



Driving Durable, Transformative Medicines Forward

June 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.



Transformative Therapeutic Potential

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



Novel Therapeutic Mechanisms

Differentiated strategy 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)



EDIT-401: Potential to Transform the Hyperlipidemia Treatment Landscape

A potential best-in-class therapy with **unprecedented ~90 percent or greater mean reduction in LDL-C and Lp(a) in non-human primates¹**

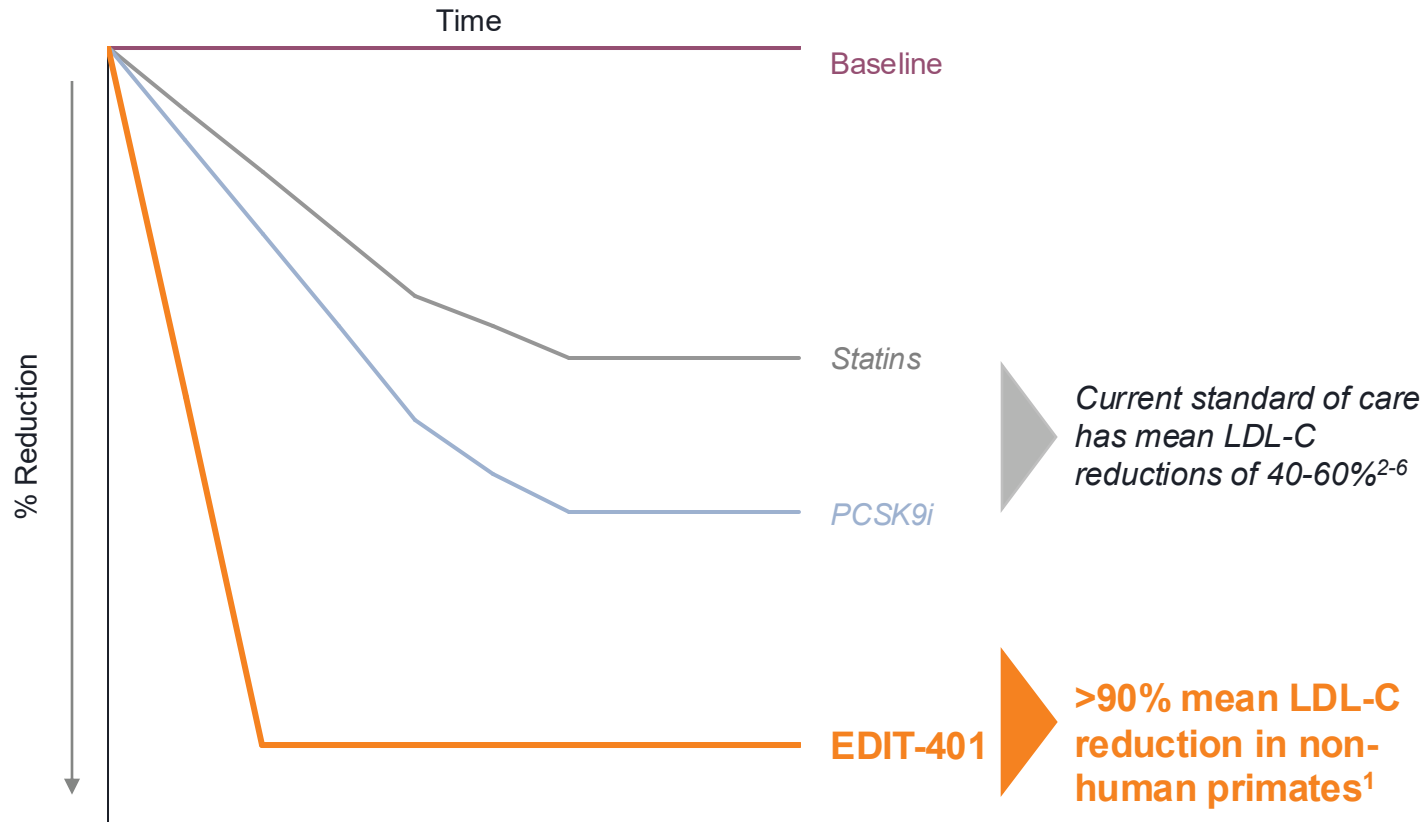
Editas' Differentiated *In Vivo* Gene Editing Strategy

	 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

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 provides maximal clinical benefit⁷



May eliminate multiple therapies with life-long administration⁸



KOLs confirm >90% mean LDL-C reduction would be transformative for the management of hyperlipidemia¹

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 goal¹ including HeFH and at-risk ASCVD patients

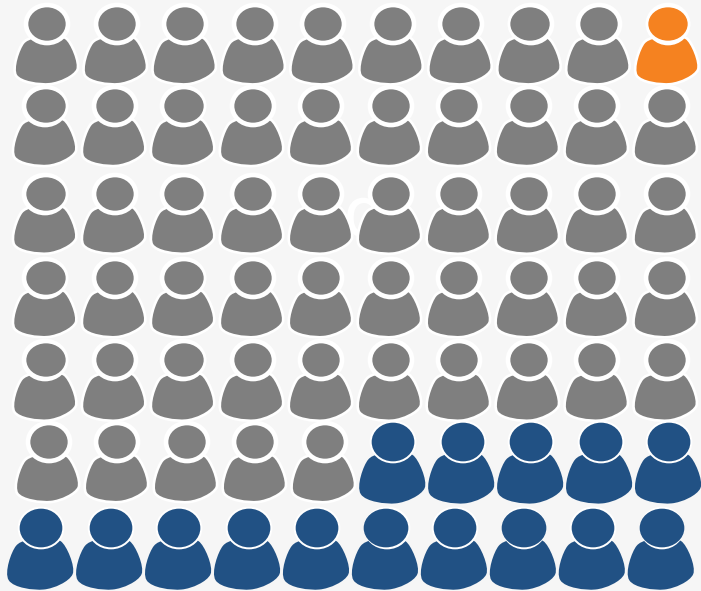
Aligning Patient Benefits with System Costs



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

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

70 million US patients with hyperlipidemia¹



HeFH

Heterozygous Familial Hypercholesterolemia

Highly prevalent monogenic genetic disorder that leads to elevated LDL-C²

1.2 Million
people are affected by HeFH²

~10–20x
increased risk of ASCVD^{3,4}

97%
of HeFH patients do not meet LDL-C goals⁶



ASCVD

At Risk ASCVD

Driven by cholesterol-rich plaque accumulation in the arteries

~15 Million
US adults with established ASCVD¹

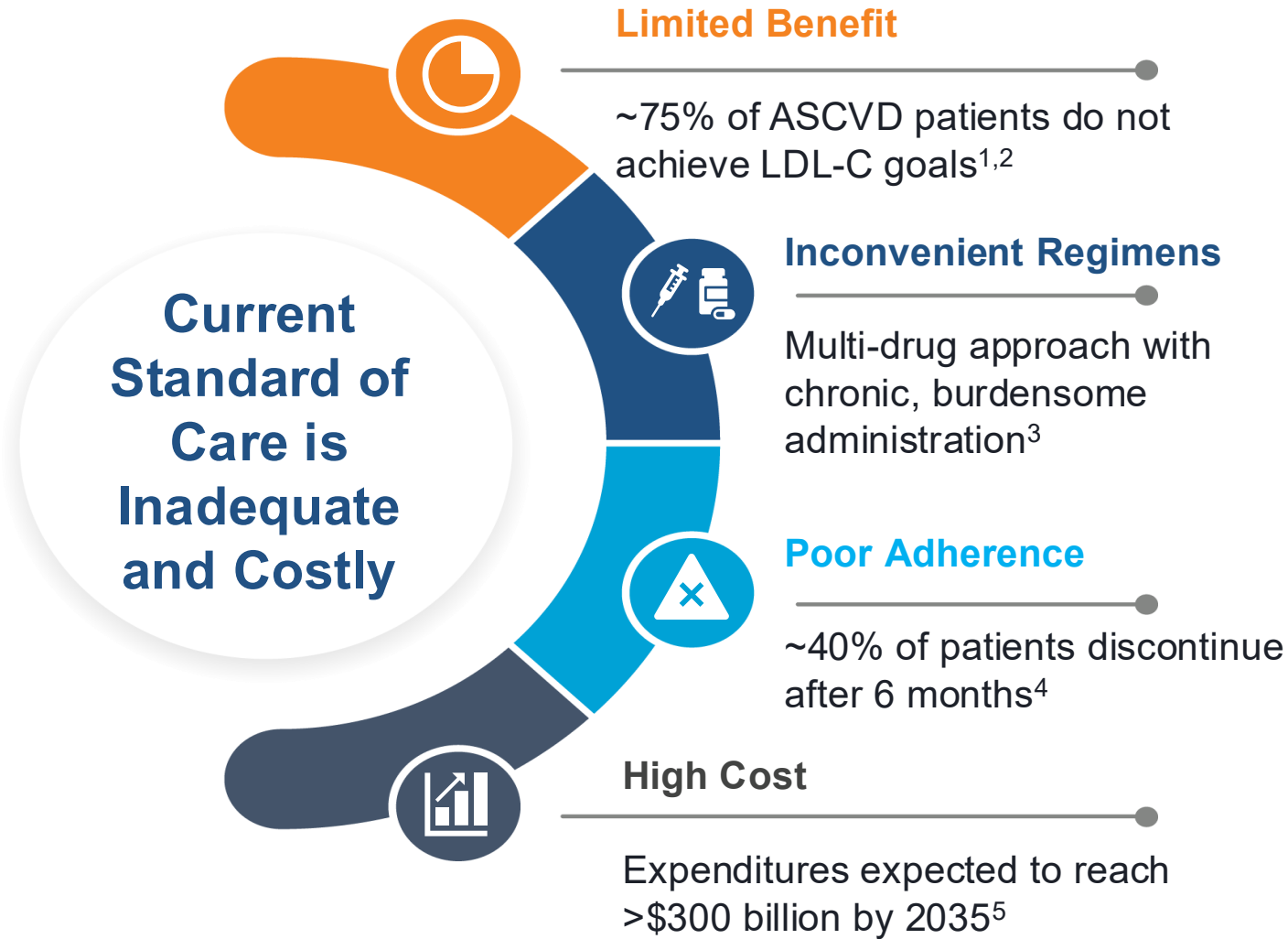
~33%
patients experience a second event⁵

~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*. 2016 Jun 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

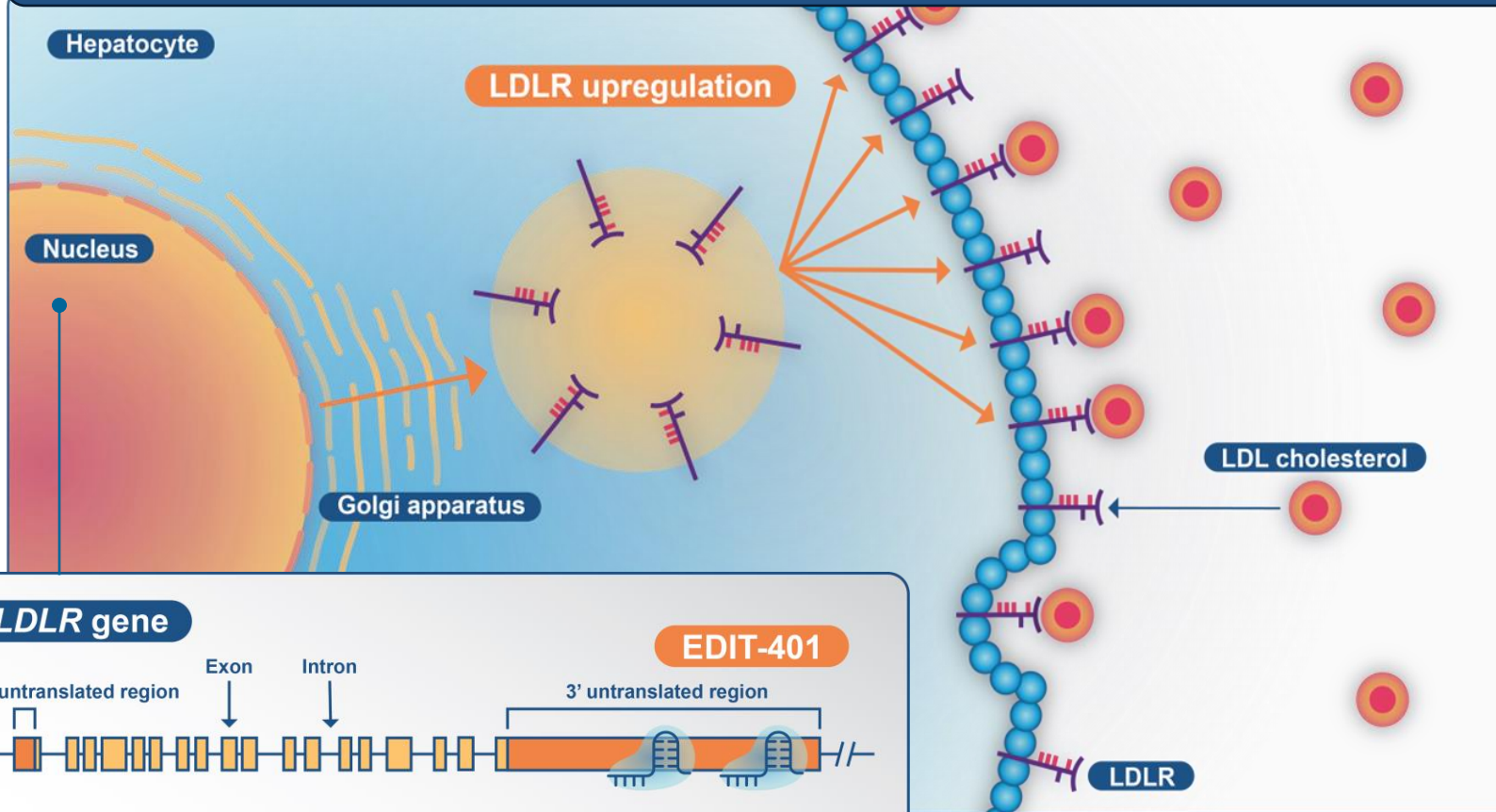


EDIT-401 is poised to transform the treatment landscape and align clinical economic value

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

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

EDIT-401 Therapeutic Strategy for LDLR upregulation



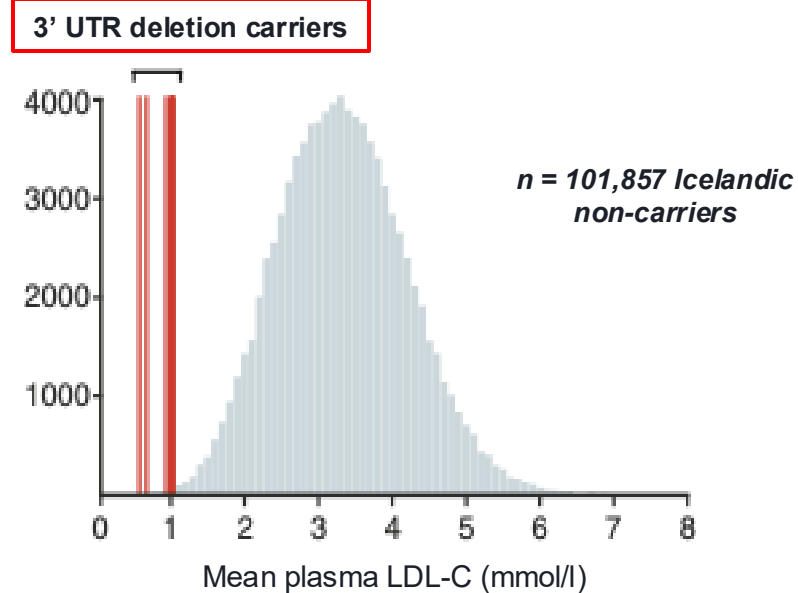
- Edits non-coding, regulatory regions
- ~10-40% functional editing achieves >90% mean LDL-C reduction in preclinical studies¹
- At least 6-fold mean LDLR protein increase in preclinical studies¹
- Differentiated upregulation strategy allows for potential best-in-class LDL-C reduction

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



7 Icelandic family members identified as carriers of partial *LDLR* 3' UTR deletion¹

Plasma LDL-C levels¹



No Adverse Events Observed in Icelandic Family Members

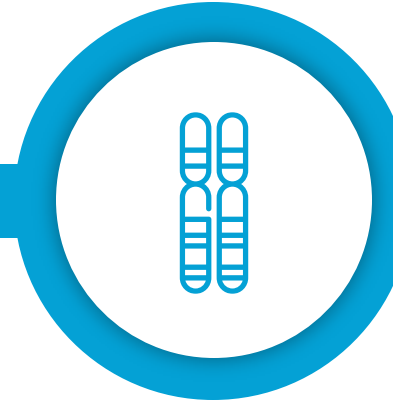
EDIT-401: Achieved ~90% or Greater Mean Reduction in LDL-C and Lp(a) in NHPs



A single dose of EDIT-401 **achieved ~90% or greater mean reduction in LDL-C and Lp(a)** across all dosed NHPs¹



LDL-C reductions remained durable through ~6 months across evaluated dose range in NHPs²



Reductions in LDL-C and Lp(a) were highly correlated, highlighting potentially transformative impact of LDLR upregulation¹



Promising preclinical safety profile with no adverse clinical observations at therapeutically relevant dose²

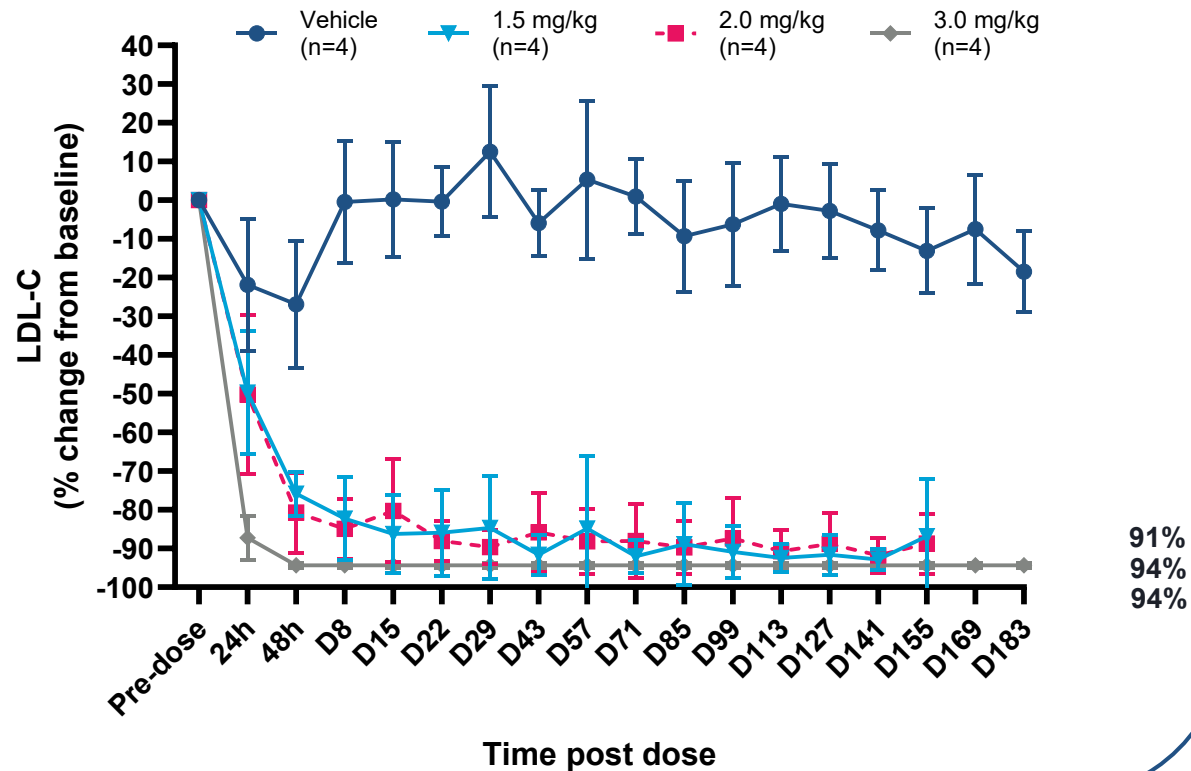
Unifying mechanism of LDLR upregulation facilitates robust reductions of multiple disease-causing lipoproteins

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



EDIT-401: Durable, Rapid, and Robust LDL-C Reduction in NHPs

Single dose of EDIT-401¹

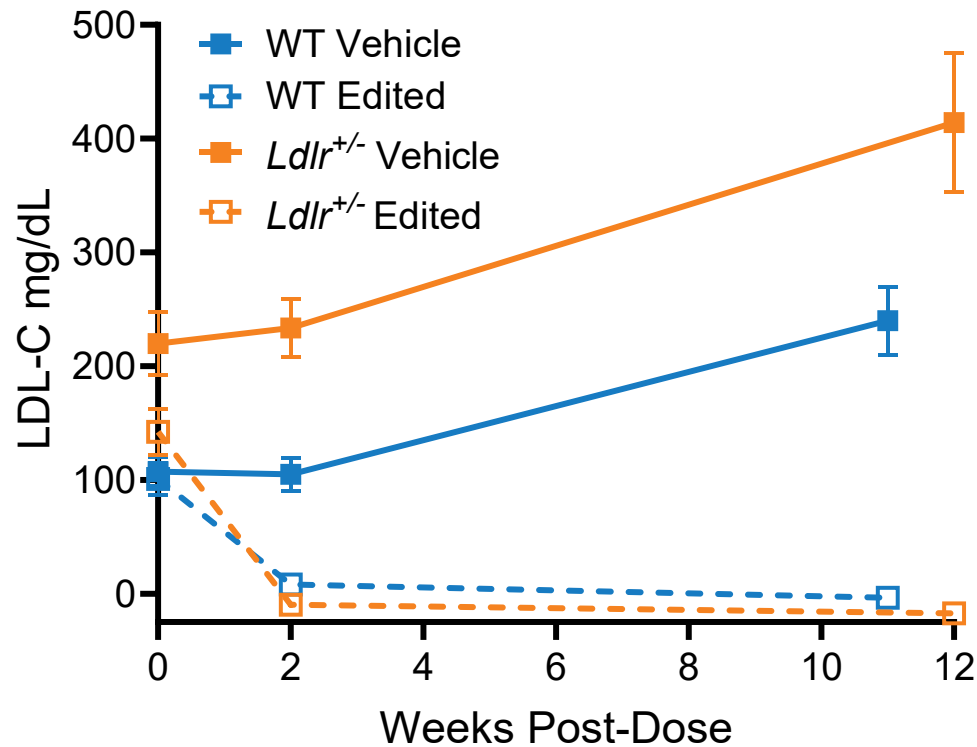


- Rapid and robust LDL-C reduction across a dose range
- At least 6-fold mean increase in LDLR protein upregulation
- Durability of LDL-C reduction at all doses through ~6 months

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-C¹



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

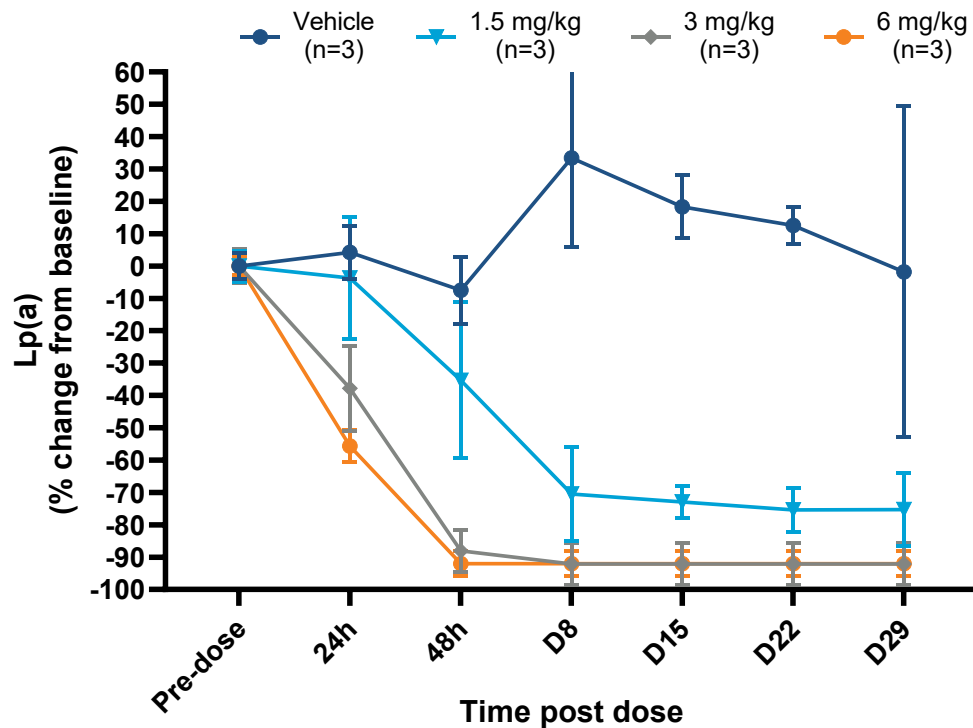
Mice on a high-fat diet had ≥ 3 -fold elevated baseline LDL-C compared with mice on a regular-fat diet. N=5 for all WT and *Ldlr*^{+/-} groups. *Ldlr*^{+/-} Edited, 100% mean LDL-C reduction from baseline at 12 weeks; WT Edited, 99% mean LDL-C reduction from baseline at 11 weeks.



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-401¹



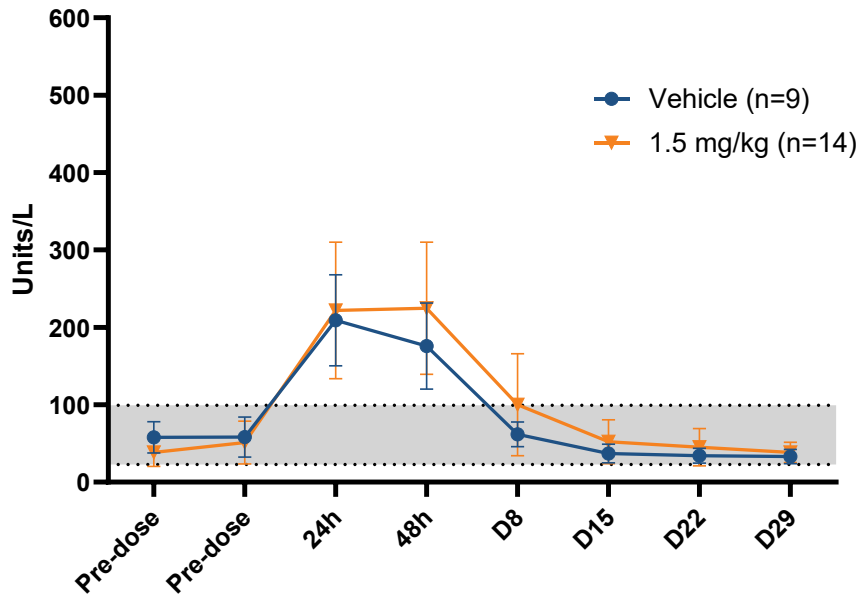
- Dose-dependent Lp(a) reduction
- Rapid Lp(a) reduction observed
- Reduction in Lp(a) correlates with LDL-C and ApoB reductions
- NHP baseline Lp(a) values approximate elevated CVD risk in humans²



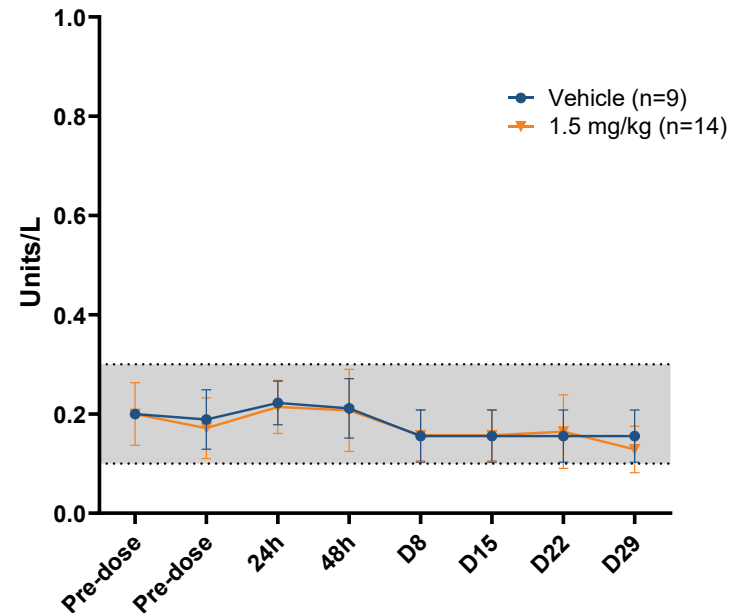
EDIT-401: Preclinical Safety Profile

Single dose of EDIT-401

Group Mean ALT



Group Mean Bilirubin



Shaded area represent normal range observed in NHPs
Vehicle dosed with saline following same pre-treatment regimen as EDIT-401

EDIT-401 safety observations at therapeutically relevant dose¹:

- Well-tolerated with no adverse clinical observations
- Observed transient ALT elevations (similar to vehicle control) resolved in ~1 week
- No liver histopathology findings

Driving Durable, Transformative Medicines Forward



EDIT-401: Key Anticipated Milestones

2026

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

2027

- Complete enrollment in clinical trial, dose-finding portion
- Topline data results available in 2027

Well capitalized to execute our strategy to advance EDIT-401

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

Programs Positioned for Development

PROGRAM (OR DISEASE CANDIDATE)	PRECLINICAL	IND/CTA ENABLING	EARLY-STAGE CLINICAL	LATE-STAGE CLINICAL	DEVELOPMENT & COMMERCIAL PARTNER
CARDIOVASCULAR					
EDIT-401: Hyperlipidemia					
HEMOGLOBIN-OPATHIES					
<i>In Vivo</i> HSC Editing – sickle cell disease					
<i>In vivo</i> HSC Editing – beta thalassemia					
OTHER ORGANS AND TISSUES					
Other Tissue Upregulation Target					
AUTOIMMUNE DISEASE					
$\alpha\beta$ T Cells - CD19 HD Allo CAR T					Bristol Myers Squibb™
CELL THERAPY					
$\alpha\beta$ T Cells (14 programs)					Bristol Myers Squibb™
$\gamma\delta$ T Cells					