Corporate Presentation

June 2026

entrada THERAPEUTICS

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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 provide a potential best-in-class outcomes in high unmet need diseases, expectations regarding Entrada’s Phase 1/2 MAD clinical study of ENTR-601-44, including the completion of the Cohort 1 open-label period by year-end 2026 and the reporting, translatability, and ability to achieve a clinically meaningful effect from additional metrics data, including 10-meter walk/run, stride velocity 95th centile, 4-stair climb, north star ambulatory, and performance upper limb, the translatability of dystrophin concentrations and exon skipping based on juvenile NHP data, expected continued benefit in muscle function in the open-label period of Cohort 1 and future Cohorts, the timing of competitive data from Cohort 2 by year-end 2026, with expected higher levels of plasma and muscle exposure (linear or better) and substantially higher dystrophin expression including the potential of double-digit dystrophin levels, increases in muscle function and continued functional response over time, the timing of data from Cohort 3 to follow, expected significantly increased exon skipping and dystrophin expression for Cohorts 2 and 3, expectations regarding the data from the clinical study of ENTR-601-44, including expected results such as higher dystrophin restoration and positive safety data, expectations regarding muscle regeneration, muscle stabilization, and greater strength in patients as measured by Time to Rise velocity, expectations regarding the connection between EEV-PMO uptake in satellite cells and muscle regeneration, speed and durability of functional improvement, expectations of lower risk of anti-drug antibody response and lower cost of goods from EEV-PMO conjugates, translatability of current data to de-risk Entrada’s DMD clinical portfolio, support dose escalations, and its ability to support U.S. Accelerated Approval, expectations regarding Entrada’s Phase 1/2 MAD clinical study of ENTR-601-45 and the timing of first-in-class data from Cohort 1 in mid-2026, expectations regarding ENTR-601-45 and its potential best-in-class clinical profile, expectations regarding regulatory filings in the EU for the planned Phase 1/2 MAD clinical study of ENTR-601-50, expectations regarding the global regulatory filings for the planned clinical study of ENTR-601-51, the ability to recruit for and complete global Phase 2 clinical studies of ENTR-601-44, ENTR-601-45, ENTR-601-50 and ENTR-601-51 and to obtain U.S. Accelerated Approval and/or full approval for each program, the ability of EEV therapies to provide a 25-50-fold improvement in endosomal escape and lower whole drug requirements compared to antibody-based therapies, the ability to continue to expand and develop additional therapeutic programs and modalities, including further exon skipping programs, 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, expectations regarding the timing of nomination of a second clinical candidate for inherited retinal disease in the second half of 2026, expectations regarding Entrada’s ENTR-801 program, including the potential for quarterly dosing, initiation of IND-enabling studies and Entrada sharing clinical strategies in 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, 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 results shared in the second half of 2026, the size of the DMD market opportunity in the U.S. and ex-US, and the sufficiency of Entrada’s cash resources into the third quarter of 2027, constitute forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995. 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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 • **Favorable safety data and early but compelling functional improvement results*** • **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

*ELEVATE-44-201 Cohort 1, **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.

Multiple clinical milestones achieved or expected in 2026

Clinical inflection points for ENTR-601-44, ENTR-601-45 and VX-670 in 2026; ENTR-601-44 safety data fundamentally de-risks the neuromuscular portfolio

Rapidly Expanding DMD Clinical Franchise

ENTR-601-44

- Cohort 1 MAD complete; All patients transitioned to open-label dosing
- Cohort 2 dose escalation cleared to initiate at 12 mg/kg; Dosing ongoing
- Cohort 1 open-label and Cohort 2 MAD data expected by year-end 2026

ENTR-601-45

- Cohort 2 dose escalation cleared to initiate at 10 mg/kg: Enrollment ongoing
- Cohort 1 data expected mid-2026

ENTR-601-50 and ENTR-601-51

- Additional regulatory filings expected following a review of the data from the ongoing ENTR-601-44 and ENTR-601-45 DMD clinical studies

Vertex Advancing VX-670 for the Treatment of DM1

- Completed SAD portion of global Phase 1/2 study
- MAD evaluation of safety and efficacy is ongoing
- Vertex to share study results in H2 2026
- Partnership terms include up to $485 million in milestones, plus royalties

Advancing Innovative Preclinical Pipeline

Inherited Retinal Diseases

- Nominated first ocular candidate, ENTR-801 for Usher syndrome type 2A
- Second program in lead optimization; Candidate declaration in H2 2026

Additional Discovery Efforts

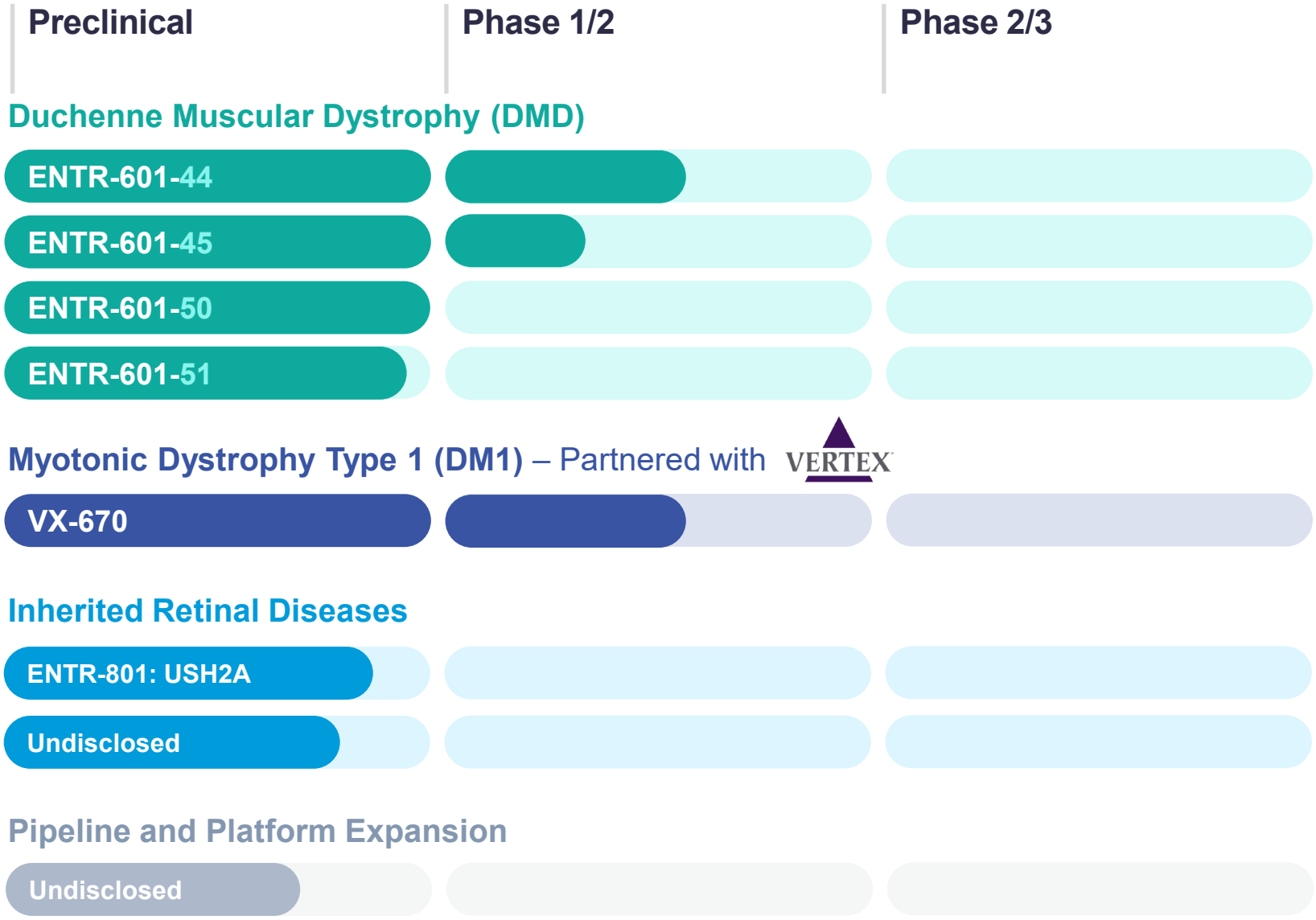
- Expansion of neuromuscular and ocular franchises leveraging next-generation EEVs and oligonucleotides
- Evaluating a range of undisclosed diseases, targets and modalities

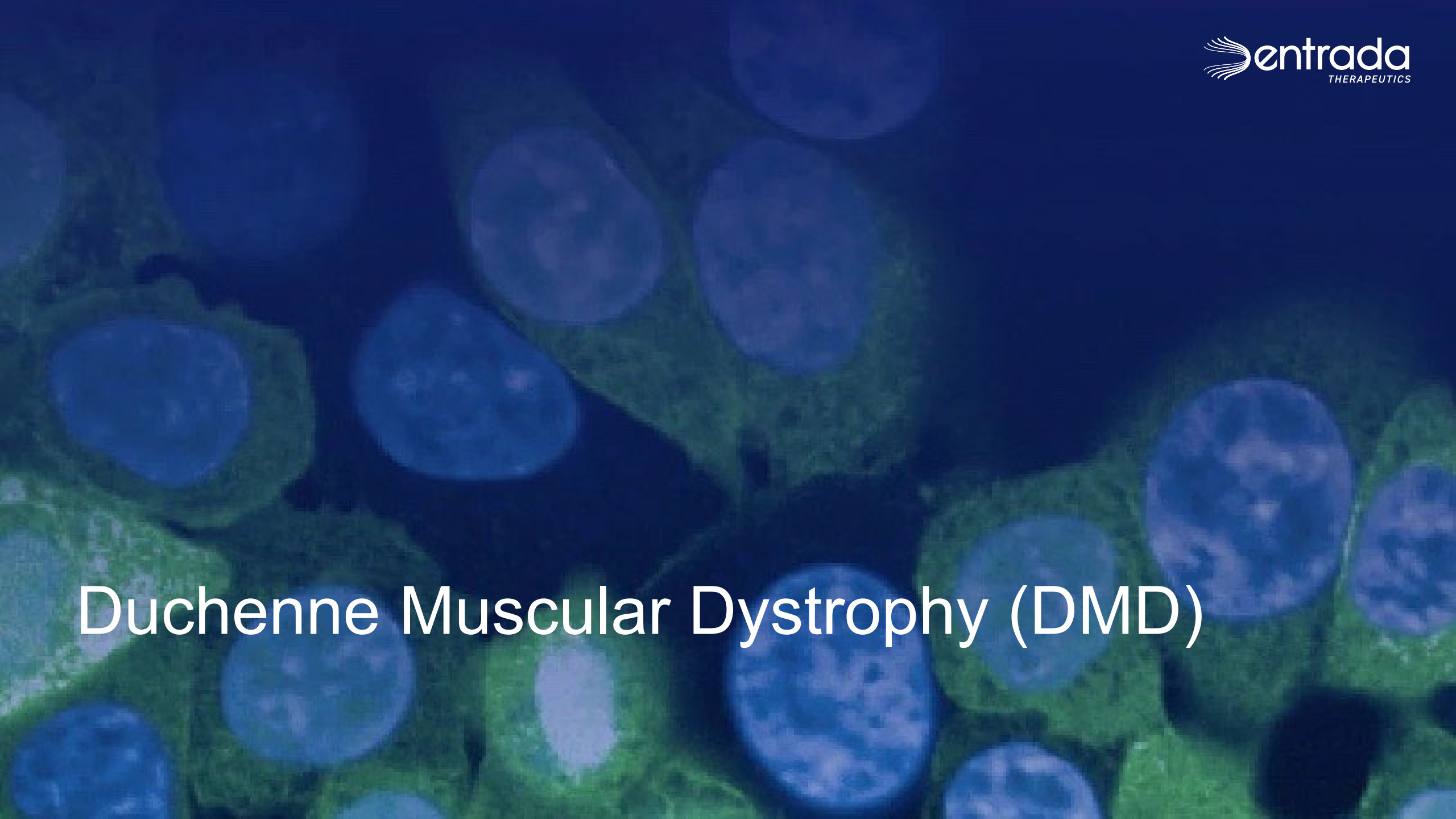
*All references in this presentation regarding planned regulatory filings and clinical study designs are subject to ongoing discussion with regulatory authorities;
DMD: Duchenne muscular dystrophy; **DM1**: Myotonic dystrophy type 1; **SAD**: Single ascending dose; **MAD**: Multiple ascending dose.

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





Duchenne Muscular Dystrophy (DMD)

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

Three strategic pillars: Differentiation, de-risking and best-in-class opportunities

Differentiated EEV™ Platform and Novel PMO Conjugates

- **Novel EEV and PMO** sequences
- **Best-in-class** non-clinical data translated into compelling safety and functional data
- Progenitor cell uptake supports **regenerative potential, speed and durability of functional improvement**
- **Lower whole drug exposure** supports lower risk of anti-drug antibody response and lower cost of goods

De-risked and Rapid Clinical Development

- Favorable ENTR-601-44 Phase 1 healthy volunteer safety profile
- Favorable ENTR-601-44 Phase 1/2 patient safety data for Cohort 1
- **Statistically significant improvement in functional benefit*** observed in ENTR-601-44 Cohort 1
- All neuromuscular programs **leverage the same EEV**
- **ENTR-601-44 Cohort 1 data de-risks the DMD franchise**

Potential for "Best-in-class" Commercial Opportunities

- Phase 1/2 data from ex-U.S. studies to support **U.S. Accelerated Approval****
- ENTR-601-44 Cohort 1 open-label and Cohort 2 MAD expected to deliver **competitive data** by year-end 2026
- **First-in-class clinical program in exon 45** projected to deliver Cohort 1 MAD data by mid-2026

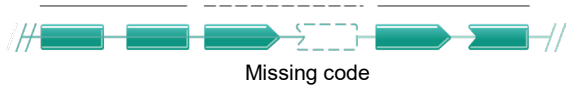
PMO: Phosphorodiamidate morpholino oligomer; Healthy volunteer may also be described as **HNV** (healthy normal volunteer); *Time to Rise velocity (TTRV), nominal p-value, *post hoc* analysis; **Accelerated Approval is subject to regulatory feedback.

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

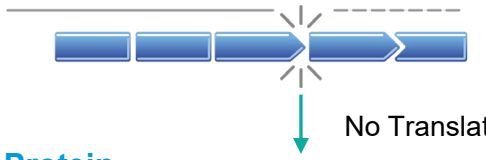
Entrada's Approach: Modulate RNA Splicing and Produce Functional Protein to Preserve and Rebuild Muscle

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

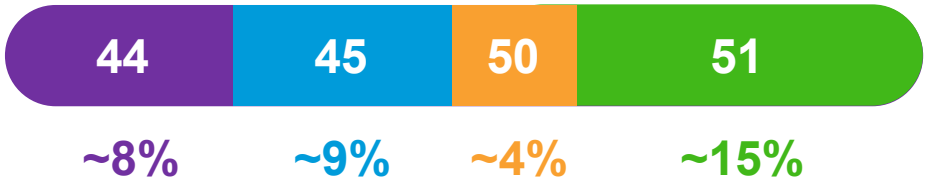
Splice modulation

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*

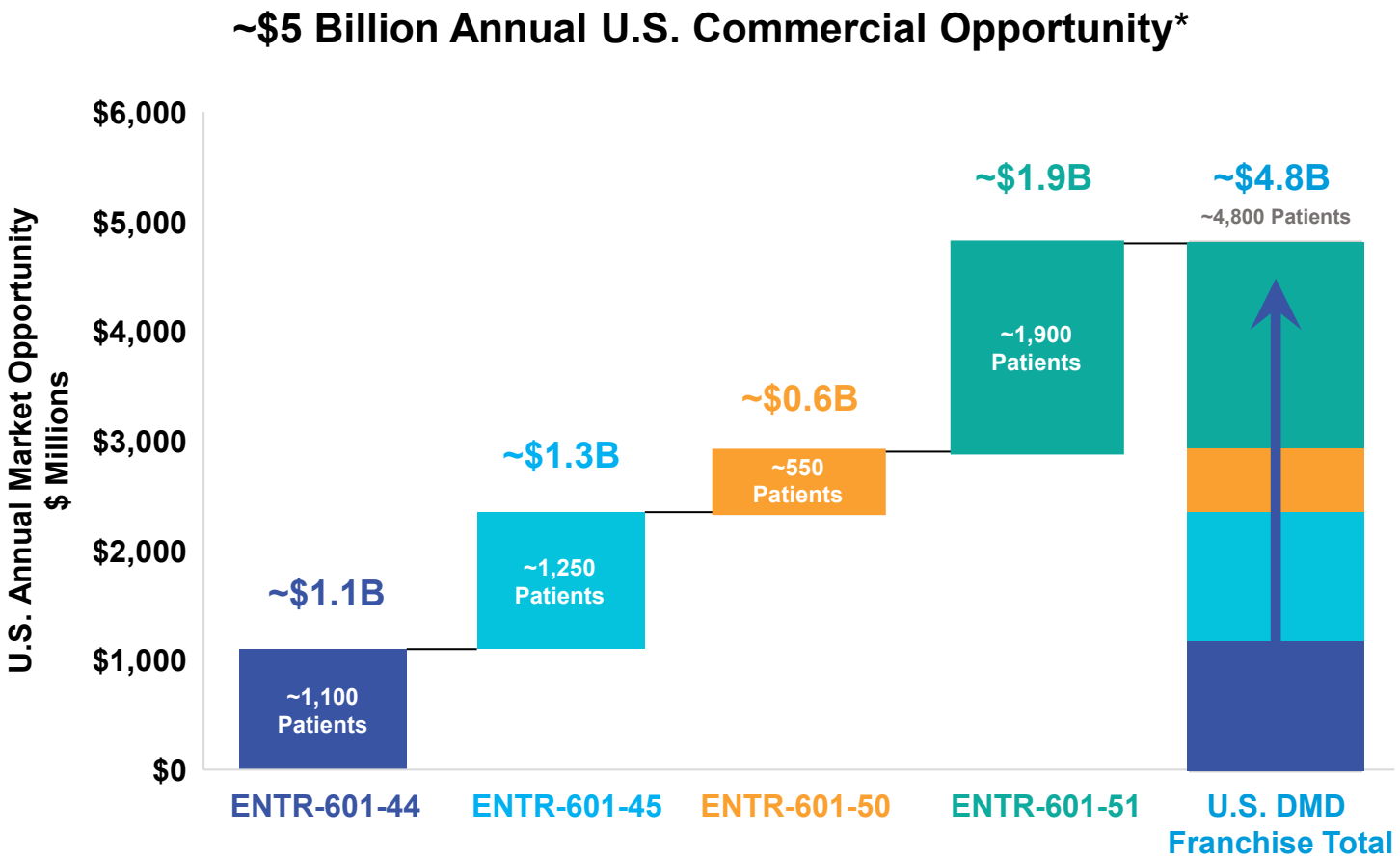
Entrada addresses mutations which result in a lack of dystrophin, muscle breakdown, progressive disability and death

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).

Entrada's DMD franchise unlocks a potential $5B U.S. market opportunity, with significant further upside ex-U.S.

Entrada's DMD franchise is positioned to address ~1/3 of all U.S. DMD patients, representing a potential ~$5B annual market opportunity in the U.S. alone



- Few available treatment options and limited competitive penetration to date
- **~5,000 U.S. patients** addressable with exon skipping therapies 44, 45, 50 and 51**
- **~6,500 European patients** addressable with exon skipping therapies 44, 45, 50 and 51**
- Ex-U.S. market not factored into projections and represents further upside potential

*$1M per patient per year market price estimated based on Lin et al., Institute for Clinical and Economic Review, April 2022, Posner et al., Journal of Manag. Care and Spec. Pharm. 2025;
**Bladen et al., HGVS Journal, January 2015; Total U.S. DMD population estimated to be ~7-8 per 100,000 males (~13,800) per Crisafulli et al., Orphanet J. Rare Disease, June 2020.

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; Importantly, Cohort 1 achieved functional benefit at the lowest dose with dystrophin levels expected to increase in Cohorts 2 and 3

- **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
- **Functional benefit demonstrated at 6 mg/kg via Time to Rise and Time to Rise Velocity (TTR and TTRV) measures**
  - TTR is a secondary endpoint of the ELEVATE-44-201 study
  - Cohort 1 resulted in a statistically significant* impact on TTR and TTRV versus placebo
  - TTRV levels were several fold above MCID after only 3 doses (127 days, 6 weeks after the last dose administered)
  - Additional functional measurement data to be shared at the end of the open-label period (year-end 2026)
- **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
- **Updated PK modeling, based on recently received juvenile NHP data, supports higher dystrophin in Cohorts 2 and 3**
  - Due to lower plasma drug exposures, dystrophin levels in Cohort 1 were lower than predicted

Early functional benefit response is compelling and statistically significant

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

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%

- **Baseline dystrophin levels seen in Cohort 1 were the lowest levels observed in recent Exon 44 studies**
- **Higher levels of baseline dystrophin correlate to higher treatment responses and vice versa**

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

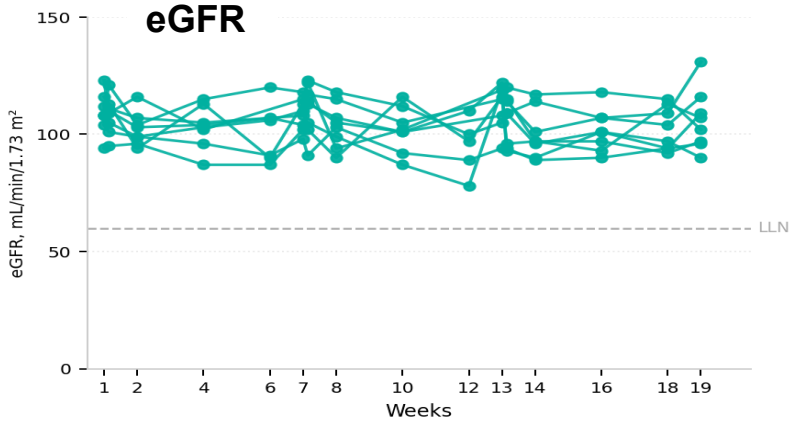
- Headache was the most common study drug–related TEAE, reported in 50% of the treatment group and 50% of the placebo group
- All events resolved
- **There were no serious TEAEs** and no study discontinuation due to any causes
- **No hypomagnesemia** or renal safety concerns were noted

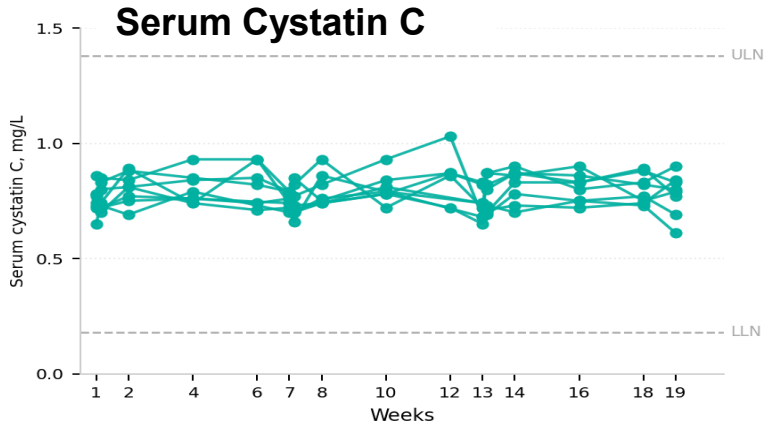
A highly favorable safety profile supports a de-risking of DMD programs

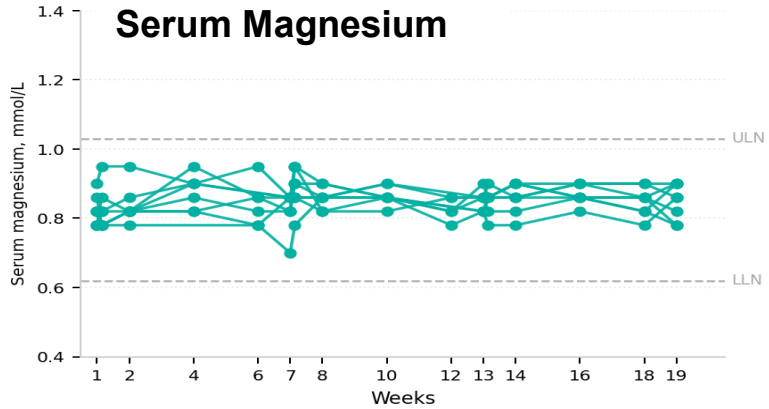
TEAE: Treatment emergent adverse event.

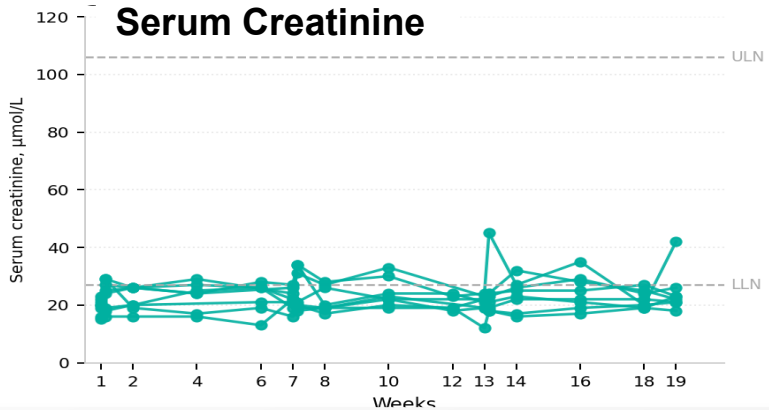
Renal markers were within normal range and comparable to placebo

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; **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 participants with DMD due to reduced muscle mass.

ELEVATE-44-201

Cohort 1: Functional Efficacy Data

Functional benefit summary

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

- **Time to Rise and Time to Rise Velocity are the first functional measurements to "move"**
  - TTR improvement versus placebo = 2.40 seconds (statistically significant)
  - TTRV improvement versus placebo = 0.09 rises per second (statistically significant)
- **Functional benefit demonstrated via Time to Rise and Time to Rise Velocity measurements**
  - TTRV is a calculation based on TTR that minimizes any imputation, outliers and noise associated with TTR
  - TTRV is a clinically significant and accepted regulatory endpoint
  - Cohort 1 resulted in a statistically significant impact on TTRV versus placebo at levels several fold above MCID after only 3 doses (127 days, 6 weeks after the last dose administered)
  - TTR and TTRV generally precede improvements in other functional measures (10MWR, etc.)
- Positive trends observed in 10MWR and will be tracked and reported at the end of the open-label period (year-end 2026)
- Additional metrics (e.g., 4SC) to be reported at the end of the open-label period (year-end 2026)

Early functional benefit response is compelling and statistically significant

10MWR: 10-meter walk/run; **4SC**: 4 stair climb.

Functional improvement
The importance and meaning of Time to Rise velocity

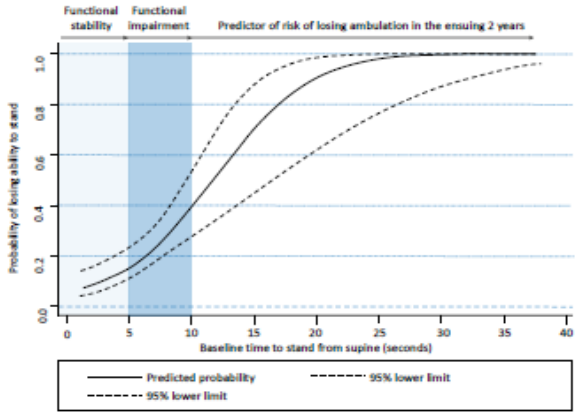
Time to Rise, a secondary endpoint in this study, is a robust, prognostic factor that predicts disease progression; Time to Rise velocity reduces the impact of imputation, outliers and noise

- **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.

Functional cascade: TTR velocity is a robust indicator of treatment effect and the earliest measurement to move

Proximal functional measures, including TTR and 4SC, detect changes before composite/distal measures like PUL and NSAA; TTR and TTRV were statistically significant versus placebo in Cohort 1

Relevant Study Period	Relevant Functional Readout	Status Of Functional Measure Collection and Readout
MAD (18 weeks)	**TTR Velocity** *Time to Rise* SV95C *Stride Velocity 95th Centile*	• **Statistically significant improvement versus placebo demonstrated in Cohort 1 MAD** portion of the study • Not collected during Cohort 1 MAD; To be reported at the end of the open-label period
Open-Label (~1 year)	**10MWR Velocity** *10-Meter Walk/Run* 4SC Velocity *4 Stair Climb*	• **Trend towards functional improvement in Cohort 1 MAD** portion of the study; Will be assessed at the end of the open-label period • Baseline not collected in Cohort 1 MAD; To be reported at the end of the open-label period
OL – LTE* (≥1 year)	NSAA *North Star Ambulatory* PUL 2.0 *Performance Upper Limb*	• Composite measure; Will be assessed at the end of the open-label period • Less sensitive in ambulatory participants; To be reported at the end of the open-label period

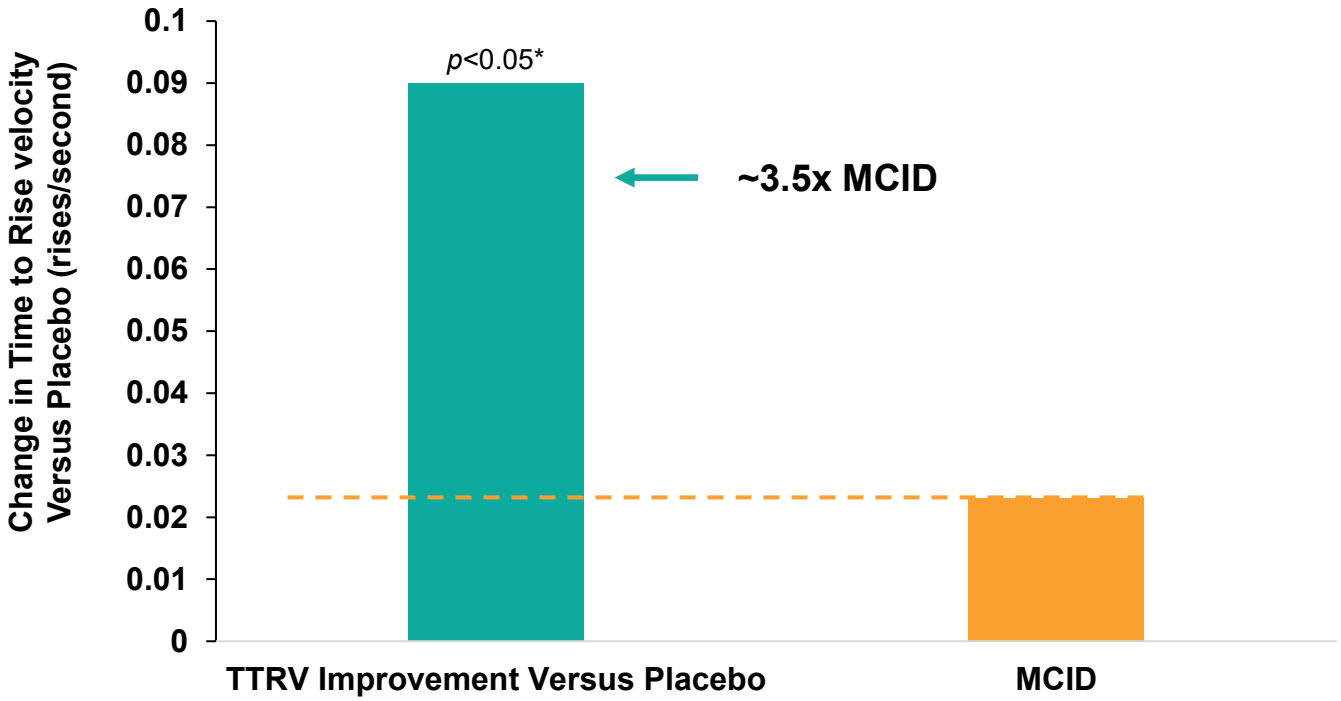
ENTR-601-44 Cohort 1 detected a statistically significant TTRV signal at Day 127 (18 weeks) which is consistent with TTRV being the earliest timed function test to detect treatment effect

Sources: Duong et al., J Neuromuscul Dis 2021;8(6):939–948; Gupta et al., PLOS ONE 2023; Rabbia et al., J Neuromuscul Dis 2024; EMA SV95C Qualification Opinion 2022; McDonald et al., Muscle Nerve 2013; Mercuri et al., NMD 2016; **OL/OLE:** Open-label extension; **LTE:** Long-term extension; **Day 127** may be referred to as D 127.

Disease-modifying functional benefit seen at lowest dose
TTRV results

Significant improvements in TTR and TTRV were observed*; TTRV is a widely accepted Phase 3 endpoint in DMD, with good statistical properties for evaluating motor function in patients

TTRV Improvement Versus Placebo*



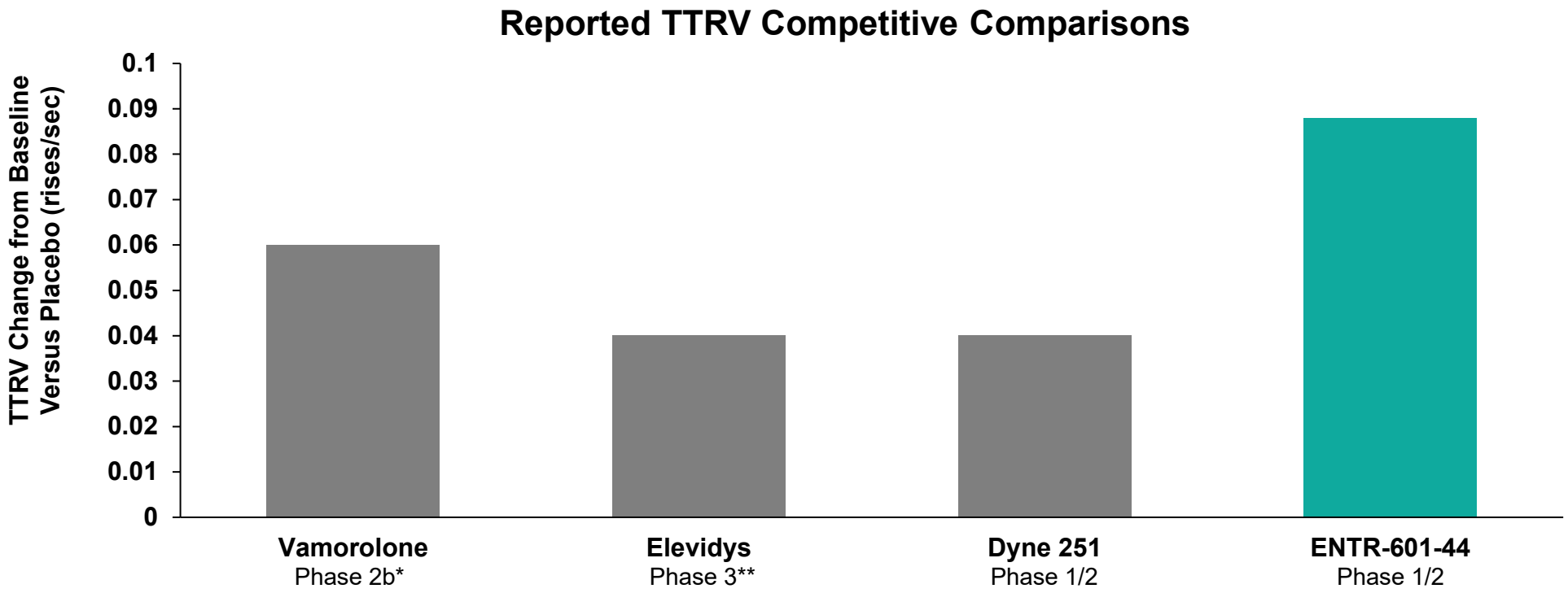
Key Takeaways

- **TTR is a pre-specified secondary endpoint in the ELEVATE-44-201 study**
- Change in TTR and in TTRV was seen across the majority of treated participants, irrespective of age
- The mean change in TTRV versus placebo was 0.09, ~3.5x higher than the MCID threshold of 0.023
- 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 magnitude of the overall response supports the hypothesis that Cohort 1's functional benefit represents a true drug-related effect
- In addition, 10MWR also demonstrated positive trends versus placebo

*Analysis on TTRV versus placebo shown rather than TTR in order to maintain the study blind, Wilcoxon rank-sum (one sided) test, nominal p-values, 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; Updated from previously disclosed topline data to reflect subsequently received information.

ENTR-601-44 delivered a differentiated impact on TTRV
TTRV is a validated registrational endpoint

ENTR-601-44's TTRV versus placebo is superior to reported numbers from steroids, gene therapy and second-generation exon skipping therapies



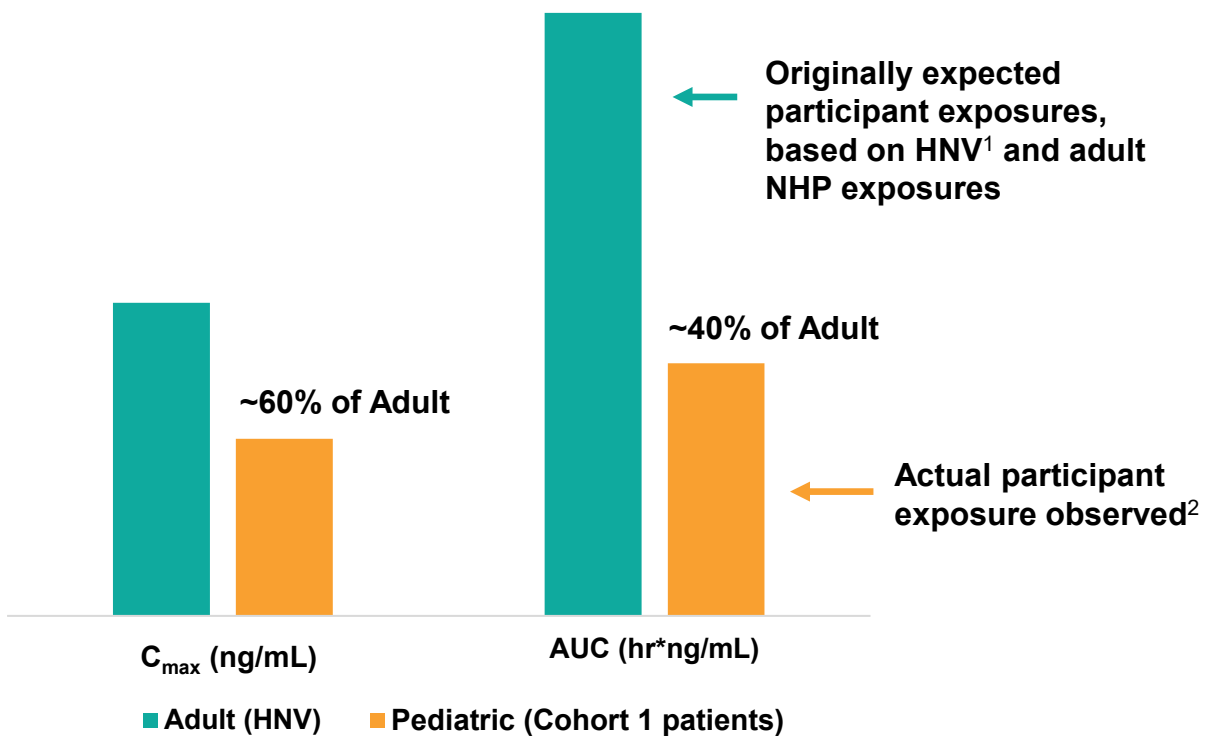
- **Phase 3 trials using TTRV as the primary endpoint: Novartis**: *del-zota*, Phase 3 (initiating); **Dyne**: 251/Z-rostudirsen, Phase 3 (initiating); **Solid Biosciences**: SGT-003, Phase 3 (ongoing); **Roche**: Elevidys, Phase 3 (to initiate in EU)

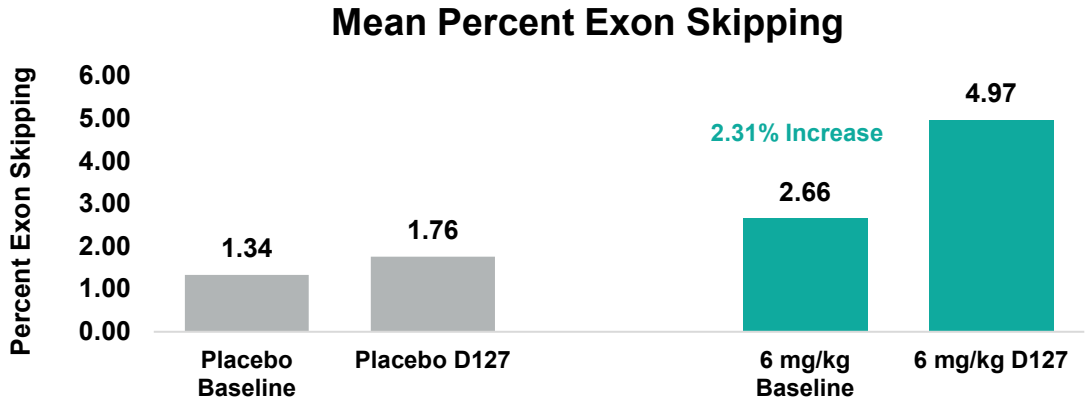
Sources: *VISION-DMD (JAMA 2022) registrational study; DELIVER (Dyne Company presentation); EMBARK (FDA Decisional Memo 2024); ENTR-601-44 (Cohort 1); No head-to-head trials have been conducted; Cross-trial comparisons may not be reliable based on differences in study designs, compound formulation and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

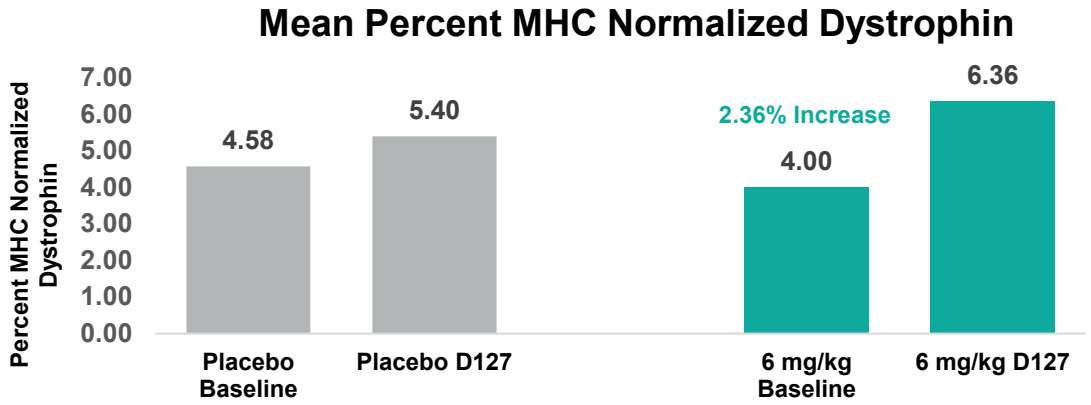
ELEVATE-44-201: Pharmacokinetics and Biomarkers

Lower-than-expected plasma exposures resulted in lower-than-expected exon skipping and dystrophin expression

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







A linear (or better) increase in exposure is expected in Cohort 2 at 12 mg/kg which should result in substantially higher dystrophin levels and continued muscle function

AUC: Area under the curve (proportions are approximate); [1]Single dose 6 mg/kg, [2]Pediatric DMD participants following third dose of 6 mg/kg every 6 weeks (similar, proportionally lower levels seen in juvenile NHP 55 week study); 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.

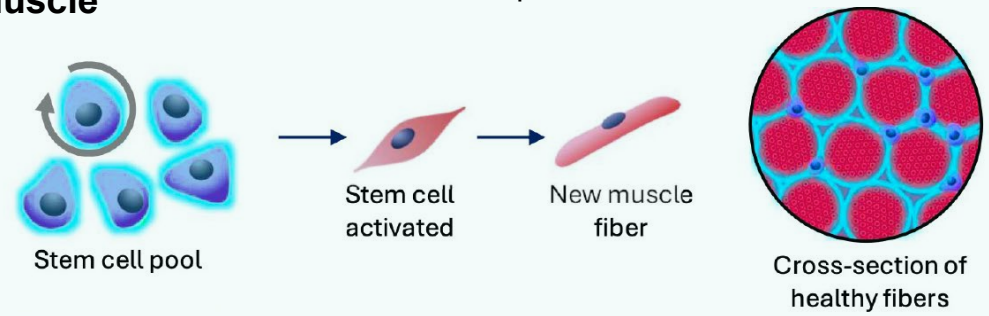
ENTR-601-44: Mechanistic Rationale

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

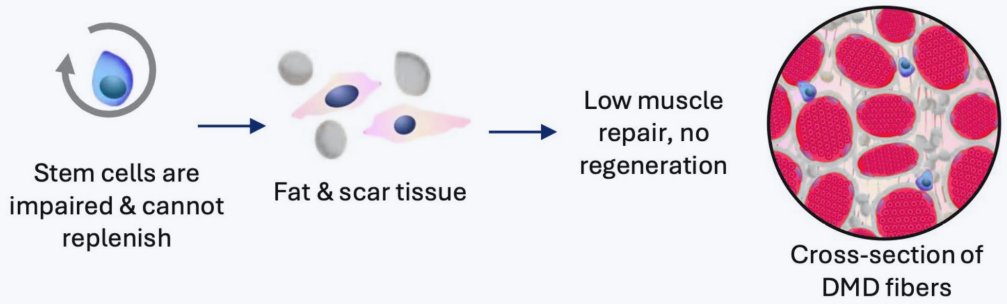
Healthy muscle

- Dystrophin supports stabilizing and repairing muscle fibers and stem cell replenishment and 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 the inability to repair and regenerate new fibers
- Lack of normal regeneration results in the replacement of muscle with fat and fibrosis

Satellite (stem) cells require 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

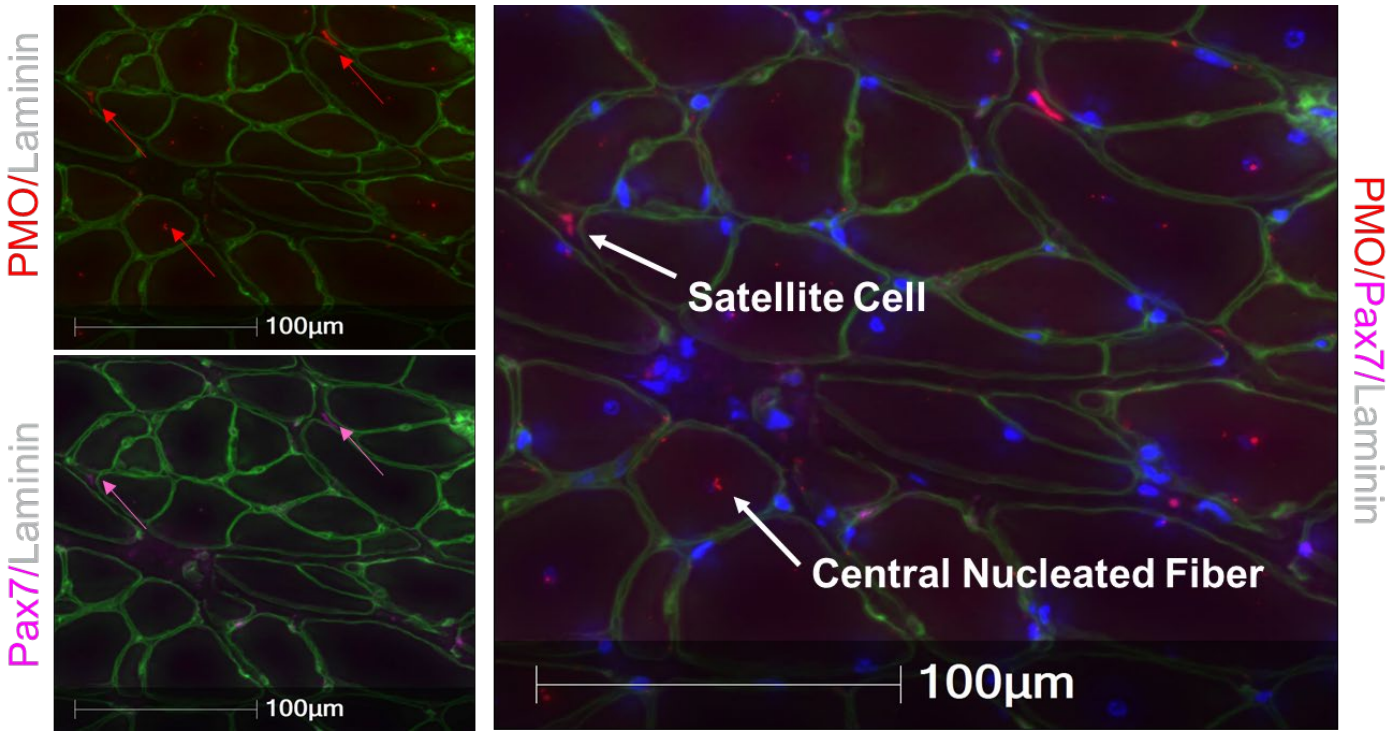
Enhanced satellite cell activity would result in the regeneration of healthy fibers and the stabilization of the overall muscle; In turn, this may convert into greater strength, as measured by TTRV

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)

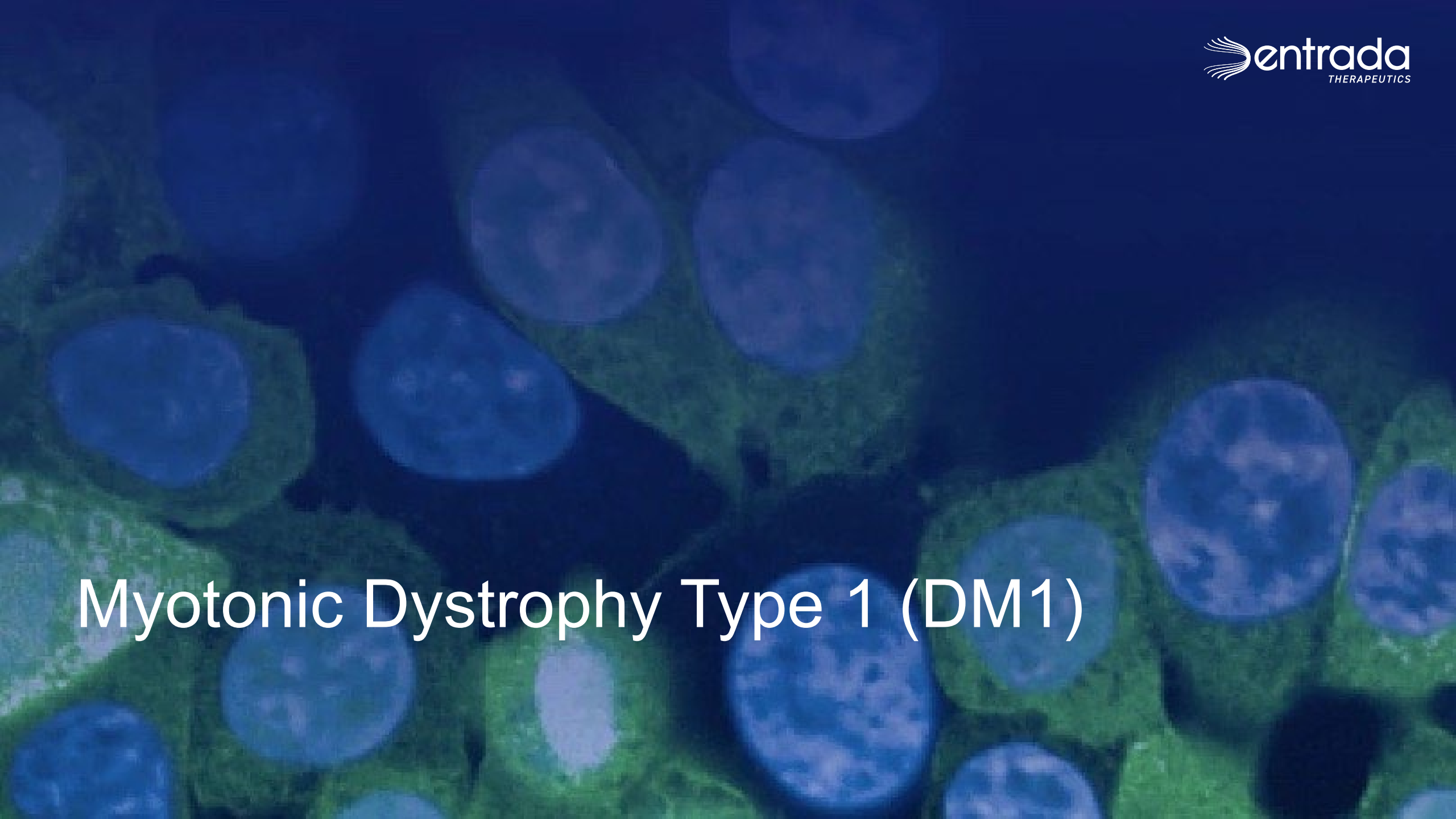
Antibody and gene therapy competitors

- 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*: 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.



Myotonic Dystrophy Type 1 (DM1)

DM1: A transformational partnership with Vertex

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

DM1 partnership established with Vertex in Q1 2023

- 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

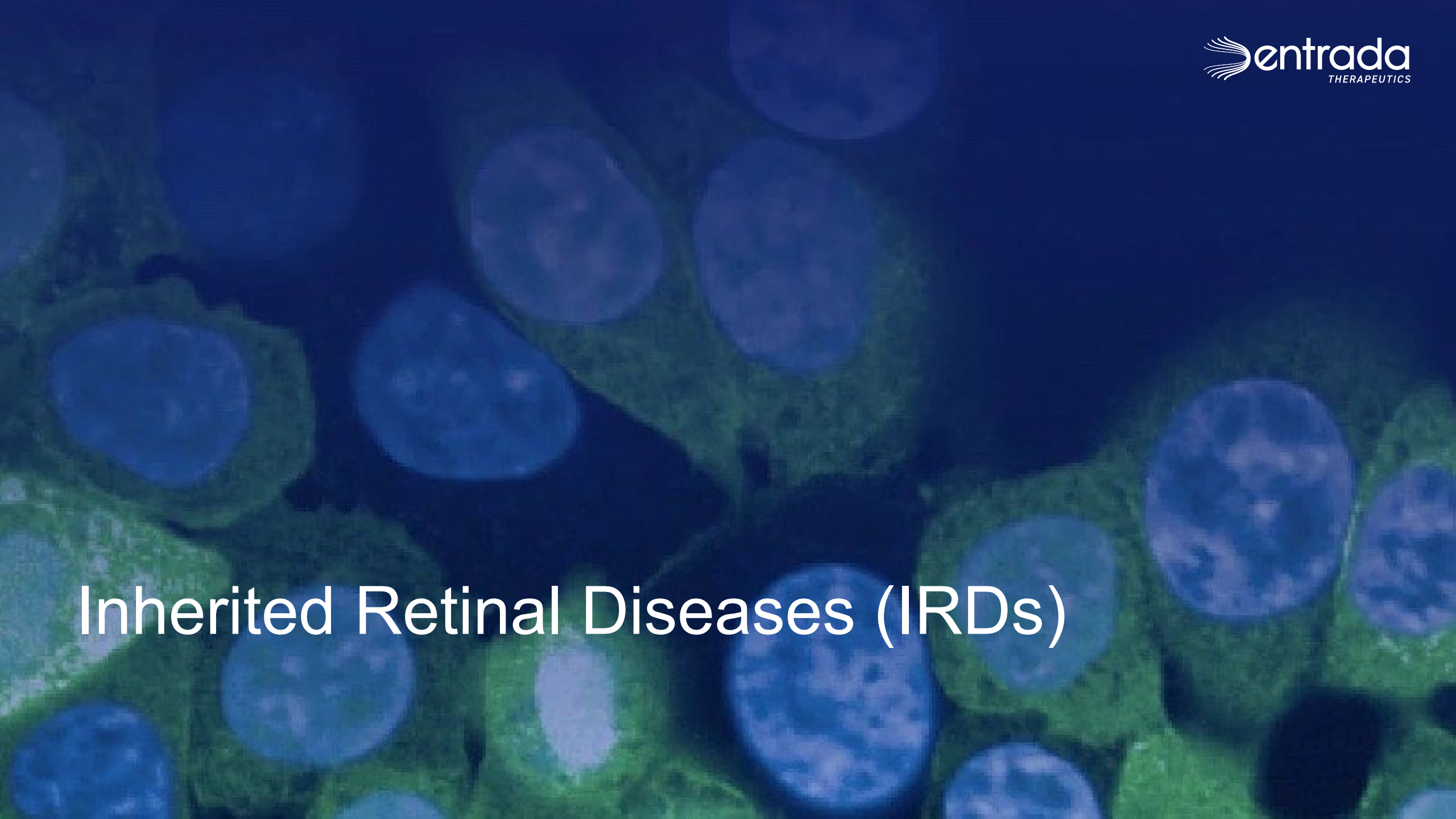
VX-670 differentiation

- 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*

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

- MAD portion of the clinical study to assess safety and efficacy is on track to read out in the second half of 2026
- Open-label extension study is enrolling
- Same EEV utilized for VX-670 as in DMD franchise

*Hasuike et al., Front. Genet., March 2022; **MBNL**: Muscleblind-like proteins; **CUG repeat**: Repeating sequence of cytosine, uracil and guanine in RNA; **DMPK**: Dystrophia myotonica protein kinase.



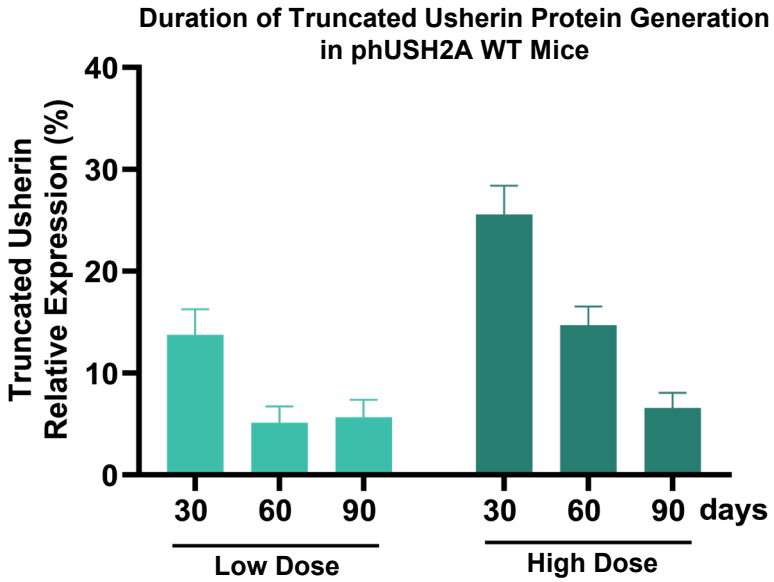
Inherited Retinal Diseases (IRDs)

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; 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 changes in the USH2A gene
- In some patients, mutations in exon 13 prevent production of the usherin protein
- Without usherin, photoreceptors (the light-sensing cells in the eye) gradually 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 usherin protein production, with the potential for quarterly dosing IVT
- IND-enabling studies will be initiated and clinical strategies will be shared in 2026
- **~15,000 patients in the U.S. and Europe are exon 13 skipping amenable***

Note: **IRD**: Inherited retinal disorders; *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

2026 inflection points

ELEVATE-44-201 Cohort 1 open-label functional data, Cohort 2 safety, 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**
  - Placebo participants crossed over into treatment via the open-label period
  - Company expects continued functional response with additional measurements collected during 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 with readout expected by year-end 2026**
  - 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 readout expected in mid-2026**
  - Data Monitoring Committee cleared Cohort 2 dosing at 10 mg/kg and enrollment has initiated
  - Significant unmet market need for new therapies
  - ELEVATE-45-201 dosing regimen of every 6 weeks is a potentially compelling point of differentiation

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

Cohort 2 cleared to dose escalate to 10 mg/kg and enrollment has initiated

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 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.

Appendix

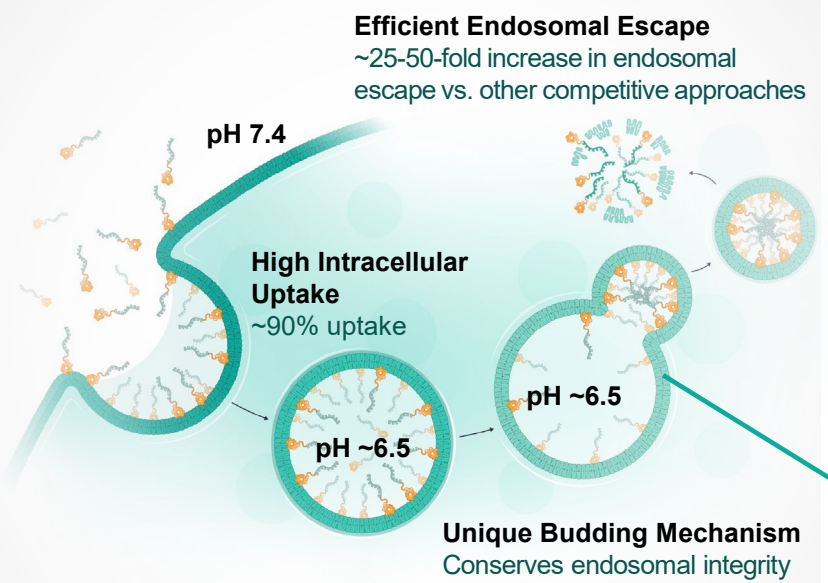
EEV™ Platform

Understanding EEV endosomal escape advantages

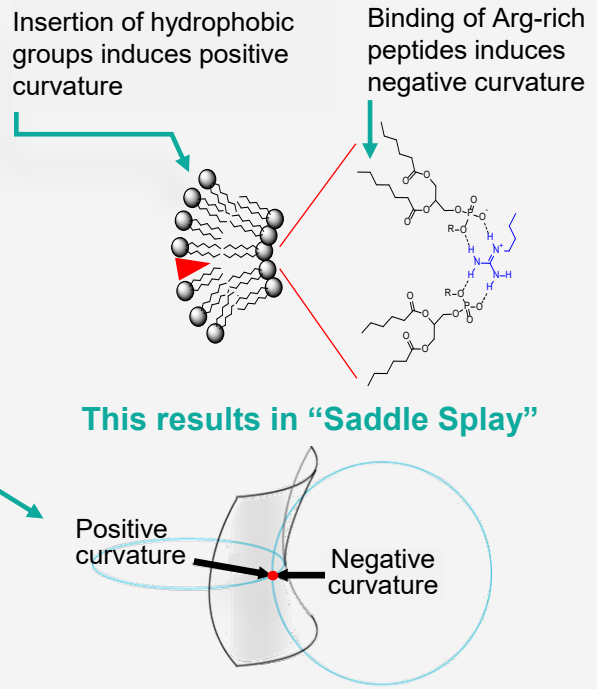
Unique pH-dependent membrane binding affinity enhances EEV endosomal escape

- 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

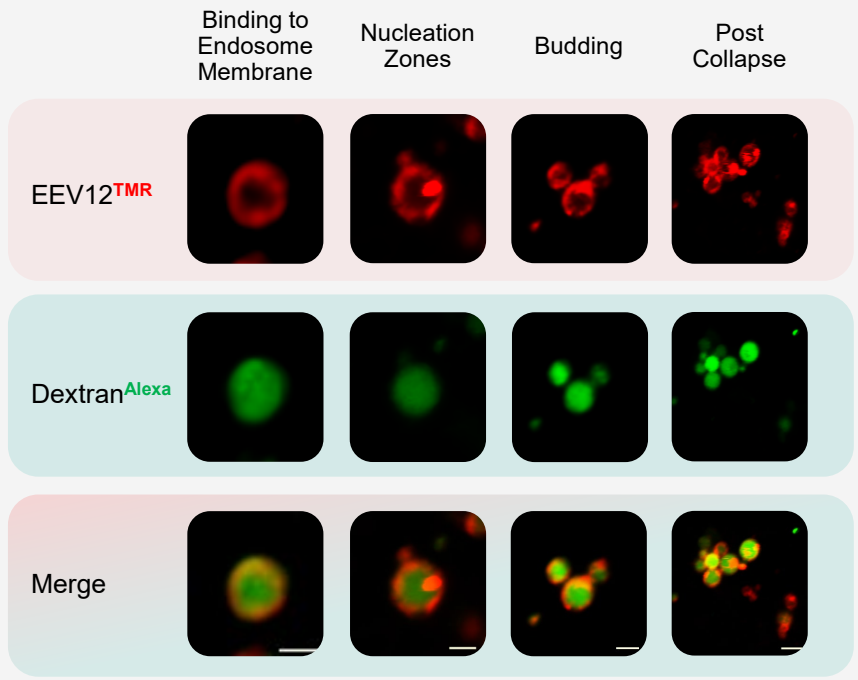
Concept



Mechanism



Visualization

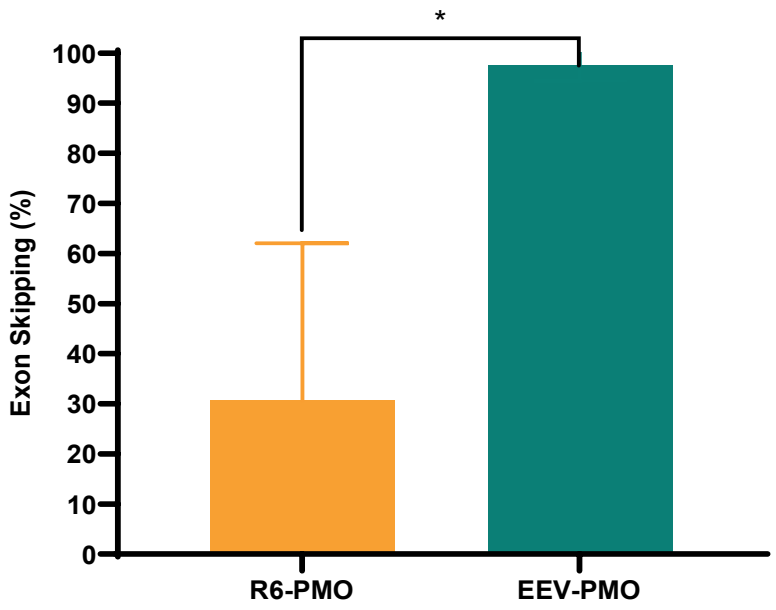


Sahni, A. et al. ACS Chem. Biol. 2021; **CPP**: Cell-penetrating peptide.

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

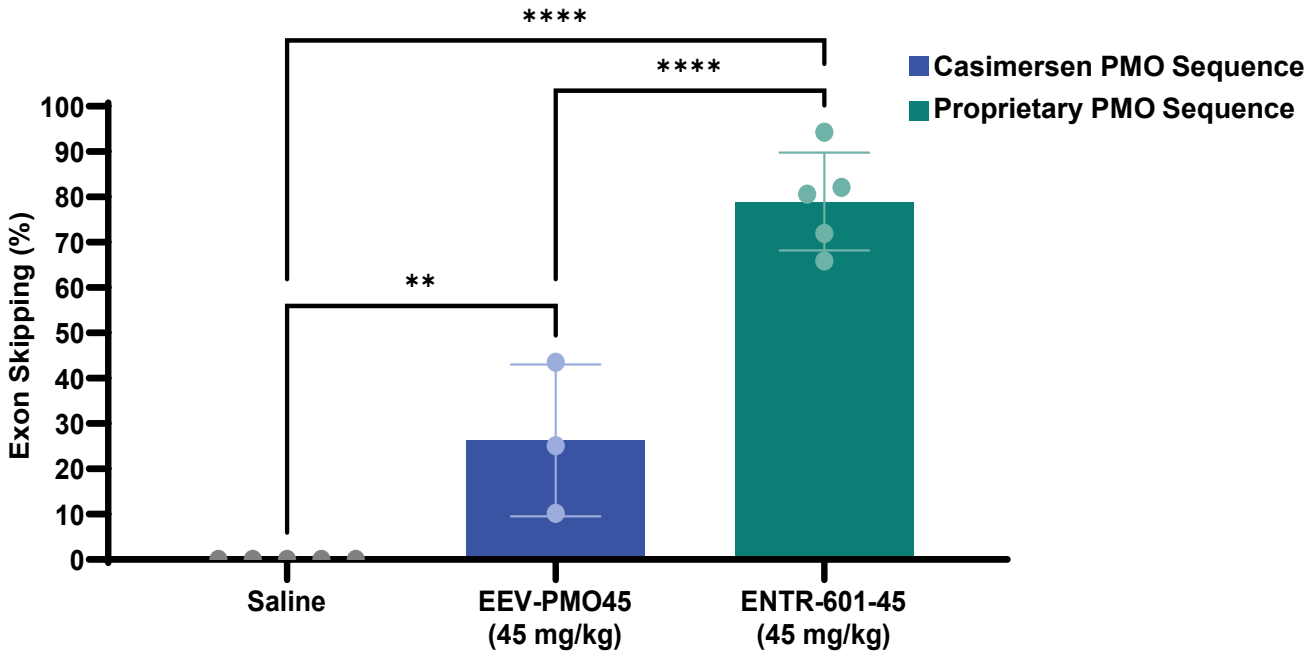
In the below DMD example, Entrada has optimized the EEV and the PMO sequence; The data represents the superiority of each component against other approaches and sequences

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



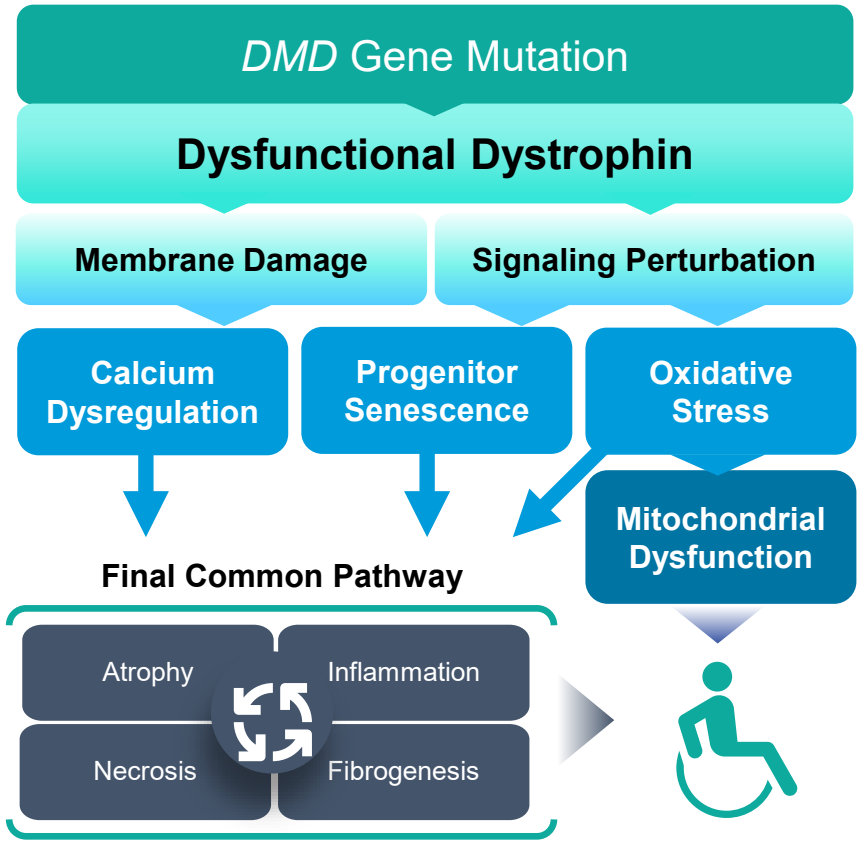
- EEVs are the same, PMO sequences are different (casimersen vs. Entrada proprietary)
- Proprietary PMO significantly improved exon skipping after 3 days in *mdx* mice as compared to competitive casimersen sequence

*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 addresses the underlying cause of DMD and restores dystrophin in muscle fibers and muscle stem cells

The Duchenne Disease Cascade



Current and Emerging Treatment Options

1. Disease-modifying agents address the root cause of morbidity and mortality

- Double-digit high-quality dystrophin production protects healthy muscle and restores regenerative capacity
- Must be safe, potent, reach both mature and progenitor cells, be infrequently dosed, be redosable and dosing is weight based to meet the needs of a growing child
- **Entrada is developing the only disease-modifying agents with the potential to meet ALL of the above**[1]

2. Supportive care agents only address downstream biology

- Myosin(i), AAK1(i), HDAC(i) may potentially enhance disease-modifying therapies and complement dystrophin restoration
- Mechanisms include membrane protection/repair and stem cell proliferation
- Symptomatic care, led by steroids, have served as standard of care despite long-term tolerability and safety concerns

Entrada's next-generation exon skippers

- Established safety at a therapeutic dose of 6 mg/kg with escalation to 12 mg/kg underway[2]
- Expected to provide durable and significant access to skeletal and cardiac muscle
- Target progenitor cells (satellite) needed to regenerate muscle and provide functional benefit
- Flexible to dose escalate and redose

Adapted from Front. Cell Dev. Biol., 18 August 2021; Sec. Molecular and Cellular Pathology; Volume 9, 2021; [1]No other exon skippers nor gene therapy fit all the disease-modifying criteria; [2]ENTR-601-44 data.

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

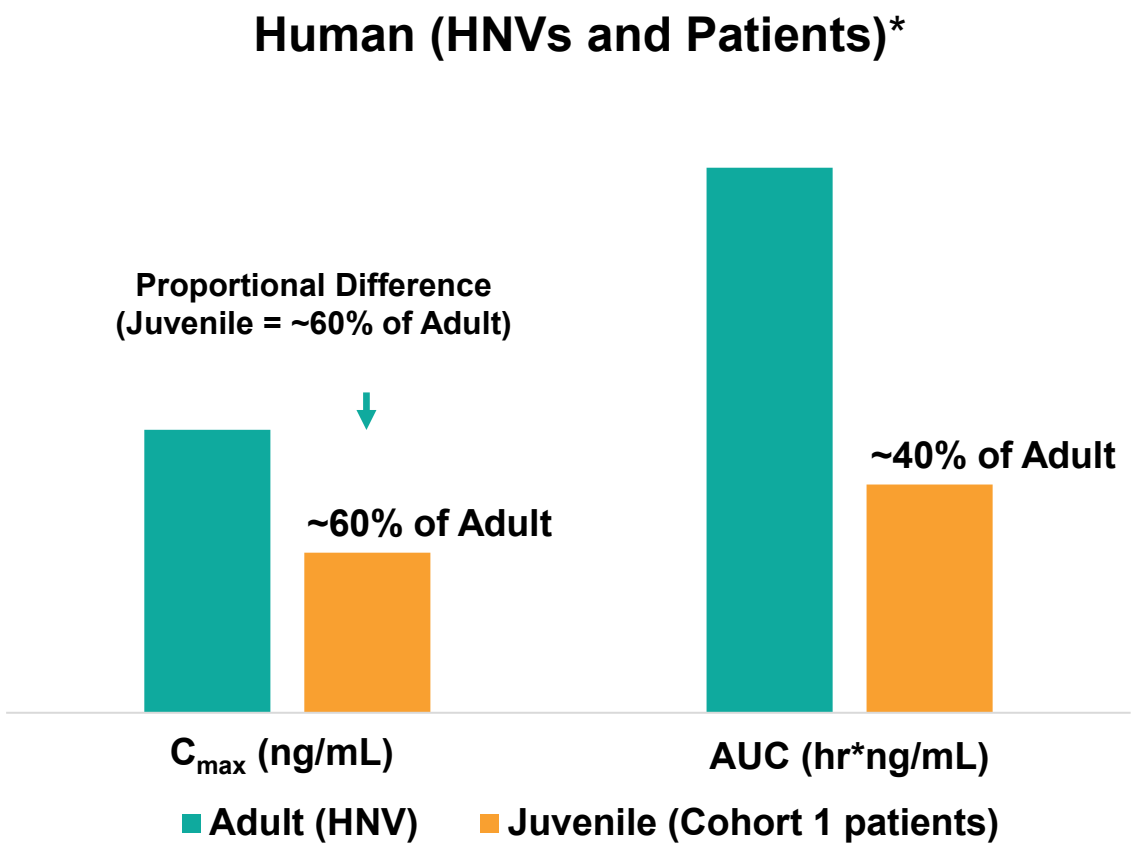
	EEV-PMO **Entrada**	**Anti-TfR1-mAb-PMO (exon 44)** Novartis/Avidity	**Anti-TfR1-Fab-PMO (exon 51)** Dyne
Design	**Cyclic, arginine-light peptide** 2.6 kDa Tissue-specific delivery; Increases stability and half-life	**Full-length antibody** ~150 kDa TfR1 specific deliver; Non-cleavable linker	**Fragment 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-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]

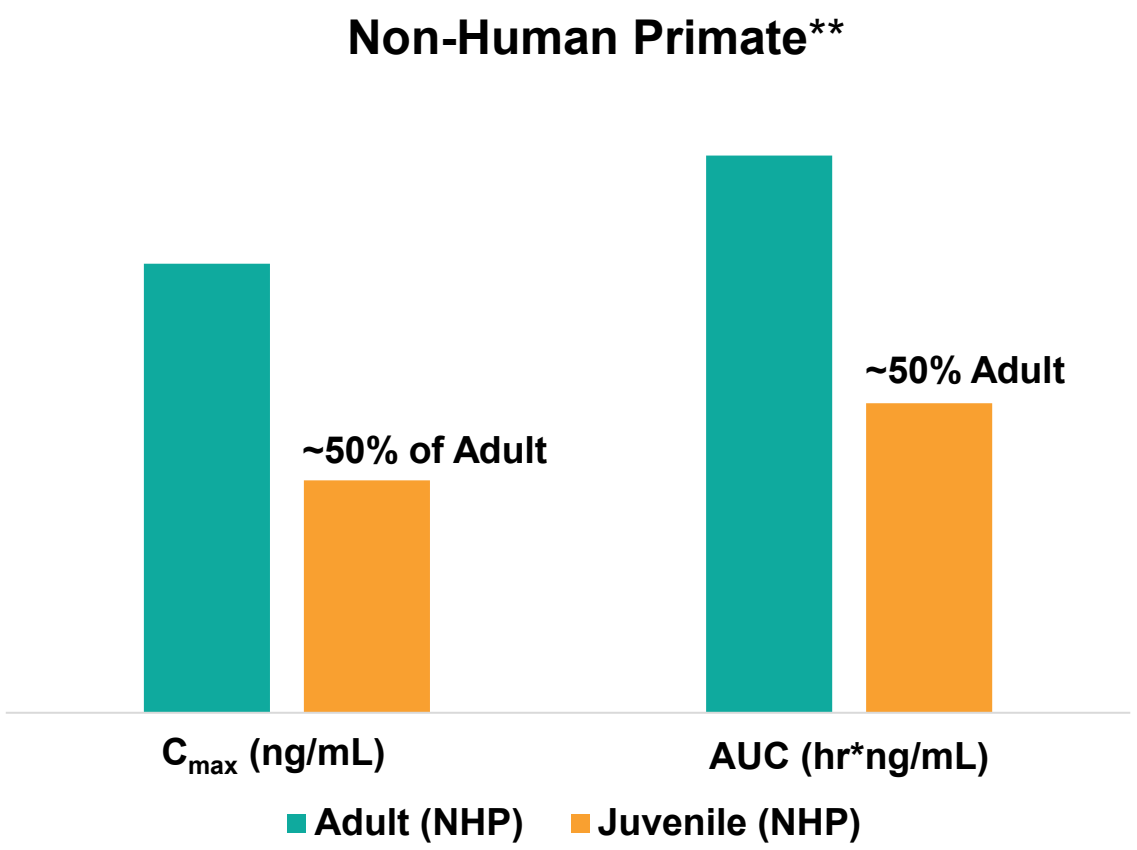
TfR1: Transferrin receptor 1; [1.]Etxaniz et al., Nucleic Acid Res., 2025; [2.]Dyne corporate materials from DELIVER Clinical Update investor call September 2025; [3.]Total mean exon skipping; Entrada internal data from Phase 1 SAD study of ENTR-601-44 in HNV; [4.]Unadjusted dystrophin, mean change from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

Pharmacokinetic Modeling

Predictive pharmacokinetics

Lower C_{max} and AUC observed in juvenile humans and NHPs; A linear (or better) increase in exposure is expected in Cohort 2 at 12 mg/kg (double dose = double exposure)



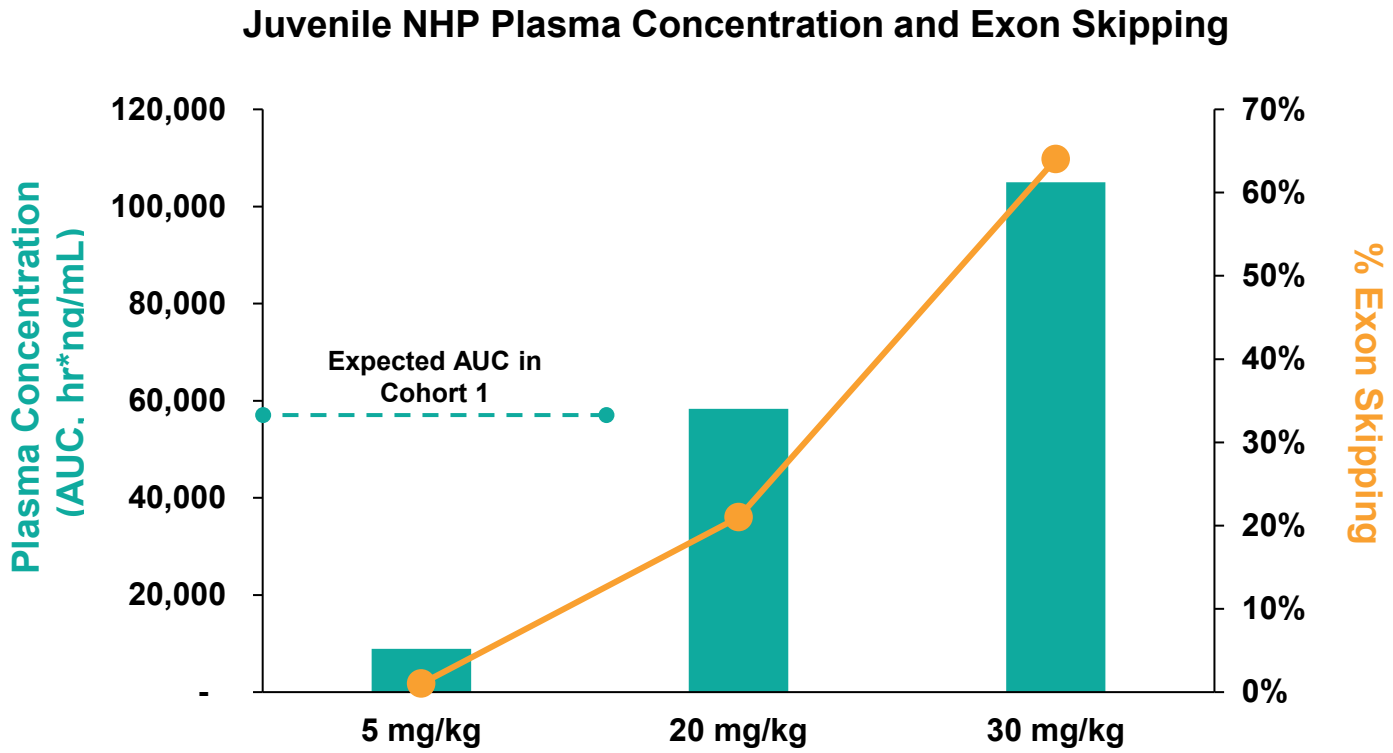


*Single dose 6 mg/kg, pediatric DMD patients following third dose; **Adult: 13-week 25 mg/kg repeat dose study, Q6W, 3 doses; Juvenile: 55-week 20 mg/kg repeat dose study, Q6W,10 doses.

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

Juvenile NHP data suggest an increase of AUC should result in substantially higher dystrophin levels and increases in muscle function

- Juvenile NHP data suggest a right-shifted double-digit dystrophin expectation starting at 12 mg/kg in Cohort 2
- As the exposure to exon skipping relationship has not changed, updated projections indicate significantly increased exon skipping and dystrophin expression expected in Cohorts 2 and 3
- Cohorts 2 and 3 dystrophin levels will be assessed for registrational potential, when combined with observed functional outcomes



NHP data from 55 weeks, QW6, 10 doses, chronic juvenile toxicology study.

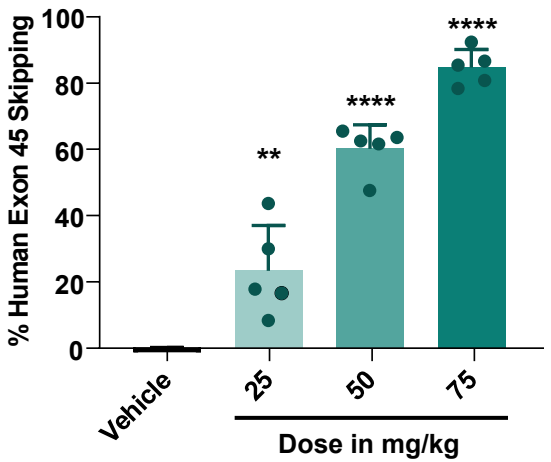
DMD Functional Data

Preclinical Models

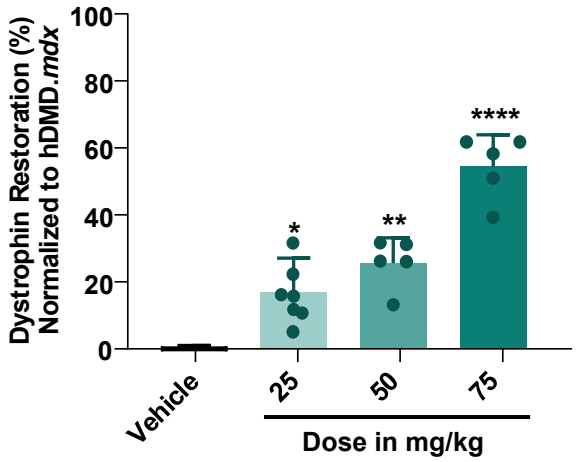
Preclinical data support potential for best-in-class clinical profile for ENTR-601-45

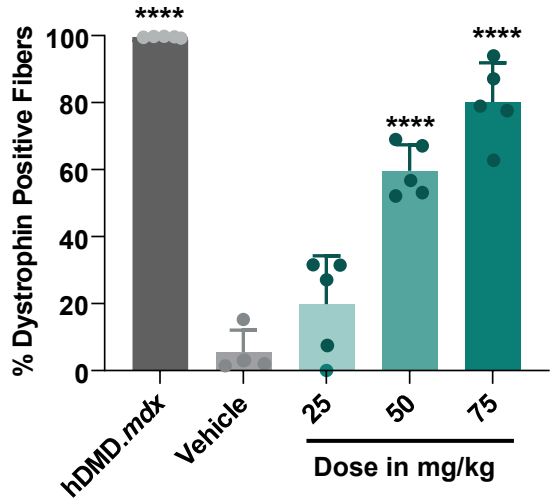
Dose-dependent increase in exon skipping and dystrophin expression correlates to complete functional correction

Exon Skipping

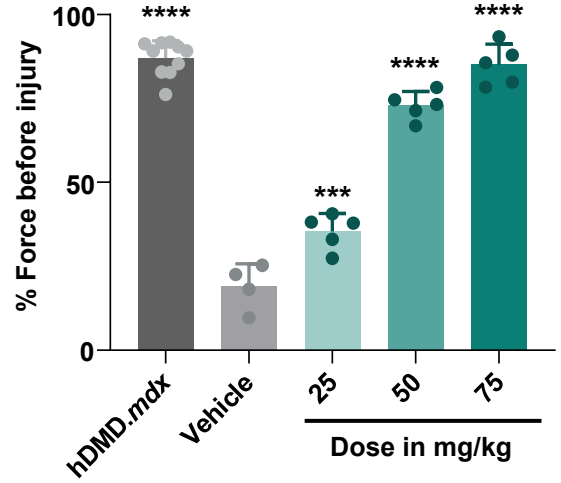


Dystrophin Restoration





Functional Correction



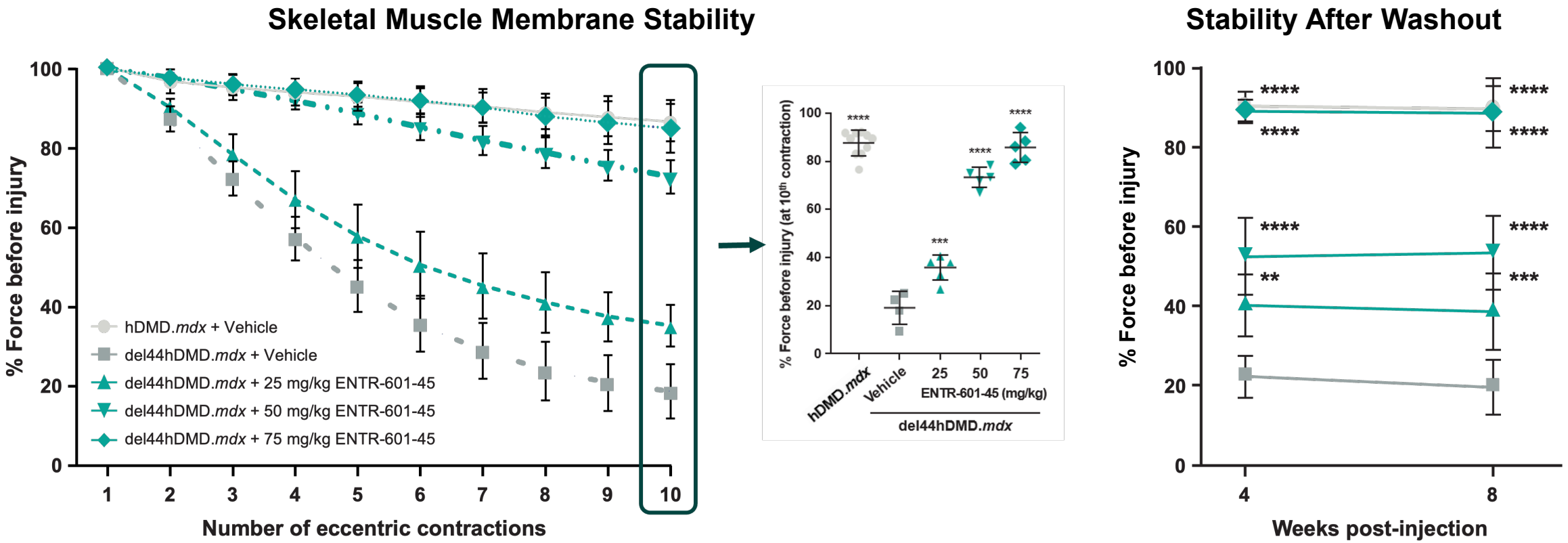
- Active and vehicle del44hDMD.*mdx* mice, n=5 per cohort, EEV-PMO-45 (Q6W x 3 doses); Control saline treated hDMD.*mdx* mice, n=10 (Q6W x 3 doses)
- Skipping (ddPCR) and dystrophin production (JESS) is significantly increased 6 weeks after the third dose of ENTR-601-45 (gastrocnemius muscle shown)

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



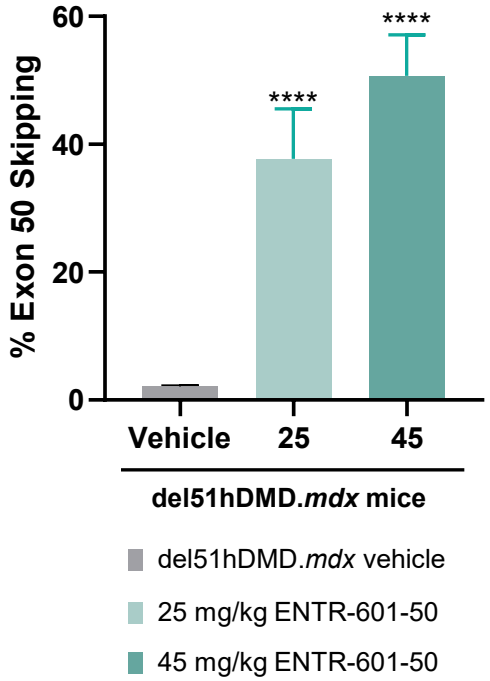
del44hDMD.*mdx* mice were treated with three Q6W IV injections of ENTR-601-45 or vehicle; ECC-induced muscle force loss generated by repeated eccentric force (ECC) contraction of the gastrocnemius muscle was assessed 5 weeks (left/center) or 4 and 8 weeks (right) after the third dose; Data (mean ± standard 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.

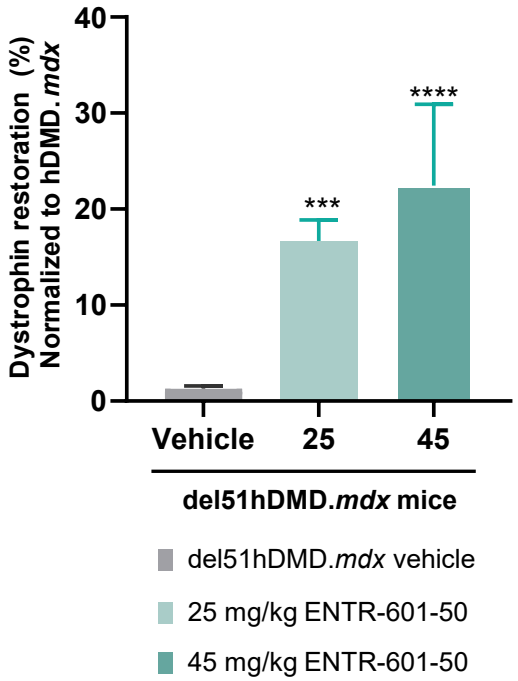
Efficacy of ENTR-601-50 in exon 50 skipping amenable mice

ENTR-601-50

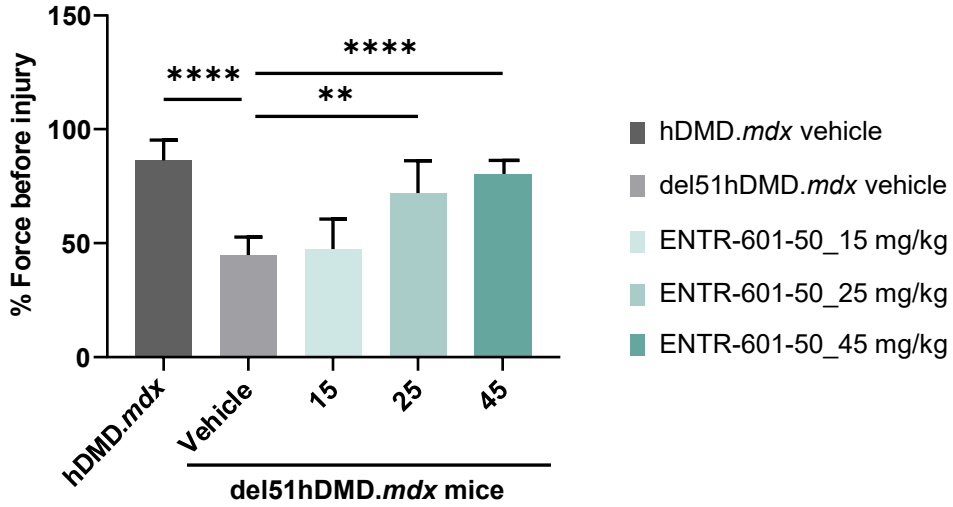
Single dose of ENTR-601-50 produced robust human *DMD* exon 50 skipping and dystrophin; Translated into improvements in muscle function within 2 weeks post-dose

Exon Skipping and Dystrophin in the Gastrocnemius





Percent Force After 10 Contractions



$p \leq 0.01$, *≤ 0.001, ****$p \leq 0.0001$ vs. Vehicle

del51hDMD.*mdx* mice were treated with a single IV injection of ENTR-601-50 or vehicle. Human *DMD* exon 50 skipping (top) and dystrophin protein expression (bottom) were analyzed in the gastrocnemius, diaphragm and heart 2 weeks post-dose; Percent dystrophin protein restoration is normalized to total protein and normalized to hDMD.*mdx* controls; Data shown as mean ± standard deviation; One-way ANOVA was used for statistical comparison; ECC-induced muscle force loss generated by repeated ECC contraction of the gastrocnemius muscle was assessed 2 weeks after dosing. Data (mean ± standard deviation) are shown across ten ECC contractions normalized as the percentage of force retained after the tenth contraction; Data presented at the 2025 World Muscle Society conference; **IV**: Intravenous.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