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): May 21, 2026

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

(Former Name or Former Address, if Changed Since Last Report)

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 May 21, 2026, PepGen Inc. (the "Company") updated its Corporate Presentation in connection with its participation in the 15th International Myotonic Dystrophy Consortium being held May 26-30, 2026, a copy of which is being furnished as Exhibit 99.1 and incorporated herein by reference.

The information in this Current Report on Form 8-K (including Exhibit 99.1 attached hereto) is intended to be furnished and shall not be deemed "filed" for 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 Current Report on Form 8-K 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.

(d) Exhibits

Exhibit Number	Description
99.1	Corporate Presentation updated as of May 2026
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: May 21, 2026

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

Exhibit 99.1

PepGen™

Company Presentation

May 2026

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 high levels of oligonucleotide to the nuclei, the promising trends and therapeutic potential and safety profile of PGN-EDODM1 based on data from the 5, 10 and 15 mg/kg cohorts of the FREEDOM study and 5 mg/kg cohort of the FREEDOM2 study, our expectations regarding the potential for significant correction of mis-splicing with more and higher doses of PGN-EDODM1 over a longer treatment period to potentially provide improved functional benefit for patients with DM1, the design, initiation and conduct of clinical trials, including expected timelines for data readouts from our FREEDOM2 trial, the potential for any functional improvements that may result from robust splicing correction with PGN-EDODM1, dose-dependent increases in splicing suggesting that PGN-EDODM1 is getting into the muscle and effectively binding to the target, the potential for PGN-EDODM1 to offer a best-in-class treatment option, ongoing and planned regulatory interactions and our financial resources and expected 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-EDODM1; our ability to enroll patients in our clinical trials, including 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-EDODM1; our product candidates, including 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, including release of the partial clinical hold placed by FDA on the FREEDOM2 study, or other regulatory feedback requiring modifications to our development programs, including with respect to the FREEDOM2 program; 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 filings 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-EDODM1, an investigational therapy, that has 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-EDODM1 or any other investigational therapy will successfully complete clinical development or gain regulatory authority approval.

Leveraging EDO Platform to Drive Meaningful Impact for Patients

OUR VISION

Develop therapies that **address the root cause** of serious genetic neuromuscular and neurological diseases—driving meaningful, functional improvement

Backed by a team of **leading neuromuscular researchers** with deep expertise in genetic disease biology and oligonucleotide drug development

Strong cash runway into **2H 2027,** through FREEDOM2 12.5 mg/kg MAD readout

EDO PLATFORM

Achieving **superior nuclear delivery and uptake** of therapeutic oligonucleotides, overcoming key limitations of prior approaches

PGN-EDODM1: *Myotonic Dystrophy Type 1*

- Best-in-class potential; selectively targets only pathogenic *DMPK* RNA
- Favorable emerging safety profile and FREEDOM2 5mg/kg results supportive of the ongoing dosing in 10 mg/kg MAD cohort
- FREEDOM2 cleared in South Korea, Australia, and New Zealand; enrollment open and active in Canada, UK, and South Korea
- Orphan Drug & Fast Track Designation (U.S.); Orphan Designation (EU)

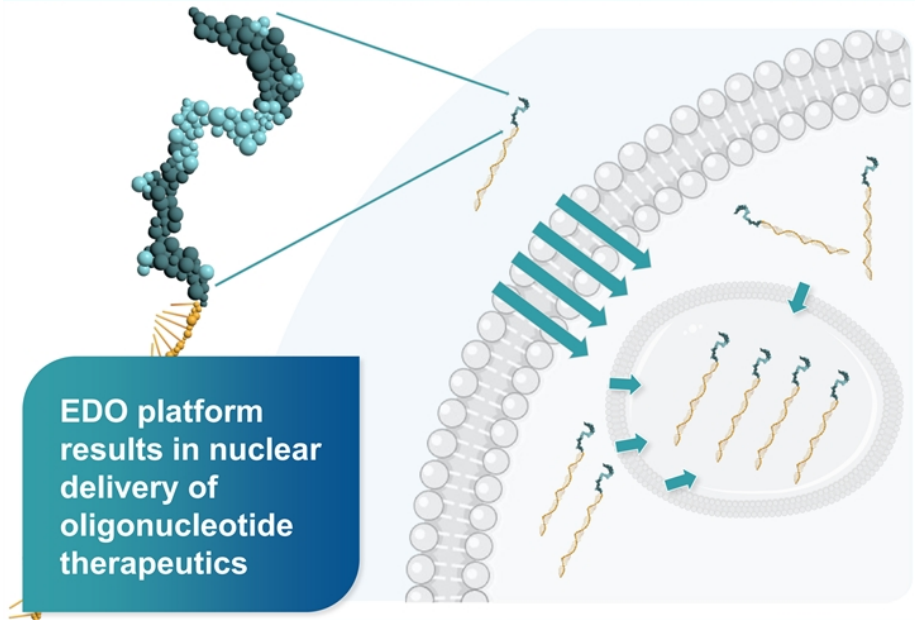
Anticipated Upcoming Milestones

- **2H 2026:** FREEDOM2 10 mg/kg clinical results
- **2027:** FREEDOM2 12.5 mg/kg clinical results

Research Pipeline

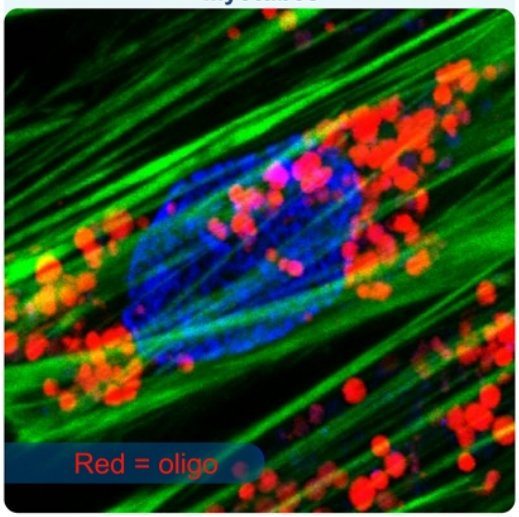
- Developing research pipeline that applies platform differentiators to address underlying neuromuscular disease drivers
- Exploring EDO's potential in genetic conditions, including Charcot-Marie-Tooth disease

PepGen's EDO Platform Has Been Designed and Developed to Solve the Delivery Challenge of Oligonucleotides



EDO platform results in nuclear delivery of oligonucleotide therapeutics

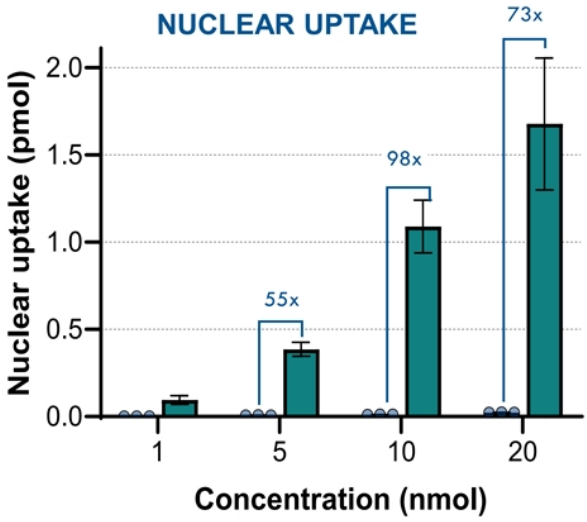
PGN-EDODM1 Delivery to DM1 Patient Myotubes



Immortalized myoblasts from 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 10 uM. Cells were visualized by confocal microscopy 24h after treatment.

EDO Technology Can Improve Endosomal Escape and Has Been Shown to Increase Nuclear Uptake up to 98-Fold

PMO Delivery in DM1 Cells



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. Graphs are presented as mean ± SD.

PGN-EDODM1: A Differentiated Drug with Best-in-Class Potential

1 Differentiated Delivery Technology

- **Receptor-independent EDO peptide delivery**
- Designed to escape the endosome – unlike TfnR targeting

2 Differentiated Target

- **Selectively targets pathogenic RNA** (CUG repeat in *DMPK*)
- Demonstrated highest rate of splicing correction ever reported in DM1 after a single dose[1]

3 Cost Effective Manufacturing

- **EDO peptide is a short linear peptide** – not cell culture product

1. FREEDOM 15 mg/kg data readout when compared with prior published data.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PGN-EDODM1 –
Myotonic Dystrophy Type 1 (DM1)

Myotonic Dystrophy Type 1 Overview and Unmet Medical Need

Overview

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

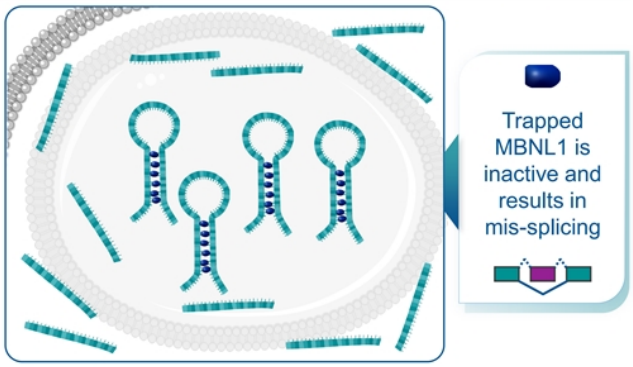
Market Opportunity

- U.S. and EU over 115,000 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
CUG: cytosine-uracil-guanine; *DMPK*: dystrophia myotonica protein kinase

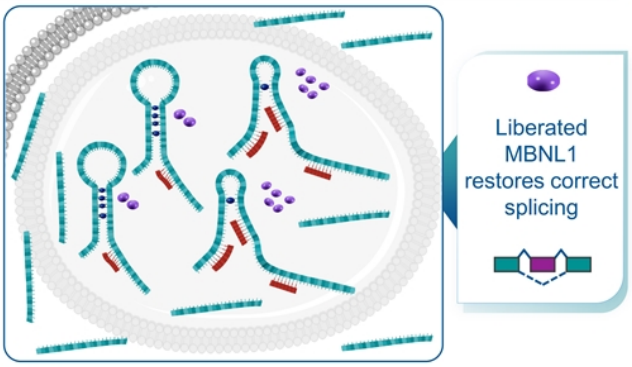
PGN-EDODM1 Blocking Approach Targets the Pathogenic CUG^{exp} Repeats *DMPK* RNA

DM1 is caused by pathogenic *DMPK* transcripts



- DM1 is caused by pathogenic *DMPK* transcripts containing CUG^{exp} repeat sequences that form hairpin loops
- These hairpin loops trap MBNL1 proteins that are needed for correct splicing of mRNAs

PGN-EDODM1 binds selectively to the pathogenic *DMPK* transcript



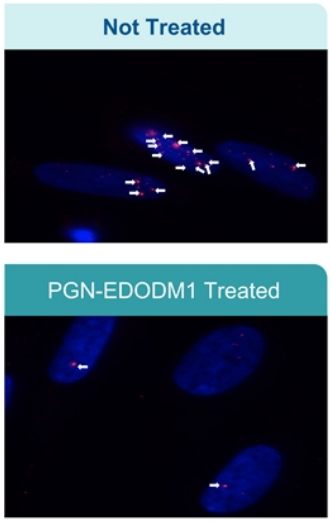
- PGN-EDODM1 binds selectively to the pathogenic *DMPK* transcript
- This reduces the ability of the CUG^{exp} repeats to form hairpin loops and sequester RNA splicing proteins

DMPK transcript | Bound MBNL1 (inactive) | PGN-EDODM1 | Free MBNL1 (active)

Wojciechowska, et al., Quantitative Methods to Monitor RNA Biomarkers in Myotonic Dystrophy, *Nature*, April 12, 2018

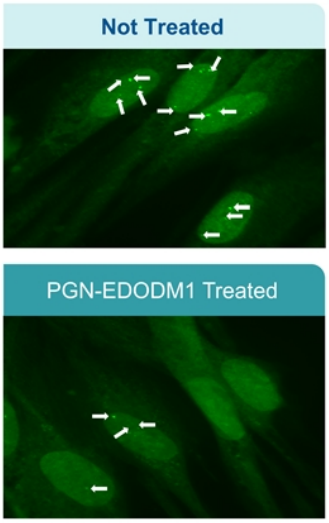
PGN-EDODM1 Reduced Pathogenic Nuclear Foci, Liberated MBNL1 and Corrected Mis-Splicing in Patient Cells with Long CUG Repeats

Foci Reduction



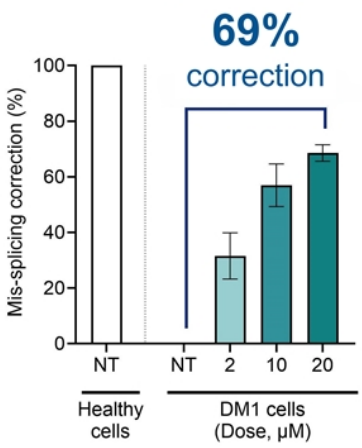
54%
reduction in toxic foci

MBNL1 Liberation



Mis-Splicing Correction

Across multiple transcripts

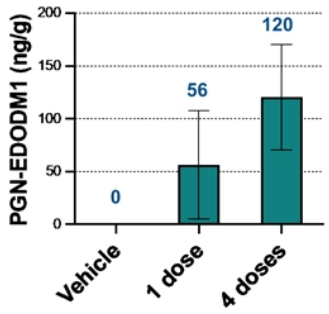


Immortalized myoblasts from healthy individual or DM1 patient with 2600 CTG repeats were cultured then differentiated for 4 days into myotubes. Treatment with peptide-PMO conjugates at concentrations given. Cells were harvested for analysis 24h after treatment. RNA isolation, RT-PCR and capillary electrophoresis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

Multiple Doses of PGN-EDODM1 Led to Greater Improvement in Splicing Correction and Myotonia vs Single Dose in Preclinical Studies

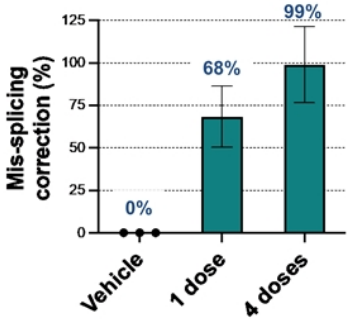
Tissue Concentration

Skeletal muscle



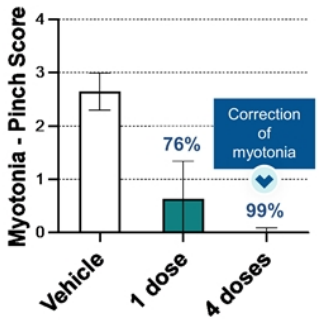
Mis-Splicing Correction

Across multiple transcripts



Correction of Myotonia

Pinch test



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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. Graphs are presented as mean ± SD; n = 12-43 per cohort.


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PGN-EDODM1 –
FREEDOM SAD Trial in DM1

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

Freedom
DM1
FREEDOM Phase 1

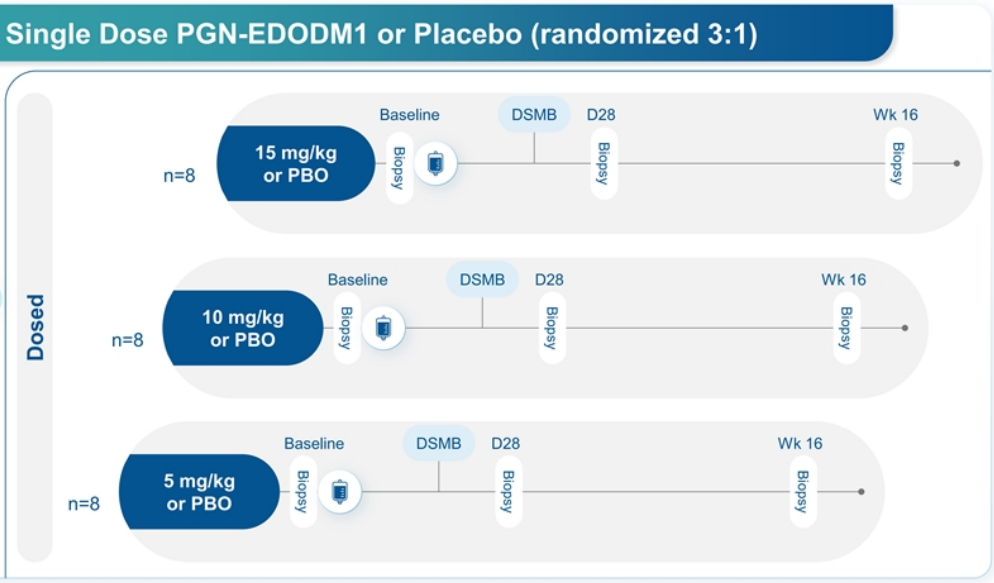
Study Overview

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

Single IV administration of PGN-EDODM1

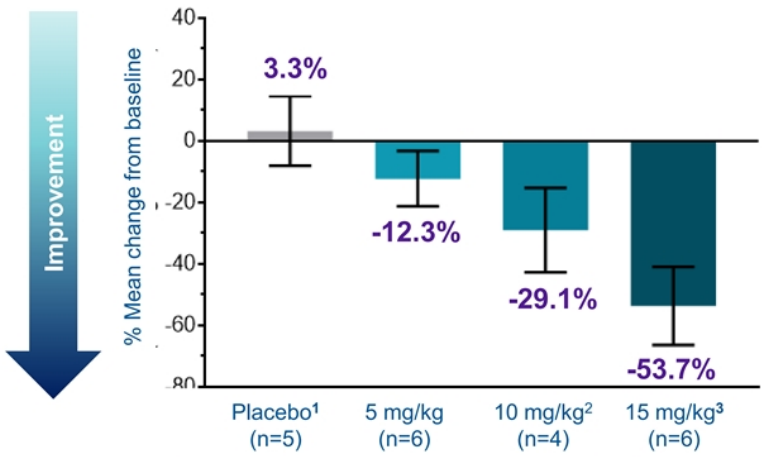
Muscle biopsies in tibialis anterior at Baseline, Day 28, Week 16

Safety, PK, correction of mis-splicing, initial functional assessments



PGN-EDODM1 Produced Dose-Dependent Best-in-Class Splicing Correction Following Single Dose

Splicing Index Changes: 22-Gene Panel* at D28

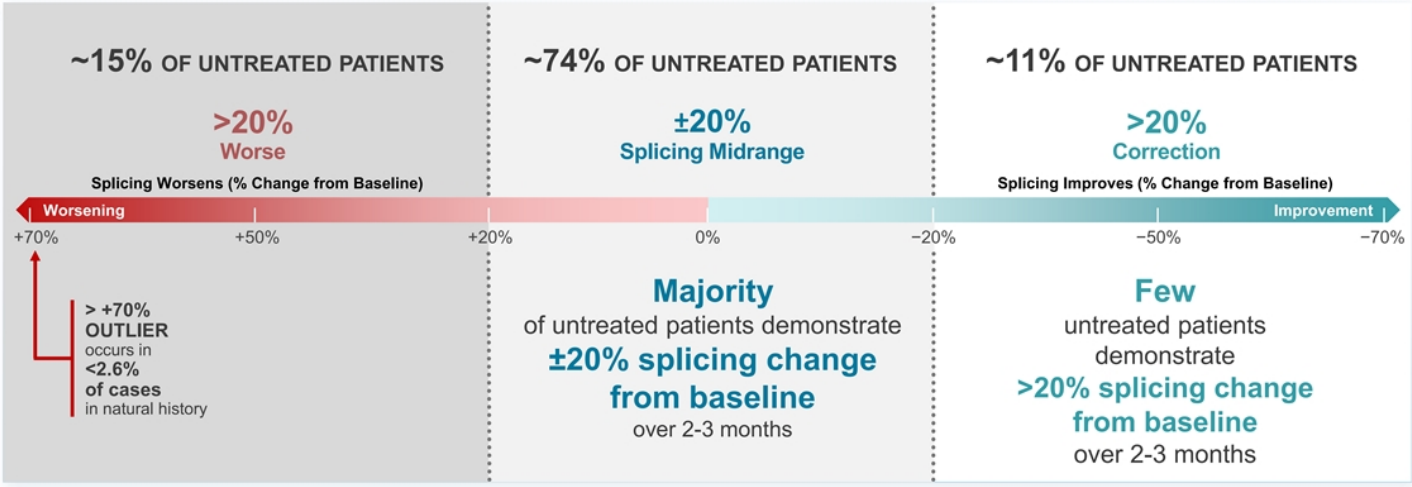


87.5%
of participants across all doses showed improved splicing

1.Missing samples due to unavailability of biopsy tissue or sample outside of assay window.
2. One subject at 10 mg/kg biopsy was not collected at day 28 due to pseudoaneurysm in connection with biopsy and one participant's splicing index fell below the pre-specified assay range at baseline and at day 28 (indicating no detectable mis-splicing)
3.One subject at 15mg/kg received 77% of the dose and was still included in the splicing index change analysis for the cohort
*Provenzano et al., The Splice Index as a prognostic biomarker of strength and function in myotonic dystrophy type 1,J Clin. Invest. 2025

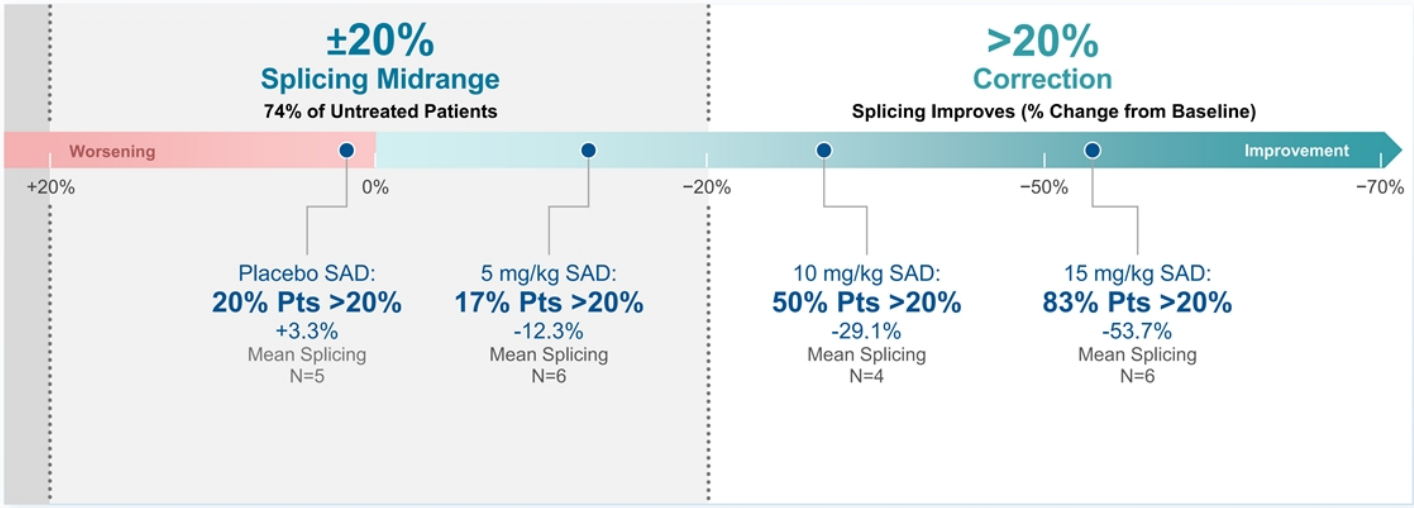
~11% of Untreated DM1 Patients Demonstrate >20% Splicing Improvement over a 2 to 3 Month Time Period

Natural History Data in Untreated DM1 Patients*



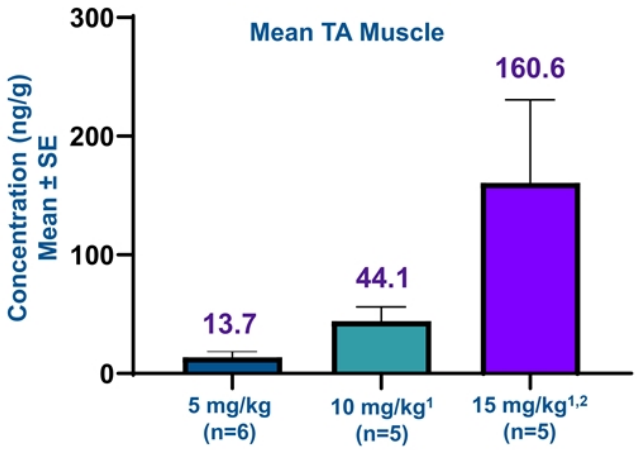
*Data from C Thornton et al., Lancet Neurology 2023; 22: 218–28. Supplement

PGN-EDODM1 Demonstrated >20% Splicing Correction in a Majority of DM1 Patients after a Single Dose ≥10 mg/kg



Robust, Greater Than Dose-Proportional Increase in Muscle Tissue Concentration Following Single Dose

Muscle Tissue Concentration at D28



1.Missing samples due to unavailability of biopsy tissue
2.One subject at 15mg/kg received 77% of the dose and was still included in the muscle tissue concentration analysis for the cohort

PGN-EDODM1 Was Generally Well Tolerated, with TEAEs Primarily Mild to Moderate Across Dose Cohorts

	Placebo (n=6) N (events)	Cohort 1 5 mg/kg (n=6)	Cohort 2 10 mg/kg (n=6)	Cohort 3 15 mg/kg (n=6)	Total (n=24)
Any TEAE, n (events)	5 (16)	3 (20)	4 (16)	5 (18)	17 (70)
Any TEAE by Max Severity Mild/Moderate Severe	 5 0	 2 1	 2 2	 5 0	 14 3
Any related TEAE, n (events)	1 (3)	1 (1)	2 (4)	4 (14)	8 (22)
Any SAE (event)	1(2)	1 (1)	2 (2)	0 (0)	4 (5)
Any related SAE	0	0	1 (1)	0	1(1)
Any TEAE leading to study withdrawal	0	0	0	0	0
Any TEAE leading to death	0	0	0	0	0

- Most frequent TEAEs: nausea, nasopharyngitis, and headache
- No electrolyte-related TEAEs or hypomagnesemia observed across dose cohorts
- No renal-related TEAEs observed at 5 and 10 mg/kg; DLT at 15 mg/kg involving a transient decrease in eGFR(cys), resolving without intervention
- Transient moderate albuminuria observed at 15 mg/kg and mild albuminuria at 10 mg/kg; Normalized within 2-7 days without intervention
- One drug-related hypersensitivity reaction (rash) during infusion at 15 mg/kg, resolving within 2 hours with oral antihistamines
- One drug-related SAE of severe abdominal pain at 10 mg/kg, confounded by off-label medication use on the day of dosing

*As of database lock on December 23, 2025. Unblinded FREEDOM safety data
TEAE: treatment-emergent adverse event, SAE: serious adverse event, DLT: Dose limiting toxicity, eGFR(cys): estimated glomerular filtration rate (cystatin equation)


PepGen™
PGN-EDODM1 –
FREEDOM2 MAD Trial in DM1

FREEDOM2: Phase 2 MAD Study Design

Freedom 2
DM1

FREEDOM2 Phase 2 Study Overview

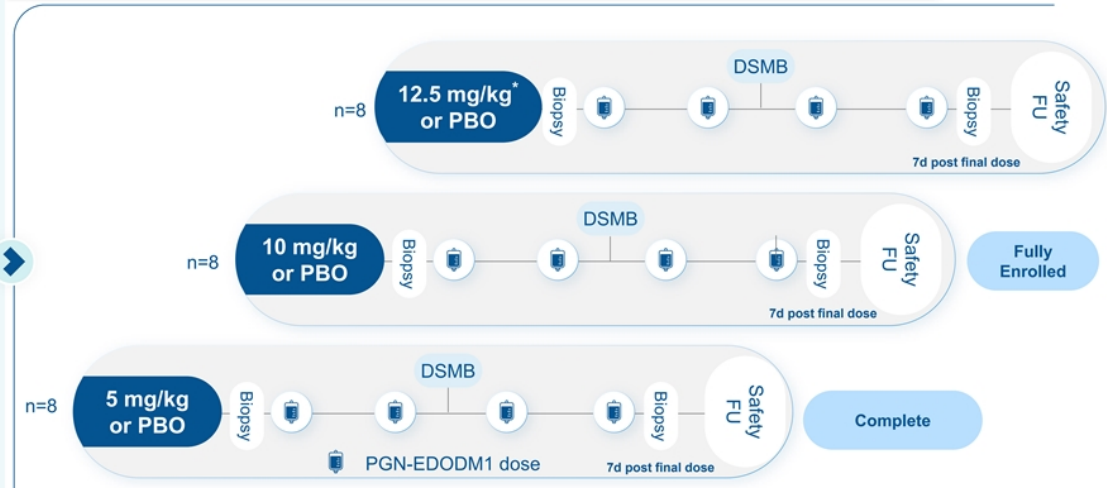
Multinational, randomized, double-blind, placebo-controlled, MAD study open in Canada, UK, NZ, Australia and South Korea**

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

FREEDOM-OLE open for patients in FREEDOM & FREEDOM2

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; OLE: open label extension
* Dose dependent on recommendations of DSMB
**The U.S. FDA recently placed a partial clinical hold on FREEDOM2-DM1

Favorable Emerging Safety Profile of PGN-EDODM1; No Increase in Toxicity with Multiple Doses at 5 mg/kg

Summary of Treatment Emergent Adverse Events (TEAEs)[1]	5 mg/kg (n=8) n(%)
Any TEAE	7 (87.5)
Mild	4 (50.0)
Moderate	3 (37.5)
Severe	0 (0.0)
Any SAE	0
Any related SAE	0
Any AESI or dose-limiting toxicities	0
Any TEAE leading to study withdrawal	0
Any TEAE leading to death	0

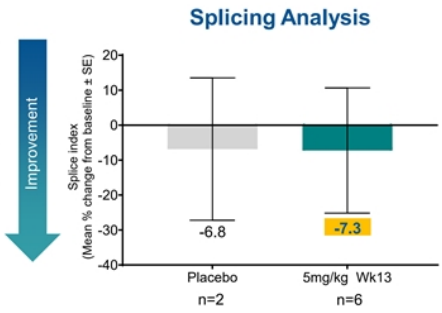
PGN-EDODM1 was Generally Well-Tolerated, with All AEs Mild or Moderate in Severity[1]

- All participants completed all 4 doses, with no evidence of cumulative AEs
- The overall AE profile of MAD 5 mg/kg is consistent with that observed in SAD 5 mg/kg
- Nausea was the most common AE
- No SAEs, AESIs, or DLTs and no signs of hypersensitivity
- eGFR and creatinine measurements within the normal range
- No hypomagnesemia
- Transient albuminuria observed – did not increase with repeat dosing

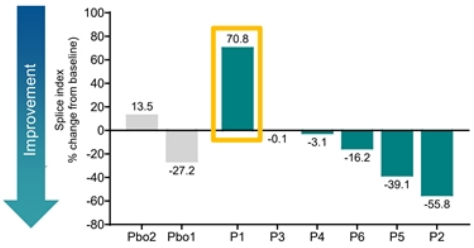
1. Data cutoff date: 04 March 2026
AE: Adverse event; AESI: Adverse event of special interest; DLT: Dose limiting toxicities; SAE: Serious adverse event; TEAE: Treatment emergent adverse event; eGFR: estimated Glomerular Filtration Rate

FREEDOM2 5 mg/kg Splicing Correction

5 mg/kg Collective Splicing Data



5 mg/kg Individual Splicing Data



Excluding notable splicing outlier mean splicing correction of 22.9% (n=5)

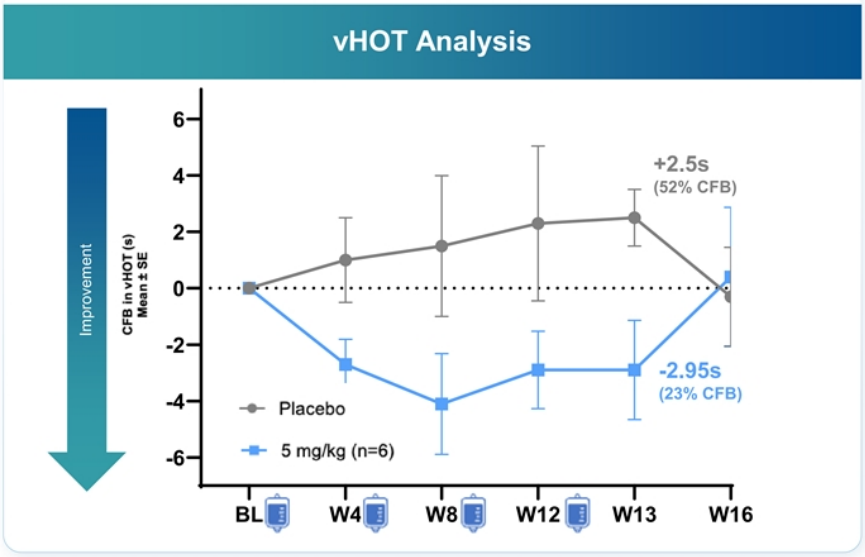
High mean muscle tissue concentration of PGN-EDODM1 of 158 ng/g at Day 7 post-dose (n=5)*

PepGen™

Data cutoff date: 04 March 2026

*One patient's muscle tissue concentration reading was pending at cutoff date.

FREEDOM2 5 mg/kg Myotonia (vHOT): PGN-EDODM1 Shows Promising Middle Finger vHOT Trends at Lowest Dose

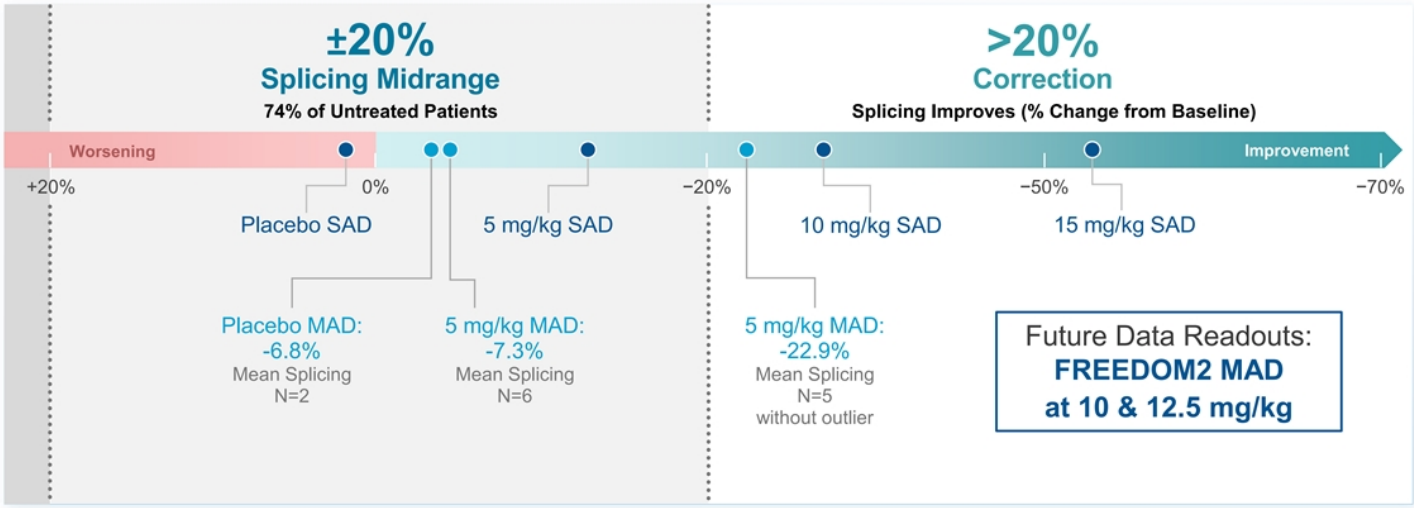


- Excluding notable splicing outlier, the active group remained below baseline (n=5)
- Splicing outlier demonstrated 22 sec difference between nadir and week 16

CFB: Change from baseline; SE: Standard error
Data cutoff date: 04 March 2026

FREEDOM2 MAD at 10 & 12.5 mg/kg has Potential to Build Upon Robust Single-Dose Splicing Correction

FREEDOM SAD
FREEDOM2 MAD



FREEDOM2 MAD

Promising Safety, Splicing and vHOT Data in FREEDOM2 Lowest Dose – Supports Ongoing 10 mg/kg MAD Cohort

SAFETY & TOLERABILITY	• PGN-EDODM1 was generally well-tolerated; all AEs were mild or moderate in severity, with no SAEs or cumulative toxicity with repeat dosing observed
SPLICING & FUNCTIONAL DATA:	• Mean splicing correction of 7.3% with PGN-EDODM1 (n=6) vs 6.8% placebo (n=2) • Analysis excluding one notable splicing outlier demonstrated mean splicing correction of 22.9% (n=5) • Promising trends observed in vHOT in PGN-EDODM1 treated group

Company on track to report clinical data from 10 mg/kg multiple dose cohort in 2H 2026

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Data cutoff date: 04 March 2026
AE: Adverse event; SAE: Serious adverse event

Summary of PGN-EDODM1 and FREEDOM Program

1. **Differentiated Delivery Technology**
2. **Differentiated Target**

FREEDOM STUDY:

PRIMARY: SAFETY

- ☑ Favorable emerging safety profile

EXPLORATORY: PD (SPLICING)

- ☑ Unprecedented splicing correction achieved with single dose

PHASE 2 FREEDOM2 MAD & OLE

Promising Safety, Splicing and vHOT Data in FREEDOM2 Lowest Dose – Supports Ongoing 10 mg/kg MAD Cohort

- Company has **completed enrollment** in the 10 mg/kg MAD cohort of FREEDOM2
- **13 patients** have enrolled in the FREEDOM-**OLE** at 5 mg/kg, including 6 patients from FREEDOM2

GUIDANCE:

- **2H 2026:** FREEDOM2 10 mg/kg clinical results
- **2027:** FREEDOM2 12.5 mg/kg clinical results

Strong cash runway into 2H **2027**, through FREEDOM2 12.5 mg/kg MAD readout


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Thank you