Corporate Presentation

March 2024

entrada THERAPEUTICS

Disclaimer

This presentation includes express and implied “forward-looking statements.” Forward looking statements include all statements that are not historical facts, and in some cases, can be identified by terms such as “may,” “might,” “will,” “could,” “would,” “should,” “expect,” “intend,” “plan,” “objective,” “anticipate,” “believe,” “estimate,” “predict,” “potential,” “continue,” “ongoing,” or the negative of these terms, or other comparable terminology intended to identify statements about the future. Forward-looking statements contained in this presentation include, but are not limited to, statements about our product development activities and clinical trials, our regulatory filings and approvals, statements related to our ability to continue to recruit for and complete its healthy volunteer trial, ENTR-601-44-101, in the United Kingdom, expectations regarding the timing of data from our Phase 1 trial for ENTR-601-44 in the second half of 2024, the ability to resolve the clinical hold for ENTR-601-44 and subsequent activities, expectations regarding the timing or content of any update regarding our regulatory filings, expectations regarding the safety and therapeutic benefits of ENTR-601-44, our ability to develop and advance our current and future product candidates and discovery programs, expectations regarding the results of preclinical studies predicting the results of later preclinical studies or any clinical trials of our therapeutic candidates, our ability to establish and maintain collaborations or strategic relationships, our ability to raise additional funding, the rate and degree of market acceptance and clinical utility of our product candidates, the potential of our EEV product candidates and EEV platform, the ability and willingness of our third-party collaborators to continue research and development activities relating to our product candidates, including our Vertex partnership for ENTR-701, expectations regarding the expected timing, progress and success of our collaboration with Vertex, including any future payments we may receive under our collaboration and license agreements, our collaborators’ ability to protect our intellectual property for our products, expectations regarding the timing of preclinical data results and planned CTA/IND submissions for ENTR-601-45 and ENTR-601-50, the continued development and advancement of ENTR-601-44, ENTR-601-45 and ENTR-601-50 for the treatment of DMD, and ENTR-701 for the treatment of DM1, and the sufficiency of our cash resources through the second quarter of 2026. By their nature, these statements are subject to numerous risks and uncertainties, including factors beyond our control, that could cause actual results, performance or achievement to differ materially and adversely from those anticipated or implied in the statements. You should not rely upon forward-looking statements as predictions of future events. Although our management believes that the expectations reflected in our statements are reasonable, we cannot guarantee that the future results, performance or events and circumstances described in the forward-looking statements will be achieved or occur. Recipients are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date such statements are made and should not be construed as statements of fact.

Certain information contained in this presentation and statements made orally during this presentation relate to or are based on studies, publications, surveys and other data obtained from third-party sources and our own internal estimates and research. While we believe these third-party studies, publications, surveys and other data to be reliable as of the date of this presentation, it has not independently verified, and makes no representations as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, no independent source has evaluated the reasonableness or accuracy of our internal estimates or research and no reliance should be made on any information or statements made in this presentation relating to or based on such internal estimates and research.

Our Mission

To Treat Devastating Diseases with Intracellular Therapeutics

entrada THERAPEUTICS

An Expanding Pipeline of Intracellular Therapeutics

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 completed dosing of a third cohort in its Phase 1 trial with data expected in H2 2024; Regulatory applications expected in Q4 2024 for global Phase 2 clinical trial
  - ENTR-601-45 regulatory applications expected in Q4 2024 for global Phase 2 clinical trial
  - ENTR-601-50 regulatory applications expected in 2025 for global Phase 2 clinical trial
- **Transformative Vertex partnership for the development of myotonic dystrophy type 1 (DM1)**
  - VX-670 (ENTR-701) Phase 1/2 clinical trial initiated
  - $224M upfront payment and $26M equity investment; Up to $485M for the achievement of certain milestones, plus royalties; Four-year global research collaboration
- **Extending the pipeline with novel intracellular therapeutic candidates by leveraging new moieties and targeting additional therapeutic areas**
- **Strong financial position with cash runway through the second quarter of 2026***

*Assumes $352 million cash, cash equivalents and marketable securities as of December 31, 2023, together with Vertex collaboration ongoing research support and achievement of certain milestones.

A Differentiated and Expanding Pipeline

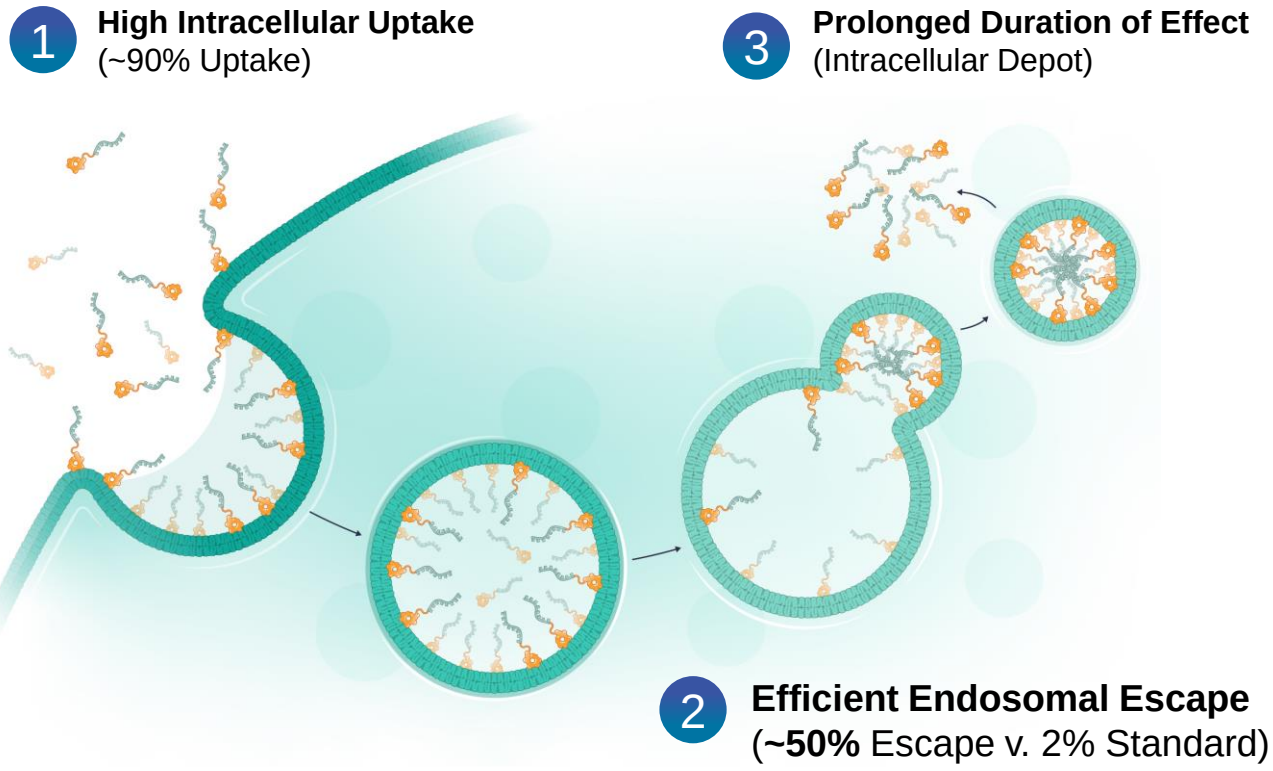
Entrada’s pipeline includes a diverse array of high potential and high value assets;
Each disease has a substantial patient population with a significant unmet medical need

Disease/Condition		Discovery	Lead Optimization	IND Enabling	Clinical
Neuromuscular	DMD	ENTR-601-44			Phase 1 data expected in H2 2024 and Phase 2 regulatory filings expected in Q4 2024
		ENTR-601-45			Phase 2 regulatory filings expected in Q4 2024
		ENTR-601-50			Phase 2 regulatory filings expected in 2025
		Exon 51			
	DM1	VX-670 (formerly ENTR-701)			VERTEX Phase 1/2 trial initiated
	POMPE	ERT/Oligonucleotide			
Beyond Neuromuscular		Immunology			
		Ocular			
Platform Expansion		Gene editing			
		mRNA delivery			
		Antibody/Protein			

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 seeks to solve 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.

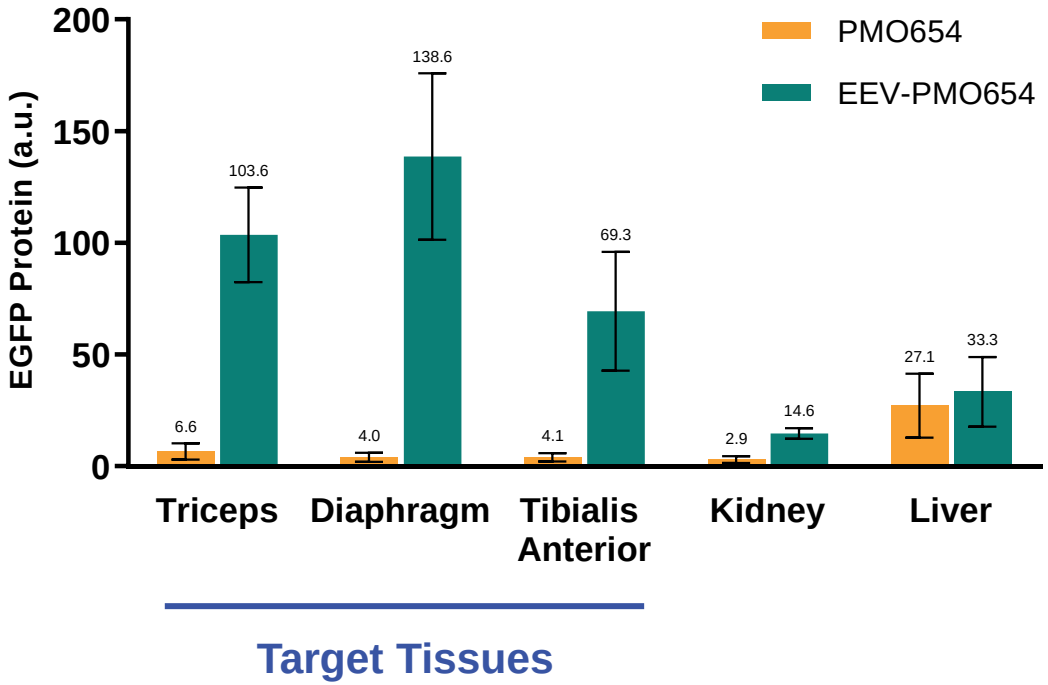
Functional Delivery for Target Tissues

EEV-therapeutic candidates can be designed to enhance functional delivery to target tissues

Discovery Engine for Intracellular Therapeutics

- High-throughput **EEV library screening** *in vitro*
- Functional validation of lead EEVs with **PMO therapeutic modality** *in vitro* and *in vivo*
- **EEV** optimized for the functional delivery to target tissues *in vivo*

Functional Delivery in the EGFP-654 Transgenic Mice



PMO, phosphorodiamidate morpholino oligomer; EGFP-654 transgenic mouse model contains an EGFP gene interrupted by human beta-globin intron 2 with mutated nt654 (Sazani, P. et al. *Nature Biotech.* 2002); PMO654, splicing switching PMO targeting nt654; shown as mean ± standard deviation.

Translation from Update to Outcomes
Murine Example

EEV-therapeutic candidates have demonstrated favorable pharmacological properties: efficient intracellular delivery, significant uptake in target tissues and potent pharmacodynamic outcomes

Tissue Uptake in Muscle

- ✓ Skeletal muscle
- ✓ Cardiac muscle

+

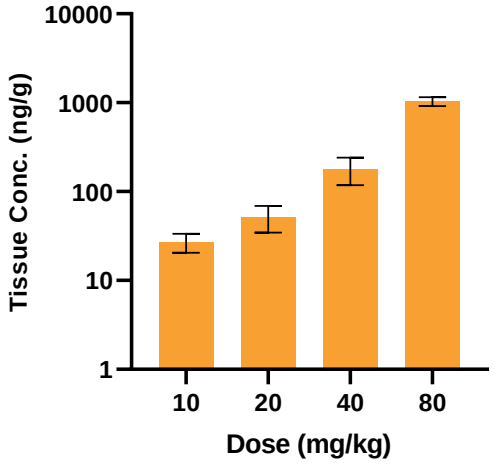
Intracellular Delivery

- ✓ Endosomal escape
- ✓ Nuclear localization

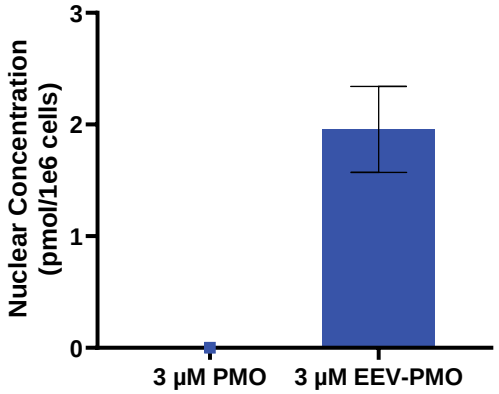
=

Pharmacodynamic Outcome

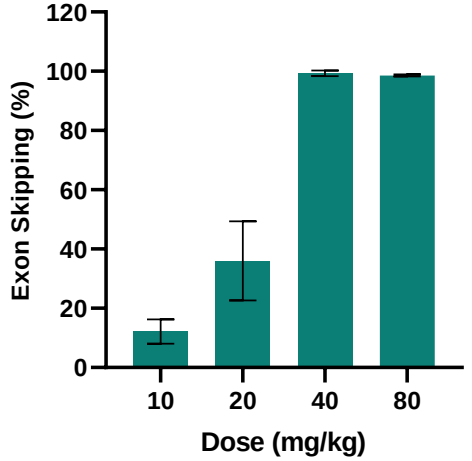
- ✓ Rapid, dose-dependent response
- ✓ Duration of at least 12 weeks



IV, hDMD mice, 5-day post injection



24-hour incubation

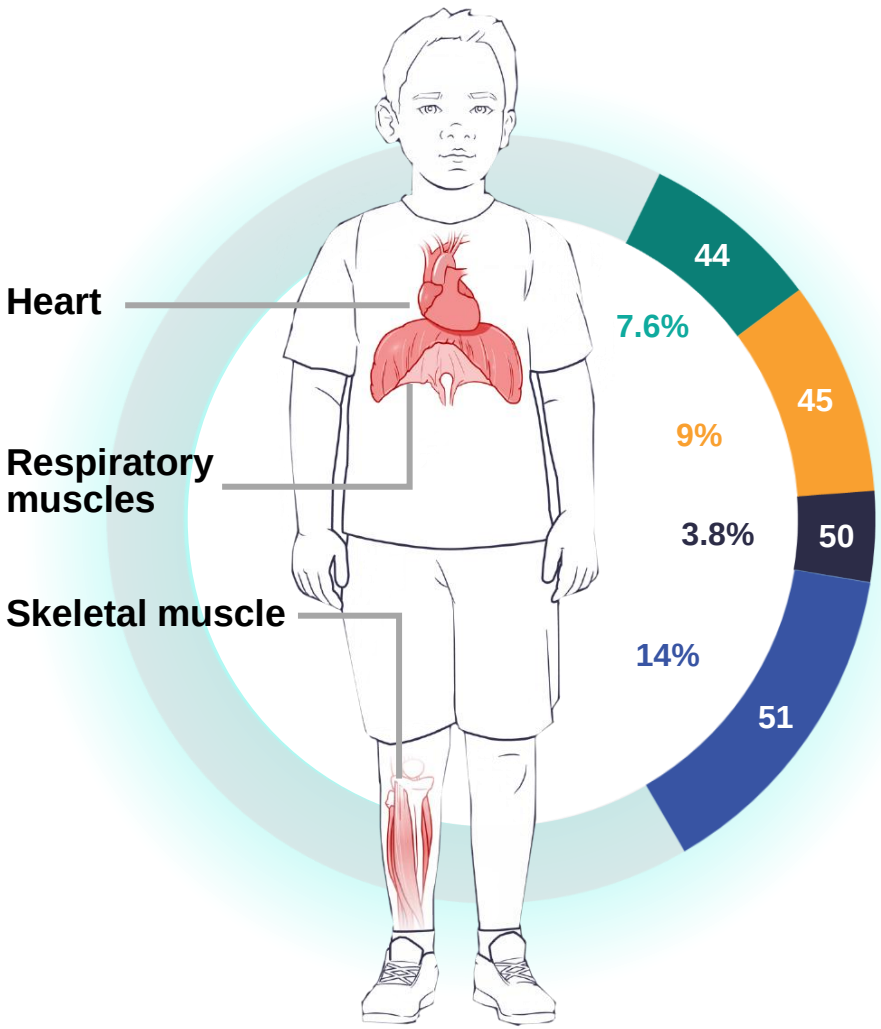


IV, hDMD mice, 5-day post injection

hDMD mice express full-length human dystrophin gene.

Duchenne Muscular Dystrophy (DMD)

DMD: 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

~40,000

people in the **U.S. and Europe** have Duchenne[1]

Duchenne Franchise

ENTR-601-44 Phase 1

Phase 1 data expected H2 2024
Phase 2 regulatory filings expected Q4 2024

ENTR-601-45 IND Enabling

Phase 2 regulatory filings expected Q4 2024

ENTR-601-50 IND Enabling

Phase 2 regulatory filings expected 2025

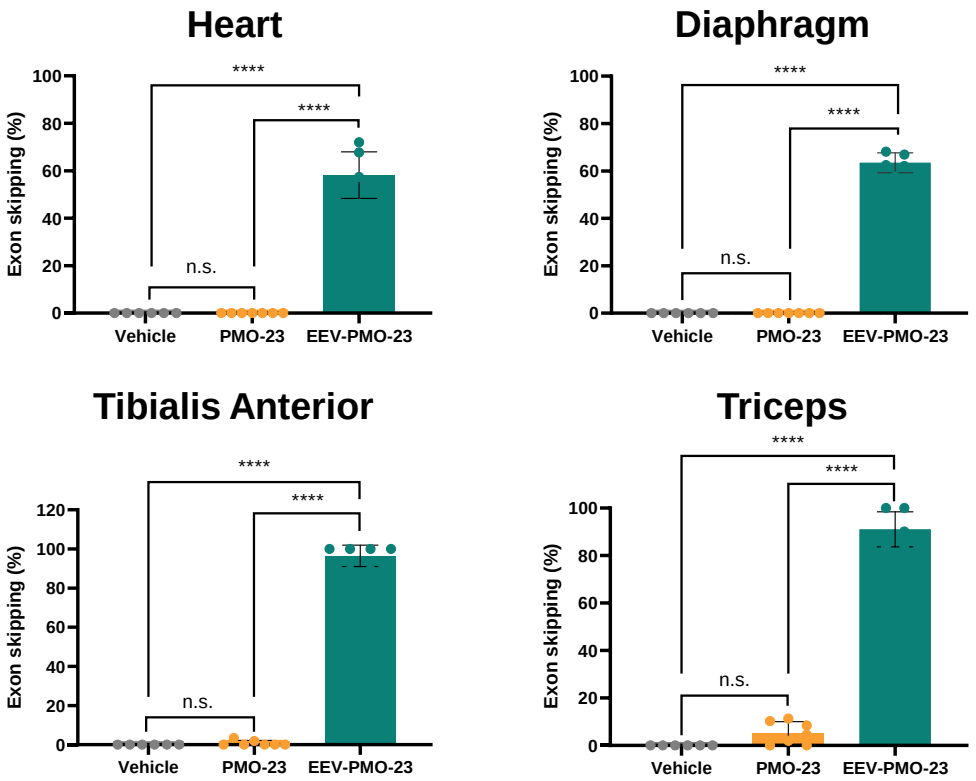
Exon 51 Lead Optimization

Candidate selection expected in 2024

[1]Parent Project Muscular Dystrophy: About Duchenne. [2]Europeans Medicines Agency: Orphan designation for the treatment of Duchenne muscular dystrophy. [3]Bladen, C.L. et al *HUMAN MUTATION*, 2015.

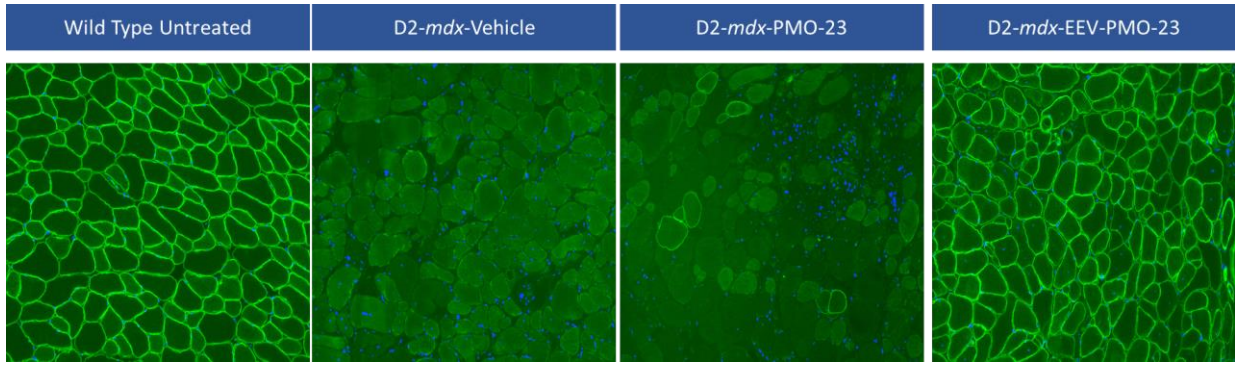
Repeat EEV-PMO Treatment Restores Muscle Integrity in D2-*mdx* Mice

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

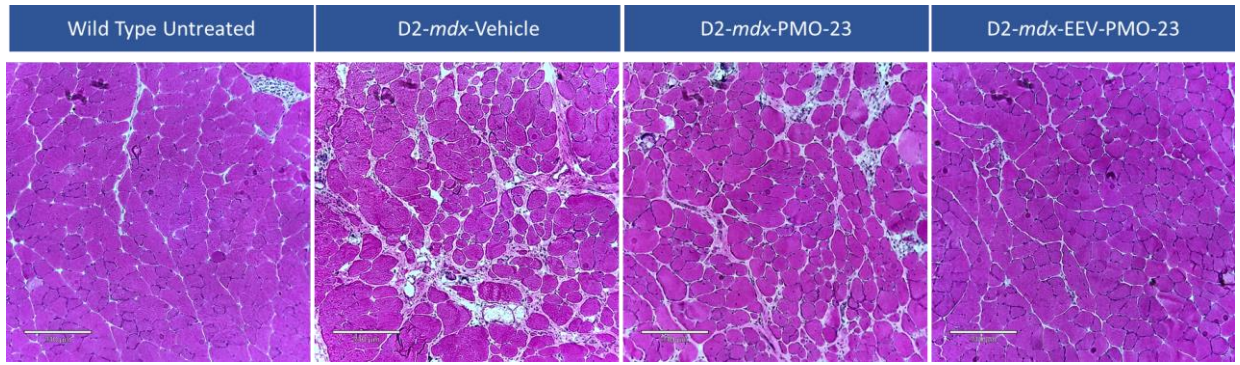


Broad dystrophin expression and restoration of muscle integrity after four monthly IV doses of EEV-PMO-23 in D2-*mdx* mice

Representative Immunofluorescence of Gastrocnemius Muscle (Dystrophin Staining in Green)



Representative Histopathology of Gastrocnemius Muscle (H&E Staining)



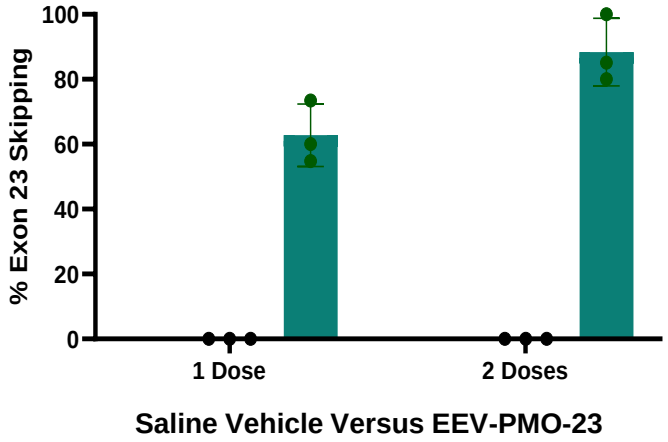
- D2-*mdx* mice (male, n=6-7) were treated with 4 monthly doses of either vehicle, 20 mg/kg PMO-23 or 20 mg/kg PMO-23 equivalent of EEV-PMO-23, and the data were collected ~4 weeks after the last dose

EEV, Endosomal Escape Vehicle; PMO-23, mouse *Dmd* exon 23 skipping phosphorodiamidate morpholino oligomer; D2-*mdx* is a DMD mouse model with a nonsense mutation in DMD exon 23 (Coley et al. *Hum. Mol. Genet.* 2016); ****p<0.0001; n.s., not significant; shown as mean ± standard deviation.

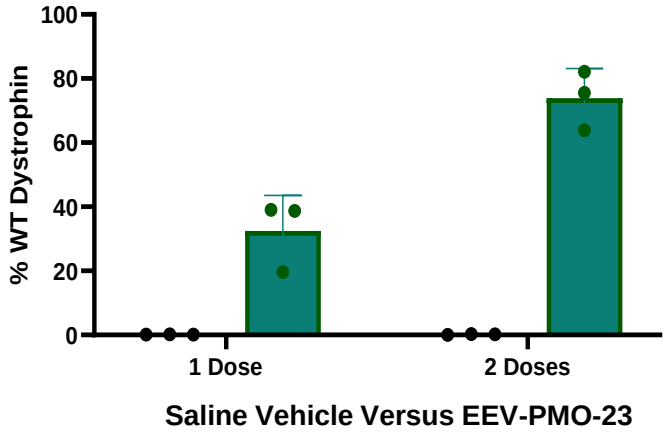
Exon 23 Skipping and Protein Restoration in D2-*mdx* Mice

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

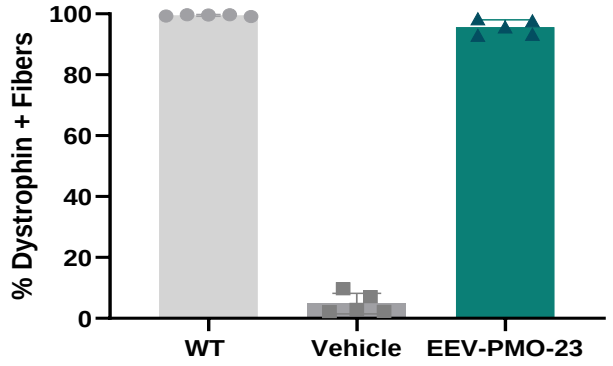
Exon Skipping



Dystrophin Production



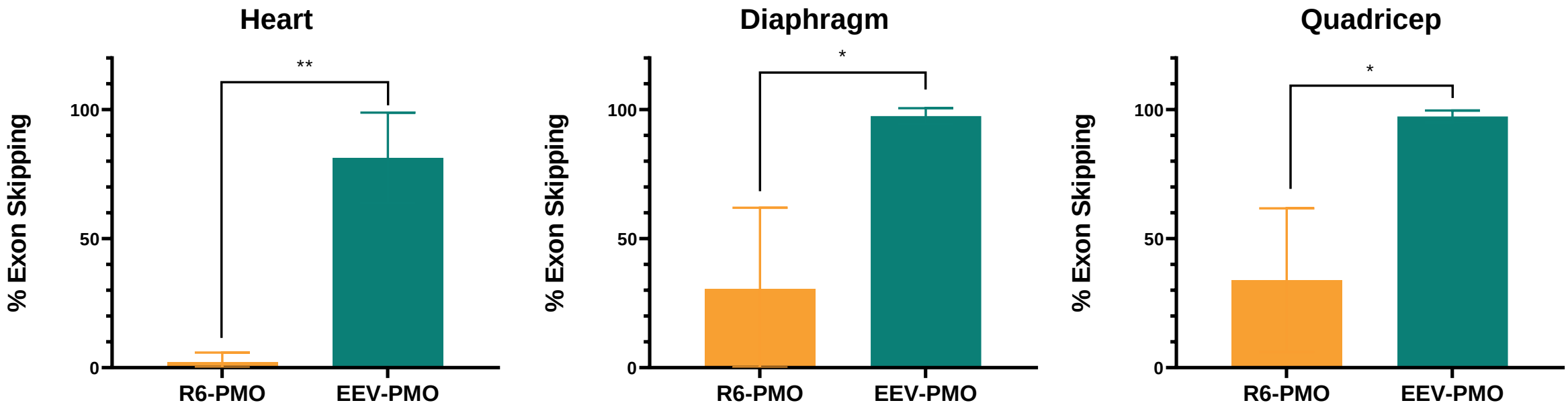
Dystrophin Positive Fibers



- 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

Comparison to Alternative R6-PMO

EEV-PMO significantly improved exon 23 skipping after 3 days in *mdx* mice as compared to competitive R6-PMO



*p<0.05, **p<0.01

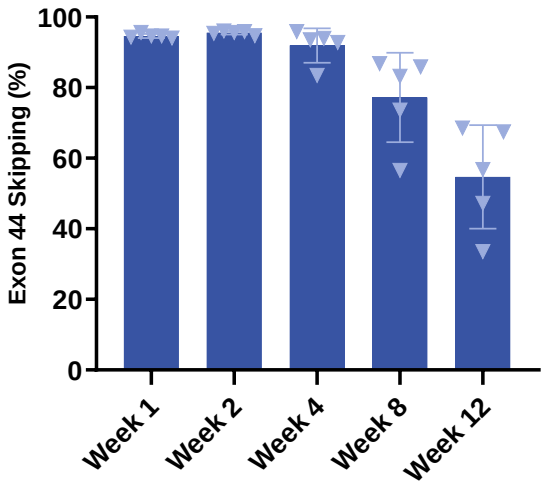
- EEV-PMO-23 demonstrates significantly improved PD effects after single 40 mg/kg IV dose in mdx mice
- Based on published patents, R6 sequence believed to be the same linear construct used for a current exon 51 skipping program

ENTR-601-44

Consistent and Durable Efficacy Demonstrated Across Species

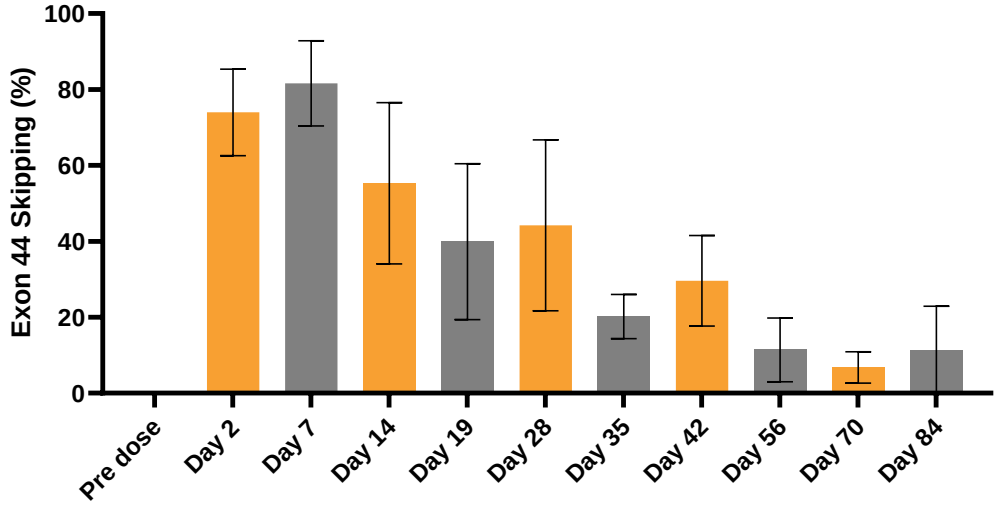
Significant patient benefit is implied by data in the mouse and the monkey at clinically relevant levels; *in vitro* data suggests much higher target engagement in patient cells

Exon 44 Skipping in hDMD Mouse



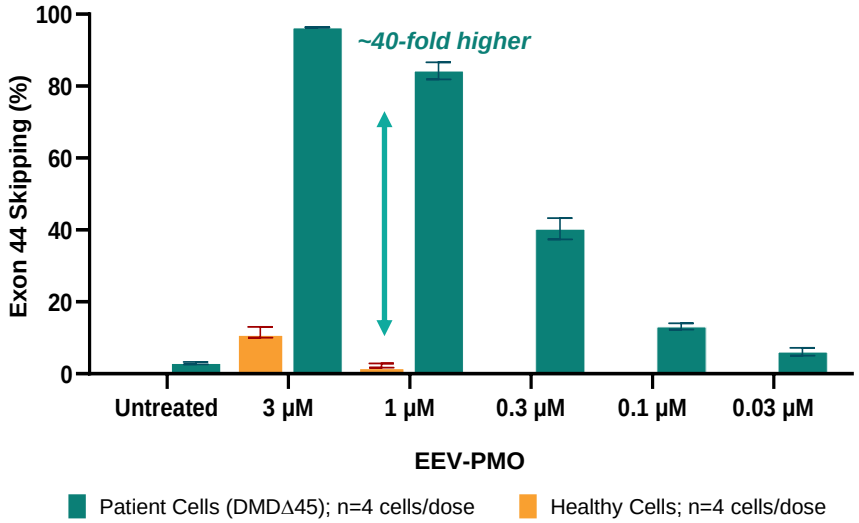
- Single 60 mg/kg dose
- Tibialis Anterior

Exon 44 Skipping in Monkey



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

Exon 44 Skipping in Healthy and Patient Myoblasts



ENTR-601-44 Data Summary

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

- ✓ High levels of exon skipping across *mdx*, D2-*mdx,* human dystrophin mouse and NHP studies
- ✓ Exon skipping translates to promising dystrophin production in heart and skeletal muscles
- ✓ Dystrophin production observed results in functional improvement in D2-*mdx* mouse
- ✓ Extended circulating half-life and durable exon skipping over 12+ weeks from a single injection of ENTR-601-44 was observed in the NHP

ENTR-601-44-101:
Phase 1 clinical trial ongoing

- First participant dosed in September 2023
- Completed dosing of cohorts 1, 2 and 3
- Data anticipated in H2 2024
- Phase 1 clinical data will support a global clinical trial in patients*

*MAD/Phase 2b study is subject to regulatory feedback and the outcome of the SAD study.

ENTR-601-44 Clinical Strategy

First-in-Human Trial

Data expected H2 2024

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

- **First subject dosed in Q3 2023**
- **Completed dosing for cohort 1, 2 and 3**
- **~40 subjects**

Outcome Measures

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

Multiple Ascending Dose/Phase 2b (Global)

Regulatory filings expected in Q4 2024

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

Phase 2b Study*
in Exon 44 Skipping Amenable Patients

Target Product Profile:

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

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

- NSAA (North Star Ambulatory Assessment), timed function tests, and other measures of function (e.g., PUL 2.0; wearable device)
- Other parameters may include cardiac MRI, FVC, QoL

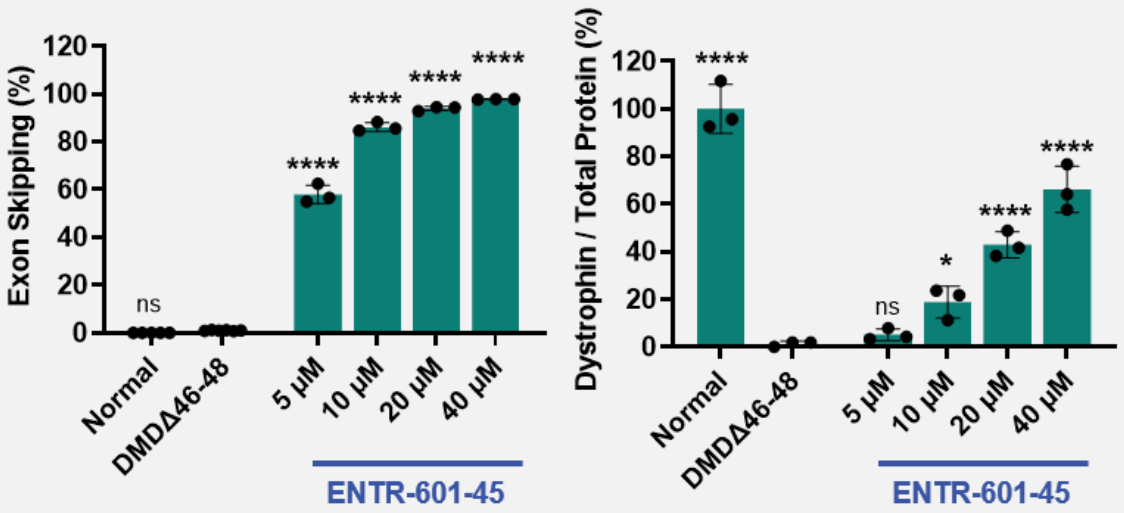
*MAD/Phase 2b study is subject to regulatory feedback and the outcome of the SAD study.

ENTR-601-45

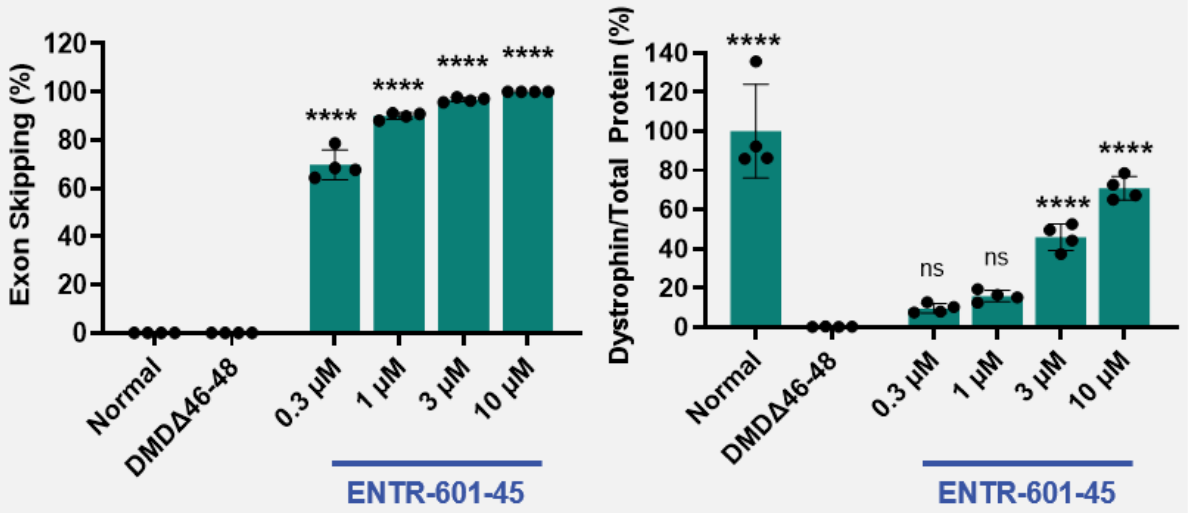
ENTR-601-45 *in vitro* Efficacy

ENTR-601-45 showed robust exon skipping and dystrophin production in patient-derived skeletal and cardiac muscle cells

ENTR-601-45 in Skeletal Muscle Cells



ENTR-601-45 in Cardiac Muscle Cells

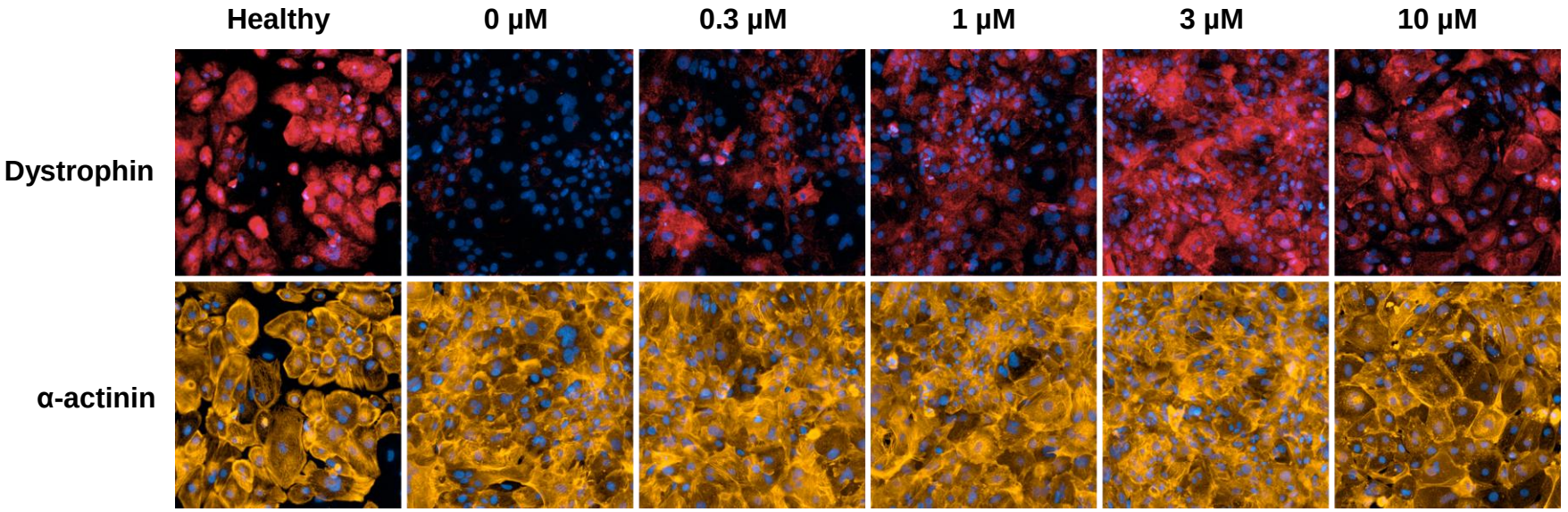


Data are shown as mean ± SD (n=3 skeletal muscle; n=4 cardiac muscle); one-way ANOVA; *p<0.05, ****p<0.0001; relative to untreated DMDΔ46-48. Concentrations provided are PMO equivalent.

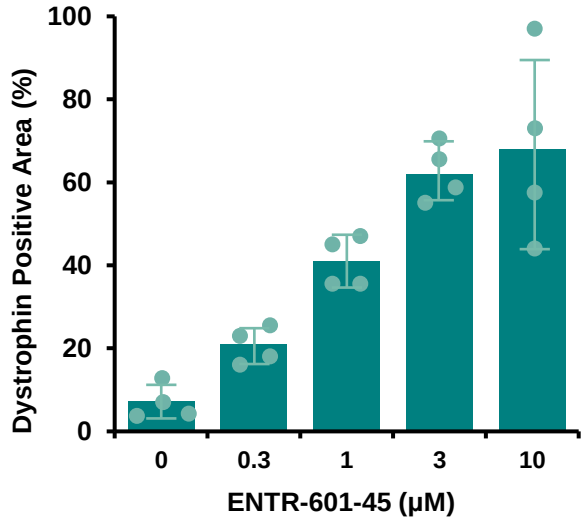
ENTR-601-45 in Cardiac Muscle Cells

ENTR-601-45 produced dose-dependent dystrophin restoration in patient-derived cardiac muscle cells

DMDΔ46-48 Cardiac Muscle Cells + ENTR-601-45



DMDΔ46-48 Cardiac Muscle Cells



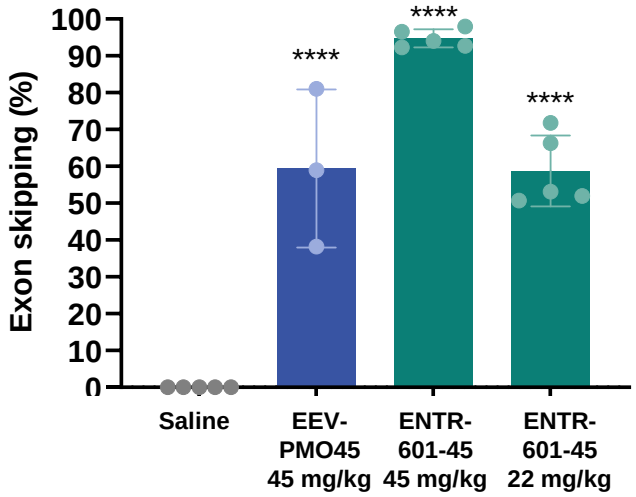
- DMD patient-derived cardiac muscle cells (DMDΔ46-48, n=4) were treated with ENTR-601-45 for 24 hours and analyzed 48 hours later

DMDΔ46-48 iPSC-derived cardiac muscle cells from an exon 45 skip amenable DMD patient harboring an exon 46-48 deletion mutation were treated for 24 hours and analyzed 48 hours later. Data are shown as mean ± SD.

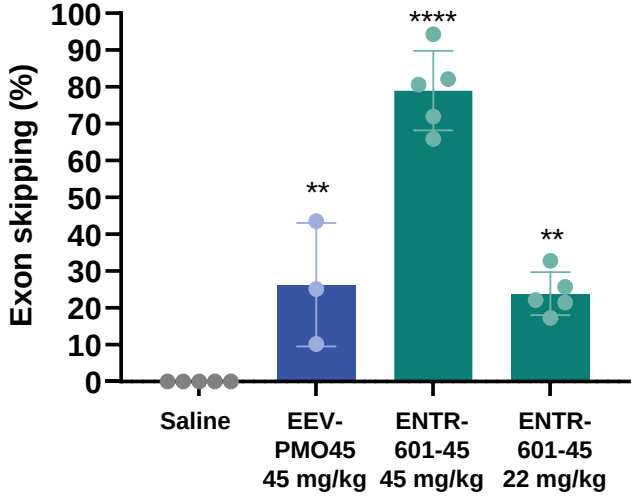
ENTR-601-45 Target Engagement in Human DMD Mice

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

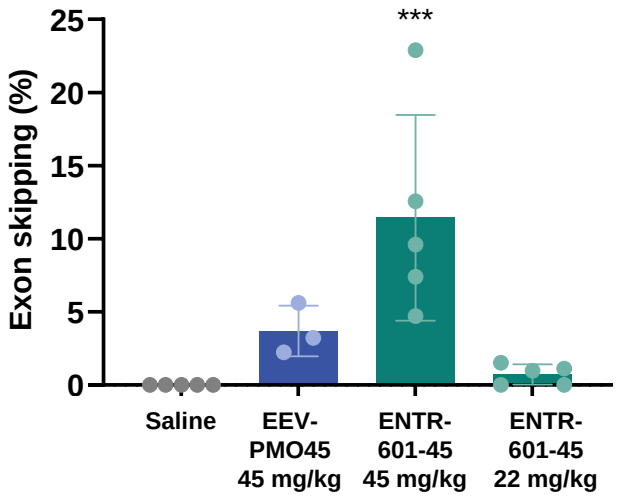
Tibialis Anterior



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 (casimersen sequence) and ENTR-601-45 utilize the same EEV
- EEV-PMO45 uses the same oligonucleotide sequence as casimersen

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.

ENTR-601-45 Data Summary

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

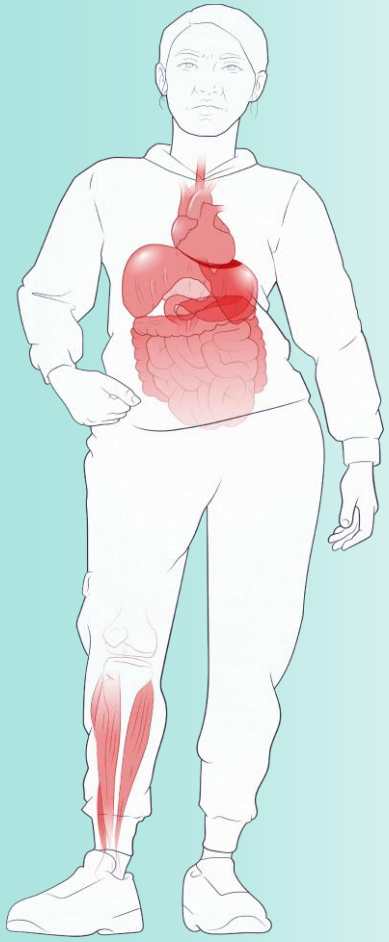
- **Patient-derived Cells**
  - ENTR-601-45 showed robust exon skipping and dystrophin protein production in patient-derived cardiac and skeletal muscle cells
- **DMD mouse models**
  - High levels of exon skipping were measured in hDMD mouse heart and skeletal muscle tissue
  - Exon 44 deletion mouse amenable to exon 45 skipping has been generated and population is being expanded externally
- **Process development**
  - Non-GMP ENTR-601-45 generated to support GLP toxicology studies
  - 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 **U.S. 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

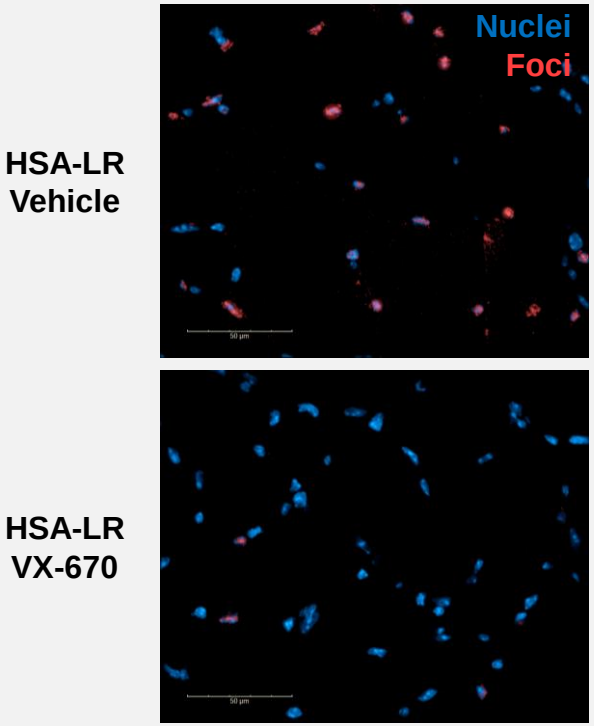
EEV-Oligonucleotide Approach

VX-670 (ENTR-701) targets the underlying cause of DM1 and has the potential to restore normal cell function via a highly-specific CUG-repeat steric blocking approach

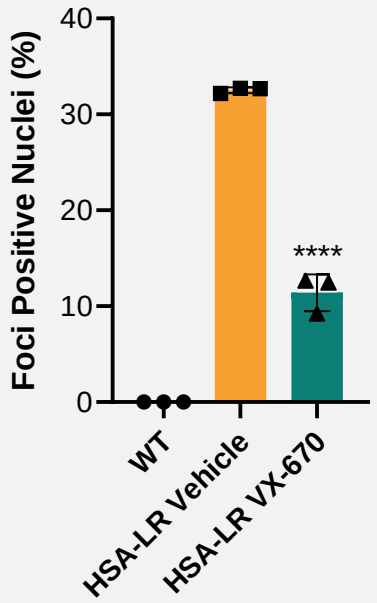
VX-670 (ENTR-701) Efficacy in HSA-LR Mice

VX-670 (ENTR-701) treatment reduced nuclear foci and CUG-repeat expansion transcript level and corrected aberrant splicing in HSA-LR mice, in a dose dependent manner

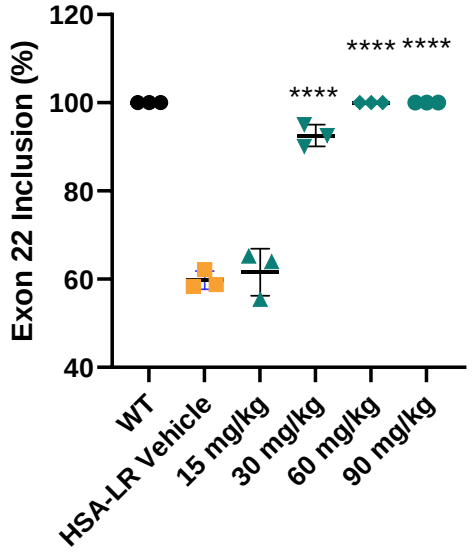
Nuclear Foci Reduction



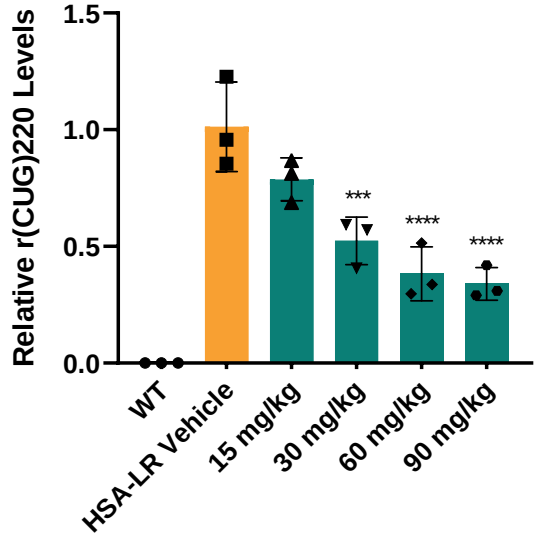
Tibialis anterior section



Atp2a1 Splicing Correction



HSA-r(CUG)220 Reduction

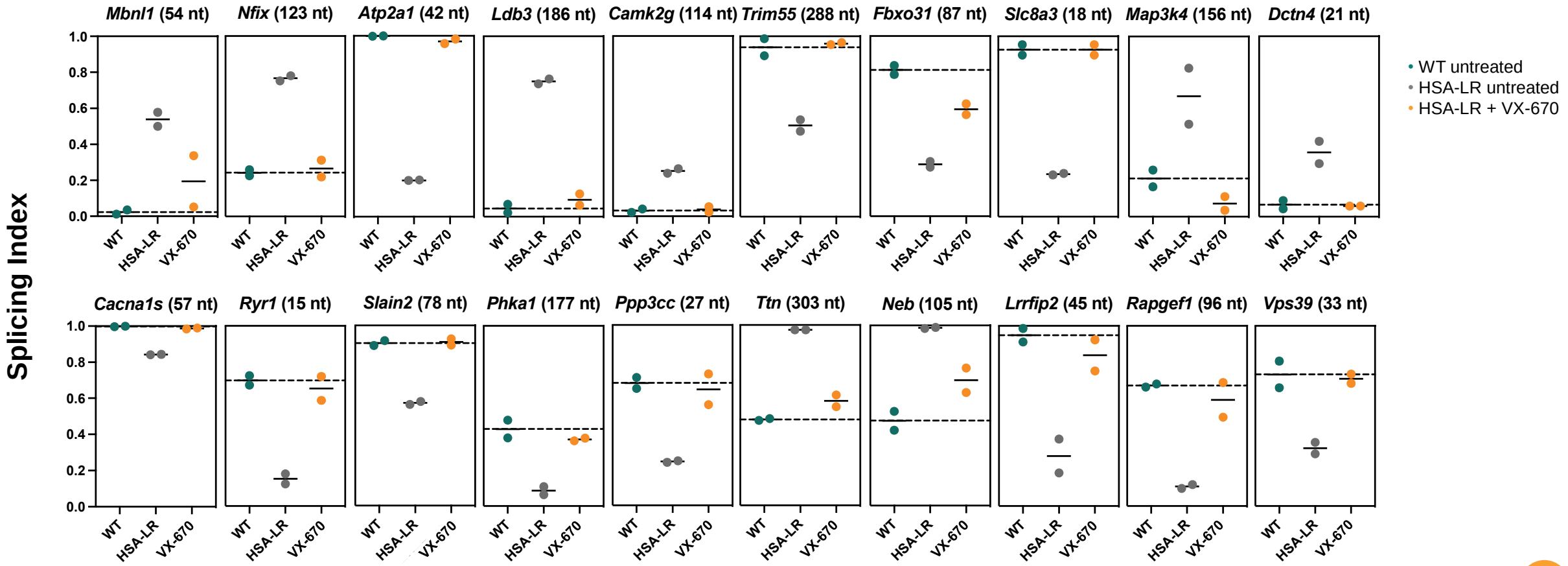


- **HSA-LR** model carries a transgene resulting in CUG repeat expansion and recapitulates DM1 phenotype and molecular pathology
- HSA-LR mice were dosed with vehicle or VX-670 and taken down 1-week post injection; tibialis anterior samples analyzed as a representative skeletal group

VX-670 (ENTR-701) is the clinical candidate selected for DM1, composed of CUG-repeat blocking PMO conjugated to EEV; ***p<0.001, ****p<0.0001, shown as mean ± standard deviation.

VX-670 (ENTR-701) Corrected Spliceopathy in HSA-LR Mice

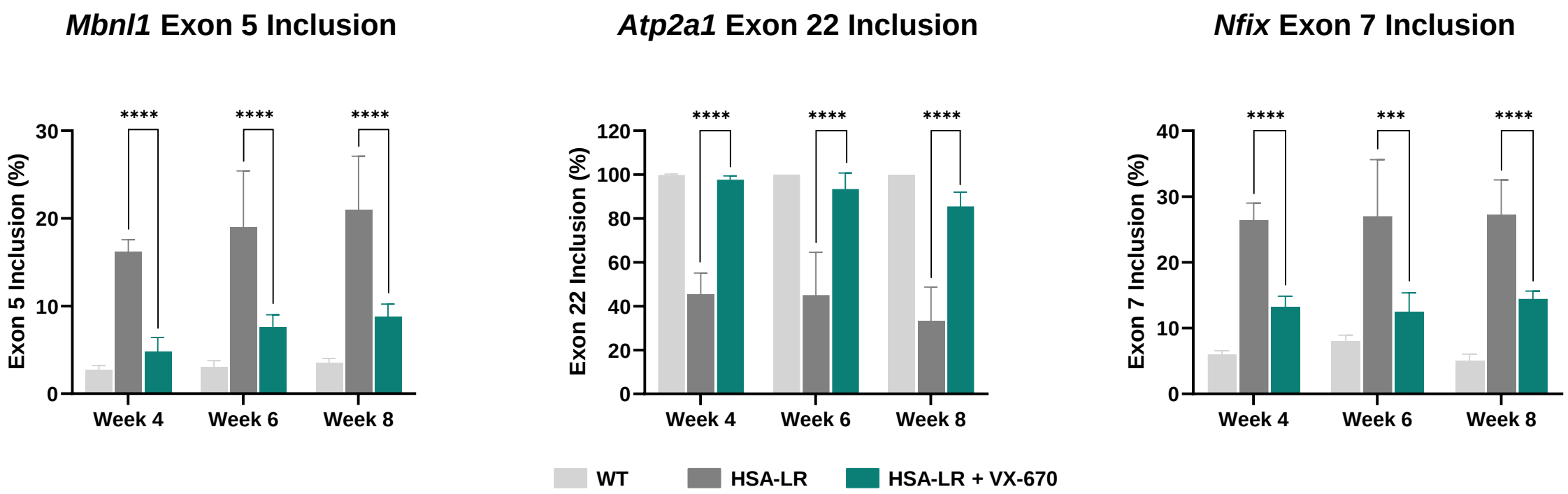
A single dose of VX-670 (ENTR-701) demonstrated substantial and robust splice correction across a panel of 20 different genes



DM1-affected splicing events analyzed by RNA-seq; VX-670 (ENTR-701) is the clinical candidate selected for DM1, composed of CUG-repeat blocking PMO conjugated to EEV.

VX-670 (ENTR-701) Durability in HSA-LR Mice

A single dose of VX-670 (ENTR-701) resulted in splicing correction in HSA-LR mice for at least 8 weeks



- Post single IV dosage of 60 mg/kg (PMO equivalent), robust splicing correction in the VX-670 treated HSA-LR mice 4, 6 or 8 weeks post injection

VX-670 (ENTR-701) is the clinical candidate selected for DM1, composed of CUG-repeat blocking PMO conjugated to EEV; *Mbnl1*, muscleblind like splicing regulator 1; *Atp2a1*, sarcoplasmic/endoplasmic reticulum calcium ATPase; *Nfix*, nuclear factor I X; ***p<0.001, ****p<0.0001, shown as mean ± standard deviation.

DM1 Program Summary

Transformational collaboration with Vertex for the discovery and development of EEV-therapeutics for the potential treatment of DM1

- Robust *in vitro* and *in vivo* data support the development of ENTR-701 (VX-670)
  - Demonstrated potential to address the underlying cause of DM1 and restore normal cell function via a CUG-repeat steric blocking approach
  - Single dose of ENTR-701 demonstrated durable splicing correction and amelioration of myotonia for at least 8 weeks post-dose in HSA-LR model
- **Vertex initiated a global Phase 1/2 clinical trial in patients with DM1 in January 2024**

VERTEX

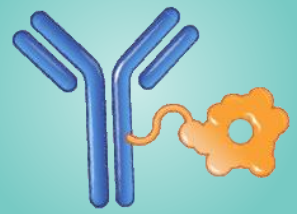
February 2023 Partnership Terms: $224M upfront payment and $26M equity investment; Up to $485M for the achievement of certain milestones, plus royalties; and Four-year global research collaboration

Pipeline Expansion

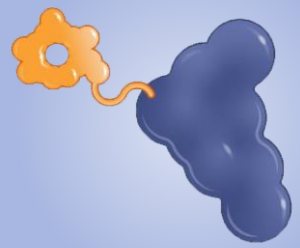
A Broadly Applicable Approach

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

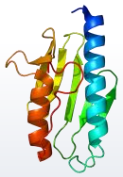
Antibodies



Enzymes

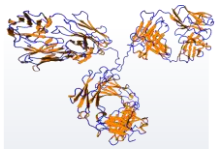


Oligonucleotides



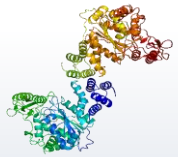
550-600 kDa

Hybrid frataxin



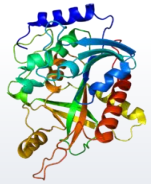
150 kDa

Antibody



98 kDa

Thymidine phosphorylase

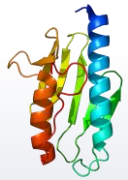


96 kDa

Purine nucleoside phosphorylase

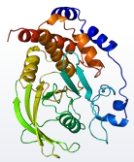
86 kDa

Alanine-glyoxylate aminotransferase



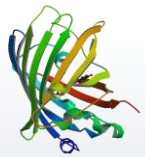
46 kDa

Human frataxin



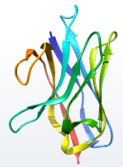
37 kDa

PTP1B catalytic domain



32 kDa

EGFP



16 kDa

Nanobody

6 kDa

Oligonucleotide

1-3 kDa

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 well 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

*Assumes $352 million cash, cash equivalents and marketable securities as of December 31, 2023, together with Vertex collaboration ongoing research support and achievement of certain milestones.

entrada THERAPEUTICS

Entrada is well capitalized to deliver ENTR-601-44 Phase 1 clinical data and progress the broader pipeline

- **Strong Financial Position**
  - Cash runway: Through the second quarter of 2026*
  - Cash, cash equivalents and marketable securities: ~$352M
  - Common shares outstanding on December 31, 2023: 33,452,041
- **Award-Winning Team and Culture**
  - ~150 employees: 75% have advanced degrees and 50% have PhDs
  - Seasoned leadership team across functions
  - Recognized as a Top Place to Work by The Boston Globe, BioSpace and MassEcon
- **Deep Patent Portfolio**
  - 58 patient families on file, including exclusive EEV platform rights
  - 12 families with one or more granted patents

Leadership Team and Board of Directors

Dipal Doshi
Chief Executive Officer

Nathan Dowden
President and Chief Operating Officer

Natarajan Sethuraman, PhD
Chief Scientific 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

An Expanding Pipeline of Intracellular Therapeutics

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 completed dosing of a third cohort in its Phase 1 trial with data expected in H2 2024; Regulatory applications expected in Q4 2024 for global Phase 2 clinical trial
  - ENTR-601-45 regulatory applications expected in Q4 2024 for global Phase 2 clinical trial
  - ENTR-601-50 regulatory applications expected in 2025 for global Phase 2 clinical trial
- **Transformative Vertex partnership for the development of myotonic dystrophy type 1 (DM1)**
  - VX-670 (ENTR-701) Phase 1/2 clinical trial initiated
  - $224M upfront payment and $26M equity investment; Up to $485M for the achievement of certain milestones, plus royalties; Four-year global research collaboration
- **Extending the pipeline with novel intracellular therapeutic candidates by leveraging new moieties and targeting additional therapeutic areas**
- **Strong financial position with cash runway through the second quarter of 2026***

*Assumes $352 million cash, cash equivalents and marketable securities as of December 31, 2023, together with Vertex collaboration ongoing research support and achievement of certain milestones.


Learn more at
EntradaTx.com
entrada
THERAPEUTICS