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): August 4, 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 5.02 Departure of Directors or Certain Officers; Election of Directors; Appointment of Certain Officers; Compensatory Arrangements of Certain Officers.

On August 1, 2022, Keros Therapeutics, Inc. (the "Company") entered into an offer letter (the "Offer Letter") with Jennifer Lachey, which superseded in full Dr. Lachey's Executive Employment Agreement dated March 16, 2020, as amended by that certain amendment dated January 1, 2022 (the "Prior Employment Agreement"), to be effective September 1, 2022. Pursuant to the Offer Letter, Dr. Lachey's schedule and benefits eligibility will be adjusted to 75% of that for a full-time employee. Dr. Lachey's base salary will also be adjusted to be 75% of her previous base salary. In addition, as of the effective date, the number of shares subject to the then unvested portions of Dr. Lachey's remaining options (the "Unvested Options") will be adjusted, such that only 75% of the number of shares subject to each of the Unvested Options will remain outstanding.

Under the terms of the Offer Letter, Dr. Lachey is no longer eligible for severance upon a termination without cause or a resignation for good reason. By entering into the Offer Letter, Dr. Lachey acknowledged that the change in her terms of employment was at her request and the Offer Letter does not constitute grounds for "good reason" pursuant to the Prior Employment Agreement.

In addition, the Board determined that the duties and responsibilities of Dr. Lachey have evolved such that she is no longer an "officer" within the meaning of Rule 16a-1(f) under the Securities Exchange Act of 1934, as amended (the "Exchange Act") or an "executive officer" within the meaning of Rule 3b-7 under the Exchange Act. She remains employed by the Company and her title has been changed from Chief Scientific Officer to Senior Vice President of Discovery.

Item 7.01 Regulation FD Disclosure.

On August 4, 2022, 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 under Item 7.01 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 Exchange Act or otherwise subject to the liabilities of that section. Such information and the accompanying Exhibit 99.1 are 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 August 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: August 4, 2022

KEROS THERAPEUTICS

Corporate Presentation

August 2022

Disclaimer

Statements contained in this presentation by Keros Therapeutics, Inc. ("Keros", "we" or "our") 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 preclinical studies and clinical trials for KER-050, KER-047 and KER-012, including its regulatory plans; 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 Keros' Annual Report on Form 10-K, filed with the SEC on March 9, 2022, and Keros' Quarterly Reports on Form 10-Q, filed with the SEC on May 5, 2022, and August 4, 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 our own internal estimates and research. While we believe these third-party sources to be reliable as of the date of this presentation, we have not independently verified, and make 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, Pulmonary, and Musculoskeletal Disorders

Program	Asset	Phase of Development	Status	Next Milestones*
Hematology	KER-050 (therapeutic protein)	Myelodysplastic syndromes (Preclinical → Phase 2)	Phase 2 clinical trial ongoing	Additional data from the Phase 2 clinical trial: End of 2022
		Myelofibrosis (Preclinical → Phase 2)	Phase 2 clinical trial ongoing	Initial data: End of 2022
	KER-047 (small molecule)	Iron-refractory iron deficiency anemia (Preclinical → Phase 2)	Phase 2 clinical trial ongoing	Initial data: End of 2022
		Iron deficiency anemia (Preclinical → Phase 1)	Completed Phase 1 clinical trial	Initiate Phase 2 clinical trial: H2 2022 Initial data: H1 2023
Pulmonary	KER-012 (therapeutic protein)	Pulmonary arterial hypertension (Preclinical → Phase 1)	Phase 1 clinical trial in healthy volunteers ongoing	Additional data from Part 2 of the Phase 1 clinical trial: H2 2022
Musculoskeletal		Bone disorders (Preclinical → Phase 1)		
Preclinical Pipeline		Musculoskeletal and hematology (Preclinical)		

*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
  - 60,000-170,000 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

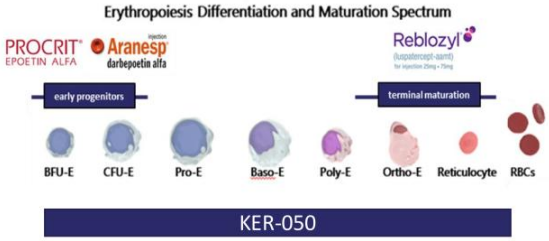
- Molecular abnormalities in the JAK-STAT pathway result in expansion of RBC and platelet precursors and subsequent ineffective hematopoiesis
- 16,000-18,500 MF patients in the US[2] with >3,000 newly diagnosed each year[3] and nearly all will become transfusion dependent[4]
- Megakaryocyte accumulation/breakdown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

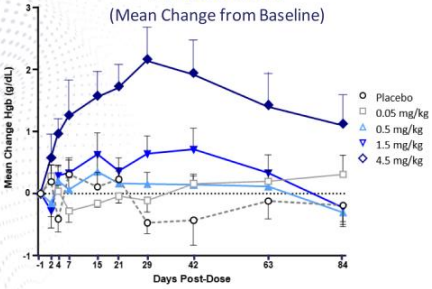


- Phase 1 clinical trial of KER-050 recapitulated preclinical data with observed rapid, sustained and dose-dependent increases in RBCs and platelets

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

HEMOGLOBIN

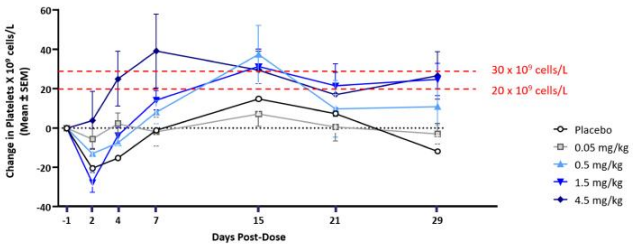
(Mean Change from Baseline)



- 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)



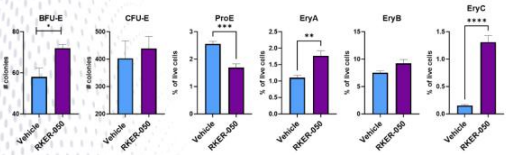
- Single doses of KER-050 observed to lead to clinically meaningful changes in platelets through day 29
- Maximum changes observed between 7-15 days post-dosing

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

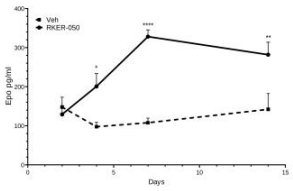
In a preclinical study of healthy mice, treatment with a mouse research form of KER-050 (RKER-050):

- 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

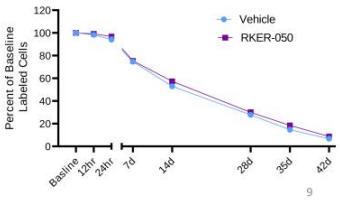
Erythropoietic tone upregulated 14 days after single RKER-050 injection



Single treatment with RKER-050 increased serum EPO



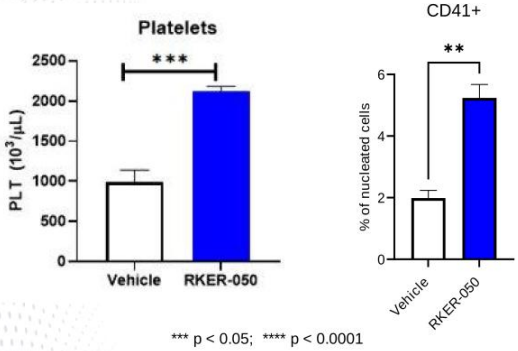
RKER-050 treatment did not alter RBC life span



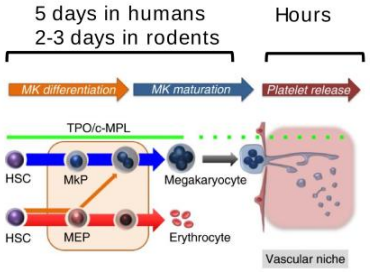
EHA 2021: Abstract # 2736

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

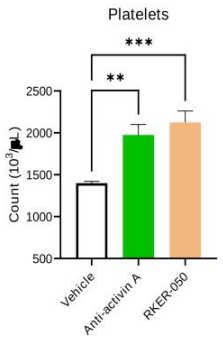


*** p < 0.05; **** p < 0.0001



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

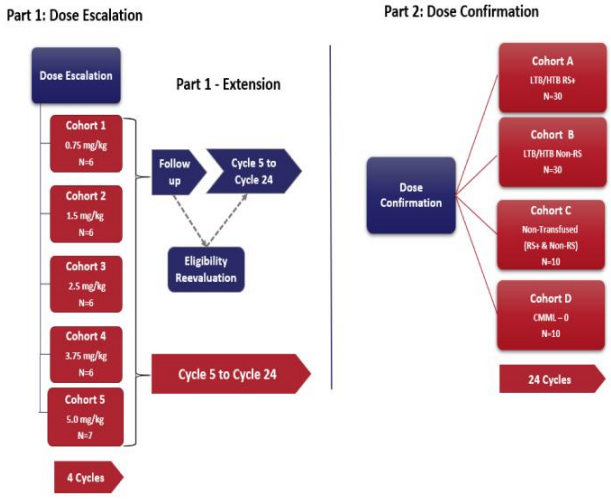
- KER-050 increased RBC parameters and platelets following single doses in a Phase 1 clinical trial in healthy volunteers
- In preclinical studies, a research form of KER-050 (RKER-050) was observed to increase RBCs and platelets, potentially through promotion of multiple stages of erythropoiesis and 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 (LR-MDS)
- KER-050 administered once every four weeks (Q4W)
- Trial objectives:
  - Part 1
    - Evaluate safety, tolerability and pharmacokinetics
    - Evaluate pharmacodynamic effects and efficacy of KER-050
  - Part 2
    - To confirm the safety, tolerability and efficacy of the dose(s) selected from Part 1
- Eligible patients in Part 1 and Part 2 may 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

- Data cut-off date: April 3, 2022
- Safety data from Cohorts 1 through 5 (0.75, 1.5, 2.5, 3.75 and 5.0 mg/kg, respectively)
  - 31 patients in Cohorts 1 through 5 received at least one dose of KER-050 as of the data cut-off date
- Efficacy data from Cohorts 1 through 5:
  - 27 patients in Cohorts 1 through 5 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:
    - 5 NT patients; 6 LTB patients; and 16 HTB patients
      - 2 LTB patients required <2 RBC units at baseline
    - Of the 20 LTB and HTB patients that required ≥2 RBC units at baseline, 8 were non-RS and 12 were RS+
    - 87% of patients had multilineage dysplasia

Safety Data as of the Data Cut-off Date (April 3, 2022)

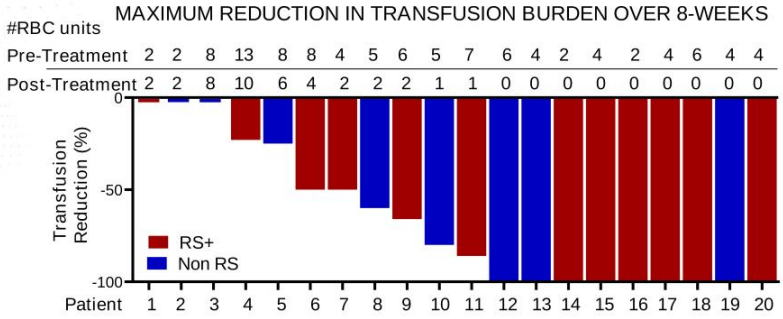
- Safety Review Committee has reviewed data from 0.75 mg/kg (Cohort 1), 1.5 mg/kg (Cohort 2), 2.5 mg/kg (Cohort 3), Cohort 4 (3.75 mg/kg) and Cohort 5 (5.0 mg/kg)
  - Following recommendation by Safety Review Committee, dosing of participants in Part 2 of the trial was initiated (starting dose of 3.75 mg/kg, with an opportunity for patients to dose escalate to 5.0 mg/kg based on individual titration rules)
  - Data from Part 2 will inform our registration plans for KER-050
- Summary of safety data as of the data cut-off date (Cohorts 1-5, n=31)
  - No drug-related serious adverse events (SAEs) or dose-limiting toxicities reported
  - 10 patients experienced treatment-emergent SAEs
  - Most commonly reported treatment-emergent adverse events (AEs):
    - Dyspnea, anemia, fatigue, diarrhea, headache and nausea
  - Treatment-related AEs reported in 5 patients were mild or moderate in severity
- 4 patients discontinued study drug: 1 withdrew consent, 1 death (unrelated to study drug, per autopsy due to obesity-associated heart disease), 2 withdrew due to unrelated TEAE
- No patients developed high-risk MDS or acute myeloid leukemia

Efficacy Summary of 8-Week Endpoints Achieved in MDS Patients

Response Summary	Response Rate, n/m (%)	
	All evaluable patients	HTB evaluable patients
Overall Erythroid Response (HI-E or TI)	14/27 (51.9%)	11/16 (68.8%)
IWG 2006 HI-E	12/26 (46.2%)	11/16 (68.8%)
Transfusion independence (TI*)	9/20 (45%)	7/16 (43.8%)
RS+	6/12 (50%)	4/9 (44.4%)
Non-RS	3/8 (37.5%)	3/7 (42.9%)

*Baseline Transfusion Requirement ≥2 RBC units
n = responders in each category; m = 8-week evaluable population as of data cutoff date

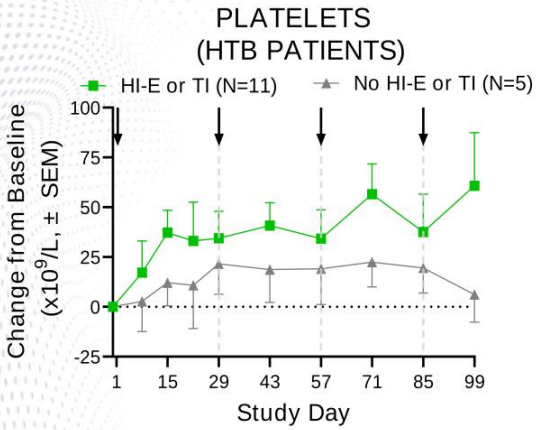
Treatment with KER-050 Resulted in HI-E and TI in Transfusion-Dependent Non-RS and RS+ MDS patients



- Improvements in transfusion burden were seen across LTB and HTB patients
- 7/16 HTB and 2/4 LTB patients achieved TI after KER-050 treatment

April 3, 2022 data cutoff date

Sustained Increase in Platelets Observed in HTB Patients Achieving HI-E or TI with KER-050 Treatment

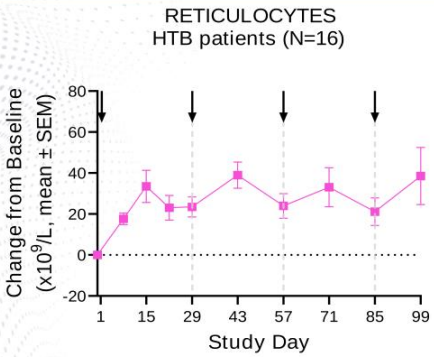


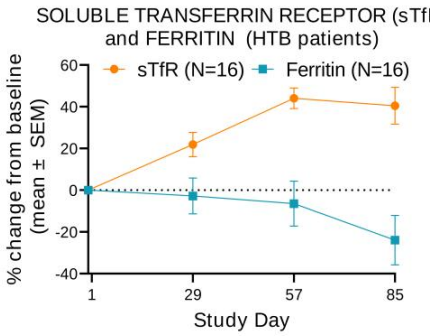
KER-050 upregulated thrombopoiesis

- Sustained increases in platelets observed in HTB 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

April 3, 2022 data cutoff date

Observed Changes in Hematologic and Ferrokinetic Biomarkers Support Induction of Erythropoiesis with KER-050 Treatment in all HTB Patients





- Increases in reticulocytes and soluble transferrin receptor, a biomarker of erythropoiesis, were observed in HTB patients
- Serum ferritin was elevated in HTB patients, indicative of transfusion-related iron overload
  - Mean baseline ferritin was 1,359.2 ng/mL
- Mean maximum reduction in ferritin was 29.1% following 3 months of treatment with KER-050

April 3, 2022 data cutoff date

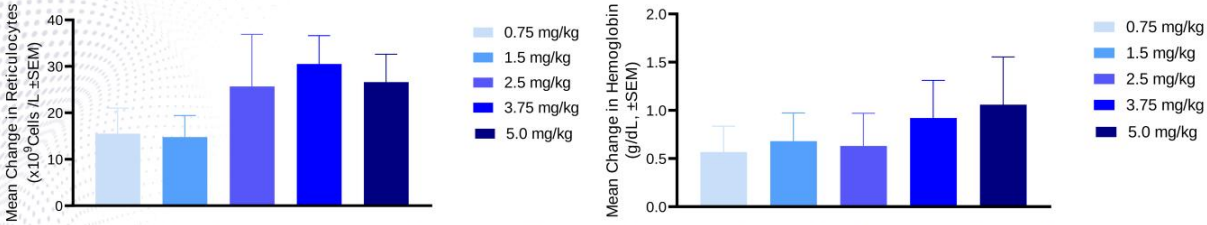
Summary of KER-050 Phase 2 Clinical Trial

- LR-MDS patients enrolled in Part 1 of this Phase 2 clinical trial were primarily transfusion-dependent with multilineage dysplasia
  - 58% of patients were HTB patients with elevated serum ferritin
- KER-050 was generally well-tolerated as of data cut-off date at doses ranging from 0.75 to 5.0 mg/kg Q4W
- No drug related SAEs or dose-limiting toxicities were observed
- Observed PD effects in reticulocytes, soluble transferrin receptor and platelets support the proposed KER-050 mechanism of increasing hematopoiesis
- HI-E and transfusion independence have been observed in both RS+ and non-RS MDS patients treated with KER-050 across varying transfusion burdens, with 44% of HTB patients achieving TI during this 3-month treatment trial
  - Reductions in serum ferritin were also observed in HTB patients
- These preliminary data support the potential of KER-050 as a treatment for multilineage cytopenias in LR-MDS, including difficult-to-treat HTB patients

April 3, 2022 data cutoff date

Phase 2 Part 2 Dose Confirmation has been Initiated

Markers of erythropoiesis during first 8 weeks of treatment



- Dose-related increases in reticulocytes and hemoglobin were observed in this primarily transfusion-dependent trial population
- Following recommendation by the Safety Review Committee dosing for Part 2 was initiated in June
  - Part 2 starting dose of 3.75 mg/kg Q4W with the option to up-titrate to 5 mg/kg Q4W
- Recommended Part 2 starting dose was based on:
  - Safety and tolerability data from patients treated with 0.75 to 5.0 mg/kg Q4W
  - Exposure response of hematological parameters, including reticulocytes and hemoglobin
  - Rates of HI-E and transfusion independence observed during 3-month treatment

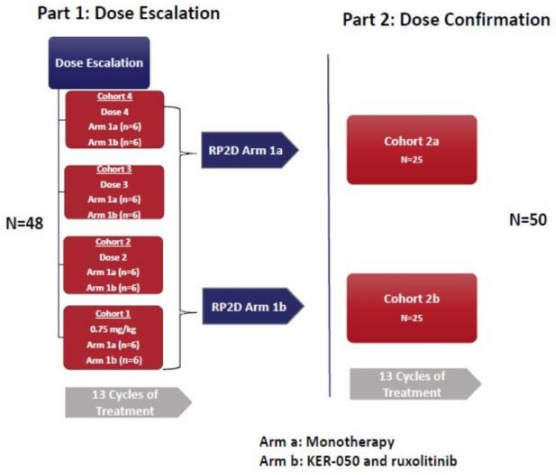
April 3, 2022 data cutoff date

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



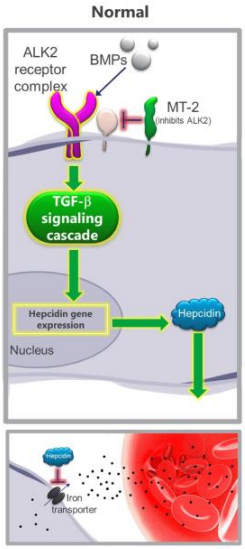
KER-047

A novel product candidate designed to address:

- Anemia resulting from iron imbalance
  - Iron Refractory Iron Deficiency Anemia (IRIDA)
  - Iron deficiency anemia (IDA)

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



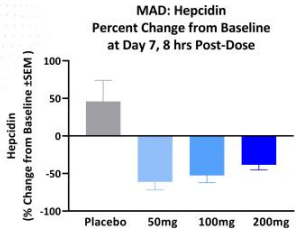
Inhibition of ALK2 Demonstrated Activity in Rodent Models of Iron Imbalance

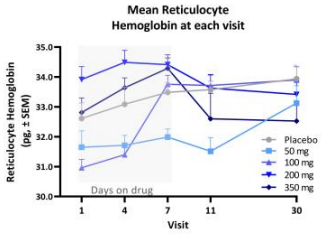
- ALK2 inhibition decreased hepcidin and increased serum iron in mice
- In a mouse model of IRIDA, treatment with ALK2 inhibitors reduced hepcidin and ameliorated anemia
- In a mouse model of chronic kidney disease, chronic inflammation resulted in increased hepcidin, reduced serum iron and anemia
  - 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 plan to initiate a Phase 2 clinical trial in patients with iron deficiency anemia in H2 2022 and expect to report initial data from this trial in H1 2023

IRIDA

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

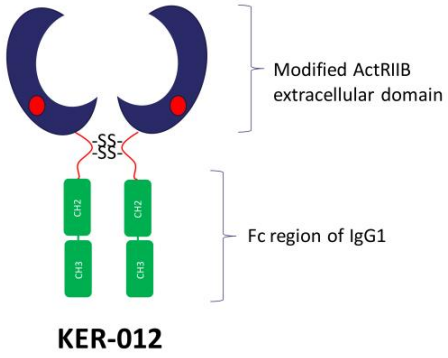
KER-012

A clinical program designed to address:

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

KER-012 is Designed to Address PAH and Bone Disorders

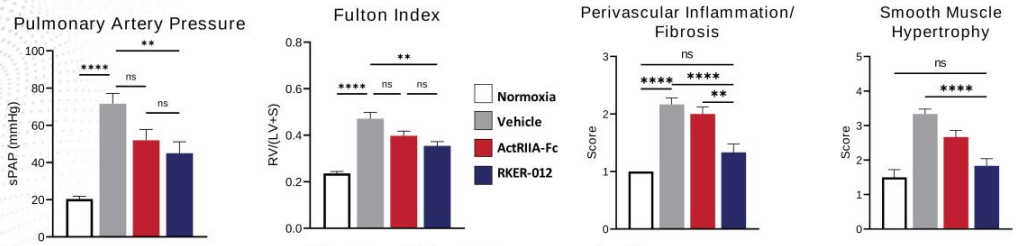
- KER-012 is a proprietary, wholly-owned, investigational ligand trap
  - Modified ActRIIB fused to the Fc region of IgG1
- KER-012 is designed to bind and inhibit activins and SMAD 2/3 signaling
- In preclinical studies, a research form of KER-012 (RKER-012):
  - Reduced inflammation, fibrosis and vascular remodeling in a rat Sugen/hypoxia model of PAH
  - Increased trabecular bone volume, bone volume fraction, trabecular number, trabecular thickness and reduced trabecular separation in the Sugen/hypoxia rat model
  - Did not increase red blood cells (RBCs) in rodents or cynomolgus monkeys in single and multiple dose studies
- A Phase 1 clinical trial in healthy postmenopausal volunteers is ongoing



KER-012

RKER-012 Reduced Pulmonary Arterial Pressure and Right Ventricle (RV) Hypertrophy in a Rat PAH Model

In a head-to-head preclinical study, ActRIIA-Fc and RKER-012 demonstrated activity in the Sugen/hypoxia rat model of PAH:

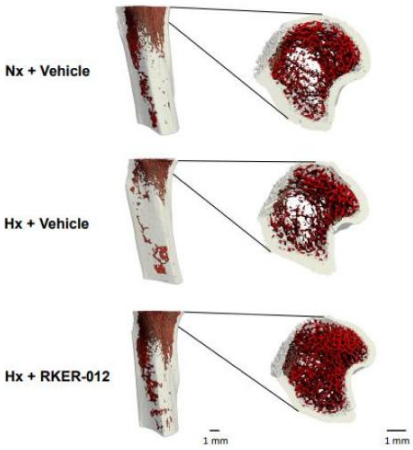


**p<0.01; ****p<0.0001; ns=not significant

- Hypoxic rats were dosed with vehicle, ActRIIA-Fc (10 mg/kg) or RKER-012 (10 mg/kg), twice weekly for three weeks
  - Normoxic rats were dosed with vehicle
- Relative to vehicle-treated hypoxic rats, RKER-012:
  - Statistically significantly reduced RV hypertrophy and pulmonary arterial pressure
  - Statistically significantly reduced lung inflammation, fibrosis and smooth muscle hypertrophy
- RKER-012 consistently showed a trend towards improved activity relative to ActRIIA-Fc in this preclinical study

RKER-012 Prevented Bone Loss in a Rat PAH Model

- In a separate preclinical study, RKER-012 demonstrated activity in improving bone mass in the Sugen/hypoxia rat model of PAH
  - Hypoxic rats were dosed with vehicle or RKER-012 (20 mg/kg), twice weekly for four weeks
  - Normoxic rats were dosed with vehicle
- Hypoxic rats dosed with vehicle exhibited decreased bone volume, bone volume fraction and trabecular number, and increased trabecular separation compared to normoxic controls
- RKER-012 prevented loss of bone volume, bone volume fraction, trabecular number, and reduced trabecular separation that was observed in vehicle-treated hypoxic rats
- Taken together, we believe this preclinical data suggests that:
  - RKER-012 potentially inhibited activins and growth differentiation factor ligands (GDFs), which are negative regulators of bone
  - Inhibition of activins and GDFs also potentially facilitated signaling of bone morphogenetic proteins (BMPs), factors that promote bone growth
  - RKER-012 protected rats from PAH-induced bone loss



(Left) Representative three-dimensional of the tibia demonstrating trabecular architecture is reduced in Hx + Vehicle compared to Nx + Vehicle and Hx + RKER-012. (Right) Transverse cross section of the proximal tibia depicting trabecular (red) and cortical (opaque) bone; Scale bar = 1 mm.

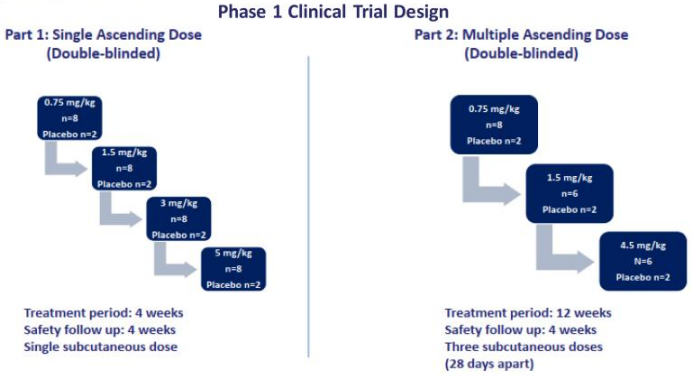
KER-012 Phase 1 Clinical Trial

A Randomized, Double-Blind, Placebo Controlled, Two-Part, Dose-Escalation Phase 1 Clinical Trial to Evaluate the Safety, Tolerability, Pharmacokinetic, and Pharmacodynamic Effects of KER-012 in Healthy Post-Menopausal Women

Phase 1 Clinical Trial of KER-012 in Healthy Post-Menopausal Women

Ongoing randomized, double-blind, placebo-controlled, two-part Phase 1 clinical trial to evaluate single and multiple ascending doses of KER-012 in healthy post-menopausal women

Phase 1 Clinical Trial Design



Part 1 endpoints: safety, pharmacokinetics (PK) and biomarkers

Status: Completed; topline data shared in this presentation

Part 2 endpoints: safety, PK, biomarkers and total body scan by dual-energy x-ray absorptiometry (DXA)

Status: Part 2 ongoing; expected to report data in H2 2022

Key Inclusion and Exclusion Criteria

Inclusion:

- Postmenopausal female aged 45 to 70 years (inclusive) at screening
  - NOTE: Postmenopausal is defined as ≥ 6 months of spontaneous amenorrhea OR 6 weeks postsurgical bilateral oophorectomy with or without hysterectomy
- Serum follicle-stimulating hormone (FSH) levels > 40 IU/L

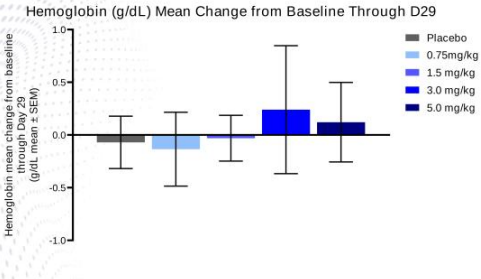
Exclusion:

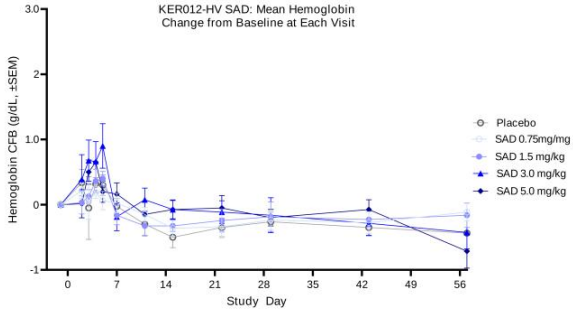
- Clinically significant (as determined by the investigator) cardiac, endocrinologic, hematologic, hepatic, immunologic, metabolic, urologic, pulmonary, neurologic, dermatologic, psychiatric, renal, and/or other major disease
- History of osteoporosis or any past treatment for osteoporosis
- Hormone replacement therapy (i.e., estrogen, or estrogen plus progesterone) within 3 months prior to dosing or plans to begin hormone replacement therapy at any time during the study. Local estrogen therapy for vaginal atrophy is permitted
- Systemic glucocorticoid therapy for more than 1 month within 6 months before screening
- Medications that may affect muscle function, including muscle anabolic agents and high intensity statins, within 3 months prior to dosing (moderate stable doses of statins are permitted)
- Antiresorptive and anabolic osteoporosis treatments within 1 year prior to dosing

Safety, Tolerability and PK (Part 1 SAD)

- KER-012 was generally well tolerated at doses up to 5 mg/kg when administered as a single dose
- There were no serious adverse events observed in Part 1
- The majority of adverse events observed in Part 1 were mild in severity (CTCAE Grade 1)
- No clinically meaningful changes in hemoglobin (Hgb), RBCs or reticulocytes were observed at doses up to 5 mg/kg when administered as a single dose
- PK parameters were generally dose proportional with increasing doses

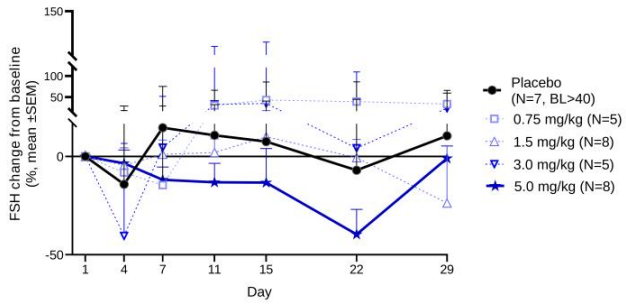
No Clinically Meaningful Change in Hgb Observed with KER-012 Administration of up to 5 mg/kg





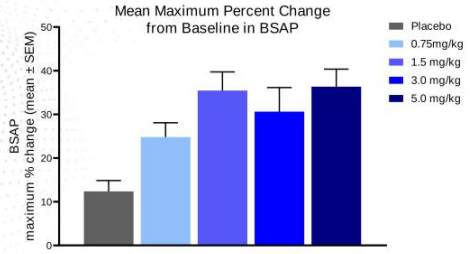
- Single dose of KER-012 was not associated with clinically meaningful changes in Hgb at all doses in Part 1 of this trial

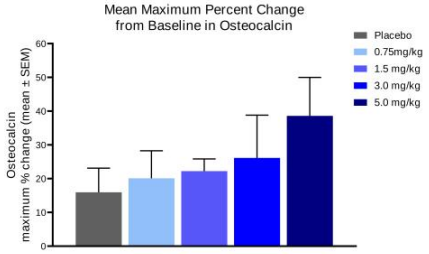
KER-012 Administration Resulted in 40% Mean Decrease in FSH at 5 mg/kg Dose



- As per the study protocol, only participants with baseline FSH ≥40 IU/L were included in the analysis for the changes in FSH with KER-012 treatment
  - Some of the participants that met the ≥40 IU/L criteria for FSH at screening dropped below the inclusion criteria at baseline
- A single dose of 5 mg/kg resulted in a 39.6% mean decrease in FSH on Day 22

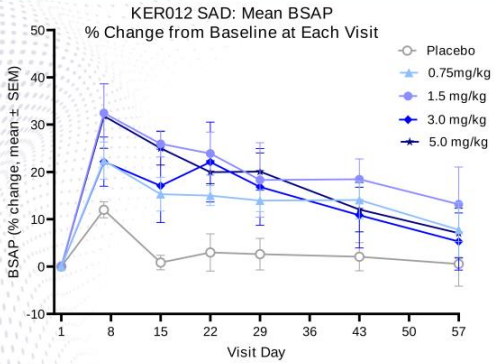
Robust Increase in Markers of Bone Formation Observed

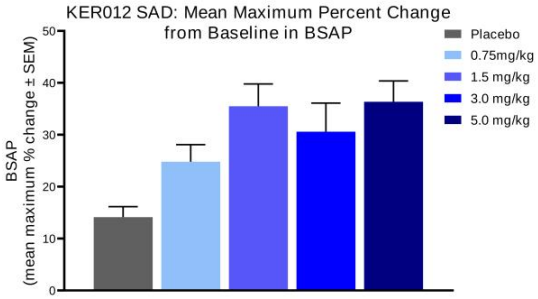




- KER-012 is designed to inhibit activins and GDFs in the bone, which we believe potentially results in reduced SMAD 2/3 signaling and increased signaling of bone morphogenetic protein (BMP) pathway (SMAD1/5/8)
  - The increased BMP signaling potentially promotes bone formation through activation/recruitment of bone forming osteoblasts and repression of osteoclasts
- Increased serum markers of osteoblast activity were observed in trial participants who were administered KER-012
  - Including bone specific alkaline phosphatase (BSAP), procollagen type 1 N-terminal propeptide (P1NP) and osteocalcin

Observed Mean Maximal Increase in BSAP at Doses of 1.5 mg/kg and Higher





- A single 0.75 mg/kg dose of KER-012 elicited a 25% mean maximum increase in BSAP, which is supportive of osteoblast activation/recruitment in bone
- A 35% mean maximum increase in BSAP was observed following a single 1.5 mg/kg dose of KER-012

KER-012 Part 1 SAD Summary

- KER-012 was generally well tolerated at all doses up to 5 mg/kg when administered as a single dose in healthy postmenopausal women
- KER-012 was associated with generally dose proportional exposure
- Maximal target engagement was observed following a single 5 mg/kg dose of KER-012 (39.6% mean reduction in FSH on Day 22)
- No clinically meaningful changes in Hgb or RBCs were observed at doses up to 5 mg/kg when administered as a single dose
- Robust changes in markers of bone formation were observed, starting at the lowest dose of 0.75 mg/kg
- Mean maximal increases in BSAP as high as 36.4% were observed at the higher dose cohorts (1.5, 3 and 5 mg/kg), which is similar to the mean maximal increase observed with other ligand traps, including KER-050
- The observed KER-012-mediated increases in BSAP are consistent with restoration of BMP signaling; Keros believes this supports the development of KER-012 as a potential treatment for patients with PAH, which is associated with reduced BMP signaling
- Keros believes the preclinical data and data from Part 1 of its ongoing Phase 1 clinical trial support that KER-012 has the potential to treat patients with PAH without a potentially dose-limiting red blood cell effect, if approved
- Part 2 of this trial (multiple ascending dose) is ongoing; expected to report data in H2 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
  - KER-047 Phase 2 trial in IRIDA initiated in Q2 2022; initiating Phase 2 trial in IDA in H2 2022
  - KER-012 is a selective activin receptor ligand trap with an ongoing Phase 1 trial in healthy volunteers; additional data expected from Part 2 of this trial in H2 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	End of 2022
• Announce initial data from Phase 2 trial in myelofibrosis	End of 2022

KER-047

• Announce initial data from Phase 2 in IRIDA	End of 2022
• Initiate Phase 2 trial in IDA	H2 2022; Initial data H1 2023

KER-012

• Announce additional data from Part 2 of Phase 1 trial	H2 2022
• Announce design of Phase 2 trial in PAH	Early 2023

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

Thank You