

Corporate Presentation

November 2026

entrada THERAPEUTICS

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This presentation contains express and implied forward-looking statements that involve substantial risks and uncertainties. All statements, other than statements of historical facts, contained in this presentation, including statements regarding Entrada's strategy, future operations, prospects and plans, objectives of management, the validation, differentiation and superiority of Entrada’s approach and EEV platform and its ability to develop best-in-class genetic medicines with the potential for competitive differentiation, expectations regarding Entrada’s Phase 1/2 MAD clinical study of ENTR-601-44, including the timing of data from the Cohort 1 open-label period by year-end 2026 and data from Cohort 2 expected in the first quarter of 2027, with Cohort 3 to follow, expectations regarding the data from ELEVATE-44-201, including the translatability of dystrophin concentrations and exon skipping based on juvenile NHP data, increased dystrophin levels and continued safety margins at higher doses for Cohorts 2 and 3, continued benefit in muscle function in the open-label period of Cohort 1 and future cohorts, the potential for stem cell uptake to support healthy muscle regeneration and function, stabilization of the overall muscle, potentially converting into greater strength as measured by Time to Rise velocity, the ability of renal marker data to further de-risk current and future DMD programs and support dose escalation to drive a higher therapeutic index, additional functional benefit metrics and safety to be received by year-end 2026, double-digit dystrophin expected starting at 12 mg/kg in Cohort 2 and continuing through 18 mg/kg in Cohort 3 based on juvenile NHP data, substantially increased exon skipping and dystrophin expression expected in Cohorts 2 and 3, functional measures to be the primary objective of Phase 3 studies and the data’s ability to support U.S. Accelerated Approval and form the basis of a global registrational program, expectations regarding the connection between EEV-PMO uptake in satellite cells and muscle regeneration, expectations regarding Entrada’s Phase 1/2 MAD clinical study of ENTR-601-45, including the timing of data from Cohort 2 in the first half of 2027, with Cohort 3 up to 15 mg/kg expected to follow pending DMC review, expectations regarding the data from ELEVATE-45-201, including meaningful exon skipping and dystrophin increases in future cohorts, the potential for achieving target concentrations at higher doses and a potential best-in-class clinical profile, expectations regarding regulatory filings and authorizations and the timing of initiation of the planned clinical studies of ENTR-601-50, and ENTR-601-51, the ability to recruit for and complete global Phase 1/2 clinical studies of ENTR-601-44, ENTR-601-45, ENTR-601-50 and ENTR-601-51, the potential for ENTR-601-44 and ENTR-601-45 to be transformative treatment options, the ability of EEV therapies to provide a 25-50-fold improvement in endosomal escape and lower whole drug exposure supporting lower risk of anti-drug antibody response and lower cost of goods, the potential therapeutic benefits of Entrada’s EEV product candidates and the ability to advance therapeutic candidates in indications beyond neuromuscular disease, including but not limited to ocular disease, the ability to continue to expand and develop additional therapeutic programs and modalities, including next-generation EEVs for neuromuscular expansion and ocular expansion into larger disease areas, expectations regarding Entrada’s ENTR-801 program, including the potential for quarterly dosing, robust exon skipping and truncated usherin protein production, the size of the USH2A market opportunity in the U.S. and ex-U.S. and Entrada sharing clinical strategies in 2026, the nomination of a second clinical candidate for inherited retinal disease by year-end 2026, expectations regarding Entrada’s next-generation exon skippers, including durable and significant access to skeletal and cardiac muscle, targeting of progenitor cells and flexibility to dose escalate and re-dose, expectations regarding the ability to develop genetic medicines to correct the mechanism of disease and slow, halt or reverse progression, the continued development and advancement of ENTR-601-44, -45, -50 and -51 for the potential treatment of DMD and ENTR-801 for the potential treatment of Usher syndrome type 2A and the partnered product candidate VX-670 for the potential treatment of DM1, expectations regarding the progress and success of Entrada’s collaboration with Vertex, including expected proceeds from program milestones plus royalties and completion of the MAD portion of the global Phase 1/2 study of the VX-670 program and interim results shared in the second half of 2026, the size of the DMD market opportunity in the U.S. and ex-U.S., and the sufficiency of Entrada’s cash resources into the fourth quarter of 2027, constitute forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995. 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. Actual results or events could differ materially from the plans, intentions and expectations disclosed in these forward-looking statements as a result of various important factors, including: uncertainties inherent in the identification and development of product candidates, including the conduct of research activities and the initiation and completion of preclinical studies and clinical studies; uncertainties as to the availability and timing of results from preclinical and clinical studies; the timing of and Entrada’s ability to submit and obtain regulatory clearance and initiate clinical studies; whether results from preclinical studies or clinical studies will be predictive of the results of later preclinical studies and clinical studies or comparative performance against competing products or product candidates; whether Entrada's cash resources will be sufficient to fund Entrada's foreseeable and unforeseeable operating expenses and capital expenditure requirements; as well as the risks and uncertainties identified in Entrada's filings with the SEC, including Entrada's most recent Form 10-K and in subsequent filings Entrada may make with the SEC. In addition, the forward-looking statements included in this presentation represent Entrada's views as of the date of this presentation. Entrada anticipates that subsequent events and developments will cause its views to change. However, while Entrada may elect to update these forward-looking statements at some point in the future, it specifically disclaims any obligation to do so. These forward-looking statements should not be relied upon as representing Entrada's views as of any date subsequent to the date of this presentation.

Entrada is developing best-in-class genetic medicines to address diseases with a significant unmet need

Proprietary Modalities

Proprietary active moieties and novel delivery constructs combine to optimize therapeutic response

Clinical Validation

Favorable participant safety and functional improvement data de-risk the neuromuscular franchise

Expanding Pipeline

Advancing programs across DMD, DM1 and inherited retinal diseases complemented by an active discovery engine

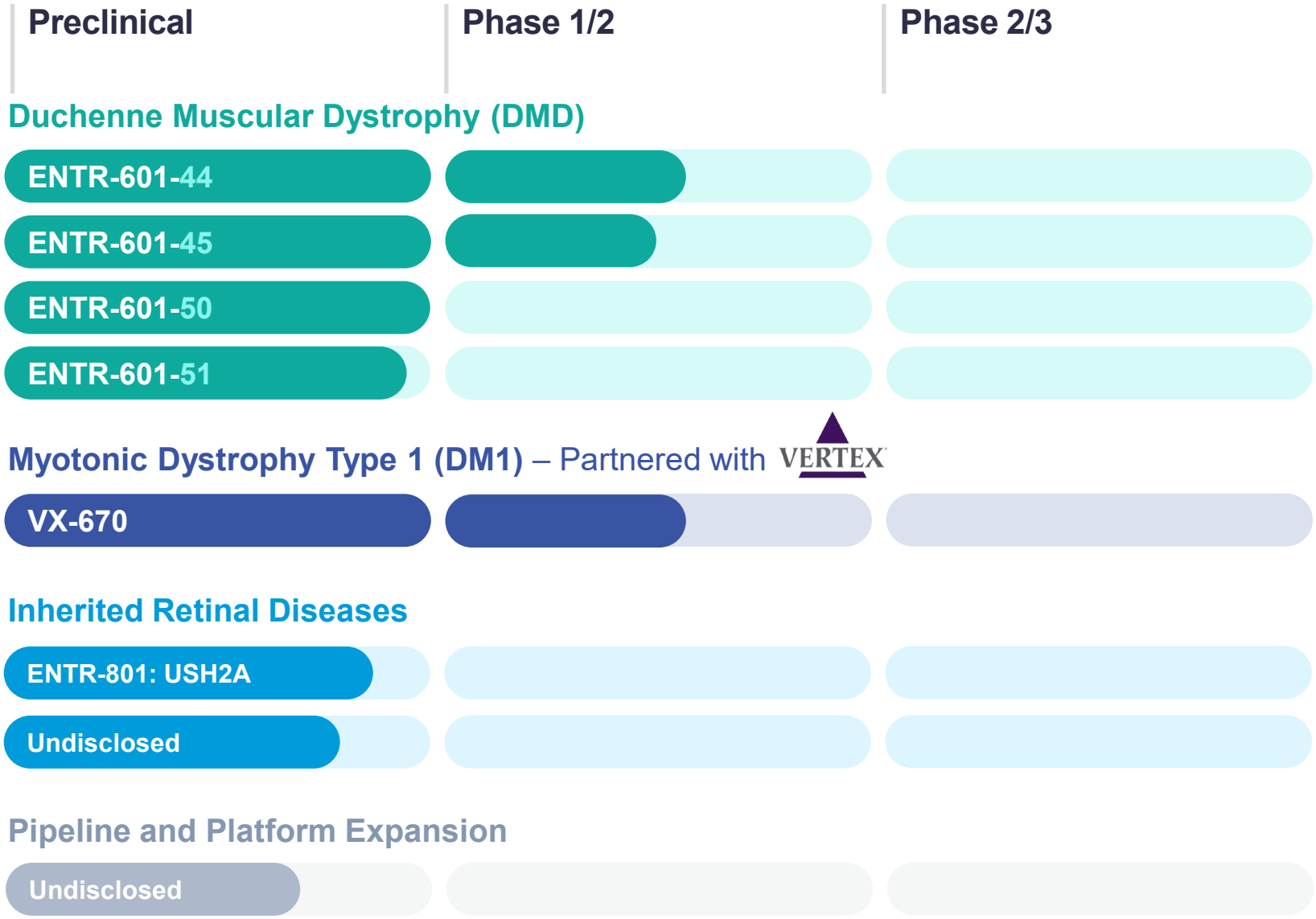
4 clinical data catalysts expected through H1 2027

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

November 2026





Neuromuscular Programs

Entrada's neuromuscular strategy is to clinically de-risk a differentiated portfolio and launch best-in-class drugs

Multiple clinical catalysts, growing platform validation and a differentiated pipeline position Entrada for significant value creation

Neuromuscular Strategy

- Neuromuscular programs **leverage the same EEV**
- **Novel PMO** sequences increase potential competitive differentiation
- **Lower whole drug exposure** supports lower risk of anti-drug antibody response and lower cost of goods
- Stem cell uptake supports **potential for healthy muscle cell regeneration**
- Phase 1/2 DMD data from ex-U.S. studies to support **U.S. Accelerated Approval***

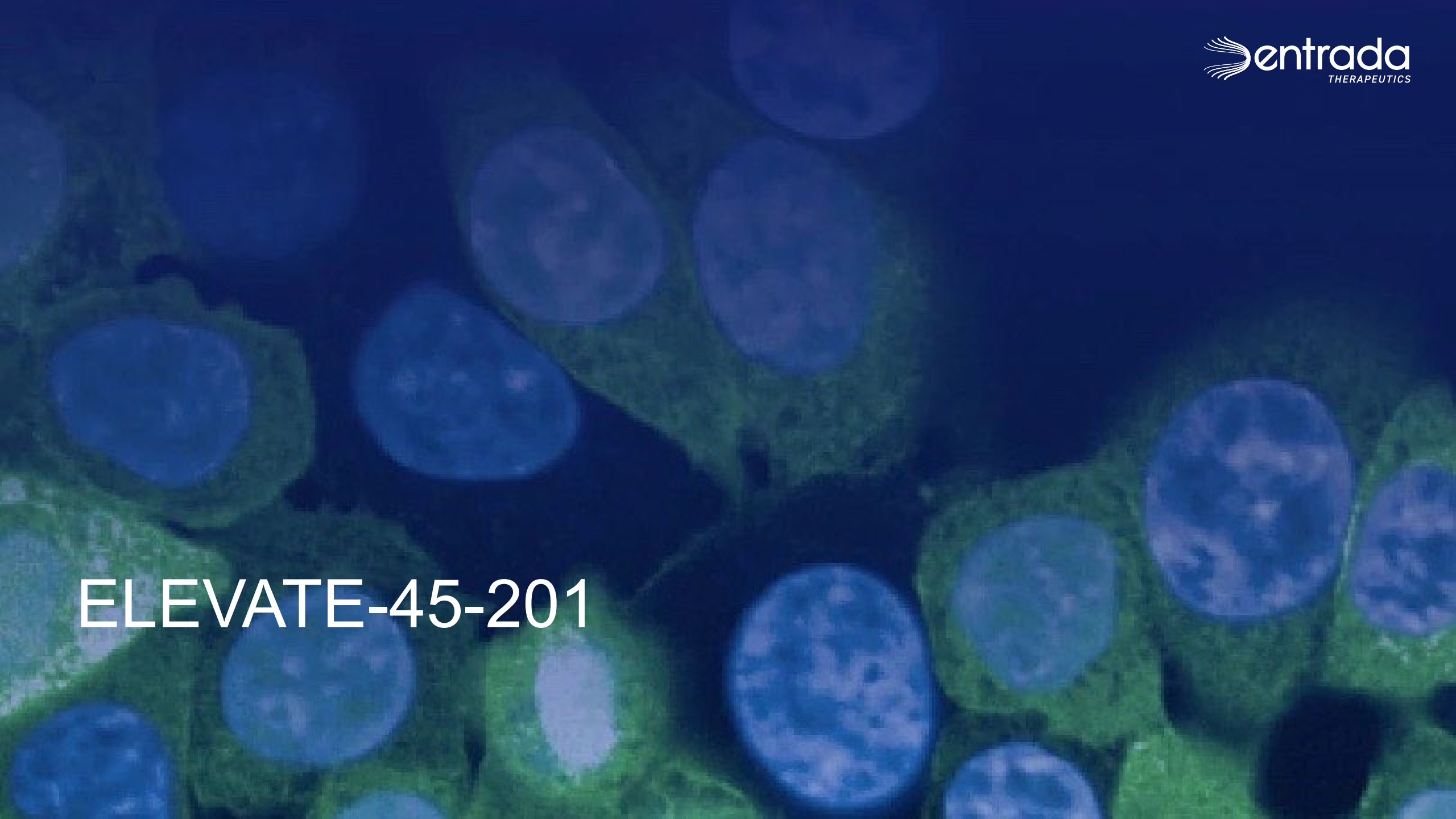
Clinical Differentiation

- **Favorable safety profile** across both Cohort 1 studies of ENTR-601-44 and ENTR-601-45
- **No discontinuations** in ENTR-601-44 or ENTR-601-45
- No clinically relevant changes in renal markers in ENTR-601-44 or ENTR-601-45
- Statistically significant improvement in functional benefit** observed in ELEVATE-44-201 Cohort 1
- **Ability to dose escalate** with Cohort 2 enrollment complete and dosing ongoing for both studies
- ELEVATE-44-201 cleared to dose participants in Cohort 3 at highest planned dose of 18 mg/kg

Near-Term Clinical Catalysts

ENTR-601-44 (DMD)	• Cohort 1 (6 mg/kg) OL dosing complete; All participants rolled into LTE study • **OL data expected to be received by year-end 2026** • Cohort 2 (12 mg/kg) enrollment complete; **Data expected in Q1 2027**
ENTR-601-45 (DMD)	• Cohort 1 (5 mg/kg) OL dosing ongoing • Cohort 2 (10 mg/kg) enrollment complete; **Data expected in H1 2027**
VX-670 (DM1)	• Vertex to share Phase 1/2 MAD **interim results in H2 2026**

All references in this presentation regarding planned regulatory filings and clinical study designs are subject to discussion with regulatory authorities; **EEV**: Endosomal Escape Vehicle; **PMO**: Phosphorodiamidate morpholino oligomer; **DMD**: Duchenne muscular dystrophy; **DM1**: Myotonic dystrophy type 1; **MAD**: Multiple ascending dose; **OL**: Open-Label; **LTE**: Long-term extension; *Accelerated Approval is subject to regulatory feedback; ****Time to Rise velocity (TTRV)**, nominal p-value, *post hoc* analysis.


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ELEVATE-45-201

ELEVATE-45-201

Cohort 1: Study Results Summary

Executive summary and key takeaways
ELEVATE-45-201 Cohort 1 results

ELEVATE-45-201 Cohort 1, with dosing at 5 mg/kg every 6 weeks, met the primary objective of safety and tolerability; Secondary objectives met guidance and are promising

Safety, Tolerability and PK

Generally well tolerated at 5 mg/kg every 6 weeks

- Mostly mild to moderate TEAEs, no discontinuations[1]
- Renal markers were generally within normal range and treated participant observations were comparable to placebo
- Plasma exposures match juvenile NHP predictions
- PK projects meaningful dystrophin increases in future Cohorts

Exon Skipping, Dystrophin and Function

- Exon skipping and dystrophin increased in treated participants when compared with baseline, as expected
- Given participants' age and baseline function, no measurable functional benefit was expected or observed

Ongoing Activity and Next Steps

Cohort 1 (5 mg/kg)	• All participants enrolled in 6-dose open-label period assessing long-term function and safety
Cohort 2 (10 mg/kg)	• Enrollment complete, dosing ongoing • **Data readout H1 2027**
Cohort 3 (up to 15 mg/kg)	• Expected to follow

[1]One participant experienced two serious TEAEs (vomiting and pyrexia); both subsequently resolved and the participant received all doses. **PK**: Pharmacokinetics; **TEAE**: Treatment-emergent adverse event; **NHP**: Non-human primate.

ELEVATE-45-201

Cohort 1: Trial Design and Treatment Population

ELEVATE-45-201 is designed to support U.S. Accelerated Approval and forms the basis of a global registrational program*

ENTR-601-45

ELEVATE-45-201 is a global, two-part, randomized, double-blind placebo-controlled Phase 1/2 study in ambulatory participants*

Primary objective: Safety and tolerability of ENTR-601-45

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 45 skipping amenable

Muscle biopsy #1

Part A

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

3:1 randomization in each cohort (ENTR-601-45 to placebo)

- ✓ **Cohort 1: 5 mg/kg or placebo**
- Cohort 2: **10** mg/kg or placebo
- Cohort 3: **≤15** 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-601-45 at the level that was administered in participant's Part A cohort
- 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 objective (U.S. confirmatory study/EU registrational study); Study participants are eligible to participate in a long-term extension platform study (ENTR-DMD-202) following completion of the open-label dosing period or expansion cohort.

Demographics and baseline characteristics

ELEVATE-45-201

Baseline Characteristics	Placebo n=2	ENTR-601-45 5 mg/kg n=6
Age, mean, years	9.0	6.3
Body mass index, mean, kg/m^2	19.75	16.62
Age at diagnosis, mean, years	4.0	1.9
Corticosteroid use, n (%)	2 (100%)	6 (100%)
Ambulatory, n (%)	2 (100%)	6 (100%)
Baseline dystrophin, mean, %	0.34%	0.49%

Dystrophin measured by western blot and normalized to MHC; Dystrophin values are actual observed values, including values <LLOQ; Two participants in the ENTR-601-45 group had baseline dystrophin levels <LLOQ and >LOD; LLOQ for dystrophin was 0.25%; LOD for dystrophin was 0.1%; Biopsy performed at baseline; **LLOQ**: Lower limit of quantification; **LOD**: Limit of detection.

ELEVATE-45-201

Cohort 1: Safety Data

ENTR-601-45 generally well tolerated at 5 mg/kg dose

ELEVATE-45-201

Participants with ≥1 TEAE, n (%)	Placebo n=2	ENTR-601-45 5 mg/kg n=6
Any TEAE	2 (100%)	6 (100%)
TEAEs related to study drug	2 (100%)	4 (67%)
Serious TEAEs	0	1 (17%)
TEAEs leading to study discontinuation	0	0
TEAEs leading to death	0	0

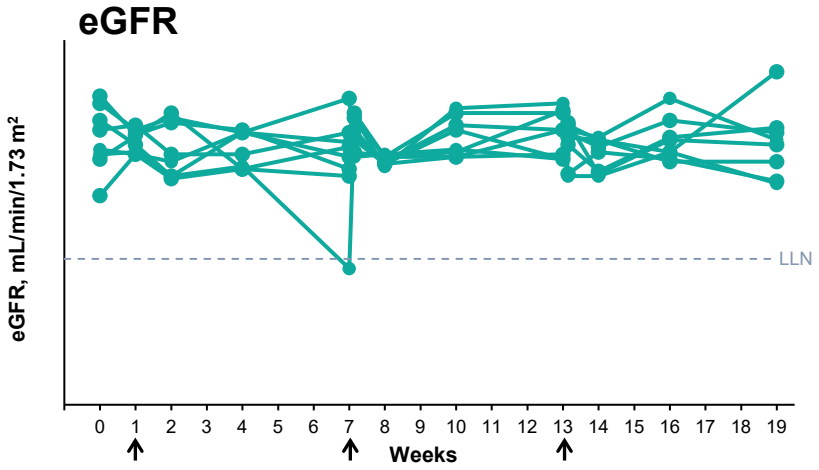
Most TEAEs were mild to moderate

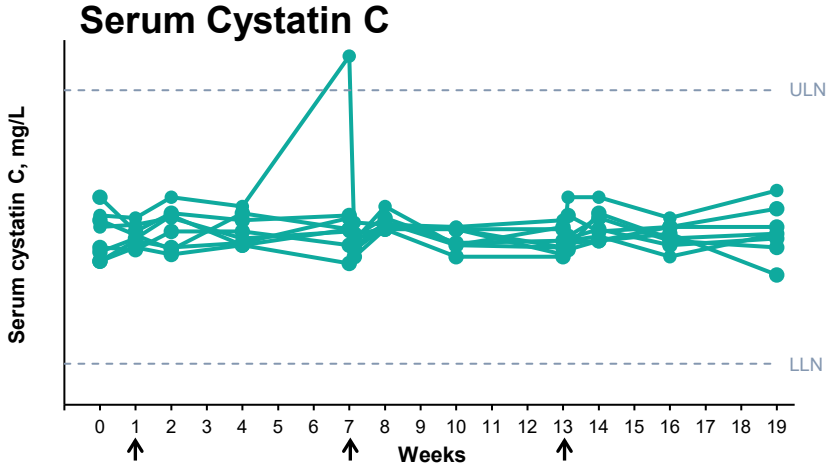
- Headache was the most common TEAE (5 events in 2 participants)
- One participant experienced 2 serious TEAEs (vomiting and pyrexia)
  - Both TEAEs resolved
- There were no study discontinuations due to any causes, and all participants enrolled in the open-label extension
- **No hypomagnesemia** or renal safety concerns were noted

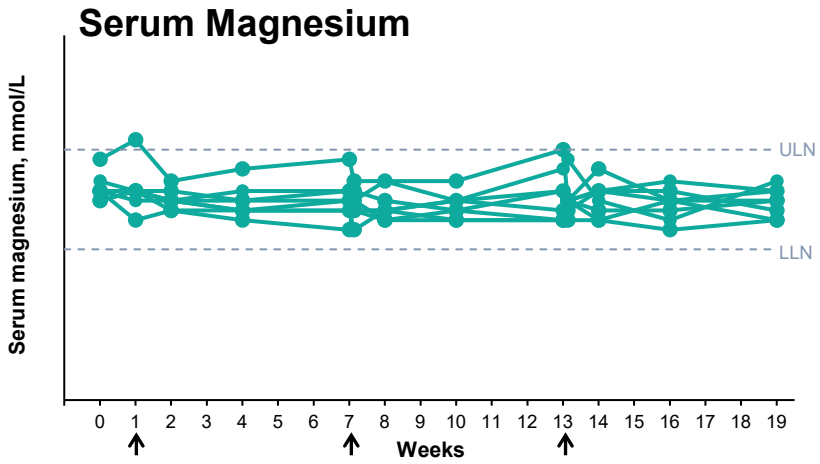
A favorable safety profile supports continued dose escalation

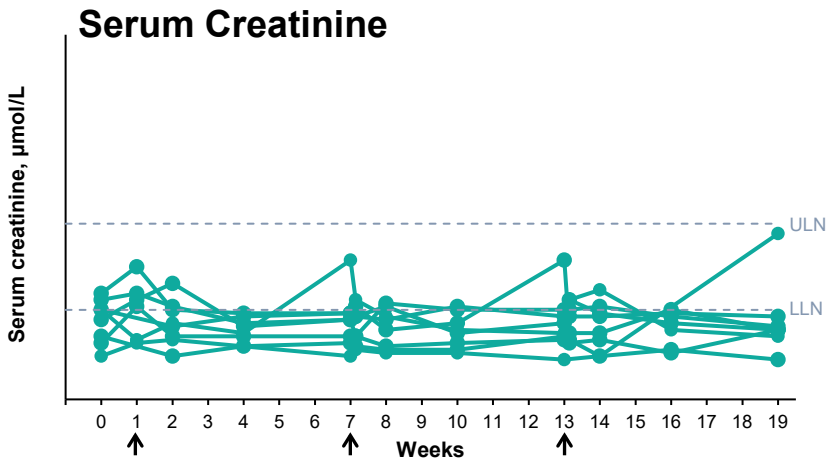
Renal markers were generally within normal range and treated participant observations were comparable to placebo

ELEVATE-45-201









Excursion outside normal ranges in eGFR and cystatin C occurred in a participant receiving placebo

↑ **Treatment**

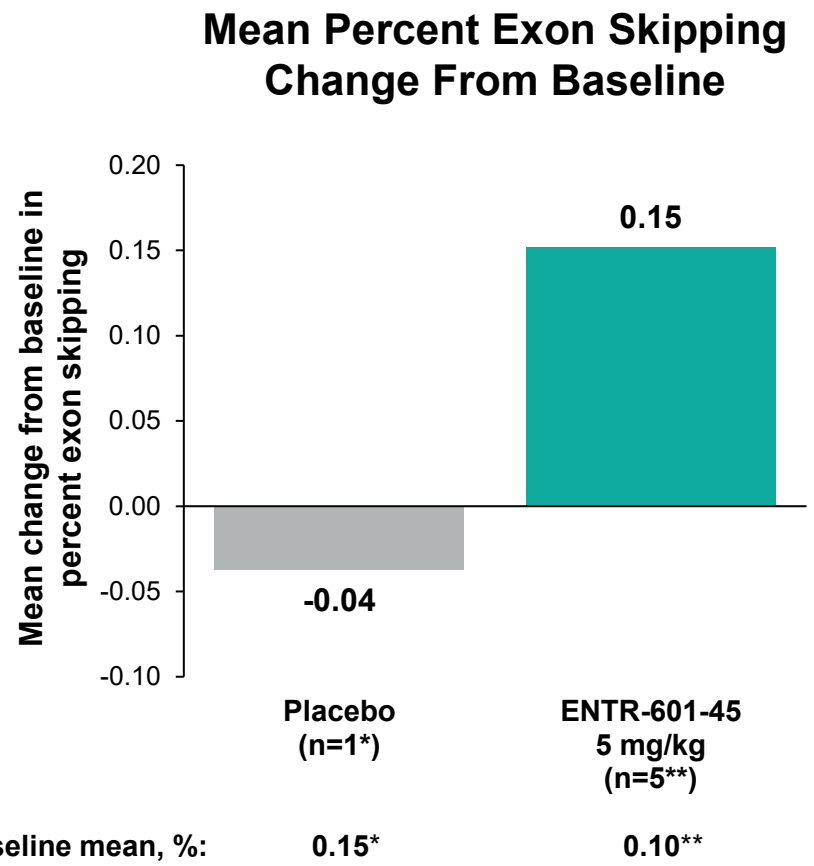
Arrowheads indicate dosing at nominal study visits; **eGFR**: Estimated glomerular filtration rate; **LLN**: Lower limit of normal; **ULN**: Upper limit of normal; Serum creatinine reference range depends on age of the participants enrolled in Cohort 1; Serum creatinine is below the reference range in individuals with DMD due to reduced muscle mass.

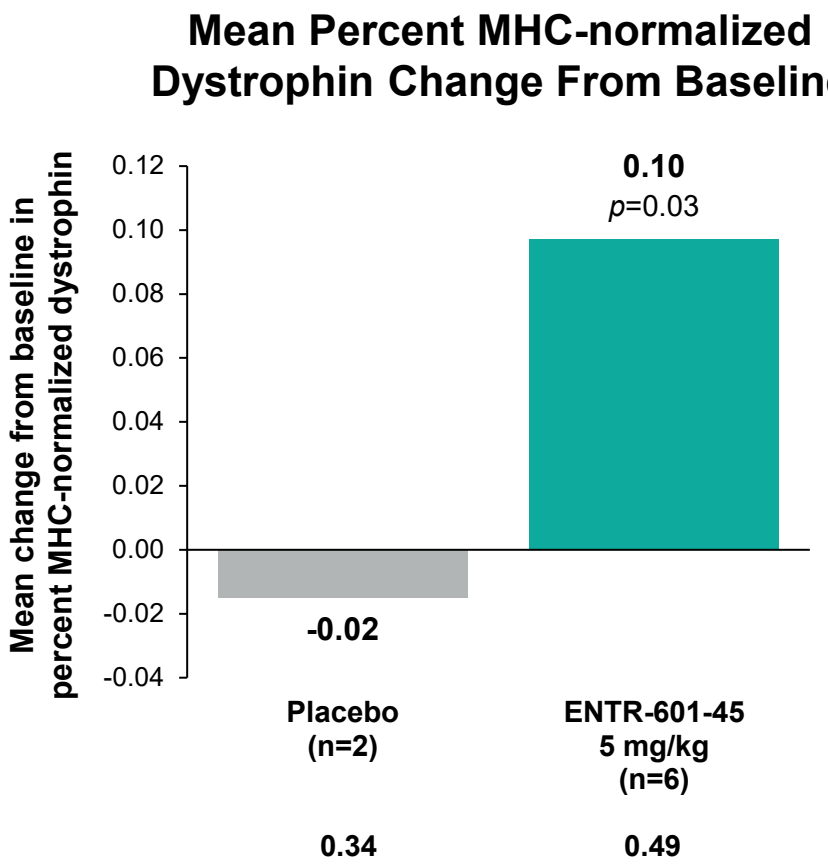
ELEVATE-45-201

Cohort 1: Efficacy Data

ELEVATE-45-201 Cohort 1
Exon skipping and dystrophin production

Cohort 1 exon skipping and dystrophin production change from baseline



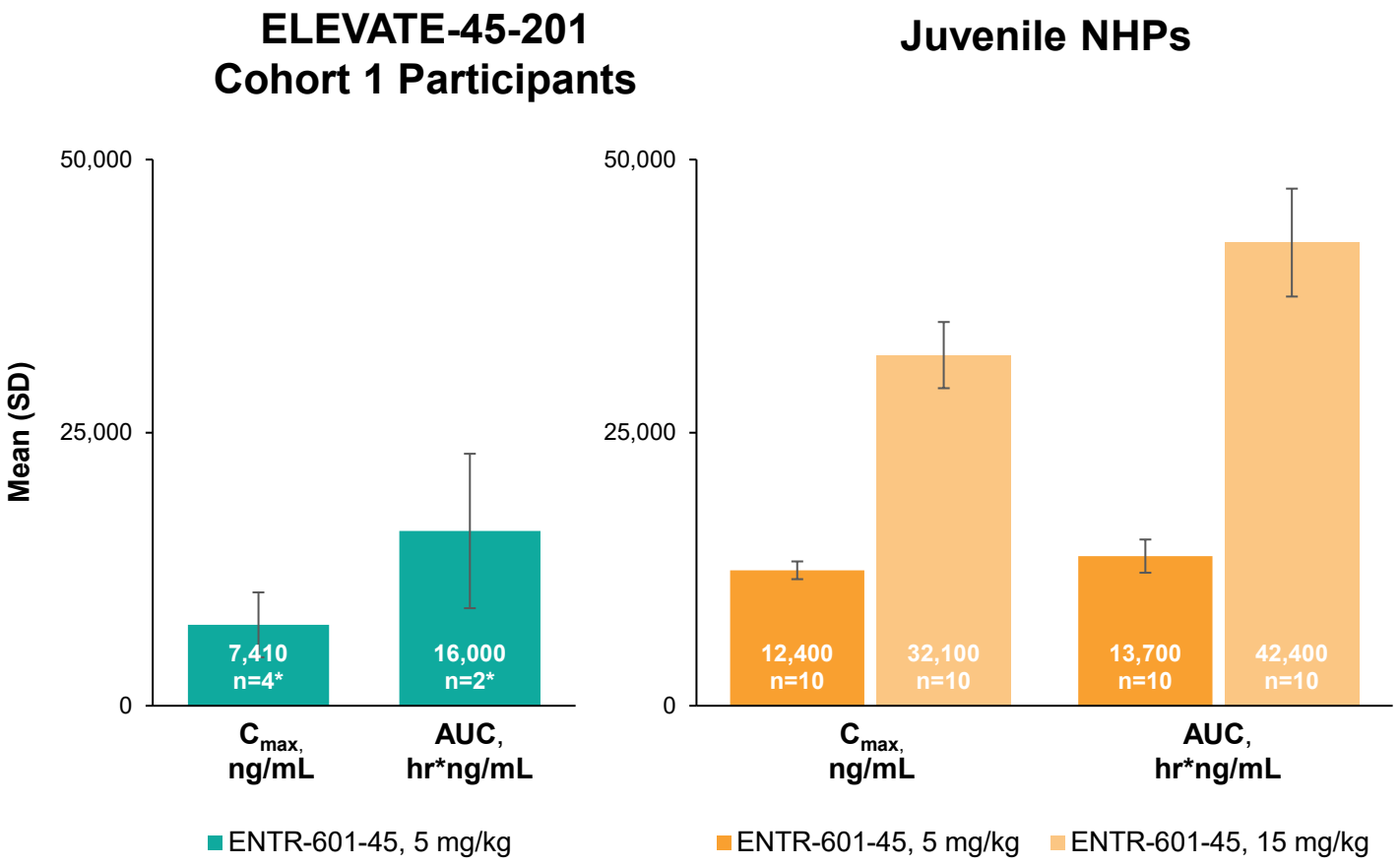


Baseline mean, %:	**0.15***	**0.10****
	0.34	**0.49**

Biopsy taken 6 weeks post last dose; Exon skipping measured by ddPCR; Dystrophin measured by western blot and normalized to MHC; Dystrophin values are actual observed values, including values <LLOQ; Two participants in the ENTR-601-45 group had baseline dystrophin levels <LLOQ and >LOD; LLOQ for dystrophin was 0.25%; LOD for dystrophin was 0.1%; Per the Statistical Analysis Plan, one sample permutation test (two-sided), hypothesis testing was only performed when n>5; Nominal *p* value; *n=2 at baseline and n=1 at week 19 due to skipped copy number at week 19 for one participant <LLOQ (LLOQ = 20 skipped copies); **One participant in the treated group had no baseline data due to sample quality issue; participant's skipped copy number at week 19 was <LLOQ (LLOQ = 20 skipped copies); **ddPCR**: Droplet digital polymerase chain reaction.

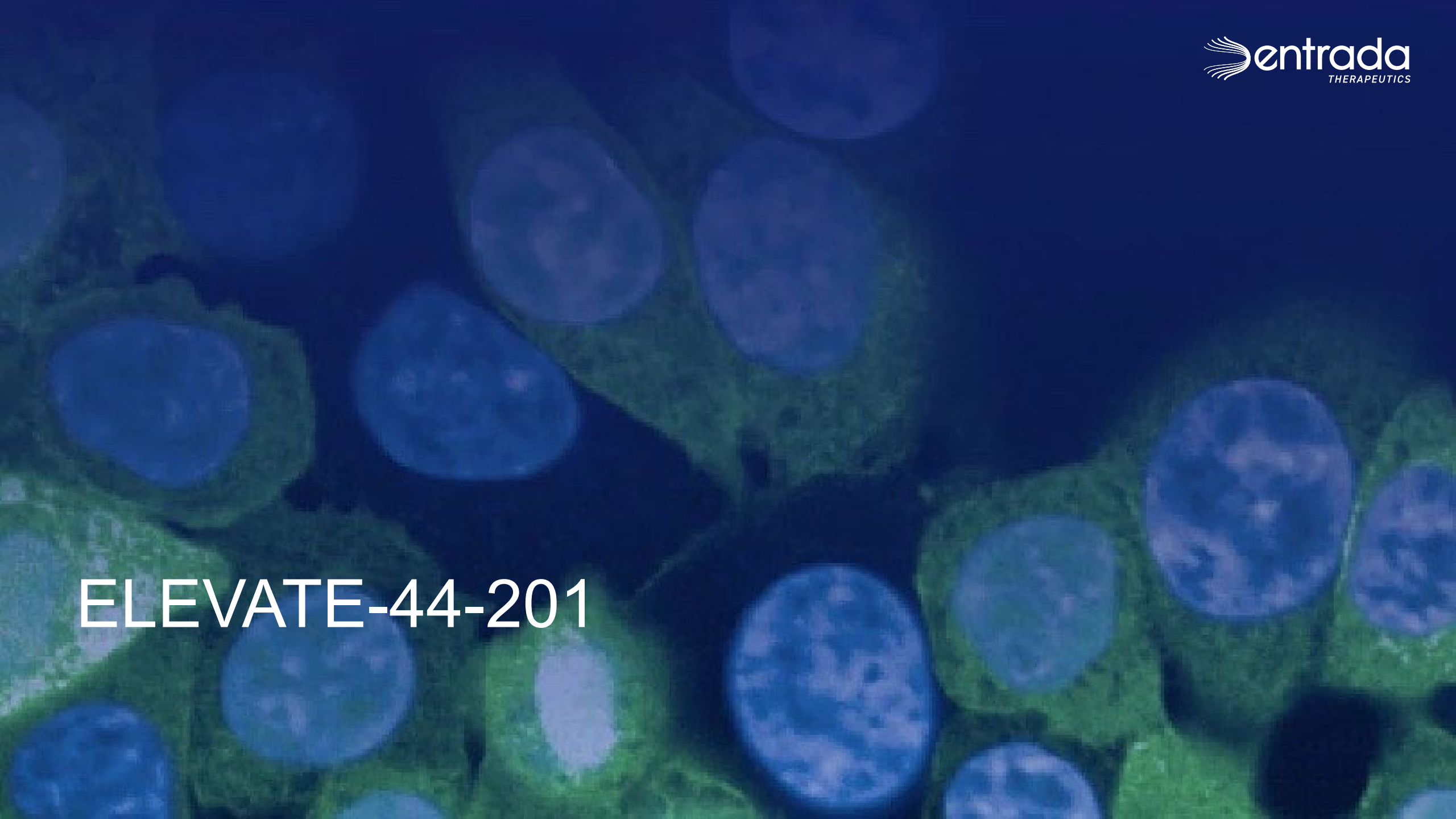
ELEVATE-45-201 Cohort 1 exposure compared to juvenile NHP

Cohort 1 exposure is consistent with juvenile NHP data and supports achieving target concentrations at higher doses



- **Exposures in Cohort 1 were comparable to juvenile NHPs at the same dose (5 mg/kg)**
- Supports use of NHP data to project pediatric exposure up to 15 mg/kg
- Juvenile NHP exposure dose-proportionally increased from approximately 5 to 15 mg/kg
- Assuming dose proportionality in ELEVATE-45-201, expected to realize meaningful exon skipping and dystrophin production in future Cohorts

*PK parameters could not be calculated in all 6 participants of Cohort 1 because PK was assessed using a sparse sampling schedule designed to limit blood draws and procedural burden for pediatric participants; Data reflect Day 1 PK/TK parameters; Juvenile NHP data are from 55-week repeat-dose study; **AUC**: Area under the concentration-time curve; **C_{max}**: Maximum concentration; **TK**: Toxicokinetic.


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ELEVATE-44-201

Cohort 1: Study Results Summary

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

Cohort 1 achieved the objective of demonstrating safety/tolerability; Functional benefit observed in Cohort 1 at the lowest dose, with dystrophin levels expected to increase in Cohorts 2 and 3

Safety and Tolerability

No SAEs; All AEs mild to moderate at 6 mg/kg

- No AEs leading to discontinuation
- Renal markers within normal range and comparable to placebo

Functional Benefit

Statistically significant* TTR and TTRV gains vs placebo

- TTR is a secondary objective of ELEVATE-44-201
- Cohort 1 resulted in a statistically significant* impact on TTR and TTRV versus placebo
- TTRV levels were several-fold above 12-month MCID after only 3 doses

Next Catalysts

Dystrophin expected to increase in Cohorts 2 and 3

- Continued expectation of safety margins at higher doses
- Continued benefit in muscle function expected, Cohort 1 open-label data received by year-end 2026
- Double-digit dystrophin expectation starting in Cohort 2 at 12 mg/kg with data in Q1 2027

Cohort 1 functional benefit response is an early and promising signal

SAEs: Serious adverse events; **AE**: Adverse event; **TTR**: Time to rise; **MCID**: Minimal clinically important difference; *Wilcoxon rank-sum (one sided) test, nominal p-values, *post hoc* analysis.

ELEVATE-44-201

Cohort 1: 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 participants*

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

3:1 randomization in each cohort (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
- 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 objective (U.S. confirmatory study/EU registrational study); Study participants are eligible to participate in a long-term extension platform study (ENTR-DMD-202) following completion of the open-label dosing period or expansion cohort.

Demographics and baseline characteristics

ELEVATE-44-201

Baseline Characteristics	Placebo n=2	ENTR-601-44 6 mg/kg n=6
Age, mean, years	13.5	9.3
Body mass index, mean, kg/m^2	17.96	20.00
Corticosteroid use, n (%)	2 (100%)	6 (100%)
Ambulatory, n (%)	2 (100%)	6 (100%)
Baseline dystrophin, mean, %	4.58%	4.00%
Time to Rise Velocity, mean (rises/second)[1]	0.191	0.126

Dystrophin measured by western blot and normalized to myosin heavy chain; Biopsy performed at baseline.[1]Time to Rise Velocity is calculated as 1/time-to-rise in seconds as an early prognostic factor for disease progression and loss of ambulation; Lower baseline number = worse.

ELEVATE-44-201

Cohort 1: 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

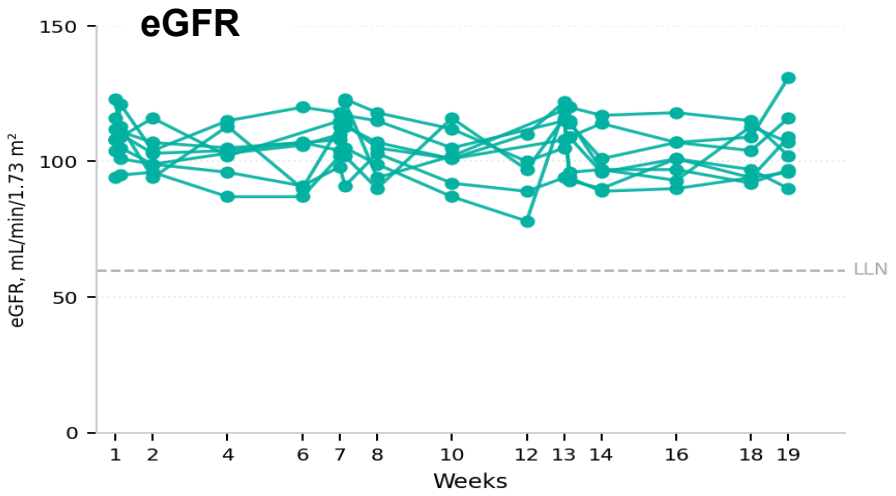
All TEAEs were mild to moderate

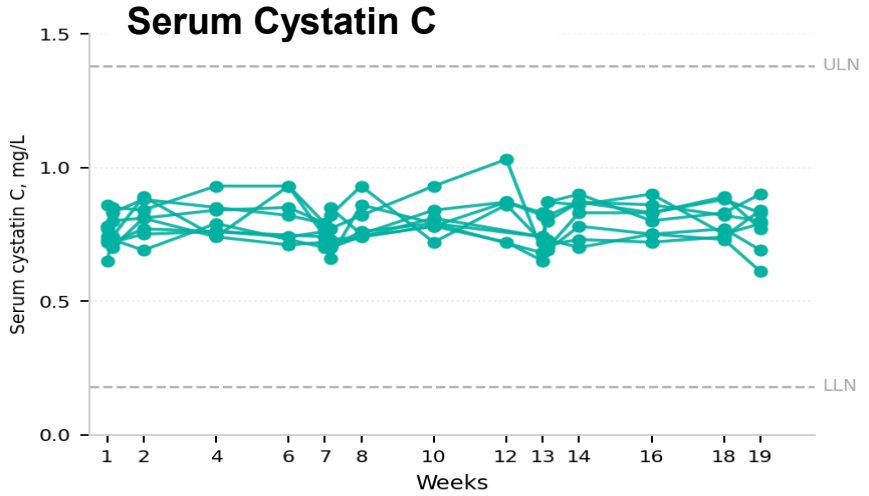
- Headache was the most common study drug–related TEAE, reported in 50% of the treatment group and 50% of the placebo group
- All events resolved
- **There were no serious TEAEs** and **no study discontinuations**
- **No hypomagnesemia** or renal safety concerns were noted

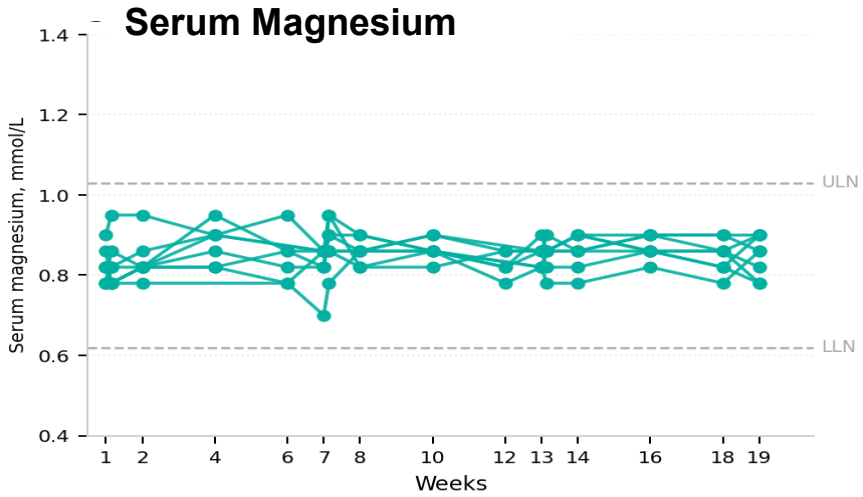
A favorable safety profile supports a de-risking of DMD programs

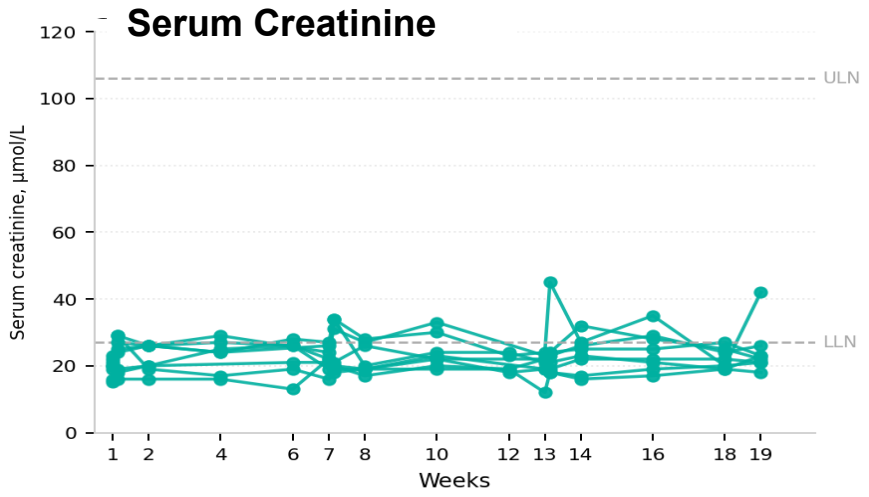
Renal markers were generally within normal range and treated participant observations were comparable to placebo

ELEVATE-44-201









Renal marker results further de-risk current and future DMD programs and support dose escalation to drive a higher therapeutic index

Data from ELEVATE-44-201; 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 participants with DMD due to reduced muscle mass.

ELEVATE-44-201

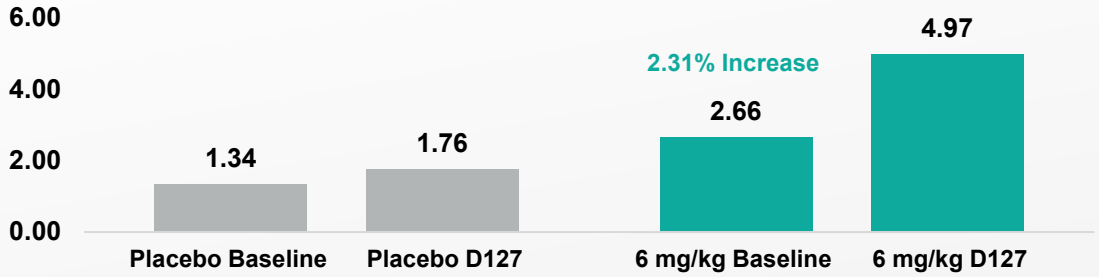
Cohort 1: Efficacy Data

ELEVATE-44-201 Cohort 1 efficacy summary

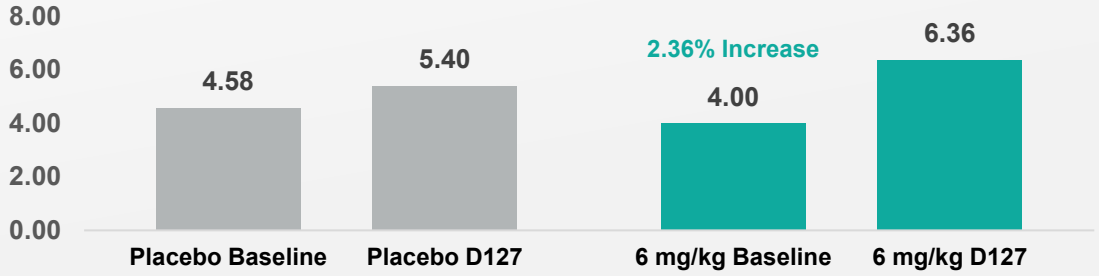
Cohort 1 achieved functional benefit via statistically significant measurements in both TTR and TTRV; 10MWR trending favorably with additional metrics expected to be received by year-end 2026

Exon Skipping and Dystrophin

Mean percent exon skipping



Mean percent MHC normalized dystrophin



Functional improvement

Improvement in both TTR and TTRV was seen across the majority of treated participants, irrespective of age

- Statistically significant* TTR improvement versus placebo of 2.40 seconds
- Statistically significant* TTRV improvement versus placebo of 0.09 rises per second
- Statistically significant correlation** between the change from baseline in TTRV and change from baseline in MHC normalized dystrophin levels
- Positive trends observed in 10MWR
- Open-label data expected to be received by year-end 2026

*Wilcoxon rank-sum (one sided) test, **Spearman Correlation, nominal p-values, *post hoc* analysis; **10MWR**: 10-meter walk/run; **MHC**: Myosin heavy chain.

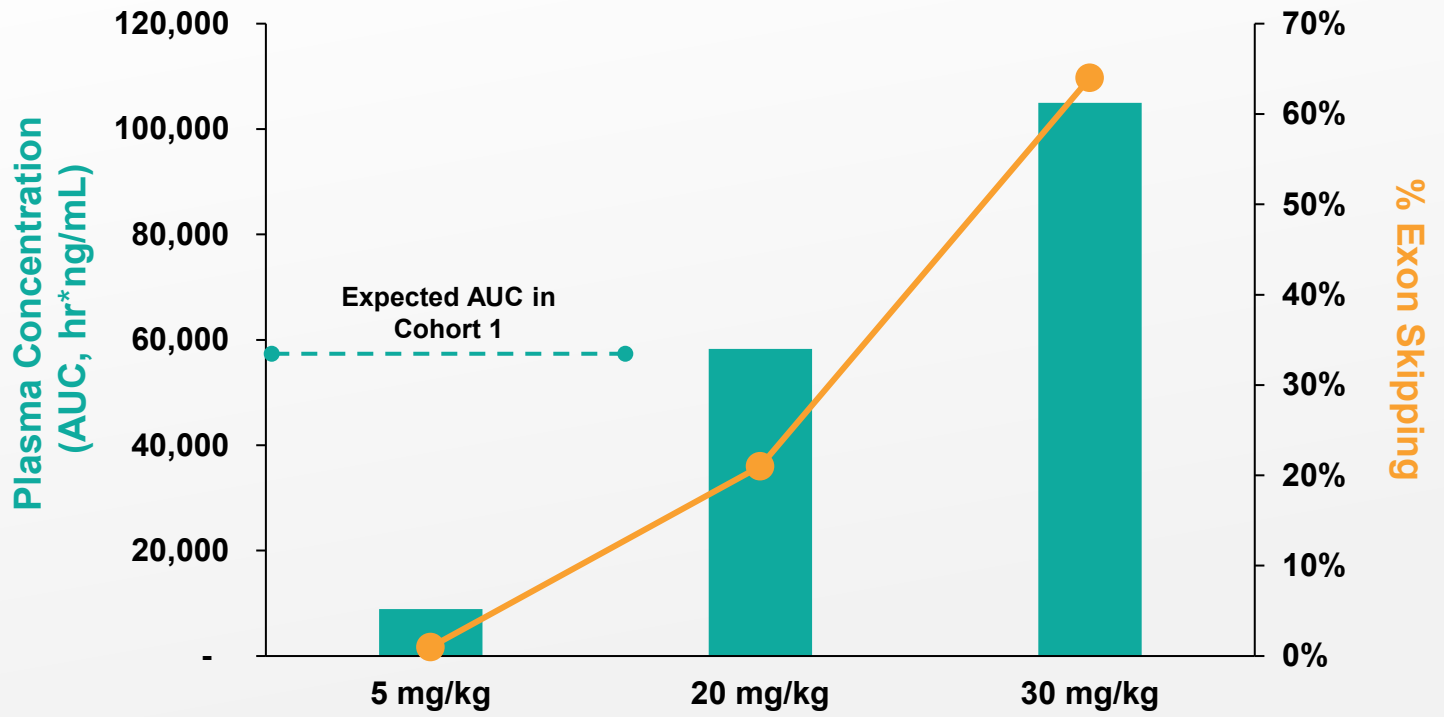
ELEVATE-44-201 expectations
Projecting Cohorts 2 and 3 based on juvenile NHP data

Juvenile NHP data suggest a right-shifted double-digit dystrophin expectation starting at 12 mg/kg in Cohort 2 and continuing through 18 mg/kg in Cohort 3

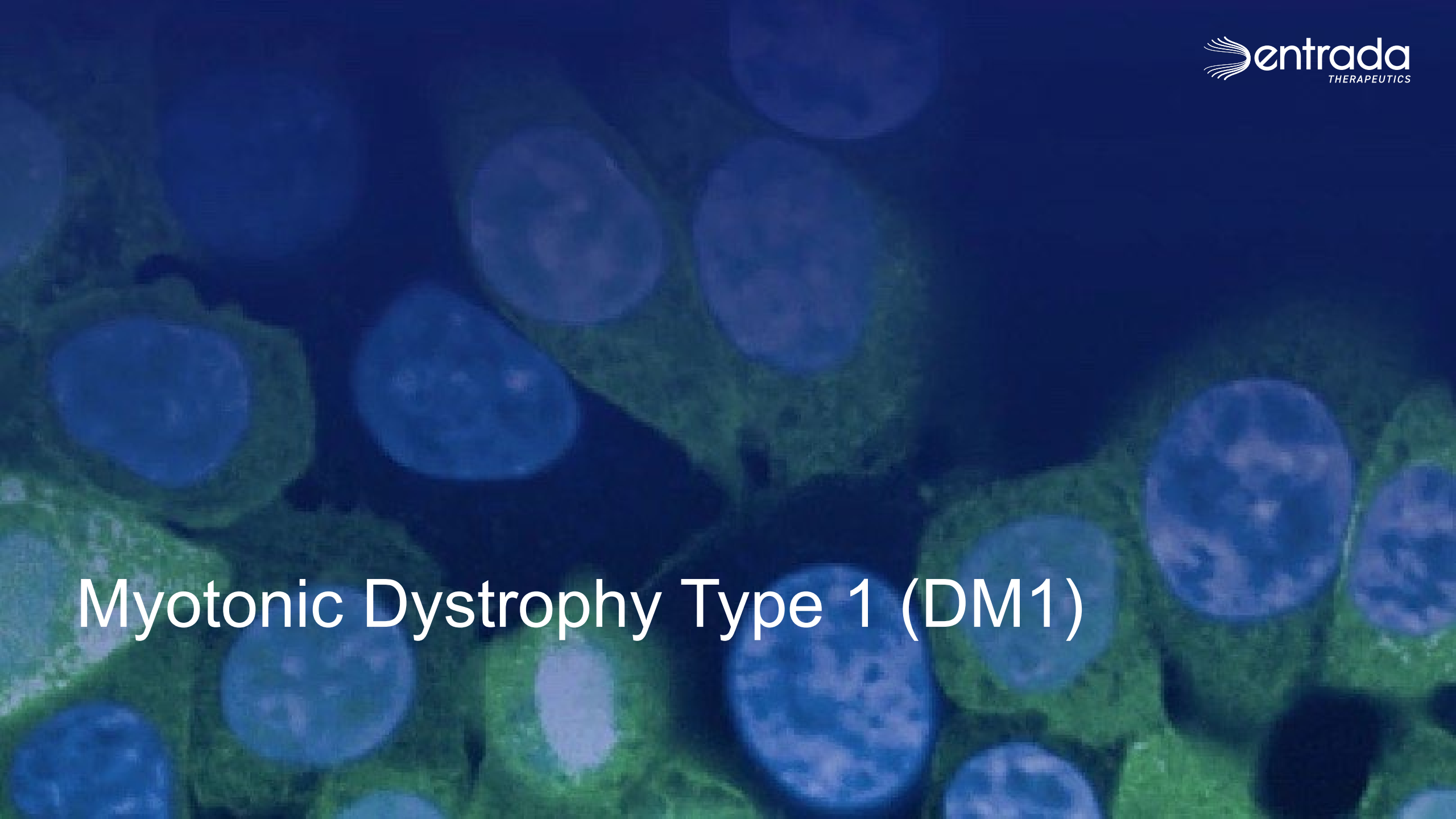
Key Expectations

- Exposure to exon skipping relationship remains unchanged – higher exposure leads to higher exon skipping
- Model projections indicate substantially increased exon skipping and dystrophin expression expected in Cohorts 2 and 3

Juvenile NHP Plasma Concentration and Exon Skipping



NHP data from 55-week, Q6W, 10 doses, chronic juvenile toxicology study.



Myotonic Dystrophy Type 1 (DM1)

Vertex DM1 partnership: Phase 1/2 readout expected in second half of 2026

No disease-modifying treatments are currently available for the more than 110,000 people in the U.S. and Europe living with DM1

Vertex completing a placebo-controlled Phase 1/2 clinical program at over 25 global sites

- Enrollment and dosing complete in the MAD portion of the GALILEO Phase 1/2 trial; Interim results expected in the second half of 2026
  - Assessing safety and preliminary efficacy, including change from baseline in splicing index and measures of muscle function and strength (vHOT and QMT)
- Open-label extension study is enrolling
- Same EEV utilized for VX-670 as in DMD franchise

VX-670 differentiation

- Unlike antibody-based approaches, VX-670's targeted and specific blocking of pathogenic CUG repeats drives global transcriptome correction while preserving healthy DMPK levels
- Satellite cell uptake may improve outcomes as CUG repeat length correlates with progenitor cell activation, proliferation and differentiation*

Significant upfront and potential future economics associated with the partnership

- Entrada received an upfront payment of $224M and an equity investment of $26M upon initiation of the collaboration
- Agreement provided for up to $485M for the achievement of milestones
- Tiered royalties on future net sales

*Hasuike et al., Front. Genet., March 2022; **CUG repeat**: Repeating sequence of cytosine, uracil and guanine in RNA; **vHOT**: Video hand opening time; **QMT**: Quantitative muscle testing; **DMPK**: Dystrophia myotonica protein kinase.



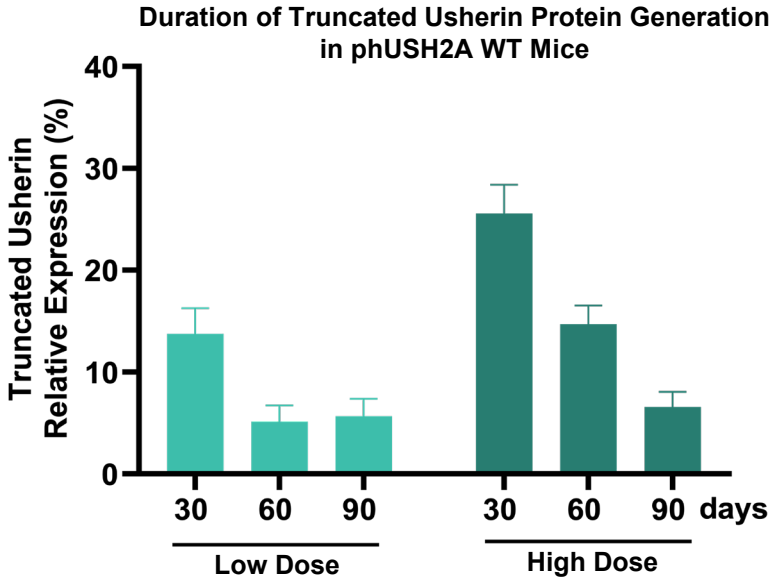
Inherited Retinal Diseases (IRDs)

Addressing areas of high unmet need in IRDs with new oligonucleotide-based therapeutics

Entrada's first IRD clinical candidate, ENTR-801, will target Usher syndrome type 2A (USH2A); Addressable population is ~15,000 patients* in the U.S. and Europe with no approved therapeutics

Usher Syndrome Type 2A (USH2A)

- An inherited eye condition caused by variants in the USH2A gene
- In approximately 30-35% of patients, mutations in exon 13 prevent production of the usherin protein
- Without usherin, photoreceptors (the light-sensing cells in the eye) progressively degenerate
- Lack of usherin leads to vision loss in early adulthood and legal blindness in mid-adulthood
- **No disease-modifying treatments are currently available that can slow or stop disease progression**



About ENTR-801

- Proprietary exon 13 skipping therapy designed to restore functional usherin protein production with the goal of preserving photoreceptor health and function
- Demonstrated robust exon skipping and truncated usherin protein production, with potential for quarterly IVT dosing
- IND-enabling studies have been initiated
- **~15,000 patients in the U.S. and Europe are exon 13 skipping amenable***

Note: **IRDs**: Inherited retinal diseases; *Delmaghani et al., Hum Genet., Apr. 2022; Sanjurjo-Soriano et al., HGG Advance, Oct. 2023; Su et al., Front Aging Neurosci., Aug. 2022; **IVT**: Intravitreal.

2026 Inflection Points

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

ENTR-601-44

Cohort 1 safety, dystrophin and functional improvement

Cohort 3 to initiate at top planned dose of 18 mg/kg

Cohort 1 OL data expected to be received by YE 2026

Cohort 2 data expected in Q1 2027

ENTR-601-45

Cohort 1 safety, tolerability and dystrophin reported

All study participants progressed to Cohort 1 OL

Cohort 2 enrollment complete at 10 mg/kg; Data expected H1 2027

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

Completed studies supporting Clinical Trial Authorization (CTA)

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

VX-670

Global Phase 1/2 program

Vertex is on track to share interim results in H2 2026

ENTR-801

Initial IRD candidate focused on Usher syndrome type 2A

Second IRD candidate nomination by year-end 2026

Pipeline Expansion

Next-generation EEVs for neuromuscular expansion

Ocular expansion into larger disease areas

Range of undisclosed diseases and modalities

Cash runway into Q4 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 ~$193M in cash, cash equivalents and marketable securities as of September 30, 2026.

Appendix

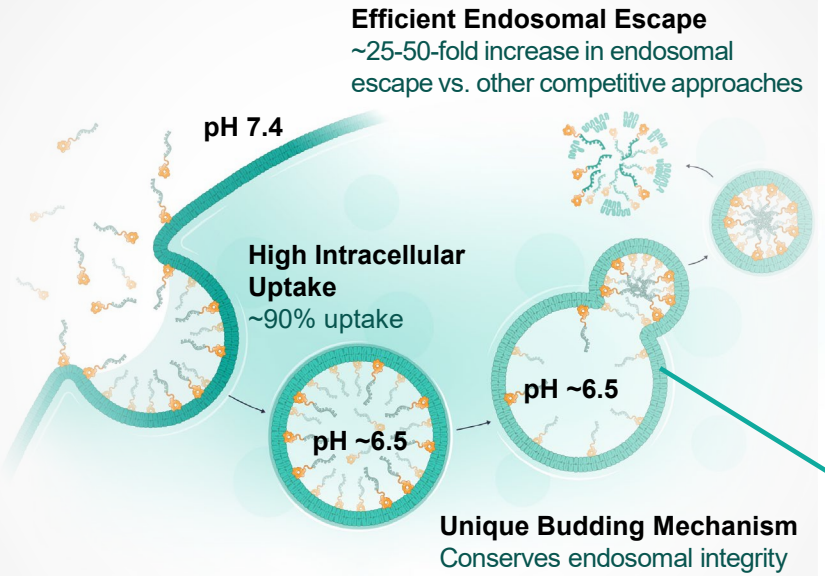
EEV™ Platform

Understanding EEV endosomal escape advantages

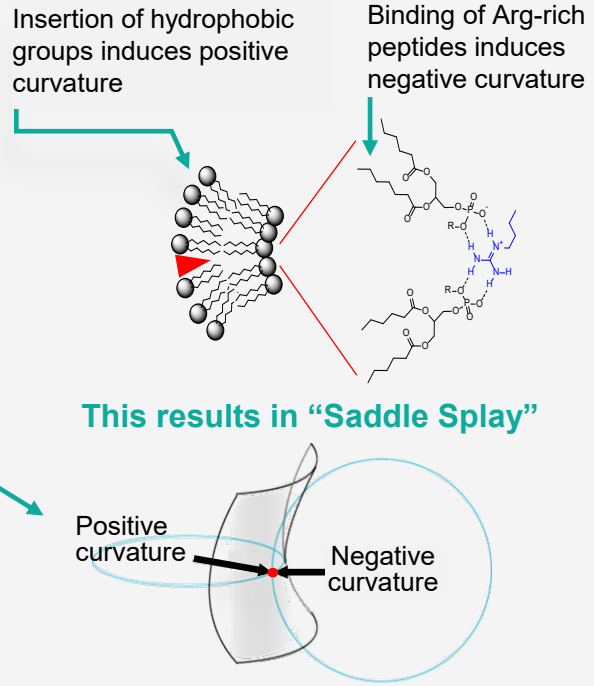
Unique pH-dependent membrane binding affinity enhances EEV endosomal escape

- EEVs have a higher affinity to phospholipids compared to traditional linear CPPs; The interaction of EEVs with phospholipids is pH-dependent
- The specific interaction of EEVs with the endosomal membrane facilitates the escape of EEVs from early endosomes, as demonstrated by the correlation between endosomal escape efficiency and endosomal membrane binding affinity

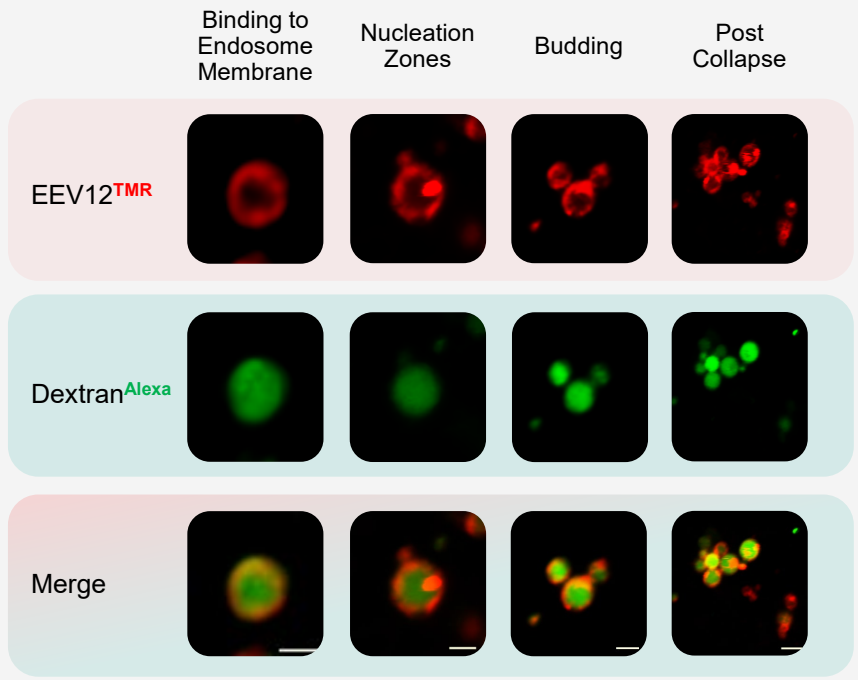
Concept



Mechanism



Visualization

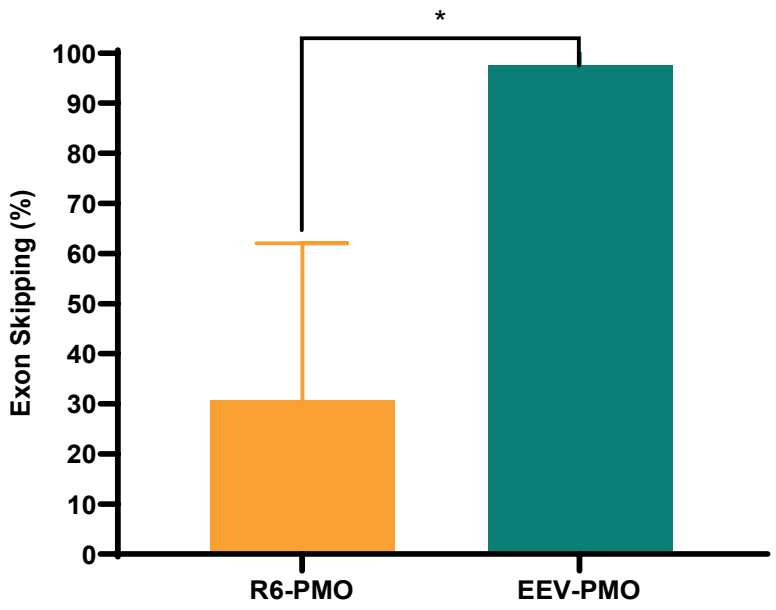


Sahni, A. et al. ACS Chem. Biol. 2021; **CPP**: Cell-penetrating peptide.

Entrada optimizes both the EEV delivery vehicle and the active conjugate to create best-in-class medicines

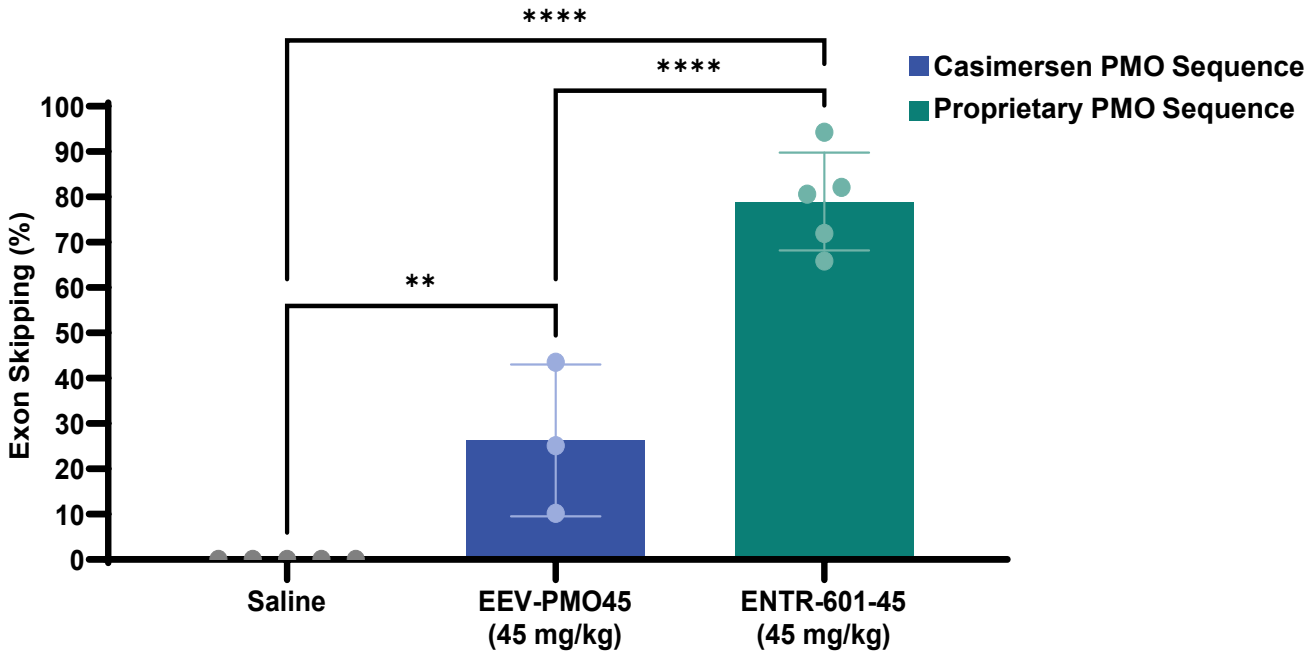
In the below DMD example, Entrada has optimized the EEV and the PMO sequence; The data represents the superiority of each component against other approaches and sequences

EEV Optimization



- PMO sequences are the same, cell penetrating peptides are different (linear vs. cyclic)
- EEV-PMO significantly improved exon skipping after 3 days in *mdx* mice as compared to competitive R6-PMO

PMO Optimization



- EEVs are the same, PMO sequences are different (casimersen vs. Entrada proprietary)
- Proprietary PMO significantly improved exon skipping after 3 days in *hDMD* mice as compared to competitive casimersen sequence

*p<0.05, **p<0.01, ***p<0.001, ****p<0.0001; relative to saline; Concentrations provided are PMO equivalent; Data presented at 2024 World Muscle Society conference.

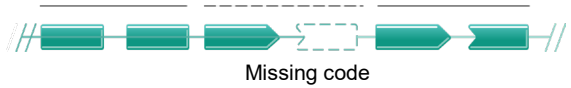
DMD Approach

Entrada is developing genetic medicines to correct the mechanism of disease and slow, halt or reverse progression

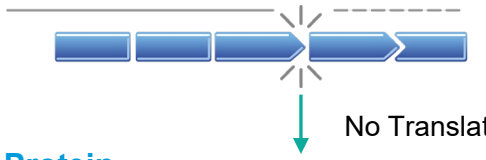
Entrada's Approach: Modulate RNA Splicing and Produce Functional Protein to Preserve and Rebuild Muscle

Individuals with Duchenne

Pre-mRNA
With exon deletion, protein program is miscoded



mRNA
Reading frame disrupted and body reads STOP!



Protein
Resultant mRNA sequence stops the production of functional dystrophin protein



Entrada Approach

Pre-mRNA
Treatment enables skipping of exon



Splice modulation

mRNA
Reading frame restored



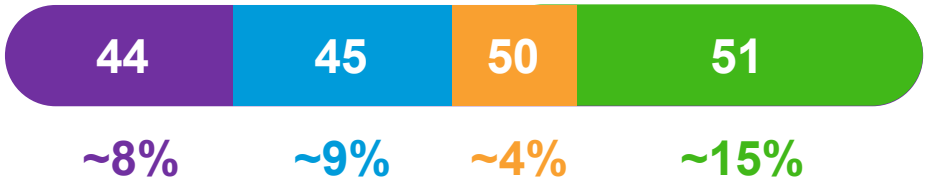
Translation

Protein
Functional dystrophin protein is translated and muscle membrane regains integrity



DMD Mutations and Tissues of Focus in the Entrada Portfolio





~35% = ~11,500 Patients*

Entrada addresses mutations which result in a lack of dystrophin, muscle breakdown, progressive disability and death

The portfolio covers ~35% of the total population totaling ~11,500 patients in the U.S. and EU*

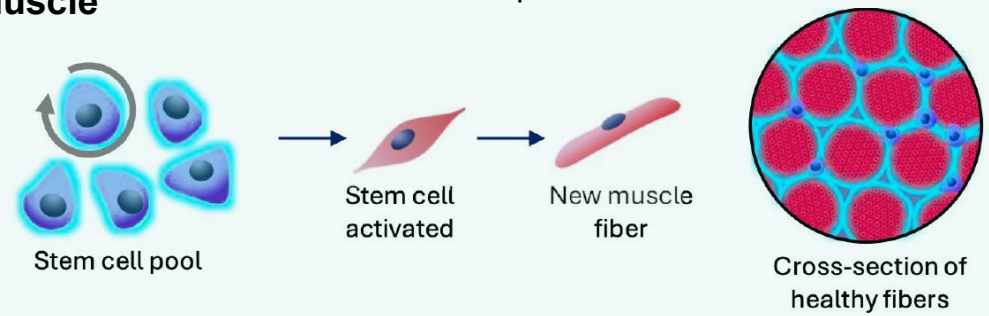
*Sub-mutation estimates rounded, from Bladen et al., HGVS Journal, January 2015; DMD population estimated to be ~7-8 per 100,000 males per Crisafulli et al., Orphanet J. Rare Disease, June 2020; Calculation based on estimates of U.S. and EU (defined as EU27 population).

The DMD challenge: Muscle damage and impaired repair

An ideal treatment is regenerative – replacing damaged, dystrophic muscle with healthy muscle; Requires satellite cell correction which enables asymmetric differentiation to develop healthy muscle

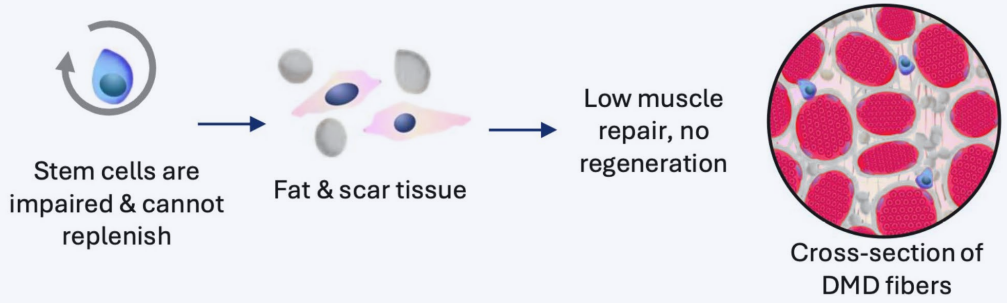
Healthy muscle

- Dystrophin supports stabilizing and repairing muscle fibers and stem cell replenishment and 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 results in a "double hit" and decline in muscle function, including:

1. Increased damage to existing muscle tissues
2. Inability to regenerate and repair with new healthy muscle fibers; Replaced with fat and fibrosis instead

Satellite (stem) cells require dystrophin to efficiently promote healthy 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

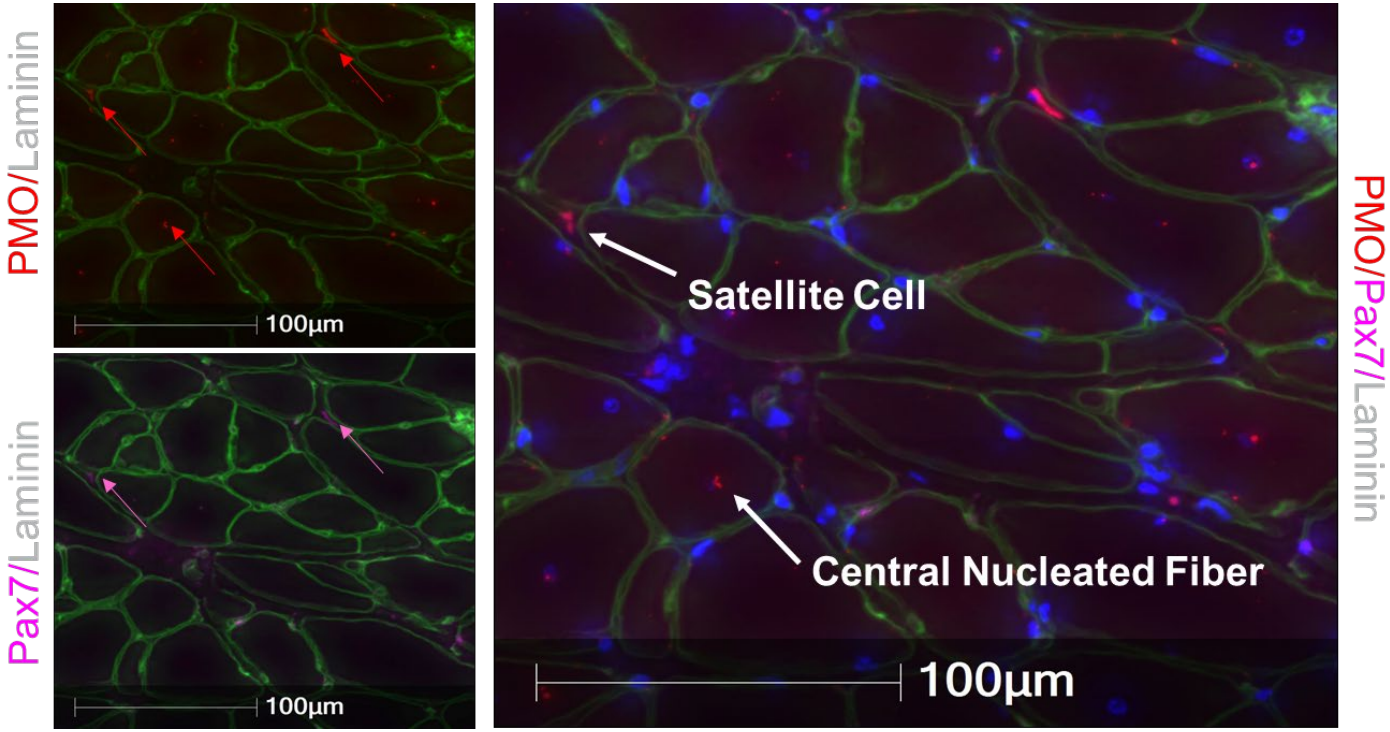
Enhanced satellite cell activity would result in the regeneration of healthy fibers and the stabilization of the overall muscle; In turn, this may convert into greater strength, as measured by TTRV

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

- Lack of transferrin receptor expressed on quiescent satellite cells so the antibodies cannot deliver PMO
- 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*: Dystrophic mouse model; 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; **Q6W**: Every 6 weeks.

Competitive Comparison

EEV therapies are expected to show superior dystrophin production and dose-dependent increases in therapeutic index

A 25-50-fold improvement in endosomal escape, novel PMO sequences, non-linear increases in PK/PD, satellite cell uptake and lower whole drug requirements compared to antibody-based therapies

	EEV-PMO **Entrada**	**Anti-TfR1-mAb-PMO (exon 44)** Novartis/Avidity	**Anti-TfR1-Fab-PMO (exon 51)** Dyne
Design	**Cyclic, arginine-light peptide** 2.6 kDa Tissue-specific delivery; Increases stability and half-life	**Full-length antibody** ~150 kDa TfR1 specific deliver; Non-cleavable linker	**Fragment antibody** ~50 kDa TfR1 specific delivery; Cleavable linker
Dose	✓ **Low conjugate doses** ~5 mg/kg PMO + 1 mg/kg EEV = **6 mg/kg whole drug (ELEVATE-44-201 Cohort 1 dose)**	– **Large conjugate doses** 5 mg/kg PMO (registrational dose) + ~23 mg/kg mAb = **~28 mg/kg whole drug dose**[1]	– **Large conjugate doses** 20 mg/kg PMO (registrational dose) + undisclosed mg/kg Fab = **undisclosed mg/kg whole drug dose**[2]
Uptake	✓ **No limit on dose response seen** **48.3%** increase in exon skipping from 3 to 6 mg/kg whole drug dose in HNV[3]	– **Apparent limit on dose response** No increase in dystrophin from 5 to 10 mg/kg PMO dose in DMD exon 44 patients: **25% vs. 26%**[4,5]	– **Apparent limit on dose response** No increase in dystrophin from 10 to 20 mg/kg PMO dose in DMD exon 51 patients: **2.97% vs. 3.14%**[2,4]
Target engagement	✓ **Enhanced endosomal escape** **~25-50-fold increase in endosomal escape** vs. other competitive approaches	– **No enhanced endosomal escape** Once dissociated from the mAb in the lysosome, the unconjugated PMO escapes at **<2% efficiency**[6]	– **No enhanced endosomal escape** Once dissociated from the Fab in the endosome, the unconjugated PMO escapes at **<2% efficiency**[6]

TfR1: Transferrin receptor 1; [1.]Etxaniz et al., Nucleic Acid Res., 2025; [2.]Dyne corporate materials from DELIVER Clinical Update investor call September 2025; [3.]Total mean exon skipping; Entrada internal data from Phase 1 SAD study of ENTR-601-44 in HNV; [4.]Unadjusted dystrophin, mean change from baseline; [5.]Avidity corporate materials from del-zota topline data investor call March 2025; [6.]Dowdy et al., Nucleic Acid Ther., 2022.

Supporting Clinical Information

Functional improvement
The importance and meaning of Time to Rise velocity

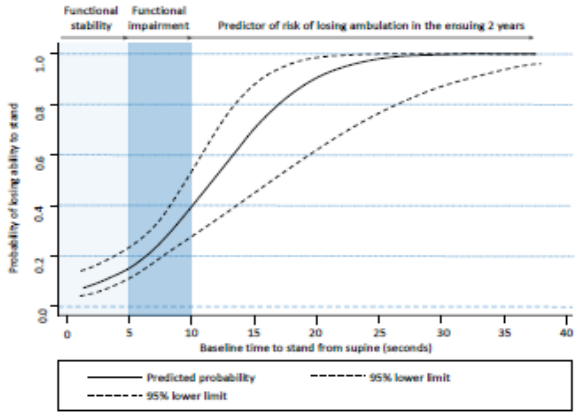
Time to Rise, a secondary objective in our ELEVATE trials, is a robust, prognostic factor that predicts disease progression; Time to Rise velocity reduces the impact of imputation, outliers and noise

- **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

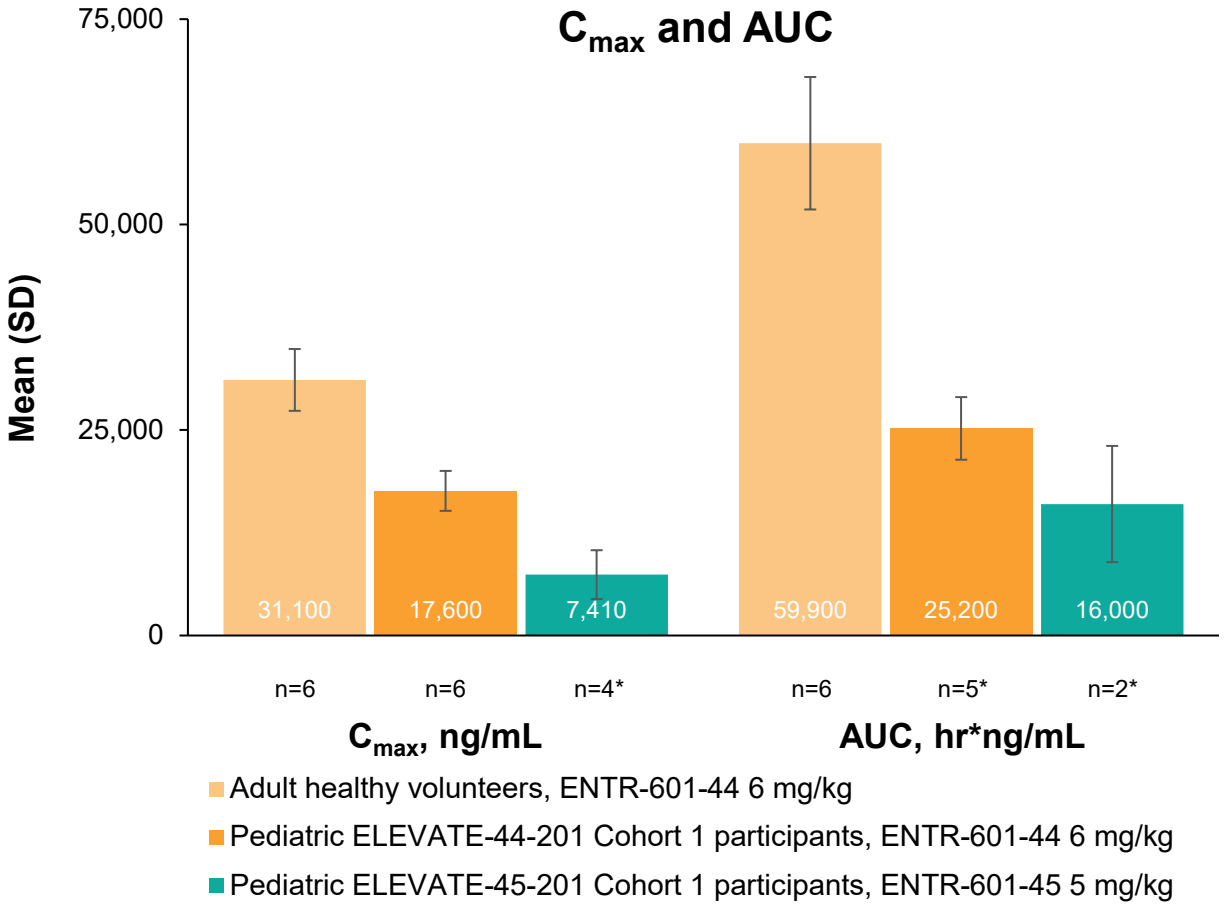
TTR baseline (seconds)	TTR end of study (seconds)	Improvement (seconds)	TTRV calculation	Δ TTRV (rises/sec)
6.0	3.89	2.11	1/3.89 – 1/6.0 = 0.257 – 0.167	**+0.09**
8.0	4.65	3.35	1/4.65 – 1/8.0 = 0.215 – 0.125	**+0.09**
10.0	5.26	4.74	1/5.26 – 1/10.0 = 0.190 – 0.100	**+0.09**

Explanation: A boy starting at a TTR of 6 seconds needs to improve by ~2 seconds to achieve +0.09 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.

ELEVATE-45-201 versus ELEVATE-44-201
Clinical PK parameters

As expected, pediatric plasma exposures are consistently lower than adult plasma exposures and appear to correlate to both age and weight



- Average treatment age of ELEVATE-44-201 Cohort 1 treated participants was 9 years while the average age of ELEVATE-45-201 Cohort 1 treated participants was 6 years (unusually young given enrollment criteria)
- In pediatric study participants, C_{max} was ~55% lower and AUC was 36% lower with ENTR-601-45 5 mg/kg versus ENTR-601-44 6 mg/kg
- Participants received 17% less drug given the difference between the ENTR-601-45 5 mg/kg and ENTR-601-44 6 mg/kg doses

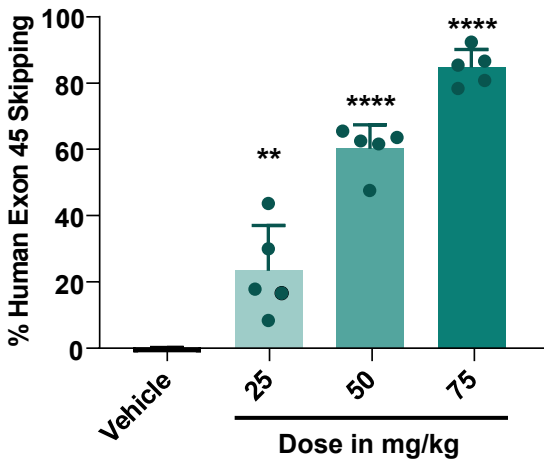
*PK parameters could not be calculated in all 6 participants of Cohort 1 of ELEVATE-44-201 or ELEVATE-45-201 due to sparse sampling.

DMD Preclinical Data

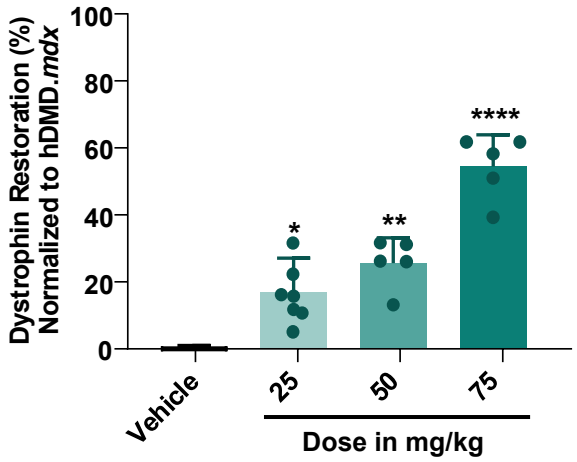
Preclinical data support potential for best-in-class clinical profile for ENTR-601-45

Dose-dependent increase in exon skipping and dystrophin expression correlates to complete functional correction

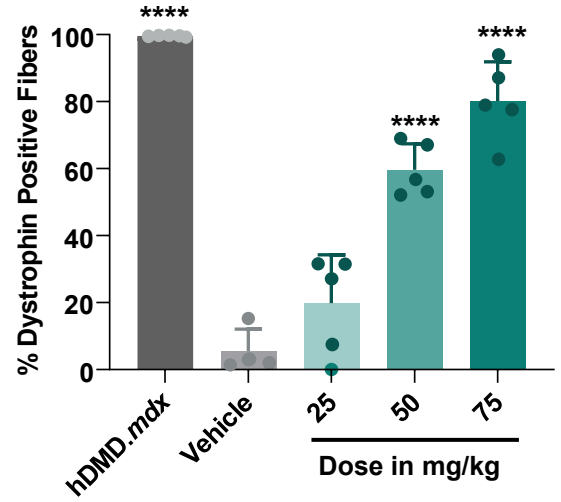
Exon Skipping

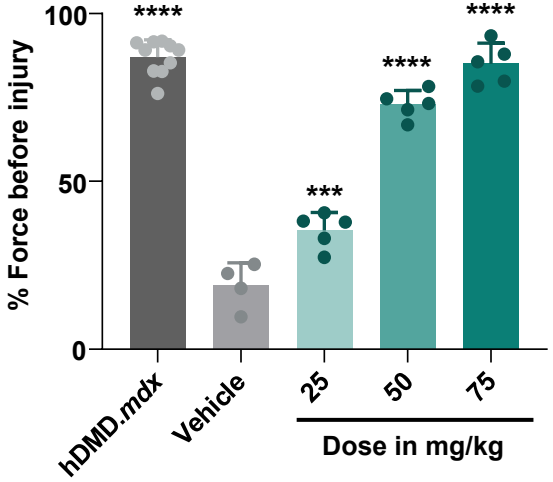


Dystrophin Restoration



Functional Correction





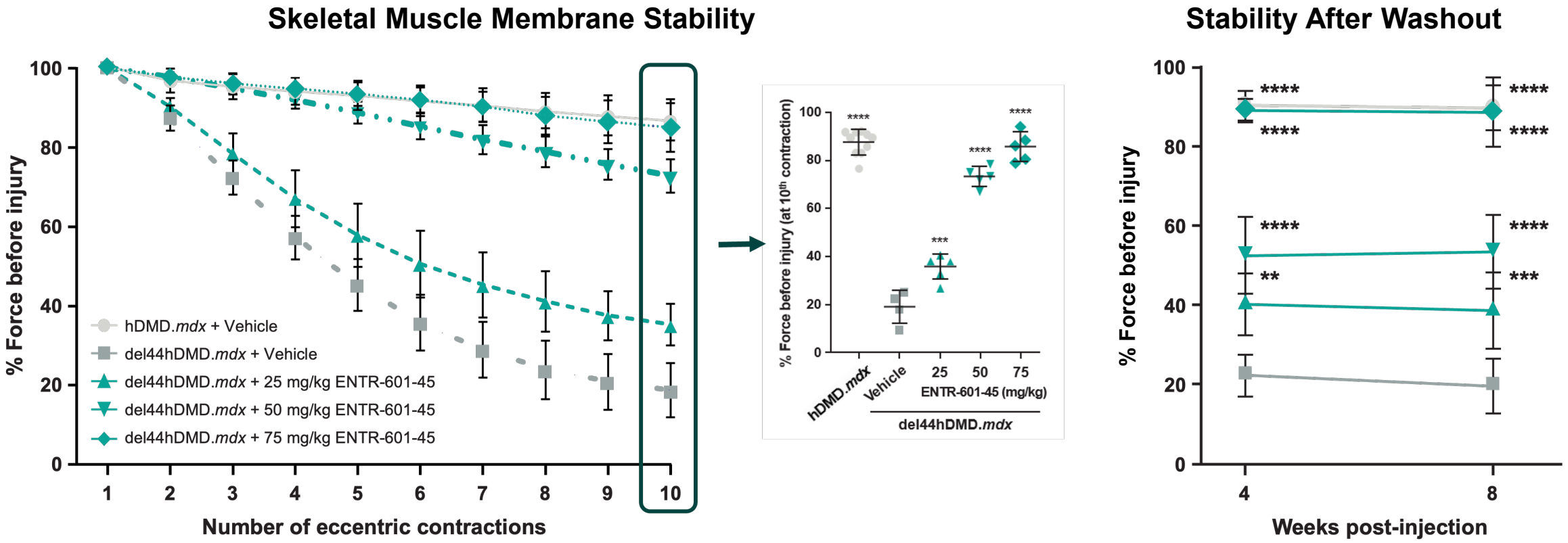
- Active and vehicle del44hDMD.*mdx* mice, n=5 per cohort, ENTR-601-45 (Q6W x 3 doses); Control saline treated hDMD.*mdx* mice, n=10 (Q6W x 3 doses)
- Skipping (ddPCR) and dystrophin production (JESS) is significantly increased 6 weeks after the third dose of ENTR-601-45 (gastrocnemius muscle shown)

Data are shown as mean ± SD; *p ≤ 0.05; **p ≤ 0.01; ***p ≤ 0.001; ****p ≤ 0.0001; 25 mg/kg correlates to ~5 mg/kg human equivalent dose (HED), 50 mg/kg correlates to ~10 mg/kg HED, 75 mg/kg correlates to ~15 mg/kg HED; Data presented at the 2024 World Muscle Society conference.

Dose-dependent and durable improvements in muscle function observed in del44hDMD.*mdx* mice

ENTR-601-45

Dose-dependent increase in percent force retention following 10 contractions; Increase maintained for at least 8 weeks after the third Q6W dose of ENTR-601-45



del44hDMD.*mdx* mice were treated with three Q6W IV injections of ENTR-601-45 or vehicle; ECC-induced muscle force loss generated by repeated eccentric force (ECC) contraction of the gastrocnemius muscle was assessed 5 weeks (left/center) or 4 and 8 weeks (right) after the third dose; Data (mean ± standard deviation) shown across 10 ECC contractions normalized into a percentage of the initial force before any ECC contractions and as the percentage of force retained after the tenth contraction; Vehicle-treated hDMD.*mdx* mice were used as a control group for normal muscle function; One-way ANOVA (Analysis of variance) was used for statistical comparison to vehicle-treated del44hDMD.*mdx* mice; **p < 0.01, ***p < 0.001, ****p < 0.0001 vs. vehicle; Data presented at the 2024 World Muscle Society conference.


Learn more at
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THERAPEUTICS