KEROS THERAPEUTICS

Corporate Presentation

November 2026



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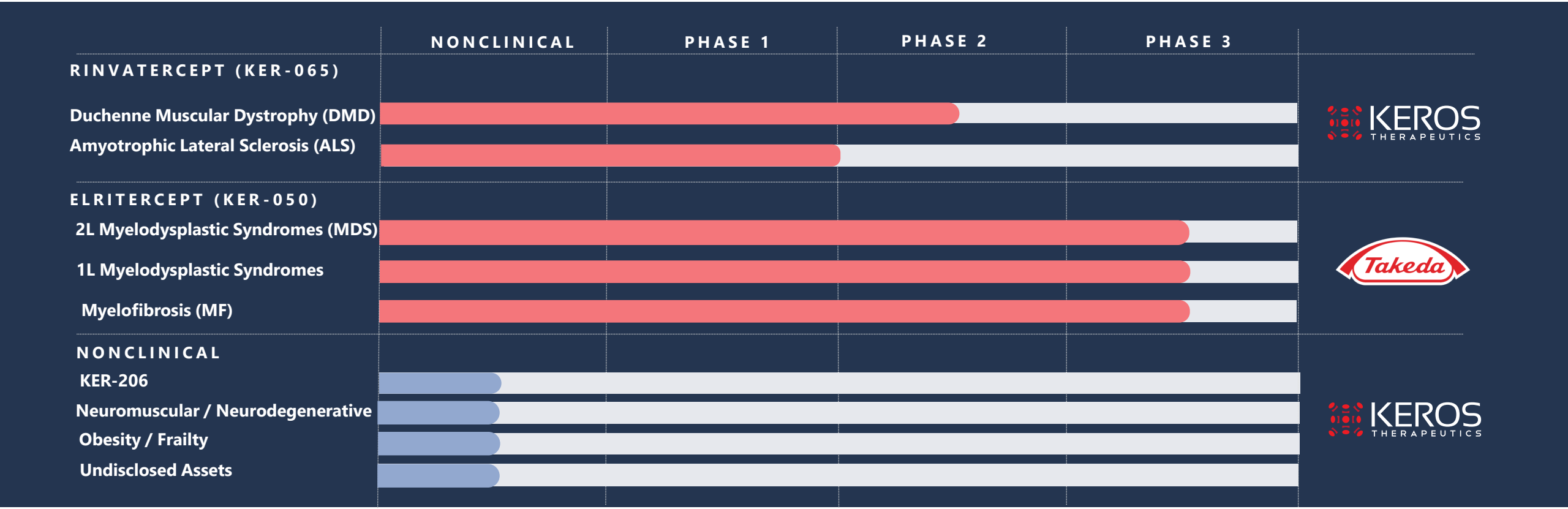
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Focused on Transforming the Lives of a Wide Range of Patients with Disorders Linked to Dysfunctional TGF-β Superfamily Signaling

Keros is a clinical-stage biopharmaceutical company
Developing potentially differentiated product candidates designed to alter transforming growth factor-beta (TGF-β) signaling and target pathways critical for the growth, repair and maintenance of a number of tissue and organ systems

We believe our product candidates have the potential to unlock the full therapeutic benefits of modulating the TGF-β superfamily and provide disease-modifying benefit to patients



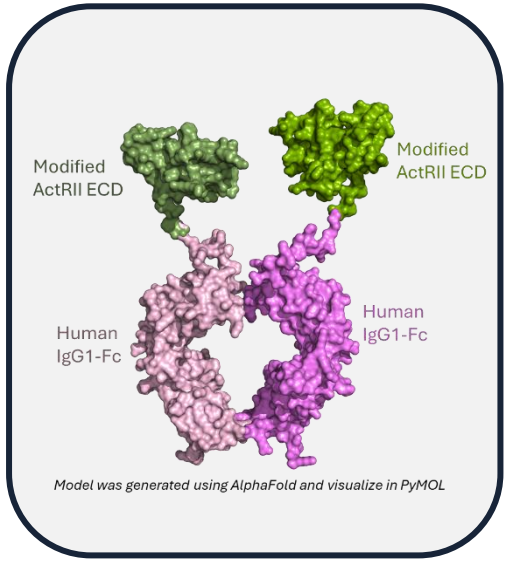


Rinvatercept (KER-065)

Rinvatercept (KER-065)

Rinvatercept is designed to bind and inhibit TGF-ß ligands, including activin A and myostatin, which are negative regulators of muscle and bone mass and strength, in order to:

- ✓ Improve skeletal muscle regeneration, increase muscle size and strength
- ✓ Inhibit and reduce fibrosis
- ✓ Inhibit inflammation
- ✓ Reduce fat accumulation
- ✓ Improve bone health through bone anabolic mechanisms

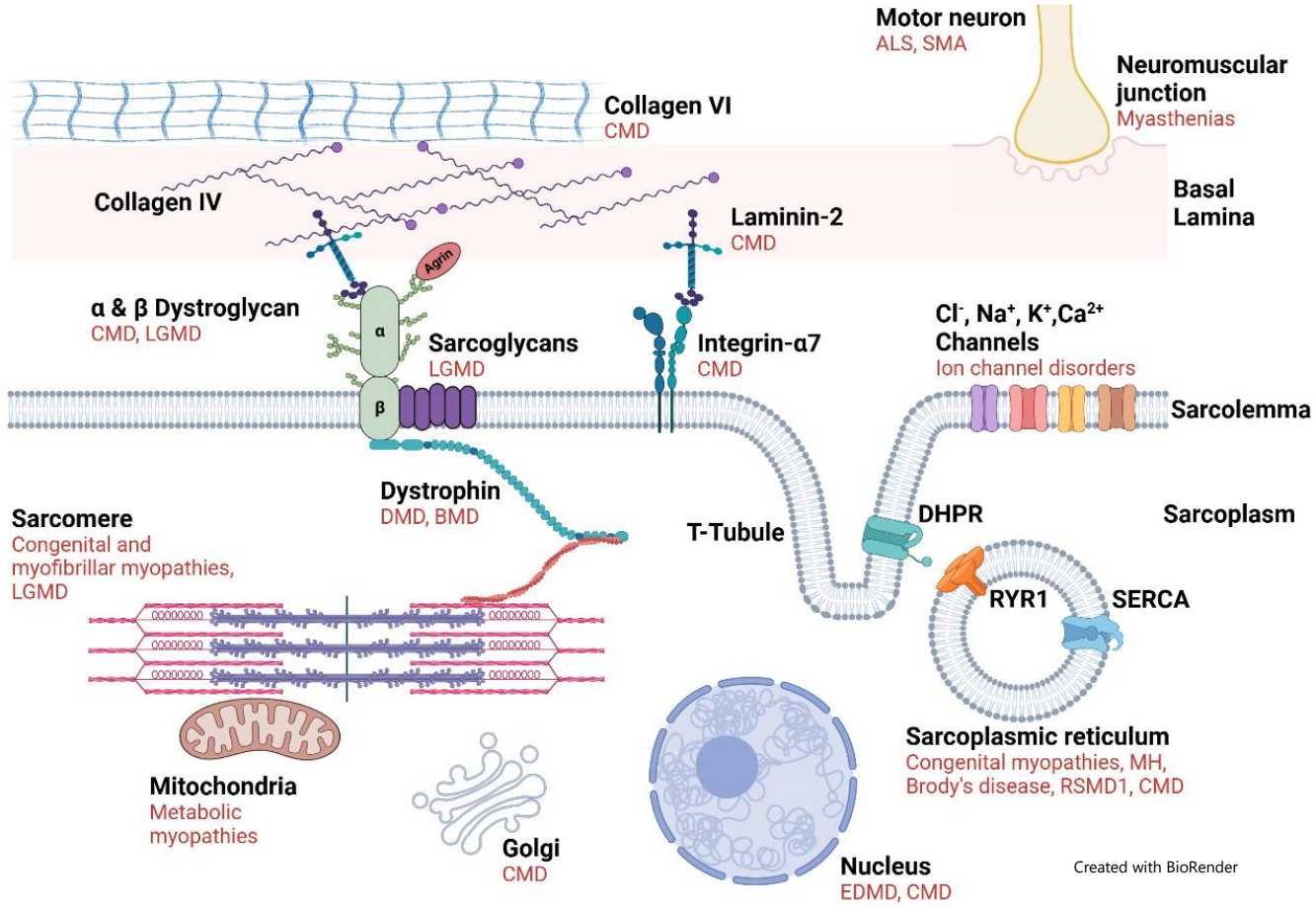


Rinvatercept is an investigational modified activin receptor type II ligand trap

Rinvatercept is designed to have reduced binding for bone morphogenic protein 9 (BMP9) to avoid the vascular/bleeding events observed with ActRIIb-Fc derived from the native sequences

Rinvatercept: Potential to Treat a Broad Range of Neuromuscular Diseases

- **Neuromuscular disorders arise from:**
  - Mutations in genes coding for structural proteins that are unable to connect the contractile apparatus to the basal lamina
    - Examples: DMD, BMD, LGMD and CMD
  - Failure of transmission of the signal from motor neuron to the muscle
    - Examples: ALS, SMA and myasthenia gravis
- **Regardless of the underlying cause, the pathology in the skeletal muscle is similar:**
  - Mutations in the structural protein gene lead to weaker muscle that is easily damaged, resulting in inflammation, inhibition of muscle regeneration, replacement of muscle with fat and fibrosis
  - Inability of the motor neuron to stimulate muscle leads to muscle wasting and replacement with fat and fibrosis



Based on observed pharmacology in nonclinical studies and the Phase 1 clinical trial, we believe rinvatercept has potential in multiple, rare neuromuscular diseases with high unmet need

BMD = Becker muscular dystrophy; LGMD = limb-girdle muscular dystrophy; CMD = congenital muscular dystrophy; ALS = amyotrophic lateral sclerosis; SMA = spinal muscular atrophy



Rinvatercept:
Duchenne Muscular Dystrophy (DMD)

Duchenne Muscular Dystrophy (DMD)

- **DMD is a rare, severe and ultimately fatal genetic disease characterized by the absence of dystrophin protein, leading to progressive muscle degeneration and weakness**
- The National Organization for Rare Disorders estimates that approximately **one in every 3,500 male births is affected by DMD worldwide**
- In DMD, **muscle undergoes continuous rounds of degeneration/regeneration, but eventually the ability of the muscle to regenerate declines due to a decline in muscle progenitor cells known as satellite cells, resulting in fibrosis and secondary fatty infiltration[1-3]**
- **Currently, there is no cure for DMD**

DMD Disease Progression[4,5]

5 to 7 Years	*Motor delay, enlarged calves, toe walking, standing from supine and climbing stairs more difficult*	Early Ambulatory
8 to 11 Years	*Increasing loss of walking mobility and part time wheelchair use*	Late Ambulatory
Early Teens	*Loss of ambulation, fulltime wheelchair use and increasing loss of upper limb function*	Early Non-Ambulatory
Teens	*Increasing respiratory impairment, ventilatory support often required and unable to perform activities of daily living*	Late Non-Ambulatory
Teens to Twenties	*Increasing cardiac dysfunction, heart failure and ultimately death*	

1. Tabebordbar, M., et al. (2013). Annu. Rev. Pathol. 8, 441–475. doi: 10.1146/annurev-pathol-011811-132450; 2.Wallace, G. Q., and McNally, E. M. (2009). Annu. Rev. Physiol. 71, 37–57. doi:10.1146/annurev.physiol.010908.1632164.4; 3. Mann, C. J.,et al. (2011). Skelet. Muscle 1:21. doi: 10.1186/2044-5040-1-21; 4. Bushby K, Connor E. Clin Investig (Lond) 2011; 1:1217-1235; 5. Cruz Guzman, et al. Int J Endocrinol 2012; 2012:485376

Current Treatment Landscape for DMD

Glucocorticoids

- Help to maintain muscle function in DMD patients
- Long-term treatment can have significant negative side effects, including bone loss, fluid retention, hyperglycemia, severe weight gain with fat deposits in the abdomen, face and neck

Exon Skipping

- Four therapies approved by the FDA, each addressing a specific exon skipping mutation
- Approved using the accelerated approval pathway on the basis of dystrophin production
- Require weekly intravenous (IV) infusions

Gene Therapy

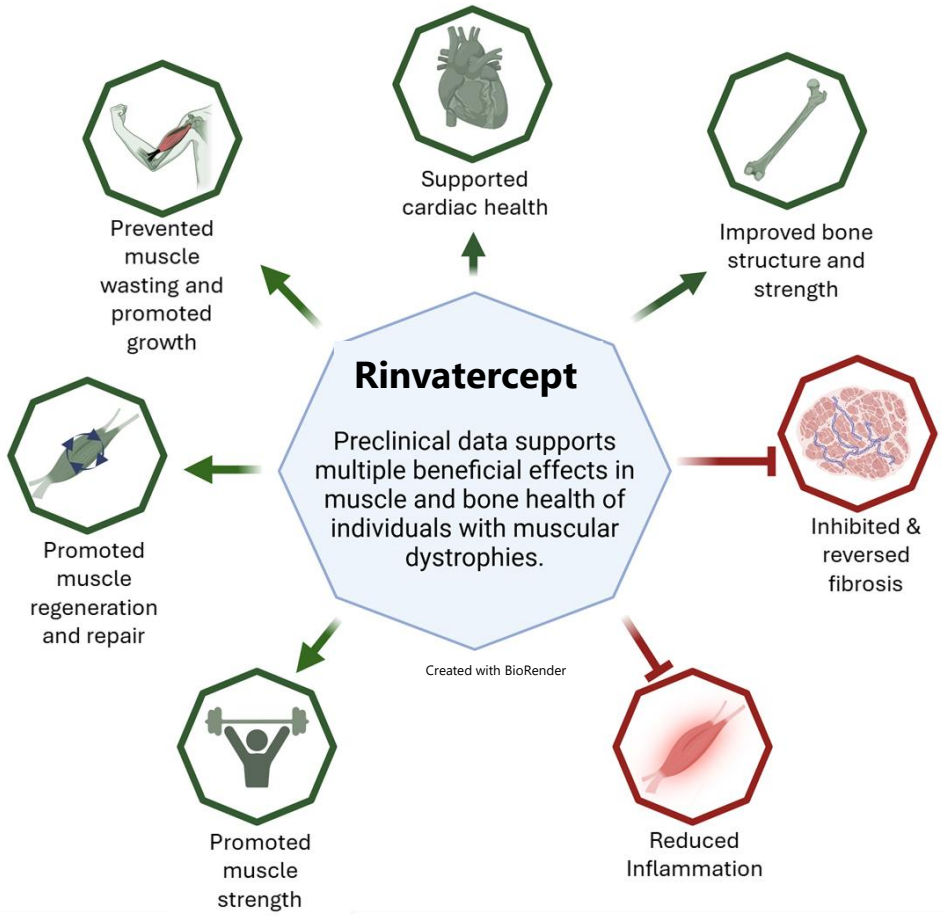
- FDA granted ELEVIDYS® full approval for the treatment of ambulatory individuals aged 4+ and accelerated approval for the treatment of non-ambulatory individuals aged 4+
- Approved using the accelerated approval pathway on basis of expression of micro-dystrophin

HDAC Inhibitors

- Modulate the deregulated activity of HDACs in dystrophic muscle
- Duvyzat™ (givinostat) was approved by the FDA in patients aged 6+
- Duvyzat™ can cause dose-related thrombocytopenia and other signs of myelosuppression, including anemia and neutropenia

Given limitations of currently available therapies, the need for additional treatments in DMD remains high

Robust Nonclinical Data Suggests Potential Benefits of Rinvatercept Through Activin A Inhibition



Activin + myostatin inhibition

✓ **Offer greater muscle and fat benefits:** Potential to increase muscle mass and decrease fat mass beyond myostatin alone, based on nonclinical data and prior experience with ActRIIB-Fc and Keros molecules

Broader potential associated with activin inhibition

✓ **Improve bone structure and strength:**
- Improved bone mineral density in nonclinical and Phase 1 healthy volunteer trial

✓ **Reduce negative effects of glucocorticoid treatment:**
- Co-treatment with prednisolone increased both muscle mass and strength
- Improved trabecular bone and strength in dystrophic D2.MDX mice treated in combination with glucocorticoids

✓ **Ameliorate inflammation:**
- A shift in the macrophage population from pro-inflammatory M1 macrophages to tissue-repairing M2 macrophages in dystrophic D2.MDX mice

✓ **Promote muscle regeneration:**
- Increased satellite cell proliferation and differentiation to myofibers in wild-type mice

✓ **Help address underlying genetic deficiency:**
- Improved lean mass and grip strength and enhanced expression of truncated dystrophin in dystrophic D2.MDX mice after combination therapy with exon-skipping therapy (PMO) compared to treatment with PMO only
- Increased utrophin expression and muscle strength in dystrophic D2.MDX mice

✓ **Protect respiratory and cardiac function**
- Potential to slow muscle damage and reduce fibrosis that leads to increased strain on the cardio-pulmonary system

D2.MDX = mouse model of DMD

Treatment with Rinvatercept Was Generally Well Tolerated in Phase 1 Healthy Volunteer Trial

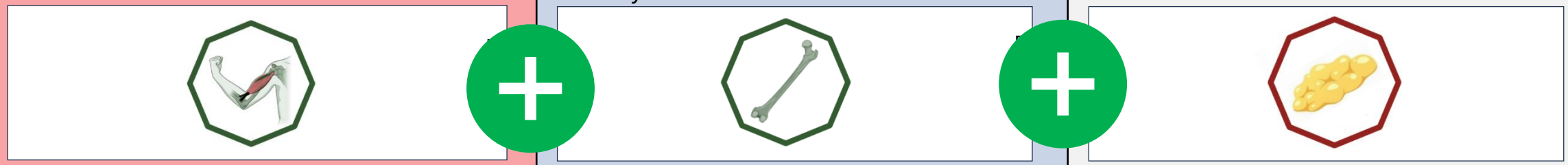
Keros completed a randomized, double-blind, placebo-controlled, two-part Phase 1 clinical trial to evaluate single (1.0 mg/kg, 3.0 mg/kg and 5.0 mg/kg) and multiple (1.25 mg/kg and 2.0 mg/kg) ascending doses of rinvatercept in healthy volunteers. The primary objectives of this trial were to assess safety, tolerability and pharmacokinetics of rinvatercept. Exploratory endpoints included assessments of the pharmacodynamic effect on bone, adipose, muscle, cardiac tissue and fibrosis

- **Most TEAEs were mild (Grade 1) to moderate (Grade 2)**
  - One unrelated Grade 4 TEAE (transient CK elevation)* was observed
- **No dose-limiting toxicities or serious adverse events were observed**
  - No bleeding events/telangiectasias were observed
  - All injection site reactions (except two Grade 3 injection site erythema AEs) were non-severe; all resolved without sequelae
  - All headache AEs (except one Grade 2) were mild; all resolved without sequelae
- **Observed increases in hemoglobin were asymptomatic and reversible**

Natarajan, H. et al, ASBMR 2025; AE grading was based on the DAIDS Table for Grading the Severity of Adult and Pediatric Adverse Events, Version 2.1 (July 2017).
CK = creatine kinase , TEAE = Treatment-emergent adverse event
*Grade 4 AE in participant receiving rinvatercept 2.0 mg/kg: CK elevation to ~17,000 that was unrelated to study drug. Participant had recently undergone 45 minutes of weightlifting. Symptoms only of mild biceps soreness after curls. CK decreased by 50% within 2 days and resolved without sequelae within 2 weeks. Participant received a subsequent dose of rinvatercept and did not experience a CK elevation.

Phase 1 Healthy Volunteer Data Support Potential for Rinvatercept to Address Multiple Aspects of DMD

Muscle	Bone	Fat
In DMD, the replacement of muscle fibers with fatty and fibrotic tissue leads to progressive loss of muscle strength and function, leading to immobility and respiratory and cardiac complications **Rinvatercept elicited:** • Increased lean muscle mass • Increased thigh muscle volume	Reduced muscle strength, loss of ambulation and use of glucocorticoids in DMD contribute to the development of secondary osteoporosis **Rinvatercept elicited:** • Increases in BSAP demonstrating mobilization of osteoblasts, which are crucial for bone formation • Decreases in CTX, a biomarker that measures the rate of bone resorption • Increased whole body bone mineral density that was durable and sustained, suggesting a balance of osteoblast and osteoclast activity	Decreased mobility and the use of glucocorticoids are associated with increased risk of obesity and related negative health consequences **Rinvatercept elicited:** • Increases in adiponectin, a biomarker of fat mobilization • Decreases in leptin, a biomarker of fat mass observed • Corresponding decreases in fat mass, both whole body and visceral fat mass, were observed



Natarajan, H. et al, ASBMR 2025

Rinvatercept: Phase 2 Clinical Trial Design

We have commenced an open-label, multi-cohort Phase 2 basket clinical trial to evaluate rinvatercept in patients with DMD and we expect to report initial data from this trial in the first half of 2027

Screening Period
Up to 6 weeks

Intra-Patient Dose Escalation*

Open-Label Treatment Period (48 weeks); last dose at week 44

Safety Follow-Up
4 weeks post-end of treatment or 8 weeks post-last dose

Late-Ambulatory DMD Patients

Cohort A1 : Stable Glucocorticoids + Rinvatercept

Early Non-Ambulatory DMD Patients

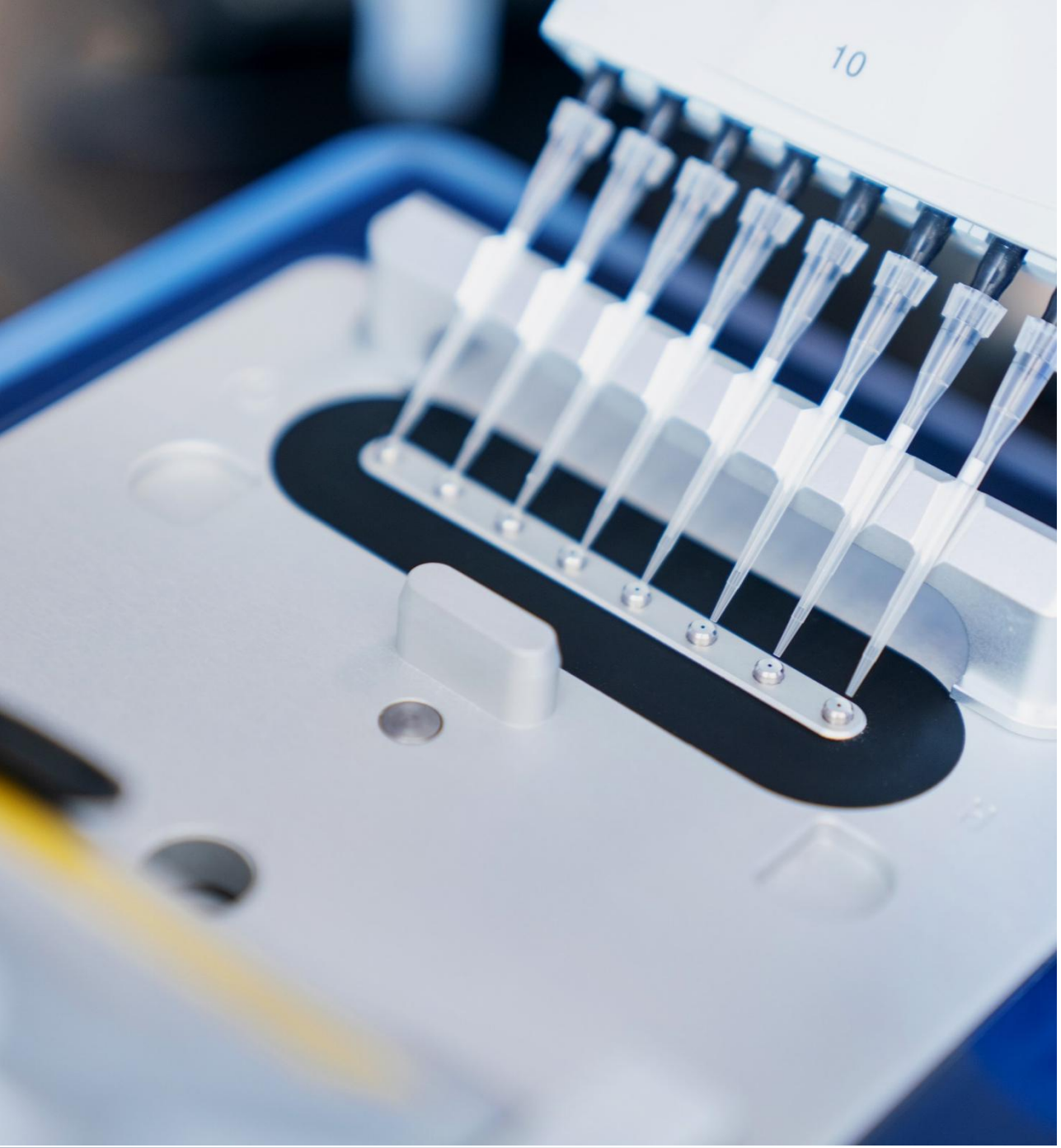
Cohort N1: Stable Glucocorticoids + Rinvatercept

Trial Population
- Total N = ~24

Endpoints
- **Primary**
  - Safety and Tolerability
- **Key Secondary**
  - Pharmacokinetics
  - Anti-drug antibodies
  - Body composition
  - Comprehensive functional improvements (skeletal muscle, motor, cardiac and pulmonary)
- **Exploratory**
  - Serum Biomarkers

*Rinvatercept is administered to patients subcutaneously once every four weeks, starting at a dose of 0.5 mg/kg. Based on individual titration rules, the dose may be escalated in 0.5 mg/kg increments after two doses, up to a maximum dose of 2.0 mg/kg

PK = pharmacokinetics; ADA = anti-drug antibodies

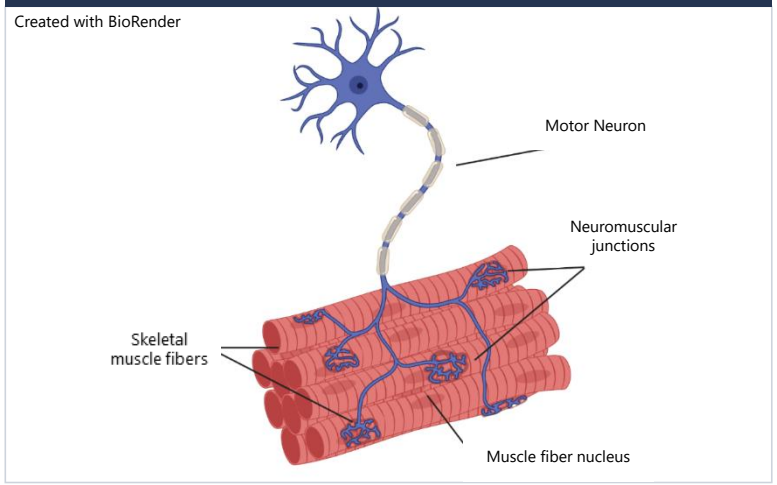


Rinvatercept:
Amyotrophic Lateral Sclerosis (ALS)

Rinvatercept and ALS: A Novel Approach

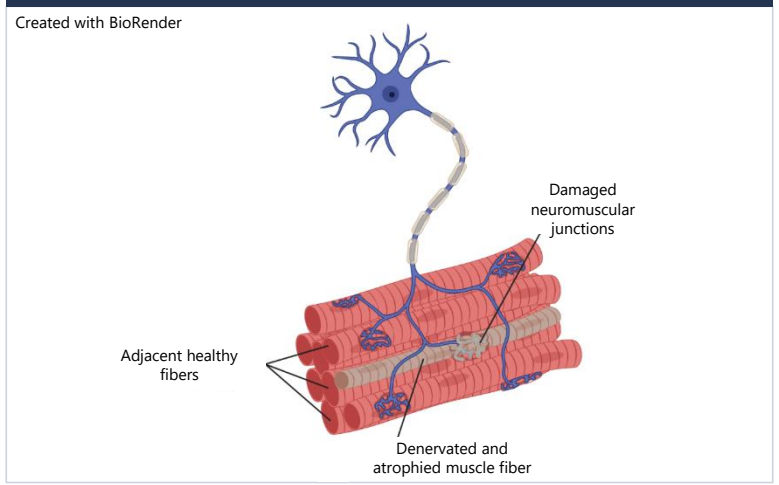
- ALS is a progressive, neurodegenerative disease that destroys motor neurons, leading to muscle weakness, loss of function and eventually paralysis[1]
- In the U.S. alone, ALS has a prevalence of ~9.9 per 100,000 people, with ~33K people estimated to have been living with ALS in 2022[2]
- The exact cause of ALS is unknown; ~90% cases are sporadic with no known family history, while ~10% of cases are familial, linked to mutations in certain genes (e.g., SOD1, C9orf72)[1,2]

Healthy Motor Unit



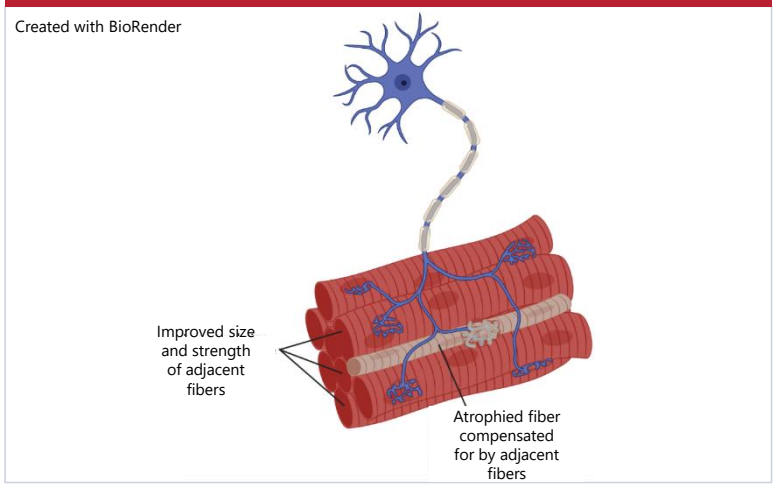
Each muscle is made up of multiple motor units. A motor unit consists of a motor neuron that is connected and activates each muscle fiber via a neuromuscular junction

ALS



In ALS, the loss of motor neurons and neuromuscular junctions leads to atrophy and a subsequent loss of connected muscle fibers. However, healthy, connected adjacent muscle fibers and motor units remain

Rinvatercept Mechanism of Action



Rinvatercept has the potential to maximize the functional strength of adjacent fibers, allowing them to compensate for atrophied fibers and motor units. This has the potential to lead to overall preservation of muscle strength and improved quality of life

Rinvatercept has the potential to preserve muscle function and maintain quality of life

1. Brown, R.H. et al, NEJM 2017, 162-172; 2. Mehta P. et al, Amyotrophic Lateral Scler. Frontotemporal Degener. 2025, 290-295

Potential Benefits of Rinvatercept Treatment for Individuals with ALS

Skeletal muscle is potentially an active participant in ALS pathology, instead of a passive victim of motor neuron degeneration[1]

By promoting muscle growth and function, rinvatercept has the potential to:

- ✓ Counteract muscle atrophy
- ✓ Preserve the neuromuscular junction
- ✓ Reduce inflammation
- ✓ Enhance muscle regeneration

We are collaborating with the Massachusetts General Hospital to design a Phase 2 clinical trial evaluating rinvatercept in patients with ALS within the Healey ALS MyMatch program, led by the Sean Healey & AMG Center for ALS at Mass General Brigham

We plan to engage regulators on the design of this Phase 2 clinical trial evaluating rinvatercept in patients with ALS in the second half of 2026

1. Duranti, E. The Role of Skeletal Muscle in Amyotrophic Lateral Sclerosis: State of the Art 2025. *Muscles* 2025 v.4, 22.

Third-Party Myostatin Inhibitor Data Demonstrated That Muscle Preservation Can Provide Functional Benefit

Motor neuron loss is a hallmark of both spinal muscular atrophy (SMA) and ALS

ISEMBYLD (Apitegromab), a third-party product candidate approved for the treatment of SMA, established proof-of-concept that preserving muscle can provide clinically meaningful benefit in patients with SMA

Keros believes the data from the SAPPHIRE Phase 3 trial of apitegromab in SMA support that muscle-targeted agents can preserve muscle function even when neuronal degeneration is slowed or persists[1]

Functional gains were observed (e.g., significant HFMSE improvement, p=0.0192)

Based on the apitegromab Phase 3 data, we believe rinvatercept could potentially sustain function in still-innervated muscle fibers in patients with ALS

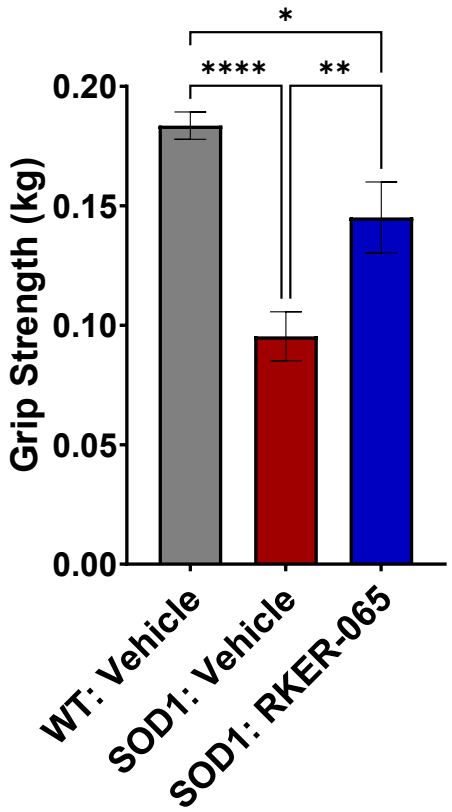
ALS trials have generally focused on preserving or reversing the motor neuron loss. With rinvatercept, we plan to target the skeletal muscle to potentially preserve the strength of innervated muscle and provide quality of life benefit

1. Crawford, T. et al; Lancet Neurology 2025, v.24, 9, 727-739

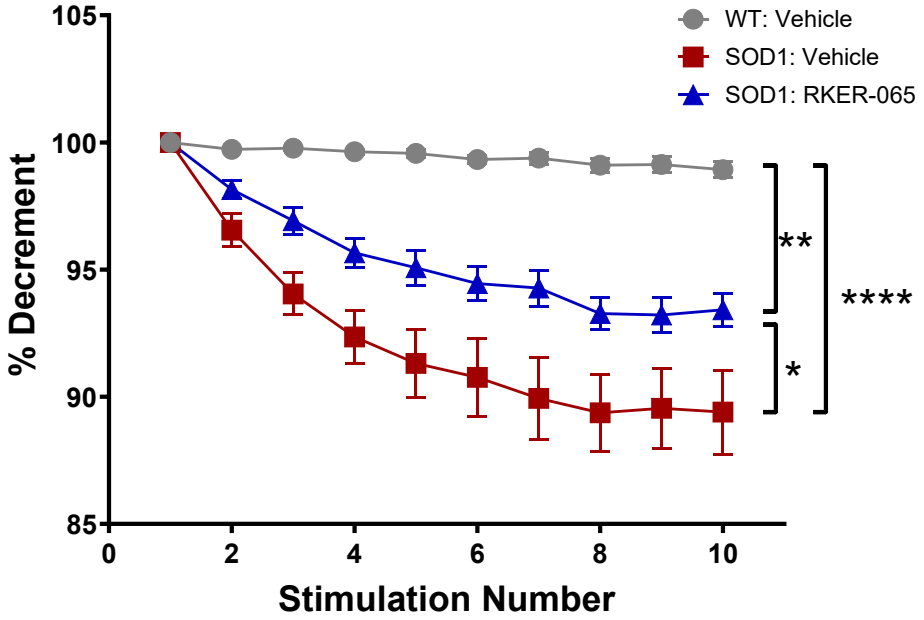
HFMSE = Hammersmith Functional Motor Scale Expanded

RKER-065 Treatment Preserved Muscle Strength and Neuromuscular Transmission in a Mouse Model of ALS

Grip Strength



Motor Unit Function (Repetitive Nerve Stimulation, RNS 10 Hz)



A decrease in repetitive nerve stimulation is supportive of a dysfunction at the neuromuscular junction, where nerve signals are unable to consistently activate the muscles

Six-week-old male SOD1-G93A (SOD1) mice (mouse model of ALS) were intraperitoneally injected with vehicle or RKER-065 (10 mg/kg, BIW) for 8 weeks. Vehicle-treated wild-type mice served as a control group (WT). Grip strength and the action potential amplitude of the tibialis anterior muscle following repetitive nerve stimulation were assessed at the end of the study. Data is shown as average ± SEM. One-way ANOVA (grip strength), 2-way ANOVA (RSN): * P≤0.05, ** P<0.01, and **** P< 0.0001.

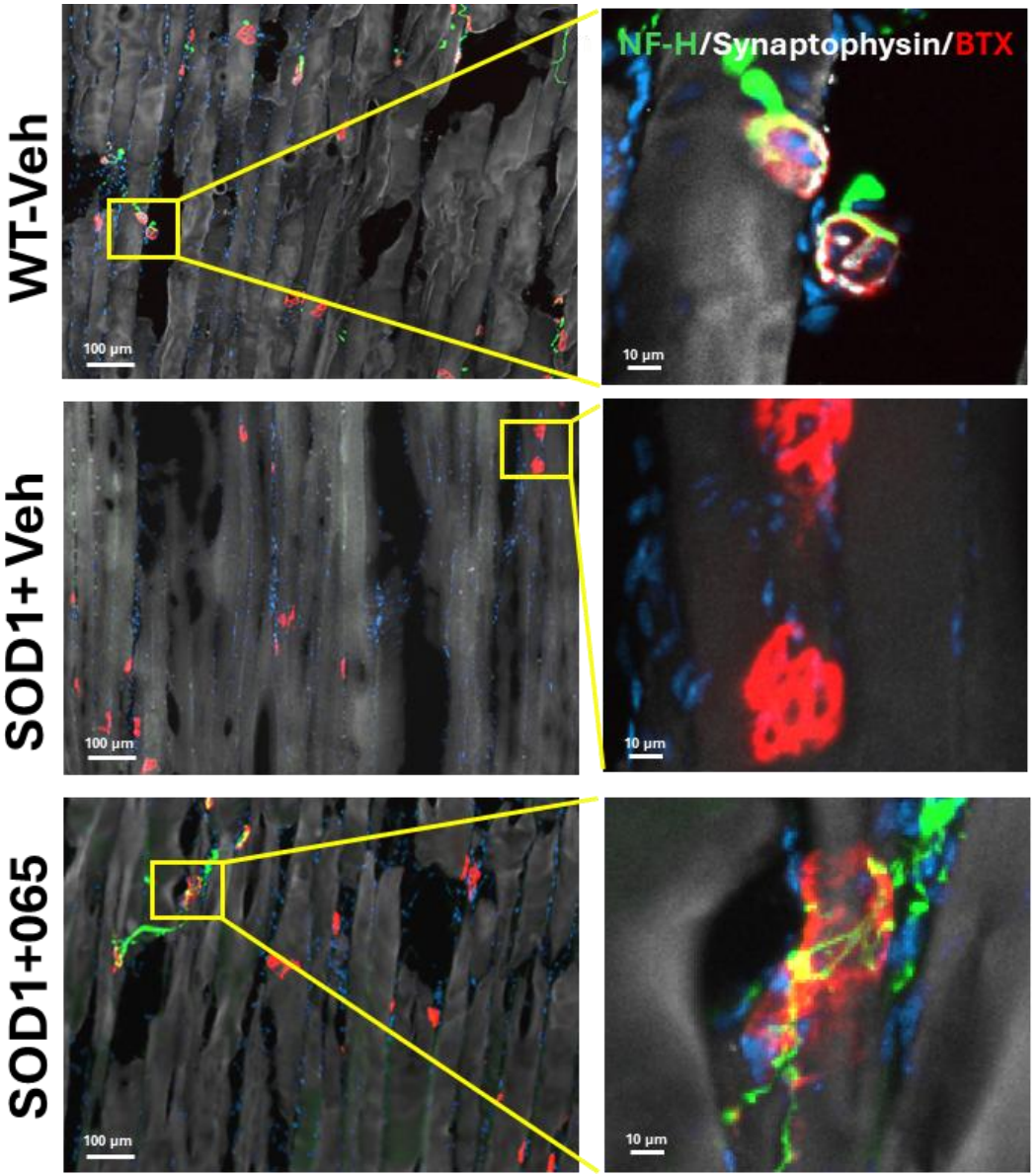
RKER-065 Treatment Protected the Neuromuscular Junction in a Mouse Model of ALS

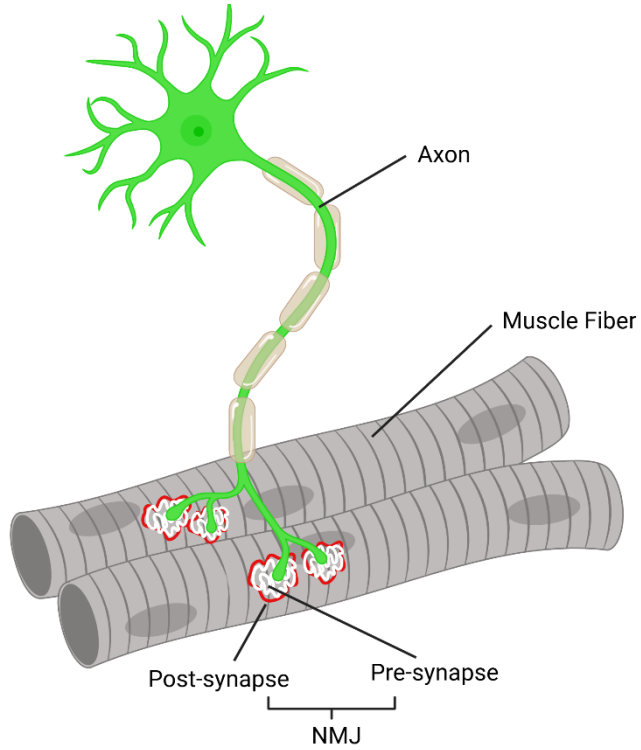
SOD1-G93 Mouse Model of ALS

In healthy neuromuscular junctions (NMJs), there is a normal complement of neurofilaments at the axon terminal

However, in certain neuromuscular disorders like ALS, there is a deficit of neurofilaments at the NMJ, leading to pathology and dysfunction

RKER-065 treatment showed a protective effect on the NMJ, suggesting rinvatercept has the potential to treat patients with ALS





Created with BioRender

NF-H: (neurofilament protein-heavy) marker of axon integrity (green); **Synaptophysin**: marker of presynaptic nerve terminal (white); **BTX**: (bungarotoxin) marker of postsynaptic acetylcholine receptors on the muscle fiber membrane (red). The nuclei were counterstained with DAPI (blue)



Elritercept (KER-050)

Global License Agreement with Takeda Pharmaceuticals

Keros entered into an exclusive license agreement with Takeda to develop, manufacture and commercialize elritercept globally, other than mainland China, Hong Kong, and Macau

Takeda is currently conducting three Phase 3 trials with elritercept for treatment of anemia and thrombocytopenia in:

RENEW	ELRiSE MDS	ELRiSE MF
2L myelodysplastic syndromes	1L myelodysplastic syndromes	Myelofibrosis

Keros received an upfront payment of $200 million

Eligible to receive development, approval and commercial milestone payments of over $1.1 billion

Development	Commercial	Sales
$90M	**$280M**	**$740M**
$10M milestone received for RENEW milestone FPD $20M milestone received for ELRiSE MDS FPD $20M milestone triggered for ELRiSE MF FPD		

Tiered royalties on net sales in the low double-digits to high teens

FPD= first patient dosed



Proprietary Discovery Approach

Proprietary Discovery Approach

Keros has a broad approach to target the TGF-β superfamily to develop product candidates with the potential to treat a broad range of indications, including neuromuscular and neurodegenerative disorders, rare bone and fibrosis diseases and obesity/frailty

Rinvatercept, elritercept and KER-206 were nominated from our proprietary library of modified ActRII ligand traps for clinical development

Multiple candidates in nonclinical development

Large library of agonists and antagonists

Proprietary library of modified ActRII ligand traps	Mono, bi and multimodal antagonist	Systemically deliverable ligands

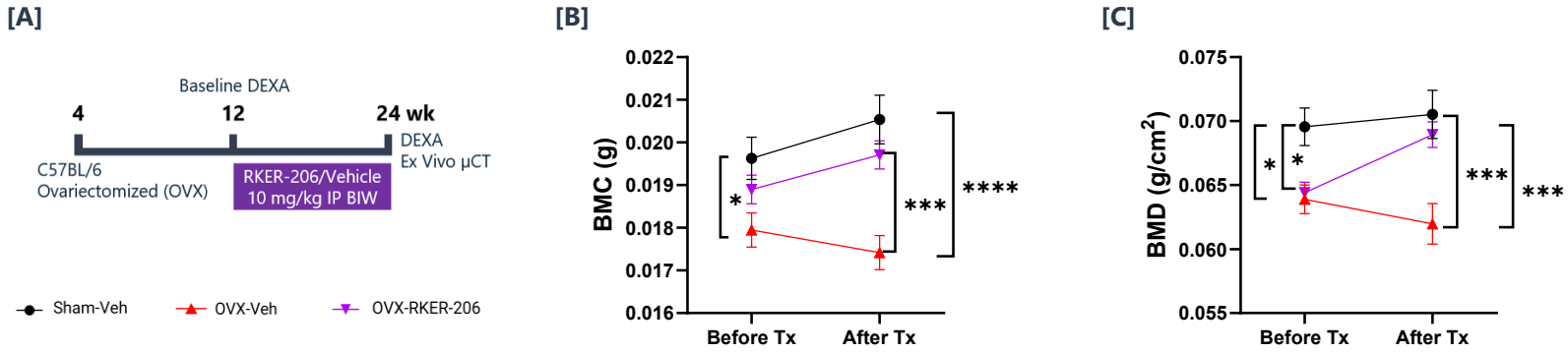
KER-206 Has the Potential to Increase Bone Mass and Improve Skeletal Integrity in Disorders Characterized by Pathological Bone Loss

KER-206 is a nonclinical asset that was nominated out of our **proprietary library of ActRII ligand traps for further investigation**:

- Novel ligand trap comprised of a modified ligand-binding domain derived from ActRIIB that is fused to the portion of the human antibody known as the Fc domain
- Designed to bind select ligands from the TGF-β superfamily, **including activin A, activin B, GDF8 and GDF11**

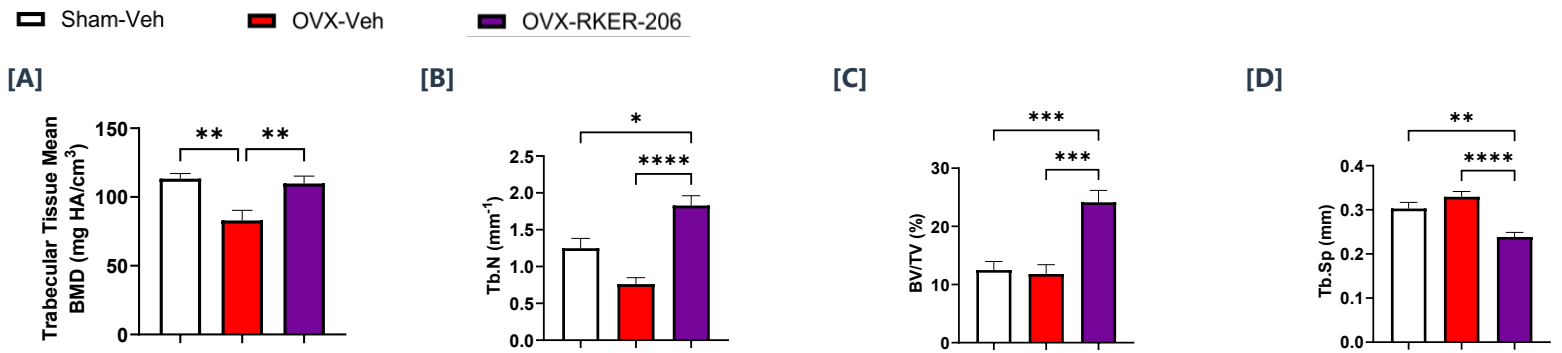
KER-206 is **designed to bind select TGF-β ligands which regulate bone remodeling through effects on differentiation and activity of bone-resorptive cells (osteoclasts) and bone-forming cells (osteoblasts)**

RKER-206 Administration Improved Bone Mass in Ovariectomized Mice



Administration of RKER-206 reversed bone loss in a mouse model of osteoporosis. [A] Schematic illustrating a study design. [B] In vivo DEXA analysis of bone mineral content (BMC) and [C] bone mineral density (BMD) of the femur before and after 12 weeks of treatment with RKER-206. Data are shown as average ± SEM. N=12-24/group. Mixed-effects analysis using restricted maximum likelihood (Tukey): *P<0.05, *** P<0.001, and **** P<0.0001. Veh: vehicle; OVX: ovariectomized; Tx: treatment.

RKER-206 Administration Improved Trabecular Bone Architecture in Ovariectomized Mice



Administration of RKER-206 improved tibia microarchitecture in a mouse model of osteoporosis. [A] trabecular tissue mean BMD, [B] bone volume fraction (BV/TV%), [C] trabecular number [Tb.N], and [D] trabecular separation (Tb.Sp). Data are shown as average ± SEM. N= 7-12/group. One-way ANOVA (Tukey): **P <0.01, *** P< 0.001, **** P< 0.0001.

Ishimwe, J. et al, ASBMR 2026