CORPORATE UPDATE

September 14th, 2026

Forward Looking Statements

This press release 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”, “plan”, “potential”, “promising”, “target”, “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. Forward-looking statements in this press release 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 planned timing for the release 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 preclinicial 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.

Agenda and Speakers

- Welcome
- Cellectis, A Gene Editing Company
- *In vivo* Gene Editing
- Strategic Transformation
- Q&A

Speakers

André Choulika, PhD
Chief Executive Officer

Adrian Kilcoyne, MD, MPH,MBA
Chief Medical Officer

Gene Editing

Cellectis is a Gene Editing Company

Cellectis since its inception over 25 years ago is a pioneer in Gene Editing

Gene Editing intervenes in cells at genome level to change gene expression by:

 Silencing

 Activating

 Repairing targeted gene(s)

Gene Editing has transformed science in numerous ways
and many experts agree that it will be a major paradigm shift in medicine

Gene Editing in Medicine

Genetics drive disease:

Cancer, diabetes, obesity, neurological disorders, immunity, longevity… all diseases are rooted in genetics: polymorphisms and aberrant gene expression.

The basic logic of gene editing: fix the gene, fix the disease.

Two pathways for gene editing

EX VIVO

Collect cells
→ edit them under GMP conditions
→ infuse (example of CAR-T cells)

Cellectis main focus so far

IN VIVO

Deliver the editor to the target tissue
→ silence, activate, repair

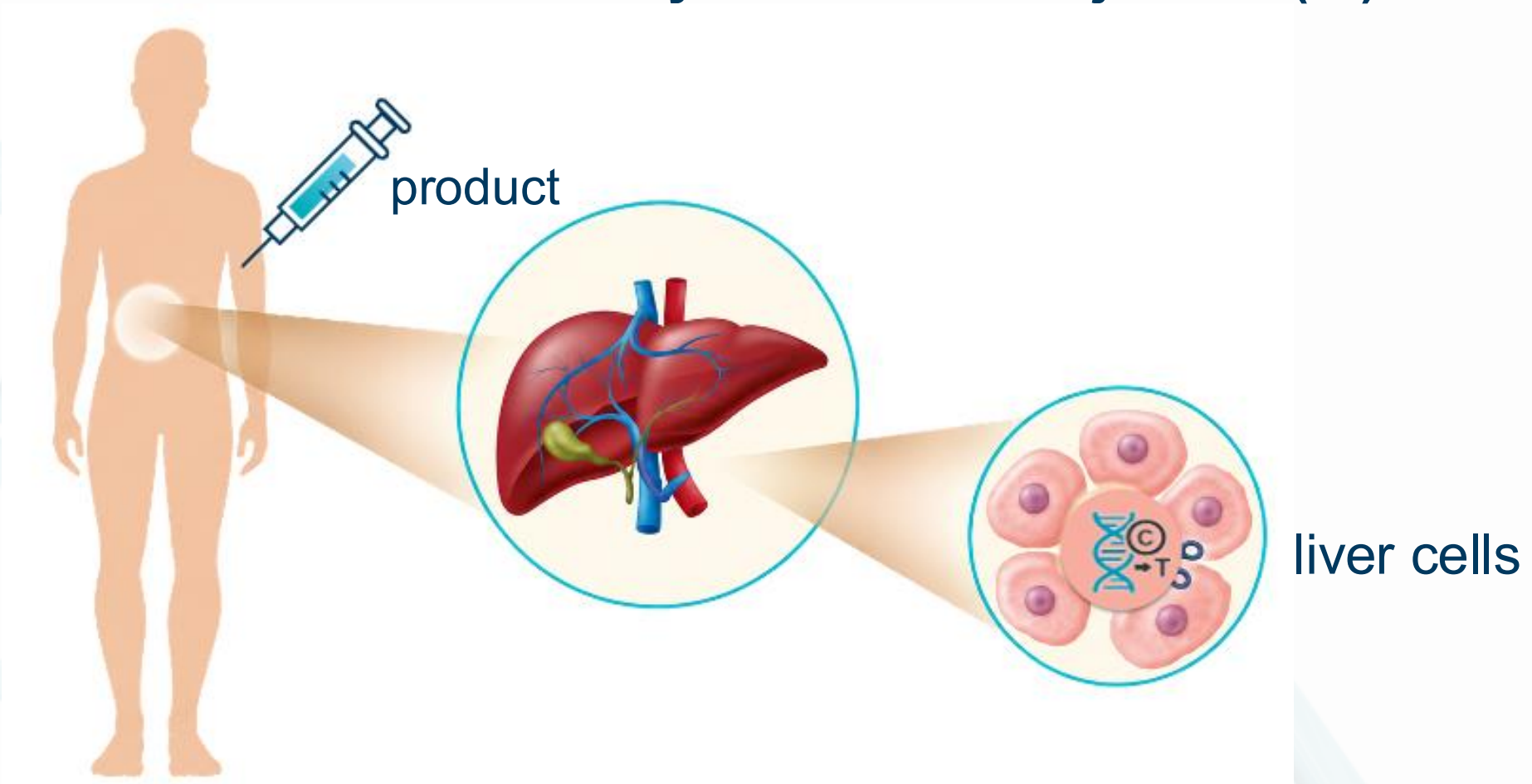
Cellectis new focus from now

Building on our *ex vivo* leadership to advance the next frontier: *in vivo* gene editing

in vivo Gene Editing

How does *In Vivo* Gene Editing work

In vivo delivered is made by intravenous injection (IV)

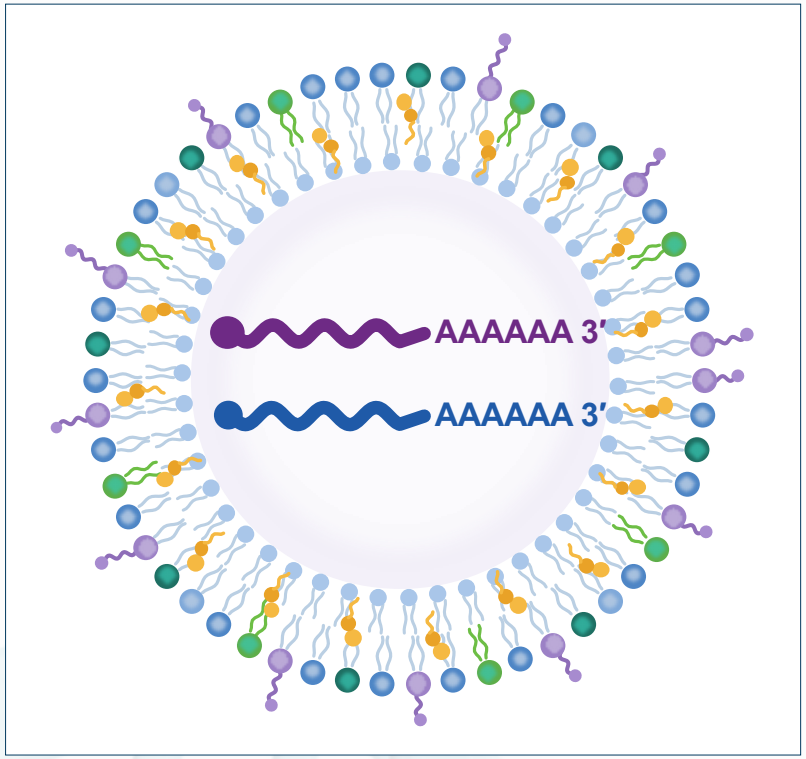


A single IV injection for a long-lasting effect

What is The *in vivo* Gene Editing Product?

Product:

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



A One-Time Treatment With a Long-Lasting Effect

Two Promising Preclinical *In Vivo* Assets

Decision to advance 2 preclinical candidate products in dyslipidemia:

.HEAL-101 (mRNA+LNP)

Targeting *APOC3* in severe hypertriglyceridemia (sHTG)

.HEAL-201 (mRNA+LNP)

Targeting *PCSK9* in severe hypercholesterolemia

- ✓ Well validated targets
- ✓ Single IV injection offering potentially long-lasting effect

IV: intravenous

.HEAL-101

GENE SURGERY

Severe Hypertriglyceridemia (sHTG): A Major Unmet Medical Need

High Clinical Burden

High risk of acute pancreatitis

Associated with cardiovascular disease

Poor quality of life and limited treatment options

.HEAL-101 Target Patient Population

High Risk sHTG (TGs ≥ 880 mg/dL or ≥ 500 mg/dL With Prior AP History)

Total: ~1-2 million patients US/EU

.HEAL-101

Indication: Severe Hypertriglyceridemia (sHTG) with high risk of acute pancreatitis

Target: *APOC3* gene is well-validated with ASO and RNAi

Tech: TALE-Base Editor disables the *APOC3* gene

Goal: One-time treatment for durable lowering of triglycerides to reduce risk of acute pancreatitis

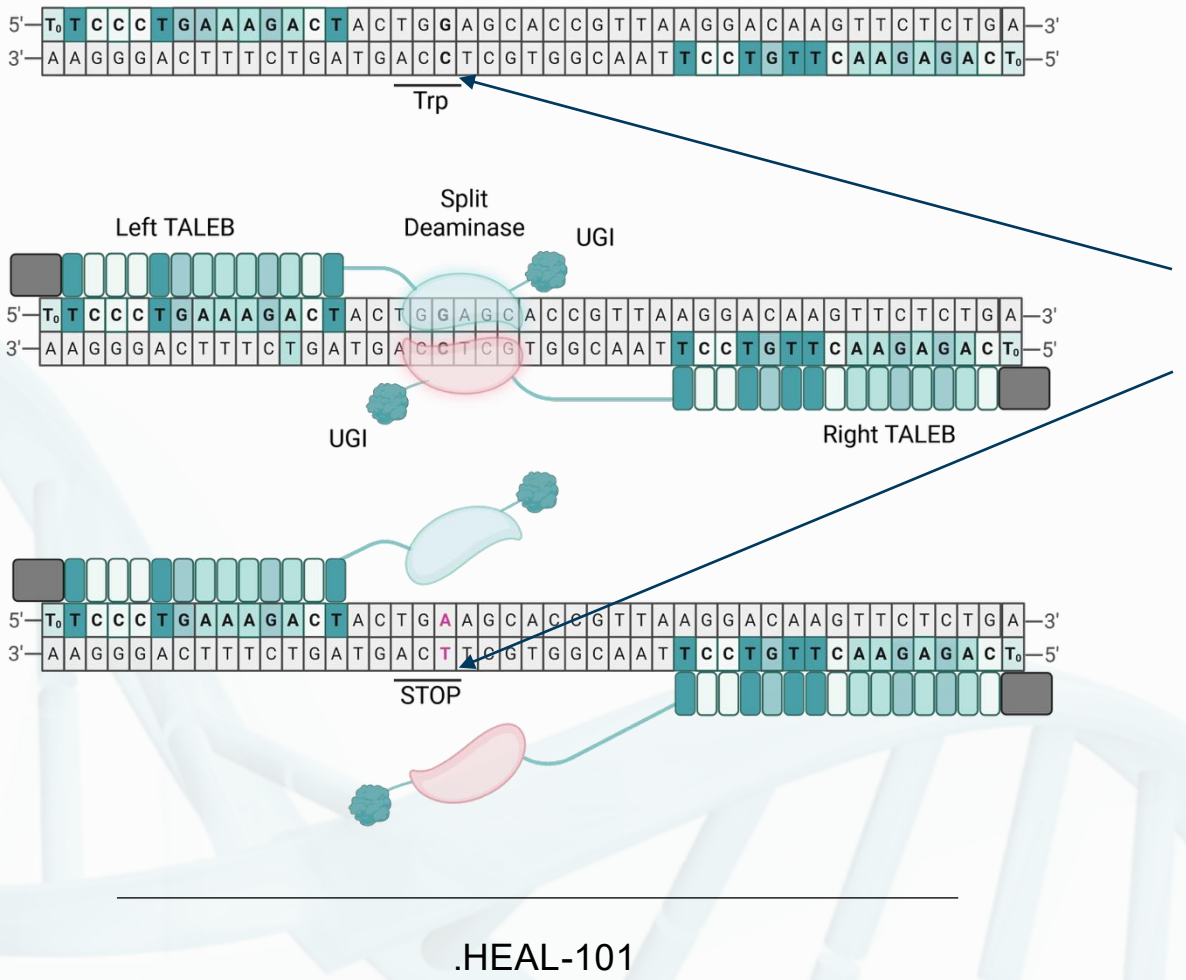
Status: Promising preclinical data supporting transition to clinic

ASO: Antisense Oligonucleotides; RNAi: interfering RNA

.HEAL-101: TALE-Base Editor Targeting *APOC3*

.HEAL-101 is a base editor

It modifies one base in DNA

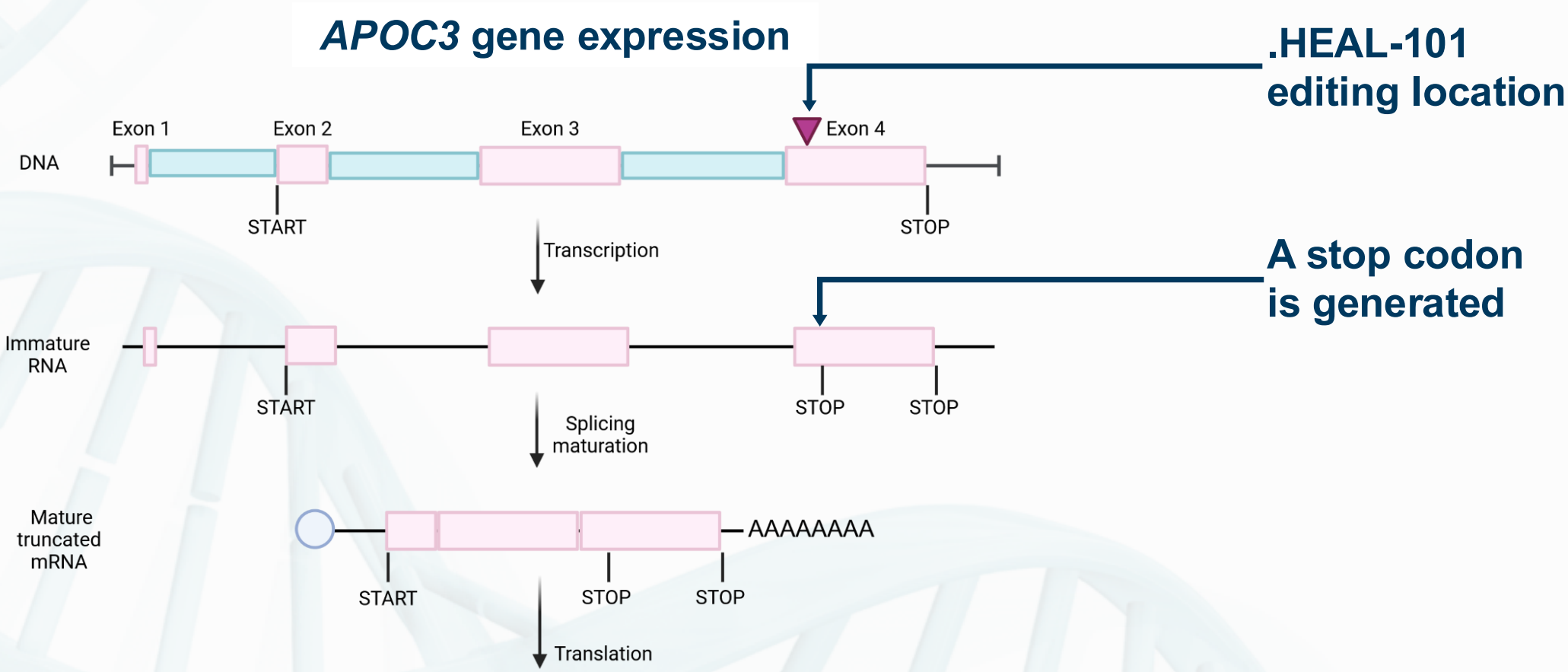


C to T conversion

.HEAL-101

No DNA break offering potential of improved safety profile

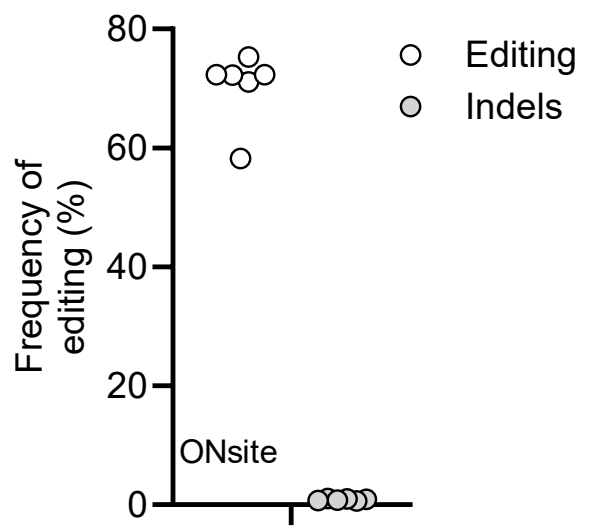
.HEAL-101: Disable *APOC3* Expression



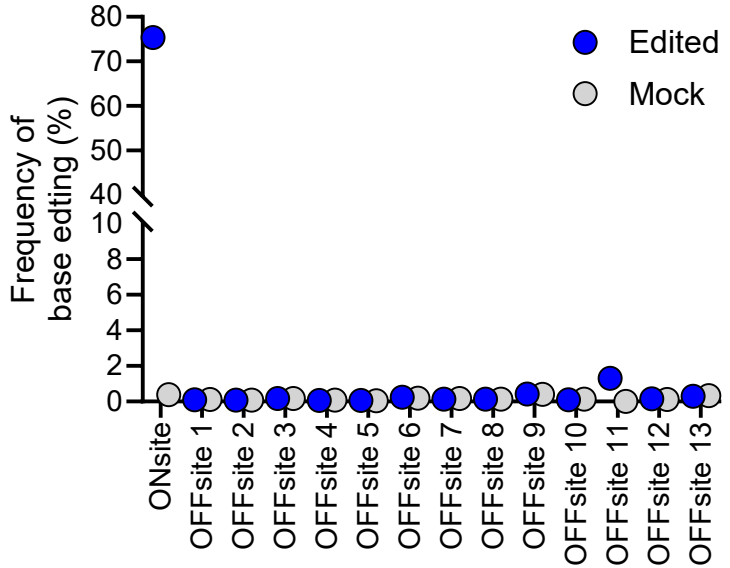
.HEAL-101 Elicits Efficient and Specific Editing of *APOC3* Gene

.HEAL-101 mRNA is introduced in hepatic cell lines

Efficient on-site modification with no DNA alteration



No modification of top putative off-sites

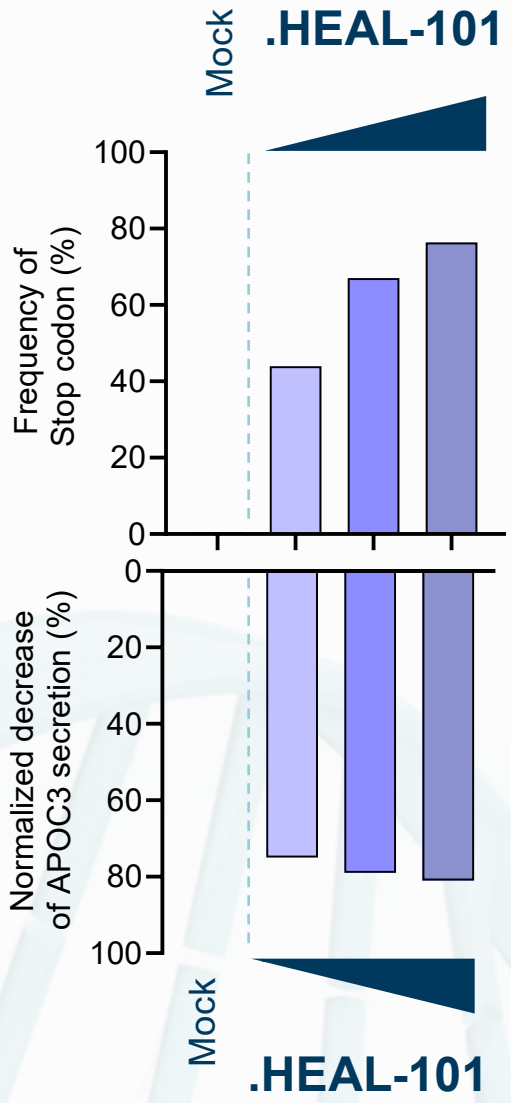


Feola et al, FASEB 2026

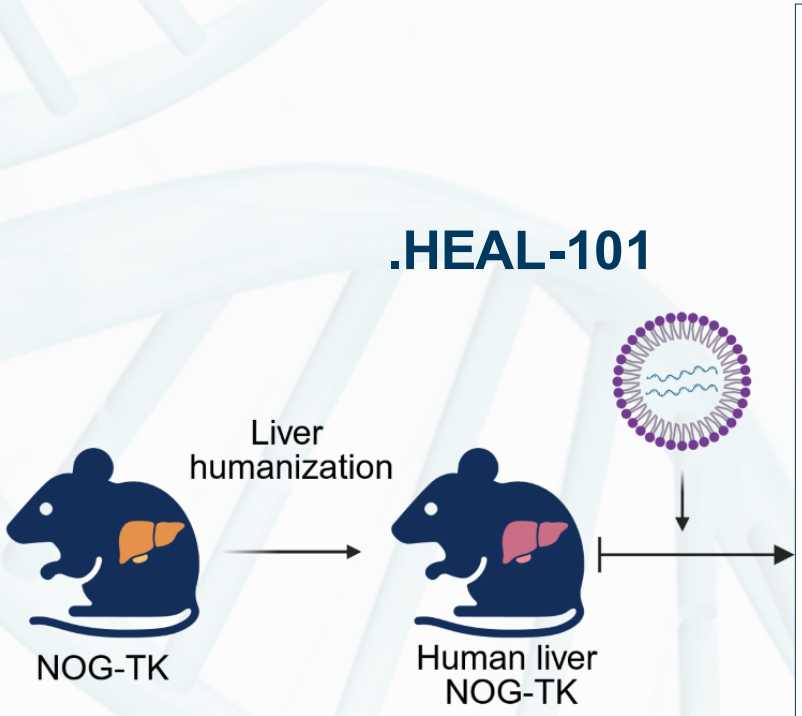
.HEAL-101 Stops *APOC3* Expression *In Vitro*

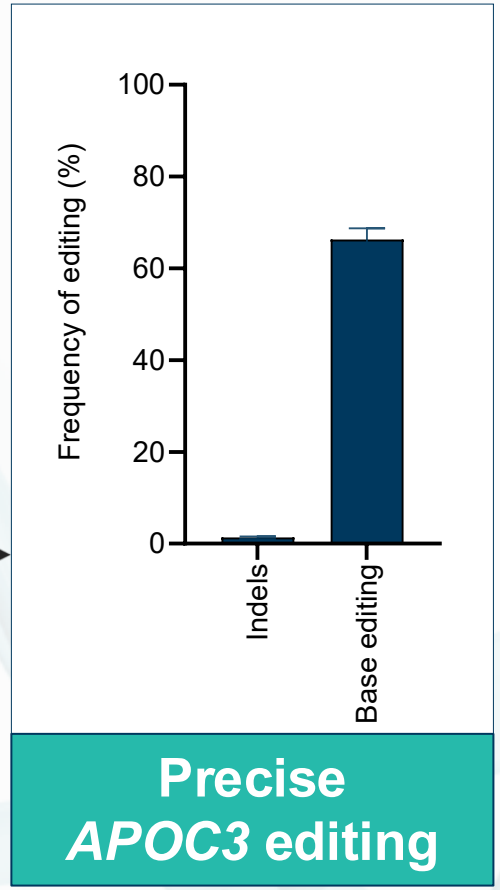
Frequency of *APOC3* editing

Quantification of *APOC3* secretion

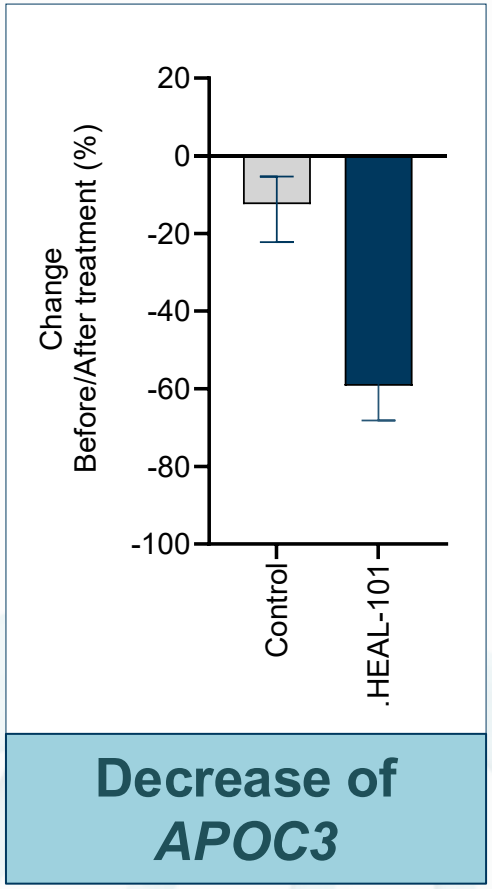


.HEAL-101 Decreases Triglycerides in Humanized Mice

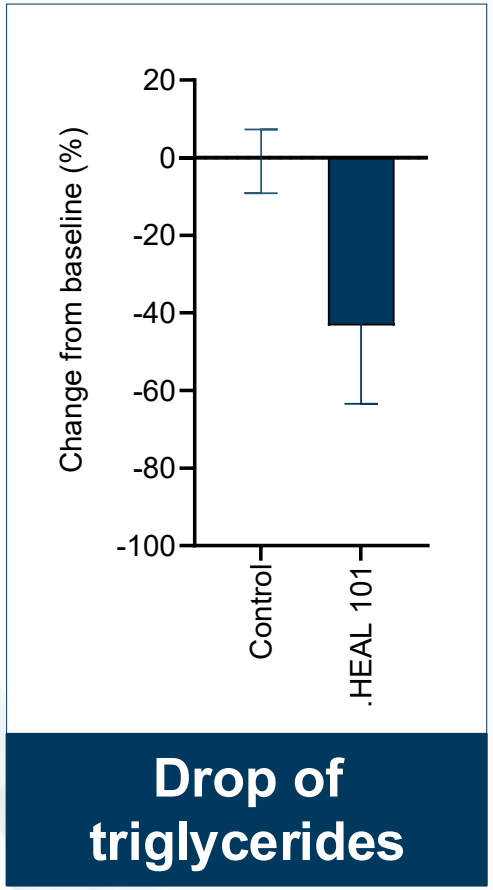




Precise *APOC3* editing

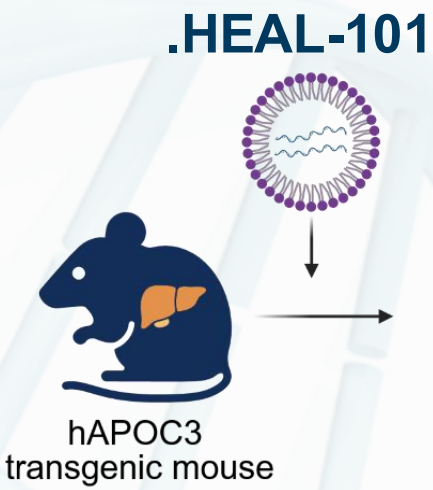


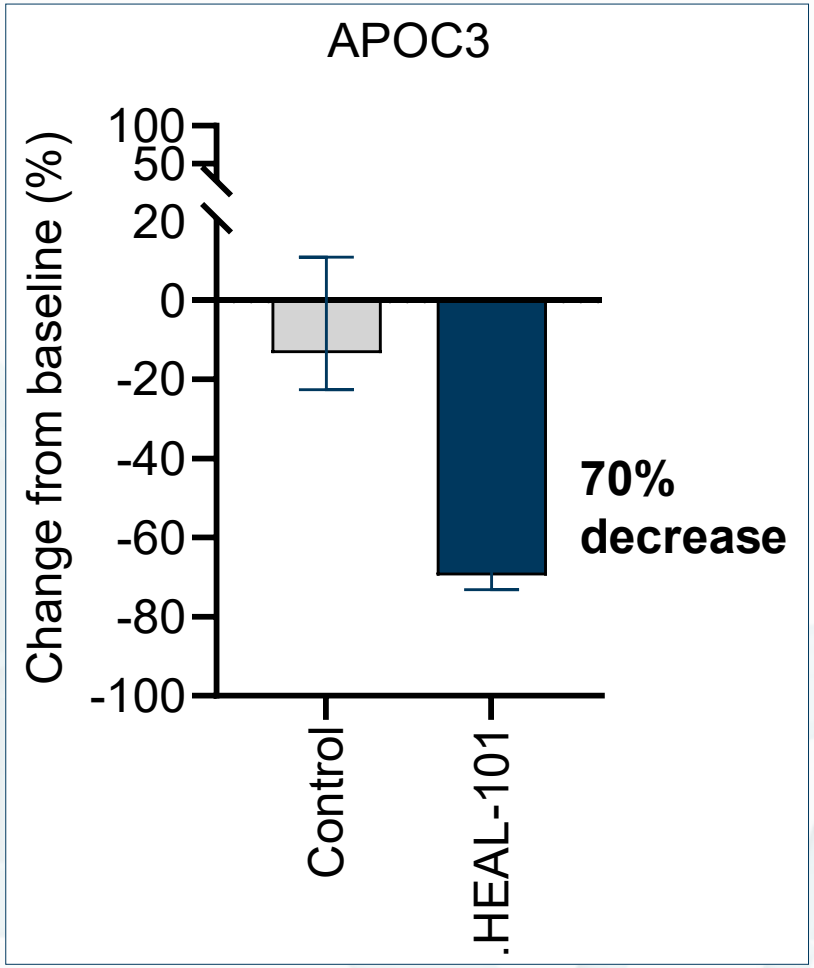
Decrease of *APOC3*

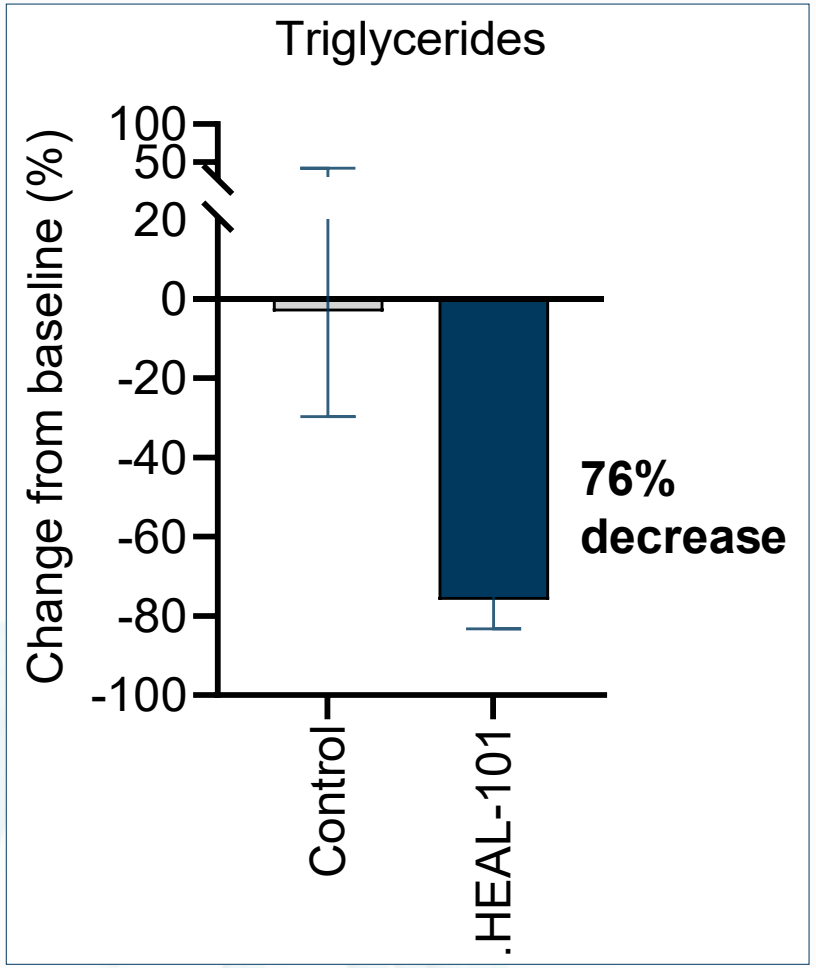


Drop of triglycerides

.HEAL-101 Induces 76% Decrease of Triglycerides in Affected Mice







.HEAL-201

GENE SURGERY

Hypercholesterolemia: A Major Unmet Medical Need

High Clinical Burden

- Leading driver of atherosclerotic cardiovascular disease
- Lowering LDL-C reduces risk of cardiovascular event (CVE)
- Many patients remain above LDL-C goals despite maximally tolerated therapy

.HEAL-201 Target Patient Population

- Younger high risk patients
- Premature Cardiovascular disease
- Persistent elevated LDL-C despite maximal therapy
- Total: ~1-2 million patients (US + EU)

LDL-C: Low-Density Lipoprotein Cholesterol

.HEAL-201

Indication: Severe Hypercholesterolemia

Target: *PCSK9* gene is a well validated target

Tech: TALE-Modulator silences the *PCSK9* gene

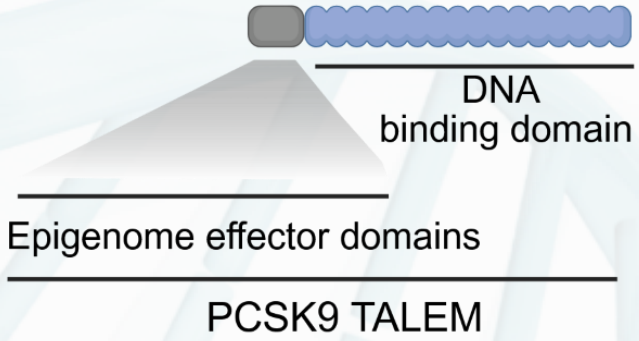
Goal: One-time treatment for durable lowering of LDL-C to reduce CVE risk

Status: Promising preclinical data supporting transition to clinic

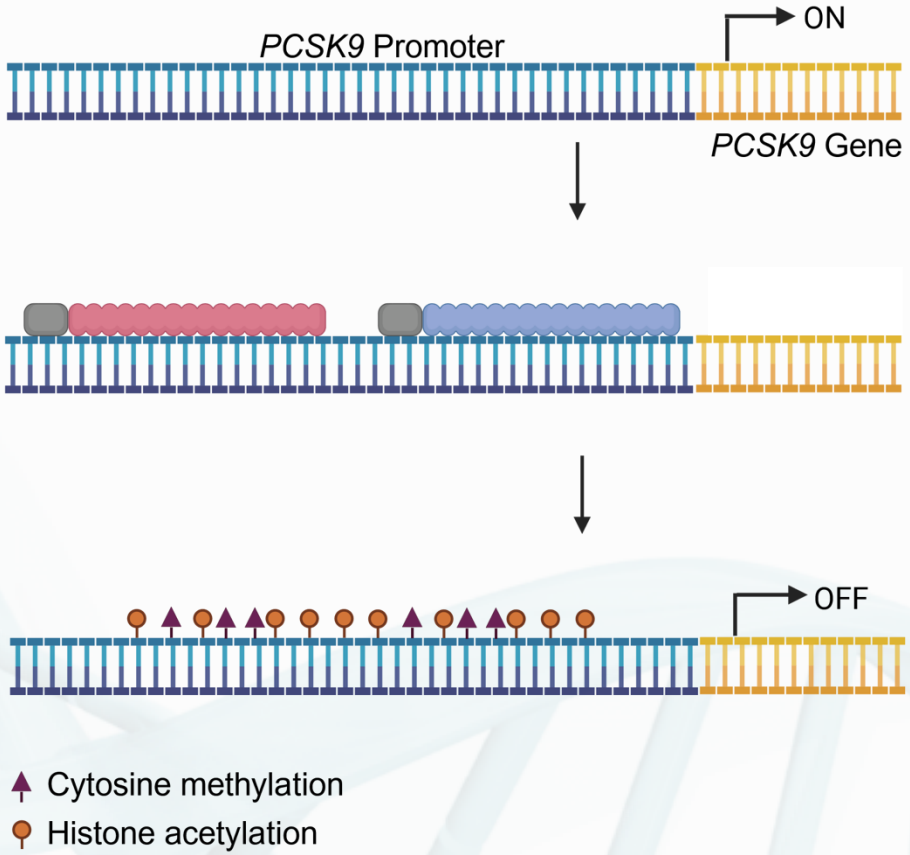
CVE: cardiovascular event

.HEAL-201: TALE-Epigenetic Modulator Targeting *PCSK9*

.HEAL-201 is an Epigenetic modulator



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



***PCSK9* is active**

.HEAL-201 binds target

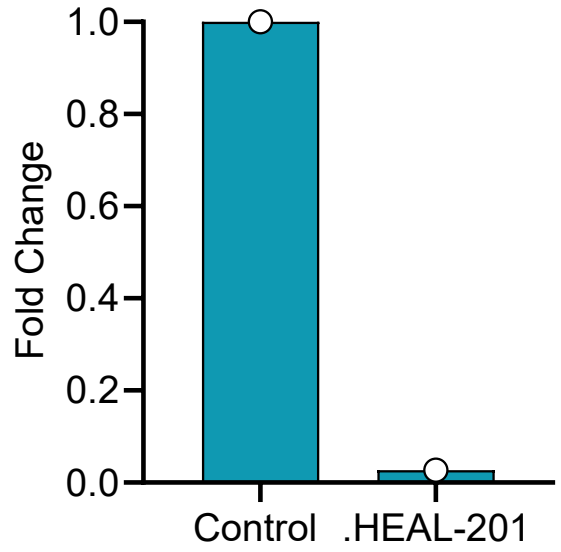
***PCSK9* is silenced**

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

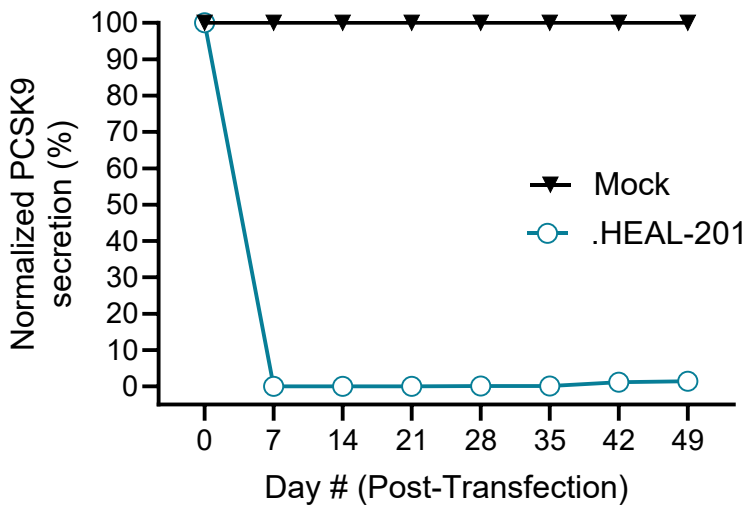


PCSK9 transcription is shut down

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

Epigenetic change in the DNA without sequence modification

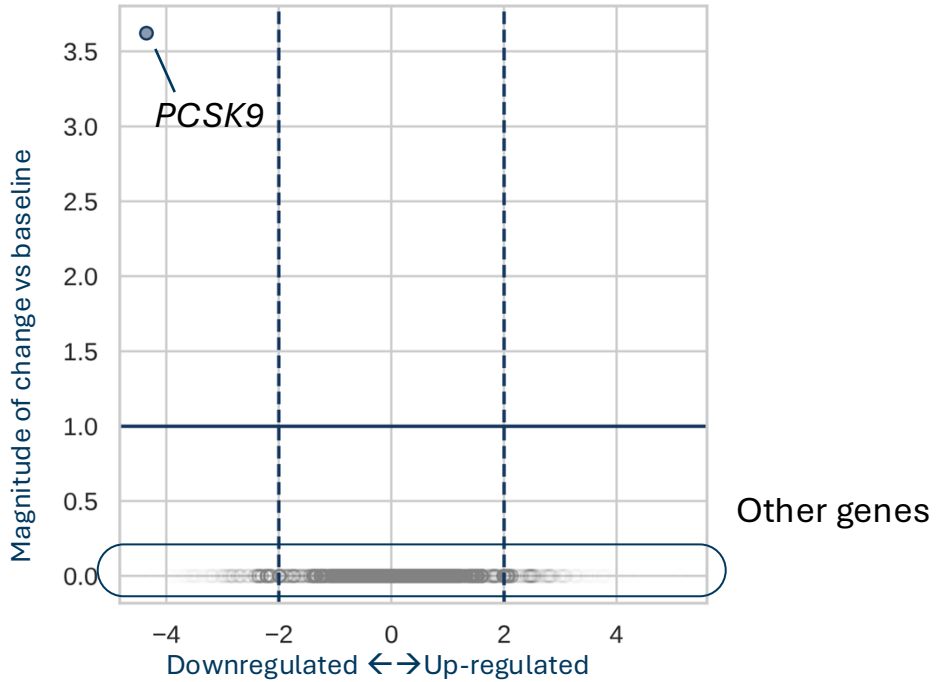
Efficient *PCSK9* protein reduction



PCSK9 protein is turned off

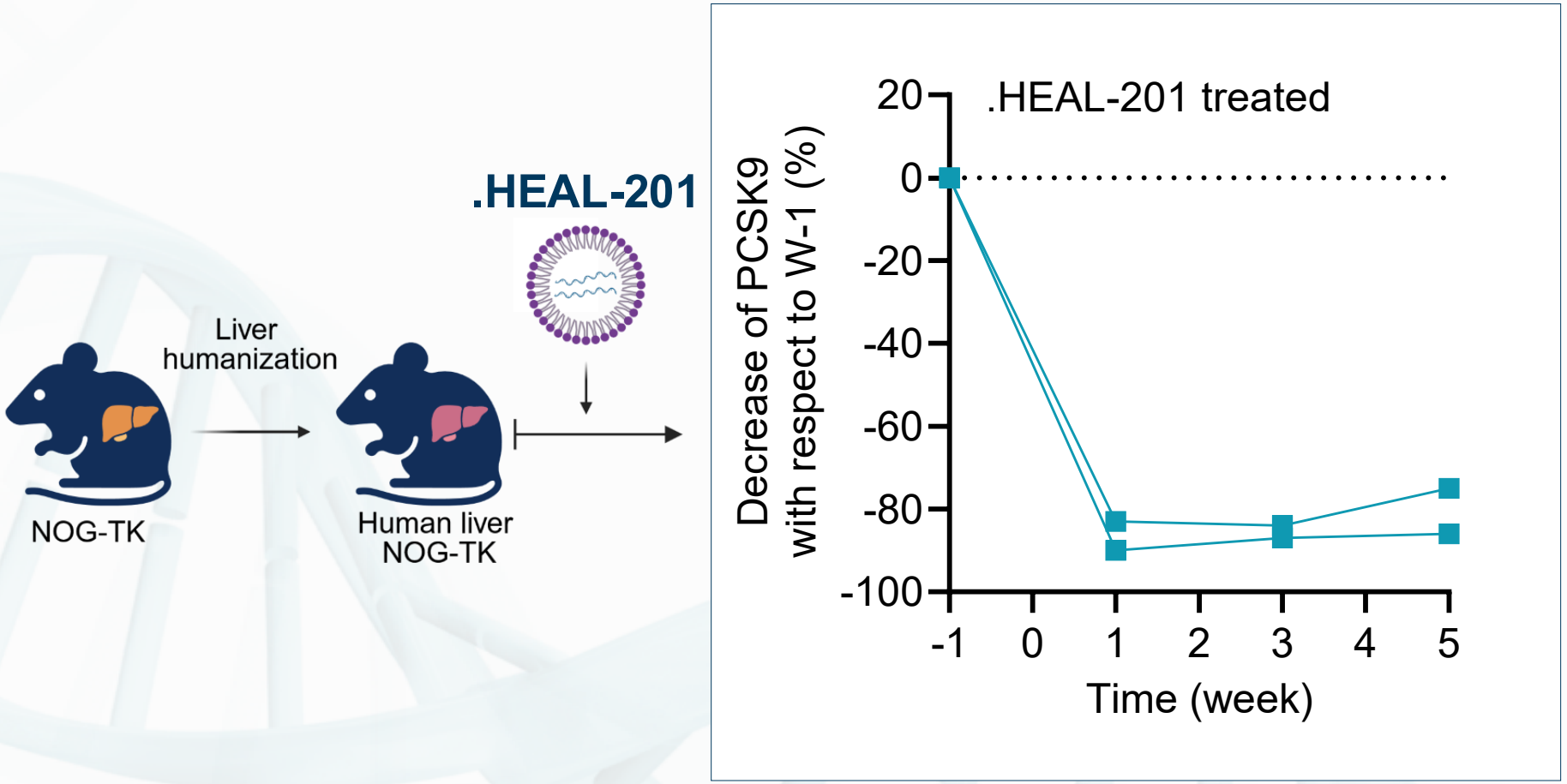
.HEAL-201 Promotes Efficient and Specific Knockdown of *PCSK9*

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*

Clinical Development Plan

.HEAL-101 Clinical Development Plan

1 Phase 1 Study

- Patient population: severe hypertriglyceridemia (sHTG)
- First-in-Human for safety and tolerability
- Establish dose efficacy in triglyceride reduction in a broader sHTG population

2 Phase 1b/2 Study

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

No cardiovascular outcomes trial required for commercialization

.HEAL-201 Clinical Development Plan

1 Phase 1 Study

- Patient Population: Severe hypercholesterolemia ≥190 mg/dL
- First-in-Human for safety and tolerability
- Establish dose efficacy in LDL-C reduction in a broader population

2 Phase 1b/2 Study

- Younger high risk patients
- 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: atherosclerosis cardiovascular disease

Advancing Toward First-In-Human Proof of Concept

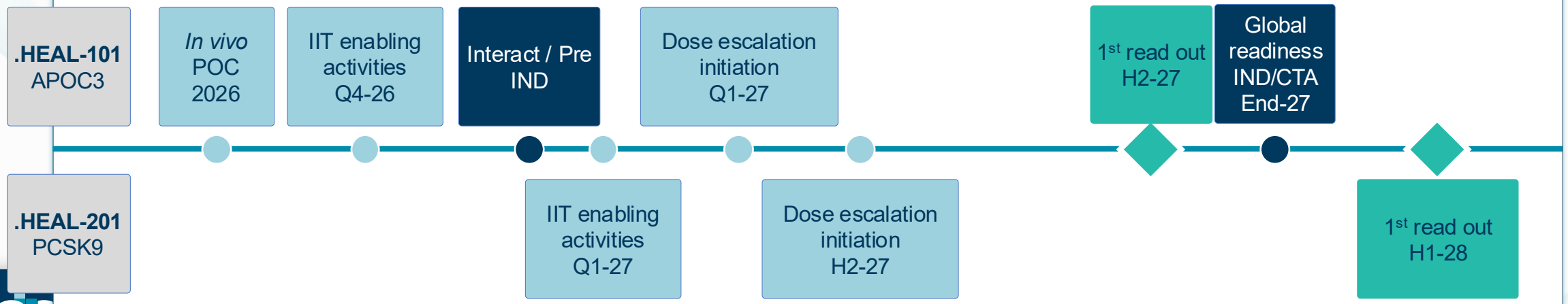
Getting to first human data

- Plan to start an IIT in China
- GMP manufacturing starting
- Phase 1 designed to be optimized for rapid dose selection

IND/CTA enabling activities

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

Potential Path to clinical value creation



A Differentiated Gene Editing Platform

Cellectis' Core Competencies

	Nuclease	Base Editor	Epigenetic	Transcription
cellectis EDITING LIFE	✓	✓ .HEAL-101	✓ .HEAL-201	✓
prime medicine	✓	✗	✗	✗
editas MEDICINE	✓	✗	✗	✗
CRISPR THERAPEUTICS	✓	✗	✗	✗
Beam THERAPEUTICS	✗	✓	✗	✗
Intellia THERAPEUTICS	✓	✓	✗	✗
scribe THERAPEUTICS	✓	✗	✓	✗
PRECISION BIOSCIENCES	✓	✗	✗	✗

Cellectis' Unique Advantages

Broad Gene Editing Toolbox

- Nuclease editing
- Base editing
- Epigenetic editing
- Transcriptional regulation

Strong Differentiation

- TALE editors
- Binding does not nick DNA
- Precision to the base pair
- 32 base pairs recognition site

Unique Advantages

- Any Gene Editing modality
- Hybrid tools combinations

Cellectis' Strategic Decision to Pivot to *in vivo* Gene Editing

Changes in the Heme-Onc Landscape in 2026

B-ALL:

- Frontline Blinatumumab consolidation reduces relapses to 15-25% (vs 45-55% before its introduction), substantially reducing 3rd line+ B-ALL patients
- New 2nd and 3rd line+ pipeline drugs with novel bispecifics and ADCs
- Focus shifts from salvage therapy to earlier intervention and long-term disease control

NHL:

- Cema-cel in frontline consolidation has demonstrated higher MRD-conversation rate
- New 2nd and 3rd line+ pipeline drugs with bispecific, ADCs and other novel combos
- *In vivo* CAR-T rush in LBCL

Earlier interventions and novel therapies are improving depth of response, durability and survival in B-ALL and NHL

ADC: antibody-drug conjugate; MRD: Minimal Residual Disease; LBCL: Large B-cell Lymphomia; B-ALL: B-cell acute lymphoblastic leukemia; NHL: Non-Hodgkin lymphoma

Cellectis Pivot

Cellectis Today

EX VIVO CELL THERAPY

Lasme-cel	Eti-cel	Cell therapy Partnered programs
in Pivotal	Phase 1	
Phase 2	Completed	

IN VIVO GENE EDITING

Promising Preclinical *in vivo* Gene Editing Assets

Cash runway expected into Q4 2027

Cellectis Tomorrow

EX VIVO CELL THERAPY

Exiting internal programs

Continuing existing collaborations

IN VIVO GENE EDITING

Advancing .HEAL-101 & .HEAL-201 with FIH in 2027

Cash runway expected into H2 2028

FIH: First in Human

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), seeking partnership

Re-aligning our organization** to our new priorities

*Excluding potential proceed from new partnership

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

FIH : First in Human

.HEAL

GENE SURGERY

By

Questions & Discussion

André Choulika, PhD
Chief Executive Officer

David Sourdive, PhD
Executive Vice President
CMC and Manufacturing

Arthur Stril
Chief Business Officer &
Chief Financial Officer

Adrian Kilcoyne, MD, MPH,MBA
Chief Medical Officer