Corporate Presentation

November 2024

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Forward Looking Statement

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 achieve key clinical milestones; the company’s SGT-003 program, including expectations for additional CTA filings, site activations, expanded clinical development, production of additional SGT-003 GMP batches, initiation and enrollment in clinical trials, dosing, and availability of clinical trial data; the company’s expectations for submission of an IND for SGT-501 and to submit additional INDs by the end of 2026; the cash runway of the company and the sufficiency of the Company’s cash, cash equivalents, and available-for-sale securities to fund its operations; 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-501, SGT-401 and other programs and platform technologies on the timelines expected or at all; obtain and maintain necessary and desirable 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; compete successfully with other companies that are seeking to develop Duchenne 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-501, SGT-401 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 presentation 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.

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Industry Leading Platform, Partners, Pipeline, and Management Team

Clinical Stage Genetic Medicines Company Targeting Neuromuscular and Cardiac Diseases

Program	Indication	Research / Discovery	Preclinical	Phase 1/2	Milestone (anticipated)	Worldwide Rights
Neuromuscular						
SGT-003	Duchenne (DMD)				FIH Data Q1 2025 [1]	✓
AVB-202-TT	FA					✓
Cardiac						
SGT-501	RYR2-Mediated CPVT				IND 1H 2025	✓
	CASQ2-Mediated CPVT					✓
SGT-601	TNNT2 DCM					✓
SGT-401	BAG3-Mediated DCM					✓
SGT-701	RBM20 DCM					✓
Platform						
Capsid Library [2]	Cardiac & NM				FIH Data Q1 2025 [3]	✓

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 Pinnacle™ 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. Initial safety, expression and biomarker data for first 3 patients dosed; 2. Cardiac Capsid Library currently in NHPs, Mice and Pigs; 3. AAV-SLB101

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Q3 Corporate Update:
INSPIRE DUCHENNE Clinical Trial Ongoing & Multiple INDs Expected Through 2026

PATIENT DOSING ONGOING IN SGT-003 PHASE 1/2 INSPIRE DUCHENNE TRIAL	First-in-human evaluation of SGT-003 for treatment of Duchenne muscular dystrophy; initial data expected Q1 2025
SGT-003 WELL TOLERATED IN FIRST 3 PATIENTS DOSED *	Numerous activities underway to accelerate SGT-003 clinical development including activation of additional sites in U.S., Canada & Europe and additional GMP manufacturing batches
PHASE 1/2 INSPIRE DUCHENNE PROTOCOL EXPANDED SEPT. 2024	Based on encouraging early results in first 3 patients, INSPIRE DUCHENNE trial protocol was amended to expand anticipated enrollment to 43 participants and broaden age cohorts to 4 to < 7 and 7 to < 12 years of age
TARGETING SUBMISSION OF 3-4 INDs BY END OF 2026	Strategically selecting neuromuscular and cardiac diseases; CPVT IND submission expected 1H 2025
STRONG PROGRESS IN CAPSID LIBRARY OUT-LICENSING	AAV-SLB101, the proprietary capsid used in SGT-003, is now being used by 13 academic labs and 1 corporation, with additional negotiations underway

* Data reported on November 6, 2024

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Neuromuscular Lead Program

Duchenne Muscular Dystrophy (Duchenne)

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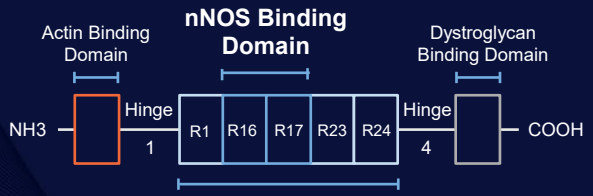
SGT-003 Utilizes an Optimized Transgene, Next Generation Capsid and Improved Manufacturing Process

Next-Generation Construct Has Shown Promising Results in Preclinical Testing

Transgene

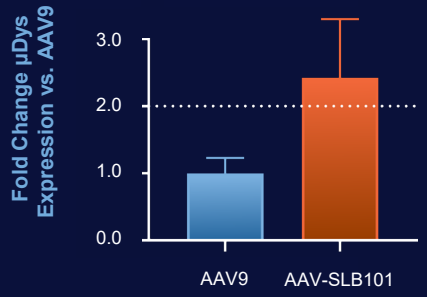
Solid's microdystrophin uniquely includes the nNOS binding domain, potentially important for prevention of activity-induced ischemia and associated muscle injury



Capsid

Rationally designed capsid with the goal of improving skeletal muscle tropism

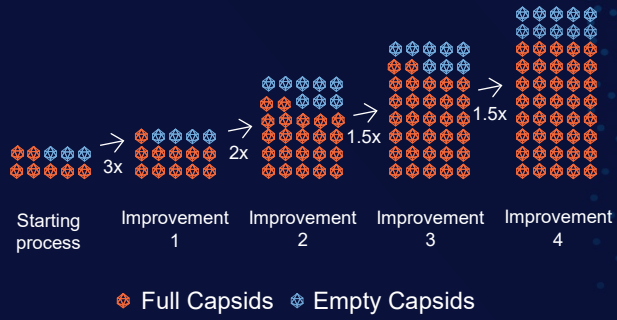
Robust µDys Expression in mdx Mouse



Manufacturing Process

Current yields and full to empty ratios have potential to significantly reduce COGS and enhance safety and efficacy for Duchenne and other gene therapies

Full/Empty and Yield Improvements



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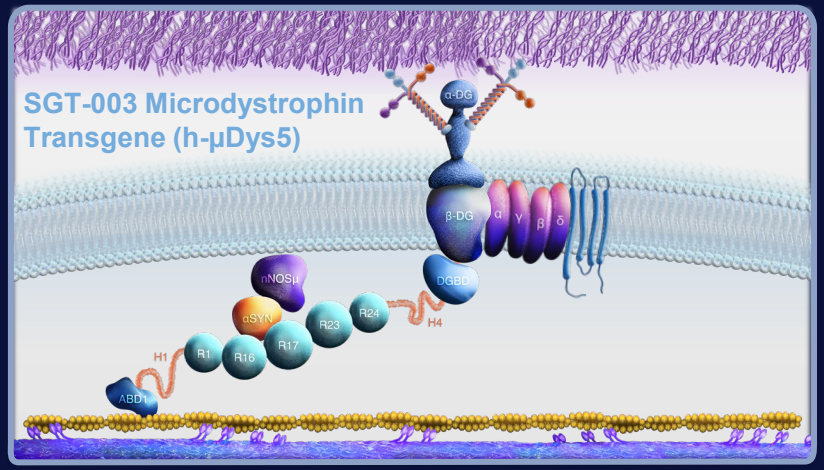
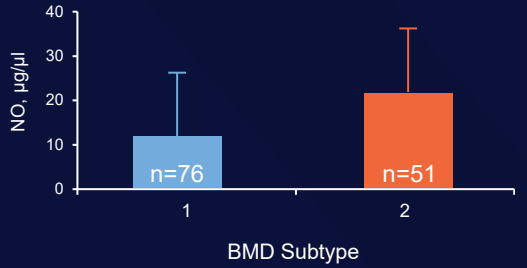
SGT-003's Unique Next-Generation Microdystrophin Strategically Designed to Enhance Functionality and Muscle Endurance

Sarcolemmal nNOS Plays an Important Role in Muscle Function



Nitric Oxide

Presence of nNOS Binding Domain is Associated With Higher Circulating Nitric Oxide in BMD Patients[1]



Loss of sarcolemmal nNOS impairs nitric oxide-mediated vasodilation and can lead to functional ischemia[2,3]

- nNOS may lead to reduction of exercise-induced fatigue associated with Duchenne[2,3]
- Microdystrophin inclusion of R16/R17 facilitates binding of α-Syntrophin, which mediates binding of nNOS in skeletal muscle[4]
- α-Syntrophin is the only dystrophin-associated syntrophin that binds nNOS; constructs without R16/R17 domain cannot recruit nNOS[4]

1. Koeks et al. *Neuromuscul Disord*. 2019. 2. Kobayashi et al. *Nature*. 2008. 3. Boehler et al. *Neuromuscul Disord*. 2019. 4. Adams et al. *Hum Mol Genet*. 2018.

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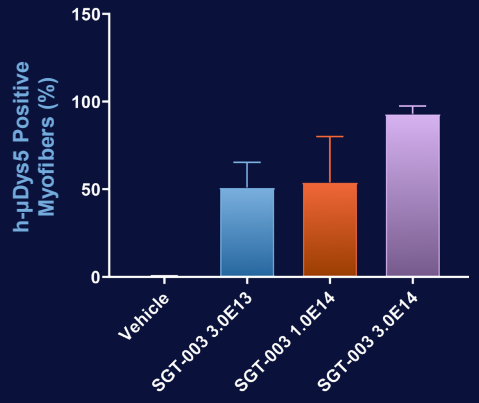
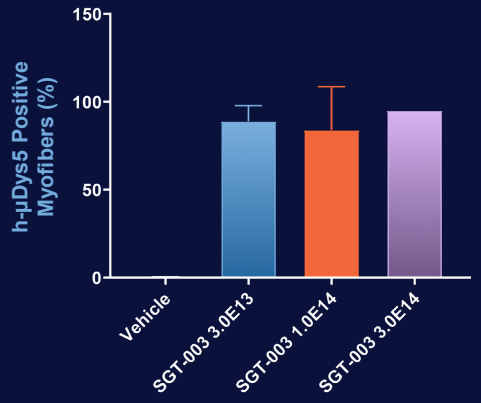
Rapid AAV-SLB101 Transduction and Expression in *mdx* Mouse Model by Day 4

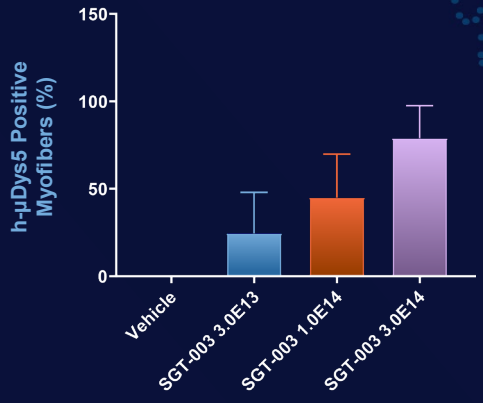
Quadriceps – Day 4



Heart – Day 4



Diaphragm – Day 4



Observations

Robust microdystrophin expression levels, as assessed by h-µDys5+ myofibers in quadriceps, heart, and diaphragm, were evident by Day 4 post-AAV-SLB101 administration

h-µDys5, Solid's microdystrophin construct

N=5 per group

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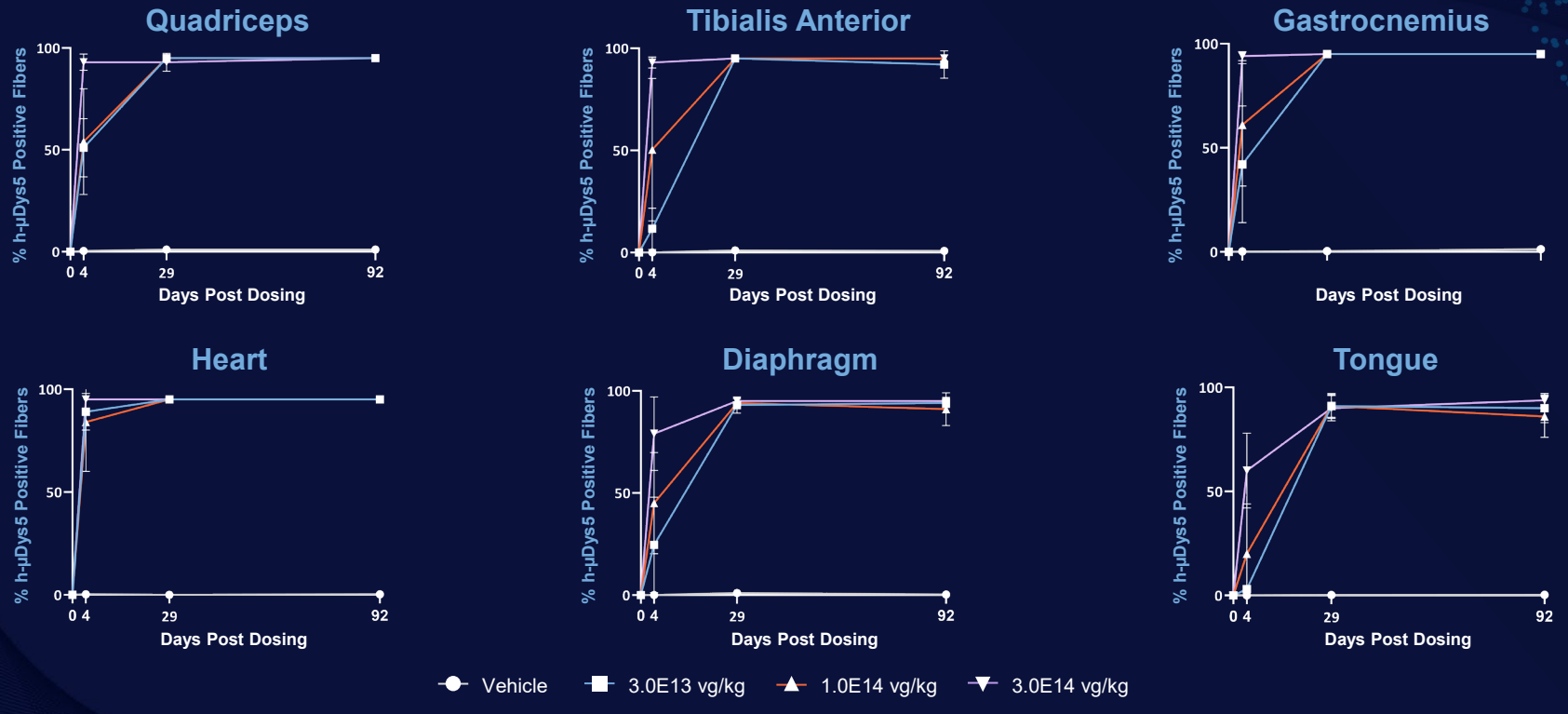
SGT-003 Showed Sustained Microdystrophin Expression in *mdx* Mouse Model

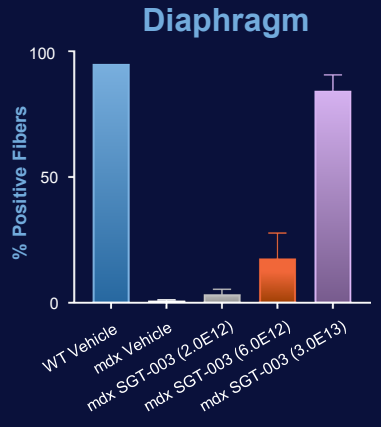


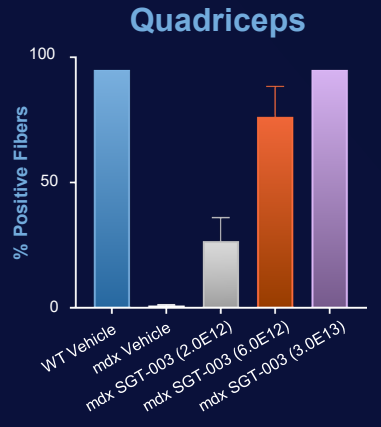
h-µDys5, Solid's microdystrophin construct

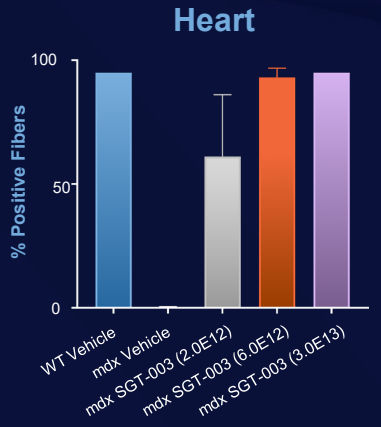
N=5 per group

High Microdystrophin Expression and nNOS Activity in Multiple Tissues at Low Doses in *mdx* Mouse Model

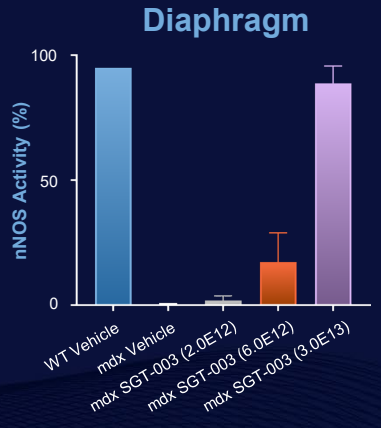
Microdystrophin

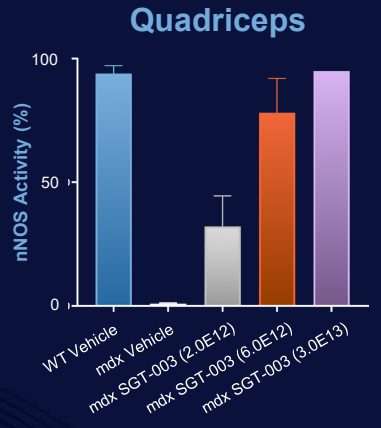






nNOS Activity





N=10 per group

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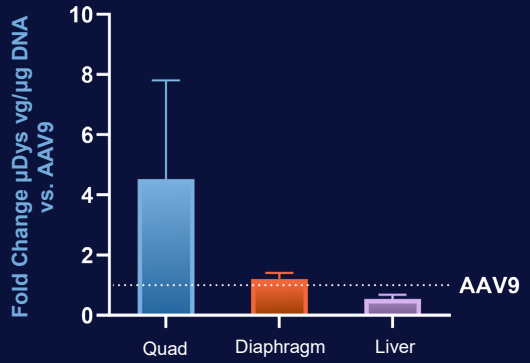
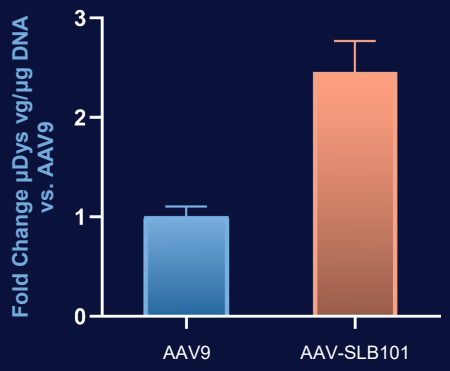
SGT-003 With AAV-SLB101 Capsid Demonstrated Superior Muscle Tropism vs AAV9

Positive Biodistribution and Expression Data Resulted in Improved Biomarker Signals in *mdx* Mouse Model

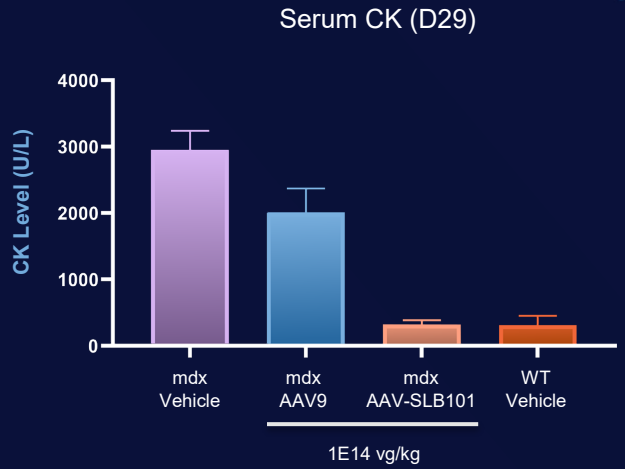
Tissue Specific Biodistribution in *mdx* Mouse



Robust µDys Expression in *mdx* Mouse



Reduced CK Levels in *mdx* Mouse



N=5 per group

Dose 1E14 vg/kg

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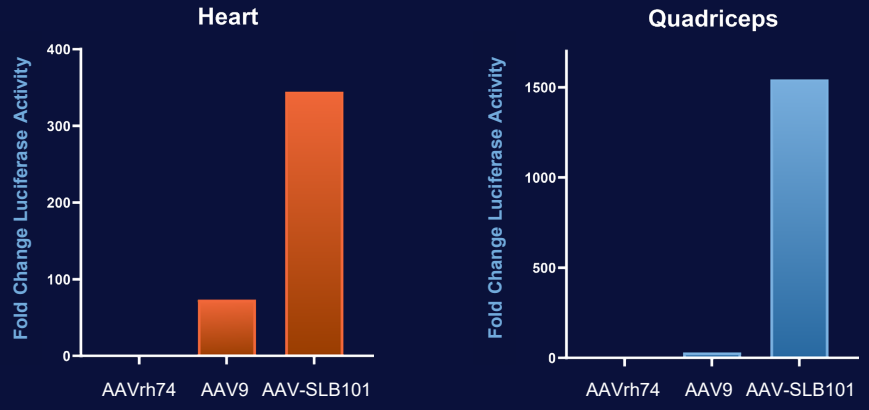
AAV-SLB101 Exhibited Superior Protein Expression Profiles vs AAV-rh74 Across Muscle Tissues of NHPs and Mice

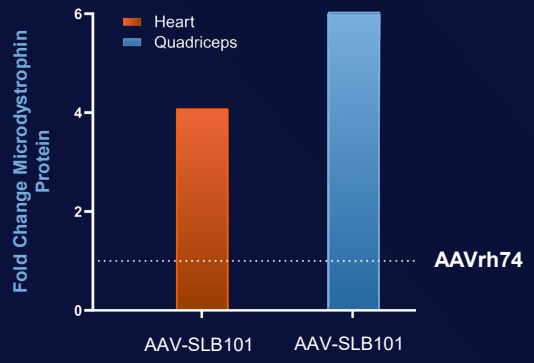
AAV-SLB101 vs AAV9 & AAV-rh74

CK8-Luciferase activity in mouse tissues at a low dose (2.0E12 vg/kg)[a]



AAV-SLB101 vs AAV-rh74

CK8-Microdystrophin protein expression by LC/MS in NHP tissues at clinical dose (1.0E14 vg/kg)[b]



Superior transgene expression profile of AAV-SLB101 in mouse and NHP muscle tissues justified candidate selection and IND-enabling studies with the AAV-SLB101-based therapeutic candidate SGT-003

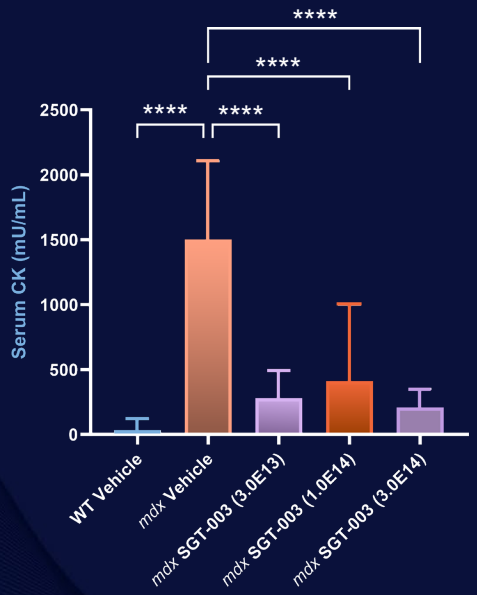
IND=investigational new drug; LCMS=liquid chromatography mass spectrometry

[a]N=5 for each tissue/capsid. [b]N=3 for AAV-SLB101. N=2 for AAV-rh74

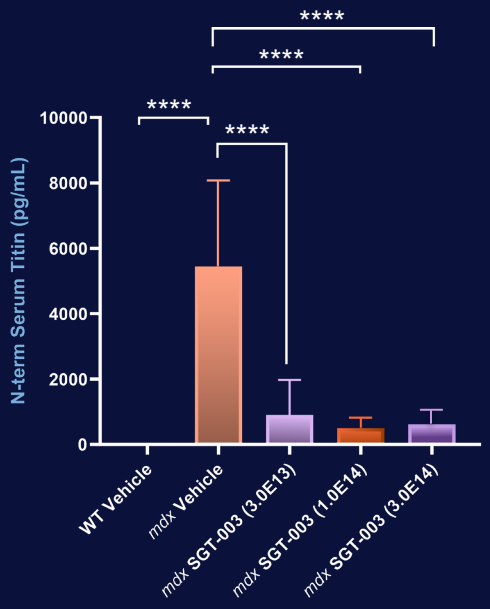
Data on file. Solid Biosciences. 2024.

Improved Serum Biomarkers of Muscle Membrane Integrity Seen in SGT-003-Treated *mdx* Mice at Doses ≥3.0E13 vg/kg

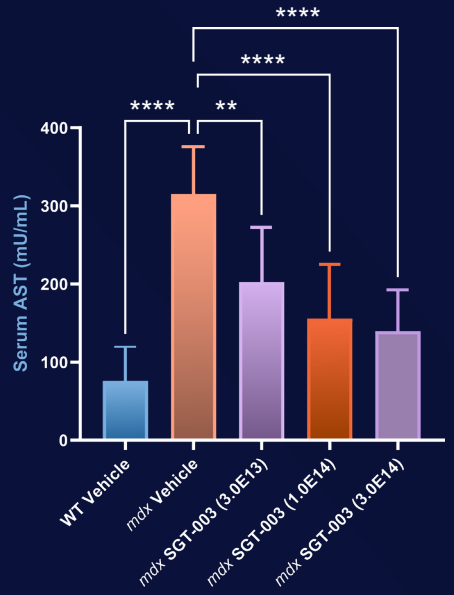
SERUM CK



SERUM TITIN

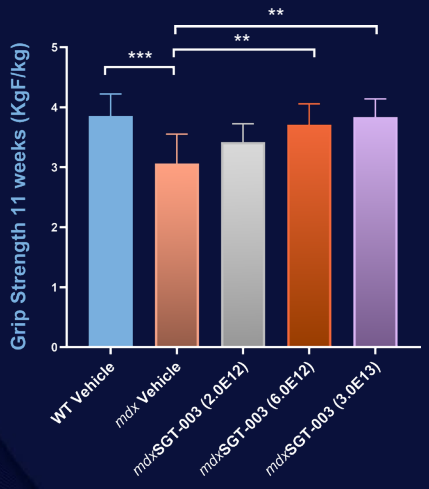


SERUM AST



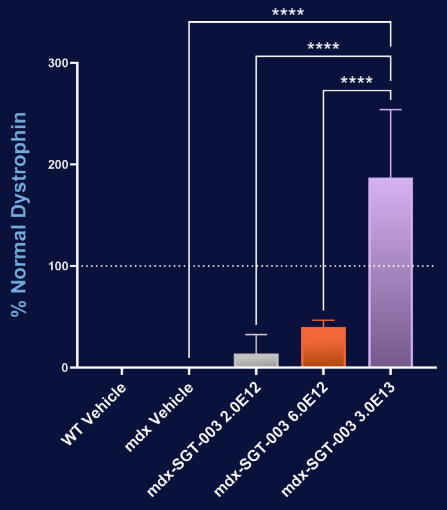
Significant Microdystrophin Expression and Functional Efficacy Observed in mdx Mouse Model at Low Doses (>6E12)

Grip Strength (11 weeks)

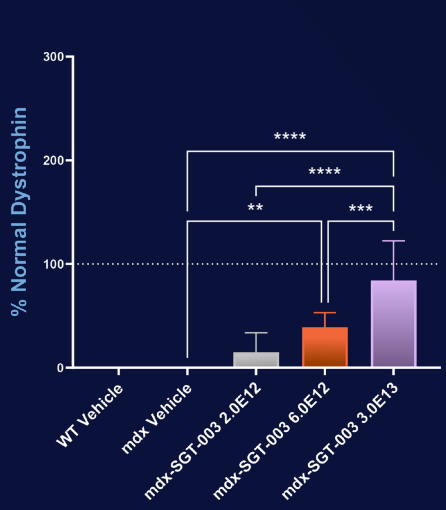


Mass Spectrometry - % Normal Dystrophin

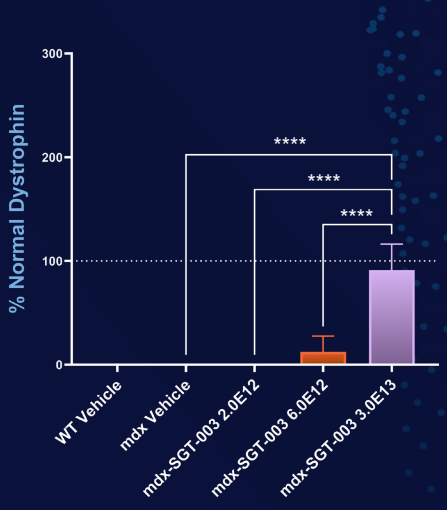
Heart



Quadriceps



Diaphragm



**p<0.005, ***p<0.0005, ****p<0.00005

Dotted line = 100% dystrophin threshold in adult skeletal muscle

N=10 per group

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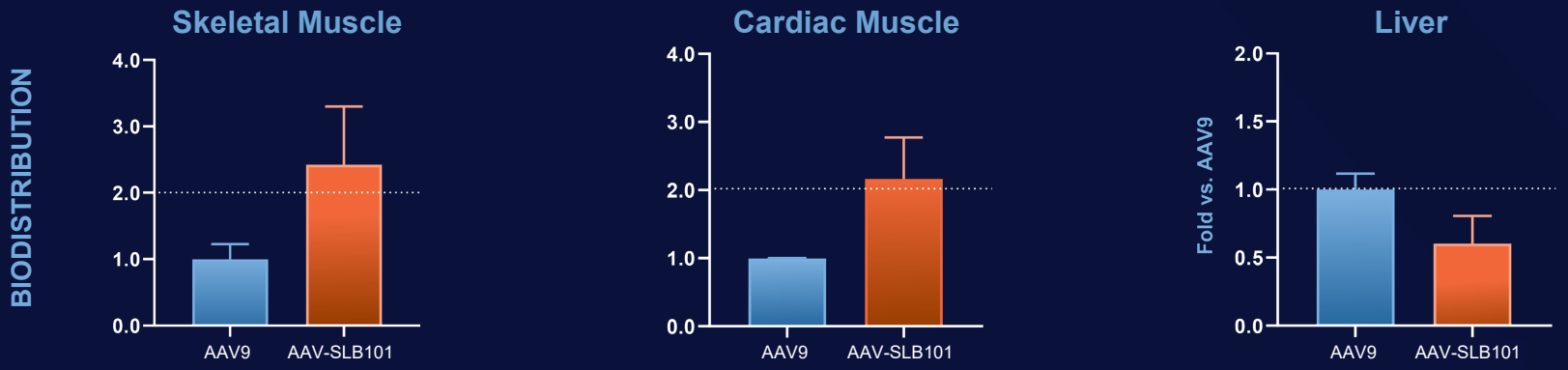
AAV-SLB101 Showed Improved Biodistribution in Cardiac & Skeletal Muscle With Decreased Hepatic Transduction as Compared to AAV9

- Increased biodistribution to skeletal & cardiac muscle correlated with increased transgene expression*
- Reduced biodistribution in liver suggests tissue de-targeting*

NHP IV Administration of AAV-SLB101 With Constitutive Promoter and Reporter Gene



*Average fold differences calculated from five skeletal muscle tissues sampled, three regions of cardiac tissue sampled, and a single liver sample.
Dose 5e12 vg/kg
N=2 per group

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Initial INSPIRE DUCHENNE Safety and Tolerability Data Consistent With GLP Toxicology NHP Study

NHP GLP TOX Study Findings

Well tolerated in both groups throughout study

No early mortality events, no unscheduled take downs

No pathology findings: organ weight changes, macroscopic or microscopic

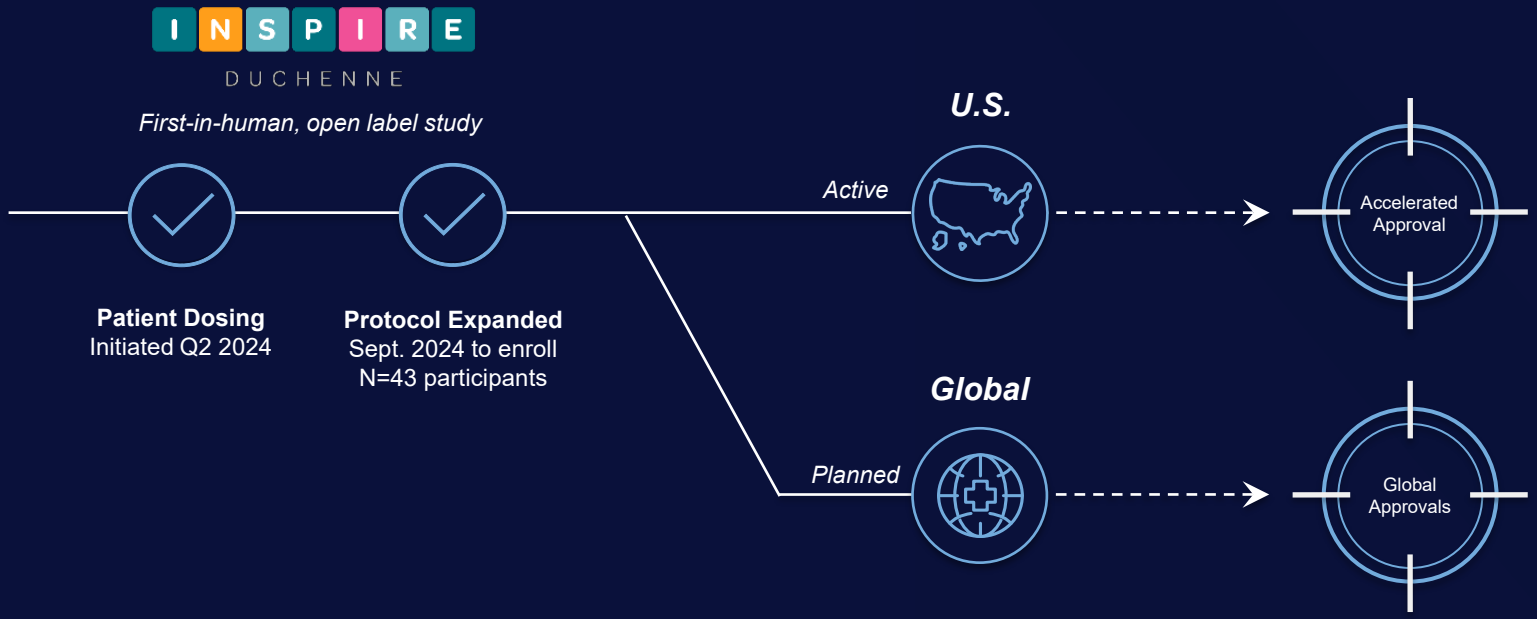
Liver enzyme levels comparable to vehicle at clinical dose level (1E14 vg/kg)

NHPs dosed at 3x clinical dose level

INSPIRE DUCHENNE Initial Safety

- Well tolerated in all patients (n=3) dosed as of Nov. 6, 2024
- No observed serious adverse events
- Immunosuppression achieved with use of steroids alone

INSPIRE DUCHENNE SGT-003 Phase 1/2 Trial Dosing Initiated: Distinct U.S. and Global Regulatory Pathways Anticipated



Solid has designed comprehensive pivotal programs to pursue accelerated approval in the US and global regulatory approvals in parallel

INSPIRE DUCHENNE Clinical Trial Design: Ongoing SGT-003 Phase 1/2 Study

First-in-Human Open-Label, Single-Dose Study

Protocol Updated September 2024 to Enroll an Anticipated 43 Patients

Objective

Primary Objective

- To investigate the **safety and tolerability** of a single intravenous 1E14vg/kg dose of SGT-003

Secondary Objective

- To investigate the **efficacy** of a single intravenous 1E14vg/kg dose of SGT-003

Design

Study includes **2 cohorts** based on age at the time of signing the informed consent:

- Cohort 1: Ambulatory participants aged 4 to < 7
- Cohort 2: Ambulatory participants aged 7 to < 12

All participants must have a genetically confirmed Duchenne diagnosis with a documented dystrophin gene mutation.

Participants must be on a stable dose of at least **0.5 mg/kg/day** of oral daily prednisone or **0.75 mg/kg/day** deflazacort for ≥12 weeks prior to entering the study

Endpoints

Primary Endpoint

- Incidence of treatment-emergent adverse events (AEs) through Day 360

Secondary Endpoints

- Change from baseline of microdystrophin protein levels at Day 90 and Day 360
- Change from baseline in the NSAA score at Day 540
- Change from baseline in stride velocity 95th centile (SV95C) at Day 540

INSPIRE DUCHENNE

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Cardiac Lead Program

Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)

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Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT): a Fatal Disorder in a Young Population

Affected Population	PREVALENCE	ESTIMATED
	1:10,000 people[1]	~33,000 patients in the US

Cause

CASQ2 & RYR2 proteins regulate cardiac calcium (Ca^{2+}), important for electrical conduction and cardiac contraction / relaxation

Postulated Mechanism: Mutations in RYR2 or CASQ2 genes disrupt Ca^{2+} release into the cytoplasm triggering abnormal contraction and relaxation leading to arrhythmias

Solid Approach

AAV-based delivery of a genetic payload to the heart to achieve safe expression of wild-type CASQ2 protein using a cardiac-selective promoter and an optimized transient transfection manufacturing process

Clinical Presentation and Unmet Need

SIGNS & SYMPTOMS

- Most commonly presents as syncope events or cardiac arrest
- Quality of life severely impacted. Risk of spontaneous arrhythmias and or sudden death
- Poor Prognosis: Up to 50% mortality by age 35[2]

AGE OF ONSET

- Typically identified in younger patients (mean onset between 7-9 y/o)[2]

STANDARD OF CARE

- Treatment landscape has not changed in decades: approved treatments – beta blockers and flecainide – do not address the underlying cause of disease, require strict compliance, and have challenging side effects

RYR2=Ryanodine receptor, CASQ2= Calsequestrin 2

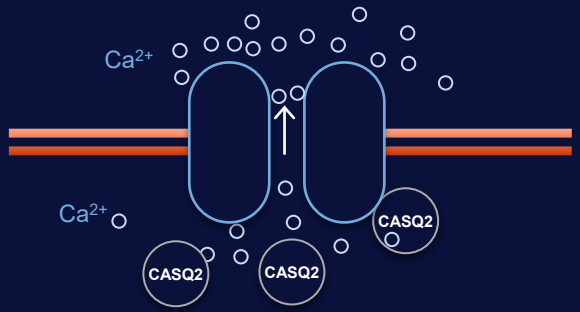
1. Napolitano, et al. 2022 2. Abbas, et al. 2022

Rationale for CASQ2 Augmentation in RYR2 CPVT

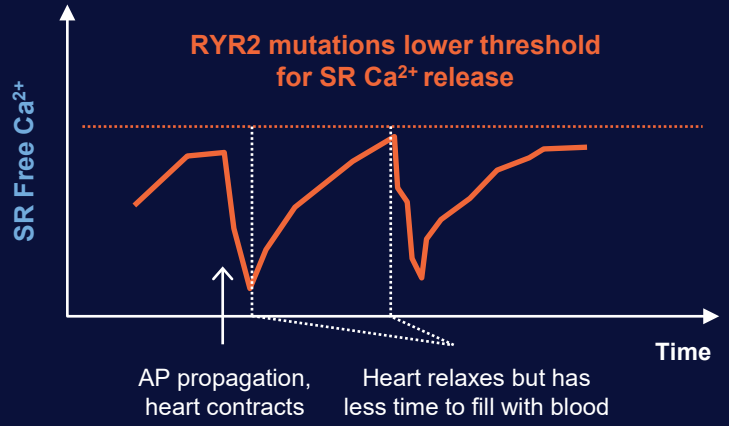
In RYR2 Pathogenic Mutations, Normal CASQ2 Levels are Insufficient to Buffer Ca^{2+} Contributing to Delayed Afterdepolarizations (DAD)

RYR2 Mutation-Related CPVT

Mutations in RYR2 make the channel more sensitive to SR Ca^{2+} levels. This can result in abnormal release of Ca^{2+} in diastole that can lead to delayed afterdepolarizations and resultant ventricular arrhythmia



Arrhythmia



RyR, Ryanodine Receptor; SR, sarcoplasmic reticulum; AP, action potential; Ca^{2+}, calcium

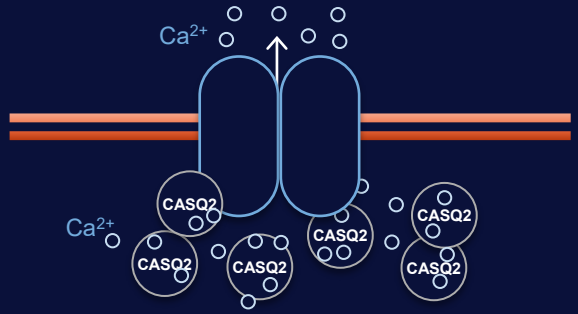
Sources: Proprietary data from Priori Lab

Rationale for CASQ2 Augmentation in RYR2 CPVT (cont.)

Cardiac Delivery of SGT-501 Leads to Increased CASQ2 Which Enhances Ca^{2+} Buffering and Counteracts Ca^{2+} Sensitivity Caused by RYR2 Pathogenic Mutations

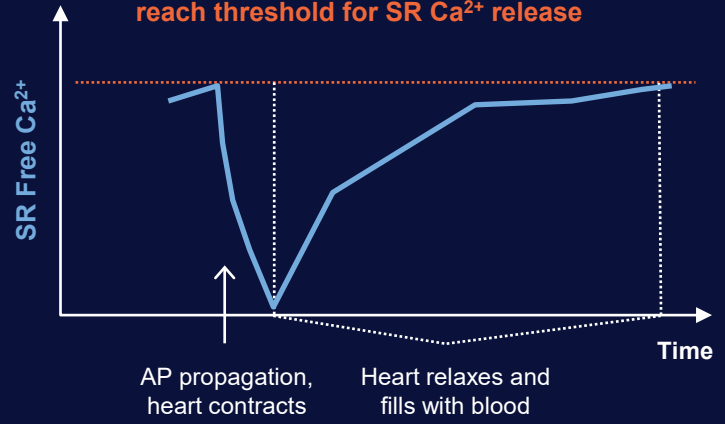
RYR2 Mutation-Related CPVT + Overexpressed CASQ2

Increased CASQ2 enhances Ca^{2+} buffering within the SR and helps stabilize RYR2 in the closed state in diastole, reducing or eliminating the probability of delayed afterdepolarizations (EAD) and resultant ventricular arrhythmia



Normal Rhythm

Overexpressed CASQ2 increases time to reach threshold for SR Ca^{2+} release



RyR, Ryanodine Receptor; SR, sarcoplasmic reticulum; AP, action potential; Ca^{2+}, calcium

Sources: Proprietary data from Priori Lab

RYR2 CPVT Transgenic Mouse Model Used To Support Proof of Concept For AAV Gene Delivery of Human CASQ2

RYR2 Transgenic Mice Have An Arrhythmogenic Phenotype Upon Challenge With β-Adrenergic Agents[1]

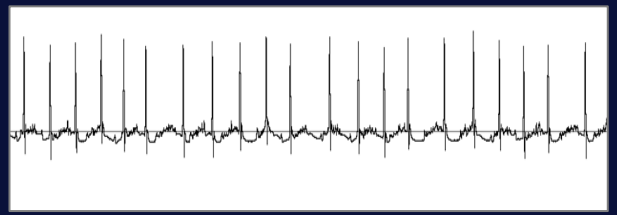
WT Mice

IP dose epinephrine & caffeine

RYR2 Transgenic Mice

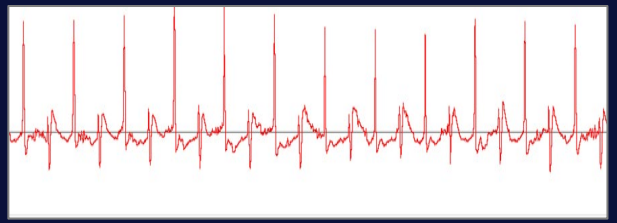
IP dose epinephrine & caffeine

Wild Type



Normal heart rhythm **in WT background strain animals**

RYR2 Transgenic



Polymorphic and/or bidirectional arrhythmic morphology **in transgenic animals**

1. Cerrone, et al. 2005

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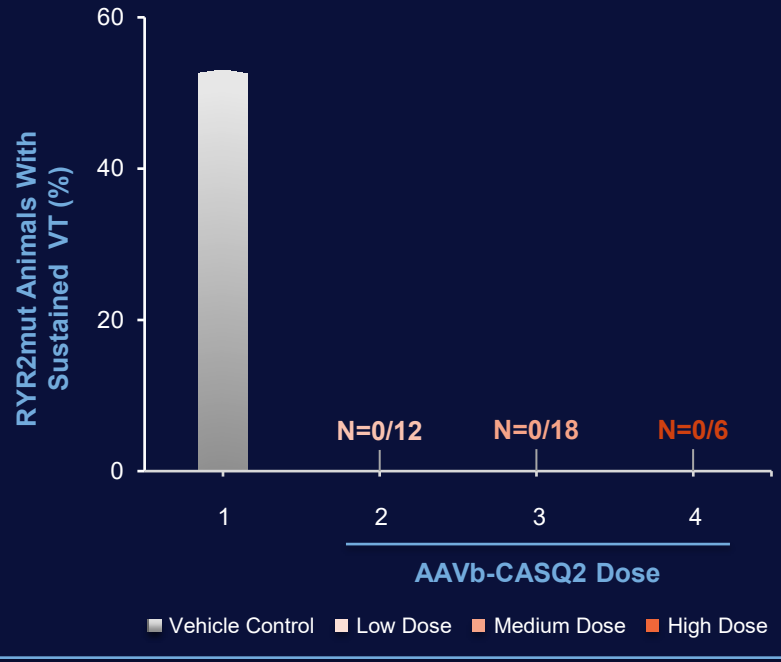
AAV-CASQ2 Treatment Eliminated Arrhythmias in RYR2 Mouse Model

Data Suggests CASQ2 Augmentation Was Well Tolerated & Highly Protective in CPVT-Relevant Transgenic Mouse Models

RYR2-Related CPVT Mouse

At 12 weeks old, **none** of the **RYR2 mutant mice** treated with AAVb-CASQ2 gene therapy exhibited arrhythmias[1]



1. Priori Lab, unpublished data

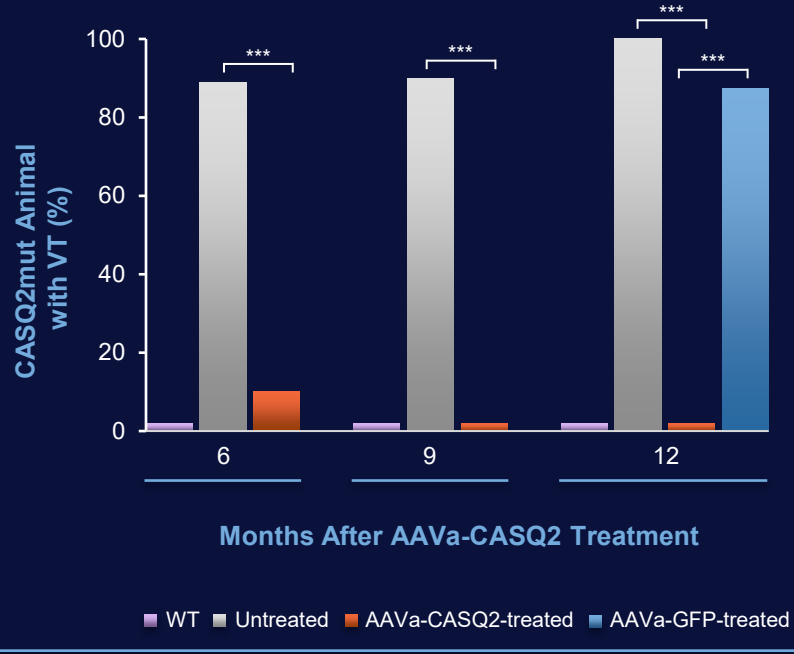
AAV-CASQ2 Treatment Eliminated Arrhythmias in CASQ2 Mouse Model

Data Suggests CASQ2 Augmentation Was Well Tolerated & Highly Protective in CPVT-Relevant Transgenic Mouse Models

CASQ2-Related CPVT Mouse

Significantly fewer **CASQ2 mutant mice** experienced arrhythmias 6-12 months after AAVa-CASQ2 gene therapy[1]

40-50% transduction, achieved in both neonates and adult mice, prevented development of VT upon β-adrenergic challenge[1]



***P<0.001, AAVa-CASQ2–treated vs untreated and AAVa-CASQ2–treated vs AAVa-GFP–treated, VT: Ventricular Tachycardia

1. Denegri, et al. 2014

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Pipeline Programs

BAG3

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BAG3-Related Dilated Cardiomyopathy (DCM): Attractive Indication, Clear Mechanistic Rationale, High Unmet Need & Significant Market Size

Affected Population	PREVALENCE	ESTIMATED	ESTIMATED
	2-4% DCM Cases[1]	~29,000 patients in the US	~33,000 patients in the EU

Cause

BAG3 mutations lead to reduced BAG3 protein leading to dilated cardiomyopathy (DCM)

Postulated mechanism: Decreased BAG3 protein leads to heat shock protein dysfunction and a build-up of dysfunctional proteins in the sarcomere, causing myofilament damage and heart failure

Solid Approach

AAV-SLB101 delivered codon-optimized BAG3 gene with a cardiac-selective promoter utilizing transient transfection manufacturing process

Clinical Presentation and Unmet Need

SIGNS & SYMPTOMS

- Most common presentation is dyspnea (but can be sudden death)
- Activities of daily life are severely impacted
- Adverse long-term prognosis, approximately 25% at one year and ~50% at five years experience severe cardiac event, intervention, or death[1]

AGE OF ONSET

- DCM caused by mutations in BAG3 is characterized by high penetrance in carriers >40 years of age and a high risk of progressive heart failure[1,2]

STANDARD OF CARE

- No approved therapies address underlying cause of disease

1. Dominguez, et al. 2018, 2. Shaw, et al. 2018

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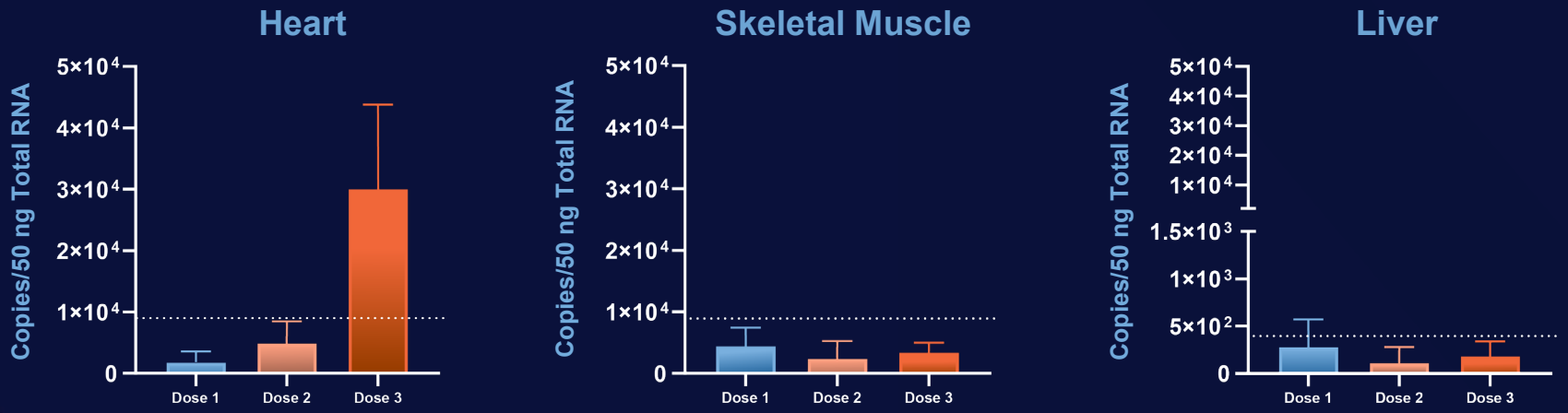
Cardiac-Selective Expression of Human BAG3 in Cardiac-Specific BAG3 Mouse Model

Expression of Human Transgene mRNA Is Below Endogenous BAG3 mRNA Levels in Off-Target Tissues

BAG3 Cardiac-Specific Knockout (cKO) Mouse IV Administration of AAV with Cardiac Promoter and Human BAG3 Transgene



Dotted line = endogenous mouse BAG3 mRNA levels in each tissue; cKO = conditional knockout

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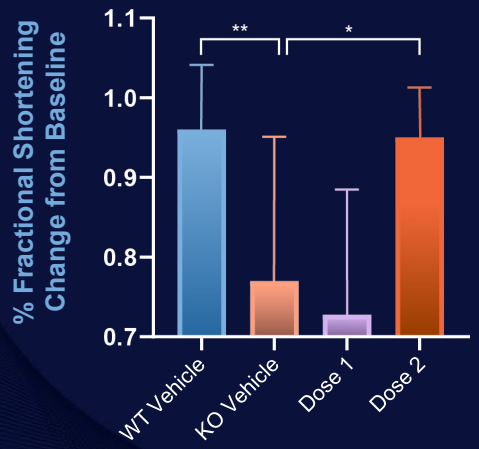
Exploratory Efficacy Study Suggests Improved Cardiac Function in BAG3 cKO Mouse Model

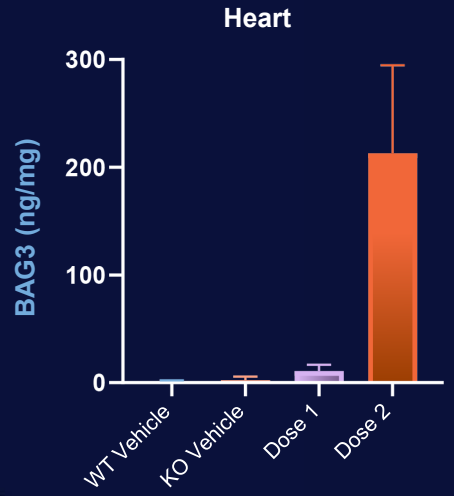
Mouse Data Support Continued Development of AAV-Mediated Gene Delivery of Human BAG3

cKO of BAG3

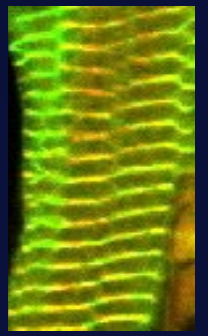
Dose-dependent Improvement in Cardiac Function



Dose-dependent Expression of Human BAG3 Protein



Human BAG3 Protein Localized to Z-line in Cardiomyocytes



Green= BAG3; Red= a-actinin

*p <0.05 One way ANOVA, Tukey's multiple comparisons test

N=5-14

Platform Technologies

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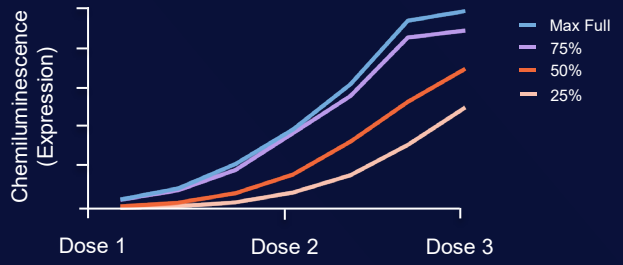
Full/Empty Capsid Ratios Can Impact Transduction and Expression of AAV Products

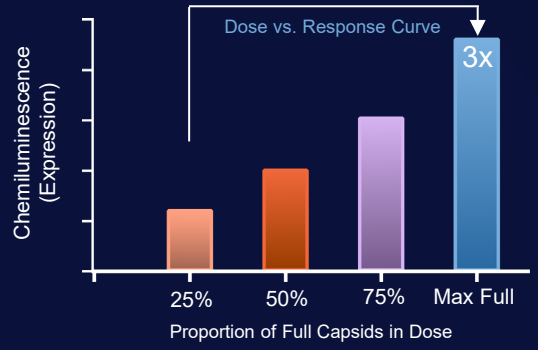
~3-Fold Difference in Chemiluminescence (Expression) Based on Percent Full/Empty

- Transducing C2C12 cells with an AAV luciferase construct allowed chemiluminescence to act as a readout of expression
- Keeping ddPCR titer constant and serially diluting with empty capsids demonstrated that expression was impacted at constant dose
- **Maximizing the percentage of full capsids has the potential to improve both expression and safety of an AAV product**

AAV DS Protein Expression vs Percent Full Capsids (Titer Match Load)[a]



AAV DS Protein Expression vs Percent Full Capsids[a]



ddPCR=Droplet Digital PCR.
[a]AAV-luciferase diluted with empty AAV capsids to yield theoretical 25%-100% full capsids with a series of dilutions based off initial gene of interest titer. N=3 per sample.
Data on file. Solid Biosciences. 2024.

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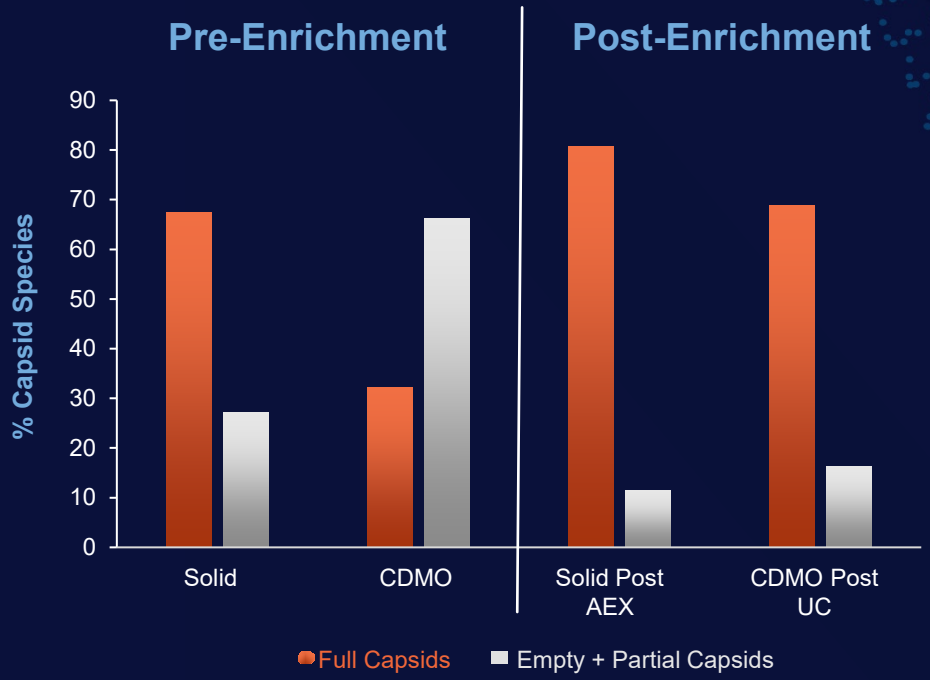
Solid's Manufacturing Platform Has Potential to Challenge Industry Yields and Redefine Full/Empty Capsid Purity

Significant Increase in Yields and Continued Improvements in Full/Empty Ratios Seen at Research Scales*

Yield & Quality Performance

Solid's pre-enrichment capsids (full vs. empty + partial) superior to leading CDMOs



*Data on file, currently at 2L scale in process development (PD), DMD clinical program currently at 1000L

Anticipated Near-Term Milestones

	Program	Milestone (anticipated)	Timing
Neuromuscular	SGT-003 for Duchenne	INSPIRE DUCHENNE Phase 1/2 patient dosing commenced	✓
		Submit multiple CTAs for global trial (already authorized in Canada)	Ongoing
		Initial 3 patient Phase 1/2 data (safety, microdystrophin expression & biomarker data)[1]	Q1 2025
Cardiac	SGT-501 for CPVT	IND-enabling NHP and mouse studies	Ongoing
		Planned submission of RYR2 IND	1H 2025
Capsids	AAV-SLB101	First-in-human data	Q1 2025
	Capsid Library (multiple capsids)	Complete rounds of NHP, mouse, and pig studies	Ongoing
Pipeline	Multiple Pipeline Assets	TNNT2 NHP & mouse studies, BAG3 preclinical studies, RBM20 preclinical work	Ongoing

1. Initial 3 patient safety, expression and biomarker data

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