KEROS THERAPEUTICS

Corporate Presentation

June 2021

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Harnessing the Powerful Biology of the TGF-β Superfamily

- Clinical-stage biopharmaceutical company developing novel therapeutics that target the TGF-β superfamily
- Approach validated by marketed products, Infuse™ (BMP2) for spinal fusion and Reblozyl® (modified activin receptor IIB) for treatment of anemia in β-thalassemia and myelodysplastic syndromes (MDS)
- 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 (MDS)	Phase 2 clinical trial ongoing	Announce additional data from Part 1 of Phase 2 clinical trial: end of 2021
		Myelofibrosis	Completed Phase 1 clinical trial in healthy volunteers	Initiate Phase 2 clinical trial: Q3 2021 Initial data: 2022
	KER-047 (small molecule)	Iron deficiency anemia	Completed expanded Phase 1 clinical trial in healthy volunteers	Initiate Phase 2 clinical trial: H2 2021 Initial data: 2022
Musculoskeletal		Anemia from high hepcidin		Initiate Phase 2 clinical trial: H2 2021 Initial data: 2022
		Fibrodysplasia ossificans progressiva (FOP)		
	KER-012 (therapeutic protein)	Bone disorders	Ongoing preclinical studies	Initiate Phase 1 clinical trial: H2 2021 Initial data: H1 2022
Pulmonary		Pulmonary arterial hypertension		
Preclinical Pipeline		Musculoskeletal and hematology		

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

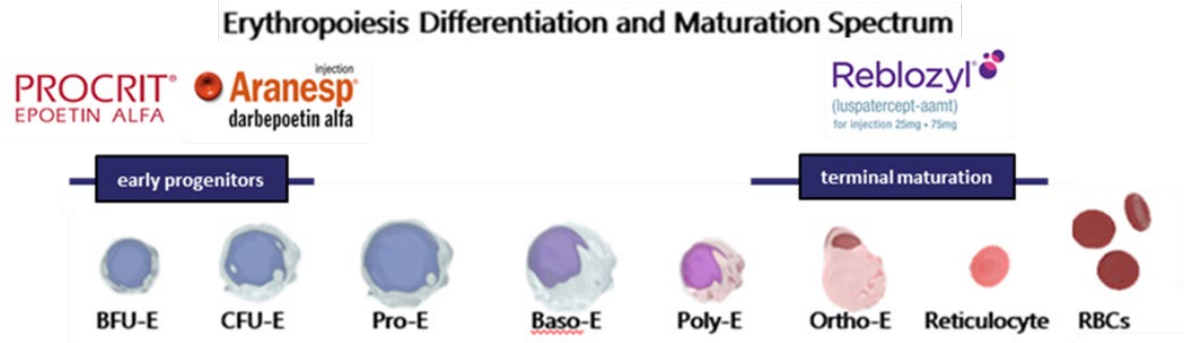
KER-050

A novel treatment designed to address diseases arising from ineffective hematopoiesis

- Myelodysplastic syndromes
- Myelofibrosis

Myelodysplastic Syndromes (MDS) Overview

- Hematologic malignancy predominantly affecting the elderly, resulting in multiple cytopenias (anemia, thrombocytopenia, neutropenia) due to ineffective hematopoiesis
  - 60,000-170,000 MDS patients in U.S.*
  - 15,000-20,000 newly diagnosed MDS patients in U.S. each year*
- 90% of patients are anemic and 40-65% have thrombocytopenia
- Platelet transfusion is the current treatment option for thrombocytopenia
- Anemia treatments include red blood cell (RBC) transfusions, erythropoiesis-stimulating agents (ESAs) and Reblozyl®
  - ESAs only impact early progenitors in red blood cell lineage and 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 requiring transfusions
  - Approximately 15% of all MDS patients are RS positive and have defects in terminal maturation
    - 38% responders vs 13% placebo
    - Similar to ESAs, benefit primarily in low transfusion burden



*MDS Foundation

Myelofibrosis (MF) is Characterized by Ineffective Hematopoiesis

- Molecular abnormalities in JAK-STAT pathway result in expansion of RBC and platelet precursors and subsequent ineffective hematopoiesis
- Megakaryocyte accumulation/breakdown is implicated in the inducement of bone marrow fibrosis
- Plan to initiate a Phase 2 trial in MF in Q3 2021, evaluating effect on platelets and RBCs
  - We believe that KER-050 has the potential to address ineffective hematopoiesis, which is central to MF

16,000-18,500

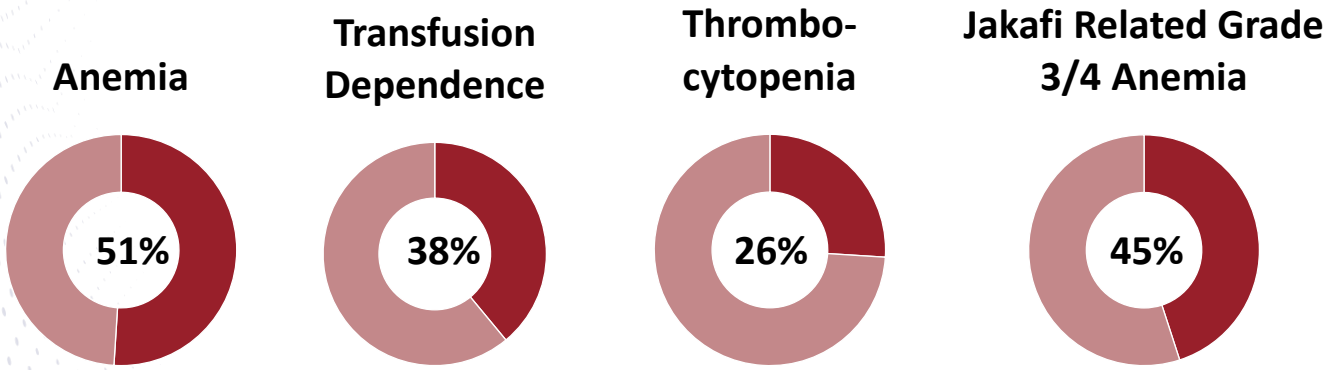
Prevalence of MF patients in US*

>3,000

New MF patients diagnosed each year**

~100 %

Nearly all MF patients will become transfusion-dependent***



Within 1 year of diagnosis

*Gangat 2011;**Srour 2016; ***Naymagon 2017

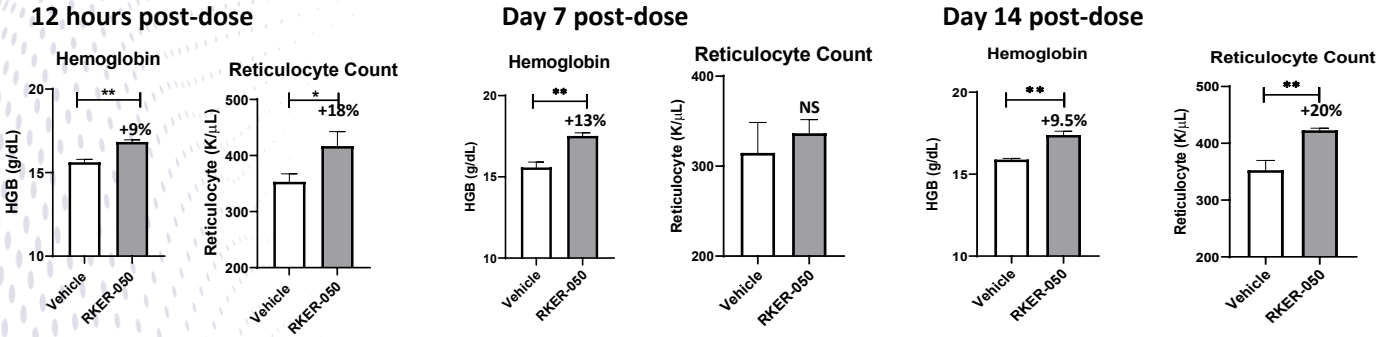
KER-050 is a Modified ActRII Fusion Protein

- 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
- Preclinical data demonstrate that increased RBCs by potentially increasing differentiation through multiple stages of erythropoiesis
  - Observed increases in platelets also potentially supports action throughout the thrombopoiesis pathway
- Phase 1 clinical trial of KER-050 recapitulated preclinical data with observed rapid, sustained and dose-dependent increases in RBCs and platelets

Treatment with RKER-050 Increased Erythropoiesis by Potentially Promoting Maturation at Multiple Stages and Increased Serum Erythropoietin

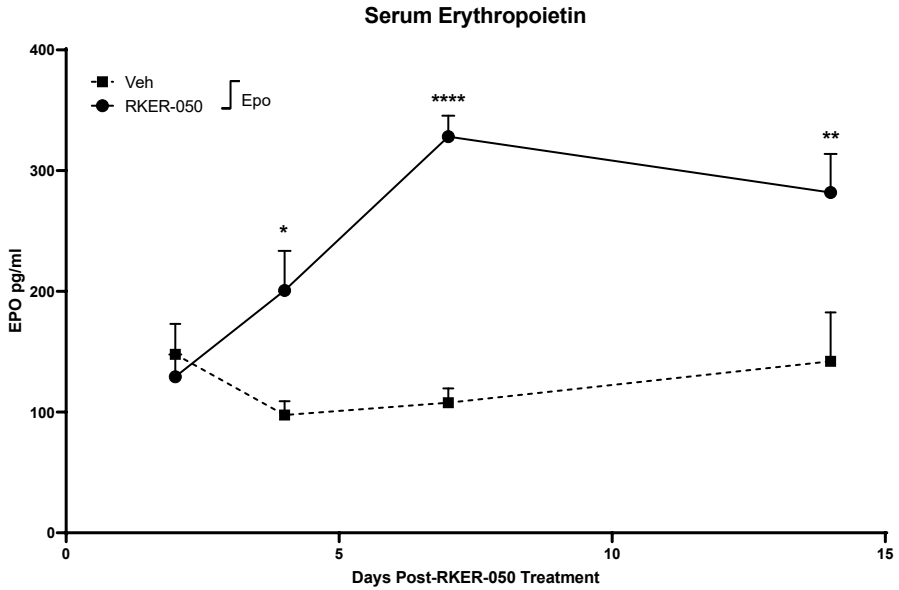
- In a preclinical study conducted in mice, a single, subcutaneous 10 mg/kg dose of a mouse version of KER-050 (RKER-050) resulted in:
  - Rapid increase in RBCs
  - Sustained increase continuing to at least 14 days post-dose
  - 2-3-fold increase in circulating erythropoietin
- KER-050 potentially acts on multiple stages across the RBC differentiation spectrum, including common myeloid cells

Increase in Red Blood Cells and Reticulocytes in Mice



*pv≤0.05; **pv≤0.01; ****pv≤0.0001

Observed Increase in Serum Erythropoietin in Mice



* P value ≤0.05; ** P value ≤0.01; **** P value ≤0.0001

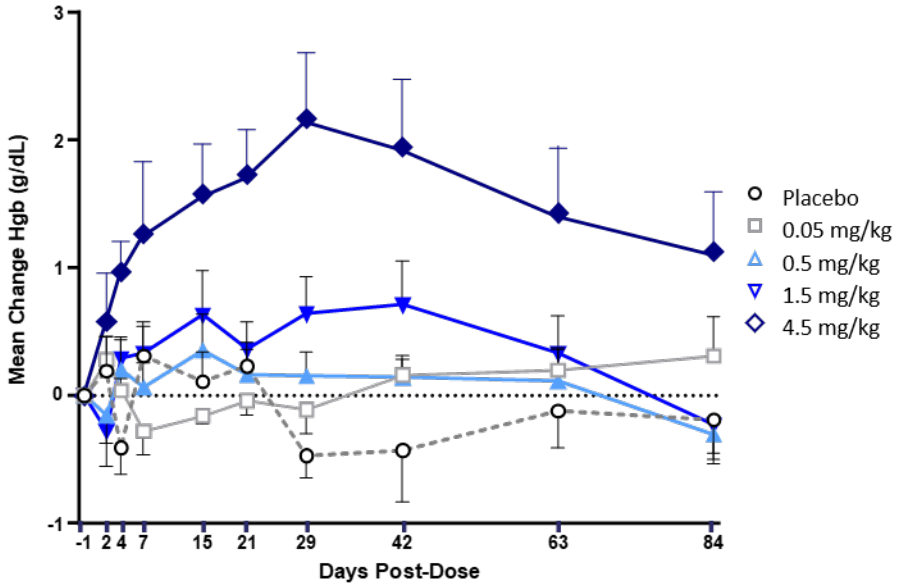
KER-050 Completed First-in-Human Trial

- First-in-human trial was designed to explore the safety, tolerability and PK in healthy volunteers with a secondary objective to evaluate changes in PD (hematology and bone biomarkers)
- Observed that KER-050 drug levels were dose proportional in Part 1 of the KER-050 Phase 1 clinical trial, with a mean half-life of approximately 12 days
  - The half-life coupled with the pharmacodynamic effect observed in the hematologic parameters support the potential for administration of monthly or less frequent dosing
- Observed to be well tolerated at dose levels up to 4.5 mg/kg, the highest dose level tested
- The most common adverse events observed in subjects in this trial were nausea, gastroenteritis, injection site erythema and, consistent with the mechanism of action of KER-050, increased hemoglobin and hypertension
  - Reversible, mild hypertension events observed in subjects with approximately 3 g/dL increase in hemoglobin

KER-050 Phase 1 Clinical Trial Recapitulated Learnings from Preclinical Studies

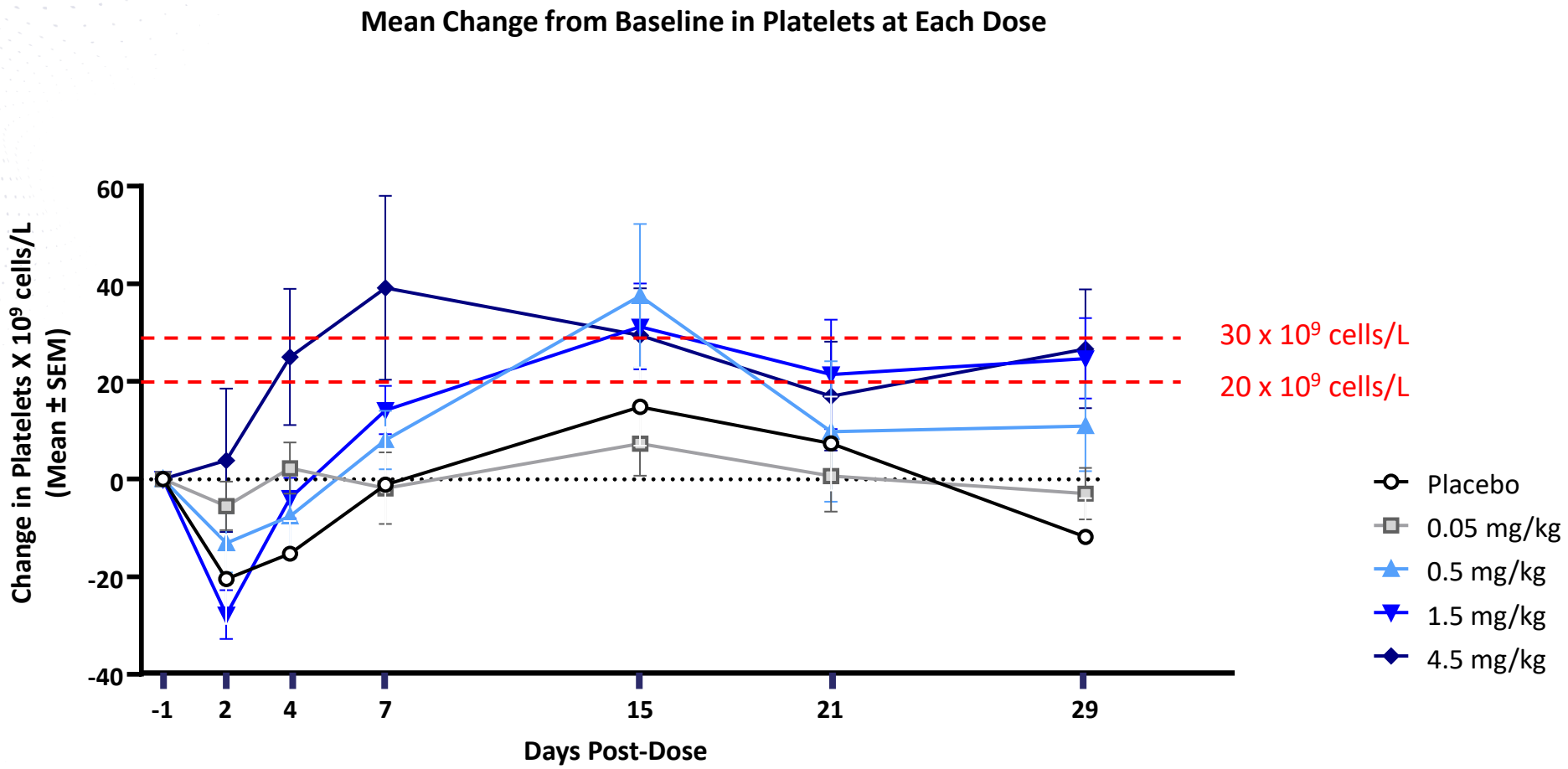
- Single, subcutaneous administration of KER-050 in healthy volunteers
- Observed rapid increase in red blood cell parameters is supportive of acceleration of maturation of late-stage precursors
  - Reticulocytes, red blood cells and hemoglobin
- Observed sustained increase from single dose supports monthly or less frequent dosing
  - Increases in RBC observed through day 29 are supportive of KER-050 acting on multiple stages of erythropoiesis
  - Maximum drug levels were observed on day 4

HEMOGLOBIN
Mean Change from Baseline



Mean change of >1.5 g/dl observed by day 15 in 4.5 mg/kg cohort

KER-050 Treatment was Observed to Lead to Clinically Meaningful Changes in Platelets after a Single Dose

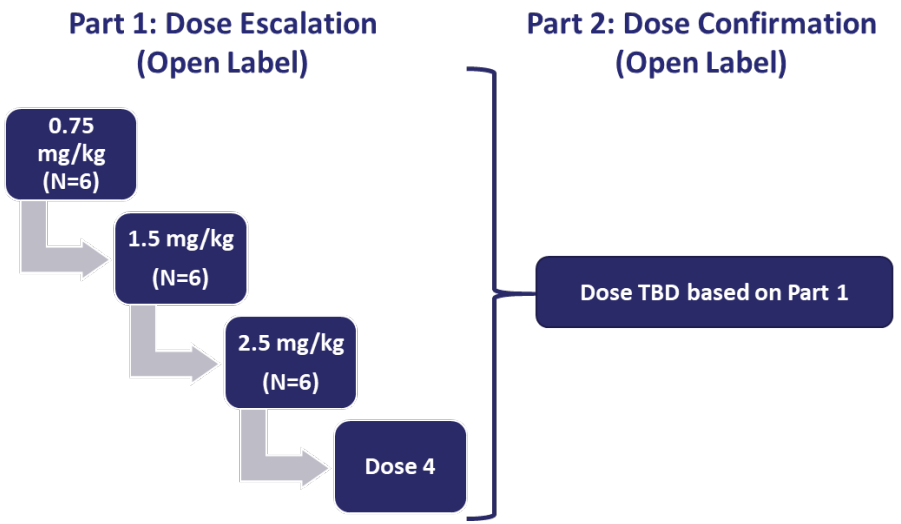


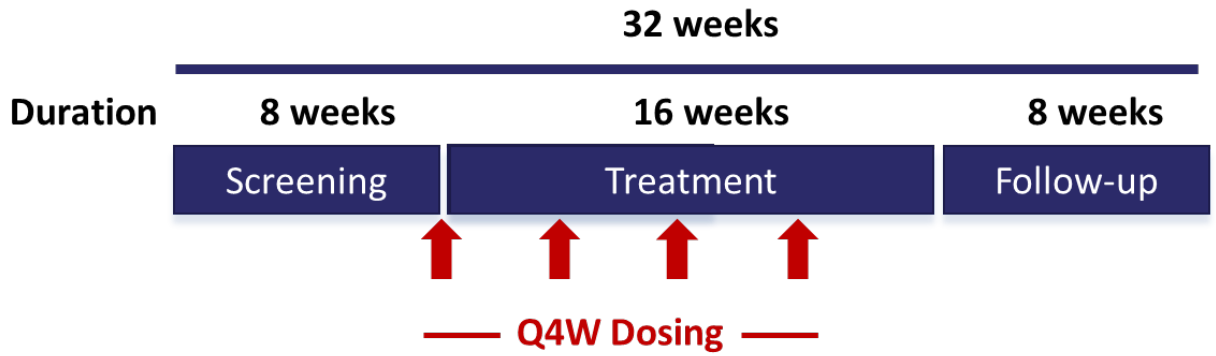
KER050-MD-201

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:
  - Safety, tolerability and pharmacokinetics
  - Evaluate pharmacodynamic effects and efficacy of KER-050
- Trial designed to evaluate KER-050 effects on hematopoiesis in:
  - Ring sideroblast (RS) positive and non-RS patients
  - ESA naïve and experienced
  - In high and low transfusion burden and non-transfused patients





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 RS positive and non-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:

- Hemoglobin increase of ≥1.5 g/ dL for 8 weeks (in NT and LTB patients)
- Reduction of ≥4 units or ≥50% units transfused over 8 weeks compared to baseline (in HTB patients)
- Transfusion independence for at least 8 weeks (in LTB and HTB patients)

Trial Status and Baseline Characteristics

- Data cut-off date: May 14, 2021
- Preliminary data presented from two lowest dose cohorts:
  - Cohort 1: 0.75 mg/kg Q4W for 12 weeks
  - Cohort 2: 1.5 mg/kg Q4W for 12 weeks
- 12 patients received at least one dose of KER-050 as of the data cut-off date
  - 9 patients completed 8 weeks of treatment with KER-050 as of the data cut-off date
  - 2 patients withdrew from the trial prior to completing 8 weeks of treatment with KER-050
  - 1 patient had not completed 8 weeks of treatment with KER-050 as of the data cut-off date
- Baseline characteristics (n=12):
  - 50% RS positive and non-RS
  - 50% had erythropoietin >100 mIU/mL
  - 50% HTB (≥ 4 units/8 weeks)
  - 85% had multilineage dysplasia
  - Mean platelet count of 192.4 x 10^9/L

Safety Profile as of May 14, 2021

- Safety Review Committee has reviewed preliminary 0.75 mg/kg (Cohort 1) and 1.5 mg/kg (Cohort 2) data
- Summary of safety profile* (Cohorts 1 and 2; n=12):
  - No drug related serious adverse events (SAEs)
    - 4 treatment-emergent SAEs deemed unrelated to study drug (anemia, febrile illness, pneumonia and death)
  - 1 treatment-related adverse event of maculopapular rash (Grade 2)
  - 2 withdrawals (death deemed unrelated to study drug; patient decision)
- Cohort 3 (2.5 mg/kg Q4W) has been initiated following Safety Review Committee recommendation

*Data cut-off date: May 14, 2021

Preliminary Results from Phase 2 Clinical Trial

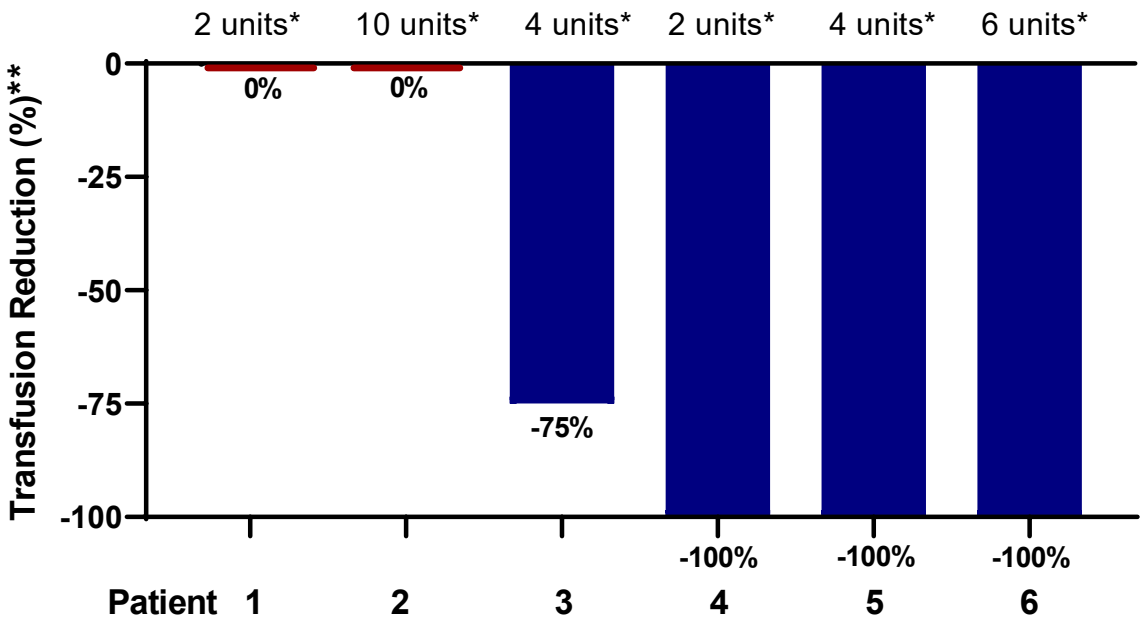
Preliminary results*:

- Consistent with the data from the Phase 1 clinical trial, increases in reticulocytes, Hgb and platelets were observed in MDS patients
- 5 patients that completed 8 weeks of treatment with KER-050 as of the data cut-off date met at least one of the following three endpoints:
  - Increase in hemoglobin ≥ 1.5 g/dL for 8 weeks, or
  - 50% reduction in transfusion requirements over 8 weeks, or
  - Transfusion independence for at least 8 weeks
- 3 patients achieved transfusion independence ≥ 8 weeks in duration
- Reduction in transfusions observed in both RS positive and non-RS patients

*Data cut-off date: May 14, 2021

Reduction in Transfusion Burden Observed+

- Patients requiring transfusions at baseline (≥2 RBC units/8 weeks) that completed 8 weeks of treatment with KER-050 as of the data cut-off date were evaluated for transfusion reduction
  - 6 patients required transfusion at baseline (2-10 RBC units over 8 weeks)
- Clinically meaningful reductions in transfusion burden as well as transfusion independence were observed with Q4W dosing schedule



+Data cut-off date: May 14, 2021
*Baseline transfusion burden over 8 weeks
**Percent transfusion reduction over 8 weeks on treatment compared to baseline transfusion burden

Summary of KER-050 Phase 2 Clinical Trial

- Keros believes the initial preliminary 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
- Increases in hematological parameters were observed in RS positive and non-RS patients that received doses of KER-050 Q4W
  - Increases in reticulocytes, hemoglobin and platelets were observed
- Clinically meaningful reductions in transfusion burden as well as transfusion independence were observed
- Lowest two doses were well tolerated as of the data cut-off date
- Cohort 3 dosing at 2.5 mg/kg Q4W has been initiated
- Keros plans to:
  - Update protocol to increase size of Part 2 (dose confirmation) to confirm response rates and guide design of registration program
  - Extend treatment duration from 12 weeks to up to 2 years to define response rate following 6 months of treatment, confirm durability of response and guide design of registration program
  - Share additional Part 1 dose-escalation data and Part 2 trial design by the end of 2021

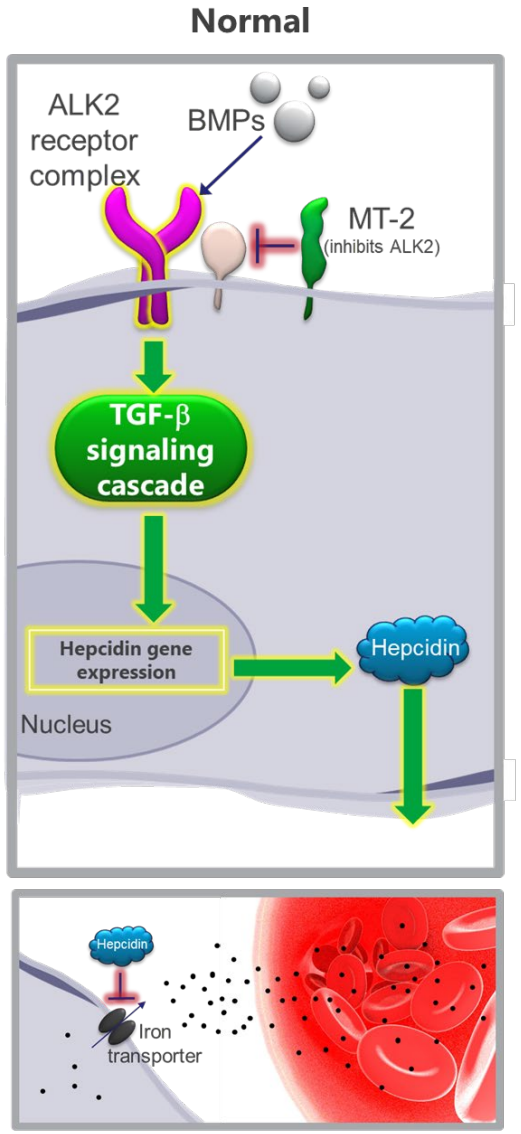
KER-047

A novel treatment designed to address:

- Anemia resulting from iron imbalance
  - Iron deficiency anemia
  - IRIDA
- Fibrodysplasia ossificans progressiva (FOP)

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

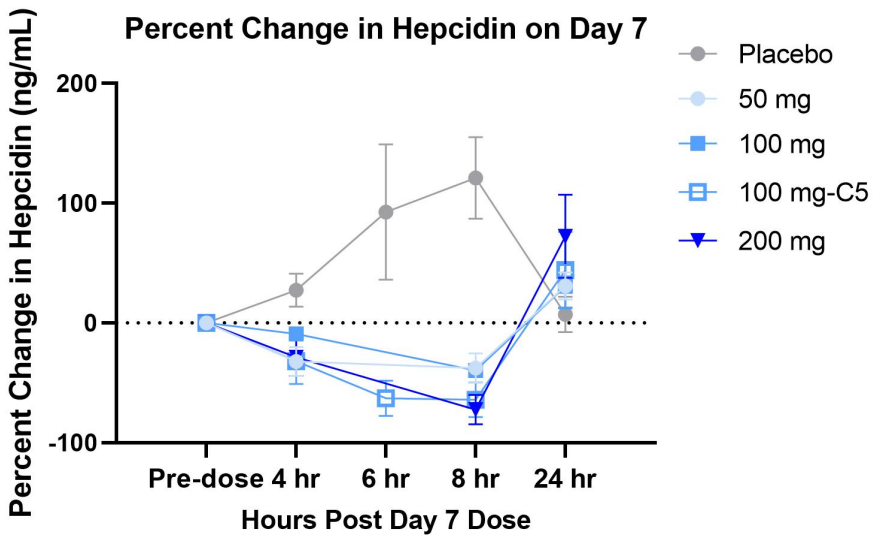
- ALK2 inhibition decreased hepcidin and increased serum iron in mice
- In a mouse model of IRIDA, treatment with ALK2 inhibitors reduced hepcidin and ameliorated anemia
- In a mouse model of chronic kidney disease, chronic inflammation resulted in increased hepcidin, reduced serum iron and anemia
  - 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

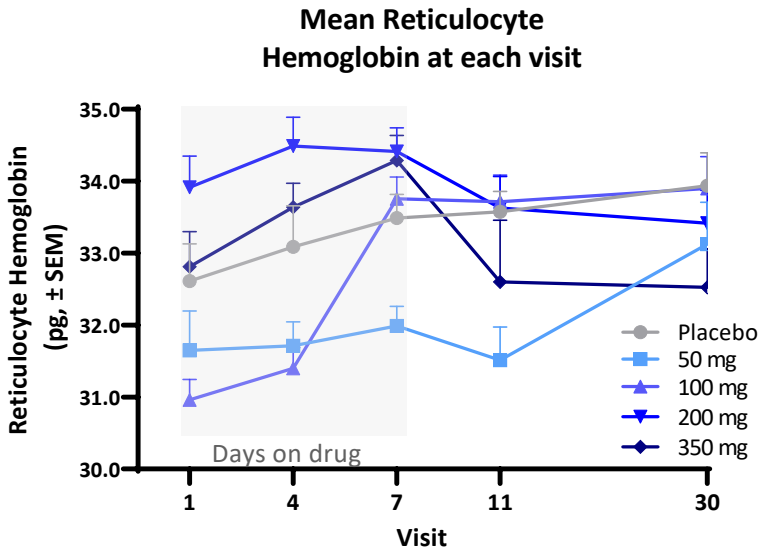
Inhibition of ALK2 has the potential to restore iron balance and treat patients with anemia and patients with iron overload

Phase 1 Clinical Trial of KER-047 in Healthy Volunteers

- KER-047 is a small molecule inhibitor of the ALK2 kinase domain with low nanomolar IC_{50}
- PK/ADME: Suitable for 1x daily oral dosing
- In November 2020, Keros completed a randomized, double-blind, placebo-controlled, two-part Phase 1 clinical trial to evaluate single and multiple ascending doses of KER-047 in healthy volunteers
- Primary objectives: Evaluate the safety, tolerability, pharmacokinetics and pharmacodynamic effects of single and multiple ascending dose levels of KER-047 in healthy volunteers
- Tolerability Profile:
  - There were no serious adverse events reported in the KER-047 Phase 1 clinical trial
  - Most common adverse events observed: abdominal discomfort, chills, decreased appetite, diarrhea, dizziness, fatigue, gastroenteritis, headache, lymphopenia, myalgia, nausea, neutropenia, pyrexia, rhinorrhea, tonsilitis, upper abdominal pain and vomiting

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





- Consistent with ALK2 inhibition, decreases in serum hepcidin were observed in Cohorts 1 through 3 of Part of the expanded trial

- 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 H2 2021 and expect to report initial data from this trial in 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 H2 2021 and expect to report initial data from this trial in 2022

KER-012

A preclinical program designed to address

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

KER-012: Preclinical Product Candidate

- Proprietary selective activin receptor ligand trap in preclinical development for the treatment of pulmonary arterial hypertension (PAH) and bone disorders
- In preclinical studies, KER-012:
  - Demonstrated 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
  - Did not increase red blood cell production in cynomolgus monkeys
- In a rat model of PAH, rats receiving a rodent version of KER-012 (RKER-012) were protected from the thickening of the right ventricular wall
  - In addition, rats receiving RKER-012 were protected from PAH-associated bone loss
- 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
- We expect to initiate a Phase 1 clinical trial in healthy volunteers in H2 2021

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 we expect to initiate a Phase 2 trial in patients with MF in Q3 2021
  - Multiple Phase 2 trials for ALK2 program (KER-047) expected to commence in H2 2021
  - KER-012 is a selective activin receptor ligand trap expected to enter a Phase 1 trial in H2 2021
  - 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 Part 1 of Phase 2 trial in MDS	End of 2021
• Initiate Phase 2 trial in myelofibrosis	Q3 2021 (initial data 2022)

KER-047

• Initiate Phase 2 trial in IDA	H2 2021 (initial data 2022)
• Initiate Phase 2 trial in IRIDA	H2 2021 (initial data 2022)

KER-012

• Present preclinical data on PAH at major conference	2021
• Initiate Phase 1 trial in healthy volunteers	H2 2021 (initial data H1 2022)

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

Thank You