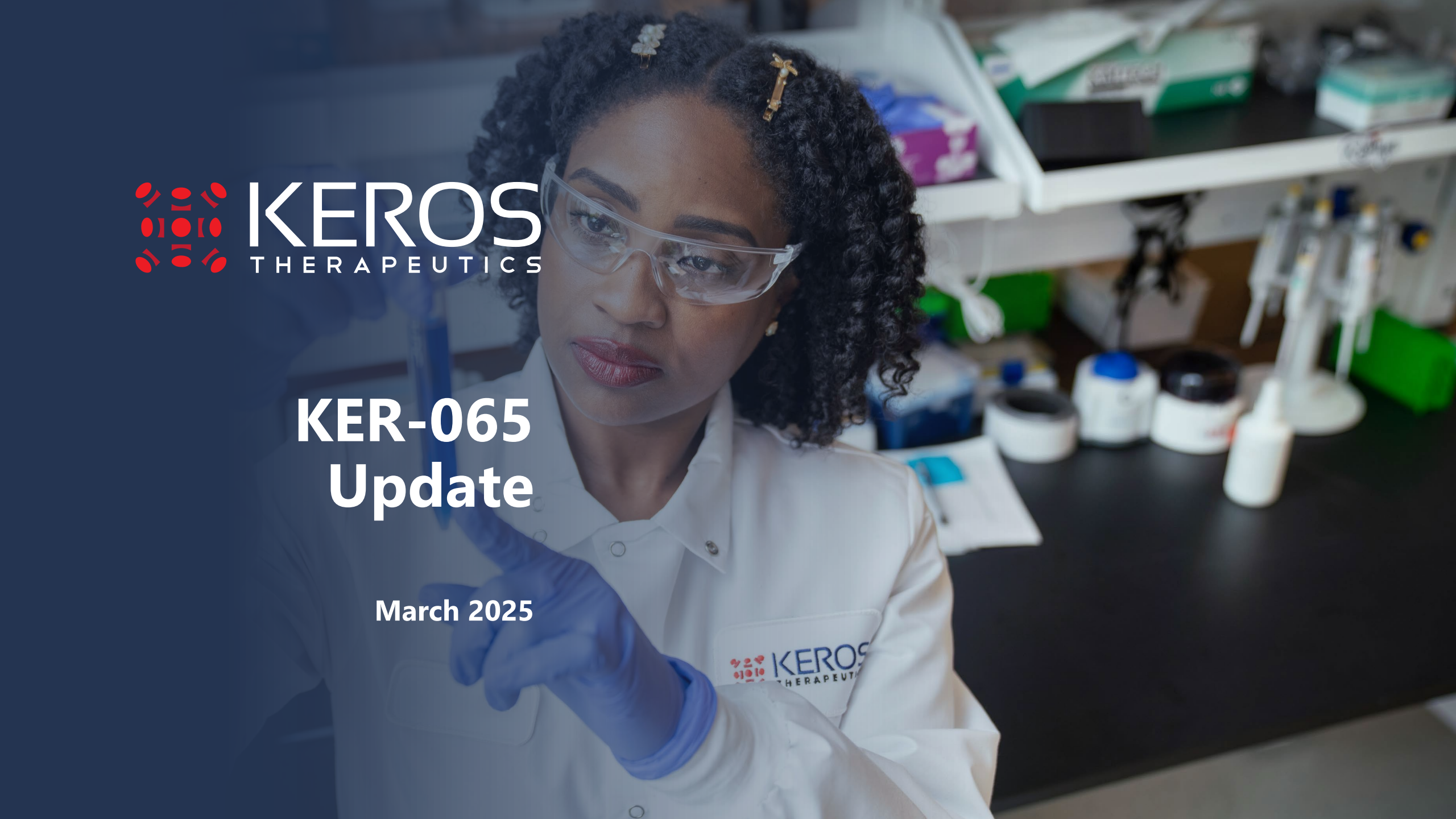

KEROS THERAPEUTICS

KER-065 Update

March 2025

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 strategy, progress, design, objectives and expected timing of its Phase 2 clinical trial for KER-065, including its regulatory plans and potential therapeutic benefits of KER-065. 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, cibotercept, KER-065 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 Annual Report on Form 10-K, filed with the SEC on February 26, 2025, 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.

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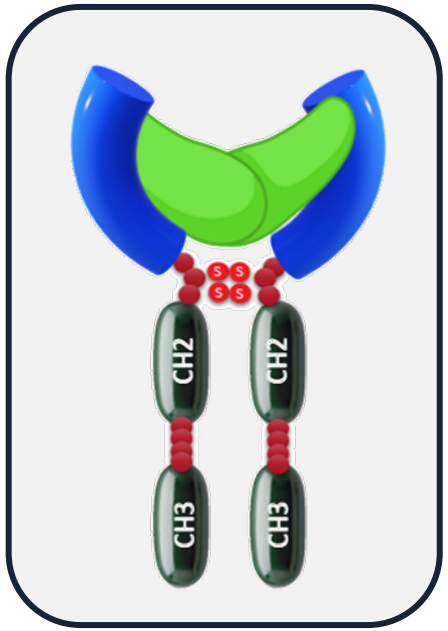
KER-065

- **KER-065 is an investigational modified activin receptor IIA (ActRIIA) and activin receptor IIB (ActRIIB) ligand trap**
  - ~50% amino acids derived from each activin receptor



- **KER-065 is designed to bind and inhibit activin A and myostatin to:**
  - Improve muscle regeneration to increase muscle size and strength
  - Inhibit and reverse fibrosis
  - Inhibit inflammation
  - Reduce fat
  - Improve bone health through bone anabolic mechanisms

- **KER-065 is designed differently from other activin receptor ligand traps:**
  - Reduced binding to bone morphogenic proteins (BMPs) to avoid the vascular/bleeding events observed with ActRIIb-Fc derived from the native sequences

KER-065: Duchenne Muscular Dystrophy (DMD)

- Chronic degenerative muscle diseases eventually lead to a collapse in the ability of muscle to regenerate and eventual loss of function
- DMD manifests as subtle motor defects postnatally leading to loss of ambulation and eventually death[1,2]
- The National Organization for Rare Disorders estimates that approximately one in every 3,500 male births is affected by DMD worldwide
- In young boys with 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[2-4]

DMD Disease Progression[5,6]

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. Parker, A. E., et al. (2005). QJM 98, 729–736. doi: 10.1093/qjmed/hci113; 2. Tabebordbar, M., et al. (2013). Annu. Rev. Pathol. 8, 441–475. doi: 10.1146/annurev-pathol-011811-132450; 3.Wallace, G. Q., and McNally, E. M. (2009). Annu. Rev. Physiol. 71, 37–57. doi:10.1146/annurev.physiol.010908.1632164.4; 4. Mann, C. J.,et al. (2011). Skelet. Muscle 1:21. doi: 10.1186/2044-5040-1-21; 5. Bushby K, Connor E. Clin Investig (Lond) 2011; 1:1217-1235; 6. 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

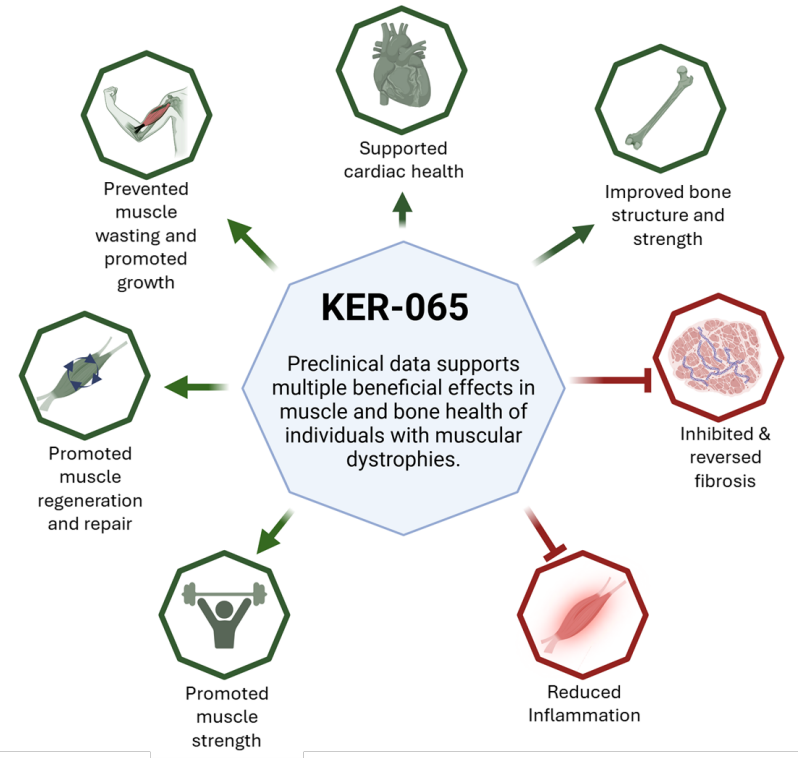
- FDA granted ELEVIDYS® full approval for the treatment of ambulatory individuals aged 4+ and accelerated approval for the treatment of non-ambulatory individuals aged 4+
- Approved using the accelerated approval pathway on basis of expression of micro-dystrophin

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 Preclinical Data Suggests Potential Benefits of KER-065



- **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

D2.MDX = mouse model of DMD

KER-065:
Phase 1 Clinical Trial Data

Encouraging Preclinical and Phase 1 Trial Data Support Rationale for Phase 2 Trial

- **Trial met key objectives for safety, tolerability, pharmacokinetics and pharmacodynamics**
  - Pharmacokinetic profile generally consistent with that of a well-behaved biologic
  - Dose exposure levels supportive of monthly dosing
- **KER-065 was generally well-tolerated, with no major safety signals observed as of the data cut-off date**
- **Pharmacodynamic data offer multiple lines of evidence that we may be achieving sufficient activin inhibition across tissues of interest, at drug exposures that we anticipate targeting in DMD**

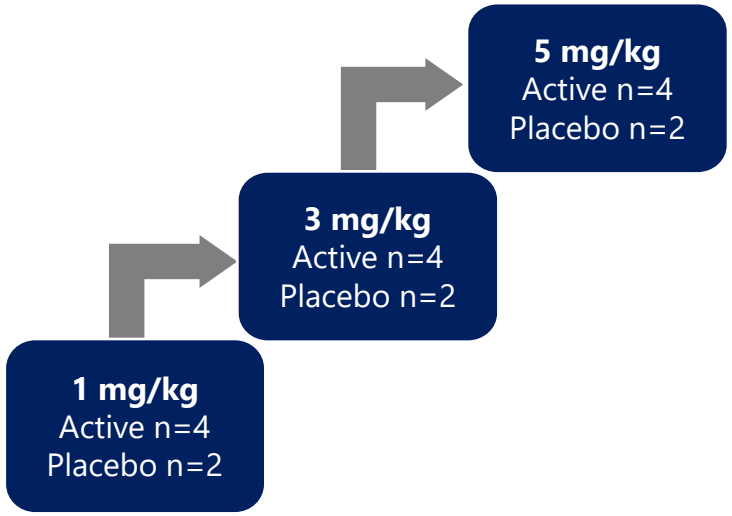
KER-065 Phase 1 Trial Design

Key Inclusion Criteria

Part 1 SAD:
Males ≥ 18 to ≤ 65 years
BMI ≥ 18 to ≤ 35 kg/m^2

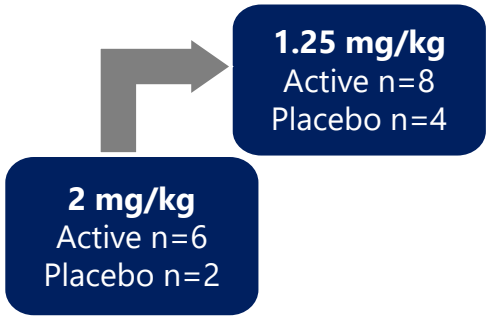
Part 2 MAD:
Males ≥ 18 to ≤ 65 years
BMI ≥ 27 to ≤ 35 kg/m^2
WHR ≥ 0.9

Part 1
Single Ascending Dose (SAD)
(Double-blinded)



Treatment period: 28 days
Safety follow up: 28 days
Single subcutaneous dose

Part 2
Multiple Ascending Dose (MAD)
(Double-blinded)



Treatment period: 85 days
Safety follow up: 56 days
Three subcutaneous doses (28 days apart)

Endpoints

Primary: Safety

Secondary: PK

Exploratory: PD (imaging and serum biomarkers)

All data in this presentation are presented as of a data cut-off date of February 6, 2025. Data are presented through the treatment period (MAD Day 85).

Baseline Demographics Consistent with Desired Population to Enroll

- All participants in this trial were male and generally healthy
- Higher baseline weight, BMI and waist-to-hip ratio in MAD relative to SAD, further supporting evaluation of body composition parameters
- Wide ranges within various characteristics, reflecting anthropometric diversity among trial population

		SAD				MAD		
		Placebo (N=6)	KER-065 1.0 mg/kg (N=4)	KER-065 3.0 mg/kg (N=4)	KER-065 5.0 mg/kg (N=4)	Placebo (N=6)	KER-065 1.25 mg/kg (N=8)	KER-065 2.0 mg/kg (N=7)*
Age (yrs)	Mean (SD)	33.0 (8.46)	28.0 (6.93)	26.3 (11.84)	26.5 (7.33)	46.2 (9.37)	38.8 (11.78)	34.6 (12.01)
	Min, Max	23, 45	22, 38	20, 44	19, 35	33, 61	26, 61	18, 52
Weight (kg)	Mean (SD)	74.36 (12.76)	76.30 (13.32)	70.18 (6.06)	67.64 (14.94)	99.72 (9.85)	94.82 (10.18)	91.11 (7.62)
	Min, Max	58.0, 94.6	59.1, 87.7	64.5, 77.8	52.1, 82.3	87.7, 117.0	74.5, 105.5	78.0, 100.0
BMI (kg/m2)	Mean (SD)	24.55 (3.42)	24.80 (3.27)	23.85 (2.52)	23.00 (3.26)	30.40 (2.94)	30.21 (2.06)	29.76 (1.53)
	Min, Max	19.4, 27.9	21.8, 29.3	21.1, 26.5	20.0, 27.3	27.5, 34.5	27.1, 33.2	27.6, 32.1
Waist-to-Hip Ratio	Mean (SD)	0.88 (0.11)	0.88 (0.04)	0.87 (0.08)	0.79 (0.04)	0.93 (0.02)	0.93(0.04)	0.92 (0.03)
	Min, Max	0.76, 1.02	0.84, 0.93	0.77, 0.98	0.76, 0.86	0.90, 0.95	0.86, 0.98	0.90, 0.99
Height (cm)	Mean (SD)	173.95 (8.3)	175.03 (9.6)	171.68 (2.6)	170.70 (9.05)	181.18 (6.1)	177.01 (6.7)	174.97 (8.4)
	Min, Max	159.3, 184.0	164.7, 187.3	168.5, 174.8	161.3, 182.0	170.7, 186.5	164.6, 186.0	160.0, 188.0

*One participant discontinued (for personal reasons and unrelated to an adverse event or study drug) following the first dose and a new participant was added to the cohort

Treatment with KER-065 Was Generally Well Tolerated

- Most TEAEs were mild (Grade 1) to moderate (Grade 2)
- No dose-limiting toxicities or serious adverse events were observed
- No bleeding events / telangiectasias were observed
- One unrelated Grade 4 TEAE* was observed (transient CK elevation)

	SAD				MAD		
	Placebo (N=6) n (%)	KER-065 1.0 mg/kg (N=4) n (%)	KER-065 3.0 mg/kg (N=4) n (%)	KER-065 5.0 mg/kg (N=4) n (%)	Placebo (N=6) n (%)	KER-065 1.25 mg/kg (N=8) n (%)	KER-065 2.0 mg/kg (N=7) n (%)
TEAE	5 (83.3)	3 (75.0)	3 (75.0)	4 (100)	5 (83.3)	8 (100)	6 (85.7)
Related TEAE	2 (33.3)	2 (50.0)	3 (75.0)	4 (100)	2 (33.3)	6 (75.0)	6 (85.7)
Gr ≥3 TEAE	0	0	1 (25.0)	0	1 (16.7)	1 (12.5)	2 (28.6)
Related TEAE of ≥Gr 3	0	0	1 (25.0)	0	0	1 (12.5)	0
TESAE	0	0	0	0	0	0	0

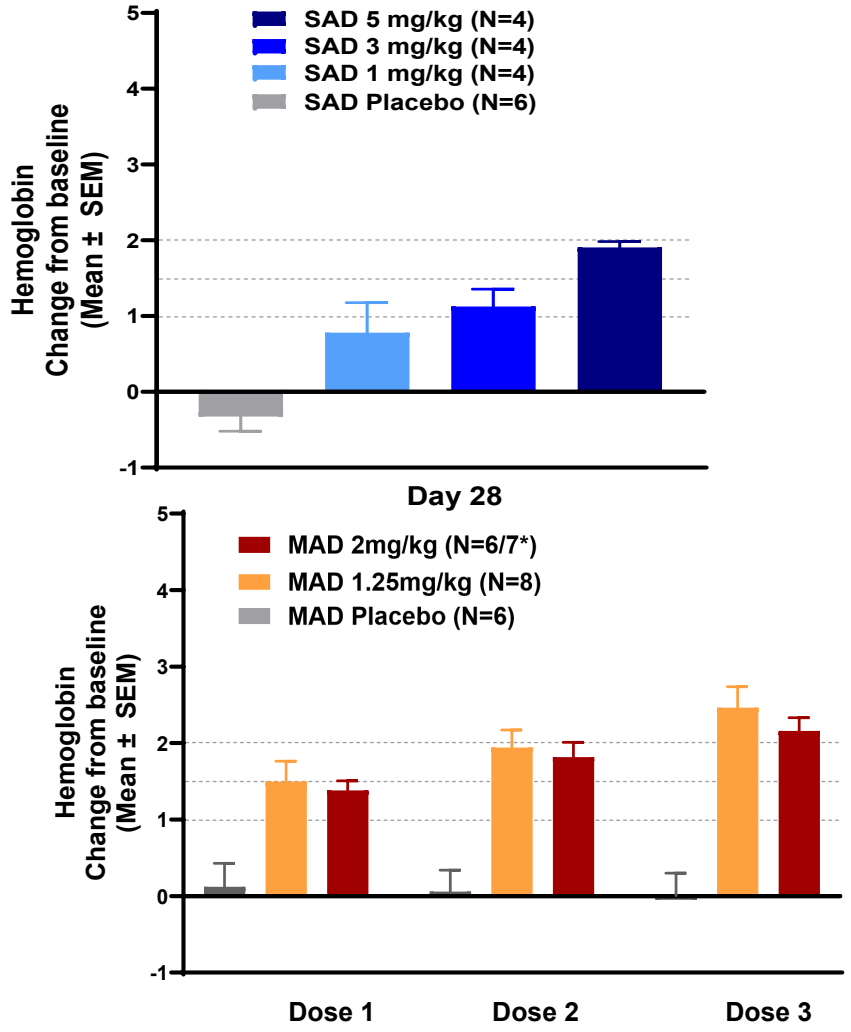
AE grading was based on the DAIDS Table for Grading the Severity of Adult and Pediatric Adverse Events, Version 2.1 (July 2017).
CK = creatine kinase , IMP = Investigational medicinal product; TEAE = Treatment-emergent adverse event; TESAE = Treatment-emergent serious adverse event
*Grade 4 AE in participant receiving KER-065 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 KER-065 and did not experience a CK elevation.

Treatment with KER-065 Was Generally Well Tolerated

- All injection site reactions (except two Grade 3 injection site erythema AEs) were not severe; all resolved without sequelae
- All headache AEs (except one Grade 2) were mild; all resolved without sequelae.
- The lab value-based AEs were transient and resolved without sequelae

Most Commonly Reported TEAEs (TEAE Preferred Term)	SAD					MAD			
	KER-065 1.0 mg/kg (N=4) n (%)	KER-065 3.0 mg/kg (N=4) n (%)	KER-065 5.0 mg/kg (N=4) n (%)	KER-065 Total (N=12) n (%)	Placebo (N=6) n (%)	KER-065 1.25 mg/kg (N=8) n (%)	KER-065 2.0 mg/kg (N=7) n (%)	KER-065 Total (N=15) n (%)	Placebo (N=6) n (%)
Injection site erythema	2 (50.0)	2 (50.0)	0	**4 (33.3)**	**2 (33.3)**	4 (50.0)	3 (42.9)	**7 (46.7)**	**0**
Headache	1 (25.0)	0	2 (50.0)	**3 (25.0)**	**0**	1 (12.5)	4 (57.1)	**5 (33.3)**	**1 (16.7)**
Blood creatine phosphokinase increased	0	2 (50.0)	1 (25.0)	**3 (25.0)**	**0**	3 (37.5)	1 (14.3)	**4 (26.7)**	**1 (16.7)**
Alanine aminotransferase increased	0	0	1 (25.0)	**1 (8.3)**	**0**	2 (25.0)	2 (28.6)	**4 (26.7)**	**2 (33.3)**
Lipase increased	0	1 (25.0)	0	**1 (8.3)**	**0**	2 (25.0)	2 (28.6)	**4 (26.7)**	**0**
Injection site pain	1 (25.0)	0	0	**1 (8.3)**	**2 (33.3)**	1 (12.5)	2 (28.6)	**3 (20.0)**	**0**
Aspartate aminotransferase increased	0	0	1 (25.0)	**1 (8.3)**	**0**	2 (25.0)	1 (14.3)	**3 (20.0)**	**3 (50.0)**
Injection site pruritus	0	1 (25.0)	0	**1 (8.3)**	**0**	1 (12.5)	2 (28.6)	**3 (20.0)**	**0**

Observed Mean Change in Hemoglobin From Baseline Dose of KER-065

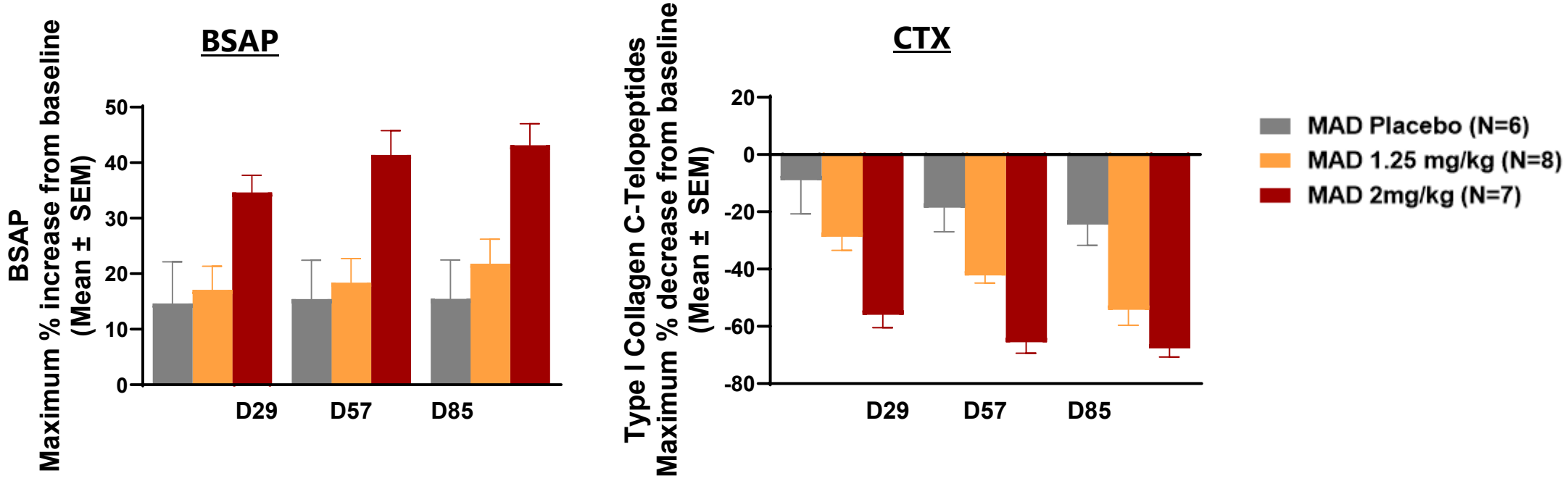


- **Increases in hemoglobin were asymptomatic and reversible**
- **SAD:**
  - Increases in hemoglobin were observed, most prominently in the highest dose
- **MAD:**
  - Hemoglobin increase observed, primarily following the initial dose and to a lesser degree upon each subsequent dose
  - Higher dose level in MAD was not associated with a greater increase in hemoglobin

*denotes N=7 after dose 1 for MAD 2mg/kg

Increases in BSAP and Decreases in CTX Observed Following KER-065 Administration

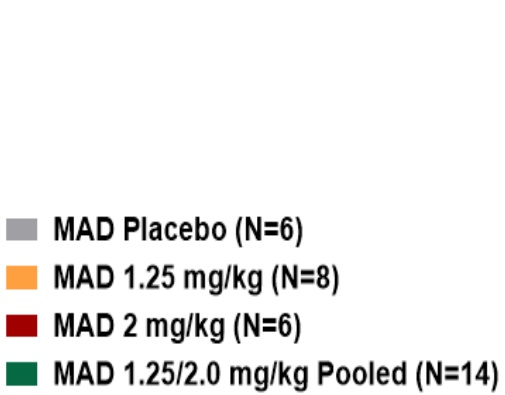
- **Bone specific alkaline phosphatase (BSAP)** is a biomarker of bone formation
- **C-terminal telopeptide (CTX)** is a biomarker of bone resorption

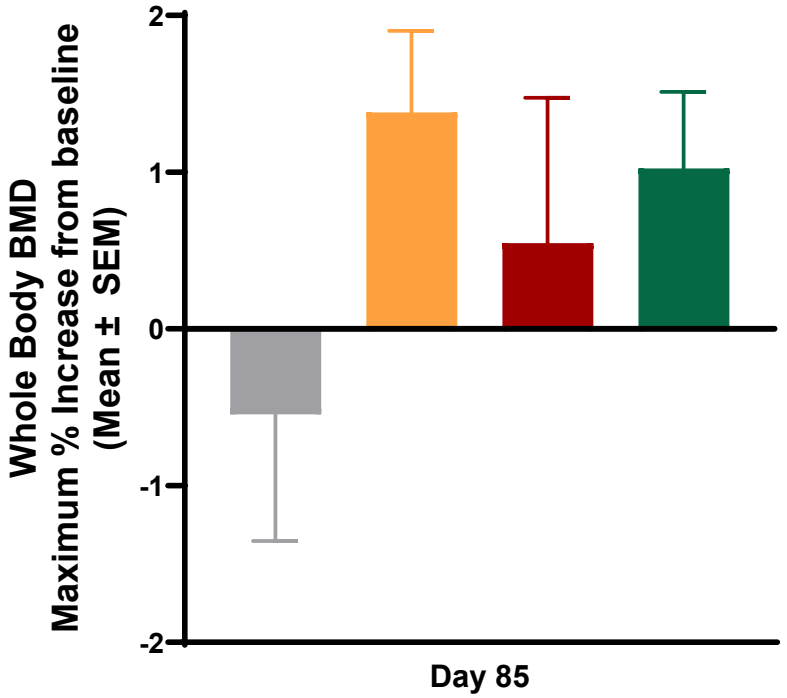


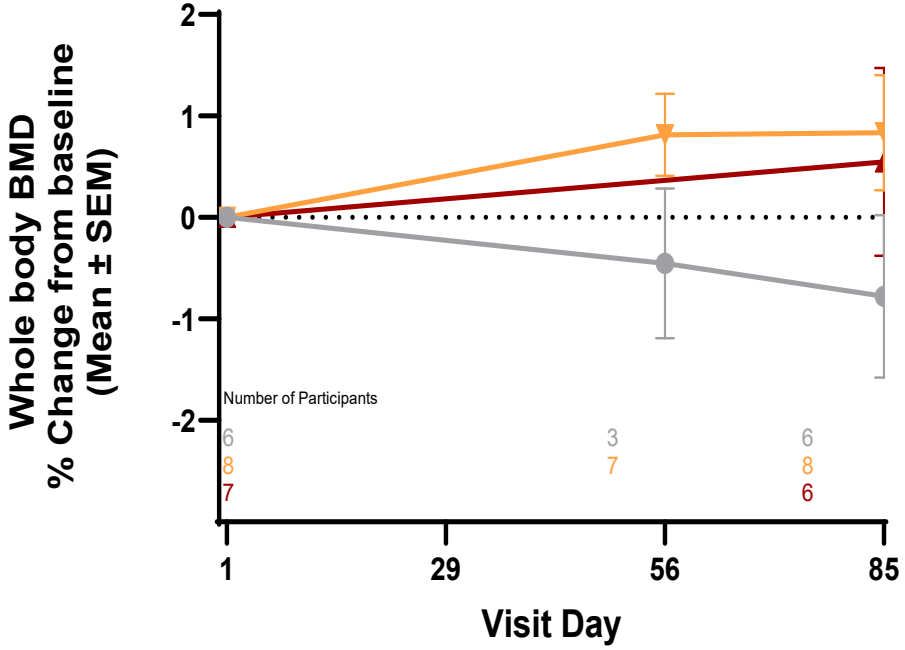
KER-065 treatment demonstrated the potential for bone anabolic activity by simultaneously increasing bone formation (BSAP) while inhibiting bone resorption (CTX)

Data represented at each timepoint is baseline through day listed

KER-065 Administration Led to Increased Whole Body Bone Mineral Density (BMD)





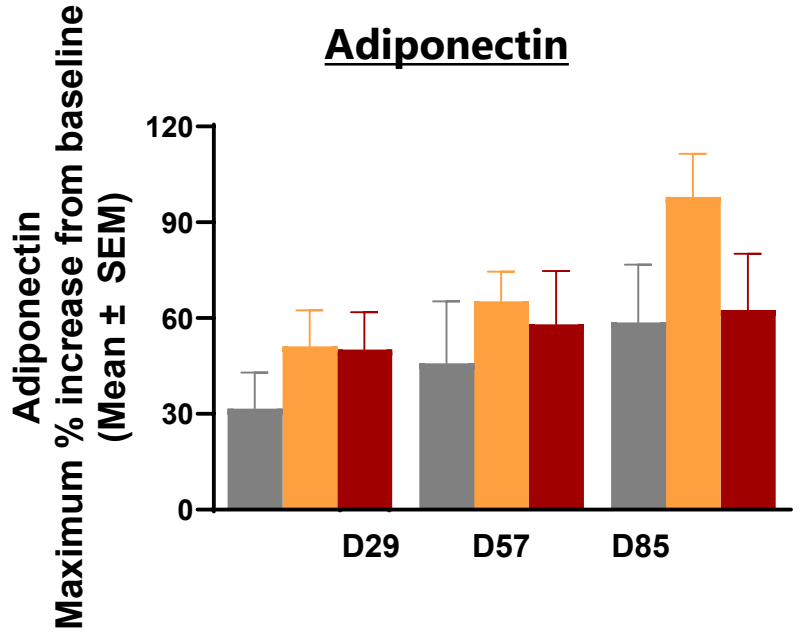


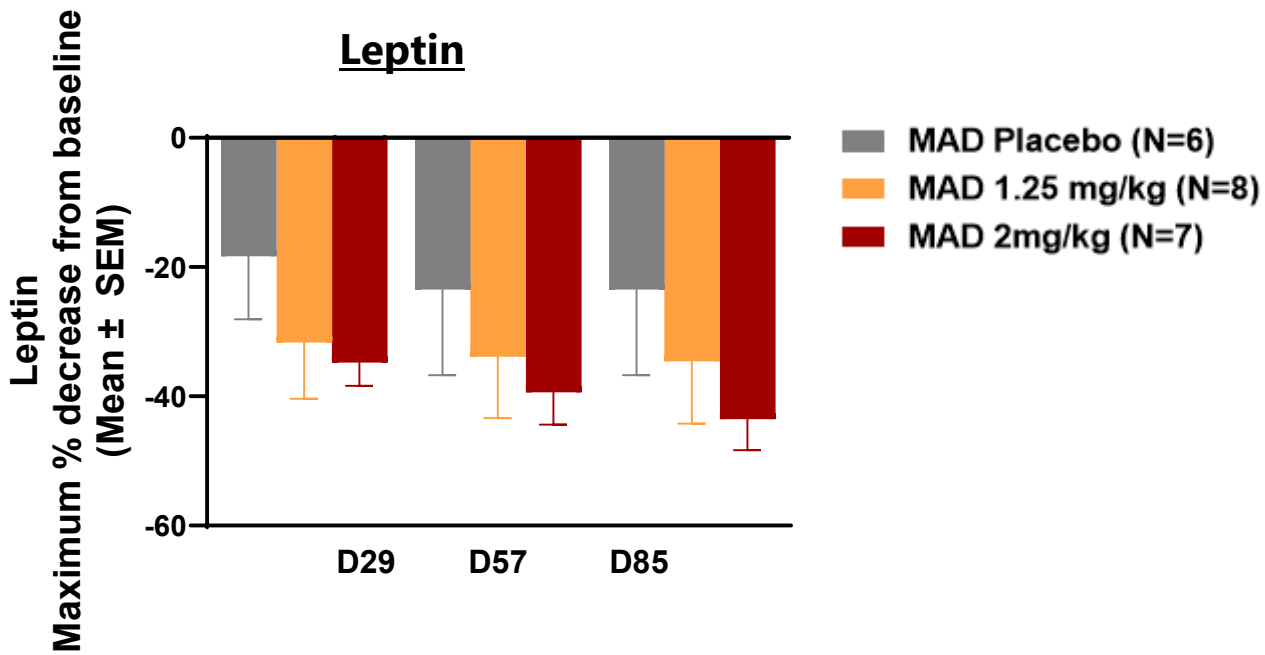
Changes in bone biomarkers of increased bone formation and reduced bone resorption were consistent with tissue level changes, as demonstrated by observed increase in BMD

Maximum increase data represented is baseline through day listed

Adiponectin Increased and Leptin Decreased Following KER-065 Administration

- **Adiponectin** is a biomarker of fat mobilization
- **Leptin** is a biomarker of fat mass

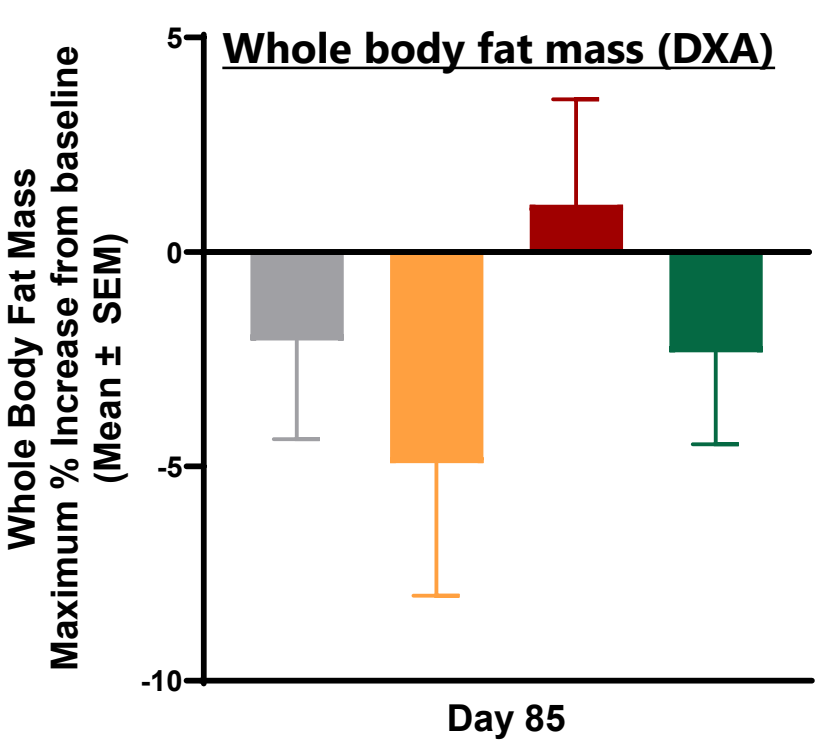


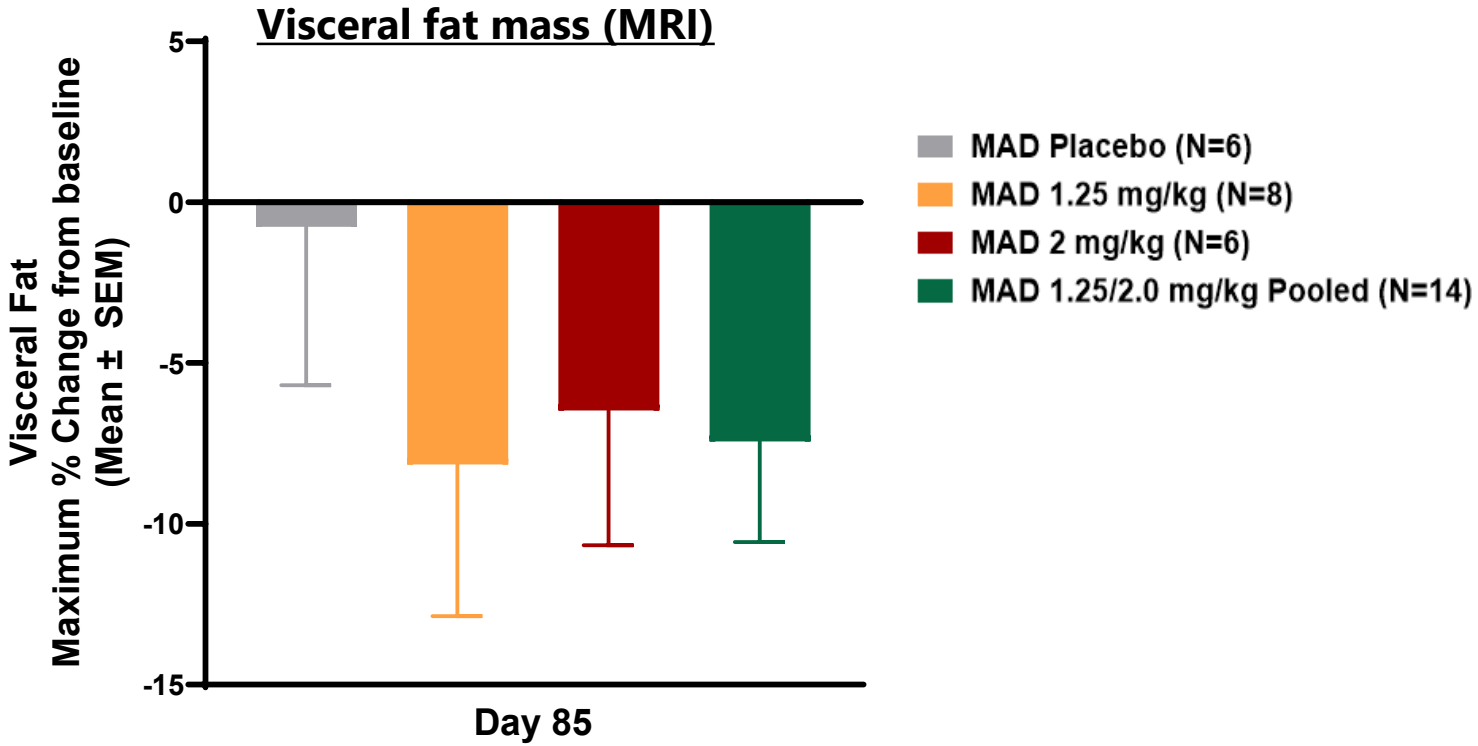


Observed increase in adiponectin and reduction in leptin are supportive of fat mobilization

Data represented at each timepoint is baseline through day listed

KER-065 Administration Led to Decreases in Fat Mass

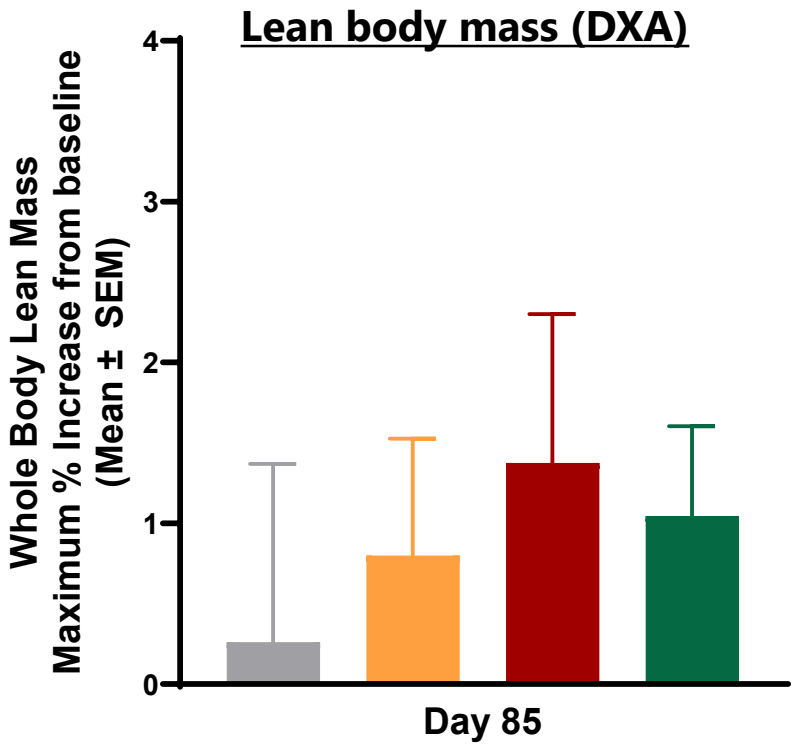


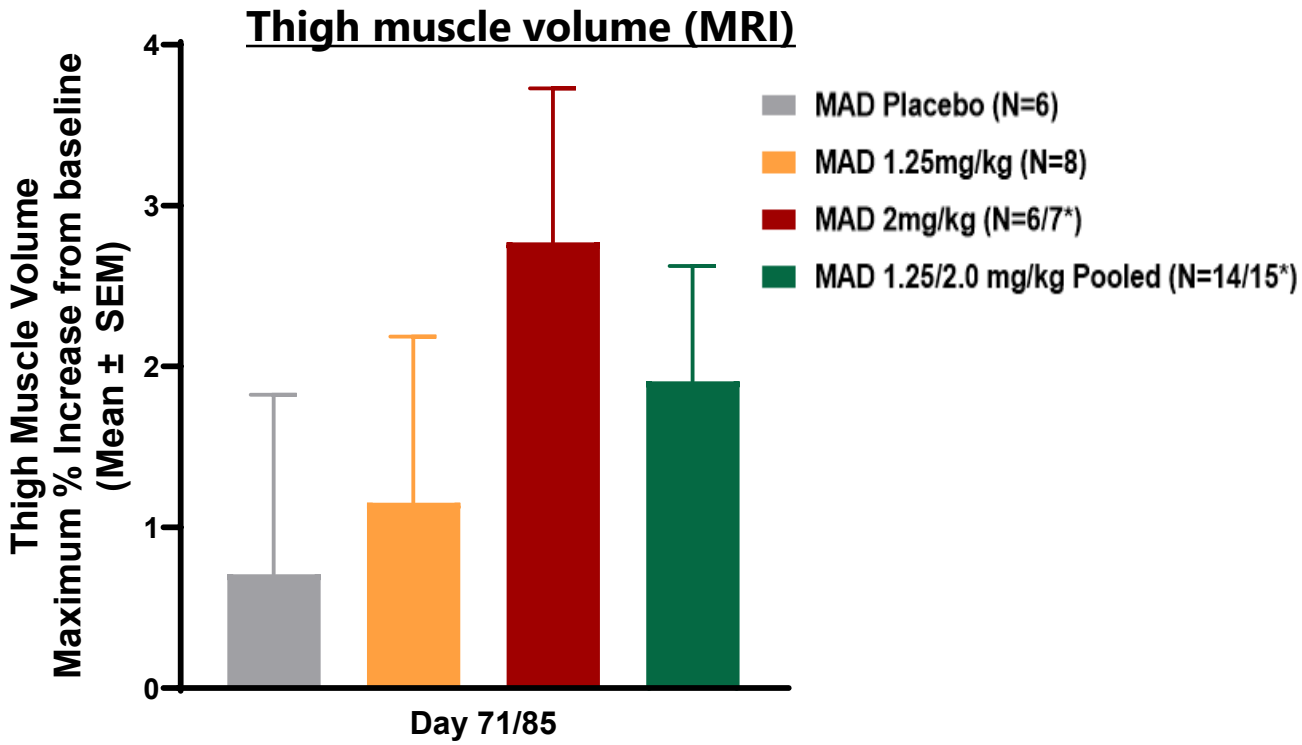


Observed changes in fat mobilization biomarkers are consistent with the observed reductions in whole body and visceral fat mass

DXA = dual-energy X-ray absorptiometry; MRI = magnetic resonance imaging
Data represented is baseline through day listed

KER-065 Administration Led to Increases in Muscle Mass





Observation of increased skeletal muscle, as demonstrated by increases in whole body lean mass (DXA) and thigh muscle volume (MRI)

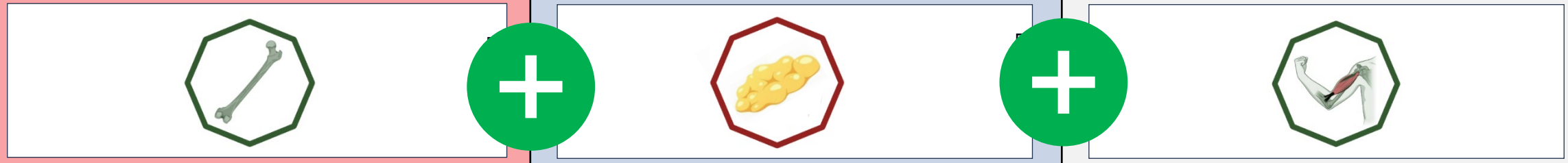
Data represented is baseline through day listed; * denotes additional participant MRI in MAD 2 mg/kg cohort

Encouraging Preclinical and Phase 1 Trial Data Support Rationale for Phase 2 Trial

- **Trial met key objectives for safety, tolerability, pharmacokinetics and pharmacodynamics**
  - Pharmacokinetic profile generally consistent with that of a well-behaved biologic
  - Dose exposure levels supportive of monthly dosing
- **KER-065 was generally well-tolerated, with no major safety signals observed as of the data cut-off date**
- **Pharmacodynamic data offer multiple lines of evidence that we may be achieving sufficient activin inhibition across tissues of interest, at drug exposures that we anticipate targeting in DMD**

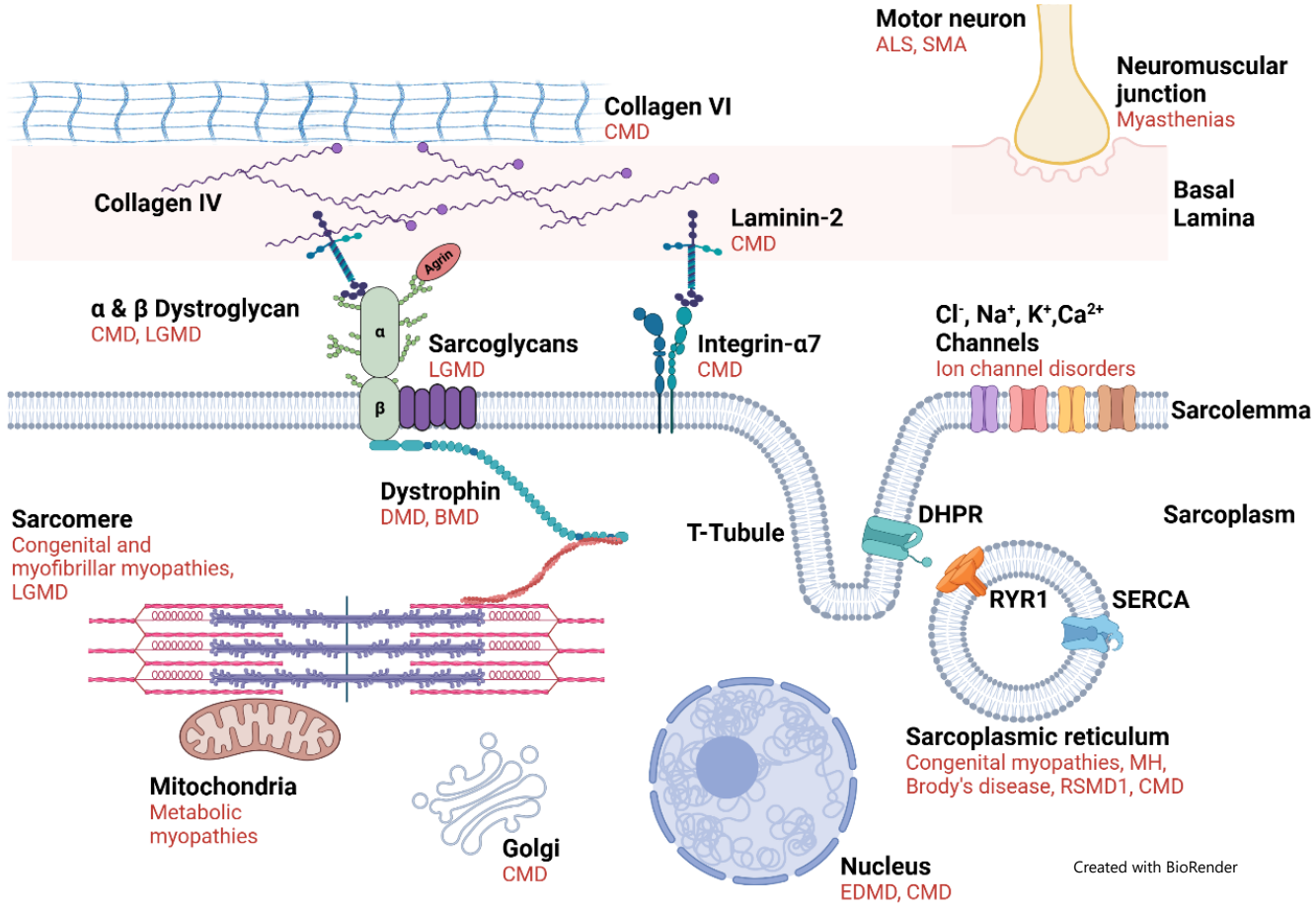
Phase 1 Data Support Potential for KER-065 to Address Multiple Aspects of DMD

Bone	Fat	Muscle
Reduced muscle strength, loss of ambulation and use of glucocorticoids in DMD contribute to the development of secondary osteoporosis **KER-065 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	Decreased mobility and the use of glucocorticoids are associated with increased risk of obesity and related negative health consequences **KER-065 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	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 **KER-065 elicited:** • Increased lean muscle mass • Increased thigh muscle volume



Therapeutic Potential in 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 motoneuron 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 motoneuron 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 KER-065 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

Next Steps for KER-065

- **We plan to engage regulators on the design of a Phase 2 clinical trial evaluating KER-065 in patients with DMD in the third quarter of 2025**
- **Subject to regulatory feedback, we plan to initiate this Phase 2 clinical trial in the first quarter of 2026**

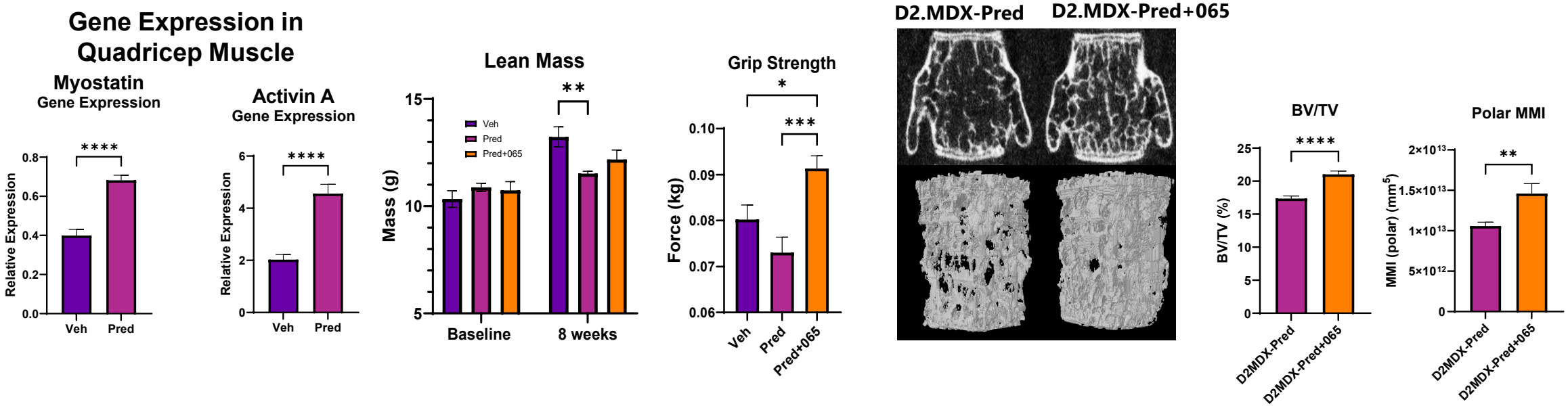
KEROS THERAPEUTICS

Q&A

KEROS THERAPEUTICS

Appendix

Glucocorticoids Increased Expression of Negative Regulators of Skeletal Muscle and Bone in a Preclinical Study

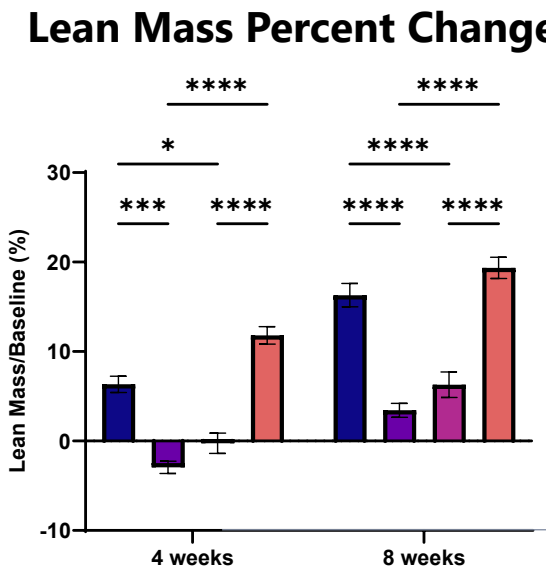


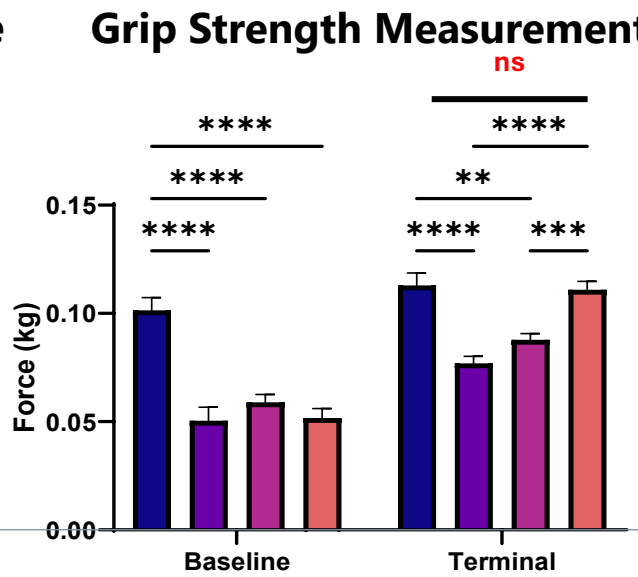
MDX mice (mouse model of DMD) were treated with vehicle (Veh) or 2-prednisolone (Pred), or cotreated with prednisolone and RKER-065 (Pred-065) (10 mg/kg, twice weekly). The changes in body weight, body composition (NMR), grip strength, skeletal muscle gene expression, and bone micro-architecture (micro-CT) were assessed.

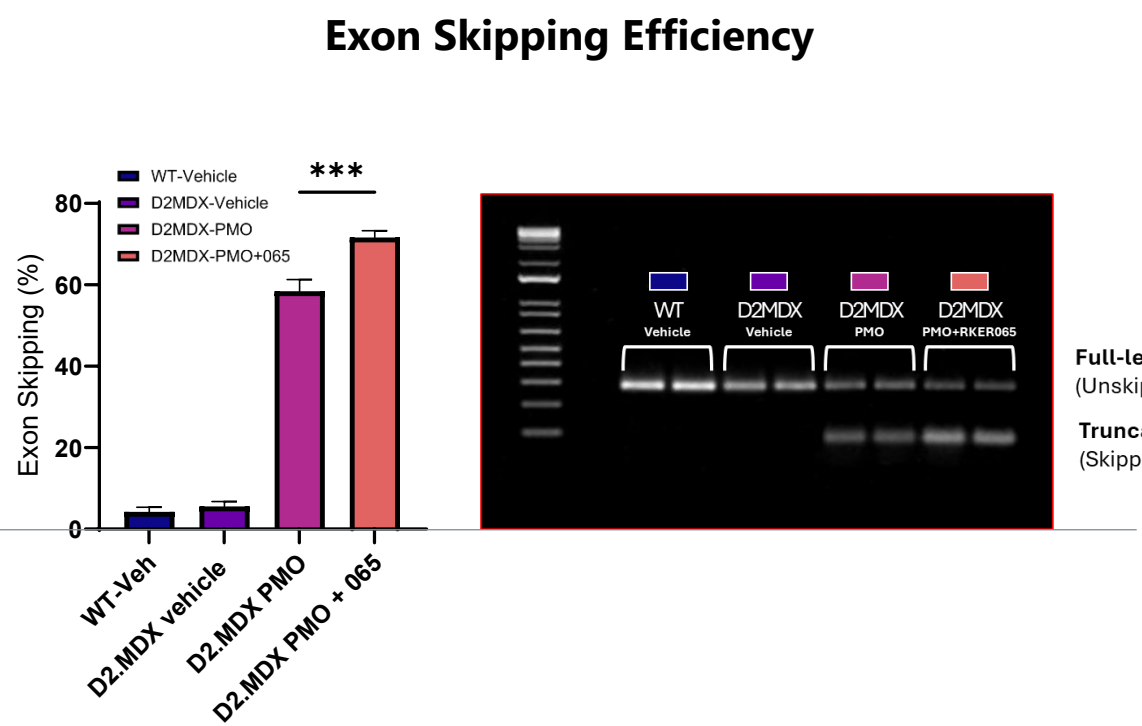
- **Prednisolone-treated MDX mice had less muscle mass and strength than vehicle-treated mice**
- **Co-treatment with prednisolone and RKER-065 increased both muscle mass and strength and trabecular bone and strength**

Data is shown as average ± SEM. * P≤0.05, ** P<0.01, *** P<0.001, and **** P< 0.0001. BV/TV = bone volume fraction; MMI = mass moment of inertia; RKER-065 = research version of KER-065

RKER-065 Treatment Improved Efficiency of Exon Skipping





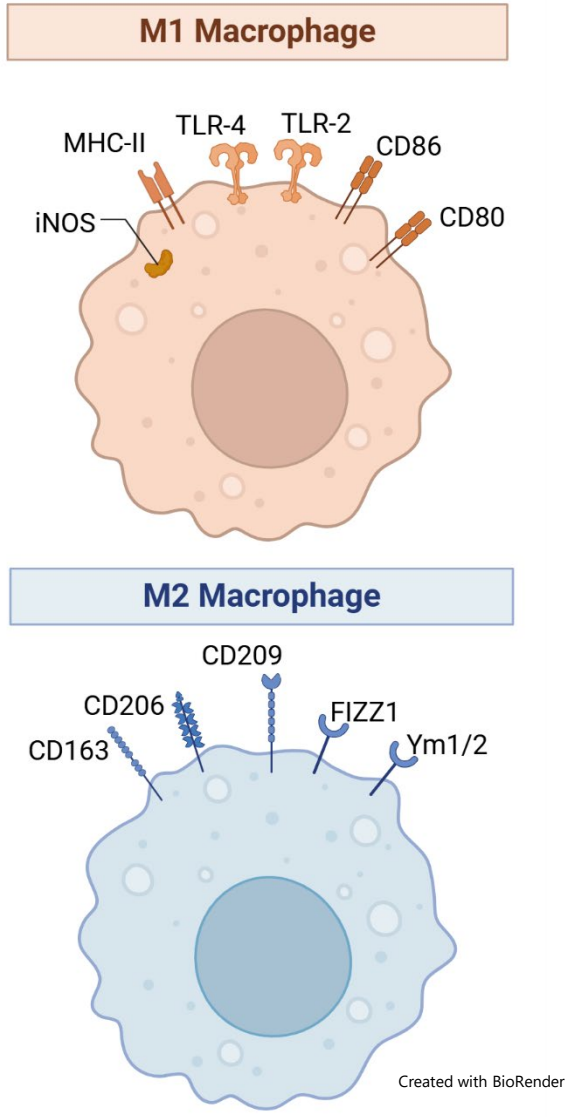


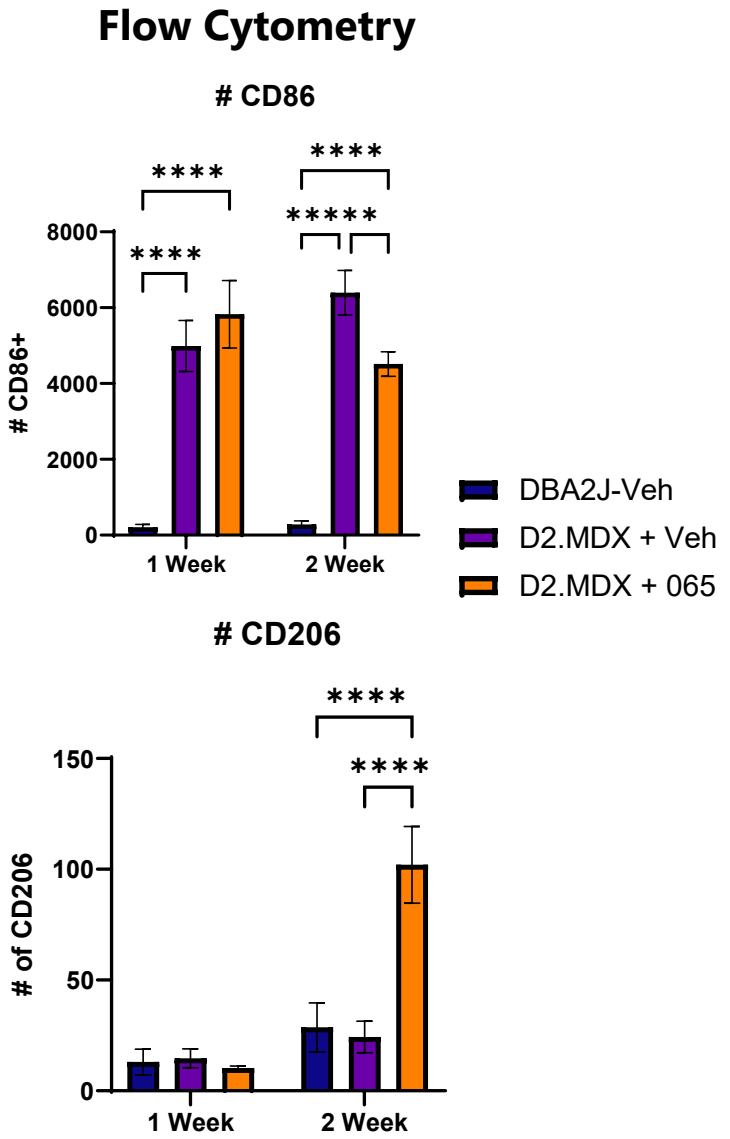
- **Treatment with PMO did not increase lean mass or muscle function**
- **Co-treatment with PMO and RKER-065 improved lean mass and grip strength**
- **RKER-065 treatment improved the efficiency of PMO driven exon skipping**

St. Pierre, M., et al. 2024 New Directions in Biology and Disease of Skeletal Muscle Conference; ns = not significant; P value: *<0.05, **<0.01, *** <0.001, **** <0.0001
PMO = Phosphorodiamidate morpholino oligomer

RKER-065 Reduced the Inflammatory Profile of Muscle Resident Macrophages and Shifted Towards Muscle Repairing

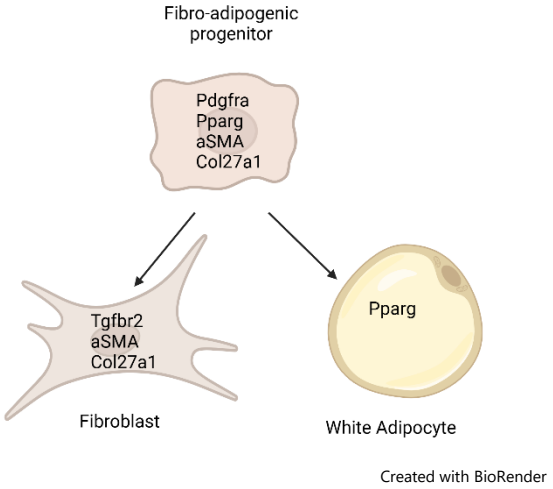
- **Under different pathophysiologic conditions, macrophages can acquire distinct functional phenotypes via undergoing different phenotypic polarization. Macrophage M1 and M2-type responses describe the opposing activities of killing or repairing**
- MDX mice were treated with a single dose of RKER-065 (10 mg/kg) or vehicle. Muscles were dissected and processed to obtain single cell suspensions on day 1, day 2, and day 4 (n=5), stained for markers of macrophage markers and analyzed by flow cytometry
- **Treatment with RKER-065 reduced the markers associated with pro-inflammatory macrophages (M1)**
- **Treatment increased markers associated with repairing macrophages (M2)**

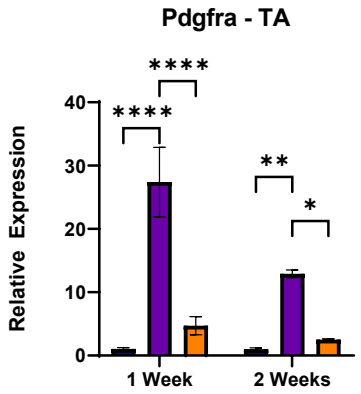


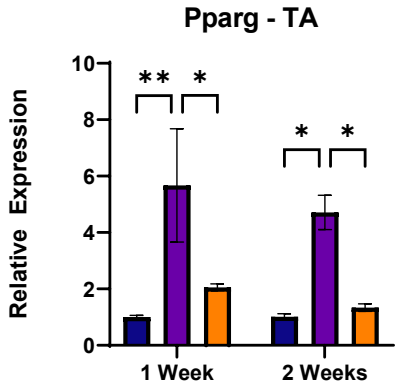


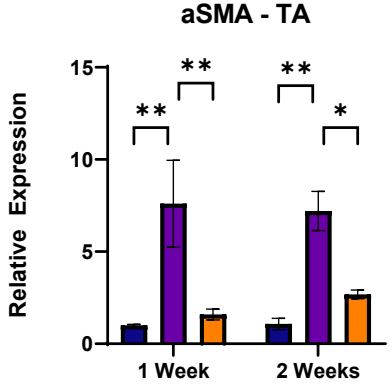
Data is shown as average ± SEM. 2-way ANOVA with repeat measures and Sidak post test. * P≤0.05, ** P<0.01, *** P<0.001, and **** P< 0.0001.

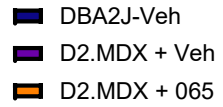
RKER-065 Reduced the Fibroblast and Fat Precursor Cells in Muscle of Dystrophic Mice



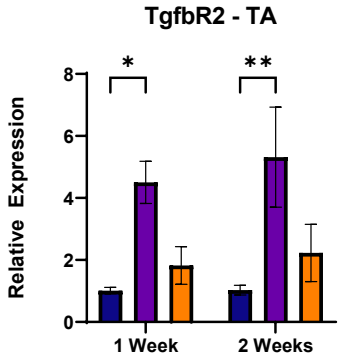


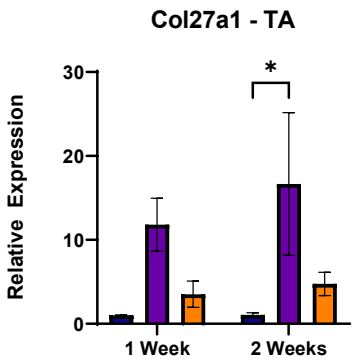






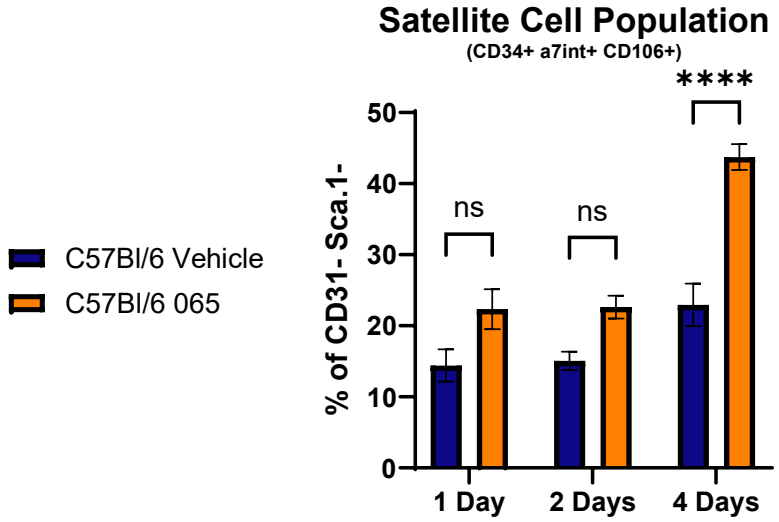
- **Failure of muscle to regenerate following injury leads to replacement of muscle fibers with fibrotic and fatty infiltrates**
- MDX mice were treated with a single dose of RKER-065 (10 mg/kg) or vehicle. Muscles were dissected and RNA isolated and analyzed by real-time QPCR
- **RKER-065 treatment reduced fibro-adipogenic progenitors, the common cell that differentiates to fibroblasts and adipocytes**



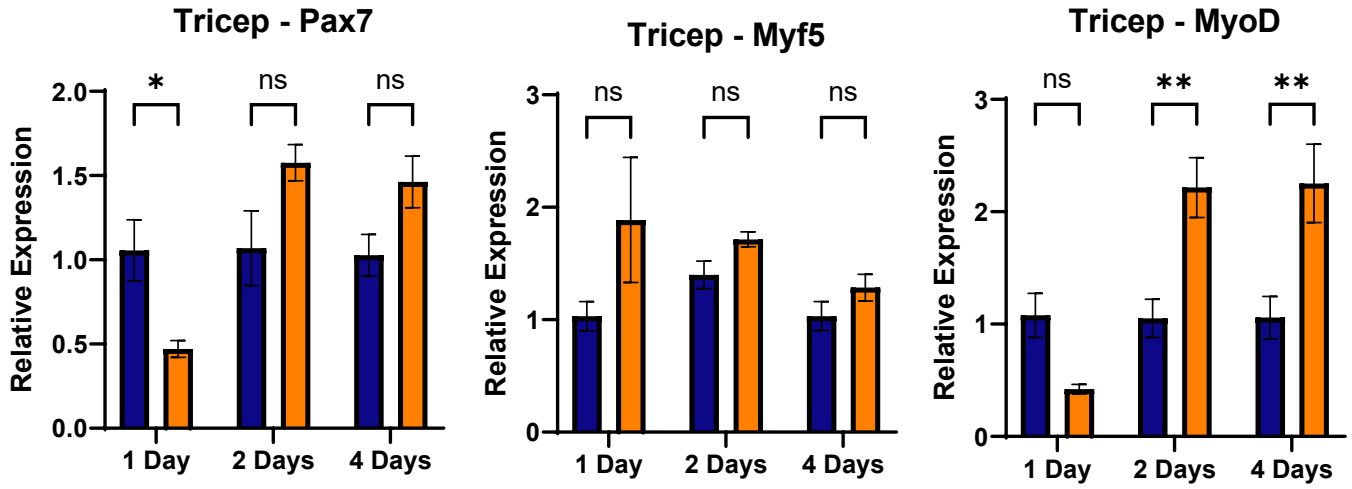


Data is shown as average ± SEM. 2-way ANOVA with repeat measures and Sidak post test. * P≤0.05, ** P<0.01, *** P<0.001, and **** P< 0.0001. TA = tibialis anterior

Treatment with RKER-065 Increased Satellite Cells in Skeletal Muscle

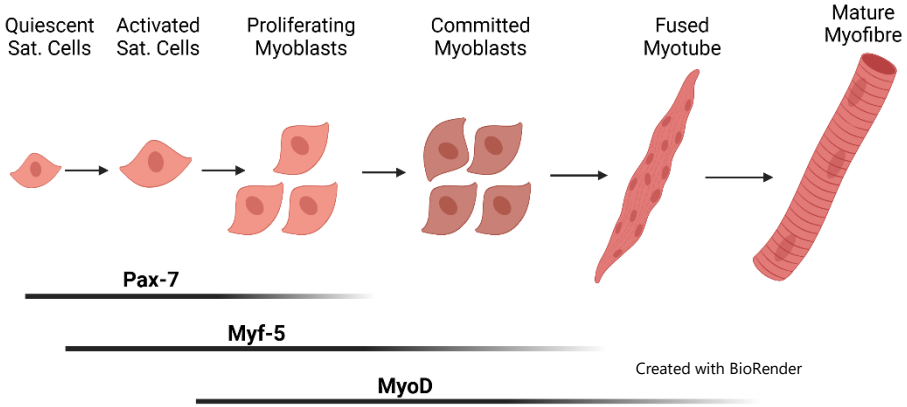


Markers of satellite cell differentiation



- Wild type mice were treated with a single dose of RKER-065 (10 mg/kg) or vehicle. Muscles were dissected and processed to obtain single cell suspensions on day 1, day 2, and day 4 (n=5, stained for markers of satellite cells (CD31, Sca.1, CD34, α7 integrin, and CD106) and analyzed by flow cytometry
- **Treatment with RKER-065 increased the pool of satellite cells in wild type mice**
- **Molecular markers demonstrated commitment/differentiation of satellite cells to muscle**

Flow Cytometry of satellite cells



* P≤0.05, ** P<0.01, *** P<0.005; Pax7 = paired box 7; Myf5 = myogenic factor 5 ; MyoD = myoblast determination protein 1

Treatment with RKER-065 Increased Utrophin Expression and Muscle Strength in Mouse Model of DMD

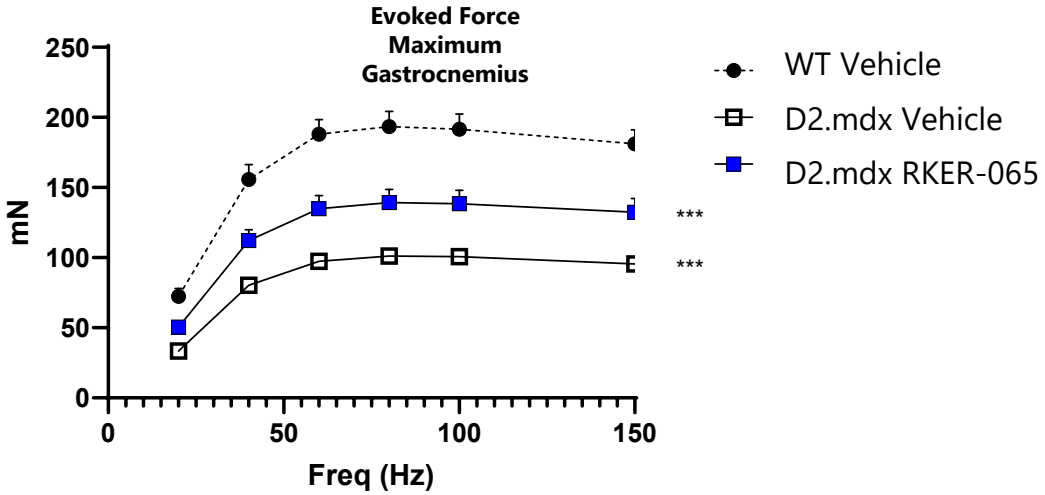
Muscle lacking dystrophin is easily damaged during the process of contraction

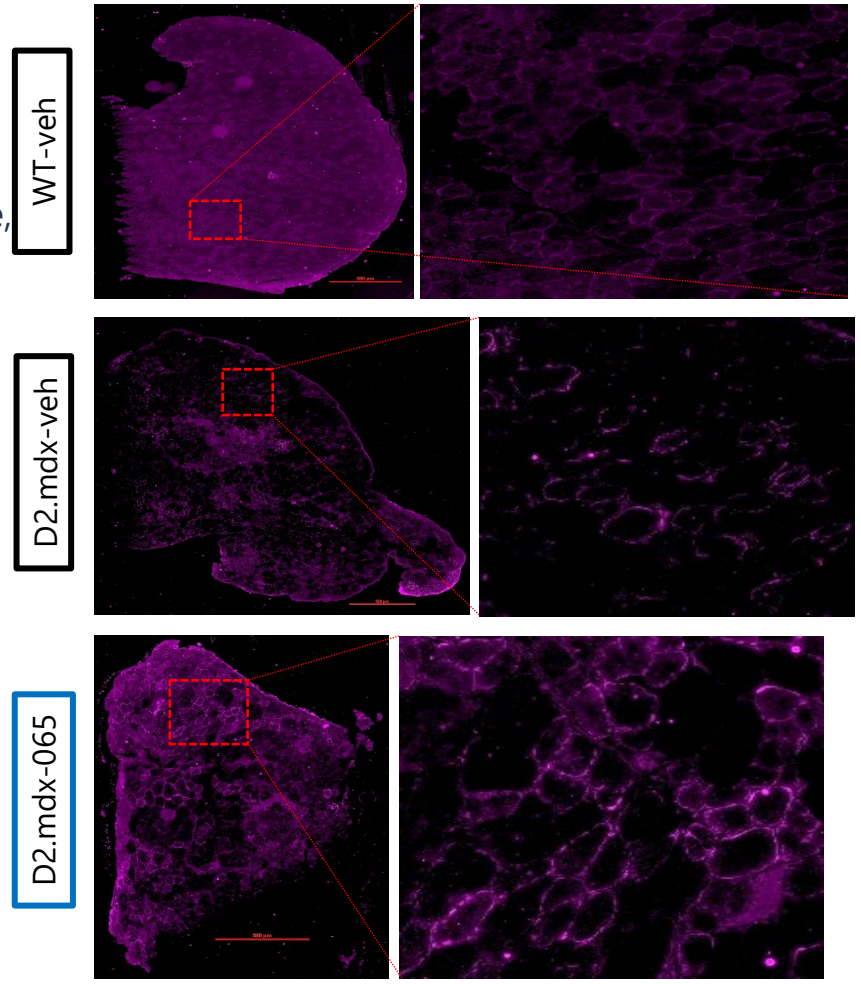
Many third-party approaches have been utilized to stabilize the muscle and provide resistance to contractile-induced damage:

- Antisense oligonucleotides to trigger exon skipping, restore the mRNA reading frame, and allow production of a truncated dystrophin protein
- Gene therapy with mini and micro dystrophin
- Increased expression of utrophin (a functional analog of dystrophin)

Treatment with RKER-065 in a mouse model of DMD led to:

- Increased expression of utrophin in muscle fibers, potentially contributing to the observed increased strength[1]





1. Nathan, R., et al. 28th International Annual Congress of the World Muscle Society; WT= wild type (control), D2.mdx = mouse model of DMD' *** P<0.001