Pioneering the Possible in Gene Editing

April 2026

Forward Looking Statements

This presentation contains forward-looking statements and information within the meaning of The Private Securities Litigation Reform Act of 1995. 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 planned clinical trials, including the Company's expectation to achieve early human proof-of-concept data for EDIT-401 by end of 2026 and complete enrollment in the EDIT-401 clinical trial (dose finding portion) with topline data results available in 2027; the timing for the Company's receipt and presentation of data from its preclinical studies, including presenting additional preclinical data for EDIT-401 by mid-2026; the potential of, and expectations for, EDIT-401 and the Company's other future *in vivo* product candidates; and the timing or likelihood of regulatory submissions and approvals, including the timing of submission of an IND/CTA for EDIT-401 by mid-2026. 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, timing, progress, and results of preclinical studies and clinical trials; uncertainty regarding availability and timing of results from preclinical studies and clinical trials; uncertainties relating to planned regulatory submissions to initiate clinical trials, including that results of preclinical studies will warrant such submissions or that regulatory agencies may require additional preclinical studies, that regulatory submissions shall occur on the expected timelines and that regulatory authorities will provide clearance for trials to be initiated; and that the Company will not be able to raise funding sufficient for its 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.

editas MEDICINE

A leader in transformative in vivo gene editing

Differentiated Upregulation Strategy

Novel therapeutic mechanisms unlocked by functionally upregulating protein expression

Delivery to Target Tissues

Delivery platform enables precise delivery of *in vivo* gene editing medicines across tissue types (liver and others)

Transformative Therapeutic Potential

Address **significant unmet needs** via one-time, durable treatment with meaningful patient and broader healthcare system impact

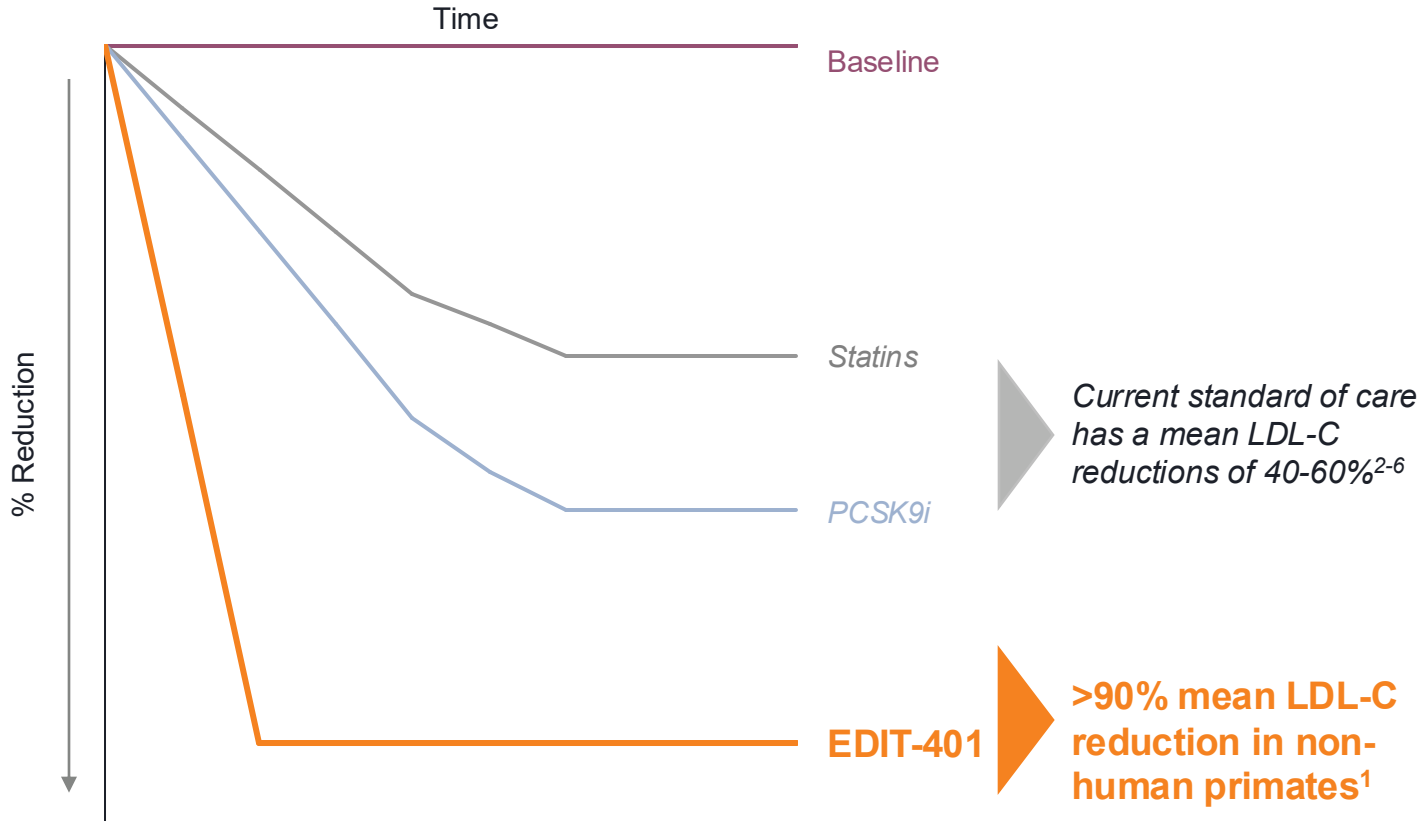
EDIT-401: Potential to Transform the Hyperlipidemia Treatment Landscape

A potential best-in-class therapy with **unprecedented >90% mean reduction in LDL-C** in non-human primates[1], **on track to achieve early human proof-of-concept data by end of 2026**

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

EDIT-401: Transformative LDL-C Lowering Potential

Average LDL-C Reduction with Current Treatments vs. EDIT-401 Potential



EDIT-401 Uniquely Positioned

- Intensive, lifelong reduction of LDL-C (<20mg/dL) provides maximal clinical benefit[7]
- Simplified treatment approach may eliminate multiple therapies with life-long administration[8]
- 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. Vasc Health Risk Manag. 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 Gaba et al. *Circulation.* 2023; 147(16):1192-1203. 8. Arnett DK et al. Circulation 2019; 140 (11): e596–e646

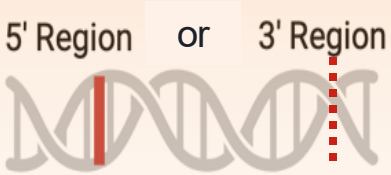
Functional Upregulation

***Differentiated use of CRISPR nuclease-based* technology**

Edits

Non-Coding, Regulatory

Regions



to *upregulate* a wild type allele or functional homolog

- ✓ **Treats diseases by increasing the level of disease mitigating protein**
- ✓ **Does not alter sequence of naturally occurring protein**
- ✓ **Genotype agnostic**

EDIT-401: Potential to Transform the Hyperlipidemia Treatment Landscape

Delivering Best-in-Class Therapy

Robust preclinical efficacy with a **>90% mean reduction of LDL-C**[1] with the potential to achieve very low LDL-C levels

Addressing Significant At-Risk Populations

~10 million U.S. patients treated for ASCVD not at goal[1] including HeFH and at-risk ASCVD patients

Aligning Patient Benefits with System Costs

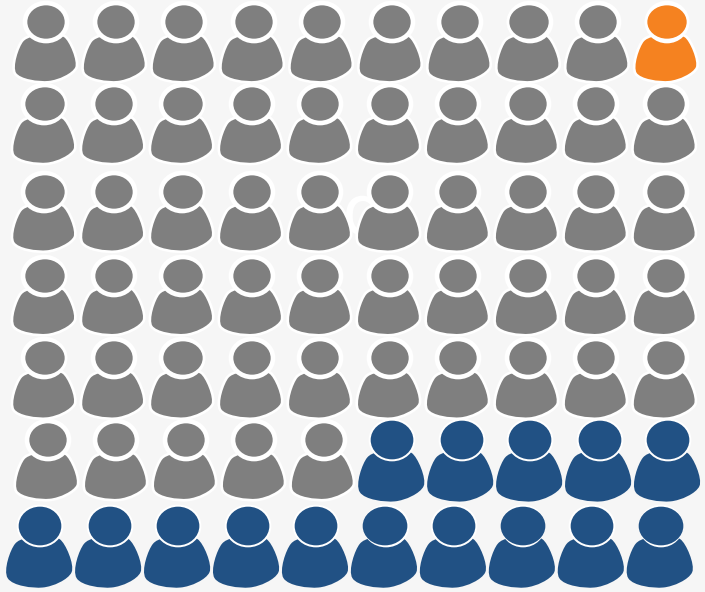
One-time treatment has potential to enable long-term patient adherence at **favorable healthcare system economics**

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

1. Editas Medicine. Data on file.

EDIT-401: Large Opportunity Across At-risk Hyperlipidemia Patient Populations

70 million US patients with hyperlipidemia[1]



Heterozygous Familial Hypercholesterolemia

Highly prevalent monogenic genetic disorder that leads to elevated LDL-C[2]

HeFH

1.2 Million
people are affected by HeFH[2]

~10–20x
increased risk of ASCVD[3,4]

97%
of HeFH patients **do not meet LDL-C goals**[6]

At Risk ASCVD

Driven by cholesterol-rich plaque accumulation in the arteries

ASCVD

~15 Million
US adults with established ASCVD[1]

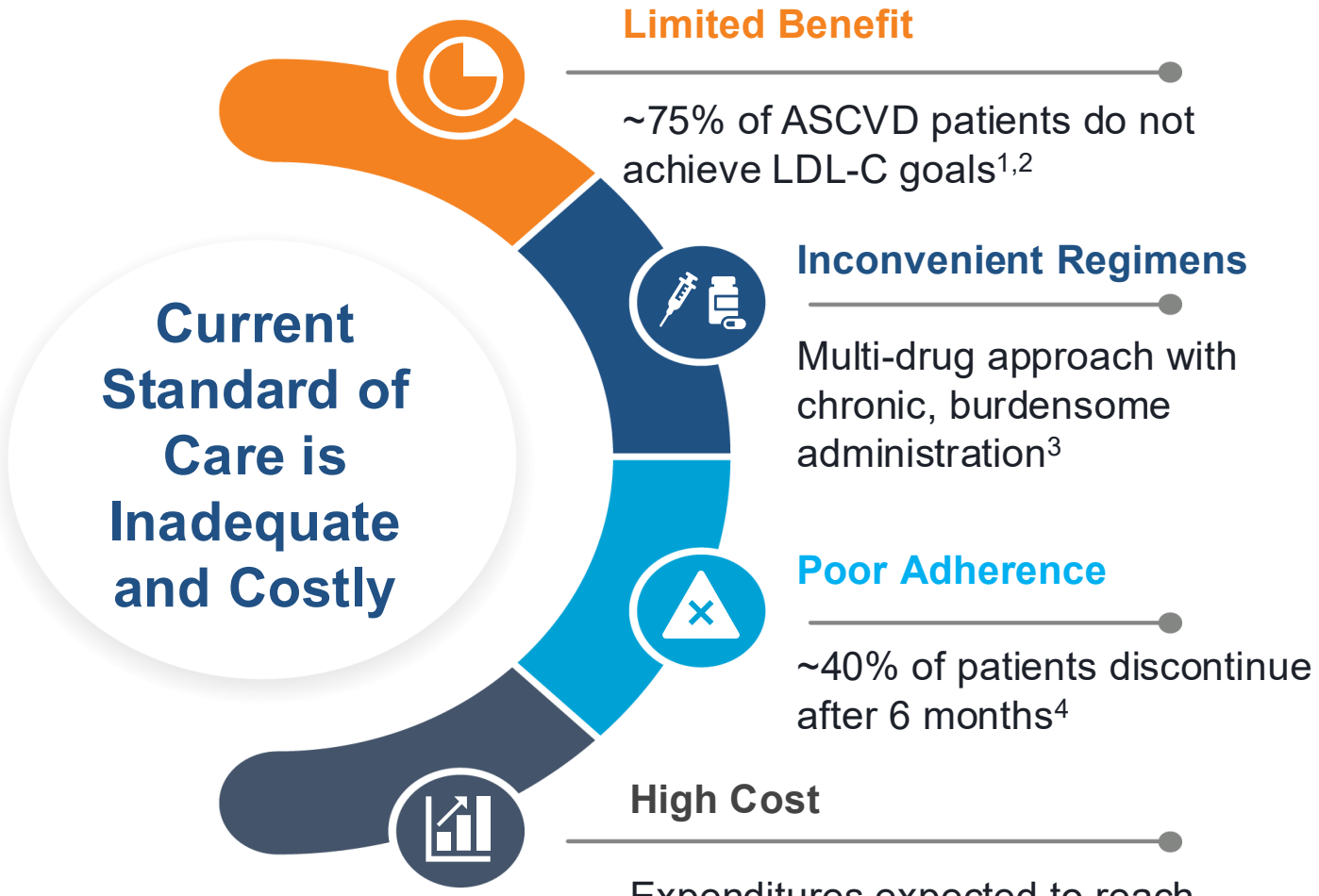
~33%
patients experience a second event[5]

~75%
of patients with ASCVD **do not meet LDL-C goals**[7,8]

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

1. Editas Medicine. Data on file. 2. Akioyamen LE, et al. BMJ Open. 2017;7(9):e016461 3. Khera AV. *et al. J Am Coll Cardiol.* 2016Jun 7;67(22):2578-89 4. Tada H *et al. Eur Heart J*. 2017 21;38:1573-1579 5. Mackinnon et al, CJC 2021 Oct 20;4(2):206–213 6. EAS Familial Hypercholesterolaemia Studies Collaboration (FHSC). Lancet. 2021; 398 (10312): 1713–1725 7. Gu J et al. Am J Prev Cardiol 2022 10: 100336; 8. Klimchak AC et al. Am J Prev Cardiol 2020;1:100010

EDIT-401: Potential to Address Significant Burden on Healthcare Systems



EDIT-401 is poised to transform the treatment landscape and align clinical economic value

- ✓ Best in class LDL-C reduction
- ✓ Single dose
- ✓ Supports long-term adherence
- ✓ Decreased healthcare costs

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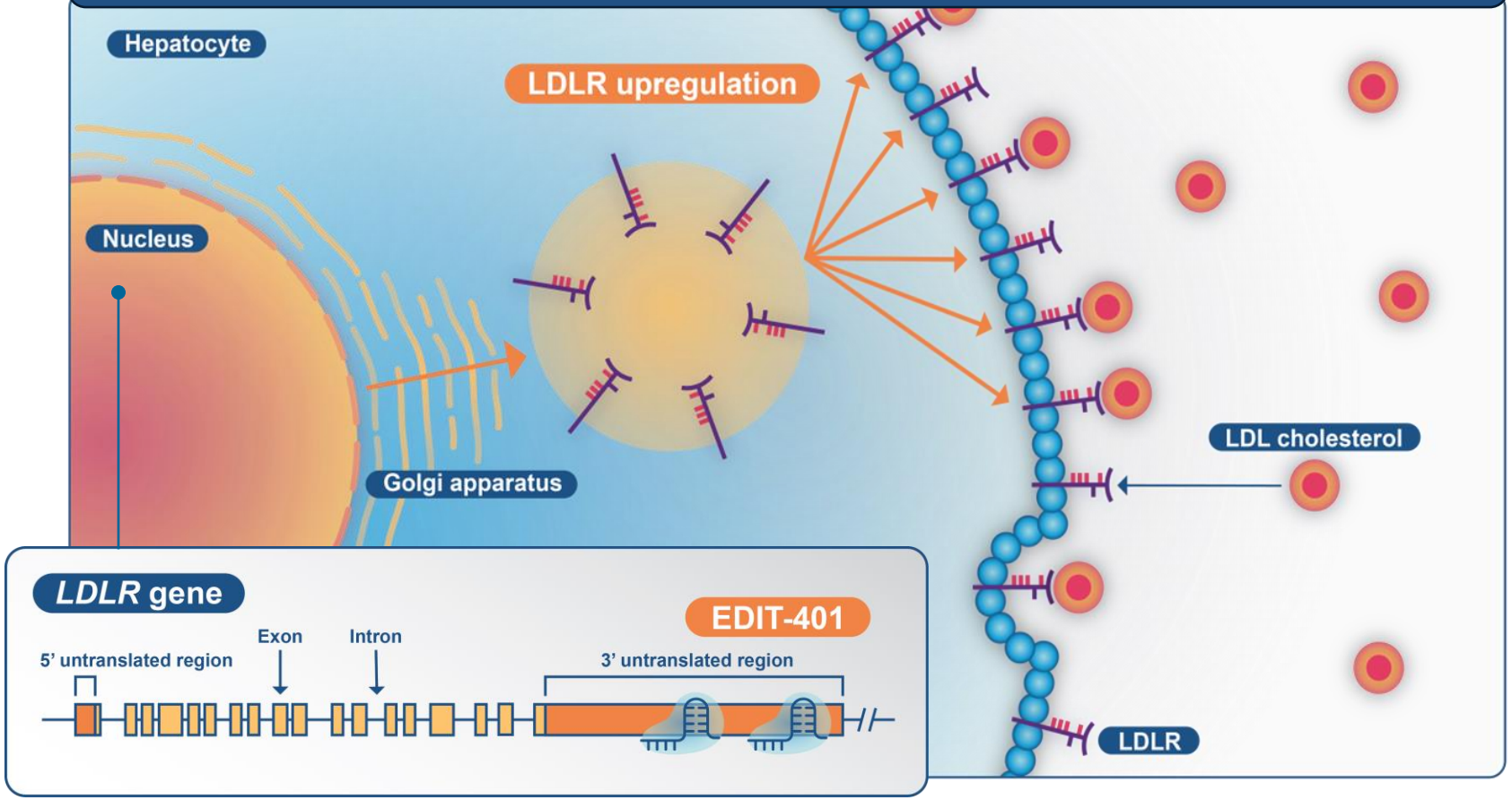
1. Gu J et al. Am J Prev Cardiol 2022; 10: 100336 2. Klimchak AC et al. Am J Prev Cardiol 2020;1:100010 3. Di Girolamo F, Pellin L, Roni C. Eur J Intern Med. 2025;140 4. Donald DR et al. J Clin Lipidol. 2022;16(3):315–324 5. Khera R et al. J Am Heart Assoc. 2020;9:e017793; https://doi.org/10.1161/CIR.0000000000001258

EDIT-401: Potential Best-in-class LDL-C Reduction

EDIT-401 Therapeutic Strategy for LDLR upregulation



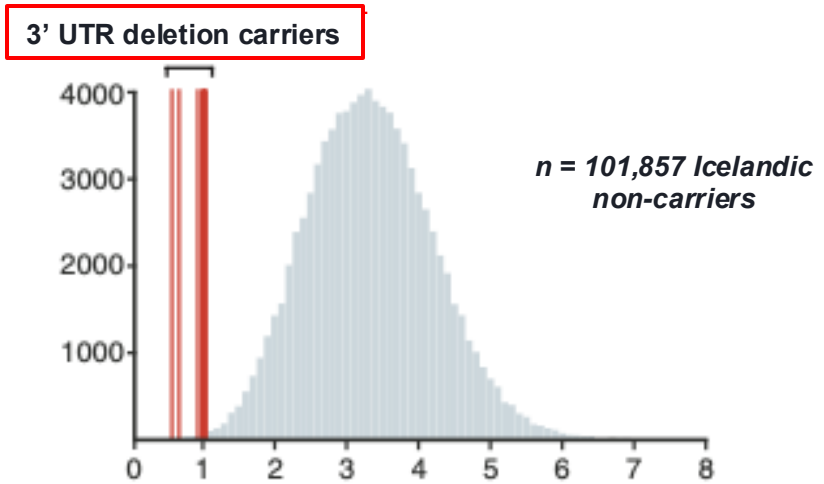
- Edits **non-coding, regulatory** regions
- **~10-40% functional editing** achieves **>90% mean LDL-C reduction in preclinical studies**[1]
- At least **6-fold mean LDLR protein** increase in preclinical studies[1]
- Differentiated **upregulation strategy** allows for potential best-in-class LDL-C reduction

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

EDIT-401: Therapeutic Strategy 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:

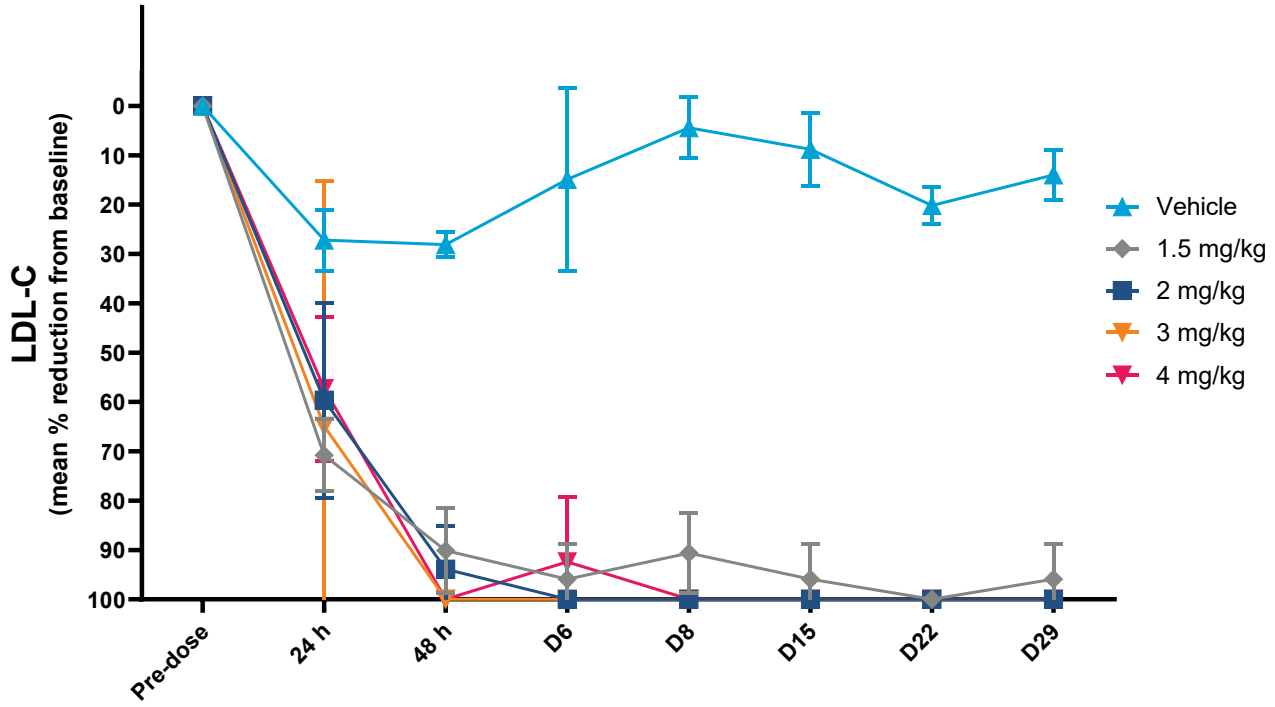
- No adverse events

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

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

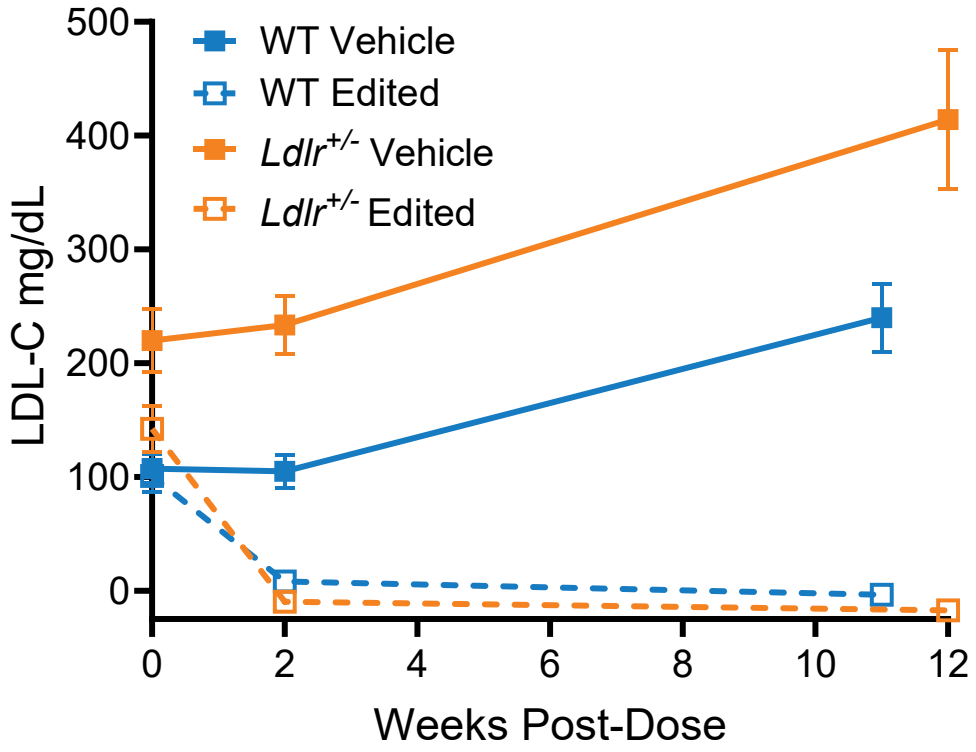
Pre-dose LDL-C was averaged across 2 timepoints to account for variability in measurements

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 Wildtype Mice with LDLR Loss-of-Function and High LDL-C

Wildtype Mice and Mice Heterozygous for LDLR Loss-of-Function with High Baseline LDL-C[1]



- ✓ >90% mean LDL-C reduction
- ✓ Single dose
- ✓ At least 8-fold mean increase in LDLR protein upregulation

Mice on a high-fat diet had ≥3-fold elevated baseline LDL-C compared with mice on a regular-fat diet. N=5 for all WT and *Ldlr*$^{+/-}$ groups. *Ldlr*$^{+/-}$ Edited, 100% mean LDL-C reduction from baseline at 12 weeks; WT Edited, 99% mean LDL-C reduction from baseline at 11 weeks.

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

1. Editas Medicine. Data on file.

Editas' Differentiated *In Vivo* Gene Editing Strategy

	editas MEDICINE Functional Upregulation	Genetic Knockdown	Genetic Correction	Non-genetic Knockdown*
Edits Non-Coding Regions	✓	✗	✗	✗
Addresses All Patients**	✓	✓	✗	✓
One-time, Curative Potential	✓	✓	✓	✗
Differentiated Therapeutic Strategy	✓			

Harnessing the power of CRISPR gene editing and differentiated therapeutic strategy to develop transformative medicines for people living with serious diseases

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*siRNA, antisense oligos, monoclonal antibody, small molecule
**Mutation-agnostic

Driving Transformative Medicines Forward

EDIT-401: Key Anticipated Milestones

2026

- Present additional preclinical data for EDIT-401 by mid-2026
- Submit IND / CTA for EDIT-401 by mid-2026
- Achieve early *in vivo* human proof of concept for EDIT-401 by end of 2026

2027

- Complete enrollment in clinical trial (dose-finding portion) with topline data results available in 2027

- ☑ *Delivering* Best-in-Class Therapy
- ☑ *Addressing* Significant At-Risk Populations
- ☑ *Aligning* Patient Benefits with System Costs

Programs Positioned for Development