PRECISION
BIOSCIENCES

Precision BioSciences PBGENE-DMD Investor Update

March 17, 2026

Forward-Looking Statements

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. All statements contained in this presentation that do not relate to matters of historical fact should be considered forward-looking statements, including, without limitation, statements regarding the key advantages of ARCUS and its key capabilities and differentiating characteristics; expectations about operational initiatives, strategies, further development, or timing of additional updates or data releases of PBGENE-HBV and PBGENE-DMD, timing and progress of IRB processes and site activations following IND clearance for the PBGENE-DMD program and FUNCTION-DMD trial; expectations to share further clinical data from the PBGENE-HBV programs at hepatitis-focused medical conferences throughout 2026; exploring multiple levers (dose level, number of dose administration & time between dose administrations) in the ELIMINATE-B clinical trial to optimize the therapeutic index of PBGENE-HBV while continuing Cohorts 3, 4 & 5 in parallel; anticipating initiating Part 2 of ELIMINATE-B after choosing optimal dosing regimen to stop nucleos(t)ide analog treatment; the suggestion of good translatability of PBGENE-DMD because the drug demonstrated as good or better transduction in nonhuman primates compared to mice; the design of PBGENE-DMD to improve function over time and address 60% of patients with DMD; the design of PBGENE-DMD to excise the 'hot-spot' region between exons 45-55 of the dystrophin gene with goal of permanently and safely restoring muscle function for the majority of patients living with DMD; the belief that PGBENE-DMD has the potential to surpass the current benchmark for functional improvement in DMD and looks to improve upon existing therapies and provide a near full length dystrophin protein and first-in-class and best-in-class therapeutic for patients ; the expectation that as little as 5% expression of functional dystrophin protein (full length or near full length dystrophin protein) is needed to provide therapeutic benefit in DMD patients; the expectation that AAV persistence not required for potential long-term effect of PBGENE-DMD because the dystrophin protein is express by the human genome; PBGENE-DMD's aim to minimize empty capsids with a >90% capsid fill ratio in manufacturing, reducing total capsids dosed and potentially improving safety; the design of the PBGENE-DMD clinical study for safety and speed and at lower or equal doses to all other AAV DMD programs approved or in development; PBGENE-DMD's immune modulation regimen (IMR) strategy designed to optimize monitoring and interventions to patient safety needs during two key safety risk periods; the expectation of clinical data for PBGENE-DMD across multiple patients in 2026; target enrollment 3-5 patients in the FUNCTION-DMD clinical trial in 2026, generation of safety data and early efficacy assessed by percentage of near full-length dystrophin protein expression from muscle biopsies at 12 weeks; translation of results in preclinical studies of ARCUS nucleases (including PBGENE-DMD) to clinical studies in humans; the preclinical and clinical development and demonstrated, potential and expected safety, efficacy, durability, and benefit of PBGENE-HBV and PBGENE-DMD, as well as our other product candidates and those being developed by partners; expectations of additional clinical data from the ongoing OTC-HOPE clinical trial in the first half of 2026; and the sufficiency of our cash runway through multiple catalysts through the end of 2028. In some cases, you can identify forward-looking statements by terms such as "aim," "anticipate," "approach," "belief", "believe," "contemplate," "could," "design," "designed," "estimate," "expect," "goal," "intend," "look," "may," "mission," "plan," "possible," "potential," "predict," "project," "pursue," "should," "strive," "suggest," "target," "will," "would," or the negative thereof and similar words and 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, and involve a 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 our capital requirements, anticipated cash runway, requirements under our current debt instruments and effects of restrictions thereunder, including our ability to raise additional capital due to market conditions and/or our market capitalization; our operating expenses and our ability to predict what those expenses will be; our limited operating history; the progression and 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; the risk that other genome-editing technologies may provide significant advantages over our ARCUS technology; our dependence on our ARCUS technology; the initiation, cost, timing, progress, achievement of milestones and results of research and development activities and preclinical and clinical studies, including clinical trial and investigational new drug applications; public perception about genome editing technology and its applications; competition in the genome editing, biopharmaceutical, and biotechnology fields; our or our collaborators' or other licensees' ability to identify, develop and commercialize product candidates; pending and potential product liability lawsuits and penalties against us or our collaborators or other licensees related to our technology and our product candidates; the U.S. and foreign regulatory landscape applicable to our and our collaborators' or other licensees' development of product candidates; our or our collaborators' or other licensees' ability to advance product candidates into, and successfully design, implement and complete, clinical trials; potential manufacturing problems associated with the development or commercialization of any of our product candidates; delays or difficulties in our and our collaborators' and other licensees' ability to enroll patients; results of preclinical studies and early clinical trials of product candidates may not be predictive of results of later studies or trials; 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; effects of system failures, cyber-attacks, and security breaches; our ability to obtain orphan drug designation, fast track designation, rare pediatric disease designation, or a priority review voucher for our product candidates, or to realize the expected benefits of these designations; our or our licensees' 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' or other licensees' 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; the success of our existing collaboration and other license 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 Annual Report on Form 10-K for the annual period ended December 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 have no 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

Alex Kelly
Chief Financial Officer
Precision BioSciences

PRECISION BIOSCIENCES

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

ARCUS:

Precision's Proprietary Gene Editing Platform

- ARCUS wholly owned by Precision BioSciences
- Derived from the homing endonuclease I-CreI found in green algae
- Naturally evolved to drive high efficiency editing
- >75 patents issued covering ARCUS and in vivo gene editing

ARCUS WORKS:
ARCUS Platform Has Multiple Points of Clinical Validation Led by Wholly Owned HBV Program

Clinical Stage Programs Validating ARCUS

Wholly Owned Programs

eliminate-B

PBGENE-HBV for Chronic Hepatitis B

- Viral gene elimination program with over 30 doses delivered across 13 patients treated in 5 cohorts to date. Responses at all dose levels reported to date
- **Additional clinical data at medical conferences throughout 2026**

functionDMD

PBGENE-DMD for Duchenne Muscular Dystrophy

- PBGENE-DMD is designed to provide durable functional muscle improvement targeting ~60% of patients with DMD
- Site activation underway following FDA IND Clearance, with initial data across multiple patients expected in late-2026

Partnered Programs

ECURE

ECUR-506 for OTC Deficiency

- First gene insertion program, utilizing ARCUS nuclease
- First patient treated and reported in Phase I study in a Complete Response
- Next clinical update in 1H 2026

IMUGENE

Azer-cel CAR T in Hematology

- Clinically tested in ~100 patients
- Pivotal trial design aligned with FDA in late 2025 for investigation in relapsed DLBCL setting. Precision earned $8.0M milestone in October 2025

TG Therapeutics

Azer-cel CAR T in Autoimmune indications

- Continued phase 1 clinical trial of azer-cel in progressive forms of multiple sclerosis
- Precision earned $7.5M for achievement of recent clinical milestone

The Focus for Today is Our Second Wholly Owned Clinical-Stage Program: **PBGENE-DMD**

Cassie Gorsuch, PhD
Chief Scientific Officer
Precision BioSciences

functionDMD

PBGENE-DMD: designed to excise the ‘hot-spot’ region between exons 45-55 of the dystrophin gene with goal of **permanently and safely restoring muscle function for the majority of patients** living with DMD

Despite the Emergence of Novel Therapies, We Believe our Approach Has the Potential to Surpass the Current Benchmark for Functional Improvement

Current Therapies Approved or in Development Face Multiple Limitations

EXAMPLES OF MICRODYSTROPHIN APPROACHES

REGENXBIO · SAREPTA THERAPEUTICS · SOLID BIOSCIENCES

- Transient Expression of Truncated Synthetic Dystrophin Proteins Missing Essential Protein Domains
- High AAV Doses Needed, Effect Expected to Dilute Over Time
- Slowing Decline, but Not Maintaining or Improving Function

EXAMPLES OF EXON SKIPPERS

SAREPTA THERAPEUTICS · WAVE LIFE SCIENCES · Dyne THERAPEUTICS

- Limited to Small Subsets of Patients
- Require Continuous Dosing
- Managing Potential Toxicities Over Time (e.g., renal)

PBGENE-DMD: Target Product Profile

PBGENE-DMD Potential to Provide Best-In-Class Therapeutic Profile

An ideal therapy would achieve the following:

- ☑ Broadly applicable to patients
- ☑ Manageable safety profile
- ☑ Improvements in muscle function as the gold standard
- ☑ Ability to reach skeletal, cardiac and diaphragm muscles
- ☑ Long-term durable benefit
- ☑ Correct the human dystrophin gene resulting in a functional dystrophin protein expression
- ☑ Single administration

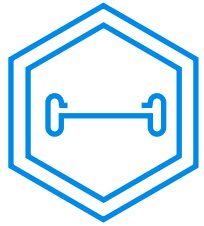
DMD, Duchenne muscular dystrophy.

Reasons to Believe in FUNCTION-DMD Phase 1/2 Trial:

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

Unmet Need Persists

Patients need better treatment options that provide proven muscle function improvements



ARCUS Gene editing

Addressing the root cause of DMD at the DNA level with the goal of long-term durability

Robust Preclinical Data

Increased dystrophin protein leading to improving muscle function across multiple time points in diseased mouse model

Clinical Trial Designed for Safety

Right patient selection criteria and comprehensive immunosuppression regimen alongside safety monitoring plan

World Class DMD Clinical Trial Sites

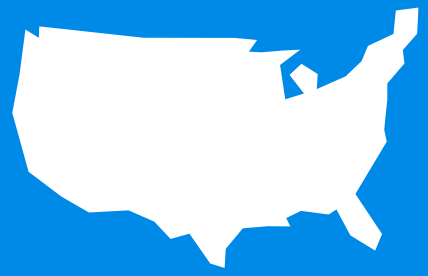
Top DMD clinical sites with highly experienced investigators and relevant clinical trial expertise

Global DMD Market Poised for Innovative Breakthroughs Like PBGENE-DMD

Prevalence & Incidence[1]

~300-400k
DMD Patients globally

~15k
DMD Patients in US

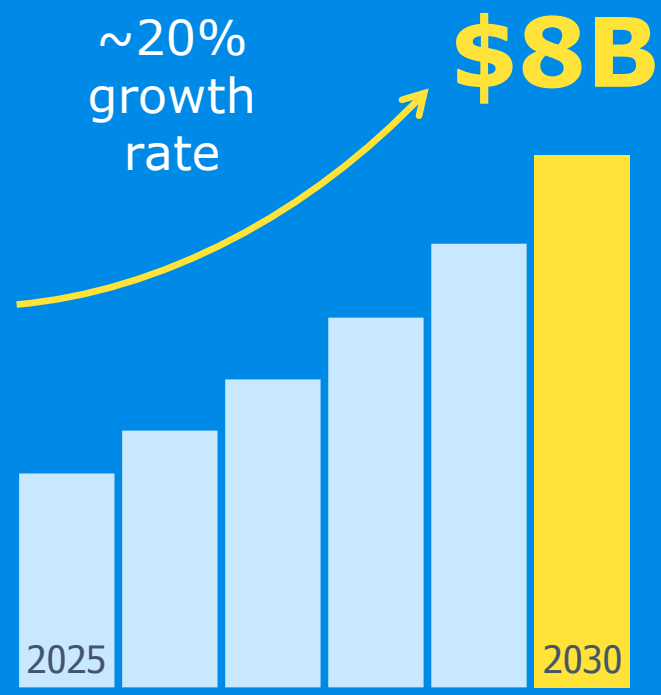


>20k births
per year globally

~550 births
per year in US

★ **Up to 60% of these patients are eligible for PBGENE-DMD**

Global Market Size Projection[2]

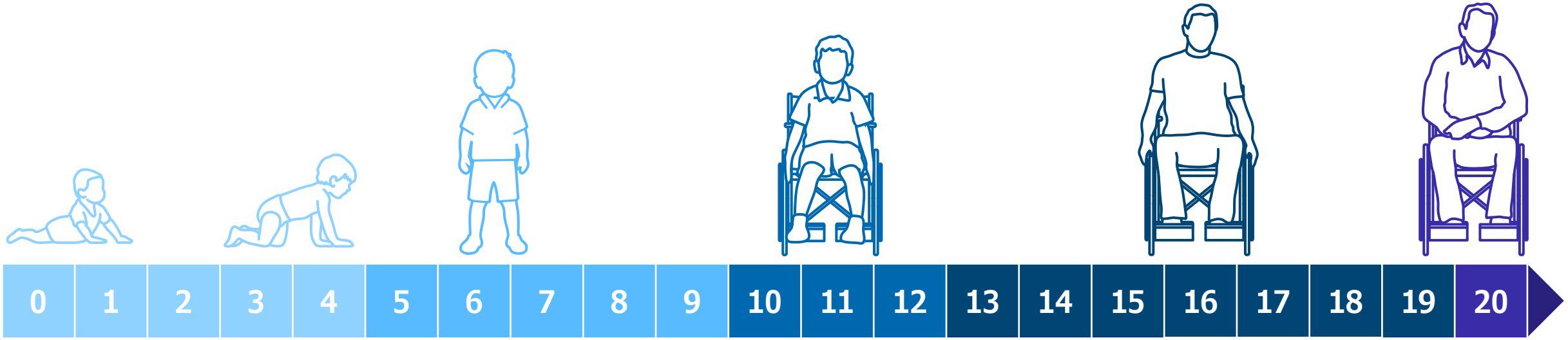


B, billion; DMD, Duchenne muscular dystrophy.
1. Prevalence and Incidence based on CureDuchenne and Orphanet Journal or Rare Diseases; k =1,000. 2. Market Size based on estimates from Evaluate Pharma 2025.

The Unmet Need Persists Despite Advancements in DMD Therapeutic Development

Pat Furlong
President & Founder
Parent Project Muscular Dystrophy (PPMD)

DMD is a Progressive Neuromuscular Disorder Leading to Early Mortality



- **From birth:** Damage starts right away, but many boys look and act **typical at first**
- **Early clues** are things like **delays in sitting, walking, running,** or **getting up** from the floor

- **Muscle weakness** often becomes clinically noticeable by age 5
- At this age, **muscle strength, stamina, and walking ability** begin to **decline** with age

- **Walking gets tougher** and slower
- Arms weaken, stamina drops
- **Wheelchairs** often become part of daily life

- **Full-time** wheelchair use is full time & more help is needed with daily tasks
- **Continued** decline in upper-limb function
- **Increasing** respiratory and trunk muscle involvement
- **Rising need for supportive care** and assistive devices

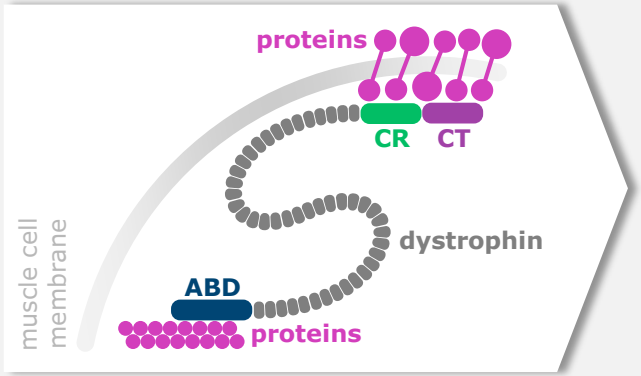
- In late-stage disease, **severe heart and lung complications** are a leading cause of **mortality**

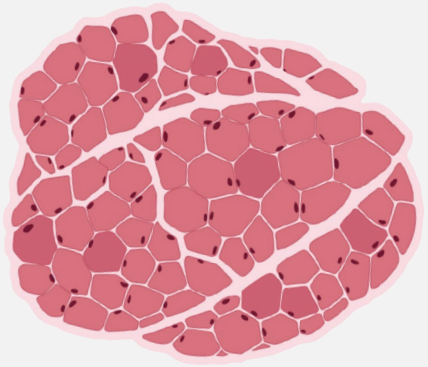
DMD, Duchenne muscular dystrophy.

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

Healthy

Dystrophin protein is necessary for muscle maintenance and repair following injury

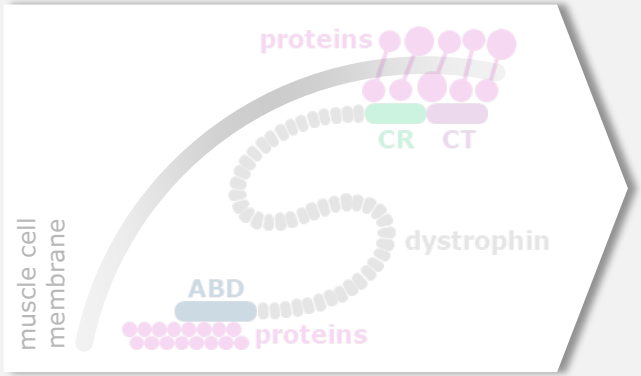


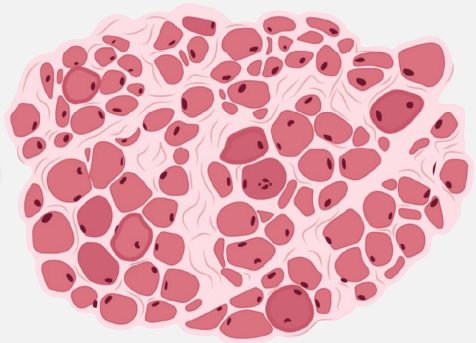


Healthy muscle

DMD

DMD is caused by mutations in the dystrophin gene prevent the production of functional dystrophin protein





Degenerating muscle

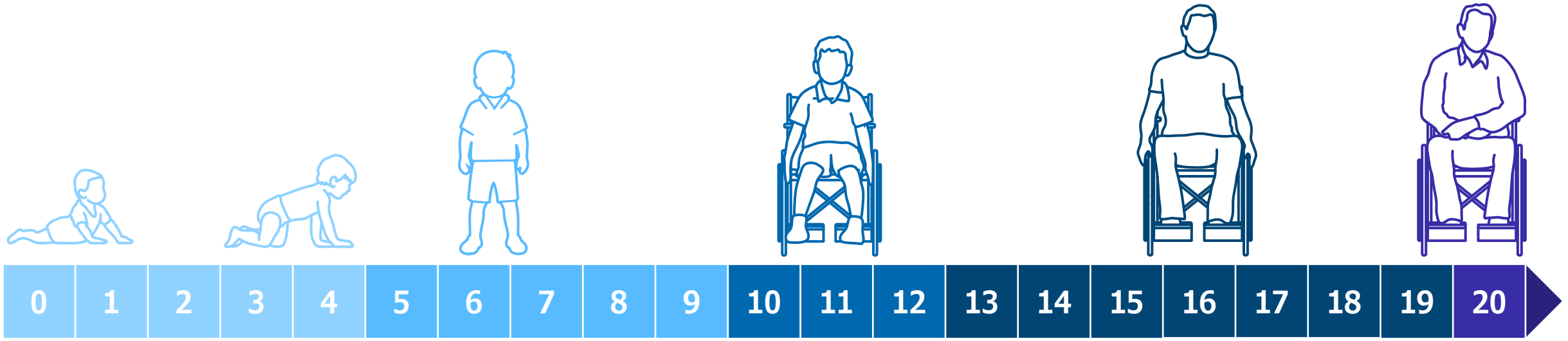
The lack of dystrophin protein leads to loss of muscle integrity and function

DMD, Duchenne muscular dystrophy.

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

The Unmet Needs Persists:

The ideal therapy would allow for **early intervention** to promote **increased muscle function**



Despite advancements in DMD therapies, the unmet need persists and continued investment in novel approaches like PBGENE-DMD are essential

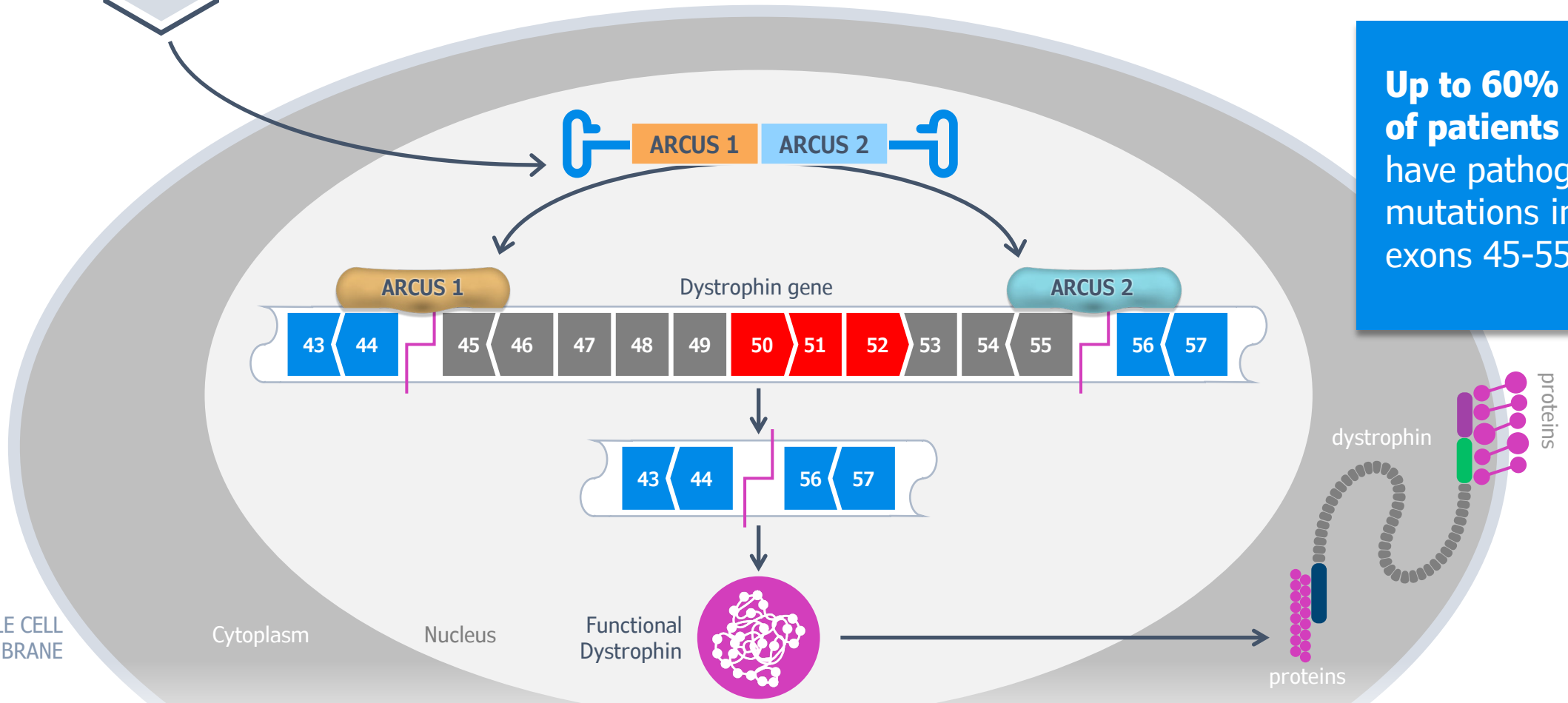
DMD, Duchenne muscular dystrophy.

PBGENE-DMD Reason-to-Believe: Novel Therapeutic Approach

Cassie Gorsuch, PhD
Chief Scientific Officer
Precision BioSciences

PBGENE-DMD Designed to Provide Durable Functional Improvement for 60% of 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



Up to 60% of patients have pathogenic mutations in exons 45-55[1]

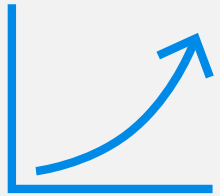
AAV, adeno-associated virus; DNA, deoxyribonucleic acid; DMD, Duchenne Muscular Dystrophy; DNA, deoxyribonucleic acid.

1. Beroud et al. *Hum Mutat.* 2007 Feb;28(2):196-202.

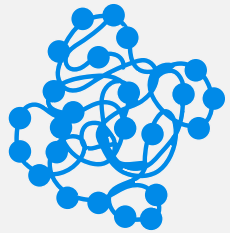
PBGENE-DMD Looks to Improve Upon Existing Therapies for DMD

Broadly applicable to most DMD patients

Improved muscle function over time



Long-term, durable benefit

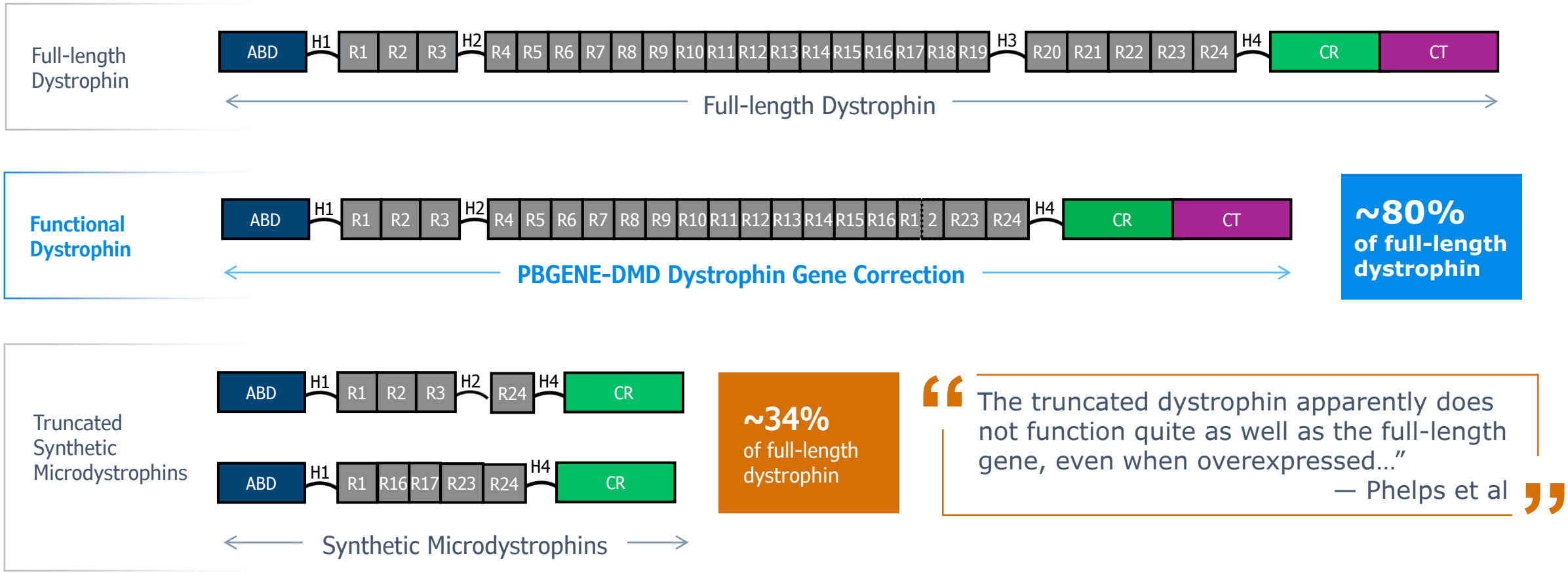


Near-full length dystrophin protein, known to function in humans

Single administration therapy

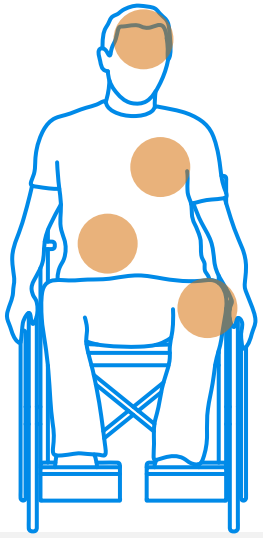
DMD, Duchenne muscular dystrophy.

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



ABD, actin-binding domain; CR, cysteine-rich domain; CT, C-terminal domain; DMD, Duchenne muscular dystrophy.
Phelps et al. *Hum Mol Genet.* 1995 Aug;4(8):1251-8.

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



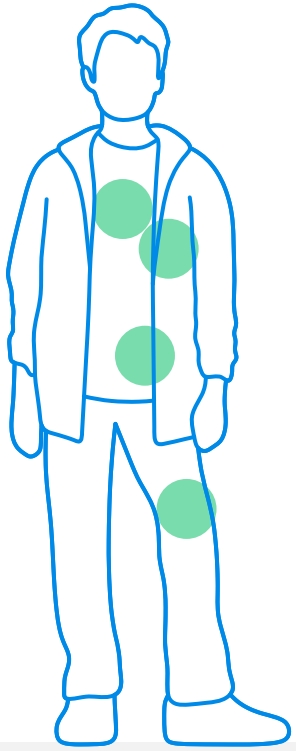
Lifespan:

- 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:

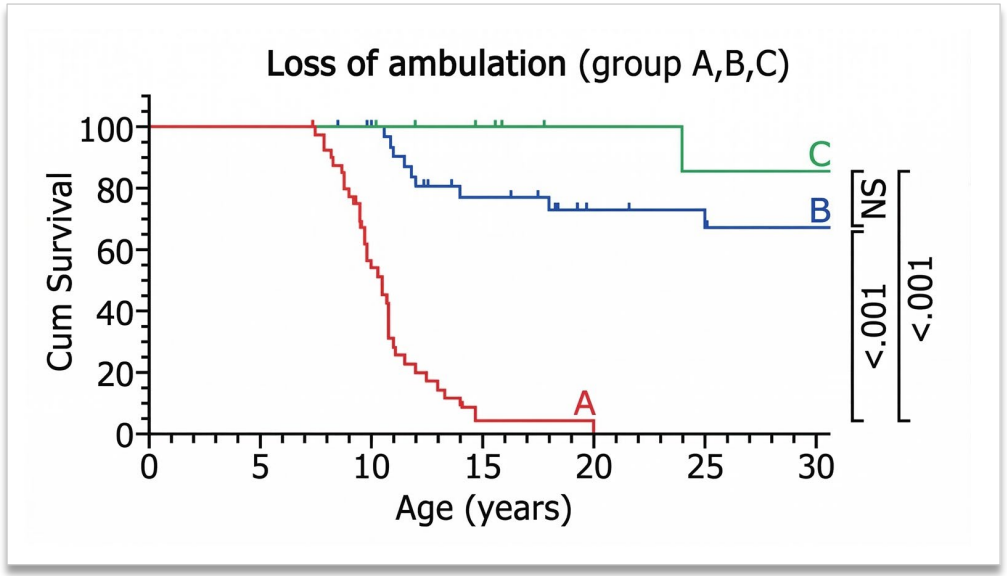
- Asymptomatic or mild symptoms[1-4]
- Normal muscle strength and ambulation throughout life[1,2]
- Normal respiratory function[2]
- Myocardial involvement, often manageable with medication[2]

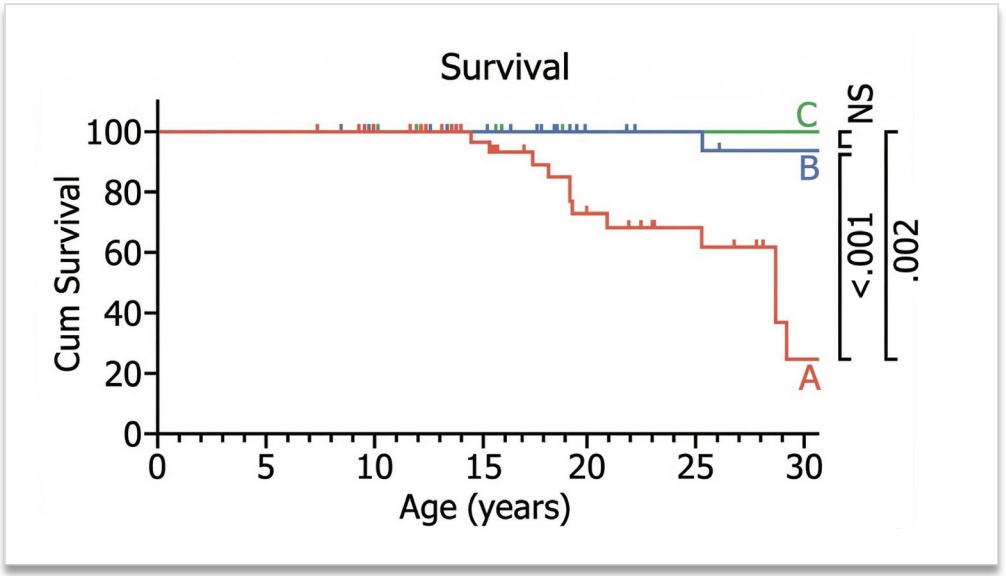
Del45-55 in-frame Dystrophin gene (BMD)

BMD, Becker Muscular Dystrophy; DMD, Duchenne Muscular Dystrophy.

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.

~5% Dystrophin is Associated with Meaningful Clinical Benefit





No dystrophin (0%)
Group A (*n*=42)

Residual dystrophin (0-5%)
Group B (*n*=34)

Residual dystrophin (≥5%)
Group C (*n*=14)

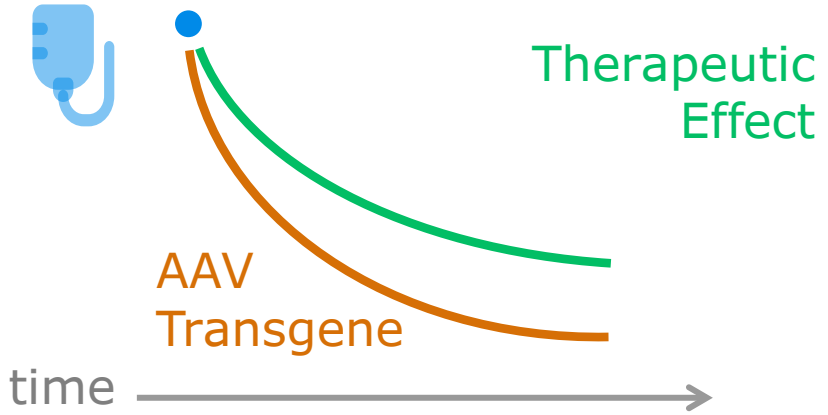
› ~5% dystrophin associated with prolonged ambulation beyond typical loss of ambulation time (~10–12 years)and linked to improved survival and milder diseases

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

Feraudy et al. Ann Neurol, 2021 Feb;89(2):280-292.

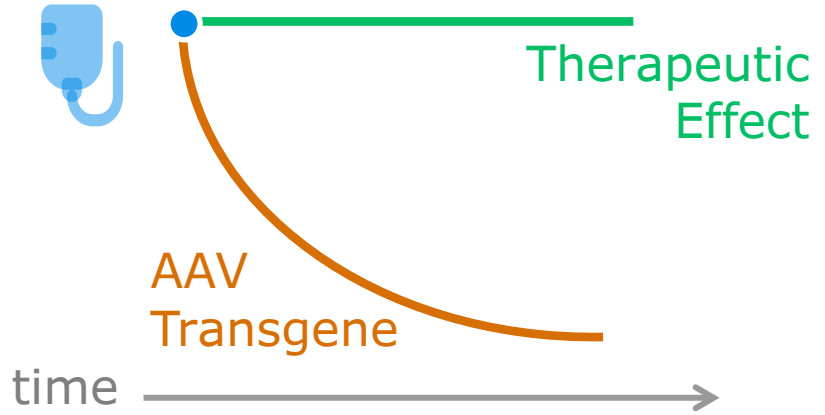
PBGENE-DMD is Designed for Durable Improvements in Muscle Function Independent of the Persistence of AAV Transgene

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



Illustrative Example

PBGENE-DMD:
The dystrophin protein is expressed by the human genome, AAV persistence not required for potential long-term effect



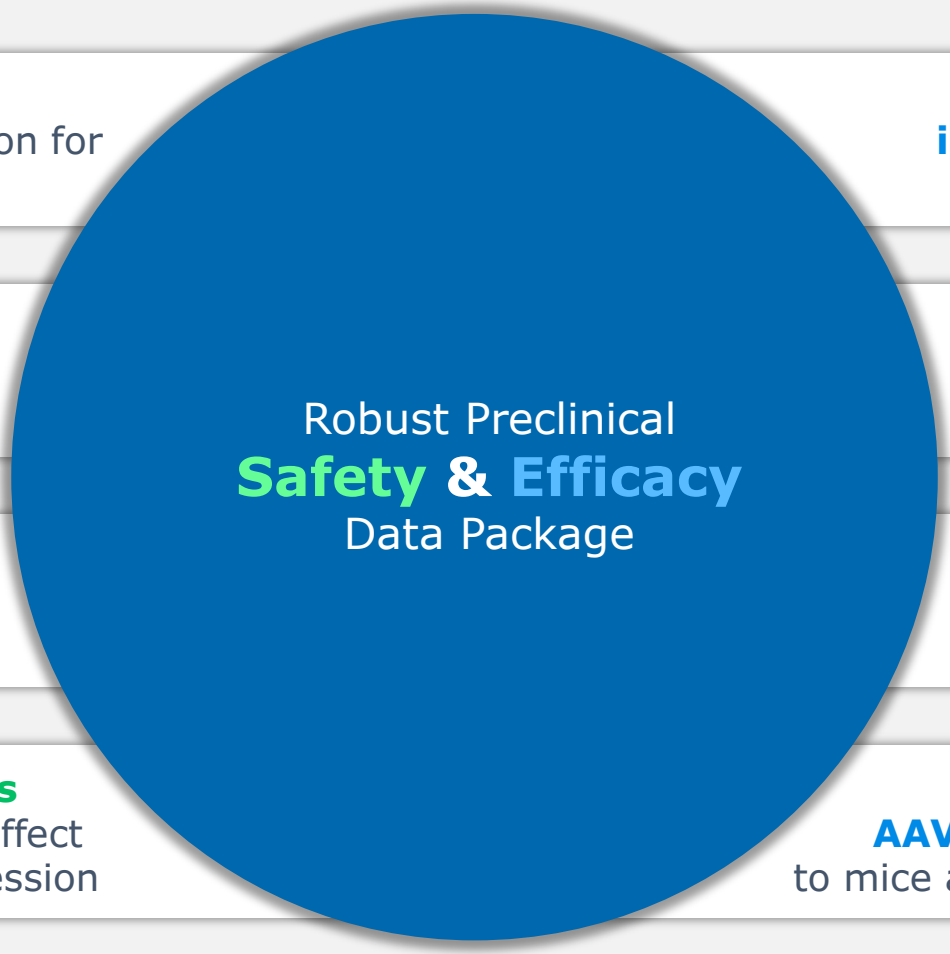
Illustrative Example

In contrast to conventional gene therapies, PBGENE-DMD is designed to deliver a durable therapeutic effect by leveraging permanent gene editing at lower doses

AAV, adeno-associated virus; DMD, Duchenne muscular dystrophy.

PBGENE-DMD Reason-to-Believe: Robust Preclinical Data

Comprehensive Preclinical Data to Support PBGENE-DMD Safety and Efficacy



ARCUS overhang cut promotes high frequency and fidelity excision for **predictable repair outcomes**

Durable muscle function improvements in DMD mice out to 9 months post dosing

Well-tolerated for ≥ 9 months in both DMD diseased and healthy mice

Increased dystrophin protein and up to 85% dystrophin-positive myofibers over time in DMD mice

Signs of **improved muscle pathology** in DMD disease mouse model after treatment

Satellite muscle cell editing consistently observed in skeletal muscle of DMD mice

Highly specific nucleases with no off-target editing effect on endogenous gene expression

Equivalent or better levels of AAV transduction in NHPs compared to mice across skeletal and cardiac muscles

AAV, adeno-associated virus; DMD, Duchenne muscular dystrophy; NHP, non-human primate.

PBGENE-DMD Drives Durable Dystrophin Protein Expression, Improvements in Muscle Integrity, and Significant Increases in Muscle Function in DMD Mice

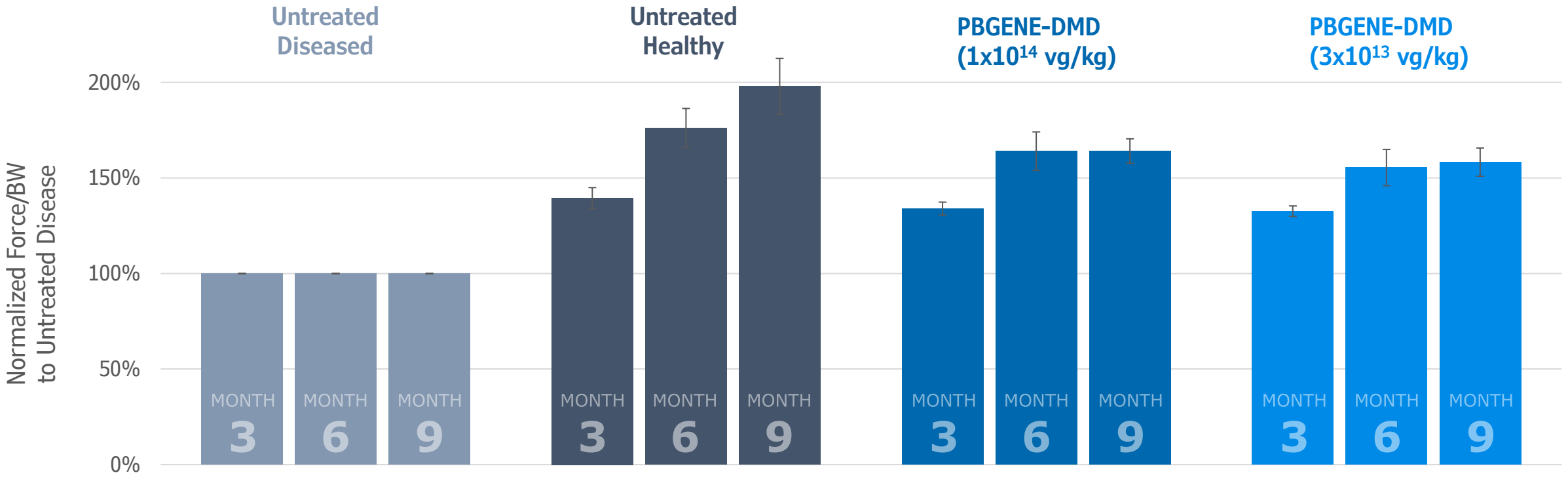
Dystrophin Protein Restoration	Muscle Integrity Biomarkers	Muscle Function Improvements
Re-establishes structural stability of muscle fibers	Indicates healthier muscle tissue following treatment	Translates into increased muscle force output

Restoring **near full-length dystrophin** protein addressed the underlying root cause of muscle degeneration in DMD and **restored muscle function**

Improved biomarkers includes creatine kinase, a key measurement of muscle health.
DMD, Duchenne muscular dystrophy.
1. Represents % dystrophin protein of healthy mice 2. Average Force Output is normalized by body weight and compared to untreated diseased animals.

PBGENE-DMD Reason-to-Believe: Bridging to Investigation in Humans

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.
AAV, adeno-associated virus; BW, body weight; DMD, Duchenne muscular dystrophy.

PBGENE-DMD Demonstrated As Good or Better Transduction in Nonhuman Primates Compared To Mice Suggesting Good Translatability

Strong Rationale for Go-Forward Dose Advancing in Humans

AAV, adeno-associated virus; DMD, Duchenne muscular dystrophy; NHP, non-human primate.

Nonclinical Data Supports the Potential for Safety and Efficacy of the Clinical Dose

SAFETY
Evaluated in DMD mouse model and NHPs

- No adverse safety findings observed at the completion of two GLP studies conducted in DMD mouse model and healthy nonhuman primates
- No off-target editing above background levels were observed at human conserved potential off-target sites in the NHP study[1]

EFFICACY
Evaluated in humanized DMD mouse model

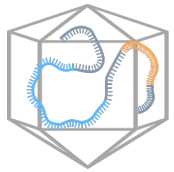
- Similar levels of dystrophin protein restoration and functional improvements observed in skeletal muscle of diseased DMD mouse model observed at both 3 x 10^{13} and 1 x 10^{14} vg/kg
- Similar levels of dystrophin protein and percent positive myofibers in the heart at both 3 x 10^{13} and 1 x 10^{14} vg/kg
- Increases in dystrophin protein restoration and percent positive myofibers in the diaphragm at 1 x 10^{14} vg/kg

Together these data support potential safety and efficacy of 1 x 10^{14} vg/kg PBGENE-DMD

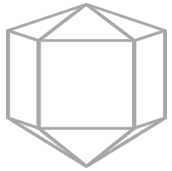
DMD, Duchenne muscular dystrophy; GLP, good laboratory practice; NHP, non-human primate.
1. Background levels of off-target editing set at 0.2% indels.

PBGENE-DMD Reason-to-Believe: High Quality AAV to Support Clinical Study

Manufacturing Process Produced High Quality AAV With >90% Full Capsids



Full Capsids

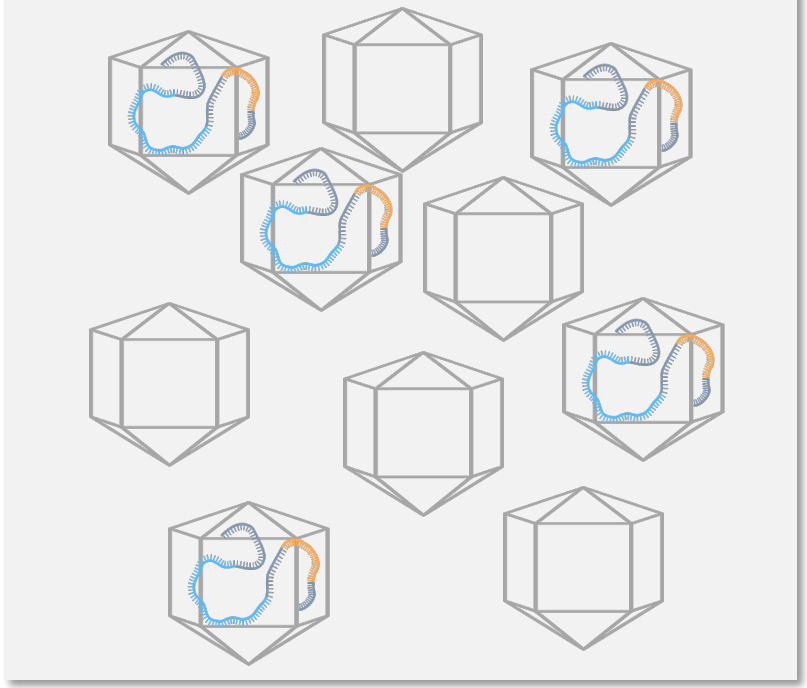


Empty Capsids

Both full and empty capsids are present in batches of AAV. The ratio of the two is an important quality attribute for safety and efficacy of AAV products.

90% Full Capsid AAV Batch

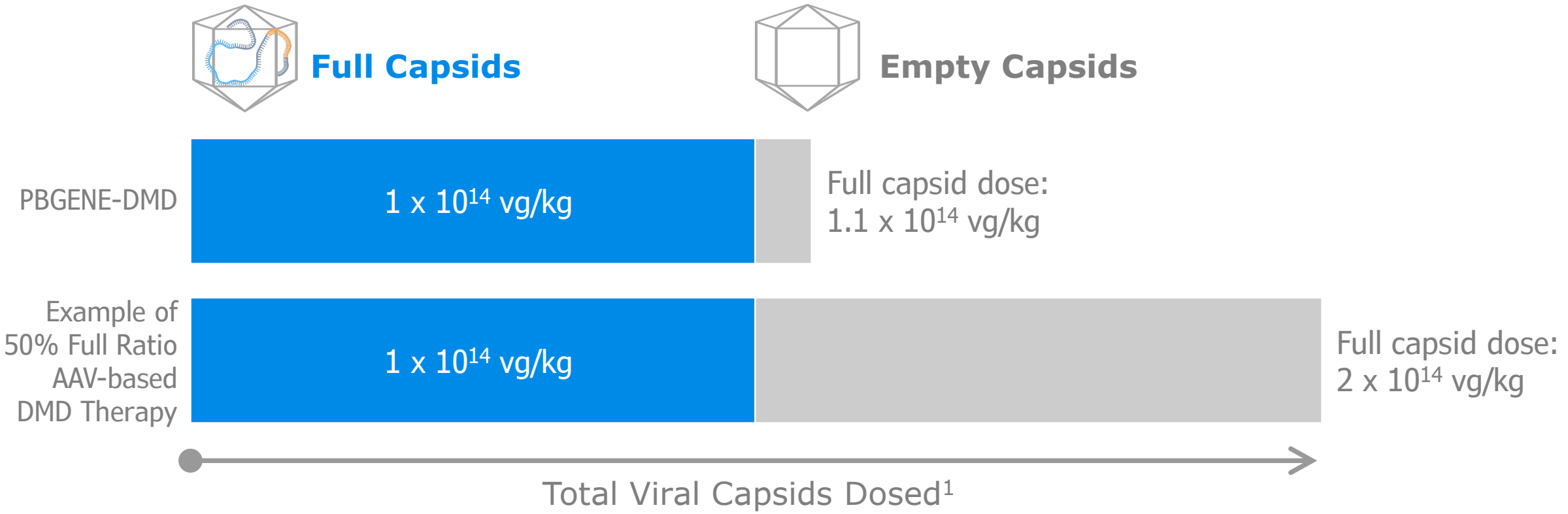
50% Full Capsid AAV Batch



PBGENE-DMD AAV clinical trial material to date have >90% full capsids

Why does this Matter?

PBGENE-DMD Aims to Minimizes Empty Capsids, Reducing Total Capsids Dosed and Potentially Improving Safety



PBGENE-DMD's full-capsid ratio enables delivery of target dose while delivering less total AAV capsids

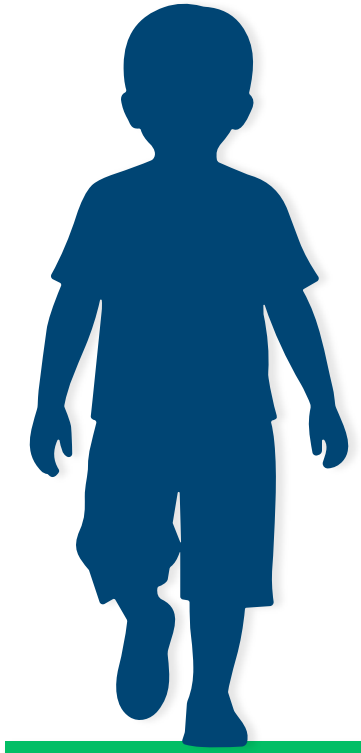
1.Byrne et al. Molecular Therapy Vol. 33 No 9 September 2025

FUNCTION-DMD Study

Aravindhan Veerapandiyan, M.D.
Director, Comprehensive Neuromuscular Program, Arkansas Children's Hospital, University of Arkansas for Medical Sciences

What to Expect in DMD patients?

Underlying disease biology drives several expected baseline findings in young DMD patients



- **Falls and gait abnormalities are common disease manifestations**
  - Frequent falls, toe walking, and mobility challenges occur as part of the natural history of Duchenne
- **Developmental and behavioral comorbidities occur in a subset of patients**
  - Speech delay, learning differences, and attention-related challenges are commonly reported in Duchenne populations
- **Elevated muscle enzymes are characteristic of the disease**
  - CK markedly elevated and AST/ALT frequently above ULN due to ongoing muscle breakdown rather than hepatic dysfunction
- **Background steroid therapy is standard of care**
  - Many patients receive prednisone or deflazacort, which may contribute to metabolic or behavioral findings independent of investigational therapy

These findings are consistent with the known baseline biology of early-stage Duchenne and may occur independent of treatment

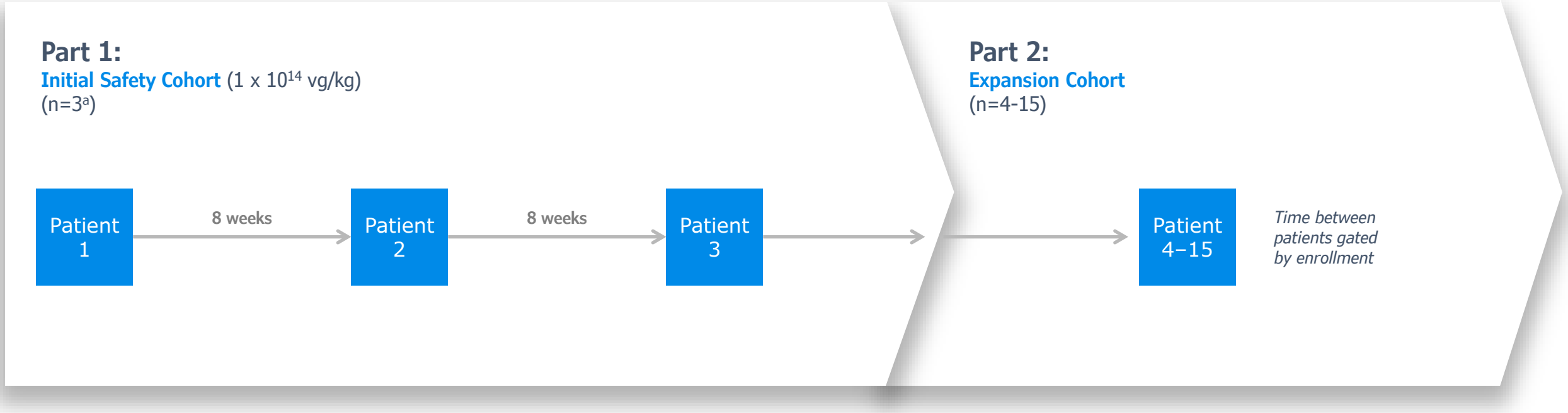
ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatine kinase; DMD, Duchenne muscular dystrophy; ULN, upper limit of normal.

Phase I/II Study Designed for Safety and Speed

Clinical Trial Identifier: NCT07429240

Inclusion criteria

- Male age 2-7
- DMD with mutations in exons 45-55
- Stable IMR regimen ≥12 weeks
- Up-to-date vaccines
- Able to complete age-appropriate motor testing assessments



[a]Up to three additional participants may be enrolled in Part 1, if needed, to further assess dosing or safety based on DSMB recommendation.

1H, first half; AE, adverse events; AAV, adeno-associated virus; BLA, biologics license application; DMD, Duchenne Muscular Dystrophy; DSMB, data safety monitoring board; IV, intravenous; vg/kg, vector genomes per kilogram.

Precision BioSciences. FUNCTION-DMD: A Phase 1/2, Open-Label Study of PBGENE-DMD Gene Editing Therapy in Duchenne Muscular Dystrophy (NCT07429240). ClinicalTrials.gov. Updated February 24, 2026. https://clinicaltrials.gov/study/NCT07429240. Accessed February 25, 2026.

PBGENE-DMD to be investigated at lower or equal doses to all other AAV DMD programs approved or in development

Company	Therapy	Vector	Dose level(s) tested
PRECISION BIOSCIENCES	**PBGENE-DMD[1]**	**AAV9**	**1 × 10^{14} vg/kg**
SOLID BIOSCIENCES	**SGT-001[2]***	AAV9	**Up to 2 × 10^{14} vg/kg**
	SGT-003[3]	AAV9-derived	**1 × 10^{14} vg/kg**
Pfizer	**fordadistrogene movaparvovec (PF-06939926)[4]***	AAV9	**Up to 3 × 10^{14} vg/kg**
SAREPTA THERAPEUTICS	**delandistrogene moxeparvovec (Elevidys)[5]**	rAAVrh74	**Up to ~1.33 × 10^{14} vg/kg**
REGENXBIO	**RGX-202[6]**	AAV8	**Up to 2 × 10^{14} GC/kg**

*Discontinued.
AAV, adeno-associated virus.
1. Precision BioSciences. PBGENE-DMD. NCT07429240. 2. Solid Biosciences. SGT-001. NCT03368742. 3. Solid Biosciences. SGT-003. NCT06138639.
4. Pfizer. Fordadistrogene movaparvovec (PF-06939926). NCT04281485. 5. Sarepta Therapeutics. delandistrogene moxeparvovec. NCT03375164. 6. RegenXBio. RGX-202. NCT05693142.

Phase I/II Study Endpoints

Clinical Trial Identifier: NCT07429240

Primary Endpoints

- Incidence, severity, and causality of TRAEs and SAEs from dosing through Week 104
- AEs and SAEs that occur or worsen after initiation of the investigational treatment

Secondary Endpoints

Biologic Activity:

Measurement of biologic activity of PBGENE-DMD through dystrophin expression in skeletal muscle biopsies at Week 12 and 52

Goal

Demonstrates target engagement and biological proof-of-concept

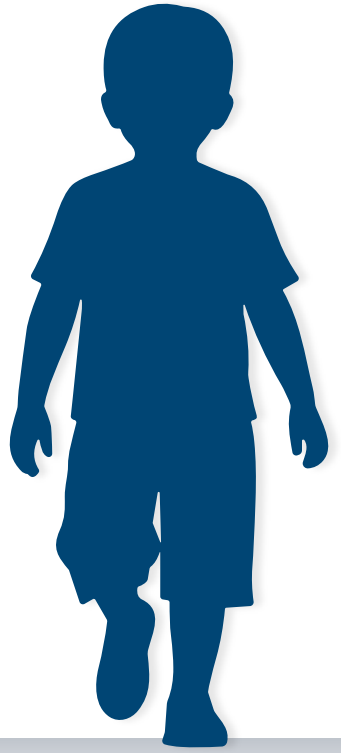
Exploratory Endpoints

Functional & Developmental Outcomes

- DMO: Stride Velocity 95th Centile
- Bayley Scales of Infant and Toddler Development, Gross Motor and Fine Motor scores (<3 years of age)
- NSAA total score (≥3 years of age)
- Timed performance tests (≥3 years of age)

*Bayley Scales of Infant and Toddler Development are 4th Edition, Bayley-4.
AE, adverse event; DMO, Digital mobility outcome; NSAA, North Star Ambulatory Assessment; SAE, serious adverse event; TRAE, treatment-related adverse events.
Precision BioSciences. FUNCTION-DMD: A Phase 1/2, Open-Label Study of PBGENE-DMD Gene Editing Therapy in Duchenne Muscular Dystrophy (NCT07429240). ClinicalTrials.gov. Updated February 24, 2026. https://clinicaltrials.gov/study/NCT07429240. Accessed February 25, 2026.

Key Inclusion Criteria



Category	Inclusion Criteria
Age / Sex	Males, 2–7 years of age (inclusive) at time of consent
Diagnosis	Genetically confirmed DMD with mutations in exons 45–55
Motor Ability	Able to complete age-appropriate motor testing assessments • Ages 2–<4: walk ≥10 meters • Ages 4–7: Must walk ≥100 meters • NSAA score: 16–29
Corticosteroid Use	Patients on corticosteroids may be included if they have been on a stable daily regimen for ≥12 weeks (temporary switch allowed before dosing)
Long-Term Follow-Up	Willing to participate in a Long-Term Follow-Up (LTFU) study after completion
Prior Therapies	Patients are eligible to enroll following a ≥6-month washout from non-AAV delivered therapies including exon-skipping treatments

DMD, Duchenne muscular dystrophy; NSAA, North Star Ambulatory Assessment.

Safety as Top Priority:

Immune Modulation Regimen (IMR) strategy ensures monitoring and interventions are optimized to patient safety needs during two key safety risk periods

Safety Mitigation: Preventing early and late serious immune complications.

AAV therapies can trigger **two predictable immune-related safety risk periods.** To keep patients safe, we plan to use a planned, phased immune-modulation:

Early Safety Period	Later Safety Period
Safety Risks: Thrombocytopenia & TMA, Cardiac Toxicity, Hyperinflammatory Syndromes, Gastrointestinal Toxicity, Flu-Like Symptoms	**Safety Risk**: Hepatotoxicity

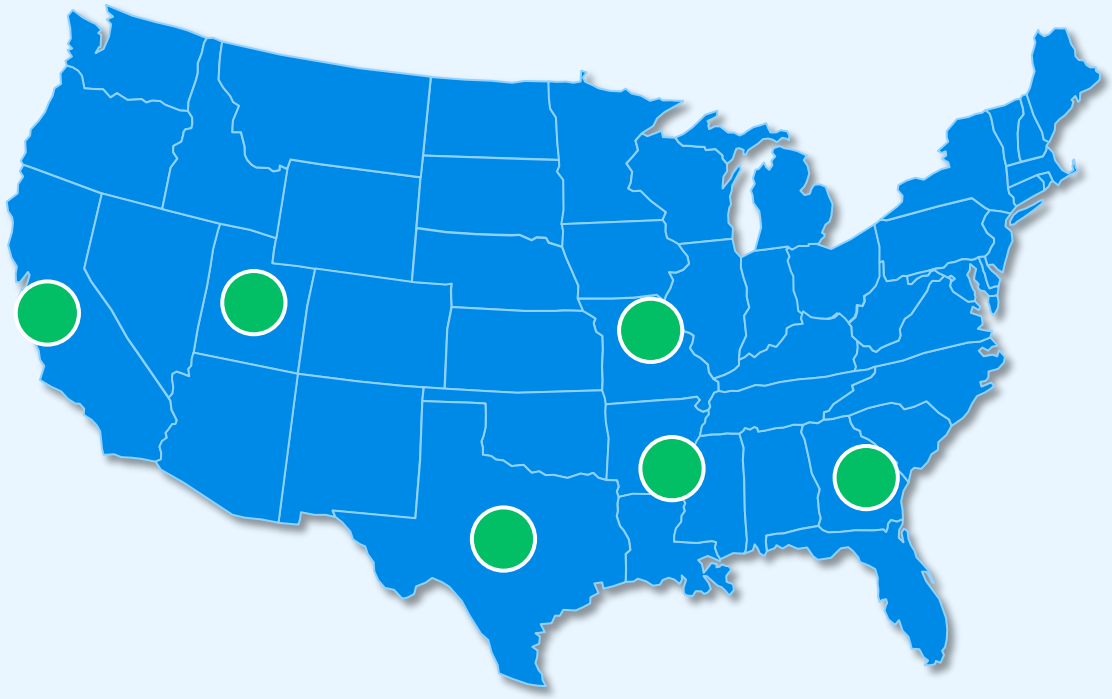
Eculizumab
Prevents immune-mediated tissue damage by blocking the complement pathway

Steroids
Rapidly controls systemic inflammation during acute immune activation

Sirolimus
Provides sustained immune regulation by limiting T-cell–driven responses

Early immune and complement activation can cause blood count changes, cardiac strain, inflammation, and flu-like symptoms
AAV, adeno-associated virus; IMR, immune modulation regimen; TMA, thrombotic microangiopathy.

World-Class Clinical Network with Proven Duchenne Expertise



- **Deep AAV gene therapy expertise** across Phase 1–3, including immune risk management and pediatric neuromuscular dosing
- **Elite Duchenne trial network** anchored by PPMD-Certified Care Centers and MDA Care Clinics with demonstrated, reliable execution
- **Decades-long Duchenne leadership** spanning pulmonary, cardiac, and neuromuscular care
- **Top-tier scientific output** with consistent publication in leading neuromuscular journals
- **Best-in-class patient** access through trusted partnerships with top patient advocacy groups

AAV, adeno-associated virus; DMD, Duchenne muscular dystrophy; MDA, Muscular Dystrophy Association; PPMD, Parent Project Muscular Dystrophy.

Closing Remarks & Program Next Steps

Alex Kelly
Chief Financial Officer
Precision BioSciences

Upcoming 2026 Milestones and Outlook

PBGENE-HBV

- Exploring multiple levers (dose level, number of dose administration & time between dose administrations) to optimize therapeutic index
  - Continuing Cohorts 3, 4 & 5 in parallel
- Additional data expected to be shared at hepatitis-focused medical conferences throughout 2026
- Anticipate initiating Part 2 of ELIMINATE-B after choosing optimal dosing regimen to stop NUCs

PBGENE-DMD

- US IND clearance by FDA in early Q1 2026
  - IRB process and site activation underway following IND clearance
- Initial data from multiple patients anticipated by year end 2026
  - Target enrollment 3-5 patients
  - Safety Data
  - Early efficacy assessed by percentage of near full-length dystrophin protein expression from muscle biopsies at 12 weeks

Cash runway through multiple catalysts:

Strong balance sheet with ~$137 million in cash (YE 2025) with expected cash runway through 2028[1]

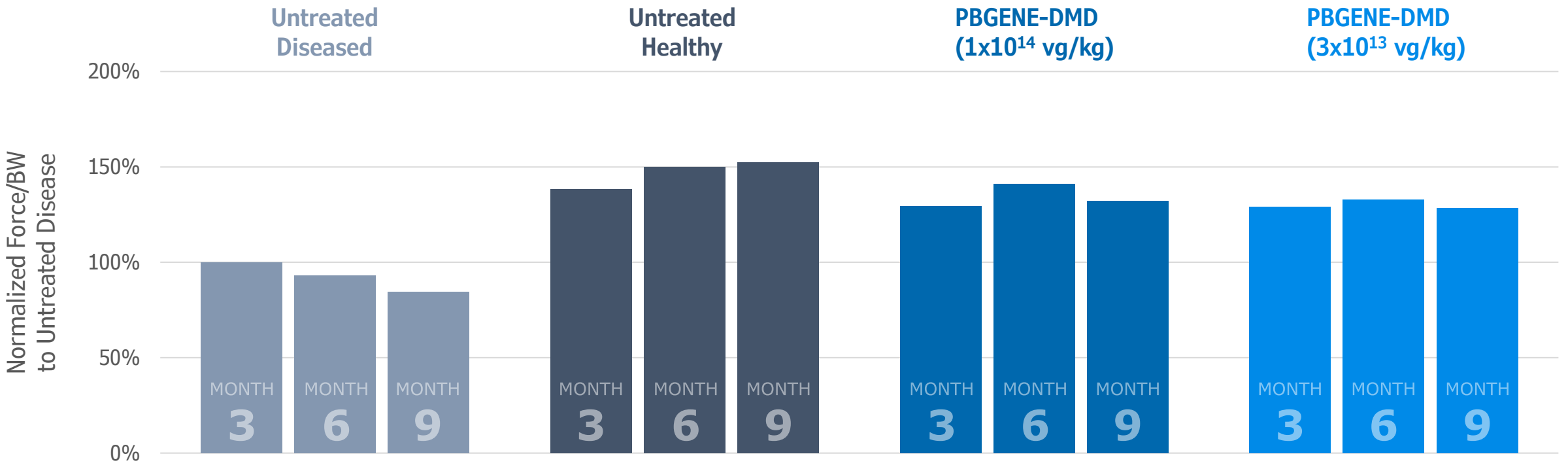
DMD, Duchenne muscular dystrophy; FDA, U.S. Food and Drug Administration; HBV, hepatitis B virus; IND, Investigational New Drug; IRB, Institutional Review Board; NUCs, nucleos(t)ide analogs; YE, year-end.

[1]$137M cash, cash equivalents, and restricted cash as of 12/31/25. The Company expects existing cash and cash equivalents, inclusive of expected azer-cel milestone proceeds, continued fiscal and operating discipline, and availability of its ATM facility are expected to provide sufficient cash runway through 2028.

Q&A

Appendix Slides

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

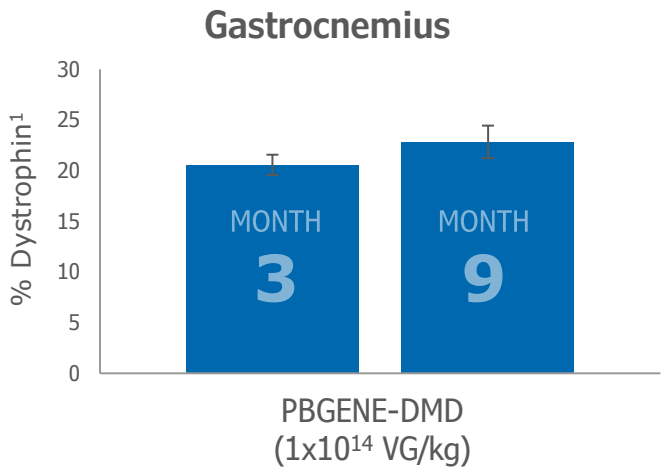


- Improved muscle function observed from 3 to 6 months.
- Durable functional improvements maintained out to 9 months.
- Benefit consistent across both experimental dose levels

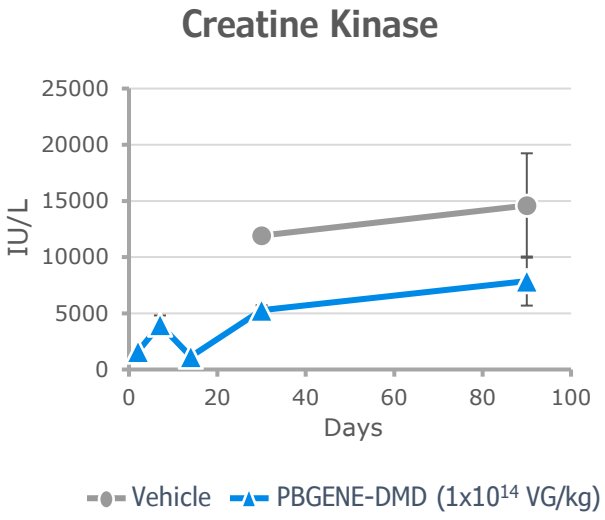
Force was measured in the calf across multiple stimulation frequencies. Averaged force normalized to bodyweight is shown. Statistically significant ($p<0.001$) increases in force were observed in both doses of PBGENE-DMD compared untreated diseased animals at both time points.
AAV, adeno-associated virus; BW, body weight; DMD, Duchenne muscular dystrophy.

PBGENE-DMD Drives Durable Dystrophin Protein Expression, Improvements in Muscle Integrity, and Significant Increases in Muscle Function in DMD Mice

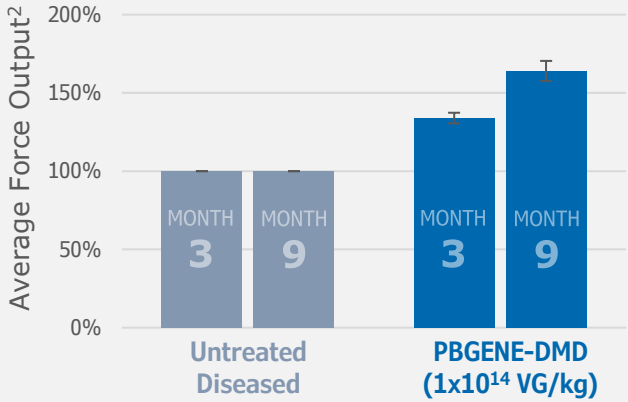
Dystrophin Protein Restoration



Muscle Integrity Biomarkers



Muscle Function Improvements



Restoring dystrophic protein, leads to improved CK, and improved muscle function.

DMD, Duchenne muscular dystrophy.
1. Represents % dystrophin protein of healthy mice 2. Average Force Output is normalized by body weight and compared to untreated diseased animals.