PRECISION
BIOSCIENCES

Corporate Presentation

August 2026

Forward-Looking Statements

This press release contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. All statements contained in this press release 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 FDA regulatory pathway, operational initiatives, strategies, further development, or timing of additional updates or data releases of PBGENE-HBV and PBGENE-DMD, timing and progress of site activation, patient recruitment, and enrollment for the FUNCTION-DMD and ELIMINATE-B clinical trials; expected timing of program or clinical updates or progress for the FUNCTION-DMD and ELIMINATE-B clinical trials by the end of 2026, including an expectation of initial safety data in the FUNCTION-DMD study; administrations of PBGENE-HBV across cohorts and the evaluation on the impact of escalating dose levels and dosing intervals; the goal of complete viral cure in PBGENE-HBV, the expectation of current and future ELIMINATE-B dataset to inform selection of the optimal dosing schedule for Part 2 expansion; the design of PBGENE-HBV to eliminate cccDNA and inactivate integrated HBV DNA with high specificity, potentially leading to cure; the suitability of PBGENE-HBV for the treatment of hepatitis B and the targeting of the root cause of the disease; the adequacy of mitigation measures in LNP-related hypotension observed during dose escalation; the design of PBGENE-DMD to durably improve function for approximately 60% of patients with DMD; adequacy of the DMD IMR strategy for patient safety; translation of results in preclinical studies of ARCUS nucleases 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 continued azer-cel development by licensees or partners; the sufficiency of our expected cash runway to extend through 2028 to enable PBGENE-HBV and PBGENE-DMD data milestones, and assumptions on market opportunity and size. 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,” "path", “plan,” “possible,” “potential,” “predict,” “project,” “pursue,” “should,” “strive,” “suggest,” “target,” "targeting", "toward", “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; 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 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; effects of any pandemic, epidemic, or outbreak of an infectious disease; 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; Annual Report on Form 10-K for the annual period ended December 31, 2025 and Quarterly Report on Form 10-Q for the quarterly period ended June 30, 2026, 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 BIOSCIENCES

Dedicated to developing novel in vivo gene editing therapeutics 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
- Ability to leverage platform into multiple high value therapeutic areas

Multiple Near-Term Data Catalysts Through Wholly Owned Programs

Clinical-Stage *In Vivo* Gene-Editing Programs

eliminate-B

PBGENE-HBV for Chronic Hepatitis B

- Viral gene elimination program with 38 doses delivered across 16 patients treated in 5 cohorts to date. Responses at all dose levels reported to date
- **First and only clinical-stage program designed to eliminate and inactivate root cause of viral replication—directly targeting cccDNA**
- **Next update targeting year end 2026**

Clinical data now validate direct targeting and elimination of cccDNA for the first time ever

functionDMD

PBGENE-DMD for Duchenne Muscular Dystrophy

- PBGENE-DMD is designed to provide durable functional muscle improvement targeting ~60% of patients with DMD
- First clinical trial sites activated at Arkansas Children's Hospital and Washington University – St. Louis
- **Next step: commence trial and execute in clinic, initial safety data targeted year end 2026**

First gene editing approach in the clinic offering potential for functional improvement in DMD

cccDNA, covalently closed circular DNA; DMD, Duchenne muscular dystrophy.

Chronic Hepatitis B Multi-Billion Dollar Market Opportunity:
Large patient population currently on non-curative treatment options

> 300 Million
cHBV infections globally

Up to 2,400,000
cHBV infections in the US

U.S. Market Opportunity of ~$10 Billion[1]

Driven by the drug treated patient population today with total global aggregate revenue estimates up to ~$500B[3]

~250,000
patients in US

~180,000
patients in Europe

~4,000,000
patients in China

~260,000
patients in Japan

Estimated ~5M patients in major markets infected with chronic HBV & treated with standard of care (SoC) nucleos(t)ide analog treatments[2,3]

M, million.
Additional 142,000 potentially drug treated in Africa.[3]

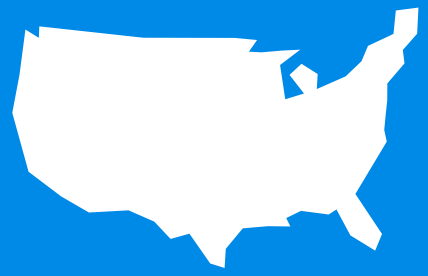
1. Chattopadhyay, Debjit, et al. "Precision BioSciences, Inc. (DTIL): The ARCUS Editing Platform — Initiating Coverage with a BUY Rating and $19 PT." Guggenheim Securities, LLC, 30 Apr. 2024.
2. Nguyen MH et al. Clin Microbiol Rev. 2020;33(2); GSK public epidemiology estimates for cHBV. **3.** Trucchio, Patrick. "Hepatitis B Breakthrough Boom: Navigating a $450-$500 Billion Frontier", H.C. Wainwright & Co. 14, Feb 2024 3. Sonderup MW, Spearman CW. HBV elimination in Africa-Current status and challenges. Clin Liver Dis (Hoboken). 2024;23(1):e0166. Published 2024 May 3.

Global DMD Market Poised for Innovative Breakthroughs Like PBGENE-DMD

Prevalence & Incidence[1]

~300-400k
DMD Patients globally

~15k
DMD Patients in US

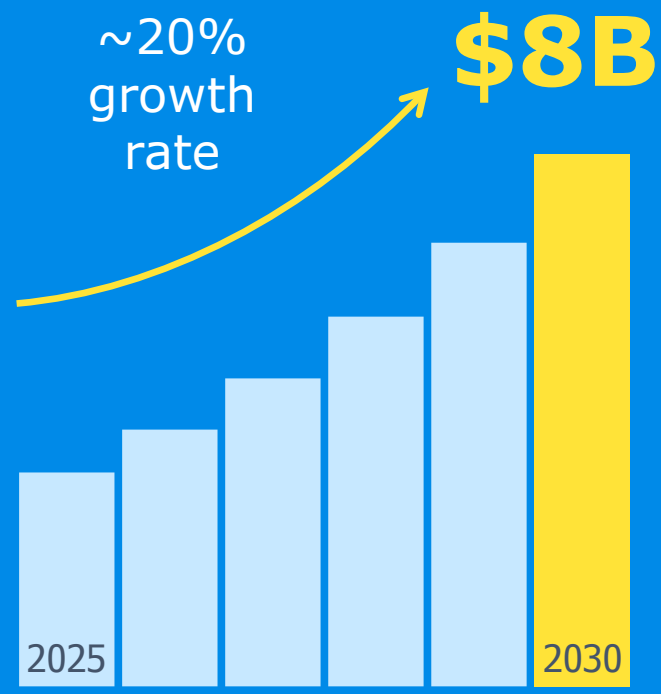


>20k births
per year globally

~550 births
per year in US

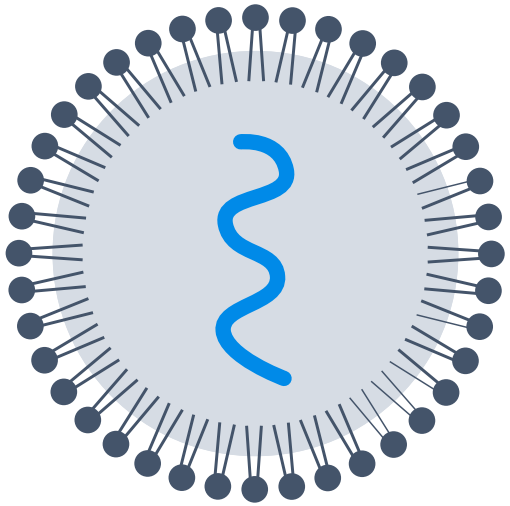
★ **Up to 60% of these patients are eligible for PBGENE-DMD**

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.

PBGENE-HBV
for Chronic Hepatitis B



HBV at the Turning Point:
From Lifelong Suppression Toward Finite, Biomarker-Guided ***Viral Cure***

"True cure of HBV infection requires eradication, degradation ...of cccDNA ..." [1]

— Dusheiko, NEJM 2023

"...assessing response to chronic hepatitis B therapies is unclear because of inconsistent correlations between HBsAg and clinical response." [2]

— FDA Guidance 2022

"First clinical evidence of reservoir-elimination emerges in HBV ... an important validation event for the ARCUS platform..."

— Trucchio, Sell-Side Analyst

1. Dusheiko G, et al. *N Engl J Med*. 2023;388(1):55–69. 2. FDA Guidance – Chronic Hepatitis B Virus Infection; Developing Drugs for Treatment; April 2022. Accessed April 30, 2026.

A Direct Antiviral Therapy Targeting & Eliminating cccDNA Is Needed to Cure HBV

Current Therapeutics Rarely Achieve Cure Because They Do Not Eliminate cccDNA

- Current therapies **do not eliminate the sole source** of infectious particles: **cccDNA**
- **Downstream targeting agents** only achieve single-digit **functional cure rates.** Do not drive **viral cures.**

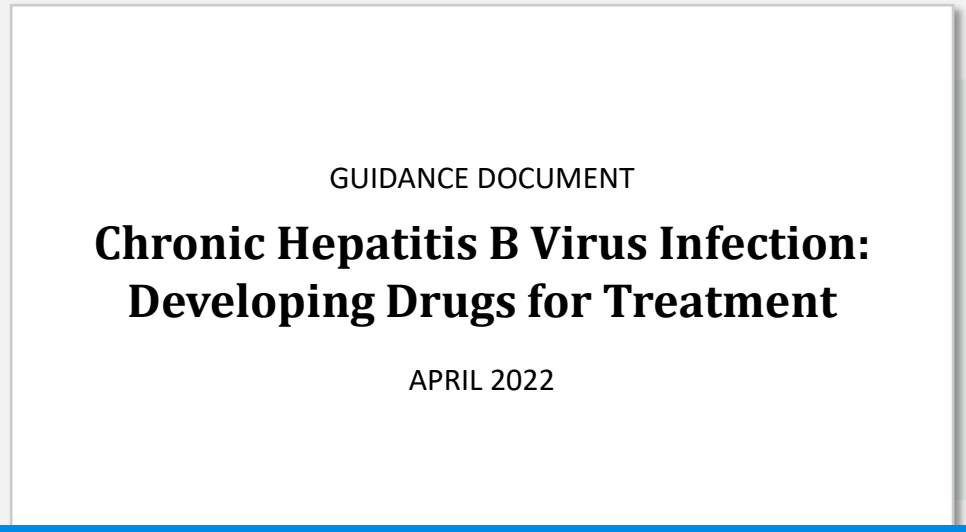
PBGENE-HBV Is the Only Program Designed to Permanently Eliminate cccDNA

- **cccDNA is the sole source of infectious viral DNA = HBV DNA**
- **FDA guidance is focused on destruction of HBV DNA** as the key element for approval

cccDNA, covalently closed circular DNA; FDA, US Food and Drug Administration; HBV, hepatitis B virus.

FDA Regulatory Pathway: Elimination of HBV DNA Is Required for Approval

PATHWAYS TO **FDA APPROVAL**[1]



1. HBV DNA undetectable only

OR

2. HBV DNA + HBsAg both undetectable

Loss of HBV DNA Is the Common Therapeutic Goal for FDA & Essential for Viral Cure

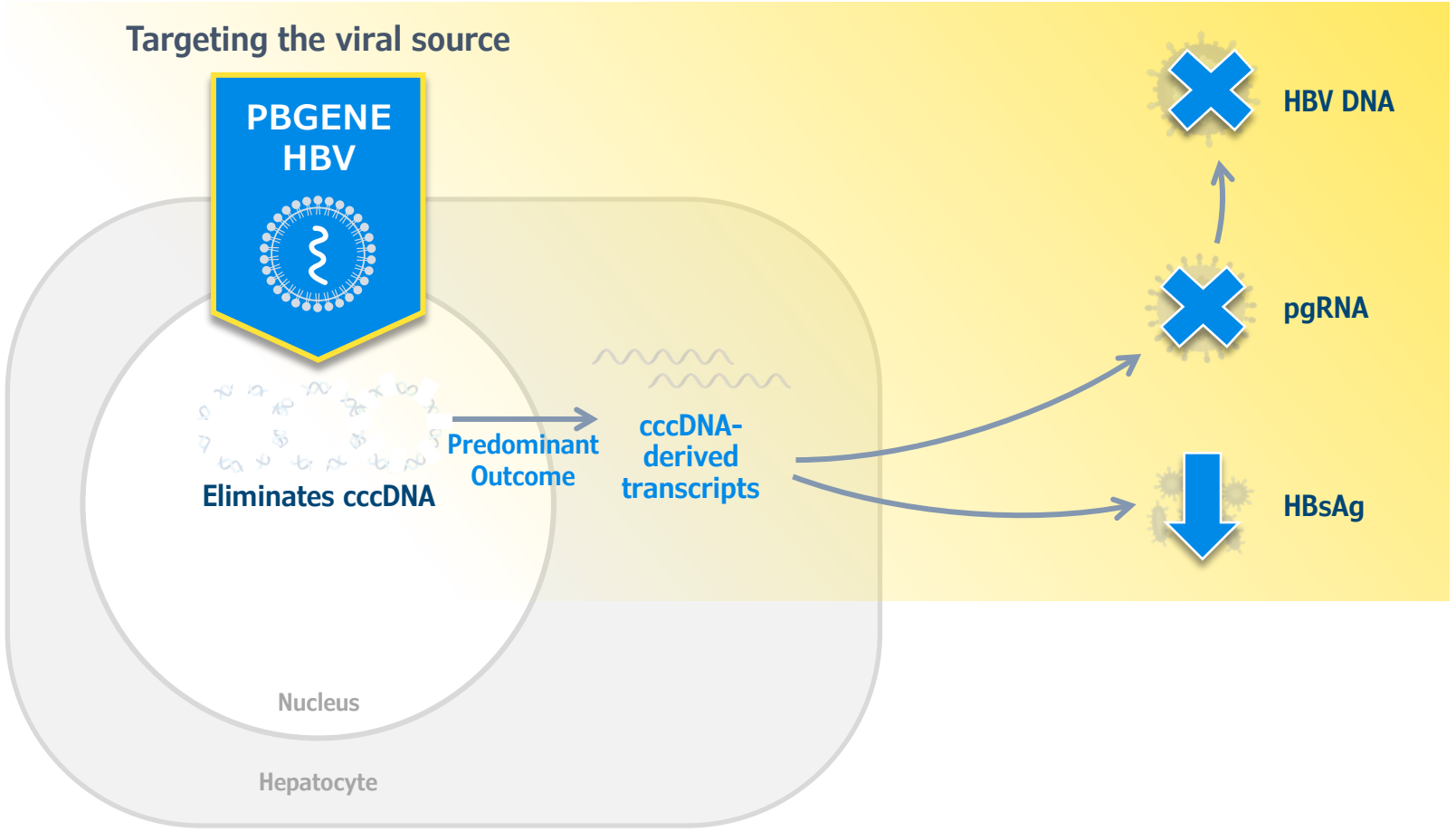
Utilizing HBsAg levels for "…assessing response to chronic hepatitis B therapies is unclear because of inconsistent correlations between HBsAg and clinical response."[1-4]

cccDNA, covalently closed circular DNA; FDA, US Food and Drug Administration; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus.

1. FDA Guidance – Chronic Hepatitis B Virus Infection; Developing Drugs for Treatment; April 2022. Accessed April 30, 2026. 2. Hu B, et al. *J Gastroenterol Hepatol.* 2018;33(7):1389-1396.
3. Thompson AJ, et al. *Hepatology*. 2010;51(6):1933-1944. 4. Chan HL, et al. *J Hepatol*. 2011;55(5):1121-1131.

Phase 1 ELIMINATE-B Trial
Efficacy Data

Direct Antiviral Targeting: Late-Breaking EASL Data Demonstrate PBGENE-HBV Eliminates cccDNA as Designed, Providing Path Toward Viral Cure

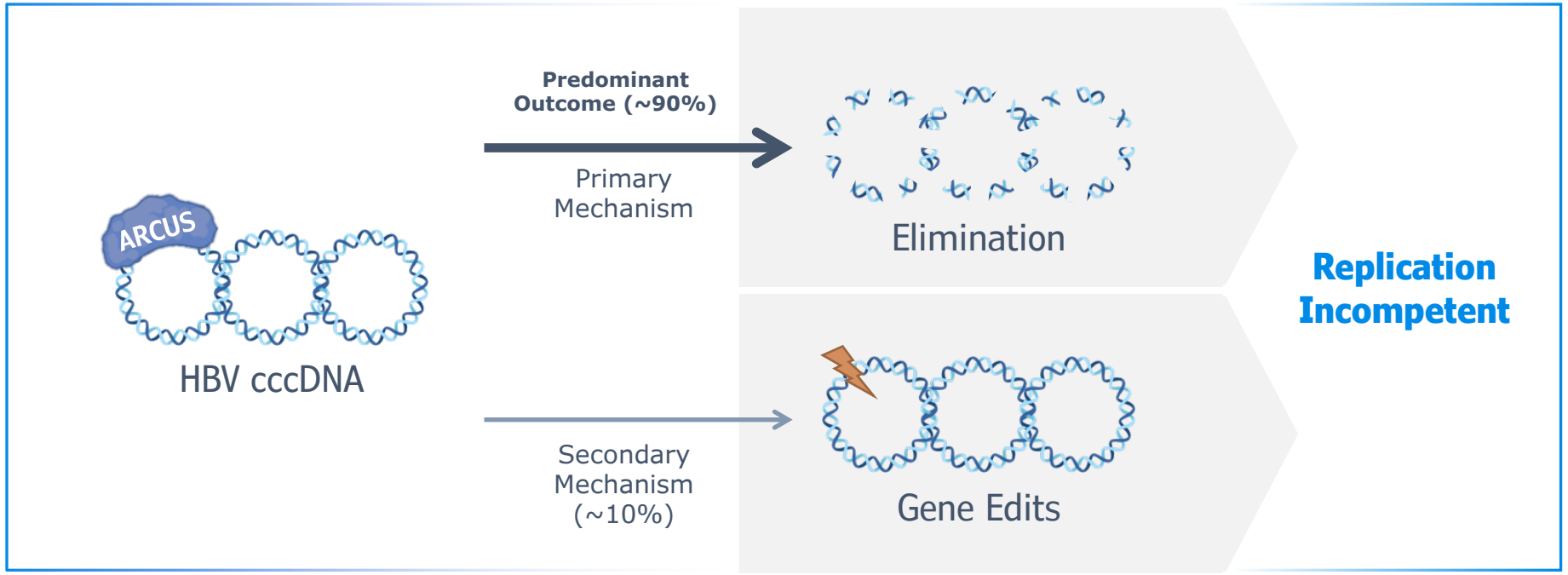


PBGENE-HBV potently eliminates cccDNA as the primary mechanism and drove **undetectable pgRNA** in all patients who were detectable at baseline

…with corresponding reductions in HBsAg

cccDNA, covalently closed circular DNA; HBsAg, hepatitis B surface antigen; pgRNA, pregenomic RNA.

Deep Dive Into PBGENE-HBV Mechanism: Potently Targeting cccDNA Through Two Productive Editing Outcomes



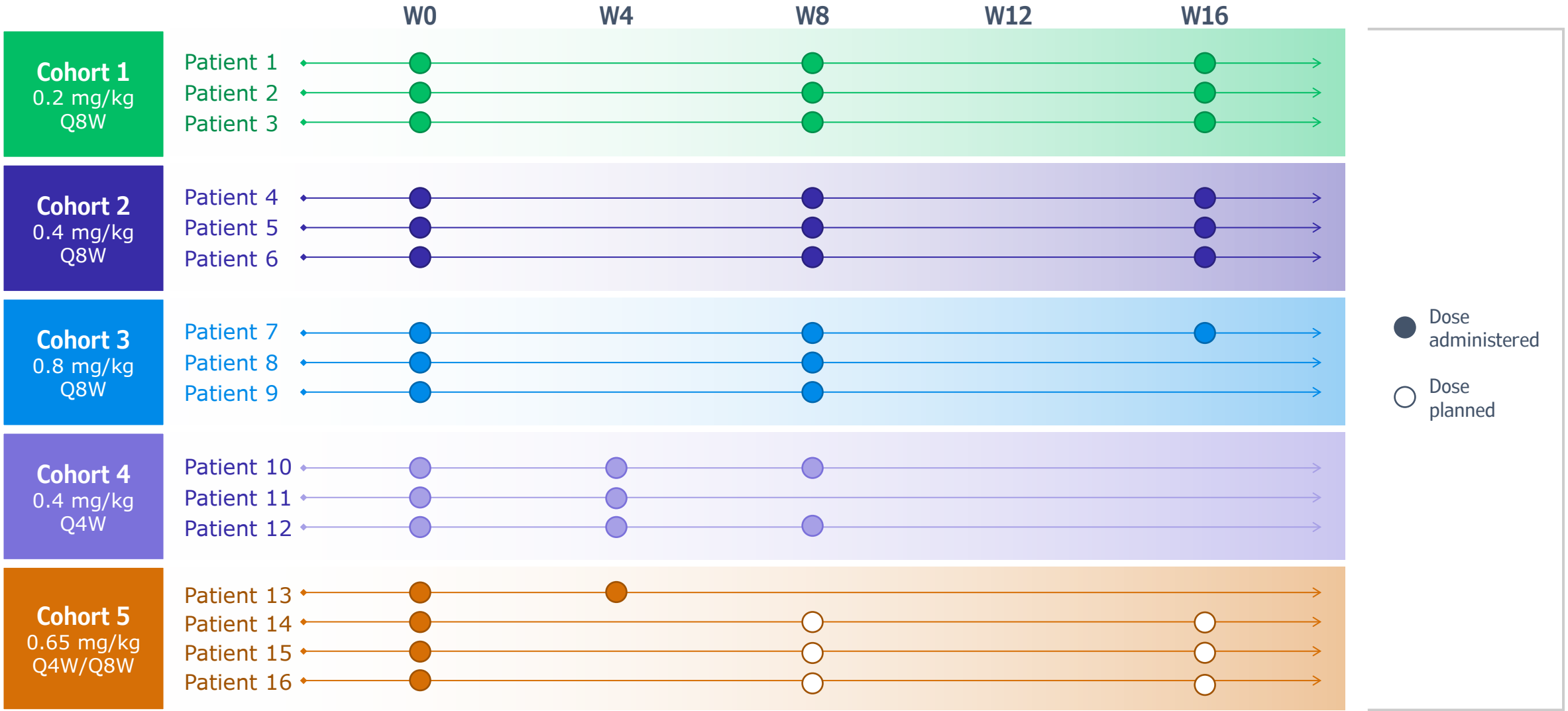
The Reason Why:

PBGENE-HBV targets cccDNA, which is more effective because there are two editing outcomes.

Both editing outcomes result in replication incompetent virus.

Based on preclinical data, elimination represents predominant editing outcome for cccDNA.
cccDNA, covalently closed circular DNA.

Broadening Patient Experience and Confidence: 38 Doses Across 16 Patients in 5 Cohorts to Date



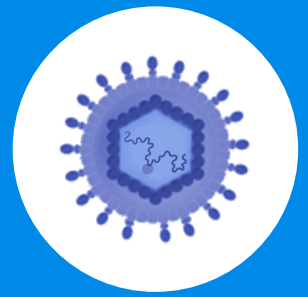
Patients without third circle are not currently planned for additional dose administrations.
Q4W, every 4 weeks; Q8W, every 8 weeks; W, week.
Data cutoff: May 4, 2026.

Multiple Datasets Support cccDNA Targeting and Permanence of Effect by PBGENE-HBV

Liver Biopsies

CONFIRMATION OF cccDNA ELIMINATION

- **10-fold (1-log) reduction in cccDNA-derived transcripts** and increases in editing with repeat administrations



pgRNA

POTENT cccDNA EFFECT

- **100% of patients (6/6) with detectable pgRNA before treatment are undetectable** after PBGENE-HBV treatment

HBsAg

SUBSTANTIAL ANTIVIRAL ACTIVITY IN ALL PATIENTS

- Durable HBsAg reductions across all patients treated; **highly supportive of cccDNA elimination** by PBGENE-HBV

cccDNA, covalently closed circular DNA; HBsAg, hepatitis B surface antigen; NA, nucleos(t)ide analog; pgRNA, pregenomic RNA.

Multiple Datasets Support cccDNA Targeting and Permanence of Effect by PBGENE-HBV

Liver Biopsies

CONFIRMATION OF cccDNA ELIMINATION

pgRNA

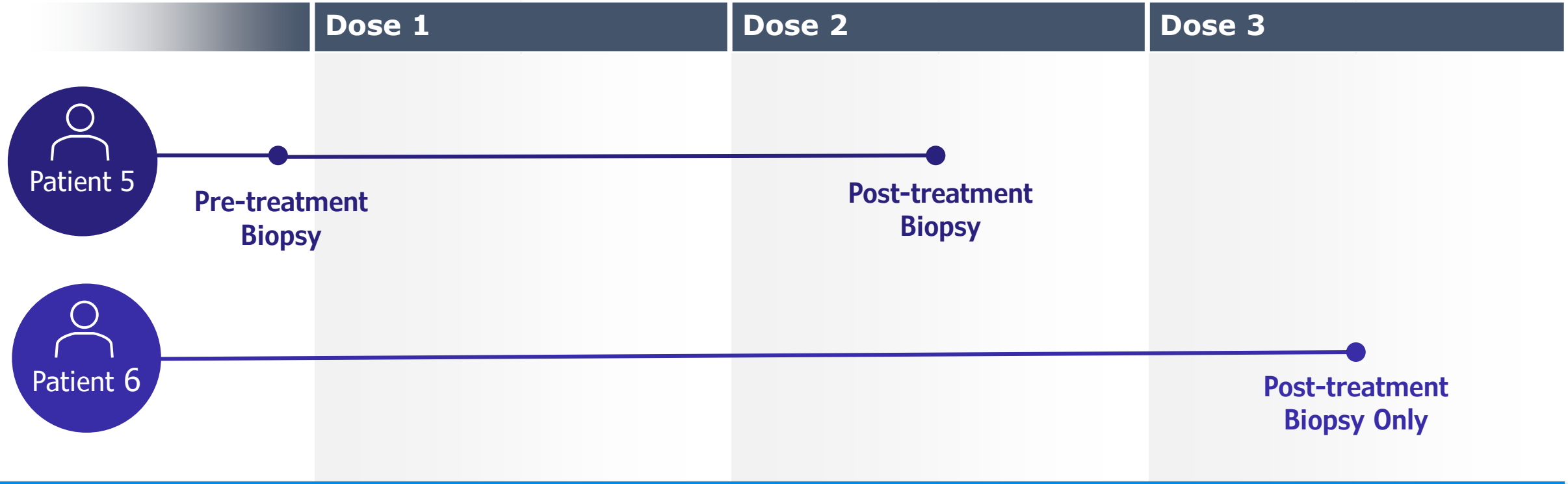
POTENT cccDNA EFFECT

HBsAg

SUBSTANTIAL ANTIVIRAL ACTIVITY IN ALL PATIENTS

cccDNA, covalently closed circular DNA; HBsAg, hepatitis B surface antigen; pgRNA, pregenomic RNA.

EASL DATA: Biopsies From Two Patients in Cohort 2 (0.4 mg/kg, Q8W)



Liver biopsy analyses

Transcript Sequencing Methodology

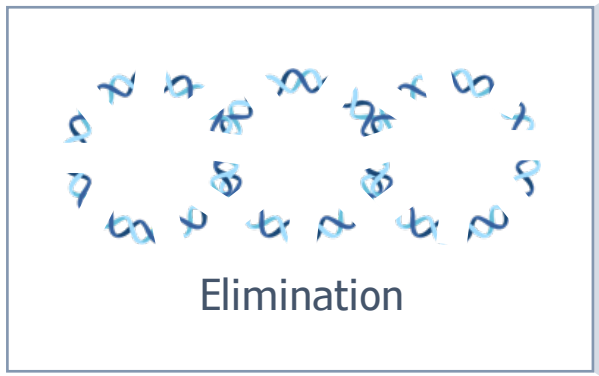
- Quantitate levels of cccDNA transcripts pre- and post-treatment
- Quantitate edits in remaining cccDNA transcripts comparing 2 and 3 dose administrations

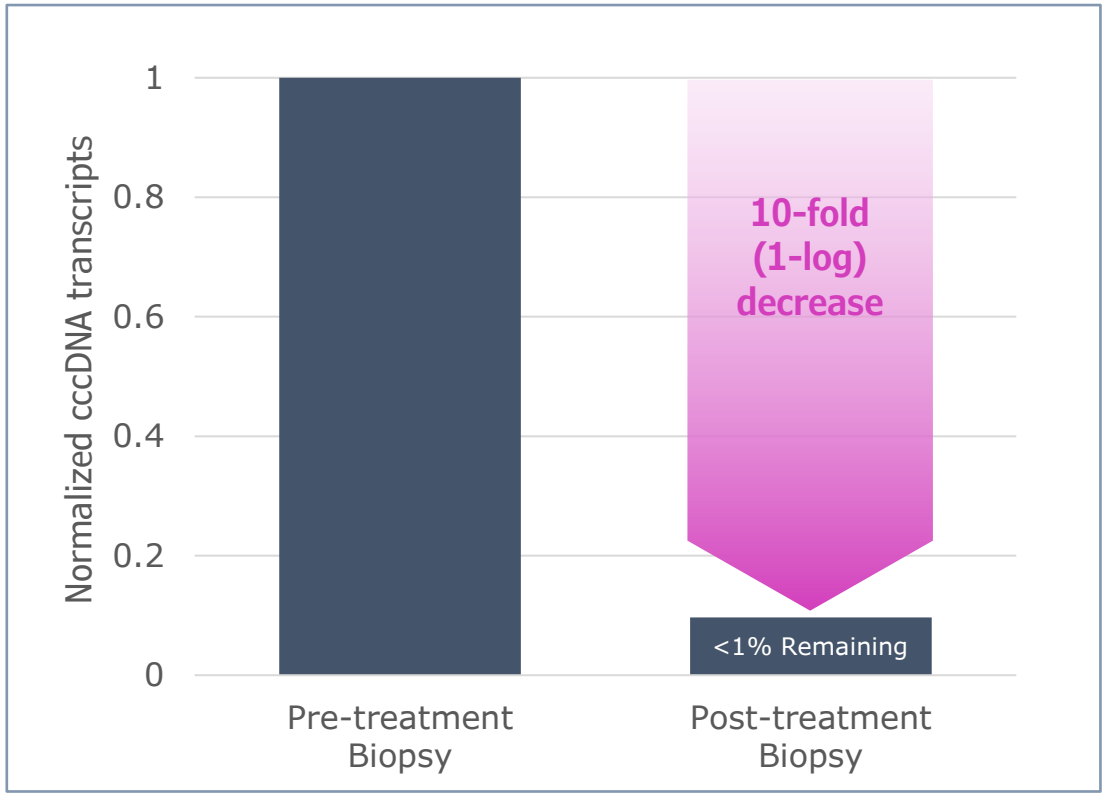
cccDNA, covalently closed circular DNA; Q8W, every 8 weeks.
Data cutoff: May 4, 2026.

EASL DATA: Primary Mechanism - PBGENE-HBV Elimination of cccDNA Resulted in 10-fold (1-log) Decrease in cccDNA

Patient 5
Cohort 2: 0.4 mg/kg

Biopsy after 2 administrations

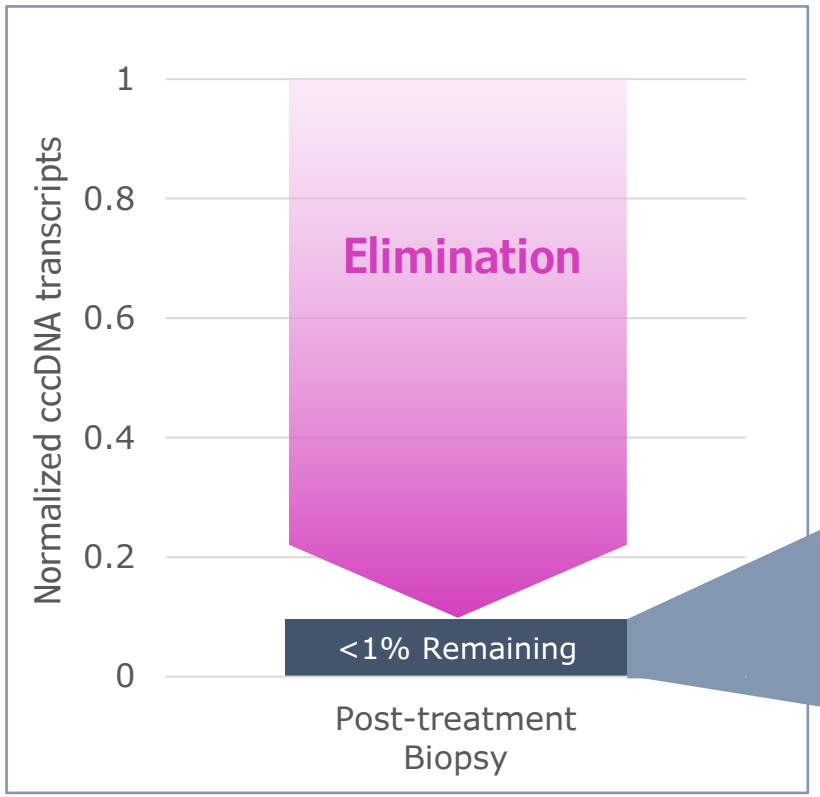


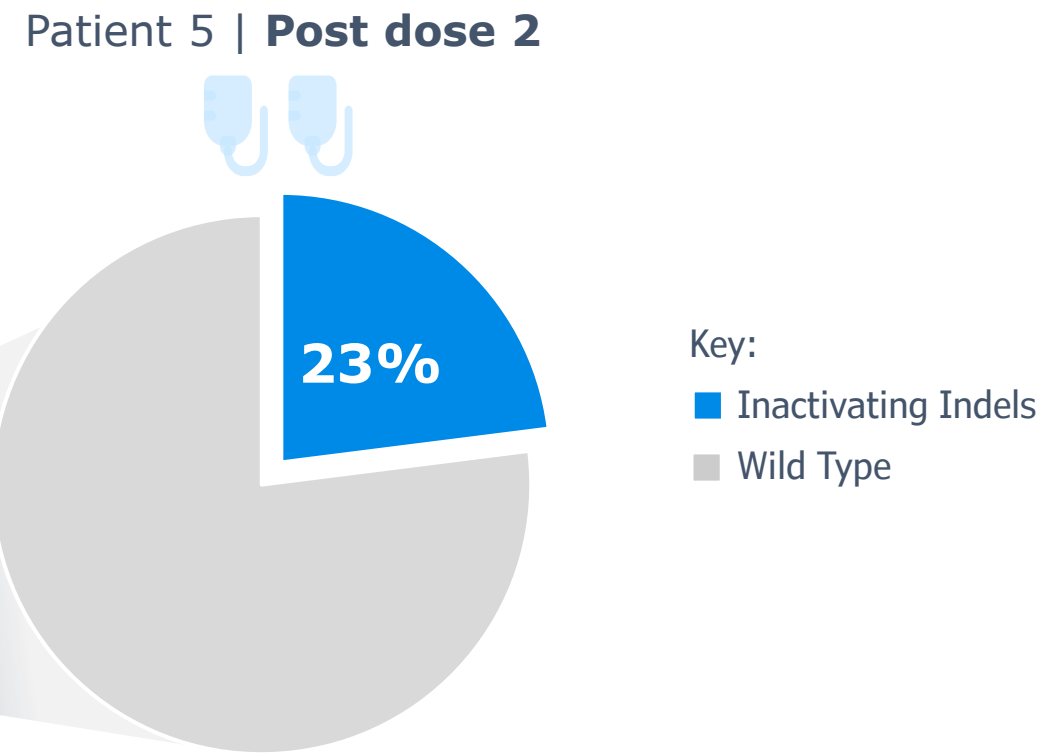


10-fold (1-log) reduction in cccDNA-derived transcripts after 2 administrations of 0.4 mg/kg PBGENE-HBV

cccDNA-derived transcripts were <1% after 2 doses of 0.4 mg/kg PBGENE-HBV.
cccDNA, covalently closed circular DNA.
Data cutoff: May 4, 2026.

EASL DATA: Of the <1% cccDNA Remaining, PBGENE-HBV Indels Permanently Inactivate Viral Replication by Knocking Out Polymerase Function

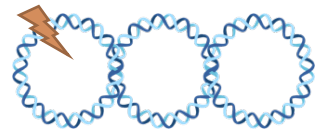




Secondary Mechanism – PBGENE-HBV indels result in complete viral inactivation in any edited cccDNA

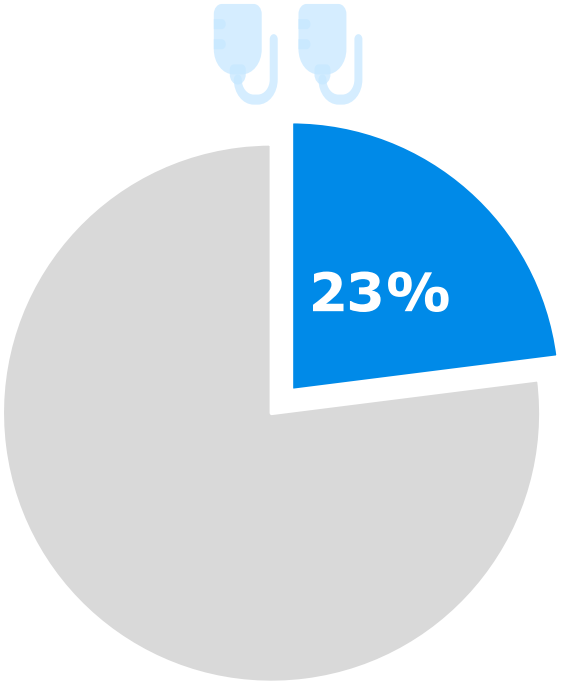
cccDNA, covalently closed circular DNA.
Data cutoff: May 4, 2026.

EASL DATA: Additional cccDNA Inactivation Occurs Upon Subsequent Administrations of PBGENE-HBV

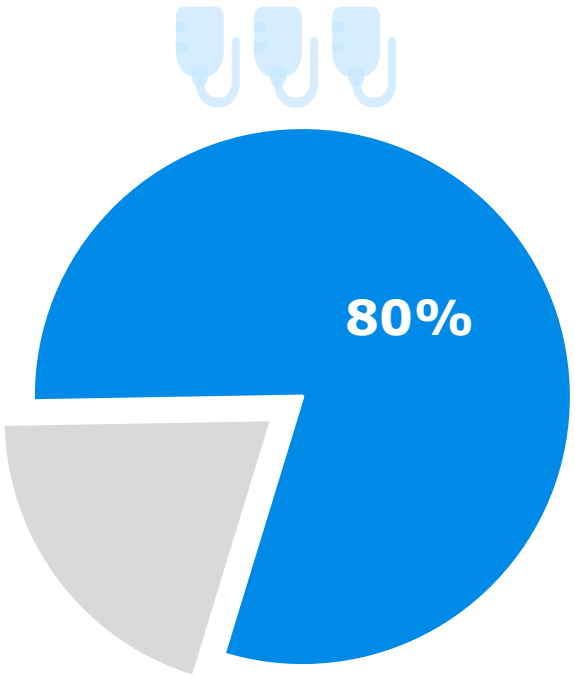


Inactivating Edits

Patient 5 | **Post dose 2**



Patient 6 | **Post dose 3**



Key:
- Inactivating Indels
- Wild Type

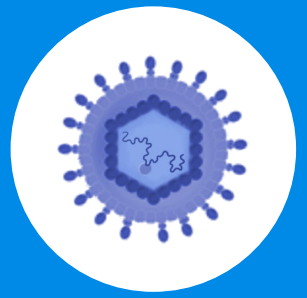
Editing in remaining cccDNA reached 80% after 3 administrations, supporting that cumulative editing leads to permanent inactivation by knocking out polymerase function

cccDNA, covalently closed circular DNA.
Data cutoff: May 4, 2026.

Multiple Datasets Support cccDNA Targeting and Permanence of Effect by PBGENE-HBV

Liver Biopsies

CONFIRMATION OF cccDNA ELIMINATION



pgRNA

POTENT cccDNA EFFECT

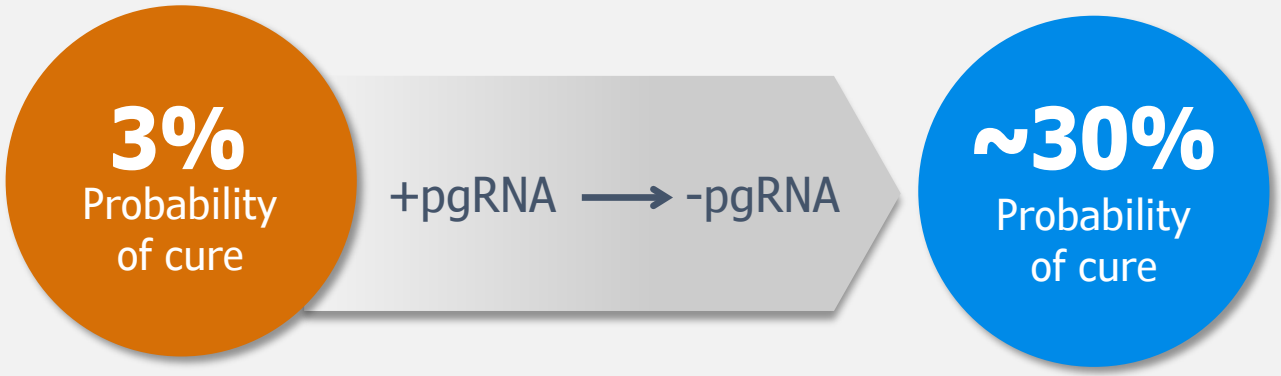
HBsAg

SUBSTANTIAL ANTIVIRAL ACTIVITY IN ALL PATIENTS

cccDNA, covalently closed circular DNA; HBsAg, hepatitis B surface antigen; pgRNA, pregenomic RNA.

Studies Demonstrate Undetectable pgRNA Is Associated With Success of Stopping Nucleoside Analogs and Increasing Probability of Cure

Probability of Cure Stratifies on pgRNA
10X Increase in Probability of Cure With Non-quantifiable pgRNA



Adapted from Terrault NA, et al.

pgRNA is a specific marker for cccDNA indicating viral load, whereas HBsAg protein measures protein load as it primarily comes from nonreplicating, integrated DNA in HBeAg negative patients[1,2]

pgRNA is a positive predictive indicator of viral cure after stopping NAs

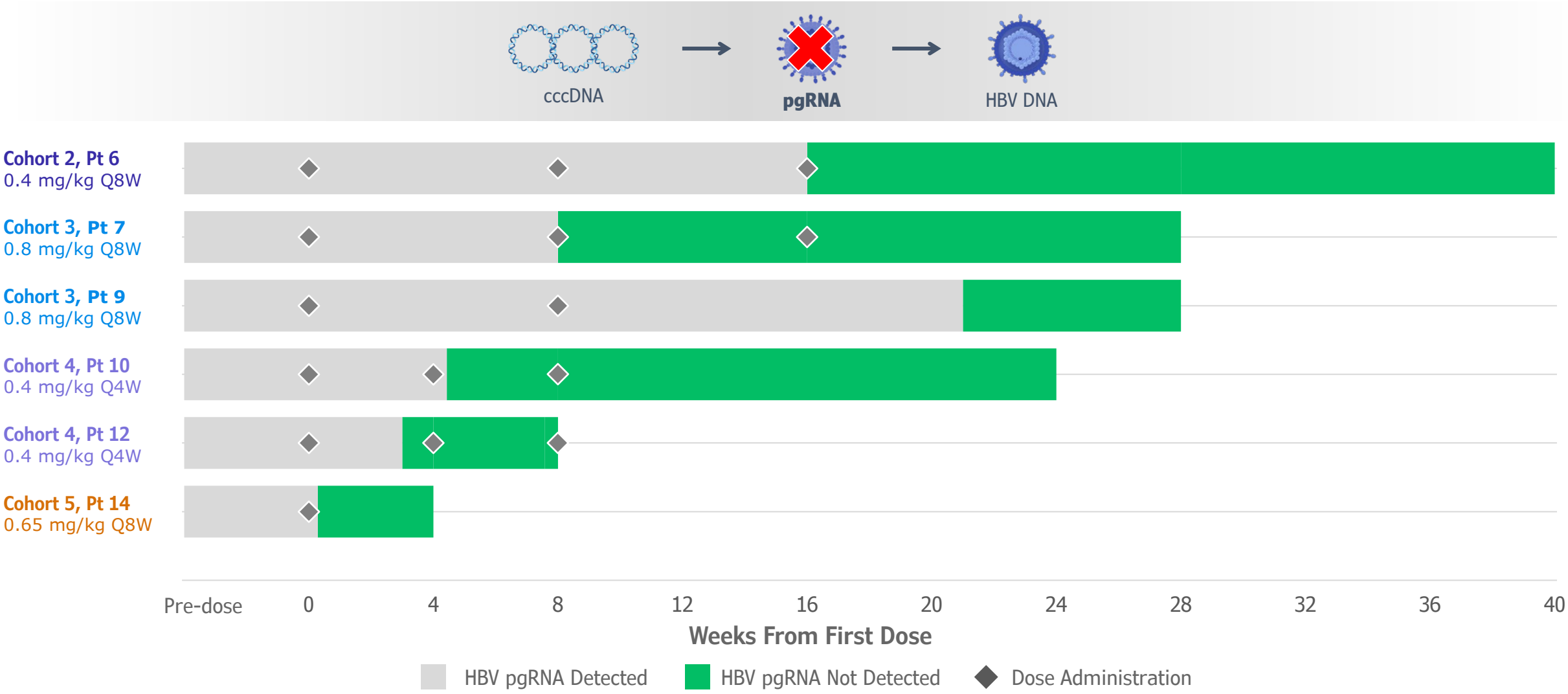
- Melbourne HBV-STOP trial (n=65)[3]
- DARING-B study (n=57)[4]
- Compilation of 23 cohort studies (n=2043)[5]

pgRNA outperforms other biomarkers (eg, HBsAg) in predicting the effective and safe withdrawal of NAs[6]

cccDNA, covalently closed circular DNA; NA, nucleos(t)ide analog; pgRNA, pregenomic RNA (also known as HBV RNA).

1. Meier MA, et al. *J Hepatol*. 2021;75(4):840-847. 2. Taddese M, et al. *JCI*. 2025;135(6):e184243. 3. Thompson AJ, et al. *Hep Comm*. 2023;7:e0188.
4. Papatheodoridi M, et al. *J Viral Hepat*. 2022;29:948-957. 5. Wang DH, et al. *J Viral Hepat*. 2026; 11:e70167. 6. Terrault NA, et al. *J Infect Dis.* 2025;231(5):1290-1298.

EASL DATA: PBGENE-HBV Drove Durable pgRNA Loss in 100% of Patients Who Had Detectable pgRNA at Baseline*



cccDNA, covalently closed circular DNA; pgRNA, pregenomic RNA; Pt, patient; Q4W, every 4 weeks; Q8W, every 8 weeks.
*Evaluable patients defined as those who have 28 days of data from initial dose.
Data cutoff: May 4, 2026.

Multiple Datasets Support cccDNA Targeting and Permanence of Effect by PBGENE-HBV

Liver Biopsies

CONFIRMATION OF cccDNA ELIMINATION

pgRNA

POTENT cccDNA EFFECT

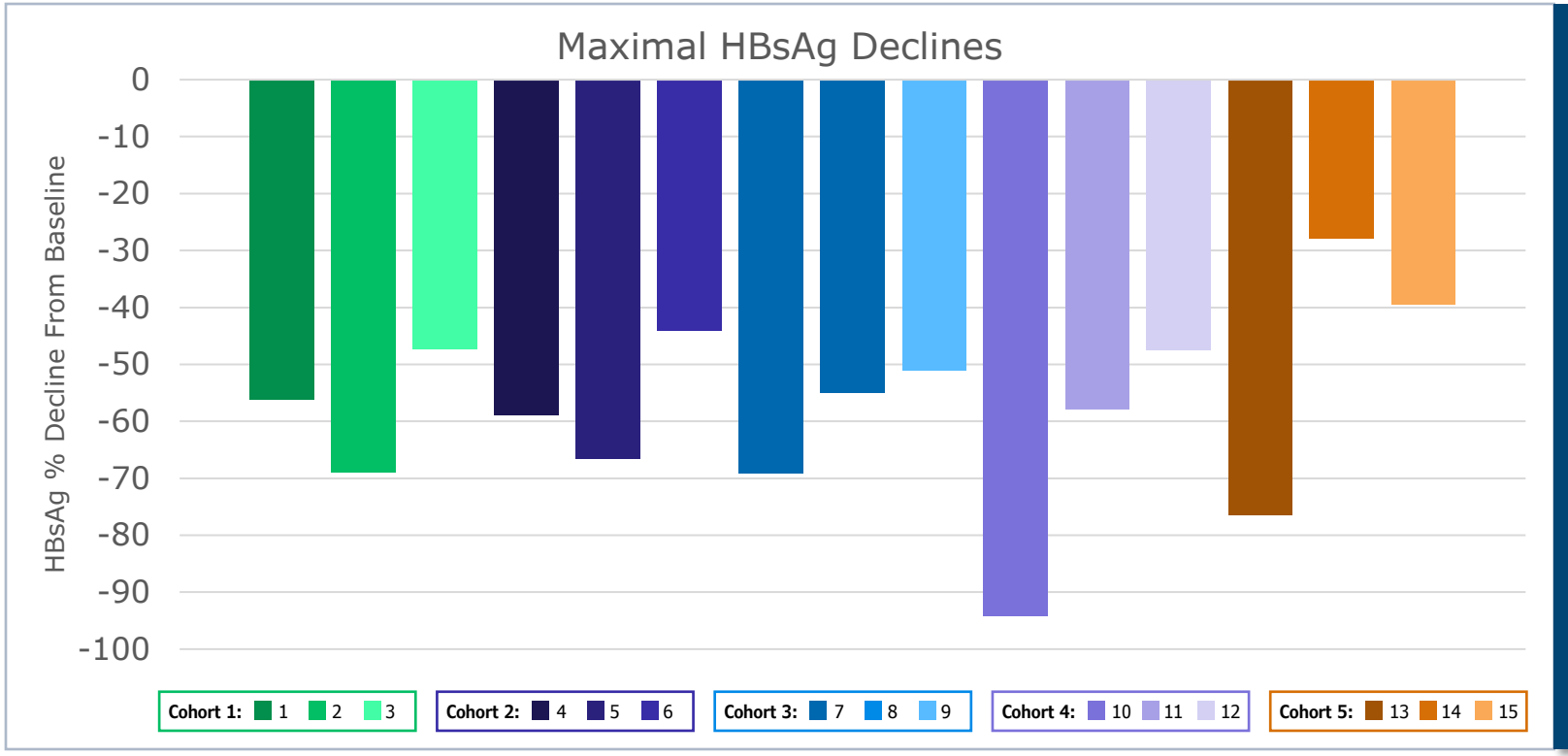
HBsAg

SUBSTANTIAL ANTIVIRAL ACTIVITY IN ALL PATIENTS

Durable HBsAg reductions across all patients treated; **highly supportive of cccDNA elimination** by PBGENE-HBV

cccDNA, covalently closed circular DNA; HBsAg, hepatitis B surface antigen; pgRNA, pregenomic RNA.

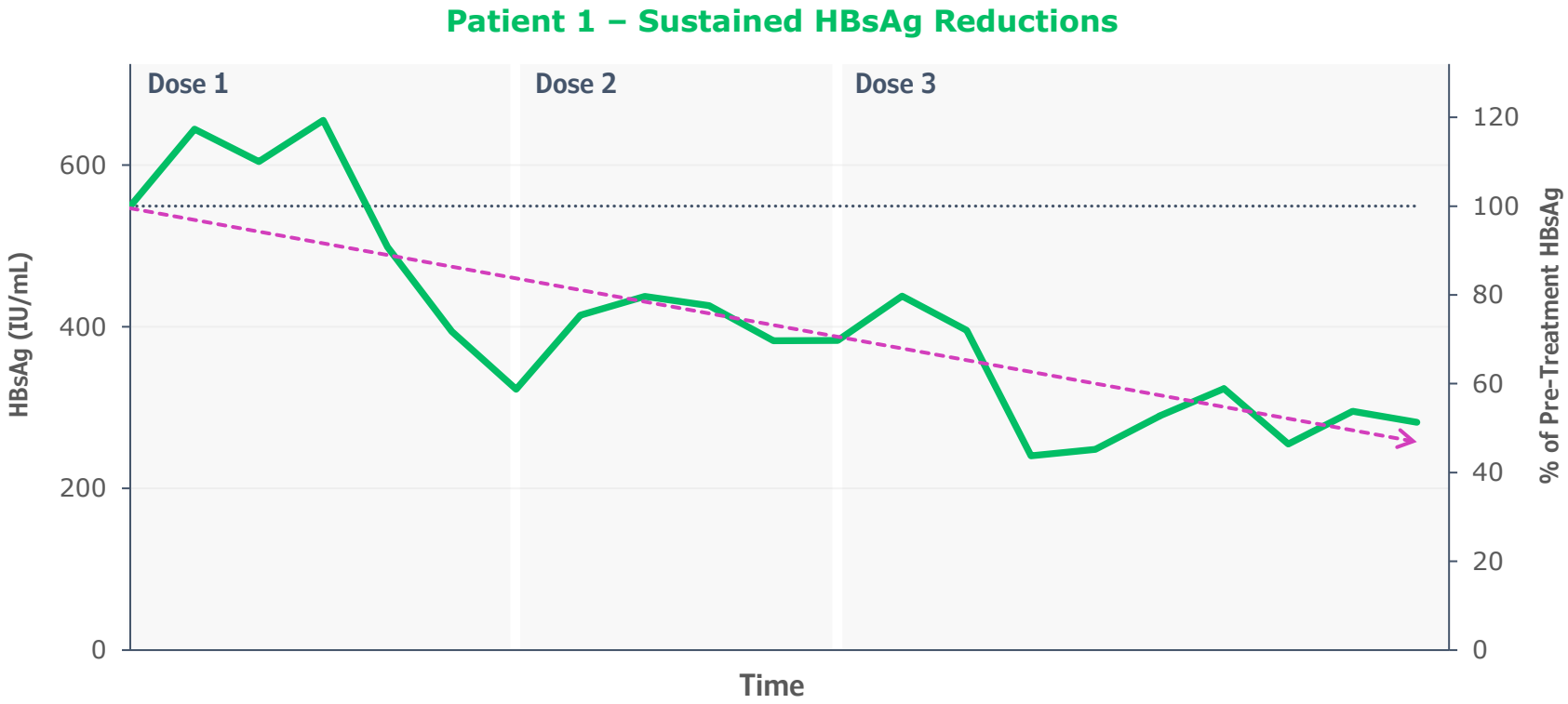
EASL DATA: PBGENE-HBV Demonstrates Proof of Editing in 100% of Evaluable Patients Across All Cohorts, Regardless of Baseline S-Antigen Levels



Significant maximal declines in HBsAg supporting PBGENE-HBV activity across all patients (n=15)

Patient 16 is not evaluable due to incomplete follow-up at time of data cut.
HBsAg, hepatitis B surface antigen.
Data cutoff: May 4, 2026.

EASL DATA: Permanent Editing Effect in First Patient Treated With PBGENE-HBV Now >1 Year Since Initiating Treatment



Stable HBsAg decline >1 year after initial dose of PBGENE-HBV supports permanence of gene editing

HBsAg, hepatitis B surface antigen.
Data cutoff: May 4, 2026

Phase 1 ELIMINATE-B Trial
Safety Data

PBGENE-HBV Safety Profile Executive Summary

- 38 doses administered across 16 patients in 5 cohorts
- No dose-limiting toxicities observed
- Most common adverse events include infusion-related reactions consistent with LNP effects, with onset and resolution within 24 hours of infusion
- While transient, reversible ALT/AST lab abnormalities have been observed, they were asymptomatic with no elevated bilirubin, and no Hy's Law in any patient and at any dose level
- Grade 3 hypotension was observed during dose escalation
- One patient in the highest-dose cohort (0.8 mg/kg) experienced two SAEs after the second LNP administration
- Etiology of hypotension is now understood, and simple measures have ameliorated clinically significant decreases in blood pressure

Experience with PBGENE-HBV broadens, as does understanding of how to manage, mitigate, and prevent future AEs

A DLT is defined in protocol as any clinically significant, organ-specific, treatment-emergent adverse event (AE) ≥grade 3 that does not decrease to ≤grade 2 within 7 days and is related to study medication.
AE, adverse event; ALT, alanine aminotransferase; AST, aspartate aminotransferase; LNP, lipid nanoparticle; SAE, serious adverse event.
Data cutoff: May 4, 2026.

Simple and Mechanism-Driven Safety Measures Have Mitigated Hypotension and Liver Lab Abnormalities

LNP-inflammatory response

- Rapid onset of complement and cytokine elevations after LNP dosing which resolve in 24 hours
- LNP-driven transient elevations of complement and cytokines drive hypotension
- Slower infusion rate and prophylactic measures have resulted in decreased inflammation and ameliorated hypotensive events

Initial prophylaxis and infusion rate

- **Antihistamine (Day -1 and 0)**
- **LNP infused over 2 hours**
- **Steroids (Day -1 and 0)**

Current prophylaxis and infusion rate

- **Antihistamine—no change**
- **LNP infused over 5 hours**
- **Steroids (Day -1 and 0)—dose increased**
- Acetaminophen (Day 0)
- Normal saline infusion

Since implementing new prophylaxis and infusion measures, no grade 3 hypotension and no LNP-related ALT/AST lab abnormalities

LNP, lipid nanoparticle.

Summary of PBGENE-HBV Clinical Data

Safety and Efficacy Findings Substantiate a Clear Therapeutic Window for Driving Potential Viral Cures in Chronic Hepatitis B

- **Established primary mechanism of direct cccDNA elimination through liver biopsy, pgRNA, and HBsAg data**
  - 1-log reduction in cccDNA transcripts observed in liver biopsies
  - All evaluable patients with detectable pgRNA at baseline show enduring pgRNA loss after treatment with PBGENE-HBV
  - Durable declines in HBsAg for all evaluable patients consistent with elimination of cccDNA
- **6 of 6 patients across various dosing cohorts demonstrated complete loss of pgRNA and significant reductions in HBsAg**
- **Robust characterization and understanding of mechanisms impacting repeat LNP dosing across 16 patients in 5 Cohorts**
- **Targeted and simple mitigations implemented have resolved primary drivers of hypotension-related AEs**
  - ~20% of all doses to date have been given with new safety mitigations, including extended infusion time & increased steroid dose
  - No grade 3 or 4 LNP-related AEs have occurred since mitigations implemented (7 of 38 doses administered)
- **Multiple dose and schedule options are viable for expansion phase of trial, with new safety mitigation protocol implemented**

AE, adverse event; cccDNA, covalently closed circular DNA; HBsAg, hepatitis B surface antigen; LNP, lipid nanoparticle; pgRNA, pregenomic RNA.
Data cutoff: May 4, 2026.

Transforming the Natural History of cHBV Through Direct Viral Targeting of cccDNA

CONTINUE

- **Expand number of patients** in cohort 4 (0.4 mg/kg) and cohort 5 (0.65 mg/kg)
  - Expand upon clinical experience, enroll patients in France & Romania
- **Increase size and strength of data set for regulators**
  - Collect additional biopsies to support undetectable pgRNA biomarker
  - Goal to establish PBGENE-HBV as foundational for HBV viral cure

STOP NUC

- **Work with global investigators to design next phase of clinical trial**

  Initial thinking on NUC stop criteria:
  - pgRNA loss for ≥6 months proxy for HBV DNA
  - Normalized liver enzymes
  - Sustained reductions in HBsAg
- **Evaluate current patients with pgRNA loss** for NUC withdrawal
  - Expand to baseline pgRNA-negative patients after viral cure proven in patients with pgRNA loss

EXPAND

- **Select optimal dosing schedule** for phase 1 expansion and phase 2
- **Lifecycle management for PBGENE-HBV**
  - Broaden to an enriched pgRNA+/cccDNA patient population, HBeAg+ patients
  - Continue to assess PBGENE-HBV as potential viral cure via monotherapy and through combination

Plan for Expanding ELIMINATE-B and Stopping NUCs

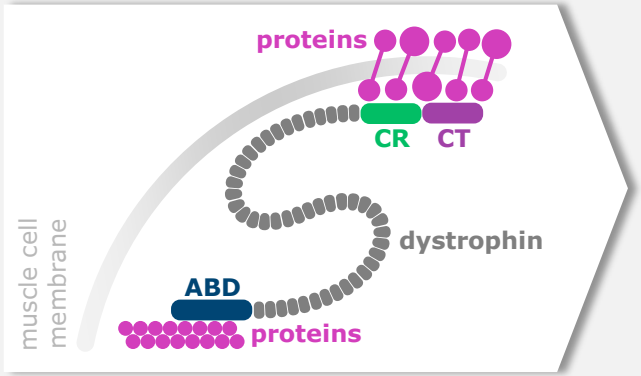
cccDNA, covalently closed circular DNA; cHBV, chronic hepatitis B virus; HBeAg, hepatitis B e antigen; HBsAg, hepatitis B surface antigen; NUC, nucleos(t)ide analog; pgRNA, pregenomic RNA.

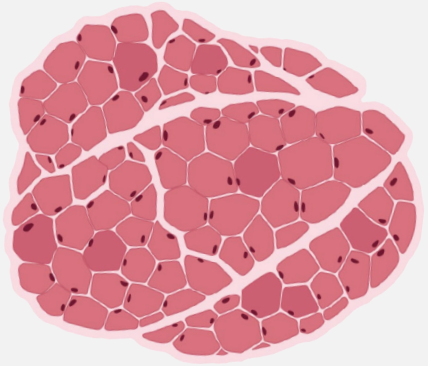
PBGENE-DMD
for Duchenne Muscular Dystrophy

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

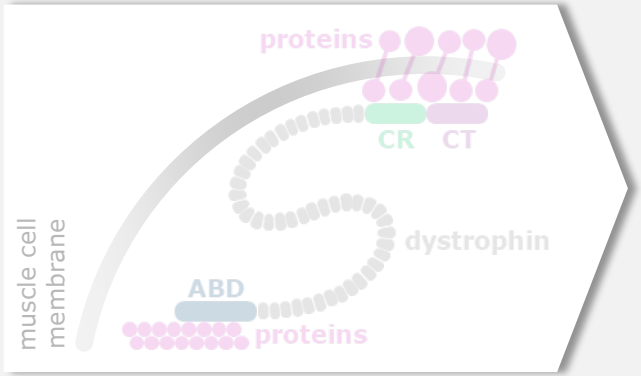


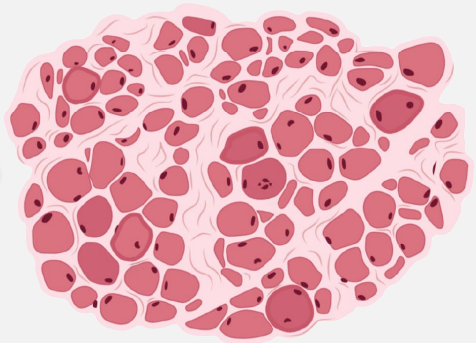


Healthy muscle

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

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

35

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

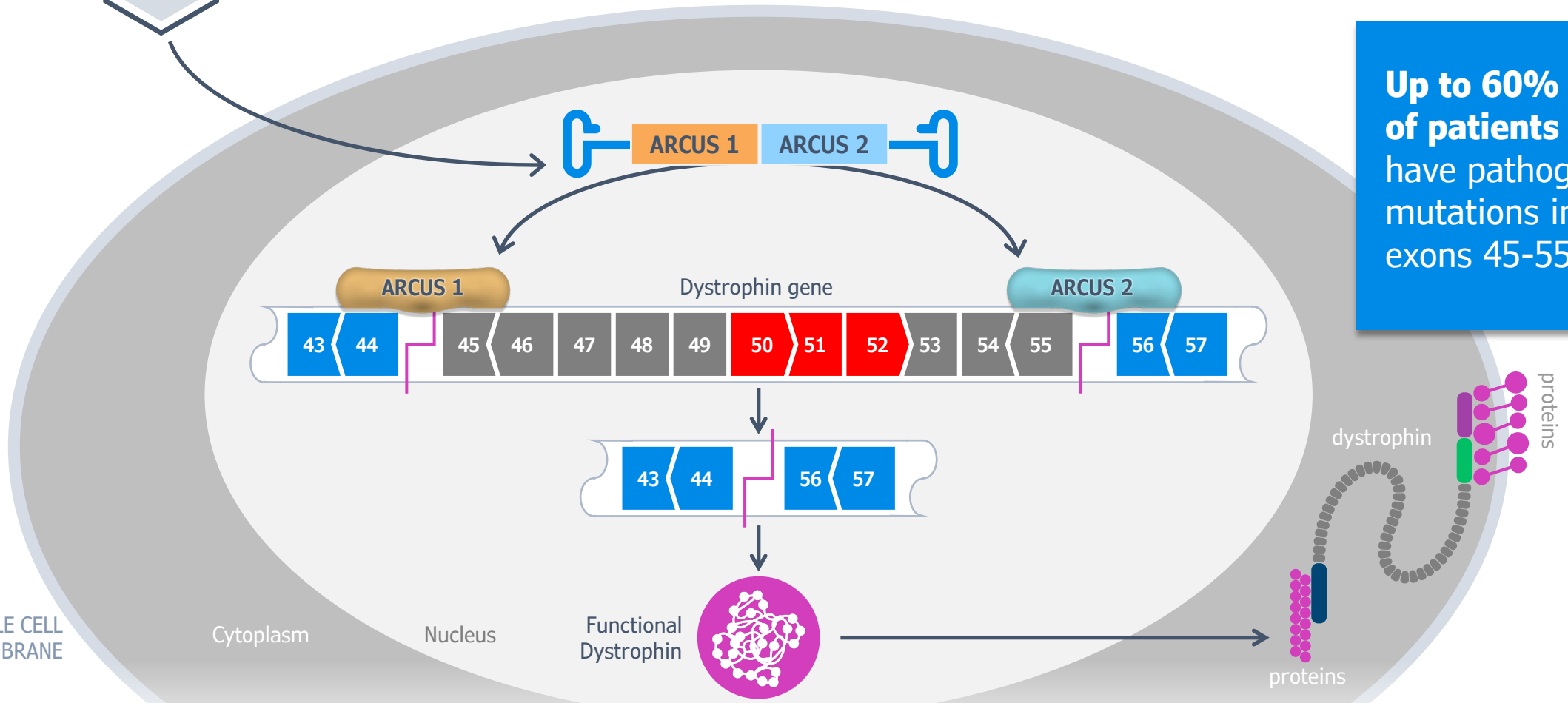
An ideal therapy would achieve the following:

- ☑ 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.

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

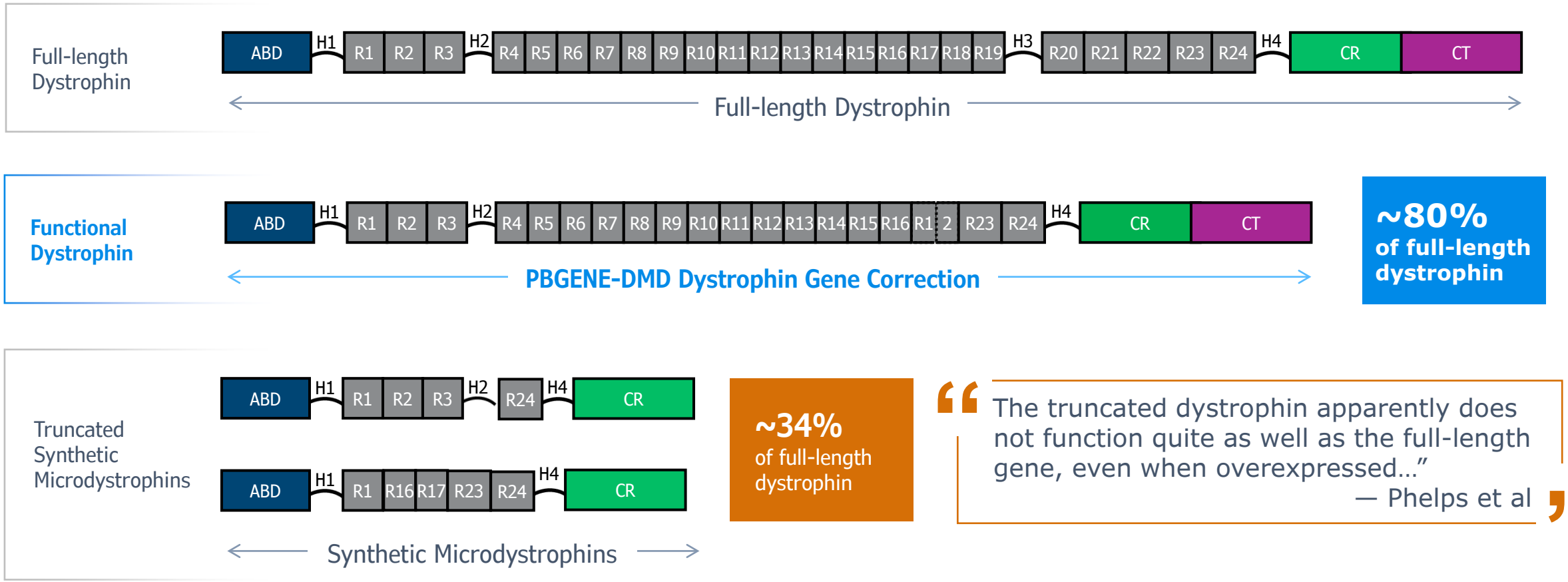


Up to 60% of patients have pathogenic mutations in exons 45-55[1]

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

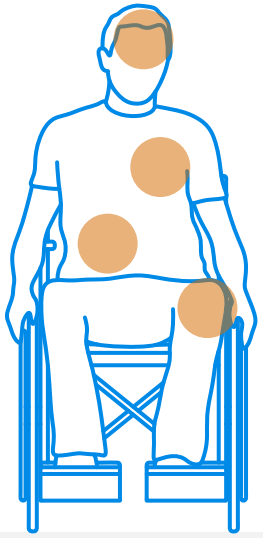


> "The truncated dystrophin apparently does not function quite as well as the full-length gene, even when overexpressed…"
>
> — Phelps et al

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



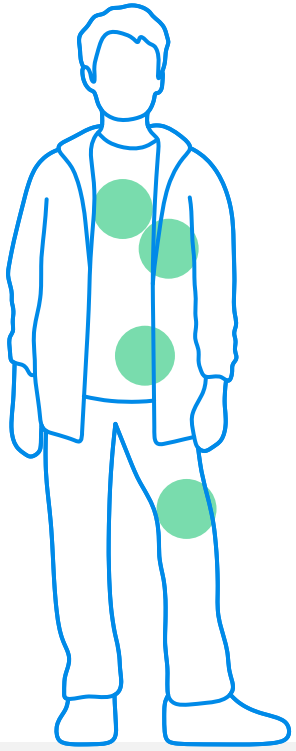
Lifespan:

- 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:

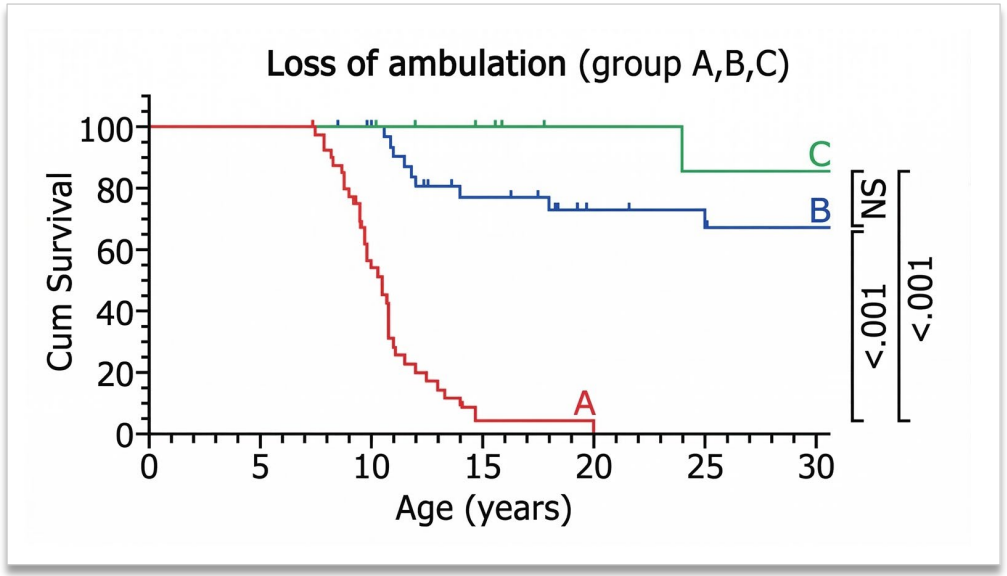
- Asymptomatic or mild symptoms[1-4]
- Normal muscle strength and ambulation throughout life[1,2]
- Normal respiratory function[2]
- Myocardial involvement, often manageable with medication[2]

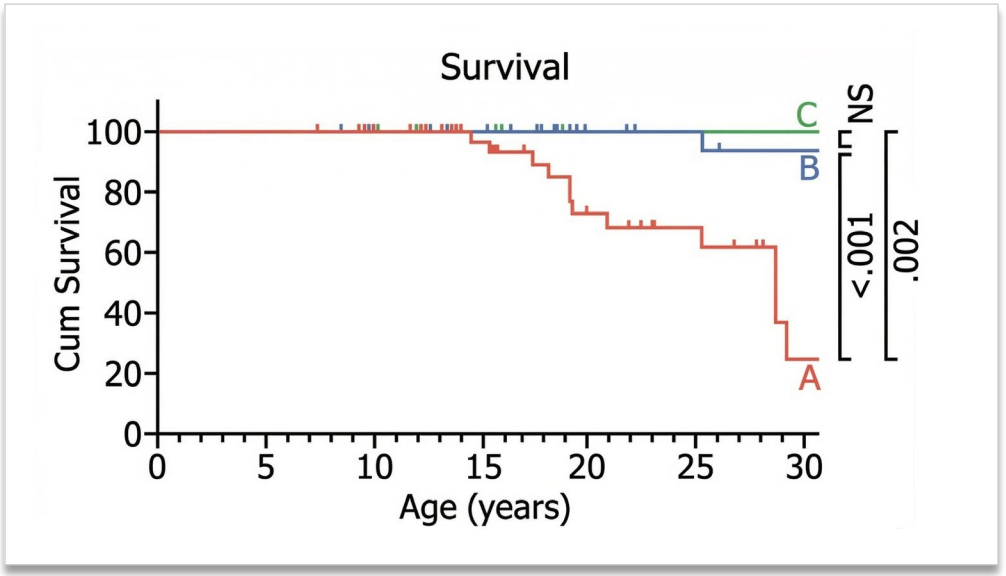
Del45-55 in-frame Dystrophin gene (BMD)

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)

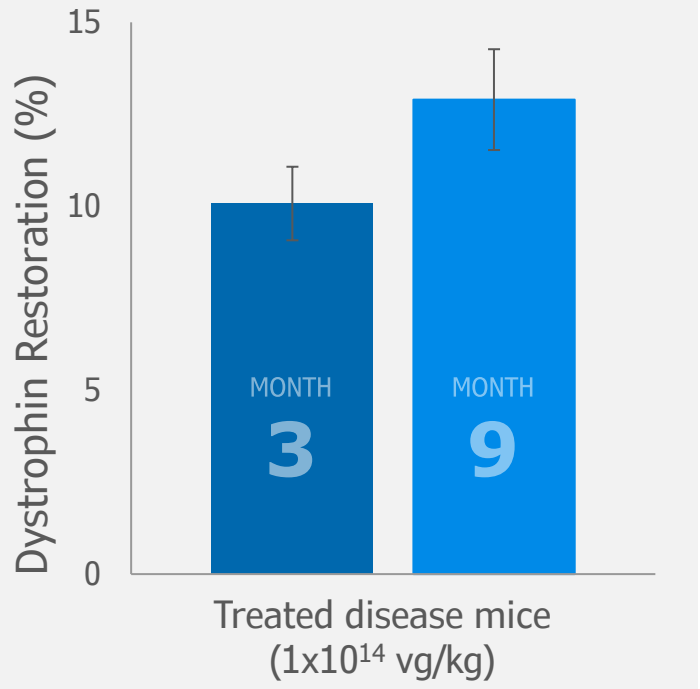
› ~5% dystrophin associated with prolonged ambulation beyond typical loss of ambulation time (~10–12 years)and linked to improved survival and milder diseases

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

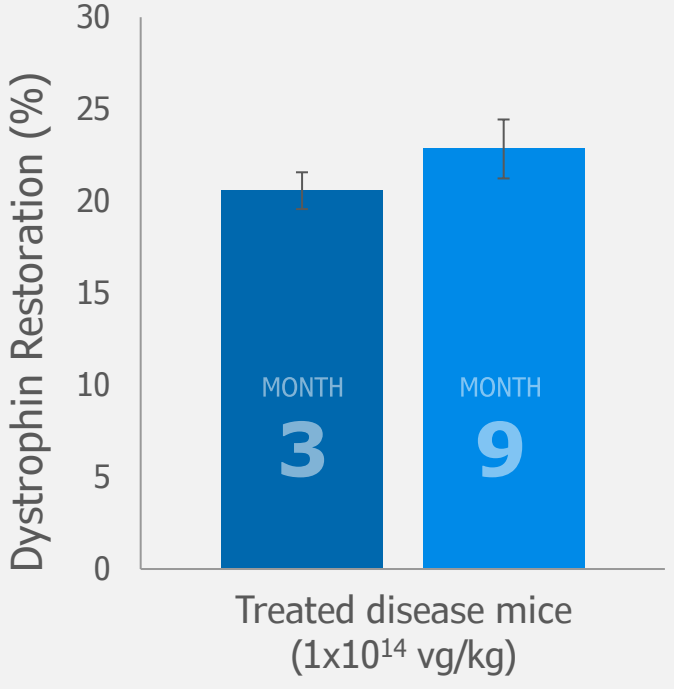
Feraudy et al. Ann Neurol, 2021 Feb;89(2):280-292.

PBGENE-DMD Resulted in Increased Dystrophin Protein Levels Over Time, Exceeding Expected Therapeutic Threshold

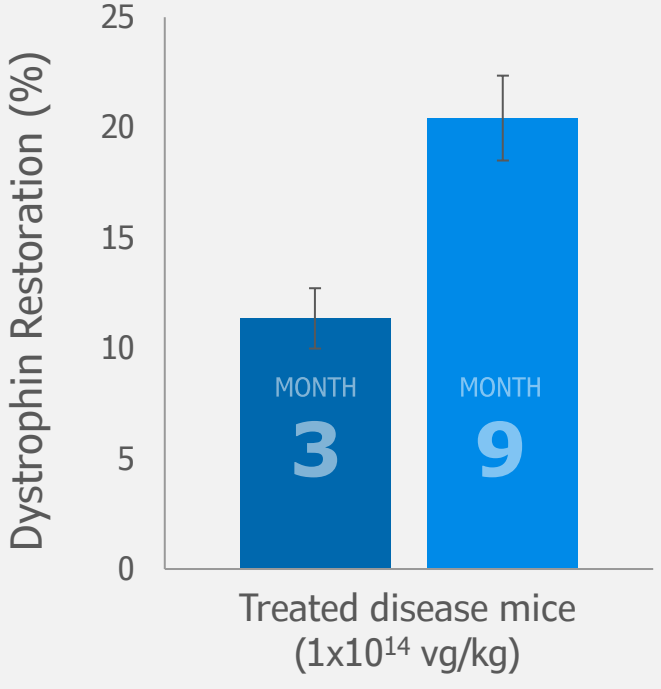
Heart



Calf



Quadriceps

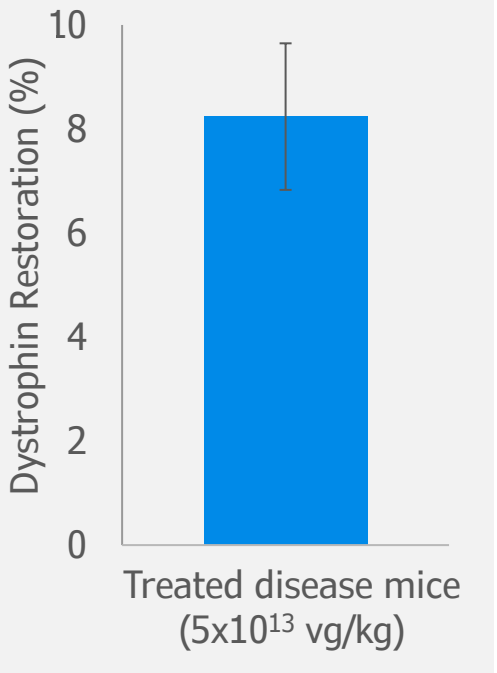


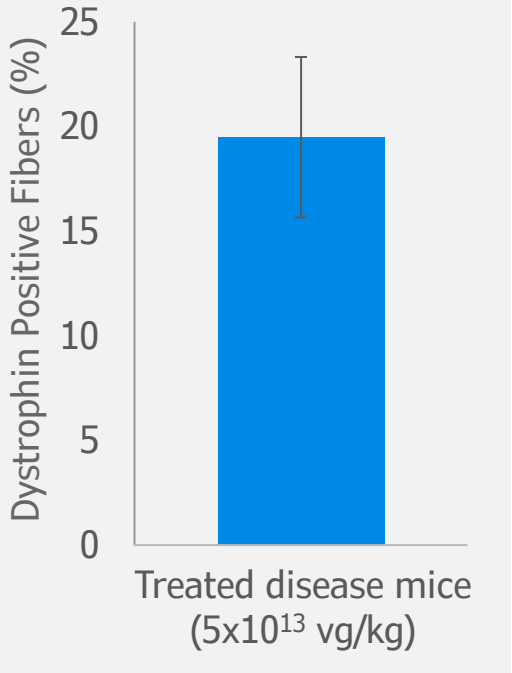
Endogenously produced, near-full–length functional dystrophin protein increases through 9 months in mice

DMD, Duchenne muscular dystrophy; vg/kg, vector genomes per kilogram.

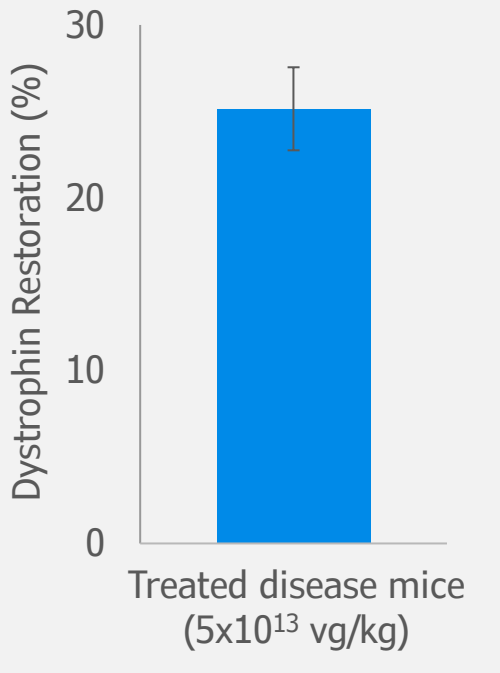
NEW ASGCT DATA: PBGENE-DMD Achieves Therapeutic Dystrophin Restoration in Critical Respiratory Muscles of Early Juvenile DMD Mice

Diaphragm





Intercostals



Dystrophin protein restoration in early juvenile mice was up to 2.5-12X higher in skeletal and respiratory muscle compared to late juvenile mice

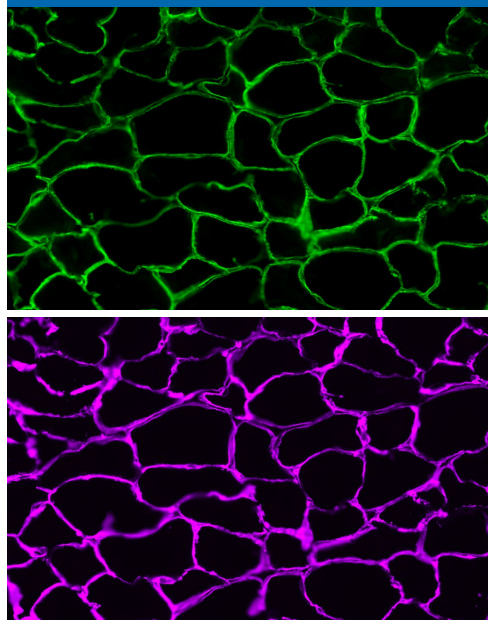
Dystrophin positive fibers in early juvenile mice were up to 2-3X higher in skeletal and respiratory muscle compared to late juvenile mice

Broad and robust efficacy in early juvenile mice supports potential benefit of earlier intervention of PBGENE-DMD in younger DMD patient population

DMD, Duchenne muscular dystrophy; vg/kg, vector genomes per kilogram.
ASGCT 2026.

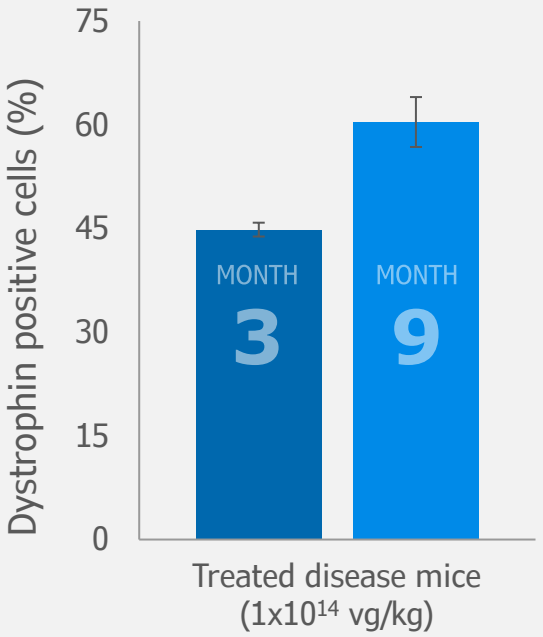
NEW ASGCT DATA: Broad Distribution of Dystrophin Positive Cells Across Skeletal, Cardiac, and Respiratory Muscles After PBGENE-DMD Treatment

PBGENE-DMD
Disease Model

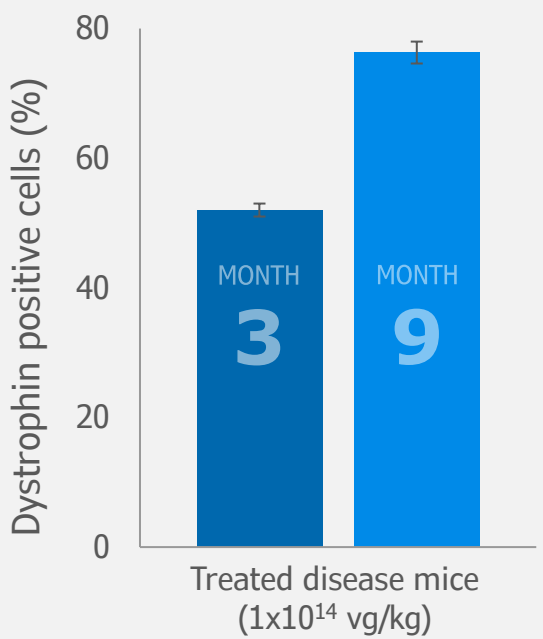


Dystrophin Laminin

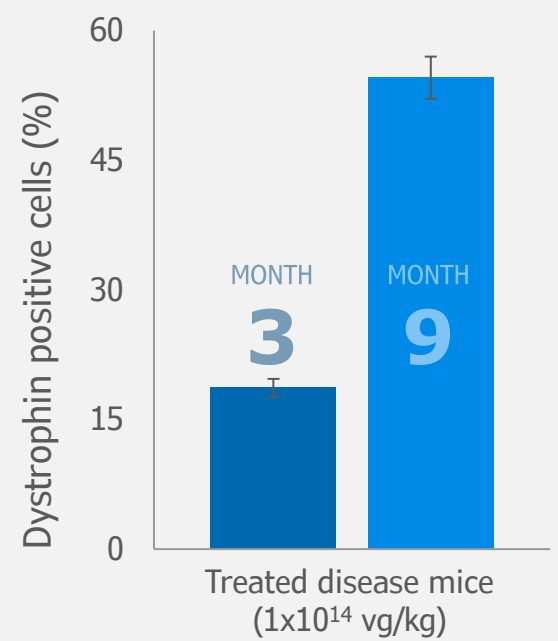
Heart



Calf



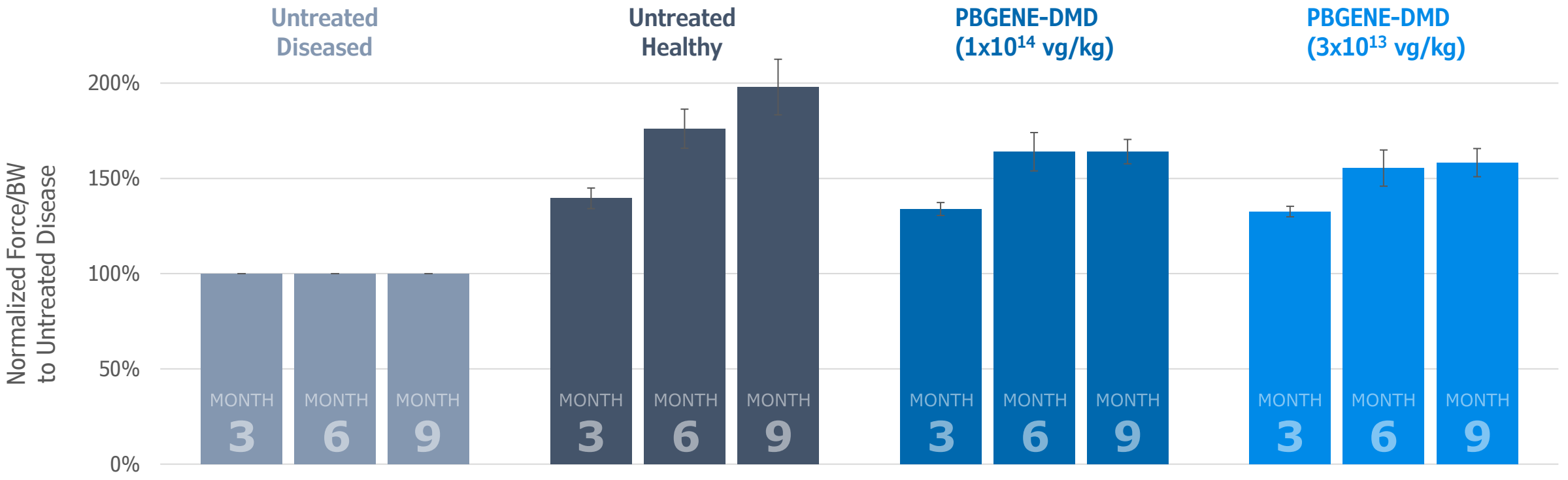
Quadriceps



Percentage of cells expressing near-full–length dystrophin protein increases over time after treatment with PBGENE-DMD

DMD, Duchenne muscular dystrophy; vg/kg, vector genomes per kilogram.
ASGCT 2026.

PBGENE-DMD Significantly Improved Muscle Function and Demonstrated Potential for 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. N=10 mice per cohort.
DMD, Duchenne Muscular Dystrophy; vg/kg, vector genomes per kilogram.
ASGCT 2025.

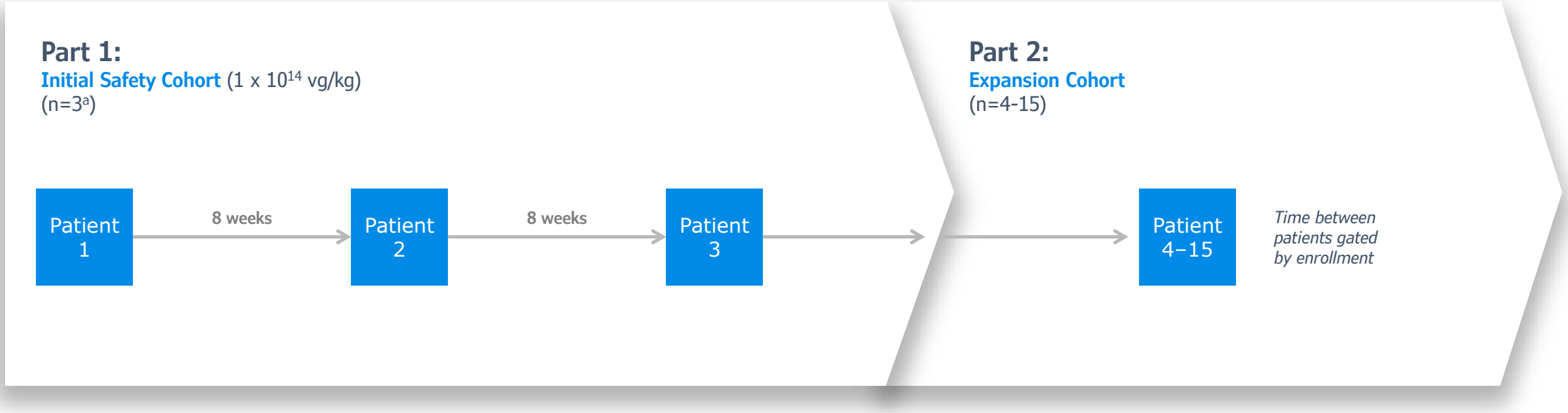
FUNCTION-DMD
Phase 1/2 Clinical Trial

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.

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

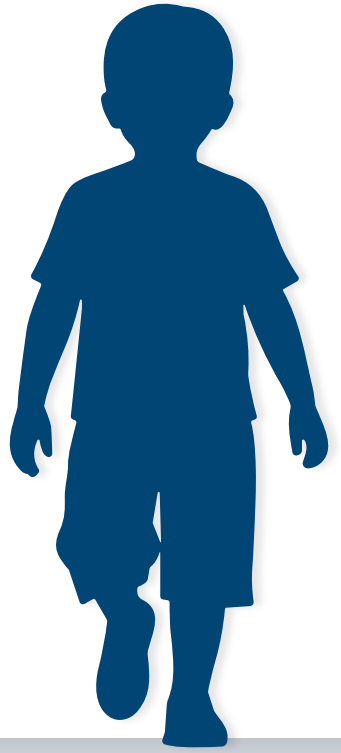
Exploratory Endpoints

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

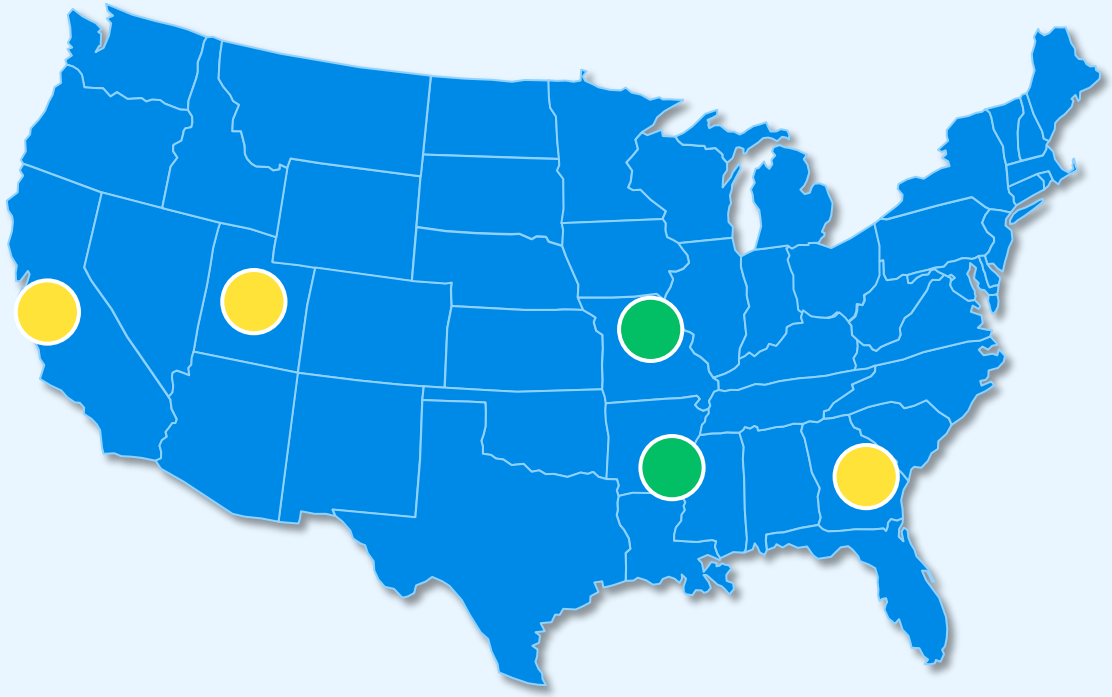
Eculizumab
Prevents immune-mediated tissue damage by blocking the complement pathway

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. Sites activated at Arkansas Children's Hospital in Little Rock and Washington University in St. Louis.
- **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.
Note: green circle denotes active site

Summary and Financials

Multiple Near-Term Data Catalysts Supported Through Cash Runway

eliminateB

PBGENE-HBV for Chronic Hepatitis B

- Viral gene elimination program with 38 doses delivered across 16 patients treated in 5 cohorts to date. Responses at all dose levels reported to date
- First and only clinical-stage program designed to eliminate and inactivate root cause of viral replication—directly targeting cccDNA
- Clinical data now validate direct targeting and elimination of cccDNA for the first time ever
- Next update targeting year end 2026

functionDMD

PBGENE-DMD for Duchenne Muscular Dystrophy

- PBGENE-DMD is designed to provide durable functional muscle improvement targeting ~60% of patients with DMD
- First clinical trial sites activated at Arkansas Children's Hospital and Washington University, St-Louis
- Next step: commence trial and execute in clinic, initial safety data targeted year end 2026

Cash runway through multiple catalysts:

Strong balance sheet with >$112 million in cash (as of 6/30/26) with expected cash runway through 2028[1]

[1]$112.4M cash, cash equivalents, and restricted cash as of 6/30/26. The Company expects existing cash and cash equivalents, continued fiscal and operating discipline, and availability of its ATM facility are expected to provide sufficient cash runway through 2028.

Precision BioSciences Leadership Team Driving Programs Through Next Clinical Milestones

Michael Amoroso
Since Oct 2021
President &
Chief Executive Officer

Cassie Gorsuch, Ph.D.
Since June 2016
Chief Scientific Officer

Alex Kelly
Since Oct 2020
Chief Operating Officer

Naresh Tanna
Since Jan 2022
Chief Financial Officer

Dario Scimeca
Since June 2019
Chief Legal Officer

Cindy Atwell
Since April 2019
Chief Development Officer

Jeff Smith, Ph.D.
Since March 2006
Chief Research Officer,
Co-Founder

Neil Leatherbury
Since March 2017
Senior VP, CMC

Juli Blanche
Since May 2022
Chief People Officer