cellectis

EDITING LIFE

Therapeutic Gene Surgery

CORPORATE PRESENTATION - NOVEMBER 2026



Forward Looking Statements

This presentation contains “forward-looking” statements within the meaning of applicable securities laws, including the Private Securities Litigation Reform Act of 1995. Forward-looking statements may be identified by words such as “design”, “expect”, “initiate”, “into”, “moving forward”, "objectives," “plan”, “potential”, “targeted”, "targeting," “will”, or similar expressions and/or the negative of these. These forward-looking statements are based on our management's current expectations and assumptions and on information currently available to management, including information provided or otherwise publicly reported by our licensed partners. Forward-looking statements in this presentation include statements regarding the Company's transition into an in vivo gene editing company, the ability of the Company to initiate an IIT in China; the timing of submission of regulatory filings, the planned timing for the first injection in human of our lead product candidates, the planned timing for the release of preliminary clinical data for the Company's lead programs, the potential for success of the Company's in vivo gene editing programs, the expected design of the clinical development of the Company's lead programs, the ability to continue advancement of cell therapy activities through existing partnerships, the anticipated impact, including potential cost savings, of the Company's decision to cease development of lasme-cel and eti-cel and to initiate a restructuring plan, the contemplated implementation of headcount reductions and other cost-savings initiatives and their planned timeframe, and the targeted extension of the Company's cash runway. Actual results, performance or events may differ materially from those projected in any forward-looking statement. Many important factors may adversely affect such forward-looking statements and cause actual results to differ from those in any forward-looking statement, including, without limitation, uncertainties inherent in the initiation and completion of preclinical and clinical studies; the performance of the IIT in China; the availability and timing of results from studies, once initiated; the acceptance by global regulatory agencies of data generated from IITs in China; the potential that pre-clinical animal model data may not translate into further preclinical development or clinical setting; promising preclinical data not yielding positive further preclinical data or clinical results; changes in the competitive landscape for *in vivo* gene editing products; expectations for regulatory approvals to conduct clinical trials; availability of funding sufficient for the Company's foreseeable and unforeseeable operating expenses and capital expenditure requirements; and impacts of the Company's headcount reductions and other cost savings initiatives, which may include operational and strategic challenges. With respect to our cash runway, our operating plans, including product development plans, may change as a result of various factors, including factors currently unknown to us. Furthermore, many other important factors, including those described in our Annual Report on Form 20-F as amended and in our annual financial report (including the management report) for the year ended December 31, 2025 and subsequent filings Cellectis makes with the Securities Exchange Commission from time to time, which are available on the SEC's website at www.sec.gov, as well as other known and unknown risks and uncertainties may adversely affect such forward-looking statements and cause our actual results, performance or achievements to be materially different from those expressed or implied by the forward-looking statements. Except as required by law, we assume no obligation to update these forward-looking statements publicly, or to update the reasons why actual results could differ materially from those anticipated in the forward-looking statements, even if new information becomes available in the future.


OUR MISSION
Life, no longer defined by disease
26 YEARS OF GENE EDITING

Cellectis at a Glance



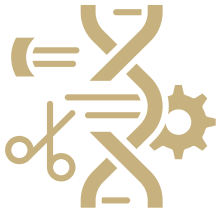
STRATEGIC REFOCUS *IN VIVO* GENE EDITING

- Exiting *ex vivo* cell therapy programs
- Continuing existing collaborations with AstraZeneca, Allogene, Servier and Iovance



LEAD PROGRAMS

- **.HEAL-101 (APOC3)**
  Severe hypertriglyceridemia
- **.HEAL-201 (PCSK9)**
  Severe hypercholesterolemia



OUR GENE EDITING PLATFORM

- A differentiated platform
- Single-base precision
- A comprehensive gene editing approach



RUNWAY INTO H2 2028

- Realignment of the operating model targeting cash runway extension into H2 2028
- Cash position*: $149m as of September 30, 2026

* Cash position i.e. cash, cash equivalents and fixed-term deposits include restricted cash of $2.3 million, classified as current and non-current financial assets, and fixed-term deposits of $118.3 million, classified as current financial assets.

A New Modality on a Platform 26 Years in the Making

26

YEARS OF GENE EDITING

Gene editing was pioneered by Cellectis

A deep understanding of editors and DNA programming

LICENSED PARTNERS

Allogene THERAPEUTICS

AstraZeneca

SERVIER moved by you

IOVANCE BIOTHERAPEUTICS

3 licensed programs in clinical development

TALEN® is a registered trademark owned by Cellectis

sHTG: A Leading Cause of Acute Pancreatitis

Severe hypertriglyceridemia (sHTG)

A chronic lipid metabolism disorder in which triglyceride-rich particles build up in the blood. Above 500 mg/dL, they can injure the pancreas and cause acute pancreatitis

~4.3M

adults in the US, EU4 and UK[1]

TG ≥500 mg/dL

TARGET POPULATION

~1.1M

high-risk population[2]

WHAT DEFINES THE HIGH-RISK TARGET GROUP

TG 500-879 mg/dL + prior acute pancreatitis

An episode has already occurred, and recurrence is frequent

TG ≥880 mg/dL

Triglycerides high enough to drive the risk of acute pancreatitis on their own

1. Prevalence of non-FCS sHTG in the US, UK and EU4 (Germany, France, Italy and Spain); estimates based on NHANES and published European epidemiology studies. 2. High-risk no FCS sHTG population defined as TG ≥880 mg/dL, plus TG 500–879 mg/dL with prior acute pancreatitis, using US claims/EHR and published European epidemiology data.
TG: Triglycerides

sHTG: Significant Burden Despite Conventional Treatment

Severe hypertriglyceridemia (sHTG)

ACUTE PANCREATITIS

3x
increased risk of pancreatitis[1]

4x
higher risk of intensive care unit admission[1]

65% of patients remained above target despite lipid-lowering therapy[2]

1. Kiss L et al. Sci Rep. 2018;8:14096. doi: 10.1038/s41598-018-32337-x. PubMed . 2. Marston NA et al. N Engl J Med. 2026;394:429–441. doi: 10.1056/NEJMoa2512761

HeFH: A Major Driver of Premature Cardiovascular Disease

Severe hypercholesterolemia (sHC)

A chronic lipid metabolism disorder in which LDL-C builds up in the blood. Prolonged exposure drives atherosclerosis and cardiovascular disease

~12M

adults in the US, EU4 and UK[1]

LDL-C ≥190 mg/dL

TARGET POPULATION

~2.3M

HeFH adults[2]

WHAT DEFINES THE TARGET GROUP

Inherited condition

LDL-C is elevated from birth, exposure accumulates for life, so must treat, to reduce cardiovascular risk

1. Cellectis estimate, derived from published prevalence of LDL-C ≥190 mg/dL (Sayed A et al. *JAMA Cardiol.* 2023; NHANES 1999–2020) applied to US, UK and EU4 adult populations (US Census Bureau, Eurostat, ONS). US prevalence applied to European populations 2. US claims/NHANES and country-specific epidemiology sources for UK and EU4 (Germany, France, Italy and Spain). 3. Sayed A, Navar AM, Slipczuk L, et al. Prevalence, Awareness, and Treatment of Elevated LDL Cholesterol in US Adults, 1999–2020. JAMA Cardiol. 2023. doi:10.1001/jamacardio.2023.3931

HeFH: Heterozygous Familial Hypercholesterolemia ; LDL-C: Low-Density Lipoprotein Cholesterol

HeFH: High Cardiovascular Risk, Poor LDL-C Control Despite Treatment

Heterozygous Familial Hypercholesterolemia (HeFH)

CARDIOVASCULAR EVENTS

10-20x

increased risk of atherosclerotic cardiovascular disease.[1] [2]

86%

of reported CV events in patients not achieving LDL-C target [3]

~97%

of treated HeFH patients do not meet LDL-C goals despite lipid lowering therapies [4]

1.Khera AV.et al.J Am CollCardiol. 2016 Jun 7; 67(22):2578-89 2. Tada Het al. EurHeart J. 2017 21;38:1573 3. 1579 Blumenthal RS et al. 2026 ACC/AHA Guideline on the Management of Dyslipidemia. J Am Coll Cardiol. 2026 3. Vrablik M et al. Atherosclerosis. 2018;277:355–361. 4. EAS Familial Hypercholesterolaemia Studies Collaboration (FHSC). Lancet. 2021; 398 (10312): 1713–1725

CV: cardiovascular ; LDL-C: Low-Density Lipoprotein Cholesterol

Two Validated Targets, Two Clear Paths to the Clinic

.HEAL-101 *APOC3*

APOC3 reduction	→	TG lowering	→	Lower risk of pancreatitis
Target engagement		*Validated registration endpoint*		*Clinical benefit*

.HEAL-201 *PCSK9*

PCSK9 reduction	→	LDL-C lowering	→	Reduced cardiovascular events
Target engagement		*Validated registration endpoint*		*Clinical benefit*

PROOF-OF-CONCEPT

.HEAL-101: −76% TG in hAPOC3 mice
.HEAL-201: **>90%** PCSK9 reduction in liver-humanized mice

TARGET VALIDATION

PCSK9 and APOC3 are two strongly validated targets

TG: Triglycerides; LDL-C: Low-Density Lipoprotein Cholesterol

Our Approach: One Injection with a Potential Long-Lasting Effect

ORAL THERAPIES

Oral therapies: often not enough on their own



Daily pills, long-term

INJECTABLE THERAPIES

Used alongside or instead of oral therapies



Repeated injections

OUR APPROACH

One injection

.HEAL-101 Edits a single base of the *APOC3* gene



.HEAL-201 Silences *PCSK9*, no DNA sequence change

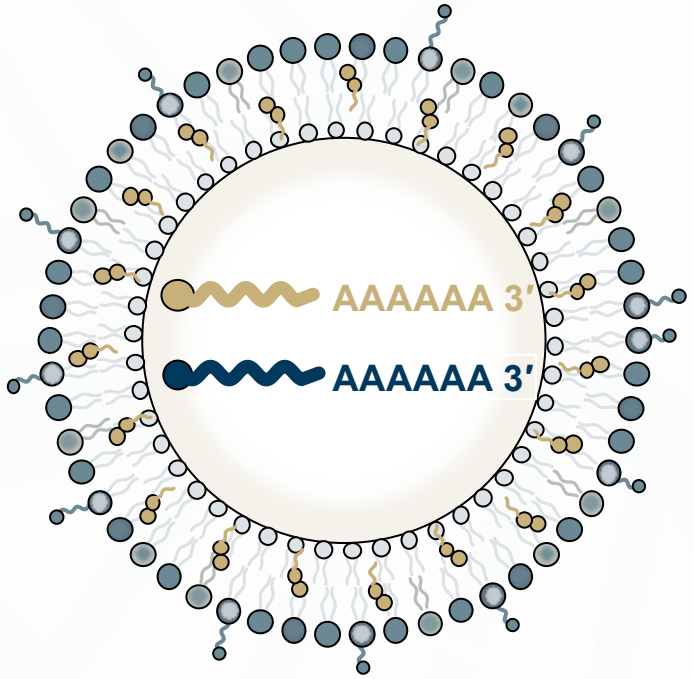


Potential long-lasting effect

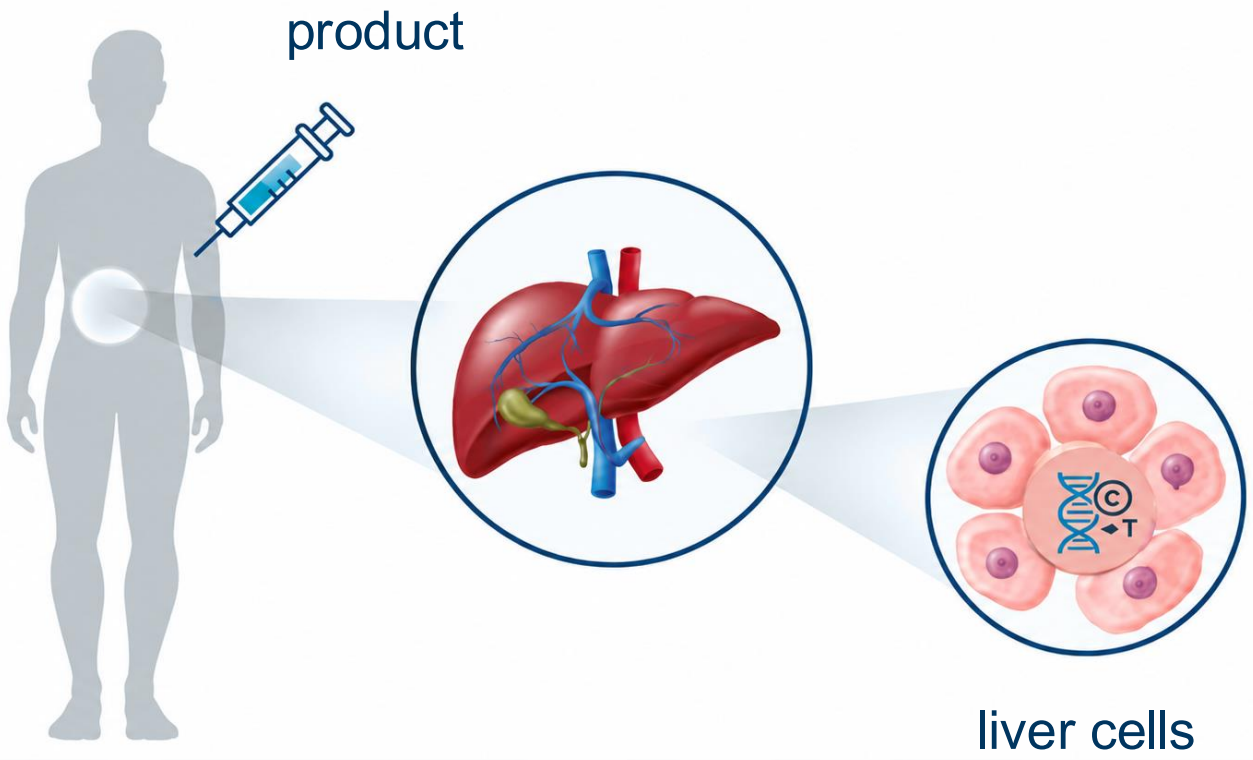


Illustrative. Based on approved dosing of the most widely used therapies: oral statins and fibrates once daily (atorvastatin 10–80 mg once daily; fenofibrate 54–160 mg once daily); injectables (olezarsen once monthly; evolocumab and alirocumab every 2 to 4 weeks). US prescribing information. Fading symbols illustrate patients who discontinue treatment over time.

How It Works: From IV Injection to Liver Cells



Product: mRNA coding for a gene editor in a lipid nanoparticle (LNP)

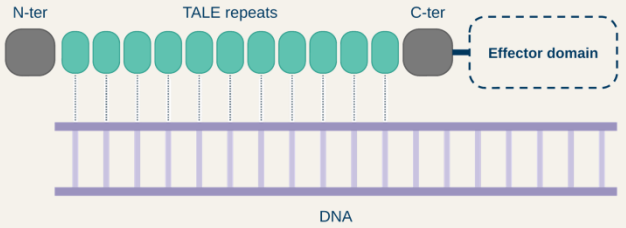


A single IV injection for a potential long-lasting effect

IV: Intravenous

One Gene Editing Platform, Multiple Editing Modalities

One TALE DNA-binding domain



Protein-based DNA recognition, no guide RNA needed

TALEN®	**NUCLEASE EDITING**	**USED FOR ALLOGENEIC CAR-T**
TALEB	**BASE EDITING**	**.HEAL-101**
TALEM	**EPIGENETIC EDITING**	**.HEAL-201**

26 years
of gene editing expertise

Intellectual Property
TALE-based technology

No DNA break
in both lead programs

TALEN® is a trademark owned by Cellectis

.HEAL-101

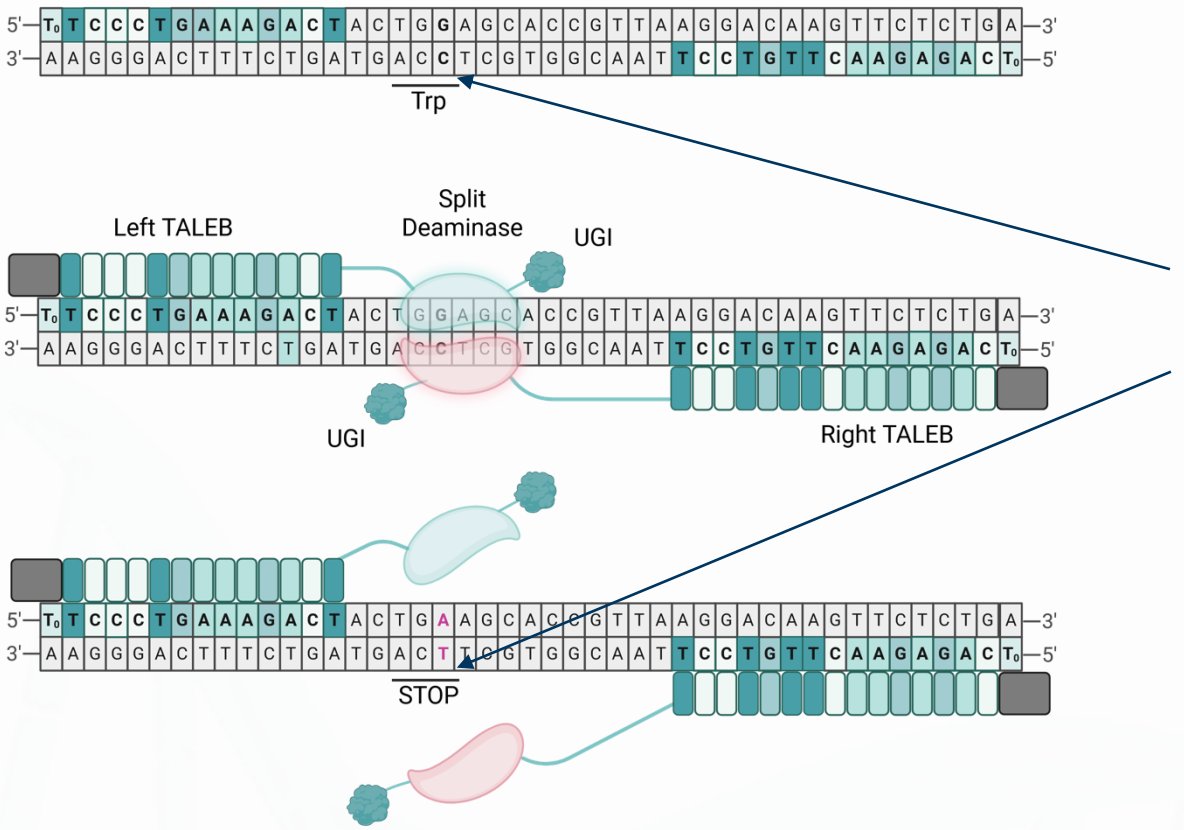
GENE SURGERY

For Severe Hypertriglyceridemia (sHTG)

.HEAL-101: A Single Base Change, No DNA Break

.HEAL-101 is a base editor

It modifies one base in DNA



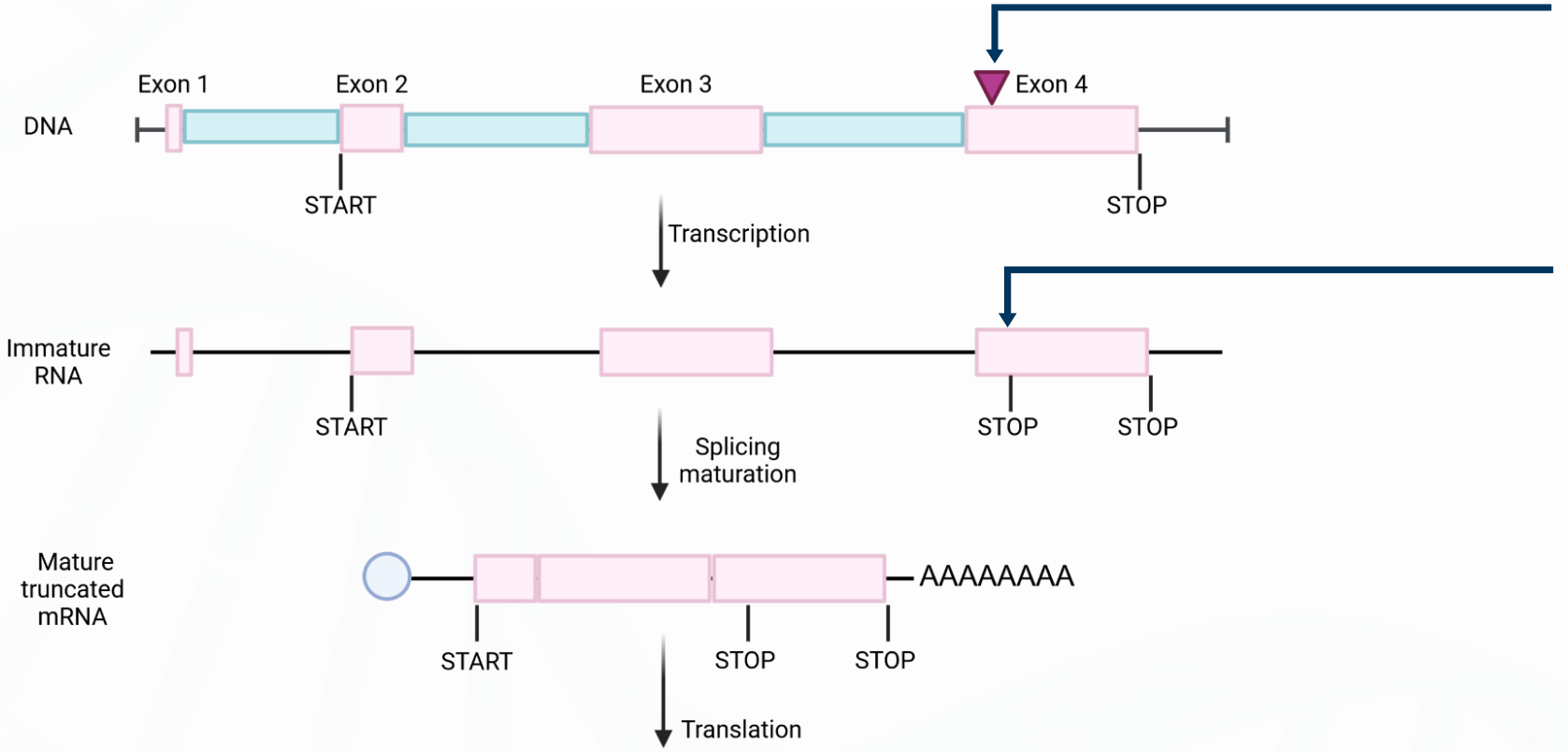
C to T conversion

.HEAL-101

No DNA break offering the potential of an improved safety profile

.HEAL-101: Disables *APOC3* Expression

APOC3 gene expression



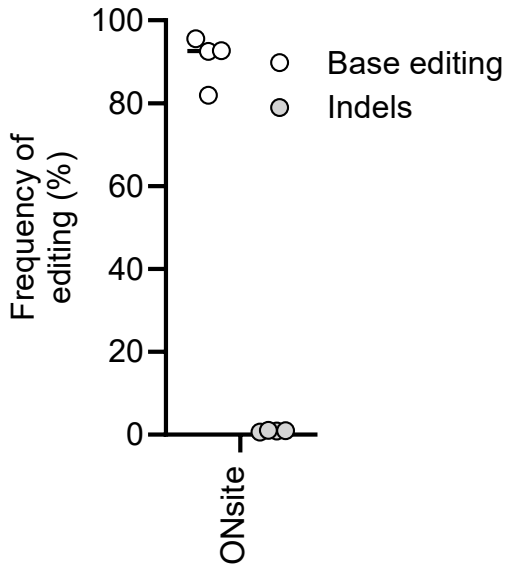
Undetectable APOC3 protein

.HEAL-101: Efficient Editing, No Detectable Off-Target Editing

.HEAL-101 mRNA is introduced in hepatic cell lines

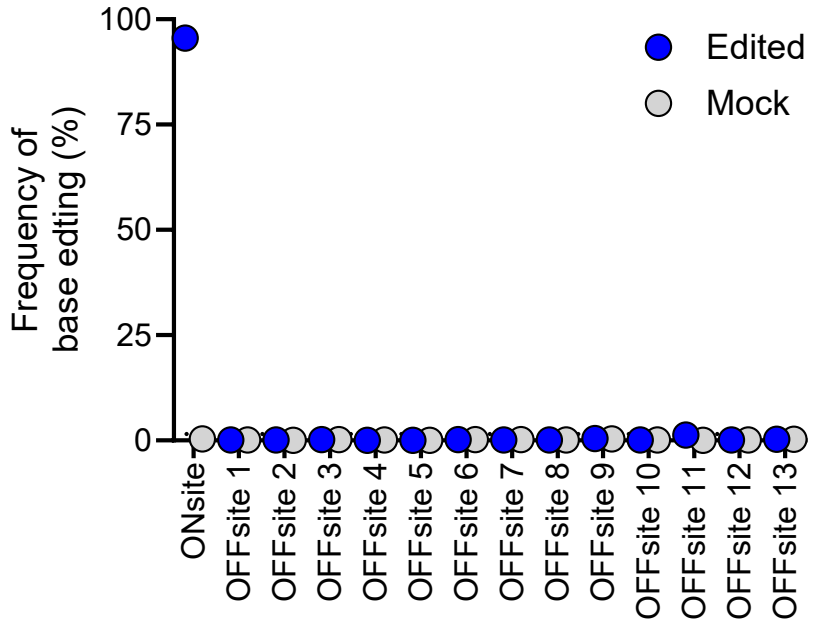
Efficient on-site modification with no DNA alteration

Efficient On-Target Base Editing
~90% with minimal indels



No modification of top putative off-sites

No Detectable Editing at Putative Off-Target Sites

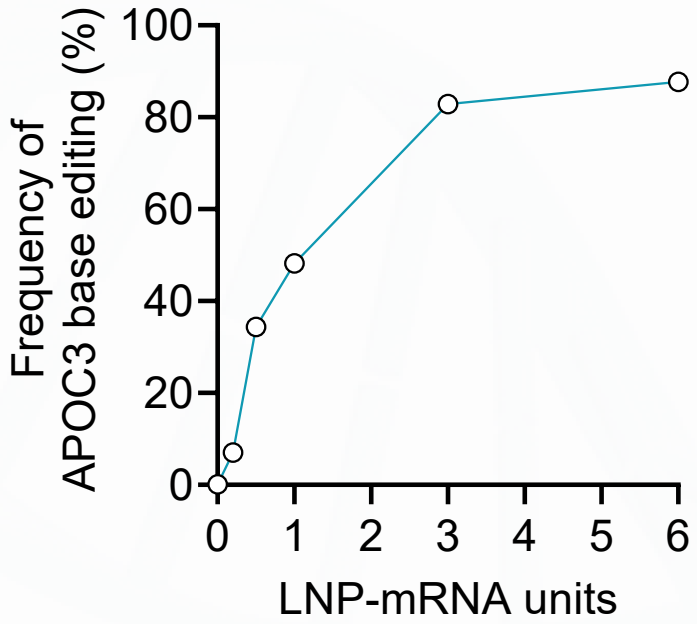


Research Data

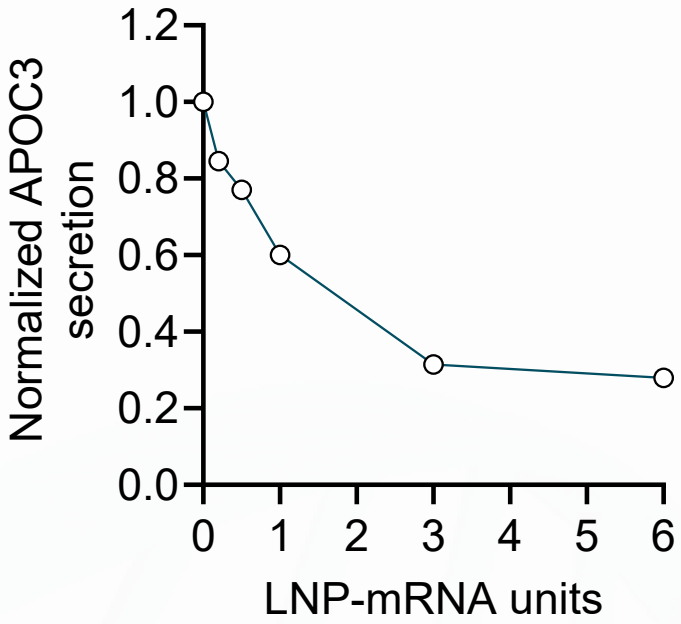
Feola et al, FASEB 2026; Letort et al, ESGCT 2026

.HEAL-101 Stops *APOC3* Expression *In Vitro*

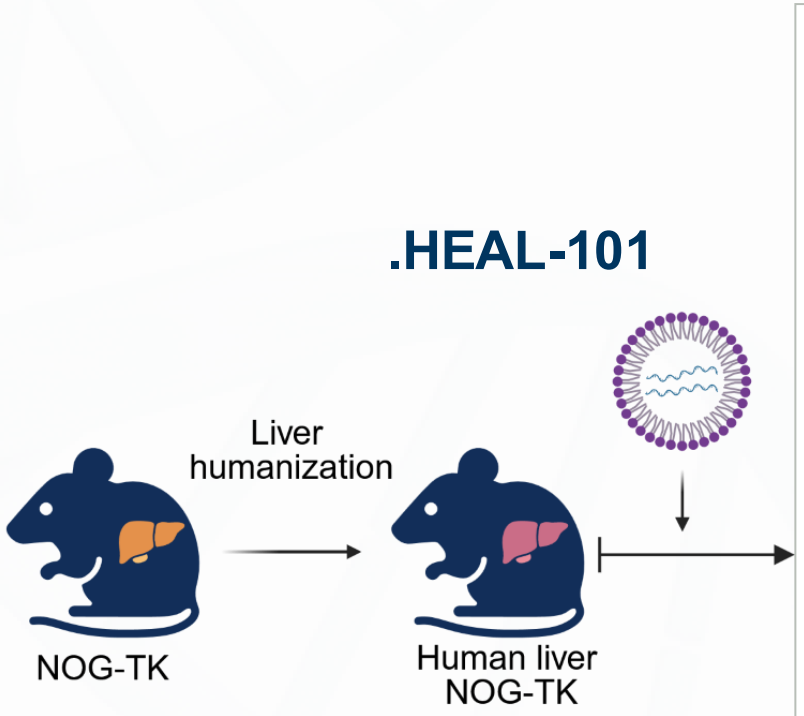
Frequency of *APOC3* editing

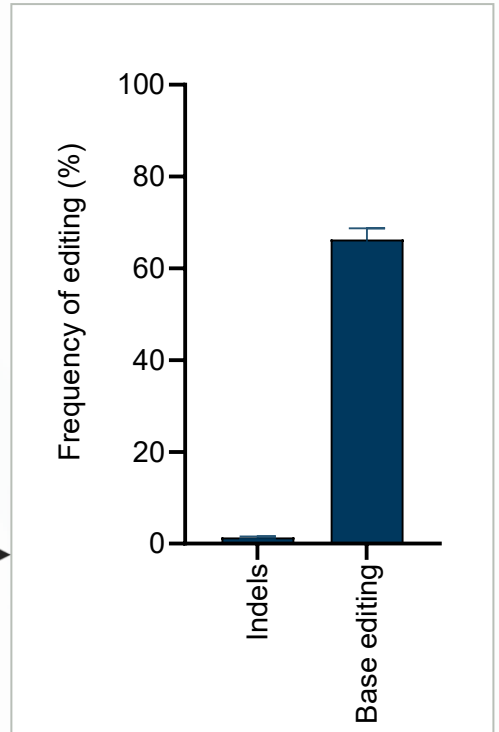


Quantification of *APOC3* secretion

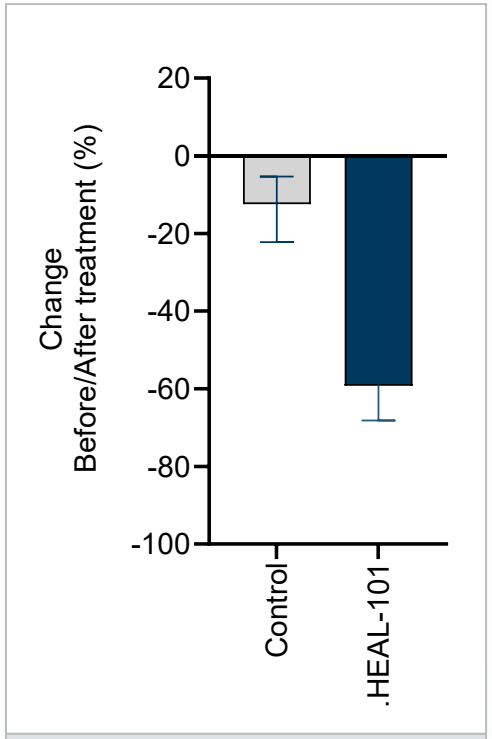


.HEAL-101 Lowers Triglycerides in Humanized Mice

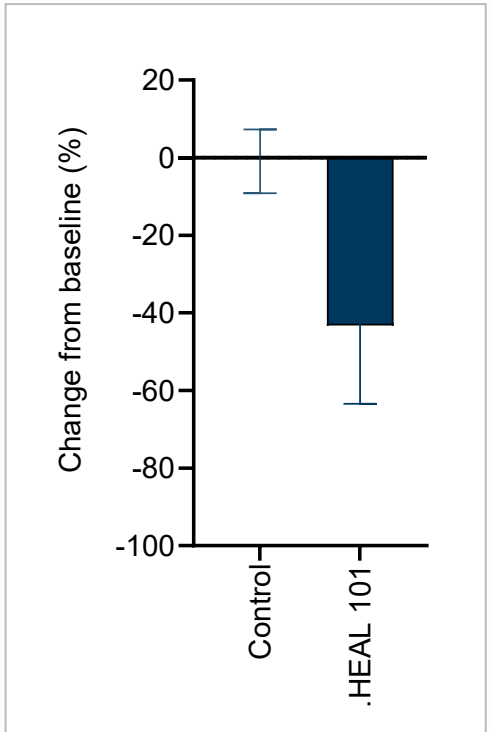




Precise *APOC3* editing



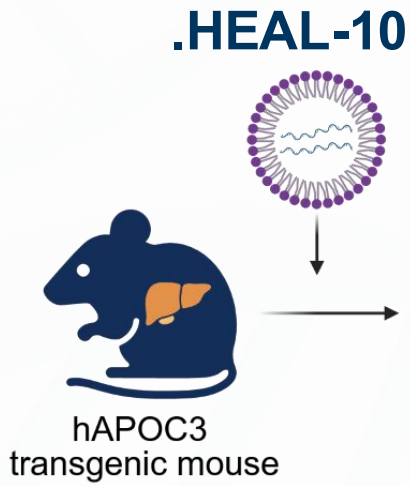
Decrease of *APOC3*

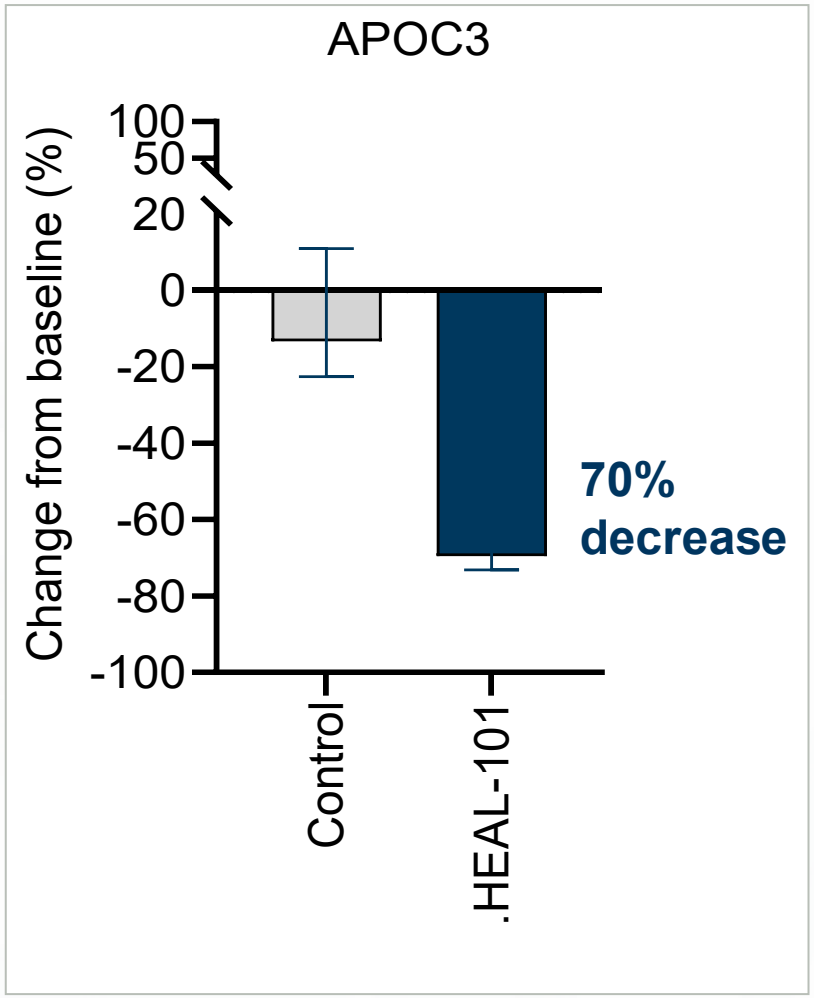


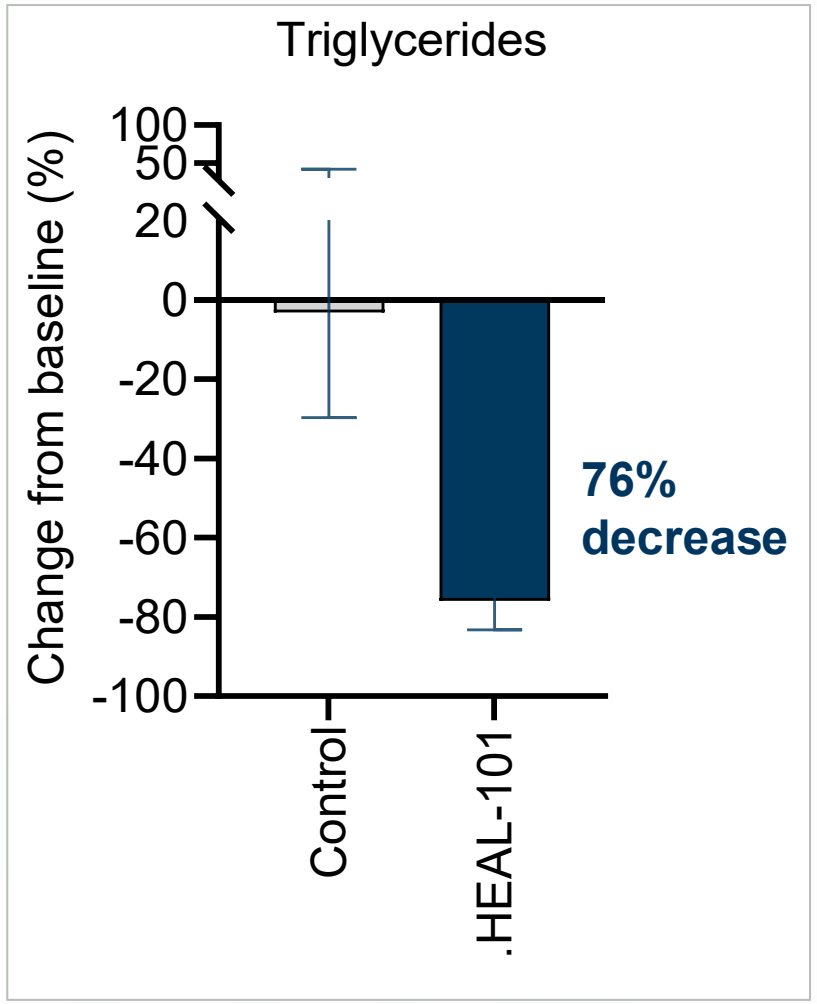
Drop of triglycerides

Research data

.HEAL-101 Lowers Triglycerides by 76% in hAPOC3 Mice







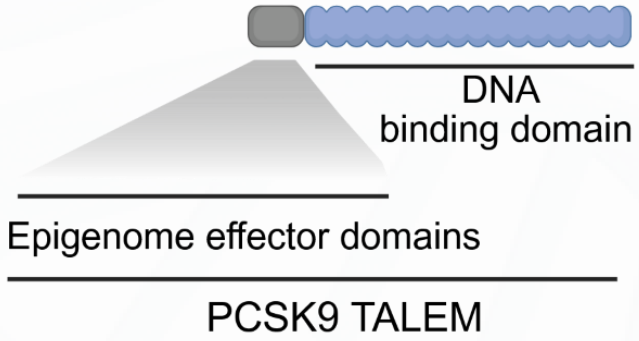
.HEAL-201

GENE SURGERY

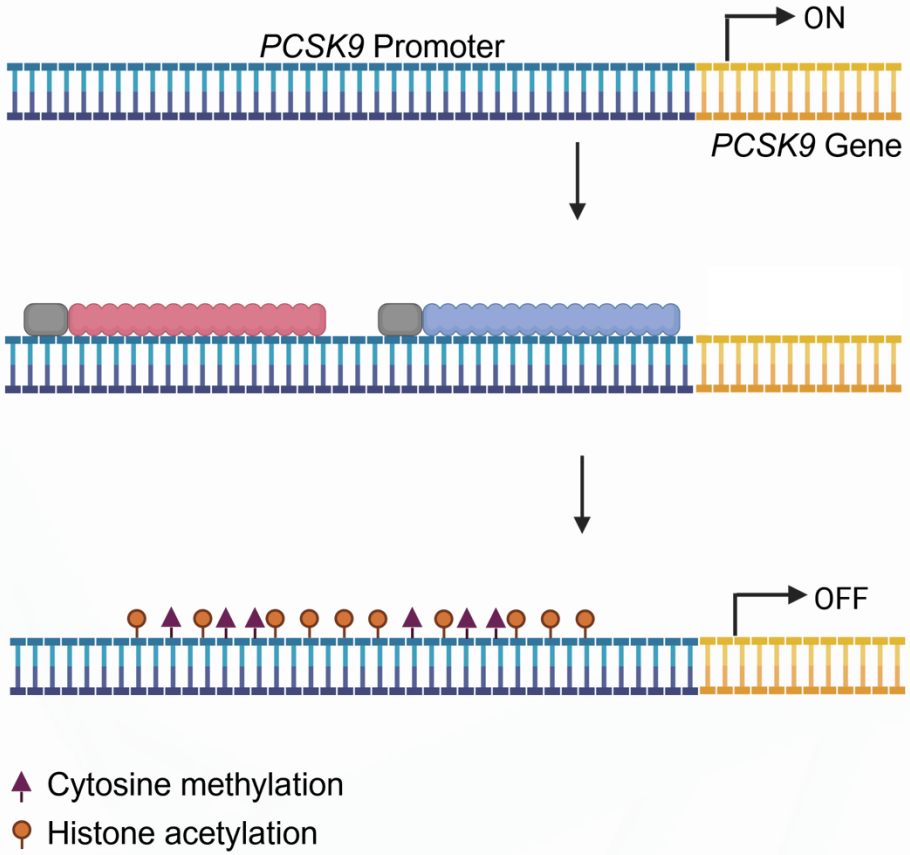
For Severe Hypercholesterolemia (sHC)

.HEAL-201: Silencing PCSK9 without Touching the DNA Sequence

.HEAL-201 is an epigenetic modulator



Epigenetically modulates the *PCSK9* gene without altering the DNA sequence



***PCSK9* is active**

.HEAL-201 binds target

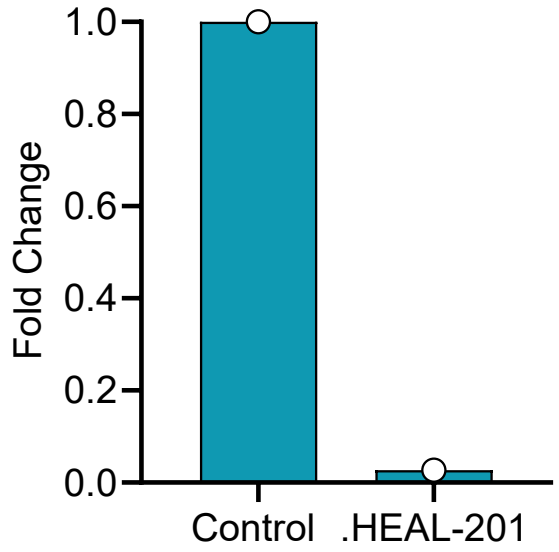
***PCSK9* is silenced**

No DNA sequence change

.HEAL-201 Promotes Efficient and Specific Knockdown of *PCSK9*

Epigenetic change in the DNA without sequence modification

Efficient *PCSK9* mRNA transcription reduction



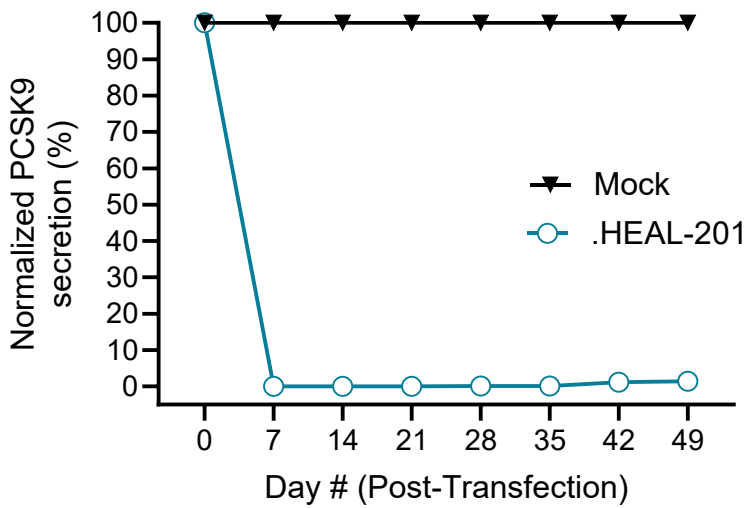
***PCSK9* transcription is shut down**

Research data

.HEAL-201 Promotes Efficient and Specific Knockdown of *PCSK9*

Epigenetic change in the DNA without sequence modification

Efficient *PCSK9* protein reduction

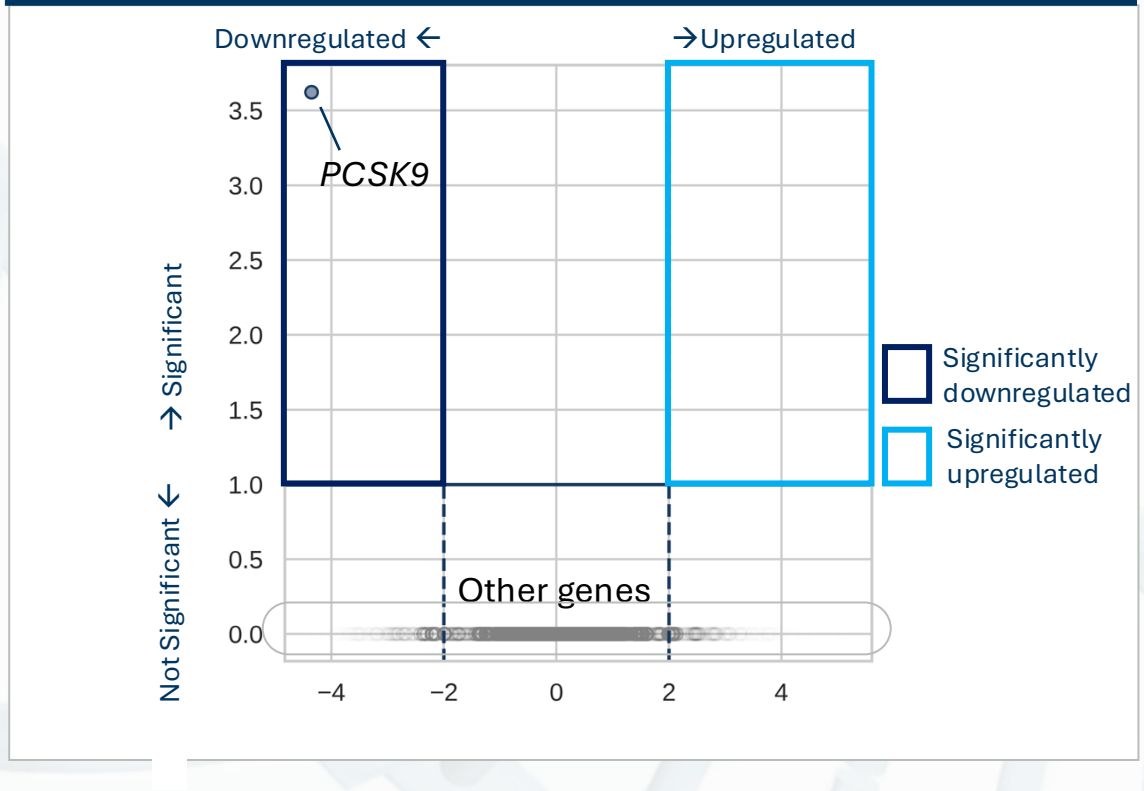


PCSK9 protein is turned off

Research data

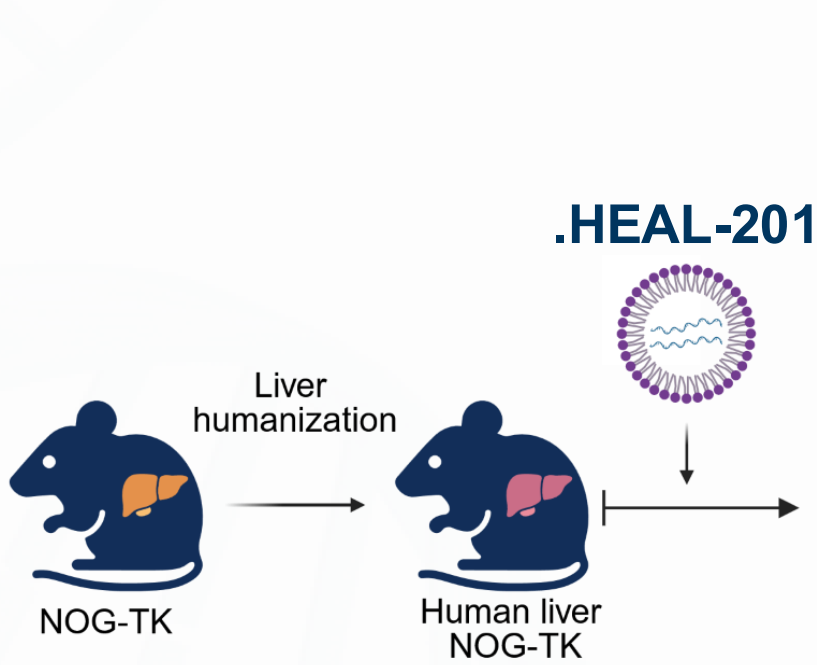
.HEAL-201 Promotes Efficient and Specific Knockdown of *PCSK9*

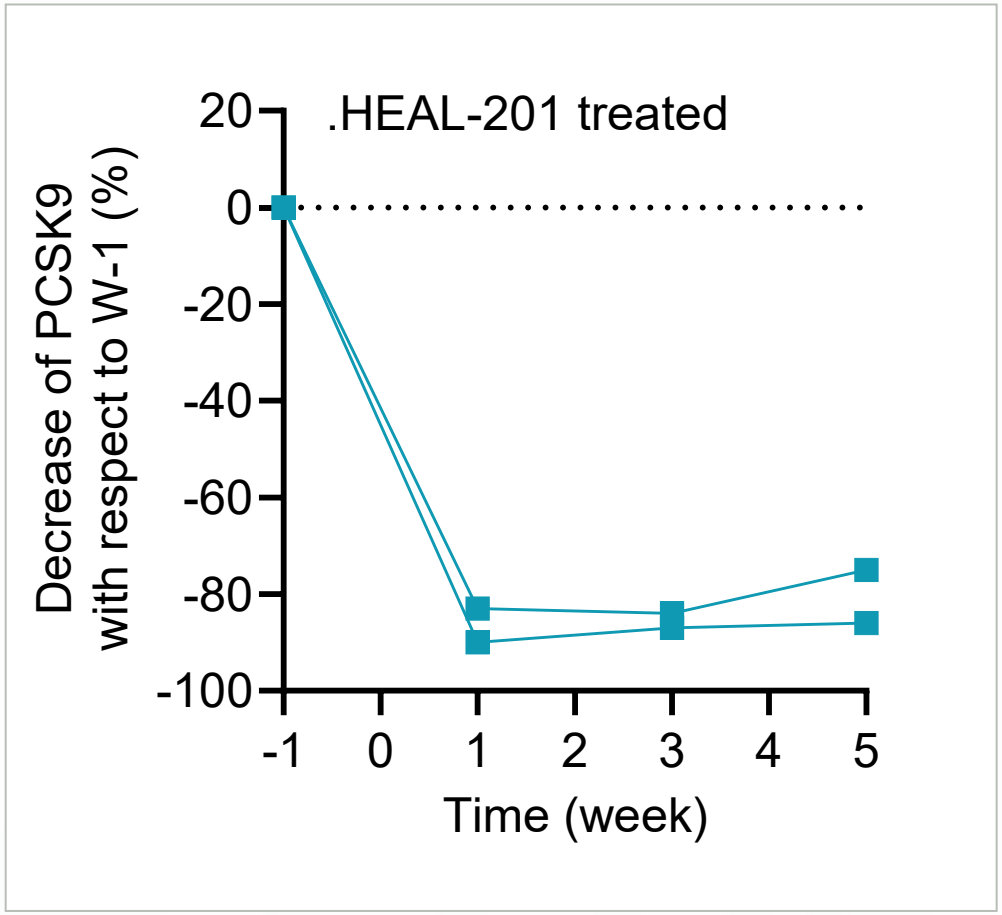
Transcriptome post .HEAL-201 treatment shows a precise modification



.HEAL-201: high specificity on *PCSK9* only

.HEAL-201 Epigenetic Modification is Stable Over Time





.HEAL-201 promotes rapid and durable decrease of *PCSK9*

Research data

Clinical Development Plan

For .HEAL-101 and .HEAL-201

.HEAL-101 Clinical Development Plan

1 Phase 1 Study

Patient population: severe hypertriglyceridemia (sHTG)

OBJECTIVES

- First-in-Human for safety and tolerability
- Establish dose efficacy in triglyceride reduction in a broader sHTG population

2 Phase 1b/2 Study

Patient population: severe high risk hypertriglyceridemia (sHTG)

OBJECTIVES

- Expand to global populations
- Dose optimization and regimen exploration
- Confirm activity and inform Phase 3 pathway

TG reduction supports traditional approval in sHTG

.HEAL-201 Clinical Development Plan

1 Phase 1 Study

Patient population: Severe hypercholesterolemia, HeFH ≥190 mg/dL

OBJECTIVES

- First-in-Human for safety and tolerability
- Establish dose efficacy in LDL-C reduction

2 Phase 1b/2 Study

Patient population: severe hypercholesterolemia, HeFH ≥190 mg/dL, high risk patients

OBJECTIVES

- Severe genetic hypercholesterolemia; LDL-C ≥190 mg/dL
- Early ASCVD - premature cardiovascular disease Persistent elevated LDL-C despite maximal therapy

Surrogate markers accepted by FDA / EMA for approval

ASCVD: atherosclerotic cardiovascular disease ; FDA: Food and Drug Administration ; EMA: European Medicines Agency

Advancing Toward First-In-Human Proof-of-Concept

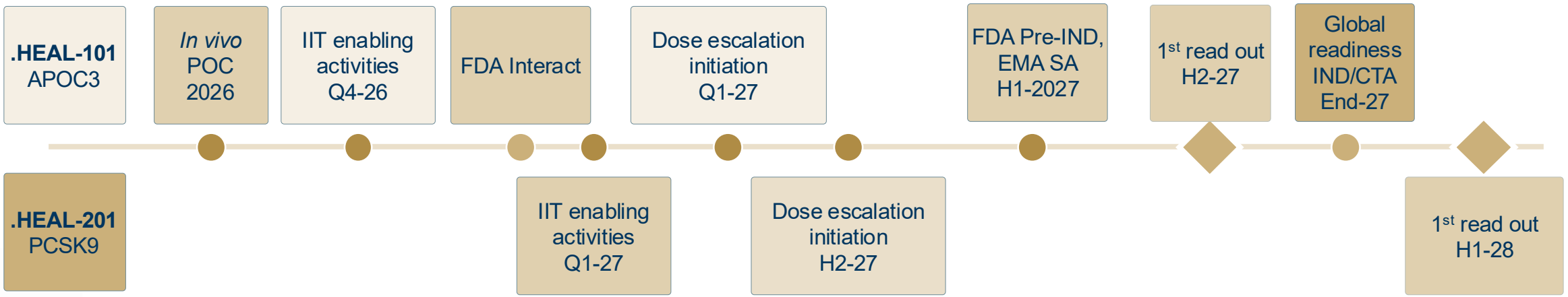
Getting to first human data

- Plan to start an IIT in China
- GMP manufacturing starting
- Phase 1 designed to accelerate dose selection

IND/CTA enabling activities

- FDA / EMA preparation initiated
- Manufacturing scope broadened toward global supply

Potential Path to clinical value creation



IIT: Investigator-Initiated trial ; GMP: Good Manufacturing Practices ; PoC: Proof-of-concept ; IND/CTA: Investigational New Drug/Clinical Trial Application

Cellectis Targeted Roadmap

Advancing 2 Lead Assets	**FIH**	**Data disclosure**
.HEAL-101 targeting *APOC3* in severe hypertriglyceridemia	H1-27	H2-27
.HEAL-201 targeting *PCSK9* in severe hypercholesterolemia	H2-27	H1-28

Continuing Existing Partnerships

AstraZeneca

Allogene THERAPEUTICS

SERVIER moved by you

IOVANCE

Targeting Cash Runway into H2 2028*

Exiting internal cell therapy development (lasme-cel & eti-cel)

Re-aligning our organization** to our new priorities

*Excluding potential proceeds from new partnerships

**Subject to the completion of the relevant works council consultation process and applicable French & US labor law requirements

FIH : First-in-Human

Thank You

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Raleigh, NC, 27616 – USA



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APPENDIX

Diversified Partnerships with Industry Leaders

SERVIER moved by you / Allogene THERAPEUTICS		Allogene THERAPEUTICS	IOVANCE BIOTHERAPEUTICS	AstraZeneca
CAR-T – CD19 cema-cel		**CAR-T – 15 targets ALLO-316 (CD70)**	**TILs IOV-4001**	**Cell and Gene Therapies**
Exclusive[1] worldwide license to CD19-directed allogeneic CAR T-cells	U.S. rights exclusively sublicensed to Allogene by Servier[2]	Exclusive worldwide license to 15 allogeneic CAR T-cell targets	Research collaboration and exclusive worldwide license agreement to develop gene-edited TILs	Joint Research and Collaboration agreement to develop up to 10 novel products in oncology, immunology and rare diseases and investment agreement
Up to $340M in Development & Sales Milestones + Low Double-Digit Royalties on Sales		Up to $2.8B in Development & Sales Milestones + High Single-Digit Royalties on Sales	Undisclosed Financials	Up to $80M to $253M Per Product in Milestones + Tiered Royalties

1. The exclusivity is subject to the December 2025 Arbitral Decision.
2. Servier grants to Allogene exclusive rights to cemacabtagene ansegedleucel in the U.S., EU and UK, and Allogene has been granted an option to extend its licensed territory to China and Japan subject to certain conditions

TIL: Tumor-Infiltrating Lymphocyte

Partnered Cell Therapy Pipeline

STUDY	INDICATION / TYPE	PRECLINICAL	PHASE 1	PHASE 2[1]	UPCOMING EXPECTED MILESTONE
ALPHA3 **Cema-cel (CD19)**[1]	LBCL (Allogeneic CAR-T)				SERVIER moved by you / Allogene THERAPEUTICS
TRAVERSE[2] **ALLO-316 (CD70)**	RCC (Allogeneic CAR-T)				Allogene THERAPEUTICS
IOV-GM1-201 **IOV-4001**	Melanoma (TIL)				IOVANCE
UNDISCLOSED					AstraZeneca

1. cemacabtagene ansegedleucel has been developed under a collaboration agreement between Servier and Allogene based on the License, Development and Commercialization Agreement signed between Cellectis and Servier. Servier grants to Allogene exclusive rights to cemacabtagene ansegedleucel in the U.S., EU and UK. The ALPHA3 study targets Large B-Cell Lymphoma (LBCL).
2. ALLO-316 utilizes TALEN® gene-editing technology pioneered and owned by Cellectis. Allogene has an exclusive license to the Cellectis technologies for allogeneic products directed at the CD70 target. Allogene holds global development and commercial rights for this investigational candidate.

LBCL: Large B-Cell Lymphoma; RCC: Renal Cell Carcinoma; TIL: Tumor Infiltrated Lymphocyte