Corporate Presentation

January 2024

SOLID BIOSCIENCES

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 priorities and achieve key preclinical and clinical milestones; the company’s plans with respect to its Phase 1/2 clinical trial for SGT-003; the company’s plans for filing an IND for SGT-501; the company’s preclinical programs, including expectations for filing INDs, and the company’s future development of preclinical and capsid programs; 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 ability to recognize the anticipated benefits of Solid’s acquisition of AavantiBio; the company’s ability to advance SGT-003, SGT-501, AVB-401, AVB-202-TT and other preclinical programs and capsid libraries 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 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, AVB-401, AVB-202-TT 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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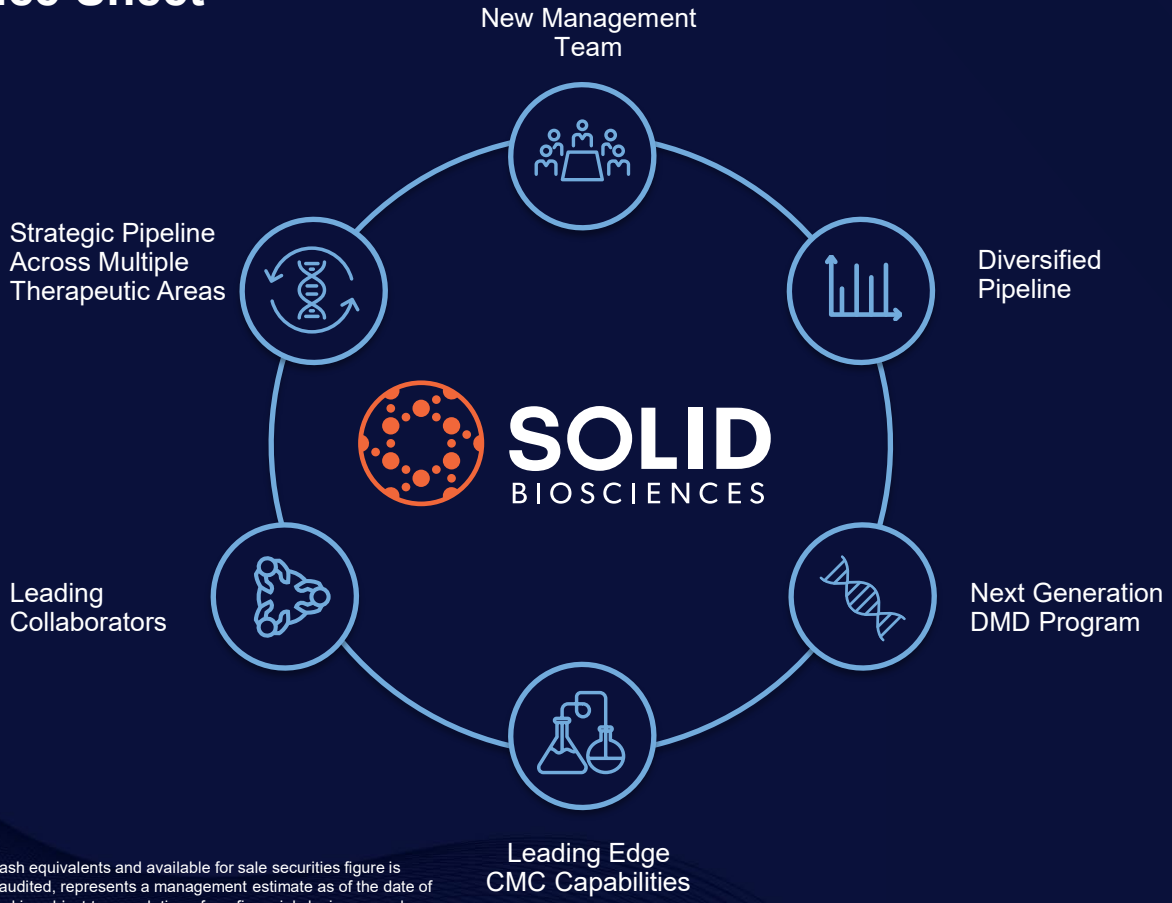
Industry Leading Platform, Partners, Pipeline, Management, and Strong Balance Sheet



$123.9M*
as of 12/31/23
&
$100.0M+
In anticipated net proceeds from PIPE announced 1/8/24

*Estimated cash, cash equivalents and available for sale securities figure is preliminary and unaudited, represents a management estimate as of the date of this presentation and is subject to completion of our financial closing procedures

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Clinical Stage Company With Additional Cardiac IND in Early 2025

Program	Indication	Research / Discovery	Preclinical	Phase 1/2	Milestone (anticipated)	Worldwide Rights
Neuromuscular						
SGT-003	Duchenne				Initial FIH Data Q3 2024	✓
AVB-202 – TT	Friedreich's Ataxia					✓
Cardiac						
SGT-501	RYR2-Mediated CPVT				IND Q1 2025	✓
	CASQ2-Mediated CPVT					✓
AVB-401	BAG3-Mediated DCM					✓
SGT-601	TNNT2 DCM					✓
SGT-701	RBM20					✓
Platform						
Capsid Library*					FIH Data Q3 2024**	✓

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. * Capsid Library currently in NHPs, Mice and Pigs, ** AAV-SLB101

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Lead Programs

(DMD & CPVT)

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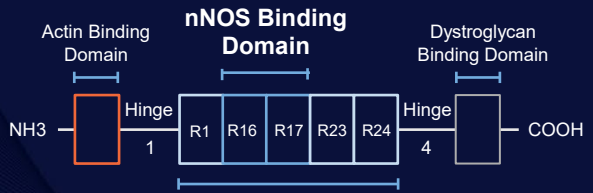
SGT-003 Utilizes an Optimized Transgene, Next Generation Capsid and Improved Manufacturing Process

Next-generation Construct Has Shown Promising Results in Preclinical Testing

Transgene

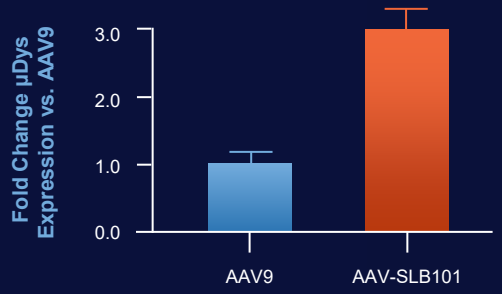
Solid's microdystrophin uniquely includes the nNOS binding domain, potentially important for prevention of activity-induced ischemia and associated muscle injury



Capsid

Rational design approach used to engineer capsid candidates with the goal of improving skeletal muscle tropism

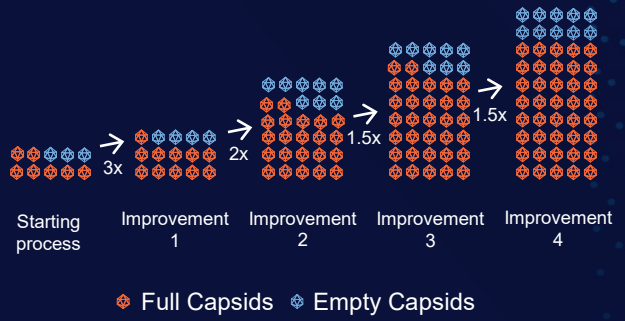
Robust µDys Expression in mdx Mouse



Manufacturing Process

Current yields and empty to full ratios have potential to significantly reduce COGs for DMD and other gene therapies

Empty/Full and Yield Improvements



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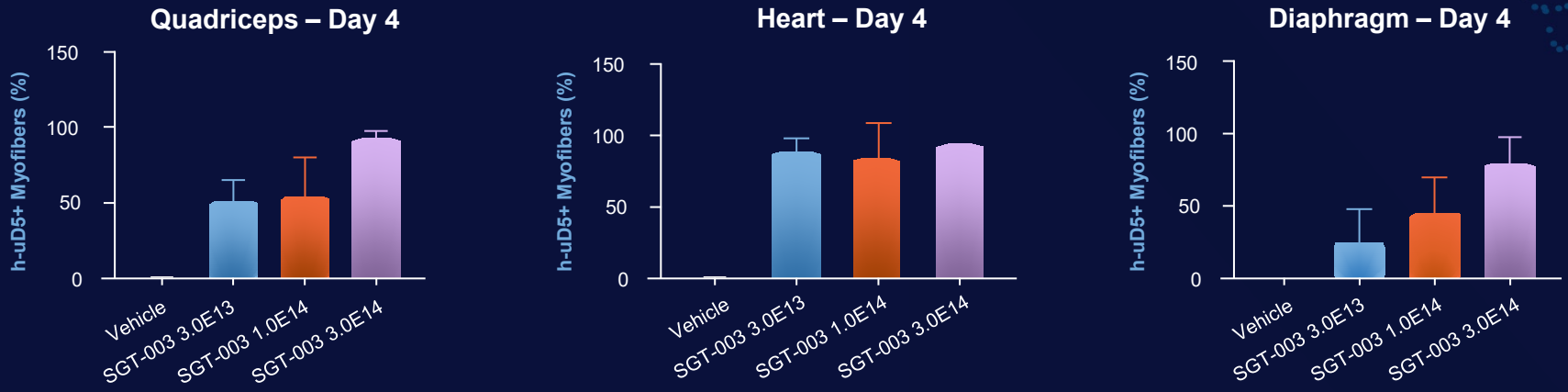
Rapid AAV-SLB101 Transduction and Expression in mdx Mouse Model by Day 4



Observations

Robust microdystrophin expression levels, as assessed by h-uDys5+ myofibers in heart, quadriceps, and diaphragm, were evident by Day 4 post-AAV-SLB101 administration

N=5 per group

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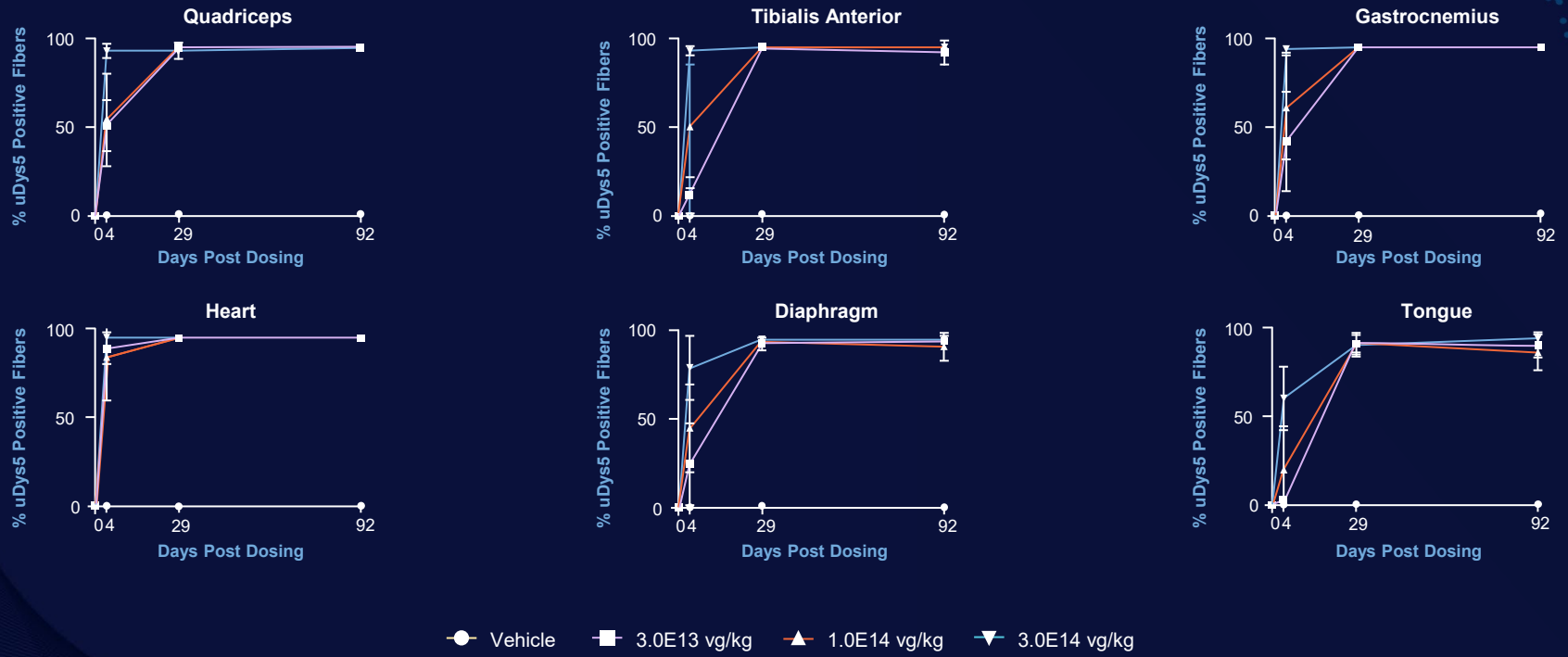
SGT-003 Showed Sustained Microdystrophin Expression in mdx Mouse Muscle



N=5 per group

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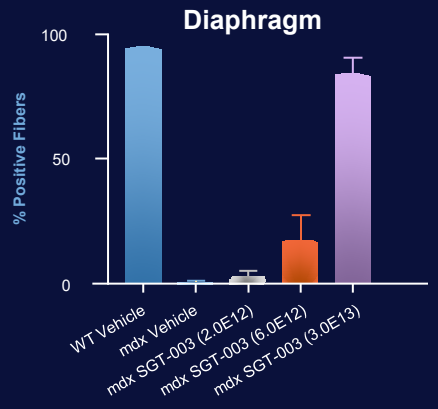
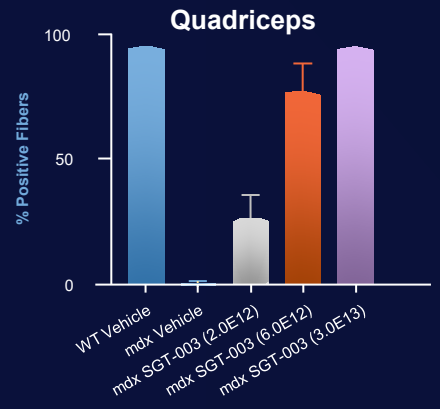
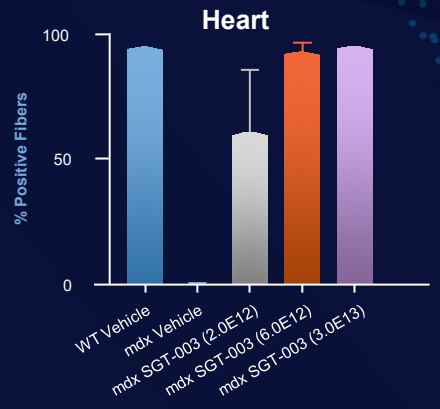
High Microdystrophin Expression and nNOS Activity in Multiple Tissues at Low Doses in mdx Mouse Model

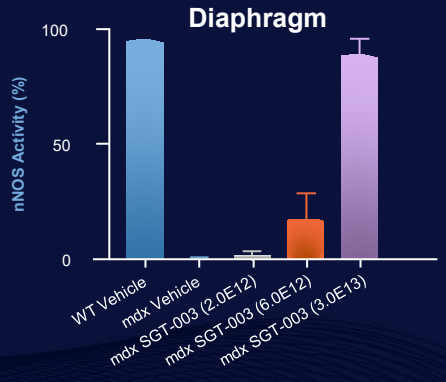
Microdystrophin

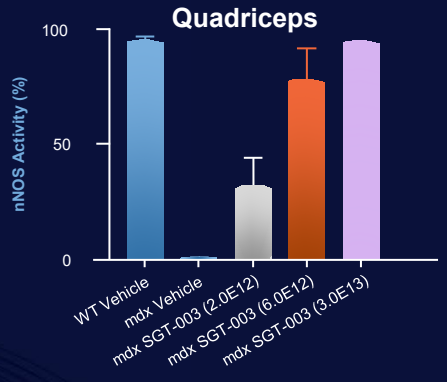






nNOS Activity





N=10 per group

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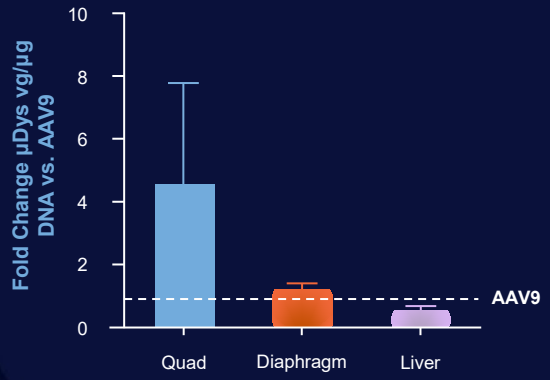
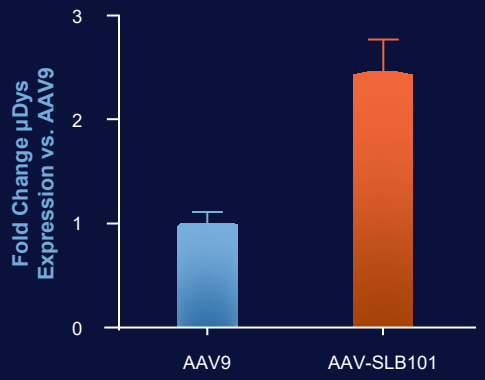
SGT-003 With AAV-SLB101 Capsid Demonstrated Superior Muscle Tropism vs AAV9

Positive Biodistribution and Expression Data Has the Potential to Translate Into Better Efficacy

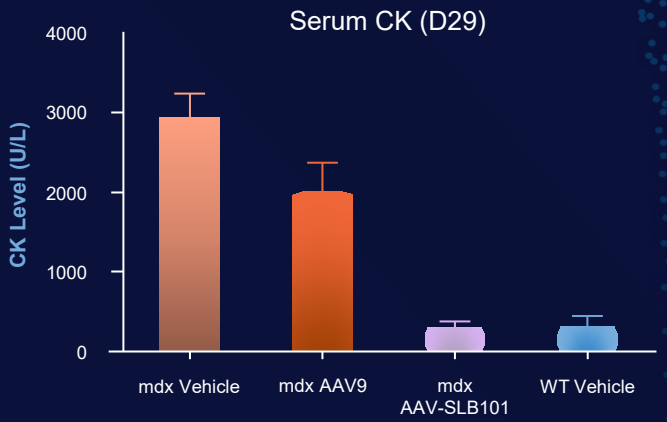
Tissue Specific Biodistribution in mdx Mouse



Robust µDys Expression in mdx Mouse



Reduced CK Levels in mdx Mouse



N=5 per group
Dose 1E14 vg/kg

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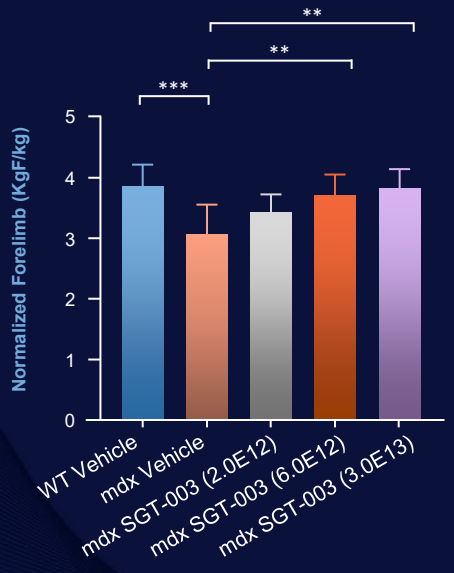
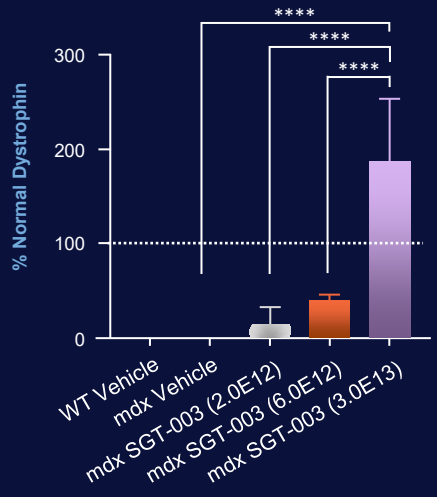
Significant Microdystrophin Expression and Functional Efficacy Observed in mdx Mouse Model at Low Doses (3E13)

Grip Strength (11 weeks)

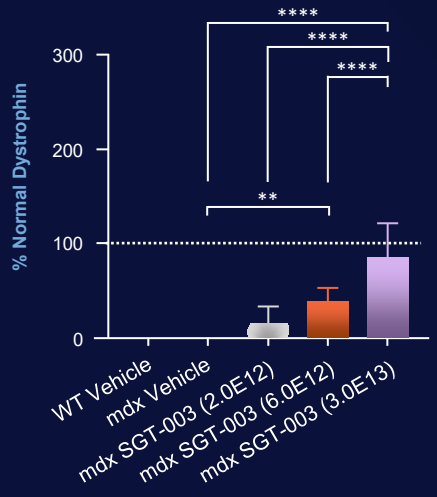


Mass Spectrometry - % Normal Dystrophin

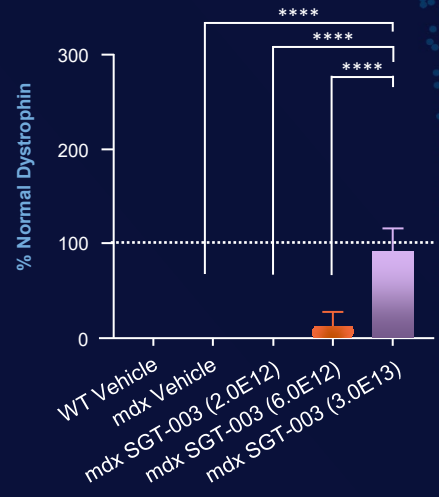
Heart



Quadriceps



Diaphragm



**p<0.005, ***p<0.0005, ****p<0.00005
N=10 per group

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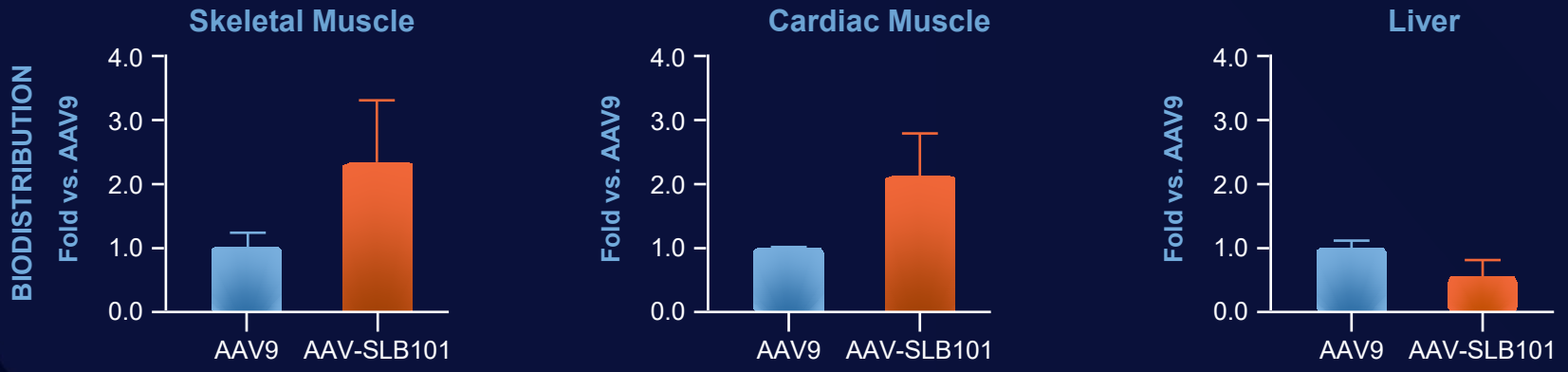
NHPs Administered AAV-SLB101 Showed Improved Biodistribution in Cardiac and Skeletal Muscle With Decreased Hepatic Transduction as Compared to AAV9

- **Increased biodistribution to skeletal & cardiac muscle** resulted in increased transgene expression*
- **Reduced biodistribution in liver** suggests tissue de-targeting and potentially improved safety profile*

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



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

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GLP Toxicology NHP Study Showed SGT-003 Was Well Tolerated

NHP GLP TOX Study

3-month study

2 treatment groups
(1E14 vg/kg & 3E14 vg/kg)

n = 3/group

> 60 tissues evaluated including skeletal muscle, liver, brain

FINDINGS

- Well tolerated in both groups throughout the 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 target clinical dose
- NHPs dosed at 3x planned first-in-human dose (1E14 vg/kg)

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FDA Cleared SGT-003 Phase 1/2 Trial Design

First-in-human Open-label, Single-dose Study to Enroll a Minimum of 3 Patients From Two Sites

Objective	Design	Endpoint

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 dose of SGT-003

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FDA Cleared SGT-003 Phase 1/2 Trial Design

First-in-human Open-label, Single-dose Study to Enroll a Minimum of 3 Patients From Two Sites

Objective | **Design** | Endpoint

Design

Study will include **2 cohorts** based on age, weight, and North Star Ambulatory Assessment (NSAA) at the time of signing the informed consent:

- Cohort 1: Participants 4 to < 6 years of age, <18 kg, NSAA total score 17 to <27
- Cohort 2: Participants 6 to < 8 years of age, <30 kg, NSAA total score 20 to <29

All participants will be required to 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

All participants must be ambulant and have a diagnosis of DMD with a documented dystrophin gene mutation confirmed by genetic testing at screening.

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FDA Cleared SGT-003 Phase 1/2 Trial Design

First-in-human Open-label, Single-dose Study to Enroll a Minimum of 3 Patients From Two Sites

Objective | Design | **Endpoint**

Primary Endpoint

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

Secondary Endpoint

- Change from baseline of microdystrophin protein levels at Day 90 and 360
- Change from baseline in the NSAA score at Day 360
- Change from baseline in 6-minute walk test (6MWT) distance at Day 360

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CPVT

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 arrythmias

Solid Approach

AAV-delivered, CASQ2 transgene with cardiac-specific promoter designed for safe expression utilizing optimized transient transfection manufacturing process

Clinical Presentation and Unmet Need

SIGNS & SYMPTOMS

- Quality of life severely impacted. Risk of spontaneous arrhythmias and or sudden death
- Poor Prognosis: ~40% mortality within 10 years of diagnosis[2]

AGE OF ONSET

- Typically identified in younger patients (mean onset between 7-12 y/o)

STANDARD OF CARE

- No available targeted therapies to address underlying disease cause

RYR2=Ryanodine receptor, CASQ2= Calsequestrin 2
1. Napolitano et al, 2022 2. Sumitomo, 2016

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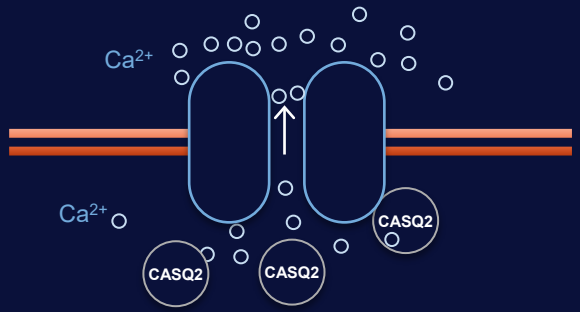
Rationale for CASQ2 Overexpression in RYR2 CPVT

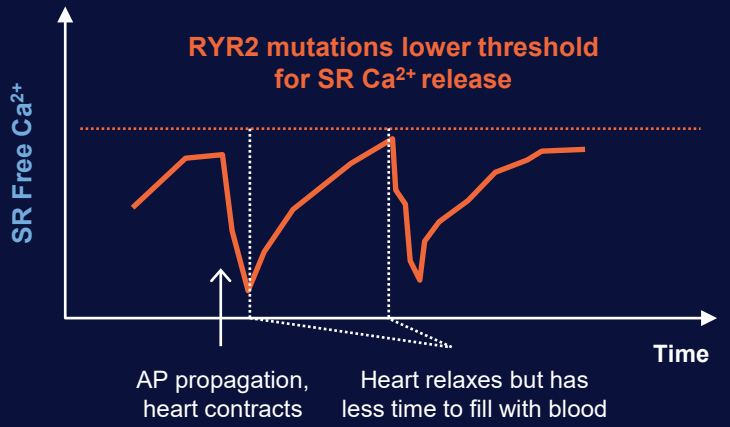
CASQ2 Overexpression Leads to Increased Ca^{2+} Buffering to Counteract Ca^{2+} Sensitivity of RYR2 Mutant

RYR2 Mutation-related CPVT

Mutations in RYR2 make the channel more sensitive to SR Ca^{2+} levels, resulting in early release of Ca^{2+} into the cytoplasm and the heart contracting when it should be filling with blood in diastole



Arrhythmia



RyR, Ryanodine Receptor; SR, sarcoplasmic reticulum; AP, action potential; Ca^{2+}, calcium
Sources: Proprietary data from Priori Lab

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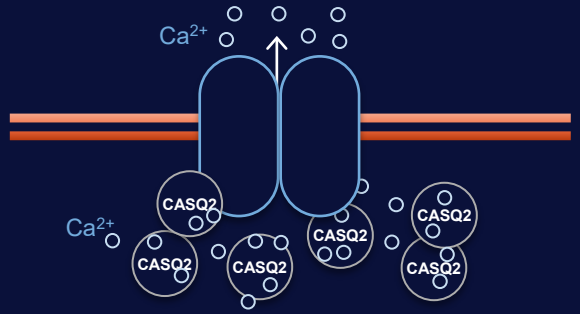
Rationale for CASQ2 Overexpression in RYR2 CPVT (cont.)

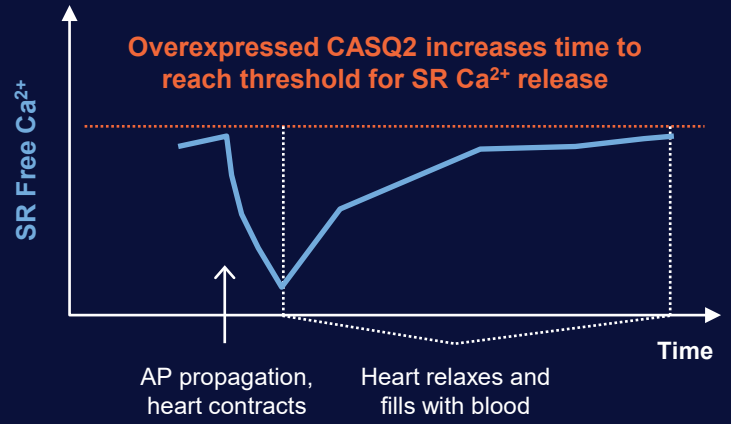
CASQ2 Overexpression Leads to Increased Ca^{2+} Buffering to Counteract Ca^{2+} Sensitivity of RYR2 Mutant

RYR2 Mutation-related CPVT + Overexpressed CASQ2

In addition to stabilizing RYR2 in the closed state, increased CASQ2 enhances Ca^{2+} buffering, so RYR2 senses less Ca^{2+} extending diastole until the typical time of contraction, allowing the heart to fill fully



Normal Rhythm



RyR, Ryanodine Receptor; SR, sarcoplasmic reticulum; AP, action potential; Ca^{2+}, calcium
Sources: Proprietary data from Priori Lab

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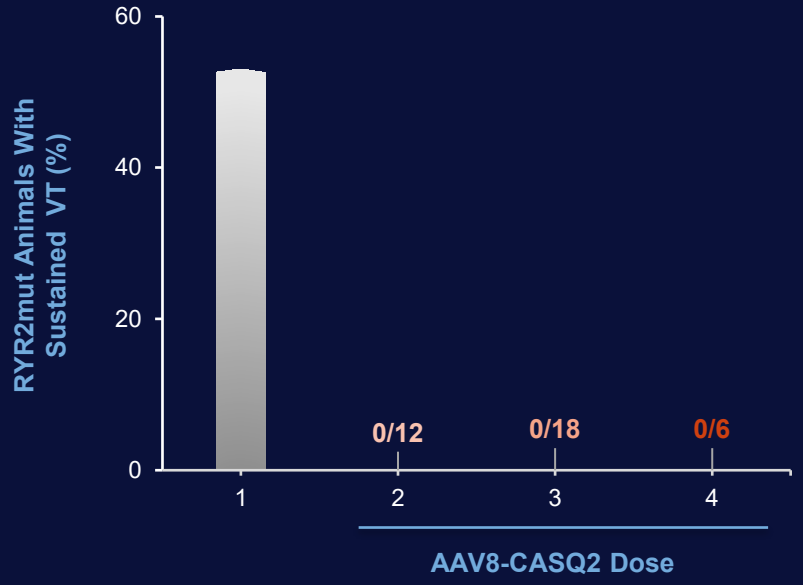
Elimination of Arrythmias in Multiple Disease State Models

Data Suggests CASQ2 Augmentation Was Well Tolerated & Highly Protective CASQ2 & RYR2 CPVT Arrhythmias

RYR2 Mouse

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



***P<0.001, AAVa-CASQ2–treated vs untreated and AAVa-CASQ2–treated vs AAVa-GFP–treated

1. Priori Lab, unpublished data.

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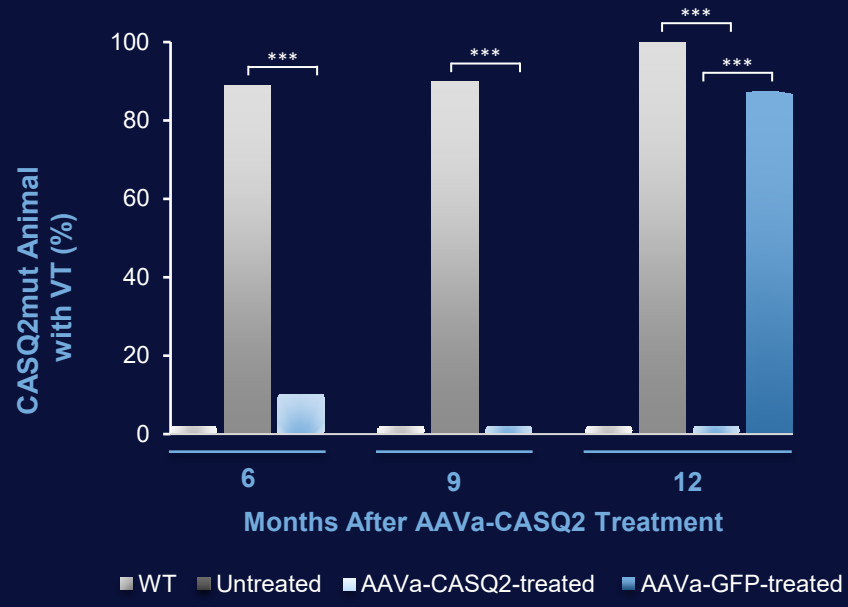
Elimination of Arrythmias in Multiple Disease State Models

Data Suggests CASQ2 Augmentation Was Well Tolerated & Highly Protective CASQ2 & RYR2 CPVT Arrhythmias

CASQ2 Mouse

Significantly fewer **CASQ2 mutant mice** experienced arrythmias two months after AVVa-CASQ2 gene therapy[1]

40-50% transduction, achieved in both neonates and adult mice, prevented propagation of triggered beats[1]



***P<0.001, AAVa-CASQ2–treated vs untreated and AAVa-CASQ2–treated vs AAVa-GFP–treated

1. Denegri, et al. 2014

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CPVT IND Planned for Q1 2025, Potentially Followed by Multiple Ex-US CTAs

Regulatory interactions intended to shed light on regulators' thinking for addressing multiple genetic mutations (RYR2 & CASQ2) with single gene replacement therapy

- Plan global health authority communications in advance of IND/CTA to de-risk IND enabling nonclinical study execution & clinical study design
- UK, NL, IT sites planned in addition to US as part of development
- Orphan Drug Designations obtained in EU and US, seeking rare pediatric disease designation in the US
- Plan to apply for Innovative Licensing & Access Pathway in the UK with aim to accelerate time to market through iterative discussions with MHRA/HTA partners

BAG3

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

AAVrh74-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 cardio 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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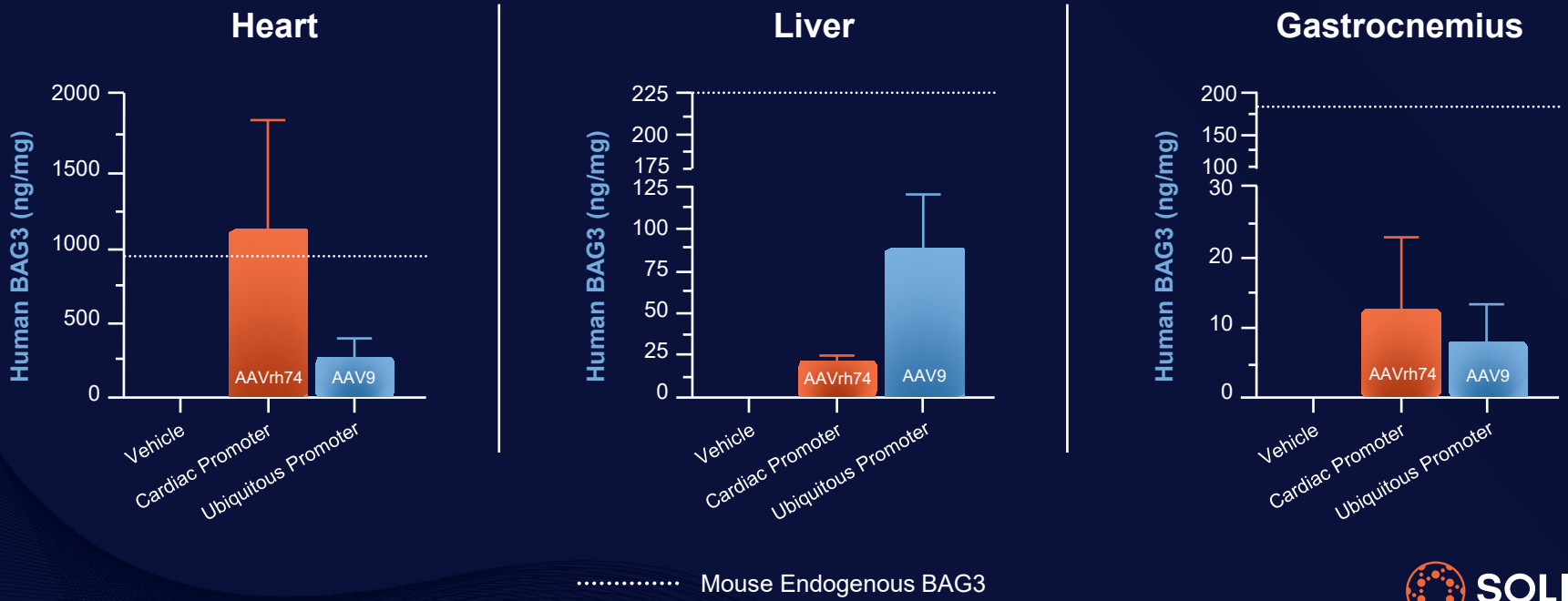
Cardiac-selective Expression of Human BAG3 Transgene Protein in Wild Type Mouse

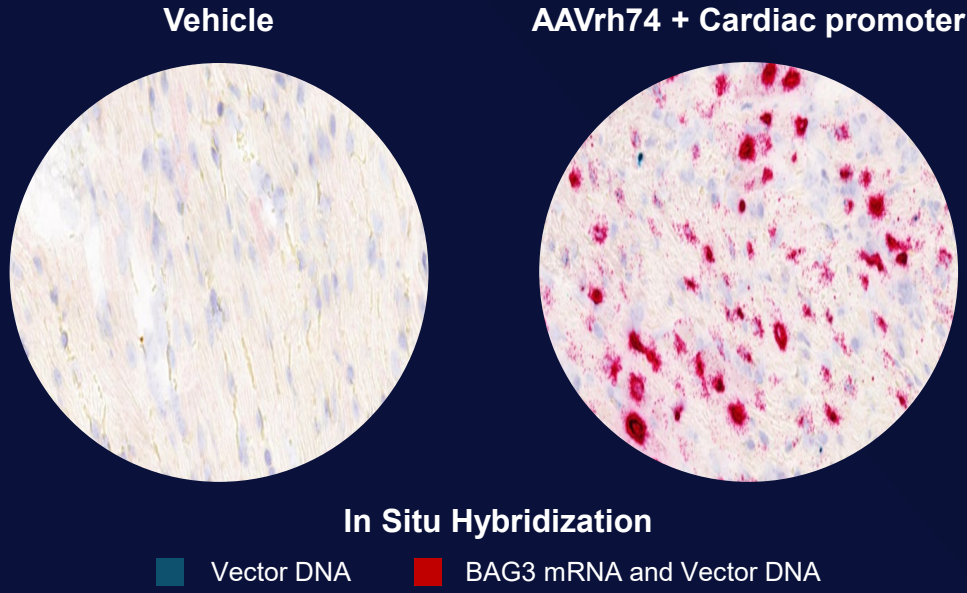
- Transgene expression using a cardiac promoter and AAVrh74 was higher in target tissue (heart) and lower in off-target tissues (e.g. liver)
- Human BAG3 expression in off-target tissues lower than endogenous BAG3



Cardiac-selective Expression of Human BAG3 Transgene Protein in Wild Type Mouse (cont.)

- Transgene expression using a cardiac promoter and AAVrh74 was higher in target tissue (heart) and lower in off-target tissues (e.g. liver)
- Human BAG3 expression in off-target tissues lower than endogenous BAG3

~80% of Cardiomyocytes positive for BAG3 mRNA expression / transduction (Red) in wild type mice

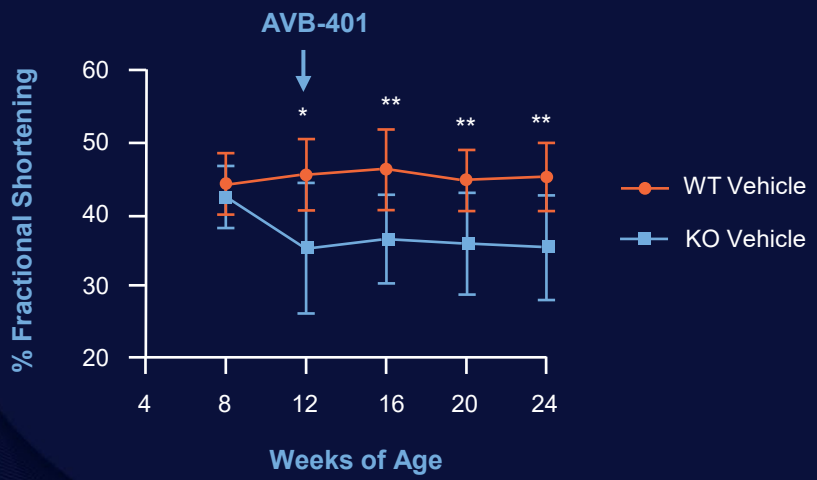


Early Data Showed the Ability of AVB-401 to Rescue Cardiac Function in BAG3 cKO Mouse Model

Mouse Data Supports Continued Development of the Candidate

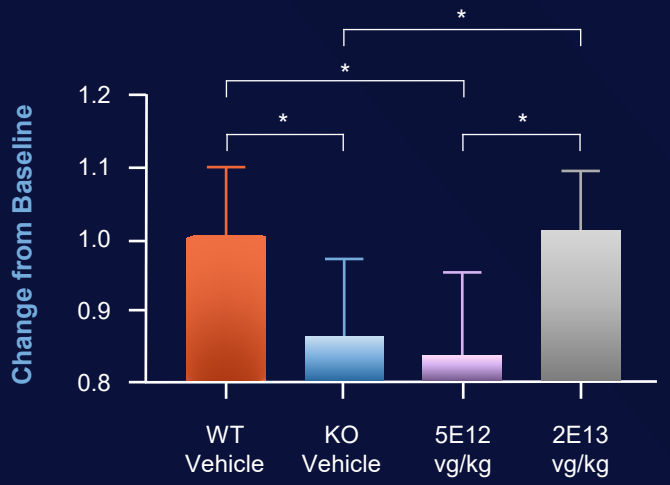
Cardiac-specific Knockout of BAG3 (ckO)

Time-dependent Decrease in Cardiac Function



*p =0.01; ** p<0.008, Mixed effects analysis, Sidaks multiple comparisons test
N=13

2E13 vg/kg Rescues Cardiac Function at 24 WOA



*p <0.05 One way ANOVA, Tukey's multiple comparisons test
N=5-11

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Platform Technologies

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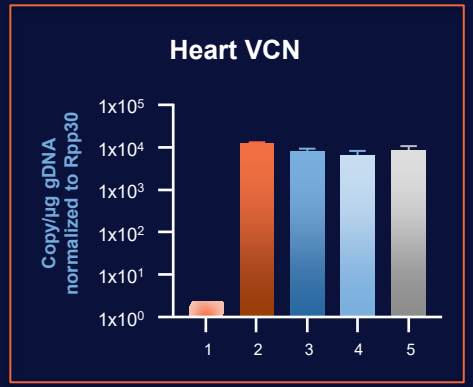
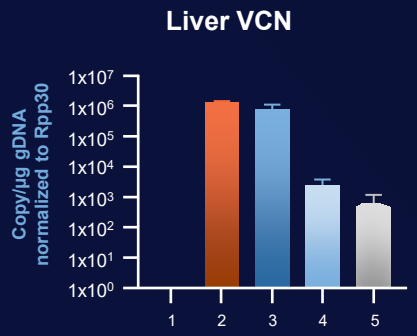
Novel Capsid AAV-SLB134, Modified for Cardiac Tropism and Liver De-targeting

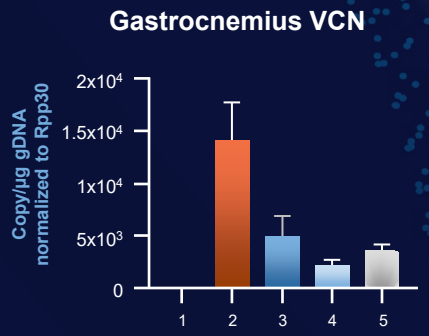
High Level of Heart Transduction with ~1000-fold Decrease In Liver Transduction in Wild Type Mice

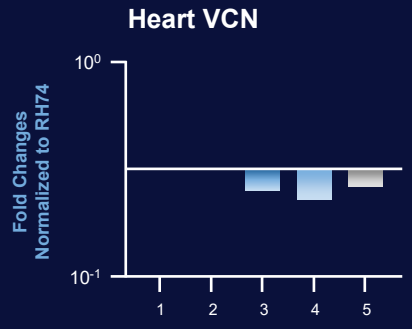
Mice Received 5 Different Treatments

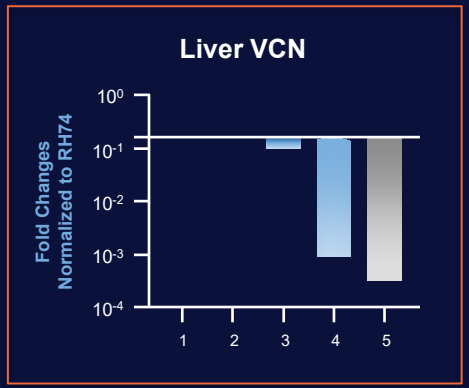
1. No Vector Control
2. Wild-type parental capsid
3. Parental capsid modified for enhanced muscle tropism
4. Parental capsid modified for liver de-targeting
5. Parental capsid modified for liver de-targeting and enhanced muscle tropism

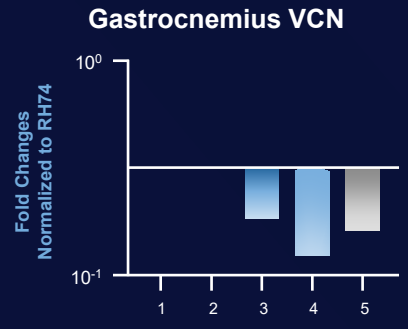












Parental Capsid Data on File, Wild Type Mice

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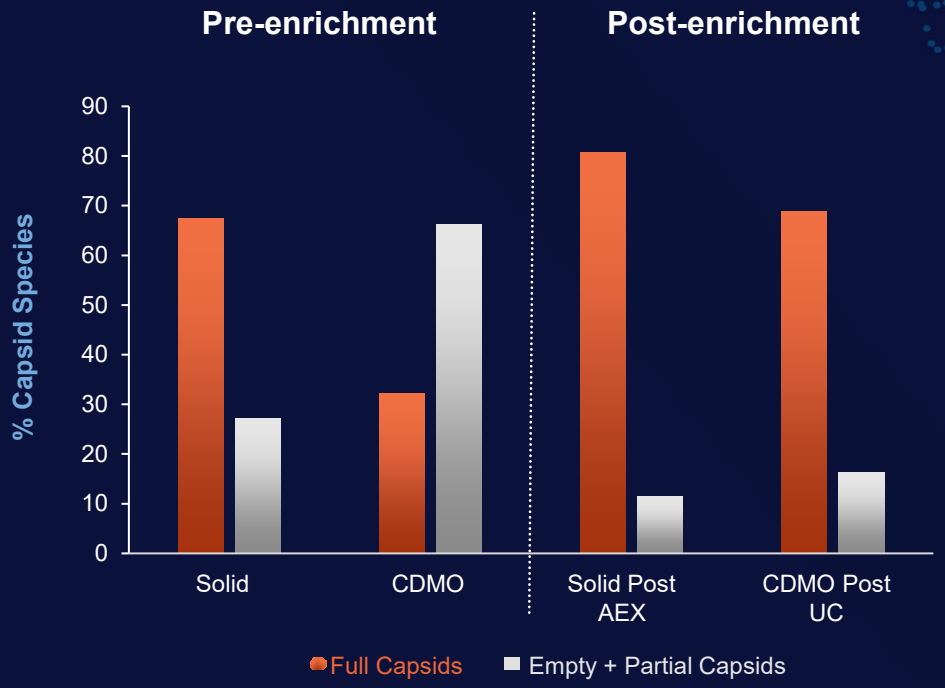
Solid's Manufacturing Platform Has Potential to Challenge Industry Yields and Empty/Full Purity

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

Yield & Quality Performance

Pre-enrichment capsids
(full vs. empty + partial) superior to leading CDMOs



*Currently at 2L scale in PD, DMD currently at 1000L

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Anticipated Near Term Milestones

	Program	Milestone (anticipated)	Timing
Neuromuscular	SGT-003 for Duchenne	First Patient Dosing	Mid-Late Q1 2024
		Phase 1/2 cohort 1 completion of dosing	Mid-2024
		Submit multiple CTAs for global trial	1H 2024
		Phase 1/2 cohort 1 data (microdystrophin expression & functional data)	Q3 2024
Cardiac	SGT-501 for CPVT	Preclinical studies ongoing in NHP and mouse (RYR2 IND planned for Q1 2025)	1H 2024
	AVB-401 for BAG3	Biodistribution & preclinical studies in mouse and NHP	2024
Capsids	AAV-SLB134	Mouse and NHP data	1H 2024
	AAV-SLB101	First-in-human data	Q3 2024
	Capsid Library (multiple capsids)	Complete rounds of NHP, mouse, and pig studies	Ongoing
Pipeline	Multiple Pipeline Assets	TNNT2 mouse studies, RBM20 preclinical work, FA mouse studies	Ongoing

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