AASLD 2026 Investor Update:

Direct cccDNA Elimination Leads to an Era of Potential Chronic Hepatitis B ***Viral Cure***

November 9, 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 design of PBGENE-HBV for viral cure through cccDNA elimination; meeting Phase 1 ELIMINATE-B objective and identifying go-forward dose of 0.4 mg/kg for a Phase 2 study; key expected inflection points through 2027 for PBGENE-HBV including initiating the ELIMINATE-B2 global Phase 2 study in Q1 2027 and rapidly enrolling that trial as well as continued Phase 2 study execution, rolling viral cure data and Phase 2 combination data compared to bepirovirsen alone B-Well studies with 19% functional cure rate in H2 2027; key expected inflection points through 2027 for PBGENE-DMD including a safety update from initial PBGENE-DMD patients, 3 month dystrophin from those initial patients and expansion into Part 2 of the FUNCTION-DMD trial in H1 2027 and 12 month dystrophin and functional data from initial patients in H2 2027; expansion expectations around reproducible liver biopsies and durability of pgRNA loss; expectations around cccDNA elimination as the only path to viral cure; goal of PBGENE-HBV to be the foundation of HBV treatment as either a monotherapy to deliver viral cure or part of a combination with an ASO to drive functional cures; expectations that PBGENE-HBV eliminates cccDNA, potentially changing the natural history of chronic Hepatitis B enabling a viral cure; expectations around PBGENE-HBV driving durable pgRNA loss in both HBeAg positive and negative patients; pgRNA loss in HBeAg positive patient with 3.7-log baseline pgRNA demonstrating PBGENE-HBV potently eliminates cccDNA in the highest viral burden population; robust efficacy data supporting 0.4 mg/kg as the go forward dose for PBGENE-HBV; favorable therapeutic index for PBGENE-HBV at 0.4 mg/kg supports further evaluation in Phase 2; reducing cccDNA load/viral burden is believed to be essential to deliver cure to more patients and PBGENE-HBV has potential to achieve this as a monotherapy and/or in combination with any other therapies; expectation that PBGENE-HBV cccDNA elimination may lead to a deeper suppression of viral transcripts with bepirovirsen potentially resulting in a higher functional cure rate; expected initiation of dosing of Phase 2 study in Q1 2027 and rolling data for viral and functional cure from ELIMINATE-B2 global Phase 2 study in H2 2027, expectations that PBGENE-DMD continues to advance in FUNCTION-DMD study and is on track to meet goal of 3 patients enrolled by end of 2026 and next steps of Part 1 (n=3) safety and 12-week biopsy readouts and continuing to Part 2 of the study (n=12, concurrent enrollment); and sufficiency of our expected cash runway into Q4 2029 to achieve key next stage inflection points in the PBGENE-HBV and PBGENE-DMD clinical trial. 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,” "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; 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 and Quarterly Report on Form 10-Q for the quarterly period ended September 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**.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and other data bout our industry. These data involve a number of assumptions and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk. Neither we nor our affiliates, advisors or representatives make any representation as to the accuracy or completeness of that data or undertake to update such data after the date of this presentation. You agree to keep any information provided herein confidential and not to disclose any of the information to any other parties without our prior express written permission. Neither the information contained in this presentation or any further information made available by us or any of our affiliates or employees, directors, representatives, officers, agents or advisers in connection with this presentation will form the basis of, or be construed as, a contract or any other legal obligation. This presentation shall not constitute an offer to sell or the solicitation of an offer to buy any of the company's securities, nor shall there be any sale of these securities in any state or other jurisdiction in which such offer, solicitation or sale would be unlawful prior to registration or qualification under the securities laws of any such state or other jurisdiction.

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.

Today's Speakers



Michael Amoroso

President & Chief Executive Officer
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Cassie Gorsuch, PhD

Chief Scientific Officer
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Li Shu Fan Medical Foundation Professor in Medicine,
The University of Hong Kong, Hong Kong

Setting the Stage for Viral Cure



Michael Amoroso
President & Chief Executive Officer
Precision BioSciences, Inc.



The Problem:
cccDNA is the root cause of chronic hepatitis B

Designed for Viral Cure Through cccDNA Elimination:

PBGENE-HBV

All other therapies suppress while preserving cccDNA:

ASO, NUCs, siRNA, CAMs, entry inhibitors, epigenetic modifiers, mAbs, immune therapies

ASO, antisense oligonucleotide; CAM, capsid assembly modulator; cccDNA, covalently closed circular DNA; HBV, hepatitis B virus; mAb, monoclonal antibody; MOA, mechanism of action; NUC, nucleos(t)ide analog; siRNA, small interfering RNA.

Overview of MOAs

Since May 2026 at EASL…

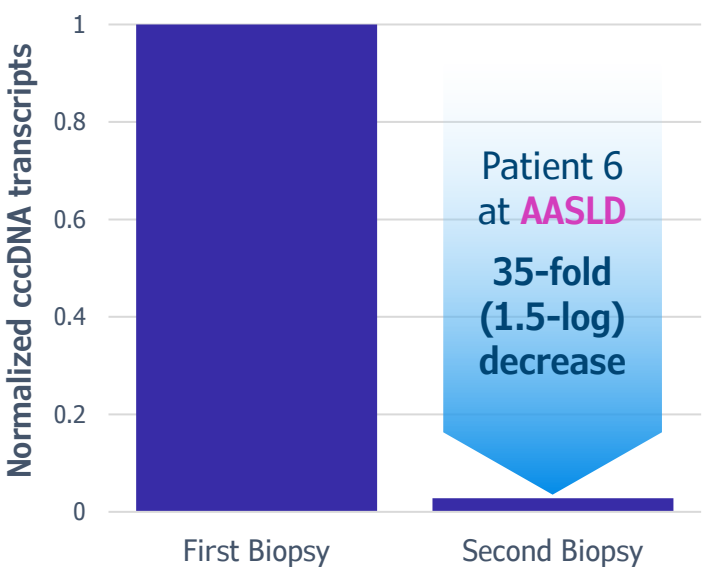
- ✓ Treatment Effect in 100% of Patients
  - 38 administrations, 16 patients
- ✓ From the Liver to the Blood → Advent of pgRNA blood biomarker
  - 100% of patients with durable pgRNA undetectable
- ✓ Evidence of Direct Elimination of cccDNA in Biopsy
  - 1log reduction
- ✓ Safety Characterization Across Doses 0.2-0.8 mg/kg

★ **Phase 1 Global ELIMINATE-B Objective Met:** With 59 Administrations in 25 Patients, Demonstrated cccDNA Elimination and Favorable Safety Profile at Go-Forward Dose of 0.4 mg/kg for Phase 2 Study

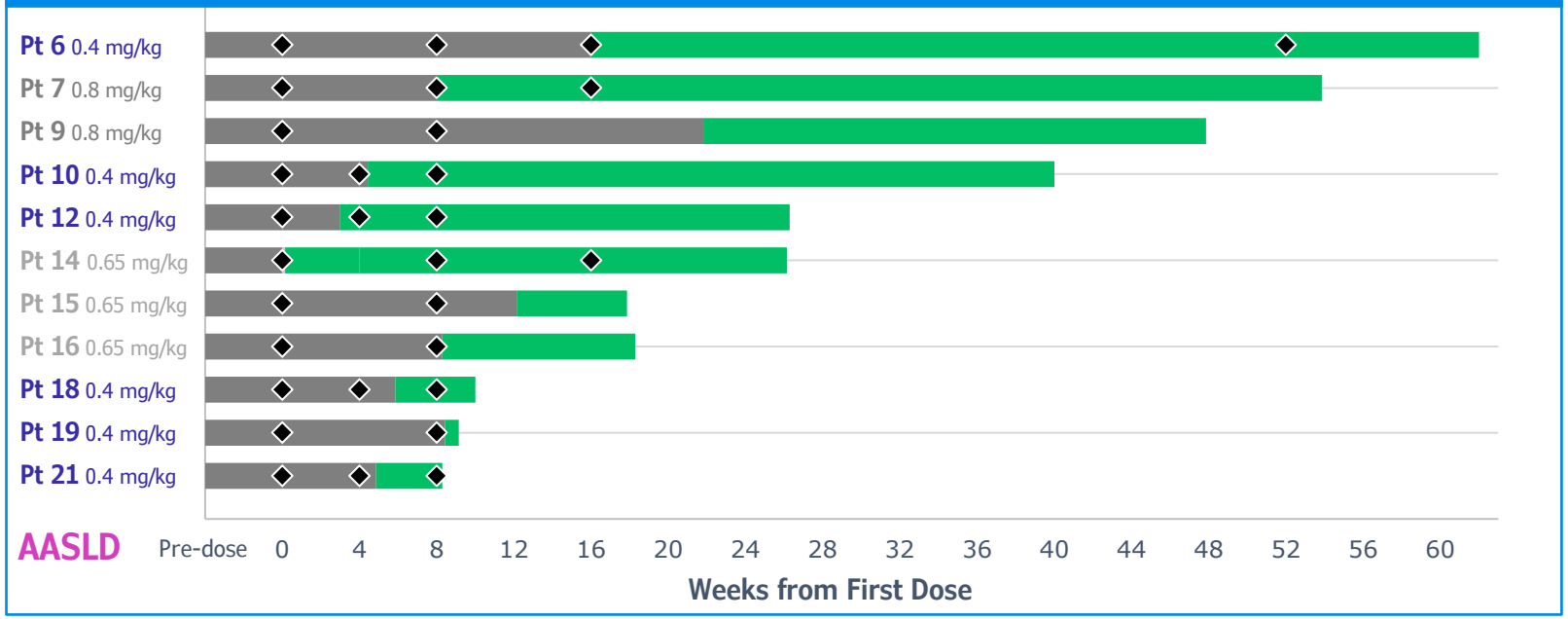
cccDNA, covalently closed circular DNA; EASL, European Association for the Study of the Liver; pgRNA, pregenomic RNA.

Late-Breaking Oral Presentation at AASLD Demonstrating cccDNA Elimination at 0.4 mg/kg

Reproducible Liver Biopsies with ≥1log cccDNA Elimination



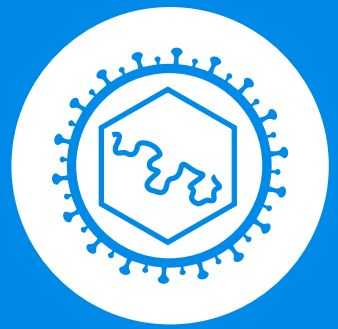
11 Patients with pgRNA Loss, Durability in 100% with ≥6 Months Evaluable



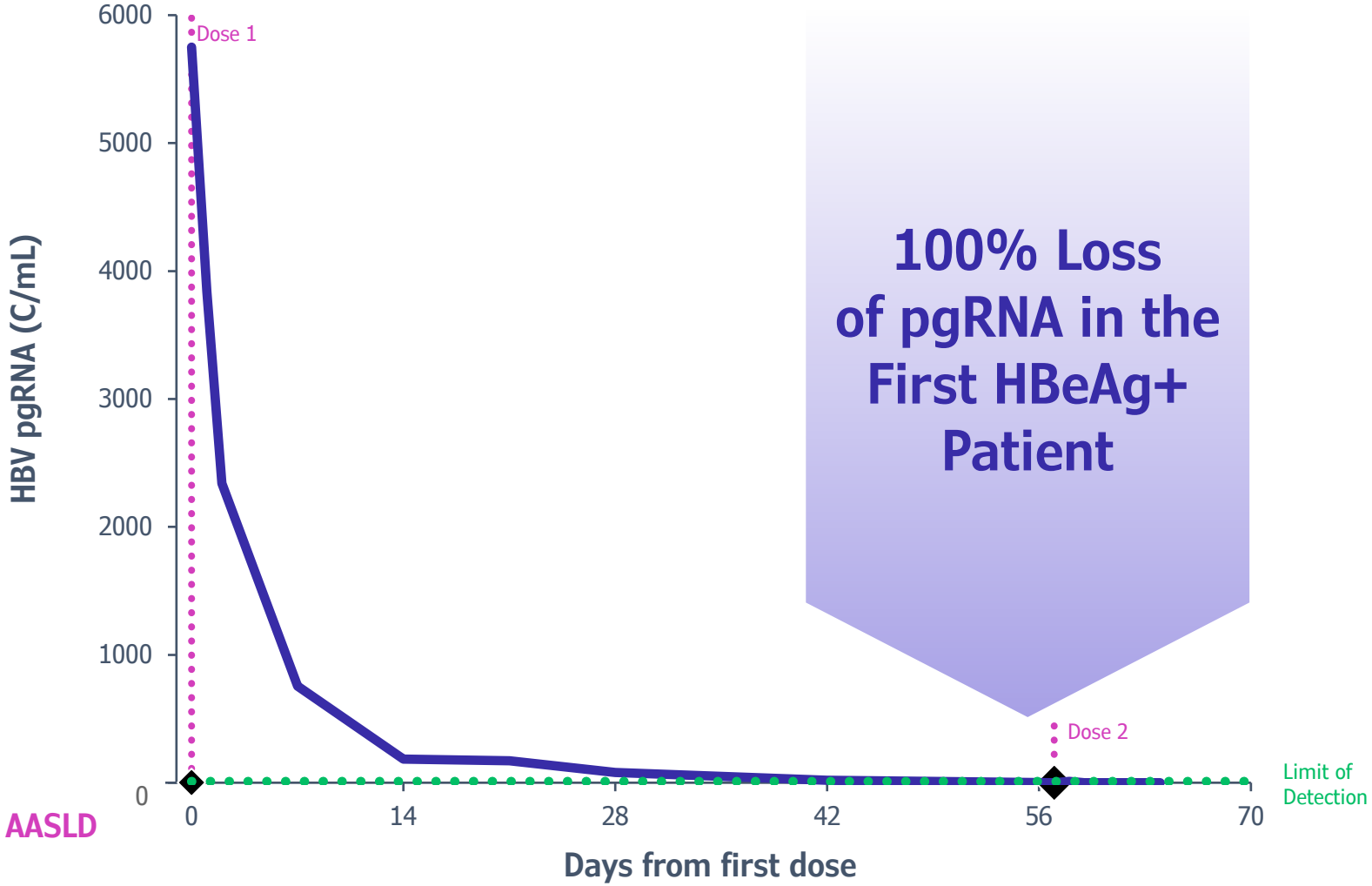
AASLD, American Association for the Study of Liver Diseases; cccDNA, covalently closed circular DNA; pgRNA, pregenomic RNA; Pt, patient.
Data cutoff: September 28, 2026.

Late-Breaking Oral Presentation at AASLD Demonstrating cccDNA Elimination at 0.4 mg/kg

Groundbreaking



100% pgRNA Loss in the First HBeAg+ Patient



AASLD, American Association for the Study of Liver Diseases; cccDNA, covalently closed circular DNA; HBeAg, hepatitis B e antigen; HBV, hepatitis B virus; pgRNA, pregenomic RNA.
Data cutoff: September 28, 2026.

Phase 1 Eliminate B Trial Success…

➡ PBGENE-HBV Moves to Phase 2

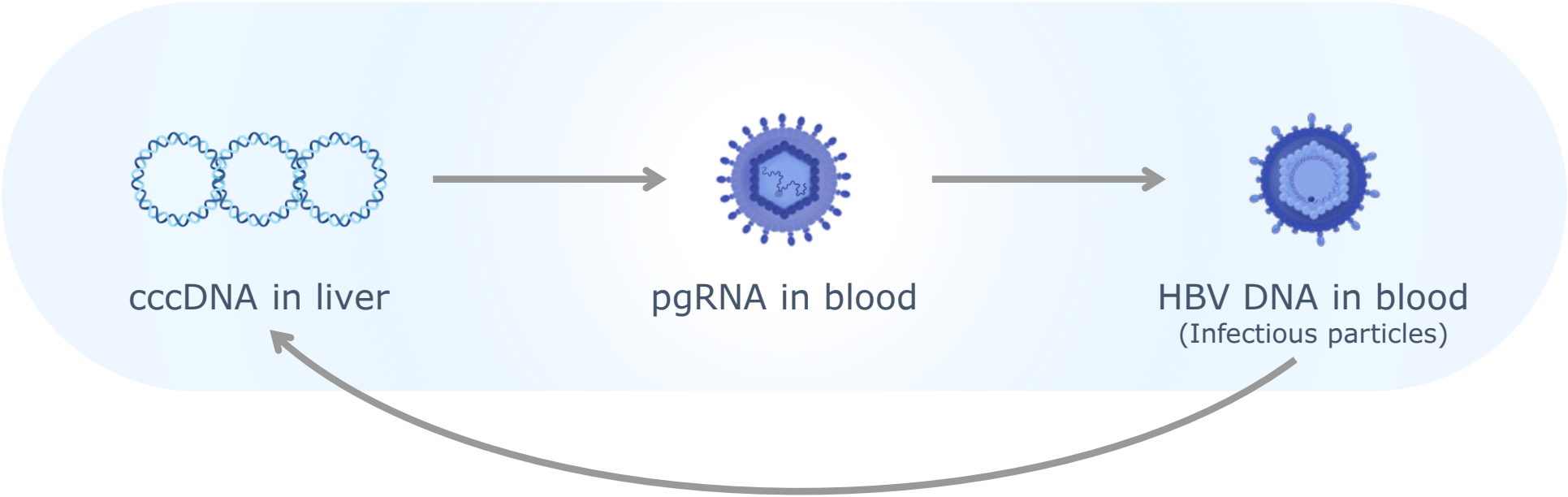
PBGENE-HBV: Eliminating cccDNA to Drive Viral Cure



Cassie Gorsuch, PhD
Chief Scientific Officer
Precision BioSciences, Inc.

cccDNA Is the Only Source of New Infectious Particles Known as HBV DNA

The Trinity of HBV Infection
cccDNA makes pgRNA makes HBV DNA

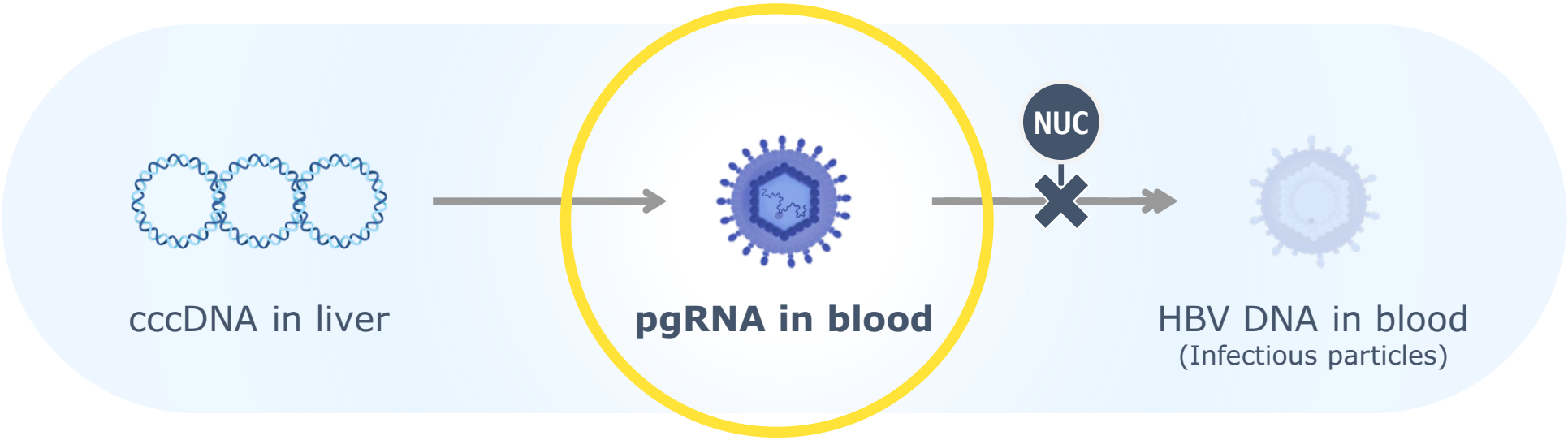


Therefore, cccDNA Elimination is the Only Path to Viral Cure

cccDNA, covalently closed circular DNA; HBV, hepatitis B virus; pgRNA, pregenomic RNA.

While on NUCs, Patients in ELIMINATE-B Have Undetectable HBV DNA

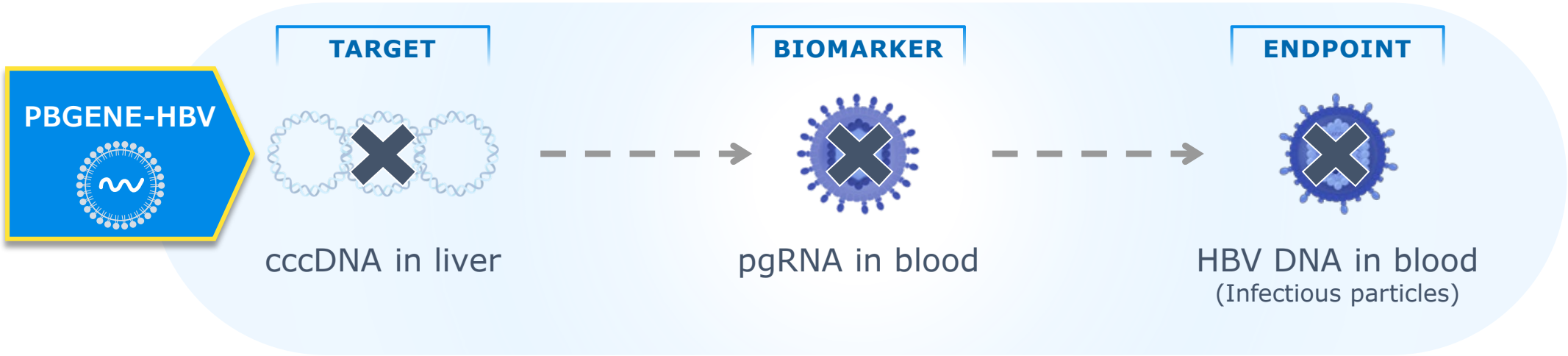
NUCs prevent conversion of pgRNA to HBV DNA



cccDNA is only present in the liver and requires biopsies
Therefore, pgRNA is the biomarker in the blood for HBV DNA

cccDNA, covalently closed circular DNA; HBV, hepatitis B virus; NUC, nucleos(t)ide analog; pgRNA, pregenomic RNA.

From Decades to Days: PBGENE-HBV Eliminates cccDNA, Potentially Changing the Natural History of Chronic Hepatitis B Enabling a Viral Cure



By directly eliminating cccDNA, PBGENE-HBV is designed to eradicate the trinity of viral replication

cccDNA, covalently closed circular DNA; HBV, hepatitis B virus; pgRNA, pregenomic RNA.

View FDA Guidance

Phase 1 ELIMINATE-B Data Support Advancement to Phase 2

Patient Baseline Characteristics (n=25)

		Number	Percent
Sex	Male	20	80
Ethnicity/ Race	Caucasian	7	28
	Asian	16	64
	Native Hawaiian or Other Pacific Islander	2	8

	Mean	Range
Age (years)	48	34-64
Time on NUCs (years)	11.8	1.2-29.1
Baseline pgRNA (C/mL)	257	TND-5,750
Baseline HBsAg (IU/mL)	4203	178-34,489

C/mL, copies per milliliter; HBeAg, hepatitis B e antigen; HBsAg, hepatitis B surface antigen; IU/mL, international units per milliliter; NUC, nucleos(t)ide analog; pgRNA, pregenomic RNA; TND, target not detected.
Screening HBsAg value used for Patient 25, awaiting value at baseline.

Comprehensive Evaluation of PBGENE-HBV: 59 Administrations in 25 Patients Across 4 Dose Levels

0.2 mg/kg	0.4 mg/kg	0.65 mg/kg	0.8 mg/kg
			

HBV, hepatitis B virus; Pt; patient.
Data cutoff: September 28, 2026.

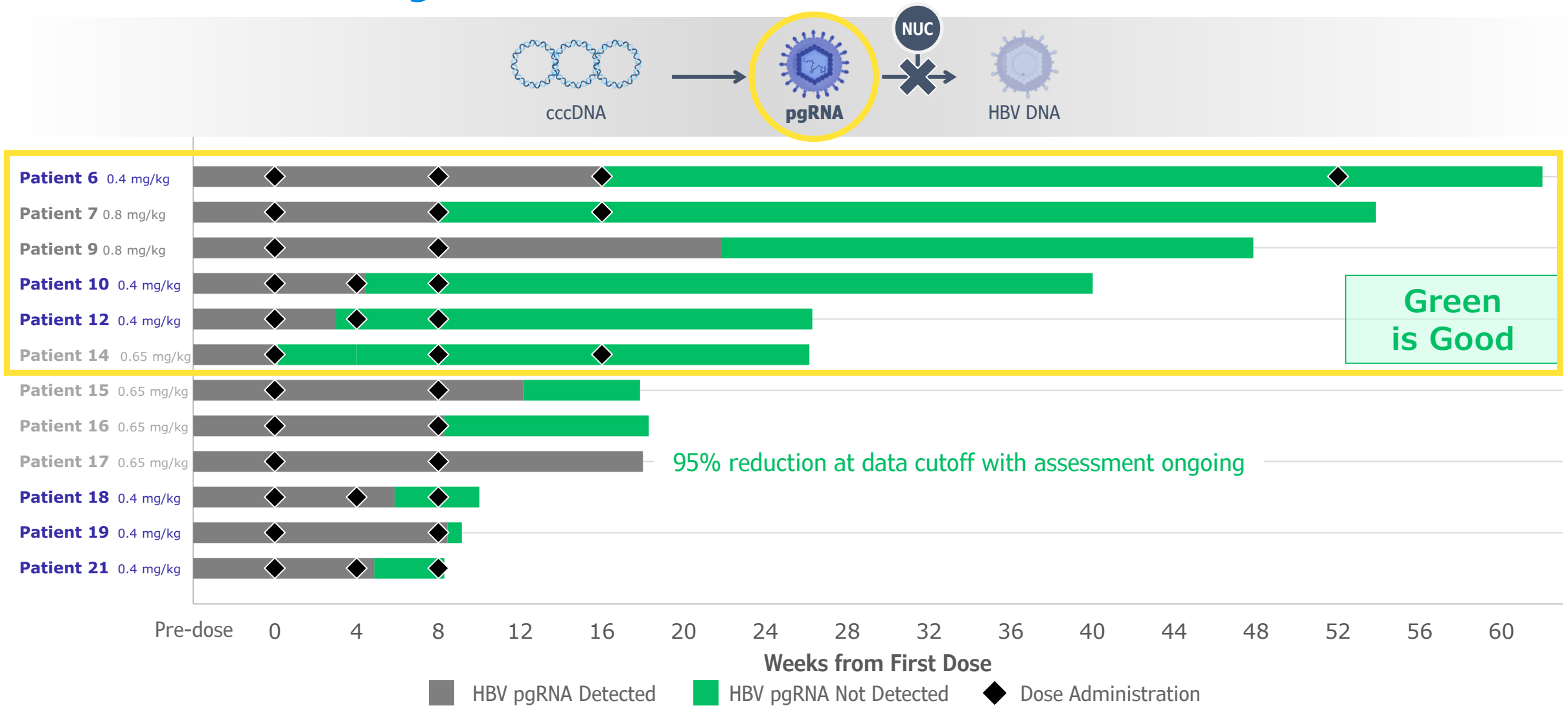
Extensive Experience with the Go-Forward Dose 0.4 mg/kg PBGENE-HBV



Achieved Target Product Profile for PBGENE-HBV as Low as 0.4 mg/kg

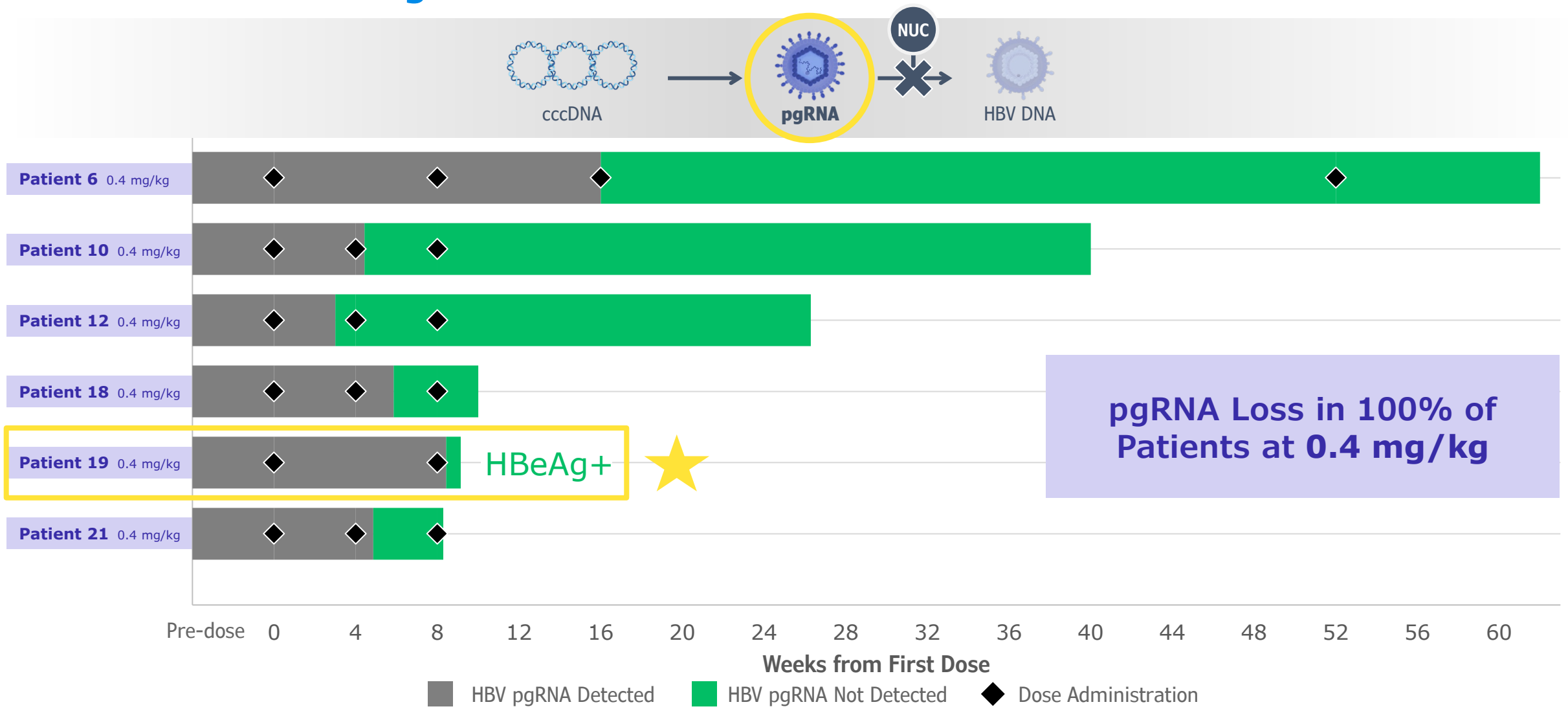
HBV, hepatitis B virus; Pt; patient.
Data cutoff: September 28, 2026.

NEW AASLD DATA: PBGENE-HBV Drives Ongoing Durable pgRNA Undetectable Through cccDNA Elimination



Roche cobas® HBV RNA test limit of detection 3.3-5 copies/mL, limit of quantitation 10 copies/mL, https://pubmed.ncbi.nlm.nih.gov/37879886/
Three additional patients are detectable for pgRNA at baseline, but not yet evaluable for pgRNA. Evaluable patients defined as those who have 28 days of data from initial dose.
AASLD, American Association for the Study of Liver Diseases; cccDNA, covalently closed circular DNA; HBeAg, hepatitis B e antigen; HBV, hepatitis B virus; NUC, nucleos(t)ide analog; pgRNA, pregenomic RNA.
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Data cutoff: September 28, 2026.

HBeAg Positive Patients:
The Most Difficult to Treat with Greatest cccDNA Burden

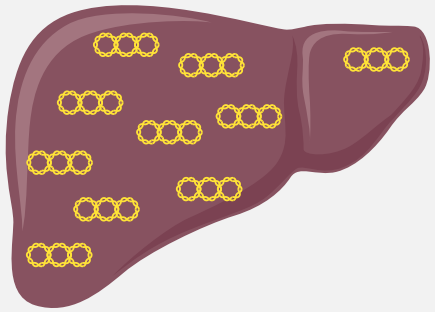
The median cccDNA levels were 2-3log higher in HBeAg+ patients compared to HBeAg- patients.

— Werle-Lapostolle et al, 2004, *Gastroenterology*

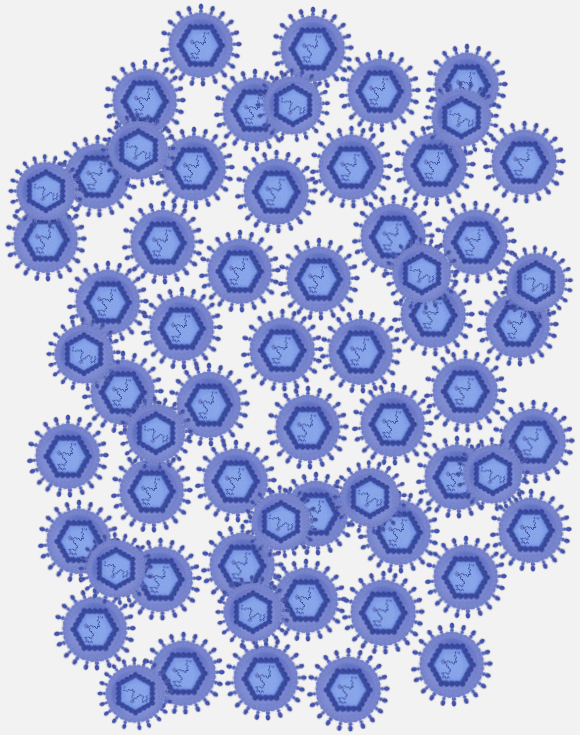
A Viral Cure is Hardest to Achieve in HBeAg Positive Patients Because They Have Logarithmically More cccDNA and Therefore More pgRNA

HBeAg Positive

cccDNA in liver

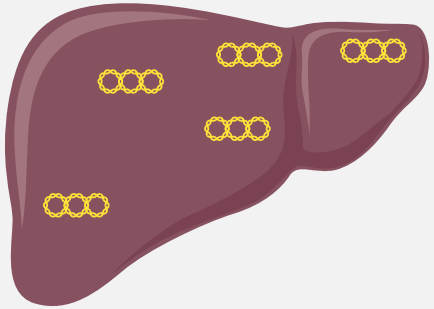


pgRNA in blood

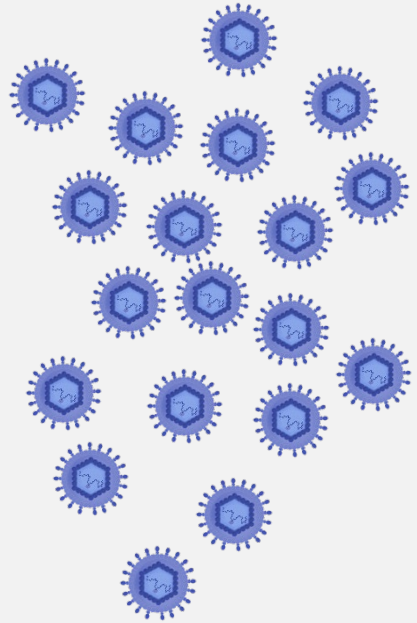


HBeAg Negative

cccDNA in liver

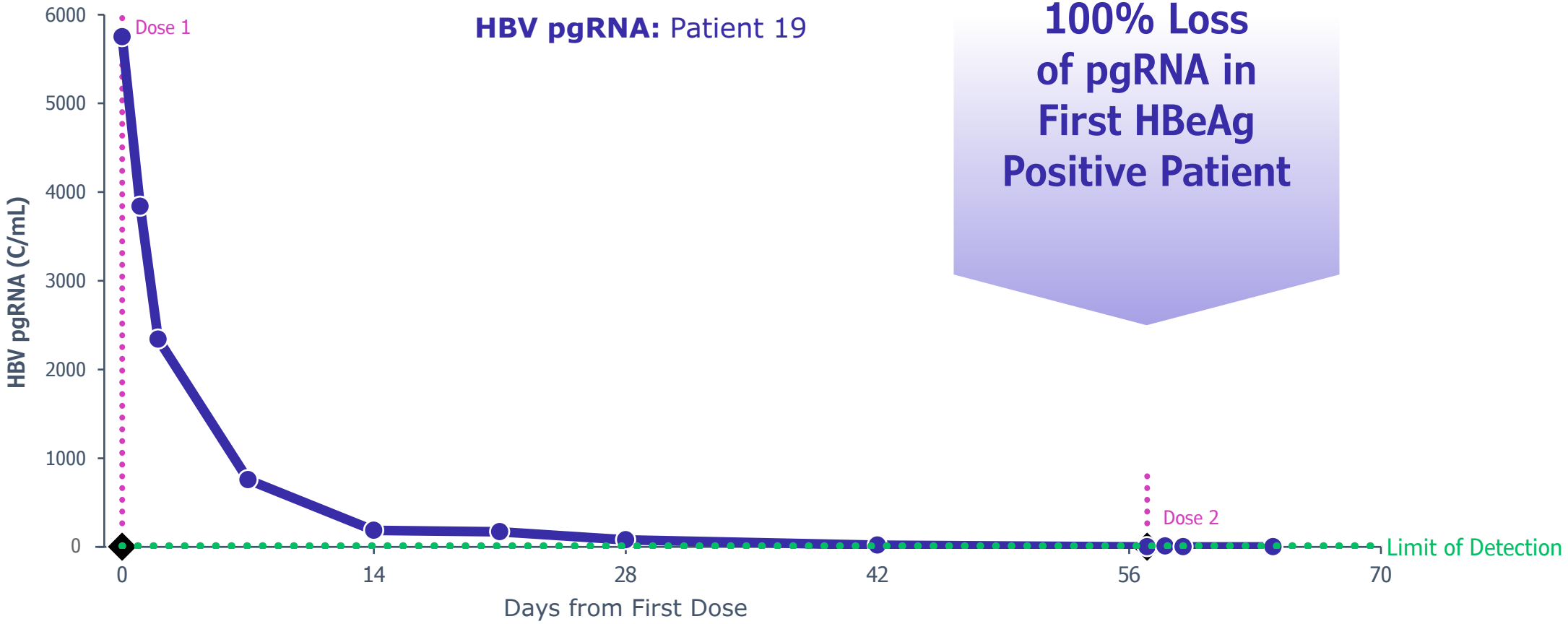


pgRNA in blood



cccDNA, covalently closed circular DNA; HBeAg, hepatitis B e antigen; pgRNA, pregenomic RNA.

NEW AASLD DATA: Eliminating cccDNA at 0.4 mg/kg Leads to 100% Loss of pgRNA in First HBeAg+ Patient



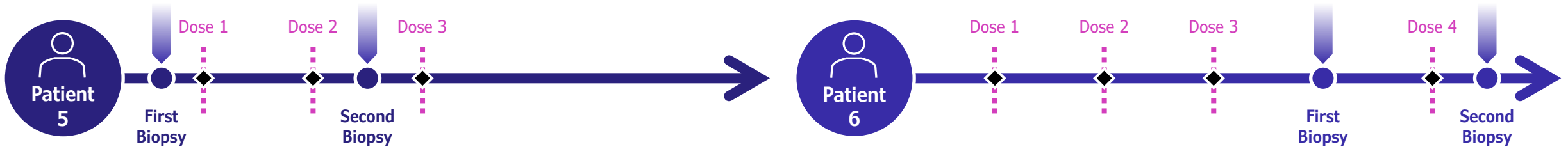
pgRNA loss in first HBeAg positive patient with 3.7-log baseline pgRNA demonstrates PBGENE-HBV potently eliminated cccDNA in the highest viral burden population

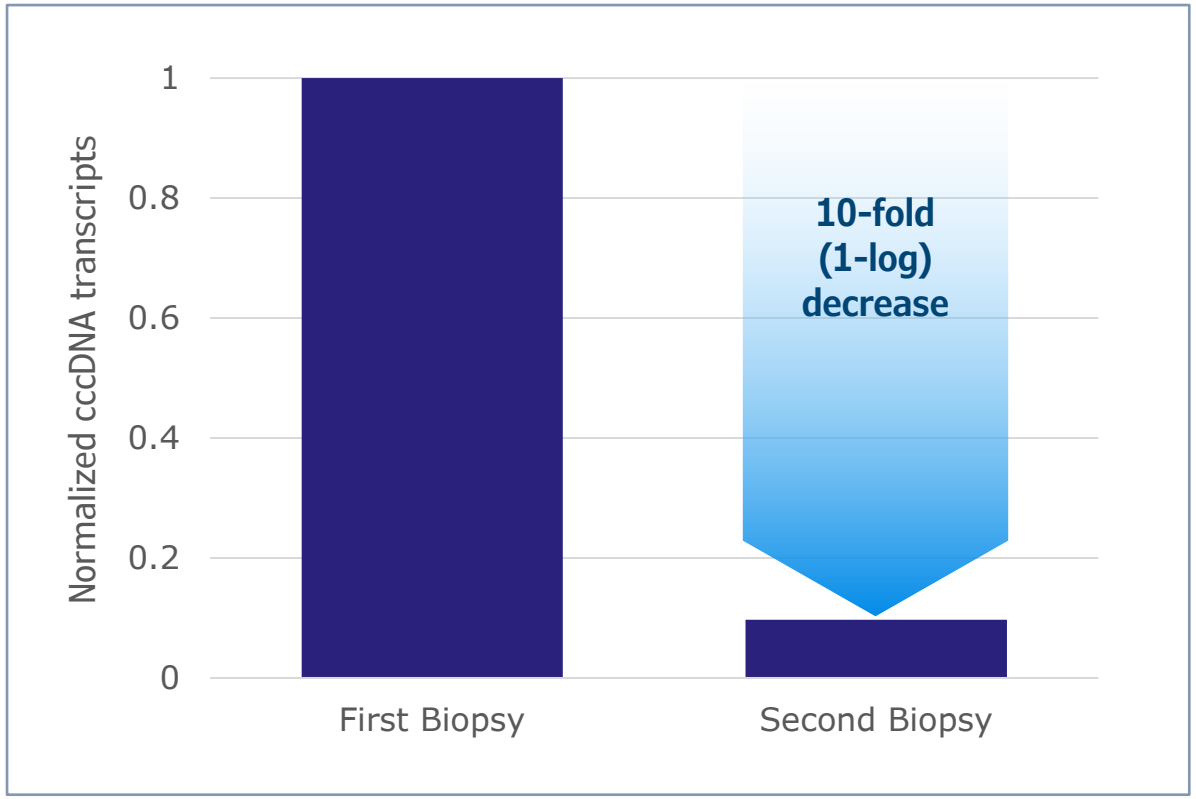
AASLD, American Association for the Study of Liver Diseases; cccDNA, covalently closed circular DNA; HBeAg, hepatitis B e antigen; HBV, hepatitis B virus; pgRNA, pregenomic RNA.
Data cutoff: September 28, 2026.

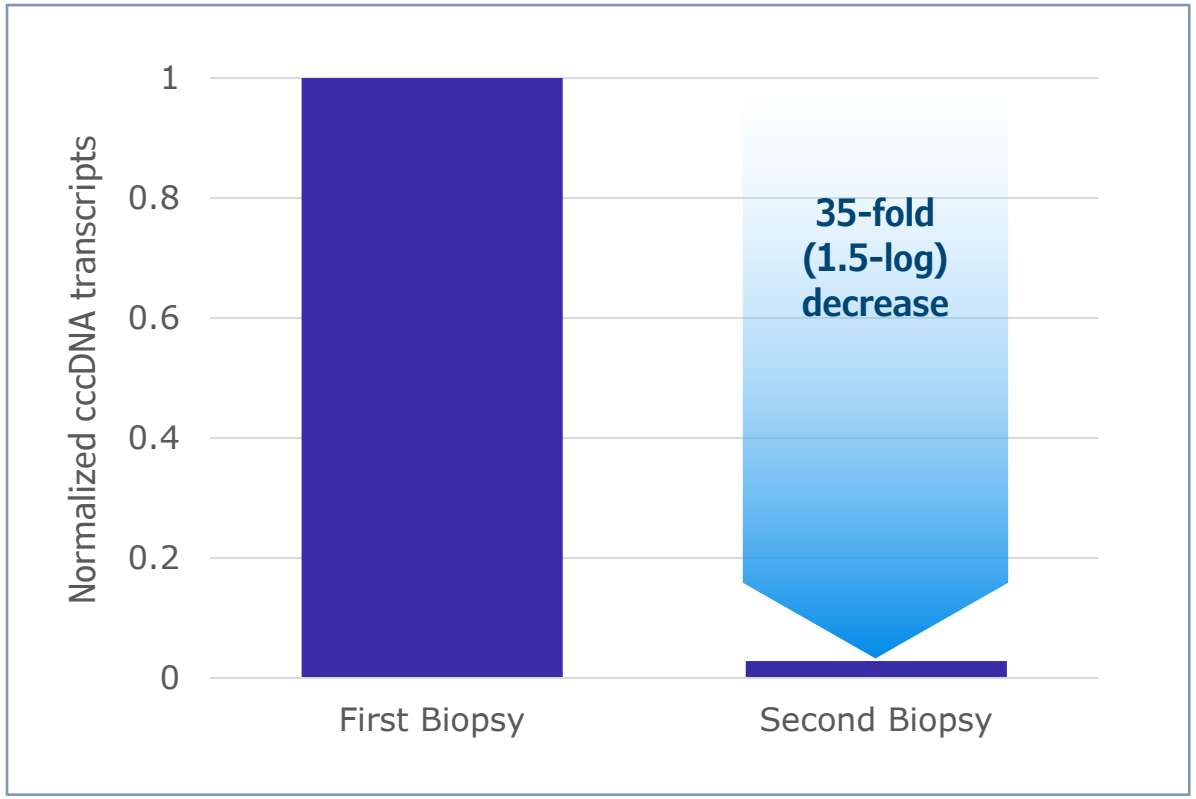
Liver Biopsy Evidence

Supports cccDNA Elimination at 0.4 mg/kg

NEW AASLD DATA: Biopsy Data Reproduced Potent cccDNA Elimination at 0.4 mg/kg







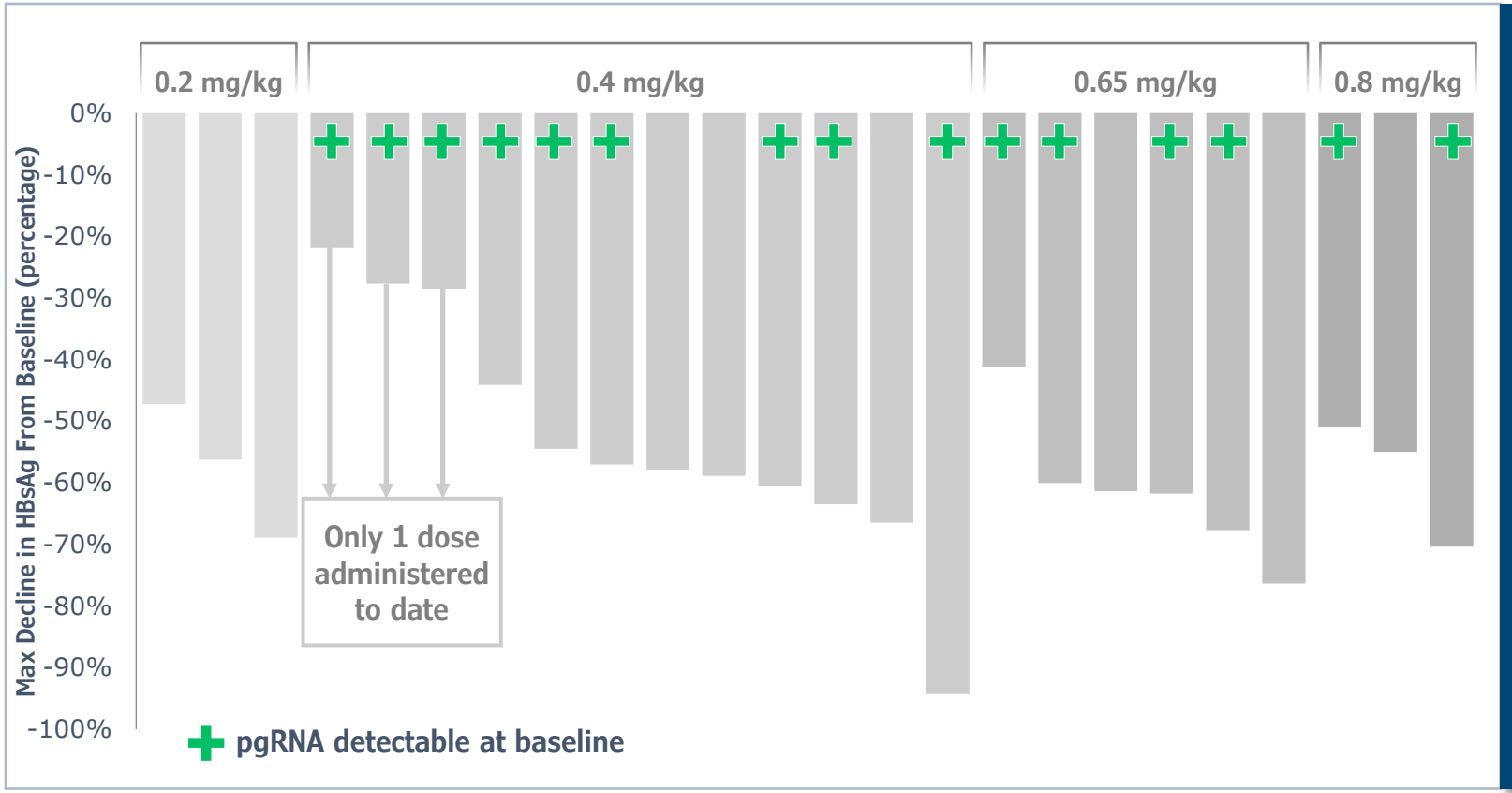
AASLD, American Association for the Study of Liver Diseases; cccDNA, covalently closed circular DNA.
Data cutoff: September 28, 2026.

Literature

Secondary Biomarker HBsAg: *Every ELIMINATE-B Patient Experienced Treatment Effect with PBGENE-HBV*

(n=24 evaluable at time of data cutoff)

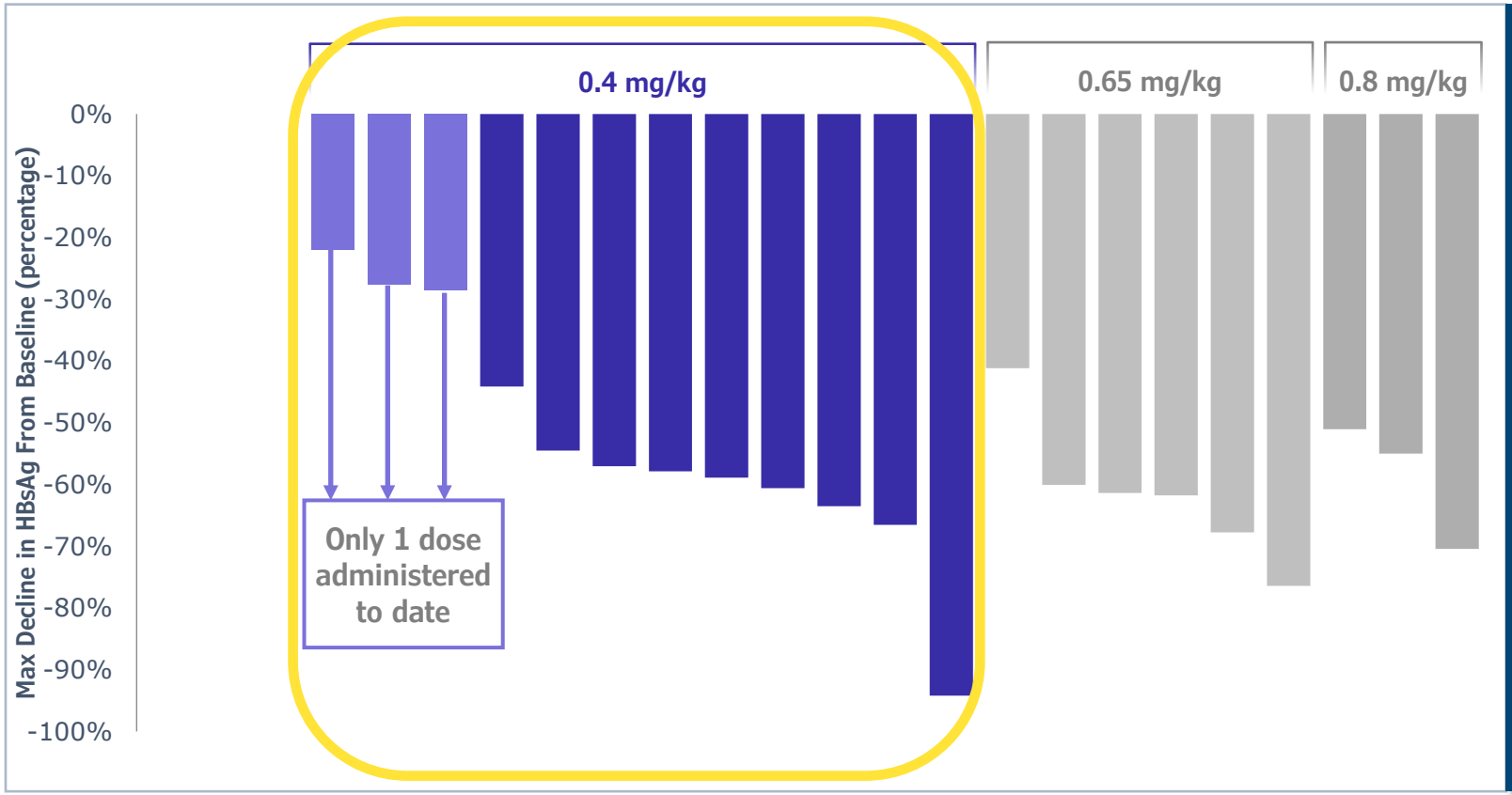
NEW AASLD DATA: Substantial HBsAg Declines Indicative of cccDNA Elimination in All Patients With or Without pgRNA



Significant maximal HBsAg declines across all patients (n=24)

The 3 patients with only 1 dose administered to date are detectable for pgRNA at baseline, but not yet evaluable for pgRNA.
Patient 25 is not yet evaluable for efficacy.
AASLD, American Association for the Study of Liver Diseases; cccDNA, covalently closed circular DNA; HBsAg, hepatitis B surface antigen; pgRNA, pregenomic RNA.
Data cutoff: September 28, 2026.

NEW AASLD DATA: 0.4 mg/kg is Sufficient for Maximal Efficacy & Durability

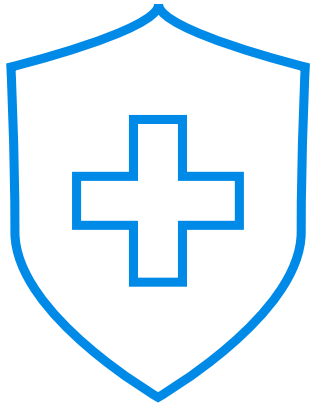


Maximal HBsAg declines observed as low as 0.4 mg/kg

Durability

Patient 25 is not yet evaluable for efficacy.
AASLD, American Association for the Study of Liver Diseases; HBsAg, hepatitis B surface antigen.
Data cutoff: September 28, 2026.

PBGENE-HBV Safety Profile Executive Summary



- 59 doses administered across 25 evaluable patients
- Primary Endpoint: No dose-limiting toxicities observed
- Most common adverse events are infusion-related reactions consistent with LNP effects, with onset and resolution within 24 hours of infusion
- Consistent with LNP effects, transient, reversible ALT/AST lab abnormalities have been observed. All were asymptomatic with no elevated bilirubin, and no cases of Hy's Law
- No Grade ≥3 hypotension has been observed at the go-forward dose of 0.4 mg/kg since implementing modified infusion protocol (n=15 administrations in 8 patients)
- Safety at 0.4 and 0.65 mg/kg is the focus of today's presentation
- Reminder from May 2026: Repeat doses at 0.8 mg/kg without current infusion protocol were not continued[1]

Favorable therapeutic index for PBGENE-HBV at 0.4 mg/kg supports further evaluation in Phase 2

ALT, alanine aminotransferase; AST, aspartate aminotransferase; HBV, hepatitis B virus; LNP, lipid nanoparticle.
Data cutoff: September 28, 2026.
1. All data from 0.8 mg/kg dose level presented in May 2026 Investor Presentation. No additional doses or safety events at 0.8 mg/kg. https://investor.precisionbiosciences.com/events/event-details/precision-biosciences-host-investor-webcast-discuss-late-breaking-pbgene-hbv

PBGENE-HBV Safety Profile: Clear Evidence of Therapeutic Window at 0.4 mg/kg

Number of patients with AEs[a]:		0.65 mg/kg n=6	0.4 mg/kg n=13	0.4 mg/kg w/ current infusion protocol n=8 of 13
Grade 3	Hypotension	4	3[b]	-
Grade 3 and 4	ALT/AST lab abnormality	4	2	1
SAEs		-	-	-

- **Current Infusion Protocol:** No Grade ≥3 hypotension has been observed at 0.4 mg/kg since implementing a simple modified infusion protocol to blunt LNP-related inflammatory response (5-hour infusion, 20 mg dexamethasone, prophylactic saline infusion)
- **Current Infusion Protocol:** One delayed ALT/AST lab abnormality was observed after third dose, with peak ALT at 306 U/L and AST at 278 U/L. No intervention was required, no changes in bilirubin were observed, and no cases of Hy's Law
- Repeat administrations at 0.65 mg/kg resulted in a higher number of patients with transient Grade 3 hypotension and ALT/AST lab abnormalities. No changes in bilirubin and no Hy's Law. These lab abnormalities were transient; 3/4 patients fully resolved on their own, 1 patient received steroid intervention and has resolved

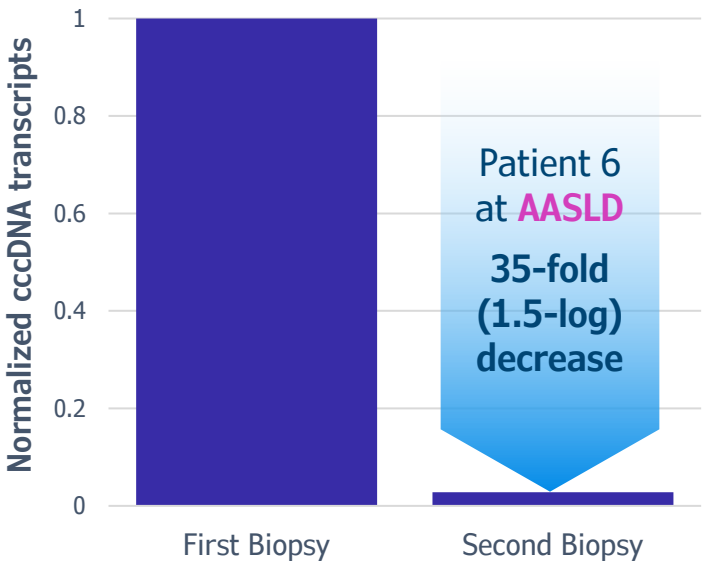
Favorable safety profile for PBGENE-HBV at 0.4 mg/kg
Aligned with DMC and Investigators

CTCAE

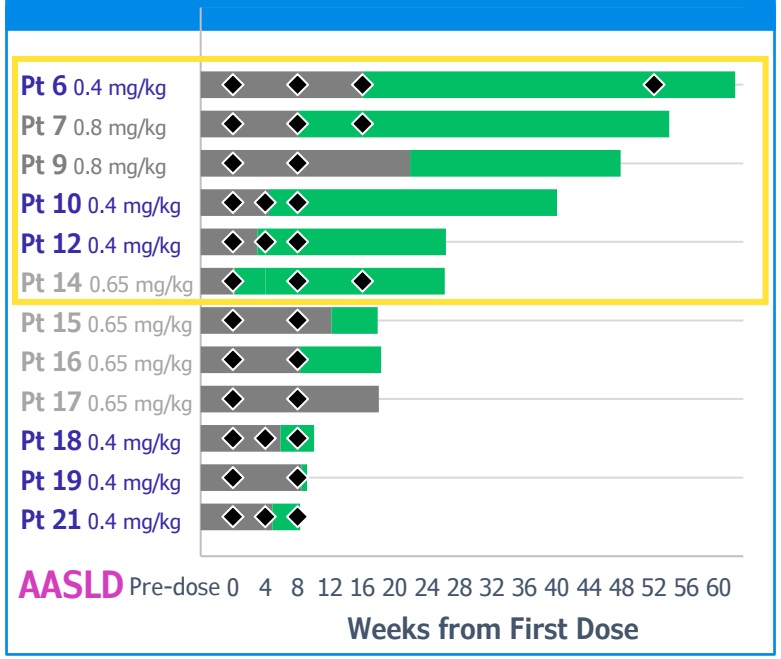
AE, adverse event; ALT, alanine aminotransferase; AST, aspartate aminotransferase; DMC, data monitoring committee; HBV, hepatitis B virus; LNP, lipid nanoparticle; SAE, serious adverse event.
Data cutoff: September 28, 2026.
[a]AEs shown are treatment-related and possibly treatment-related. [b]Grade 3 hypotension was observed at 0.4 mg/kg prior to current infusion protocol.
1. Safety profile at 0.2 and 0.8 mg/kg were previously presented in May 2026, https://investor.precisionbiosciences.com/events/event-details/precision-biosciences-host-investor-webcast-discuss-late-breaking-pbgene-hbv
2. Two SAEs were observed in the same patient dosed at 0.8 mg/kg. A grade 2 myocardial ischemia, mechanistically linked to acute hypotension, was considered treatment-related and resolved with no heart damage. An intracerebral hemorrhage occurred on day 30 post second dose and is considered possibly related.

NEW AASLD DATA: Definitive Phase 1 Efficacy Data Support 0.4 mg/kg as Go-Forward Dose

Reproducible Liver Biopsies with ≥1-log cccDNA Elimination

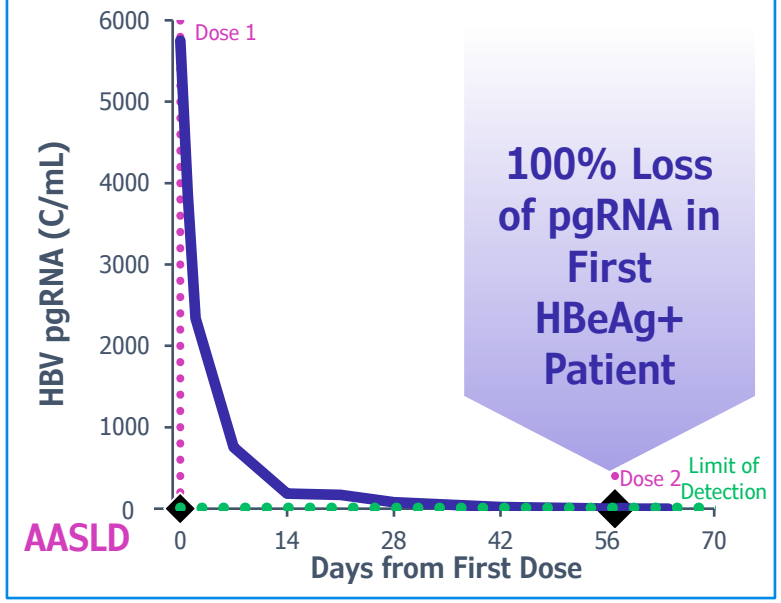


Durable pgRNA Loss ≥6 Months



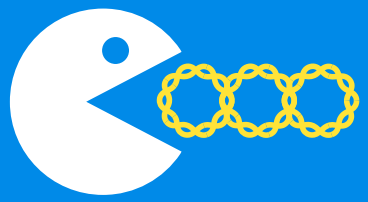
Groundbreaking

100% pgRNA Loss in First HBeAg+ Patient



AASLD, American Association for the Study of Liver Diseases; cccDNA, covalently closed circular DNA; HBeAg, hepatitis B e antigen; HBV, hepatitis B virus; pgRNA, pregenomic RNA; Pt, patient.
Data cutoff: September 28, 2026.

Go-Forward Phase 2 Dose of 0.4 mg/kg for PBGENE-HBV: Selected Based on Compelling Evidence



Efficacy:

- 100% of patients experienced a treatment effect with PBGENE-HBV (n=24)
- 92% (11/12) of patients achieved pgRNA loss to undetectable
  - Last remaining patient had 95% pgRNA reduction at data cutoff with assessment ongoing
- 100% of patients with ≥6 months follow-up have maintained durable pgRNA loss to undetectable
  - Maximal durability now up to ~1 year demonstrates permanence of cccDNA elimination
  - 100% of patients at 0.4 mg/kg have pgRNA loss to undetectable and remain undetectable
- 100% loss of pgRNA in hardest to treat patient, HBeAg+ patient
  - From 5,750 (3.7log) copies/mL at baseline to undetectable after 2 doses of 0.4 mg/kg
- Biopsies in 2 patients at 0.4 mg/kg show 1-log and 1.5-log reduction in cccDNA



Safety:

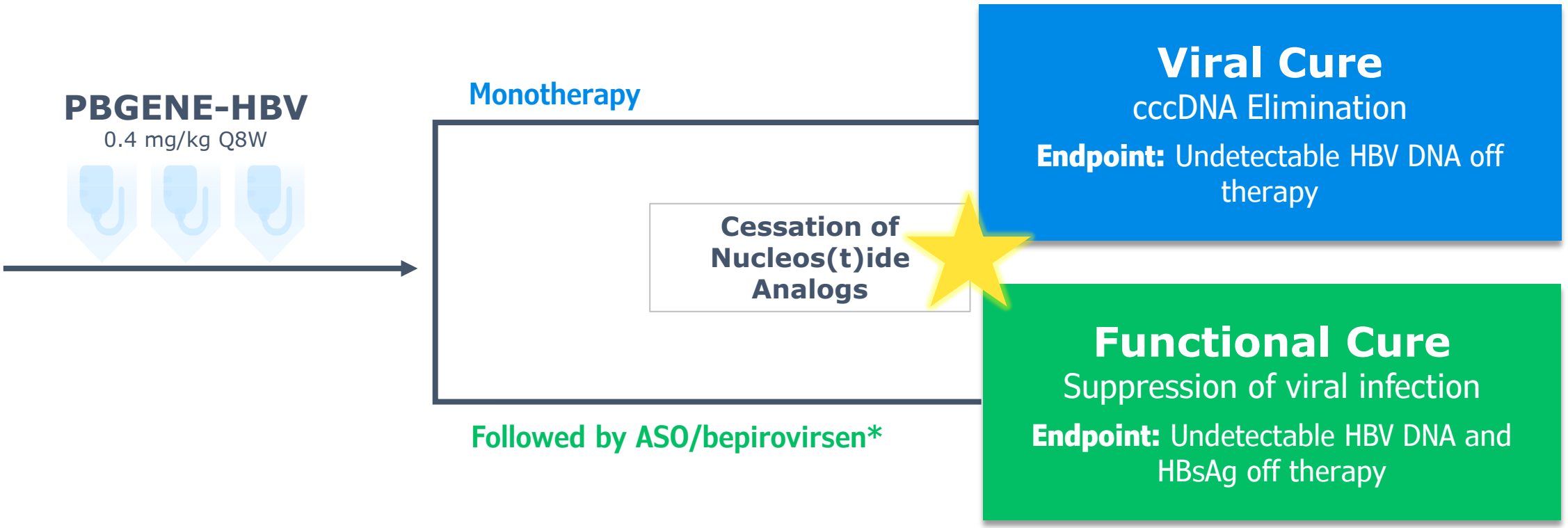
- At 0.4 mg/kg with current infusion protocol, PBGENE-HBV has a manageable safety profile with no SAEs, with all patients completing planned doses
- PBGENE-HBV has a well characterized and predictable safety profile; 59 dose administrations across 25 patients delivered globally in the ELIMINATE-B trial
- No Grade ≥3 hypotension at 0.4 mg/kg with current infusion protocol
- In the Global Phase 1 ELIMINATE-B trial, there were no cases of elevated bilirubin, Hy's Law, or evidence of synthetic liver dysfunction at any dose level

cccDNA, covalently closed circular DNA; HBeAg, hepatitis B e antigen; HBV, hepatitis B virus; pgRNA, pregenomic RNA; SAE, serious adverse event.

What's Next:

PBGENE-HBV Moves to Phase 2 Global Expansion

Establishing PBGENE-HBV as the Foundation of Treatment in HBV: ELIMINATE-B Trial Moves to Phase 2 Expansion



*Japan MHLW Approval Date; August 24, 2026. US FDA Approval Date; October 20, 2026.
ASO, antisense oligonucleotide; cccDNA, covalently closed circular DNA; FDA, US Food and Drug Administration; HBsAg, hepatitis B surface antigen;
HBV, hepatitis B virus; MHLW, Ministry of Health, Labour and Welfare; Q8W, every 8 weeks.

Details

PBGENE-HBV Goal to Improve Upon Cure Rates in CHB

Monotherapy

Viral Cure

Complete cccDNA elimination in the liver

FDA Endpoint: Undetectable HBV DNA for 6 months (off therapy including NUCs)[1]

- Viral Cure rate today: 0%
- Goal for PBGENE-HBV: Enable first ever viral cure

+ Combination with ASO

Functional Cure

Suppression of viral infection

FDA Endpoint: Undetectable HBV DNA and HBsAg for 6 months (off therapy including NUCs)[1]

- Functional Cure rate today: 19% (bepirovirsen*)[2]
- Goal in the field: 30% functional cure[3]

*Japan MHLW Approval Date; August 24, 2026. US FDA Approval Date; October 20, 2026.
ASO, antisense oligonucleotide; cccDNA, covalently closed circular DNA; CHB, chronic hepatitis B; FDA, US Food and Drug Administration; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; MHLW, Ministry of Health, Labour and Welfare.

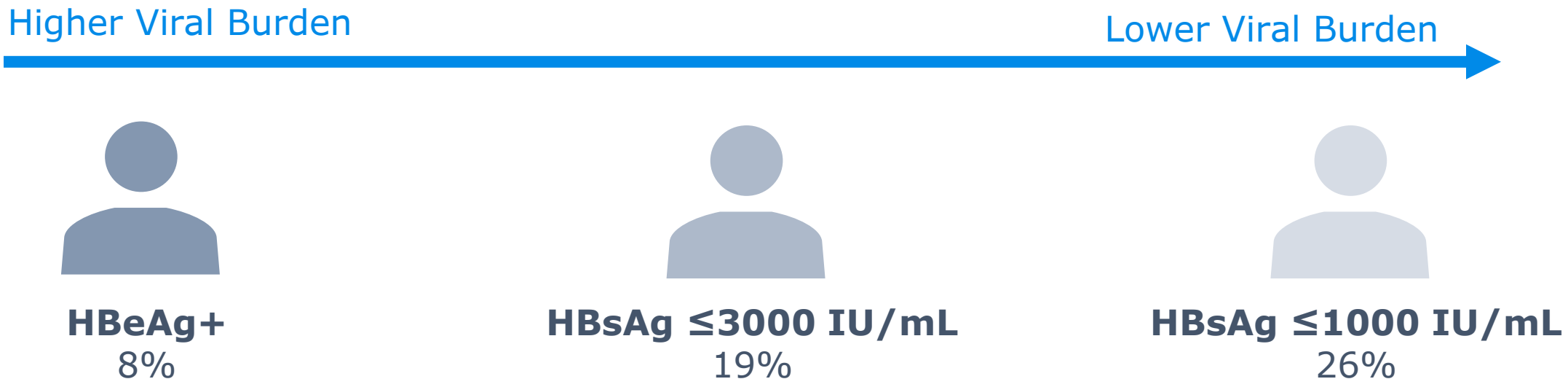
1. FDA Guidance – Chronic Hepatitis B Virus Infection; Developing Drugs for Treatment; April 2022. Accessed April 30, 2026. 2. Hou J, et al. *N Engl J Med.* 2026;394(24):2395-2406.
3. Ren S, et al. *Front Public Health*. 2026;14:1707447.

View FDA Guidance

Rationale for Potential Combination

Bepirovirsen Most Effective in Patients with Lower Viral Burden; 19% Functional Cure Rate in B-Well 1 and B-Well 2 Studies

Functional Cure Rates in B-Well 1 and B-Well 2 Phase 3 Studies

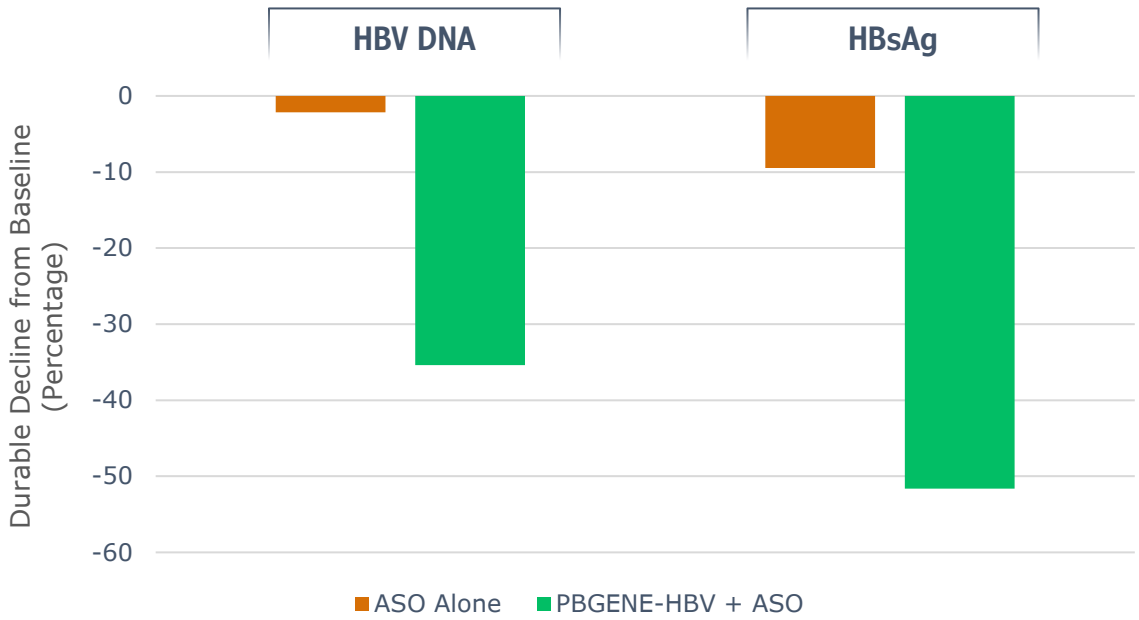


HBeAg, hepatitis B e antigen; HBsAg, hepatitis B surface antigen.

Hou J, et al. *N Engl J Med.* 2026;394(24):2395-2406.

Synergy of PBGENE-HBV + ASO in HBV Mouse Model Drove Deeper Reductions in HBV DNA and HBsAg

Improved Depth and Durability of HBV DNA and HBsAg Declines with PBGENE-HBV + ASO



HBV Transgenic Mouse Model Treated with ASO alone or PBGENE-HBV + ASO

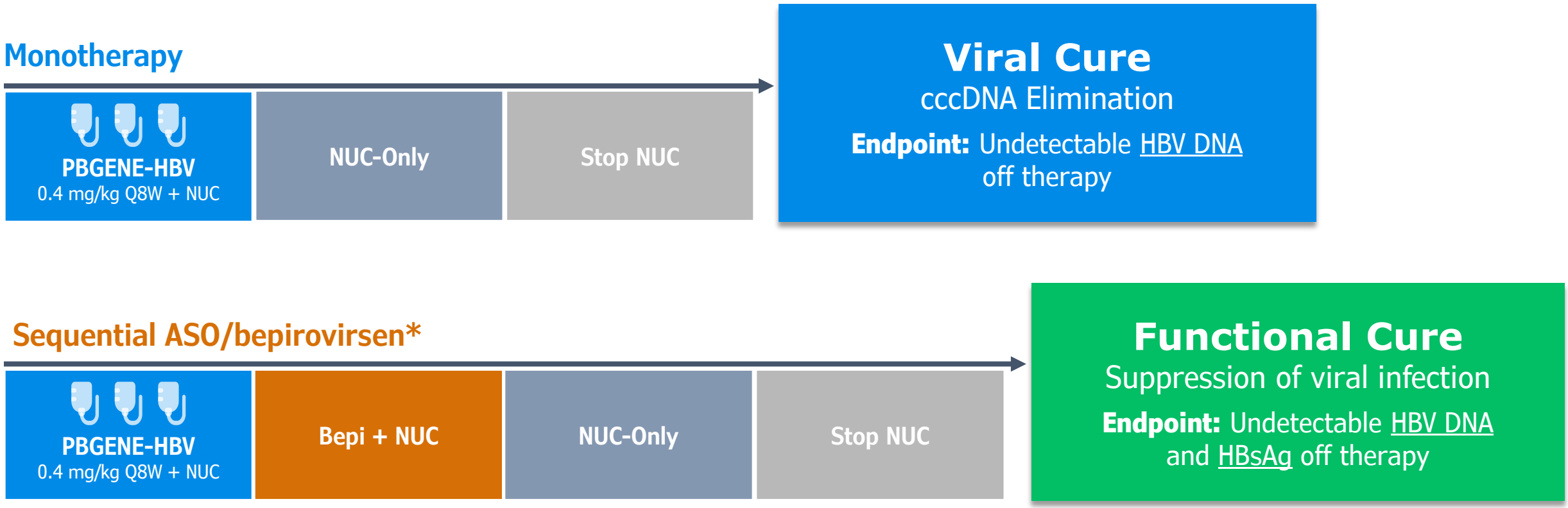
- Single dose of PBGENE-HBV
- Finite course of treatment with ASO (biosimilar to bepirovirsen)
- Off-treatment monitoring period with durability of the effect measured at the end of the study

PBGENE-HBV + ASO Combination Resulted in Substantially Deeper Reductions in HBV DNA and HBsAg Compared to ASO Alone

ASO, antisense oligonucleotide; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus.
Data on file.

Bepi MOA

Establishing PBGENE-HBV as the Foundation of Treatment in HBV: ELIMINATE-B Trial Moves to Phase 2 Expansion



*Japan MHLW Approval Date; August 24, 2026. US FDA Approval Date; October 20, 2026.
ASO, antisense oligonucleotide; cccDNA, covalently closed circular DNA; FDA, US Food and Drug Administration; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; MHLW, Ministry of Health, Labour and Welfare; Q8W, every 8 weeks.

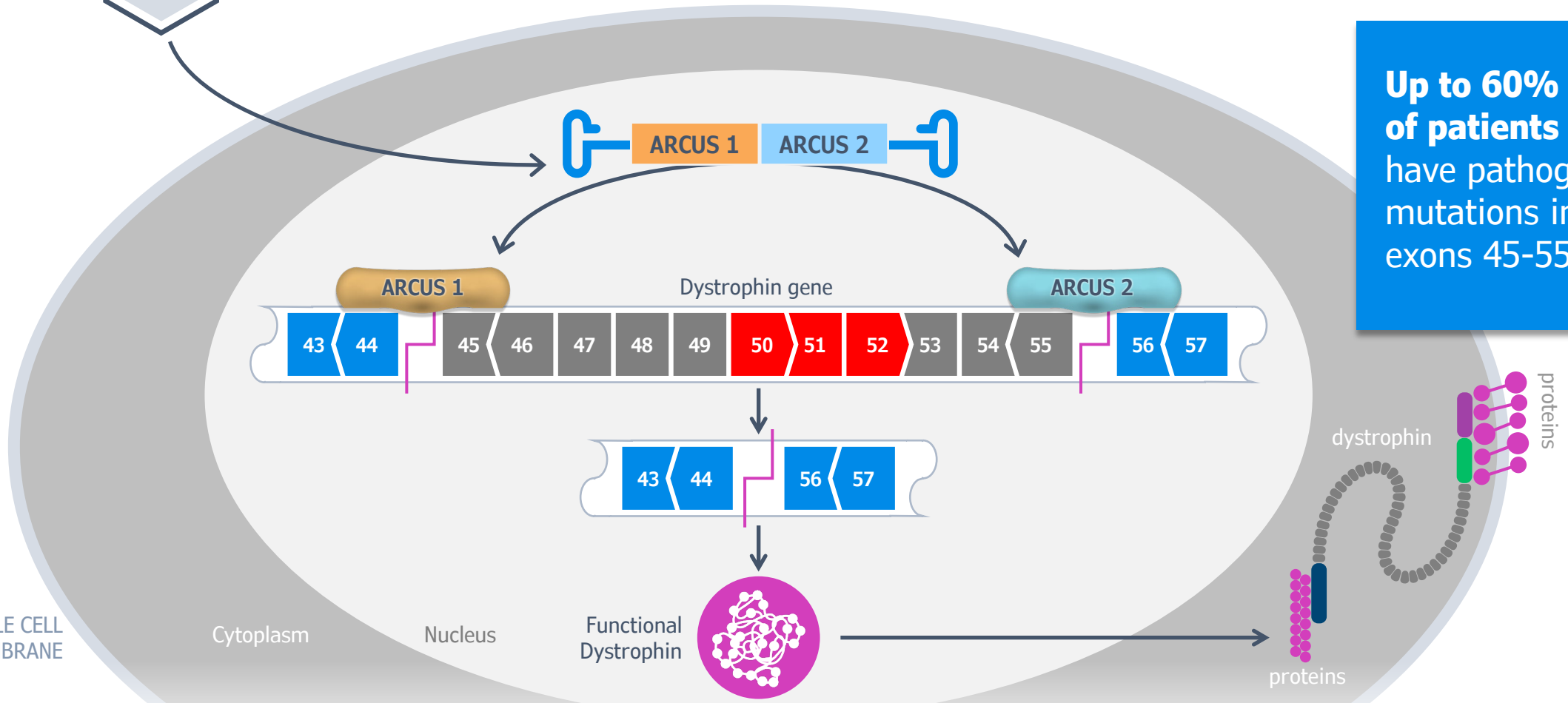
Schema

FUNCTION-DMD

Clinical Momentum Continues with PBGENE-DMD

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

PBGENE-DMD: A single AAV encodes 2 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; DMD, Duchenne muscular dystrophy.

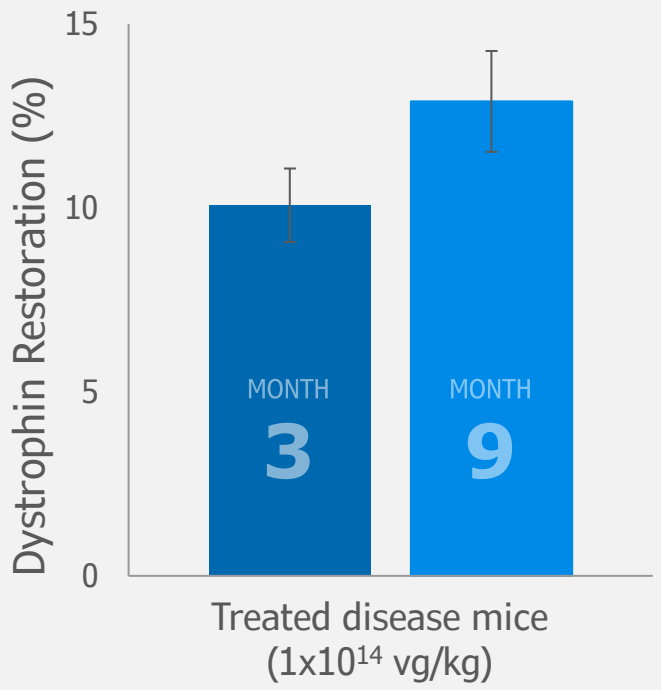
1. Béroud C, et al. *Hum Mutat*. 2007;28(2):196-202. .

Preclinical Data Supports Potential of PBGENE-DMD to Provide Therapeutic Benefit

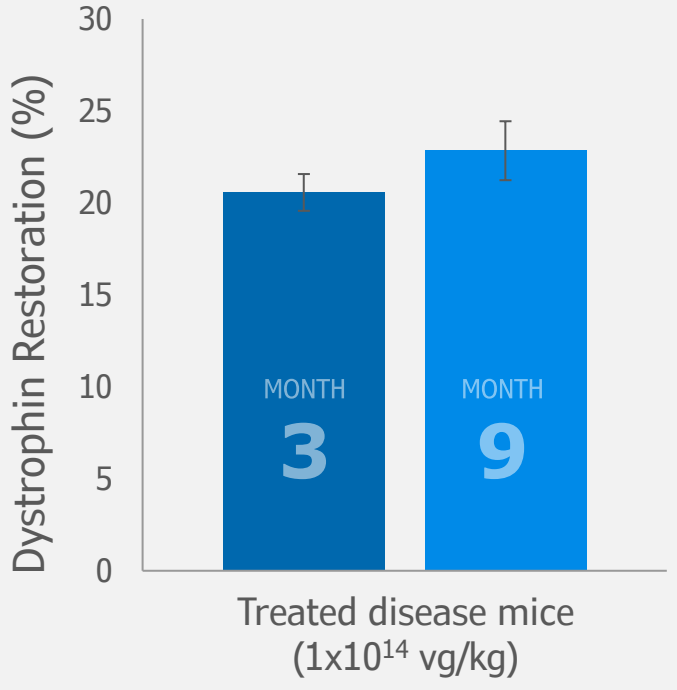
Based on natural history data, as little as 5% expression of functional dystrophin protein is expected to provide therapeutic benefit in patients with DMD

Preclinical Data from Humanized DMD Mouse Model Demonstrates >5% Dystrophin Protein Expression

Heart



Calf

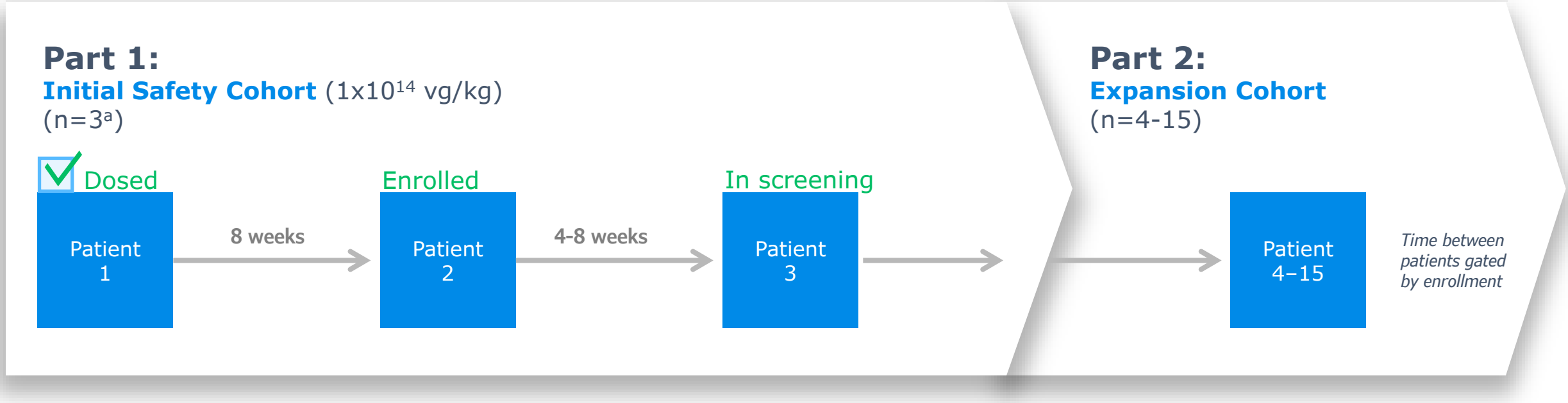


Phase 1/2 Study Designed for Safety and Speed

Clinical Trial Identifier: NCT07429240

Inclusion criteria

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



[a]Up to 3 additional participants may be enrolled in Part 1, if needed, to further assess dosing or safety based on DSMB recommendation.
DMD, Duchenne muscular dystrophy; DSMB, data safety monitoring board; IMR, immune modulation regimen; MOA; mechanism of action; 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 Continues To Advance In FUNCTION-DMD Study

1st patient successfully dosed August 2026

- No serious adverse events, no adverse events of special interest (including no TMA, myocarditis, or acute liver injury)

DSMB has reviewed patient 1 data through week 8

- No safety concerns identified
- Endorsed decision to dose patients 2 & 3 without amendment to the protocol or need for further DSMB review within part 1

On track to meet goal of 3 patients enrolled by end of year

- Participants pre-screened and undergoing baseline assessments

What's next:

- Part 1 (n=3) safety and 12-week biopsy readouts
- Continuation to Part 2 of the study (n=12, concurrent enrollment)

DMD, Duchenne muscular dystrophy; DSMB, data safety monitoring board; TMA, thrombotic microangiopathy.

Closing Thoughts



Michael Amoroso
President & Chief Executive Officer
Precision BioSciences, Inc.

Precision BioSciences Has Numerous Upcoming Catalysts in Next 12 Months

Key Expected Inflection Points Through 2027

		PBGENE-HBV LNP Program	PBGENE-DMD AAV Program
2026	Q4	✓ AASLD Late-Breaking Oral Data Update & Phase 2 Study Plan	✓ Safety Update from Initial Patients
2027	H1	〉 Initiate ELIMINATE-B2 Global Ph2 Study in Q1 〉 Rapid Enrollment Across Multiple Continents	〉 3-month Dystrophin from Initial Patients 〉 Expansion into Part 2
	H2	〉 Continued Phase 2 Study Execution 〉 Generate Open Label Phase 2 Viral Cure Data 〉 Generate Open Label Phase 2 Combination Data Compared to Bepirovirsen Alone (19% functional cure)	〉 12-month Dystrophin from Initial Patients 〉 Functional Data from Initial Patients

AASLD, American Association for the Study of Liver Diseases; AAV, adeno-associated virus; DMD, Duchenne muscular dystrophy; H1, first half; H2, second half; HBV, hepatitis B virus; LNP, lipid nanoparticle; Ph2, phase 2; Q1, first quarter; Q4, fourth quarter.

Expected Cash Runway into Q4 2029:
Funds Both Clinical Programs Through Key Next Stage Inflection Points

PBGENE-HBV

- Funds PBGENE-HBV through global Phase 2 study (ELIMINATE-B2)
- Open Label Data results:
  - PBGENE-HBV monotherapy viral cure
  - PBGENE-HBV with bepirovirsen (ASO) for functional cure

PBGENE-DMD

- Funds PBGENE-DMD through pivotal trial clinical execution
- Data results:
  - Phase 1/2 complete data set in N=10 patients
  - Pivotal data generation beginning in 2028

Recent $125M catalyst-driven financing adds capital to Precision's previously reported cash balance of $108M (9/30/26) **and extends expected runway into Q4 2029**[1]

[1]$108M cash, cash equivalents, and restricted cash as of 9/30/26. 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 into Q4 2029.

Q&A



MF Yuen, MD, PhD

Chair Professor
Division of Gastroenterology & Hepatology
Li Shu Fan Medical Foundation Professor in Medicine,
The University of Hong Kong, Hong Kong



Mark Sulkowski, MD

Professor of Medicine
Director, Division of Infectious Diseases Johns Hopkins Bayview Medical Center
Johns Hopkins University & Medicine



Cassie Gorsuch, PhD

Chief Scientific Officer
Precision BioSciences, Inc.



Michael Amoroso

President & Chief Executive Officer
Precision BioSciences, Inc.

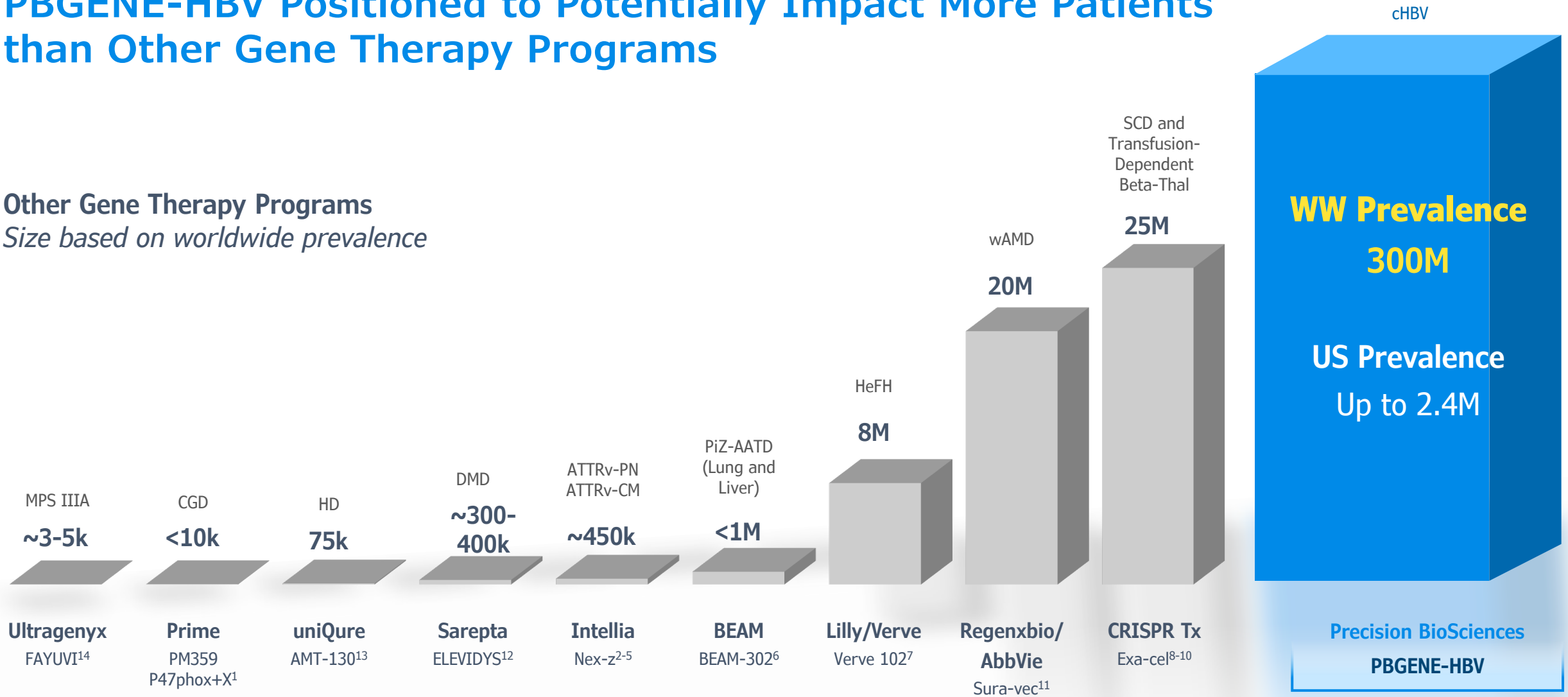
PRECISION
BIOSCIENCES

Appendix

PBGENE-HBV Positioned to Potentially Impact More Patients than Other Gene Therapy Programs

Other Gene Therapy Programs

Size based on worldwide prevalence



AATD, alpha-1 antitrypsin deficiency; ATTRv-CM, hereditary transthyretin amyloidosis with cardiomyopathy; ATTRv-PN, hereditary transthyretin amyloidosis with polyneuropathy; Beta-Thal, beta thalassemia; cHBV, chronic hepatitis B; CGD, chronic granulomatous disease; DMD, Duchenne muscular dystrophy; HD, Huntington's disease; HeFH, heterozygous familial hypercholesterolemia; k, thousand; M, million; MPS IIIA, mucopolysaccharidosis type IIIA; SCD, sickle cell disease; TD, transfusion-dependent; wAMD, wet age-related macular degeneration; WW, worldwide.

Prevalence does not equal addressable market.

1. Winkelstein JA, et al. Chronic granulomatous disease. Medicine (Baltimore). 2000;79(3):155. 2. Lovely M, et al. *Journal of Patient-Reported Outcomes*. 2021. 3. Lin H, et al. *Value Health*. 2019. 4. Morris A, et al. *Clin Ther*. 2022. 5. Detecting amyloidosis. AstraZeneca website. www.astrazeneca.com/media-centre/articles/2023/detecting-amyloidosis-understanding-the-different-types-of-attr.html. Published 2023. Accessed October 2, 2024. 6. Leme A, et al. *J Clin Invest*. 2021. 7. Familial hypercholesterolemia in adults: Overview. UpToDate website. www.uptodate.com/contents/familial-hypercholesterolemia-in-adults-overview#H1321616434. Accessed October 2, 2024. 8. Original Article: *Lancet Haematol*. 2023;10(8). 9. Correction: *Lancet Haematol*. 2023;10(8). 10. CRISPR Tx. EU application https://www.ema.europa.eu/en/documents/rmp/casgevy-epar-risk-management-plan_en.pdf. 11. Regenxbio. Sura-vec (ABBV-RGX-314) corporate presentation October 2026. Accessed October 27, 2026. 12. Prevalence and Incidence based on CureDuchenne and Orphanet Journal or Rare Diseases. 13. uniQure announces submission of biologics license application for AMT-130 for the treatment of Huntington's disease. News release. uniQure; September 2026. Accessed October 27, 2026. 14. Ultragenyx announces approval of FAYUVI™ gene therapy, the first-ever treatment for Sanfilippo syndrome type A. News release. Ultragenyx; September 2026. Accessed October 27, 2026.

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

US 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	~180,000	~4,000,000	~260,000
patients in US	patients in Europe	patients in China	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]

Main

B, billion; cHBV, chronic hepatitis B; HBV, hepatitis B virus; M, million.
Additional 142,000 potentially drug treated in Africa.[3]

1. Chattopadhyay D, 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):e00046-19. 3. Trucchio P. "Hepatitis B Breakthrough Boom: Navigating a $450-$500 Billion Frontier", H.C. Wainwright & Co. 14 Feb 2024.

Phase 2 Study Endpoints Aligned to Current HBV Regulatory Guidance

GUIDANCE DOCUMENT

Chronic Hepatitis B Virus Infection: Developing Drugs for Treatment

APRIL 2022

New finite duration therapies could be evaluated in clinical trials using any of the following efficacy endpoints:

- Sustained suppression (6 months or longer) of **HBV DNA** (less than LLOQ, TD or TND) off-treatment after a finite duration of therapy
- Sustained suppression (6 months or longer) of **HBV DNA** (less than LLOQ, TD or TND) off-treatment with HBsAg loss (less than 0.05 international unit/milliliter (IU/mL)) with or without HBsAb seroconversion after a finite duration of therapy

DNA, deoxyribonucleic acid; FDA, US Food and Drug Administration; HBsAb, hepatitis B surface antibody; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; LLOQ, lower limit of quantification; TD, target detected; TND, target not detected.

FDA Guidance – Chronic Hepatitis B Virus Infection; Developing Drugs for Treatment; April 2022. Accessed April 30, 2026.

PBGENE-HBV Goal

Phase 2 Design

PRECISION BIOSCIENCES

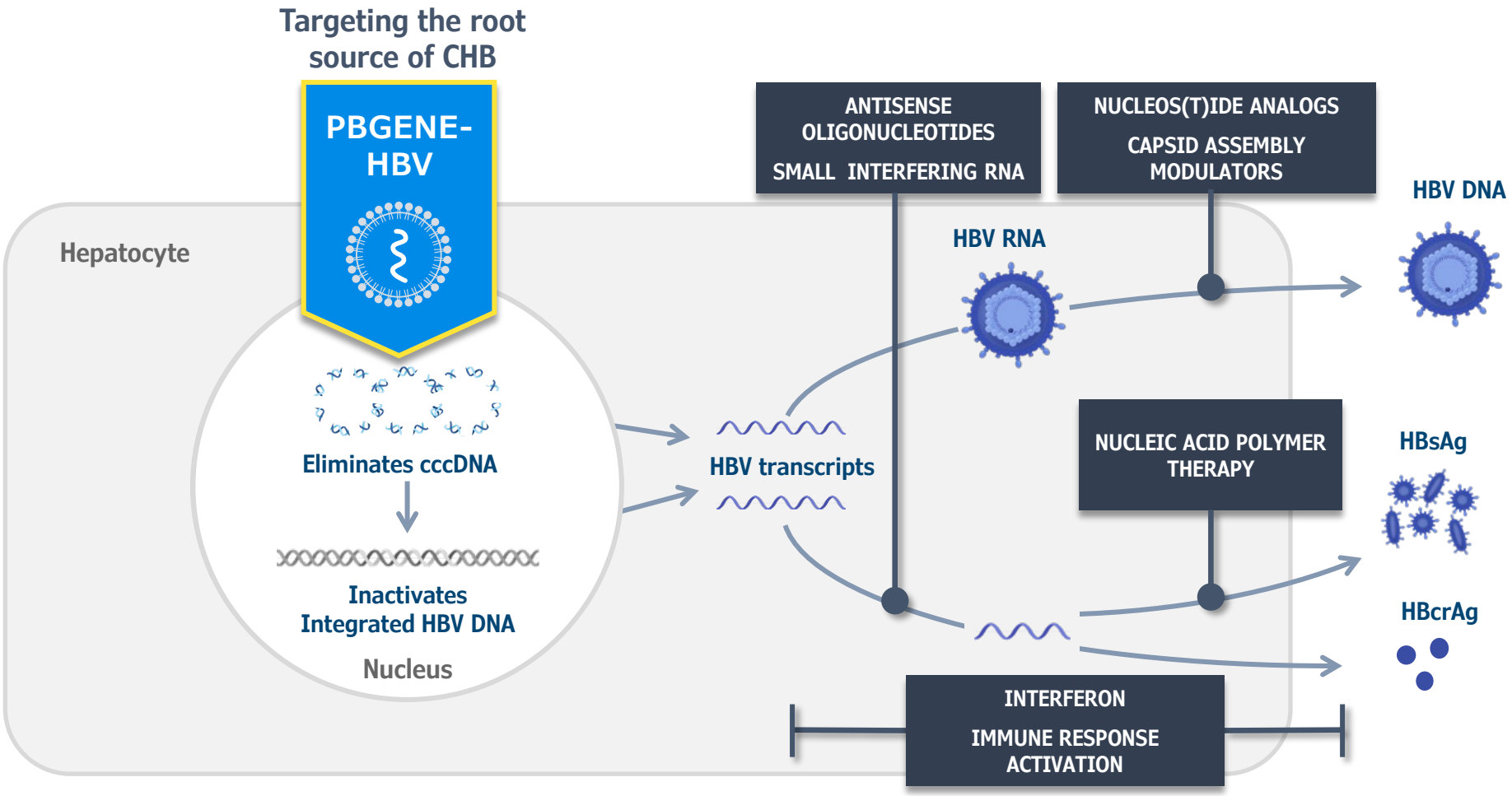
Founded in 2006 and **dedicated to developing novel therapeutics designed with goal of curing difficult-to-treat diseases with high unmet need,** including infectious and **rare genetic diseases.**

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

PBGENE-HBV Directly Eliminates cccDNA; All Other Therapies Suppress While Preserving cccDNA

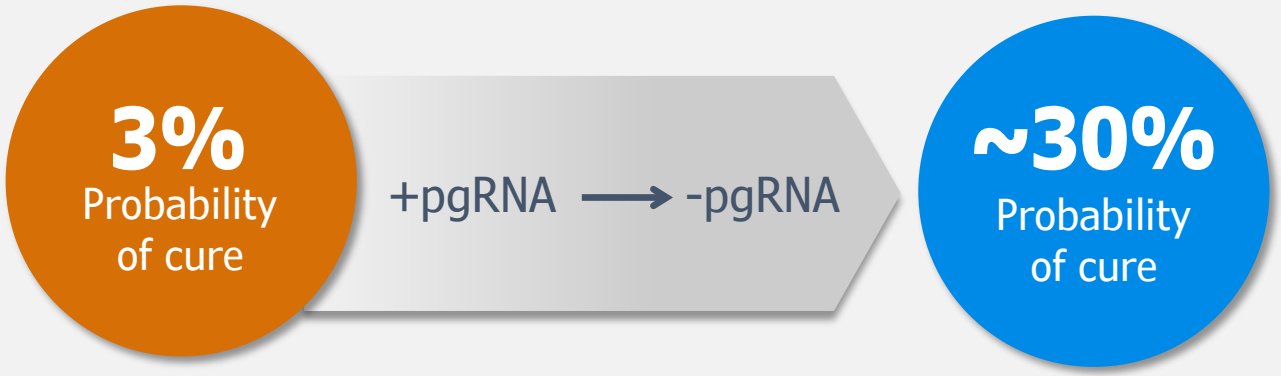


cccDNA, covalently closed circular DNA; CHB, chronic hepatitis B; HBcrAg, hepatitis B core-related antigen; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus.
Adapted from https://www.natap.org/2017/HBV/090517_02.htm

"The Problem"

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

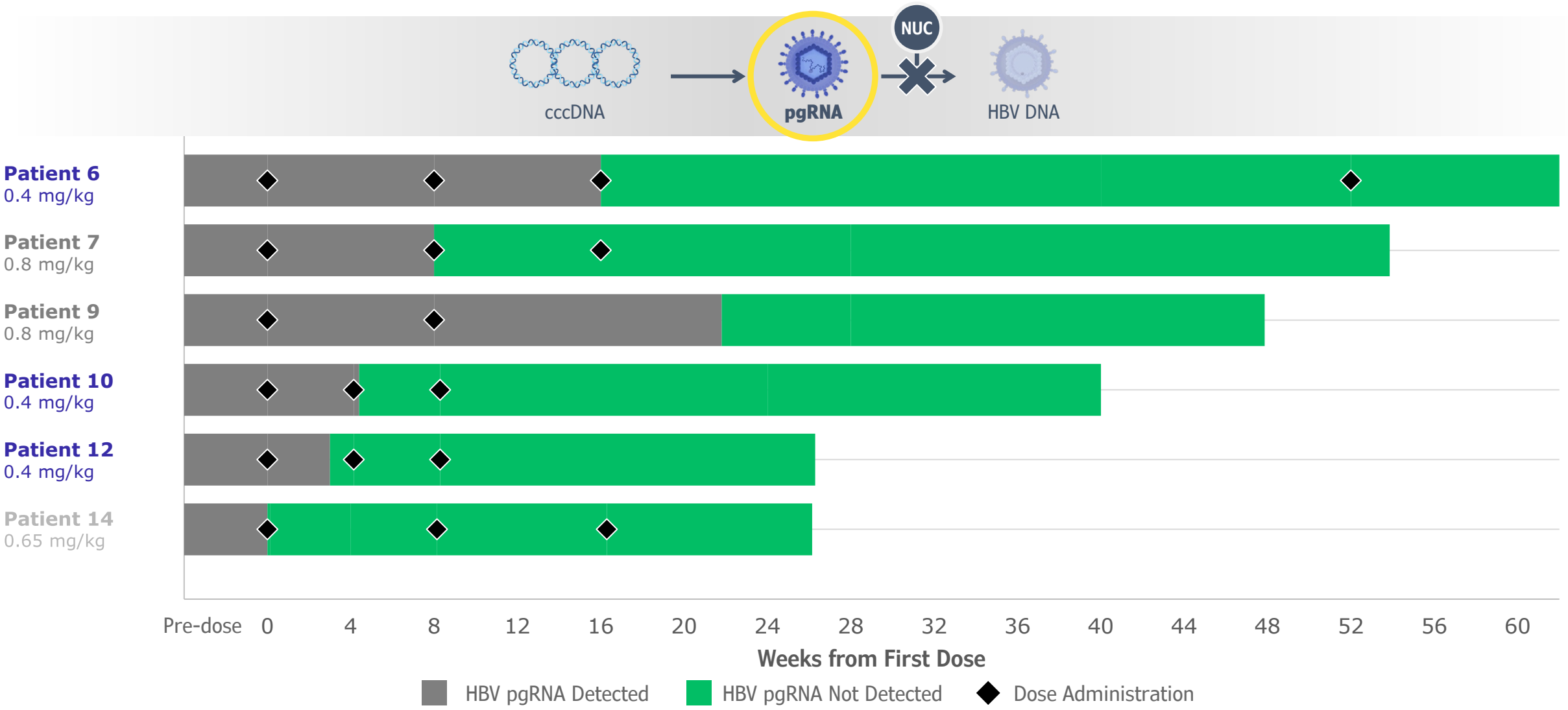
- Melbourne HBV-STOP trial (n=65)[3]
- DARING-B study (n=57)[4]
- Compilation of 23 cohort studies (n=2043)[5]

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.

NEW AASLD DATA: ALL EASL Patients Have Achieved 6+ Months of Durable pgRNA Loss



AASLD, American Association for the Study of Liver Diseases; cccDNA, covalently closed circular DNA; EASL, European Association for the Study of the Liver; HBV, hepatitis B virus; NUC, nucleos(t)ide analog; pgRNA, pregenomic RNA
Evaluable patients defined as those who have 28 days of data from initial dose.
Data cutoff: September 28, 2026.

Back to All Patients

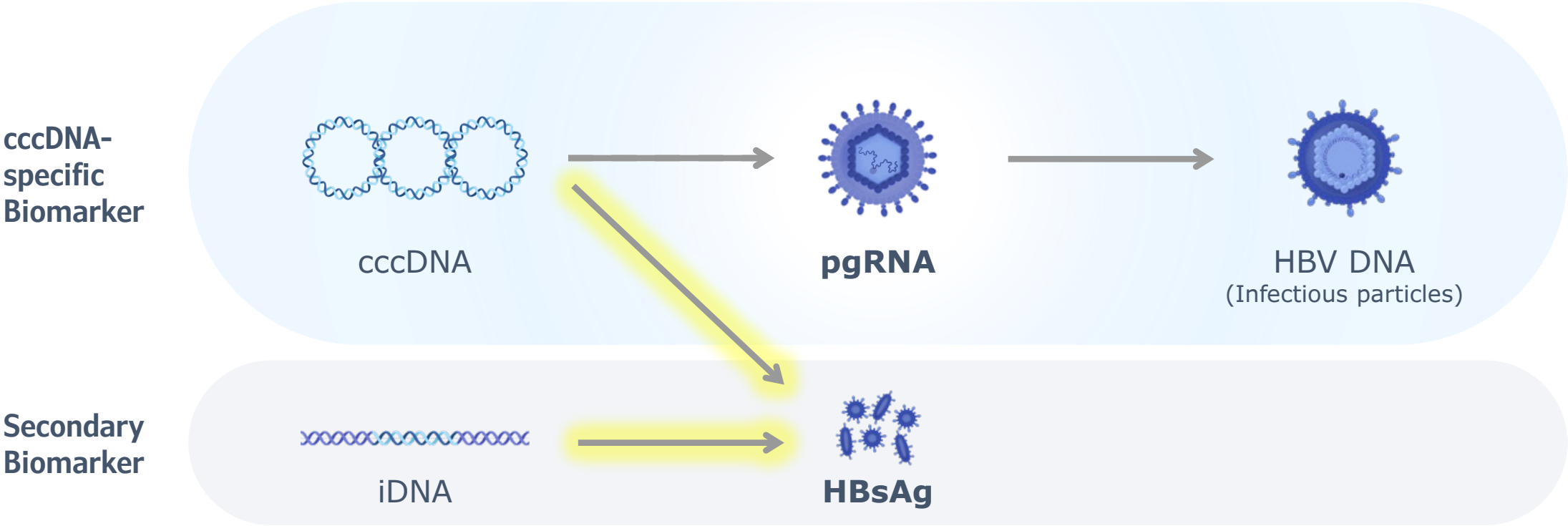
Lower cccDNA and Blood Biomarkers Track with Cure Rates

- Lower cccDNA in the liver is associated with significantly better odds of a sustained virologic response (adjusted odds ratio for log cccDNA was 5.3 [95% CI: 1.5-18.2, *P* = .009])
  - Sung et al, 2005, *Gastroenterology*
- Functionally cured patients show lower cccDNA and fewer HBsAg-positive hepatocytes than uncured patients; residual pool of cccDNA persists in functionally cured patients (eg, 2.70 copies/cell)
  - Gao et al, 2023, *Aliment Pharmacol Ther*
  - Xu et al, 2024, *Front Med*
- Blood pgRNA levels correlate with cccDNA levels more closely than HBsAg and are a significant predictor of off-treatment viral relapse
  - Wang et al, 2026, *J Viral Hepat*
  - Wang et al, 2021, *Virol J*

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

Back to Biopsies

HBsAg as a Secondary Biomarker Derived From cccDNA and Integrated HBV DNA



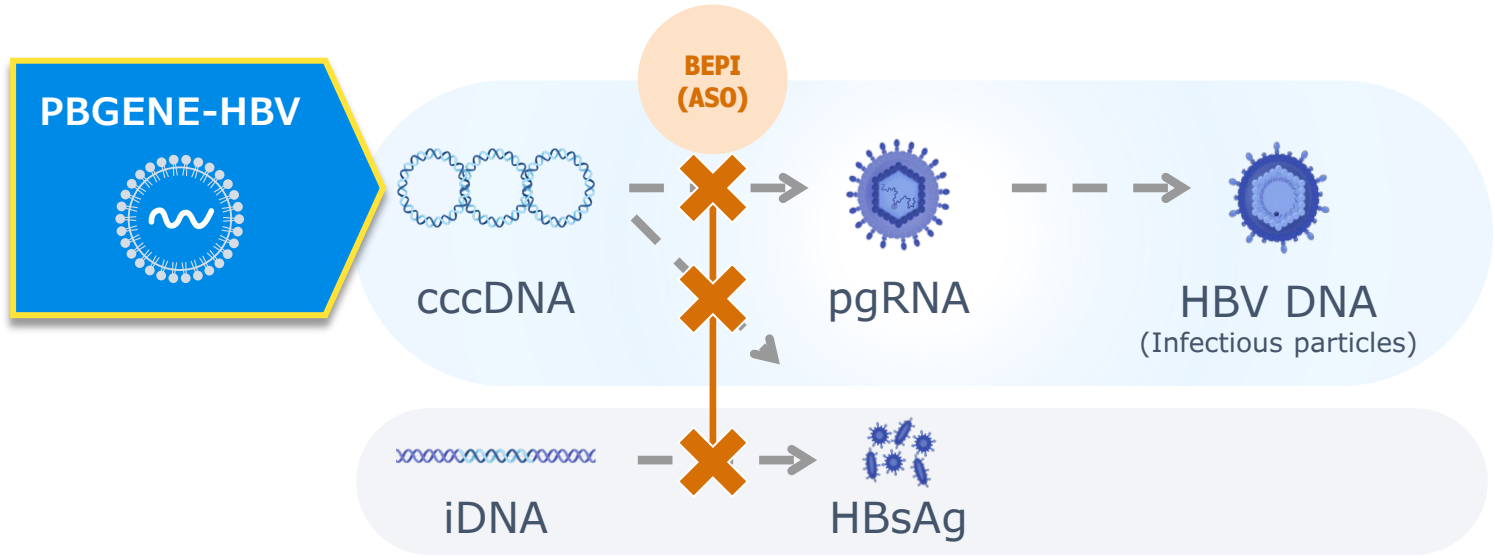
pgRNA is the cccDNA-specific biomarker
HBsAg is a secondary biomarker expected to be reduced with cccDNA elimination

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

Back to HBsAg data

Bepirovirsen Most Effective in Patients with Low Viral Burden; PBGENE-HBV Lowers Viral Burden and Potential to Increase Functional Cure Rate

Bepirovirsen works at the RNA level, not the DNA level



1 **PBGENE-HBV** Eliminates cccDNA to reduce viral burden

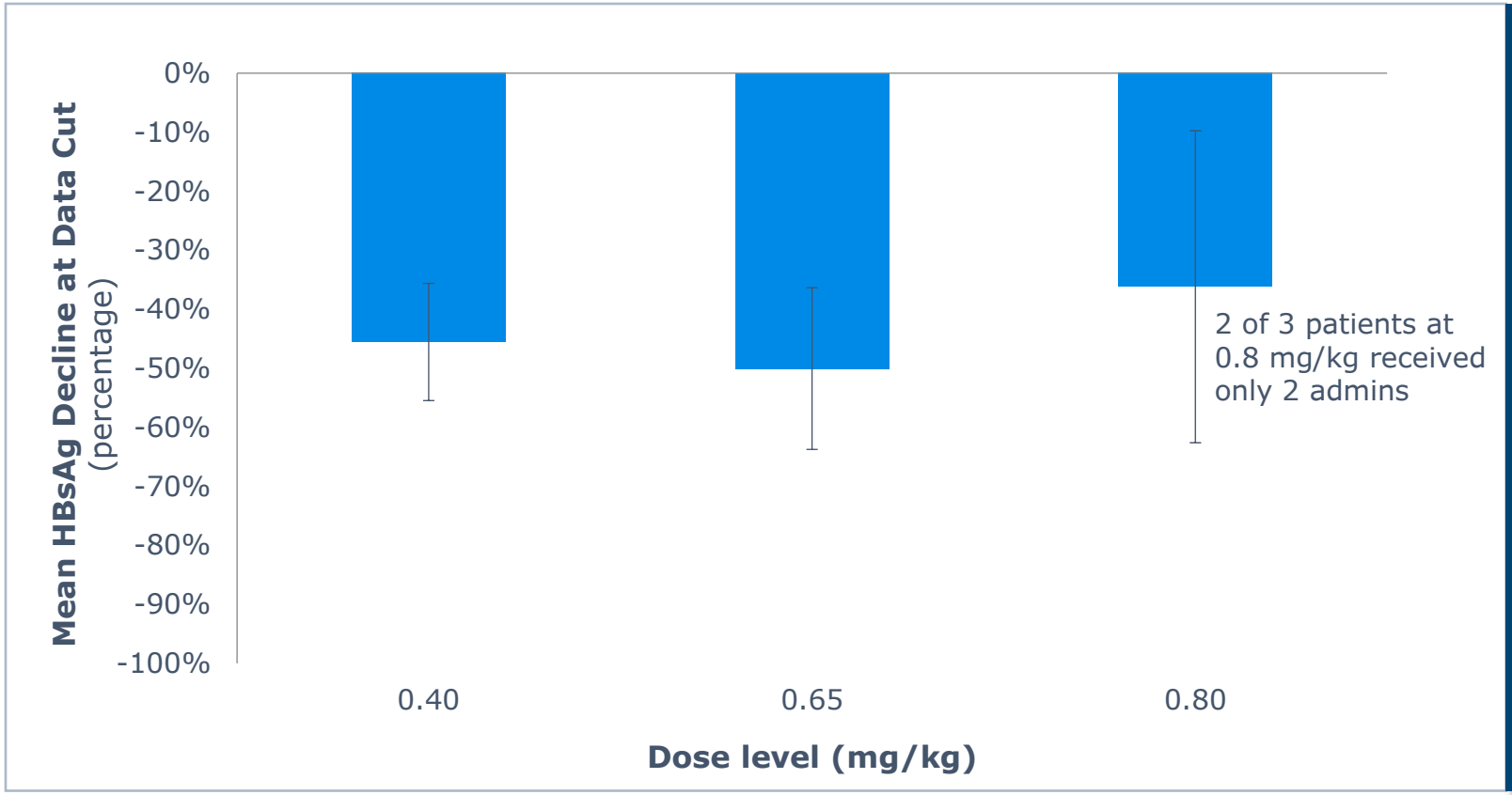
2 Goal of deeper suppression of viral transcripts with **Bepirovirsen**

3 Potential for a higher **functional cure** rate

ASO, antisense oligonucleotide; BEPI, bepirovirsen; cccDNA, covalently closed circular DNA; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus;
iDNA, integrated DNA; pgRNA, pregenomic RNA.

Combo Rationale

PBGENE-HBV Drove Durable HBsAg Declines at All Dose Levels ≥0.4 mg/kg



Durable HBsAg Declines at All Dose Levels ≥0.4 mg/kg

Patients with only 1 dose at 0.4 mg/kg as of the data cutoff have been excluded from this analysis.
HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus.
Data cutoff: September 28, 2026.

HBsAg Main Deck

PBGENE-HBV Safety Profile with CTCAE—Planned for Phase 2

CTCAE v6.0 –Planned for Phase 2

Number of patients with AEs[a]:		0.65 mg/kg n=6	0.4 mg/kg n=13	0.4 mg/kg w/ current infusion protocol n=8 of 13
Grade 3	Hypotension	-	-	-
Grade 3 and 4	ALT/AST lab abnormality	4	2	1
SAEs		-	-	-

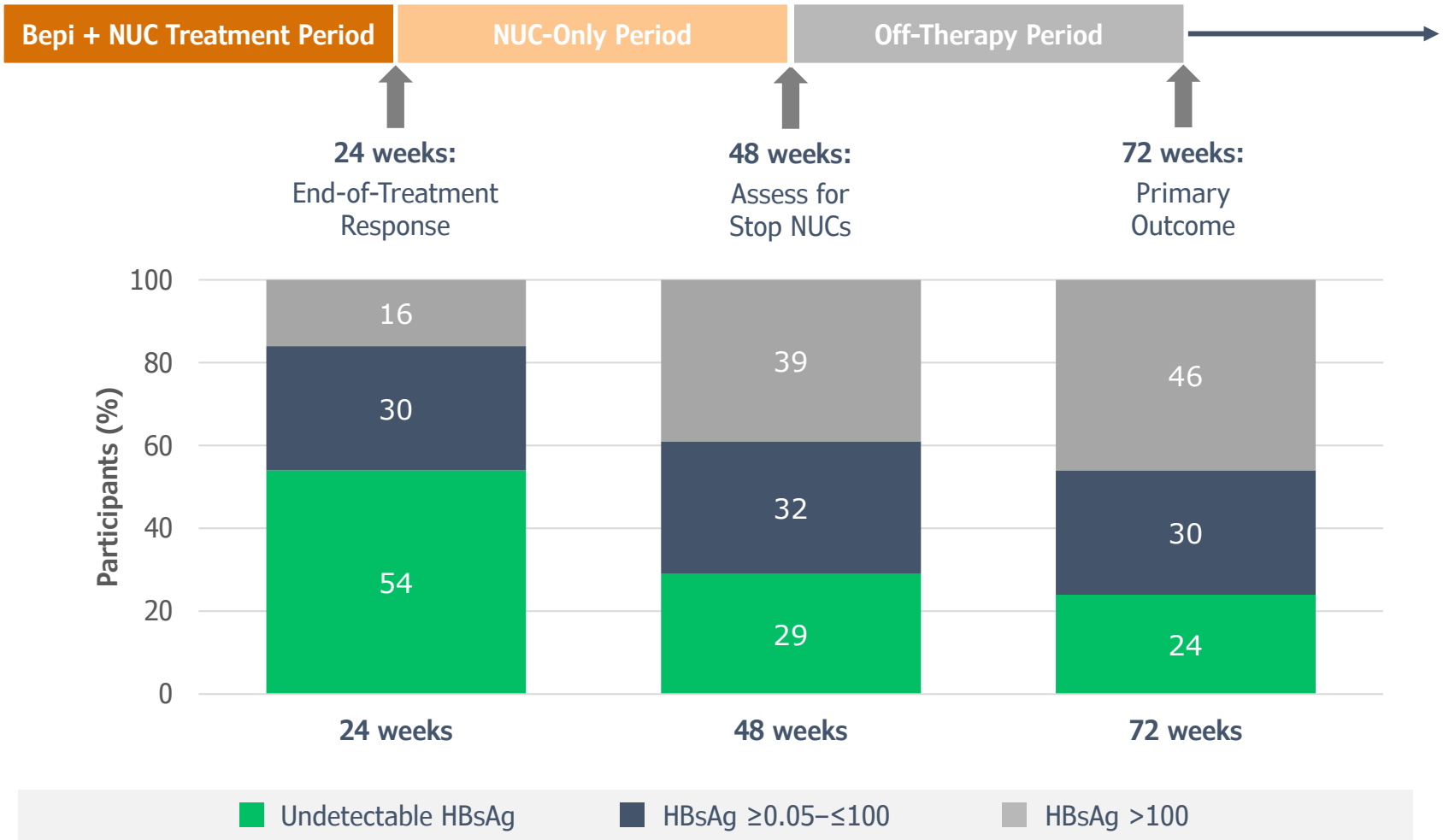
- An independent pharmacovigilance firm has assessed adverse events of interest in the Phase 1 ELIMINATE-B study and provided an analysis of these events graded under CTCAE, which will be used in the Phase 2 ELIMINATE-B2 study
- Currently, ELIMINATE-B has utilized a DAIDS framework for assigning grades to adverse events
  - DAIDS often used with small molecules in infectious disease studies
- For Phase 2, ELIMINATE-B2 will utilize CTCAE framework for assigning grades to adverse events
  - CTCAE is commonly used in studies utilizing LNPs[1,2,3]
  - Infusion-related reactions are a common effect of LNPs, and CTCAE has a dedicated grading system outlined for IRRs and DAIDS does not

CTCAE, Common Terminology Criteria for Adverse Events; AE, adverse event; ALT, alanine aminotransferase; AST, aspartate aminotransferase; DMC, data monitoring committee; HBV, hepatitis B virus; LNP, lipid nanoparticle; SAE, serious adverse event.
Data cutoff: September 28, 2026. Independent retrospective analysis was performed on hypotension and transaminase lab abnormalities.
[a]AEs shown are treatment-related and possibly treatment-related.

1. Gillmore, et al. *N Engl J Med*. 2021;385:493-502.
2. Cohn, et al. *N Engl J Med*. 2026;395:1471-1482.
3. Vafai, et al. *N Engl J Med*. 2026;395:648-659.

AE Table

Bepirovirsen Does Not Eliminate cccDNA, Thus End of Treatment Response is Not Maintained



End-of-Treatment Response: **54%**

Off-Therapy Response: **24%**

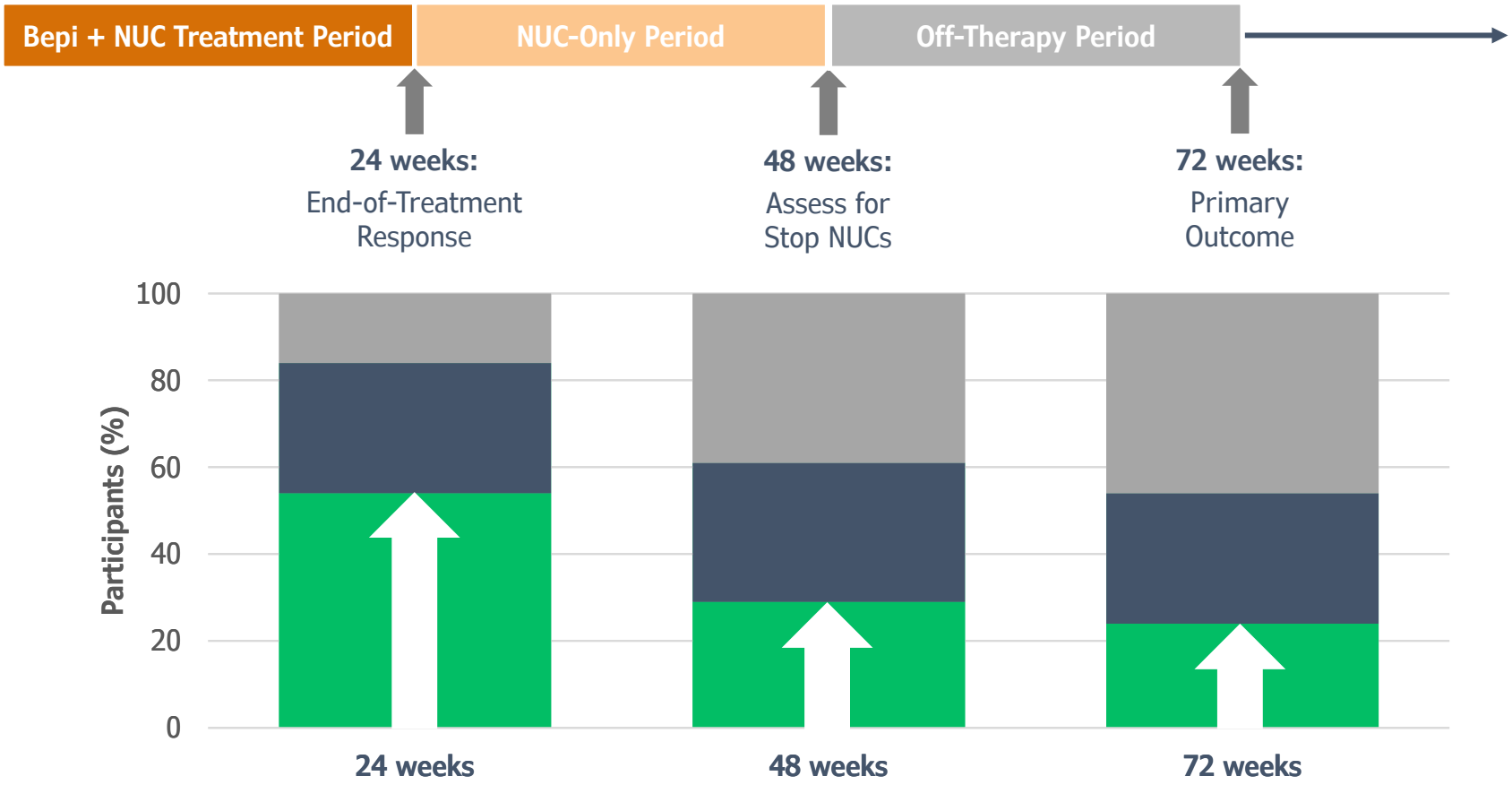
Phase 3 Results of Bepirovirsen Treatment for Chronic Hepatitis B Virus Infection

The NEW ENGLAND JOURNAL of MEDICINE

Discussion: "Sequential or combination strategies with complementary HBV therapies may increase the percentage of patients who can have a functional cure with bepirovirsen."

Bepi, bepirovirsen; cccDNA, covalently closed circular DNA; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; NUC, nucleos(t)ide analog.
Hou J, et al. *N Engl J Med.* 2026;394(24):2395-2406.

Combination of PBGENE-HBV With Bepirovirsen Potentially Improves Depth and Durability of Antiviral Activity



Goal of combination
is to increase
the bepirovirsen responders
by reducing the viral burden
with PBGENE-HBV
and thus drive higher
functional cure rates

Bepi, bepirovirsen; HBV, hepatitis B virus; NUC, nucleos(t)ide analog.

Hou J, et al. *N Engl J Med.* 2026;394(24):2395-2406.

PBGENE-DMD

Large Valuation Opportunity for Transformative Gene Therapy Drugs in DMD



FDA NEWS RELEASE

FDA Approves First Gene Therapy for Treatment of Certain Patients with Duchenne Muscular Dystrophy

For Immediate Release: June 22, 2023

Español

Today, the U.S. Food and Drug Administration approved Elevidys, the first gene therapy for the treatment of pediatric patients 4 through 5 years of age with Duchenne muscular dystrophy (DMD) with a confirmed mutation in the DMD gene who do not have a pre-existing medical reason preventing treatment with this therapy.

"Today's approval addresses an urgent unmet medical need and is an important advancement in the treatment of Duchenne muscular dystrophy, a devastating condition with limited treatment options, that leads to a progressive deterioration of an individual's health over time," said Peter Marks, M.D., Ph.D., director of the FDA's Center for Biologics Evaluation and Research. "The FDA remains committed to facilitating the development of innovative new therapies to reduce the impact of debilitating diseases and to improve outcomes and quality of life for those affected."

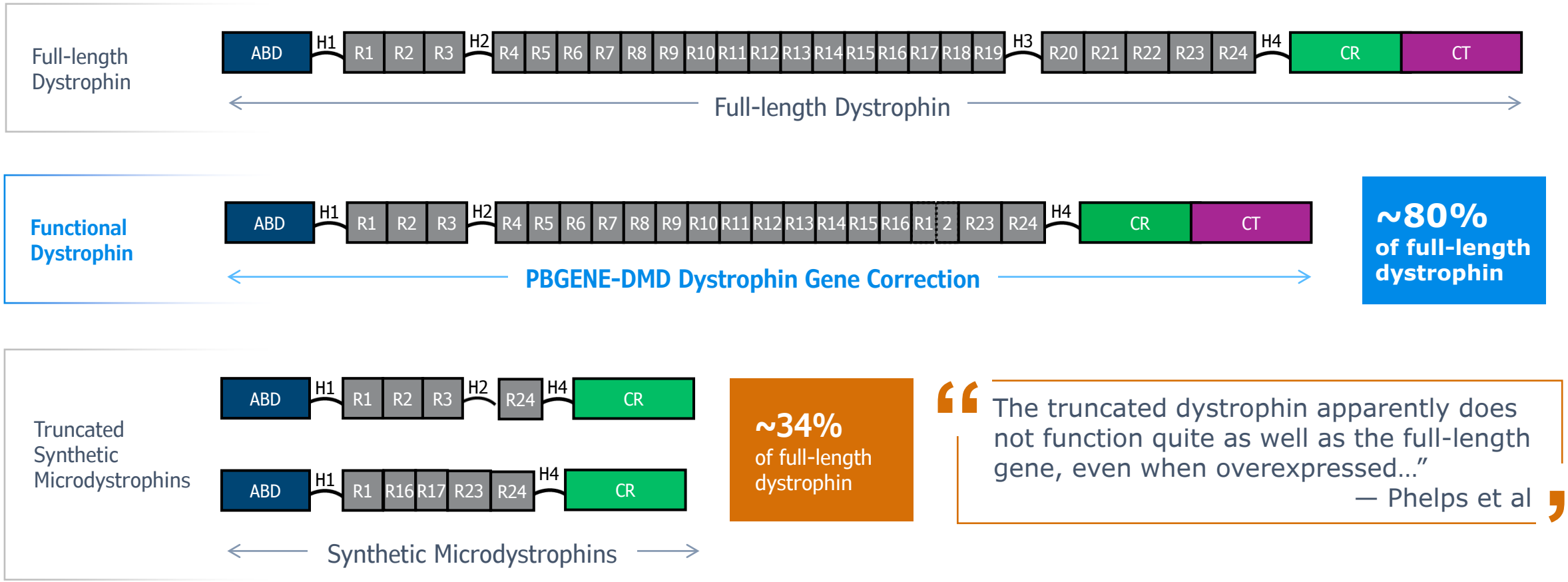
After receiving approval for its gene therapy Elevidys for DMD, Sarepta reached a peak market capitalization of ~$15.7B in June 2024.

This was an **increase from ~$3B in late 2017** when it received IND approval to initiate Phase 1 trials for Elevidys

https://clinicaltrials.gov/study/NCT03375164
Sources: https://www.macrotrends.net/stocks/charts/SRPT/sarepta-therapeutics/market-cap

Main

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

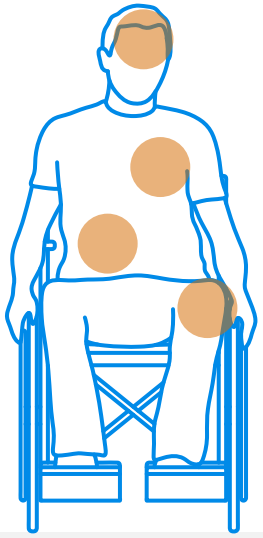


> "The truncated dystrophin apparently does not function quite as well as the full-length gene, even when overexpressed…"
>
> — Phelps et al

ABD, actin-binding domain; CR, cysteine-rich domain; CT, C-terminal domain; DMD, Duchenne muscular dystrophy.

Phelps SF, et al. *Hum Mol Genet.* 1995;4(8):1251-1258.

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



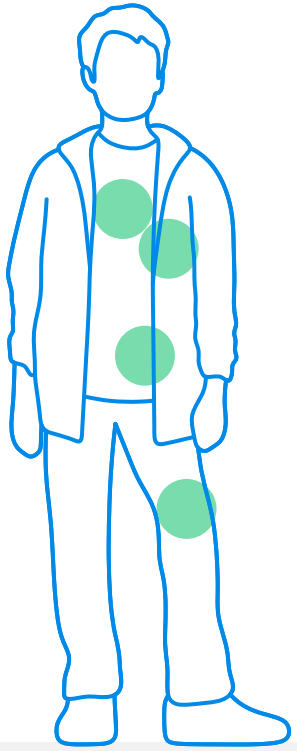
Lifespan:

- 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 60s-70s[1-3]

Clinical Presentation:

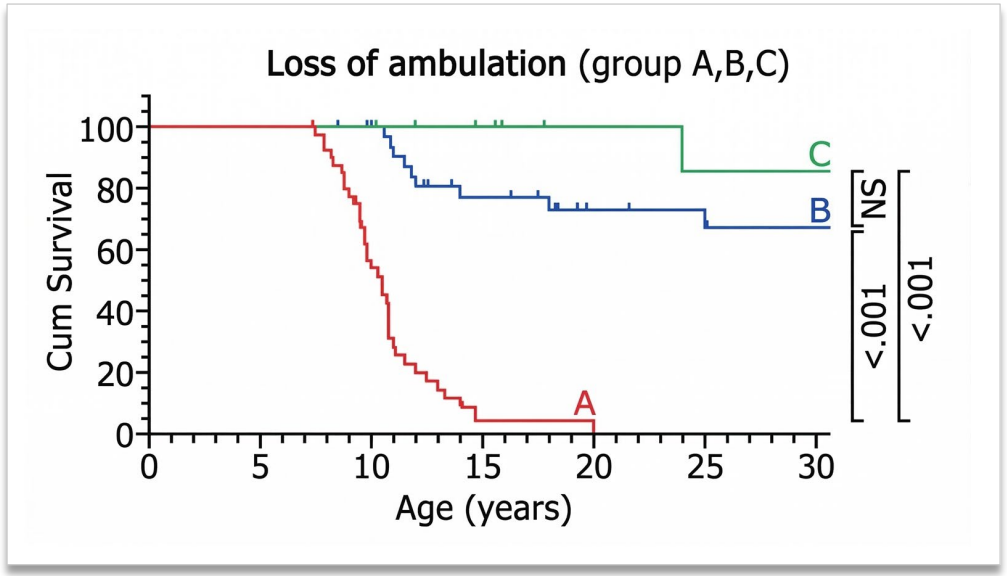
- Asymptomatic or mild symptoms[1-4]
- Normal muscle strength and ambulation throughout life[1,2]
- Normal respiratory function[2]
- Myocardial involvement, often manageable with medication[2]

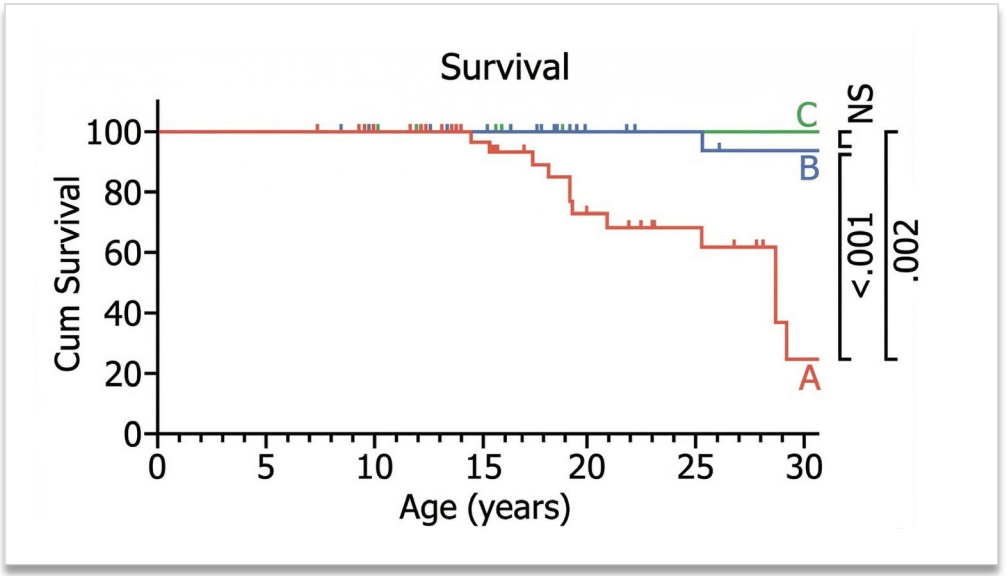
Del45-55 in-frame dystrophin gene (BMD)

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

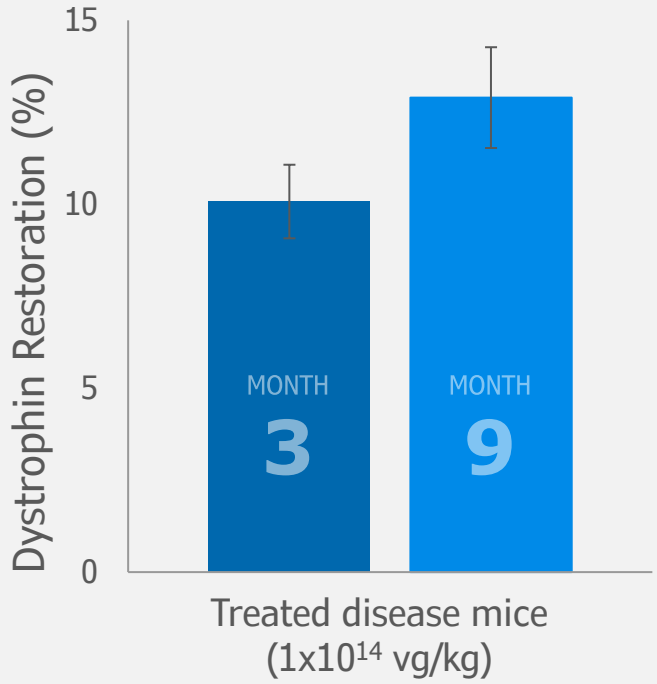
It is expected that as little as 5% expression of functional dystrophin protein is needed to provide therapeutic benefit in patients with DMD

DMD, Duchenne muscular dystrophy.
de Feraudy Y, et al. *Ann Neurol*. 2021;89(2):280-292.

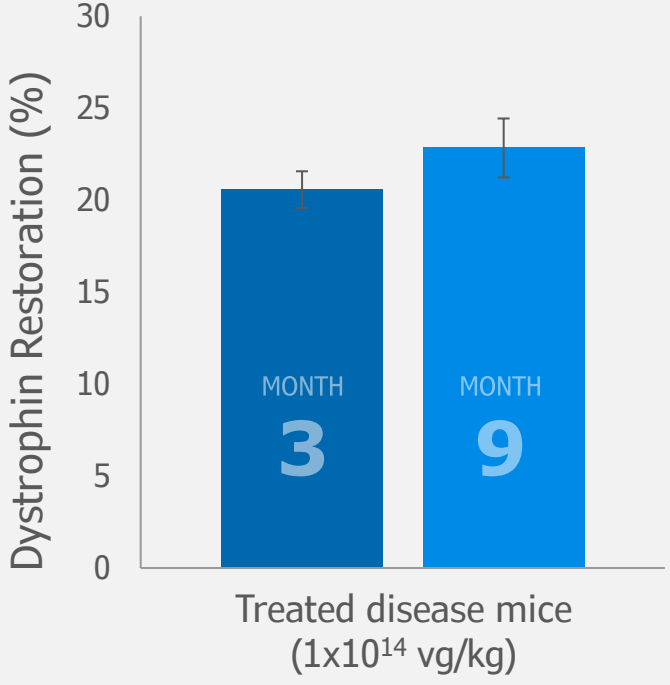
PBGENE-DMD Reason-to-Believe: Bridging to Investigation in Humans

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

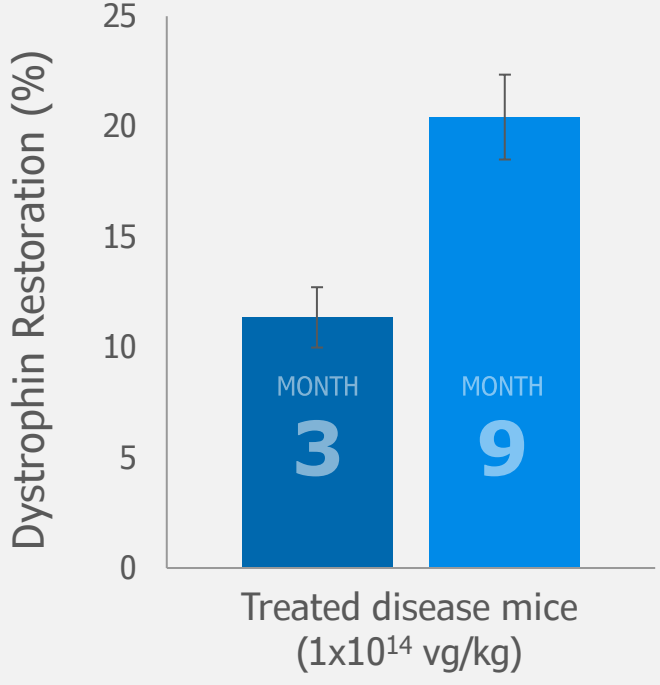
Heart



Calf



Quadriceps



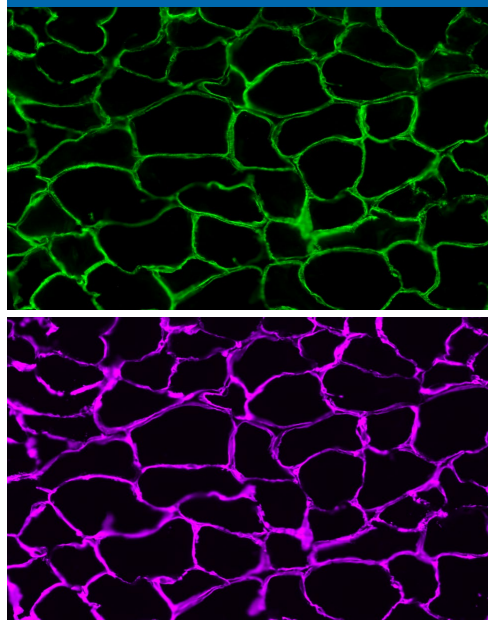


Endogenously produced, near-full-length functional dystrophin protein increases through 9 months in mice

DMD, Duchenne muscular dystrophy; vg/kg, vector genomes per kilogram.
https://investor.precisionbiosciences.com/events/event-details/precision-biosciences-virtual-investor-event-duchenne-muscular-dystrophy-dmd

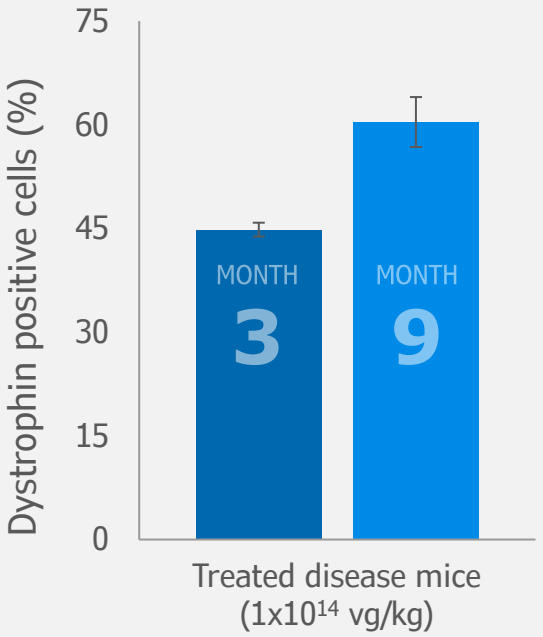
Broad Distribution of Dystrophin Positive Cells Across Skeletal, Cardiac, and Respiratory Muscles After PBGENE-DMD Treatment

PBGENE-DMD
Disease Model

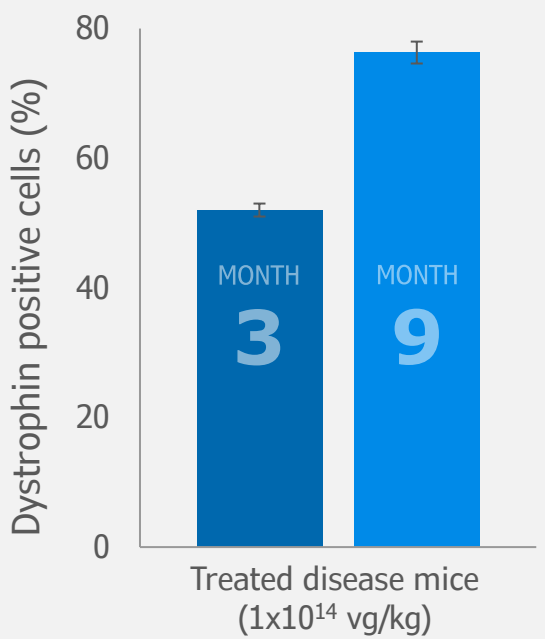


Dystrophin Laminin

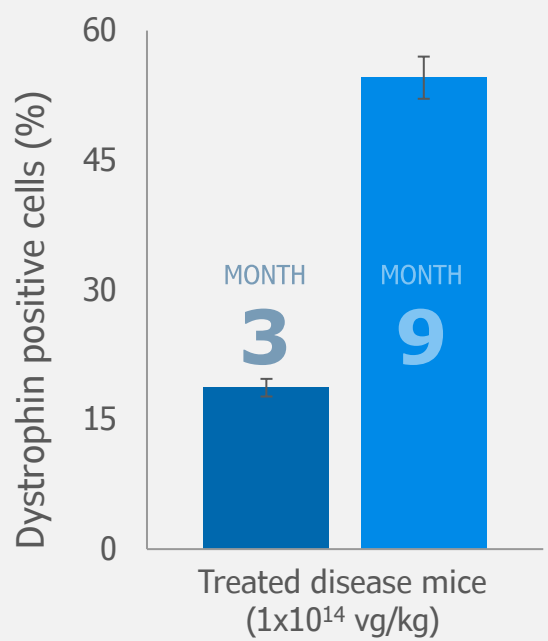
Heart



Calf



Quadriceps



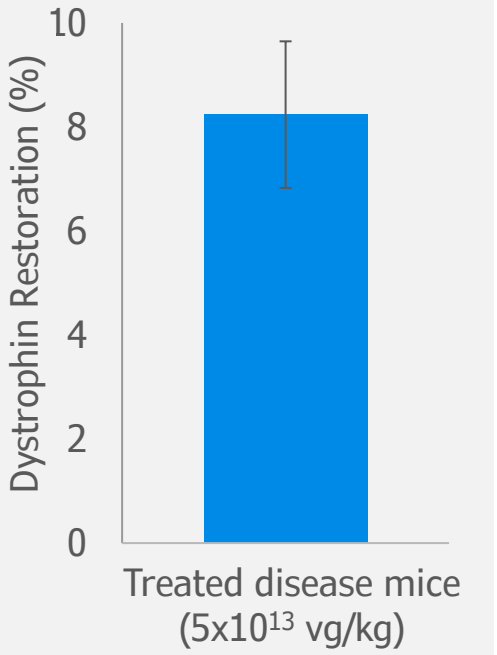


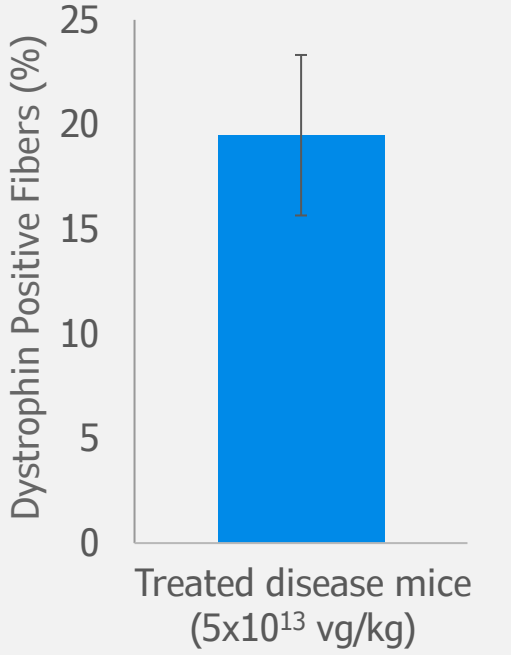
Percentage of cells expressing near-full-length dystrophin protein increases over time after treatment with PBGENE-DMD

DMD, Duchenne muscular dystrophy; vg/kg, vector genomes per kilogram.
https://investor.precisionbiosciences.com/events/event-details/precision-biosciences-virtual-investor-event-duchenne-muscular-dystrophy-dmd

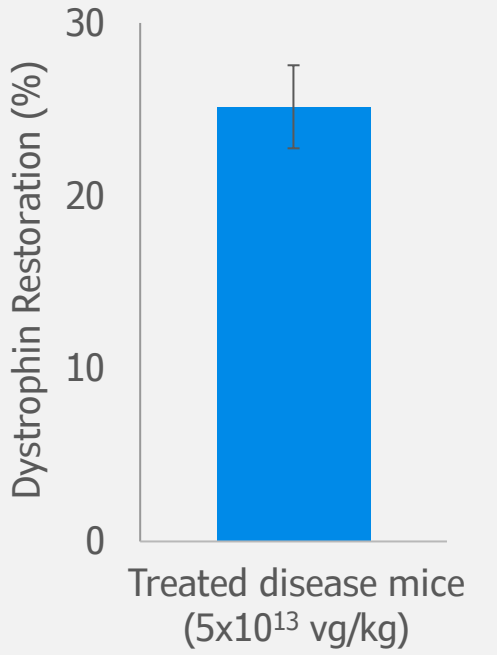
PBGENE-DMD Achieves Therapeutic Dystrophin Restoration in Critical Respiratory Muscles of Early Juvenile DMD Mice

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

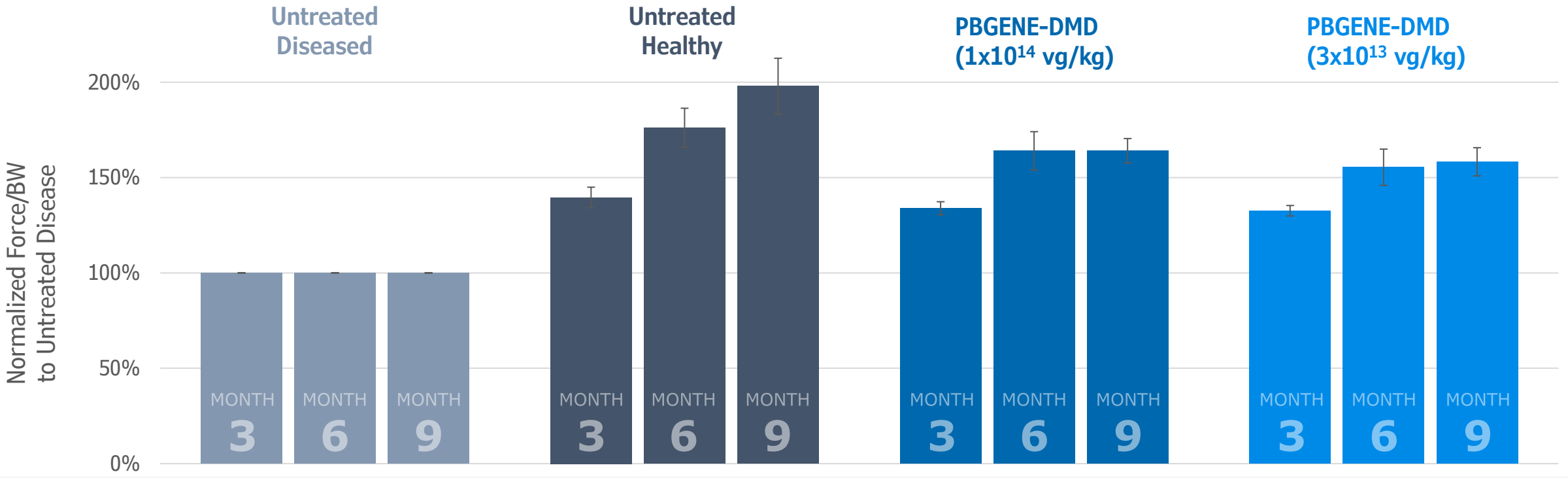
Dystrophin positive fibers in early juvenile mice were up to 2-3X higher in skeletal and respiratory muscle compared to late juvenile mice



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.
https://investor.precisionbiosciences.com/events/event-details/precision-biosciences-virtual-investor-event-duchenne-muscular-dystrophy-dmd

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

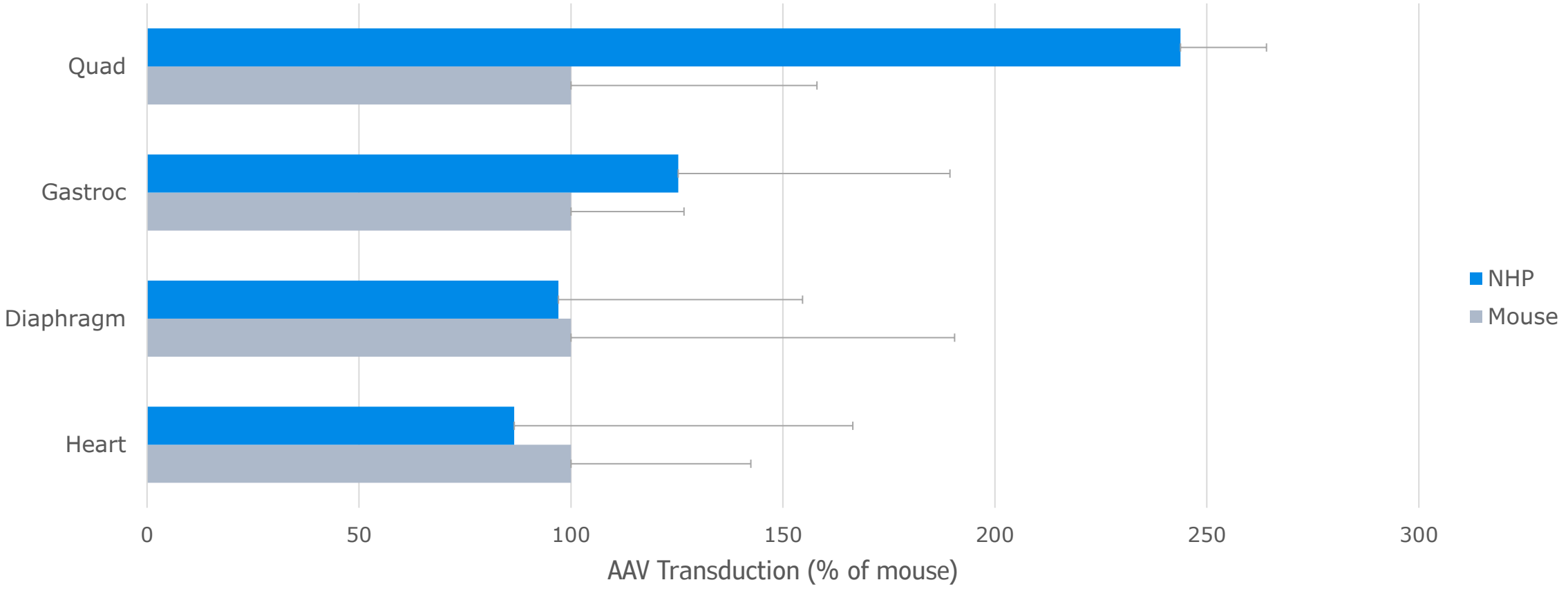




- Improved muscle function observed from 3 to 6 months
- Durable functional improvements maintained out to 9 months
- Benefit consistent across both experimental dose levels

Force was measured in the calf across multiple stimulation frequencies. Averaged force normalized to body weight is shown. Statistically significant ($p<0.001$) increases in force were observed in both doses of PBGENE-DMD compared with untreated diseased animals at both time points.
BW, body weight; DMD, Duchenne muscular dystrophy.
https://investor.precisionbiosciences.com/events/event-details/precision-biosciences-virtual-investor-event-duchenne-muscular-dystrophy-dmd

PBGENE-DMD Demonstrated as Good as 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.
https://investor.precisionbiosciences.com/events/event-details/precision-biosciences-virtual-investor-event-duchenne-muscular-dystrophy-dmd

Nonclinical Data Support 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 2 GLP studies conducted in DMD mouse model and healthy nonhuman primates
- No off-target editing above background levels was 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 $3x10^{13}$ and $1x10^{14}$ vg/kg
- Similar levels of dystrophin protein and percent positive myofibers in the heart at both $3x10^{13}$ and $1x10^{14}$ vg/kg
- Increases in dystrophin protein restoration and percent positive myofibers in the diaphragm at $1x10^{14}$ vg/kg

Together these data support potential safety and efficacy of $1x10^{14}$ 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.
https://investor.precisionbiosciences.com/events/event-details/precision-biosciences-virtual-investor-event-duchenne-muscular-dystrophy-dmd

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**	**1×10¹⁴ vg/kg**
SOLID BIOSCIENCES	**SGT-001[2]***	AAV9	**Up to 2×10¹⁴ vg/kg**
	SGT-003[3]	AAV9-derived	**1×10¹⁴ vg/kg**
Pfizer	**fordadistrogene movaparvovec (PF-06939926)[4]***	AAV9	**Up to 3×10¹⁴ vg/kg**
SAREPTA THERAPEUTICS	**delandistrogene moxeparvovec (Elevidys)[5]**	rAAVrh74	**Up to ~1.33×10¹⁴ vg/kg**
REGENXBIO	**RGX-202[6]**	AAV8	**Up to 2×10¹⁴ GC/kg**

*Discontinued.
AAV, adeno-associated virus; DMD, Duchenne muscular dystrophy; GC/kg, genome copies per kilogram; vg/kg, viral genomes per kilogram.
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 1/2 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 Weeks 12 and 52

Goal

Demonstrates target engagement and biological proof-of-concept

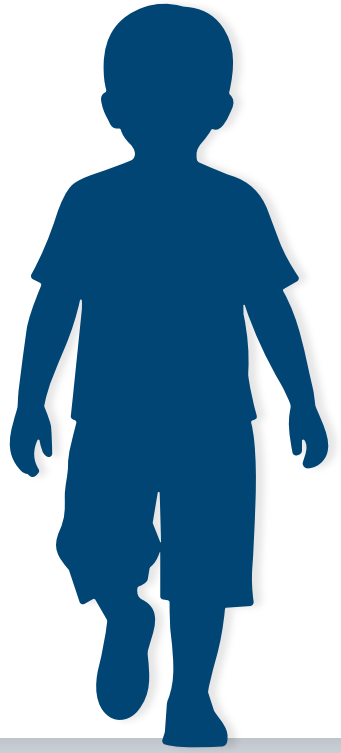
Exploratory Endpoints

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; DMD, Duchenne muscular dystrophy; DMO, digital mobility outcome; NSAA, North Star Ambulatory Assessment; SAE, serious adverse event; TRAE, treatment-related adverse event.
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

AAV, adeno-associated virus; DMD, Duchenne muscular dystrophy; NSAA, North Star Ambulatory Assessment.
Updated February 24, 2026. https://clinicaltrials.gov/study/NCT07429240. Accessed February 25, 2026.

Safety as Top Priority:

Immune Modulation Regimen (IMR) strategy ensures monitoring and interventions are optimized to patient safety needs during 2 key safety risk periods

Safety Mitigation: Preventing early and late serious immune complications.

AAV therapies can trigger **2 predictable immune-related safety risk periods.** To keep patients safe, we plan to use a 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 control 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; TMA, thrombotic microangiopathy.
https://investor.precisionbiosciences.com/events/event-details/precision-biosciences-virtual-investor-event-duchenne-muscular-dystrophy-dmd