UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): March 27, 2023

PepGen Inc.

(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-41374**	**85-3819886**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

321 Harrison Avenue **8th Floor** **Boston, Massachusetts**	**02118**
(Address of Principal Executive Offices)	**(Zip Code)**

Registrant's Telephone Number, Including Area Code: 781 797-0979

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common stock, par value $0.0001 per share	PEPG	Nasdaq Global Select Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

On March 27, 2023, PepGen Inc. (the "Company") updated its Corporate Presentation, a copy of which is being furnished as Exhibit 99.1 and incorporated herein by reference. The information in this report (including Exhibit 99.1) is being furnished pursuant to Item 7.01 and shall not be deemed to be "filed" for the purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such filing. This report will not be deemed an admission as to the materiality of any information in this Item 7.01 (including Exhibit 99.1).

Item 9.01 Financial Statements and Exhibits.

Exhibit Number	Description
99.1	Corporate Presentation updated on March 27, 2023
104	Cover Page Interactive Data File (embedded within Inline XBRL document)

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

PEPGEN INC.

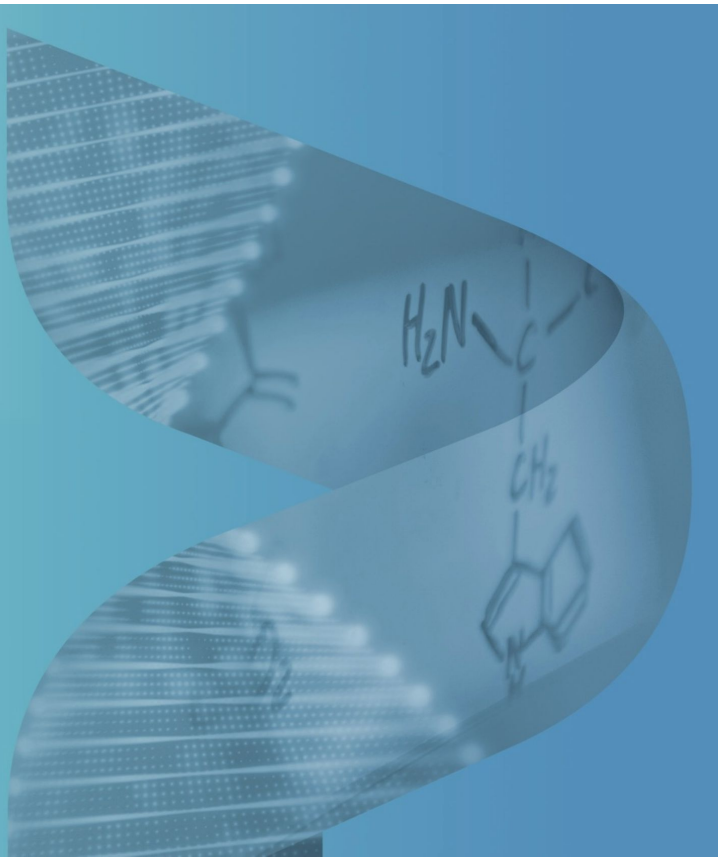
Date: March 27, 2023

By: /s/ Noel Donnelly
Noel Donnelly, Chief Financial Officer

PepGen

EMPOWERING OLIGONUCLEOTIDE THERAPEUTICS

COMPANY PRESENTATION
MARCH 2023



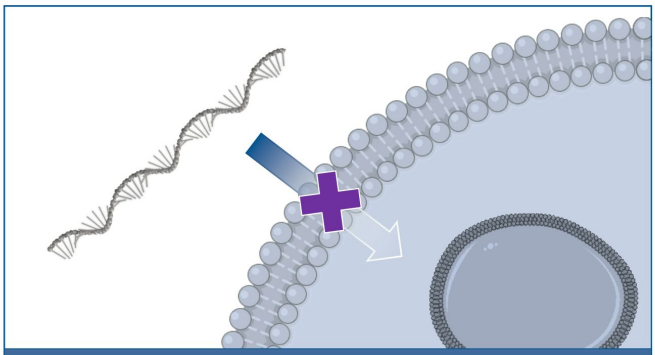
DISCLAIMERS

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.

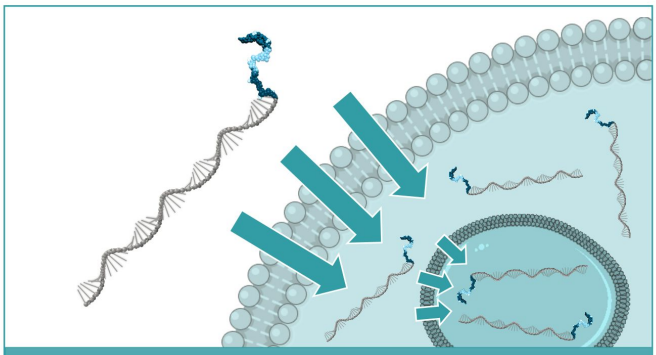
PEPGEN'S EDO TECHNOLOGY IS DESIGNED TO ADDRESS THE DELIVERY CHALLENGES THAT LIMIT OLIGONUCLEOTIDE THERAPEUTICS

THE CHALLENGE



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

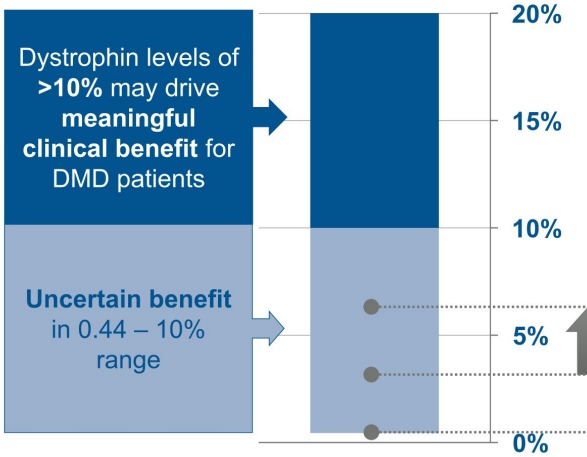
THE EDO SOLUTION



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

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

DYSTROPHIN PRODUCTION (%)



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**

6.55%**

SRP-5051 (vesleteplirsen) – *Sarepta Therapeutics*

- Dystrophin level at 20 and 30 mg/kg
- Currently in Ph2b

3.06%**

0.44%*

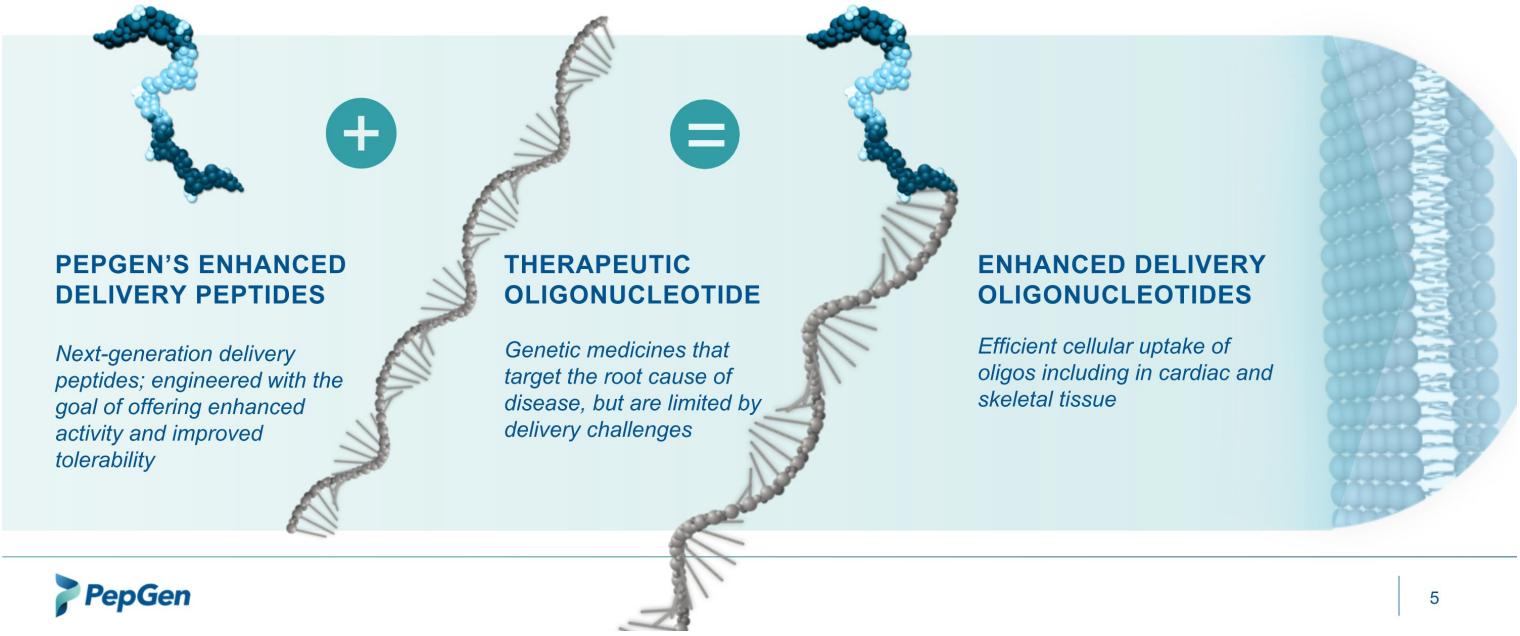
EXONDYS 51® (eteplirsen) – *Sarepta Therapeutics*

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

* Clinical data included in drug label (FDA). **Source: Sarepta MOMENTUM study update, 20 and 30 mg/kg cohort, 03May21. *** 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.

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

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.

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

PEPGEN: EXPERIENCED TEAM OF COMPANY BUILDERS, SCIENTISTS, AND CLINICIANS

Management team

JAMES MCARTHUR, PhD
(CEO & President)
imara · RACAPITAL · nightstar THERAPEUTICS · Cydan · Vtesse

NOEL DONNELLY
(CFO)
EIP PHARMA · Takeda · Shire

JAYA GOYAL, PhD
(EVP Research & Preclinical Development)
WAVE LIFE SCIENCES · Biogen

MICHELLE MELLION, MD
(SVP Clinical Development)
Fulcrum Therapeutics · Biogen · VERTEX

NIELS SVENSTRUP, PhD
(SVP Chemistry & Manufacturing)
BAYER · ascendis pharma · Cydan · Lundbeck

Board of Directors*

LAURIE KEATING, JD
(Chair)
Immuneering · IMAGO BioSciences

JOSH RESNICK, MD
(Director)
RACAPITAL

HABIB DABLE
(Director)
BAYER · ACCELERON PHARMA

CHRIS ASHTON, PhD
(Director)
SCIENCE ENTERPRISES

HEIDI HENSON
(Director)
Pardes Biosciences

* Plus Dr. McArthur (Director).

PGN-EDO51 FOR DUCHENNE MUSCULAR DYSTROPHY

DUCHENNE MUSCULAR DYSTROPHY IS A DEBILITATING, PROGRESSIVE MUSCLE-WASTING DISEASE

ROOT CAUSE OF DISEASE	EXON 51 PATIENT POPULATION*	EXON 51 THERAPEUTIC LANDSCAPE	PEPGEN'S TREATMENT APPROACH
• Caused by mutations in the dystrophin gene • Absence of dystrophin leads to muscle degeneration	~2,000 *(US)* ~3,200 *(EEA)* ~700 *(JP)*	• Exondys51® approved in US on the basis of <1% dystrophin restoration • Not approved in EEA or JP	Exon 51 skipping to drive production of a truncated, yet functional dystrophin protein

*DMD patient numbers: 15k US + 25k EEA + 5k JP whole population (range used: Crisafulli et al 2020 – 7.1/100k males; Orphanet 2021 – 4.78/100k pop). Exon 51 population 13% of total.

AN ABSENCE OF THE DYSTROPHIN PROTEIN DRIVES THE PATHOLOGIES OBSERVED IN PEOPLE WITH DMD

ROLE OF DYSTROPHIN

- Acts as a **shock absorber** to protect muscle cells from mechanical stress
- **DMD patients produce little or no dystrophin**
- In the absence of this critical protein, muscles cells are **no longer protected** from contractile forces, leading to **replacement of muscle with fatty/fibrotic tissue and muscle degeneration**

STAGES OF DISEASE

- **Early ambulatory (childhood):** difficulty walking (may walk on toes), motor delays, enlarged calves
  - **Late ambulatory (late childhood):** walking, climbing stairs, rising from floor becomes increasingly difficult, cognitive impairment may become apparent
    - **Early non-ambulatory (early adolescence):** full-time wheelchair use, upper limb function impaired
      - **Late non-ambulatory (adolescence/adulthood):** life-threatening heart and respiratory conditions common, DMD is typically fatal by early adulthood

We believe dystrophin restoration is a compelling therapeutic strategy – levels of >10% of normal may halt, slow or even reverse disease progression

Sources: Busby et al, *Lancet Neurol* 2010;9:77-93; https://www.parentprojectmd.org/care/care-guidelines/by-stage/

PGN-EDO51 WAS ENGINEERED TO TRANSFORM THE TREATMENT OF DMD AMENABLE TO EXON 51 SKIPPING

PGN-EDO51 DEVELOPMENT DATA SUMMARY

- **Highest level of exon skipping and oligonucleotide delivery in humans** following a single dose*
- Generally well-tolerated

- **Greatest exon skipping potency at tolerable target dose levels** compared to any approved exon 51 therapeutic or known development candidate

- High levels of dystrophin expression and exon skipping in *mdx* mouse model

* 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 on both head-to-head and cross-trial comparisons with other exon 51 skipping therapeutics that have been assessed in NHP.

PepGen

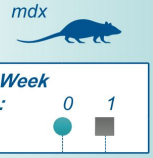
PRECLINICAL DATA

THE ACTIVITY OF OUR EDO PLATFORM IN DMD HAS BEEN EVALUATED IN MULTIPLE PRECLINICAL MODELS

		Species	Key readouts observed
Non-GLP pharmacology studies	**Patient cells** *PGN-EDO51*	 *DMD patient*	• High levels of exon 51 skipping
	Single & repeat dose *PGN-EDO23*	 *mdx*	• Normalization of serum creatine kinase • High levels of exon 23 skipping and dystrophin restoration • Accumulation of exon skipping and dystrophin levels with repeat dosing
	Single dose *PGN-EDO51*	 *WT*	• High levels of exon 51 skipping
	Repeat dose *PGN-EDO51*	 *WT*	• High levels of exon 51 skipping • Accumulation of exon skipping levels with repeat dosing

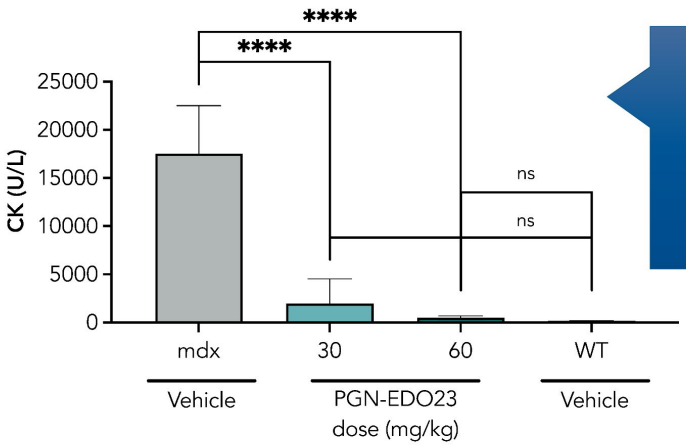
MDX MICE: A SINGLE DOSE OF PGN-EDO23 WAS OBSERVED TO NORMALIZE CREATINE KINASE, A MARKER OF MUSCLE DAMAGE

PGN-EDO23
(murine analogue of PGN-EDO51)



PGN-EDO23 dose
Serum analysis

SERUM CREATINE KINASE



This result suggests that PGN-EDO23 potentially **restored muscle cell integrity** following a single dose at tolerable levels

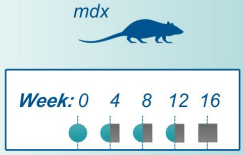
PGN-EDO23 utilizes the **same EDO delivery peptide** as our clinical candidate

Protocol: peptide-PMO conjugate and a saline control were administered intravenously (IV) to *mdx* and WT mice; serum creatine kinase measured 7 days after injection. Mean ± SD; **** = p≤0.0001; **ns** = p≥0.05; n = 3 for control groups and 5 for treated group.

MDX MICE: SIGNIFICANT INCREASE IN DYSTROPHIN OBSERVED WITH REPEAT DOSING

PGN-EDO23
(murine analogue of PGN-EDO51)

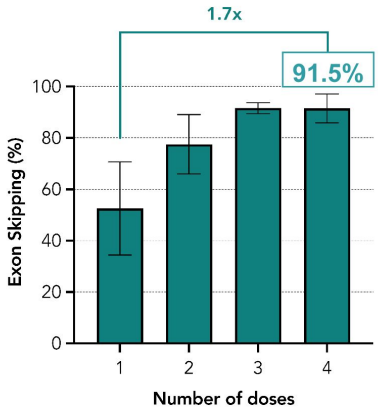
mdx



PGN-EDO23 dose
Tissue analysis

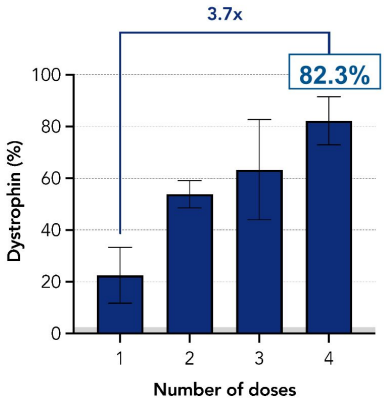
EXON SKIPPING

Biceps, 30 mg/kg, Q4W



DYSTROPHIN

Biceps, 30 mg/kg, Q4W



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

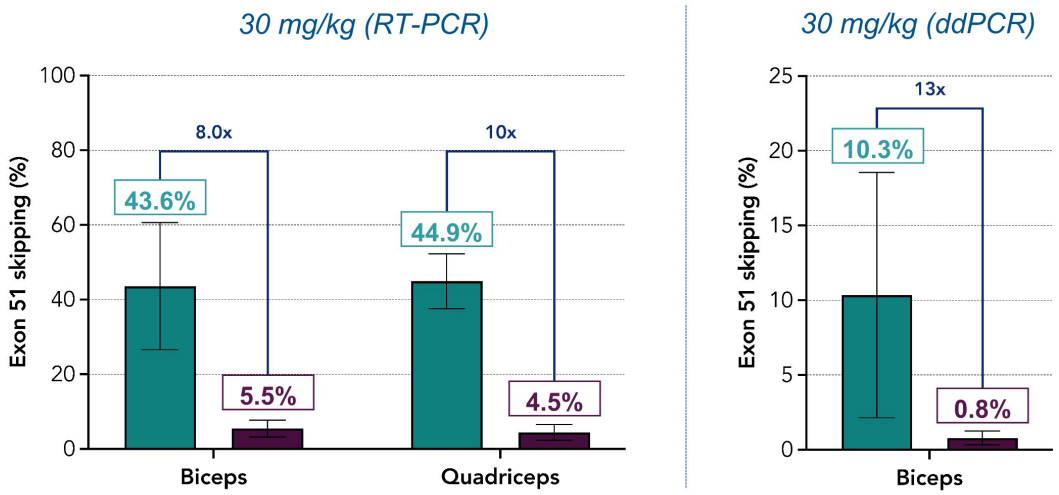
Protocol: *mdx* mice were dosed 2, 3 or 4 doses, with 4-week intervals between doses. Tissue samples were collected 4 weeks post-each dose at time points indicated. Exon skipping was performed by RT-PCR and dystrophin evaluation by western blot. Graph is presented as mean ± SD; *n* = 4-5 per cohort;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

NHP: MARKEDLY HIGHER SINGLE DOSE EXON SKIPPING LEVELS OBSERVED FOR PGN-EDO51 COMPARED TO R_6G-PMO

PGN-EDO51
R_6G-PMO51

PGN-EDO51
WT
Week: 0 1
PPMO dose
Tissue analysis

EXON SKIPPING

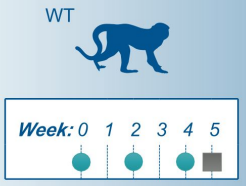


Protocol: PGN-EDO51 and R_6G-PMO were administered to NHP by IV infusion over 30 min at the dose indicated (n=3). Biopsies taken 7 days post single dose. Study not powered for statistical significance. Data shown as mean ± SD; n = 3 per group. R_6G-PMO51 is believed to be structurally equivalent to SRP-5051.

NHP: Q2W REPEAT DOSE EXON SKIPPING LEVELS OF >70% OBSERVED IN SKELETAL MUSCLES AT 30 MG/KG

PGN-EDO51

WT

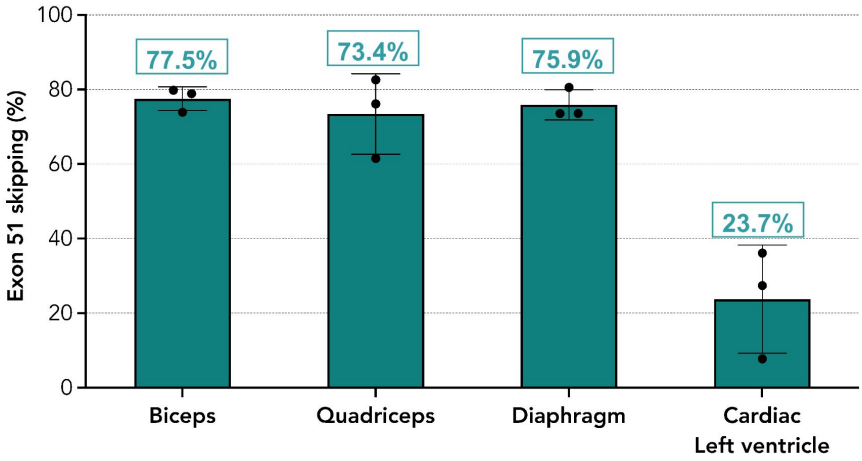


PPMO dose

Tissue analysis

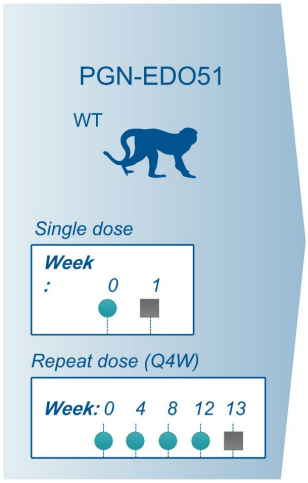
EXON SKIPPING

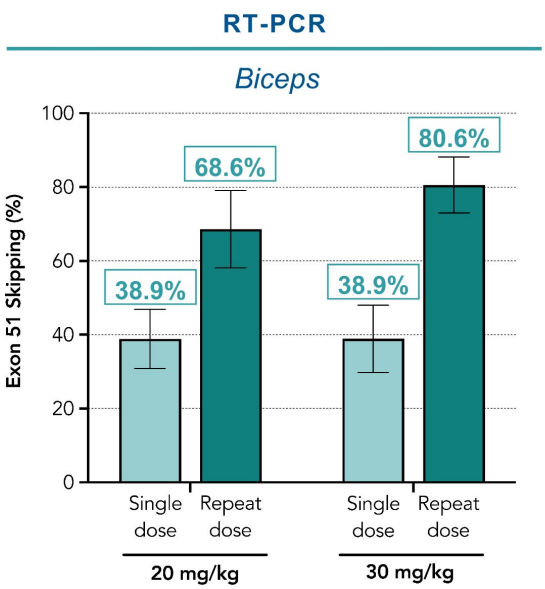
30 mg/kg

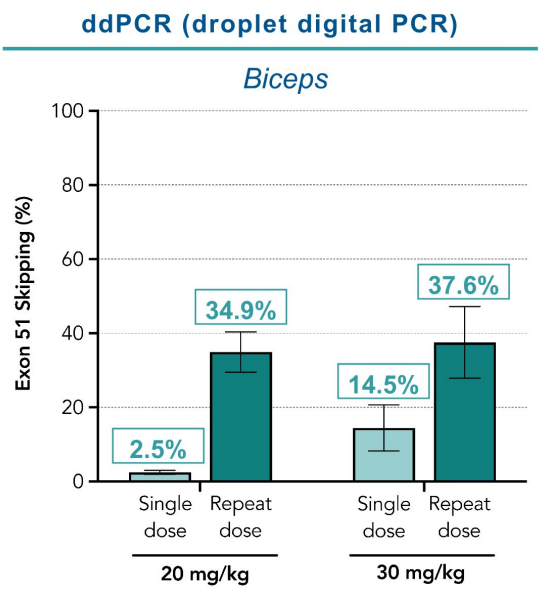


Protocol: PGN-EDO51 and R_6G-PMO were administered to NHP by IV infusion over 30 min at the doses indicated (n=3). Q2W, three doses administered, saline control. Tissues were harvested 7 days after final administration. Shown as mean ± SD; n = 3 per group. Study was not powered for statistical significance.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

NHP: EXON SKIPPING LEVELS ACCUMULATED WITH Q4W REPEAT DOSE ADMINISTRATION OF PGN-EDO51 BY BOTH RT-PCR AND ddPCR







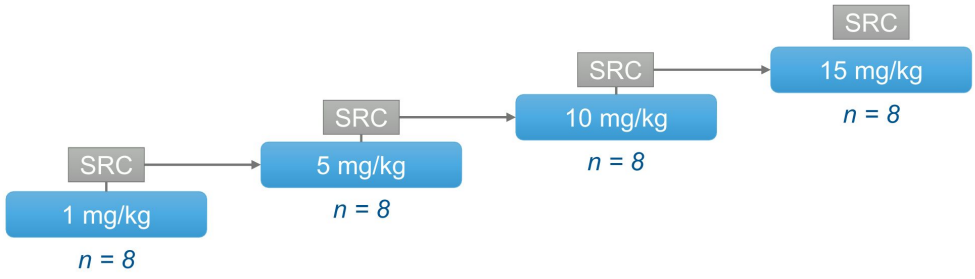
NHP protocol: Single (30 min) or repeat (60 min) IV doses with PGN-EDO51 were performed in male NHP. For repeat evaluation, NHP received 4 doses with 4-week intervals between doses. Tissue samples were collected 1-week post-final dose as indicated on graphs. Exon skipping was performed by RT-PCR and ddPCR. Graph is presented as mean ± SD; *n* = 3-8 per group.

PepGen

PH 1
CLINICAL DATA

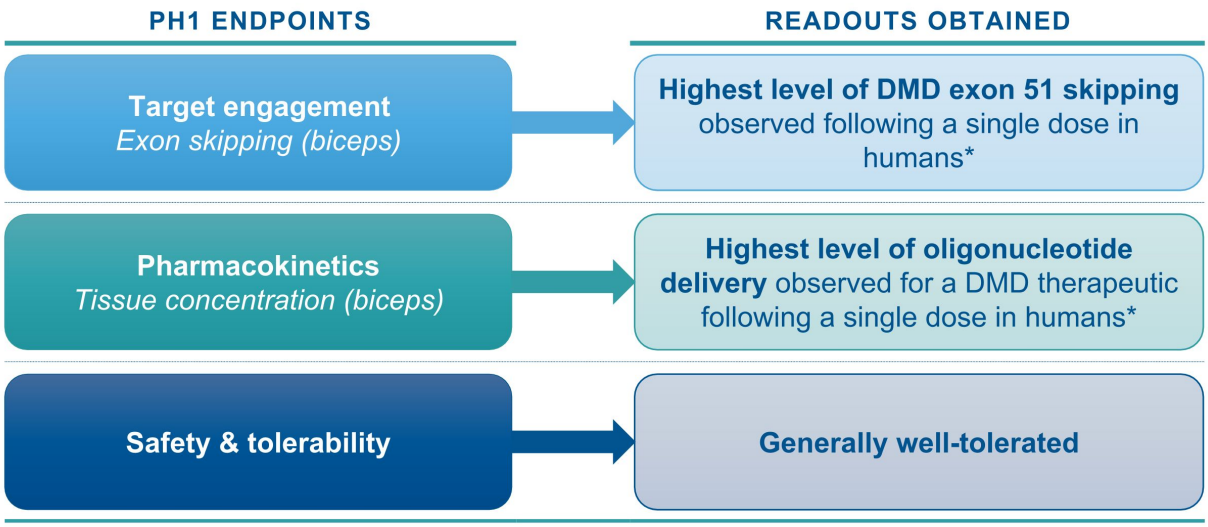
HV: WE HAVE COMPLETED A SINGLE ASCENDING DOSE PH1 TRIAL OF PGN-EDO51 IN HEALTHY NORMAL VOLUNTEERS

PH1 HEALTHY VOLUNTEER (HV) TRIAL SUMMARY

Overview	• **Study population:** Healthy adult males (n = 32, 3:1 PGN-EDO51:placebo) • **Dosing:** Single dose, i.v. administration • **Placebo control** • Biceps biopsies conducted on **Day 10 and Day 28**
Trial summary	

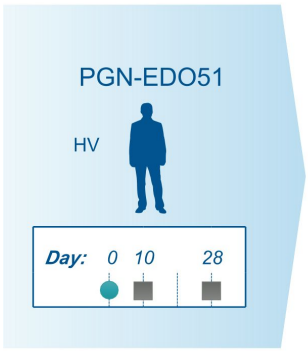
SRC = safety review committee.

HV: HIGHEST LEVELS OF OLIGO DELIVERY & EXON 51 SKIPPING OBSERVED, SUPPORTING FURTHER DEVELOPMENT OF PGN-EDO51



* 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.

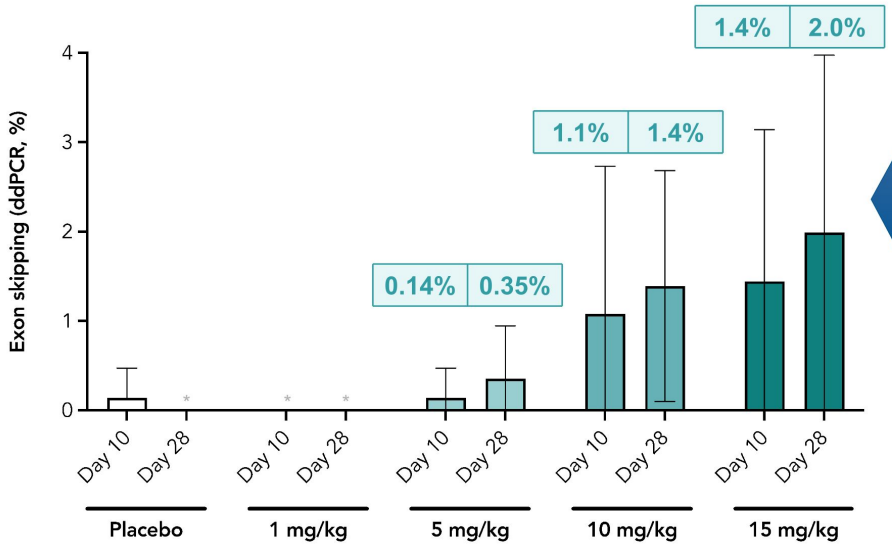
HV: HIGHEST LEVELS OF EXON 51 SKIPPING OBSERVED IN HUMANS FOLLOWING A SINGLE DOSE



PGN-EDO51 dose

Biceps biopsy

EXON SKIPPING (BICEPS)

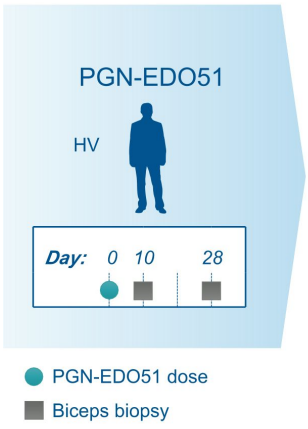


These results further support our belief that repeat dosing of PGN-EDO51 may lead to **accumulation of skipped transcript and dystrophin** in DMD patients

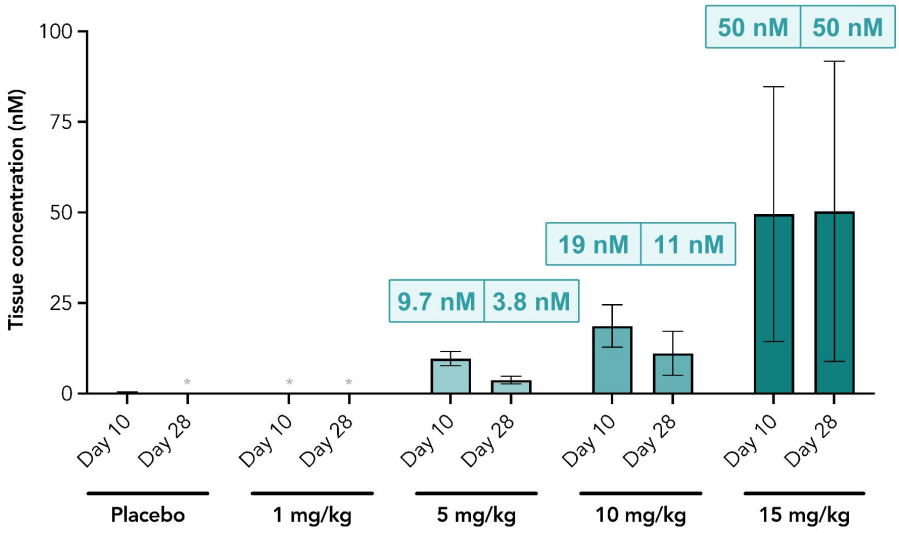
PepGen

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 were administered by IV infusion at doses indicated. Participants were followed for 28 day period following dose administration to evaluate safety, tolerability, pharmacokinetics (PK), and pharmacodynamics (PD). Needle biopsies of biceps muscle were taken on Day 10 and Day 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). Asterix indicates that values were under the lower limit of quantitation. 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.

HV: HIGH, PERSISTENT TISSUE CONCENTRATIONS OF OLIGONUCLEOTIDE WERE OBSERVED



TISSUE CONCENTRATION (BICEPS)



PepGen

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 were administered by IV infusion at doses indicated. Participants were followed for 28 day period following dose administration to evaluate safety, tolerability, PK, and PD. Needle biopsies of biceps muscle were taken on Day 10 and Day 28. Tissue concentration measured by ELISA. Shown as mean ± SD; n = 6 PGN-EDO51: 2 Placebo per cohort (n = 5 for D10 at 15 mg/kg. Asterix indicates that values were under the lower limit of quantitation.

HV: PGN-EDO51 WAS GENERALLY WELL-TOLERATED IN PH1 SAD TRIAL

SAFETY & TOLERABILITY SUMMARY

At **10 mg/kg**:

- All participants (n = 6) completed the study with **no discontinuations**.
- All related treatment-emergent adverse events (TEAEs) were assessed as **mild and resolved without any intervention**.
- Serum cystatin C, the recommended biomarker to assess renal function in DMD, **did not change**.
- There was **no evidence of hypomagnesemia**.

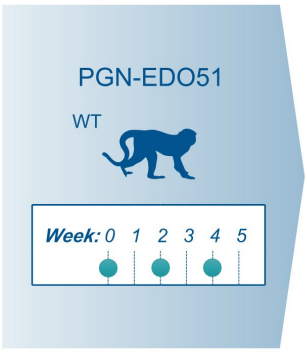
HV: TEAEs MILD AND RESOLVED WITHOUT INTERVENTION AT SELECTED CLINICALLY-RELEVANT DOSES

PH1 TRIAL SAFETY & TOLERABILITY SUMMARY

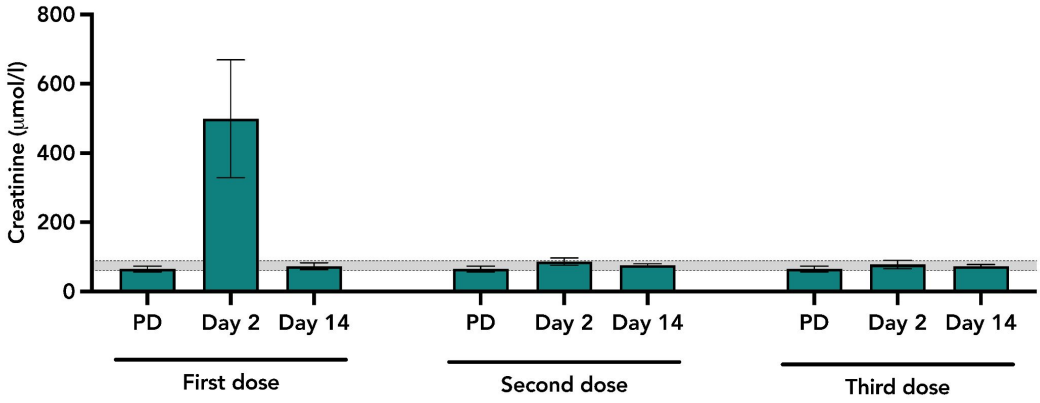
Healthy Volunteers (HV) with ≥1 AE, n (%)	Placebo (n=8)	Cohort A: 1 mg/kg (n=6)	Cohort B: 5 mg/kg (n=6)	Cohort C: 10mg/kg (n=6)	Cohort D: 15 mg/kg (n=6)	PGN-EDO51 Total (n=24)
Any AE	4 (50)	4 (66.7)	2 (33.3)	5 (83.3)	6 (100)	17 (70.8)
Related to study drug	1 (12.5)	2 (33.3)	0	4 (66.7)	6 (100)	12 (50)
Serious AE related to study drug	0	0	0	0	1 (16.7)	1 (4.2)
AE leading to discontinuation	0	0	0	0	0	0
AE leading to death	0	0	0	0	0	0
Number of Related TEAEs by CTCAE v5.0 grading*						
Grade 1 (Mild)	1	1	0	7	12	20
Grade 2 (Moderate)	0	1	0	0	3	4
Grade 3 (Severe)	0	0	0	0	1	1

* No Grade 4 or 5 recorded; There were transient, reversible changes in kidney biomarkers that resolved without intervention at higher doses. At 15 mg/kg there was one non-life threatening serious adverse event (SAE) related to changes in kidney biomarkers that were transient and reversible. This HV was admitted to the hospital for less than 24 hours, received hydration and then was re-admitted to the Phase 1 unit and completed the study. Transient mild (Grade 1) to moderate (Grade 2) hypomagnesemia was observed in two participants at the 15 mg/kg dose and did not require any intervention. In light of higher than anticipated oligo levels and exon skipping levels in muscle observed at 5 mg/kg and 10 mg/kg, further does escalation was not deemed necessary by sponsor. Under this Phase 1 protocol any non-life-threatening SAE was considered a dose-limiting toxicity (DLT), however study was not halted by the SRC nor put on hold by Health Canada.

IN NHP REPEAT DOSE STUDY, KIDNEY BIOMARKER ELEVATIONS WERE REDUCED AFTER FIRST DOSE OF PGN-EDO51



REPEAT-DOSE SERUM CREATININE LEVELS *(HIGH-DOSE COHORT)*



We believe that these results support the potential tolerability of PGN-EDO51 with repeat dosing

PD = pre-dose. Protocol: PGN-EDO51 was administered to NHP by IV infusion over 30 min at a given dose level (n=3). Q2W, three doses administered, saline control. Shown as mean ± SD; n = 3 per group. Study was not powered for statistical significance. Grey bar shows normal range.

PepGen

PH 2
CLINICAL PLANS

FOLLOWING ENCOURAGING PH1 HV DATA IN 2022, WE ANTICIPATE OPENING TWO PH2 DMD MAD PATIENT STUDIES IN 2023

2022
Completed

- **2Q:** First HV dosed in Ph1 trial ✓
- **3Q:** **Ph1 data:**
  - **Highest single dose levels of exon skipping & oligo delivery*** ✓
  - PGN-EDO51 generally well-tolerated
- **4Q:** Completion of Ph2-enabling tox studies ✓

2023
Anticipated

- **1H:** Initiation of **CONNECT1-EDO51 Ph 2** patient study in Canada

Connect 1
EDO51

- **2H:** Initiation of **CONNECT2-EDO51 Ph2** global patient study

Connect 2
EDO51

2024
Anticipated

- Ph2 data in DMD patients:
  - **Dystrophin**
  - **Exon skipping**
  - **Safety**

* 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.

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

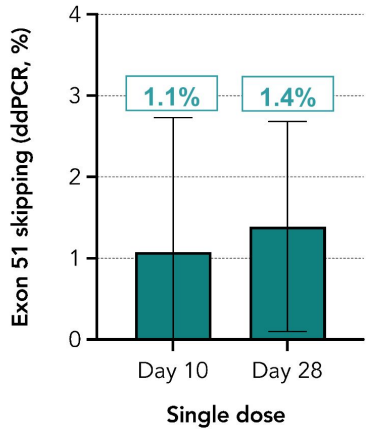
Dystrophin, exon skipping and safety data anticipated in 2024

- 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**

MARKED INCREASE IN EXON SKIPPING OBSERVED IN NHP WITH Q4W REPEAT DOSING REGIMEN

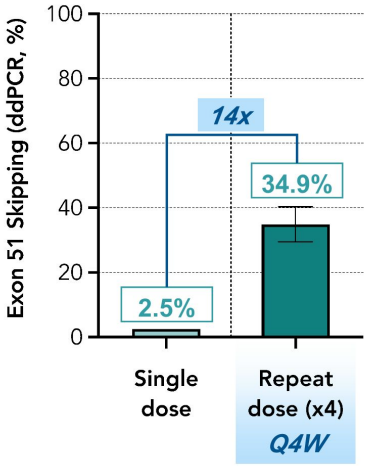
HV SINGLE DOSE

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 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

PGN-EDODM1 FOR MYOTONIC DYSTROPHY TYPE 1 (DM1)

MYOTONIC DYSTROPHY TYPE 1 IS A PROGRESSIVE, DEBILITATING NEUROMUSCULAR DISORDER WITH GREAT UNMET NEED

ROOT CAUSE OF DISEASE	PATIENT POPULATION**	THERAPEUTIC LANDSCAPE	PEPGEN'S TREATMENT APPROACH
• Due to a CTG repeat expansion mutation in the *DMPK* gene • Leads to downstream dysregulation of a broad set of proteins	~40,000 *(US)* ~75,000 *(EEA)* ~15,000 *(JP)*	• No approved disease-modifying therapeutics • Standards of care focused on symptom management	PGN-EDODM1 binds *DMPK* transcript, reducing toxic foci and liberating MBNL1 to restore physiological splicing

* CNS symptoms include cognitive deficits, excessive daytime sleepiness and behavioral impairments.
** Johnson et al 2021, NORD (1 in 8,000 prevalent population)

PGN-EDODM1 WAS ENGINEERED TO LIBERATE MBNL1 FROM *DMPK*-CUG_{exp} TOXIC FOCI AND CORRECT MIS-SPLICING

PGN-EDODM1 DEVELOPMENT DATA SUMMARY

- Correction of mis-splicing observed in preclinical models with **long and short CTG repeats**
- **Reduction of toxic foci and liberation of MBNL1** observed in patient cells
- In DM1 mouse model, **robust mis-splicing correction and reversal of myotonia** observed with a single dose; durable mis-splicing corrections observed **through 24 weeks**
- Observed to be **well-tolerated through 90 mg/kg in NHP single-dose GLP toxicology studies**
- **Not designed to degrade** CUG-containing transcripts, including *DMPK* – a potentially important safety feature
- **No impact observed on other transcripts containing >10 CUG repeats**
- EDO technology observed to enable:
  - Delivery of **pharmacologically active** levels of oligonucleotide to **muscle in humans**
  - Delivery of oligonucleotides to the **CNS** in NHPs

DM1 IS A MULTI-SYSTEMIC DISEASE THAT HAS A SIGNIFICANT IMPACT ON QUALITY OF LIFE

Musculoskeletal: Myotonia (a temporary inability to relax a muscle after contraction), muscle weakness & wasting

Cardiac: Conduction defects

Respiratory: breathing difficulties, sleep apnea

GI: Dysphagia (difficulty swallowing), constipation, IBS

CNS: Cognitive impairments, behavioral / psychologic disorders, excessive daytime sleepiness

Vision: Early-onset cataracts, retinal damage

Endocrine: Thyroid dysfunction, diabetes

Other pathologies: skin, immune, reproductive, increased cancer risk

QoL considerations: **Shortened lifespan:** ~45 – 55 years for more severe forms of disease, 60+ for milder forms; **genetic anticipation:** disease severity may increase, and age of onset may decrease in subsequent generations

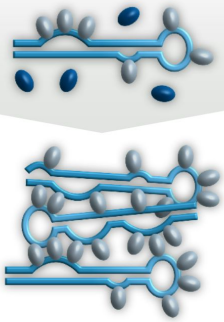
We believe that a potential therapeutic approach with a broad biodistribution profile may allow for the treatment of such multi-systemic pathologies

Sources: https://www.mda.org/disease/myotonic-dystrophy/signs-and-symptoms; www.musculardystrophy.com; Mathieu J et al, Neurology. 1999;52:1658–62

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

DM1 PATHOLOGY

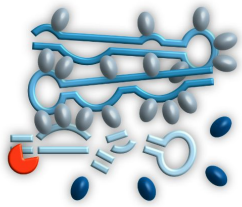
***DMPK* transcript CUG repeat hairpin loops bind MBNL1 and form cross-linked foci**



Expanding toxic foci trap more MBNL1

DMPK KNOCKDOWN

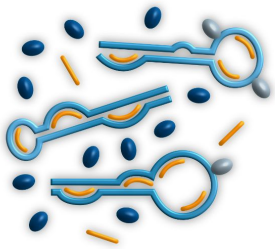
ASO / siRNA degrade *DMPK* transcript to reduce toxic foci



- 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

PepGen

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 IS DIFFERENTIATED OVER OTHER APPROACHES AS A POTENTIAL TREATMENT FOR DM1

KEY ADVANTAGES OF OUR PGN-EDODM1 APPROACH

- PGN-EDODM1 targets MBNL1 binding to *DMPK* transcripts
  - This may provide **greater tolerability** – no degradation of *DMPK* or risk of haploinsufficiency
  - Avoids potential **disconnect between DMPK knockdown and correction of mis-splicing**
- PGN-EDODM1 PMO does not require RISC or RNAseH proteins – **potentially better accessibility to toxic aggregated *DMPK*-CUG$_{exp}$ nuclear foci**
- **Considerably higher levels of oligonucleotide delivery observed in human muscle tissue** when compared to competing approaches in DM1*
- EDO platform has demonstrated **successful delivery of therapeutic PMOs to the nucleus**

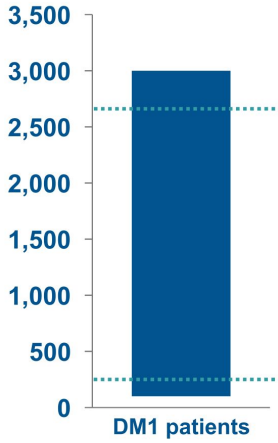
* Comparative statements are based on cross-trial comparisons with publicly-available data for other approaches

THE PHARMACOLOGY OF PGN-EDODM1 HAS BEEN EVALUATED IN MULTIPLE PRECLINICAL MODELS

		Species	Key readouts observed
Non-GLP pharmacology studies	**Patient cells** *PGN-EDODM1*	 *DM1 patient*	• Reduction in nuclear foci, liberation of MBNL1 • Correction of downstream transcript mis-splicing
	Single dose *PGN-EDODM1*	 *HSA^{LR}*	• Correction of downstream transcript mis-splicing • Normalization of myotonia
	Duration of effect *PGN-EDODM1*	 *HSA^{LR}*	• Correction of downstream transcript mis-splicing for at least 24 weeks post-dose
Non-GLP dose-range finding (DRF) studies	**Single dose** *PGN-EDODM1*	 *WT*	• Doses identified for GLP toxicology studies
	Repeat dose *PGN-EDODM1*	 *WT*	• No change observed in *DMPK* levels

PGN-EDODM1 ACTIVITY HAS BEEN OBSERVED IN PRECLINICAL MODELS WITH A WIDE RANGE OF CTG REPEATS

CTG REPEAT LENGTH



PRECLINICAL MODELS ASSESSED

DM1 patient cells: 2,600 CTG repeats
Reduction of nuclear foci and correction of downstream transcript mis-splicing observed

Full spectrum of patient CTG repeats addressed in preclinical testing

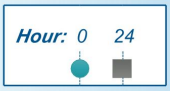
HSA^{LR} mouse model: 220 – 250 CTG repeats
Correction of downstream transcript mis-splicing and normalization of myotonia observed

IN VITRO: PGN-EDODM1 REDUCED PATHOGENIC NUCLEAR FOCI

FOCI REDUCTION

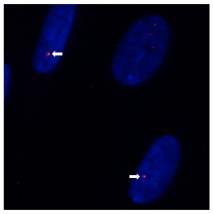
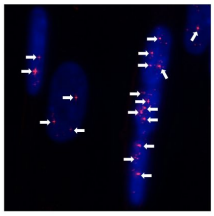
PGN-EDODM1

DM1 patient cells
(2,600 CTG repeats)

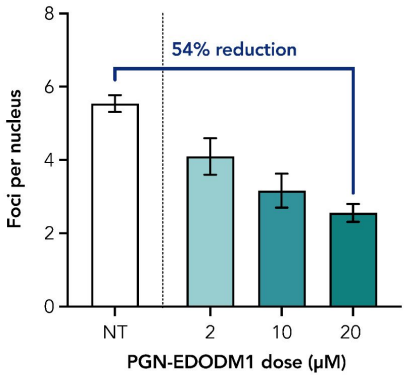


PGN-EDODM1 dose

Analysis

Not treated (NT)	PGN-EDODM1	PMO
		
Foci observable in patient cells	**Robust reduction** in number of foci following PGN-EDODM1 treatment	**No foci reduction with unconjugated PMO**

Foci quantification



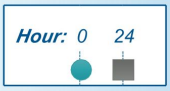
Immortalized myoblasts from healthy individual or DM1 patient with 2600 CTG repeats were cultured then differentiated for 4 days into myotubes. Treatment with PMO or peptide-PMO conjugates at concentrations given. Cells were harvested for analysis 24h after treatment. Visualisation with FISH and immunofluorescence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

IN VITRO: PGN-EDODM1 LIBERATED FOCI-BOUND MBNL1

FOCI REDUCTION & LIBERATION OF MBNL1

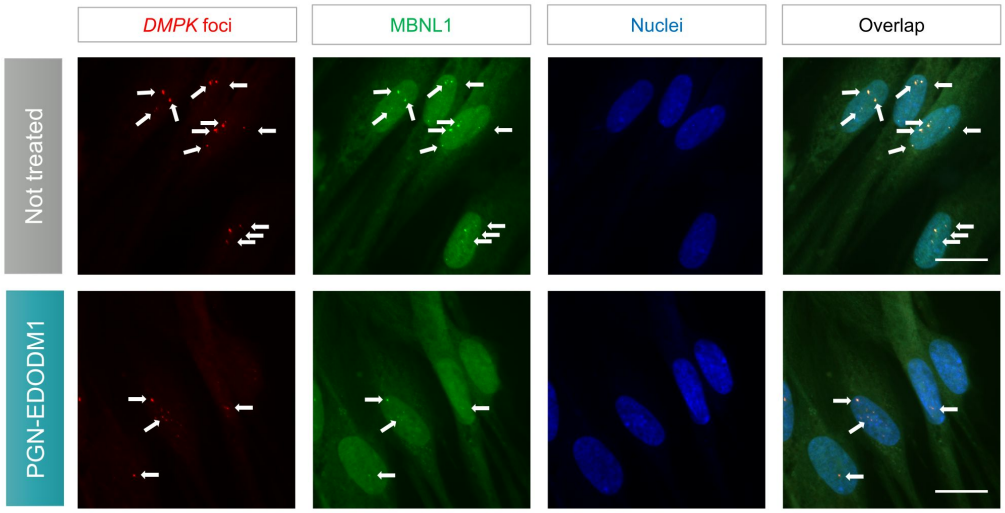
PGN-EDODM1

DM1 patient cells
(2,600 CTG repeats)



PGN-EDODM1 dose

Analysis

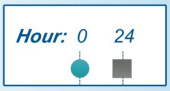


Immortalized myoblasts from healthy individual or DM1 patient with 2600 CTG repeats were cultured then differentiated for 4 days into myotubes. Cells were treated with PGN-EDODM1 and harvested for analysis 24h after treatment. Visualisation with FISH and immunofluorescence microscopy.

IN VITRO: PGN-EDODM1 CORRECTED DOWNSTREAM TRANSCRIPT MIS-SPLICING EVENTS

PGN-EDODM1

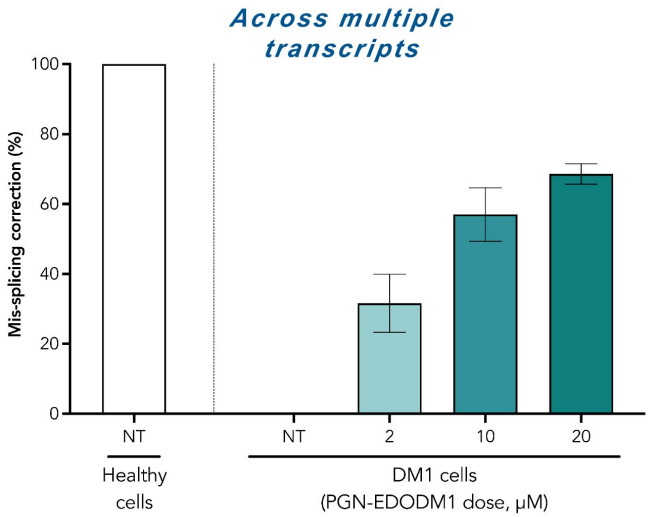
DM1 patient cells
(2,600 CTG repeats)



PGN-EDODM1 dose

Analysis

MIS-SPLICING CORRECTION



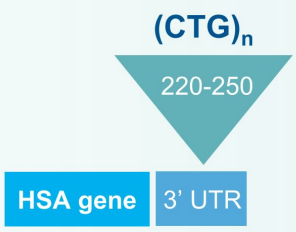
Almost **70% mis-splicing correction** observed at highest dose level

PepGen

Immortalized myoblasts from healthy individual or DM1 patient with 2600 CTG repeats were cultivated then differentiated for 4 days. Treatment with PGN-EDODM1 at concentrations given. Cells were harvested for analysis 24h after treatment. RNA isolation, RT-PCR and capillary electrophoresis (QIAxcel) analysis was performed. Mean ± SD; n = 5 per group.

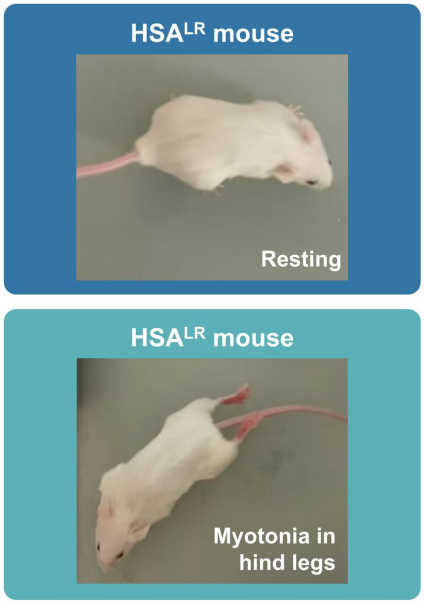
HSA^{LR} MOUSE DISPLAYS MOLECULAR AND FUNCTIONAL DM1 PHENOTYPE

REPEAT EXPANSION IN HSA GENE UTR



DM1 ASSOCIATED ABNORMALITIES

- Skeletal muscle specific CUG_{exp}
- MBNL1 sequestration in the nucleus
- Downstream mis-splicing events
- Myotonia



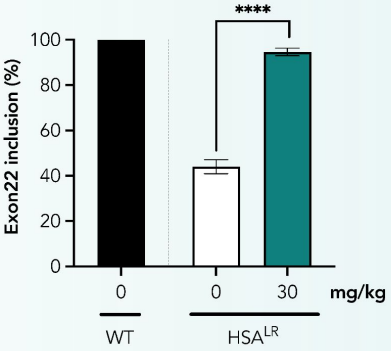
Source: Mankodi et al., SCIENCE 2000.

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

CORRECTION OF MIS-SPLICING

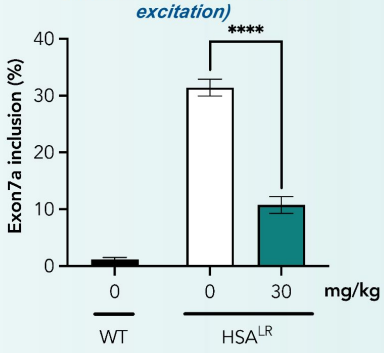
Atp2a1

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



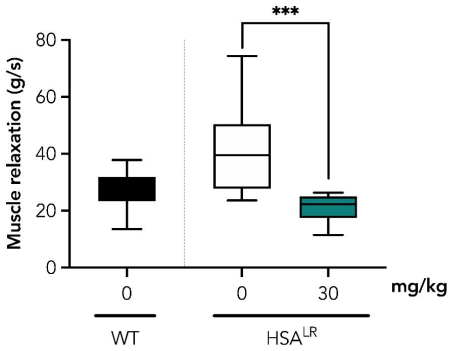
Clcn1

(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

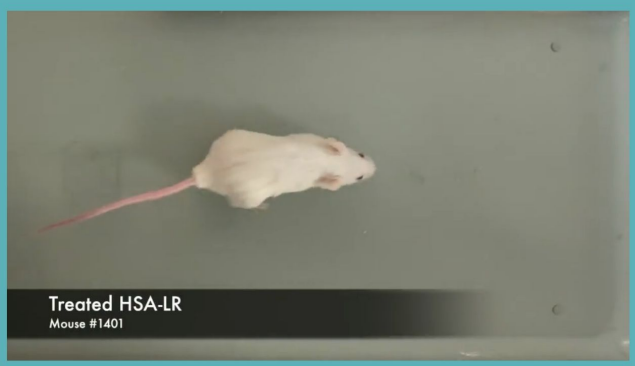
PepGen

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.

HSA^{LR}: SPLICING CORRECTION TRANSLATED TO PHENOTYPIC IMPROVEMENT OF DM1 MICE TREATED WITH PGN-EDODM1

UNTREATED HSA^{LR}

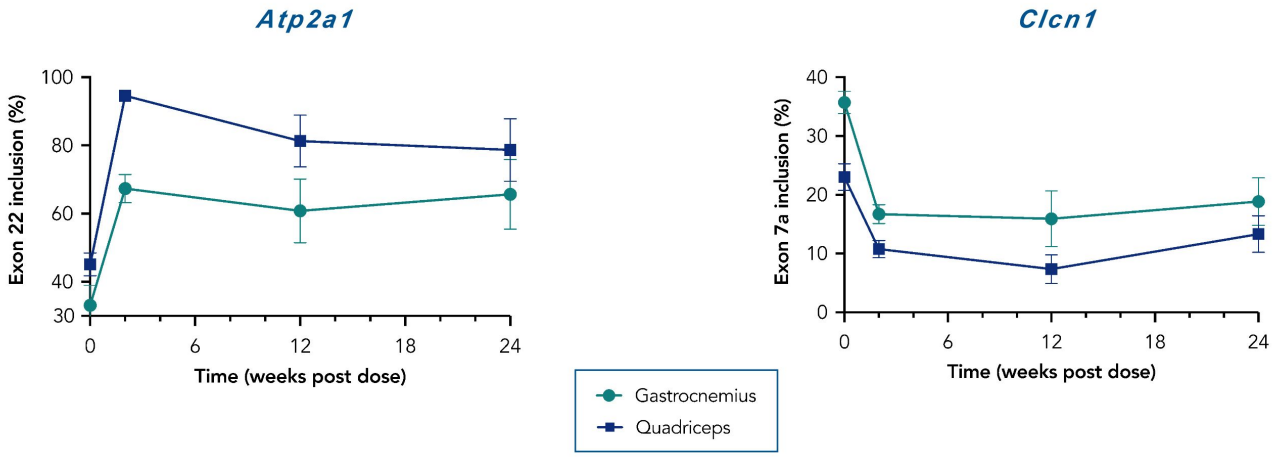
TREATED HSA^{LR}



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.

HSA^{LR}: SINGLE DOSE TREATMENT OF PGN-EDODM1 LED TO DURABLE IMPROVEMENTS IN SPLICING THROUGH 24 WEEKS

CORRECTION OF MIS-SPLICING

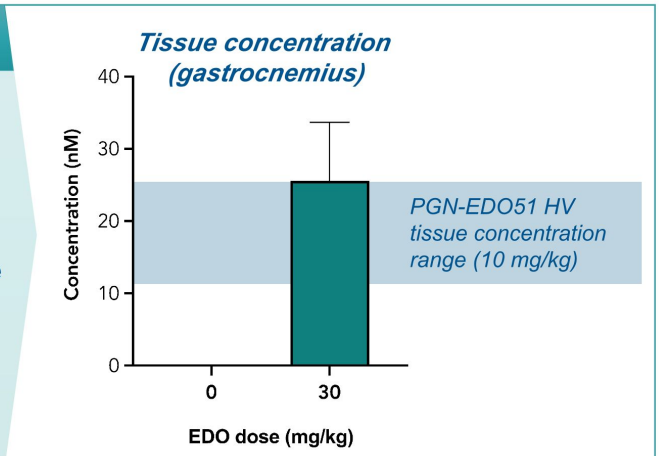


Protocol: PGN-EDODM1 was administered intravenously (IV) to WT and HSA^{LR} mice at 30 mg/kg; gastrocnemius muscle harvested 2 (n=8), 12 (n=8) or 24 (n=5) weeks post-administration; graph plotted as mean ±SEM; n = 7 for 0 timepoint, 8 for 2- and 12-week timepoints; 5 for 24-week timepoint.

HUMAN: PGN-EDO51 TISSUE CONCENTRATIONS WERE COMPARABLE TO THOSE ACHIEVED IN HSA^{LR} MOUSE MODEL WITH PGN-EDODM1

HSA^{LR} MOUSE

Robust mis-splicing correction and reversal of myotonia were observed after a **single dose of 30 mg/kg**



PGN-EDO51 Ph1

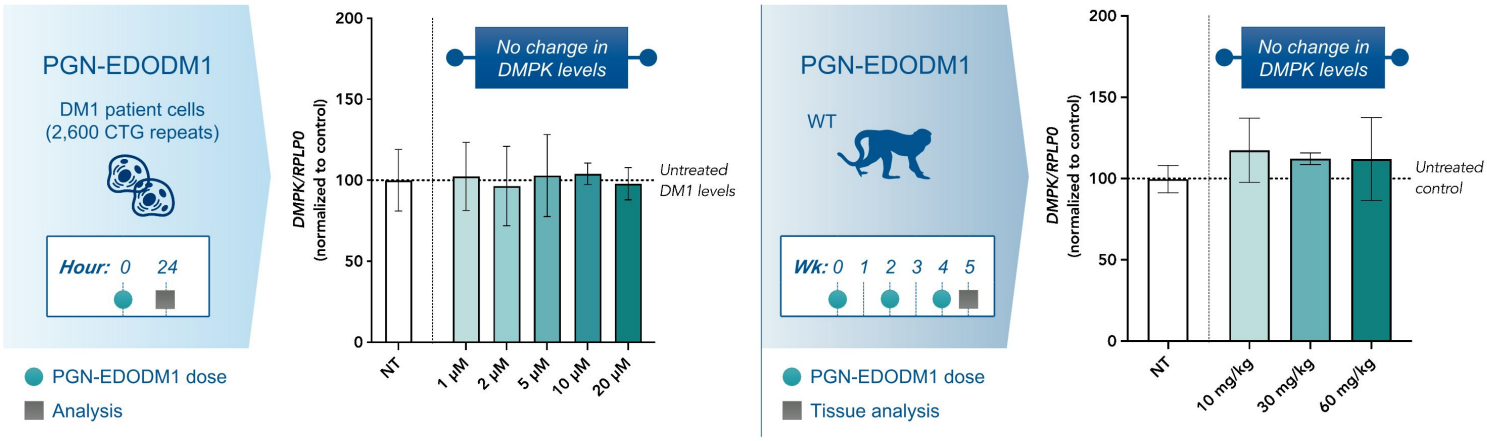
Following a single 10 mg/kg dose of PGN-EDO51 in our Ph1 HV trial, **tissue concentrations were similar** to those measured for PGN-EDODM1 at 30 mg/kg in HSA^{LR} mouse

We believe that PGN-EDODM1 has the potential to achieve concentrations in DM1 patients that could lead to clinically-meaningful outcomes, supporting further development of this candidate

Protocol: PGN-EDODM1 was administered IV to HSA^{LR} mice at 30 (n=8) mg/kg against a wild-type (WT) saline control (n=8). Tissue concentration in the gastrocnemius muscle assessed by HPLC.

IN VITRO + NHP: OUR STERIC BLOCKING MECHANISM OF ACTION WAS NOT OBSERVED TO TARGET *DMPK* FOR DEGRADATION

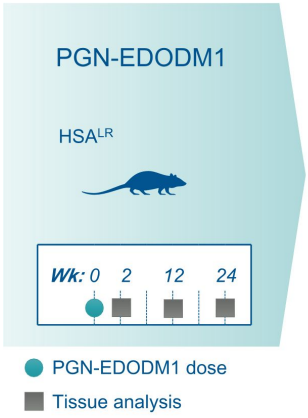
DMPK TRANSCRIPT LEVELS



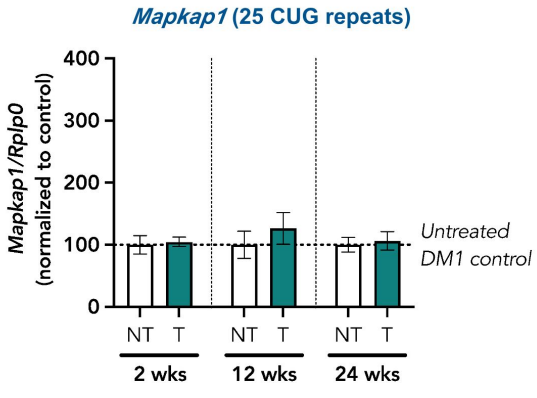
***DMPK* transcript levels remained unchanged across multiple preclinical models**

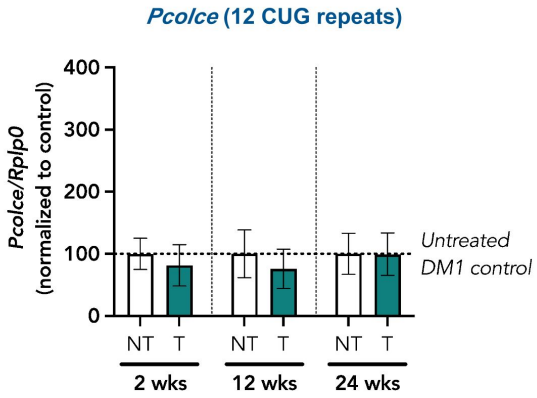
In vitro: Immortalized myoblasts from DM1 patient with 2600 CTG repeats were differentiated for 4 days to myotubes and treated for 24h hours with PGN-EDODM1 from 1-20 µM. NT = not treated. *DMPK* transcript levels were evaluated by qPCR and normalised to *RPLP0*. Graphs plotted as mean ± SD, *n*=3-4.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

HSA^{LR}: NO IMPACT OBSERVED ON OTHER TRANSCRIPTS CONTAINING >10 CUG REPEATS



TRANSCRIPT LEVELS IN HSA^{LR} MOUSE (QUADS)





No evidence of off-target effects in human cell, mouse and NHP studies with PGN-EDODM1

Protocol: PGN-EDODM1 was administered once intravenously (IV) to HSA^{LR} mice at 30 mg/kg; quadriceps muscle harvested 2,-12 or 24 weeks post-administration; graph plotted as mean ± SD; *n*=7 for 0 timepoint, 8 for 2- and 12-week timepoints; 5 for 24-week timepoint. Transcript levels measured by qPCR and normalized to *Rplp0*. NT = not treated.

NHP: IND-ENABLING STUDIES DOSED THROUGH 90 MG/KG, RESULTS SUPPORT ANTICIPATED INITIATION OF PATIENT TRIAL IN 1H23

PGN-EDODM1

Single-dose

NHP SINGLE-DOSE GLP TOXICOLOGY STUDIES

SUMMARY OF FINDINGS

- PGN-EDODM1 was well-tolerated through 90 mg/kg
- No adverse effects on kidney function
- No adverse effects on liver function
- No adverse effects on cardiovascular function

PepGen

Protocol: PGN-EDODM1 was administered as a single dose by intravenous infusion of 60 minutes to cynomolgus monkeys at a range of dose levels, and safety and tolerability endpoints were assessed.

PepGen

CLINICAL PLANS

BUILDING ON A ROBUST PRECLINICAL DATASET, WE ANTICIPATE ADVANCING TO THE CLINIC IN 2023

2022
Completed

- **2Q:** NHP dose range-finding study ✓
- **3Q:** **Clinical potential supported by PGN-EDO51 trial** ✓
- **2H:** **Ph1-enabling studies** ✓

2023
Anticipated

- **1H:** Initiation of **FREEDOM-DM1 Ph1** SAD DM1 patient trial

Freedom DM1

2024
Anticipated

- DM1 patient data:
  - **Functional assessments**
  - **Correction of mis-splicing**
  - **Safety**
- Initiation of **Ph2 MAD** DM1 patient trial

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

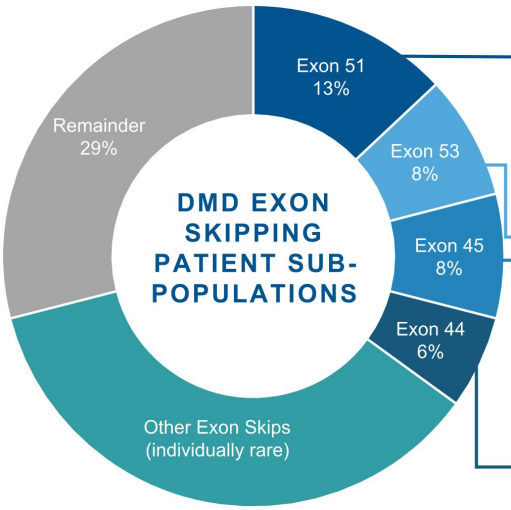
PHASE 2
Multiple ascending dose

- 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

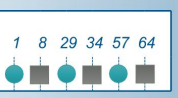
- *High levels of exon skipping observed in human cells*

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.

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

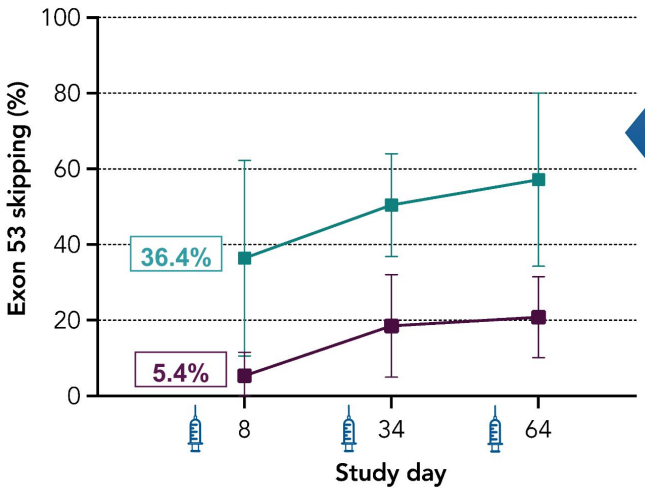
PGN-EDO53
R_6G-PMO53

PGN-EDO53
WT



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.

PGN-EDO45: HIGH, DOSE-DEPENDENT LEVELS OF EXON 45 SKIPPING WERE OBSERVED IN WILD-TYPE HUMAN MYOBLASTS

PGN-EDO45
R_6G-PMO45

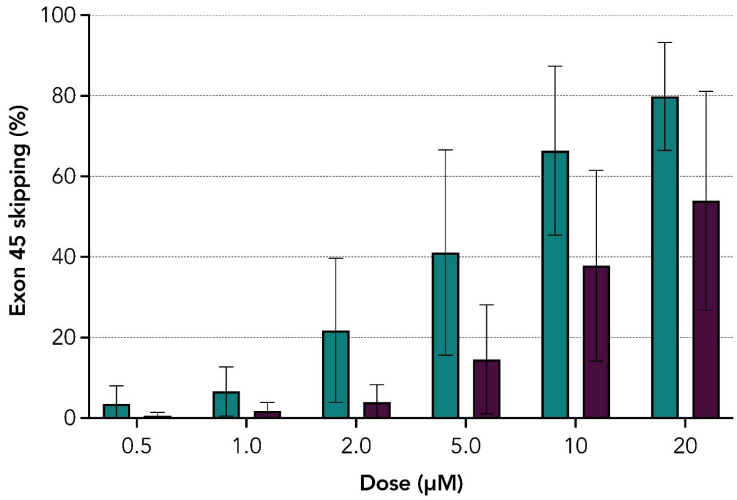
PGN-EDO45

WT human myoblasts

Hour: 0 48

PPMO dose
Analysis

EXON SKIPPING



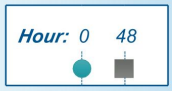
PGN-EDO45 candidate nominated for development **outperformed R_6G-PMO45 comparator at every dose level**

Protocol: WT human myoblasts were differentiated, treated for 48 hours with PGN-EDO45 or R_6G-PMO45 and then evaluated for exon 45 skipping levels by RT-PCR. Data is presented as mean ± SD of 4 biological replicates (n = 3 for R_6G-PMO at two top dose levels), which includes two technical replicates within each biological replicate. R_6G-PMO45 was selected as a relevant comparator PPMO approach.

PGN-EDO44: HIGH, DOSE-DEPENDENT LEVELS OF EXON 44 SKIPPING WERE OBSERVED IN WILD-TYPE HUMAN MYOBLASTS

PGN-EDO44

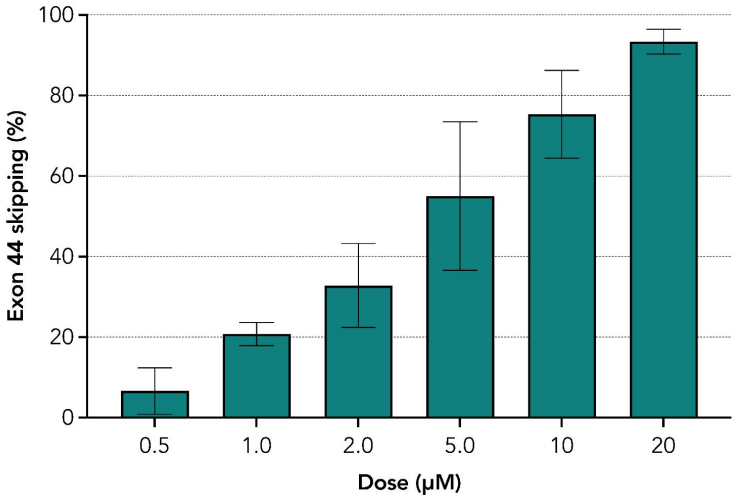
WT human myoblasts



PGN-EDO44 dose

Analysis

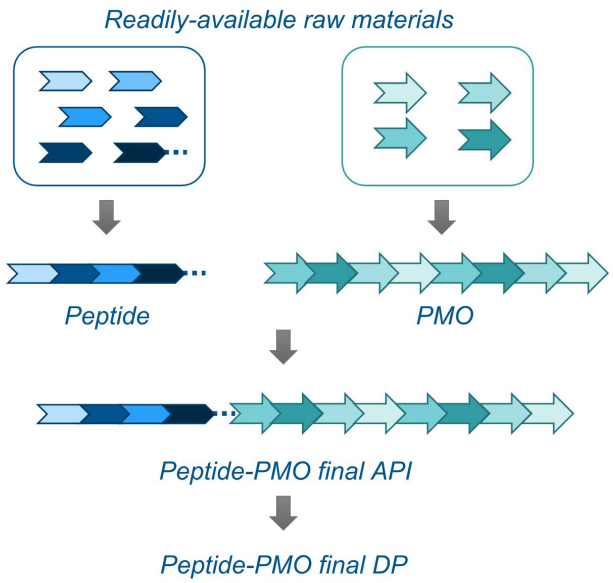
EXON SKIPPING



PGN-EDO44 candidate **nominated for development**

Protocol: WT human myoblasts were differentiated, treated for 48 hours with PPMOs and then evaluated for exon 44 skipping levels by RT-PCR. Data is presented as mean ± SD of 3 biological replicates, which includes two technical replicates within each biological replicate.

CURRENT MANUFACTURING CAPABILITIES DESIGNED TO SUPPORT ALL PLANNED CLINICAL TRIALS AND COMMERCIALIZATION



HIGHLIGHTS:

- **Fully synthetic** manufacturing process; **no cell-based steps**
- Product and intermediates are **readily characterized**
- Research to date suggests product has **robust stability**
- **Multiple cGMP** batches have been **manufactured and released**

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.

PepGen

THANK YOU