

Corporate Presentation

August 2026



Forward looking statements

This presentation may contain some statements that may be considered “Forward-Looking Statements”, within the meaning of the US Securities Laws. Thus, any forward-looking statement relating to financial projections or other statements relating to the Company’s plans, objectives, expectations or intentions involve risks and uncertainties that may cause actual results to differ materially. For a discussion of such risks and uncertainties as they relate to us, please refer to our 2025 Form 20-F, filed with US Securities and Exchange Commission, in particular Item 3, Section D, titled “Risk Factors.”



Alterity is a late clinical stage biopharmaceutical company dedicated to developing treatments for neurodegenerative diseases

 Alterity means the state of being different

 Our goal is to slow the course of disease progression

 We strive to create an alternate future and improve patient quality of life

Redefining Neurodegenerative Disease Therapy

A Potential First-in-Class, Disease-Modifying Therapy for Multiple System Atrophy (MSA)

Compelling Phase 2 Efficacy on FDA-Endorsed Endpoint

Up to 46% slowing of disease progression v. placebo ($p < 0.05$) on FDA-endorsed endpoint*
Favorable safety profile with no drug-related serious adverse events

Unmet Need with Significant Commercial Potential

MSA is a rare, rapidly progressive disease (up to 50,000 U.S. patients)
~\$US2.4B global peak sales opportunity in MSA with patent protection to 2045

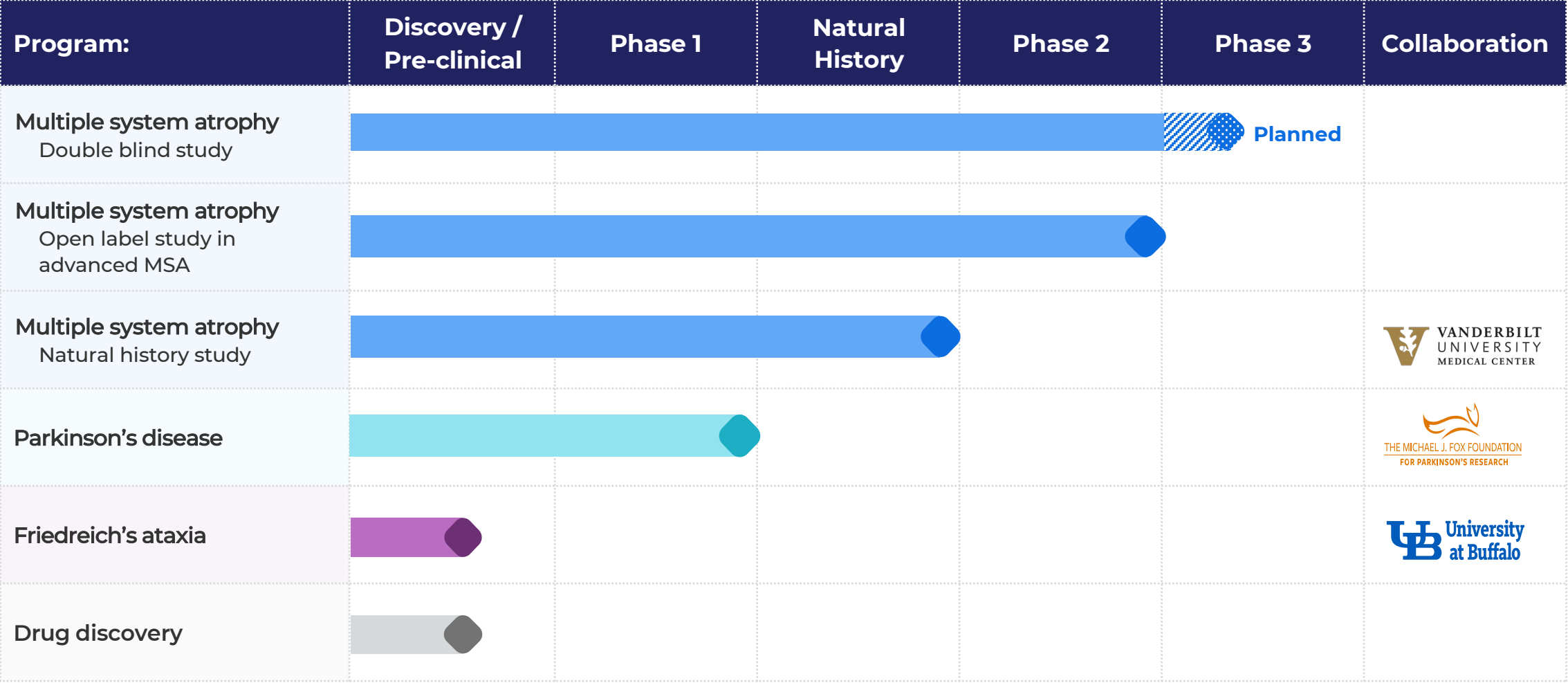
Differentiated, Disease- Modifying Approach for MSA

Oral iron chaperone ATH434 targets excess reactive iron and α -synucleinopathies
Blood brain barrier penetrant small molecule

Pivotal Advancement in 2026 with Clear Regulatory Path

Successful End-of-Phase 2 FDA meeting obtained alignment on Phase 3 design
Veteran development team with 3 FDA approvals in Neurology area

Broad opportunity for ATH434 in neurodegenerative disease



Multiple System Atrophy (MSA): Parkinsonian disorder with no approved treatment

Rapidly progressive

Highly debilitating

Up to 50,000

patients in U.S.

Disease characteristics:

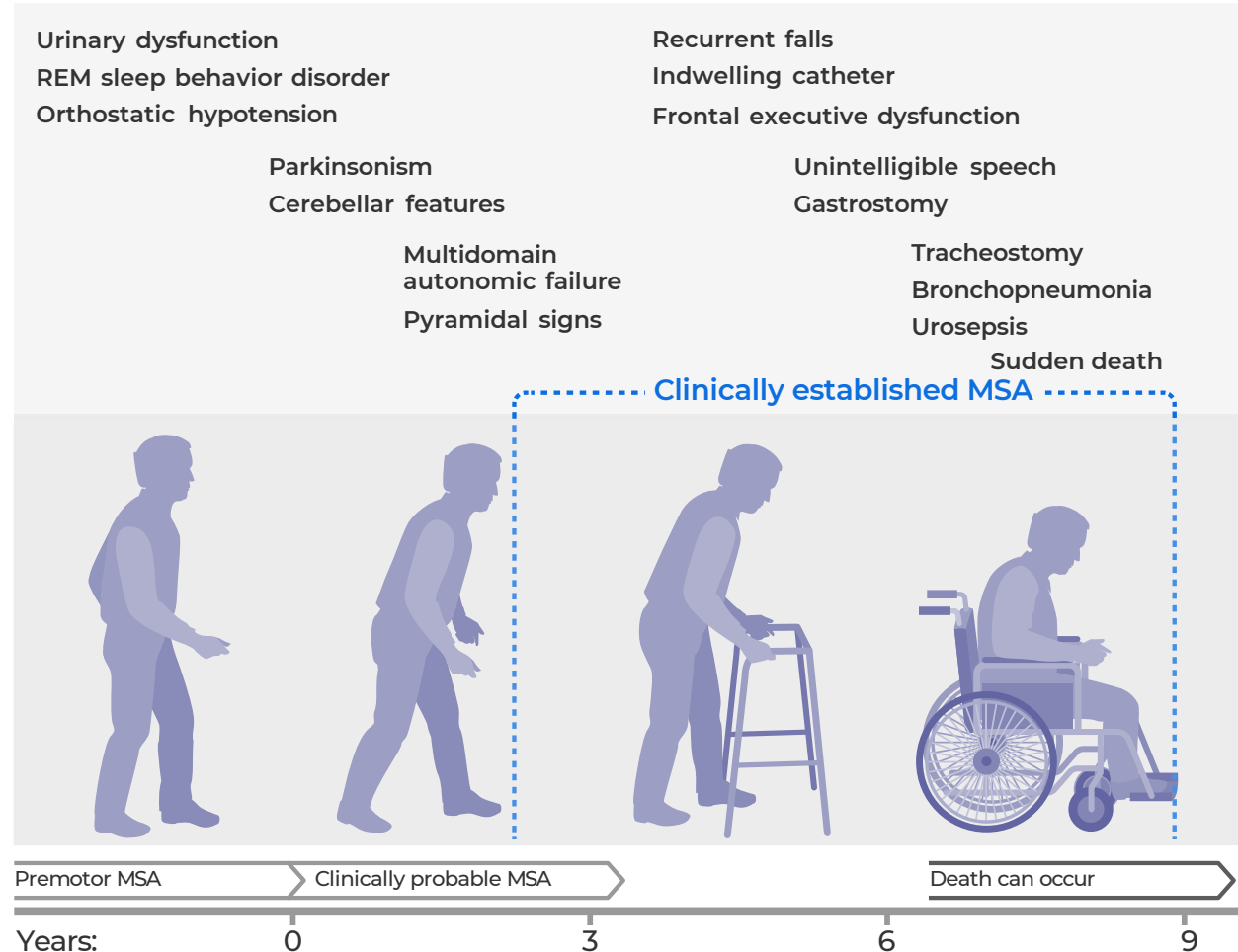
- Motor: Parkinsonism, uncoordinated movements, balance problems, falls
- Autonomic dysfunction: blood pressure maintenance, bladder control, bowel function
- Atrophy and α -synuclein accumulation in multiple brain regions

Over 50%

require wheelchair
in 5 years

7.5 years

median survival
after symptom onset

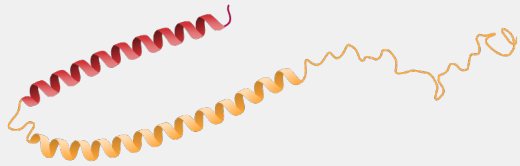


Targeting the pathology in Parkinsonian disorders



Targeting key players in MSA pathology

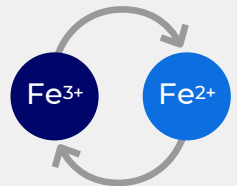
Alpha-synuclein and iron balance in health and disease



α -Synuclein protein

- Present in all neurons
- Enables neuronal communication

In disease: α -Synuclein aggregates in neurons in MSA impairing communication and leading to dysfunction



Fe^{2+} Reactive
 Fe^{3+} Stable

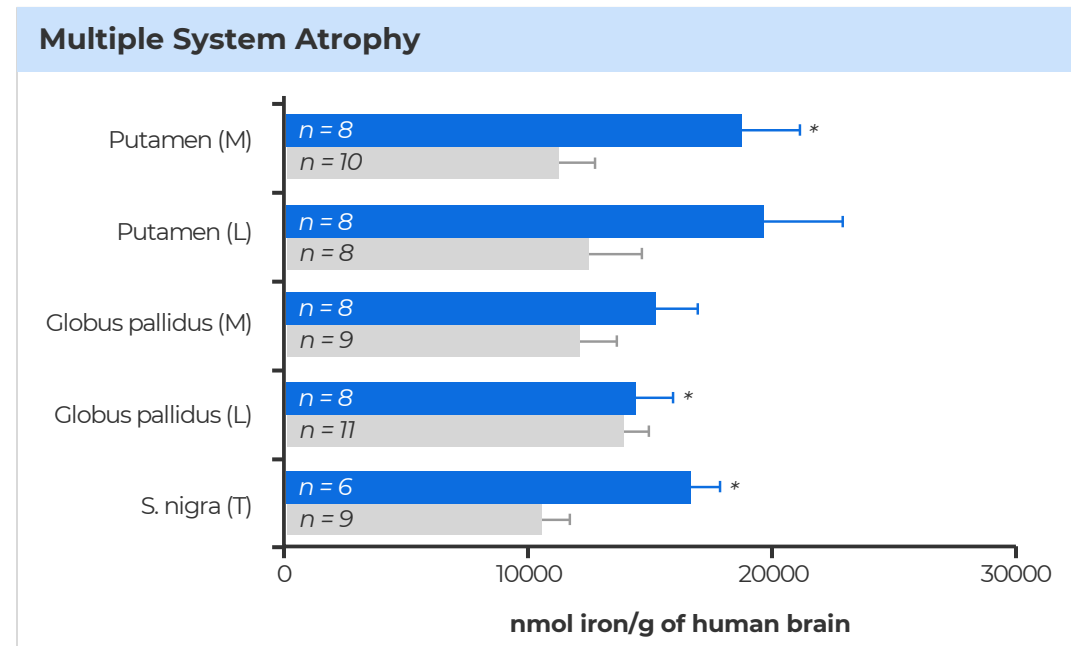
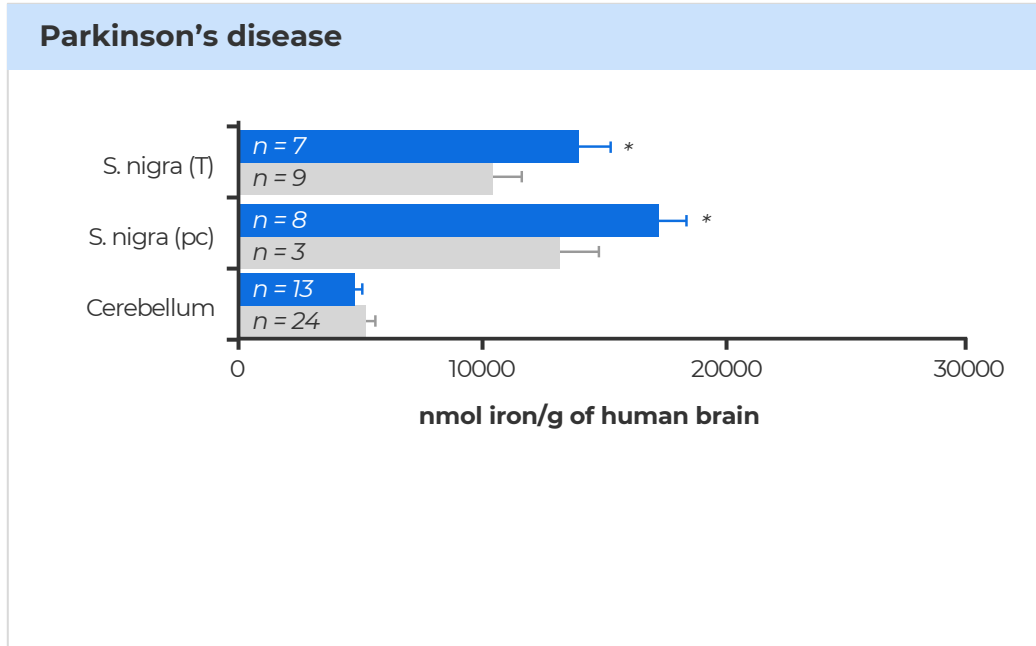
Two forms of iron required for cellular function

- Neurotransmitter synthesis (e.g., dopamine)
- Myelin synthesis (allows fast signal transmission)

In disease: Excess reactive iron drives α -synuclein aggregation and oxidative injury

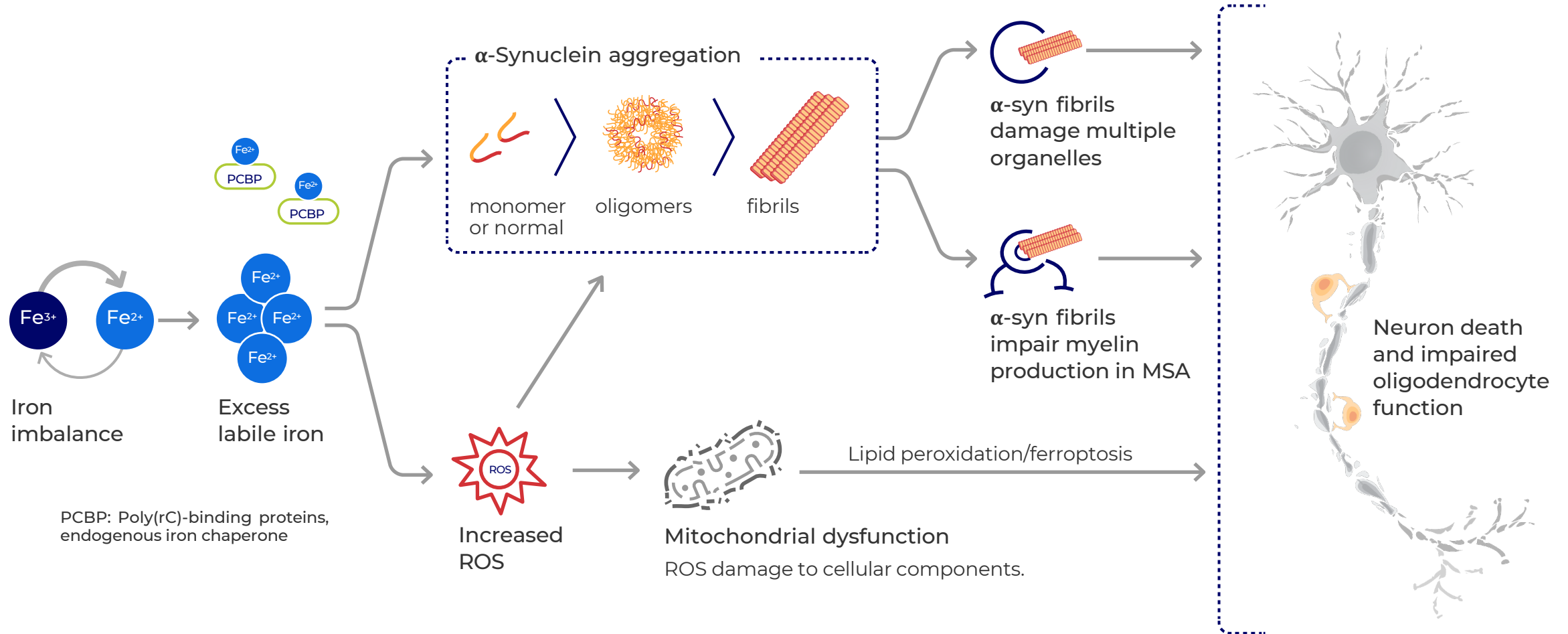
Pathology of Parkinsonian disorders

Increased Brain Iron in Areas of Pathology



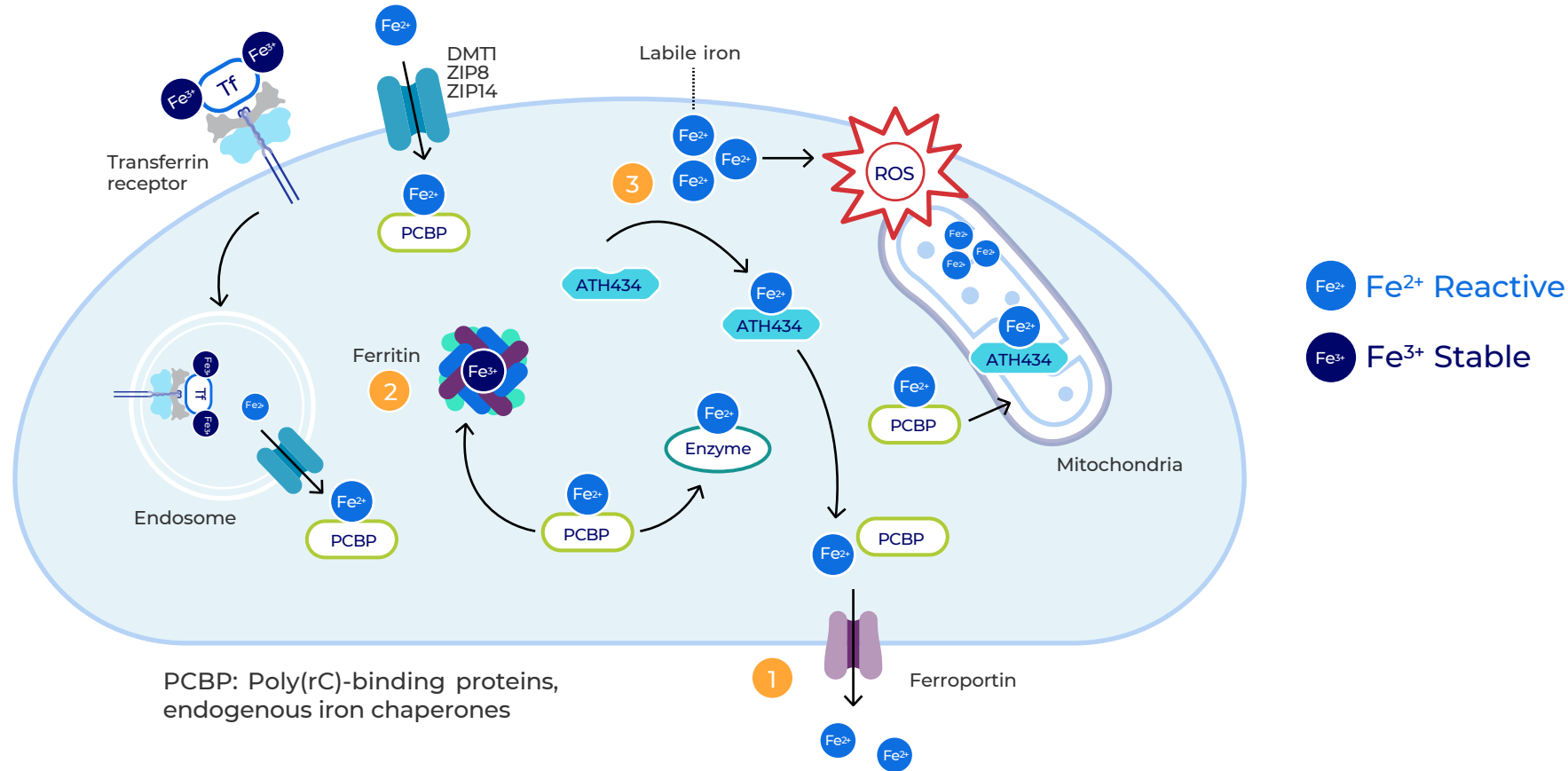
■ Patients ■ Healthy controls

Excess labile iron is a key driver of pathology causing α -synuclein aggregation and oxidative injury



ATH434 mechanism of action: Iron chaperone

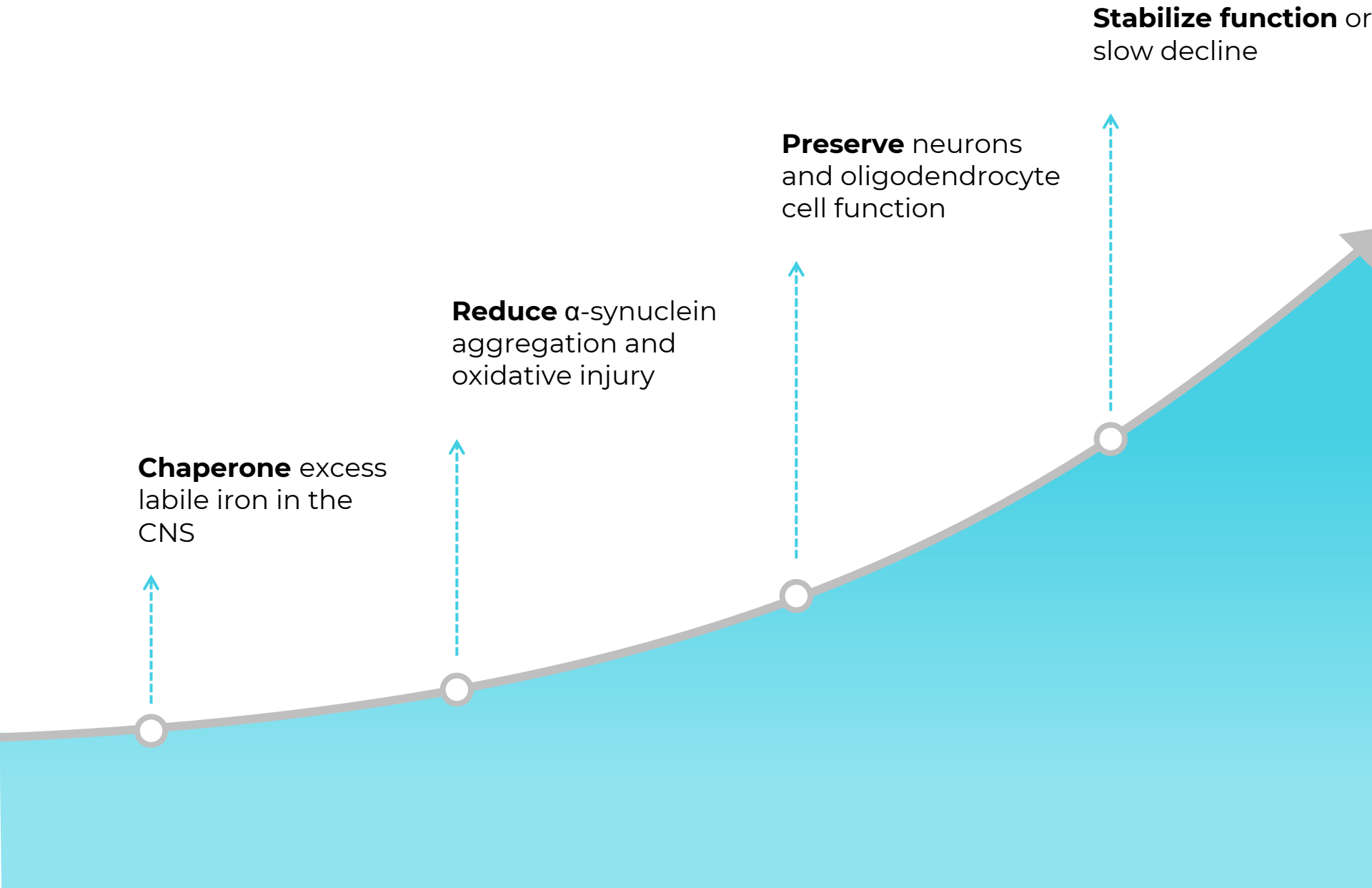
ATH434 chaperones excess labile (reactive) iron to reduce neuronal injury



Chaperone mechanisms:

- 1 Efflux iron from cell (ferroportin)
- 2 Increase iron storage (ferritin)
- 3 Buffering Fe^{2+} in labile iron pool

Treatment approach: Address underlying pathology

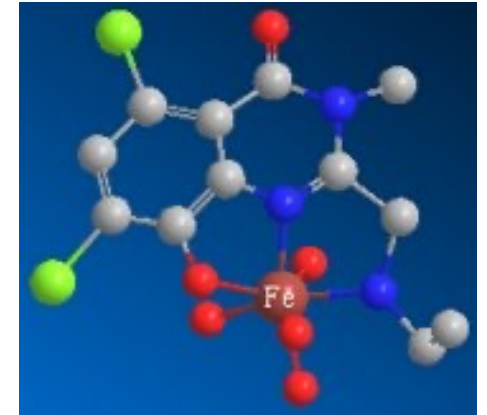


Based on mechanism of action, ATH434 is a potential disease modifying therapy

ATH434: Small molecule drug candidate

- ✓ Oral administration Preferred by patients and doctors vs infusions (IV, intrathecal) or injections
- ✓ Blood-brain barrier penetrant Acts intracellularly to address underlying pathology
- ✓ Iron chaperone Moderate binding affinity, redistributes excess labile iron in CNS
- ✓ Broad treatment potential Potential to treat many neurodegenerative diseases (e.g., Parkinson's, Friedreich Ataxia)
- ✓ Orphan & Fast Track designations US FDA Fast Track Designation and Orphan drug designation in U.S. and EU

ATH434 binding to labile iron



Multiple models of neurodegenerative disease demonstrate ATH434 efficacy

Target Disease	Model	Midbrain iron (incl. s. nigra)	α -Synuclein	Preserve neurons/ function	Clinical observations
MSA ¹	PLP- α -syn	↓	↓	↑	Improved motor performance
MSA ²	PLP- α -syn	↔ to ↓	↓	↑	Improved motor performance
Parkinson's	Monkey MPTP	↔ to ↓	n/a	↑	Improved motor performance
Parkinson's	Mouse MPTP	↓	↓	↑	Improved motor performance
Parkinson's	Mouse A53T	↓	↓	↑	Improved motor performance
Parkinson's	Mouse tau knockout	↓	↓	↑	Improved motor performance

↔ Stable

ATH434 consistently improved motor performance by reducing α -synuclein aggregation and preserving neurons



ATH434 clinical development program in MSA

Diligent approach to de-risk development program

Natural History Study

bioMUSE

- Observational study in *21 participants with clinically probable MSA*
- Designed to de-risk clinical development program
- Identify biomarkers to improve accuracy of patient selection

Phase 2

ATH434-201

Randomized double-blind placebo-controlled trial

Results: clinically meaningful efficacy on MSA rating scale, measures of orthostatic hypotension, disease severity

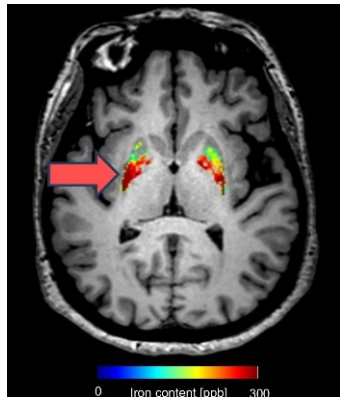
ATH434-202

Open label trial in advanced MSA patients

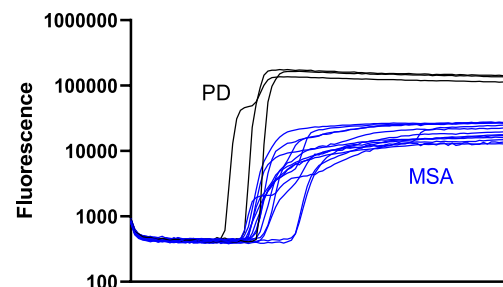
Results: showed improved neurological symptoms in more advanced patients and favorable safety

Optimized patient selection in Phase 2 trials

Advanced MRI methods



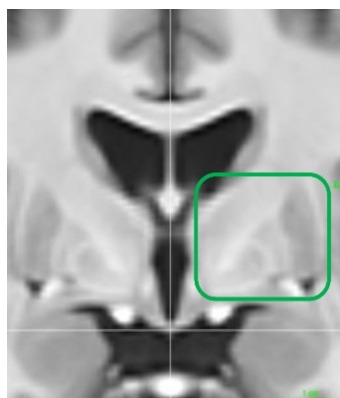
α -synuclein SAA in CSF



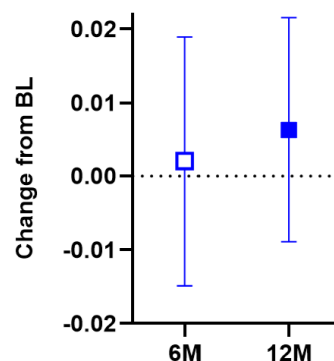
- ✓ Identified "iron signature" of early MSA
- ✓ Differentiated MSA from Parkinson's disease (PD)
- ✓ Revised selection criteria in ATH434-201 and ATH434-202 protocols to exclude PD patients

Precision biomarker assessment

Structural mapping



Iron content in pallidum



- ✓ Improved precision of volume measurements
- ✓ Novel strategies for measuring brain iron in individual regions
- ✓ State of the art methods enabled precise measurements of brain iron and volume with MRI

ATH434-201: Randomized, double-blind, placebo-controlled study

ATH434-201

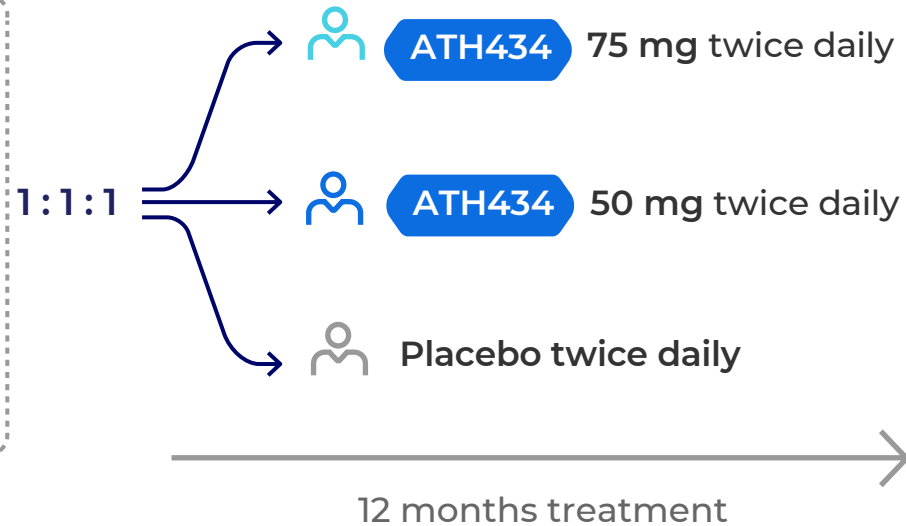
Patient criteria:



N = 77

- Clinical diagnosis of MSA
- Motor symptoms ≤4 years
- No severe impairment
- Elevated brain iron on MRI
- Elevated plasma NfL

Study design:



Endpoints:



Key clinical endpoint:
MSA Rating Scale



Additional secondary endpoints:
CGI-S, OHSA, Wearable Sensors, Safety



Key biomarker endpoint:
brain iron content by MRI

Importance of the Unified MSA Rating Scale Part I (UMSARS I)

ATH434-201

UMSARS Part I Items:

- | | |
|------------------------------------|--|
| ☐ Speech | ☐ Walking |
| ☐ Swallowing | ☐ Falling |
| ☐ Handwriting | ☐ Orthostatic symptoms |
| ☐ Cutting food | ☐ Urinary function |
| ☐ Dressing | ☐ Bowel function |
| ☐ Hygiene | ☐ Sexual function^ |

Rated from 0 to 48
higher scores worse

UMSARS is the FDA endorsed endpoint to support approval for the treatment of MSA

Primary endpoint in Phase 3

Validated rating scale to assess MSA disease severity
Rates functional impairment in domains affected in MSA

Baseline characteristics

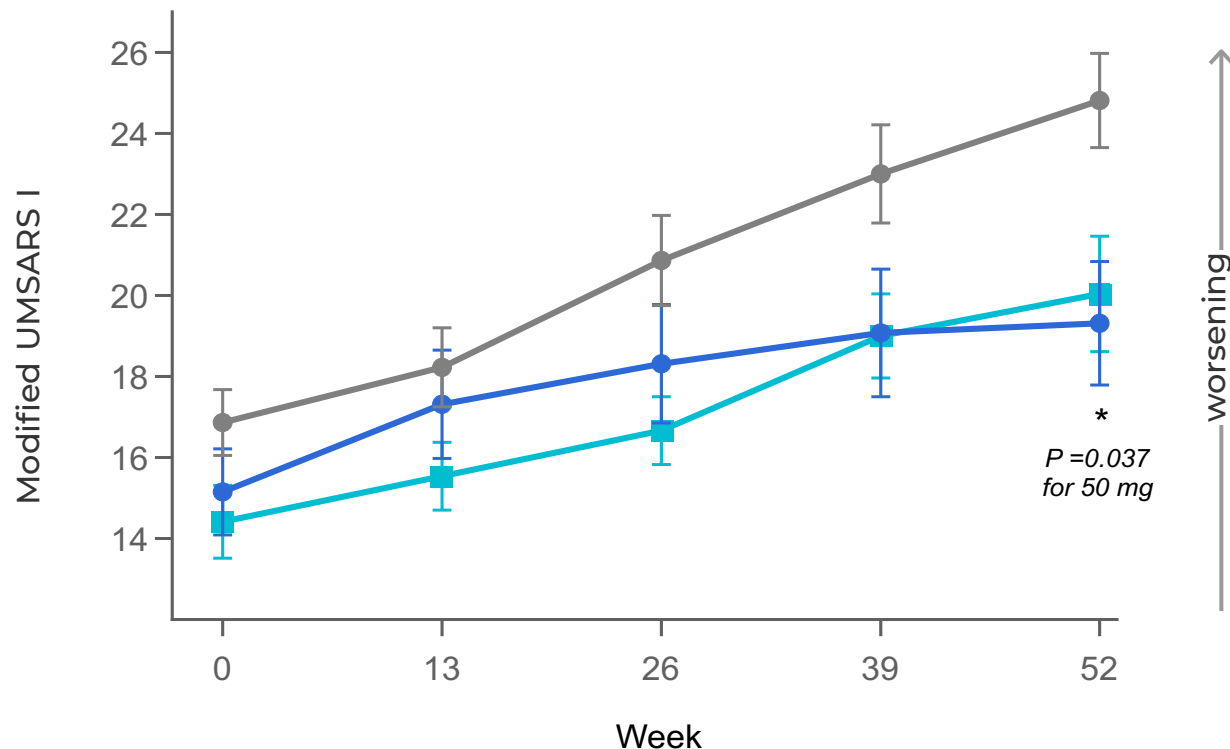
ATH434-201

	Placebo  N=22	ATH434-201 50 mg twice daily  N=25	ATH434-201 75 mg twice daily  N=24
Age (y)	61.3 (6.6)	63.1 (6.1)	63.9 (6.7)
Gender (% male)	63.6%	52.0%	62.5%
Duration of motor symptoms (y)	2.5 (0.8)	2.6 (0.8)	2.3 (0.9)
Modified UMSARS I	16.9 (3.9)	15.2 (5.4)	14.4 (4.4)
Motor score of Parkinson plus scale ¹	57.6 (14.2)	47.8 (18.4)	48.9 (16.8)
Plasma NfL (pg/mL)	34.9 (12.5)	31.1 (9.1)	32.3 (9.0)
CSF aggregating α -syn SAA (+)	91%	92%	96%
OH symptom assessment	13.5 (9.8)	13.8 (13.2)	15.0 (12.2)
Clinical phenotype: MSA-P (%)	59.1%	60.0%	70.8%
Severe orthostatic hypotension ²	4.5%	4.0%	29.2% 

Groups balanced at baseline except for severe orthostatic hypotension – a predictor of rapid disease progression

Clinically Significant Efficacy on 11-Item UMSARS I Change from Baseline to Week 52

ATH434-201



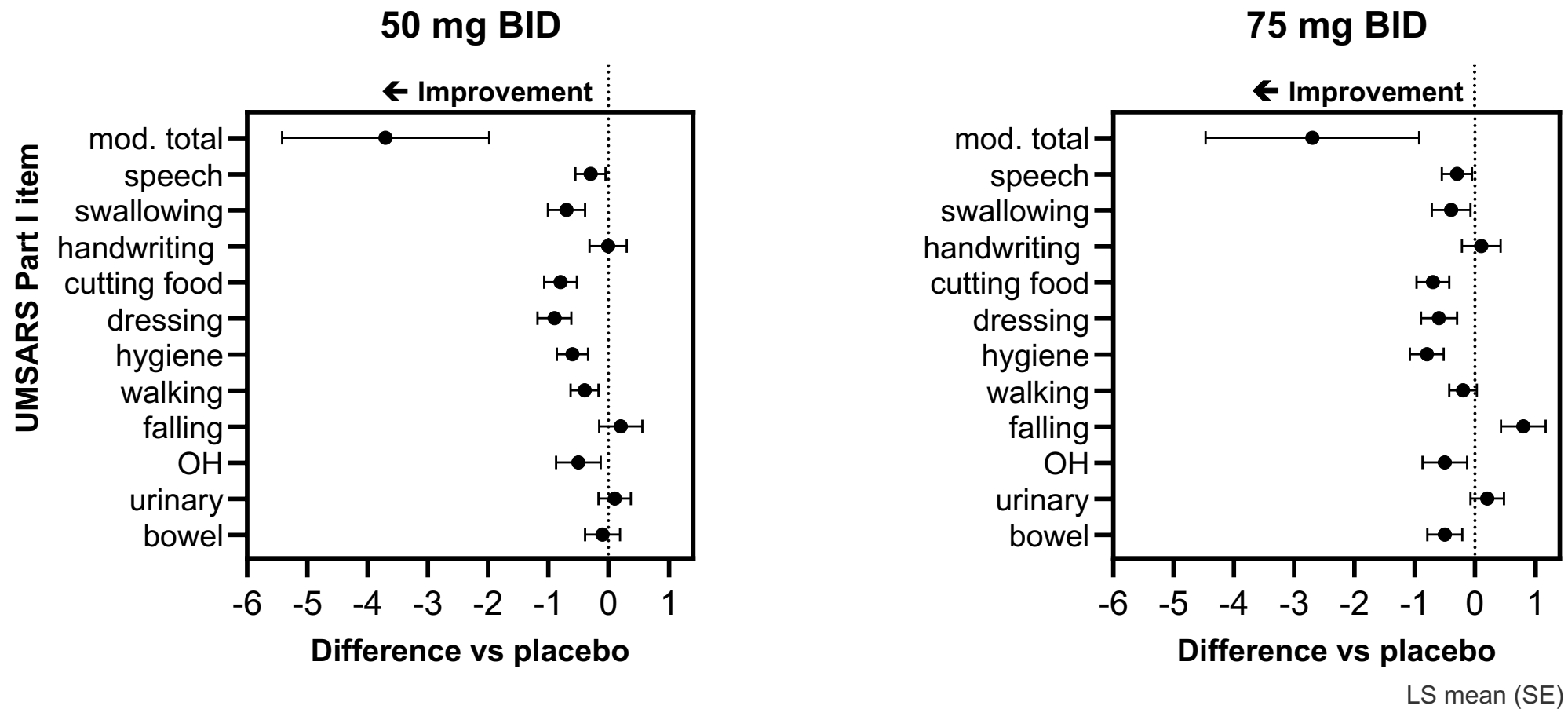
Placebo N=22	Difference v. placebo LS mean (SE)	Relative treatment effect
ATH434 50 mg N=25	-3.7 (1.7)	46%
ATH434 75 mg N=24	-2.7 (1.8)	34%

Minimal clinically important
difference (MCID) on UMSARS I¹ = -1.5 points

$$\text{Relative Treatment Effect:} \\ \frac{\text{Change}_{\text{ATH434}} - \text{Change}_{\text{Placebo}}}{\text{Change}_{\text{Placebo}}}$$

Item Analysis of 11-Item UMSARS I: Consistent efficacy across multiple domains

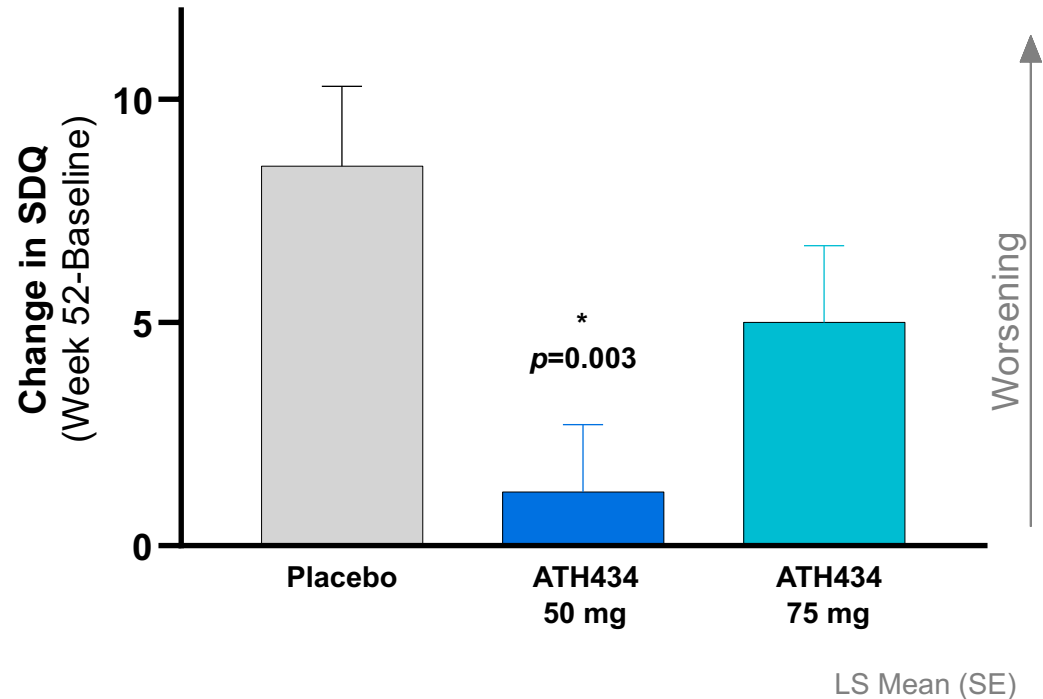
ATH434 treatment effect vs. placebo at 52 weeks



Similar pattern of efficacy in both dose groups

Swallowing Disturbance Questionnaire (SDQ) Change from baseline to week 52

ATH434-201



Patient reported outcome

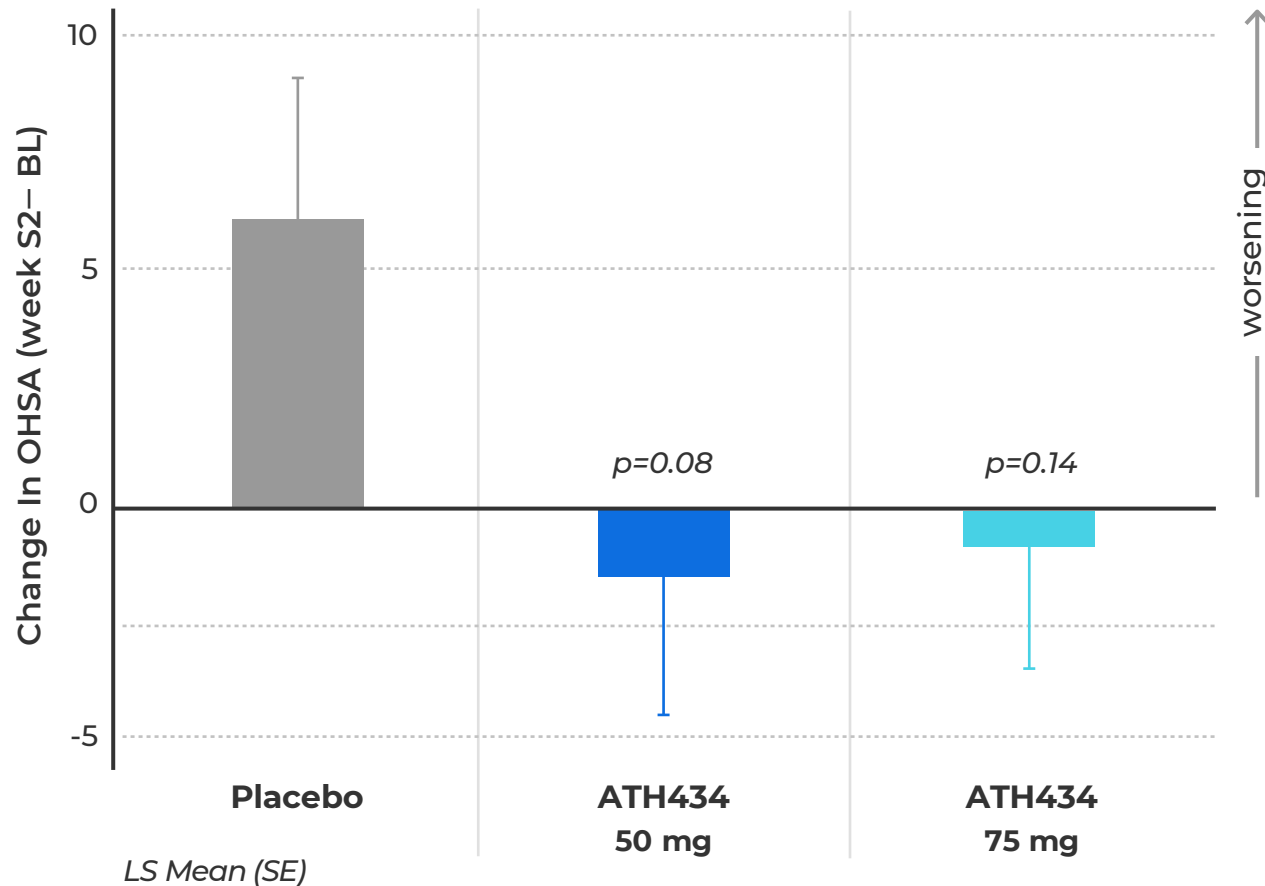
Validated Scale for Detecting Dysphagia

1. Do you experience difficulty chewing solid food like an apple, cookie or a cracker?
2. Are there any food residues in your mouth, cheeks, under your tongue or stuck to your palate after swallowing?
3. Does food or liquid come out of your nose when you eat or drink?
4. Does chewed up food dribble from your mouth?
5. Do you feel you have too much saliva in your mouth; do you drool or have difficulty swallowing your saliva?
6. Do you swallow chewed up food several times before it goes down your throat?
7. Do you experience difficulty in swallowing solid food (i.e., do apples or crackers get stuck in your throat)?
8. Do you experience difficulty in swallowing pureed food?
9. While eating, do you feel as if a lump of food is stuck in your throat?
10. Do you cough while swallowing liquids?
11. Do you cough while swallowing solid foods?
12. Immediately after eating or drinking, do you experience a change in your voice, such as hoarseness or reduced?
13. Other than during meals, do you experience coughing or difficulty breathing as a result of saliva entering your windpipe?
14. Do you experience difficulty in breathing during meals?
15. Have you suffered from a respiratory infection (pneumonia, bronchitis) during the past year?

Orthostatic Hypotension Symptom Assessment (OHSA)

Change from baseline to week 52

ATH434-201



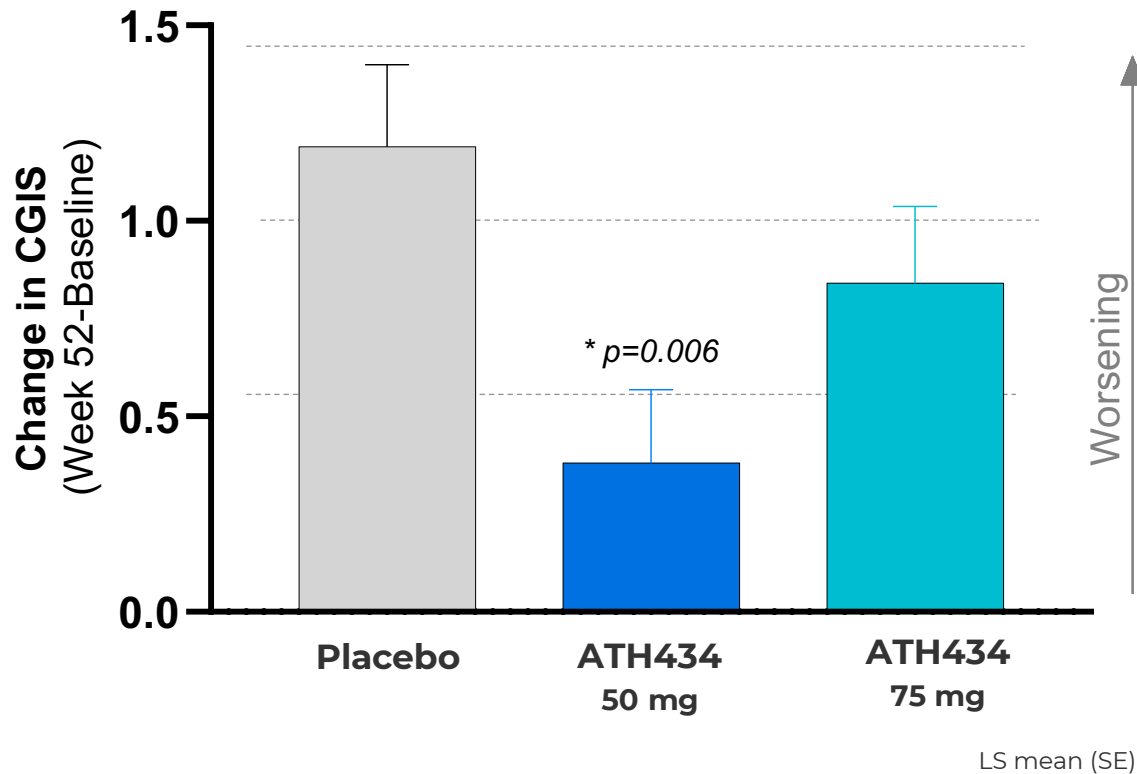
Patient reported outcome



- Assesses symptoms of low blood pressure when going from sitting to standing (e.g., dizziness / feeling faint / lightheadedness)

Efficacy on Clinical Global Impression of Severity (CGI-S) scale Change from baseline to week 52

ATH434-201

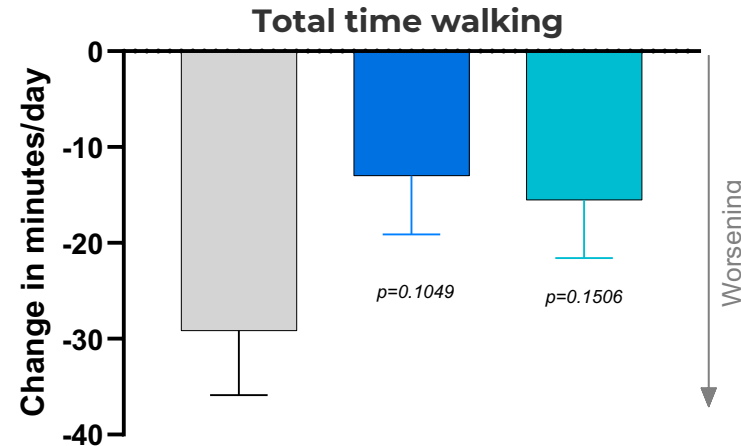
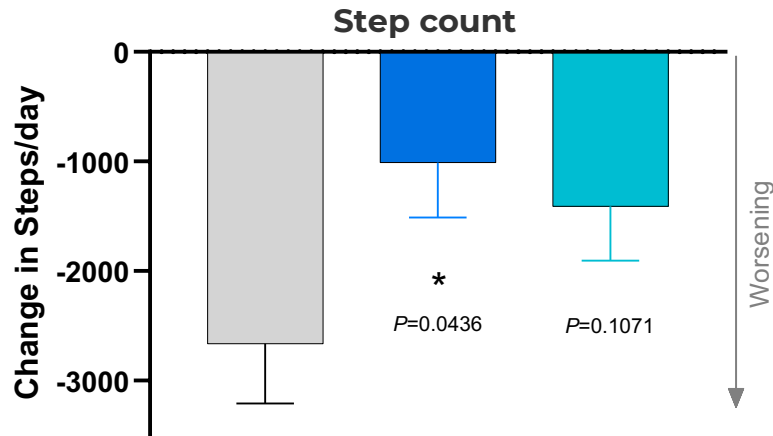


- CGI-S
 - 7-point scale
 - higher score indicates a worse outcome
- Assesses total picture over prior 28 days
 - illness severity, impact of illness on function, level of distress and any other aspects of impairment

ATH434 preserved walking in outpatient setting

Change from baseline to week 52

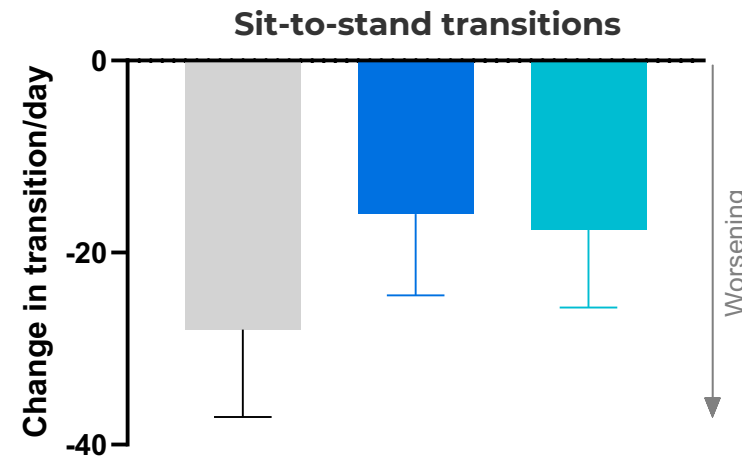
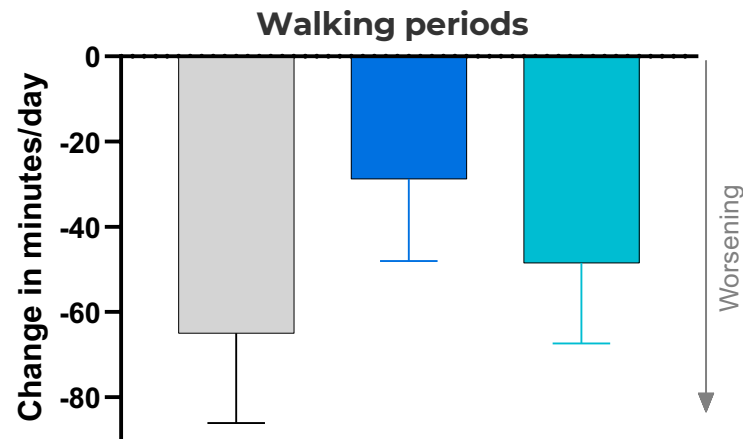
ATH434-201



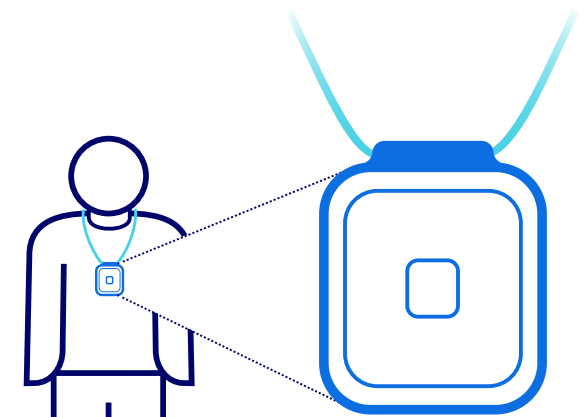
Placebo

ATH434-201
50 mg

ATH434-201
75 mg



Pendant
Wearable movement sensor



Adverse Events

	Placebo twice daily  N=26	ATH434-201 50 mg  N=25	ATH434-201 75 mg  N=26
N (%) of subjects ¹			
Any Adverse Event (AE)	24 (92.3%)	21 (84.0%)	25 (96.2%)
UTI	14 (53.8%)	10 (40.0%)	7 (26.9%)
Fall	8 (30.8%)	7 (28.0%)	8 (30.8%)
Covid-19	1 (3.8%)	6 (24.0%)	4 (15.4%)
Fatigue	2 (7.7%)	1 (4.0%)	5 (19.2%)
Back pain	1 (3.8%)	3 (12.0%)	2 (7.7%)
Severe AEs ²	8 (30.8%)	3 (12.0%)	6 (23.1%)
Serious AEs ²	10 (38.5%)	5 (20.0%)	7 (26.9%)

- Similar rates of AEs in ATH434 and placebo participants
- No severe or serious AEs related to study drug
- No hematologic side effects

1 - Reporting one or more event
2 - None related to Study Drug



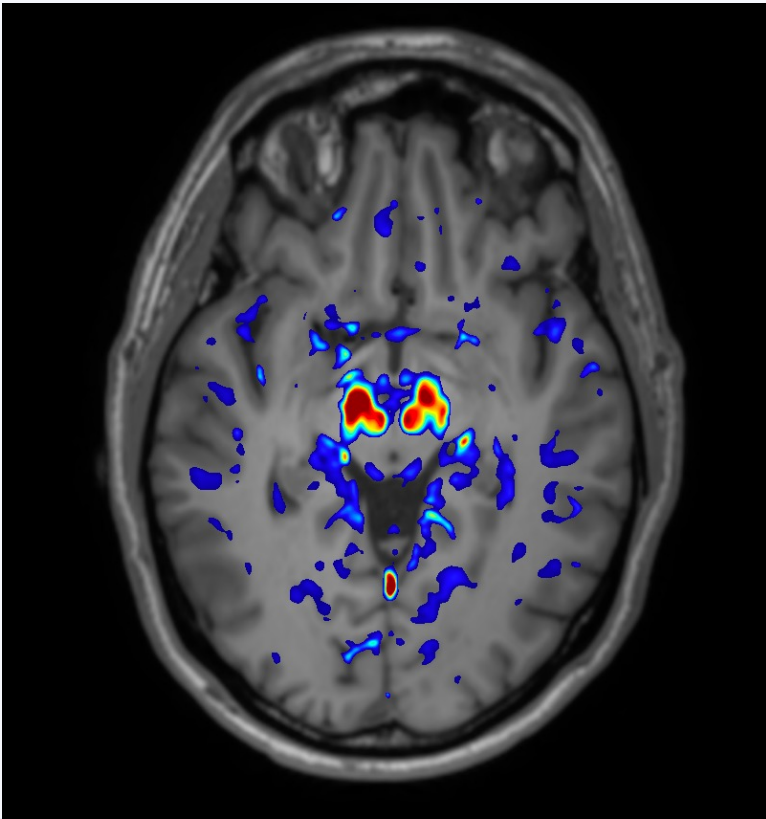
Neuroimaging

Measuring Iron Content with MRI (QSM)

Regional increases in iron in MSA Patient

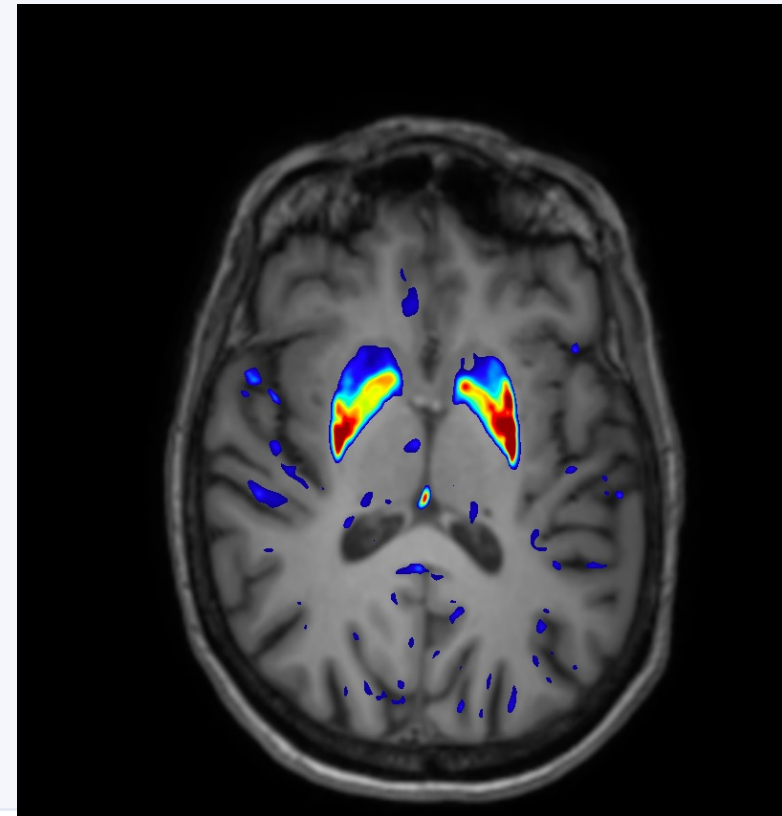
Substantia Nigra

Midbrain axial view



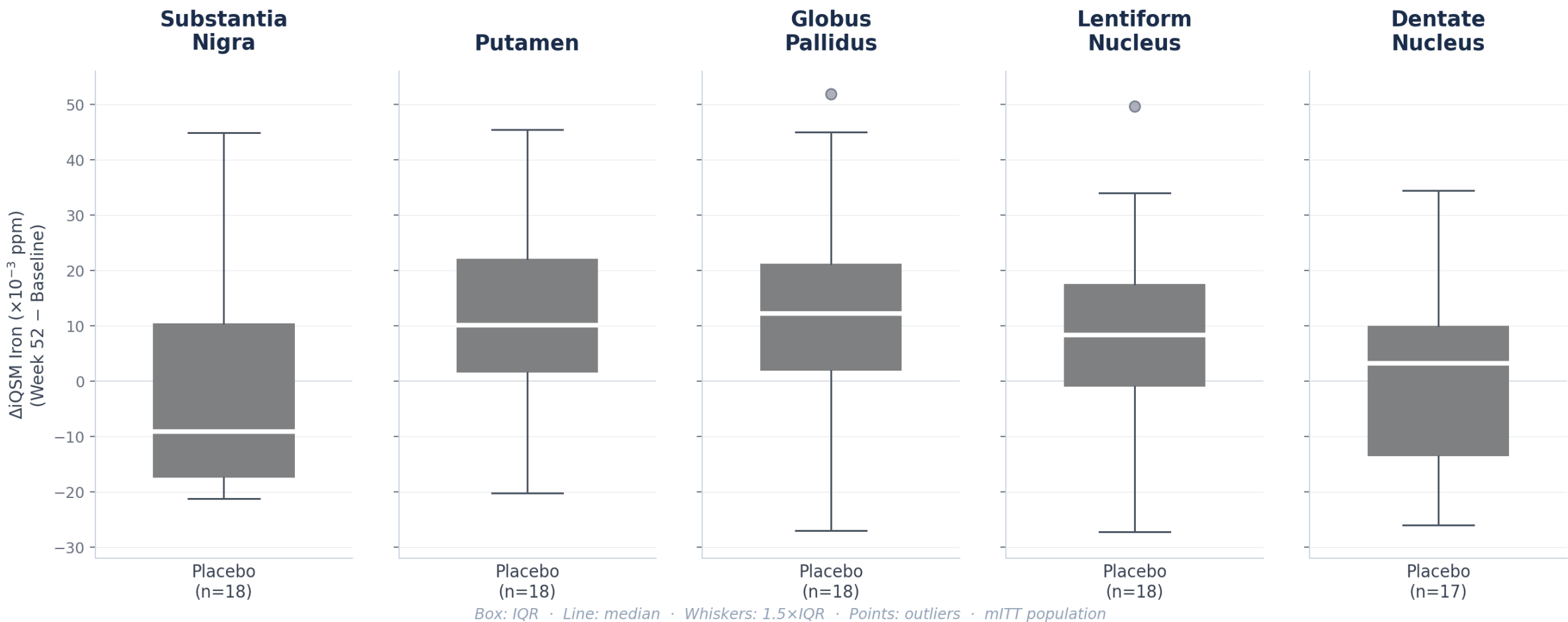
Putamen / Globus Pallidus

Basal ganglia axial view



Iron Deposition on MRI in Placebo: Change Baseline → Week 52

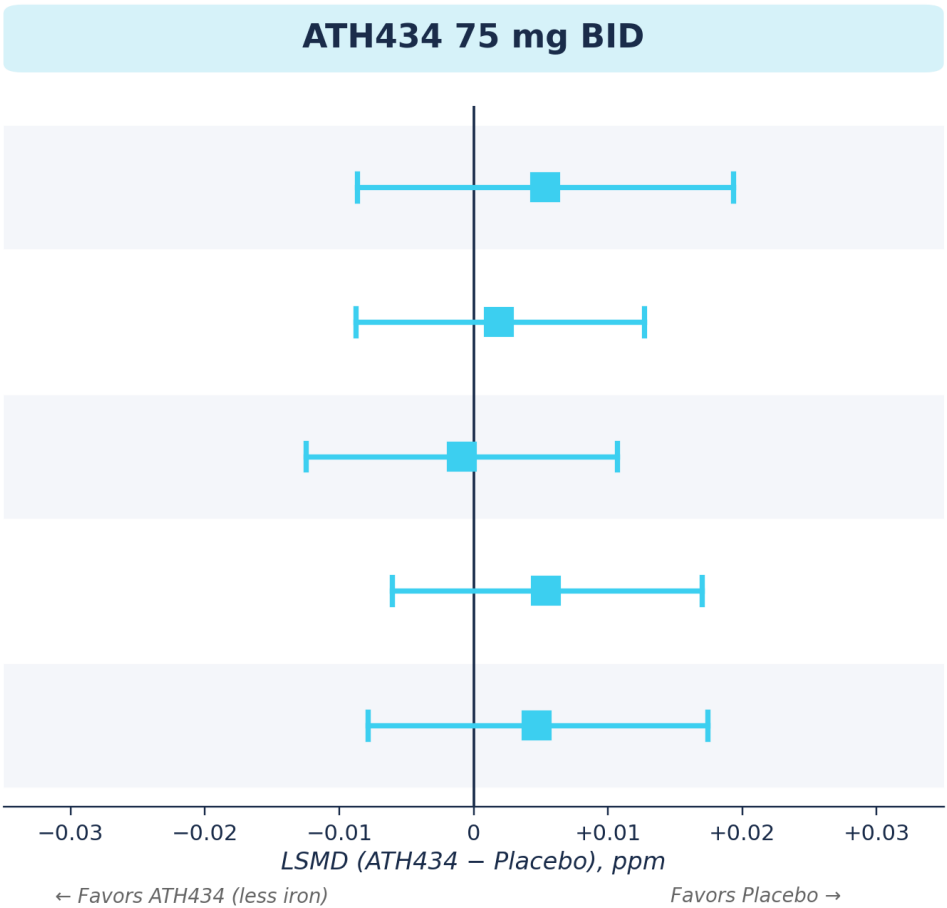
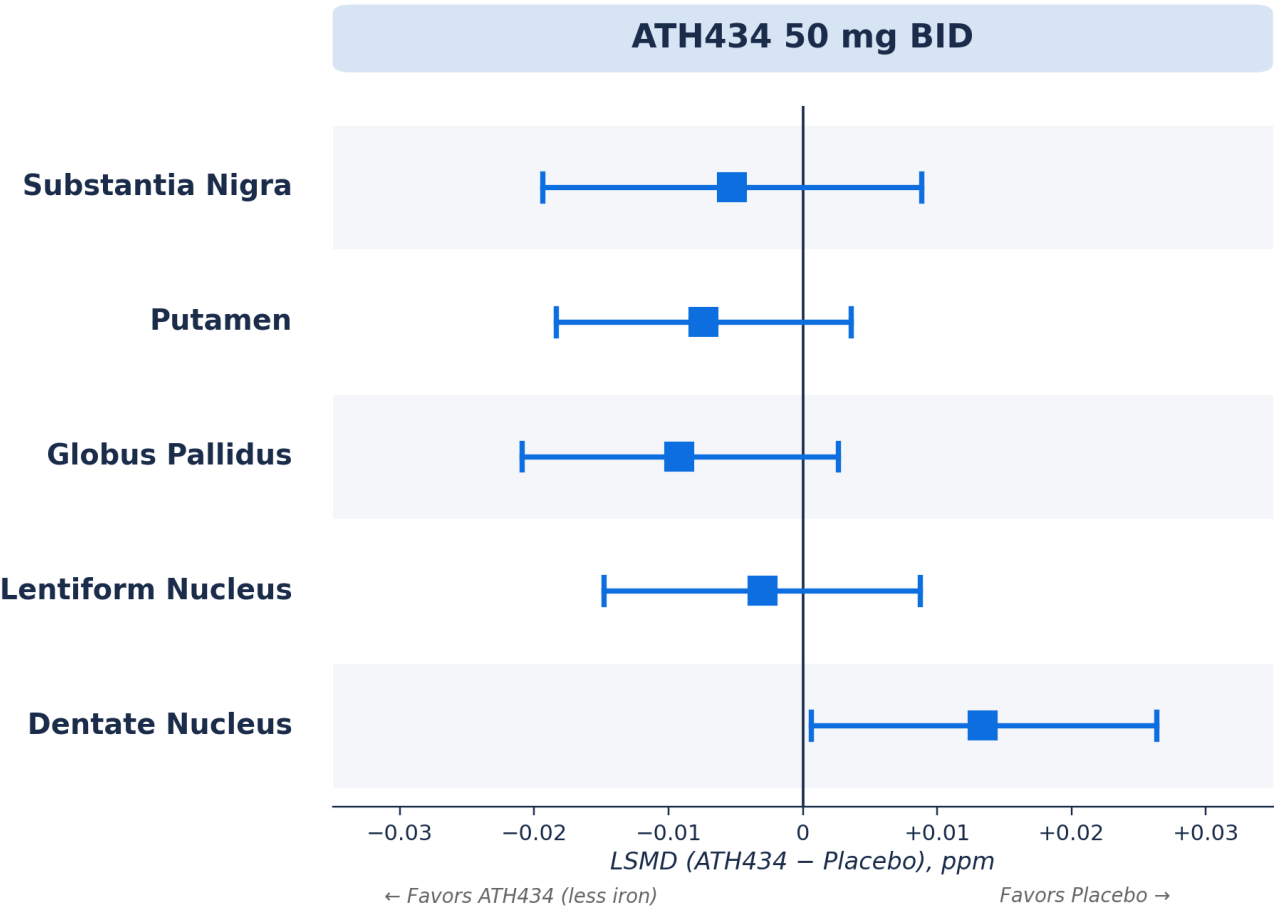
ATH434-201



Mean $\pm$ 95% CI

Iron Deposition on MRI: ATH434 vs. Placebo

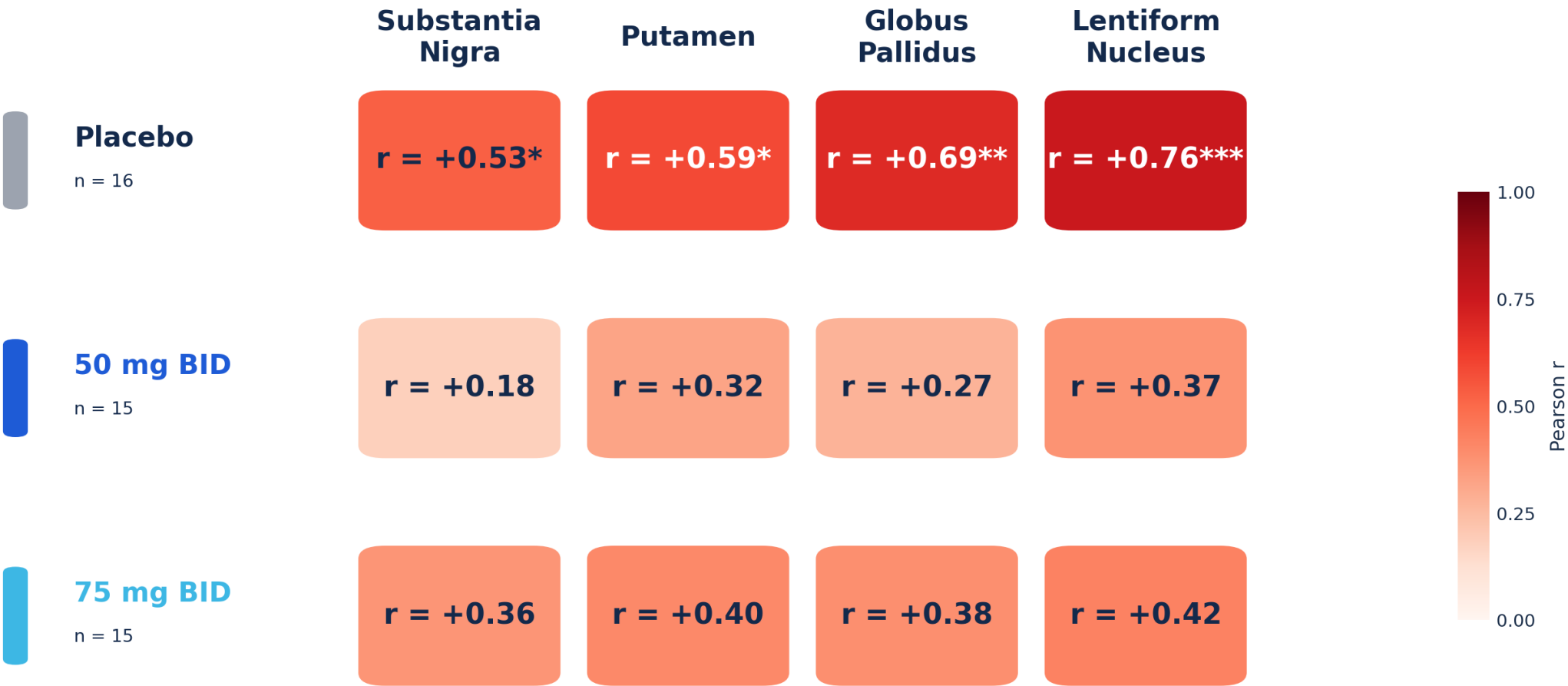
ATH434-201



MMRM with sex, age, baseline CSF NfL, and baseline iron as covariates

Correlation Between Change in Iron and Change in UMSARS I Score at Week 52

ATH434 decouples iron accumulation from clinical worsening

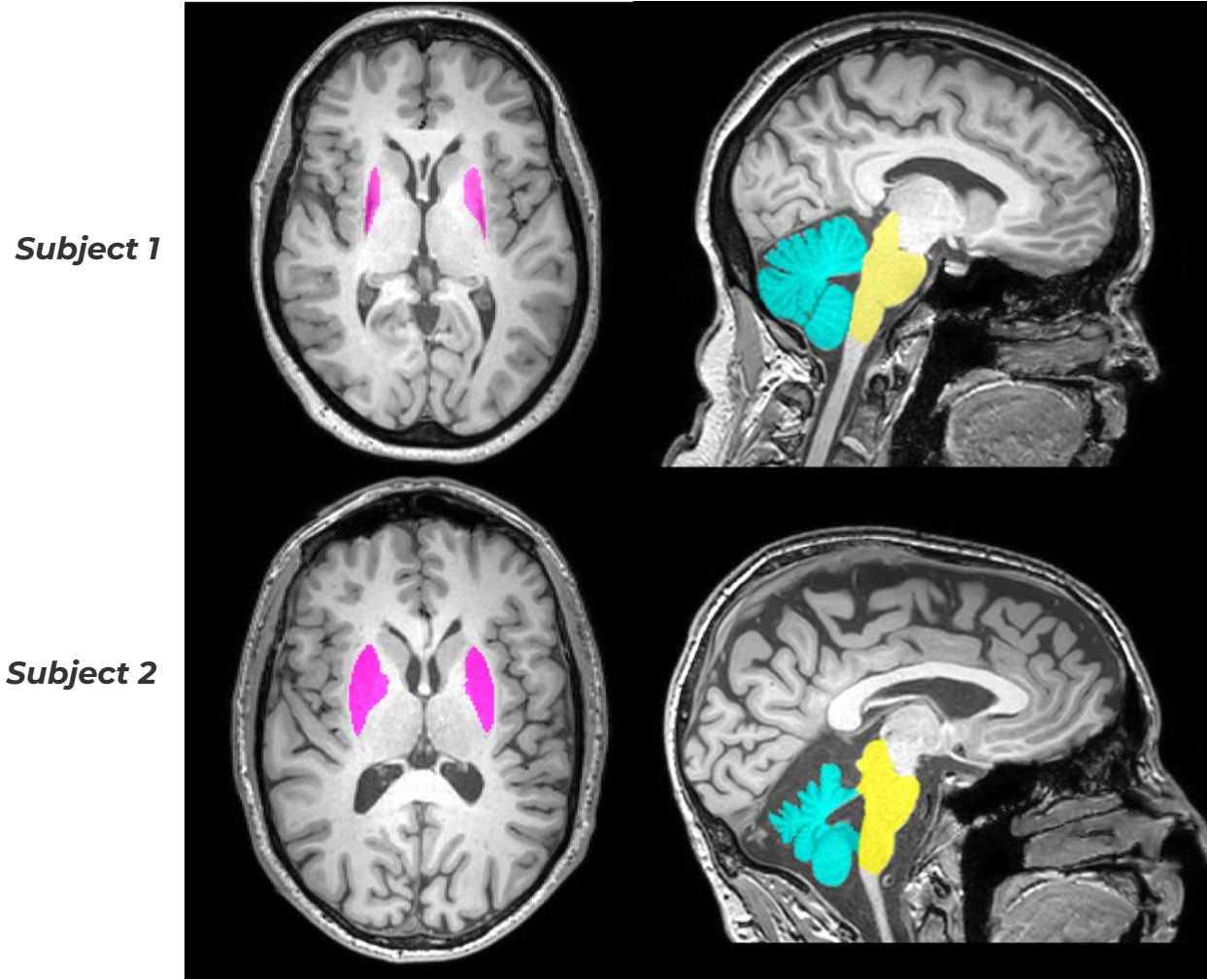


† p<0.10 * p<0.05 ** p<0.01 *** p<0.001 | Residual partial correlation Controlling for Sex, Age, Baseline CSF NfL, Baseline QSM

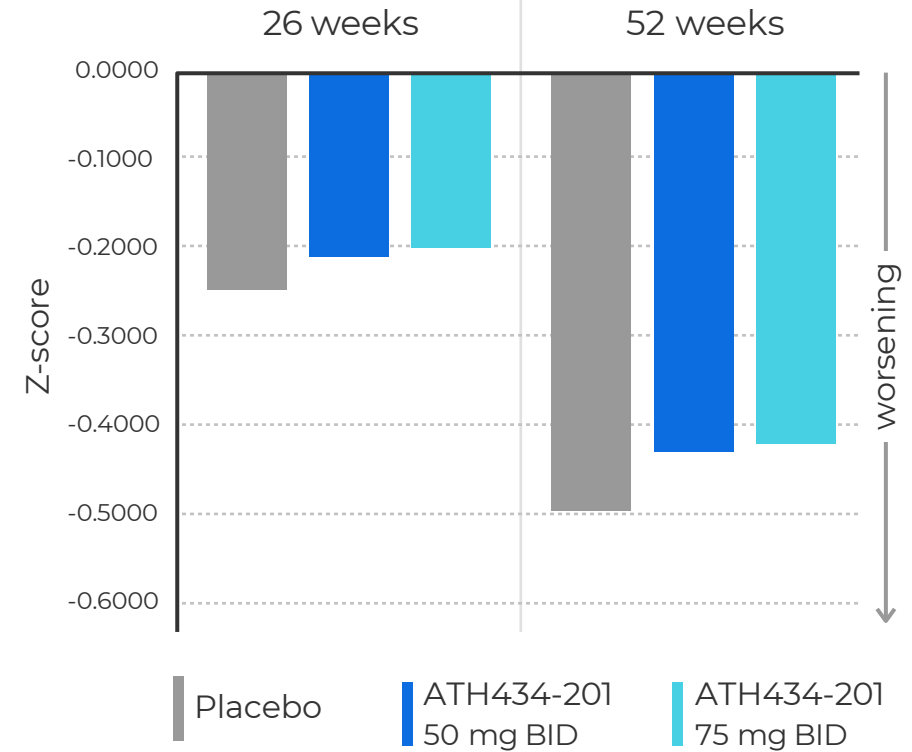
Summary: ATH434 Interrupts the Pathologic Process in MSA

- Iron accumulates in the basal ganglia of MSA patients, most prominently in the globus pallidus and putamen
- QSM allows quantitation of total brain iron but does not distinguish between reactive (toxic in excess) and stable forms of iron
- In placebo-treated patients, change in iron **strongly** correlates with change in disease severity
- In ATH434-treated patients, change in iron **weakly** correlates with change in disease severity
 - ATH434 leads to a **decoupling** of iron accumulation and clinical worsening as assessed by UMSARS I

Measuring Brain Volume in MSA Regions of Interest



Change in Brain Volume*



ATH434 showed trends in preserving brain volume

* MSA Atrophy Index: composite z-score of putamen, GP, cerebellum and brainstem vs. healthy age-matched population

Design	Single arm, open-label
Objective	Assess safety and efficacy in advanced MSA
Population	Advanced MSA (n=10)
Treatment	ATH434 75 mg BID x 12 months
Brain MRI Biomarkers	Volume, Iron
Key Clinical Measure	UMSARS I

Outcomes:

- ✓ Comparable efficacy observed at same dose in double blind study
- ✓ No serious Adverse Events (AEs) related to study drug
- ✓ AEs consistent with underlying disease

The study indicates the potential of ATH434 to slow disease progression in advanced MSA

Carefully designed Phase 2 program demonstrates potential for ATH434 in MSA

ATH434 demonstrated clinically significant efficacy in slowing disease progression in MSA



Both dose levels efficacious on UMSARS I and important secondary endpoints



Demonstrated target engagement with reduced iron accumulation in MSA affected brain regions



Stabilized orthostatic hypotension, one of the most challenging MSA symptoms to manage



Preserved walking in outpatient setting as measured with objective digital biomarker



Open-label trial showed comparable safety and efficacy in advanced MSA



No safety signals and well-tolerated
No serious AEs related to study drug

Successful End-of-Phase 2 meeting with FDA

Alignment on on key elements of the Phase 3 design

- ✓ **Primary endpoint:** 11-Item UMSARS Part I
- ✓ **Population :** ~200 patients with clinical and biomarker evidence of MSA
- ✓ **Dosing regimen:** 50 mg dose administered twice-daily for 12 months
- ✓ **Key secondary endpoints:** Swallowing Disturbance Questionnaire (SDQ), Orthostatic Hypotension Symptom Assessment (OHSA), and the Clinical Global Impression of Severity (CGIS)
- ✓ **Statistics:** Methods for analyzing primary and secondary efficacy endpoints acceptable
- ✓ **Safety database:** Anticipated safety database at the conclusion of Phase 3 reasonable

FDA Alignment on Phase 3 trial Design

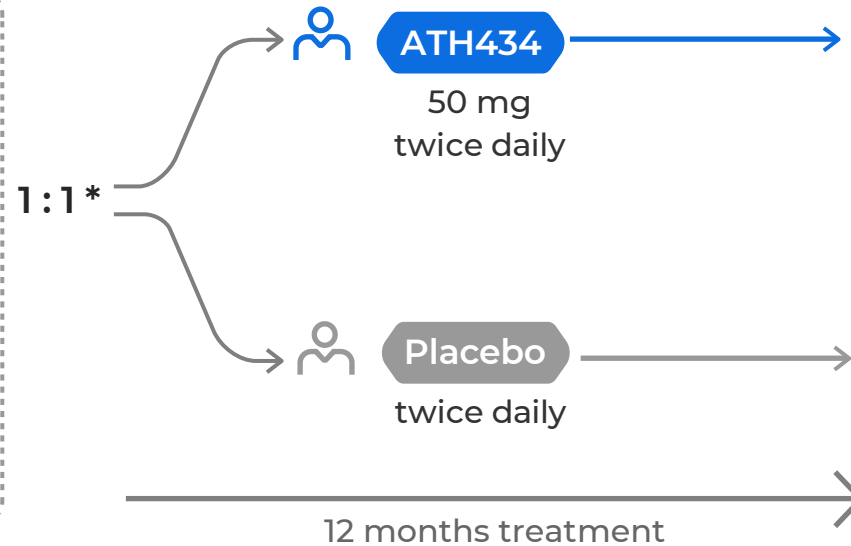
Patient criteria:



N = ~200

- Clinical diagnosis of MSA
- Ambulatory without assistance
- No severe impairment
- Brain atrophy in MSA affected regions on MRI
- Elevated plasma NfL

Study design:



* Randomization stratified by screening plasma NfL

Efficacy Endpoints:



Primary endpoint

11-Item UMSARS Part I
FDA endorsed clinical endpoint



Key Secondary endpoints

SDQ, OHSA, CGIS



Efficacy Analysis

Includes baseline CSF NfL as covariate

Expect to initiate trial activities by YE 2026



Commercial assessment & corporate overview

Independent commercial assessment in MSA

Target product profile based on positive Phase 2 data



Strong Intent to Prescribe

Over 70% of neurologists were “extremely likely” or “very likely” to prescribe ATH434 based on its profile



Substantial Unmet Need

Severely debilitating illness with no approved treatment ripe for new entrants

Critical need for a tolerable, disease modifying therapy



Targeted Mechanism of Action

Importance of inhibiting α -synuclein aggregation to address the underlying pathology of disease



Efficacy is the Key Driver

Slowing disease progression is key driver of physician interest

Stabilizing orthostatic hypotension[^], one of the most challenging symptoms in MSA, strongly positions ATH434

USD \$2.4 Billion

Potential worldwide annual peak sales for ATH434 in MSA

Well Positioned for 2026 Catalysts

Finalize Regulatory Strategy - Align with U.S. FDA on Phase 3 clinical trial

- ✓ Reached alignment in Type C meeting related to clinical pharmacology and non-clinical elements
- ✓ Reached alignment in Type C meeting related to Chemistry Manufacturing & Controls (CMC)
- ✓ Agreement with FDA on Phase 3 study design at End-of-Phase 2 meeting

Phase 3 Readiness activities

- Clinical site identification and qualification
- Manufacture and package of clinical drug supply
- Initiate start-up activities for the trial by year end

Build for Scalable Growth

- ✓ Expanded IP protection with new U.S. Composition of Matter patent to 2045
- Evaluate additional high-value indications to grow the pipeline
- Strengthen the team to enhance organizational capabilities

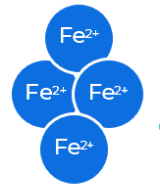
ASX: ATH
NASDAQ: ATHE





APPENDIX

Inadequately chaperoned cellular iron drives MSA pathology



Excess
labile iron

Promotes α -synuclein cross linking¹

Directly increases α -synuclein translation²

ODG toxicity due to limited endogenous glutathione³

Free radicals promote α -synuclein aggregation⁴

Impaired lysosomal autophagy⁵

The Relevance of Iron in the Pathogenesis of Multiple System Atrophy: A Viewpoint

Christine Kaundlstorfer, Kurt A. Jellinger, Sabine Eschlböck, Nadia Stefanova, Günter Weiss, Gregor K. Wenning

Journal of Alzheimer's Disease (2018) DOI 10.3233/JAD-170601

Iron converts native α -SYN into a β -sheet conformation and promotes its aggregation either directly or via increasing levels of oxidative stress. The disturbance of iron homeostasis leads to abnormal iron deposition in the brain and causes neurotoxicity via generation of free radicals and oxidative stress.

Cellular iron deposition patterns predict clinical subtypes of multiple system atrophy

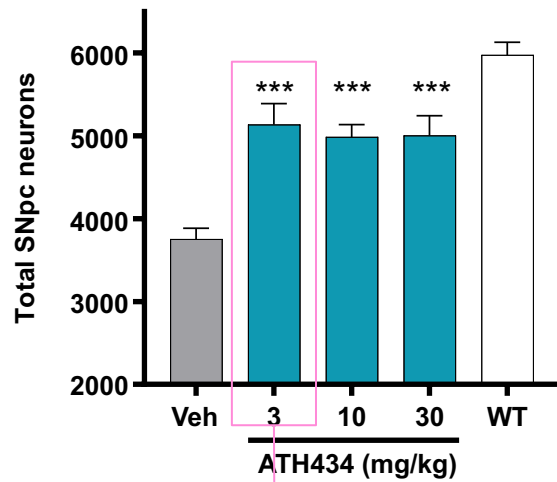
Seojin Lee, Ivan Martinez-Valbuena, Anthony E. Lang, Gabor G. Kovacs

Neurobiology of Disease. 2024. <https://doi.org/10.1016/j.nbd.2024.106535>

Importantly, extensive evidence suggests a molecular relationship between iron accumulation and α -syn pathology.

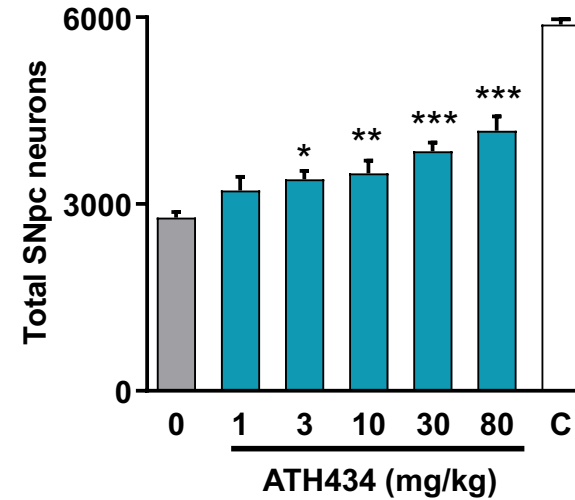
Animal data supporting Phase 2 dose selection

PLP- α -syn MSA Mouse model



Exposure at 3 mg/kg
~ to 50 mg BID in man

MPTP Mouse model



ATH434 affinity for Fe²⁺ and Fe³⁺ relative to endogenous iron binding proteins

	Fe ²⁺		Fe ³⁺	
	μM	constant	μM	constant
ATH434	4.6 ^a	10 ^{-5.33}	0.0003 ^b	10 ^{-9.6}
PCBPs	0.9 ^c 5.8 ^c	10 ^{-6.04} 10 ^{-5.24}		
Glutathione	~8 ^d	10 ^{-5.10}		
Transferrin				10 ^{-23 e}

a: Pall et al 2024

b: Finkelstein et al 2017, consistent with data from (a) collected at pH 5.5 when adjusted for neutral pH. Traditional chelators have constants of ~ 10⁻³⁰ (Crisponi 2015, Hilder 2014)

c: Shi 2008; binds irons with 2 constants

d: Hider 2011

e: Aisen 1978

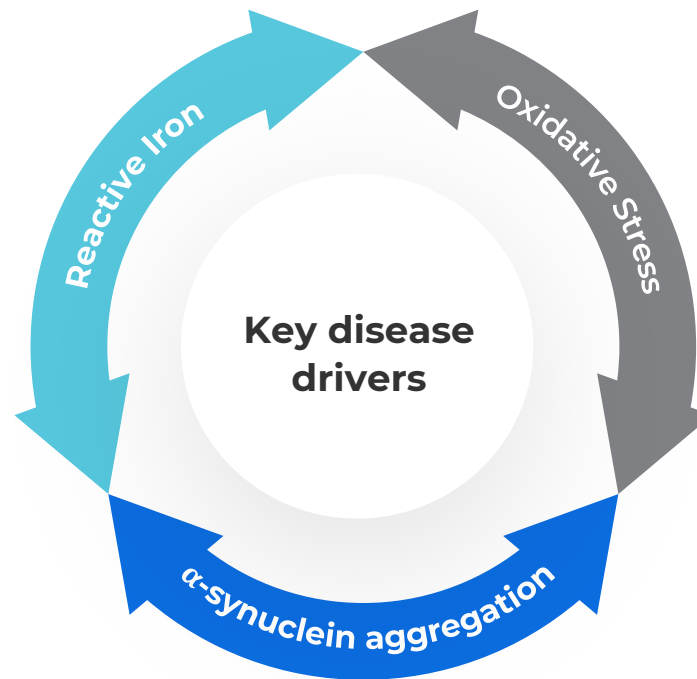
Iron is a key driver of the MSA Pathology

ATH434 chaperones excess iron to reduce neuronal injury

MSA Pathology Cycle

Disrupted Control of CNS Iron

Overwhelms natural iron buffering systems, leading to iron accumulation in MSA brain regions



Reactive iron

Generates free radicals
Promotes α -synuclein aggregation

Oxidative stress

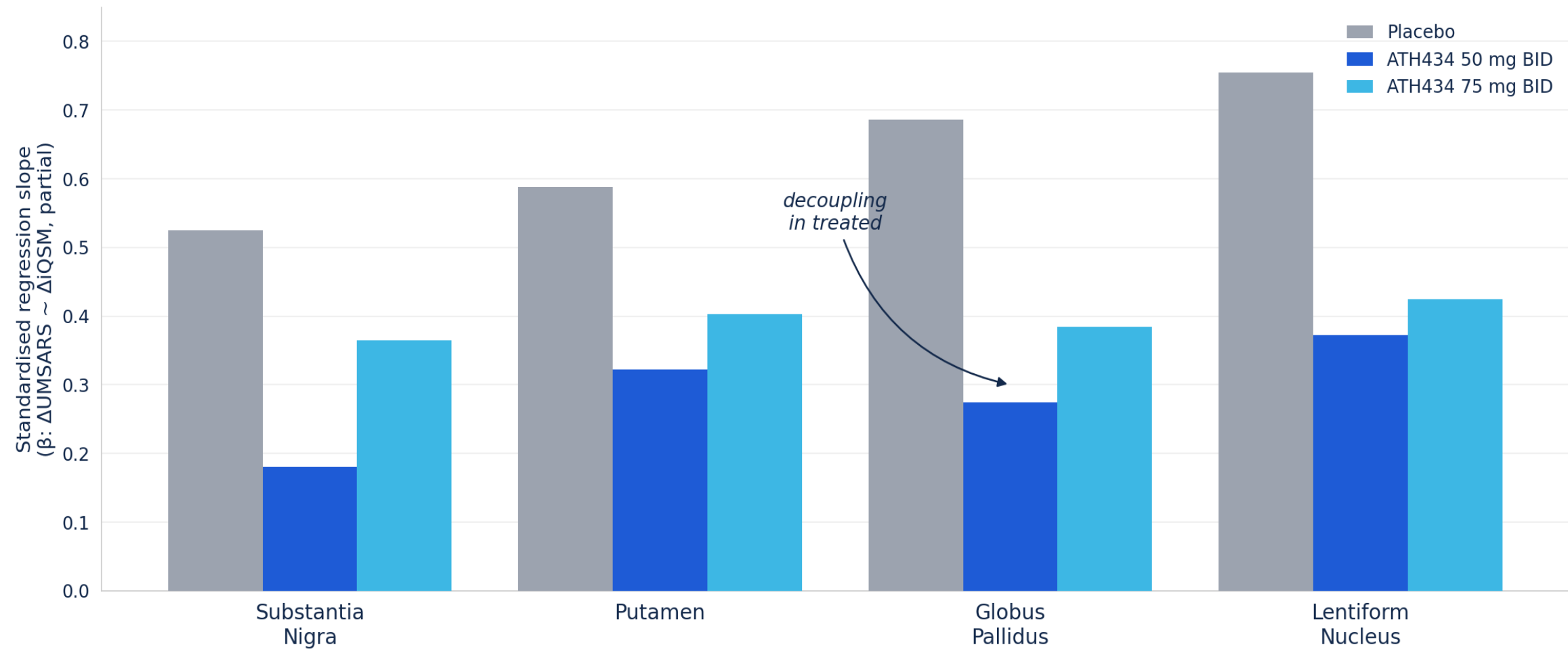
Disrupts multiple cellular functions
Promotes α -synuclein aggregation

α -synuclein aggregation

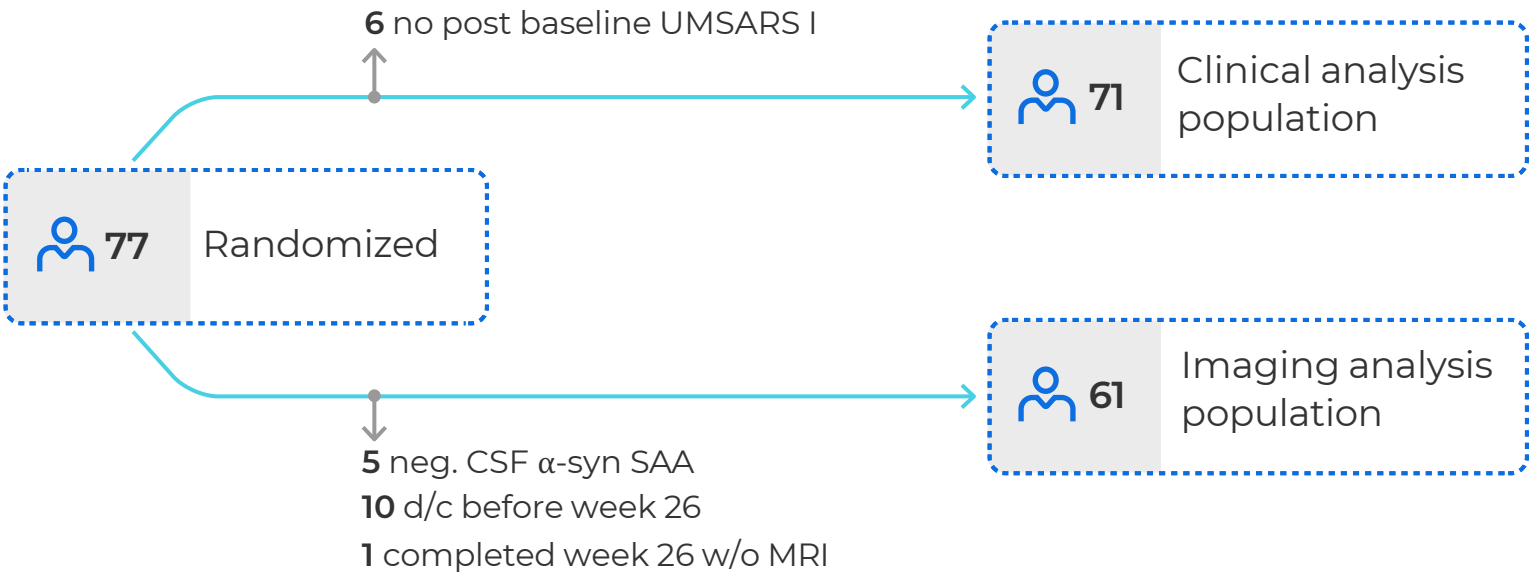
Neuronal toxicity
Impaired myelin production

Correlation Between Change in Iron and Change in UMSARS I Score at Week 52

ATH434 decouples iron accumulation from clinical worsening



Controlling for Sex, Age, Baseline CSF NfL, Baseline QSM



Endpoint	Change from BL to week 52	Population
Biomarker (Primary)	Iron content in s. nigra by MRI	Imaging
Clinical (Key secondary)	Change in Modified UMSARS Part I	Clinical

ATH434-202: Open label study in advanced MSA

Parameter	ATH434-202 75 mg BID  N=10	ATH434-201 75mg BID  N=24
Age (yr)	64.5 (7.5)	63.9 (6.7)
Duration of motor symptoms (yr)	3.9 (1.8)	2.3 (0.9)
Modified UMSARS I ¹	19.2 (5.3)	14.4 (4.4)
Motor score of Parkinson Plus Scale2	57.5 (20.4)	48.9 (16.8)
Plasma NfL (pg/mL)	42.1 (14.1)	32.3 (9.0)
OH Symptom Assessment	16.7 (14.8)	15.0 (12.2)
Severe Orthostatic Hypotension	40.0%	29.2%
	Mean (SD)	

Key objective was to assess efficacy and safety of ATH434 75 mg dose for comparison to 75 mg dose in 201 double-blind study

¹ MSA affected areas by MSA-atrophy index. Trujillo, P. et al Annals of Clin and Trans Neuro, 2025

ATH434-202: Key data at 75 mg dose

Comparison to double blind study at 12 mo

Change over 12 Months	ATH434-202 75 mg BID  N=10	ATH434-201 75mg BID  N=24
Modified UMSARS I	3.5 (4.7)	5.6 (5.6)
Clinical global impression of change (% stable)	30%	21%
Patient global impression of change (% stable)	30%	26.4%
Brain volume ¹	-0.44 (0.14)	-0.42 (0.29)
	Mean (SD)	

The 75 mg dose demonstrated comparable efficacy to that observed in the double-blind study

- No serious AEs related to study drug
- AEs consistent with underlying disease

¹In MSA affected areas by MSA-atrophy index. Trujillo, P. et al. Annals of Clin and Trans Neuro, 2025