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): June 12, 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 June 12, 2020, Keros Therapeutics, Inc. (the "Company") issued a press release announcing updated data from its Phase 1 clinical trial evaluating the safety, tolerability, pharmacokinetics and pharmacodynamic effects of ascending dose levels of KER-050 in healthy post-menopausal women, along with preclinical data on KER-050 from multiple animal models and additional preclinical data evaluating the role of ALK2 inhibition in regulating hepcidin and serum iron, being presented at the 25th Annual Congress of European Hematology Association, held as a virtual event from June 11-21, 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 June 12, 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: June 12, 2020

Exhibit 99.1

Keros Therapeutics Presents Results from Clinical and Preclinical Studies Investigating KER-050, along with Preclinical Data from its ALK2 Inhibitor Program, at the European Hematology Association 2020 Annual Meeting

- *Poster presentation highlights Phase 1 clinical trial data demonstrating tolerability, pharmacokinetics and pharmacodynamics of KER-050 in healthy postmenopausal women.*
- *Poster presentations introduce data demonstrating KER-050 robustly promoted hematopoiesis in multiple animal species in preclinical studies and support a potentially novel mechanism of action.*
- *Oral presentation on a novel ALK2 kinase inhibitor that suppressed hepcidin production and subsequently ameliorated anemia in a mouse model of iron-refractory iron deficiency anemia.*

Lexington, Massachusetts – June 12, 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 updated data from its Phase 1 clinical trial evaluating the safety, tolerability, pharmacokinetics and pharmacodynamic effects of ascending dose levels of KER-050 in healthy post-menopausal women, along with preclinical data on KER-050 from multiple animal models and additional preclinical data evaluating the role of ALK2 inhibition in regulating hepcidin and serum iron.

These data were presented at the 25th Annual Congress of European Hematology Association (EHA), held as a virtual event from June 11-21, 2020.

"The presentations by Keros at the 25th Congress of EHA showcase the breadth and depth of the Keros franchise in hematological disorders," said Jasbir S. Seehra, Ph.D., President and Chief Executive Officer of Keros. "KER-050 was well tolerated in healthy volunteers in our Phase 1 clinical trial across increasing doses and after multiple administrations. Importantly, we also observed a robust, dose-dependent response of multiple hematological parameters. Based on these findings and the results from preclinical studies, we believe KER-050 has a differentiated pharmacologic effect on red blood cells and platelets and has the potential to treat multiple cytopenias in diseases of ineffective hematopoiesis such as myelodysplastic syndromes and myelofibrosis. In addition, our preclinical data from our ALK2 program demonstrated that ALK2 inhibition was able to reverse elevated hepcidin and anemia in a mouse model of iron-refractory iron deficiency anemia. We believe these findings support that ALK2 inhibition is a potentially promising therapeutic for improving iron levels and anemia in IRIDA patients and could potentially provide benefit in other hepcidin-mediated anemias such as chronic kidney disease or anemia of inflammation."

Clinical Data Highlight Potential of KER-050 to Address Multiple Cytopenias

- *Administration of KER-050, a Novel ActRIIA Ligand Trap, to Healthy Participants Elicited Robust and Sustained Increases in Hemoglobin and Platelets - Abstract Number*: EP806.

Administration of KER-050 in healthy subjects was observed to elicit rapid, robust, sustained and dose-dependent increases in hemoglobin, reticulocytes and red blood cells, in addition to clinically meaningful increases in platelets. In Part 1 of the Phase 1 first-in-human clinical trial, 38 subjects received either a single dose of KER-050 (0.05, 0.5, 1.5 or 4.5 mg/kg) or placebo, and in Part 2 of the trial, 10 subjects received either two doses, 28 days apart, of KER-050 (0.75 mg/kg) or placebo. KER-050 was well tolerated at dose levels up to 4.5 mg/kg as a single dose, and 0.75 mg/kg after two doses. No treatment-related serious adverse events were reported. There were no discontinuations due to adverse events, which in KER-050-treated subjects were all mild or moderate in severity; as expected with the KER-050 mechanism of action, reversible mild hypertension events were observed in subjects with approximately 3g/dL increase in hemoglobin.

A dose-dependent increase in the proportion of subjects with a hemoglobin increase of ≥1.5 g/dL was observed. The proportion of subjects treated with KER-050 that demonstrated this hemoglobin increase in Part 1 of the trial was 12.5% (0.05 mg/kg dose), 12.5% (0.5 mg/kg dose), 50% (1.5 mg/kg dose), 66.7% (4.5 mg/kg dose) and 0% (placebo group), and in Part 2 of the trial was 25% (0.75 mg/kg dose) and 0% (placebo group). Overall, there was a dose-dependent increase in hemoglobin observed in Part 1 of this trial, with the highest dose of 4.5 mg/kg eliciting a mean change from baseline in hemoglobin of 2.1 g/dL on Day 29. In addition, clinically relevant increases in platelet counts were observed, with a mean change from baseline of 39.2 x 10^9 cells/L in the 4.5 mg/kg group on Day 7.

Additionally, in the Phase 1 clinical trial of KER-050:

- The mean half-life of KER-050 observed in the 0.5, 1.5 and 4.5 mg/kg doses in Part 1 and after the first 0.75 mg/kg dose was administered in Part 2 ranged from 9.7 to 11.9 days.
- In the 4.5 mg/kg group, the mean percent change (decrease) from baseline in follicle-stimulating hormone, a biomarker of activin inhibition, on Day 11 was 18.6%.
- In Part 1, the maximum percentage change from baseline observed in bone specific alkaline phosphatase, a biomarker of bone remodeling, was 9.6% (0.05 mg/kg dose), 21.8% (0.5 mg/kg dose), 21.8% (1.5 mg/kg dose), 34.1% (4.5 mg/kg dose) and 13.3% (placebo group).

Preclinical Studies of KER-050 Demonstrated Robust Effects on Hematopoiesis and Differentiated Mechanism of Action

• *KER-050, a Novel Modified ActRIIA Ligand Trap, Increases Red Blood Cell Production in Cynomolgus Monkeys - Abstract Number*: EP782.

Treatment with KER-050 resulted in robust and dose-dependent increases in red blood cell (“RBC”) number, hemoglobin (“Hgb”), hematocrit (“Hct”) and reticulocyte (“RET”) number in male and female cynomolgus monkeys dosed subcutaneously with KER-050 at doses of 3, 10 or 50 mg/kg for three months compared to vehicle-treated cohorts:

- RBC number and Hct both showed statistically significant increases in treated animals compared to vehicle at all doses in females and at 10 mg/kg and 50 mg/kg in males.
- Hgb increases from baseline were all statistically significant in treated animals compared to vehicle at all doses in both females and males.
- RET number increase was statistically significant in both female and male treated animals compared to vehicle at the 50 mg/kg dose.

KER-050 increased RBC number, Hct, Hgb and RET number in both male and female monkeys across multiple doses, demonstrating the effect of KER-050 on erythropoiesis in higher order species.

• *KER-050, a Novel Inhibitor of TGF-b Superfamily Signaling, Induces Red Blood Cell Production and is a Potential Candidate for the Treatment of Ineffective Erythropoiesis - Abstract Number*: EP786.

In healthy mice dosed with 10 mg/kg of RKER-050 (a research form of KER-050), rapid increases in circulating reticulocytes, RBCs, hemoglobin and hematocrit were observed compared to vehicle-treated mice, supporting an effect on maturation of late-stage erythroid precursors. Additionally, bone marrow from mice harvested four days after treatment with RKER-050 had an increased proerythroblast population, with a concomitant reduction in burst-forming unit-erythroids (BFU-Es) and colony-forming unit-erythroids (CFU-Es), which is consistent with RKER-050 affecting early stages of erythropoiesis.

In a subsequent study evaluating the involvement of erythropoietin in the RKER-050 mechanism of action, groups of mice received vehicle, 10 mg/kg of RKER-050, 5.5 mg/kg of neutralizing erythropoietin (“EPO”) antibody (“mEpo Ab”), or a combination of both RKER-050 and mEPO Ab. While treatment with

mEpo Ab significantly reduced hematologic parameters, RKER-050 administered in combination with mEpo Ab resulted in RBC numbers comparable to vehicle.

Keros believes these data support that KER-050 has the potential to stimulate terminal maturation of late-stage erythroid precursors, increase the early stage precursor population and acts via a mechanism of action that is distinct from EPO.

Administration of ALK2 Inhibitor Reduced Hepcidin and Resolved Anemia in Preclinical IRIDA Model

- *Selective Inhibition of ALK2 Signaling Ameliorates Disease in a Novel Model of Iron Refractory Iron Deficiency Anemia (IRIDA) - Abstract Number*: S308

An ALK2 inhibitor ("KTI-2338") was used to test the effect of suppressing ALK2 signaling on regulation of hepcidin, serum iron and hematological parameters in a mouse model of IRIDA.

IRIDA is a disease that arises from mutations in the TMPRSS6 gene, resulting in excessive signaling of the ALK2 receptor and subsequent hepcidin-mediated anemia. Keros developed a mouse model of IRIDA using an siRNA to knock down TMPRSS6 expression. Mice receiving the TMPRSS6 siRNA replicated the anemia in IRIDA patients, including having elevated hepcidin levels and anemia. In the preclinical study, mice received TMPRSS6 siRNA and were confirmed to have high hepcidin and anemia prior to receiving treatment with the ALK2 inhibitor KTI-2338. Daily treatment of KTI-2338 in mice with established disease resulted in increases in hemoglobin, hematocrit, RBCs and serum iron with a concomitant decrease in hepcidin.

These data demonstrate ALK2 inhibition can overcome changes in hepcidin, serum iron and anemia resulting from TMPRSS6-deficiency and therefore, Keros believes ALK2 inhibition has the potential to treat patients with IRIDA.

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 myelodysplastic syndromes, or MDS, and in patients with myelofibrosis. Keros recently completed a Phase 1 clinical trial evaluating the safety, tolerability and pharmacokinetics of KER-050 in healthy post-menopausal women. Keros plans to commence a Phase 2 clinical trial in patients with MDS evaluating KER-050 for the treatment of cytopenias, including anemia and thrombocytopenia, in the second half of 2020. Keros also plans to commence a Phase 2 clinical trial evaluating KER-050 for the treatment of patients with myelofibrosis-associated cytopenias 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 elevated levels of hepcidin, the key regulator of iron absorption and recycling, 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; the potential of KER-050 to treat multiple cytopenias in diseases of ineffective hematopoiesis; 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 May 22, 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