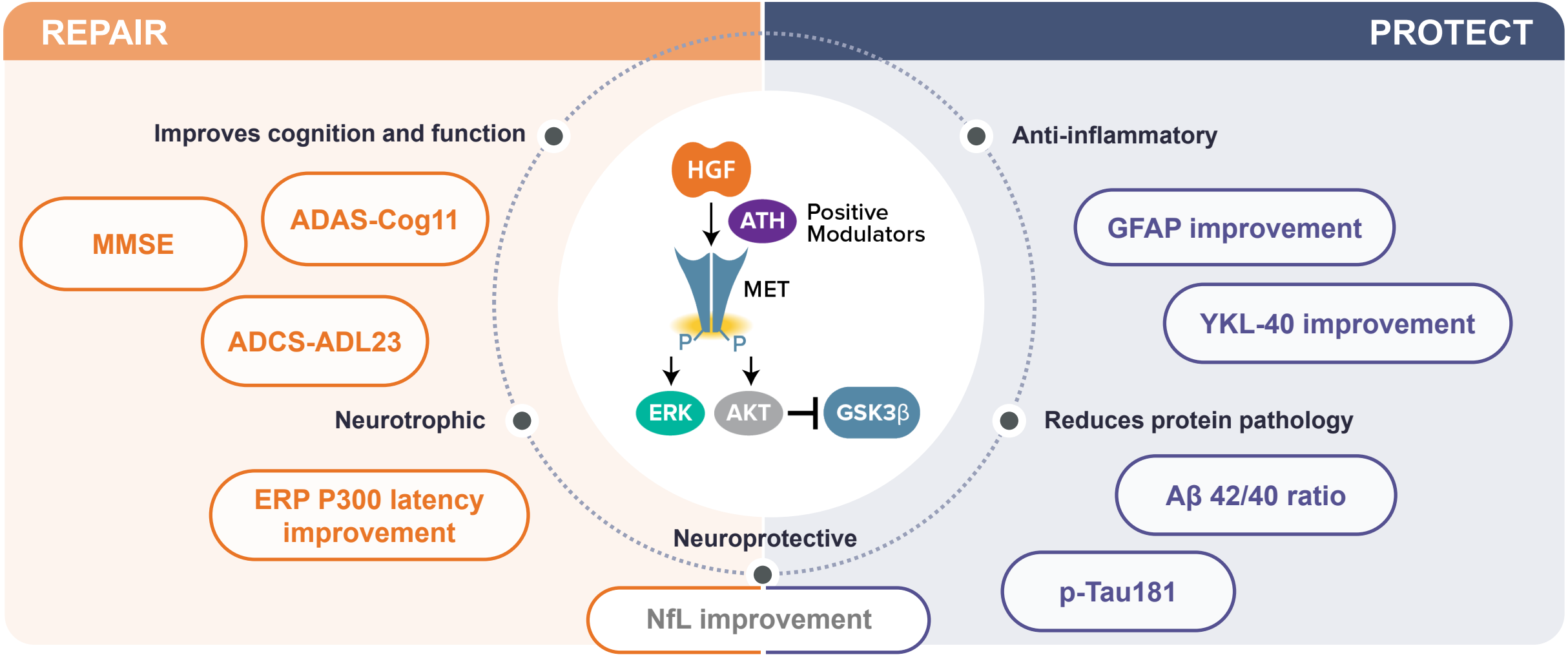


n at each visit	W2	W6	W12	W20	W26
Placebo	8	8	7	7	6
Fosgonimeton	19	19	17	16	17

Data from mITT population without background therapy and presented as unadjusted mean ± SEM; n.s., not statistically significant.
AD, Alzheimer's disease; ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living; AE, adverse event; mITT, modified intent-to-treat; W, week.

Clinical findings support therapeutic potential of fosgonimeton

SUMMARY OF ACT-AD RESULTS[1]

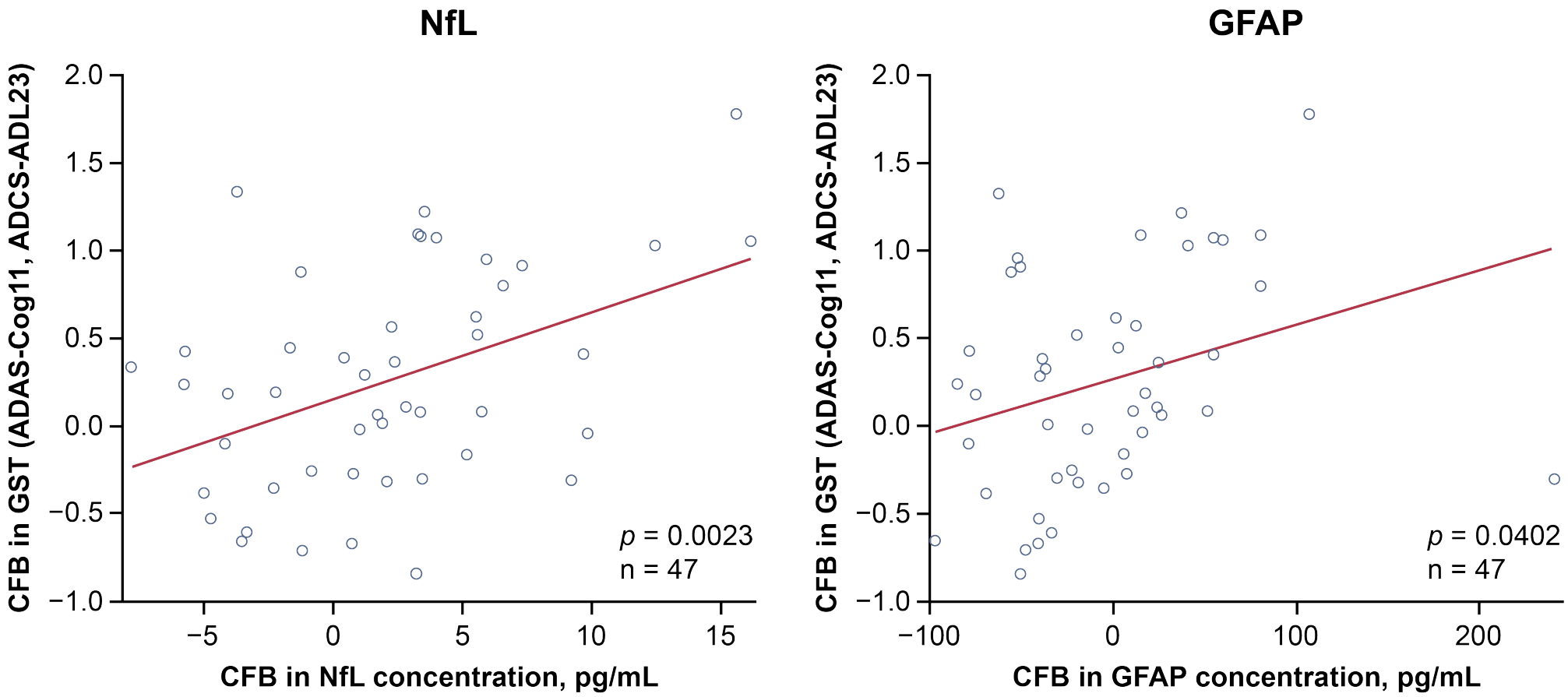


[1]Treatment effect of fosgonimeton without concomitant AChEI compared to placebo.

Aβ, amyloid beta; ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living; AKT, protein kinase B; ERK, extracellular-signal regulated kinase; ERP, event-related potential; GFAP, glial fibrillary acidic protein; GSK3β, glycogen synthase kinase-3 beta; HGF, hepatocyte growth factor; NfL, neurofilament light chain; p-Tau181, tau phosphorylated at threonine-181; YKL-40, chitinase-3–like protein 1.

Decreases in disease state biomarkers significantly correlate with improvements in cognitive and functional measures

EXPLORATORY ANALYSIS IN FULL STUDY POPULATION



ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living; CFB, change from baseline; GFAP, glial fibrillary acidic protein; GST, global statistical test; NfL, neurofilament light chain.

Fosgonimeton phase 2/3 LIFT-AD trial after amendment

LEARNINGS FROM ACT-AD INFORM TRIAL DESIGN OPTIMIZATION

POPULATION

N = ~300 participants without concomitant AChEI

Mild-to-moderate AD (MMSE 14-24; CDR 1-2)

26-week randomized, double-blind treatment, + optional 30-month OLEX

- Fosgonimeton (40 mg)
- Placebo

TIMELINE: To be updated following End of Phase 2 Meeting with FDA

ENDPOINTS

PRIMARY

- Composite endpoint of cognition and function (ADAS-Cog11 and ADCS-ADL23)
- Safety

SECONDARY

- Plasma NfL biomarker
- Cognition: ADAS-Cog11
- Function: ADCS-ADL23

EXPLORATORY

- Additional clinical endpoints and plasma biomarkers

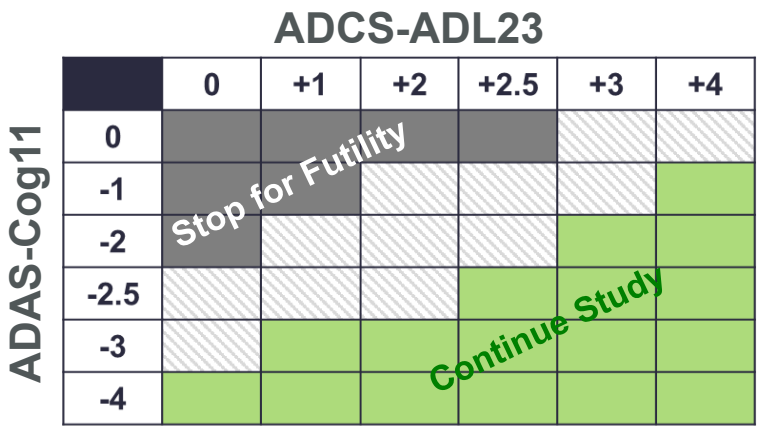
Aβ, amyloid beta; AD, Alzheimer's disease; ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living; ADCS-CGIC, Alzheimer's Disease Cooperative Study–Clinical Global Impression of Change; CDR, clinical dementia rating; COWAT, controlled oral word association test; EQ-5D-5L, 5-level EQ-5D version; GFAP, glial fibrillary acidic protein; GST, global statistical test; NfL, neurofilament light chain; NPI, neuropsychiatric inventory; OLEX, open-label extension; p-Tau181, tau phosphorylated at threonine 181; p-Tau217, tau phosphorylated at threonine 217; YKL-40, chitinase-3–like protein 1; ZBI, zarit burden interview.

Independent unblinded analysis by DMC supports the potential clinically meaningful activity of fosgonimeton

DEVELOPMENT PLAN OPTIMIZED WITH MITIGATED RISK

Pre-specified Methodology[1]

- Adaptive method enabling sample-size re-estimation based on interim findings that measures a candidate therapy's performance
- Monte Carlo simulations run to inform pre-specified decision framework
- Pre-specified constraints included maximum enrollment limit and minimum target power



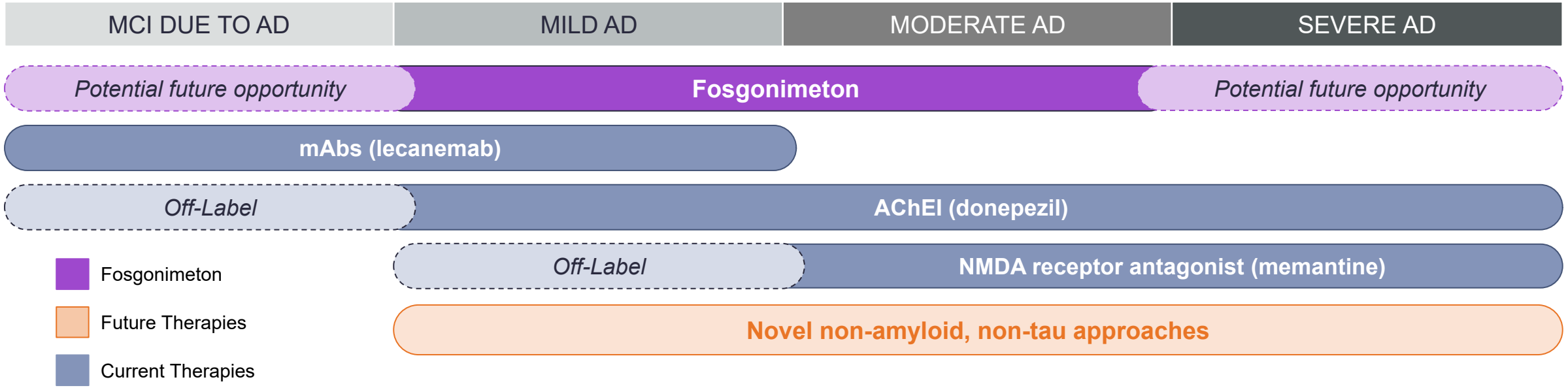
ANALYSIS & OUTCOME

- Efficacy and futility analysis performed on 100 patients without concomitant AChEIs who completed the trial
- DMC Recommendation (Oct 2022): **Continue LIFT-AD Study**
- Supports potential to achieve the primary endpoint, a composite score measuring cognition and function

[1]Conducted by DMC: Chair neurologist (MD) and two biostatisticians (PhD); [2]Primary endpoint is the global statistical test, an unweighted composite of ADAS-Cog11 and ADCS-ADL23.
AD, Alzheimer's disease; ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living; DMC, data monitoring committee.

Significant opportunity in Alzheimer's disease

EXTERNAL ENVIRONMENT



81% of all patients diagnosed with Alzheimer's disease are mild-to-moderate

2.1 million mild-to-moderate Alzheimer's patients diagnosed in 2021 in the US

Few available options

75% of patients in the US move to a second-line treatment in less than a year

Evolving regulatory environment favoring biomarker data and composite endpoints

Data sources: Decision Resources Group, accessed 2H2022; National Institute on Aging; Yuan et al., *Journal of Alzheimer's Disease* 2021.

AChEI, acetylcholinesterase inhibitor; AD, Alzheimer's disease; mAbs, monoclonal antibodies; MCI, mild cognitive impairment; NANT, non-amyloid, non-tau; NMDA, N-methyl-aspartate.

Strong rationale to advance fosgonimeton

SIGNIFICANT OPPORTUNITY TO TRANSFORM THE TREATMENT PARADIGM FOR NEURODEGENERATIVE DISEASES

- Anti-Inflammatory
- Neuroprotective
- Improves Cognition
- Improves Function
- Disease Modifying
- Favorable Safety and Tolerability Profile
- Risk Mitigated Ph 2/3 LIFT-AD following Interim Analysis

Differentiated and Risk Mitigated

+

- Evolving Regulatory Environment
- High Unmet Need

Favorable external landscape

AD, Alzheimer's disease.

Amyotrophic Lateral Sclerosis

ATH-1105

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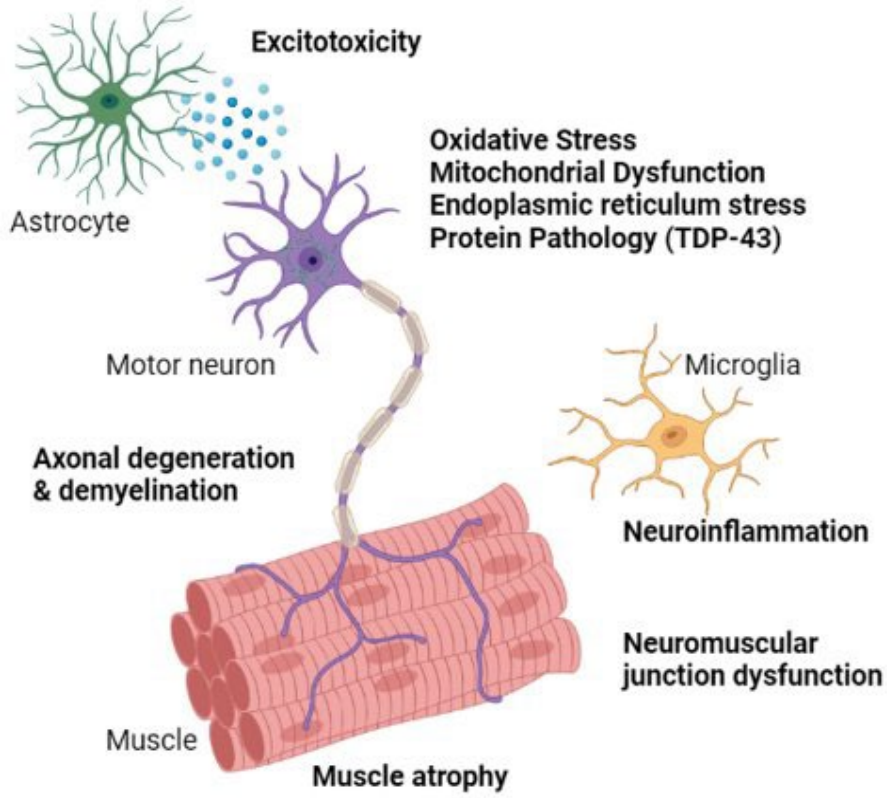
Positive modulation of HGF/MET as a potential treatment for ALS

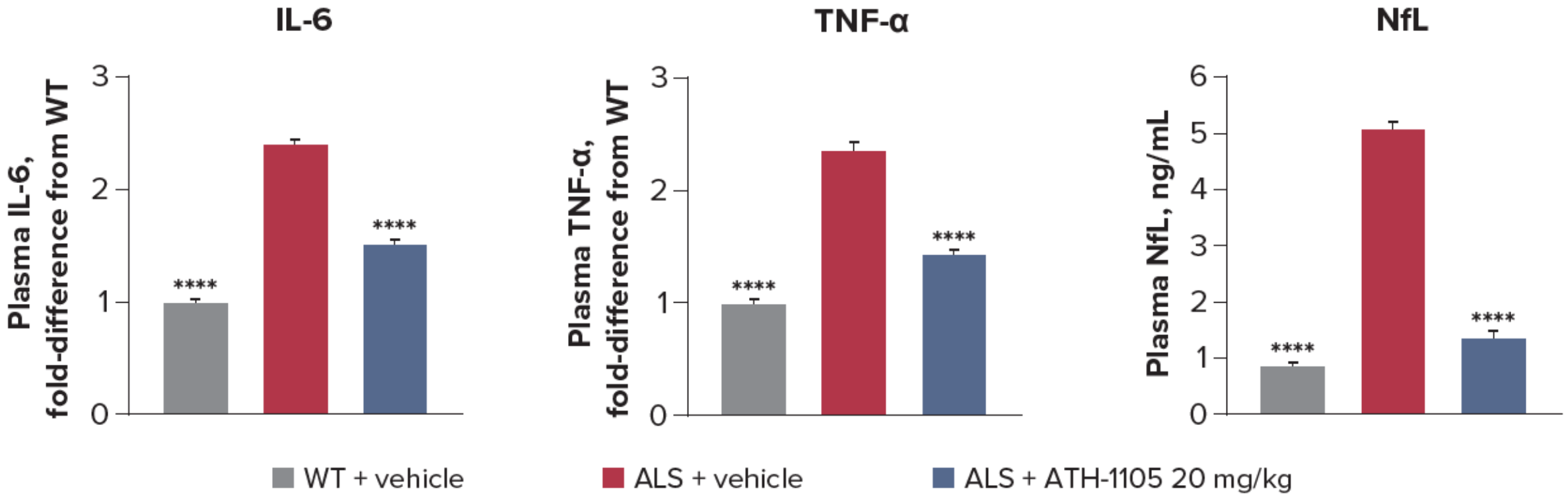
- ALS is a devastating progressive neurodegenerative disease
  - Characterized by degradation of motor neurons due to several factors including glutamate excitotoxicity, TDP-43 protein pathology, and systemic inflammation
- Approximately 97% of ALS patients have TDP-43 pathology[1]
  - TDP-43 is a nuclear protein under normal conditions but in ALS forms toxic aggregates in the cytoplasm of motor neurons
  - TDP-43 mouse models have been developed that exhibit TDP-43 pathology and ALS-like symptoms
- Promotion of HGF/MET activity has been reported to have beneficial effects in preclinical models of ALS
  - HGF delays disease progression in ALS animal models[2,3]
  - HGF reduces muscle impairment and motor neuron loss in an ALS mouse model[4]



[1]Scotter et al., *Neurotherapeutics* 2015.
[2]Ishigaki et al., *J Neuropathol Exp Neurol* 2007.
[3]Lee et al., *Acta Neuropathol Commun* 2019.
[4]Vallarola et al., *Int J Mol Sci* 2020.

Anti-inflammatory and neuroprotective: ATH-1105 reduces markers of inflammation and neurodegeneration

TDP-43 MOUSE MODEL OF ALS



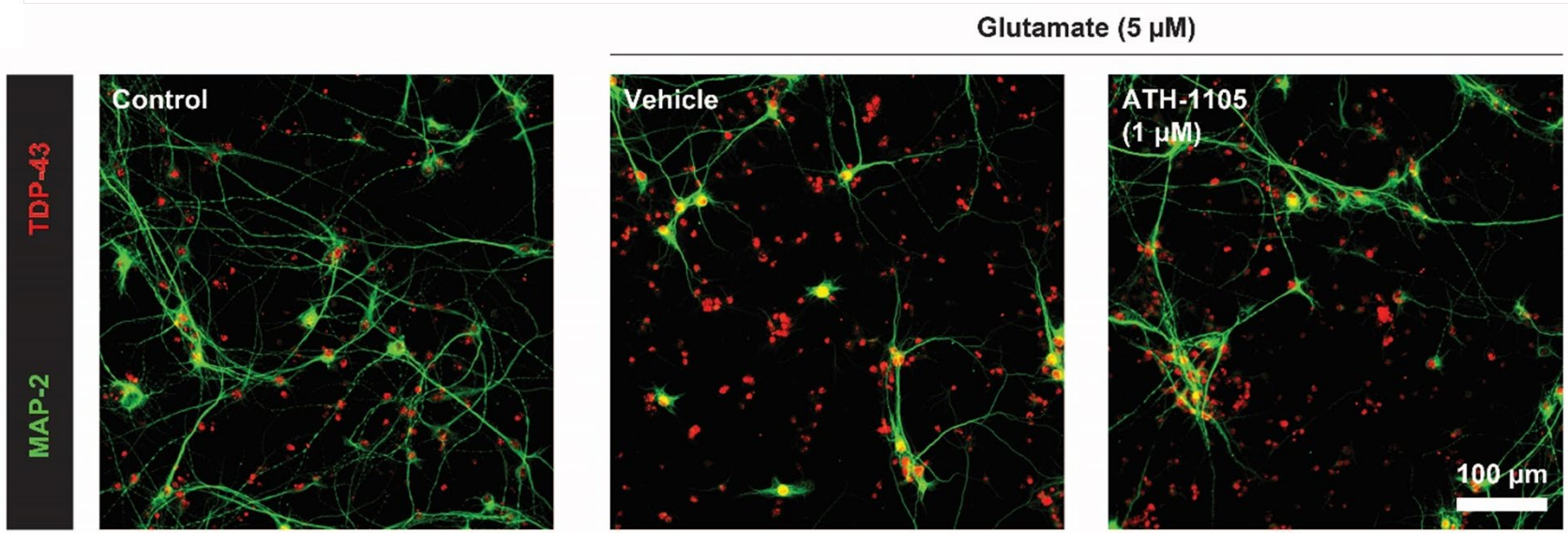
Data presented as mean ± SEM.

Statistical significance was determined by 1-way ANOVA with Dunnett's test versus ALS + vehicle. ****p < 0.0001.

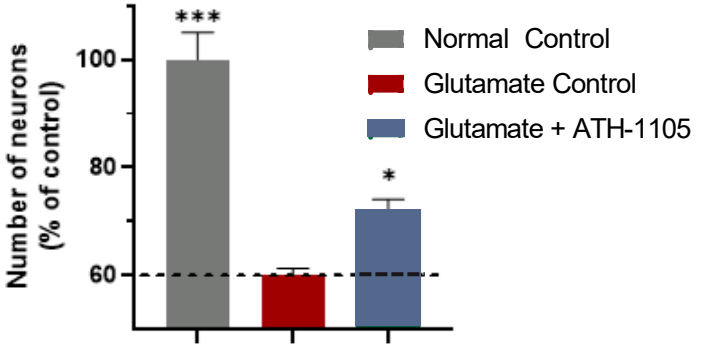
ALS, amyotrophic lateral sclerosis; IL-6, interleukin 6; NfL, neurofilament light chain; TDP-43, TAR DNA-binding protein 43; TNF-α, tumor necrosis factor alpha; WT, wild-type.

Neuroprotection and protein pathology: ATH-1105 reduces extranuclear TDP-43 accumulation and enhances neuron survival

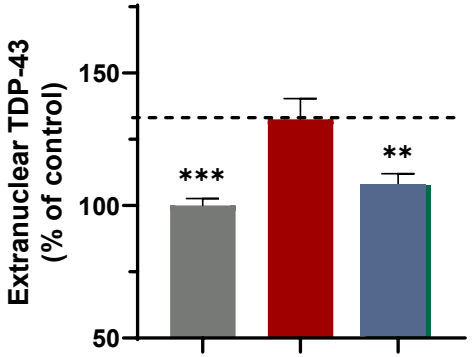
GLUTAMATE CHALLENGE MODEL IN MOTOR NEURON CULTURES



Motor neuron survival following glutamate challenge



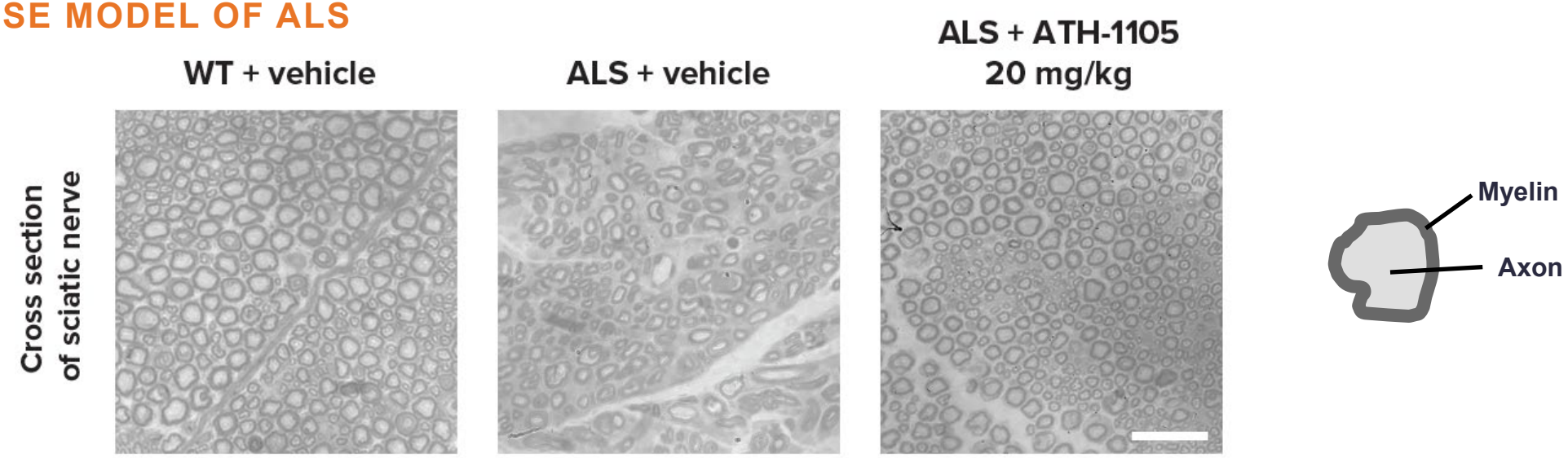
Glutamate-induced TDP-43 accumulation in spinal motor neurons

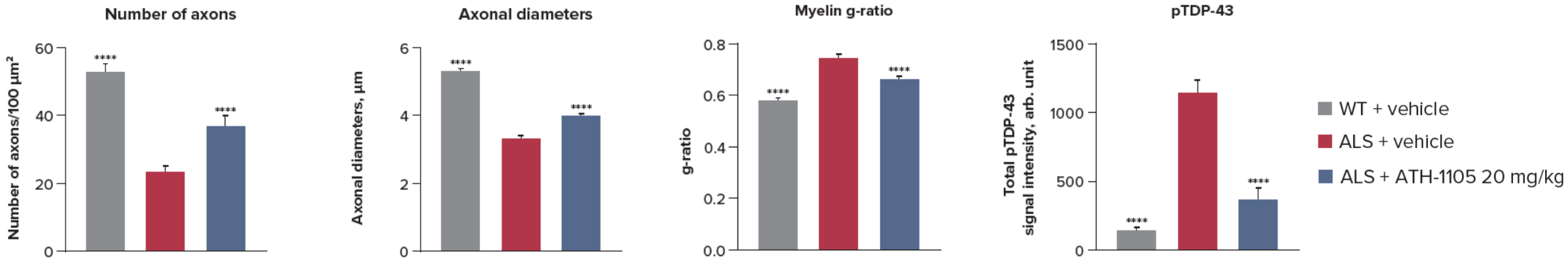


Primary rat spinal motor neurons. Cultures treated with vehicle control or 5 µM glutamate.
Data presented as mean ± SEM. Statistics applied: 1-way ANOVA with Fisher least significant difference test. *p < 0.05, **p < 0.01, ***p < 0.001 versus Glutamate Control; n = 6.
Scale bar: 100 µm, applies to all images.
MAP-2, microtubule-associated protein-2; TDP-43, TAR DNA-binding protein 43.

Neuroprotective: ATH-1105 protects against axon degeneration and demyelination, and reduces pTDP-43

TDP-43 MOUSE MODEL OF ALS





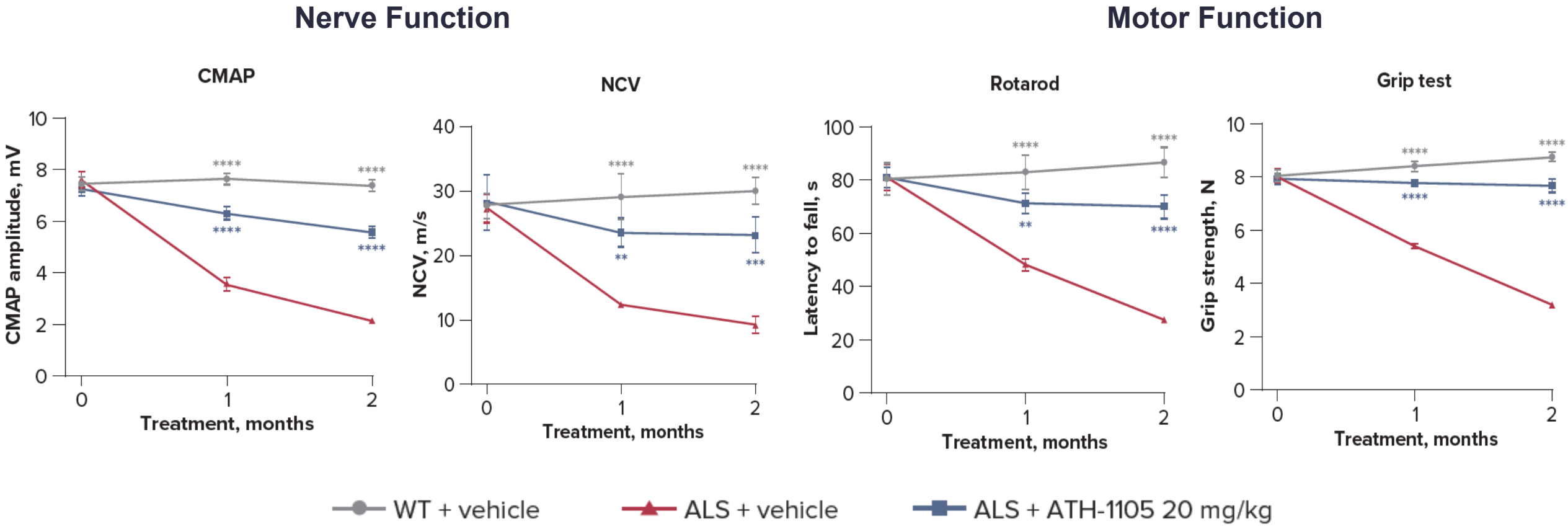
Graphical representation of the number of axons (per 100 µm2), axonal diameter (in micrometers), and mean of myelin g-ratio, defined as the ratio of the inner axonal diameter to the total axonal diameter, following 2 months of treatment. Data presented as mean + SEM. Statistical significance was determined by 1-way ANOVA with Dunnett's test versus ALS + vehicle. ****p < 0.0001.

Scale bar: 10 µm, applies to all images.

ALS, amyotrophic lateral sclerosis; pTDP-43, phosphorylated TDP-43; TDP-43, TAR DNA-binding protein 43; WT, wild-type.

Function: ATH-1105 improves nerve and motor function

TDP-43 MOUSE MODEL OF ALS



Data presented as mean ± SEM.

Statistical significance was determined by 2-way ANOVA with Dunnett's test versus ALS + vehicle. **p < 0.01; ***p < 0.001; ****p < 0.0001. n = 10 mice per group.

ALS, amyotrophic lateral sclerosis; CMAP, compound muscle action potential; NCV, nerve conduction velocity; TDP-43, TAR DNA-binding protein 43; WT, wild-type.

Survival: ATH-1105 prolongs survival and delays time to first mortality

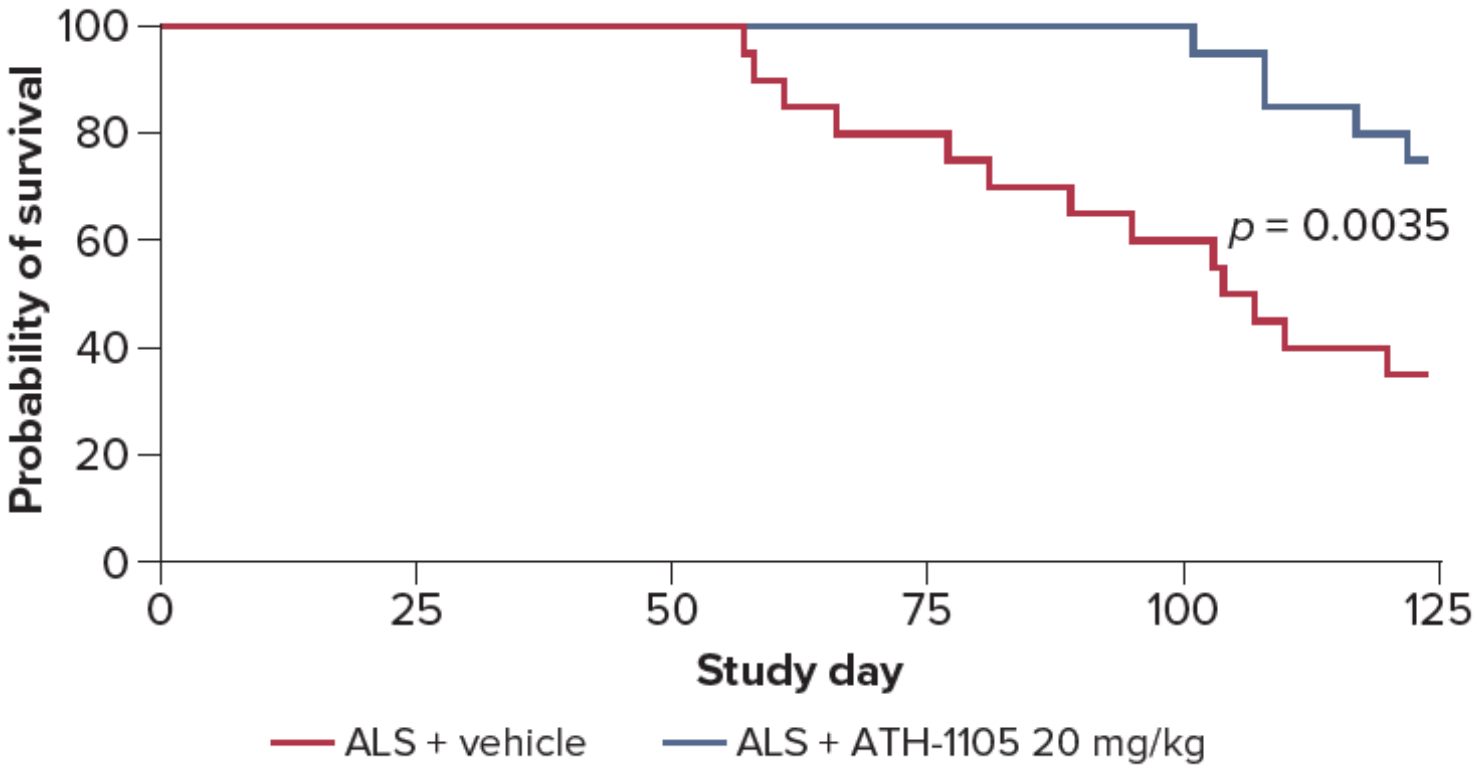
TDP-43 MOUSE MODEL OF ALS

Time to first mortality

- Day 57 in ALS + vehicle group
- Day 101 in ALS + ATH-1105 20 mg/kg

Percent survival at 5 months of age

- 35% in ALS + vehicle group
- 75% in ALS + ATH-1105 20 mg/kg



Data presented as Kaplan-Meier survival curves
Statistical significance was determined by log-rank (Mantel-Cox) test. n = 20 mice per group.
ALS, amyotrophic lateral sclerosis; TDP-43, TAR DNA-binding protein 43; WT, wild-type.

ATH-1105 preclinical data summary

In the TDP-43 mouse model of ALS, daily oral treatment of ATH-1105 resulted in:

- Improvement in motor function, and protection against body weight reduction
- Preservation of nerve function and structure
- Reduction of plasma biomarkers of systemic inflammation and neurodegeneration
- Prolonged survival and delayed time to first mortality

These results highlight the therapeutic potential of ATH-1105 in ALS and support further development

ALS, amyotrophic lateral sclerosis; TDP-43, TAR DNA-binding protein 43.

Significant unmet need: Amyotrophic Lateral Sclerosis (ALS)

~75,000[1]

People globally affected by ALS with the 40% of those cases in the US

Only Four[1]

Approved drugs specifically indicated for the treatment of ALS

Zero[1]

ALS drugs targeting neurotrophic factor systems with a multimodal mechanism of action

Global Market Size for ALS[1]

2019:	2029 Projected:
$197M	**$781M**

Drugs in Development[1,2]

Limited differentiated and multifactorial approaches

Tofersen, a late-clinical stage asset, addresses a familial form of ALS accounting for <3% of all cases

Significant Opportunity for ATH-1105

Limited approved treatment options exist for ALS patients

Multimodal mechanism of action – neuroprotective, anti-inflammatory and potentially disease modifying

Positive modulation of a naturally occurring repair mechanism

Athira PHARMA

[1]GlobalData ALS Market Reports; ALS Association
[2]Berdynski, et al., *Nature* 2022.

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Athira management team with significant CNS product development and approval experience

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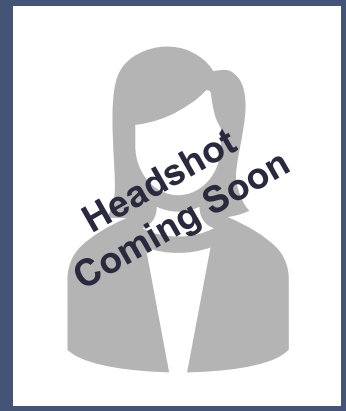

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Moving forward

- ✓ Independent, unblinded interim analysis of Phase 2/3 LIFT-AD
- ✓ Focus on 40 mg dose

→ Timelines to be updated following End of Phase 2 meeting with FDA

- ✓ Enrolled 28 patients in exploratory SHAPE trial in PD and dementia with Lewy bodies

→ Complete SHAPE and evaluate next steps

- ✓ Prolonged survival and delayed time to first mortality, and demonstrated consistent improvements in motor function, nerve function, biomarkers and nerve morphology in transgenic mouse model of ALS

→ Advance ATH-1105 in ALS and target initiating first-in-human Phase 1 trial in 2024

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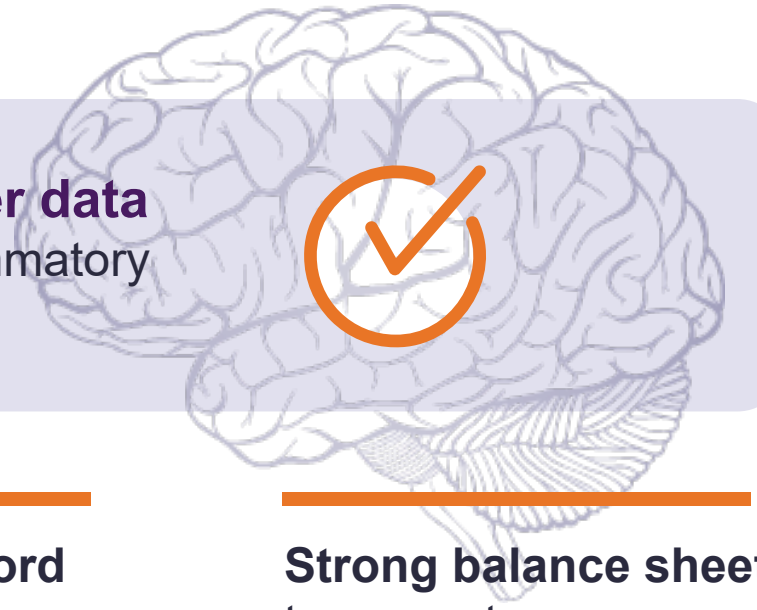
AD, Alzheimer's disease; ALS, amyotrophic lateral sclerosis; IND, investigational new drug; PD, Parkinson's disease; POC, proof-of-concept.

Well positioned to lead with innovative approach to battling neurodegenerative diseases



Consistent and correlative preclinical, clinical and biomarker data showing the potential of fosgonimeton to be neuroprotective, anti-inflammatory and disease modifying in a number of neurodegenerative diseases

Mitigated development risk through independent, unblinded interim analysis of Phase 2/3 LIFT-AD trial

Evolving regulatory environment and favorable competitive landscape

Strong track record of execution and leadership team with significant CNS product development and approval experience

Strong balance sheet to support programs through to key inflection points

AD, Alzheimer's disease; CNS, central nervous system.

Thank You

Athira PHARMA