UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of The Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): May 25, 2026

Editas Medicine, Inc.
(Exact Name of Registrant as Specified in its Charter)

Delaware	**001-37687**	**46-4097528**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(IRS Employer Identification No.)

11 Hurley Street **Cambridge, Massachusetts**	**02141**
(Address of Principal Executive Offices)	(Zip Code)

Registrant's telephone number, including area code: **(617) 401-9000**

(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (*see* General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, $0.0001 par value per share	EDIT	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

On May 26, 2026, Editas Medicine, Inc. (the "Company") issued a press release titled "Editas Medicine Presents EDIT-401 Preclinical Data Demonstrating Robust Reductions in LDL-C, Lp(a), and ApoB in Non-Human Primates at the 94th European Atherosclerosis Society Congress", a copy of which is furnished as Exhibit 99.1 hereto.

The information in this Item 7.01, including Exhibit 99.1 attached hereto, is intended to be furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 8.01 Other Events.

On May 25, 2026, the Company announced in an oral presentation at the 94th European Atherosclerosis Society (EAS) Congress new preclinical data for EDIT-401, its lead *in vivo* development candidate, from preclinical studies in non-human primates ("NHPs"). In the data presented, EDIT-401 achieved robust reductions in LDL-cholesterol ("LDL-C"), lipoprotein(a) ("Lp(a)"), and apolipoprotein B ("ApoB"). The findings presented included:

- A single dose of EDIT-401 achieved a 90% or greater mean reduction in LDL-C, with rapid and dose-dependent effect, across a dose range and with durability through six months.
- A single dose of EDIT-401 achieved rapid, dose-dependent mean reduction of approximately 90% in Lp(a), an independent risk factor for atherosclerotic cardiovascular disease ("ASCVD").
- A single dose of EDIT-401 achieved rapid, dose-dependent mean reduction of approximately 90% in ApoB, a key measure of total plaque-causing cholesterol particles and predictive measure for ASCVD.
- The reductions in LDL-C, Lp(a), and ApoB were highly correlated, supporting a unified mechanism facilitated by upregulation of the LDL receptor.

The Company continues to advance preclinical studies for EDIT-401, including an ongoing Good Laboratory Practice ("GLP") toxicology study in NHPs. Interim results from this study were consistent with the Company's other preclinical studies, each of which demonstrated EDIT-401 was well-tolerated at the therapeutically relevant dose of 1.5 mg/kg with no adverse clinical observations, no notable treatment-related liver enzyme elevations, and no liver histopathology findings in non-GLP toxicology. EDIT-401 showed low functional liver editing rates with a mean of 12.4% at the therapeutically relevant dose of 1.5 mg/kg. Low editing was detected in the adrenal gland, spleen and ovary, and no significant editing was observed in any other of 31 total extrahepatic tissues compared to the vehicle control. Doses of 3 mg/kg and 6 mg/kg, which are greater than the therapeutically relevant dose, were also evaluated. There were minimal to marked liver enzyme increases and non-adverse liver findings in NHPs administered 3 mg/kg and adverse clinical observations were observed in one NHP at the highest dose of 6 mg/kg.

The Company plans to submit a Clinical Trial Notification ("CTN") in Australia to the Therapeutic Goods Administration (TGA) in mid-2026, with the goal of initiating a first-in-human clinical trial of EDIT-401 in patients with Heterozygous Familial Hypercholesterolemia ("HeFH") later in 2026 and expects to have early in vivo human proof of concept data for EDIT-401 by the end of 2026. The Company anticipates that the clinical trial will initially be designed as a two-part Phase 1/2 trial. Part 1 is expected to be a single ascending dose, dose-finding study and is expected to enroll approximately 18 patients with HeFH in three dosing arms. Eligible patients will have a clinical diagnosis of HeFH with an elevated LDL-C despite treatment with two or more lipid lowering therapies. The Company plans to complete enrollment of Part 1 and have topline data results available in 2027. The Company expects that Part 2 of the trial will be a single-dose randomized, placebo-controlled expansion study with approximately 28 patients, and that if the data from the Phase 1/2 trial warrant advancing EDIT-401, the Company would advance EDIT-401 into a single, pivotal randomized, placebo-controlled Phase 3 clinical trial in patients with HeFH, with or without existing ASCVD or coronary artery disease. These clinical trial plans are subject to further discussion and alignment with regulatory authorities.

The Company also received pre-IND feedback from the U.S. Food and Drug Administration ("FDA") on its nonclinical package, CMC plans, and study design to support an Investigational New Drug Application ("IND") for EDIT-401, which the Company believes provides optionality for submitting an IND supportive of the Company's anticipated clinical development strategy.

Forward-Looking Statements

This Current Report on Form 8-K 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 Current Report on Form 8-K include statements regarding the initiation, timing, progress and results of the Company's preclinical studies and its research and development programs, including initiating a first-in-human study for EDIT-401 in 2026 and achievement of early *in vivo* human proof-of-concept data for EDIT-401 by the end of 2026; the potential of, and expectations for, EDIT-401; and the timing or likelihood of regulatory filings and approvals, including submitting a CTN in Australia by mid-2026 for EDIT-401. 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; 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; that the results and outcome of preclinical studies may not be predictive of the results of clinical trials; and the availability of funding sufficient for 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 Current Report on Form 8-K 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.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	**Description**
99.1	Press release issued by the Company on May 26, 2026*
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

*This exhibit shall be deemed to be furnished and not filed.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, as amended, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

EDITAS MEDICINE, INC.

Date: May 26, 2026

By: /s/ Amy Parison
Amy Parison
Chief Financial Officer

Exhibit 99.1

Editas Medicine Presents EDIT-401 Preclinical Data Demonstrating Robust Reductions in LDL-C, Lp(a), and ApoB in Non-Human Primates at the 94th European Atherosclerosis Society Congress

Single dose of EDIT-401 achieved ~90% or greater mean reductions in LDL-C, Lp(a), and ApoB in non-human primates

Data reinforce differentiated LDLR upregulation approach with rapid, dose-dependent effects on multiple atherogenic lipoproteins

Company on track to submit CTN by mid-2026 for EDIT-401 and achieve early in vivo *human proof-of-concept data by the end of 2026*

CAMBRIDGE, Mass., May 26, 2026 – Editas Medicine, Inc. (Nasdaq: EDIT), a pioneering gene editing company focused on developing transformative medicines for serious diseases, presented new preclinical data for EDIT-401, its lead *in vivo* development candidate, in an oral presentation at the 94th European Atherosclerosis Society (EAS) Congress in Athens, Greece on May 25, 2026. In the data presented, EDIT-401 achieved robust reductions in LDL-cholesterol (LDL-C), lipoprotein(a) (Lp(a)), and apolipoprotein B (ApoB) in non-human primates (NHPs), supporting its potential as a best-in-class medicine for hyperlipidemia.

Key EDIT-401 preclinical data in NHPs presented include:

- A single dose of EDIT-401 achieved ≥90% mean reduction in LDL-C, with rapid and dose-dependent effect.
- EDIT-401 achieved rapid, dose dependent ~90% mean reduction in Lp(a), an independent risk factor for atherosclerotic cardiovascular disease (ASCVD).
- EDIT-401 achieved rapid, dose-dependent ~90% mean reduction in ApoB, a key measure of total plaque-causing cholesterol particles and predictive measure for ASCVD.
- Reductions in LDL-C, Lp(a), and ApoB were highly correlated, supporting a unified mechanism facilitated by LDLR upregulation.

“The consistent reductions of ~≥90 percent with EDIT-401 in LDL-C, Lp(a), and ApoB observed in these preclinical studies highlight the transformative potential of our LDLR upregulation approach to address multiple drivers of cardiovascular risk, including residual risk beyond LDL-C alone,” said Linda C. Burkly, Ph.D., Executive Vice President and Chief Scientific Officer, Editas Medicine. “These robust and consistent reductions across multiple atherogenic lipoproteins with a single dose further support EDIT-401 as a potentially best-in-class *in vivo* gene editing medicine for people living with hyperlipidemia.”

The abstract can be accessed on the EAS website, and the presentation is available on the Editas Medicine website.

Editas continues to advance preclinical studies for EDIT-401, including an ongoing Good Laboratory Practice (GLP) toxicology study in NHPs. Interim results from this study demonstrated EDIT-401 was well-tolerated with no adverse clinical observations, no notable treatment-related liver enzyme elevations, and no liver histopathology findings in non-GLP toxicology at the therapeutically relevant dose of 1.5 mg/kg.

The Company also received positive pre-IND feedback from the U.S. Food and Drug Administration (FDA) on its nonclinical package, CMC plans, and study design to support an Investigational New Drug Application (IND). The Company plans to submit a Clinical Trial Notification (CTN) in Australia to the Therapeutic Goods Administration (TGA) by mid-2026 to initiate a first-in-human clinical trial of EDIT-401 in patients with Heterozygous Familial Hypercholesterolemia (HeFH) later this year, and is on track to have early *in vivo* human proof-of-concept data for EDIT-401 by the end of 2026.

About Editas Medicine
As a pioneering gene editing company, Editas Medicine is focused on translating the power and potential of CRISPR genome editing systems into a robust pipeline of transformative *in vivo* medicines for people living with serious diseases around the world. Editas Medicine aims to discover, develop, manufacture, and commercialize durable, precision *in vivo* gene editing medicines for a broad class of diseases. Editas Medicine is the exclusive licensee of Broad Institute's Cas12a patent estate and Broad Institute and Harvard University's Cas9 patent estates for human medicines. For the latest information and scientific presentations, please visit www.editasmedicine.com.

Forward-Looking Statements

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