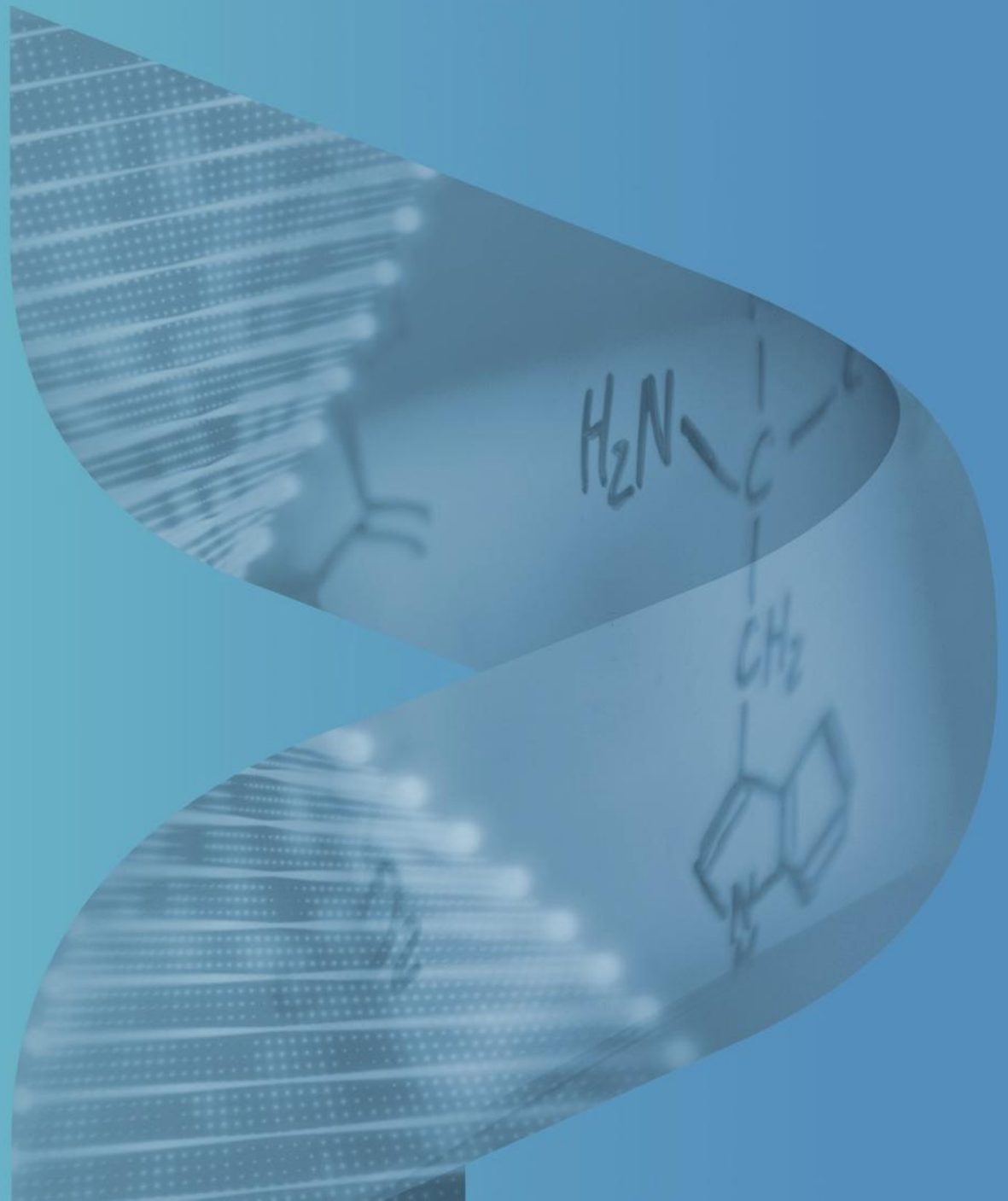
PepGen™

Company Presentation

December 2024

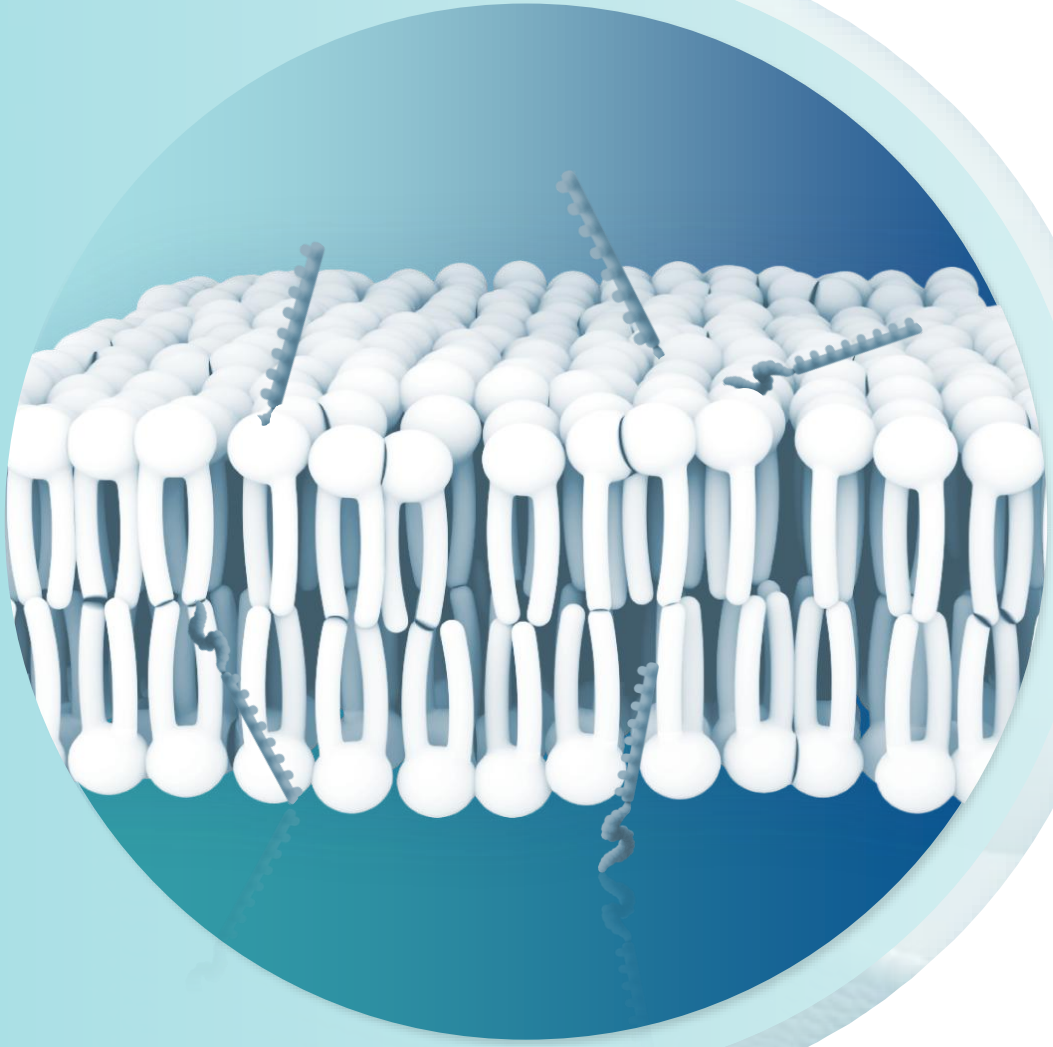


Forward-Looking Statements

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 presentation that are not statements of historical fact may be deemed to be forward-looking statements. These forward-looking statements include, without limitation, statements regarding the potential of our EDO platform to deliver higher levels of oligonucleotide to the nuclei and to dramatically improve the lives of people living with severe neuromuscular and neurological diseases, the therapeutic potential and safety profile of our product candidates, including based on early data, PGN-EDO51 and PGN-EDODM1, the design, initiation and conduct of clinical trials, including expected timelines for our CONNECT1-EDO51 and CONNECT2-EDO51 Phase 2 trials, and our FREEDOM1-DM1 Phase 1 and FREEDOM2-DM1 Phase 2 trials, the expected timing for additional results from our CONNECT1 Phase 2 trial and results from our FREEDOM Phase 1 trial, ongoing and planned regulatory interactions, the evaluation of PGN-EDO53 in IND/CTA enabling studies, and our financial resources and cash runway.

Any forward-looking statements in this presentation are based on current expectations, estimates and projections only as of the date of this presentation 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: delays or failure to successfully initiate or complete our ongoing and planned development activities for our product candidates, including PGN-EDO51, PGN-EDODM1 and PGN-EDO53; our ability to enroll patients in our clinical trials, including CONNECT1, CONNECT2, FREEDOM and FREEDOM2; that our interpretation of clinical and preclinical study results may be incorrect, or that we may not observe the levels of therapeutic activity in clinical testing that we anticipate based on prior clinical or preclinical results, including for PGN-EDO51 and PGN-EDODM1; our product candidates, including PGN-EDO51 and PGN-EDODM1, may not be safe and effective or otherwise demonstrate safety and efficacy in our clinical trials; adverse outcomes from our regulatory interactions, including delays in regulatory review, clearance to proceed or approval by regulatory authorities with respect to our programs, including clearance to commence planned clinical studies of our product candidates, or other regulatory feedback requiring modifications to our development programs, including in each case with respect to our including CONNECT1, CONNECT2, FREEDOM and FREEDOM2 clinical trials; changes in regulatory framework that are out of our control; our ability to obtain, maintain and protect our intellectual property; our ability to enforce our patents against infringers and defend our patent portfolio against challenges from third parties; competition from others developing therapies for the indications we are pursuing; unexpected increases in the expenses associated with our development activities or other events that adversely impact our financial resources and cash runway; and our dependence 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 our most recent annual report on Form 10-K and quarterly report on Form 10-Q that are filed with the SEC. PepGen explicitly disclaims any obligation to update any forward-looking statements except to the extent required by law.

This presentation discusses PGN-EDO51 and PGN-EDODM1, investigational therapies that have not been approved for use in any country and is not intended to convey conclusions about their efficacy or safety. There is no guarantee that PGN-EDO51, PGN-EDODM1 or any other investigational therapy will successfully complete clinical development or gain regulatory authority approval.



Driven by our proprietary Enhanced Delivery Oligonucleotide (EDO) platform, PepGen is creating a pipeline of disease-modifying therapeutics with the potential to safely and effectively target the underlying cause of serious genetic neuromuscular and neurological diseases

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PepGen's Pipeline Enabled by EDO Technology

INVESTIGATIONAL CANDIDATES	CLINICAL PROGRAMS	INDICATIONS	PRECLINICAL	PHASE 1	PHASE 2	PIVOTAL
PGN-EDO51	Connect	DMD – *Exon 51*				
PGN-EDODM1	Freedom	DM1 – *DMPK*				
PGN-EDO53		DMD – *Exon 53*				

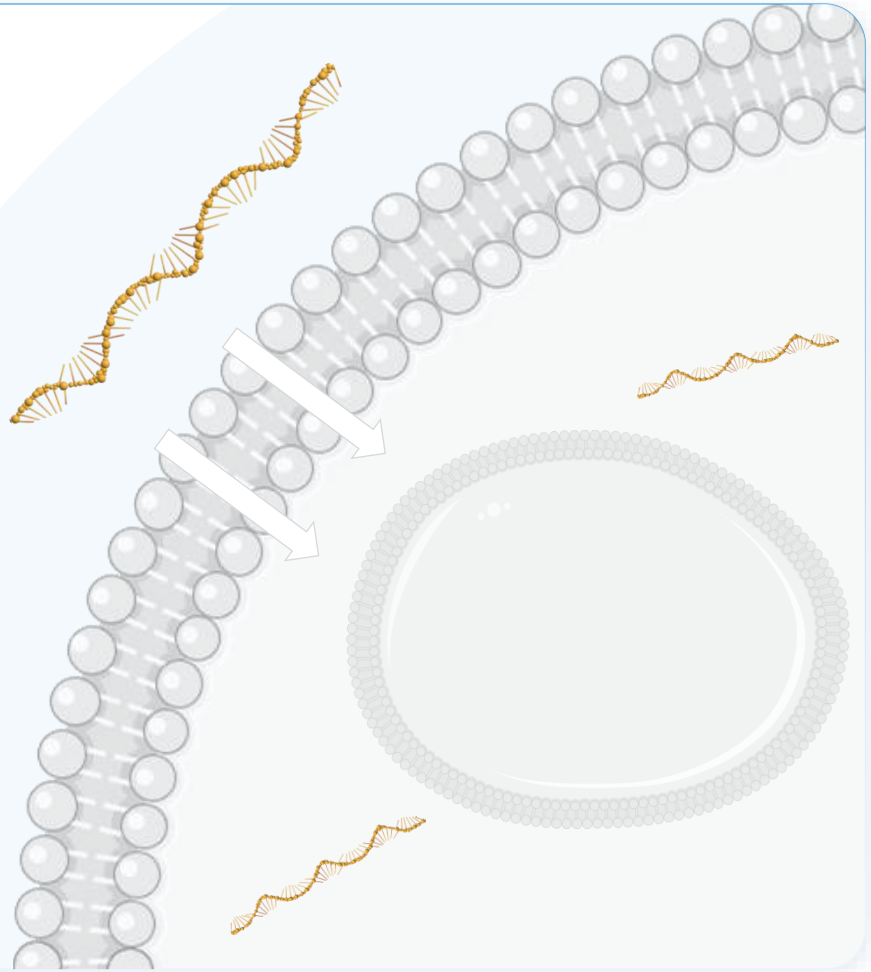
Research

- DMD *Exon 45, Exon 44*
- Additional neuromuscular diseases
- Neurological diseases

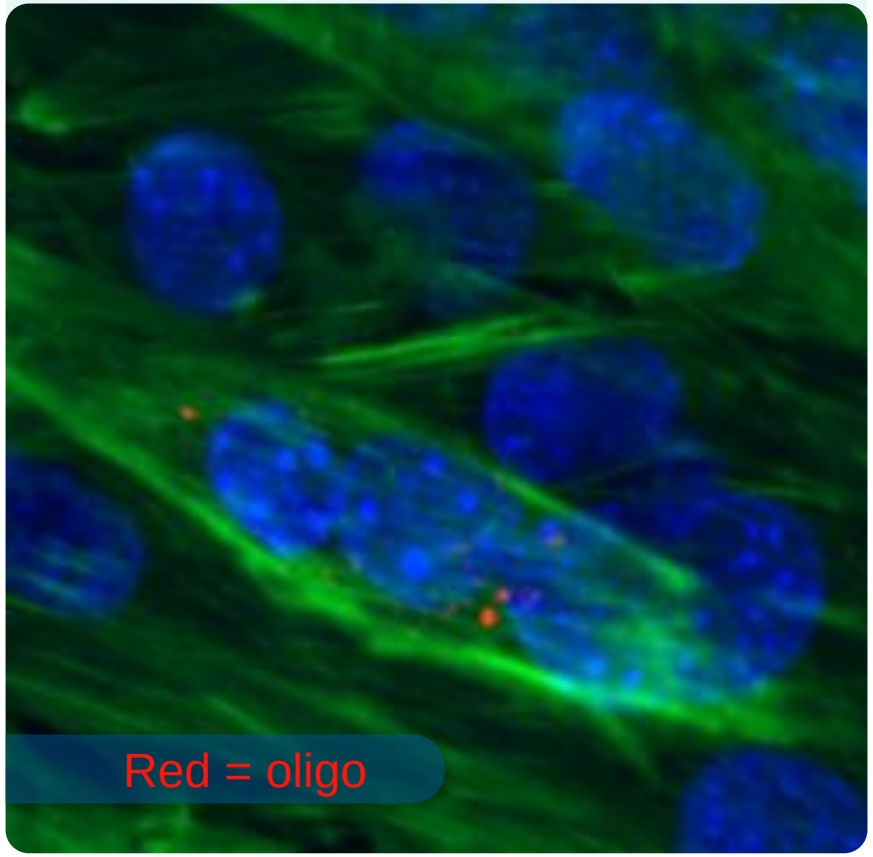
Note: Clinical plans are subject to alignment with regulatory authorities.

The Challenge of Oligonucleotides

Naked oligonucleotides do not efficiently penetrate muscle cells and nucleus



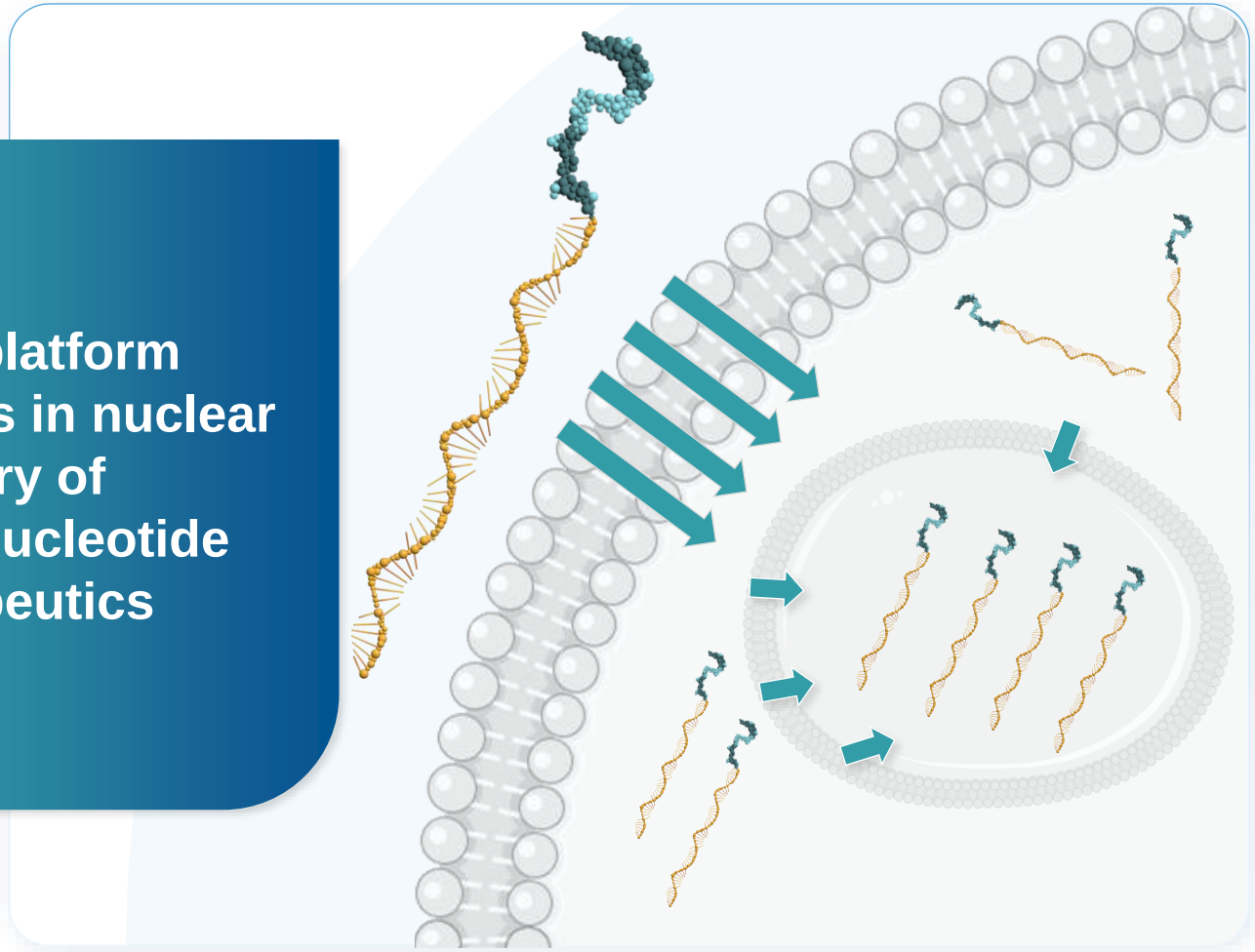
Naked Oligonucleotide (PMO)



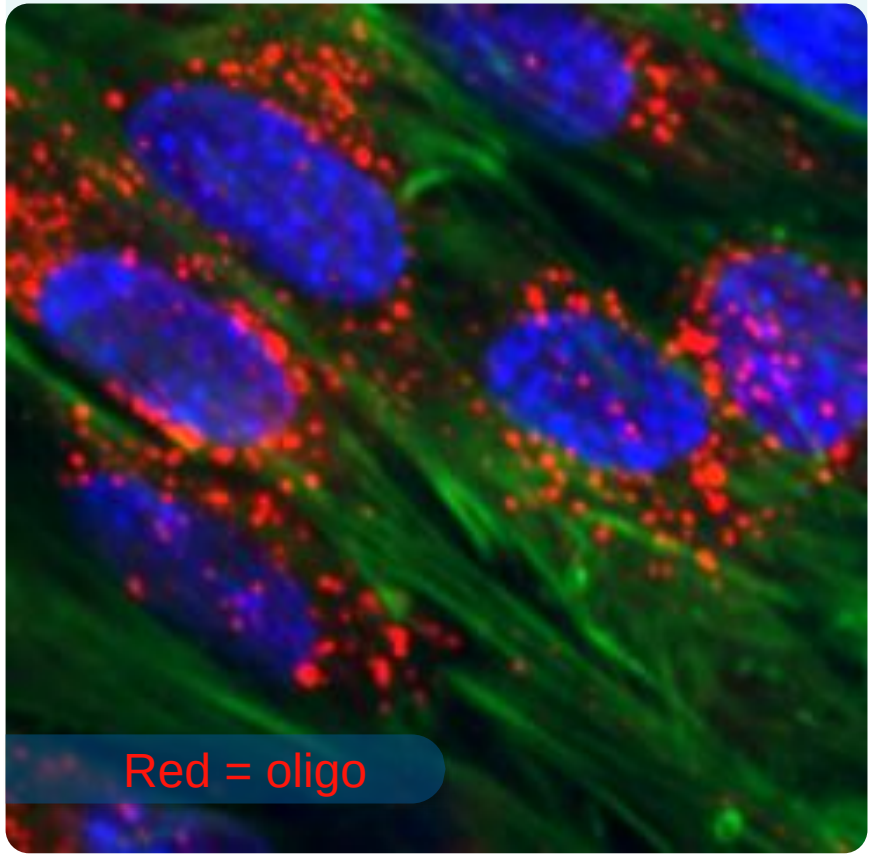
In vitro staining image is shown with 10µM concentration of PMO23 (naked oligonucleotide). C2C12 mouse cells were differentiated for 4 days into myotubes and treated with fluorescently tagged compounds for 24h. PMO: phosphorodiamidate morpholino oligonucleotide

PepGen's EDO Platform Has Been Designed and Developed to Solve this Decades Long Problem

EDO platform results in nuclear delivery of oligonucleotide therapeutics



PepGen's EDO: Up to 25X Higher Nuclear Uptake of Oligonucleotide



In vitro staining image is shown with 10µM conc. of EDO23. C2C12 mouse cells were differentiated for 4 days into myotubes and treated with fluorescently tagged compounds for 24h.

Latest Achieved Milestones

DM1: PGN-EDODM1

- FREEDOM-DM1 (Phase 1) open in US, Canada and UK
- FREEDOM2-DM1 (Phase 2) open in Canada and UK
  - First participant dosed in Q4 2024

DMD: PGN-EDO51

- CONNECT1-EDO51 (Phase 2) at 5 mg/kg
  - Encouraging levels of muscle adjusted dystrophin production (0.70%) and total dystrophin production (0.26%) after 3 months and 4 doses
  - High levels of exon 51 skipping (2.15%)
  - Data demonstrates EDO technology delivers high levels of oligonucleotides to the nucleus
- CONNECT1 10 mg/kg – all 4 participants have received at least one dose

Anticipated Upcoming Milestones

DM1

- FREEDOM 5 mg/kg and 10 mg/kg clinical results by end of Q1 2025
- FREEDOM 15 mg/kg clinical results in 2H 2025

2025

DMD

- CONNECT1 10 mg/kg clinical results by year-end 2025

PGN-EDODM1 for DM1

Myotonic Dystrophy Type 1 (DM1) Overview and Unmet Medical Need

Overview

- Mutation in DMPK gene
- Onset of symptoms variable-childhood to adulthood
  - Myotonia
  - Muscle weakness
  - Cardiac arrythmias
  - Loss of lung function
- Average life expectancy is 50-60 years for non-congenital forms of DM1

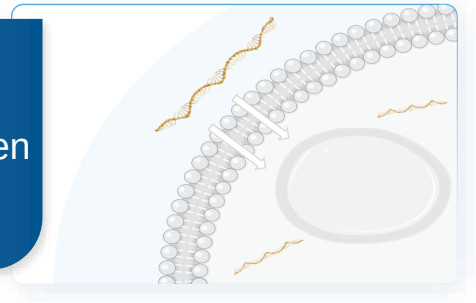
Market opportunity

- US and EU over 110,000 patients
- No approved therapies that address underlying cause of the disease

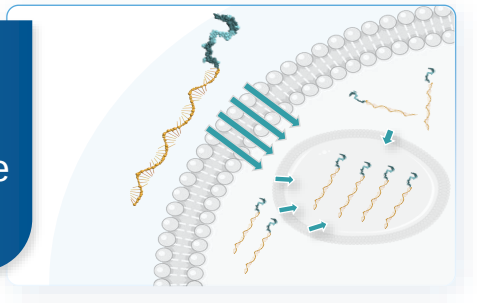
Sources: Neuroepidemiology (2022) 56 (3): 163–173., Neurology 2021 Feb 16;96(7):e1045-e1053

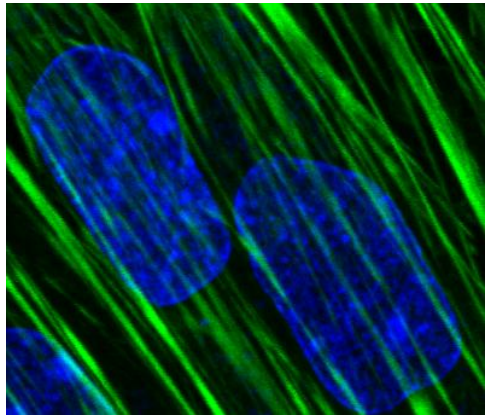
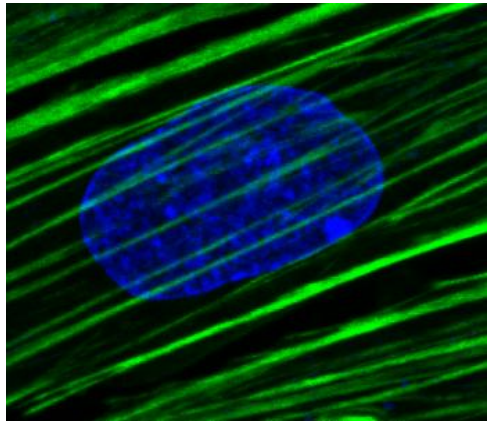
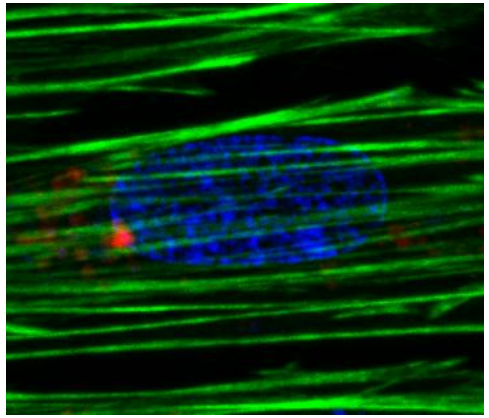
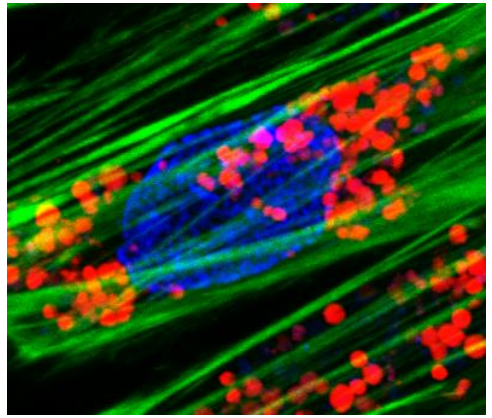
PGN-EDODM1 is Efficiently Delivered to the Nuclei of Muscle Cells

Naked oligonucleotides not efficiently taken up into muscle cells & nucleus



EDO's enhance nuclear delivery of oligonucleotide therapeutics



Not treated	PGN-PMODM1	PGN-EDODM1	
			
0 µM	20 µM	2 µM	10 µM

PGN-EDODM1 / Actin / Nucleus

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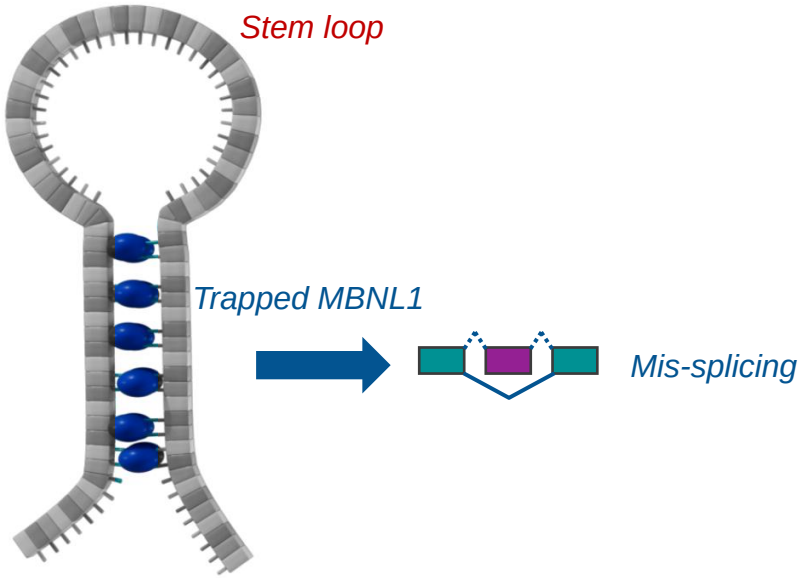
Immortalized myoblasts from a healthy individual or a DM1 patient with 2600 CTG repeats were cultured then differentiated for 4 days into myotubes and then treated with fluorescently tagged PGN-PMODM1 (PMO) or PGN-EDODM1 (PPMO) at concentrations detailed above. Cells were visualized by confocal microscopy 24h after treatment.

PepGen's Novel Therapeutic Approach to Treat Root Cause of DM1

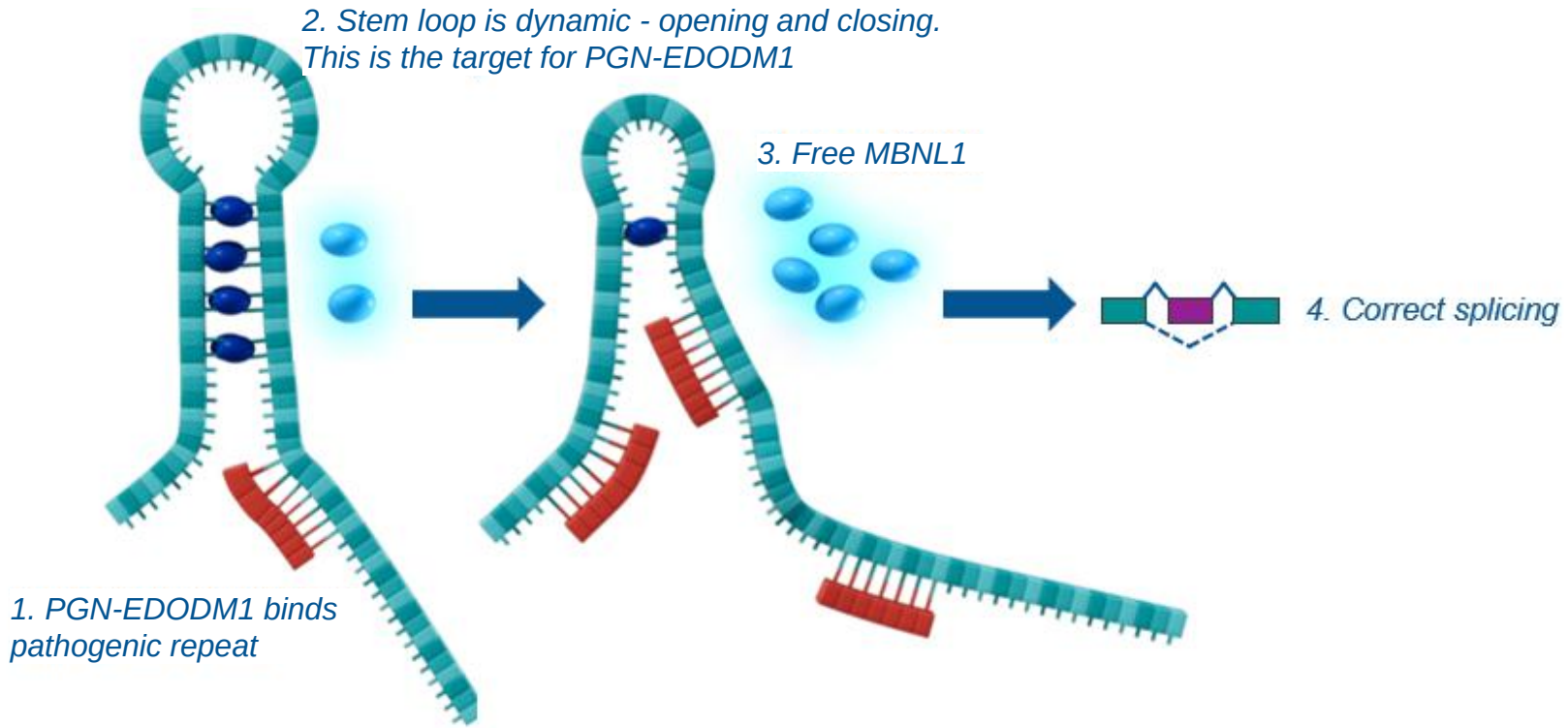
DM1

Repeating RNA "CUG" sequences, associated with DM1, form stem loop, trapping MBNL1 proteins



PGN-EDODM1 Targets Pathogenic DMPK

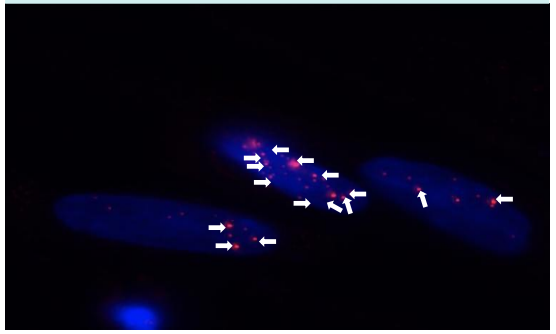
EDO enhances delivery of PMO cargo to the nucleus, targeting pathogenic DMPK with expanded CUG repeats



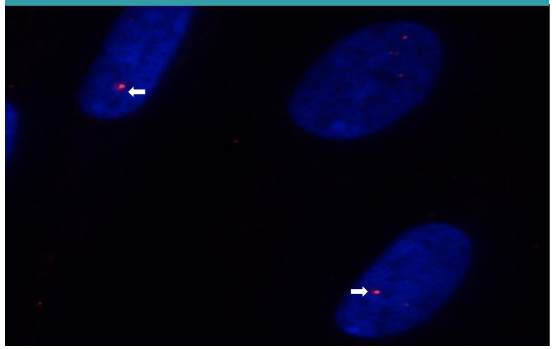
PGN-EDODM1 Reduced Pathogenic Nuclear Foci, Liberated MBNL1 and Corrected Mis-Splicing in Patient Cells with Long CUG Repeats

Foci Reduction

Not Treated



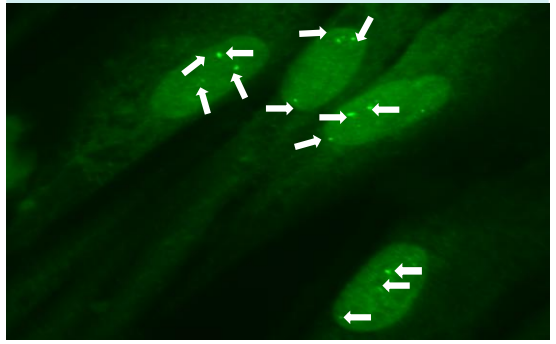
PGN-EDODM1 Treated



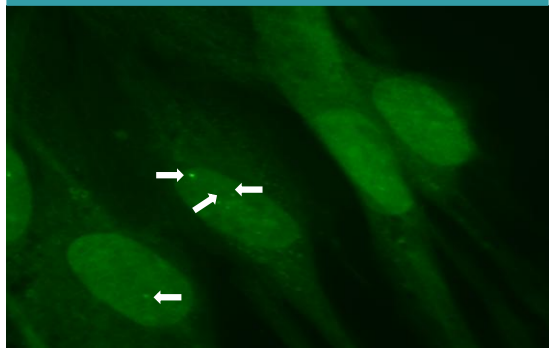
54%
reduction in toxic foci

MBNL1 Liberation

Not Treated



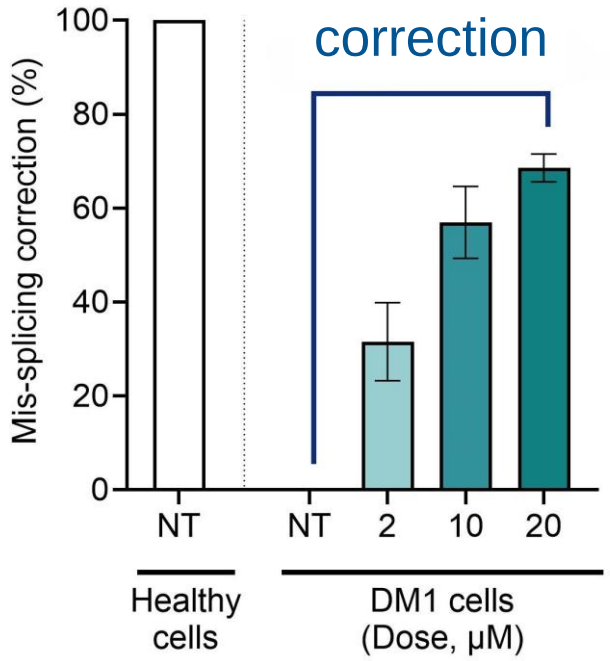
PGN-EDODM1 Treated



Mis-Splicing Correction

Across multiple transcripts

69%
correction



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

PGN-EDODM1 Corrected Movement Disorder of DM1 Mouse Model

Non-Treated HSA^{LR}

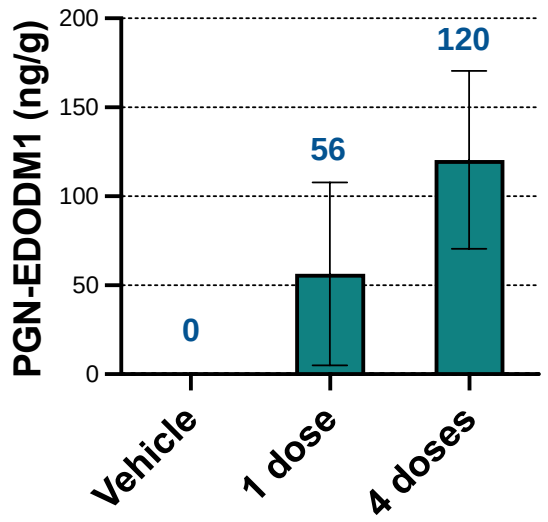
Treated HSA^{LR}

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

Multiple Doses of EDOs Have Been Shown to Lead to Greater Efficacy Than Single Doses in Both Preclinical and Clinical Healthy Volunteer (HV) Studies

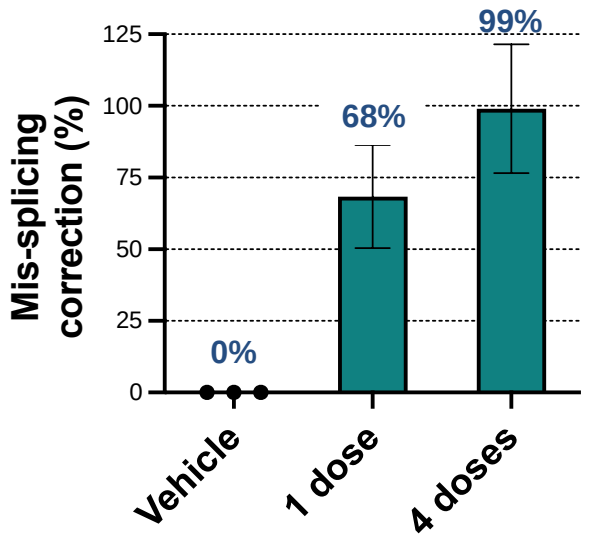
Tissue Concentration

Skeletal muscle



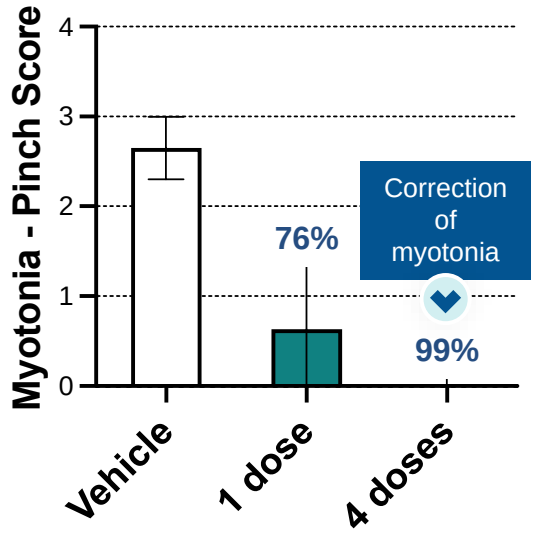
Mis-Splicing Correction

Across multiple transcripts



Correction of Myotonia

Pinch test



EDO technology (PGN-EDO51) resulted in activity in HVs while achieving PMO concentration of 136 ng/g with a single dose; therefore, we expect meaningful improvement in key biomarkers with a single dose of PGN-EDODM1 in FREEDOM

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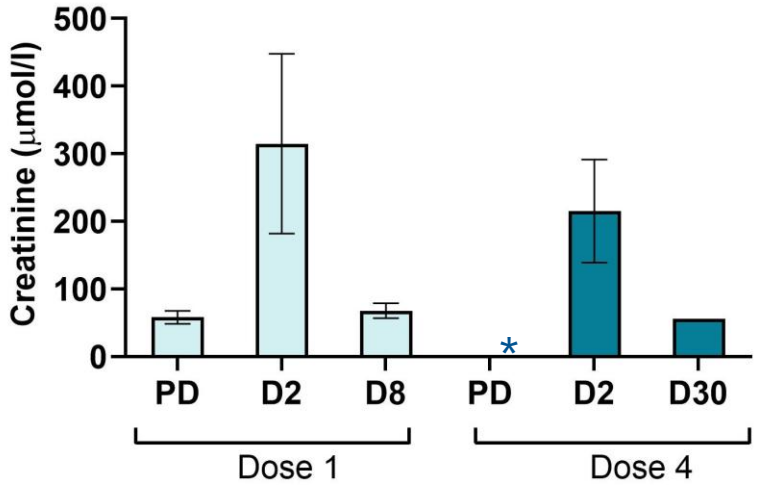
Protocol: HSA^{LR} mice received 1 or 4 doses of PGN-EDODM1, with 4-week intervals between doses. Skeletal muscle tissues were collected 4 weeks post-final dose. Skeletal muscle tissue concentration was measured by fluorescent based HPLC method. Graph is presented as mean ± SD; n = 8-12 per cohort. Mis-splicing analysis considers multiple transcripts. Graph is presented as mean ± SD; n = 8-12 per cohort per transcript. Action myotonia evaluation (pinch test) was performed 4 weeks post-final dose. Grade 3 = Clear sign of myotonia strong AND reproducible, Grade 2 = Clear sign of myotonia, strong OR reproducible, Grade 1 = Clear sign of myotonia but non reproducible, Grade 0 = No sign of myotonia. Graph is presented as mean ± SD; n = 12-43 per cohort.

Preclinical Safety: Observed Changes in Creatinine are Transient and Not Associated with Adverse Kidney Findings

We believe these results support the potential tolerability of PGN-EDODM1 with repeat dosing

PGN-EDODM1 REPEAT-DOSE SERUM CREATININE



Dosing schedule

Month:	0	1	2	3
	●	●	●	●

● PPMO dose: 60 mg/kg

- Transient increase in serum creatinine resolved within a week post-dose
- No adverse findings in the kidney even after 4 doses up to 60 mg/kg
- No notable hematologic, cardiovascular or hepatic effects in 13-week study

* Data not collected. PD = pre-dose. NHP protocol: PGN-EDODM1 was administered to NHP (males and females) by IV infusion at 60 mg/kg over 60 min (n=5/sex) for 4 doses. Dosing schedule was once every 28 days. Male and female results were comparable with male-only data presented. Shown as mean ± SD.

FREEDOM: Phase 1 PGN-EDODM1 Single-Ascending Dose Study Design

Freedom
DM1

FREEDOM Study Overview

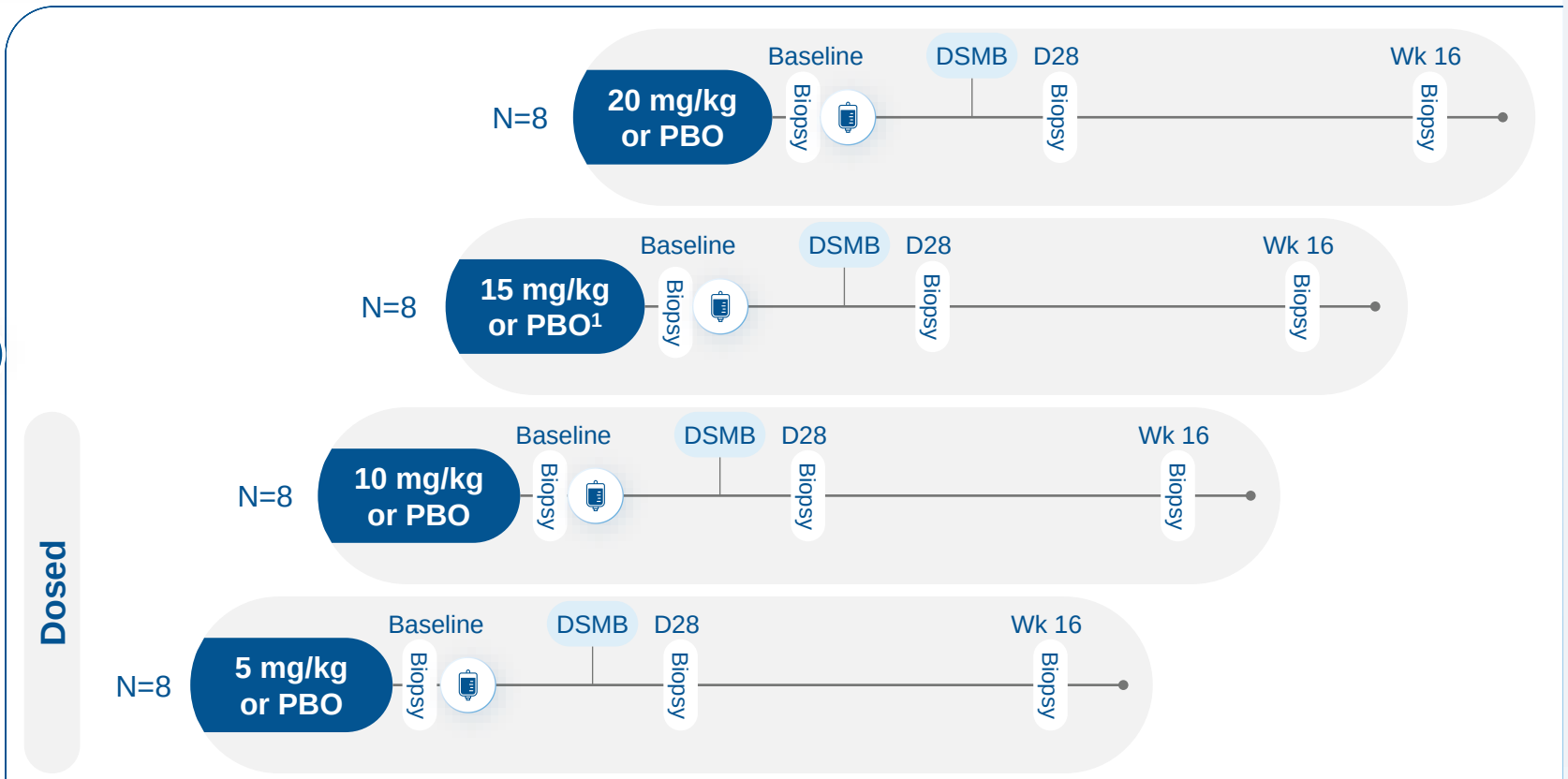
Multinational, randomized, double-blind, placebo-controlled SAD study in patients

Single IV administration of PGN-EDODM1

Muscle biopsies in tibialis anterior at baseline, day 28, week 16

Initial functional assessments, correction of mis-splicing and safety

Single Dose PGN-EDODM1 or Placebo (randomized 3:1)



1.15 mg/kg cohort added to expand pharmacokinetic and pharmacodynamic understanding.
DSMB: data safety monitoring board; IV: intravenous; PBO: placebo; SAD: single-ascending dose

FREEDOM Informed Design of Phase 2 FREEDOM2 Trial

Freedom DM1

Enrolling in US, Canada and UK

- Phase 1 randomized, double-blind, placebo-controlled SAD study in patients
- Key anticipated readouts: Functional assessments, correction of mis-splicing, safety data

Freedom 2 DM1

Multinational Study Initiated

- Phase 2 randomized, double-blind, placebo-controlled multiple-ascending dose (MAD) study in patients
- Dosing initiated in FREEDOM2 in Q4 2024
  - IV administration of PGN-EDODM1 every 4 weeks up to 12 weeks
  - Key anticipated readouts: Functional assessments, correction of mis-splicing, safety data

FREEDOM2: Phase 2 Multiple-Ascending Dose Study Design

Freedom 2
DM1

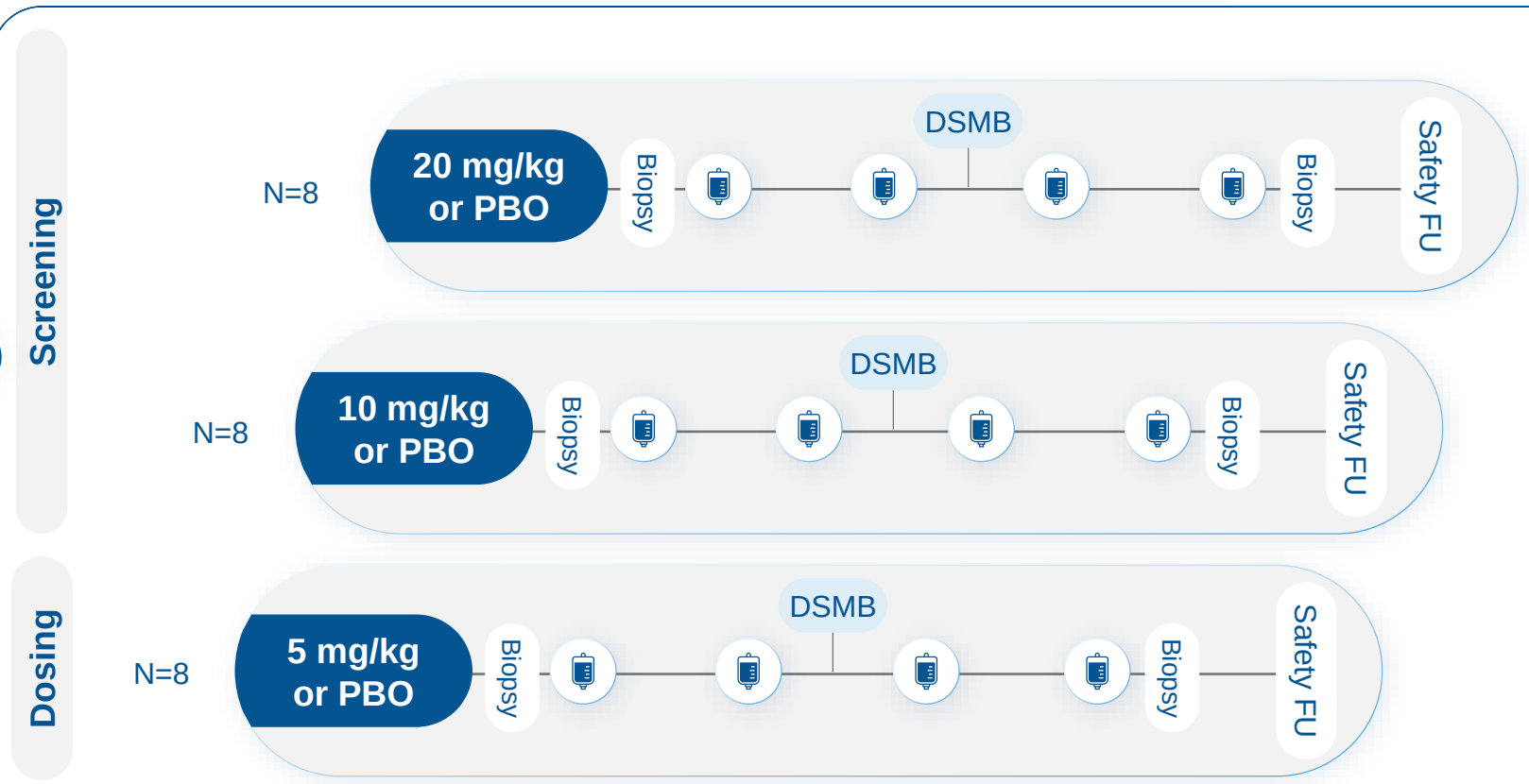
FREEDOM2 Study Overview

Multinational, randomized, double-blind, placebo-controlled, MAD study

IV administration of PGN-EDODM1 or placebo every 4 weeks for a period of 12 weeks

Key endpoints: Safety, PK, correction of splicing, functional assessments: vHOT, hand grip, 10-meter walk run test

4 Doses of PGN-EDODM1 or Placebo (randomized 3:1)



DSMB: data safety monitoring board; FU follow-up; IV: intravenous; MAD: multiple-ascending dose; PBO: placebo; PK: pharmacokinetics; vHOT: video hand opening test

PGN-EDODM1 dose every 4 weeks

PGN-EDO51 for DMD

Duchenne Muscular Dystrophy (DMD) Overview and Unmet Medical Need

Overview

- Caused by mutation in dystrophin gene resulting in progressive muscle damage
- Onset of symptoms in early childhood
  - Loss of ambulation by early adolescence
  - Loss of respiratory and cardiac function resulting in early adulthood mortality

Market opportunity

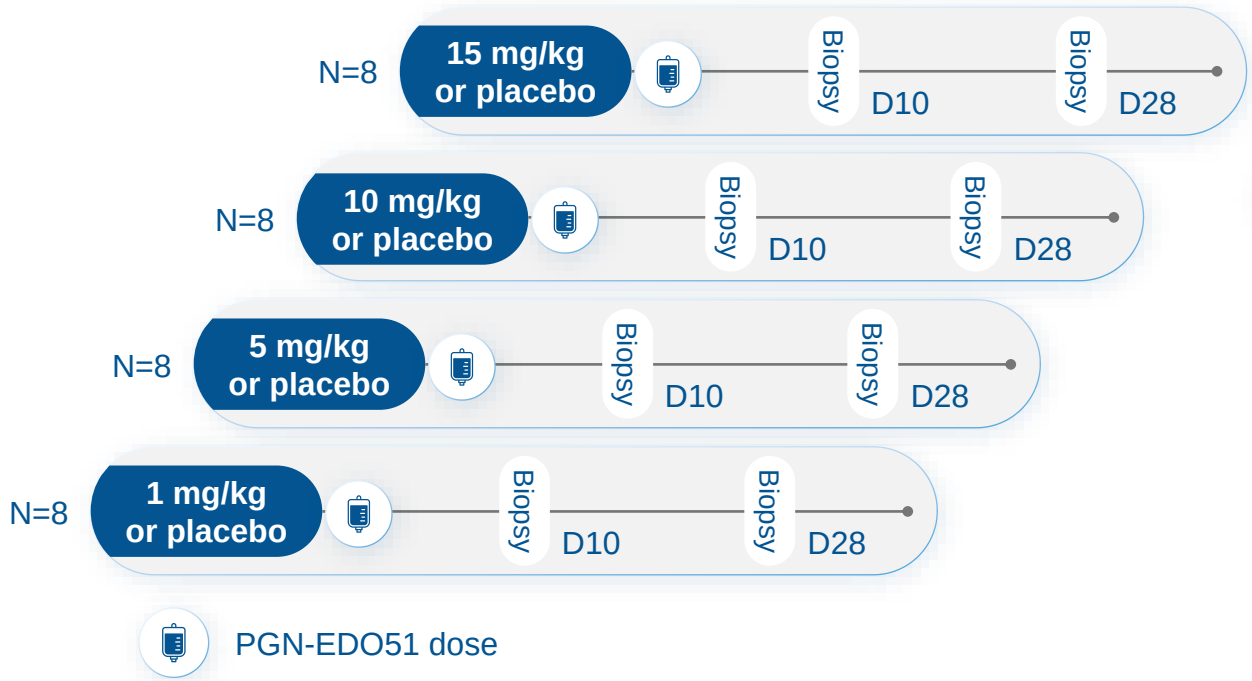
- US and EU ~40,000 patients
- ~21% patients amenable to:
  - PGN-EDO51: Phase 2 (exon 51)
  - PGN-EDO53: CTA/IND enabling studies advancing (exon 53)
- Novel therapies needed to restore functional dystrophin and prevent loss of muscle function and early mortality

Source: https://www.cureduchenne.org/cure/exon-skipping/.

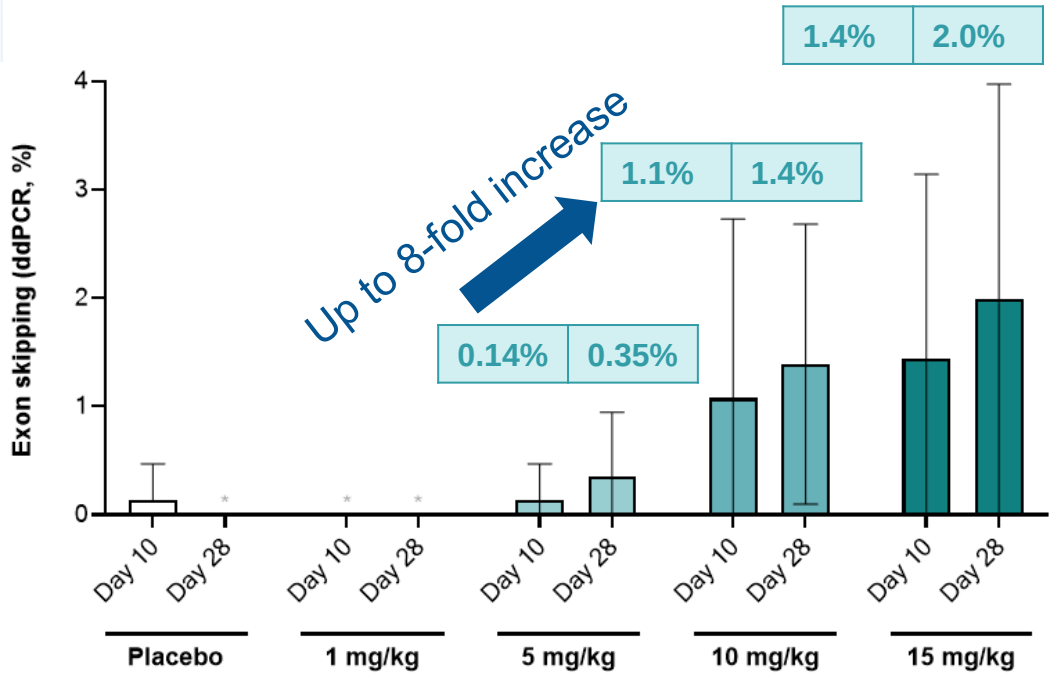
Healthy Volunteer Study Results Led to CONNECT1: Highest Levels of Exon 51 Skipping in Humans Following Single Dose of PGN-EDO51[1]

Phase 1 Healthy Volunteer (HV) Trial Design

- Study population: Healthy adult males (n=32; 8 per cohort, 3:1 PGN-EDO51:placebo)
- Dosing: Single dose, IV administration
- Biceps biopsies conducted on Day 10 and Day 28



Trial Results: Exon Skipping (Biceps)



Protocol PGN-EDO51-101: Phase 1, first in human, randomized double blind, placebo controlled, single ascending dose study in healthy adult volunteers. Single dose of either PGN-EDO51 or placebo administered by IV infusion at doses indicated. Participants were followed for 28-day period following dose administration to evaluate safety, tolerability, pharmacokinetics, and pharmacodynamics. Needle biopsies of biceps muscle were taken on Days 10 and 28. Exon skipping measured by ddPCR. Shown as mean ± SD; n = 6 PGN-EDO51: 2 placebo per cohort (n=5 for D10 at 15 mg/kg). Asterisk indicates values that were under the lower level of quantification. 1.Comparative statement based on cross-trial comparison of Phase 1 HV data of single dose administration of EDO51 with publicly available Phase 1 HV data following a single dose of other exon skipping approaches (vesleteplirsen and eteplirsen).

CONNECT1: Designed to Establish Proof-of-Concept and Inform CONNECT2 Clinical Trial Design

Connect 1
EDO51

CONNECT1 Study Overview

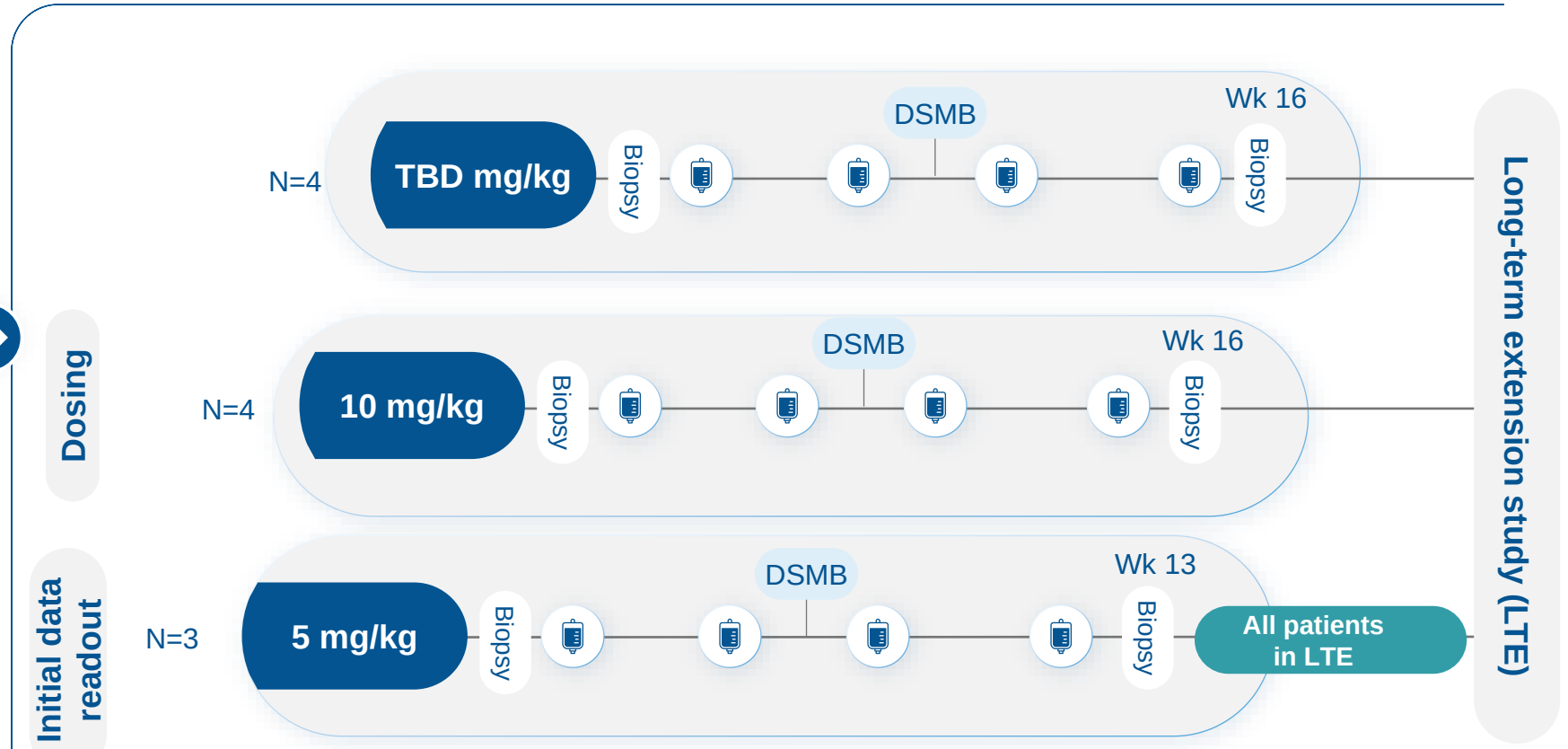
Open label, multiple ascending dose (MAD) clinical trial in Canada

DMD patients (n=11) with exon 51 skippable mutation

Ages 6-16, ambulatory and non-ambulatory

Key endpoints: Safety and tolerability, dystrophin production, muscle tissue concentration of PGN-EDO51, exon skipping

Open Label Study in Patients with DMD Amenable to Exon 51 Skipping Therapy



DSMB: data safety monitoring board

PGN-EDO51 dose every 4 weeks

CONNECT1 5 mg/kg: Baseline Characteristics of Participants (N=3)

	Mean (SD)
Age (years)	11.7 (1.5)
BMI (kg/m^2)	19.8 (2.7)
Height (cm)	132.0 (9.9)
Weight (kg)	34.4 (3.9)
Age of DMD genetic diagnosis (years)	6.3 (1.5)
Number of patients on daily corticosteroid dosing regimen	3
Number of ambulatory patients	3
Number of patients previously on DMD therapy	0

CONNECT1 5 mg/kg: Safety Profile[1]

MAD Period	N(%)
Any TEAEs, n(%)	3 (100)
Related to study drug	1 (33.3)
• Mild • Moderate • Severe	1 (33.3) 0 0
Serious Adverse Events (AEs)	0
AEs leading to dose modification/ discontinuation/interruption	0
AEs leading to death	0

- All treatment emergent adverse events (TEAEs) were mild and resolved
- Related TEAE was mild (abdominal pain, flatulence)
- No discontinuations, dose modifications or dose interruptions
  - All participants rolled over to the long-term extension study
- No sustained elevation in kidney biomarkers
- No changes in electrolytes
  - No hypomagnesemia or hypokalemia
- No changes in hepatic function
- No anemia or thrombocytopenia

At 5 mg/kg, a total of 31 doses have been administered (12 doses in the MAD period+19 doses in the LTE period)[2]

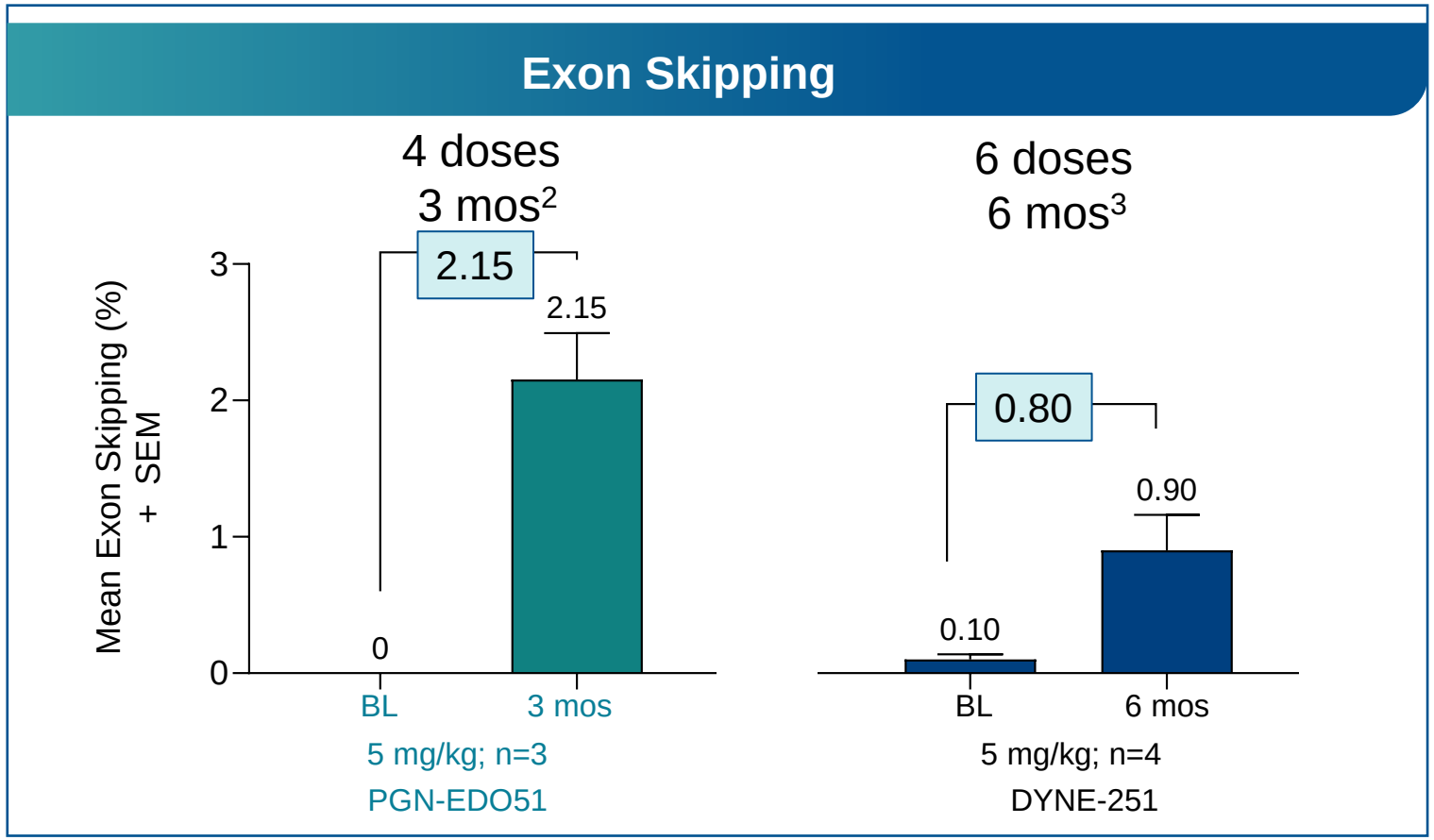
1. As of May 31, 2024
2. As of November 5, 2024

Dosing Continues in Cohort 2 at 10 mg/kg[1]

- Emerging safety profile is favorable
- At 10 mg/kg, a total of 10 doses have been administered
- All treatment-related adverse events have been mild or moderate
- No treatment-related serious adverse events
- Asymptomatic hypomagnesemia has been observed in 2 patients. They have been treated with oral supplementation and remain on study
- No dose discontinuations, dose modifications or dose interruptions
- No sustained elevation in kidney biomarkers
- No changes in hepatic function
- No hypokalemia, anemia or thrombocytopenia

1. As per the data obtained through October 31, 2024

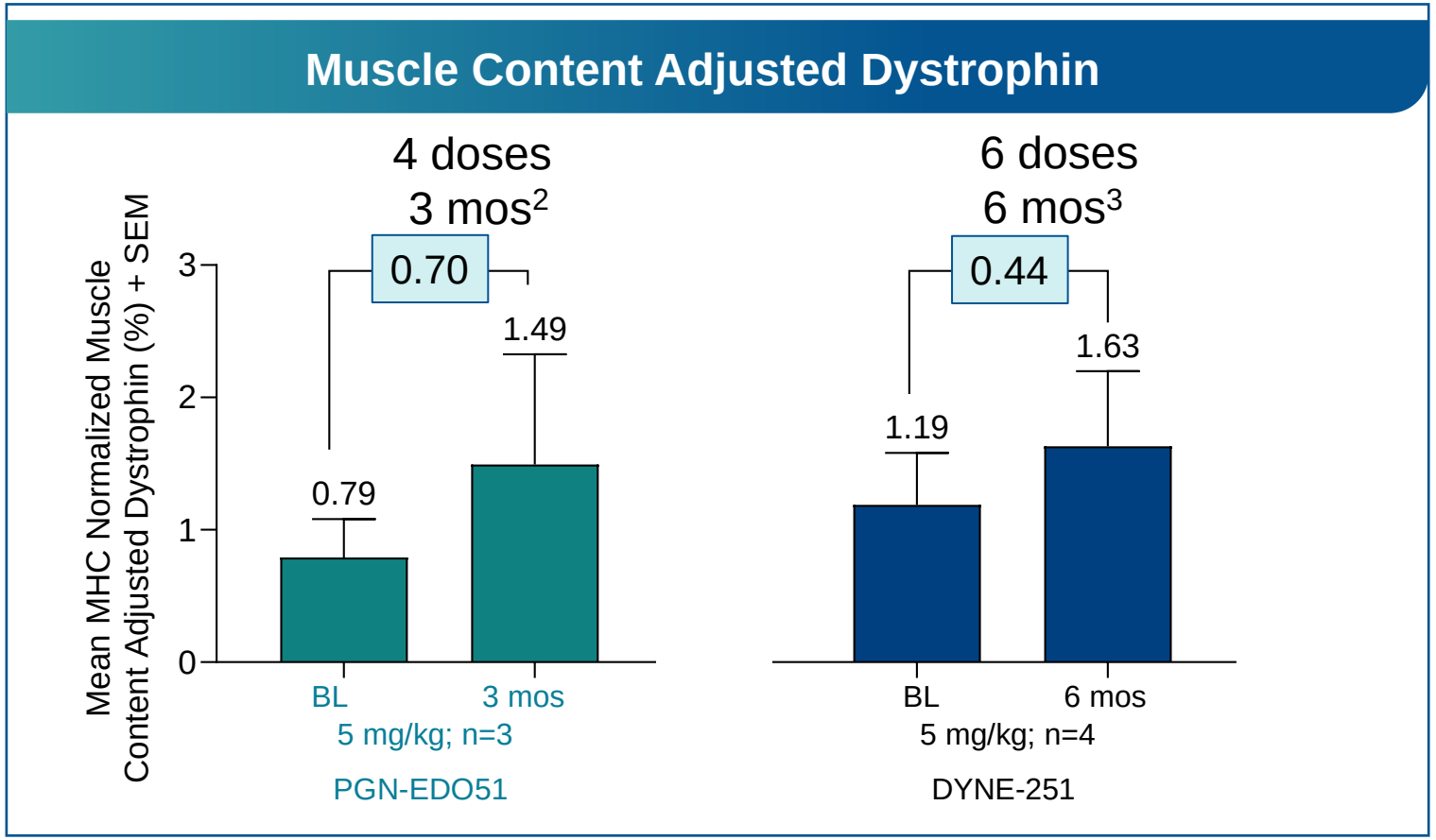
PGN-EDO51 Showed High Levels of Mean Exon Skipping



2. PGN-EDO51 muscle biopsy taken approximately 7 days after last dose
3. DYNE-251 muscle biopsy taken approximately 28 days after last dose

1. No head-to-head trials have been conducted comparing PGN-EDO51 to DYNE-251. Data from studies of these clinical candidates may not be directly comparable due to differences in molecule composition, trial protocols, dosing regimens, and patient populations and characteristics. Accordingly, cross-trial comparisons may not be reliable.DYNE-251 DELIVER clinical data update, May 20, 2024. Note: DYNE-251 error bars estimated based on public presentations.

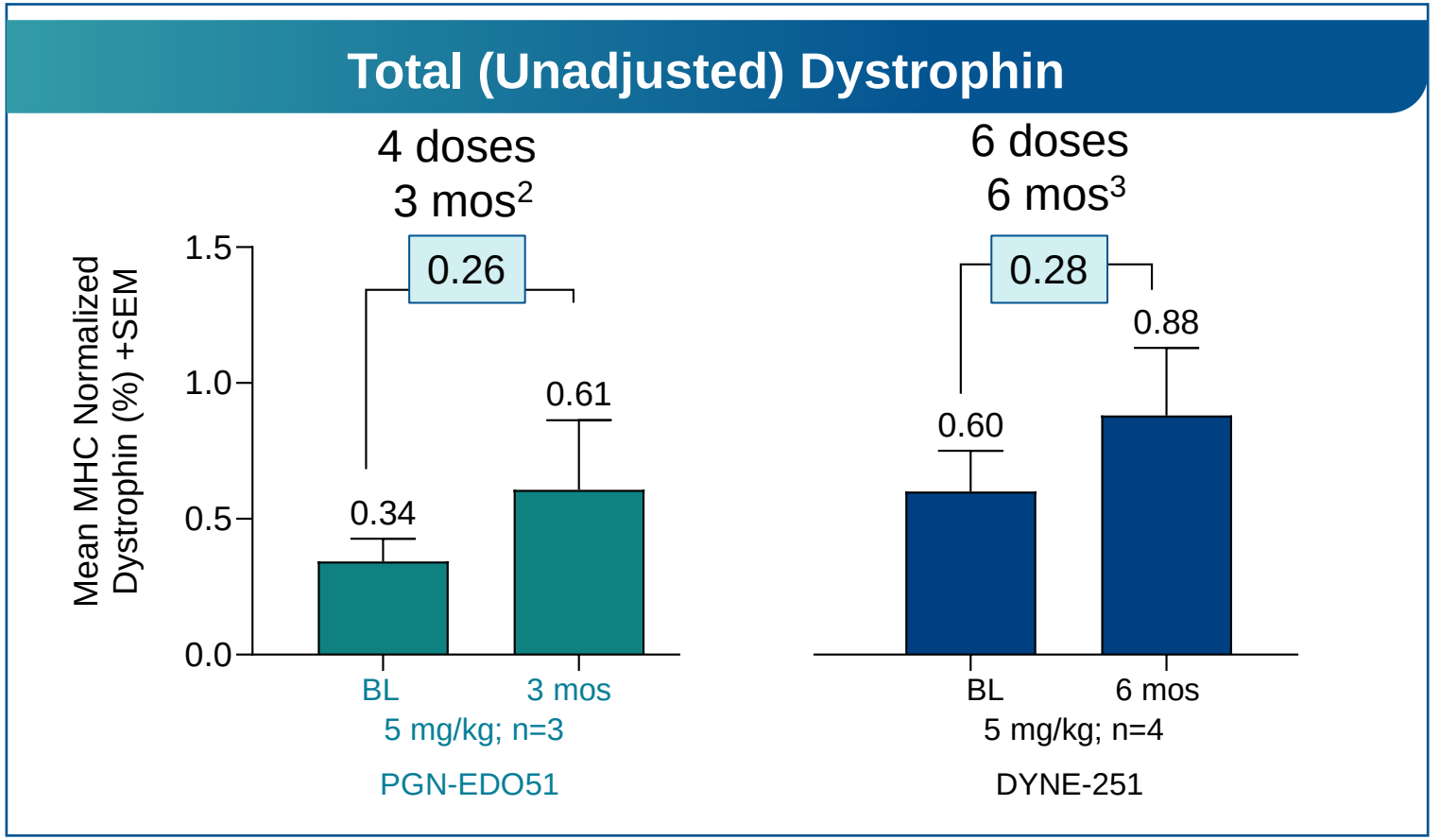
PGN-EDO51 Produced Greater Muscle Content Adjusted Dystrophin Increase in Half the Treatment Duration and Fewer Doses[1]



2. PGN-EDO51 muscle biopsy taken approximately 7 days after last dose
3. DYNE-251 muscle biopsy taken approximately 28 days after last dose

1. No head-to-head trials have been conducted comparing PGN-EDO51 to DYNE-251. Data from studies of these clinical candidates may not be directly comparable due to differences in molecule composition, trial protocols, methodologies for calculating muscle content adjusted dystrophin, dosing regimens, and patient populations and characteristics. Accordingly, cross-trial comparisons may not be reliable.DYNE-251 DELIVER clinical data update, May 20, 2024. Note: Dyne-251 error bars estimated based on public presentations.

PGN-EDO51 Produced Similar Dystrophin Increase in Half the Treatment Duration[1]



2. PGN-EDO51 muscle biopsy taken approximately 7 days after last dose
3. DYNE-251 muscle biopsy taken approximately 28 days after last dose

1. No head-to-head trials have been conducted comparing PGN-EDO51 to DYNE-251. Data from studies of these clinical candidates may not be directly comparable due to differences in molecule composition, trial protocols, dosing regimens, and patient populations and characteristics. Accordingly, cross-trial comparisons may not be reliable.DYNE-251 DELIVER clinical data update, May 20, 2024. Note: Dyne-251 error bars estimated based on public presentations.

CONNECT1 Key Takeaways

- Emerging safety profile is favorable[1]
- All patients dosed at 5 mg/kg demonstrated increased exon skipping and dystrophin production and have continued into the long-term extension study
- PGN-EDO51 generated encouraging levels of muscle adjusted dystrophin production (0.70%) and total dystrophin production (0.26%) after just 3 months and 4 doses at 5 mg/kg
- PGN-EDO51 produced high levels of mean exon 51 skipping (2.15%) after just 3 months and 4 doses at 5 mg/kg
- Initial results support that our EDO technology delivers high levels of oligonucleotides to the nucleus

Potentially higher levels of dystrophin production are expected with higher doses of PGN-EDO51 over longer treatment periods

1. For the 5 mg/kg and 10 mg/kg cohorts, as of October 31, 2024

PGN-EDO51 Development Path to Support Registration

Ongoing

Phase 2: Open-label MAD trial

Open in Canada

Proof-of-concept: Dystrophin expression at 13-16 weeks[1]

Open

Phase 2: Randomized, double-blind, placebo-controlled MAD trial

Multinational trial; open in United Kingdom

Potential to support accelerated approval[2]: Dystrophin expression at 25 weeks

1. Dystrophin expression measured at 13 weeks in Cohort 1 and at 16 weeks in Cohorts 2 and 3
2. Subject to alignment with regulatory authority feedback

CONNECT2: Phase 2 PGN-EDO51 MAD Study

Connect 2
EDO51

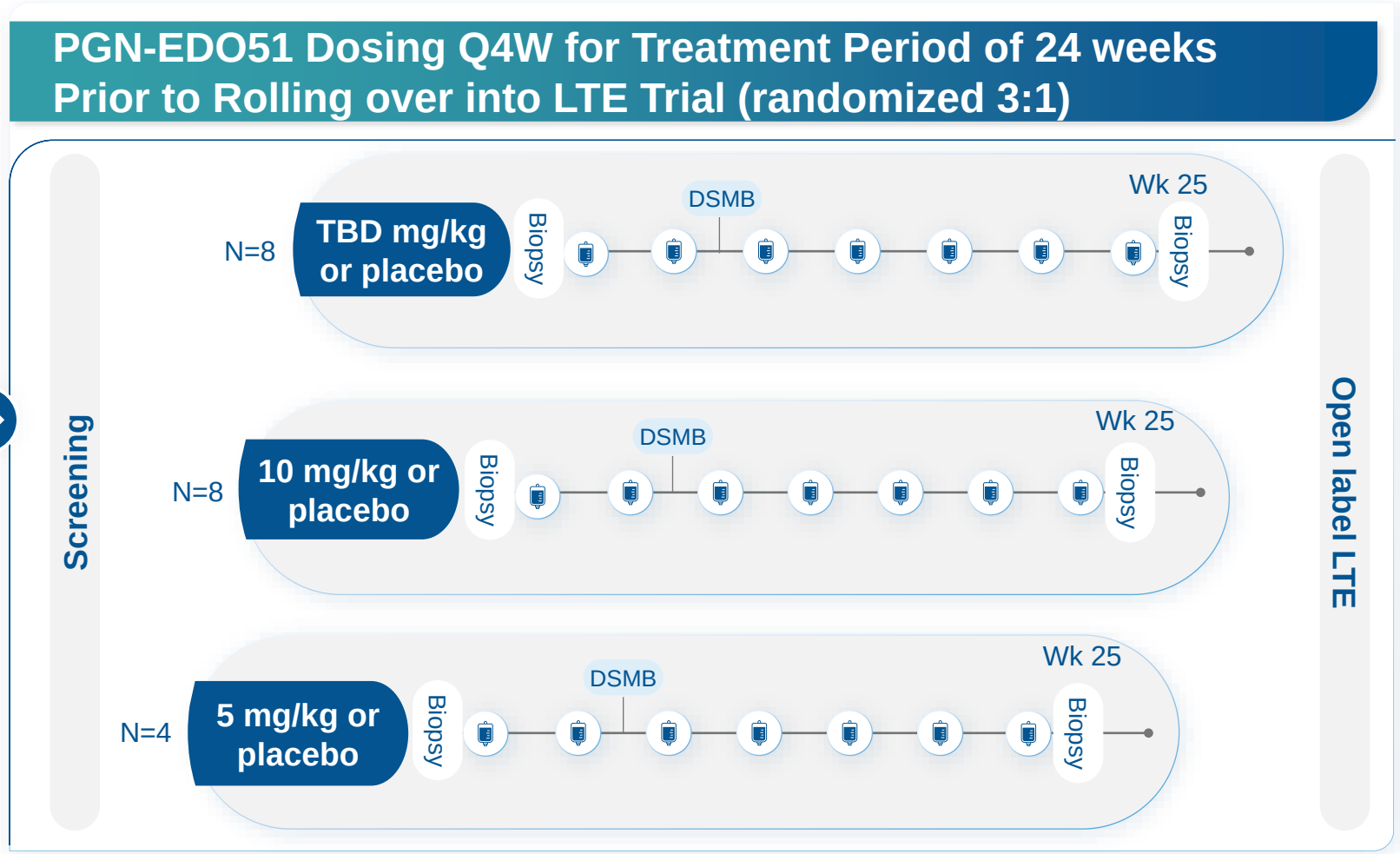
CONNECT2 Study Overview

Multinational, randomized, double-blind, placebo-controlled trial

IV administration of PGN-EDO51 or placebo every 4 weeks

Muscle biopsies in biceps at baseline and week 25

Key endpoints: Safety biomarkers, dystrophin, exon skipping, North Star Ambulatory Assessment (NSAA), Time to stand from supine, Performance of Upper Limb



Conclusion

Key Readouts Starting in Q1 2025 with Existing Cash Funding Planned Operations into 2026

Key expected data readouts/milestones

PGN-EDODM1
DM1

- FREEDOM 5 mg/kg and 10 mg/kg clinical results by end of Q1 2025
- FREEDOM 15 mg/kg clinical results in 2H 2025
- FREEDOM2 first participant dosed in Q4 2024

PGN-EDO51
DMD Exon 51

- CONNECT1 10 mg/kg clinical results by year-end 2025
- CONNECT2
  - Currently open in UK
  - Engaging with EU regulators
  - Received clinical hold notice December 2024 from US FDA