Corporate Presentation

January 2026

SOLID BIOSCIENCES

Cautionary Note Regarding Forward Looking Statements

This presentation contains “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995, including statements regarding future expectations, plans and prospects for the company; the ability to successfully achieve and execute on the company’s goals, priorities and key clinical and preclinical milestones; strategies and expectations for the company’s SGT-003, SGT-212, SGT-501 and SGT-601 programs; expectations for additional site activations, planned enrollment, planned regulatory interactions and the potential approval pathways for SGT-003; timing of planned clinical trials of SGT-501; and other statements containing the words “anticipate,” “believe,” “continue,” “could,” “estimate,” “expect,” “intend,” “may,” “plan,” “potential,” “predict,” “project,” “should,” “target,” “would,” “working” and similar expressions. Any forward-looking statements are based on management’s current expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in, or implied by, such forward-looking statements. These risks and uncertainties include, but are not limited to, risks associated with the company’s ability to advance SGT-003, SGT-212, SGT-501, SGT-601 and other preclinical programs, capsid libraries and other enabling technologies on the timelines expected or at all; obtain and maintain necessary approvals from the FDA and other regulatory authorities; replicate in clinical trials positive results found in preclinical studies and early-stage clinical trials of the company’s product candidates; obtain, maintain or protect intellectual property rights related to its product candidates; replicate preliminary or interim data from early-stage clinicals trials in the final data of such trials; compete successfully with other companies that are seeking to develop Duchenne, FA, CPVT and other neuromuscular and cardiac treatments and gene therapies; manage expenses; and raise the substantial additional capital needed, on the timeline necessary, to continue development of SGT-003, SGT-212, SGT-501, SGT-601 and other candidates, achieve its other business objectives and continue as a going concern. For a discussion of other risks and uncertainties, and other important factors, any of which could cause the company’s actual results to differ from those contained in the forward-looking statements, see the “Risk Factors” section, as well as discussions of potential risks, uncertainties and other important factors, in the company’s most recent filings with the Securities and Exchange Commission. In addition, the forward-looking statements included in this press release represent the company’s views as of the date hereof and should not be relied upon as representing the company’s views as of any date subsequent to the date hereof. The company anticipates that subsequent events and developments will cause the company's views to change. However, while the company may elect to update these forward-looking statements at some point in the future, the company specifically disclaims any obligation to do so.

This presentation contains estimates and other statistical data made by independent parties and by us relating to market size and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such data and estimates. In addition, projections, assumptions and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk.

Shaping a Brighter Future for Tomorrow's Gene Therapies

PROGRAM	INDICATION	RESEARCH / DISCOVERY	PRECLINICAL	PHASE 1/2	PHASE 3	WORLDWIDE RIGHTS
Neuromuscular						
SGT-003	Duchenne muscular dystrophy	INSPIRE DUCHENNE				✓
		IMPACT DUCHENNE				✓
SGT-212	Friedreich's ataxia (FA)	FALCON				✓
Cardiac						
SGT-501	RYR2-Mediated CPVT	ARTEMIS				✓
	CASQ2-Mediated CPVT					✓
SGT-601	TNNT2 DCM					✓
SGT-401	BAG3-Mediated DCM					✓
SGT-701	RBM20 DCM					✓
Mayo Clinic Collaboration	Six Undisclosed Targets					✓
Platform						
Capsid Library[1]	Cardiac & NM					✓

Notes: In 2020, Solid entered into a collaboration agreement with Ultragenyx for the development of UX810, a next generation Duchenne construct comprised of Solid's proprietary nNOS microdystrophin and Ultragenyx's PinnacleTM PCL manufacturing platform for use with AAV8 and Clade E variants thereof. Solid has the option to co-fund collaboration programs in return for a profit share or increased royalty payments at proof-of-concept. 1. Cardiac Capsid Library currently in NHPs, Mice and Pigs.

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Consistent Execution in 2025 Positions Solid for a Pivotal 2026

Lead Programs	2025 *(achievements)*
AAV-SLB101 Proprietary Cardiac & Neuromuscular Capsid	✓ First-in-human data & interim update from Phase 1/2 INSPIRE DUCHENNE ✓ Exceeded 30+ executed licenses and agreements announced as of Nov. 3
SGT-003 Duchenne Muscular Dystrophy (Duchenne)	✓ Initial and interim Phase 1/2 INSPIRE DUCHENNE data ✓ Exceeded INSPIRE DUCHENNE participant enrollment targets ✓ Activated sites & enrolled first participant in ex-US Phase 3 trial, IMPACT DUCHENNE ✓ U.K. Innovative Licensing and Access Pathway (ILAP) designation granted
SGT-212 Friedreich's ataxia (FA)	✓ Announced FDA IND clearance ✓ Granted FDA Fast Track, Rare Pediatric Disease and Orphan Drug Disease designations ✓ Activated sites & enrolled first participant in Phase 1b clinical trial, FALCON
SGT-501 Catecholaminergic polymorphic ventricular tachycardia (CPVT)	✓ Announced FDA IND clearance and Health Canada CTA approval ✓ Granted FDA Fast Track designation ✓ Activated sites in Phase 1b clinical trial, ARTEMIS

2026: A Potentially Transformational Year

4 clinical programs in 3 markets with significant opportunity with multiple key anticipated clinical catalysts

Lead Programs	2026 *(anticipated milestones)*
AAV-SLB101 Capsid Library ***Building next-generation technology infrastructure***	• Continued out-licensing efforts designed to seed early-stage cardiac & neuromuscular research • Creation of proprietary next-generation capsids
SGT-003 Duchenne ***Establish a potential best-in-class Duchenne gene therapy***	• **Q1 2026:** First participant has been enrolled, with initiation of dosing expected in Phase 3 IMPACT DUCHENNE trial • **H1 2026:** Planned interactions with U.S. FDA for potential alignment on approval pathway • **Mid-2026:** Expected update on FDA interactions, including potential accelerated approval pathway[1]; additional data expected from INSPIRE DUCHENNE trial
SGT-212 FA ***Only therapy in development designed to address neurologic & cardiac manifestations***	✓ **Jan 2026:** First participant dosed in Phase 1b FALCON trial • **H2 2026:** Initial data expected from FALCON trial[2]
SGT-501 CPVT ***Potential first-in-class therapy with significant market opportunity***	• **Q1 2026:** First participant expected to be dosed in Phase 1b ARTEMIS trial • **H2 2026:** Initial safety data expected from ARTEMIS trial[2]

1. Expected timing, subject to scheduling and formal receipt of meeting minutes. 2. Subject to participant enrollment

SGT-003

Investigational Duchenne Muscular Dystrophy (Duchenne) Gene Therapy

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SGT-003 – Differentiated Profile With Significant Potential

Compelling Clinical Profile Demonstrated in Phase 1/2 INSPIRE DUCHENNE Trial

- Compelling levels of microdystrophin expression observed (Mean 58%, N=10, September 29, 2025, cutoff)
- Concordant restoration of DAPC & comprehensive improvements across muscle injury biomarkers suggest coordinated downstream effect (N=10-14, September 29, 2025, cutoff)
- Early data suggests potential cardiac benefit through observed troponin I reductions & improvements in systolic function as measured by LVEF (N=14, September 29, 2025, cutoff)

Safety and Tolerability Profile Continues to be Promising & Consistent

- SGT-003 has been generally well tolerated in all (N=33) participants dosed as of January 9, 2026[1]
- No drug-induced liver injury (DILI), thrombotic microangiopathy (TMA), atypical hemolytic uremic syndrome (aHUS) or myocarditis observed

Potential Significant Commercial Opportunity

- Majority of U.S. Duchenne prevalent population (~10-15k[2] with ~5k aged 0-11) has not been treated with a gene therapy
- U.S. incidence rate of at least 400 annual Duchenne births[3]
- EU population larger than U.S. population with no approved therapies available
- FDA-approved gene therapy, ELEVIDYS®, priced $3M+ per dose in U.S.
- **Pricing and available U.S. & EU patient populations supports an estimated $20B+ total addressable market**

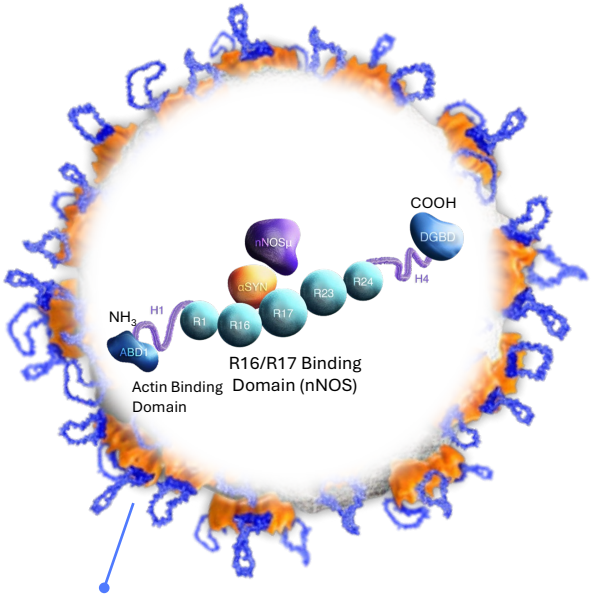
Near-Term Catalysts

- Multiple interactions with U.S. FDA expected throughout H1 2026 to align on Phase 3 confirmatory trial design and on necessary confirmatory evidence required to support potential accelerated approval

DAPC=dystrophin-associated protein complex; LVEF=left ventricular ejection fraction; TAM=total addressable market.
1. The 33rd participant was dosed in early January and therefore has limited safety data availability. 2. CureDuchenne – What is Duchenne? https://cureduchenne.org/about/what-is-Duchenne/. 3. MedlinePlus – Duchenne and Becker muscular dystrophy: https://medlineplus.gov/genetics/condition/duchenne-and-becker-muscular-dystrophy/#frequency.

SGT-003 Leverages Next-Generation Technology to Deliver Cutting Edge Duchenne Gene Therapy

SGT-003 is the only Duchenne gene therapy to use a next-generation capsid (AAV-SLB101)



Optimized Transgene Design

- Uniquely contains R16/R17 binding domain, which localizes nNOS to the muscle membrane
- nNOS restoration may help improve muscle perfusion and performance, reduce pathology and increase cardiac protection[1-6]

Minimally Burdensome Immunomodulation Regimen

- SGT-003 has been generally well-tolerated with no DILI, TMA, aHUS, myocarditis observed[7]
- Steroid-only prophylactic immunomodulatory regimen

Extensive Early Analysis Demonstrated Compelling Biologic Profile

- Interim data demonstrated compelling microdystrophin expression, reconstitution of key DAPC elements and improvement in a panel of muscle integrity biomarkers[8]

Lower Viral Dose & High Level of Capsid Purity

- Lower viral dose (1.0E14 vg/kg) vs. FDA-approved Duchenne gene therapy (1.33E14 vg/kg)
- ~75% full-to-empty capsid ratios at 1,000L GMP scale

nNOS=neuronal nitric oxide synthase; DILI=drug-induced liver injury; TMA=thrombotic microangiopathy; aHUS=atypical hemolytic uremic syndrome; DAPC=dystrophin-associated protein complex; GMP=good manufacturing practice.

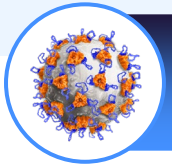
1. Tidball JC and Wehling-Henricks, M. *J Physiol.* 2014;592(Pt 21):4627-4638. 2. Lai Y, et al. *J Clin Invest.* 2009;119(3):624-35. 3. Rodino-Klapac LR, et al. *Hum Mol Genet.* 2013;22(24):4929-37. 4. Froehner SC, et al. *Hum Mol Genet.* 2014;24(2):492-505. 5. Zhang YH, et al. *J Physiol.* 2014;592(Pt 15):3189-3200. 6. Ziolo MT, et al. *J Mol Cell Cardiol.* 2012;45(5):625-632. 7. Safety cutoff as of January 5, 2026, N=33. 8. Data as of September 29, 2025.

Industry and Academic Interest in AAV-SLB101 Continues to Gain Momentum Quarter Over Quarter

At Solid, we believe that delivery is the foundation of gene therapy – and that improving delivery technologies will make gene therapies safer, more effective and more accessible for all

50+

Agreements, including licenses, executed with corporations, institutions and academic labs for use of AAV-SLB101[1]



"AAV-SLB101 Will Be the Workhorse for AAV Gene Therapy for Years to Come"

- Expert U.S. Duchenne Clinician

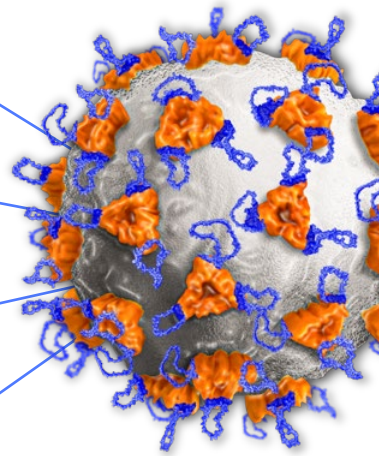
Contact our business development team to partner with us:
businessdevelopment@solidbio.com

1. Announced as of January 13, 2026.

AAV-SLB101 is a Next-Generation Capsid That has Demonstrated Rapid Transduction, Expression and Encouraging Safety Data

AAV-SLB101 was rationally engineered to include an RGD motif with high-affinity binding to integrin receptors that improve cardiac & skeletal muscle cell uptake and reduce liver biodistribution

- Improved skeletal & cardiac muscle tropism with reduced off-target effects observed in preclinical models
- Designed with intention to reduce liver biodistribution
- Rapid transduction and transgene expression observed in preclinical models
- Improved product purity, yields and COGS

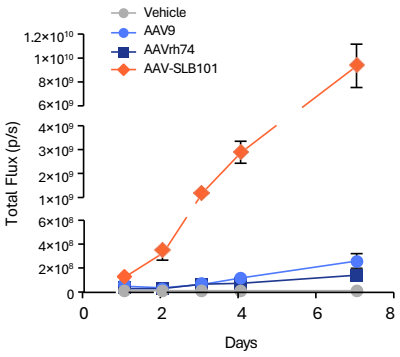


Data on file. Solid Biosciences.

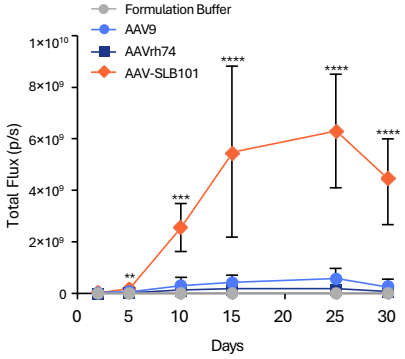
AAV-SLB101 has Demonstrated Rapid and Robust Transduction and Enhanced Expression Compared to First-Generation Capsids

Luciferase Activity Measured by Whole Body IVIS

5E13 vg/kg



2E12 vg/kg

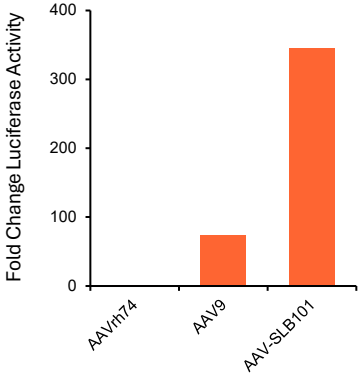


Rapid transduction with expression via AAV-SLB101 observed as early as 2 days post-dose, remaining significantly higher than AAV9 or AAVrh74

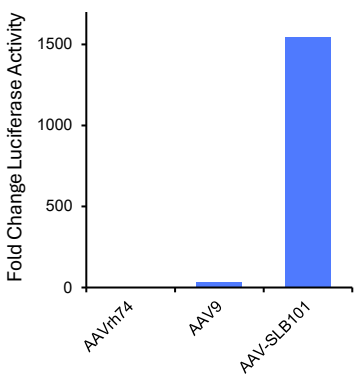
Luciferase Activity in Isolated Tissues

2.0E12 vg/kg (n=5 per tissue/capsid)

HEART



QUADRICEPS



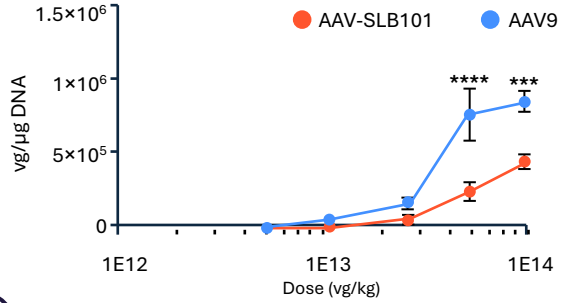
Highly improved target tissue expression achieved with AAV-SLB101 at low dose vs AAV9 or AAVrh74

IVIS=*in vivo* imaging system

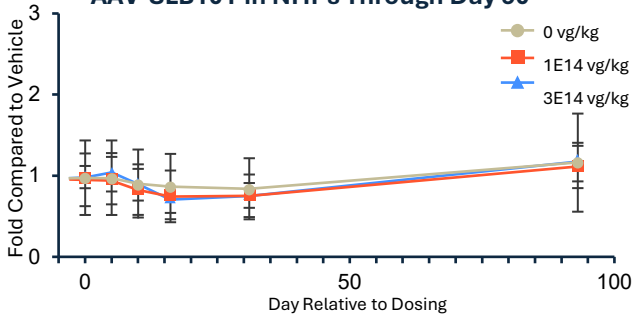
Compelling AAV-SLB101 Preclinical Liver Detargeting Data Reflected in Phase 1/2 INSPIRE DUCHENNE Trial

As of January 9, 2026, 33 participants have been dosed with no drug-induced liver injury observed

Decreased Liver Biodistribution in Mice[1]

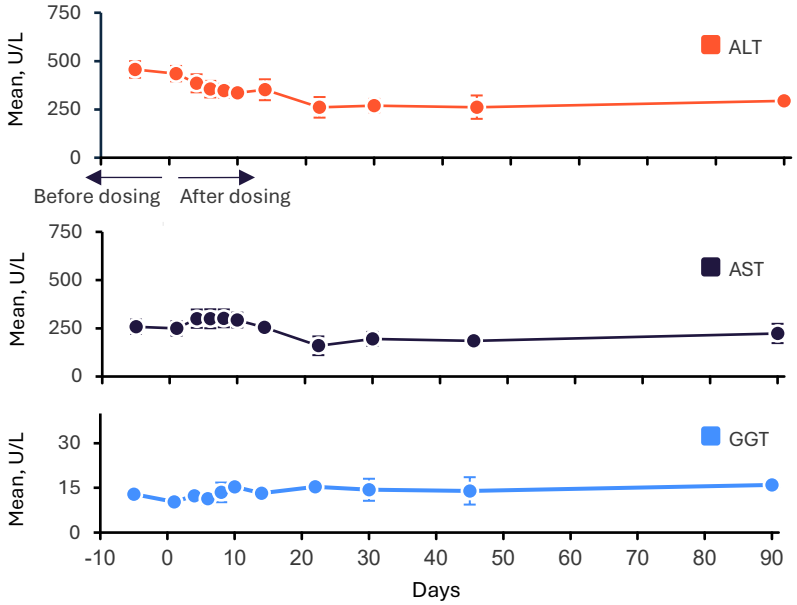


Stable GGT Levels at Multiple Dose Levels of AAV-SLB101 in NHPs Through Day 90[1]



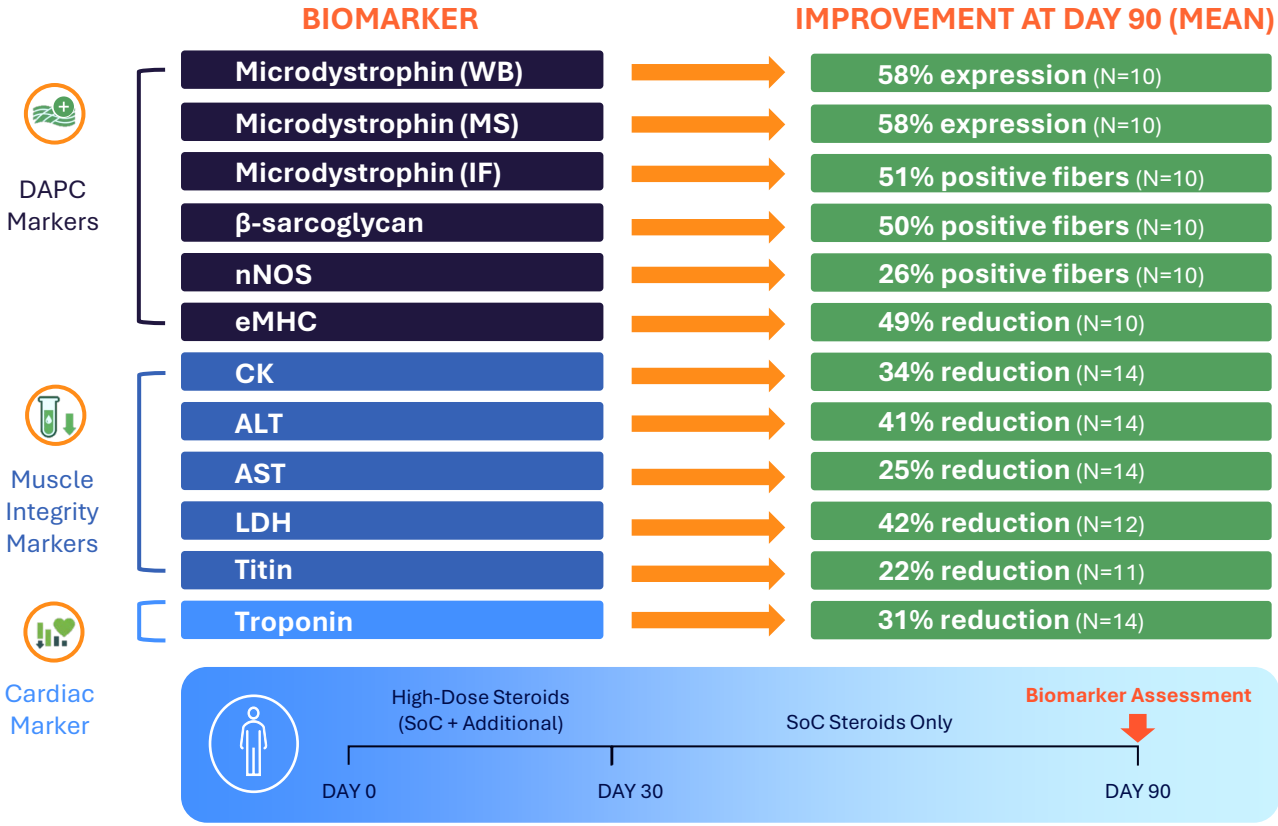
Liver Biomarkers – INSPIRE DUCHENNE Trial[2]

n=14



P-values: ***p<0.001; ****p<0.0001. NHP=Non-human primate; ALT=alanine transaminase; AST=aspartate aminotransferase; GGT=gamma-glutamyl transferase.
1. Data on file. Solid Biosciences. 2. Data cutoff of September 29, 2025. Values are means ± standard error of the mean.

Thorough Analysis of Skeletal & Cardiac Muscle Biomarkers Suggests Beneficial Biological Treatment Effect & Improved Muscle Integrity



Comprehensive improvements across 12 biomarkers contribute to desired target product profile (TPP)

Minimally burdensome prophylactic regimen supported by rigorous safety monitoring

WB=western blot; MS=mass spectrometry; IF=immunofluorescence; nNOS=neuronal nitric oxide synthase; eMHC=embryonic myosin heavy chain; CK=creatine kinase; ALT=alanine transaminase; AST=aspartate aminotransferase; LDH=lactate dehydrogenase; SoC=standard-of-care. Data as of September 29, 2025. Solid Biosciences.

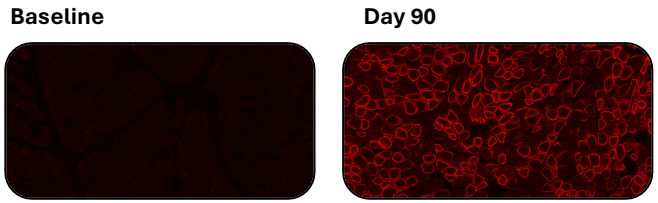
Microdystrophin Transduction and Expression Observed at Days 90 & 360 Post SGT-003 Treatment

Comprehensive orthogonal measurements showed consistent microdystrophin expression across three measures

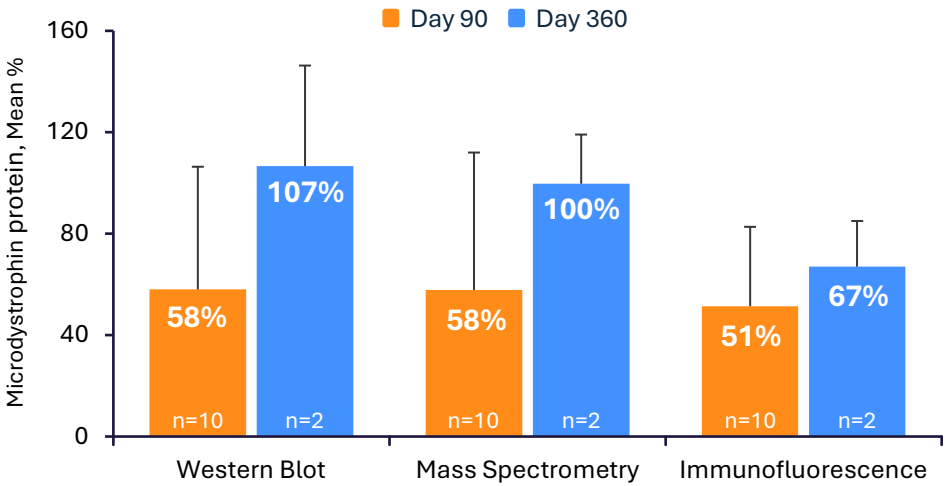
Microdystrophin Transduction

DOSE	VECTOR COPIES/NUCLEUS DAY 90 (N=10)	VECTOR COPIES/NUCLEUS DAY 360 (N=2)
1.0E14 vg/kg	13	12

Example Microdystrophin Biopsy[2]

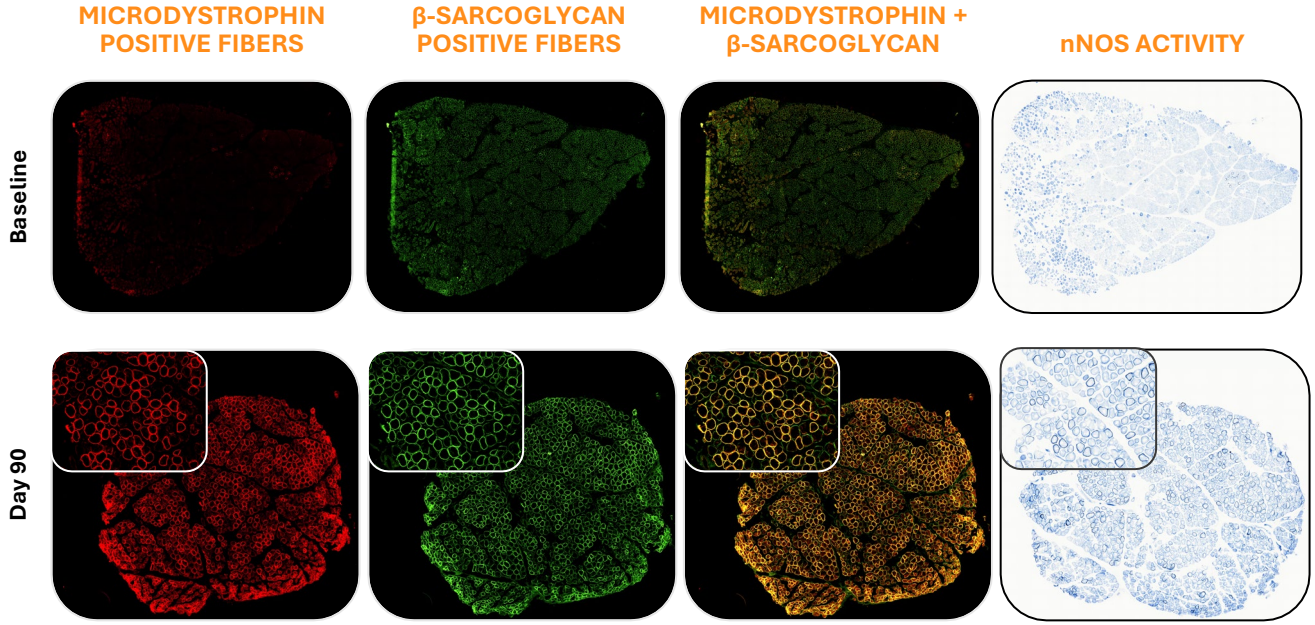


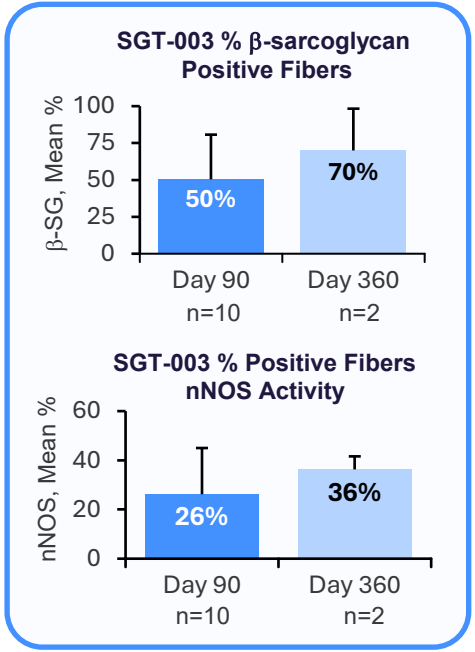
Mean SGT-003 Microdystrophin Expression[1]



1. Data cutoff of September 29, 2025. Solid Biosciences. Baseline Western Blot and Mass Spectrometry were both 0% mean normal dystrophin. Baseline mean dystrophin positive fibers were 1.5% measured by IF. Dystrophin positive fibers are not adjusted for fat and fibrosis; these are absolute numbers. 2. Representative images are shown.

Microdystrophin Positive Fibers With Concordant DAPC Restoration Observed After SGT-003 Treatment



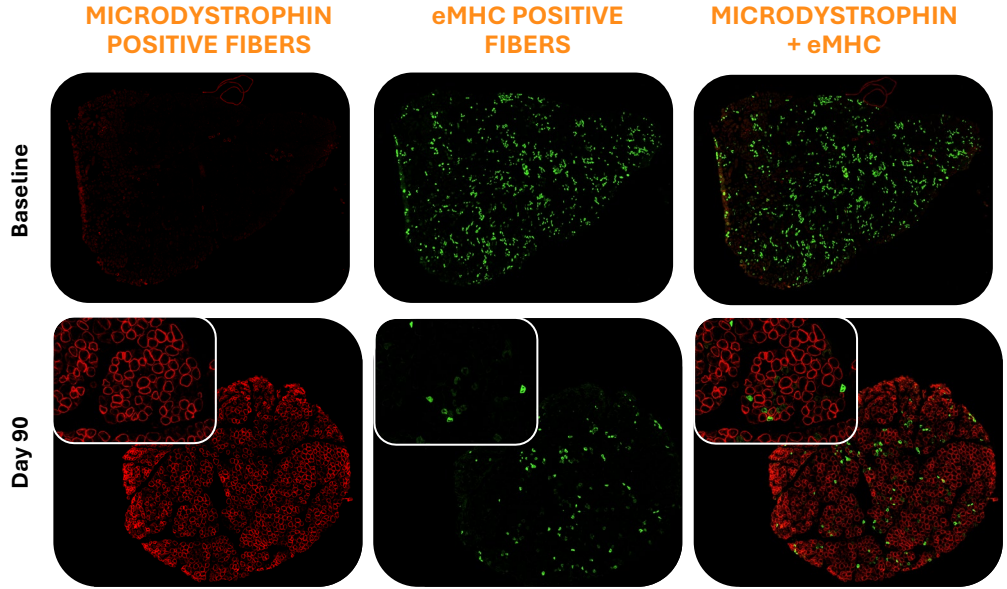


SGT-003 microdystrophin positive fibers (%) achieved a statistical correlation with both β-sarcoglycan positive fibers (%) and nNOS activity positive fibers (%) ($r_{Pearson}$ =0.95)

β-SG= β-sarcoglycan.
Data cutoff of September 29, 2025. Solid Biosciences. β-SG was measured by immunofluorescence and nNOS was measured using an activity assay. Representative images from the same participant are shown.

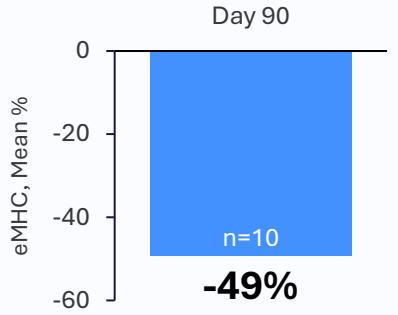
Full Slide Scans of Muscle Biopsy Sections Showed Uniform Improvements in eMHC[1,2]

Muscle stem cells (satellite cells) are activated to repair & replace damaged muscle fibers–during this process, newly formed, dystrophic muscle fibers transiently express embryonic myosin heavy chain (eMHC)[1,2]



A treatment-mediated decrease in eMHC is favorable and may suggest muscle preservation

SGT-003 % eMHC Positive Fibers



% positive microdystrophin fibers & the reduction in eMHC positive fibers at Day 90 were **negatively correlated**

($r_{Pearson}$ = -0.51)

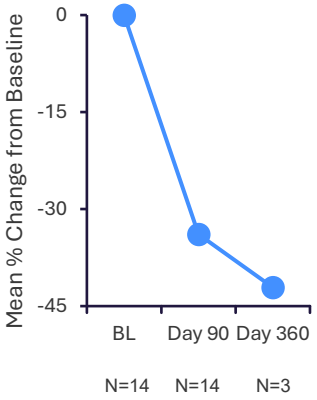
Data cutoff of September 29, 2025. Solid Biosciences. Representative images from the same participant are shown.

1. Dubuisson N, et al. *Int J Mol Sci*. 2022;23(24):16080. 2. Cardone N, et al. *Acta Neuropathol Commun*. 2023;11(1):167.

Improved Measures of Muscle Integrity and Resilience Were Observed After SGT-003 Treatment

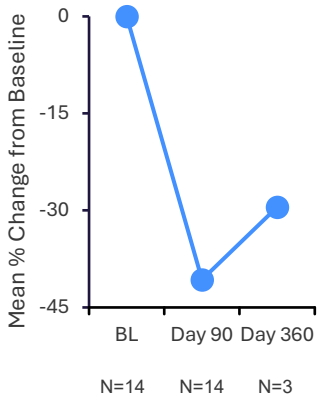
Comprehensive and thorough assessment of muscle injury and metabolism

SERUM CK % Change From Baseline



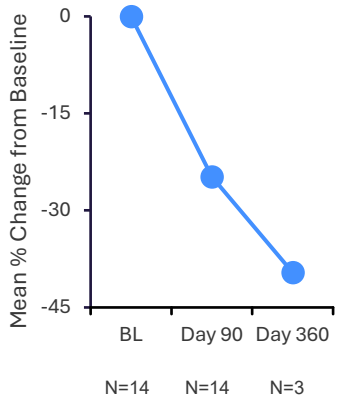
- ⬇ 34% at Day 90
- ⬇ 42% at Day 360

SERUM ALT % Change From Baseline



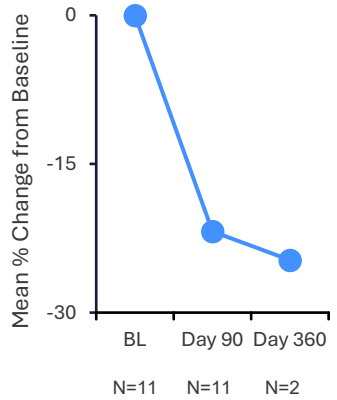
- ⬇ 41% at Day 90
- ⬇ 29% at Day 360

SERUM AST % Change From Baseline



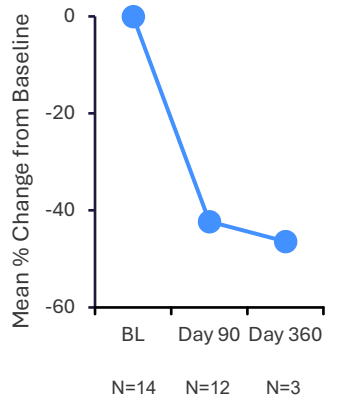
- ⬇ 25% at Day 90
- ⬇ 40% at Day 360

SERUM Titin % Change From Baseline[a]



- ⬇ 22% at Day 90
- ⬇ 25% at Day 360

SERUM LDH % Change From Baseline[b]

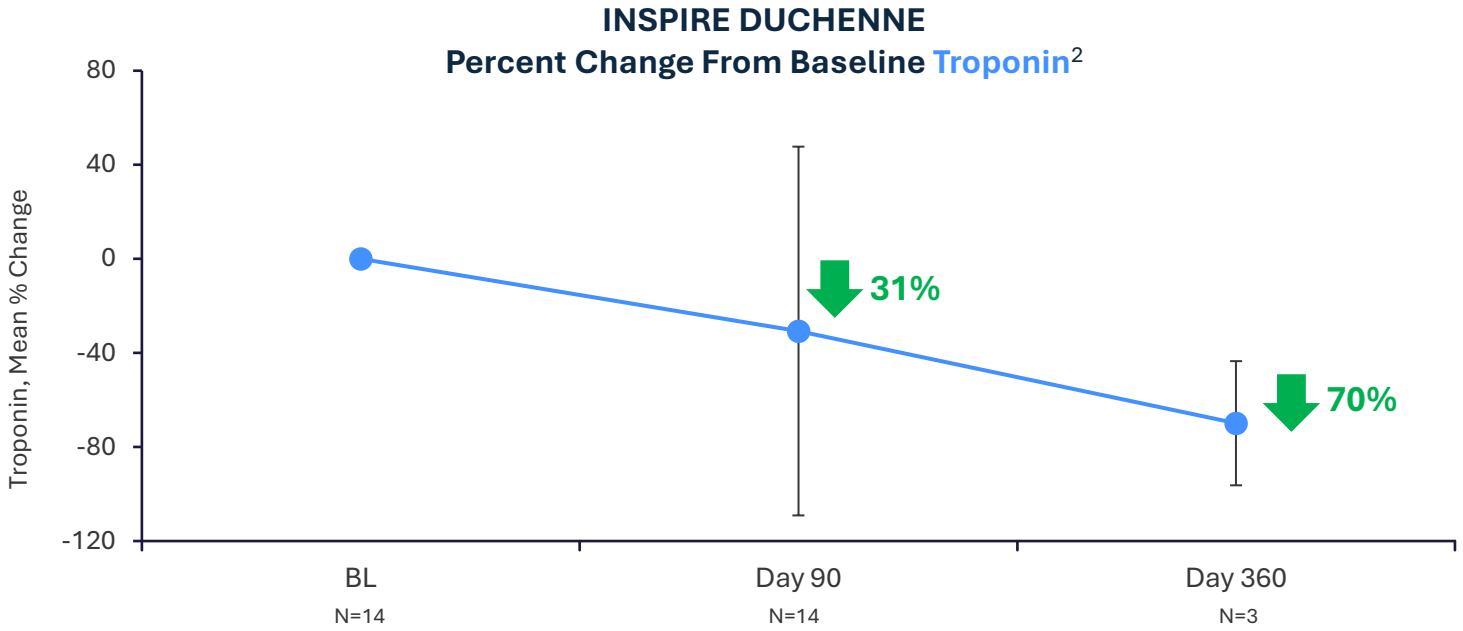


- ⬇ 42% at Day 90
- ⬇ 46% at Day 360

[a]Titin was batch analyzed at an earlier data cutoff date. [b]Two LDH samples hemolyzed at day 90 and are not available for inclusion.
Data cutoff of September 29, 2025. Solid Biosciences.

Troponin Reductions May Indicate Early Signals of SGT-003 Cardiac Treatment Effect

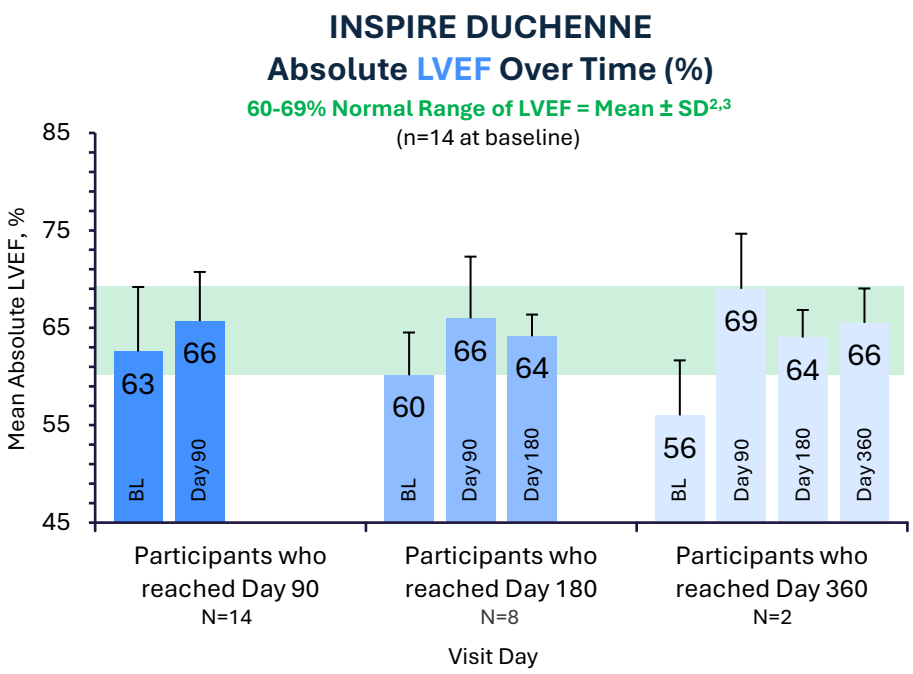
Troponin I is released during myocardial cell injury, acting as a signal of muscle breakdown and a surrogate for cardiac myocyte damage[1]

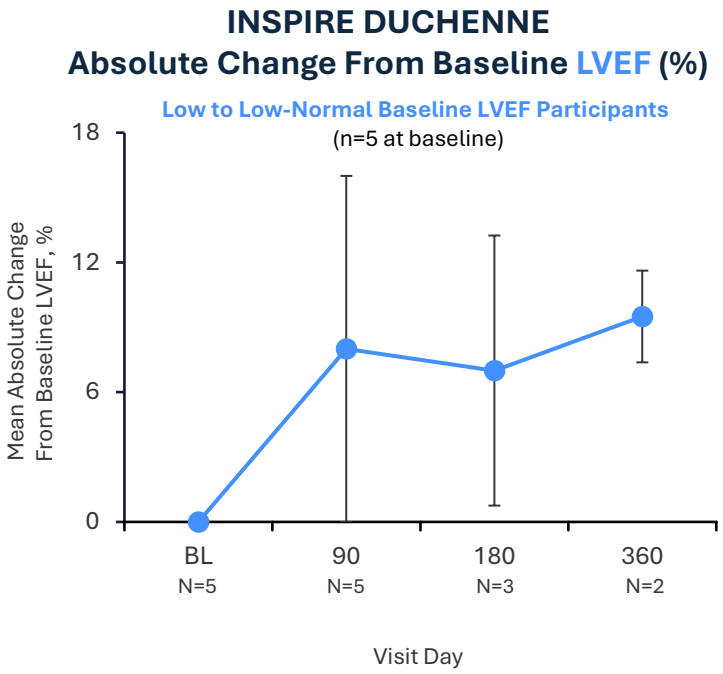


1. Sheybani A, et al. *Pediatr Res*. 2022;92(6):1613-1620. 2. Data cutoff of September 29, 2025. Solid Biosciences.

Stable-to-Improved Cardiac Function Observed After SGT-003 Dosing

Early observations of improved cardiac function driven by participants with low to low-normal baseline left ventricular ejection fraction (LVEF)[1]





BL=baseline; CFB=change from baseline.

1. Data cutoff of September 29, 2025. Solid Biosciences. 2. The mean ± SD normal LVEF range is 60% to 69% for this age-matched population. 3. Romanowicz J, et al. *J Am Soc Echocardiogr*. 2023;36(3):310-323.

INSPIRE DUCHENNE Interim Safety Summary for All Participants

33 participants have received SGT-003 (1.0E14 vg/kg) at ages ranging from 1 to 10 years as of January 9, 2026

COHORTS	ELIGIBLE AGE RANGE (YEARS)	AGE AT ENROLLMENT (YEARS)	WEIGHT FOR DOSING (KG)	PARTICIPANTS ENROLLED (N)
1-3	0 to <12	1 to 10	9.9 to 39.7	33

SGT-003 TREATMENT-RELATED ADVERSE EVENTS as of January 9, 2026 (N=33)[1]		N (%)
Serious Adverse Events (SAEs)		1 (3.0)[2]
Most Common Treatment-related Adverse Events (AEs)	Vomiting	22 (66.7)
	Nausea	21 (63.6)
	Thrombocytopenia / Platelet Count Decreased	14 (42.4)
	Decreased Appetite	11 (33.3)
	Headache	6 (18.2)

Based upon compelling safety & tolerability profile, **SGT-003 is now being administered in an outpatient setting**

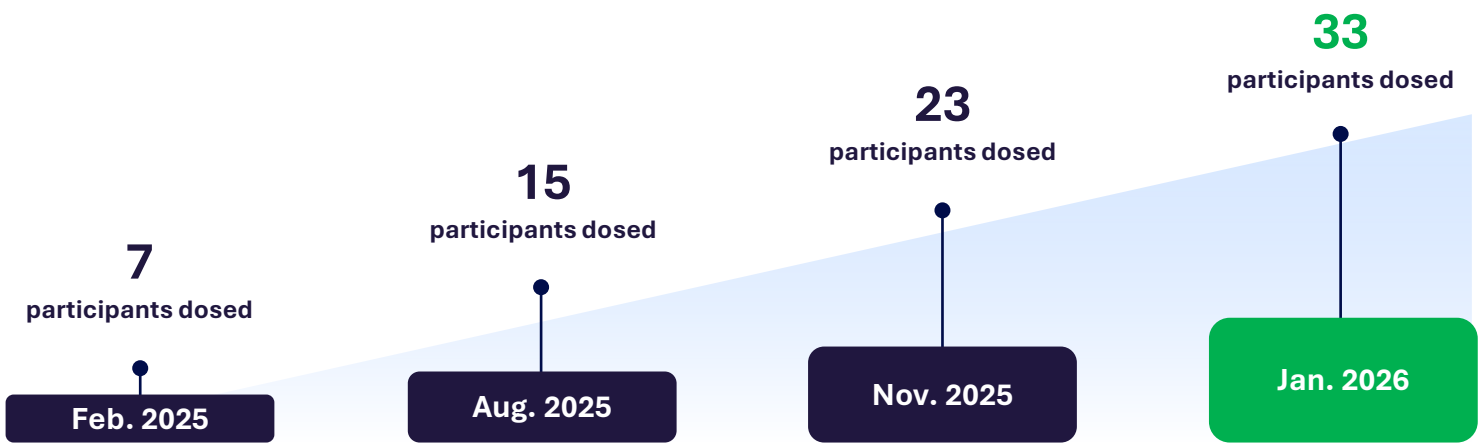
2. One (n=1) CTCAE Grade 3 serious adverse event of immune-mediated myositis. The myositis was not associated with muscle pain or weakness. The participant responded promptly to steroid treatment, with all clinical symptoms noted at presentation resolving and with CK levels declining well below baseline.

CTCAE, common terminology criteria for adverse events.
1. The 33rd participant was dosed in early January and therefore has limited safety data available.

Acceleration of Participant Dosing Reflects Participant Demand

Participant enrollment rate in the INSPIRE DUCHENNE trial has repeatedly outpaced prior guidance, suggesting strong Duchenne community appetite for new therapies

INSPIRE DUCHENNE



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2026: Integrated U.S. & Ex-U.S. Pivotal Trial Strategy Underway to Support Potential Accelerated Regulatory Authorizations

Novel therapeutic design, rapid participant enrollment, favorable tolerability profile and robust biomarker data continue to build position of strength for SGT-003 both in the U.S. and ex-U.S.

INSPIRE DUCHENNE

Phase 1/2 open-label, non-randomized clinical trial designed to support potential accelerated approval in the U.S.

STATUS: Active & enrolling ambulatory boys aged 0 to < 12; N=33 dosed as of January 9, 2026

PRIMARY ENDPOINT: Change from baseline in microdystrophin protein levels; incidence of TEAEs

TRIAL SITES: 15 sites activated across U.S., Canada, U.K. & Italy

IMPACT DUCHENNE

Phase 3 randomized, double-blind, placebo-controlled clinical trial designed to support ex-U.S. regulatory authorizations

STATUS: Active & enrolling ambulatory boys aged 7 to 11

PRIMARY ENDPOINT: Change from baseline in Time to Rise (TTR) from supine at Day 540 (18 months)

TRIAL SITES: 2 sites activated in Canada and Australia with planned expansion into additional countries, including in Europe, beginning in mid-year 2026, subject to regulatory approvals

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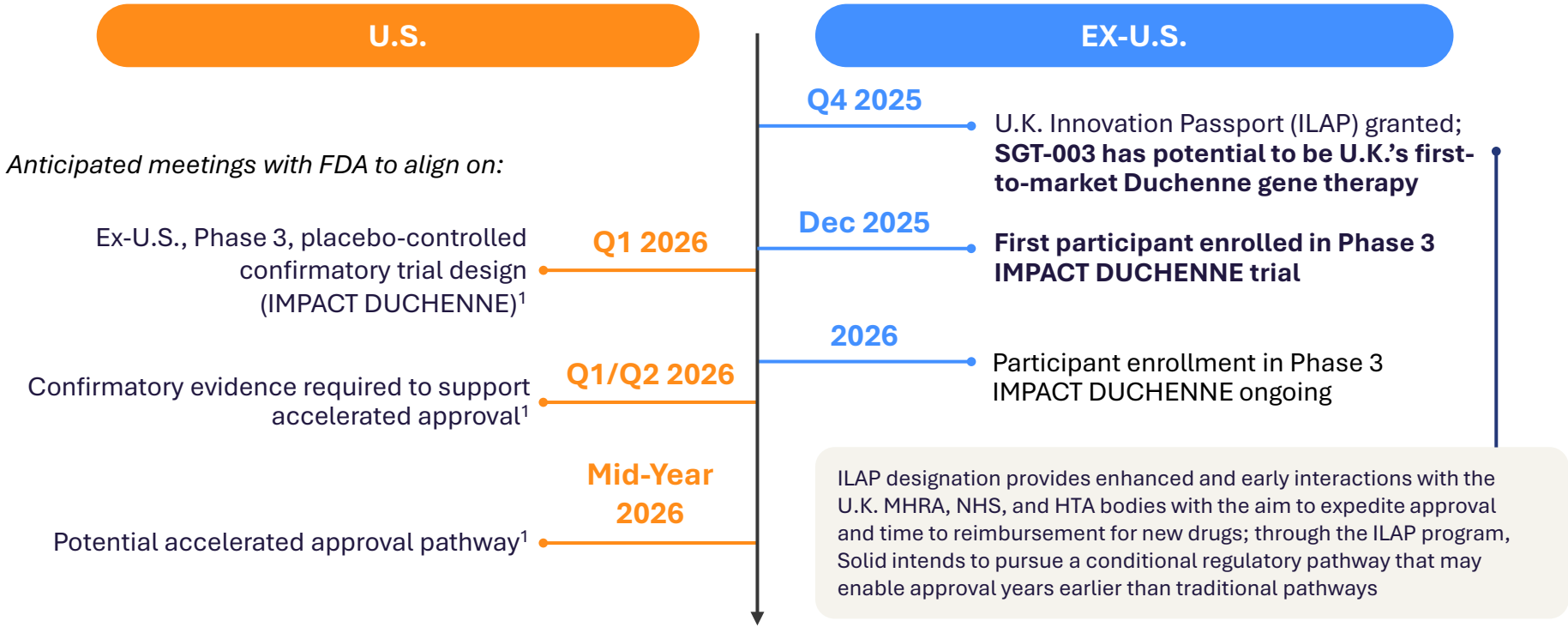
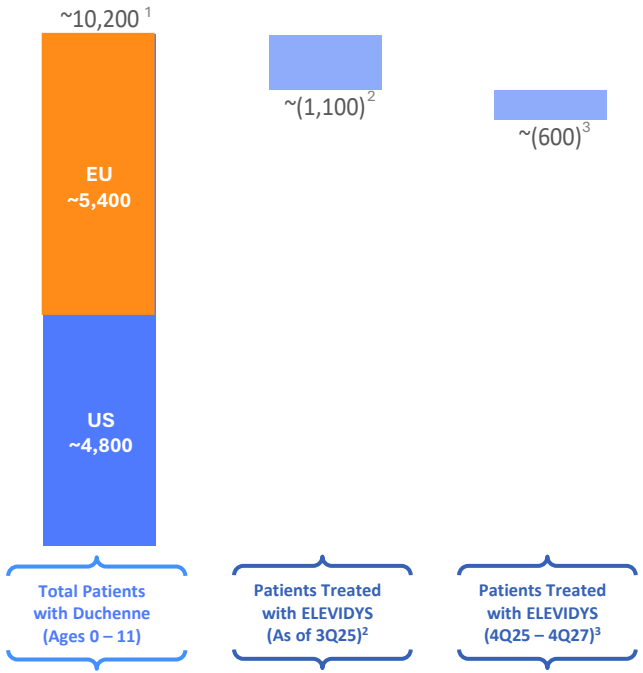
2026: Integrated U.S. & Ex-U.S. Pivotal Trial Strategy Underway to Support Potential Accelerated Regulatory Authorizations *(cont.)*



1. Expected timing, subject to scheduling.

Duchenne Remains a Mega-Blockbuster Commercial Opportunity

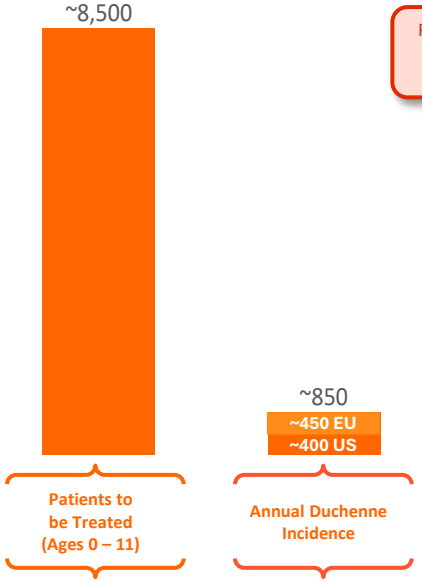
US and EU Duchenne Patients (Ages 0 – 11)



Potential Market Opportunity – Available Ambulatory

~$25B Patients to be Treated + ~$3B Annual Incidence = ~$28B+ Market Opportunity

Assuming a net price of $3M per patient[4]



Potential to expand further to APAC and non-ambulatory patients

Vast majority of treatment-eligible Duchenne patients remain undosed, with current revenue trajectory well below incident population

Potential opportunities:

- Currently eligible, but untreated
- Younger patients (< 4 years) becoming eligible
- Annual Duchenne incidence
- Ex-US patients

1. Estimates based on annual birth rates in the EU, assuming 50% male births, and a Duchenne prevalence rate of 1 in every 3,500-5,000 male births, and annual incidence rate of 400 Duchenne births in the US, in the 0 through 12 year population. 2. Sarepta Therapeutics' filings and website; includes global clinical and commercial patients treated with ELEVIDYS. 3. Wall Street Research, data as of December 2025: estimated ~600 US patients will be treated with ELEVIDYS between 4Q25 and 4Q27 based on broker sales projections of ~50–70 patients / quarter and assumes no patients in EU as ELEVIDYS is not approved. 4. Estimate only, actual pricing not yet determined and is subject to applicable regulatory approval on pricing.

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SGT-212

Investigational Friedreich’s Ataxia (FA) Gene Therapy

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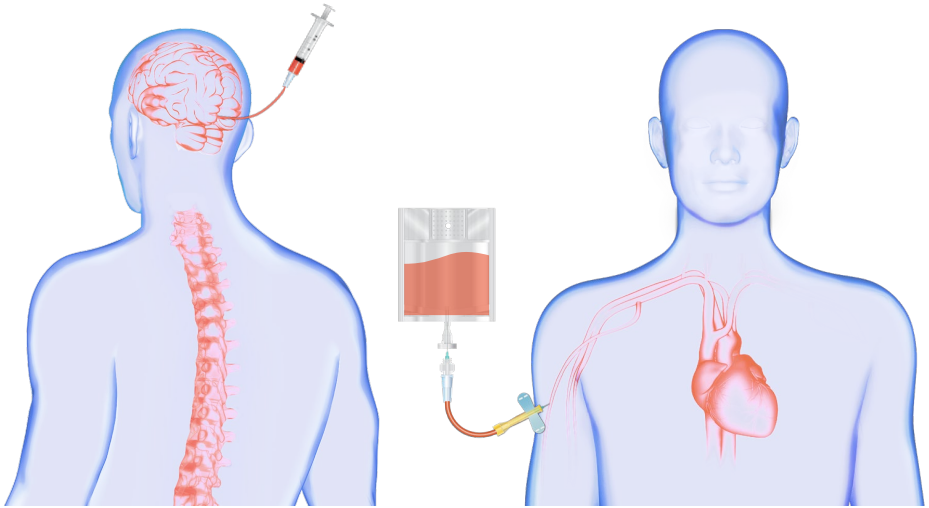
SGT-212 Leverages Precision Technology to Address Quality of Life and Cardiac Mortality

SGT-212 is the only dual route gene therapy (IDN & IV) approach for the treatment of Friedreich's ataxia – designed to address the neurologic, systemic and cardiac manifestations of the disease

SGT-212:

- *Capsid*: AAVhu68
- *Dosing Cadence*: IDN first, followed by IV
- Ubiquitous promoter



Slowing or halting disease progression is the most meaningful treatment impact for the FA community[1]

Treatment approaches that address both quality of life *and* mortality/lifespan represent the future **standard for FA disease management**

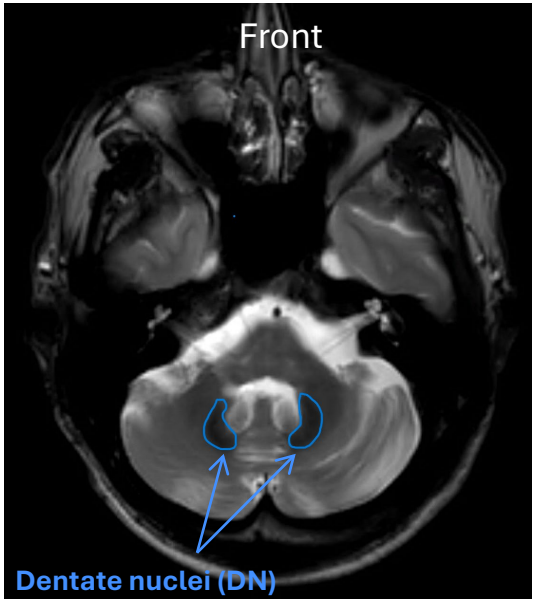
IDN=intradentate nuclei; IV=intravenous

1. Friedreich's Ataxia Research Alliance (FARA) *Voice of the Patient* Survey, 2017.

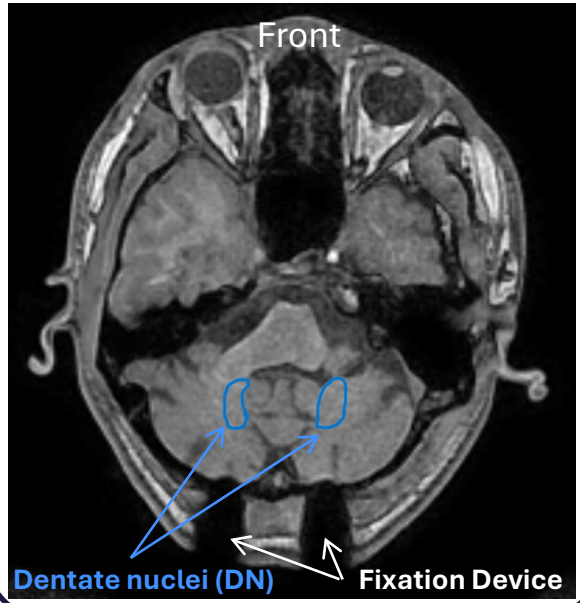
SGT-212: First Participant Dosed in Phase 1b FALCON Trial

Intra-procedural MRI-imaging confirmed precise delivery of SGT-212 into the dentate nuclei

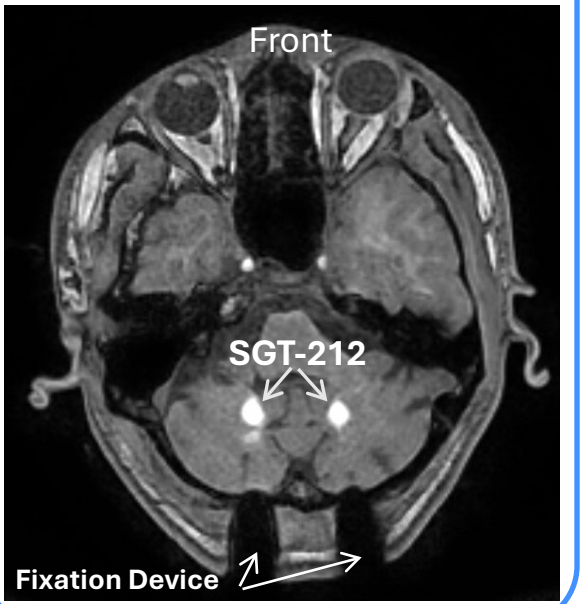
Baseline MRI



Pre-Treatment With SGT-212



Post-Treatment With SGT-212



FALCON Phase 1b Trial Design

A first-in-human, open-label, multi-center trial designed to evaluate the safety and tolerability of contemporaneous IDN and systemic IV infusion of SGT-212 gene therapy in participants with FA

Cohorts

Study to enroll approximately 10 participants aged 18-40 years with FA and documented cardiac hypertrophy

1. **Non-ambulatory**
2. **Ambulatory**
3. **Ambulatory & Non-Ambulatory** *(Optional)*

Study Endpoints

Primary Endpoint

Incidence and severity of TEAEs from baseline to month 12

Secondary & Exploratory Endpoints

Change from baseline in serum biomarkers

Change from baseline cardiac frataxin expression at Day 90 and Month 18

Change from baseline in assessments measuring key aspects of the disease, such as neuromuscular function, fatigue and speech

Change from baseline cardiac structure and function

Community Interest

- Multiple inquiries received from interested FA community members located both in and ex-U.S.
- Solid has collaborated with experts from 5 leading institutions to develop, test and refine the stereotactic, MRI-guided neurosurgical IDN-dosing procedure used in FALCON

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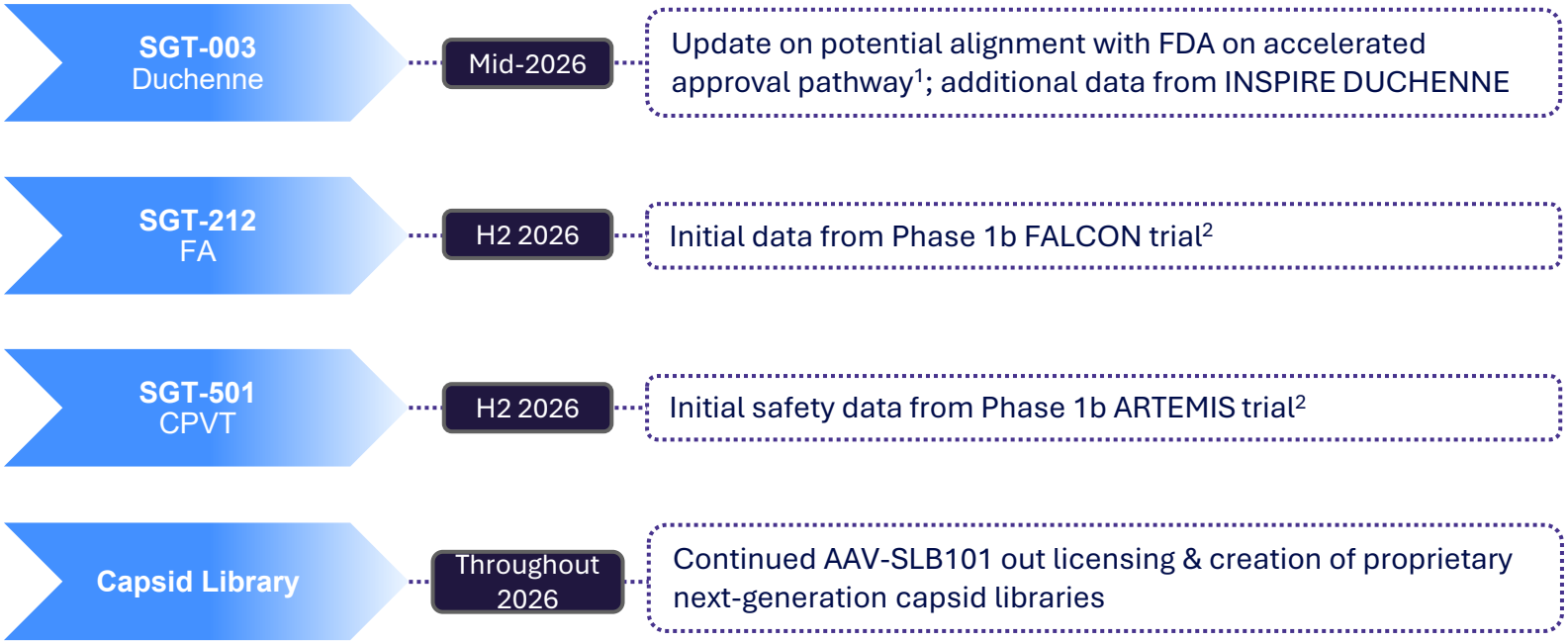
Poised for Transformational Value Inflection in 2026 & Beyond

Major milestones anticipated across all lead programs create multiple opportunities for value inflection



1. Expected timing, subject to scheduling and formal receipt of meeting minutes. 2. Subject to participant enrollment

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Learn about ways to partner with us: www.solidbio.com/partners/