CRISPR THERAPEUTICS®

Creating transformative gene-based medicines for serious diseases

Corporate Overview | Q4 2023

Forward-Looking Statements

The presentation and other related materials may contain a number of "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995, as amended, including statements regarding CRISPR Therapeutics' expectations about any or all of the following: (i) its plans and expectations for its preclinical studies, clinical trials and pipeline products and programs; (ii) the safety, efficacy and clinical progress of its various clinical programs; (iii) the status of preclinical studies and clinical trials (including, without limitation, the expected timing of data releases, announcement of additional programs and activities at clinical trial sites, and discussions with regulatory authorities) and expectations regarding the data that is being presented; (iv) the data that will be generated by ongoing and planned preclinical studies and clinical trials and the ability to use that data for the design and initiation of additional preclinical studies and clinical trials; (v) regulatory submissions and authorizations, including timelines for and expectations regarding additional regulatory agency decisions; (vi) manufacturing activities; (vii) the activities under its collaborations and the expected benefits thereof; (viii) its intellectual property coverage and positions of its, its licensors and third parties as well as the status and potential outcome of proceedings involving any such intellectual property; (ix) the sufficiency of its cash resources; and (x) the therapeutic value, development, and commercial potential of CRISPR/Cas9 gene editing technologies and therapies, including as compared to other therapies. Without limiting the foregoing, the words "believes," "anticipates," "plans," "expects" and similar expressions are intended to identify forward-looking statements. You are cautioned that forward-looking statements are inherently uncertain. Although CRISPR Therapeutics believes that such statements are based on reasonable assumptions within the bounds of its knowledge of its business and operations, forward-looking statements are neither promises nor guarantees and they are necessarily subject to a high degree of uncertainty and risk. Actual performance and results may differ materially from those projected or suggested in the forward-looking statements due to various risks and uncertainties. These risks and uncertainties include, among others, that: the efficacy and safety results from ongoing clinical trials will not continue or be repeated in ongoing or planned clinical trials or may not support regulatory submissions; the FDA or other regulatory authorities may not approve exa-cel on a timely basis or at all; adequate pricing or reimbursement may not be secured to support continued development or commercialization of exa-cel following regulatory approval; the potential that clinical trial results may not be favorable; one or more of its product candidate programs will not proceed as planned for technical, scientific or commercial reasons; future competitive or other market factors may adversely affect the commercial potential for its product candidates; initiation and completion of preclinical studies for its product candidates is uncertain and results from such studies may not be predictive of future results of future studies or clinical trials; regulatory approvals to conduct trials or to market products are uncertain; it may not realize the potential benefits of its collaborations; uncertainties regarding the intellectual property protection for its technology and intellectual property belonging to third parties, and the outcome of proceedings (such as an interference, an opposition or a similar proceeding) involving all or any portion of such intellectual property; and those risks and uncertainties described under the heading "Risk Factors" in its most recent annual report on Form 10-K, quarterly report on Form 10-Q, and in any other subsequent filings made by it with the U.S. Securities and Exchange Commission, which are available on the SEC's website at www.sec.gov. Existing and prospective investors are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date they are made. CRISPR Therapeutics disclaims any obligation or undertaking to update or revise any forward-looking statements contained in this presentation, other than to the extent required by law.

Caution should be exercised when interpreting results from separate trials involving separate product candidates. There are differences in the clinical trial design, patient populations, and the product candidates themselves, and the results from the clinical trials of autologous products may have no interpretative value on our existing or future results.

CRISPR Therapeutics Highlights

Leading gene editing company | Broad pipeline | Best-in-class platform and capabilities

Broad pipeline of *ex vivo* and *in vivo* programs across four franchises: hemoglobinopathies, immuno-oncology, regenerative medicine, and *in vivo* approaches

Historic first approval of a CRISPR-based gene-editing therapy with CASGEVY™ (exa-cel), now approved in the U.S. for certain eligible patients with sickle cell disease

Next-generation allogeneic CAR T programs, CTX112 and CTX131, advancing in the clinic with potency edits to improve tumor killing capacity and resistance to suppression

Proven track record of execution with best in-class capabilities and state-of-the-art internal GMP manufacturing facility

Preeminent CRISPR technology platform focused on the innovation that matters for transformative medicines

Several catalysts upcoming across each franchise

Transforming Medicine Across Four Core Franchises

Hemoglobinopathies

First approval of a CRISPR-based gene-editing therapy in the world[1]

Immuno-oncology

Next-generation edited allogeneic immune cells for cancer

Regenerative Medicine

Edited, stem cell-derived beta cells for diabetes

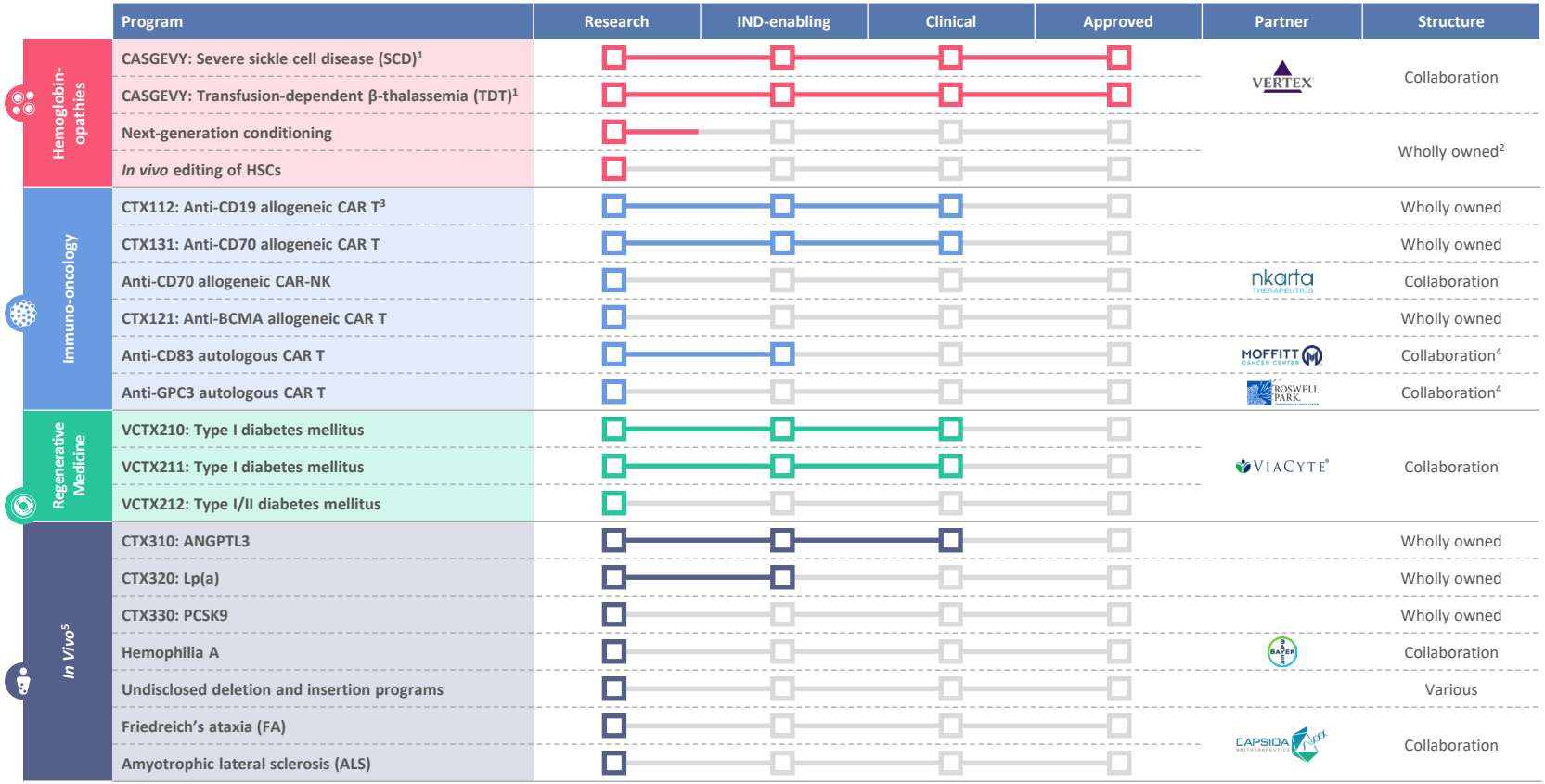
In vivo

>10 programs using both LNP and AAV approaches

Platform (next-generation editing and delivery)

(1) CASGEVY approved by the U.S. FDA for certain eligible patients with SCD and granted conditional marketing authorization from the UK MHRA and Bahrain NHRA for certain eligible patients with SCD or TDT in Q4 2023

Our Pipeline



(1) Currently approved in some countries for certain eligible patients with SCD or TDT; (2) Collaboration with Vertex for applications in SCD and β-thalassemia; (3) Initiation of additional trial in systemic lupus erythematosus planned for 1H 2024; (4) CRISPR retains commercial rights; (5) Partnered with Vertex on several additional disease areas, including DMD, DM1, and CF

Potential Functional Cure with CASGEVY (exa-cel)

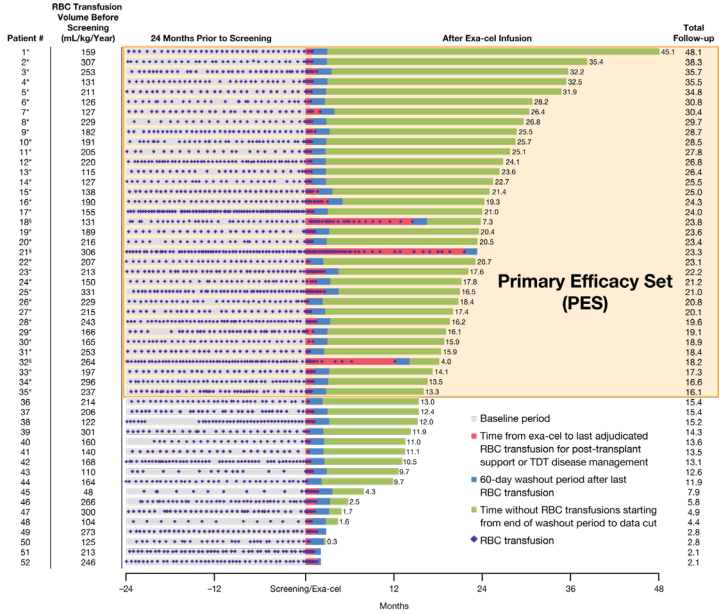
- **CASGEVY (exagamglogene autotemcel [exa-cel]) approved by the U.S. FDA** for the treatment of SCD in eligible patients 12 years and older with recurrent vaso-occlusive crises, and **granted conditional marketing authorization from the UK MHRA and Bahrain NHRA** for certain eligible patients with SCD or TDT in Q4 2023
- **U.S. FDA PDUFA target action date of March 30, 2024, for TDT (Standard Review);** MAA for SCD and TDT filed and validated in the EU
- **CASGEVY could address >30K patients** in the U.S. and EU with severe SCD and TDT if approved, with the **opportunity to expand the market even further** with targeted conditioning and *in vivo* editing

Program	Research	IND-enabling	Clinical	Approved	Status	Partner	Structure
CASGEVY: Severe sickle cell disease (SCD)					Approved in some countries for certain eligible patients	VERTEX	Collaboration
CASGEVY: Transfusion-dependent β-thalassemia (TDT)							
Next-generation conditioning							Wholly-owned[1]
***In vivo* editing of HSCs**							

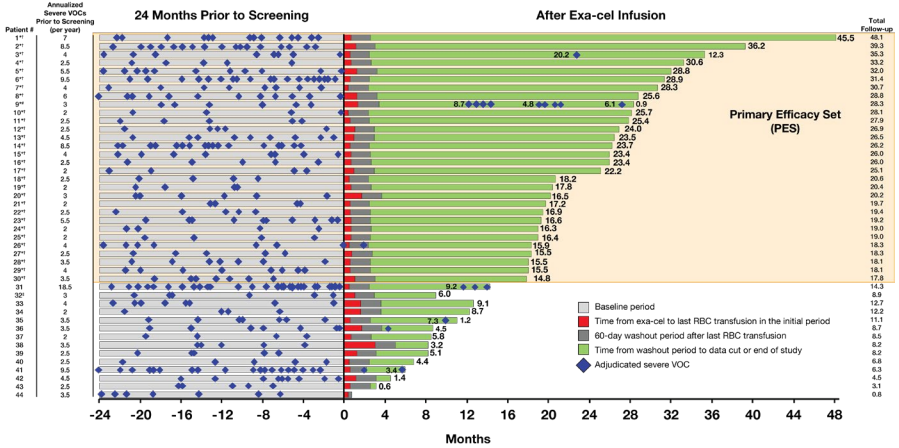
(1) Collaboration with Vertex for applications in SCD and β-thalassemia

Exa-cel: Groundbreaking Data Across >95 Patients

TDT: Transfusion independence achieved out to 45 months



SCD: VOC-free and no in-patient hospitalizations for VOCs achieved out to 45.5 months



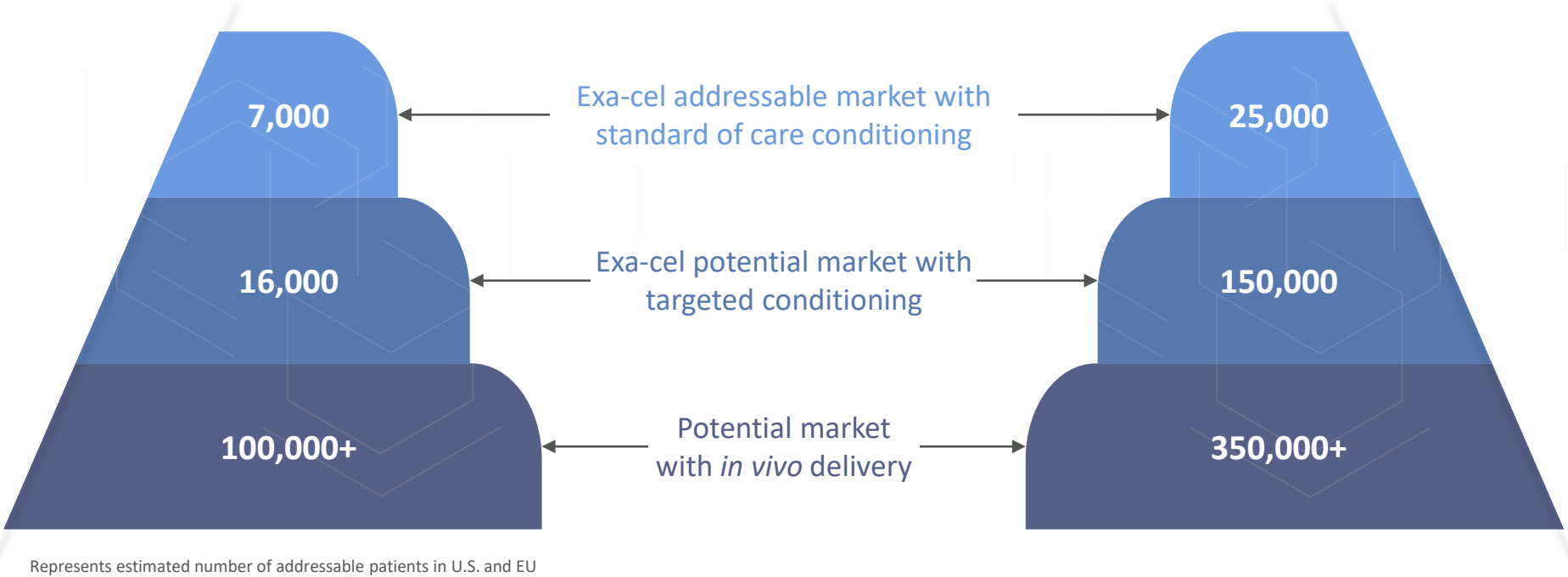
Exa-cel treatment resulted in early and sustained increases in Hb and HbF leading to **transfusion independence (TI12) in 91.4% of patients** with TDT, **elimination of VOCs (VF12) and inpatient hospitalization for VOCs (HF12) in 96.7% and 100% of patients** with SCD, respectively

* Participant evaluable for the primary endpoint; † participant achieved TI12 (TDT) or VF12 (SCD); § participant did not achieve TI12; # participant did not achieve VF12; ‡ Death from respiratory failure due to COVID-19 infection
Hb, hemoglobin; HbF, fetal hemoglobin; HF12, proportion of participants free from inpatient hospitalization for severe VOCs for ≥12 months; RBC, red blood cell; TI12, proportion of patients transfusion independent for 12 consecutive months while maintaining weighted average Hb ≥9 g/dL; VF12, proportion of participants free of severe VOCs for ≥12 months; VOC, vaso-occlusive crisis

Presented at the American Society of Hematology Annual Meeting. 11 Dec 2023

Exa-cel has a Large Addressable Market

Opportunity to broaden market via innovation in conditioning and delivery



Represents estimated number of addressable patients in U.S. and EU

Robust Immuno-Oncology Pipeline

- **Allogenic platform allows immediate "off-the-shelf" dosing,** alleviating the complex supply barriers associated with approved autologous cell therapies
- **CTX112 and CTX131 advancing in the clinic:** Next-generation CAR T candidates with potency edits to improve tumor killing capacity and resistance to suppression, manufactured at internal GMP facility
- **Proof of concept that allogeneic CAR T cells can produce durable complete remissions following a standard lymphodepletion regimen** demonstrated by first-generation programs

	Program		Research	IND-enabling	Clinical	Approved	Status	Partner	Structure
Allo	CD19	CTX112[1]					Enrolling		Wholly owned
	CD70	CTX131					Enrolling		Wholly owned
		Anti-CD70 CAR-NK						nkarta THERAPEUTICS	Collaboration
	Other targets	CTX121 (anti-BCMA)							Wholly owned
		Other CAR T programs							Wholly owned
Auto	Novel targets	Anti-CD83 CAR T						MOFFITT CANCER CENTER	Collaboration[2]
		Anti-GPC3 CAR T						ROSWELL PARK	Collaboration[2]

(1) Initiation of additional trial in systemic lupus erythematosus planned for 1H 2024; (2) CRISPR retains commercial rights

Our Gene-Edited Allogeneic CAR T Franchise

First-generation | **Next-generation with novel potency edits**

Single-dose

PoC that allogeneic CAR T cells can produce durable remissions following a standard LD regimen

Consolidation dosing

Improved 6-month complete response rate

CTX112 and CTX131

Preliminary clinical data suggest next-gen may improve upon the clinical profile of first-gen

- Significantly higher CAR T cell expansion and functional persistence
- Increased manufacturing robustness; manufactured at internal GMP facility
- Opportunity to expedite development based on clinical and regulatory learnings from CTX110 and CTX130

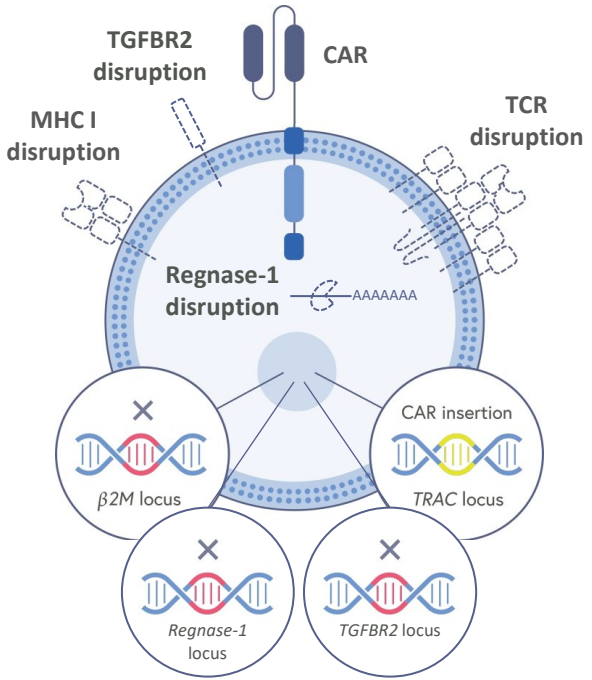
Further indications/targets

Advancing next-gen candidates in new areas, e.g., CTX112 in autoimmune disease, GPC3-targeted autologous CAR T with Roswell Park, and others in the pipeline

CTX112 and CTX131 Incorporate Novel Potency Edits

Next-generation CRISPR gene-edited allogeneic CAR T chassis:

- **MHC I KO:** Improve persistence in the allogeneic setting and avoid need for more toxic lymphodepletion
- **TGFBR2 KO:** Reduce tumor microenvironment inhibition of multiple CAR T cell functions



- **TCR KO:** Prevent GvHD
- **Regnase-1 KO:** Increase functional persistence, cytokine secretion and sensitivity, and effector function
- **CAR KI:** Site-specific insertion into TRAC locus without using lentivirus

CTX112 and CTX131 utilize the same CRISPR-edited allogeneic T cell design, *but CTX112 incorporates a CD19-targeted CAR while CTX131 incorporates a CD70-targeted CAR and knock-out of CD70*

Regnase-1 and TGFBR2 Knock-Outs Work Synergistically

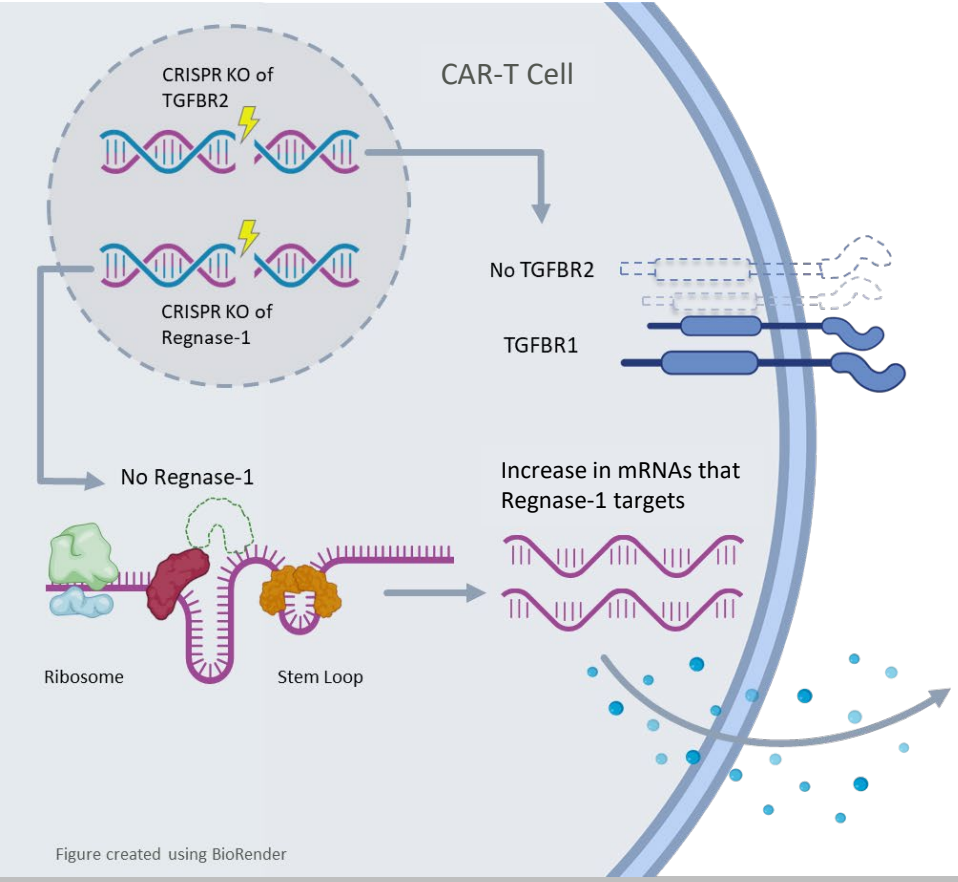
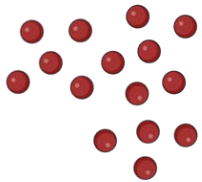


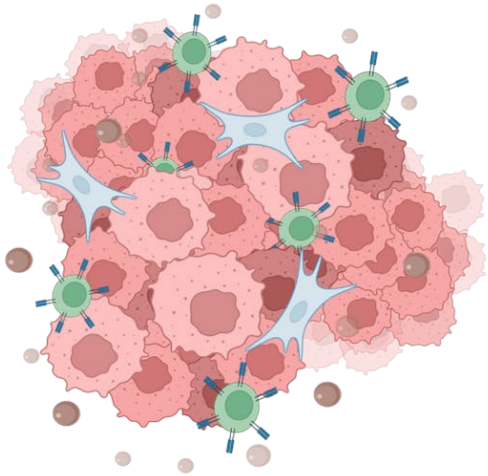
Figure created using BioRender

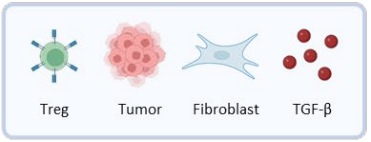
- **No TGF-β mediated inhibition**



- **Increased proliferation**
- **Broad cytokine secretion**
- **Increased cytotoxicity**
- **Repeat response to antigen challenge**

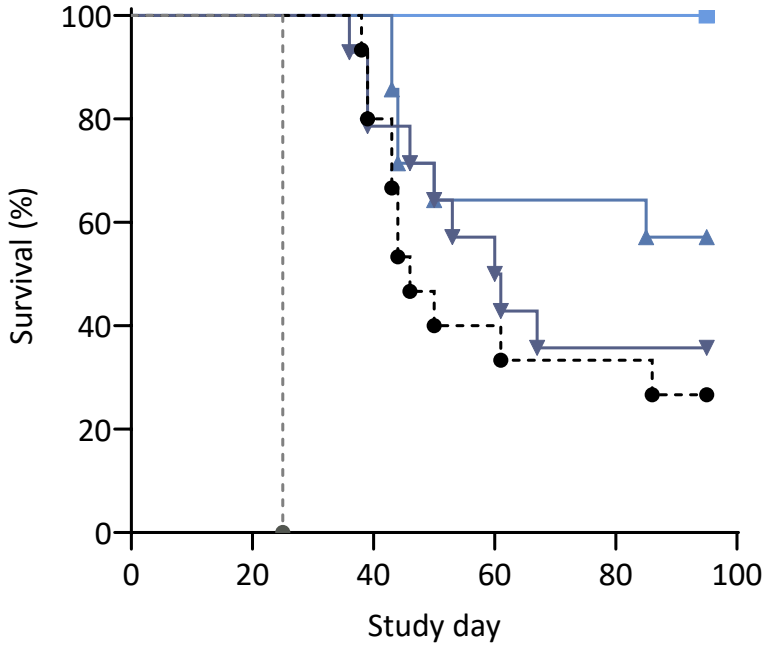
Tumor microenvironment





CTX112: Regnase-1/TGFBR2 KO Enhances Potency

Potency edits in CTX112 lead to extended survival in Nalm6-Luc mice



- CTX112: TGFBR2 KO + Regnase-1 KO
- Regnase-1 KO
- TGFBR2 KO
- CTX110
- Untreated

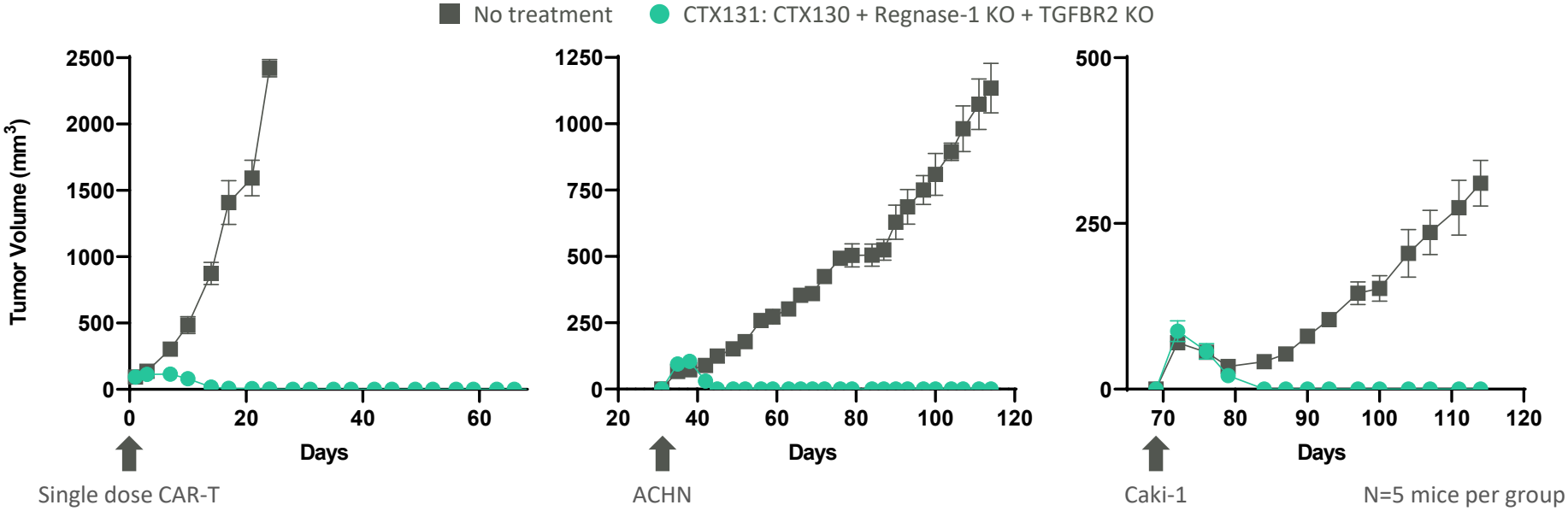
N=15 mice per treated group
N=5 mice for untreated group

Presented at the American Association for Cancer Research Annual Meeting. 16 April 2023

CTX131 Eliminates Three Successive Tumor Models *In Vivo*

CTX131 eliminates three different xenograft tumor models in succession without exhaustion

Tumor 1: *NCI-H1975 (Lung)*	**Tumor 2:** *Rechallenge 1 with ACHN (RCC)*	**Tumor 3:** *Rechallenge 2 with Caki-1 (RCC)*



CTX112 and CTX131 Clinical Trials

	Phase 1/2 safety and efficacy study evaluating CTX112	Phase 1/2 safety and efficacy study evaluating CTX131
Indications	▪ Relapsed or refractory B-cell malignancies ▪ Expanding into autoimmune diseases with planned trial initiation in 1H 2024 in systemic lupus erythematosus (SLE)	▪ Relapsed or refractory solid tumors starting with renal cell carcinoma (RCC) ▪ Expanding into hematological malignancies
LD regimen	▪ Standard lymphodepletion regimen of cyclophosphamide (500 mg/m^2) and fludarabine (30 mg/m^2) for 3 days	

Allogeneic CAR T enables simplified trial design with short screening timeframe, no apheresis, no bridging chemotherapy, and on-site availability of CAR T cell product

Collaborations with Top Cancer Centers on New Targets

Clinical trial to begin in next 12 months

- **First-in-human trial for autologous CAR T therapy targeting CD83**
- **CD83:** Expressed on certain cancers and activated T cells – potential in AML and other oncology and autoimmune indications
- Additional research in collaboration with the Masonic Cancer Center, University of Minnesota

IND-enabling studies to begin this year

- **Initial trial for gene-edited, autologous CAR T therapy targeting GPC3**
- **GPC3:** Solid tumor target for hepatocellular carcinoma (HCC) with limited expression in healthy tissues – potency edits have potential to enhance CAR-T activity against solid tumors

Cancer centers conduct viral vector manufacturing, cell manufacturing, and Phase I trial
CRISPR retains commercial rights

CRISPR Enables Regenerative Medicine 2.0

- **CRISPR gene editing and pluripotent stem cell technology enable a new class of cell replacement therapies**
- **Developing a beta-cell replacement product that aims to treat diabetes without requiring immunosuppression in partnership with ViaCyte** – gene editing key to achieve this goal
- **Clinical trial initiated for VCTX211, which includes novel edits to promote cell survival** – CRISPR platform enables continuous innovation with next-generation products incorporating incremental edits to increase benefit

Program	Research	IND-enabling	Clinical	Approved	Status	Partner	Structure
VCTX210: Type I diabetes mellitus							
VCTX211: Type I diabetes mellitus					Enrolling	VIACYTE®	Collaboration
VCTX212: Type I/II diabetes mellitus							

Multi-staged Product Strategy

Perforated Device Approach
- Progenitor cells (stage 4)
- Retrievable, enabling broader initial patient population

Deviceless approach
- Immature β-cells (stage 6)
- Portal vein injection

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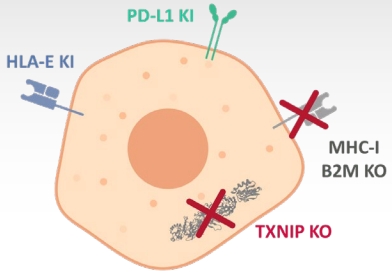
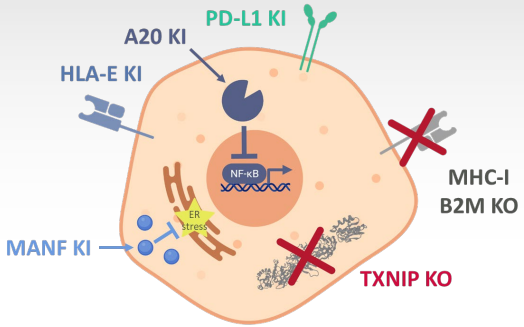


- Entered clinic Nov 2021
- Safety and immune evasion
- Informs 211 trial design

211



- Two additional edits to promote cell survival
- CTA cleared in 2H 2022

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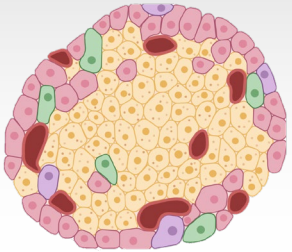
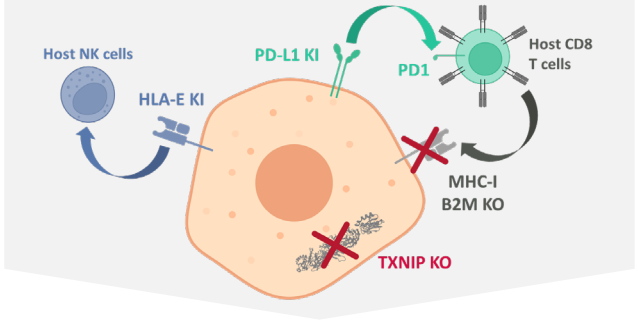


- Unencapsulated, stage 6 cell aggregates containing additional edits beyond 211
- Research stage program

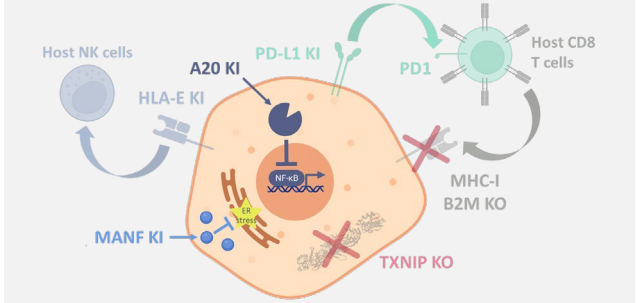
VCTX211: Further Optimized for Cell Fitness

VCTX211 has 2 gene KOs and 4 insertions to improve functionality

VCTX210



VCTX211



Immune evasion

- **MHC-I KO** eliminates T cell mediated rejection
- **PD-L1 KI** reduces immune rejection, particularly from T cells
- **HLA-E KI** further reduces immune rejection, particularly from NK cells

Cell fitness

- **Thioredoxin interacting protein (TXNIP) KO** protects from oxidative and ER stress

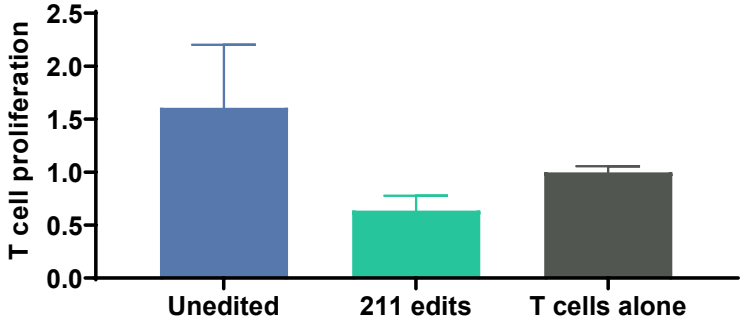
+

- **A20 (TNFAIP3) KI** induces graft acceptance and protection from cytokine induced apoptosis
- **MANF KI** enhances β cell proliferation and protection against inflammatory stress

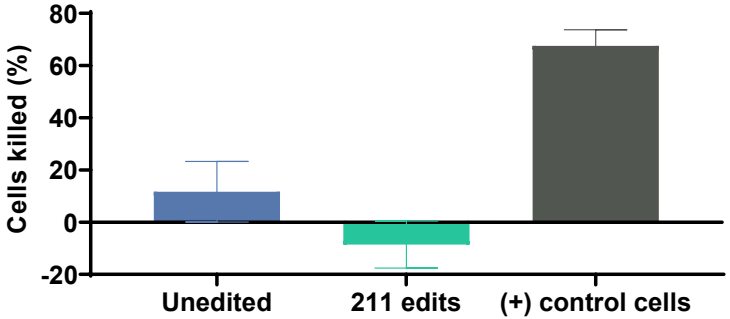
Sources: Qian, et al. *Immunology*. 1996; 88(1):124-9. Gornalusse, et al. *Nat Biotechnology.* 2017;35(8):765-72. El Khatib, et al. *Gene Therapy*. 2015;22(5):430-8. Chen, et al. *FASEB J.* 2008;22(10):3581-94. Shalev. *Biochem Soc Trans*. 2008;36(5):963-5. Lindahl, et al. *Cell Rep.* 2014;24(7):366-75. Zammit, et al. *JCI Insight.* 2019;4(21)

Edited Cells Evade Immunity *In Vitro* and *In Vivo*

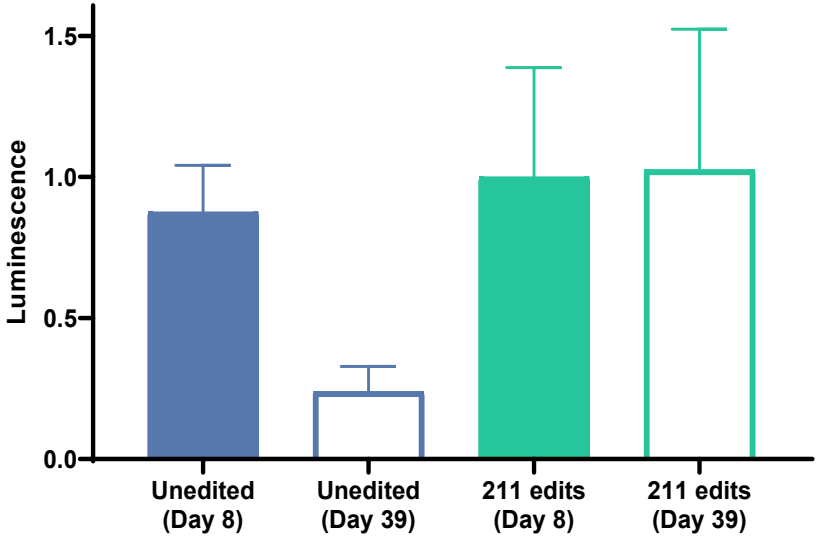
Adaptive – T cells do not respond to 211 cells *in vitro*



Innate – 211 cells resist NK attack *in vitro*



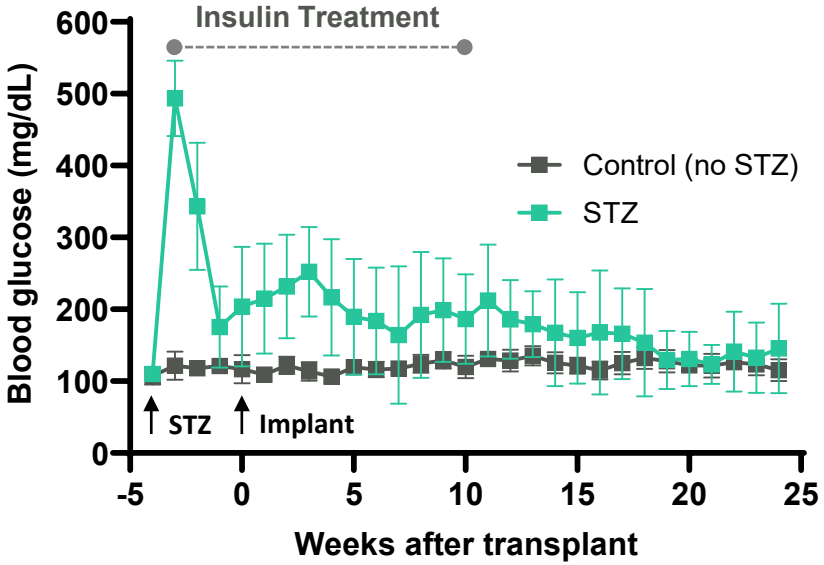
Adaptive & Innate – 211 cells survive in humanized mouse model



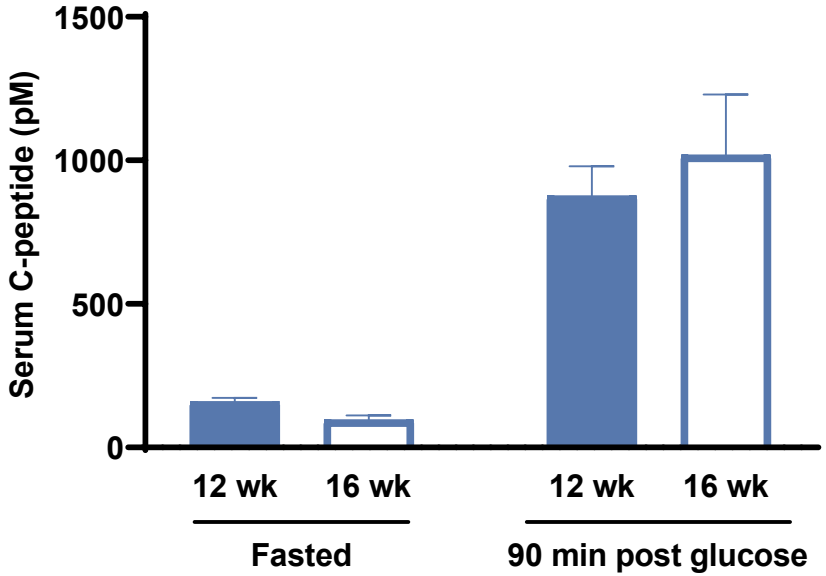
Demonstrates broad immune evasive potential of 211 cells – humanized mouse model contains human DC, B cells, T cells, NK cells, and monocytes

VCTX211 Reverses Hyperglycemia in Diabetic Rat Model

Normalization of blood glucose by 12-16 weeks



Treated rats maintain glucose sensitivity



Rats either treated with STZ ~4 weeks before VCTX211 implantation or untreated (normoglycemic control)

STZ: Streptozotocin (β-cell toxin)

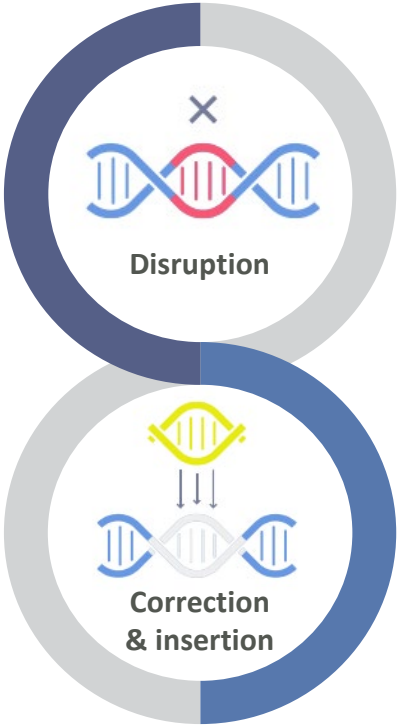
In Vivo Platform Advancing Rapidly

- **90% of the most prevalent severe monogenic diseases only addressable with gene disruption and/or whole gene correction**
- **Established plug-and-play LNP/mRNA platform for *in vivo* gene disruption,** starting in the liver
- **Developing a multi-modal whole gene correction platform,** starting with AAV+LNP in the liver and advancing to AAV-free, HDR-independent methodologies
- **Advancing a broad portfolio across both rare and common diseases** leveraging our translational capabilities and balance sheet

	Program		Research	IND-enabling	Clinical	Approved	Partner	Structure
LNP	Disruption or deletion	CTX310: ANGPTL3						Wholly-owned
		CTX320: Lp(a)						Wholly-owned
		CTX330: PCSK9						Wholly-owned
		Undisclosed CV programs						Wholly-owned
		Other gene disruption programs						Wholly-owned
		Undisclosed ocular program					BAYER	Collaboration
	Insertion	Hemophilia A						
		Undisclosed insertion program						Wholly-owned
AAV	Disruption or deletion	Friedreich's ataxia (FA)					CAPSIDA BIOTHERAPEUTICS	Collaboration
		Amyotrophic lateral sclerosis (ALS)						

Partnered with Vertex on several additional disease areas, including Duchenne muscular dystrophy (DMD), myotonic dystrophy type 1 (DM1), and cystic fibrosis (CF)

Becoming an *In Vivo* Leader – Our Strategy

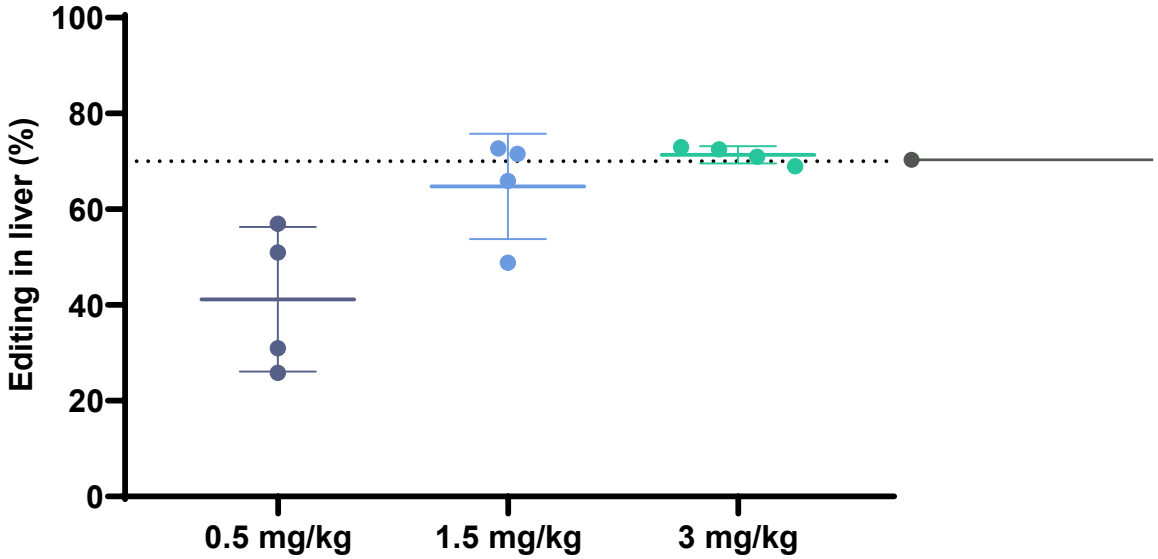


Focus on disruption and whole gene correction – needed to address ~90% of the most prevalent severe monogenic diseases

- **Establish a leading platform for *in vivo* gene disruption,** starting in the liver
- **Advance a broad portfolio of programs across both rare and common diseases,** leveraging our translational capabilities, balance sheet, and plug-and-play LNP/mRNA platform
  - Targets/indications include ANGPTL3, Lp(a), PCSK9, HAE, TTR, PH1, and other undisclosed ocular and liver targets
  - Wholly-owned portfolio creates opportunity for internal development or partnership
- **Develop leading whole gene correction platform,** starting with AAV+LNP in the liver and advancing to AAV-free, HDR-independent methodologies

Established a Leading mRNA/LNP Platform for Gene Disruption

Dose-dependent liver editing up to 70% in NHPs



70+% editing in whole liver typically equates to 90+% hepatocyte editing and reduction in serum protein levels

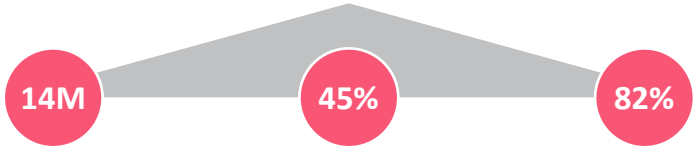
Single intravenous dose of LNP formulated with Cas9 mRNA and gRNA

Our Initial *In Vivo* Programs Could Transform the Treatment Paradigm for ASCVD

The chronic care model

Daily medications | Weekly injections | Multiple infusions annually | Surgical interventions

Heavy burden for patients and the healthcare system



people with very high CV risk[1,2] in U.S. and Europe, including **~4M** with genetic dyslipidemias[3,4]

not adherent to lipid-lowering therapy despite prior major CV events[5]

not at LDL-C target goal[2]

A new treatment paradigm

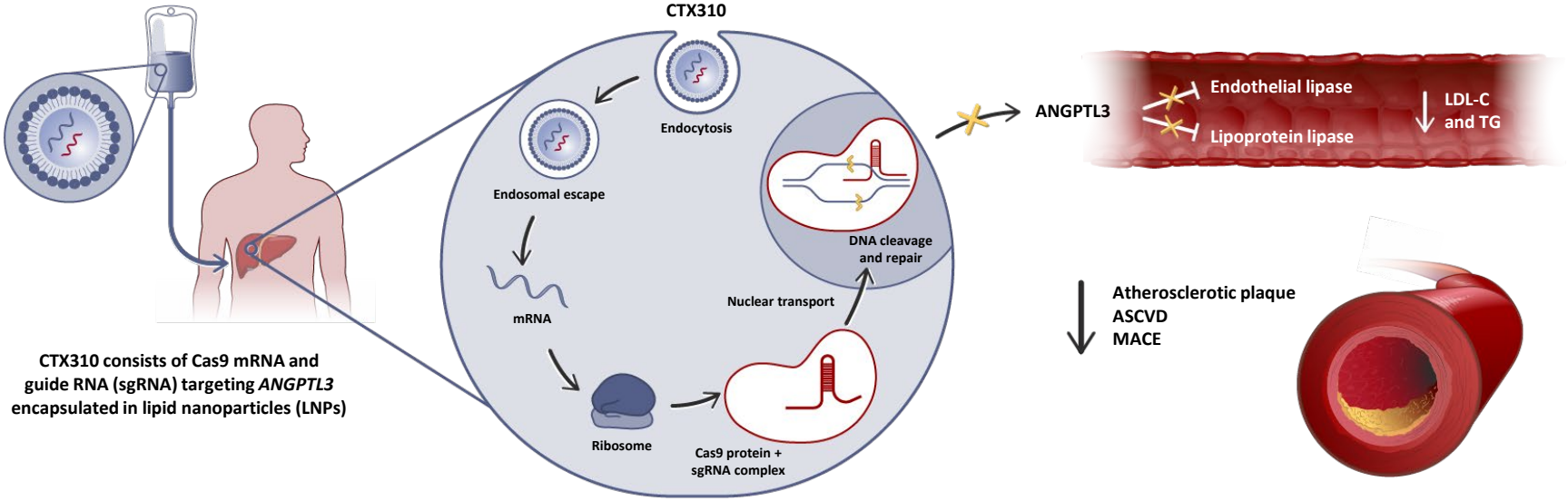
One-time CRISPR-based therapies with the potential to:

- Recapitulate the proven benefit of targets like ANGPTL3, as validated by natural human genetics and other therapeutic modalities
- Improve long-term cardiovascular outcomes by durably lowering atherogenic lipoproteins for a patient's lifetime
- Minimize or eliminate the need for additional treatments
- Treat both severe disease and much larger ASCVD patient populations

ASCVD: atherosclerotic cardiovascular disease; (1) Gu et al. 2022; (2) Ray et al. 2021; (3) Hu et al. 2020; (4) Dron et al. 2018; (5) Guglielmi et al. 2017

CTX310: A One-Time Dose to Stop Expression of ANGPTL3

Intravenous delivery targeting the liver → **CRISPR/Cas9-based editing of *ANGPTL3*** → **Reduced atherogenic lipoprotein concentrations**



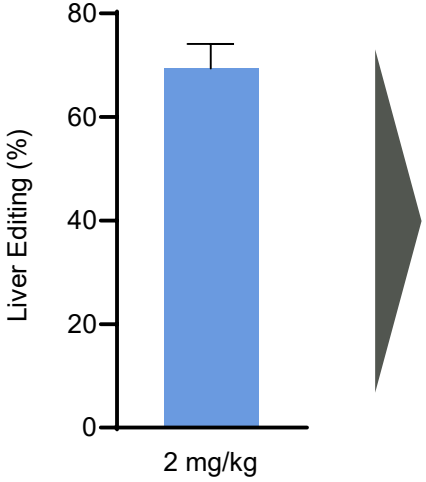
CTX310 consists of Cas9 mRNA and guide RNA (sgRNA) targeting *ANGPTL3* encapsulated in lipid nanoparticles (LNPs)

Transient expression of Cas9 and sgRNA in hepatocytes to reduce ANGPTL3 expression permanently

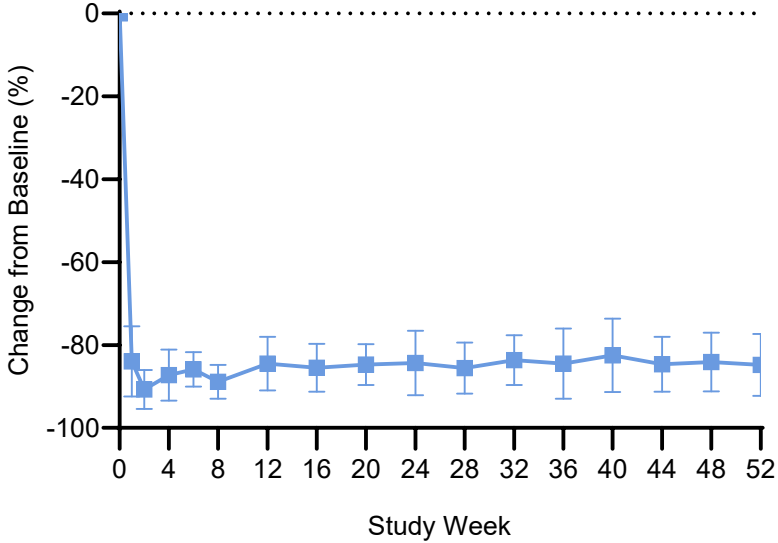
Clinical trial of CTX310 initiated

A Single Dose of CTX310 Resulted in Durable Reduction in ANGPTL3 and Triglycerides in Non-Human Primates

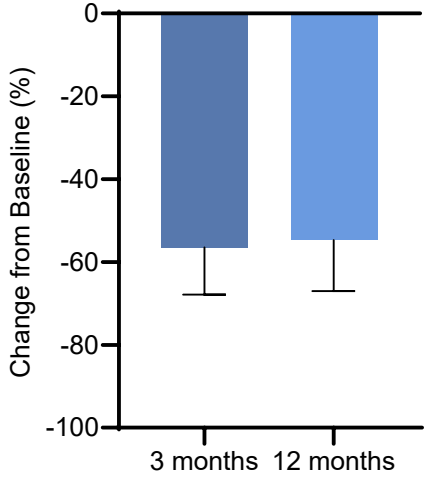
~70% editing of *ANGPTL3* at 1 year



Sustained reduction in plasma ANGPTL3 at 1 year



Sustained reduction in TG at 1 year



Single dose of CTX310 (2 mg/kg) administered to NHPs (N=8) on Day 1; dose levels reflect mg total RNA; study ongoing

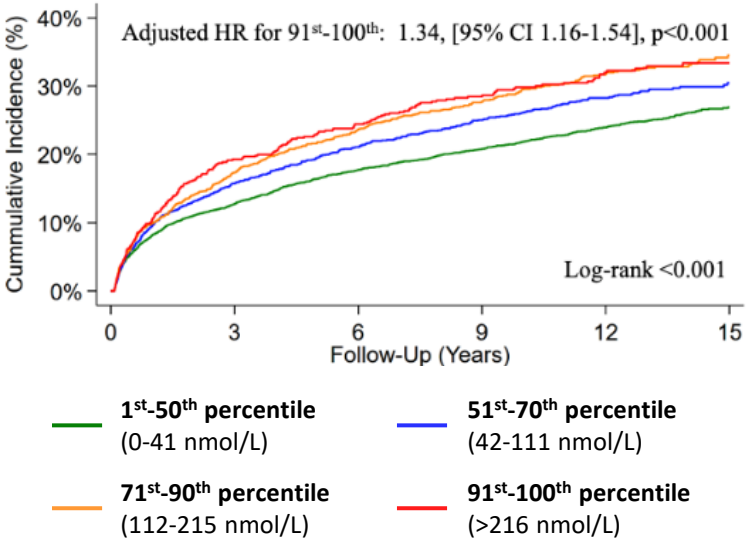
Presented at the American Heart Association Scientific Sessions. 11 Nov 2023

Lp(a): An Independent Risk Factor for ASCVD

- **Lipoprotein(a):** an LDL-like lipoprotein synthesized and secreted by hepatocytes that contains apo(a) bound to ApoB
- **The *LPA* gene encodes apo(a) and determines plasma Lp(a) levels**
- **Epidemiologic studies, Mendelian randomization, and GWAS have shown that elevated Lp(a) levels increase ASCVD risk**, whereas those with low Lp(a) levels (~12.5 nmol/L) have better cardiometabolic outcomes, e.g., 29% reduced risk of coronary heart disease, 37% reduced risk of aortic valve stenosis[1,2,3,6,7,8]
- **>20% of the global population have elevated circulating Lp(a)** concentrations above ~125 nmol/L[4,5]

A one-time, CRISPR-based therapy could recapitulate the protective effect of naturally low Lp(a) levels

Independent association with long-term MACE[8]

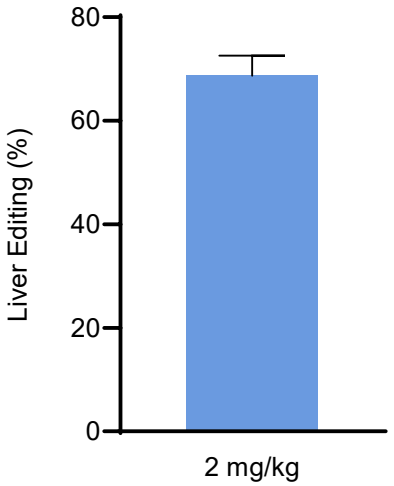


Association of Lp(a) with MACE among individuals with a history of ASCVD, adjusted for age, sex, self-reported race and ethnicity, hypertension, chronic kidney disease status, non-Lp(a) hyperlipidemia, diabetes, insulin use (in diabetic individuals), and smoking status; N=10,181

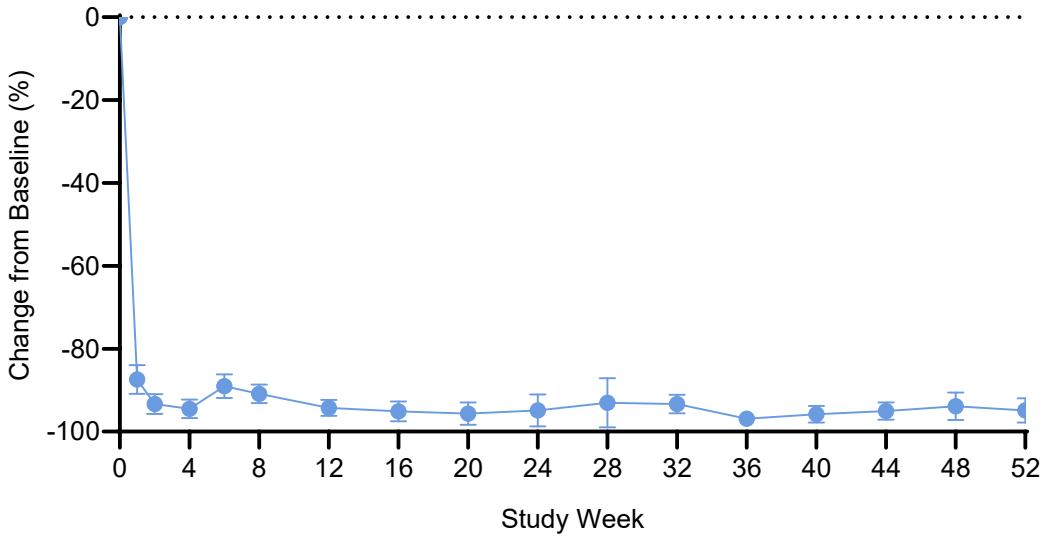
(1) Enas et al. 2019; (2) Gurdasani et al. 2012; (3) Laschkolnig et al. 2014; (4) Nordestgaard et al. 2010; (5) Varvel et al. 2016; (6) Langsted et al. 2021; (7) Emdin et al. 2016; (8) Berman et al. 2023

A Single Dose of CTX320 Resulted in Durable Lp(a) Reduction in Non-Human Primates

~70% editing of *LPA* at 1 year



~95% reduction in plasma Lp(a) sustained at 1 year

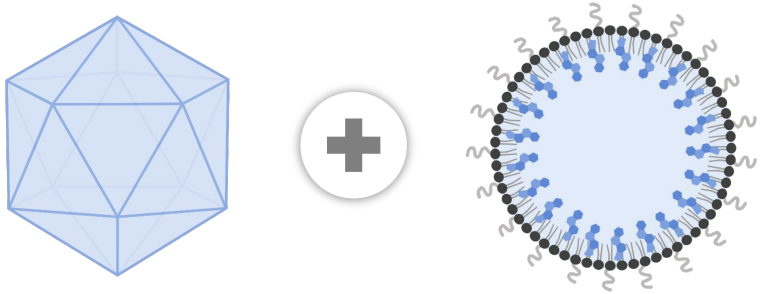


Single dose of CTX320 (2 mg/kg) administered to NHPs (N=4) on Day 1; study ongoing

Presented at the American Heart Association Scientific Sessions. 11 Nov 2023

Unlocking Whole Gene Correction and Insertion

AAV + LNP



- Proven technologies allow whole gene correction via repair mechanisms at specific loci
- Potential for improved consistency and durability compared to episomal gene transfer via AAV
- Ability to address majority of monogenic diseases, where mutations span the length of the gene

Next-generation technologies

CRISPR-X

- Dedicated internal group focused on emerging technologies to allow HDR-independent and/or AAV-free whole gene correction/insertion
- Natural systems require further optimization of efficiency and specificity for clinical application
- Research ongoing focused on non-viral DNA delivery and all-RNA systems

Strong U.S. and Global Foundational IP Position

United States

CVC granted patents of broad scope; multiple applications progressing

- **55** Patents of broad scope granted
- **16** Additional patent applications moving forward in parallel with both broad and narrow claims
- PTAB decision in CVC/Broad interference appealed to the CAFC; interferences between CVC/Sigma, CVC/ToolGen, Broad/Sigma and Broad/ToolGen put “on hold” by PTAB

Europe and Global

CVC granted foundational patents, including use in eukaryotes

- **46** Patents of broad scope granted in the EU, Canada, China, Japan, Brazil, Mexico, Singapore, Hong Kong, Ukraine, Israel, UAE, Australia, New Zealand, South Africa, etc.
- **~80** Jurisdictions worldwide in which CVC has patent protection
- In August, CVC prevailed against ToolGen’s challenge to CVC’s Japanese patent; challenges pending in China and India

CVC: Charpentier, University of California, and University of Vienna

As of Q3 2023

Building a Great Company