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AASLD 2025 Business Update

November 2025

Forward-Looking Statements

This presentation contains forward-looking statements, as may any related presentations, within the meaning of the Private Securities Litigation Reform Act of 1995. The Company intends such forward-looking statements to be covered by the safe harbor provisions for forward-looking statements contained in Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. All statements contained herein and in any related presentation that do not relate to matters of historical fact should be considered forward-looking statements, including, without limitation, statements regarding our financial position, including our expected cash runway and the sufficiency of our cash runway extending through 2028, the clinical development and expected safety, efficacy and benefit of our product candidates (including PBGENE-HBV) and gene editing approaches including editing efficiency; our expected cash runway, including availability of our ATM facility, and the sufficiency of our cash runway extending through 2028 enabling achievement of certain clinical and regulatory milestones for PBGENE-HBV and PBGENE-DMD; the design of PBGENE-HBV to eliminate cccDNA and inactivate integrated HBV DNA with high specificity and driving functional cures; the differentiation of ARCUS from other gene editing approaches; the expected timing of regulatory processes (including filings such as IND's and CTA's and studies for PBGENE-HBV and the acceptance of these filings by regulatory agencies); the translation of preclinical safety and efficacy studies and models to safety and efficacy in humans, the suitability of PBGENE-HBV for the treatment of hepatitis and the targeting of the root cause of the disease; clinical safety and efficacy data demonstrating PBGENE-HBV was well tolerated and demonstrated proof of activity as well as signs of cumulative and durable HBsAG reductions; expectations about operational initiatives, strategies, and further development of our programs; expectations about achievement of key milestones; and anticipated timing of regulatory filings, regulatory acceptances and clinical data. In some cases, you can identify forward-looking statements by terms such as "aim," "anticipate," "approach," "believe," "contemplate," "could," "designed to", "estimate," "expect," "goal," "intend," "look," "may," "mission," "plan," "possible," "potential," "predict," "project," "promise," "pursue," "should," "target," "will," "would," and other similar words or expressions, or the negative of these words or similar words or expressions, are intended to identify forward-looking statements, though not all forward-looking statements use these words or expressions.

Forward-looking statements are based on management's current expectations, beliefs and assumptions and on information currently available to us. These statements are neither promises nor guarantees, but involve number of known and unknown risks, uncertainties and assumptions, and actual results may differ materially from those expressed or implied in the forward-looking statements due to various important factors, including, but not limited to: our ability to become profitable; our ability to procure sufficient funding to advance our programs; risks associated with raising additional capital and requirements under our current debt instruments and effects of restrictions thereunder; our operating expenses and our ability to predict what those expenses will be; our limited operating history; the success of our programs and product candidates in which we expend our resources; our limited ability or inability to assess the safety and efficacy of our product candidates; our dependence on our ARCUS technology; the initiation, cost, timing, progress, achievement of milestones and results of research and development activities, preclinical studies and clinical trials; public perception about genome editing technology and its applications; competition in the genome editing, biopharmaceutical, and biotechnology fields; our or our collaborators' ability to identify, develop and commercialize product candidates; potential product liability lawsuits and penalties against us or our collaborators related to our technology and our product candidates; the U.S. and foreign regulatory landscape applicable to our and our collaborators' development of product candidates; our or our collaborators' or other licensees' ability to advance product candidates into, and successfully design, implement and complete, clinical or field trials; potential manufacturing problems associated with the development or commercialization of any of our product candidates; delays or difficulties in our and our collaborators' ability to enroll patients; changes in interim "top-line" and initial data that we announce or publish; if our product candidates do not work as intended or cause undesirable side effects; risks associated with applicable healthcare, data protection, privacy and security regulations and our compliance therewith; our ability to obtain orphan drug designation or fast track designation for our product candidates or to realize the expected benefits of these designations; our or our collaborators' ability to obtain and maintain regulatory approval of our product candidates, and any related restrictions, limitations and/or warnings in the label of an approved product candidate; the rate and degree of market acceptance of any of our product candidates; our ability to effectively manage the growth of our operations; our ability to attract, retain, and motivate executives and personnel; effects of system failures and security breaches; insurance expenses and exposure to uninsured liabilities; effects of tax rules; effects of any pandemic, epidemic, or outbreak of an infectious disease; the success of our existing collaboration agreements, and our ability to enter into new collaboration arrangements; our current and future relationships with and reliance on third parties including suppliers and manufacturers; our ability to obtain and maintain intellectual property protection for our technology and any of our product candidates; potential litigation relating to infringement or misappropriation of intellectual property rights; effects of natural and manmade disasters, public health emergencies and other natural catastrophic events; effects of sustained inflation, supply chain disruptions and major central bank policy actions; market and economic conditions; risks related to ownership of our common stock, including fluctuations in our stock price; our ability to meet the requirements of and maintain listing of our common stock on NASDAQ or other public stock exchanges; and other important factors discussed under the caption "Risk Factors" in our annual Report on Form 10-K for the fiscal year ended December 31, 2024 and our Quarterly Reports on Form 10-Q for the quarters ended March 31, 2025, June 30, 2025, and September 30, 2025, as any such factors may be updated from time to time in our other filings with the SEC, which are accessible on the SEC's website at www.sec.gov and the Investors page of our website under SEC Filings at investor.precisionbiosciences.com.

All forward-looking statements speak only as of the date of this presentation and, except as required by applicable law, we have no obligation to update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise. Precision consults with various presentation speakers and compensates them for their time and expertise.

ARCUS WORKS: ARCUS Platform Has Multiple Points of Clinical Validation Led by Wholly Owned HBV Program with an Emerging Path to Expansion & Phase II

ARCUS: DTIL Proprietary Gene Editing Platform

- ARCUS wholly-owned by Precision BioSciences
- Derived from the homing endonuclease I-CreI found in green algae
- Naturally evolved to drive high efficiency editing
- > 65 patents issued covering ARCUS and in vivo gene editing

Clinical Stage Programs Validating ARCUS

Wholly Owned Program:

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PBGENE-HBV for Chronic Hepatitis B

- Viral gene elimination program with over 22 doses delivered across 9 patients treated to date with responses at all dose levels
- **AASLD Late breaker ELIMINATE-B phase 1 oral presentation on November 10th**
- **Clinical data emerging may support path to expansion phase & Phase II earlier than expected**

Partnered Programs:

ECURE

ECUR-506 for OTC Deficiency

- Gene insertion program utilizing ARCUS nuclease
- First patient treated and reported in Phase I study in a complete response; now over 1.5 years old

IMUGENE

Azer-cel CAR T in Hematology

- Clinically tested in ~100 patients
- Pivotal Dose & Combination Identified
- November FDA meeting in relapsed DLBCL setting to align on pivotal design

AASLD, American Association for the Study of Liver Diseases; CAR T, chimeric antigen receptor T cell; DLBCL, diffuse large B-cell lymphoma; HBV, hepatitis B virus; OTC, ornithine transcarbamylase; PRV, Priority Review Voucher.

Successful $75M Financing Fuels Further Development of Lead Programs While Generating Shareholder Value Through Multiple Clinical Milestones Expected Over Next 12-24 Months

Wholly owned In Vivo Gene Editing Programs

		Expected First Clinical Data Timing
PBGENE-HBV **Phase 1/2a Program for Chronic Hepatitis B**	〉 First-in-Class Mechanism targeting cccDNA elimination 〉 Well Tolerated Safety Profile even with repeat dosing 〉 Active Drug Across Doses and HBsAg Levels 〉 Durable responses in 7 of 9 patients 〉 **Near-term path towards stopping NUCs**	**2025** eliminate-B
PBGENE-DMD **Duchenne Muscular Dystrophy**	〉 PBGENE-DMD is designed to provide durable functional muscle improvement **for ~60% of patients with DMD** 〉 PBGENE-DMD significantly **improved muscle function** and demonstrated **long-term durability** in DMD mouse model 〉 Targeting IND filing in late 2025 and dosing patients in 2026 with BLA filing in 2028	**2026** functionDMD

Multiple Clinical Datasets Expected to Read-Out within Extended Current Cash Runway through 2028[1]

Financing with Top Tier Fundamental Investors Expected to Fuel Further Development of PBGENE-HBV through Phase 2 Study and to PBGENE-DMD BLA Submission

cccDNA, covalently closed circular DNA; DMD, Duchenne Muscular Dystrophy; FDA, Food and Drug Administration; H1, first-half of the year; H2, second-half of the year; HBV, Hepatitis B virus; HBsAg, Hepatitis B surface antigen; IND, investigational new drug; MHRA, Medicines & Healthcare products Regulatory Agency; NUCs, nucleos(t)ide analogs

1. $71.2M cash and restricted cash as of 9/30/25. The Company expects existing cash and cash equivalents, its recent financing, potential near-term cash from CAR T transactions, along with expected operating efficiencies, operational receipts, and availability of Precision's at-the-market (ATM) facility to extend Precision's cash runway through 2028

Dr. MF Yuen Presented Late Breaking Oral Presentation on PBGENE-HBV at AASLD

*Phase 1 datasets rarely receive late breaking oral presentations**

AASLD The Liver Meeting® NOV. 7-11, 2025

Late-Breaking Abstract Presentation

Abstract title

PBGENE-HBV, a First-in-class Gene Editing Therapy for Chronic Hepatitis B, Demonstrates Safety and Antiviral Activity in Early Cohorts

Publication number: **5017** | Type of presentation: **Late-breaking oral presentation**

Presenter	Date and time
MF Yuen, MD, PhD	**November 10th (Monday)** 5:30pm-6:00pm EST

Authors
Man-Fung Yuen, Alina Jucov, Edward Gane, Emily B. Harrison, Andrew Van Cott, Abhishek Chandiramani, Neil Leatherbury, John Fry, Jeff Smith, Cassandra L. Gorsuch, Stanley Frankel, Mark Sulkowski.

MF Yuen, MD, PhD

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

- Leading enroller for early phase HBV trials—*presented over 360 lectures worldwide, and is leading international trials in HBV*
- Demonstrated success with biopsies
- Lead investigator for GSK late-stage trial in HBV
- Prolific publication record (>560) in top tier journals

AASLD, American Association for the Study of Liver Diseases; HBV, hepatitis B virus.
*AASLD The Liver Meeting Late Breaking Abstract Submission Guidelines, https://www.aasld.org/the-liver-meeting/late-breaking-abstract-submissions

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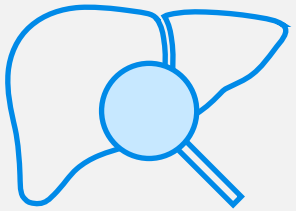
PBGENE-HBV
Designed to Eliminate
Source of Hepatitis B Virus

The First and Only Gene Editor for Chronic Hepatitis B:
Clinical Data to Date Demonstrates Repeat Safety and Meaningful Antiviral Activity Across First Three Cohorts

New Rules for a New Modality:

Resetting the Ground Rules for Direct Viral Elimination of Hepatitis B the ONLY Path to Complete Cure

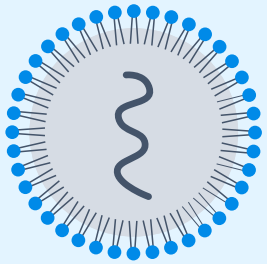


Reducing HBsAg is Not Enough:

- **HBsAg reductions alone,** even by multiple logs, have largely **not established the immune balance** needed for viral suppression

Direct Viral Elimination Drives HBsAg Reductions:

- **Eliminating the viral source, cccDNA,** and thus reducing viral transcription and antigen production, including HBsAg, drives viral suppression



PBGENE-HBV

First and Only Clinical Stage Program Demonstrating Promising Data with Path Towards Stopping NUCs to Test for Cure

- PBGENE-HBV has been **well tolerated** after multiple doses and up to 0.8 mg/kg; shows ability to **safely repeat dose with predictable and manageable profile**
- **Dose-dependent antiviral effects and molecular biopsy data** demonstrate first ever **proof of activity** for a **gene editor designed to eliminate the viral source of hepatitis B**
- **There is an emerging path for PBGENE-HBV to stop NUCs, test for cure, and progress to expansion/phase 2**

cccDNA, covalently closed circular DNA; HBV, hepatitis B virus; HBsAg, Hepatitis B surface antigen; mg/kg, milligram per kilogram; NUCs, nucleos(t)ide analogs.

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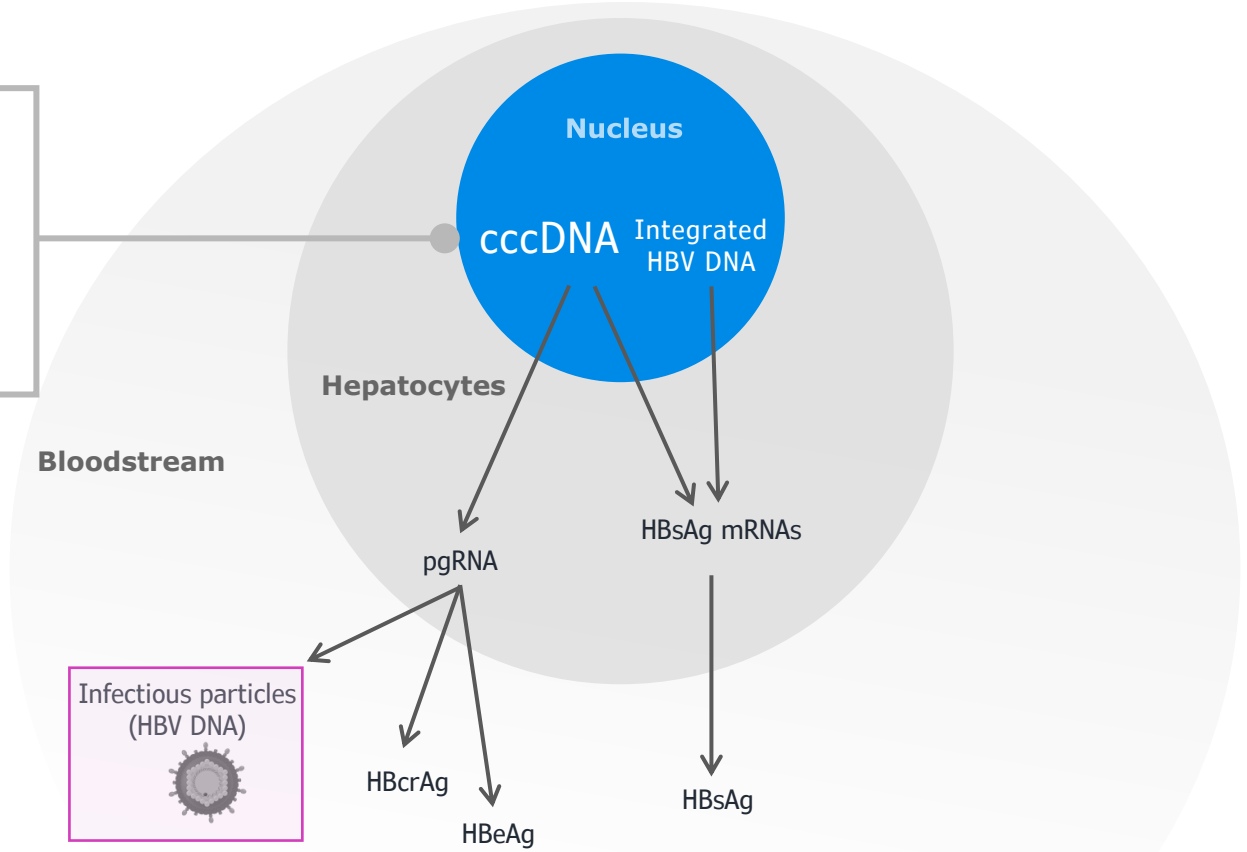
Large Unmet Need Remains for Patients with Hepatitis B

Current Standard of Care Nucleoside Analogs (NUC) and Therapies in Development Have Never Addressed the Root Cause of Hepatitis B and Rarely Cures

cccDNA is the Only Source of New Infectious Particles

cccDNA is the source of pregenomic RNA (pgRNA) that is necessary for infectious particles (HBV DNA)



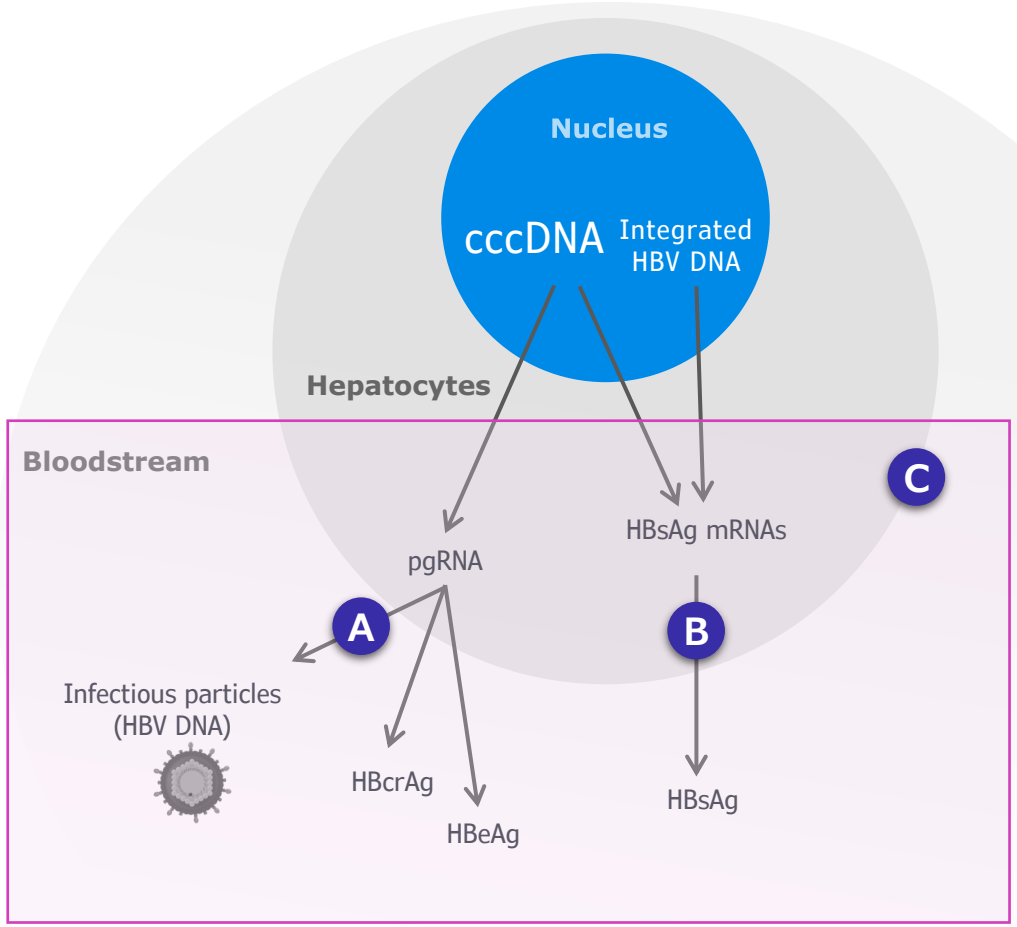
Chronic HBV is driven by persistence of cccDNA

cccDNA, covalently closed circular DNA; DNA, deoxyribonucleic acid; HBeAg, hepatitis B e antigen; HBcrAg, hepatitis B core-related antigen; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; mRNA, messenger RNA; NUCs, nucleos(t)ide analogs; pgRNA, pregenomic RNA; RNA, ribonucleic acid.

Even with Latest Therapies in Development Functional Cure Remains Elusive:

Downstream targeting therapies do not eliminate cccDNA

Therapies in development target downstream components of the viral life cycle



Treatment with NUCs only results in a 1-3% functional cure rate and patients continue to have an elevated risk of hepatocellular carcinoma (HCC)[1,2]

- **A** Nucleos(t)ide Analogs
  Capsid Assembly Modulator
- **B** Nucleic Acid Polymer Therapy
  Antisense Oligonucleotides
  siRNA
- **C** Immune modulators

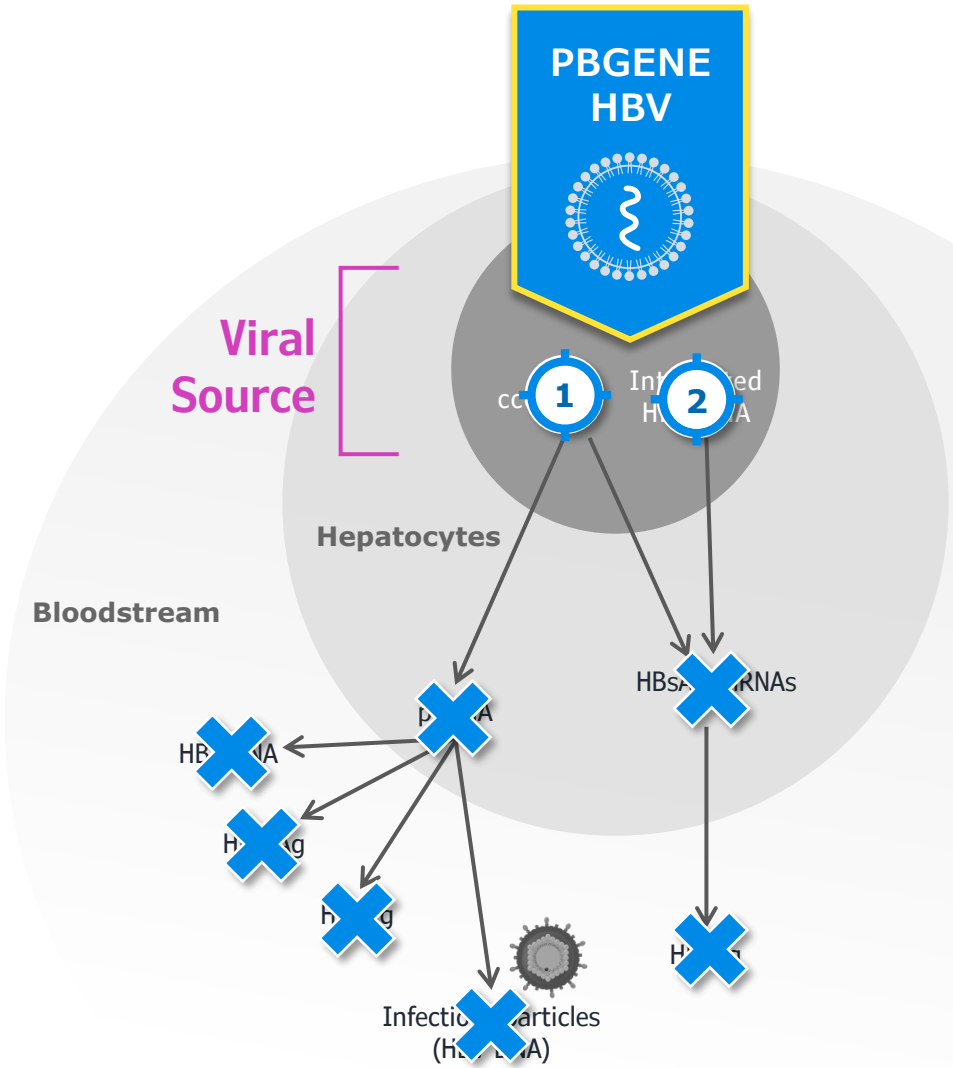
cccDNA, covalently closed circular DNA; DNA, deoxyribonucleic acid; HBeAg, hepatitis B e antigen; HBcrAg, hepatitis B core-related antigen; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; mRNA, messenger RNA; NUCs, nucleos(t)ide analogs; pgRNA, pregenomic RNA; RNA, ribonucleic acid; siRNA, small interfering RNA.

1. Cornberg et al., J Hepatol 2020 Mar;72(3):539-557.

HBV Cure Requires a Novel Approach Targeting the Viral Replication Source

Eliminating cccDNA has a Biologic Rationale for Cure

1. Eliminates cccDNA
2. Inactivates Integrated HBV DNA
3. Treatment at the Source of the Viral Pathway Results in Reductions of Downstream Markers



"The ideal therapeutic strategy for curative approaches includes reduction or elimination of the whole cccDNA pool."

—Ligat et al. 2020

PBGENE-HBV is uniquely designed to achieve a complete cure by eliminating cccDNA and inactivating integrated DNA at the source of HBV, preventing the chance of viral relapse

cccDNA, covalently closed circular DNA; DNA, deoxyribonucleic acid; HBeAg, hepatitis B e antigen; HBcrAg, hepatitis B core-related antigen; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; mRNA, messenger RNA; NUCs, nucleos(t)ide analogs; pgRNA, pregenomic RNA; RNA, ribonucleic acid; siRNA, small interfering RNA.

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ELIMINATE-B Trial Results:

Testing PBGENE-HBV the First Clinical Modality Designed to Eliminate cccDNA

Phase 1/2a Study Design

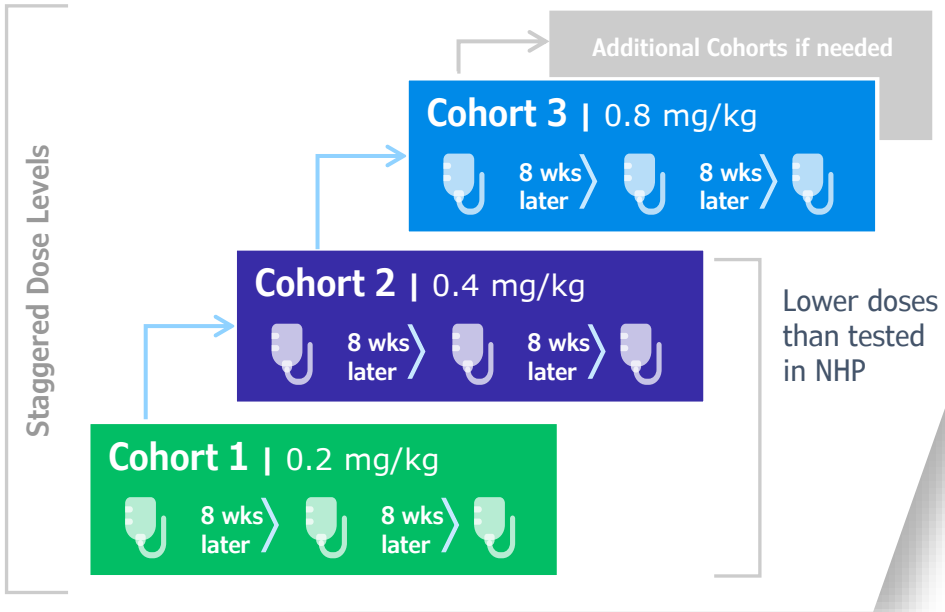
Potential for rapid evaluation of safety and efficacy of PBGENE-HBV in 2025

Part 1: Multiple Ascending Dose Escalation

Finite Treatment: Patient receives up to 3 dose administrations*

HBeAg negative patients on NUCs

N=3 dosed at each cohort



Safety & Efficacy Evaluation

Part 2: Dose Expansion

Advance optimized dose and schedule to eliminate cccDNA and drive cure

Go Forward Dose

Optimal

- Dose level
- Number of doses
- Time between doses

N = Up to 45 patients total across both Part 1 and 2 of Phase 1 study

GOAL: Establish a finite treatment course resulting in sustained viral suppression

*3 + 3 standard design with sentinel dosing of patients. Note- protocol permits dosing additional dose administrations beyond 3 in Part 2 of protocol.
cccDNA, covalently closed circular DNA; HBeAg, hepatitis B e antigen; HBV, hepatitis B virus; mg/kg, milligram per kilogram; N, total number of patients; NHP, non-human primate; NUCs, nucleos(t)ide analogs; wks, weeks.
ClinicalTrials.gov identifier NCT06680232.

PBGENE-HBV was Well Tolerated with Repeat Dosing Across All Dose Levels

Adverse events were predictable and manageable

Number of patients experiencing treatment-related:		0.2 mg/kg n=3	0.4 mg/kg n=3	0.8 mg/kg n=3
Any Grade 1 AEs		2	3	3
Grade 2 AEs	Chills	2		
	Fever	3	3	2
	Headache	1	1	1
	Myalgia	1	1	
	ALT			1
Grade 3 AEs	AST			1
	Hypotension[1]		2	2

No DLTs have been observed across all 22 doses given[2]

AEs were transient and generally resolved within 12 hours

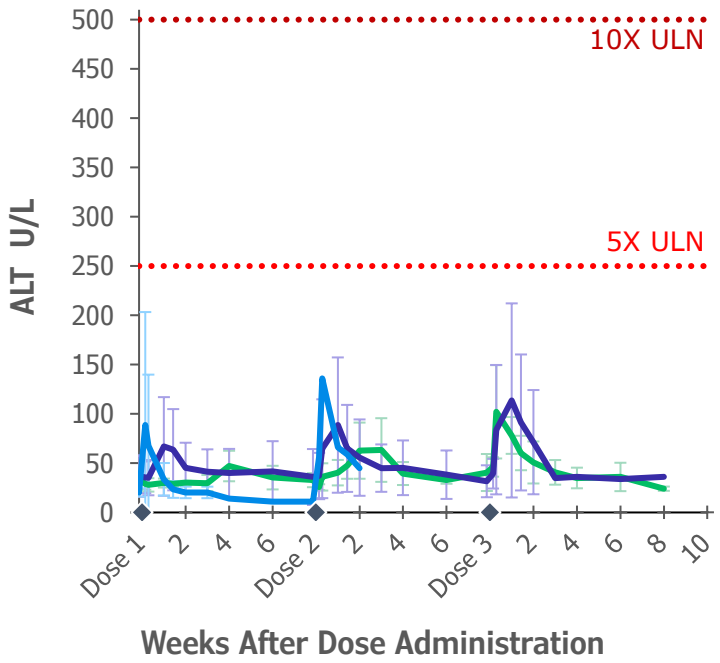
Grade 3 AST elevation resolved within ~3 days; was reviewed by independent ALT Flare Committee and deemed not dose-limiting. Hypotension events resolved in <24 hours post dosing.

AEs were consistent with infusion related reactions and were predictable and manageable

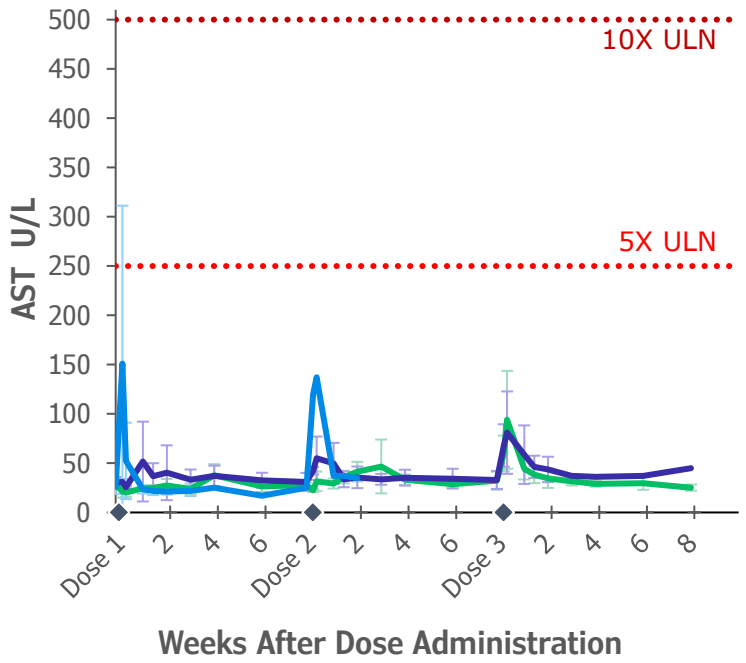
*One participant did not complete dosing due to a transient, reversible infusion reaction. The DMC did not deem this to be dose-related or dose-limiting.
**AEs shown represent treatment-related or likely treatment related events.
ALT/AST elevations graded per DAIDS criteria (Grade 2 = 2.5–5× ULN, Grade 3 = 5–10× ULN, Grade 4 = >10× ULN).
1. Hypotension was transient, occurring immediately after PBGENE-HBV infusion, and responsive to IV normal saline.
2. A DLT is any clinically significant, organ-specific, treatment-emergent adverse event (AE) ≥ Grade 3 that does not decrease to ≤ Grade 2 within 7 days and is related to study medication
AE, adverse event; AST, aspartate aminotransferase; DLT, dose-limiting toxicity; HBV, hepatitis B virus; mg/kg, milligram per kilogram; SAE, serious adverse event.
Data cutoff date was October 31, 2025.

Diving Into Most Important Safety Markers: Lab Values Below Thresholds for LNP Safety Concerns Even Following Repeat Administrations Across Cohorts

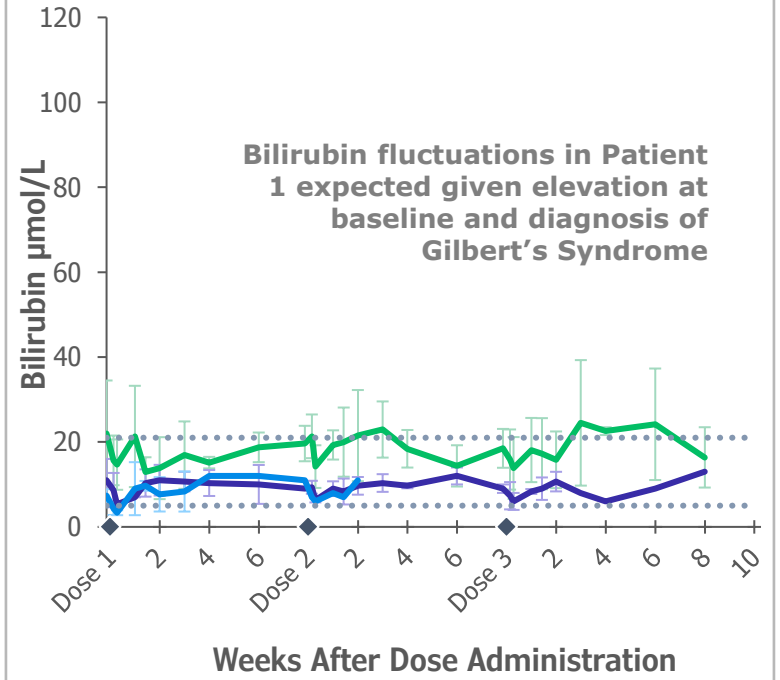
Transient ALT elevations resolved quickly and were <5X ULN



Transient AST elevations resolved quickly



Bilirubin fluctuations near normal range



Cohort 1 — Cohort 2 — Cohort 3 ◆ Dosing ····· Reference Range

- Transaminase elevations were transient with no associated changes in bilirubin and no evidence of liver dysfunction
- No changes in transaminases outside of normal limits after 8 weeks post 3rd administration

ALT, alanine aminotransferase; AST, aspartate aminotransferase; L, liter; LNP, lipid nanoparticle; U, unit; ULN, upper limit of normal; µmol, micromole.
Data shown represent mean +/- standard deviation.
ULN ranges across labs: ALT: 45-58 U/L, AST: 38-50 U/L, Bilirubin: 21-25 µmol/L
Data cutoff date was October 31, 2025.

PBGENE-HBV: Well Tolerated Across All Three Cohorts

- PBGENE-HBV has been **well tolerated with repeat doses** spanning dose levels of 0.2 mg/kg, 0.4 mg/kg and 0.8 mg/kg (n=22 doses administered)
- Adverse events were **predictable, manageable, and quickly resolved across broad range of global sites**
- Transaminase elevations were **transient, without elevations in bilirubin,** and resolved without intervention
- Platelet fluctuations have been **transient and asymptomatic**

Demonstrating safety for multiple doses of PBGENE-HBV was paramount to establish efficacious path to stopping NUCs and testing for cure

HBV, hepatitis B virus; mg/kg, milligram per kilogram; n, number of patients; NUCs, nucleos(t)ide analogs.

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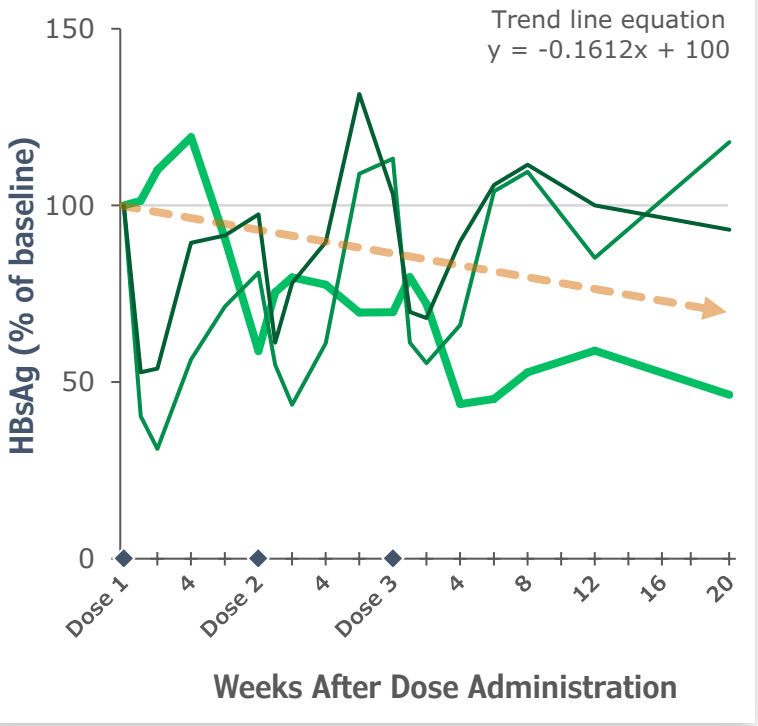
Clear Antiviral Activity Alongside Durable Responses Established Through Increasing Dose Levels with Path Towards Stopping NUCs

Antiviral Activity Established Across All Three Patients at Lowest Dose with One Patient Durable 9 Months After First Dose

Cohort 1 | 0.2 mg/kg

Activity in 3 of 3 patients

Durable response in 1 of 3 patients



Cohort 1: Patient 1 Patient 2 Patient 3 **Cohort 2:** Patient 4 Patient 5 Patient 6 **Cohort 3:** Patient 7 Patient 8 Patient 9 Dosing Baseline Trendline

HBsAg, hepatitis B surface antigen; mg/kg, milligram per kilogram.
Data cutoff date was October 31, 2025.

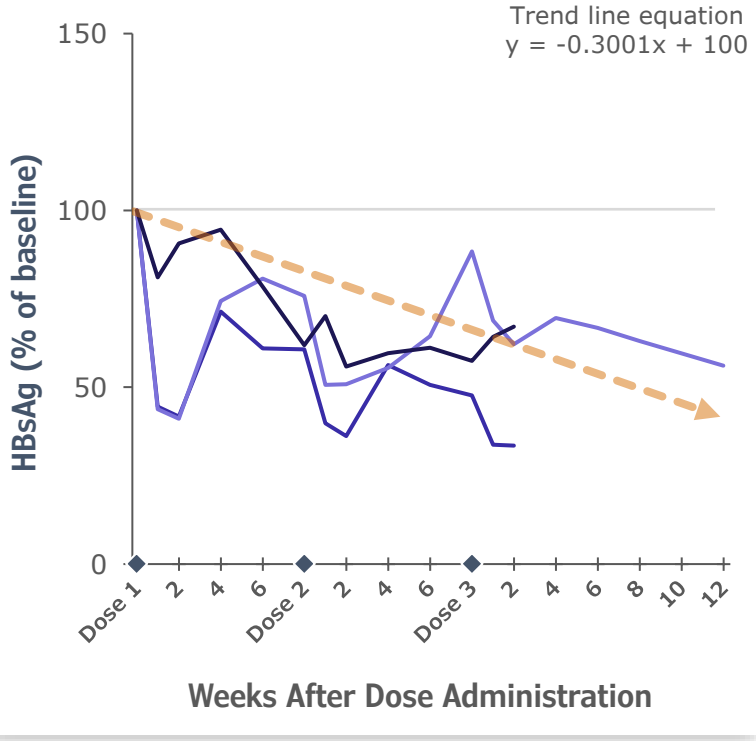
Cohort 1 HBsAg charts

At Double the Dose from Cohort 1, Cohort 2 Demonstrated Antiviral Activity and Durable Responses in All Three Patients

Cohort 2 | 0.4 mg/kg

Activity in 3 of 3 patients

Durable response in 3 of 3 patients



Cohort 1: Patient 1 Patient 2 Patient 3 **Cohort 2:** Patient 4 Patient 5 Patient 6 **Cohort 3:** Patient 7 Patient 8 Patient 9 Dosing Baseline Trendline

HBsAg, hepatitis B surface antigen; mg/kg, milligram per kilogram.
Data cutoff date was October 31, 2025.

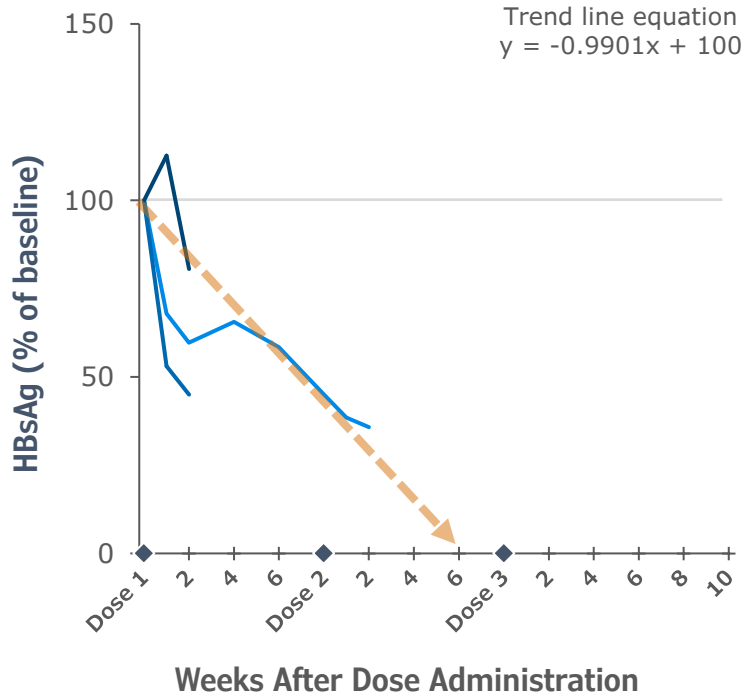
Cohort 2 HBsAg charts

Data to Date Shows that Increasing Dose to 0.8 mg/kg Leads to a Greater Rate of HBsAg Decline

Cohort 3 | 0.8 mg/kg

Activity in 3 of 3 patients

Dose dependent effect established



Cohort 1: Patient 1 Patient 2 Patient 3 **Cohort 2:** Patient 4 Patient 5 Patient 6 **Cohort 3:** Patient 7 Patient 8 Patient 9 Dosing Baseline Trendline

Interim day 21 HBsAg data was collected for patients 8 and 9 but not for patient 7 or any patients in prior cohorts. That data, which awaits final confirmation from the clinical lab, shows a similar trend to patient 7 between days 14 and 28 after the first administration.
HBsAg, hepatitis B surface antigen; mg/kg, milligram per kilogram.
Data cutoff date was October 31, 2025.

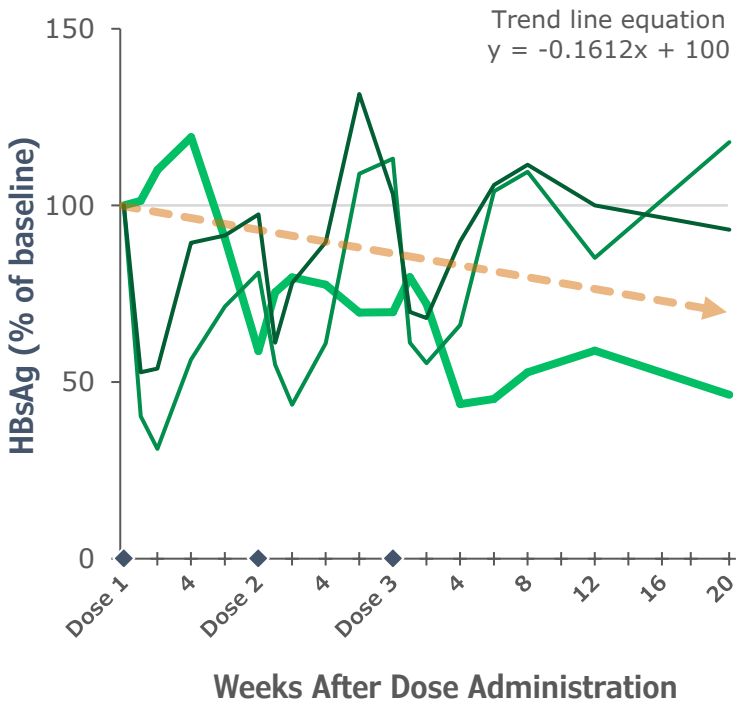
Cohort 3 HBsAg charts

Clear Antiviral Activity Alongside Durable Responses and Dose Dependent Effects Established Through Increasing Dose Levels

Cohort 1 | 0.2 mg/kg

Activity in 3 of 3 patients

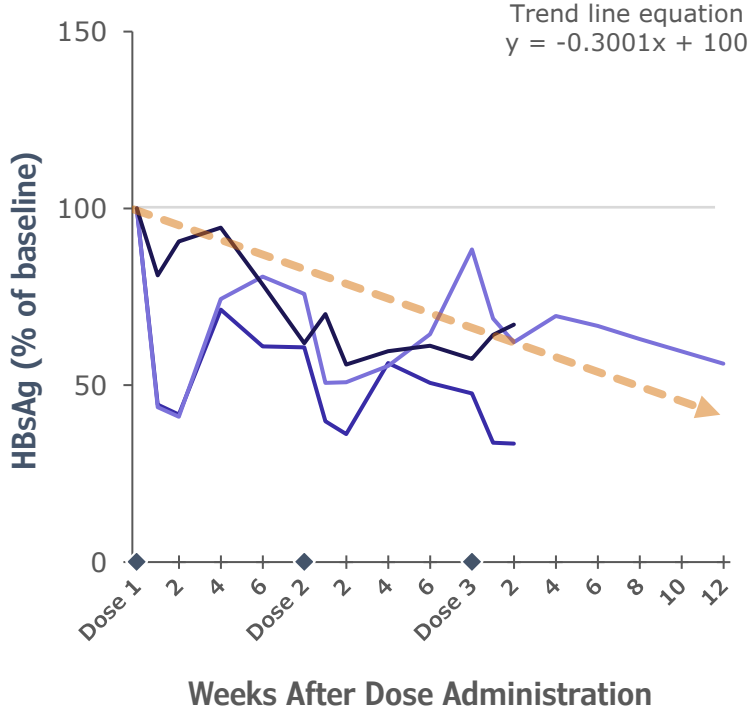
Durable response in 1 of 3 patients



Cohort 2 | 0.4 mg/kg

Activity in 3 of 3 patients

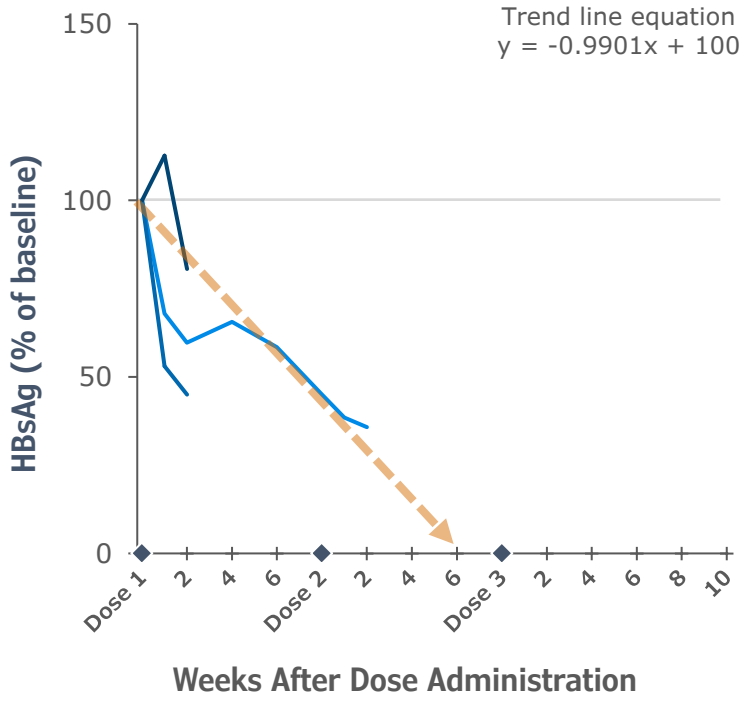
Durable response in 3 of 3 patients



Cohort 3 | 0.8 mg/kg

Activity in 3 of 3 patients

Dose dependent effect established



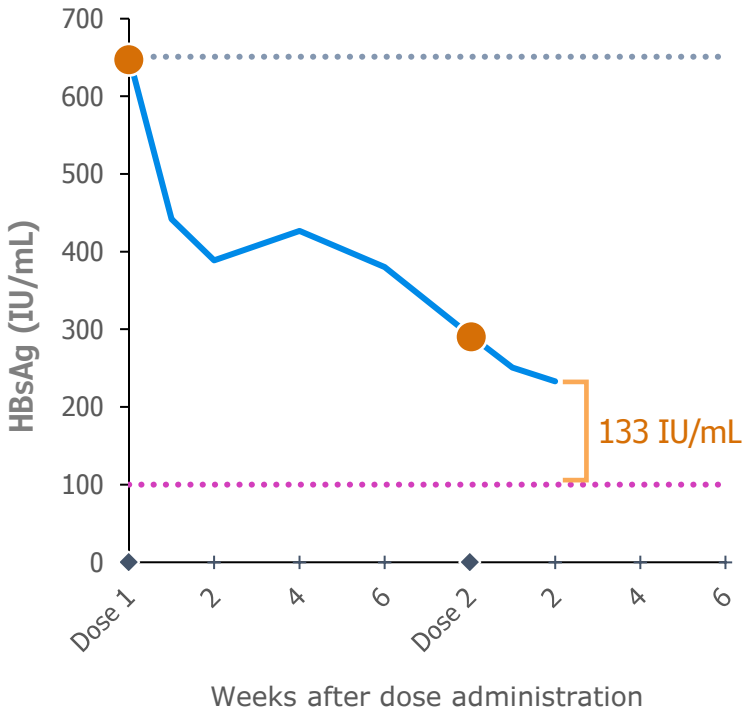
Cohort 1: Patient 1 Patient 2 Patient 3 **Cohort 2:** Patient 4 Patient 5 Patient 6 **Cohort 3:** Patient 7 Patient 8 Patient 9 Dosing Baseline Trendline

Interim day 21 HBsAg data was collected for patients 8 and 9 but not for patient 7 or any patients in prior cohorts. That data, which awaits final confirmation from the clinical lab, shows a similar trend to patient 7 between days 14 and 28 after the first administration.
HBsAg, hepatitis B surface antigen; mg/kg, milligram per kilogram.
Data cutoff date was October 31, 2025.

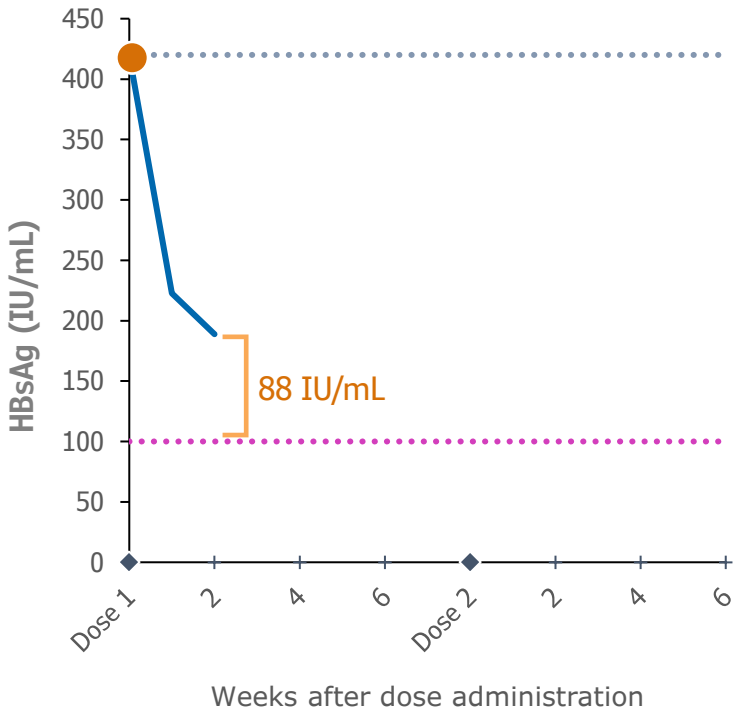
Best Responder by Cohort

Deep Dive Into Cohort 3: At 0.8 mg/kg All Three Patients Continue to Demonstrate Deepening Responses Towards Benchmark to Consider Stopping NUCs to Test for Cure

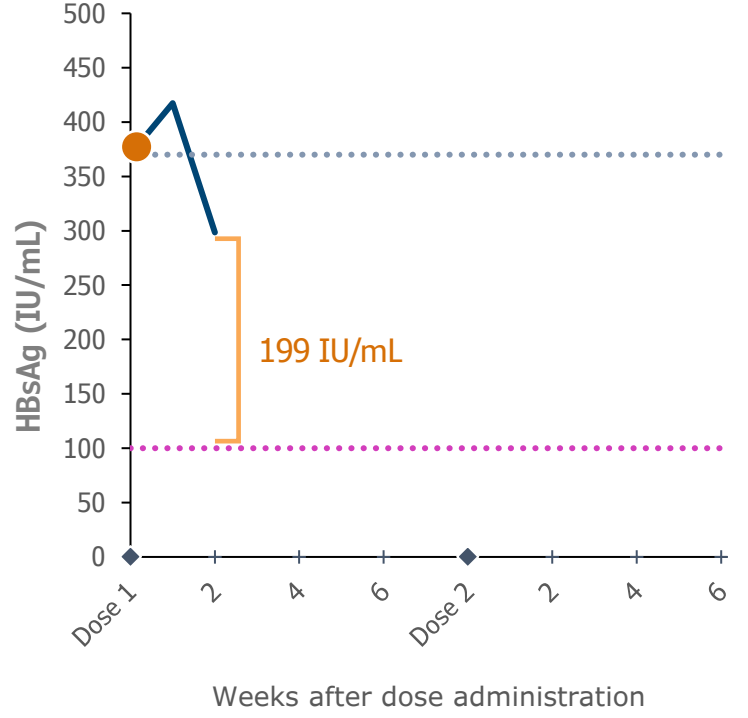
Patient 7



Patient 8



Patient 9



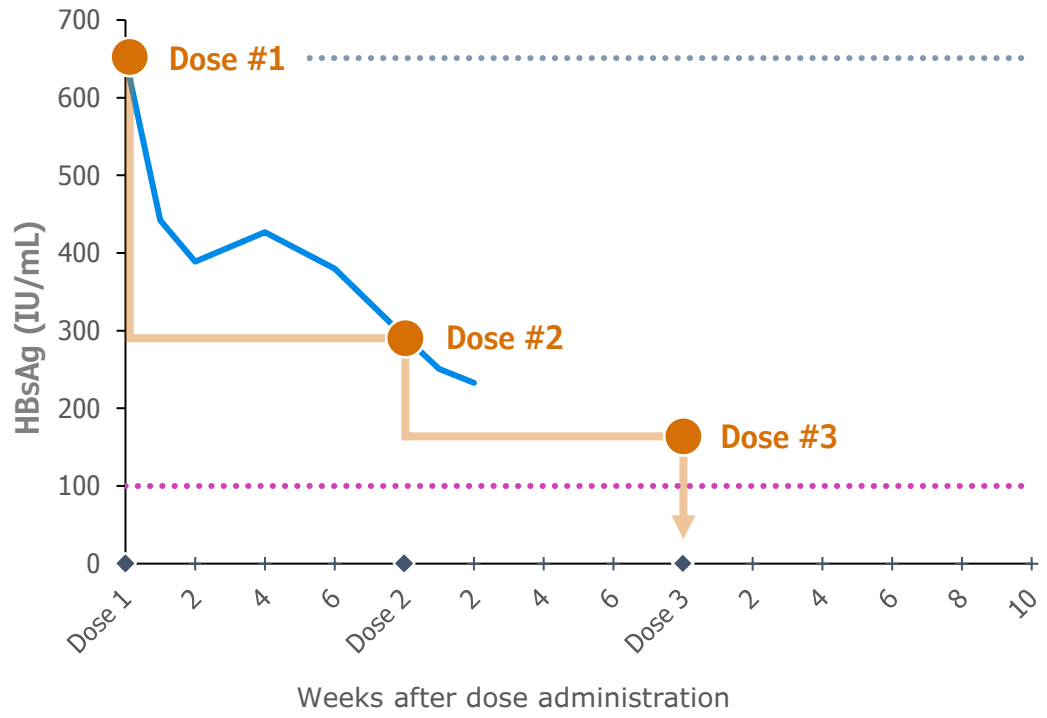
Base Line Consider stopping NUCs*

All three patients at Dose Level 3 are showing reductions in HBsAg with more dose administrations to come, potentially approaching a benchmark to consider stopping NUCs to test for cure

Interim day 21 HBsAg data was collected for patients 8 and 9 but not for patient 7 or any patients in prior cohorts. That data, which awaits final confirmation from the clinical lab, shows a similar trend to patient 7 between days 14 and 28 after the first administration.
EASL, European Organization for Hepatology; HBsAg, hepatitis B surface antigen; IU/mL, international units per milliliter; mg/kg, milligram per kilogram; NUCs, nucleo(t)side analogs.
EASL Clinical Practice Guidelines on the management of hepatitis B virus infection. J Hepatology. 2025;83(2):502-583.
Data cutoff date was October 31, 2025.

With Dose Dependent and Durable Antiviral Effects, PBGENE-HBV is on Potential Path to Stopping NUCs and Testing For Cure

Repeat dosing designed to drive cumulative antiviral effects and cure



- Only mechanism targeting cccDNA elimination in development with potential for complete cure
- Well tolerated and manageable safety profile across multiple geographies, including with repeat administrations
- All patients demonstrated antiviral activity after PBGENE-HBV treatment
- Dose responsive durability is consistently observed in later cohorts
- **Emerging near-term path towards stopping NUCs, testing for cure, and moving into Part 2 expansion**

cccDNA, covalently closed circular DNA; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; IU/mL, international units per milliliter; NUCs, nucleo(t)side analogs.
*EASL Clinical Practice Guidelines on the management of hepatitis B virus infection. *J Hepatology*. 2025;83(2):502-583.
Data cutoff date was October 31, 2025.

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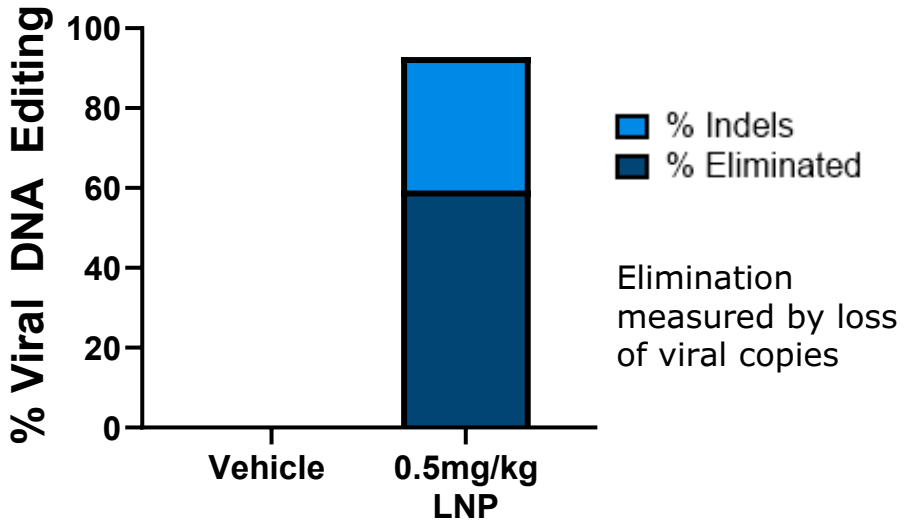
eliminate-B

Supportive Biopsy Data Confirms PBGENE-HBV Mechanism of Action Editing Viral DNA

Hot off The Press - Proof of Mechanism: Evidence of PBGENE-HBV Activity on Viral DNA

Preclinical NHP Viral DNA Editing
(Two Admins at 0.5 mg/kg)



Elimination measured by loss of viral copies

	Clinical Biopsy Data (Two Admins at 0.4 mg/kg)
Inactivation with indels in remaining DNA	**28%**
Elimination of viral DNA (Expected to be the primary editing outcome)	**Data pending** (pgRNA and cccDNA quantitation)

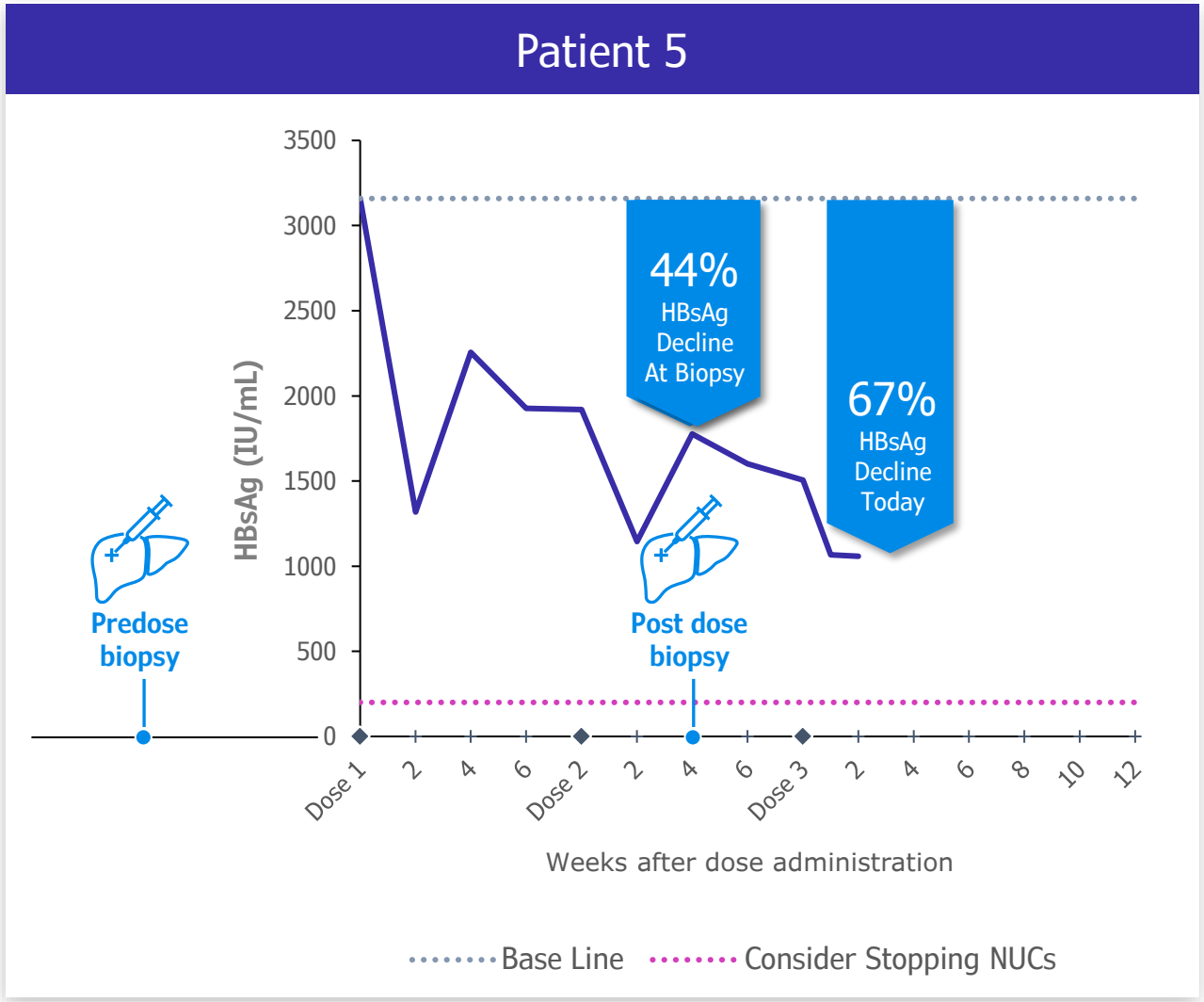
Preclinical data demonstrated viral DNA elimination as the primary outcome after editing with PBGENE-HBV

*Preclinical NHP data showed 92% total viral DNA editing (indels + elimination) after 2 administrations at 0.5 mg/kg. Assay development underway for clinical biopsy evaluation of viral DNA elimination.

cccDNA, covalently closed circular DNA; indels, insertions and deletions; mg/kg, milligram per kilogram; HBV, hepatitis B virus; W, week. IU/mL, international units per milliliter.

Measuring the Viral Source with Biopsy

Participant 5 underwent pre and post treatment biopsy



- Molecular biopsy data in Patient 5 was taken after 2 dose administrations and corresponds to a 44% decline in HBsAg
- After the third dose administration, Patient 5 has further reduced HBsAg to 67% from baseline

Given the durable and cumulative HBsAg reductions observed in Patient 7, we expect even greater editing rates have been achieved in Cohort 3 at 0.8 mg/kg

Next Steps: Planning for Progression Into Part 2 Expansion and Beyond

Complete Dosing for Cohort 3

All 3 doses expected to complete in Q1 2026

Stop NUCs, Test for Cure

Will consider stopping NUCs after observing two sustained, low HBsAg measurement and with other supporting clinical markers*

Expand to Part 2

Expect to dose up to 45 patients between Part 1 and 2 with paired biopsies

Site expansion underway to enable rapid enrollment

IN PARALLEL: Dosing Optimization

Given tolerability and durability of responses observed to date in Cohorts 2 and 3, evaluate 4-week dosing interval at 0.4 mg/kg and potential for additional dose administrations in parallel to completing doses in Cohort 3

HBsAg, hepatitis B surface antigen; IU/mL, international units per milliliter; mg/kg, milligram per kilogram; NUCs, nucleo(t)side analogs; Q, quarter.
*Other clinical markers to consider when stopping NUCs: No advanced liver disease, Undetectable HBV DNA, HBV RNA, and HBcrAg

What Does Success for ELIMINATE-B Look Like?

Two FDA Regulatory Pathways Support Advancement of Therapies in Hepatitis B

Current therapies in development have not been able to directly target cccDNA and hence the path towards approval has focused on functional cure through HBsAg loss

However, if able to eliminate cccDNA (the only source of HBV DNA), PBGENE-HBV has an additional potential path forward per FDA guidelines…

GUIDANCE DOCUMENT

Chronic Hepatitis B Virus Infection: Developing Drug 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

cccDNA, covalently closed circular DNA; DNA, deoxyribonucleic acid; FDA, Food and Drug Administration; HBsAb, hepatitis B s antigen; HBV, hepatitis B virus; IU/mL, international unit per milliliter; LLOQ, lower limit of quantification; TD, targeted direction; TND, target not directed.

1. FDA Guidance – Chronic Hepatitis B Virus Infection; Developing Drugs for Treatment; April 2022.

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functionDMD

PBGENE-DMD

Precision's lead muscle program utilizing novel gene excision approach for majority of patients with Duchenne Muscular Dystrophy (DMD)

PBGENE-DMD: A Novel Clinical Candidate with Advantages Beyond Existing Treatments

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

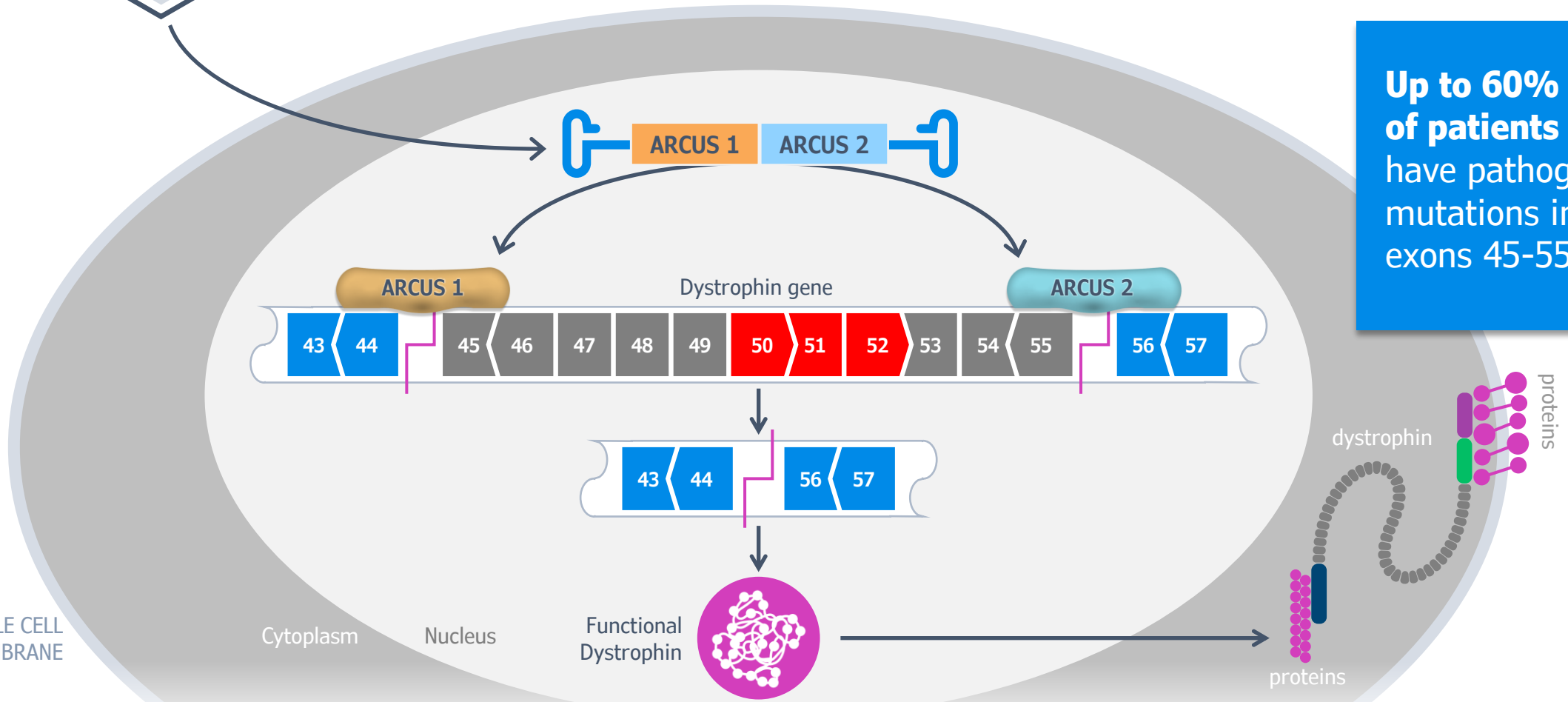
Limited Benefits

Patients with DMD are in dire need with limited therapeutic options

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

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

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

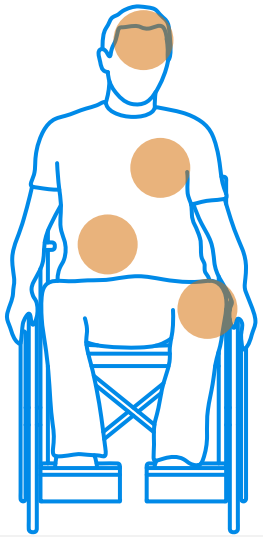


Up to 60% of patients have pathogenic mutations in exons 45-55[1]

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

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

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



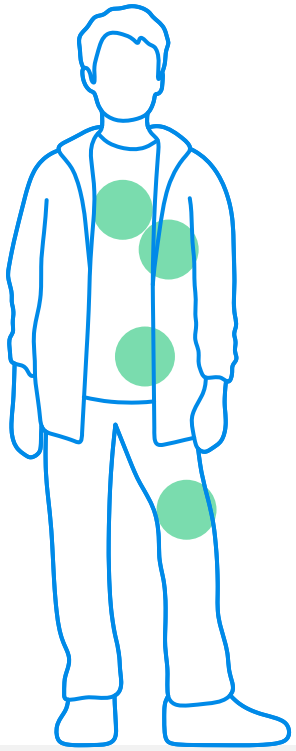
Lifespan:

- Early death in late teens or 20s

Clinical Presentation:

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

Out of frame dystrophin gene (DMD)



Lifespan:

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

Clinical Presentation:

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

Del45-55 in-frame Dystrophin gene (BMD)

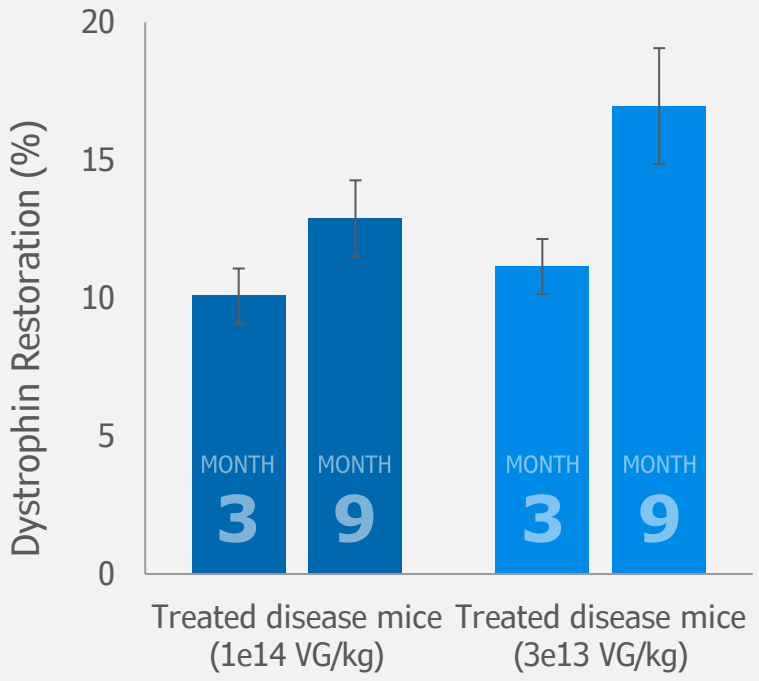
It is expected that as little as 5% expression of functional dystrophin protein is needed to provide therapeutic benefit in DMD patients[5]

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

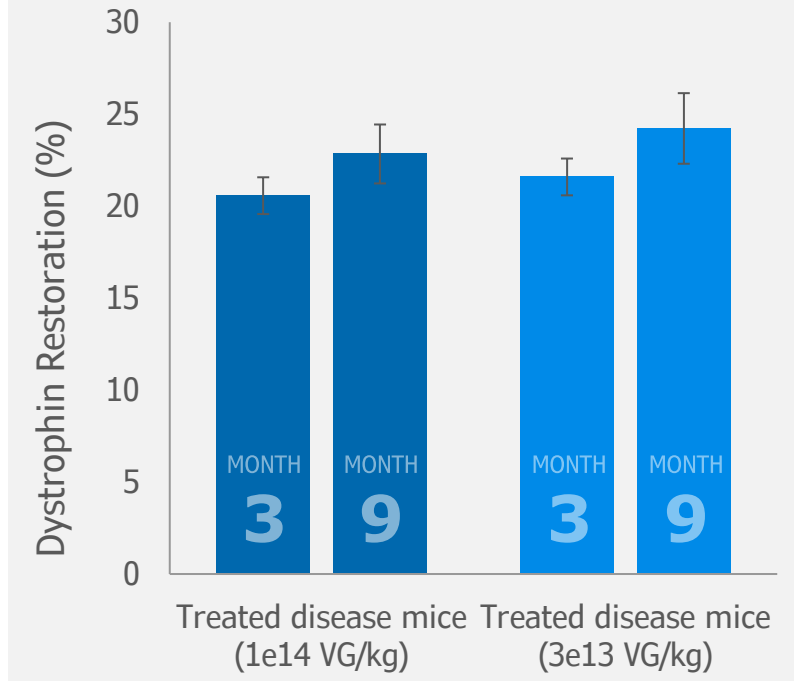
1. Taglia A, et al. Acta Myol. 2015;34(1):9-13. 2. Echigoya Y, et al. J Pers Med. 2018;8(4):41. 3. Béroud C, et al. Hum Mutat. 2007;28(2):196-202. 4. Nakamura A. J Hum Genet. 2017;62(10):871-876. 5. Feraudy et al. Ann Neurol, 2021 Feb;89(2):280-292.

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

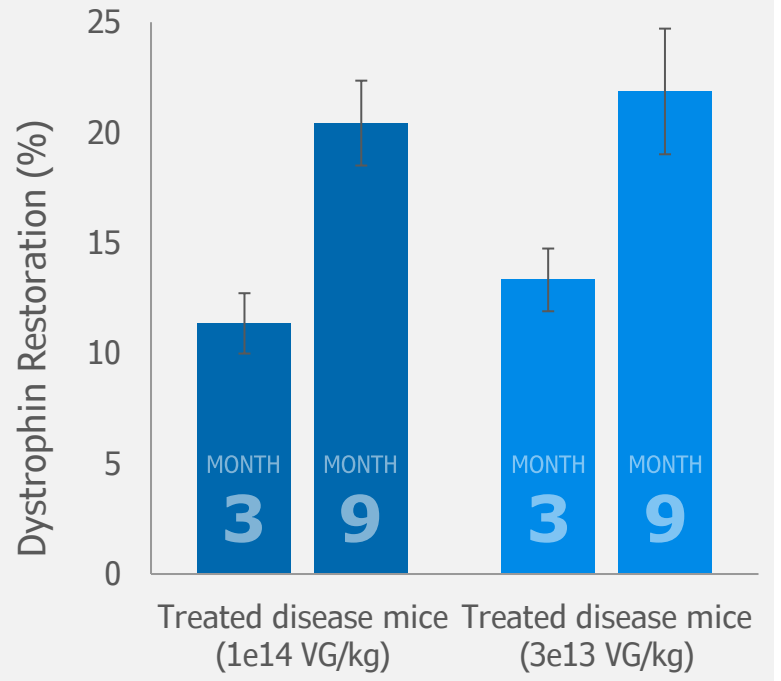
Heart



Calf



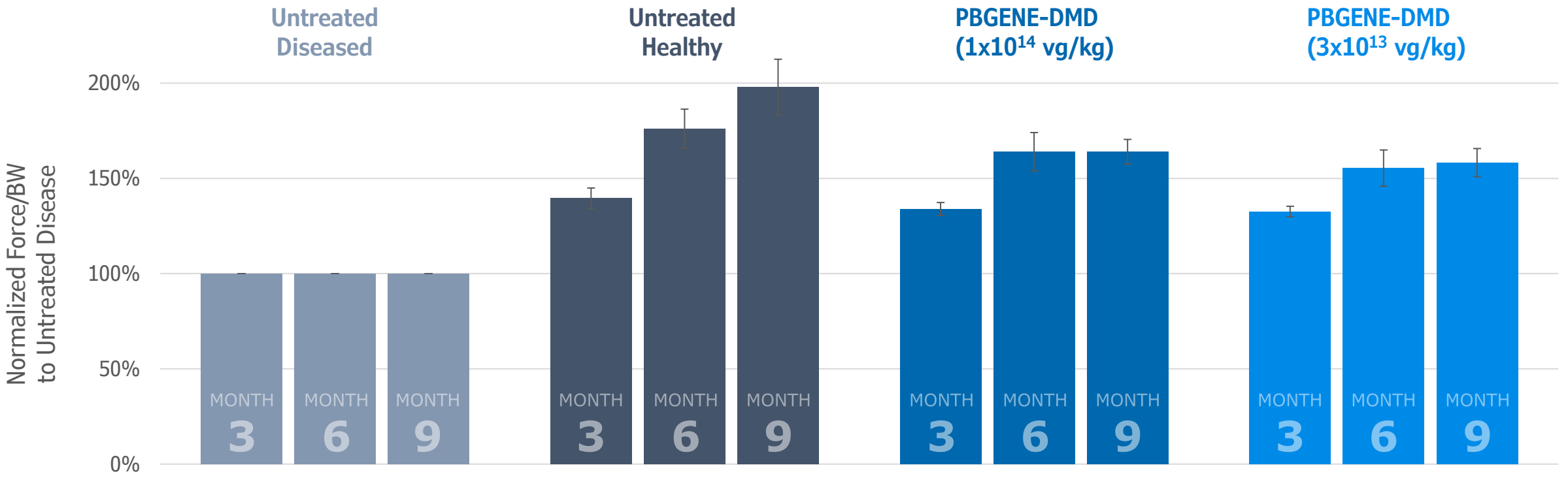
Quadricep



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

DMD, Duchenne Muscular Dystrophy; vg/kg, vector genomes per kilogram.
ASGCT 2025.

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



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

Force was measured in the calf across multiple stimulation frequencies. Averaged force normalized to bodyweight is shown. Statistically significant ($p<0.001$) increases in force were observed in both doses of PBGENE-DMD compared untreated diseased animals at both time points. N=10 mice per cohort.
DMD, Duchenne Muscular Dystrophy; vg/kg, vector genomes per kilogram.
ASGCT 2025.

Phase I/II/III Study Designed for Safety and Speed

Phase I/II – Establish Safety and Efficacy

Single-Dose Level Design
Final dose to be aligned with regulators (e.g., 1e14 vg/kg)

Plan:

- Enroll 5-8 ambulatory patients across multiple clinical trial sites in 2026
- After treating a total of 10-15 patients, meet with regulatory agencies to align on pivotal study and path forward
- Assess safety and efficacy through near full-length dystrophin% and functional measures

Move to Pivotal

Planned Pivotal Trial

- Treat a total of 35 to 40 patients across Phase I/II/III
- Targeted BLA filing in 2028

Keeping Safety Top of Mind

Employing an aggressive immune modulation regimen and safety monitoring program at world class DMD specialized sites to mitigate any potential AAV safety risks

1H, first half; AAV, adeno-associated virus; BLA, biologics license application; DMD, Duchenne Muscular Dystrophy; vg/kg, vector genomes per kilogram.
IND/clinical trial application for PBGENE-DMD pending regulatory submission and clearance prior to initiation of clinical program.

Successful $75M Financing Fuels Further Development of Lead Programs While Generating Shareholder Value Through Multiple Clinical Milestones Expected Over Next 12-24 Months

Wholly owned In Vivo Gene Editing Programs

		Expected First Clinical Data Timing
PBGENE-HBV **Phase 1/2a Program for Chronic Hepatitis B**	〉 First-in-Class Mechanism targeting cccDNA elimination 〉 Well Tolerated Safety Profile even with repeat dosing 〉 Active Drug Across Doses and HBsAg Levels 〉 Durable responses in 7 of 9 patients 〉 **Near-term path towards stopping NUCs**	**2025** eliminate B
PBGENE-DMD **Duchenne Muscular Dystrophy**	〉 PBGENE-DMD is designed to provide durable functional muscle improvement **for ~60% of patients with DMD** 〉 PBGENE-DMD significantly **improved muscle function** and demonstrated **long-term durability** in DMD mouse model 〉 Targeting IND filing in late 2025 and dosing patients in 2026 with BLA filing in 2028	**2026** functionDMD

Multiple Clinical Datasets Expected to Read-Out within Extended Current Cash Runway through 2028[1]

Financing with Top Tier Fundamental Investors Expected to Fuel Further Development of PBGENE-HBV through Phase 2 Study and to PBGENE-DMD BLA Submission

cccDNA, covalently closed circular DNA; DMD, Duchenne Muscular Dystrophy; FDA, Food and Drug Administration; H1, first-half of the year; H2, second-half of the year; HBV, Hepatitis B virus; HBsAg, Hepatitis B surface antigen; IND, investigational new drug; MHRA, Medicines & Healthcare products Regulatory Agency; NUCs, nucleos(t)ide analogs

1. $71.2M cash and restricted cash as of 9/30/25. The Company expects existing cash and cash equivalents, its recent financing, potential near-term cash from CAR T transactions, along with expected operating efficiencies, operational receipts, and availability of Precision's at-the-market (ATM) facility to extend Precision's cash runway through 2028

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Appendix

Treatment Goal Today:
≥30% Functional Cure Rate Set By HBV Experts Has Remained Elusive

Review > J Hepatol. 2020 Mar;72(3):539-557. doi: 10.1016/j.jhep.2019.11.003.
Epub 2019 Nov 12.

Guidance for design and endpoints of clinical trials in chronic hepatitis B - Report from the 2019 EASL-AASLD HBV Treatment Endpoints Conference‡

Markus Cornberg [1], Anna Suk-Fong Lok [2], Norah A Terrault [3], Fabien Zoulim [4];
2019 EASL-AASLD HBV Treatment Endpoints Conference Faculty

PMID: 31730789 DOI: 10.1016/j.jhep.2019.11.003

Abstract

Representatives from academia, industry, regulatory agencies, and patient groups convened in March 2019 with the primary goal of developing agreement on chronic HBV treatment endpoints to guide clinical trials aiming to 'cure' HBV. Agreement among the conference participants was reached on some key points. 'Functional' but not sterilising cure is achievable and should be defined as sustained HBsAg loss in addition to undetectable HBV DNA 6 months post-treatment. The primary endpoint of phase III trials should be functional cure; HBsAg loss in ≥30% of patients was suggested as an acceptable rate of response in these trials. Sustained virologic suppression (undetectable serum HBV DNA) without HBsAg loss 6 months after discontinuation of treatment would be an intermediate goal. Demonstrated validity for the prediction of sustained HBsAg loss was considered the most appropriate criterion for the approval of new HBV assays to determine efficacy endpoints. Clinical trials aimed at HBV functional cure should initially focus on patients with HBeAg-positive or negative chronic hepatitis, who are treatment-naïve or virally suppressed on nucleos(t)ide analogues. A hepatitis flare associated with an increase in bilirubin or international

AASLD, American Association for the Study of Liver Diseases; DNA, deoxyribonucleic acid; EASL, European Professional Organization for Hepatology; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus.

Enrolling a Real-World Population of HBeAg Negative Patients Across Range of Key Demographics and Baseline Characteristics

	Cohort 1 (0.2 mg/kg)		
	Patient 1	Patient 2	Patient 3
Sex	Male	Male	Male
Age (years)	40	39	44
Ethnicity/Race	Caucasian	Caucasian	Caucasian
Region of Origin	Eastern Europe	Eastern Europe	Eastern Europe
Time with HBV (years)	9	39	8
Time on NUCs (years)	6	7	7
Baseline HBsAg (IU/mL)	562	11,813	788
HBV Treatment (NUCs)	TDF	TDF	TDF
Medical History	Gilbert Syndrome*	Obesity	None

*Participant 1 was diagnosed with Gilbert Syndrome-a common, benign genetic disorder where the liver processes bilirubin, more slowly than usual, leading to slightly elevated levels in the blood.

HBsAg, Hepatitis B surface antigen; HBV, hepatitis B virus; IU, international units; mg/kg, milligram per kilogram; mL, milliliter; NUCs, nucleo(t)side analogs; TDF, tenofovir disoproxil fumarate.

Enrolling a Real-World Population Across Range of Key Demographics and Baseline Characteristics

	Cohort 2 (0.4 mg/kg)		
	Patient 4	**Patient 5**	**Patient 6**
Sex	Male	Male	Male
Age (years)	50	45	52
Ethnicity/Race	Asian	Asian	Asian
Region of Origin	Asia	Asia	Asia
Time with HBV (years)	34	37	26
Time on NUCs (years)	25	16	4
Baseline HBsAg (IU/mL)	1,402	3,159	827
HBV Treatment (NUCs)	TDF	ETV	ETV
Medical History	Liver cyst and hemangioma	Fatty liver, obesity, hypertension	None

ETV, entecavir; HBsAg, Hepatitis B surface antigen; HBV, hepatitis B virus; IU, international units; mg/kg, milligram per kilogram; mL, milliliter; NUCs, nucleo(t)side analogs; TDF, tenofovir disoproxil fumarate.

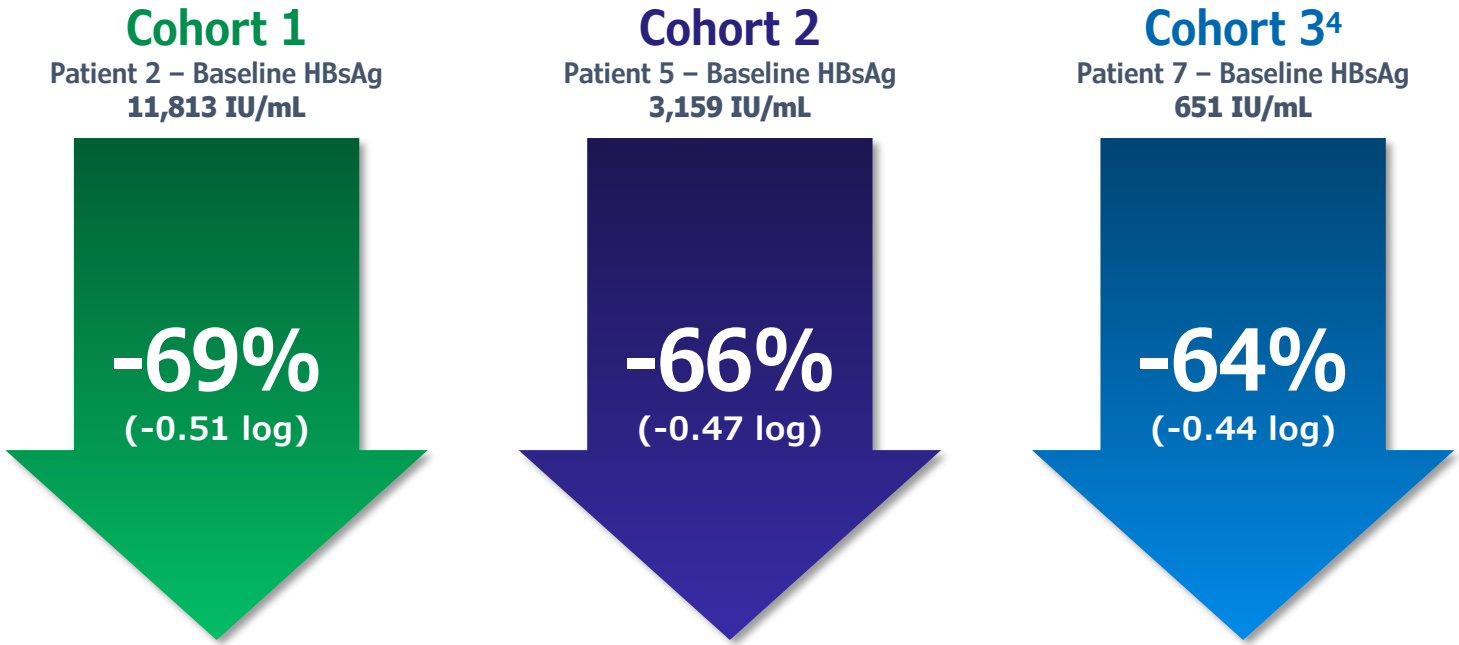
Enrolling a Real-World Population Across Range of Key Demographics and Baseline Characteristics

	Cohort 3 (0.8 mg/kg)		
	Patient 7	**Patient 8**	**Patient 9**
Sex	Male	Male	Male
Age (years)	64	53	54
Ethnicity/Race	Asian	Native Hawaiian or Other Pacific Islander	Asian
Region of Origin	Asia	Polynesia	Asia
Time with HBV (years)	32	13	19
Time on NUCs (years)	16	8	7
Baseline HBsAg (IU/mL)	651	420	370
HBV Treatment (NUCs)	ETV	ETV	ETV
Medical History	Hyperlipidemia	Hypertension, Hyperlipidemia	Hyperlipidemia

ETV, entecavir; HBsAg, Hepatitis B surface antigen; HBV, hepatitis B virus; IU, international units; mg/kg, milligram per kilogram; mL, milliliter; NUCs, nucleo(t)side analogs; TDF, tenofovir disoproxil fumarate.

PBGENE-HBV Activity Independent of HBsAg Baseline Levels

HBsAg Change From Baseline at Nadir During Course of Treatment for Best Responder



Note natural HBsAg fluctuations including assay variability expected to be ~20-25% for this population[1-3]

Activity across a broad range of baseline HBsAg levels, up to 11,813 IU/mL

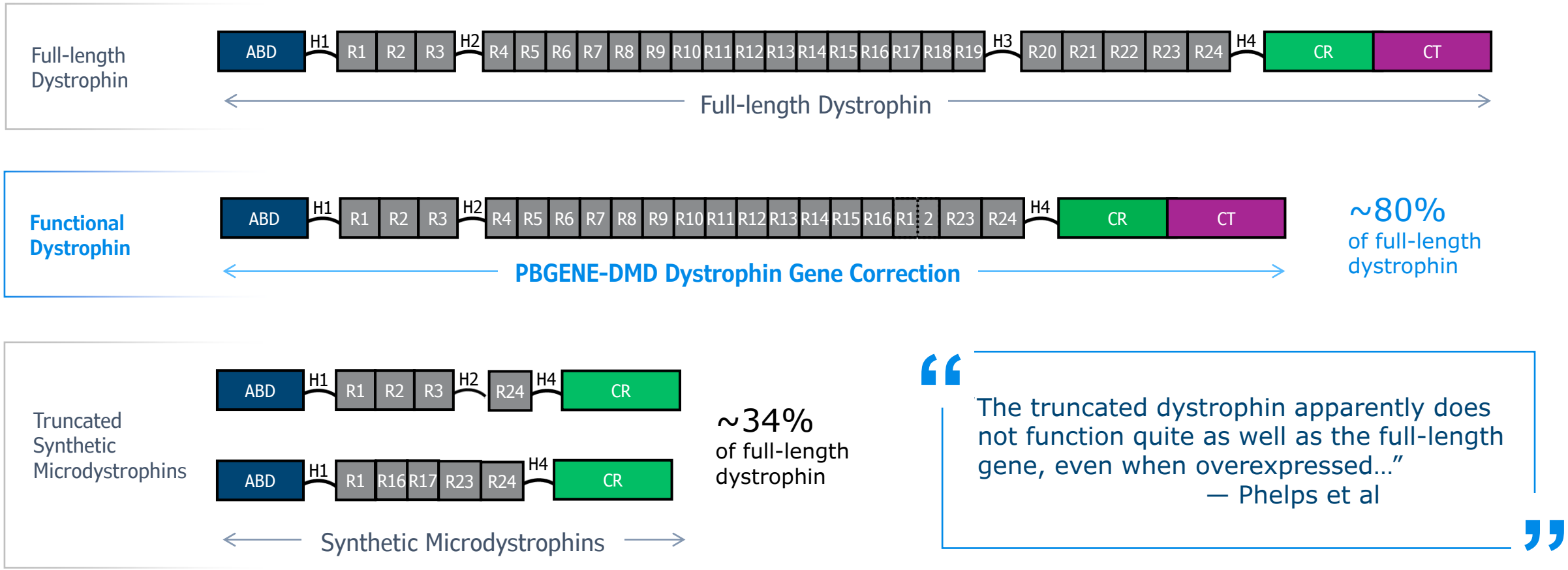
HBsAg, Hepatitis B surface antigen; IU/mL, international units per milliliter.

1. Lee HA, et al. Clin Mol Hepatol 2016;22:382-389. 2. Yeo YH, et al. Gastroenterology. 2019;156:635-646. 3.Elecsys HBsAg II quant II Roche Diagnostics GmbH, Germany. Package Insert, Version 2.0. June 2020. https://assets.roche.com/f/173850/x/29ae54d551/can-pi-elecsys-hbsag-ii-quant-ii-08814899190-v2-en.pdf . Accessed July 30, 2025.

4. Patient 7 only through 2 dose administration as of data cutoff date of October 31, 2025.

Summary

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



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

DMD, Duchenne Muscular Dystrophy.
Phelps et al. *Hum Mol Genet.* 1995 Aug;4(8):1251-8.