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): March 9, 2022

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 7.01 Regulation FD Disclosure.

On March 9, 2022, Keros Therapeutics, Inc. (the "Company") updated its corporate presentation for use in meetings with investors, analysts and others. The presentation is available through the Company's website and a copy is attached as Exhibit 99.1 to this Current Report on Form 8-K.

The information in this Current Report on Form 8-K, including Exhibit 99.1 attached hereto, is being 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. The information contained herein and in the accompanying exhibit is not incorporated by reference in any filing of the Company under the Securities Act of 1933, as amended, or the Exchange Act, whether made before or after the date hereof, regardless of any general incorporation language in such filing, except as shall be expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Corporate Presentation dated March 2022.
104	Cover Page Interactive Data File (the cover page XBRL tags are embedded within the Inline XBRL document)

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: March 9, 2022

KEROS THERAPEUTICS

Corporate Presentation

March 2022

Disclaimer

Statements contained in this presentation 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 the design, objectives and timing of its preclinical studies and clinical trials for KER-050, KER-047 and KER-012; the potential impact of COVID-19 on Keros’ ongoing and planned preclinical studies, clinical trials, business and operations; and the potential of Keros’ proprietary discovery approach. 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 Annual Report on Form 10-K, filed with the SEC on March 9, 2022, and its other documents subsequently filed with or furnished to the SEC. All forward-looking statements contained in this presentation 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.

Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and the Company’s own internal estimates and research. While we believe these third-party sources to be reliable as of the date of this presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third -party sources. Finally, while we believe our own internal research is reliable, such research has not been verified by any independent source.

The trademarks included in this presentation are the property of the owners thereof and are used for reference purposes only.

Harnessing the Powerful Biology of the TGF-β Superfamily

- Clinical-stage biopharmaceutical company developing novel therapeutics that target the TGF-β superfamily
- Leveraging our extensive experience in TGF-β superfamily protein structure, function and protein engineering to generate a clinical pipeline of differentiated therapeutics:

Hematology

KER-050: Modified activin receptor IIA (ActRIIA) ligand trap
- Designed to address ineffective hematopoiesis by modulating TGF-β superfamily signaling
- Potential to correct multiple cytopenias in patients with MDS and myelofibrosis (MF)

KER-047: Activin receptor-like kinase-2 (ALK2) inhibitor
- Designed to address anemias resulting from iron imbalance
- Potential to treat iron-refractory iron deficiency anemia (IRIDA), iron deficiency anemia and other diseases

Pulmonary and Musculoskeletal

KER-012: Modified activin receptor IIB ligand trap
- Designed to inhibit vascular remodeling and bone loss
- Potential to treat pulmonary arterial hypertension (PAH) and bone loss in osteogenesis imperfecta and osteoporosis

Keros is Developing Differentiated Clinical Assets in Hematological and Musculoskeletal Disorders

Program	Asset	Phase of Development	Status	Next Milestones*
Hematology	KER-050 (therapeutic protein)	Myelodysplastic syndromes	Phase 2 clinical trial ongoing	Additional data from the Phase 2 clinical trial: mid-2022
		Myelofibrosis	Phase 2 clinical trial ongoing	Initial data: End of 2022
	KER-047 (small molecule)	Iron deficiency anemia	Completed Phase 1 clinical trial	Initiate Phase 2 clinical trial: H1 2022 Initial data: End of 2022
		Anemia from high hepcidin		Initiate Phase 2 clinical trial: H1 2022 Initial data: End of 2022
Musculoskeletal	KER-012 (therapeutic protein)	Bone disorders	Phase 1 clinical trial in healthy volunteers ongoing	Initial data: Q2 2022
Pulmonary		Pulmonary arterial hypertension		
Preclinical Pipeline		Musculoskeletal and hematology		

* Anticipated clinical milestones are subject to the impact of COVID-19 on our business.

KER-050

A novel product candidate designed to address diseases arising from ineffective hematopoiesis

- Myelodysplastic syndromes
- Myelofibrosis

KER-050: A potential treatment for ineffective hematopoiesis in Myelodysplastic Syndromes (MDS) and Myelofibrosis (MF)

MDS

- Hematologic malignancy predominantly affecting the elderly, resulting in multiple cytopenias (anemia, thrombocytopenia, neutropenia) due to ineffective hematopoiesis
  - 60-170,000 MDS patients in the US with 15,000-20,000 newly diagnosed each year[1]
- Platelet transfusion is the current treatment option for thrombocytopenia
- Anemia treatments include red blood cell (RBC) transfusions, erythropoiesis-stimulating agents (ESAs) and Reblozyl®
  - ESAs benefit is limited to patients with low transfusion burden and low endogenous EPO levels
  - Reblozyl® approved for treatment of anemia failing ESA in RS positive patients (~15% of MDS patients) requiring transfusions
    - 38% responders vs 13% placebo
    - Similar to ESAs, benefit primarily in low transfusion burden

MF

- Molecular abnormalities in JAK-STAT pathway result in expansion of RBC and platelet precursors and subsequent ineffective hematopoiesis
- 16,000-18,500 MF patients in the US[2] with >3,000 newly diagnosed each year[3] and nearly all will become transfusion dependent[4]
- Megakaryocyte accumulation/breakdown is implicated in the inducement of bone marrow fibrosis

- We believe that KER-050 has the potential to address ineffective hematopoiesis, which is central to MDS and MF

[1]MDS Foundation; [2]Gangat 2011 [3]Srour 2016; [4]Naymagon 2017

KER-050 is a Modified ActRII Fusion Protein

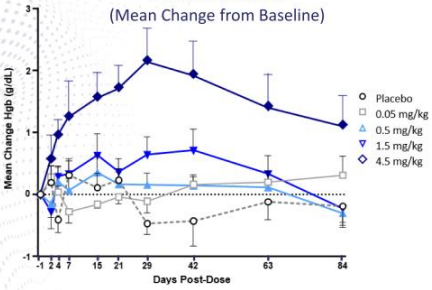
- Activin receptors are expressed on hematopoietic cells and modulate differentiation of precursor cells
- KER-050 is a ligand trap composed of a modified extracellular domain of activin receptor IIA (ActRIIA) fused to the Fc region of human IgG
- KER-050 is designed to increase RBC and platelet production by inhibiting the signaling of ligands through activin receptors

- Phase 1 clinical trial of KER-050 recapitulated preclinical data with observed rapid, sustained and dose-dependent increases in RBCs and platelets
  - Observed to be well tolerated at dose levels up to 4.5 mg/kg, the highest dose level tested

KER-050 Increased RBC Parameters and Platelets Following Single Doses in a Phase 1 Clinical Trial in Healthy Volunteers

HEMOGLOBIN

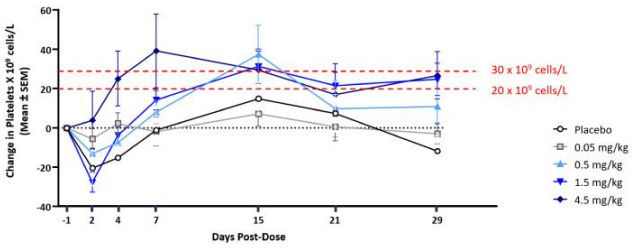
(Mean Change from Baseline)



- Observed increase in RBC parameters continued to build through day 29 which we believe is supportive of KER-050 acting on multiple stages of erythropoiesis
- Observed sustained increase in hemoglobin through day 84 after a single dose supports monthly or less frequent dosing

PLATELETS

(Mean Change from Baseline in Platelets at Each Dose)



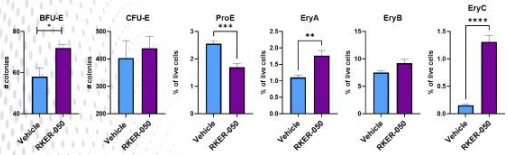
- Single doses of KER-050 observed to lead to clinically meaningful changes in platelets through day 29
- Maximum changes observed between 7-15 days post-dosing

KER-050 Preclinical Data Support Potential to Promote Multiple Stages of Erythropoiesis

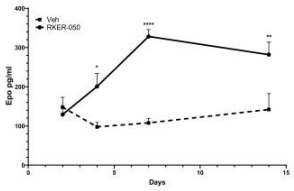
In a preclinical study of healthy mice, treatment with a mouse research form of KER-050 (RKER-050):

- Stimulated terminal maturation of late-stage erythroid precursors and increased the outflux of late-stage reticulocytes into circulation
- Expanded the early-stage precursor population that differentiate to replenish the late-stage erythroblast pools
- Increased erythropoietin levels
- Unaltered life span of RBCs

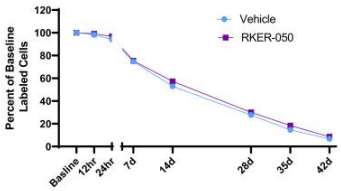
Erythropoietic tone upregulated 14 days after single RKER-050 injection



Single treatment with RKER-050 increased serum EPO



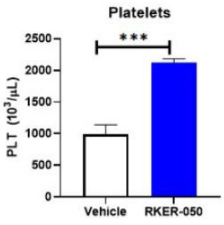
RKER-050 treatment did not alter RBC life span

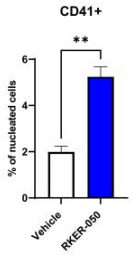


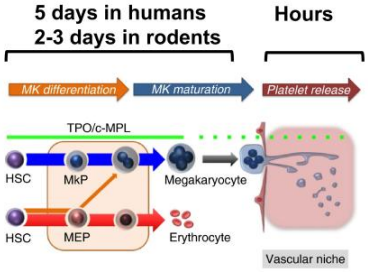
EHA 2021: Abstract # 2736

RKER-050 Preclinical Data Support Potential to Promote Multiple Stages of Thrombopoiesis

- Observed rapid onset of platelet increase in mouse models is consistent with terminal maturation of proplatelets
- Observed increase in the number of CD41+ cells and the polyploid in mouse models is consistent with upregulation in the early stages of thrombopoiesis



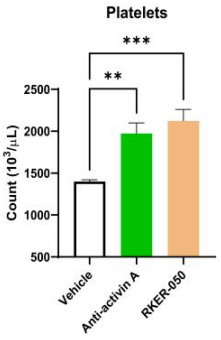




*** $p < 0.05$; **** $p < 0.0001$

RKER-050 Increased Platelets Potentially Through Activin A Inhibition

- Treatment with Activin A inhibited differentiation of platelet production in a preclinical study in mice
  - Decreased number of polyploid CD41+ cells (megakaryocytes)
- In contrast, inhibition of activin A through administration of an activin A neutralizing antibody in a preclinical study increased platelet count
- KER-050 (and RKER-050) is designed to inhibit a subset of TGF-β superfamily ligands, including activin A, activin B, GDF8 and GDF11
- In a preclinical study in mice, RKER-050 administration resulted in rapid and sustained increases in platelets, potentially through RKER-050's inhibition of activin A



** p < 0.01, *** p < 0.001.

KER-050 Summary

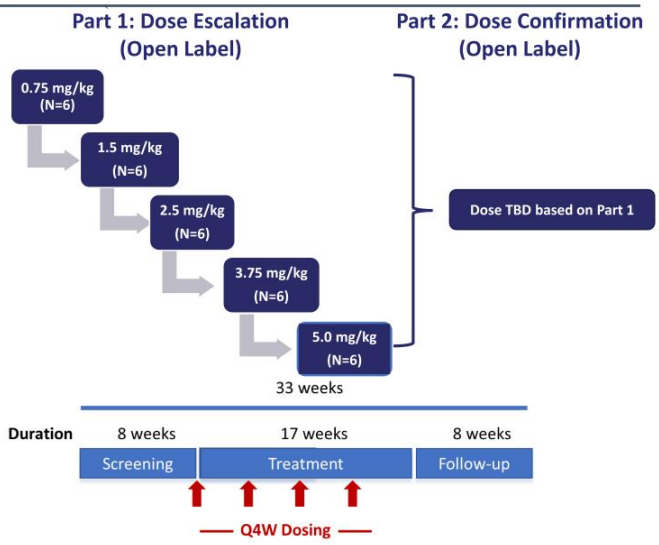
- KER-050 increased RBC parameters and platelets following single doses in a Phase 1 clinical trial in healthy volunteers
- In preclinical studies, a research form of KER-050 (RKER-050) was observed to increase RBCs and platelets, potentially through promotion of multiple stages of erythropoiesis and thrombopoiesis
- We believe that data from our preclinical studies and our Phase 1 clinical trial support that treatment with KER-050 has the potential to address ineffective hematopoiesis in diseases where multiple cytopenias arise from the blockage in progression of progenitor cells to mature blood cells, such as in MDS and myelofibrosis

KER050-MD-201

A Phase 2 Clinical Trial Of KER-050 For The Treatment Of Anemia In Patients With Very Low, Low Or Intermediate Risk Myelodysplastic Syndromes (MDS)

Phase 2 Clinical Trial of KER-050 in MDS

- Phase 2, multicenter, open-label clinical trial in very low-, low- and intermediate-risk MDS patients
- KER-050 administered once every four weeks (Q4W) for 12 weeks
- Trial objectives:
  - Evaluate safety, tolerability and pharmacokinetics
  - Evaluate pharmacodynamic effects and efficacy of KER-050
- Protocol was amended to allow patients in Part 1 and Part 2 to remain on treatment up to 24 cycles (2 years)



Phase 2 Clinical Trial of KER-050 in MDS

Key Eligibility Criteria:

- MDS with very low-, low-, or intermediate-risk disease, as classified by the International Prognostic Scoring System-Revised, including both patients that did not have ring sideroblasts (non-RS) and patients that have ring sideroblasts (RS+)
- ESA naïve and experienced patients are eligible
- No prior treatment with azacitidine, decitabine, lenalidomide, luspatercept or sotatercept
- Anemia, categorized in one of the following three groups:
  - Non-transfused (NT): hemoglobin (Hgb) <10 g/dL
  - Low transfusion burden (LTB): 1-3 units of RBC/8 weeks, Hgb <10 g/dL
  - High transfusion burden (HTB): ≥4 units of RBC/8 weeks

Select Efficacy Endpoints:

- IWG 2006 Hematological improvement-erythroid (HI-E)
  - Hemoglobin increase of ≥1.5 g/dL for 8 weeks (in NT and LTB patients)
  - Reduction of ≥4 RBC units transfused over 8 weeks compared to baseline (in HTB patients)
- Transfusion independence (TI) for at least 8 weeks in patients who require ≥ 2 RBC units transfused at baseline

Trial Status and Baseline Characteristics

- Abstract announcing additional data from our ongoing Phase 2 clinical trial of KER-050 in MDS was presented at the 63rd American Society of Hematology (ASH) Annual Meeting and Exposition
- Data cut-off date: October 25, 2021
- Efficacy data presented from Cohorts 1, 2 and 3:
  - Cohort 1: 0.75 mg/kg Q4W
  - Cohort 2: 1.5 mg/kg Q4W
  - Cohort 3: 2.5 mg/kg Q4W
- Safety data presented from Cohorts 1, 2, 3 and 4 (3.75 mg/kg)
- 24 patients in Cohorts 1, 2, 3 and 4 received at least one dose of KER-050 as of the data cut-off date
  - 16 patients in Cohorts 1, 2 and 3 completed 8 weeks of evaluation and treatment with KER-050 as of the data cut-off date (which we refer to as the “evaluable patients”), comprised of:
    - 4 NT patients; 3 LTB patients; and 9 HTB patients
    - 8 were non-RS and 8 were RS+
  - 2 patients in Cohort 2 were not efficacy-evaluable due to withdrawal of consent (n=1) and death (n=1)
  - 6 patients in Cohort 4 were not efficacy-evaluable as they had not completed 8 weeks of evaluation and treatment as of the data cut-off date

Biomarkers of Ineffective Hematopoiesis in Very Low-, Low- and Intermediate-Risk MDS Patients by RS Status

Biomarker	RS Status Mean (SD)	
	RS+ (N=13)	Non-RS (N=11)
EPO (IU/L)	326.0 (826.2)	490.2 (855.7)
Reticulocytes (10^9/L)	39.1 (28.0)	29.8 (24.8)
Platelets (10^9/L)	228.4 (67.8)	134.6 (55.5)
TPO (pg/mL)	74.2 (69.3)	42.8 (60.1)
sTfR (mg/L)	1.9 (1.0)	1.2 (0.6)

- MDS is characterized by ineffective hematopoiesis due to dysregulated differentiation of myeloid, erythroid and/or megakaryocytic lineages which results in multilineage cytopenias
- Non-RS patients had lower reticulocyte and platelet counts, higher endogenous EPO levels and lower sTFR than RS+ patients at baseline, which suggested a greater degree of ineffective hematopoiesis

Safety Data as of the Data Cut-off Date (October 25, 2021)

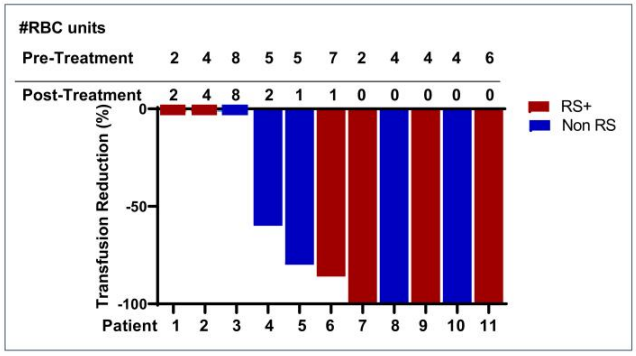
- Safety Review Committee has reviewed data from 0.75 mg/kg (Cohort 1), 1.5 mg/kg (Cohort 2), 2.5 mg/kg (Cohort 3) and Cohort 4 (3.75 mg/kg)
- Summary of safety data as of the data cut-off date (Cohorts 1-4, n=24)
  - No drug-related serious adverse events (SAEs), dose-limiting toxicities or drug-related dose modifications reported
  - 6 treatment-emergent SAEs in 5 patients, all of which were deemed unrelated to study drug:
    - Grade 2 (pyrexia, cardiac failure congestive)
    - Grade 3 (anaemia, pneumonia, pneumothorax)
    - Grade 5 (death – obesity-related heart disease)
  - Most frequent treatment-emergent adverse events (AEs):
    - Diarrhea, dyspnea, fatigue and nausea
  - Treatment-related AEs, reported in 4 patients, by maximum grade:
    - Grade 1 (headache, pain in extremity, abdominal pain)
    - Grade 2 (rash, diarrhea, nausea, peripheral edema)
      - The treatment-related AE of maculopapular rash was reported in one patient, after the patient's first dose, and resolved without recurrence following subsequent doses
- 2 withdrawals (death deemed unrelated to study drug; patient decision)
- No patients developed high-risk MDS or acute myeloid leukemia

Preliminary Results from Phase 2 Clinical Trial

Preliminary results*:

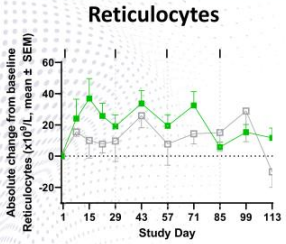
- **n=8/16 (50%)** of the evaluable patients met at least one of the following endpoints:
  - 2006 IWG HI-E criteria: **n=7/16 (43.8%)**
    - Hemoglobin increase of ≥1.5 g/dL for 8 weeks (in NT and LTB patients)
    - Reduction of ≥4 RBC units transfused over 8 weeks compared to baseline (in HTB patients)
  - Transfusion independence for at least 8 weeks in patients who require ≥ 2 RBC units transfused at baseline: **n=5/11 (45.5%)**
    - RS+ 3/6
    - Non-RS 2/5

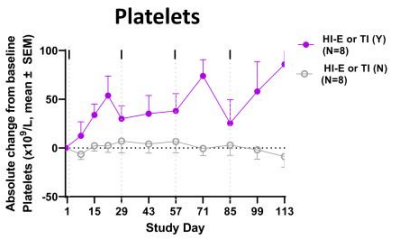
Maximum Reduction in Transfusions Over 8 Weeks*

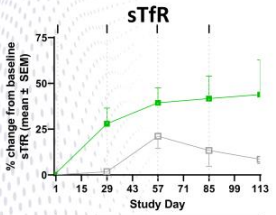


*Data cut-off date: October 25, 2021

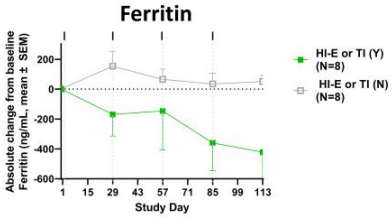
KER-050 Demonstrated Improvement in Erythropoiesis and Thrombopoiesis







Baseline sTfR mg/L:
HI-E or TI (Y): 1.97 ± 1.06
HI-E or TI (N): 1.42 ± 0.62



Baseline Ferritin 867±748 ng/mL
4/16 patients are on iron chelation
3/4 on iron chelation achieved HI-E or TI

KER-050 upregulated erythropoiesis

- Reticulocyte increases observed in patients achieving HI-E or TI endpoints
- Increases in serum soluble transferrin receptor (sTfR) and decreases in serum ferritin observed in patients achieving HI-E or TI endpoints

KER-050 upregulated thrombopoiesis

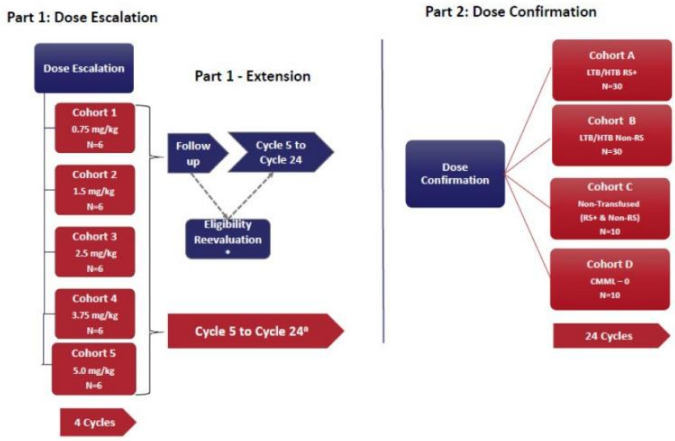
- Sustained increases in platelets observed in patients achieving HI-E or TI endpoints
- No patients required dose reduction due to thrombocytosis
- Preclinical data demonstrate this effect could potentially be mediated by KER-050 inhibition of activin A

Summary of KER-050 Phase 2 Clinical Trial

- Keros believes the additional data from this 12-week treatment Phase 2 clinical trial demonstrate proof-of-concept of KER-050 in patients with very low-, low- or intermediate-risk MDS
  - Data consistent with observations from the Phase 1 clinical trial in healthy volunteers
- **Preliminary Efficacy Data**
  - Clinically meaningful reductions in transfusion burden as well as transfusion independence were observed in both RS+ and non-RS patients as of the data cut-off date
- **Safety Data**
  - Dose levels as of the data cut-off date were generally well tolerated as of the data cut-off date
- **PD Markers**
  - Increases in hematological parameters were observed in RS+ and non-RS patients that received doses of KER-050 Q4W as of the data cut-off date
    - Increases in reticulocytes, hemoglobin and platelets were observed
  - Observed increases in reticulocytes and soluble transferrin receptor and observed decreases in serum ferritin suggest that administration of KER-050 is potentially associated with increased erythropoiesis, with a broader effect on hematopoiesis being suggested by the increase in platelets

Updated Design of KER050-MD-201

- Cohort 4 Safety Review Committee permitted dosing of participants in Cohort 5 at 5.0 mg/kg, Q4W for 24 months
- Preparing to initiate Part 2 of this trial, with centers participating globally
- Data from Part 2 will inform our registration plans for KER-050
- Part 2 will explore KER-050 in larger cohorts of RS+ and non-RS patients
- A group of non-transfused patients and one of chronic myelomonocytic leukemia will also be included

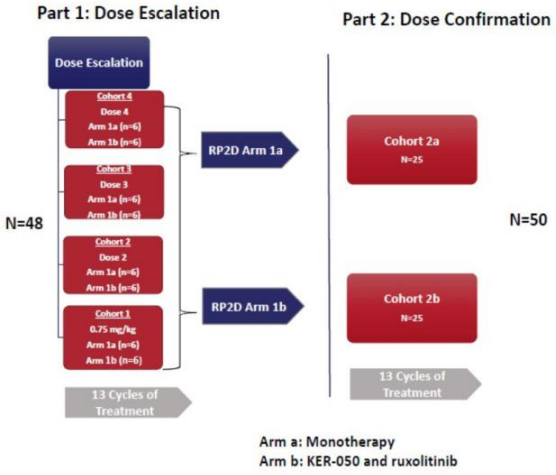


KER050-MF-301

A Phase 2 Clinical Trial to Evaluate KER-050 as a Monotherapy or in Combination With Ruxolitinib in Myelofibrosis

Phase 2 Clinical Trial of KER-050 in Patients with Myelofibrosis-Associated Cytopenias

- Ongoing open label, two-part, multiple ascending dose Phase 2 clinical trial to evaluate KER-050 as a monotherapy and in combination with ruxolitinib in participants with myelofibrosis-associated cytopenias
- Primary objectives: safety and tolerability
- Secondary objectives: evaluate the pharmacokinetics, pharmacodynamics and efficacy of KER-050 administered with or without ruxolitinib
- Expect to report initial data from this trial by the end of 2022



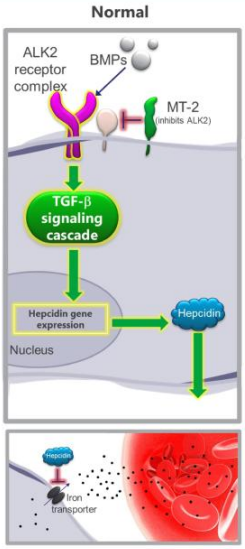
KER-047

A novel product candidate designed to address:

- Anemia resulting from iron imbalance
  - Iron deficiency anemia
  - IRIDA

ALK2 Regulates Hepcidin and Iron Homeostasis

- ALK2 signaling in the liver controls hepcidin expression, a hormone that controls iron homeostasis
- Excessive ALK2 signaling results in high hepcidin and a shortage of iron availability for RBC production
- ALK2 signaling requires BMP ligand and the co-receptor hemojuvelin
- Hepcidin expression is tightly regulated and controls expression of the ALK2 suppressor protease MT-2
  - The genetic disease iron-refractory iron deficiency anemia (IRIDA) is characterized by loss of MT-2
  - High hepcidin has also been implicated in anemia of inflammation
- Modulating ALK2 signaling will normalize high hepcidin levels, restore serum iron and ameliorate anemia



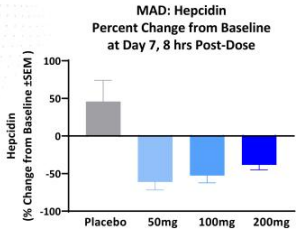
Inhibition of ALK2 Demonstrated Activity in Rodent Models of Iron Imbalance

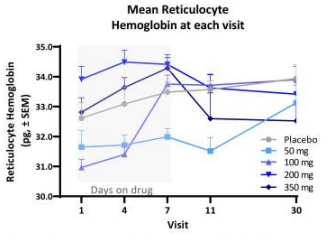
- ALK2 inhibition decreased hepcidin and increased serum iron in mice
- In a mouse model of IRIDA, treatment with ALK2 inhibitors reduced hepcidin and ameliorated anemia
- In a mouse model of chronic kidney disease, chronic inflammation resulted in increased hepcidin, reduced serum iron and anemia
  - Treatment with an ALK2 inhibitor reduced hepcidin, increased serum iron and resolved anemia
- Frequent infusions of red blood cells or iron (intravenous) results in iron overload in the liver, heart and other tissue
  - Treatment with an ALK2 inhibitor mobilized the iron and reduced iron deposits in the liver in mice

Inhibition of ALK2 has the potential to restore iron balance and treat patients with anemia and patients with iron overload

Phase 1 Clinical Trial: KER-047 Treatment Led to Reduced Hepcidin Levels and Increased Hemoglobin Content in Reticulocytes

- KER-047 is a small molecule inhibitor of ALK2 with low nanomolar IC_{50}
- PK/ADME: Suitable for 1x daily oral dosing
- There were no serious adverse events reported in the randomized, double-blind, placebo-controlled two-part Phase 1 clinical trial of KER-047 in healthy volunteers





- Consistent with ALK2 inhibition, decreases in serum hepcidin were observed in Cohorts 1 through 3 of Part 2 of the expanded trial
- Treatment related decreases in hepcidin resulted in increased serum iron

- An increase in reticulocyte hemoglobin was observed in Cohorts 1 through 4 of Part 2 of the expanded trial, starting on Day 4 of treatment
- Pronounced increase in reticulocyte hemoglobin observed in cohorts with lower baseline reticulocyte hemoglobin

Phase 2 Trials to Provide Proof-of-Concept for Treatment of Anemia Resulting from Iron Imbalance, Including IDA and IRIDA

Iron Deficiency Anemia

- KER-047 is designed to re-establish normal iron homeostasis by mobilizing iron out of tissues, thereby ameliorating anemia
- We expect to initiate a Phase 2 clinical trial in patients with iron deficiency anemia in H1 2022 and expect to report initial data from this trial by the end of 2022

IRIDA

- KER-047 is designed to normalize high hepcidin levels, restore serum iron and ameliorate anemia
- We expect to initiate a Phase 2 clinical trial in patients with IRIDA in H1 2022 and expect to report initial data from this trial by the end of 2022

KER-012

A clinical program designed to address:

- Pulmonary arterial hypertension (PAH)
- Bone loss disorders such as osteoporosis and osteogenesis imperfecta

KER-012: Product Candidate for Treatment of PAH and Bone Disorders

Proprietary selective activin receptor ligand trap in clinical development for the treatment of pulmonary arterial hypertension (PAH) and bone disorders

In preclinical studies, KER-012:

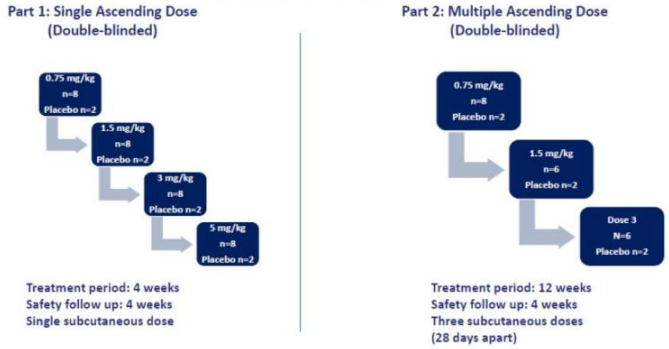
- Demonstrated effects on bone
  - Exhibited high affinity for, and potent inhibition of, ligands involved in the regulation of bone homeostasis
  - Increased bone mineral density and trabecular bone volume in wild-type mice and mice with established osteoporosis
  - Rats receiving a rodent version of KER-012 (RKER-012) were protected from hypoxia-associated bone loss
- Demonstrated benefit in models of PAH
  - In a rat model of PAH, rats receiving RKER-012 were protected from the thickening of the right ventricular wall
  - In a mouse model of pulmonary arterial banding (PAB), RKER-012 was observed to protect against both the PAB-related cardiac dysfunction and remodeling
- Did not increase red blood cell production in cynomolgus monkeys

We believe KER-012 has the potential to increase the signaling of BMP pathways by inhibiting activin A and activin B signaling and, consequently, treat diseases such as PAH that are associated with reduced BMP signaling

Phase 1 Clinical Trial of KER-012 in Healthy Volunteers

- Ongoing randomized, double-blind, placebo-controlled, two-part Phase 1 clinical trial to evaluate single and multiple ascending doses of KER-012 in healthy volunteers
- Trial objectives: safety, tolerability and pharmacokinetics

Phase 1 Clinical Trial Design



- Expect to report initial data from Part 1 of this trial in the second quarter of 2022 and additional data from Part 2 of this trial in the second half of 2022

Keros Summary

We Believe Keros is Positioned for Clinical and Commercial Success

- Keros is focused on the development of novel TGF-β therapeutics
  - Robust biology that has been validated in the clinic
- Keros is well-positioned to harness the potential of the TGF-β superfamily
  - ActRII program (KER-050) is in a Phase 2 trial in patients with MDS and a Phase 2 trial in patients with MF
  - Multiple Phase 2 trials for ALK2 program (KER-047) expected to commence in H1 2022
  - KER-012 is a selective activin receptor ligand trap with an ongoing Phase 1 trial in healthy volunteers; initial data expected from Part 1 of this trial in Q2 2022
  - Clinical programs have potentially differentiated mechanism of action
- Our discovery approach has the potential to identify additional molecules with differentiated profiles from existing third-party products and product candidates
  - Pipeline of preclinical assets: bone, muscle and pulmonary

Anticipated Key Milestones*

KER-050

• Announce additional data from Phase 2 trial in MDS	Mid-2022
• Announce initial data from Phase 2 trial in myelofibrosis	End of 2022

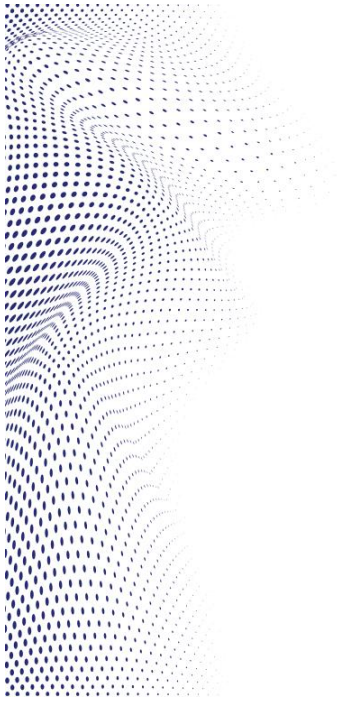
KER-047

• Initiate Phase 2 trial in IDA	H1 2022 (initial data end of 2022)
• Initiate Phase 2 trial in IRIDA	H1 2022 (initial data end of 2022)

KER-012

• Announce initial data from Part 1 of Phase 1 trial	Q2 2022
• Announce additional data from Part 2 of Phase 1 trial	H2 2022

*Anticipated preclinical and clinical milestones are subject to the impact of COVID-19 on our business.



Thank You