Athira PHARMA

Corporate Presentation

January 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, and other neurodegenerative diseases, such as amyotrophic lateral sclerosis, the anticipated reporting of data, the potential learnings from 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, preliminary and unaudited estimates of our cash, cash equivalents, and investments, and the impact of pandemics on our business and operations.

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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

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; trial well powered to meet primary endpoint based on anticipated effect size
  - 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

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* The exploratory SHAPE trial did not meet its primary endpoint..
ALS, amyotrophic lateral sclerosis; HGF, hepatocyte growth factor.

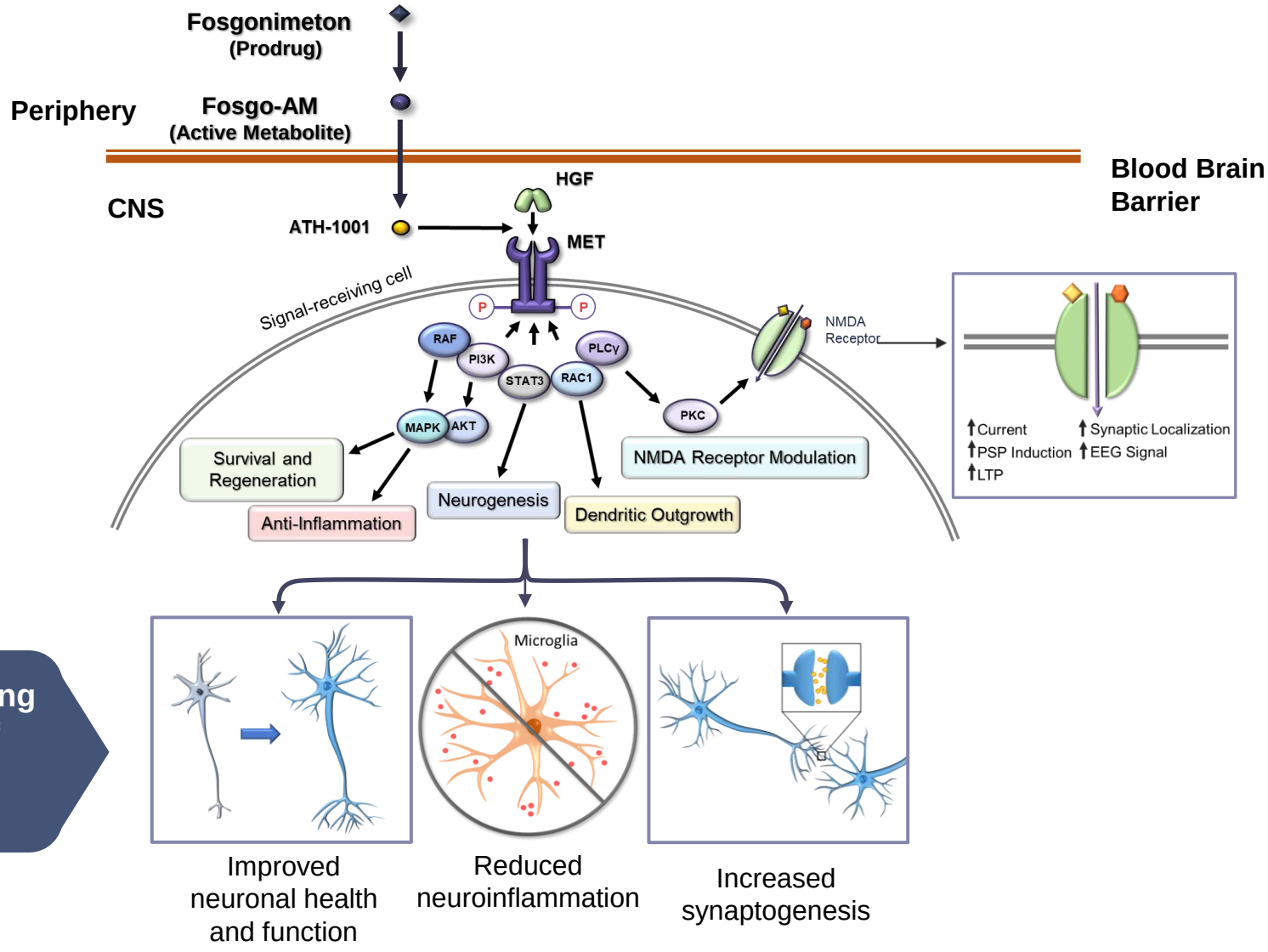
HGF is a critical neuroprotective system

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

HGF/MET signaling is a multimodal signaling pathway

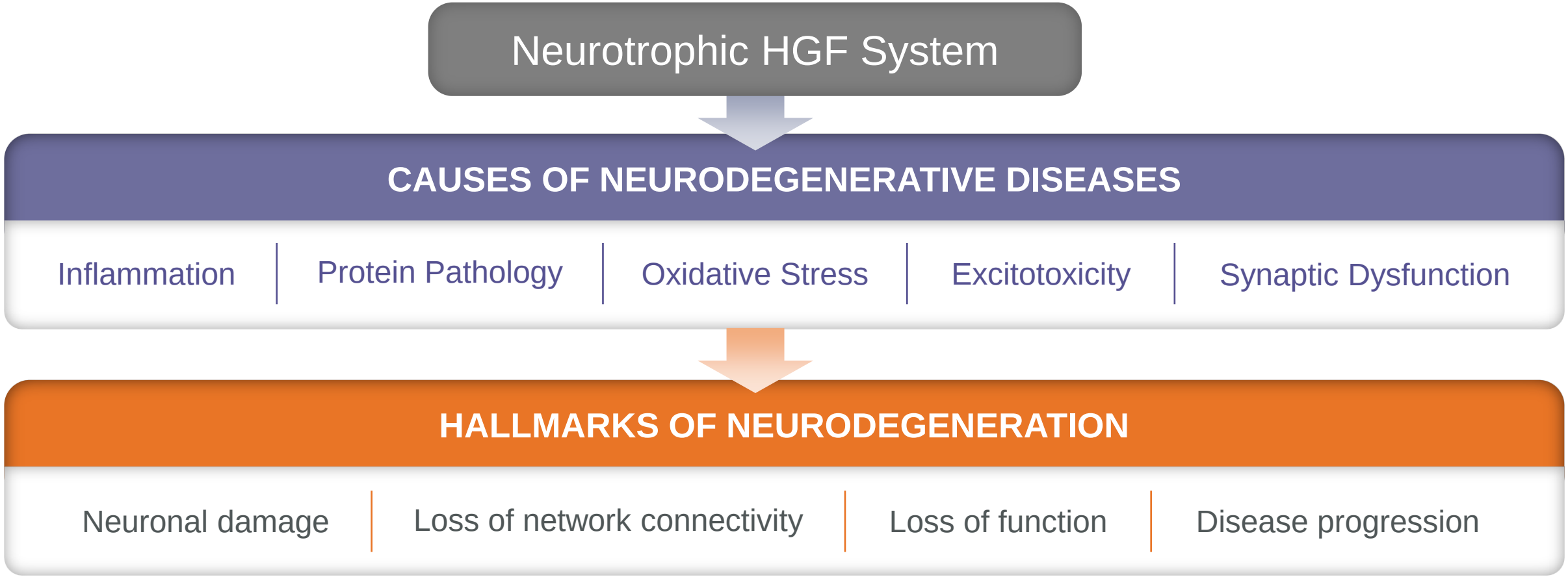
- Activation of MET leads to activation of multiple kinase signaling cascades
- These signaling events influence neuron health, morphology, inflammatory response, and function

Fosgonimeton-enhanced HGF/MET signaling has the potential to lead to promotion of multiple beneficial cell behaviors by activating a range of pathways



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

HGF system impacts multifactorial complex pathologies that lead to neurodegeneration



Positive modulators of the HGF system could be therapeutic

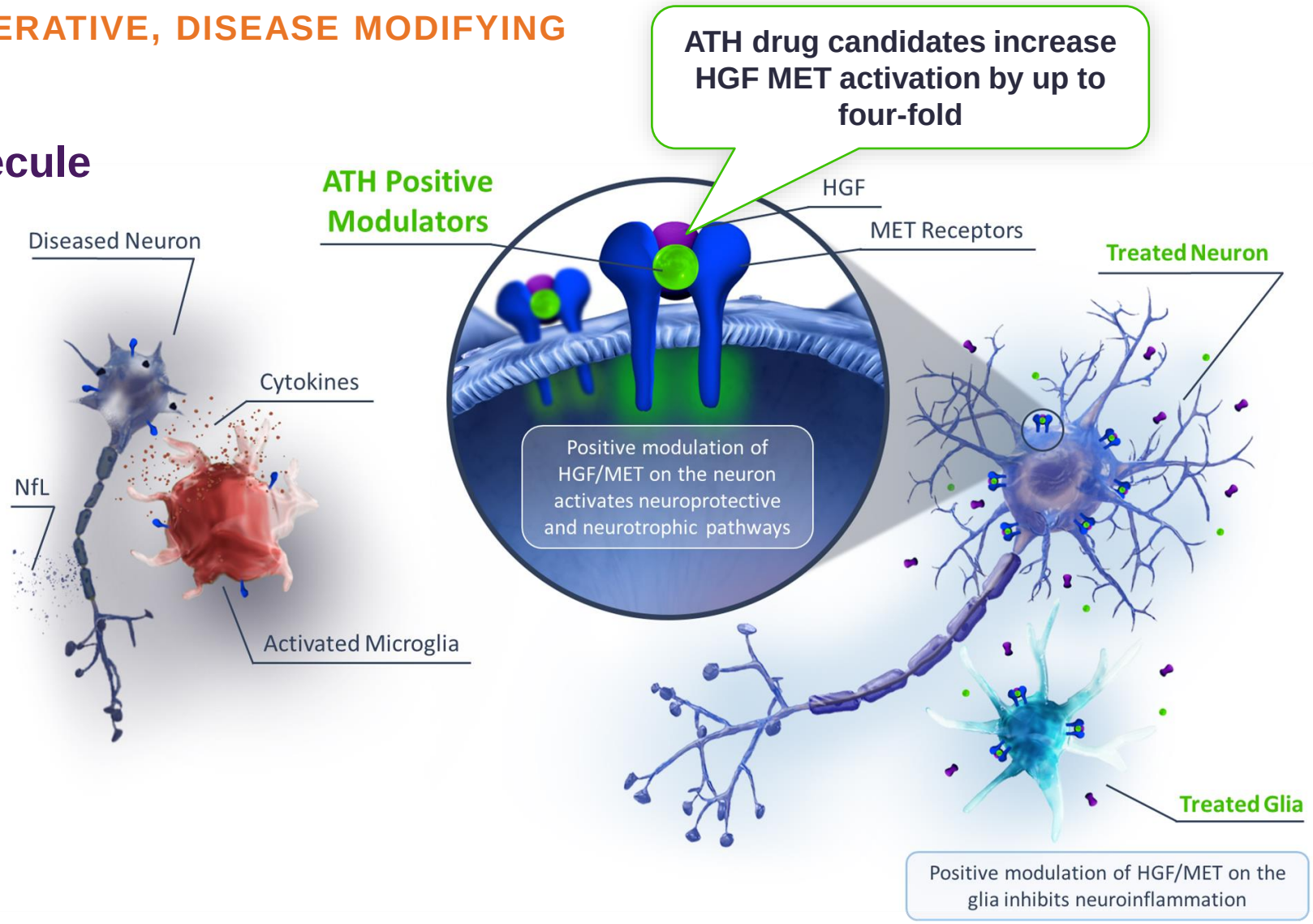
MULTIMODAL, PROTECTIVE, REGENERATIVE, DISEASE MODIFYING

Potential first-in-class small molecule drug candidates

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

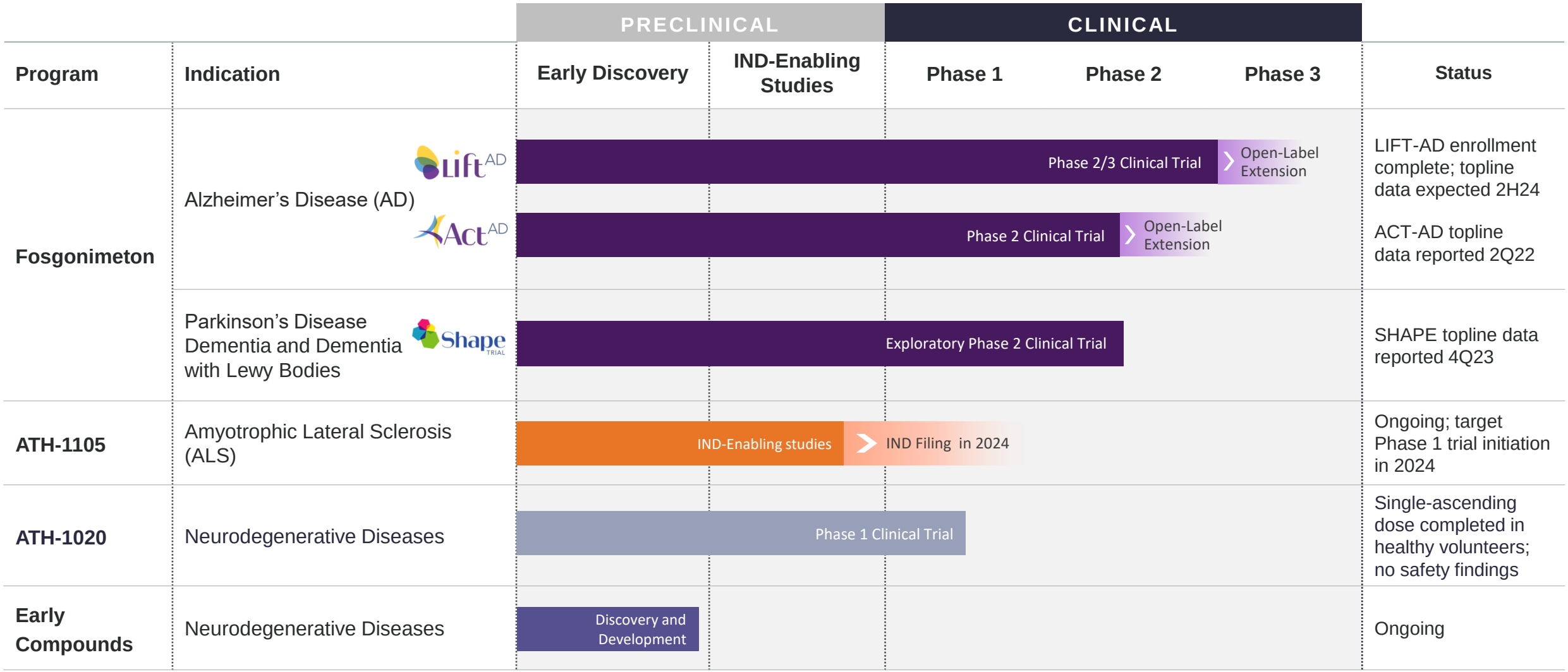
Mechanism of action may

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



HGF, hepatocyte growth factor.

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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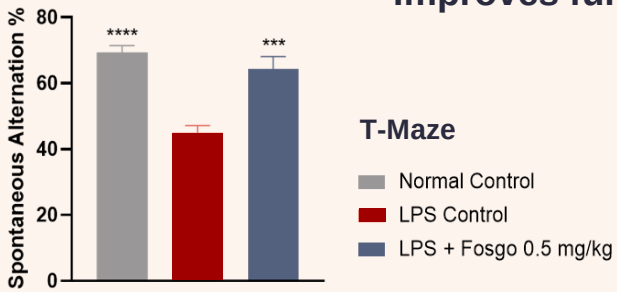
Fosgonimeton protects and repairs neuronal networks in preclinical models

MULTIMODAL APPROACH WITH POTENTIAL FOR DISEASE MODIFICATION

REPAIR | **PROTECT**

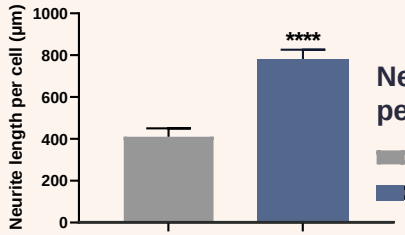
Improves function



T-Maze

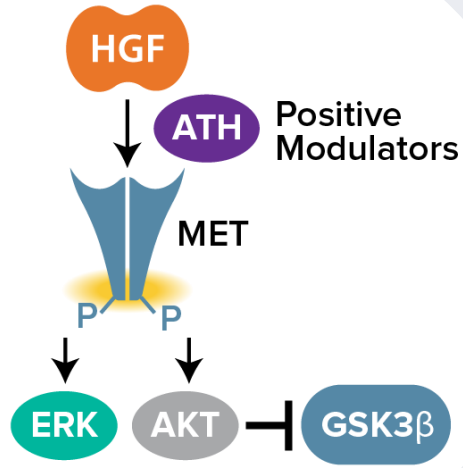
- Normal Control
- LPS Control
- LPS + Fosgo 0.5 mg/kg

Neurotrophic

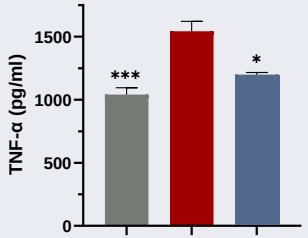


Neurite length per cell

- Control
- Fosgo-AM



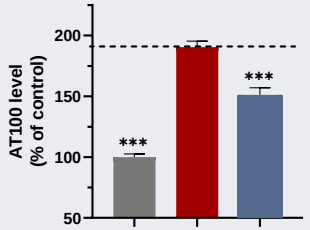
Anti-inflammatory



LPS-induced TNFα release in immune cells

- Normal Control
- LPS Control
- LPS + Fosgo-AM

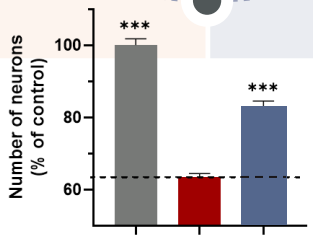
Reduces protein pathology



Aβ-induced tau phosphorylation in cortical neurons

- Normal Control
- Aβ Control
- Aβ + Fosgo-AM

Neuroprotective



Aβ-induced cortical neuron death

- Normal Control
- Aβ Control
- Aβ + Fosgo-AM

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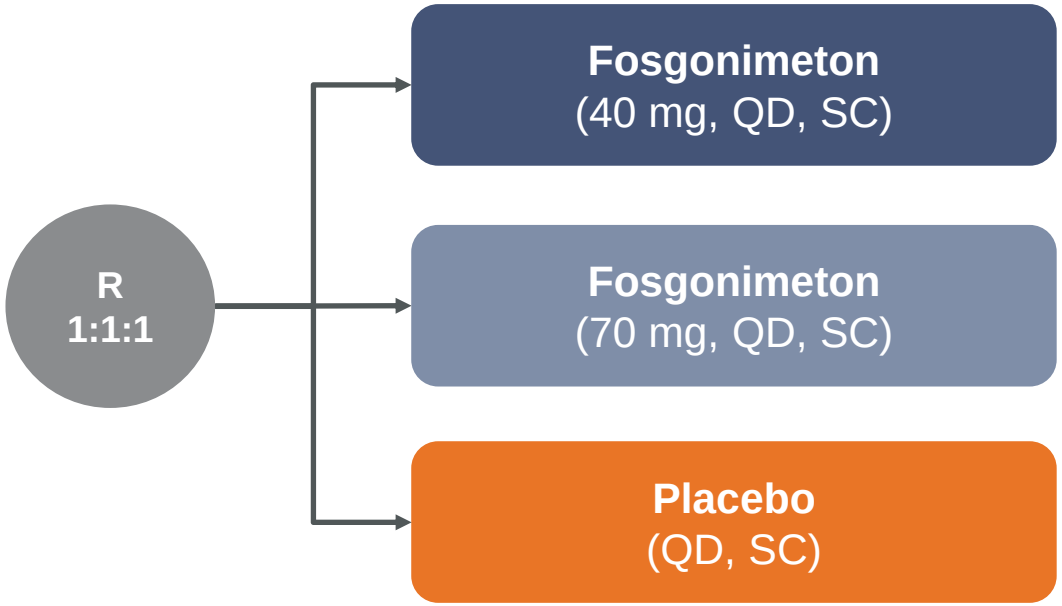
Fosgonimeton exploratory phase 2 trial in mild-to-moderate Alzheimer's disease*

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



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

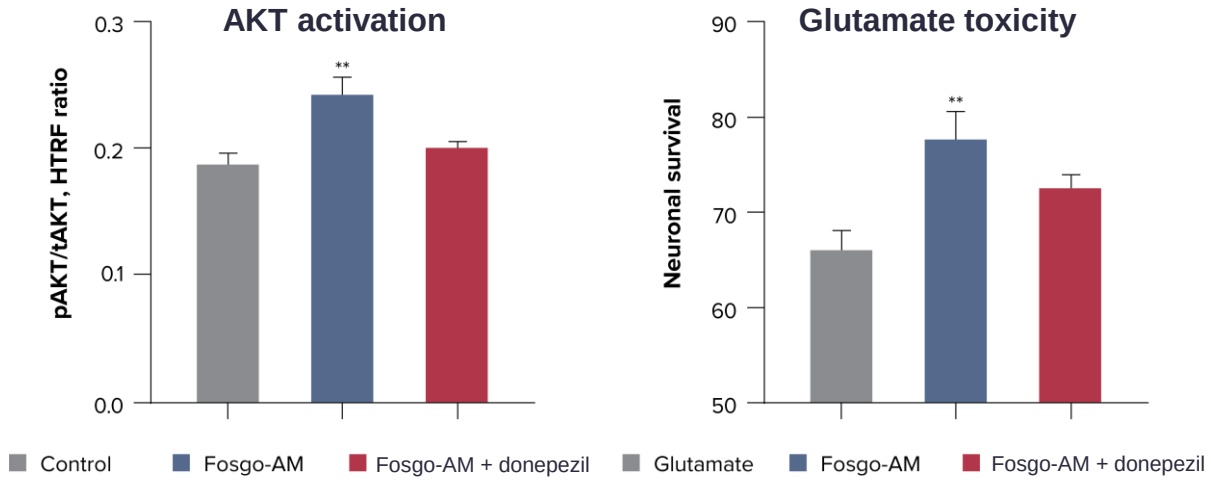
* Trial did not meet its primary endpoint.
AChEI, acetylcholinesterase inhibitor; AD, Alzheimer's disease; ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living; ADCS-CGIC, Alzheimer's Disease Cooperative Study–Clinical Global Impression of Change; ERP P300, event-related potential; OLEX, open-label extension; QD, once daily; R, randomization; SC, subcutaneous

Neuroprotective effects of fosgonimeton are reduced with exposure to AChEIs

Preclinical

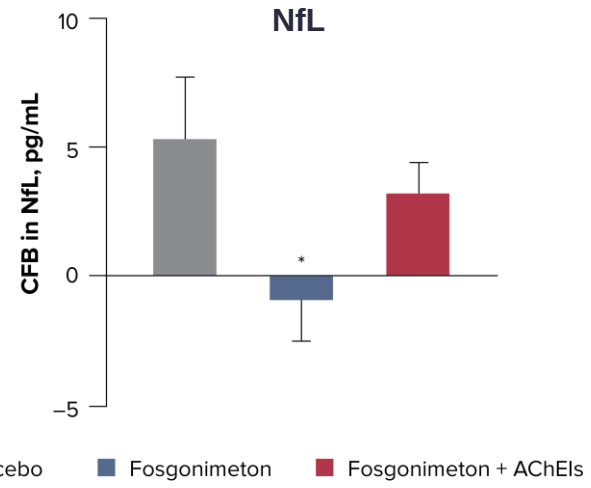
- Combination of fosgo-AM with donepezil interferes with fosgo-AM-induced AKT activation
- Neuroprotective effects of fosgo-AM are reduced when combined with donepezil

  Likely the result of observed decrease in fosgo-AM-induced AKT activation



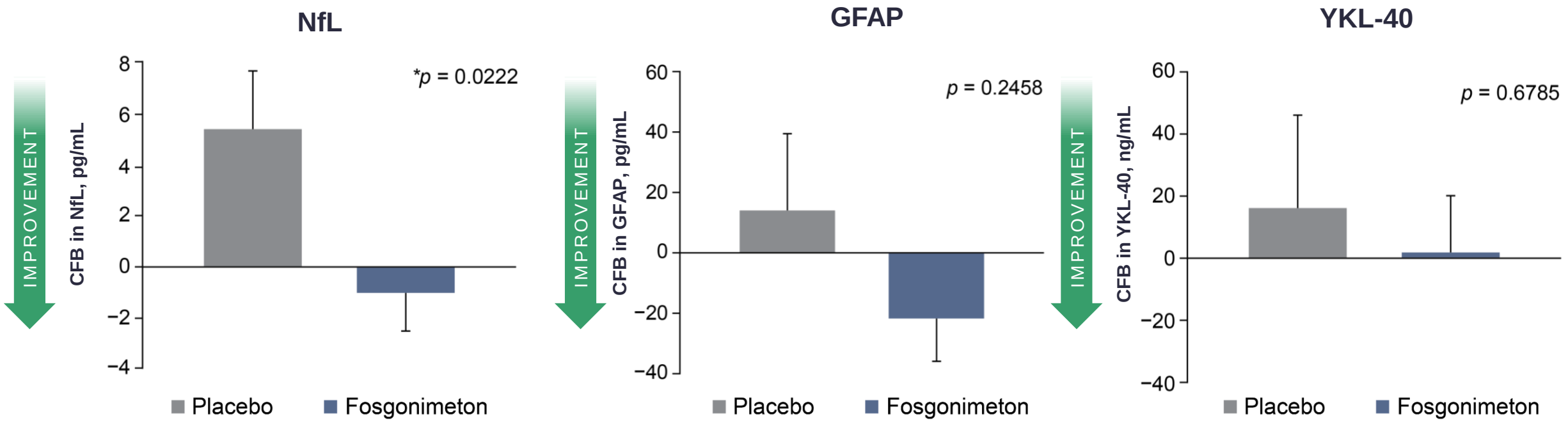
Clinical

- 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; NfL, neurofilament light chain.
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

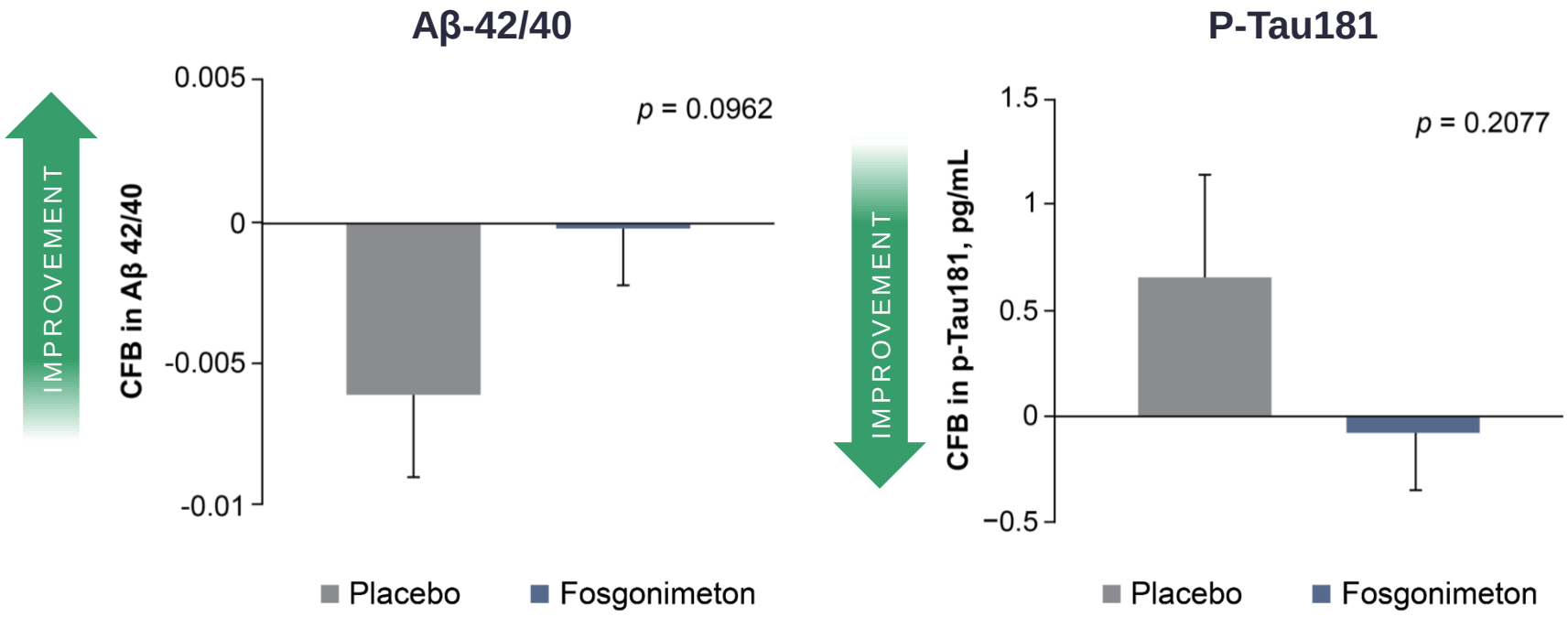
- GFAP and YKL-40 are markers of neuroinflammation
- Magnitude of decrease is encouraging in this continuously progressive condition

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

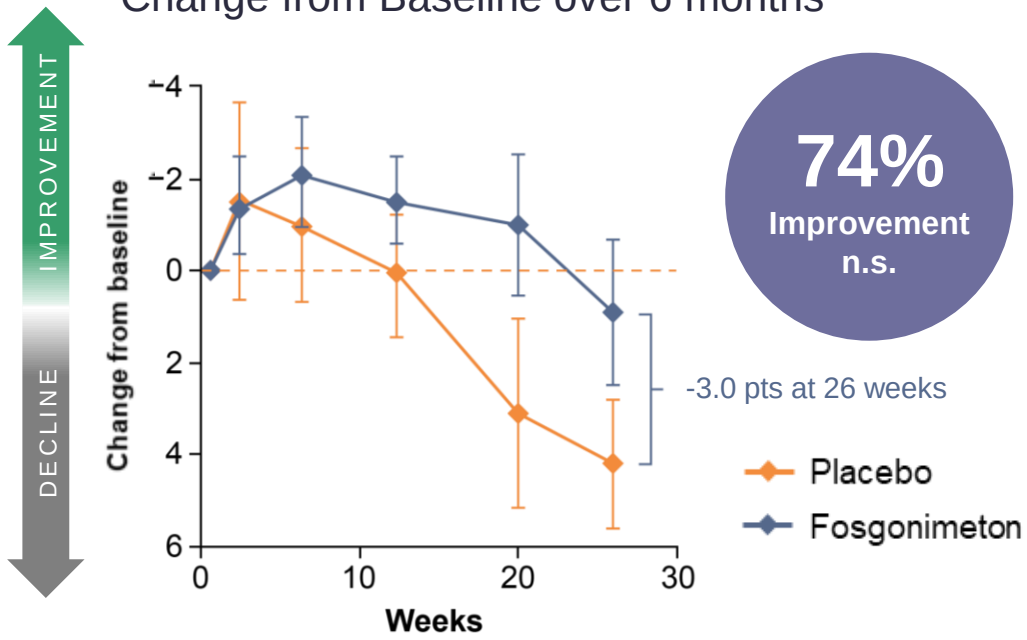
41% | **+1.7** points n.s.

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.

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 30-month OLEX

- Fosgonimeton (40 mg)
- Placebo

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

ENDPOINTS

PRIMARY

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

SECONDARY

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

EXPLORATORY

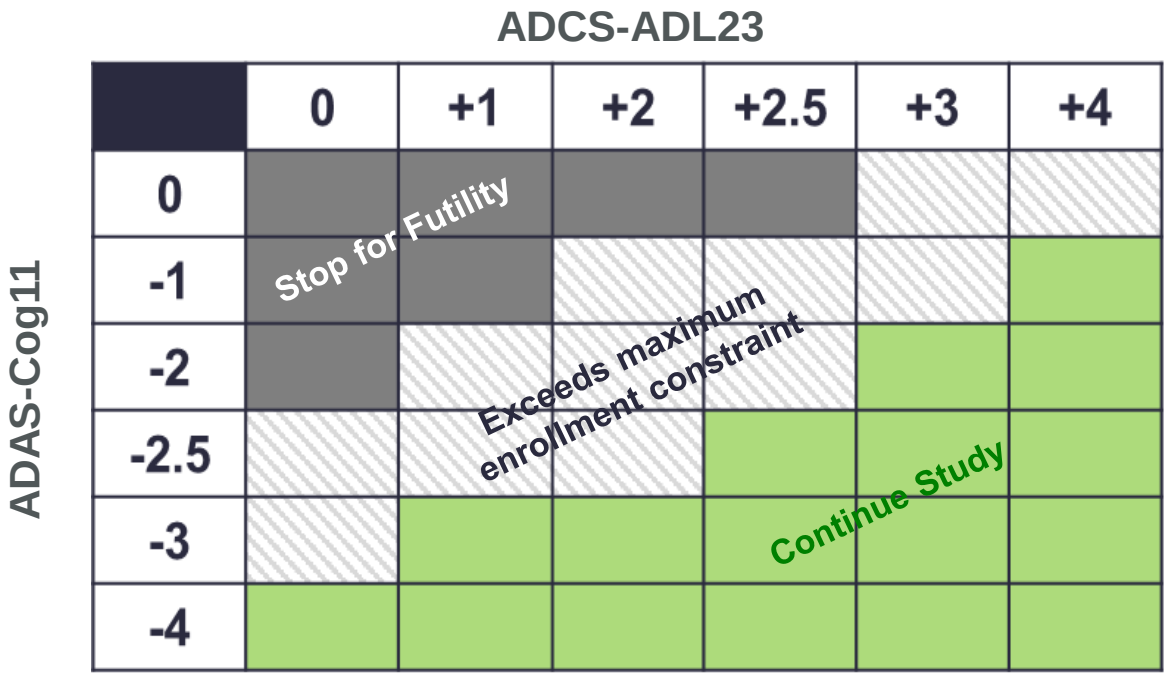
- Additional clinical endpoints and plasma biomarkers

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**

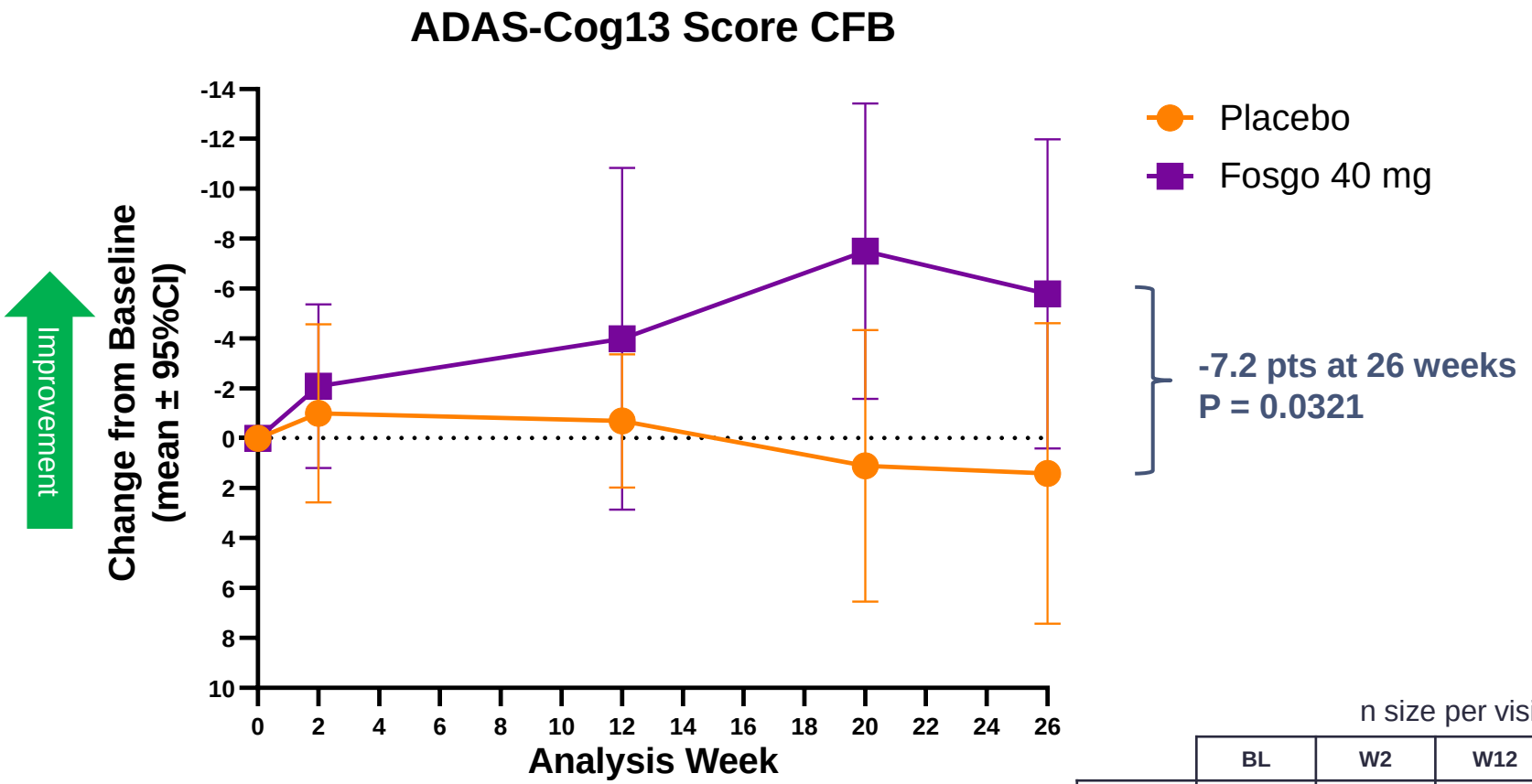


DMC analysis suggests greater potential of LIFT-AD success

[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

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

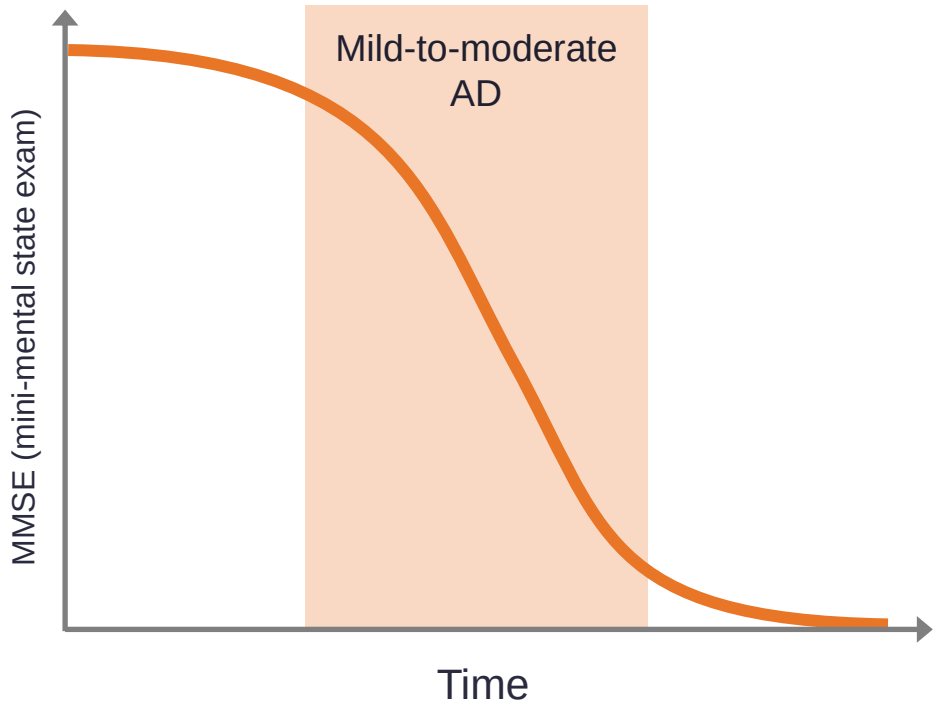
New Treatment Options Needed for Mild-to-Moderate Alzheimer's Disease

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]



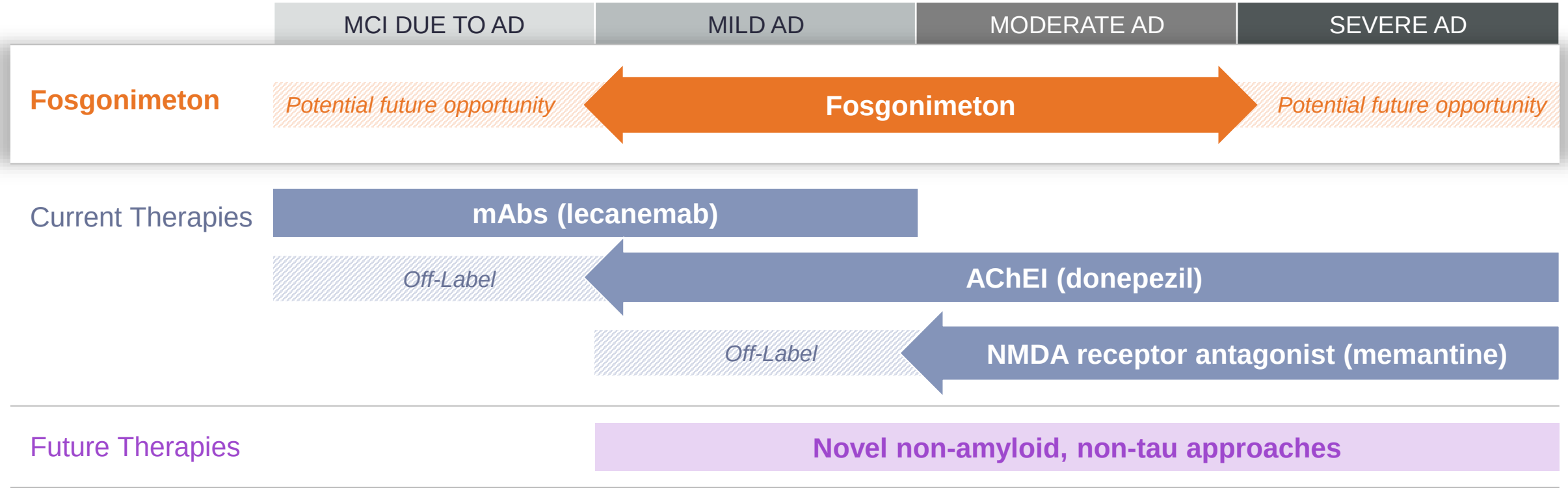
Reduced development risk:

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 – preclinically and clinically
- Improves function – preclinically and clinically
- Reduces inflammation – preclinically and clinically
- Prevents nerve cell death – preclinically
- 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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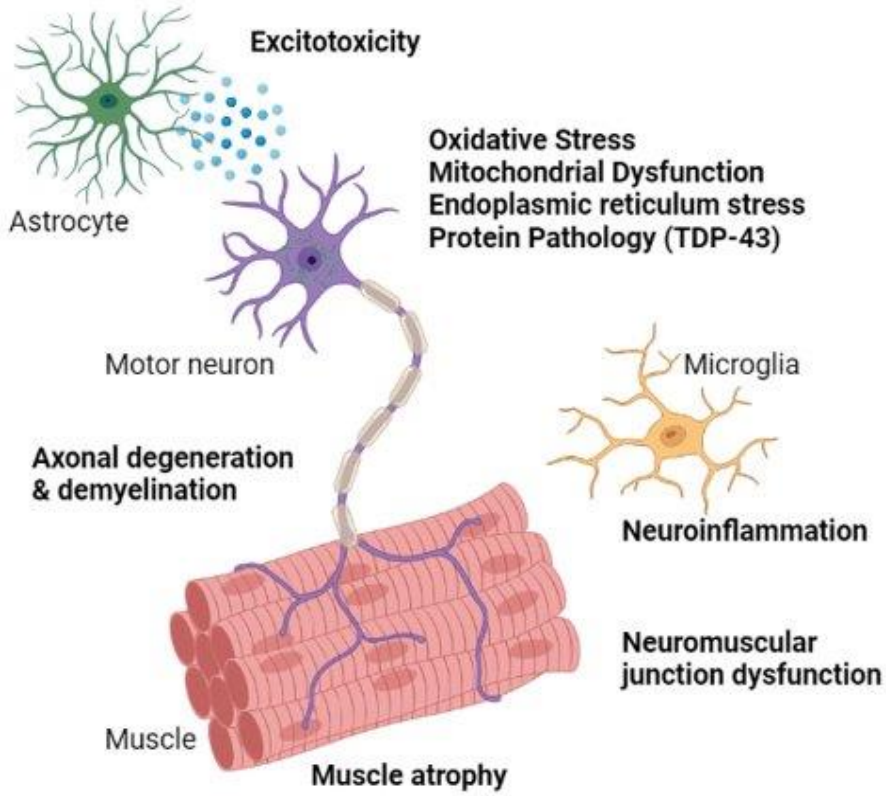
Positive modulation of HGF as a potential treatment for ALS

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



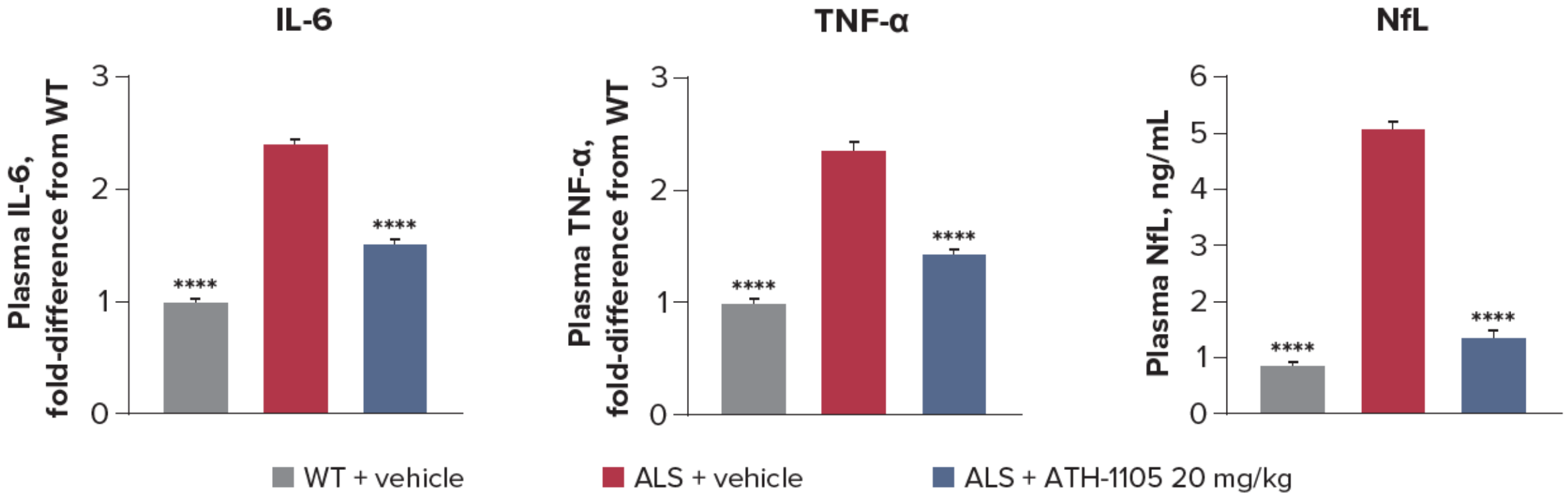
Promotion of HGF/MET activity has been reported to have beneficial effects in preclinical models of ALS

- 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.

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

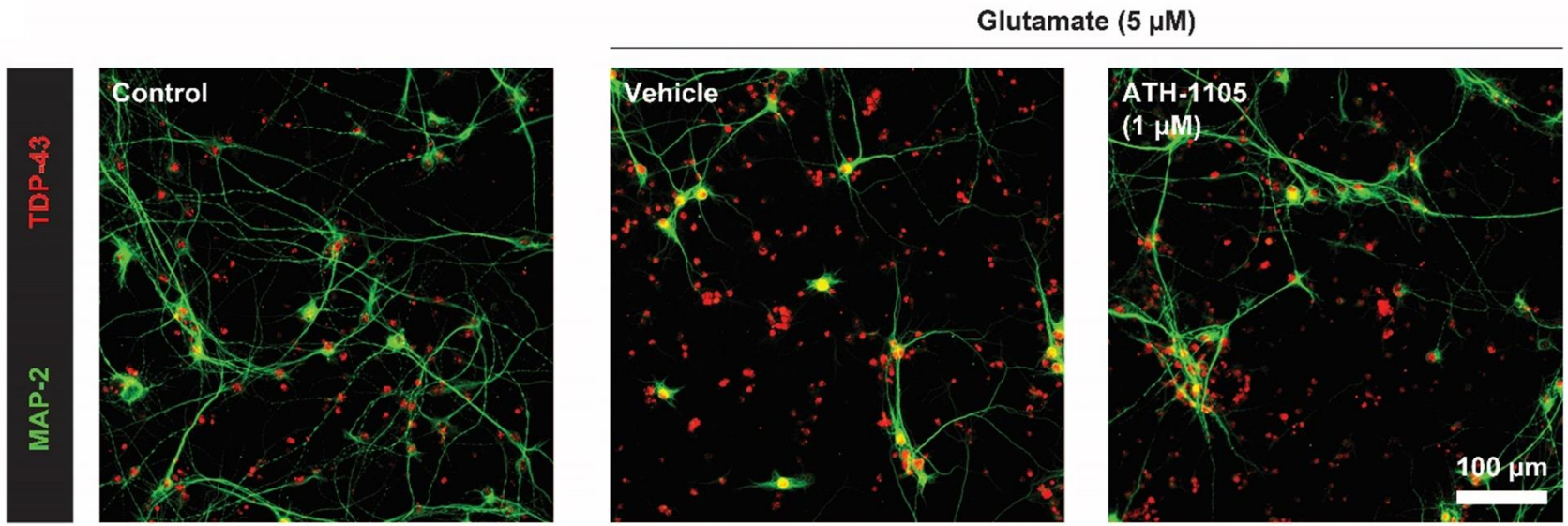
TDP-43 MOUSE MODEL OF ALS



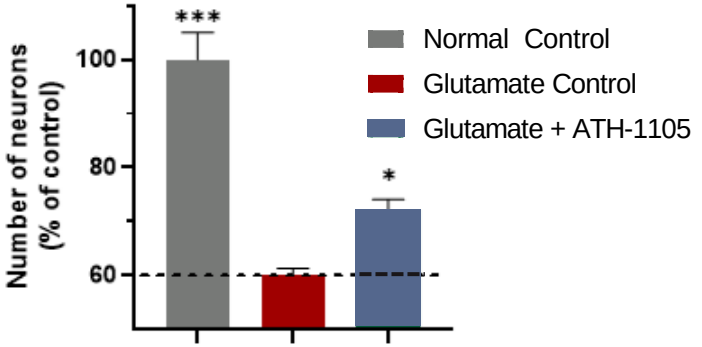
Data presented as mean ± SEM.
Statistical significance was determined by 1-way ANOVA with Dunnett's test versus ALS + vehicle. ****p < 0.0001.
ALS, amyotrophic lateral sclerosis; IL-6, interleukin 6; NfL, neurofilament light chain; TDP-43, TAR DNA-binding protein 43; TNF-α, tumor necrosis factor alpha;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

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

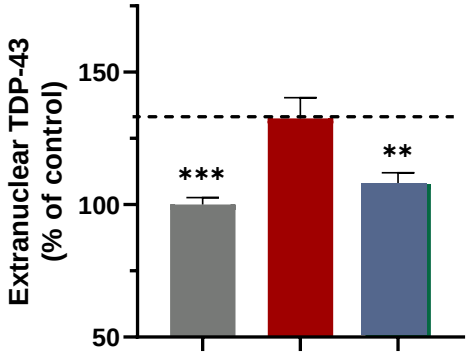
GLUTAMATE CHALLENGE MODEL IN MOTOR NEURON CULTURES



Motor neuron survival following glutamate challenge



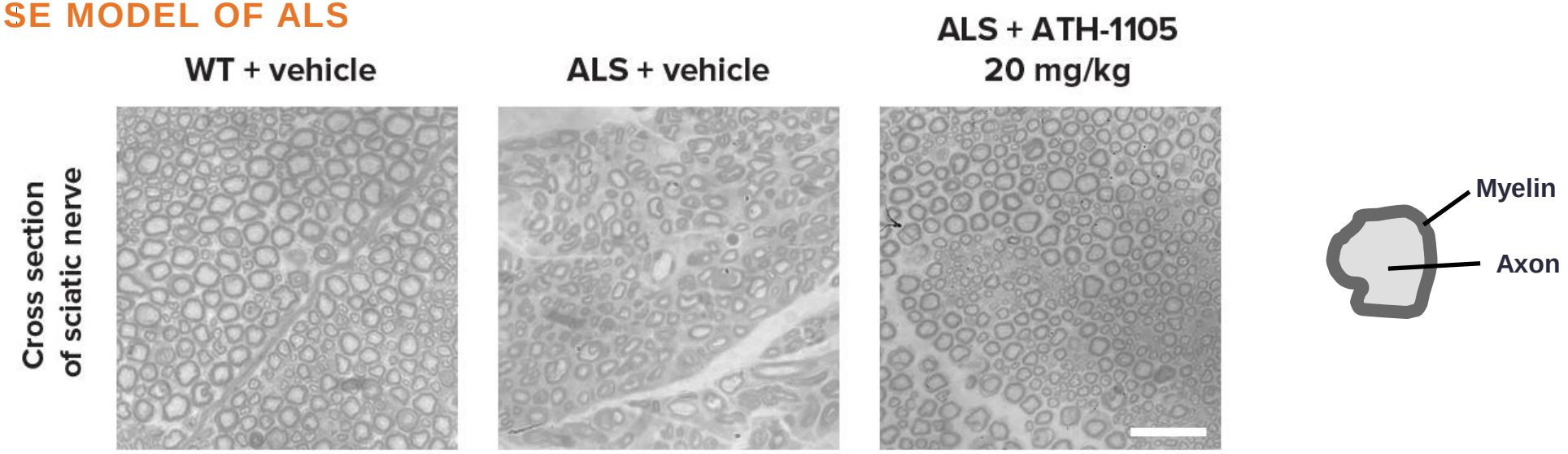
Glutamate-induced TDP-43 accumulation in spinal motor neurons

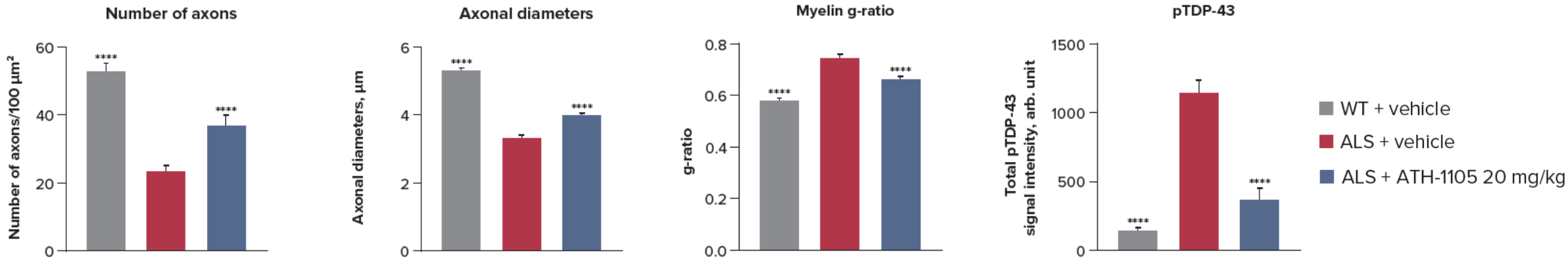


Primary rat spinal motor neurons. Cultures treated with vehicle control or 5 µM glutamate.
Data presented as mean ± SEM. Statistics applied: 1-way ANOVA with Fisher least significant difference test. *p < 0.05, **p < 0.01, ***p < 0.001 versus Glutamate Control; n = 6.
Scale bar: 100 µm, applies to all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





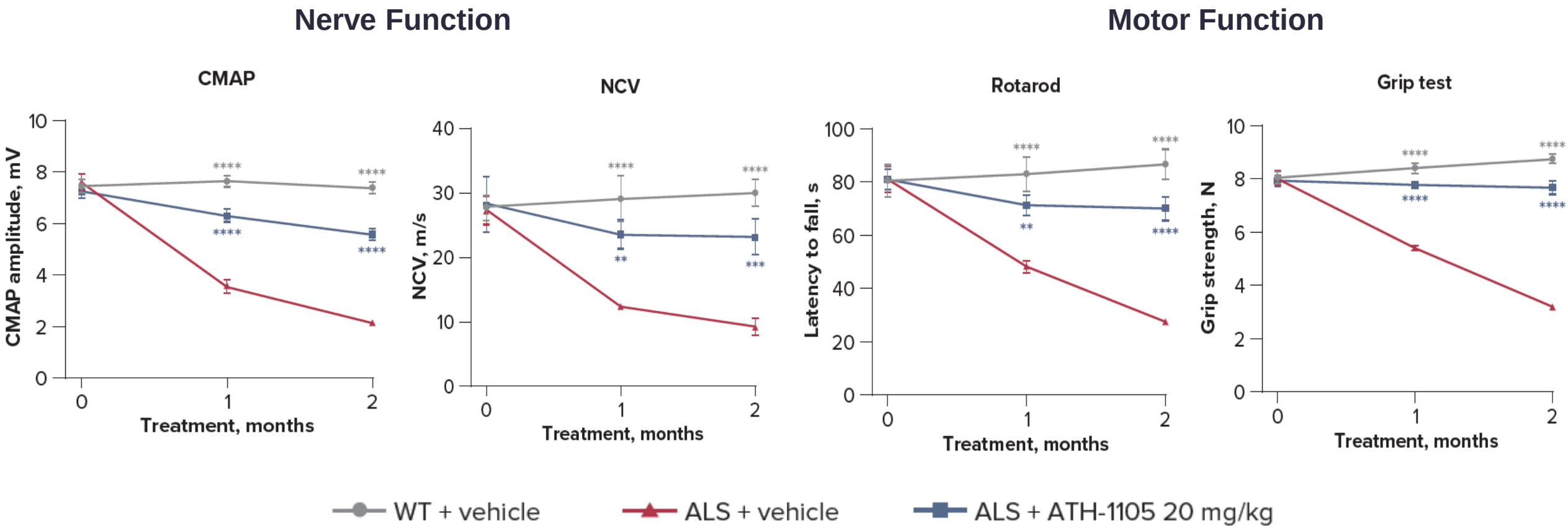
Graphical representation of the number of axons (per 100 µm2), axonal diameter (in micrometers), and mean of myelin g-ratio, defined as the ratio of the inner axonal diameter to the total axonal diameter, following 2 months of treatment. Data presented as mean + SEM. Statistical significance was determined 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

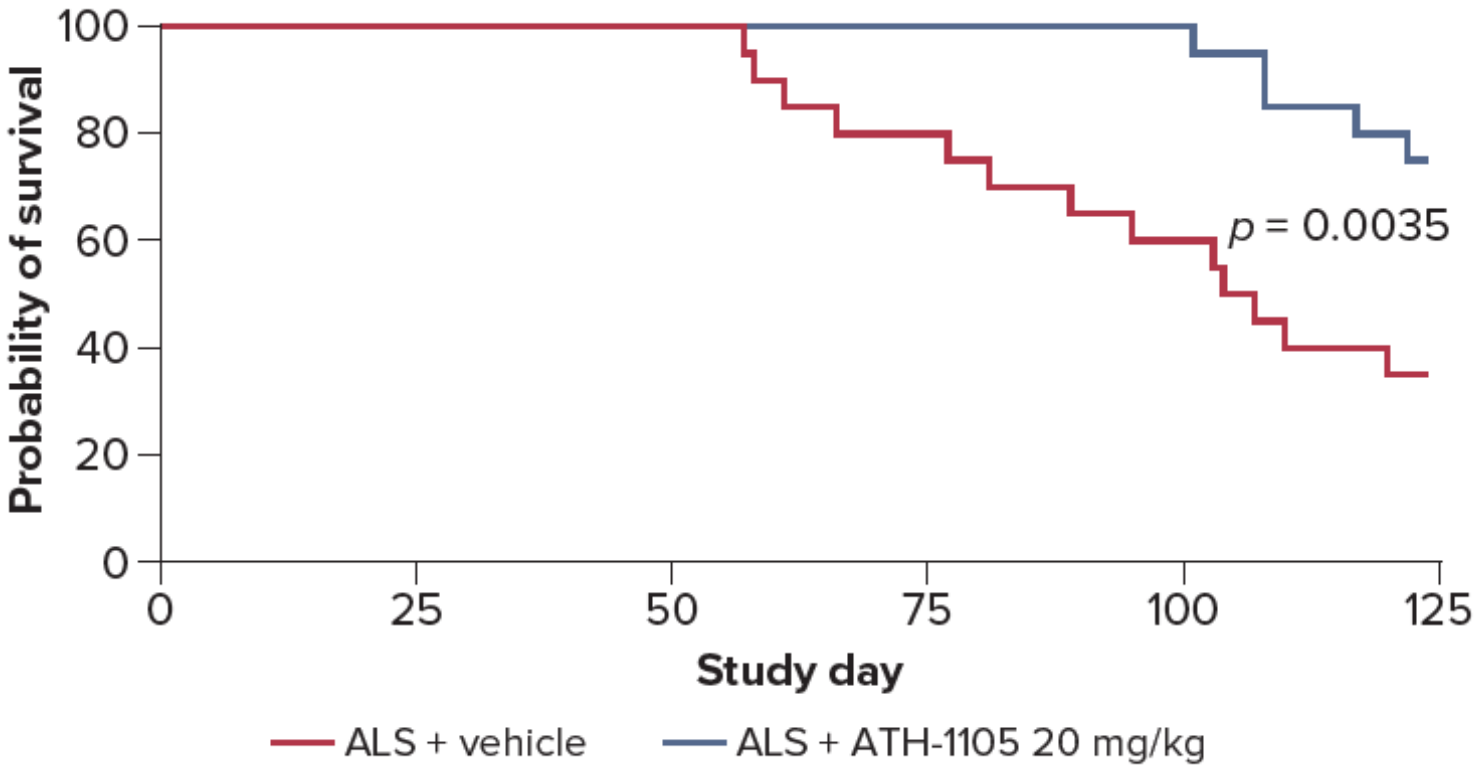
TDP-43 MOUSE MODEL OF ALS

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

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

Athira PHARMA

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

Corporate

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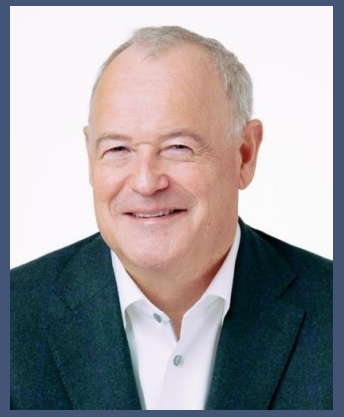
Athira management team with significant CNS product development and approval experience

EXECUTIVE LEADERSHIP



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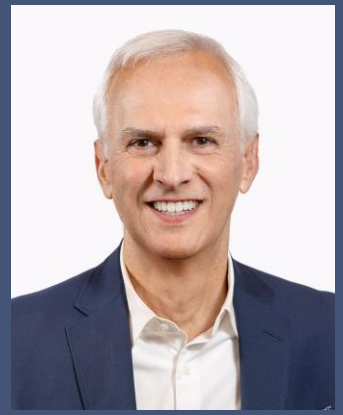
COO



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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 of 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

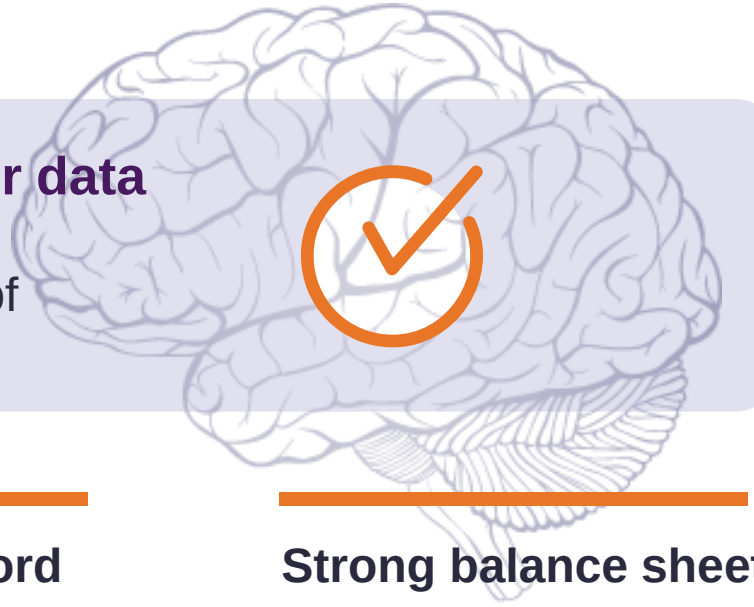
→
- Complete IND enabling studies
- File IND
- Initiate first-in-human Phase 1 testing of ATH-1105 in 1H2024

Athira PHARMA

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 (~$147M in cash*)

* Approximation of unaudited cash, cash equivalents and investments at 12/31/2023. The preliminary expectations regarding year-end cash, cash equivalents, and investments remain subject to management's review as well as to audit by our independent registered public accounting firm, and therefore actual results could differ from management's expectations.

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