satellos

Corporate Overview · July 2026

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OUR MISSION

To change the lives of people living with Duchenne muscular dystrophy and other muscle diseases — by restoring the body's natural ability to regenerate muscle.

Grounded in science. Inspired by need.

CHARLIE · age 8, living with Duchenne

COMPANY OVERVIEW

Who We Are

Founded **2018** Stage **Clinical** **TSX: MSCL** **NASDAQ: MSLE**

SCIENTIFIC FOUNDATION

Dr. Michael Rudnicki

Scientific Founder & Chief Discovery Officer

DMD is a disease of failed muscle regeneration — not merely muscle breakdown.

Dr. Rudnicki's landmark discovery reframed the disease — and the opportunity to treat it.

FOUNDATIONAL RESEARCH

Nature Medicine (2015) **Cell** (2007)

OUR APPROACH

SAT-3247 — first-in-class oral, once-daily tablet

- Not just stabilizing — designed to regenerate muscle
- Potential to be disease modifying
- Suitable for all patients regardless of genetic mutation
- Potential as stand-alone or add-on therapy
- Designed to be safe, tolerable and non-immunogenic

Children Phase 2 · ages 7–9 · N=51 · placebo-controlled

Adults Phase 2 · adults ≥16 · open-label

2026 data readout Top-line data from both trials

A Large, Unmet Need

Therapeutic need in DMD

1 in 5,000 male births worldwide — a fatal genetic disease

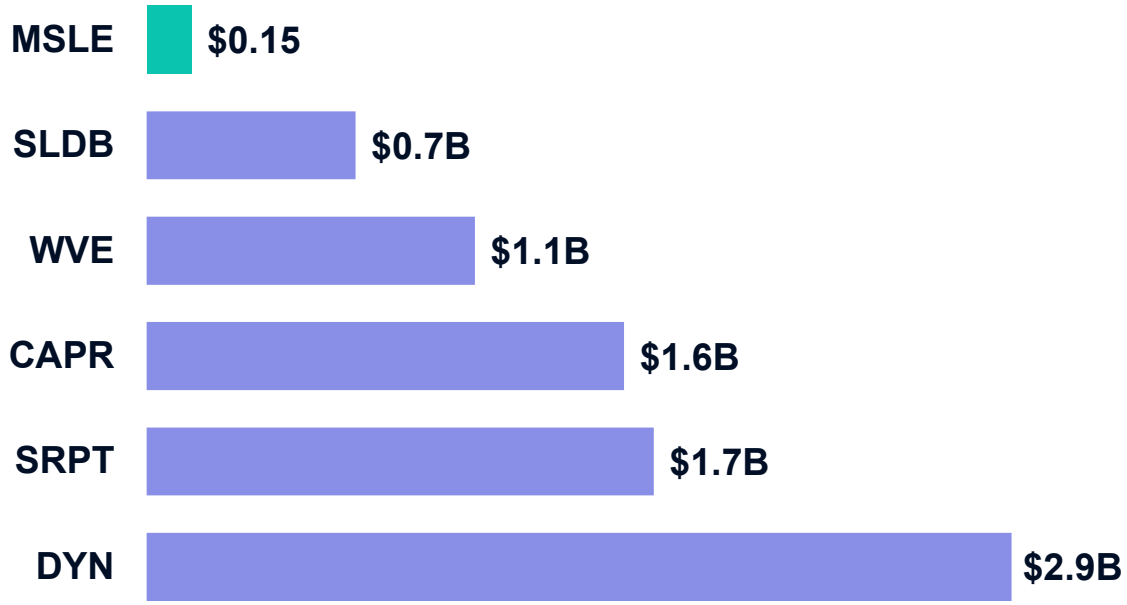
~12,000 individuals affected in the U.S. alone

0 approved therapies designed to regenerate muscle

Standard-of-care corticosteroids carry toxic side effects; the lone gene therapy is restricted to young children, and exon skippers reach only narrow genetic subsets.

Significant value created by DMD-focused peers

Market capitalization, US$BN (June 2, 2026)



THE PROBLEM

Rewriting the Duchenne Story

Infants–4 yrs

EARLY CHILDHOOD

Children have functional muscle in early years

5–9 yrs

TIPPING POINT

Muscle loss begins to exceed muscle gains

10–19 yrs

DECLINE

Progressive loss of muscle mass & function

20 yrs +

LATE STAGE

Premature death, typically cardiorespiratory failure

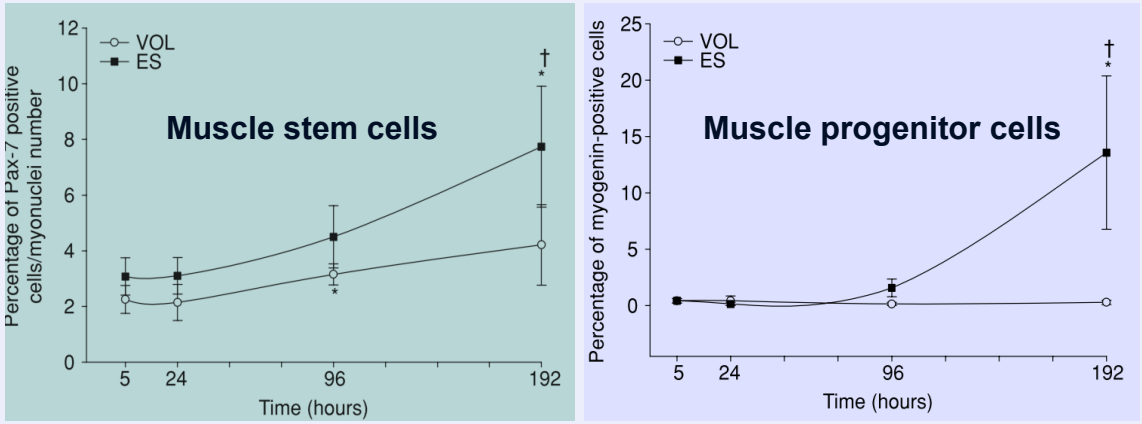
Satellos focus: restoring muscle regeneration and function with SAT-3247

Why Regeneration Fails in Duchenne

In DMD, muscle stem cells divide — but fail to produce the progenitor cells needed to repair damage faster than it accumulates.

Ratio of Muscle Progenitor to Stem Cells Normally Increases in Response to Damage

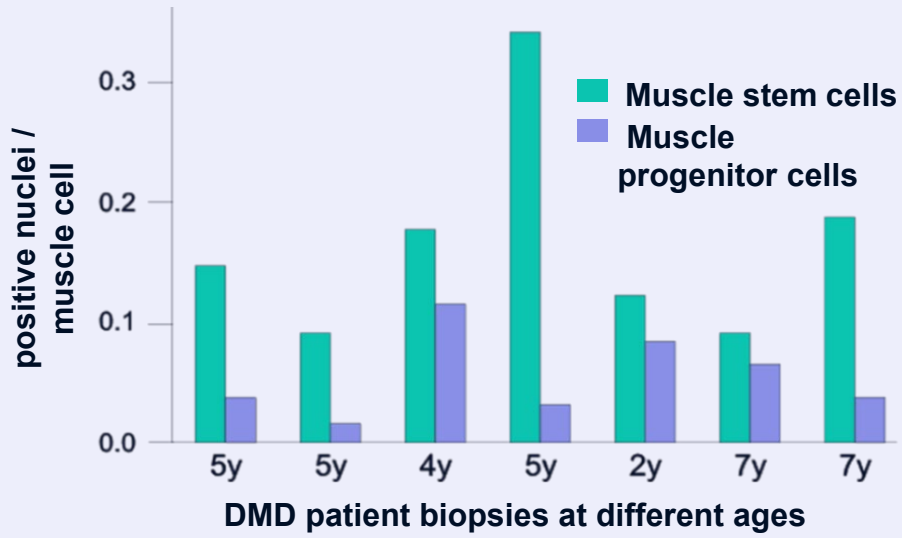
- Electrical stimulation injury model in healthy volunteers
- Rapid regeneration response within 4 – 8 days
- Higher ratio of progenitors to stem cells



J Physiol 583.1 (2007) pp 365–380

VOL: Voluntary
ES: Electrical Stimulation

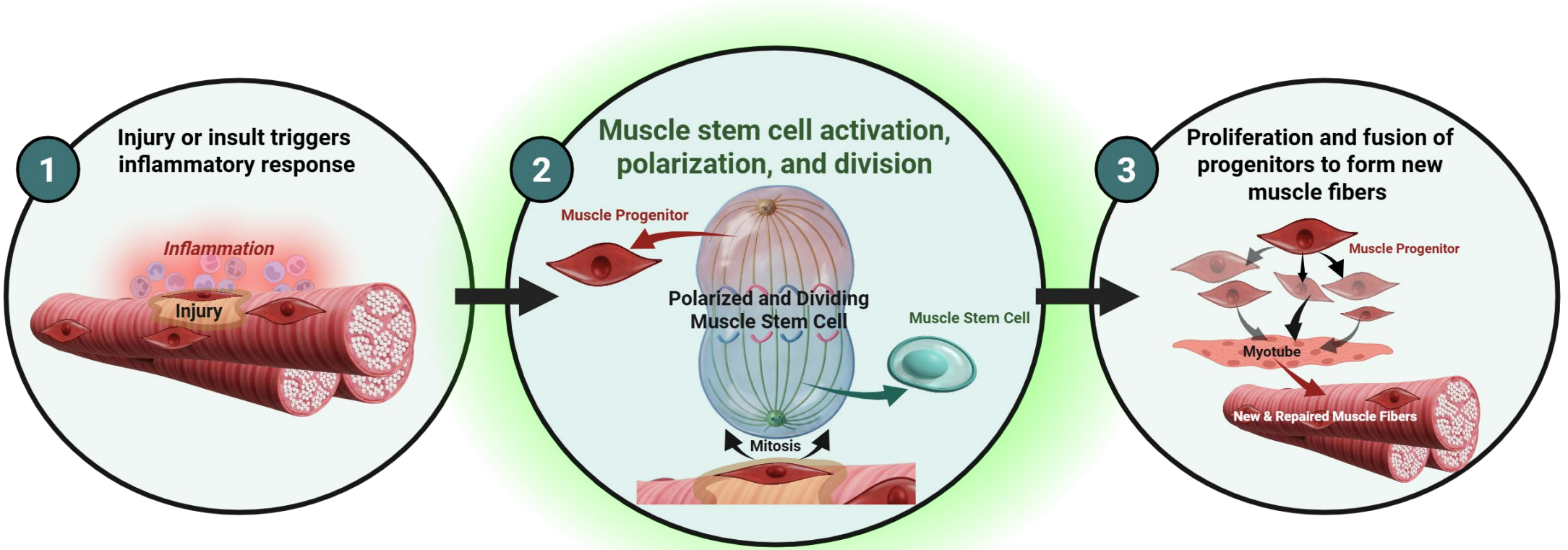
Ratio of Muscle Progenitor to Stem Cells Consistently Inverted in DMD Patients



Michael Kottlors et al., Cell Tissue Research. 2010

Muscle regeneration is a three-phase process that repairs muscle and produces new muscle fibers

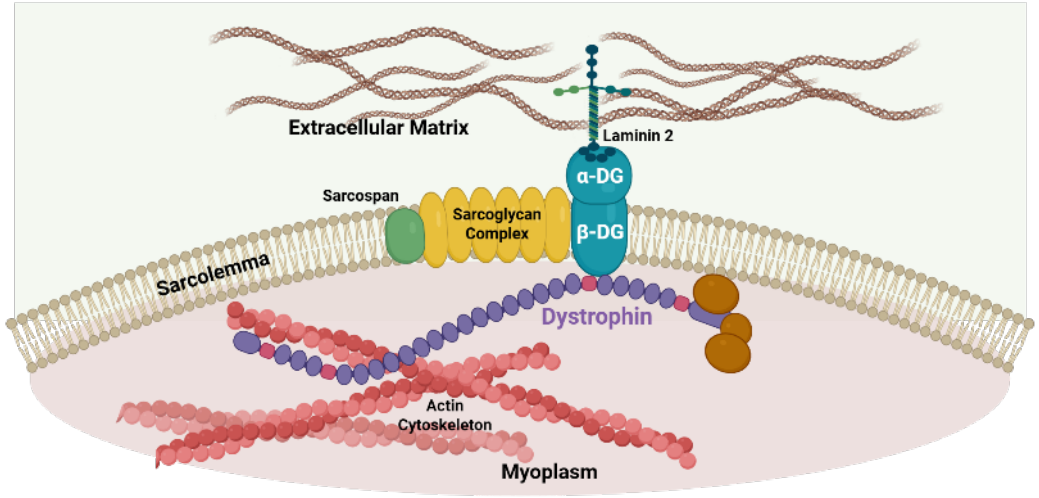
Muscle stem cell division and function plays a central role in muscle regeneration.



Dystrophin Serves Two Critical Functions in Skeletal Muscle

1

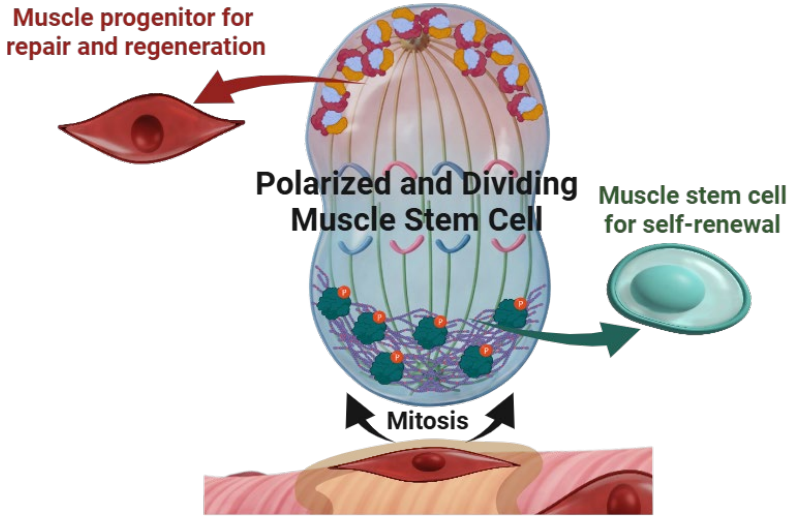
Structural reinforcement



Low or no dystrophin expression ~ **ongoing muscle damage**

2

Regulation of muscle stem cells



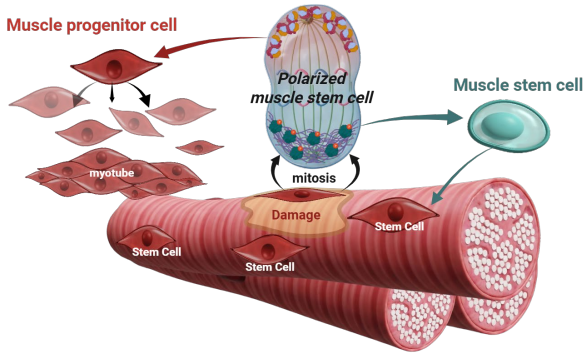
Low or no dystrophin expression ~ **ongoing loss of muscle mass**

SAT-3247 is Designed to Enhance Muscle Regeneration

SAT-3247 inhibits AAK1* to re-establish muscle stem cell polarity — resetting repair and regeneration.

HEALTHY

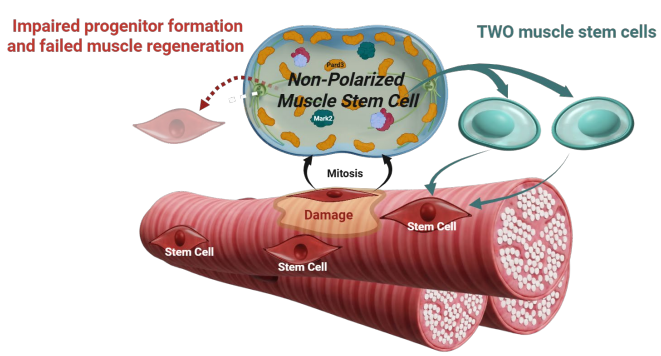
Muscle Repair = Muscle Damage



✓ Dystrophin enables muscle stem cell polarity to make new muscle cells for repair and regeneration

DMD

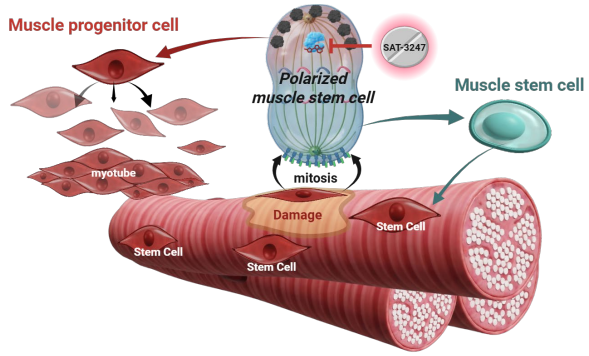
Muscle Repair ≪ Muscle Damage



✗ Absence of dystrophin impairs the ability to make enough new muscle cells for repair and regeneration

With SAT-3247

Muscle Repair = Muscle Damage



✓ SAT-3247 inhibits AAK1 to re-establish muscle stem cell polarity — enabling repair and regeneration

*AAK1: Adaptor-associated kinase 1

Progenitor formation can be restored

Inhibiting **AAK1** with **SAT-3247** replaces the repair signal lost when dystrophin is absent — restoring asymmetric division.

01

Dystrophin is absent

The repair signal that drives asymmetric stem-cell division is lost.

→

02

SAT-3247 inhibits AAK1

An oral small molecule restores the missing signal at its source.

→

03

Regeneration restored

Stem cells again produce progenitors — 1 progenitor + 1 stem cell.

01

THE EVIDENCE

Overview of Data

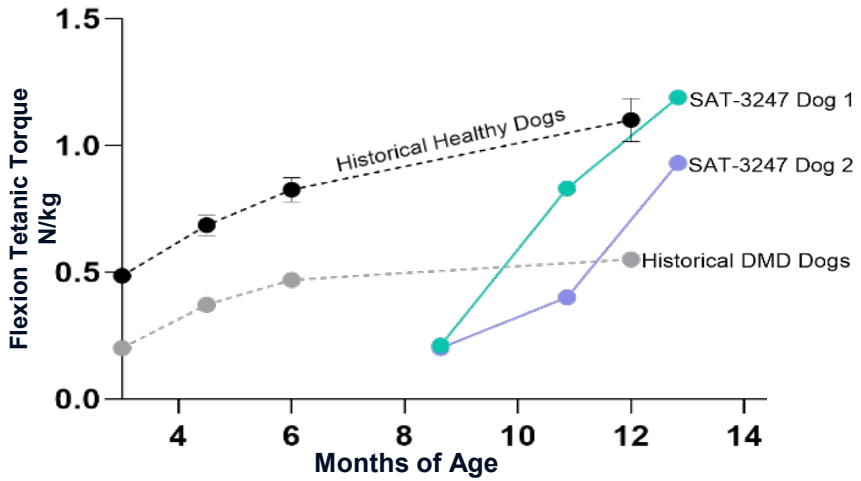
Preclinical and clinical results across canine and human studies

PRECLINICAL

Promising Effects in a Canine Model of DMD

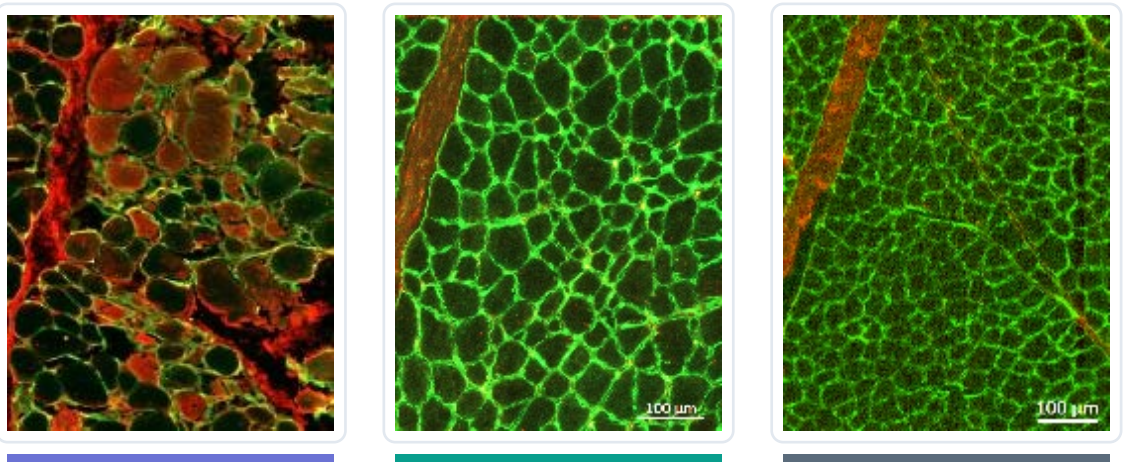
Muscle strength increased

Flexion tetanic torque — up to 2x and 4x at 2 and 4 months



Muscle regeneration enhanced

Immunofluorescence of bicep femoris biopsies at 4 months



Before treatment | **After treatment** | **Healthy animal**

4x

strength gain at 4 months

2x

strength gain at 2 months

STUDY OVERVIEW

- 2 DMD golden retriever / labrador canines, ~9 months of age at study start
- Oral SAT-3247 dosing: 10 mg/kg once daily (4 days on, 3 days off)
- 4 months of treatment; study terminated per institute protocol
- Serial functional and serum measurements, with biopsy (bicep femoris)

CLINICAL · PHASE 1

First-in-Human Trial of SAT-3247

PHASE 1a · HEALTHY VOLUNTEERS (n=72)

SAD study

10–400 mg · 5 cohorts of 8

Single dose / 1 day

MAD study

60–240 mg · 4 cohorts of 8

Single dose / 7 days

PHASE 1b · DMD ADULTS

28-day open-label study

- 60 mg daily dose, weekdays only for 4 weeks
- 5 patients enrolled and completed
- Age range 20 – 27, all on steroids

PRIMARY

Safety & tolerability — labs, vitals, ECG, exam

SECONDARY

Pharmacokinetic exposure (PK)

EXPLORATORY (1b only)

Grip/pinch strength, lung function, serum markers

SAT-3247 was well tolerated with a favorable safety profile across the Phase 1a & 1b studies

CLINICAL · PHASE 1B

SAT-3247-CL-101: Phase 1b of SAT-3247 Safety, Tolerability, PK, and Efficacy

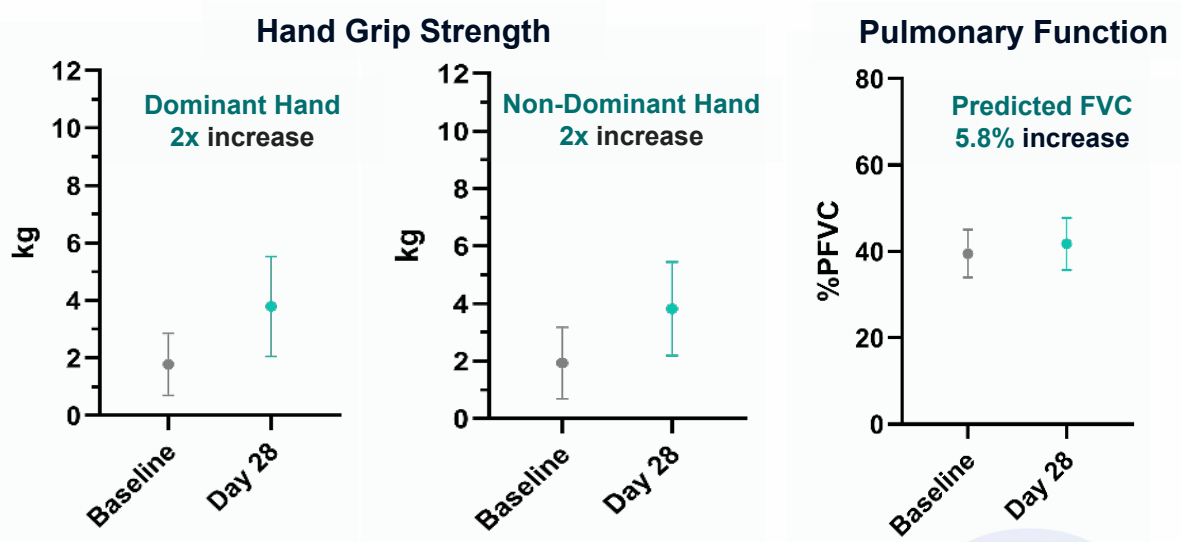
SAFETY SUMMARY

AE Category	SAD (n=40)	MAD (n=32)	FE (n=8)	Ph 1b (n=5)
Any TEAE	9	18	4	4
Any related TEAE	1[a]	2[b]	0	2[d]
Any serious TEAE	0	0	0	2
Any related serious TEAE	0	0	0	0
Any TEAE leading to withdrawal	0	1[c]	0	0
Any TEAE leading to death	0	0	0	0

[a] Mild nausea/abdominal pain (resolved)
[b] Mild somnolence (resolved), mild abdominal pain (resolved)
[c] Covid-19 (unrelated, resolved)
[d] Mild nausea (resolved), elevated ALT ((ref range (5, 40) – baseline(29), day 28(47) - resolved))

**PK: Pharmacokinetics; TEAE: Treatment Emergent Adverse Event; FVC: Forced Vital Capacity*

GRIP STRENGTH AND PULMONARY FUNCTION

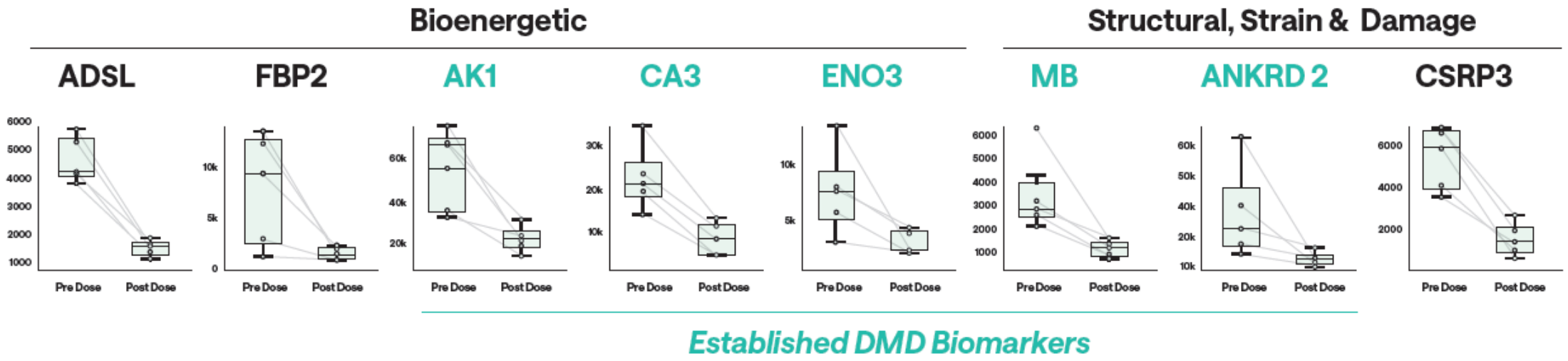


KEY TAKEAWAY

SAT-3247 demonstrated a **favorable safety profile**, predictable, dose-proportional PK, and **early signs of functional benefit** after 28 days of dosing in adults with Duchenne.

SAT-3247 Reduced DMD Biomarkers of Muscle Injury

The SomaScan® proteomic tool employs single-stranded DNA aptimers to capture & measure concentrations of ~11,000 proteins in biologic samples

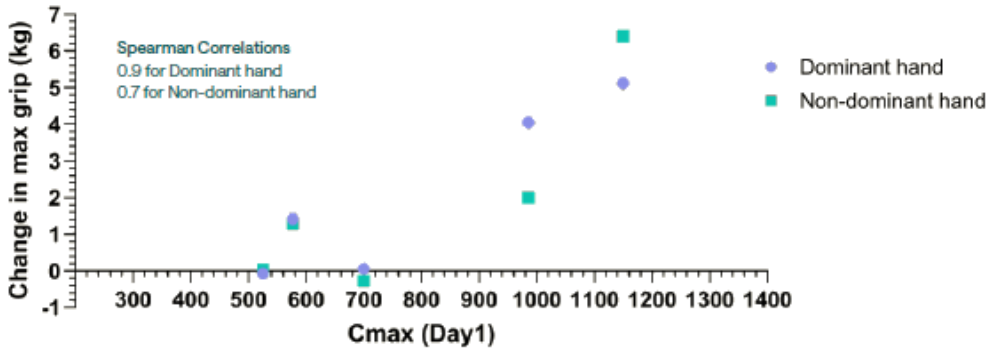


- Proteomic biomarker **changes were detected after just 15 days** of SAT-3247 dosing
- Confirms SAT-3247 is biologically active in the muscle of DMD patients
- Establishes a clear pathway to biomarker development for the program

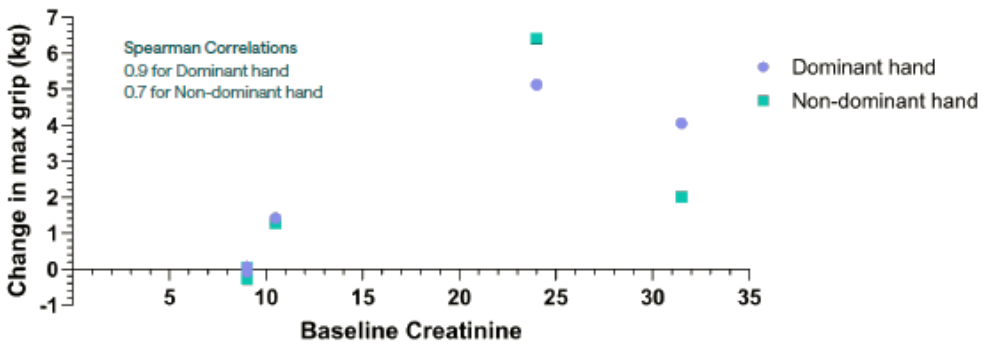
Gains Correlate with Exposure and Muscle Mass

Grip-strength gains track with both drug exposure **(Cmax)** and baseline muscle mass **(creatinine)** — strong Spearman correlations (0.9 dominant, 0.7 non-dominant).

MAXIMUM GRIP STRENGTH VS CMAX



MAXIMUM GRIP STRENGTH VS BASELINE CREATININE



POSITIVE IMPLICATIONS FOR BASECAMP

More muscle mass: the pediatric age group is expected to have more muscle mass than adults

Higher exposure: BASECAMP tests two dose levels, allowing for a higher potential Cmax

02

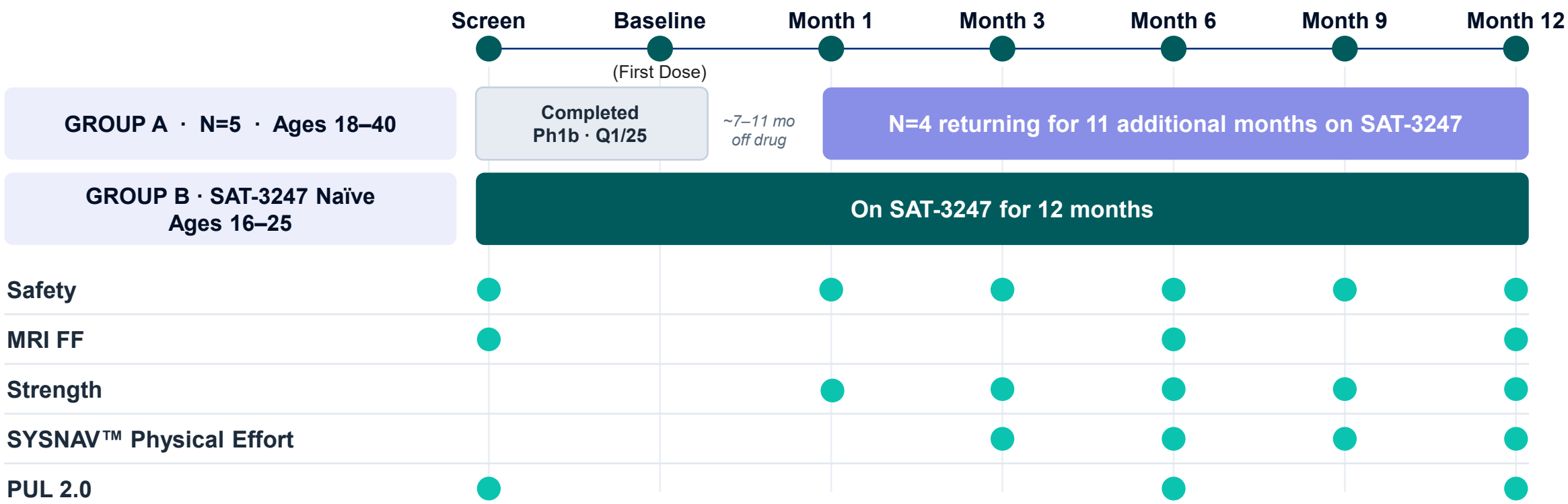
CLINICAL PROGRAMS

TRAILHEAD & BASECAMP

Two concurrent Phase 2 trials — adults and children

TRAILHEAD (NCT06867107) Phase 2 Study Design

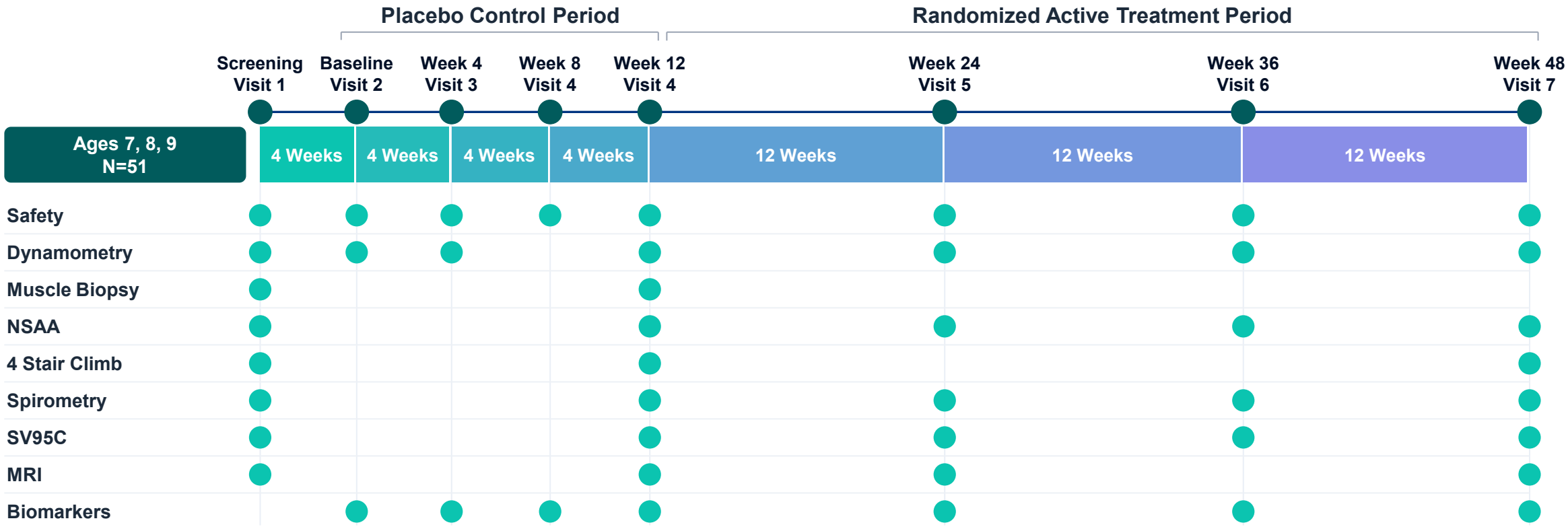
Open-label, long-term study of SAT-3247



*MRI FF=MRI fat fraction; PUL 2.0=Performance of Upper Limb 2.0; Strength=grip & pinch, elbow extension/flexion, shoulder abduction/flexion via dynamometry. *SYSNAV™ at protocol-defined visits.*

BASECAMP Phase 2 Study Design

12-week placebo-controlled period followed by a 36-week randomized active-treatment period.



Full enrollment of the BASECAMP study is expected Q3 2026

03

CLINICAL DATA

Six-Month TRAILHEAD Data

Reduced muscle fat fraction, increased total effort, stable strength and favorable safety following treatment with SAT-3247

6-MONTH READOUT

A Consistent Picture of Stable or Improving Outcomes

Six-month findings in four adults (aged 21–28) with Duchenne who originally enrolled in CL-101 — one of the most challenging populations in which to show treatment effects.

Favorable

Safety profile — no serious treatment-emergent adverse events (TEAEs), 100% compliance

−3.68%

MRI muscle fat fraction — reduced in all four participants

+~34%

Total effort (TE99C) — increased in all four participants

~2×

Handgrip near-doubling during CL-101 maintained

KEY TAKEAWAY

Across muscle composition, effort, strength, and safety, demonstrated outcomes were stable or improving through six months of SAT-3247 treatment.

SAT-3247 is an investigational agent not yet approved in any country or region

SAFETY & TOLERABILITY

SAT-3247 Well Tolerated; Favorable Safety Profile Maintained Through Six Months

0

serious TEAEs

0

TEAEs leading to withdrawal or discontinuation

100%

treatment compliance

186

days mean drug exposure

KEY TAKEAWAY

No serious or treatment-limiting safety signals were observed — supporting continued clinical evaluation of SAT-3247.

**Data as of 18 May 2026*

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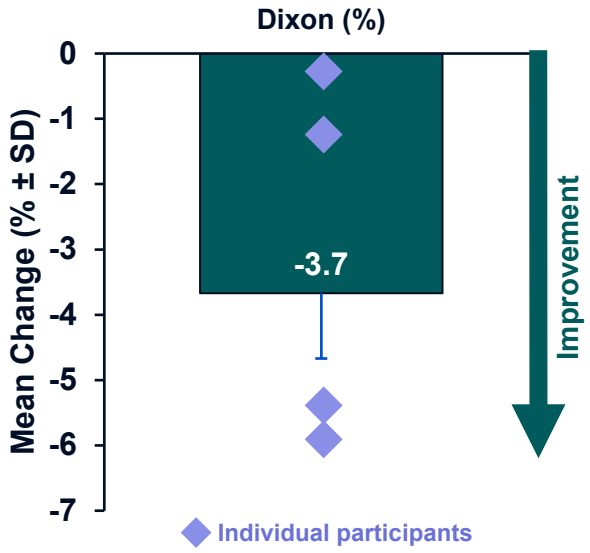
MRI Muscle Fat-Fraction Improved in All Participants

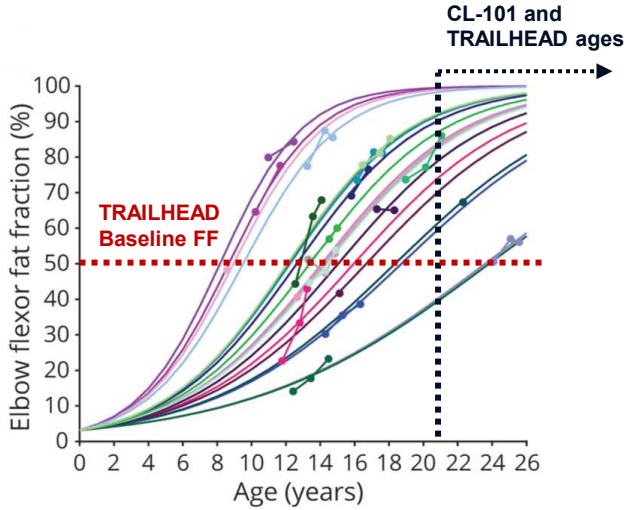
Individual Values and Changes

	Muscle MRI Fat Fraction (%)		
	Baseline	Month 5	Change
Participant 1	81.4%	75.3%	-6.2%
Participant 2	35.3%	34.4%	-0.9%
Participant 3	27.9%	22.1%	-5.8%
Participant 4	54.1%	52.2%	-1.9%
Mean ± SD	49.7 ± 23.9%	46.0 ± 23.1%	-3.7 ± 2.7%

Mean Changes



Natural History of Elbow Flexor Fat-Fraction[1]



[1]Naarding KJ, et al. Neurology 2021. 97:e1737-e1742
Elbow flexor muscles included biceps and brachialis muscles

KEY TAKEAWAY

Muscle fat fraction showed a decrease in every participant, compared with a 5.9%[1] annual increase reported in natural history studies.

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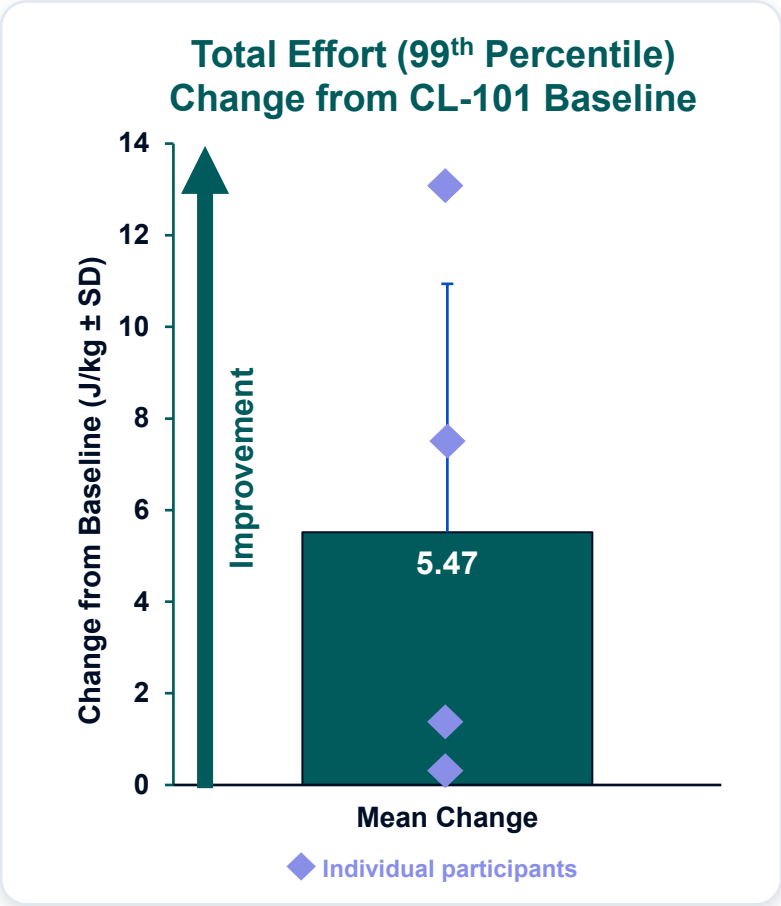
REAL-WORLD FUNCTION

Total Effort (TE99C) Improved Over Six Months



+~34%

From mean 16.1 joules/kg at baseline to 21.6 at month 6; increases observed in all four participants

TE99C (99th-percentile total effort) is captured continuously at home by the SYSNAV Syde® medical-grade wearable

KEY TAKEAWAY

Demonstrated increase in maximum effort in every participant — an emerging real-world activity signal that complements the imaging and strength findings.

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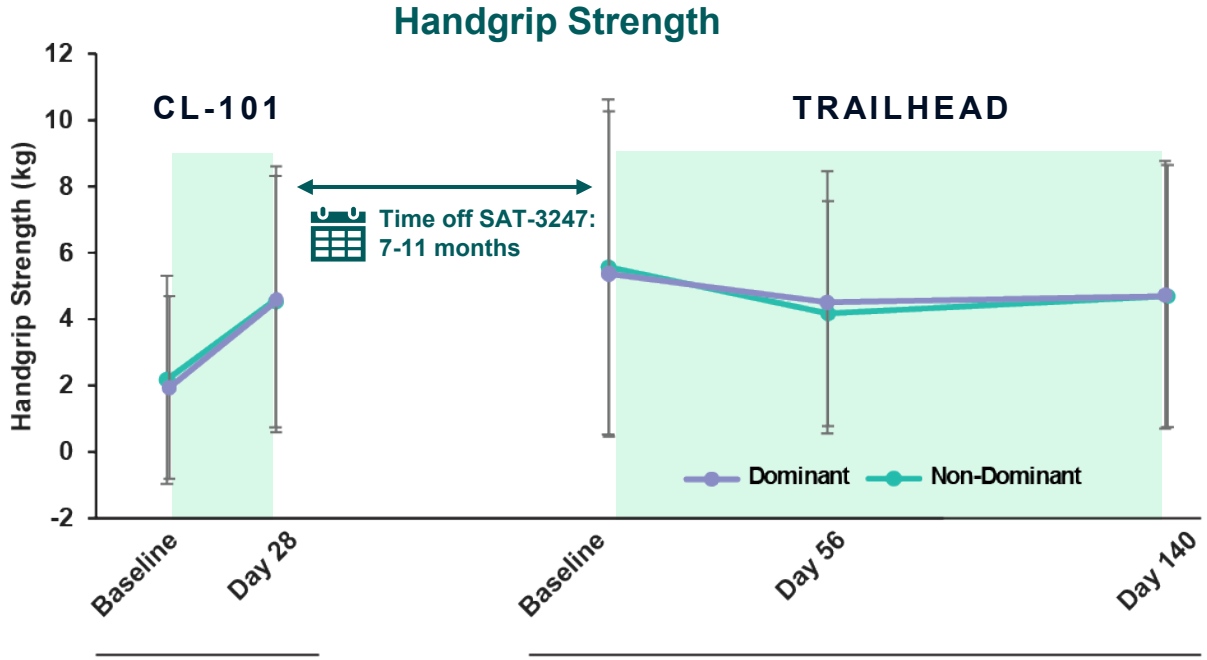
Upper-Extremity Strength Maintained

The near-doubling of handgrip strength seen in the 28-day CL-101 study was maintained through six months of TRAILHEAD follow-up.



STABLE ACROSS ALL MUSCLE GROUPS

- ✓ **Handgrip**
- ✓ **Elbow**
- ✓ **Shoulder**

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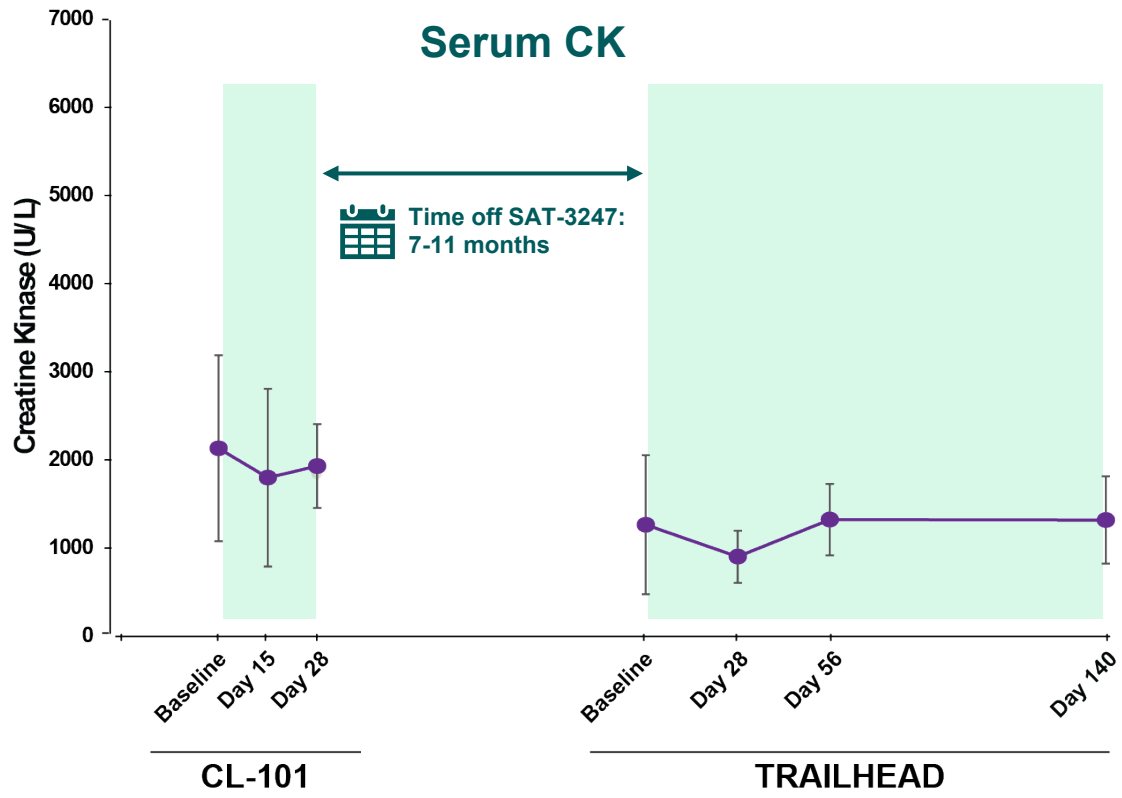
KEY TAKEAWAY

Strength demonstrated stability across every measure, with previously reported near-doubling handgrip strength preserved, not the decline expected in untreated adults.

Mean Serum Creatine Kinase (CK) Concentrations Demonstrated 38% Decline



Mean CK declined 38% from CL-101 baseline (2130 u/l) through month 6 in TRAILHEAD (1315 u/l).

CK levels in TRAILHEAD are lower than those observed in CL-101

After 28 days in CL-101, an interval of 7-11 months, and 140 days in TRAILHEAD

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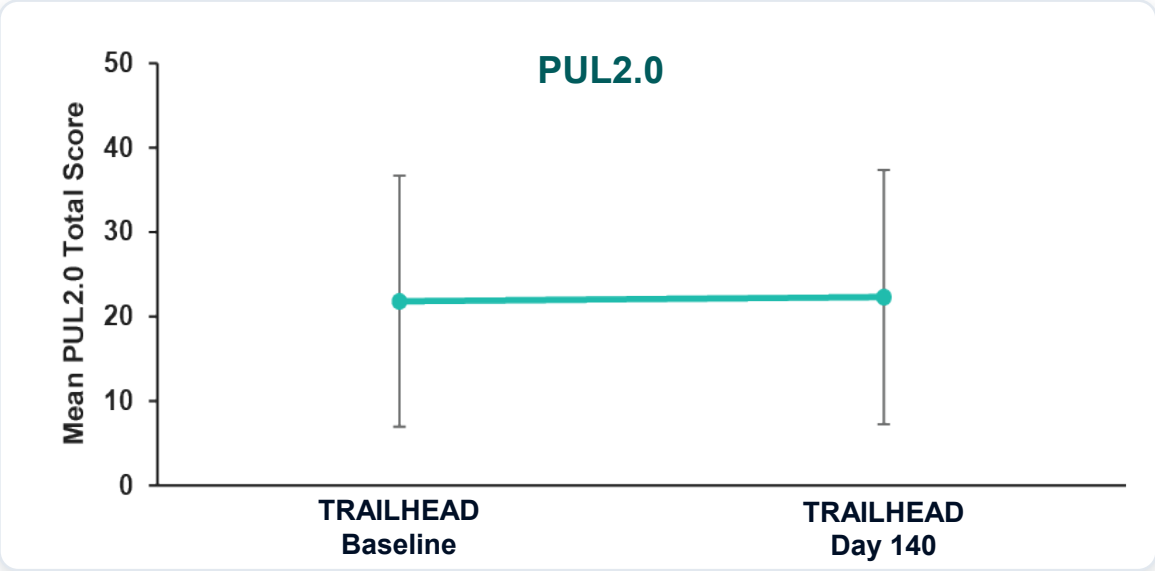
Performance of Upper Limb 2.0 (PUL2.0) Function Maintained



2 participants

improved by 1 point

2 participants

remained stable

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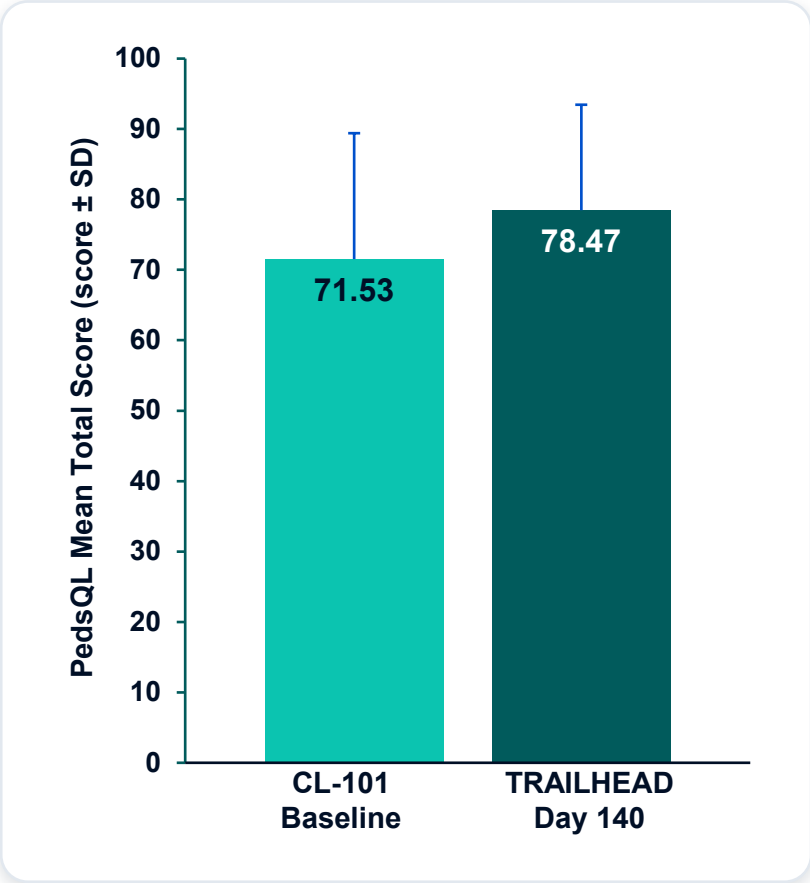
KEY TAKEAWAY

In the natural history of DMD, PUL2.0 function is generally expected to decline over time.[1]

Pane M, et al. *J Neuromuscul Dis*. 2023.

Patient Reported Quality-of-life Measure (PedsQL) Scores Demonstrated Improvement



+6.94

Mean PedsQL-MFS scores increased (CL-101 baseline → month 6 in TRAILHEAD)

PedsQL designed to quantify fatigue and adapted for adults

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03

THE COMPANY

Inside Satellos

Catalysts, financial position, and the road ahead

Clinical Catalysts: BASECAMP & TRAILHEAD

Q2

TRAILHEAD: FDA submission to start in U.S.

BASECAMP: trial-progress update

Q3

TRAILHEAD: progress / potential data

BASECAMP: anticipate completing enrollment, guidance update

Q4

TRAILHEAD: one-year Ph 1b data readout expected

BASECAMP: study completion & top-line data expected

Also, in 2H 2026: plan to submit an IND/CTA to initiate an FSHD clinical trial in the U.S. and Canada, with poster sessions and presentations throughout the year.

Financial Summary

Balance sheet supports execution on upcoming milestones

US$69.9M

Q1 2026 cash balance

24,281,712

common shares outstanding*

NASDAQ: MSLE **TSX: MSCL**

As of May 15, 2026; includes 3,450,522 pre-funded warrants outstanding.

Research analyst coverage

Canaccord	Tania Armstrong-Whitworth
Oppenheimer	Kostas Biliouris
Guggenheim	Debjit Chattopadhyay
H.C. Wainwright	Arthur He
Leede Financial	Douglas Loe
Leerink	Joseph P. Schwartz
Cantor	Yanni Souroutzidis

THE ROAD AHEAD

Reimagine

how Duchenne muscular dystrophy is treated

Regenerate

muscle with an oral, once-daily medicine

Realize

a new future for patients and families