Clinical Trial of ENTR-601-44, an Endosomal Escape Vehicle (EEV™)-Oligonucleotide Conjugate for the Treatment of Duchenne Muscular Dystrophy

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entrada THERAPEUTICS

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This presentation contains forward-looking statements that involve substantial risks and uncertainties. All statements, other than statements of historical facts, contained in this presentation, including statements regarding the Company's strategy, future operations, prospects and plans, objectives of management, the validation and differentiation of Entrada's approach and EEV platform and its ability to provide a potential treatment for patients, expectations regarding significant accumulation of exon skipping and dystrophin production in patients, expectations regarding the importance of endosomal escape to therapeutic index optimization, the translatability of the data from the Phase 1 clinical study for ENTR-601-44 to our planned DMD clinical studies, expectations regarding the ability of the Company's preclinical studies and clinical studies to demonstrate safety and efficacy of its therapeutic candidates, and other positive results, expectations regarding the Company's planned Phase 1/2 multiple ascending dose ("MAD") clinical studies of ENTR-601-44 and -45, including their study initiations in Q2 2025 and Q3 2025, respectively, expectations regarding the Company's planned global Phase 1/2 MAD clinical study of ENTR-601-50, including its initiation in Q4 2025, the ability to recruit for, enroll, and complete a global Phase 1/2 study for ENTR-601-44, -45, -50, and -51, the ability to recruit for, enroll, and complete a Phase 1b study for ENTR-601-44 in the US, expectations regarding the approvals and specific protocols for the Company's planned Phase 1/2 clinical studies for ENTR-601-44, -45, and -50, the timing of regulatory filings for the planned Phase 1/2 clinical studies for ENTR-601-50 in the second half of 2025 and ENTR-601-51 in 2026, candidate selection for ENTR-601-51 in December 2024, the potential of its EEV product candidates and EEV platform, including the potential for ENTR-601-44, -45, -50, and -51 to be transformative treatment options, the continued development and advancement of ENTR-601-44, -45, -50, and -51 for the treatment of Duchenne and the partnered product VX-670 for the treatment of myotonic dystrophy type 1, and the sufficiency of the Company's cash resources extending into 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. The Company 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; timing of and expectations regarding the Company's ability to submit and obtain regulatory authorization and initiate clinical studies; whether results from preclinical studies will be predictive of the results of later preclinical studies and clinical studies; whether earlier clinical data will be predictive of later clinical data; our ability to establish and maintain collaborations or strategic relationships; whether the Company's cash resources will be sufficient to fund the Company's foreseeable and unforeseeable operating expenses and capital expenditure requirements; as well as the risks and uncertainties identified in the Company's filings with the SEC, including the Company's most recent Form 10-K and in subsequent filings the Company may make with the SEC. In addition, the forward-looking statements included in this presentation represent the Company's views as of the date of this presentation. The Company anticipates that subsequent events and developments will cause its views to change. However, while the Company 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 the Company's views as of any date subsequent to the date of this presentation.

DMD, Duchenne muscular dystrophy; **EEV**, endosomal escape vehicle.

OUR MISSION:

To Treat Devastating Diseases With Intracellular Therapeutics

We're proud to share the stories of JJ, Andrew, Max and Franklin – all living with Duchenne muscular dystrophy

EEV™ PLATFORM

ENDOSOMAL ESCAPE VEHICLE (EEV™)-BASED THERAPIES

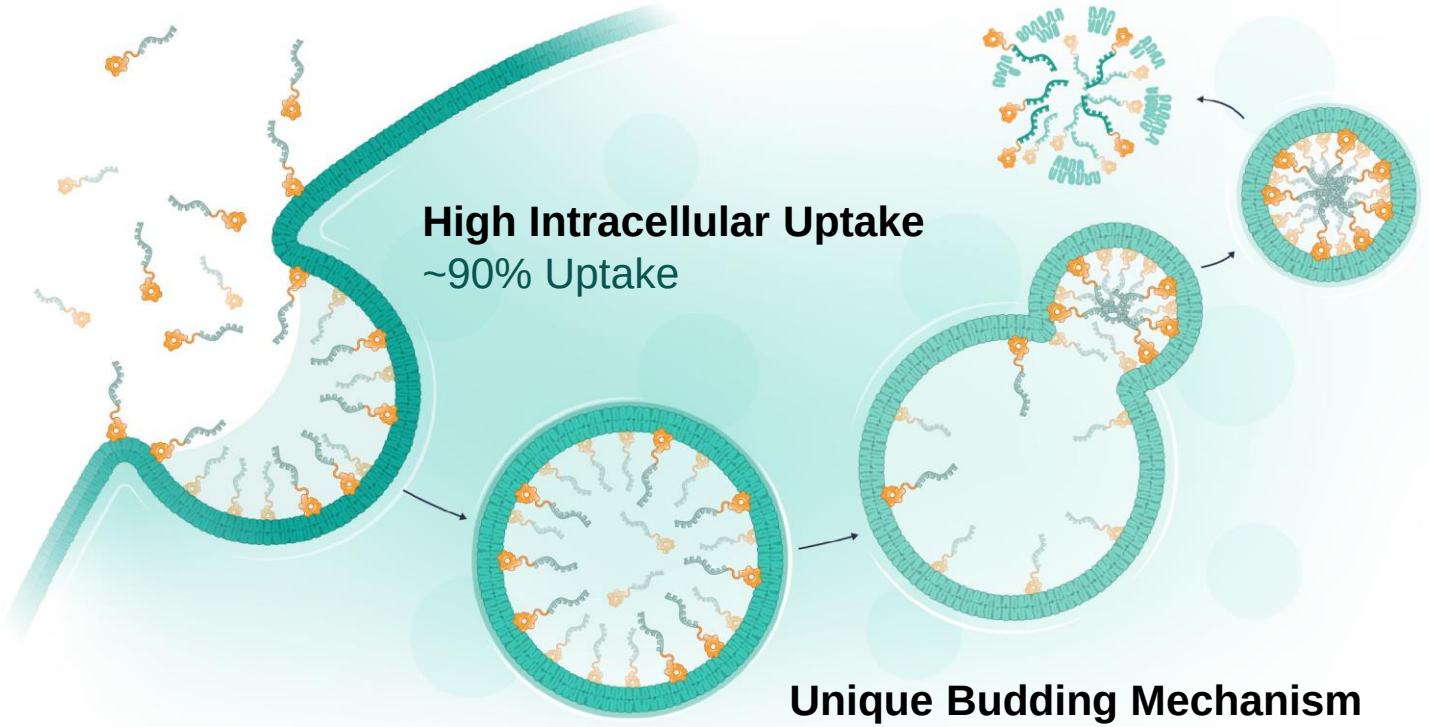
Unique chemistry
Improved uptake and endosomal escape

Cyclic structure
Extended half-life and increased stability

Phospholipid binding
Broad biodistribution to all cells

Consistent and predictable pharmacokinetics
Same EEV used across initial programs

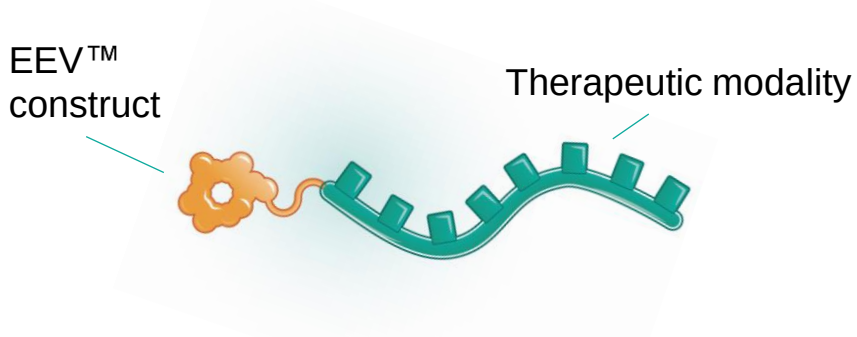
Efficient Endosomal Escape
~50% Escape vs. ~2% Standard



Qian, Z. et al. ACS Chem. Biol. 2013; Qian, Z. et al. Biochemistry 2014; Qian, Z. et al. Biochemistry 2016; Sahni, A. et al. ACS Chem. Biol. 2020; Pei, D. Acc. Chem. Res. 2022.

EEV LIBRARY: SCREENING AND OPTIMIZATION

Discovery Engine for Intracellular Therapeutics



- Cyclic peptide library design and combinatorial synthesis to generate **EEV library**
- Delivery and counter-screening assays enabled for in vitro **high throughput screening**
- Functional screening of lead EEVs in vivo to select for **pharmacodynamic activity** in target tissues
- Optimize **conjugation chemistry** for desired therapeutic modality

Screening Cascade for EEV Candidates

Chemically-diverse >500 member EEV library

EEVs with robust target cell uptake and efficacy

In vitro **functional validation** in relevant cell types with therapeutic payload

Well-tolerated EEVs with desired tissue functional delivery

Assess in vivo **functional delivery** in wild-type and disease models

Fit-for-purpose EEV candidate for target indication

Identify EEV candidate with desired **therapeutic profile**

EEV, endosomal escape vehicle.

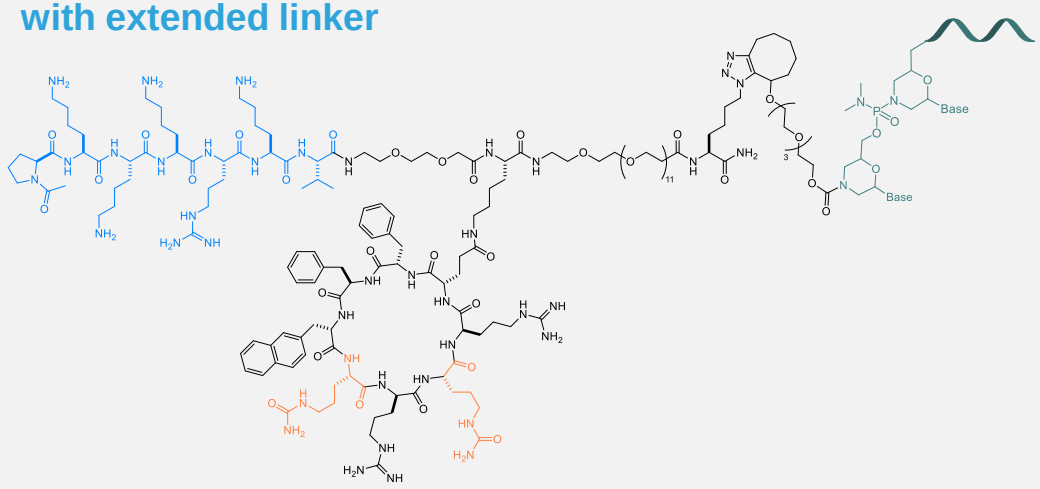
OPTIMIZATION OF EEV FOR MUSCLE DELIVERY

Rational substitution of cationic residues with a surrogate results in robust functional delivery to skeletal and cardiac muscle

EEV3-PMO654 Structure and Medicinal Chemistry

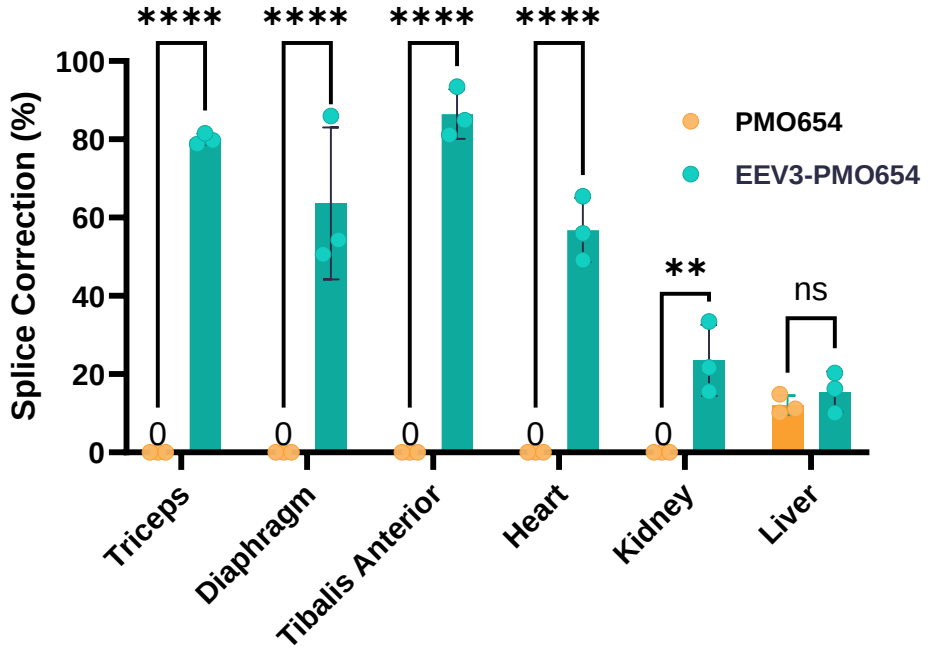
Exocyclic peptide sequence with extended linker

Conjugation with PMO



Substitution of positively charged arginine residues with neutral charged citrullines

Enhanced Functional Delivery to Muscle



- *EGFP654* mice were evaluated for splice correction 7 days following three weekly 10 mg/kg IV injections of PMO654 or EEV3-PMO654

p<0.01, ****p<0.0001; values are shown as mean ± standard deviation. **EEV, endosomal escape vehicle; **IV,** intravenous; **ns**, not significant; **PMO**, phosphorodiamidate morpholino oligomer.

DUCHENNE MUSCULAR DYSTROPHY

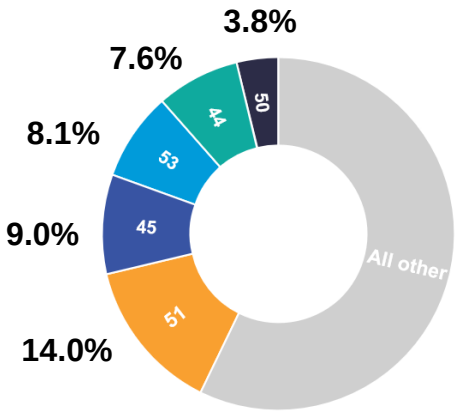
SIGNIFICANT THERAPEUTIC NEED EXISTS WITHIN A VALIDATED DUCHENNE MARKET

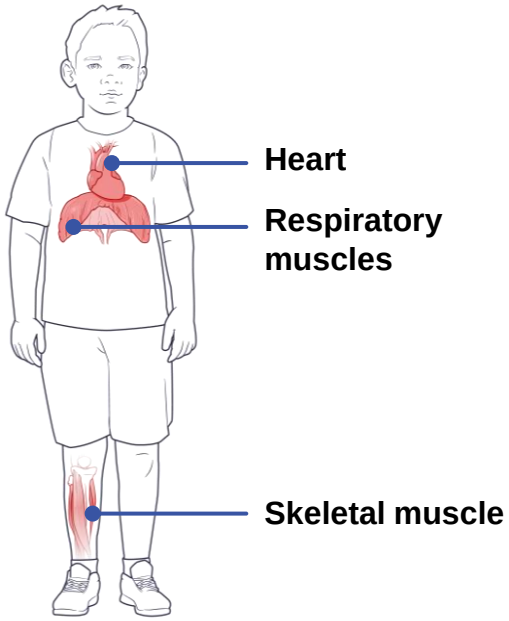
Duchenne is caused by **mutations in the *DMD* gene, which lead to a lack of functional dystrophin**, causing progressive loss of muscle function throughout the body

Exon skipping therapeutics have been approved based on **modest improvement in dystrophin levels ranging from ~1% to 6%**[4-7]

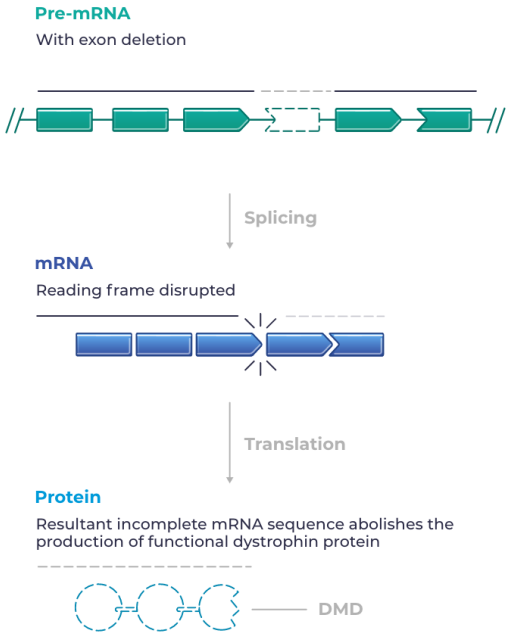
Approximately **41,000** people in the **U.S.**[1] **and Europe**[2] have Duchenne

>40% of patients with Duchenne[3] have mutations amenable to exon skipping of exons 44, 45, 50, 51 and 53

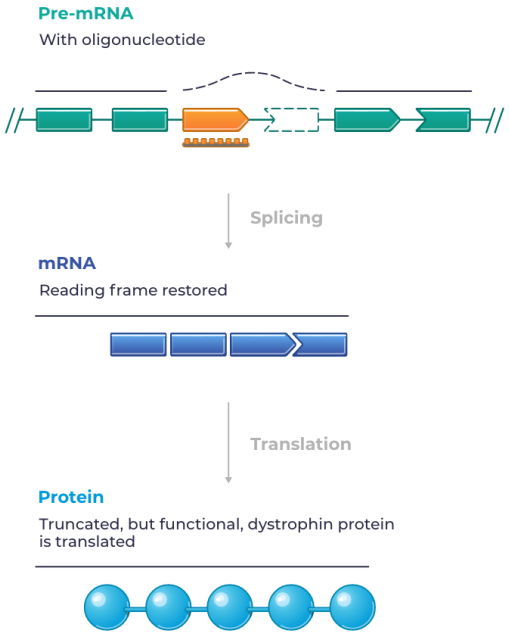




Patients with Duchenne



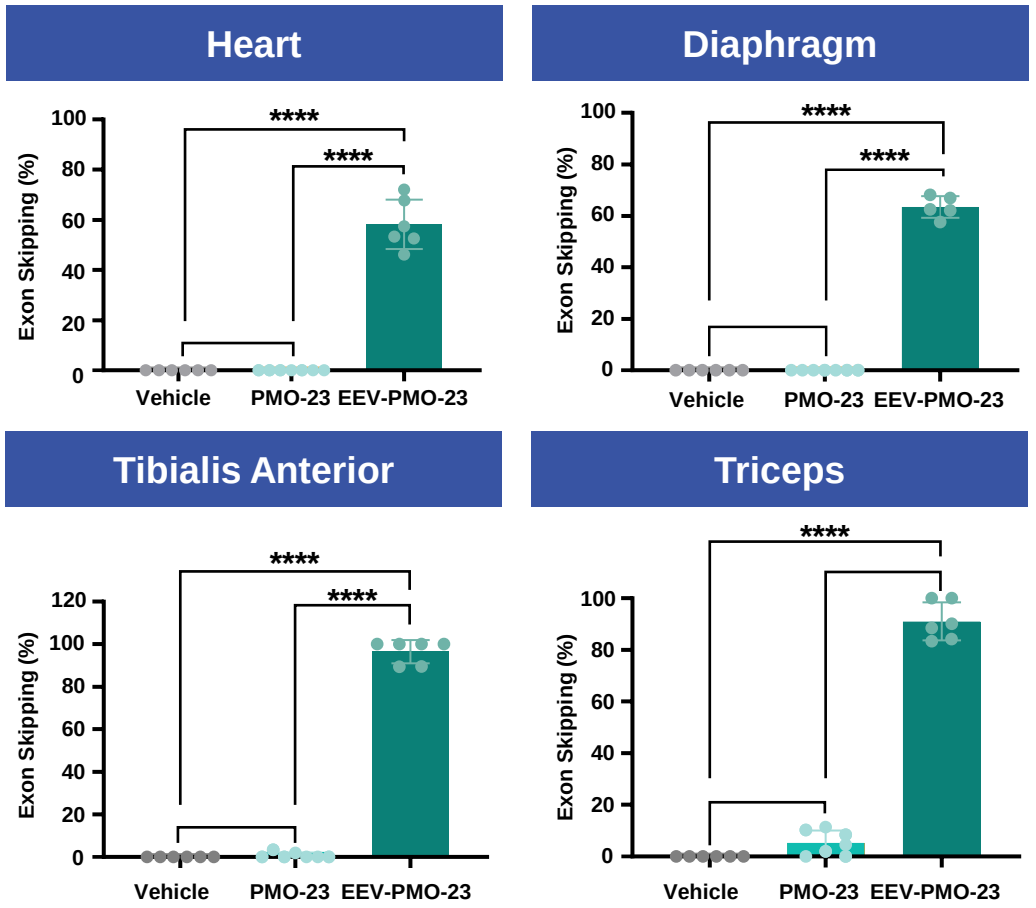
EEV-Oligonucleotide Approach



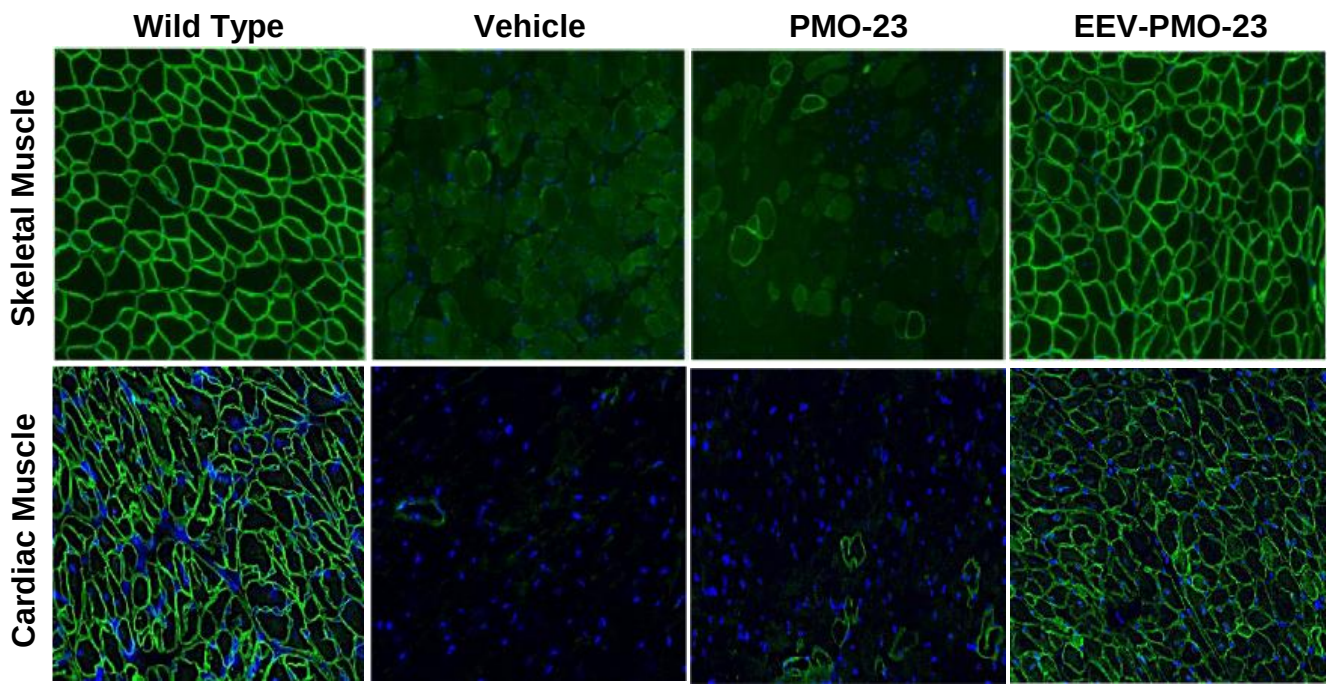
1. Parent Project Muscular Dystrophy. https://www.parentprojectmd.org/about-duchenne/. Accessed August 18, 2023. **2.** Europeans Medicines Agency. https://www.ema.europa.eu/en/medicines/human/orphan-designations/eu3202375. Accessed August 18, 2023. **3.** Bladen, C.L. et al. *Hum Mutat*. 2015. **4.** AMONDYS 45 PI. **5.** VILTEPSO PI. **6.** VYONDYS 53 PI. **7.** EXONDYS 51 PI. **DMD**, Duchenne muscular dystrophy; **EEV**, endosomal escape vehicle

REPEAT EEV-PMO-23 TREATMENT IN D2-*mdx* MICE

Robust exon 23 skipping after 4 monthly IV doses of EEV-PMO-23 in D2-*mdx* mice



Broad dystrophin expression and restoration of muscle integrity after four monthly IV doses of EEV-PMO-23 in D2-*mdx* mice



- D2-*mdx* mice (male, n=6-7) were treated with 4 monthly doses of either vehicle, 20 mg/kg unconjugated PMO-23 or 20 mg/kg PMO-23 equivalent of EEV-PMO-23, and the data were collected ~4 weeks after the last dose.

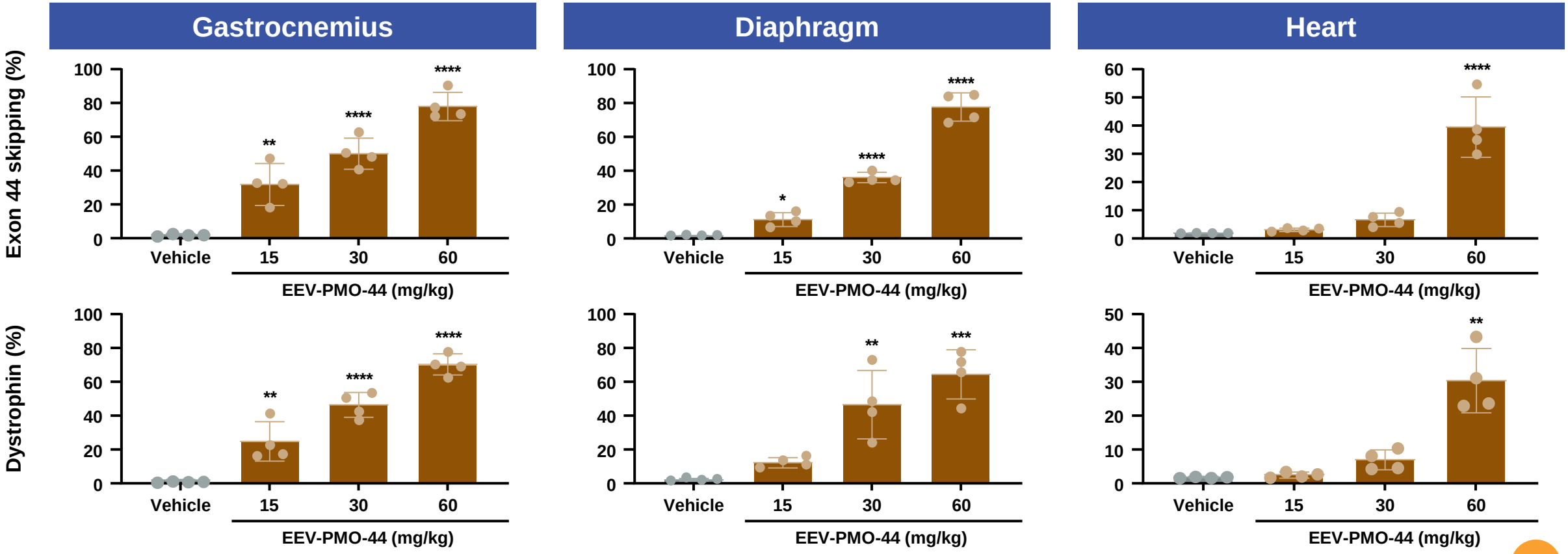
****p<0.0001. Values are shown as mean ± standard deviation. **D2-*mdx*** is a DMD mouse model with a nonsense mutation in *DMD* exon 23 (Coley, W.D. et al. *Hum. Mol. Genet*. 2016). Li, X. et al. *Mol. Ther. Nucleic Acids* 2023. **EEV**, endosomal escape vehicle; **DMD**, Duchenne muscular dystrophy; **IV,** intravenous; **ns**, not significant; **PMO**, phosphorodiamidate morpholino oligomer; **PMO-23**, mouse *DMD* exon 23 skipping phosphorodiamidate morpholino oligomer.

EEV-PMO PRECLINICAL STUDIES

EEV-PMO-44 EFFICACY IN del45hDMD.*mdx* MICE

A single dose of EEV-PMO-44 produced robust human *DMD* exon 44 skipping and dystrophin production 2 weeks post-dose in mice amenable to exon 44 skipping

*$p \leq 0.05$, **$p \leq 0.01$, ***$p \leq 0.001$, ****$p \leq 0.0001$ vs. Vehicle



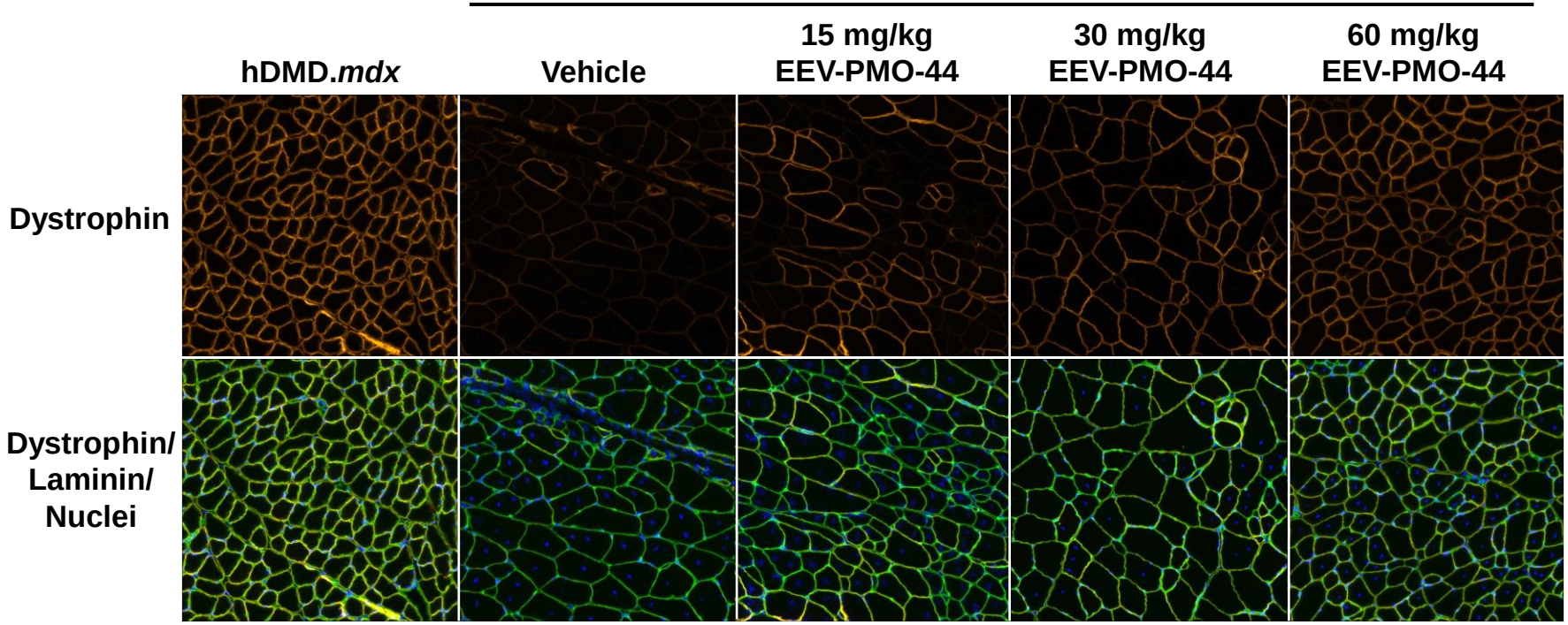
del45hDMD.*mdx* mice were treated with a single IV injection of EEV-PMO-44 (*DMD* exon 44 skipping EEV-oligonucleotide construct) or vehicle. Human *DMD* exon 44 skipping (top) and dystrophin protein expression (bottom) were analyzed in the gastrocnemius, diaphragm, and heart 2 weeks after the final 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; **ANOVA**, analysis of variance; **hDMD**, human dystrophin transgene; **IV**, intravenous.

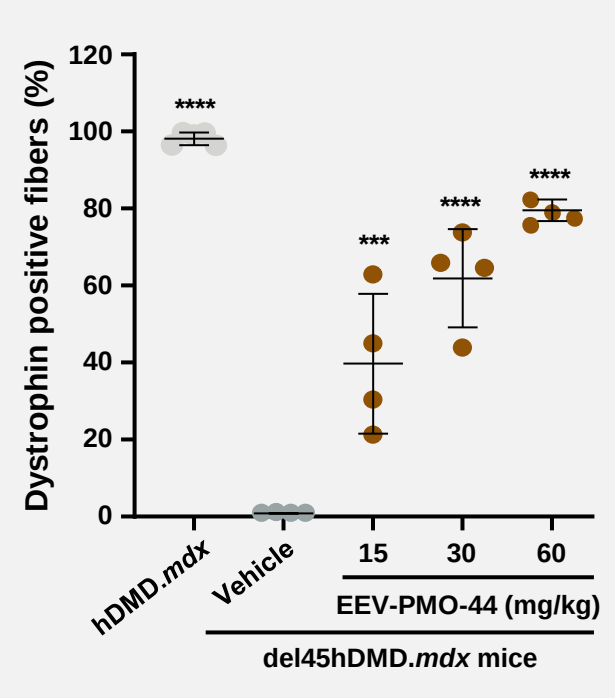
DYSTROPHIN LOCALIZATION WITH EEV-PMO-44 IN del45hDMD.*mdx* Mice

EEV-PMO-44 produced dose-dependent increases in dystrophin-positive muscle fibers localized to the sarcolemma of del45hDMD.*mdx* mice 2 weeks post-dose

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

del45hDMD.*mdx* Skeletal Muscle





- del45hDMD.*mdx* mice were treated with a single IV dose of EEV-PMO-44 or vehicle. Dystrophin protein distribution and cellular localization were analyzed by immunofluorescence in the gastrocnemius 2 weeks post-dose.

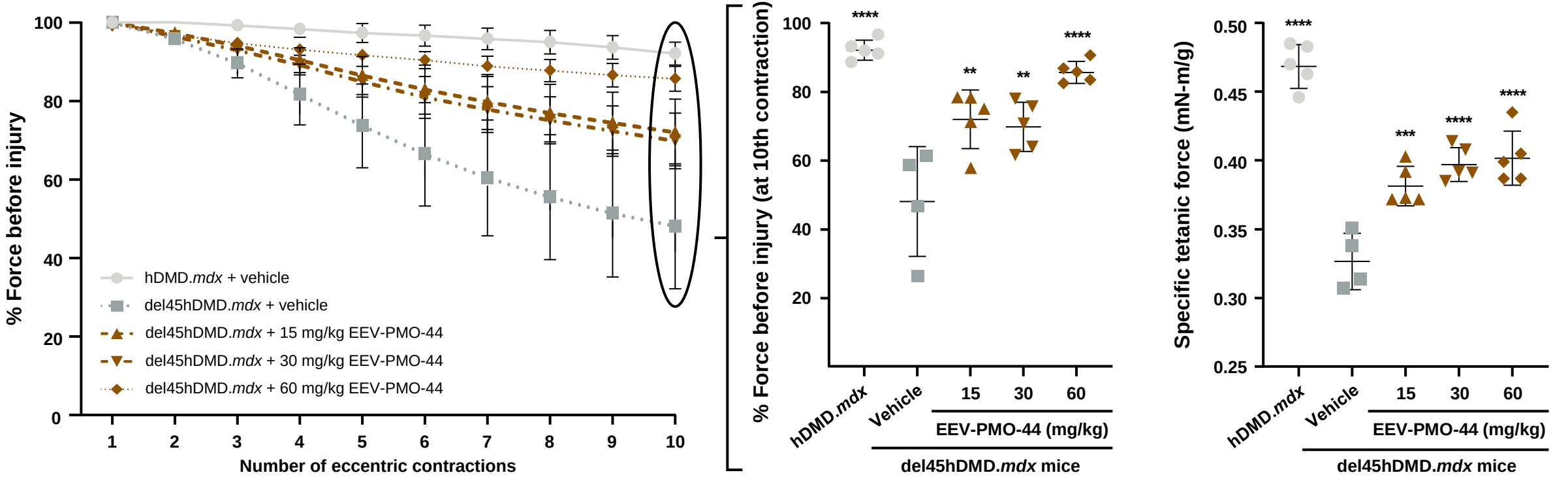
Quantification via Halo Image Analysis Software is shown as the percentage of dystrophin-positive muscle fibers relative to the total number of muscle fibers as determined by laminin staining (green) and dystrophin staining (red); co-localization to the sarcolemma appears yellow and nuclei appear blue. Data shown as mean ± standard deviation. One-way ANOVA was used for statistical comparison; **ANOVA**, analysis of variance; **hDMD**, human dystrophin transgene; **IV**, intravenous.

EEV-PMO-44 IMPROVES MUSCLE FUNCTION IN del45hDMD.*mdx* Mice

A dose-dependent increase in resistance to membrane damage was observed following the tenth contraction, as well as an increase in tetanic force 2 weeks post-dose of EEV-PMO-44

***p*<0.01, ****p*<0.001, *****p*<0.0001 vs. Vehicle

Skeletal Muscle Membrane Stability

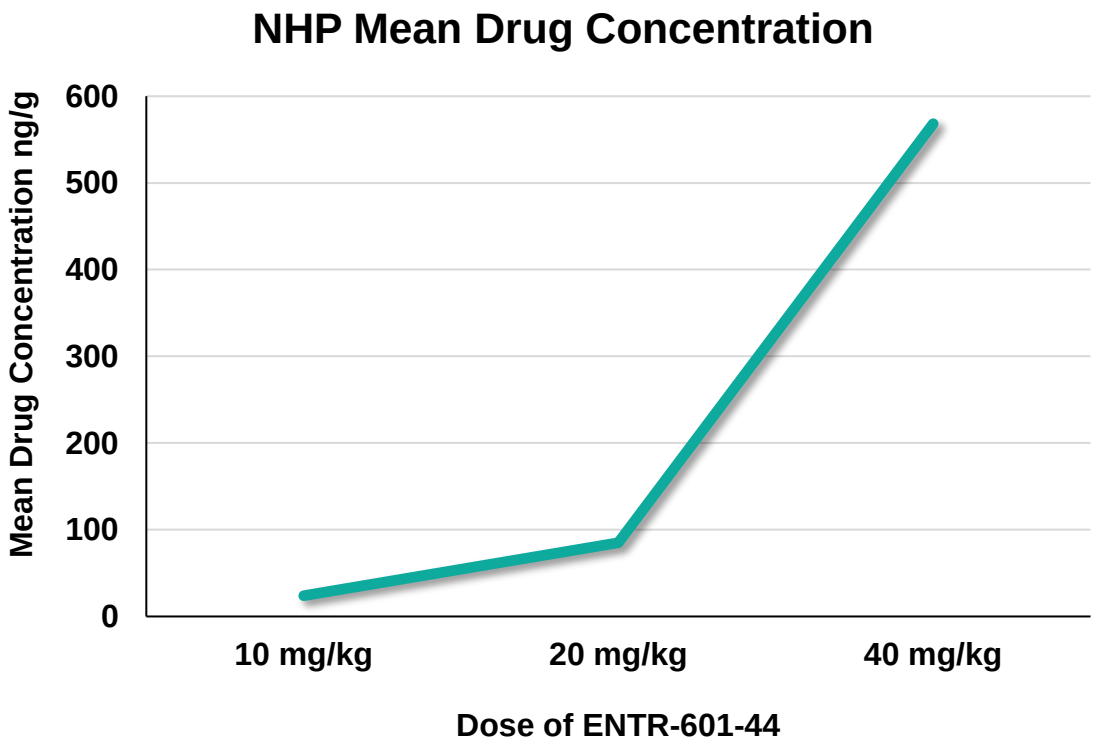


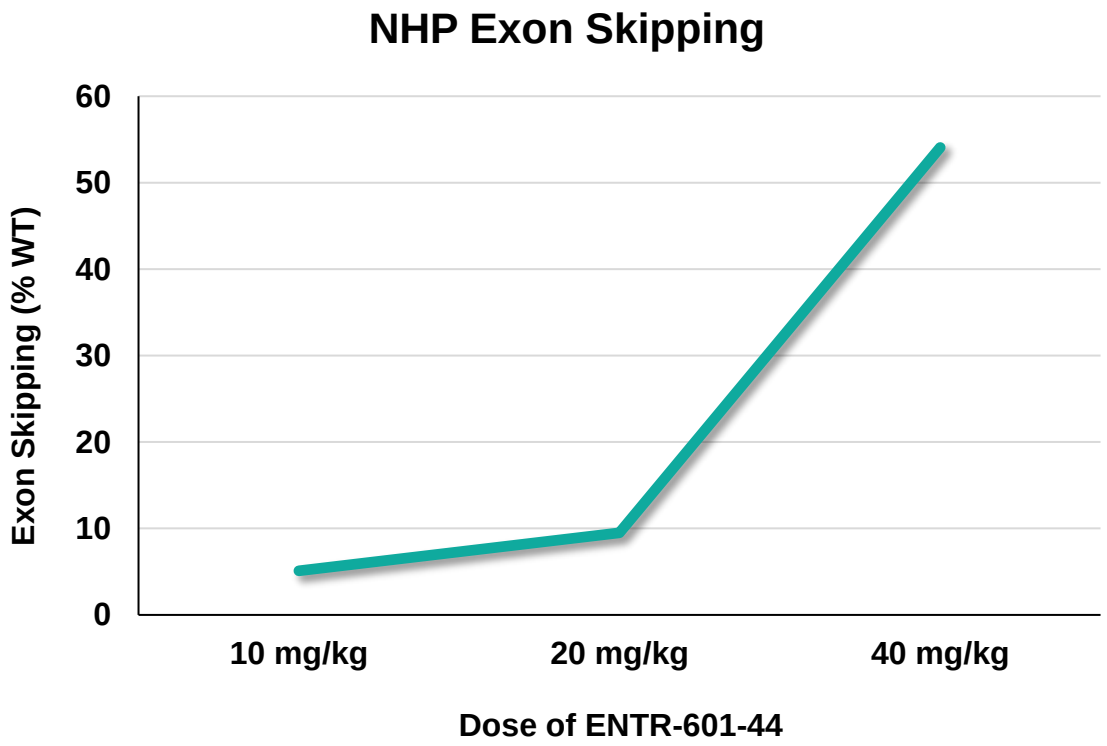
Specific Tetanic Force

del45hDMD.*mdx* mice were treated with a single IV injection of EEV-PMO-44 or vehicle. ECC-induced muscle force loss generated by repeated ECC contraction and tetanic force of the gastrocnemius muscle was assessed 2 weeks post-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 10th contraction. Vehicle-treated hDMD.*mdx* mice were used as a control group for normal muscle function. One-way ANOVA was used for statistical comparison to vehicle-treated del44hDMD.*mdx* mice. **ECC**, eccentric force; **hDMD**, human dystrophin transgene; **IV**, intravenous.

DOSE-DEPENDENT PK/PD WITH ENTR-601-44 IN NHP

NHP data demonstrated exponential increases in exon skipping at higher doses;
A close correlation between drug concentration and exon skipping was observed



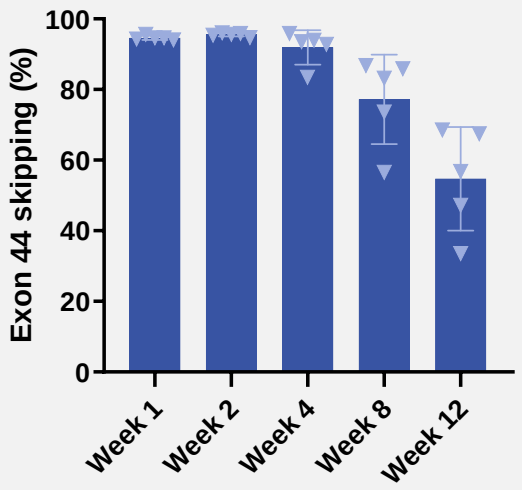


Non-human primates were administered a single intravenous dose of ENTR-601-44. Bicep biopsy was taken at 48 hours post-infusion; *R^2=0.9996; **PK**: pharmacokinetics; **PD**: pharmacodynamics; **ENTR-601-44** is a *DMD* exon 44 skipping EEV-oligonucleotide construct; **NHP,** Non-human primates.

CONSISTENT AND DURABLE EFFICACY OF EEV-PMO WAS DEMONSTRATED ACROSS SPECIES

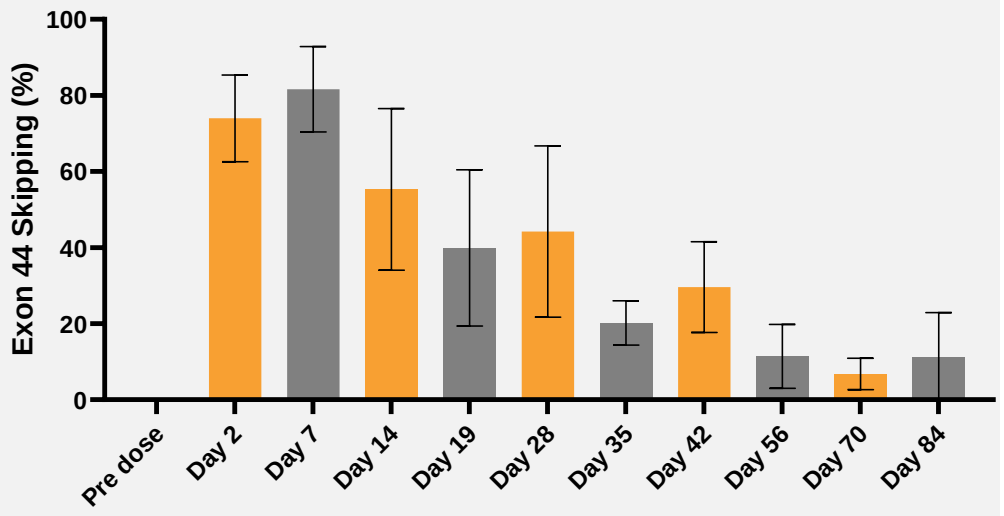
Significant patient benefit is implied by data in the mouse and the monkey at clinically relevant levels; *in vitro* data suggests much higher target engagement in patient cells

Exon 44 Skipping in hDMD Mouse



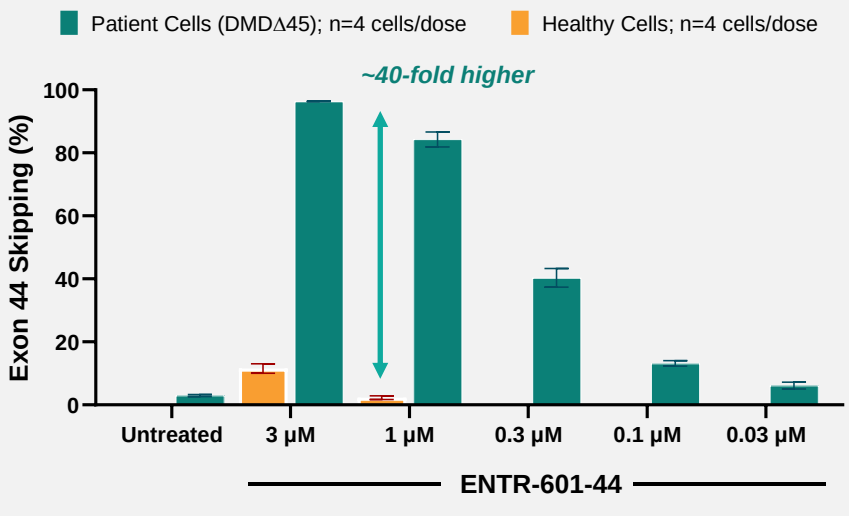
- Single IV 80 mg/kg dose of ENTR-601-44
- Tibialis Anterior

Exon 44 Skipping in Monkey



- Post IV infusion of single 45 mg/kg dose of ENTR-601-44**,** robust exon 44 skipping observed in biceps of treated monkeys (n=3 per cohort) for at least 12 weeks

Exon 44 Skipping in Healthy and Patient-Derived Muscle Cells

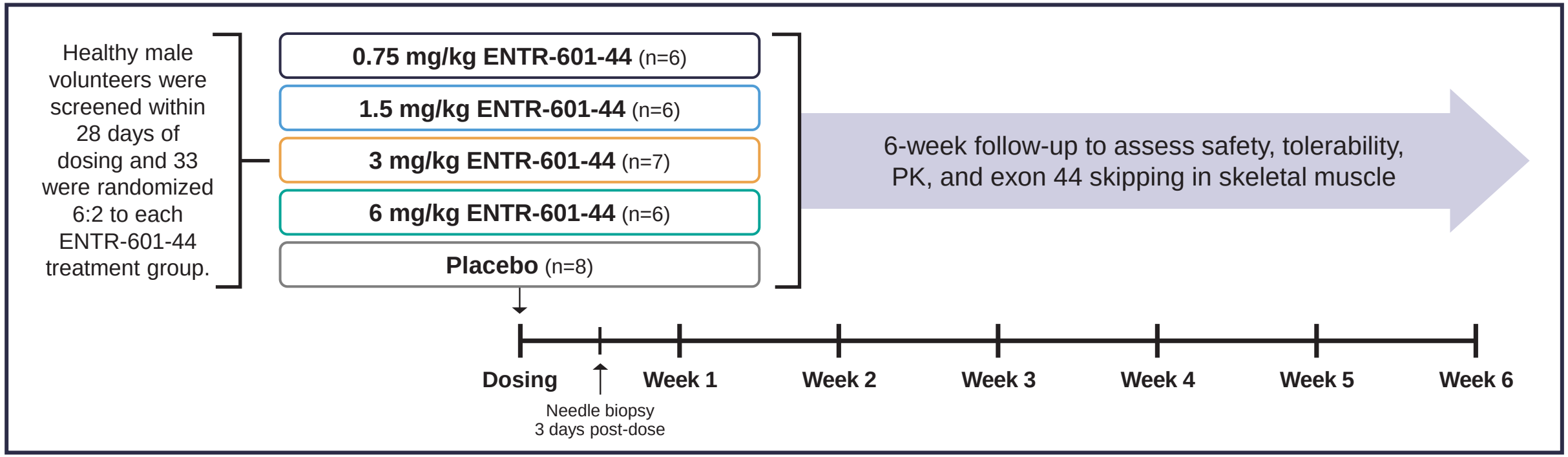


- Robust dose-dependent exon 44 skipping was observed in DMD patient-derived muscle cells harboring an exon 44 skip-amenable mutation

hDMD transgenic mice (left) express full-length human dystrophin gene ('t Hoen, A.C. et al. *J. Biol. Chem.* 2008). **DMDΔ45** (right) are immortalized myoblasts from DMD patients harboring an out-of-frame exon 45 deletion and further differentiated into myotubes. Values are shown as mean ± standard deviation. **ENTR-601-44** is a *DMD* exon 44 skipping EEV-oligonucleotide construct. **DMD**, Duchenne muscular dystrophy; **hDMD,** human Duchenne muscular dystrophy; **IV,** intravenous.

ENTR-601-44-101 PHASE 1 STUDY

ENTR-601-44-101: STUDY DESIGN



Key Inclusion Criteria

- Healthy males aged 18–55 years, inclusive.
- Body mass index (BMI) of 18.0 to 32.0 kg/m², inclusive, and a minimum weight of 50 kg at screening.

Key Exclusion Criteria

- No current or prior history of clinically significant illness, organ transplant, cardiac disease, hypertension, long QT syndrome, hepatitis B, or diabetes.

Volunteers were required to stay overnight between dosing and Week 1, as well as 1 day at Weeks 2, 3, and 4 study visits. Volunteers underwent a needle biopsy on the biceps brachii 3 days after dosing. Safety, tolerability, and PK parameters were assessed at each study visit. **IV**, intravenous; **PK**, pharmacokinetics. **BMI**, body mass index; **PD**, pharmacodynamics; **PK**, pharmacokinetics; **QoL**, quality of life.

ENTR-601-44-101: SAFETY AND TOLERABILITY

A single IV dose of ENTR-601-44 was well-tolerated in healthy human volunteers up to a dose of 6 mg/kg. No treatment-related adverse events were reported in the study.

- No AEs related to study drug
- Most common AE was headache (n=7; 5 mild and 2 moderate)
- No clinically significant findings with lab values, ECG or vital signs
- No adverse findings or clinically relevant changes to biomarkers of renal toxicity at highest dose of 6 mg/kg

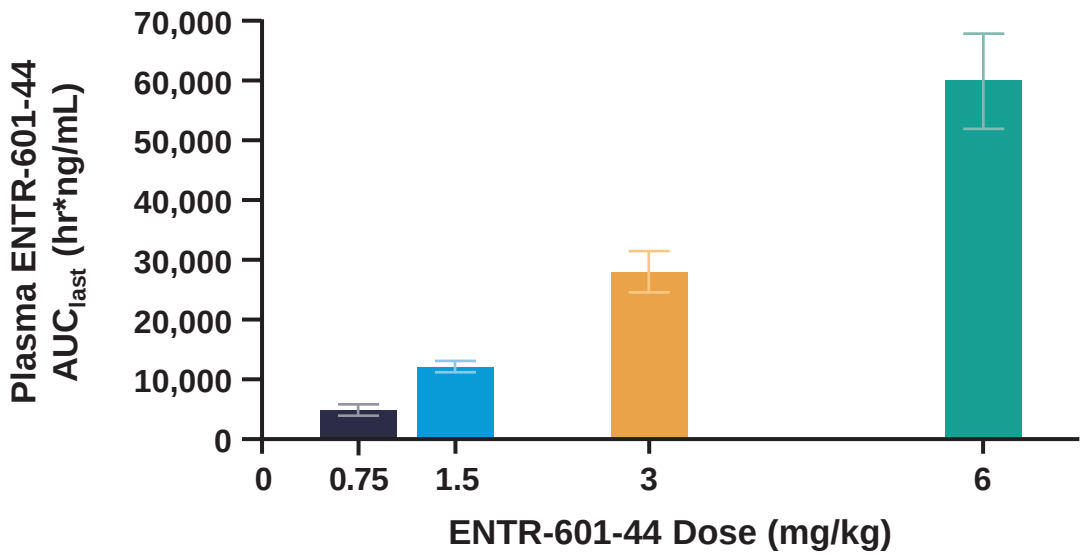
n (%)	Pooled placebo (N=8)	ENTR-601-44				
		0.75 mg/kg (n=6)	1.5 mg/kg (n=6)	3.0 mg/kg (n=7)	6.0 mg/kg (n=6)	Total (N=25)
Randomized	8 (100)	6 (100)	6 (100)	7 (100)	6 (100)	25 (100)
Dosed	8 (100)	6 (100)	6 (100)	6 (85.7)	6 (100)	24 (96)
Completed study	8 (100)	6 (100)	6 (100)	6 (85.7)	6 (100)	24 (96)
Any TEAE	1 (12.5)	5 (83.3)	2 (33.3)	3 (50)	3 (50)	13 (54)
Treatment-related TEAE	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Severe AEs	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
SAEs	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)

Safety and tolerability were assessed at each study visit following a single IV dose of ENTR-601-44 or placebo. One participant enrolled and randomized into Cohort 3 was removed prior to dosing. Renal biomarkers assessed using FNIH and the C-Path. Kidney Safety CM Biomarker User's Guide v1.1, 2019. **AE**, adverse event; **SAE**, serious adverse event; **TEAE**, treatment-emergent adverse event.

ENTR-601-44-101: PHARMACOKINETICS

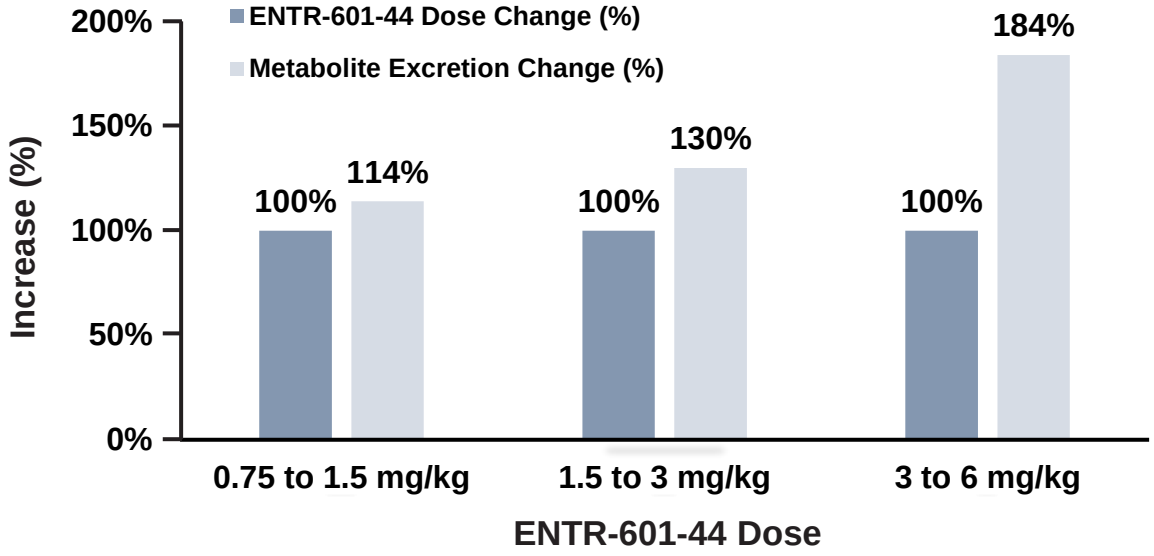
Pharmacokinetic analysis of ENTR-601-44 demonstrates dose-dependent increases in plasma concentration and urinary excretion.

Plasma Concentration



- Dose-dependent increase in mean C_{max} (range) of 3530 (2970–4530), 7380 (6750–8000), 15,400 (12,400–18,500), and 30,900 (26,300–34,200) ng/mL in the 0.75, 1.5, 3.0, and 6.0 mg/kg dose groups, respectively

Urinary Excretion of Final PMO-44 Metabolite



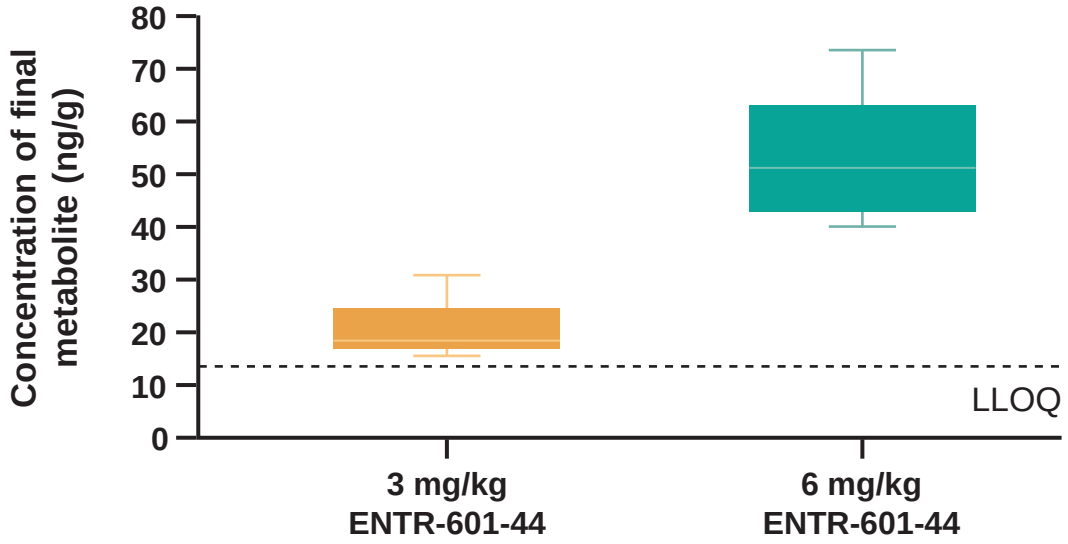
- Urinary excretion of the final metabolite is consistent with preclinical data, which demonstrate urinary excretion as the primary route of elimination.

(Left) Blood samples for PK assessment were collected at 2 hours pre-dose and post-end of infusion: 5 minutes, 1h, 4h, 8h, 16h, 24h, and every 24 hours after. Additional samples were taken at follow-up study visits. **(Right)** 24-hour urine samples for PK assessment were collected the day prior to dosing and every 24 hours after. Additional samples were taken at follow-up study visits. Data shown as mean ± standard deviation. **PMO**, phosphorodiamidate morpholino oligomer.

ENTR-601-44-101: MUSCLE CONCENTRATION AND EXON SKIPPING

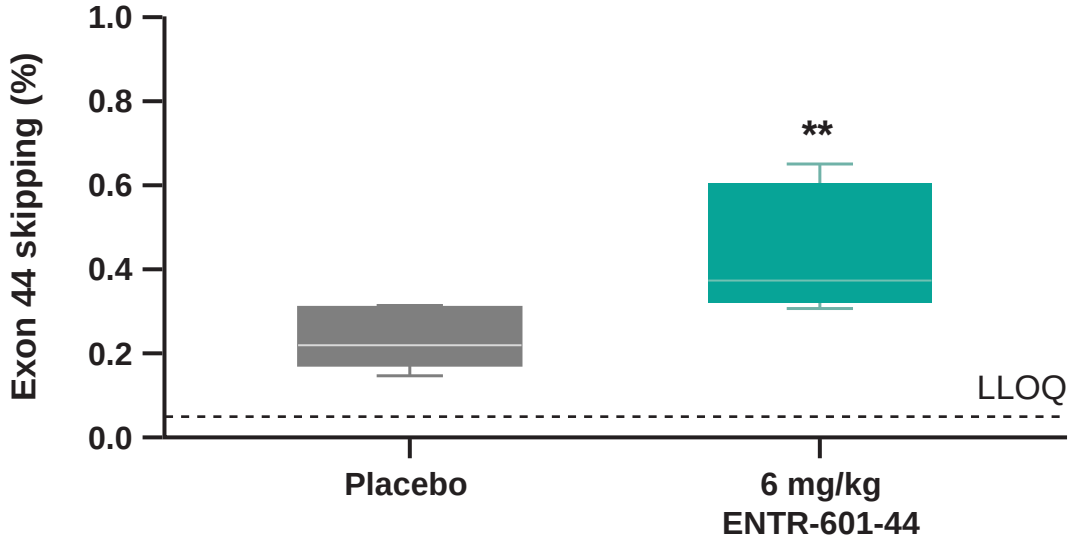
Dose-dependent increases in muscle concentration and *DMD* exon 44 skipping were observed 72 hours following a single IV dose of ENTR-601-44

Skeletal Muscle Concentration



- All six volunteers in the 6 mg/kg dose group had detectable levels of PMO-44 metabolite in skeletal muscle (mean 52.4 ng/g, range 40.0–73.5 ng/g)
- Concentrations of PMO-44 metabolite were below LLOQ in 3 of 6 volunteers in the 3 mg/kg dose group and all volunteers in the 0.75 and 1.5 mg/kg dose groups

DMD Exon 44 Skipping

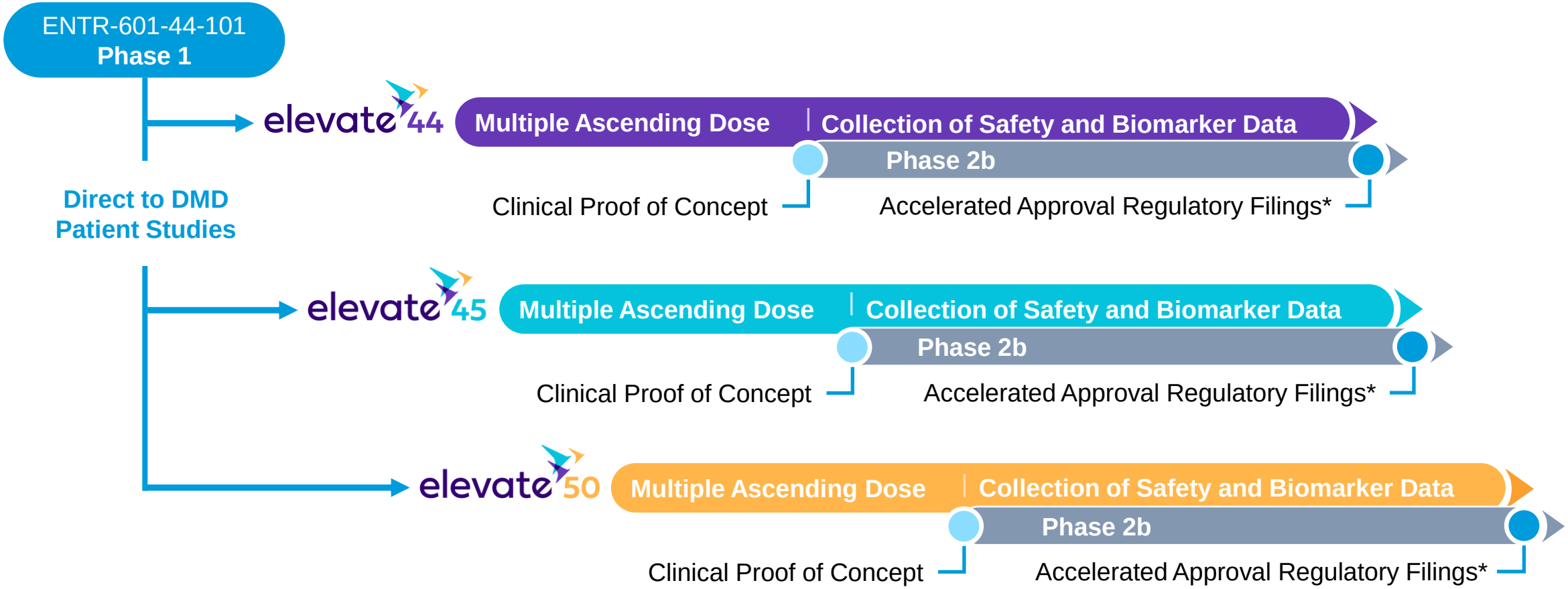


- Statistically significant *DMD* exon 44 skipping was observed with 6 mg/kg ENTR-601-44 (mean 0.44%, range 0.30%–0.65%) in comparison with placebo (mean 0.22%, range 0.14%–0.31%)
- No other ENTR-601-44 dose group was statistically significant in comparison with placebo.

Muscle concentrations and exon skipping were assessed using a needle muscle biopsy taken from biceps brachii 72 hours (±4 hours) post-dose of ENTR-601-44. Box and whisker plot illustration (right): the boxes represent the IQR and median. Whiskers show the smallest and largest values within 1.5 times the IQR. ** $p<0.005$ vs. placebo using Mann-Whitney U test. **IQR**, interquartile range; **LLOQ**, lower level of quantification; **PMO**, phosphorodiamidate morpholino oligomer.

CLINICAL STRATEGY IS DESIGNED FOR EFFICIENT REGULATORY PATH

All ENTR-601-series programs will follow a similar clinical and regulatory approach



Protocols pending regulatory feedback; *Potential for Accelerated Approval in the US, followed by confirmatory Phase 3 studies to obtain Full Approval in the US and ex-US countries.

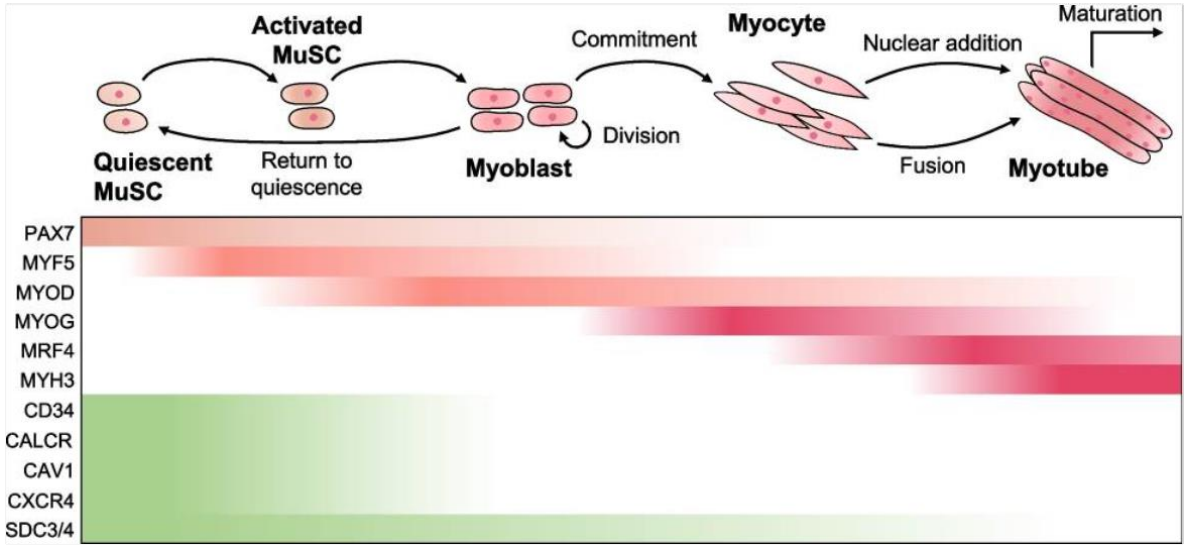
DELIVERY OF PMO TO SATELLITE CELLS

MUSCLE SATELLITE CELLS

Muscle satellite cells as a new therapeutic target with potential in several neuromuscular disorders

- **Satellite cells are muscle stem cells responsible for generating myoblasts for early muscle growth**
  - Mitotically quiescent in mature muscle, satellite cells maintain their own population by self renewal
  - In post-mitotic muscles, they can be activated to generate myoblast for homeostasis, repair, and hypertrophy
- **Quiescent satellite cells are historically challenging to access by therapeutic modalities**
- **EEV-mediated delivery to access quiescent satellite cells could enable early disease intervention**
  - Ability to deliver to myonuclei of muscle fibers and to quiescent satellite cells holds potential in several neuromuscular disorders
- **Currently evaluating satellite cell-opathies (primary and secondary) to identify attractive target-indication pairs, e.g., FSHD**

Developmental Stages of Muscle Satellite Cells



- **The developmental stages of muscle satellite cells can be delineated by various myogenic factors**
  - Pax7, a canonical myogenic marker essential for orchestrating proper muscle regeneration, is mainly expressed in **quiescent** state, and at a lower level in activated state
  - Stage-specific expression of myogenic factors provides tools for studying EEV-PMO uptake at different developmental stages of satellite cells

Image from Byun et al., Inflamm Regen, 2024

DISTRIBUTION OF EEV-PMO TO SATELLITE CELLS

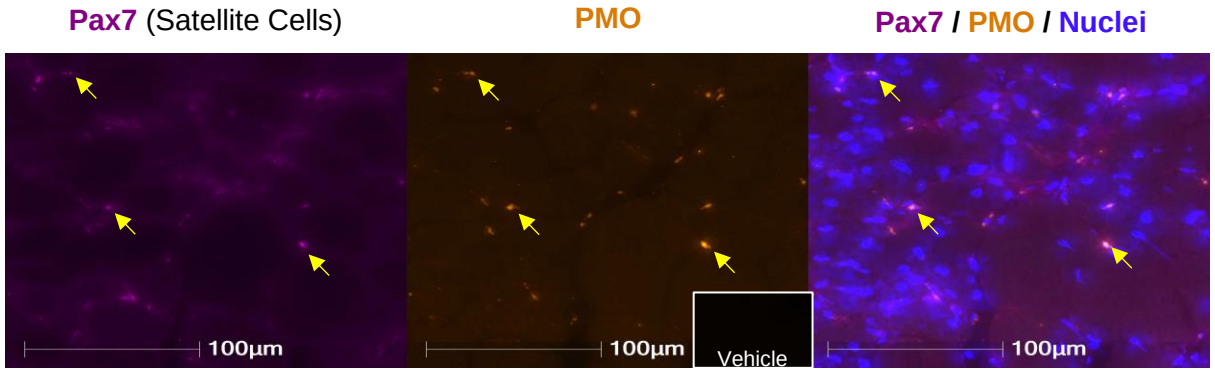
EEV-PMO shows 100% co-localization with quiescent satellite cells (Pax7 positive) at 48 hours; Qualitative data demonstrates that co-localization lasts at least 1-week post-dose

- **Two independent molecular techniques were utilized to determine EEV-PMO distribution within specific cell lineages across muscle tissue**
  - RNA-ISH: Highly selective and sensitive technique to assess specific cell lineages across muscle tissue
  - Immunohistochemistry Assessment
- **Quantification analysis of RNA-ISH data confirms that EEV-PMO is co-localized in 100% of satellite cells at 48 hours**
  - Quantitative assessment confirms qualitative data (data not shown)
- **Qualitative assessment of IHC data demonstrates co-localization of satellite cells in hDMD mice with EEV-PMO at 7 days**

PMO Distribution (48 hours, RNA-ISH Quantitation)

Treatment Group (D2-*mdx* mice)	% Pax7 Positive Cells	% Pax7 + PMO Positive Cells
Saline	1-10%	0%
EEV-PMO Treated	1-10%	1-10%

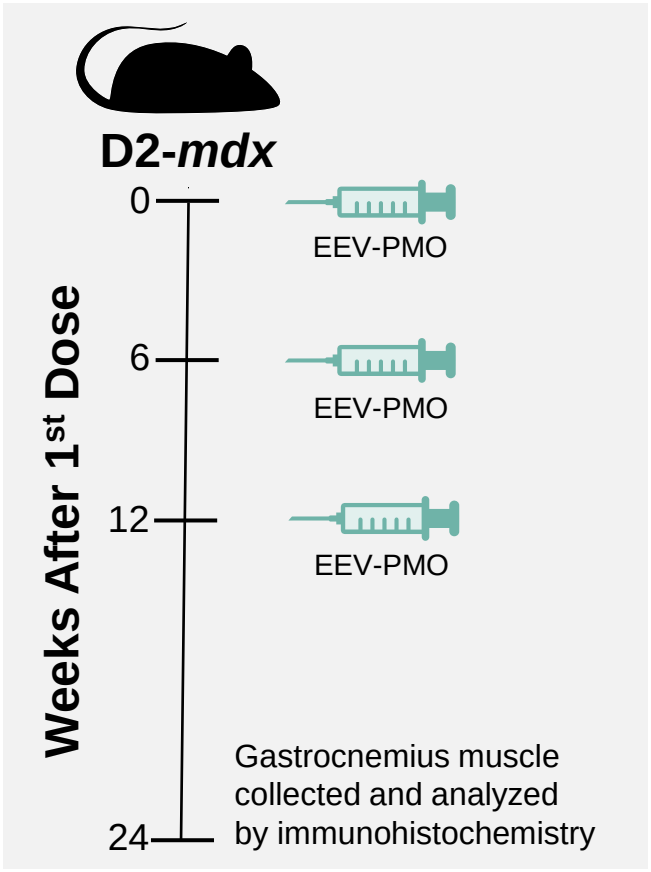
PMO Distribution in hDMD Mice (Day 7, IHC)

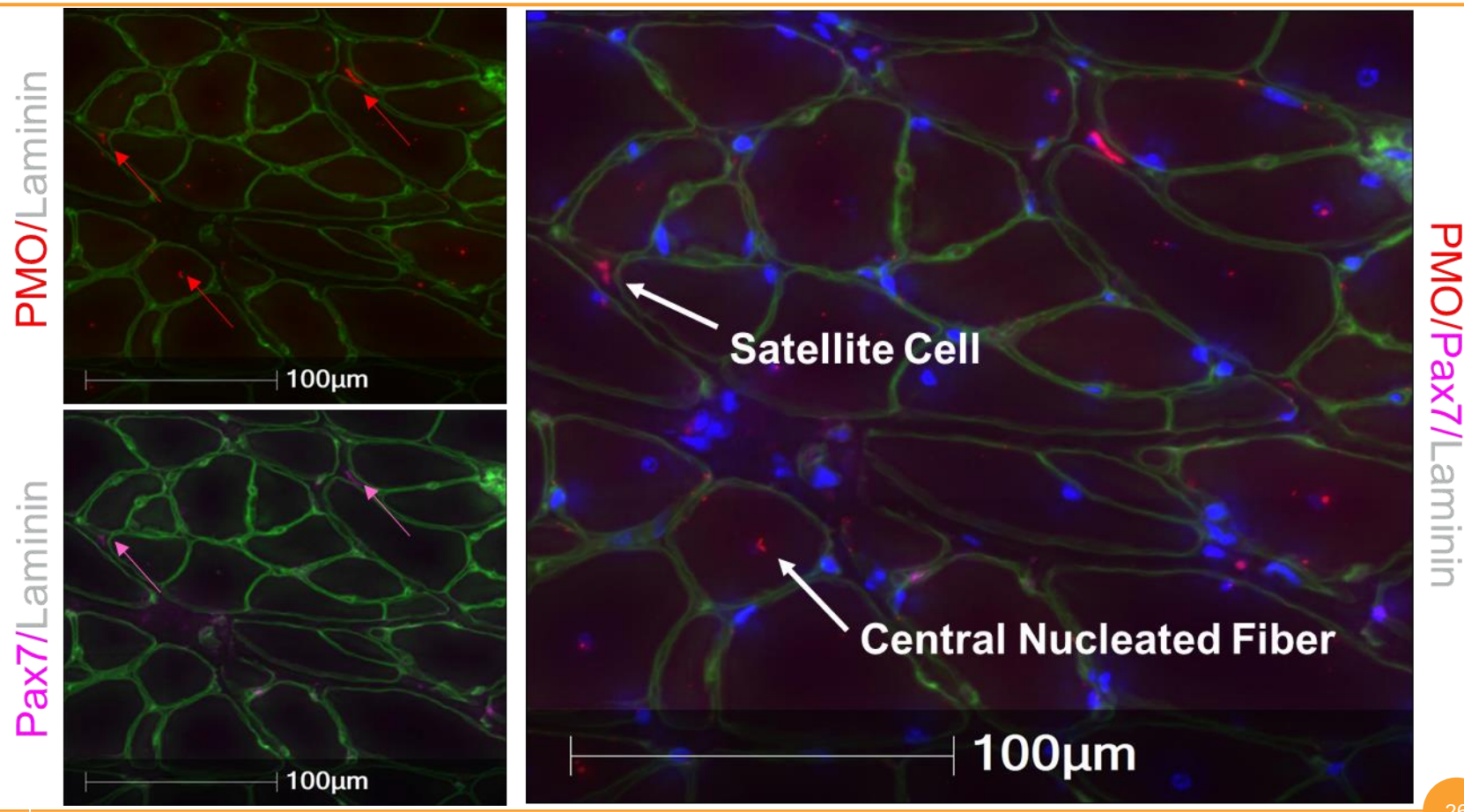


(Top right) D2-*mdx* mice were treated with a single IV dose of EEV-PMO; Gastrocnemius was collected at 48 hours and analyzed by RNA-ISH. (Bottom right) hDMD mice were treated with a single IV dose of EEV-PMO; Gastrocnemius was collected at 7 days and tissues analyzed by IHC. Pax7 is a satellite cell marker (Seale, P. et al *Cell* 2000). **hDMD,** human Duchenne muscular dystrophy; **IV,** intravenous; **RNA-ISH**: RNA *in situ* Hybridization image analysis; **IHC**: immunohistochemistry.

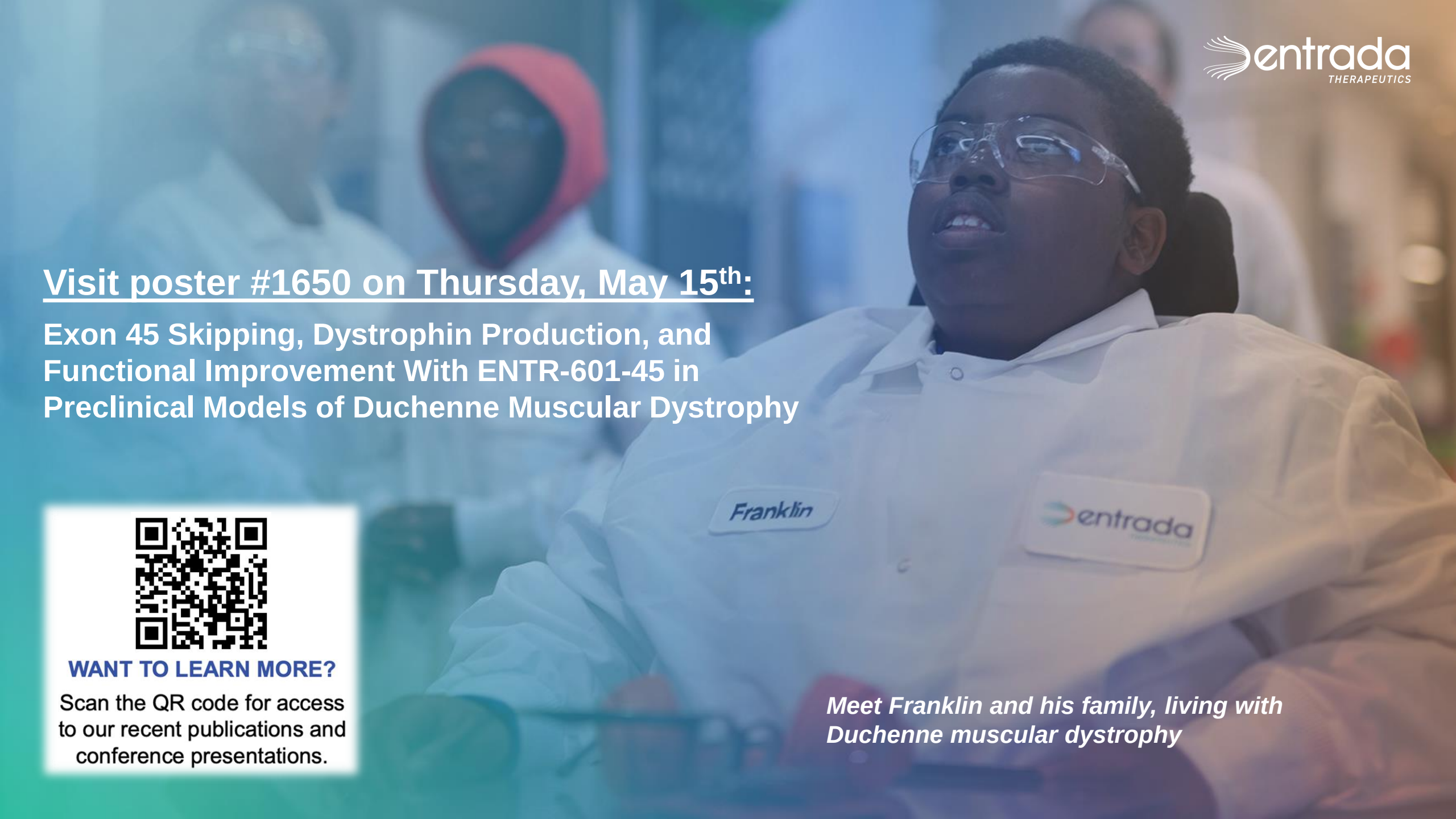
EEV-PMO PERSISTS IN SATELLITE CELLS 12 WEEKS AFTER FINAL DOSE

PMO co-localizes with satellite cells and newly regenerated centrally nucleated fibers 12 weeks post washout after 3 Q6W doses





D2-*mdx* mice were treated with 3 IV doses of EEV-PMO administered every six weeks; Gastrocnemius was collected 12 weeks after the 3rd dose and analyzed by immunohistochemistry. **Q6W**: every six weeks.



entrada THERAPEUTICS

Visit poster #1650 on Thursday, May 15th:

Exon 45 Skipping, Dystrophin Production, and Functional Improvement With ENTR-601-45 in Preclinical Models of Duchenne Muscular Dystrophy

WANT TO LEARN MORE?

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Meet Franklin and his family, living with Duchenne muscular dystrophy