KEROS THERAPEUTICS

Corporate Update

May 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 of KER-012 to treat diseases such as pulmonary arterial hypertension without a dose-limiting red blood cell effect; 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 Quarterly Report on Form 10-Q, filed with the SEC on May 5, 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.

Introductions

- Jasbir Seehra, President and Chief Executive Officer
- Keith Regnante, Chief Financial Officer
- Simon Cooper, Chief Medical Officer
- Jenn Lachey, Chief Scientific Officer
- Christopher Rovaldi, Chief Operating Officer

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 (ActRIIB) 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

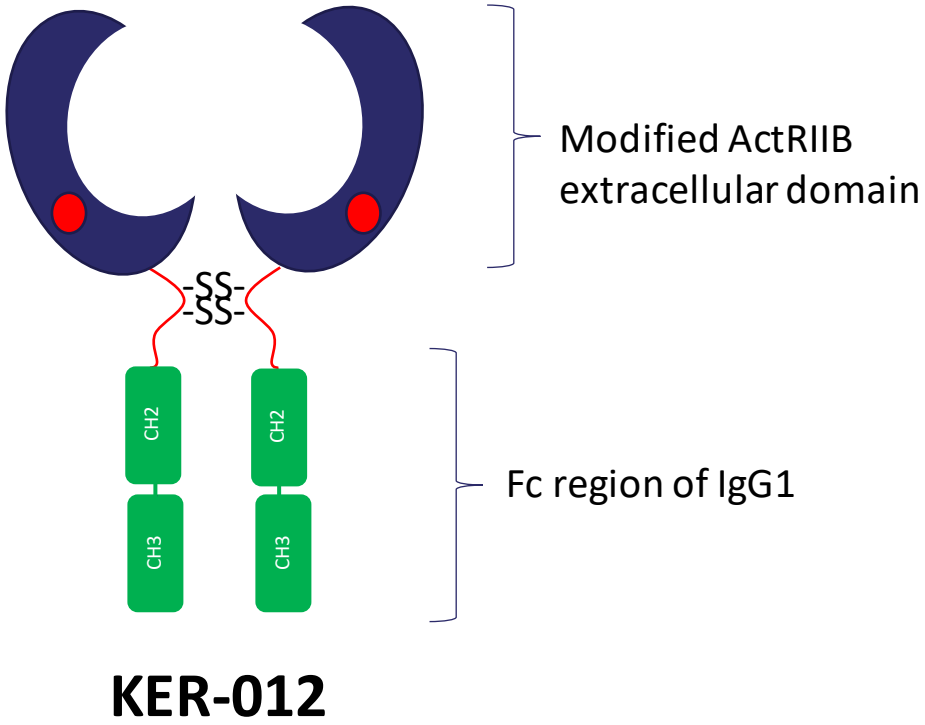
Program	Asset	Phase of Development	Status	Next Milestones*
Hematology	KER-050 (therapeutic protein)	Myelodysplastic syndromes	Phase 2 clinical trial ongoing	Additional data from the Phase 2 clinical trial: mid-2022
		Myelofibrosis	Phase 2 clinical trial ongoing	Initial data: End of 2022
	KER-047 (small 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
Pulmonary	KER-012 (therapeutic protein)	Pulmonary arterial hypertension	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 Pipeline		Musculoskeletal and hematology		

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

KER-012

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
- Phase 1 clinical trial in healthy postmenopausal volunteers is ongoing



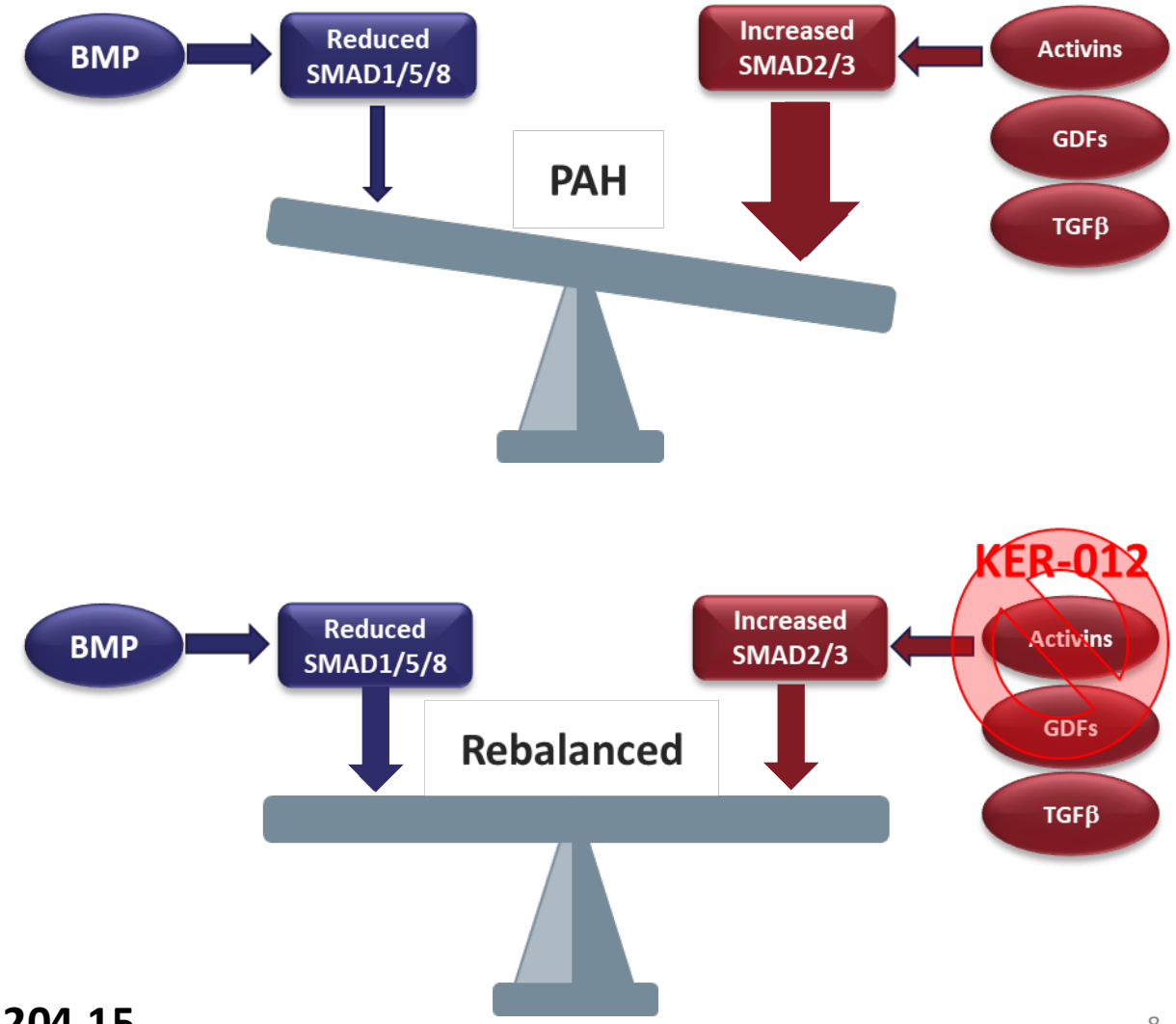
KER-012

Role of TGF-β in Pulmonary Arterial Hypertension

Pulmonary arterial hypertension (PAH) is a debilitating disorder characterized by elevated pulmonary vascular resistance, resulting in diminished oxygenation, impaired cardiac output, and right ventricle (RV) overload

PAH is associated with imbalanced TGF-ß superfamily signaling, including insufficient SMAD 1/5/8 signaling*

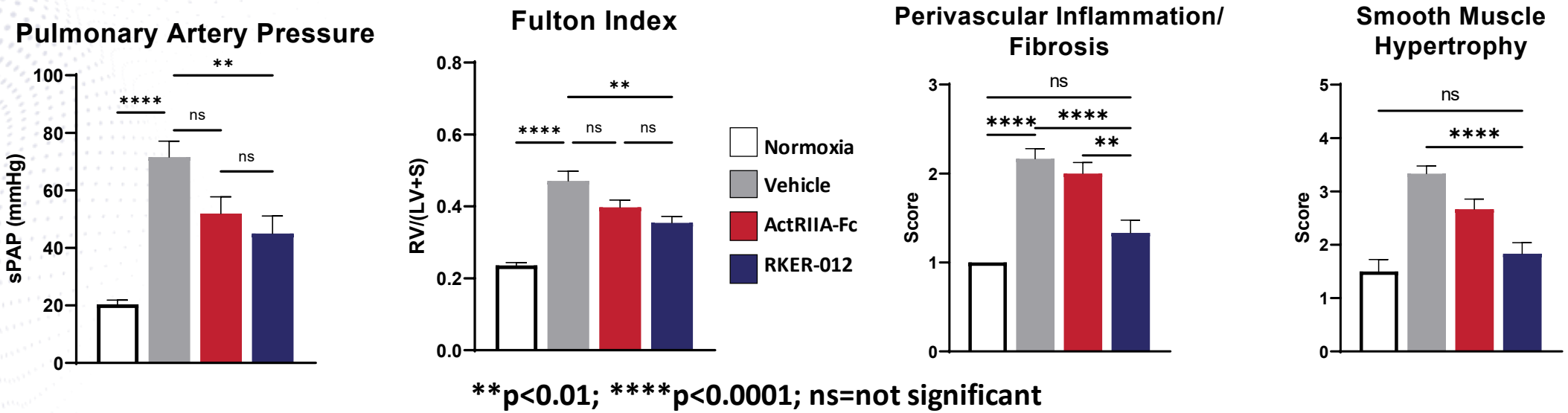
A third-party Phase 2 clinical trial demonstrated that rebalancing SMAD signaling by inhibiting ligands that bind ActRIIA provided benefit but was accompanied by a potentially dose-limiting increase in red blood cells (RBCs)*



*Data from: Humbert, M. et. al., N Engl J Med 2021;384:1204-15

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:

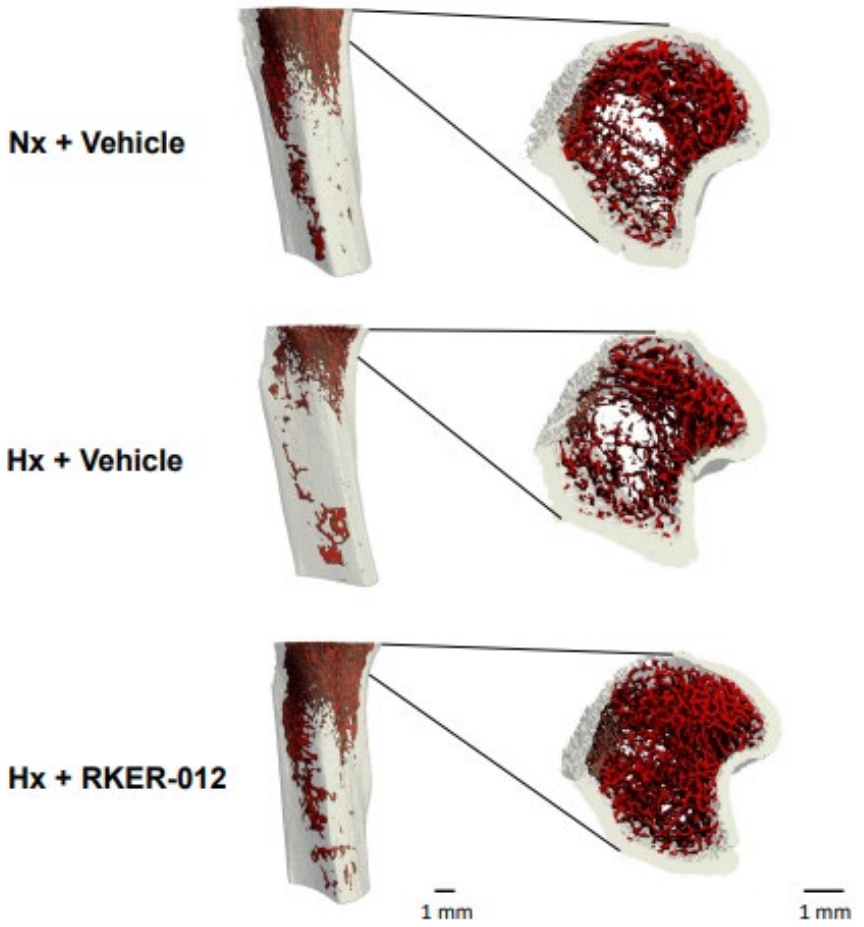


**p<0.01; ****p<0.0001; ns=not significant

- 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.

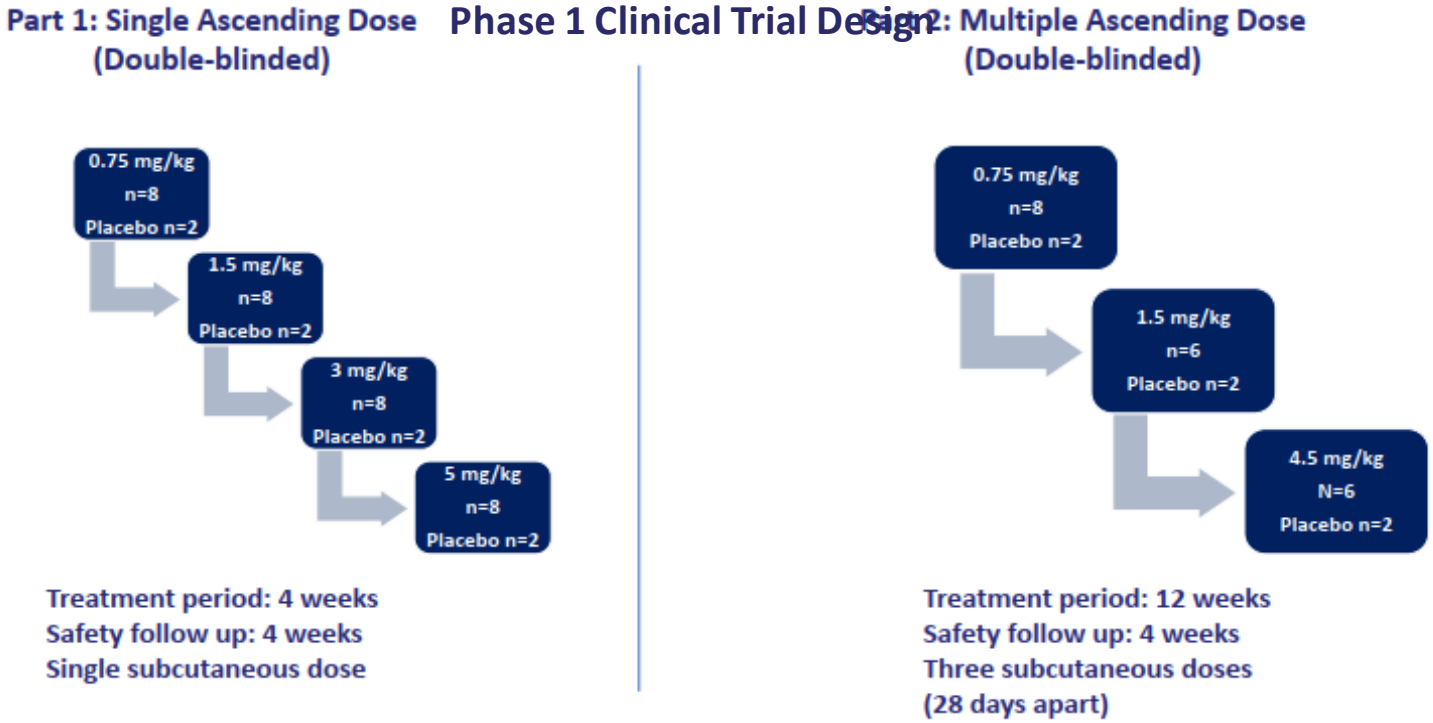
KEROS THERAPEUTICS

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

Demographics and Disposition (Part 1 SAD)

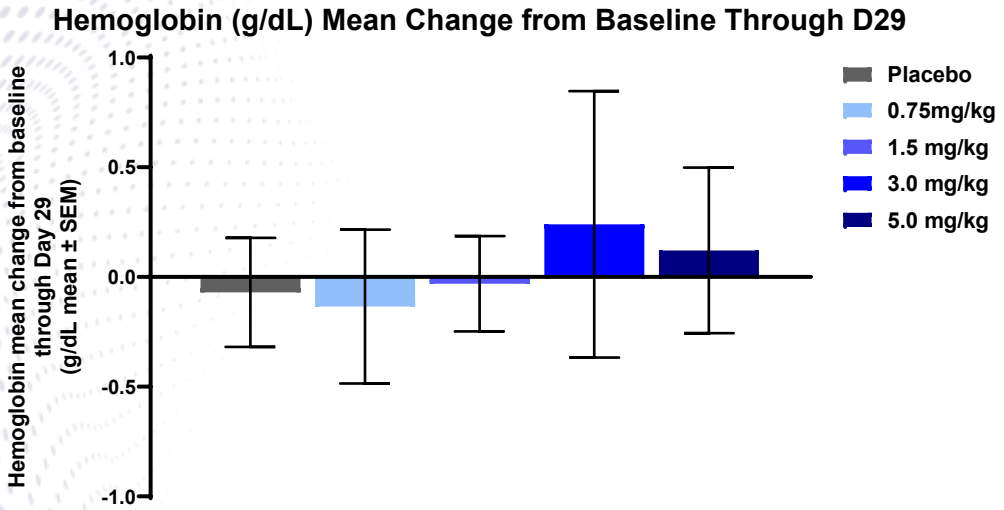
	PBO (N=8)	0.75 mg/kg (N=8)	1.5 mg/kg (N=8)	3.0 mg/kg (N=8)	5.0 mg/kg (N=8)	All Subjects (N=40)
Age, years mean (range)	56.0 (48 – 60)	58.3 (52 -70)	54.9 (50 - 59)	57.8 (50 - 66)	59.3 (53 - 68)	57.2 (48 - 70)
Race, n (%)						
White	8 (100)	8 (100)	8 (100)	7 (87.5)	8 (100)	39 (97.5)
Multiple*	0	0	0	1 (12.5)	0	1 (2.5)
Weight, kg mean (SD)	68.4 (10.09)	71.6 (9.60)	67.5 (8.05)	68.1 (9.49)	67.1 (10.35)	68.6 (9.19)
FSH, IU/L mean (SD) [range]						
at Screening	88.9 (16.34) [62, 107]	75.5 (19.87) [56, 112]	95.0 (22.93) [64, 133]	77.9 (26.31) [60, 127]	91.0 (35.02) [45, 146]	85.6 (25.02) [45, 146]
at C1D1	70.4 (28.91) [18, 105]	53.3 (28.16) [26, 103]	86.5 (16.64) [64, 109]	49.5 (23.65) [21, 92]	87.1 (35.49) [63, 162]	68.9 (30.18) [18, 162]
%chg from SCRN	-16.9 (35.65) [-83.2, 11.9]	-31.9 (23.02) [-58.3, 1.1]	-7.7 (8.78) [-18.3, 7.1]	-33.3 (24.57) [-83.5, 2.6]	4.4 (17.71) [-17.0, 40.0]	-17.7 (26.38) [-83.5, 40.0]
Disposition						
Completed Study, n (%)	8 (100%)	8 (100%)	7 (87.5%)	8 (100%)	8 (100%)	39 (97.5)
Discontinuation, n (%)	0	0	1** (12.5%)	0	0	1** (2.5)

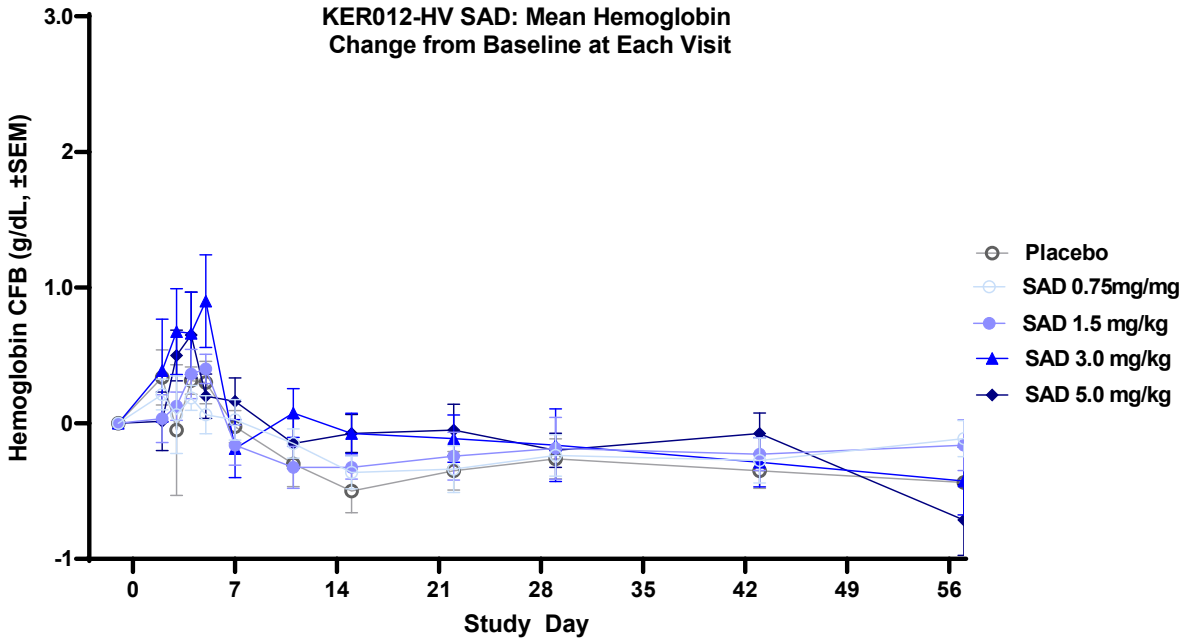
* More than one race was reported.
** 1 subject prematurely discontinued after receiving KER-012 due to withdrawal of consent.

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

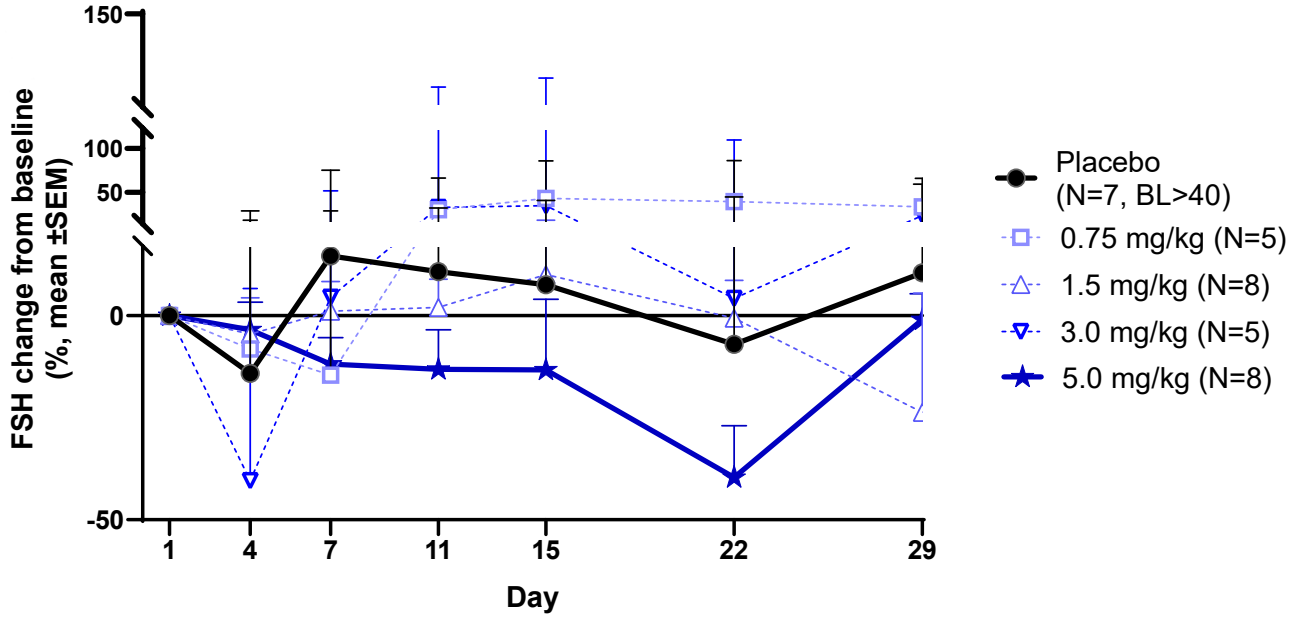
No Clinically Meaningful Change in Hgb Observed with KER-012 Administration of up to 5 mg/kg





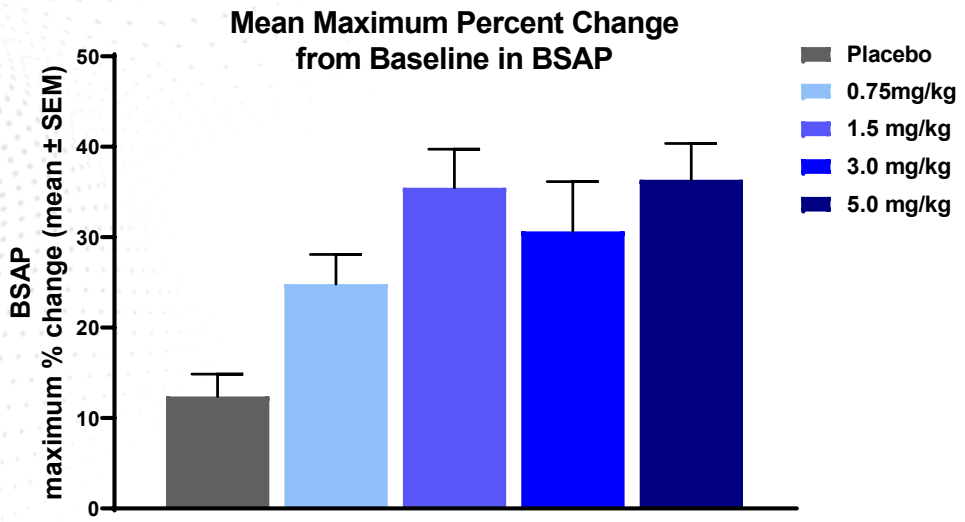
- Single dose of KER-012 was not associated with clinically meaningful changes in Hgb at all doses in Part 1 of this trial

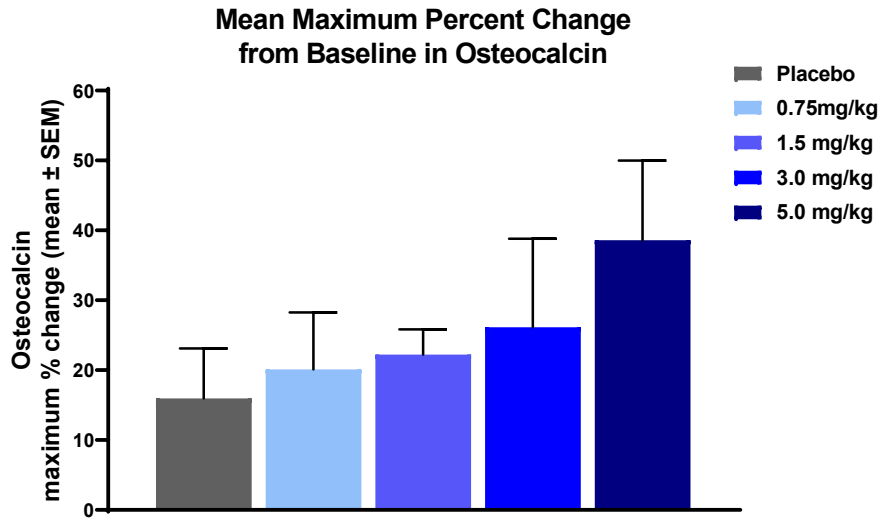
KER-012 Administration Resulted in 40% Mean Decrease in FSH at 5 mg/kg Dose



- 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 40% mean decrease in FSH on Day 22

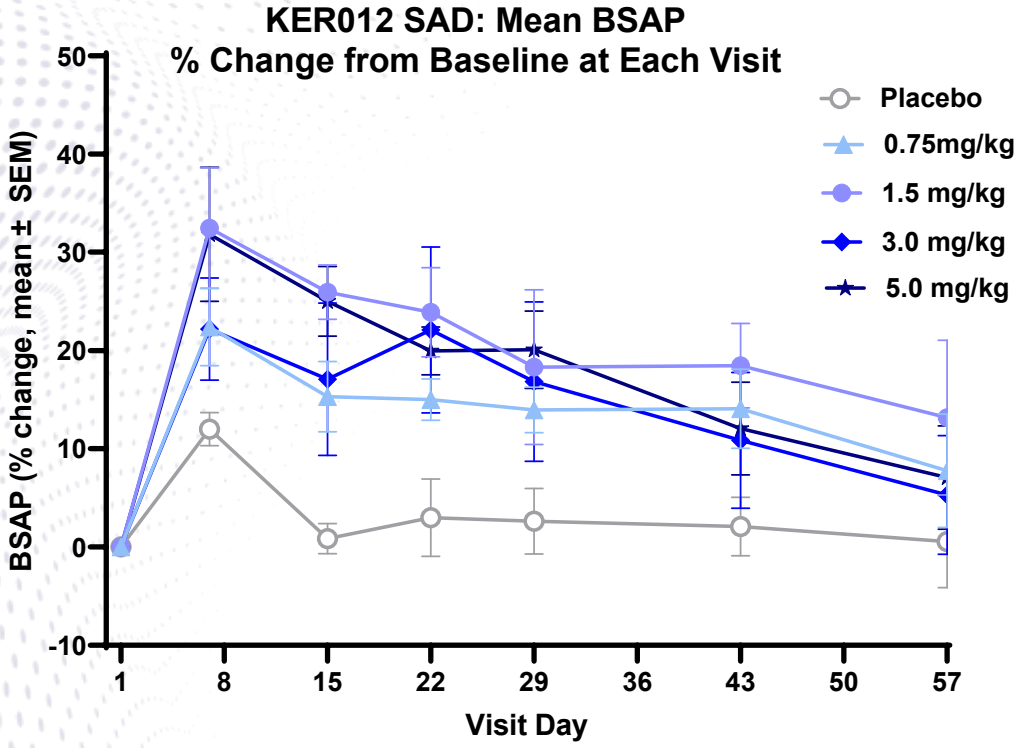
Robust Increase in Markers of Bone Formation Observed

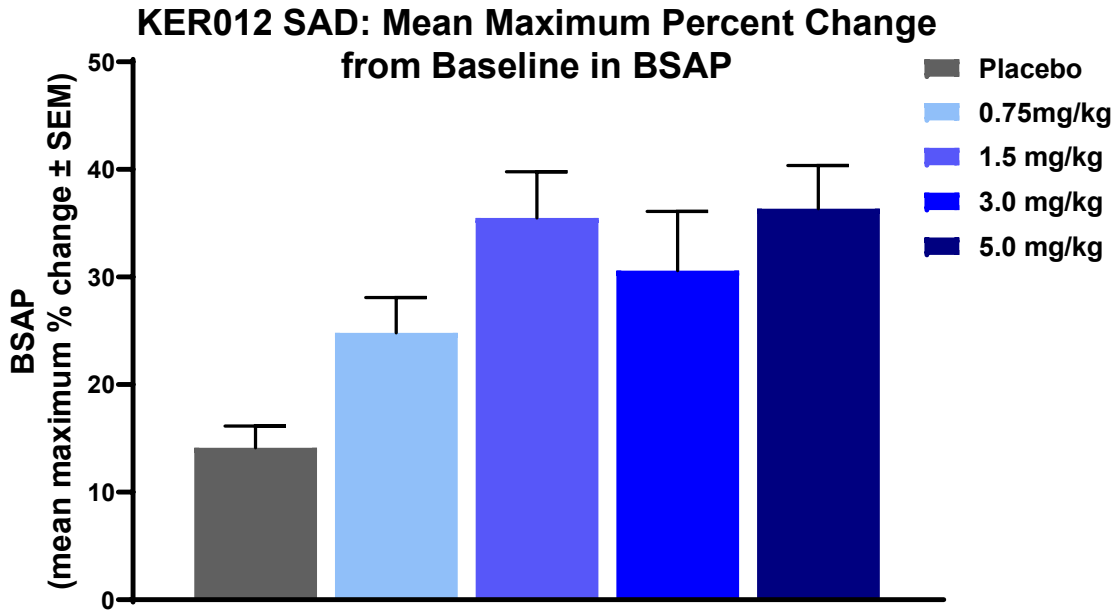




- KER-012 is designed to inhibit activins and GDFs in the bone, which we believe potentially results in reduced SMAD 2/3 signaling and increased signaling of bone morphogenetic protein (BMP) pathway (SMAD1/5/8)
  - The increased BMP signaling potentially promotes bone formation through activation/recruitment 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



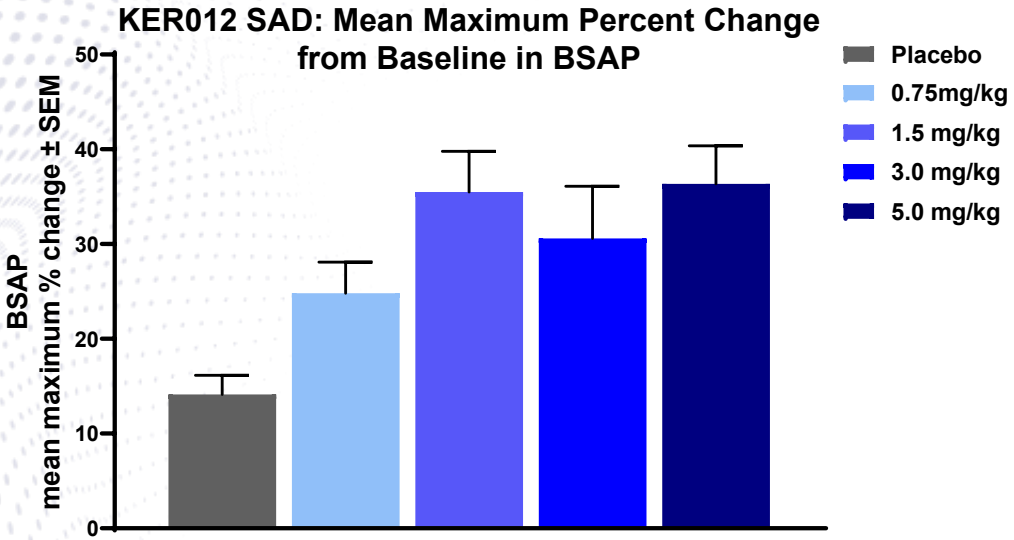


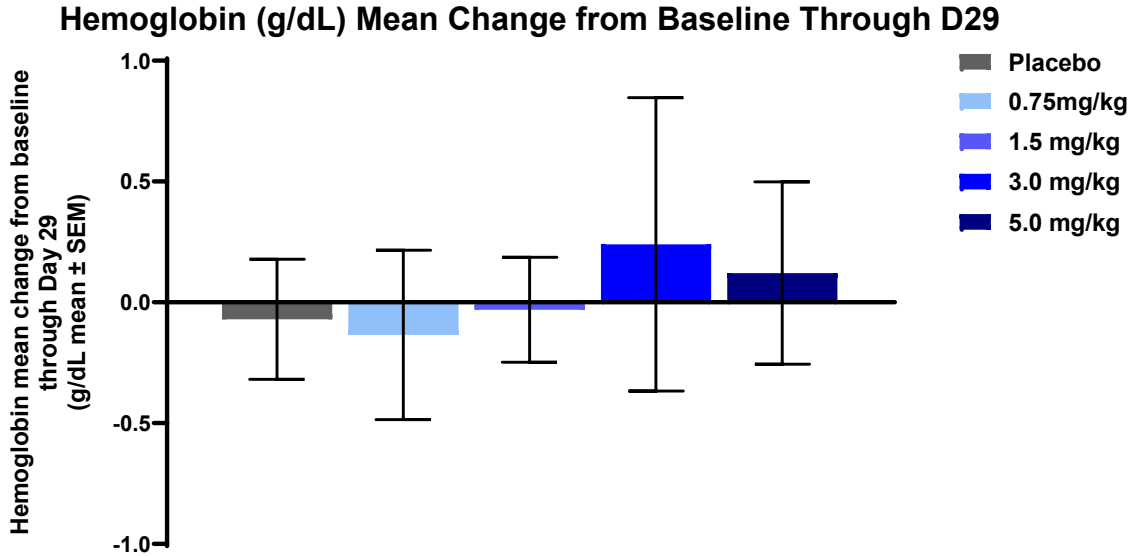
- A single 0.75 mg/kg dose of KER-012 elicited a 25% mean maximum increase in BSAP, which is supportive of osteoblast activation/recruitment in bone
- A 35% mean maximum increase in BSAP was observed following a single 1.5 mg/kg dose of KER-012

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 (40% 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 35% 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

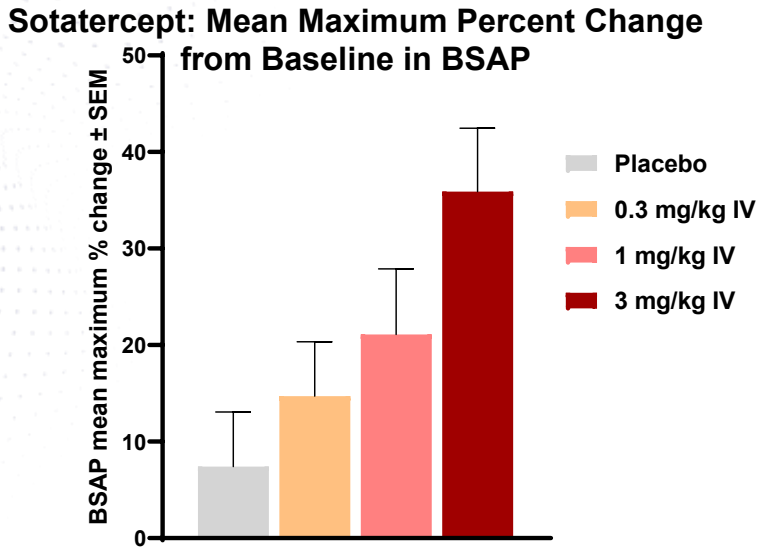
KER-012 Elicited Maximum Increases in Bone-Specific Alkaline Phosphatase (BSAP)





- Increasing doses of KER-012 was observed to elicit maximal target engagement
  - Observed FSH decrease of up to 40%
  - Observed increases in BSAP, P1NP and osteocalcin
- Mean maximal increases in BSAP were observed at the higher dose cohorts (1.5, 3 and 5 mg/kg) with a single dose of KER-012
- No clinically meaningful changes in hemoglobin were observed following single doses of KER-012 ranging from 0.75 to 5.0 mg/kg

Sotatercept Increased BSAP Concurrently with Observed Increases in Hemoglobin in a Third-Party Phase 1 Clinical Trial*



Dose (iv) (mg/kg)	Placebo	0.3	1.0	3.0
Max change in Hemoglobin (g/dL)	0.4	1.7	1.7	2.4
Standard Deviation	0.3	0.6	0.6	0.7

- Results from a third-party single ascending dose Phase 1 clinical trial of sotatercept in healthy postmenopausal women was previously reported*
- Dose dependent increases in BSAP were observed with sotatercept*
- Sotatercept elicited mean maximal target engagement in BSAP at 3.0 mg/kg (i.v.)*
- Treatment with a single dose of sotatercept resulted in sustained increase in hemoglobin*

***All data on this slide from: Ruckle, J. et. al., JMBR 2009;24:744-752**

KER-012: Next Steps

- Part 2 of this trial (multiple ascending dose) is ongoing; expected to report data in H2 2022
  - Expect to confirm SAD biomarkers and include changes in bone mineral density by dual-energy x-ray absorptiometry
- Keros expects to initiate a Phase 2 clinical trial of KER-012 in PAH patients following the completion of the Phase 1 clinical trial
  - Keros expects to announce the design of this Phase 2 clinical trial in early 2023

Anticipated Key Milestones*

KER-050

• Announce additional data from Phase 2 trial in MDS	Mid-2022 (EHA 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 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.

Q&A