Editas Medicine to Host Virtual Event to Discuss EDIT-301 Clinical Data from the RUBY Trial for Severe Sickle Cell Disease and the EDITHAL Trial for Transfusion-dependent Beta Thalassemia

June 6, 2023

CAMBRIDGE, Mass., June 06, 2023 (GLOBE NEWSWIRE) -- Editas Medicine, Inc. (Nasdaq: EDIT), a clinical-stage genome editing company, today announced that it will host a live webinar on Monday, June 12, at 8:00 a.m. ET to present clinical data from the RUBY trial of EDIT-301 for the treatment of severe sickle cell disease. The Company will also present initial clinical data from the EDITHAL trial of EDIT-301 for the treatment of transfusion-dependent beta thalassemia.

The webinar follows the oral presentation of the RUBY clinical trial update at the European Hematology Association (EHA) Hybrid Congress in Frankfurt, Germany, and via live stream. The EHA oral presentation is scheduled for Saturday, June 10, from 5:30-5:45 p.m. CEST/11:30-11:45 a.m. EDT.

Key data from four patients treated in the RUBY trial and one patient treated in the EDITHAL trial will be shared in the Company's webinar, including:

- Initial safety and efficacy data from four patients treated in the RUBY trial with 2-10 months follow-up, covering total hemoglobin, fetal hemoglobin, and vaso-occlusive events (VOEs) post-EDIT-301 infusion.
- Initial safety data from one patient treated in the EDITHAL trial with 1.5 months follow-up, including neutrophil and platelet engraftment, and initial efficacy data covering total hemoglobin and fetal hemoglobin post-EDIT-301 infusion.

The live and archived webcast of the Company's webinar presentation will be accessible through this webcast link, or through the Events & Presentations page of the "Investors" section of the Company's website.

A replay of the webinar will be available upon conclusion of the webinar in the Investors section of the Editas Medicine website at https://www.editasmedicine.com/.

About Sickle Cell Disease
Sickle cell disease is an inherited blood disorder caused by a mutation in the beta-globin gene that leads to polymerization of the sickle hemoglobin protein (HbS). In sickle cell disease, the red blood cells are misshapen in a sickle shape instead of a typical disc shape. The abnormal shape causes the red blood cells (RBCs) to have shortened lifespan and to block blood flow causing anemia, pain crises, organ failure, and early death. There are an estimated 100,000 people in the United States currently living with sickle cell disease. Higher levels of fetal hemoglobin (HbF) inhibit HbS polymerization, thus reducing the clinical manifestation of RBCs sickling.

About Beta Thalassemia
Beta thalassemia is a common autosomal recessive disorder with an estimated annual incidence rate of 1 in 100,000 worldwide for symptomatic individuals. Beta thalassemia mutations reduce or abrogate beta globin expression. Insufficient beta globin production leads to ineffective red blood cell production, chronic hemolytic anemia due to the destruction of red blood cells, and compensatory extramedullary hematopoiesis (creation of blood cells). Based on clinical severity and transfusion requirements, beta thalassemia can be classified into non-transfusion-dependent (NTDT) and transfusion-dependent beta thalassemia (TDT). TDT is the most severe form of beta thalassemia, and patients require lifelong regular red blood cell transfusions to prevent organ failure and death. Chronic red blood cell transfusions are complicated by iron overload leading to organ dysfunction and failure. Left untreated, the mortality rate among TDT patients is high, with a survival rate of only 15 percent at age five due to severe anemia. It is estimated that there are approximately 1,000 people in the United States currently living with transfusion-dependent beta thalassemia. Higher levels of fetal hemoglobin (HbF) ameliorate anemia thereby reducing the need for regular red blood cell transfusions.

About EDIT-301
EDIT-301 is an experimental cell therapy medicine under investigation for the treatment of severe sickle cell disease (SCD) and transfusion-dependent beta thalassemia (TDT). EDIT-301 consists of patient-derived $CD34^+$ hematopoietic stem and progenitor cells edited at the gamma globin gene (HBG1 and HBG2) promoters, where naturally occurring fetal hemoglobin (HbF) inducing mutations reside, by a highly specific and efficient proprietary engineered AsCas12a nuclease. Red blood cells derived from EDIT-301 $CD34^+$ cells demonstrate a sustained increase in fetal hemoglobin production, which has the potential to provide a one-time, durable treatment benefit for people living with severe SCD and TDT.

About RUBY
The RUBY trial is a single-arm, open-label, multi-center Phase 1/2 study designed to assess the safety and efficacy of EDIT-301 in patients with severe sickle cell disease. Patients will receive a single administration of EDIT-301. Additional details are available on www.clinicaltrials.gov (NCT# 04853576).

About EDITHAL
The EDITHAL trial is a single-arm, open label, multi-center Phase 1/2 study designed to assess the safety and efficacy of EDIT-301 in patients with transfusion-dependent beta thalassemia. Patients will receive a single administration of EDIT-301. Additional details are available on www.clinicaltrials.gov (NCT# 05444894).

About Editas Medicine
As a clinical-stage genome editing company, Editas Medicine is focused on translating the power and potential of the CRISPR/Cas12a and Cas9 genome editing systems into a robust pipeline of treatments for people living with serious diseases around the world. Editas Medicine aims to discover, develop, manufacture, and commercialize transformative, durable, precision genomic 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.

Media and Investor Contact:
Cristi Barnett
(617) 401-0113
cristi.barnett@editasmed.com
ir@editasmed.com

Source: Editas Medicine, Inc.