Building the World's Leading Neuromuscular Disease Company

COMPANY OVERVIEW | JANUARY 2025

Sarah, *living with DM1*

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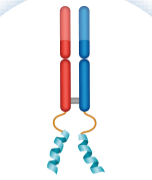
OUR MISSION

Life-transforming therapies

for patients with serious muscle diseases

Aiming to Deliver Transformative Therapies for Neuromuscular Diseases

LATE-STAGE PIPELINE	Two clinical programs moving to registrational expansion cohorts for DM1 and DMD following positive proof-of-concept data
NEAR-TERM VALUE DRIVERS	Key data readouts in 2025 & 2026 potentially enabling two submissions for U.S. Accelerated Approval in 2026
DIFFERENTIATED PLATFORM	FORCE™ platform enables targeted delivery to muscle and CNS; broader pipeline includes FSHD and Pompe
STRONG FINANCIAL POSITION	Cash position of ~ $642 million (as of 12/31/24)* with expected runway into H2 2026; all assets fully owned

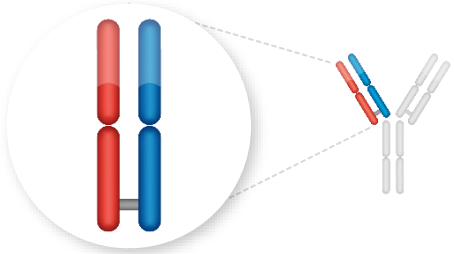


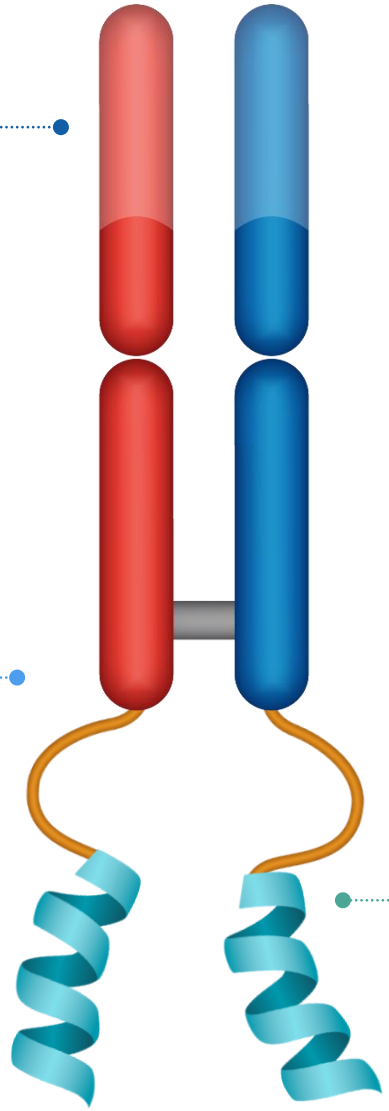
Note: DM1 = myotonic dystrophy type 1; DMD = Duchenne muscular dystrophy; FSHD = facioscapulohumeral muscular dystrophy
* Preliminary and unaudited

Dyne FORCE™ Platform: Modern Oligo Therapeutics for Muscle Diseases

ANTIBODY

Proprietary Fab targets TfR1 to enable muscle delivery



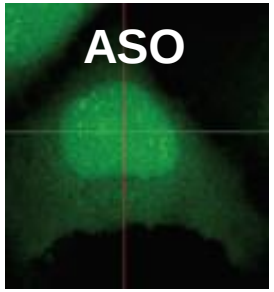


LINKER

Clinically validated, enables precise conjugation of multiple payloads to a single Fab

PAYLOAD

Modularity enables rational selection of payload to target the genetic basis of disease

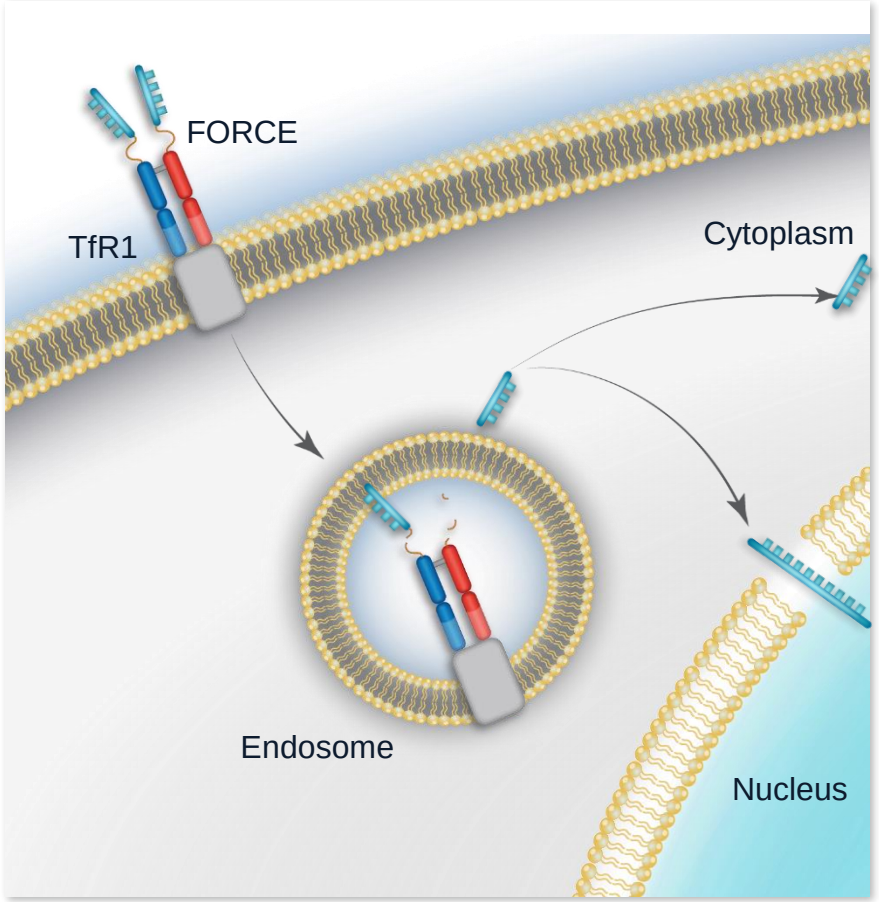


Nuclear localization

Cytoplasmic localization

Adapted from Ohrt T., et al. *Nucleic Acids Res* 2006;34:1369.

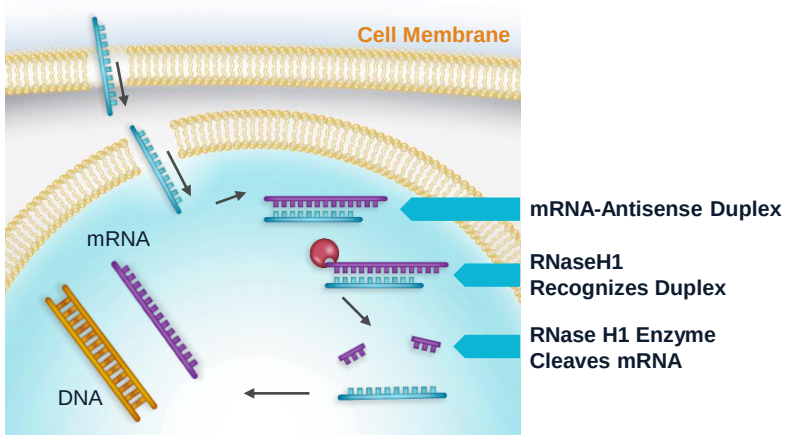
FORCE Platform Harnesses Cell Biology to Modify Disease



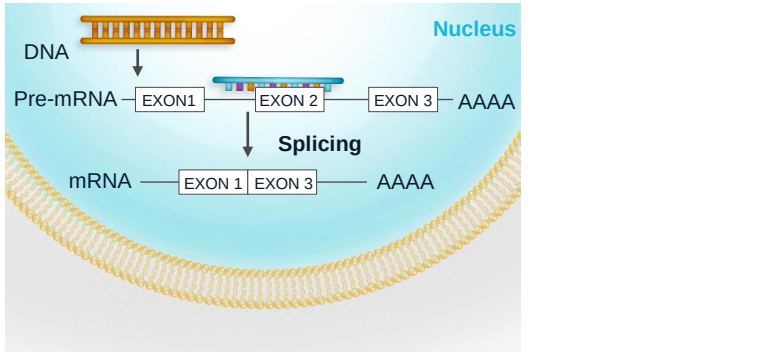
- Harnesses natural mechanism of TfR1 receptor-mediated delivery to transport therapeutics across the cell membrane
- Achieves endosomal escape without any membrane-destabilizing agents
- Distinctive pharmacokinetic profile creates opportunity for durable target engagement and wide therapeutic index

Rationally Select Payload to Target Genetic Basis of Disease

ASO acts in the nucleus and cytoplasm

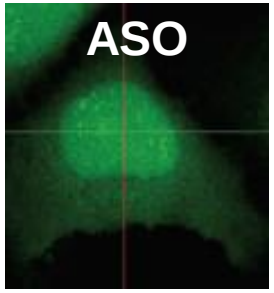


Splice-modulating ASO

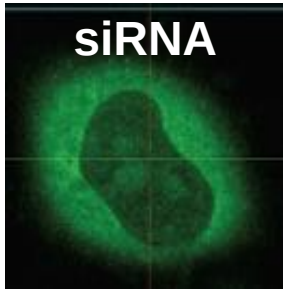


Single-Stranded Antisense

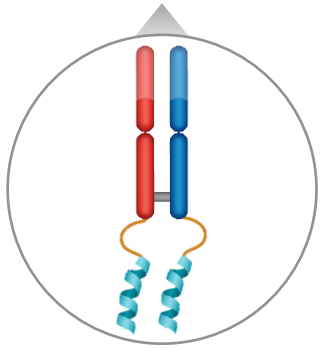
Subcellular distribution of ASO and siRNA



Nuclear localization

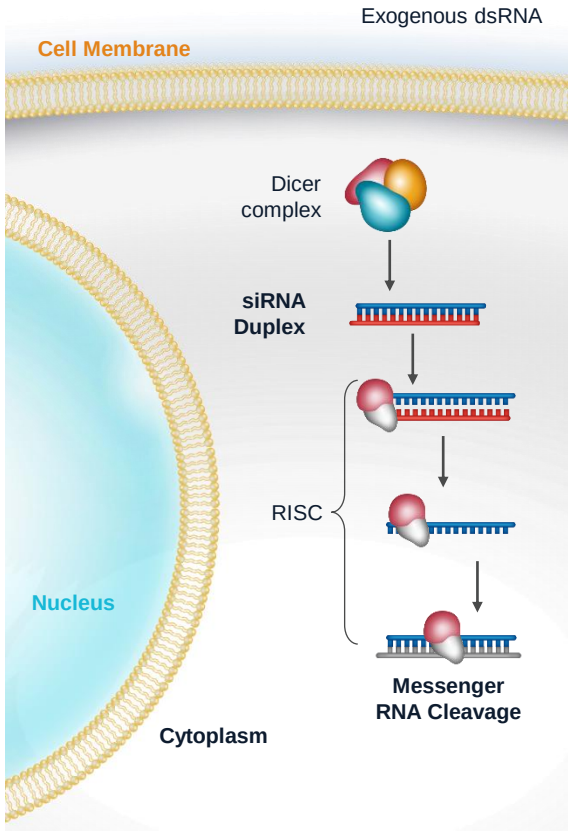


Cytoplasmic localization



FORCE delivers **ASO** payload for nuclear targets, **siRNA** payload for cytoplasmic targets

siRNA acts in the cytoplasm



Double-Stranded Antisense (siRNA)

FORCE Platform Designed to Deliver Significant Advantages

Targeted Muscle Delivery

Leverages TfR1 expression
on skeletal, cardiac and smooth muscle

Targets Genetic Basis of Disease

Rationally select payloads
to match target biology

Redosable Administration

Potential for individualized patient
titration and longer-term efficacy

Enhanced Tolerability

Targeted delivery limits systemic
drug exposure

Extended Durability

Potential for prolonged disease-modifying
effects, enabling less frequent dosing

Reduced Development and Manufacturing Costs

A single Fab and linker
utilized across all programs

Robust Portfolio Focused on Neuromuscular Diseases with Opportunities to Expand by Leveraging FORCE Delivery

DISEASE	TARGET	DISCOVERY / PRECLINICAL / PHASE 1/2	ESTIMATED PATIENTS
Myotonic Dystrophy Type 1 (DM1)	DMPK	DYNE-101	US: **>40,000** Europe: **>74,000**
Duchenne Muscular Dystrophy (DMD)	Exon 51	DYNE-251	US: **~12,000-15,000** Europe: **~25,000**
	Exon 53		
	Exon 45		
	Exon 44		
	Other Exons		
Facioscapulohumeral Muscular Dystrophy (FSHD)	DUX4	DYNE-302	US: **~16,000-38,000** Europe: **~35,000**
Pompe disease	GAA	DYNE-401	US: **~3,800** Europe: **~7,000**

Pipeline expansion opportunities in CNS, rare skeletal, cardiac and metabolic

Developing Transformative Therapies for People Living with DM1

Overview

- Mutation in the *DMPK* gene
- Onset at any point, depending on DM1 phenotype
- Life expectancy of 45 - 60 years

Clinical Presentation

- Myotonia
- Muscle weakness
- Cardiac arrhythmia
- Pulmonary abnormalities
- CNS manifestations

Population

- >40,000 (US)
- >74,000 (Europe)

NO approved therapies

OUR APPROACH

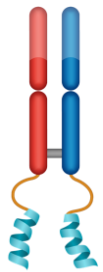
Disease-Modifying Nuclear *DMPK* Knockdown

Targeting toxic gain of function *DMPK* RNA to potentially **stop or reverse** disease progression

Note: CNS = central nervous system

DYNE-101 Addressing the Central Pathobiology of DM1 to Enable Broad Functional Improvement[1]

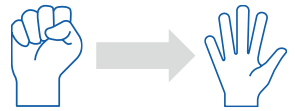
Robust and widespread delivery → ***DMPK* degradation in the nucleus** → **MBNL release and splicing correction** → **Correction of myotonia and broad functional improvement**



DYNE-101

Differentiated MOA

MBNL released:
Splicing corrected



Early effect on myotonia

Muscle Strength:
Quantitative Muscle Testing

Functional Assessments:
10-Meter Walk / Run;
5 Times Sit to Stand

Patient Reported Outcomes:
Myotonic Dystrophy Health Index (MDHI)

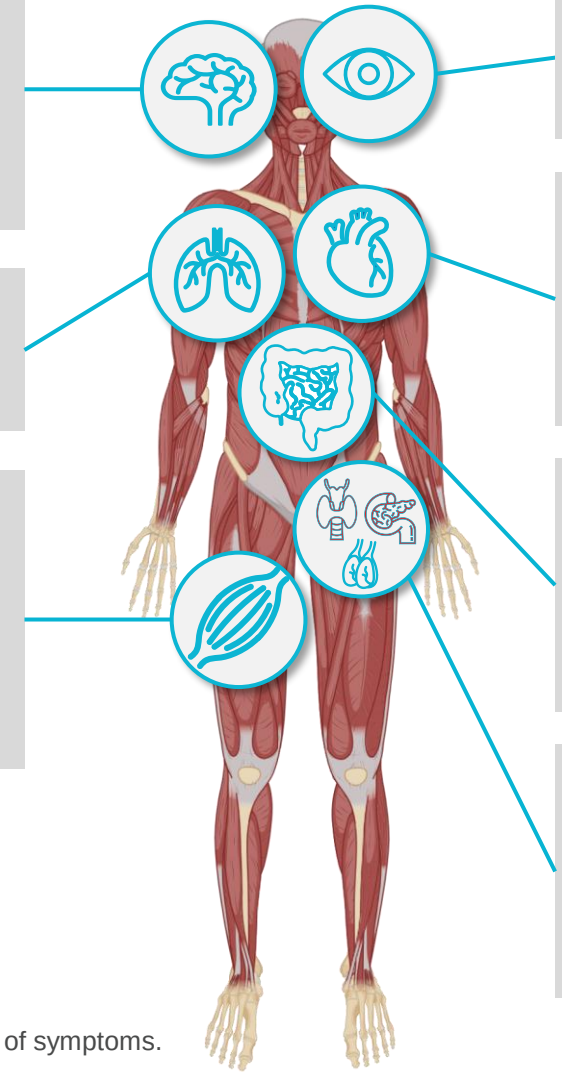
1. Image depicts the intended DYNE-101 mechanism of action.

DM1 is a Heterogeneous Disease with Widespread Muscle and CNS Manifestations

CNS[1–4]
- Fatigue
- Excessive daytime sleepiness
- Difficulty concentrating
- Behavioral/personality changes

Skeletal muscle (respiratory)[1–4]
- Restrictive ventilatory pattern
- Shortness of breath

Skeletal muscle[1–4]
- Muscle weakness
- Myotonia
- Balance issues
- Muscle pain
- Atrophy



Ocular[1–4]
- Cataracts
- Ptosis

Cardiac[1–4]
- Conduction disturbances
- Arrythmia
- Cardiomyopathy
- Sudden death

Smooth muscle[1–4]
- Dysphagia
- Constipation
- Heartburn
- Regurgitation

Endocrine[1–4]
- Thyroid disorders
- Diabetes
- Male hypogonadism
- Vitamin D deficiency

Slide does not represent an exhaustive list of symptoms.

1. Thornton CA. *Neurol Clin.* 2014;32:705-719; 2. Ho G, et al. *World J Clin Pediatr*. 2015;4:66–80.
3. Hagerman KA, et al. *Muscle Nerve.* 2019;59:457–464; 4. Gutierrez Gutierrez G, et al. Neurologia (Engl Ed). 2020;35:185–206.

Multiple Ascending Dose (MAD) Portion of ACHIEVE is Complete

ACHIEVE

Population	Primary Endpoints	Additional Endpoints	Stages of ACHIEVE
• Adult patients living with DM1 • Ages 18 to 49 years	• Safety and tolerability	• Pharmacokinetics • Change from baseline of: – Splicing – *DMPK* RNA expression – Multiple assessments of muscle strength and function – Patient-reported outcomes, including DM1-ACTIV^{c} and MDHI	✓ Multiple Ascending Dose (MAD): 24 weeks • Open-Label Extension (OLE): 24 weeks • Long-Term Extension (LTE): 96 weeks ↓ **Registrational Expansion Cohort**

Additional endpoints include select secondary and exploratory endpoints. DM1-ACTIV^{c}: Myotonic Dystrophy type 1 Activity and participation scale. MDHI: Myotonic Dystrophy Health Index.

ACHIEVE Baseline Participant Characteristics: By Treatment

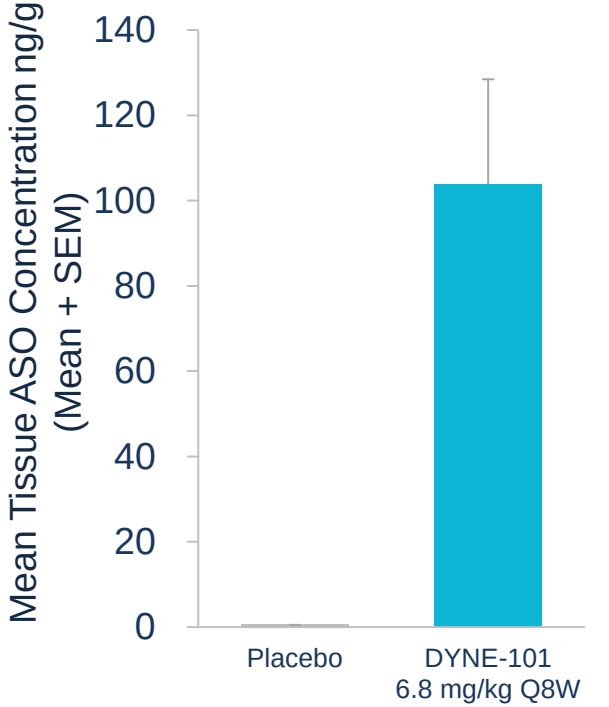
Mean (SD)	Placebo (N=14)	1.8 mg/kg Q4W (N=6)	3.4 mg/kg Q4W (N=6)	5.4 mg/kg Q8W (N=6)	6.8 mg/kg Q8W (N=6)
Age (years)	32.6 (9.6)	37.0 (10.5)	31.2(4.4)	40.2 (6.5)	37.2 (9.7)
BMI (kg/m^2)	24.4 (4.7)	21.6 (5.8)	21.1 (1.8)	21.4 (2.5)	23.4 (5.6)
CASI-22	0.68 (0.20)	0.64 (0.25)	0.75 (0.12)	0.82 (0.16)	0.74 (0.25)
CTG Repeats	597 (246)	303 (163)	652 (258)	482 (236)	542 (191)
vHOT (sec) (middle finger)	7.5 (3.0)	11.3 (4.4)	6.6 (3.9)	11.9 (5.7)	7.8 (3.8)
QMT Total (% predicted)	51.5 (14.3)	48.1 (10.6)	42.0 (12.6)	46.6 (17.7)	51.3 (10.4)
10MWR (sec)	3.34 (0.48)	3.39 (0.55)	3.48 (0.67)	5.1 (2.40)	3.94 (1.56)
5 Times Sit to Stand (sec)	9.24 (2.03)	9.47 (2.04)	8.75 (1.88)	12.78 (6.79)	9.98 (3.33)
DM1-ACTIV^c Total	47 (NA[a])	46 (4.59)	38 (4.65)	44 (6.99)	43.4 (NA[a])
MDHI Total	18.7 (13.8)	23.5 (23.2)	30.2 (23.2)	14.8 (7.4)	26.5 (13.7)

[a] SDs for DM1-ACTIV ^{c} are not reported to maintain blinding.

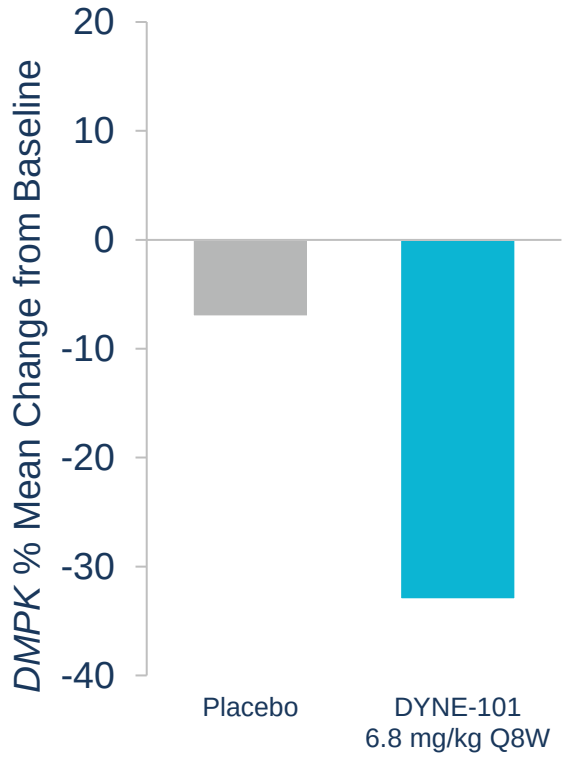
DYNE-101 Addresses Central Pathobiology: Differentiated Pharmacology with Potential to Lead to Broad Functional Benefit

Delivery and exposure → ***DMPK* knockdown** → **Splicing correction** → **Broad functional benefit**

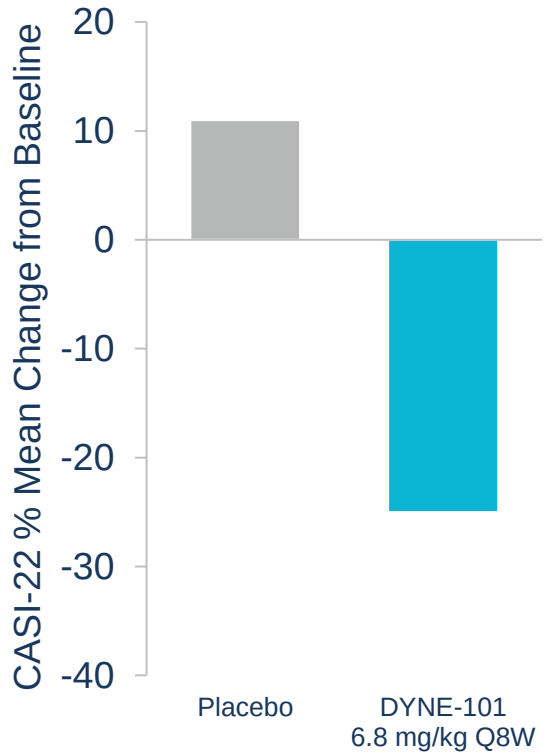
Muscle delivery at 3 mo



Target engagement at 3 mo



Biomarker response at 3 mo



Benefit at 6 mo

Notes: CASI-22 = composite alternative splicing index; vHOT = video hand opening time; QMT = quantitative muscle testing; 10MWR = 10-meter walk/run test; 5xSTS = 5 times sit to stand; 3 months = 85 days, 6 months = 169 days.

DYNE-101: Favorable Safety Profile with No Serious Related TEAEs

Summary of Treatment Emergent Adverse Events (TEAEs)[1]

TEAE Category	Participants with ≥1 TEAE – n (%)					
	1.8 mg/kg Q4W+Rec. N=16	3.4 mg/kg Q4W+Rec. N=16	3.4 mg/kg Q8W N=8	5.4 mg/kg Q8W N=8	6.8 mg/kg Q8W N=8	Overall (N=56)
Any TEAE	16 (100%)	16 (100%)	8 (100%)	8 (100%)	8 (100%)	56 (100%)
Any related TEAE	9 (56%)	9 (56%)	2 (25%)	3 (38%)	6 (75%)	29 (52%)
Any serious TEAE	4 (25%)	0	1 (13%)	0	0	5 (9%)
Any serious related TEAE	0	0	0	0	0	0
Any TEAE leading to withdrawal from study	0	0	0	0	0	0
Any TEAE leading to death	0	0	0	0	0	0

Most TEAEs Were Mild or Moderate in Intensity[1]

- 6 serious TEAEs unrelated to study drug
  - Atrioventricular block first degree (1)[2]
  - Pneumonia (2 events in same participant)
  - Pulmonary embolism (1)[3]
  - Hyponatremia (1)
  - Influenza (1)
- Most common TEAEs (≥20% participant incidence)[4]
  - Nasopharyngitis (38%)
  - Procedural pain (30%)
  - Influenza (27%)
  - Infusion-related reaction (25%)
  - Diarrhea; headache (each 21%)

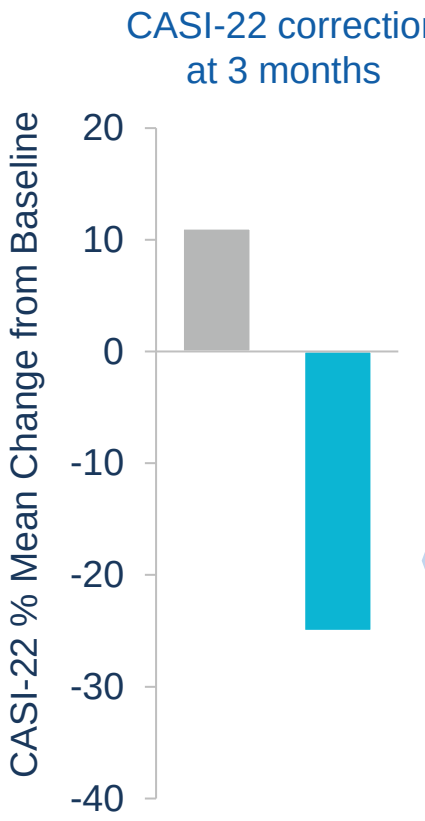
Additional Safety Data

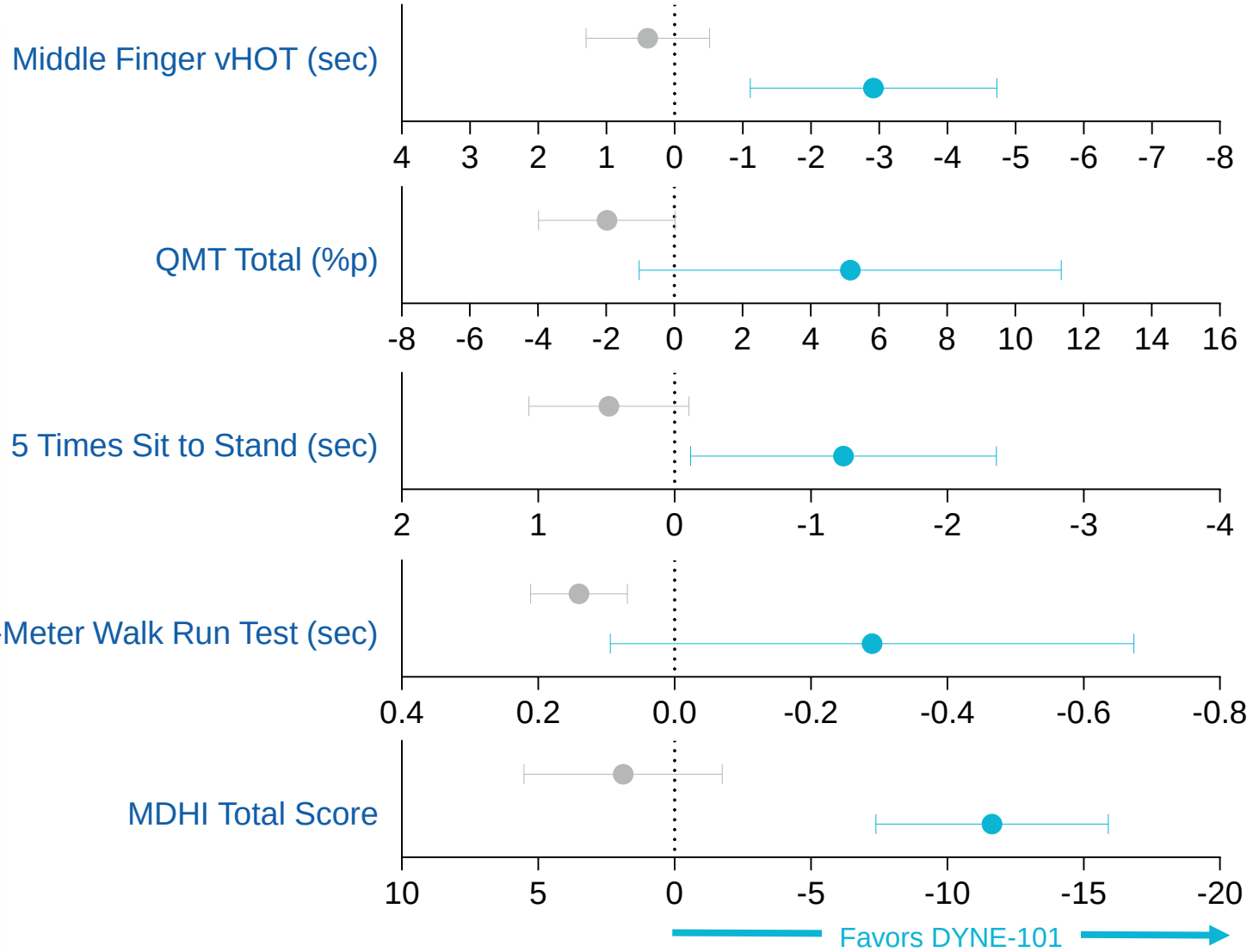
- Liver enzyme elevations have been observed in a minority of participants
  - No impact on liver function (bilirubin or coagulation)
  - Interpretation is complicated by underlying disease and elevated baseline values up to ~2.5x greater than the upper limit of normal
- No participants have demonstrated persistent related anemia or thrombocytopenia

~855 Doses Administered to Date Representing Over 72 Patient-Years of Follow-Up[1]

1. Data as of December 6, 2024; 2. Transient worsening of atrioventricular (AV) block in a participant with ongoing medical history of first-degree AV block; 3. Attributed to risk factors for pulmonary embolism; 4. All cohorts combined; preferred terms are reported.

Broad Improvement Demonstrated at 6 Months with Planned Registrational Dose of 6.8 mg/kg Q8W





Note: 3 months = 85 days; 6 months = 169 days.

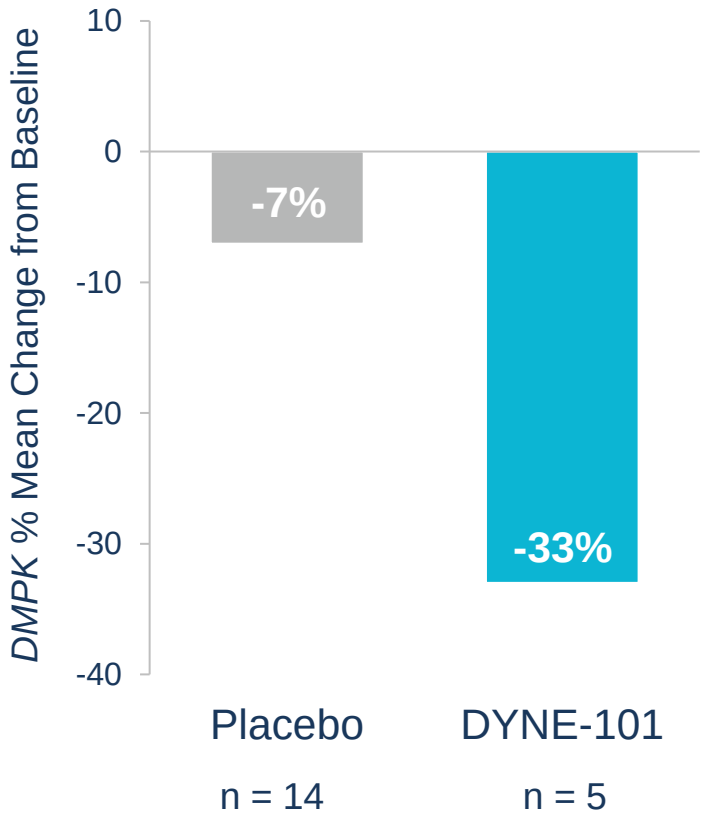
Placebo (N = 14)

DYNE-101 6.8 mg/kg Q8W (N = 6)

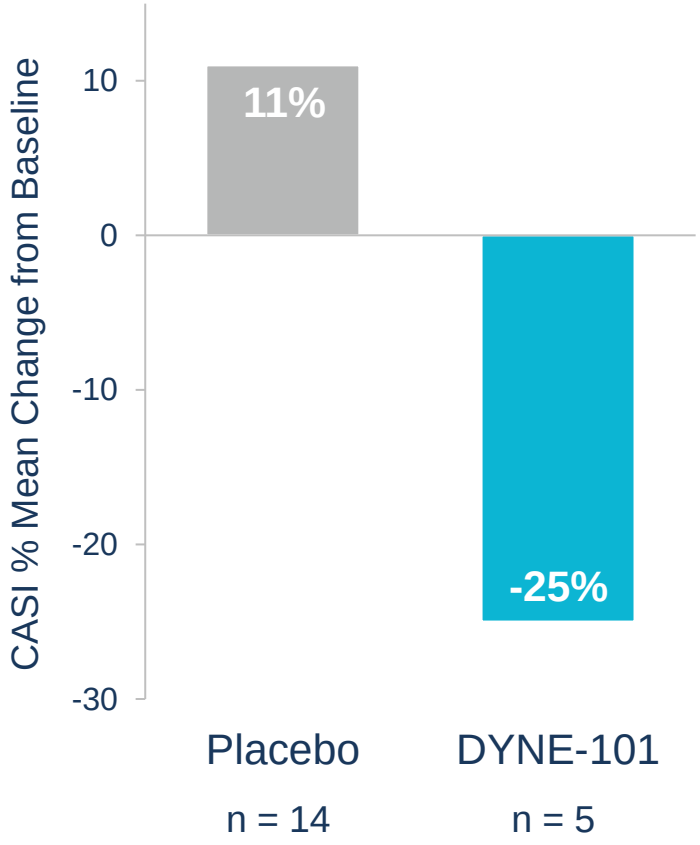
Data presented are mean change from baseline ± SEM

DYNE-101 at 6.8 mg/kg Q8W Improved Foundational Pathobiology of DM1 at 3 Months

DMPK

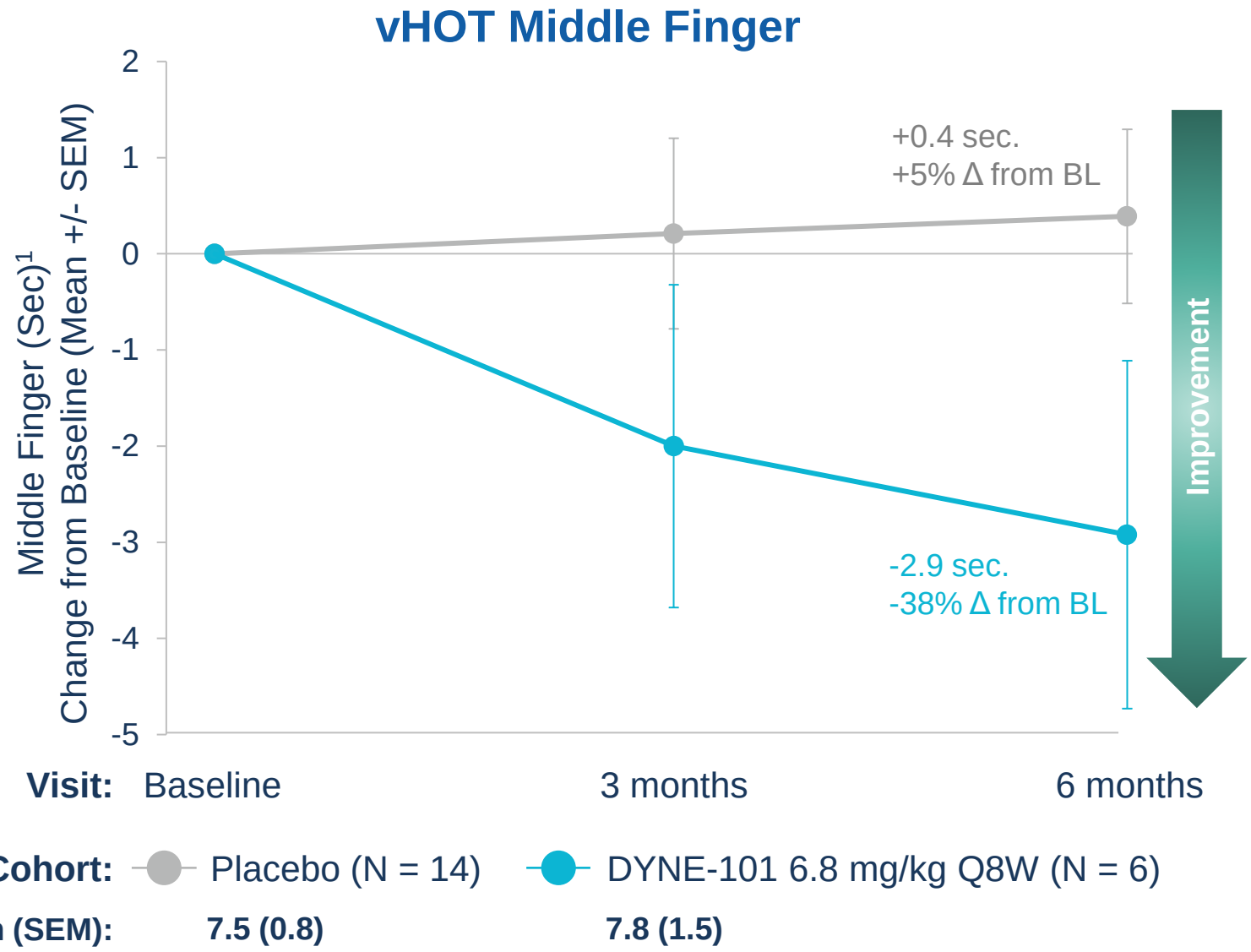


Splicing correction



Notes: One baseline sample in 6.8 mg/kg treatment groups not included within splicing assay as the sample did not meet QC criteria. 3 months = 85 days.

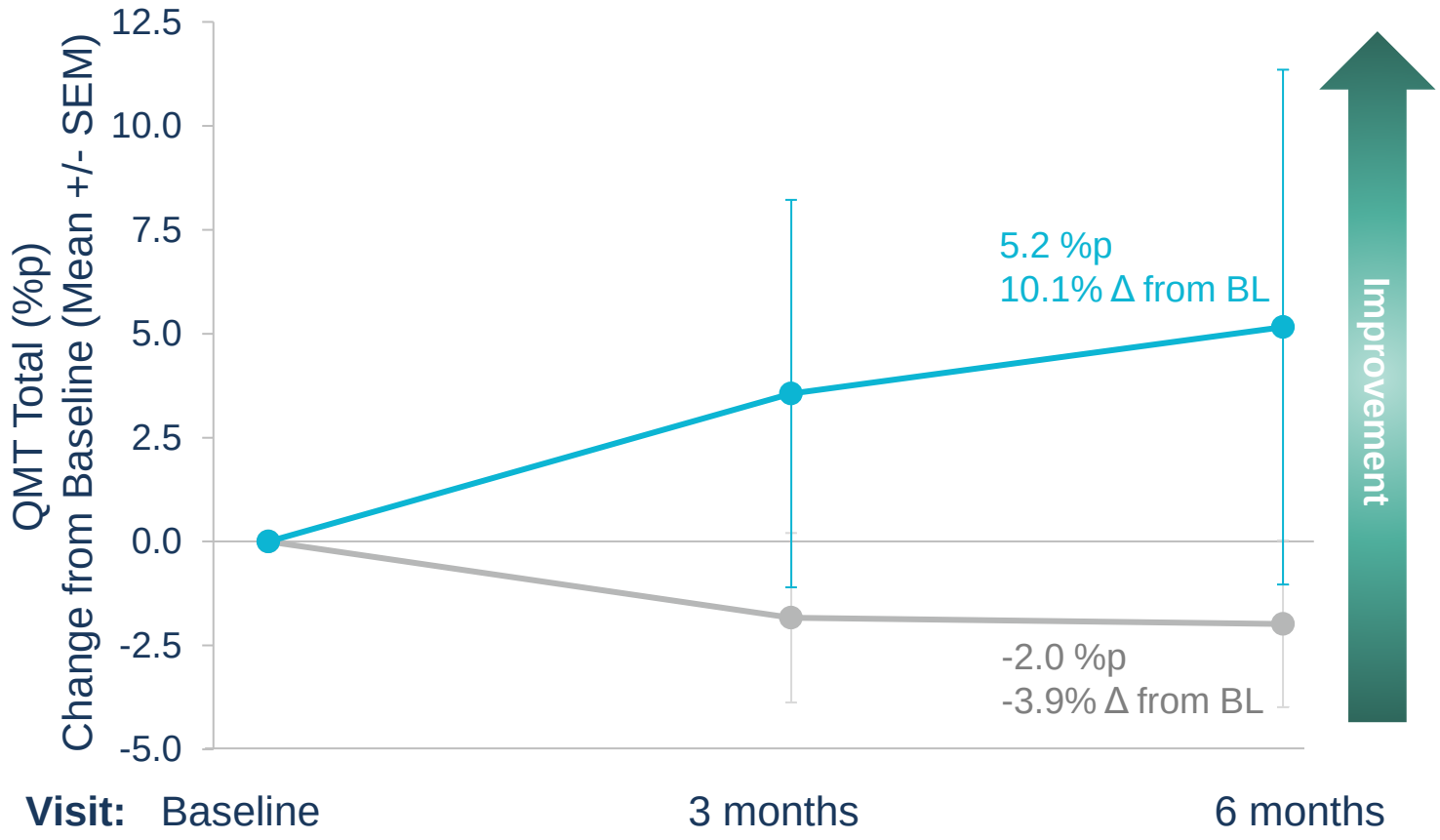
Early and Robust Improvement in Functional Myotonia



Notes: 1. vHOT Middle Finger (sec) is the average of all myotonia trials for an individual participant in ACHIEVE; BL = baseline. 3 months = 85 days; 6 months = 169 days.

Improved Muscle Strength at 6 Months

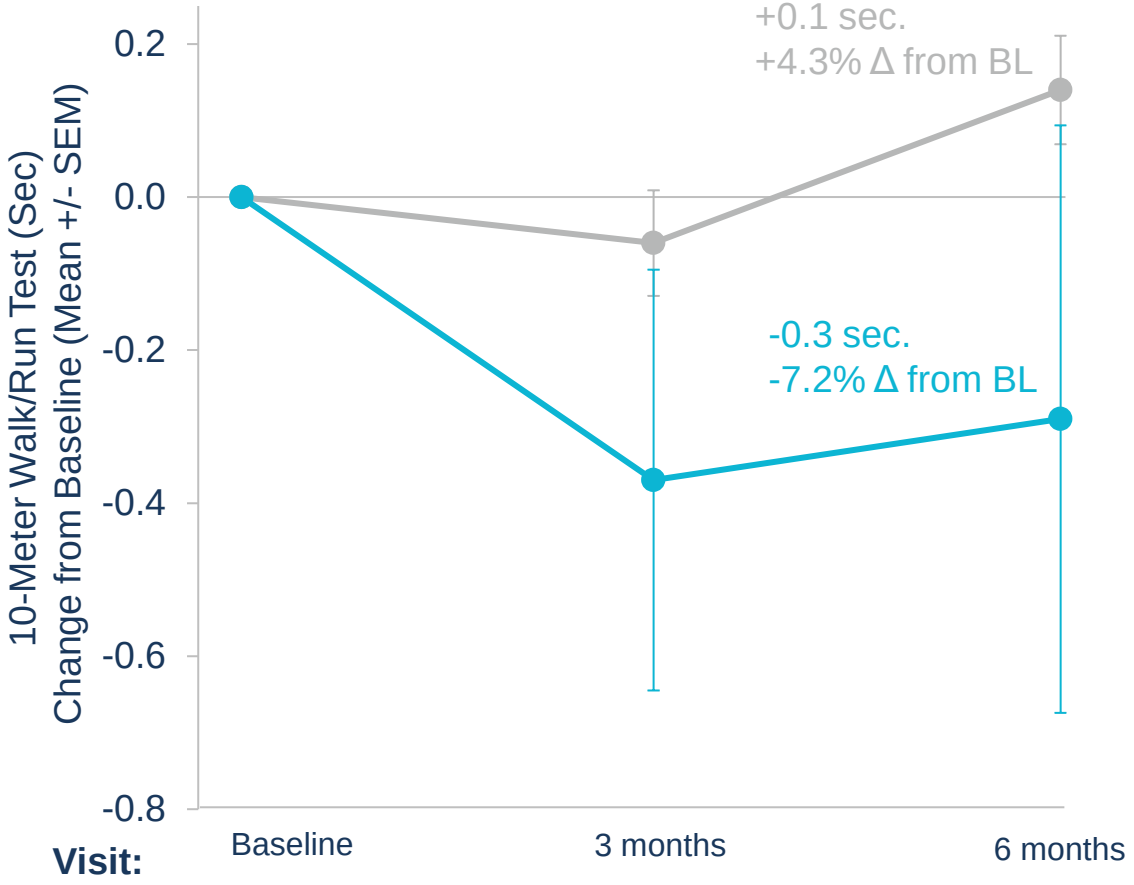
Quantitative Muscle Testing (QMT) Total Score



Notes: BL = baseline; 3 months = 85 days; 6 months = 169 days.

Early and Robust Benefit Across Multiple Timed Function Tests

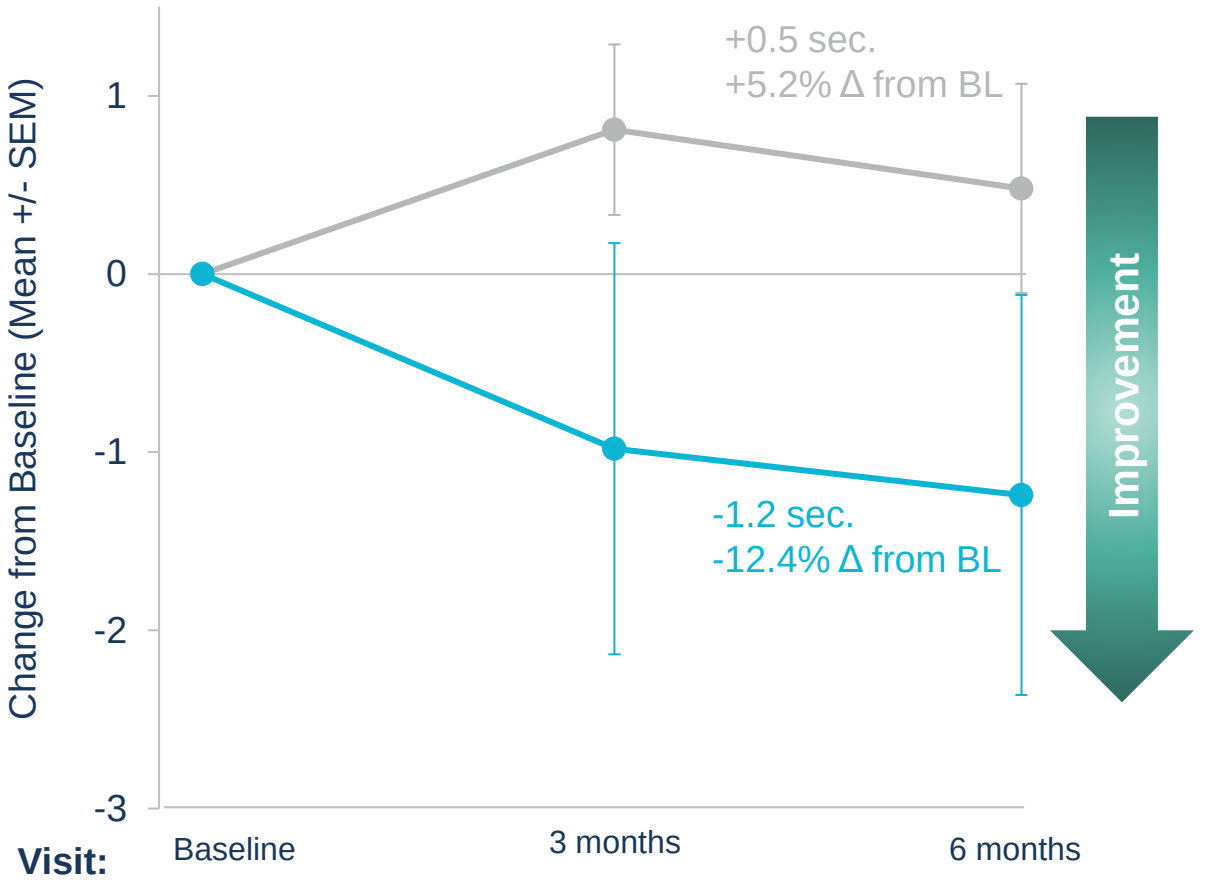
10-Meter Walk/Run Test



Cohort: Placebo (N = 14) DYNE-101 6.8 mg/kg Q8W (N = 6)

Baseline (sec), mean (SEM):	**3.3 (0.1)**	**3.9 (0.6)**

5 Times Sit to Stand

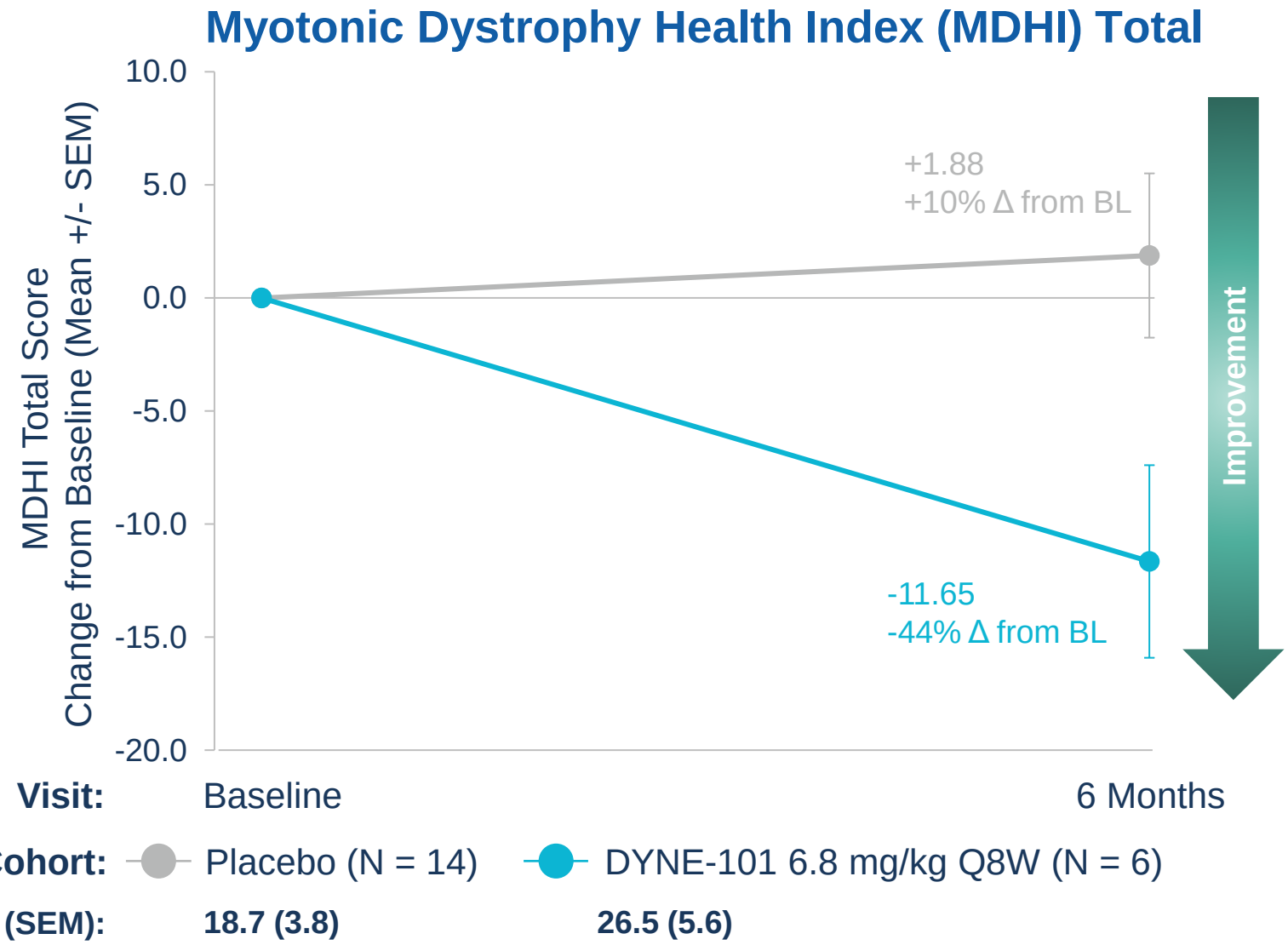


Cohort: Placebo (N = 14) DYNE-101 6.8 mg/kg Q8W (N = 6)

Baseline (sec), mean (SEM):	**9.2 (0.5)**	**10.0 (1.4)**

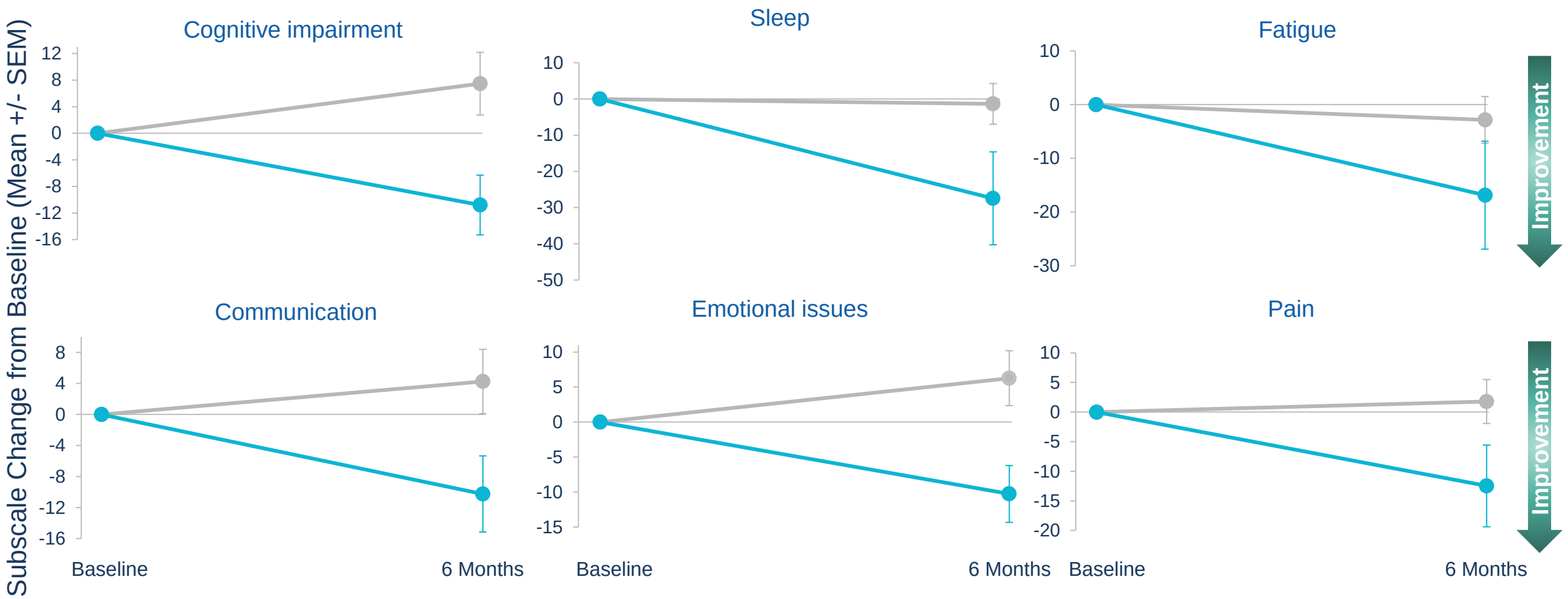
Notes: BL = baseline; 3 months = 85 days; 6 months = 169 days.

Improvement in MDHI Total Indicates Encouraging Patient Reported Outcome Trends



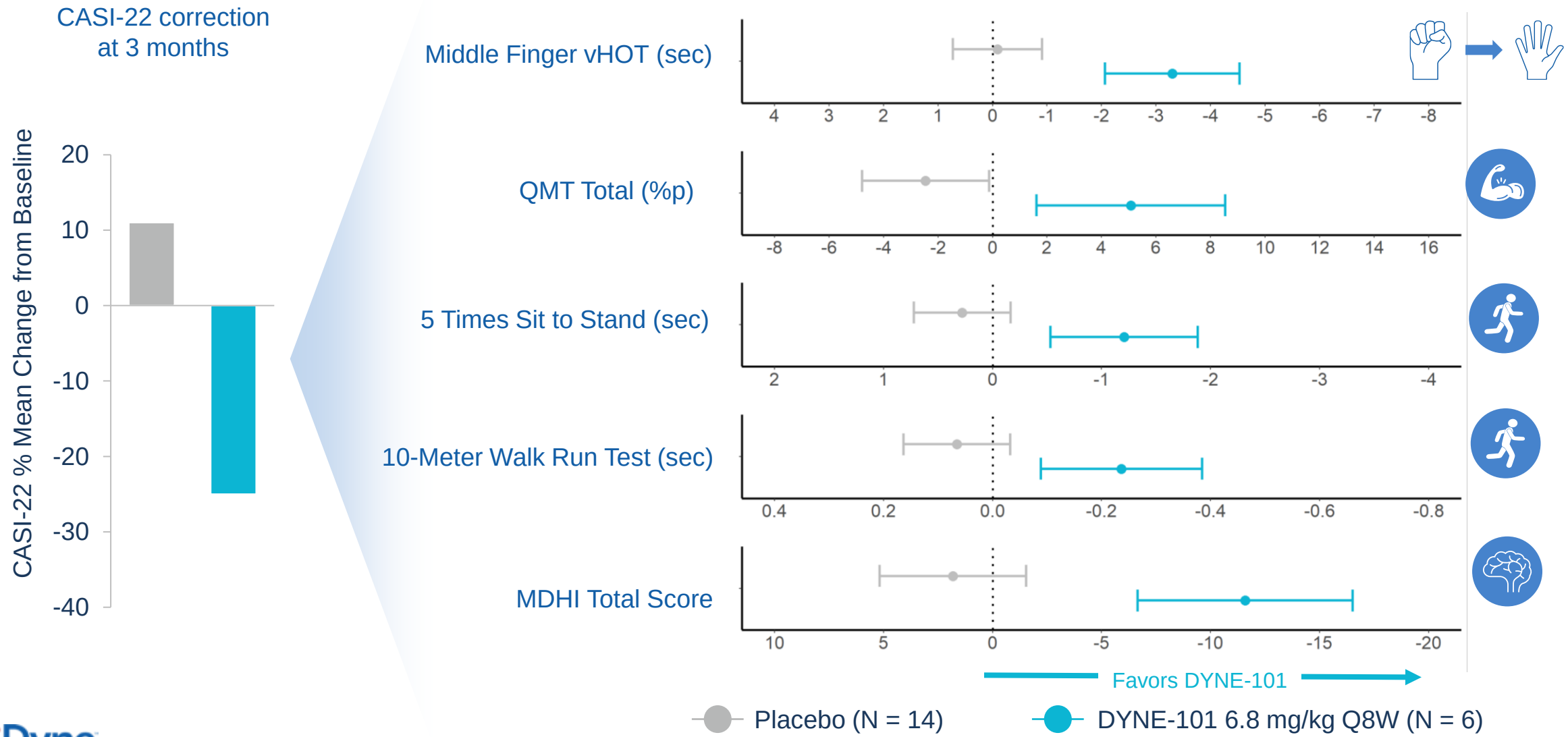
Note: Patient-reported outcomes (PRO) including Myotonic Dystrophy Health Index (MDHI) collected at baseline (BL) and 6 months (169 days).

CNS-related MDHI Subscales Show Improvement at Registrational DYNE-101 Dose



Note: Patient-reported outcomes (PRO) including Myotonic Dystrophy Health Index (MDHI) collected at baseline and 6 months (169 days).

Benefit at 6 Months Strengthened when Adjusting for Baseline Imbalances



Mixed model for repeated measures (MMRM): fixed effects: dose, visit, baseline, dose by visit interaction, baseline by visit interaction. Data: all dose groups except recovery group; excluding placebo data after 6 months; Data presented are least squares (LS) mean change from baseline ± SE; 3 months = 85 days; 6 months = 169 days.

Registrational Expansion Cohort Timeline Enables Potential Submission for U.S. Accelerated Approval in H1 2026

Accelerated Approval Path Enables Speed to Filing with Functional Benefit

ACHIEVE Registrational Expansion Cohort

N ~ 32, 3:1
6.8 mg/kg Q8W

- **Full Enrollment (mid-2025)**
- **Splicing Data (3 months)**
- **Functional Endpoints (6 months)**

Potential Submission for U.S. Accelerated Approval (H1 2026)

Potential Profile

Planned Primary Endpoint (3 months)

- Change from baseline in CASI

Planned Secondary Endpoints (6 months)

- Change from baseline in
  - vHOT (middle finger)
  - 10MWR
  - QMT
  - 5xSTS
  - MDHI Total Score

Myotonic Dystrophy Type 1 (DM1) Clinical Data Show Broad Functional Impact and Support Potential for Accelerated Approval

- **Initiating Registrational Expansion Cohort: primary endpoint of splicing correction at 3 months, supported by functional endpoints and PROs; full enrollment expected mid-2025**
- **6.8 mg/kg Q8W dose showed robust splicing correction at 3 months and broad functional improvement, starting at 3 months and continuing at 6 months**
- **Continued favorable safety profile[1]; no serious related TEAEs**

1. DYNE-101 safety data as of December 6, 2024

Building a Global DMD Franchise of Transformative Therapies

Overview

- Mutation in the *DMD* gene that encodes for dystrophin
- Onset in first few years of life
- Life expectancy ~30 years

Clinical Presentation

- Muscle weakness
- Progressive loss of function
- Loss of ambulation
- Respiratory/cardiac failure

Population

- ~12,000 - 15,000 (US)
- ~ 25,000 (Europe)

Current Approved Exon 51 Therapies Only Increased Dystrophin Production

<1%

OUR APPROACH

Potential Best-in-class Targeted Exon Skipping

Increase dystrophin expression and enable less frequent dosing to potentially **stop or reverse disease progression**

Registrational Expansion Cohort of DELIVER Rapidly Enrolling

DELIVER

Population

- Male patients with DMD with mutations amenable to exon 51 skipping therapy
- Ages 4 to 16 years
- Ambulant and non-ambulant

Primary Endpoints

- Safety and tolerability
- Change from baseline in dystrophin protein levels by Western Blot

Additional Endpoints

- Pharmacokinetics
- Change from baseline of:
  - Exon 51 skipping levels
  - Muscle tissue PDPF
  - Multiple assessments of muscle function, including NSAA score, SV95C and certain timed functional tests

Stages of DELIVER

✓ Multiple Ascending Dose (MAD): 24 weeks

- Open-Label Extension (OLE): 24 weeks
- Long-Term Extension (LTE): 192 weeks

Registrational Expansion Cohort

Note: Additional endpoints include select secondary and exploratory endpoints
PDPF: percent dystrophin-positive fibers; NSAA: North Star Ambulatory Assessment; SV95C: Stride Velocity 95th Centile.

DELIVER Baseline Participant Characteristics: By Cohort

mean (SD) or n(%)	0.7 mg/kg (N=6)	1.4 mg/kg (N=6)	2.8 mg/kg (N=6)	5 mg/kg (N=6)	10 mg/kg (N=8)	20 mg/kg (N=8)
Age (years)	10.8 (2.2)	8.0 (3.5)	10.7 (2.9)	8.3 (2.8)	6.6 (2.2)	8.1 (2.4)
BMI (kg/m^2)	19.5 (3.4)	18.6 (2.2)	22.6 (6.3)	20.9 (1.6)	18.3 (3.2)	18.6 (5.1)
Age of Symptom Onset (years)	3.7 (1.8)	4.5 (2.1)	2.8 (1.8)	3.7 (3.1)	2.8 (1.6)	2.9 (2.0)
Corticosteroid dosing regimen (n (%))[1] Daily Other	 4 (66.7%) 2 (33.3%)	 4 (66.7%) 3 (50.0%)	 5 (83.3%) 2 (33.3%)	 6 (100.0%) 0	 8 (100.0%) 0	 8 (100.0%) 2 (25.0%)
Prior DMD Therapy (n (%)) Eteplirsen Other	 4 (66.7%) 2 (33.3%)	 2 (33.3%) 1 (16.7%)	 5 (83.3%) 0	 1 (16.7%) 0	 1 (12.5%) 1 (12.5%)	 0 2 (25.0%)
NSAA Total Score	22.2 (7.2)	22.8 (10.5)	20.3 (9.0)	21.0 (7.0)	25.3 (6.4)	15.6 (5.1)
10 Meter Run/Walk (sec)	6.1 (1.5)	6.3 (5.2)	6.9 (3.6)	5.1 (1.5)	4.6 (1.9)	7.7 (3.8)
Time Rise From Floor (sec)	8.5 (4.0)	3.1 (0.3)	6.9 (4.9)	5.0 (2.6)	6.3 (5.6)	5.1 (2.3)
Stride Velocity 95th Percentile (m/sec)	N/A	N/A	N/A	N/A	1.9 (0.5)	1.4 (0.5)

Note: Q4W and placebo arms are reported together for baseline characteristics. N/A: not applicable as data not collected.
1. Historical corticosteroid regimen based on medical history; a participant can be counted in multiple categories.

DYNE-251 Safety Profile Is Favorable

Summary of Treatment Emergent Adverse Events (TEAEs)[1]

TEAE Category	Participants with ≥1 TEAE – n (%)								
	0.7mg/kg Q4W N=6	1.4 mg/kg Q4W N=6	2.8 mg/kg Q4W N=6	5 mg/kg Q4W N=6	10 mg/kg Q4W N=8	20 mg/kg Q4W N=8	40 mg/kg Q8W N=8	40 mg/kg Q4W N=6	Overall[1] N=54
Any TEAE	6 (100%)	6 (100%)	6 (100%)	6 (100%)	7 (88%)	8 (100%)	7 (88%)	5 (83%)	51 (94%)
Any related TEAE	3 (50%)	3 (50%)	0	6 (100%)	3 (38%)	4 (50%)	2 (25%)	3 (50%)	24 (44%)
Any serious TEAE	0	0	1 (17%)	0	0	1 (13%)	2 (25%)	2 (33%)	6 (11%)
Any serious related TEAE	0	0	0	0	0	0	0	2 (33%)	2 (4%)
Any TEAE leading to withdrawal from study	0	0	0	0	0	0	0	0	0
Any TEAE leading to death	0	0	0	0	0	0	0	0	0

Most TEAEs Were Mild or Moderate in Intensity[1]

- 3 serious TEAEs potentially related to study drug in two participants
  - Acute kidney injury (1); thrombocytopenia (1)[2]
  - Pancytopenia (1)[3]
- 6 serious TEAEs unrelated to study drug
  - Dehydration due to gastroenteritis (1)
  - Femoral neck fracture (1); gastric volvulus (1)[4]
  - Tibia fracture (1)
  - Febrile convulsion (1); pyrexia (1)[5]
- Most common TEAEs (≥20% participant incidence)[6]
  - Pyrexia (44%)
  - Fall; vomiting (each 33%)
  - Headache (32%)
  - Nasopharyngitis (28%)
  - Cough; infusion-related reaction[7] (each 20%)

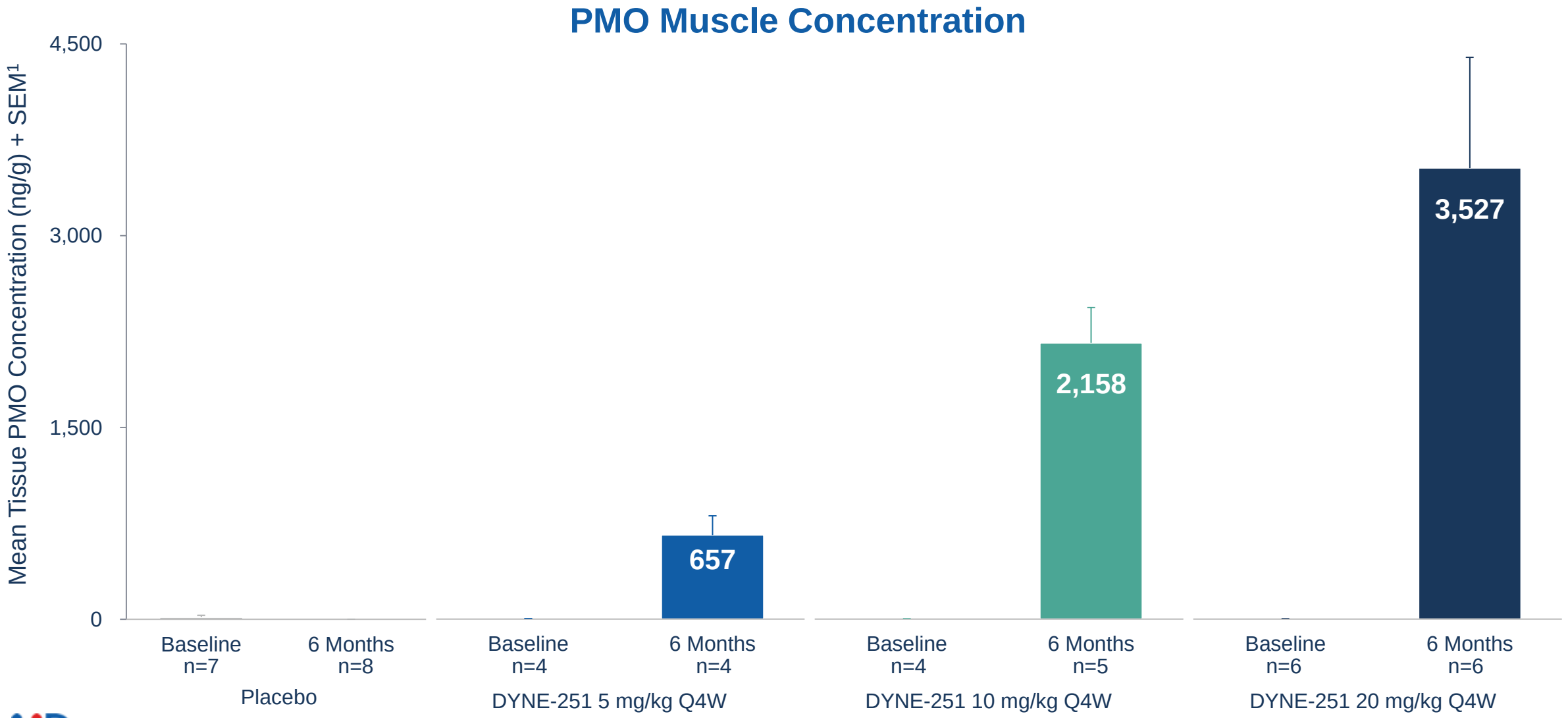
Additional Safety Data

- Other than two participants with serious TEAEs in 40 mg/kg Q4W cohort:
  - No participants have demonstrated persistent related anemia or thrombocytopenia
  - No participants have demonstrated kidney injury
- No participants have demonstrated clinically meaningful changes in electrolytes, including magnesium

837 Doses Administered to Date Representing Over 65 Patient-Years of Follow-Up[1]

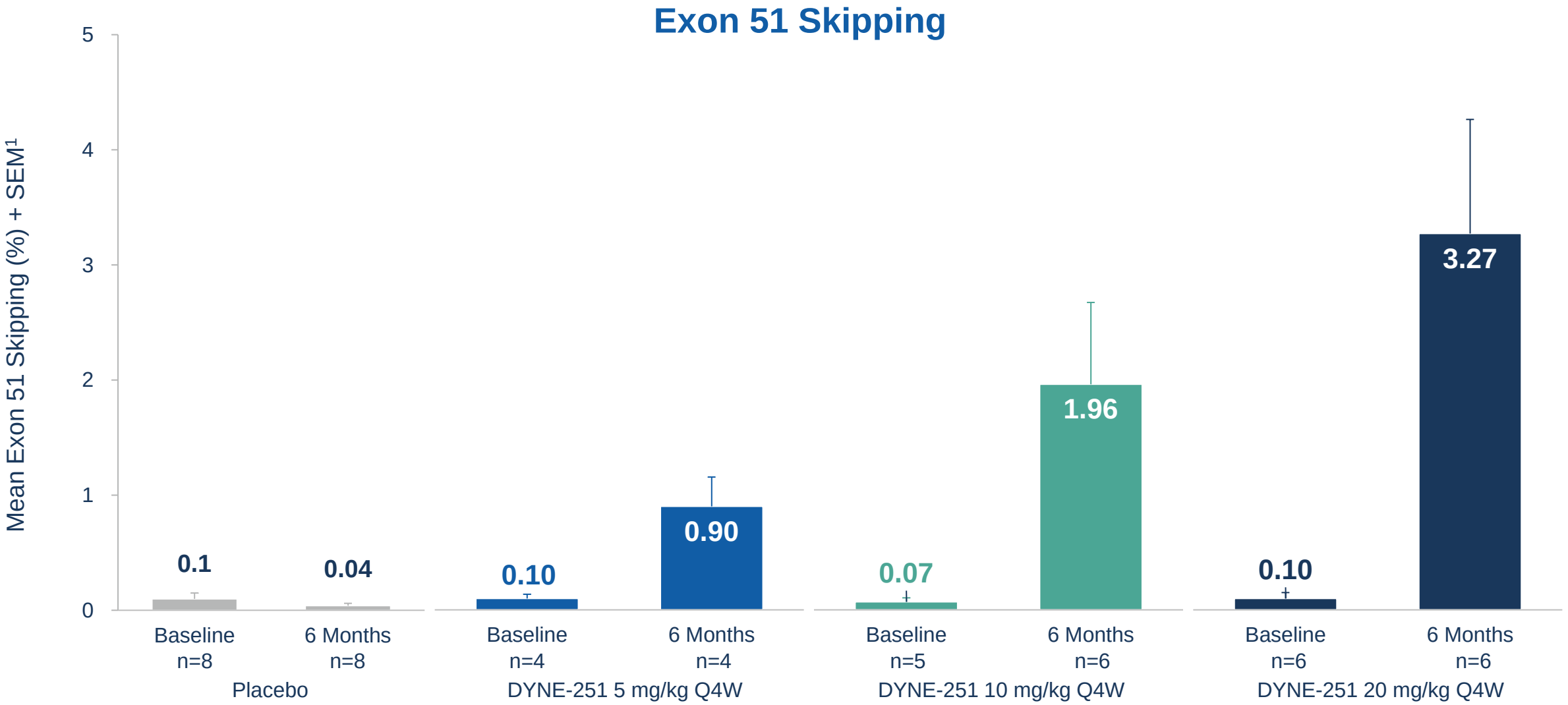
1. Data as of November 21, 2024; 2. Events have same day of onset in a single participant with a nonserious related TEAE of anemia in the context of fever, hemolysis, diarrhea and positive blood in stool; together these events are consistent with hemolytic uremic syndrome (HUS) with a possible infectious etiology; 3. Participant has a history of hemolytic anemia of unidentified etiology; presented with fever and tonsilitis; symptoms resolved without therapeutic intervention; 4. Events occurred in same participant at different times; 5. Events occurred in same participant at different times; 6. All cohorts combined; preferred terms are reported; 7. All infusion-related reactions have been mild or moderate in intensity; dosing has continued in all participants who experienced infusion-related reactions.

DYNE-251 Drove Dose Dependent Delivery of PMO to Muscle



1. DELIVER biopsy taken approximately 28 days after most recent dose; 6 months = Week 25 for DELIVER.

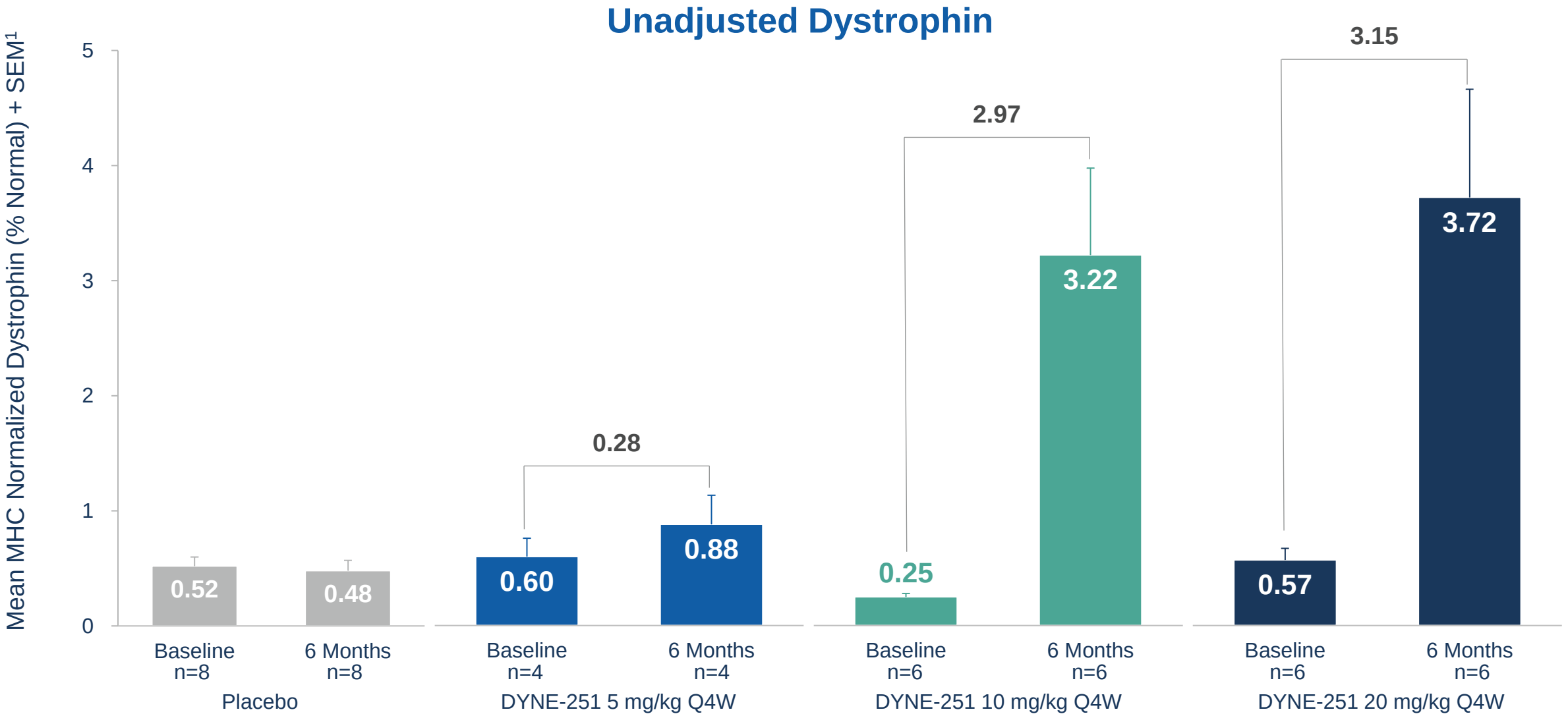
DYNE-251 Demonstrated Dose-Dependent Exon Skipping



1. DELIVER biopsy taken approximately 28 days after most recent dose; 6 months = Week 25 for DELIVER.

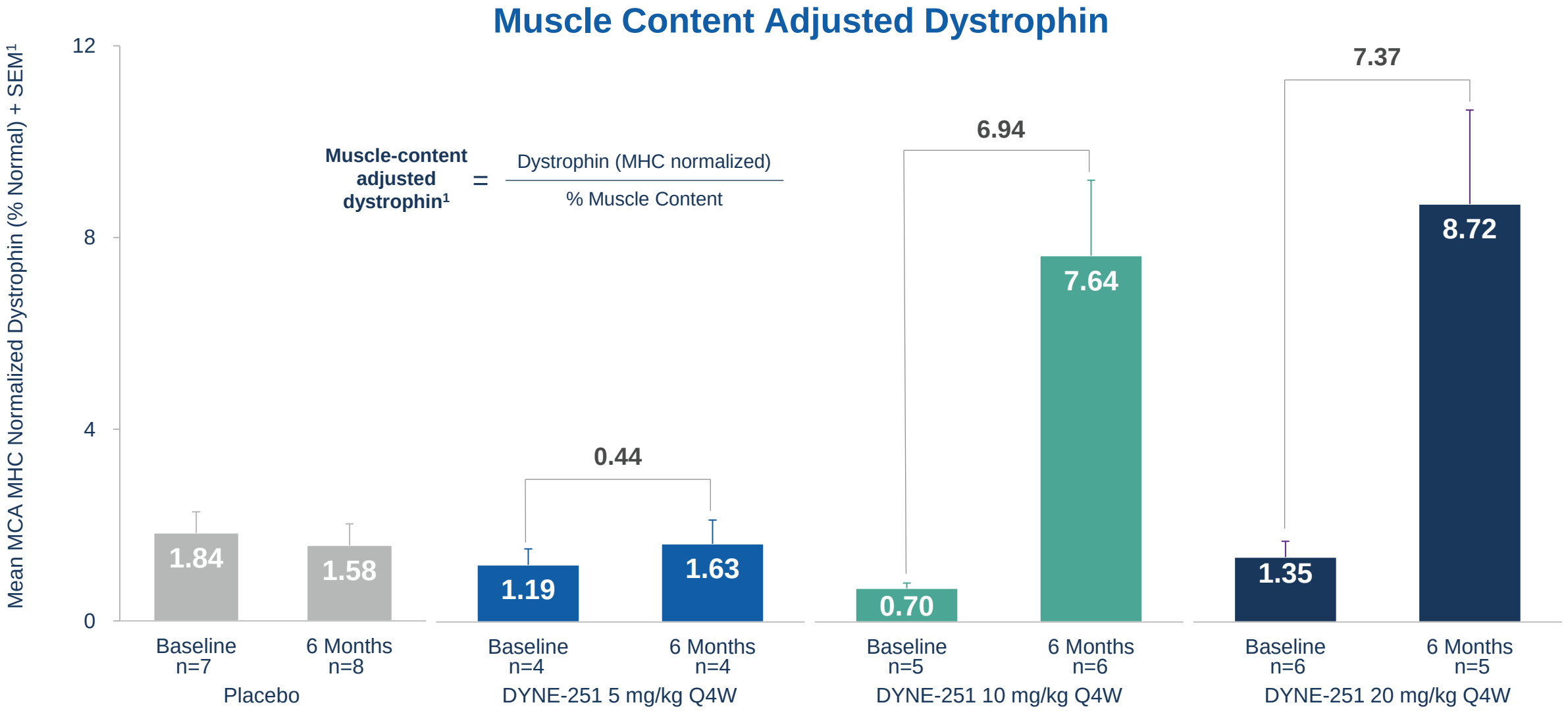
Higher Doses of DYNE-251 Continued to Drive Robust Dystrophin Expression

DYNE-251 Showed 3.7% Unadjusted Dystrophin at 6 Months



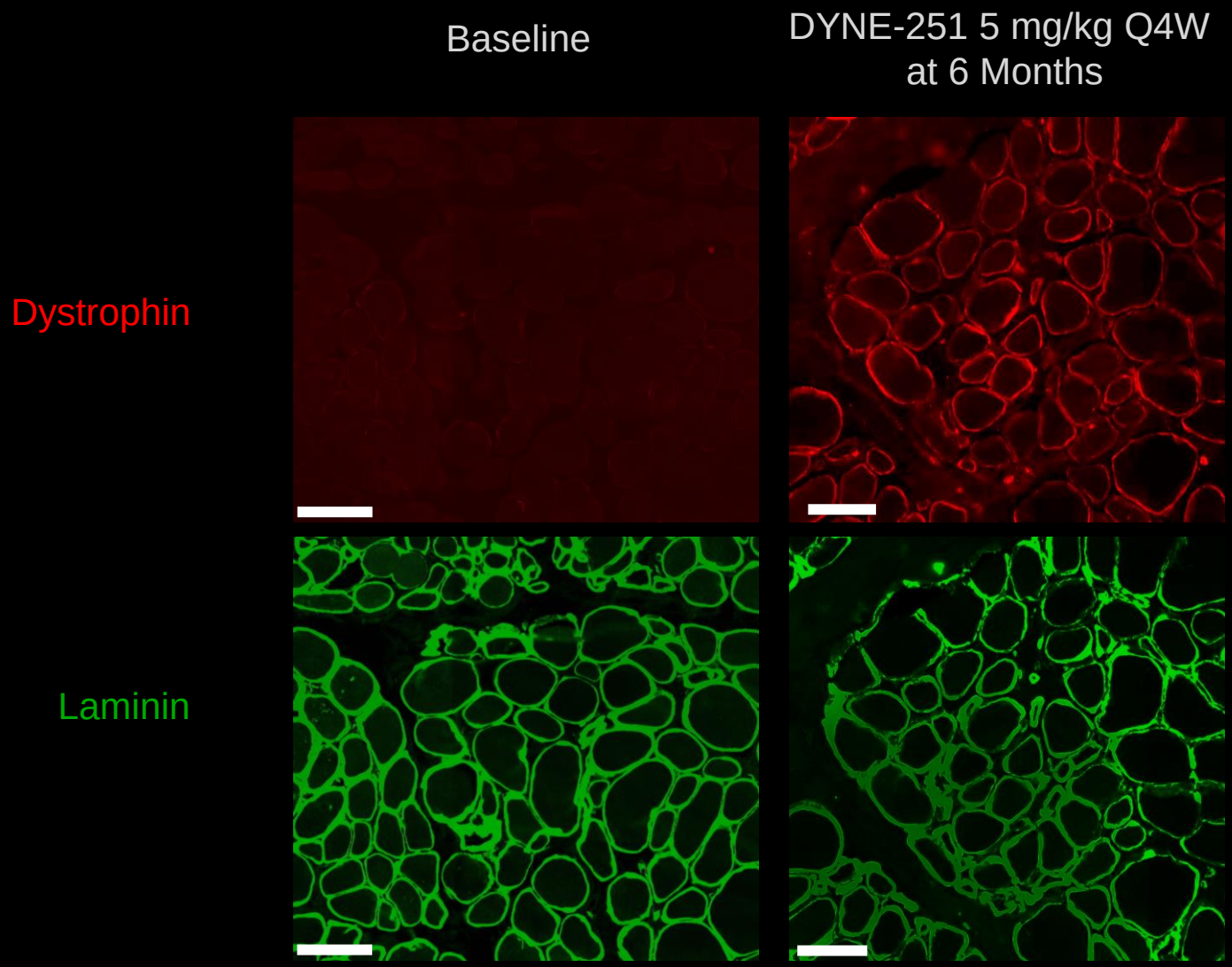
1. DELIVER biopsy taken approximately 28 days after most recent dose; 6 months = Week 25 for DELIVER.

DYNE-251 Positioned as a Potentially Best-in-Class Next Generation Exon Skipper, Achieving 8.7% Muscle Content Adjusted Dystrophin at 6 Months



1. DELIVER biopsy taken approximately 28 days after most recent dose; 6 months = Week 25 for DELIVER.

PDPF: Clear Improvement in Dystrophin Localization to Sarcolemma

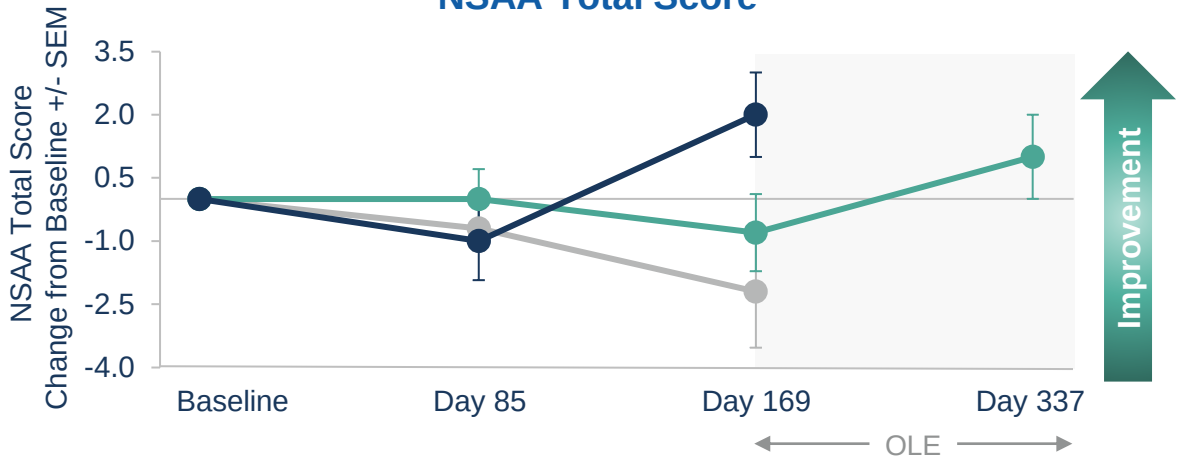


Notes: DELIVER biopsy taken approximately 28 days after most recent dose. Notes: scale bar is 100 µm.

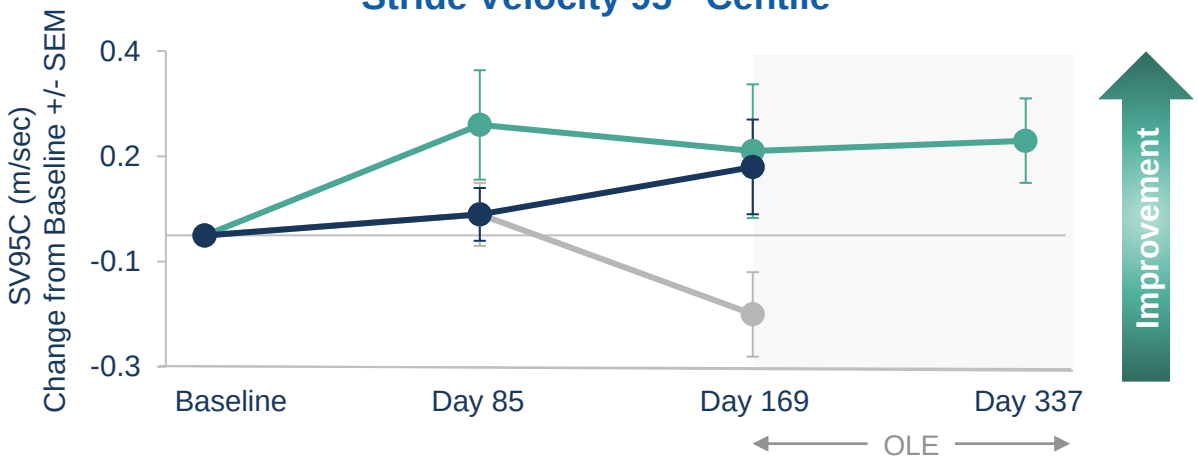
Improvements Across Multiple Functional Endpoints in Multiple Cohorts

Baseline Values Inform Interpretation of Data; Ongoing Exploration of Longer Timepoints

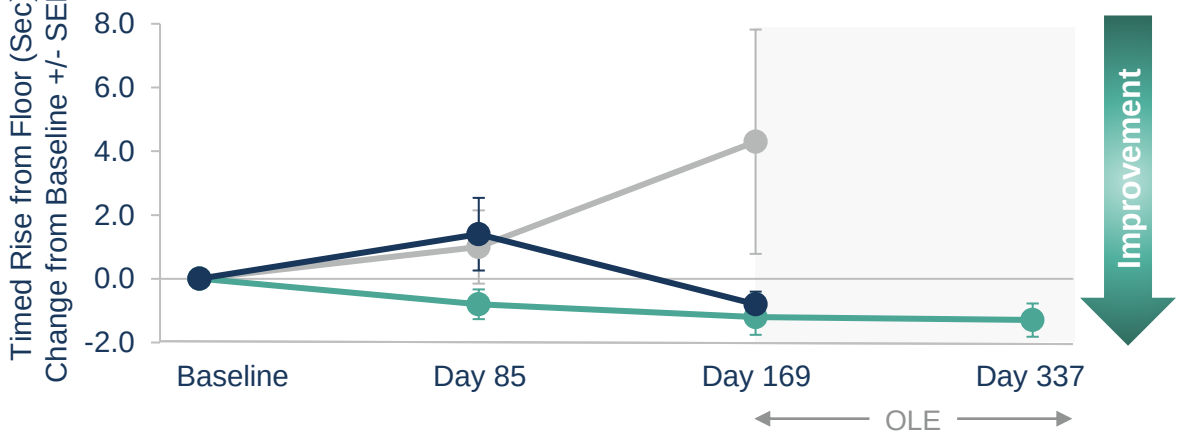
NSAA Total Score



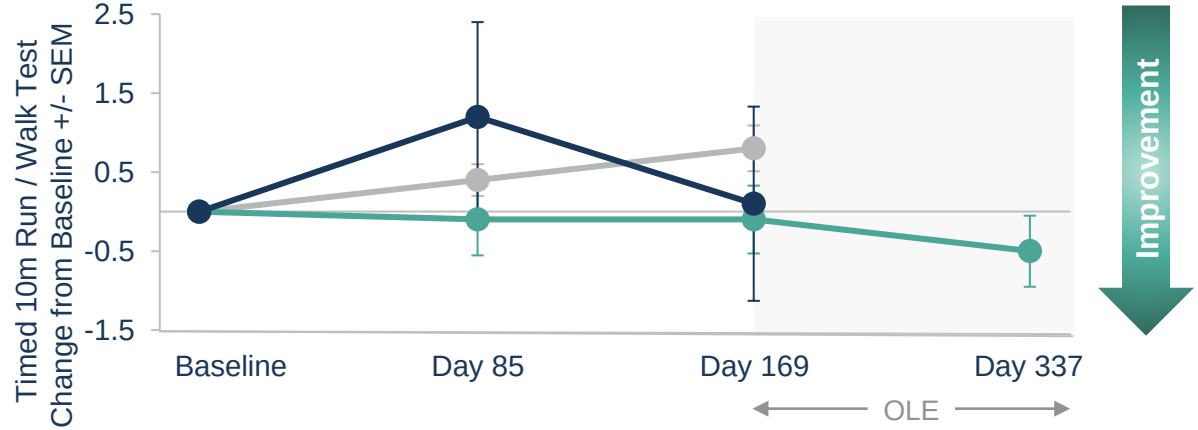
Stride Velocity 95th Centile



Timed Rise from Floor



Timed 10-Meter Walk / Run Test



Placebo (n=6 for SV95C and n=14 for other endpoints)

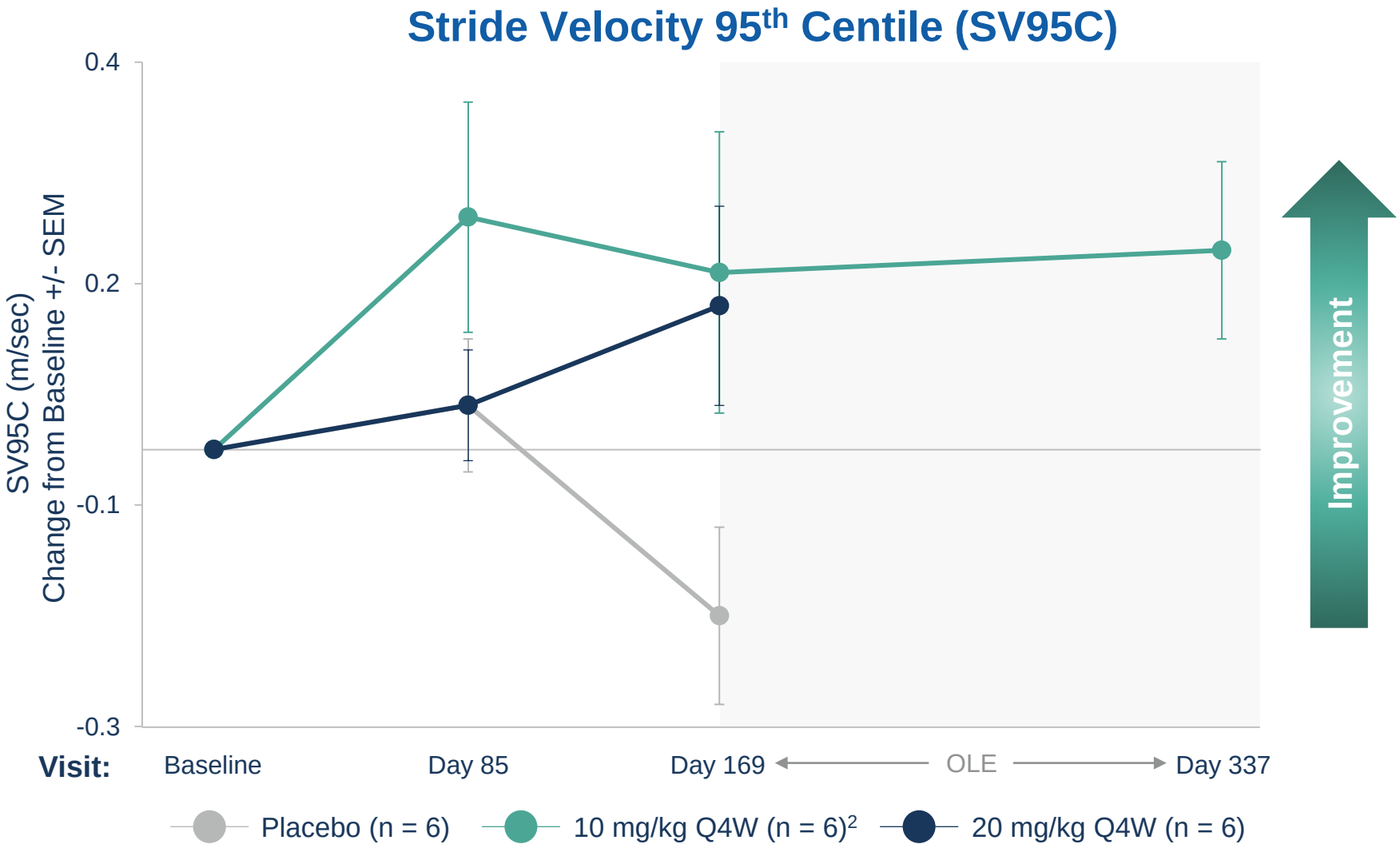
DYNE-251 10 mg/kg Q4W (n = 6 for all endpoints) [1]

DYNE-251 20 mg/kg Q4W (n = 6 for all endpoints)

1. During the OLE, all participants in 10 mg/kg cohort were dose escalated to 20 mg/kg Q4W regimen.

DYNE-251 Drove Clinically Meaningful Improvements in Stride Velocity 95th Centile

SV95C is a Qualified Primary Endpoint for Duchenne Trials in Europe and Leveraged Across Global Trials

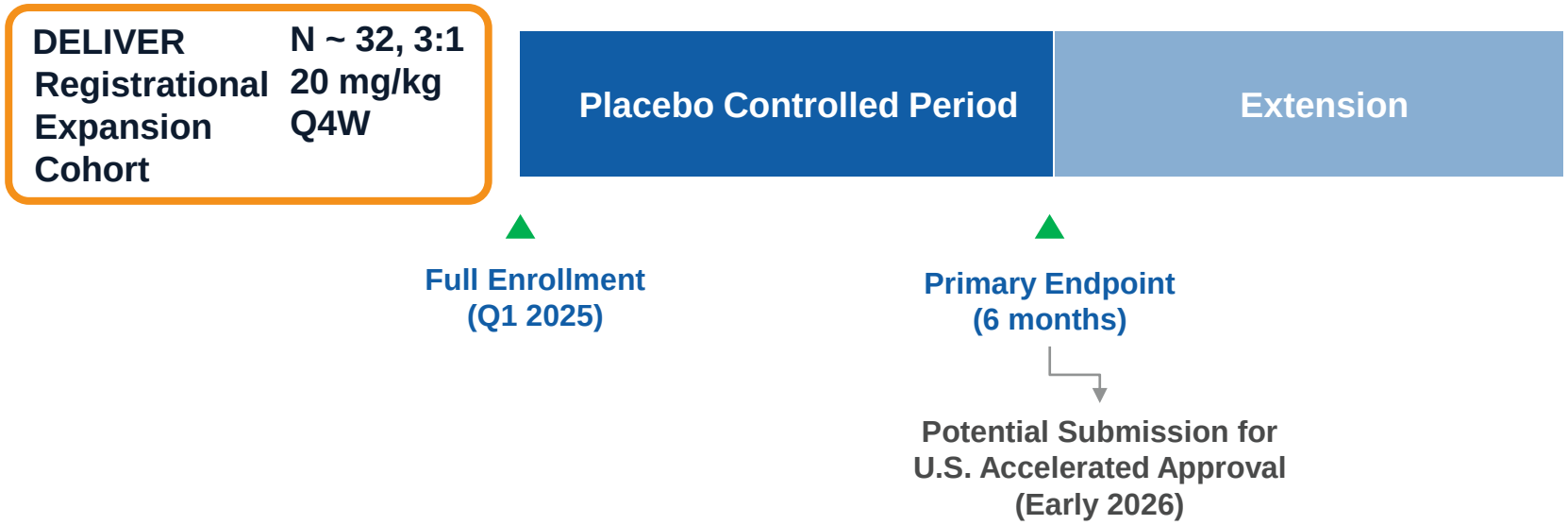


- SV95C is a digital objective endpoint of ambulatory performance in patients' normal daily environment
- Patients in DELIVER wore the device on each ankle for 3 weeks prior to the clinic visits
- The change from baseline met the published MCID by the EMA[1]

1. Minimal clinically important difference (MCID) as defined by EMA in its qualification opinion for SV95C as primary endpoint in studies in ambulatory DMD studies. 2. During the OLE, all participants in 10 mg/kg cohort were dose escalated to 20 mg/kg Q4W regimen.

Registrational Expansion Cohort Timeline Enables Potential Submission for U.S. Accelerated Approval in Early 2026

Accelerated Approval Path Enables Speed to Filing



Potential Profile

Primary Endpoint (6 months)

- Change from baseline in dystrophin protein levels by Western Blot

Additional Endpoints

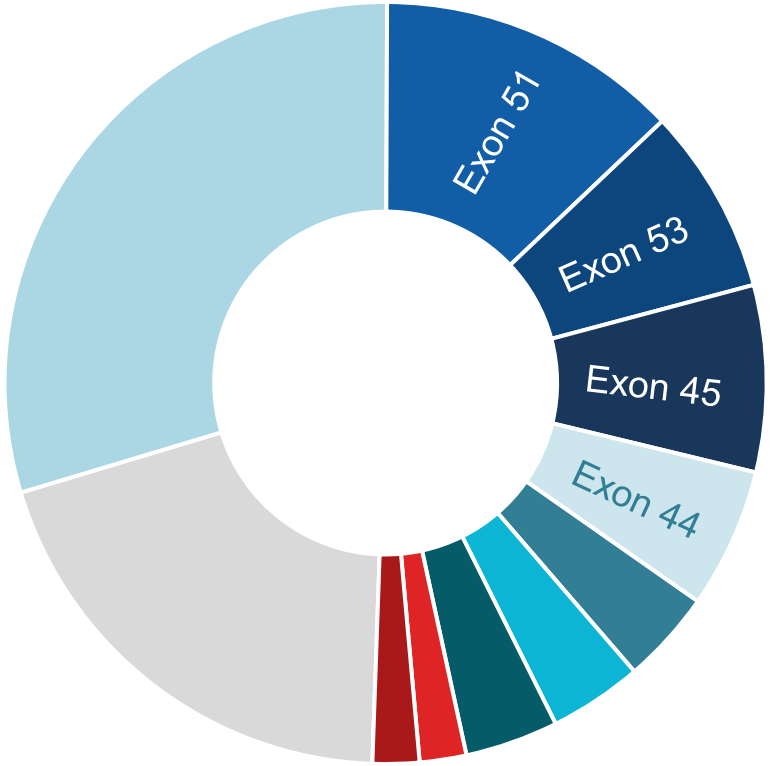
- Change from baseline of:
  - Exon 51 skipping levels
  - Muscle tissue PDPF
- NSAA
- SV95C

Opportunity to Build a Global DMD Franchise: Leading with DYNE-251, Payloads Identified for Exons 53, 45, 44

Approximately

80% of patients

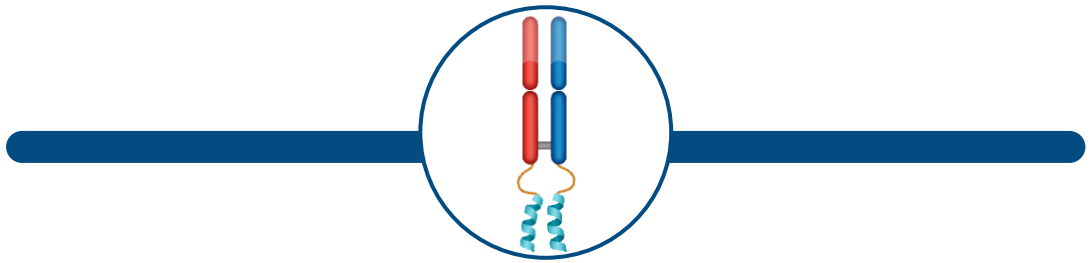
have genotypes amenable to exon skipping



- Exon 51 – **13%**
- Exon 53 – **8%**
- Exon 45 – **8%**
- Exon 44 – **6%**
- Exon 50 – **4%**
- Exon 52 – **4%**
- Exon 43 – **4%**
- Exon 55 – **2%**
- Exon 8 – **2%**
- Other exon skips – **~30%**
- May not be amenable to exon skipping – **~20%**

FORCE Positions Dyne With Potential Leading Role in Evolving DMD Therapeutic Landscape

FORCE



Potency	✓ Targeted muscle delivery, near full-length dystrophin
Durability	✓ Durable target engagement
Dosing	✓ Re-dosable, titratable
Tolerability	✓ Favorable safety profile
Manufacturing	✓ Well-established, scalable

- Muscle delivery is the challenge
- Clinical data to date validates FORCE's targeted delivery to muscle
- Non-targeted delivery modalities face significant challenges, including an acceptable therapeutic index
- SMA landscape strong analogue to DMD with ZOLGENSMA (gene therapy) and SPINRAZA (oligo) playing an important role in evolving standard of care

Advancing DYNE-251 Towards Potentially Registrational Data Set

- ✓ Unprecedented level of dystrophin generated, with 3.7% unadjusted and 8.7% muscle content adjusted dystrophin
- ✓ Improvements in multiple functional outcomes, including SV95C, an approvable endpoint in Europe, in multiple cohorts
- ✓ Favorable safety profile to date[1]; enrolling registrational cohort at 20 mg/kg Q4W
- ✓ Supports further development of DMD global franchise

Enrolling registrational cohort based on regulatory interactions and strength of data

1. Safety data as of November 21, 2024.

Building Momentum Toward Potential Launches in 2027

	2024	2025	2026
DYNE-101 for DM1	✓ MAD Complete ✓ Registrational dose selected	Fully enroll Registrational Expansion Cohort (mid-2025)	Registrational Expansion Cohort readout (H1 2026) Submission for U.S. Accelerated Approval (H1 2026)
DYNE-251 for Exon 51 DMD	✓ Registrational Expansion Cohort initiated	Registrational Expansion Cohort readout (late 2025)	Submission for U.S. Accelerated Approval (early 2026)

FSHD Program

Overview

- Aberrant expression of DUX4
- Onset in teen years or young adulthood
- Normal life expectancy

Clinical Presentation

- Progressive wasting and skeletal muscle loss
- Significant physical limitations

Population

- ~16,000 - 38,000 (US)
- ~35,000 (Europe)

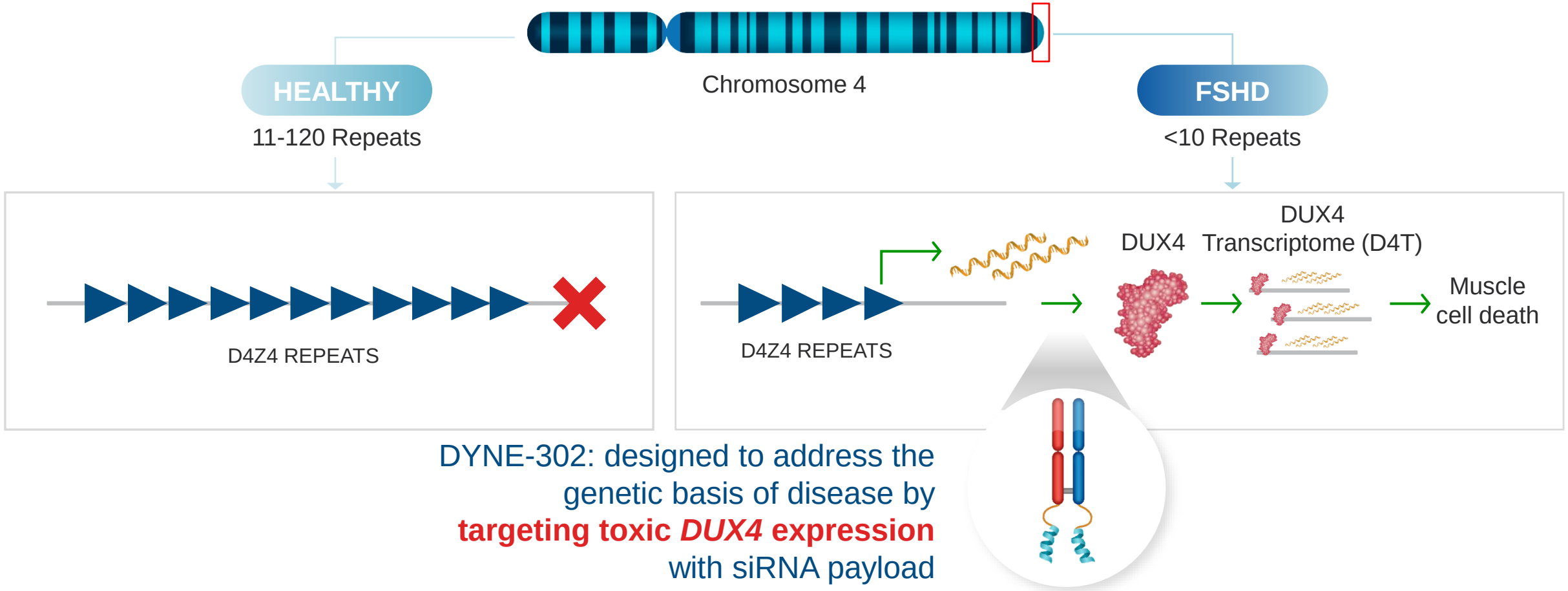
NO approved therapies

OUR APPROACH

Disease-Modifying DUX4 Knockdown

Targeting toxic *DUX4* mRNA expression to potentially **stop or reverse disease progression**

DYNE-302 Targets the Genetic Basis of FSHD

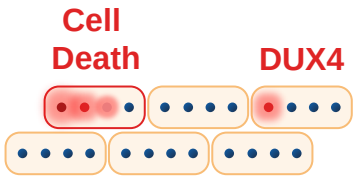


DYNE-302: designed to address the genetic basis of disease by **targeting toxic *DUX4* expression** with siRNA payload

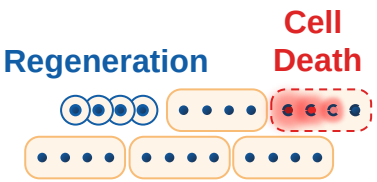
The hTfR1/iFLExD Mouse Model Recapitulates Multiple Aspects of Human FSHD

FSHD

Sporadic DUX4 activation



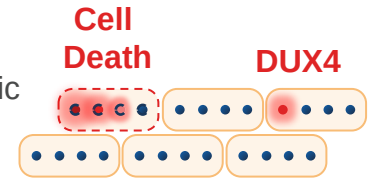
Years



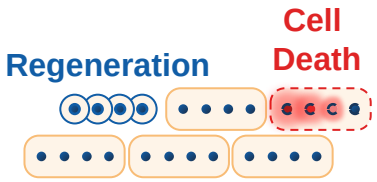
Slowly progressing myopathy and functional decline

hTfR1/iFLExD (Uninduced)

Chronic, sporadic DUX4 activation



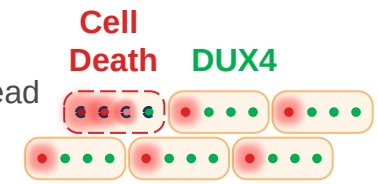
Weeks



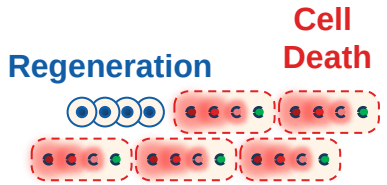
Slowly progressing myopathy without functional decline

hTfR1/iFLExD (Induced)

Acute, widespread DUX4 activation



Days

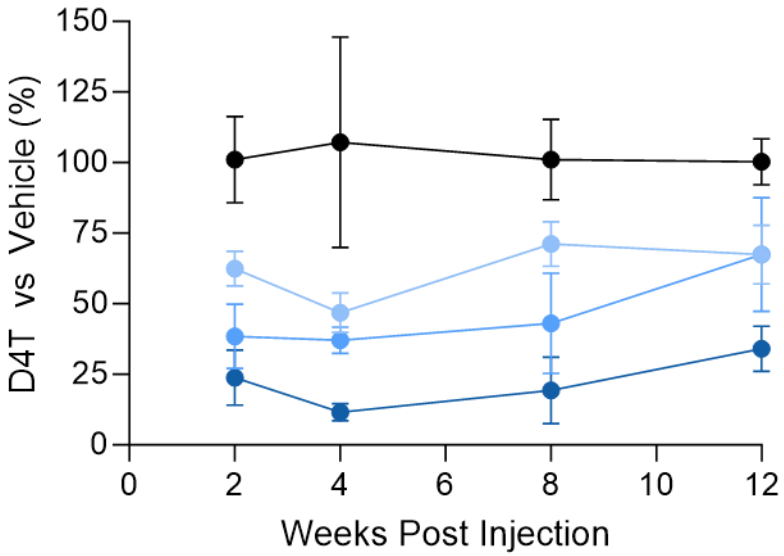


Rapidly progressing myopathy with functional decline

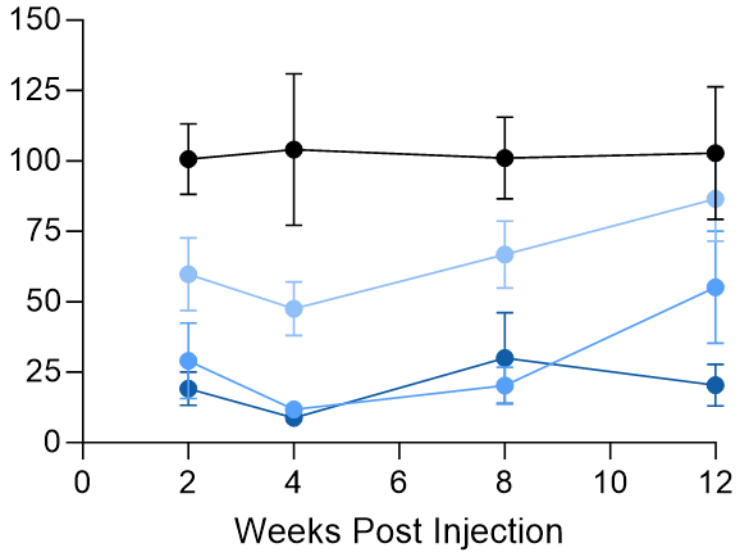
Notes: Jones et al. (2020) DOI: 1186/s13395-020-00227-4

Single Dose of DYNE-302 Achieved Robust, Durable, and Dose-Dependent D4T Knockdown in Skeletal Muscle of hTfR1/iFLExD FSHD Mice

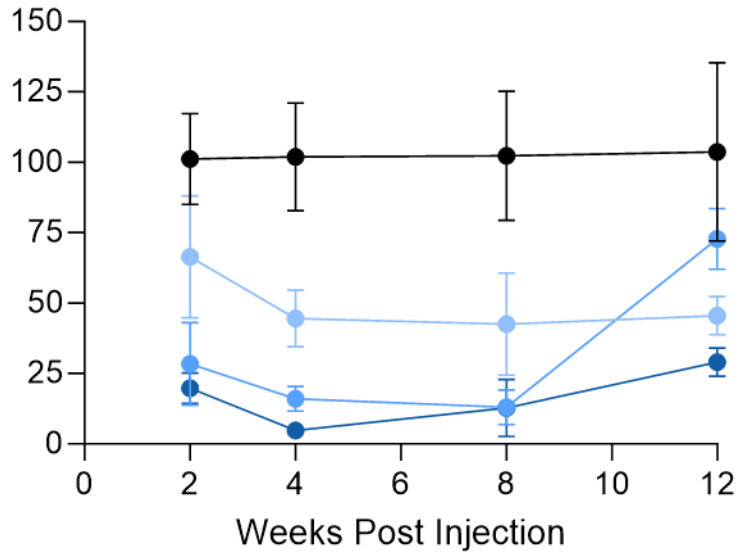
Quadriceps



Gastrocnemius



Tibialis Anterior



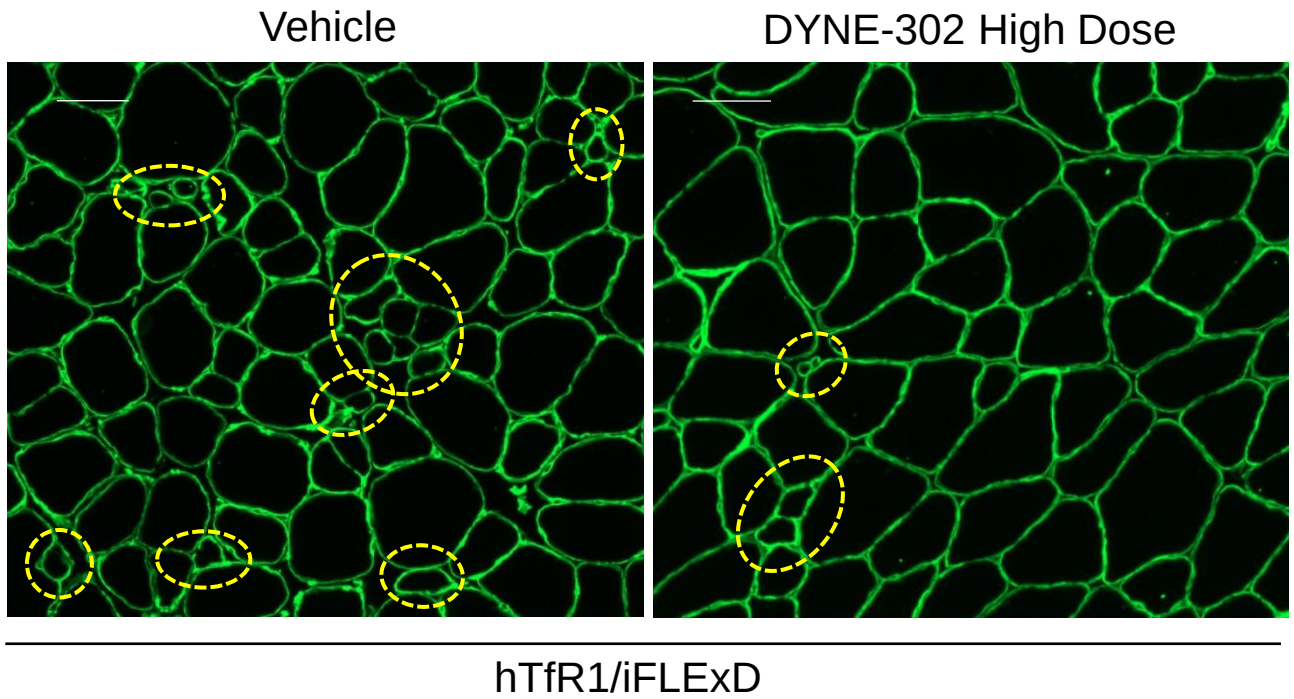
Vehicle | DYNE-302 Low Dose | DYNE-302 Medium Dose | DYNE-302 High Dose

DYNE-302 demonstrates potential for infrequent dosing, out to Q12W

Notes: Uninduced hTfR1/iFLExD mice dosed with vehicle or DYNE-302 on day 0, analyzed at indicated weeks. Data are means ± SD; n = 4 - 12. D4T is an average of mouse *Wfdc3*, *Sord*, and *Serpinb6c* mRNA markers.

Single Dose of DYNE-302 Corrected Muscle Pathology in Quadriceps of the Uninduced hTfR1/iFLExD FSHD Model at 12 Weeks

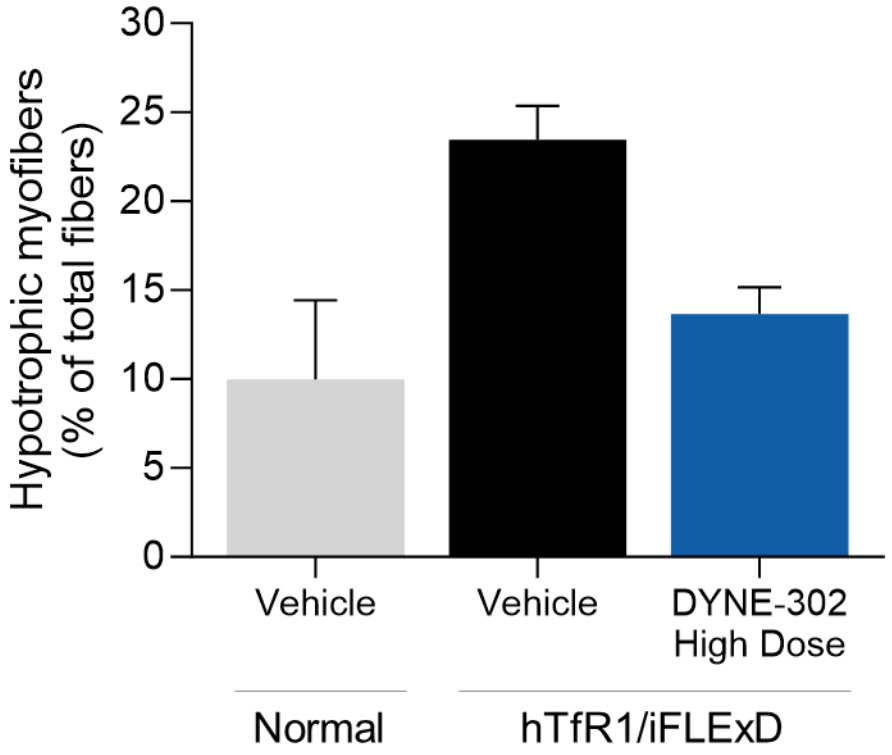
DYNE-302 reduces hypotrophic myofibers



Laminin

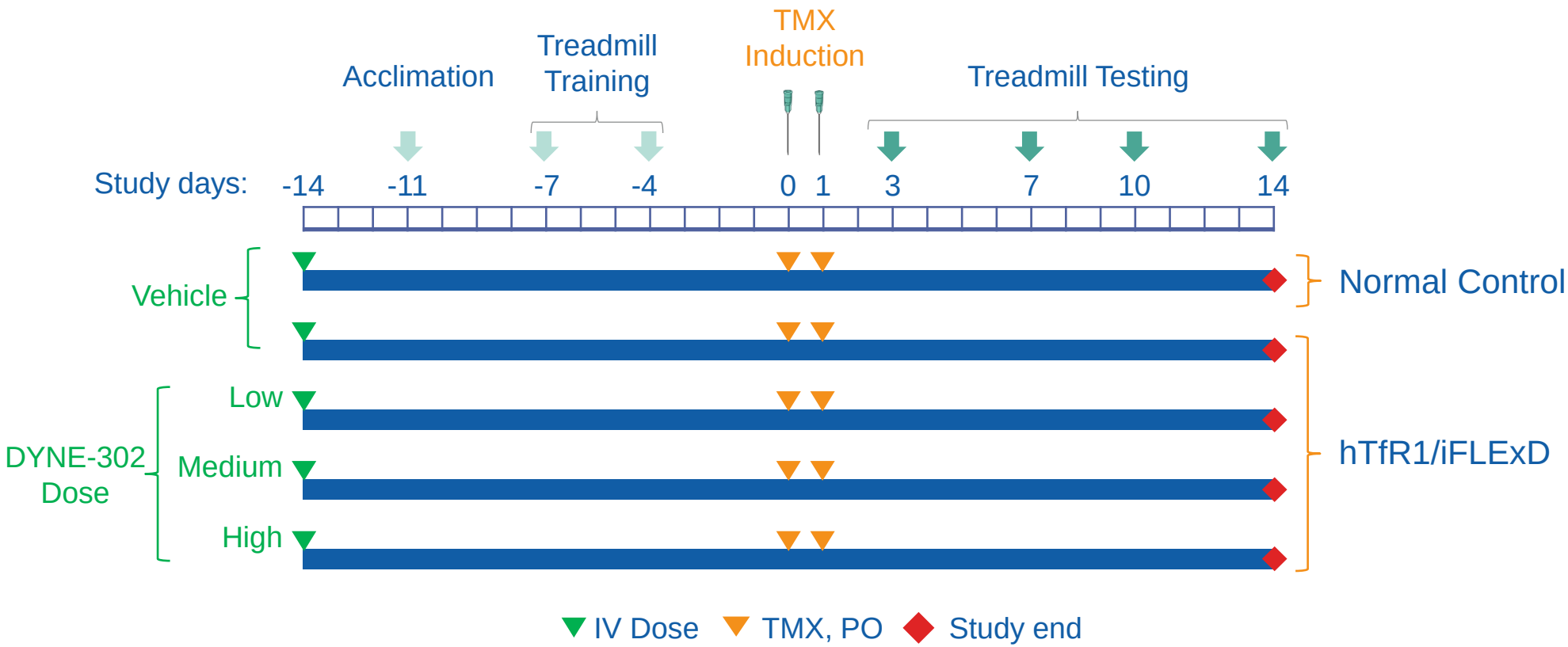
Fiber splitting (hypotrophic myofibers)

Quantification of hypotrophic myofiber reduction



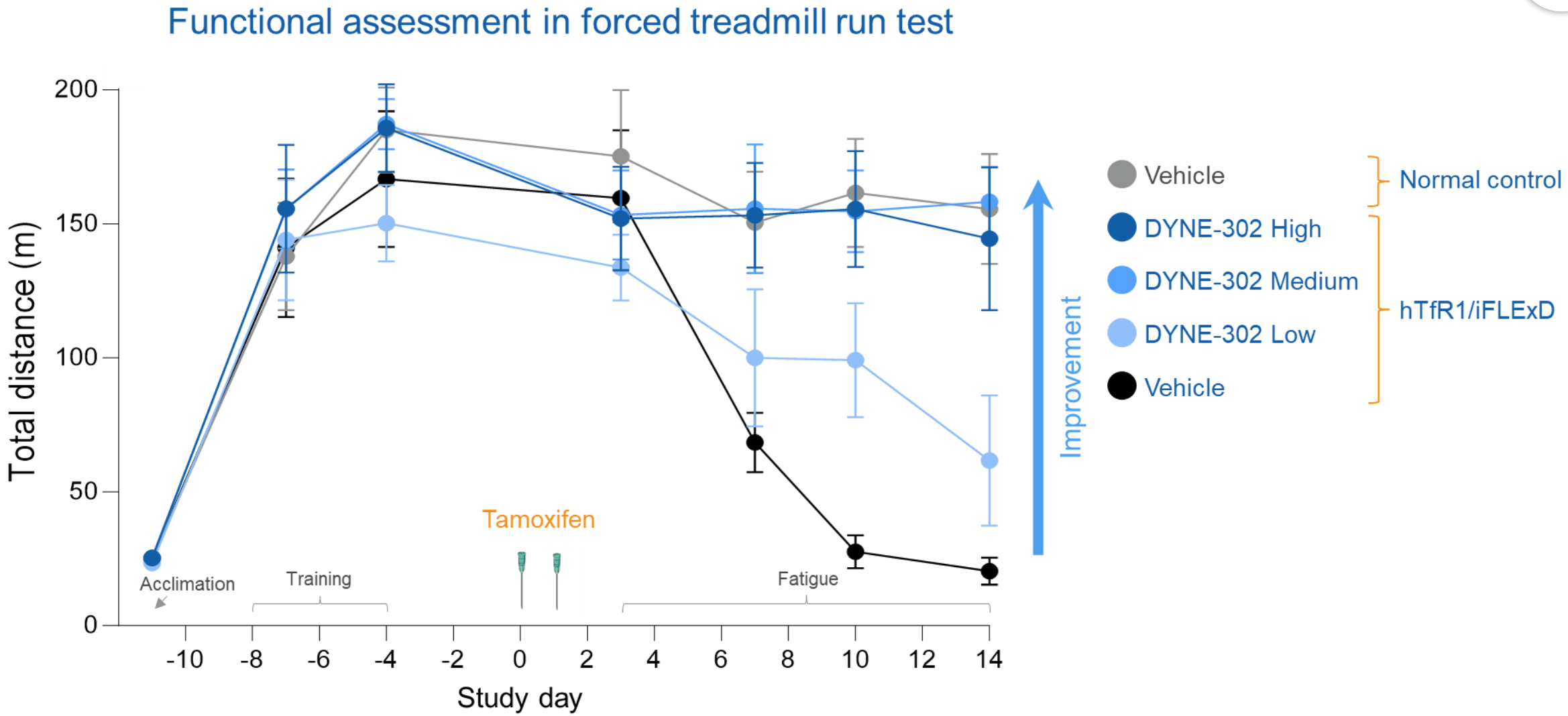
Notes: Uninduced hTfR1/iFLExD mice dosed with vehicle or DYNE-302 on day 0, analyzed on week 12. Data are means + SD; n = 4 – 9.

Study to Establish DYNE-302 Functional Benefit in the Induced hTfR1/iFLExD FSHD Mouse Model



Notes: IV = intravenous; PO = oral gavage; TMX = tamoxifen; hTfR1/iFLExD mice lacking ACTA-CRE-ER transgene were used as controls for TMX effect on function (normal control). hTfR1/iFLExD mice that were not induced with tamoxifen were also included as uninduced control (not shown).

Single Dose of DYNE-302 Demonstrated Functional Benefit in the Induced hTfR1/iFLExD FSHD Mouse Model



Notes: Mice dosed with vehicle or DYNE-302 on Day -14. Data are means ± SEM; n = 5 - 6.