Building the World's Leading Muscle Disease Company

COMPANY OVERVIEW | MAY 2023

Jordan, *living with DMD*

Forward-Looking Statements

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 Dyne's strategy, future operations, prospects and plans, objectives of management, the potential of the FORCE platform, the anticipated timelines for reporting data for the DYNE-251 and DYNE-101 trials, the trial design of the DYNE-251 and DYNE-101 clinical trials, and the sufficiency of Dyne's existing cash resources for the period anticipated, 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. Dyne 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 initiation and completion of preclinical studies and clinical trials; uncertainties as to the availability and timing of results from preclinical studies and clinical trials; the timing of and Dyne's ability to initiate and enroll patients in clinical trials; whether results from preclinical studies will be predictive of the results of later preclinical studies and clinical trials; whether Dyne'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 Dyne's filings with the Securities and Exchange Commission (SEC), including the Company's most recent Form 10-Q and in subsequent filings Dyne may make with the SEC. In addition, the forward-looking statements included in this presentation represent Dyne's views as of the date of this presentation. Dyne anticipates that subsequent events and developments will cause its views to change. However, while Dyne 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 Dyne's views as of any date subsequent to the date of this presentation.

This presentation also contains estimates, projections and other statistical data made by independent parties and by the Company relating to market size and growth and other data about the Company's industry and business. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. The Company has not independently verified the accuracy and completeness of the information obtained by third parties included in this presentation. In addition, projections, assumptions and estimates of the Company's future performance and the future performance of the markets in which the Company operates are necessarily subject to a high degree of uncertainty and risk.

OUR MISSION

Life-transforming therapies

for patients with serious muscle diseases

Dyne: Building the Leading Muscle Disease Company

Proprietary FORCE Platform

- Modern oligo therapeutics for muscle diseases
- Overcoming challenge of muscle delivery
- Plug-and-play therapeutics for multiple targets in skeletal, cardiac and smooth muscle diseases

Rare Muscle Disease Focus

- Robust pipeline: DM1, DMD, and FSHD
- Set standard for evaluating PD in DM1 disease model
- Significant exon skipping & dystrophin expression in DMD
- Significant market opportunities

Delivering for Patients

- Developing multiple first-in-class or best-in-class therapies
- Two clinical-stage programs in DM1 and DMD, advancing FSHD to the clinic
- Clinical data in DM1 and DMD expected in H2 2023

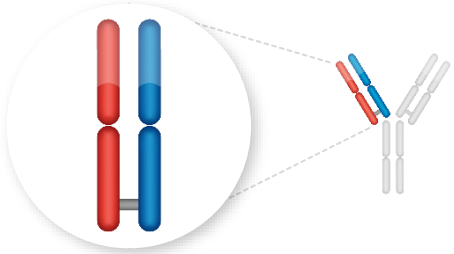
Exceptional Team

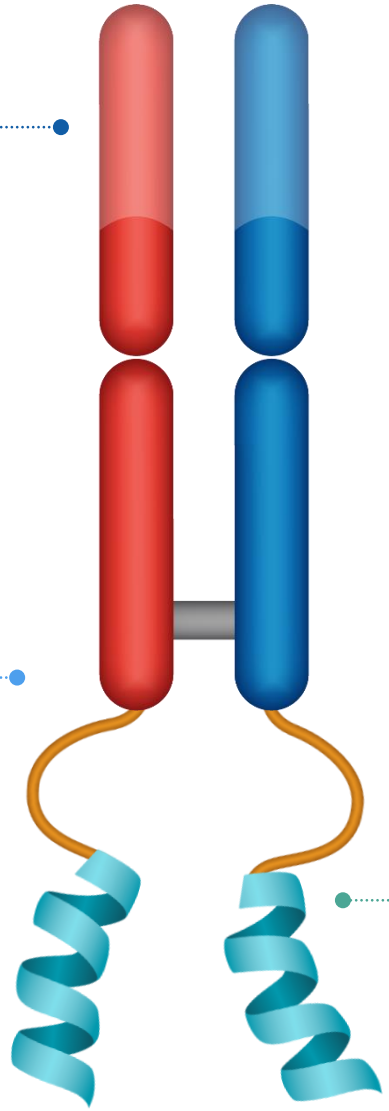
- Deep knowledge of muscle diseases and novel therapeutic modalities
- World-class scientific advisory board
- Supported by leading healthcare investors

Dyne FORCE™ Platform: Modern Oligo Therapeutics for Muscle Diseases

ANTIBODY

Proprietary Fab targets TfR1 to enable muscle delivery



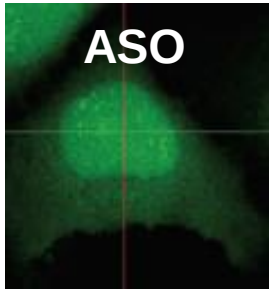


LINKER

Clinically validated, enables precise conjugation of multiple payloads to a single Fab

PAYLOAD

Modularity enables rational selection of payload to target the genetic basis of disease

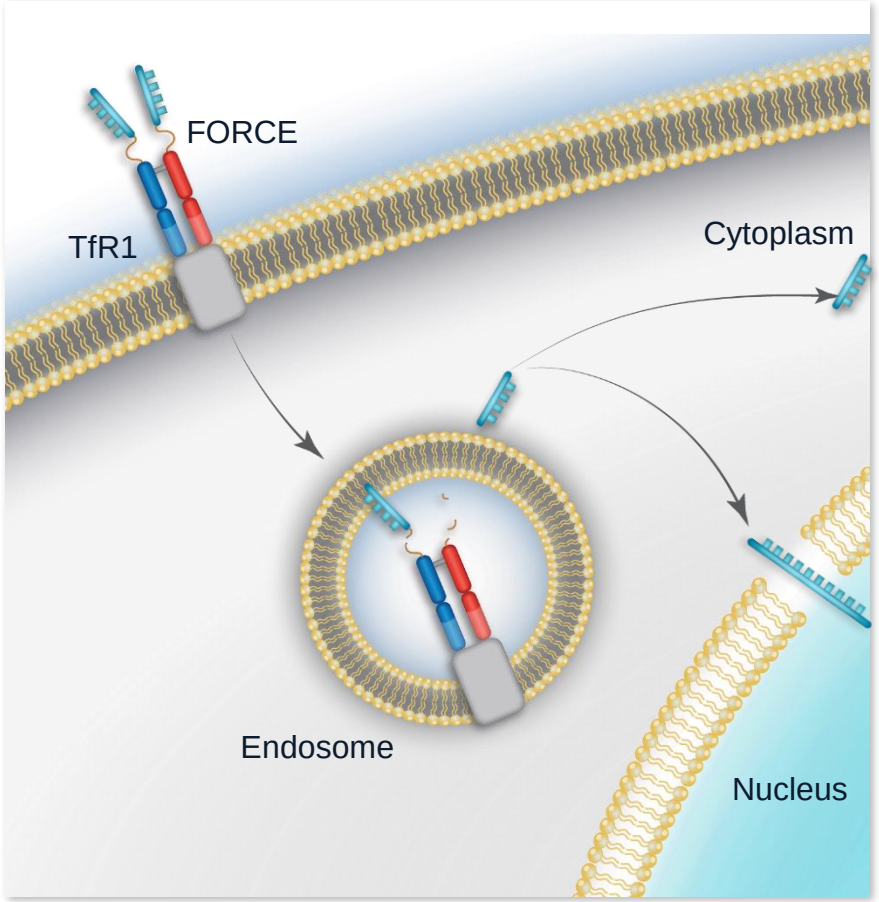


Nuclear localization

Cytoplasmic localization

Adapted from Ohrt T., et al. *Nucleic Acids Res* 2006;34:1369.

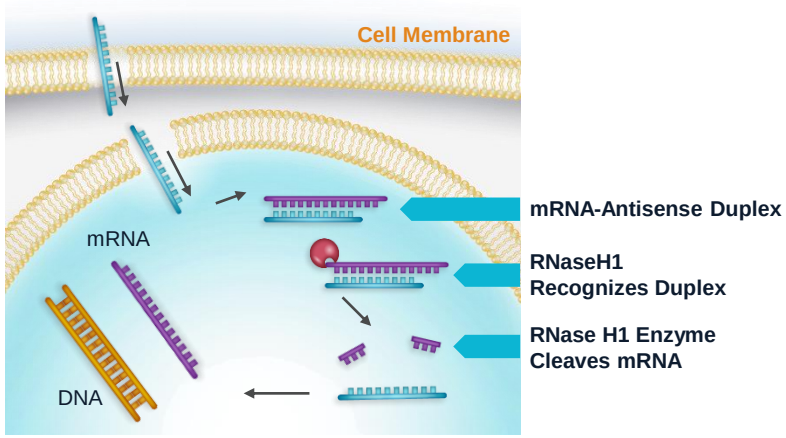
FORCE Platform Harnesses Cell Biology to Modify Disease



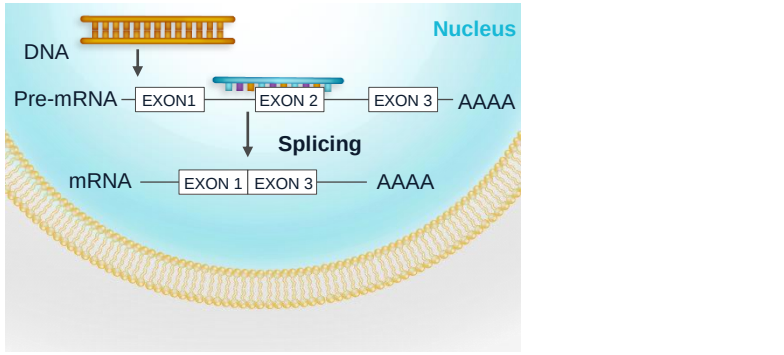
- Harnesses natural mechanism of TfR1 receptor-mediated delivery to transport therapeutics across the cell membrane
- Achieves endosomal escape without any membrane-destabilizing agents
- Distinctive pharmacokinetic profile creates opportunity for durable target engagement and wide therapeutic index

Rationally Select Payload to Target Genetic Basis of Disease

ASO acts in the nucleus and cytoplasm

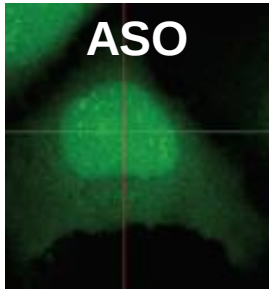


Splice-modulating ASO

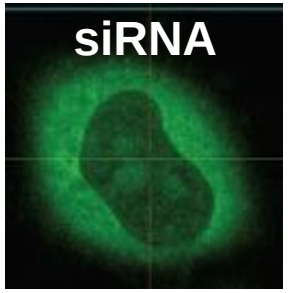


Single-Stranded Antisense

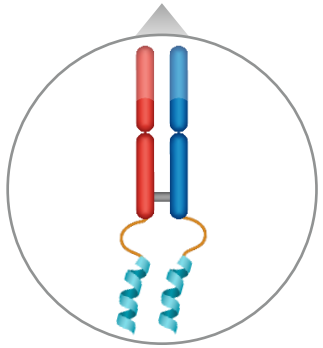
Subcellular distribution of ASO and siRNA



Nuclear localization

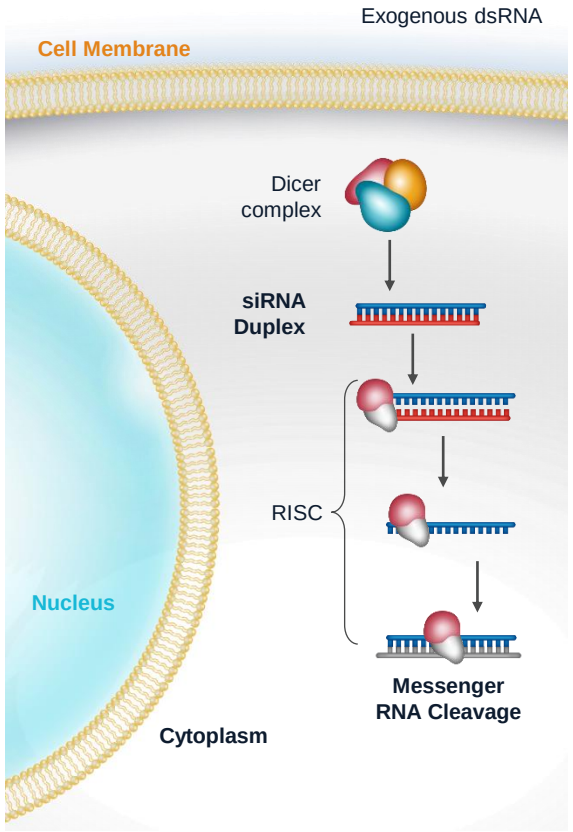


Cytoplasmic localization



FORCE delivers **ASO** payload for nuclear targets, **siRNA** payload for cytoplasmic targets

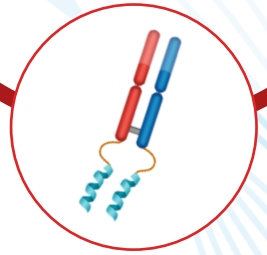
siRNA acts in the cytoplasm



Double-Stranded Antisense (siRNA)

FORCE Platform Designed to Deliver Significant Advantages

Stop or Reverse Disease Progression



Targeted Muscle Delivery

Leverages TfR1 expression on skeletal, cardiac and smooth muscle

Targets Genetic Basis of Disease

Rationally select payloads to match target biology

Redosable Administration

Potential for individualized patient titration and longer-term efficacy

Enhanced Tolerability

Targeted delivery limits systemic drug exposure

Extended Durability

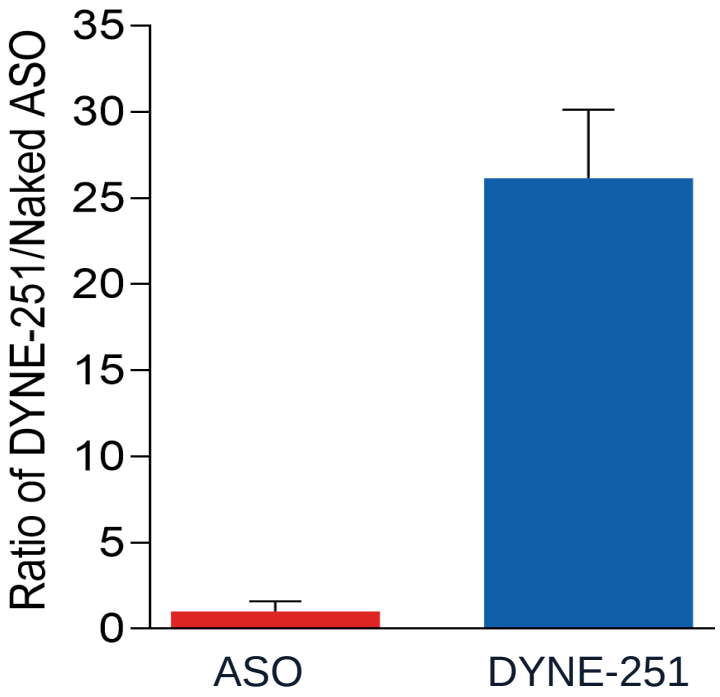
Potential for prolonged disease-modifying effects, enabling less frequent dosing

Reduced Development and Manufacturing Costs

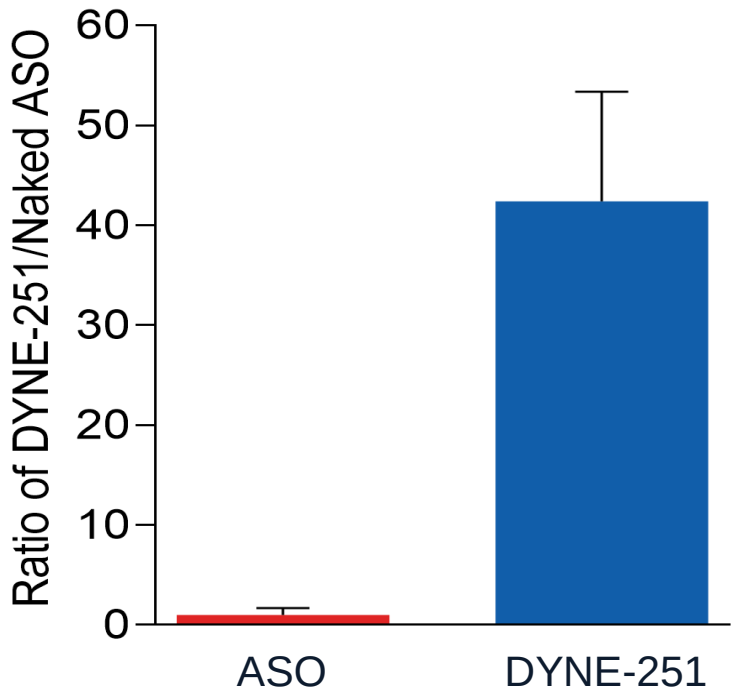
A single Fab and linker utilized across all programs

FORCE's Targeted Delivery Brings the Power of Modern Oligo Therapeutics to Patients Living with Serious Muscle Diseases

Heart



Diaphragm



Note: Results after repeat IV dose of naked ASO or DYNE-251 in male cynomolgus monkeys, 2 x 30 mg/kg on day 0 and day 7, analyzed day 28; n = 4 – 5.

FORCE Platform Delivered Comprehensive & Validating Data Across Three Programs – Poised to Execute in the Clinic

DMD

In vitro:

- Enhanced exon skipping

In vivo:

- Robust, durable exon skipping and dystrophin expression in *mdx* model
- Transformative exon skipping in NHP cardiac and skeletal muscles
- NHP GLP tox results support favorable safety profile

DM1

In vitro:

- *DMPK* KD, reduction in nuclear foci, splicing correction

In vivo:

- Correction of splicing & reversal of myotonia in HSA^{LR} model
- Robust knockdown of toxic nuclear *DMPK* in hTfR1/DMSXL model, foci reduction & correction of splicing
- NHP GLP tox results support favorable safety profile

FSHD

In vitro:

- Reduced expression of key DUX4 biomarkers

In vivo:

- Enhanced tissue distribution in NHP

Robust Portfolio Focused on Muscle Diseases

DISEASE	TARGET	DISCOVERY / PRECLINICAL / PHASE 1/2	ESTIMATED PATIENTS
Myotonic Dystrophy Type 1 (DM1)	DMPK	**DYNE-101** — *Safety, Tolerability & Splicing Data Expected in H2'23*	US: **>40,000** Europe: **>74,000**
Duchenne Muscular Dystrophy (DMD)	Exon 51	**DYNE-251** — *Safety, Tolerability & Dystrophin Data Expected in H2'23*	US: **~12,000-15,000** Europe: **~25,000**
	Exon 53		
	Exon 45		
	Exon 44		
	Other Exons		
Facioscapulohumeral Muscular Dystrophy (FSHD)	DUX4	**DYNE-301**	US: **~16,000-38,000** Europe: **~35,000**

Pipeline Expansion Opportunities

Rare Skeletal

Cardiac

Metabolic

Driving Toward Meaningful Clinical Data in DM1 & DMD in H2 2023

ACHIEVE

DELIVER

Global, Randomized Placebo-Controlled Trials Designed to Be Registrational

Dosing Patients at Predicted Pharmacologically Active Doses

Significant Unmet Patient Need Provides Confidence in Ability to Enroll Rapidly

Safety, Tolerability & Splicing Data Expected in H2 2023

Safety, Tolerability & Dystrophin Data Expected in H2 2023

Cash Runway Expected Through 2024

Experienced, Successful Team Committed to Our Shared Vision

Joshua Brumm
President & CEO
Kaleido · PROTEOLIX · pharmacyclics An AbbVie Company · ZELTIQ

Susanna High
COO
bluebirdbio · Alnylam PHARMACEUTICALS

Wildon Farwell
CMO
Biogen

Oxana Beskrovnaya
CSO
SANOFI GENZYME

Ashish Dugar
Chief Medical Affairs Officer
SAREPTA THERAPEUTICS · Pfizer

Jonathan McNeill
SVP, Business Development
editas MEDICINE · BCG

Debra Feldman
SVP, Head of Regulatory Affairs
Sage Therapeutics™

John Najim
Chief Technical Officer
PROTEON THERAPEUTICS

Amy Reilly
SVP, Head of Corporate Communications & IR
Kaleido · Biogen · CUBIST

Jason Rhodes
Chairman, Founder
ATLAS VENTURE

Muscle Disease Expertise	Rare Disease Drug Development	Manufacture & Commercialization of Novel Therapeutics

Developing Transformative Therapies for People Living with DM1

Overview

- Mutation in the *DMPK* gene
- Onset at any point, depending on DM1 phenotype
- Life expectancy of 45 - 60 years

Clinical Presentation

- Myotonia
- Muscle weakness
- Cardiac arrhythmia
- Pulmonary abnormalities

Population

- >40,000 (US)
- >74,000 (Europe)

NO approved therapies

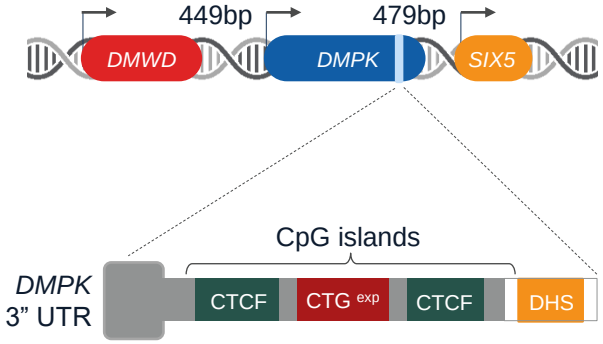
OUR APPROACH

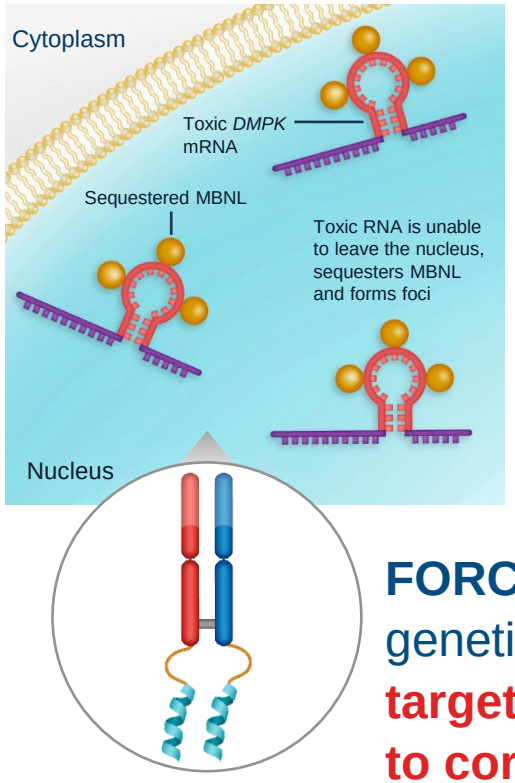
Disease-Modifying Nuclear *DMPK* Knockdown

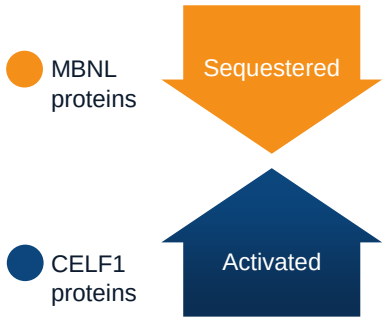
Targeting toxic gain of function *DMPK* RNA to potentially **stop or reverse** disease progression

FORCE Targets the Genetic Basis of DM1 to Correct Splicing

DNA Triplet Repeats → **Toxic RNA Forms Foci in Nucleus** → **RNA Binds Splicing Proteins** → **Clinical Presentation**



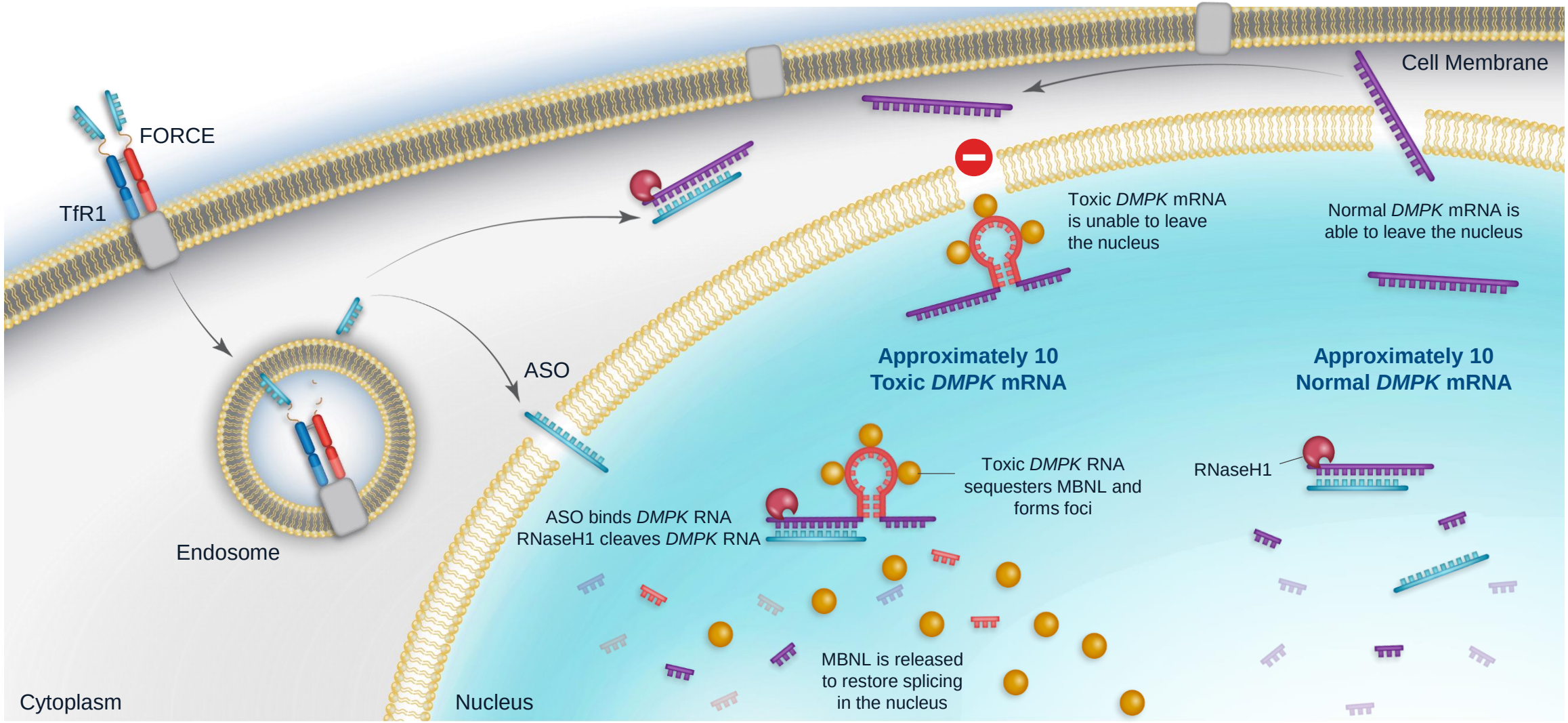




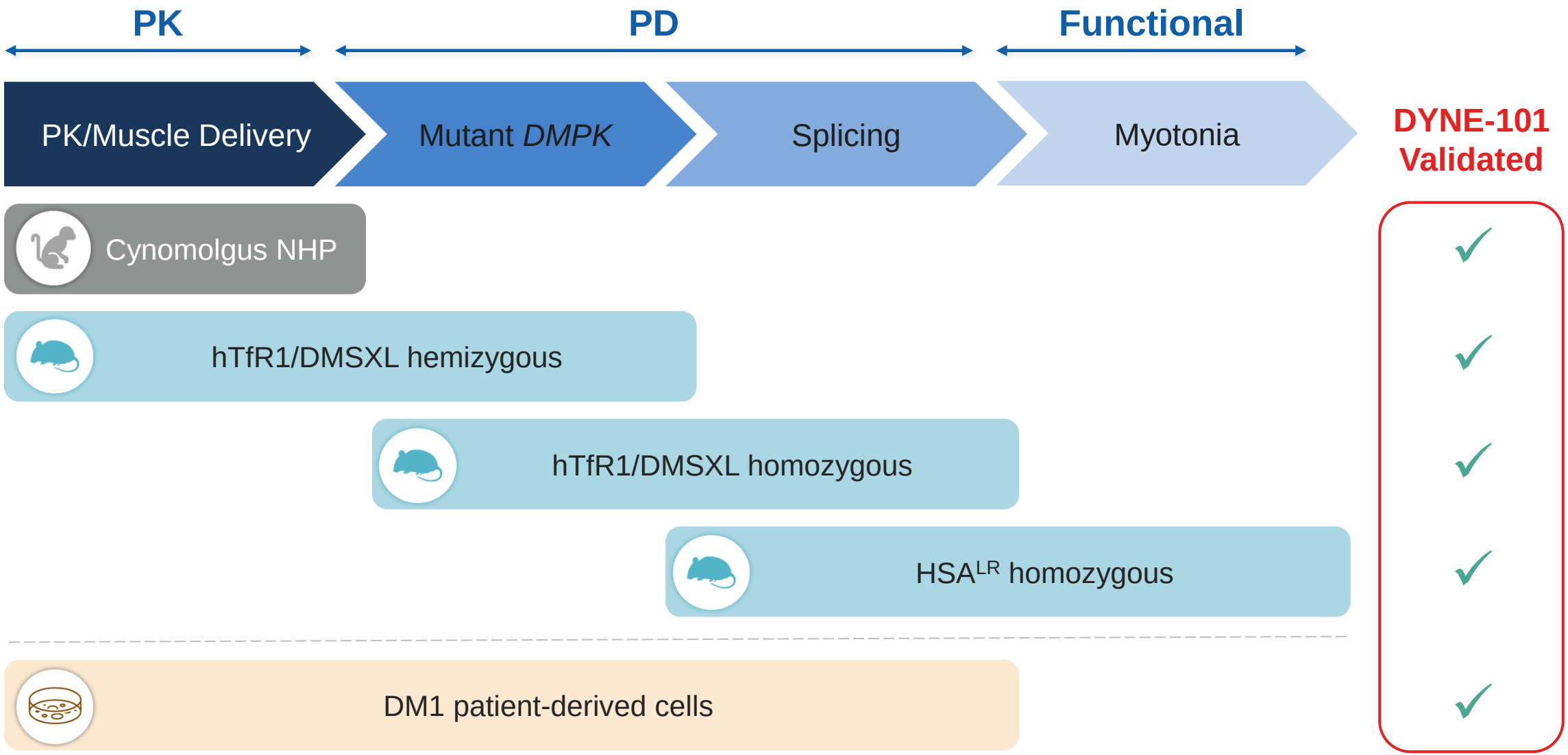
- Myotonia
- Muscle weakness
- Cardiac arrhythmia
- Pulmonary abnormalities

FORCE designed to address the genetic basis of disease by **targeting toxic nuclear *DMPK* RNA to correct spliceopathy**

FORCE Targets Toxic Nuclear *DMPK* RNA



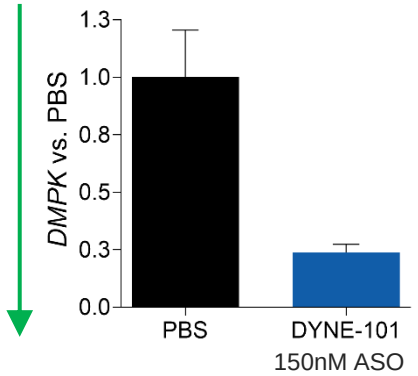
Robust Preclinical Data Supporting the Potential of DYNE-101 to Drive Disease Modification in the Clinic



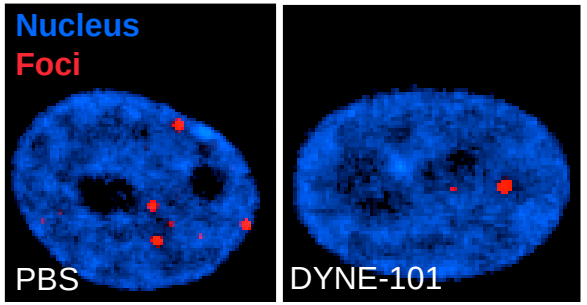
DYNE-101 Demonstrated Robust Dose-Dependent *DMPK* KD, Foci Reduction, and Splicing Correction

380 CTG Repeats DM1 Myotubes

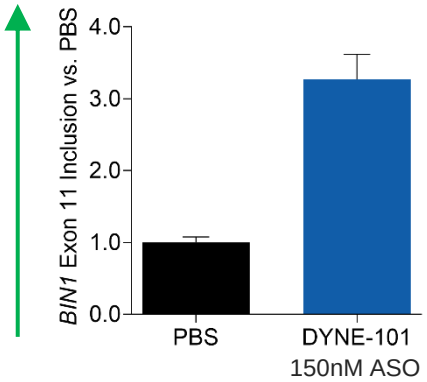
DMPK mRNA KD by qPCR



DMPK foci reduction by FISH

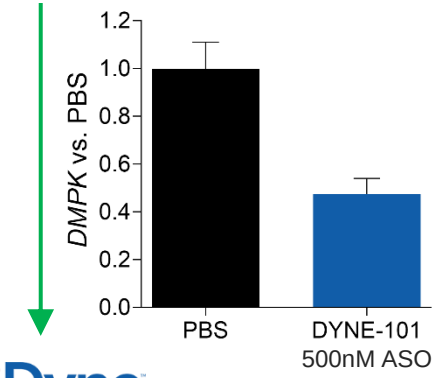


BIN1 mis-splicing correction by qPCR

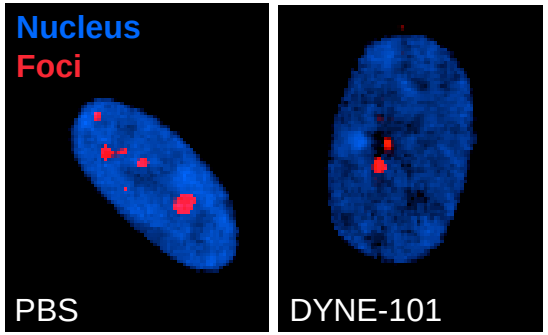


2,600 CTG Repeats DM1 Myotubes

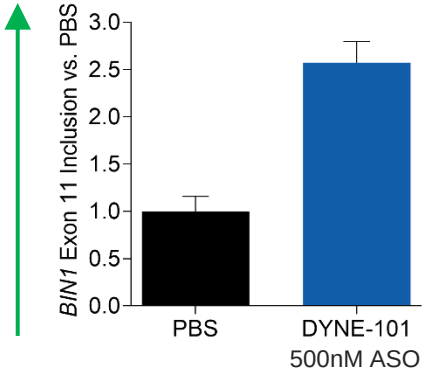
DMPK mRNA KD by qPCR



DMPK foci reduction by FISH



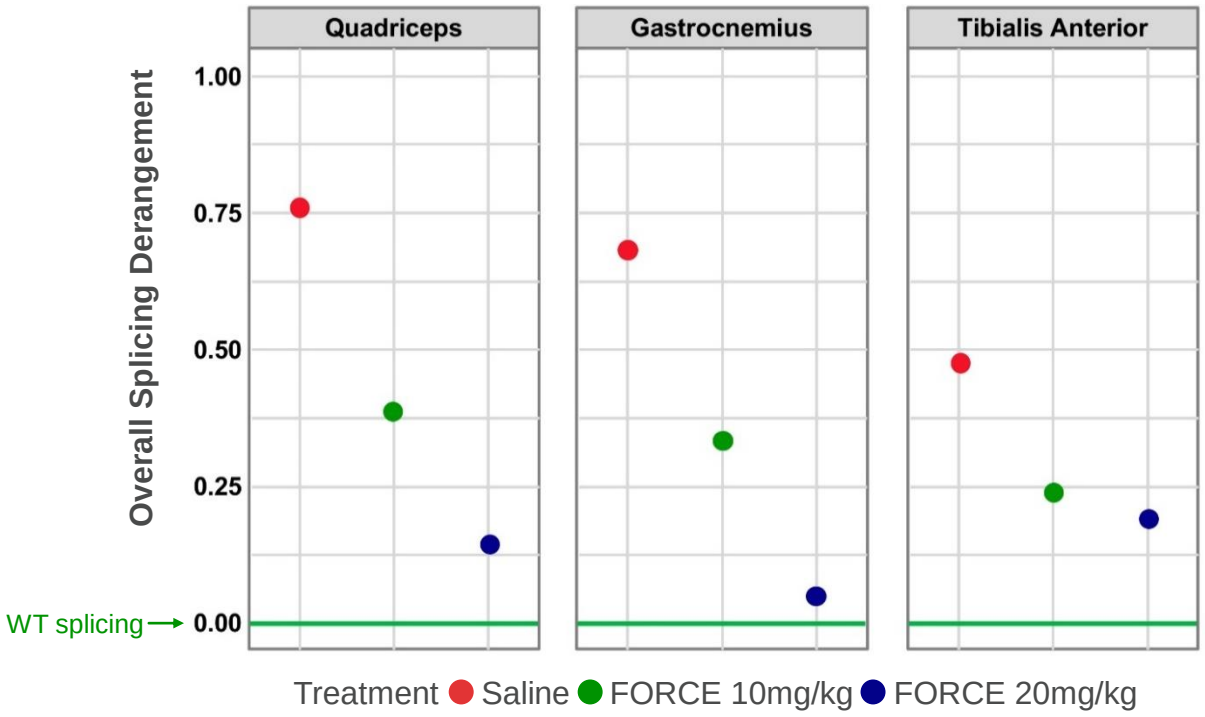
BIN1 mis-splicing correction by qPCR



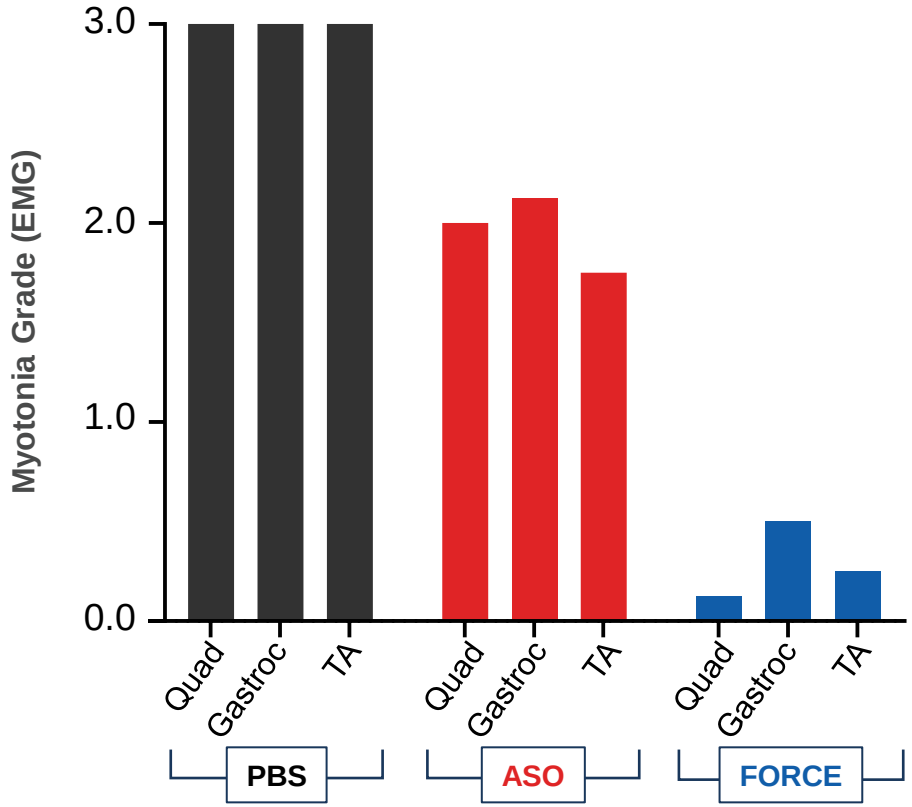
Note: Data are mean ±SD, n=4. Foci reduction based on foci area corrected for nuclear area.

FORCE Dose-Dependently Corrected Splicing and Reversed Myotonia in the HSA^{LR} DM1 Mouse Model

Splicing Correction in Multiple Muscles

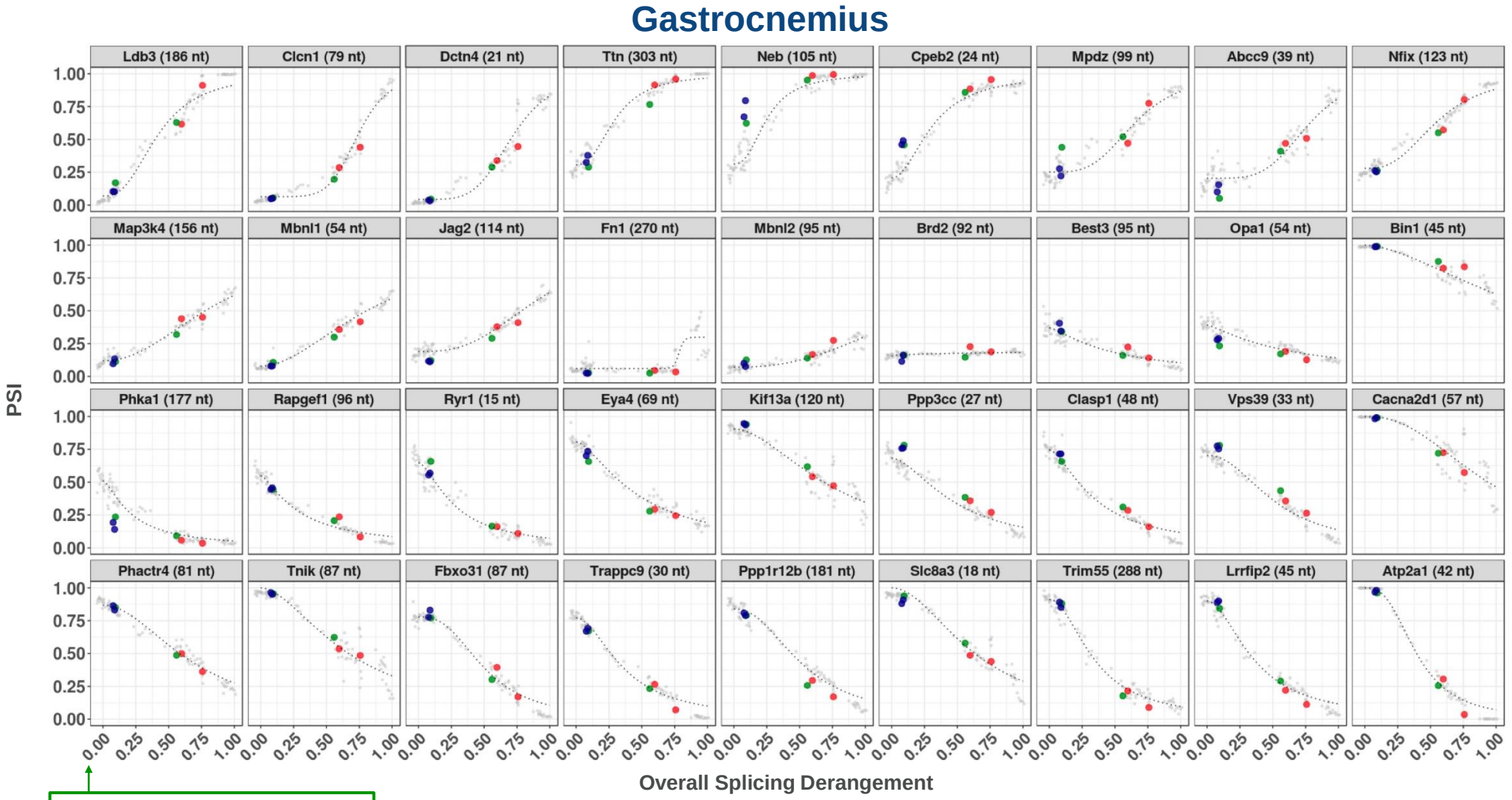


Near Complete Myotonia Reversal Within 14 Days After a Single Low Dose

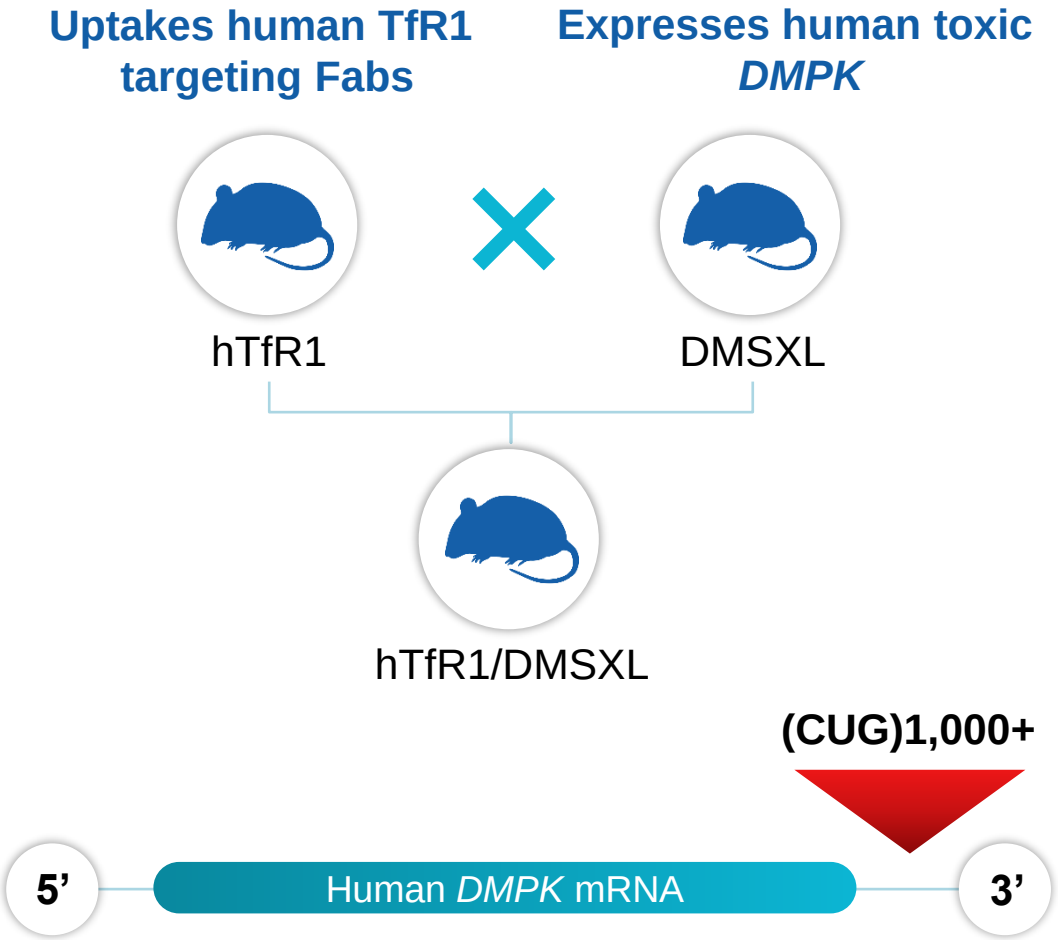


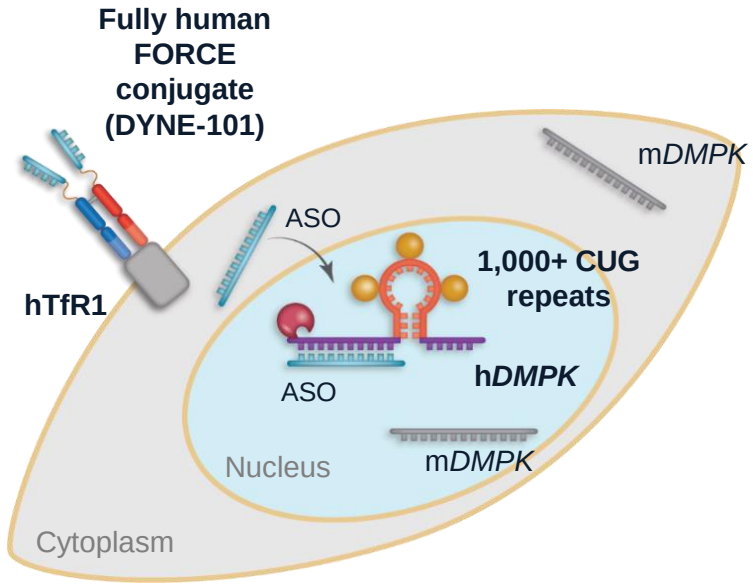
Note: HSA^{LR} mice, single dose 14-day study. Overall splicing derangement indexed to WT level of 0.00. EMG myotonic discharges were graded by a blinded examiner on a 4-point scale: 0, no myotonia; 1, occasional myotonic discharge in less than 50% of needle insertions; 2, myotonic discharge in greater than 50% of needle insertions; 3, myotonic discharge with nearly every insertion.

Robust Nuclear Knockdown by FORCE Drives Dose-Dependent Splicing Correction Across Full Panel of Genes in HSA^{LR} DM1 Mouse Model



hTfR1/DMSXL: Innovative Model Developed by Dyne to Evaluate PD By Measuring Toxic Human Nuclear *DMPK* KD



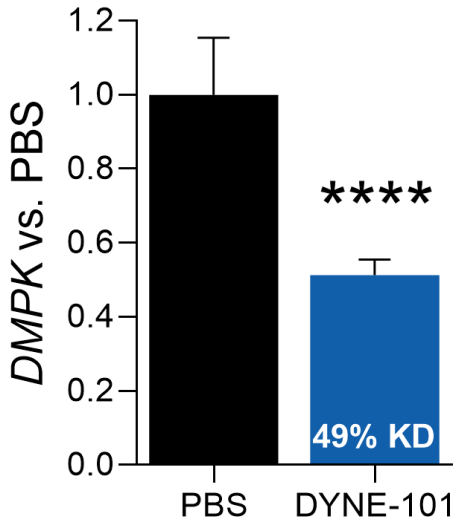


- Expresses human TfR1 receptor, enabling use of human TfR1-targeting Fab
- Underestimates potency, expressing >10 times less human toxic *DMPK* vs. mouse *DMPK*

Note: DMSXL mice first described in PLOS Genetics 2012, 8(11):e1003043.

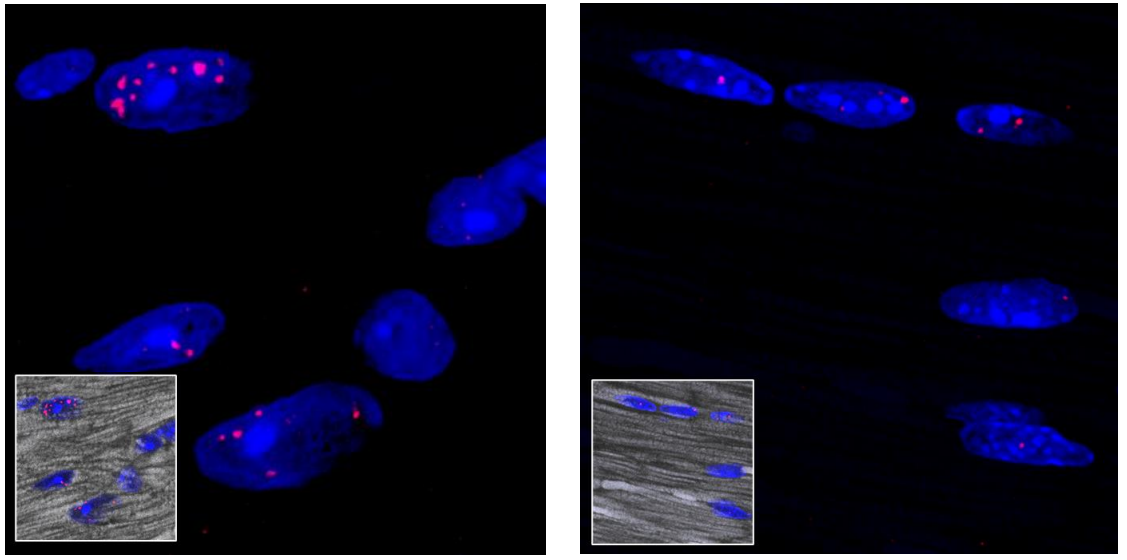
DYNE-101 Demonstrated Toxic *DMPK* KD, Foci Reduction and Splicing Correction in Heart of hTfR1/DMSXL Homozygous Model

Toxic Human *DMPK* RNA KD



Toxic Human *DMPK* Foci Reduction

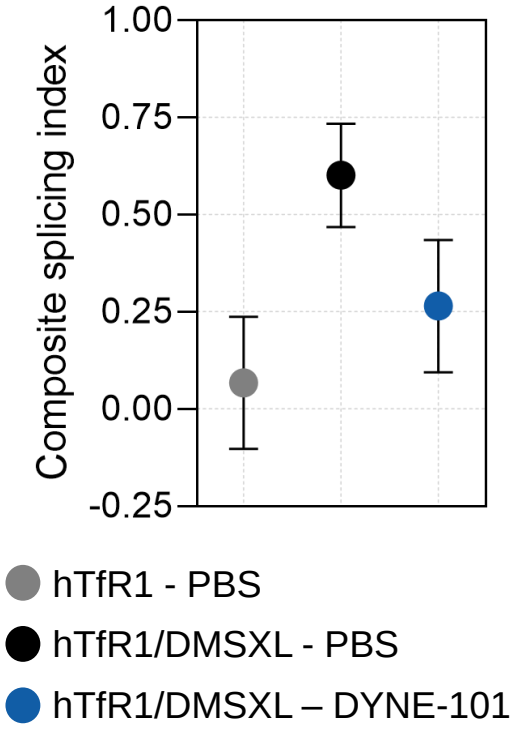
DMPK Foci Nuclei Myofibers



PBS DYNE-101

DYNE-101 reduces foci area by 49%*

Splicing Correction

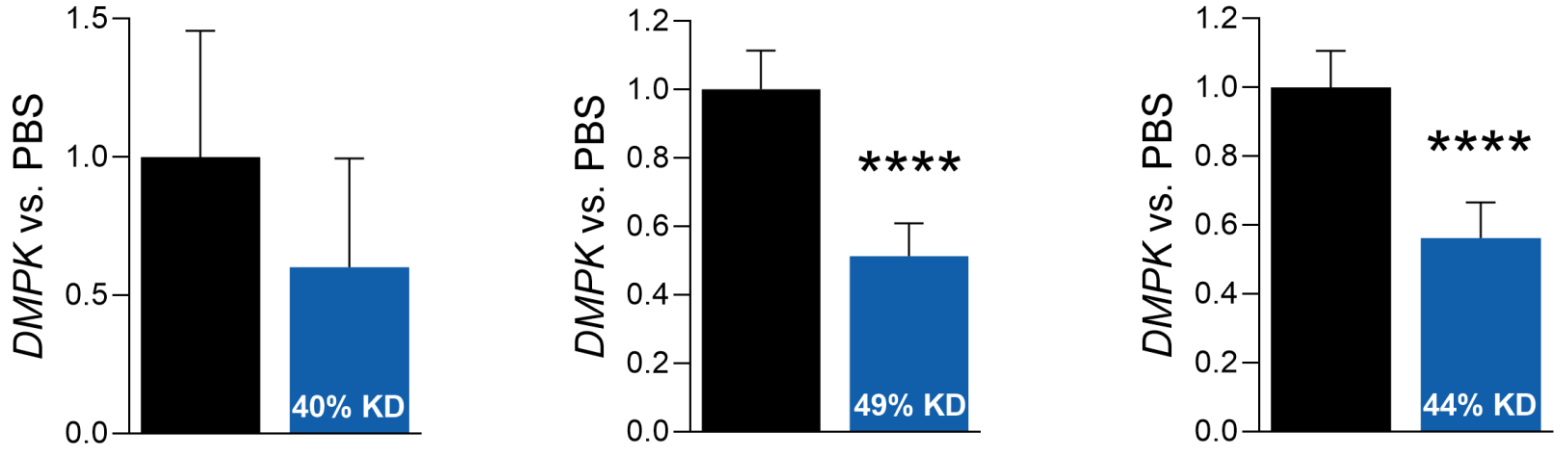


Note: hTfR1/DMSXL homozygous model. 2 x 10 mg/kg on d0 and d7, analyzed d28.
Composite splicing index includes changes in *Ldb3* exon (E) 11, *Mbnl2* E6, and *Nfix* E7. Data are mean ± SD, n = 6 - 7.; * p <0.05; **** p < 0.0001.

DYNE-101 Demonstrated Toxic *DMPK* KD and Splicing Correction in Muscle of hTfR1/DMSXL Homozygous Model

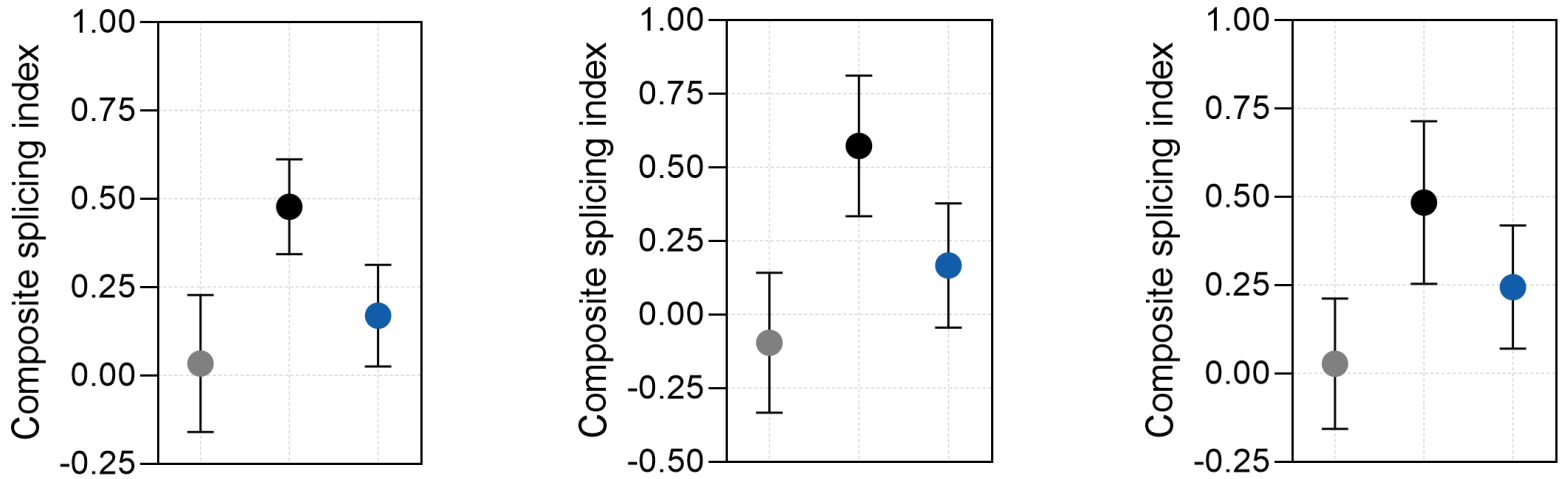
Toxic Human *DMPK* KD

- hTfR1/DMSXL - PBS
- hTfR1/DMSXL – DYNE-101



Splicing correction

- hTfR1 - PBS
- hTfR1/DMSXL - PBS
- hTfR1/DMSXL – DYNE-101



Note: hTfR1/DMSXL homozygous model. 2 x 10 mg/kg on d0 and d7, analyzed d28.
Composite splicing indices include *Bin1* E11, *Insr* E11, *Ldb3* E11, *Mbnl2* E6, *Nfix* E7, and *Ttn* E313 mis-splicing measured by qRT-PCR. Data are means ± SD; n = 4–7; **** $p < 0.0001$ by *t*-test.

DYNE-101 Achieved *DMPK* Knockdown & Well Tolerated in NHPs

Robust WT *DMPK* KD Achieved in Skeletal, Cardiac and Smooth Muscles

- Up to 70% *DMPK* KD at 2 months with low monthly dosing

13-Week GLP Toxicology Study[1]

- No dose limiting toxicity observed up to a maximally feasible dose[2]
- No changes in cardiac, respiratory, neurologic or ophthalmic endpoints
- No effect on kidney function
- No effect on liver function
- No effect on coagulation
- NOAEL was identified at the highest dose tested

Notes: 1. Based on conclusions of report from third-party CRO. 2. Dosed once every three weeks.
NOAEL: No-observed-adverse-effect level

ACHIEVE Trial Informed by Input from Multiple Stakeholders

Global, Multi-disciplinary KOL and Regulatory Input

- Overall design for the MAD portion in patients ages 18 to 49
- Splicing, myotonia, measures of strength & function, key safety considerations
- Natural history data to contextualize clinical trial data for patients and families, clinicians, payers, regulators

Global Advocacy Leaders, Patient and Caregiver Input

- Considerations for trial selection
- Clinical trial protocol and visit schedule
- Minimizing patient burden during trial conduct
- Ensuring support and education to patients and families

Phase 1/2 Clinical Trial to Evaluate DYNE-101 in Patients with DM1

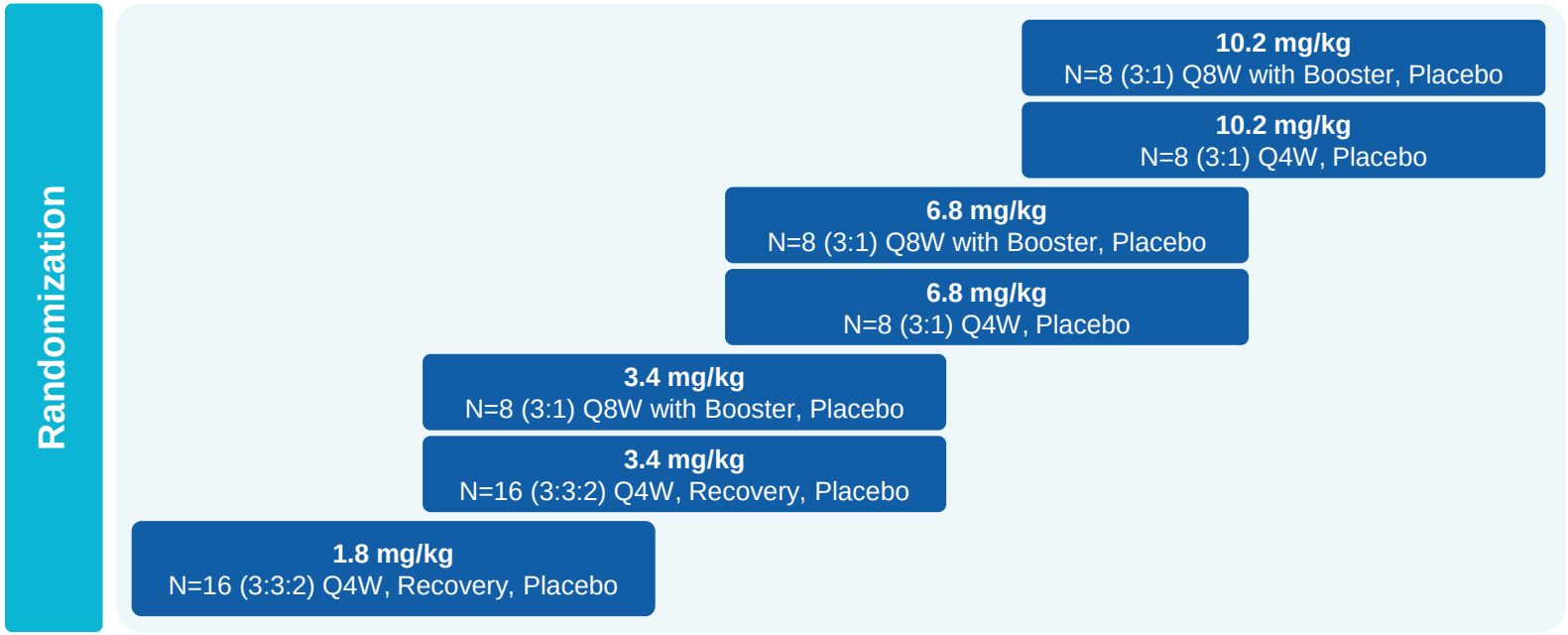
Population	Primary Endpoints	Key Secondary Endpoints	Stages of ACHIEVE
• Adult patients living with DM1 • Ages 18 to 49 years • ~72 adult participants	• Safety and tolerability	• Pharmacokinetics • Change from baseline of: – Splicing – *DMPK* RNA expression – Multiple assessments of muscle strength and function	• Multiple Ascending Dose (MAD): 24 weeks • Open-Label Extension (OLE): 24 weeks • Long-Term Extension (LTE): 96 weeks

Initial Safety, Tolerability & Splicing Data Expected in H2 2023

Multiple Ascending Dose Stage of ACHIEVE

ACHIEVE

Global, Randomized, Placebo-Controlled Stage Evaluating Once Monthly or Less Frequent Administration of DYNE-101 in ~72 Adult Patients Living with DM1



MAD Study Details

- IV administration of DYNE-101 or placebo every 4 weeks or every 8 weeks
- Muscle biopsies: Baseline, 12 weeks, 24 weeks
- Patients in MAD study escalated to highest tolerable dose in OLE and LTE

Global Strategy & Adaptive Trial Design Enable Rapid Achievement of Potentially Registrational Clinical Data

Doses provided refer to ASO component of DYNE-101. Recovery cohort Q4W x 2 doses then placebo for the remainder of the 24W placebo-controlled period. Q8W with booster includes Q4W x 3 doses then Q8W dosing.

DM1 Program Summary

Validating Data

- **Targeted** toxic *DMPK* in the nucleus in patient cells
- **Robust and durable toxic human *DMPK* KD** in novel hTfR1/DMSXL model
- **Reduced nuclear foci** *in vitro & in vivo*
- **Corrected splicing** changes *in vitro & in vivo*
- **Reversed myotonia** in HSA^{LR} model
- **Delivered** *DMPK* targeting ASO to mouse and NHP muscle tissues
- **Favorable safety profile in NHP GLP tox study**

Potential Advantages

- **Tractable development** with rapid path to human PoC
- **Efficient** commercial model, addressable with focused sales force

Initial Safety, Tolerability & Splicing Data Expected in H2 2023

Building a Global DMD Franchise of Transformative Therapies

Overview

- Mutation in the *DMD* gene that encodes for dystrophin
- Onset in first few years of life
- Life expectancy ~30 years

Clinical Presentation

- Muscle weakness
- Progressive loss of function
- Loss of ambulation
- Respiratory/cardiac failure

Population

- ~12,000 - 15,000 (US)
- ~ 25,000 (Europe)

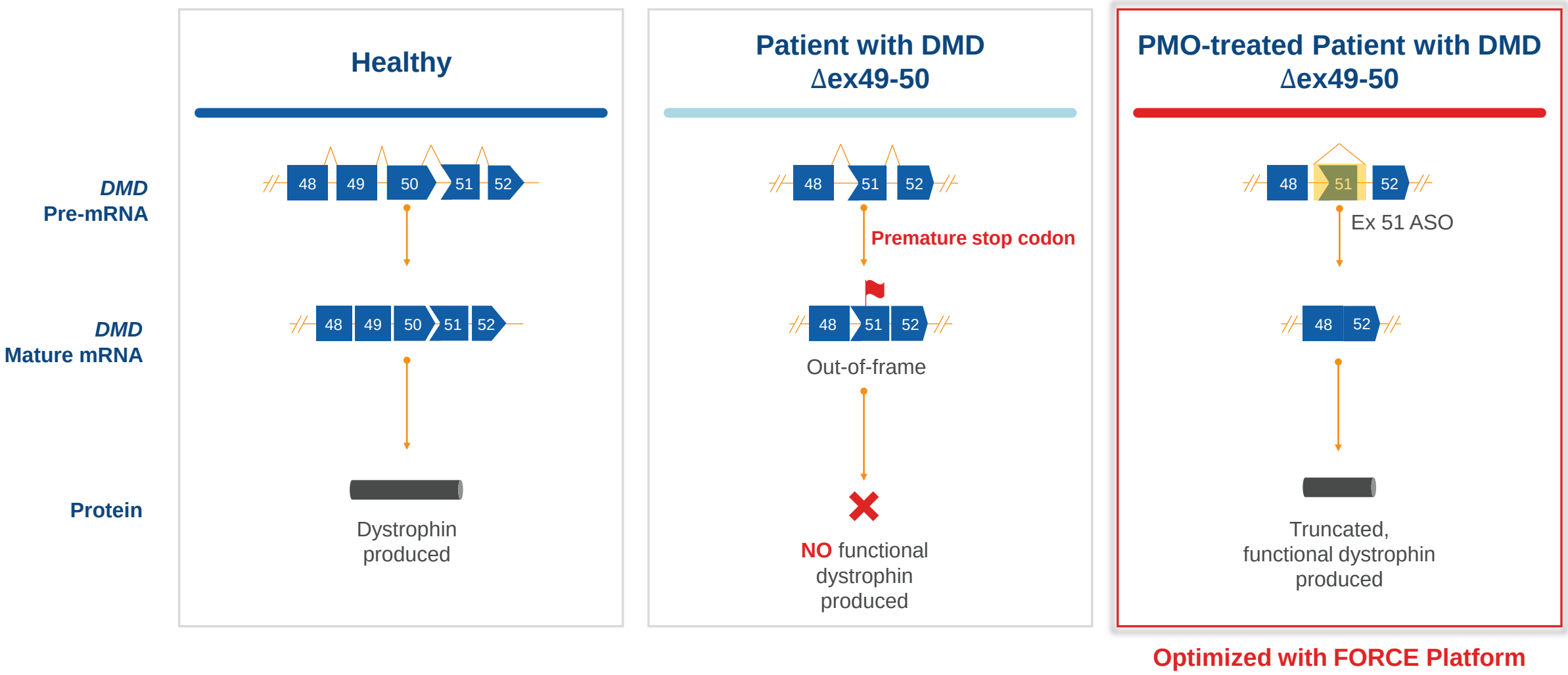
Current Approved Exon 51 Therapies Only Increased Dystrophin Production <1%

OUR APPROACH

Best-in-class Targeted Exon Skipping

Increase dystrophin expression and enable less frequent dosing to potentially **stop or reverse disease progression**

Targeting the Genetic Basis of DMD with a Proven Mechanism

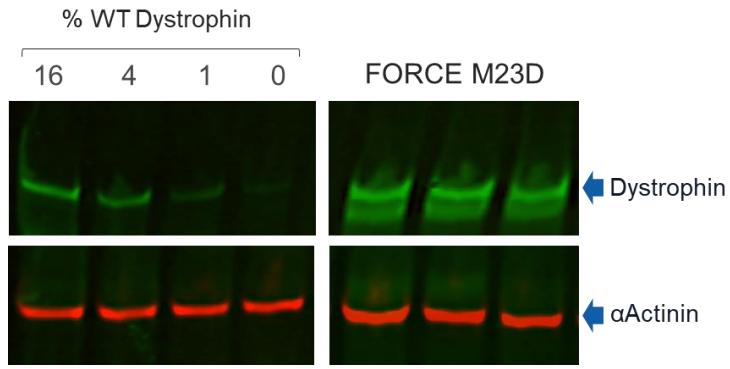


Optimized with FORCE Platform

Adapted from Lim KRQ., et al. *Drug Des Devel Ther* 2017;11:533.

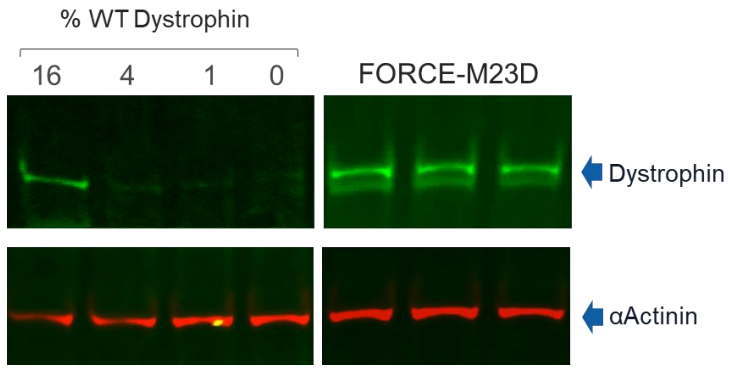
FORCE Achieved Robust and Durable Dystrophin Expression and Sarcolemma Localization in Muscle

Quadriceps



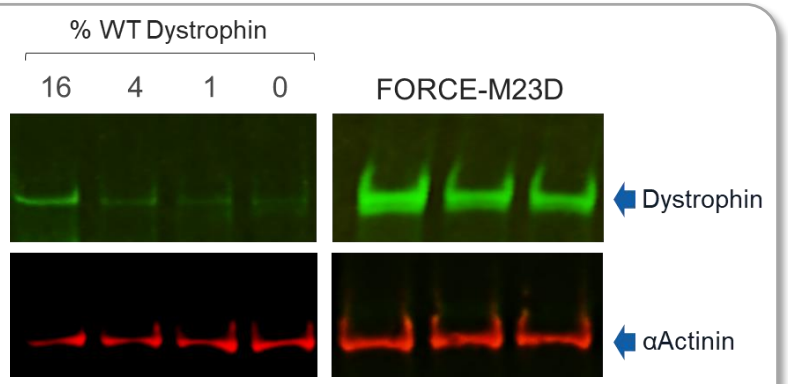
51% of wild-type dystrophin

Diaphragm

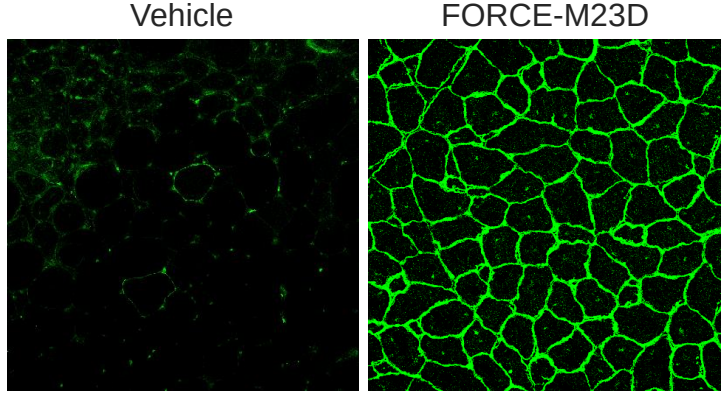


90% of wild-type dystrophin

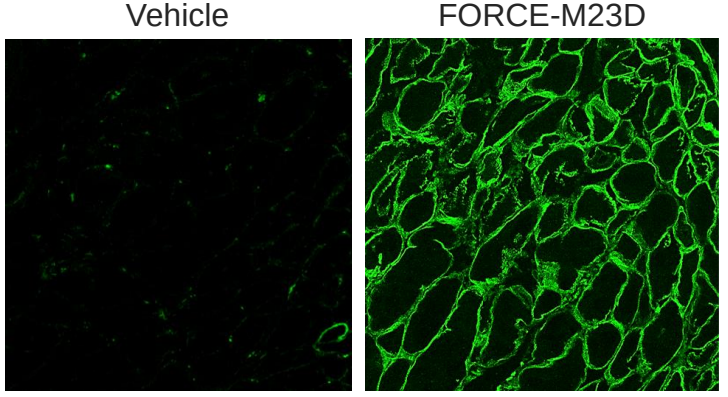
Heart



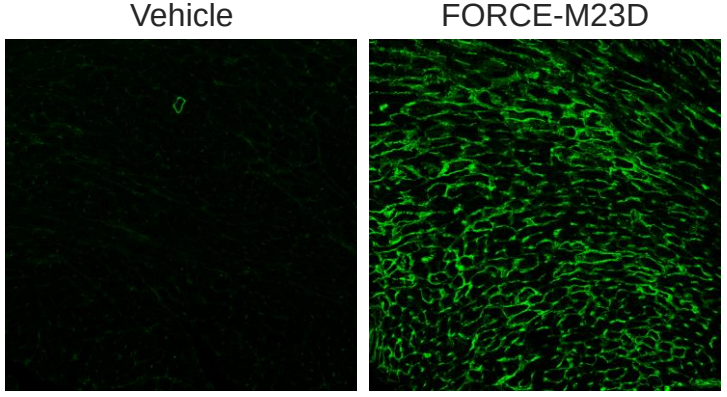
77% of wild-type dystrophin



68% dystrophin-positive fibers



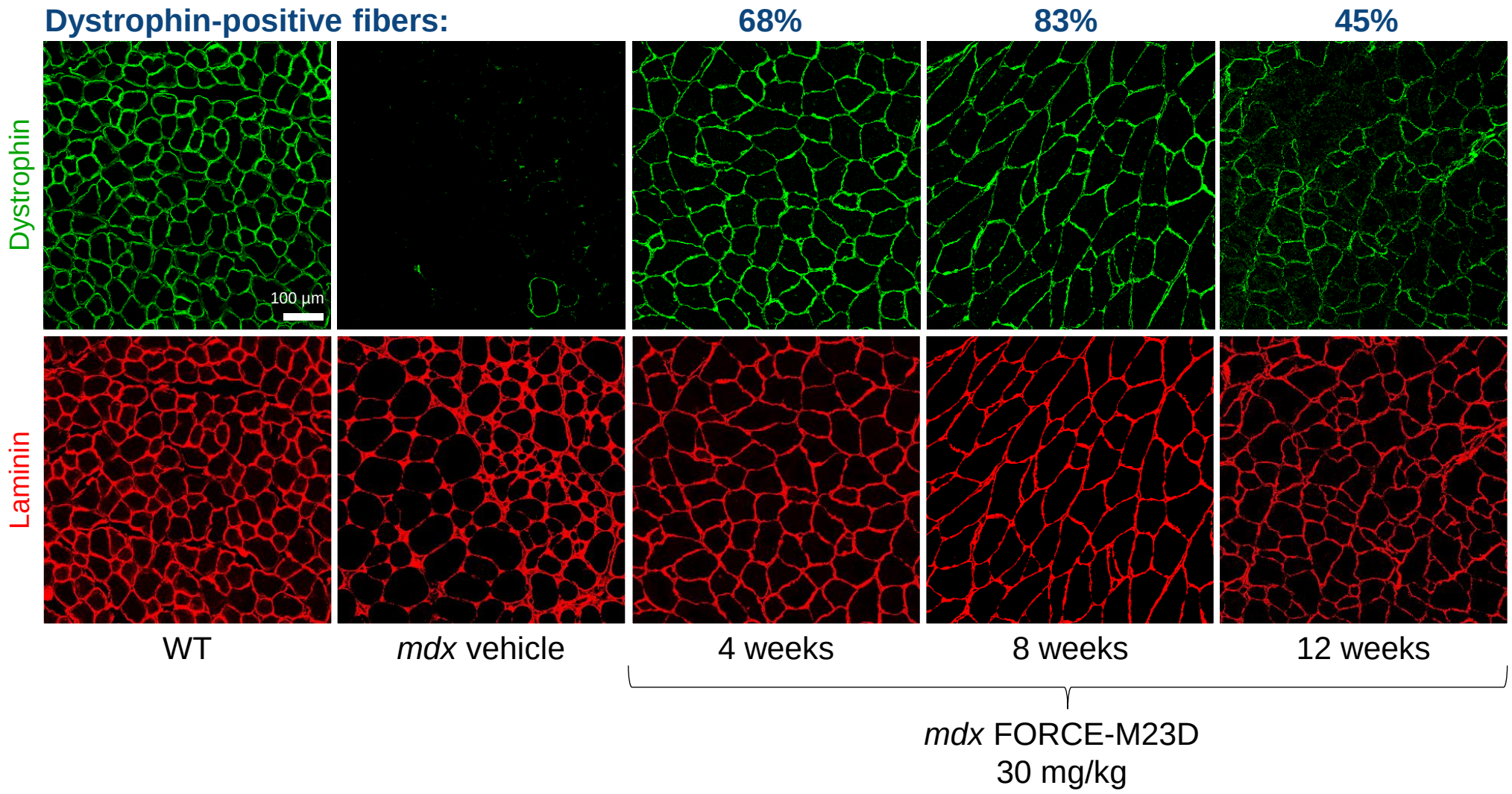
~80% dystrophin-positive fibers



~80% dystrophin-positive fibers

Note: Single IV 30 mg/kg dose of FORCE-M23D in *mdx* mouse model on day 0; analysis on week 4 for all muscles. N= 3 - 5 per cohort.

FORCE Achieved Durable Dystrophin Localization to Sarcolemma in Quadriceps



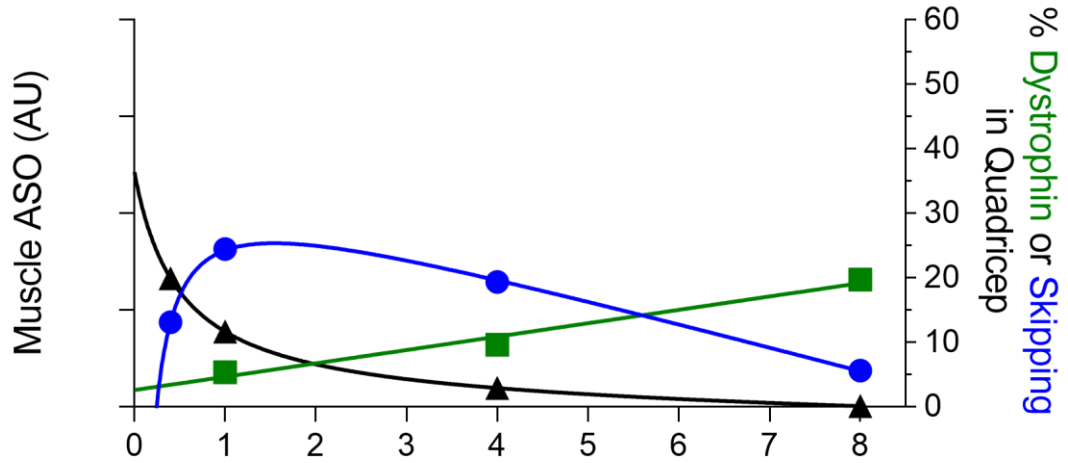
Note: Single IV 30 mg/kg dose of FORCE-M23D in *mdx* mouse model on day 0, analysis at specified time intervals.

FORCE's Distinctive Pharmacokinetic Profile Delivered Substantial and Durable Dystrophin Expression with a Single Dose

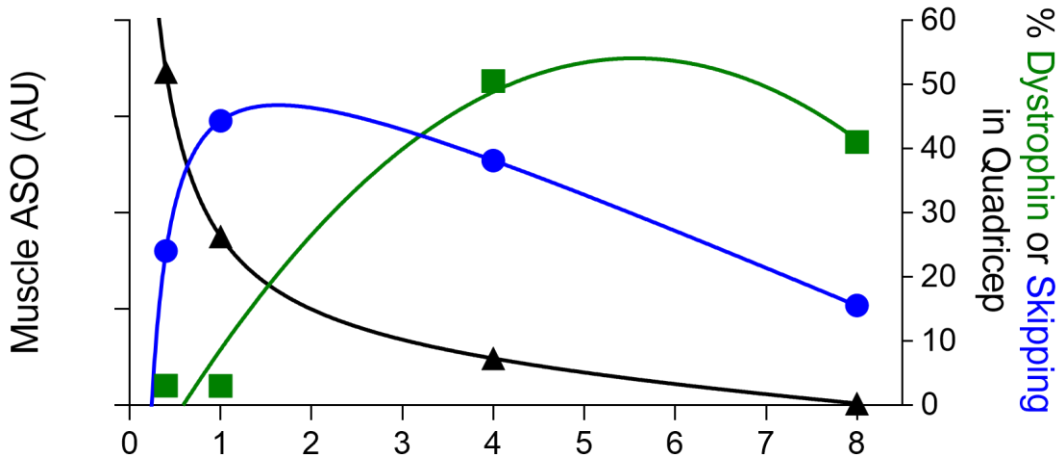
Pharmacokinetics | **Pharmacodynamics**

Muscle ASO concentration → Exon skipping → Dystrophin restoration

FORCE-M23D 10 mg/kg



FORCE-M23D 30 mg/kg

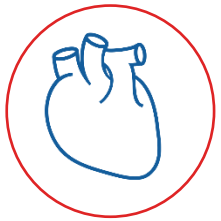


Muscle ASO | Skipping | Dystrophin

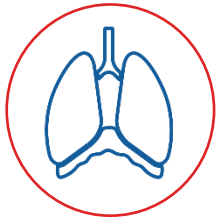
Note: Single IV dose of FORCE-M23D in *mdx* mouse model on day 0. N = 3 - 5 per cohort. ASO levels expressed as arbitrary units (AU).

DYNE-251 Demonstrated Robust Exon Skipping & Favorable Safety Profile in NHPs

High Level of Exon 51 Skipping Achieved in Key Muscles at 2 Months[1]



43% in heart



52% in diaphragm

18% in quadriceps

GLP Toxicology Studies: 5-Week & 13-Week[2]

- No dose limiting toxicity observed up to a maximally feasible dose
- No changes in cardiac, respiratory, neurologic or ophthalmic endpoints
- No effect on kidney function
- No effect on liver function
- No effect on coagulation
- NOAEL was identified at the highest dose tested

Notes: 1. Based on 5 weekly doses (Week 0 – Week 4) with tissue analysis at 8 weeks. 2. Based on conclusions of report from third-party CRO.
NOAEL: No-observed-adverse-effect level

DELIVER Trial Informed by Input from Multiple Stakeholders

Global, Multi-disciplinary KOL and Regulatory Input

- Overall design for the MAD portion in patients with DMD amenable to exon 51 skipping
- Patient population, biomarker and functional endpoints, and key safety considerations
- Natural history data to contextualize clinical trial data for patients and families, clinicians, payers, regulators

Global Advocacy Leaders, Patient and Caregiver Input

- Considerations for trial selection
- Clinical trial protocol and visit schedule
- Minimizing patient burden during trial conduct
- Ensuring support and education to patients and families

Phase 1/2 Clinical Trial to Evaluate DYNE-251 in Patients with DMD

Population

- Patients with DMD with mutations amenable to exon 51 skipping therapy
- Ages 4 to 16 years
- ~48 male participants
- Ambulant and non-ambulant

Primary Endpoints

- Safety and tolerability
- Change from baseline in dystrophin protein levels by Western Blot

Key Secondary Endpoints

- Pharmacokinetics
- Change from baseline of:
  - Exon 51 skipping levels
  - Muscle tissue PDPF
  - Multiple assessments of muscle function, including NSAA score and certain timed functional tests

Stages of DELIVER

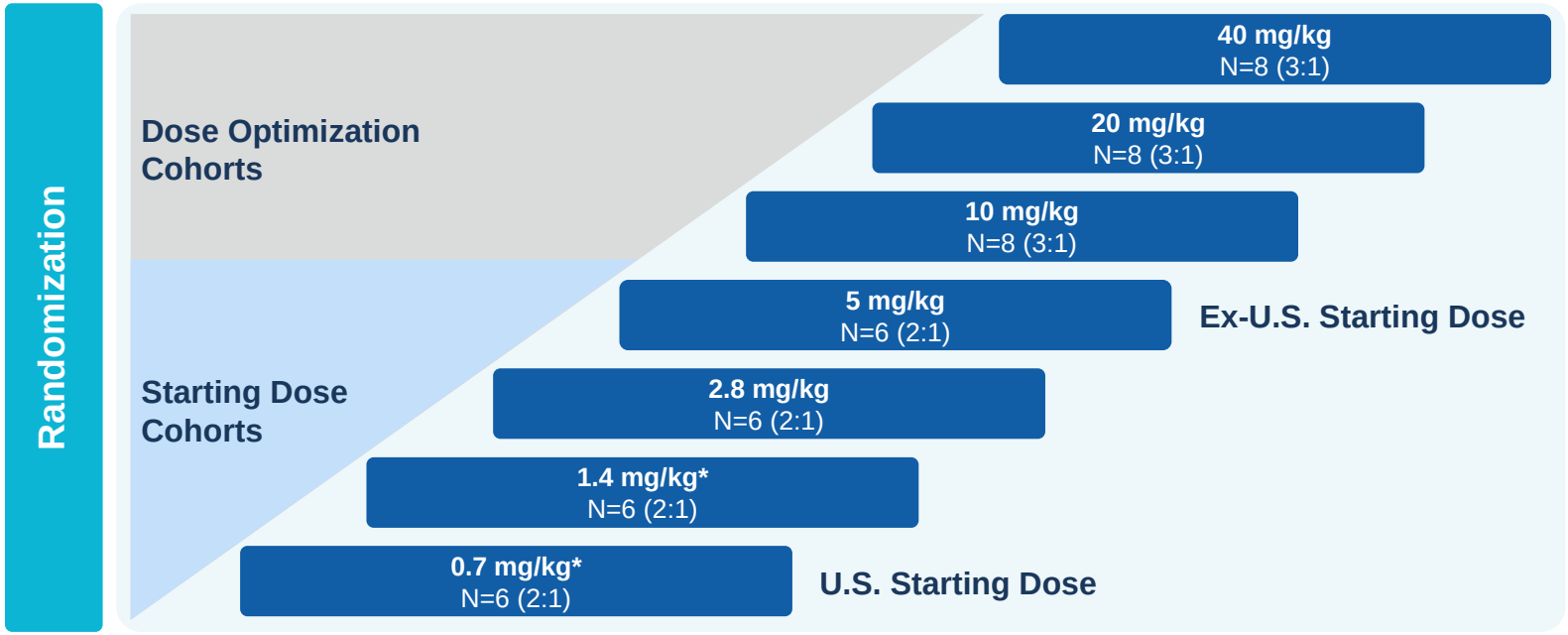
- Multiple Ascending Dose (MAD): 24 weeks
- Open-Label Extension (OLE): 24 weeks
- Long-Term Extension (LTE): 96 weeks

Initial Safety, Tolerability & Dystrophin Data Expected in H2 2023

Multiple Ascending Dose Stage of DELIVER

DELIVER

Global, Randomized, Placebo-Controlled Stage Evaluating Once Monthly Administration of DYNE-251 in ~48 Ambulant and Non-Ambulant Male DMD Patients with Mutations Amenable to Exon 51 Skipping Therapy



MAD Study Details

- IV administration of DYNE-251 or placebo every 4 weeks
- Muscle biopsies: Baseline and 24 weeks in 2.8 mg/kg and higher cohorts
- Patients in MAD study escalated to highest tolerable dose in OLE and LTE

Global Trial Designed to be Registrational and Enable Rapid Achievement of Predicted Pharmacologically Active Dose Levels

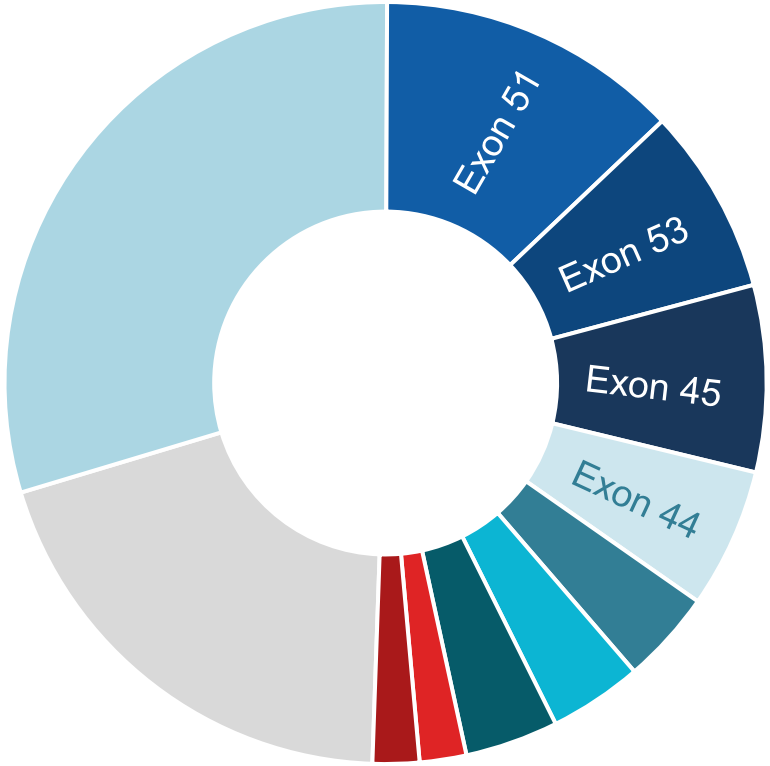
Patient cohorts will be dosed from 0.7 mg/kg to 40 mg/kg in the U.S. Outside the U.S., patient cohorts will be dosed from 5 mg/kg to 40 mg/kg. Doses provided refer to PMO component of DYNE-251.
* Muscle biopsies taken at baseline and 24 weeks in 2.8 mg/kg and higher cohorts; biopsies not taken in 0.7 mg/kg and 1.4 mg/kg cohorts.

Dyne is Committed to Developing Global DMD Franchise

Approximately

80% of patients

have genotypes amenable to exon skipping



- Exon 51 – **13%**
- Exon 53 – **8%**
- Exon 45 – **8%**
- Exon 44 – **6%**
- Exon 50 – **4%**
- Exon 52 – **4%**
- Exon 43 – **4%**
- Exon 55 – **2%**
- Exon 8 – **2%**
- Other exon skips – **~30%**
- May not be amenable to exon skipping – **~20%**

DMD Program Summary

Validating Data

mdx Model

- **Achieved robust and durable** exon skipping in skeletal and cardiac muscle
- **Dose-dependently increased dystrophin** expression up to 90% of WT based on western blot and ~80% dystrophin-positive fibers
- **Reduced serum CK levels**
- **Demonstrated functional benefit** in multiple standardized assessments

DYNE-251

- **Robust and dose-dependent exon skipping** in DMD patient myotubes (exon 51)
- **Transformative exon skipping in NHP** cardiac and skeletal muscles
- **Favorable safety profile in NHP GLP tox studies**

Potential Advantages

- **Established** clinical and regulatory path
- FDA granted **Fast Track designation for DYNE-251**
- **Opportunity to accelerate DMD franchise** expansion (exon 53, exon 45, exon 44) to reach additional patient populations

Safety, Tolerability & Dystrophin Data Expected in H2 2023

FSHD Program

Overview

- Aberrant expression of DUX4
- Onset in teen years or young adulthood
- Normal life expectancy

Clinical Presentation

- Progressive wasting and skeletal muscle loss
- Significant physical limitations

Population

- ~16,000 - 38,000 (US)
- ~35,000 (Europe)

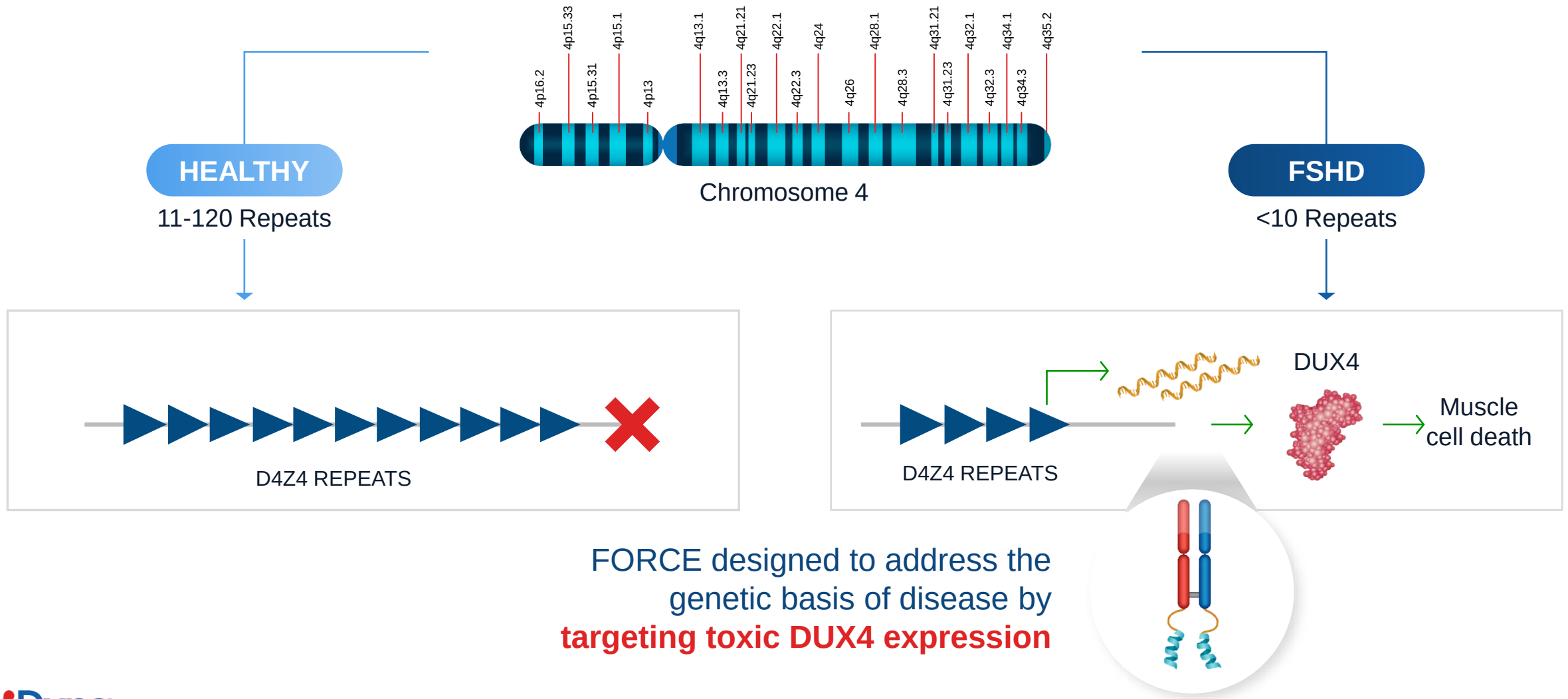
NO approved therapies

OUR APPROACH

Disease-Modifying DUX4 Knockdown

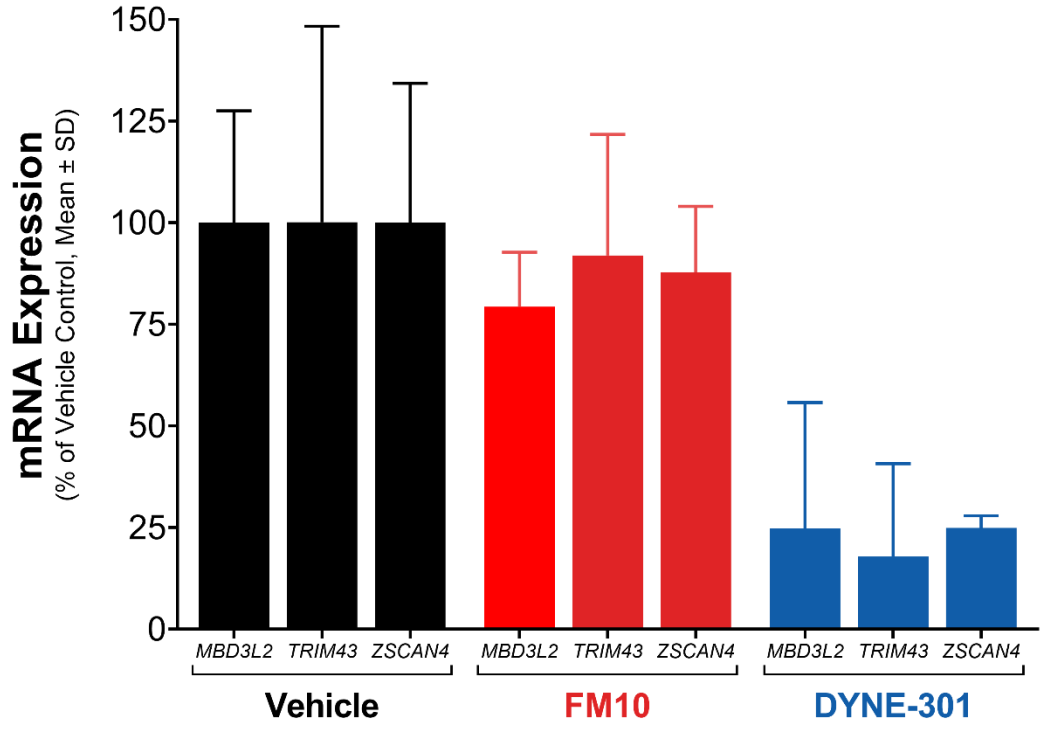
Targeting toxic *DUX4* mRNA expression to potentially **stop or reverse disease progression**

FORCE Targets the Genetic Basis of FSHD

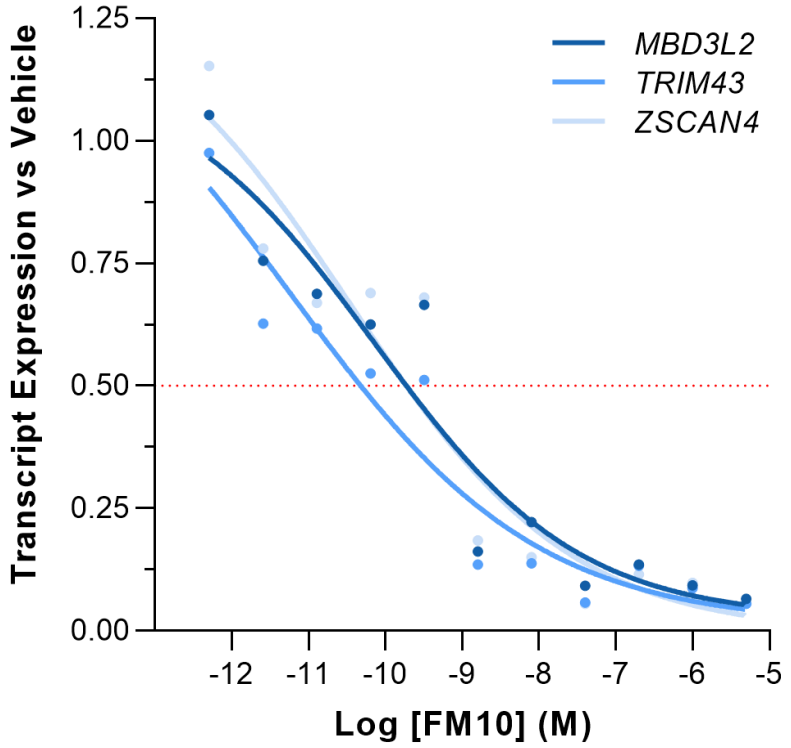


FORCE designed to address the genetic basis of disease by **targeting toxic DUX4 expression**

DYNE-301 Highly Potent and Suppressed Expression of Key DUX4 Biomarkers in FSHD Patient Myotubes



FSHD patient cells were treated with either 8 nM of naked FM10 PMO or FORCE-FM10 (DYNE-301)



DUX4 Transcriptome Marker	IC_{50}
MBD3L2	0.2 nM
TRIM43	0.05 nM
ZSCAN4	0.2 nM

FSHD Program Summary

Validating Data

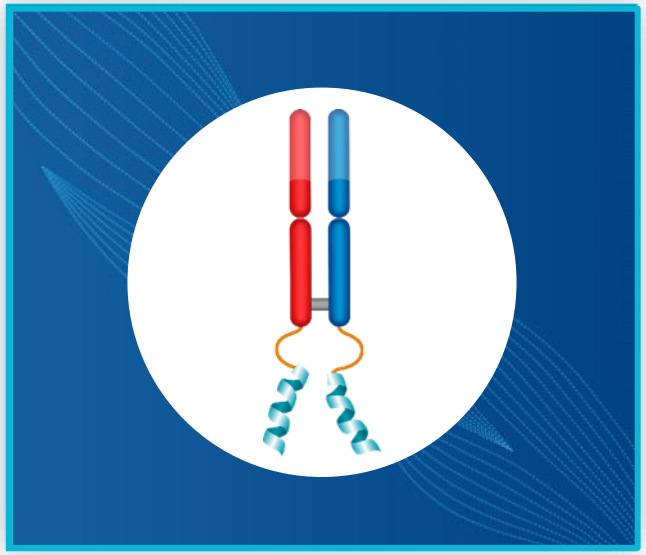
- **Reduced** toxic gain-of-function *DUX4* mRNA
- **Superior** suppression of DUX4 biomarkers observed over naked ASO
- **Enhanced** muscle distribution
- **Translatability** to primates expected; based on platform data in multiple NHP studies

Potential Advantages

- **Exclusive license** to potent *DUX4* targeting payloads
- **Tractable development** with rapid path to human PoC

Building the World's Leading Muscle Disease Company

Win in DM1, DMD, FSHD



Own Muscle Delivery & Leverage FORCE

Dynamo Culture