Fulcrum Therapeutics to Present 12-Week Data from the 20 mg Dose Cohort of the Phase 1b PIONEER Trial of Pociredir in Sickle Cell Disease

February 17, 2026 at 8:00 AM EST

CAMBRIDGE, Mass., Feb. 17, 2026 (GLOBE NEWSWIRE) -- Fulcrum Therapeutics, Inc.® (Fulcrum) (Nasdaq: FULC), a clinical-stage biopharmaceutical company focused on developing small molecules to improve the lives of patients with genetically defined rare diseases, will host a conference call and webcast on Tuesday, February 24, 2026 beginning at 8:00 a.m. ET to present 12-week results from the 20 mg dose cohort of the Phase 1b PIONEER trial of pociredir in sickle cell disease.

Members of Fulcrum management will be joined by Dr. Martin Steinberg, Professor of Medicine, Pediatrics, Pathology and Laboratory Medicine at Boston University Chobanian and Avedisian School of Medicine.

To register for this event, please click here or visit the “Events and Presentations” section of Fulcrum’s website. A replay will be available on Fulcrum’s website following the event.

About Martin H Steinberg, MD
Dr. Martin H Steinberg, MD is a hematologist with a clinical and research focus on disorders of the red blood cell with special emphasis on sickle cell disease. He has published more than 450 articles and 3 textbooks on the science and clinical features of sickle cell disease and related disorders. A graduate of Cornell University and Tufts University School of Medicine he completed post-graduate training in New York and Boston. He has participated in basic, translational, and clinical studies devoted to understanding the pathophysiology and genetic basis of phenotypic heterogeneity in sickle cell disease. Using candidate gene, genome-wide association studies, next-generation sequencing, and induced pluripotent stem cells to understand the genetic determinants of sickle cell disease heterogeneity, Dr. Steinberg and his coworkers modeled disease severity and selected subphenotypes of disease to discover hitherto unsuspected genetic associations. He has also helped develop a widely accepted paradigm reimagining the pathophysiology of sickle cell disease as a combination of both sickle vasoocclusion and intravascular hemolysis. His most recent work focusses on the distribution of HbF concentrations among red cells of patients before and following HbF induction therapeutics.

About Fulcrum Therapeutics
Fulcrum Therapeutics is a clinical-stage biopharmaceutical company focused on developing small molecules to improve the lives of patients with genetically defined rare diseases in areas of high unmet medical need. Fulcrum’s lead clinical program is pociredir, a small molecule designed to increase expression of fetal hemoglobin for the treatment of sickle cell disease. Fulcrum uses proprietary technology to identify drug targets that can modulate gene expression to treat the known root cause of gene mis-expression. For more information, visit www.fulcrumtx.com and follow us on X (@FulcrumTx) and LinkedIn.

About Pociredir
Pociredir is an investigational oral small-molecule inhibitor of Embryonic Ectoderm Development (EED) that was discovered using Fulcrum’s proprietary discovery technology. Inhibition of EED leads to potent downregulation of key fetal globin repressors, including BCL11A, thereby causing an increase in fetal hemoglobin (HbF). Pociredir is being developed for the treatment of SCD. In the PIONEER Phase 1b clinical trial in people with SCD, pociredir has demonstrated dose-dependent increases in HbF, pan-cellular HbF induction, and improvements in markers of hemolysis and anemia. Across the 12 mg and 20 mg dose cohorts, pociredir has been generally well-tolerated with up to three months of exposure, with no treatment-related serious adverse events reported. Pociredir has been granted Fast Track and Orphan Drug Designation from the FDA for the treatment of SCD. To learn more about clinical trials of pociredir please visit ClinicalTrials.gov.

About Sickle Cell Disease
SCD is a genetic disorder of the red blood cells caused by a mutation in the HBB gene. This gene encodes a protein that is a key component of hemoglobin, a protein complex whose function is to transport oxygen in the body. The result of the mutation is less efficient oxygen transport and the formation of red blood cells that have a sickle shape. These sickle shaped cells are much less flexible than healthy cells and can block blood vessels or rupture cells. People with SCD typically suffer from serious clinical consequences, which may include anemia, pain, infections, stroke, heart disease, pulmonary hypertension, kidney failure, liver disease, and reduced life expectancy.

Contact:
Kevin Gardner
LifeSci Advisors, LLC
kgardner@lifesciadvisors.com
617-283-2856