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): December 7, 2020

Keros Therapeutics, Inc.
(Exact name of registrant as specified in its charter)

Delaware	**001-39264**	**81-1173868**
(state or other jurisdiction of incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

99 Hayden Avenue, Suite 120, Building E **Lexington, Massachusetts**	**02421**
(Address of principal executive offices)	**(Zip Code)**

Registrant's telephone number, including area code: (617) 314-6297

Not applicable

(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:

☐ 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	Name of each exchange on which registered
Common Stock, $0.0001 par value per share	**KROS**	**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 8.01 Other Events.

On December 7, 2020, Keros Therapeutics, Inc. (the "Company") issued a press release announcing data from its Phase 1 clinical trial evaluating the safety, tolerability, pharmacokinetics and pharmacodynamic effects of ascending dose levels of KER-047 in healthy volunteers, preclinical data evaluating the role of activin receptor-like kinase-2 inhibition in regulating hepcidin and serum iron, and preclinical data on the differentiated mechanism of action of KER-050 and its activity in multiple anemia models, being presented at the 62nd American Society of Hematology Annual Meeting, held as a virtual event from December 5-8, 2020.

A copy of the press release is attached as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Press release dated December 7, 2020.

SIGNATURES

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

KEROS THERAPEUTICS, INC.

By: /s/ Jasbir Seehra

Jasbir Seehra, Ph.D.
Chief Executive Officer

Dated: December 7, 2020

Keros Therapeutics Presents Clinical Trial and Preclinical Study Results from its ALK2 Inhibitor Program, Including KER-047 Phase 1 Data, and KER-050 Preclinical Data, at the Virtual 62nd American Society of Hematology Annual Meeting and Exposition

- *Presentation highlights data from Keros' Phase 1 clinical trial of KER-047, which demonstrated robust and sustained dose-related increases in serum iron and transferrin saturation that were associated with decreases in ferritin and hepcidin.*
- *Preclinical presentation demonstrates that inhibition of activin receptor-like kinase-2 ("ALK2") signaling through both small molecule and biologic approaches has potentially positive effects on hepcidin-mediated iron deficiency.*
- *Additional preclinical presentations demonstrate that KER-050 potentially works at multiple stages of erythroid differentiation and resolved anemia in multiple preclinical models.*

Lexington, Massachusetts – December 7, 2020 – Keros Therapeutics, Inc. ("Keros") (Nasdaq: KROS), a clinical-stage biopharmaceutical company focused on the discovery, development and commercialization of novel treatments for patients suffering from hematological and musculoskeletal disorders with high unmet medical need, today announced that it presented data from its Phase 1 clinical trial evaluating the safety, tolerability, pharmacokinetics and pharmacodynamic effects of ascending dose levels of KER-047 in healthy volunteers and preclinical data evaluating the role of ALK2 inhibition in regulating hepcidin and serum iron at the virtual 62nd American Society of Hematology Annual Meeting and Exposition ("ASH") held December 5 through 8, 2020. In addition, Keros announced preclinical data on the differentiated mechanism of action of KER-050 and its activity in multiple anemia models.

"We were pleased to present complete data from all cohorts of our Phase 1 first-in-human clinical trial of KER-047, following our initial data announcement on August 4, 2020. In addition to characterizing the tolerability profile of KER-047 in healthy volunteers across increasing doses and after multiple administrations, we observed rapid, robust and sustained dose-related increases in serum iron and transferrin saturation that were associated with decreases in ferritin and hepcidin. Reticulocyte hemoglobin was also observed to increase, which we believe suggests that the iron mobilized with treatment was incorporated into hemoglobin. Based on these findings, we believe that the differentiated pharmacologic effect of KER-047 on hepcidin and iron mobilization has the potential to treat diseases arising from iron imbalance," said Jasbir S. Seehra, Ph.D., President and Chief Executive Officer of Keros. "In addition, the data from our preclinical studies of KER-050 further elucidate its differentiated mechanism of action and ability to potentially target multiple stages along the spectrum of erythropoiesis and resolve anemia due to multiple causes. These data further support our belief that KER-050 can potentially treat diseases that cause cytopenias due to ineffective hematopoiesis, including myelodysplastic syndromes ("MDS") and myelofibrosis."

Clinical Data Highlight the Effects of KER-047 on Iron Mobilization

- *Administration of KER-047, a Novel ALK2 Inhibitor, Elicited Robust and Sustained Increases in Serum Iron in Healthy Participants - Publication Number*: 769.

In Part 1 of the Phase 1 first-in-human clinical trial, two formulations of KER-047 were evaluated in single ascending oral doses, ranging from 1 mg to 300 mg of a capsule formulation, and 30 mg to 450 mg of a liquid formulation or placebo. In Part 2 of this trial, the liquid formulation was evaluated in multiple ascending doses (50 mg, 100 mg, 200 mg and 350 mg) of KER-047 or placebo, administered daily for 7 to 14 days.

In healthy participants, administration of KER-047 elicited rapid, robust and sustained dose-related increases in serum iron and transferrin saturation that were associated with decreases in ferritin, an effect which is consistent with mobilization of iron stores [Figures 1, 2 and 3].

Keros also observed decreases in serum hepcidin in Cohorts 1 through 3 of Part 2 of this trial (50 mg, 100 mg and 200 mg dose groups) [Figure 4]. These data were not collected in Part 1 of this trial or in Cohort 4 of Part 2 of this trial (350 mg dose group).

Additionally, Keros observed a decrease in hepcidin as early as four hours after administration of the first dose in Cohort 5 of Part 2 of this trial (100 mg dose group).

Keros also observed an increase in reticulocyte hemoglobin in Cohorts 1 through 4 of Part 2 of this trial, starting on Day 4 of treatment [Figure 5].

We believe the observed decrease in ferritin and hepcidin coupled with the observed increases in reticulocyte hemoglobin content are indicative of increased iron mobilization, resulting in increased iron incorporation into hemoglobin.

The tolerability profile of KER-047 in healthy participants was characterized in this Phase 1 trial. There were no serious adverse events ("SAEs") in either part of the trial, and the majority of adverse events ("AEs") observed were mild or moderate in severity.

In Part 2 of this trial, 10 of 40 (25%) participants administered KER-047 and 1 of 11 (9.1%) participants administered placebo discontinued study drug due to AEs. AEs that led to study drug discontinuation in three or more participants in the KER-047 groups included lymphopenia and chills. Details on AEs were as follows:

- Severe AEs were reported in 1 of 8 (12.5%) participants in the 350 mg and 100 mg (Cohort 5) dose groups.
- AEs reported in two or more participants and more common in the KER-047 groups than placebo were: abdominal discomfort, abdominal pain (upper), chills, decreased appetite, diarrhea, dizziness, fatigue, gastroenteritis, headache, lymphopenia, myalgia, nausea, neutropenia, pyrexia, rhinorrhea, tonsilitis, vomiting.
- Lymphopenia, which Keros believes is consistent with KER-047's mechanism of action, was observed after multiple doses that was reversible after discontinuation of study drug.

MAD: Average Change from Baseline in Serum Iron Through Day 7

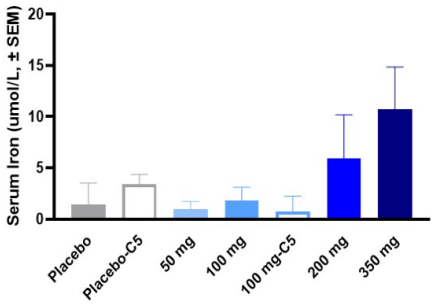

Figure 1

MAD: Average Change from Baseline in Transferrin Saturation Through Day 7

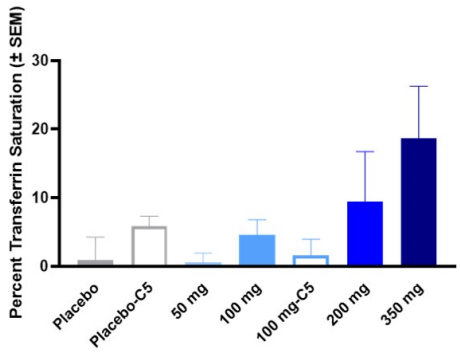

Figure 2

MAD: Average Change from Baseline in Ferritin Through Day 7

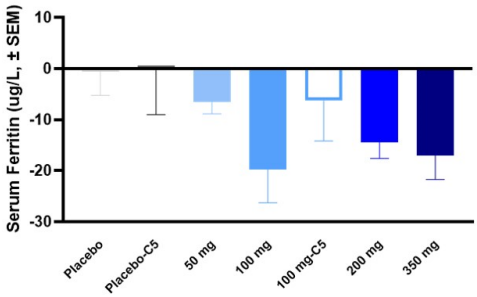

Figure 3

MAD: Hepcidin Percent Change from Baseline at Day 7, 8 hrs Post-Dose

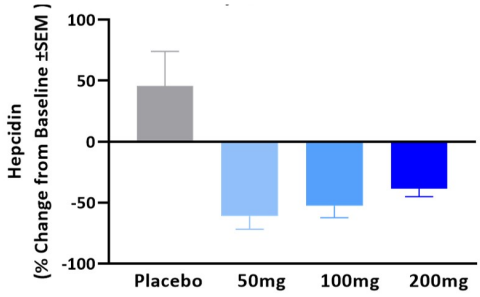

Figure 4

Mean Reticulocyte Hemoglobin at each visit

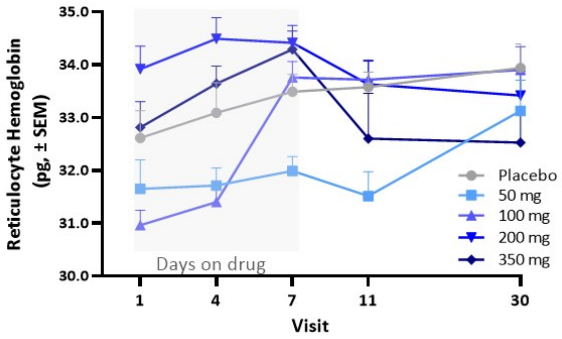

Figure 5

Multiple Modalities of ALK2 Inhibition Reduced Hepcidin and Resolved Anemia in IRIDA Model

- *Selective Inhibition of ALK2 Signaling Suppresses Serum Hepcidin and Increases Serum Iron – Publication Number*: 771

In an siRNA-based model of iron-refractory iron deficiency anemia ("IRIDA"), administration of either KTI-2338, a small molecule ALK2 kinase inhibitor, or KTI-A2.0MAb, a novel neutralizing ALK2 antibody, resulted in the rescue of hemoglobin ("HGB"), hematocrit ("HCT"), serum hepcidin and serum iron from the disease state. Following treatment, HGB, HCT and serum iron were increased and serum hepcidin was decreased in treated groups compared to control cohorts receiving vehicle.

These data further show that inhibition of ALK2 signaling via either modality in anemic mice potentially contributes to a decrease in serum hepcidin and an increase in serum iron levels, which we believe suggests that ALK2 signaling is an integral part of hepcidin-mediated iron mobilization, and illustrate the potential therapeutic benefit of ALK2 inhibition (with a small molecule inhibitor or a neutralizing monoclonal antibody) in anemia of high hepcidin, including IRIDA and anemia of inflammation.

Preclinical Studies of KER-050 Demonstrated Effects on Multiple Stages in the Erythropoiesis Cascade and in Multiple Models of Anemia

- *KER-050, a Modified ActRIIA Ligand Trap, Alleviates Cytopenia Arising from Multiple Etiologies – Presentation Number*: 2582.

Administration of a research form of KER-050 ("RKER-050") was shown to alleviate cytopenias caused by multiple conditions:

- Aged (2-year-old), vehicle-treated mice had significantly lower red blood cells ("RBCs"), HGB and HCT (-14.0%, -13.5%, -10.9%, respectively) relative to 11-week-old young vehicle-treated mice after six weeks of twice weekly treatment. However, aged mice treated with RKER-050 had higher RBCs, HGB and HCT (+12.3%, +10.0%, +9.1%, respectively) compared to aged mice receiving vehicle, with levels indistinguishable from those of young controls.
- Anemic NUP98-HOXD13 mice (a murine model of MDS) dosed twice weekly with RKER-050 for six weeks had increases in RBCs, HGB and HCT (+10.9%, +11.2%, + 9.8%, respectively), achieving values comparable to the wild-type control animal of the same age, while vehicle-treated MDS mice continued to have significantly reduced RBCs, HGB and HCT.
- In Sprague-Dawley rats, where anemia was induced by bleeding 20% of total blood volume, treatment with RKER-050 twice weekly showed early and robust increases in both RBCs and HGB

that exceeded baseline levels, whereas decreased RBCs and HGB in the vehicle-treated group persisted longer. RKER-050 treatment also resulted in an increase in platelet count at Day 3 post-phlebotomy, which remained elevated at Day 6.

- *KER-050, a Novel Inhibitor of TGF-β Superfamily Signaling, Induces Red Blood Cell Production by Promoting Multiple Stages of Erythroid Differentiation – Presentation Number*: 2736.

Mice treated with a single 10 mg/kg dose of RKER-050 had increased RBCs, HGB and HCT (+8%, +9%, +7%, respectively) 12 hours after administration compared to vehicle-treated mice. This effect was further increased on Day 7 and persisted to at least Day 14. The following was also observed:

- A reduction in the number of enucleated erythroid cells in the bone marrow and a parallel increase in the percent of immature reticulocytes ("RET") in peripheral blood, suggesting an increased outflux of RET into circulation.
- Increases in colony forming unit-erythroid progenitors, which is consistent with an effect on early stages of erythropoiesis.
- Changes in polyerythrochromatic/early orthochromatic erythroid precursors and late orthochromatic erythroblasts/RETs, which is consistent with progression of cells through erythropoiesis.
- A greater than two-fold increase in serum levels of erythropoietin ("Epo") at Days 4, 7 and 14.

Overall, we believe these data demonstrate that KER-050 stimulates terminal maturation of late-stage erythroid precursors, expands the early-stage precursor population and progresses precursors through erythropoiesis. Additionally, KER-050 was observed to increase Epo concurrent with elevated RBCs.

About KER-050

Keros' lead protein therapeutic product candidate, KER-050, is an engineered ligand trap comprised of a modified ligand-binding domain of the Transforming Growth Factor-Beta receptor known as activin receptor type IIA that is fused to the portion of the human antibody known as the Fc domain. KER-050 is being developed for the treatment of low blood cell counts, or cytopenias, including anemia and thrombocytopenia, in patients with MDS and in patients with myelofibrosis. Keros has initiated a Phase 2 clinical trial in patients with MDS evaluating KER-050 for the treatment of cytopenias, including anemia and thrombocytopenia, and expects to report initial data from Part 1 of this trial in mid-2021. Keros also plans to initiate a Phase 2 clinical trial evaluating KER-050 for the treatment of patients with myelofibrosis-associated cytopenias in 2021.

About KER-047

Keros' lead small molecule product candidate, KER-047, is designed to selectively and potently inhibit ALK2, a Transforming Growth Factor-Beta receptor. KER-047 is being developed for the treatment of anemia resulting from iron imbalance, as well as for the treatment of fibrodysplasia ossificans progressiva, a rare musculoskeletal disorder. Keros expects to commence two Phase 2 clinical trials of KER-047, one in patients with iron deficiency anemia and one in patients with IRIDA, in 2021.

About Keros Therapeutics, Inc.

Keros is a clinical-stage biopharmaceutical company focused on the discovery, development and commercialization of novel treatments for patients suffering from hematologic and musculoskeletal disorders with high unmet medical need. Keros is a leader in understanding the role of the Transforming Growth Factor-Beta family of proteins, which are master regulators of red blood cell and platelet production as well as of the growth, repair and maintenance of muscle and bone. Keros' lead protein

therapeutic product candidate, KER-050, is being developed for the treatment of low blood cell counts, or cytopenias, including anemia and thrombocytopenia, in patients with myelodysplastic syndromes and in patients with myelofibrosis. Keros' lead small molecule product candidate, KER-047, is being developed for the treatment of anemia resulting from iron imbalance, as well as for the treatment of fibrodysplasia ossificans progressiva. Keros' third product candidate, KER-012, is being developed for the treatment of disorders associated with bone loss, such as osteoporosis and osteogenesis imperfecta, and for the treatment of pulmonary arterial hypertension.

Cautionary Note Regarding Forward-Looking Statements

Statements contained in this press release regarding matters that are not historical facts are "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Words such as "anticipates," "believes," "expects," "intends," "plans," "potential," "projects," "would" and "future" or similar expressions are intended to identify forward-looking statements. Examples of these forward-looking statements include statements concerning: Keros' expectations regarding its growth, strategy, progress and timing of its clinical trials for KER-050 and KER-047; the potential of KER-050 to treat multiple cytopenias in diseases of ineffective hematopoiesis; the potential of KER-047 to treat diseases arising from iron imbalance; and the potential of ALK2 inhibition to treat IRIDA and other hepcidin-mediated anemias. Because such statements are subject to risks and uncertainties, actual results may differ materially from those expressed or implied by such forward-looking statements. These risks and uncertainties include, among others: Keros' limited operating history and historical losses; Keros' ability to raise additional funding to complete the development and any commercialization of its product candidates; Keros' dependence on the success of its lead product candidates, KER-050 and KER-047; that Keros may be delayed in initiating, enrolling or completing any clinical trials; competition from third parties that are developing products for similar uses; Keros' ability to obtain, maintain and protect its intellectual property; Keros' dependence on third parties in connection with manufacturing, clinical trials and pre-clinical studies; and risks relating to the impact on our business of the COVID-19 pandemic or similar public health crises.

These and other risks are described more fully in Keros' filings with the Securities and Exchange Commission ("SEC"), including the "Risk Factors" section of the Company's Quarterly Report on Form 10-Q, filed with the SEC on November 10, 2020, and its other documents subsequently filed with or furnished to the SEC. All forward-looking statements contained in this press release speak only as of the date on which they were made. Except to the extent required by law, Keros undertakes no obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made.

Investor Contact:

Julia Balanova

jbalanova@soleburytrout.com

646-378-2936