PRECISION
BIOSCIENCES

Corporate Deck

September 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 safety, efficacy and benefit of our and our partners' and licensees' product candidates (including PBGENE-HBV and PBGENE-DMD as a potentially curative therapies targeting root cause); the safety, efficacy and expected benefit of our gene editing approaches including editing efficiency, and the suitability of ARCUS nucleases due to their cut, size and simplicity for gene insertion, gene elimination and gene excision and differentiation from other gene editing approaches; the expected timing of regulatory processes and clinical operations (including IND and/or CTA filings, studies, enrollment and clinical data for PBGENE-HBV, PBGENE-DMD, PBGENE-3243 and 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 potential target product profile of PBGENE-DMD to potentially provide a best-in-class therapeutic profile; the ability of PBGENE-DMD to provide significant functional dystrophin protein production in different types of muscle at levels expected to provide therapeutic benefit in a humanized DMD-diseased mouse model; the ability of PBGENE-DMD to edit satellite muscle stem cells, a potential predictor of durable functional benefit; the suitability of PBGENE-DMD for the treatment of DMD and restoration of functional dystrophin protein at a therapeutic level, expectations about the commercial potential, market opportunity, operational initiatives, strategies, and further development of our programs and those of our collaboration partners; the translation of results in preclinical studies of ARCUS nucleases including PBGENE-HBV and PBGENE-DMD to clinical studies in humans; the potential eligibility of PBGENE-DMD for a Priority Review Voucher (PRV) valued ~$150 million upon BLA approval; expectations about our and our partners' operational initiatives, strategies, and further development of our programs; expectations and updates around our partnerships and collaborations and our ability to enter into new collaborations, license agreements or other arrangements; our expected cash runway and available credit; the sufficiency of our cash runway extending into the second half of 2027 and potential to advance 3 programs to Phase 1 clinical data; expectations about achievement of key milestones and receipt of any milestone, royalty, or other payments; expectations regarding our liquidity and capital resources; and anticipated timing of clinical data. 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," "suggest," "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 Quarterly Report on Form 10-Q for the quarterly period ended June 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.

Precision BioSciences Leadership Team Brings Strategic and Operational Experience

Michael Amoroso
Since Oct 2021
President &
Chief Executive Officer

Jeff Smith, Ph.D.
Since March 2006
Chief Research Officer,
Co-Founder

Cassie Gorsuch, Ph.D.
Since June 2016
Chief Scientific Officer

Alex Kelly
Since Oct 2020
Chief Financial Officer

Cindy Atwell
Since April 2019
Chief Development &
Business Officer

Juli Blanche
Since May 2022
Chief People Officer

Dario Scimeca
Since June 2019
General Counsel

Neil Leatherbury
Since March 2017
Senior VP, CMC

Naresh Tanna
Since Jan 2022
Chief of Staff & Head of
Investor Relations

Precision BioSciences is a Clinical Stage In Vivo Gene Editing Company

Multiple Programs in or Nearing Clinical Data

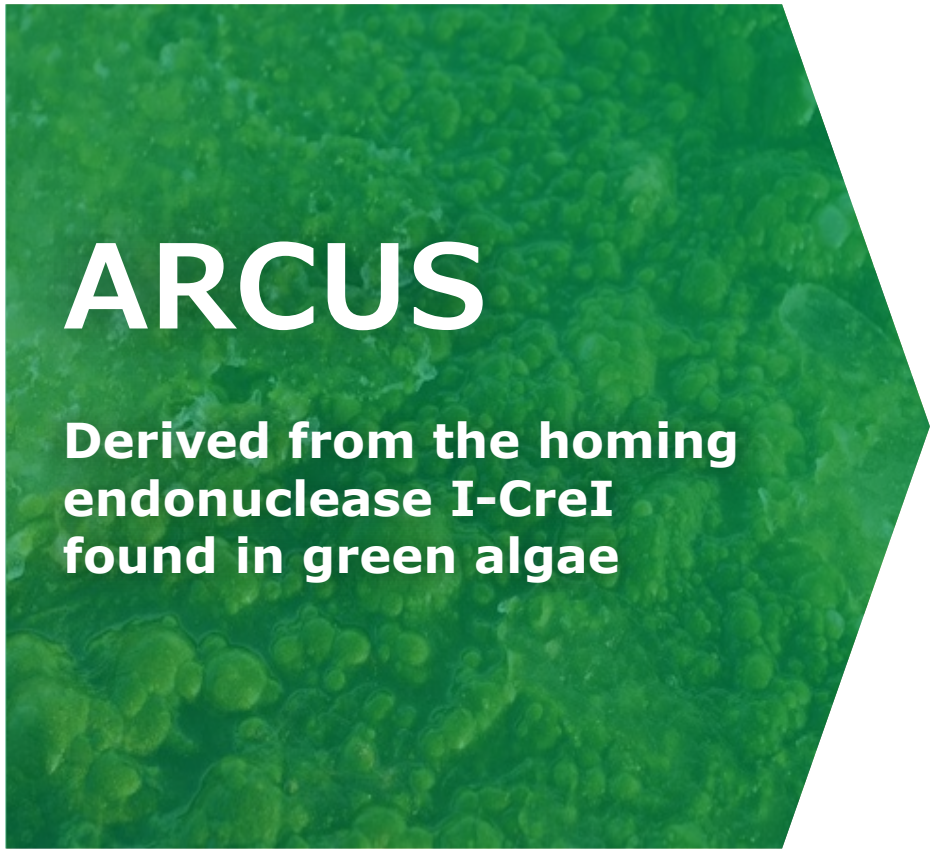
			Expected First Clinical Data Timing
Wholly owned	**PBGENE-HBV** ***In Vivo* Gene Editing Program for Chronic Hepatitis B**	〉 Investigational New Drug (IND) clearance and Fast Track designation received in United States 〉 Global phase 1/2a ELIMINATE-B trial on-going with low dose cohort demonstrating safety and antiviral activity with HBsAg reductions 〉 Data updates expected throughout 2025	**2025** eliminate B
	PBGENE-DMD ***In Vivo* Gene Editing Program Duchenne Muscular Dystrophy**	〉 PBGENE-DMD is Precision's second wholly owned program 〉 Targeting IND and/or CTA filing in 2025 〉 Potentially eligible for Priority Review Voucher (PRV) valued approximately $150M upon BLA approval	**2026**
Partnered Program Employing ARCUS Nuclease	**iECURE-OTC***	〉 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**

Three Clinical Datasets Read-Out through 2026 and Within Cash Runway
Cash Runway Now Extended into 2H 2027

*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

We Believe our Gene Editing Platform is Best Suited for the Indications We are Pursuing

ARCUS is Our Proprietary Gene Editing Platform: Naturally Evolved to Drive High Efficiency Editing



- **ARCUS wholly owned by Precision BioSciences**
- **Evolved to safely edit by inserting in genome, adding function**
  - CRISPR-based editing tools engineered from enzymes evolved to knockout DNA only
- **> 65 patents issued** covering ARCUS and in vivo gene editing
- **Unique Features of ARCUS Nucleases Drive Defined Outcomes***
  - **CUT:** 3’ overhang cuts drive homology-mediated insertion and re-ligation after excision with high efficiency
  - **SIZE**: Small nucleases (~1000bp) enables delivery flexibility, driving safety and efficacy
  - **SIMPLICITY**: DNA recognition and catalytic activity in one protein with no gRNA required, enabling high specificity and efficiency

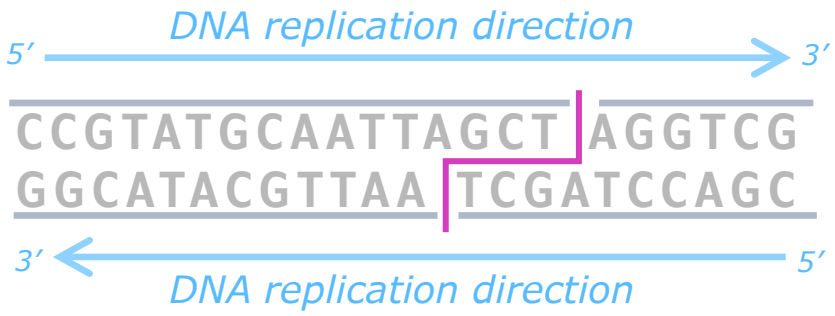
***Defined Outcome** = desired therapeutic edit and effect

The ARCUS Cut is Uniquely Designed to Drive Defined Outcomes

ARCUS cut leads to HDR or “Perfect” Re-ligation

Generates a 3′ overhang required for Defined Outcomes by HDR or Perfect Re-ligation

The ARCUS Cut



Cas9

CCGTATGCAATTA | GCTAGGTCG
GGCATACGTTAAT | CGATCCAGC

Blunt cut induces NHEJ

TALEN/ZFN

CCGTATGCA | ATTAGCTAGGTCG
GGCATACGTTAAT | CGATCCAGC

Cas12a

CCGTATGCAATTAGCTA | GGCAT
GGCATACGTTAATCGATCCGT | A

Staggered cut in wrong 5′ direction for HDR, therefore NHEJ repair

Base Editors

CCGTAGCAATTAGCTA | GGATG
GGCATCGTTAATCGATCCGTAC

Prime Editors

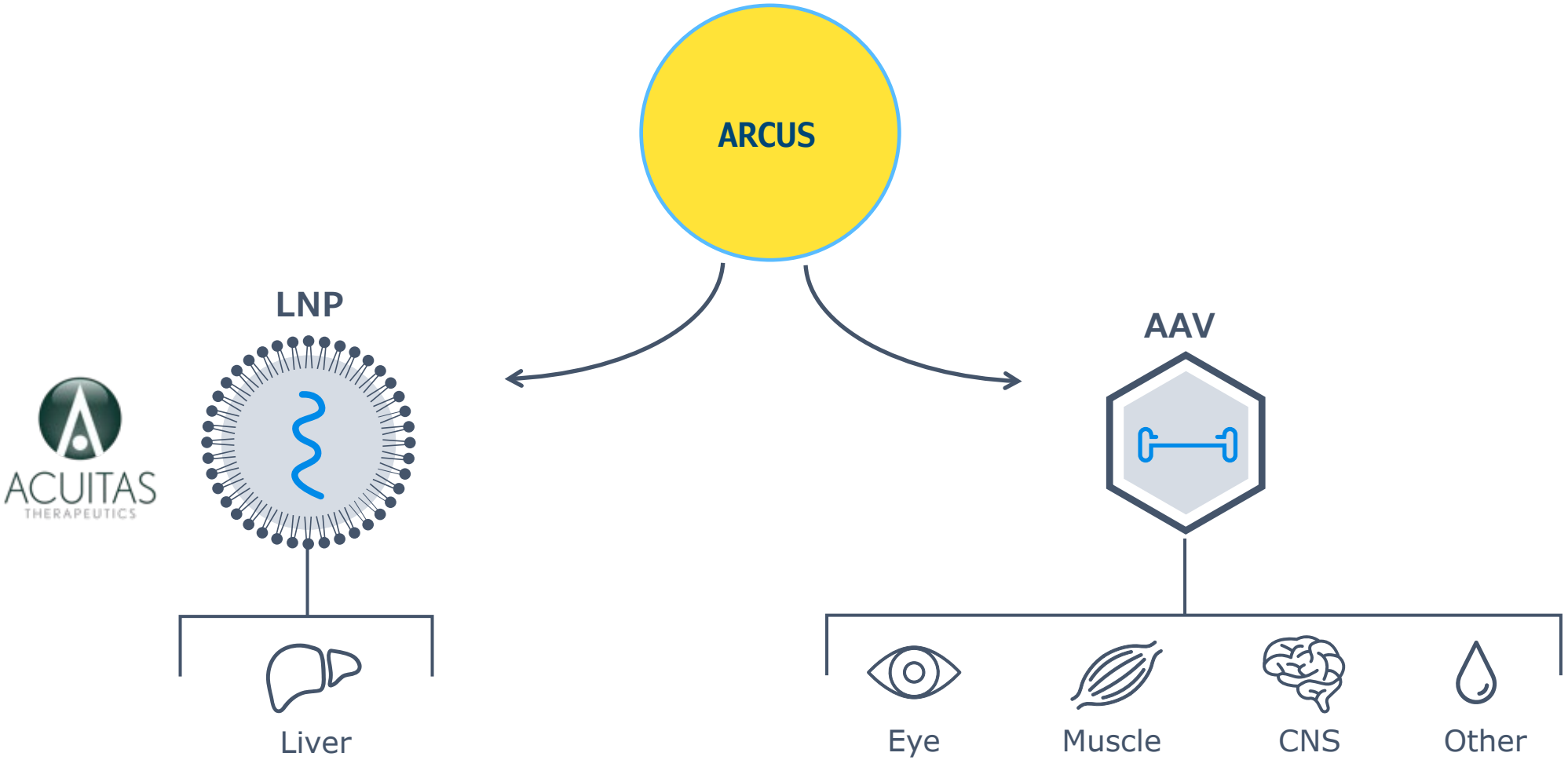
CCGTATGCAATTAGCTA | GGCAT
GGCA | TACGTTAATCGATCCGTA

Single, double, or unintended DNA breaks results in interrupted repair state*, HDR not attainable

**Genotoxic effects of base and prime editing in human hematopoietic stem cells*; Nature Biotechnology, 2023, Fiumara, M.

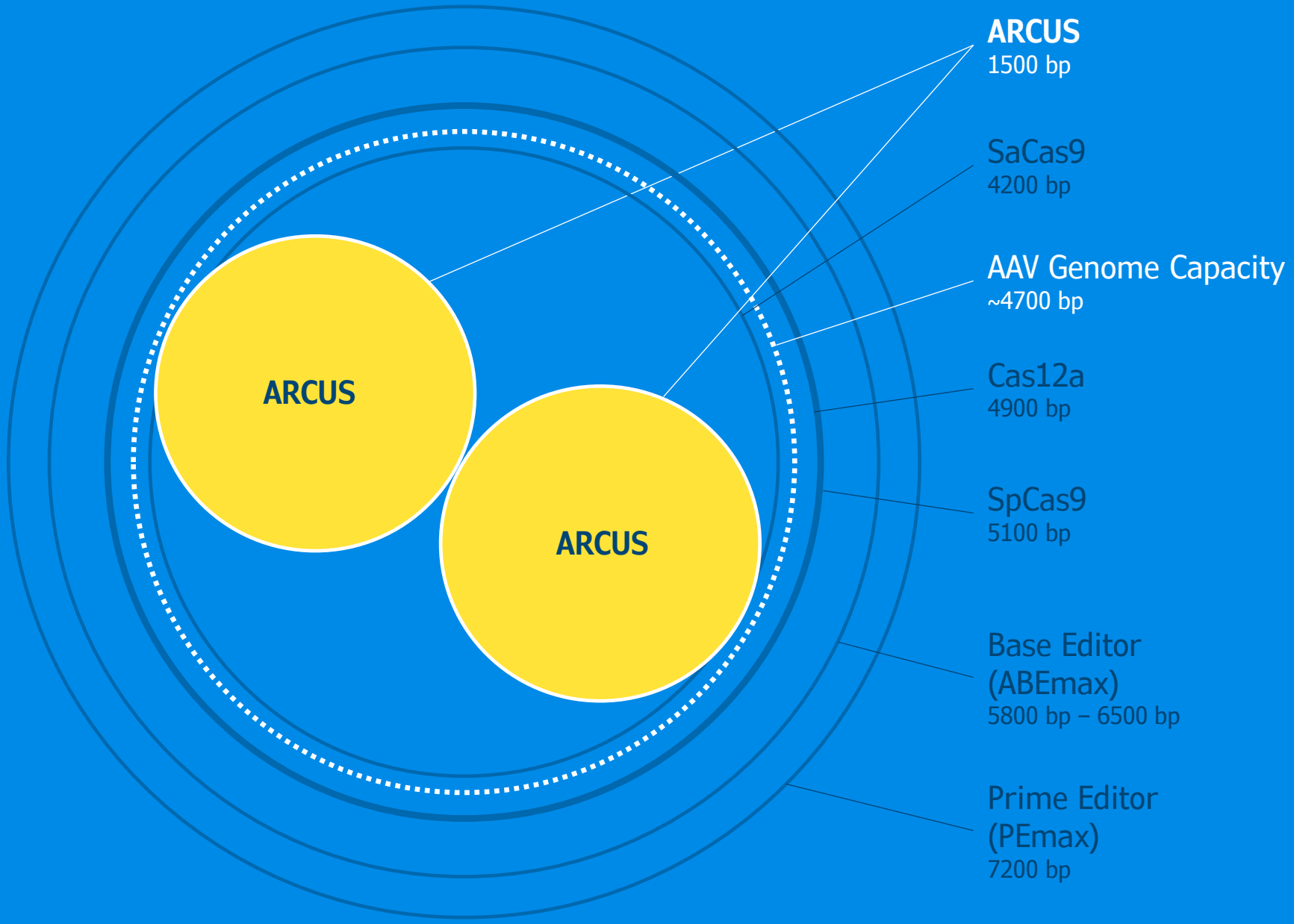
Size Matters for Where You Can Deliver

ARCUS can use different delivery vehicles to target diverse tissue types



Size Matters for **What** You Can Deliver:

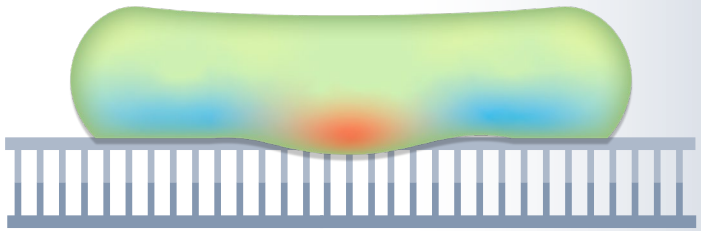
Small ARCUS Size Allows Two Nucleases in One AAV for **Gene Excision** as in **our PBGENE-DMD program**



Sizes include editor and expression machinery; ARCUS nuclease is 1062 bp

Simplicity: ARCUS is the Only Single Protein Component Editor

1 ARCUS



Single protein with a DNA recognition motif and catalytic activity all in one; **no guide RNA required**

Editing outcome not dependent on simultaneous delivery of multiple components leading to **higher efficiency**

Single component **requires less** AAV and potentially less LNP

〉 **Easy to deliver**

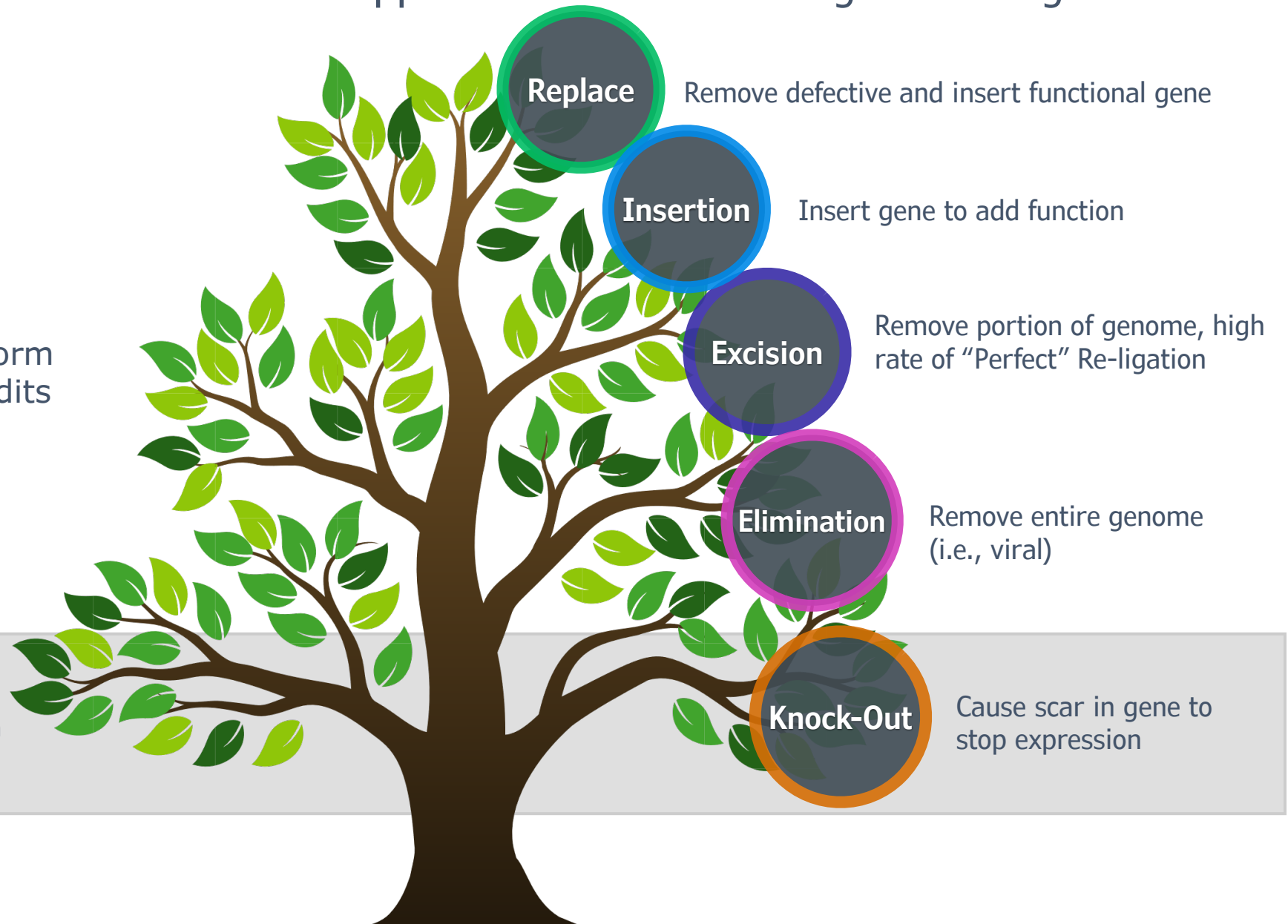
〉 **High efficiency**

〉 **Low dose improves safety**

ARCUS for the More Sophisticated Gene Edit

Designed by nature for a multitude of applications versus other gene editing modalities

ARCUS
Capability To Perform
"Sophisticated" Edits



"Low Hanging Fruit"
Commoditized application for all gene editors

Degree of Difficulty

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

ECURE

Partnered Program Employing ARCUS Nuclease

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]

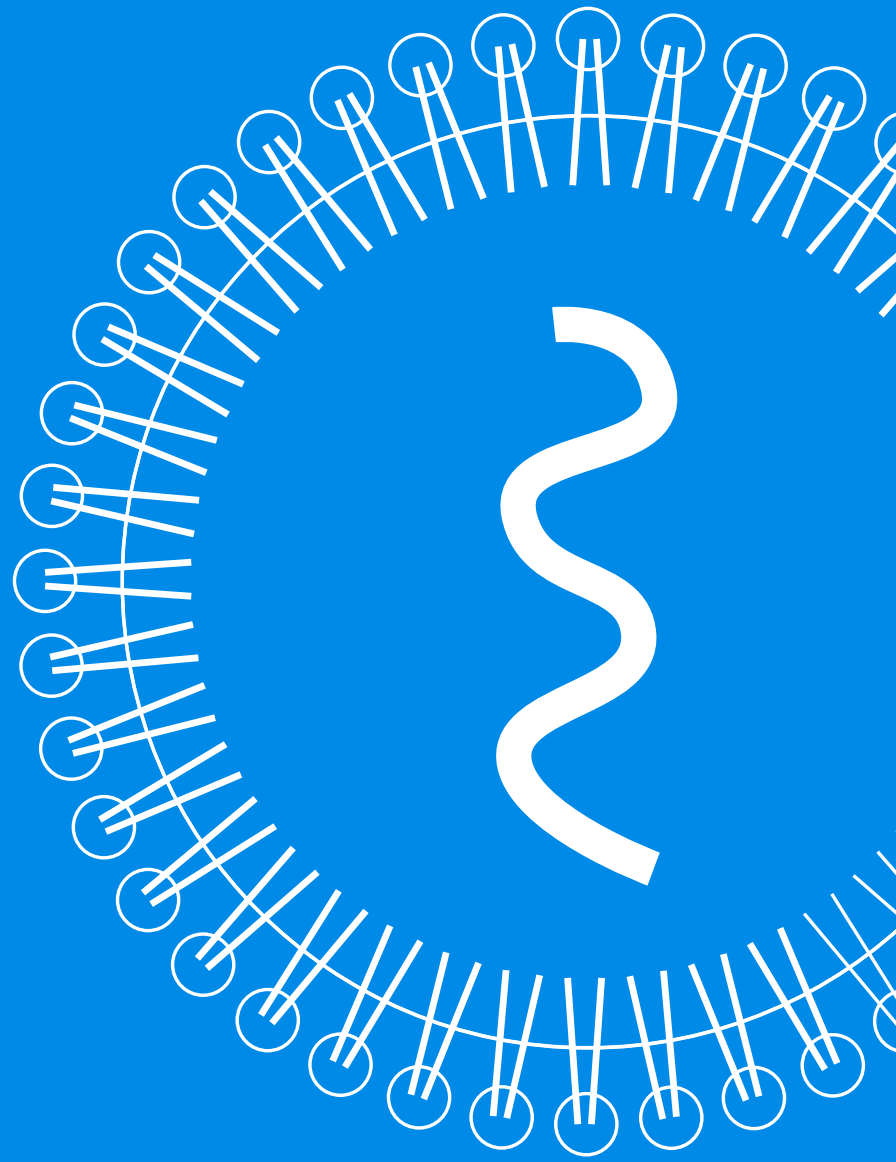
~10,000

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



Chronic Hepatitis B Multi-Billion Dollar Market Opportunity:

Large patient population currently on non-curative treatment options

> 300 Million
cHBV infections globally

Up to 2,400,000
cHBV infections in the US

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.

Chronic Hepatitis B Program Validated In Clinical Study:

Phase 1 Safety and Efficacy Established in Cohort 1 with Proof-of-Activity and Well-Tolerated Profile Observed in All Patients

- **Investigational New Drug Application cleared in the U.S. and Clinical Trial Applications approved in Moldova, Hong Kong, and New Zealand and United Kingdom**
  - Fast Track designation from FDA received in March 2025
  - Rapid patient recruitment with eight patients dosed by August 2025

- **PBGENE-HBV, the first gene editing medicine designed to eliminate cccDNA and provide complete cure for chronic HBV, was safe and well tolerated after all doses administered at lowest dose (Cohort 1, 0.2 mg/kg dose) and substantial reductions in HBsAg (47-69% from baseline) were observed in all patients**
  - No grade >2 treatment-related adverse events or serious adverse events
  - One of three patients demonstrated persistent ~50% HBsAg decline seven months after initial treatment

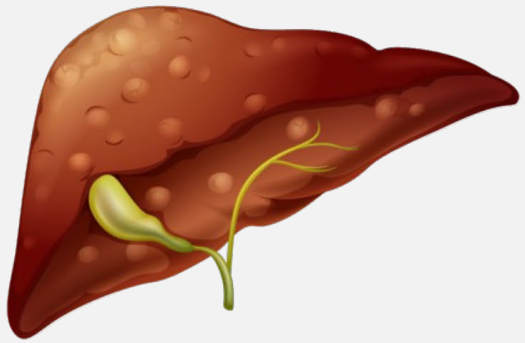
- **PBGENE-HBV shows good safety profile at 0.4 mg/kg dose level in one patient after three administrations and two patients after one administration**
  - Cohort 2 continues with subsequent dose administrations

- **Cohort 3 commenced in August 2025**

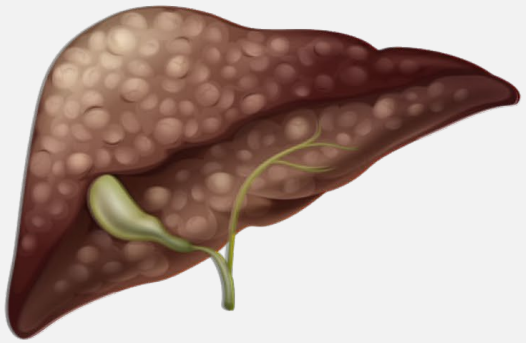
Risk of Liver Cancer, Complications, and Mortality Remain on Current SoC:

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

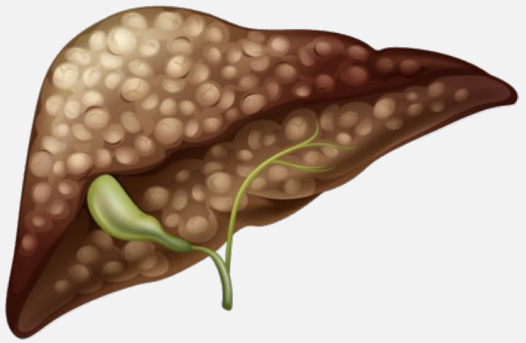
Up to **40% of patients** with chronic HBV infections may develop life-threatening complications including cirrhosis and/or HCC[1]



Cirrhosis



Liver Failure



Hepatocellular Carcinoma (HCC)

cHBV Results in

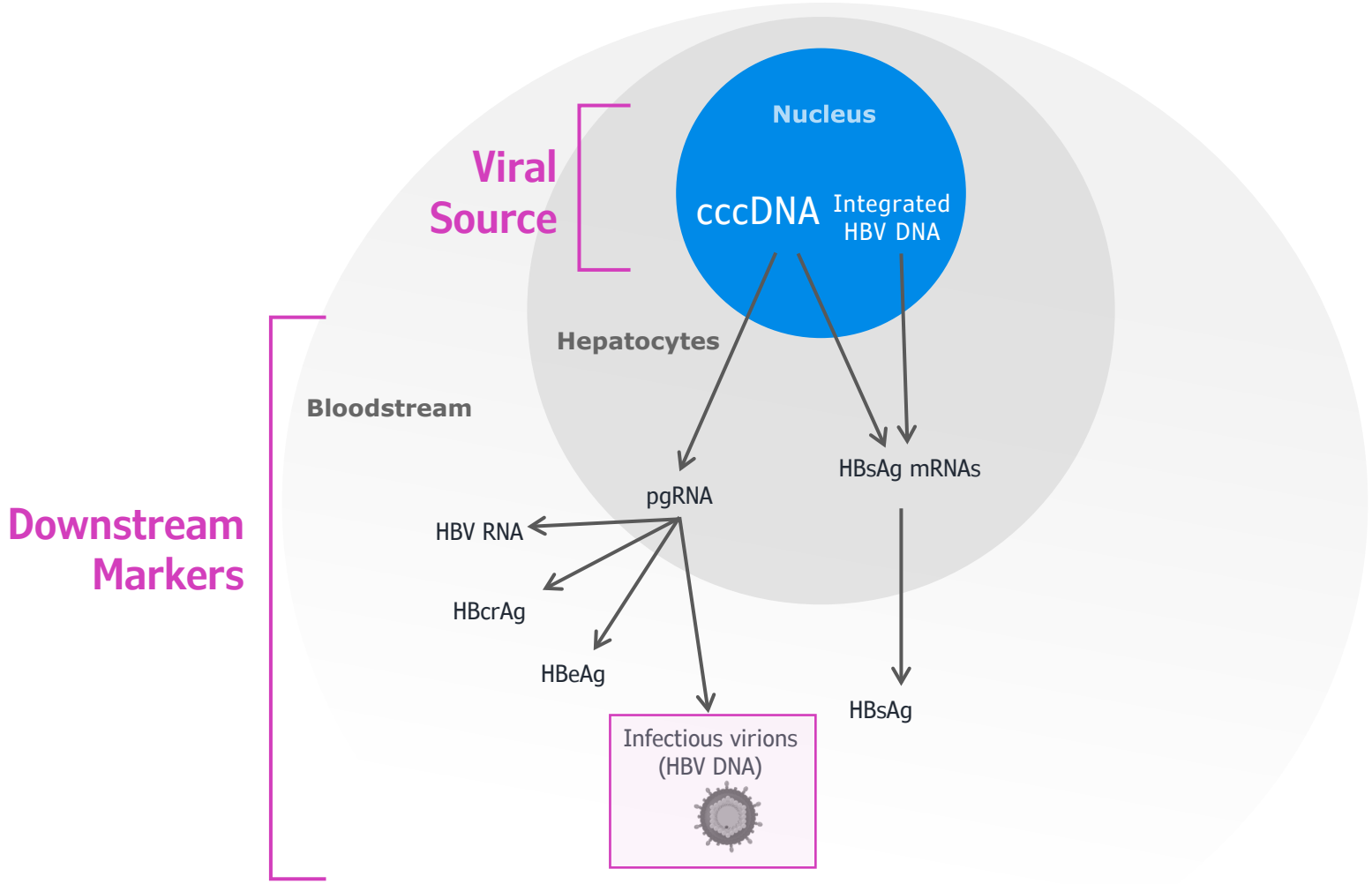
> 1 Million Global Deaths

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.

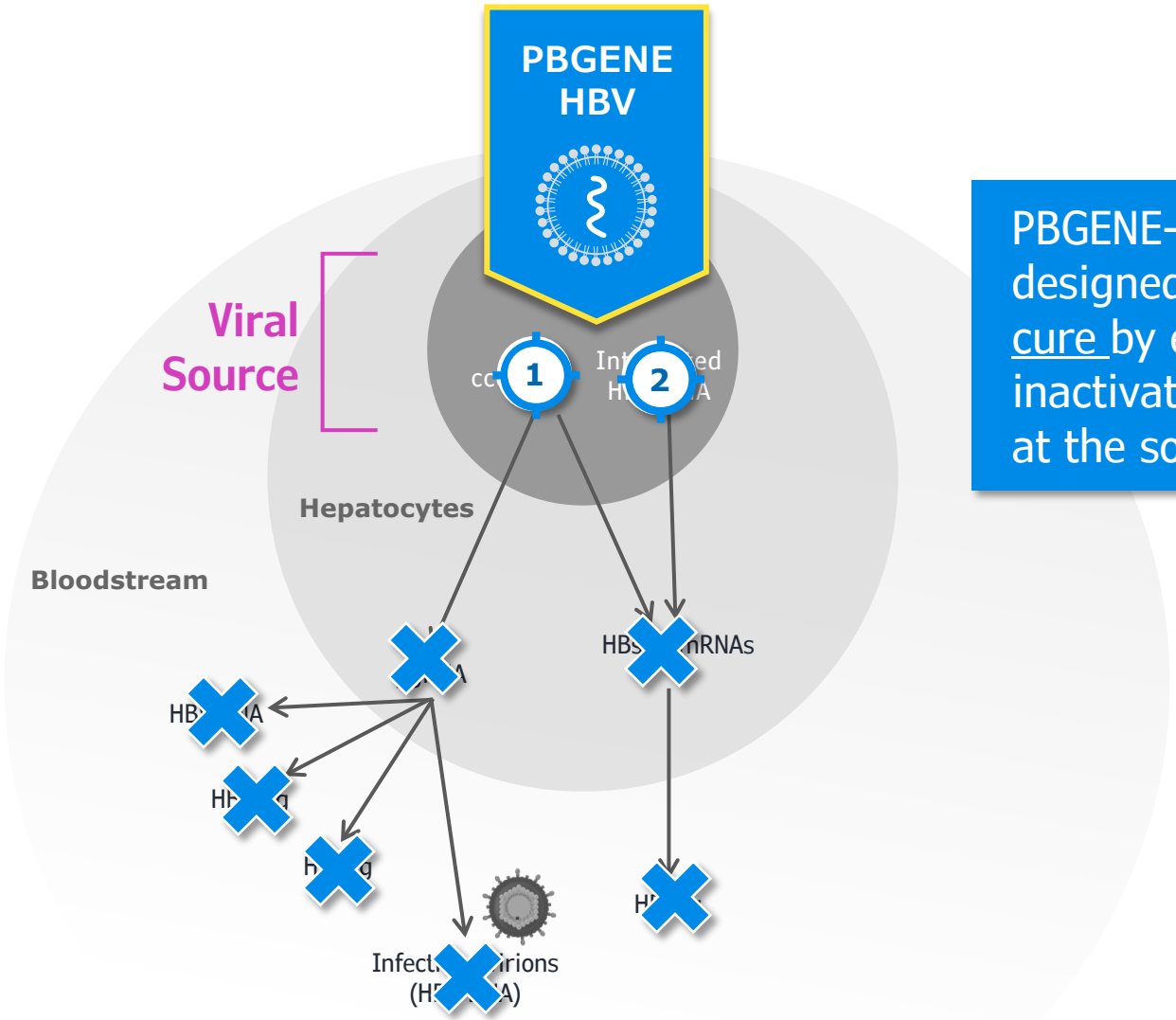
cccDNA and Integrated HBV DNA are the Viral Source of Downstream Markers



cccDNA, covalently closed circular DNA; DNA, deoxyribonucleic acid; HBeAg, hepatitis B e antigen; HBcrAg, hepatitis B core-related antigen; HBsAg, hepatitis B surface antigen; mRNA, messenger RNA; pgRNA, pregenomic RNA; RNA, ribonucleic acid.

HBV Cure Requires a Novel Approach Targeting The Viral Replication Source

Eliminating cccDNA Has a Clear Biologic Rationale For Cure



PBGENE-HBV is uniquely designed to seek a complete cure by eliminating cccDNA and inactivating integrated HBV DNA at the source of HBV

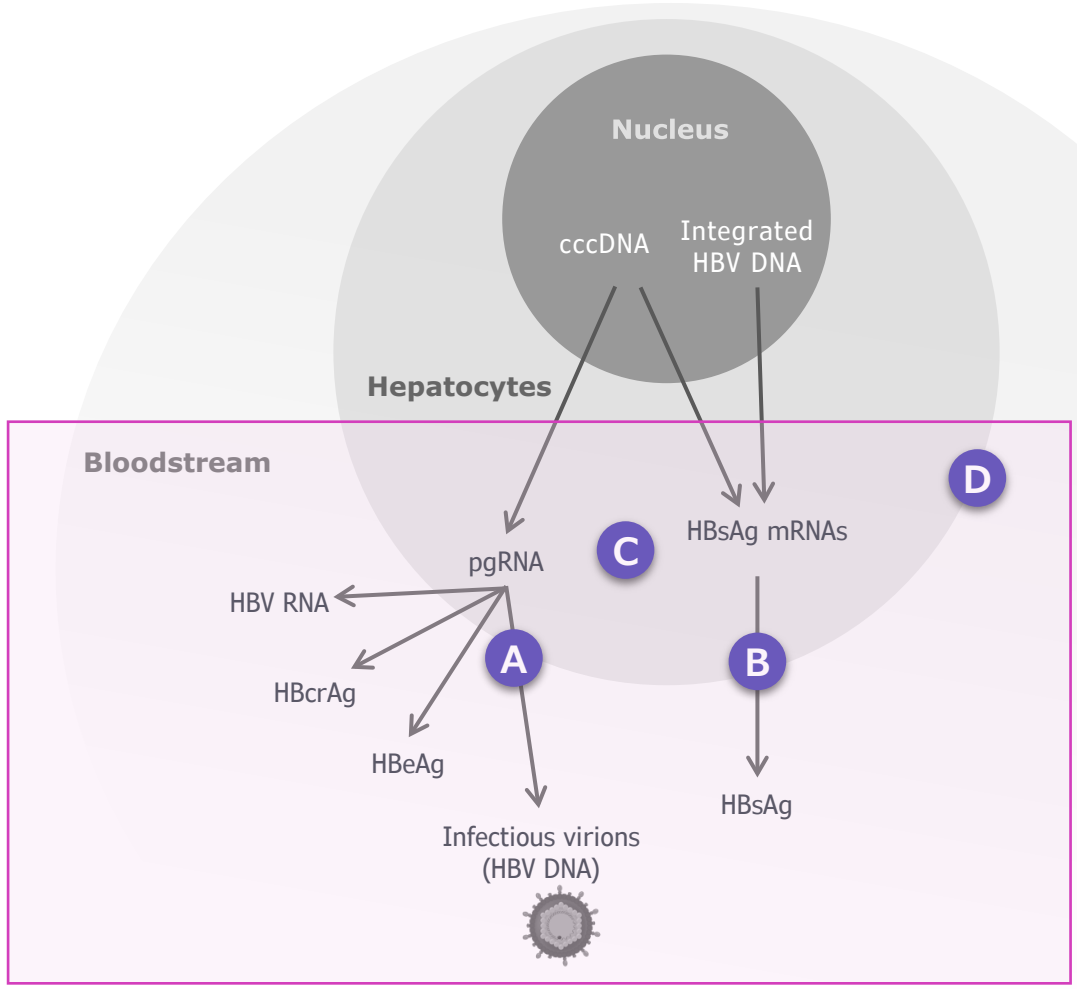
1 Eliminates cccDNA

2 Inactivates Integrated HBV DNA

Treatment at the Source of the Viral Pathway Results in Reductions of Downstream Markers

Current Therapeutic Strategies Rarely Achieve Functional Cure Because They Leave cccDNA Present

Current therapies in development target downstream components of the viral life cycle



Even combining antivirals and immune modulators, functional cure is rarely achieved and far from the ≥30% functional cure rate set by HBV experts

A Nucleos(t)ide Analogs Capsid Assembly Modulator

B Nucleic Acid Polymer Therapy

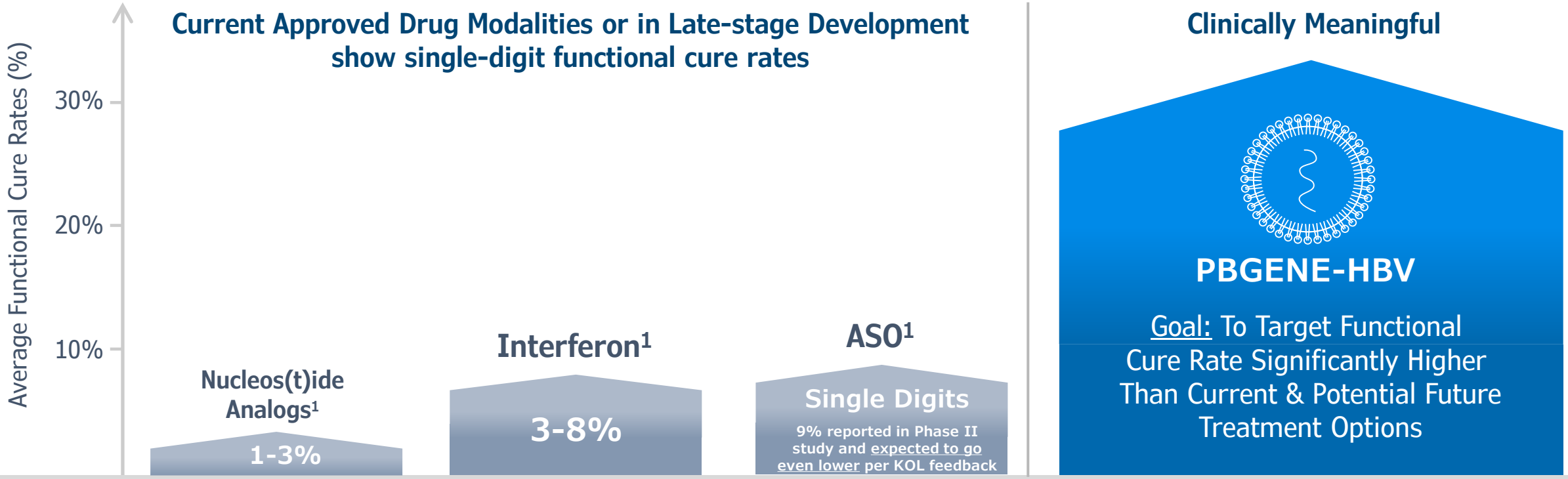
C Antisense Oligonucleotides siRNA

D Immune modulators

SiRNA, small interfering RNA.

≥30% Functional Cure Rate Set By HBV Experts Has Remained Elusive

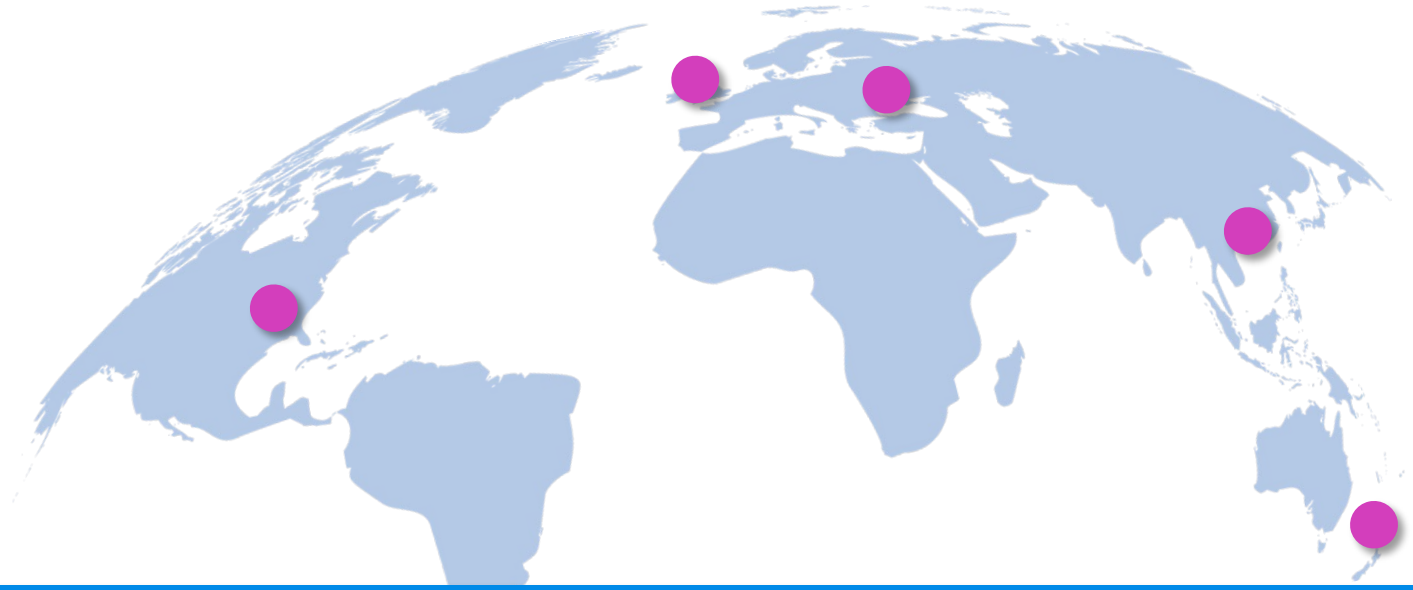
PBGENE-HBV has opportunity to be clinically meaningful by increasing functional cures



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

eliminateB



Global Phase 1/2a Study Across

5 Countries

Up to 45 Patients

Patient Population

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

ClinicalTrials.gov identifier NCT06680232.

Phase 1/2a Study Design

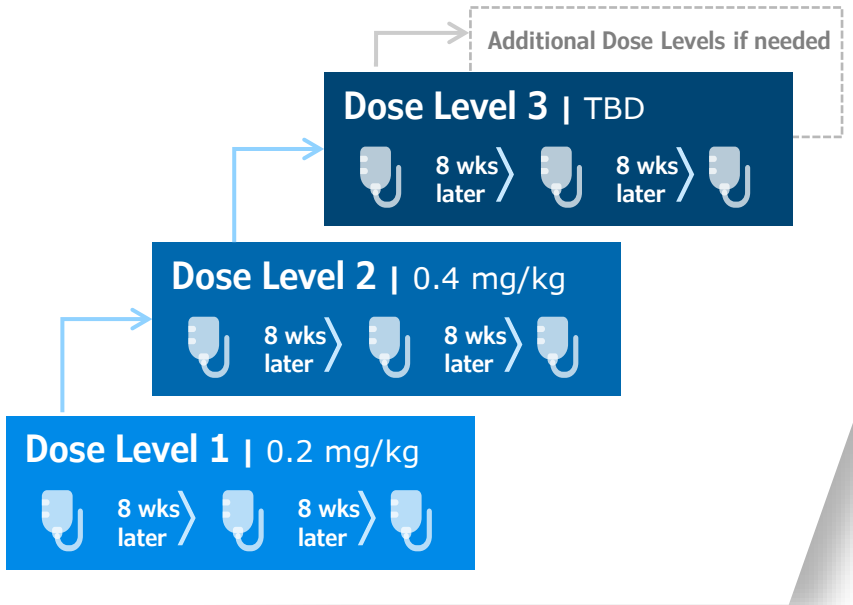
Potential for rapid evaluation of safety and efficacy of PBGENE-HBV in 2025

Part 1: Multiple Ascending Dose Escalation

Finite Treatment: Patient receives up to 3 dose administrations*

HBeAg negative patients on NUCs

Staggered Dose Levels



Safety & Efficacy Evaluation

Part 2: Dose Expansion

Advance optimized dose and schedule to eliminate cccDNA and drive complete 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 complete cure

*3 + 3 standard design with sentinel dosing of patients. Note- protocol permits dosing additional dose administrations beyond 3 in Part 2 of protocol.
mg/kg, milligram per kilogram; N, total number of patients; wks, weeks.
ClinicalTrials.gov identifier NCT06680232

Phase 1/2a 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

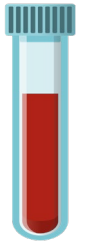
Monitoring Biomarkers

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.

Status - Completed Dosing Cohort 1, Cohort 2 is Partially Complete and Cohort 3 is Underway

DATA CUT:
We have now successfully enrolled 6 evaluable patients into cohort 1 (n=3) and cohort 2 (n=3) with full analysis

Safety

- In Cohort 1 at 0.2 mg/kg no adverse events >Grade 2, no serious adverse events, and no dose-limiting toxicities were observed
- In evaluable patients[1] from Cohort 2 at 0.4 mg/kg no adverse events >Grade 2, no serious adverse events, and no dose-limiting toxicities were observed
- Safety data supports continued exploration of higher dose levels and shorter dose interval
- Cohort 3 at higher dose level commenced in August 2025

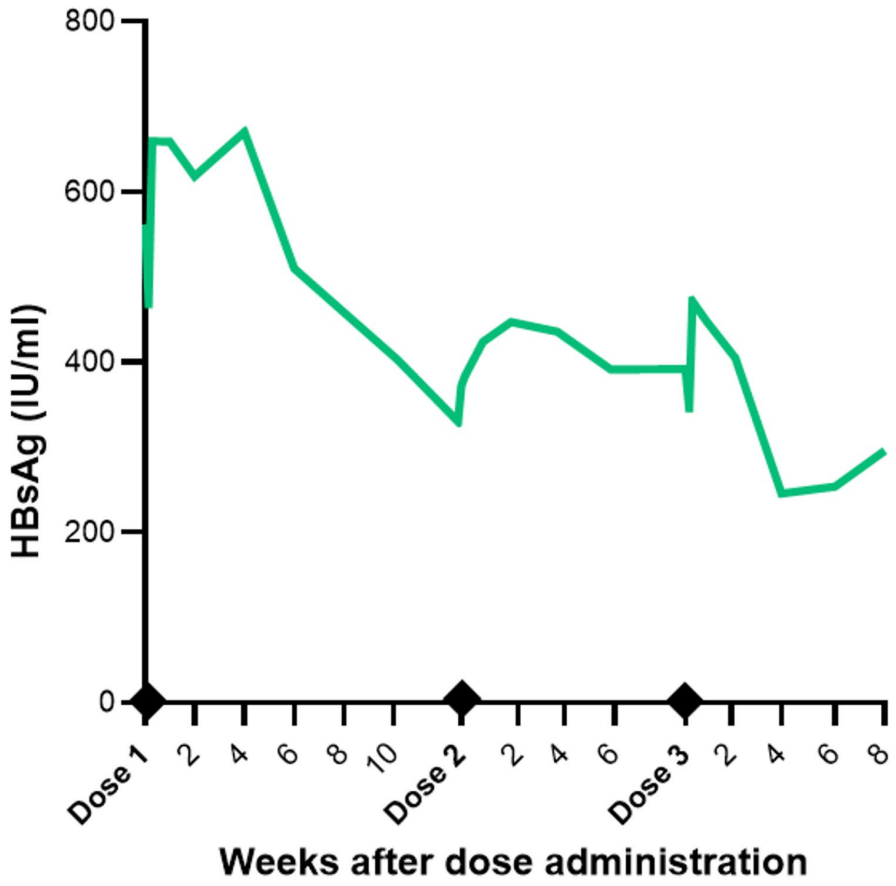
Efficacy

- Proof-of-activity established for PBGENE-HBV, the first and only clinical modality designed to eliminate covalently closed circular DNA (cccDNA) and inactivate integrated DNA, with the goal of complete cure
- PBGENE-HBV demonstrated substantial antiviral activity in all three patients in Cohort 1 at 0.2 mg/kg, with best responses achieving a 47-69% Hepatitis B surface antigen (HBsAg) reduction
  - Durable HBsAg reduction of approximately 50% in patient 1 is observed 7 months after initial dose administration

August 6, 2025 Company Press Release
n, number of patients.
1. Received three doses
ClinicalTrials.gov identifier NCT06680232

EFFICACY: Sustained HBsAg Reduction Seen in 1 of 3 Patients at Lowest Dose Level

Figure 1. Sustained HBsAg reduction in patient 1



+/- 20% natural fluctuations of HBsAg

PRECISION
BIOSCIENCES

PBGENE-DMD

Excision of a section of the Dystrophin Gene responsible for ~60% of the defects in Duchenne's Muscular Dystrophy (DMD)

PBGENE-DMD Opportunity:

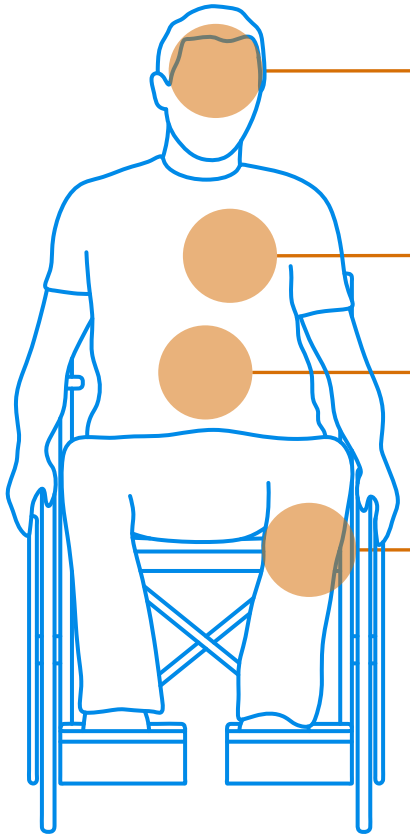
Potential to provide a first-in-class and best-in-class therapeutic for patients

High Unmet Need

Clear Regulatory Guidance

Growing Opportunity for Innovative Treatments

DMD is a Genetic Disorder Resulting in Progressive Muscle Degeneration and Early Death



Brain

- Learning disability or serious cognitive disability in a subset of patients

Heart

- Cardiomyopathy and arrythmias
- Eventual cardiac failure leading to premature death in late teens or 20s

Diaphragm & Intercostal Muscles

- Weakened respiratory muscles increase risk of infections
- Pulmonary insufficiency leading to premature death in late teens or 20s

Skeletal Muscle

- Progressive muscle weakness leading to loss of ambulation around 12

Content adapted from www.mda.org.

Today, Patients with DMD are in Dire Need with Limited Therapeutic Options

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

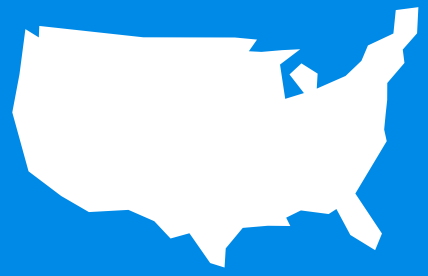
PBGENE-DMD Target Product profile, Exon Skipper and Microdystrophins based on data presentations and earnings release from related companies.

Global DMD Market Poised for Innovative Breakthroughs Like PBGENE-DMD

Prevalence & Incidence[1]

~300-400k
DMD Patients globally

~15k
DMD Patients in US

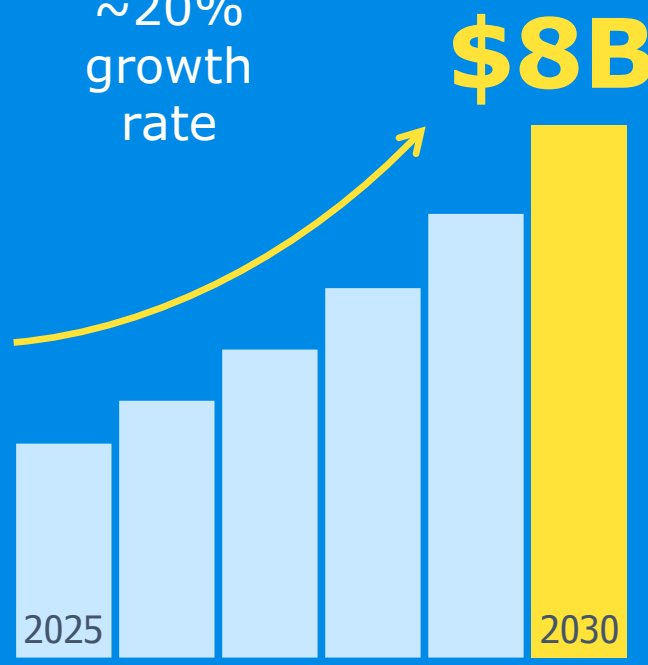


>20k births
per year globally

~550 births
per year in US

Global Market Size Projection[2]

~20% growth rate



1. Prevalence and Incidence based on CureDuchenne and Orphanet Journal or Rare Diseases; k =1,000.
2. Market Size based on estimates from Evaluate Pharma 2025.

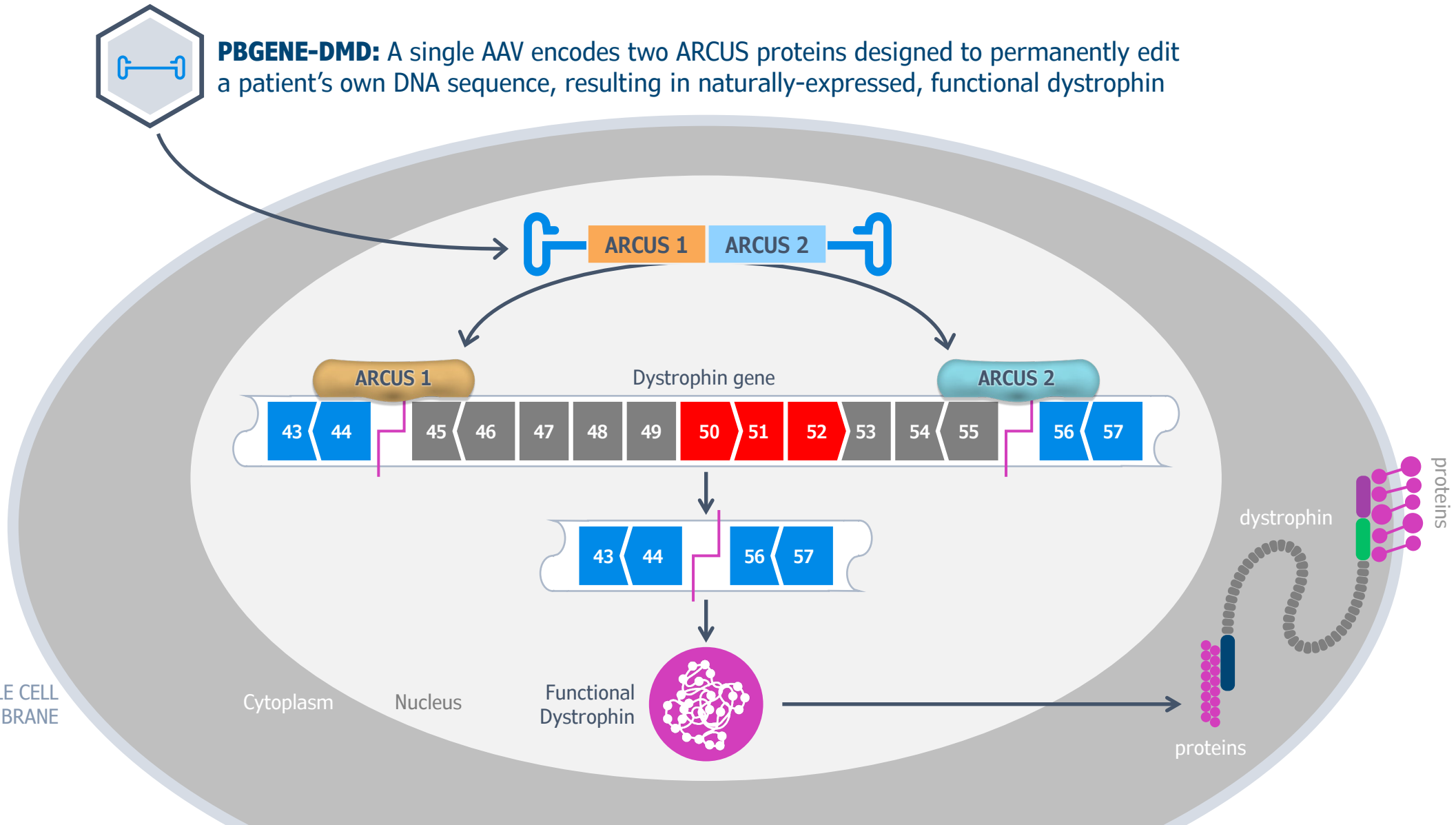
PBGENE-DMD is Designed to Provide Durable **Functional Muscle Improvement** for the Majority of Patients with DMD

Novel Mechanism Corrects Human Dystrophin Gene Resulting in a Functional Dystrophin Protein

Designed For Safety Through Lower Dose AAV

PBGENE-DMD Designed to Provide Durable Functional Improvement for Majority 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



ARCUS is Uniquely Positioned for DMD Gene Editing

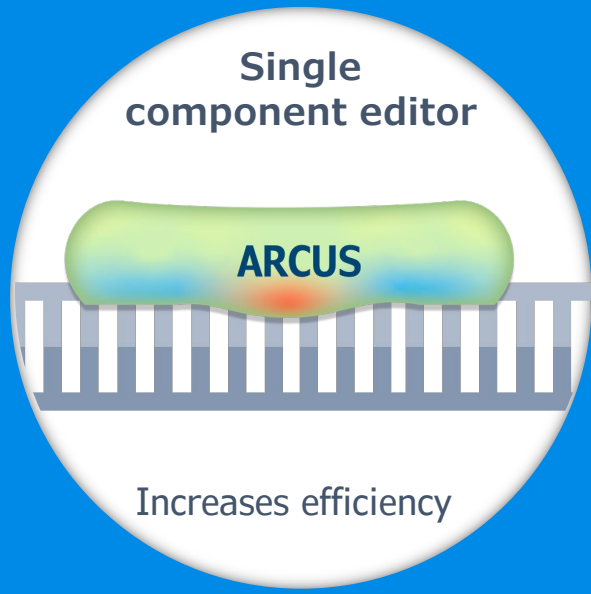
Why Other Gene Editors Cannot Follow

DMD: Potential Safety and Efficacy

Predictable repair enhances reliability of excision; Unique cut allows for more complete characterization of specificity

DMD: Potential Safety and Efficacy

Both nucleases are delivered to cells in a single AAV, enabling lower doses and higher efficiency



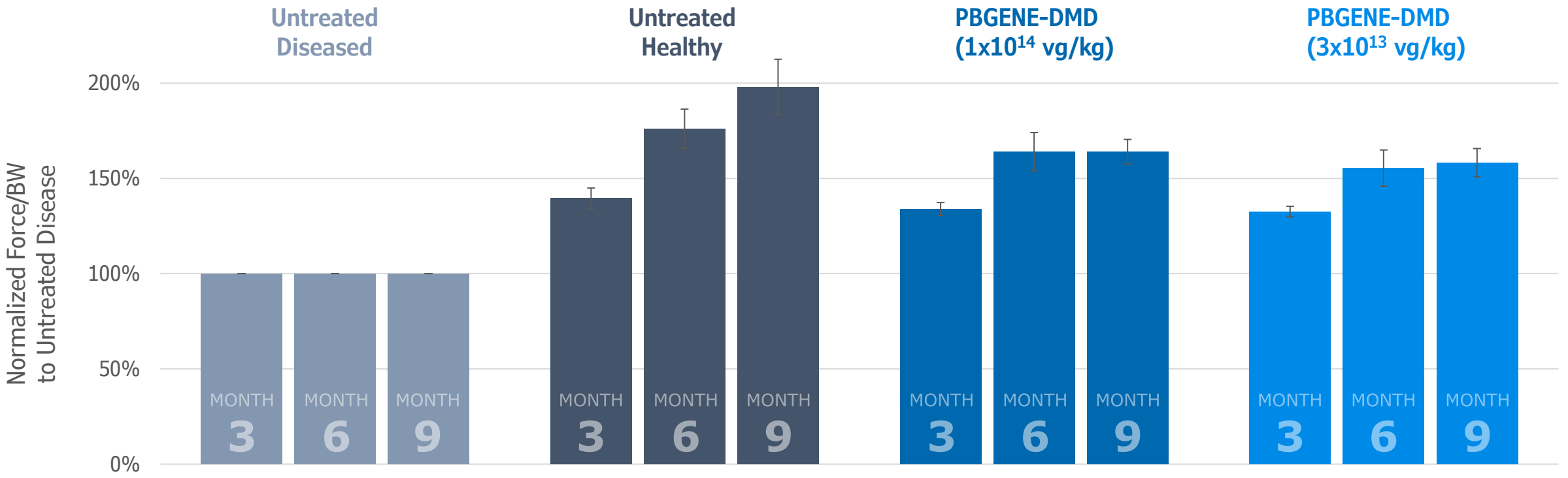
DMD: Potential Efficacy

Iterative protein engineering enabled co-evolution of two ARCUS nucleases for coordinated, efficient excision due to kinetic design

PBGENE-DMD Showed Durable Functional Improvement Over Time

New Data

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



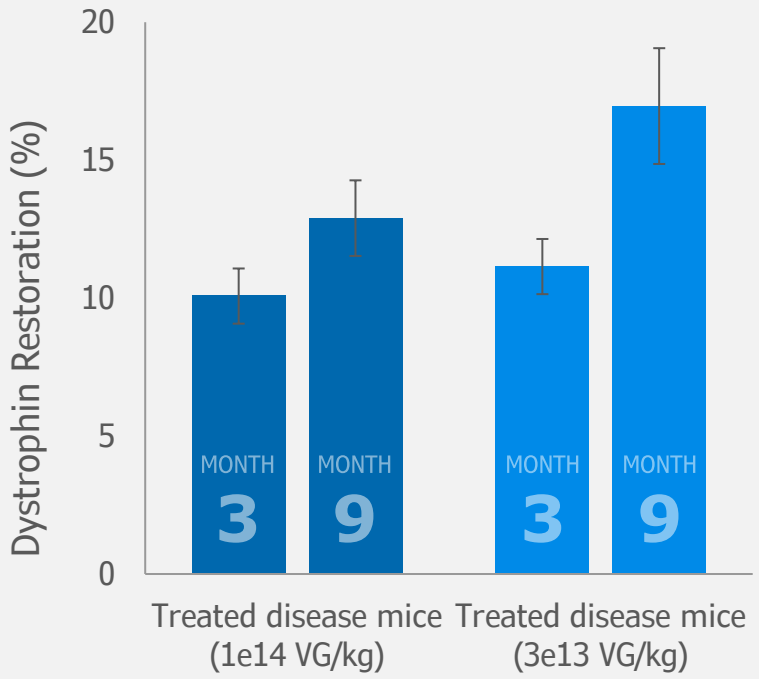
- Improved muscle function observed from 3 to 6 months.
- Durable functional improvements maintained out to 9 months.
- Benefit consistent across both experimental dose levels

Force was measured in the calf across multiple stimulation frequencies. Averaged force normalized to 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.

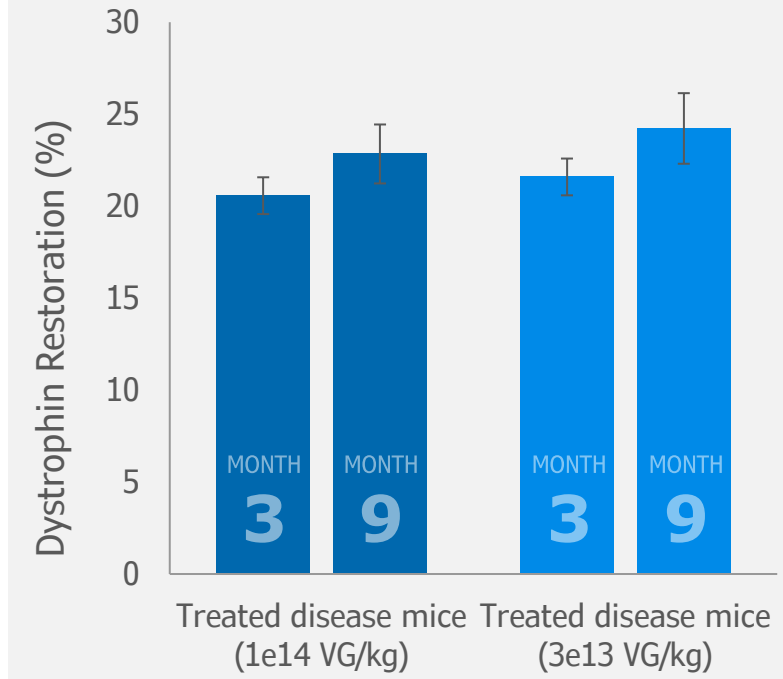
New Data

Long-term Functional Improvement Driven by Increasing and Stable Levels of Dystrophin Protein Expression

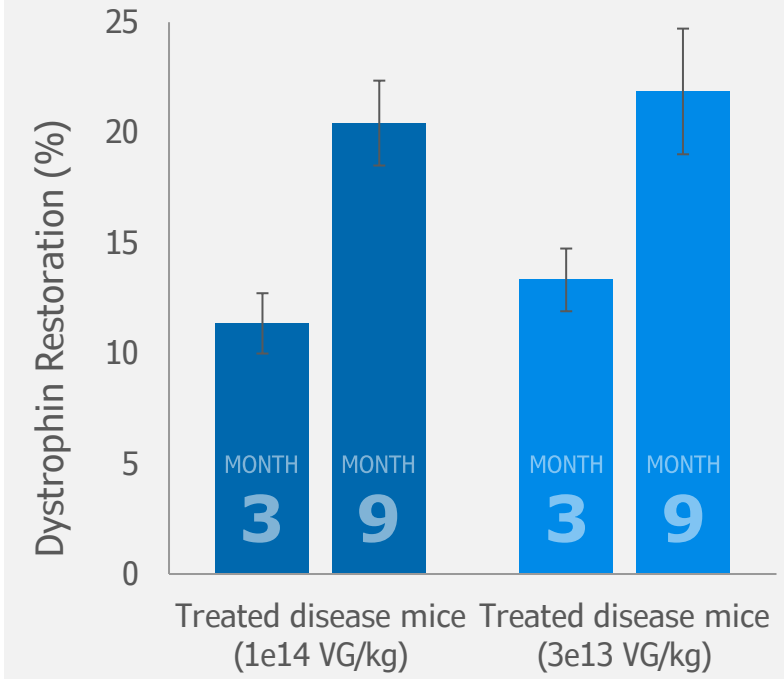
Heart



Calf



Quadricep

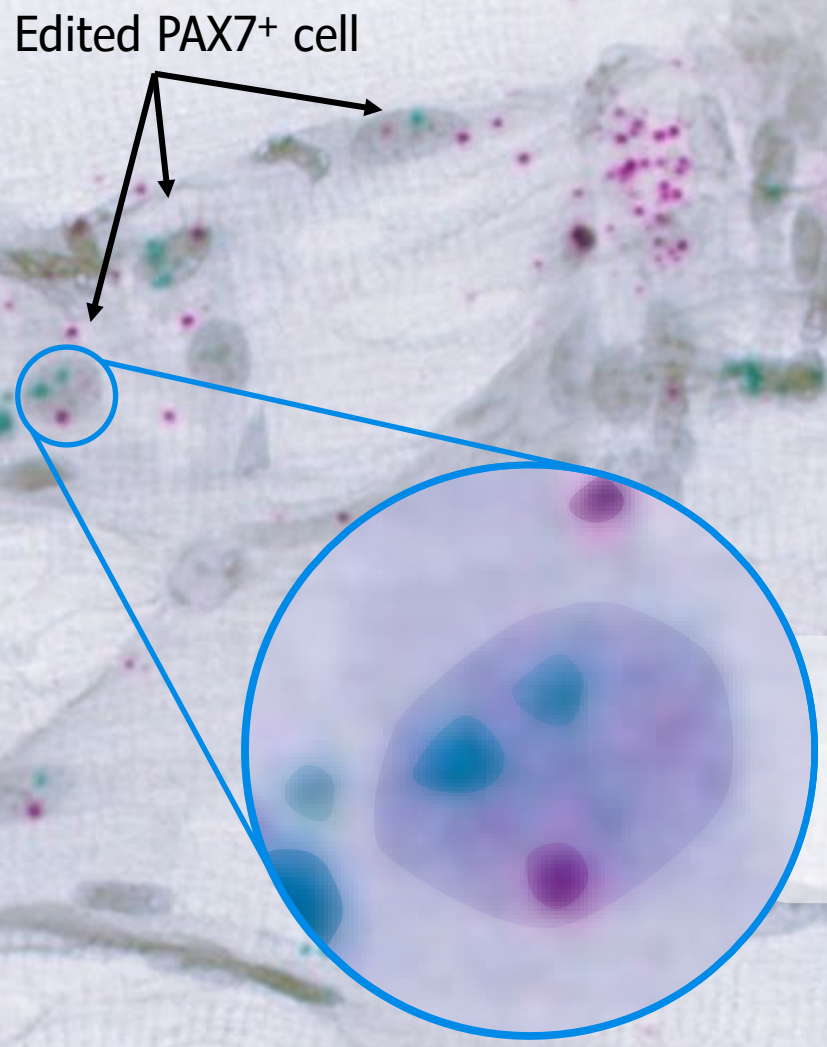


Naturally-produced, near full-length functional dystrophin protein increases through 9 months in mice

PBGENE-DMD Edited Muscle Satellite Stem Cells, Providing Potential for Durable Functional Improvement

PBGENE-DMD has demonstrated permanent editing of satellite cells, beyond transient transduction

Observed edited dystrophin mRNA in PAX7+ cells, **a marker for muscle satellite stem cells**

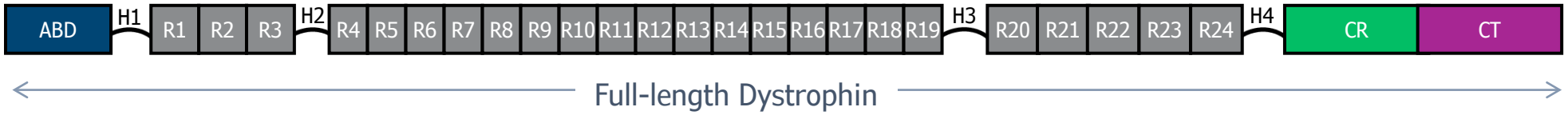


Pax7+
Edited DMD 44-56 mRNA

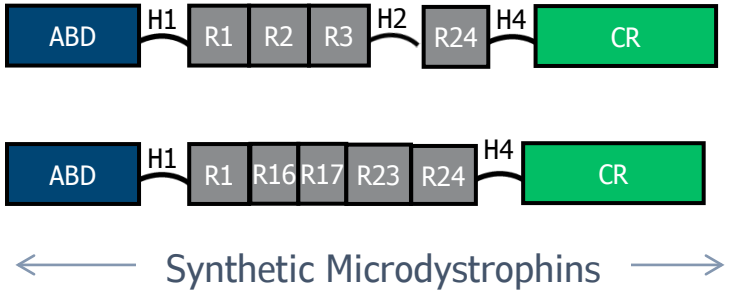
Novel Mechanism and Proven Dystrophin Protein

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

Full-length Dystrophin



Truncated Synthetic Microdystrophins

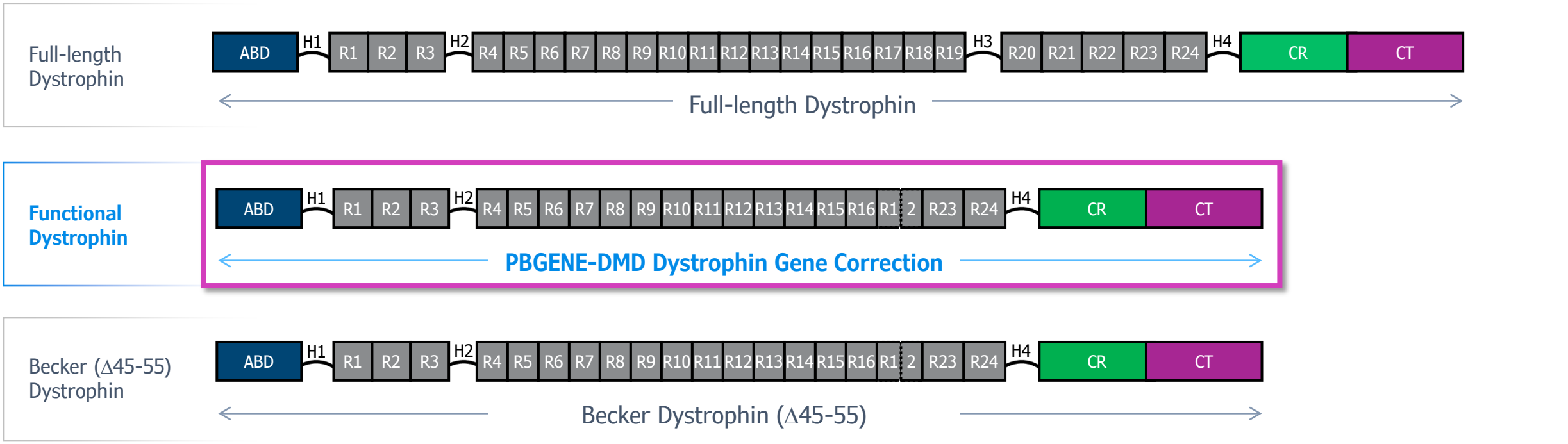


> The truncated dystrophin apparently does not function quite as well as the full-length gene, even when overexpressed…"
>
> — Phelps et al

Functional Dystrophin

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

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

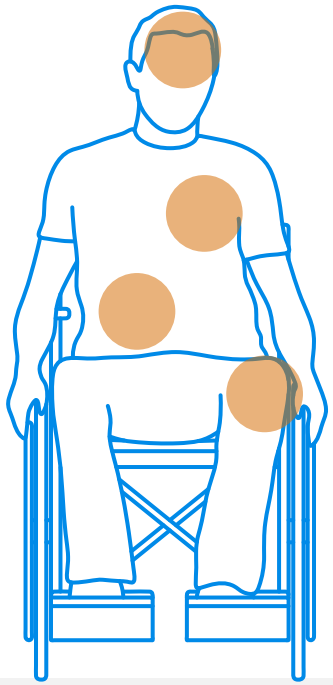


PBGENE-DMD functional dystrophin is present in a subset of Becker patients, who often have mild to asymptomatic phenotypes[1]

It is expected that as little as 5% expression of functional dystrophin protein is needed to provide therapeutic benefit in DMD patients[2]

1. Taglia et al. *Acta Myol*, 2015 May;34(1):9-13. 2. Feraudy et al. *Ann Neurol*, 2021 Feb;89(2):280-292.

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



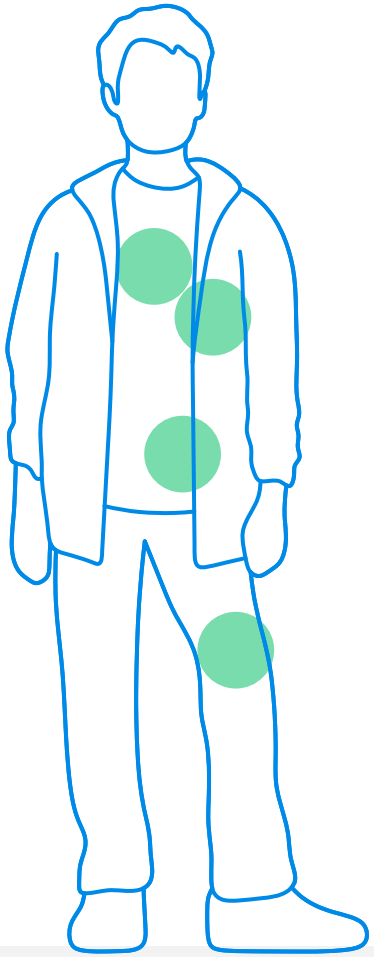
Lifespan:

- 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

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.

PBGENE-DMD Program is Quickly Moving Towards Clinical Stage

- [x] Identified Clinical Candidate
- [x] Received Positive Pre-IND Feedback From FDA
- [x] Initiated GLP Toxicology Studies
- [ ] Manufacture Clinical Trial Material
- [ ] Target Filing of CTA and/or IND in 2025
- [ ] Initiate First-in-Human Clinical Study

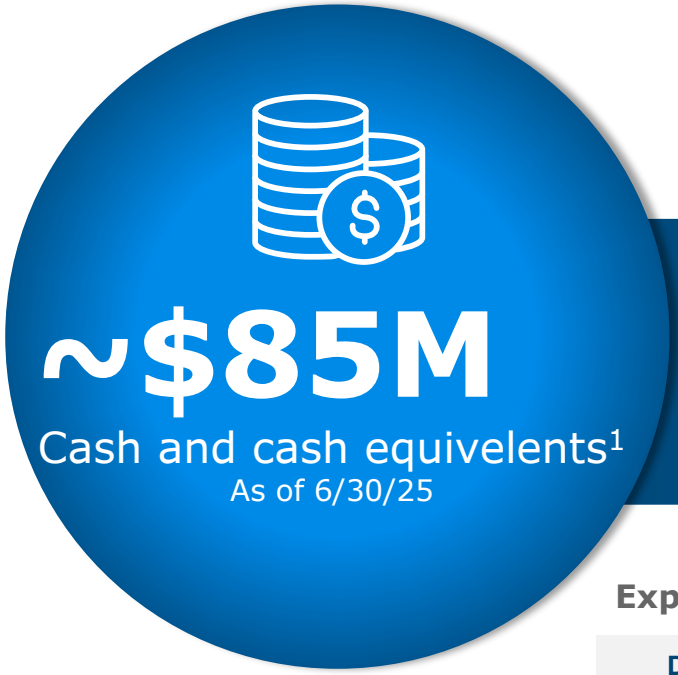
PBGENE-DMD: Highly Desirable Target Product Profile

Ideal therapy would have:	PBGENE-DMD Potential to Provide Best-In-Class Therapeutic Profile
Improvements in muscle function	✓ Nonclinical evidence of muscle function improvement over time
Long-term durable benefit	✓ Data demonstrating improvement is durable to at least 9 months in mice
Broadly applicable to patients	✓ Up to 60% of patients
Corrects human dystrophin gene resulting in a functional dystrophin protein	✓ Protein produced is known to have functional benefit in humans as evidenced by Del45-55 subset of Becker patients with favorable prognosis
Single administration	✓ One time administration
Good Safety	✓ PBGENE-DMD has been safe in nonclinical studies[1] to date
Ability to reach skeletal and cardiac muscles	✓ Robust protein expression across broad range of skeletal and cardiac muscles in mice

1. Diseased mouse model

Precision BioSciences Offering Meaningful Catalysts in Next 6-18 Months

Ample cash runway to fund regulatory submissions and clinical data readouts



CASH RUNWAY INTO 2H 2027

Potential to Advance 3 Programs to Phase I Clinical Data

Expected Inflection Points

PBGENE-HBV — Global phase 1/2a ELIMINATE-B trial on-going with initial data demonstrating safety and antiviral activity with HBsAg reductions; Data throughout 2025

ECUR-506 for OTCD — Complete clinical response in first patient dosed; OTC-HOPE Phase 1/2 trial expected to finish enrollment in Q4 2025 with data readout for all patients expected in 1Q 2026

PBGENE-DMD — IND and/or CTA Filing targeted for 2025; clinical data expected in 2026

[1]Source: Form 10-Q filed 5/15/25.