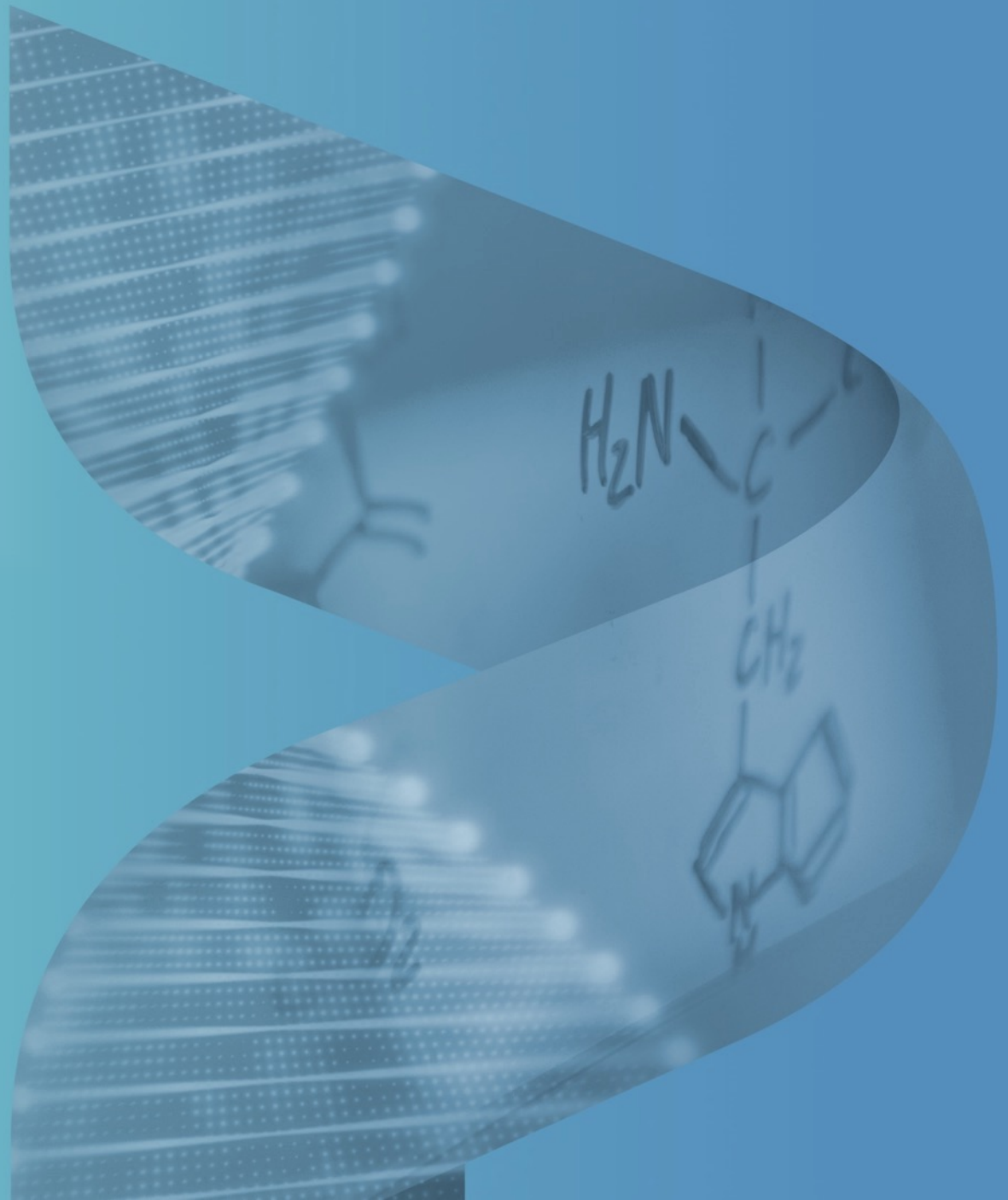
PepGen

EMPOWERING OLIGONUCLEOTIDE THERAPEUTICS

COMPANY PRESENTATION

APRIL 2023



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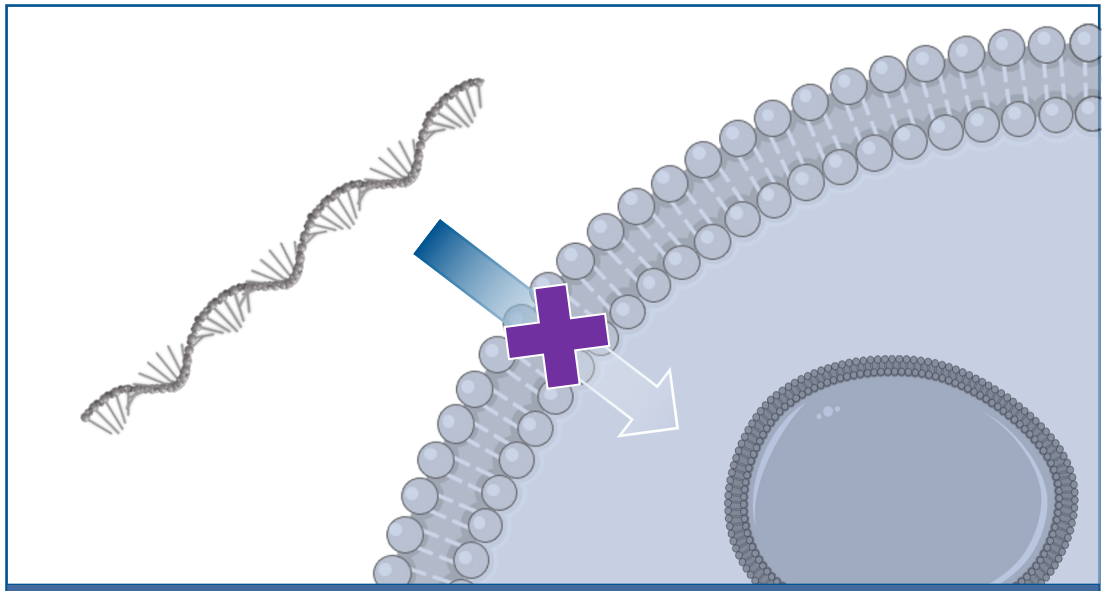
This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. These statements may be identified by words such as "aims," "anticipates," "believes," "could," "estimates," "expects," "forecasts," "goal," "intends," "may," "plans," "possible," "potential," "seeks," "will," and variations of these words or similar expressions that are intended to identify forward-looking statements. Any such statements in this press release that are not statements of historical fact may be deemed to be forward-looking statements. These forward-looking statements include, without limitation, statements about our clinical and preclinical programs, product candidates, including their planned development and therapeutic potential, plans for future development, preclinical studies and clinical trials in our programs, including the planned initiation of a Phase 2a MAD trial of PGN-EDO51 in DMD patients, achievement of milestones, and corporate and clinical/preclinical strategies.

Any forward-looking statements in this presentation are based on current expectations, estimates and projections only as of the date of this release and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward-looking statements. These risks and uncertainties include, but are not limited to that we may fail to successfully complete preclinical studies and clinical trials of our product candidates or to obtain regulatory approval for marketing of such products; initial preclinical study or clinical trial results for one or more of our product candidates may not be predictive of future trial results for such candidates; our product candidates may not be safe and effective; there may be delays in regulatory clearance or changes in regulatory framework that are out of our control; we may not be able to nominate new drug candidates within the estimated timeframes; our estimation of addressable markets of our product candidates may be inaccurate; we may need additional funding before the end of our expected cash runway and may fail to timely raise such additional required funding; more efficient competitors or more effective competing treatments may emerge; we may be involved in disputes surrounding the use of our intellectual property crucial to our success; we may not be able to attract and retain key employees and qualified personnel; earlier-stage trial results may not be predictive of later stage trial outcomes; and we are dependent on third parties for some or all aspects of our product manufacturing, research and preclinical and clinical testing. Additional risks concerning PepGen's programs and operations are described in its most recent annual report on Form 10-K and/or quarterly report on Form 10-Q on file with the SEC. PepGen explicitly disclaims any obligation to update any forward-looking statements except to the extent required by law.

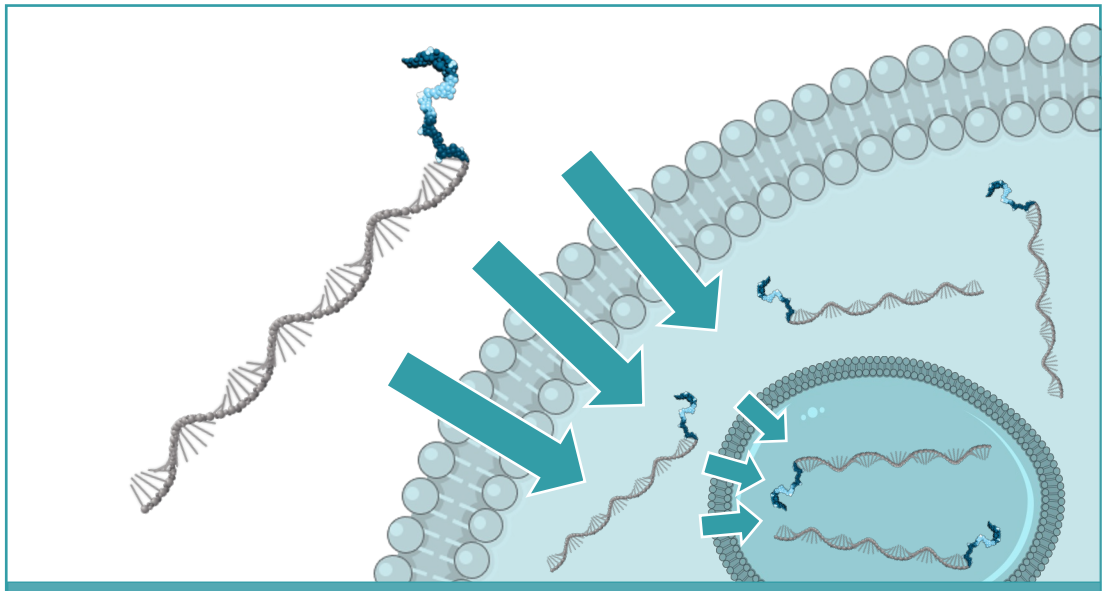
PEPGEN'S EDO TECHNOLOGY IS DESIGNED TO ADDRESS THE DELIVERY CHALLENGES THAT LIMIT OLIGONUCLEOTIDE THERAPEUTICS

THE CHALLENGE



Unconjugated oligonucleotides are **not readily distributed to muscle**, and are **not efficiently taken up into cells and the nucleus**

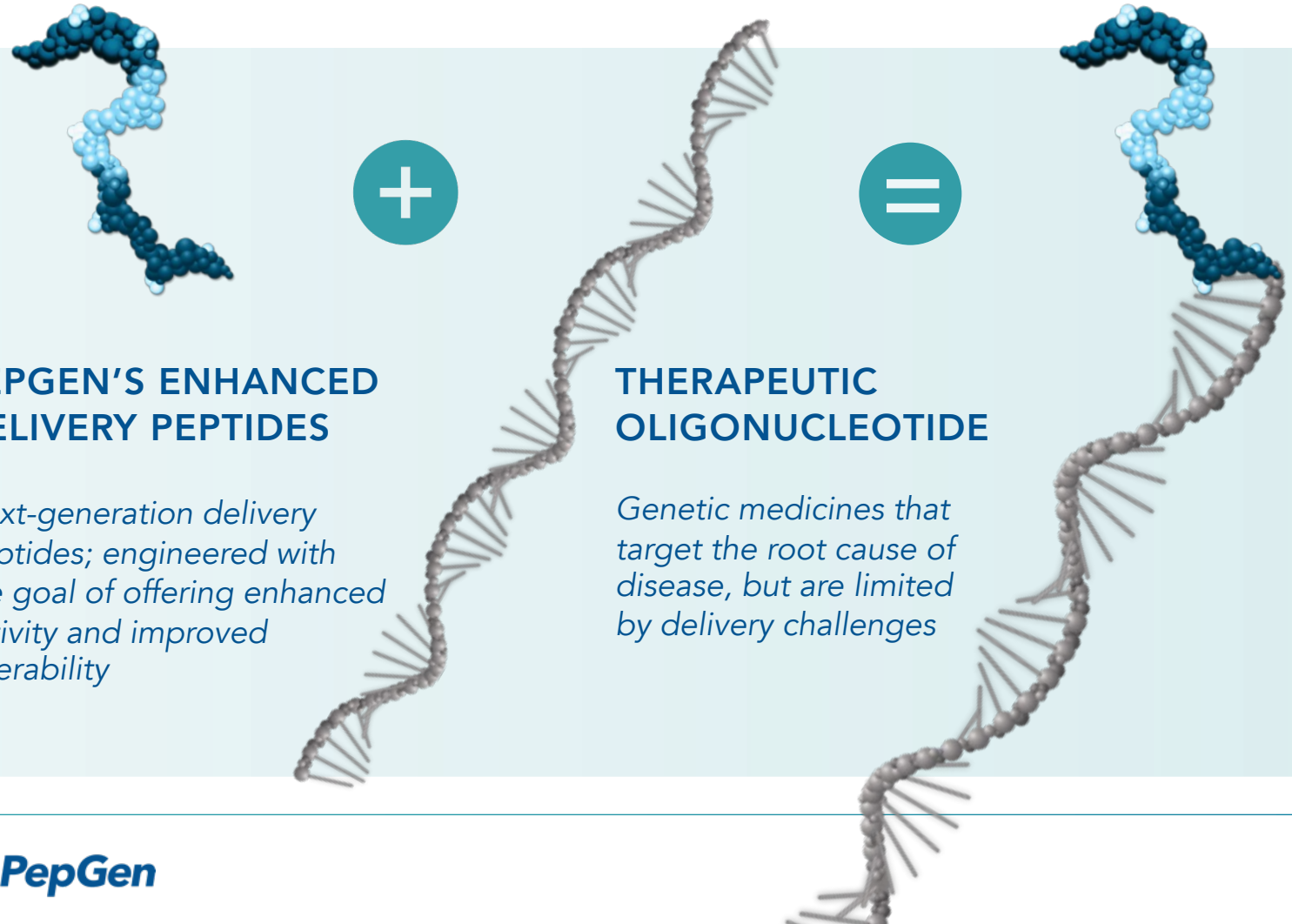
THE EDO SOLUTION



Our EDO platform is engineered to optimize the **tissue penetration, cellular uptake and nuclear delivery** of oligonucleotide therapeutics

THE POWER OF EDOs

Enhanced Delivery Oligonucleotides are well-characterized therapeutic PMO oligonucleotides conjugated to proprietary delivery-enhancing peptides



PEPGEN'S ENHANCED DELIVERY PEPTIDES

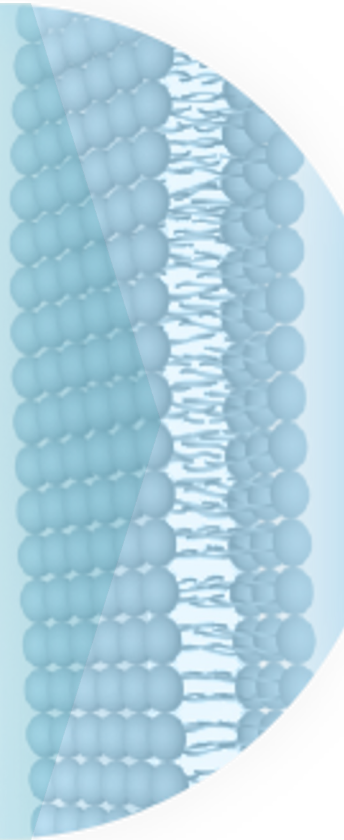
Next-generation delivery peptides; engineered with the goal of offering enhanced activity and improved tolerability

THERAPEUTIC OLIGONUCLEOTIDE

Genetic medicines that target the root cause of disease, but are limited by delivery challenges

ENHANCED DELIVERY OLIGONUCLEOTIDES

Efficient cellular uptake of oligos including in cardiac and skeletal tissue



A NEXT-GENERATION OLIGONUCLEOTIDE DELIVERY PLATFORM WITH THE POTENTIAL TO TRANSFORM PATIENT OUTCOMES

Empowering oligonucleotide therapeutics

Our **Enhanced Delivery Oligonucleotide (EDO)** platform is engineered to offer enhanced therapeutic activity and improved tolerability, with **greater skeletal, diaphragm and cardiac muscle penetrance**

PGN-EDO51 for DMD Exon 51

- PGN-EDO51 treatment resulted in the **highest levels of oligo delivery & exon 51 skipping in humans** following a single dose*
- **Highest level of exon 51 skipping in NHP** skeletal muscle at tolerable target dose levels, and **highest level of dystrophin production in *mdx*** mouse skeletal muscle**
- Generally **well-tolerated**
- **CONNECT1-EDO51 Ph2 patient MAD trial anticipated to open in 1H23, CONNECT2-EDO51 in 2H23*****

PGN-EDODM1 for DM1

- EDO technology delivered to human muscle levels of oligonucleotide which were **pharmacologically active** in DM1 mouse model
- **Foci reduction and liberation of MBNL1** observed in patient cells
- EDO-mediated **delivery of therapeutic oligonucleotides to the CNS** observed in NHP studies
- **FREEDOM-DM1 patient SAD trial anticipated to open in 1H23*****

A robust pipeline

- Lead assets target potentially **large, multi-$B market opportunity**
- Potential for EDO platform to address **50% of DMD exon skipping amenable patients**
- Broad NMD therapeutic portfolio

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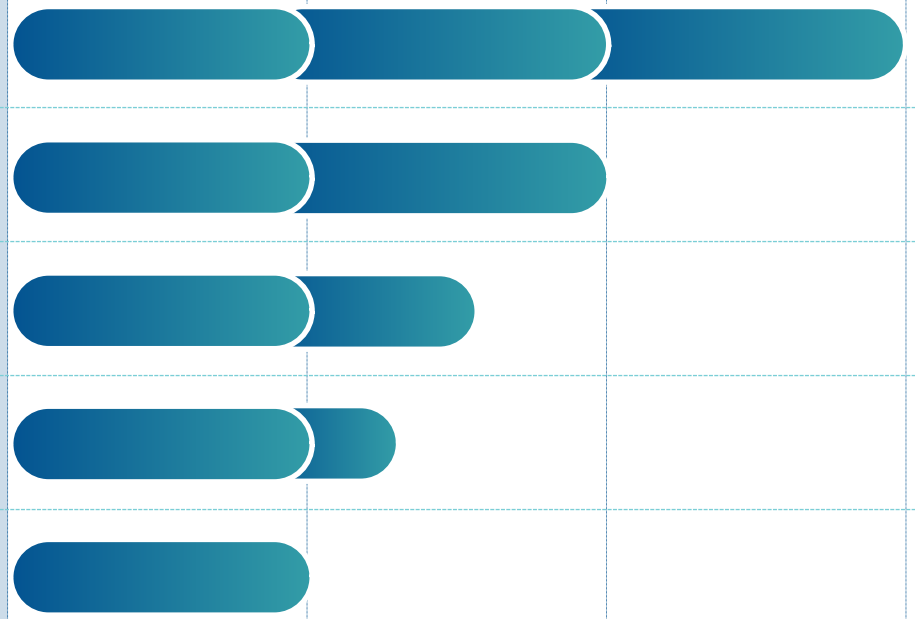
DMD = Duchenne muscular dystrophy; DM1 = myotonic dystrophy Type 1; NMD = neuromuscular disease.
* Comparative statements are based on cross-trial comparisons with publicly-available data for other exon skipping approaches that have been assessed following a single dose in humans, and following single and multiple doses in NHP. ** Of clinical-stage DMD therapies. *** Subject to approval from regulatory authorities.

SCALABLE EDO TECHNOLOGY DESIGNED TO ENABLE BROAD PORTFOLIO

PROGRAM	INDICATION *TARGET*	DISCOVERY	PRECLINICAL	PHASE 1	PHASE 2	REGISTRA-TIONAL*
PGN-EDO51	Duchenne muscular dystrophy *Exon 51*					
PGN-EDODM1	Myotonic dystrophy type 1 *DMPK*					
PGN-EDO53	Duchenne muscular dystrophy *Exon 53*					
PGN-EDO45	Duchenne muscular dystrophy *Exon 45*					
PGN-EDO44	Duchenne muscular dystrophy *Exon 44*					



FUTURE PIPELINE OPPORTUNITIES
Additional neuromuscular indications
Neurologic indications

*A registrational study is designed to generate data in order to support a regulatory application, subject to alignment with regulatory authorities.

WE BELIEVE THAT OUR DELIVERY PLATFORM HAS THE POWER TO UNLOCK THE THERAPEUTIC POTENTIAL OF OLIGONUCLEOTIDES

PGN-EDO51 – *PepGen's step change*

- Enhanced delivery to key muscle tissues
- **Highest levels of exon skipping in humans** following a single dose*
- **Potential for greater dystrophin production**

SRP-5051 (vesleteplirsen) – *Sarepta Therapeutics*

- Currently in Ph2b

EXONDYS 51® (eteplirsen) – *Sarepta Therapeutics*

- Approved 2016, 2022 sales: **$512M****

	EXON SKIPPING		DYSTROPHIN
	1 dose (HV)	*>3 doses (patients)*	*>3 doses (patients)*
15 mg/kg	**1.4%**	CONNECT1 study	
10 mg/kg	**1.1%**		
30 mg/kg	-	10.79%***	6.55%***
20 mg/kg	~0.18%***	2.57%***	3.06%***
30 mg/kg	<0.05%***	0.59%***	0.44%****

Dystrophin levels of **>10%** may drive **meaningful clinical benefit** for DMD patients

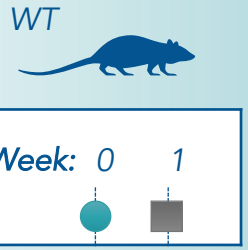
Uncertain benefit in 0 – 10% range

* Comparative statements are based on cross-trial comparisons with publicly-available data for other exon skipping approaches that have been assessed following a single dose. ** Source: Sarepta 2022 10K filing. **Source: Sarepta MOMENTUM study updates, 07Dec20 and 03May21. **** Clinical data included in drug label (FDA).

PGN-EDO51 FOR DUCHENNE MUSCULAR DYSTROPHY

EDO TECHNOLOGY INCREASES THE POTENCY OF EXON SKIPPING OLIGONUCLEOTIDES

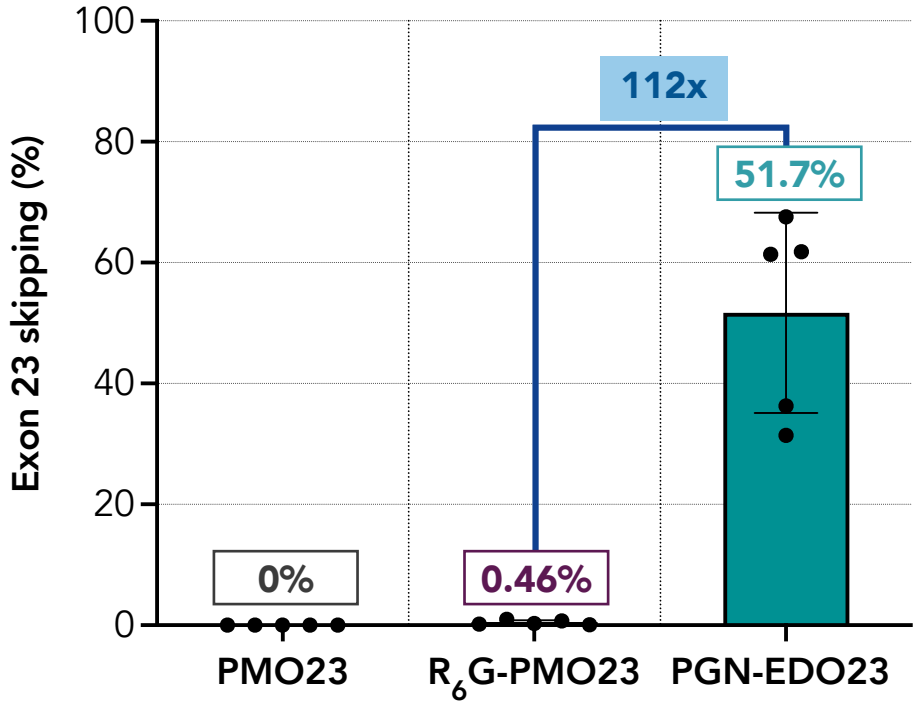
PGN-EDO23
(murine analogue of PGN-EDO51)



- PMO or PPMO dose
- Tissue analysis

EXON SKIPPING

Biceps, 10 mg/kg (qPCR)

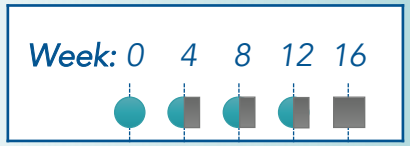


Protocol: PMO23, PGN-EDO23 or R_6G-PMO23 were administered a single intravenous (IV) 10 mg/kg dose to WT male mice; tissues collected 7 days after injection. Exon skipping was evaluated by qPCR. Graph plotted as mean ± SD, *n* = 5. R_6G-PMO23 is believed to be structurally equivalent to the peptide component of SRP-5051 conjugated to a murine exon 23 skipping oligonucleotide.

SIGNIFICANT INCREASE IN DYSTROPHIN OBSERVED WITH REPEAT DOSING

PGN-EDO23
(murine analogue of PGN-EDO51)

mdx

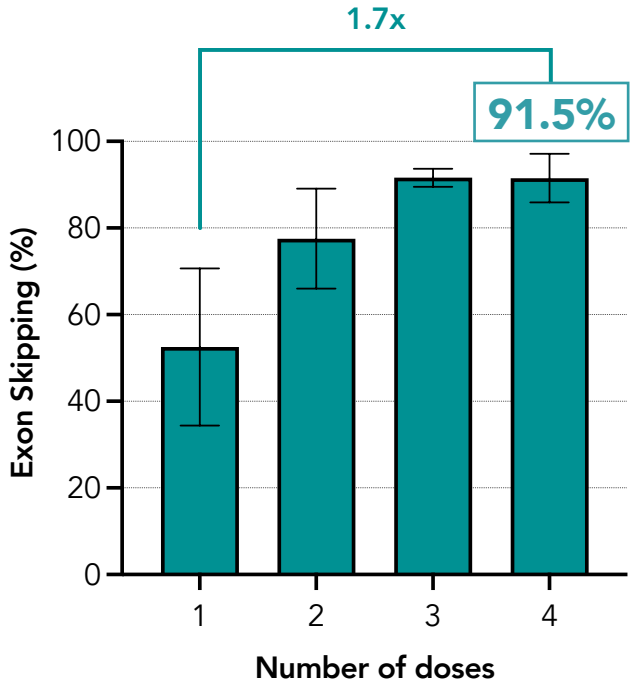


PGN-EDO23 dose

Tissue analysis

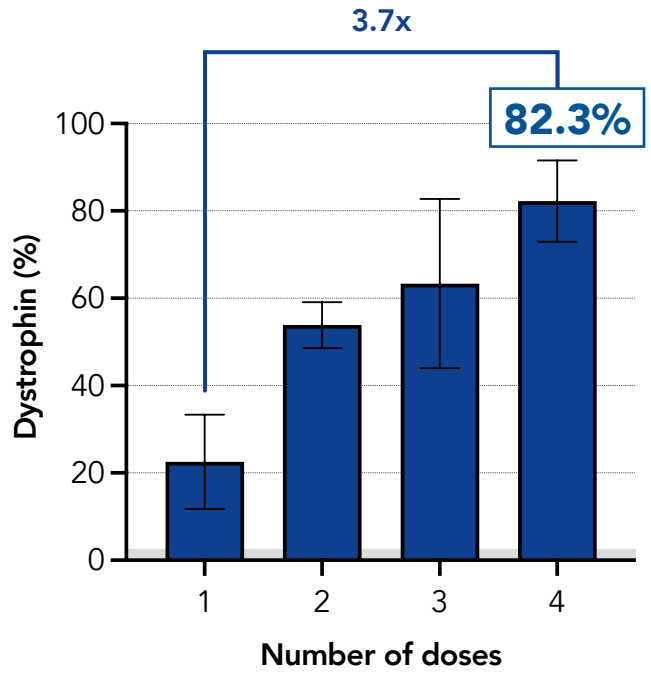
EXON SKIPPING

Biceps, 30 mg/kg, Q4W



DYSTROPHIN

Biceps, 30 mg/kg, Q4W



We believe these findings **support Q4W dosing in the clinic**

Protocol: *mdx* mice were dosed 2, 3 or 4 doses, with 4-week intervals between doses. Tissue samples were collected 4 weeks post-each dose at time points indicated. Exon skipping was performed by RT-PCR and dystrophin evaluation by western blot. Graph is presented as mean ± SD; *n* = 4-5 per cohort; grey band is dystrophin LLOQ (2.5%).

EDO TECHNOLOGY INCREASES THE POTENCY OF EXON SKIPPING OLIGONUCLEOTIDES

PGN-EDO51
R_6G-PMO51

PGN-EDO51

WT

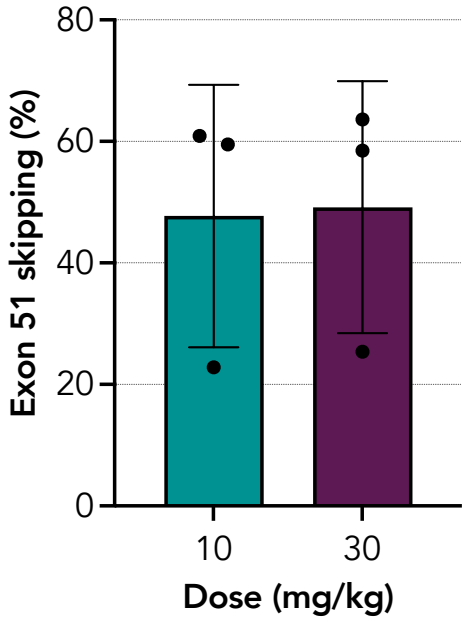
Week: 0 1 2 3 4 5

PPMO dose

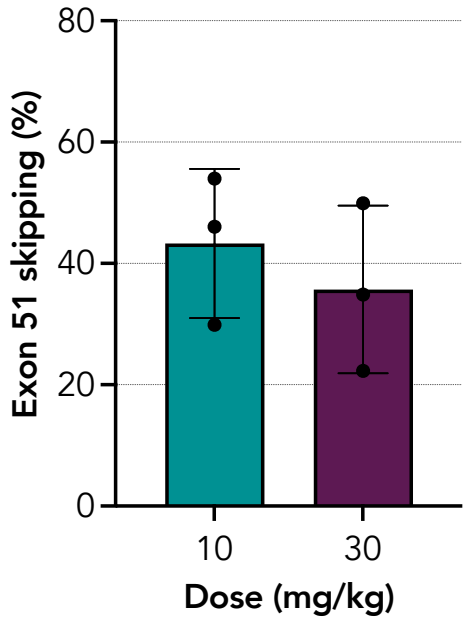
Tissue analysis

10 mg/kg of PGN-EDO51 has similar activity to 30 mg/kg of R_6G-PMO

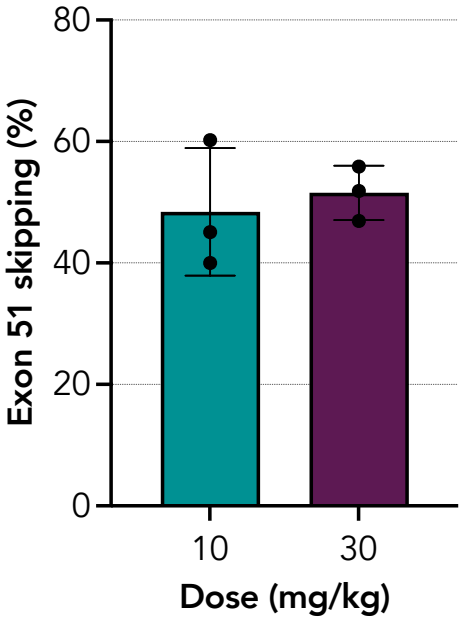
BICEPS



QUADRICEPS



DIAPHRAGM



Building on this data, we anticipate that:

- **10 mg/kg PGN-EDO51 is likely to be more active than 20 mg/kg R_6G-PMO**
- **15 mg/kg PGN-EDO51 is likely to be more active than 30 mg/kg R_6G-PMO**

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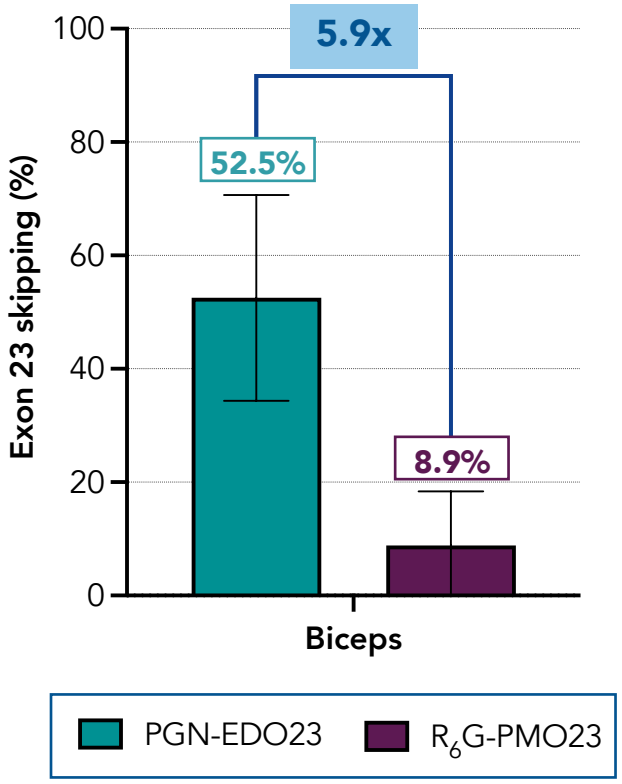
Protocol: PGN-EDO51 and R_6G-PMO were administered to NHP by IV infusion over 30 min at the doses indicated (n=3). Q2W, three doses administered, plus saline control. Tissues were harvested 7 days after final administration, exon skipping analyzed by RT-PCR. Shown as mean ± SD; n = 3 per group. Study was not powered for statistical significance. R_6G-PMO51 is believed to be structurally equivalent to SRP-5051.

CONSISTENT POTENCY OF EDO PLATFORM: MOUSE ➔ NHP ➔ HUMAN

MDX SINGLE DOSE

30 mg/kg, RT-PCR

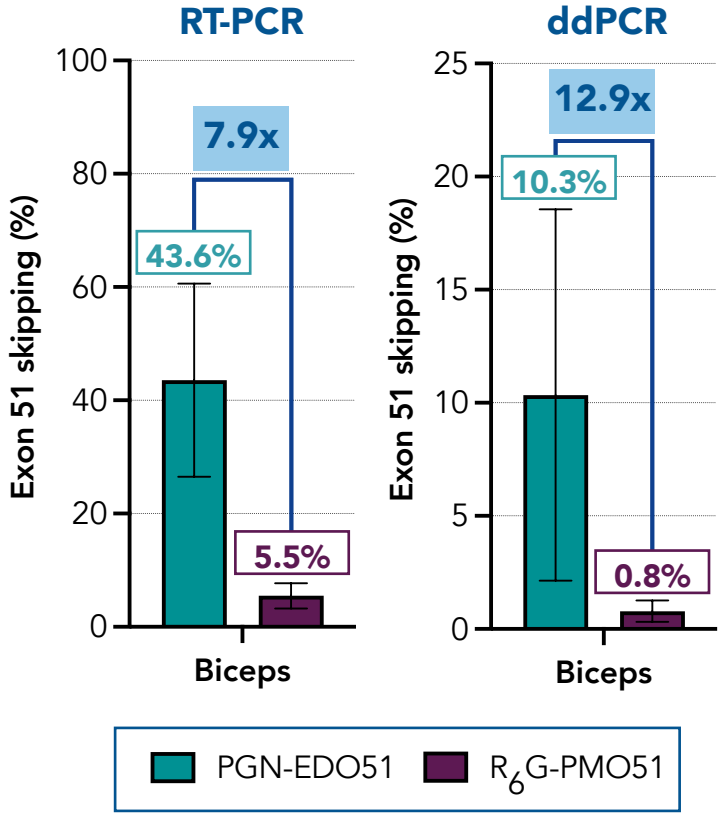
*Generally well-tolerated; no adverse kidney findings**



NHP SINGLE DOSE

30 mg/kg, RT-PCR & ddPCR

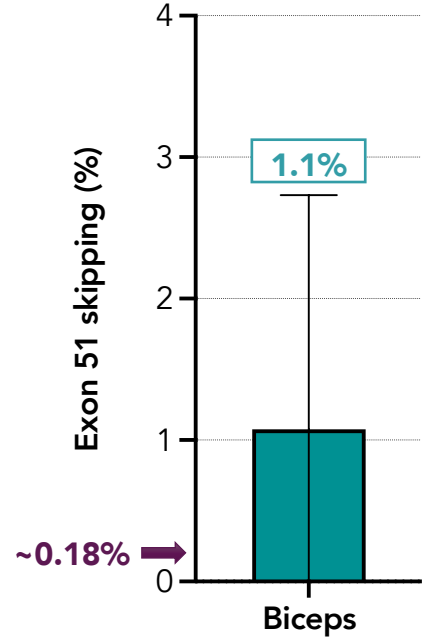
Generally well-tolerated; no adverse kidney findings



HV SINGLE DOSE

10 mg/kg, ddPCR

All AEs were mild & reversible



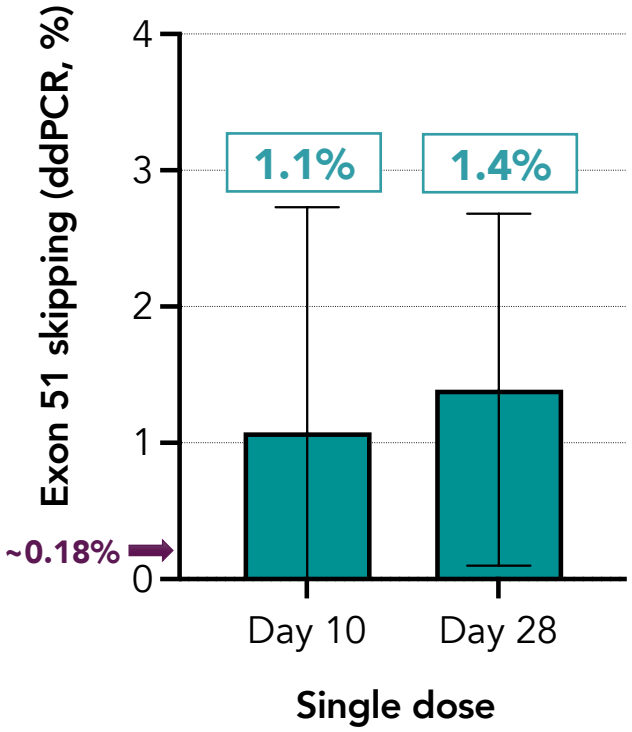
~6x higher than previously reported following a single dose

Mdx protocol: PGN-EDO23 and R_6G-PMO23 were administered to *mdx* mice via a single bolus i.v. injection at the dose indicated. Tissues were harvested after four weeks and exon skipping was analyzed via RT-PCR. Graph presented as mean ± SD; n = 4 per group. *Tolerability statement supported by single dose GLP study conducted in WT mice. HV protocol: see slide 25 (Day 10 data). SRP-5051 20 mg/kg HV data from Momentum update, 07Dec20 (comparative statements for human data are based on cross-trial comparisons). R_6G-PMO51 is believed to be structurally equivalent to SRP-5051, R_6G-PMO23 utilizes the murine exon 23 skipping oligonucleotide.

HIGHEST LEVELS OF EXON 51 SKIPPING IN HUMANS FOLLOWING SINGLE DOSE → INCREASED EXON SKIPPING IN NHP WITH Q4W REPEAT DOSING

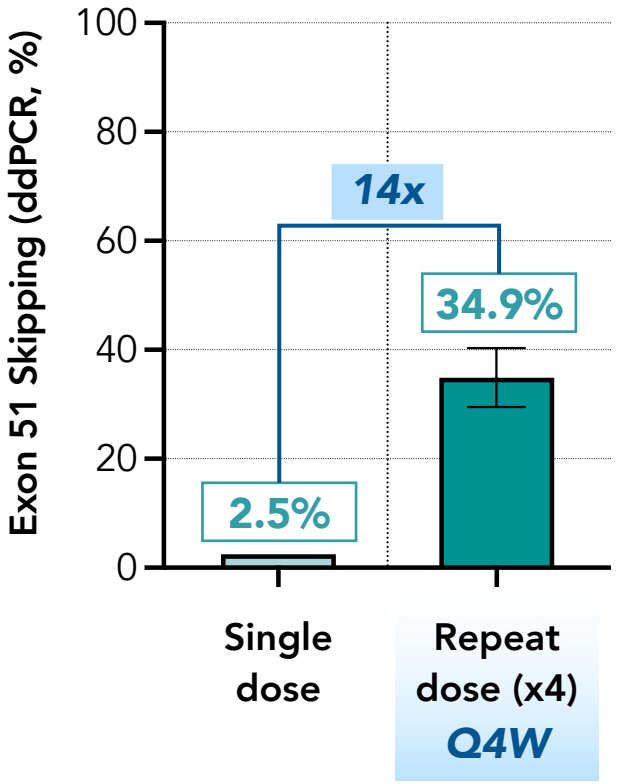
HV SINGLE DOSE

Biceps, 10 mg/kg, ddPCR



NHP SINGLE VS REPEAT

Biceps, 20 mg/kg, ddPCR



PATIENT REPEAT DOSE

Studies initiating in 2023

Ph2 MAD studies in DMD patients anticipated to utilize **Q4W dosing**

We believe this NHP data supports potential for **meaningful increases in exon skipping under this dosing regimen**

NHP protocol: Single (30 min) or repeat (60 min) IV doses with PGN-EDO51 were performed in male NHP. For repeat dose evaluation, NHP received 4 doses with 4-week intervals between doses. Tissue samples were collected 1-week post-final dose as indicated on graphs. Exon skipping was performed by ddPCR. Graph is presented as mean ± SD; *n* = 3-8 per group. HV protocol: see slide 25. SD = single dose, RD = repeat dose. * Comparative statements are based on cross-trial comparisons with publicly-available data for other exon skipping approaches that have been assessed following a single dose in humans. SRP-5051 data from Momentum update, 07Dec20.

TWO PH2 MAD STUDIES SUPPORT CLINICAL DATA READOUT ANTICIPATED IN 2024 AND POTENTIAL ACCELERATED APPROVAL

CONNECT1-EDO51

Ph2 open-label MAD study in patients *(planned initiation 1H23)*

- Initial dystrophin, exon skipping and safety data anticipated in 2024

Connect 1
EDO51

CONNECT2-EDO51

Ph2 randomized, double-blind, placebo-controlled MAD study in patients *(planned initiation 2H23)*

- Potential to support accelerated approval

Connect 2
EDO51

Dystrophin, exon skipping and safety data anticipated in 2024

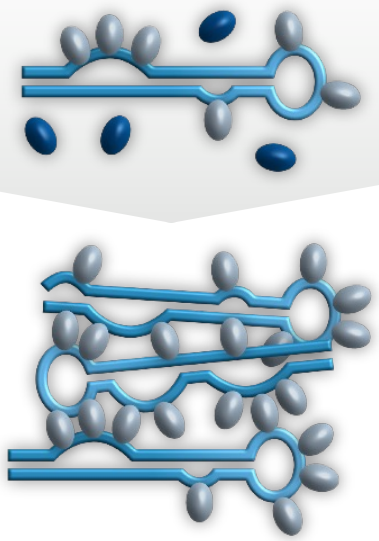
- Preclinical data suggests **Q4W repeat dosing** has the potential to drive **meaningful clinical benefit in individuals with DMD**
- Studies to be conducted in parallel
- Designed to provide **potential path to accelerated approval**

PGN-EDODM1 FOR MYOTONIC DYSTROPHY TYPE 1 (DM1)

OLIGO-BASED THERAPEUTIC MODALITIES FOR DM1 ARE FOCUSED ON TWO DISTINCT MECHANISTIC APPROACHES

DM1 PATHOLOGY

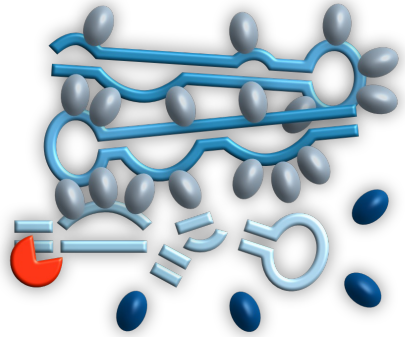
DMPK transcript CUG repeat hairpin loops bind MBNL1 and form cross-linked foci



Expanding toxic foci trap more MBNL1

DMPK KNOCKDOWN

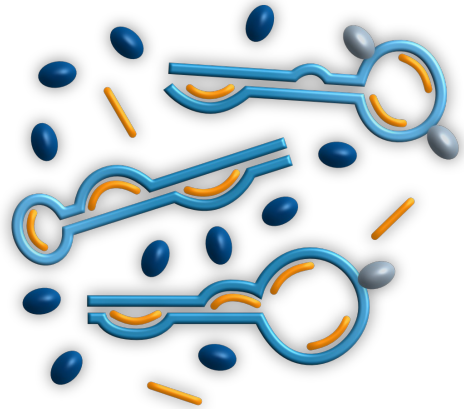
ASO / siRNA degrade *DMPK* transcript to reduce toxic foci



- Treatment results in non-specific degradation of *DMPK* transcript; potential risk of haploinsufficiency
- Correlation between level of knockdown and level of splicing correction is uncertain

DMPK COMPETITION

PGN-EDODM1 binds *DMPK* transcript, reducing toxic foci



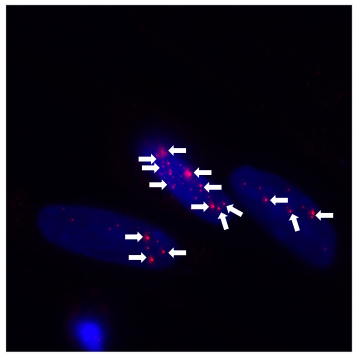
- Binding of PGN-EDODM1 liberates MBNL1, restoring physiological splicing
- *DMPK* transcript retained; role in cellular processes uninterrupted

denotes free (active) MBNL1, denotes bound (inactive) MBNL1, denotes PGN-EDODM1, denotes knockdown approach.
Other approaches also in development, including gene and RNA editing, and treatment of downstream pathologies.

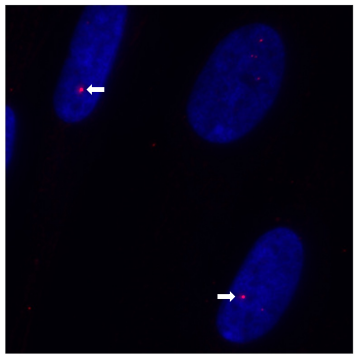
PGN-EDODM1 REDUCED PATHOGENIC NUCLEAR FOCI, LIBERATED MBNL1 AND CORRECTED MIS-SPLICING IN PATIENT CELLS

FOCI REDUCTION

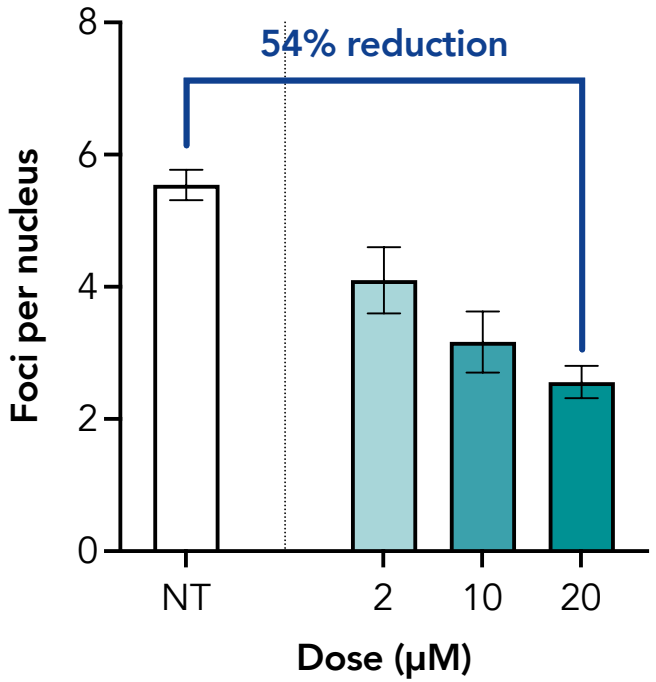
Not treated (NT)



PGN-EDODM1

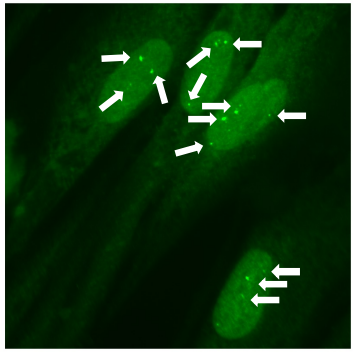


Foci quantification

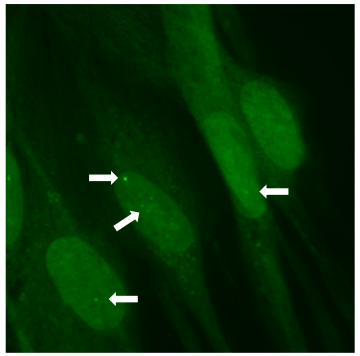


MBNL1 LIBERATION

Not treated (NT)

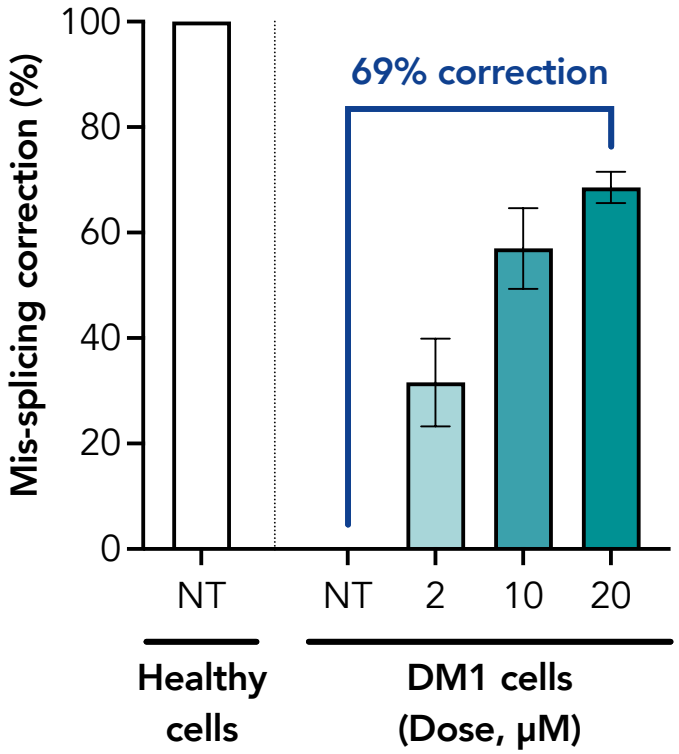


PGN-EDODM1



SPLICING CORRECTION

Across multiple transcripts



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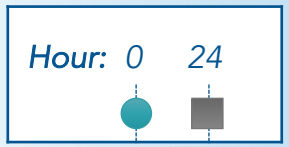
Immortalized myoblasts from healthy individual or DM1 patient with 2600 CTG repeats were cultured then differentiated for 4 days into myotubes. Treatment with PMO or peptide-PMO conjugates at concentrations given. Cells were harvested for analysis 24h after treatment. RNA isolation, RT-PCR and capillary electrophoresis (QIAxcel) analysis was performed. Visualisation with FISH and immunofluorescence microscopy. Mean ± SD; n = 5 per group.

OUR STERIC BLOCKING MECHANISM OF ACTION WAS NOT OBSERVED TO TARGET *DMPK* FOR DEGRADATION

DMPK TRANSCRIPT LEVELS

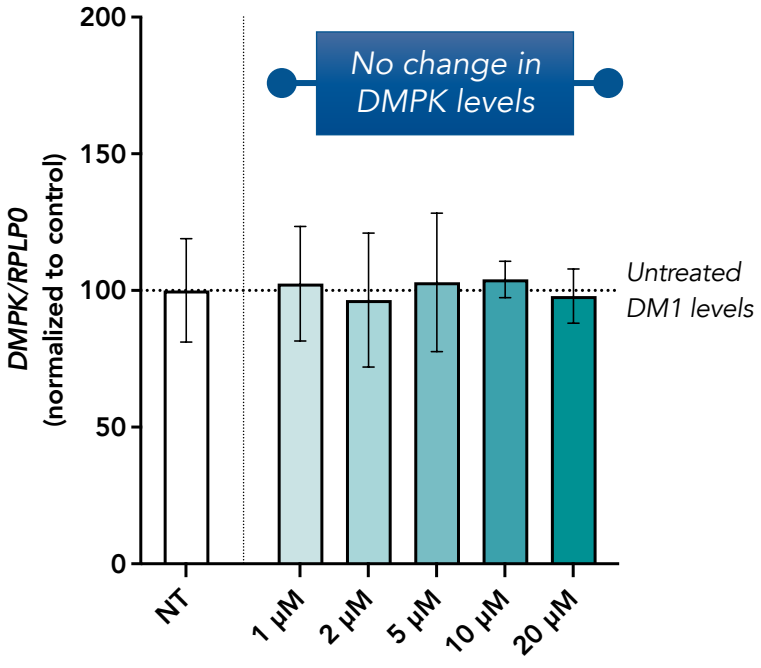
PGN-EDODM1

DM1 patient cells
(2,600 CTG repeats)



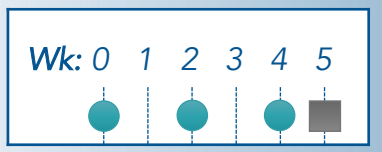
PGN-EDODM1 dose

Analysis



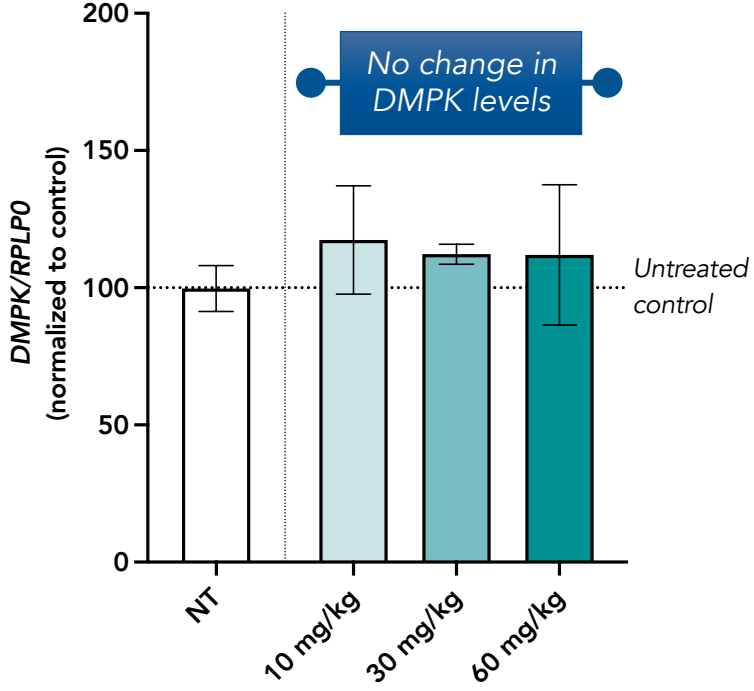
PGN-EDODM1

WT



PGN-EDODM1 dose

Tissue analysis



DMPK transcript levels remained unchanged across multiple preclinical models

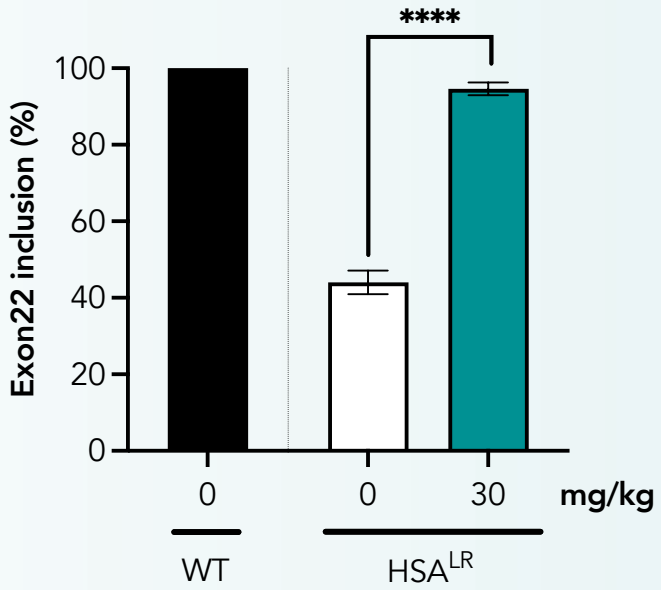
In vitro: Immortalized myoblasts from DM1 patient with 2600 CTG repeats were differentiated for 4 days to myotubes and treated for 24h hours with PGN-EDODM1 from 1-20 µM. NT = not treated. *DMPK* transcript levels were evaluated by qPCR and normalised to *RPLP0*. Graphs plotted as mean ± SD, *n*=3-4. NHP: PGN-EDODM1 was administered to NHP at the doses and regimen indicated. *DMPK* transcript levels were evaluated by RT-PCR and normalised to *RPLP0*. Graphs plotted as mean ± SD, *n*=4. NT = not treated.

PGN-EDODM1 ACHIEVED >68% CORRECTION OF MIS-SPLICING AND COMPLETE REVERSAL OF MYOTONIA AT 30 MG/KG IN HSA^{LR} MICE

CORRECTION OF MIS-SPLICING

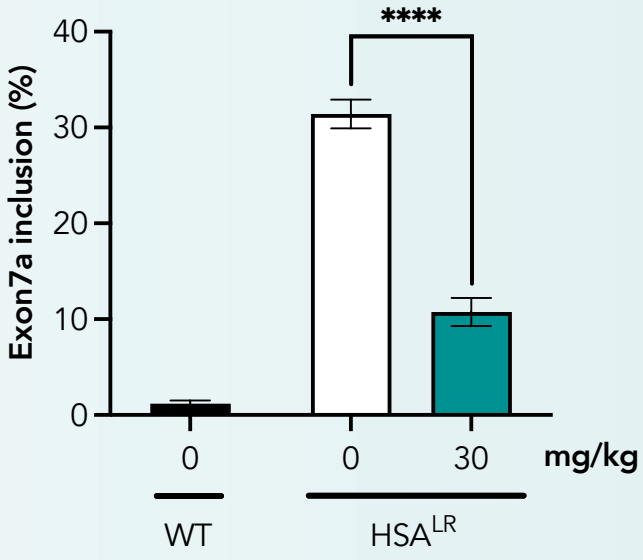
Atp2a1

(encodes SERCA1 – intracellular Ca^{+} pump involved in muscle excitation)



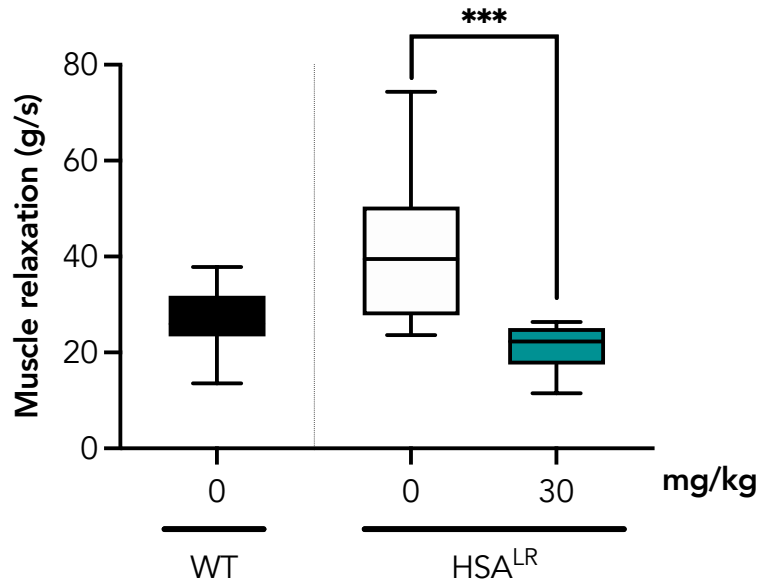
Clcn1

(encodes voltage-dependent Cl^{-} channel involved in muscle excitation)



REVERSAL OF MYOTONIA

Rate of muscle relaxation



91% correction of Atp2a1 splicing

68% correction of Clcn1 splicing

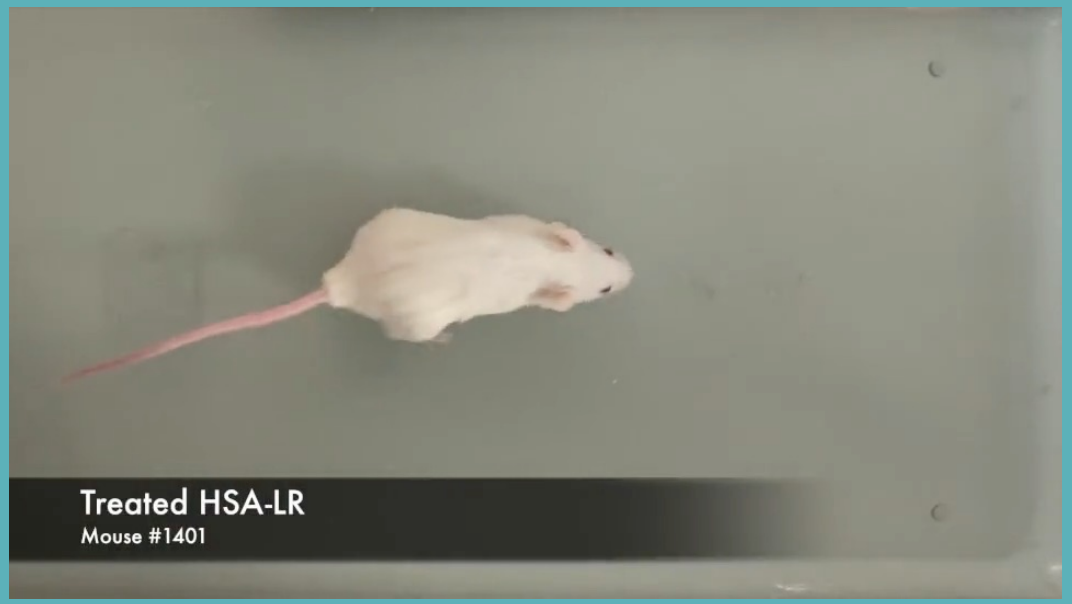
Complete correction of myotonia observed after single dose

Protocol: PGN-EDODM1 was administered IV to HSA^{LR} mice at 30 mg/kg (n=8) against a saline control (n=16) and wild-type (WT) saline control (n=8). Myotonia assessed, tissues harvested 2 weeks post-administration. Mis-splicing data is quadriceps. Mean ± SEM or min to max. **** = p≤0.0001; *** = p≤0.001. Mis-splicing correction of Mbnl1 was also assessed.

SPLICING CORRECTION TRANSLATED TO PHENOTYPIC IMPROVEMENT OF DM1 MICE TREATED WITH PGN-EDODM1

UNTREATED HSA^{LR}

TREATED HSA^{LR}

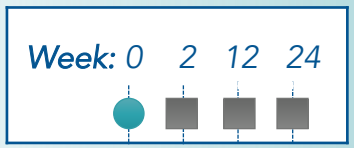


Protocol: PGN-EDODM1 was administered intravenously (IV) to WT and HSA^{LR} mice at 50 mg/kg (n=4-16); myotonia assessed two weeks post-administration.

SINGLE DOSE TREATMENT OF PGN-EDODM1 LED TO DURABLE IMPROVEMENTS IN SPLICING THROUGH 24 WEEKS

CORRECTION OF MIS-SPLICING

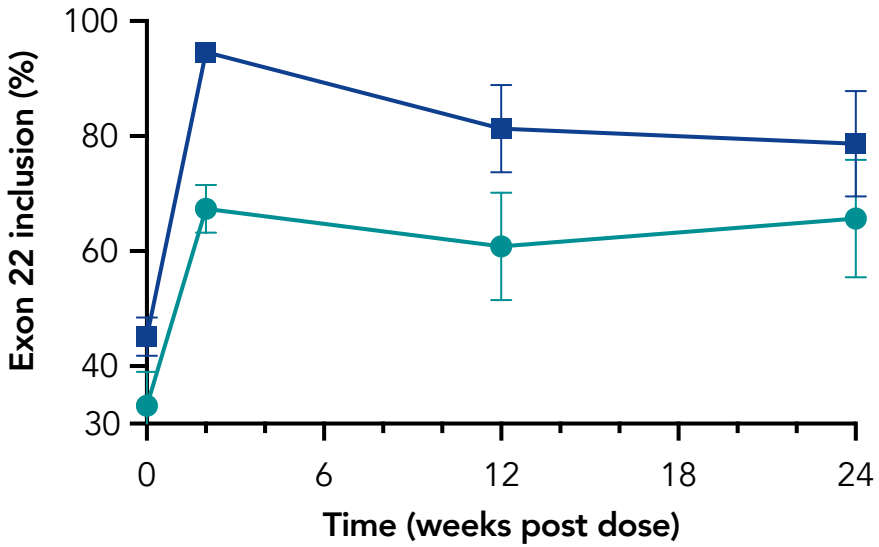
PGN-EDODM1



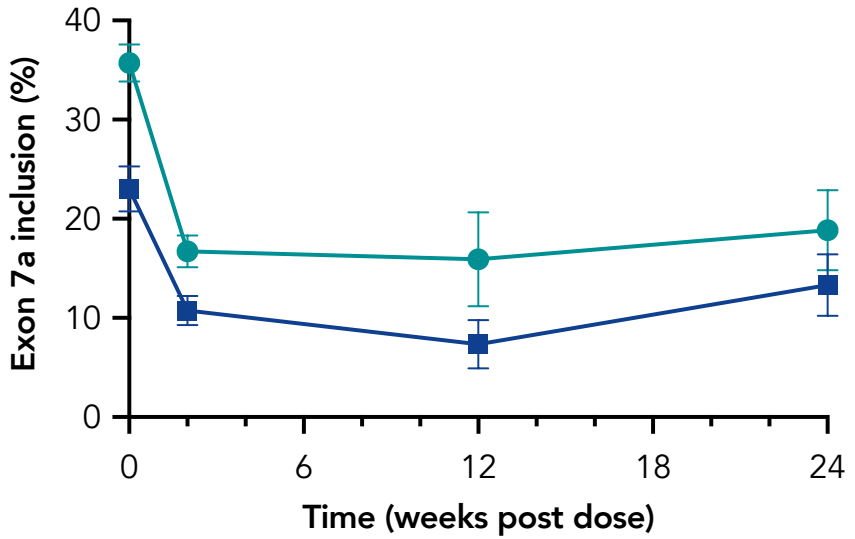
PGN-EDODM1 dose

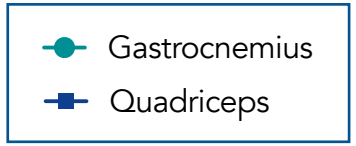
Tissue analysis

Atp2a1



Clcn1





Protocol: PGN-EDODM1 was administered intravenously (IV) to WT and HSA^{LR} mice at 30 mg/kg; gastrocnemius muscle harvested 2 (n=8), 12 (n=8) or 24 (n=5) weeks post-administration; graph plotted as mean ±SEM; n = 7 for 0 timepoint, 8 for 2- and 12-week timepoints; 5 for 24-week timepoint.

FREEDOM-DM1 PH1 STUDY ANTICIPATED TO OPEN IN 1H23, WITH PATIENT DATA IN 2024

FREEDOM-DM1: PHASE 1
Single ascending dose

- To be conducted in DM1 patients
- Randomized, double-blind, placebo-controlled trial
- Key anticipated readouts: **functional assessments, correction of mis-splicing, safety data**

Freedom
DM1

Planned to open in 1H23; **data anticipated in 2024**

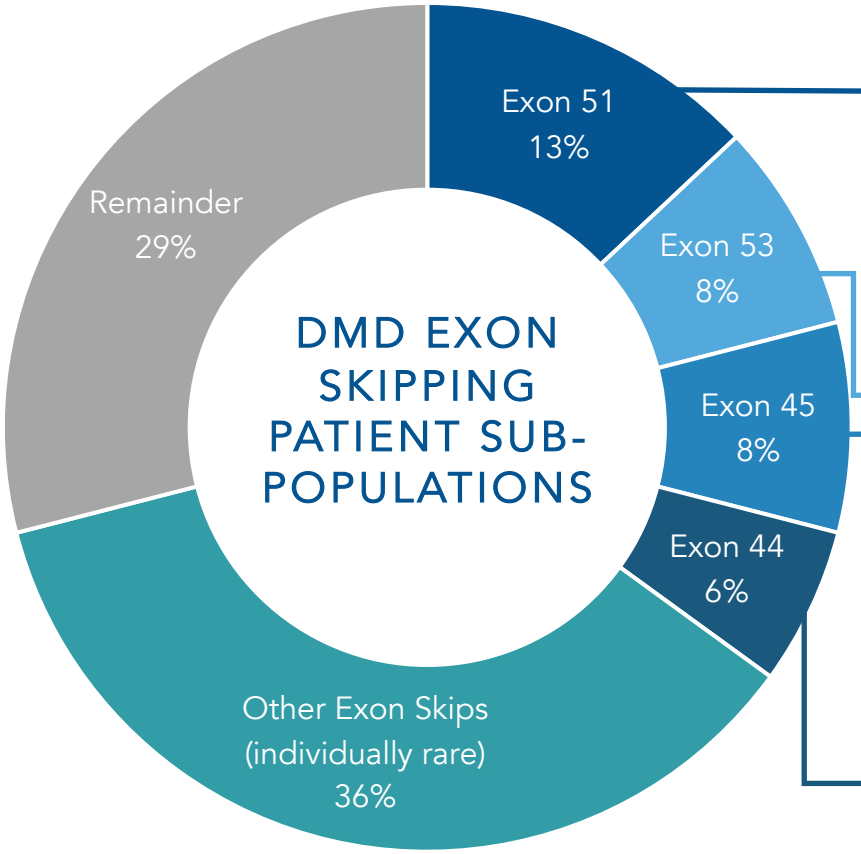
PHASE 2
Multiple ascending dose

- Informed by Ph1 study
- To be conducted in DM1 patients
- Randomized, double-blind, placebo-controlled trial
- Key anticipated readouts: **functional assessments, correction of mis-splicing, safety data**

Planned to open in 2024; designed to potentially support **regulatory approvals**

EDO PIPELINE

PEPGEN'S LEAD PROGRAM TARGETS LARGEST EXON SKIPPING PATIENT POPULATION IN DMD



PepGen's clinical program

Exon 51: PGN-EDO51

- *Highest levels of exon skipping observed in humans following a single dose**

PepGen's preclinical programs

Exon 53: PGN-EDO53

- *High levels of exon skipping observed in NHP*

Exon 45: PGN-EDO45

- *High levels of exon skipping observed in human cells*

Exon 44: PGN-EDO44

- *High levels of exon skipping observed in human cells*

PepGen

Source: https://www.cureduchenne.org/cure/exon-skipping/. * Comparative statements are based on cross-trial comparisons with publicly-available data for other exon skipping approaches that have been assessed following a single dose in humans.

SINGLE-DOSE EXON SKIPPING LEVELS FOR PGN-EDO53 ALMOST 7X HIGHER THAN FOR R_6G-PMO53 COMPARATOR

PGN-EDO53
R_6G-PMO53

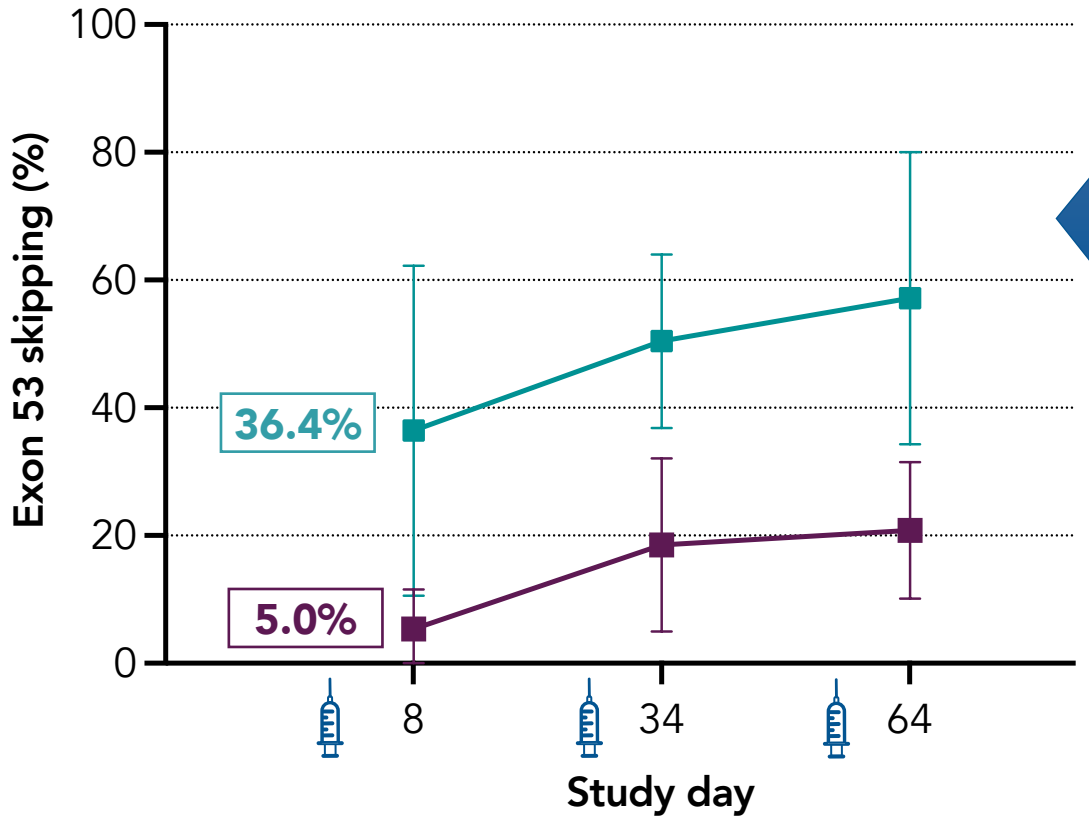
PGN-EDO53

WT

Day: 1 8 29 34 57 64

PPMO dose (Q4W)
Tissue analysis

BICEPS *(30 mg/kg)*



PGN-EDO53 candidate nominated for development **outperformed R_6G-PMO53** comparator after single and repeat doses

Protocol: PGN-EDO53 and R_6G-PMO53 were administered to NHP by IV infusion over 60 min (n=3). Q4W, three doses administered, PBS control. Biopsies taken 5 - 7 days after first and second administration; terminal samples collected 7 days after final dose. Study not powered for statistical significance. Data shown as mean ± SD; n = 3 per group. R_6G-PMO53 was selected as a relevant comparator PPMO approach.

ANTICIPATE INITIATING THREE PATIENT CLINICAL TRIALS IN 2023, WITH CLINICAL READOUTS EXPECTED IN 2024

PGN-EDO51
DMD Exon 51

CONNECT1-EDO51: Ph2 open-label MAD study in patients (planned initiation 1H23)
- Initial dystrophin, exon skipping and safety data anticipated in 2024

Connect 1
EDO51

CONNECT2-EDO51: Ph2 randomized, double-blind, placebo-controlled MAD study in patients (planned initiation 2H23)
- Potential to support accelerated approval

Connect 2
EDO51

PGN-EDODM1
DM1

FREEDOM-DM1: Ph1 randomized, double-blind, placebo-controlled SAD study in patients (planned initiation 1H23)
- Initial clinical function, correction of mis-splicing and safety data anticipated in 2024

Freedom
DM1

Clinical plans are subject to alignment with regulatory authorities

CONCLUSION

THE FUTURE OF PEPGEN

		2023	2024
PGN-EDO51 *DMD Exon 51*	**Highest level of single-dose exon skipping & oligo delivery in humans***	➢ **1H:** Initiation of **CONNECT1-EDO51** (Canada Ph2 MAD) ➢ **2H:** Initiation of **CONNECT2-EDO51** (global Ph2 MAD)	➢ **Dystrophin, exon skipping** and safety data in DMD patients
PGN-EDODM1 *DM1*	**Differentiated approach with robust preclinical dataset**	➢ **1H:** Initiation of **FREEDOM-DM1** (Ph1 SAD)	➢ **Functional assessments, correction of mis-splicing** and safety data in DM1 patients ➢ Initiation of Ph2 patient MAD
Pipeline	• Five neuromuscular disease candidates in pipeline • Work underway to **leverage EDO platform** to expand to new tissues and new indications		

PepGen

* Comparative statements are based on cross-trial comparisons with publicly-available data for other exon skipping approaches that have been assessed following a single dose.
Clinical plans are subject to alignment with regulatory authorities.