PRECISION
BIOSCIENCES

Precision BioSciences Muscle Programs Update

May 2025

Forward-Looking Statements

This presentation contains forward-looking statements, as may any related presentations, within the meaning of the Private Securities Litigation Reform Act of 1995. The Company intends such forward-looking statements to be covered by the safe harbor provisions for forward-looking statements contained in Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. All statements contained herein and in any related presentation that do not relate to matters of historical fact should be considered forward-looking statements, including, without limitation, statements regarding the clinical development and expected safety, efficacy and benefit of our and our partners' and licensees' product candidates (including PBGENE-DMD as a potentially curative therapy for DMD), the safety, efficacy and expected benefit of our gene editing approaches including editing efficiency, and the suitability of ARCUS nucleases due to their cut, size and simplicity for gene insertion, gene elimination and gene excision and differentiation from other gene editing approaches; the expected timing of regulatory processes and clinical operations (including filings, studies, enrollment and clinical data for PBGENE-HBV, PBGENE-DMD, PBGENE-3243 and iECURE-OTC); the potential target product profile of PBGENE-DMD to potentially provide a best-in-class therapeutic profile; the ability of PBGENE-DMD to provide significant functional dystrophin protein production in different types of muscle at levels expected to provide therapeutic benefit in a humanized DMD-diseased mouse model; the ability of PBGENE-DMD to edit satellite muscle stem cells, a potential predictor of durable functional benefit; the suitability of PBGENE-DMD for the treatment of DMD and restoration of functional dystrophin protein at a therapeutic level, expectations about the commercial potential, market opportunity, operational initiatives, strategies, and further development of our programs and those of our collaboration partners; the translation of results in preclinical studies of ARCUS nucleases including PBGENE-DMD to clinical studies in humans; the potential eligibility of PBGENE-DMD for a Priority Review Voucher (PRV) valued ~$100-$150 million upon BLA approval; expectations about our and our partners' operational initiatives, strategies, and further development of our programs; expectations and updates around our partnerships and collaborations and our ability to enter into new collaborations, license agreements or other arrangements; our expected cash runway and available credit; the sufficiency of our cash runway extending into the second half of 2026 and realizing Phase 1 clinical data for multiple in vivo gene editing programs; expectations about achievement of key milestones and receipt of any milestone, royalty, or other payments; expectations regarding our liquidity and capital resources; and anticipated timing of clinical data. In some cases, you can identify forward-looking statements by terms such as "aim," "anticipate," "approach," "believe," "contemplate," "could," "designed to", "endeavor", "estimate," "expect," "goal," "intend," "look," "may," "mission," "plan," "possible," "potential," "predict," "project," "promise," "pursue," "should," "suggest," "target," "will," "would," and other similar words or expressions, or the negative of these words or similar words or expressions, are intended to identify forward-looking statements, though not all forward-looking statements use these words or expressions.

Forward-looking statements are based on management's current expectations, beliefs and assumptions and on information currently available to us. These statements are neither promises nor guarantees, but involve number of known and unknown risks, uncertainties and assumptions, and actual results may differ materially from those expressed or implied in the forward-looking statements due to various important factors, including, but not limited to: our ability to become profitable; our ability to procure sufficient funding to advance our programs; risks associated with raising additional capital and requirements under our current debt instruments and effects of restrictions thereunder; our operating expenses and our ability to predict what those expenses will be; our limited operating history; the success of our programs and product candidates in which we expend our resources; our limited ability or inability to assess the safety and efficacy of our product candidates; our dependence on our ARCUS technology; the initiation, cost, timing, progress, achievement of milestones and results of research and development activities, preclinical studies and clinical trials; public perception about genome editing technology and its applications; competition in the genome editing, biopharmaceutical, and biotechnology fields; our or our collaborators' ability to identify, develop and commercialize product candidates; potential product liability lawsuits and penalties against us or our collaborators related to our technology and our product candidates; the U.S. and foreign regulatory landscape applicable to our and our collaborators' development of product candidates; our or our collaborators' or other licensees' ability to advance product candidates into, and successfully design, implement and complete, clinical or field trials; potential manufacturing problems associated with the development or commercialization of any of our product candidates; delays or difficulties in our and our collaborators' ability to enroll patients; changes in interim "top-line" and initial data that we announce or publish; if our product candidates do not work as intended or cause undesirable side effects; risks associated with applicable healthcare, data protection, privacy and security regulations and our compliance therewith; our ability to obtain orphan drug designation or fast track designation for our product candidates or to realize the expected benefits of these designations; our or our collaborators' ability to obtain and maintain regulatory approval of our product candidates, and any related restrictions, limitations and/or warnings in the label of an approved product candidate; the rate and degree of market acceptance of any of our product candidates; our ability to effectively manage the growth of our operations; our ability to attract, retain, and motivate executives and personnel; effects of system failures and security breaches; insurance expenses and exposure to uninsured liabilities; effects of tax rules; effects of any pandemic, epidemic, or outbreak of an infectious disease; the success of our existing collaboration agreements, and our ability to enter into new collaboration arrangements; our current and future relationships with and reliance on third parties including suppliers and manufacturers; our ability to obtain and maintain intellectual property protection for our technology and any of our product candidates; potential litigation relating to infringement or misappropriation of intellectual property rights; effects of natural and manmade disasters, public health emergencies and other natural catastrophic events; effects of sustained inflation, supply chain disruptions and major central bank policy actions; market and economic conditions; risks related to ownership of our common stock, including fluctuations in our stock price; our ability to meet the requirements of and maintain listing of our common stock on NASDAQ or other public stock exchanges; and other important factors discussed under the caption "Risk Factors" in our Quarterly Report on Form 10-Q for the annual period ended March 31, 2025, as any such factors may be updated from time to time in our other filings with the SEC, which are accessible on the SEC's website at www.sec.gov and the Investors page of our website under SEC Filings at investor.precisionbiosciences.com.

All forward-looking statements speak only as of the date of this presentation and, except as required by applicable law, we haveno obligation to update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise. Precision consults with various presentation speakers and compensates them for their time and expertise.

Opening Remarks and Overview

Michael Amoroso
President & Chief Executive Officer
Precision BioSciences

Precision BioSciences Continues to Validate ARCUS Clinically Through 2025

Through our clinical programs, over 100 patients have received ARCUS-based therapies safely for hematological malignancies, chronic hepatitis B, and OTC deficiency.

Wholly-owned PBGENE-HBV program demonstrates initial safety and antiviral activity for chronic hepatitis B

U.S. IND Cleared and Fast Track Designation received

ECUR-506 partnered program utilizing ARCUS nuclease safely demonstrates complete clinical response in OTC deficiency

PRECISION BIOSCIENCES

Founded in 2006 and **dedicated to developing novel therapeutics designed to overcome and potentially cure difficult-to-treat diseases with high unmet need**, including infectious and **rare genetic diseases.**

Precision's therapies are developed utilizing **ARCUS, a proprietary next generation genome editing platform** with a unique cut, smaller size and simpler structure than CRISPR, enabling more efficient and sophisticated edits.[1]

Today, Precision is announcing prioritized internal development of our wholly-owned program for **Duchenne muscular dystrophy (DMD)**

- **PBGENE-DMD: excising the hot-spot region of DMD with goal to permanently and safely restore muscle function for the majority of patients**

1. Precision Biosciences 2023 R&D Day. View PDF presentation.

PBGENE-DMD is Designed to Provide Durable **Functional Muscle Improvement** for the Majority of Patients with DMD

Novel Mechanism Corrects Human Dystrophin Gene Resulting in a Functional Dystrophin Protein

Designed For Safety Through Lower Dose AAV

PBGENE-DMD Opportunity:

Potential to provide a first-in-class and best-in-class therapeutic for patients

High Unmet Need

Clear Regulatory Guidance

Growing Opportunity for Innovative Treatments

Today, Patients with DMD are in Dire Need with Limited Therapeutic Options

Ideal therapy would have:	Microdystrophin Gene Therapies	Exon Skipping Therapies	PBGENE-DMD Target Product Profile
Improved muscle function over time	✗	✗	✓
Long-term durable benefit	✗	✗	✓
Broadly applicable to patients	✓	✗	✓
Corrects human dystrophin gene resulting in a functional dystrophin protein	✗	✗	✓
Single administration	✓	✗	✓

Limited Benefits

PBGENE-DMD Target Product profile, Exon Skipper and Mirodystrophins based on data presentations and earnings release from related companies.

Clear Regulatory Guidance on Clinical Development Path to Approval

Validated Clinical Trial Design with Potential for Accelerated Approval

- Pivotal design and accelerated pathway aligned with the FDA in DMD space

Phase I/II

Dose Evaluation

Evaluate dose levels to establish initial clinical activity and determine go-forward dose level

Expansion Cohort

Expand study to add additional patients and evaluate therapeutic index

Phase III

Pivotal Study

Broader study aimed at evaluating effectiveness through **dystrophin % biomarker linked to demonstrated functional outcomes**

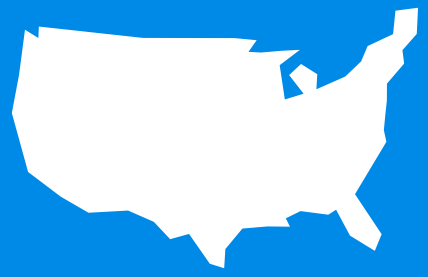
Regenx Bio Nov 2024 Press Release; Internal Analysis.

Global DMD Market Poised for Innovative Breakthroughs Like PBGENE-DMD

Prevalence & Incidence[1]

~300-400k
DMD Patients globally

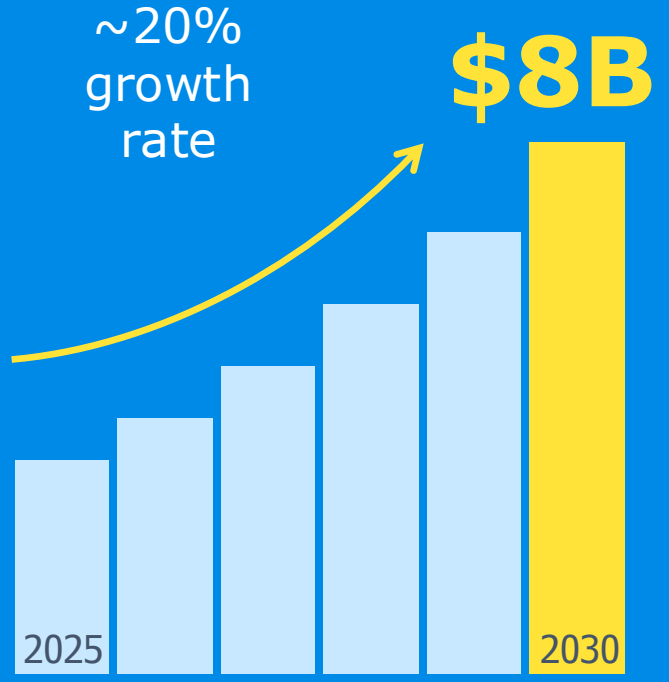
~15k
DMD Patients in US



>20k births
per year globally

~550 births
per year in US

Global Market Size Projection[2]

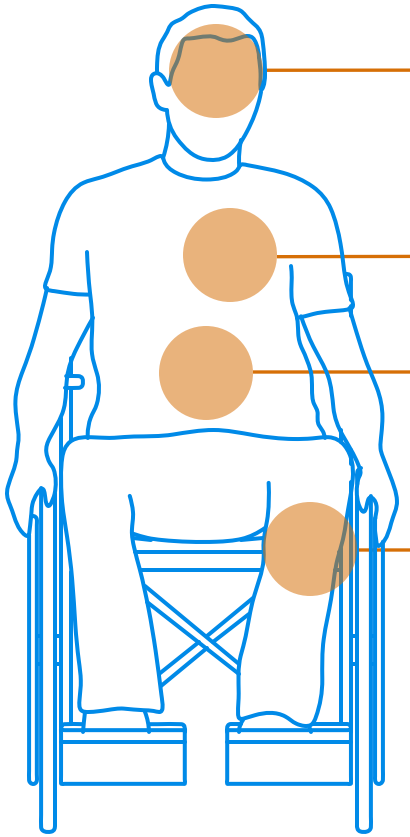


1. Prevalence and Incidence based on CureDuchenne and Orphanet Journal or Rare Diseases; k =1,000.
2. Market Size based on estimates from Evaluate Pharma 2025.

PBGENE-DMD Therapeutic Approach

Cassie Gorsuch, PhD
Chief Scientific Officer
Precision BioSciences, Inc

DMD is a Genetic Disorder Resulting in Progressive Muscle Degeneration and Early Death



Brain

- Learning disability or serious cognitive disability in a subset of patients

Heart

- Cardiomyopathy and arrythmias
- Eventual cardiac failure leading to premature death in late teens or 20s

Diaphragm & Intercostal Muscles

- Weakened respiratory muscles increase risk of infections
- Pulmonary insufficiency leading to premature death in late teens or 20s

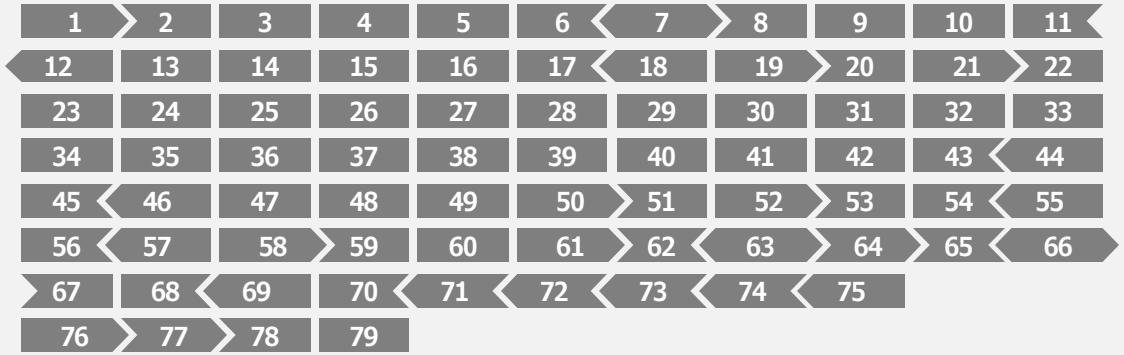
Skeletal Muscle

- Progressive muscle weakness leading to loss of ambulation around 12

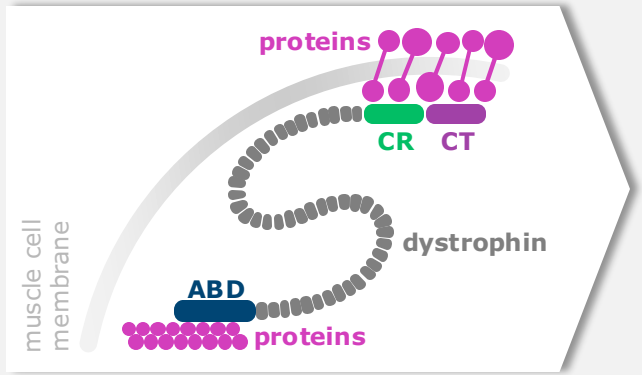
Content adapted from www.mda.org.

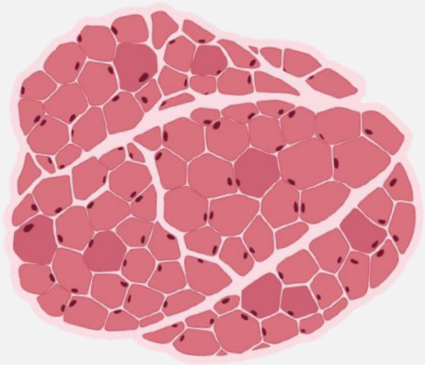
DMD is Caused by Mutations in the Dystrophin Gene That Prevent Production of Dystrophin Protein

Healthy



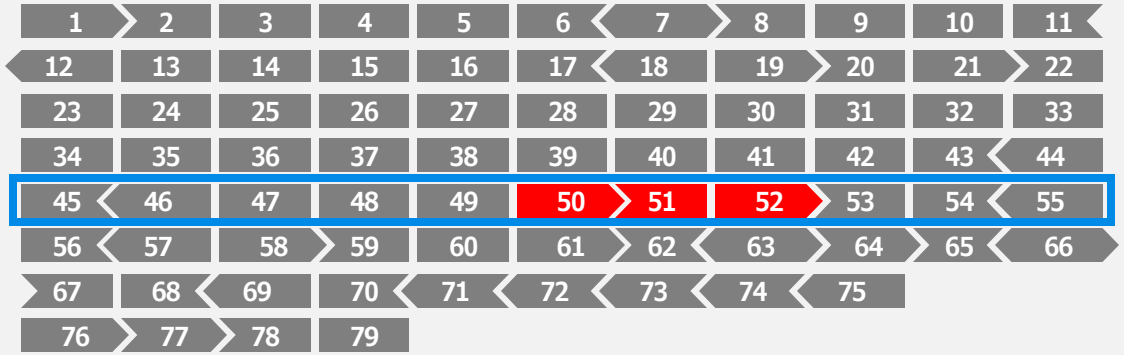
Dystrophin gene produces dystrophin protein, which is necessary for muscle maintenance and repair following injury





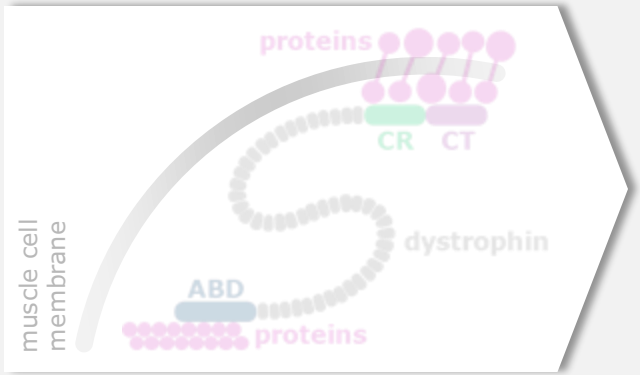
Healthy muscle

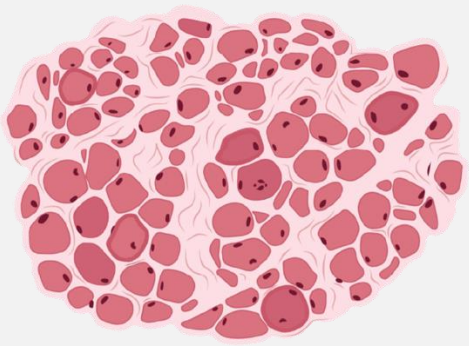
DMD



Mutations in exons prevent the production of dystrophin protein, causing DMD

Up to 60% of patients have mutations in exons 45-55



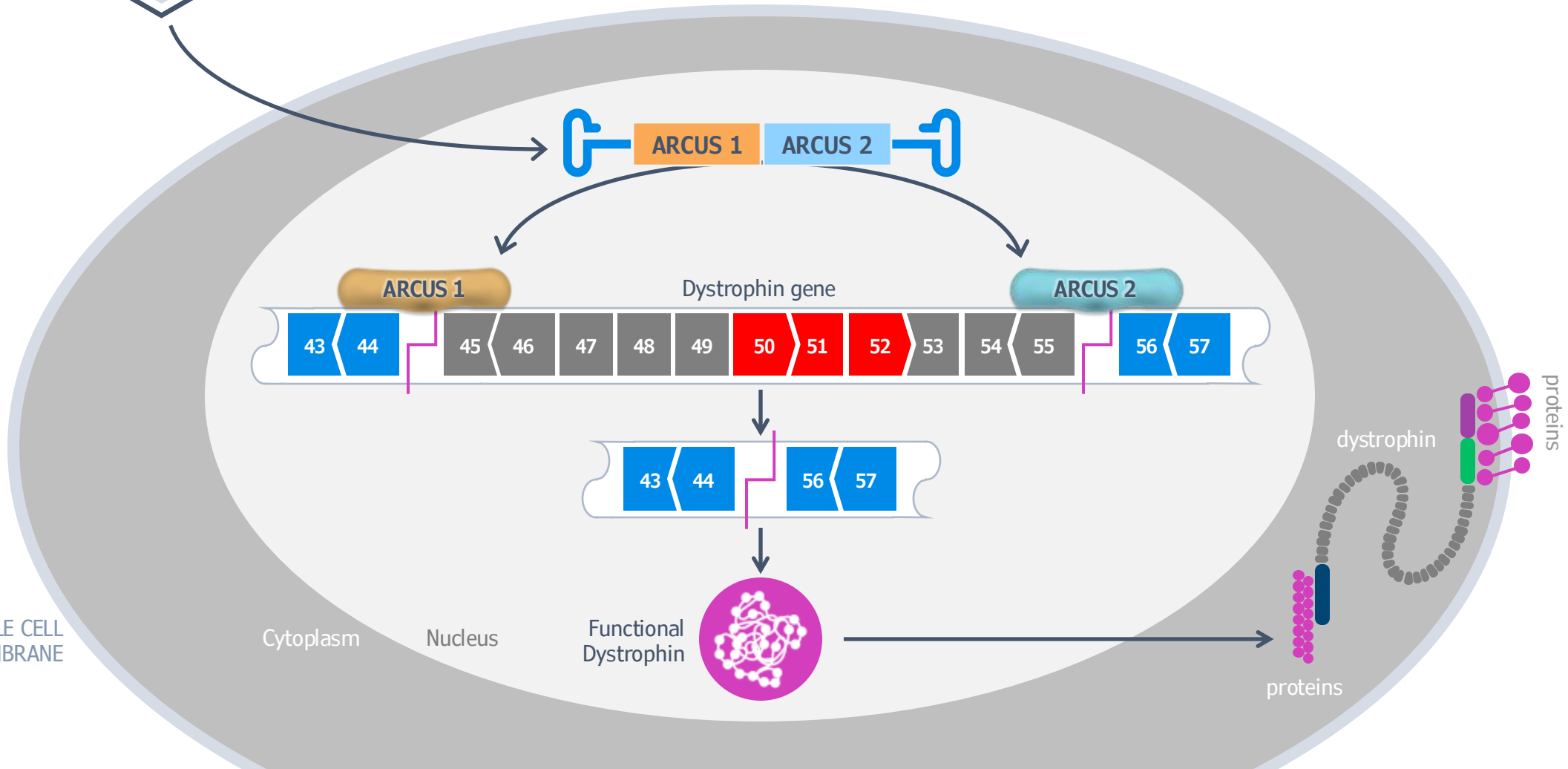


Degenerating muscle

Duan, D., Goemans, N., Takeda, S. et al. Duchenne muscular dystrophy. Nat Rev Dis Primers 7, 13 (2021). https://doi.org/10.1038/s41572-021-00248-3

PBGENE-DMD Designed to Provide Durable Functional Improvement for Majority Patients with DMD

PBGENE-DMD: A single AAV encodes two ARCUS proteins designed to permanently edit a patient's own DNA sequence, resulting in naturally-expressed, functional dystrophin



ARCUS is Uniquely Positioned for DMD Gene Editing

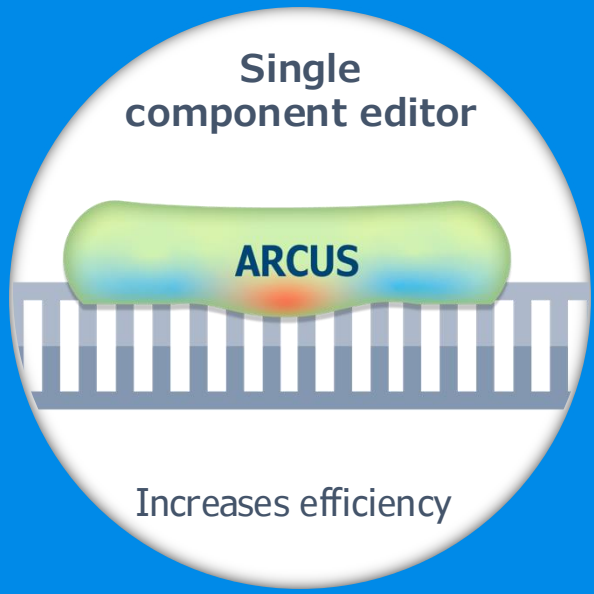
Why Other Gene Editors Can Not Follow

DMD: Safety and Efficacy

Predictable repair enhances reliability of excision; Unique cut allows for superior characterization of specificity

DMD: Safety and Efficacy

Both nucleases are delivered to cells in a single AAV, enabling lower doses and higher efficiency



DMD: Efficacy

Iterative protein engineering enabled co-evolution of two ARCUS nucleases for coordinated, efficient excision due to kinetic design

PBGENE-DMD is Designed to Provide Durable **Functional Muscle Improvement** for the Majority of Patients with DMD

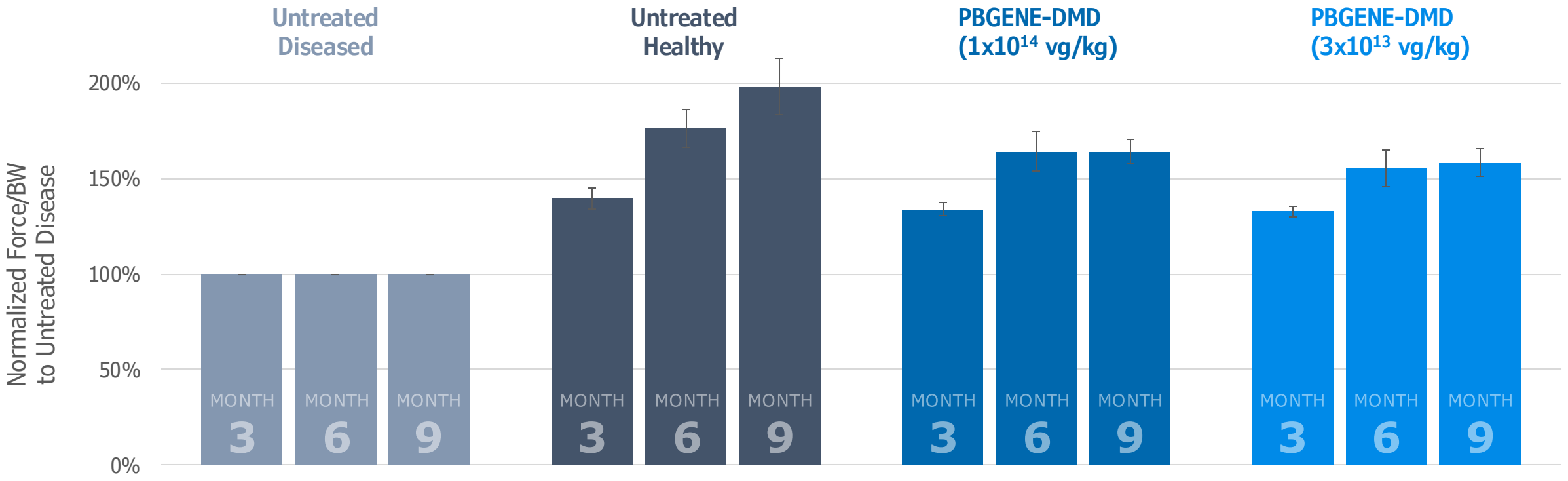
Novel Mechanism Corrects Human Dystrophin Gene Resulting in a Functional Dystrophin Protein

Designed For Safety Through Lower Dose AAV

PBGENE-DMD Showed Durable Functional Improvement Over Time

New Data

PBGENE-DMD Significantly Improved Muscle Function and Demonstrated Long-Term Durability



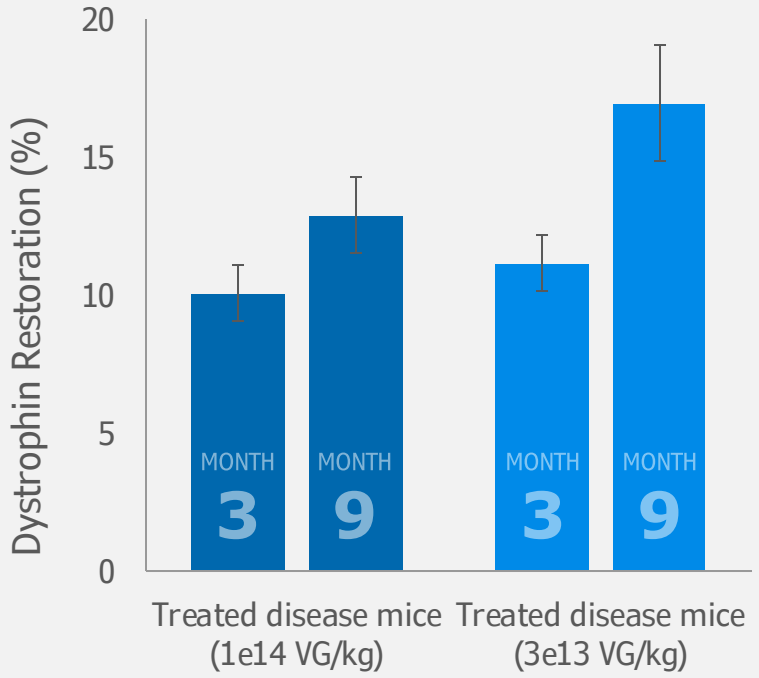
- Improved muscle function observed from 3 to 6 months.
- Durable functional improvements maintained out to 9 months.
- Benefit consistent across both experimental dose levels

Force was measured in the calf across multiple stimulation frequencies. Averaged force normalized to bodyweight is shown. Statistically significant ($p<0.001$) increases in force were observed in both doses of PBGENE-DMD compared untreated diseased animals at both time points.

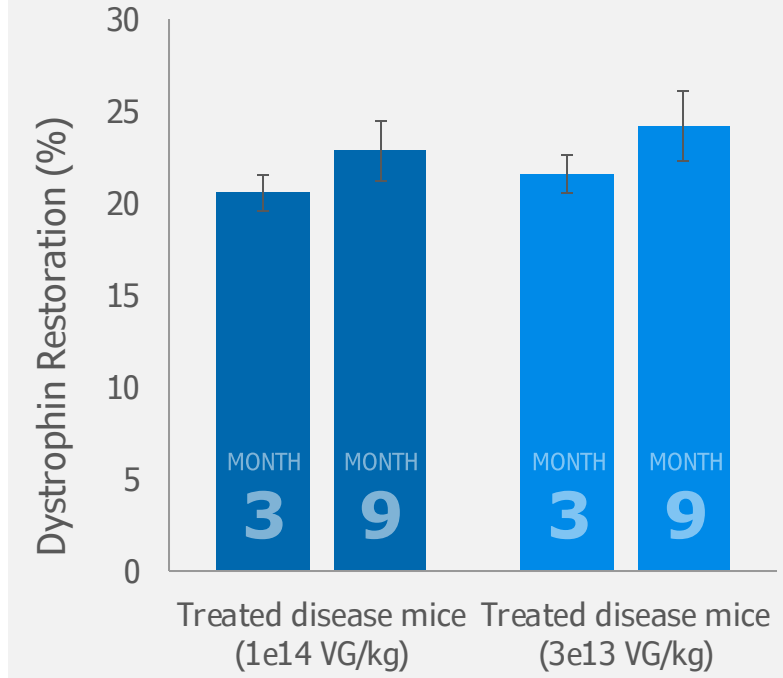
New Data

Long-term Functional Improvement Driven by Increasing and Stable Levels of Dystrophin Protein Expression

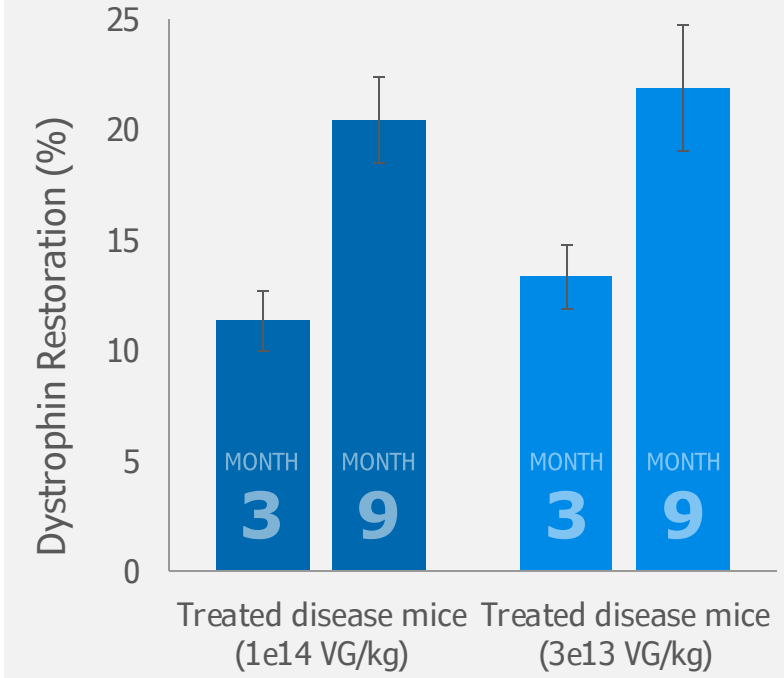
Heart



Calf

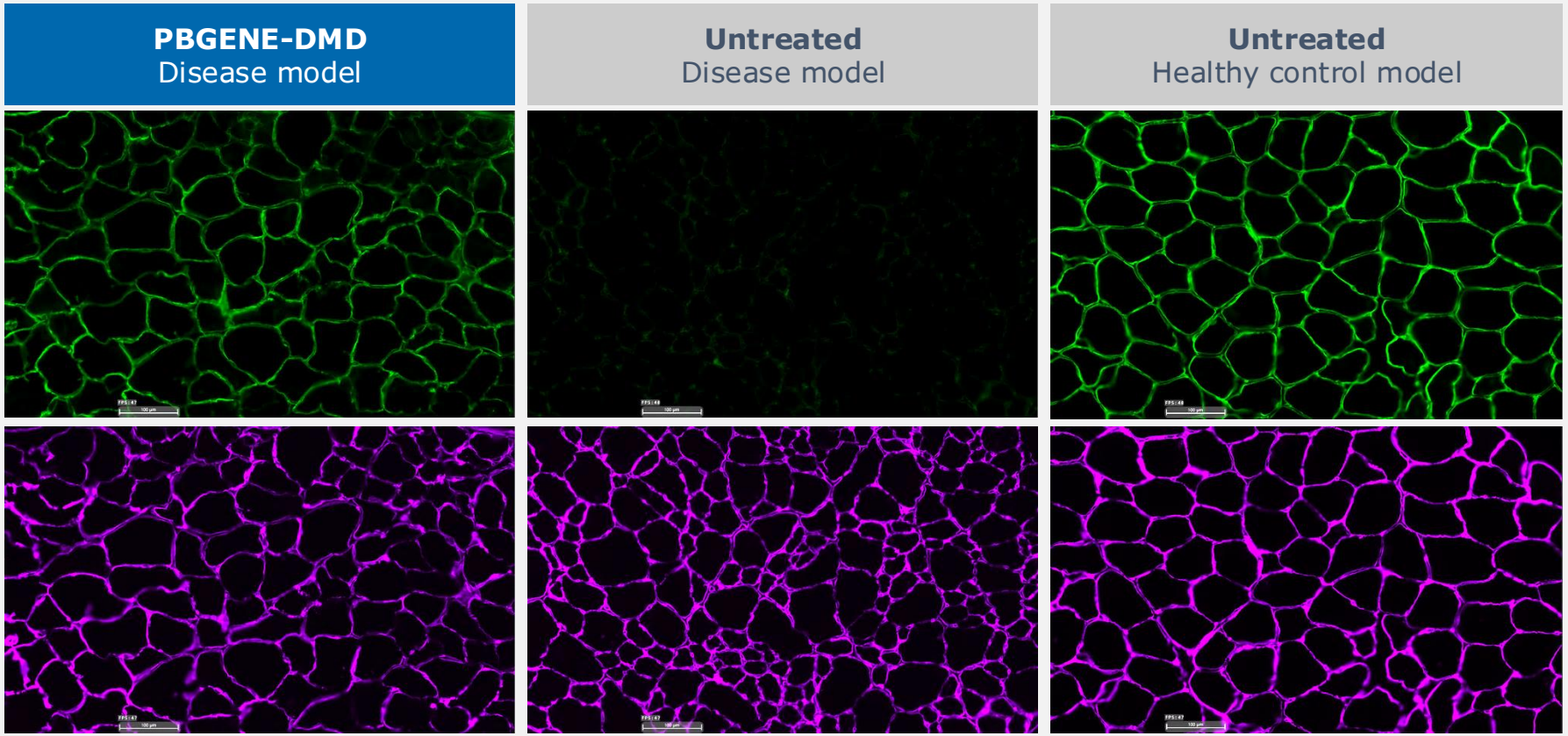


Quadricep



Naturally-produced, near full-length functional dystrophin protein increases through 9 months in mice

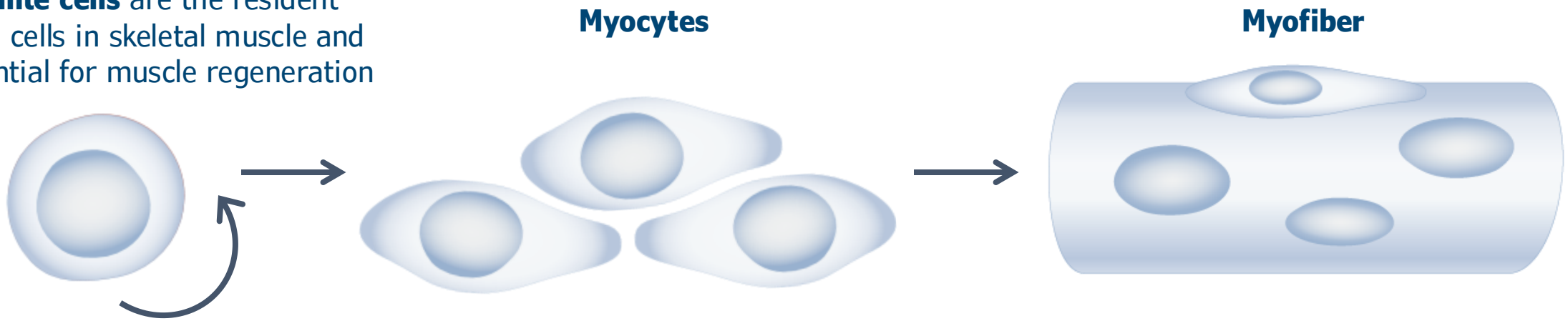
PBGENE-DMD Restored Functional Dystrophin Protein in Majority of Myofibers in Calf



Broad and substantial near full-length functional dystrophin restoration across muscles

Editing Satellite Cells is Essential for Durable Effect

Satellite cells are the resident stem cells in skeletal muscle and essential for muscle regeneration

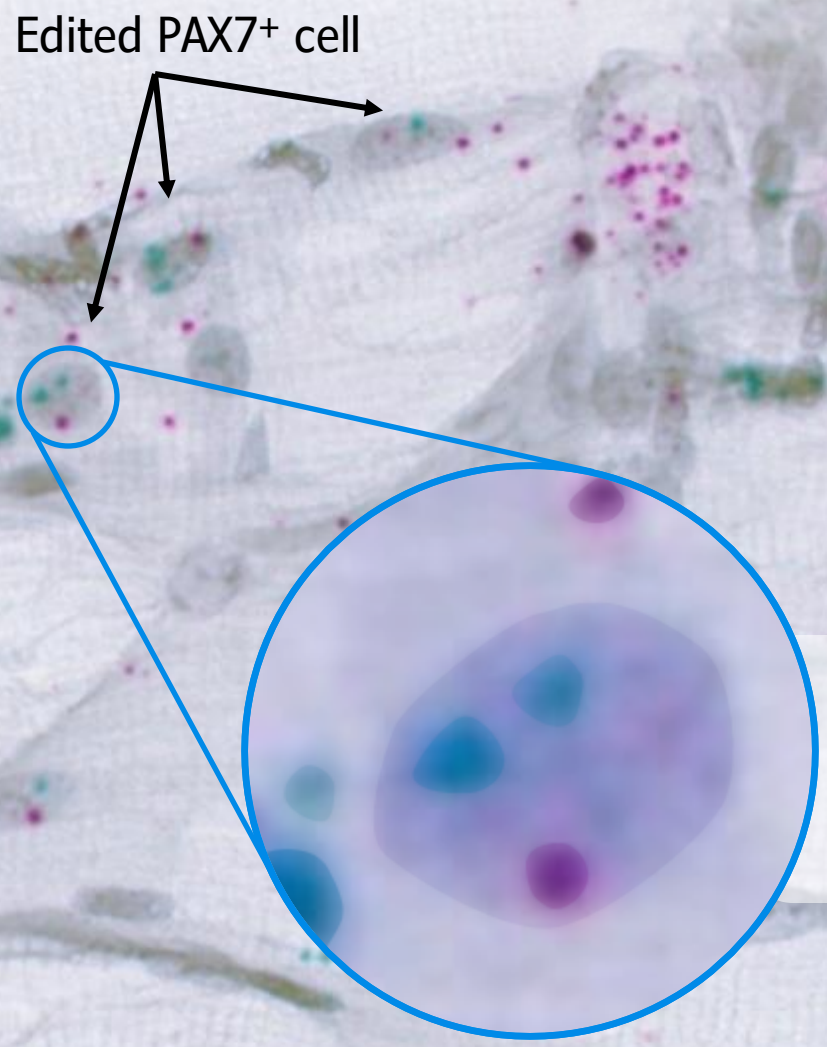


In DMD where myofiber degeneration is continuous,
editing satellite cells is essential for long-term therapeutic effect

PBGENE-DMD Edited Muscle Satellite Stem Cells, Providing Potential for Durable Functional Improvement

PBGENE-DMD has demonstrated permanent editing of satellite cells, beyond transient transduction

Observed edited dystrophin mRNA in PAX7+ cells, **a marker for muscle satellite stem cells**

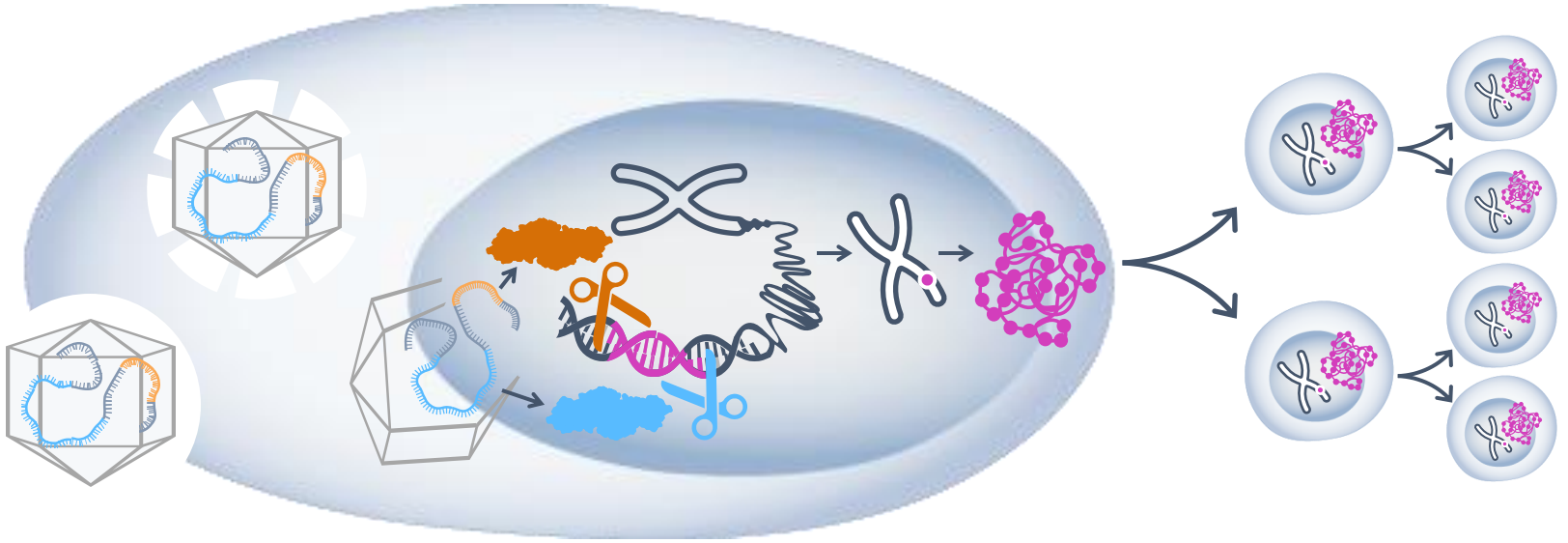


Pax7+
Edited DMD 44-56 mRNA

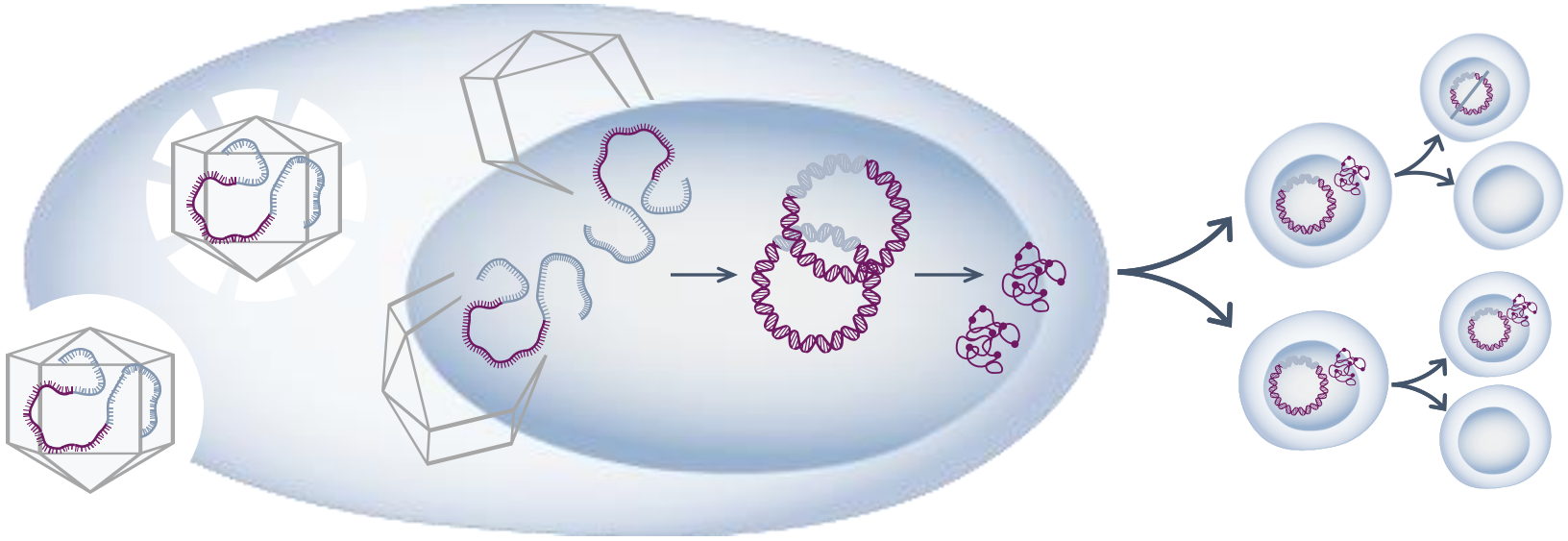
Novel Mechanism and Proven Dystrophin Protein

PBGENE-DMD's Novel Mechanism Potentially Enables Durable Improvements in Muscle Function Independent of the Persistence of AAV

PBGENE-DMD:
The dystrophin protein is expressed by the human genome, preventing the need for AAV persistence

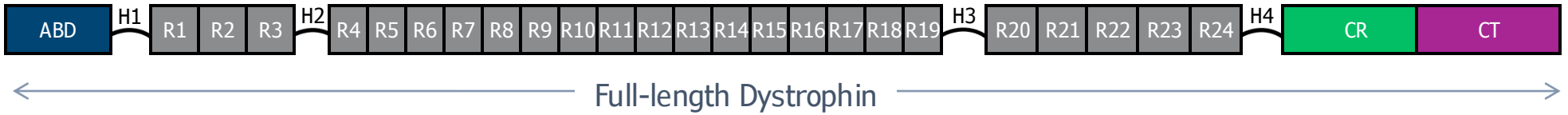


Microdystrophin Gene Therapies:
Microdystrophin protein is expressed from the AAV, requiring persistence of the AAV genome for microdystrophin expression

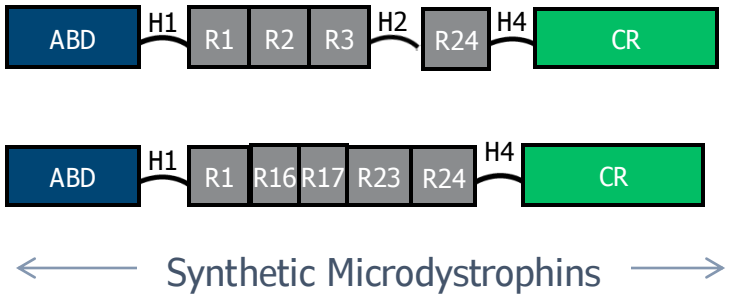


PBGENE-DMD Designed to Produce a Near Full-Length Dystrophin Protein, Proven to be Functional in Humans

Full-length Dystrophin



Truncated Synthetic Microdystrophins

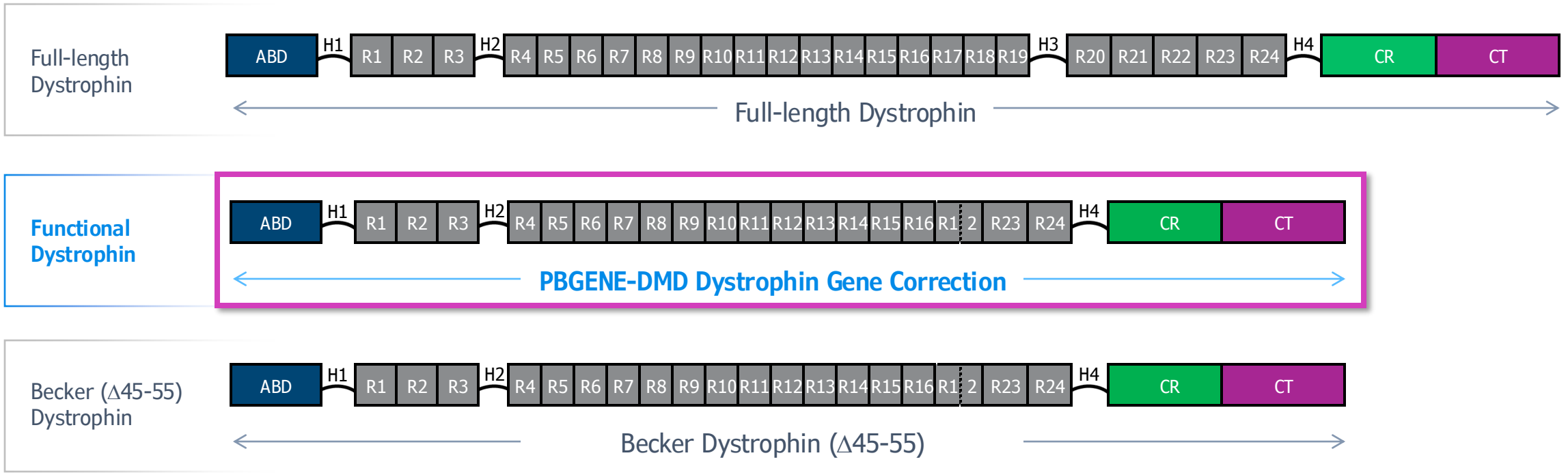


> "The truncated dystrophin apparently does not function quite as well as the full-length gene, even when overexpressed…"
>
> — Phelps et al

Functional Dystrophin

Phelps et al. *Hum Mol Genet.* 1995 Aug;4(8):1251-8.

PBGENE-DMD Designed to Produce a Near Full-Length Dystrophin Protein, Proven to be Functional in Humans



PBGENE-DMD functional dystrophin is present in a subset of Becker patients, who often have mild to asymptomatic phenotypes[1]

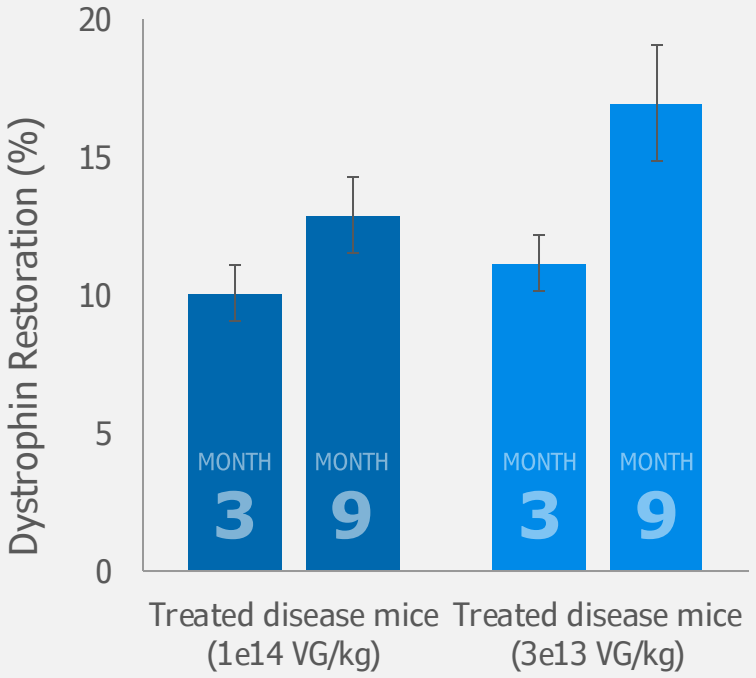
It is expected that as little as 5% expression of functional dystrophin protein is needed to provide therapeutic benefit in DMD patients[2]

1. Taglia et al. *Acta Myol*, 2015 May;34(1):9-13. 2. Feraudy et al. *Ann Neurol*, 2021 Feb;89(2):280-292.

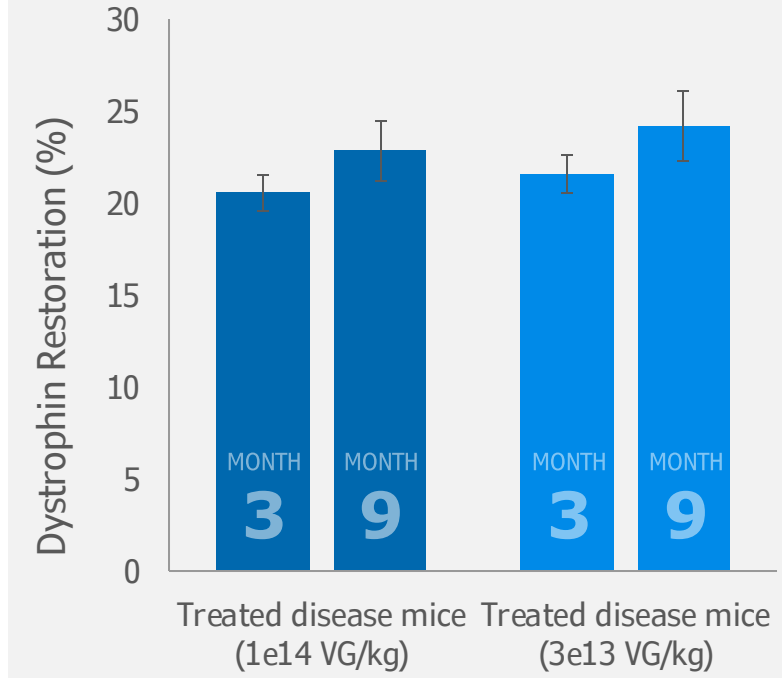
New Data

Long-term Functional Improvement Driven by Increasing and Stable Levels of Dystrophin Protein Expression

Heart



Calf



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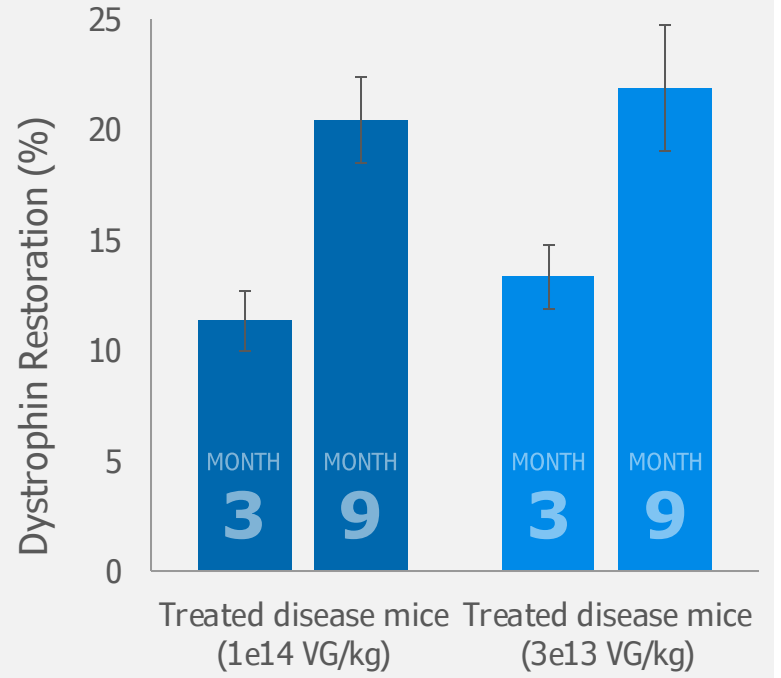
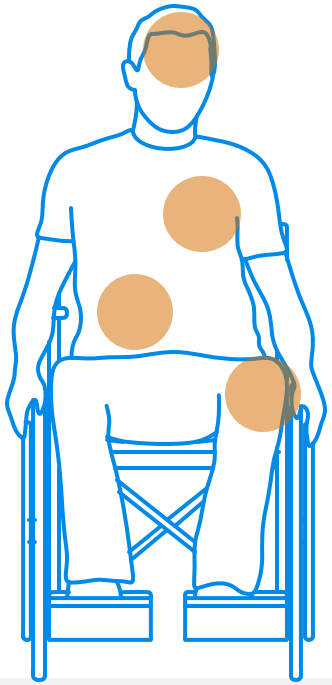


Naturally-produced, near full-length functional dystrophin protein increases through 9 months in mice

Near Full Length Dystrophin Protein Has Proven Function in Individuals with Dystrophin Del45-55 Genotype



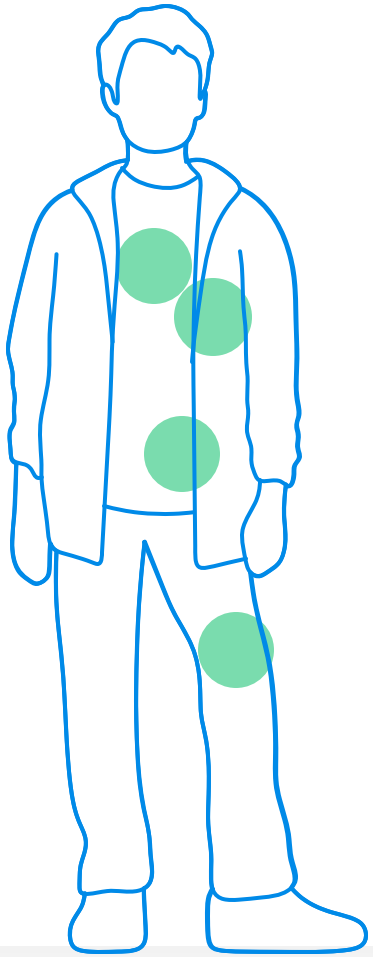
Lifespan:

- Early death in late teens or 20s

Clinical Presentation:

- Progressive muscle weakness leading to loss of ambulation
- Respiratory difficulties often contributing to early death
- Cardiac complications contributing to early death
- Neurological impairment in some patients

Out of frame dystrophin gene (DMD)



Lifespan:

- Can live into 60-70s[1-3]

Clinical Presentation:

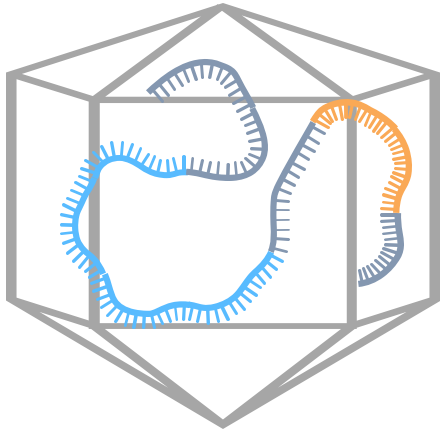
- Asymptomatic or mild symptoms[1-4]
- Normal muscle strength and ambulation throughout life[1,2]
- Normal respiratory function[2]
- Occasional myocardial involvement, manageable with medication[2]

Del45-55 in-frame Dystrophin gene

1. Taglia A, et al. *Acta Myol*. 2015;34(1):9-13. 2. Echigoya Y, et al. *J Pers Med*. 2018;8(4):41. 3. Béroud C, et al. *Hum Mutat*. 2007;28(2):196-202.
4. Nakamura A. *J Hum Genet*. 2017;62(10):871-876.

PBGENE-DMD Designed With Safety Top Of Mind

PBGENE-DMD Designed For Safety Through Low Dose and High Quality AAV



Permanently correct the gene using just enough AAV

Because PBGENE-DMD corrects the DNA, AAV persistence is not necessary, and therefore lowest efficacious dose is the goal

Consistently produce high quality AAV

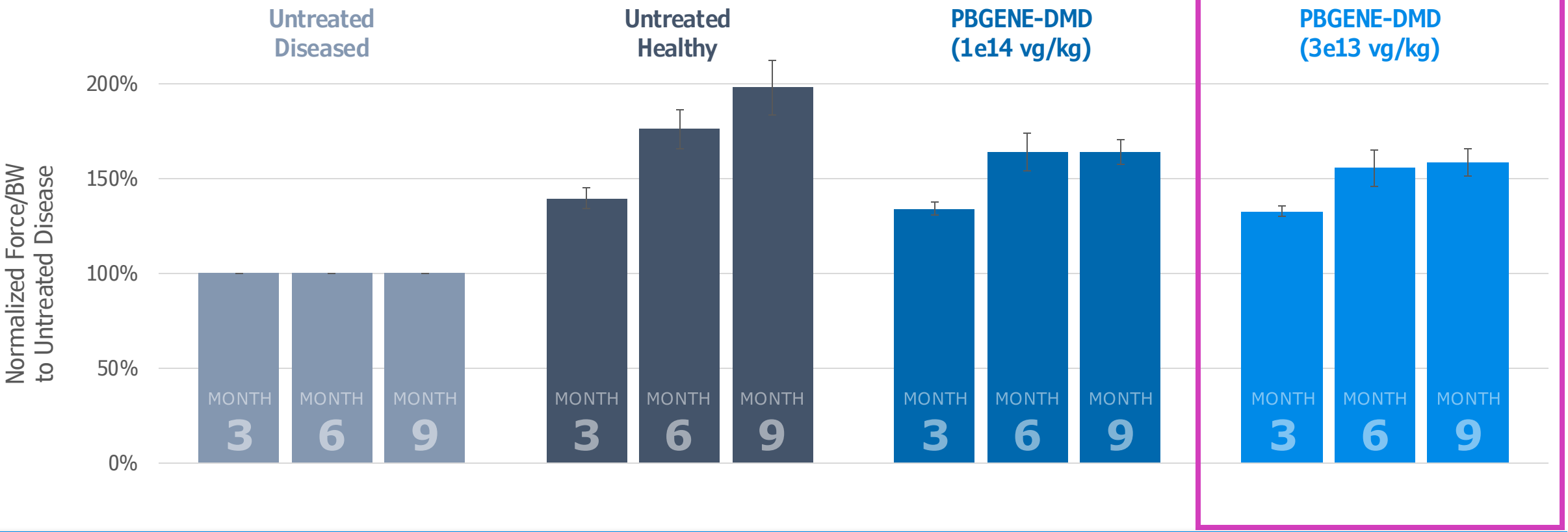
Precision BioSciences has developed capability to produce high quality AAV with >85% full capsid ratios, designed to improve safety and efficacy compared to earlier generation AAV products

Experienced, world-class clinical teams

Taking advantage of the extensive clinical experience of AAV, as well as effective immunosuppression regimens

New Data

PBGENE-DMD Significantly Improved Muscle Function at even the Lowest Dose Level



Significant Improvement in Muscle Force Even with the Lowest Dose Level AAV

Force was measured in the calf across multiple stimulation frequencies. Averaged force normalized to bodyweight is shown. Statistically significant ($p<0.001$) increases in force were observed in both doses of PBGENE-DMD compared untreated diseased animals at both time points.

PBGENE-DMD: Highly Desirable Target Product Profile

Ideal therapy would have:	PBGENE-DMD Potential to Provide Best-In-Class Therapeutic Profile
Improvements in muscle function	✓ Nonclinical evidence of muscle function improvement over time
Long-term durable benefit	✓ Data demonstrating improvement is durable to at least 9 months in mice
Broadly applicable to patients	✓ Up to 60% of patients
Corrects human dystrophin gene resulting in a functional dystrophin protein	✓ Protein produced is known to have functional benefit in humans as evidenced by Del45-55 subset of Becker patients with favorable prognosis
Single administration	✓ One time administration
Good Safety	✓ PBGENE-DMD has been safe in nonclinical studies to date
Ability to reach skeletal and cardiac muscles	✓ Robust protein expression across broad range of skeletal and cardiac muscles in mice

KOL Panel Discussion

Russell Butterfield, MD, PhD

Director, Center for Gene Therapy

Associate Professor, Child Neurology

University of Utah, School of Medicine

Cassie Gorsuch, PhD

Chief Scientific Officer

Precision BioSciences, Inc

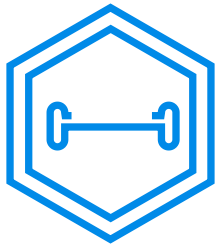
Program Next Steps

Including Implications to the PBGENE-3243 Program

PBGENE-DMD Program is Quickly Moving Towards Clinical Stage

- [x] Identified Clinical Candidate
- [x] Received Positive Pre-IND Feedback From FDA
- [x] Initiated GLP Toxicology Studies
- [ ] Manufacture Clinical Trial Material
- [ ] Target Filing Of CTA and/or IND in 2025
- [ ] Initiate First-in-Human Clinical Study

PBGENE-3243 Program Development Paused —*Exploring Opportunities for Advancement*



Precision BioSciences remains committed to our goal of continuing to advance ARCUS-based therapies for patients with high unmet needs, such as patients with mitochondrial diseases

We are encouraged by the preclinical data supporting the potential for PBGENE-3243 to provide therapeutically meaningful levels of heteroplasmy shift and continue to explore opportunities to advance this program internally or through a partnership

We are grateful to the mitochondrial community including all our current partners and hope to continue working together in the future

Precision BioSciences is a Clinical Stage *In Vivo* Gene Editing Company

Multiple Programs in or Nearing Clinical Data

		Expected First Clinical Data Timing
Wholly owned	**PBGENE-HBV** ***In Vivo* Gene Editing Program for Chronic Hepatitis B** 〉 Investigational New Drug (IND) clearance and Fast Track designation received in United States 〉 Global phase 1/2a ELIMINATE-B trial on-going with initial data demonstrating safety and antiviral activity with HBsAg reductions 〉 Data updates expected throughout 2025	**2025** eliminate B
	PBGENE-DMD ***In Vivo* Gene Editing Program Duchenne Muscular Dystrophy** 〉 PBGENE-DMD is Precision's second wholly-owned program 〉 Targeting IND and/or CTA filing in 2025 〉 Potentially eligible for Priority Review Voucher (PRV) valued approximately $150M upon BLA approval	**2026**
Partnered Program Employing ARCUS Nuclease	**iECURE-OTC*** 〉 Global phase 1 trial on-going in U.S., U.K., Australia and Spain 〉 Complete clinical response in first patient dosed; OTC-HOPE Phase 1/2 trial expected to finish enrollment in 2025 with full data in 1H 2026 〉 Initial clinical validation for ARCUS *in vivo* gene editing	**2025**

Three Clinical Datasets Read-Out through 2026, Within Cash Runway

*Also known as ECUR-506; approved for clinical trials by the U.S. Food and Drug Administration (FDA), U.K. Medicines & Healthcare products Regulatory Agency (MHRA), Australian Therapeutic Goods Administration (TGA) and Spanish Agency of Medicines and Medical Devices (AEMPS) in Spain. iECURE responsible for all development costs for ECUR-506

Q&A