EMPOWERING OLIGONUCLEOTIDE THERAPEUTICS

COMPANY PRESENTATION
NOVEMBER 2022
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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 our most recent 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.
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
- Ph2a patient trial anticipated to initiate in 1H23 (potentially pivotal)

PGN-EDODM1 for DM1

- EDO technology delivered to human muscle levels of oligonucleotide which were pharmacologically active in DM1 mouse model
- EDO-mediated delivery of therapeutic oligonucleotides to the CNS observed in NHP studies
- Ph1/2 patient trial anticipated to initiate 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.
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 (%)

- **Exondys 51® (eteplirsen)** – Sarepta Therapeutics
  - Approved 2016, 2021 sales: $454M

- **SRP-5051 (vesleteplirsen)** – Sarepta Therapeutics
  - Dystrophin level at 30 mg/kg (3 or 5 doses)
  - Currently in Ph2b

- **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

- **EXONDYS 51® (eteplirsen)** – Sarepta Therapeutics
  - Approved 2016, 2021 sales: $454M

* Clinical data included in drug label (FDA). **Source: Sarepta MOMENTUM study update, 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 2021 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
<table>
<thead>
<tr>
<th>PROGRAM</th>
<th>INDICATION</th>
<th>DISCOVERY</th>
<th>PRECLINICAL</th>
<th>PHASE 1</th>
<th>PHASE 2</th>
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<tbody>
<tr>
<td>PGN-EDO51</td>
<td>Duchenne muscular dystrophy</td>
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<tr>
<td></td>
<td>Exon 51</td>
<td></td>
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<td>PGN-EDOM1</td>
<td>Myotonic dystrophy type 1</td>
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<td>PGN-EDO53</td>
<td>Duchenne muscular dystrophy</td>
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<td></td>
<td>Exon 53</td>
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<td>PGN-EDO45</td>
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<td>Exon 45</td>
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<td>PGN-EDO44</td>
<td>Duchenne muscular dystrophy</td>
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<td></td>
<td>Exon 44</td>
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</table>

**FUTURE PIPELINE OPPORTUNITIES**

Additional neuromuscular indications
Neurologic indications
PEPGEN: EXPERIENCED TEAM OF COMPANY BUILDERS, SCIENTISTS, AND CLINICIANS

Management team

JAMES MCArTHUR, PhD (CEO & President)

NOEL DONNELLY (CFO)

JAYA GOYAL, PhD (EVP Research & Preclinical Development)

NIELS SVENSTRUP, PhD (SVP Chemistry & Manufacturing)

MICHIELLE MELLION, MD (SVP Clinical Development)

SONIA BRACEGIRDLE, DPhil (SVP Strategy & Operations)

Board of Directors*

LAURIE KEATING, JD (Chair)

JOSH RESNICK, MD (Director)

HABIB DABLE (Director)

CHRIS ASHTON, PhD (Director)

HEIDI HENSON (Director)

* Plus Dr. McArthur (Director).
PGN-EDO51 FOR DUCHENNE MUSCULAR DYSTROPHY
DUCHENNE MUSCULAR DYSTROPHY IS A DEBILITATING, PROGRESSIVE MUSCLE-WASTING DISEASE

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

*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 or even reverse disease progression

PGN-EDO51 WAS ENGINEERED TO TRANSFORM THE TREATMENT OF EXON 51 DUCHENNE MUSCULAR DYSTROPHY

PGN-EDO51 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

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* 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.
PRECLINICAL DATA
**The activity of our EDO platform in DMD has been evaluated in multiple preclinical models**

<table>
<thead>
<tr>
<th>Species</th>
<th>Study design</th>
<th>Key readouts observed</th>
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<tbody>
<tr>
<td><strong>Non-GLP</strong></td>
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<tr>
<td><strong>pharmacology</strong></td>
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<tr>
<td><strong>studies</strong></td>
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<tr>
<td>Patient cells</td>
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<td></td>
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<tr>
<td><strong>PGN-EDO51</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DMD patient</td>
<td></td>
<td>• High levels of exon 51 skipping</td>
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<tr>
<td></td>
<td>Single dose</td>
<td></td>
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<td><strong>PGN-EDO23</strong></td>
<td></td>
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<tr>
<td><em>mdx</em></td>
<td></td>
<td>• Normalization of serum creatine kinase</td>
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<tr>
<td></td>
<td></td>
<td>• High levels of exon 23 skipping and dystrophin restoration</td>
</tr>
<tr>
<td></td>
<td>Single dose</td>
<td></td>
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<tr>
<td><strong>PGN-EDO51</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>WT</em></td>
<td></td>
<td>• High levels of exon 51 skipping</td>
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<tr>
<td></td>
<td>Repeat dose</td>
<td></td>
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<tr>
<td><strong>PGN-EDO51</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>WT</em></td>
<td></td>
<td>• High levels of exon 51 skipping</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Accumulation of exon skipping levels with repeat dosing</td>
</tr>
</tbody>
</table>
MDX MICE: A SINGLE DOSE OF PGN-EDO23 WAS OBSERVED TO NORMALIZE CREATINE KINASE, A MARKER OF MUSCLE DAMAGE

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: ROBUST DYSTROPHIN RESTORATION OBSERVED 7 DAYS AFTER A SINGLE, GENERALLY WELL-TOLERATED DOSE OF PGN-EDO23

**EXON SKIPPING**

![Bar chart showing exon skipping percentages for different muscle groups.]

- **QUAD**: 86.3%
- **BICEPS**: 93.1%
- **DIAPH**: 76.6%
- **HEART**: 62.3%

**DYSTROPHIN**

![Bar chart showing dystrophin percentages for different muscle groups.]

- **QUAD**: 90.4%
- **BICEPS**: 99.7%
- **DIAPH**: 80.6%
- **HEART**: 25.7%

**Protocol:** peptide-PMO conjugate and a saline control were administered intravenously (IV) to mdx and WT mice; exon skipping and dystrophin restoration measured 7 days after injection. Mean ± SD; n = 3 for control groups and 3 for treated groups. NT = not treated.
NHP: 10 MG/KG OF PGN-EDO51 SHOWED SIMILAR POTENCY TO 30 MG/KG OF R6G-PMO IN QUADS & BICEPS

Protocol: PGN-EDO51 and R6G-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. R6G-PMO51 is believed to be structurally equivalent to SRP-5051.

Exon skipping levels of >70% observed in skeletal muscles at 30 mg/kg for PGN-EDO51
NHP: 10 MG/KG OF PGN-EDO51 SHOWED SIMILAR POTENCY TO 30 MG/KG OF R₆G-PMO IN DIAPHRAGM

A single dose of 20 mg/kg of PGN-EDO51 afforded 19% exon 51 skipping in whole heart

Protocol: PGN-EDO51 and R₆G-PMO were administered to NHP by IV infusion over 30 min at the doses indicated (n=3). Q4W, 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. R₆G-PMO51 is believed to be structurally equivalent to SRP-5051.
NHP: EXON SKIPPING LEVELS ACCUMULATED WITH REPEAT DOSE ADMINISTRATION OF PGN-EDO51

**Protocol:** PGN-EDO51 and R6G-PMO were administered to NHP by IV infusion over 30 min at the doses indicated (n=3). Q2W, three doses administered, saline control. Biopsies taken 7 days after each administration; terminal samples collected 7 days after final dose. Study not powered for statistical significance. Data shown as mean ± SD; n = 3 per group. R6G-PMO51 is believed to be structurally equivalent to SRP-5051.

**QUADRICEPS**

30 mg/kg

<table>
<thead>
<tr>
<th>Week</th>
<th>PPMO dose</th>
<th>Tissue analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
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<td>1</td>
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<tr>
<td>5</td>
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</tbody>
</table>

**Exon 51 skipping (%)**

- **0 mg/kg:** WT
- **30 mg/kg:** PGN-EDO51 (44.9%), R6G-PMO51 (4.5%)

**BICEPS**

30 mg/kg

<table>
<thead>
<tr>
<th>Week</th>
<th>PPMO dose</th>
<th>Tissue analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
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<tr>
<td>4</td>
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</tbody>
</table>

**Exon 51 skipping (%)**

- **0 mg/kg:** WT
- **30 mg/kg:** PGN-EDO51 (43.6%), R6G-PMO51 (5.5%)

Q2W regimen employed in this study; differential with R6G-PMO may increase with monthly dosing.
CLINICAL DATA
**PH1 HEALTHY NORMAL VOLUNTEER (HNV) 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**

<table>
<thead>
<tr>
<th>Dose Level</th>
<th>Participants</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mg/kg</td>
<td>n = 8</td>
</tr>
<tr>
<td>5 mg/kg</td>
<td>n = 8</td>
</tr>
<tr>
<td>10 mg/kg</td>
<td>n = 8</td>
</tr>
<tr>
<td>15 mg/kg</td>
<td>n = 8</td>
</tr>
</tbody>
</table>

**SRC** = safety review committee.
HNV: HIGHEST LEVELS OF OLIGO DELIVERY & EXON 51 SKIPPING OBSERVED, SUPPORTING FURTHER DEVELOPMENT OF PGN-EDO51

<table>
<thead>
<tr>
<th>PH1 ENDPOINTS</th>
<th>READOUTS OBTAINED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Target engagement</td>
<td>Highest level of DMD exon 51 skipping observed following a single dose in humans*</td>
</tr>
<tr>
<td>Exon skipping (biceps)</td>
<td></td>
</tr>
<tr>
<td>Pharmacokinetics</td>
<td>Highest level of oligonucleotide delivery observed for a DMD therapeutic</td>
</tr>
<tr>
<td>Tissue concentration (biceps)</td>
<td>following a single dose in humans</td>
</tr>
<tr>
<td>Safety &amp; tolerability</td>
<td>Generally well-tolerated</td>
</tr>
</tbody>
</table>

* 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.
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 was 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.
HNV: HIGH, PERSISTENT TISSUE CONCENTRATIONS OF OLIGONUCLEOTIDE WERE OBSERVED

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.

These results further support our belief that repeat dosing of PGN-EDO51 may lead to accumulation of skipped transcript and dystrophin in DMD patients.
At 10 mg/kg:

- All participants 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.
PH1 TRIAL SAFETY & TOLERABILITY SUMMARY

<table>
<thead>
<tr>
<th>Healthy Normal Volunteers (HNV) with ≥1 AE, n (%)</th>
<th>Placebo (n=8)</th>
<th>Cohort A: 1 mg/kg (n=6)</th>
<th>Cohort B: 5 mg/kg (n=6)</th>
<th>Cohort C: 10 mg/kg (n=6)</th>
<th>Cohort D: 15 mg/kg (n=6)</th>
<th>PGN-EDO51 Total (n=24)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any AE</td>
<td>4 (50)</td>
<td>4 (66.7)</td>
<td>2 (33.3)</td>
<td>5 (83.3)</td>
<td>6 (100)</td>
<td>17 (70.8)</td>
</tr>
<tr>
<td>Related to study drug</td>
<td>1 (12.5)</td>
<td>2 (33.3)</td>
<td>0</td>
<td>4 (66.7)</td>
<td>6 (100)</td>
<td>12 (50)</td>
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<td>Serious AE related to study drug</td>
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<td>0</td>
<td>1 (16.7)</td>
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<td>AE leading to discontinuation</td>
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<td>AE leading to death</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<td>0</td>
</tr>
<tr>
<td>Number of Related TEAEs by CTCAE v5.0 grading*</td>
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</tr>
<tr>
<td>Grade 1 (Mild)</td>
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<td>1</td>
<td>0</td>
<td>7</td>
<td>12</td>
<td>20</td>
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<td>Grade 2 (Moderate)</td>
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<td>Grade 3 (Severe)</td>
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* 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 HNV 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 dose 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.
PEPGEN HAS COMPLETED A PH1 HNV TRIAL FOR PGN-EDO51; ON TRACK TO INITIATE DMD PATIENT CLINICAL TRIAL IN 1H23

<table>
<thead>
<tr>
<th>Anticipated milestones</th>
<th>2022</th>
<th>2023</th>
<th>2024</th>
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<tbody>
<tr>
<td></td>
<td><strong>2Q</strong>: First HNV dosed in Ph1 trial</td>
<td><strong>1H</strong>: Initiation of Ph2 DMD patient trial, potentially pivotal</td>
<td>Safety and <strong>dystrophin data</strong> in DMD patients (Ph2a)</td>
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<td></td>
<td><strong>3Q</strong>: Ph1 clinical safety, oligo delivery &amp; exon skipping data</td>
<td><strong>4Q</strong>: Completion of Ph2-enabling tox studies</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Overview</th>
<th>2022</th>
<th>2023</th>
<th>2024</th>
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<tbody>
<tr>
<td></td>
<td>Ph1 trial showed highest single-dose levels of exon skipping &amp; oligo delivery*</td>
<td>Trial will assess safety and tolerability, exon skipping and dystrophin in DMD patients</td>
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<td></td>
<td>PGN-EDO51 was generally well-tolerated</td>
<td>Safety readouts from HNV trial anticipated to support MAD initiation at higher dose levels</td>
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<tr>
<td></td>
<td>We believe readouts support progression to Ph2a</td>
<td>Precedents suggest that exon skipping readouts will be higher in patients than in HNVs at the same dose level</td>
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<td></td>
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<td>Anticipate trial will be conducted in multiple geographies, including the U.S.</td>
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* 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.
PGN-EDODM1 FOR MYOTONIC DYSTROPHY TYPE 1 (DM1)
MYOTONIC DYSTROPHY TYPE 1 IS A PROGRESSIVE, DEBILITATING NEUROMUSCULAR DISORDER WITH GREAT UNMET NEED

<table>
<thead>
<tr>
<th>ROOT CAUSE OF DISEASE</th>
<th>PATIENT POPULATION**</th>
<th>THERAPEUTIC LANDSCAPE</th>
<th>PEPGEN’S TREATMENT APPROACH</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Due to a CTG repeat expansion mutation in the DMPK gene</td>
<td>~40,000 (US)</td>
<td>• No approved disease-modifying therapeutics</td>
<td></td>
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<tr>
<td>• Leads to downstream dysregulation of a broad set of proteins</td>
<td>~75,000 (EEA)</td>
<td>• Standards of care focused on symptom management</td>
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<tr>
<td></td>
<td>~15,000 (JP)</td>
<td></td>
<td>Steric blocking of CUG repeat expansion in DPMK transcript to correct downstream mis-splicing events</td>
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</table>

* 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 TRANSFORM THE TREATMENT OF MYOTONIC DYSTROPHY TYPE 1

PGN-EDODM1 DATA SUMMARY

- Correction of mis-splicing observed in preclinical models with long and short CTG repeats
- In DM1 mouse model, robust mis-splicing correction and reversal of myotonia with a single dose; durable mis-splicing corrections observed through 24 weeks
- Not designed to degrade CUG-containing transcripts, including DMPK
- Results from PGN-EDO51 Ph1 clinical trial showed that the EDO technology delivered levels of oligonucleotide to human muscle which were observed to be pharmacologically active in DM1 mouse model
- EDO-mediated delivery of therapeutic oligonucleotides to the CNS observed in NHP studies
**DM1 IS A MULTI-SYSTEMIC DISEASE THAT HAS A SIGNIFICANT IMPACT ON QUALITY OF LIFE**

<table>
<thead>
<tr>
<th><strong>Musculoskeletal:</strong></th>
<th>Myotonia (a temporary inability to relax a muscle after contraction), muscle weakness &amp; wasting</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiac:</strong></td>
<td>Conduction defects</td>
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<tr>
<td><strong>Respiratory:</strong></td>
<td>Breathing difficulties, sleep apnea</td>
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<tr>
<td><strong>GI:</strong></td>
<td>Dysphagia (difficulty swallowing), constipation, IBS</td>
</tr>
<tr>
<td><strong>CNS:</strong></td>
<td>Cognitive impairments, behavioral / psychologic disorders, excessive daytime sleepiness</td>
</tr>
<tr>
<td><strong>Vision:</strong></td>
<td>Early-onset cataracts, retinal damage</td>
</tr>
<tr>
<td><strong>Endocrine:</strong></td>
<td>Thyroid dysfunction, diabetes</td>
</tr>
<tr>
<td><strong>Other pathologies:</strong></td>
<td>skin, immune, reproductive, increased cancer risk</td>
</tr>
</tbody>
</table>

**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

DM1 is caused by CUG triplet expansion hairpin loop in DMPK RNA sequestering MBNL1 protein.

**Without Treatment**
- CUG repeats form ‘hairpin loops’ in the DMPK RNA, which sequester a key RNA processing protein (MBNL1).
- Downstream mis-splicing events and aberrant protein expression give rise to disease phenotypes.

**With PGN-EDODM1 Treatment**
- PGN-EDODM1 binds toxic CUG repeats in DMPK RNA and blocks MBNL1 binding.
- Downstream splicing patterns are restored.

PGN-EDODM1 is designed to restore MBNL1 functions and correct downstream mis-splicing events.
### The Pharmacology of PGN-EDODM1 has been evaluated in multiple preclinical models

<table>
<thead>
<tr>
<th>Species</th>
<th>Study design</th>
<th>Key readouts observed</th>
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<tbody>
<tr>
<td><strong>Non-GLP pharmacology studies</strong></td>
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<tr>
<td>Patient cells</td>
<td></td>
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<tr>
<td>PGN-EDODM1</td>
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</table>
| DM1 patient |  | • Reduction in nuclear foci  
|  | Hour: 0 24  | • Correction of downstream transcript mis-splicing |
| Single dose |  | • Correction of downstream transcript mis-splicing  
| PGN-EDODM1 |  | • Normalization of myotonia |
| HSA<sup>LR</sup> | Week: 0 1 2  | |
| Duration of effect |  | • Correction of downstream transcript mis-splicing for at least 24 weeks post-dose |
| PGN-EDODM1 |  | |
| HSA<sup>LR</sup> | Week: 0 12 24  | |
| **Non-GLP dose-range finding (DRF) studies** | | |
| Single dose |  | • In progress |
| PGN-EDODM1 |  | |
| WT | Week: 0 1  | |
| Repeat dose |  | • In progress |
| PGN-EDODM1 |  | |
| WT | Week: 0 1 2 3 4 5  | |
PGN-EDODM1 activity has been observed in preclinical models with a wide range of CTG repeats.

**CTG REPEAT LENGTH**

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

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

**PRECLINICAL MODELS ASSESSED**
IN VITRO: PGN-EDODM1 REDUCED PATHOGENIC NUCLEAR FOCSI AND CORRECTED DOWNSTREAM TRANSCRIPT MIS-SPLICING

FOCI REDUCTION

- Not treated (NT)
- PGN-EDODM1
- PMO

MIS-SPLICING CORRECTION

Across multiple transcripts

Foci observable in patient cells

Robust reduction in number of foci following PGN-EDODM1 treatment

No foci reduction with unconjugated PMO

DM1 patient cells (2,600 CTG repeats)

Hour: 0 24

PGN-EDODM1 dose
Analysis

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

(CTG)$_n$

220-250

HSA gene 3’ UTR

DM1 ASSOCIATED ABNORMALITIES

• Skeletal muscle specific CUGexp
• MBNL1 sequestration in the nucleus
• Downstream mis-splicing events
• Myotonia

Source: Mankodi et al., SCIENCE 2000.
Protocol: PGN-EDODM1 was administered IV to HSA<sup>LR</sup> mice at doses (mg/kg): 10, 20, 30 (n=8), 50 (n=4) 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.
Protocol: PGN-EDODM1 was administered intravenously (IV) to WT and HSA<sup>LR</sup> mice at 50 mg/kg (n=4-16); myotonia assessed two weeks post-administration.
**HSA\textsuperscript{LR}: SINGLE DOSE TREATMENT OF PGN-EDODM1 LED TO DURABLE IMPROVEMENTS IN SPLICING THROUGH 24 WEEKS**

**CORRECTION OF MIS-SPLICING**

**Atp2a1**

**Clcn1**

Protocol: PGN-EDODM1 was administered intravenously (IV) to WT and HSA\textsuperscript{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\textsuperscript{LR} MOUSE MODEL

**HSA\textsuperscript{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 or 15 mg/kg dose of PGN-EDO51 in our Ph1 HNV trial, **tissue concentrations were similar** to those measured for PGN-EDODM1 at 30 mg/kg

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\textsuperscript{LR} mice at doses (mg/kg): 10, 20, 30 (n=8), 50 (n=4) against a wild-type (WT) saline control (n=8). Tissue concentration assessed by HPLC.
NHP: EDO PLATFORM CAN DELIVER TO CNS, SUPPORTING THE POTENTIAL OF PGN-EDODM1 TO ADDRESS NEUROLOGICAL SYMPTOMS

TISSUE PMO QUANTIFICATION

Protocol: NHPs received one slow bolus IV infusion on Day 1, study was terminated on Day 8 and PMO levels were quantified in key tissues. Shown as mean ± SEM; n = 2 per group. CC = cerebral cortex; LLOQ = lower limit of quantification.

Robust delivery across skeletal, cardiac and smooth muscles observed

Delivery across the blood-brain barrier observed
PEPGEN IS ON TRACK TO INITIATE A DM1 PATIENT CLINICAL TRIAL IN 1H23

Anticipated milestones

2022

- **2Q**: NHP dose range-finding study
- **2H**: IND-enabling studies

2023

- **1H**: Initiation of Ph1/2 DM1 patient trial

2024

- Safety and **proof of concept** in DM1 patients (Ph1/2)

Overview

- We believe oligonucleotide tissue concentration readouts from PGN-EDO51 Ph1 study support clinical potential of PGN-EDODM1

- Aim of clinical trials is to assess safety, tolerability and efficacy of PGN-EDODM1 in DM1 patients
EDO PIPELINE
PEPGEN’S LEAD PROGRAM TARGETS LARGEST EXON SKIPPING PATIENT POPULATION IN DMD

DMD EXON SKIPPING PATIENT SUB-POPULATIONS

- Exon 51: PGN-EDO51
  - Highest levels of exon skipping observed in humans following a single dose*

- 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

Other Exon Skips (individually rare) 36%

Remainder 29%

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.
OUR SCREENING CASCADE IS DESIGNED TO ENABLE THE RAPID DEVELOPMENT & TRANSLATION OF OUR PIPELINE PROGRAMS

In vitro screening

5 – 8 candidates screened in patient cells with established control

NHP exon skipping

1 – 2 candidates evaluated in NHP

GLP toxicology

Lead candidate advanced to GLP tox

PGN-EDO53
PGN-EDO45
PGN-EDO44
Exon Skipping Levels after a single dose of PGN-EDO53 were almost 7x higher than those observed for R_6G-PMO53 comparator.
PGN-EDO45: HIGH, DOSE-DEPENDENT LEVELS OF EXON 45 SKIPPING WERE OBSERVED IN WILD-TYPE HUMAN MYOBLASTS

EXON SKIPPING

Protocol: WT human myoblasts were differentiated, treated for 48 hours with PGN-EDO45 or R₆G-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 R₆G-PMO at two top dose levels), which includes two technical replicates within each biological replicate. R₆G-PMO45 was selected as a relevant comparator PPMO approach.

PGN-EDO45 candidate nominated for development outperformed R₆G-PMO45 comparator at every dose level
PGN-EDO44: HIGH, DOSE-DEPENDENT LEVELS OF EXON 44 SKIPPING WERE OBSERVED IN WILD-TYPE HUMAN MYOBLASTS

EXON SKIPPING

[Graph showing exon 44 skipping (%) against dose (μM)]

- PGN-EDO44 dose
- Analysis

PGN-EDO44 candidate nominated for development

WT cells

Mean ± SD

n = 2 per group; study was not powered for statistical significance

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.

PGN-EDO44
WT human myoblasts

Hour: 0 48
WE WILL LEVERAGE OUR EDO PEPTIDE PLATFORM TO:

- **REACH NEW TISSUES**
  - Explore full potential of EDO platform across **multiple tissue types**, including:
    - Deep brain structures via IT administration
    - Peripheral nerves via IV administration
    - Other tissue and cell types

- **DELIVER NEW CARGOS**
  - Utilize **modular nature** of EDO platform to evaluate new cargo technologies
  - Explore potential for **non-PMO oligo** and small molecule delivery

- **DEVELOP NEW THERAPIES**
  - Identify opportunities for novel EDO therapeutics
  - Maximize EDO platform and pipeline value through **strategic collaborations**
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** DP batches have been manufactured and released

Readily-available raw materials

Peptide-PMO final DP
CONCLUSION
THE FUTURE OF PEPGEN

<table>
<thead>
<tr>
<th>Year</th>
<th>Event</th>
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</table>
| 2022 | PGN-EDO51 (DMD exon 51) Ph1 HNV trial showed:  
- Highest level of single-dose exon skipping and oligo delivery observed in a clinical trial*  
- PGN-EDO51 was generally well-tolerated |
| 2023 | Anticipate **initiation of patient clinical trials** for DMD & DM1 |
| 2024 | Anticipate **clinical POC in two indications:**  
- Patient dystrophin data (DMD)  
- Patient splicing data (DM1) |

- 5 neuromuscular disease candidates in pipeline  
- Work underway to **leverage EDO platform** to expand to new tissues & new indications

* 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.
THANK YOU