Positive Topline Data from Cohort 1 of ELEVATE-44-201 Phase 1/2 Study of ENTR-601-44 in DMD

May 7, 2026

entrada THERAPEUTICS

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The words “anticipate,” “believe,” “continue,” “could,” “estimate,” “expect,” “intend,” “may,” “might,” “objective,” “ongoing,” “plan,” “predict,” “project,” “potential,” “should,” or “would,” or the negative of these terms, or other comparable terminology are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Entrada may not actually achieve the plans, intentions or expectations disclosed in these forward-looking statements and you should not place undue reliance on these forward-looking statements. 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Today's speakers

Dipal Doshi
Chief Executive Officer

Natarajan Sethuraman, PhD
President of Research & Development

Laurent Servais, MD, PhD
Professor of Pediatric Neuromuscular Diseases, University of Oxford

Today's agenda

Establishing a New Class of Genetics Medicines
Dipal Doshi
Chief Executive Officer

DMD Landscape and Patient Need
Laurent Servais, MD, PhD
Professor of Pediatric Neuromuscular Diseases, MDUK Oxford Neuromuscular Centre

Topline Data from Cohort 1 of ELEVATE-44-201
Natarajan Sethuraman, PhD
President of Research & Development

Upcoming Catalysts
Dipal Doshi
Chief Executive Officer

Q&A
Entrada Management & Laurent Servais, MD, PhD

2026 is a transformational year for Entrada

Entrada is a clinical-stage biopharma company developing proprietary genetic medicines to deliver best-in-class outcomes in high unmet need diseases

Deep pipeline with value inflection points in 2026

- **5 clinical-stage programs** in DMD and DM1
- **4 clinical data catalysts** in DMD and DM1 patients achieved or expected in 2026
- **2 development candidates** in inherited retinal diseases projected by year-end 2026

Differentiated programs in untapped markets

- **$5 billion U.S. DMD market**, with limited competitive penetration; Untapped global markets
- **FDA Accelerated Approval strategy** to be supported by Phase 1/2 data from ex-U.S. DMD studies
- **Proprietary and differentiated** delivery and active moiety sequences in all candidate programs
- **Best-in-class preclinical data** in all declared clinical and preclinical programs

Capitalized to realize value catalysts

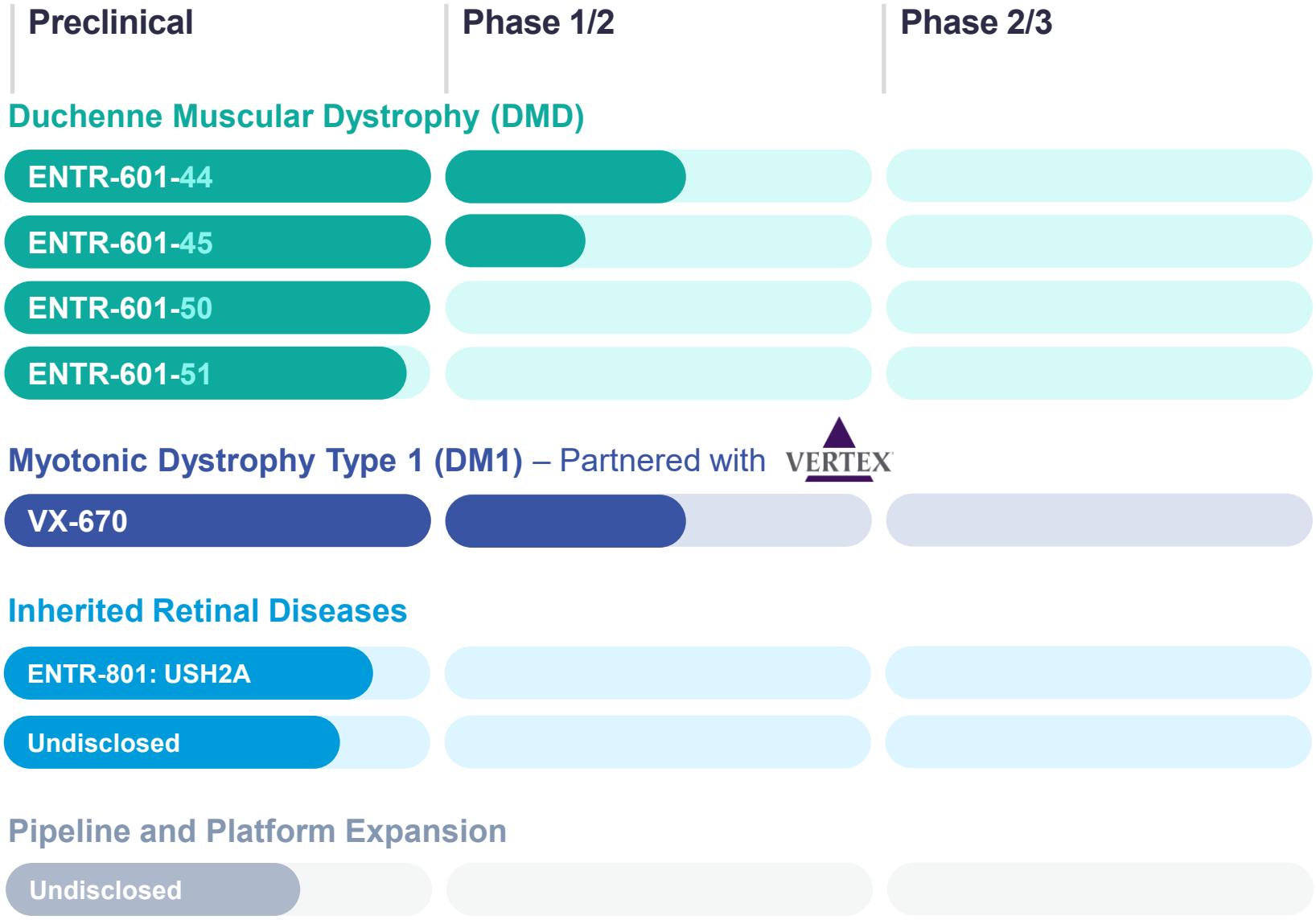
- **Cash runway into Q3 2027**
- **Up to $485 million in DM1 milestones**, plus royalties associated with Vertex partnership

DMD: Duchenne muscular dystrophy; **DM1**: Myotonic dystrophy type 1; Cash runway based on current operating plans and ending cash, cash equivalents and marketable securities as of March 31, 2026.

Advancing five fully owned programs and one partnered program

Phase 1/2 data from ex-U.S. DMD studies to support Accelerated Approval regulatory filings in the U.S.

Each target disease has a substantial patient population with a significant unmet medical need



DMD Landscape and Patient Need

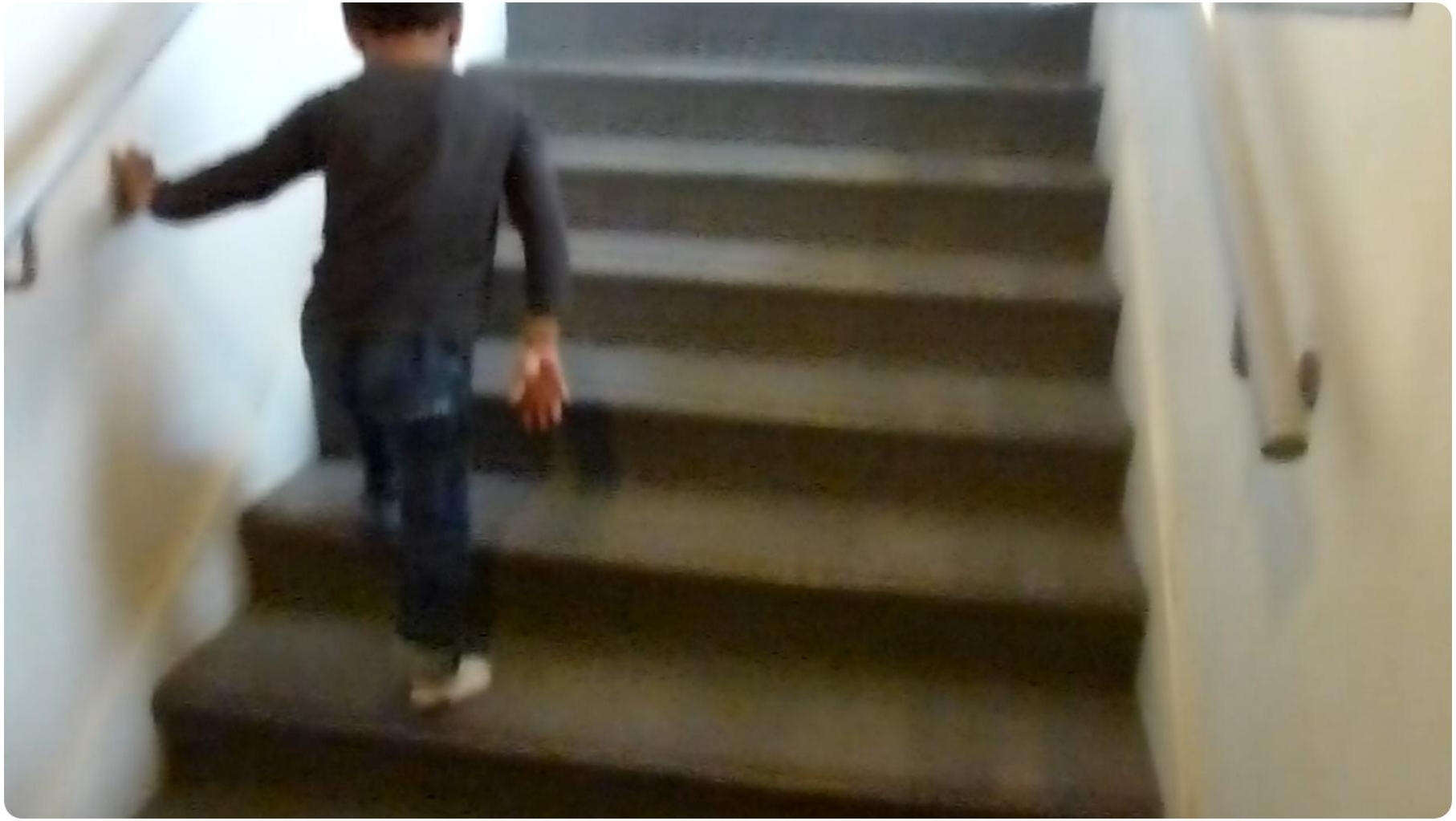
Dr. Laurent Servais, MD, PhD

Professor of Pediatric Neuromuscular Diseases, University of Oxford

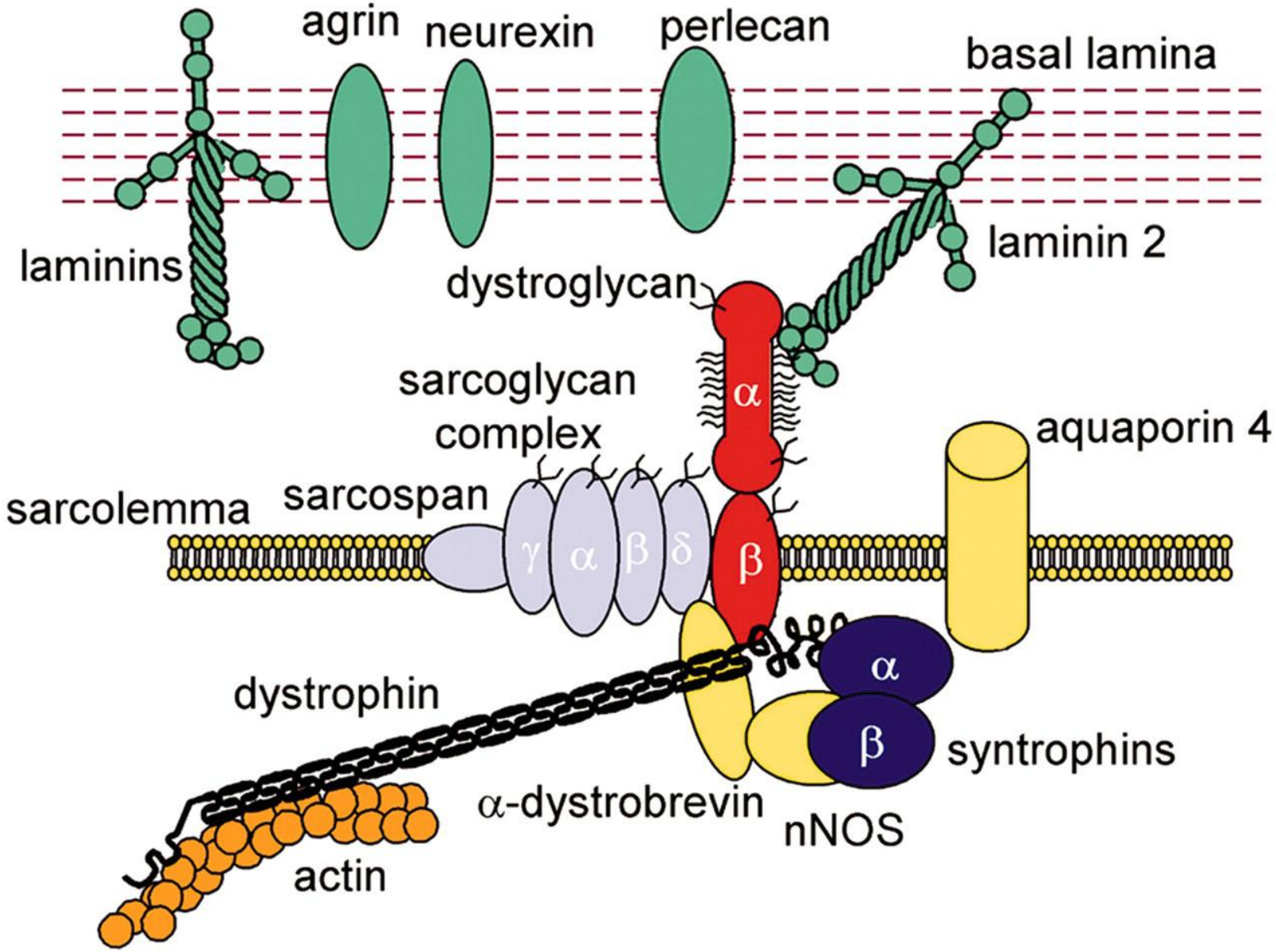
Principal Investigator in the ELEVATE-44-201 clinical study

Example DMD Functional Test: 4-Stair Climb (4SC)

For illustrative purposes only



Professeur Laurent Servais, MD, PhD

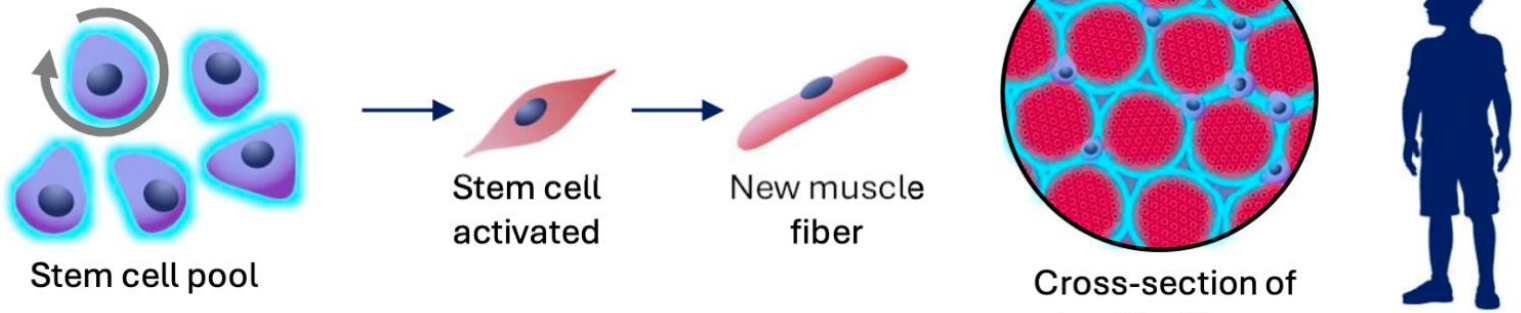


From Michel & Campbell 2003

Dystrophin: Dual Roles in Muscle Health

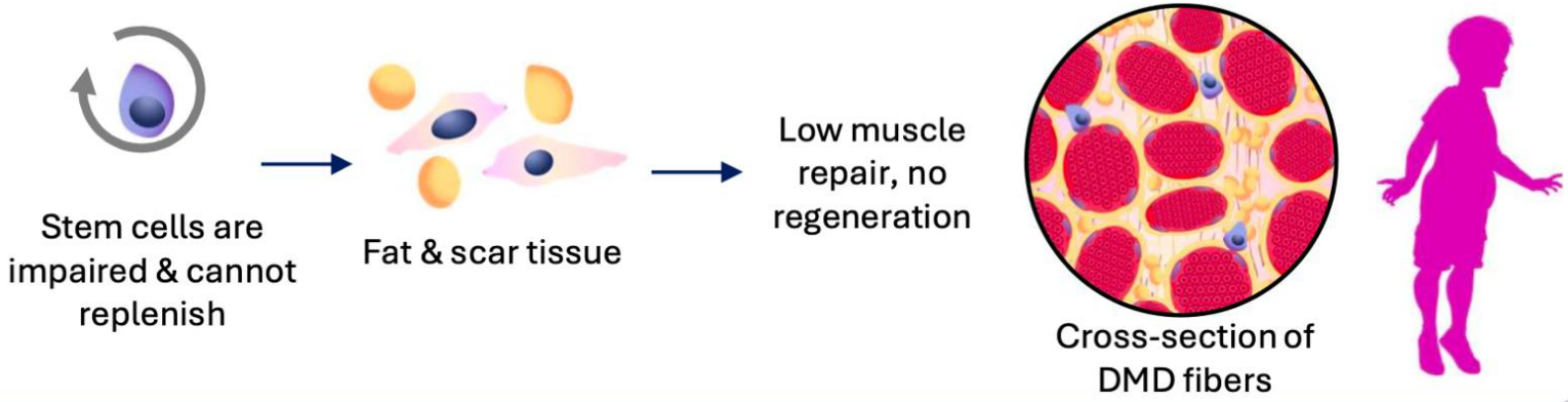
Healthy muscle

- Dystrophin supports stabilizing and repairing muscle fibers and stem cell replenishment & function



DMD muscle lacking dystrophin

- Muscle fibers susceptible to damage
- Stem cells and regeneration impaired
- Muscle replaced with fat and scar tissue



Lack of dystrophin leads to progressive muscle deterioration in DMD

Professeur Laurent Servais, MD, PhD

Example DMD Symptom Map and Bodily Impact

For illustrative purposes only

Cognition…. *School support, methylphenidate*

Scoliose? *Arthrodèse*

Respiratory? *Vaccination, ventilation, infection management*

Heart management

Feeding, constipation

Bone health

Endocrine

Mobility support… wheelchair

Contracture…. Orthopaedic management… Surgery?

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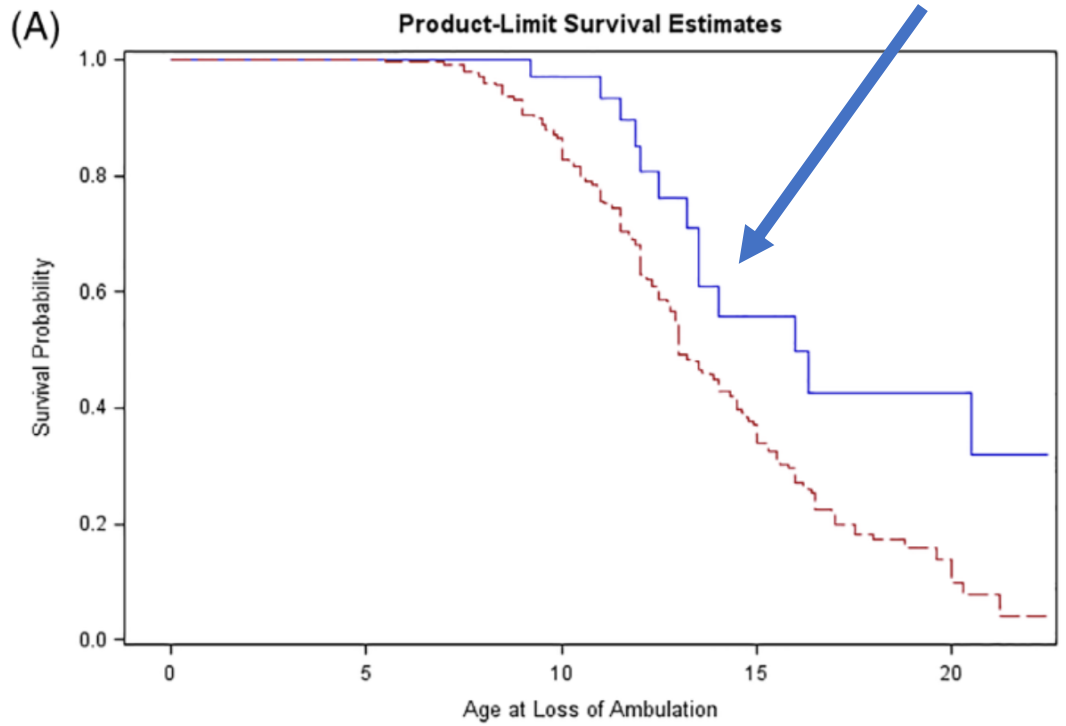
CLINICAL RESEARCH ARTICLE

MUSCLE&NERVE WILEY

The impact of genotype on age at loss of ambulation in individuals with Duchenne muscular dystrophy treated with corticosteroids: A single-center study of 555 patients

Alexander Zygmunt MD[1,2] | Brenda Wong MD[3] | David Moon MD[4] | Paul Horn PhD[1,2] | Richard Rathbun MS[1] | Joshua Lambert MS[5] | Jean Bange MS[1] | Irina Rybalsky MD[1] | Lisa Reebals APRN-CNP[1] | Cuixia Tian MD[1,2]

Skippable 44



Delta with skippable 51 for median LoA: 4.5 years

5% expression of truncated dystrophin since birth could translate in 4-5 years gain in ambulation

ELEVATE-44-201:
Cohort 1 Study Results Summary

Executive summary and key takeaways
ELEVATE-44-201 Cohort 1 results

- **Favorable safety and tolerability profile at 6 mg/kg; All AEs were mild to moderate**
  - No reported SAEs nor AEs leading to discontinuation
  - Renal markers within normal range and comparable to placebo
- **Updated PK modeling, based on recently completed juvenile NHP data, supports path to higher dystrophin levels in Cohort 2 and 3 and a complete understanding as to why Cohort 1 did not result in double-digit dystrophin levels**
  - Dystrophin levels in Cohort 1 were lower than predicted, due to lower than predicted plasma drug exposures
- **Importantly, a potential disease-modifying effect with functional benefit has been demonstrated at 6 mg/kg**
  - TTRV is a robust, early prognostic factor for disease progression
  - Consistent and statistically significant impact on Time to Rise velocity, an unbiased Phase 3 registrational endpoint at levels several fold above MCID after only 3 doses (127 days)
- **Mechanistic advantages support early functional benefit observations**
  - EEV-PMO uptake in satellite cells can lead to the proliferation, activation and asymmetric division which is the basis of muscle regeneration
  - Continued benefit in muscle function expected in the open-label period of Cohort 1 and future cohorts

PK modeling led to lower-than-expected exposure which explains the Cohort 1 dystrophin levels; Early functional benefit response is compelling and statistically significant

AE: Adverse event; **SAE**: Serious adverse events **TTR**: Time to Rise, *post hoc* analysis; **TTRV**: Time to Rise velocity; **MCID**: Minimal clinically important difference; Pharmacokinetics may be abbreviated to **PK** and Pharmacodynamics may be abbreviated to **PD**.

ELEVATE-44-201:
Trial Design and Treatment Population

ELEVATE-44-201 is designed to support U.S. Accelerated Approval and forms the basis of a global registrational program*

ENTR-601-44

ELEVATE-44-201 is a global, two-part, randomized, double-blind placebo-controlled Phase 1/2 study in ambulatory patients*

Primary objective: Safety and tolerability of ENTR-601-44

Secondary objectives: Evaluation of pharmacokinetics, exon skipping, dystrophin production and measures of function

Screening	Double-Blind Period	Open-Label Period	Follow-up Period
Up to 6 weeks	19 weeks	31 weeks	6 weeks

Ambulatory participants with DMD who are exon 44 skipping amenable

Muscle biopsy #1

Part A

N=24 participants across three cohorts, n=8 per cohort

Within each cohort, randomization 3:1 (ENTR-601-44 to placebo)

- ✓ **Cohort 1: 6 mg/kg or placebo**
- Cohort 2: **12** mg/kg or placebo
- Cohort 3: **≤18** mg/kg or placebo

x3 doses
Days 1, 43 and 85

Muscle biopsy #2
Day 127

Participants who complete Part A are able to continue into open-label period

- Receive 6 doses of ENTR-601-44 at the level that was administered in participant's Part A cohort
- Dosing initiated
- Study blinded to end of open-label period

- ✓ Complete
- Ongoing

Expansion Cohort

*Expansion cohort and potential for Accelerated Approval is subject to regulatory feedback; Global registration and approval will require Phase 3 studies to follow the Phase 1/2 studies; Phase 3 studies will likely include functional measures as the primary endpoint (U.S. confirmatory study/EU registrational study).

Demographics and baseline characteristics
Cohort 1

ELEVATE-44-201

	Placebo n=2	ENTR-601-44 6 mg/kg n=6
Age, mean	**13.5**	**9.3**
Body mass index, mean, kg/m^2	17.96	20.00
Age at disease onset, mean, years	1.0	2.2
Corticosteroid use, n (%)	2 (100%)	6 (100%)
Ambulatory, n (%)	2 (100%)	6 (100%)
Baseline dystrophin	4.6%	4.0%

Note: The baseline dystrophin levels seen in Cohort 1 were the lowest levels observed in recent studies of Exon 44 skipping programs

ELEVATE-44-201: Safety Data

Safety was consistent and unremarkable at the 6 mg/kg dose

ELEVATE-44-201

Participants with ≥1 TEAE, n (%)	Placebo n=2	ENTR-601-44 6 mg/kg n=6
Any TEAE	2 (100%)	6 (100%)
TEAEs related to study drug	1 (50%)	5 (83%)
Serious TEAEs	**0**	**0**
TEAEs leading to study discontinuation	0	0
TEAEs leading to death	0	0

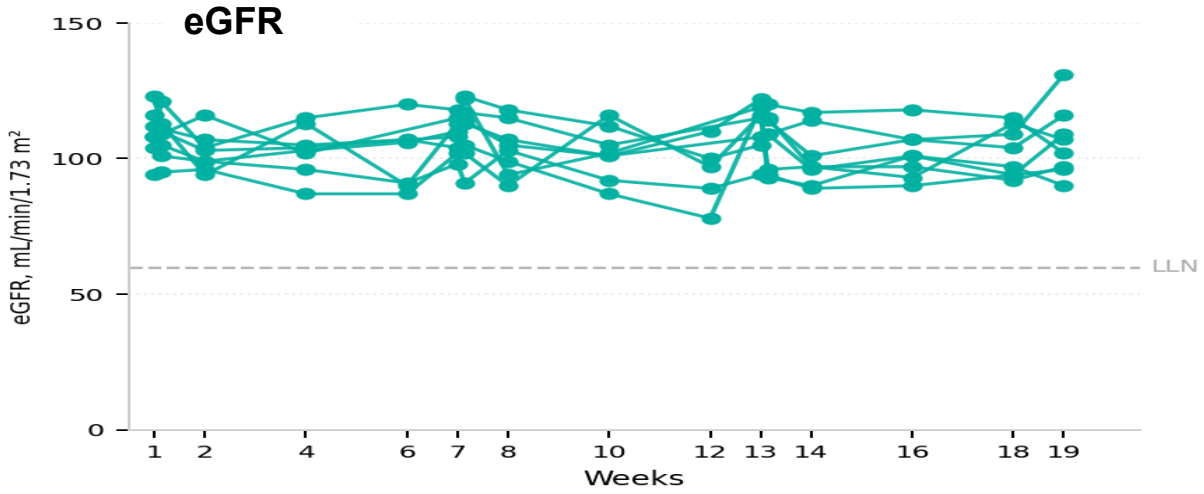
All TEAEs were mild to moderate

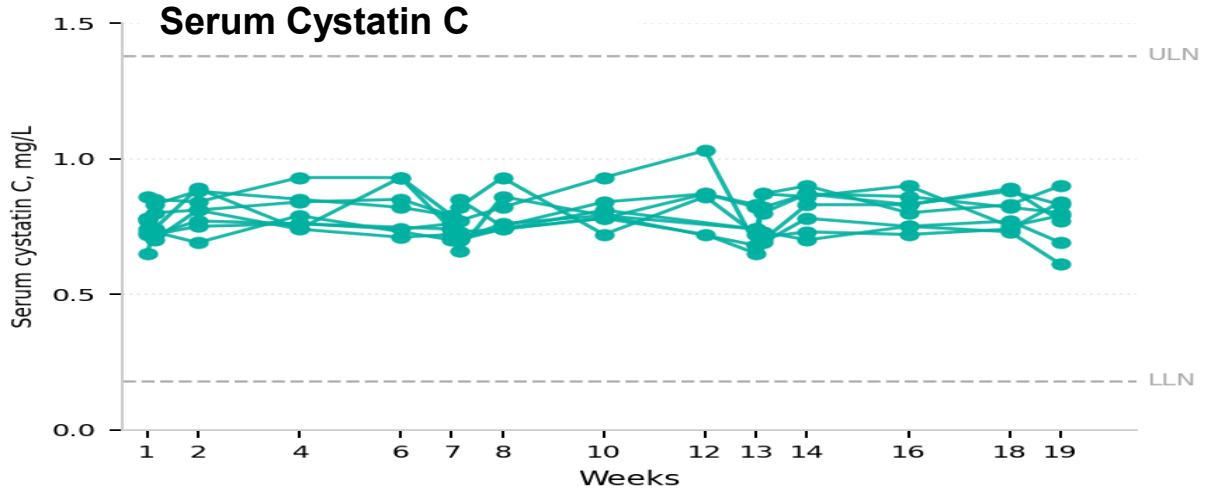
- Headache was the most common study drug-related TEAE, reported in 50% of the treatment group and 50% of the placebo group
- All events resolved
- **There were no serious TEAEs** and no study discontinuation due to any causes
- **No hypomagnesemia** or renal safety concerns were noted

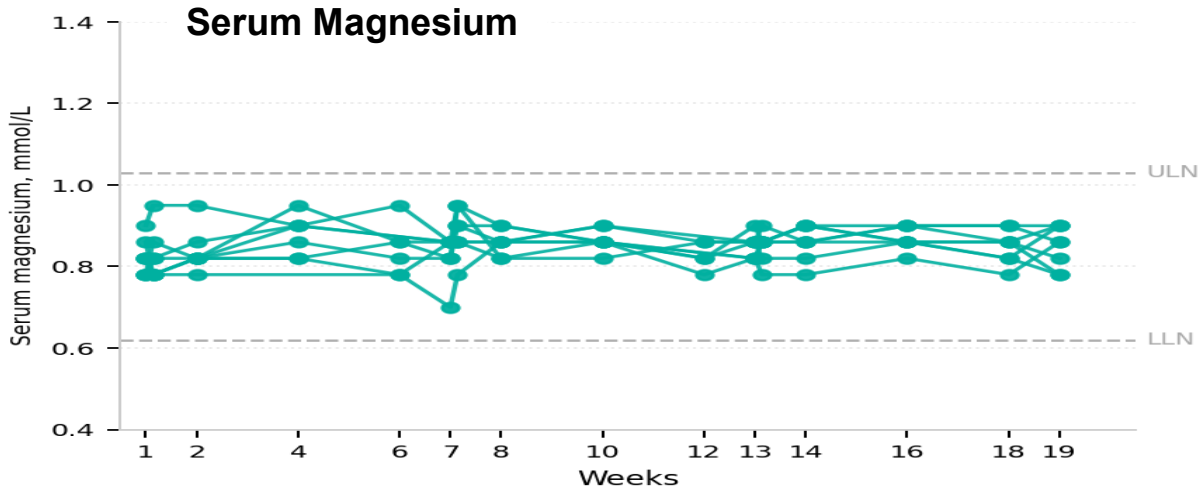
TEAE: Treatment emergent adverse event.

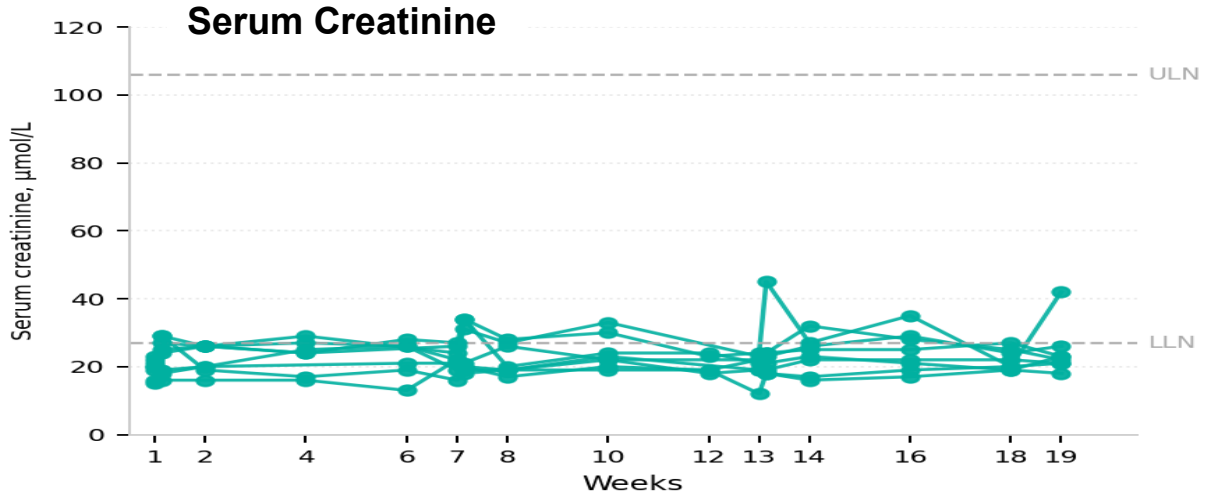
Renal markers were within normal range and comparable to placebo

ELEVATE-44-201







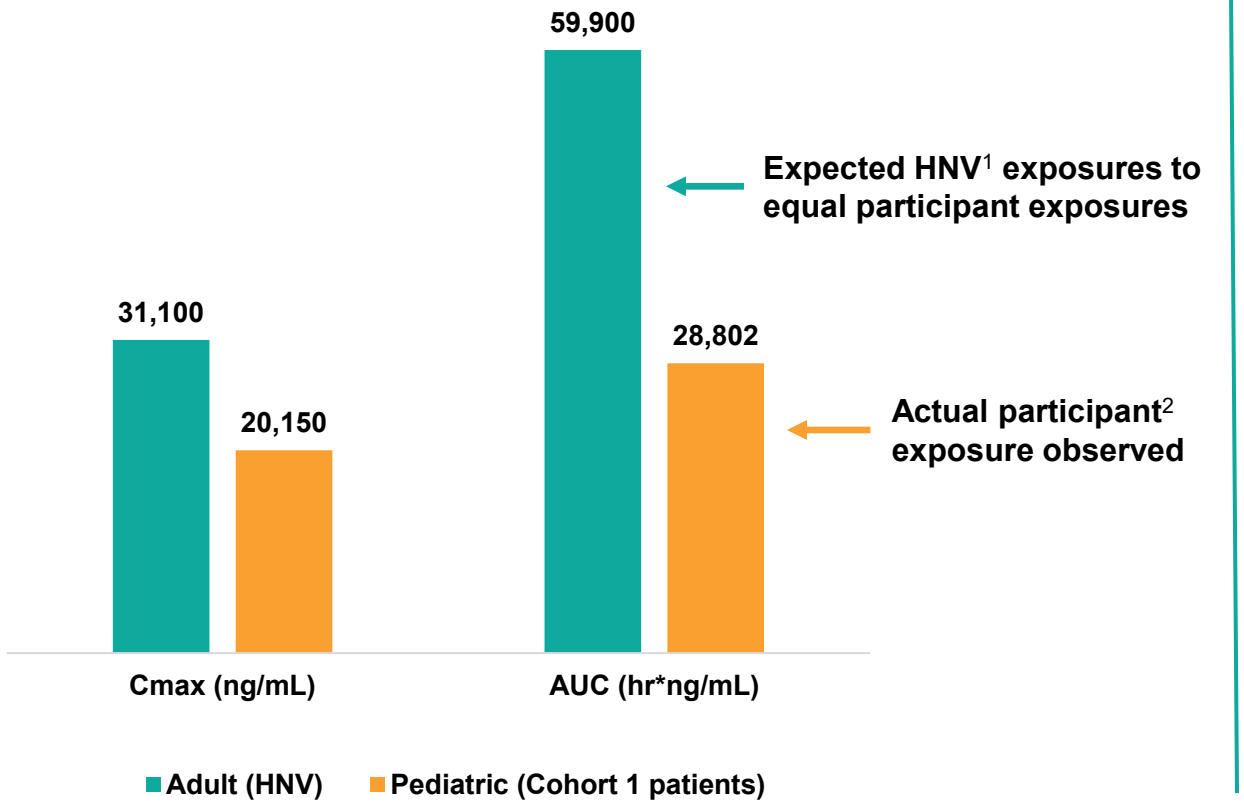


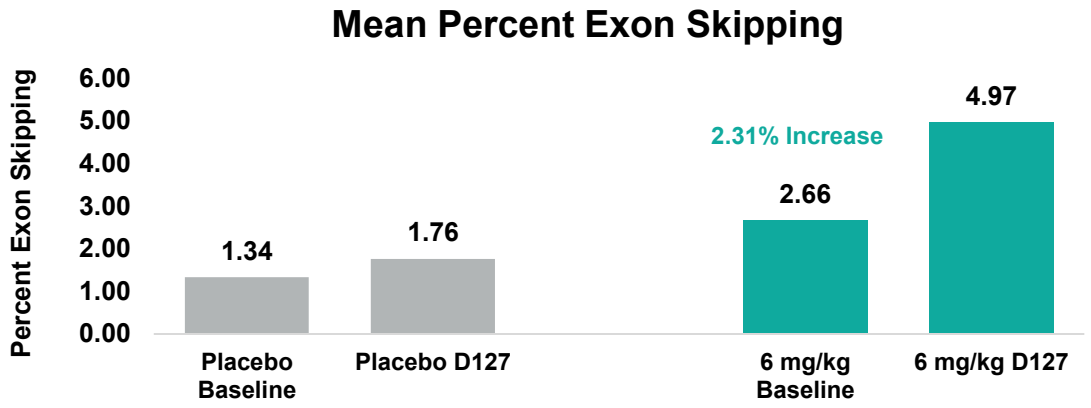
Data from ELEVATE-44-201; eGFR: Estimated glomerular filtration rate; LLN: Lower limit of normal; ULN: Upper limit of normal; Serum creatinine reference range shown is 27–106 µmol/L and is dependent on age of the participant enrolled in Cohort 1; Serum creatinine is below normal in patients with DMD due to reduced muscle mass.

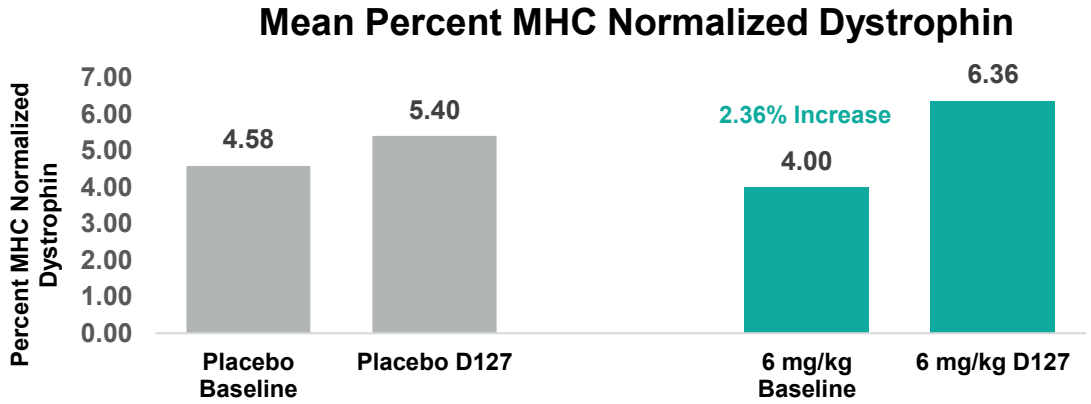
ELEVATE-44-201: Pharmacokinetics and Biomarkers

Pharmacokinetic modeling led to lower than predicted exposures, resulting in lower exon skipping and dystrophin expression

Lower C_{max} and AUC observed in pediatric participants when compared with healthy adults





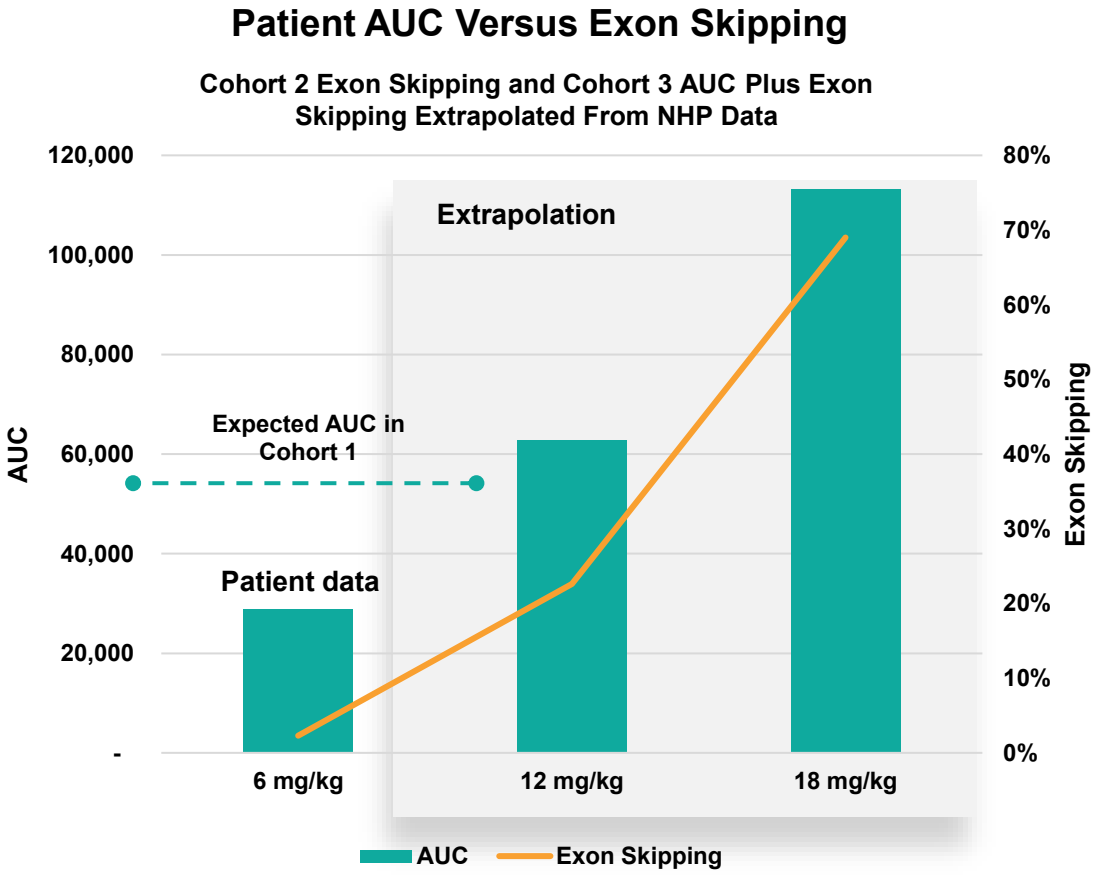


AUC: Area under the curve; **D127**: Day 127; [1] Healthy normal volunteer, single dose 6 mg/kg, [2] Pediatric DMD participants following third dose of 6 mg/kg every 6 weeks; **MHC**: Myosin heavy chain; Note: As biopsies were taken 6 weeks post final dose, muscle concentration levels were 10-fold below projected peak, in-line with plasma concentration and expectations.

Improved predictive modeling based on new data point to higher dystrophin levels in Cohorts 2 and 3

Participant data from Cohort 1, combined with juvenile NHP data, suggests an increase of AUC should result in substantially higher dystrophin levels and increases in muscle function

- **Updated projections indicate significantly increased exon skipping and dystrophin expression expected in Cohort 2 and Cohort 3**
- Original “double-digit” dystrophin assumption was based upon AUC levels that are expected to be achieved in Cohort 2
- Cohort 2 and 3 doses will be assessed for registrational potential, when combined with observed functional outcomes



NHP data from 55 weeks, QW6, 10 doses, chronic tox study; Extrapolation based on exon skipping/AUC at each dose (dose normalized on a mg/kg basis across non-clinical and clinical).

PK analysis
Conclusions and looking ahead

Updated PK analysis has determined that future cohorts should generate higher dystrophin restoration

- Initial PK modeling overestimated the exposures in participants and exon-skipping/dystrophin expression that could be achieved at the Cohort 1 dose due to a reliance on adult projections (NHP and humans)
- The lower levels of plasma exposures seen in juveniles when compared to the plasma exposures seen in adults were similar across the non-clinical and clinical settings
- This new finding explains the delta between the original exposure and biomarker projections and observed exposure and biomarker findings in patients
- Updated PK analysis projects higher levels of plasma concentration, exon skipping and dystrophin
  - A statistically significant improvement in dystrophin production has been used to predict functional benefit and, in conjunction with a trend towards functional improvement, continues to be accepted as the basis for Accelerated Approval in the U.S.

Path to Accelerated Approval and Full Approval has not changed

ELEVATE-44-201: Functional Efficacy Data

Functional improvement
The interpretation and importance of Time to Rise velocity

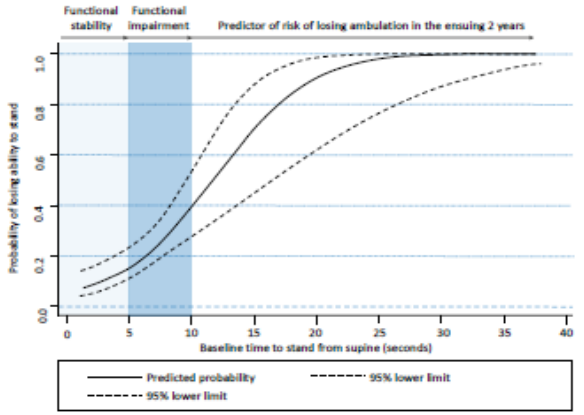
Time to Rise is a robust, prognostic factor that predicts disease progression; Time to Rise velocity reduces the impact of imputation, outliers and noise

- **TTR declines rapidly** over time in patients with DMD and is an early **prognostic factor for disease progression and loss of ambulation**[1]
  - The largest absolute and proportional annual signal among functional measures and early prognostic factor for disease progression and loss of ambulation
  - Robust, as rising from the floor depends on proximal strength and postural control, functions affected early in disease progression[1]
  - Precedes improvements in 4-stair climb or 10-meter walk test and less complicated than NSAA[2]
- **TTRV** is calculated as 1/TTR, expressed as rises/second and is designed to reduce the impact of outliers and imputed data (illustration at right)
  - Handles the unable to perform problem by scoring that observation at zero avoiding arbitrary scoring
  - Dampens clinically meaningless scoring noise between visits
  - Compresses the long tail and produces a distribution that is much closer to normal, which matters for parametric statistics

Table 1. Clinical relevance of TTSTAND measurements[1]

TTSTAND	Clinical relevance
<5 seconds	Suggests functional stability[2,3]
≥5 seconds	Indicates functional impairment[2,3] Shown to predict disease progression over 48 weeks[2,3]
≥10 seconds	Shown to predict risk of losing ambulation in the ensuing 2 years[3]

Fig 1. Predicted probability of losing ability to stand at 24 months given baseline time to stand from supine assessment (TTSTAND). Adjustment made for baseline age and cumulative steroid use at baseline



Illustrative Data and Representative Calculation of TTRV

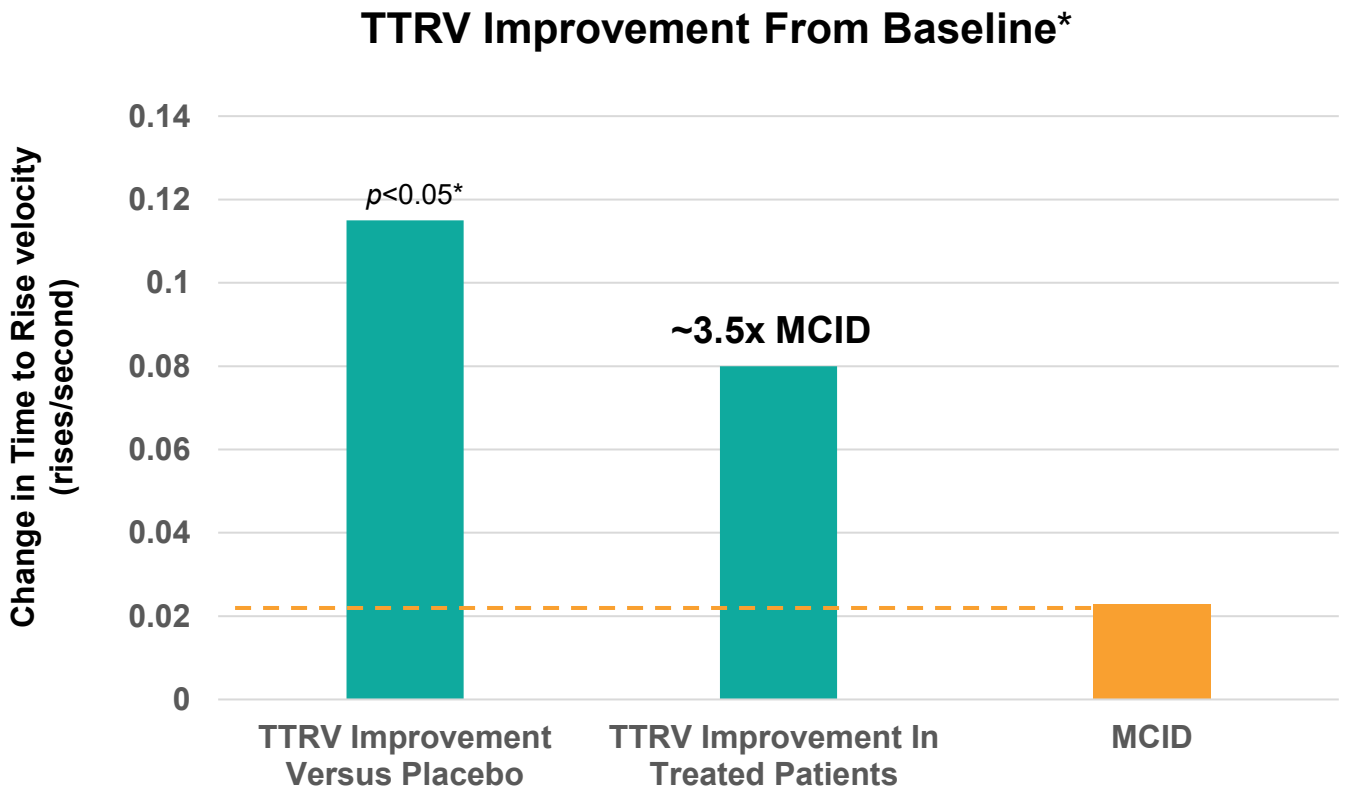
TTR baseline (seconds)	TTR end of study (seconds)	Improvement (seconds)	TTRV calculation	Δ TTRV (rises/sec)
6.0	4.05	1.95	1/4.05 – 1/6.0 = 0.247 – 0.167	**+0.08**
8.0	4.88	3.12	1/4.88 – 1/8.0 = 0.205 – 0.125	**+0.08**
10.0	5.56	4.44	1/5.56 – 1/10.0 = 0.180 – 0.100	**+0.08**

Explanation: A boy starting at a TTR of 6 seconds needs to improve by ~2 seconds to achieve +0.08 ΔTTRV, but a boy starting at a TTR of 10 seconds needs to improve by ~4.5 seconds for the same velocity change

[1]Cicala et al., Italian DMD Network (WMS 2025); [2]McDonald et al., CINRG DNHS (PPMD 2021); **NSAA:** North Star Ambulatory Assessment.

Disease-modifying functional benefit seen at lowest dose
TTRV results

A significant improvement in TTRV was observed*; TTRV is a widely accepted Phase 3 endpoint in DMD, with good statistical properties for evaluating motor function in patients



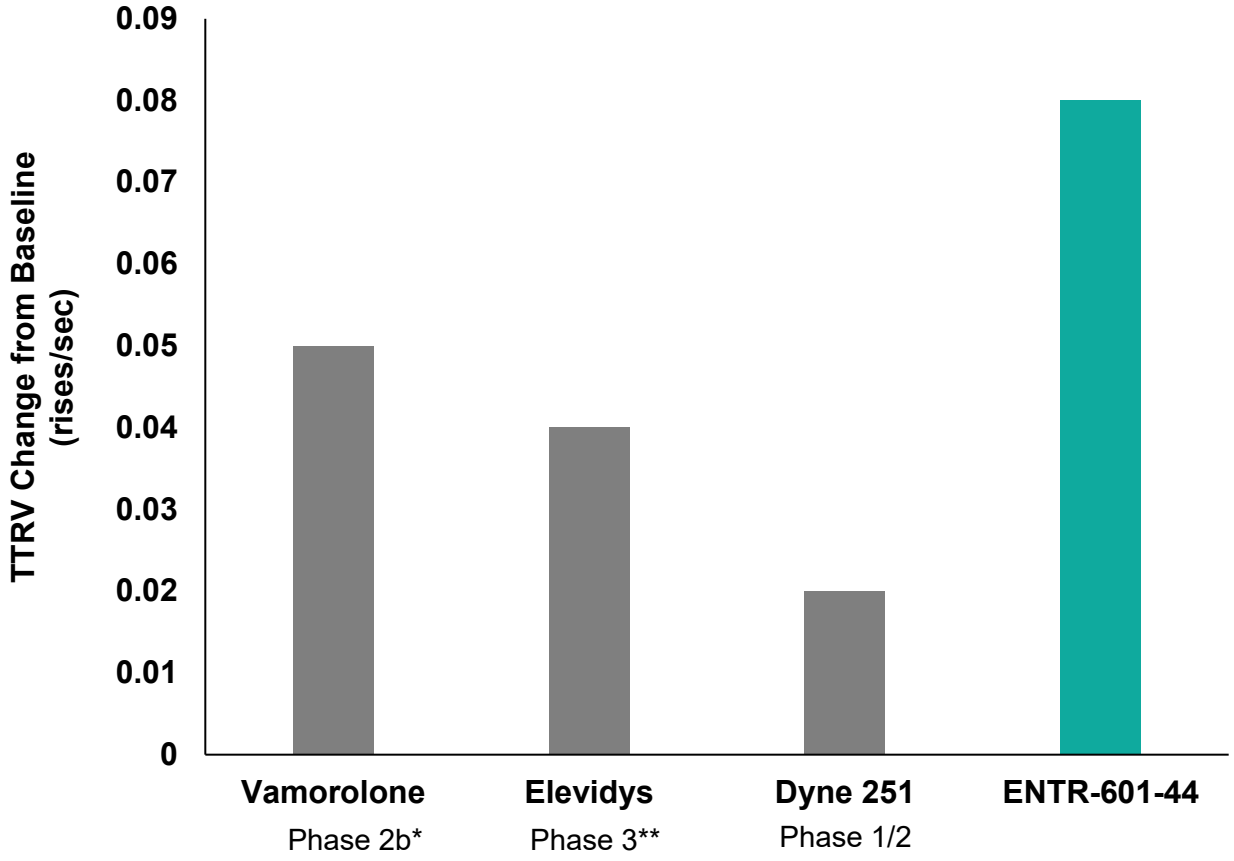
- Change in TTRV was seen across the majority of participants, irrespective of disease severity or age
- The mean change in TTRV was 0.08, 3.5x higher than the MCID threshold of 0.023; Mean change versus placebo was 0.115
- End of Cohort 1 dystrophin levels correlated with the end of Cohort 1 TTRV, suggesting that dystrophin production may have crossed a critical threshold for functional improvement
- The consistency of the data supports the hypothesis that Cohort 1's functional benefit represents a true drug-related effect
- In addition, 10-meter walk/run also demonstrated positive trends versus placebo

*Not the primary endpoint in the study, *post hoc* analysis; MCID**:** "The Minimal Clinical Important Difference (MCID) in Annual Rate of Change of Timed Function Tests in Boys with DMD," Duong T, Canbek J, Birkmeier M, Nelson L, Siener C, Fernandez-Fernandez A, Henricson E, McDonald CM, Gordish-Dressman H, and the CINRG-DNHS Investigators; Journal of Neuromuscular Diseases. 2021;8(6):939–948.

ENTR-601-44 delivered a differentiated impact on TTRV

TTRV is a validated registrational endpoint

Reported TTRV Competitive Comparisons*



Primary Endpoint In Registrational Clinical Trials

- **Santhera**: Vamorolone, Phase 2b (0.048 TTRV)
- **NS Pharma**: Vitolarsen, Phase 3 (did not report TTRV)
- **Solid Biosciences**: SGT-003, Phase 3 (ongoing)
- **REGENEXBIO**: RGX-202, Phase 3 trial (ongoing)
- **Roche**: Elevidys, Phase 3 trial (to initiate in EU)
- **Novartis**: *del-zota*, Phase 3 trial (ongoing)

Sources: *VISION-DMD (JAMA 2022) registrational study; DELIVER (Dyne Company presentation); EMBARK (FDA Decisional Memo 2024 – note this is versus placebo); ENTR-601-44 (Cohort 1); No head-to-head trials have been conducted; Cross-trial comparisons may not be reliable based on differences in study designs, compound formulation and mechanism, patient populations, etc.; **Supplement to original Phase 3 filing in U.S.

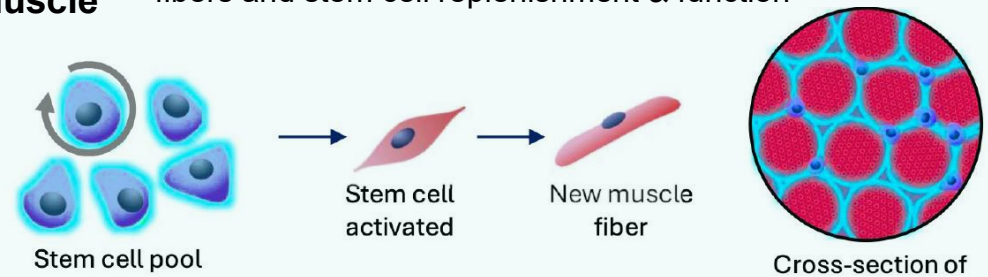
ENTR-601-44: Mechanistic Rationale

The DMD challenge: Muscle damage and impaired repair

An ideal treatment is regenerative – replacing damaged, dystrophic muscle with healthy muscle; This requires satellite cell correction which then promotes asymmetric differentiation

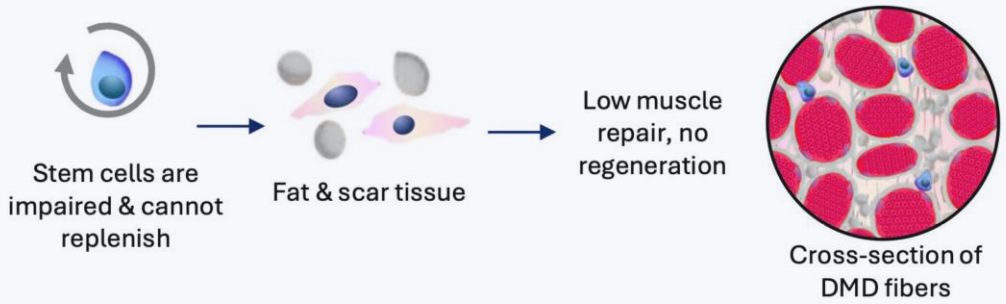
Healthy muscle

Dystrophin supports stabilizing and repairing muscle fibers and stem cell replenishment & function



DMD muscle lacking dystrophin

- Muscle fibers susceptible to damage
- Stem cells and regeneration impaired
- Muscle replaced with fat and scar tissue



The dystrophin "double hit" results in a decline in function

- Lack of dystrophin results in muscle damage
- Lack of dystrophin also results in lack of ability to repair and regenerate new fibers
- Lack of normal regeneration results in the replacement of muscle with fat and fibrosis

Satellite (stem) cells need dystrophin to efficiently promote muscle regeneration

- Deficit inhibits asymmetric differentiation and activation, thus impairing regeneration

Source: Servais L, et al. ICNMD 2026.

EEV-enabled stem cell uptake implies the potential for the return of healthy muscle and function

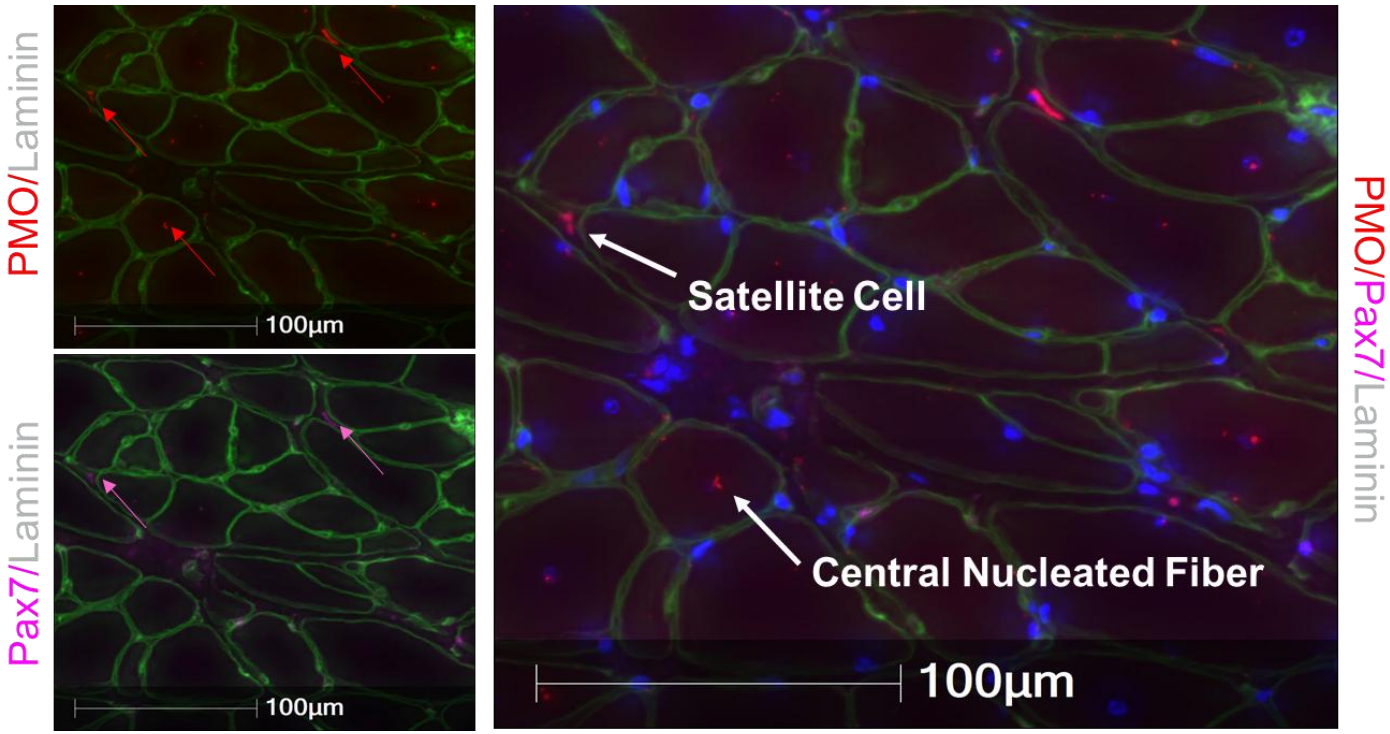
Enhanced satellite cell activity would result in the regeneration of healthy fibers and the stabilization of the overall muscle; In turn, this may manifest in greater strength as measured by TTRV

Entrada differentiation

- Immunohistochemistry data demonstrates PMO in satellite cells and newly regenerated centrally nucleated fibers 12 weeks post-washout after 3 Q6W doses (D2-*mdx* mice)

Antibody and gene therapy competitors

- No transferrin receptor expressed on these cells so the antibodies cannot reach them
- AAV-enabled gene therapies lack ability to efficiently reach satellite cells which limits response durability



Immunohistochemistry data demonstrates PMO in satellite cells and newly regenerated centrally nucleated fibers 12-weeks post-washout after 3 Q6W doses (D2-*mdx* mice)

D2-*mdx* mice were treated with 3 IV doses of EEV-PMO administered every six weeks; Gastrocnemius was collected 12 weeks after the third dose and analyzed by immunohistochemistry; Data presented at International Conference on Muscle Wasting 2025; CK: Creatine kinase; Q6W: Every 6 weeks; D2-*mdx*: Dystrophic mouse model.

2026 Inflection Points

2026 inflection points

ELEVATE-44-201 Cohort 1 open-label functional data, Cohort 2 dystrophin and functional data, and ELEVATE-45-201 Cohort 1 data represent additional 2026 catalysts

- **All ELEVATE-44-201 Cohort 1 participants have rolled over into the open-label portion of the study**
  - Company expects to see a deepening of functional response over time
  - Placebo participants crossing over into treatment via the open-label period
  - Completion of open-label period expected by year-end 2026
- **ELEVATE-44-201 Cohort 2 at 12 mg/kg continues to enroll**
  - Higher levels of plasma and muscle exposure expected
  - Concomitant increases in exon skipping and dystrophin production are expected
  - Continued functional responses expected at higher doses
- **ELEVATE-45-201 Cohort 1 at 5 mg/kg fully enrolled with a readout expected in mid-2026**
  - Plasma exposures expected to be similar to those seen in ELEVATE-44-201 Cohort 1 (due to previous modeling)
  - Implies potential for lower-than-expected exon skipping and dystrophin, but potential for functional improvement at the 5 mg/kg dose

Thank you!

To the individuals and families living with Duchenne who choose to courageously participate in clinical studies

To the clinical investigators and site teams who care for patients with dedication and integrity

To the advocacy organizations whose partnership and leadership strengthen this entire community

Multiple 2026 near-term value drivers anticipated across expanding pipeline of intracellular therapeutics

ENTR-601-44

Global Phase 1/2 MAD study ongoing

Cohort 1 data demonstrated safety, dystrophin production and functional improvement

Cohort 2 data expected by year-end 2026

ENTR-601-45

Global Phase 1/2 MAD study ongoing

Cohort 1 data expected mid-2026

ENTR-601-50

Received authorization for Phase 1/2 MAD study (U.K.)

EU regulatory filing expected following a review of data from the ongoing DMD clinical studies

ENTR-601-51

Global regulatory filings expected following a review of data from the ongoing DMD clinical studies

VX-670

Vertex is on track to complete MAD participant enrollment and dosing, and share results in H2 2026

ENTR-801

Initial IRD candidate focused on Usher syndrome type 2A

Additional data and clinical plans will be shared in 2026

Second IRD candidate declaration in H2 2026

Pipeline Expansion

Next-generation EEVs for neuromuscular expansion

Ocular expansion into larger disease areas

Range of undisclosed diseases and modalities

Cash runway into Q3 2027*

Note: All references in this presentation regarding planned regulatory filings and clinical study designs are subject to ongoing discussion with regulatory authorities; *Based on current operating plans and ~$255M in cash, cash equivalents and marketable securities as of March 31, 2026.

Q&A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