

*EASL 2026 Investor Update:
PBGENE-HBV ELIMINATE-B Program*

May 27, 2026



Forward-Looking Statements

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. All statements contained in this presentation that do not relate to matters of historical fact should be considered forward-looking statements, including, without limitation, statements regarding PBGENE-HBV being the only program designed to permanently eliminate cccDNA and inactivate integrated HBV DNA; expectations around HBV at a turning point from lifelong suppression toward finite, biomarker-guided viral cure; statements regarding PBGENE-HBV's primary mechanism for eliminating cccDNA and inactivating integrated HBV DNA, secondary mechanism of indels resulting in complete viral inactivation in any edited DNA and reducing HBsAg from both cccDNA and integrated DNA; statements that late-breaking data prove PBGENE-HBV directly targets and eliminates cccDNA providing a path toward cure and FDA approval; statements that pgRNA is the appropriate blood biomarker and is unique to cccDNA elimination in the blood; statements that the field has been focused on the wrong target and biomarker in cHBV; expectations about FDA guidance focused on destruction of HBV DNA as a key or required element of the regulatory pathway and approval; expectations about operational initiatives, strategies, further development, or timing of additional updates or data releases of PBGENE-HBV; statements that undetectable viral DNA equals potential cure and lack of relapse; expectations around PBGENE-HBV's target product profile; statements that studies demonstrate undetectable pgRNA is associated with success of stopping nucleoside analogs and increasing probability of cure; expectations around the durability of pgRNA loss and sustained HBsAg reduction in patients treated with PBGENE-HBV; expectations around permanent and/or cumulative gene editing in patients treated with PBGENE-HBV; statements that liver biopsies in combination with key biomarkers reflect cccDNA elimination and support increased probability of cure after treatment with PBGENE-HBV; expectations that the etiology of hypotension is now understood and simple measures have ameliorated clinically significant decreases in blood pressure; expectations that ALT/AST lab abnormalities were transient and asymptomatic not clinically significant; statements that the tolerability of PBGENE-HBV has improved after implementing new safety mitigations; expectations that the Company will expand the number of patients in cohorts 4 and 5 and enroll patients in France and Romania; expectations that additional liver biopsies will be collected to support the undetectable pgRNA biomarker with the goal to establish PBGENE-HBV as transformational for viral cure; expectations around confirming global alignment for nucleoside analog withdrawal framework with clinicians around pgRNA loss for ≥ 6 months, normalized liver enzymes and sustained reductions in HBsAg; expectations around evaluating current patients with pgRNA loss ≥ 6 months for nucleoside analog withdrawal and expansion to pgRNA negative patients after viral cure proven in those with pgRNA loss; expectations around selecting the optimal dosing schedule for PBGENE-HBV phase I expansion and Phase 2; statements around broadening to an enriched pgRNA+ patient population, HBeAg+ patients and continued assessment of PBGENE-HBV as potential viral cure via monotherapy and combination; expectations around an HBV turning point from lifelong suppression to finite, biomarker-guided viral cure; statements that the next PBGENE-HBV update will take place by year-end 2026; and statements that PBGENE-DMD is designed to provide durable functional muscle improvement targeting ~60% of patients with DMD and next steps to execute in the clinic through 2026 with initial clinical data expected end of year. In some cases, you can identify forward-looking statements by terms such as "aim," "anticipate," "approach," "belief," "believe," "contemplate," "could," "design," "designed," "estimate," "expect," "goal," "intend," "look," "may," "mission," "path", "plan," "possible," "potential," "predict," "project," "pursue," "should," "strive," "suggest," "target," "toward", "will," "would," or the negative thereof and similar words and expressions

Forward-looking statements are based on management's current expectations, beliefs, and assumptions and on information currently available to us. These statements are neither promises nor guarantees, and involve a number of known and unknown risks, uncertainties and assumptions, and actual results may differ materially from those expressed or implied in the forward-looking statements due to various important factors, including, but not limited to, our ability to become profitable; our ability to procure sufficient funding to advance our programs; risks associated with our capital requirements, anticipated cash runway, requirements under our current debt instruments and effects of restrictions thereunder, including our ability to raise additional capital due to market conditions and/or our market capitalization; our operating expenses and our ability to predict what those expenses will be; our limited operating history; the progression and success of our programs and product candidates in which we expend our resources; our limited ability or inability to assess the safety and efficacy of our product candidates; the risk that other genome-editing technologies may provide significant advantages over our ARCUS technology; our dependence on our ARCUS technology; the initiation, cost, timing, progress, achievement of milestones and results of research and development activities and preclinical and clinical studies, including clinical trial and investigational new drug applications; public perception about genome editing technology and its applications; competition in the genome editing, biopharmaceutical, and biotechnology fields; our or our collaborators' or other licensees' ability to identify, develop and commercialize product candidates; pending and potential product liability lawsuits and penalties against us or our collaborators or other licensees related to our technology and our product candidates; the U.S. and foreign regulatory landscape applicable to our and our collaborators' or other licensees' development of product candidates; our or our collaborators' or other licensees' ability to advance product candidates into, and successfully design, implement and complete, clinical trials; potential manufacturing problems associated with the development or commercialization of any of our product candidates; delays or difficulties in our and our collaborators' and other licensees' ability to enroll patients; changes in interim "top-line" and initial data that we announce or publish; if our product candidates do not work as intended or cause undesirable side effects; risks associated with applicable healthcare, data protection, privacy and security regulations and our compliance therewith; our or our licensees' ability to obtain orphan drug designation or fast track designation for our product candidates or to realize the expected benefits of these designations; our or our collaborators' or other licensees' ability to obtain and maintain regulatory approval of our product candidates, and any related restrictions, limitations and/or warnings in the label of an approved product candidate; the rate and degree of market acceptance of any of our product candidates; our ability to effectively manage the growth of our operations; our ability to attract, retain, and motivate executives and personnel; effects of system failures and security breaches; insurance expenses and exposure to uninsured liabilities; effects of tax rules; effects of any pandemic, epidemic, or outbreak of an infectious disease; the success of our existing collaboration and other license agreements, and our ability to enter into new collaboration arrangements; our current and future relationships with and reliance on third parties including suppliers and manufacturers; our ability to obtain and maintain intellectual property protection for our technology and any of our product candidates; potential litigation relating to infringement or misappropriation of intellectual property rights; effects of natural and manmade disasters, public health emergencies and other natural catastrophic events; effects of sustained inflation, supply chain disruptions and major central bank policy actions; market and economic conditions; risks related to ownership of our common stock, including fluctuations in our stock price; our ability to meet the requirements of and maintain listing of our common stock on Nasdaq or other public stock exchanges; and other important factors discussed under the caption "Risk Factors" in our Quarterly Report on Form 10-Q for the quarterly period ended March 31, 2026, as any such factors may be updated from time to time in our other filings with the SEC, which are accessible on the SEC's website at www.sec.gov and the Investors page of our website under SEC Filings at investor.precisionbiosciences.com.

All forward-looking statements speak only as of the date of this presentation and, except as required by applicable law, we have no obligation to update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise.



EASL 2026 Investor Update: PBGENE-HBV ELIMINATE-B Program



Michael Amoroso

President & Chief Executive Officer
Precision BioSciences, Inc.



Key Hepatitis Expert Presenters and Panelists



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



Mark Sulkowski, MD

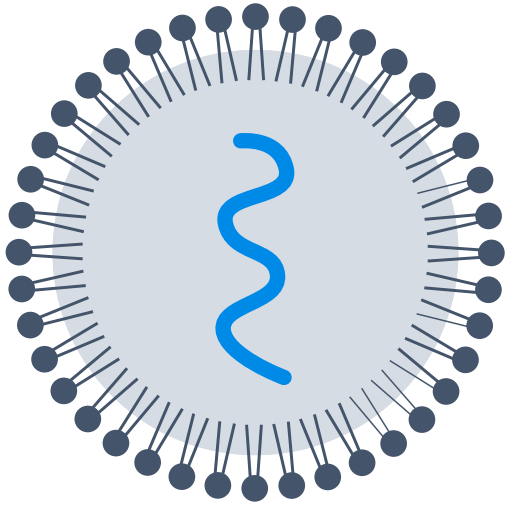
Professor of Medicine
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Cassie Gorsuch, PhD

Chief Scientific Officer
Precision BioSciences, Inc.





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

“True cure of HBV infection requires eradication, degradation ...of cccDNA ...”¹

— Dusheiko, NEJM 2023

“...assessing response to chronic hepatitis B therapies is unclear because of inconsistent correlations between HBsAg and clinical response.”²

— FDA Guidance 2022

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

— Trucchio, Sell-Side Analyst



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



Current Therapeutics Rarely Achieve Cure Because They Do Not Eliminate cccDNA

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



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

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



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

PATHWAYS TO FDA APPROVAL¹

GUIDANCE DOCUMENT

Chronic Hepatitis B Virus Infection: Developing Drugs for Treatment

APRIL 2022

1

HBV DNA
undetectable
only

OR

2

HBV DNA +
HBsAg both
undetectable



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

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

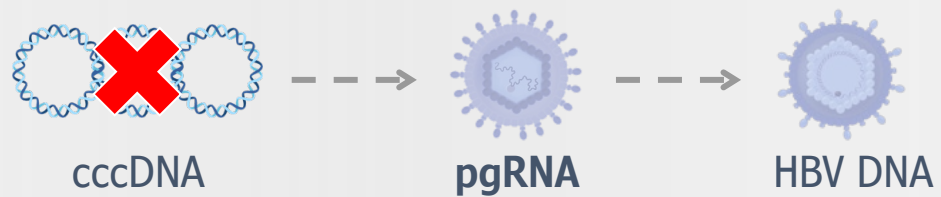
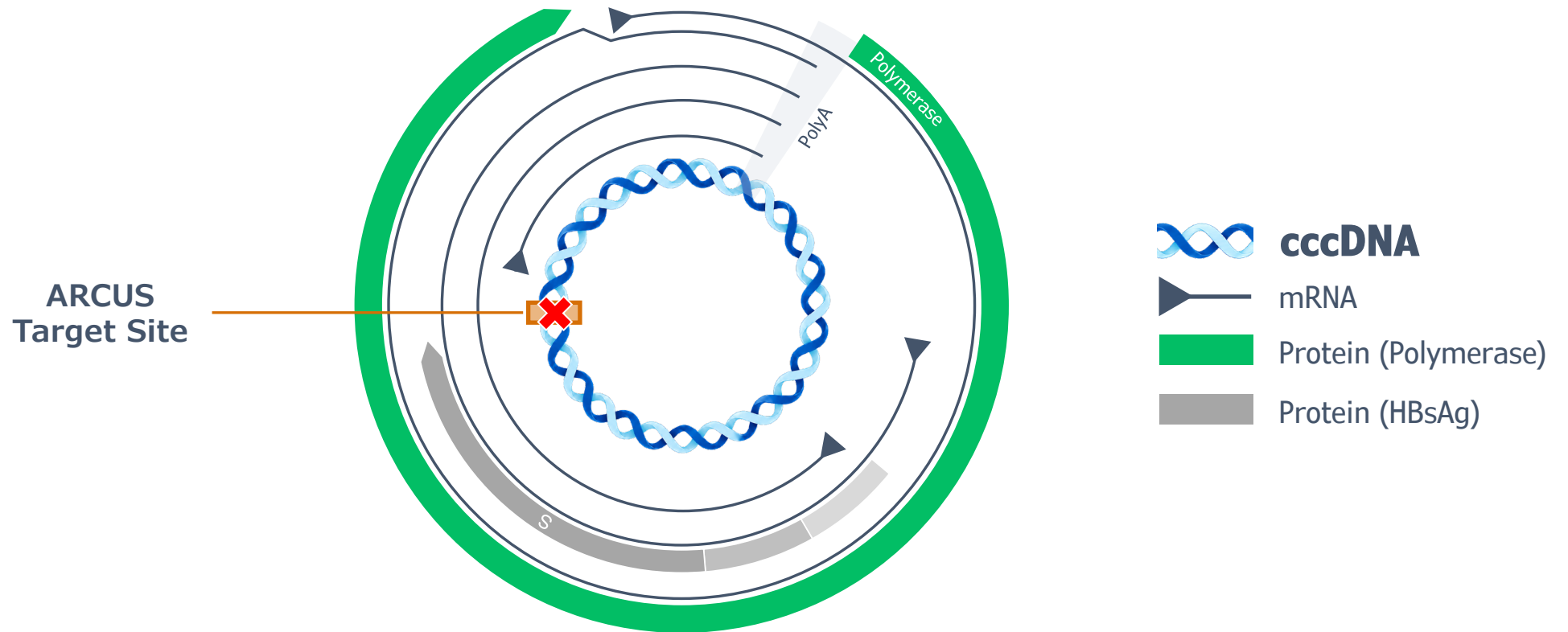


cccDNA, covalently closed circular DNA; FDA, US Food and Drug Administration; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus.

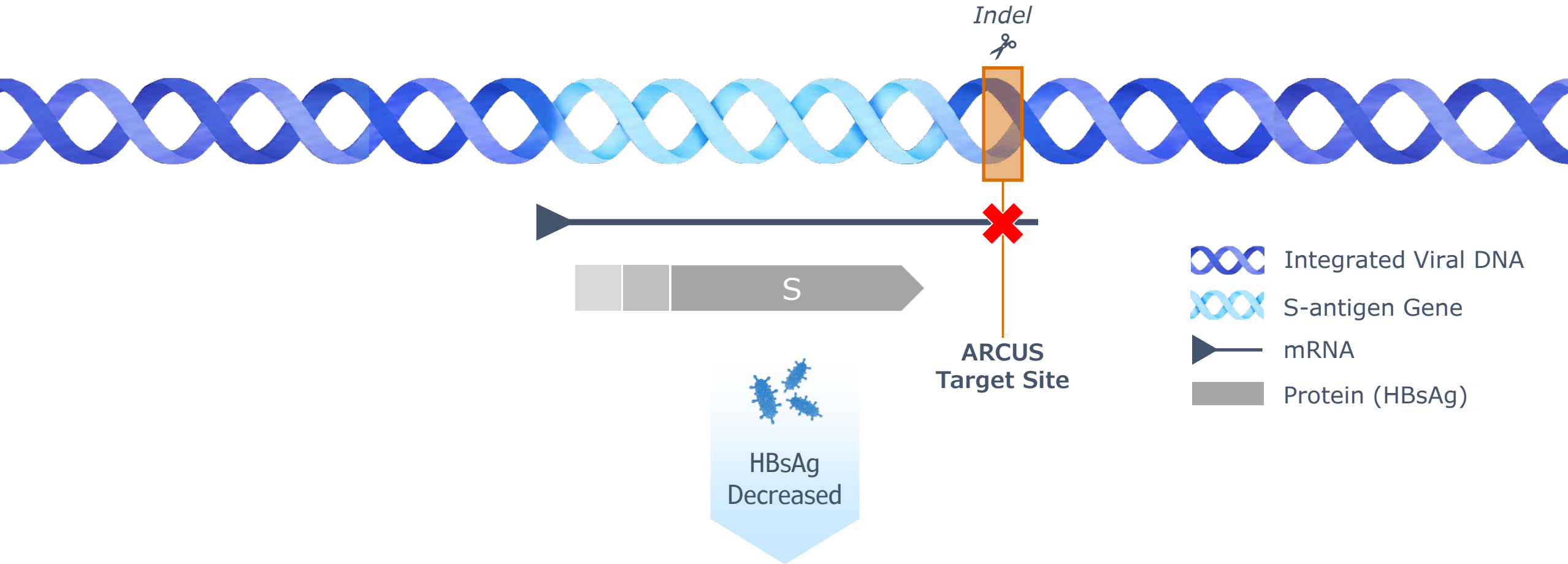
1. FDA Guidance – Chronic Hepatitis B Virus Infection; Developing Drugs for Treatment; April 2022. Accessed April 30, 2026. 2. Hu B, et al. *J Gastroenterol Hepatol.* 2018;33(7):1389-1396.

3. Thompson AJ, et al. *Hepatology.* 2010;51(6):1933-1944. 4. Chan HL, et al. *J Hepatol.* 2011;55(5):1121-1131.

PBGENE-HBV Primary Mechanism: Directly Targets and Eliminates cccDNA



PBGENE-HBV Secondary Mechanism: Also Targets and Reduces HBsAg, a Replication-Incompetent Protein, by Editing Integrated Viral DNA



LATE-BREAKING DATA Prove PBGENE-HBV Directly Targets and Eliminates cccDNA, Providing Path Toward Viral Cure

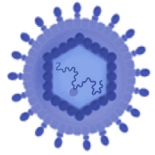


- › **Primary antiviral mechanism established** through multiple biopsies, confirming targeting and elimination of cccDNA
- › **Potent cccDNA targeting drives direct antiviral effect**
 - **10-fold (1-log) reduction in cccDNA**-derived transcripts in **liver biopsies**
 - Additionally, edits in remaining cccDNA **permanently inactivate** viral replication through disruption of polymerase function
- › Biopsy evidence of **cumulative antiviral cccDNA effect** after repeat PBGENE-HBV administrations

 PBGENE-HBV direct viral targeting mechanism directly aligns to FDA approval through loss of HBV DNA

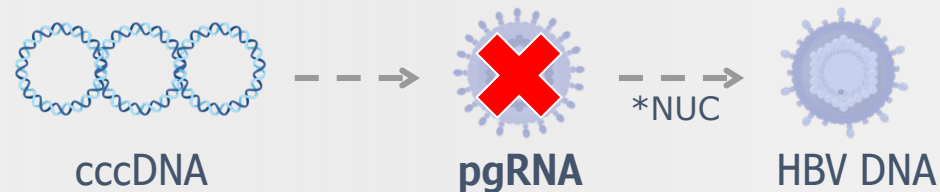


NEW EASL DATA: pgRNA Is the Appropriate Blood Biomarker as it Directly Comes From cccDNA and Is the Necessary Precursor of HBV DNA Replication

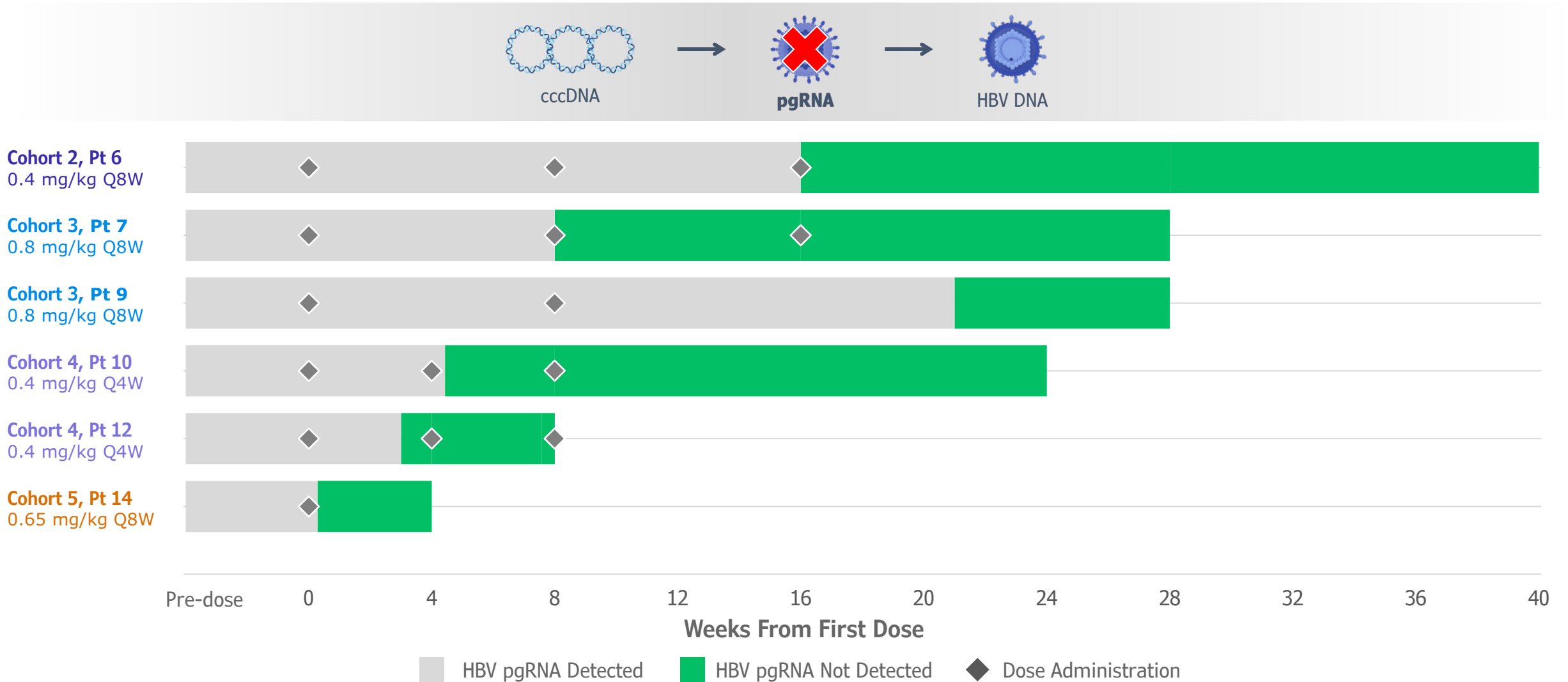


Based on Clinical Biopsy and Biomarker Data, PBGENE-HBV's Primary Mechanism of Eliminating cccDNA Results in Durable pgRNA Loss

- › Loss of blood pgRNA in 100% of patients (6/6)
 - Detectable (pre-treatment) → undetectable (post-treatment with PBGENE-HBV)
- › Loss of pgRNA in blood corresponded to undetectable pgRNA in post-PBGENE-HBV-treated liver biopsy
- › Innovative mechanism = distinct biomarker
 - pgRNA is the **APPROPRIATE** marker for PBGENE-HBV elimination and inactivation of cccDNA when controlled on NUCs

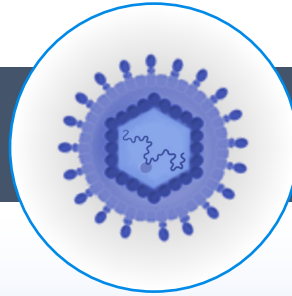


NEW EASL DATA: PBGENE-HBV Drove Durable pgRNA Loss in 100% of Patients Who Had Detectable pgRNA at Baseline*



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

Destroying Hepatitis at Its Source Is Foundational to Viral Cure



Hepatitis C Virus
RNA Virus

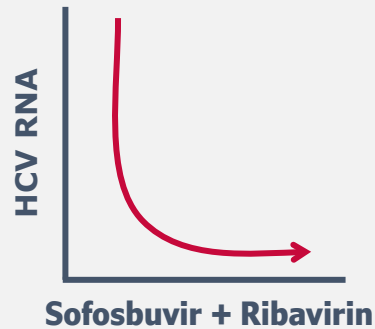
Hepatitis B Virus
DNA Virus

SOVALDI® (sofosbuvir)¹

PBGENE-HBV

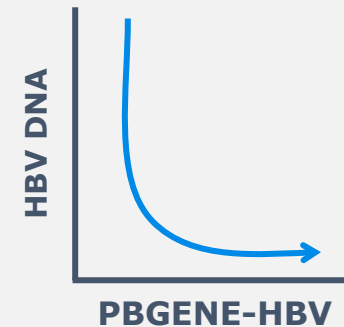


HCV RNA Loss
correlates to sustained virologic response



**Undetectable
Levels of Virus
=
Lack of Relapse
& Potential Cure**

**Direct Elimination of
HBV cccDNA**



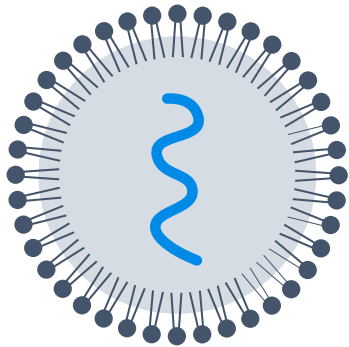
*Note: HBV DNA is not detectable on NUCs



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Graphs are for illustrative purposes only.
cccDNA, covalently closed circular DNA; HCV, hepatitis C virus; NUC, nucleos(t)ide analog; pgRNA, pregenomic RNA.
1. Gane EJ, et al. *N Engl J Med.* 2013;368(1):34-44. 2. Wang DH, et al. *J Viral Hepat.* 2026;33(4):e70167.

PBGENE-HBV Product Profile: Designed to Directly Target and Eliminate cccDNA and Drive Viral Cure

PBGENE-HBV Target Product Profile



A curative antiviral therapy would achieve the following:

- › **Directly target & destroy cccDNA** – the only source of new infectious virus (HBV DNA)
- › **Permanent mechanism with finite treatment course resulting in durable benefit**
 - cccDNA must stay eliminated
- › **MOA removes viral precursor (pgRNA) directly linked to viral source – cccDNA**
- › Maintain **predictable and manageable safety profile**
- › **Applicable for patients across all phases** of chronic hepatitis B natural history—comparable treatment effectiveness regardless of baseline disease load



PBGENE-HBV Primary Mechanism of Targeting cccDNA Is Now Validated



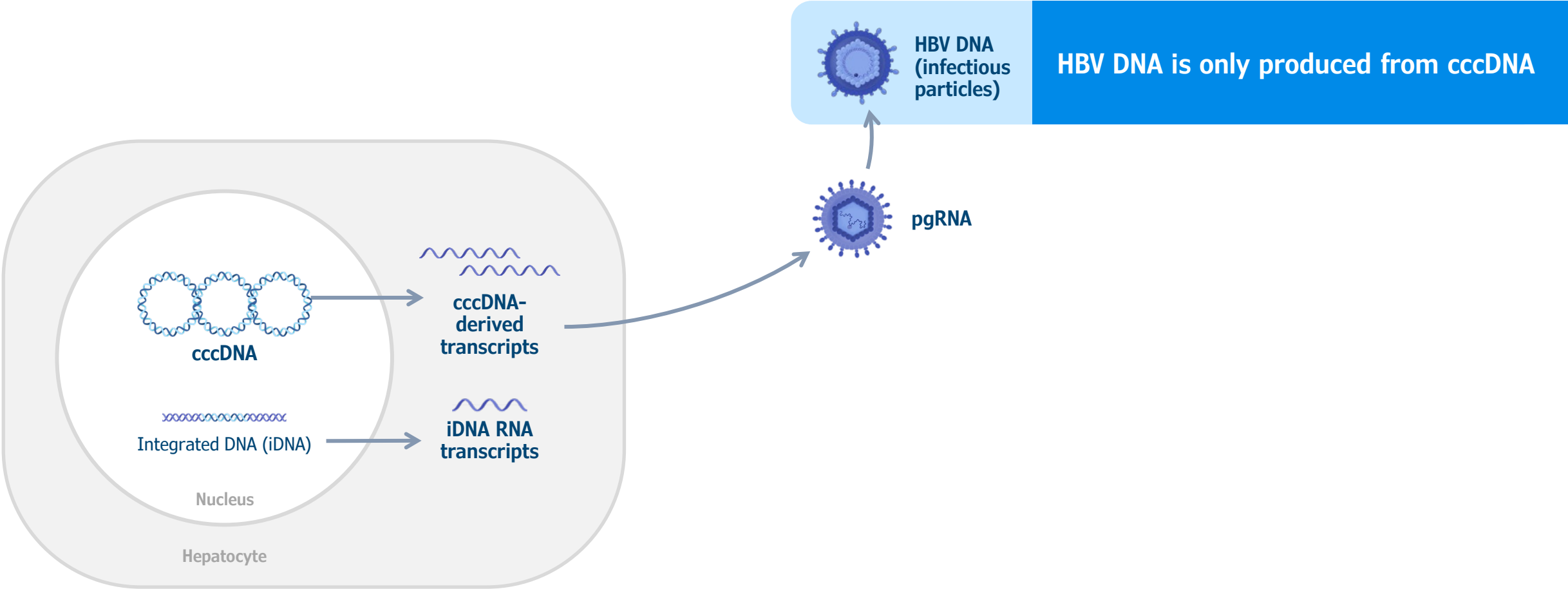
Cassie Gorsuch, PhD
Chief Scientific Officer
Precision BioSciences, Inc.



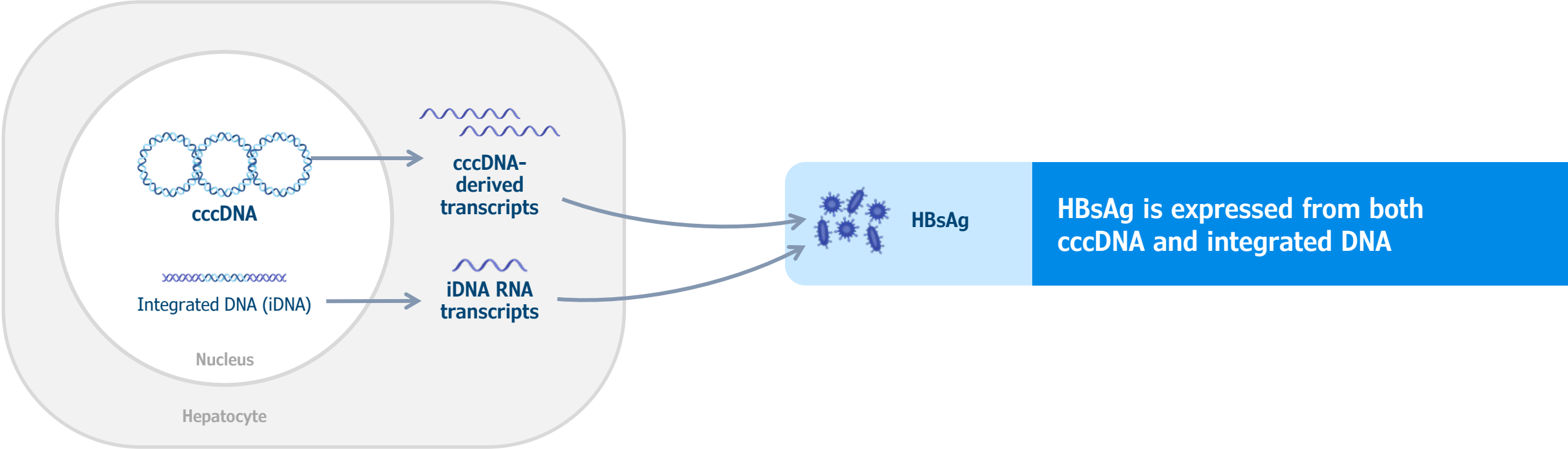
Chronic Hepatitis B Disease Biology



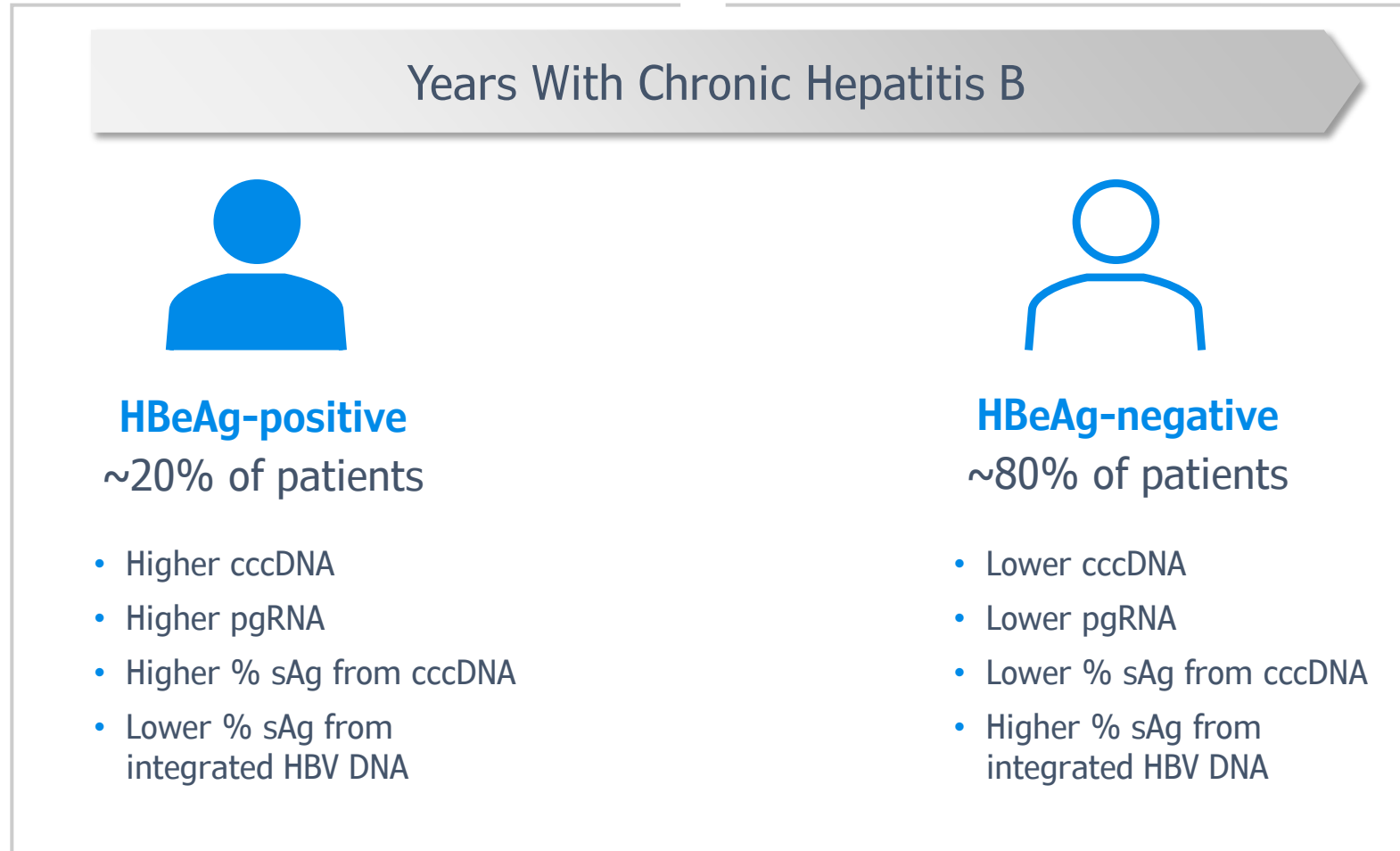
cccDNA Is the Only Source of New Infectious Virus (HBV DNA) and the Therapeutic Target to Cure Hepatitis B



Integrated DNA Cannot Produce HBV DNA (Infectious Virus) and Therefore Is Less Important Than cccDNA



In Fact, in HBeAg-Negative Patients, HBsAg Is Primarily Produced From iDNA, Limiting Its Utility as a Biomarker for Antiviral Effect on cccDNA



The Problem: Because the Field Lacked Tools Directly Targeting cccDNA, We've Been Focused on the Wrong Target and Biomarker

- › Hepatitis B is a viral infection in the liver that can cause cirrhosis and liver cancer
- › cccDNA is the source of the viral infection
- › The goal of therapy MUST be to eliminate cccDNA
- › Much of the clinical development work in chronic hepatitis B has focused on HBsAg protein targeting and reduction, which does not lead to a viral cure
- › cccDNA is not the primary source of HBsAg in most patients (HBeAg-negative), and HBsAg isn't specific to cccDNA

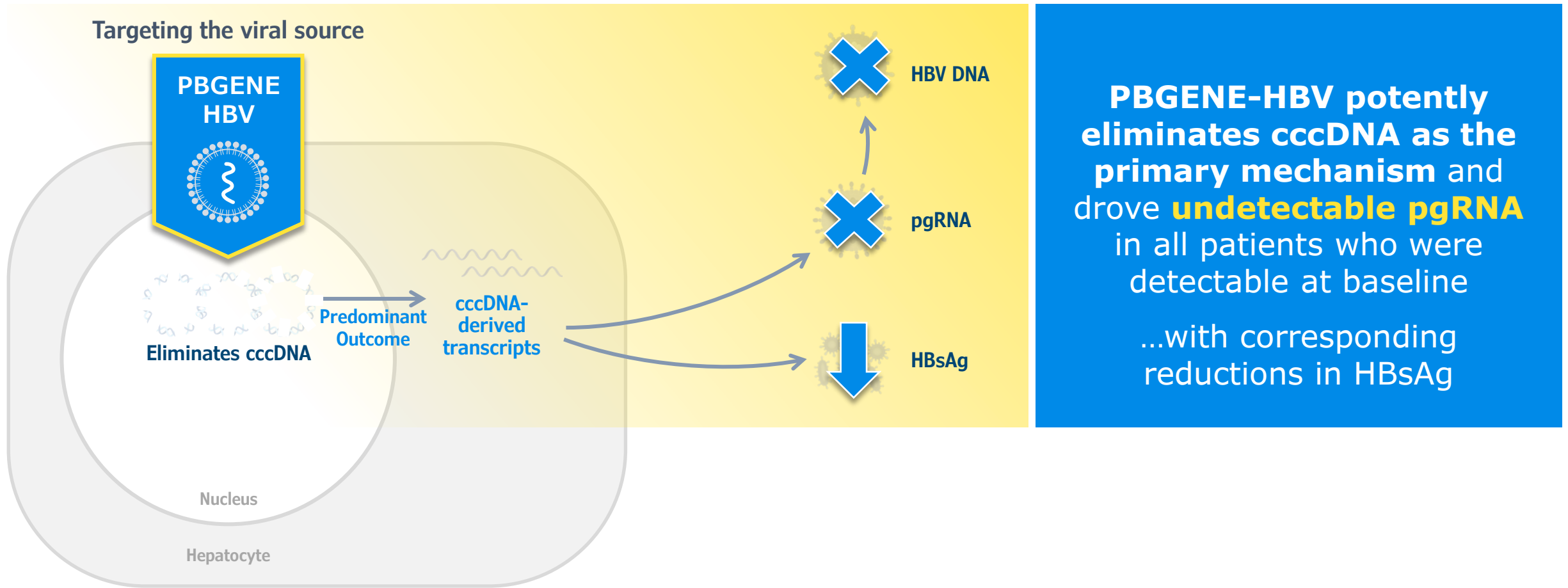
We now have a therapeutic approach that directly eliminates cccDNA – PBGENE-HBV



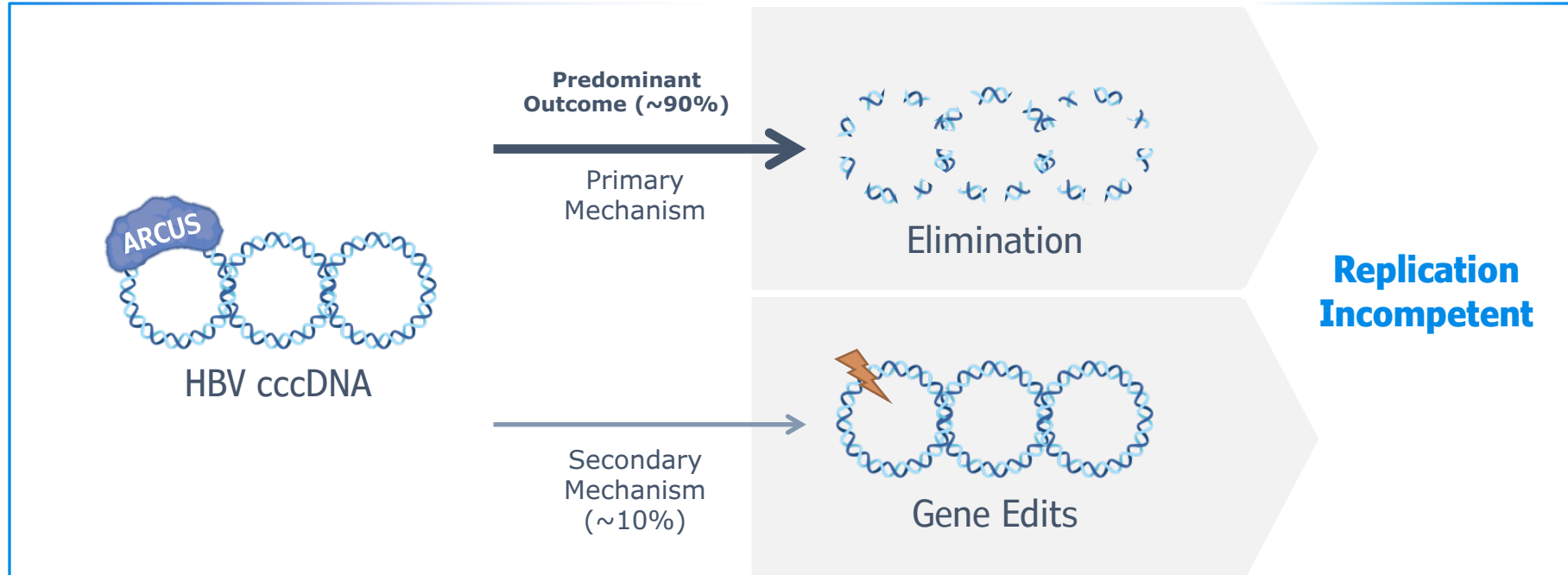
PBGENE-HBV Mechanism of Action



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



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



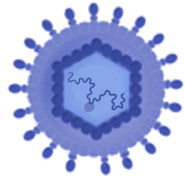
The Reason Why:

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

Both editing outcomes result in replication incompetent virus.

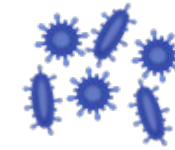


For a Direct Targeting cccDNA Mechanism Like PBGENE-HBV, pgRNA Is the Specific Blood Biomarker for Measuring Elimination of cccDNA¹⁻²



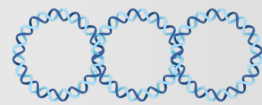
Source of pgRNA/HBV DNA

100% from cccDNA
0% from integrated DNA (iDNA)

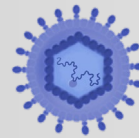


Source of HBsAg

<10% from cccDNA
>90% from integrated DNA (iDNA)

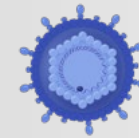


cccDNA



pgRNA

*NUC

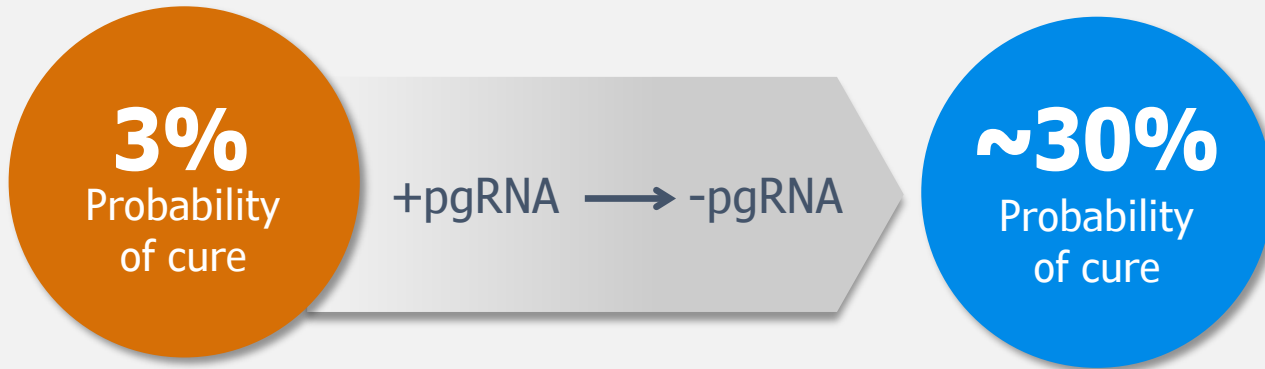


HBV DNA



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

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



Adapted from Terrault NA, et al.

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

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

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

pgRNA outperforms other biomarkers (eg, HBsAg) in predicting the effective and safe withdrawal of NAs⁶



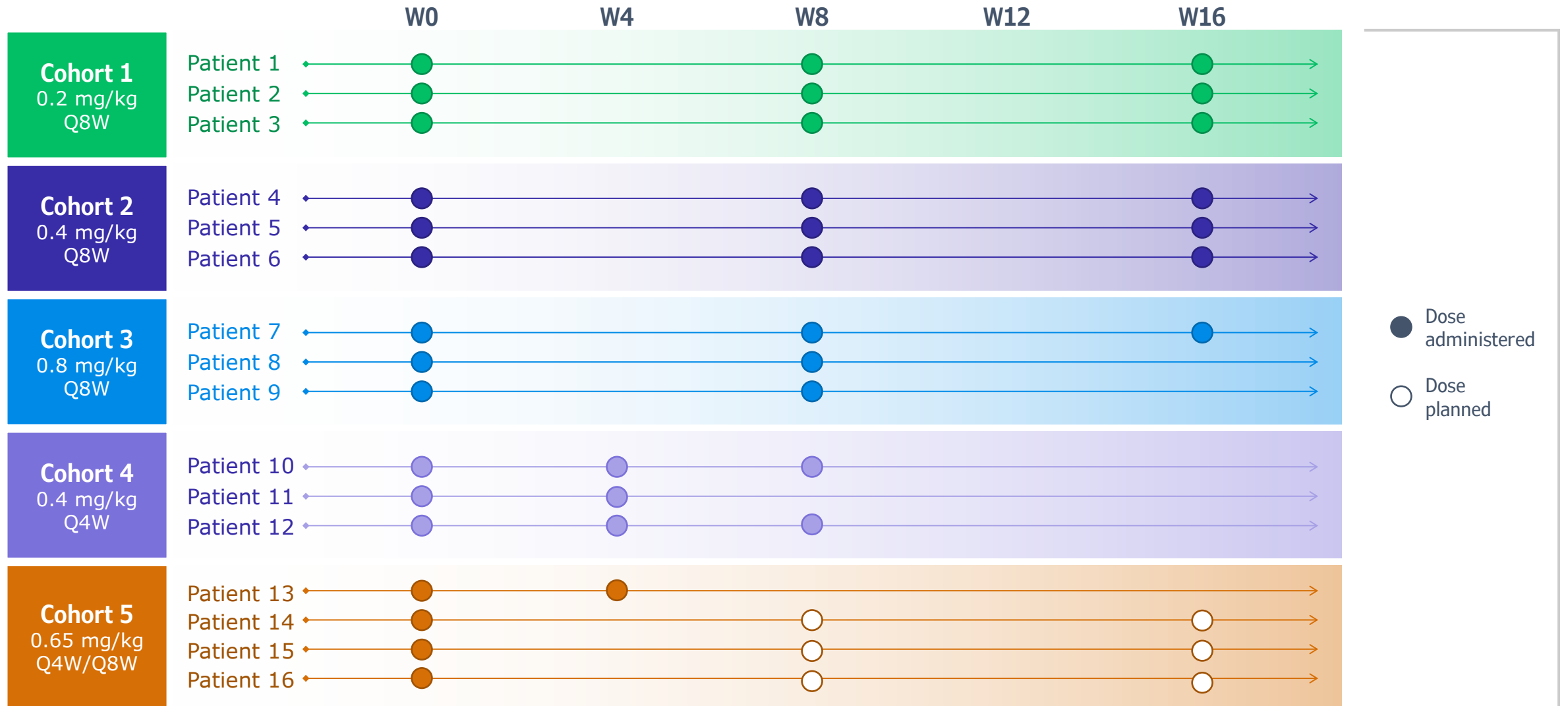
cccDNA, covalently closed circular DNA; NA, nucleos(t)ide analog; pgRNA, pregenomic RNA (also known as HBV RNA).

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.

*Phase 1 ELIMINATE-B Trial
Continues to Rapidly Progress*



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



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

*Efficacy:
PBGENE-HBV Shows Potent Elimination of
cccDNA as Primary Mechanism of Action*



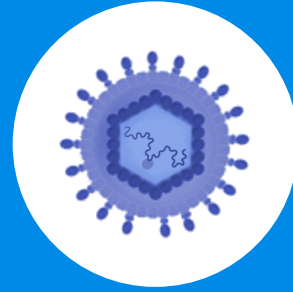
Multiple Datasets Support cccDNA Targeting and Permanence of Effect by PBGENE-HBV



Liver Biopsies

CONFIRMATION OF cccDNA ELIMINATION

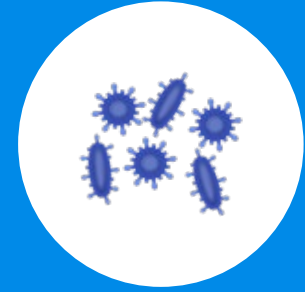
- › **10-fold (1-log) reduction in cccDNA-derived transcripts** and increases in editing with repeat administrations



pgRNA

POTENT cccDNA EFFECT

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



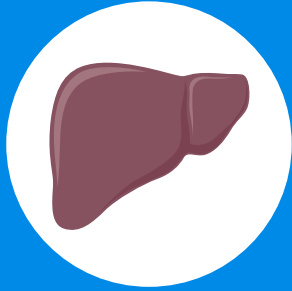
HBsAg

SUBSTANTIAL ANTIVIRAL ACTIVITY IN ALL PATIENTS

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

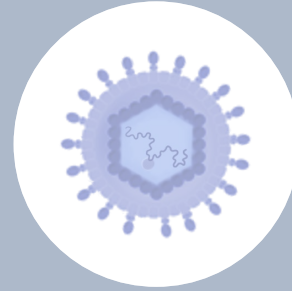


Multiple Datasets Support cccDNA Targeting and Permanence of Effect by PBGENE-HBV



Liver Biopsies

**CONFIRMATION OF
cccDNA ELIMINATION**



pgRNA

**POTENT cccDNA
EFFECT**

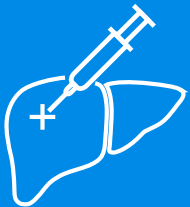
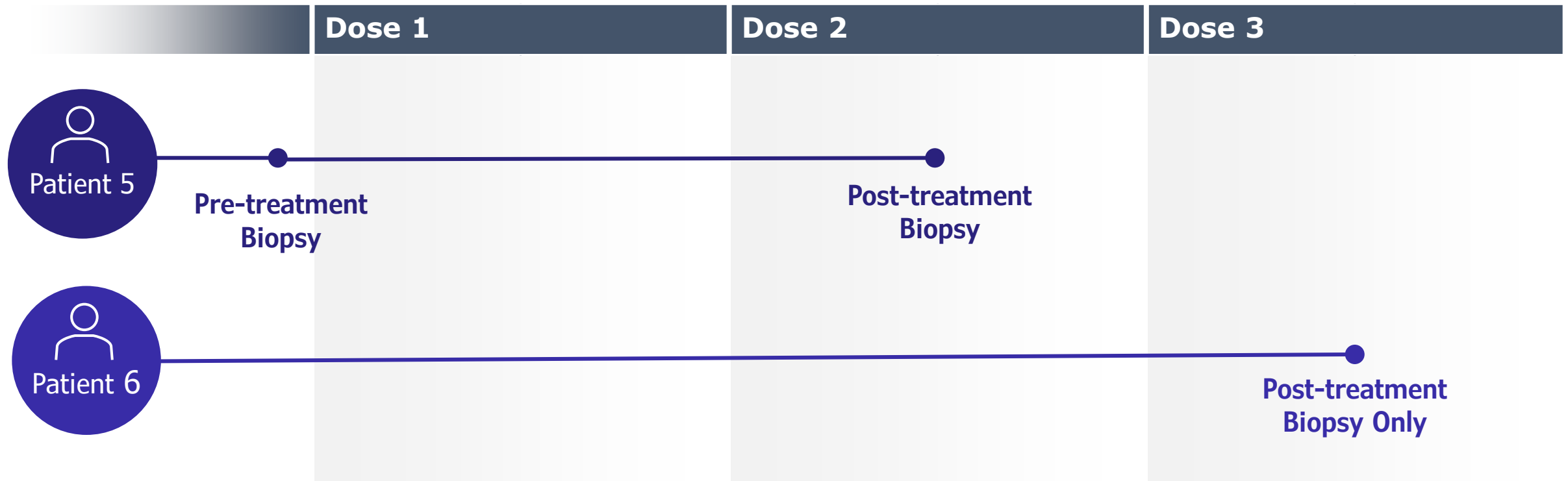


HBsAg

**SUBSTANTIAL ANTIVIRAL
ACTIVITY IN ALL PATIENTS**



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





Liver biopsy analyses

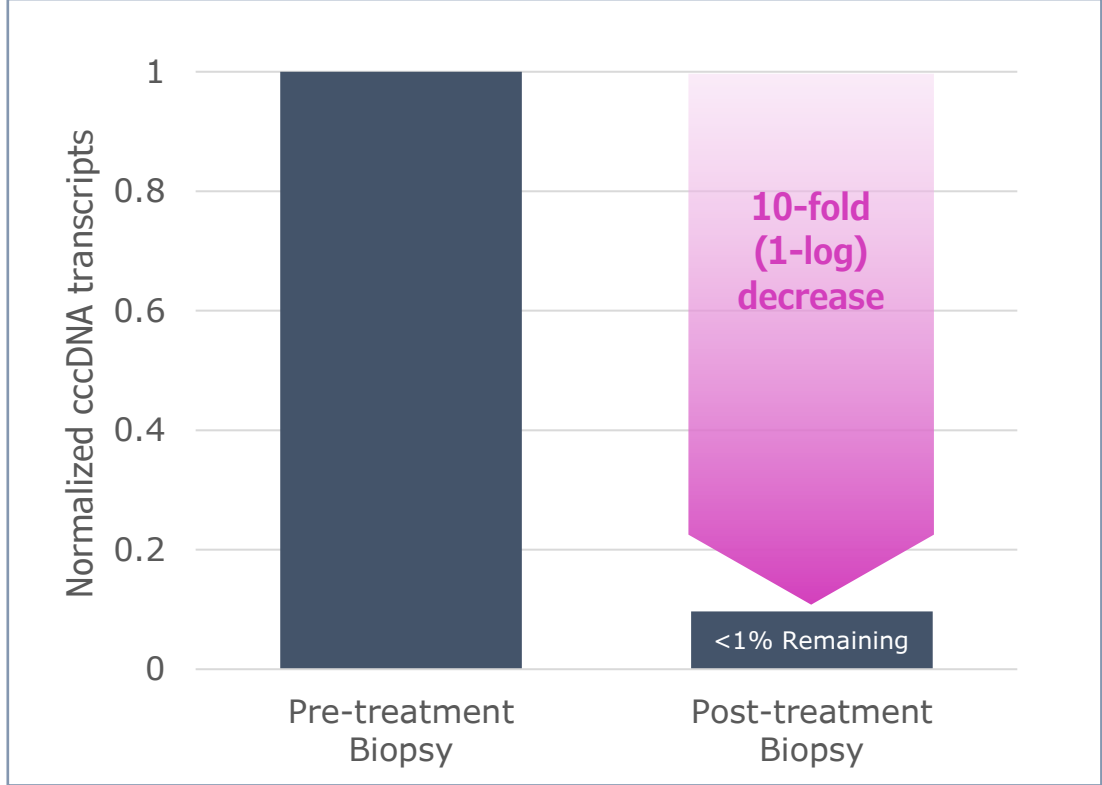
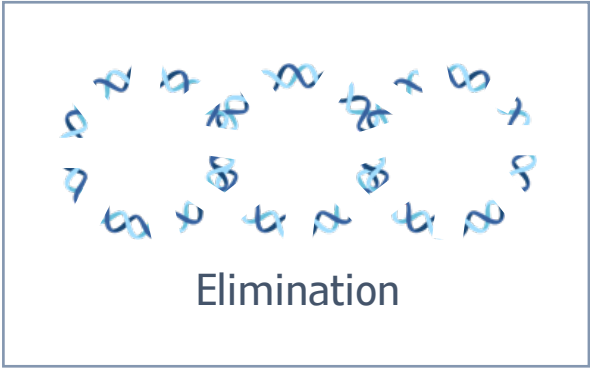
Transcript Sequencing Methodology

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




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

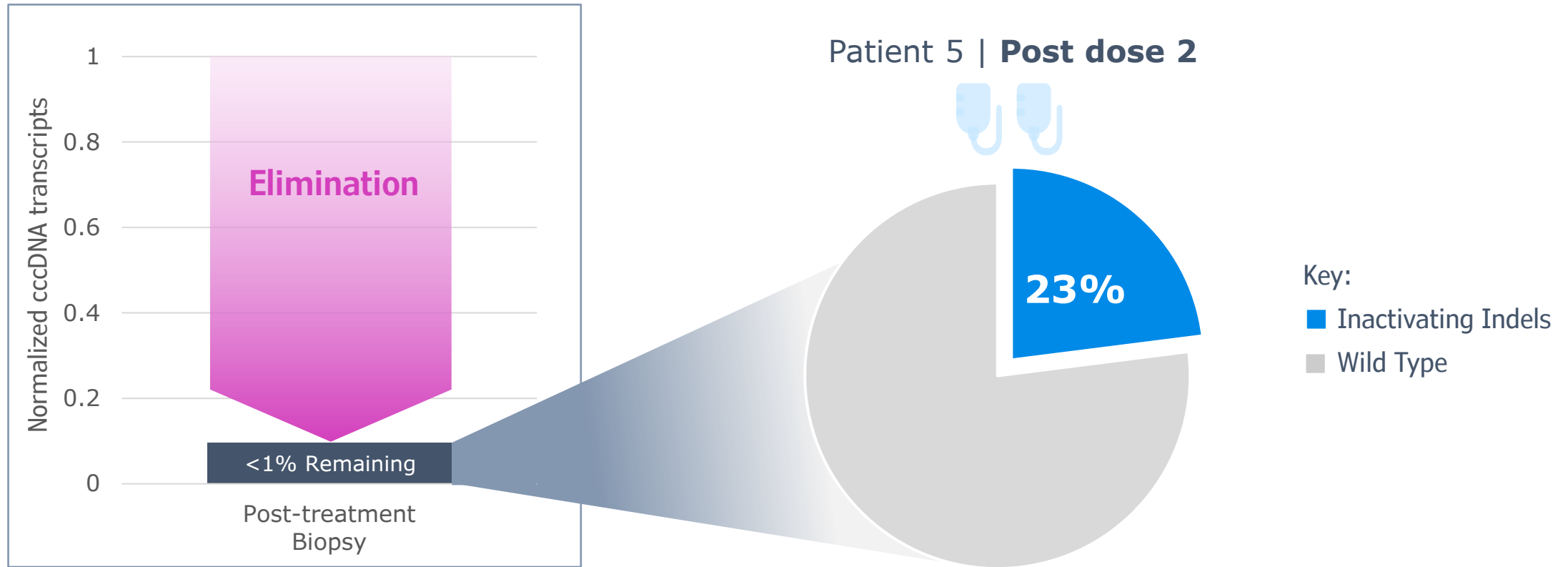

Patient 5
Patient 5
Cohort 2: 0.4 mg/kg
Biopsy after 2 administrations




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

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

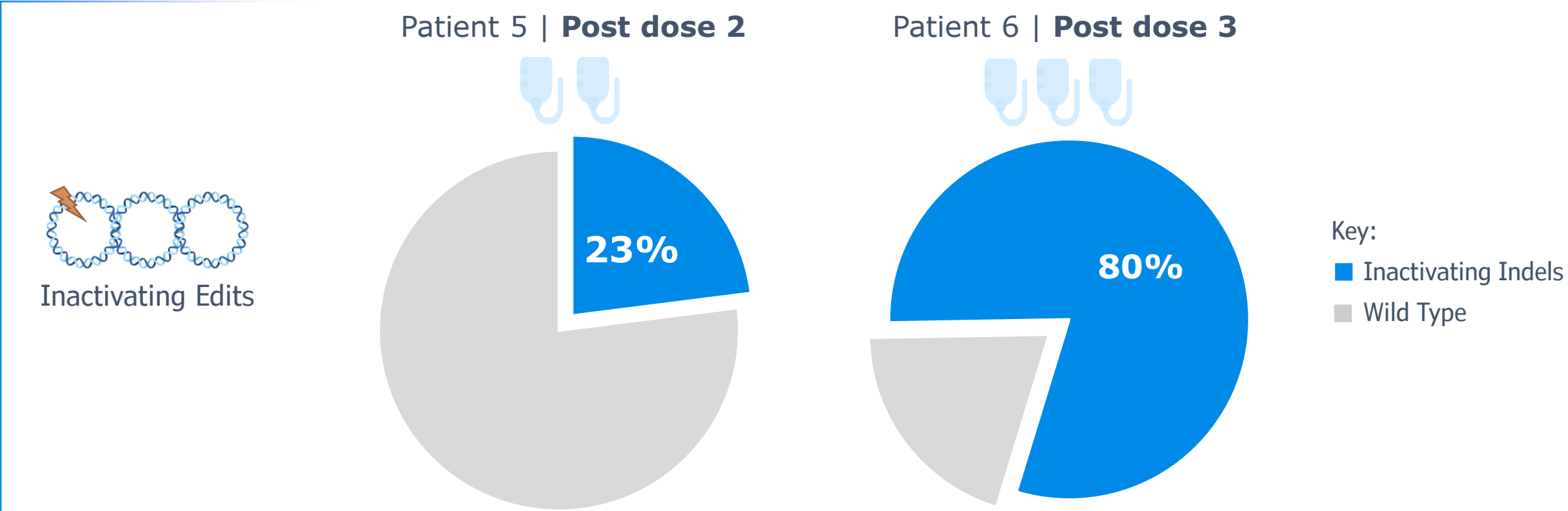
NEW EASL DATA: Of the <1% cccDNA Remaining, PBGENE-HBV Indels Permanently Inactivate Viral Replication by Knocking Out Polymerase Function



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



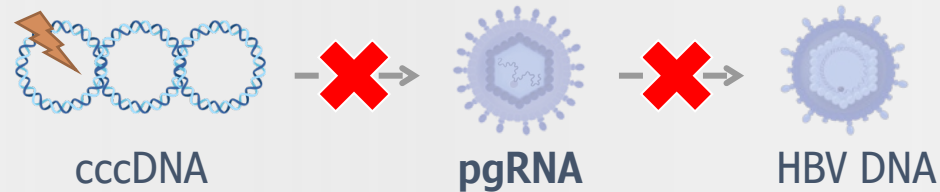
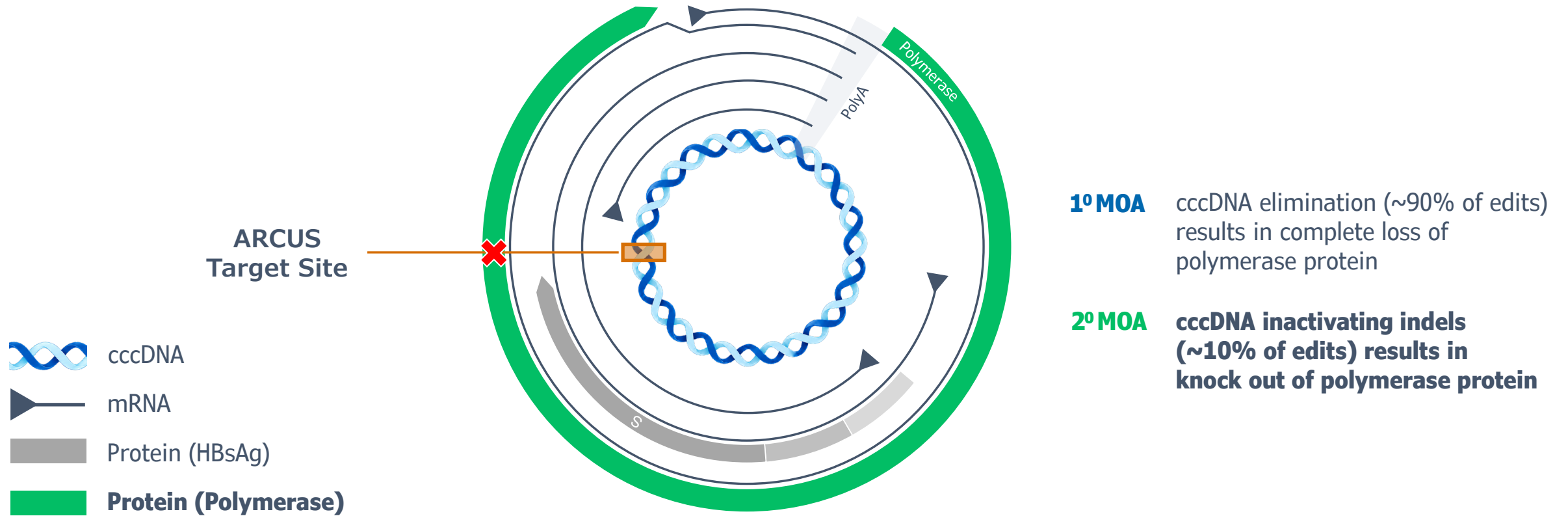
NEW EASL DATA: Additional cccDNA Inactivation Occurs Upon Subsequent Administrations of PBGENE-HBV



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



NEW EASL DATA: Secondary Mechanism – All Indels in cccDNA Knock Out Polymerase Function, Resulting in Complete Viral Inactivation



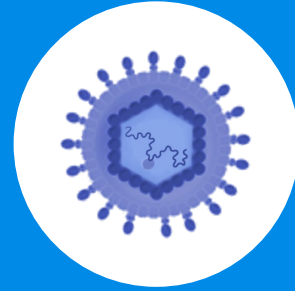
cccDNA, covalently closed circular DNA; HBsAg, hepatitis B surface antigen; MOA, mechanism of action; pgRNA, pregenomic RNA.
 1. Gorsuch CL, et al. *Mol Ther*. 2022;30(suppl 9):2909-2922. 2. Image source adapted from: Tu T, et al. *Viruses*. 2021;13(2):180.
 3. Bartenschlager R, et al. *J Virol*. 1990;64(11):5324-5332. 4. Chen Y, et al. *J Virol*. 1994;68(8):5232-5238.

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



Liver Biopsies

**CONFIRMATION OF
cccDNA ELIMINATION**



pgRNA

**POTENT cccDNA
EFFECT**

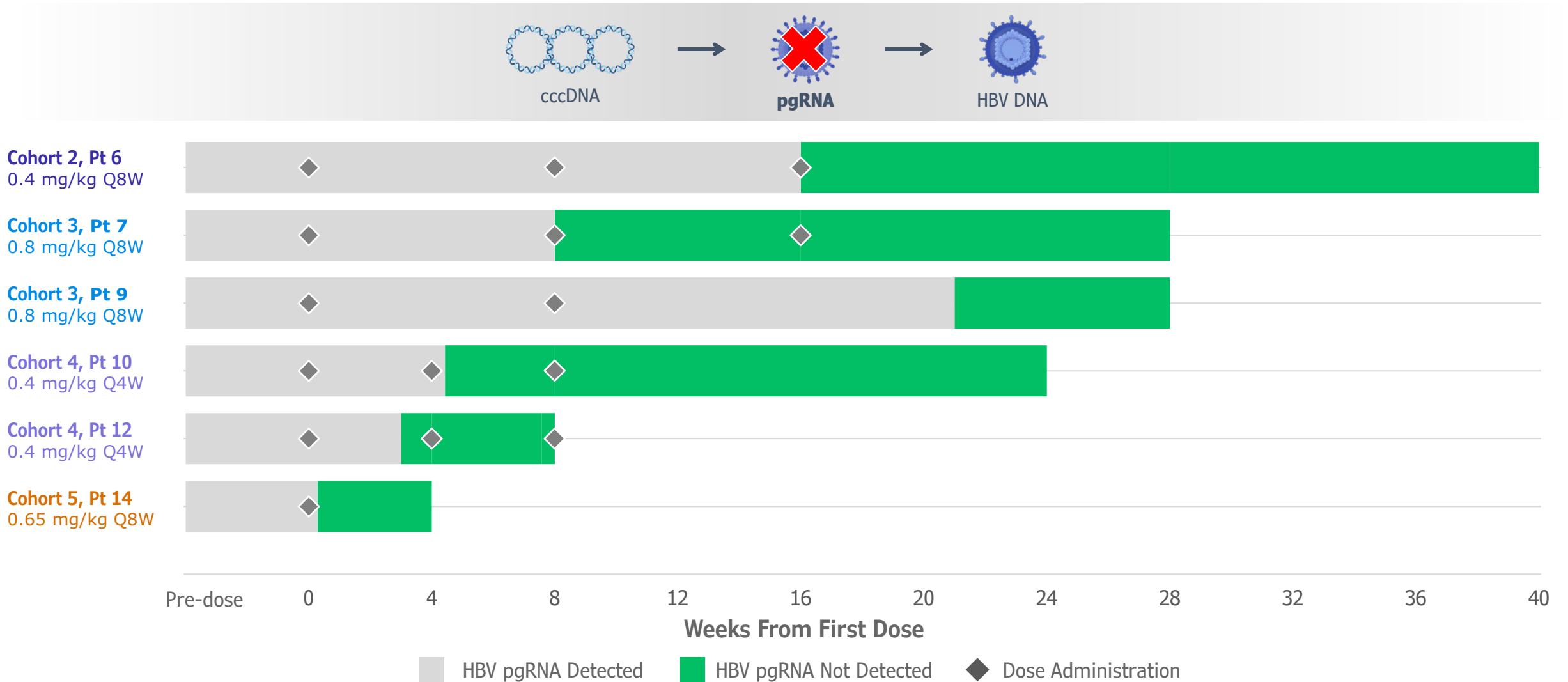


HBsAg

**SUBSTANTIAL ANTIVIRAL
ACTIVITY IN ALL PATIENTS**

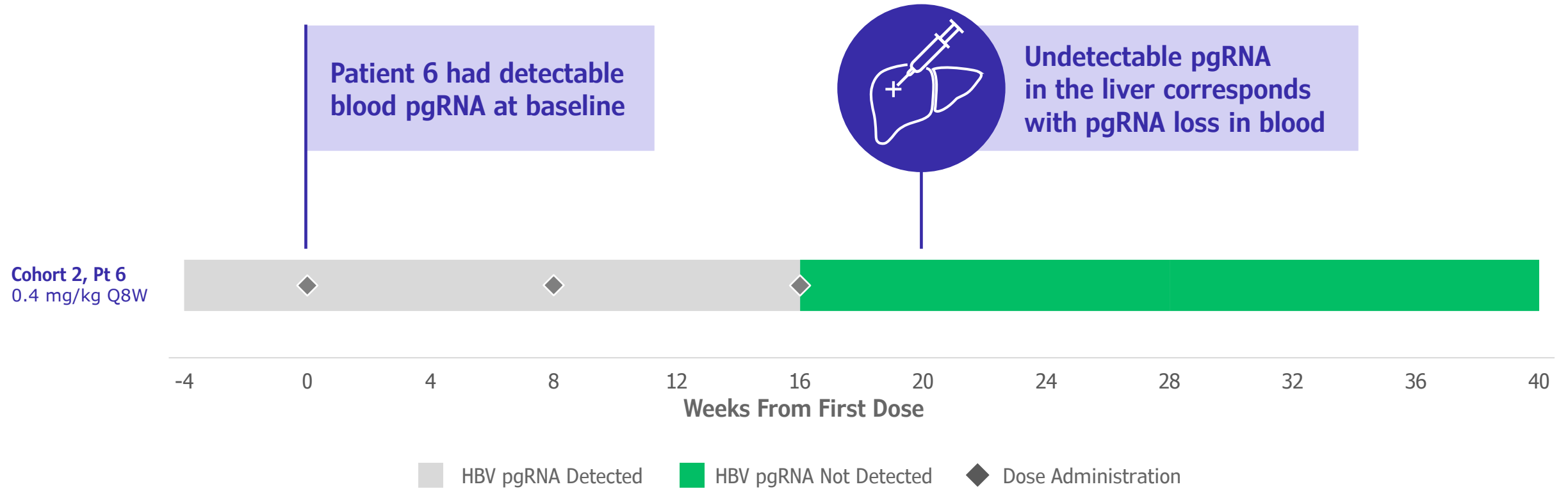


NEW EASL DATA: PBGENE-HBV Drove Durable pgRNA Loss in 100% of Patients Who Had Detectable pgRNA at Baseline*



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

NEW EASL DATA: Patient 6 – Undetectable pgRNA in Both Blood and Liver Biopsy After Third Administration

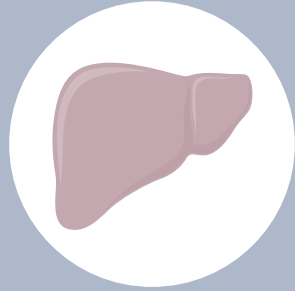


Liver biopsy data further support pgRNA as the appropriate blood biomarker



Patient 6 had detectable blood pgRNA at baseline, which went to undetectable after treatment, corresponding with undetectable pgRNA in liver biopsy collected after third administration. cccDNA, covalently closed circular DNA; pgRNA, pregenomic RNA; Pt, patient; Q8W, every 8 weeks. Data cutoff: May 4, 2026.

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



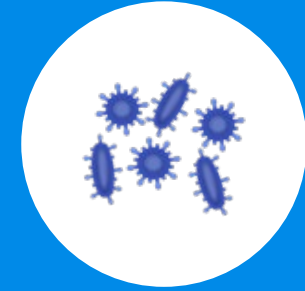
Liver Biopsies

**CONFIRMATION OF
cccDNA ELIMINATION**



pgRNA

**POTENT cccDNA
EFFECT**



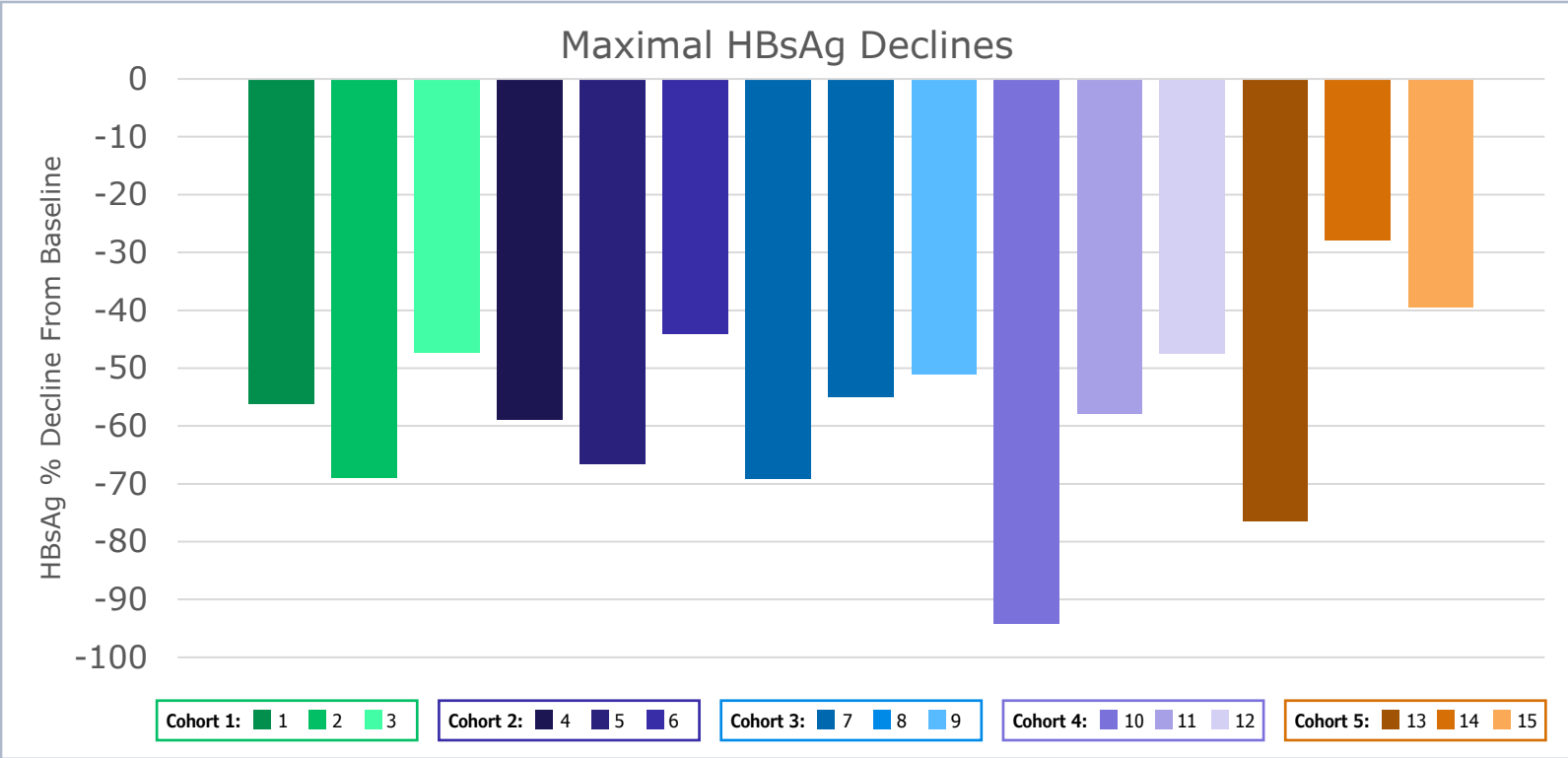
HBsAg

**SUBSTANTIAL ANTIVIRAL
ACTIVITY IN ALL PATIENTS**

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



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

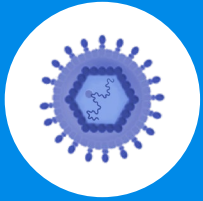


Significant maximal declines in HBsAg supporting PBGENE-HBV activity across all patients (n=15)



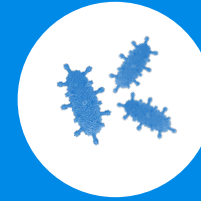
Patient 16 is not evaluable due to incomplete follow-up at time of data cut.
HBsAg, hepatitis B surface antigen.
Data cutoff: May 4, 2026.

NEW EASL DATA: Biomarker Proof of cccDNA Elimination – 100% of Patients With pgRNA Complete Loss and S-Antigen Decline Across All Evaluable Patients



pgRNA (n=6)
Durable biomarker; proof of cccDNA elimination

- › 6 patients detectable at baseline
- › 100% achieved pgRNA loss
- › Duration of response ranging from 1 to 6+ months ongoing (at data cutoff)



HBsAg (n=13)
Supportive biomarker correlated with cccDNA elimination

- › 100% of all patients had a substantial reduction in HBsAg
- › Duration of response ranging from 1.5 months to 1 year ongoing across all patients* (at data cutoff)

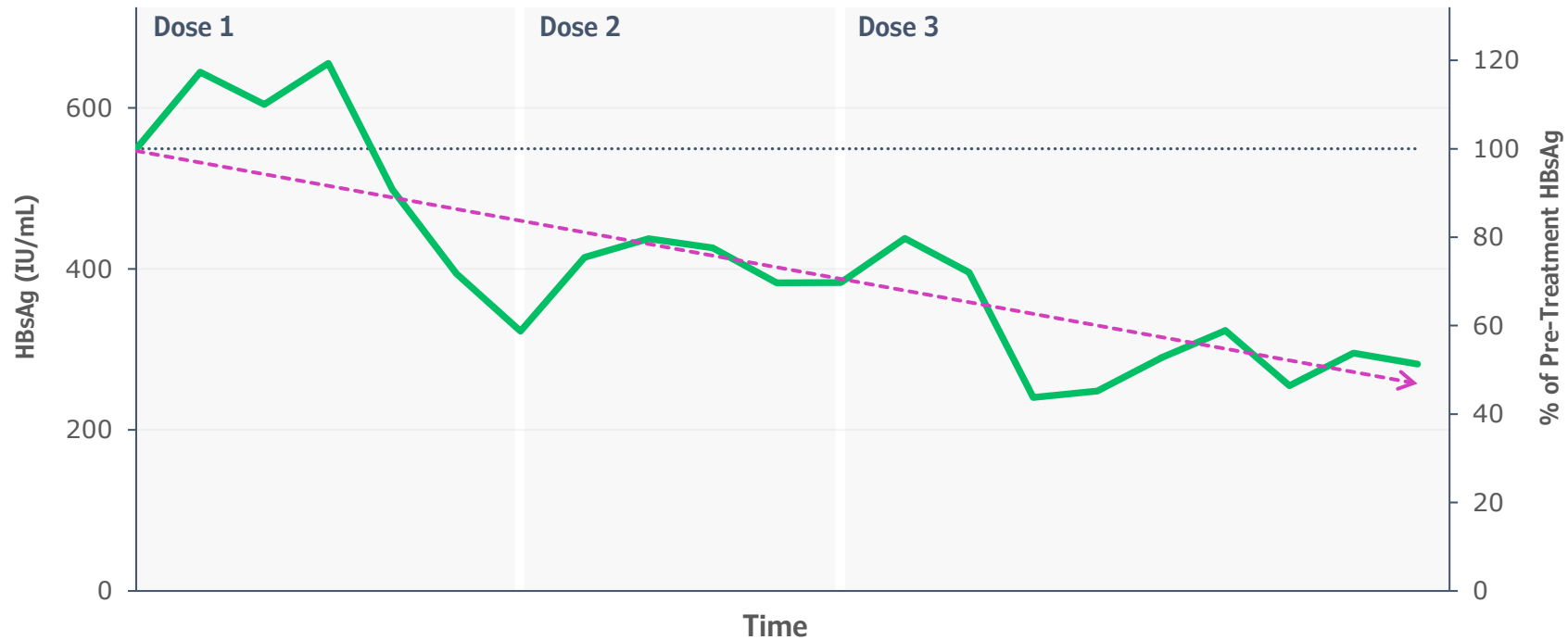
All biomarker trends support cccDNA elimination



*Includes all patients who received repeat LNP doses as of data cutoff (n=13 patients with necessary follow-up at data cutoff).
cccDNA, covalently closed circular DNA; HBsAg, hepatitis B surface antigen; pgRNA, pregenomic RNA.
Data cutoff: May 4, 2026.

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

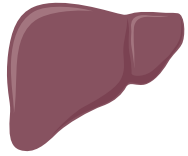
Patient 1 – Sustained HBsAg Reductions



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

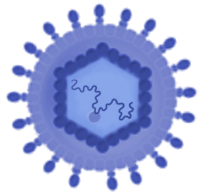


Efficacy Summary: PBGENE-HBV Potentially the Foundation for Viral Cure



Liver Biopsies

- › First evidence of cccDNA elimination by a direct antiviral gene-editing modality
- › 1-log / 10-fold reduction in cccDNA-derived transcripts after only 2 dose administrations
- › Of the <1% of the cccDNA that remains, edits permanently inactivate viral replication knocking out polymerase
- › Cumulative editing is evident after 3 vs 2 dose administrations



pgRNA

- › Durable loss of blood pgRNA in 100% of patients (6/6) with detectable levels pre-treatment
- › Loss of blood pgRNA confirmed undetectable pgRNA in post-treatment liver biopsy
- › Loss of pgRNA across multiple dosing cohorts provides multiple paths forward for ongoing clinical development



HBsAg

- › 100% of patients demonstrate substantial HBsAg declines, indicating broad activity across any baseline HBsAg levels
- › Sustained declines in HBsAg consistent with elimination of cccDNA demonstrated in biopsy data
- › Longest patient experience, stable HBsAg decline >1 year after initial dose supports permanence of gene editing

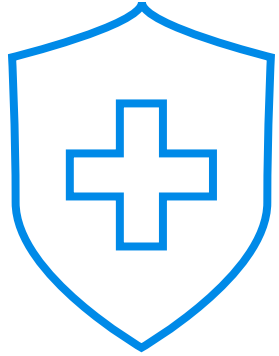
Liver biopsies in combination with key blood biomarkers reflect cccDNA elimination and support increased probability of cure after treatment with PBGENE-HBV



*Safety:
Clinical Safety Update From ELIMINATE-B*



PBGENE-HBV Safety Profile Executive Summary



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

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



A DLT is defined in protocol as any clinically significant, organ-specific, treatment-emergent adverse event (AE) \geq grade 3 that does not decrease to \leq grade 2 within 7 days and is related to study medication.

AE, adverse event; ALT, alanine aminotransferase; AST, aspartate aminotransferase; LNP, lipid nanoparticle; SAE, serious adverse event.

Data cutoff: May 4, 2026.

NEW EASL DATA: PBGENE-HBV Safety Profile Summary – Clear Evidence of Therapeutic Window

Number of patients with treatment-related and possibly treatment-related AEs:		0.2 mg/kg Q8W Cohort 1 n=3	0.4 mg/kg Q8W Cohort 2 n=3	0.4 mg/kg Q4W Cohort 4 n=3	0.65 mg/kg Q4W/Q8W Cohort 5 n=4	0.8 mg/kg Q8W Cohort 3 n=3
Grade 3	Hypotension	-	2	1	1*	3
	ALT/AST lab abnormality	-	-	-	-	-
	Binocular visual dysfunction	-	-	-	-	1*
Grade 4	ALT/AST lab abnormality	-	-	1	1*	2
SAEs	Myocardial ischemia (Grade 2)	-	-	-	-	1*
	Intracerebral hemorrhage (Grade 3)	-	-	-	-	1*

- › No dose-limiting toxicities.
- › No liver-related SAEs.
- › ALT/AST lab abnormalities were transient and asymptomatic, not clinically significant, and not associated with changes in bilirubin. No Hy's Law criteria experienced in any patient.
- › LNP-related elevations were transient and within expected time frame.
- › Grade 3 hypotension did not require vasopressors and resolved with saline infusion.
- › One patient in cohort 3 experienced 2 SAEs after second LNP administration:
 - Grade 2 myocardial ischemia characterized by a mild troponin elevation and EKG finding on day of infusion. Deemed mechanistically linked to acute hypotension and treatment-related. Patient was discharged in 48 hours following a normal CT angiogram. Upon follow-up, echocardiogram showed no heart damage, and normal function and troponin levels were normal.
 - Intracerebral hemorrhage occurred on day 30 post-dosing. Patient is ambulatory, home, and stable. No clear pathophysiologic mechanism attributable to PBGENE-HBV has been identified. The patient had a history of atherosclerosis, and aspirin therapy had been initiated 4 weeks prior. The event is considered possibly related.



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

LNP-inflammatory response

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

Initial prophylaxis and infusion rate

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

Current prophylaxis and infusion rate

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



NEW EASL DATA: Tolerability of PBGENE-HBV Has Improved After Implementing New Safety Mitigations

Across 5 Patients and 7 Administrations With Repeat Administrations Across Cohorts

Number of AEs associated with doses given after implementing new safety mitigations		0.4 mg/kg Q4W Cohort 4 n=2	0.65 mg/kg Q8W* Cohort 5 n=3
Grade 3	Hypotension	-	-
	ALT/AST lab abnormality	-	-
Grade 4	ALT/AST lab abnormality	1	-
SAEs		-	-

- › New prophylaxis and infusion measures have significantly improved tolerability of PBGENE-HBV, with no grade 3 hypotension observed in any doses given
- › One delayed ALT/AST elevation beyond LNP exposure was observed in a patient
- › Associated with a delayed reduction in HBsAg, hypothesized to be indicative of an efficacy-related immune ALT flare

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



*Participants include 3 patients who received Q8W dosing.
 AE, adverse event; ALT, alanine aminotransferase; AST, aspartate aminotransferase; Q4W, every 4 weeks; Q8W, every 8 weeks; SAE, serious adverse event.
 Data cutoff: May 4, 2026.

Summary of PBGENE-HBV Clinical Data



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

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



Next Steps



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



Michael Amoroso

President & Chief Executive Officer
Precision BioSciences, Inc.



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

CONTINUE

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

STOP NUC

- › **Confirm global alignment for NUC withdrawal framework** with clinicians
 - Initial thinking
 - pgRNA loss for ≥ 6 months proxy for HBV DNA
 - Normalized liver enzymes
 - Sustained reductions in HBsAg
- › **Evaluate current patients with pgRNA loss** for NUC withdrawal
 - Expand to baseline pgRNA-negative patients after viral cure proven in patients with pgRNA loss

EXPAND

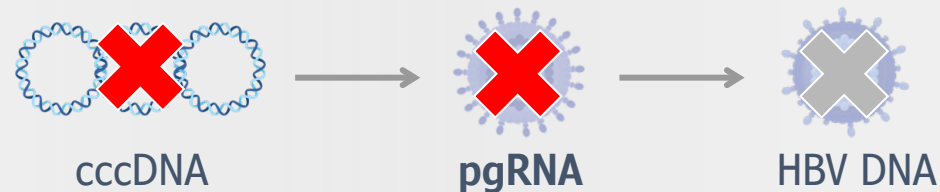
- › **Select optimal dosing schedule** for phase 1 expansion and phase 2
- › **Lifecycle management for PBGENE-HBV**
 - Broaden to an enriched pgRNA+/cccDNA patient population, HBeAg+ patients
 - Continue to assess PBGENE-HBV as potential viral cure via monotherapy and through combination

Plan for Stopping NUCs and Expanding ELIMINATE-B



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

- ✓ Eliminating cccDNA drives undetectable HBV DNA → consistent with FDA approval guidance
- ✓ Destruction of replication-competent virus by PBGENE-HBV directly eliminating and inactivating cccDNA
- ✓ pgRNA is the biomarker to directly measure cccDNA elimination by PBGENE-HBV
- ✓ PBGENE-HBV achieved pgRNA loss in 100% of patients with detectable pgRNA at baseline
- ✓ PBGENE-HBV has a clear therapeutic window with a well-characterized and manageable safety profile across multiple dosing regimens



Multiple Near-Term Data Catalysts Through Wholly Owned Programs

Clinical-Stage Programs Validating ARCUS

Wholly Owned In Vivo Gene-Editing Programs



PBGENE-HBV for Chronic Hepatitis B

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

Clinical data now validate direct targeting and elimination of cccDNA



PBGENE-DMD for Duchenne Muscular Dystrophy

- › PBGENE-DMD is designed to provide durable functional muscle improvement targeting ~60% of patients with DMD
- › First clinical trial site activated at Arkansas Children's Hospital
- › **Next step: execute in clinic through 2026, with initial clinical data expected end of year**





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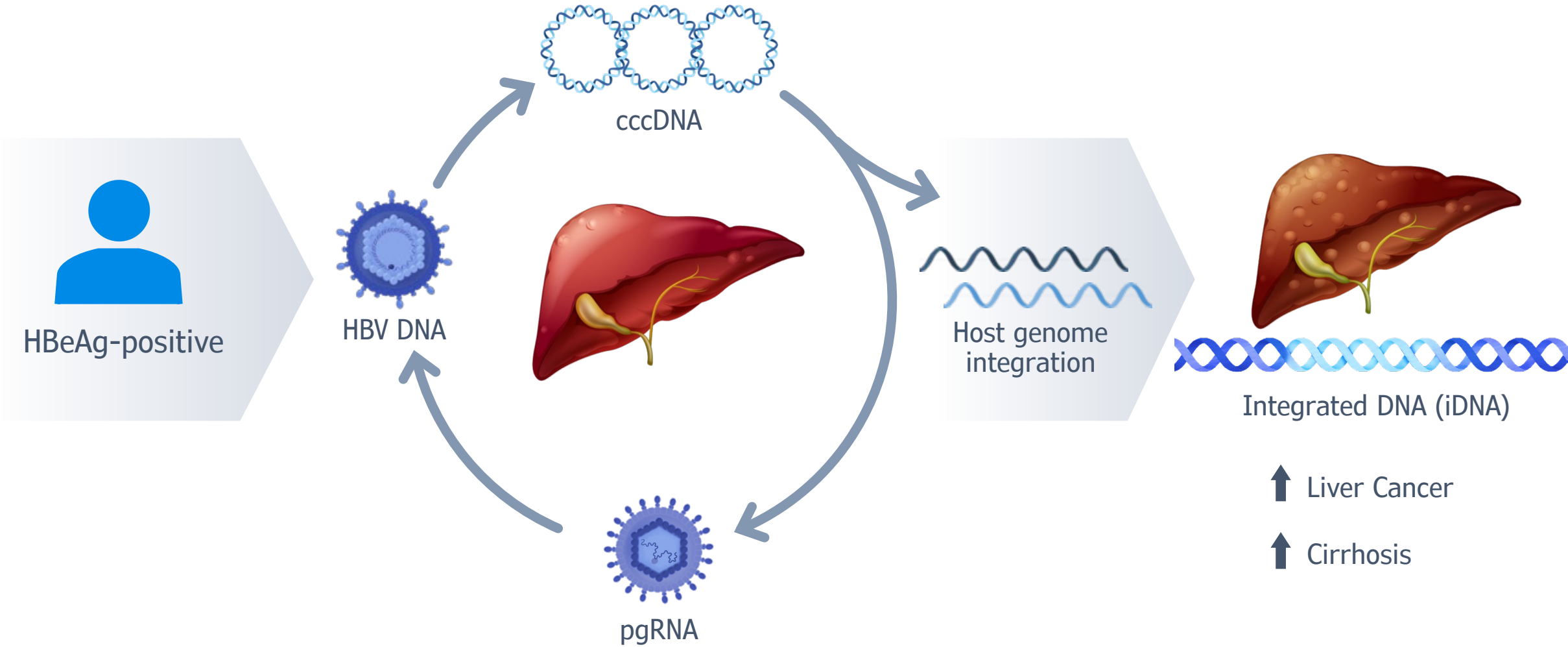
President & Chief Executive Officer
Precision BioSciences, Inc.



Appendix



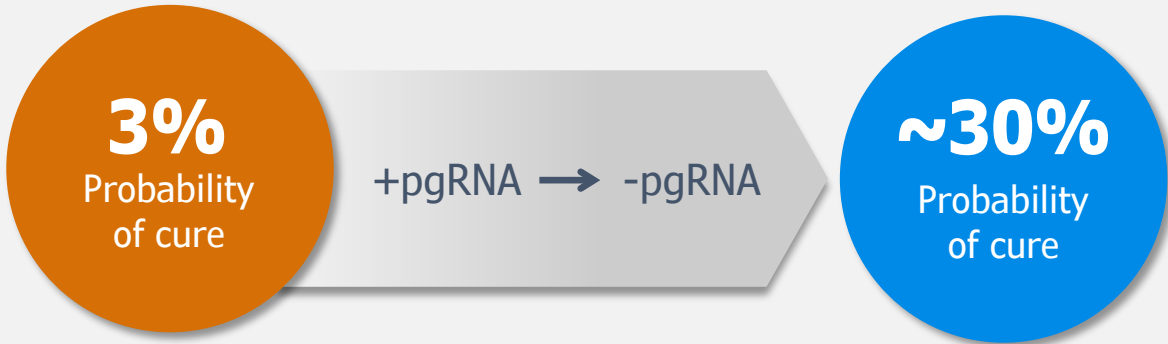
Viral Replication Drives Increased Integrations, Resulting in a Higher Risk of Liver Cirrhosis and Liver Cancer



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

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

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



Adapted from Terrault NA, et al.

