

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 3, 2026

Keros Therapeutics, Inc.
(Exact name of registrant as specified in its charter)

Delaware
(state or other jurisdiction
of incorporation)

001-39264
(Commission
File Number)

81-1173868
(I.R.S. Employer
Identification No.)

1050 Waltham Street, Suite 302

Lexington, Massachusetts
(Address of principal executive offices)

02421
(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 7.01 Regulation FD Disclosure.

On August 3, 2026, Keros Therapeutics, Inc. (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 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 Securities Exchange Act of 1934, as amended (the “Exchange Act”) or otherwise subject to the liabilities of that section. The information contained herein and in the accompanying exhibit is 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 2026.
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 3, 2026



Corporate Presentation

August 2026



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, expected results and timing of its preclinical studies and clinical trials for rinwatercept (KER-065), including its regulatory plans; the potential of rinwatercept to treat patients with Duchenne muscular dystrophy and patients with amyotrophic lateral sclerosis; and the potential of Keros’ proprietary discovery approach, including additional indications which we may pursue. 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 product candidates, rinwatercept and elritercept; 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; and Keros’ dependence on third parties in connection with manufacturing, clinical trials and preclinical studies.

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 August 3, 2026, 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.

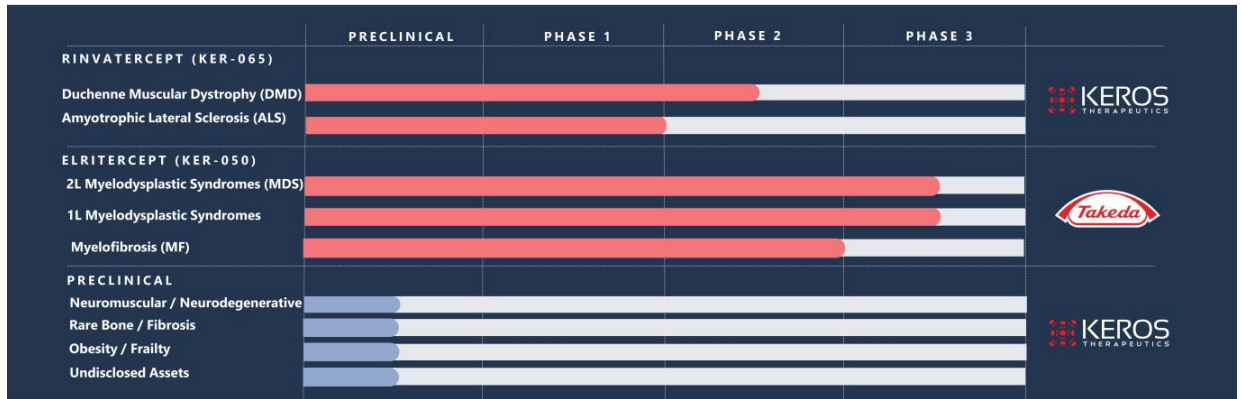
The trademarks included in this presentation are the property of the owners thereof and are used for reference purposes only.



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

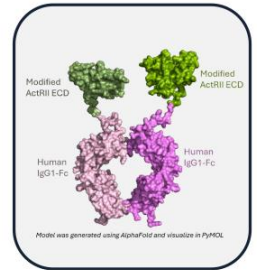




Rinvatercept (KER-065)

Rinvatercept (KER-065)

- Rinvatercept is an investigational modified activin receptor type II ligand trap
- Rinvatercept is designed to bind and inhibit TGF- β ligands, including activin A and myostatin, which are negative regulators of muscle and bone mass and strength, 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 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



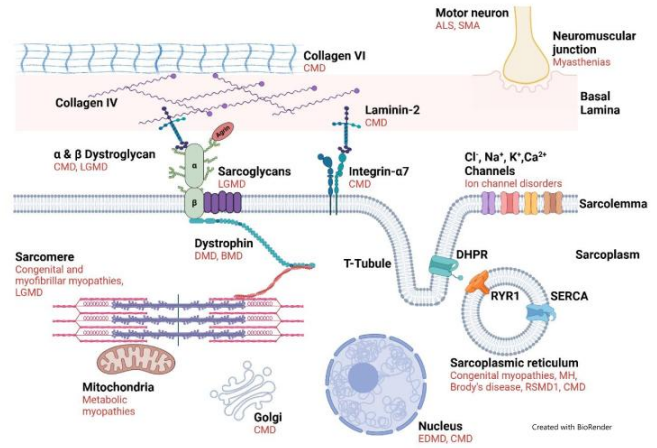
Rinvatercept: Potential to Treat a Broad Range of Neuromuscular Diseases

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 preclinical studies and the Phase 1 clinical trial, we believe rinvatercept has potential in multiple, rare neuromuscular diseases with high unmet need





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 infiltration¹⁻³
- 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. Tabeibordbar, 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

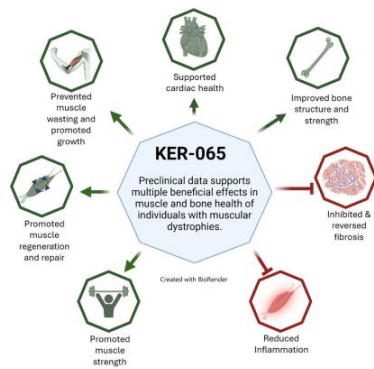
HDAC Inhibitors

- Modulate the deregulated activity of HDACs in dystrophic muscle
- Duvyzt™ (givinostat) was approved by the FDA in patients aged 6+
- Duvyzt™ 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 Preclinical Data Suggests Potential Benefits of Rinvatercept



 D2.MDX = mouse model of DMD

- **Offer muscle, bone and fat benefits:** Potential to increase muscle mass, decrease fat mass and improve BMD, based on preclinical data and prior experience with ActRIIB-Fc and Keros molecules
- **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

Inhibition of both activins and myostatin can potentially offer greater benefit than myostatin inhibition alone

Activin inhibition (but not myostatin inhibition) offers therapeutic potential

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

We 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.

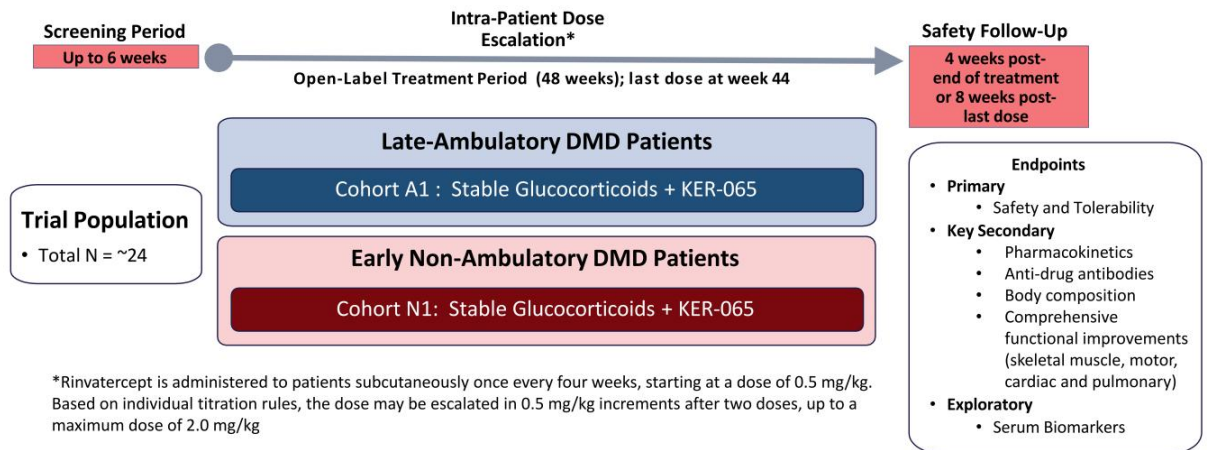


Phase 1 Healthy Volunteer Data Support Potential for Rinvatercept to Address Multiple Aspects of DMD

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
		

Rinvatercept: Phase 2 Clinical Trial Design

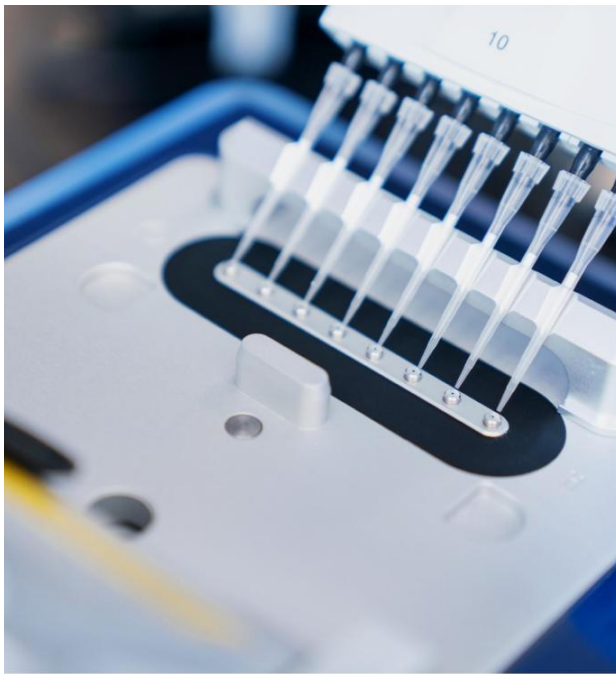
We have initiated an open-label, multi-cohort Phase 2 basket clinical trial to evaluate rinvatercept in patients with DMD and we expect to report initial data from this trial in the first half of 2027



PK = pharmacokinetics; ADA = anti-drug antibodies

Corporate Presentation

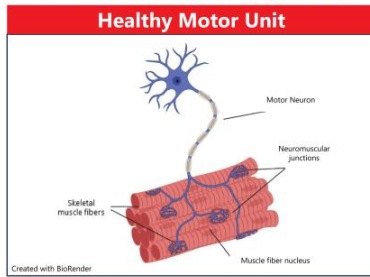
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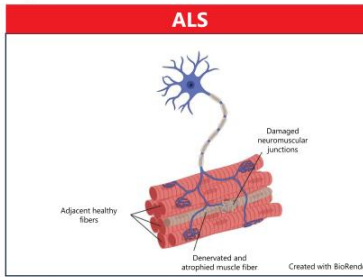
Rinvatercept: *Amyotrophic Lateral Sclerosis (ALS)*

Rinvatercept and ALS: A Novel Approach

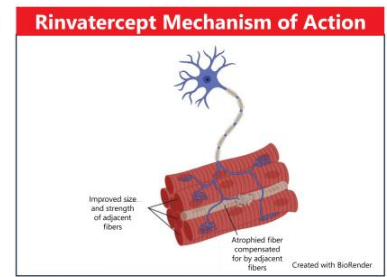
- ALS is a progressive, neurodegenerative disease that destroys motor neurons, leading to muscle weakness, loss of function and eventually paralysis¹
- In the U.S. alone, ALS has a prevalence of ~9.9 per 100,000 people, with ~33K people estimated to have been living with ALS in 2022²
- The exact cause 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}



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



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 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
Corporate Presentation

Third-Party Product Candidate (Apitegromab) Phase 3 Data Demonstrated That Muscle Preservation Can Provide Benefit

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

- ▶ Apitegromab, a third-party product candidate being developed for the treatment of SMA, established proof-of-concept that preserving muscle can provide clinically meaningful benefit in patients with SMA
- ▶ We believe 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 persists¹
 - ▶ 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

Potential Benefits of Rinvatercept Treatment for Individuals with ALS

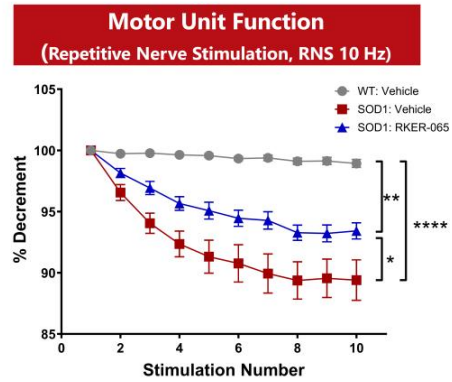
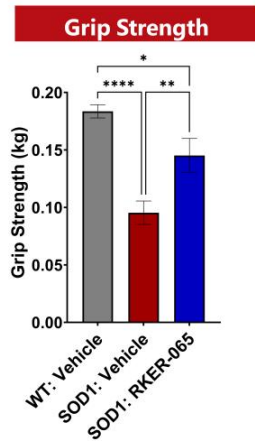
- **Skeletal muscle is potentially an active participant in ALS pathology, instead of a passive victim of motor neuron degeneration¹**
- By promoting muscle growth and function, rinvatercept has the potential to:
 - Counteract muscle atrophy
 - Preserve the neuromuscular junction
 - Reduce inflammation
 - 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.

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



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 $\pm$ SEM. One-way ANOVA (grip strength), 2-way ANOVA (RSN): * $P \leq 0.05$, ** $P < 0.01$, and **** $P < 0.0001$.



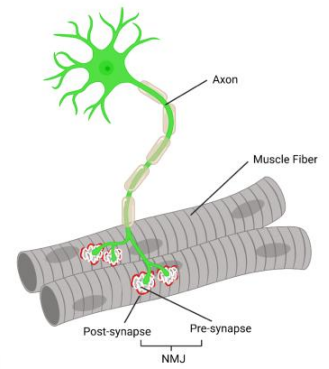
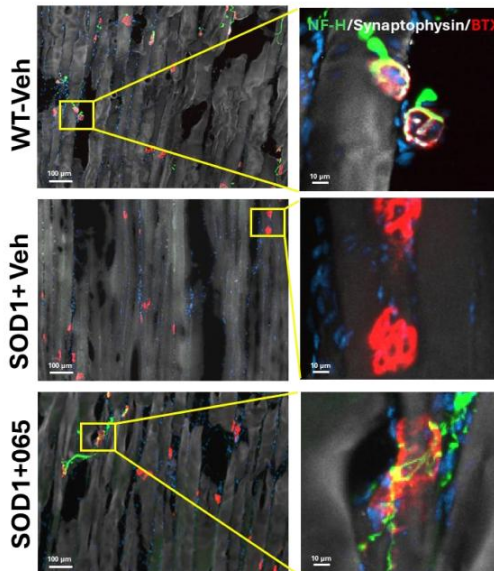
RKER-065 Treatment Protected the Neuromuscular Junction in a Mouse Model of ALS

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)





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Elritercept (KER-050)

**Investigational Treatment for Anemia and
Thrombocytopenia in Patients with
Myelodysplastic Syndromes and in Patients
with Myelofibrosis**



On December 3, 2024, Keros announced it had entered into an exclusive license agreement with Takeda to develop, manufacture and commercialize elritercept globally, other than mainland China, Hong Kong, and Macau

Financials:

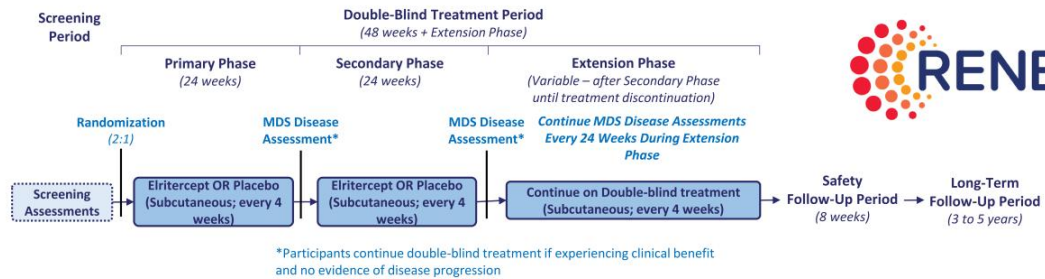
- Keros received an upfront payment of \$200 million
- Eligible to receive development, approval and commercial milestone payments of over \$1.1 billion
 - Development: \$90M (\$10M received in 2025 for Phase 3 RENEW clinical trial first patient dosed)
 - Commercial: \$280M
 - Sales: \$740M
- Tiered royalties on net sales in the low double-digits to high teens

Under the terms of the agreement, in January 2025, Takeda became responsible for all clinical development, manufacturing and commercialization of elritercept in its territory



RENEW Trial: Ongoing Phase 3 Clinical Trial to Evaluate Elritercept in Patients with MDS

- Based on data from our Phase 2 clinical trial of elritercept in patients with MDS, a global, multicenter, double-blind, randomized, placebo-controlled Phase 3 RENEW clinical trial was initiated to evaluate the efficacy and safety of elritercept versus placebo in patients with transfusion-dependent anemia with lower-risk MDS
- The primary endpoint is the proportion of patients achieving transfusion independence for at least eight weeks from baseline through week 24
- A key secondary endpoint is the proportion of patients achieving transfusion independence for at least 24 weeks from baseline through week 48





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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
 - Large library of agonists and antagonists
 - Proprietary library of modified ActRII ligand traps
 - Mono, bi and multimodal antagonist
 - Systemically deliverable ligands
 - Multiple candidates in preclinical development
- ✓ **Rinvatercept** and **elritercept** were both nominated from our proprietary library of modified ActRII ligand traps for clinical development



