UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): November 5, 2026

Precision BioSciences, Inc.

(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-38841**	**20-4206017**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

302 East Pettigrew St.
Suite A-100
Durham, North Carolina **27701**

(Address of Principal Executive Offices) **(Zip Code)**

Registrant's Telephone Number, Including Area Code: 919 314-5512

(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, par value $0.000005 per share	DTIL	The Nasdaq Capital Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure

On November 5, 2026, Precision BioSciences, Inc. (the "Company") issued a press release to provide an update on its ELIMINATE-B clinical trial evaluating PBGENE-HBV for chronic hepatitis B (the "HBV News Release"). A copy of the HBV News Release is furnished as Exhibit 99.1 to this Current Report on Form 8-K. The abstract for the Company's late-breaking oral presentation at the American Association for the Study of Liver Diseases ("AASLD") The Liver Meeting® 2026 was published on November 5, 2026 and is furnished as Exhibit 99.2 to this Current Report on Form 8-K (the "PBGENE-HBV Late Breaker Oral Abstract").

On November 5, 2026, the Company also issued a press release to announce the first patient dosed with PBGENE-DMD completed the eight week observation period following treatment with no reported safety concerns, including no serious adverse events or adverse events of special interest (the "DMD News Release" and, together with the HBV News Release, the "News Releases"). The independent Data Safety Monitoring Board ("DSMB") for the Phase 1/2 FUNCTION-DMD clinical trial for Duchenne muscular dystrophy ("DMD") endorsed proceeding with dosing additional patients in Part 1 of the study. A copy of the DMD News Release is furnished as Exhibit 99.3 to this Current Report on Form 8-K.

As described in the HBV News Release, the Company will host a live webcast on Monday, November 9, 2026 at 8:00 a.m., Eastern Time, to present the late-breaking AASLD Phase 1 data. The webcast link is https://lifescievents.com/event/pgy32le/. A replay of the presentation will available following the event on the Company's website in the Investors section under Events & Presentations: https://investor.precisionbiosciences.com/events-and-presentations.

The Company is also furnishing its updated corporate deck (the "Corporate Presentation") for use by management. A copy of the Corporate Presentation is furnished as Exhibit 99.4 to this Current Report on Form 8-K.

Pursuant to General Instruction B.2 of Current Report on Form 8-K, the information contained in, or incorporated into, this Item 7.01 (including the News Releases, PBGENE-HBV Late Breaker Oral Abstract and Corporate Presentation attached hereto as Exhibits 99.1, 99.2, 99.3 and 99.4) of this Current Report on Form 8-K is being furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that Section, nor shall it be deemed to be incorporated by reference into any registration statement or other filing of the Company under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 8.01 Other Events

On November 5, 2026, the Company issued the HBV News Release announcing a late-breaking oral presentation at the American Association for the Study of Liver Diseases (AASLD) The Liver Meeting® 2026. This presentation includes new and updated preliminary data from the ongoing Phase 1 ELIMINATE-B study of PBGENE-HBV, the first and only in vivo gene editing therapy designed to eliminate cccDNA, the root cause of chronic hepatitis B. The PBGENE Late Breaker Abstract for this presentation was published on November 5, 2026.

As of the September 28, 2026 data cutoff date, 25 patients had received a total of 59 administrations of PBGENE-HBV across four dose levels. Key findings include:

- Phase 1 ELIMINATE-B study successful, PBGENE-HBV program advancing to Phase 2
- PBGENE-HBV achieves target profile at 0.4 mg/kg dose, evaluating both monotherapy and combination therapy for chronic hepatitis B, with dosing expected to begin in the first quarter of 2027
- Achieved undetectable pgRNA in 100% of evaluable patients at expected go forward dose 0.4 mg/kg (one patient, dosed with two administrations at 0.65mg/kg, experienced a 95% reduction in pgRNA at the time of the data cutoff, with further assessment ongoing); Durable and ongoing pgRNA loss in 100% of patients who achieved undetectable pgRNA and had ≥6 months of follow-up
- First HBeAg positive patient achieved a 100% pgRNA loss from a baseline of 3.7-log copies/mL at 0.4 mg/kg dose

- Reproducible liver biopsy findings demonstrated 1-log to 1.5-log reductions in cccDNA at the 0.4 mg/kg expected go forward dose across multiple patients
- Treatment effect observed in 100% of evaluable patients as measured by HBsAg reduction; maximal HBSAg reductions approached 94% at 0.4 mg/kg, with antiviral activity observed in patients with and without pgRNA at baseline
- PBGENE-HBV demonstrated a well-characterized and manageable safety profile across 59 administrations in 25 patients, with no dose-limiting toxicities and no serious adverse events at the 0.4 mg/kg go-forward dose

On November 5, 2026, the Company also issued the DMD News Release. The DMD News Release announced that the first patient dosed with PBGENE-DMD completed the eight week observation period following PBGENE-DMD treatment with no reported safety concerns, including no serious adverse events or adverse events of special interest reported through week eight, and the independent DSMB for the FUNCTION-DMD clinical trial endorsed proceeding with dosing additional patients in Part 1 of the study. Safety and efficacy updates are expected from the FUNCTION-DMD trial throughout 2027.

Forward-Looking Statements

Statements in this Current Report on Form 8-K regarding management's future expectations, beliefs, intentions, goals, strategies, plans or prospects are forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, including but not limited to statements regarding the further development and potential of our PBGENE-HBV program and ELIMINATE-B clinical trial and our PBGENE-DMD program and FUNCTION-DMD clinical trial, the ability of the ELIMINATE-B trial to advance into a global Phase 2 trial and the timing and progress of the study; and the expected timing of clinical milestones, including the expectation of safety and efficacy updates from the FUNCTION-DMD trial throughout 2027. In some cases, you can identify forward-looking statements by terms such as "aim," "anticipate," "approach," "believe," "contemplate," "could," "estimate," "expect," "goal," "intend," "look," "may," "mission," "plan," "potential," "predict," "project," "should," "target," "will," "would," or the negative thereof and similar words and expressions. Such forward-looking statements involve known and unknown risks, uncertainties and other important factors, including, without limitation, the risks referred to under the section "Risk Factors" in the Company's Annual Report on Form 10-K for the fiscal year ended December 31, 2025 and its Quarterly Reports on Form 10-Q for the quarterly periods ended March 31, 2026, June 30, 2026 and September 30, 2026, as such factors may be updated from time to time in the Company's other filings with the Securities and Exchange Commission ("SEC"), which filings are accessible on the SEC's website at www.sec.gov and the Investors & Media page of the Company's website. All forward-looking statements speak only as of the date of this Current Report on Form 8-K, 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.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	HBV News Release of Precision BioSciences, Inc. dated November 5, 2026.
99.2	PBGENE-HBV Late Breaker Oral Abstract dated November 5, 2026.
99.3	DMD News Release of Precision BioSciences, Inc. dated November 5, 2026.
99.4	Corporate Presentation.
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

PRECISION BIOSCIENCES, INC.

Date: November 5, 2026

By: /s/ Naresh Tanna

Naresh Tanna

Chief Financial Officer

Precision BioSciences Announces Late-Breaking PBGENE-HBV Data at AASLD The Liver Meeting® 2026 Further Demonstrating cccDNA Elimination and Potential to Drive Viral Cure

- Phase 1 ELIMINATE-B study successful, PBGENE-HBV Program Advancing to Phase 2-

- PBGENE-HBV achieves target product profile at 0.4 mg/kg dose-

- PBGENE-HBV *achieved pgRNA loss to undetectable in 100% of evaluable patients at expected go-forward dose of 0.4 mg/kg; Durable and ongoing pgRNA loss in 100% of patients with ≥6 months of follow-up -*

- In hard-to-treat patient population: HBeAg-positive patient achieved a 100% pgRNA loss to undetectable from a baseline of 3.7-log copies/mL at 0.4 mg/kg dose, patient's pgRNA remained undetectable at data cut-off -

- Reproducible liver biopsy findings: PBGENE-HBV demonstrated a 1-log and 1.5-log reduction in cccDNA at 0.4 mg/kg the expected go forward dose across multiple patients -

- Treatment effect observed in 100% of evaluable patients as measured by HBsAg reduction -

- PBGENE-HBV demonstrated a well-characterized and manageable safety profile across 59 administrations in 25 patients, with no dose-limiting toxicities -

- Company to host investor and KOL webcast on Monday, November 9, 2026, at 8:00 a.m. ET presenting the late-breaking AASLD Phase 1 data -

DURHAM, N.C., November 5, 2026 -- Precision BioSciences, Inc. (Nasdaq: DTIL), a clinical stage gene editing company utilizing its novel proprietary ARCUS® platform to develop *in vivo* gene editing therapies for high unmet need diseases, today announced new and updated Phase 1 clinical data from the ongoing ELIMINATE-B study of PBGENE-HBV to be presented in a late-breaking oral presentation on Sunday, November 8 at 2:15pm MST, at the American Association for the Study of Liver Diseases (AASLD) The Liver Meeting® 2026. PBGENE-HBV is the first and only *in vivo* gene editing therapy designed to eliminate the root cause of chronic hepatitis B, covalently closed circular DNA (cccDNA), to drive viral cure measured by sustained undetectable HBV DNA.

The oral presentation will be delivered by our ELIMINATE-B lead investigator, Man Fung Yuen, MD, Ph.D., Chair Professor of The University of Hong Kong and the Chief of the Division of Gastroenterology and Hepatology, Queen Mary Hospital. The presentation features new and updated data from 25 patients across 59 administrations in the Phase 1 ELIMINATE-B trial. A copy of the presentation will be available on the Company's website following Dr. Yuen's oral presentation.

"The new clinical data from ELIMINATE-B represent a breakthrough in our efforts to address the underlying cause of chronic hepatitis B, cccDNA," said Michael Amoroso, President and Chief Executive Officer of Precision BioSciences. "The consistency of the findings at the 0.4 mg/kg dose, including complete and durable pgRNA loss to undetectable levels, reproducible liver biopsies demonstrating evidence of cccDNA elimination, and a treatment effect in all patients with a manageable safety profile, supports advancement to a global Phase 2 study. In the Phase 2 study, we plan to evaluate cure rates after a finite course of treatment and cessation of nucleos(t)ide analogs. Patients have long desired a cure for their virus and living a life free from daily medication - today's data is a step in that direction."

AASLD Presentation Shows Evidence of cccDNA Elimination and Favorable Safety Profile at 0.4 mg/kg

As of the data cutoff date, September 28, 2026, 25 patients had received a total of 59 administrations of PBGENE-HBV across four dose levels.

“We’re thrilled to share the complete and durable loss of pgRNA in 100% of evaluable patients at our go-forward dose of 0.4 mg/kg, as well as durable, ongoing pgRNA loss in all patients who had at least 6-months of follow up, which we believe is indicative of permanent elimination of cccDNA in the liver. In total, across all dose levels, 11 evaluable patients achieved pgRNA loss to undetectable and remained undetectable through data cut off. The highlight of the data was demonstrating pgRNA loss to undetectable levels in our first HBeAg positive patient dosed on study, indicating potent effect of PBGENE-HBV in a patient population that is very difficult to treat due to logarithmically higher amounts of cccDNA and viral load in the liver,” said Cassie Gorsuch, PhD, Chief Scientific Officer of Precision BioSciences.

Consistent pgRNA loss to undetectable in evaluable patients

Among 12 evaluable patients with detectable pregenomic RNA (pgRNA) at baseline, 11 (92%) achieved undetectable pgRNA following treatment with PBGENE-HBV, including all six evaluable patients at 0.4 mg/kg. The remaining patient, dosed with only two administrations at 0.65 mg/kg, experienced a 95% reduction in pgRNA at the time of the data cutoff and will continue to be followed to evaluate pgRNA at go forward visits. In groundbreaking data, the first HBeAg-positive patient treated in the study achieved undetectable pgRNA upon the second administration of PBGENE-HBV at 0.4 mg/kg, declining from 5,750 copies/mL at baseline. As a blood-based biomarker of cccDNA transcription, pgRNA provides evidence of PBGENE-HBV's intended mechanism of cccDNA elimination in the liver.

Durable pgRNA loss to undetectable sustained over time

Among patients who achieved undetectable pgRNA and had at least six months of follow-up, 100% maintained undetectable pgRNA through the data cutoff. The longest observed duration of pgRNA loss now approaches one year, extending the findings reported in May 2026. The sustained absence of detectable pgRNA across patients provides validation evidence and confidence that as designed, PBGENE-HBV is eliminating the cccDNA viral reservoir that drives chronic hepatitis B.

Reproducible liver biopsy evidence of cccDNA elimination at 0.4 mg/kg

Paired liver biopsies from two patients treated with PBGENE-HBV at 0.4 mg/kg, the go forward Phase 2 dose demonstrated approximately 1-log (10-fold) and 1.5-log (35-fold) reductions in cccDNA , respectively across multiple patients. These findings reproduce and extend the initial liver biopsy evidence of cccDNA elimination reported previously at EASL 2026. The concordance between elimination of cccDNA-derived transcripts in liver tissue and loss of circulating pgRNA provides additional evidence supporting PBGENE-HBV's intended mechanism of directly targeting and eliminating the persistent cccDNA viral reservoir responsible for chronic hepatitis B infection.

HBsAg reductions observed across all evaluable patients

All 24 evaluable patients experienced reductions in the secondary biomarker hepatitis B surface antigen (HBsAg) following treatment with PBGENE-HBV at all dose levels. Maximal HBsAg reductions approached 94% at 0.4 mg/kg, with similar antiviral activity observed in patients with and without detectable pgRNA at baseline. HBsAg is produced from both cccDNA and integrated HBV DNA, therefore elimination of cccDNA is expected to result in sustained and durable reduction of HBsAg. The HBsAg findings provide consistent supportive evidence of antiviral activity across a broad patient population and, together with pgRNA loss and liver biopsy findings, further support PBGENE-HBV's intended mechanism of action and the expected go-forward dose.

Well-characterized safety and tolerability profile with no dose-limiting toxicities

PBGENE-HBV demonstrated a well-characterized and manageable safety profile across 59 administrations in 25 patients, with no dose-limiting toxicities (DLTs) observed. The most common adverse events were infusion-related reactions consistent with lipid nanoparticle (LNP) effects, with

onset and resolution generally occurring within 24 hours of infusion. The 0.4mg/kg dose was better tolerated than the 0.65mg/kg, dose, allowing all dose administrations to be delivered and further supporting the go-forward dose in Phase 2. At the expected Phase 2 dose of 0.4 mg/kg, no serious adverse events (SAEs) have been observed. Under the current infusion protocol designed to mitigate potential LNP-related inflammatory responses, no Grade 3 or higher hypotension events have occurred across 15 administrations in eight patients at the Phase 2 dose of 0.4 mg/kg.

Transient, reversible elevations in alanine aminotransferase (ALT) and aspartate aminotransferase (AST) have been observed following PBGENE-HBV administration, consistent with LNP-associated effects. These laboratory abnormalities were asymptomatic, with no associated elevations in bilirubin, cases of Hy's Law or evidence of hepatic synthetic dysfunction across any dose level. At 0.4 mg/kg under the current infusion protocol, one delayed ALT/AST elevation was observed following the third administration, with peak ALT of 306 U/L and AST of 278 U/L. The elevation was transient and resolved to normal with no intervention required and no associated bilirubin changes were observed. Collectively, the antiviral activity and safety profile of PBGENE-HBV support selection of 0.4 mg/kg for further evaluation in Phase 2.

Next Steps and Expected Milestones

“For patients with chronic hepatitis B, the persistent viral reservoir remains the central obstacle to achieving a cure,” said Dr. Man-Fung Yuen, lead investigator of the ELIMINATE-B study. “Reproducing the liver biopsy evidence of cccDNA elimination in a second patient treated at 0.4 mg/kg, together with durable pgRNA loss in patients with longer follow-up, is particularly important because it links the intended mechanism of action, cccDNA elimination, to ongoing antiviral activity. These findings provide a meaningful foundation for Phase 2 testing and for assessing whether directly targeting cccDNA can ultimately enable patients to stop long-term suppressive therapy and achieve cure.”

Based on the Phase 1 ELIMINATE-B findings, Precision has selected 0.4 mg/kg as the go-forward dose for PBGENE-HBV and is initiating a global ELIMINATE-B2 Phase 2 study. The study is designed to evaluate PBGENE-HBV as both a monotherapy and in combination with an antisense oligonucleotide (ASO), to assess its potential to achieve viral and functional cures in patients with chronic hepatitis B.

Precision is finalizing the Phase 2 protocol and onboarding clinical sites globally. The Company expects to initiate dosing in the ELIMINATE-B2 Phase 2 study in the first quarter of 2027, with enrollment planned across multiple continents. Precision anticipates generating initial open-label data from the Phase 2 study in the second half of 2027.

Conference Call Details

The webcast link for Precision’s investor update on Monday, November 9th at 8:00 AM ET can be found **here**. A replay of the presentation will be available on the Company’s Investor Relations Events and Presentations webpage following the event.

About PBGENE-HBV (Hepatitis B cccDNA Elimination Program):

PBGENE-HBV is Precision’s wholly owned *in vivo* gene editing program being evaluated in ELIMINATE-B, a global clinical trial, as a potential viral cure for chronic hepatitis B. PBGENE-HBV is the only clinical stage program that targets and eliminates covalently closed circular DNA (cccDNA), the sole source of viral replication, leading to undetectable sustained loss of pregenomic RNA (pgRNA), the precursor for HBV DNA infectious particles. PBGENE-HBV is the first and only *in vivo* gene elimination approach to prospectively employ repeat lipid nanoparticle (LNP) administration in chronic hepatitis B with the goal of viral cure, as measured by sustained undetectable HBV DNA off nucleos(t)ide therapy. The FDA has

previously provided guidance that undetectable HBV DNA is the validated endpoint for chronic hepatitis B. PBGENE-HBV has been granted Fast Track designation by the FDA.

Further details on the trial can be found on Precision's website and on **clinicaltrials.gov** under identifier NCT06680232.

About Hepatitis B:

Chronic hepatitis B viral (HBV) infection causes inflammation and damage to the liver, leading to increased risk of death from liver cancer or cirrhosis. To date, there is no cure for chronic hepatitis B as current treatments do not result in a viral cure due to persistence of cccDNA in the liver. In patients with chronic hepatitis B, cccDNA, which contains the full-length viral genome, persists long term, produces pgRNA, and is the sole source of new infectious viral particles known as HBV DNA.

Since cccDNA is the only source of infectious particles known as HBV DNA, Precision's clinical development goals are centered around the elimination of cccDNA potentially resulting in a viral cure of chronic hepatitis B. Sustained loss of HBV DNA as a result of cccDNA elimination is the validated FDA approval endpoint for finite curative treatment and goal for the PBGENE-HBV target product profile.

About Precision BioSciences, Inc.

Precision BioSciences, Inc. is a clinical stage gene editing company dedicated to improving life (DTIL) with its novel and proprietary ARCUS® genome editing platform that differs from other technologies in the way it cuts, its smaller size, and its simpler structure. These features are intended for ARCUS nucleases to drive more defined therapeutic outcomes. Using ARCUS, the Company's pipeline is comprised of clinical stage *in vivo* gene editing candidates designed to deliver lasting cures for the broadest range of genetic and infectious diseases where no adequate treatments exist. For more information about Precision BioSciences, please visit **www.precisionbiosciences.com**.

The ARCUS® platform is being used to develop *in vivo* gene editing therapies for gene elimination (removing a viral genome e.g. viral DNA such as in the Company's PBGENE-HBV program), gene excision (removing a large portion of a defective gene by delivering two ARCUS nucleases in a single AAV such as in the Company's PBGENE-DMD program), and gene insertion (inserting DNA into a gene to cause expression or add function).

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, expectations about the goal of PBGENE-HBV of viral cure as measured by sustained undetectable HBV DNA off nucleos(t)ide therapy; expectations around durable pgRNA loss in both HBeAg positive and negative patients being treated with PBGENE-HBV supporting a path towards viral cure; the potential and expected safety, efficacy, durability, and benefit of PBGENE-HBV, including the durability of pgRNA loss; the ability to advance to a global Phase 2 ELIMINATE-B trial and timing and progress of the study; belief that elimination of cccDNA could result in a viral cure of chronic hepatitis B; and the expectation that sustained loss of HBV DNA is a validated endpoint for the FDA and highly relevant for PBGENE-HBV. In some cases, you can identify forward-looking statements by terms such as

“aim,” “anticipate,” “approach,” “belief,” “believe,” “contemplate,” “could,” “design,” “designed,” “estimate,” “expect,” “goal,” “intend,” “look,” “may,” “mission,” “plan,” “possible,” “potential,” “predict,” “project,” “pursue,” “should,” “strive,” “suggest,” “target,” “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.

All forward-looking statements speak only as of the date of this press release 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.

Investor and Media Contact:
Naresh Tanna
Chief Financial Officer
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Durable pgRNA loss in both HBeAg positive and negative patients treated with PBGENE-HBV supports path towards viral cure

Man-Fung Yuen[1], Anca Streinu-Cercel[2,3], Vlad Andrei Ichim[4,5], Emily B. Harrison[5], James M. Harris[6], Keely Dulmage[5], Andrew Van Cott[5], Abhishek Chandiramani[5], Neil Leatherbury[5], Janel Lape[5], Peter Balfe[6], Jeff Smith[5], Cassandra L. Gorsuch[5], Stanley Frankel[5], Oby Sogbetun[5], Jane A. McKeating[6], Mark Sulkowski[5,7] Edward Gane[8]

[1]Queen Mary Hospital, The University of Hong Kong, Hong Kong, [2]Carol Davila University of Medicine and Pharmacy, Bucharest, Romania, [3]National Institute for Infectious Disease 'Prof. Dr. Matei Balș', Bucharest, Romania, [4]"Luliu Hațieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania, [5]Cluj Emergency County Hospital, Cluj-Napoca, Romania[8]Precision BioSciences Inc., [6]Nuffield Department of Medicine, University of Oxford,[7]Division of Infectious Diseases Johns Hopkins Bayview Medical Center Johns Hopkins University & Medicine, [8]New Zealand Clinical Research

Background and Aims: The root cause of chronic hepatitis B (CHB) infection is covalently closed circular DNA (cccDNA). cccDNA is the sole source of pregenomic RNA (pgRNA), the precursor molecule to infectious particles, HBV DNA. The persistence of cccDNA and pgRNA upstream of HBV DNA suppression by nucleoside analogue (NA) therapy drives ongoing risk of disease progression and hepatocellular carcinoma. Therefore, cccDNA elimination is key for chronic hepatitis B viral cure. PBGENE-HBV, an LNP containing ARCUS nuclease mRNA, is designed to eliminate cccDNA. ELIMINATE-B is a Phase 1b study evaluating the safety and antiviral activity of PBGENE-HBV in HBeAg-negative patients and now expanded to include HBeAg-positive patients — an important population with notably higher levels of cccDNA and pgRNA.

Method: Multiple ascending doses of PBGENE-HBV were tested in NA-treated, CHB patients, with repeat infusions dosed at ≥4-week intervals. The primary endpoint was safety, defined by the frequency and severity of dose limiting toxicities (DLTs). Other endpoints included assessment of antiviral activity from blood and liver biopsy.

Results: After PBGENE-HBV treatment, elimination and inactivation of cccDNA was confirmed in liver biopsies, and durable loss of pgRNA was demonstrated in serial blood samples (repeat measurements below the lower limit of detection, 5 copies/mL). In the first HBeAg-positive patient, treated at 0.4 mg/kg, pgRNA decreased from 5,750 copies/mL at baseline to undetectable. To date, of 11 HBeAg-negative patients with detectable pgRNA at baseline, 10 currently have undetectable pgRNA after PBGENE-HBV treatment, with 5-month average durability and ongoing follow-up. The first 6 participants with pgRNA loss have now surpassed the important 6-month durability milestone since their initial undetectable measurement. Loss of pgRNA occurred after one or two administrations at dose levels ranging from 0.4 to 0.8 mg/kg. Transcriptomic analysis from liver biopsies showed a 10-fold (1-log) reduction in cccDNA derived transcripts after 2 administrations at 0.4 mg/kg in one patient, with additional biopsy data pending.

PBGENE-HBV was well-tolerated with no DLTs across 58 doses in 24 subjects, from 0.2-0.8 mg/kg. Infusion-related reactions were transient and manageable supporting a wide therapeutic window.

Conclusion: Elimination and inactivation of cccDNA by PBGENE-HBV was confirmed by liver biopsies as well as durable loss of pgRNA in the blood. In an HBeAg positive patient, loss of pgRNA after treatment with PBGENE-HBV at 0.4 mg/kg shows elimination of cccDNA is not limited to patients with lower cccDNA burden. Safety and efficacy data support ongoing development of PBGENE-HBV as a potentially curative regimen for chronic hepatitis B.

Precision BioSciences Announces Positive DSMB Safety Review Supporting Additional PBGENE-DMD Dosing in Phase 1/2 FUNCTION-DMD Study

- First patient completed observation period following PBGENE-DMD treatment with no reported safety concerns -

- Independent Data Safety Monitoring Board endorses proceeding with dosing additional patients -

DURHAM, N.C., November 5, 2026 -- Precision BioSciences, Inc. (Nasdaq: DTIL), a clinical stage gene editing company utilizing its novel proprietary ARCUS® platform to develop *in vivo* gene editing therapies for high unmet need diseases, today announced that the independent Data Safety Monitoring Board (DSMB) has completed its review of safety data from the first patient treated with PBGENE-DMD in the Phase 1/2 FUNCTION-DMD clinical trial for Duchenne muscular dystrophy (DMD). No serious adverse events or adverse events of special interest were reported in the first patient through week eight. Following completion of the patient's initial eight-week safety observation period, the DSMB identified no safety concerns and endorsed proceeding with dosing additional patients in Part 1 of the study.

“Successful completion of the initial safety assessment and DSMB authorization to proceed with treatment of additional patients represents an important milestone for the FUNCTION-DMD study,” said Dr. Sam Collins, MBBS, Ph.D., Senior Vice President, DMD Clinical Development at Precision BioSciences. “This initial eight-week period is key in monitoring potential early safety events known to be associated with all AAV-based therapies, and we are encouraged by the DSMB’s agreement that the safety results in the first patient support completing enrollment in Part 1 of the study. We remain diligent about safety monitoring as the trial progresses and are grateful to the patients, families, and clinical teams whose commitment is making it possible to evaluate this novel gene editing approach for Duchenne muscular dystrophy.”

“We believe PBGENE-DMD has the potential to fundamentally change the treatment paradigm by addressing the underlying genetic cause of DMD. Unlike truncated microdystrophin treatments, PBGENE-DMD is designed to utilize the body’s native dystrophin gene, excising the frequently mutated hotspot region to restore near full-length, functional dystrophin,” said Cassie Gorsuch, PhD, Chief Scientific Officer of Precision BioSciences. “We remain committed to generating the clinical evidence needed to evaluate this novel approach for patients and families living with DMD, and look forward to providing safety and efficacy updates from the FUNCTION-DMD study throughout 2027.”

About FUNCTION-DMD Trial

The Phase 1/2 FUNCTION-DMD study is enrolling ambulatory DMD patients between the ages of 2 and 7 with mutations between exons 45 and 55 representing up to 60% of boys with DMD. The objective of the FUNCTION-DMD study is to evaluate safety, tolerability, and efficacy, including dystrophin protein expression and functional outcomes in patients living with DMD. For more information about this clinical trial and contact information, please visit **www.clinicaltrials.gov** and search for NCT07429240.

About PBGENE-DMD, A Muscle-Targeted Excision Program

PBGENE-DMD is Precision’s clinical program for the treatment of DMD. DMD is a genetic disease caused by mutations in the dystrophin gene that prevent production of the dystrophin protein and affects approximately 15,000 patients in the U.S. alone. There are currently no approved therapies that can drive durable and significant functional improvements over time. PBGENE-DMD is designed to improve

function by employing two complementary ARCUS nucleases delivered in a single AAV to excise exons 45-55 of the dystrophin gene. The aim of this approach is to restore a near full-length functional dystrophin protein native to the body as opposed to foreign and synthetic, truncated microdystrophin approaches which have led to limited functional benefit. The Phase 1/2 FUNCTION-DMD study is enrolling ambulatory DMD patients with mutations between exons 45 and 55 impacting up to 60% of boys with DMD. The clinical trial employs an appropriate immune modulation regimen and safety monitoring program to treat ambulatory patients at world-class specialized DMD clinical sites.

PBGENE-DMD was granted Orphan Drug Designation by the FDA in July 2025. PBGENE-DMD has received Rare Pediatric Disease Designation from the FDA for the treatment of DMD, and therefore PBGENE-DMD may be eligible for a Priority Review Voucher (PRV). PBGENE-DMD received Fast Track designation from the FDA in February 2026.

About Precision BioSciences, Inc.

Precision BioSciences, Inc. is a clinical stage gene editing company dedicated to improving life (DTIL) with its novel and proprietary ARCUS® genome editing platform that differs from other technologies in the way it cuts, its smaller size, and its simpler structure. These features are intended for ARCUS nucleases to drive more defined therapeutic outcomes. Using ARCUS, the Company's pipeline is comprised of clinical stage in vivo gene editing candidates designed to deliver lasting cures for the broadest range of genetic and infectious diseases where no adequate treatments exist. For more information about Precision BioSciences, please visit **www.precisionbiosciences.com**.

The ARCUS® platform is being used to develop *in vivo* gene editing therapies for sophisticated gene edits, including gene elimination (removing a viral genome e.g. viral DNA such as in the Company's PBGENE-HBV program), and excision (removing a large portion of a defective gene by delivering two ARCUS nucleases in a single AAV such as in the Company's PBGENE-DMD program) and gene insertion (inserting DNA into a gene to cause expression or add function).

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, expectations about expectations around timing of site activation, patient enrollment and data releases of PBGENE-DMD and the FUNCTION-DMD trial; the potential of PBGENE-DMD to fundamentally change the treatment paradigm by addressing the underlying genetic cause of DMD; translation of results in preclinical studies of PBGENE-DMD to clinical studies in humans; the design of PBGENE-DMD to potentially provide durable functional muscle improvement for DMD patients with mutations in exons 45-55 impacting up to 60% of patients with DMD; and the use of an appropriate immune modulation regimen. In some cases, you can identify forward-looking statements by terms such as "aim," "anticipate," "approach," "belief", "believe," "contemplate," "could," "design," "designed," "estimate," "expect," "goal," "intend," "look," "may," "mission," "plan," "possible," "potential," "predict," "project," "pursue," "should," "strive," "suggest," "target," "will," "would," or the negative thereof and similar words and expressions.

Forward-looking statements are based on management's current expectations, beliefs, and assumptions and on information currently available to us. These statements are neither promises nor guarantees, and involve a number of known and unknown risks, uncertainties and assumptions, and actual results

may differ materially from those expressed or implied in the forward-looking statements due to various important factors, including, but not limited to, our ability to become profitable; our ability to procure sufficient funding to advance our programs; risks associated with our capital requirements, anticipated cash runway, requirements under our current debt instruments and effects of restrictions thereunder, including our ability to raise additional capital due to market conditions and/or our market capitalization; our operating expenses and our ability to predict what those expenses will be; our limited operating history; the progression and success of our programs and product candidates in which we expend our resources; our limited ability or inability to assess the safety and efficacy of our product candidates; the risk that other genome-editing technologies may provide significant advantages over our ARCUS technology; our dependence on our ARCUS technology; the initiation, cost, timing, progress, achievement of milestones and results of research and development activities and preclinical and clinical studies, including clinical trial and investigational new drug applications; public perception about genome editing technology and its applications; competition in the genome editing, biopharmaceutical, and biotechnology fields; our or our collaborators' or other licensees' ability to identify, develop and commercialize product candidates; pending and potential product liability lawsuits and penalties against us or our collaborators or other licensees related to our technology and our product candidates; the U.S. and foreign regulatory landscape applicable to our and our collaborators' or other licensees' development of product candidates; our or our collaborators' or other licensees' ability to advance product candidates into, and successfully design, implement and complete, clinical trials; potential manufacturing problems associated with the development or commercialization of any of our product candidates; delays or difficulties in our and our collaborators' and other licensees' ability to enroll patients; 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.**

All forward-looking statements speak only as of the date of this press release 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.

Investor and Media Contact:
Naresh Tanna
Chief Financial Officer
naresh.tanna@precisionbiosciences.com

AASLD 2026 Investor Update:

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



Confidenti

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 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 expect 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 cor 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 an 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 cc 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 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 p 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 furth 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 PBGE 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; and 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 bio readouts and continuing to Part 2 of the study (n=12, concurrent enrollment). In some cases, you can identify forward-looking statements by terms such as "aim," "anticipate," "approach," "belief", "believe," "contemplate," "could," "design," "designe "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 expr

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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 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 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 cor 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 whic offer, solicitation or sale would be unlawful prior to registration or qualification under the securities laws of any such state or other jurisdiction.

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Setting the Stage for Viral Cure





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

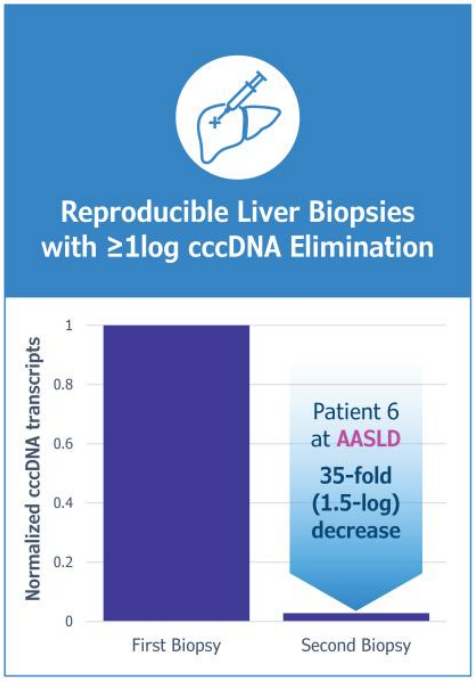
Since May 2026 at EASL…

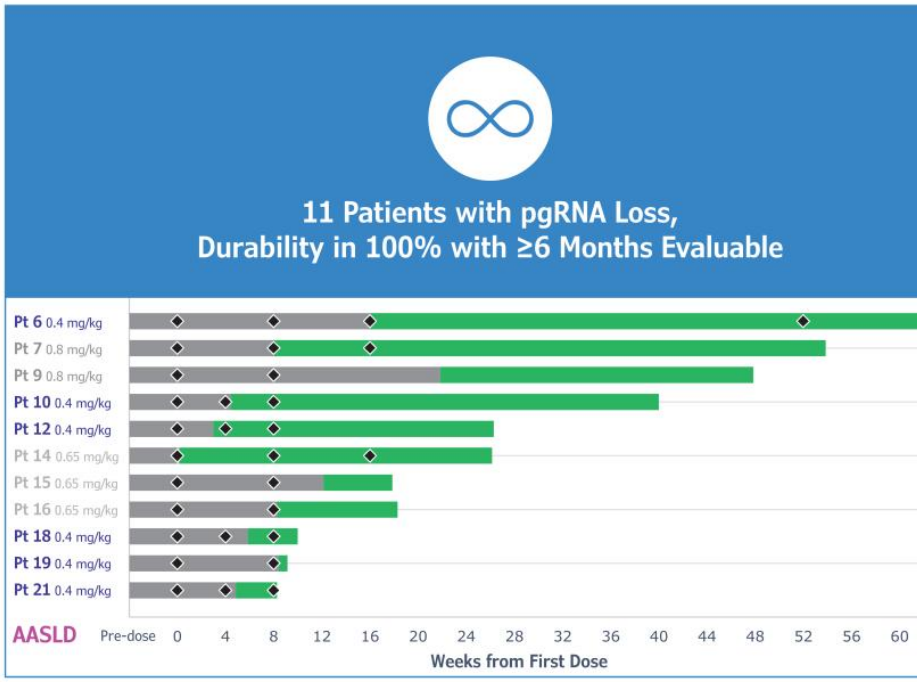
- ✓ Treatment Effect in 100% of Patients
  - 〉 38 administrations, 16 patients
- ✓ From the Liver to the Blood → Advent of pgRNA blood biomarker
  - 〉 100% of patients with durable pgRNA undetectable
- ✓ Evidence of Direct Elimination of cccDNA in Biopsy
  - 〉 1log reduction
- ✓ Safety Characterization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

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

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



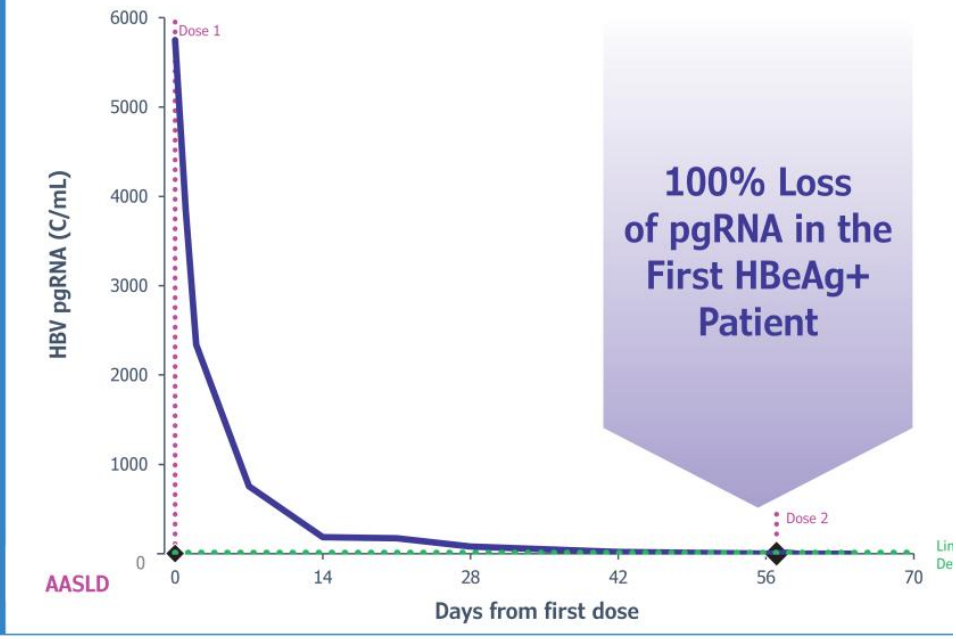


AASLD, American Association for the Study of Liver Diseases; cccDNA, covalently closed circular DNA; pgRNA, pregenomic RNA; Pt, patient.
Data cutoff: September 28, 2026.

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

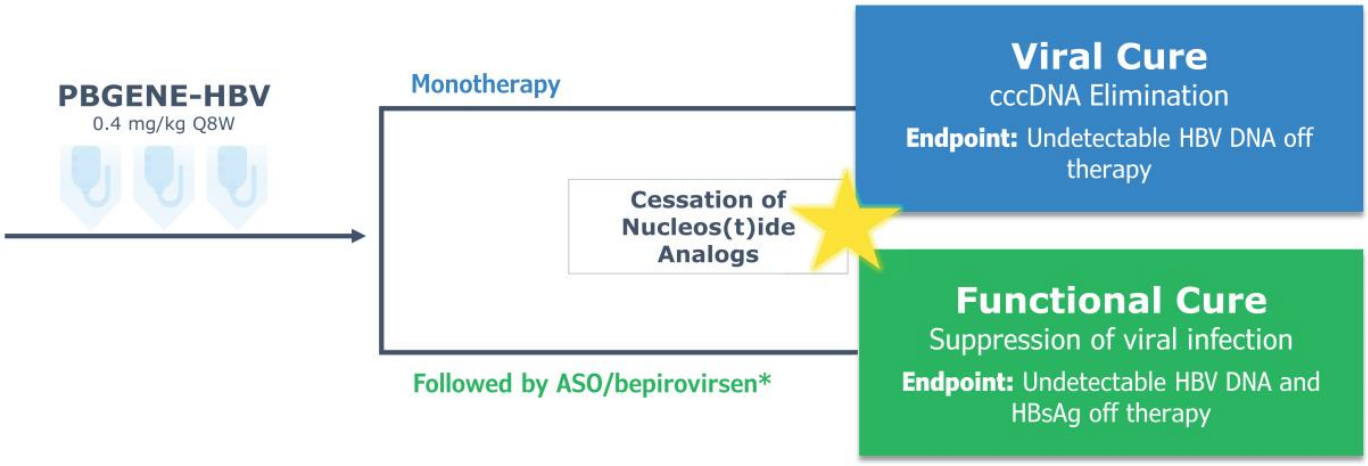
Groundbreaking

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.

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



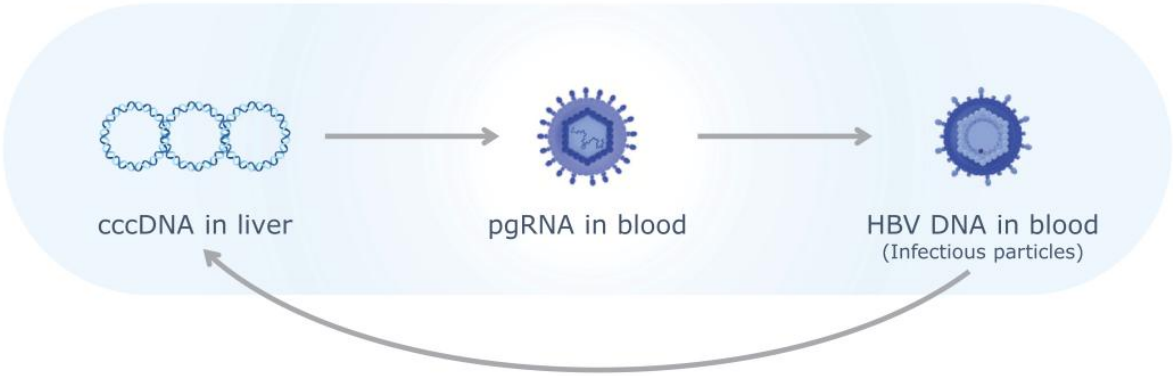
*Japan MHLW Approval Date; August 24, 2026. US FDA Approval Date; October 20, 2026.
ASO, antisense oligonucleotide; cccDNA, covalently closed circular DNA; FDA, US Food and Drug Administration; HBsAg, hepatitis B surface antigen;
HBV, hepatitis B virus; MHLW, Ministry of Health, Labour and Welfare; Q8W,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

Endpoints

PBGENE-HBV: Eliminating cccDNA to Drive Viral Cure

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

The Trinity of HBV Infection
cccDNA makes pgRNA makes HBV DNA

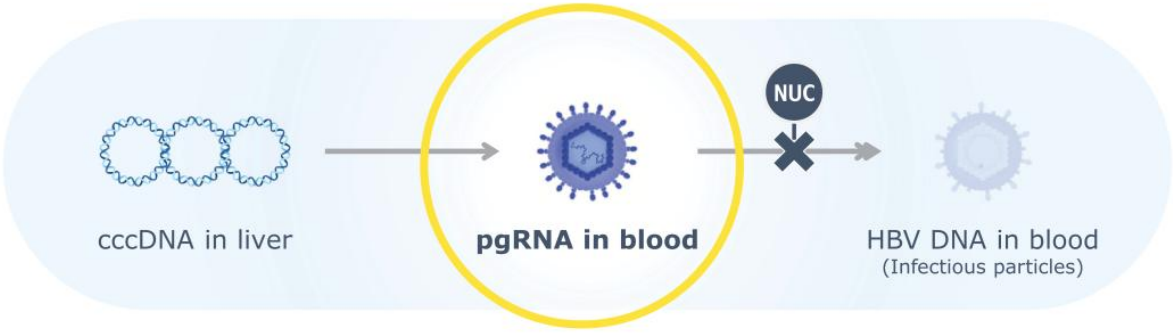


Therefore, cccDNA Elimination is the Only Path to Viral Cure

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

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

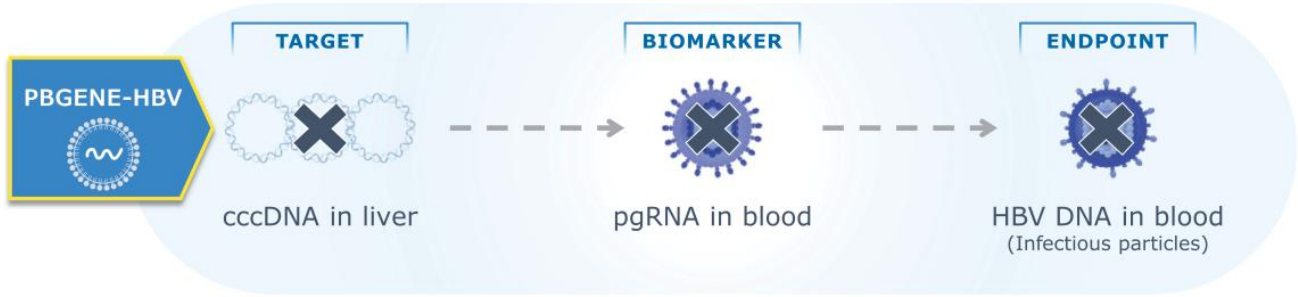
NUCs prevent conversion of pgRNA to HBV DNA



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 Chang 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 Level



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

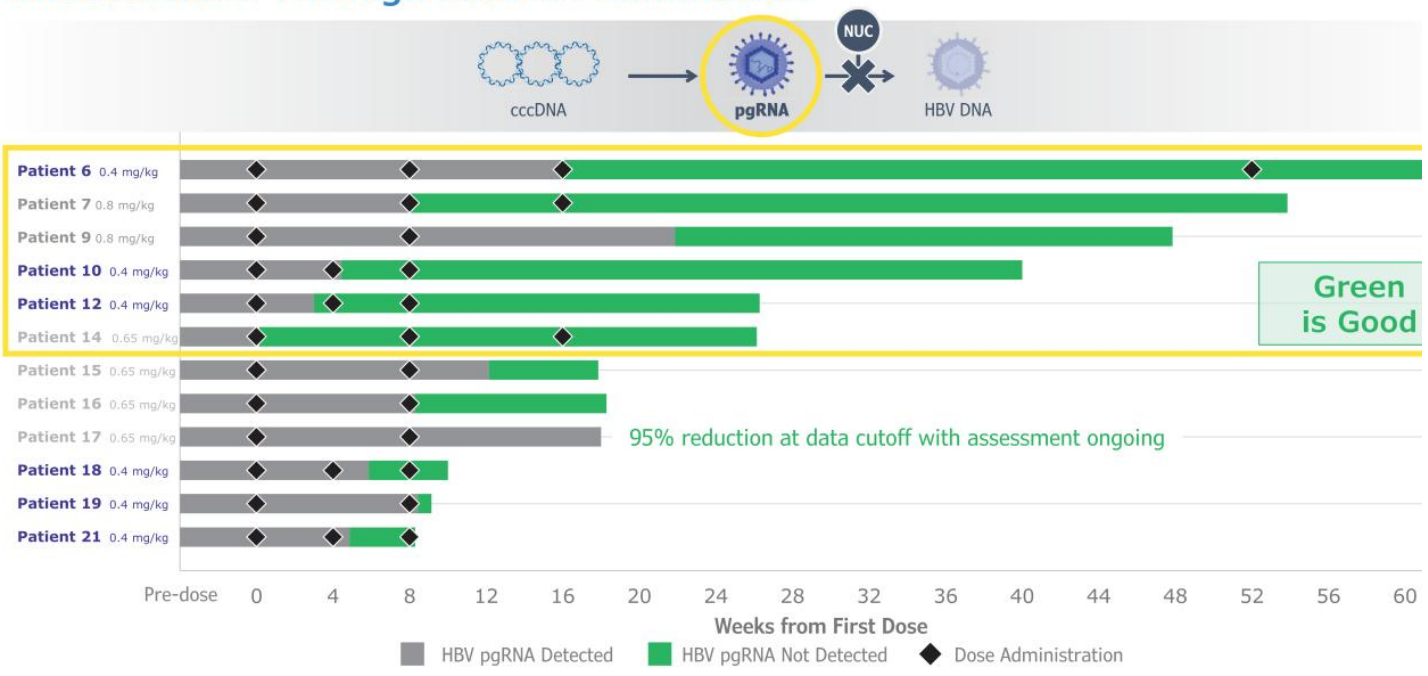
Extensive Experience with the Go-Forward Dose 0.4 mg/kg PBGENE-HBV



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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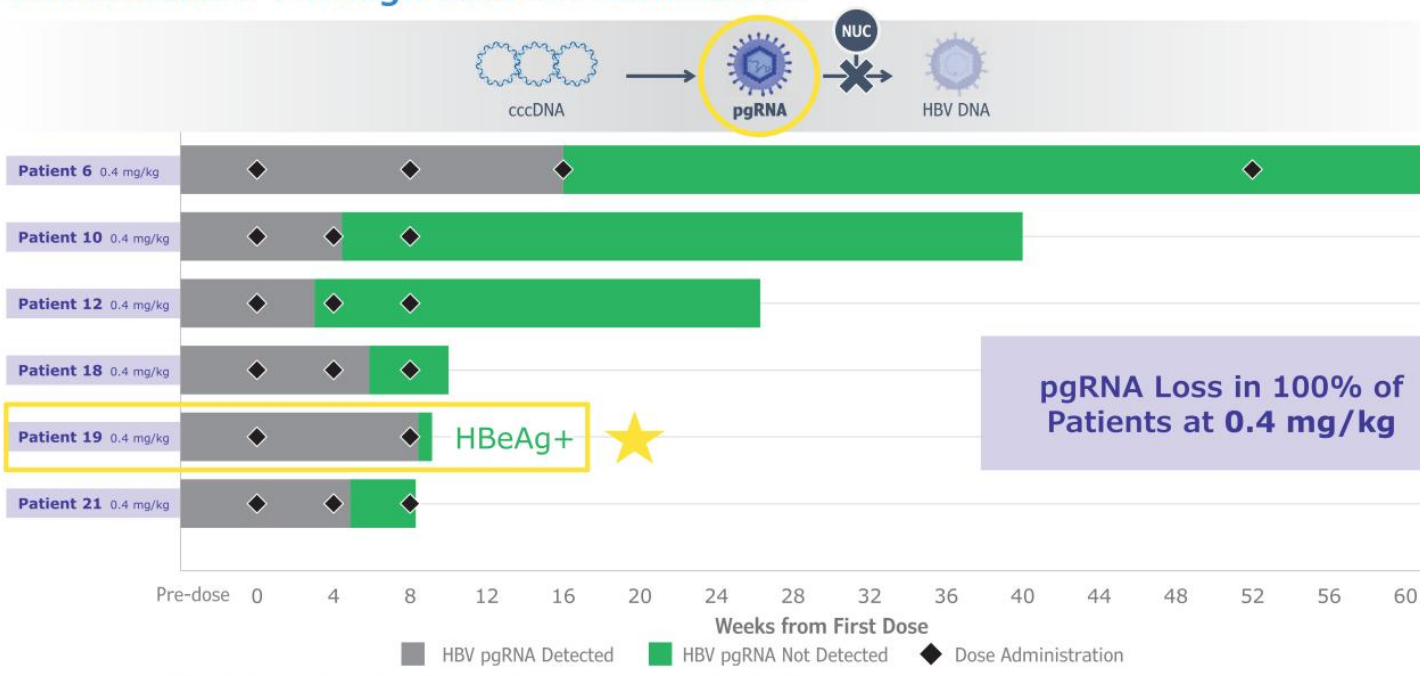

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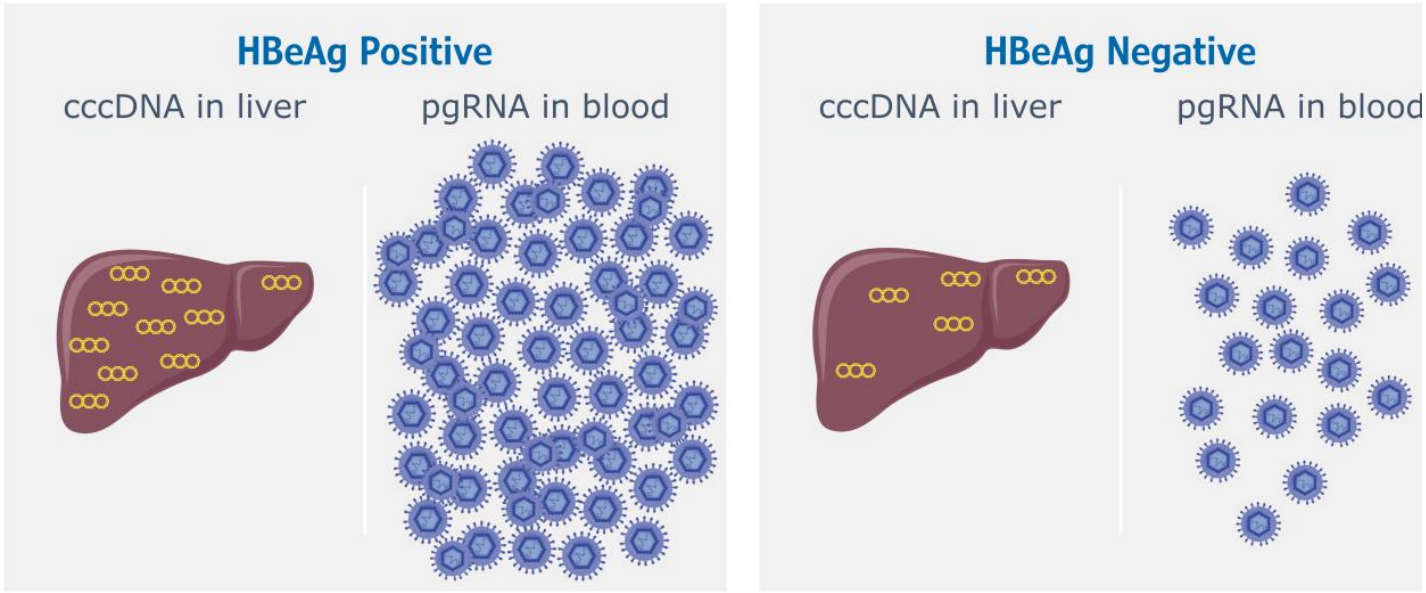
AASLD, American Association for the Study of Liver Diseases; cccDNA, covalently closed circular DNA; HBeAg, hepatitis B e antigen; HBV, hepatitis B virus; NUC, nucleos(t)ide analog; pgRNA, pregenomic RNA.
Data cutoff: September 28, 2026.

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.

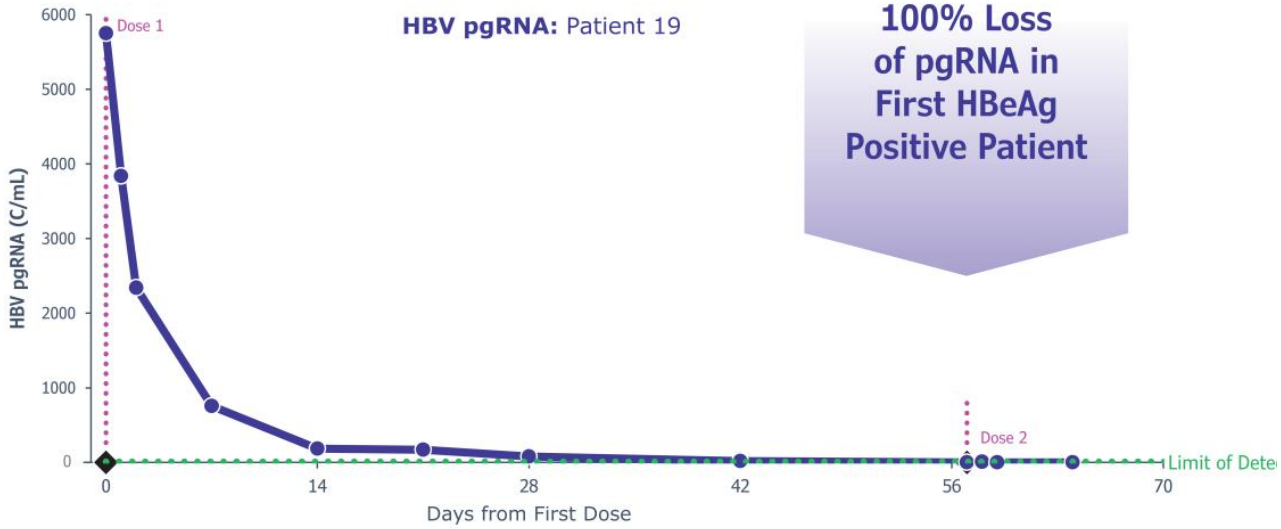
— Werle-Lapostolle et al, 2004, *Gastroenterology*

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



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



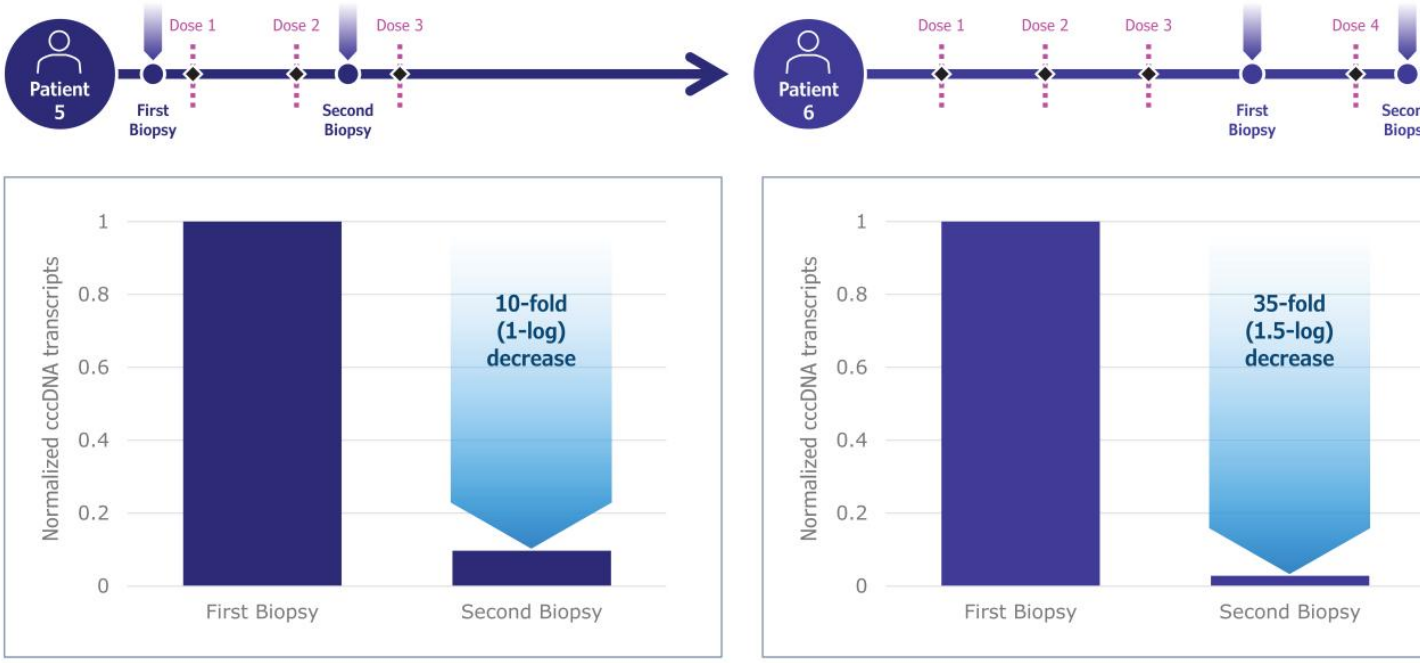
pgRNA loss in first HBeAg positive patient with 3.7-log baseline pgRNA demonstrates PBGENE-HBV potently eliminated cccDNA in the highest viral burden population

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/



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

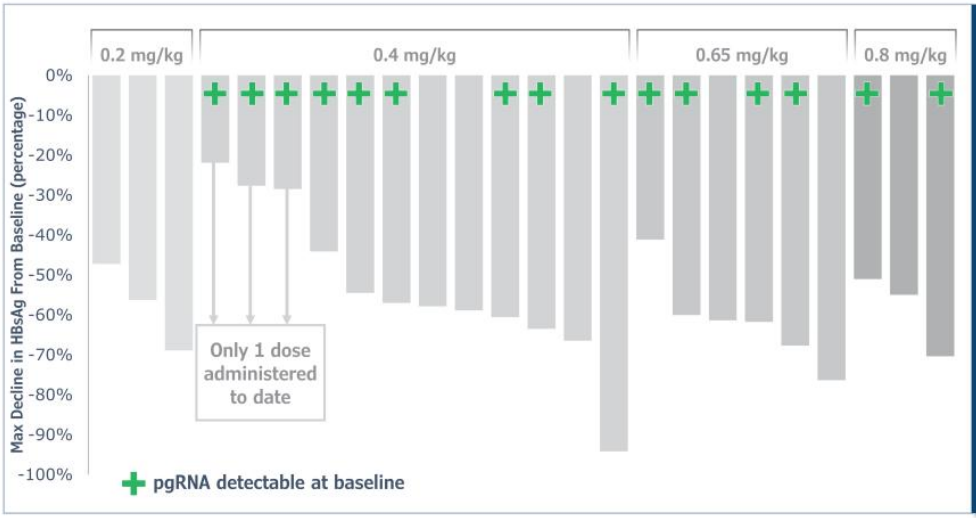
Literature

Secondary Biomarker HBsAg:

Every ELIMINATE-B Patient Experienced Treatment Effect with PBGENE-HBV

(n=24 evaluable at time of data cutoff)

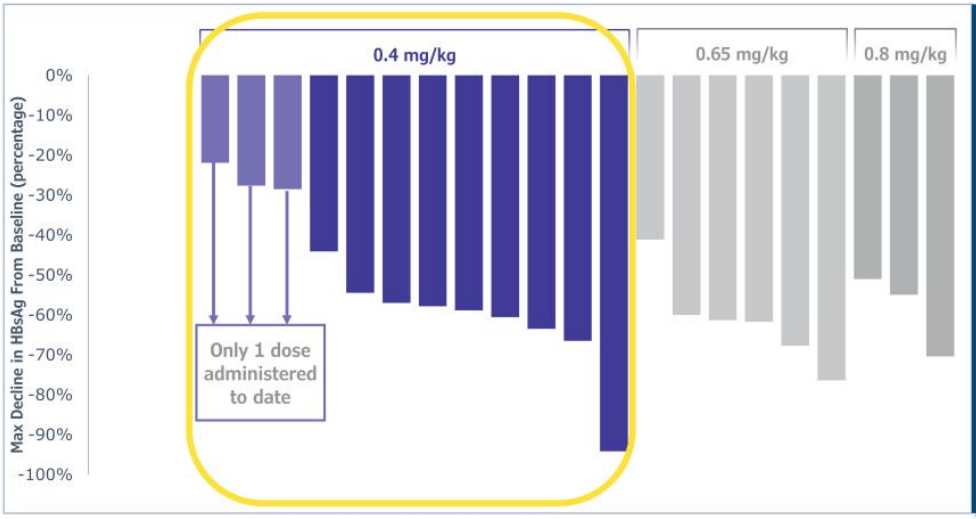
NEW AASLD DATA: Substantial HBsAg Declines Indicative of cccDNA Elimination in All Patients With or Without pgRNA



Significant maximal HBs/ declines across all patien (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 & Durabilit

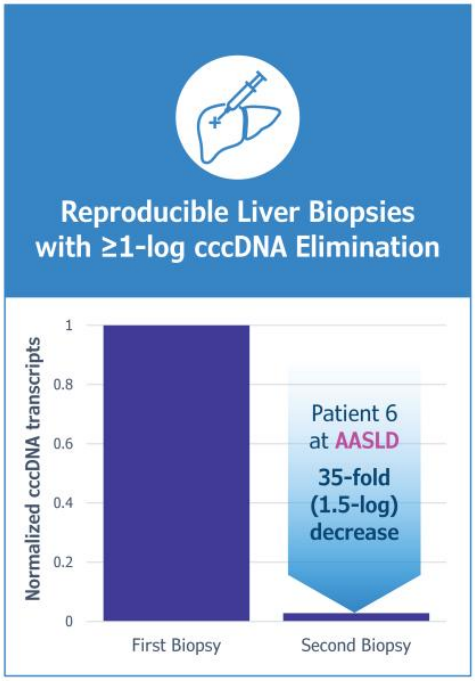


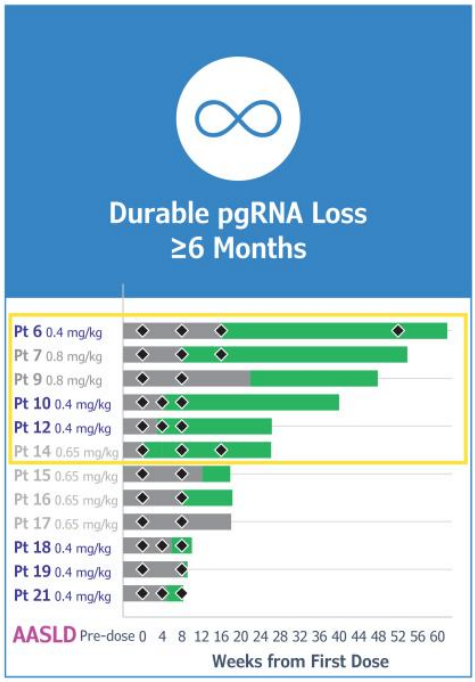
Maximal HBsAg declines observed as low as 0.4 mg/kg

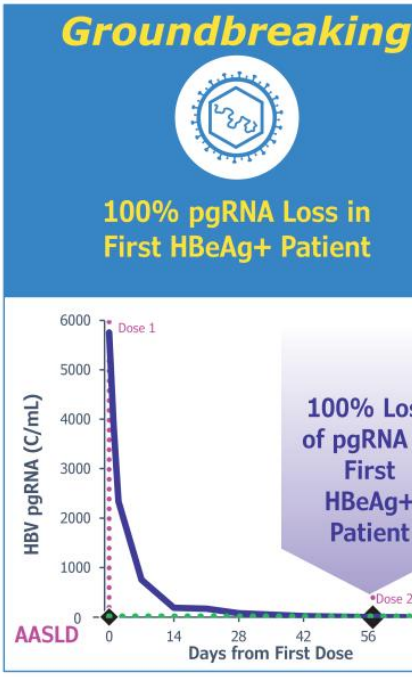
Durability

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.

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







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.

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/

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 proto n=8 of 13
Grade 3	Hypotension	4	3[b]	-
Grade 3 and 4	ALT/AST lab abnormality	4	2	1
SAEs		-	-	-

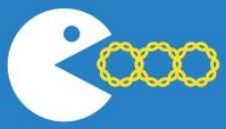
- **Current Infusion Protocol:** No Grade ≥3 hypotension has been observed at 0.4 mg/kg since implementing a simple modified infusion protocol to blunt LNP-related inflammatory response (5-hour infusion, 20 mg dexamethasone, prophylactic saline infusion)
- **Current Infusion Protocol:** One delayed ALT/AST 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 biliru 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.

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



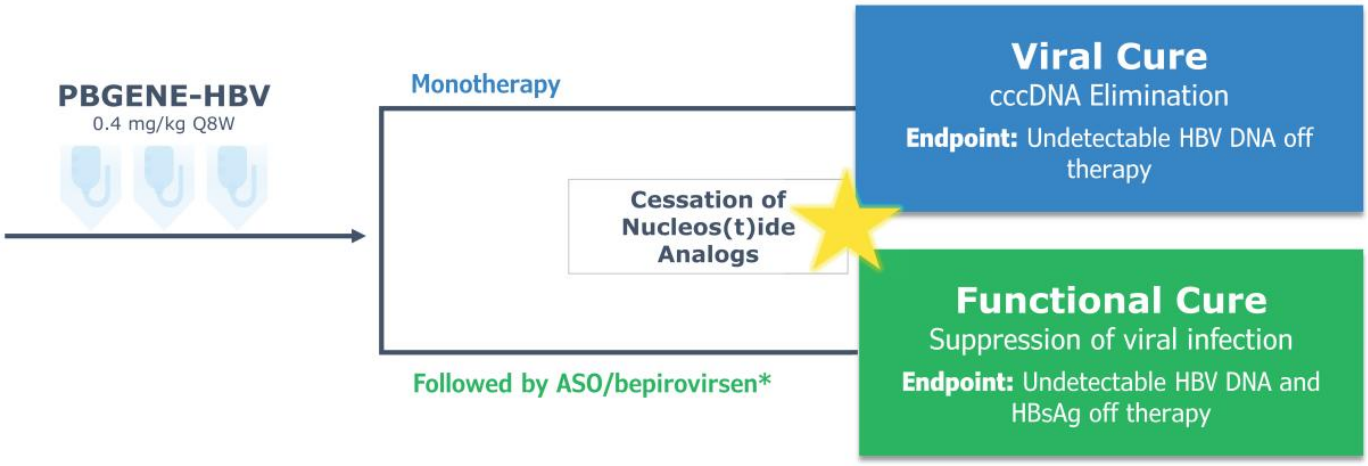
cccDNA, covalently closed circular DNA; HBeAg, hepatitis B e antigen; HBV, hepatitis B virus; pgRNA, pregenomic RNA; SAE, serious adverse event.

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.

View FDA Guida

Details

PBGENE-HBV Goal to Improve Upon Cure Rates in CHB

Monotherapy

+ Combination with ASO

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

Functional Cure

Suppression of viral infection

FDA Endpoint: Undetectable HBV DNA and HBsA 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.

Bepi MOA

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

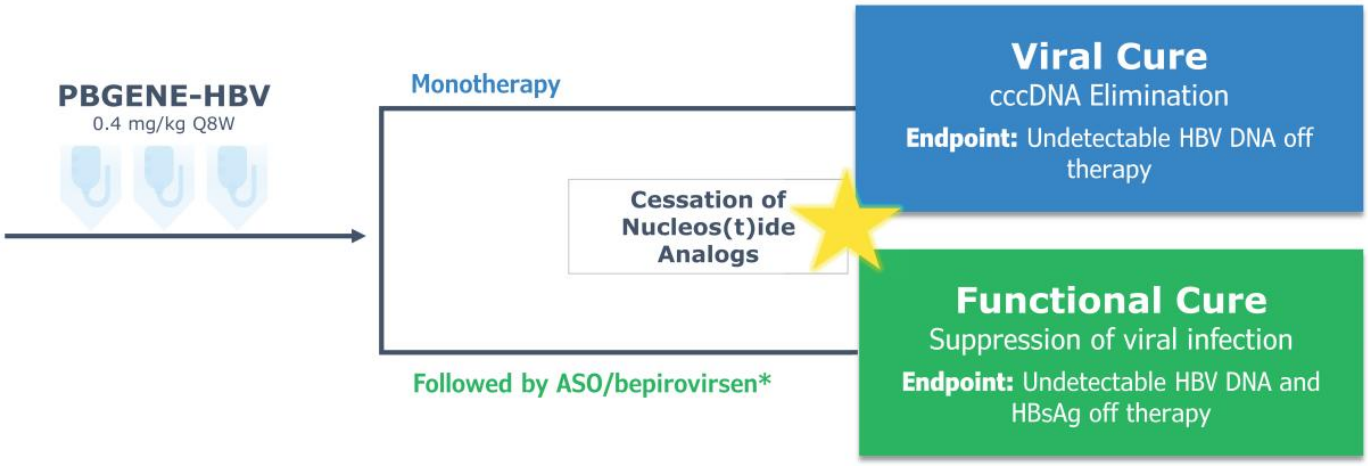
- Single dose of PBGENE-HBV
- Finite course of treatment with ASO (biosimilar to bepirivirse
- Off-treatment monitoring period with durability of the effect measured at the end of the study

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



View FDA Guida

*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.

FUNCTION-DMD

Clinical Momentum Continues with PBGENE-DMD

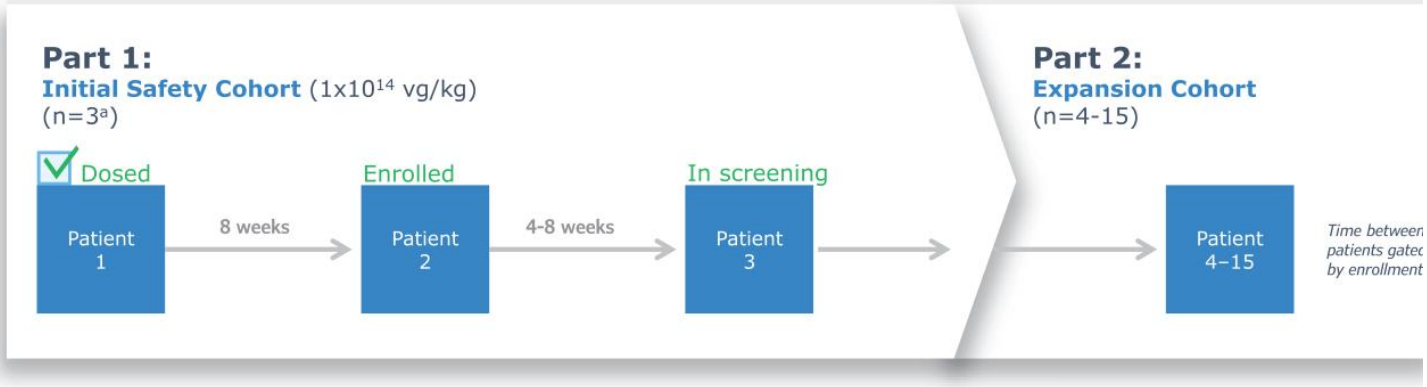


Phase 1/2 Study Designed for Safety and Speed

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.

DMD MOA

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



Precision BioSciences Has Numerous Upcoming Catalysts in Next 12 Month

Key Expected Inflection Points Through 2027

		eliminate B2	functionDMD
		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

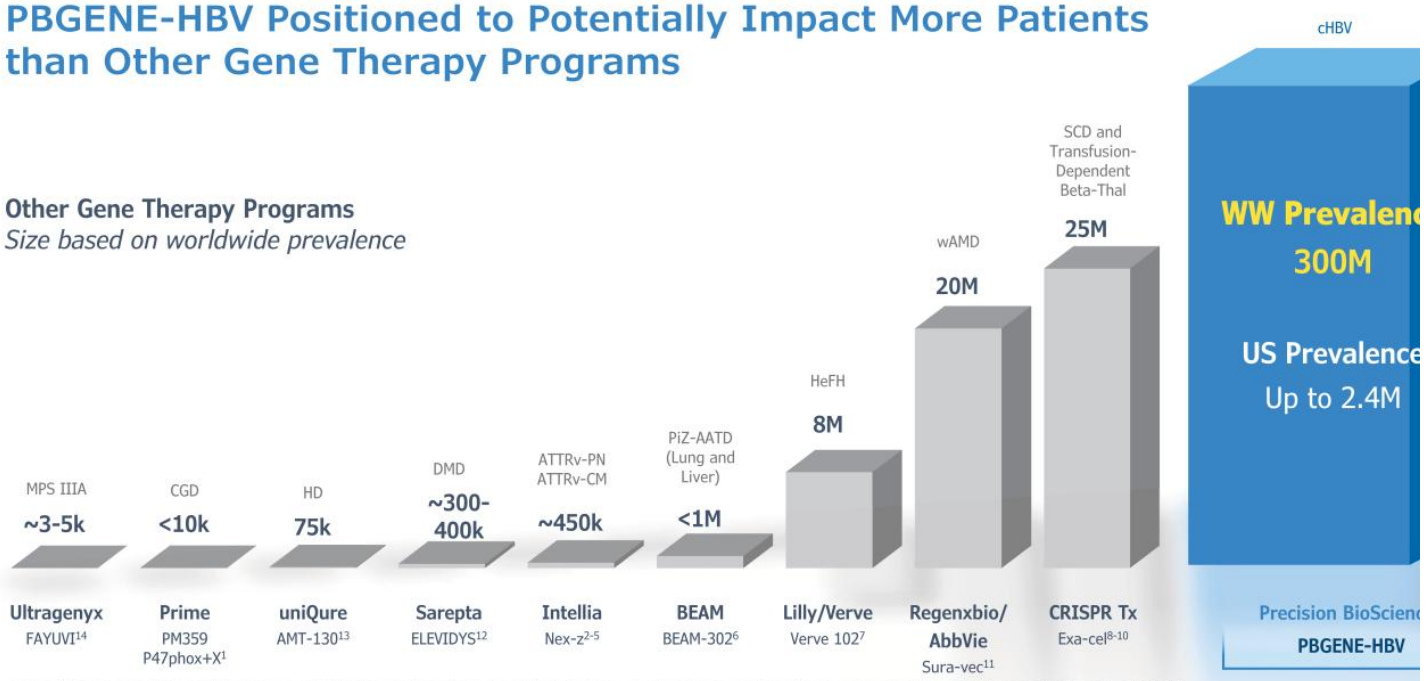
AASLD, American Association for the Study of Liver Diseases; AAV, adeno-associated virus; DMD, Duchenne muscular dystrophy; H1, first half; H2, second half; HBV, hepatitis B virus; LNP, lipid nanoparticle; Ph2, phase 2; Q1, first quarter; Q4, fourth quarter.

HBV Steps

PRECISION
BIOSCIENCES

Appendix

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



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.

Main

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]

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

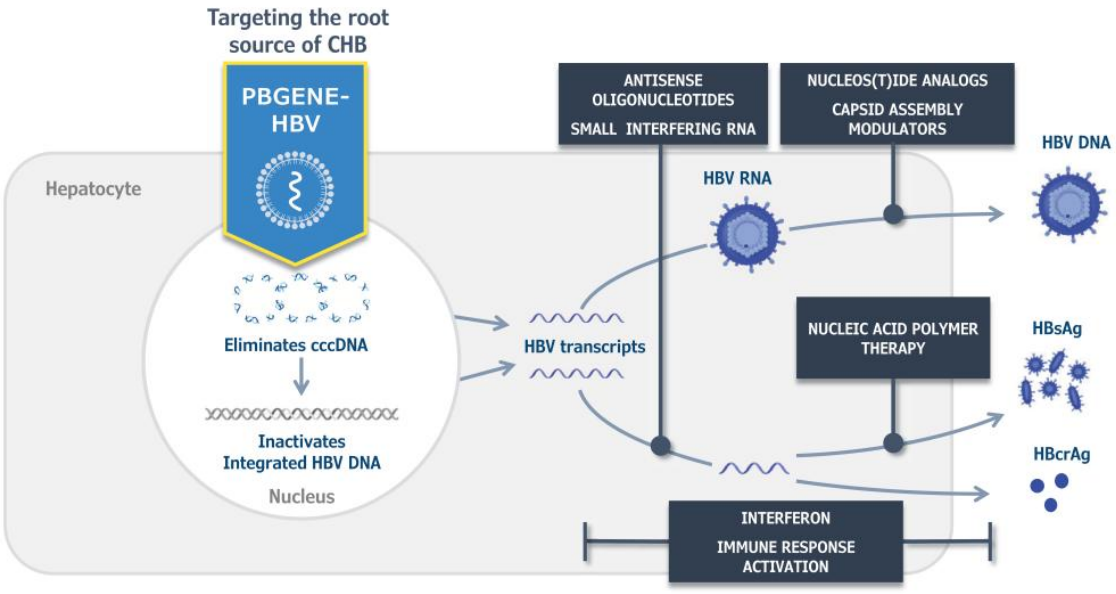
Founded in 2006 and **dedicated to developing novel therapeutics designed with goal of curing difficult-to-treat diseases with high unmet need,** including infectious and **rare genetic diseases.**

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 editi
- >75 patents issued covering ARCUS and in vivo gene editing

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

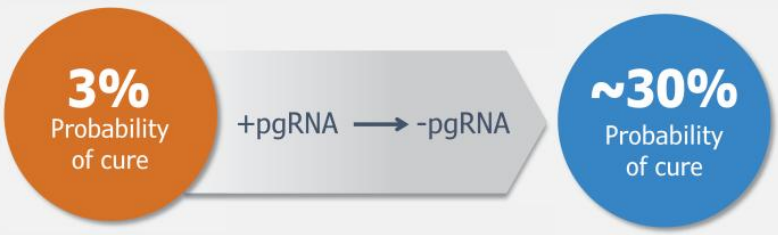


cccDNA, covalently closed circular DNA; CHB, chronic hepatitis B; HBcrAg, hepatitis B core-related antigen; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus.
Adapted from https://www.natap.org/2017/HBV/090517_02.htm

"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 prote load as it primarily comes from nonreplicating, integrated DNA in HBeAg negative patients[1,2]

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

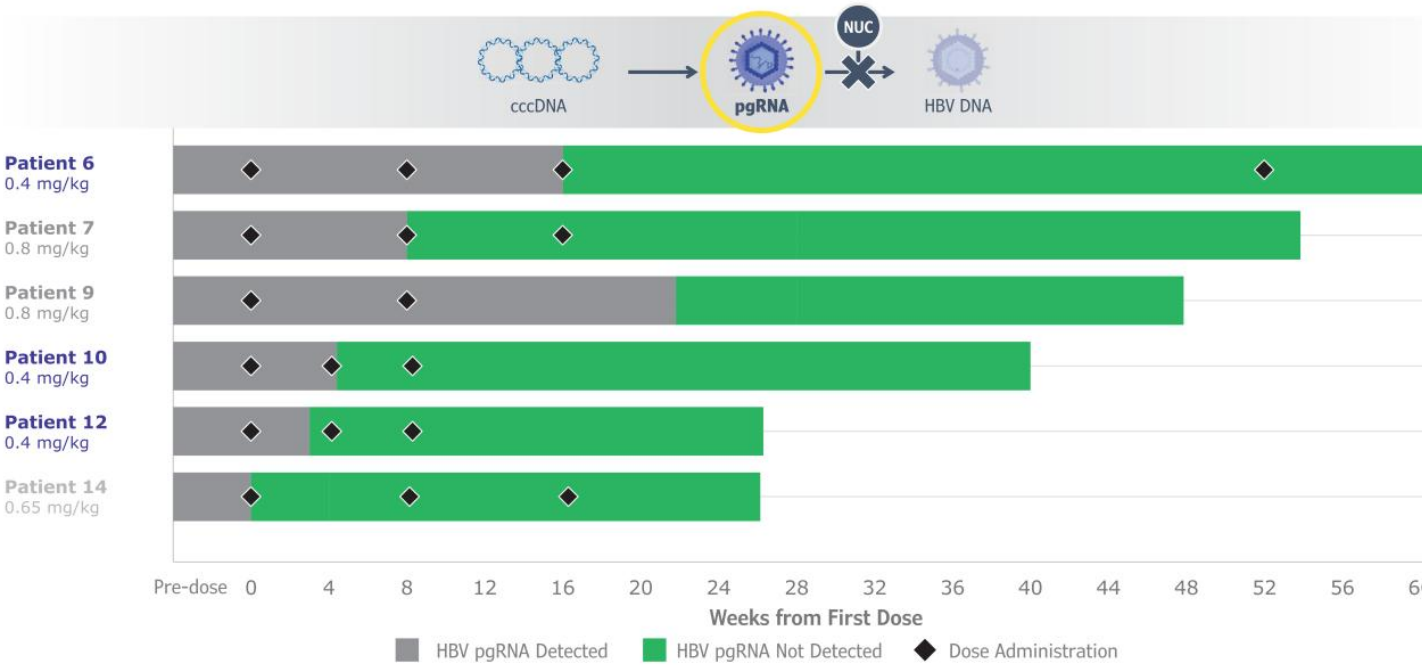
- Melbourne HBV-STOP trial (n=65)[3]
- DARING-B study (n=57)[4]
- Compilation of 23 cohort studies (n=2043)[5]

pgRNA outperforms other biomarkers (eg, HBsAg) predicting the effective and safe withdrawal of NA

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 Los



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

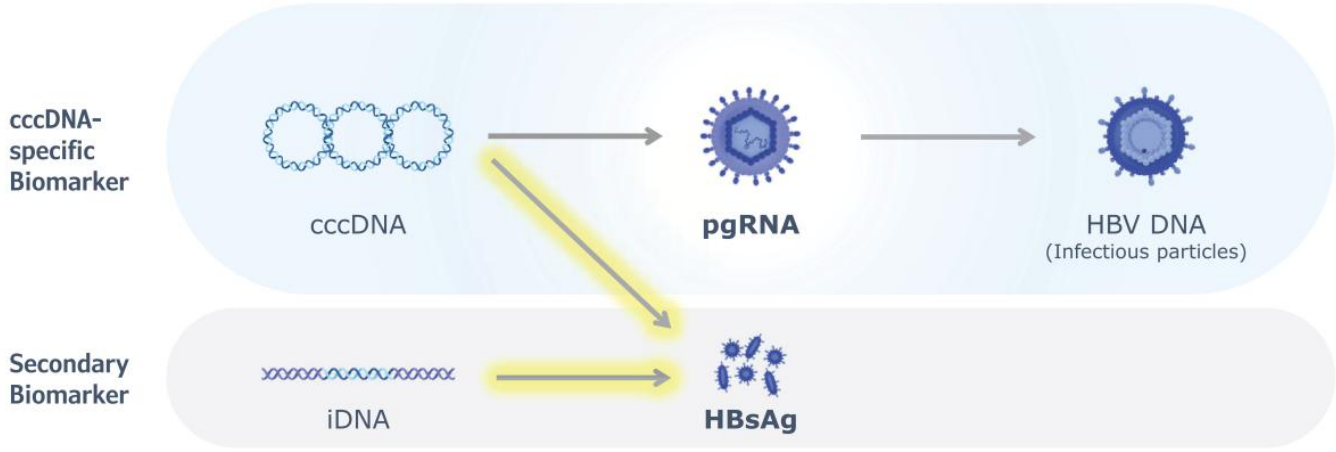
- Lower cccDNA in the liver is associated with significantly better odds of a sustained virologic response (adjusted odds ratio for log cccDNA was 5.3 [95% CI: 1.5-18.2, *P* = .009])
  - Sung et al, 2005, *Gastroenterology*
- Functionally cured patients show lower cccDNA and fewer HBsAg-positive hepatocytes than uncure 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

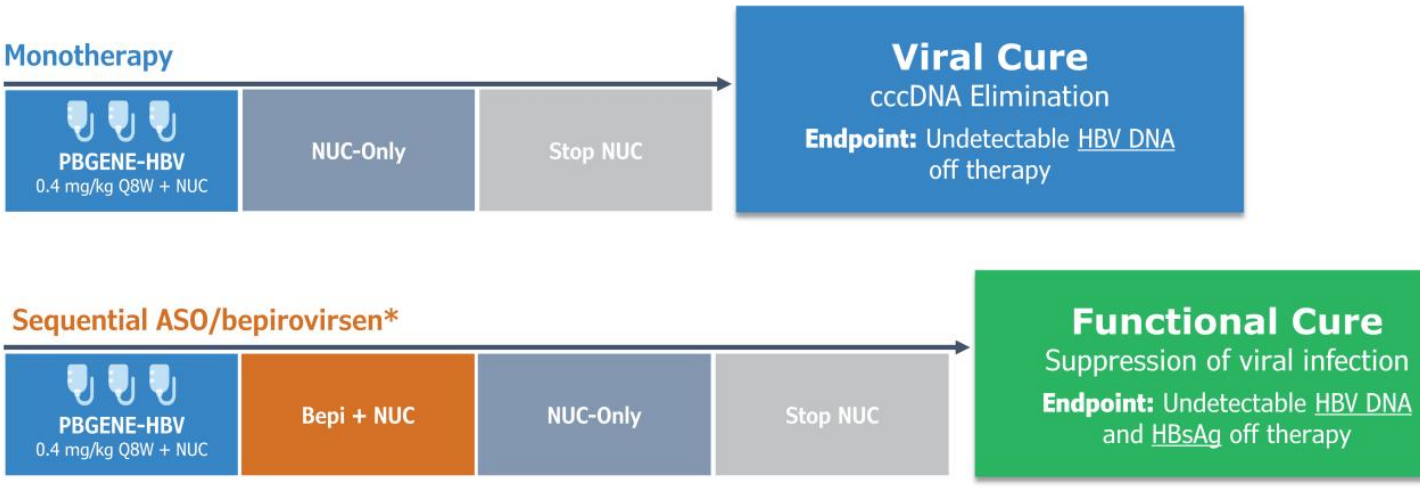


pgRNA is the cccDNA-specific biomarker
HBsAg is a secondary biomarker expected to be reduced with cccDNA elimination

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

Back to HBsAg data

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



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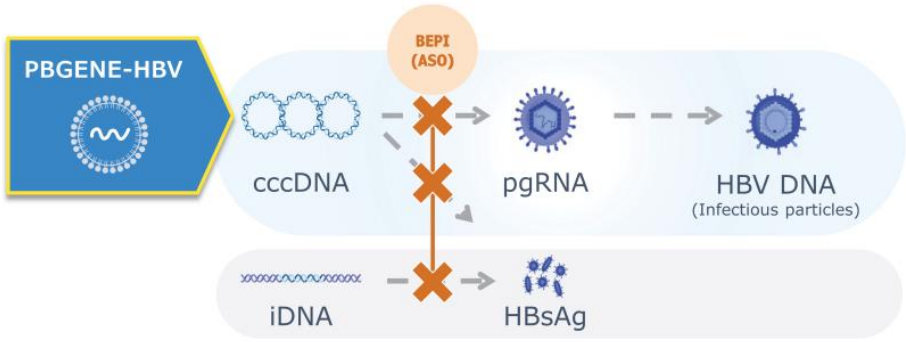
ASO, antisense oligonucleotide; cccDNA, covalently closed circular DNA; FDA, US Food and Drug Administration; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; MHLW, Ministry of Health, Labour and Welfare; Q8W, every 8 weeks.

Schema

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

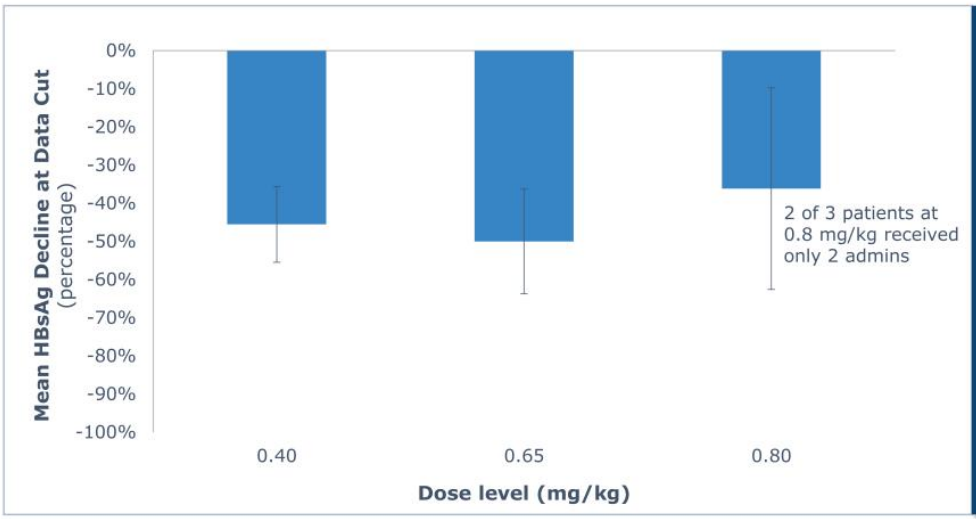
2 Goal of deeper suppression of viral transcripts with **Bepirovirsen**

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
All Dose Levels ≥0.4 mg/

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 proto n=8 of 13
Grade 3	Hypotension	-	-	-
Grade 3 and 4	ALT/AST lab abnormality	4	2	1
SAEs		-	-	-

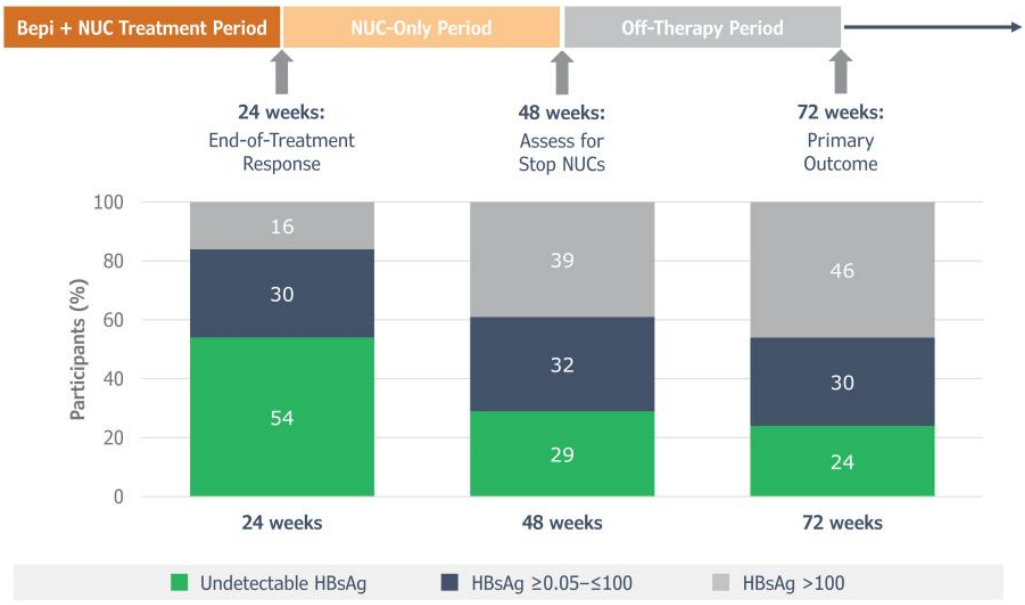
- An independent pharmacovigilance firm has assessed adverse events of interest in the Phase 1 ELIMINATE-B study and provided an analysis these events graded under CTCAE, which will be used in the Phase 2 ELIMINATE-B2 study
- Currently, ELIMINATE-B has utilized a DAIDS framework for assigning grades 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 r

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 Treatn for Chronic Hepatitis B Virus Infectio

The NEW ENGLAND JOURNAL of MEDICINE

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

PBGENE-DMD

Large Valuation Opportunity for Transformative Gene Therapy Drugs in DM



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."

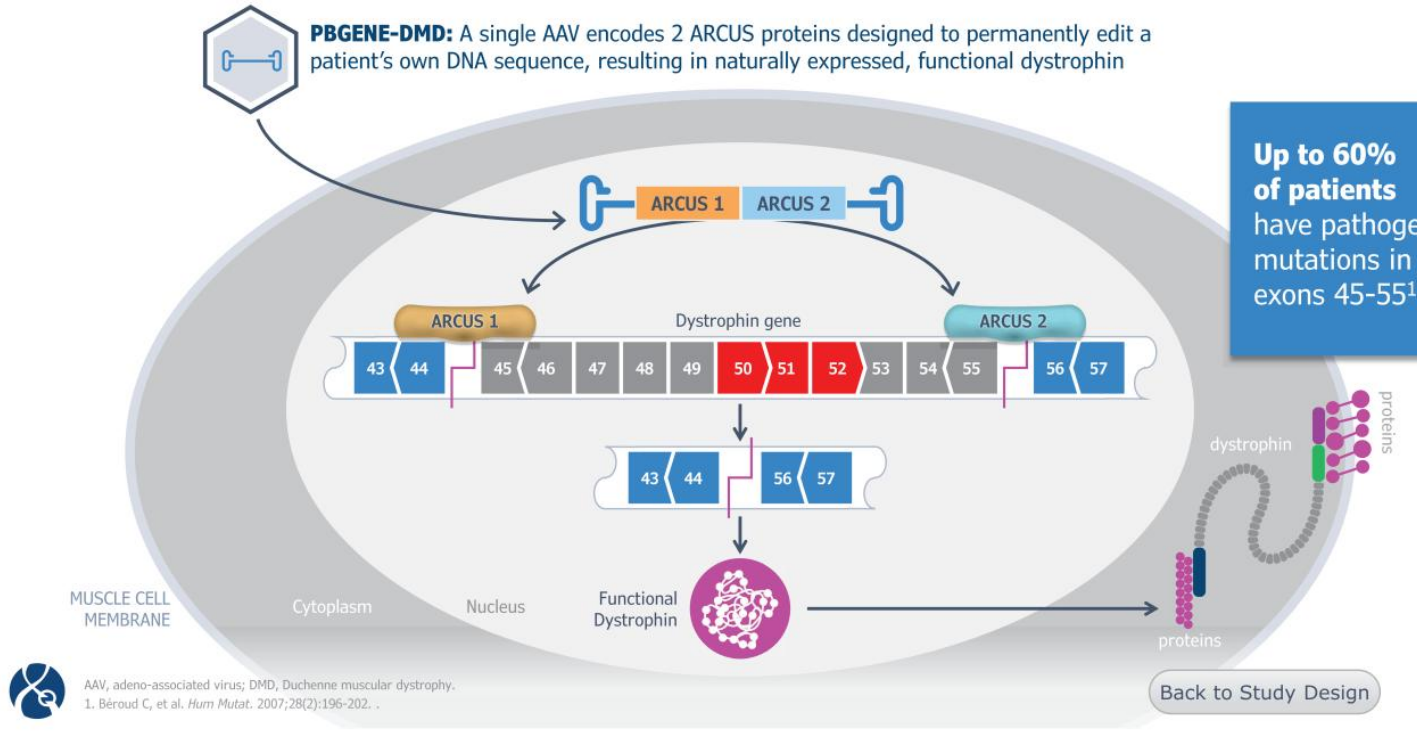
After receiving approval for its gene therapy Elevidys for DMD, Sarepta reached a peak market capitalization of ~$15.7B in June 2024.

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

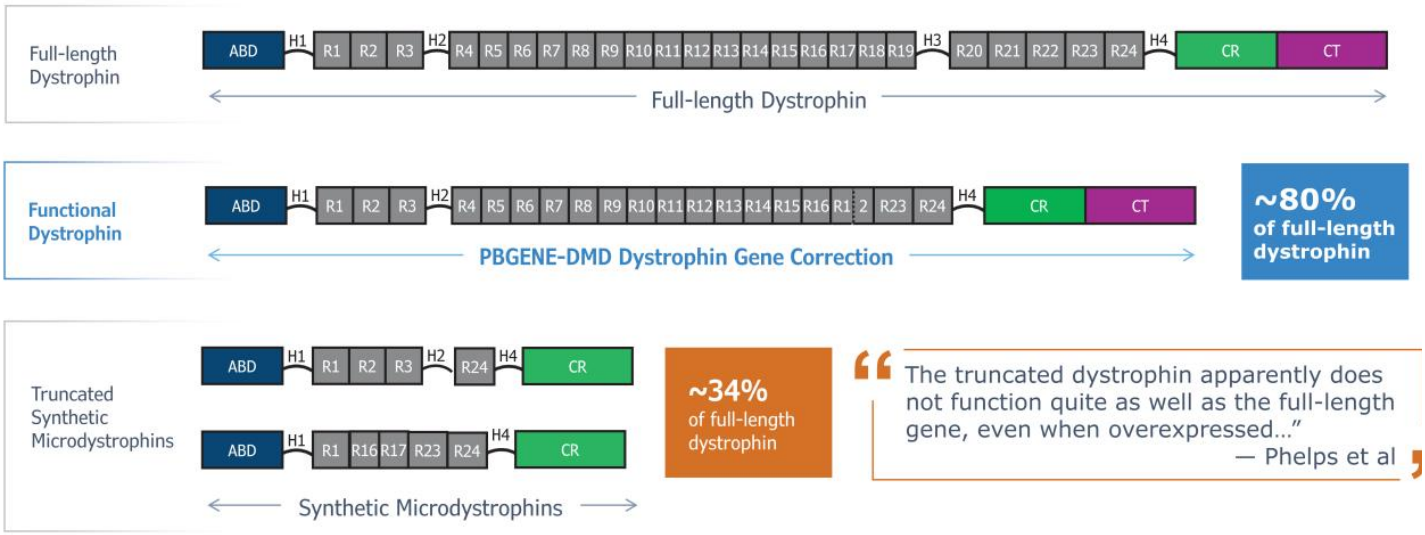
https://clinicaltrials.gov/study/NCT03375164
Sources: https://www.macrotrends.net/stocks/charts/SRPT/sarepta-therapeutics/market-cap

Main

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

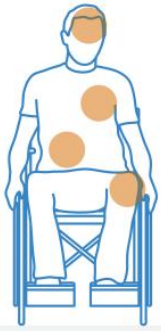


PBGENE-DMD Designed to Produce a Near-Full-Length Dystrophin Protein, Demonstrated to be Functional in Humans



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

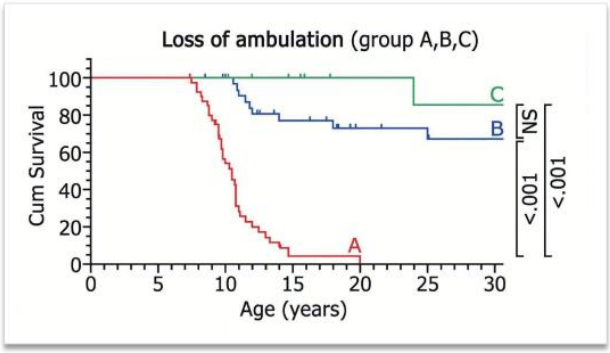
- Asymptomatic or mild symptoms[1-4]
- Normal muscle strength and ambulation throughout life[1,2]
- Normal respiratory function[2]
- Myocardial involvement, often manageable with medication[2]

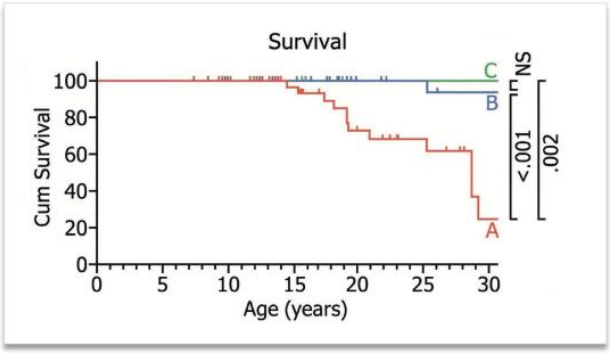
Del45-55 in-frame dystrophin gene (BMD)

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

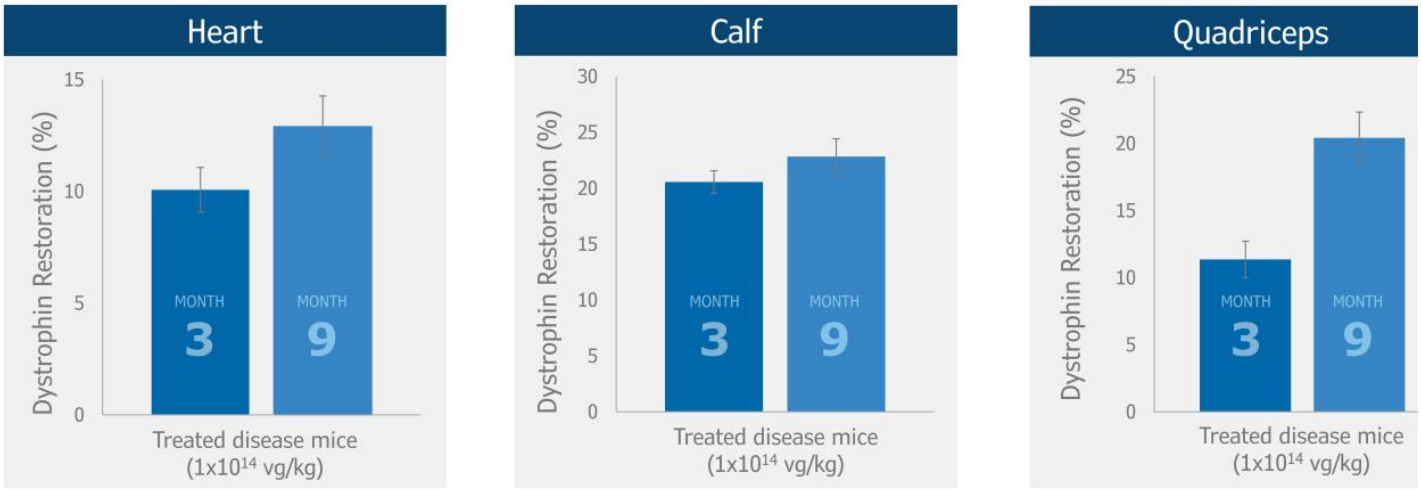
It is expected that as little as 5% expression of functional dystrophin protein is needed to provide therapeutic benefit in patients with DMD

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



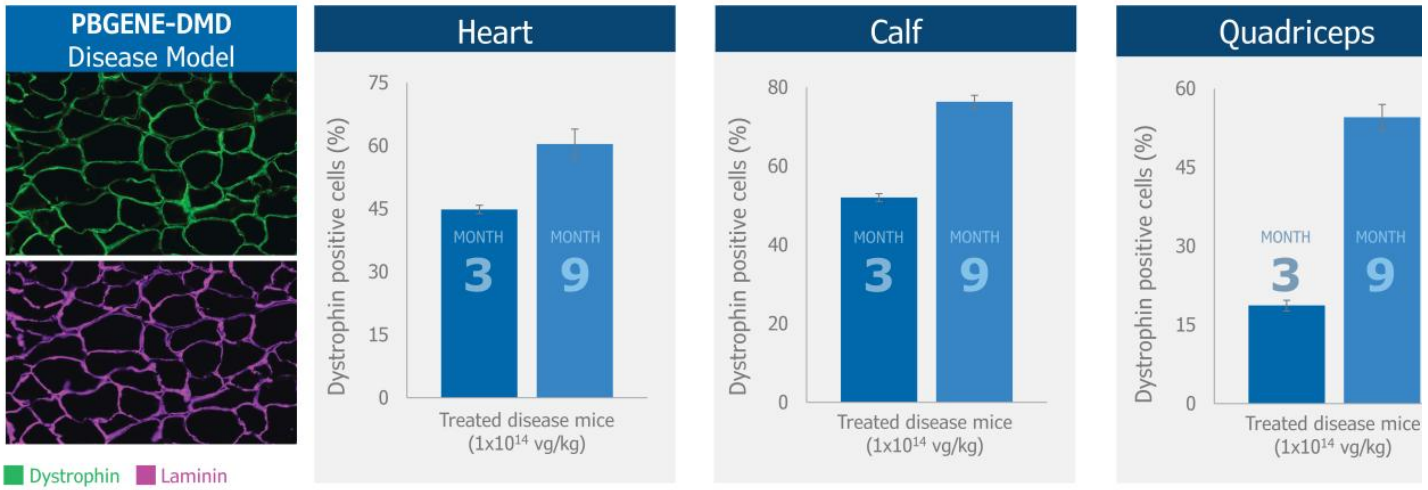
PBGENE-DMD Resulted in Increased Dystrophin Protein Levels Over Time, Exceeding Expected Therapeutic Threshold



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

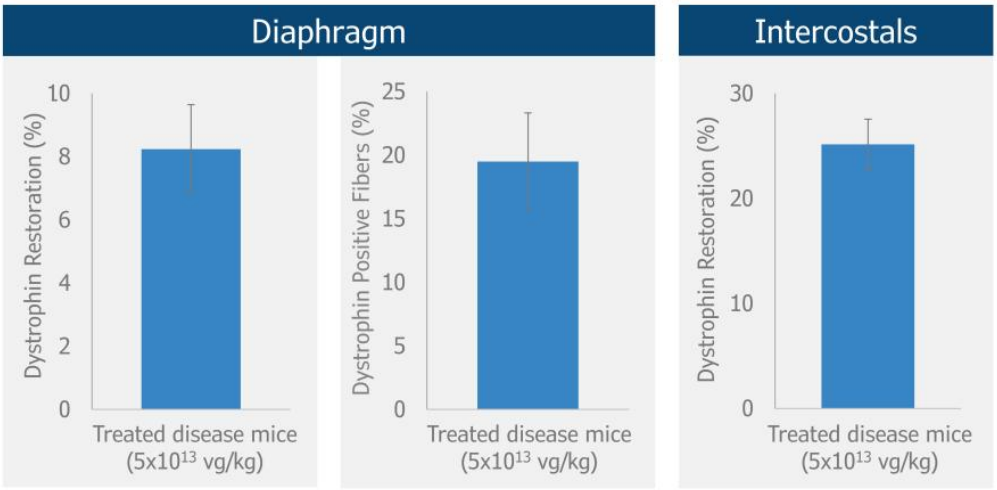
Broad Distribution of Dystrophin Positive Cells Across Skeletal, Cardiac, and Respiratory Muscles After PBGENE-DMD Treatment



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



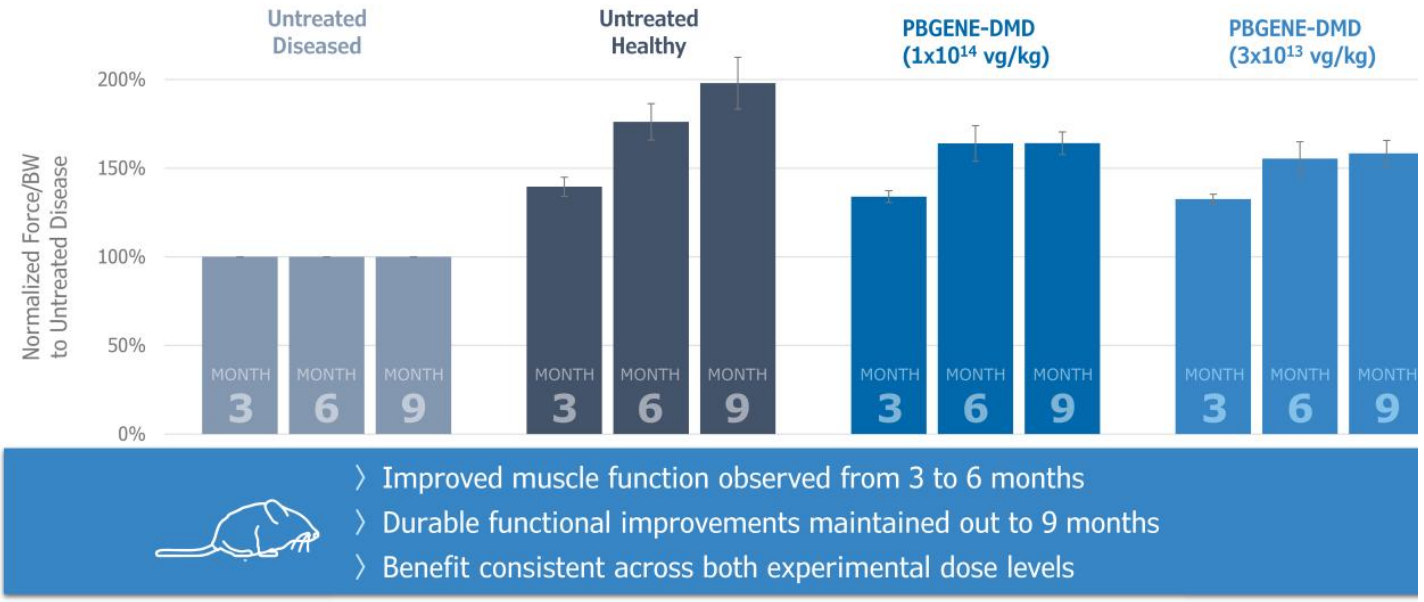
Dystrophin protein restoratio
early juvenile mice was up t
2.5-12X higher in skeletal ar
respiratory muscle compared
late juvenile mice

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

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

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

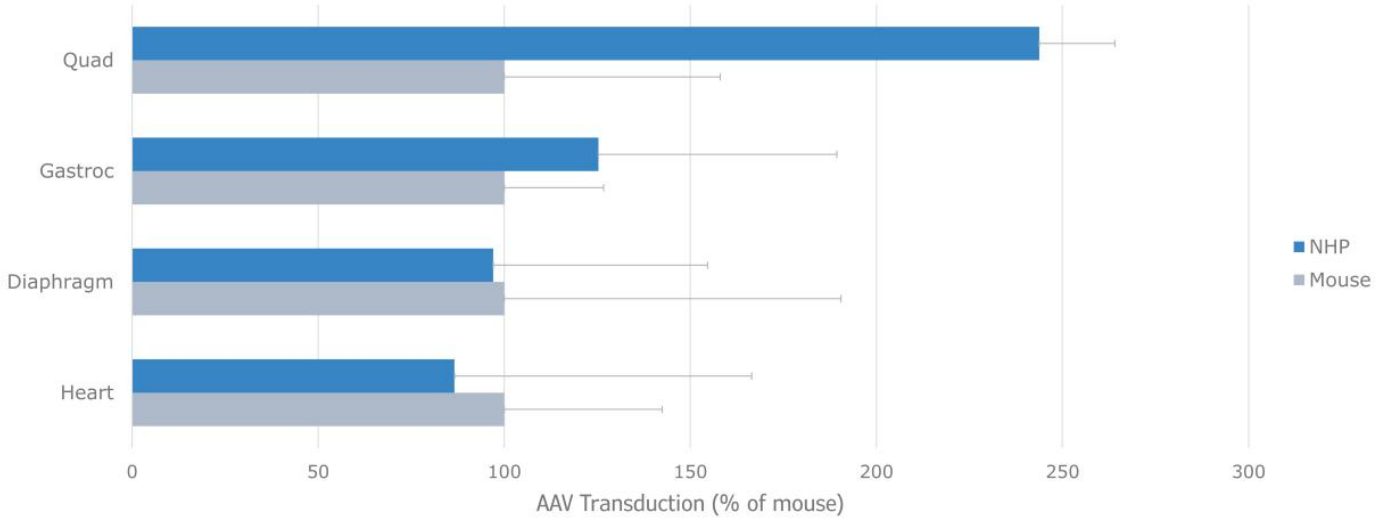
PBGENE-DMD Significantly Improved Muscle Function and Demonstrated Long-Term Durability



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

*Discontinued.
AAV, adeno-associated virus; 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.

functionDMD

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:	Goal
Measurement of biologic activity of PBGENE-DMD through dystrophin expression in skeletal muscle biopsies at Weeks 12 and 52	Demonstrates target engagement and biological proof-of-concept

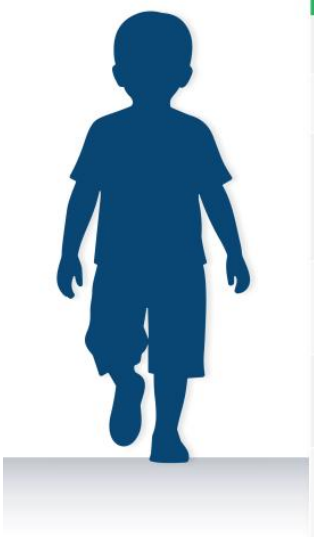
Exploratory Endpoints

Functional & Developmental Outcom

- DMO: Stride Velocity 95th Centile
- Bayley Scales of Infant and Toddler Developme Gross Motor and Fine Motor scores (<3 years o
- 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.

functionDMD | 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