Corporate Presentation

October 2024

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 translatability of the data from the Phase 1 clinical trial for ENTR-601-44 to future clinical trials for ENTR-601-44, expectations regarding the ability of the Company’s preclinical studies and clinical trials to demonstrate safety and efficacy of its therapeutic candidates, and other positive results, expectations regarding the starting dose for the Company’s planned Phase 2 clinical trial for ENTR-601-44, the timing of Phase 2 regulatory filings for ENTR-601-44 and ENTR-601-45 clinical trials in the fourth quarter of 2024, and ENTR-601-50 in 2025, the ability to recruit for and complete a global Phase 2 trial for ENTR-601-44, ENTR-601-45 and ENTR-601-50, the potential of its EEV product candidates and EEV platform, the continued development and advancement of ENTR-601-44, ENTR-601-45 and ENTR-601-50 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 trials; uncertainties as to the availability and timing of results from preclinical and clinical studies; the timing of and the Company's ability to submit and obtain regulatory clearance and initiate clinical trials; whether results from preclinical studies will be predictive of the results of later preclinical studies and clinical trials; whether earlier clinical data will be predictive of later clinical data; 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.

Our Mission

To Treat Devastating Diseases with Intracellular Therapeutics

entrada THERAPEUTICS

Meet Max and his family, living with Duchenne muscular dystrophy

Corporate Summary

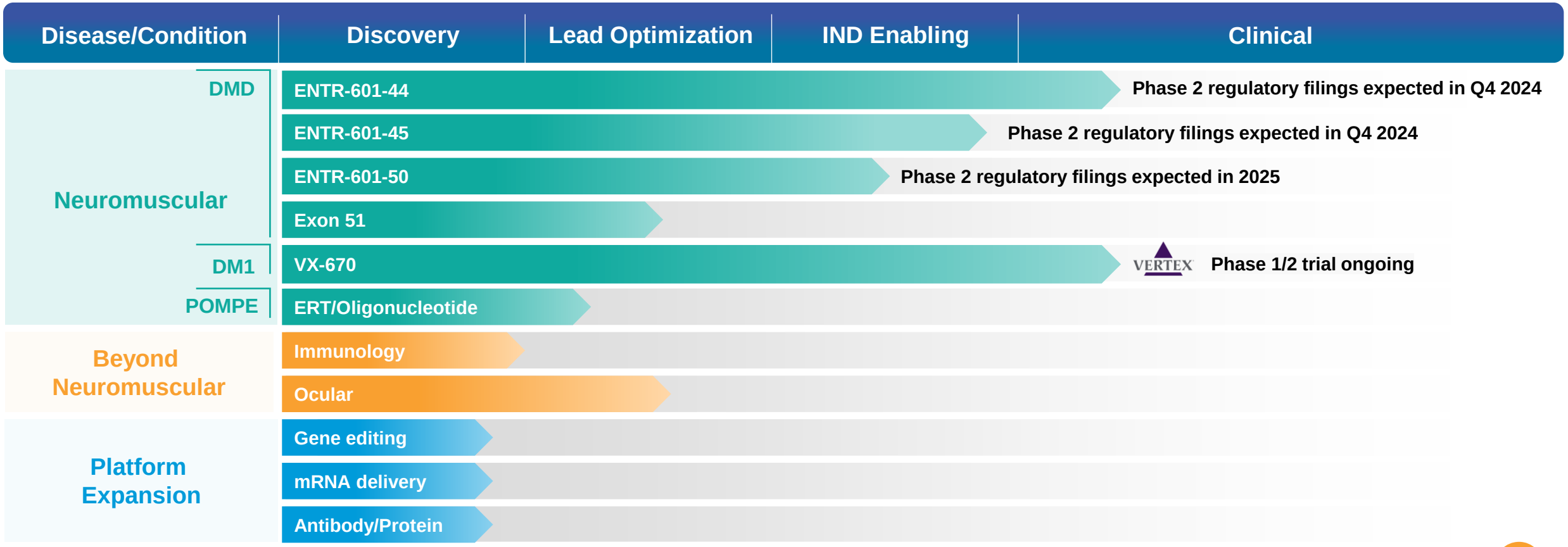
Entrada is leveraging its Endosomal Escape Vehicle platform (EEV™) to create a diverse and expanding development portfolio of RNA-, antibody- and enzyme-based therapeutics

- **Advancing new therapeutic options for people living with Duchenne muscular dystrophy (DMD)**
  - ENTR-601-44: Regulatory filings expected in Q4 2024 for global Phase 2 clinical trial in patients
    - Positive Phase 1 study shows dose-dependent response, significant plasma concentration, muscle concentration and exon skipping with no serious adverse events and no clinically significant changes in laboratory assessments
    - Data demonstrates the translation of ENTR-601-44's nonclinical studies to healthy volunteers
  - ENTR-601-45: Regulatory filings expected in Q4 2024 for global Phase 2 clinical trial in patients
  - ENTR-601-50: Regulatory filings expected in 2025 for global Phase 2 clinical trial in patients
- **Transformative Vertex partnership for the development of myotonic dystrophy type 1 (DM1)**
  - VX-670: Global Phase 1/2 clinical trial ongoing; Completion of SAD portion of the study expected by end of 2024
- **Expanding pipeline by leveraging new moieties and extending into new therapeutic areas**
- **Strong financial position with cash runway into 2027***

Note: All references in this presentation regarding planned regulatory filings and clinical trial designs are subject to ongoing discussion with US and international regulatory authorities; SAD: Single ascending dose; *Based on current operating plans and $469.7M in cash, cash equivalents and marketable securities as of June 30, 2024.

An Expanding Pipeline of Intracellular Therapeutics

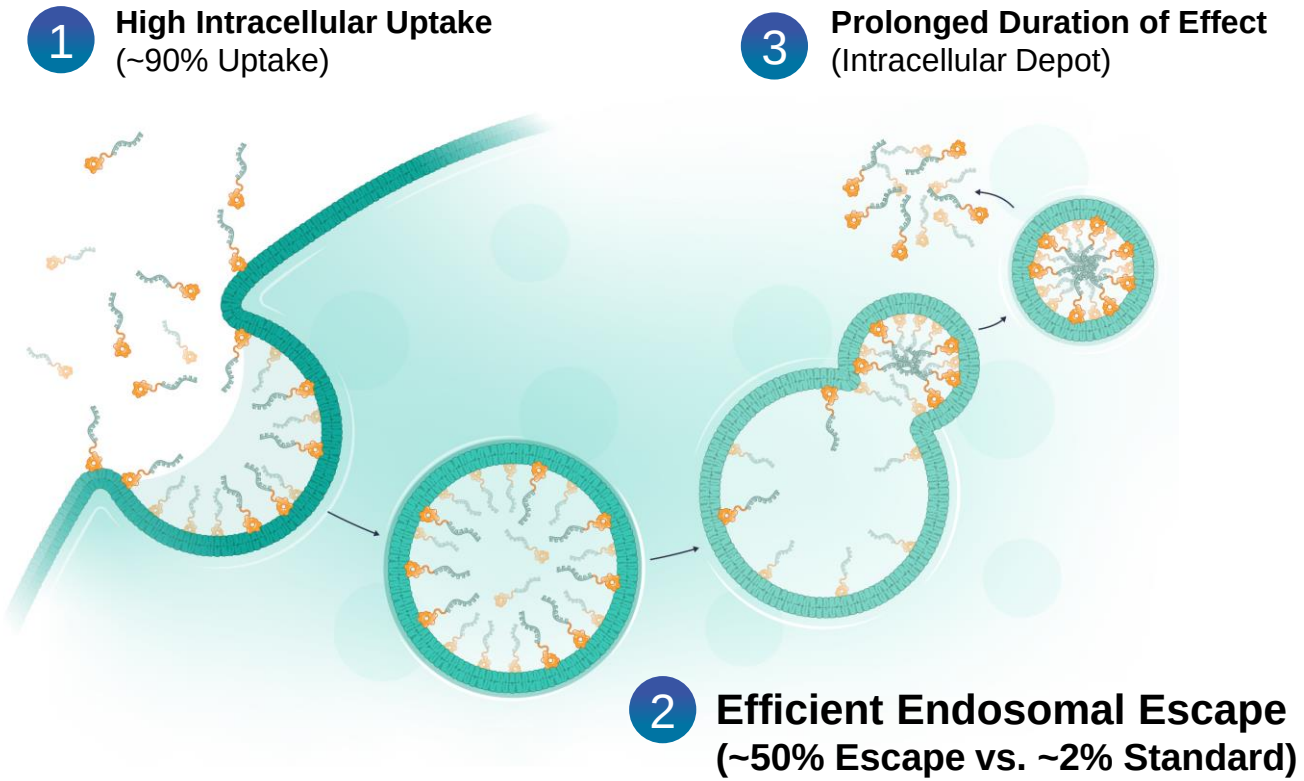
Entrada's pipeline includes a diverse array of high potential and high value assets;
Each target disease has a substantial patient population with a significant unmet medical need



Endosomal Escape Vehicle (EEV™) Therapeutics

- Unique chemistry results in **improved uptake and endosomal escape**
- Cyclic structure designed to **extend half life and increase stability**
- Phospholipid binding potentially **enables broad biodistribution to all cells**
- Mechanism of **internalization conserved across species**

Entrada solves a fundamental problem: A lack of efficient cellular uptake and escape from the endosome; Both are critical to intracellular target engagement and therapeutic benefit

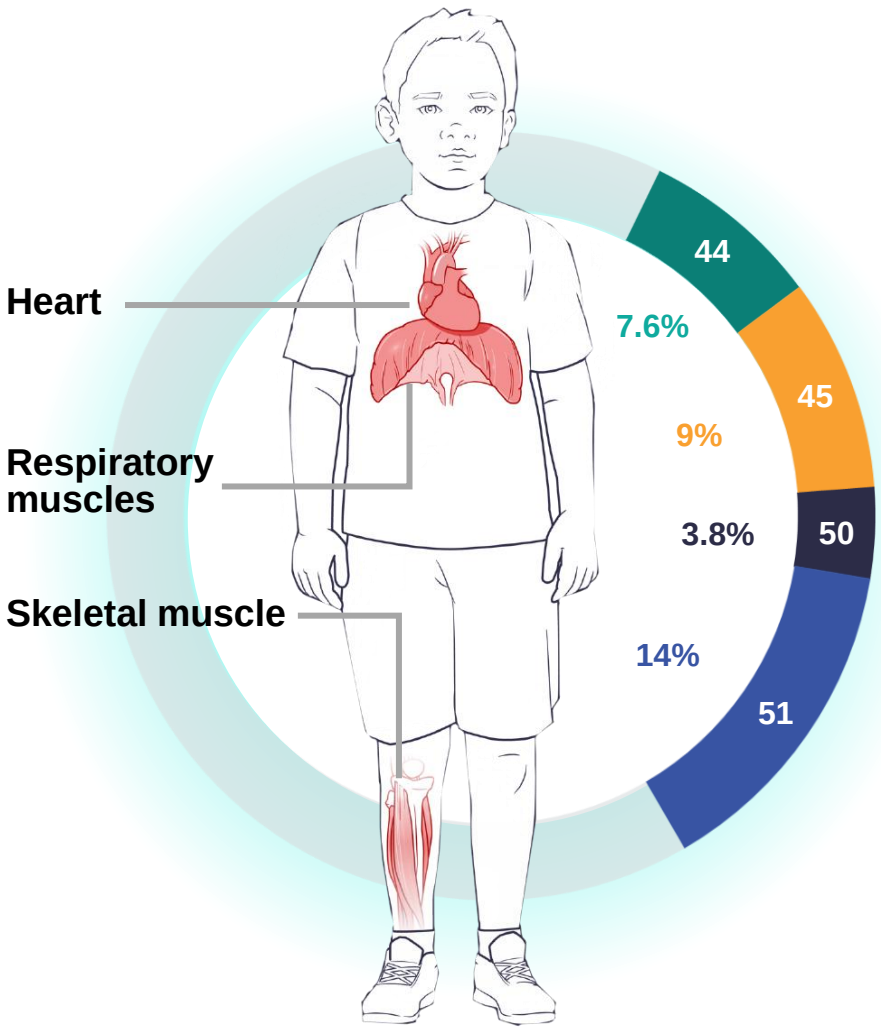


Qian, Z. et al. *ACS Chem. Biol.* 2013; Qian, Z. et al. *Biochemistry* 2014; Qian, Z. et al. *Biochemistry* 2016; Sahni, A. et al. *ACS Chem. Biol.* 2020; Pei, D. *Acc. Chem. Res.* 2022.

Duchenne Muscular Dystrophy (DMD)

Meet Franklin and his family, living with Duchenne muscular dystrophy

Duchenne: Significant Unmet Need



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

Progression generally leads to death via **cardiac and/or respiratory failure** in the third or fourth decade

~40,000

people in the **US and Europe** have Duchenne[1]

Duchenne Franchise

ENTR-601-44

Phase 1: Positive data reported in June 2024

Phase 2: Regulatory filings expected Q4 2024

ENTR-601-45

Phase 2: Regulatory filings expected Q4 2024

ENTR-601-50

Phase 2: Regulatory filings expected 2025

Exon 51

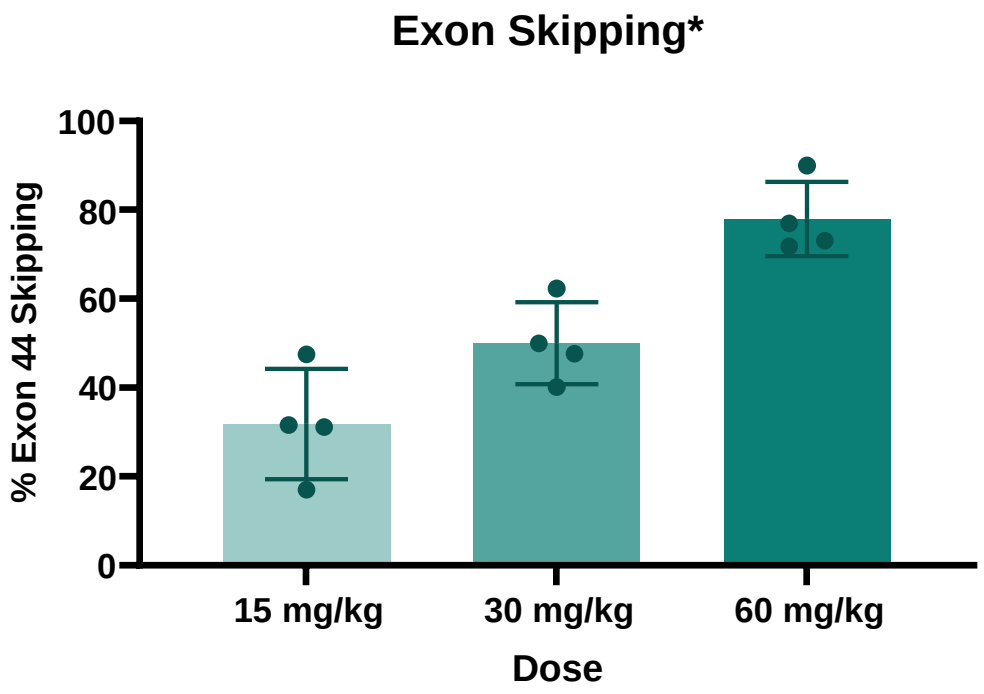
Candidate selection expected in 2024

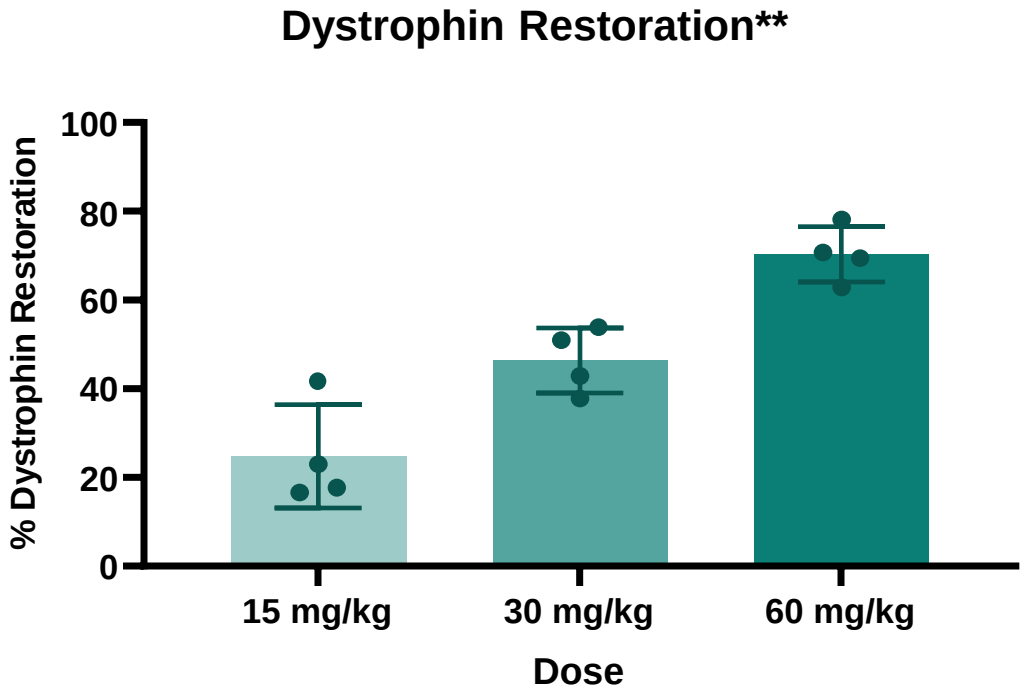
[1]Parent Project Muscular Dystrophy: About Duchenne.

Duchenne Franchise: Preclinical Data

Dose-Dependent Exon Skipping and Dystrophin
Strong Potential for Best-in-Class Clinical Profile

Dose-dependent response at a minimally effective dose of 15 mg/kg is observed, with near saturation at a clinically relevant dose of 60 mg/kg implying a wide therapeutic index





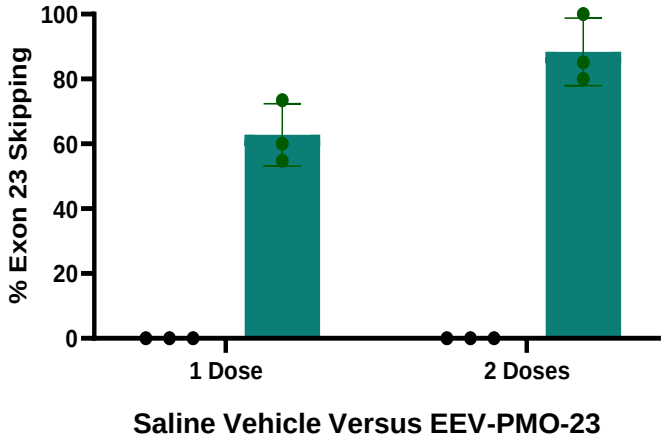
- Del45hDMD.*mdx* mice dosed with EEV-PMO-44
- n=4, gastrocnemius sample collection 2 weeks post injection

*ddPCR: double drop PCR; **JESS: automated western blot system; **PMO**, phosphorodiamidate morpholino oligomer.

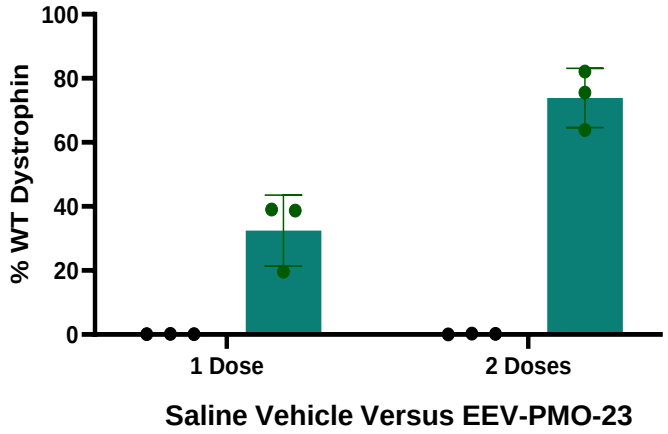
Accumulation of Exon Skipping and Dystrophin Restoration

Significant increase in and accumulation of exon 23 skipping and dystrophin expression following two doses of EEV-PMO-23 in D2-*mdx* mice, as measured six weeks after each dose

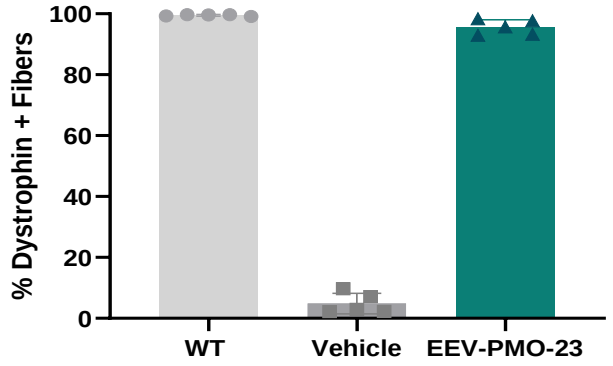
Exon Skipping



Dystrophin Production



Dystrophin Positive Fibers

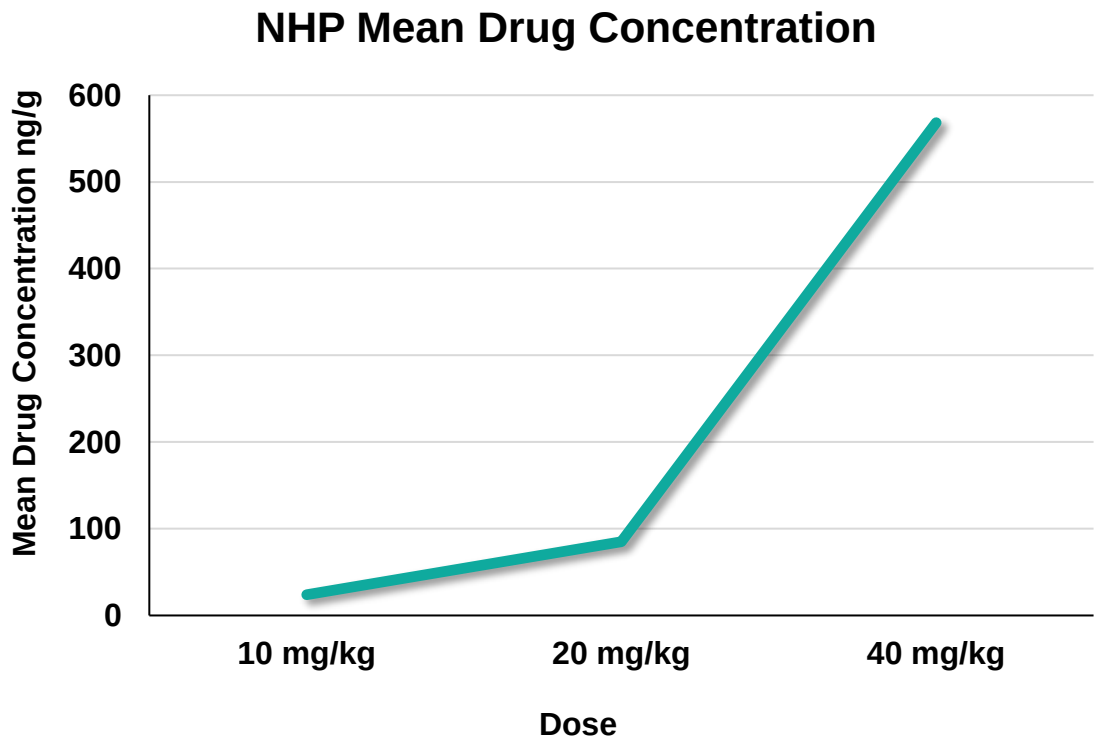


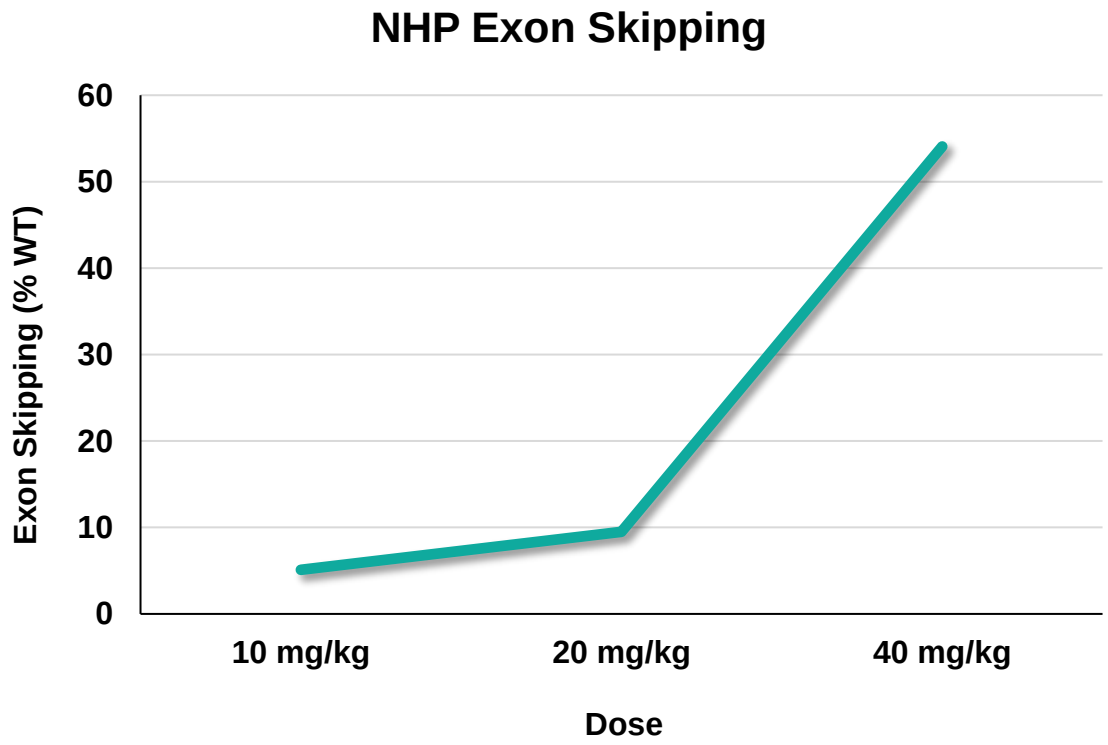
- D2-*mdx* mice (male, n=6) were treated with 2 doses of either vehicle or 80 mg/kg of EEV-PMO-23, 6 weeks apart and analyzed ~6 weeks after the last dose; Samples from gastrocnemius

Data presented at the 2024 Muscular Dystrophy Association (MDA) Clinical and Scientific Conference.

Dose-Dependent PK/PD in NHPs

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



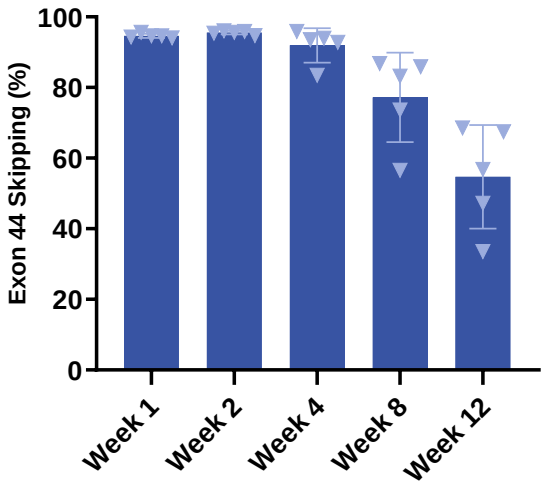


Single dose, bicep biopsy at 48 hours post-infusion; *R^2=0.9996; NHP: Non-human primates.

Consistent and Durable Efficacy Demonstrated Across Species

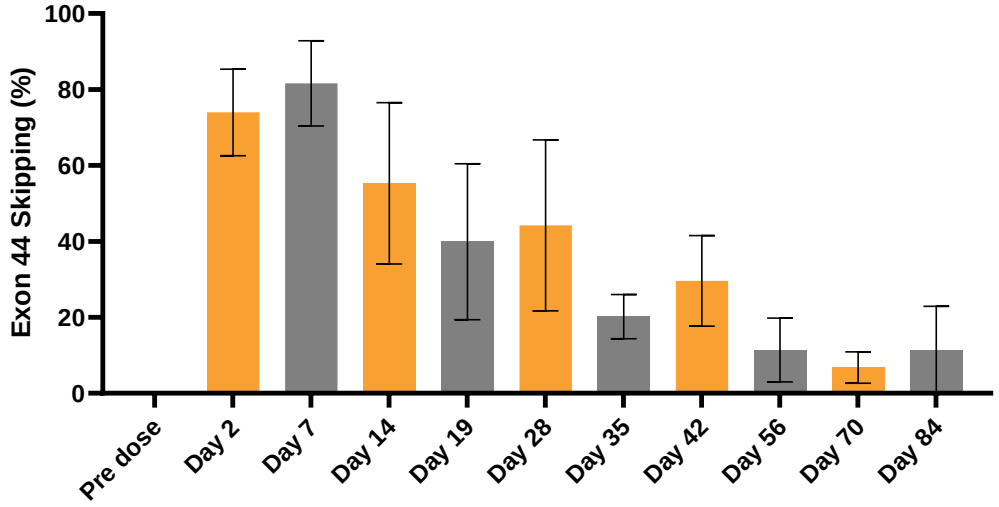
Significant potential patient benefit is supported by data in the mouse and the NHP at clinically relevant levels; *in vitro* data suggests much higher target engagement in patient cells

Exon 44 Skipping in hDMD Mouse



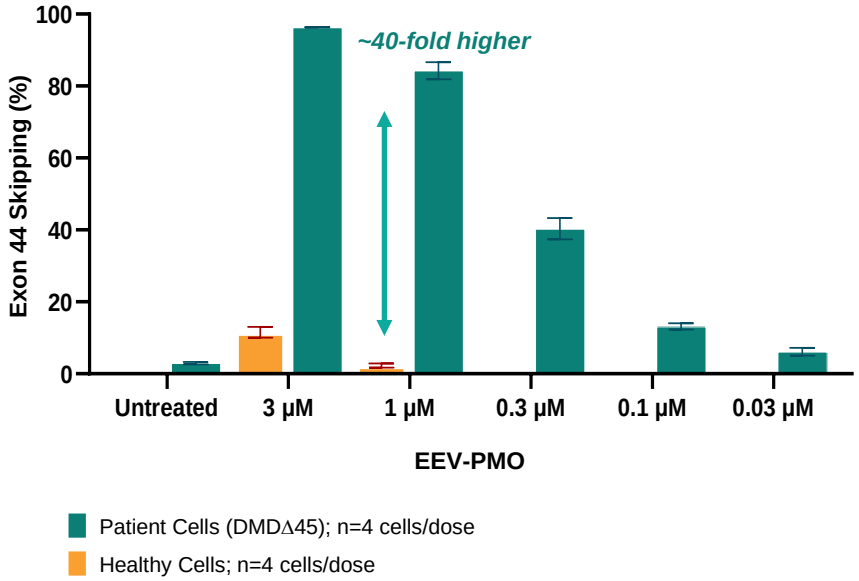
- Single 60 mg/kg dose
- Tibialis anterior

Exon 44 Skipping in NHP



- Post IV infusion of single 35 mg/kg dose, robust exon 44 skipping observed in biceps in the ENTR-601-44 treated NHPs (n=3 per cohort) for at least 12 weeks

Exon 44 Skipping in Healthy and Patient Myoblasts



Data presented at the 2022 MDA Clinical and Scientific Conference, TIDES 2022, and OPT 2024.

ENTR-601-44 Clinical Program

ENTR-601-44: Clinical Strategy

First-in-Human

Complete: Positive data support Phase 2 initiation

Single Ascending Dose (SAD) Study*
in Healthy Volunteers (ENTR-601-44-101)

- **32 adult subjects**
- **Placebo controlled**
- **6:2 randomization**
- **4 SAD cohorts**
- **Dosing 0.75, 1.5, 3 and 6 mg/kg**

Outcomes Measured

- ✓ Safety and tolerability
- ✓ Evaluation of PK and PD
- ✓ Target engagement as measured via exon skipping

Planned Multiple Ascending Dose/Phase 2b (Global)

Regulatory filings expected in Q4 2024

Multiple Ascending Dose (MAD) Study**
in Exon 44 Skipping Amenable Patients

- Juvenile patients
- 3 MAD cohorts (final design TBD)
- Dosing initiation target of 6 mg/kg
- Dosing interval ≥ every 6 weeks

Phase 2b Study**
in Exon 44 Skipping Amenable Patients

Target Product Profile:

- Double digit dystrophin improvement from baseline
- Dosing interval ≥ every 6 weeks

File for Accelerated Approval

Outcome Measures

- Safety and tolerability
- Evaluation of PK and PD
- Evaluation of exon skipping and dystrophin production (skeletal muscle)

Primary Efficacy Measures

- Change in dystrophin level (skeletal muscle)

Secondary/Exploratory Efficacy Measures

- Change from baseline in the 10-meter walk/run
- Change from baseline in the timed rise from floor
- Other parameters may include NSAA, FVC, QoL

October 2024

*Phase 1 data presented at World Muscle Society in Prague, Czechia, October 8-12, 2024; **MAD/Phase 2b study is subject to regulatory feedback. **ENTR-601-44** is a DMD exon 44 skipping EEV-oligonucleotide construct. **FVC**, forced vital capacity; **NSAA**, North Star Ambulatory Assessment: **PD**, pharmacodynamics; **Q,** quarter; **QoL**, quality of life; **TBD**, to be determined.

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 were deemed related to study drug by the investigator
- Most common AE was headache (n=7; 5 were mild and 2 were moderate)
  - All AEs resolved by study completion
  - No severe or serious AEs were reported in any dose group throughout the study
- No clinically significant findings were observed with laboratory values, electrocardiogram or vital signs
- No adverse findings or clinically relevant changes to any biomarkers of renal toxicity at the highest dose tested (6 mg/kg)

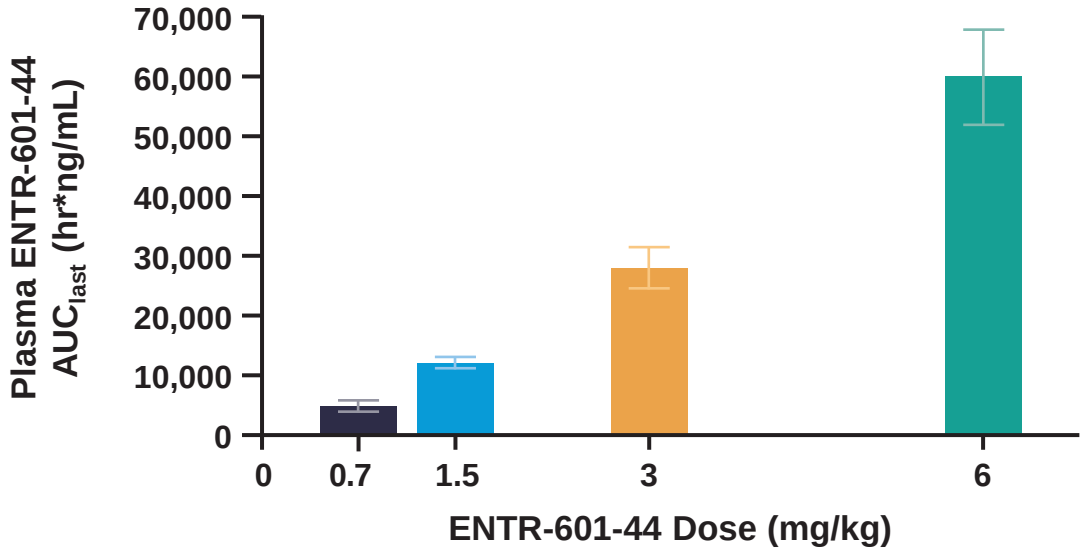
n (%)	Pooled placebo (N=8)	ENTR-601-44				
		0.75 mg/kg (n=6)	1.5 mg/kg (n=6)	3.0 mg/kg (n=7)	6.0 mg/kg (n=6)	Total (N=25)
Randomized	8 (100)	6 (100)	6 (100)	7 (100)	6 (100)	25 (100)
Dosed	8 (100)	6 (100)	6 (100)	6 (85.7)	6 (100)	24 (96)
Completed Study	8 (100)	6 (100)	6 (100)	6 (85.7)	6 (100)	24 (96)
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. Data presented at WMS 2024.

ENTR-601-44-101: Pharmacokinetics

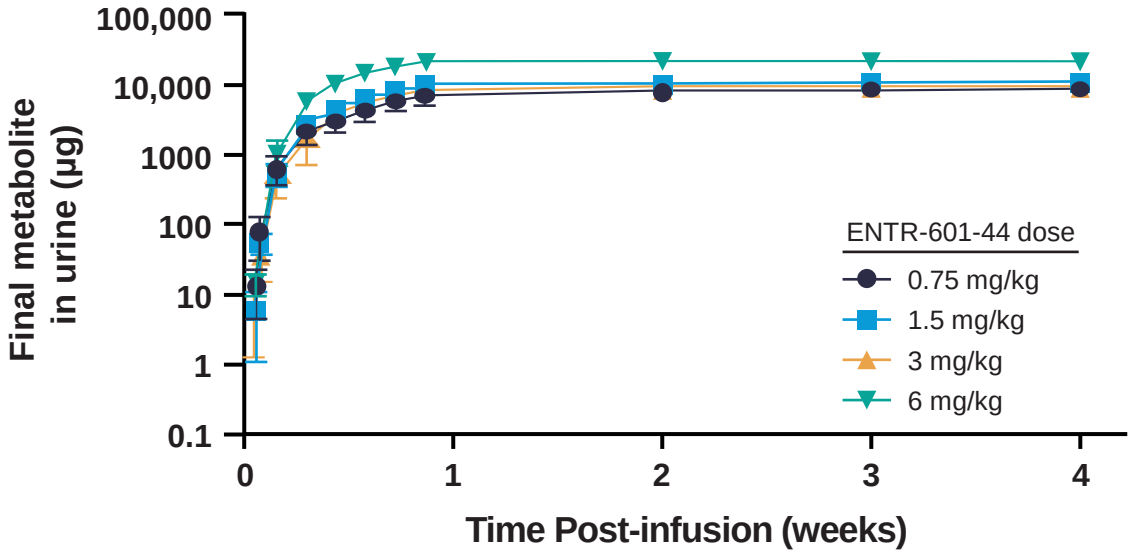
Remarkable dose-dependent pharmacokinetics, as measured by plasma AUC and urinary excretion, were observed in the trial, supporting the potential for efficacy at low doses in patients

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



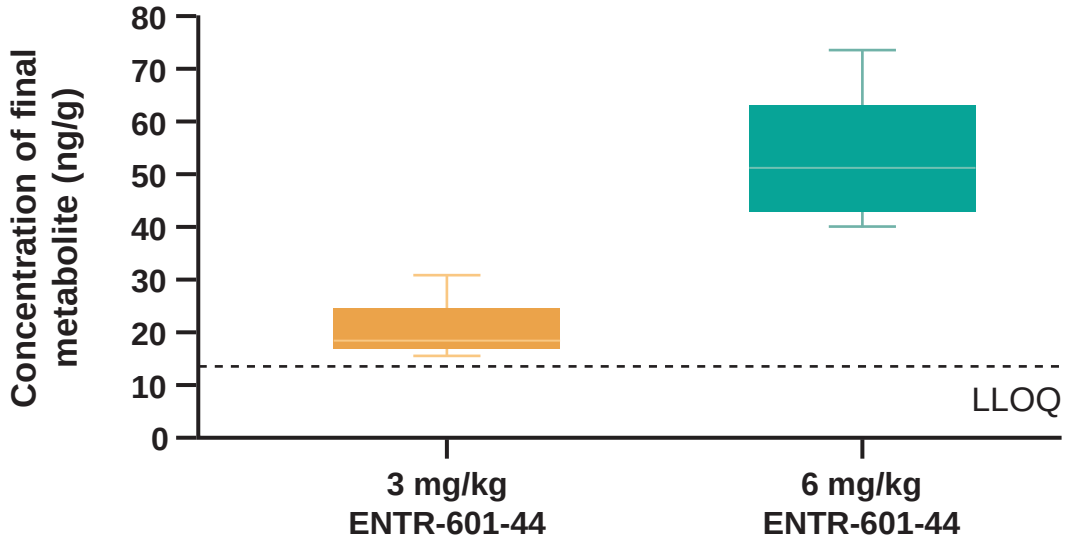
- Results suggest saturation of receptor-mediated re-uptake in human kidney on a dose-adjusted basis, contributing to lower dose-proportional renal exposure and lower possible renal toxicity in comparison with non-clinical models

(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. **AUClast**, area under the plasma concentration-time curve to the last measurable plasma concentration; **Cmax**, maximum concentration; **PK**, pharmacokinetics. Data presented at The 29th Annual Congress of the World Muscle Society (WMS 2024).

ENTR-601-44-101: Dose-Dependent Muscle Concentration and Exon Skipping

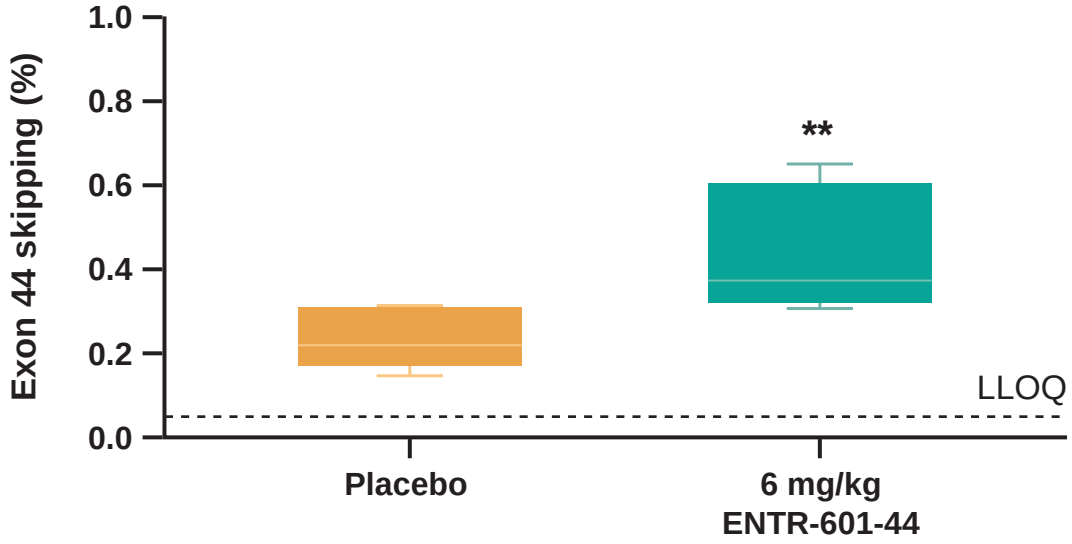
Clear muscle concentration dose response and separation from placebo at 6 mg/kg for exon skipping suggest the potential for a clinically relevant starting dose in the MAD/Phase 2

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. Data presented at WMS 2024.

ENTR-601-44: Clinical Strategy

First-in-Human

Complete: Positive data support Phase 2 initiation

Single Ascending Dose (SAD) Study*
in Healthy Volunteers (ENTR-601-44-101)

- 32 adult subjects
- Placebo controlled
- 6:2 randomization
- 4 SAD cohorts
- Dosing 0.75, 1.5, 3 and 6 mg/kg

Outcomes Measured

- ✓ Safety and tolerability
- ✓ Evaluation of PK and PD
- ✓ Target engagement as measured via exon skipping

Planned Multiple Ascending Dose/Phase 2b (Global)

Regulatory filings expected in Q4 2024

Multiple Ascending Dose (MAD) Study**
in Exon 44 Skipping Amenable Patients

- **Juvenile patients**
- **3 MAD cohorts (final design TBD)**
- **Dosing initiation target of 6 mg/kg**
- **Dosing interval ≥ every 6 weeks**

Phase 2b Study**
in Exon 44 Skipping Amenable Patients

Target Product Profile:

- **Double digit dystrophin improvement from baseline**
- **Dosing interval ≥ every 6 weeks**

File for Accelerated Approval

Phase 2b → **Open-label Extension**

Outcome Measures

- Safety and tolerability
- Evaluation of PK and PD
- Evaluation of exon skipping and dystrophin production (skeletal muscle)

Primary Efficacy Measures

- Change in dystrophin level (skeletal muscle)

Secondary/Exploratory Efficacy Measures

- Change from baseline in the 10-meter walk/run
- Change from baseline in the timed rise from floor
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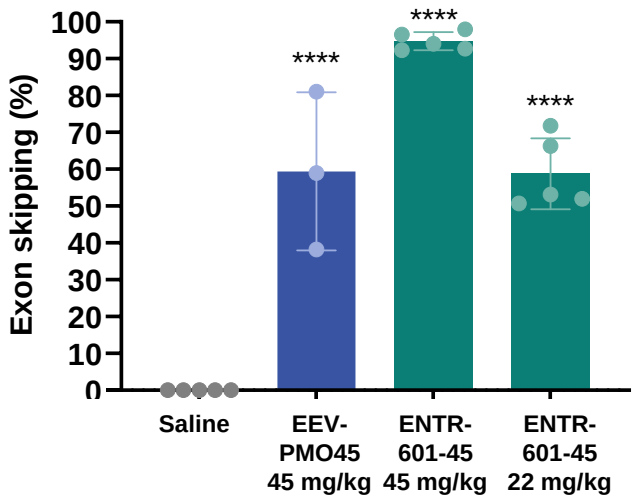
ENTR-601-45

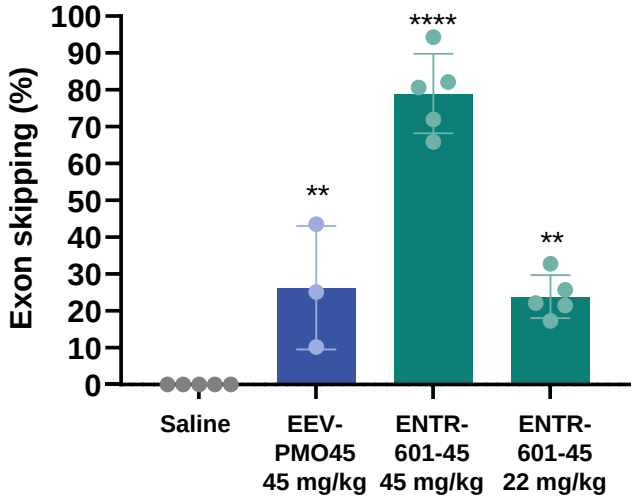
ENTR-601-45 Target Engagement in hDMD Mice

ENTR-601-45 delivered up to a two-fold improvement in exon skipping when compared to an equivalent dose of the same EEV conjugated to a casimersen sequence

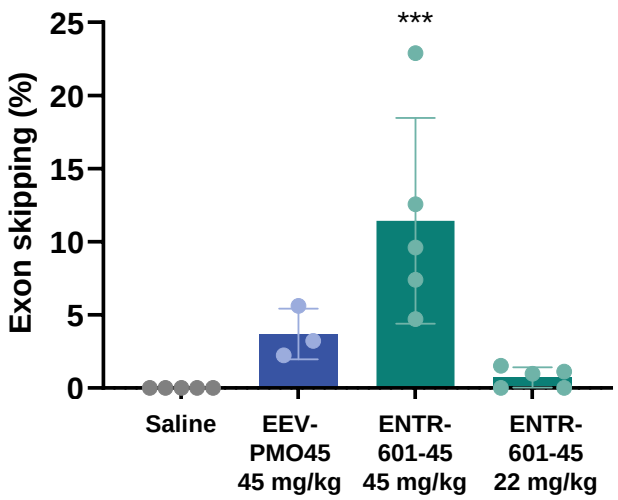
Tibialis Anterior



Diaphragm



Heart

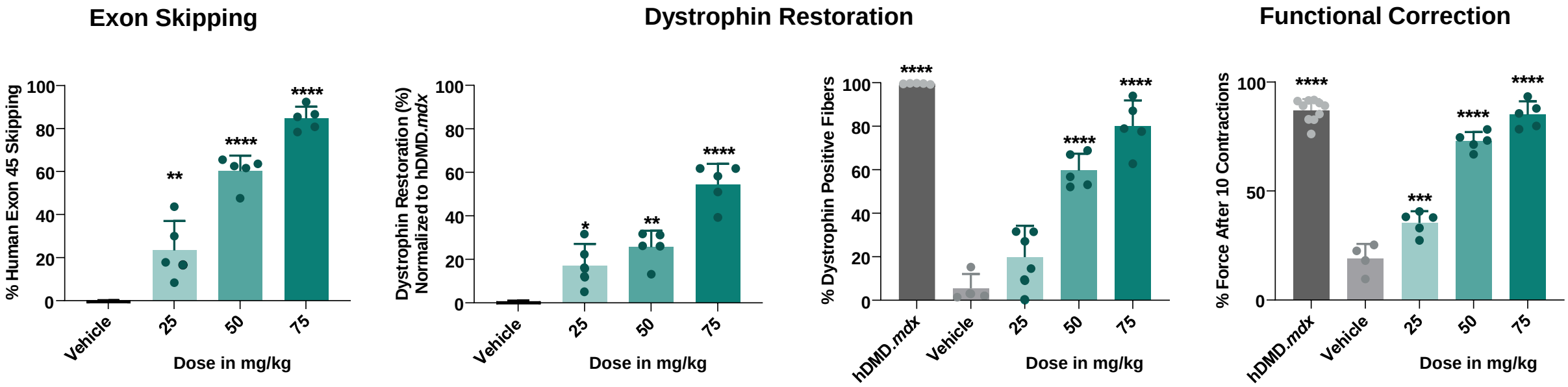


- A single IV dose of ENTR-601-45 resulted in high levels of exon skipping in hDMD in the mouse skeletal muscle and heart after 1 week
- Both EEV-PMO45 and ENTR-601-45 utilize the same EEV
- EEV-PMO45 uses the same oligonucleotide sequence as casimersen; ENTR-601-45 uses our proprietary PMO sequence

Data are shown as mean ± SD (n=3-5); One-way ANOVA; **p<0.01, ***p<0.001, ****p<0.0001; Relative to saline; Concentrations provided are PMO equivalent. Data presented at the 2023 MDA Clinical and Scientific Conference.

Dose-Dependent Functional Correction
Strong Potential for Best-In-Class Clinical Profile

Significant dose-dependent increase in exon 45 skipping and dystrophin expression following three doses in del44hDMD.*mdx* mice correlates to functional correction to wild type



- Active and vehicle n=5 del44hDMD.*mdx* mice per cohort, dosing EEV-PMO-45 Q6W 3X; Control standard n=10 saline treated hDMD.*mdx,* mice dosing Q6W 3X
- Skipping (ddPCR) and dystrophin production (JESS) is significantly increased 6 weeks the 3rd dose of ENTR-601-45 (gastrocnemius muscle shown)
- 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 are shown as mean ± SD *p ≤ 0.05 **p ≤ 0.01 ***p ≤ 0.001 ****p ≤ 0.0001; JESS: automated western blot system.

ENTR-601-45 Data Summary

ENTR-601-45 consistently demonstrated robust *in vitro* and *in vivo* data;
Regulatory submissions planned in Q4 2024

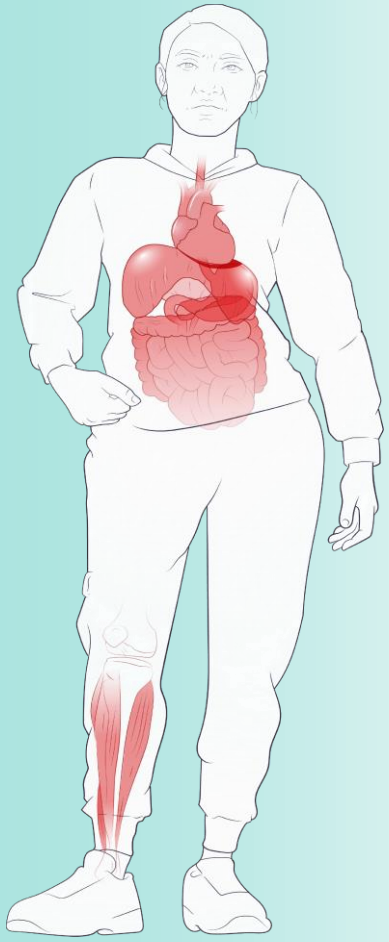
- **Patient-derived cells**
  - ENTR-601-45 showed robust exon skipping and dystrophin protein production in patient-derived cardiac and skeletal muscle cells
- **DMD mouse models**
  - High levels of exon skipping were measured in hDMD mouse heart and skeletal muscle tissue
  - Significant dose-dependent increase in exon 45 skipping and dystrophin expression following 3 doses in del44hDMD.*mdx* mice correlates to functional correction to wild type
- **Process development**
  - GMP drug substance production complete

Next Steps

- Planning for a global MAD trial in Duchenne patients
- Regulatory submissions expected in Q4 2024

Myotonic Dystrophy Type 1 (DM1)

DM1 is a Debilitating, Multisystemic Disease with No Available Treatments



~110,000

people in the **US and Europe** are living with DM1

Symptoms include:

- Myotonia (or delayed relaxation of skeletal muscles)
- Fatigue and excessive sleepiness
- Cardiac conduction irregularities
- Respiratory muscle impairment
- Gastrointestinal complications
- Incontinence
- Generalized limb weakness

EEV-Oligonucleotide Approach

VX-670 targets the underlying cause of DM1 and has the potential to restore normal cell function via a highly-specific CUG-repeat steric blocking approach

Transformational Collaboration with Vertex

Entered into a partnership for the discovery and development of EEV-therapeutics for DM1 in 2023

The four-year global research collaboration includes $224M upfront payment and $26M equity investment, up to $485M for the achievement of certain milestones, plus royalties

Program Highlights

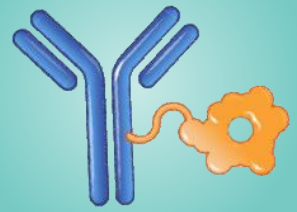
- VX-670 engages the CUG repeat RNA and liberates bound splicing factors. Through this mechanism, VX-670 aims to correct mis-splicing and is being investigated to address the underlying cause of disease
- Global Phase 1/2 clinical trial for VX-670 in people with DM1 is ongoing and Vertex expects to complete the SAD portion of the study by the end of 2024
- Upon completion of the SAD, Vertex will move into the MAD portion of the trial, where both the safety and efficacy of VX-670 will be evaluated

Pipeline Expansion

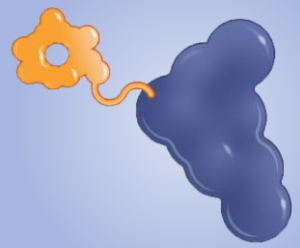
A Broadly Applicable Approach

Entrada has demonstrated intracellular uptake of unique moieties ranging from 1 kDa to 600 kDa

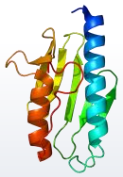
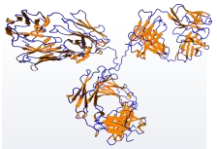
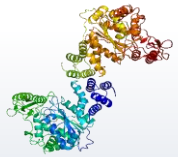
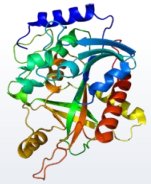
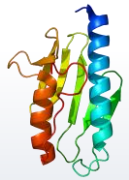
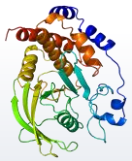
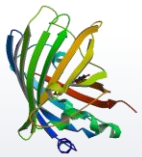
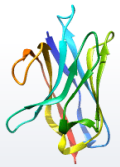
Antibodies



Enzymes



Oligonucleotides

										
550-600 kDa	**150 kDa**	**98 kDa**	**96 kDa**	**86 kDa**	**46 kDa**	**37 kDa**	**32 kDa**	**16 kDa**	**6 kDa**	**1-3 kDa**
Hybrid frataxin	Antibody	Thymidine phosphorylase	Purine nucleoside phosphorylase	Alanine-glyoxylate aminotransferase	Human frataxin	PTP1B catalytic domain	EGFP	Nanobody	Oligonucleotide	Various peptide cargos

Multiple Pipeline Expansion Opportunities

Entrada is extending its efforts to develop novel intracellular therapeutic candidates by leveraging new moieties and targeting additional therapeutic areas

	DNA	RNA			PROTEINS		
TARGET							
APPROACH	**Gene** Editing	**RNA** Editing	**RNA** Splicing	**RNA** Blocking	**RNA** Silencing	**Protein** Replacement	**Protein** Inhibition	**Protein** Degradation
GOAL	Deliver CRISPR enzyme and repair gene function with guide RNA	Deliver oligonucleotide therapeutics for RNA editing	Modify RNA via exon/intron splicing to activate protein expression	Block trinucleotide repeats in RNA to inhibit adverse binding	Silence or knockdown RNA to prevent protein expression	Replace proteins and enzymes	Inhibit protein signaling pathways	Degrade disease-causing proteins

Corporate Highlights

Entrada is positioned for execution, growth and diversification

The Boston Globe
TOP PLACES TO WORK 2023
DIVERSITY, EQUITY, AND INCLUSION CHAMPION

2023 BEST PLACES to WORK
PRESENTED BY BioSpace

We are a proud Massachusetts company!
WINNER
MassEcon Impact Awards

*Based on current operating plans and $469.7M in cash, cash equivalents and marketable securities as of June 30, 2024.

entrada THERAPEUTICS

Entrada is well capitalized to deliver ENTR-601-44 and ENTR-601-45 through early interim patient data and progress the broader pipeline

- **Strong Financial Position (June 30, 2024)**
  - Cash runway: Into 2027*
  - Cash, cash equivalents and marketable securities: ~$470M
  - Common shares outstanding: 37.2M
- **Award-Winning Team and Culture**
  - ~170 employees: 75% have advanced degrees and 50% have PhDs
  - Seasoned leadership team across functions
  - Top Place to Work: *The Boston Globe*, *BioSpace* and MassEcon
- **Deep Patent Portfolio**
  - 70 patent families on file, including exclusive EEV platform rights
  - 14 families with one or more granted patents

Leadership Team and Board of Directors

Dipal Doshi
Chief Executive Officer

Natarajan Sethuraman, PhD
President of R&D

Nathan Dowden
President and Chief Operating Officer

Kory Wentworth, CPA
Chief Financial Officer

Kerry Robert
Senior Vice President, People

Jared Cohen, PhD, JD
General Counsel

Karla MacDonald
Chief Corporate Affairs Officer

Kevin Healy, PhD
Senior Vice President, Regulatory

Board of Directors

Kush Parmar, MD, PhD
Managing Partner
5AM Ventures
(Board Chairman)

Peter S. Kim, PhD
Virginia and D.K. Ludwig Prof. of Biochemistry
Stanford University

Gina Chapman
President and Chief Executive Officer
CARGO Therapeutics

Mary Thistle
Industry Leader and Independent
Board Member

Bernie Zeiher, MD
Industry Leader and Independent
Board Member

Dipal Doshi
Chief Executive Officer

Corporate Summary

Entrada is leveraging its Endosomal Escape Vehicle platform (EEV™) to create a diverse and expanding development portfolio of RNA-, antibody- and enzyme-based therapeutics

- **Advancing new therapeutic options for people living with Duchenne muscular dystrophy (DMD)**
  - ENTR-601-44: Regulatory filings expected in Q4 2024 for global Phase 2 clinical trial in patients
    - Positive Phase 1 study shows dose-dependent response, significant plasma concentration, muscle concentration and exon skipping with no serious adverse events and no clinically significant changes in laboratory assessments
    - Data demonstrates the translation of ENTR-601-44's nonclinical studies to healthy volunteers
  - ENTR-601-45: Regulatory filings expected in Q4 2024 for global Phase 2 clinical trial in patients
  - ENTR-601-50: Regulatory filings expected in 2025 for global Phase 2 clinical trial in patients
- **Transformative Vertex partnership for the development of myotonic dystrophy type 1 (DM1)**
  - VX-670: Global Phase 1/2 clinical trial ongoing; Completion of SAD portion of the study expected by end of 2024
- **Expanding pipeline by leveraging new moieties and extending into new therapeutic areas**
- **Strong financial position with cash runway into 2027***

Note: All references in this presentation regarding planned regulatory filings and clinical trial designs are subject to ongoing discussion with US and international regulatory authorities; SAD: Single ascending dose; *Based on current operating plans and $469.7M in cash, cash equivalents and marketable securities as of June 30, 2024.


Learn more at
EntradaTx.com
entrada
THERAPEUTICS