Athira PHARMA

September 3, 2024

LIFT-AD Topline Readout

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, amyotrophic lateral sclerosis, and other neurodegenerative diseases, the anticipated reporting of data, and the potential learnings from preclinical studies and other nonclinical data, the LIFT-AD trial and their ability to inform and improve future clinical development plans.

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.

In addition, statements that "we believe" and similar statements reflect our beliefs and opinions on the relevant subject. These statements are based upon information available to us as of the date of this presentation, and although we believe such information forms a reasonable basis for such statements, such information may be limited or incomplete, and our statements should not be read to indicate that we have conducted a thorough inquiry into, or review of, all potentially available relevant information. These statements are inherently uncertain and readers are cautioned not to unduly rely upon these statements. Furthermore, if our forward-looking statements prove to be inaccurate, the inaccuracy may be material. In light of the significant uncertainties in these forward-looking statements, you should not regard these statements as a representation or warranty by us or any other person that we will achieve our objectives and plans in any specified time frame, or at all. We undertake no obligation to publicly update any forward-looking statements, whether as a result of new information, future events or otherwise, except as required by law.

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.

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.

We announce material information to the public through a variety of means, including filings with the SEC, press releases, public conference calls, our website (www.athira.com), our investor relations website (investors.athira.com), and our news site (investors.athira.com/news-and-events/press-releases). We use these channels, as well as social media, including our LinkedIn (https://www.linkedin.com/company/athirapharma), Facebook (https://www.facebook.com/athirapharmainc) and @athirapharma on X (formerly known as Twitter) and Instagram to communicate with investors and the public about Athira, our products, and other matters. Therefore, we encourage investors, the media, and others interested in Athira to review the information we make public in these locations, as such information could be deemed to be material information.

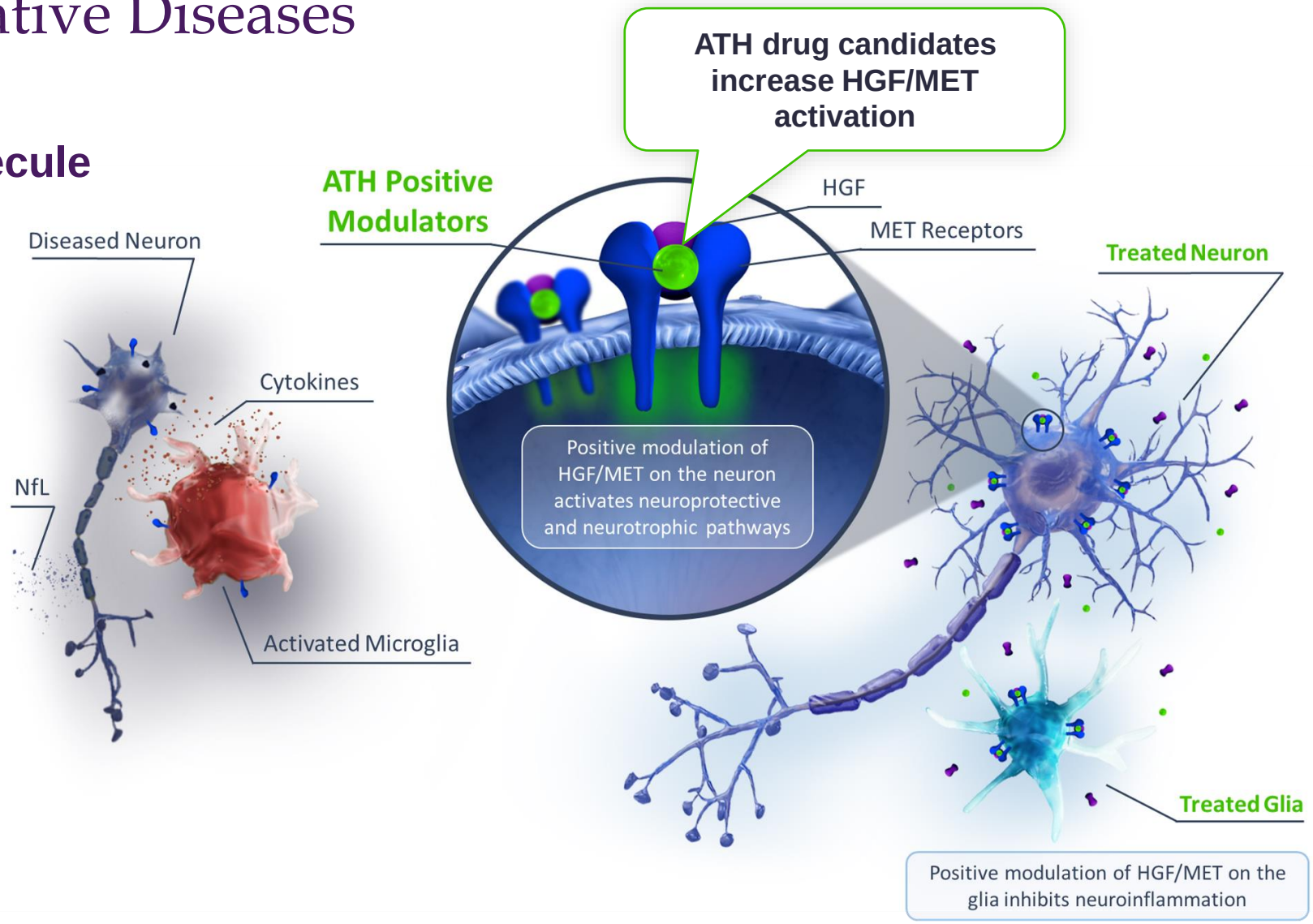
Therapeutic Potential of HGF Positive Modulation as a Neurotrophic Factor for Neurodegenerative Diseases

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.

LIFT-AD Study Design in Mild-to-Moderate Alzheimer's Disease

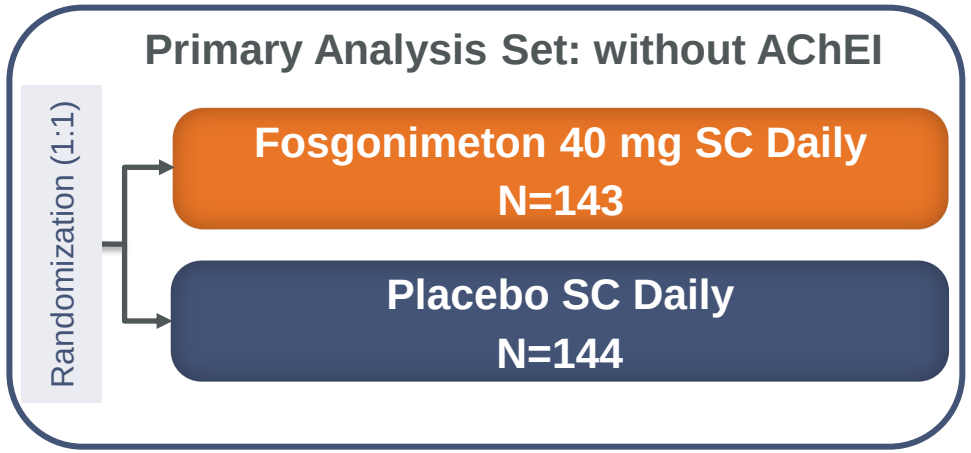
Randomized, Double-blind, Placebo-controlled, 26-week Trial

POPULATION

- 55-88 years of age
- Clinical diagnosis of probable AD
- Mild-to-moderate dementia
  - MMSE score of 14-24
  - CDR global score of 1 or 2

ANALYSIS SET

- **Primary analysis**: 312 enrolled
  287 evaluable participants without concomitant AChEI
- **Safety analysis**: 549 participants



ENDPOINTS

PRIMARY

- Global Statistical Test - composite of ADAS-Cog11 and ADCS-ADL23
- Safety

SECONDARY

- ADAS-Cog11
- ADCS-ADL23
- Plasma NfL

EXPLORATORY PLASMA BIOMARKERS

- Aβ42/40, p-Tau181, p-Tau217, and GFAP

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; CDR, clinical dementia rating; GFAP, glial fibrillary acidic protein; MMSE, Mini Mental State Examination; NfL, neurofilament light chain; p-Tau, phosphorylated tau; Aβ, Amyloid-ß; SC, subcutaneous

Baseline Characteristics and Demographics are Well Balanced

Characteristic	Primary Analysis Population (No Concomitant AChEI)	
	Placebo (N=144)	**Fosgonimeton 40 mg** (N=143)
Mean (SD) age, years	73.4 (7.1)	72.6 (6.9)
Female, n (%)	82 (56.9)	76 (53.1)
White, n (%)	118 (81.9)	116 (81.1)
APOE4 carriers, n (%)	74 (51.4)	74 (51.7)
Heterozygotes	59 (41.0)	59 (41.3)
Homozygotes	15 (10.4)	15 (10.5)
Concomitant AChEI, n (%)	0 (0)	0 (0)
Mean (SD) MMSE Score	19.3 (3.4)	19.9 (3.5)
MMSE ≥20 (mild), n (%)	74 (51.4)	81 (56.6)
MMSE <20 (moderate), n (%)	70 (48.6)	61 (42.7)
CDR Score, n (%)		
0.5	1 (0.7)	1 (0.7)
1	123 (85.4)	122 (85.3)
2	19 (13.2)	20 (14.0)
Mean (SD) ADAS-Cog 11	22.3 (7.6)	20.7 (7.8)
Mean (SD) ADCS-ADL23	62.3 (10.0)	62.5 (9.9)
Mean (SD) NfL, pg/mL	27.7 (16.4)	26.3 (25.5)

AChEI, acetylcholinesterase inhibitor; ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living; CDR, clinical dementia rating; MMSE, Mini Mental State Examination; NfL, neurofilament light chain; APOE, Apolipoprotein E; SD, standard deviation

Overview of Primary and Secondary Endpoints

Primary and secondary endpoints did not reach statistical significance

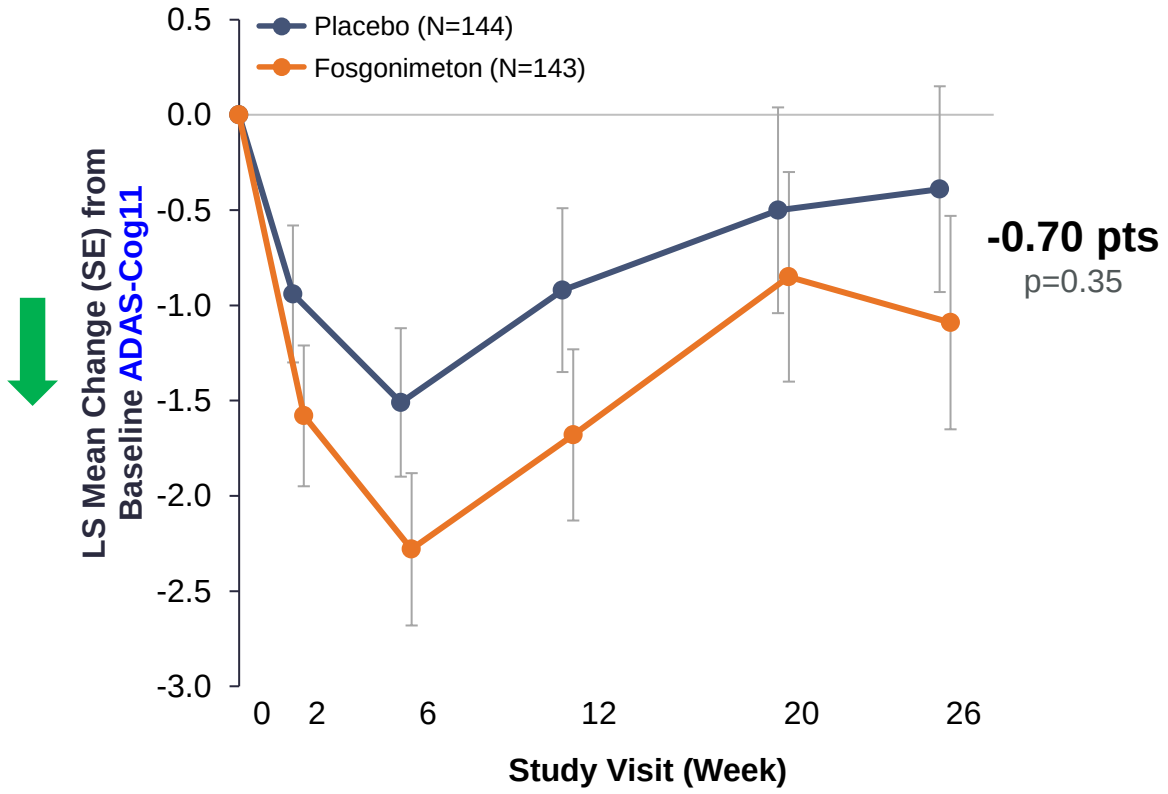
Measure (Direction of Improvement)	**LS Mean Change (SE) from Baseline at Week 26** (Primary Analysis Population)		
	Placebo (N=144)	**Fosgonimeton 40 mg** (N=143)	**Difference vs Placebo** (N=287)
GST ↓	-0.13 (0.07)	-0.21 (0.07)	-0.08 (0.10) p=0.70
ADAS-Cog11 ↓	-0.39 (0.54)	-1.09 (0.56)	-0.70 (0.77) p=0.35
ADCS-ADL23 ↑	-0.02 (0.65)	0.65 (0.67)	0.67 (0.92) p=0.61
NfL (pg/mL) ↓	2.95 (2.49)	-0.96 (2.48)	-3.91 (3.46) (p=0.26)*

GST, global statistical test; ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living; NfL, neurofilament light chain; LS mean, least squares mean; SE, standard error

*Weighted model p-value not reported due to lack of model convergence; Non-weighted analysis p-value shown

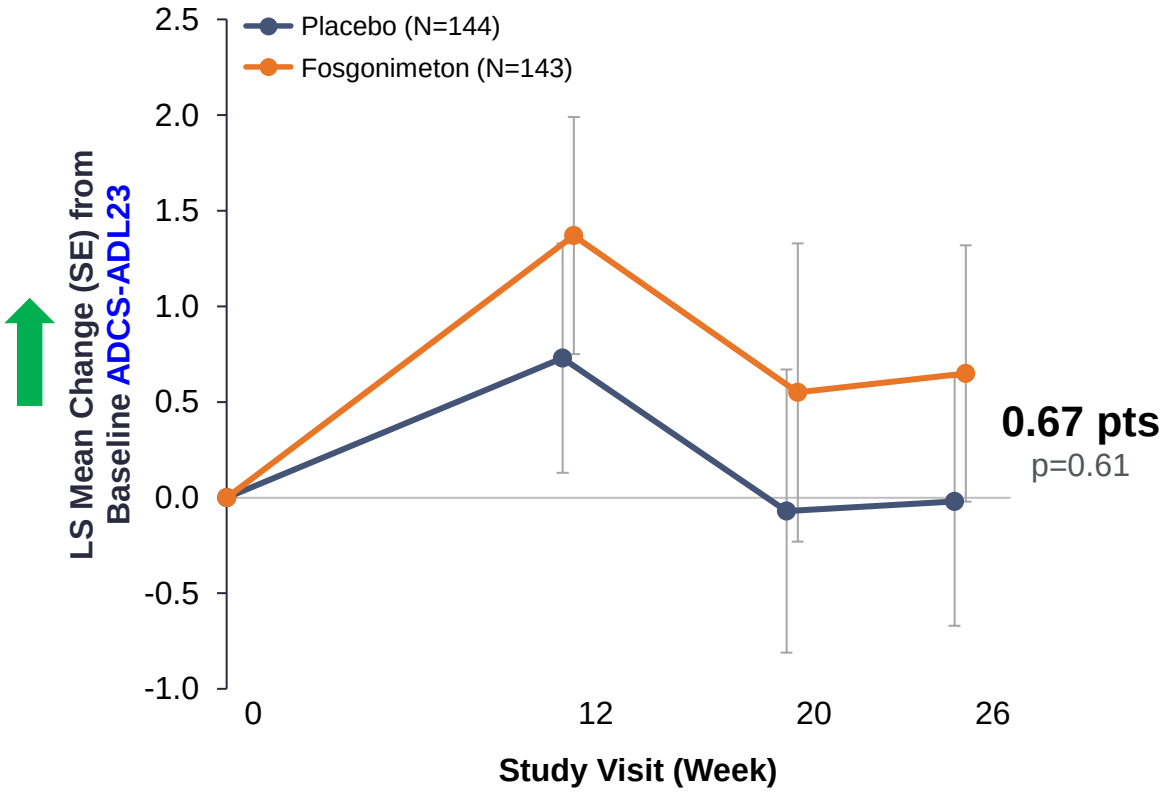
Change in Cognition and Activities of Daily Living

ADAS-Cog11



	Placebo (N=144)	Fosgonimeton (N=143)
Baseline, Mean (SD)	22.3 (7.6)	20.7 (7.8)
CFB at Week 26, Mean (SE)	-0.39 (0.54)	-1.09 (0.56)

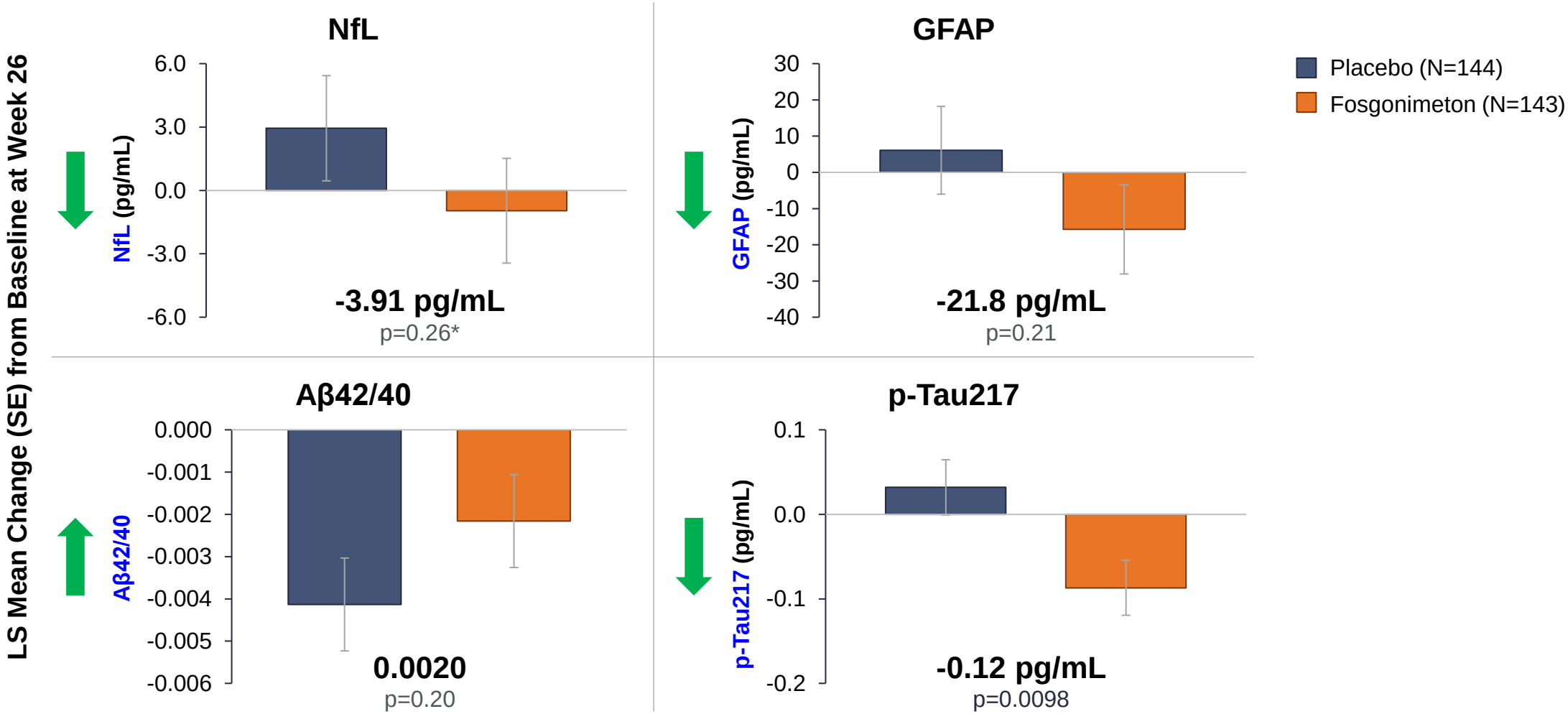
ADCS-ADL23



	Placebo (N=144)	Fosgonimeton (N=143)
Baseline, Mean (SD)	62.3 (10.0)	62.5 (9.9)
CFB at Week 26, Mean (SE)	-0.02 (0.65)	-0.65 (0.67)

ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; ADCS-ADL23, Alzheimer's Disease Cooperative Study–Activities of Daily Living; CFB, change from baseline; LS mean, least squares mean; SE, standard error; SD, standard deviation

Fosgonimeton Shows a Neuroprotective Effect Across Plasma Biomarkers of Neurodegeneration (NfL), Inflammation (GFAP), and Protein Pathology (Aβ42/40 and p-Tau 217)

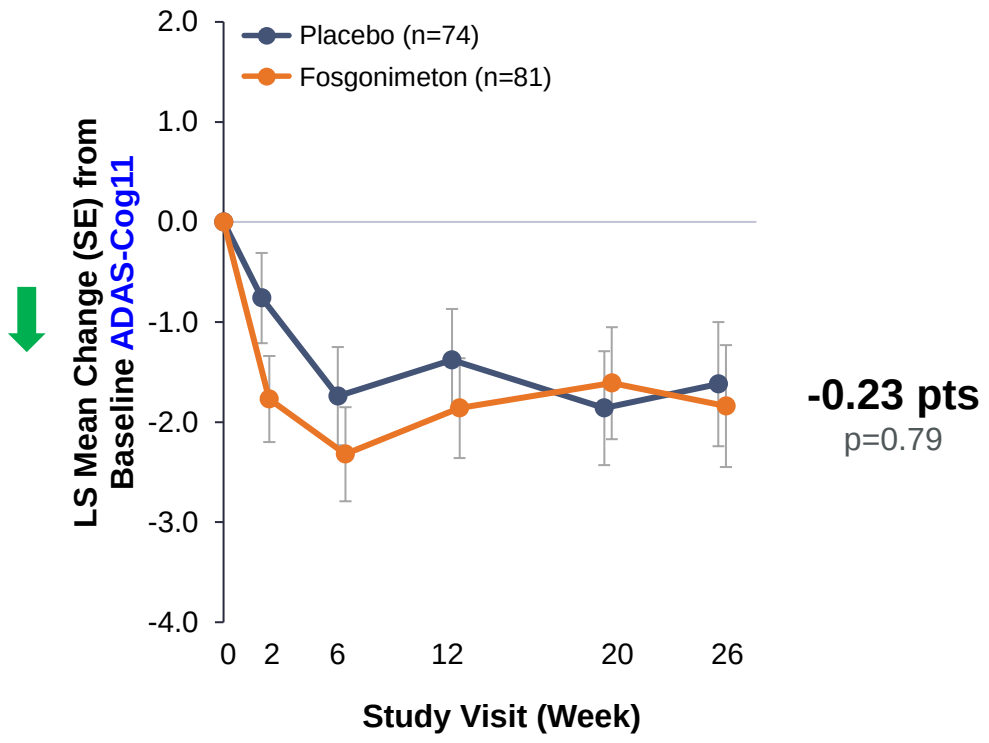


NfL, neurofilament light chain; GFAP, glial fibrillary acidic protein; Aβ, Amyloid-β; LS mean, least squares mean; SE, standard error

**Weighted model p-value not reported due to lack of model convergence; Non-weighted analysis p-value = 0.26*

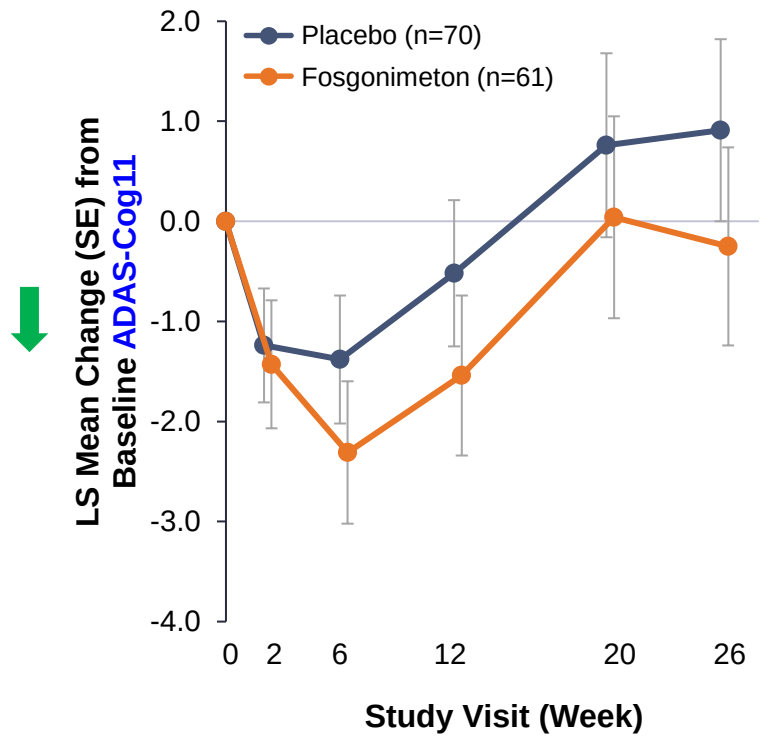
Change in Cognition in Mild and Moderate AD by MMSE

Mild Baseline MMSE (20 – 24)



-0.23 pts
p=0.79

Moderate Baseline MMSE (14 – 19)

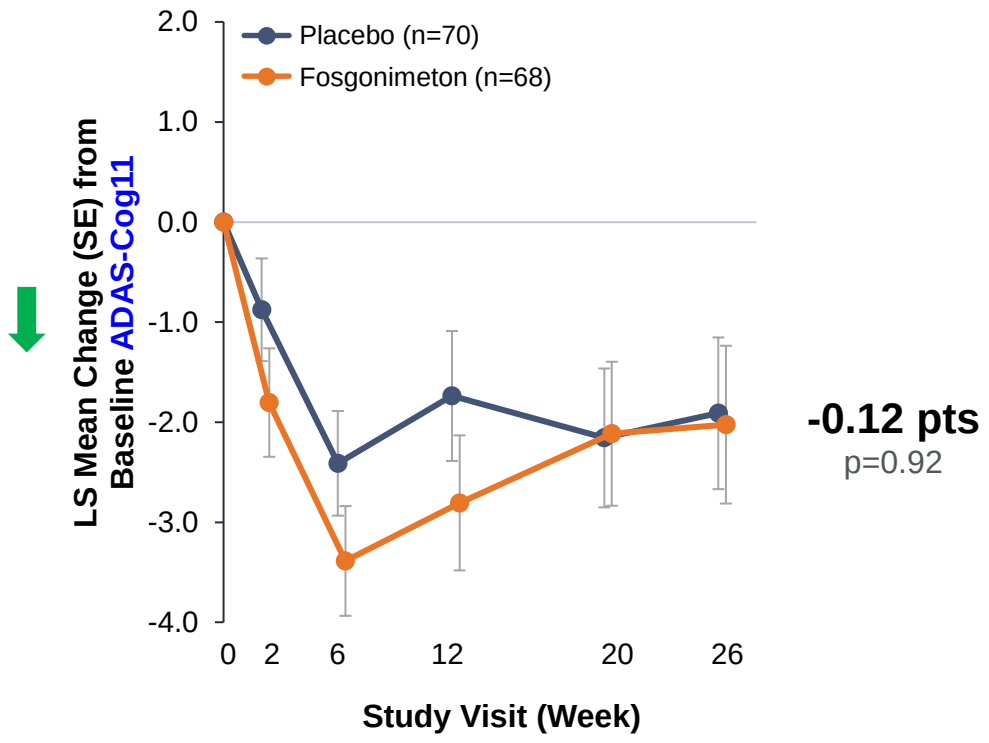


-1.16 pts
p=0.39

ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; MMSE, Mini Mental State Examination; LS mean, least squares mean; SE, standard error

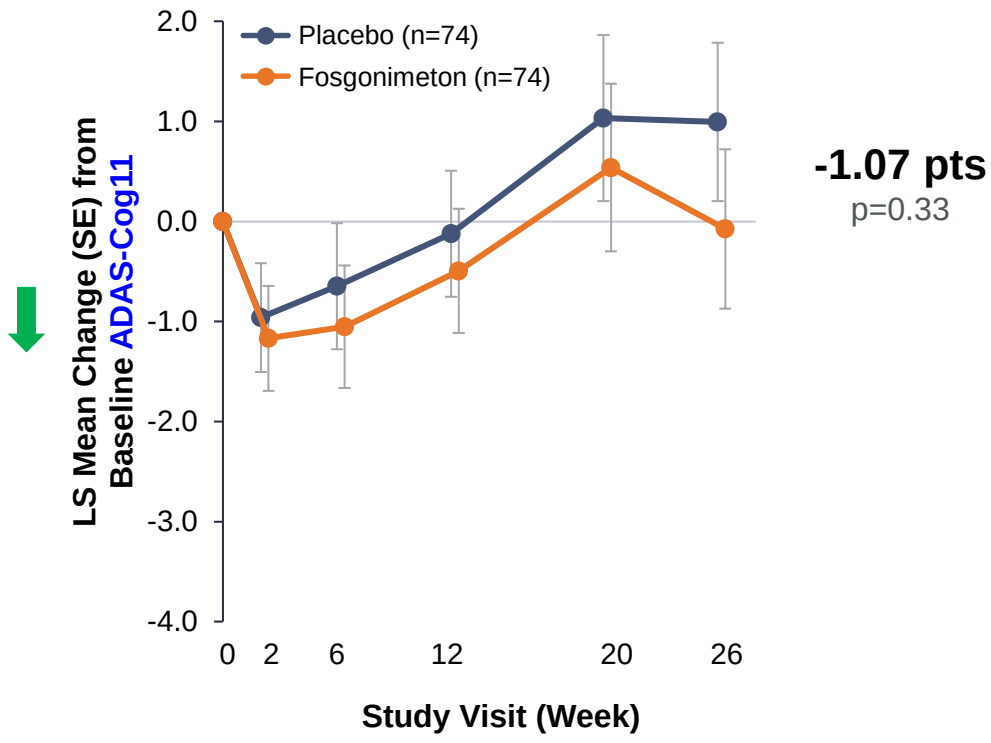
Change in Cognition Assessed by ADAS-Cog11 in APOE4 Carriers

APOE4 Non-Carrier



-0.12 pts
p=0.92

APOE4 Carrier

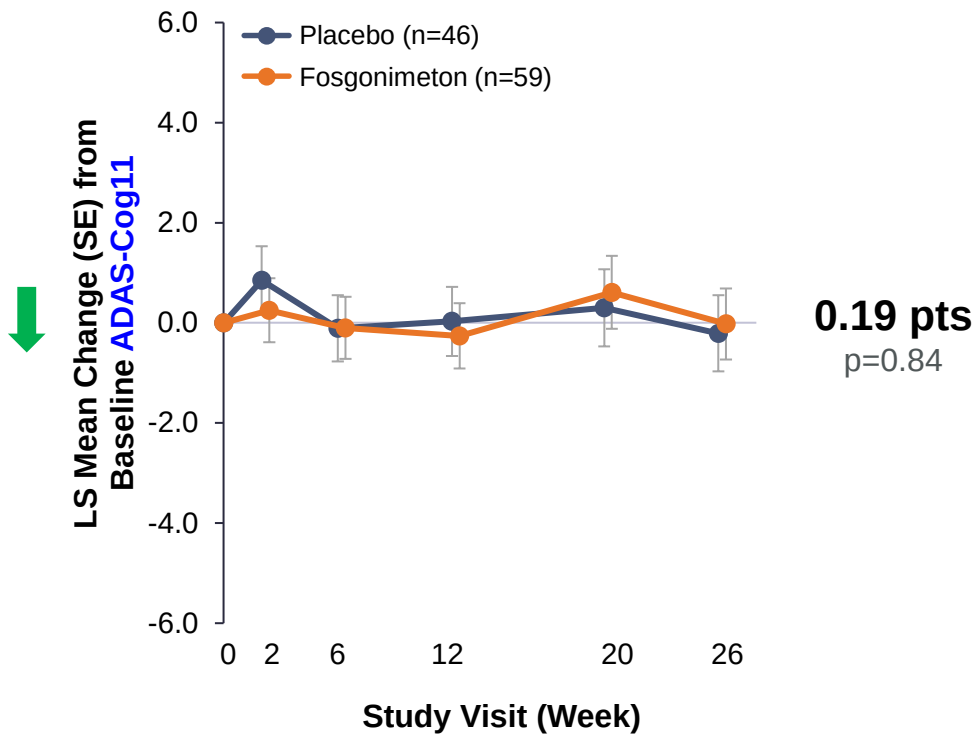


-1.07 pts
p=0.33

ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; MMSE, Mini Mental State Examination; APOE, Apolipoprotein E; LS mean, least squares mean; SE, standard error

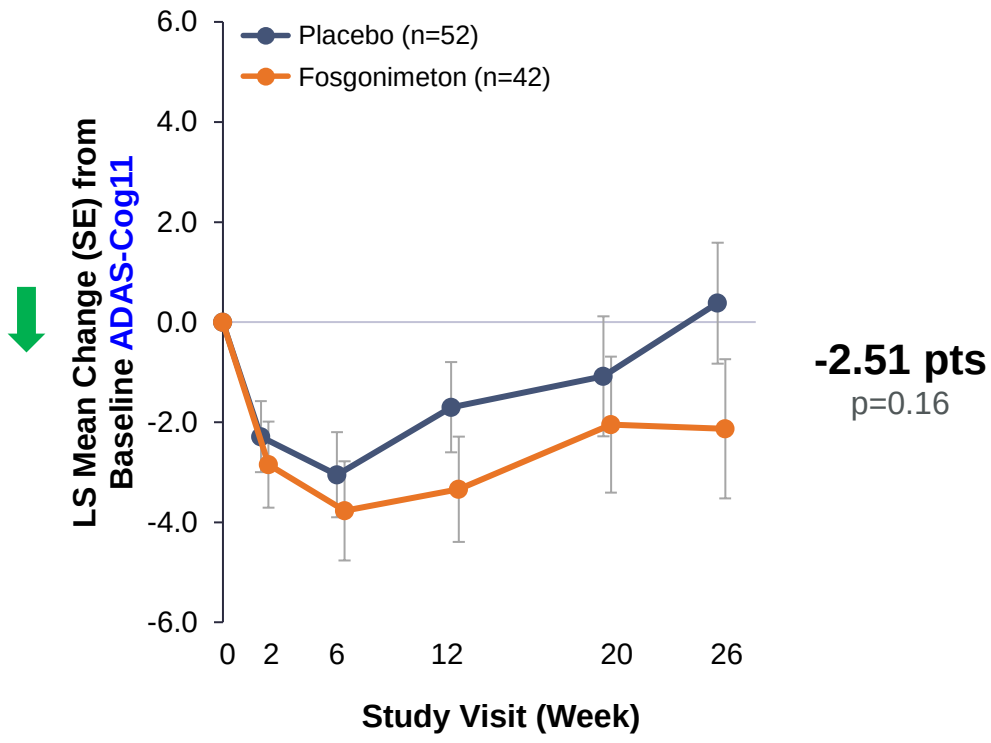
Change in Cognition in the Lowest and Highest ADAS-Cog11 Tertile Subsets at Baseline

Lowest Tertile



0.19 pts
p=0.84

Highest Tertile

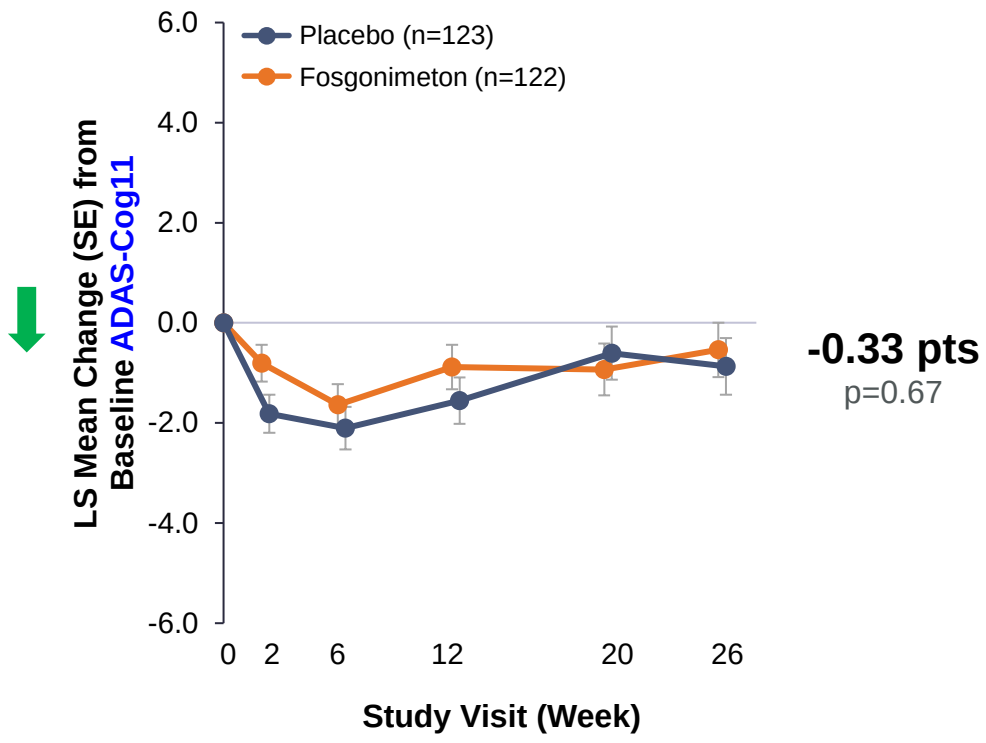


-2.51 pts
p=0.16

ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; MMSE, Mini Mental State Examination; LS mean, least squares mean; SE, standard error

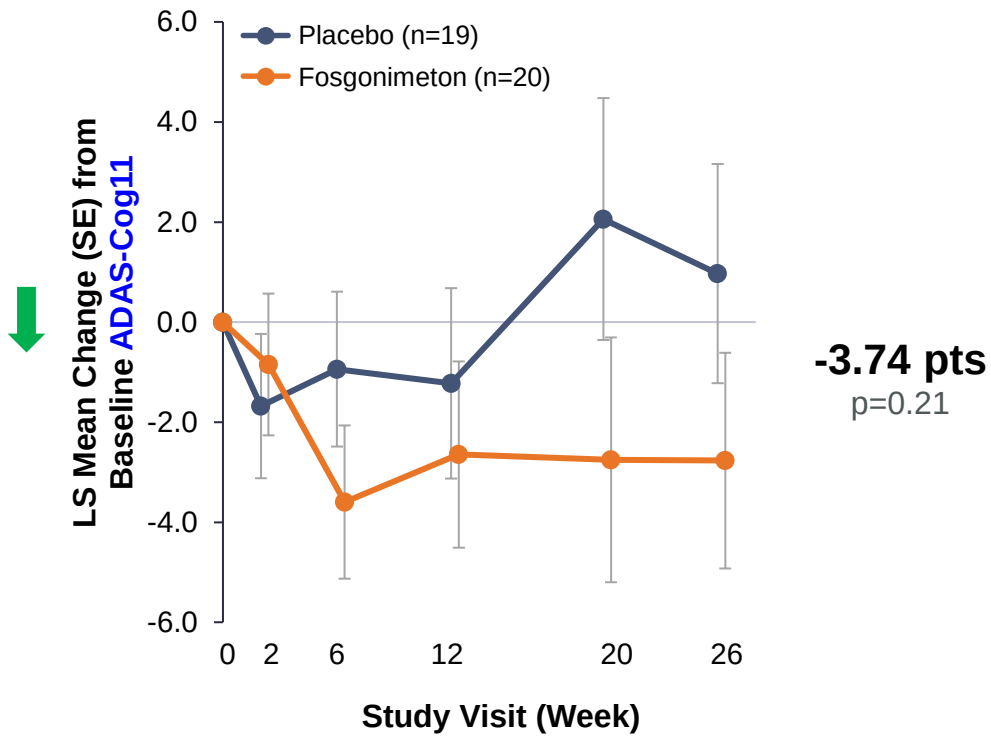
Change in Cognition in CDR Subgroups

CDR 1



-0.33 pts
p=0.67

CDR 2 (moderate AD)



-3.74 pts
p=0.21

ADAS-Cog11, Alzheimer's Disease Assessment Scale–Cognitive Subscale; MMSE, Mini Mental State Examination; CDR: Clinical Dementia Rating; LS mean, least squares mean; SE, standard error

Summary of Treatment-Emergent Adverse Events (Safety Analysis Population)

Fosgonimeton was generally well tolerated, with a favorable safety profile

Subject Incidence, n (%)	Placebo (N=218)	Fosgonimeton 40 mg (N=224)	Fosgonimeton 70 mg (N=107)
Any AE	136 (62.4)	177 (79.0)	94 (87.9)
TEAEs	132 (60.6)	175 (78.1)	94 (87.9)
Treatment-related TEAEs	54 (24.8)	155 (69.2)	86 (80.4)
Serious TEAEs	15 (6.9)	11 (4.9)	3 (2.8)
Treatment-related serious TEAEs	0	3 (1.3)	2 (1.9)
TEAEs leading to study drug withdrawal	9 (4.1)	24 (10.7)	23 (21.5)
TEAEs leading to study drug interruption	9 (4.1)	29 (12.9)	11 (10.3)
TEAEs leading to study withdrawal	10 (4.6)	24 (10.7)	23 (21.5)
Deaths	0	0	0

AE, adverse event; TEAE, treatment-emergent adverse event
Most TEAE discontinuations were due to injection site reactions

Summary and Interpretation

- LIFT-AD trial did not meet primary endpoint of GST and key secondary endpoints; fosgonimeton compares favorably to placebo numerically despite the very small decline in the placebo group
- In subgroups[1] of patients with moderate AD, or APOE4 carriers or those with greatest impairment in cognition by ADAS-Cog11 fosgonimeton showed a larger effect size
- Fosgonimeton treatment was associated with changes in biomarkers of Alzheimer's disease pathology consistent with the broad neuroprotective mechanism of HGF modulation
- Fosgonimeton was generally well tolerated, with a favorable safety profile

Totality of the data suggests that positive modulation of HGF signaling may have potential beneficial effects in neurodegenerative diseases

1. Prespecified Analysis – MMSE and APOE4; Post Hoc – ADAS-Cog11 Tertiles and CDR

Thank You

Athira PHARMA

