

SAT-3247 KOL Event

*Unmet Need & Current Treatment Landscape
for Duchenne Muscular Dystrophy*

Feb. 24, 2026

satellos

REGENERATING MUSCLE FROM WITHIN™

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Agenda



Welcome

Frank Gleeson *Co-founder & Chief Executive Officer, Satellos*



Duchenne and the Need for Additional Therapeutic Approaches

Dr. Kevin M. Flanigan *Wolfe Foundation Endowed Chair in Neuromuscular Research, Nationwide Children's Hospital; Professor of Pediatrics & Neurology, Ohio State University College of Medicine*



The Science Behind SAT-3247

Dr. Michael Rudnicki *Co-founder & Chief Discovery Officer, Satellos; Senior Scientist & Director, Sprott Centre for Stem Cell Research, Ottawa Hospital Research Institute*



SAT-3247 Clinical Development Overview

Dr. Wildon Farwell *Chief Medical Officer, Satellos*

Q&A

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Duchenne Muscular Dystrophy and the Need for Additional Therapeutic Approaches

Kevin M. Flanigan, MD

Director, Center for Gene Therapy

Robert F. & Edgar T. Wolfe Foundation Endowed Chair in Neuromuscular Research

Director, NCH Wellstone Muscular Dystrophy Specialized Research Center

Nationwide Children's Hospital

Professor of Pediatrics and Neurology

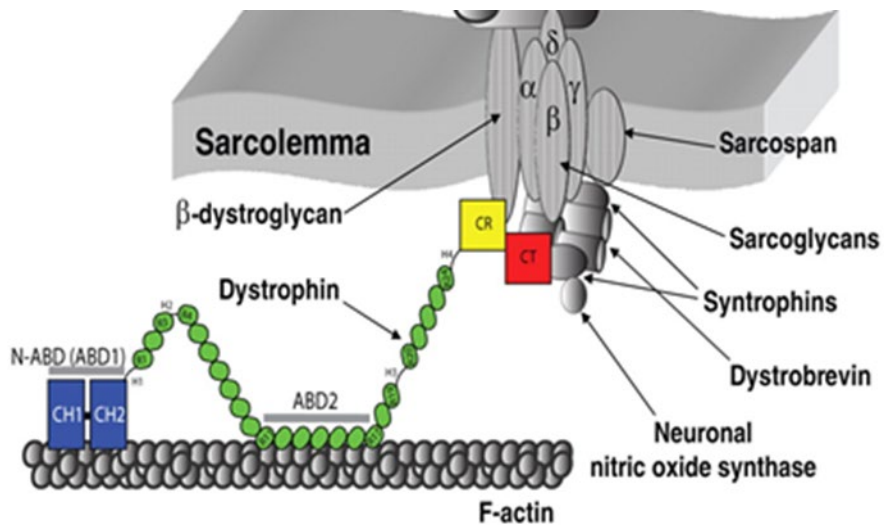
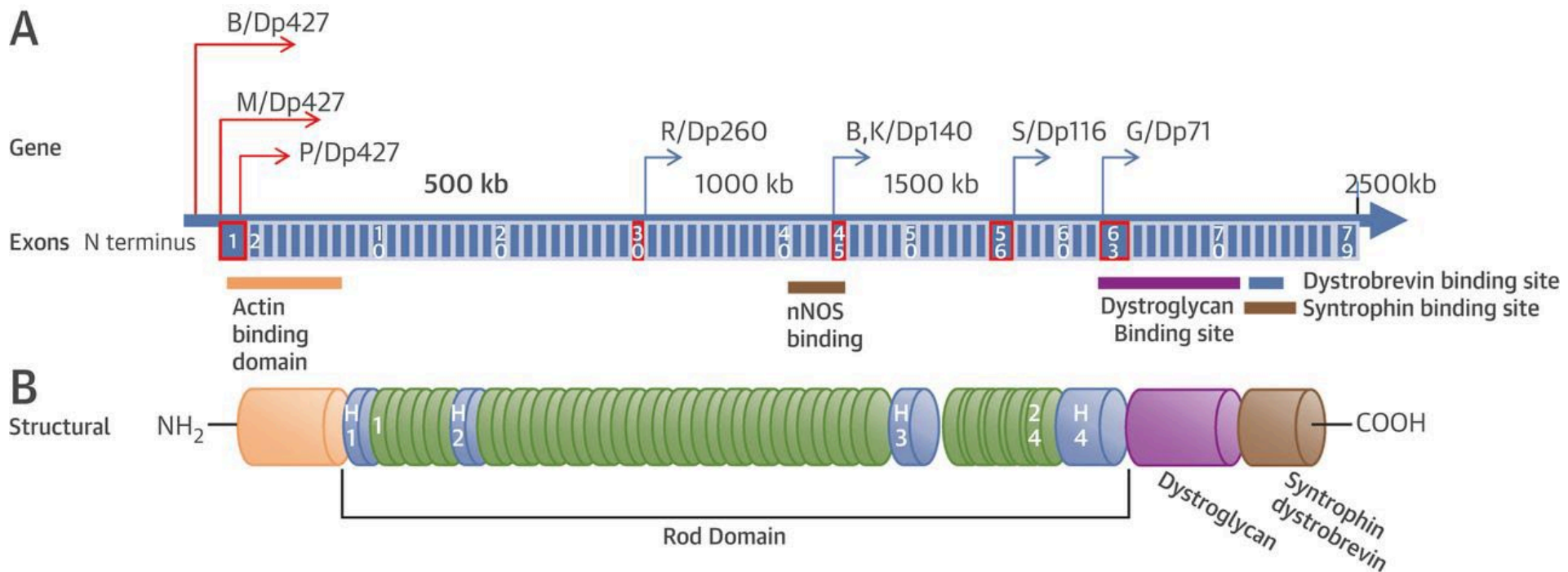
The Ohio State University



Financial Disclosure

- Clinical trial support: Sarepta, Dyne, Avidity, Ultragenyx, Solid
- Advisory boards: Armatus, Encoded, Insmmed, Dyne, Solid, Precision Biosciences
- Past Royalties: stellas

The Dystrophin Gene (A) and Protein (B)



Dystrophinopathies: Clinical Categorization

- DMD:
 - Onset age 3-5
 - Pelvic girdle weakness
 - Tight heel cords
 - CK 50-100X normal
 - Loss of ambulation by age 12
- BMD:
 - Classic definition: loss of ambulation > age 12
 - Alternatively:
 - “intermediate muscular dystrophy” for loss of ambulation ages 12 through 15
 - BMD for loss of ambulation > age 15
 - Limb-girdle syndromes in adulthood
 - Myalgias
 - Isolated cardiomyopathy

Dystrophinopathies are a continuum



Dystrophin Mutations

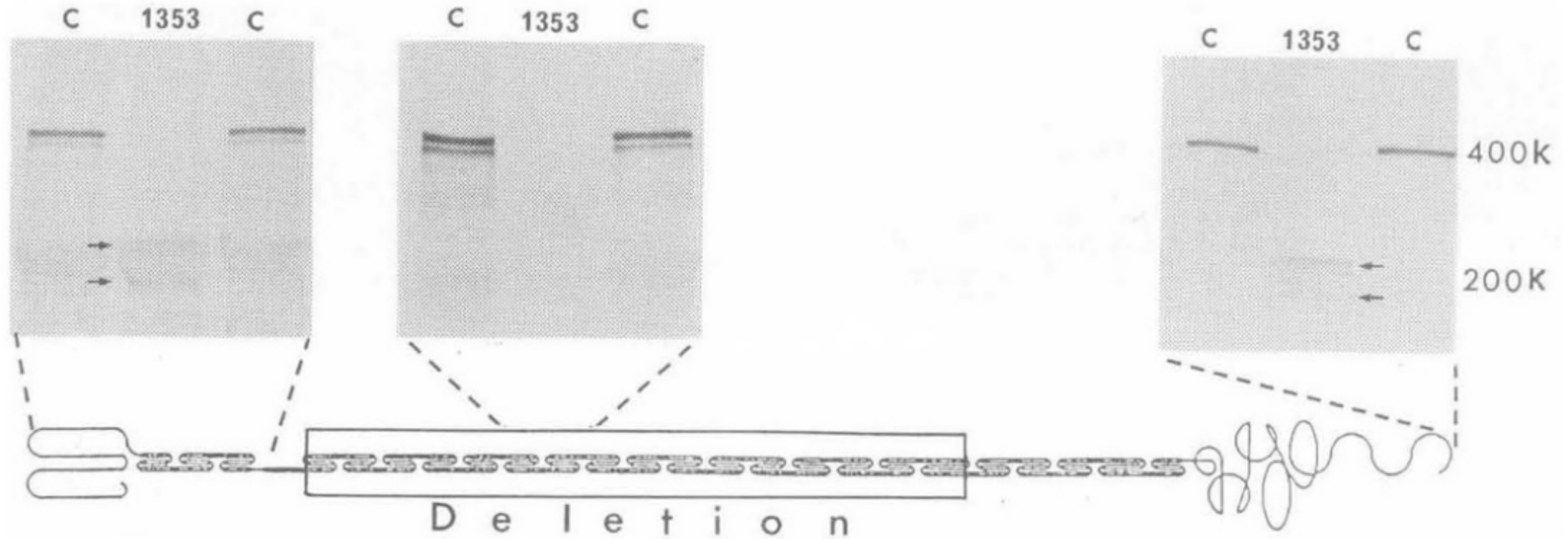
- Dystrophin gene (*DMD*) on Xp21.1 is huge:
 - 2.4 million base pairs
 - 79 exons and 8 promoters
- Large deletions (≥ 1 exon) account for ~65% of DMD/BMD patients
- ~5% have duplications
- ~15% of boys have nonsense mutations
- Remainder are frameshifting insertions/deletions, splice site mutations, missense mutations

Dystrophin Mutations and the Reading Frame Rule: Duchenne vs Becker

The reading frame rule:

- Mutations that maintain an open mRNA reading frame (“in-frame mutations”) will result in translation of a protein with partial function (Becker)
- Those that ablate the reading frame (“out-of-frame”) will result in no dystrophin protein and a Duchenne phenotype
- This is the basis of exon-skipping therapies and microdystrophin gene delivery

Microdystrophin: Derived from Clinical Observation

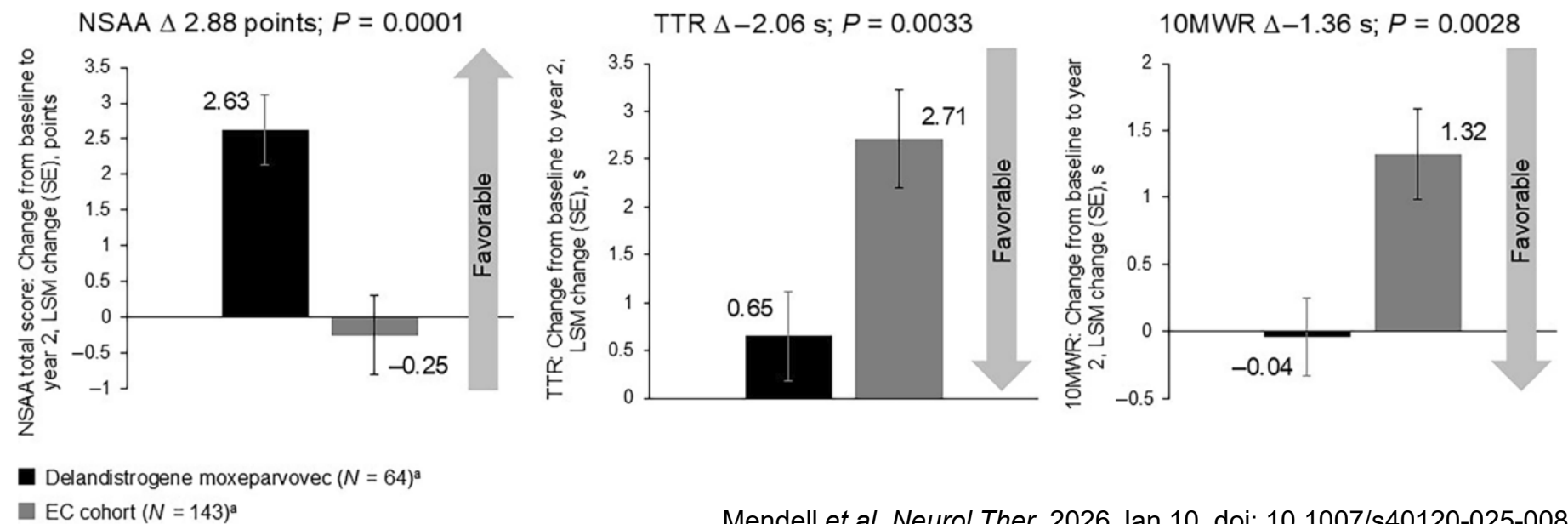
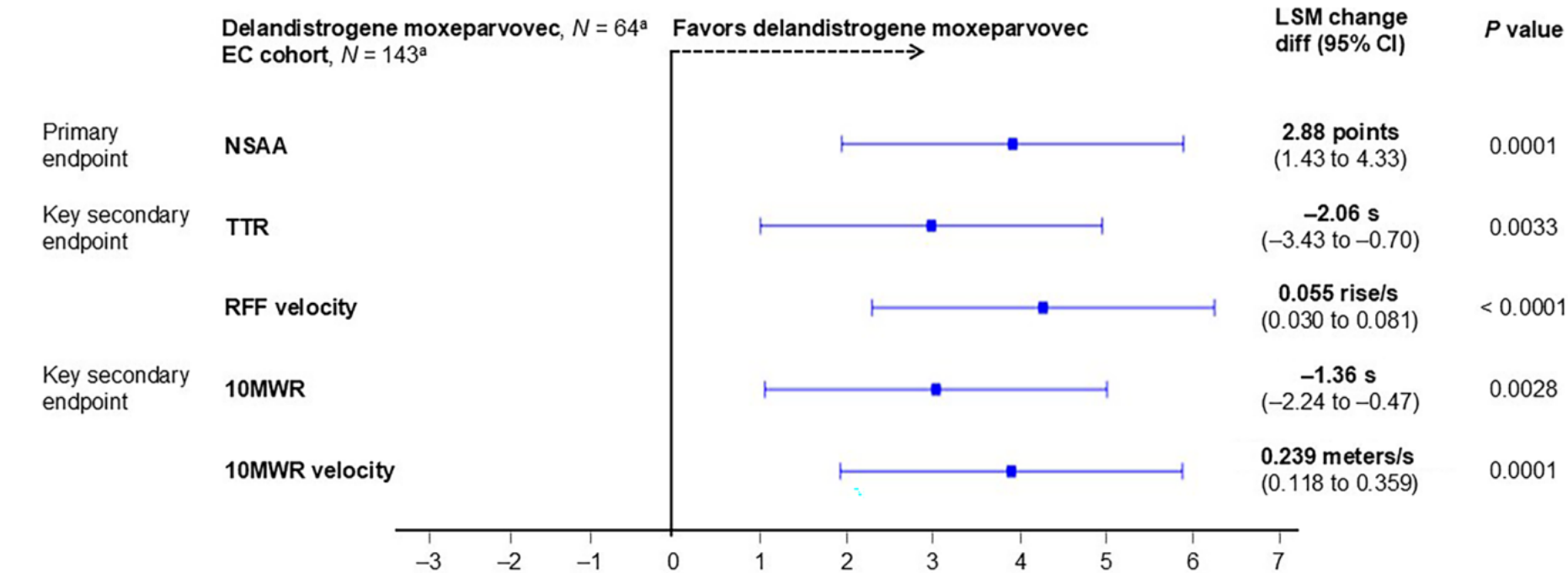


- Deletion of 46% of protein (exons 17-48)
- Ambulant to at least age 61

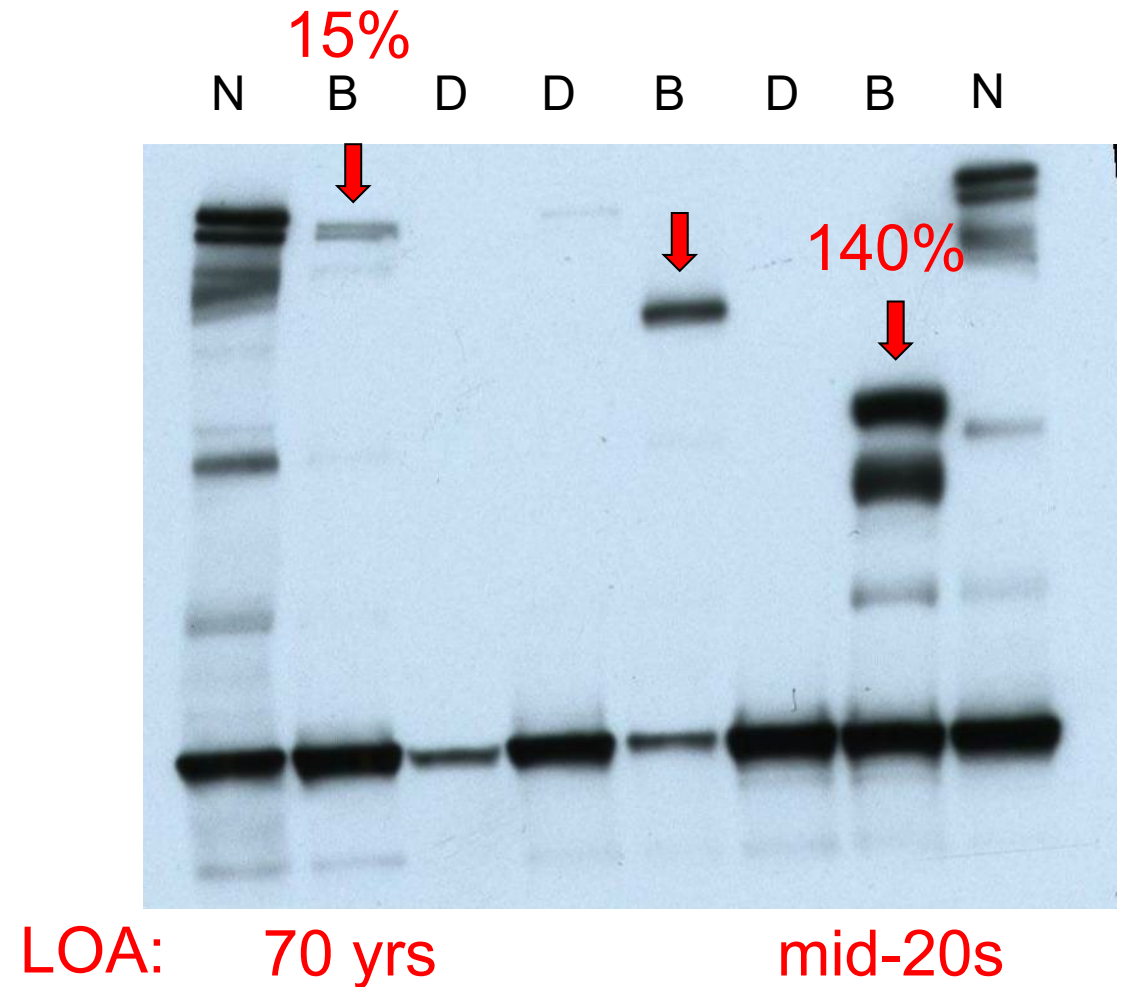
England *et al*, *Nature* 343; 180–182 (1990)

Davies and Vogt, *Neuromuscul Disord* 2024 Jun;39:5-9.

Delandistrogene
moxeparvovec:
Improvements in
function at 2 years
post-treatment
(EMBARK trial)

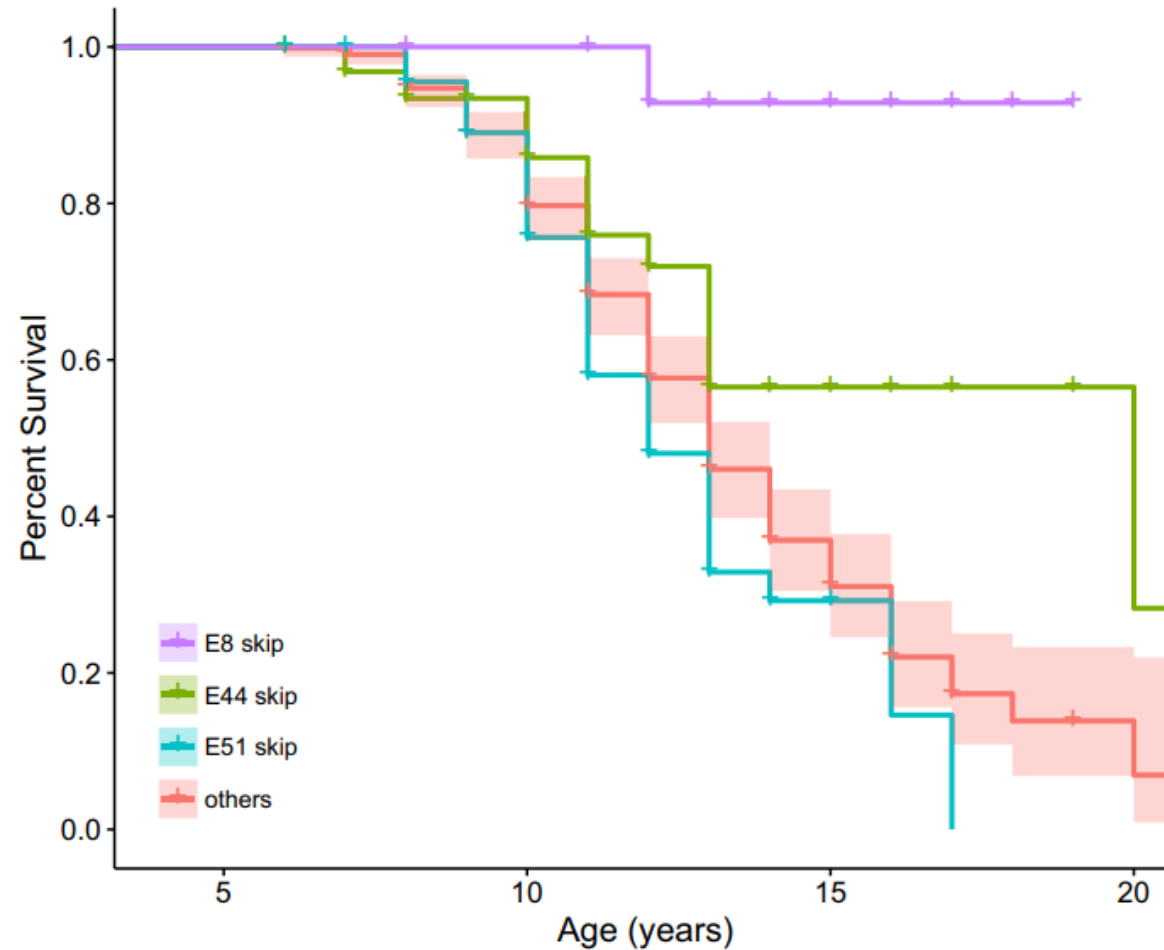


Not all BMD-associated dystrophin proteins will have the same functional outcome.



- Amount of internally-deleted dystrophin alone is insufficient to predict outcome.

Not All Boys with Out-of-frame Mutations Will Have the Same Outcomes



What Can Account for These Differences?

- Multiple pathways of pathogenesis

And therefore

- Multiple potential genes relevant as potential modifiers of disease

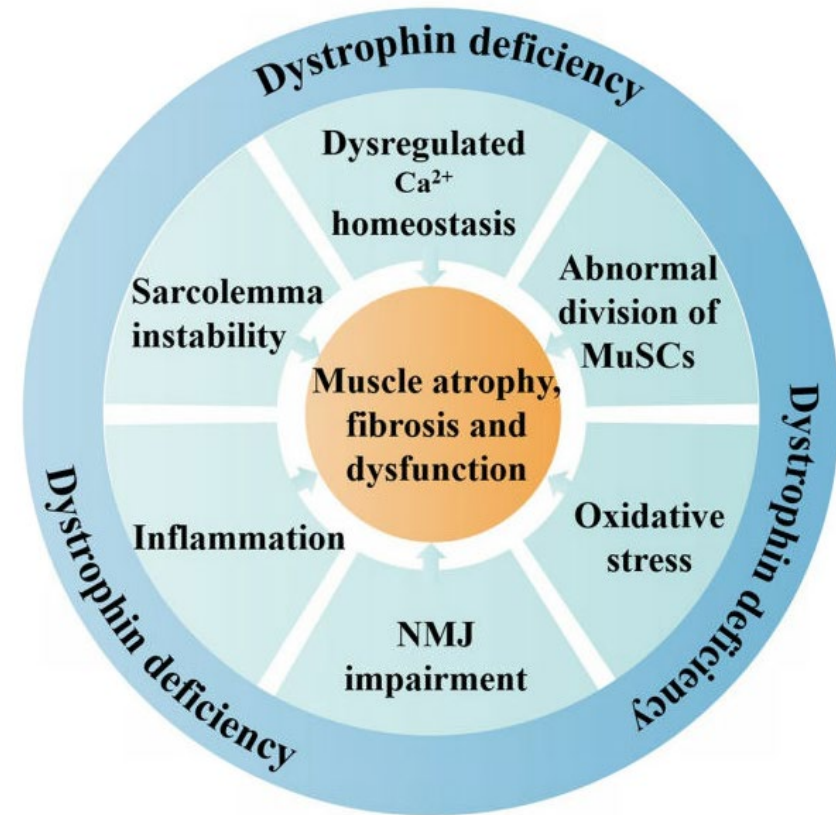
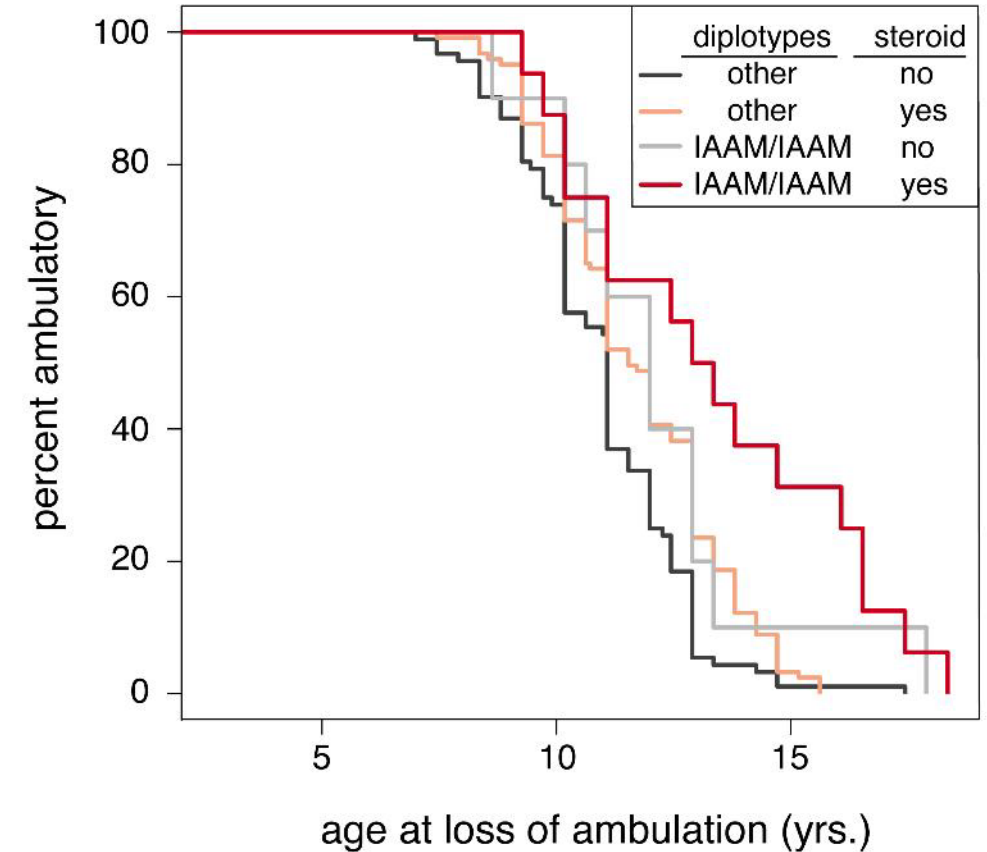
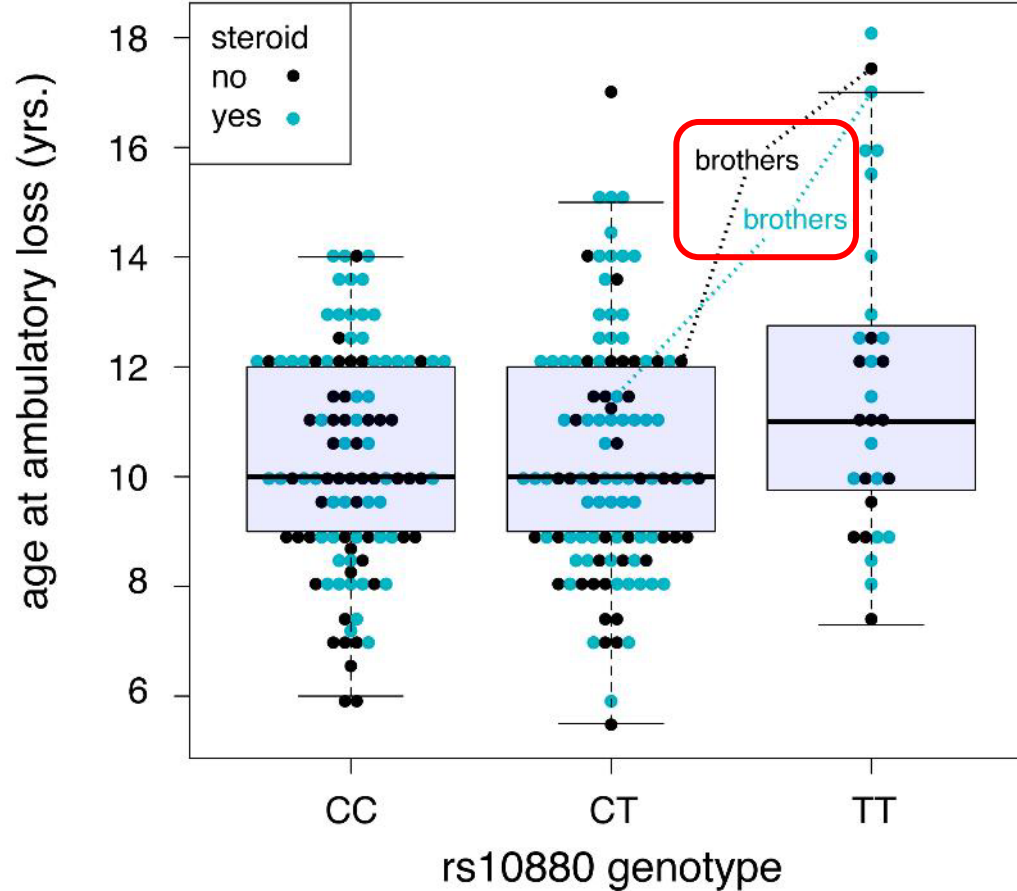


Fig. 2 Pathogenesis of Duchenne muscular dystrophy. *NMJ* neuro-muscular junction, *MuSCs* muscle satellite cells

One Example: There is a Protective Genotype Associated With the *LTBP4* Gene



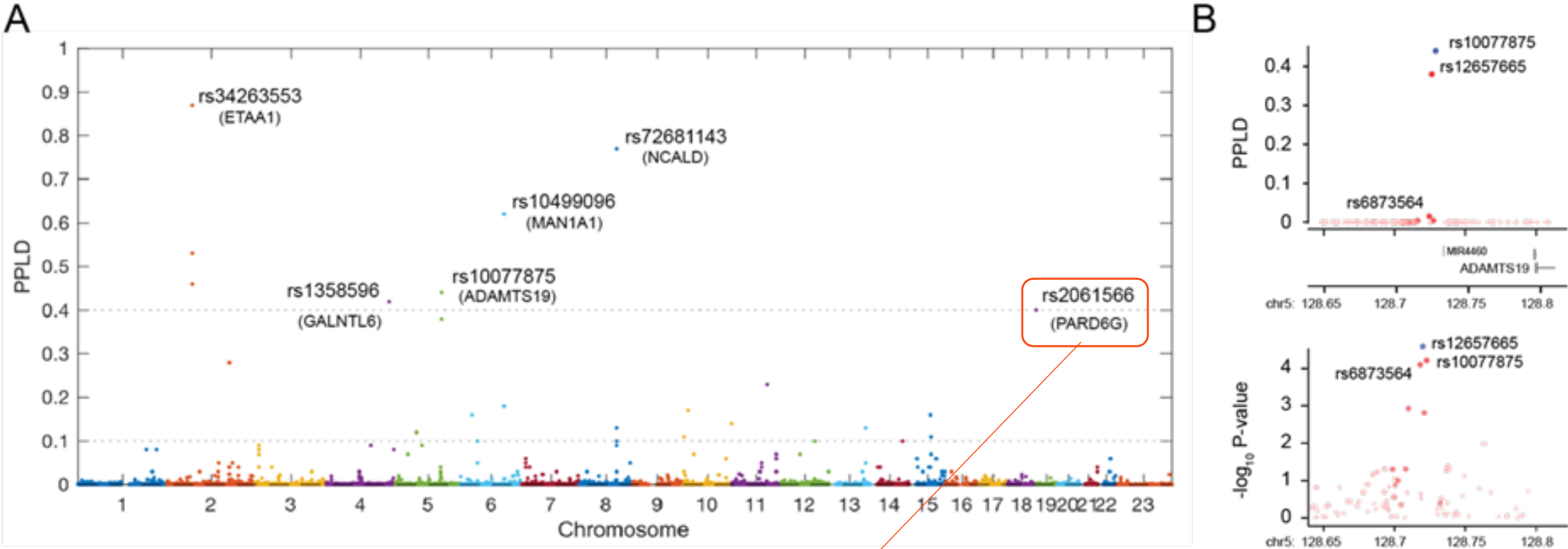
- May account for differences between brothers

Mean age of ambulatory loss for “IAAM” versus other haplotypes:

- In steroid-treated patients: 12.5 ± 3.3 years vs. 10.7 ± 2.1 years
- In steroid-naïve patients: 11.2 ± 2.7 vs. 9.8 ± 2.0 years

A genome-wide association analysis of loss of ambulation in dystrophinopathy patients suggests multiple candidate modifiers of disease severity

Kevin M. Flanigan^{1,2,3}, Megan A. Waldrop^{1,2,3}, Paul T. Martin^{1,2}, Roxane Alles¹, Diane M. Dunn⁴, Lindsay N. Alfano^{1,2}, Tabatha R. Simmons¹, Melissa Moore-Clingenpeel^{5,6}, John Burian⁵, Sang-Cheol Seok⁵, Robert B. Weiss^{4,8} and Veronica J. Vieland^{2,5,6,7,8}

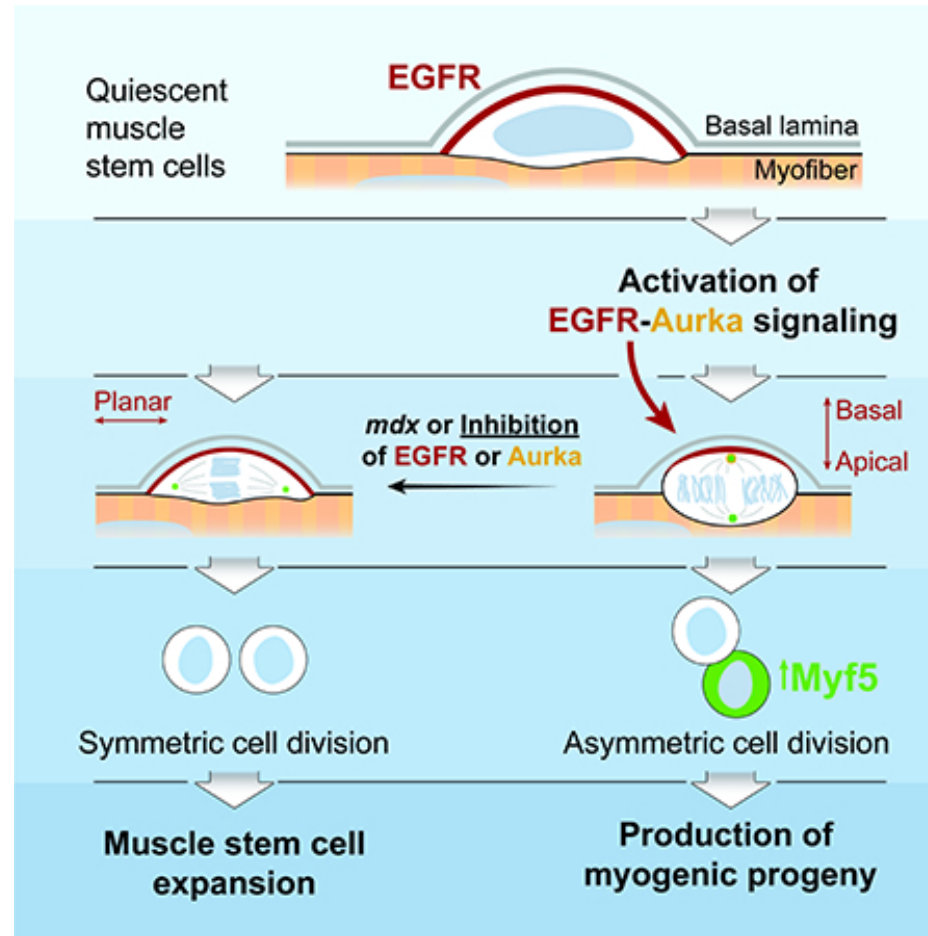
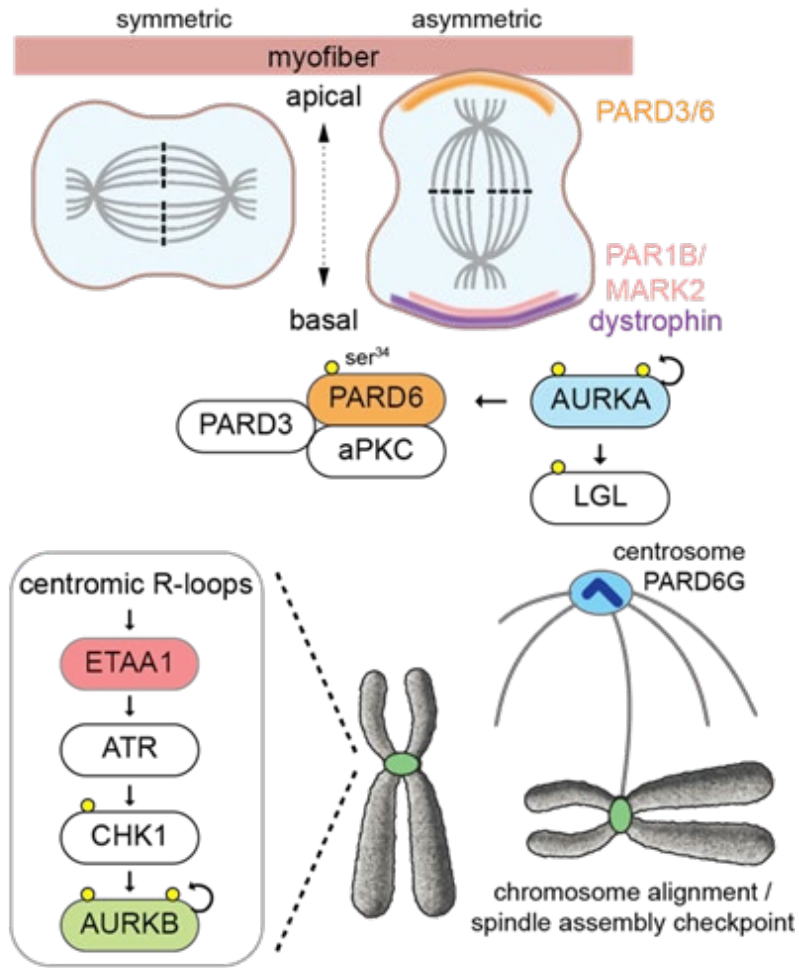


The rs2061566 SNP alters an enhancer linked to *PAR6G*

Six Novel Candidate Gene Modifiers

Chromosome	Gene	Function	Plausible role
2	<i>ETAA1</i>	activating protein for ATR	maintaining genome integrity and chromosomal stability of the muscle stem cell population
4	<i>GALNTL6</i>	O-GalNAc transferase	may dictate a switch between a propensity of muscles to be predisposed to endurance versus strength training.
5	<i>ADAMTS19</i>	protease	involved in extracellular matrix formation, interactions, and remodeling
6	<i>MAN1A1</i>	mannosidase	may (by altering N-linked sialic acid) ameliorate muscular dystrophy and alter muscle regeneration and muscle mitochondria and stem cell function via KLOTHO dependent pathway
8	<i>NCALD</i>	myristoylated calcium sensor	may regulate an alternate cGMP pathway and impact downstream signaling cascades disrupted by the loss of NO-activated cGMP synthesis
18	<i>PARD6G</i>	member of the PAR polarity complex	may affect muscle satellite cell polarity and regeneration

PARD6 is Implicated in Asymmetric Satellite Cell Division



Promotion of asymmetric cell division and production of myogenic cells = **Objective of SAT-3247**

Where Does DMD Therapy Stand Today?

- Corticosteroids and related steroids
 - Provide benefit in sustaining ambulation, with significant side effects
- AAV-delivered microdystrophin gene therapy
 - One is approved and others are in trials, but not all boys can receive it
 - AAV antibody positivity
 - Specific genotypes are excluded
 - We do not yet know durability of effect
 - Safety in older boys/men is not yet established
- Exon skipping
 - Currently approved drugs show limited benefit
 - Second-generation exon skipping drugs are quite promising, but a limited number of genotypes will be treatable with them

But none of these address regeneration of muscle.

Ongoing Therapeutic Needs in DMD:

Therapies that:

- Address other pathways of pathogenesis, including inflammation
- Improve regenerative capacity of or reduce histopathology of muscle
- Are suitable as add-on or “cocktail” therapies following dystrophin (or microdystrophin) restoration

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Muscle Regeneration Primer



Skeletal Muscle is a Highly Regenerative Tissue



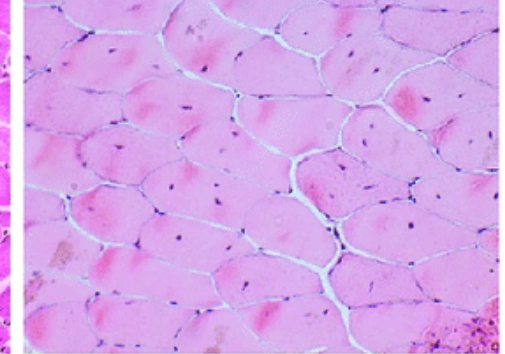
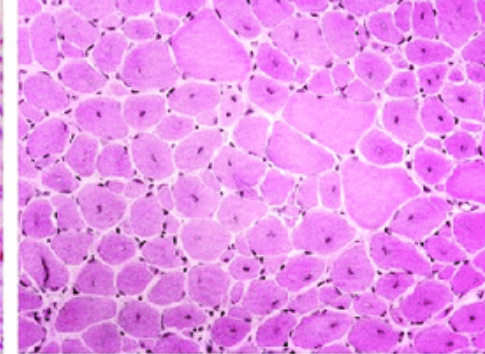
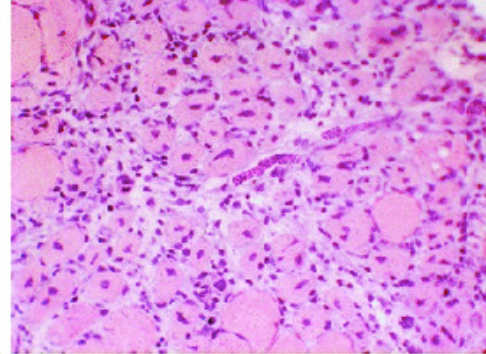
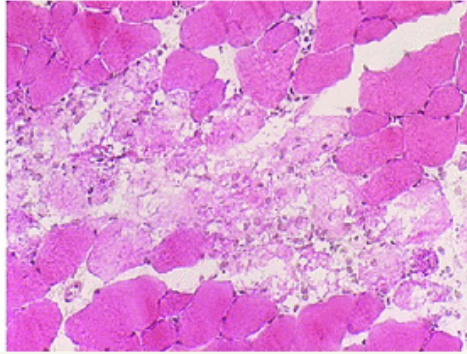
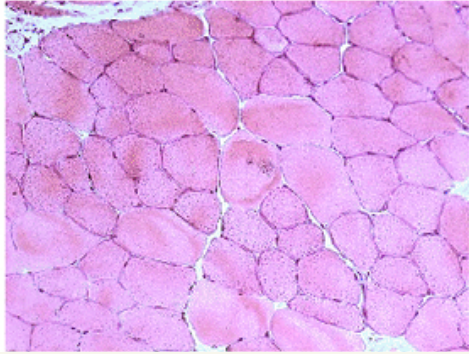
Healthy muscle

Injury (toxin)

Cell proliferation

Cell maturation

Repaired muscle

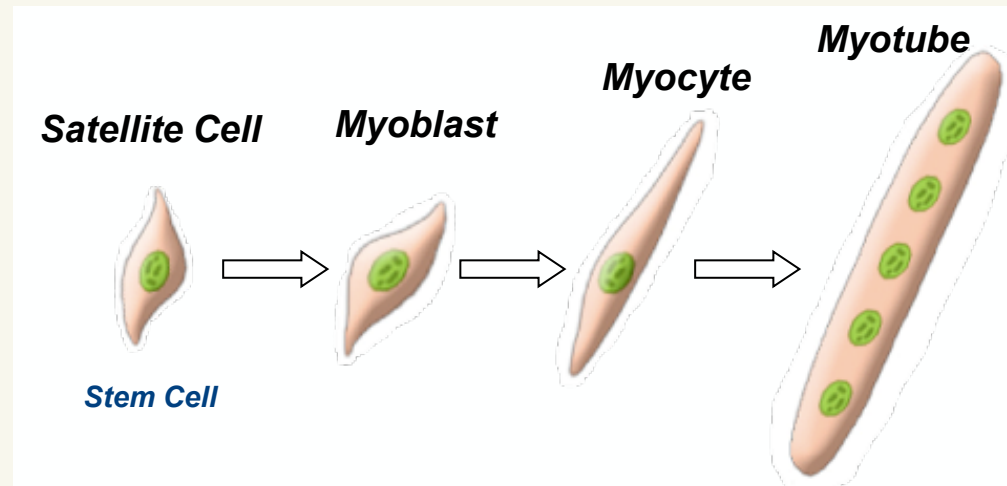


Day 0

Day 5

Day 10

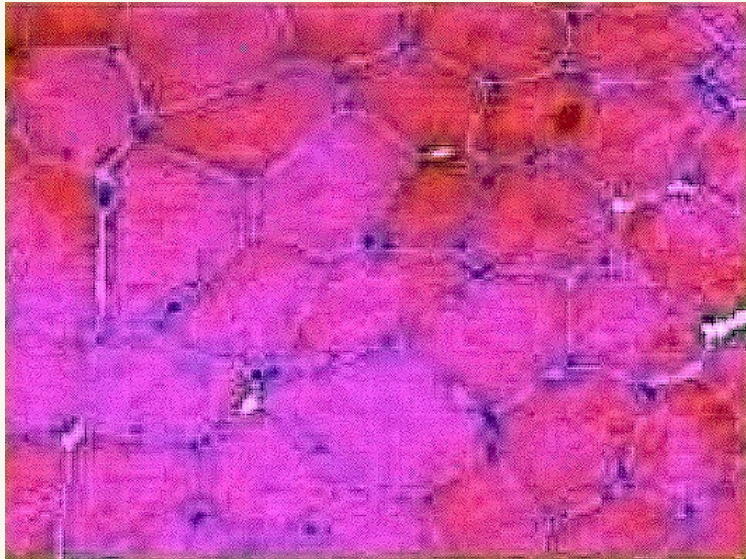
Day 20



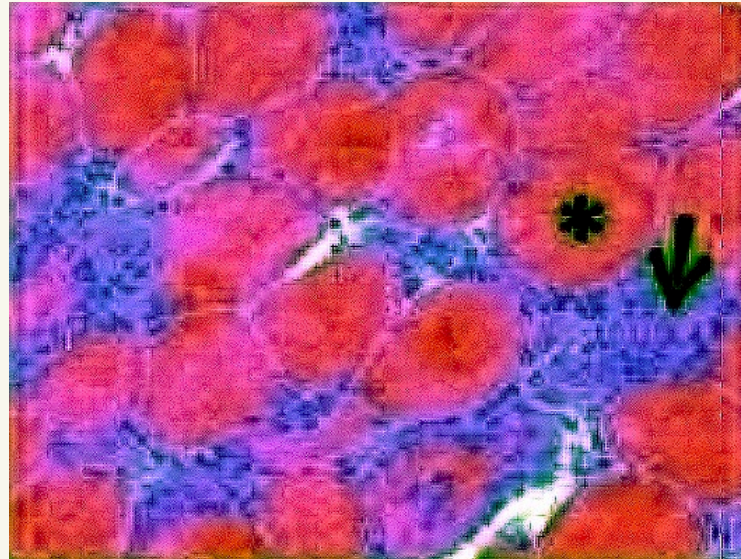
Skeletal Muscle is a Highly Regenerative Tissue in People!



Healthy muscle

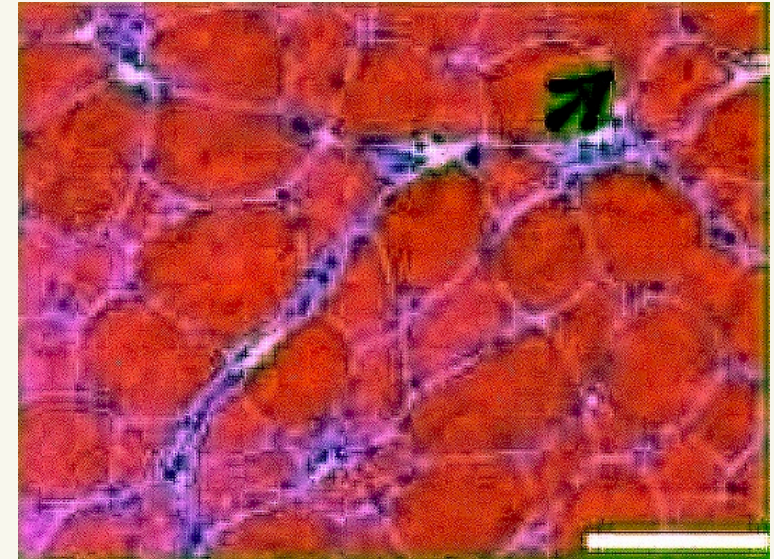


Electrically stimulated injury



7 days post injury

Repaired muscle



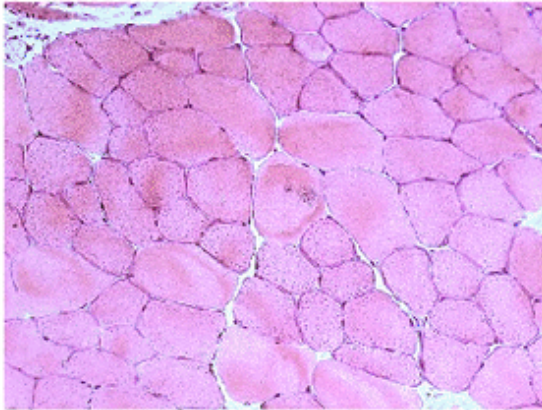
30 days post injury

(images enhanced from Mackey et al. 2016 for clarity and contrast using AI)

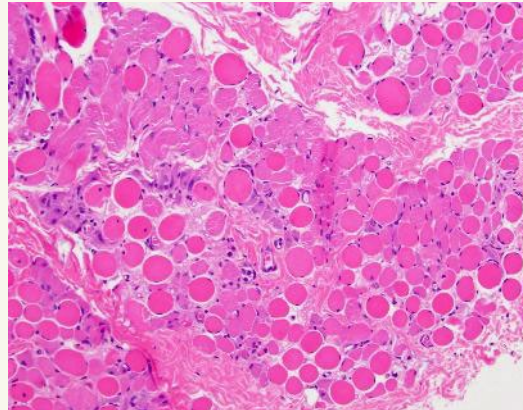
In Duchenne, Muscle Regeneration Loses the Battle With Constant Degeneration, Failing to Maintain Muscle



Healthy

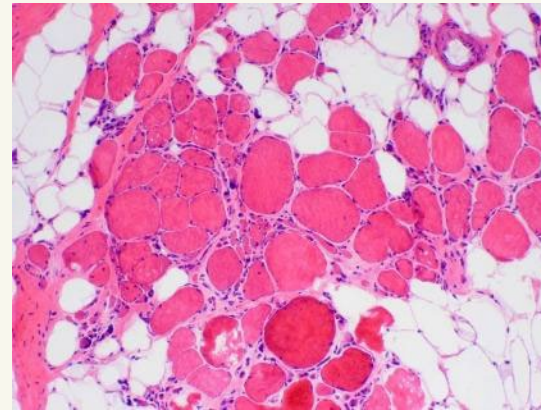


Myopathic changes



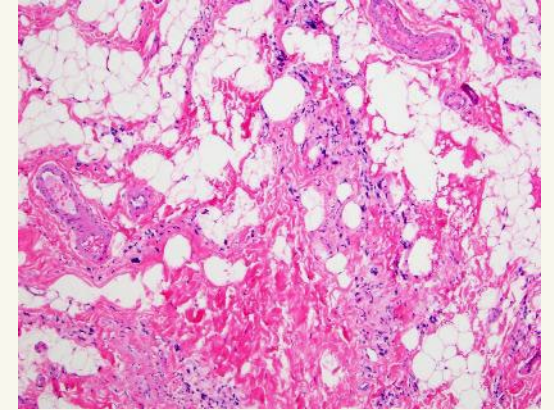
Early stage

Fatty replacement



Mid stage

Fibrosis



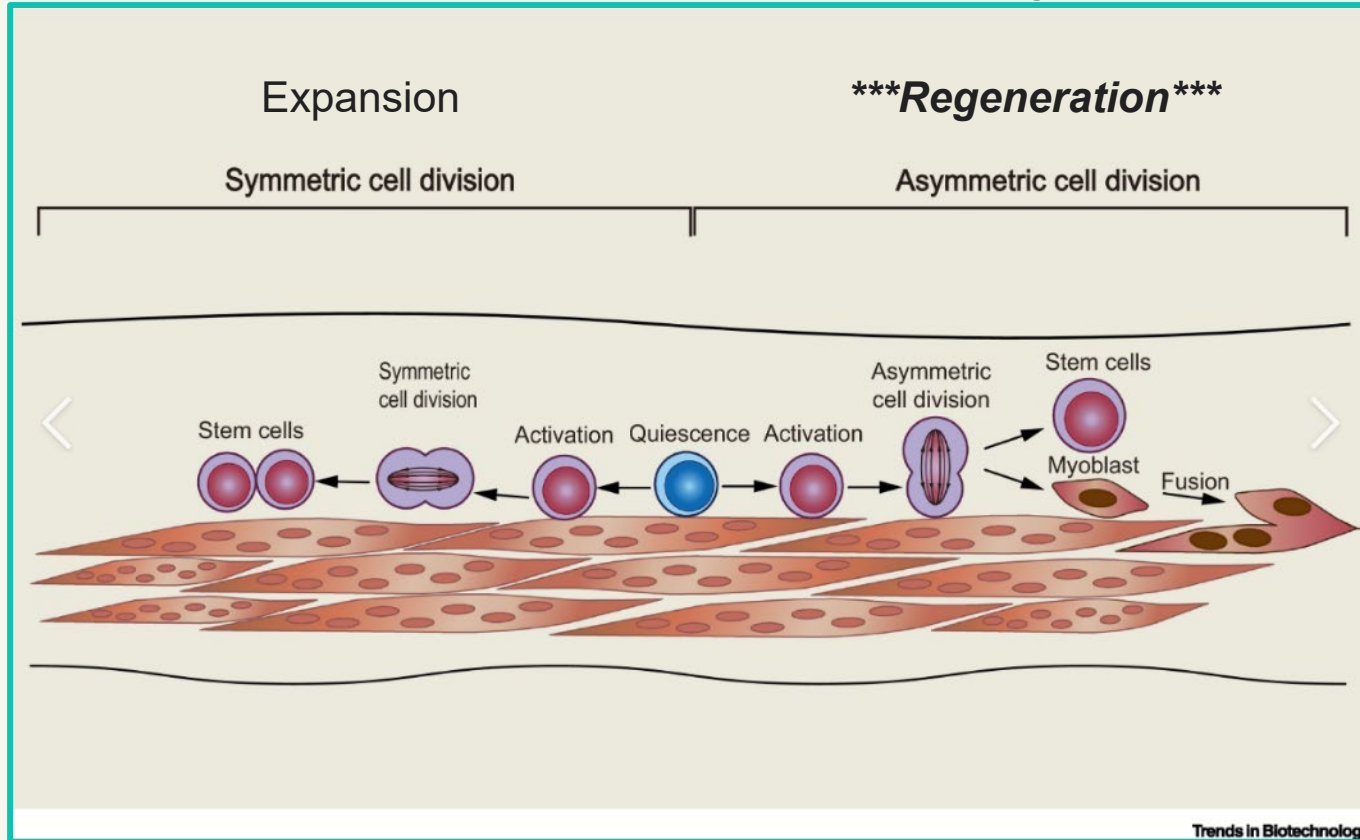
Late stage

Why Does Regeneration Fail in Duchenne?



Regeneration is Driven by Muscle Stem Cells

Stem cell division takes place on myofibers

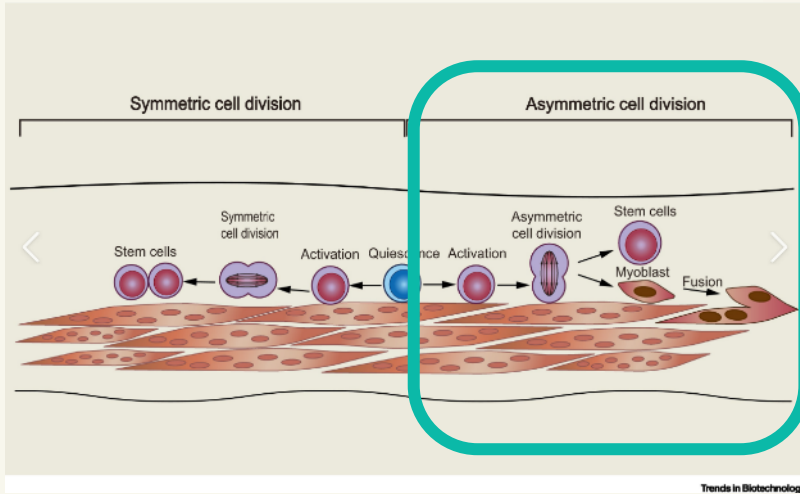


Stem Cell Division Key Terms

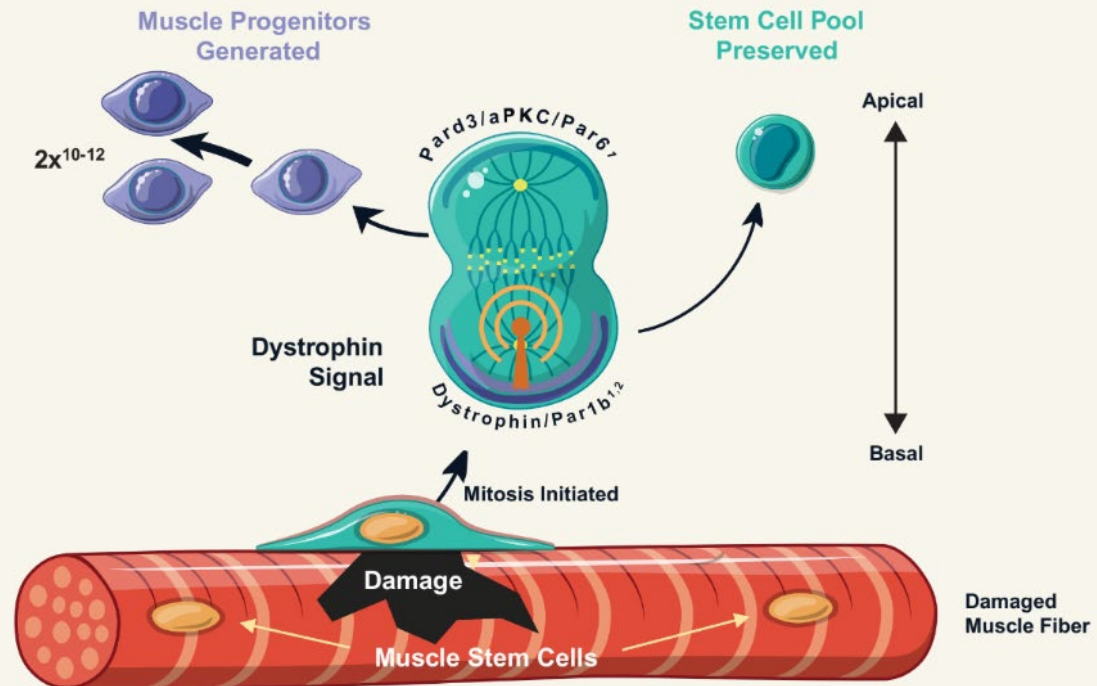
1. **Symmetric cell division:** expanding the stem cell pool

2. **Asymmetric cell division:** producing new muscle tissue and maintaining the pool
(this is regeneration)

Core Discovery: Asymmetric Cell Division (*Regeneration*) is Regulated by Dystrophin

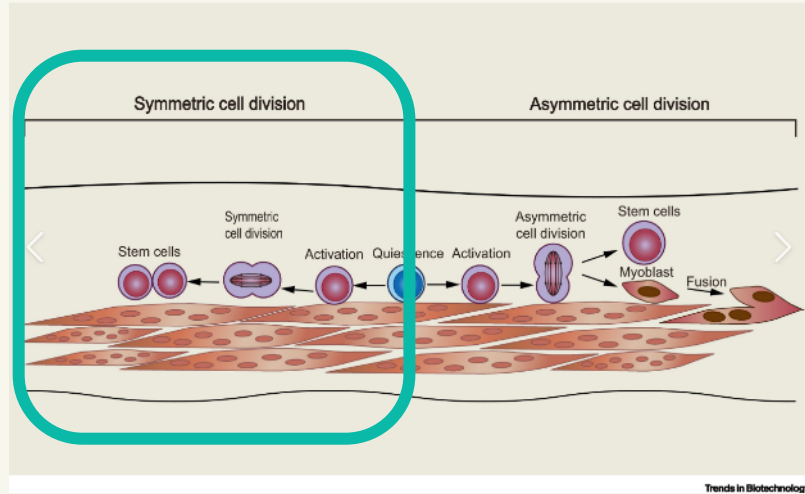


Dystrophin 'signal' present: Repair and regeneration occurs

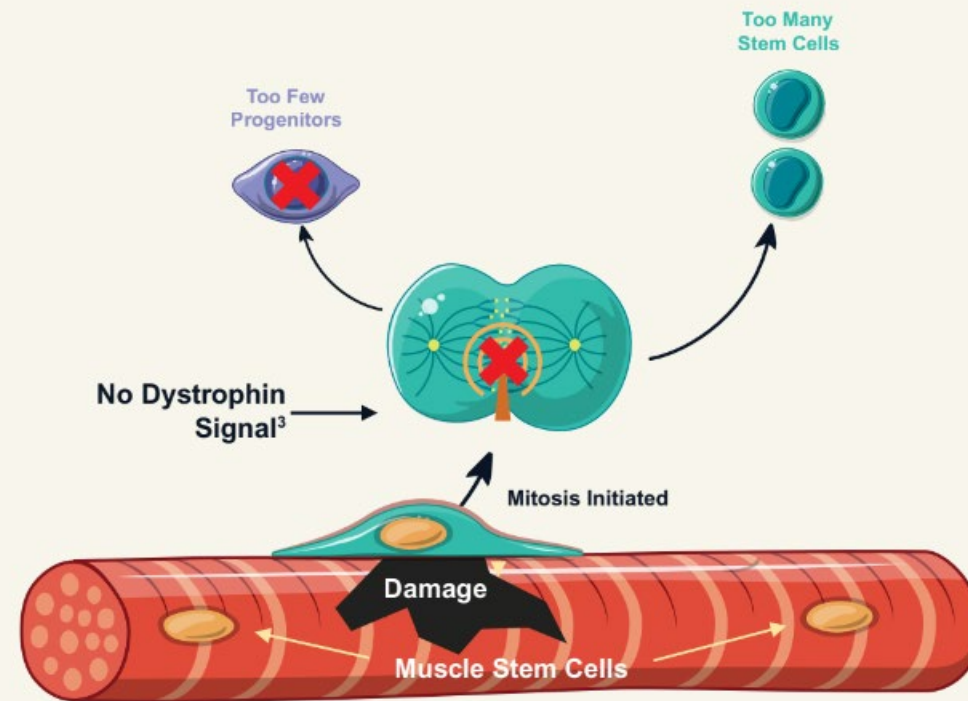


1. *Par3, APKC, Par6, and Par1b form the Par Polarity Complex*
2. *Dystrophin phosphorylates Par1b to establish the basal pole of the cell and release Pard3 et al to form the apical pole*
3. *Absent Dystrophin, phosphorylation of Par1b does not occur, polarity is impaired and the balance of stem cells to progenitors is distorted*

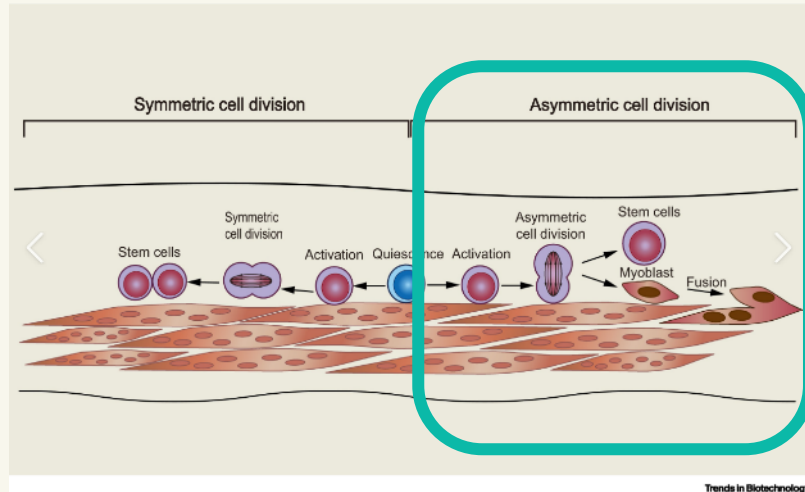
Core Discovery: Asymmetric Cell Division (*Regeneration*) is Impaired in Duchenne



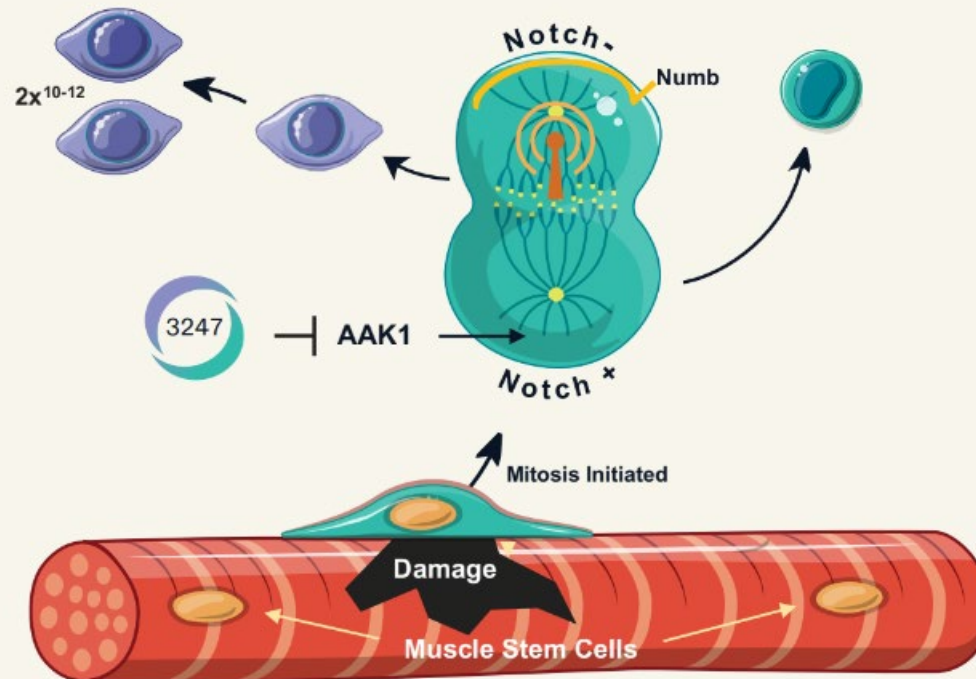
Dystrophin 'signal' absent: repair and regeneration impaired



Core Discovery: Asymmetric Cell Division (*Regeneration*) Can Be Functionally Rescued via Inhibition of AAK1



Missing 'signal' replaced: repair and regeneration occurs



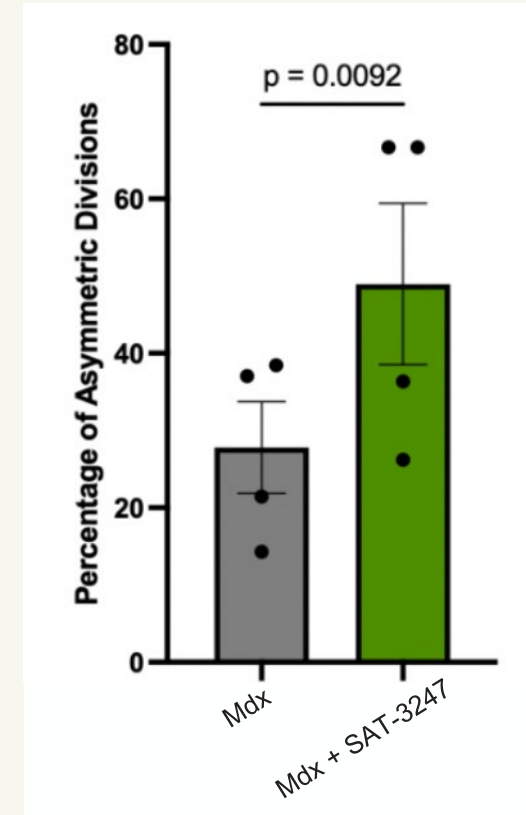
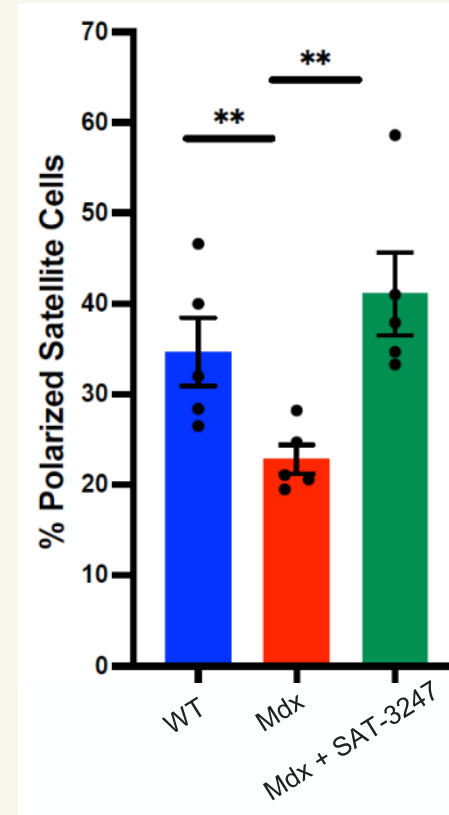
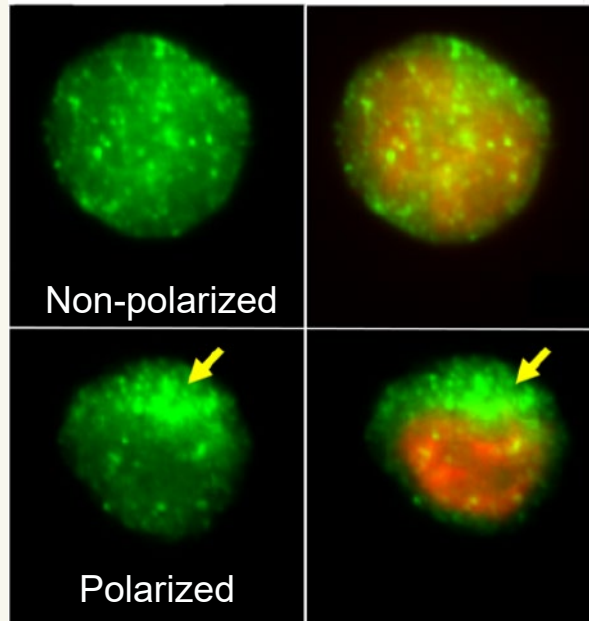
SAT-3247 Polarizes Notch Signalling Inside of Duchenne Muscle Stem Cells Resulting in Increased Rates of Asymmetric Division



Mdx muscle stem cells

NUMB

NUMB Pax7



What did Satellos discover?

1. Regeneration is regulated by dystrophin
2. Regeneration is impaired in Duchenne, due to loss of dystrophin
3. Regeneration can be functionally rescued via inhibition of AAK1, even in the absence of dystrophin

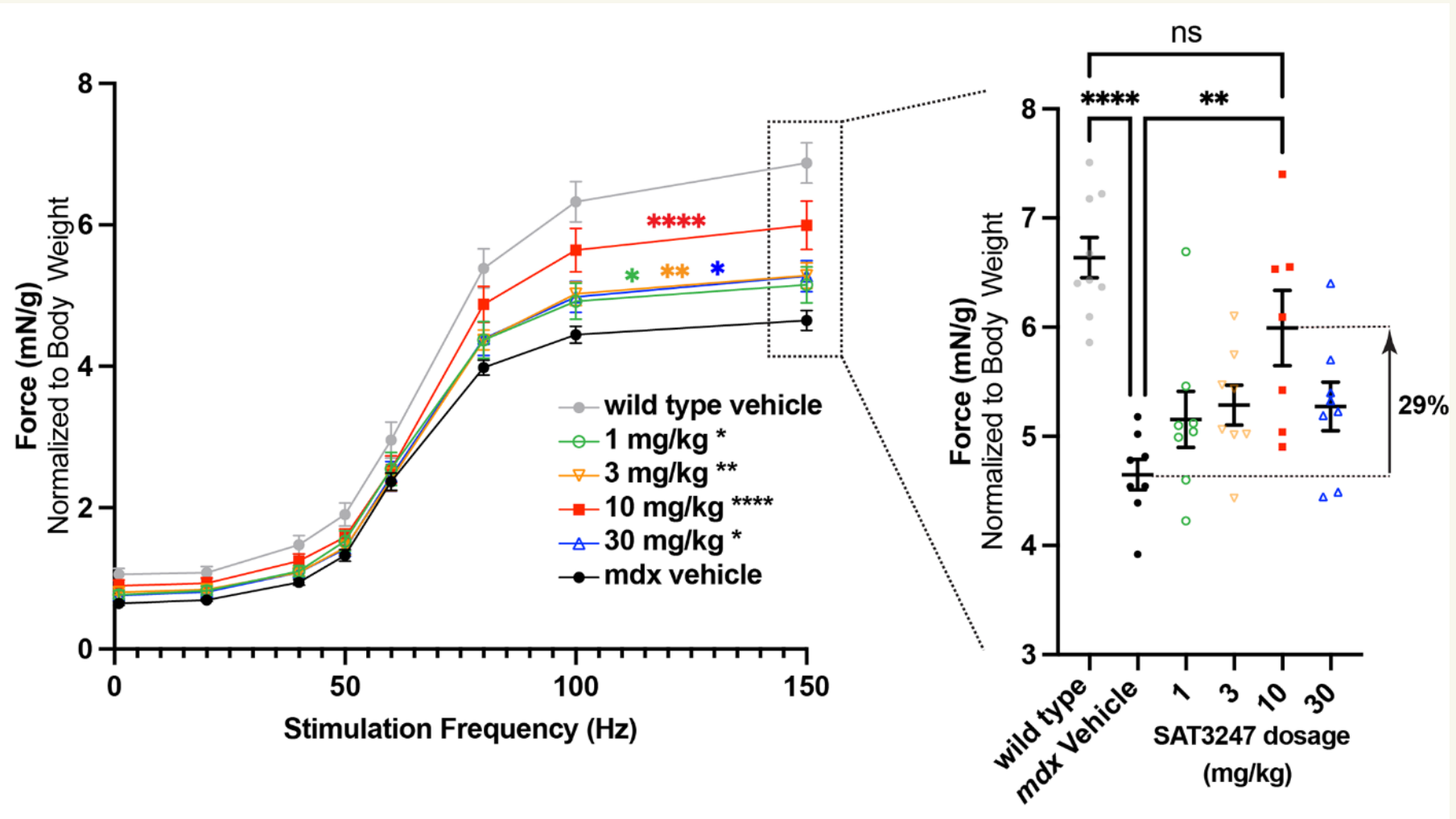


SAT-3247 enables the natural muscle regeneration process to better respond to ongoing damage

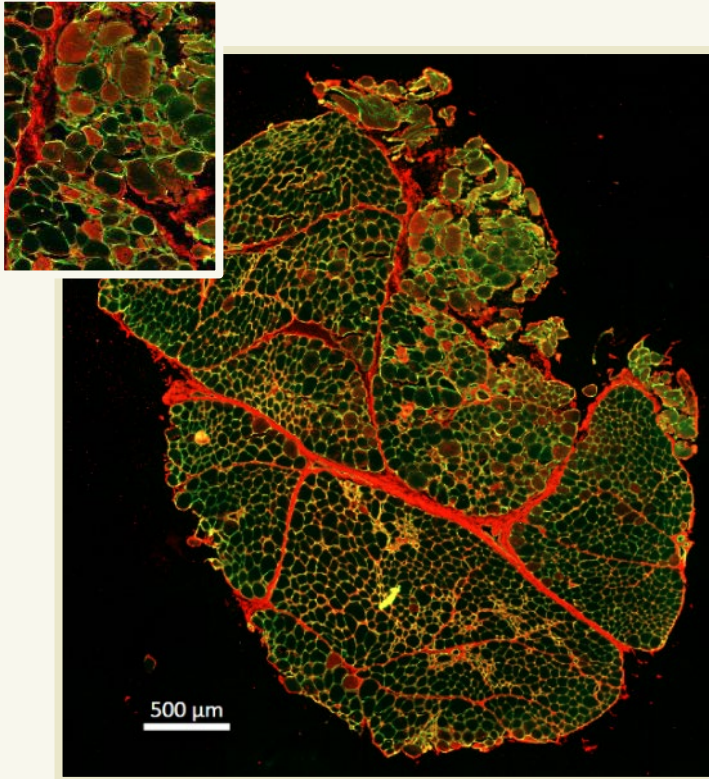
Preclinical Highlights



Enhancing Progenitor Production Through Increased Asymmetric Division Leads to Improved Muscle Strength

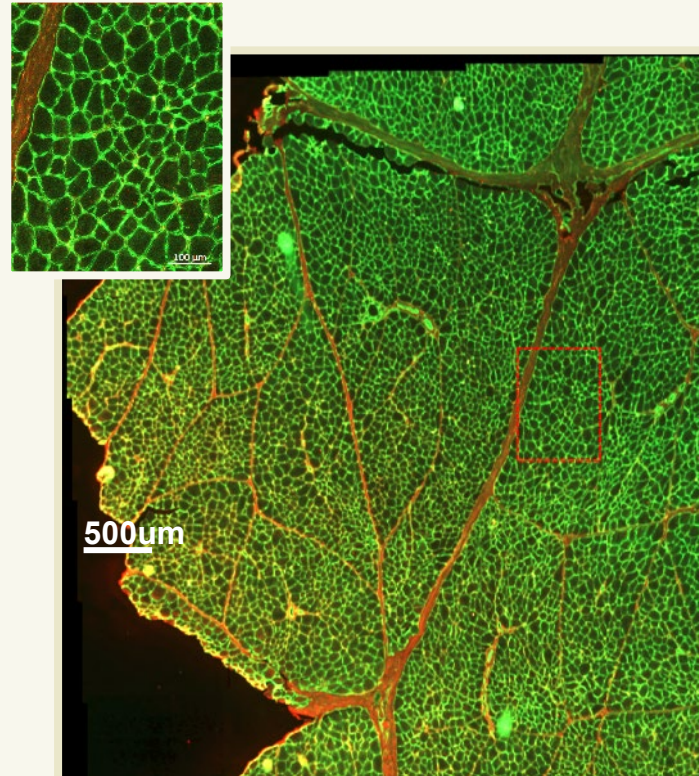


SAT-3247 Treatment Shows Muscle Regeneration in Canine Model of DMD



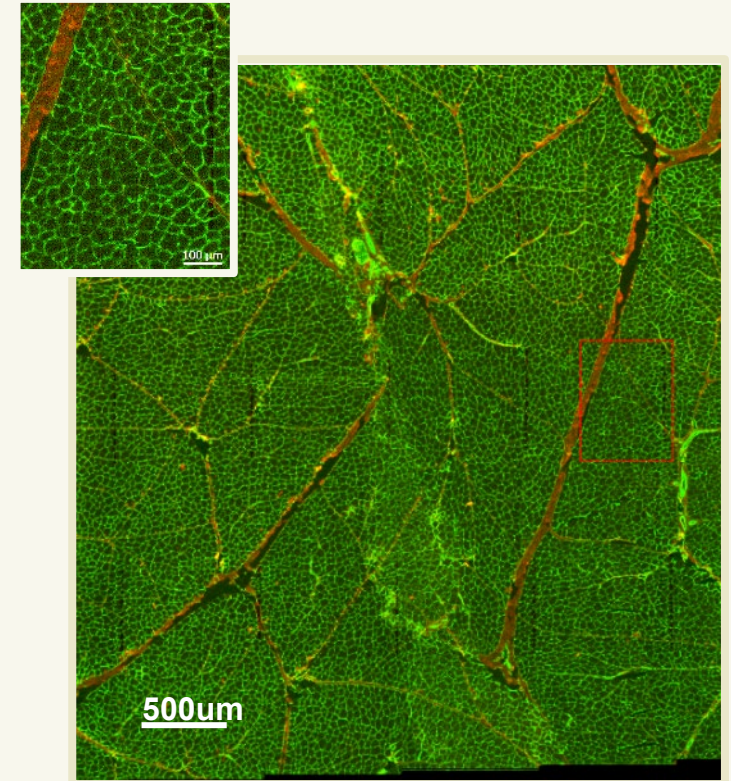
SAT-3247 Study

DMD Animal / Pre-Treatment
Bicep Femoris



SAT-3247 Study

DMD Animal / Post-Treatment
Bicep Femoris



Healthy Age-Matched

Non-DMD Comparator
Bicep Femoris

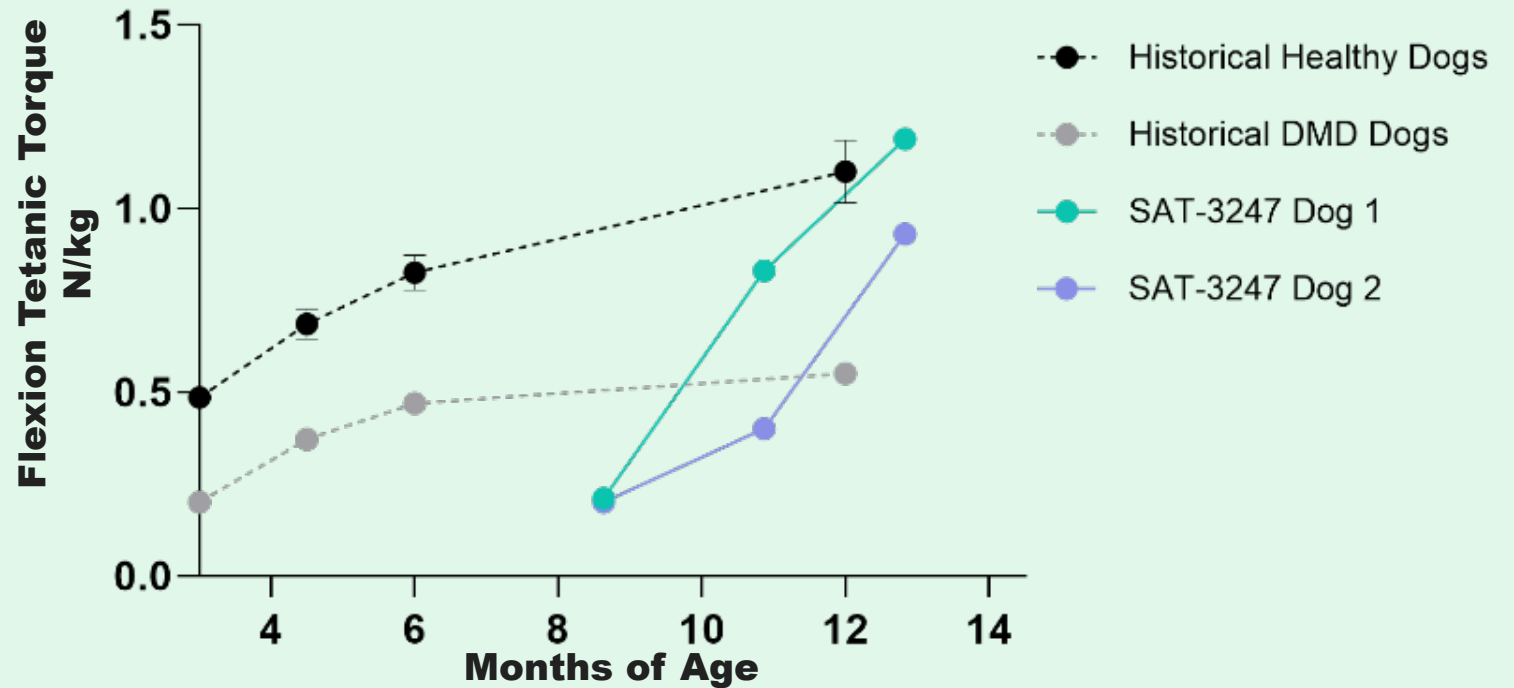
60 mg oral dose | 4x/wk | 4 months of treatment | ~9 – 13 months of age

SAT-3247 Treatment Restores Muscle Strength in Canine Model of DMD

Study Overview

- 2 DMD dogs: each ~9 mos. of age at start of study
- Oral dosing with **SAT-3247**:
 - 1x per day, 60 mg
 - 4 days on, 3 days off
 - 4 months of treatment
- Multiple measurements throughout
- Study terminated at 4 months

SAT-3247 treated vs. historical controls¹



1. Kornegay, J.N., et. al., Journal of Neurological Sciences, 1999

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SAT-3247 Aims to Enhance Muscle Regeneration and Improve Function

Approximately 12,000 boys or young men currently live with DMD in the US

Genetic approaches to DMD

- Administered by infusion or injection
- Gene therapy (DNA) or exon skipping (RNA)
- Aims to improve function by stabilizing muscle
- Limited by exon status or potential immunogenicity concerns

WAVE
LIFE SCIENCES

AVIDITY
BIOSCIENCES

Dyne
THERAPEUTICS

SAREPTA
THERAPEUTICS

SOLID
BIOSCIENCES

Satellos approach to DMD

- Oral, once-daily pill
- Designed to be safe, tolerable and non-immunogenic
- Aims to improve function by regenerating muscle
- Intended for all patients regardless of exon mutation
- Potential as stand-alone or add-on therapy

SAT-3247 Phase 1 First-in-Human Clinical Trial

Ph 1a: Healthy Volunteers (n=72)		Ph 1b: DMD Adults
SAD* Study	MAD* Study	28-day, Open-Label Study
10 - 400 mg 5 cohorts of 8 - Single dose / 1 day	60 - 240 mg 4 cohorts of 8 - Single dose / 7 days	60 mg daily dose, weekdays only for 4 weeks 5 patients enrolled and completed - Age range 20 – 27, all on steroids

Endpoints

Primary (Ph 1a & 1b): Safety & tolerability (labs, vitals, ECG, physical exam)

Secondary (Ph 1a & 1b): Pharmacokinetic exposure (PK)

Exploratory (Ph 1b only): Grip/pinch strength, upper body effort, lung function (FVC), serum markers

**SAD: single ascending dose; *MAD: multiple ascending dose.*

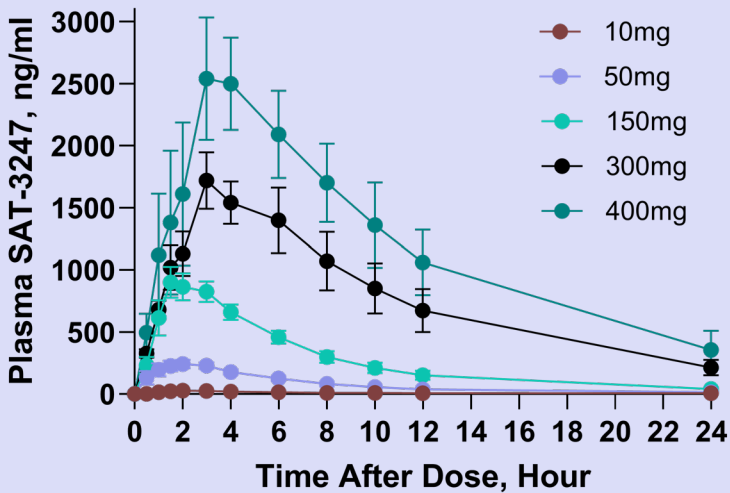
SAT-3247 Was Safe and Achieved Target PK in Healthy Volunteers

Safety Profile

AE Category	Part A (n=40)	Part B (n=32)	Part C (n=8)
Any TEAE	9	18	4
Any related TEAE	1 ^a	2 ^b	0
Any serious TEAE	0	0	0
Any related serious TEAE	0	0	0
Any TEAE leading to withdrawal	0	1 ^c	0
Any TEAE leading to death	0	0	0

^a Mild nausea/abdominal pain (resolved); ^b Mild somnolence (resolved), mild abdominal pain (resolved); ^c Covid-19 (unrelated, resolved)

PK Profile



- ✓ Dose proportional exposure levels
- ✓ Drug clearance within 24 hours

SAT-3247 Was Safe and Achieved Target PK in Adults with DMD

5 adults with DMD ages 20 to 27 years, all receiving steroid therapy

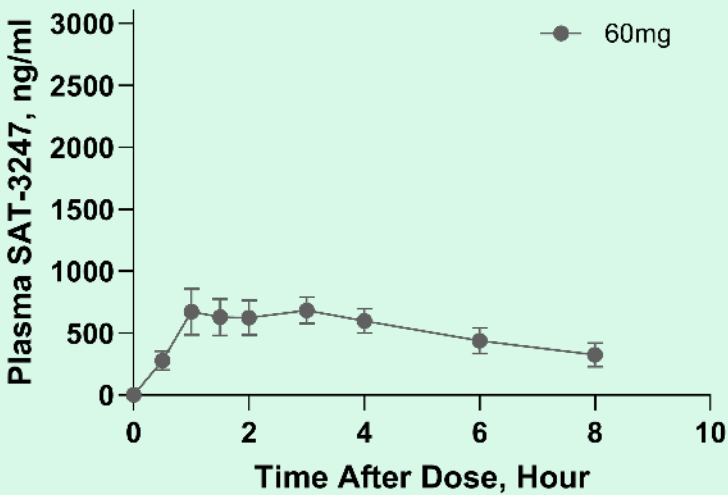
Safety Profile

AE Category	Part D (n=5)
Any TEAE	4
Any related TEAE	2 ^d
Any serious TEAE	2 ^e
Any related serious TEAE	0
Any TEAE leading to withdrawal	0
Any TEAE leading to death	0

^d Mild nausea (resolved), elevated ALT ((ref range (5, 40) – baseline(29), day 28(47) - resolved))

^e Diverticulitis, Pneumothorax

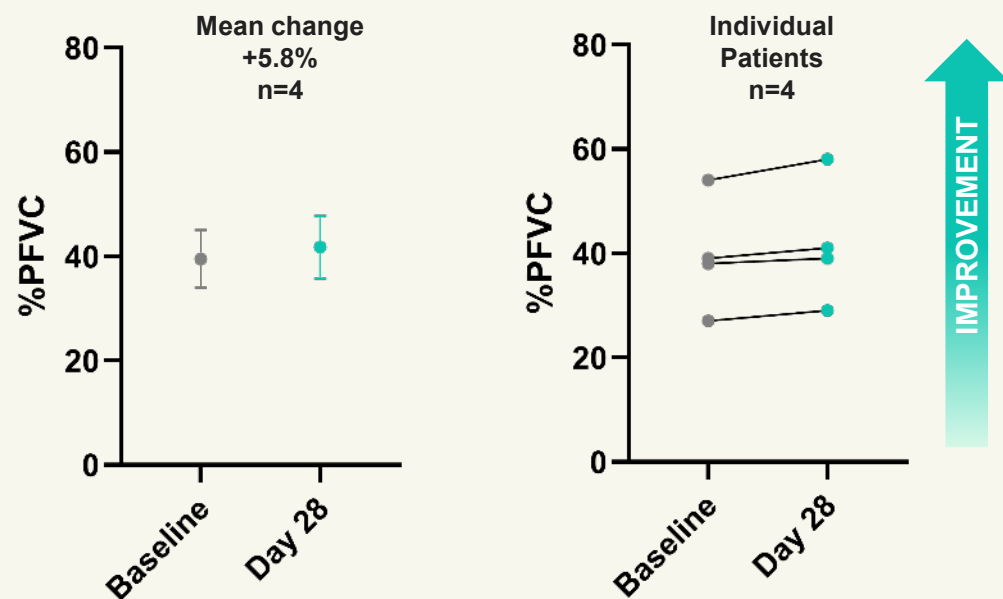
PK Profile



✓ Desired exposure met in patients

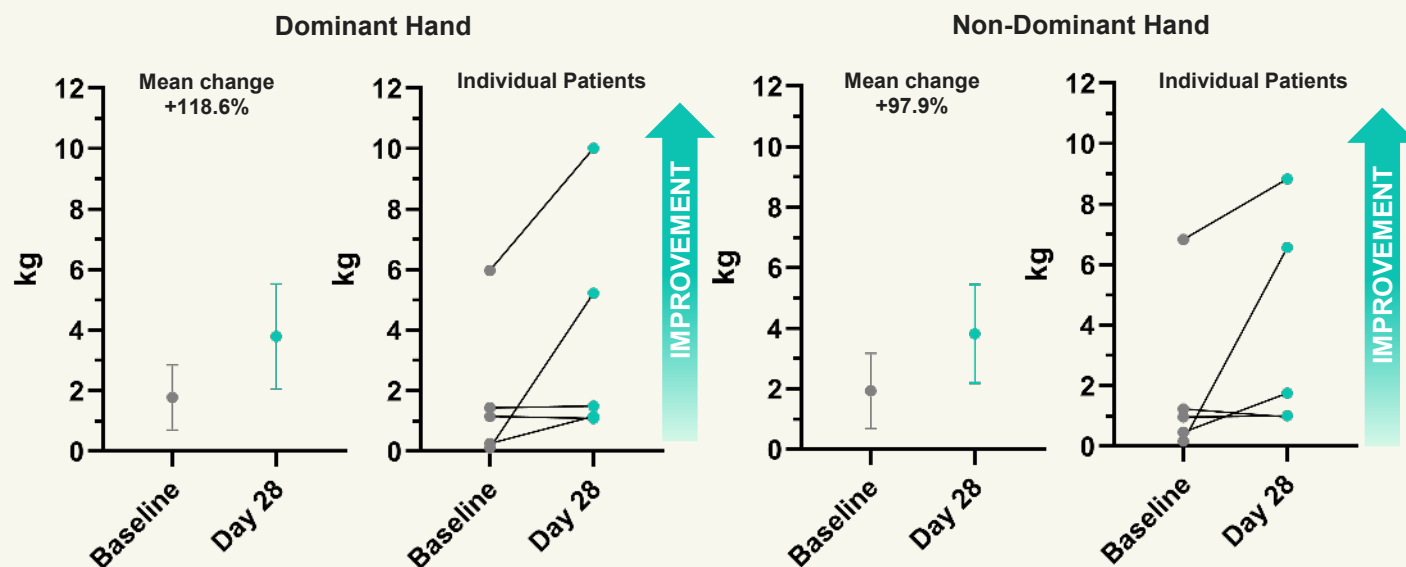
SAT-3247: Grip Strength Shows Encouraging Benefit in 28 Days

5.8% Increase in Predicted Forced Vital Capacity (FVC)



Typically, DMD patients in this age group experience a 5% annual decrease in FVC

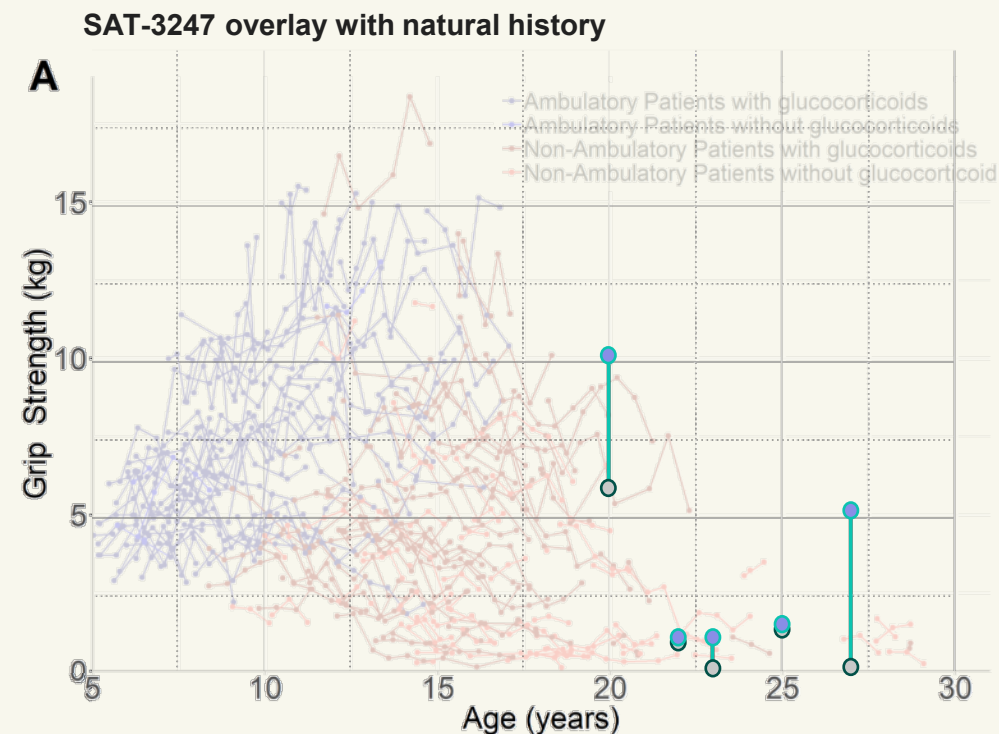
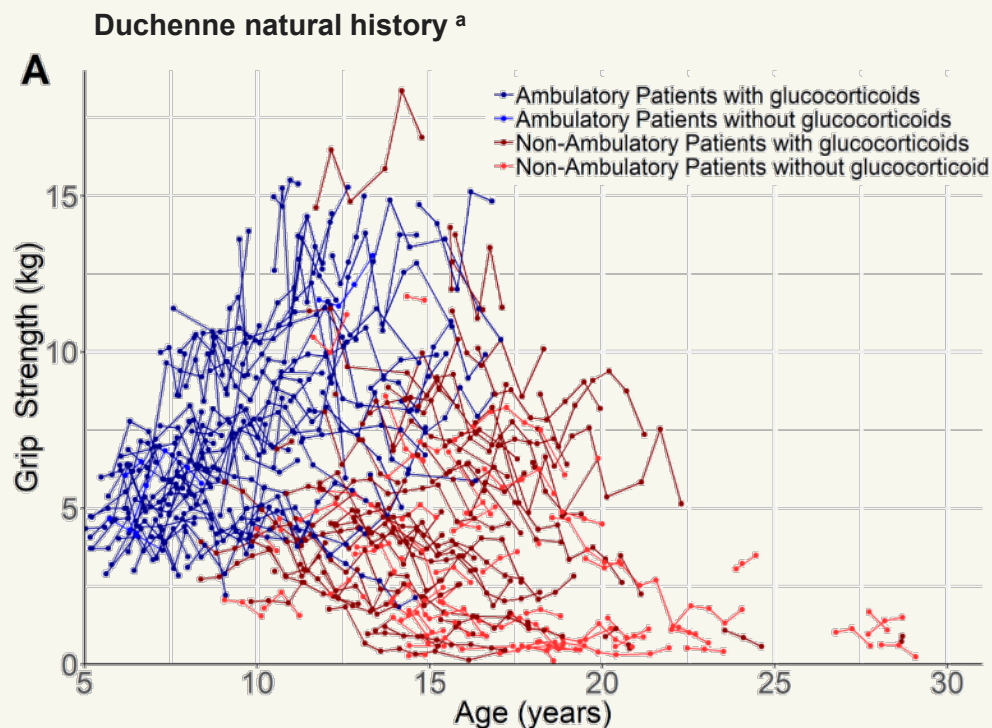
118.6% and 97.9% Increases in Grip Strength in Dominant and Non-Dominant hands



Improvements seen with SAT-3247 are beyond reported experimental variability with the grip device

Increased Grip Strength with SAT-3247

Treatment Exceeds Results Seen in Natural History

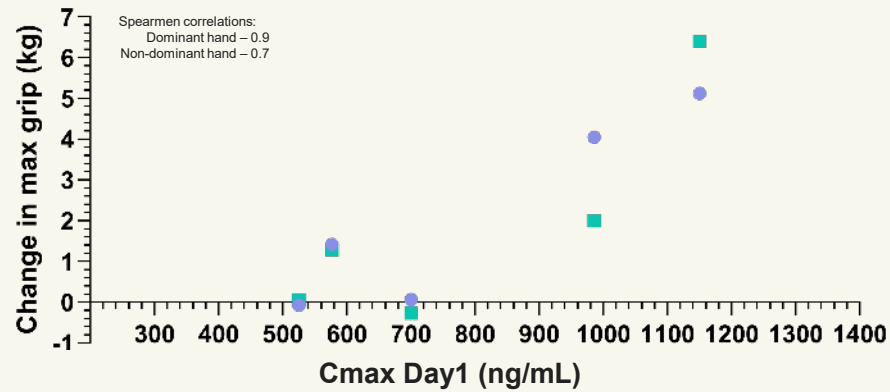


- **Improvement with SAT-3247** beyond what is reported for natural history
 - Absolute grip strength is consistently below 10 kg after age 20 years and below 5 kg after age 25 years
- **Increases seen in 3 patients after 28 days** appear to be moving into “**white space**”
 - Improvements of this nature (if sustained) would provide opportunity for early approval consideration

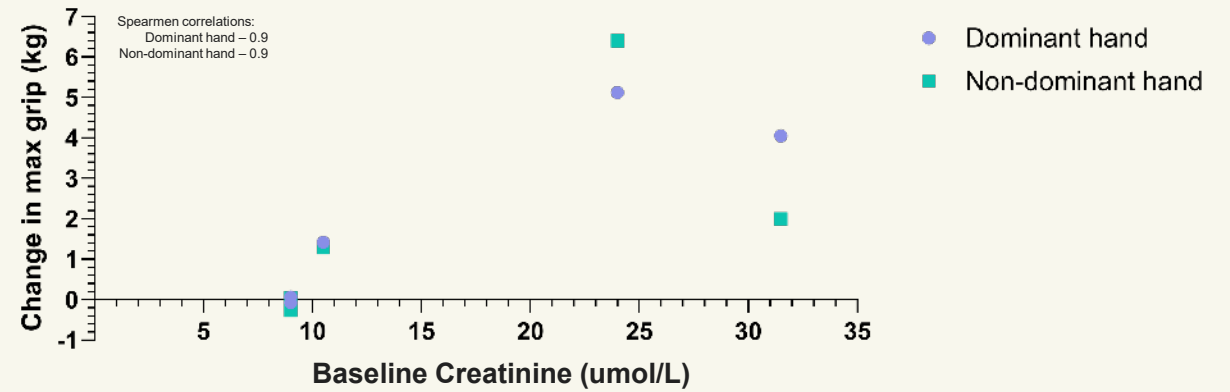
Grip Strength Improvements Correlate with Cmax and Creatinine Levels

Supports rationale that results seen are drug related

Maximum Grip Strength vs Cmax



Maximum Grip Strength vs Baseline Creatinine



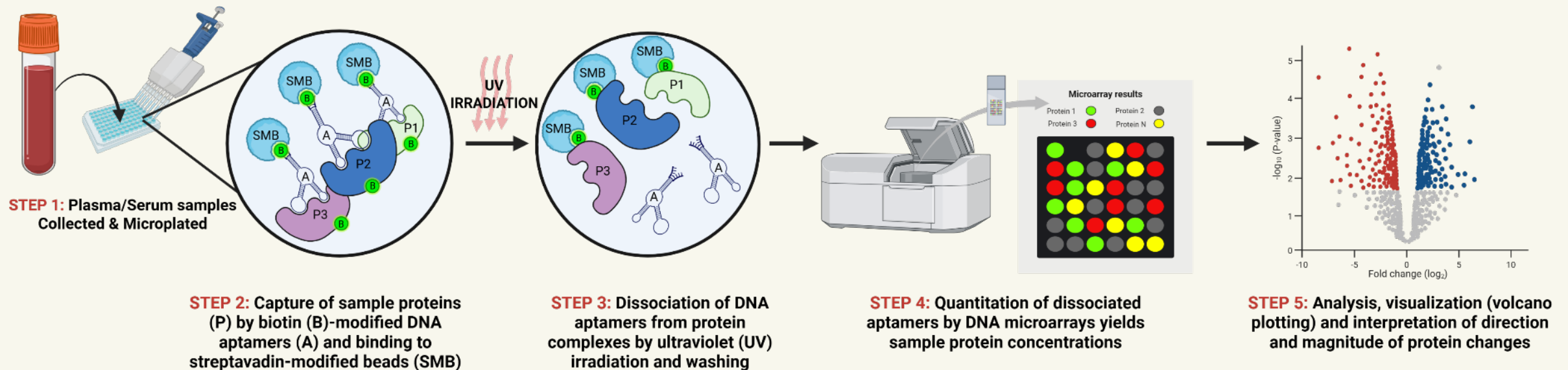
- **Greatest improvements** in grip strength occurred in participants with:
 - **Highest Cmax on Day 1**; and
 - **Highest baseline creatinine** (surrogate marker for muscle mass)

Emerging Biomarker Insights in DMD Following SAT-3247 Treatment

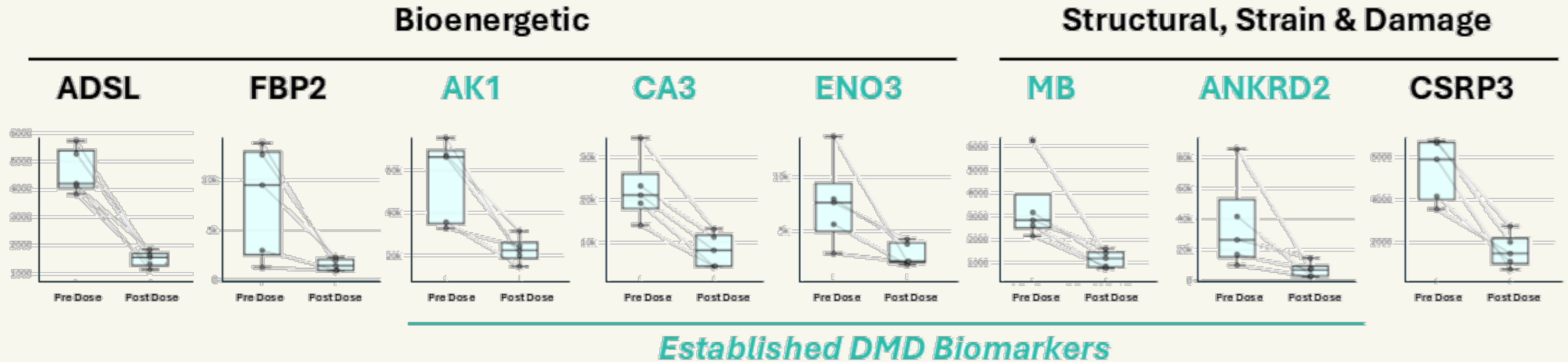


Broad Proteomic Profiling Enables Assessment of Multiple Biological Pathways

- Measures thousands of circulating proteins simultaneously
- Captures coordinated biological signals beyond single biomarkers
- Provides relative changes over time
- Used by Satellos to explore treatment-related biological effects in DMD



Consistent Declines Across Established DMD Biomarkers



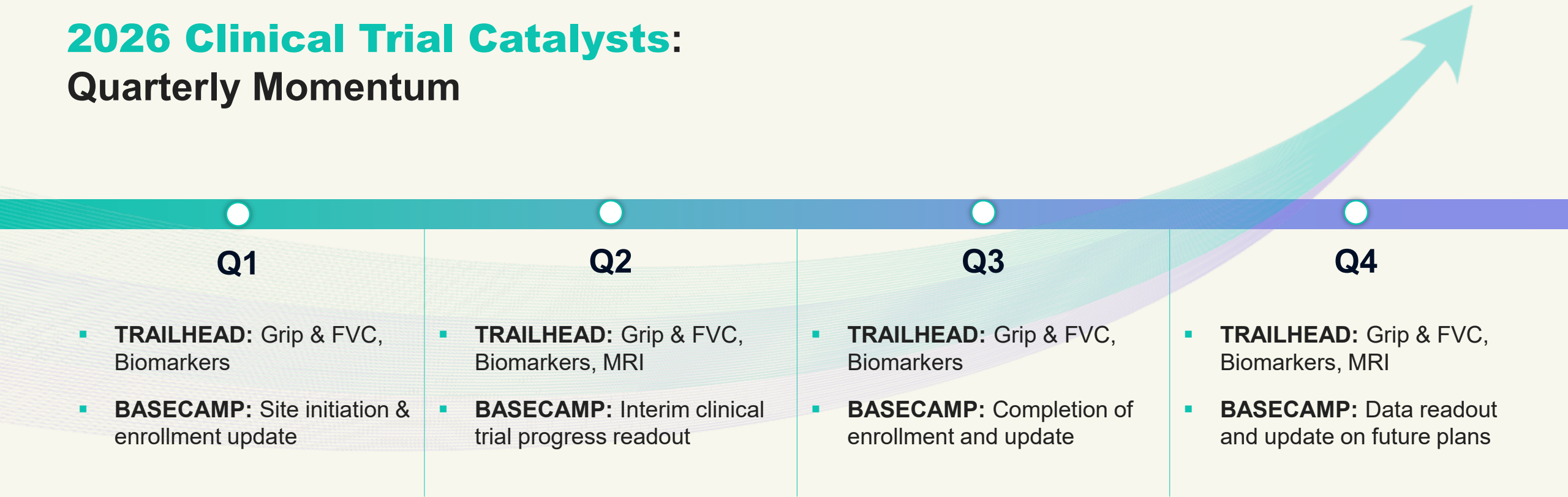
Abbreviations: ADSL=adenylosuccinate lyase; AK1=adenylate kinase isoenzyme 1; ANKRD2=ankryn repeat domain-containing protein 2; ENO3=beta-enolase; CA3=carbonic anhydrase 3; CSRP3=cysteine and glycine-rich protein 3; FBP2=fructose-1,6-bisphosphatase isoenzyme 2; MB=myoglobin

- **Reductions in classical DMD biomarkers of muscle injury** known to correlate with CK (ie. AK1, CA3, MB, ENO3) after 15 days of dosing
- Reduction in recently identified potential biomarkers of muscle injury (ie. ANKRD2)
- Additional work ongoing to further characterize the relationship between SAT-3247 and potential biomarkers identified with proteomic profiling

Current Clinical Trials to Evaluate SAT-3247 in People with DMD

	TRAILHEAD (LT-001)	BASECAMP (CL-201)
Participants	Up to 30 adults with DMD >=18 years	51 ambulatory DMD participants aged ≥ 7 and < 10 years of age
Summary	Will assess the long-term safety, tolerability and potential efficacy of a 60 mg dose of SAT-3247 with a weekday regimen in an 11-month, open-label study.	Will study 60 and 120 mg of SAT-3247 in a randomized, double-blind, placebo-controlled weekday regimen for 12 weeks to determine the optimal dose, safety, tolerability, and preliminary efficacy.
Locations	Australia: up to 10 participants USA: planning to file with FDA and enroll up to 20 additional participants	U.S., UK, EU (Belgium, Spain, Poland), Serbia, Canada, and Australia.
Current Status	1 site activated; 4 participants being dosed	5 sites activated in U.S.; 1 participant being dosed

2026 Clinical Trial Catalysts: Quarterly Momentum



LT-001: Duchenne open-label long-term follow-up study in patients 16-40
BASECAMP (CL-201): Randomized, placebo-controlled, global study in pediatric DMD patients

Poster and Oral Presentations To Be Presented at MDA, March 8-11, 2026

Agenda



Welcome

Frank Gleeson *Co-founder & Chief Executive Officer, Satellos*



Duchenne and the Need for Additional Therapeutic Approaches

Dr. Kevin M. Flanigan *Wolfe Foundation Endowed Chair in Neuromuscular Research, Nationwide Children's Hospital; Professor of Pediatrics & Neurology, Ohio State University College of Medicine*



The Science Behind SAT-3247

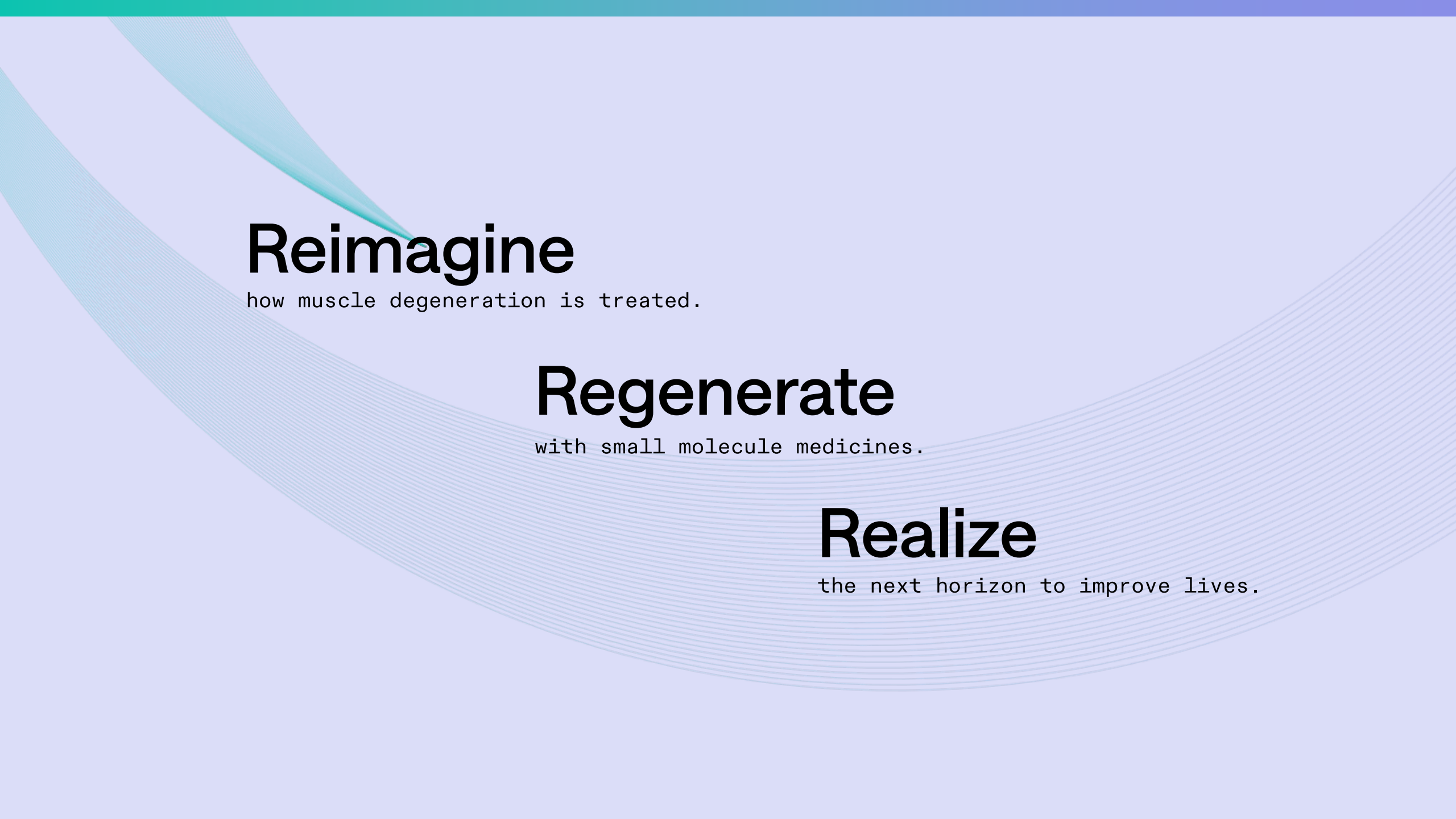
Dr. Michael Rudnicki *Co-founder & Chief Discovery Officer, Satellos; Senior Scientist & Director, Sprott Centre for Stem Cell Research, Ottawa Hospital Research Institute*



SAT-3247 Clinical Development Overview

Dr. Wildon Farwell *Chief Medical Officer, Satellos*

Q&A



Reimagine

how muscle degeneration is treated.

Regenerate

with small molecule medicines.

Realize

the next horizon to improve lives.