



*Precision BioSciences
Corporate Presentation*

January 2023



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. All statements contained in this herein and in any related presentation that do not relate to matters of historical fact should be considered forward-looking statements, including, without limitation, statements regarding our growth strategy, research advancement, clinical development and regulatory review of our product candidates, the expected timing of updates regarding our CAR T and in vivo gene editing programs and research, the expected timing of our communications with regulators, the expected advancement toward and timing of IND and CTA filings, expected efficacy and benefit of our platform, programs, product candidates, and manufacturing improvements and optimizations, the progress and success of collaborations with Lilly, Novartis, and other partners, including the receipt of any milestone, royalty, or other payments pursuant to and satisfaction of obligations under collaboration agreements, the goal of displacing autologous CAR T therapy, the goal of providing a one time, potentially curative treatment for genetic diseases, the goal of complex edits of the genome, the potential of “safe harbor” gene insertion, the ability of AAV and LNP delivery capabilities, the ability of our product candidates, if approved, to become best-in-class or first-in-class, expectations about our operational initiatives and business strategy, achieving key milestones and additional collaborations, and expectations regarding our liquidity and ability to fund operating expenses and capital expenditures requirements. In some cases, you can identify forward-looking statements by terms such as “aim,” “anticipate,” “approach,” “believe,” “contemplate,” “could,” “estimate,” “expect,” “goal,” “intend,” “look,” “may,” “mission,” “plan,” “possible,” “potential,” “predict,” “project,” “should,” “target,” “will,” “would,” or the negative thereof and similar words and expressions.

Forward-looking statements are based on management’s current expectations, beliefs and assumptions and on information currently available to us. Such statements are subject to 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 and requirements under our current debt instruments and effects of restrictions thereunder; risks associated with raising additional capital; 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; pending and potential 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’ 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; our or our collaborators’ 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; our ability to obtain an adequate supply of T cells from qualified donors; our ability to achieve our anticipated operating efficiencies at our manufacturing facility; 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 COVID-19 pandemic and variants thereof, or any pandemic, epidemic or outbreak of an infectious disease; insurance expenses and exposure to uninsured liabilities; effects of tax rules; risks related to ownership of our common stock 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, 2022, 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.

The 2022 financial results included in presentation are unaudited and preliminary, and this presentation does not present all information necessary for an understanding of the Company’s financial condition as of December 31, 2022 and its results of operations for the three months and year ended December 31, 2022. The Company’s actual results may differ from the preliminary estimates above due to the completion of the Company’s year-end accounting procedures, including execution of the Company’s internal control over financial reporting, and audit of the Company’s financial statements for the year ended December 31, 2022 by the Company’s independent registered public accounting firm, which are ongoing.

Precision's Growth Strategy:

Leverage the ARCUS gene editing platform in oncology and genetic diseases

1

Advance a first-in-class and a best-in-class allogeneic CAR T treatment for hematologic cancer

- Lead anti-CD19 allogeneic candidate, azercabtagene zapreleucel (azer-cel) potential first-in-class for R/R NHL* patients who relapsed post auto CAR T
- Next-generation anti-CD19 candidate, PBCAR19B with the goal of displacing auto CAR T in 2nd /3rd line R/R NHL* patients

2

Unlock full potential of ARCUS *in vivo* gene editing platform

- **Differentiate ARCUS** on safety, gene insertion and complex edits
- **Advance first *in vivo* gene editing programs to the clinic** to address serious genetic diseases and chronic hepatitis B

3

Secure selective premium *in vivo* gene editing collaborations

- Unlock additional development opportunities, reach more patients and provide capital for advancing wholly owned programs

Building an end-to-end gene editing company, spanning research through commercialization

Precision BioSciences: A Clinical Stage Gene Editing Company Built on Wholly Owned ARCUS Genome Editing Platform

ARCUS® Platform for therapeutic drug development

Diversified Pipeline



Ex Vivo:

Allogeneic CAR T immunotherapies

- Single-gene edit, donor-derived, single-dose



In Vivo:

Gene editing for complex genetic diseases

- Potentially curative, one-time treatment



Pioneers in genome editing technology development



Scalable in-house manufacturing capabilities



ARCUS platform and nucleases are unencumbered by third-party IP; control of over 100 issued patents related to ARCUS and its applications



Strong balance sheet provides 2+ years of runway¹

Agenda

- › 2022 Accomplishments
- › Allogeneic CAR T Program Update
- › Differentiating the ARCUS Platform
- › ARCUS *In Vivo* Gene Editing Program Highlights
- › 2023 Priorities & Milestones

2022 Accomplishments

- ✓ Presented **compelling clinical allogeneic CAR T data to date; 100% ORR 73% CR¹**
- ✓ Secured **\$75M for *in vivo* gene editing collaboration with Novartis** for hemoglobinopathies
- ✓ Raised **\$50M in equity financing** from new and existing investors
- ✓ Extended the **cash runway**; Runway greater than 2 years²
- ✓ Optimized **CAR T manufacturing process** designed to improve attributes for safety and efficacy
- ✓ Published first **NHP proof-of-concept data supporting HBV *in vivo* gene editing program**
- ✓ Progressed **Lilly and Novartis preclinical *in vivo* gene editing programs**, including DMD and SCD
- ✓ Received favorable **Type C feedback from FDA** regarding CMC plan for azer-cel

¹ Results announced on June 8, 2022 (n=12) were as of May 31, 2022

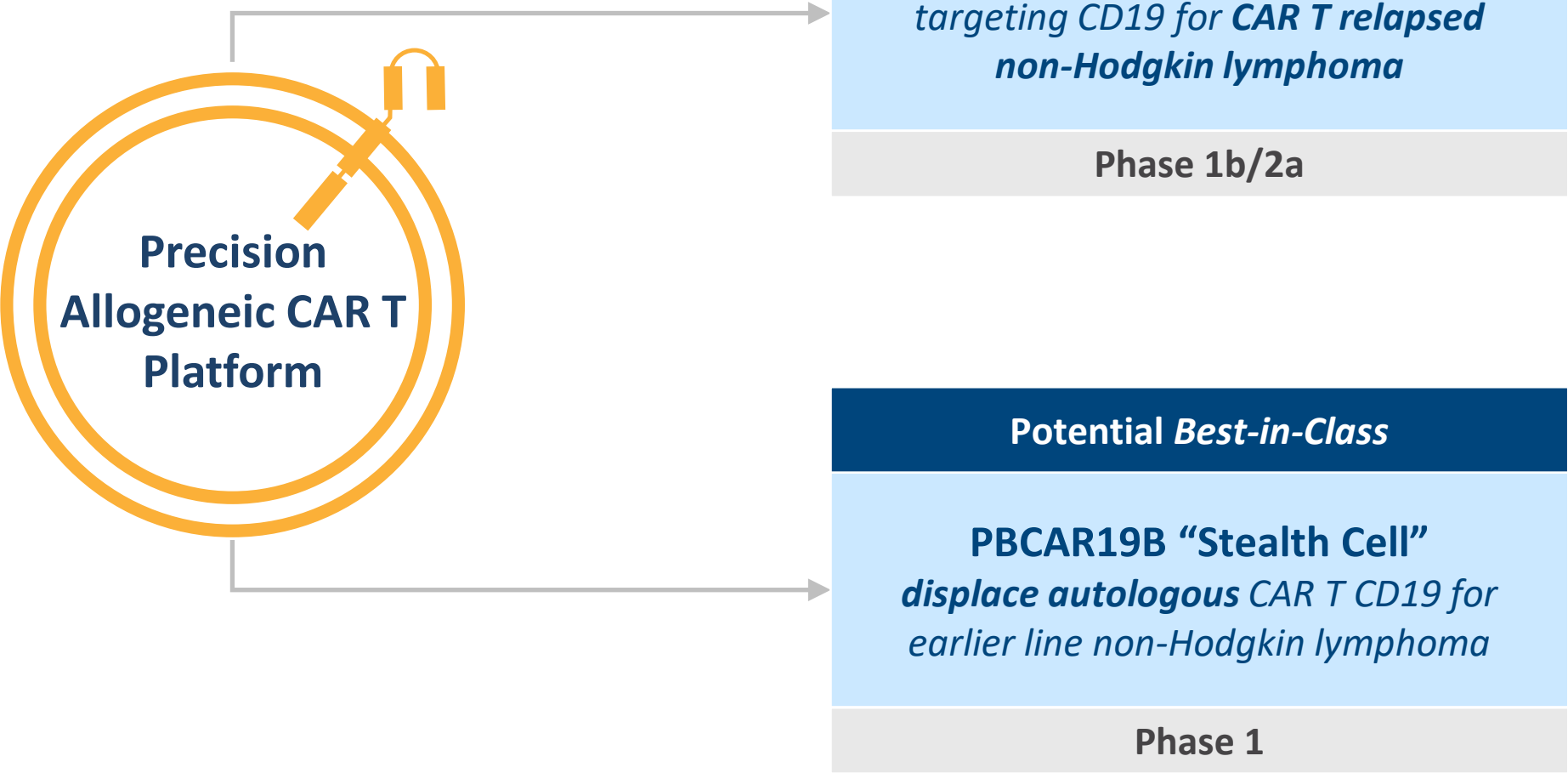
² As of December 31, 2022, Precision had approximately \$190 million in cash and cash equivalents. The Company expects that existing cash and cash equivalents, expected operational receipts, and available credit will be sufficient to fund its operating expenses and capital expenditure requirements into 2025.

Allogeneic CAR T Programs

Potential first-in-class and best-in-class approaches



Ex Vivo Allogeneic CAR T Pipeline Focused on Potential First-in-Class and Best-in-Class Approaches



Allogeneic CAR T Program Executive Summary

- 1** In autologous CAR T relapsed patients, azer-cel has shown high response rates¹, with **evidence of durability** extending greater than 18 months; **including peak CAR T levels in the same range as autologous CAR T**
- 2** **Manufacturing optimization resulted in improved product attributes** supporting reduced lymphodepletion dose in combination with azer-cel
- 3** **Favorable Type C feedback from FDA** on chemistry, manufacturing, and controls strategies support ongoing late-stage development for azer-cel
- 4** **Will proceed with request for FDA clinical meeting** if promising activity and safety observed in next azer-cel cohort
- 5** **Recruiting additional patients for PBCAR19B** in earlier line R/R DLBCL to complete Phase 1

Azer-cel: CAR T Relapsed Market for DLBCL Expected to Grow ~4-5x by 2025

Driven by auto CAR T becoming 2nd line standard-of-care (SoC)

Ex Vivo

Azer-cel

CAR T Relapsed Eligible Patients in US

2022 | 3rd line+ DLBCL patients



Total available 3rd Line + CAR T patient pool: ~5,000² (excludes clinical trials)

Auto CAR T becomes 2nd line SoC
ZUMA-7

2025 | 2nd line DLBCL patients



Total:
~14,000-15,000

By 2025, the auto CAR T relapsed population is expected to be 1.5x larger than the entire 3rd line+ auto CAR T market opportunity today

Auto CAR T relapsed patients

¹ LEK Research (HCP interviews, Evaluate Pharma, DRG), Internal assumptions

² <https://doi.org/10.6004/jnccn.2020.77422> CancerMPact, ZUMA-1 and ZUMA-7 Clinical Studies

Note: ZUMA-1 and ZUMA-7 relapse rate estimated off 2-year EFS. Assumes higher drug treatment rate of 90% in 2025 from the advancement of ZUMA-7 into 2nd line setting offering potential curative outcomes/new standard-of-care

Azer-cel: Target Product Profile (TPP) for CAR T Relapsed Patient Population

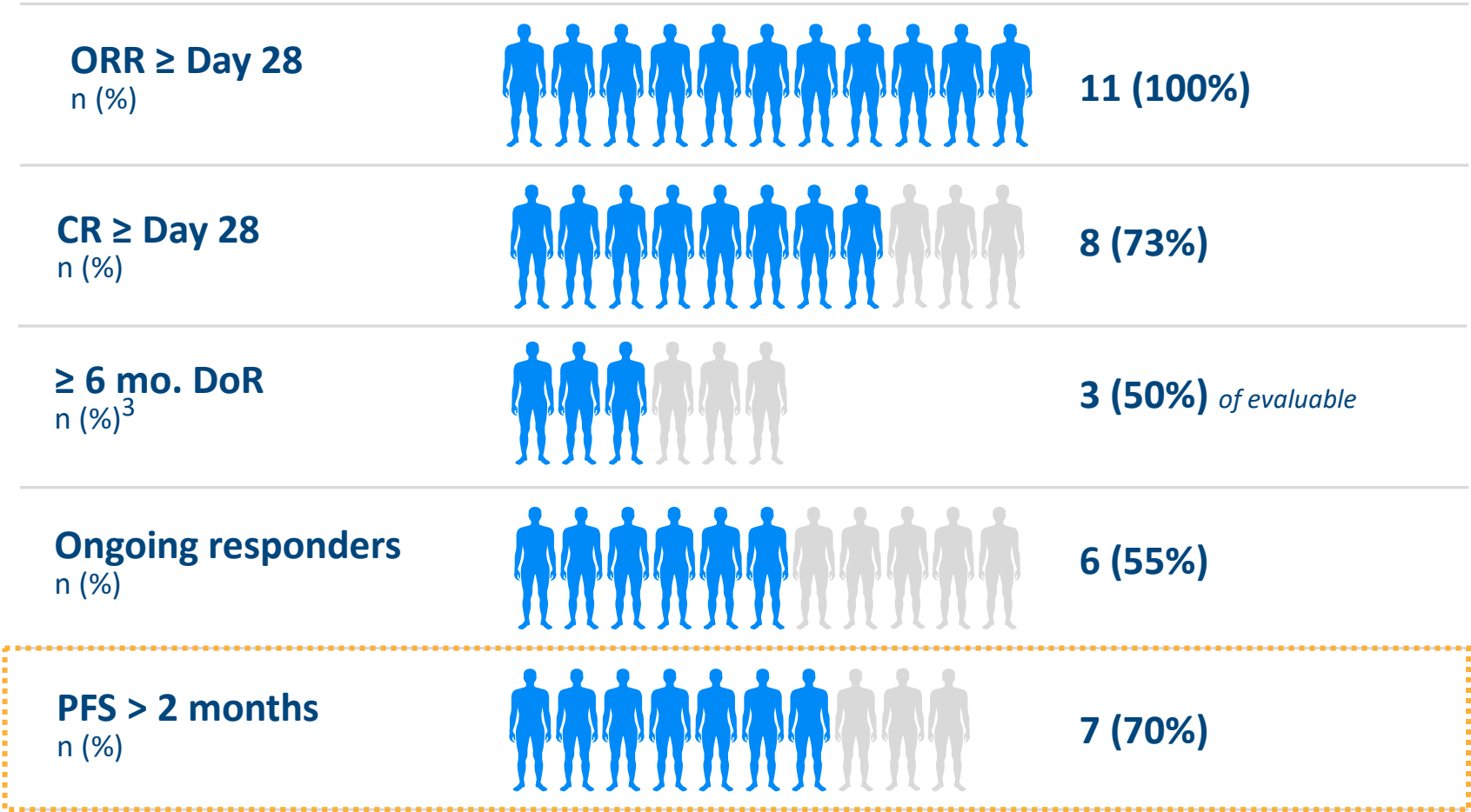
Ex Vivo

Azer-cel

	CAR T relapse	3 rd line NHL
Progression Free Survival (mPFS)	> 3 months	> 6-7 months
Duration of Response (DoR)	> 50% @ 3 months	~35% CR at 6 months; ~32% CR at 1 year plus
Overall Response Rate (ORR)	> 50%	> 70% at 28 days
Overall Survival (mOS)	> 6 months	26 months ³
Safety	Highest risk salvage population	Same or better than auto CAR T
Potential Regulatory Path	Single-arm study with historical control (to be discussed with FDA)	Head-to-head vs. auto CAR T/auto transplant

Azer-cel: Efficacy Results¹: *100% ORR, 73% CR with 50% durable responses at > 6 months*

CAR T relapsed
median 5+ prior lines
(n = 11 evaluable)^{2,3}



Clinicaltrials.gov identifier: NCT03666000

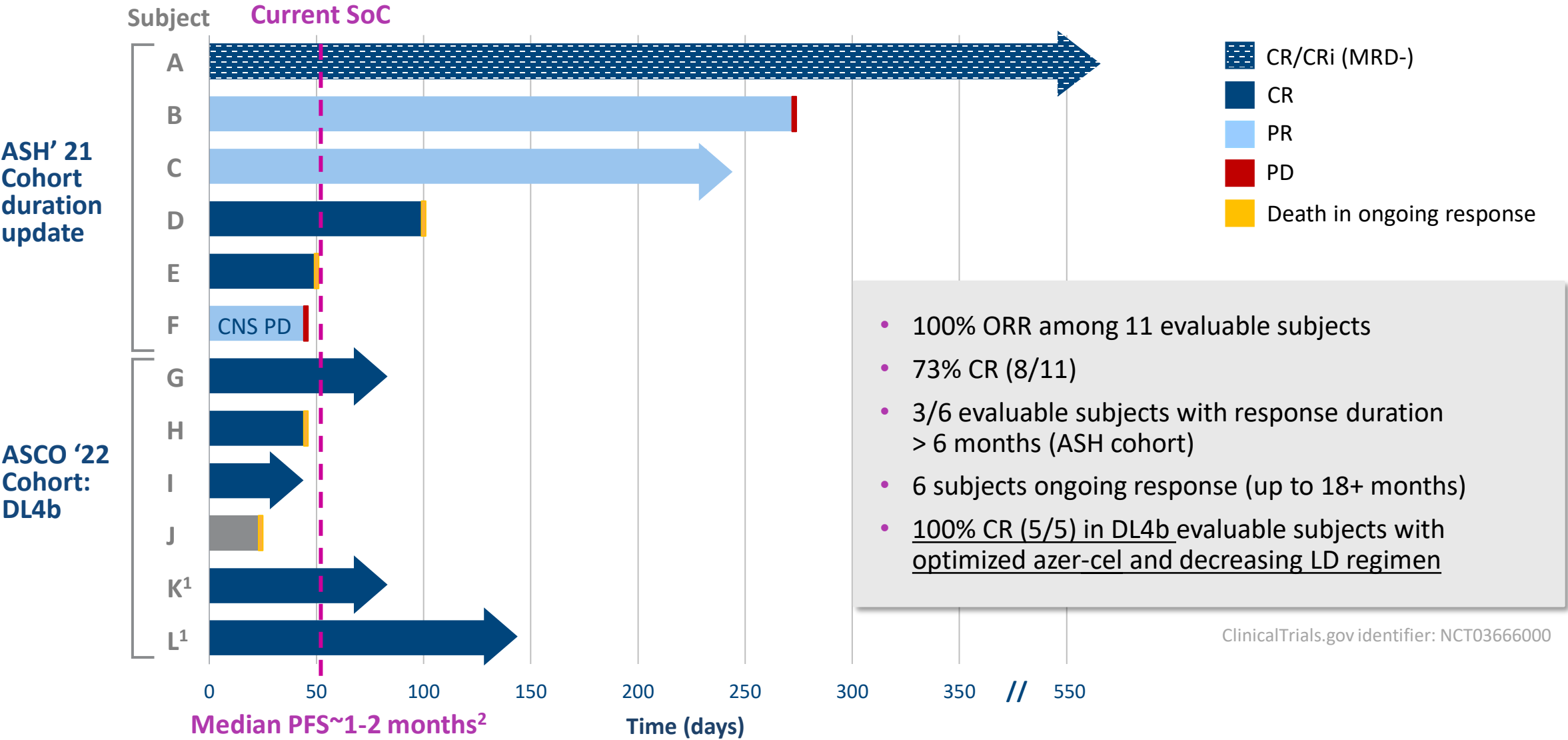
Potential Regulatory Hurdle

¹ Interim efficacy results as of May 31, 2022. Data presented at American Society of Clinical Oncology 2022 Annual Meeting

² One subject non-evaluable for efficacy at Day 28 assessment due to death from suspected fludarabine associated neurotoxicity at Day 23; patient had complete resolution on PET/CT scan at Day 21

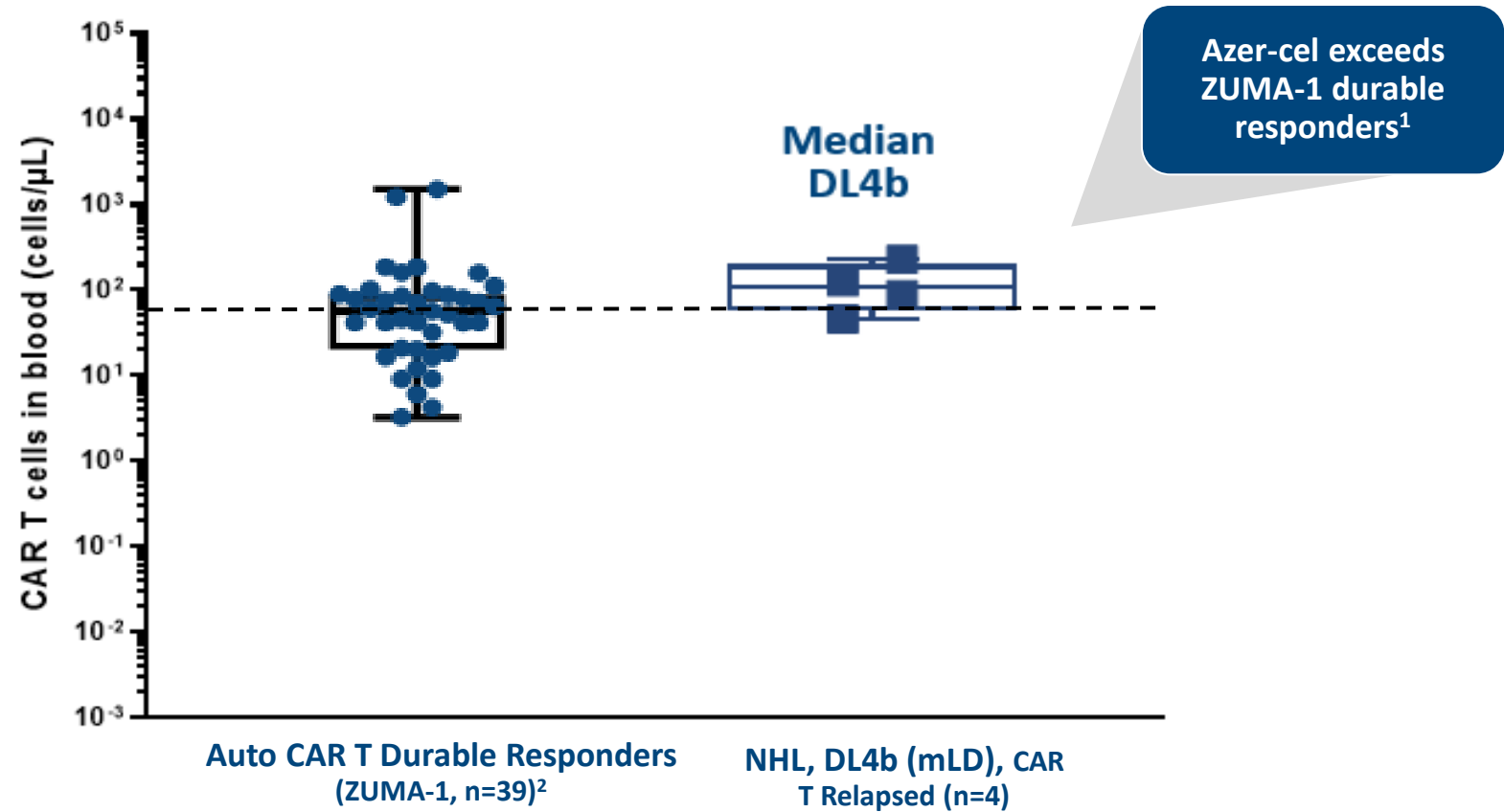
³ 4 of 6 (67%) evaluable patients have achieved remission inversions when comparing against prior therapy received

Azer-cel: Response Rates and Durability Exceed Current Standard-of-Care (SoC) for CAR T Relapsed Subjects



1 Subject K and subject L relapsed after allo CAR T, initially dosed at dose level 3. Original response: K – CR for 178 days before PD; L – PR for 28 days before PD
2. US CAR T Consortium Study - <https://pubmed.ncbi.nlm.nih.gov/33156925/>

Azer-cel: Peak Expansion Equivalent to Auto CAR T Levels in Long Term Durable Responders from ZUMA-1



★ Azer-cel program is first allogeneic CAR T to show peak expansion equivalent to auto CAR T with single dose

¹ For illustrative purposes only - not a head-to-head comparison. Differences exist between trial designs and subject characteristics, and caution should be exercised when comparing data across studies. Both ZUMA-1 and PBCAR01091 clinical study analyzed utilizing flow cytometry.

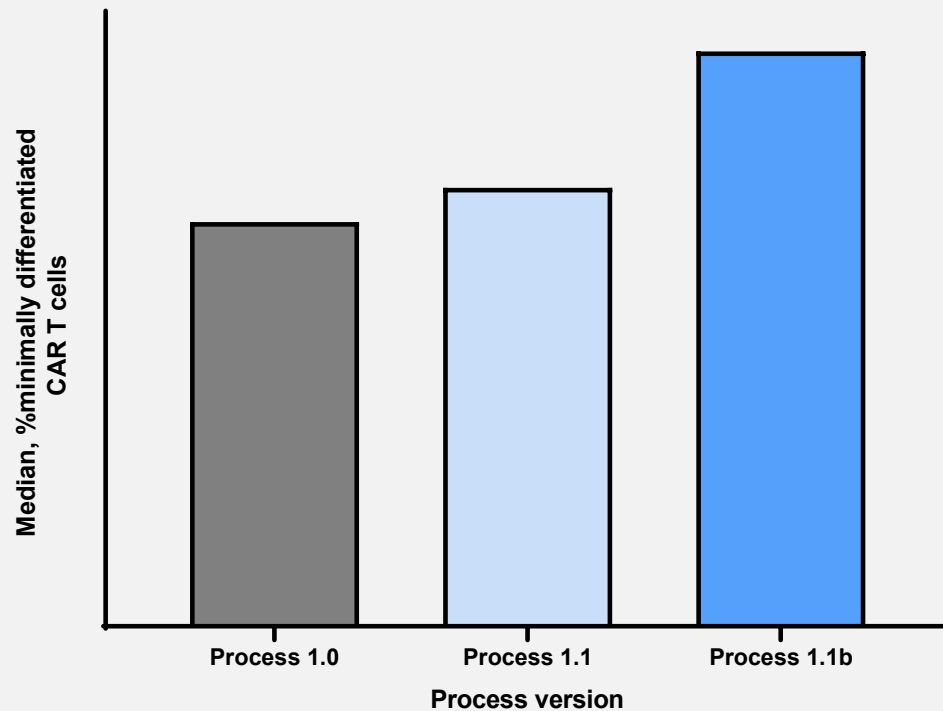
² Locke, et al. 2020

Improvements to Manufacturing Platform Enhanced CAR T Attributes Associated with Efficacy and Safety

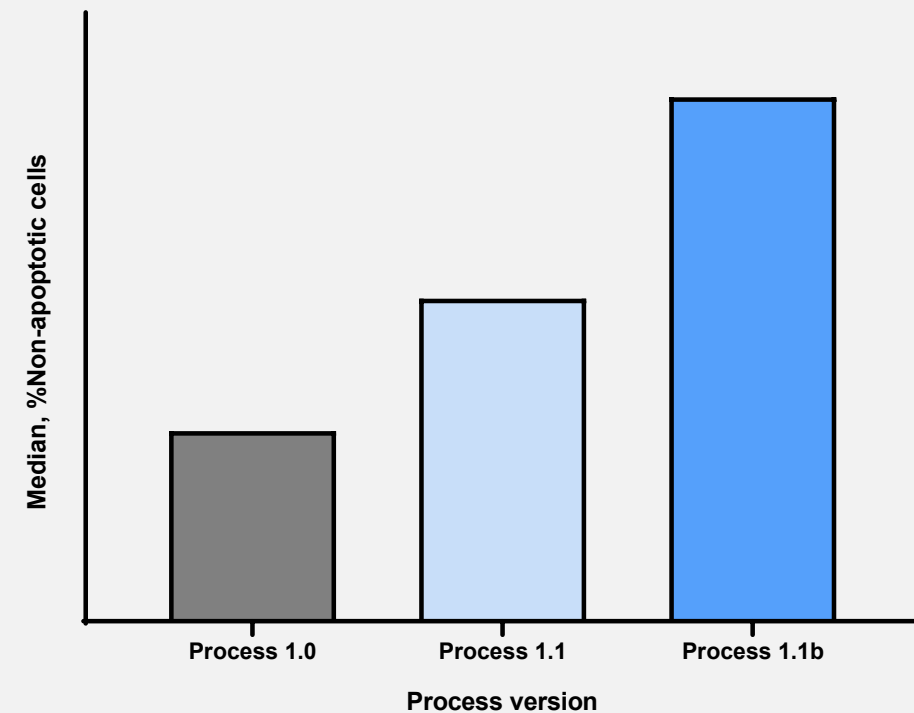
Improved manufacturing process resulted in:

- Less cell differentiation, correlated with efficacy
- Improved cell health by >175% and desired memory T cell subtype, correlated with efficacy
- Tighter CD4:CD8 ratio, potentially important for safety

%Minimally differentiated CAR T cells



%Non-apoptotic CAR T cells

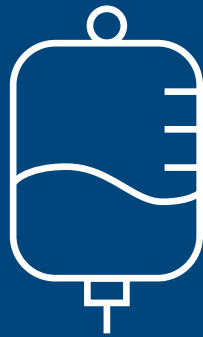


Optimize Therapeutic Index: Maximize Safety While Maintaining Efficacy

Profile Remains Key Next Step

Ex Vivo

Azer-cel



Reduce total fludarabine exposure during lymphodepletion

4
days



3
days

Fludarabine dose of
30 mg/m² per day

★ Given CAR T relapsed patient population is extremely fragile and already have received lymphodepletion, we have reduced the total fludarabine exposure

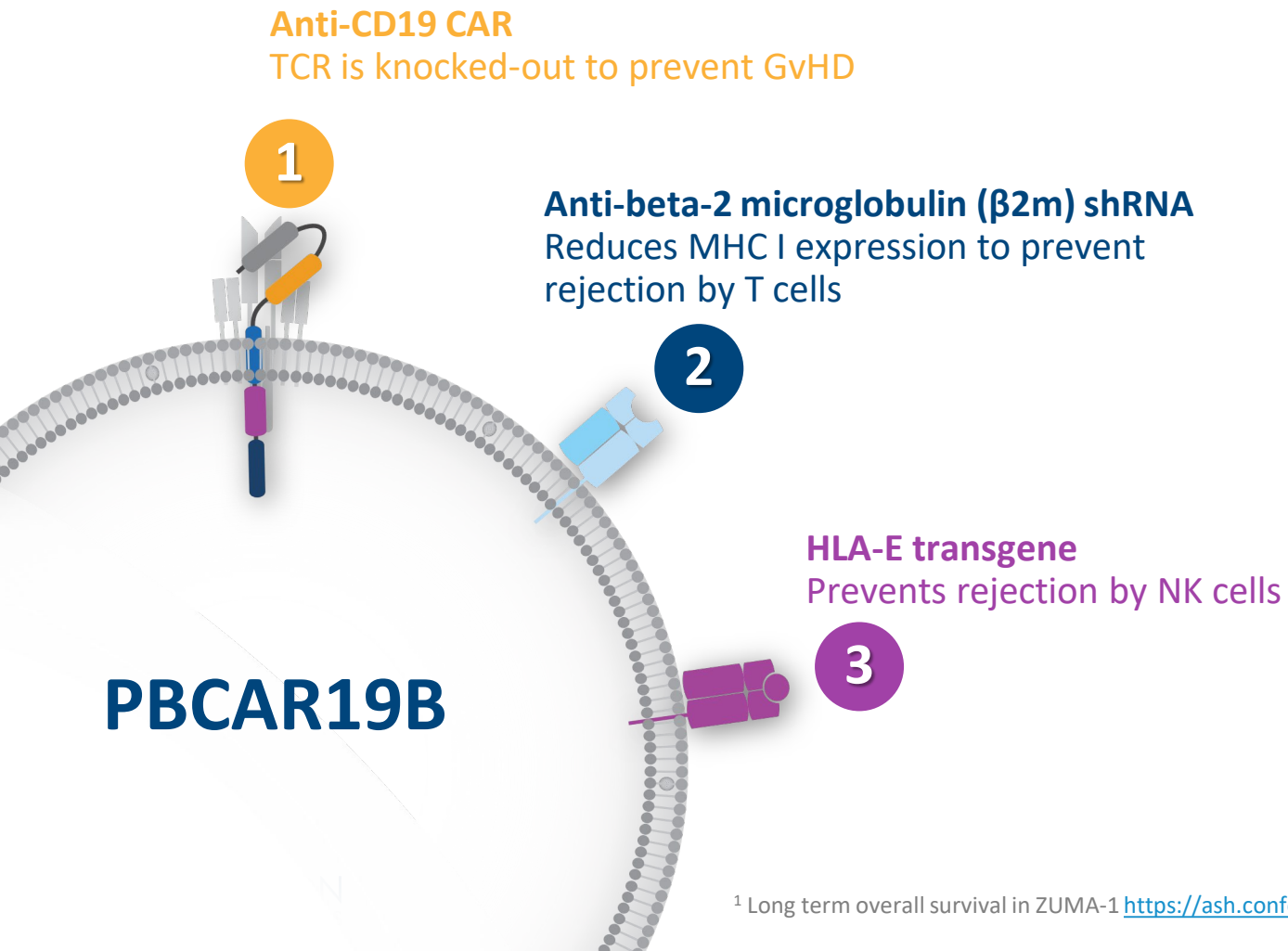
PBCAR19B “Stealth Cell”

Anti-CD19 allogeneic CAR T designed to evade immune rejection and displace 2nd/3rd line auto CAR T

PBCAR19B: Anti-CD19 Allogeneic CAR T Designed to Evade Immune Rejection and Displace 2nd/3rd Line Auto CAR T

Ex Vivo

PBCAR19B



3rd line+ NHL Target Product Profile to Replace Auto CAR T

Median Overall Survival (mOS)	>	~26 months ¹
Overall Response Rate (ORR)	>	> 70% at 28 days
Duration of Response (DoR)	>	~35% at 6 months to 1 year+
Safety	>	Same or better than auto CAR T
Expected Regulatory Hurdle	>	Head-to-head vs. auto CAR T/transplant

¹ Long term overall survival in ZUMA-1 <https://ash.confex.com/ash/2021/webprogram/Paper148078.html>

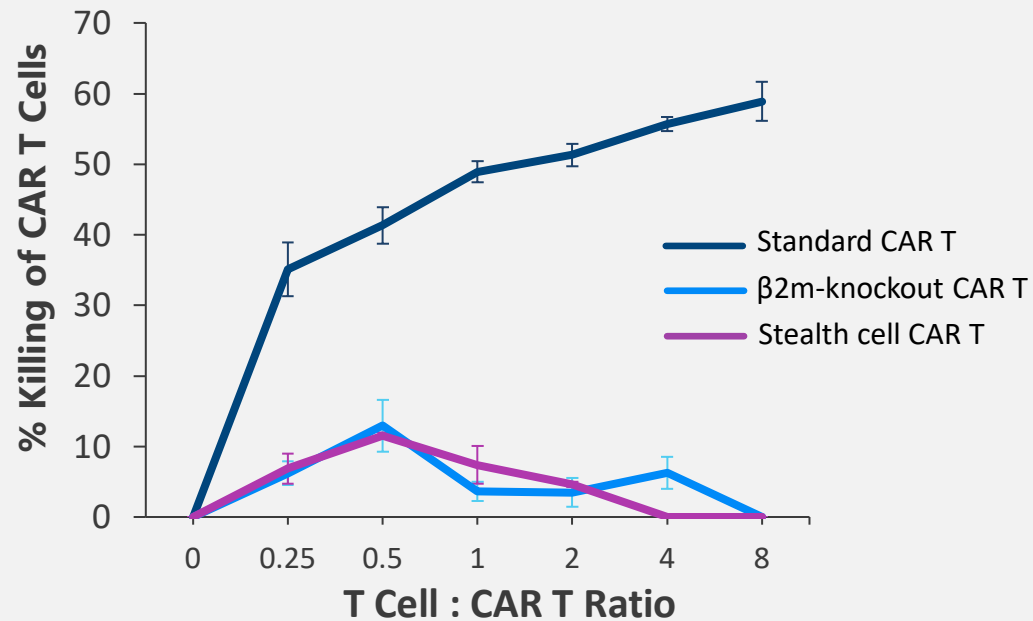
PBCAR19B: Designed to Overcome Rejection by T Cells and NK Cells in Mixed Lymphocyte Reactions

Ex Vivo

PBCAR19B

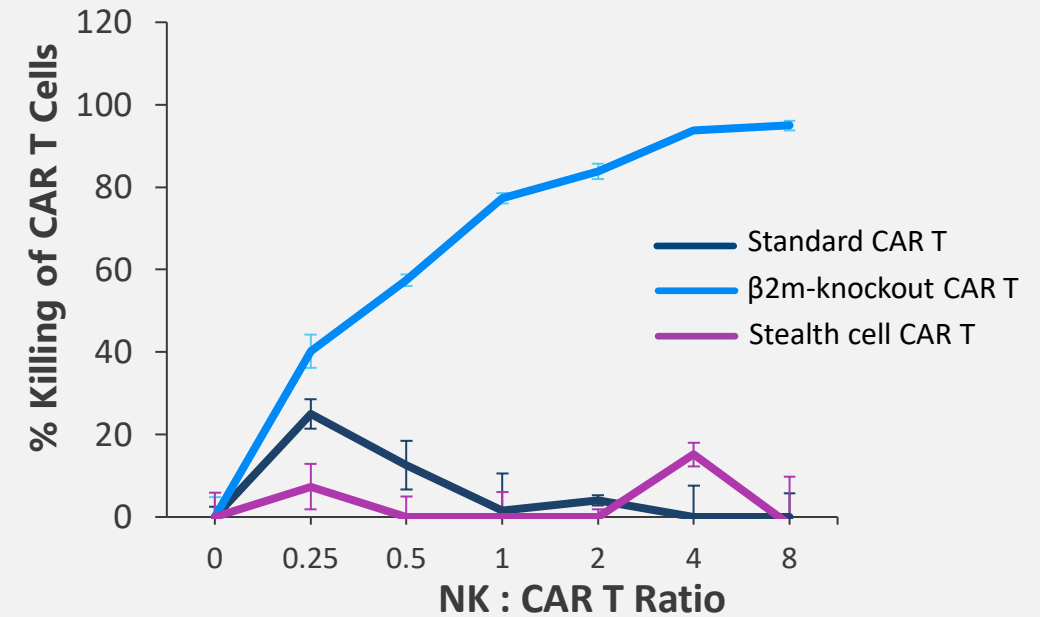
Rejection by T cells

(n=4 mismatched donors)

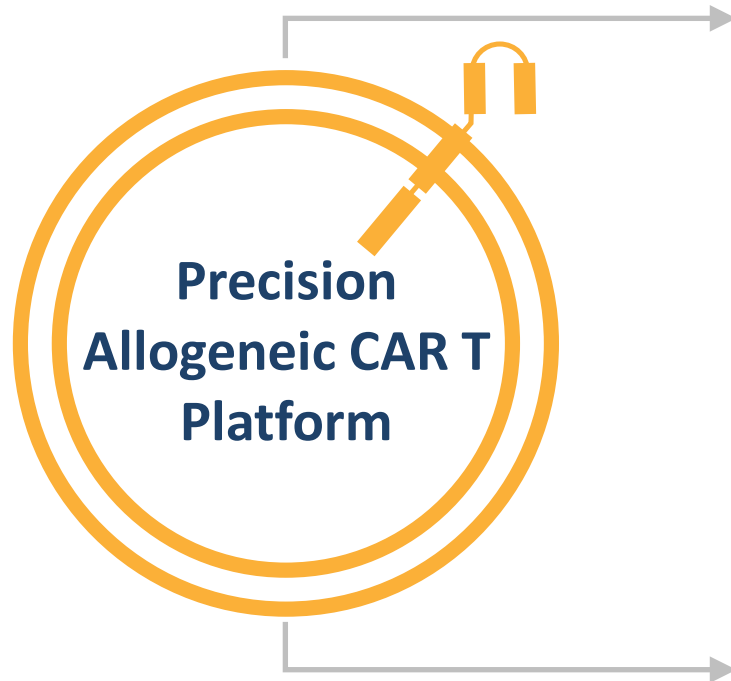


Rejection by NK cells

(n=3 mismatched donors, strong activation protocol)



What to Expect in Q1 2023 Program Update



Azer-cel

- Additional long-term follow up from ASH 2021 and ASCO 2022 cohorts
- New subjects treated with optimized cells at DL4 (500M cells) and less intense LD
 - Peak expansion data (pharmacokinetics)
 - Safety
 - Response rates in evaluable subjects
- If data is supportive, proceed with request for FDA clinical meeting

PBCAR19B

- Subjects treated with optimized cells at DL2 (540M cells)
 - Peak expansion data (pharmacokinetics)
 - Safety
 - Response rates in evaluable subjects
- Recruiting additional patients for PBCAR19B program in R/R DLBCL to complete Phase 1



Differentiating the ARCUS platform

Highly differentiated genome editing platform for
high unmet needs in genetic diseases

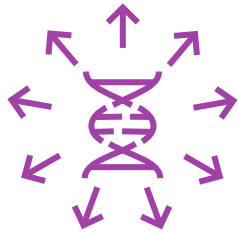
Three Distinct Advantages of ARCUS Genome Editing Platform



Precision (Safety & Specificity)



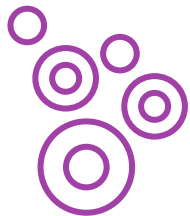
- ARCUS is inactive until it binds to target DNA
- Off-target editing easier to detect and eliminate
- Iterative nuclease optimization process to customize safety and efficacy profile



Versatility



- Gene insertion, deletion or repair/complex edit
- DNA cuts are preferentially repaired by homology-directed repair (HDR)



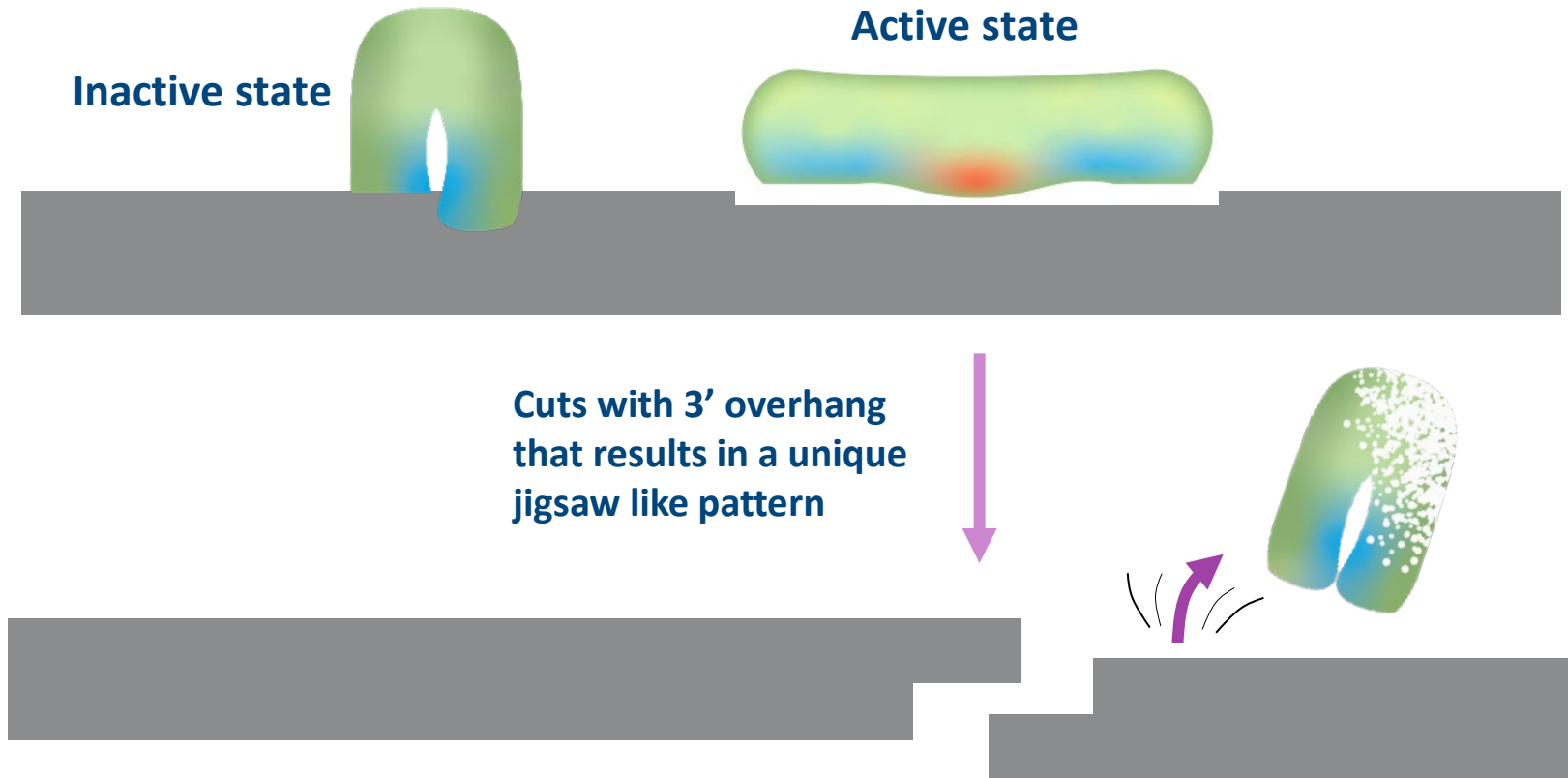
Delivery



- ARCUS nucleases are small (364 amino acids)
- Delivery to broad array of tissues and cells using adeno-associated virus (AAV) and/or lipid nanoparticle (LNP)

ARCUS: Engineering Nucleases that Mimic Nature's Genome Editing System

The ARCUS nuclease is the only gene editor derived from a natural homing endonuclease



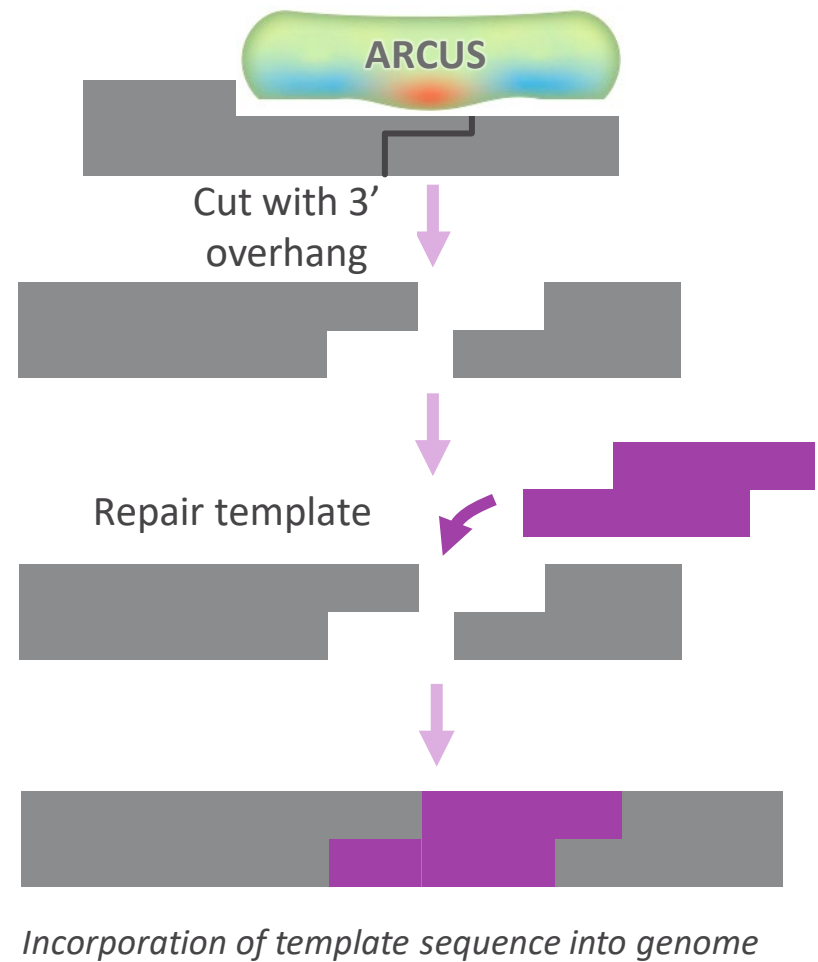
The ARCUS nuclease cuts DNA at a specified site – can result in simple and complex edits

ARCUS is Capable of Simple Edits and Optimally Suited for Complex Edits

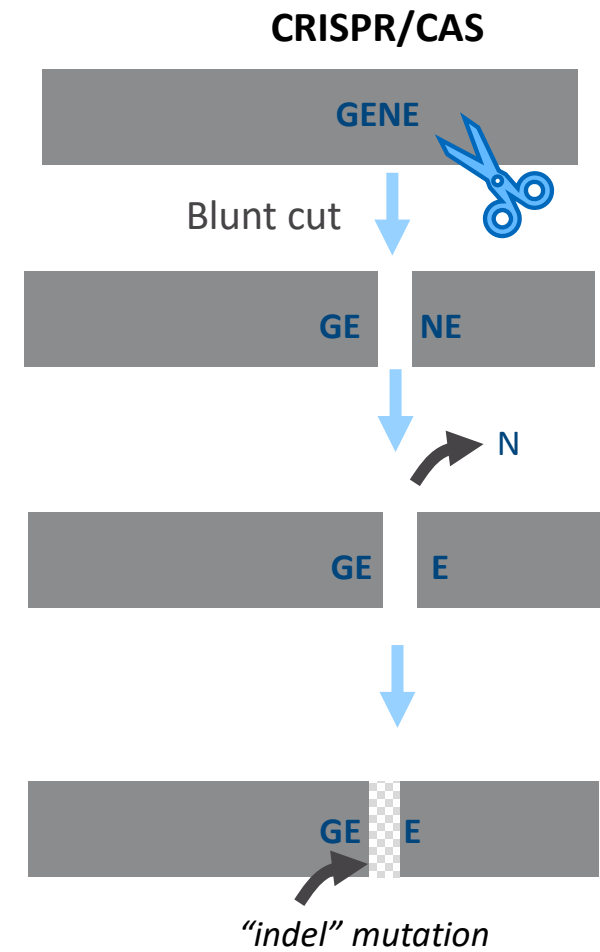
Simple Edit		Deletion	A deletion (also known as <u>knock-out</u>) is when a gene is made inoperative through a permanent change in the DNA; therefore, the gene no longer expresses a functional protein
Complex Edits		Insertion	An insertion (also known as <u>knock-in</u>) is when a healthy copy of a gene is inserted into the genome through a permanent change in the DNA
		Excision	An excision is when a gene is altered through permanent removal of a portion of the genome, requiring multiple cuts in the DNA
		Replacement	A replacement is when a healthy copy of a gene is inserted into the genome at the same time as the defective gene is removed from the genome

Precise 3' Overhang Cuts are Unique - Designed to Enable Gene Insertion and Complex Edits and Provide Identifiable Signature for On-Target Editing

Complex/Homology-directed Repair (HDR)

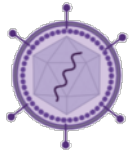


Simple/Non-Homologous End Joining (NHEJ)



Effective Gene Editing Requires Both AAV and LNP Delivery Capabilities

ARCUS can be effectively delivered with both AAV and LNP



AAV



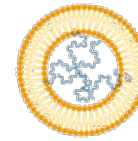
PROS

- Sustained nuclease expression may provide increased efficacy
- **Gene insertion requires AAV to deliver DNA repair template**
- **AAV has been well-tolerated** in NHP studies to date
- **Tissue specificity:** target expression in target tissue only (via promoters)
- **Can deliver to broad tissue** (CNS, muscle, stem cells, etc.)



CONS

- **Pre-existing neutralizing antibodies** and prior **AAV based therapies** may limit patient access
- **Safety risk:** AAV integrations, risk of long-term immune response to long expression of the nuclease
- **Size limitations** on cargo capacity is a challenge for most gene editing technologies; **ARCUS is small and fits in AAV**



LNP-mRNA



PROS

- **Potential to repeat dose** to maximize therapeutic impact
- **Scalability** for manufacturing
- **Transient (~48 hours)** nuclease expression **may have better safety profile**
- **No risk of insertion of the payload into the genome** since mRNA delivered by LNP cannot integrate into the genome



CONS

- **Transient (~48 hours)** nuclease expression **may limit efficacy**
- **Tolerability/Immunogenicity** of mRNA is a concern and high doses increase risk of complement activation
- **LNPs are largely restricted to use for liver** delivery today (novel approaches may permit LNPs beyond liver in future)
- **Payload (insertion) can not directly deliver DNA into the nucleus;** requires additional delivery system (e.g., AAV)

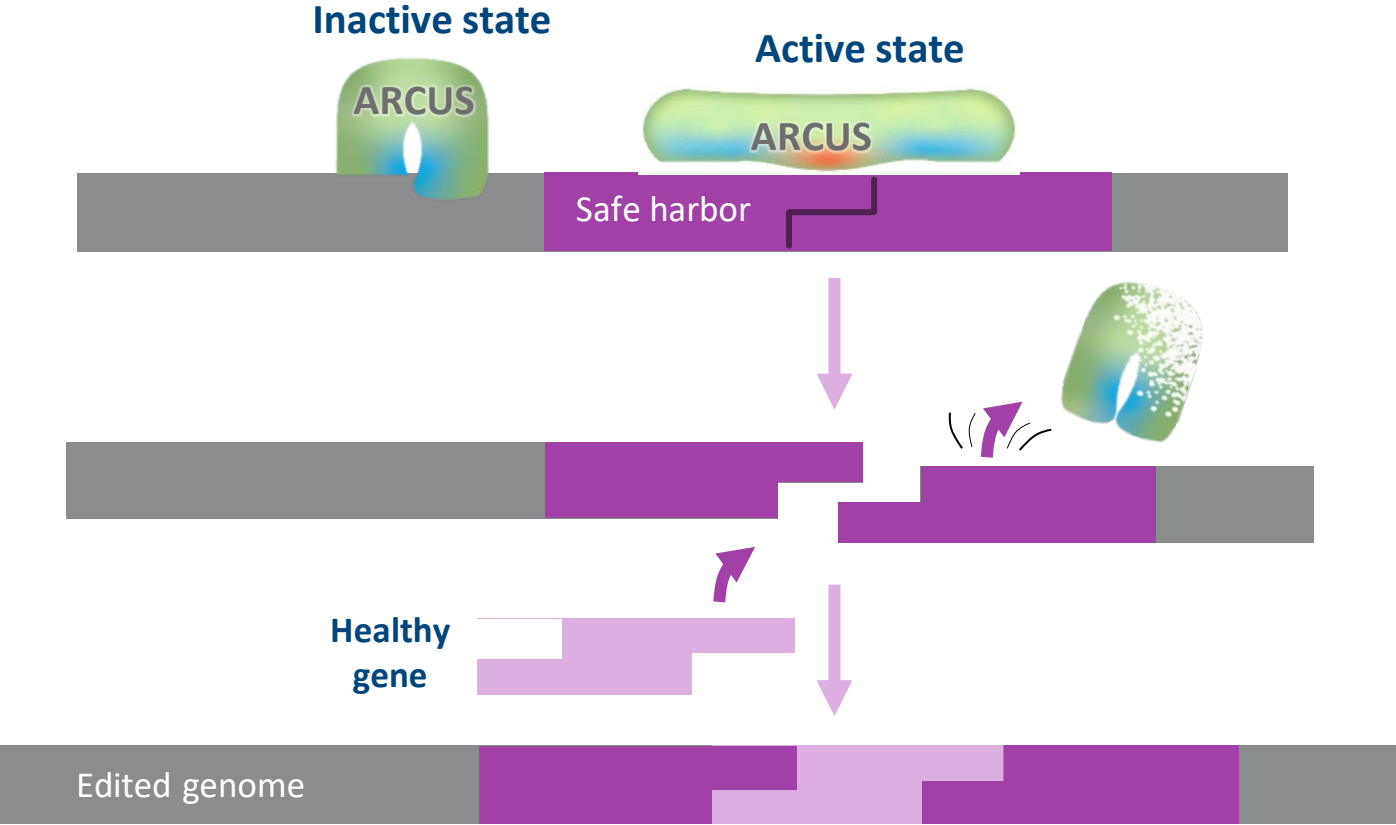
Unlocking Greater *In Vivo* Editing Potential via Safe Harbor, ARCUS Gene Insertion

A “safe harbor” ARCUS nuclease may be used to **develop multiple products to insert a different gene for each disease**

Complex Edit



Insertion

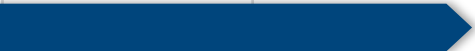
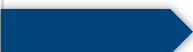


In Vivo Gene Editing Programs

Precision *in vivo* gene insertion and complex gene editing programs highlight unique ARCUS attributes



Broad and Deep *In Vivo* Pipeline Showcases ARCUS for Gene Insertion and Complex Edits

PROGRAM	INDICATION	TISSUE	TARGET	COMPLEX EDIT TYPE / DELIVERY	RESEARCH	CANDIDATE SELECTION	IND-ENABLING	PARTNER
PBGENE-HBV	Chronic hepatitis B	Liver	HBV	Deletion/LNP				
PBGENE-HbE	Sickle cell disease/ beta thalassemia	HSCs	—	Insertion/—				
PBGENE-DMD	Duchenne muscular dystrophy	Muscle	DMD	Excision/AAV				
PBGENE-LLY2	Undisclosed	Liver	—	—				
PBGENE-LLY3	Undisclosed	CNS	—	—				
iECURE-OTC	Ornithine transcarbamylase deficiency	Liver	OTC	Insertion/AAV				
iECURE-PKU	Phenylketonuria	Liver	PAH	Insertion/AAV				



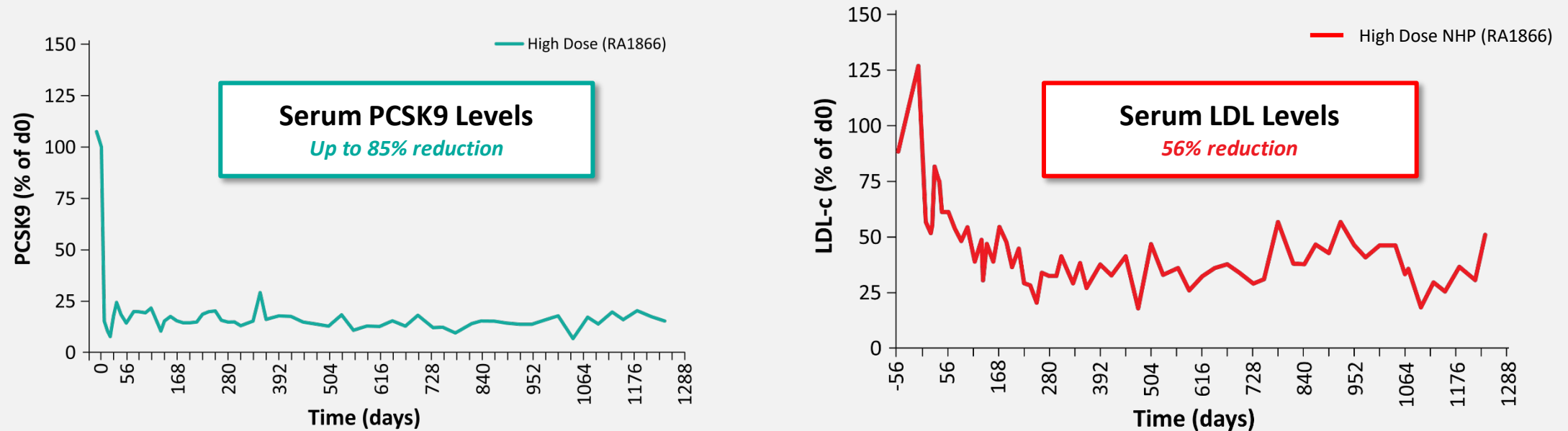
ARCUS Safety Supported by Long-term Non-Human Primate Data

Differentiated ability to track and minimize off-target editing

Five Years of Follow-up for ARCUS in NHP; Longest, Publicly Known Gene Editing Data

Non-Human Primate Animal Model

One-time delivery of an **AAV-ARCUS** vector into PCSK9 safe harbor for deletion resulted in long-term reductions of serum PCSK9 and LDL¹ (5-year timepoint approaching) with no additional safety concerns

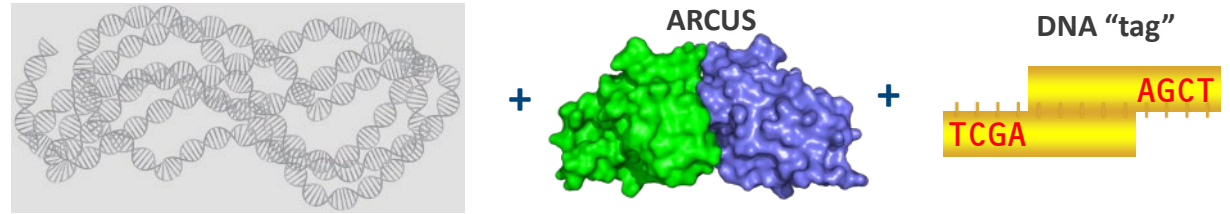


★ **ARCUS delivered by AAV has been studied in the most extensive large animal data set of any gene editing tool showing sustained, safe deletion in long term NHP studies**

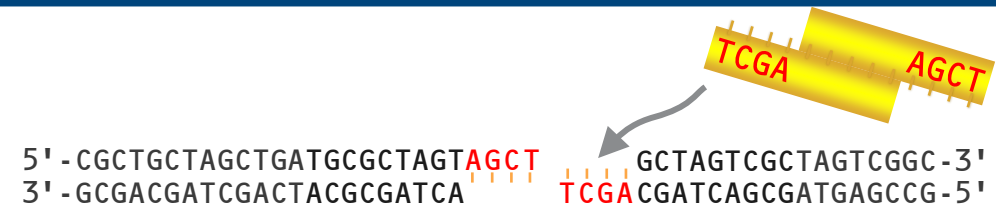
ARCUS Safety and Specificity for Tracking Off-Target Editing

Oligo Capture: A genome-wide assay for ARCUS off-target editing

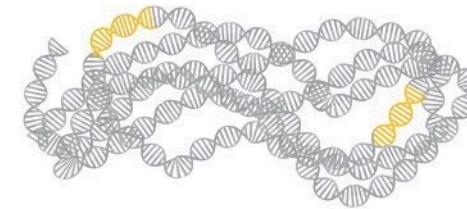
Step 1: Transfect cells with ARCUS and a DNA “tag”



Step 2: The “tag” is captured at DNA breaks resulting from ARCUS cleavage



Step 3: Genomic DNA is isolated from cells and evaluated on a next-gen sequencer to identify all sites where the “tag” was captured



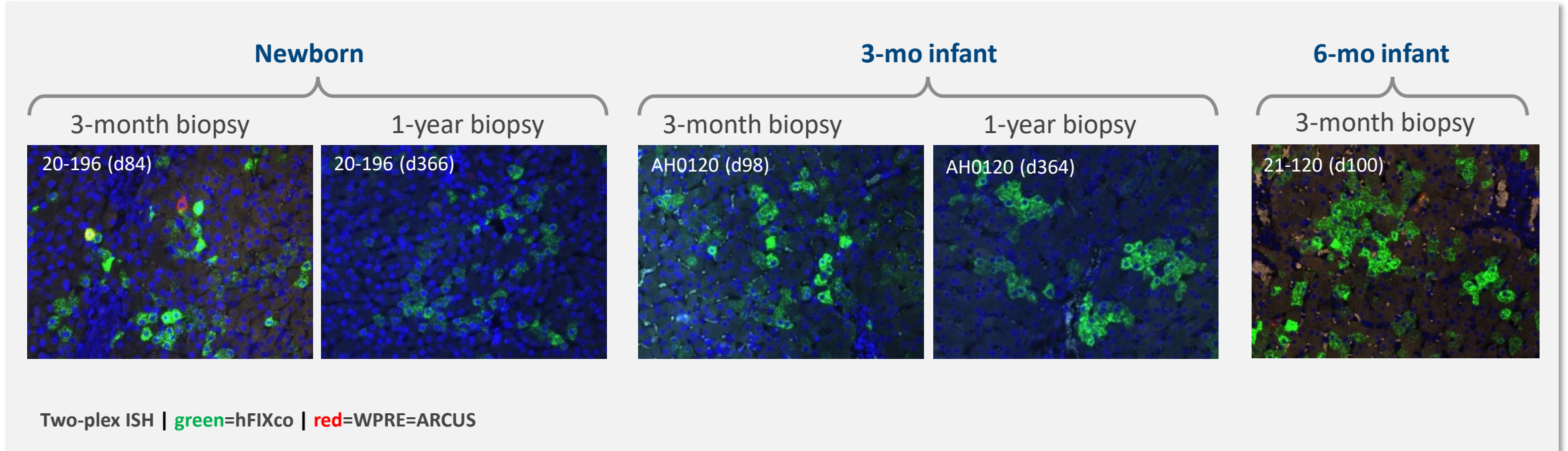
Step 4: Sites of on- and off-target capture of the DNA “tag” are identified and deep sequenced to confirm editing

```
ATGCTAGCTAGCTAGCTGATCGATGCTAGCTAGCTAGCTA
GTAGCTAGTAAGCTGATCGTAGCTGCCGCTGCTAGCTGA
TGCGCTAGTAGCTGCTAGTCGCTAGTCGGCAGTCGATGC
TGCTAGCTAGTAGCTGCATGCTAGCTAGTGTGTCGATGT
```

ARCUS for Gene Insertion

Wholly owned and partnered programs, including those for large gene insertions

Hemophilia B Gene Insertion: Stable Transduction of Factor IX in Newborn and Infant Macaque Liver (Non-Human Primates)¹



Green cells show that the Factor 9 (FIX) gene has been inserted and stable over 1 year

★ Factor 9 (FIX) gene insertion demonstrated in both newborn and infant NHPs to address Hemophilia B

Ornithine Transcarbamylase Deficiency (OTC) Clinical Candidate Shows Stable Gene Insertion at Year 1¹

In Vivo

Gene Insertion
for OTC >

Delivery of twin AAV-based vectors carrying ARCUS nuclease vector (GTP-506A) and therapeutic donor vector (GTP-506D) via PCSK9 “safe harbor” site



Long-term stability of the edited genome in NHPs was demonstrated in newborn and infant macaques

- › Efficient targeted insertion was achieved in NHPs up to three months of age, and studies of older infants are ongoing
- › 12-month follow-up biopsies continued to demonstrate durability, with gene insertion efficiency up to 28.2%², well above the expected threshold for clinical benefit
- › AAV delivery well tolerated; no evidence of liver histopathology in any ARCUS-treated animals

★ Durable gene editing efficiency continues to be demonstrated in NHP studies using ARCUS

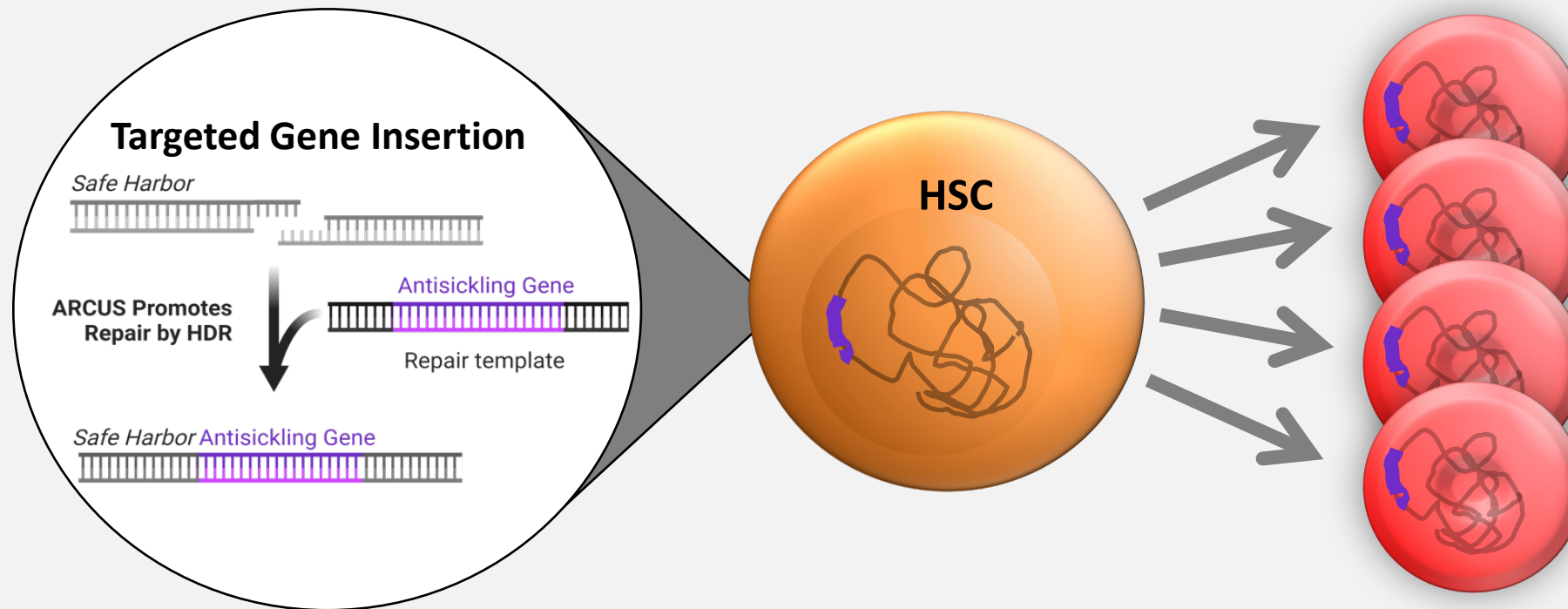
Gene Insertion to a “Safe Harbor” Locus in Hematopoietic Stem Cells

In Vivo

[Gene Insertion for
SCD and Beta Thal >](#)

ARCUS will be used to add an antisickling gene to hematopoietic stem cells (HSCs)

Permanent integration of an antisickling gene into a “safe harbor” locus in HSCs is expected to prevent the sickle cell phenotype in mature erythrocytes.



ARCUS for Complex Edits

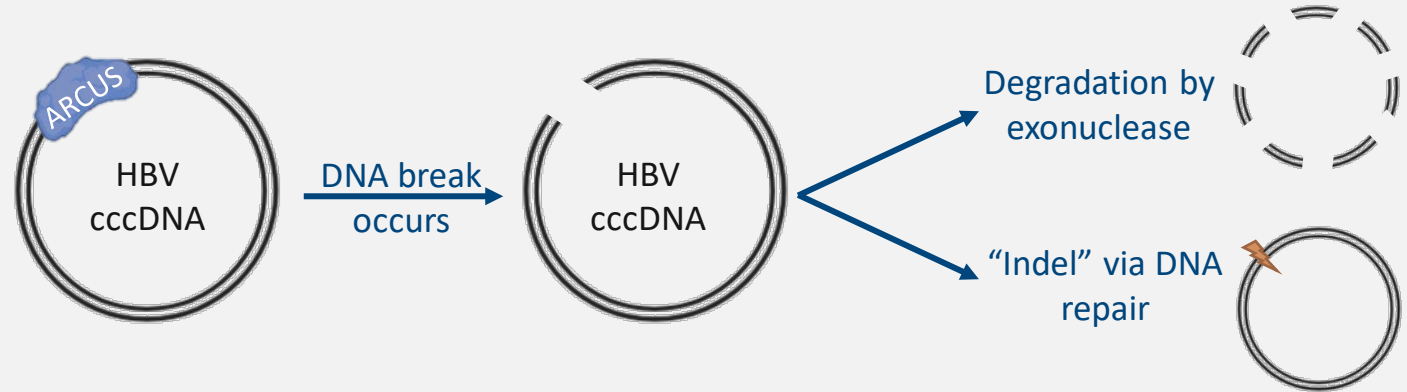
- › HBV approach edits DNA at two locations
- › DMD approach makes a 500,000 base pair edit

Precision Approach in HBV: Excision in Two Locations in the Liver

Curing HBV requires targeting two key viral components – cccDNA and integrated HBV DNA

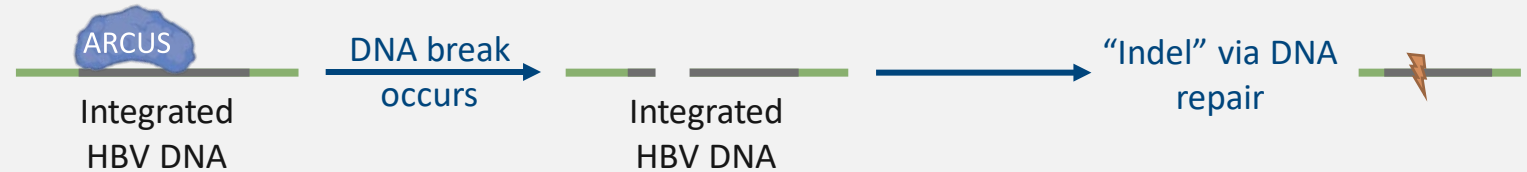
1. cccDNA

cccDNA is a pool of minichromosomes that hangs out in nucleus of liver cells and can re-establish infection



2. Integrated HBV DNA

Integrated HBV DNA attaches into the DNA and genome of liver cells



★ Precision's therapeutic approach targets both HBV viral components enabling a path towards a potential cure

ARCUS HBV Program has Shown Exciting Efficacy Data During *In Vivo* POC Studies Demonstrating Ability to Reduce Both HBV S-Antigen and HBV cccDNA

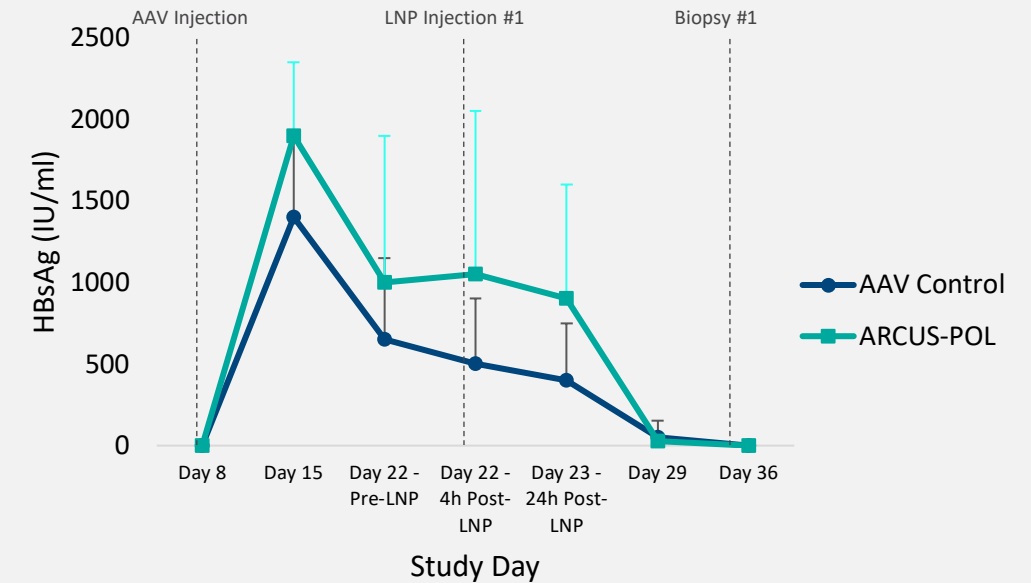
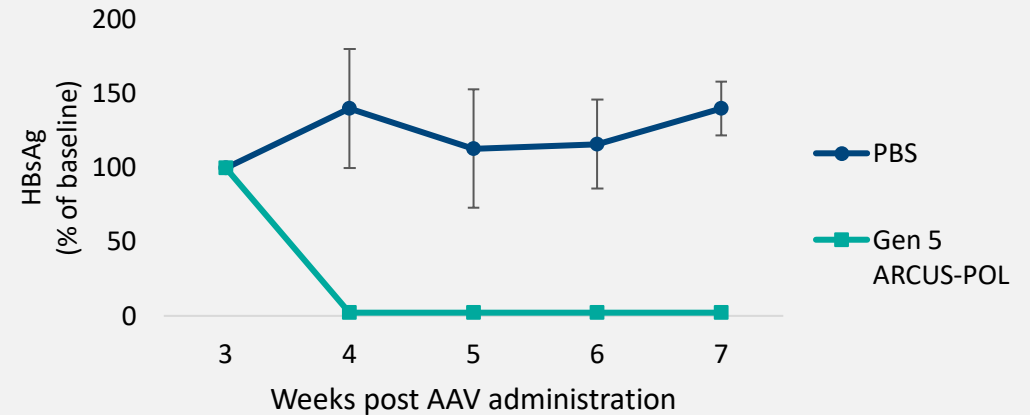
In Vivo

[Gene Excision for Chronic Hep B >](#)

Data from *in vivo* models¹ treated with ARCUS-HBV nuclease via LNP showed potential for delivering cures

★ **96% reduction** in serum **HBsAg levels** and substantial reduction in the liver

★ **83% reduction** in HBV cccDNA

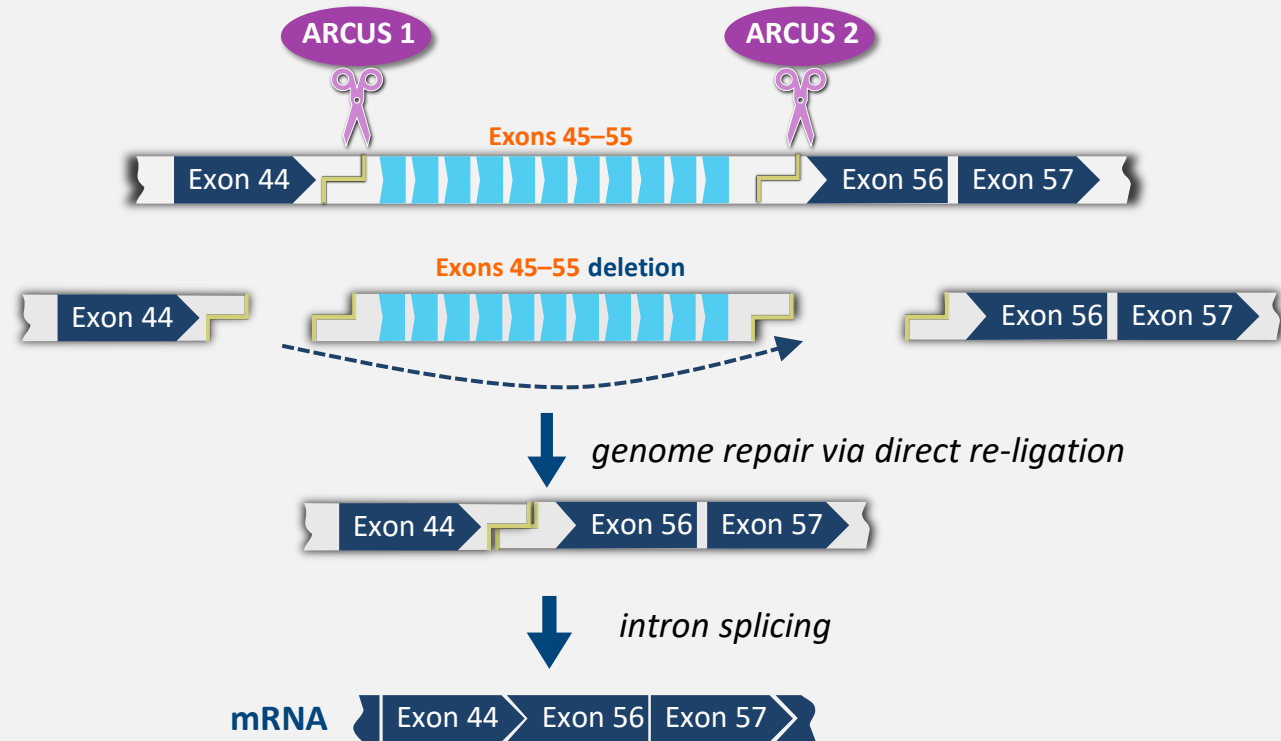


Restore dystrophin expression

Deleting exons 45-55 using a pair of ARCUS nucleases intended to remove a mutation hotspot responsible for >50% of DMD

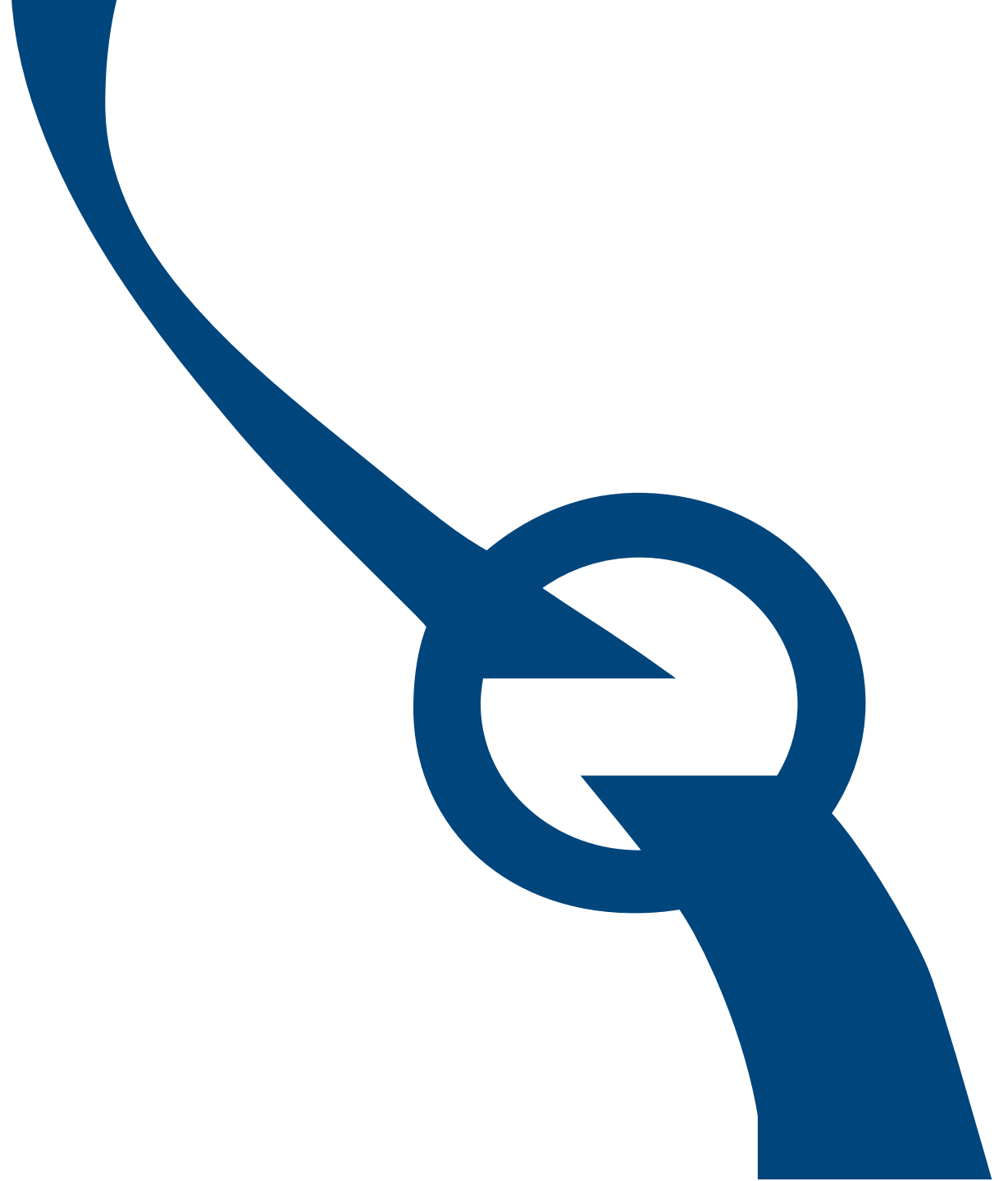
GOAL

ARCUS nucleases to make complex edit of the genome and make a variant of the dystrophin protein that is functionally competent





2023 Priorities



2023 Priorities and Milestones

- › Complete Phase 1b for azer-cel to support decision point for Phase 2
- › Complete Phase 1 dosing trial for PBCAR19B
- › Host CAR T update in first quarter of 2023
- › Nominate the final drug candidate for HBV *in vivo* program
- › Advance the first ARCUS *in vivo* nuclease to CTA through a partner
- › Publish new preclinical data to further support *in vivo* gene editing programs
- › Host *in vivo* gene editing R&D Day around mid-2023
- › Further extend the cash runway

**Building the leading
therapeutic gene editing
company** focused on high
unmet needs in oncology and
genetic diseases

Technology: ARCUS, a premier
genome editing platform

People: Fortified Senior Leadership
Team with 15+ years perfecting
ARCUS protein engineering

Focus & Discipline: Fiscal resources
support 2+ years runway

Precision BioSciences: Senior Leadership Team



Michael Amoroso

Since Sep 2021
President &
Chief Executive Officer



Derek Jantz, Ph.D.

Since Mar 2006
Chief Scientific Officer,
Co-Founder



**Jeff Smith,
Ph.D.**

Since Mar 2006
Chief Research Officer,
Co-Founder



Alex Kelly

Since Oct 2020
Chief Financial Officer



Cindy Atwell

Since Apr 2019
Chief Business Officer



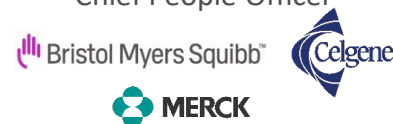
Alan List, M.D.

Since Apr 2021
Chief Medical Officer



Juli Blanche

Since May 2022
Chief People Officer



Dario Scimeca

Since Jun 2019
General Counsel



Neil Leatherbury

Since Mar 2017
VP, CMC



Precision BioSciences: Board of Directors



Kevin Buehler

Since Jul 2019
Board Chair



Michael Amoroso

Since Oct 2021



Melinda Brown

Since May 2022
Audit Chair



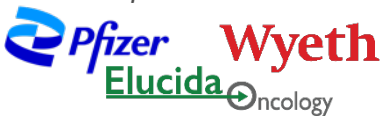
Stanley Frankel, M.D.

Since Apr 2021
Science & Technology Chair



Geno Germano

Since Mar 2020
Compensation Chair



Shari Lisa Piré, J.D.

Since Nov 2021



Sam Wadsworth, Ph.D.

Since Nov 2021



Appendix



ARCUS for Chronic Hepatitis B Virus (cHBV) Targeting cccDNA

In Vivo

Gene Insertion for
Chronic Hep B

ARCUS-mediated **inactivation** of cccDNA and **integrated HBV** could result in a functional cure

Chronic HBV is one of greatest racial health disparities in the U.S.

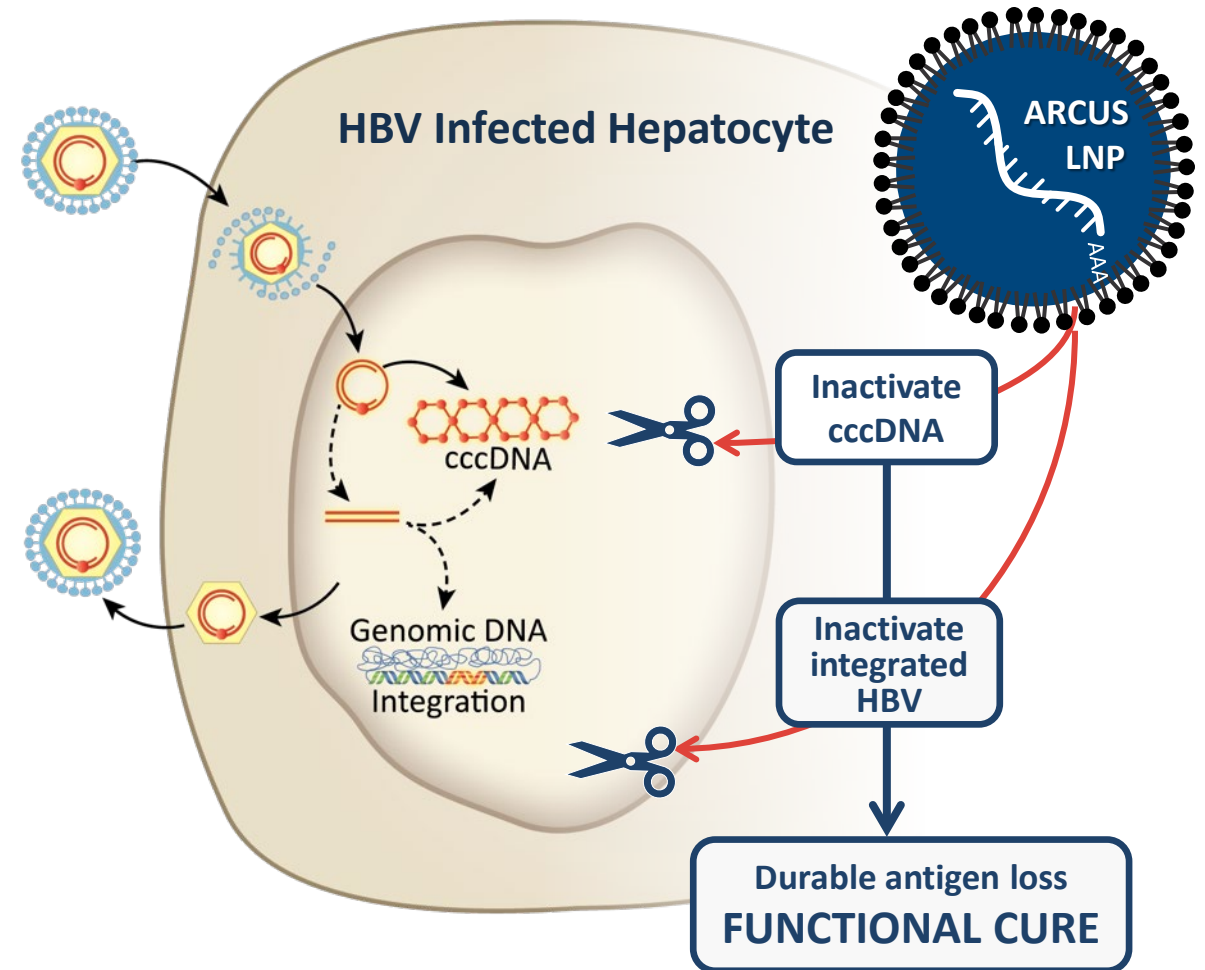


> **860,000**
cHBV infections in the US



> **290 million**
cHBV infections globally

- > 90% of infected infants develop cHBV
- ≤ 50% of infected children 1-5 years develop cHBV
- 5-10% of infected healthy adults develop chive



Novartis Collaboration Complementary to Precision's Existing *In Vivo* Gene Editing Partnership with Lilly

In Vivo

Gene Insertion
for DMD

Research collaboration and license agreement aimed at treating challenging genetic diseases



+

Lilly

Pre-IND R&D

IND to Commercial

3 Initial collaboration for **three targets**, including Duchenne muscular dystrophy and **two other undisclosed programs targeting the liver and CNS**

+

3 Lilly retains right to select up to **three additional gene targets**

- › Upfront payment of \$135M including \$35M equity
- › Up to \$420M per target in development and commercialization milestones
- › Mid-single digit to low tweens tiered royalties

Duchenne Muscular Dystrophy Currently Lacks a Curative Treatment

In Vivo

Gene Insertion
for DMD

On average,
children **lose
their ability
to walk** by
age 12



Mutation on the X chromosome interferes with dystrophin protein production, which is needed to form and maintain healthy muscle

Affects approximately
1 in 3,500
live male births



In Vivo Gene Insertion Collaboration with Novartis for Hemoglobinopathies

In Vivo

Gene Insertion for
Hemoglobinopathies

Builds on the unique gene insertion capabilities of ARCUS® and further validates ARCUS as a premium genome editing platform



+



NOVARTIS

Develop single
ARCUS nuclease

Preclinical to
commercial

Collaboration with Novartis,
a global gene therapy leader

**Precision to develop a single
ARCUS nuclease** for certain
hemoglobinopathies such as
sickle cell disease and beta
thalassemia

**Goal to design ARCUS
nuclease for safe and efficient
in vivo gene insertion**

- › Precision receives \$75M upfront for a single target/single nuclease
- › Eligible to receive up to an additional \$1.4B in milestones and tiered royalties on sales of licensed products
- › Collaboration adds hematopoietic stem cells (HSCs) to existing *in vivo* gene editing programs targeting the liver, muscle and central nervous system
- › One-time, treatment for hard-to-treat genetic blood disorders such as sickle cell disease (SCD) and beta thalassemia

Hemoglobinopathies are a Major World Health Problem

In Vivo

Gene Insertion for
Hemoglobinopathies

Sickle Cell Disease (SCD)

Affects the structure/function of hemoglobin, reducing the ability of red blood cells to transport oxygen

- Acute sicklecell pain crises and life-threatening complications

Beta Thalassemia

One of the most common genetic diseases caused by a disruption of normal hemoglobin production

- Complications: Overproduction of red blood cells inside and outside of the bone marrow, heart disease, chronic liver hepatitis, defects of the reproductive system, diabetes, and rare skin disorders

Sickle Cell Disease Affects

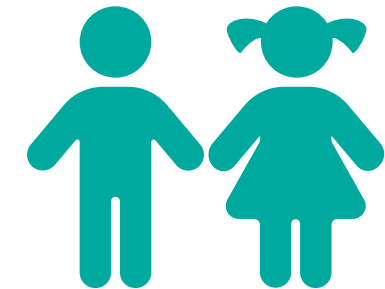
>300,000

newborns
annually



~1,000 children

in Africa are born with SCD
every day and > 50% will not
reach their 5th birthday



~68,000

children born with
thalassemia each year

OTC is a Severe, Ultra Rare Genetic Condition With Extremely High Unmet Medical Needs Across Phenotypes

Ornithine Transcarbamylase (OTC) Deficiency

- OTC deficiency is the most common **urea cycle disorder**
- Disease prevalence is between **1 in 60,000** and **1 in 72,000**
- Neonatal onset has been associated with **mortality rates as high as 74%**¹
- A **liver transplant** is typically required by six months of age and is the only known curative treatment
- OTC deficiency is also associated with numerous **neuropsychological complications**
- **High ammonia levels** can lead to development delays, learning and intellectual disabilities, ADHD and executive function deficits, seizures, coma and death

~ 4,200 People with OTC in the US²

Neonatal-onset (<30 days since birth)	Late-onset Symptomatic (>30days to 16yo)	Late-onset Asymptomatic Adult (>16yo)
Majority male X-linked / No enzymatic activity	Majority females w/skewed X-inactivation / Limited enzymatic activity	Majority female, asymptomatic
Catastrophic disease managed by liver transplants & aggressive medical mgt. / Significant neurocognitive problems and lower life expectancy	Severe disease managed by medical mgt. High risk for neurocognitive problems Liver transplant / mortality risks exist	Usually manifest during stress situation (surgery, post-childbirth) Rare but high mortality at initial event

¹ Complete removal of OTC activity results in severe neonatal disease, while decreased OTC results in late-onset.

² Onset may occur at any age though is more common in infancy. HAC: Hyperammonemic Crisis, defined as plasma ammonia levels $\geq 150 \mu\text{mol/L}$ together with clinical symptoms probably related to hyperammonemia. 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.