Lead *In Vivo* Development Candidate Webinar

September 2, 2025

Forward Looking Statements

This presentation contains forward-looking statements and information within the meaning of The Private Securities Litigation Reform Act of 1995, including statements regarding the potential market for EDIT-401, if approved. The words ‘‘anticipate,’’ ‘‘believe,’’ ‘‘continue,’’ ‘‘could,’’ ‘‘estimate,’’ ‘‘expect,’’ ‘‘intend,’’ ‘‘may,’’ ‘‘plan,’’ ‘‘potential,’’ ‘‘predict,’’ ‘‘project,’’ ‘‘target,’’ ‘‘should,’’ ‘‘would,’’ and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Forward-looking statements in this presentation include statements regarding the initiation, timing, progress and results of the Company’s preclinical studies and its research and development programs, including the Company’s expectation to achieve human proof-of-concept data for EDIT-401 by end of 2026 and identify and disclose an additional target cell type or tissue by the end of 2025; the potential of, and expectations for, EDIT-401; the timing or likelihood of regulatory filings and approvals, including submission of an IND or CTA for EDIT-401 by mid-2026; and the Company’s expectations regarding its cash runway. The Company may not actually achieve the plans, intentions, or expectations disclosed in these forward-looking statements, and you should not place undue reliance on these forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in these forward-looking statements as a result of various important factors, including: uncertainties inherent in the initiation and completion of preclinical studies and clinical trials; availability and timing of results from preclinical studies and clinical trials; expectations for regulatory approvals to conduct trials; that the outcome of preclinical testing may not be indicative of the results of clinical trials and early clinical trials may not be predictive of the outcomes of later clinical trials; that preclinical data of one compound may not be comparable with clinical data for another compound; that the market opportunity for EDIT-401 may not be as significant as the Company expects; and uncertainties with respect to the availability of resources and financing sufficient to fund the Company’s foreseeable and unforeseeable operating expenses and capital expenditure requirements. These and other risks are described in greater detail under the caption “Risk Factors” included in the Company’s most recent Annual Report on Form 10-K, which is on file with the Securities and Exchange Commission, as updated by the Company’s subsequent filings with the Securities and Exchange Commission, and in other filings that the Company may make with the Securities and Exchange Commission in the future. Any forward-looking statements contained in this presentation represent the Company’s views only as of the date hereof and should not be relied upon as representing its views as of any subsequent date. Except as required by law, the Company explicitly disclaims any obligation to update any forward-looking statements.

Agenda and Speakers

- Welcome and Strategic Overview
- EDIT-401 Overview & Opportunity
- Upregulation Strategy & EDIT-401 Preclinical Data
- Anticipated Key Milestones & Closing Remarks
- Q&A

SPEAKERS

Gilmore O'Neill, MB, MMSc
President and Chief Executive Officer

Linda Burkly, P.h.D
Chief Scientific Officer

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Differentiated Science

Strategic Execution

Best-in-Class *In Vivo* Editing Pipeline

- **Differentiated upregulation-only strategy** unlocks novel therapeutic mechanisms not addressable by traditional gene disruption or correction
- Solved for delivery using **proprietary, targeted LNPs (tLNPs) that allow targeting of multiple tissues**
- **"Plug 'n play" *in vivo* editing** enables rapid development of new therapies by simply reprogramming guide RNAs
- Driven management team with a **proven track record** of drug development and commercialization and focus on execution
- Strong cash position with **operational runway into Q2 2027**

EDIT-401: A Potential Best-In-Class, *In Vivo* Gene Editing Medicine to Transform the Hyperlipidemia Treatment Paradigm

Robust preclinical efficacy data with a **~90% mean reduction of LDL-C**[1]

Potential **one-time treatment** designed for lifelong benefit

Sizeable market potential with favorable health care system economics[1]

Attractive business model with expected typical biopharma margins[1]

Compelling preclinical data supporting rapid progression to **human proof-of-concept data by end of 2026**

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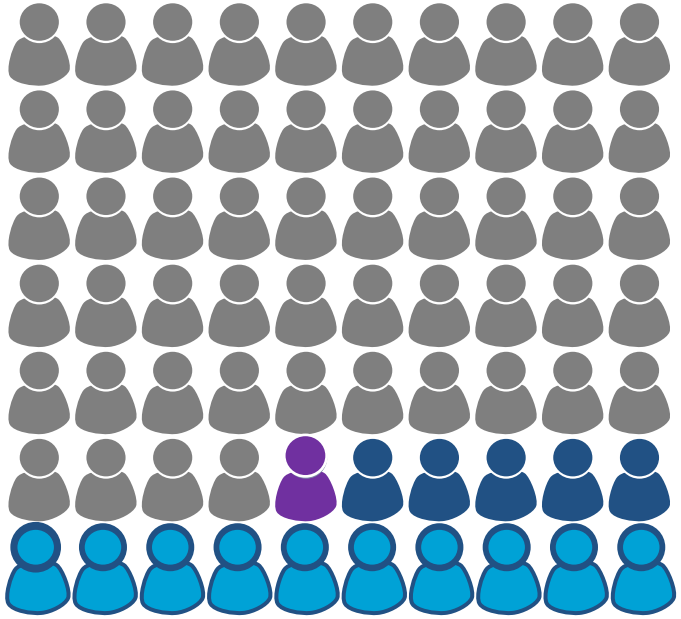
LDL-C, low-density lipoprotein cholesterol
1. Editas Medicine. Data on file.

Large Opportunity Across At-Risk Hyperlipidemia Patient Populations

- ASCVD is the **leading cause of death worldwide**[1]
- National US expenditures expected to reach over **$300 billion by 2035**[2]
- **~75% of patients** with established cardiovascular disease **do not achieve LDL-C goals**[3, 5]
- Lower LDL-C is well correlated to **reduced risk of cardiovascular events**[1]
- Standard of care requires **multiple therapies** and **life-long administration**[1]

70 million US patients with hyperlipidemia[4]



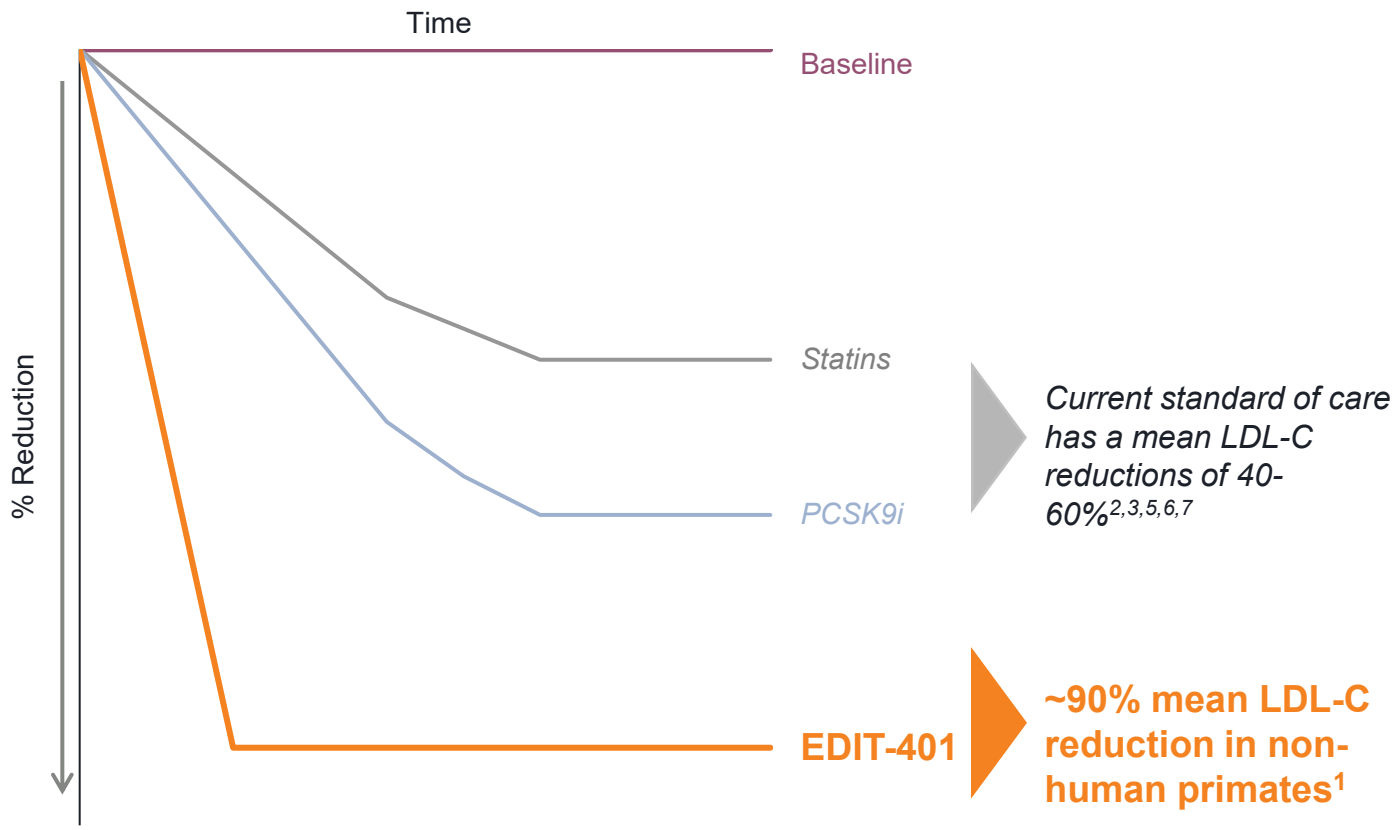
Unmet need across multiple at-risk segments of patients with hyperlipidemia

ASCVD, atherosclerotic cardiovascular disease; LDL-C, low-density lipoprotein cholesterol; HeFH, heterozygous familial hypercholesterolemia

1. Arnett DK *et al*. *Circulation* 2019; 140 (11): e596–e646; 2. Khera R et al. J Am Heart Assoc. 2020;9:e017793; 3. Gu J *et al*. *Am J Prev Cardiol* 2022; 10: 100336; 4. Editas Medicine. Data on file. 5. Klimchak AC et al. Am J Prev Cardiol 2020;1:100010

Transformative LDL-C Lowering Potential with EDIT-401

Current Treatments Provide Insufficient Reduction of LDL-C



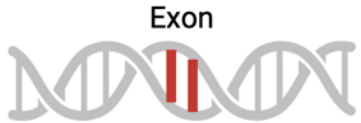
EDIT-401 Uniquely Positioned

- Intensively reducing LDL-C long-term provides maximal benefit[4,5]
- KOLs confirm ~90% mean LDL-C reduction would be transformative for the management of hyperlipidemia[1]

LDL-C, low-density lipoprotein cholesterol; KOL, key opinion leader

1. Editas Medicine. Data on file. 2. Arca M et al. J Am Heart Assoc. 2023;12 3. Goldenberg et al. Vascular Health and Risk Management 2009:5 369–376. 4. Nawrocki JW et al. Arterioscler Thromb Vasc Biology 1995;15:678-682. 5. Robinson JG et al. N Engl J Med 2015; 372:1489-1499. 6. Koskinas KC et al. J Am Coll Cardiol 2019;74:2452–62. 7. Cohen et al. N Engl J Med 2006;354:1264-72. 8. Gaba et al. *Circulation.* 2023; 147(16):1192-1203.

Editas' Differentiated *In Vivo* Gene Editing Upregulation Strategy

	editas MEDICINE	Other Approaches	
Therapeutic strategy	**Functional upregulation***	Knockdown	Gene correction
Gene Editing approach			
Non gene Editing modality		siRNA, antisense oligos, monoclonal antibody, and small molecule (pill)	
Patient population	**All patients (mutation agnostic)**	All patients (mutation agnostic)	Subset of patients (single mutation)
Therapeutic potential	**First/best-in class opportunities for loss of function diseases; cannot be addressed via knockdown**	Diseases that can be addressed by protein reduction similar to ASO and siRNA	Correction limited to subset of all patients with given disease

*editing of regulatory region, e.g., 5' or 3' region to upregulate a wild type allele or functional homolog to address loss of function or deleterious mutations

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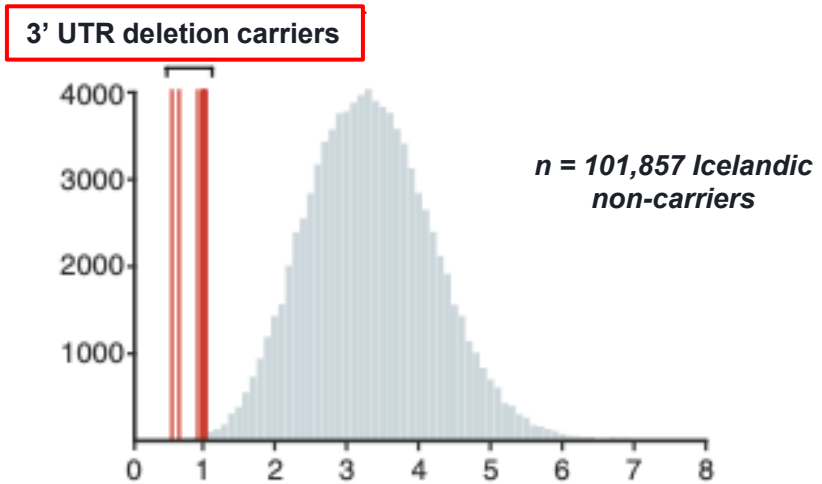
Therapeutic Strategy for EDIT-401 De-Risked by Human Genetics

7 Icelandic family members identified as carriers of partial *LDLR* 3' UTR deletion[1]

Plasma LDL-C levels[1]



No Adverse Impacts on Health[1]

LDL-C:

- **13-72 mg/dL** in plasma

LDLR:

- **1.5–2.5-fold** higher surface LDLR

Safety:

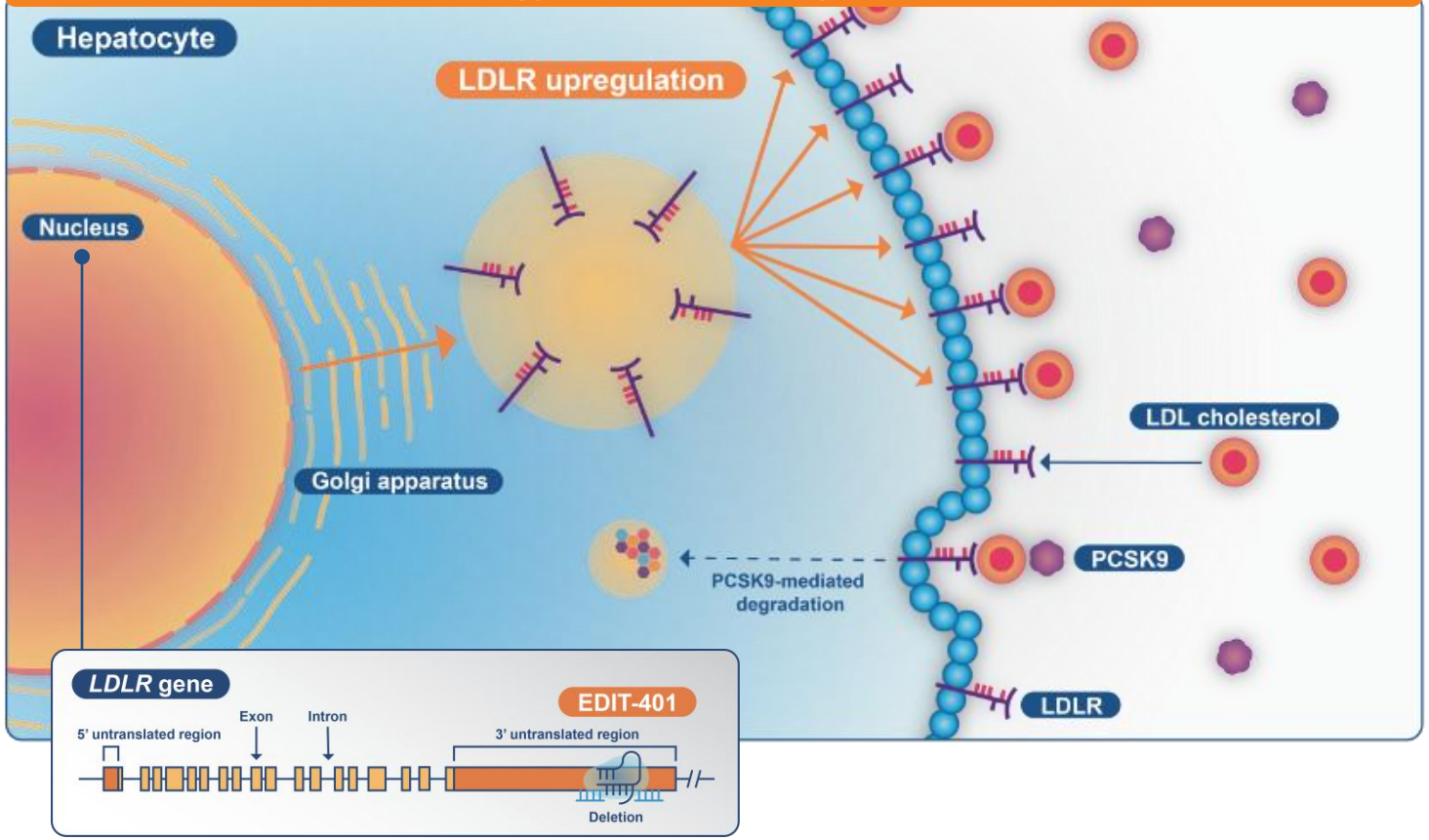
- No adverse events

LDL-C, low-density lipoprotein cholesterol; LDLR, low-density lipoprotein receptor; 3' UTR, three prime untranslated region

1. Bjornsson *et al*. *Circ Genom Precis Med* 2021;14:e003029

Robust LDL-C Reduction Driven by at Least 6-Fold Mean Increase in LDLR Protein Upregulation

EDIT-401 Therapeutic Strategy for LDLR Upregulation



Deletion of regulatory elements increases the stability of the LDLR mRNA resulting in increased production of LDLR protein

EDIT-401

EDIT-401 increased LDLR protein by at least 6-fold mean in preclinical studies[1]

PCSK9

Targeting PCSK9 prevents LDLR degradation, resulting in limited increase in LDLR protein[2,3]

LDLR, low-density lipoprotein receptor; PCSK9, proprotein convertase subtilisin/kexin type 9.

References: 1. Editas Medicine. Data on file. 2. https://biocytogen.com/gene-humanized-models/b-hpcsk9-mice. Accessed Aug 29, 2025. 3. Rashid et al. *PNAS* 2005;102(15): 5374 –5379.

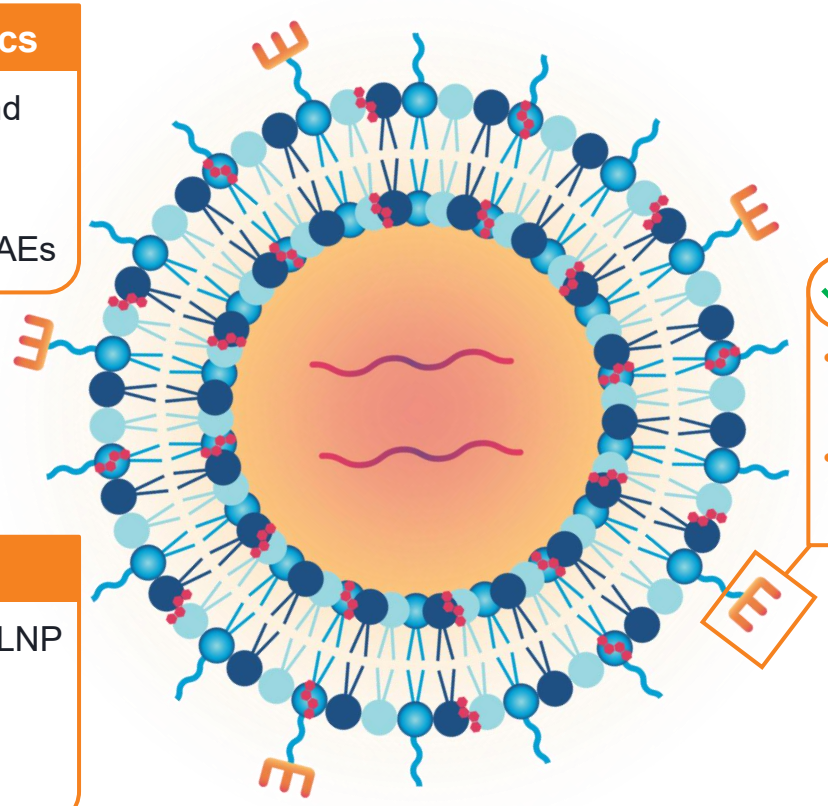
EDIT-401 Combines Editas' CRISPR and LNP Expertise

Gene Target Informed by Human Genetics

- Excision of LDLR 3′ UTR segment with Cas9 and dual gRNAs
- De-risked strategy – models naturally occurring gain-of-function deletion to lower LDL-C with no AEs

LNP Delivery Strategy

- Encapsulation of mRNA and gRNAs in a single LNP for transient expression of drug product
- Clinically validated LNP components through strategic partnership with Genevant



GalNAc-Targeted LNP for Liver Delivery

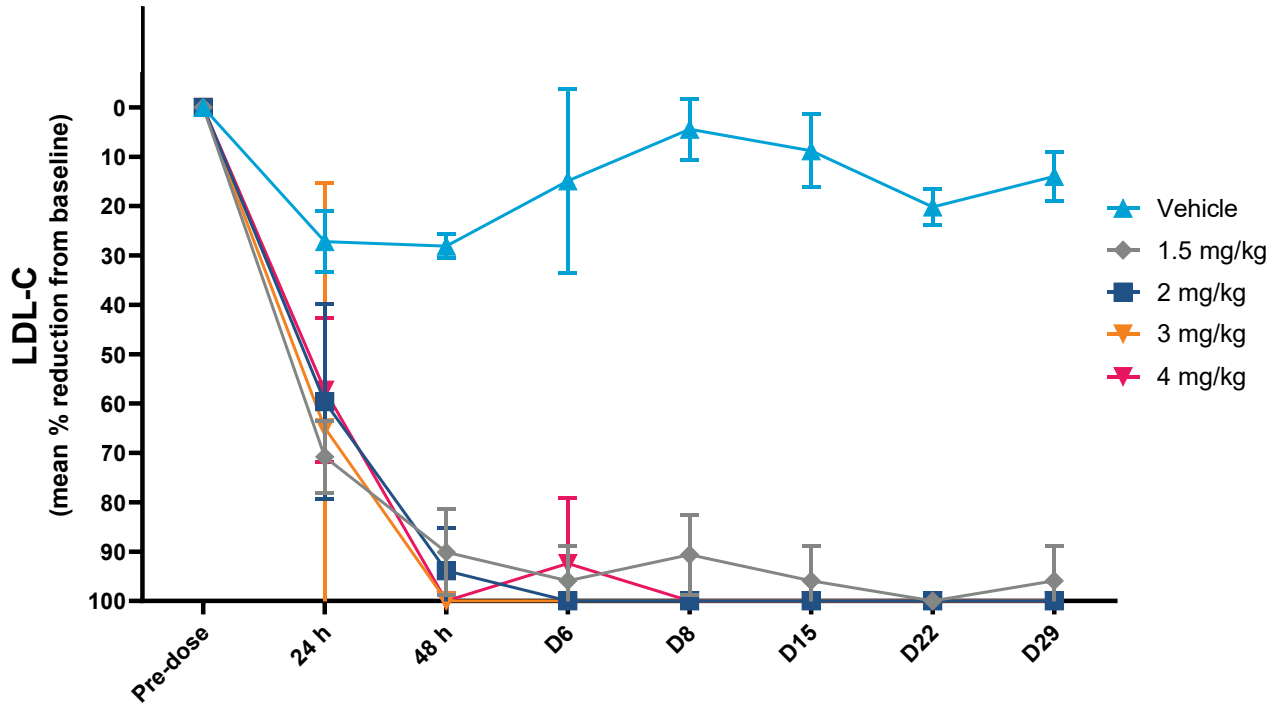
- GalNAc targeting moiety enabling delivery if lacking sufficient LDLR
- GalNAc-LNP leveraged through strategic partnership with Genevant

Integration of multiple unique components has enabled EDIT-401 to achieve ~90% mean LDL-C reduction in preclinical studies

LNP, lipid nanoparticle; AE, adverse event; Cas9, CRISPR-associated protein 9; CRISPR, clustered regularly interspaced short palindromic repeats; GalNAc, N-acetylgalactosamine; gRNA, guide RNA; LDL-C, low-density lipoprotein cholesterol; LDLR, low-density lipoprotein receptor; c; mRNA, messenger RNA; RNA, ribonucleic acid; 3′ UTR, three prime untranslated region.

EDIT-401 Achieved ~90% Mean LDL-C Reduction in NHPs

Non-human Primates[1]



- ✓ ~90% mean LDL-C reduction at each dose level
- ✓ Single dose
- ✓ At least 6-fold mean increase in LDLR protein upregulation
- ✓ No adverse clinical observations noted
- ✓ Transient increases in liver enzymes resolved within 1 week

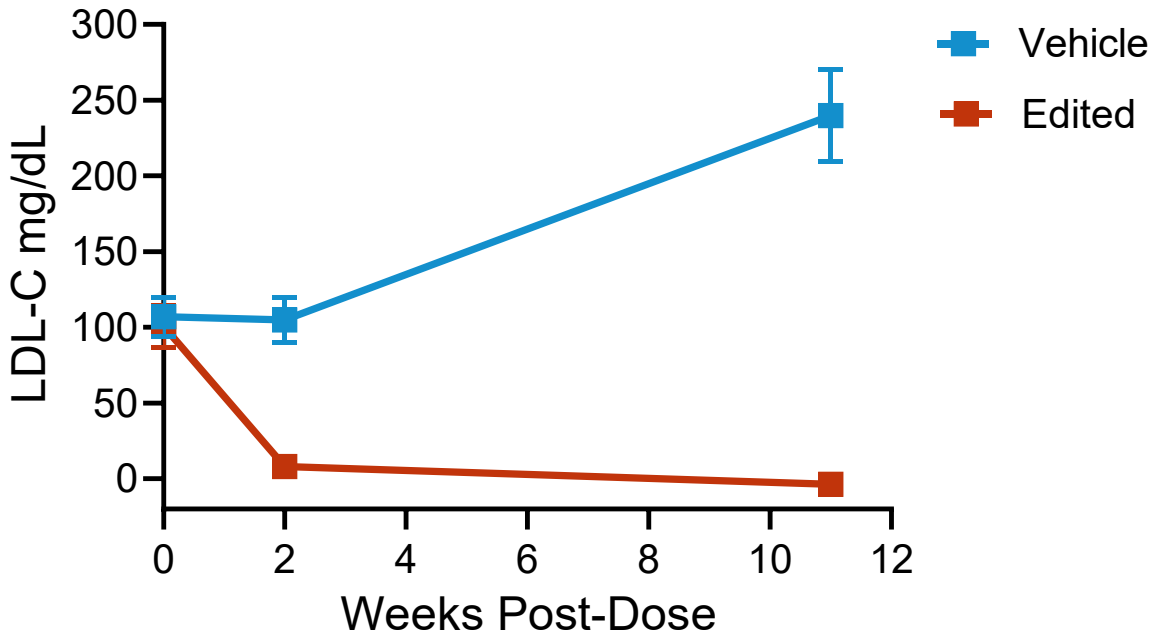
Pre-dose LDL-C was averaged across 2 timepoints to account for variability in measurements

LDL-C, low-density lipoprotein cholesterol; LDLR, low-density lipoprotein receptor

1. Editas Medicine. Data on file.

EDIT-401 Achieved ~90% Mean LDL-C Reduction in Mice with High Baseline LDL-C

Wild-type Mice on High-Fat Diet[1]



Mice on a high fat diet had 3-fold elevated baseline LDL-C compared to mice on a regular fat diet

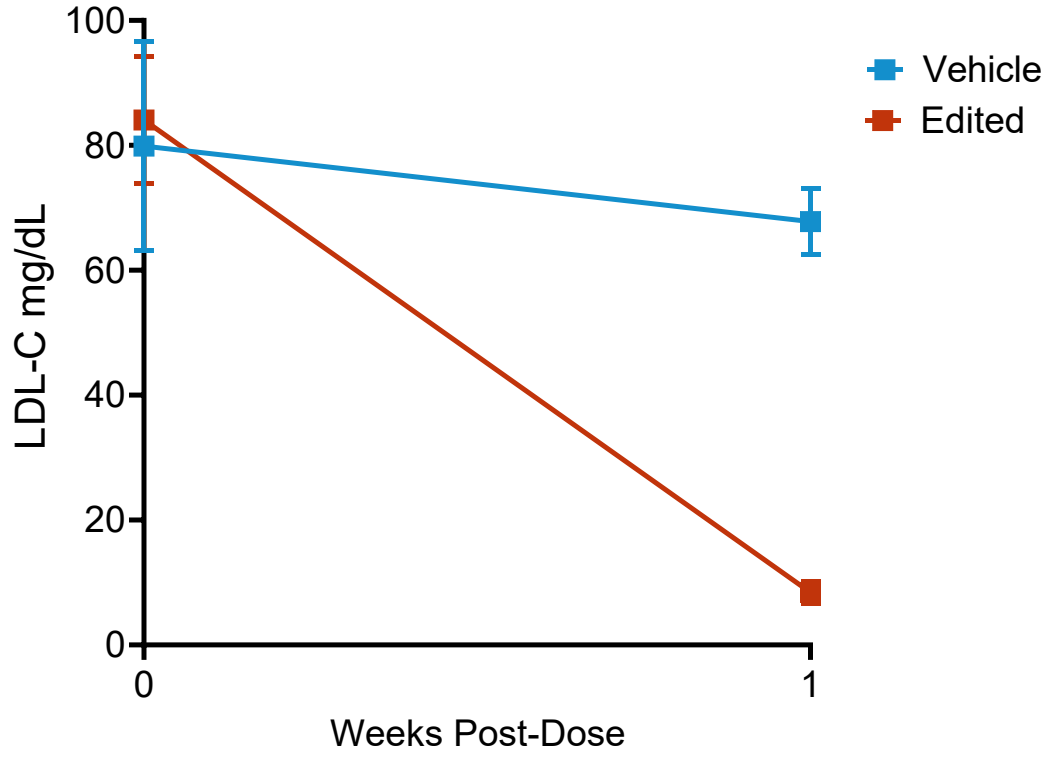
- ✓ ~90% mean LDL-C reduction
- ✓ Single dose
- ✓ Greater than 10-fold LDLR protein upregulation

LDL-C, low-density lipoprotein cholesterol; LDLR, low-density lipoprotein receptor

1. Editas Medicine. Data on file.

EDIT-401 Achieved ~90% Mean LDL-C Reduction in Mice Heterozygous for LDLR Loss-of-Function

Mice Heterozygous for LDLR Loss-of-Function[1]



Mice heterozygous for LDLR loss-of-function had 2-fold increase in baseline LDL-C

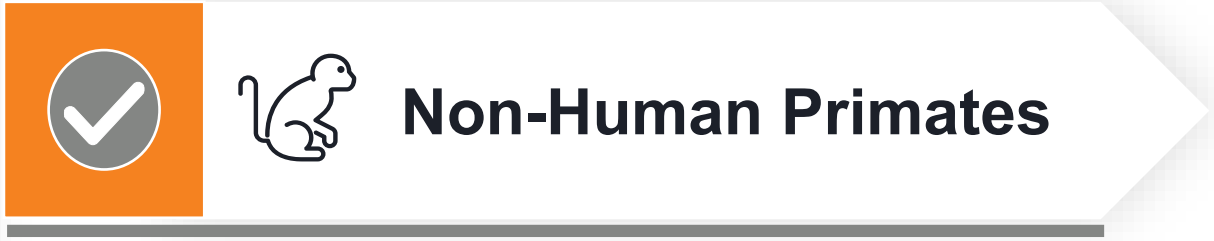
- ✓ ~90% mean LDL-C reduction
- ✓ Single dose
- ✓ Greater than 10-fold LDLR protein upregulation

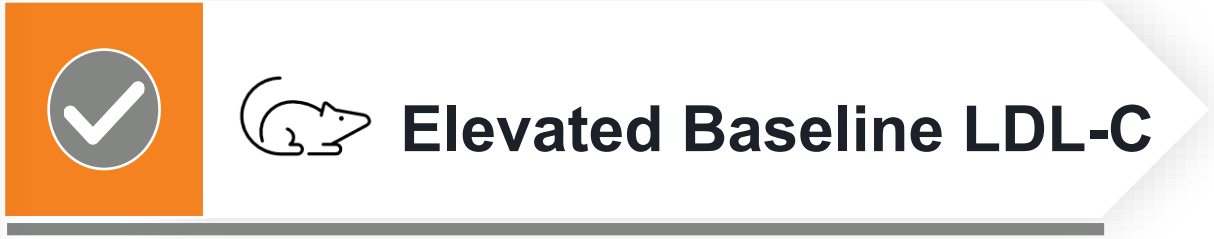
LDL-C, low-density lipoprotein cholesterol; LDLR, low-density lipoprotein receptor

1. Editas Medicine. Data on file.

Potential to Deliver Meaningful Benefits Across the Hyperlipidemia Population

EDIT-401
achieved
~90% mean LDL-C reduction





LDL-C, low-density lipoprotein cholesterol; LDLR, low-density lipoprotein receptor
1. Editas Medicine. Data on file.

EDIT-401: Editas' Lead Development Candidate

A potential best-in-class, in vivo *gene editing medicine to transform the hyperlipidemia treatment paradigm*

Robust preclinical efficacy data with a **~90% mean reduction of LDL-C**[1]

Potential **one-time treatment** designed for lifelong benefit

Sizeable market potential with favorable health care system economics[1]

Attractive business model with expected typical biopharma margins[1]

Preclinical data supports rapid progression to **human proof-of-concept** data by **end of 2026**

LDL-C, low-density lipoprotein cholesterol
1. Editas Medicine. Data on file.

Key Anticipated Milestones & Strategic Priorities

Selection of EDIT-401 reaffirms our strategic vision to be a leader in* in vivo *programmable gene editing with a potential best-in-class medicine that could meaningfully impact patients and the healthcare system

Questions & Discussion

Gilmore O'Neill, MB, MMSc
President and Chief Executive Officer

Linda Burkly, PhD
Chief Scientific Officer

Amy Parison
Chief Financial Officer