SOLID BIOSCIENCES

Q2 2022 Business Update and Financial Results

August 2022

Financial Information and Forward-Looking Statements

This presentation release contains “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995, including statements regarding the Company’s plans to present data from IGNITE DMD, the implication of interim clinical data, the safety or potential treatment benefits of SGT-001 or SGT-003 in patients with Duchenne, the Company’s regulatory plans and discussions, the Company’s plan to continue dosing with SGT-001, the Company’s SGT-003 program, including the Company’s expectation for filing an IND, timelines, 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 its SGT-001 and SGT-003 programs on the timelines expected or at all; obtain and maintain necessary approvals from the FDA and other regulatory authorities; obtain and maintain the necessary approvals from investigational review boards at clinical trial sites and independent data safety monitoring board; replicate in clinical trials positive results found in preclinical studies and early-stage clinical trials of its product candidates; whether the interim data referenced in this release will be predicative of the final results of the trial or will demonstrate a safe or effective treatment benefit of SGT-001 or SGT-003; whether the methodologies, assumptions and applications the Company utilizes to assess particular safety or efficacy parameters will yield meaningful statistical results; advance the development of its product candidates under the timelines it anticipates in current and future clinical trials; successfully transition, optimize and scale its manufacturing process; obtain, maintain or protect intellectual property rights related to its product candidates; compete successfully with other companies that are seeking to develop Duchenne treatments and gene therapies; manage expenses; and raise the substantial additional capital needed, on the timeline necessary, to continue development of SGT-001, SGT-003 and other product 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.

Solid Biosciences At-a-Glance

Advance SGT-001 with new manufacturing process

- IGNITE DMD functional and durability data supports program advancement
- Program is transitioning to a commercially scaled transient transfection-based manufacturing process with clinical entry in 2023
- Additional IGNITE DMD data expected to be released in early-2023, including one-year primary analysis and three-year durability data

Accelerate SGT-003 pipeline program to the clinic

- Mid-2023 IND submission anticipated
- Novel capsid development continues following release of new preclinical data in NHP exploratory study
  - ≥2X increases in muscle targeting, decreased liver uptake
  - ≥10X increase in reporter gene expression in muscle and heart

Solid is positioned for success

- Strategic program and manufacturing process alignment supports funding of operations through important clinical milestones and into Q2 2024
- $162.9m in cash and investments as of June 30, 2022

SOLID BIOSCIENCES AND DUCHENNE MUSCULAR DYSTROPHY

Bringing Meaningful Therapies to Patients Through the Development of Genetic Medicines

2013-2015
ESTABLISHING PATIENT CENTRICITY

Since its founding, the patient experience has been uniquely central to Solid. Clinical studies are powered by input from the patient community and designed to gain meaningful insights in order to best understand all facets of the disease

2016-2017
COLLABORATIONS DRIVE SOLUTIONS

Establishment of Solid's gene therapy focus and SGT-001 as its lead candidate was influenced by the convergence of ideas and knowledge through collaborations with industry, academia, government and community

2018-2019
ENTERING THE CLINIC

Patients 1-6 dosed in IGNITE DMD Phase I/II clinical trial of SGT-001; First 250L GMP AAV production process using A1; First GMP HSV runs at scale; Qualified all first gen release and characterization assays

2020-2021
ESTABLISHING A CENTER OF EXCELLENCE

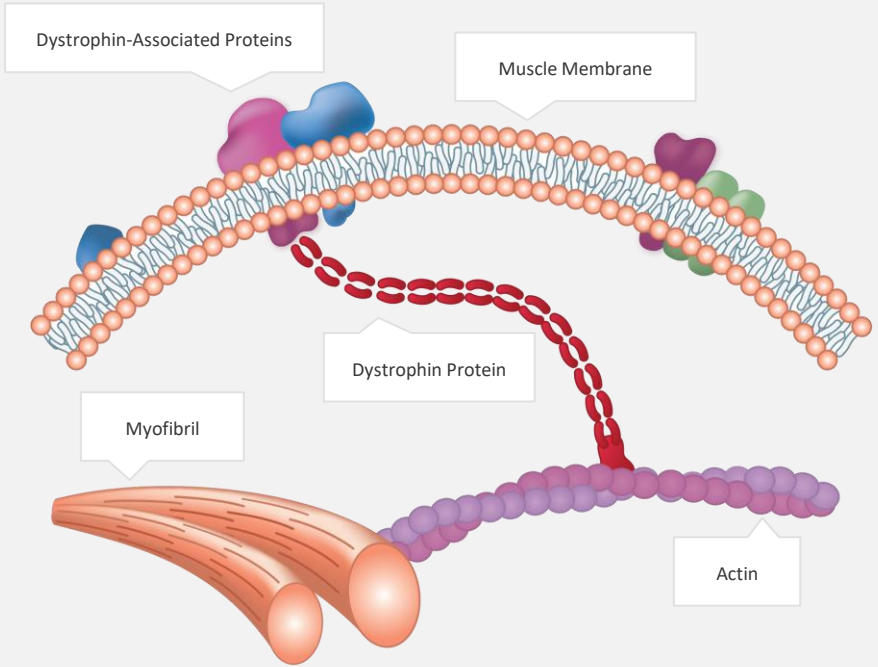
IGNITE DMD dosing resumed, and first clinical data released. Company explores collaborations to utilize expertise in muscle biology, gene therapy, clinical, regulatory and patient advocacy more broadly

2022
BUILDING TOWARD THE FUTURE

Company enters next phase positioned for success with strong financial resources and internal infrastructure to support expansion

Solid's Gene Therapy Programs Are Designed To Address The Genetic Cause Of Duchenne

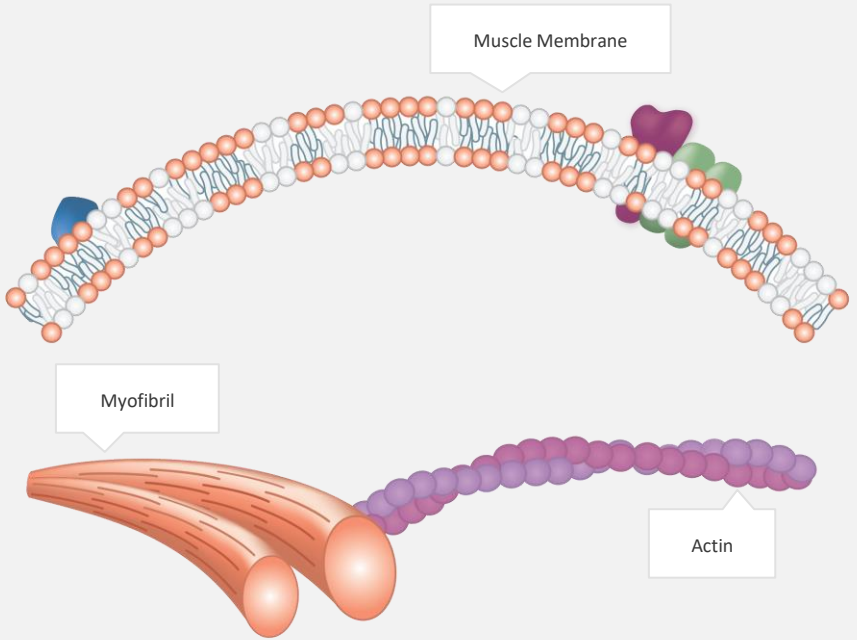
HEALTHY MUSCLE DYSTROPHIC MUSCLE TREATED MUSCLE



Visual representation only

Solid's Gene Therapy Programs Are Designed To Address The Genetic Cause Of Duchenne

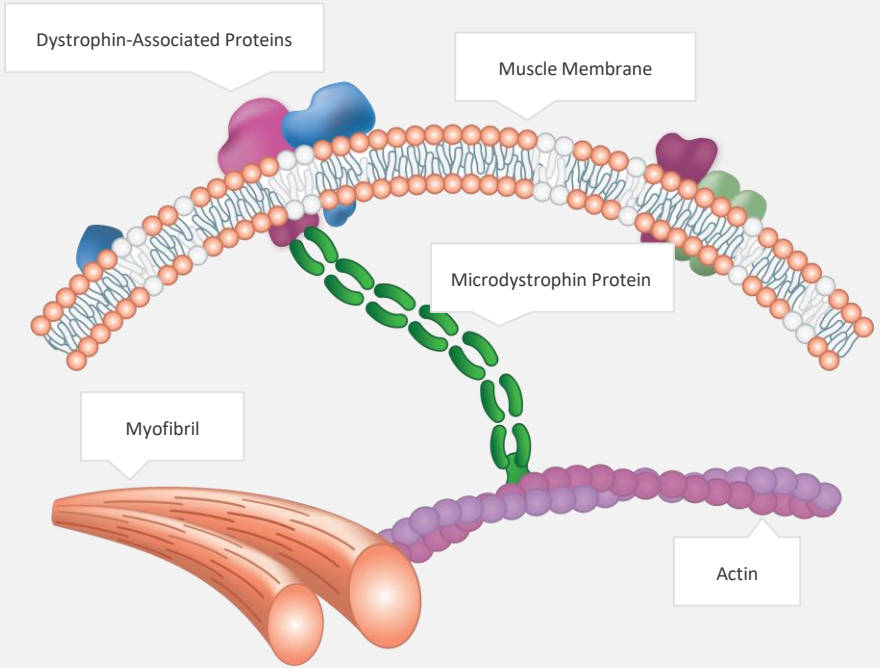
HEALTHY MUSCLE **DYSTROPHIC MUSCLE** TREATED MUSCLE



Visual representation only

Solid's Gene Therapy Programs Are Designed To Address The Genetic Cause Of Duchenne

HEALTHY MUSCLE DYSTROPHIC MUSCLE **TREATED MUSCLE**

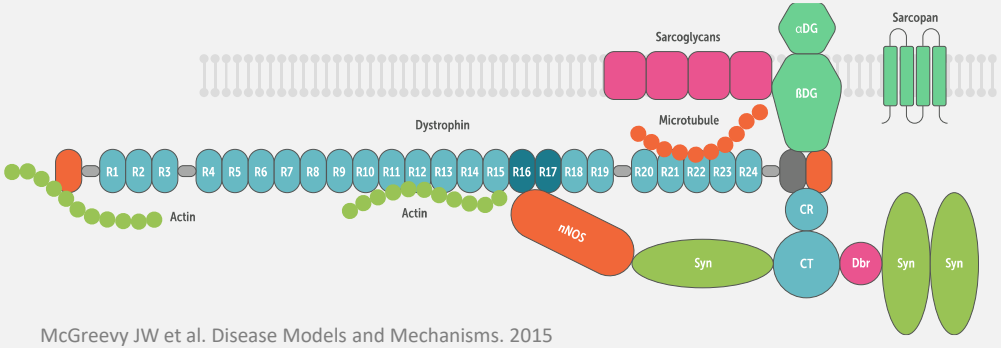


Visual representation only

Solid's nNOS Microdystrophin Construct Has Demonstrated Differentiation

Dystrophin and the Glycoprotein Complex

- Stabilizes the muscle membrane
- Acts as a molecular shock absorber
- Prevents muscle tissue damage and death
- Absent in Duchenne muscular dystrophy (Duchenne)

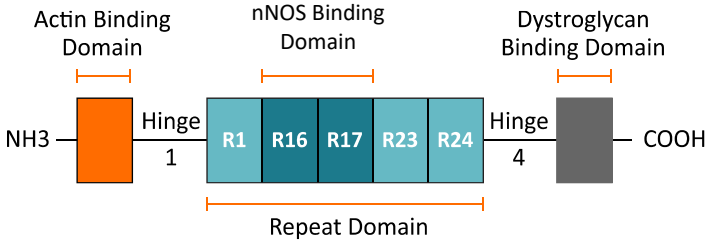


McGreevy JW et al. Disease Models and Mechanisms. 2015

Solid nNOS Microdystrophin

- Microdystrophin is a rationally designed recombinant protein
- Able to be packaged into an AAV vector
- **Uniquely includes the nNOS* binding domain**
  - Important for prevention of activity-induced ischemia and associated muscle injury
  - Presence correlated with milder phenotypes of Becker muscular dystrophy (BMD)
- Acts as a functional surrogate of full-length dystrophin

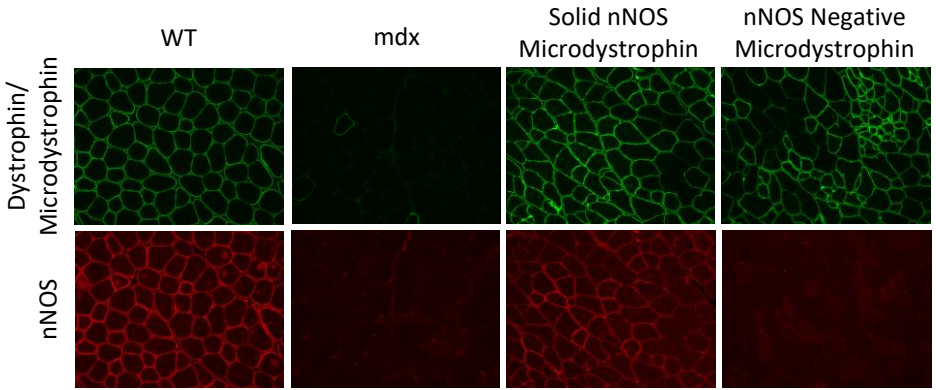
SGT-001: Retains key dystrophin protein functional domains



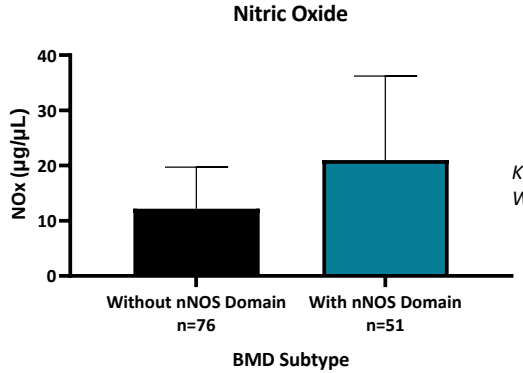
*nNOS: neuronal nitric oxide synthase

The nNOS Protein Produces Nitric Oxide, Preventing Fatigue and Muscle Injury when Anchored by Dystrophin to the Muscle Membrane

- nNOS activation at the muscle membrane results in Nitric Oxide signaling to increase blood flow to meet energy demand
- In the absence of the R16/R17 nNOS binding domain in Duchenne or BMD, Nitric Oxide signaling cannot properly occur and muscles are susceptible to ischemic injury
- Expression of the nNOS binding domain in either BMD or via a microdystrophin containing the R16/R17 domains restores nNOS and its physiological function at the membrane

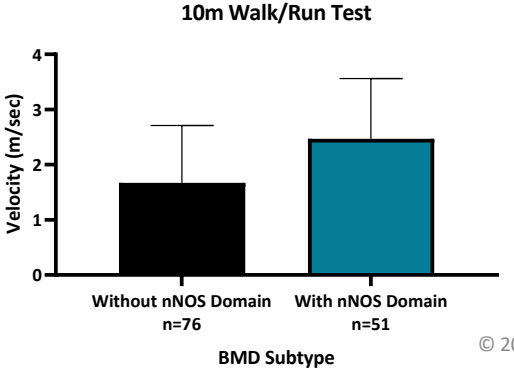


Presence of the nNOS Binding Domain is Associated with Higher Circulating Nitric Oxide in BMD Patients



Koeks, et al. WMS 2019

Clinical Evidence of Improved Muscle Function When nNOS Localization is Preserved in BMD Patients



Koeks, et al. WMS 2019

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SGT-001 / IGNITE DMD

Each Component Of SGT-001 Was Carefully Selected

Transgene → Restore key functions of a complex protein → **nNOS microdystrophin**

Promoter → Expression is highly targeted → **CK8**

Capsid → Skeletal and cardiac transduction → **AAV9**

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IGNITE DMD Clinical Trial

STUDY SUMMARY

IGNITE DMD enrollment has concluded

- 12 Total Subjects Enrolled; 9 Dosed
  - n=3 subjects analyzed as controls
  - n=3 subjects at 5E13 vg/kg
  - n=6 subjects at 2E14 vg/kg
- Both non-ambulant and ambulant subjects were enrolled
  - n=1 non-ambulant dosed (14.4 yrs at dosing); n=1 control
  - n=8 ambulant subjects dosed (4.5-10.7 yrs at dosing); n=2 controls

Primary & Secondary Endpoints (Baseline to 1 Year)

- Primary Endpoints: Incidence of adverse events and change in microdystrophin protein levels in muscle biopsies by Western blot
- Secondary Endpoints: 6-Minute Walk Test (6MWT); North Star Ambulatory Assessment (NSAA); Pulmonary Function Tests (PFTs); Quality of Life via Pediatric Outcomes Data Collection Instrument (PODCI)

For more information, please visit clinicaltrials.gov **NCT03368742**

SAFETY FINDINGS

Clinical Adverse Events

- Most Common: Nausea (9/9), vomiting (8/9), fever (7/9)
- Less Common: Cytokine release syndrome, generalized edema, acute kidney injury, thrombotic microangiopathy

Serious Adverse Events

- 3 Treatment-Associated: Systemic inflammatory response syndrome (2), thrombocytopenia (1) due to complement activation
- 2 Unrelated: Immune hepatitis (1), giardiasis (1)
- All resolved

Laboratory Abnormalities

- Most Common: Thrombocytopenia/decreased platelets, anemia, proteinuria, and increases in fibrin D-dimer, sC5b9 and LDH
- Less Common: Increased CPK, decreased complement, increased liver enzymes, increased troponin, decreased hemoglobin, increased haptoglobin, urinary casts, leukocytosis

No treatment-associated AEs have occurred in any subject after 90 days post-infusion, with follow-up periods of >6 months to >4 years

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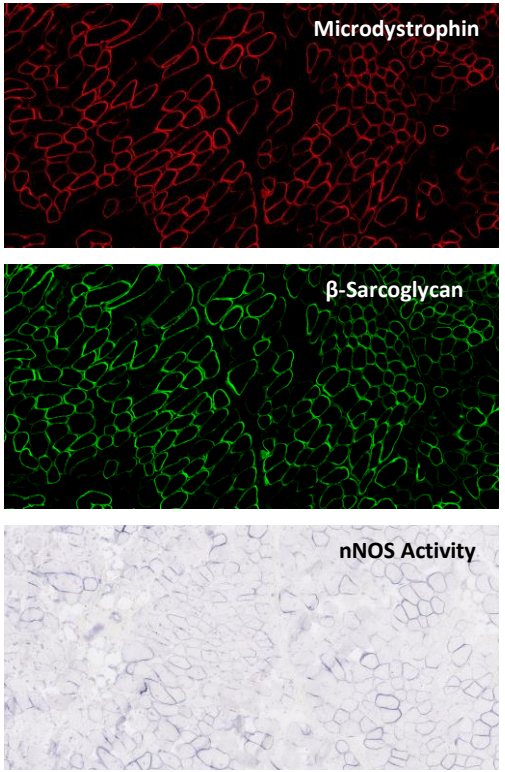
Microdystrophin Expression and Protein Function in Biopsies

Intermediate and Durable Expression and Protein Function from Biopsies Collected at 3 months and Timepoints Ranging from 12-24 Months Post-Dosing

Intermediate (3 month) and Long-term Microdystrophin Expression in Patients 4-9 (2E14 vg/kg)

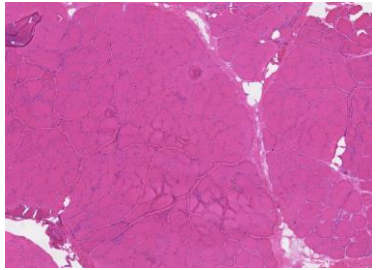
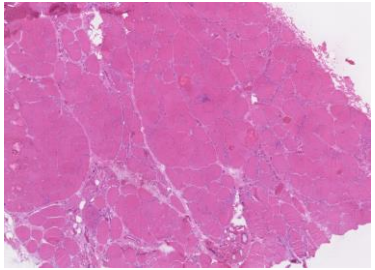
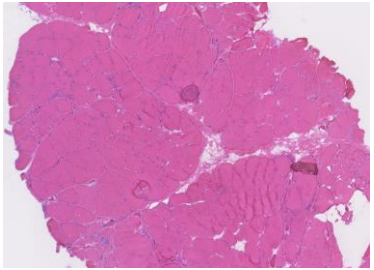
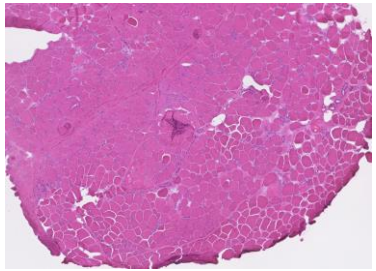
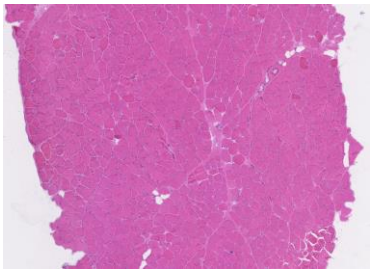
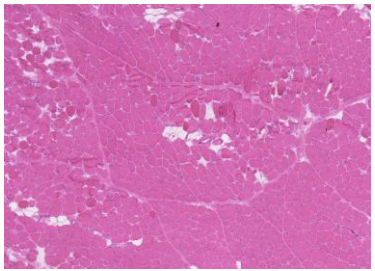
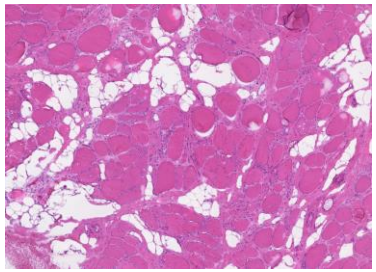
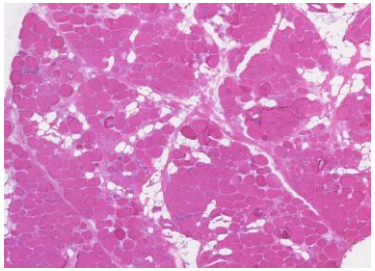
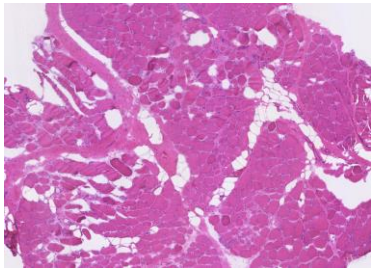
Biopsy Timepoint	Patient Number	% Positive Fibers (Immunofluorescence)	% of Normal Dystrophin (Western Blot)
3 months (n=6)	Pt. 4-9 (n=6)	1% to 70%	BLQ* to 17.5%
12 months (n=1)	Pt. 4	50-60%	20.3%
18 months (n=1)	Pt. 5	85%	69.8%
24 months (n=1)	Pt. 6	10-30%	BLQ



Biopsy from Pt. 5 at 18 months

*BLQ: Below the 5% limit of quantification

Limited Dystrophic Pathology Progression Over 12-24 Months

	Baseline	3 Months	Last Timepoint	
Pt 4 (Age at Dosing: 10.7 yrs)				**24-Month Biopsy (Age at Biopsy: 12.7 yrs)** *Very mild active dystrophic pathology*
Pt 5 (Age at Dosing: 6.9 yrs)				**18-Month Biopsy (Age at Biopsy: 8.3 yrs)** *No active dystrophic pathology*
Pt 6 (Age at Dosing: 7.7 yrs)				**12-Month Biopsy (Age at Biopsy: 8.7 yrs)** *Very mild active dystrophic pathology*

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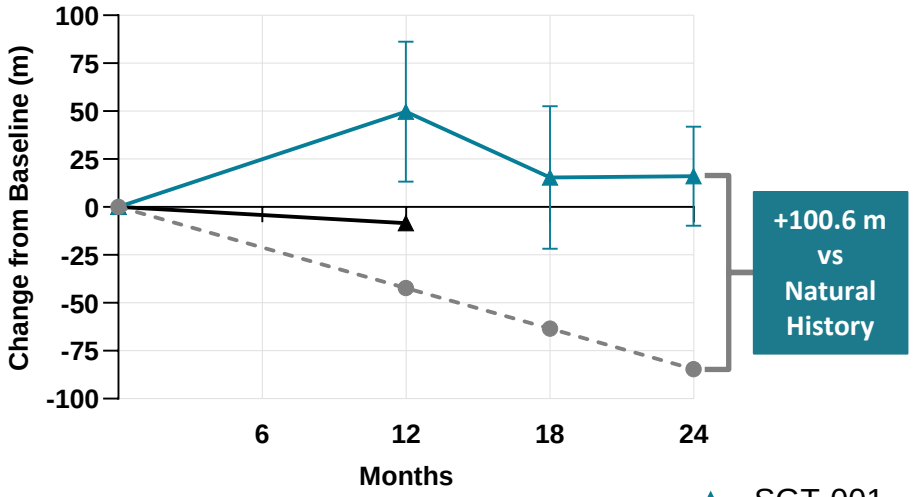
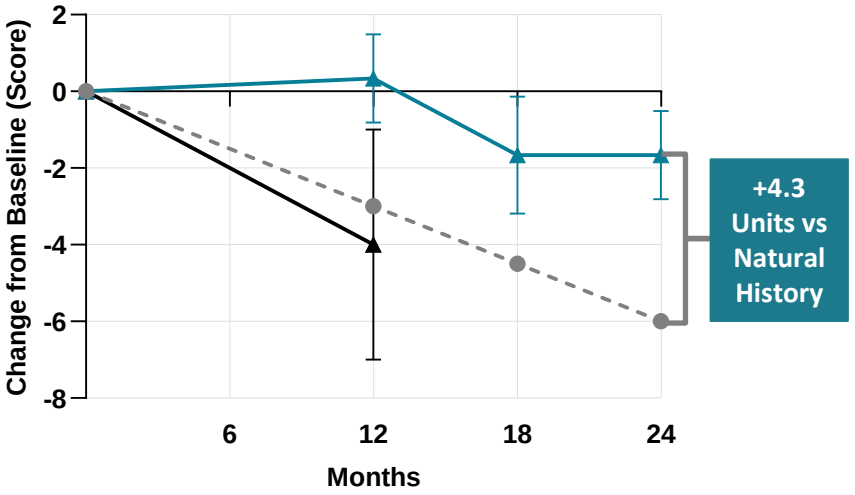
SGT-001 Treated Patients 4-6 Continue to Show Consistent, Stable Motor Function Across 6MWT and NSAA at 2 Years Post-Dosing Compared to Natural History

6 Minute Walk Test (6MWT)



North Star Ambulatory Assessment (NSAA)



SGT-001
DMD Natural History
Control (Age-Matched)

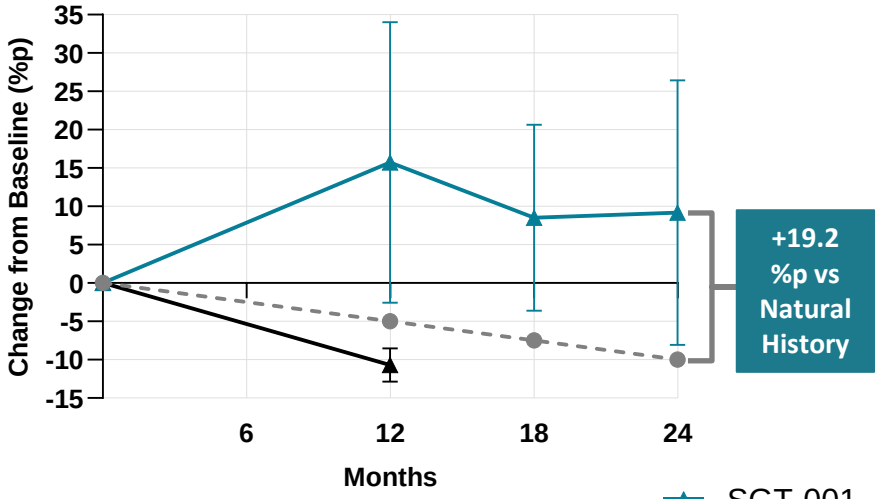
-84.6 m expected decline in 24 months after age 7 (Mercuri et al 2016)

-6.0 unit expected decline in 24 months after age 6.3 (Muntoni et al 2019)

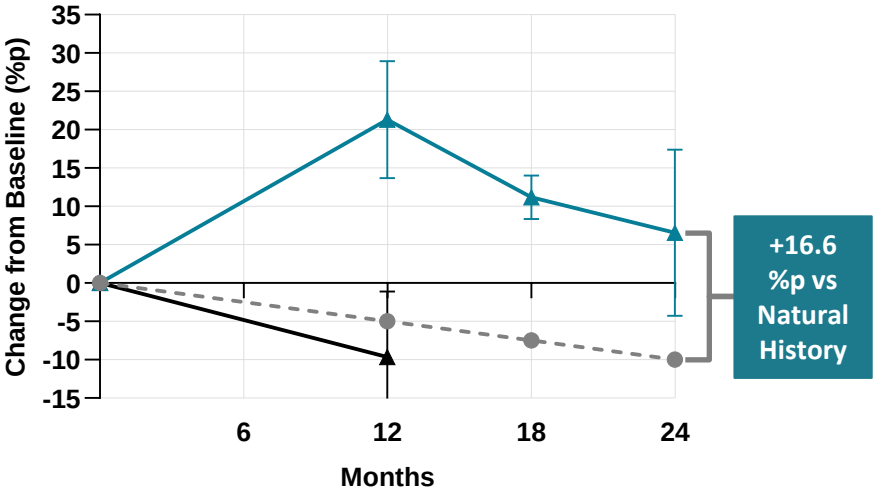
Interim data
Mean calculation based on IGNITE DMD Patients 4-6

Pulmonary Function Tests Show Durable Improvements in SGT-001 Treated Patients 4-6 across 2 Years after Dosing when Compared to Natural History

Forced Vital Capacity % Predicted (FVC %p)



Peak Expiratory Flow % Predicted (PEF %p)



SGT-001
DMD Natural History
Control (Age-Matched)

-10.0 %p expected decline in 24 months after age 6 (Mayer et al 2015)

-10.0 %p expected decline in 24 months after age 6 (Mayer et al 2015)

Interim data
Mean calculation based on IGNITE DMD Patients 4-6

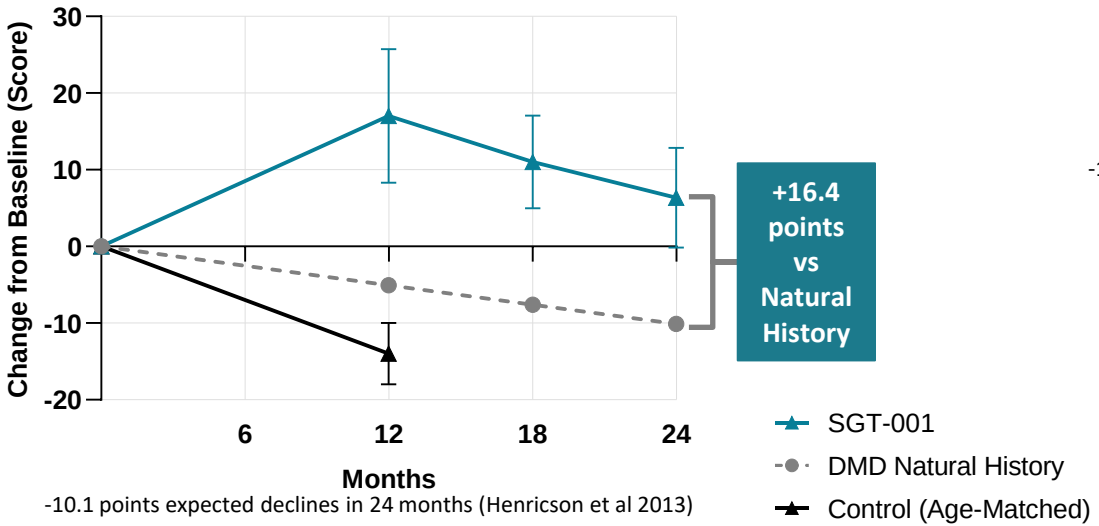
SGT-001 Treated Patients 4-6 Report Stability or Improvements in Key Functional Domains of the PODCI after 2 Years when Compared to Natural History

Supports the Potential Benefits Observed in Ambulatory and Pulmonary Functional Assessments

PODCI: Pediatric Outcomes Data Collection Instrument

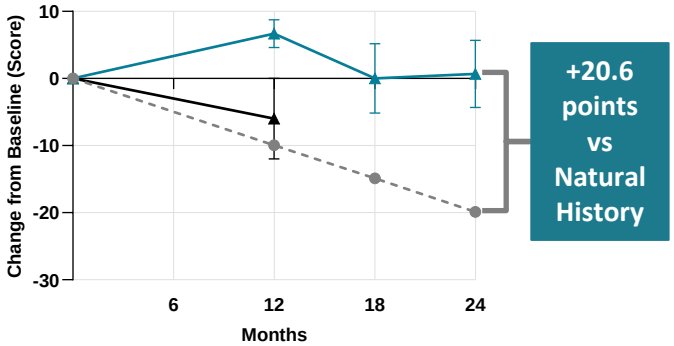
- **Method**: Questionnaire-based Patient Reported Outcome Measure
- **Domains**: Global Function, Transfer/Basic Mobility, Sports/Physical Function, Upper Extremity Function, Pain/Comfort, Happiness with Physical Condition

Global Function



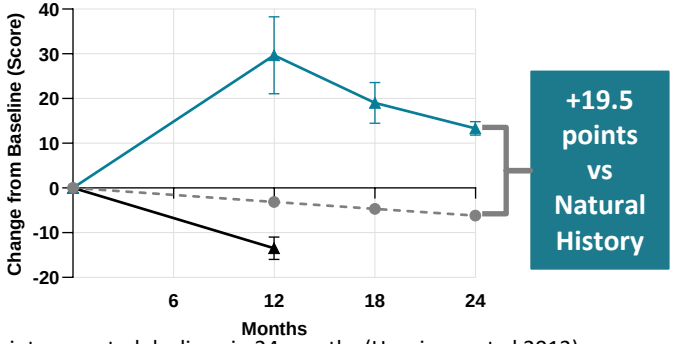
-10.1 points expected declines in 24 months (Henricson et al 2013)

Transfer / Basic Mobility



-19.9 points expected declines in 24 months (Henricson et al 2013)

Sports / Physical Functioning



-6.2 points expected declines in 24 months (Henricson et al 2013)

Interim data
Mean calculation based on IGNITE DMD Patients 4-6
Standardized scores calculated from pediatric and adolescent parent-reported responses

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Key Takeaways From Interim Analysis of IGNITE DMD

Sustained motor function

- ✓ Stable 6 Minute Walk Test (6MWT) distances and North Star Ambulatory Assessment (NSAA) scores compared to natural history

Improved pulmonary function

- ✓ Improvements in Forced Vital Capacity (FVC %p) and Peak Expiratory Flow (PEF %p) compared to baseline and natural history

Continued meaningful improvements in patient reported outcomes

- ✓ Stable or improved scores across functional domains of the PODCI compared to baseline and natural history

All patients dosed with SGT-001 in the high dose cohort have demonstrated microdystrophin expression and localization

- ✓ 90-day biopsy data from Patients 7-9 within the range of expression for Patients 4-6
- ✓ Long-term biopsy data from Patients 4-6 demonstrate durable microdystrophin expression at 12-24 months post-dosing

SGT-001 treated patients show consistent, durable improvements in function across assessments 2 years after dosing compared to expected natural history declines

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Solid's Path Forward for SGT-001

Manufacturing

Solid to transition to new process with target to have product available in early-2023

IGNITE DMD

Solid has decided to conclude enrollment in IGNITE DMD and transition to FDA engagement and planning for future clinical activities. Solid to continue monitoring patients for 5-years post-treatment and release functional data

Clinical Development

Solid to refine clinical development strategy with future patients being dosed with SGT-001 produced via transient production. Solid to initiate Natural History Study in early-2023

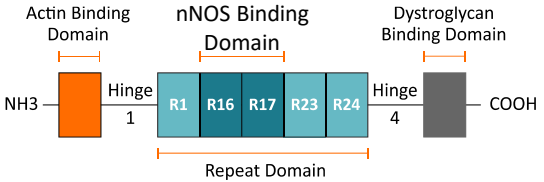
Solid expects to initiate dosing patients in 2023, following GMP production and FDA discussions

SGT-003 and Novel Capsids

SGT-003 is Rapidly Advancing to the Clinic Using a Next Generation Muscle-Tropic Capsid

Novel Capsid

- Rational design of next-generation novel capsid
- Demonstrated differentiated muscle tropism with improved efficacy per dose compared to AAV9



nNOS Microdystrophin

- Continue to deliver best-in-class, optimized microdystrophin construct with nNOS domain

Data collected on microdystrophin construct as part of SGT-001's IGNITE DMD clinical trial to support SGT-003

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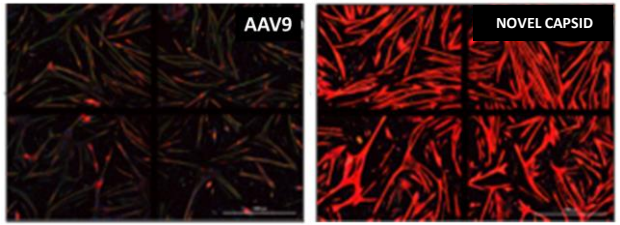
Compelling Data Resulted in Advancement of SGT-003 Program

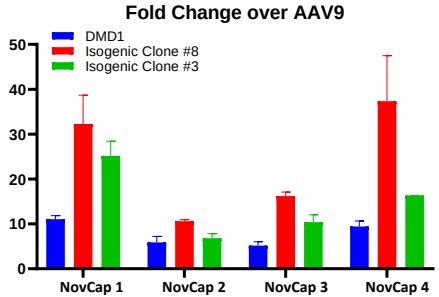
Capsid library development *in vitro* screening and *in vivo* testing

Cell Based *In Vitro* Assays
WT Mouse and Human Dystrophic Cells

C2C12 Microdystrophin Protein Expression

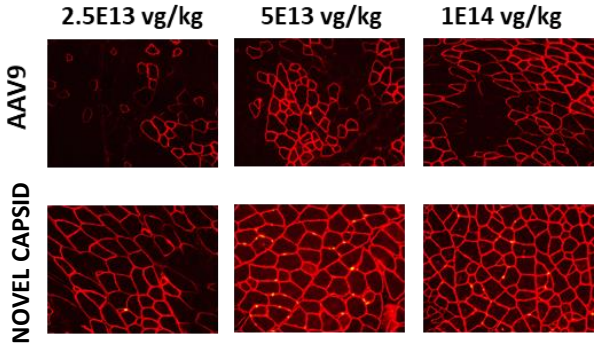


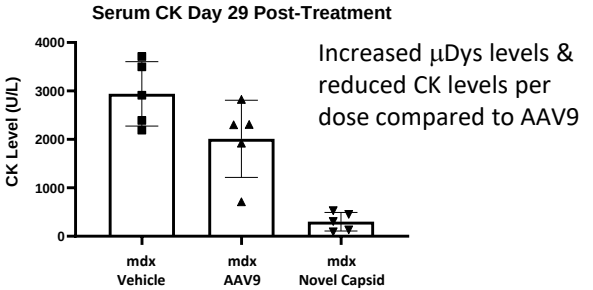
Human DMD Cell Microdystrophin Expression



In Vivo Disease Model (mdx) Testing

IF Imaging of mdx Quads



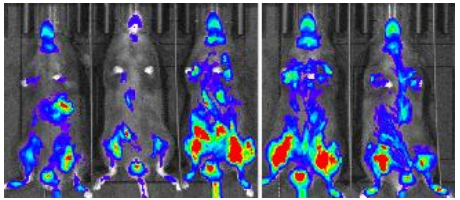


Increased µDys levels & reduced CK levels per dose compared to AAV9

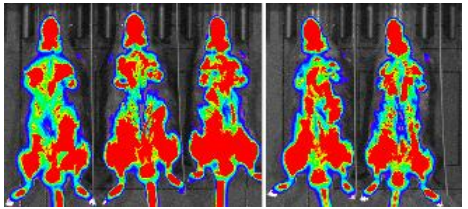
Interim data

In Vivo WT Mouse Reporter

AAV9 3E14 vg/kg



Novel Capsid 3E14 vg/kg



Advanced to Program status: SGT-003

SOLID BIOSCIENCES

Confirming Earlier Preclinical Data in a Non-Human Primate (NHP) Exploratory Study

Program Goal
Improve expression at a lower dose, allowing for decreased Total Viral Load to Patients

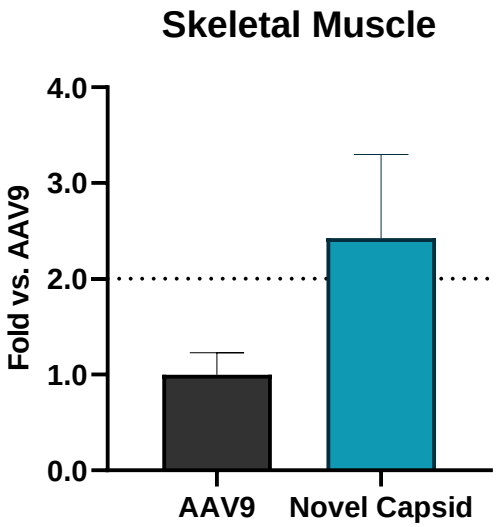
Microdystrophin Mouse Studies
Novel capsids show increases in muscle biodistribution and microdystrophin expression compared to AAV9 in dystrophic animals

Novel Capsid Mouse Models
Novel capsid library demonstrates improved muscle tropism in multiple animal models

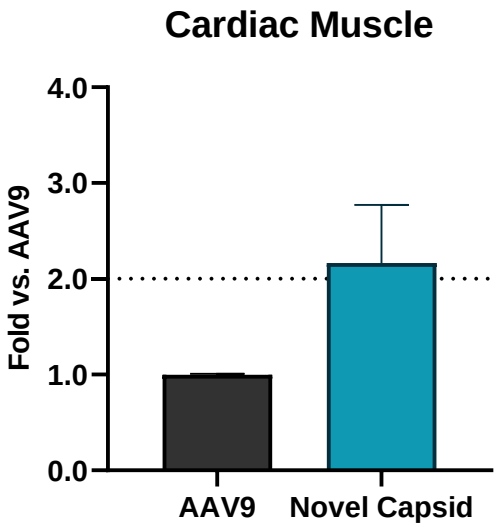
NHP Exploratory Study
Confirm safety and expression; Eight 2-year-old monkeys, 4 male / 4 female; 4 dosed with AAV9 / 4 dosed with Solid's novel Capsid

SOLID BIOSCIENCES

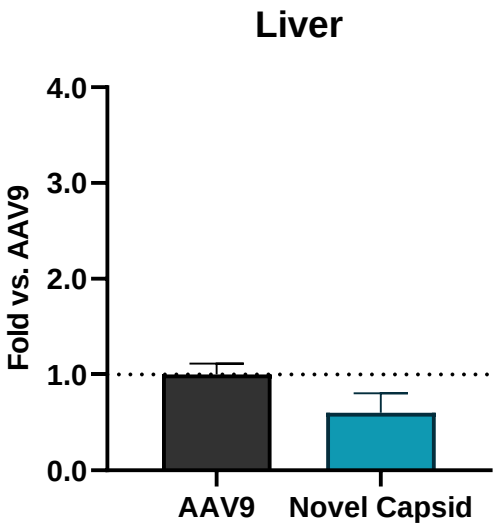
NHPs Administered a Novel Capsid Show Increased Muscle Biodistribution and Decreased Biodistribution to the Liver Relative to AAV9



>2X vs AAV9



>2X vs AAV9

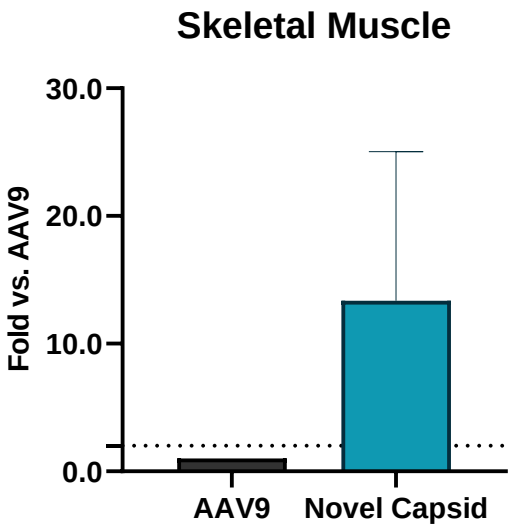


>0.5X vs AAV9

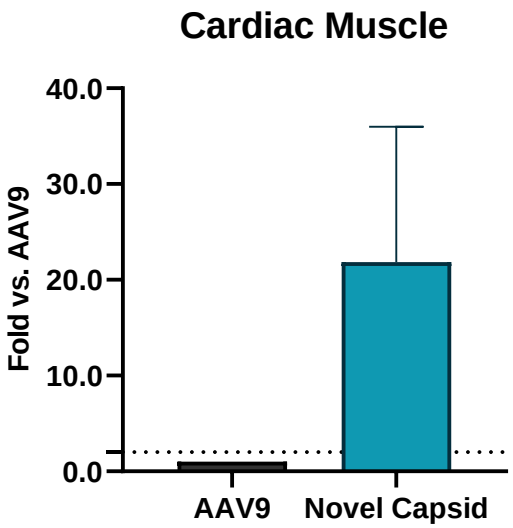
* Average fold differences calculated from the four skeletal muscle tissues sampled, three cardiac muscles sampled, and the single liver sample

SOLID BIOSCIENCES

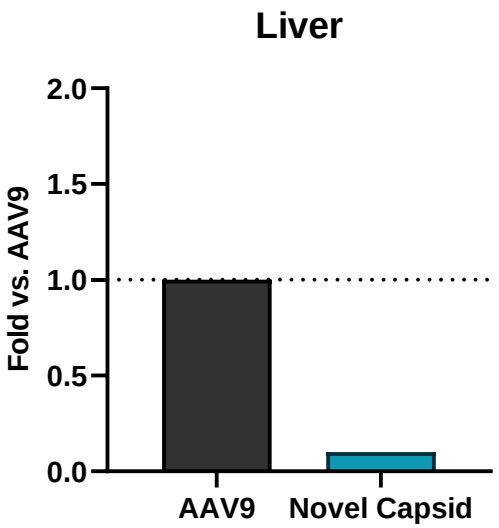
Luciferase Transgene Expression Data Support Relative Differences Observed in Biodistribution between a Novel Capsid and AAV9



>10X vs AAV9



>20X vs AAV9



<0.5X vs AAV9

* Average fold differences calculated from the five skeletal muscle tissues sampled, three cardiac muscles sampled, and the single liver sample

SOLID BIOSCIENCES

SGT-003 Moving Towards and Early 2023 IND Submission

Novel Capsid Development

- Evaluation of exploratory toxicity studies and other research activities are ongoing
- Company to continue discussions with other potential partners in muscle-related disorders

SGT-003

- Advancing toward IND submission that could enable Solid to bring next generation microdystrophin to the clinic in 2023
- Translatability activities across species continue, along with other pre-IND research activities

Totality of data is encouraging for Duchenne and potentially other muscle-related disorders

SOLID BIOSCIENCES

DRIVING THE FUTURE

Solid Biosciences Highlights

SGT-001

- IGNITE DMD functional and durability data supports program advancement
- Transition to transient transfection-based manufacturing process on track with clinical entry in 2023
- Additional IGNITE DMD data expected to be released in early-2023

SGT-003 AND NOVEL CAPSIDS

- Mid-2023 IND submission anticipated for SGT-003
- Novel capsid development continues following release of new preclinical data
  - ≥2X increases in muscle targeting; decreased liver uptake
  - ≥10X increase in reporter gene expression in muscle and heart

POSITIONED FOR SUCCESS

- $162.9m in cash and investments as of June 30, 2022 will enable Solid to advance key strategic priorities into Q2 2024

SOLID BIOSCIENCES


THANK YOU