KEROS THERAPEUTICS

KER-050 Update

June 2022

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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 deficiency anemia (Preclinical → Phase 1)	Completed Phase 1 clinical trial	Initiate Phase 2 clinical trial: Q3 2022 Initial data: End of 2022
		Iron-refractory iron deficiency anemia (Preclinical → Phase 1)		Initiate Phase 2 clinical trial: H1 2022 Initial data: End of 2022
Pulmonary **Musculoskeletal**	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
		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

KER-050 is an investigational modified ActRIIA ligand trap designed to inhibit a subset of TGF-β superfamily ligands, including activin A, activin B, GDF8, and GDF11

In preclinical studies, treatment with a mouse research form of KER-050 (RKER-050) has been shown to increase both erythropoiesis and thrombopoiesis:

- Induced red blood cell (RBC) production by promoting multiple stages of erythroid differentiation
- Increased platelet production by increasing megakaryocyte progenitors as well as promoting terminal maturation

27th Annual Congress of the European Hematology Association

KER-050 Presentations:

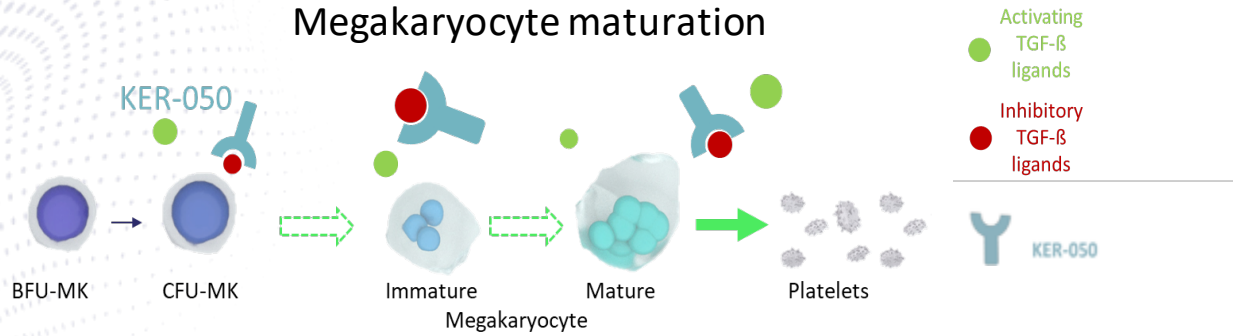
Preclinical

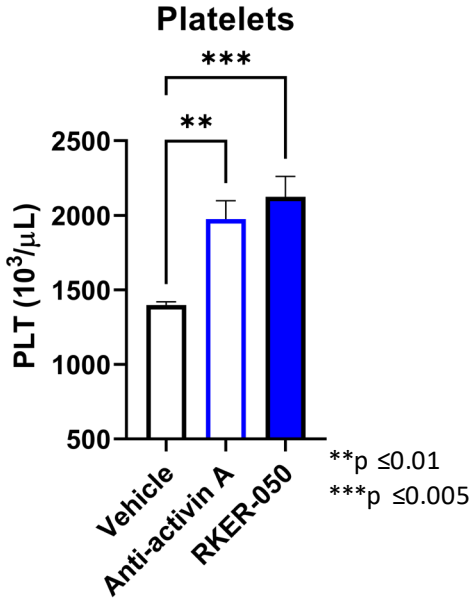
- "RKER-050, a novel inhibitor of TGF-β superfamily signaling, induced platelet production in healthy mouse megakaryocytes"

Clinical

- "A Phase 2, open-label, ascending dose study of KER-050 for the treatment of anemia in patients with very low, low, or intermediate risk myelodysplastic syndromes"

Treatment with RKER-050 Increased Platelet Production in Mice Potentially by Blocking Inhibitory TGF-β Ligands such as Activin A while Permitting BMP Signaling

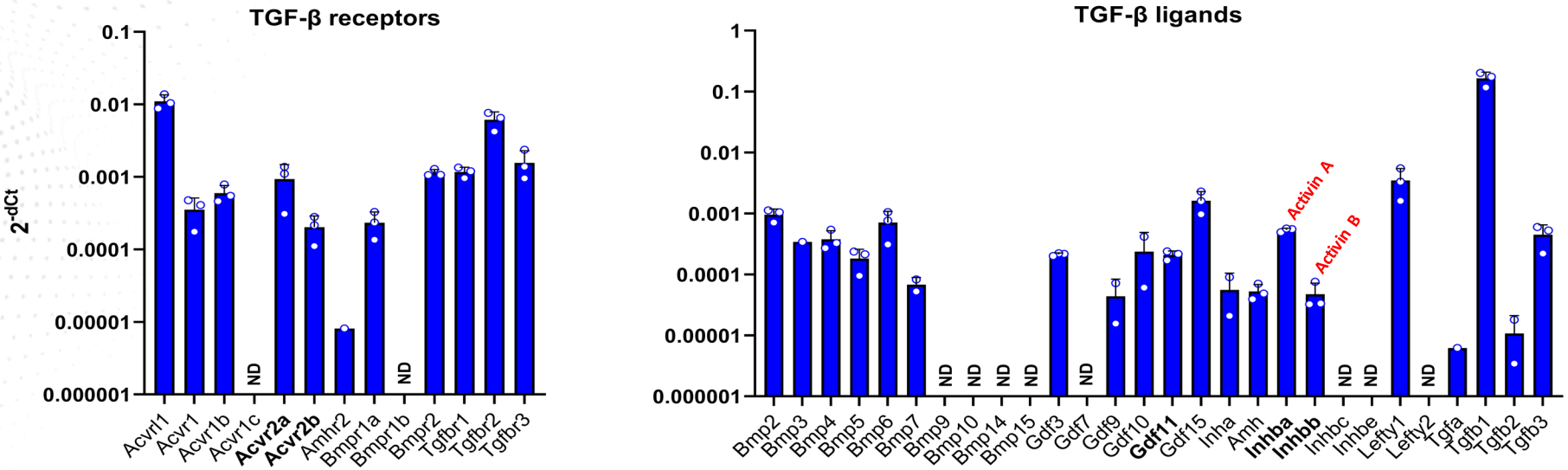




- Inhibitory TGF-β ligands, including activin A, drive SMAD 2/3 quiescence signaling and prevent megakaryocyte maturation
- Activating TGF-β ligands, including the bone morphogenetic proteins (BMPs), increase SMAD 1/5/9 signaling and promote thrombopoiesis

- Both anti-activin A and RKER-050 significantly increased platelet numbers in mice, suggesting:
  - activin A inhibition may be a partial driver for the observed effects of RKER-050 on platelets; and
  - RKER-050 is potentially acting by blocking SMAD 2/3-driven hematopoietic quiescence signaling

Megakaryocyte Precursor Cells Express Activins, GDFs, BMPs and TGF-β Ligands and their Cognate Receptors



- qPCR was performed on RNA from bone marrow-derived megakaryocyte precursor cells isolated from untreated mice to assess TGF-β ligands and receptors gene expression
- Murine bone marrow megakaryocyte precursors express activins, GDFs, BMPs and TGF-β ligands and their cognate receptors, supporting the role of TGF-β superfamily signaling in differentiation of megakaryocytes
  - KER-050 is designed to bind activin A, activin B and GDF11 expressed by megakaryocytes
  - By comparison, luspatercept reportedly binds activin B, GDF8 and GDF11, but does not bind activin A expressed by megakaryocytes (Sako et. al. J Biol Chem. 2010; 285(27): 21037–21048)

KER-050 Overview

- In a Phase 1 clinical trial of KER-050, treatment led to rapid, sustained and dose-dependent increases in RBCs and platelets
  - Inhibition of activin A in the bone resulted in increases in bone specific alkaline phosphatase (BSAP), a marker of osteoblast activity, which is supportive of change in the bone marrow microenvironment
- 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 like myelodysplastic syndromes and myelofibrosis

Myelodysplastic Syndromes (MDS)

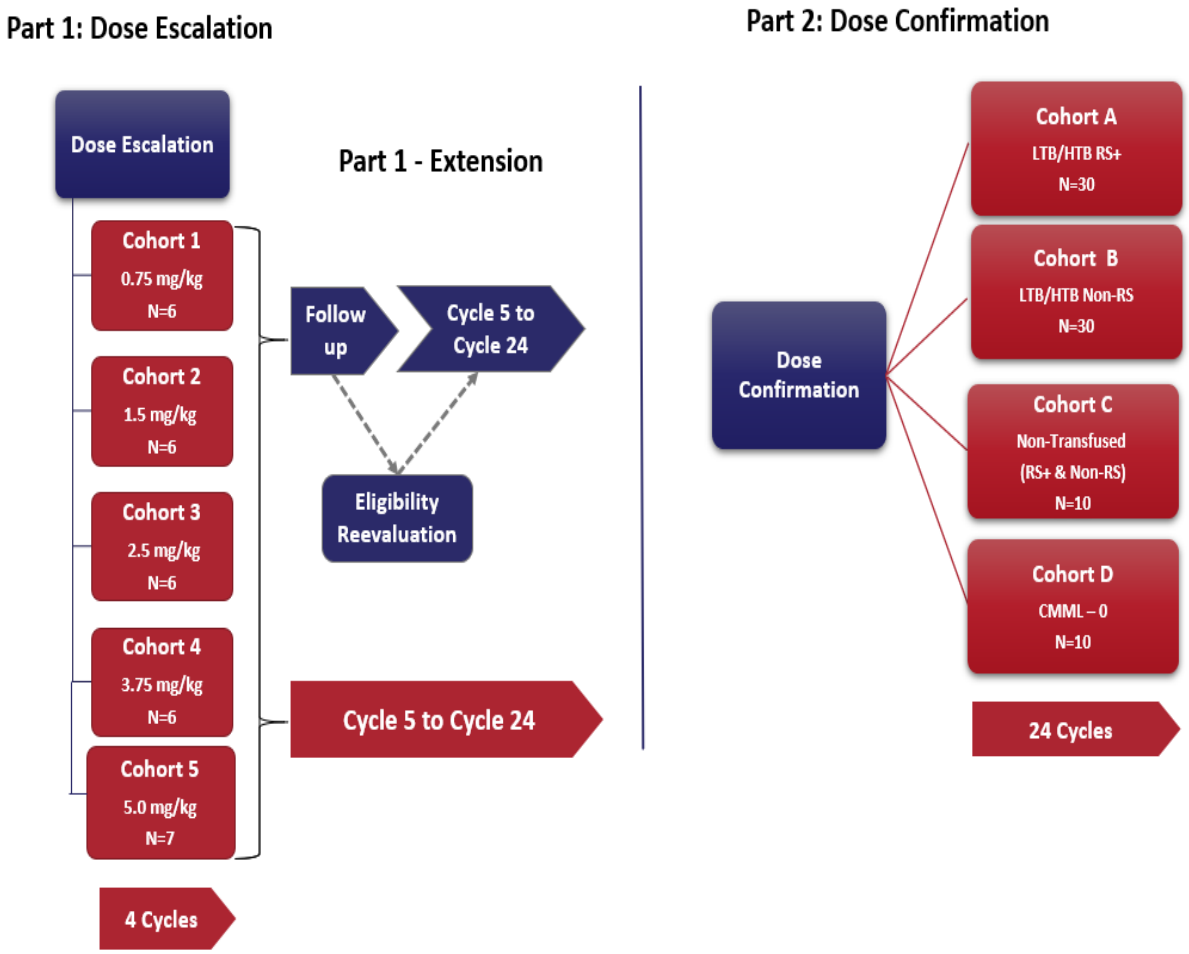
- MDS are a heterogeneous group of myeloid neoplasms characterized by clonal proliferation of hematopoietic stem cells, recurrent genetic abnormalities, myelodysplasia and ineffective hematopoiesis
  - Abnormalities in the bone marrow microenvironment, including altered hematopoietic–stromal interactions, results in ineffective hematopoiesis
  - This results in peripheral blood cytopenias with approximately 90% of lower risk MDS (LR-MDS) patients experiencing anemia
  - Patients have a high risk of evolution to acute myeloid leukemia (AML)
  - Patients are also at high risk of morbidity and mortality, including increased risk of infection, hemorrhage, iron overload due to transfusions and cardiovascular disease
- Treatment options for LR-MDS patients include erythroid stimulating agents (ESA), erythroid maturation agent (EMA), RBC transfusions and iron chelation therapy
  - However, the benefit of RBC agents is limited in certain patients including patients with high transfusion burden

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)
- The data included in this presentation represent available data from a data cut-off date of April 3, 2022



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 for Hgb ≤9 g/dL
  - High transfusion burden (HTB): ≥4 units of RBC/8 weeks for Hgb ≤9 g/dL

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

Demographics and Baseline Characteristics from Part 1 Dose Escalation

	KER-050 Dose Level (mg/kg)					
	0.75 (N=6)	1.5 (N=6)	2.5 (N=6)	3.75 (N=6)	5 (N=7)	All (N=31)
Age, Mean (range)	75.5	68.3	72	73.3	76.7	73.3 (55-88)
Female, n (%)	5	1	4	2	0	12 (38.7%)
RS positive	3	3	3	4	4	17 (54.8%)
Transfusion Burden						
Non-Transfused (NT), 0 units	3	0	1	1	0	5 (16.1%)
Low Transfusion Burden (LTB), <4 units	2	0	1	2	3	8 (25.8%)
High Transfusion Burden (HTB), ≥4 units	1	6	4	3	4	18 (58.1%)
WHO MDS Classification, n (%)						
MDS-MLD	3	3	3	1	3	13 (41.9%)
MDS-RS-MLD	2	2	3	4	3	14 (45.2%)
MDS-RS-SLD	0	1	0	0	0	1 (3.2%)
MDS with isolated del(5q)	1	0	0	0	0	1 (3.2%)
N/A	0	0	0	1	1	2 (6.5%)
Prior ESA Therapy, n (%)	0	0	2	1	0	3 (9.7%)
Iron chelator, n (%)	0	2	2	2	1	7 (22.6%)
Efficacy Evaluable Patients*, n(%)	6	4	6	6	5	27 (87.1%)
Efficacy Evaluable HTB Patients, n (%)**	1	4	4	3	4	16/18 (88.9%)

*Patients with at least 8 weeks of post-treatment hemoglobin and transfusion assessments were defined as efficacy evaluable.
** Percentage was based on all HTB patients.

KER-050 was Generally Well-Tolerated at all Doses Tested in Part 1

n (%)	KER-050 Dose Level (mg/kg)					
	0.75 (N=6)	1.5 (N=6)	2.5 (N=6)	3.75 (N=6)	5 (N=7)	All (N=31)
TEAE	6 (100)	6 (100)	6 (100)	5 (83.3)	6 (85.7)	29 (93.5)
Related TEAE	1 (16.7)	0	1 (16.7)	1 (16.7)	2 (28.6)	5 (16.1)
Grade ≥3	1 (16.7)	1 (16.7)	3 (50.0)	2 (33.3)	5 (71.4)	12 (38.7)
SAE	1 (16.7)	2 (33.3)	1 (16.7)	2 (33.3)	4 (57.1)	10 (32.3)
TEAE requiring dose modification	0	0	0	1 (16.7)	1 (14.3)	2 (6.5)
Death	0	1 (16.7)	0	0	0	1 (3.2)

- No drug related serious adverse events or dose-limiting toxicities were reported
- 10 patients experienced treatment-emergent SAEs
- 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 progressed to AML

April 3, 2022 data cutoff

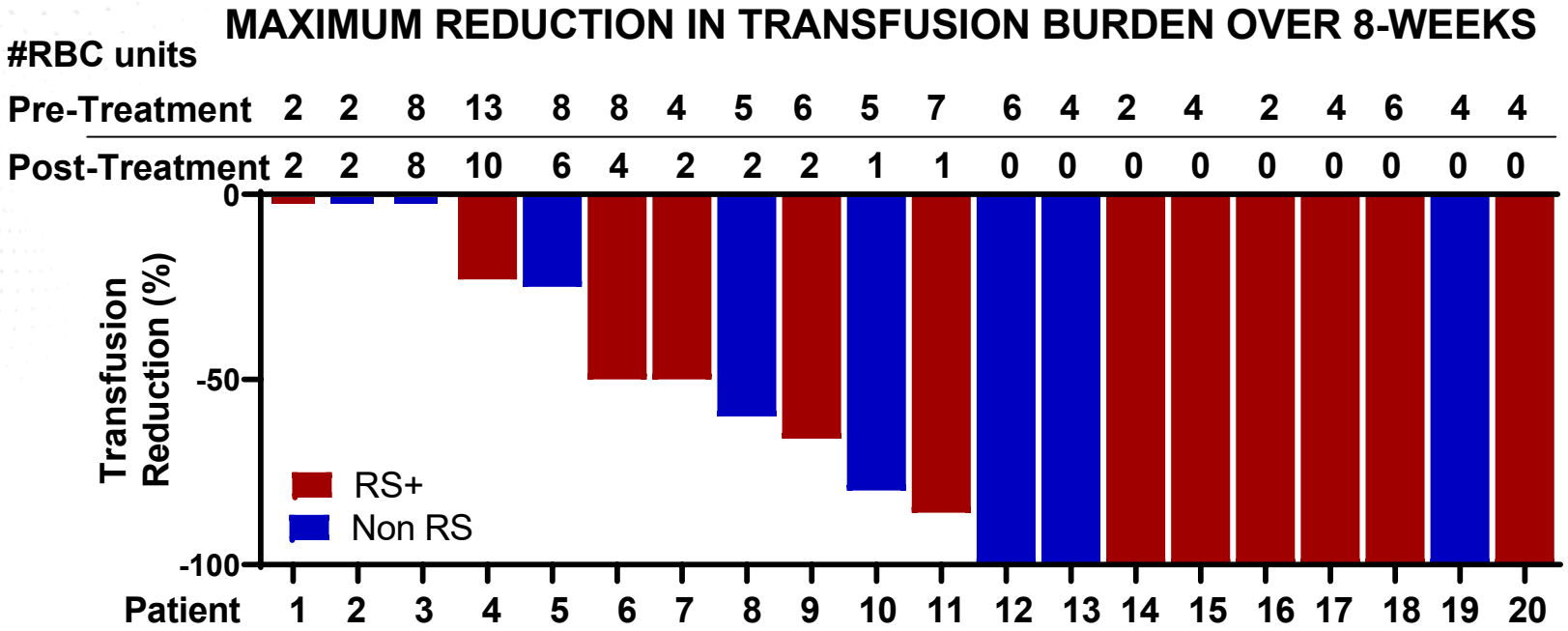
Efficacy Summary of 8-Week Endpoints Achieved in MDS Patients

Response Summary	Response Rate, n/m (%)	
	All evaluable patients	HTB evaluable patients
Overall Erythroid Response (HI-E or TI)	14/27 (51.8%)	11/16 (68.8%)
IWG 2006 HI-E	12/26 (46.2%)	11/16 (68.8%)
Transfusion independence (TI*) **RS+** **Non-RS**	9/20 (45%) 6/12 (50%) 3/8 (37.5%)	7/16 (43.8%) 4/9 (44.4%) 3/7 (42.9%)

*Baseline Transfusion Requirement ≥2 RBC units

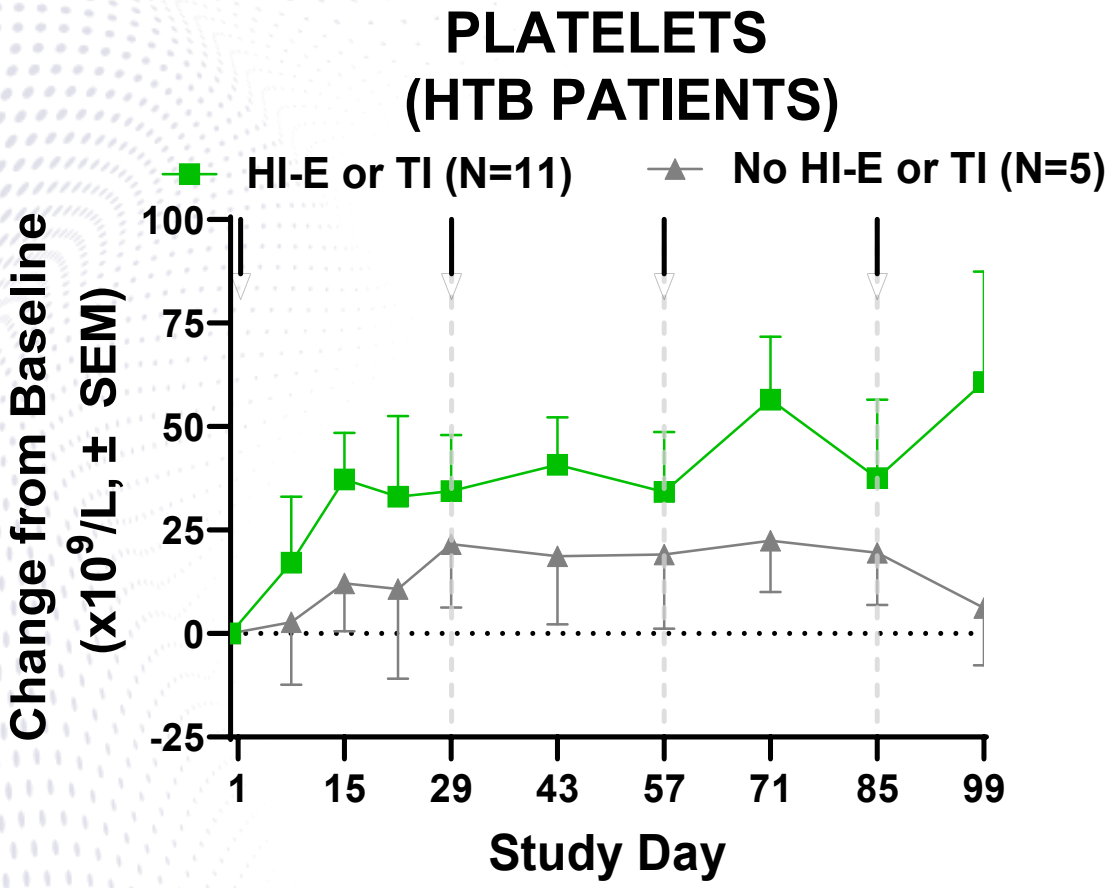
n = responders in each category; m = 8-week evaluable population as of data cutoff date

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

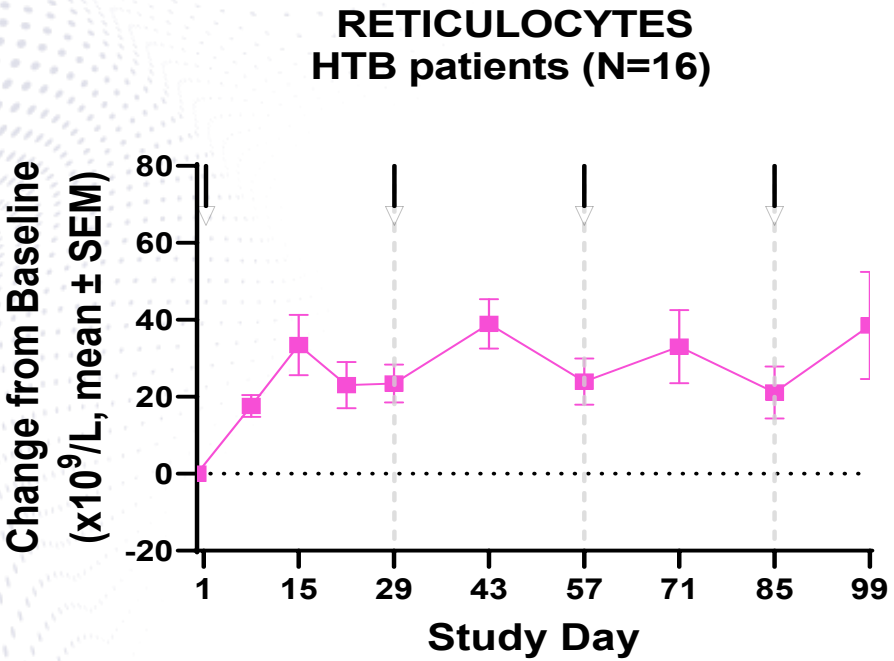
Sustained Increase in Platelets Observed in HTB Patients Achieving HI-E or TI with KER-050 Treatment

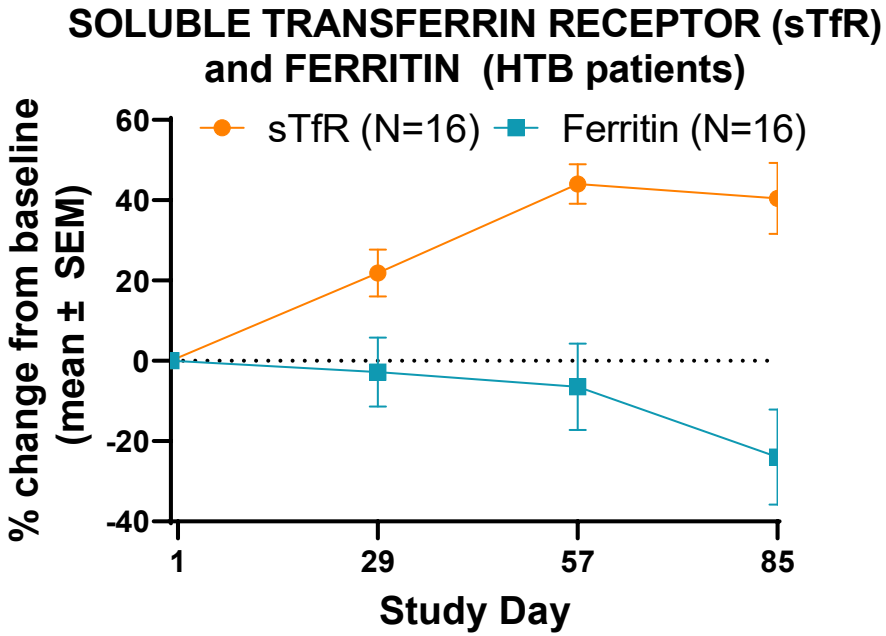


KER-050 upregulated thrombopoiesis

- 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

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 1359.2 ng/mL
- Mean maximum reduction in ferritin was 29.1% following 3 months of treatment with KER-050

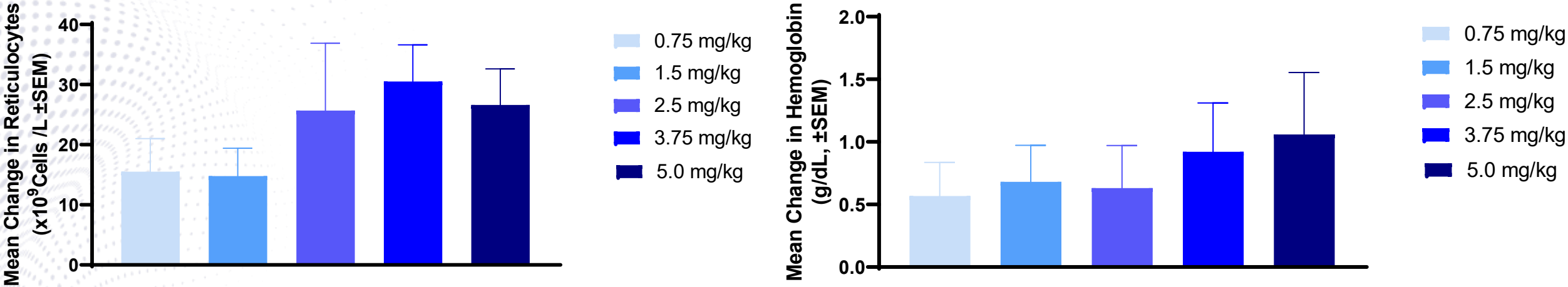
April 3, 2022 data cutoff

Summary

- 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

MD-201 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
- Safety Review Committee recommended initiation of Part 2 dose confirmation of the Phase 2 clinical trial
  - 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

Anticipated Key Milestones*

KER-050

- Announce additional data from Phase 2 trial in MDS — End 2022
- Announce initial data from Phase 2 trial in myelofibrosis — End 2022

KER-047

- Initiate Phase 2 trial in IRIDA — H1 2022 (initial data end of 2022)
- Initiate Phase 2 trial in IDA — Q3 2022 (initial data end of 2022)

KER-012

- Announce additional data from Part 2 of Phase 1 trial — H2 2022

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

Thank You