PRECISION
BIOSCIENCES

Corporate Deck

January 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 the clinical development and expected gene editing efficiency, safety, efficacy and benefit of our product candidates (including PBGENE-HBV and PBGENE-3243) and those of our collaboration partners (including iECURE-OTC); the design of PBGENE-HBV to eliminate cccDNA and inactivate integrated HBV DNA with high specificity and driving sterilizing or functional cures with finite treatment; 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, expectations about the commercial potential, market opportunity, operational initiatives, strategies, and further development of our programs and those of our collaboration partners (including iECURE-OTC); expectations about achievement of key milestones and receipt of any milestone, royalty or other payments; anticipated timing of regulatory filings, regulatory acceptances and clinical data, our expected cash runway and available credit; the sufficiency of our cash runway extending into the second half of 2026 and realizing Phase 1 clinical data for multiple in vivo gene editing programs. In some cases, you can identify forward-looking statements by terms such as "aim," "anticipate," "approach," "believe," "contemplate," "could," "designed to", "endeavor", "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 risk that other genome-editing technologies may provide significant advantages over 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; our or our collaborators' 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; the rate and degree of market acceptance of any of our product candidates; 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; our ability to effectively manage the growth of our operations; our ability to attract, retain, and motivate key executives and personnel; market and economic conditions; effects of system failures and security breaches; 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; insurance expenses and exposure to uninsured liabilities; effects of tax rules; risks related to ownership of our common stock; 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 September 30, 2024, 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.

Precision BioSciences is a Clinical Stage In Vivo Gene Editing Company

Multiple Programs in or Nearing Clinical Data

		Expected First Clinical Data Timing
Wholly owned	**PBGENE-HBV** ***In Vivo* Gene Editing Program for Chronic Hepatitis B** 〉 Multiple Clinical Trial Application (CTA) approvals received including in Moldova, Hong Kong & New Zealand 〉 First-in-human trial ELIMINATE-B initiated 〉 U.S. clinical site will be part of phase 1 trial eliminate b	**2025**
	PBGENE-3243 ***In Vivo* Gene Editing Program for m3243 mitochondrial disease** 〉 IND and/or CTA filing in 2025 〉 Currently in late-stage preclinical development	**2026**
Partnered Program Employing ARCUS Nuclease	**iECURE-OTC[1]** 〉 Global phase 1 trial on-going in U.S., U.K., Australia and Spain 〉 Complete clinical response in first patient dosed; OTC-HOPE Phase 1/2 trial expected to finish enrollment in 2025 with full data in 1H 2026 〉 Initial clinical validation for ARCUS *in vivo* gene editing	**2025**

~$109 Million to Fund Company Operations into 2H 2026
Three Phase 1 Clinical Data Read-Outs Within Cash Runway[2]

1. Also known as ECUR-506; approved for clinical trials by the U.S. Food and Drug Administration (FDA), U.K. Medicines & Healthcare products Regulatory Agency (MHRA), Australian Therapeutic Goods Administration (TGA) and Spanish Agency of Medicines and Medical Devices (AEMPS) in Spain. iECURE responsible for all development costs for ECUR-506
2. Cash, cash equivalents, and restricted cash as of 12/31/24 (8K – 1/8/2025); $108.5M reported and does not include $2.5M from TG Therapeutics and additional cash inflows in 2025

Ornithine Transcarbamylase (OTC) Deficiency Program: First ARCUS in vivo Gene Editing Program Validated In Clinical Study

Initial Clinical Validation for ARCUS *in vivo* Gene Insertion

First Infant Achieves Complete Clinical Response in Our Partner iECURE's Phase 1/2 OTC-Hope trial

- Showcases that a complete clinical response can be achieved through ARCUS in vivo gene editing for children born with devastating OTC disease
- ECUR-506 was generally well tolerated with no significant clinical safety concerns
- iECURE has received IND and CTA approvals in the U.S., Australia, U.K., and Spain for the initiation of a first-in-human Phase 1/2 trial OTC-HOPE evaluating ECUR-506, which employs an ARCUS nuclease, for the treatment of OTC deficiency in pediatric (or neonatal) patients[3]
- OTC-Hope trial enrollment expected to be completed in 2025 with data readout for all patients expected in 1H 2026[3]

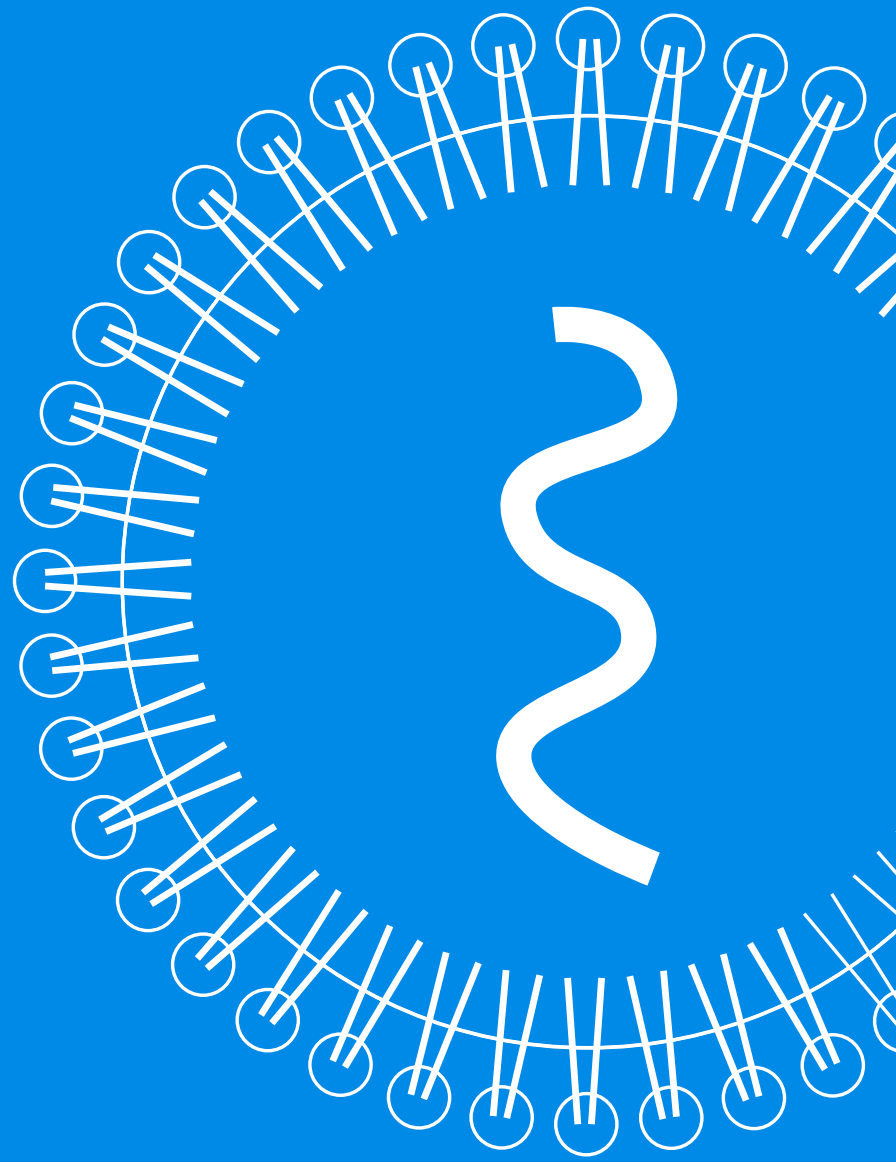
~10,000

People WW with OTCD[1,2]

- Occurs in over 1,000 births per year globally
- Disease prevalence is between 1 in 60,000 - 72,000
- **Neonatal onset has been associated with mortality rates as high as 74%**

[1] Complete absence of OTC protein results in severe neonatal disease, while decreased OTC levels results in late-onset disease.
[2] Onset may occur at any age though is more common in infancy. OTC: Ornithine Transcarbamylase. Source: UpToDate; Orphanet; Hasegawa et. Al. J Pediatr Surg. 1995. Ah et. Al. GeneReviews. 2017. NORD; Lamb et. Al. BJM. 2016. Brassier et. Al. Orphanet Journal of Rare Disease 2015.; Unsinn et. Al. Orphanet Journal of Rare Diseases. 2016; Summar et al. NIH. 2008; Buerger et. Al. J. Inherit. Metab. Dis. 2013; ClearView Analysis.
3. iECURE press release – January 2025

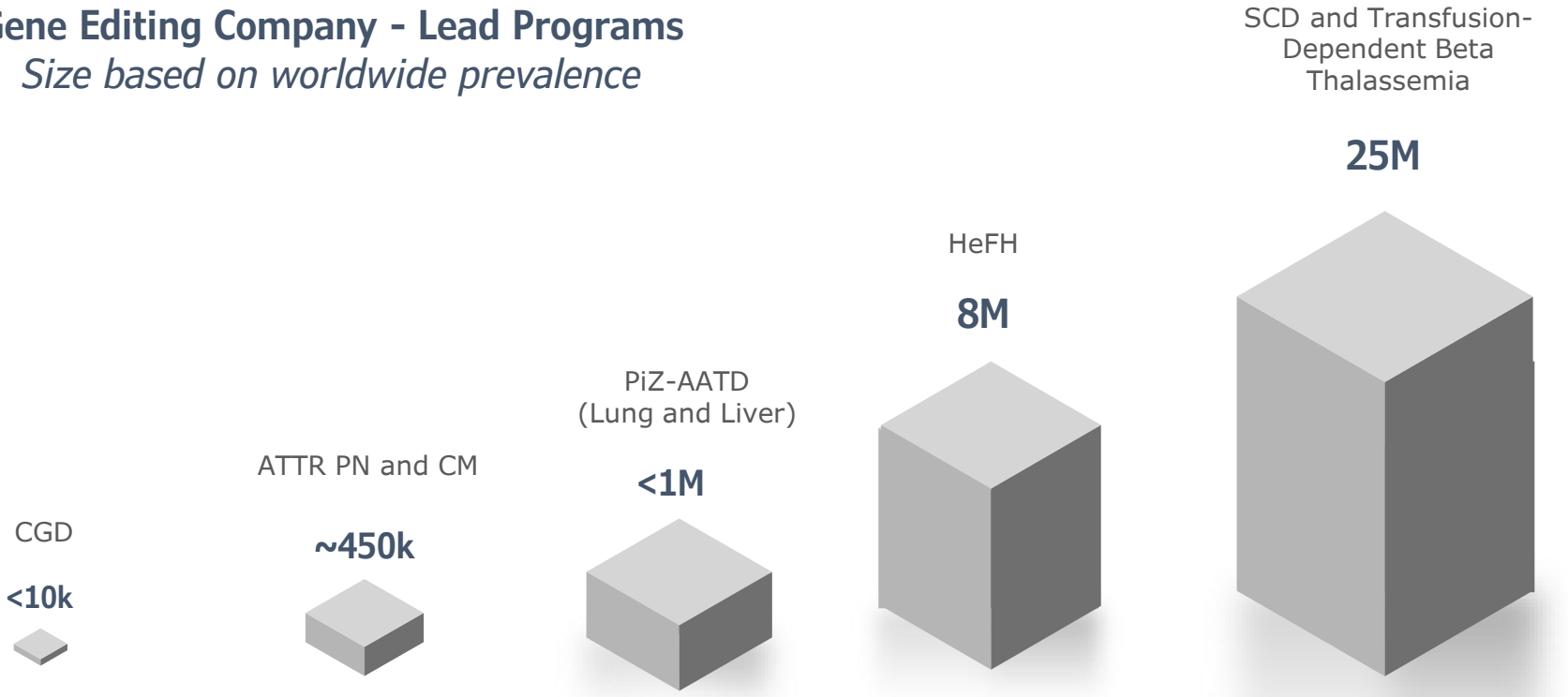
PBGENE-HBV is
First Wholly Owned
In Vivo Gene Editing
Application of ARCUS

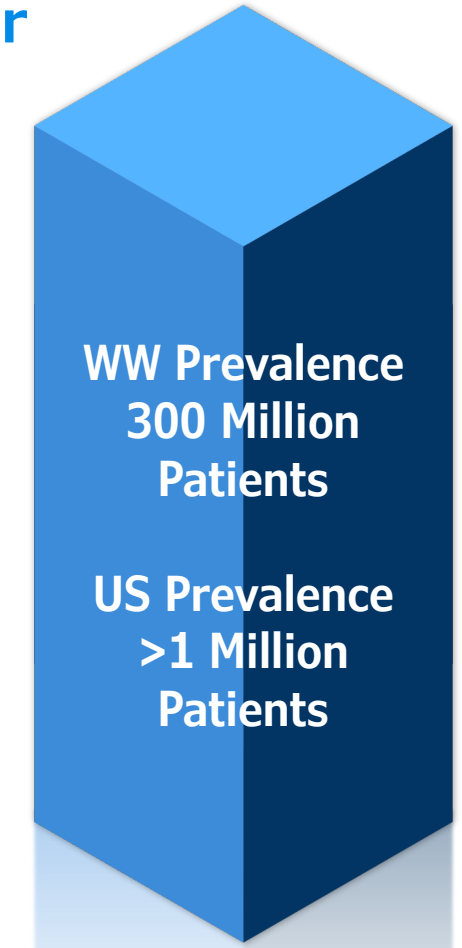


PBGENE-HBV Positioned to Impact More Patients than Other Gene Editing Programs

Gene Editing Company - Lead Programs

Size based on worldwide prevalence





Precision BioSciences
PBGENE-HBV

Lead Program for
Chronic Hepatitis B (cHBV)

Beta-Thal, beta thalassemia; CGD, chronic granulomatous disease; CM, cardiomyopathy; HeFH, heterozygous familial hypercholesterolemia; PN, polyneuropathy; SCD, sickle cell disease; TD, transfusion-dependent; US, United States; WW, worldwide. k, thousand; M, million.

1. Winkelstein JA, et al. Chronic granulomatous disease. Medicine (Baltimore). 2000;79(3):155. **2.** Lovely M, et al. Journal of Patient-Reported Outcomes. 2021. **3.** Lin H, et al. Value Health. 2019. **4.** Morris A, et al. Clin Ther. 2022. **5.** Detecting amyloidosis. AstraZeneca website. www.astrazeneca.com/media-centre/articles/2023/detecting-amyloidosis-understanding-the-different-types-of-attr.html. Published 2023. Accessed October 2, 2024. **6.** Leme A, et al. J Clin Invest. 2021. **7.** Familial hypercholesterolemia in adults: Overview. UpToDate website. www.uptodate.com/contents/familial-hypercholesterolemia-in-adults-overview#H1321616434. Accessed October 2, 2024. **8.** Original Article: Lancet Haematol. 2023;10(8). **9.** Correction: Lancet Haematol. 2023;10(8). **10.** CRISPR Tx. EU application https://www.ema.europa.eu/en/documents/rmp/casgevy-epar-risk-management-plan_en.pdf

Chronic Hepatitis B Multi-Billion Dollar Market Opportunity:

Large patient population currently on non-curative treatment options

> 300 Million
cHBV infections globally

> 1,000,000
cHBV infections in the US

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

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

~250,000
patients in US

~180,000
patients in Europe

~4,000,000
patients in China

~260,000
patients in Japan

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

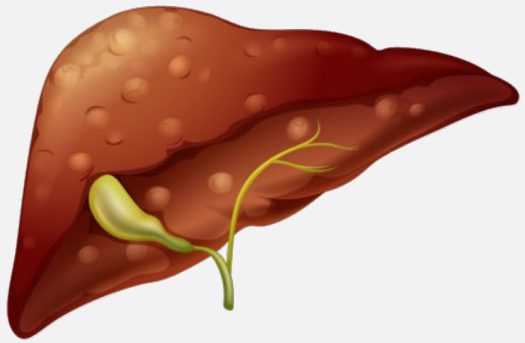
M, million.
Additional 142,000 potentially drug treated in Africa.[3]

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

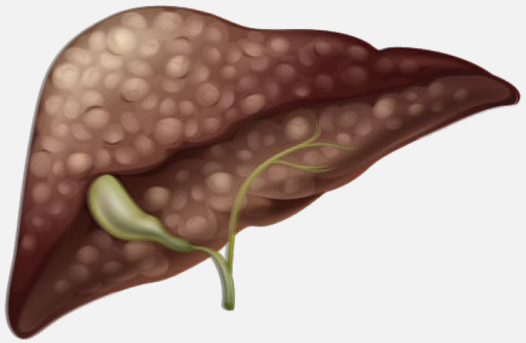
Risk of Liver Cancer, Complications, and Mortality Remain on Current SoC

cHBV Patients Have Up to a 30% Risk of Liver Cancer Over 10 years on Nucleos(t)ide Analogs

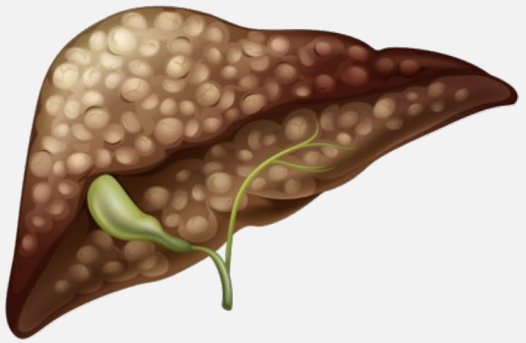
Up to **40% of patients** with chronic HBV infections may develop life-threatening complications including cirrhosis and/or HCC[1]



Cirrhosis



Liver Failure



Hepatocellular Carcinoma (HCC)

cHBV Results in

> 1 Million Global Deaths

Every Year[2,3]

Even when virally suppressed on nucleos(t)ide analogs, risk for HCC remains with a 10-year cumulative incidence up to 30%[4]

1. Centers for Disease Control and Prevention. Hepatitis B. CDC Yellow Book 2024: Health Information for International Travel. Accessed October 18, 2024. https://wwwnc.cdc.gov/travel/yellowbook/2024/infections-diseases/hepatitis-b. **2.** World Health Organization. Hepatitis B. World Health Organization. Published June 27, 2022. Accessed October 21, 2024. https://www.who.int/news-room/fact-sheets/detail/hepatitis-b. **3.** Hepatitis B Foundation. Hepatitis B Fast Facts. Doylestown, PA: Hepatitis B Foundation; 2007. Available from: https://www.hepb.org. **4.** Abd El Aziz MA, et al. Nucleos(t)ide analogues and Hepatitis B virus-related hepatocellular carcinoma: A literature review. Antivir Chem Chemother. 2020;28:2040206620921331.

Phase 1 Clinical Momentum for PBGENE-HBV Program

Potentially Curative Finite Treatment for cHBV

First-in-Human Trial Initiated

Precision's Hepatitis Scientific Advisory Board

Operational execution guided by world-class Scientific Advisory Board and leading clinical investigators

Mark Sulkowski, M.D.

Jordan Feld, M.D., MPH

Ray Schinazi, Ph.D., DSc

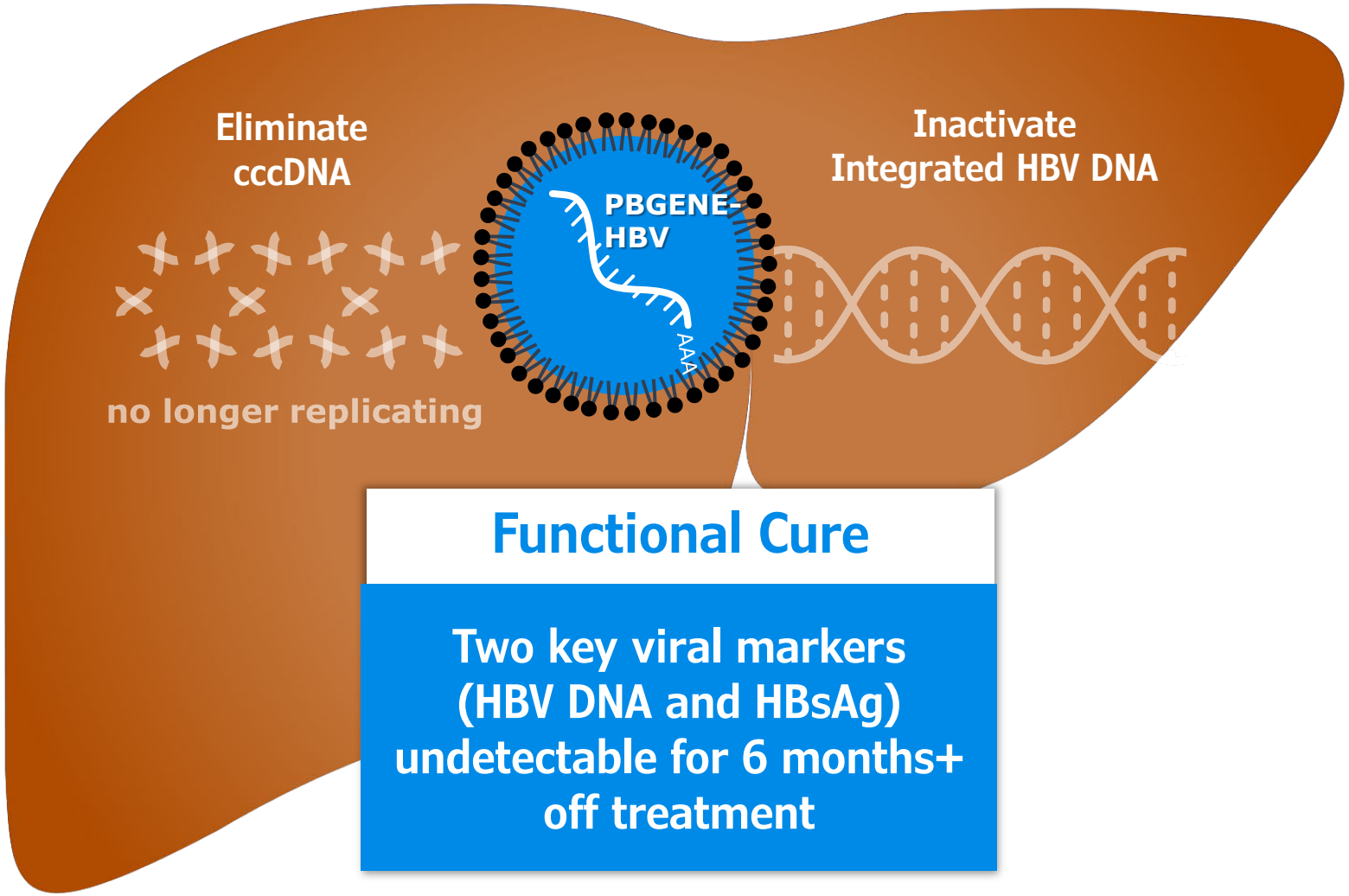
- CTA approved in Moldova, Hong Kong, and New Zealand
- Additional CTA under regulatory review
- U.S. clinical site expected to be part of Phase 1 study
- Global Phase 1 multi-site clinical trial initiated and moving to dose patients in up to 5 countries
- Broad patient inclusion across all global genotypes

Consistent with the Hepatitis C treatment paradigm:
The goal is to drive high cure rates for patients through a finite treatment course

Advancement to Clinical Trials Supported by Strong Preclinical Rationale

PBGENE-HBV Dual Mechanism:

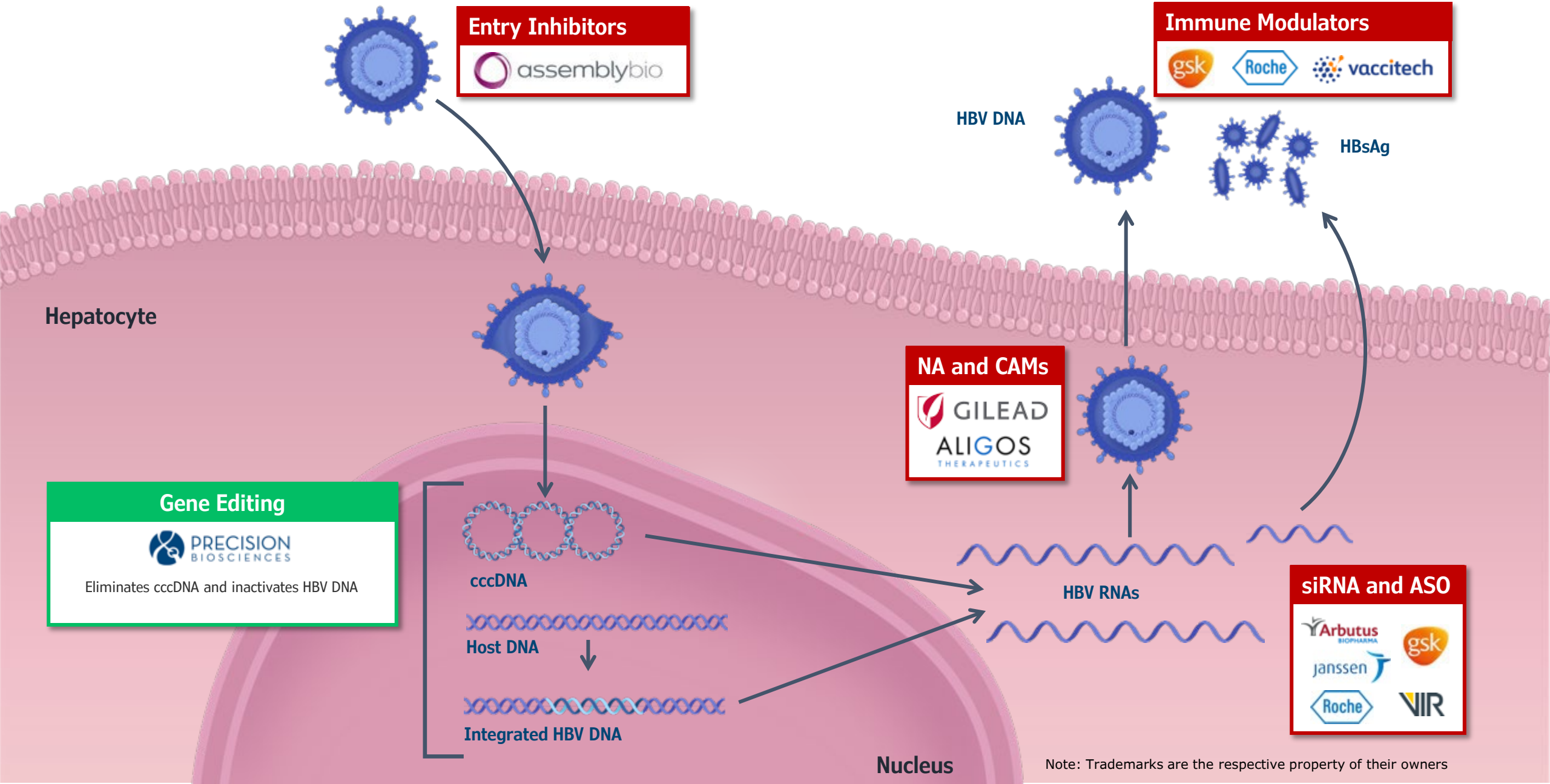
Designed to Drive Potential Cures By Targeting Root Cause of Disease & Eliminate



Functional Cure

Two key viral markers (HBV DNA and HBsAg) undetectable for 6 months+ off treatment

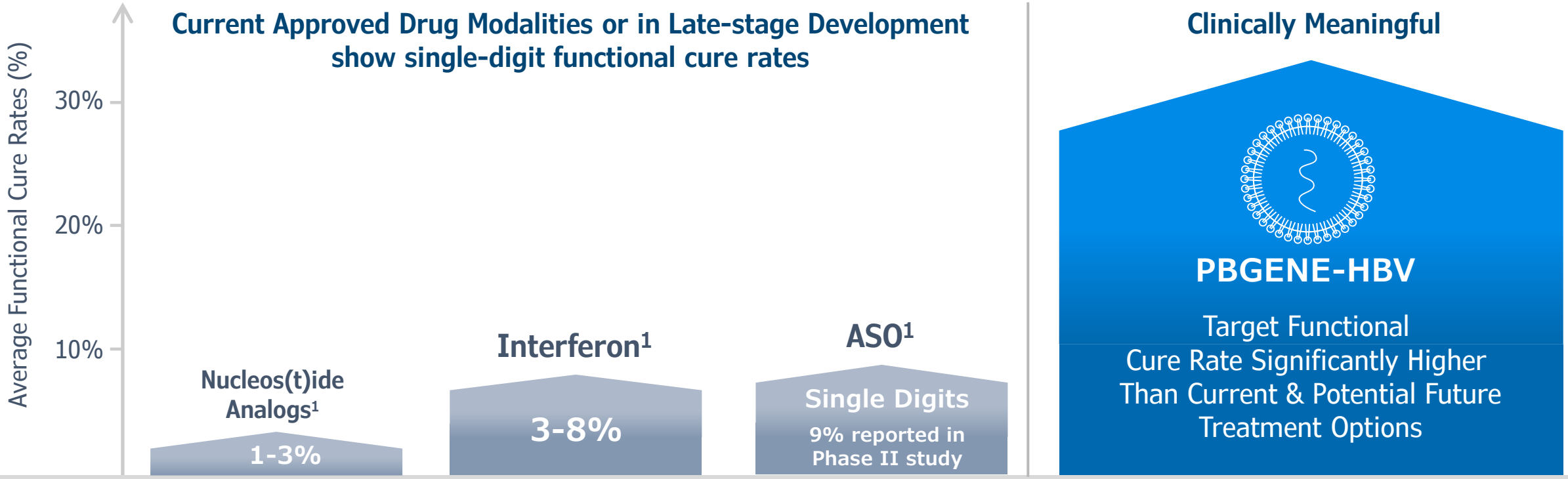
Only ARCUS is designed to eliminate cccDNA and inactivate HBV DNA



Note: Trademarks are the respective property of their owners

Very Few Patients Get To Functional Cure Today:

PBGENE-HBV has opportunity to be clinically meaningful by increasing functional cures



ASO, antisense oligonucleotide.

1. Gopalakrishna H, Ghany MG. Perspective on Emerging Therapies to Achieve Functional Cure of Chronic Hepatitis B. Curr Hepatol Rep. 2024;23(2):241-252.;
9% functional cure rate from cohort treated on Nucleo(s)tide analogs

Eliminate cccDNA to Cure Hepatitis B

"Realization of a functional or complete cure for chronic HBV infections requires innovative therapeutic approaches aimed at disabling and **eliminating the persistent episomal cccDNA**."

–Bloom et al 2018

"At present, the **cccDNA cannot be completely eliminated by standard treatments**. There is an urgent need to develop drugs or therapies that can **reduce HBV cccDNA levels** in infected cells."

–Jin et al 2023

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

–Ligat et al 2020

Bloom et al. *Genes*. 2018; 9(4), 207. DOI: 10.3390/genes9040207
Jin et al. *Drug Discov Today*. 2023; 28(7):103617. DOI: 10.1016/j.drudis.2023.103617
Ligat et al. *Curr Hepatol Rep*. 2020; 19(3):235-244. DOI: 10.1007/s11901-020-00534-w

ARCUS is The Only Clinical Stage Gene Editor Designed to Eliminate cccDNA

ARCUS Gene Editing

Designed to **ELIMINATE** cccDNA and **INACTIVATE** integrated HBV DNA

Single-component

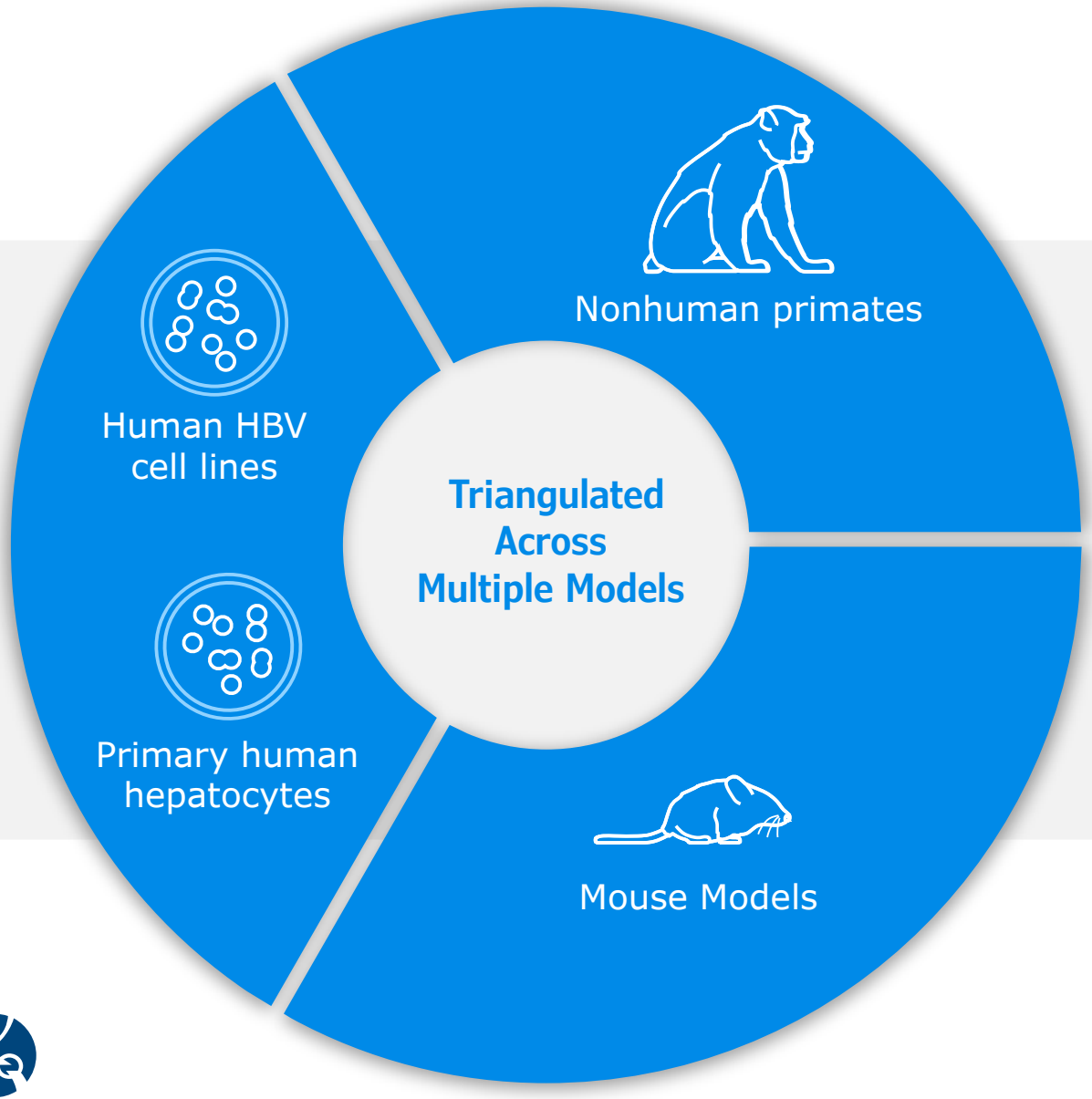
ARCUS protein interacts with cccDNA directly, not through use of a guide RNA

Small size

Enables delivery efficiency and accessibility to cccDNA

Demonstrating Efficacy & Safety: Preclinical Evidence for PBGENE-HBV

Supports Advancement of PBGENE-HBV to First-In-Human Clinical Studies



Robust Preclinical Evidence, Including in Gold Standard NHP Models

Supports PBGENE-HBV to Safely Eliminate Viral Source of Replication and Integrated Disease

Safety: PBGENE-HBV Demonstrates Robust Safety Package Supporting Advancement Towards Clinical Trials

- Robust and thorough specificity pipeline demonstrated **high degree of specificity for PBGENE-HBV** with no increased risks of translocations or integrations in HBV-infected PHH
- PBGENE-HBV **does not distribute to germ cells**, as evidenced by NHP studies
- PBGENE-HBV was **well tolerated** in NHPs **over multiple administrations** with rapid clearance after each dose administration
- **High-quality mRNA** and an **optimized LNP formulation** contribute to a robust safety profile of PBGENE-HBV
- **Favorable safety profile** compared to other clinical stage gene editing LNP programs[1,2,3]

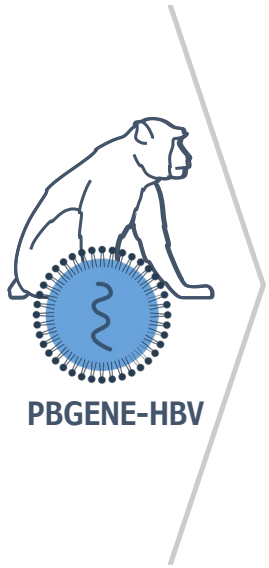
Lee et al. Circulation (2023) | CRISPR, AHA 2023 (CTX310) | CRISPR, AHA 2023 (CTX320)

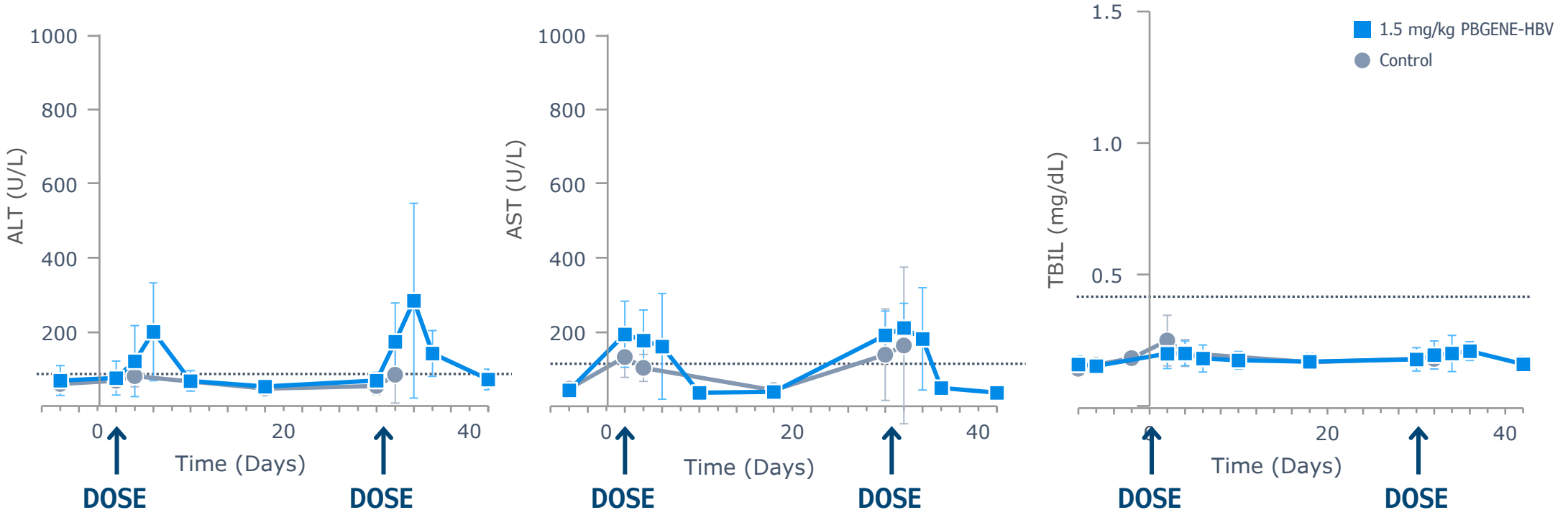
Data sources with hyperlinks are provided for convenience.

PHH: primary human hepatocytes; NHP: nonhuman primates; ULN: upper limit of normal; LNP: lipid nanoparticle

Safety: PBGENE-HBV Showed Minor and Transient Elevations in Liver Transaminases

NHP data supports safety of multi-dosing to achieve functional cure



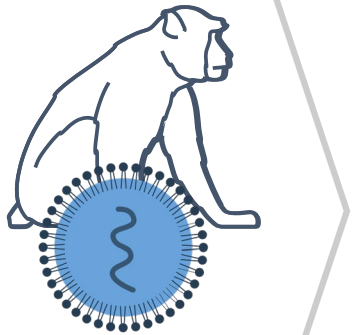


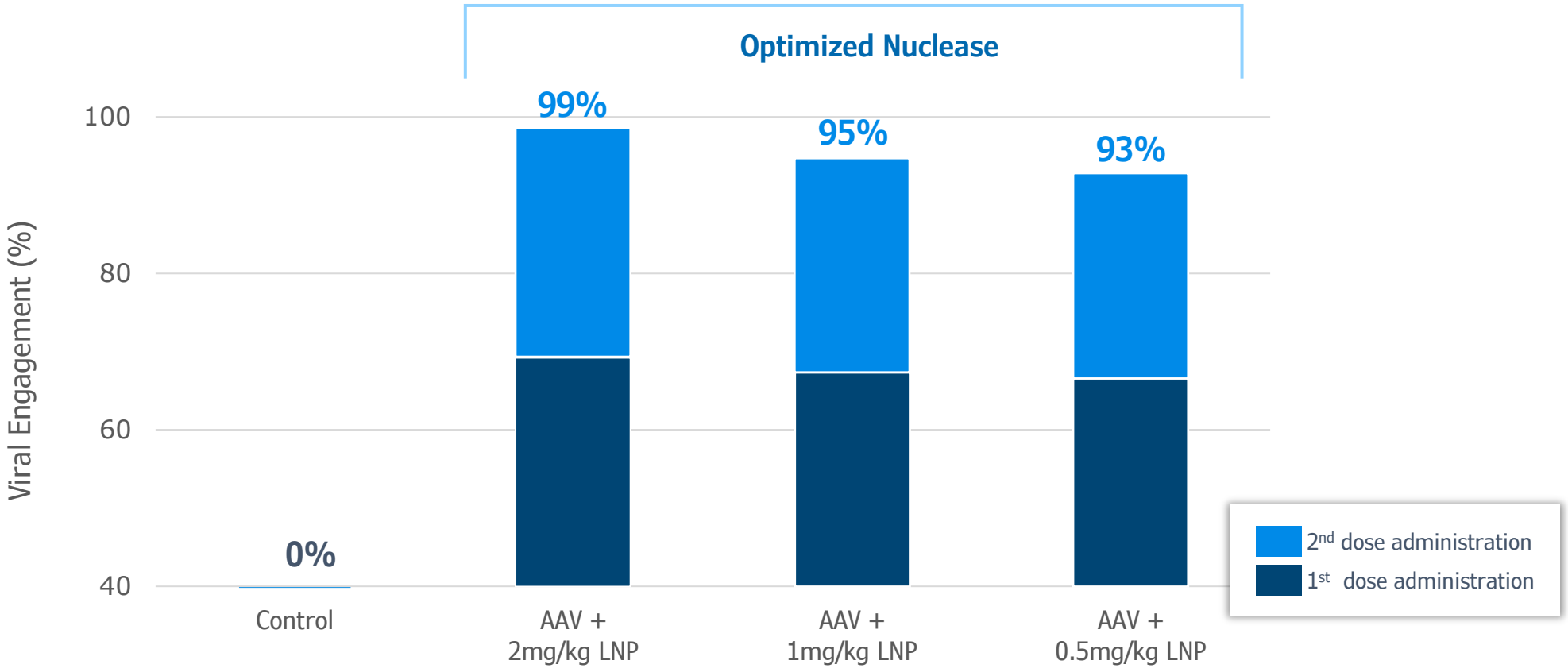
PBGENE-HBV demonstrates transient liver enzyme elevations <3x ULN and non-adverse changes in blood parameters

1. LNP Technology provided by Acuitas Therapeutics, Inc.

····· *Upper limit of normal*

Efficacy: NHP Study Demonstrated Up to 99% Viral Engagement, Suggestive of Strong Potential Efficacy Profile of PBGENE-HBV



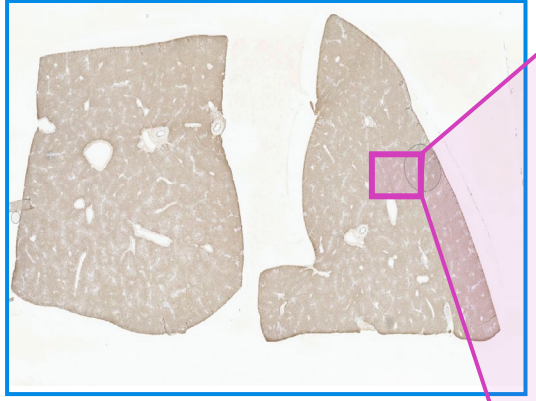


Increased editing observed with 2 dose administrations supports driving efficacy through multidosing in phase 1 study

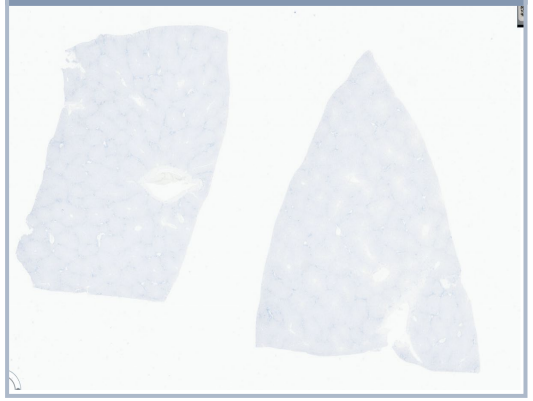
1. Final optimized candidate nuclease derived from optimized nuclease - only one amino acid difference with similar target product profile
2. Non-human primate (NHP) study- 2 doses of LNP 42 days apart; viral engagement (elimination + inactivation through indels) measured at D90
3. LNP Technology provided by Acuitas Therapeutics, Inc.

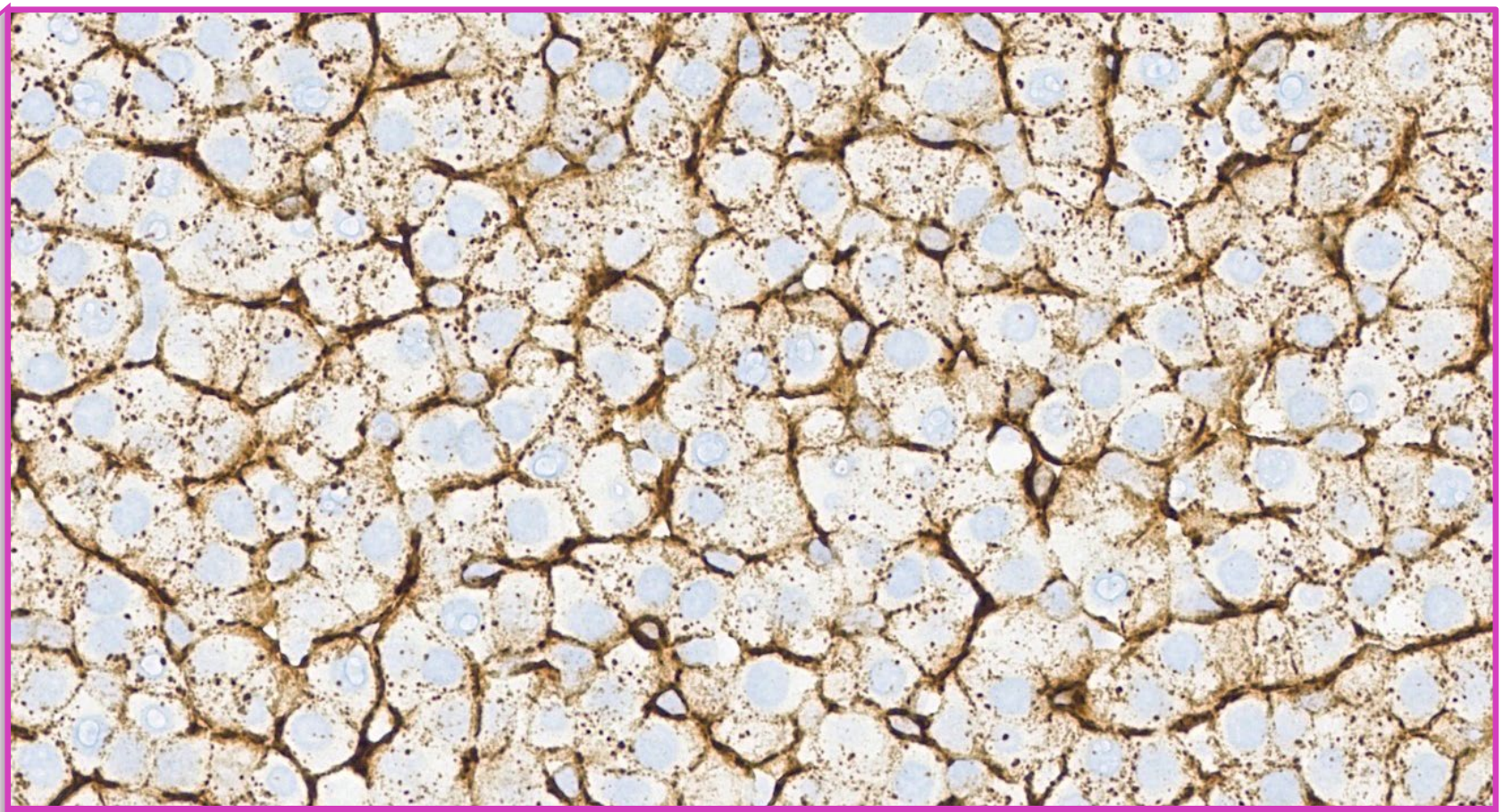
Efficacy: PBGENE-HBV Achieved Broad Distribution Across NHP Liver

PBGENE-HBV NHP Liver



Control NHP Liver

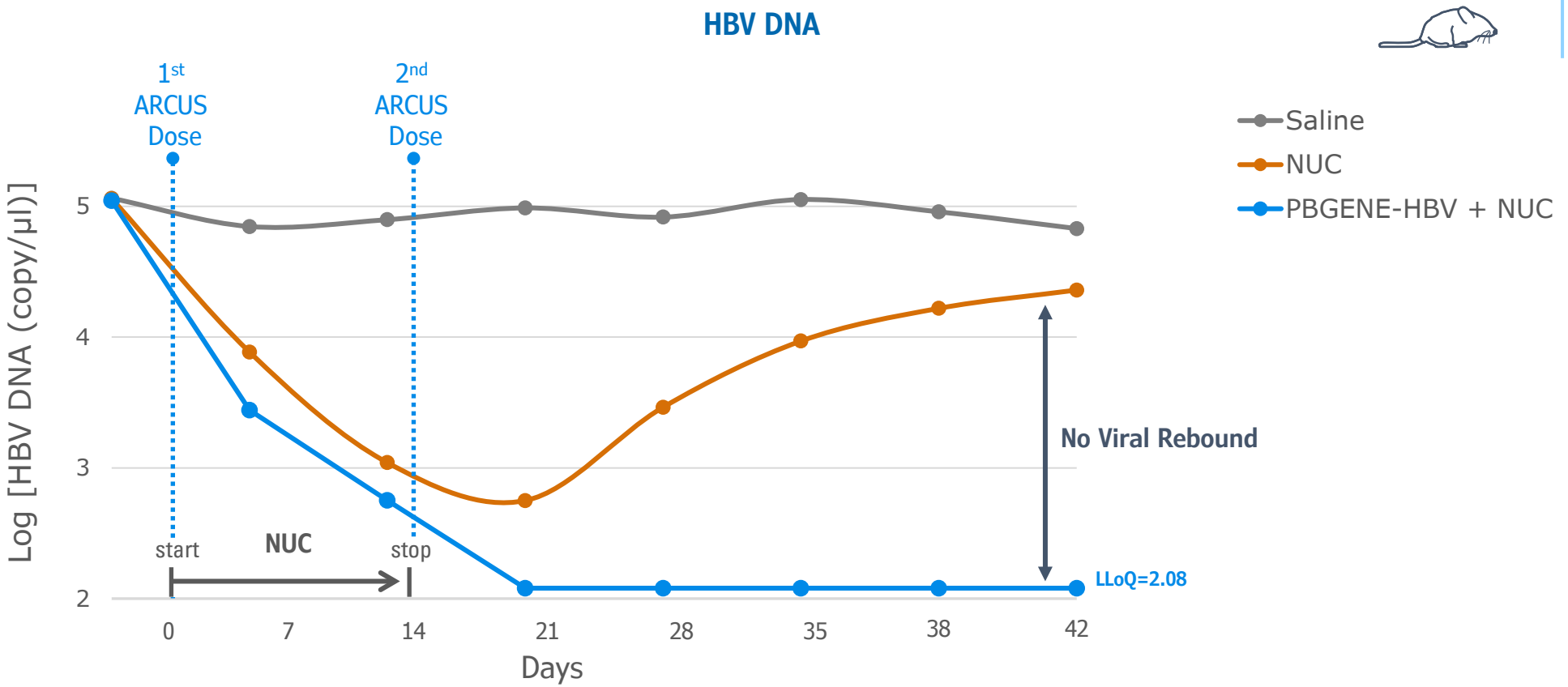




- ARCUS mRNA reaches all hepatocytes[1]
- ~10,000 ARCUS mRNA molecules per cell are detected in liver[2]

1. ISH quantification shows that ≥ 80% of cells are ARCUS+ after PBGENE-HBV administration; ~70-80% of the liver is hepatocytes and the primary target of the LNP formulation.
2. Assumes 1.22e5 cells/mg liver tissue in NHP.
3. LNP Technology provided by Acuitas Therapeutics, Inc.

Efficacy: PBGENE-HBV Significantly and Sustainably Reduced HBV DNA as a Monotherapy in Transgenic Mouse Model



Even after stopping NUC, PBGENE-HBV durably reduced HBV DNA as seen in combination cohort. Supports potential for stopping NUC and functional cures in phase 1 study

1. NUC = nucleos(t)ide analog, entecavir used in this study
2. HBV DNA levels measured in plasma; produced from multiple tissues in this mouse model
3. LNP Technology provided by Acuitas Therapeutics, Inc.

PBGENE-HBV: Robust Preclinical Safety and Efficacy Profile

IntDNA INACTIVATION:
Dose-dependent editing and HBsAg reductions

Nov 2023

SAFETY:
Comprehensive off-target analysis demonstrates high specificity

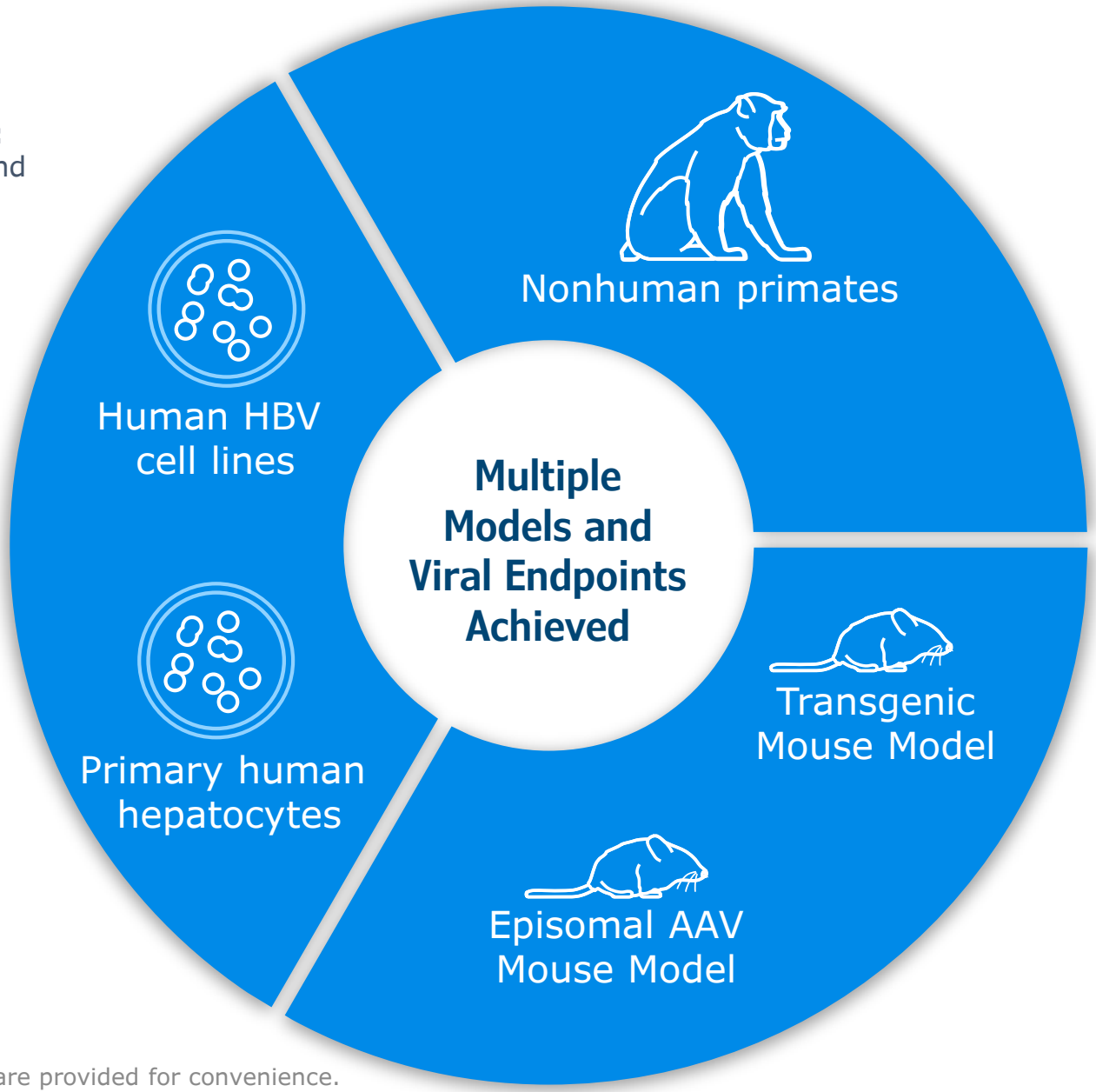
Nov 2023

cccDNA ELIMINATION:
Dose-dependent elimination of cccDNA and inhibition of viral markers

Nov 2023

SAFETY:
Comprehensive off-target analysis demonstrates high specificity

June 2024



cccDNA ELIMINATION:
99% viral engagement at highest dose

Nov 2023

SAFETY:
Multiple doses were well-tolerated

June 2024

IntDNA INACTIVATION:
Significant and sustainable reduction of HBV DNA in HBV transgenic mouse model

Nov 2023

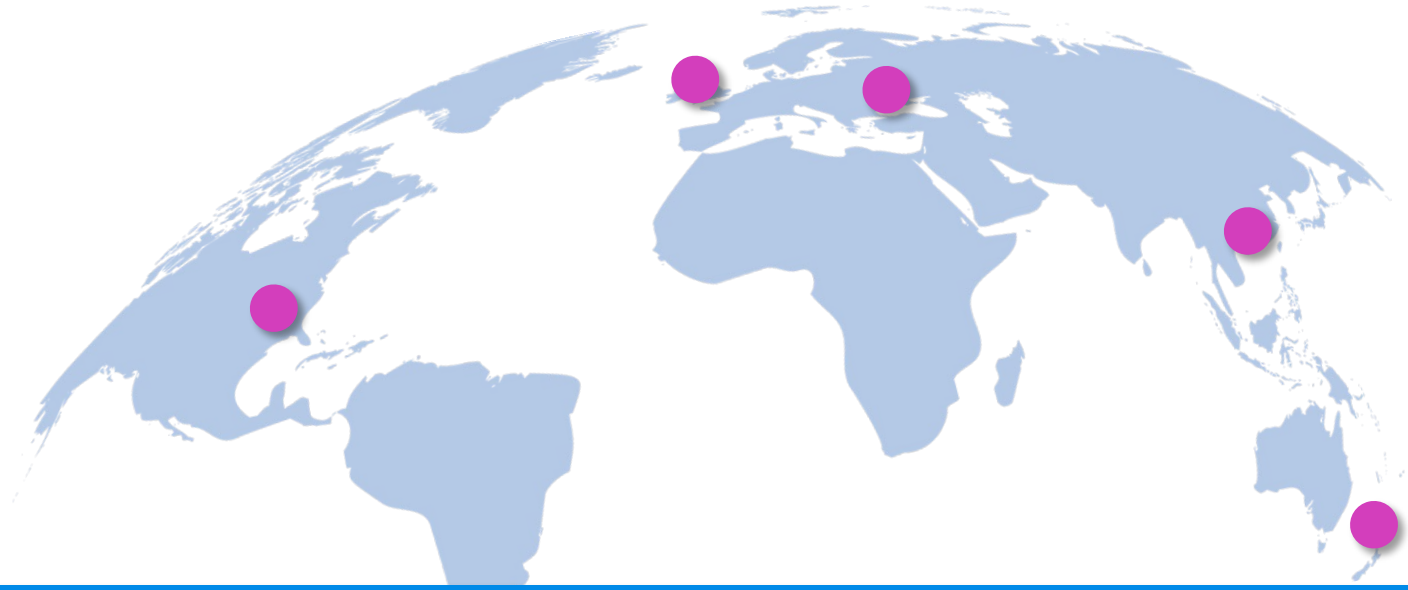
cccDNA ELIMINATION:
>95% durable HBsAg reduction across multiple dose levels in AAV mouse model

Nov 2023

Data sources with hyperlinks are provided for convenience.

Phase 1 Clinical Trial – Targeting Functional Cure

eliminateB



Global Phase 1 Study Across

Up to 5 Countries

Up to 45 Patients

Patient Population

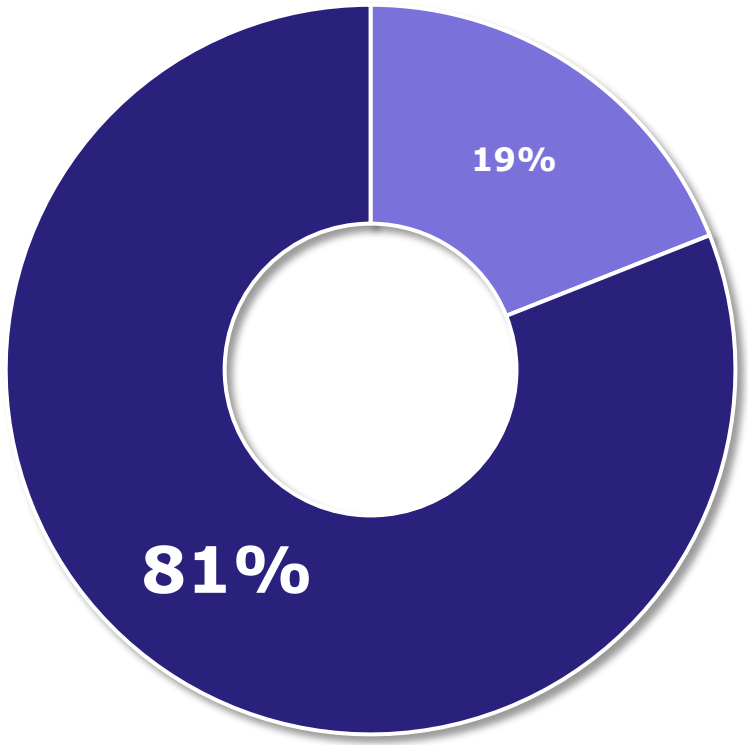
HBeAg-negative patients controlled on nucleos(t)ide analogs

ClinicalTrials.gov identifier NCT06680232.

Eliminate-B Trial is Starting in Largest Real World Patient Segment

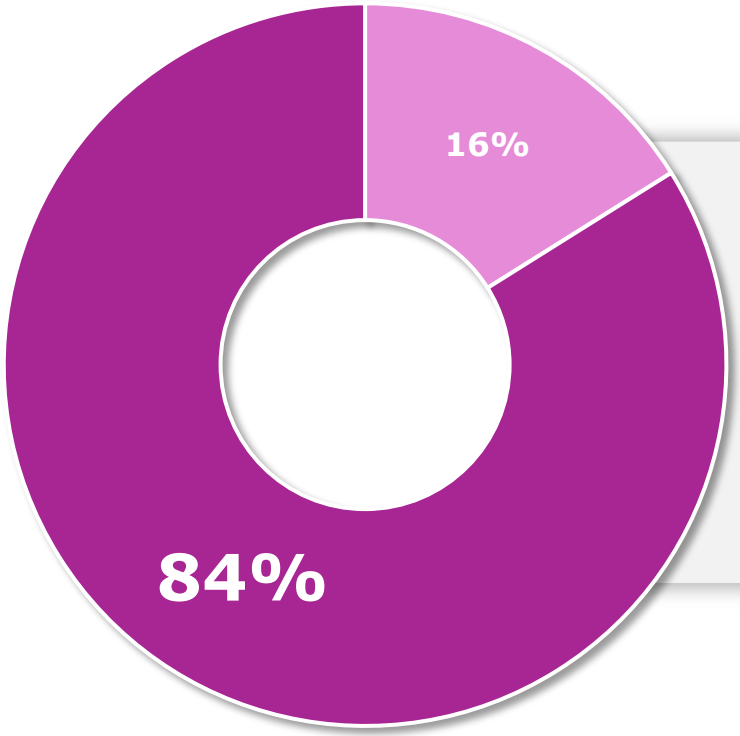
More than 80% of HBV Patients are E-Negative

81% of Diagnosed Patients are E-Negative



HBeAg Positive HBeAg Negative

84% of Patients Treated on Nucleos(t)ide Analogs are E-Negative



HBeAg Positive HBeAg Negative

Still only 1-3% achieve functional cure and up to 30% 10-year cumulative incidence for HCC

Literature search average including global meta-analysis.[1]
For the 45% of patients treated with nucleos(t)ide analogs leads estimated 34% achieve E antigen loss on TAF.[2]

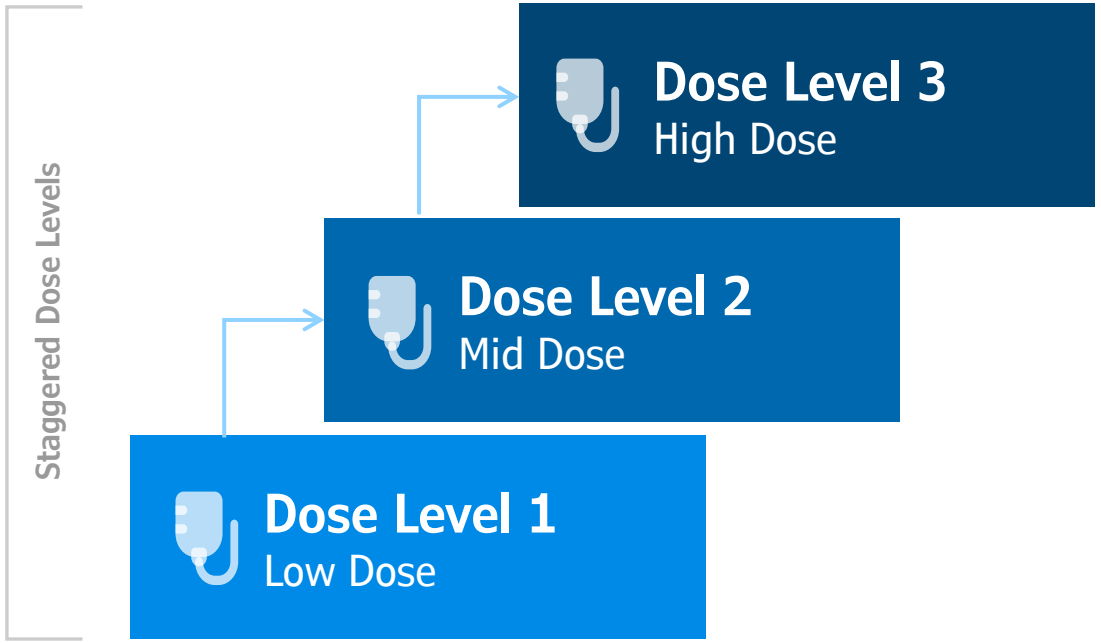
1. Tan M, et al. Postoperative long-term prognosis and its predictors in hepatocellular carcinoma patients after liver transplantation: a single-center experience. Front Oncol. 2021;10:7801814.
2. Chan HLY, et al. Long-term treatment with tenofovir alafenamide for chronic hepatitis B results in high rates of viral suppression and favorable renal and bone safety. Am J Gastroenterol. 2024;119(3):486-496.

Phase 1 Study Design

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

Part 1: Multiple Ascending Dose Escalation

N= 3-6 Patients at Each Dose Level
Finite Treatment: Patient Receives up to 3 dose administrations



3 + 3 standard design with sentinel dosing of patients

Safety & Efficacy Evaluation

Part 2 : Dose Expansion

Go Forward Dose

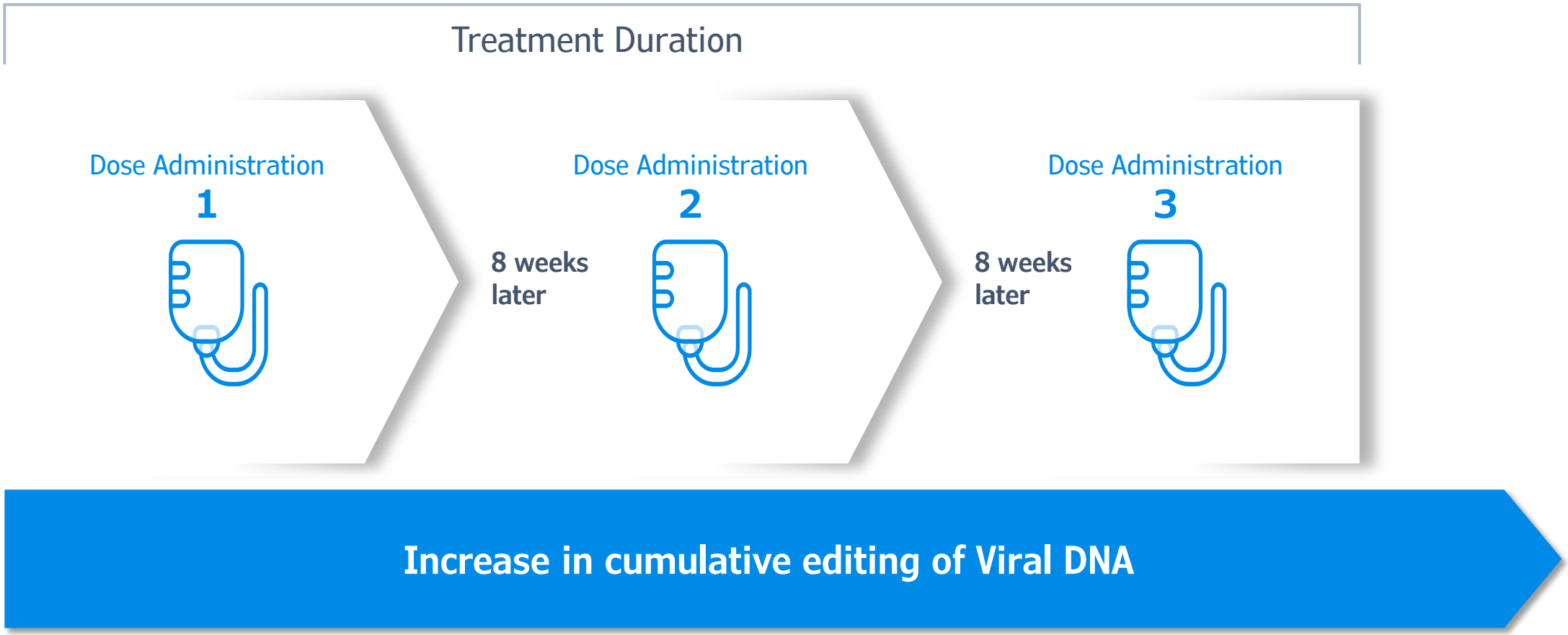
N = Up to 45 patients total across both Part 1 and 2 of Phase 1 study

Finite Treatment:
Patient receives maximum of 3 dose administrations in Part 1 of trial

ClinicalTrials.gov identifier NCT06680232.

PBGENE-HBV Finite Treatment Duration:

Increased Cumulative Viral Editing Through Up to Three Dose Administrations



ClinicalTrials.gov identifier NCT06680232.

Phase 1 Study Goals to Demonstrate Both Safety and Efficacy

Key Endpoints

Safety determined by:

Frequency and severity of dose-limiting toxicities (DLTs)

Efficacy determined by:

Antiviral activity through fixed duration PBGENE-HBV treatment

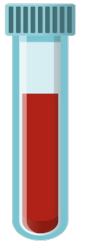
Monitoring Biomarkers

Reduction/Negativity in HBsAg:
Change from baseline in HBsAg and anti-HBs levels

— Proportion of participants with **undetectable HBsAg levels** at each study visit

Sustained HBV DNA Negativity:

— Supported by **reduction in HBV RNA levels**



Tested using blood sample

- HBsAg
- HBcrAg
- HBV DNA & HBV RNA
- Anti-HBs
- Part II: liver biopsy

Driving Patient Outcomes

Stopping SoC Nucleos(t)ide Analogs:
Proportion of participants who can discontinue NA therapy

Partial Cure:
Defined as a **decline in HBsAg to < 50 IU/mL** & continued HBV DNA suppression and ALT concentrations <1.5 × ULN for **6 months post-therapy**

Functional Cure:
Defined as **sustained seroclearance of HBsAg** with/without seroconversion of HBsAb & continued HBV DNA suppression for **>6 months post-therapy**

ALT, alanine aminotransferase; cccDNA, covalently closed circular DNA; HBsAg, hepatitis B surface antigen; HBV DNA, hepatitis B virus DNA; HBV RNA, hepatitis B virus RNA; SoC, standard of care; ULN, upper limit of normal.
ClinicalTrials.gov identifier NCT06680232.

United Kingdom

Kosh Agarwal, MD

Hepatologist and Transplant Physician Institute of Liver Studies, King's College Hospital NHS Foundation Trust

Moldova

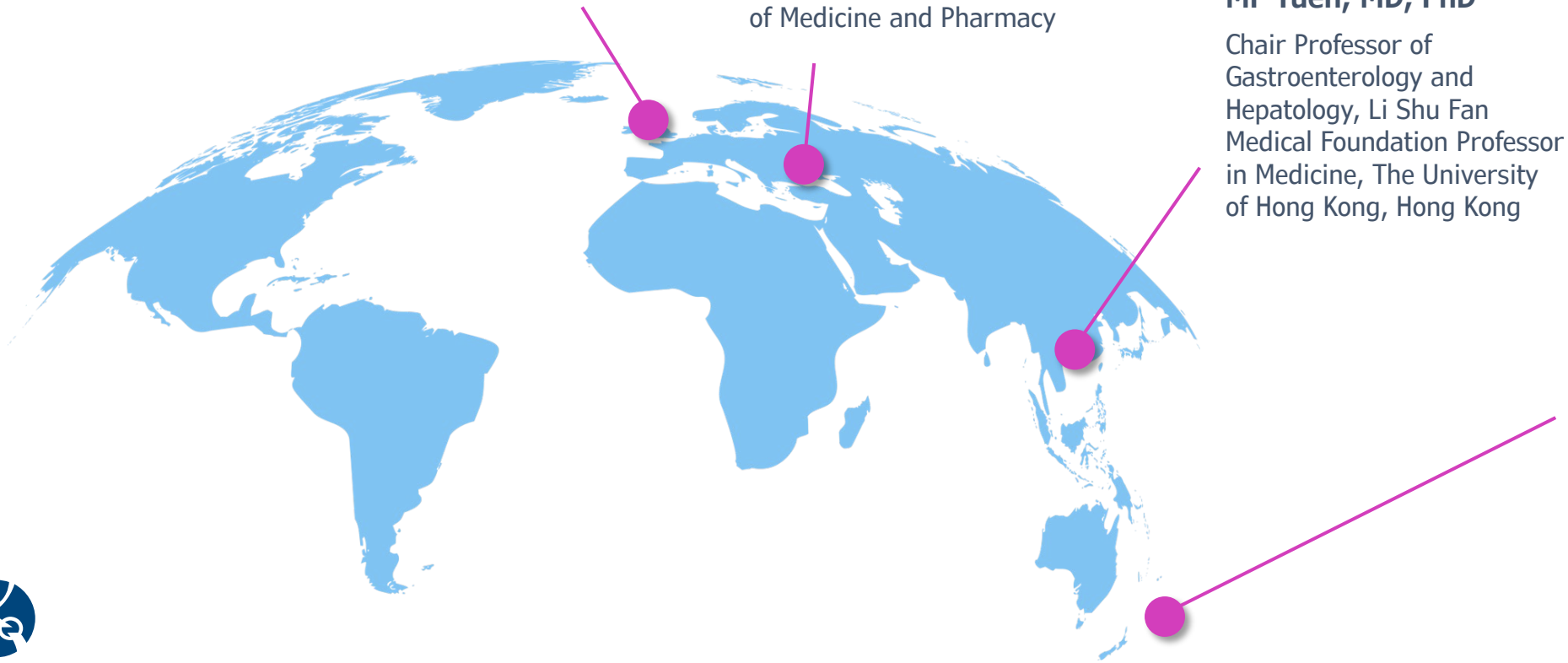
Alina Jucov, MD, PhD

Principal Investigator, ARENSIA Research Clinic, Chisinau, Moldova
Assistant Professor, Department of Gastroenterology, State University of Medicine and Pharmacy

Hong Kong

MF Yuen, MD, PhD

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



New Zealand

Ed Gane, MD

Professor of Medicine, University of Auckland, Deputy Director, New Zealand Liver Transplant Unit Chief Medical Advisor, New Zealand Clinical Research

PBGENE-HBV: Establishing A New Paradigm for Cures in cHBV

Goal of Sterilizing Cure with Initial Target of Functional Cure
Uniquely designed to eliminate cccDNA and inactivate integrated DNA, addressing the root cause of HBV

Right Clinical Design Backed by Robust Preclinical Data & Regulatory Alignment
Demonstrated 99% viral DNA editing and safety profile in NHPs

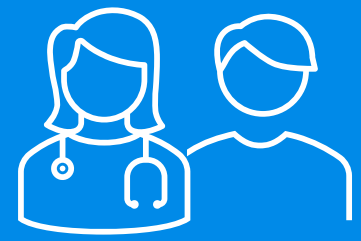
Global Phase 1 Study Up to 5 Countries in Largest Real World Patient Population
Phase 1 trial designed to rapidly translate preclinical safety and antiviral efficacy in patients, starting with the largest E-negative patient population

Clinical Program and Execution Guided by the Leading HBV Investigators and Advisors
Experienced Scientific Advisory Board and Phase I investigators across both infectious disease and gene editing clinical studies

On Track for Clinical Data Readouts in 2025
Phase 1 study initiated & moving towards dosing patients

H1 '25: lower dose cohorts including safety and efficacy

H2 '25: higher dose cohorts including safety, efficacy and durability



Next Steps

- Finalize additional CTA and IND regulatory approvals
- In parallel, dose patients across multiple global clinical sites

PRECISION
BIOSCIENCES

Target CTA/IND
2025

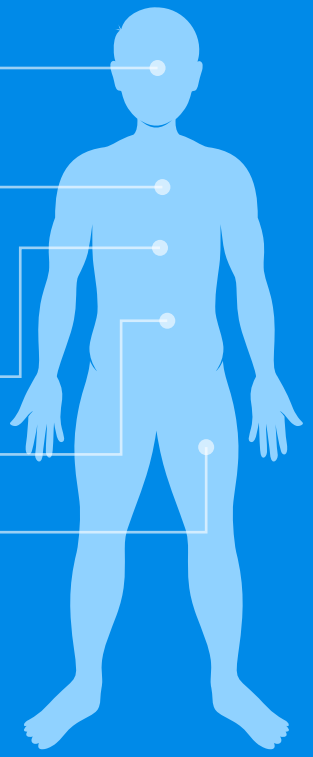
PBGENE-3243

Mutant Mitochondrial DNA Elimination

m.3243 Mitochondrial Disease Currently Lacks a Curative Treatment
~20K m.3243 mitochondrial disease *patients in the US alone*

m.3243-associated mitochondrial diseases often lead to defects in energy production affecting high energy-demand tissues (e.g. skeletal muscle)

- Chronic progressive external ophthalmoplegia
- Respiratory failure
- Exercise Intolerance
- Multi organ dysfunction
- Impaired Mobility
- Generalized weakness



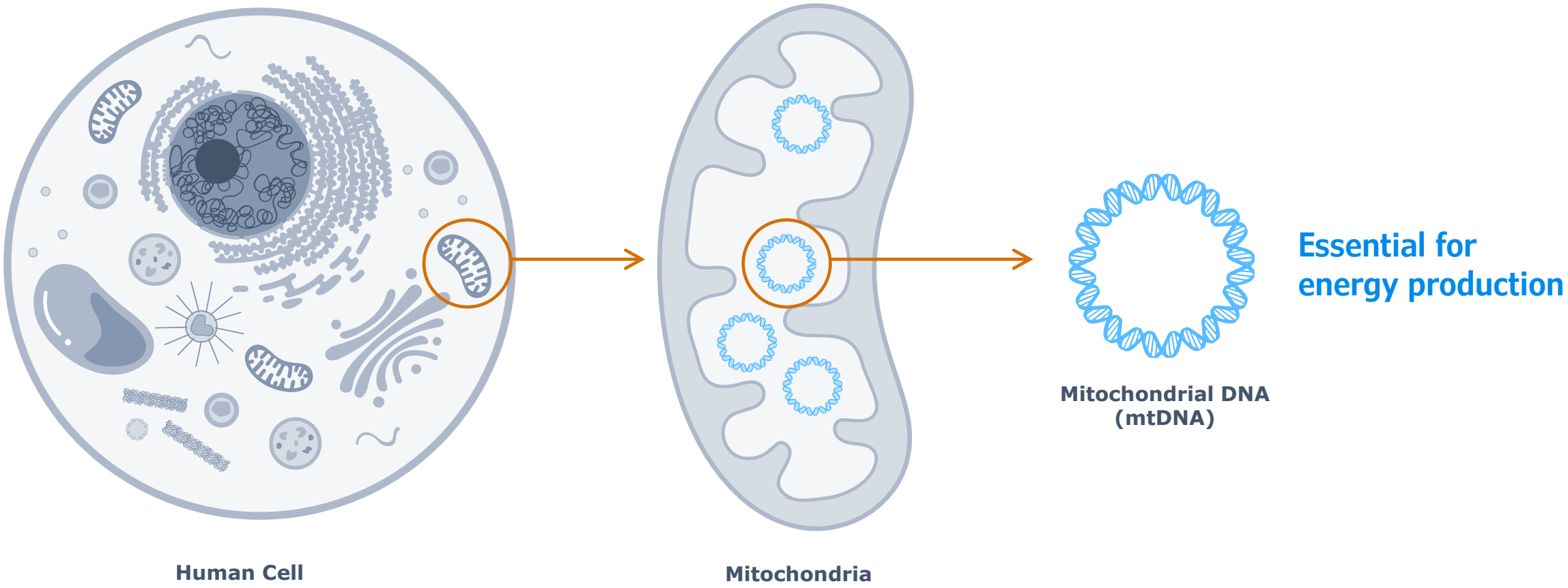
Patients today lack curative treatments and receive supportive care only through "mito cocktails"[1]

m.3243A>G

m.3243-associated mitochondrial disease estimated at

Sources:
1. https://www.ninds.nih.gov/health-information/disorders/mitochondrial-myopathies; https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6938233/
2. Calculated based disease epidemiology studies and secondary literature

Multi-Copy Mitochondrial DNA (mtDNA) is Critical for Mitochondrial Function



PBGENE-3243 Distinguishes a Single Base Difference at m.3243

m.3243A>G

- **Mutation Prevalence of 1/500**[1]
- **~36% of Mitochondrial Diseases are driven by m.3243A>G**[2]

m.3243 associated mitochondrial disease estimated at **~20k patients in the US alone**

Mutant mtDNA sequence

5′-C A G **G** G C C C G G T A A T C G C A T A A A-3′

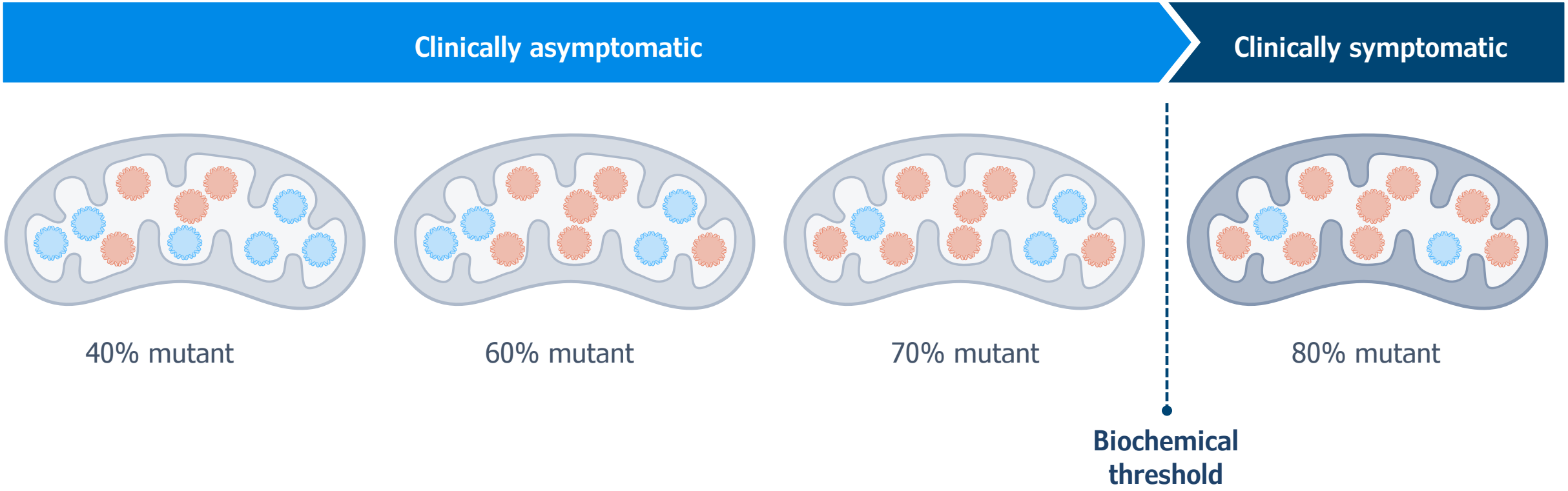
5′-C A G **A** G C C C G G T A A T C G C A T A A A-3′

Wild-type (healthy) mtDNA sequence

1.Manwaring et al 2006 Population prevalence of the MELAS A3243G mutation. Mitochondrion
2.Schon et al., 2023, National Mitochondrial Disease Registry in England... Euromit2023 Conference, Bologna, Italy, June 13, 2023;

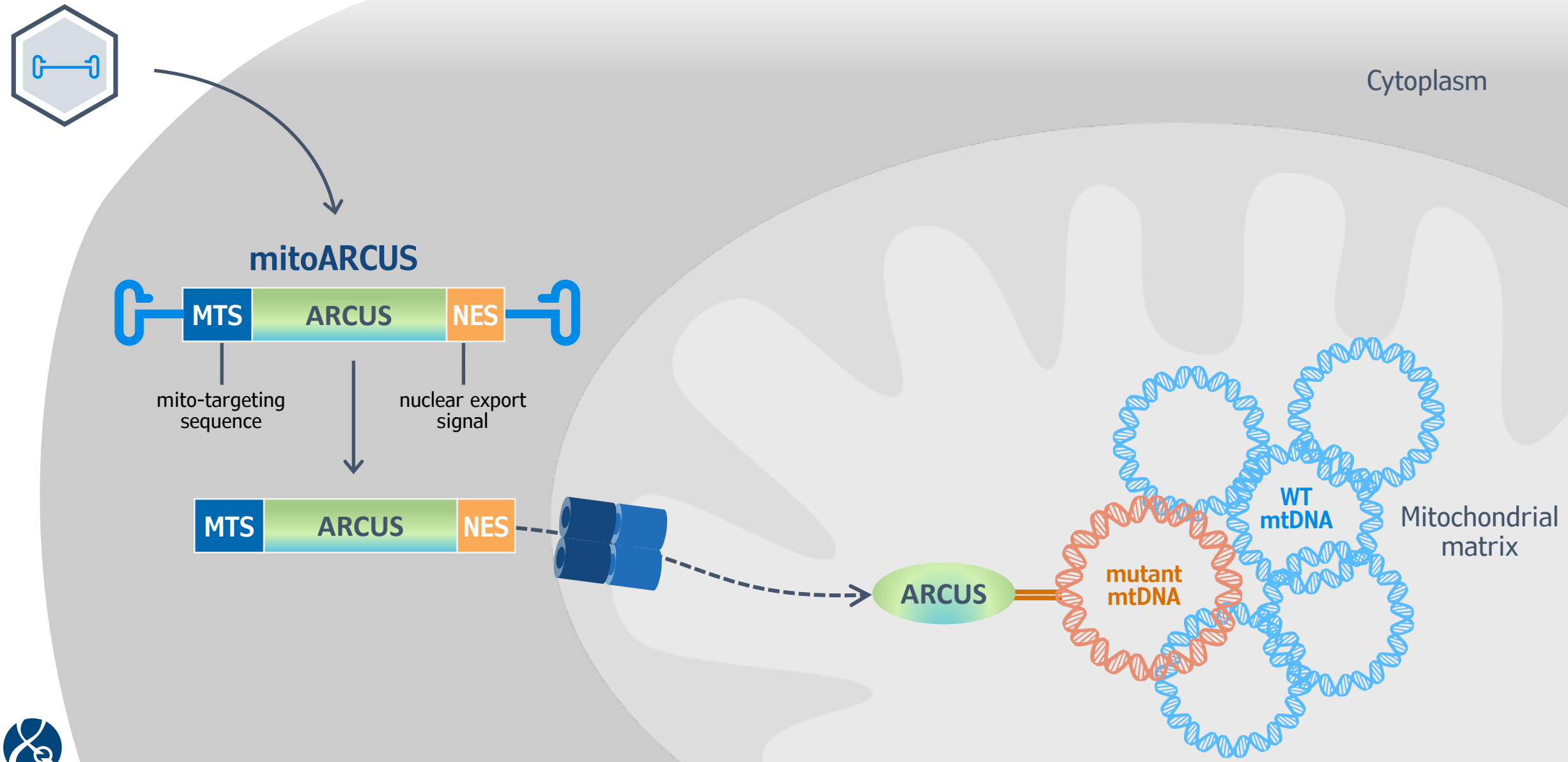
mtDNA Mutations Are Commonly Heteroplasmic

Situation where two or more mtDNA variants exist in same mitochondria

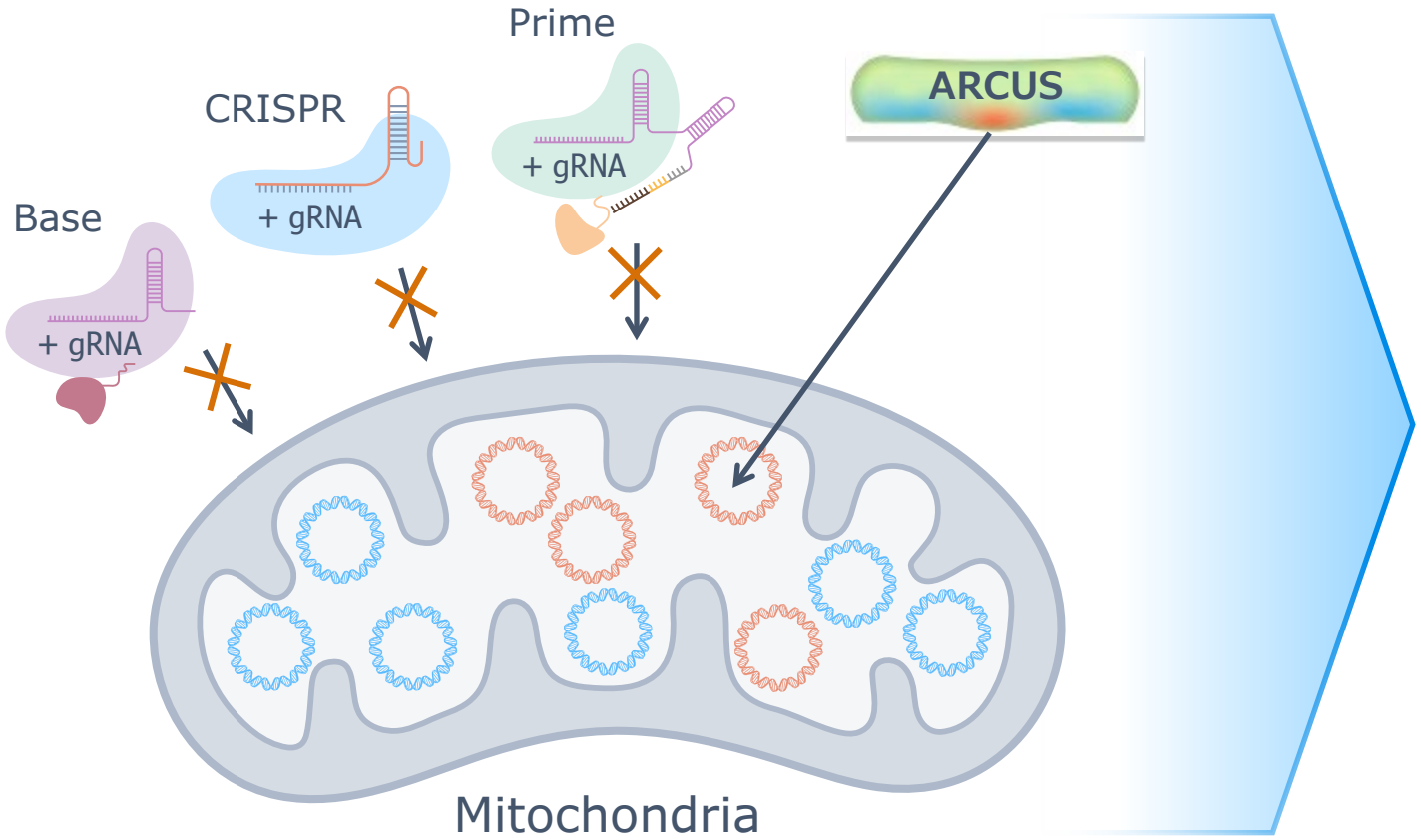


mtDNA, mitochondrial DNA.

mitoARCUS Therapeutic Approach to Shift Heteroplasmy



Simplicity: ARCUS Can Go Where Few Other Gene Editors Can Follow



nature metabolism

Article

https://doi.org/10.1038/s42255-023-00932-6

Efficient elimination of MELAS-associated m.3243G mutant mitochondrial DNA by an engineered mitoARCUS nuclease

Received: 18 May 2023

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Published online: 30 November 2023

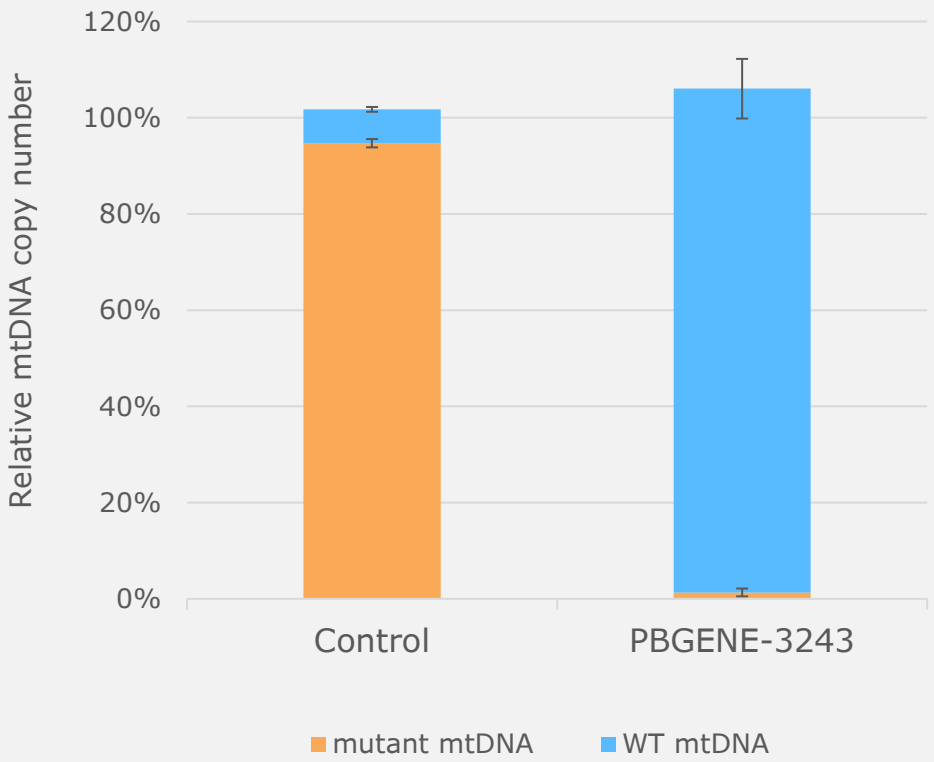
Wendy K. Shoop[1,2], Janel Lape[1], Megan Trum[1], Alea Powell[1], Emma Sevigny[1], Adam Mischler[1], Sandra R. Bacman[3], Flavia Fontanesi[3], Jeff Smith[1], Derek Jantz[1], Cassandra L. Gorsuch[1] & Carlos T. Moraes[3]

PBGENE-3243 Program Highlights

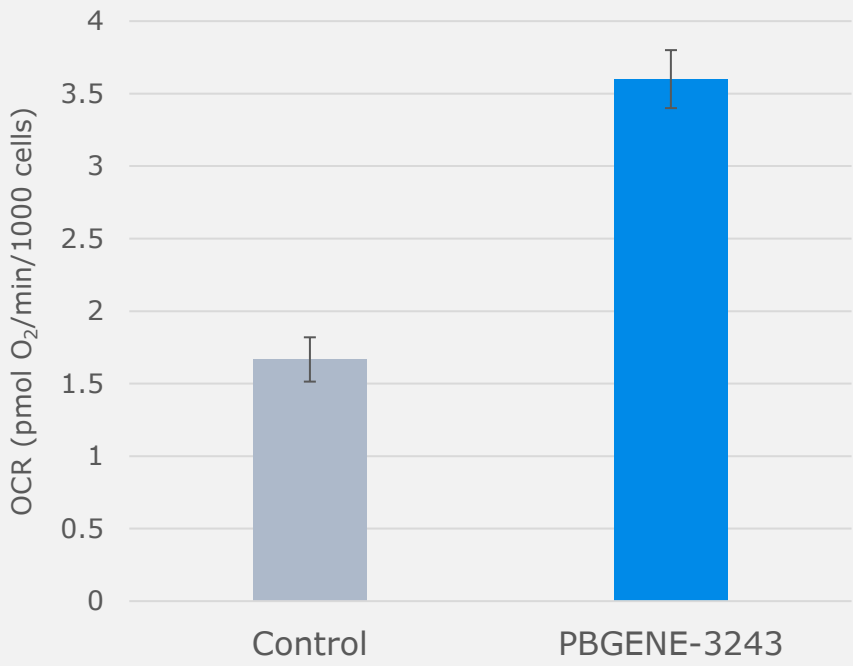
- Single component nature of ARCUS allows specific editing of mutant mtDNA with no off-target editing
- ARCUS-induced heteroplasmy shift resulting in improved mitochondrial and respiratory function in edited cells
- No evidence of mitoARCUS editing nuclear DNA

PBGENE-3243 Improved Mitochondrial Function

On target efficacy



Impact on energy production



mtDNA, mitochondrial DNA; WT, wild-type; experiments in cybrid cells.

Precision BioSciences Offering Meaningful Catalysts in Next 6-18 Months

Ample cash runway to fund multiple potential regulatory submissions and clinical data readouts

CASH RUNWAY INTO 2H 2026

Potential to Advance 3 Programs to Phase I Clinical Data

Bolstered by

~$50M[2]
in business development (last 12 months)

+

$40M
in equity raise @ $16/share (March '24)

Expected Inflection Points

ECUR-506 for OTCD — Complete clinical response in first patient dosed; OTC-HOPE Phase 1/2 trial expected to finish enrollment in 2025 with data readout for all patients expected in 1H 2026

PBGENE-HBV — CTA approved in Moldova, Hong Kong, and New Zealand with additional regulatory applications in-progress; Data expected in 2025

PBGENE-3243 — CTA and/or IND Filing in 2025; Data expected in 2026

[1]Source: Form 8-K filed 1/8/25 does not includes $2.5M from TG Therapeutics and additional cash inflows in 2025. Cash balance includes cash, cash equivalents, and restricted cash. [2]Includes upfront payments and expected near-term milestones.