Delivering Durable Medicines: Bold Science in Motion

November 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 planned clinical trials, including the Company’s expectation to complete enrollment in Part 1, the dose-finding portion of the clinical trial of EDIT-401, with topline safety and efficacy data results available in the second half of 2027; the timing for the Company’s receipt and presentation of data from its preclinical and planned clinical studies, including reporting initial safety and tolerability data in the clinical trial of EDIT-401 in the first quarter of 2027; the potential of, and expectations for, EDIT-401; 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, 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 on the expected timelines or at all; and uncertainties as to whether the Company’s cash resources are sufficient to fund its foreseeable and unforeseeable operating expenses and capital expenditure requirements for the period anticipated. 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 Differentiated Genetic Medicine Engine

Accessible, Durable Medicines: Single-dose, Lifelong Benefit

Challenge the status quo and change the course of disease

Focus on simple-to-use, single-dose medicines with low COGS manufacturing to enable commercial viability



Harness gene editing to unlock new possibilities, such as increasing levels of disease-mitigating proteins

Maximize probability of clinical success through human-validated targets, the right editing strategy, and purpose-built delivery

EDIT-401 Lead Program with the Potential to Transform Hyperlipidemia Standard of Care

Editas' Differentiated *In Vivo* Gene Editing Strategy

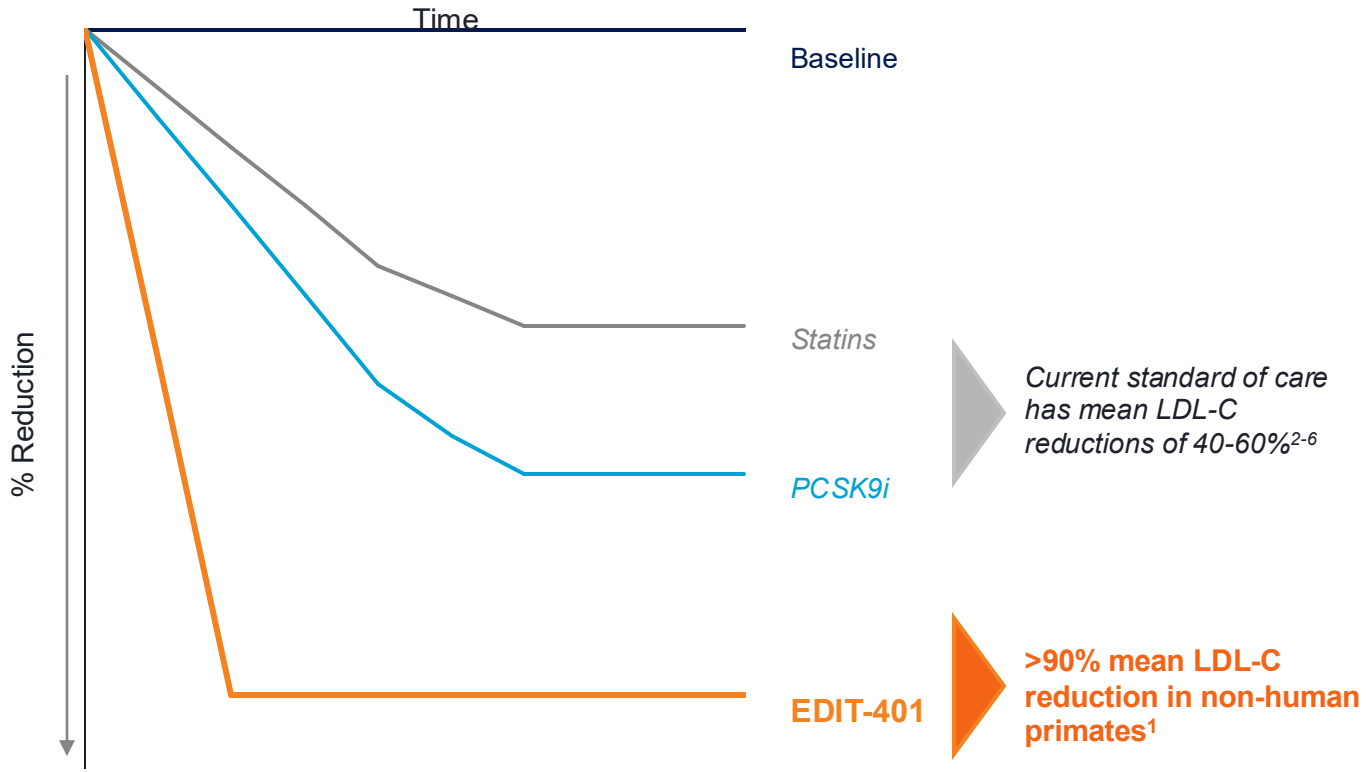
	editas MEDICINE Functional Upregulation	Genetic Knockdown	Genetic Correction	Non-genetic Knockdown*
Edits Non-Coding Regions	✓	✗	✗	✗
Addresses All Patients**	✓	✓	✗	✓
One-time, Curative Potential	✓	✓	✓	✗
Differentiated Therapeutic Strategy	✓			

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

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

EDIT-401: Transformative LDL-C Lowering Potential

Average LDL-C Reduction with Current Treatments and EDIT-401 Potential



EDIT-401 Uniquely Positioned

- Intensive, lifelong reduction of LDL-C provides maximal clinical benefit[7]
- 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

EDIT-401: Potential to Transform the Treatment of Hyperlipidemia

Delivering Best-in-Class Therapy



Single dose of EDIT-401 **achieved ~90% or greater mean reduction** across multiple atherogenic lipoproteins including **LDL-C and Lp(a)** in NHPs

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



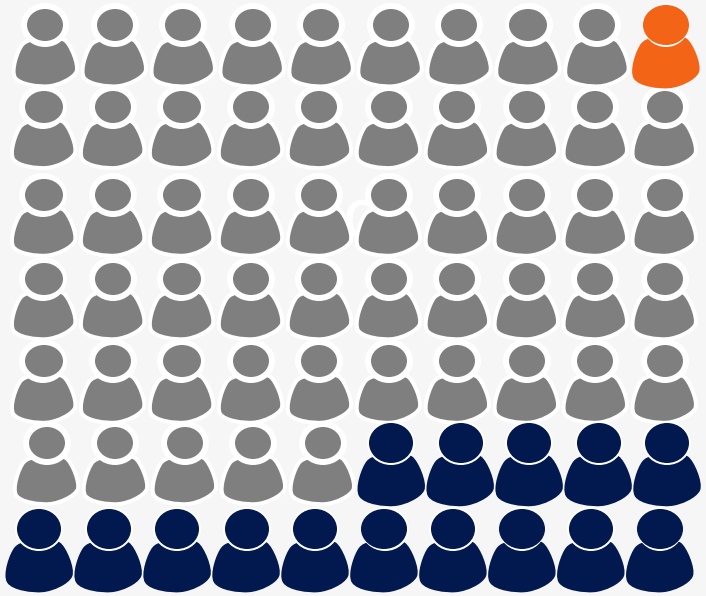
One-time treatment has potential to enable long-term patient benefit with **favorable healthcare system economics**

LDL-C, low-density lipoprotein cholesterol; Lp(a), lipoprotein(a); NHPs, non-human primates; 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]



HeFH

Heterozygous Familial Hypercholesterolemia

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

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]

ASCVD

At Risk ASCVD

Driven by cholesterol-rich plaque accumulation in the arteries

~15 Million
US adults with established ASCVD[1]

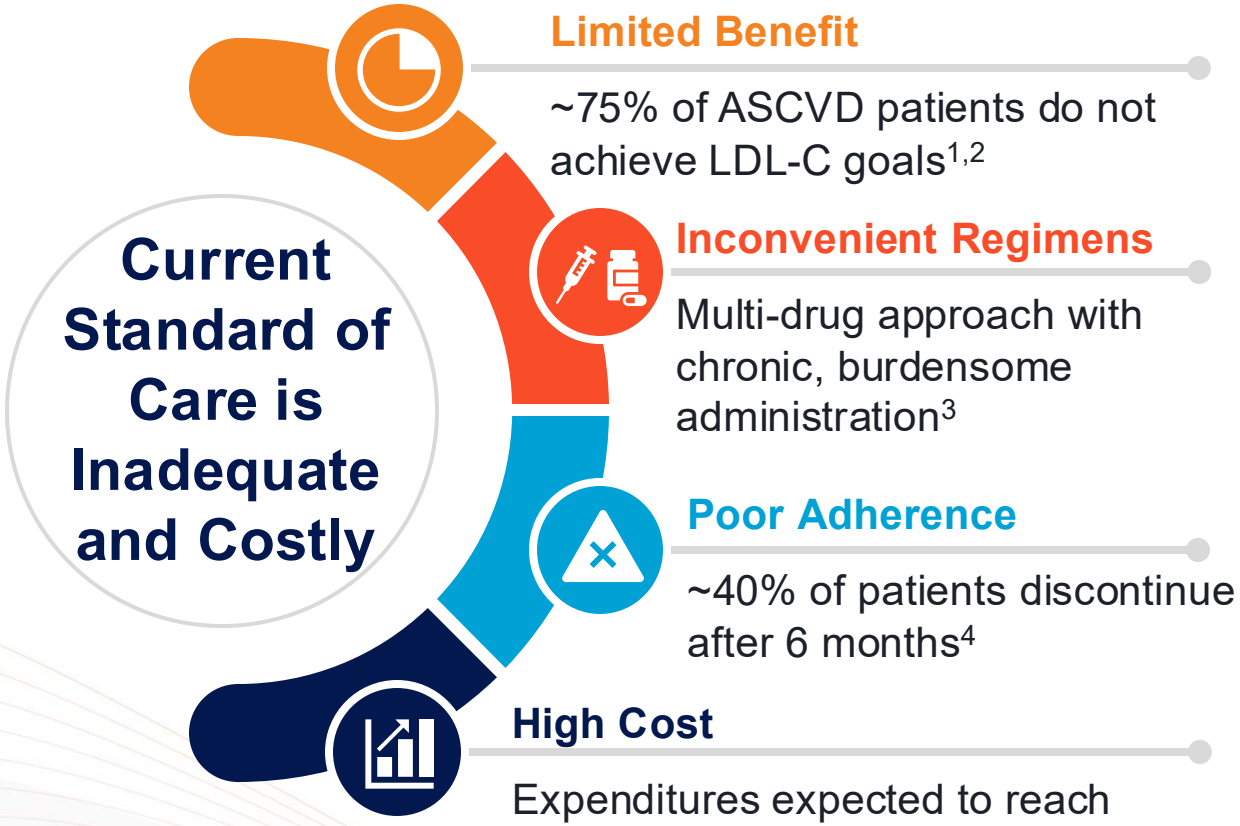
~33%
patients experience a second event[5]

~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 and economic value

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

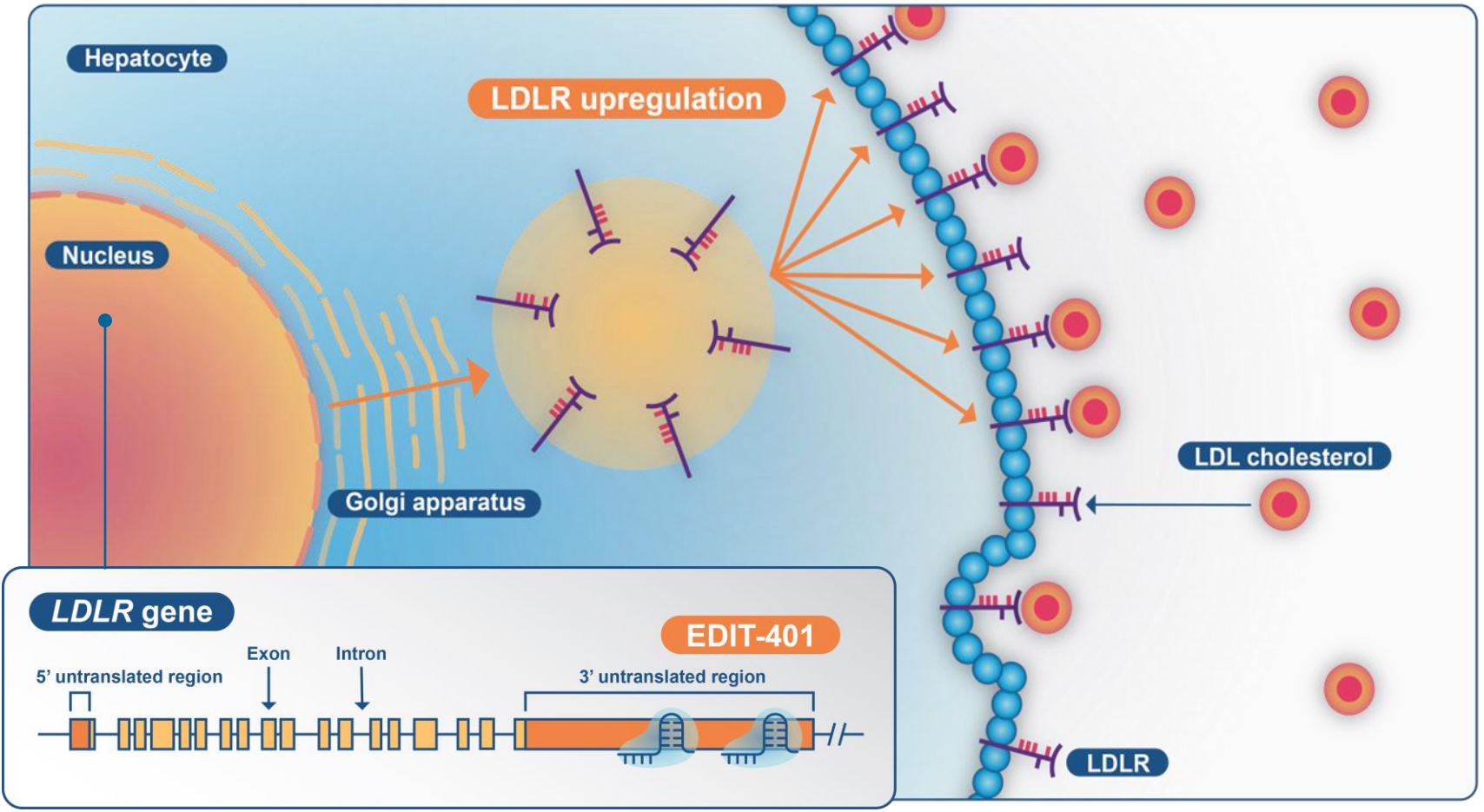
ASCVD, atherosclerotic cardiovascular disease; LDL-C, low-density lipoprotein cholesterol

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

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

EDIT-401 Therapeutic Strategy for LDLR upregulation



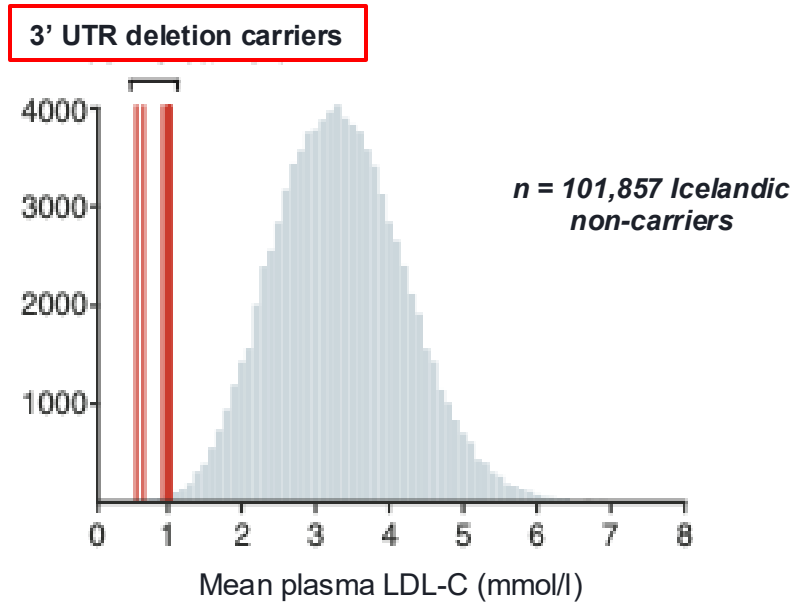
- Edits **non-coding, regulatory** regions
- ~10-40% functional editing achieves **>90% mean LDL-C reduction** in preclinical studies[1]
- At least 6-fold mean LDLR protein increase in preclinical studies[1]
- Differentiated **upregulation strategy** allows for potential best-in-class LDL-C reduction

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

1. Editas Medicine. Data on file.

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

Plasma LDL-C levels[1]



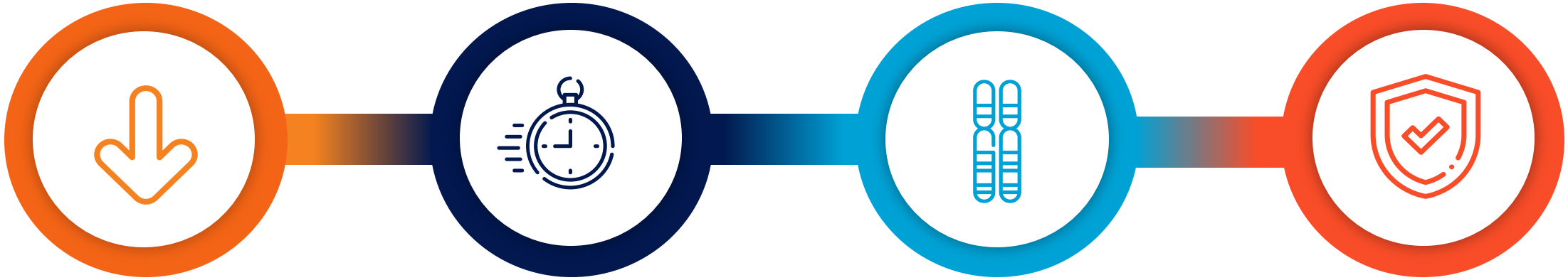
No Adverse Events Observed in Icelandic Family Members

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

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% or Greater Mean Reduction in LDL-C and Lp(a) in NHPs



A single dose of EDIT-401 **achieved ~90% or greater mean reduction in LDL-C and Lp(a)** across all dosed NHPs[1]

LDL-C reductions remained durable through ~10 months across evaluated dose range in NHPs[2]

Reductions in LDL-C and Lp(a) were highly correlated, highlighting potentially transformative impact of LDLR upregulation[1]

Promising preclinical safety profile with no adverse clinical observations at therapeutically relevant dose[2]

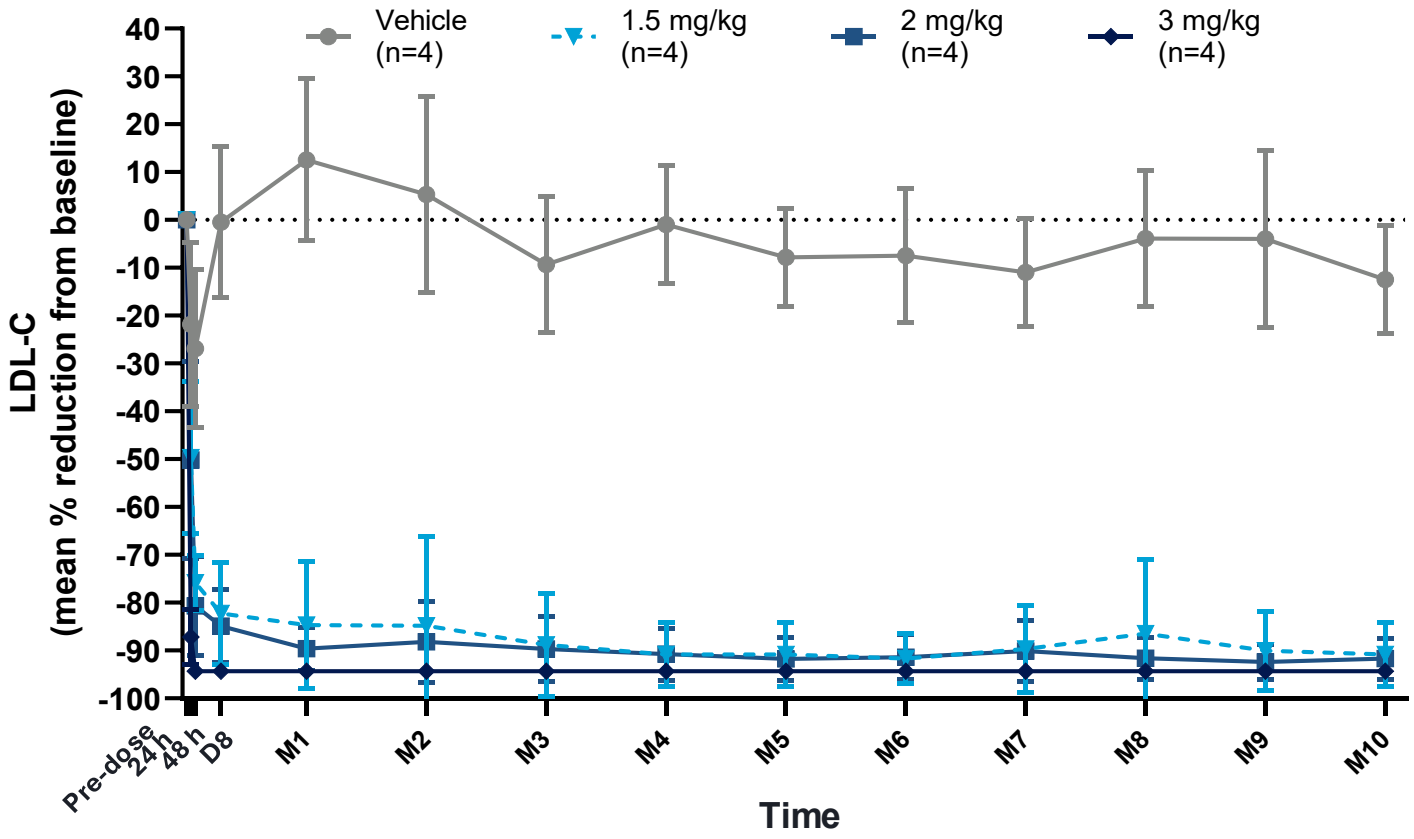
Unifying mechanism of LDLR upregulation facilitates robust reductions of multiple disease-causing lipoproteins

LDL-C, low-density lipoprotein cholesterol; Lp(a), lipoprotein(a); ApoB, apolipoprotein; LDL-C, low-density lipoprotein cholesterol; NHP, non-human primate; LDLR, low-density lipoprotein receptor

1. Wrighton, *et al.* EDIT-401: A potentially transformative investigational in vivo CRISPR gene editing medicine upregulates LDLR and meaningfully reduces LDL-C in non-human primates. Presented at: European Atherosclerosis Society (EAS) Congress; 2026 May 25 2. American Heart Association Scientific Sessions; Nov 2026

EDIT-401: Robust and Durable >90% Mean LDL-C Reduction in NHPs

Single dose of EDIT-401[1]



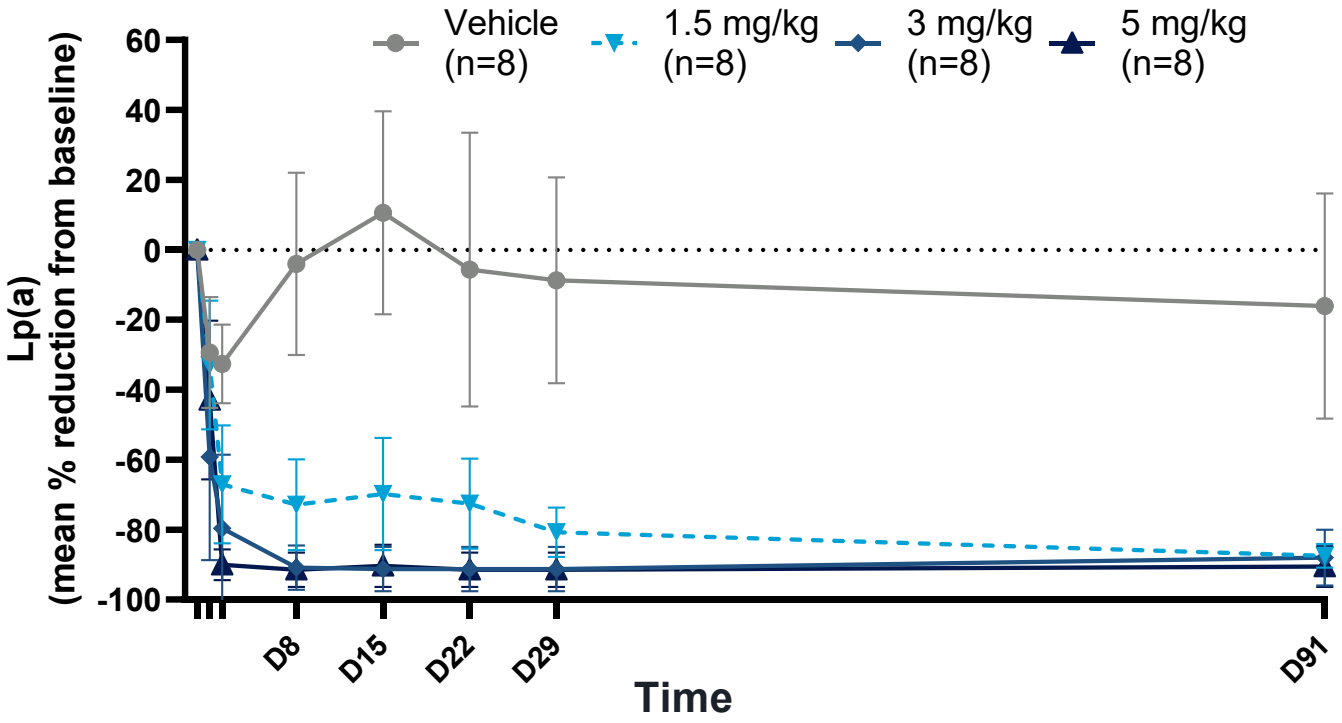
- Rapid and robust LDL-C reduction across a dose range
- At least 6-fold mean increase in LDLR protein upregulation
- Durability of LDL-C reduction at all doses through ~10 months

Mean values are shown ± SD. Values below lower limit of quantitation assigned as LLoQ/2.
D, day. h, hours; LDL-C, low-density lipoprotein cholesterol; LDLR, low-density lipoprotein receptor; LLoQ, lower limit of quantification; LNP, lipid nanoparticle; M, month; SD, standard deviation.
LNPs licensed from Genevant Sciences GmbH.
1. American Heart Association Scientific Sessions; Nov 2026

EDIT-401: Up To ~90% Mean Lp(a) Reduction Achieved in NHPs

Elevated Lp(a) adds independent genetically driven risk that can persist even when LDL-C is at goal

Single dose of EDIT-401[1]



- Dose-dependent Lp(a) reduction
- Durability of Lp(a) reduction at all doses through 3 months
- Reduction in Lp(a) correlates with LDL-C and ApoB reductions
- NHP baseline Lp(a) values approximate elevated CVD risk in humans[2]

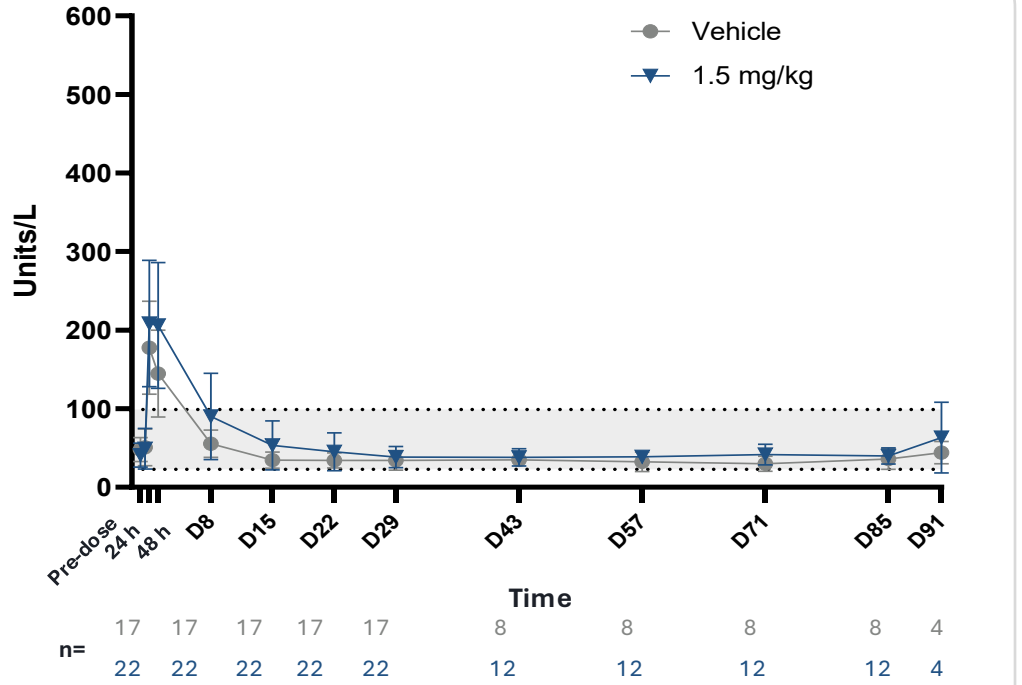
Lp(a), lipoprotein(a); LDL-C, low-density lipoprotein cholesterol; ApoB, apolipoprotein B; NHP, non-human primate; CVD, cardiovascular disease; D, day;

1. American Heart Association Scientific Sessions; Nov 2026; 2. Irvine, A *et al. Front Cardiovasc Med* 2026;12:1710557

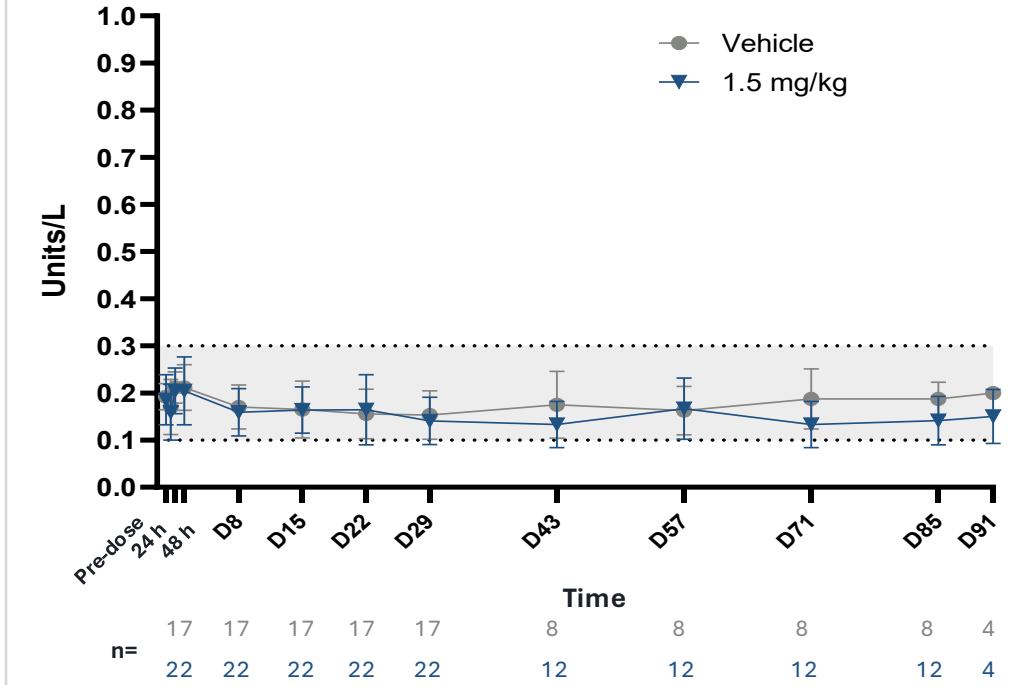
EDIT-401: No Adverse Liver Findings in NHPs

Transient ALT elevations | **No bilirubin elevations** | **No histopathologic changes**

Mean (SD) ALT[1]



Mean (SD) Bilirubin[1]



ALT and bilirubin data presented from 4 NHP studies. Shaded area represents normal range observed in NHPs. Vehicle dosed with saline following same pre-treatment regimen as EDIT-401.
ALT, alanine aminotransferase; D, day; h, hours; NHP, non-human primate; SD, standard deviation. LNPs licensed from Genevant Sciences GmbH.

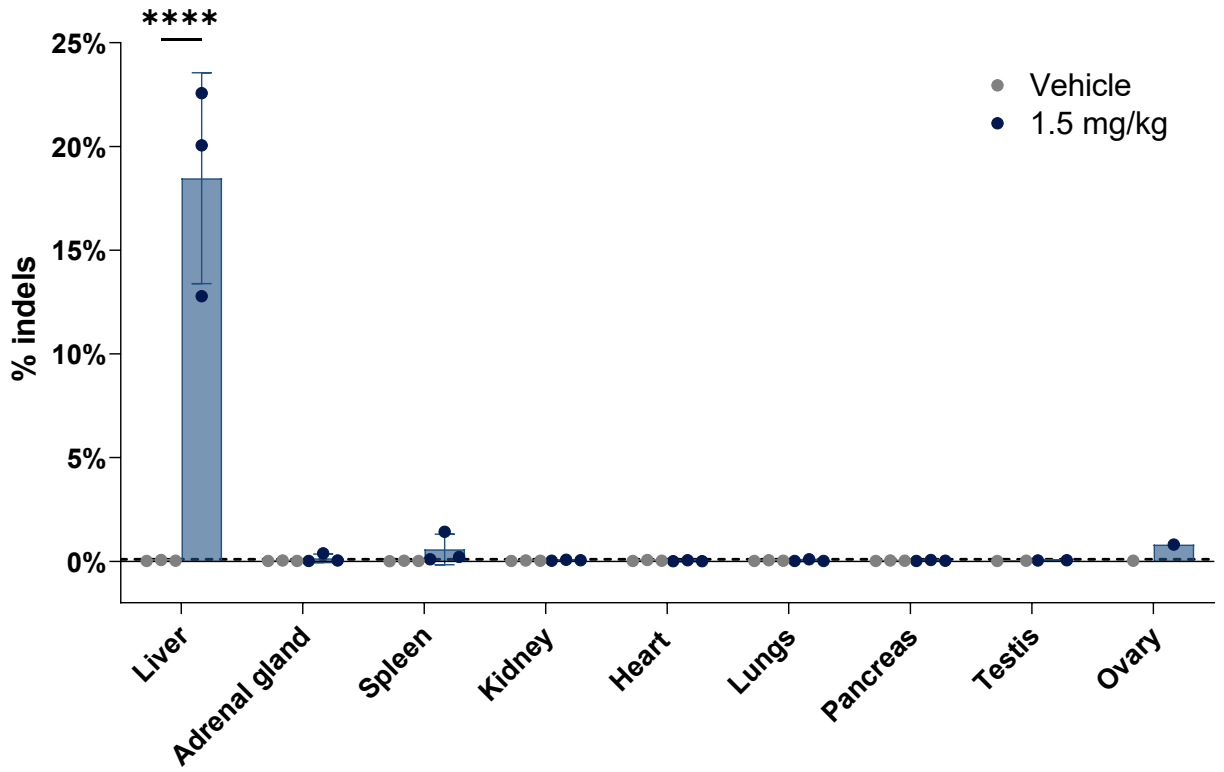
1. American Heart Association Scientific Sessions; Nov 2026

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EDIT-401: Targeted Delivery to the Liver with No Evidence of Genotoxicity in NHPs

- **High delivery to hepatocytes** with undetectable Cas9 mRNA in oocytes*
- **No off-target editing detected** in a panel of ~1500 candidate sites with frequencies of structural variants measured below LLoQ†

EDIT-401 delivers liver-targeted editing[1]



Graph show % indels in *LDLR* 3' UTR across liver and all non-target tissues. Representative data shown with one of the EDIT-401 gRNAs by targeted amplicon sequencing. ****P<0.0001

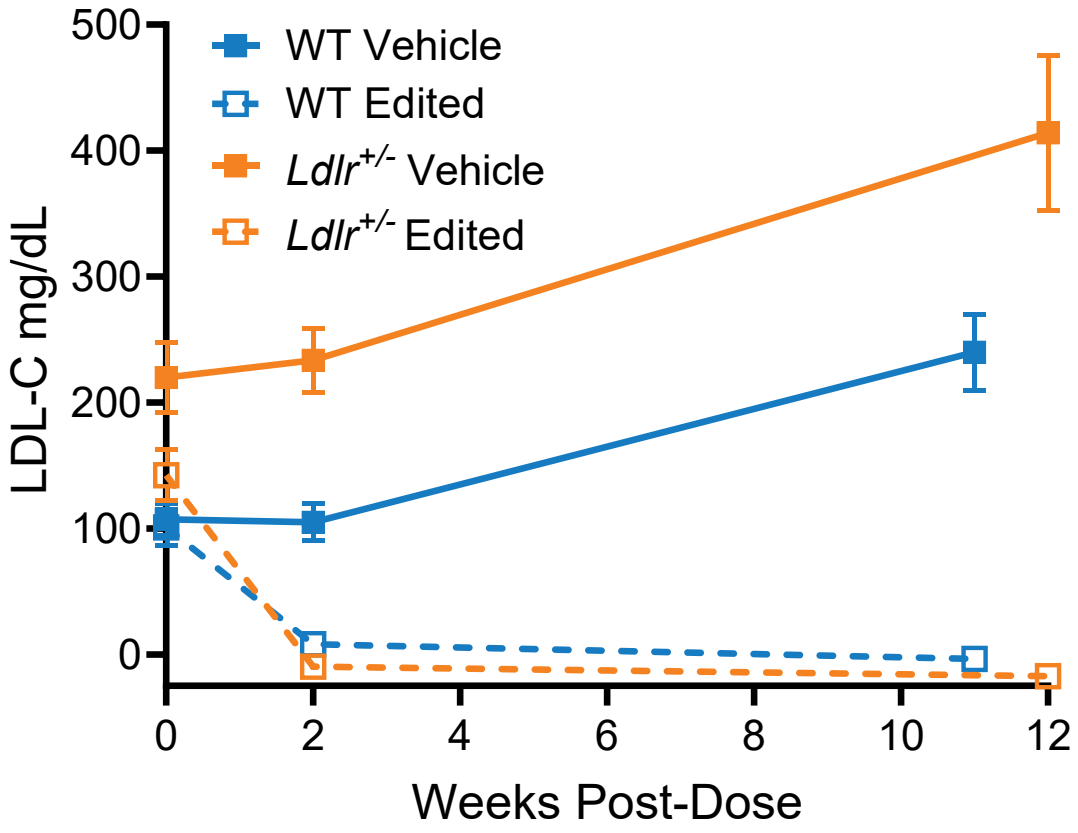
*In situ hybridization at 1.5 mg/kg. †Using an *in vitro* assessment in relevant human primary cell that represents clinical editing levels.
NHP, non-human primate; Cas9, CRISPR-associated protein 9; CRISPR, clustered regularly interspaced short palindromic repeats; LDLR, low-density lipoprotein receptor; LLoQ, lower limit of quantification; LNP, lipid nanoparticle; UTR, untranslated region. LNPs licensed from Genevant Sciences GmbH.

1. American Heart Association Scientific Sessions; Nov 2026

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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.

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Driving Durable, Transformative Medicines Forward

EDIT-401 Strive™ Study: Key Anticipated Milestones

2026

- ✓ Present additional preclinical data for EDIT-401 by mid-2026
- ✓ Submit CTN for EDIT-401 by mid-2026

2027

- Report initial safety and tolerability data for Strive study in the first quarter of 2027
- Complete enrollment in Part 1 Strive study, with topline safety and efficacy results from Part 1 available in the second half of 2027

Well capitalized to execute our strategy to advance EDIT-401, with cash runway extending to the second half of 2028

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

Programs Positioned for Development

