Athira PHARMA

Corporate Presentation

May 2024

ADVANCING NEW THERAPIES FOR NEURONAL HEALTH

Disclaimer

This presentation and the accompanying oral commentary contain forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. These forward-looking statements are based on our management's beliefs and assumptions and on information currently available to our management. Forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified. In some cases, you can identify forward-looking statements by terminology such as "may," "will," "should," "could," "expect," "plan," anticipate," "believe," "estimate," "predict," "intend," "potential," "would," "continue," "ongoing" or the negative of these terms or other comparable terminology. Forward-looking statements include all statements other than statements of historical fact contained in this presentation, including information concerning our future financial performance, business plans and objectives, timing and success of our planned development activities, our ability to obtain regulatory approval, the potential therapeutic benefits and economic value of our product candidates as a potential treatment for Alzheimer's disease, Parkinson's disease, Parkinson's disease dementia, dementia with Lewy bodies, amyotrophic lateral sclerosis, and other neurodegenerative diseases, the anticipated reporting of data, and the potential learnings from preclinical studies and other nonclinical data, the ACT-AD and SHAPE trials and LIFT-AD unblinded interim efficacy and futility analysis and their ability to inform and improve future clinical development plans, potential growth opportunities, competitive position, and industry environment and potential market opportunities.

Forward-looking statements are subject to known and unknown risks, uncertainties, assumptions and other factors. It is not possible for our management to predict all risks, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in any forward-looking statements we may make. These factors, together with those that are described in greater detail in our filings with the Securities and Exchange Commission ("SEC") may cause our actual results, performance or achievements to differ materially and adversely from those anticipated or implied by our forward-looking statements.

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By attending or receiving this presentation you acknowledge that you will be solely responsible for your own assessment of the market and our market position and that you will conduct your own analysis and be solely responsible for forming your own view of the potential future performance of our business. This presentation contains estimates, projections and other information concerning market, industry and other data. We obtained this data from our own internal estimates and research and from academic and industry research, publications, surveys, and studies conducted by third parties, including governmental agencies. These data involve a number of assumptions and limitations, are subject to risks and uncertainties, and are subject to change based on various factors, including those discussed in our filings with the SEC. These and other factors could cause results to differ materially from those expressed in the estimates made by the independent parties and by us. While we believe such information is generally reliable, we have not independently verified any third-party information.

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

Harnessing the power of the neurotrophic HGF system for the treatment of neurodegenerative diseases

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Investment highlights

Leveraging deep knowledge, broad IP, and HGF biology to develop potential first-in-class and next-generation small molecules to protect and repair neuronal networks

Strong balance sheet
to support clinical programs through key inflection points

Leadership team with significant CNS product development and approval experience

Neurotrophic HGF system is critical to normal brain function and plays a key role in neurodegenerative diseases

Fully enrolled late-stage clinical trial of fosgonimeton in Alzheimer's disease (AD) with data expected in 2H24

- Factors that support the potential success of the Phase 2/3 LIFT-AD trial
  - Exploratory ACT-AD trial showed improvement across key AD indicators and informed LIFT-AD design
  - Independent unblinded interim analysis of LIFT-AD trial cleared stringent go/no go criteria; DMC recommended that the study continue
  - Ongoing open label extension trial shows high participation rate and long duration of investigational treatment
  - Exploratory SHAPE trial showed potential improvements in cognition in a different patient population treated with the same dose as being tested in the LIFT-AD trial
  - Well-tolerated with a favorable safety profile
- Targeting the mild-to-moderate patient population representing an enormous opportunity – likely >3M in US alone

Robust pipeline of proprietary small molecules targeting HGF biology

- Restoring the impaired HGF system in disease states has the potential to treat Alzheimer's, Parkinson's, ALS, neuropathic pain, and other neurological diseases
- We own or in-license worldwide IP rights to all of our drug candidates

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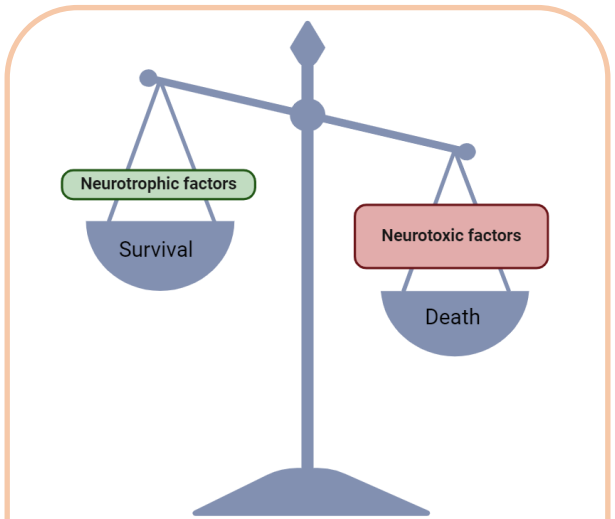
ALS, amyotrophic lateral sclerosis; HGF, hepatocyte growth factor.

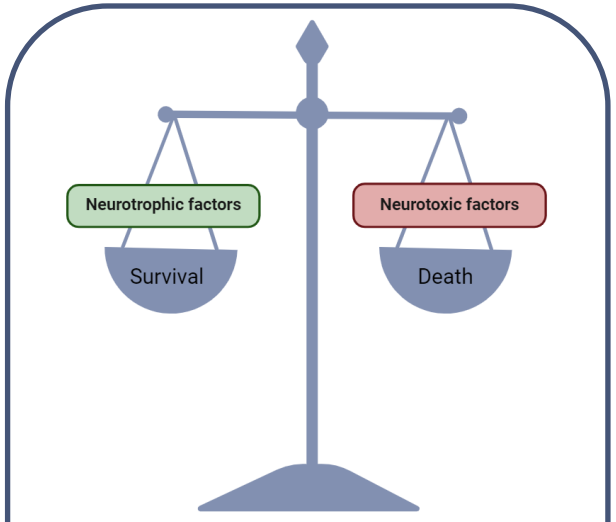
Neurotrophic factors, including HGF, are a less appreciated approach to addressing neurodegenerative disease

- We have developed a series of **small molecules** designed to enhance the activity of the neurotrophic HGF system
  - Our small molecule approach overcomes a major hurdle in evaluating the potential of therapies targeting neurotrophic factors
  - ATH small molecules can cross the BBB and distribute to relevant nervous system tissue

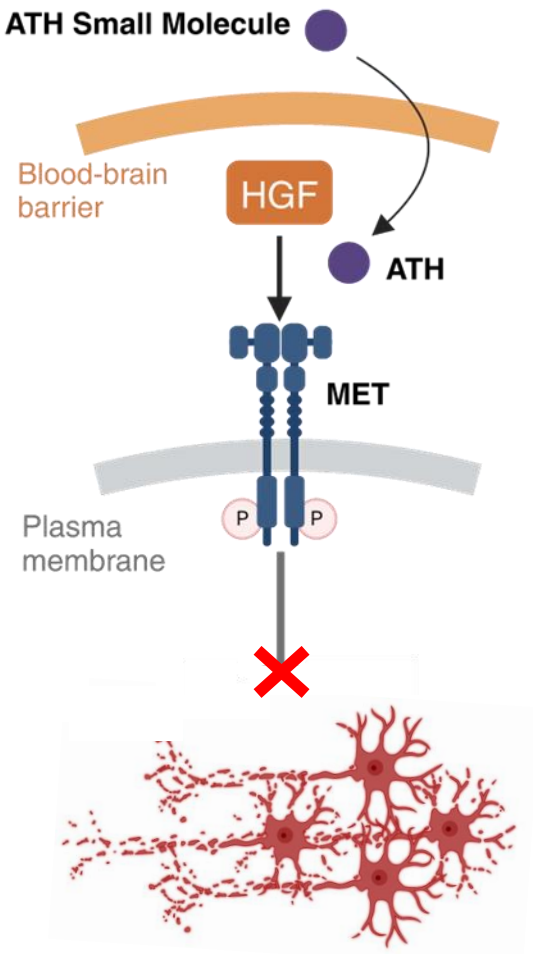


Increased levels of neurotoxic factors (oxidative stress, protein pathology, neuroinflammation, and mitochondrial dysfunction), and neurotrophic deficits, drive **neurodegeneration**

ATH small molecules



Promotion of neurotrophic HGF signaling has the potential to counteract neurotoxic factors and restore neuronal health and function



Neurodegeneration

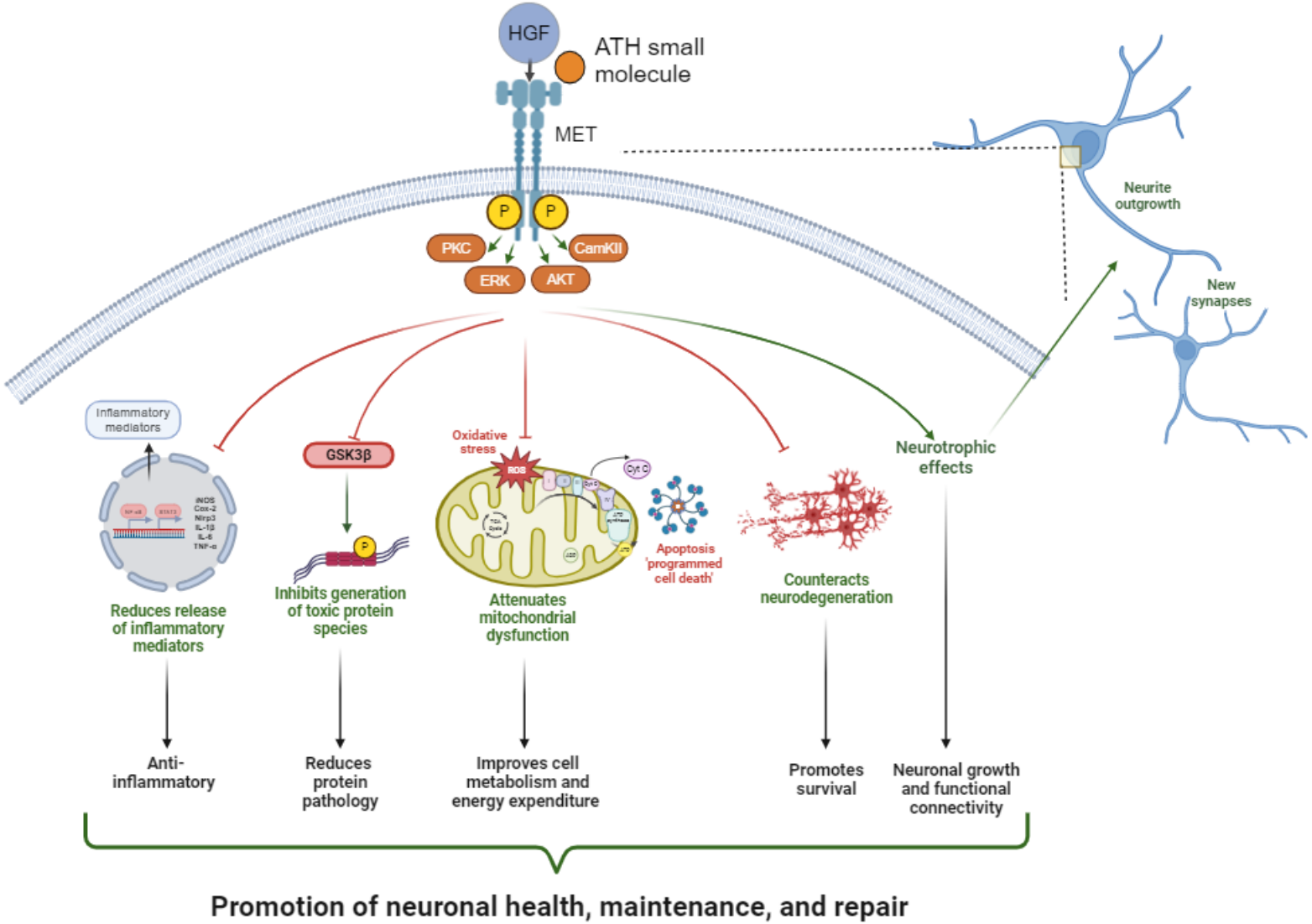
HGF is a critical neuroprotective system

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

HGF signaling has pleiotropic downstream signaling effects

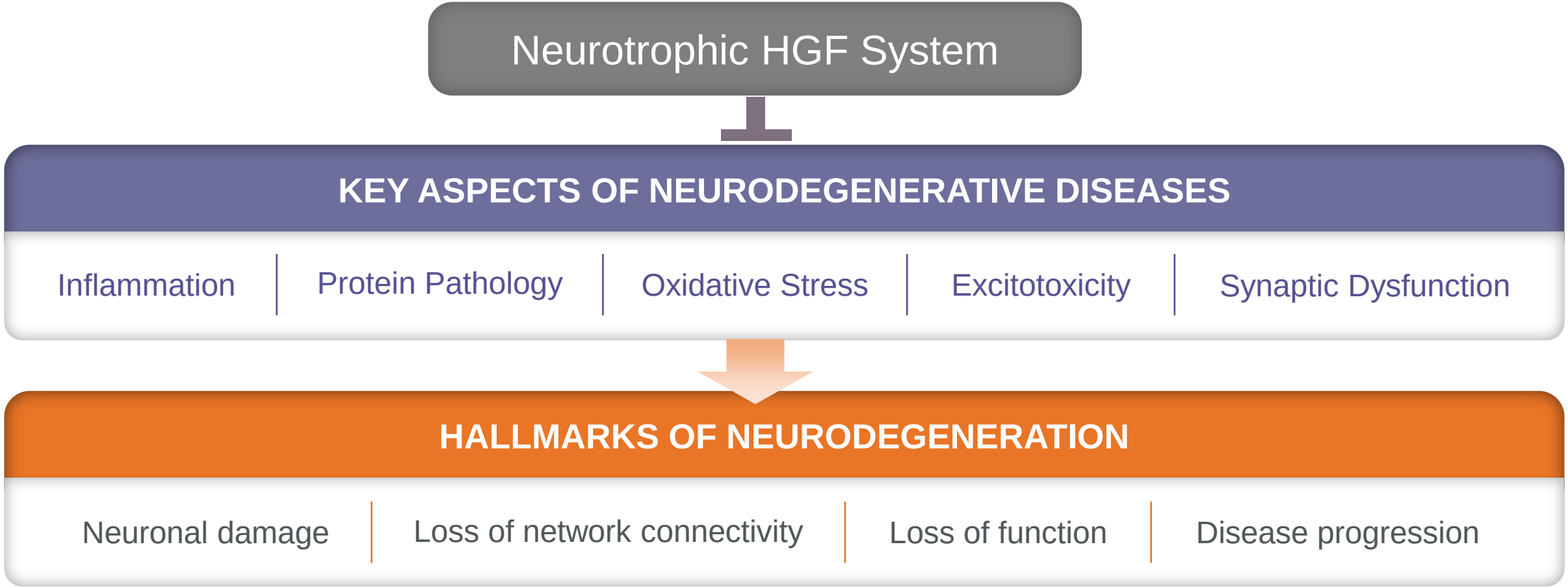
- Activation of MET (HGF receptor) leads to activation of multiple signaling cascades
- These signaling events influence the ability of the cell to counteract neurodegenerative hallmarks and undergo repair

Positive modulation of HGF signaling has the potential to promote neuronal health and repair by activating a range of pathways that alleviate key components of neurodegeneration

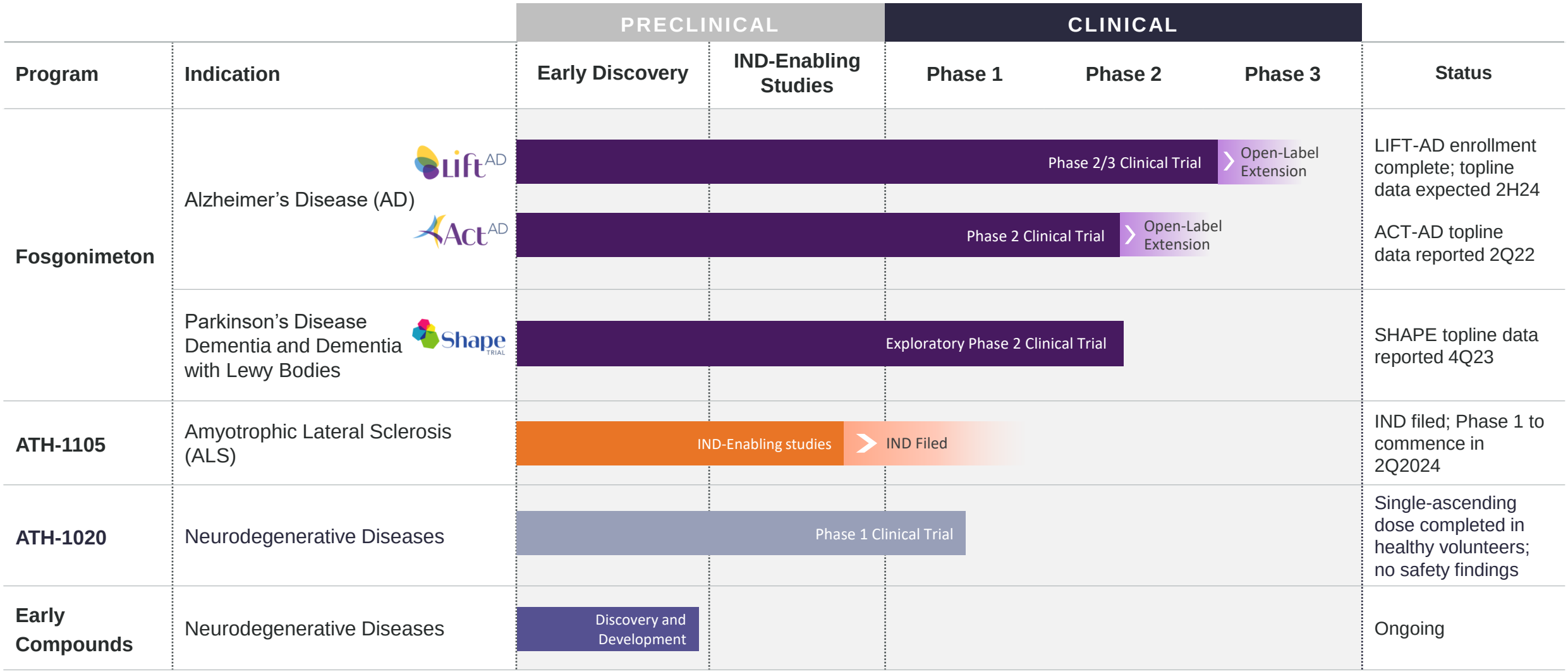


*Desole et al., *Frontiers in Cell and Developmental Biology* 2021.

HGF system impacts several of the complex pathologies that lead to neurodegeneration



Therapeutic potential across a broad range of clinical indications



IND, investigational new drug.

Fosgonimeton for the treatment of mild-to-moderate Alzheimer's disease

Phase 2/3 LIFT-AD Trial Fully Enrolled with Topline Data Expected in 2H2024

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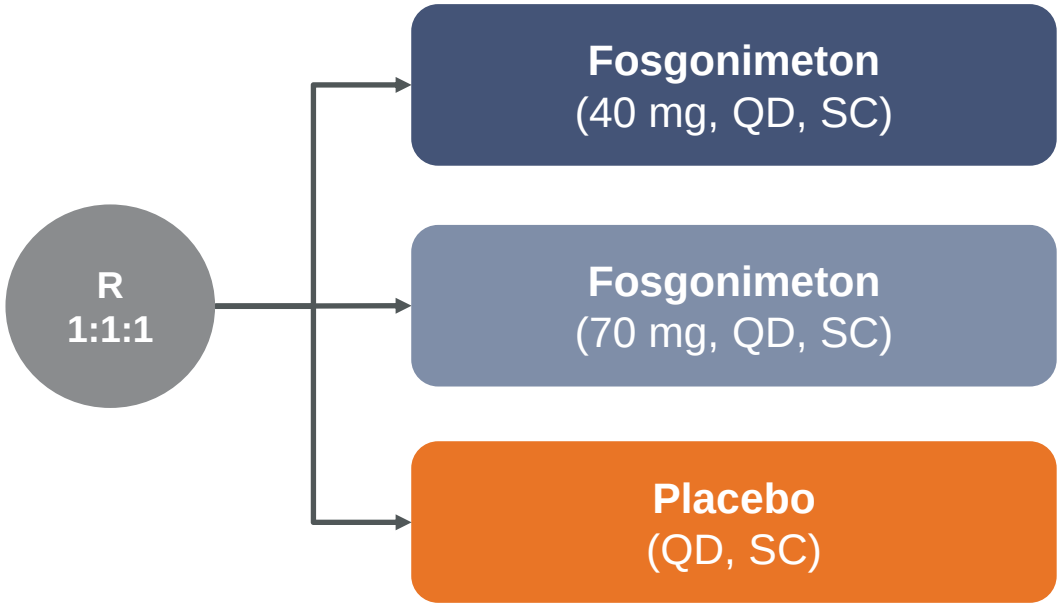
Fosgonimeton exploratory phase 2 trial in mild-to-moderate Alzheimer's disease*

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



ENDPOINTS

Primary: Change in ERP P300 latency, 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
- P300 not appropriate endpoint given variability

* Trial did not meet its primary endpoint.
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; ERP P300, event-related potential; OLEX, open-label extension; QD, once daily; R, randomization; SC, subcutaneous

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

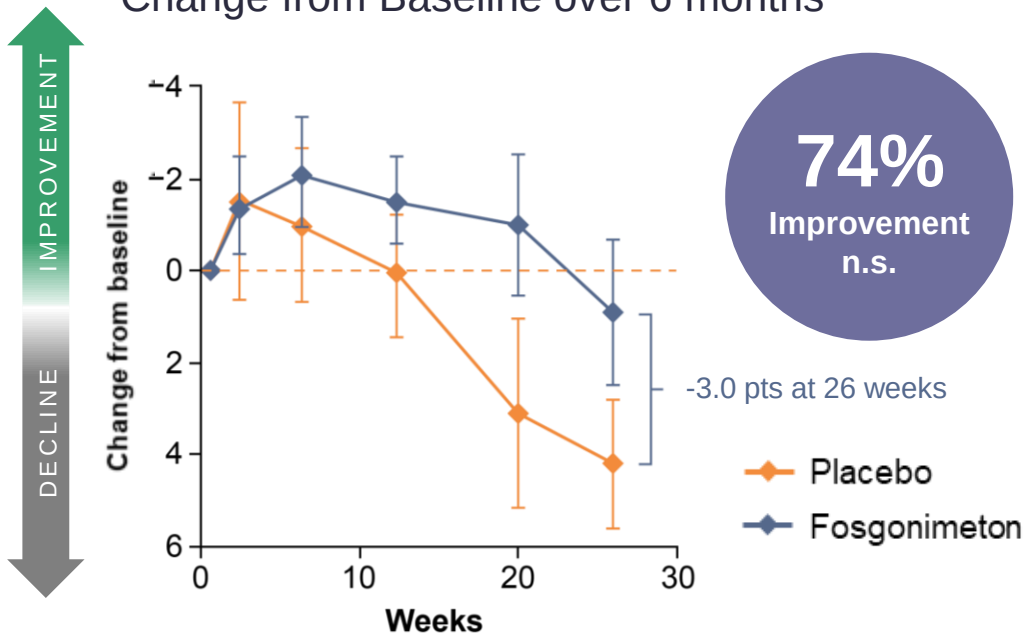
41% | **+1.7** points n.s.

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

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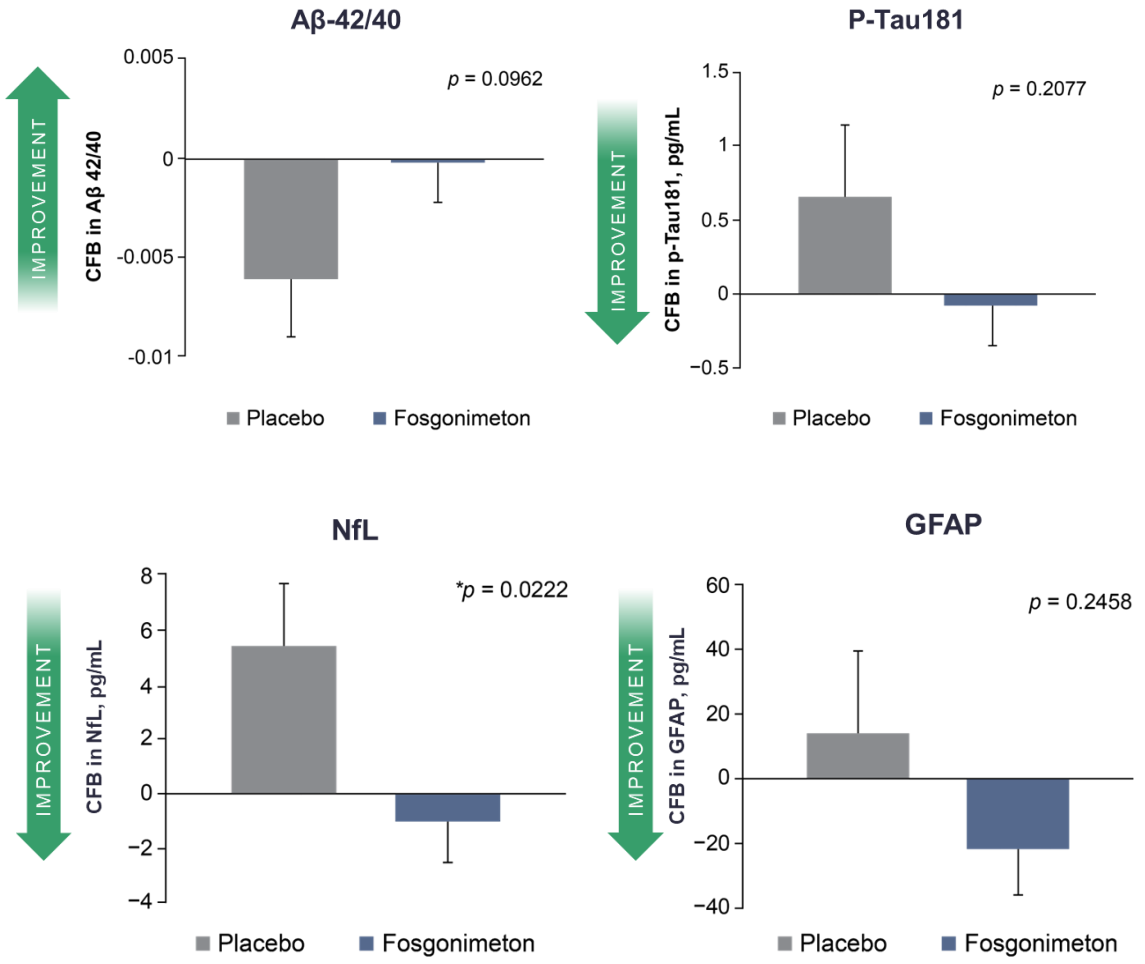
Data from mITT population without background therapy and presented as unadjusted mean ± SEM; n.s., not statistically significant.
AD, Alzheimer's disease; ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living;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

Fosgonimeton induces directional improvements in hallmarks of Alzheimer's disease

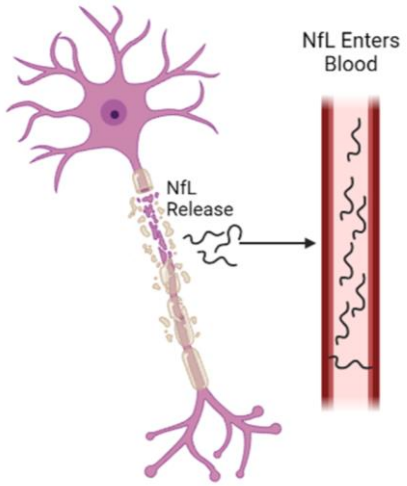
- Decreased Aβ 42/40 ratio and increased absolute p-Tau values are hallmarks of Alzheimer's disease
- NfL is an established, objective biomarker of neurodegeneration, and GFAP is a marker of neuroinflammation
- Changes support relevance of the HGF/MET pathway to Alzheimer's disease pathology
- Supports 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=12 for fosgonimeton treatment without concomitant AChEI.

Aβ, amyloid beta; CFB, change from baseline; GFAP, glial fibrillary acidic protein; HGF, hepatocyte growth factor; NfL, neurofilament light chain; p-Tau181, tau phosphorylated at threonine-181; SE, standard error

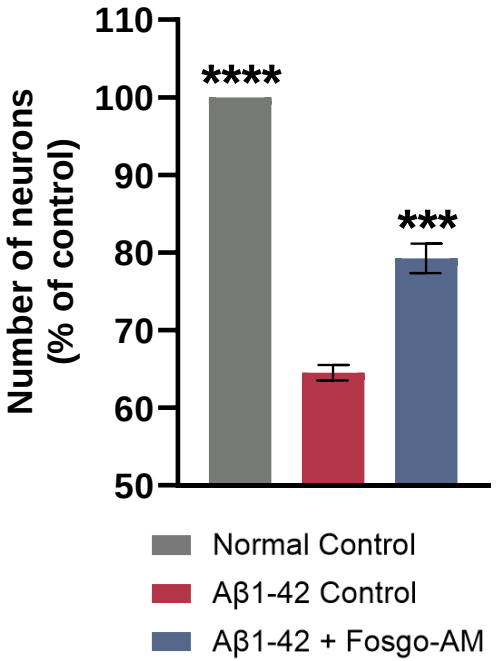
Neuroprotective - Fosgonimeton reduces neurofilament light, an established marker of neurodegeneration



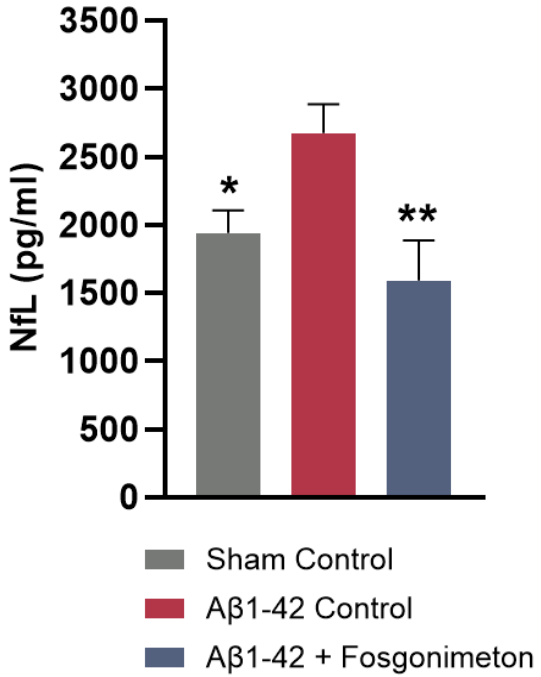
NfL is released into the blood following neuronal injury and neurodegeneration

Preclinical

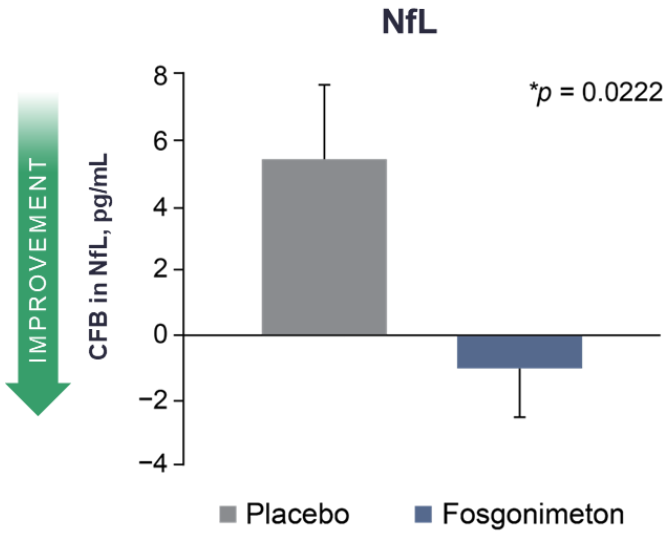
Cortical neuron survival following Aβ1-42 exposure



NfL in Aβ1-42 aged mouse model



Clinical



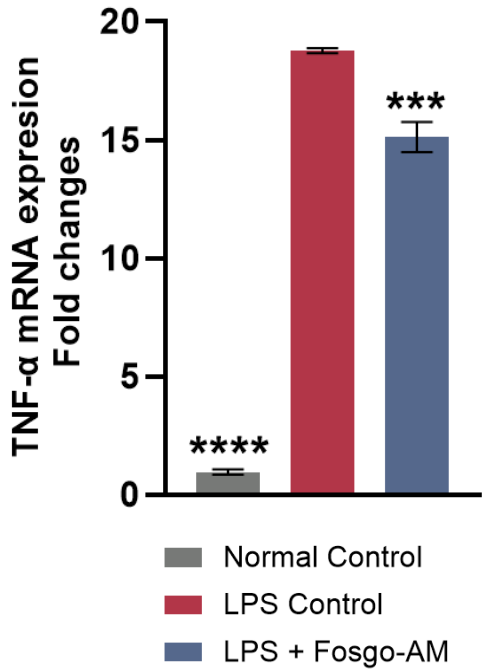
- Neurofilament light (NfL) is an established, objective biomarker of neurodegeneration
- Decrease of NfL to below baseline levels is suggestive of repair in this continuously progressive disease

Aβ, amyloid-beta; CFB, change from baseline; NfL, neurofilament light chain.
For in vitro assays, fosgonimeton was administered as the active metabolite, fosgo-AM; One-way ANOVA with Fisher's LSD; ***p<0.001, ****p<0.0001 vs. Aβ1-42 control; N = 3 biological replicates (n = 4-6 technical replicates). In vivo: NfL measured in CSF from intrahippocampal Aβ1-42 aged mouse model following 28-day fosgonimeton treatment. One-way ANOVA with Fisher's LSD; *p< 0.05, **p<0.01 vs. Aβ1-42 control; n = 12 mice per group 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).

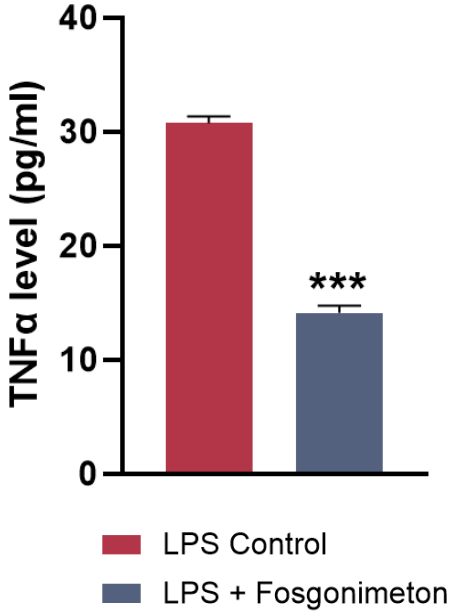
Anti-inflammatory - Fosgonimeton reduces markers of neuroinflammation

Preclinical

TNF-α expression in microglia following LPS exposure

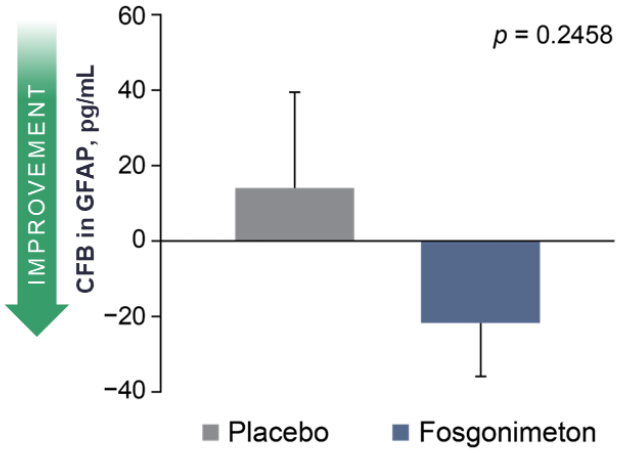


TNF-α in brain homogenates from LPS mouse model



Clinical

GFAP

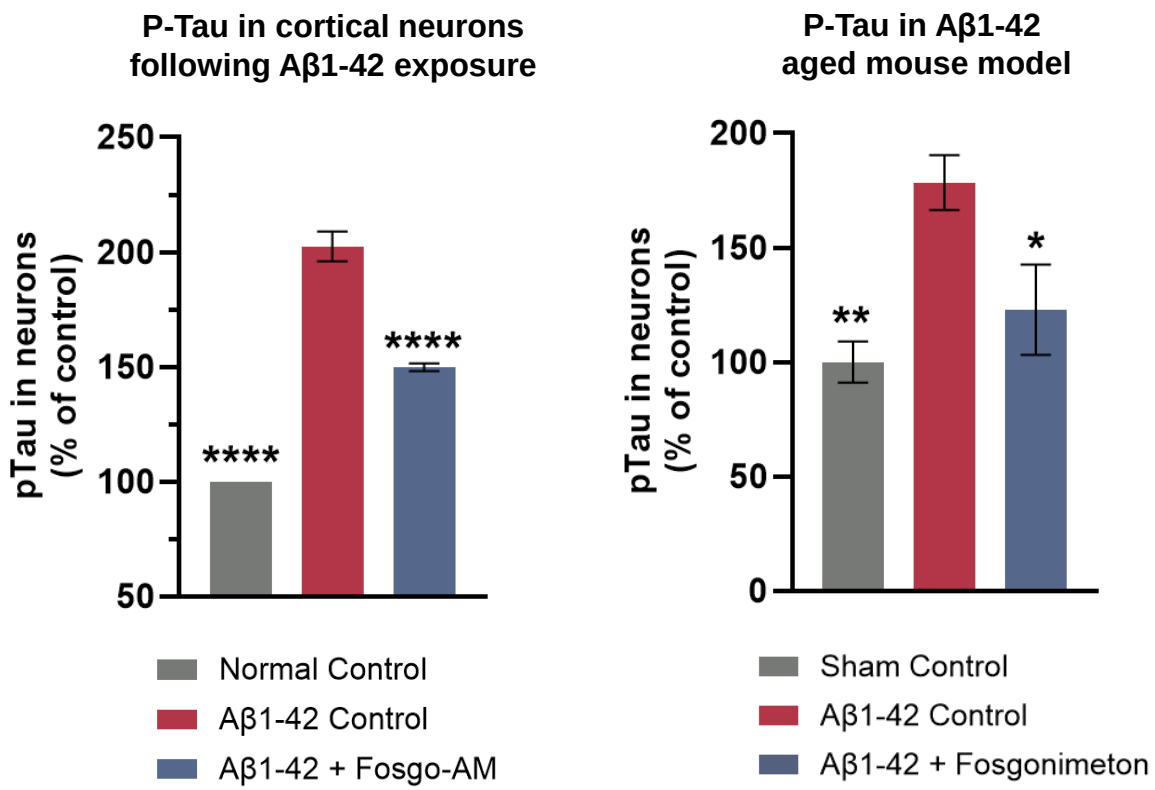


- GFAP is a marker of neuroinflammation
- Magnitude of decrease is encouraging in this continuously progressive conditions

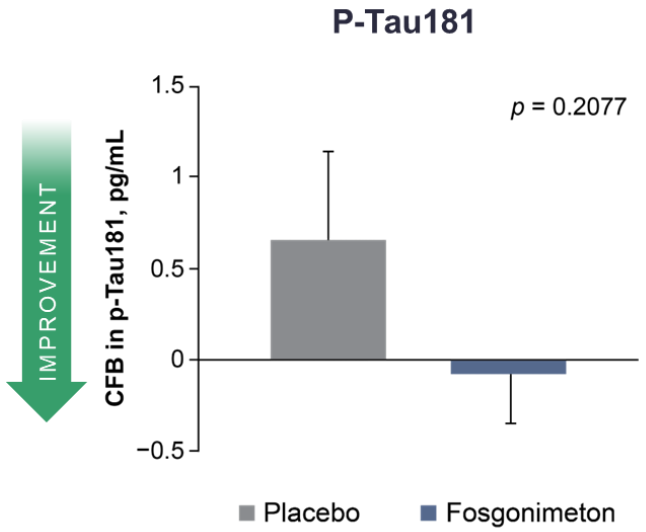
CFB, change from baseline; LPS, lipopolysaccharide; GFAP, glial fibrillary acidic protein; TNFα, tumor necrosis factor alpha
For in vitro assays, fosgonimeton was administered as the active metabolite, fosgo-AM; One-way ANOVA with Dunnett's post-test; ***p<0.001, ****p<0.0001 vs. LPS control; n = 3 technical replicates. In vivo: TNFα measured in brain homogenates 1.5h post LPS intraperitoneal administration. Fosgonimeton was administered 20 minute prior to LPS administration. One-way ANOVA with Dunnett's post-test; ***p<0.001 vs. LPS control; n = 10 mice per group mean + SEM.
For clinical data, 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),

Protein Pathology - Fosgonimeton reduces tau phosphorylation (pTau)

Preclinical



Clinical



- Increase p-Tau levels are a hallmark of Alzheimer's disease
- Changes in levels of pTau support relevance of HGF/MET pathway to Alzheimer's specific protein pathology and are suggestive of disease modification

Aβ, amyloid-beta; CFB, change from baseline; P-Tau, phospho-Tau; p-Tau181, tau phosphorylated at threonine-181
For in vitro assays, fosgonimeton was administered as the active metabolite, fosgo-AM; One-way ANOVA with Fisher's LSD; ****p<0.0001 vs. Aβ1-42 control; N = 3 biological replicates (n = 4-6 technical replicates). In vivo: P-Tau measured in hippocampal slices from intrahippocampal Aβ1-42 aged mouse model following 28-day fosgonimeton treatment One-way ANOVA with Fisher's LSD; *p<0.05, **p<0.01 vs. Aβ1-42 control; n = 12 mice per group mean + SEM.
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Fosgonimeton phase 2/3 LIFT-AD trial after amendments

LEARNINGS FROM ACT-AD INFORM TRIAL DESIGN OPTIMIZATION

POPULATION

N = ~315 participants without concomitant AChEI

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

26-week randomized, double-blind treatment, + optional 48-month OLEX

- Fosgonimeton (40 mg)
- Placebo

TIMELINE: **Enrollment Complete; Topline results expected in 2H24**

ENDPOINTS

PRIMARY

- GST, which combines results of co-key secondary endpoints of cognition and function
- Safety

SECONDARY

- Cognition: ADAS-Cog11
- Function: ADCS-ADL23

EXPLORATORY

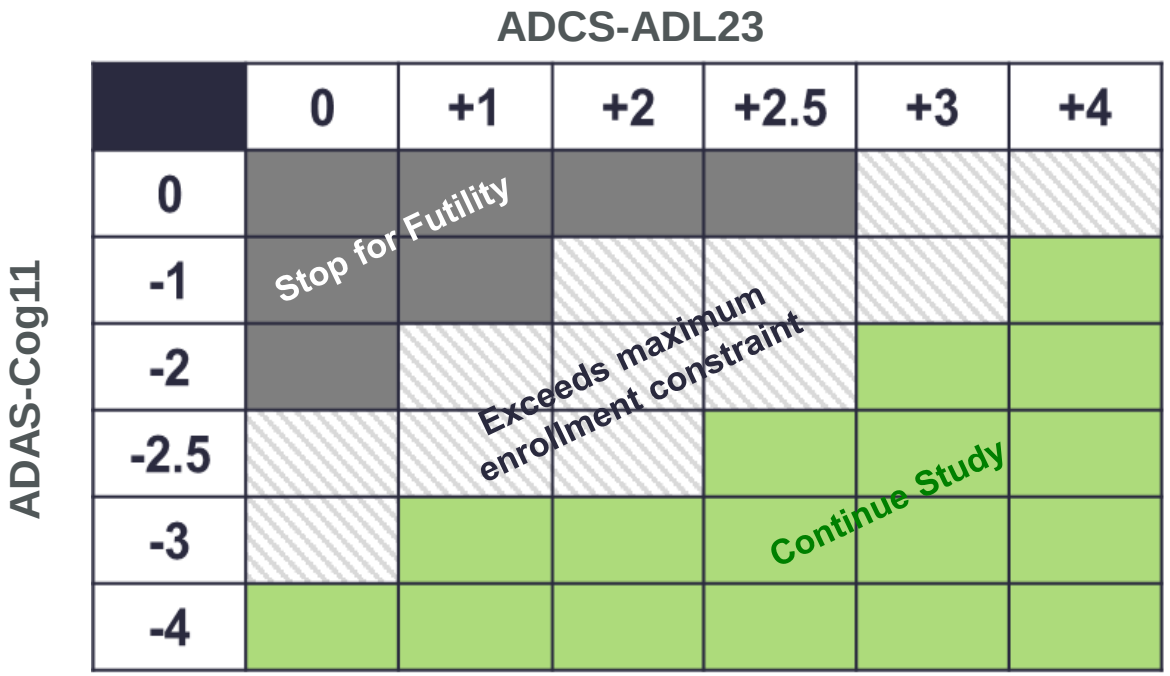
- Additional clinical endpoints and plasma biomarkers

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; MMSA, Mini Mental State Examination

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

DMC ANALYSIS

- Efficacy and futility analysis performed on first 100 patients who completed the six-month trial of fosgonimeton compared with placebo
- DMC Recommendation (Oct 2022): **Continue LIFT-AD Study**

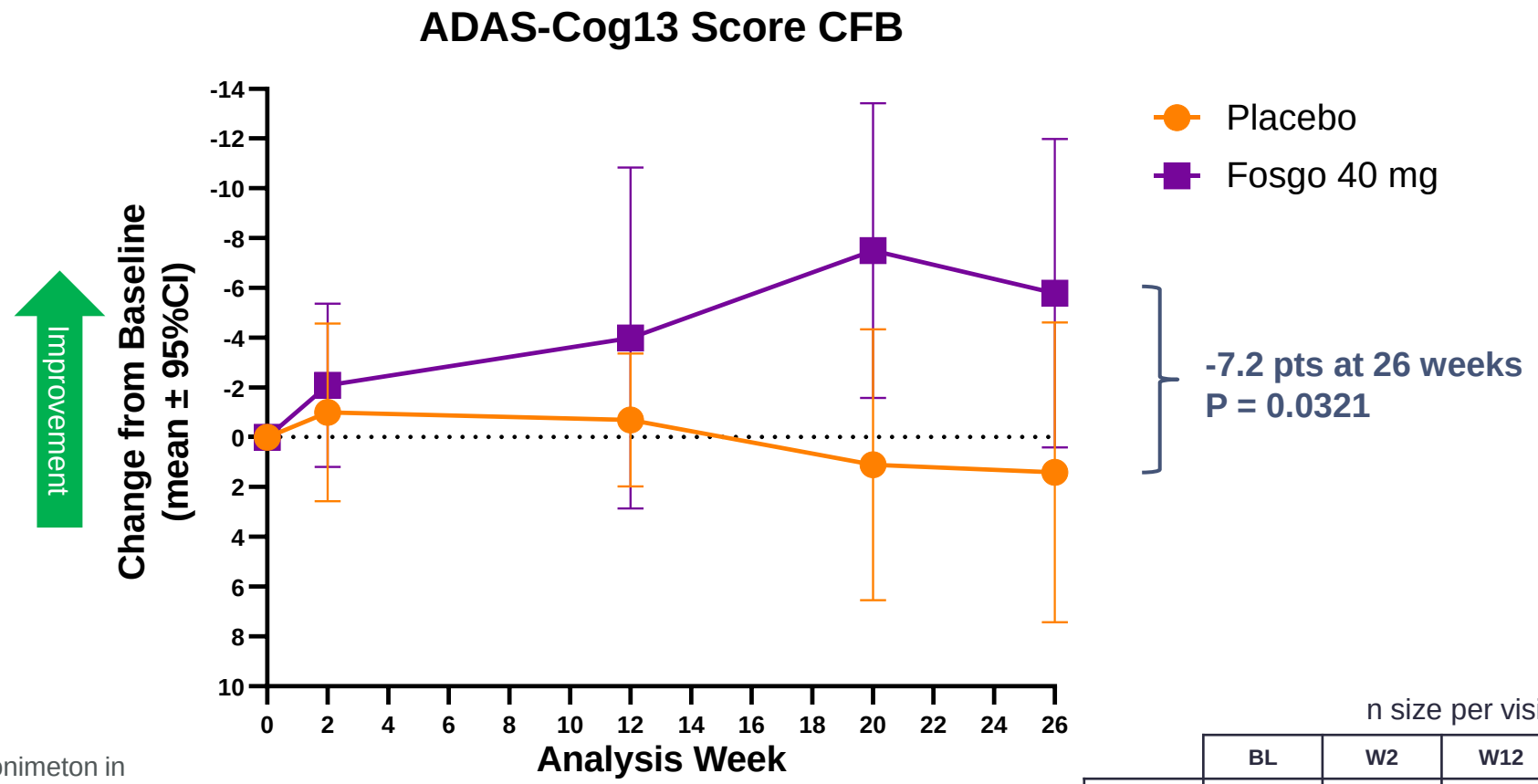


DMC analysis suggests greater potential of LIFT-AD success

[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.

In a different disease setting* fosgo improved cognition with 40mg dose compared to placebo

ADAS-COG13 SCORE CHANGE FROM BASELINE MEAN AND 95% CI – MODIFIED INTENT TO TREAT POPULATION



n size per visit

	BL	W2	W12	W20	W26
Placebo	9	9	9	8	7
40 mg	7	7	7	6	5

* The SHAPE trial investigated fosgonimeton in Parkinson's disease dementia and dementia with Lewy bodies; trial did not meet its primary endpoint.

ADAS-Cog13: Alzheimer's Disease Assessment Scale – Cognitive Subscale 13-item version; CI: Confidence Interval; Baseline is defined as the mean of the pre-dose measurements. Multiple efficacy assessments are averaged if they fall at the same visit. P-value is one-sided

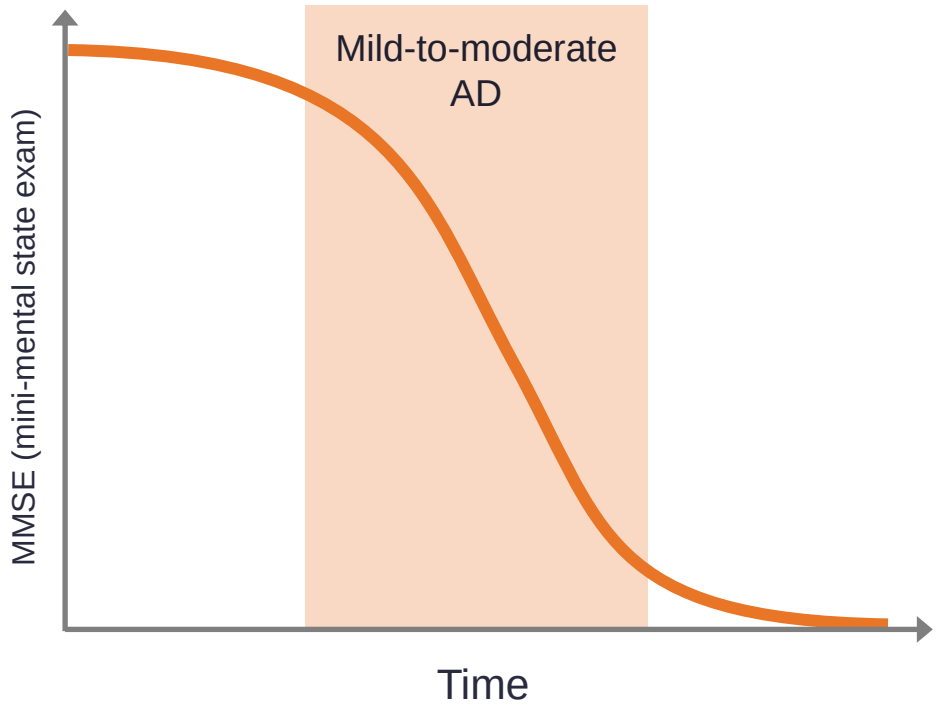
New Treatment Options Needed for Mild-to-Moderate Alzheimer's Disease

Medical need:

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

Few treatment options with only modest effects[3]

Higher financial burden than pre-dementia[4]



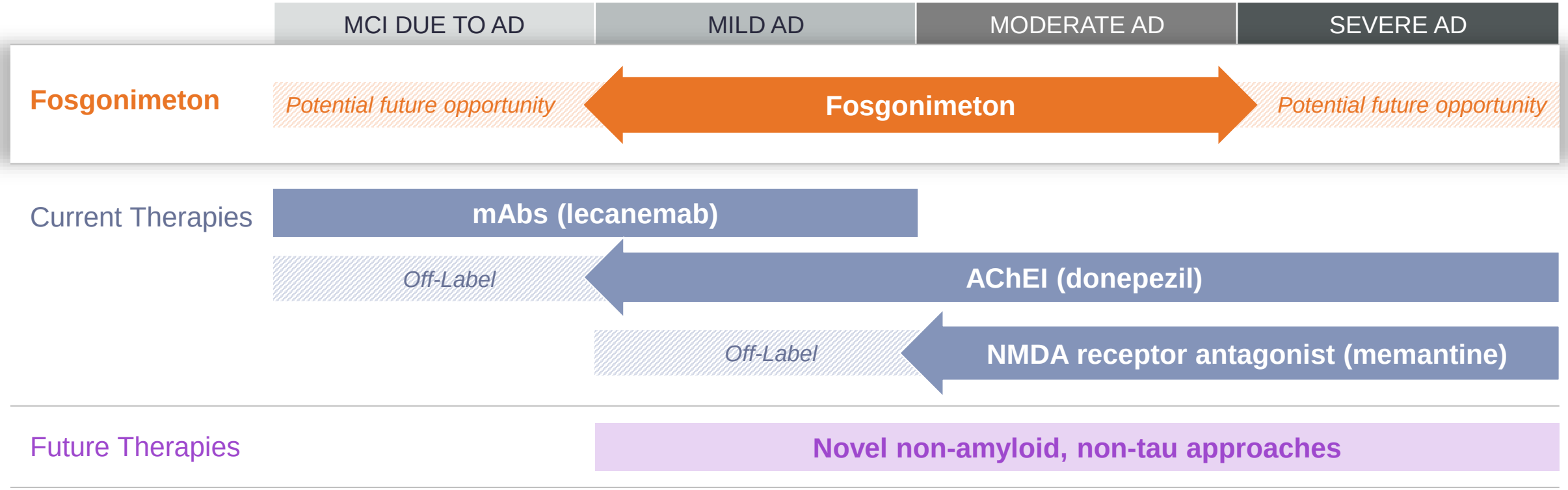
Reduced development risk:

Clinical, syndromal diagnosis is possible[5]

Increased likelihood of tangible placebo decline

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

Significant opportunity in Alzheimer's disease



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

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.

Significant opportunity in Alzheimer's disease

55 million
People living with Alzheimer's dementia today[1]

Multi-Billion $ Market
Despite generic entries

Only Two
New drugs launched since 2003 - two anti-amyloid antibodies

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]

Mild to Moderate comprises 81% of all patients with Alzheimer's Disease

78.5% of these patients receive Rx therapies[3]

Significant opportunity for fosgonimeton

Market research suggests favorable reaction and receptivity to fosgonimeton base case target product profile[4]

[1] https://www.alzint.org/about/dementia-facts-figures/
[2] https://www.alz.org/media/documents/alzheimers-facts-and-figures.pdf
[3] https://www.nia.nih.gov/news/half-alzheimers-disease-cases-may-be-mild
[4] ClearView Healthcare Partners Market Research Analysis

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Strong rationale to advance fosgonimeton

SIGNIFICANT OPPORTUNITY TO TRANSFORM THE TREATMENT PARADIGM FOR NEURODEGENERATIVE DISEASES

- Disease modifying
- Improves cognition
- Improves function
- Reduces inflammation
- Prevents nerve cell death
- Favorable safety and tolerability profile
- Risk mitigated Ph 2/3 LIFT-AD following interim analysis

Differentiated and Risk Mitigated

+

- High unmet need
- Enormous potential market

Favorable external landscape

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Amyotrophic Lateral Sclerosis

ATH-1105

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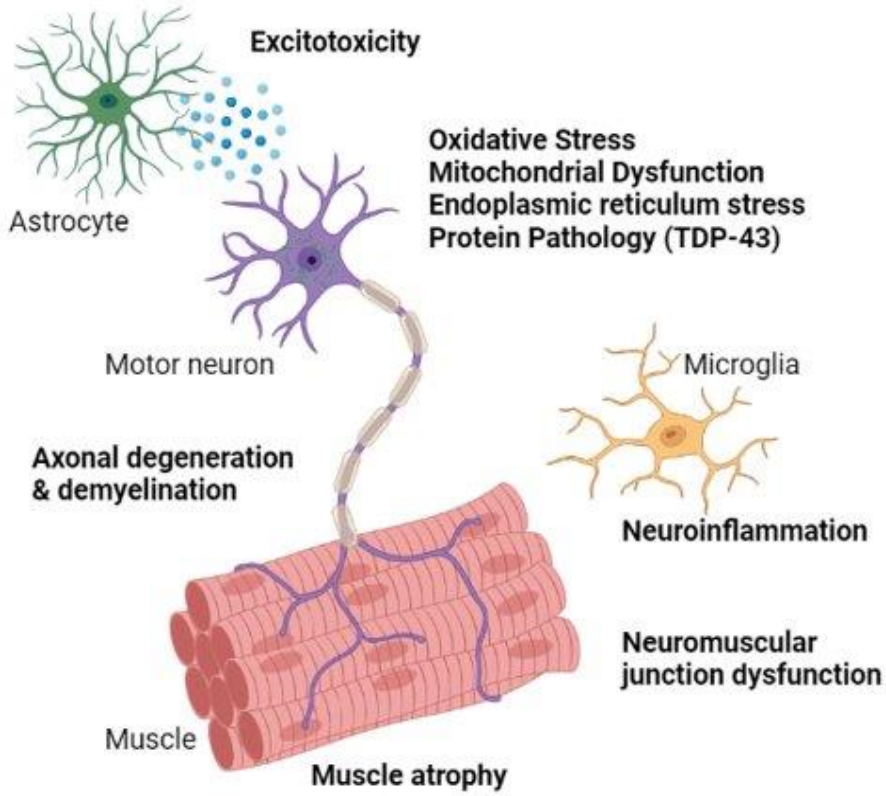
Positive modulation of HGF as a potential treatment for ALS

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



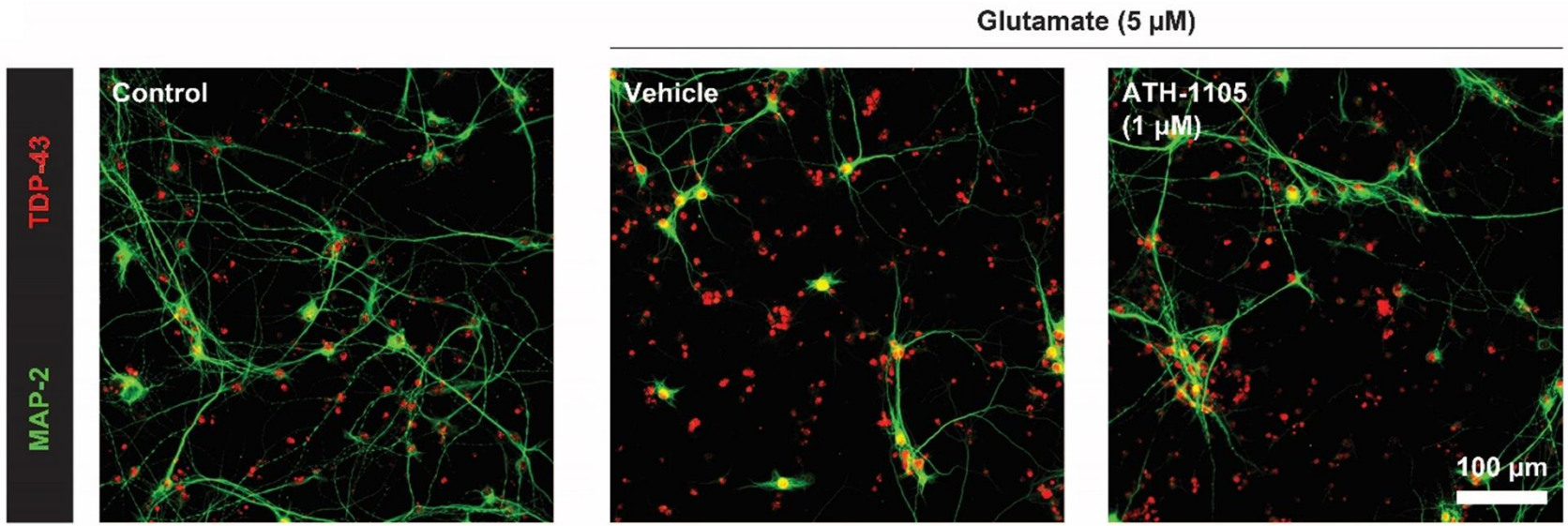
Promotion of HGF/MET activity has been reported to have beneficial effects in preclinical models of ALS

- HGF delays disease progression in ALS animal models[2,3]
- HGF reduces muscle impairment and motor neuron loss in an ALS mouse model[4]

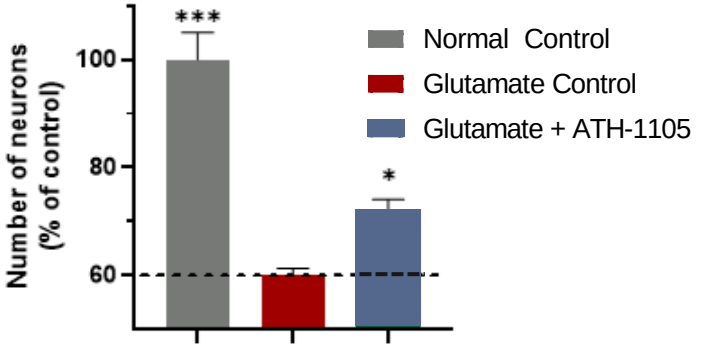
[1]Scotter et al., *Neurotherapeutics* 2015.
[2]Ishigaki et al., *J Neuropathol Exp Neurol* 2007.
[3]Lee et al., *Acta Neuropathol Commun* 2019.
[4]Vallarola et al., *Int J Mol Sci* 2020.

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

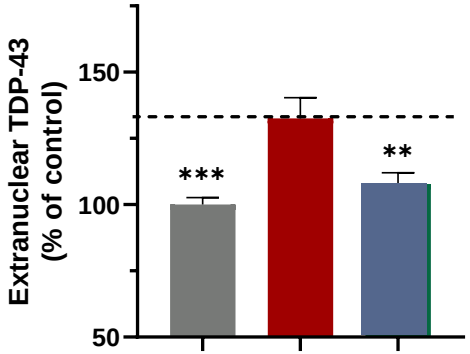
GLUTAMATE CHALLENGE MODEL IN MOTOR NEURON CULTURES



Motor neuron survival following glutamate challenge



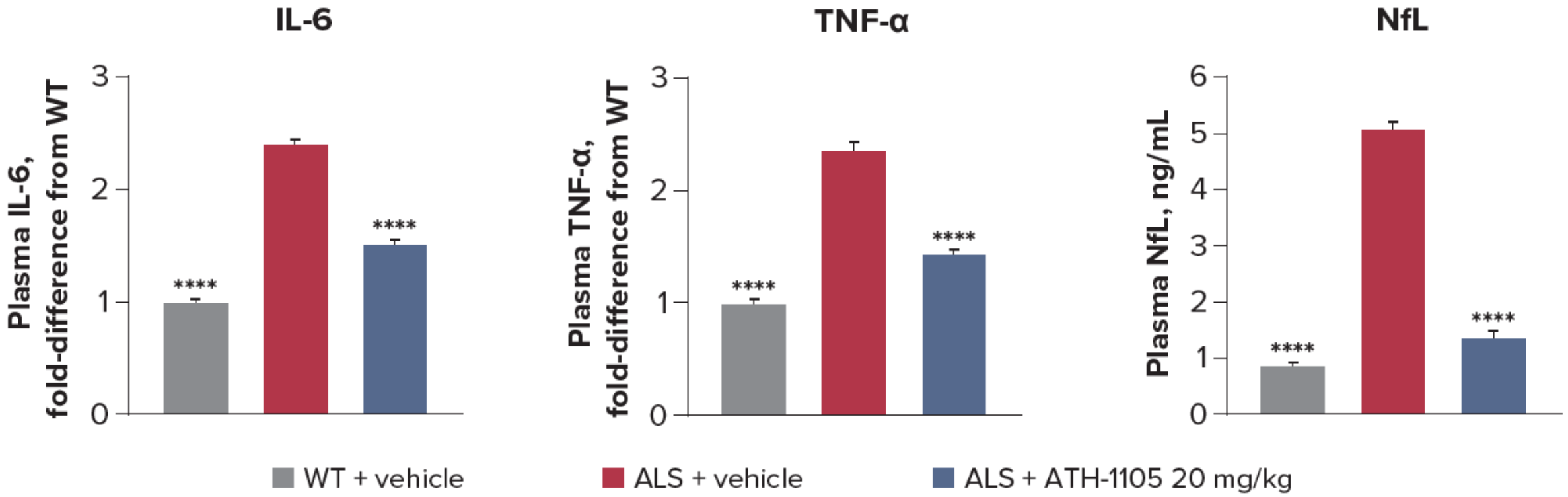
Glutamate-induced TDP-43 accumulation in spinal motor neurons



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.

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

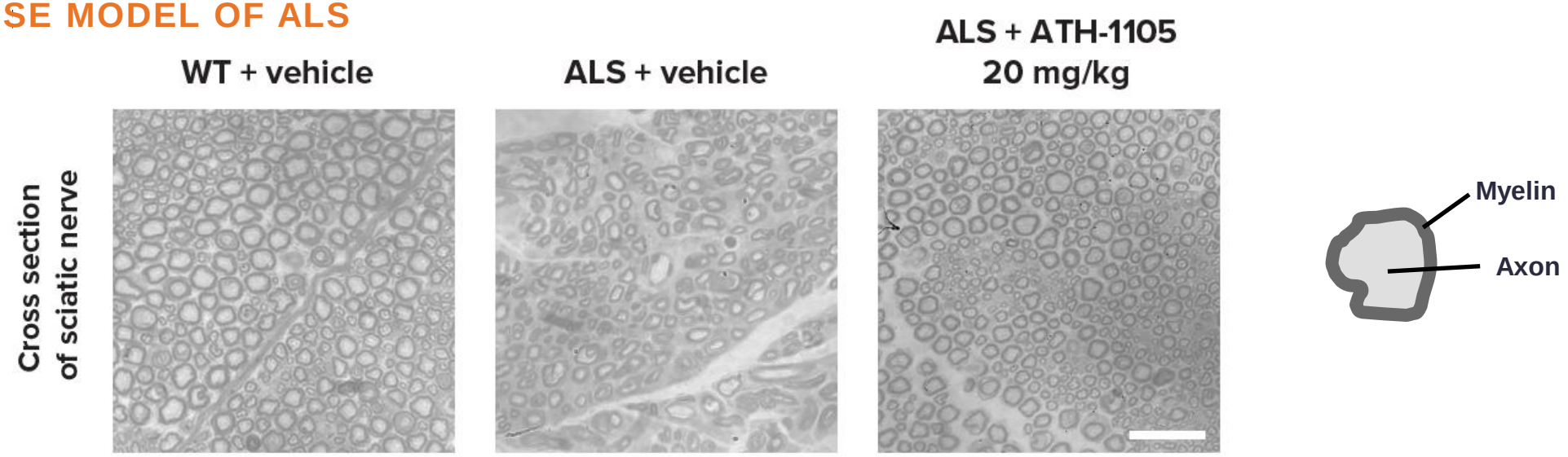
TDP-43 MOUSE MODEL OF ALS

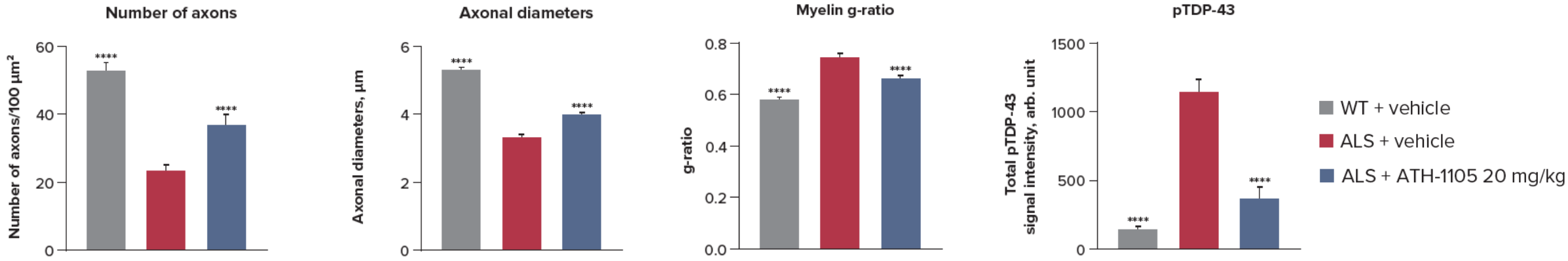


Data shown is from plasma collected following 2 months of ATH-1105 or vehicle treatment. Data presented as mean ± SEM.
Statistical significance was determined by 1-way ANOVA with Dunnett's test versus ALS + vehicle. ****p < 0.0001.
ALS, amyotrophic lateral sclerosis; IL-6, interleukin 6; NfL, neurofilament light chain; TDP-43, TAR DNA-binding protein 43; TNF-α, tumor necrosis factor alpha; WT, wild-type.

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.

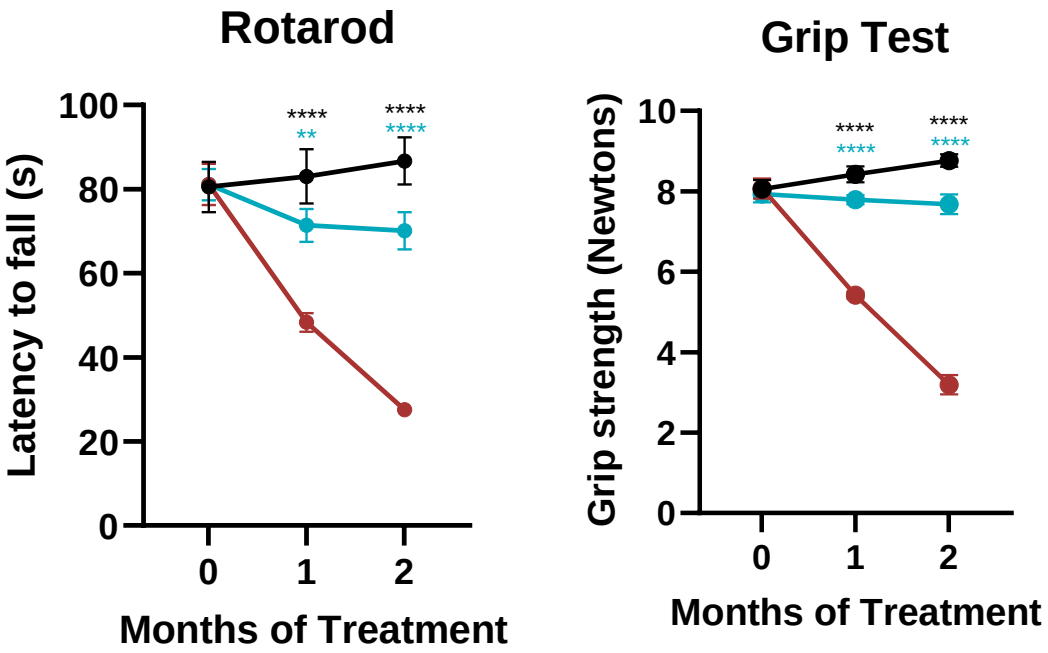
Scale bar: 10 µm, applies to all images.

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

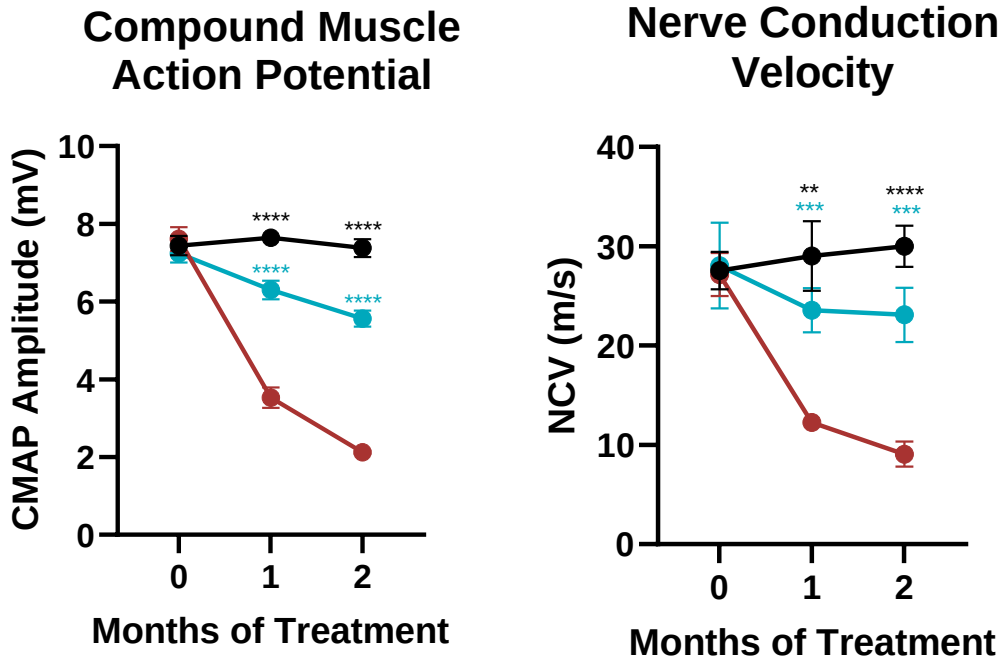
Function: ATH-1105 prevents nerve and motor function decline

TDP-43 MOUSE MODEL OF ALS

Motor Function



Nerve Function



WT + vehicle ALS + vehicle ALS + ATH-1105 20 mg/kg

Model: Prp-TDP43A315T mouse model of ALS

Data presented as mean ± SEM

Statistics applied: 2-way ANOVA with the Dunnett test versus ALS + vehicle. *p < 0.05 ,**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

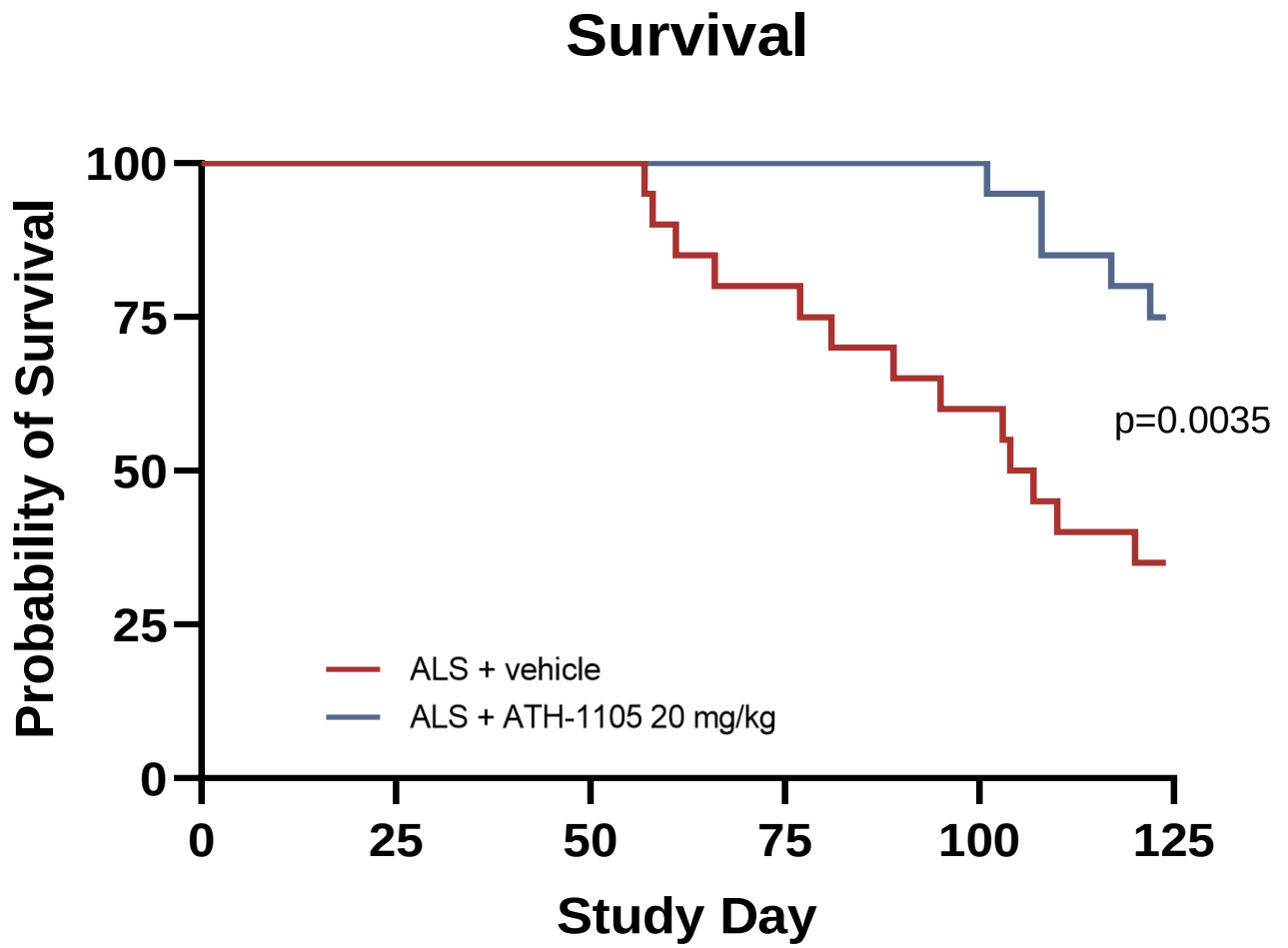
TDP-43 MOUSE MODEL OF ALS

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 40% of those cases in the US

Six[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 addresses a familial form of ALS accounting for <3% of all cases (recently received accelerated approval on the basis of NfL)

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

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[1]ALS Association
[2]Berdynski, et al., *Nature* 2022.

Corporate

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Athira management team with significant CNS product development and approval experience

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Moving forward

- ✓ Independent, unblinded interim analysis of fosgonimeton Phase 2/3 LIFT-AD
- ✓ LIFT-AD completed enrollment

→ Topline results expected in 2H2024

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

→ Topline results reported 4Q2023; evaluating next steps

- ✓ ATH-1105 prolonged survival, delayed time to first mortality, and demonstrated consistent improvements in motor function, nerve function, biomarkers and nerve morphology in a transgenic mouse model of ALS

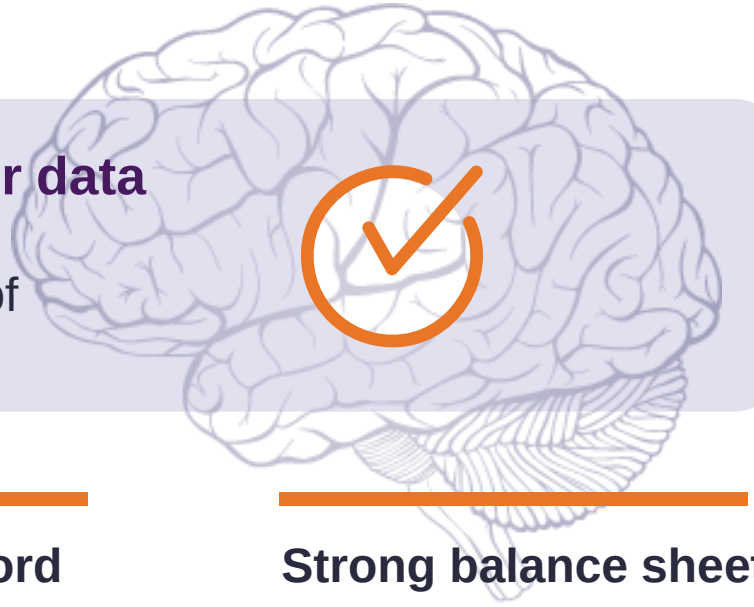
→ IND filed

→ Initiate first-in-human Phase 1 testing in 2Q2024

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

Well positioned to lead with innovative approach to battling neurodegenerative diseases

Consistent and correlative preclinical, clinical and biomarker data showing that enhancing HGF neurotrophic system has potential 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 key inflection points (~$122M in cash at end of 1Q2024)

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