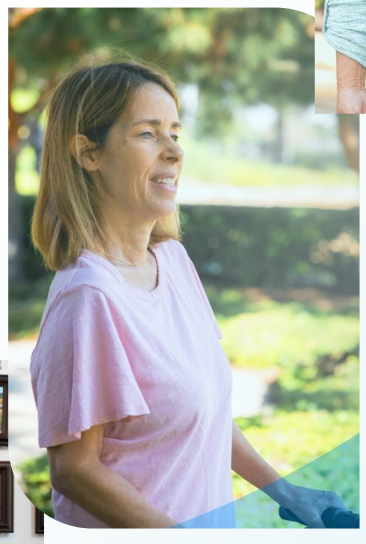
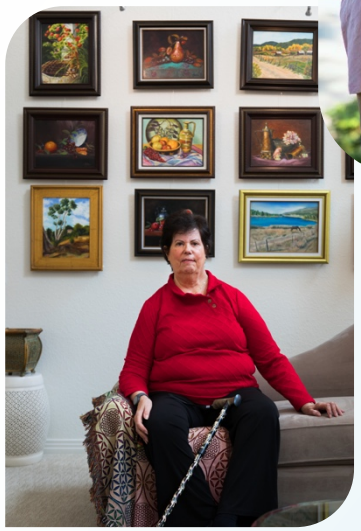
AVIDITY® BIOSCIENCES





Delivering on the RNA Revolution

May 2021

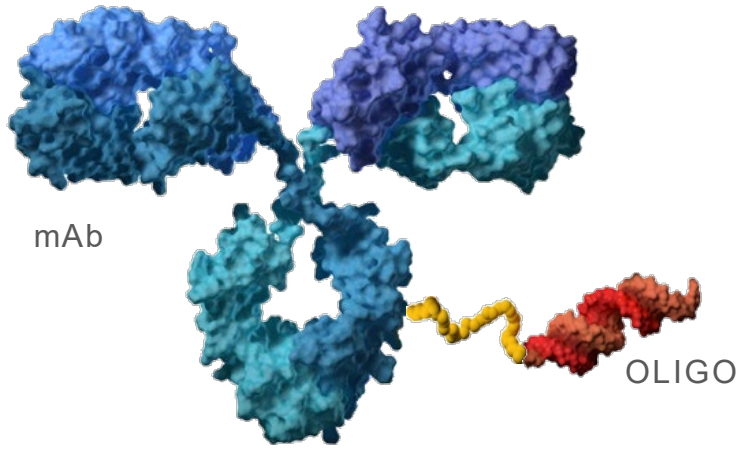
Forward Looking Statements

We caution the reader that this presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation, including statements regarding our future results of operations and financial position, business strategy, the anticipated timing, costs, design and conduct of our ongoing and planned preclinical studies and planned clinical trials, research and development plans, timing and likelihood of success, prospective products, product approvals, plans and objectives of management for future operations, and future results of anticipated product development efforts, are forward-looking statements. In some cases, the reader can identify forward-looking statements by terms such as “may,” “will,” “should,” “expect,” “plan,” “anticipate,” “could,” “intend,” “target,” “project,” “contemplates,” “believes,” “estimates,” “predicts,” “potential” or “continue” or the negative of these terms or other similar expressions. The inclusion of forward-looking statements should not be regarded as a representation by Avidity that any of our plans will be achieved. Actual results may differ from those set forth in this presentation due to the risks and uncertainties inherent in our business, including, without limitation: we are early in our development efforts and all of our development programs are in the preclinical or discovery stage; our approach to the discovery and development of product candidates based on our AOC platform is unproven, and we do not know whether we will be able to develop any products of commercial value; the success of our preclinical studies and clinical trials for our product candidates; the results of preclinical studies and early clinical trials are not necessarily predictive of future results; potential delays in the commencement, enrollment and completion of clinical trials; our dependence on third parties in connection with preclinical testing and product manufacturing; disruption to our operations from the COVID-19 pandemic; unexpected adverse side effects or inadequate efficacy of our product candidates that may limit their development, regulatory approval and/or commercialization, or may result in recalls or product liability claims; regulatory developments in the United States and foreign countries, including acceptance of INDs and similar foreign regulatory filings and our proposed design of future clinical trials; our ability to obtain and maintain intellectual property protection for our product candidates and proprietary technologies; we may use our capital resources sooner than we expect; and other risks described in our filings with the SEC, including under the heading “Risk Factors” in our Form 10K for the year ending on December 31, 2020, filed with the SEC on March 15, 2021, and any subsequent filings with the SEC. The reader is cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date hereof, and except as required by applicable law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise. All forward-looking statements are qualified in their entirety by this cautionary statement, which is made under the safe harbor provisions of the Private Securities Litigation Reform Act of 1995.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and the reader is cautioned not to give undue weight to such 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. These and other factors could cause results to differ materially from those expressed in the estimates made by the independent parties and by us.

AOCs – A Powerful New Class of Drugs

Utilizing decades of proven science to unlock the power of oligonucleotides



ANTIBODY OLIGONUCLEOTIDE CONJUGATE (AOC)

- Combines the proven and safe technologies of monoclonal antibodies and oligonucleotides
  - ✓ Specificity of targeting with mAbs
  - ✓ Potency & precision of oligonucleotides
  - ✓ Targets tissues with potent and durable agents
- Engineered to deliver to tissues previously untreatable with RNA therapeutics
- Focused first on muscle, broadening to other tissues (i.e. cardiac) and cell types (i.e. B Cells)
- Readily scalable with many experienced manufacturers

Delivering on Our Vision

DISRUPTIVE & BROAD
PLATFORM

- Delivering a New Class of RNA Therapies
- Demonstrating preclinical proof of concept in multiple tissues
- Broadening to other tissues & cell types through partnerships & internal discovery

ADVANCING & EXPANDING
PIPELINE

- Progressing robust pipeline in muscle
- Entering the clinic with AOC 1001 in 2H 2021
- Planning for clinical initiations in FSHD & lead DMD program in 2022

AGILE & DIVERSE
COMPANY

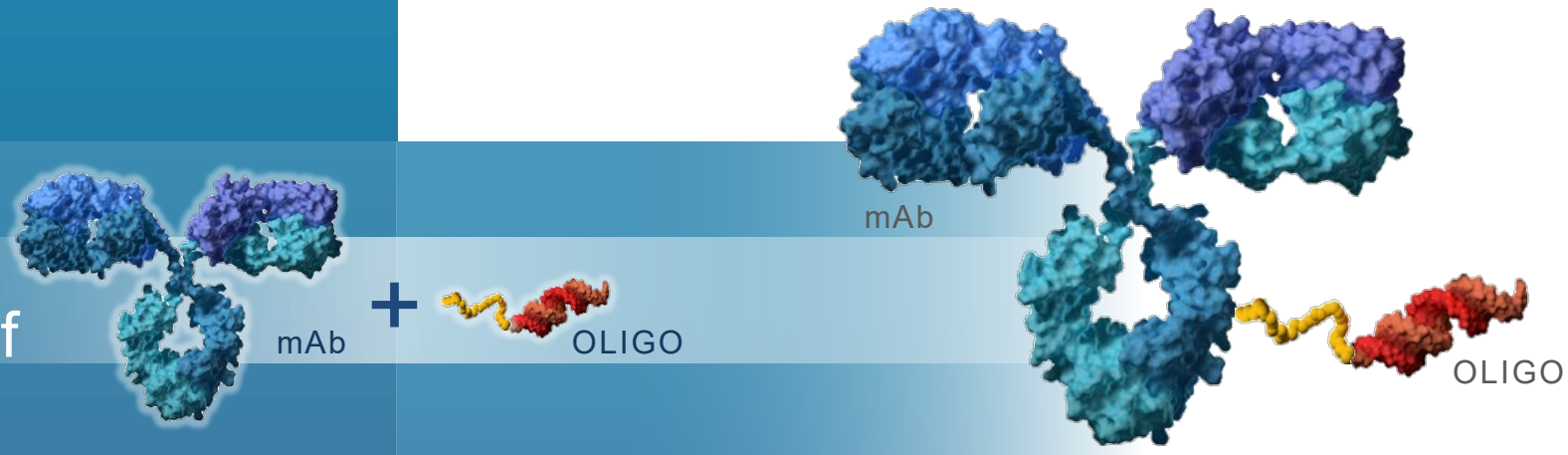
- Leveraging expertise in clinical and commercial execution
- Assembling an experienced team in rare & RNA therapies
- Building an integrated and diverse company in service of our patients

Our AOC™ Platform

A New Class of RNA Therapeutics

Engineering an AOC Therapeutic

Combine the potency and precision of oligonucleotide therapies with the specificity of mAbs



MONOCLONAL ANTIBODIES

OLIGONUCLEOTIDE THERAPIES

ANTIBODY OLIGONUCLEOTIDE CONJUGATE (AOC)

AVIDITY BIOSCIENCES

AOCs: Designed to Deliver Efficacious & Safe Therapeutics

Multiple programs utilizing two proven technologies

mAbs

- Over 100 approved drugs
- Suitable for chronic therapies because of well-established safety profile
- High specificity and affinity
- Readily reproducible with many experienced manufacturers
- Long half-life

siRNA

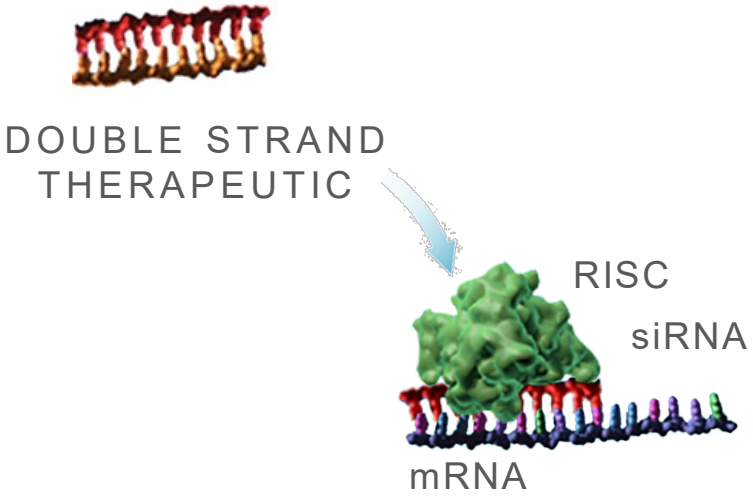
- Multiple approved drugs
- Attractive safety profile with no known thrombocytopenia, liver or renal toxicity
- Potency in the nanomolar range
- Readily reproducible with many experienced manufacturers
- Sustained activity in both cytoplasm and the nucleus

Proven Safe Durable Scalable Potent

Optimizing the Specificity of our AOCs
Targeting the RNA with the Right Oligonucleotide

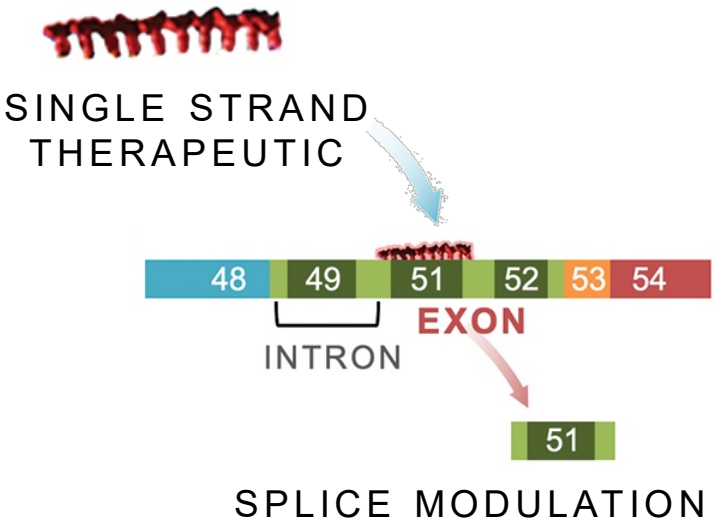
RNA DEGRADATION

Reducing the expression of a disease-related RNA with siRNAs



RNA SPLICING

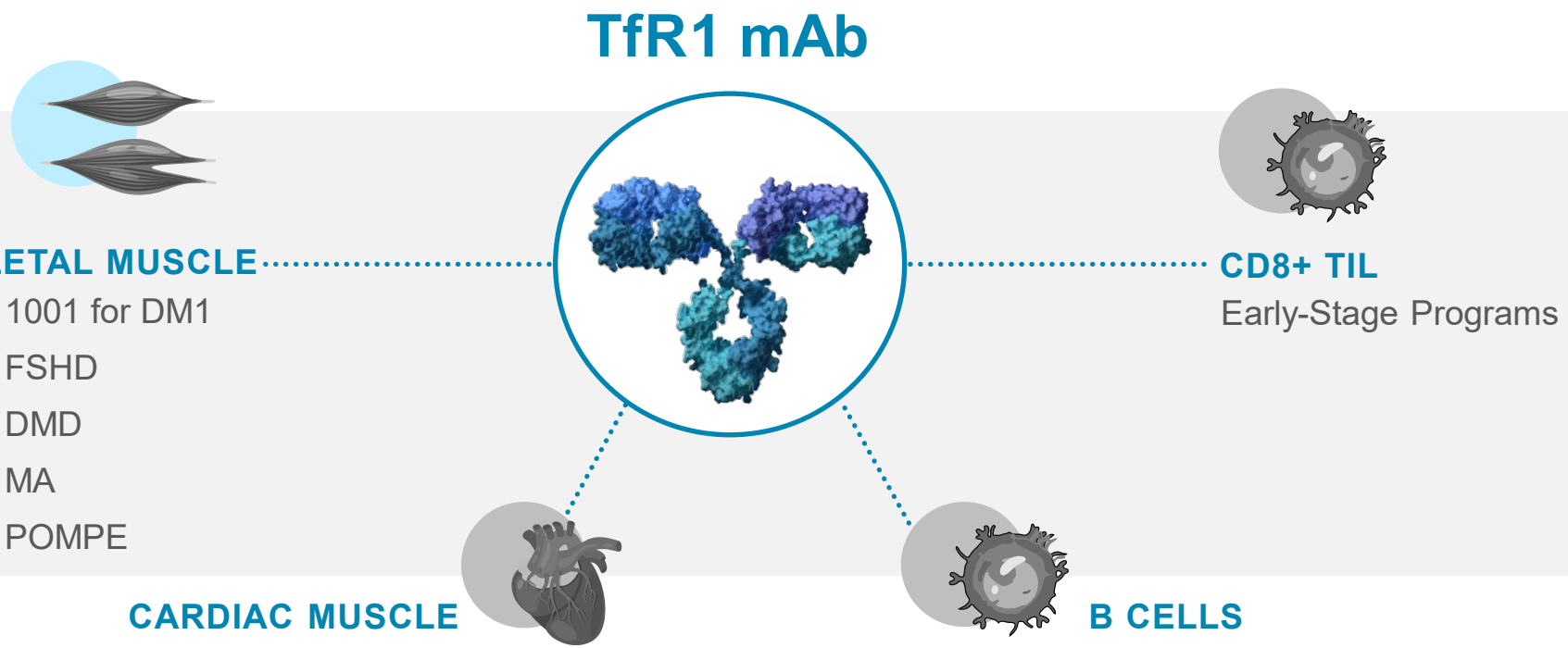
Correction of aberrant processing of RNAs with splice modifying oligonucleotides



We use oligonucleotides that are tailored to modulate a given disease process

AVIDITY BIOSCIENCES

Leveraging the Receptor-Mediated mAb Across Multiple Programs and Tissue Types



AOC Platform: Delivering on the RNA Revolution

Expanding scope of diseases beyond liver	Selecting most potent oligonucleotide type	Enabling infrequent dosing	Readily reproducible and scalable
Targeting new tissue and cell types with specificity, including muscle, immune cells and others	Achieving ED50s at the nanomolar concentration	Maximizing durability with sustained single dose RNA reductions in NHP beyond 12 weeks	Utilizing the same monoclonal antibody across multiple programs

Advancing our Muscle Disease Franchise of AOCs

PROGRAM / INDICATION	TARGET	DISCOVERY/ LEAD OPTIMIZATION	IND ENABLING	CLINICAL
MUSCLE DISORDERS				
AOC 1001: Myotonic Dystrophy Type 1 (DM1)	DMPK			Planned Phase 1/2 Trial in H2 2021
AOC FSHD: Facioscapulohumeral Muscular Dystrophy (FSHD)	DUX4		Clinical trial initiations planned for 2022	
AOC DMD: Duchenne Muscular Dystrophy (DMD)	Exon 44 Dystrophin		Clinical trial initiations planned for 2022	
	Exon 51 Dystrophin			
	Exon 45 Dystrophin			
AOC Muscle Atrophy: Muscle Atrophy*	MuRF1			
AOC Pompe Disease: Pompe Disease	GYS1			

AVIDITY BIOSCIENCES

* Opportunity for a rare disease indication

AOC 1001 for Myotonic Dystrophy Type 1 (DM1) Program

"Some days I don't have the energy to take another step."

Karin, Living with DM1

Myotonic Dystrophy Type 1: Disease Overview

>40,000
PEOPLE WITH DM1 IN THE US

SIMILAR PREVELANCE
ESTIMATES FOR EUROPE

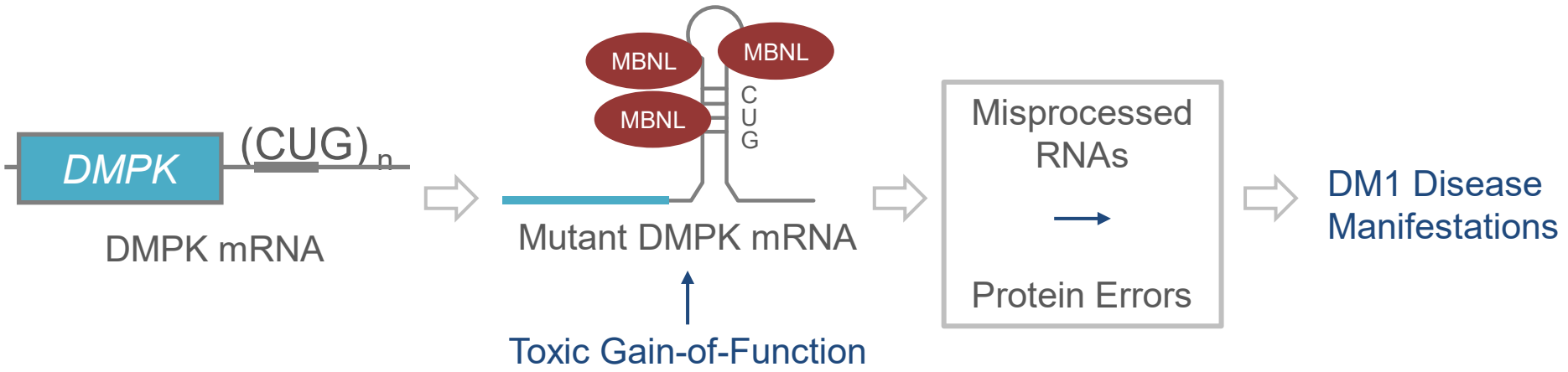
0
APPROVED THERAPIES

- DM1 is a complex disease with symptoms that present with high variability from patient to patient
- Monogenic, autosomal dominant, progressive disease that primarily affects skeletal and cardiac muscle
- Mutated dystrophy myotonic protein kinase (DMPK) gene leads to misprocessing of multiple pre-mRNAs that encode key proteins
- Myotonia and muscle weakness, respiratory problems, fatigue, hypersomnia, cardiac abnormalities, severe gastrointestinal complications, and cognitive and behavioral impairment

DM1, Caused by a Toxic Gain-of-Function mRNA, is Well Suited to an siRNA Approach

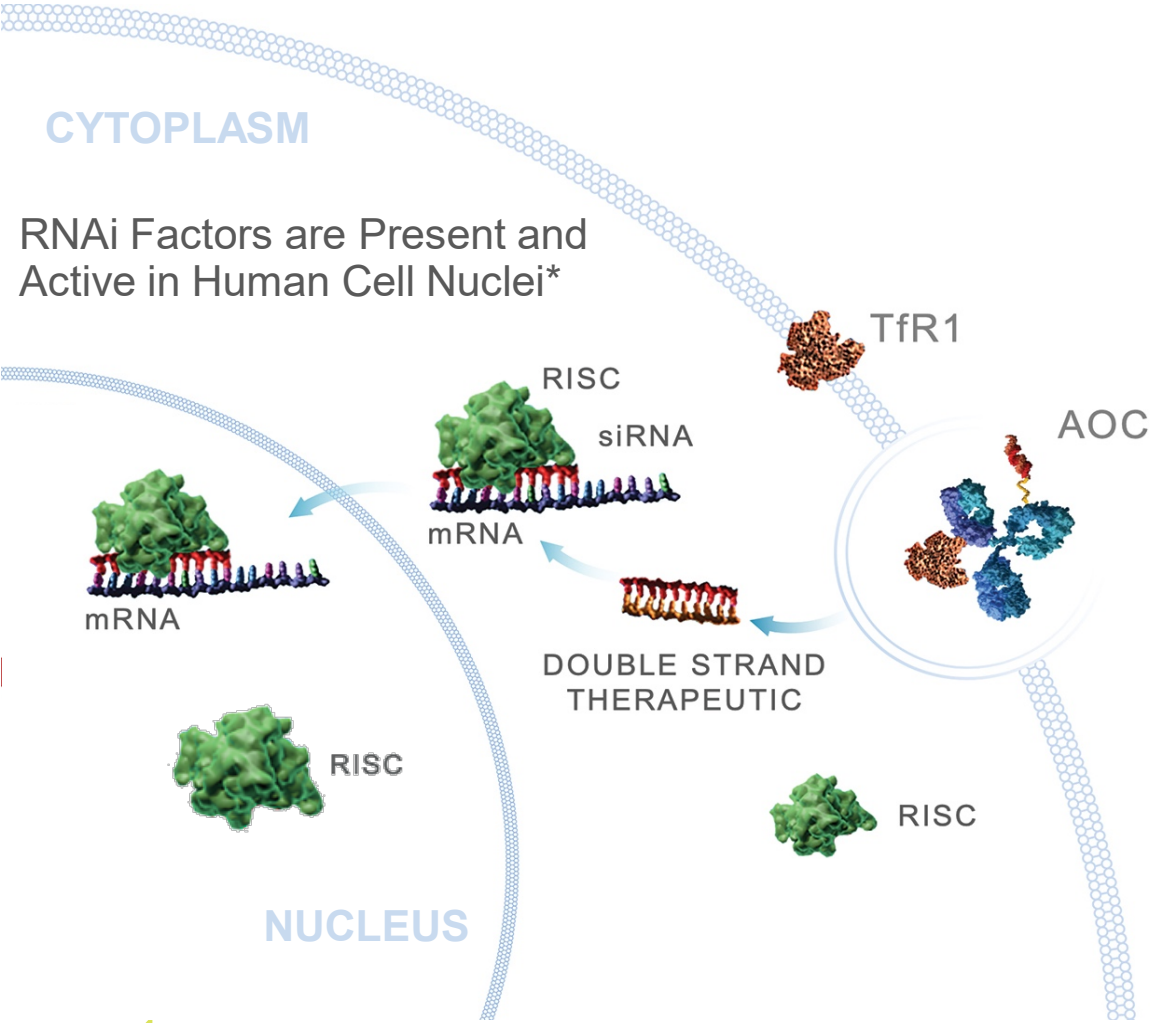
MECHANISM OF DISEASE:

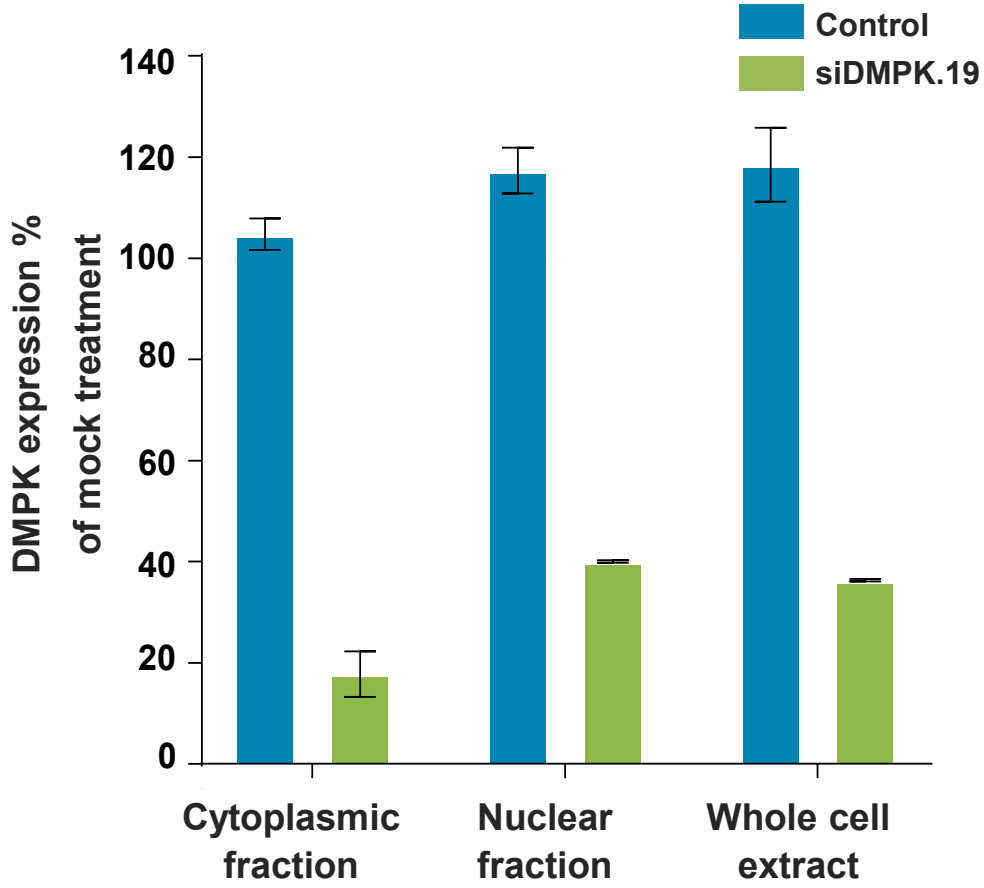
DM1 is Caused by a Toxic Gain-of-Function of the Mutant DMPK mRNA



- Trinucleotide expansion in DMPK mRNA sequesters muscleblind-like (MBNL), an RNA splicing protein
- Depleted MBNL leads to RNA splicing errors in multiple muscle-related RNAs
- Therapeutic Approach: Reduce DMPK mRNA to minimize RNA splicing errors, improve muscle function, and reverse the course of the disease

AOCs are Active in the Nucleus and Cytoplasm



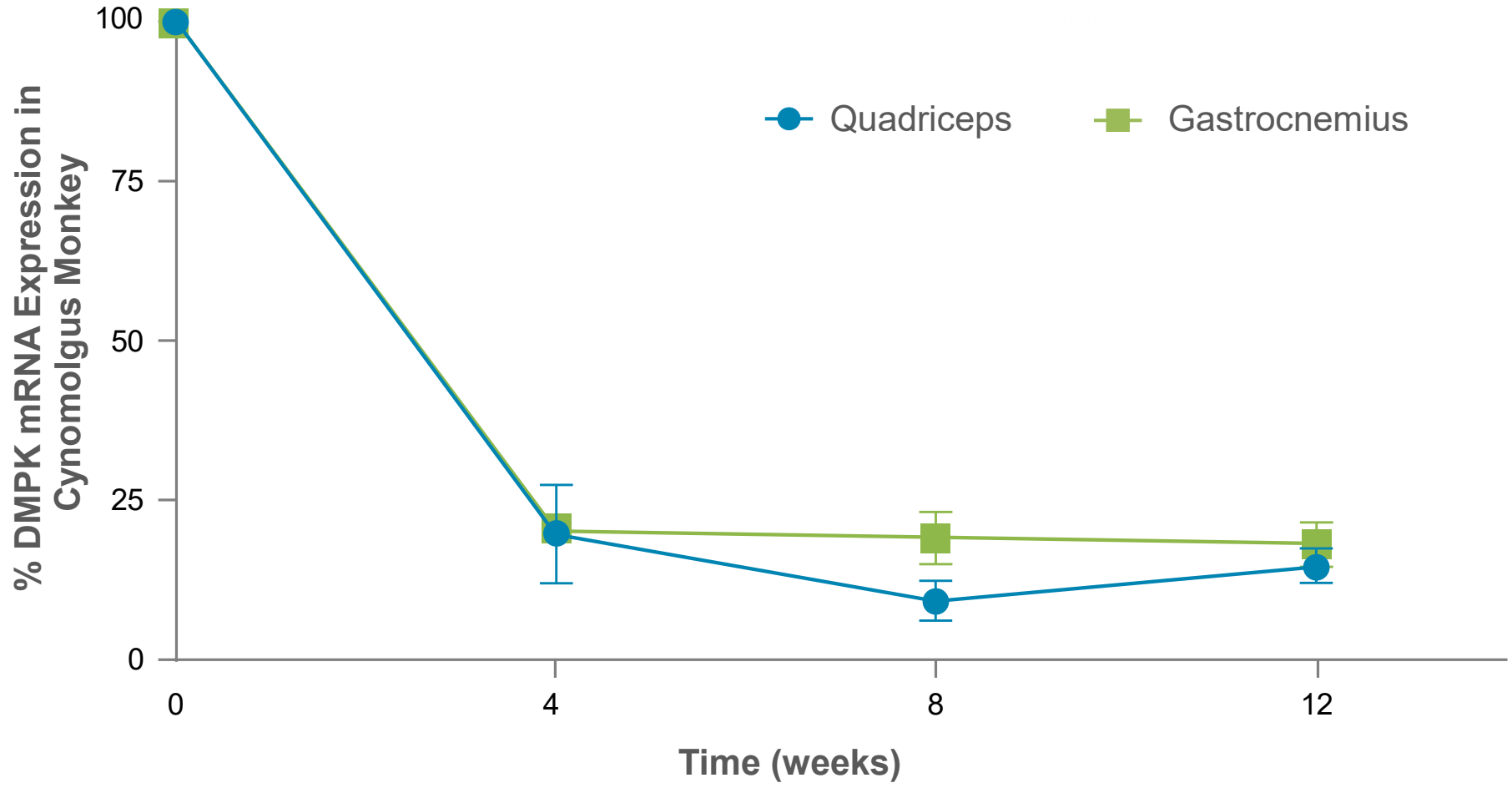


Nuclear and Cytoplasmic DMPK mRNA Levels in DM1 Patient-Derived Muscle Cells; Data: N=2 mean with range

AVIDITY BIOSCIENCES

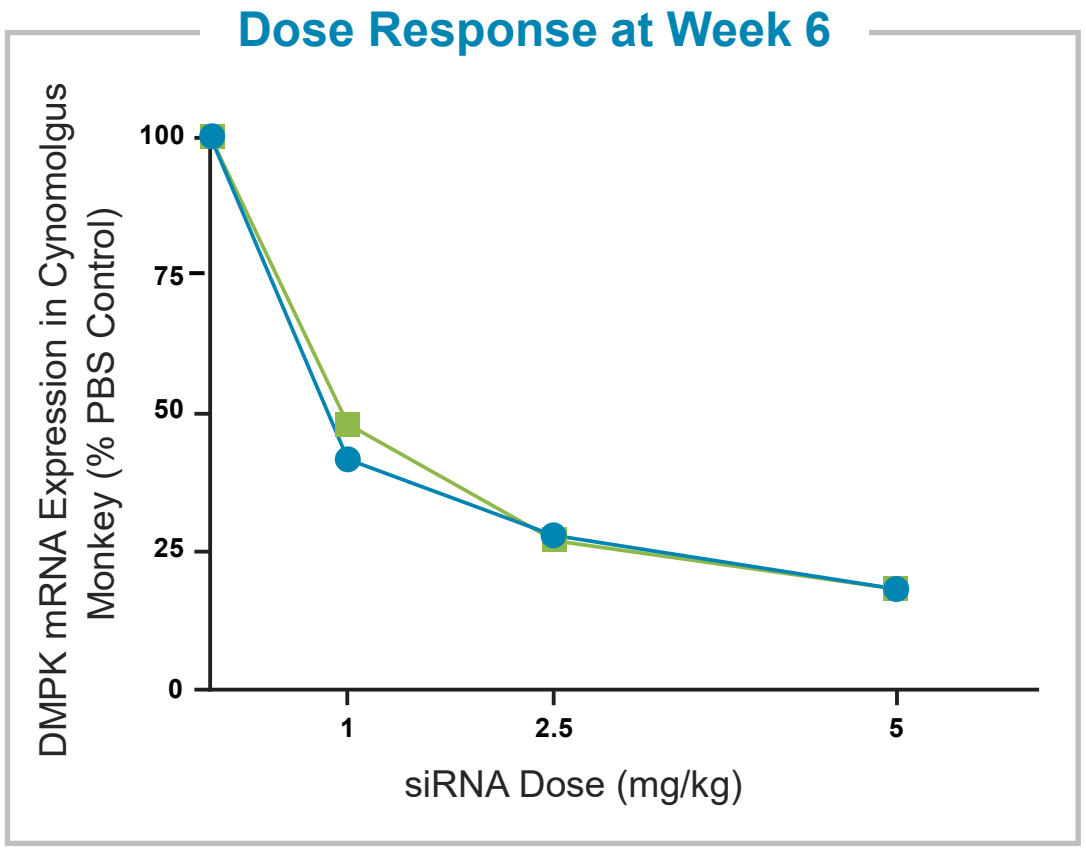
*Gagnon et al. Cell Rep. (2014) 6: 211-21

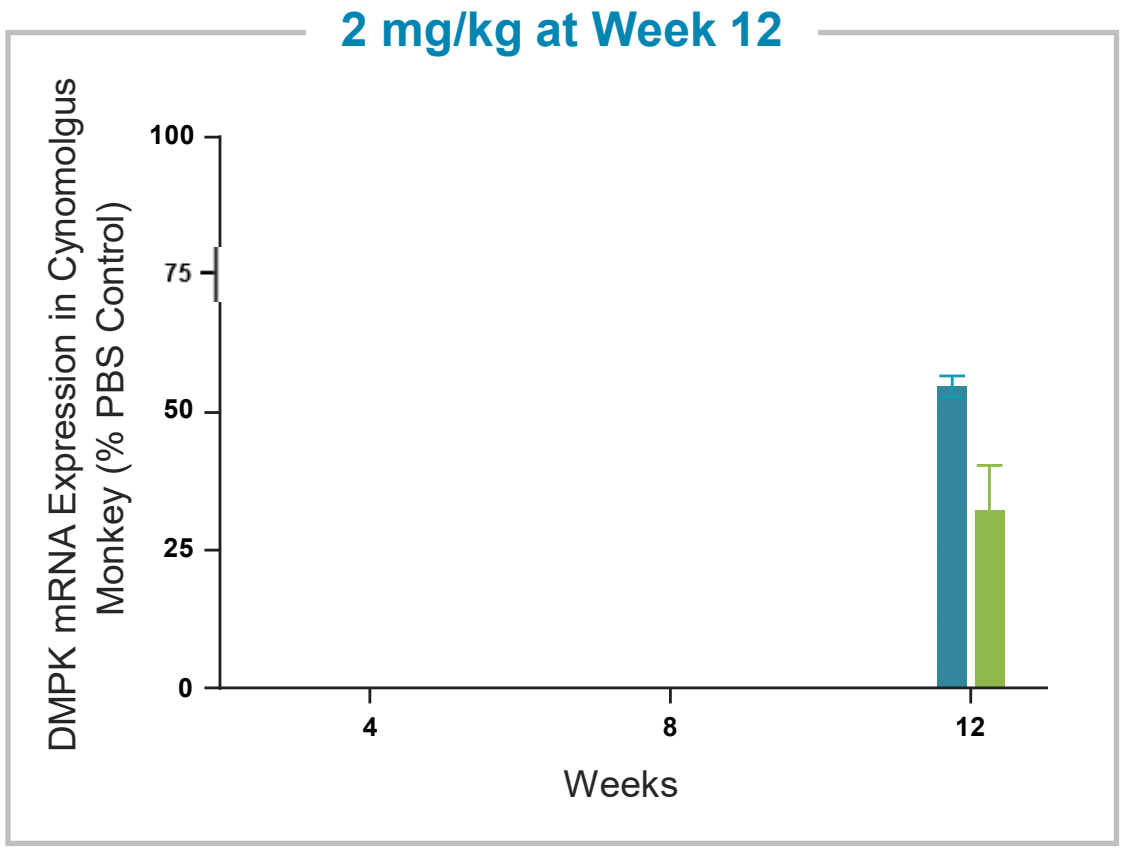
~75% Reduction of DMPK mRNA in Skeletal Muscles After a Single Dose of 2mg/kg of siDMPK.19



AVIDITY BIOSCIENCES

Robust and Durable Reductions of DMPK Levels in Cardiac Muscle and Diaphragm After a Single Dose

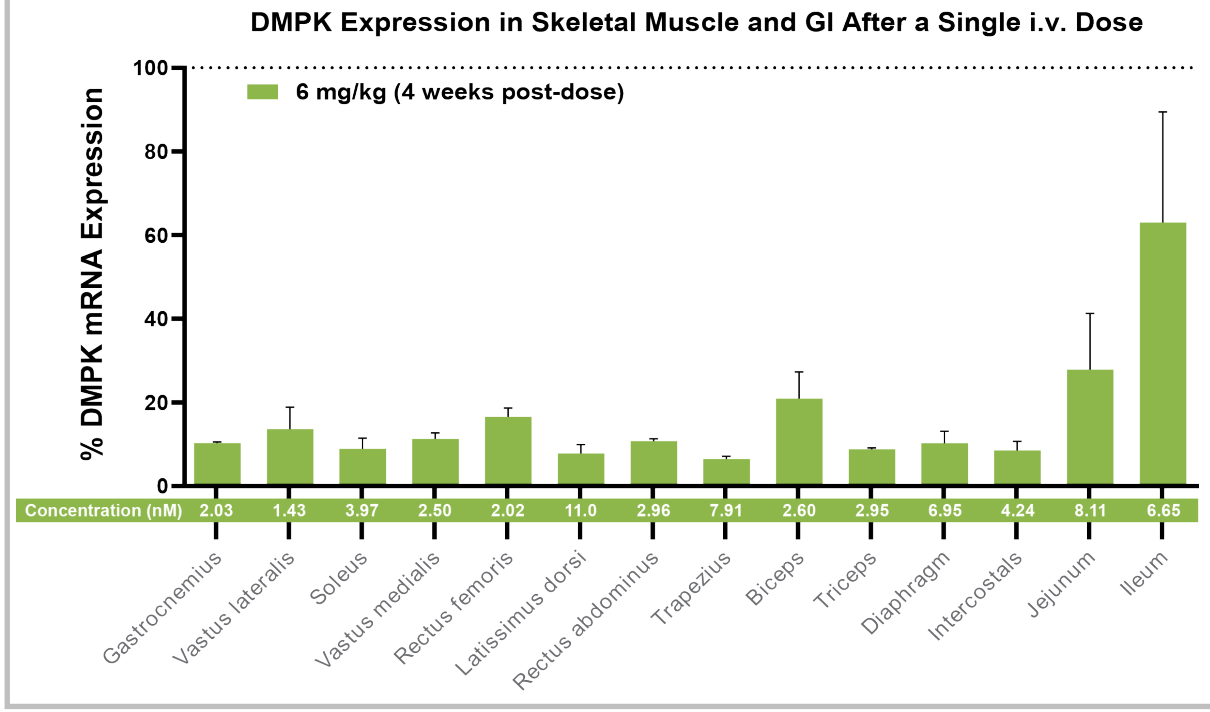


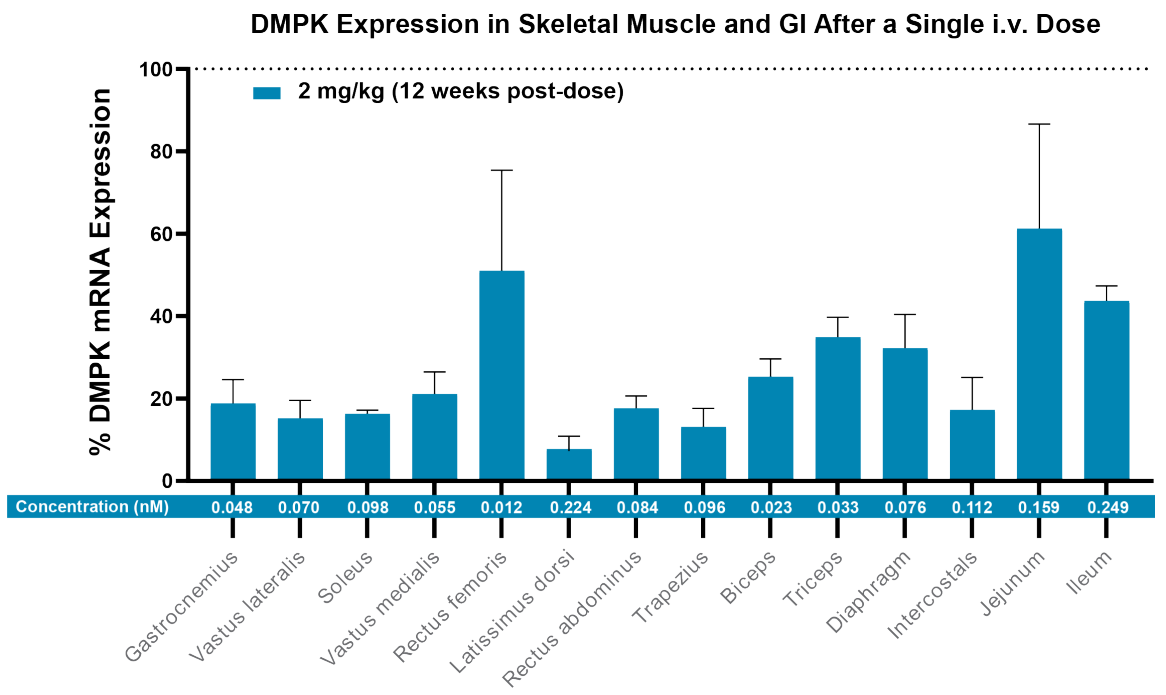


Heart Diaphragm

Reduction of DMPK mRNA in a Wide Range of Skeletal Muscle and GI Tissues in Cynomolgus Monkey at nM Concentrations

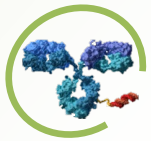
DMPK Expression in Skeletal Muscle and GI After a Single i.v. Dose





AOC 1001 for DM1 Has a Favorable Preclinical Profile

- ✓ Reduction of target mRNA in relevant disease models
- ✓ Long duration of action
- ✓ Well-established and scalable methods for manufacturing
- ✓ US Patent No. 10,881,743 for AOC 1001 issued in January 2021



DELIVERING ON DM1

APPROACHING THE CLINIC

Planned Phase 1/2 study initiation in **H2 2021**

AVIDITY BIOSCIENCES

Facioscapulohumeral Muscular Dystrophy (FSHD) Program

"Living with FSHD feels like an imprisonment in your own body."

Amy, Living with FSHD

FSHD Disease Overview

AFFECTS

~16,000 - 38,000
PEOPLE IN THE US

~1 in 20,000
INDIVIDUALS IN THE US

0
APPROVED THERAPIES

- FSHD is one of the most common forms of muscular dystrophy characterized by progressive skeletal muscle loss; initial signs appear in the face, shoulders, arms and trunk
- The disease is caused by the abnormal expression of DUX4 (double homeobox 4), a gene involved in embryonic development but not typically expressed in adults

FSHD is Caused by Aberrant Expression of DUX4 Gene After Birth

HEALTHY

DUX4 is located on human chromosome 4, in the D4Z4 region

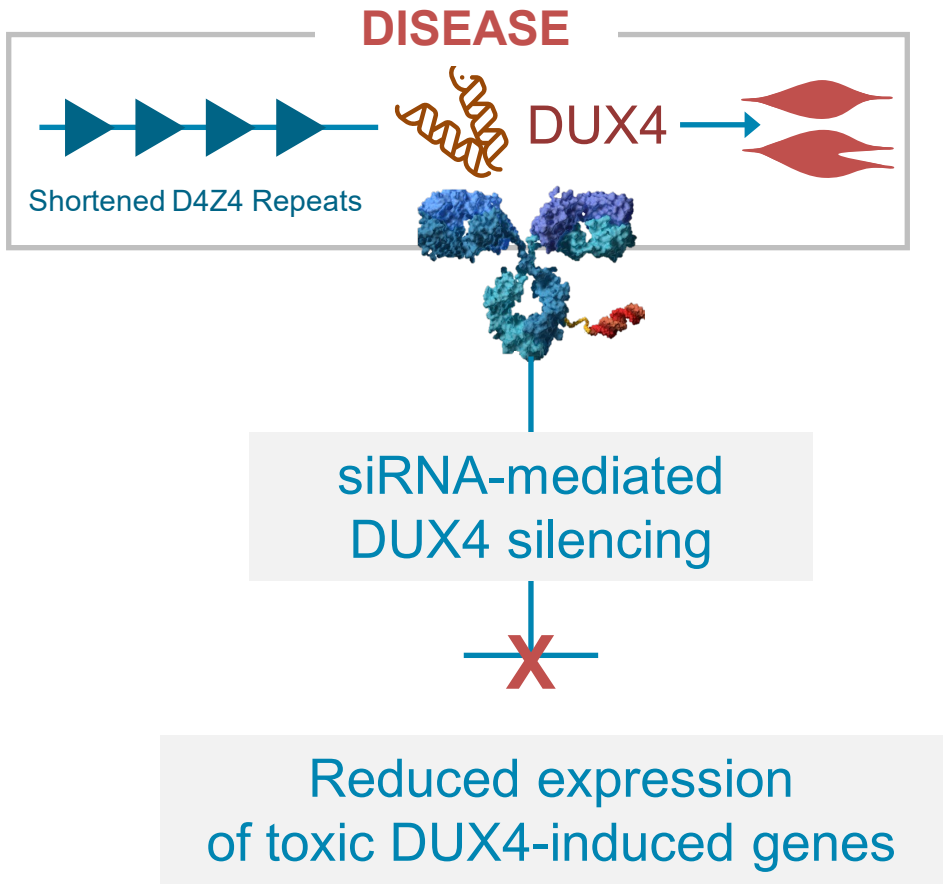
- DUX4 is normally only expressed during embryonic development
- DUX4 gene is repressed in healthy muscles

DISEASE

D4Z4 is open and expressed in FSHD muscles

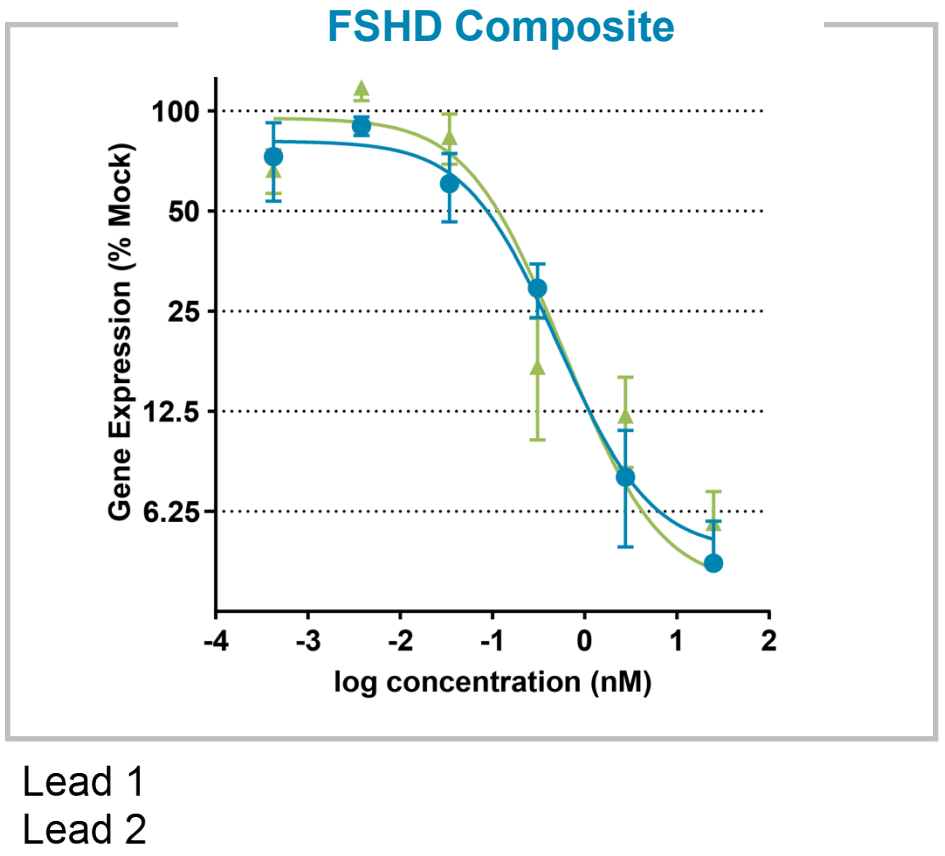
- DUX4 creates changes in gene expression that result in muscle degeneration
- Inappropriate DUX4 expression in muscles thus results in FSHD

Our AOC is Designed to Treat the Underlying Cause of FSHD



- Our AOC FSHD silences DUX4 expression to inhibit the expression of toxic downstream genes to a more normal state

Potent Reductions in DUX4 Target Gene Expression in Muscle Cells with Lead siRNA Candidates

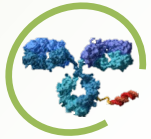


- Reduction in gene expression in vitro of a composite of 4 known biomarkers in cells from patients with FSHD
- Similar results were observed across a range of cells from patients
- Lead siRNA candidates are very potent, with IC_{50}<1nM

Genes in composite LEUTX, MBD3L2, ZSCAN4, KHDC1L

FSHD Program Summary

- ✓ Aberrant expression of DUX4 well established as cause of FSHD
- ✓ Mouse model with human DUX4 gene available
- ✓ AOC FSHD reduced expression of key DUX4 biomarkers in FSHD patient myotubes
- ✓ Natural history study planned



DELIVERING ON FSHD

PRECLINICAL STUDIES ONGOING

Planned regulatory filing to support initiation of a clinical trial in **2022**

AVIDITY BIOSCIENCES

Duchenne Muscular Dystrophy (DMD) Program

Three programs advancing toward the clinic

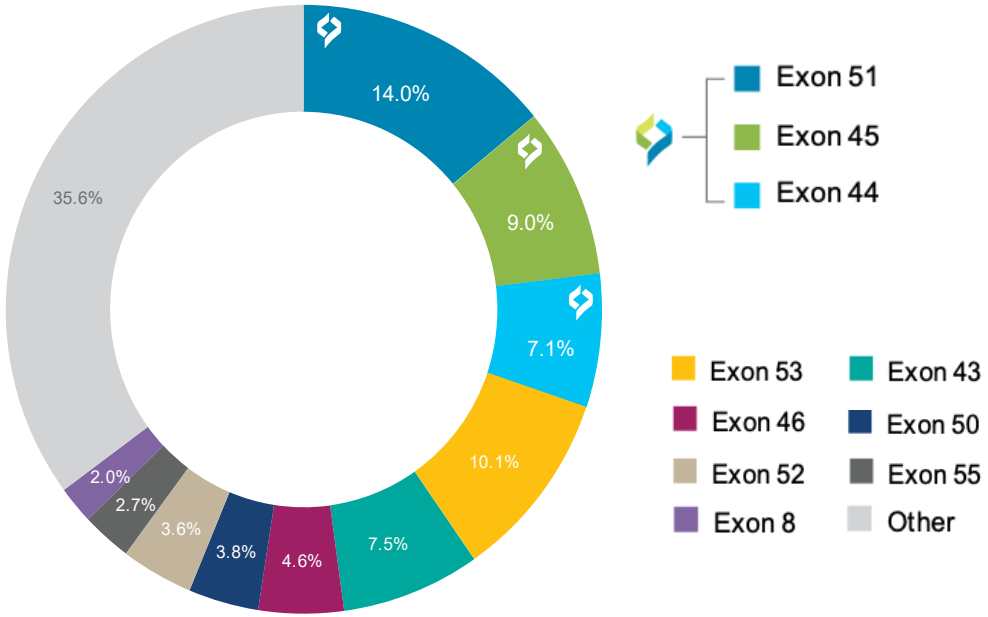
Duchenne Muscular Dystrophy: Disease Overview

~10,000 - 15,000
DMD BOYS IN THE US

SIMILAR PREVELANCE
ESTIMATES FOR EUROPE

APPROVED THERAPIES HAVE NOT ESTABLISHED A CLINICAL BENEFIT

- DMD is a monogenic, X-linked, recessive, neuromuscular disease
- Caused by mutations in the DMD gene, which encodes for the protein “dystrophin”
- Lack of functional dystrophin leads to stress and tears of muscle cell membranes, resulting in muscle cell death and the progressive loss of muscle function

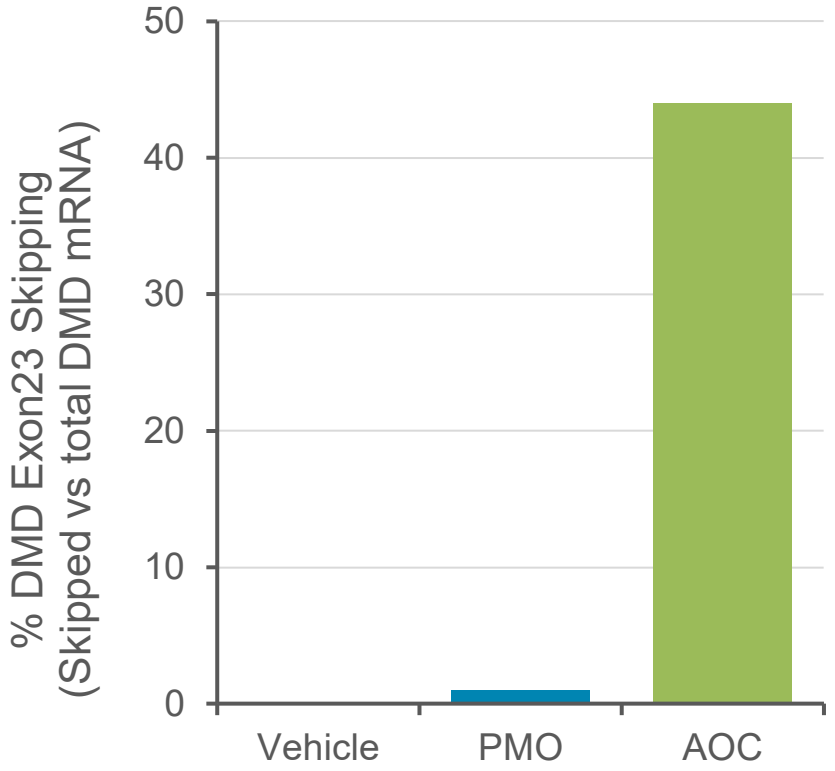


Exons 44, 51 and 45 represent ~30% of the total mutations observed amenable to skipping

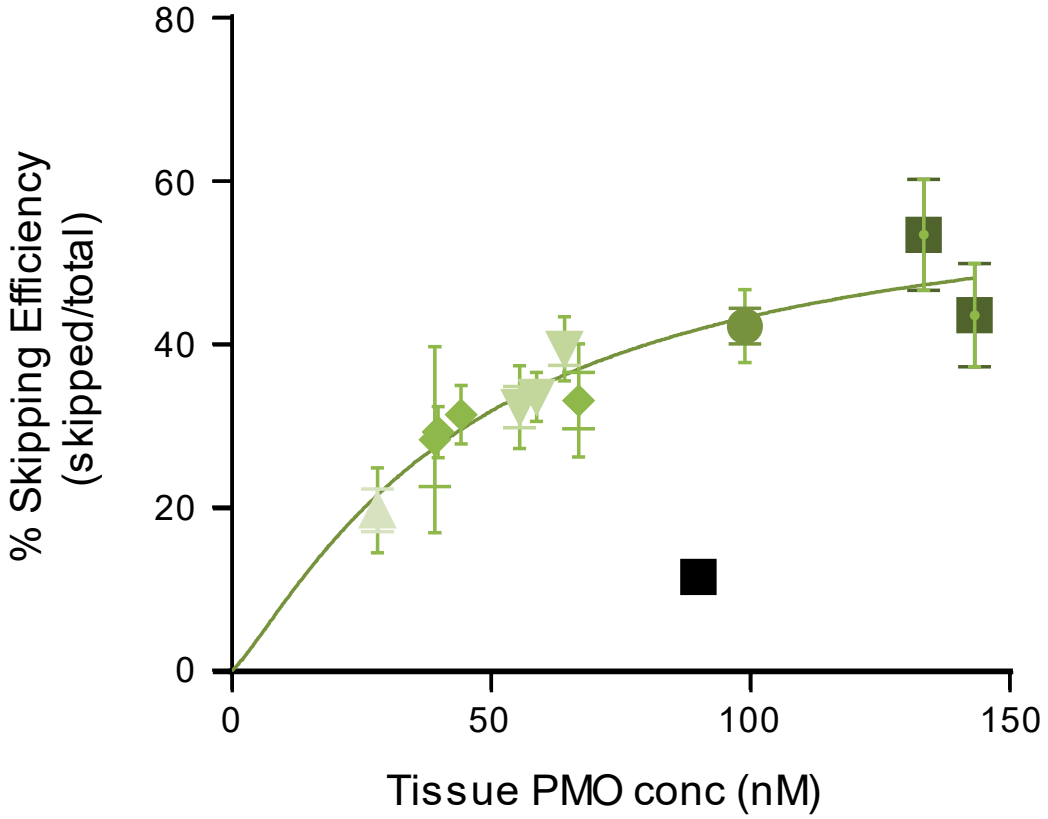
AVIDITY BIOSCIENCES

AOC Treatment Increased Exon Skipping > 50-fold Compared to an Unconjugated Oligo in DMD Model

- mdx mouse model of DMD
- Single injection of 8 mg/kg of phosphorodiamidate morpholino oligomer (PMO)
  - Vehicle = PBS
  - PMO = Dystrophin (DMD) exon 23 skipping PMO
  - AOC = TfR1 mAb + DMD exon 23 skipping PMO
- Exon skipping was measured 14 days post dose



Exon Skipping with a TfR1 mAb AOC or Control mAb AOC



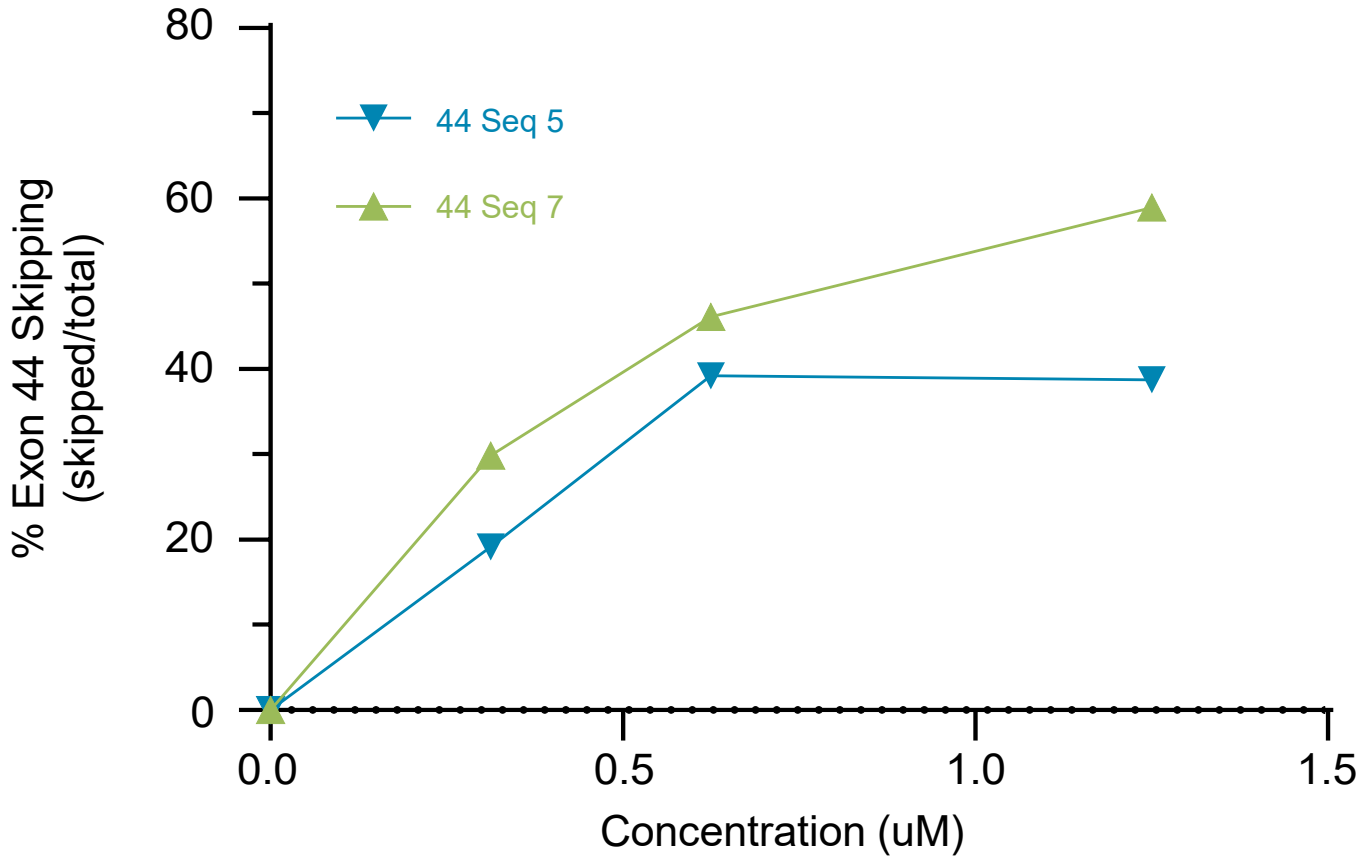
TfR1.mAb AOC 1.9 mg/kg
TfR1.mAb AOC 3.3 mg/kg
TfR1.mAb AOC 5.7 mg/kg
TfR1.mAb AOC 8.5 mg/kg
TfR1.mAb AOC 10 mg/kg
Control.mAb AOC 10 mg/kg

Data provides us with a framework for establishing target concentration in clinical trials

The PMO conjugated to a non-targeting mAb (black square) allowed for uptake into muscle, but skipping was substantially less compared to the TfR1 targeted AOC

Dystrophin Exon 44-Skipping Lead Candidates

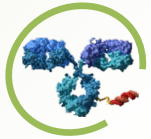
Substantial skipping of Exon 44 of dystrophin in human primary myocytes when incubated with two lead development candidates



AVIDITY BIOSCIENCES

DMD Program Summary

- ✓ Skipping of exons in relevant disease models
- ✓ Long duration of action
- ✓ Specificity of the target exon
- ✓ Effective delivery of PMO
- ✓ Well-established and scalable methods for manufacturing
- ✓ Potential for DMD mutations beyond exons 44, 51 and 45



DELIVERING ON DMD

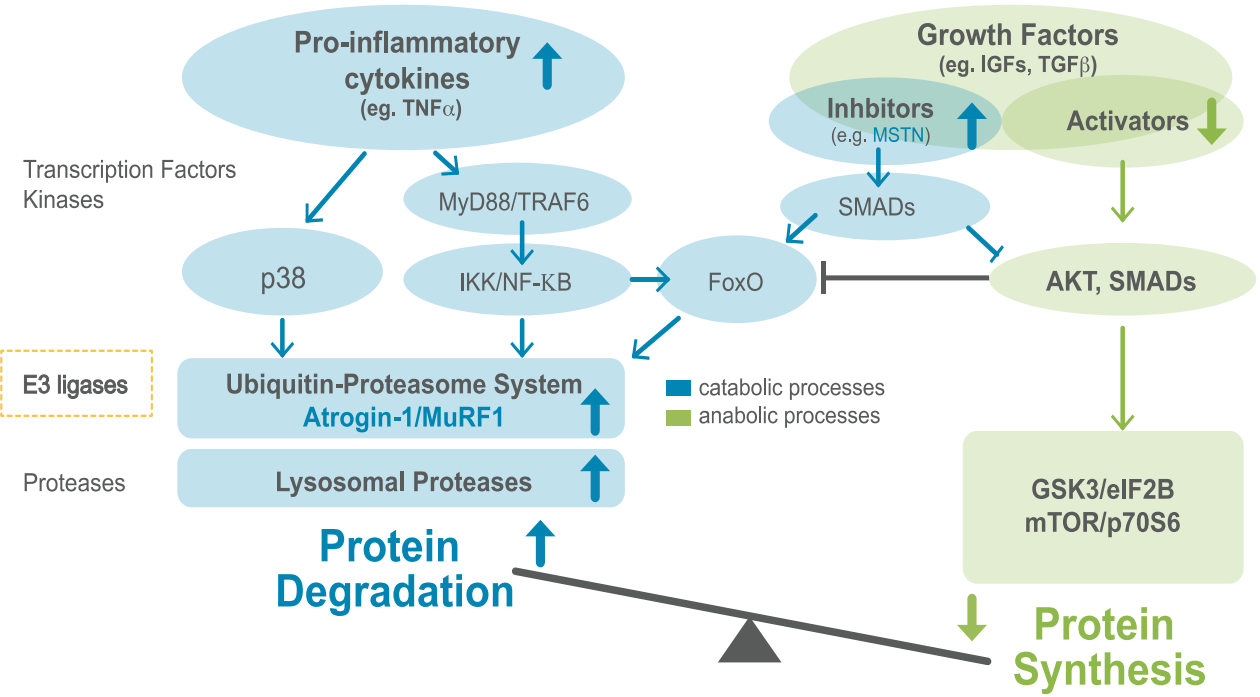
THREE PROGRAMS ADVANCING TOWARD THE CLINIC

Planned regulatory filing to support initiation of a clinical trial for the exon 44 program in **2022**

AVIDITY BIOSCIENCES

Muscle Atrophy Program

Muscle Atrophy Overview and Opportunity for AOCs



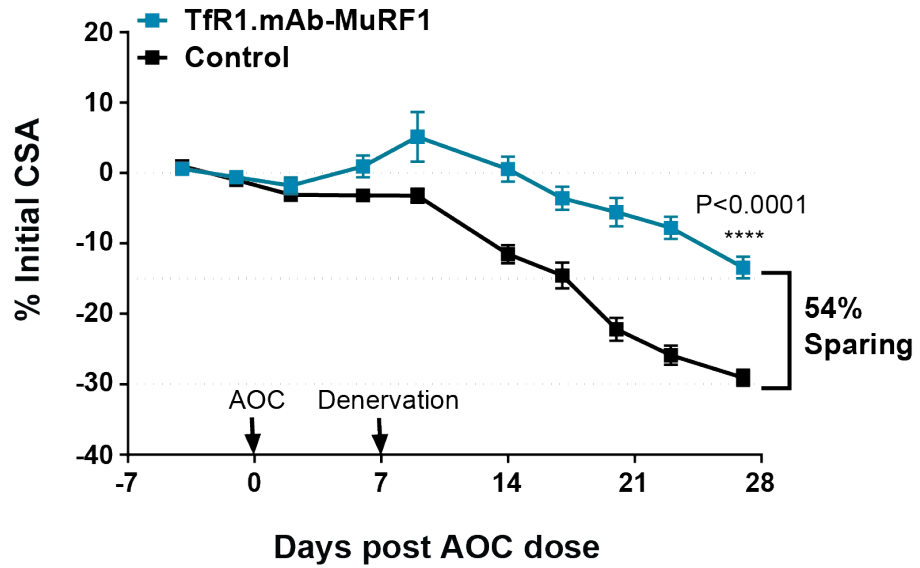
Muscle atrophy is caused by a change in the balance between catabolic and anabolic signals

By targeting MuRF1, our AOC is designed to employ both the catabolic and anabolic pathways associated with the degradation of protein in muscle cells

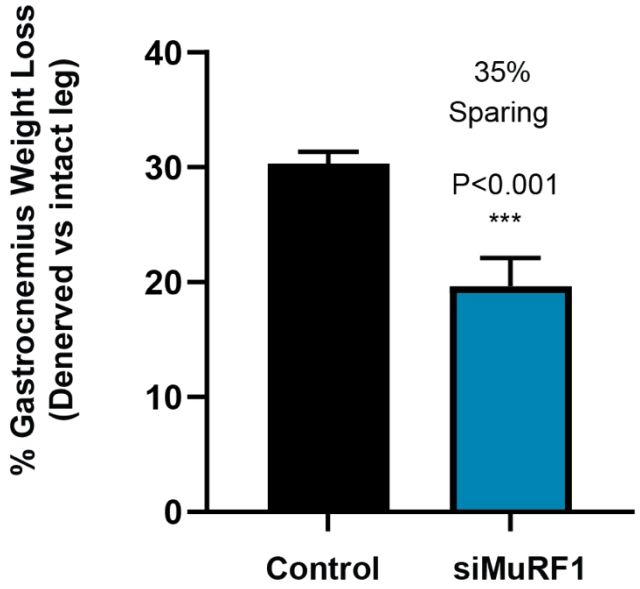
AVIDITY BIOSCIENCES

Impact of Downregulation of MuRF1 on Denervation-Induced Muscle Atrophy in Mice

Lower Leg Cross Sectional Area (CSA)

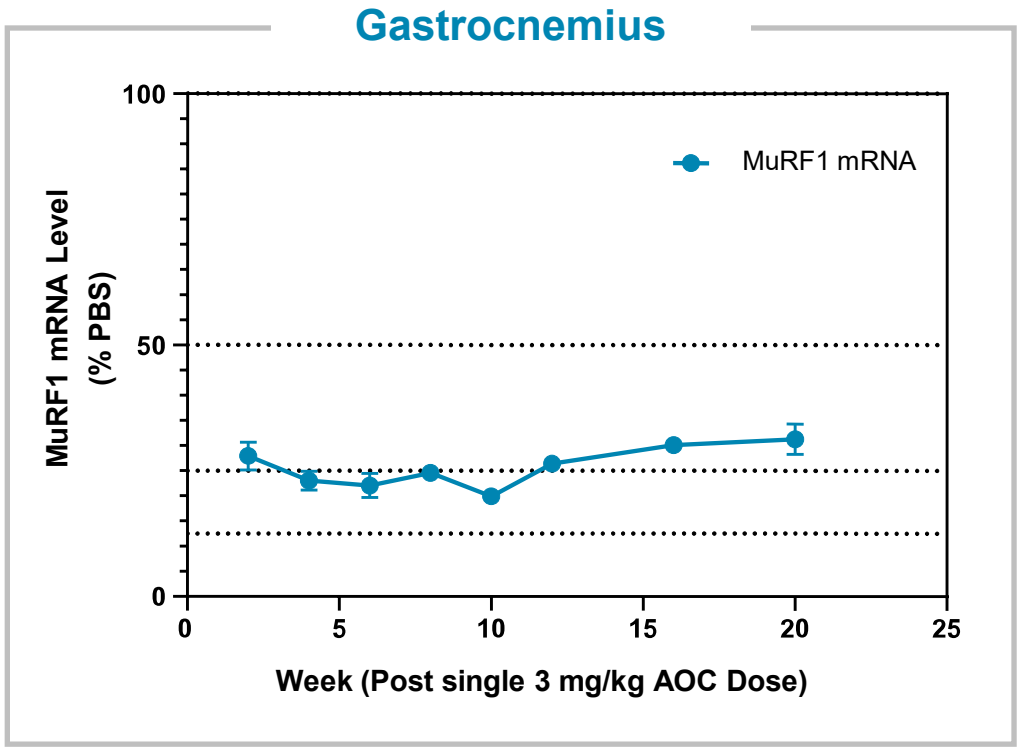


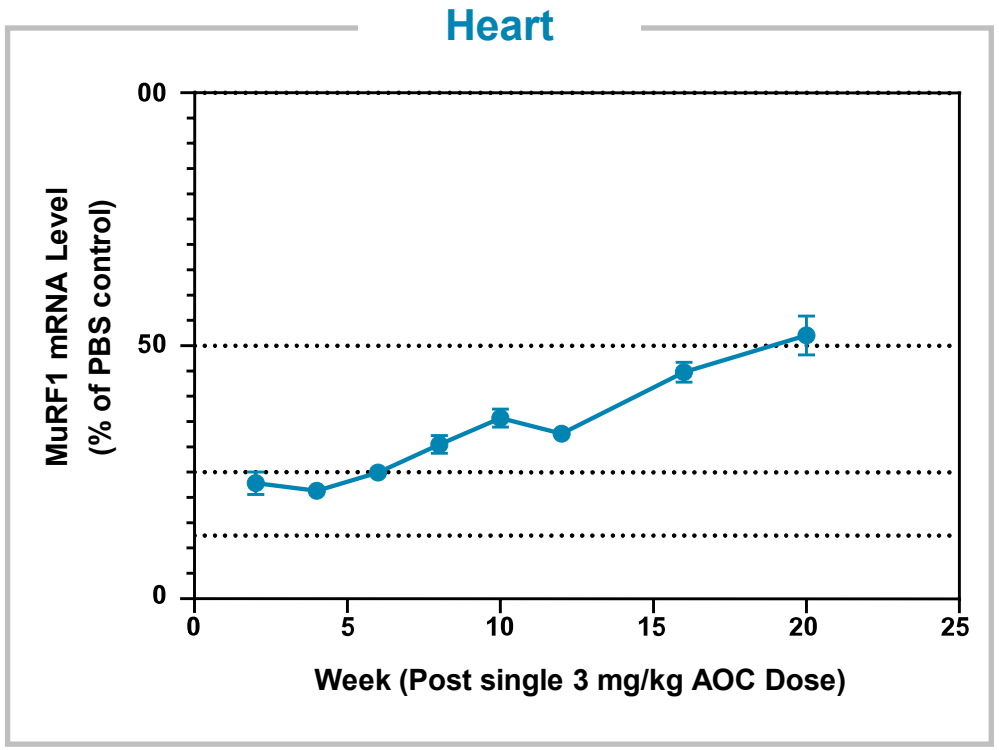
Gastrocnemius Weight



- The muscle sparing activity of AOC-mediated MuRF1 downregulation mimics the muscle-sparing activity observed upon genetic ablation of MuRF1
- Evaluation of MuRF1 AOCs in other models of muscle atrophy is in progress

A Single 3 mg/kg Dose Produced >50% Reduction of MuRF1 in Skeletal Muscle in Mice for More Than 20 Weeks



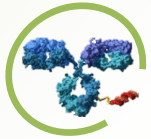


Data are expressed as mean (SE) compared to saline controls

AVIDITY BIOSCIENCES

Muscle Atrophy Preclinical Profile

- ✓ Improvements in disease model of muscle atrophy
- ✓ Most potent oligonucleotide type
- ✓ Long duration of action
- ✓ Specificity of the target mRNA
- ✓ Effective delivery of siRNA
- ✓ Well-established and scalable methods for manufacturing



DELIVERING ON MUSCLE ATROPHY

PRECLINICAL STUDIES ONGOING

Planned regulatory filing to support initiation of a clinical trial in **2022**

AVIDITY BIOSCIENCES

Pompe Disease Program

Pompe Disease: Disease Overview

~1 in 40,000
INDIVIDUALS IN THE US

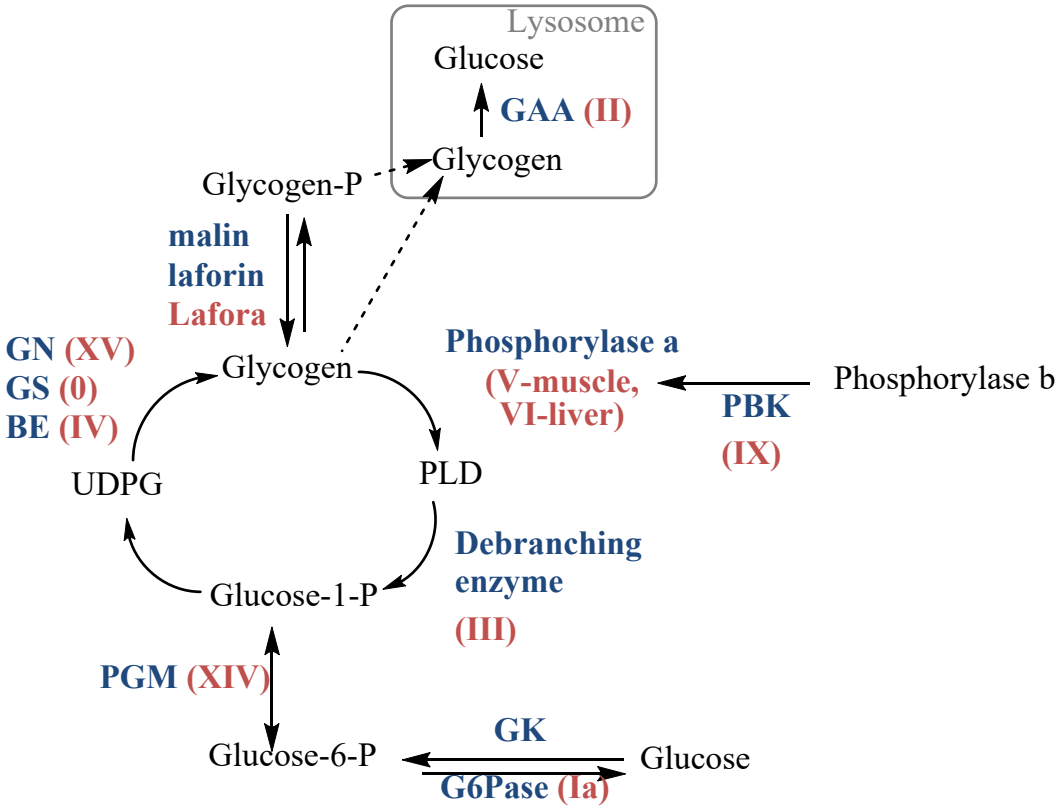
AFFECTS
~5,000 - 10,000
PEOPLE WORLDWIDE

HIGH UNMET NEED
DESPITE ONE APPROVED THERAPY
(Lumizyme, enzyme replacement therapy or ERT)

- Pompe is a rare, autosomal recessive lysosomal storage disease
- Caused by a mutation in the gene that encodes glucosidase alpha acid (GAA) that results in the buildup of glycogen in the body's cells
- The accumulation of glycogen in certain organs and tissues, especially muscles, impairs normal tissue and organ function
- ERT does not adequately address the breakdown of muscle tissue associated with the disease

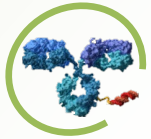
AOCs Targeting GYS1 May Overcome Limitations of Existing Therapies for Pompe

- Glycogen synthase 1 (GYS1) is predominantly expressed in muscle tissue where it serves as the main enzyme for glycogen synthesis
- GYS1 knockdown will inhibit glycogen production in muscle tissue and reduce glycogen accumulation in lysosomes
- We believe GYS1 targeted substrate reduction therapy will be complementary to:
  - Existing enzyme replacement therapy and enzyme enhancement therapy
  - Future gene therapies



Pompe Program Summary

- ✓ GYS1 specific AOCs identified
- ✓ Significant unmet need despite existing therapy
- ✓ GYS1 knockdown will inhibit toxic glycogen production in muscle tissue
- ✓ AOCs have the potential to improve the quality and life expectancy for Pompe patients



DELIVERING ON POMPE

PRECLINICAL STUDIES ONGOING

Immunology and Other Areas

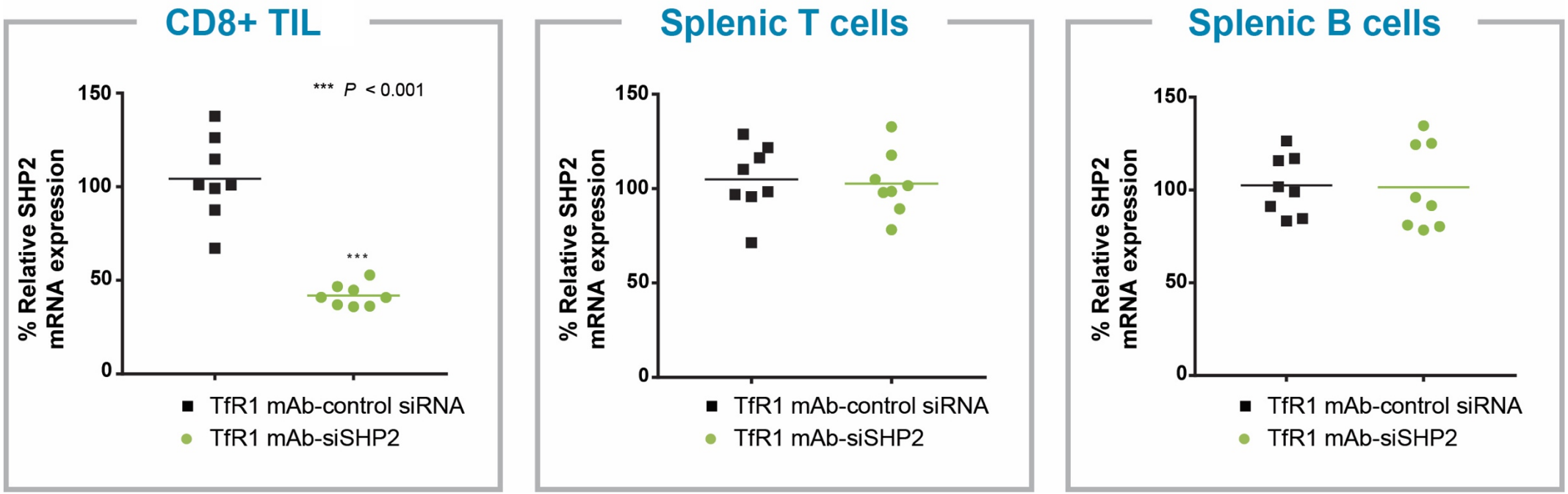
AOCs Have Multiple Applications in Immunology

- *In vitro* receptor-antibody pairs were identified for siRNA delivery to
  - $CD8^+$, $CD4^+$ tumor infiltrating T cells
  - Activated macrophages
- *In vivo* AOC POCs were established in
  - Transiently activated splenic T and B cells
  - Tumor infiltrating $CD8^+$ and $CD4^+$ T cells
  - Peritoneal macrophages MRL/lpr mice

AOCs Modulate Tregs, Which may be an Effective Approach to Treat Cancer

- Tregs are a type of CD4+ T cells that suppress the potentially adverse effect of the immune system, prevent autoimmune disease, and maintain self-tolerance
  - Tumor Tregs correlate with poor prognosis and resistance to anti-PD-L1 antibody therapy
- AOCs have shown TIL selectivity over peripheral T and B cells

Localized Activity of AOCs in Cells Involved in Immune-Responses to Tumors Without Affecting Lymphocytes Outside the Tumor



- Syngeneic CT-26 tumor model with an intravenous dose of 6 mg/kg
- Significant AOC-mediated mRNA reduction in CD4+ TIL and T-regs from mice treated with an AOC comprised of a mAb targeting TfR1 and an siRNA targeting SHP2
- AOCs had no effect on the mRNA levels in T or B cells in the spleen

Building for the Future

Experienced Leadership with Significant RNA Therapeutic Expertise

Management Team

Sarah Boyce *President & CEO*	**Art Levin, PhD** *CSO*	**Jae Kim, MD, FACC** *CMO*	**W. Michael Flanagan, PhD** *CTO*
Michael MacLean *CFO*	**John Wallen III, PhD, JD** *General Counsel*	**Joseph Baroldi** *COO*	**Teresa McCarthy** *CHRO*

AKCEA THERAPEUTICS · ALEXION · Alnylam PHARMACEUTICALS · AMGEN · Biogen · Genentech A Member of the Roche Group · IONIS · janssen · miRagen · MYOKARDIA · NOVARTIS · Roche · santaris pharma a/s RNA Medicines for the 21st Century

Board of Directors

Troy Wilson, PhD, JD *Chairman & Avidity Founder, CEO of Kura Oncology*	**Carsten Boess** *Board Member*	**Edward Kaye, MD** *CEO & Director, Stoke Therapeutics*	**Jean Kim** *Board Member*
Noreen Henig, MD *Chief Medical Officer, Kezar Life Sciences*	**Roderick Wong, MD** *Managing Partner, RTW Investments*	**Tamar Thompson** *VP, Govt. Affairs and Policy, Alexion Pharmaceuticals*	**Sarah Boyce** *President & CEO, Avidity*

Partnering Strategy to Accelerate and Expand the Utility of AOCs Outside of Rare Diseases

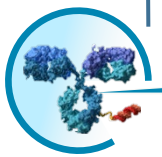
IMMUNOLOGY

Lilly

Collaboration focused on immunology and other select indications, but not muscle diseases

$405M

Potential milestone payments per target, plus mid-single to double-digit tiered royalties



CARDIAC MUSCLE

MYOKARDIA

Research collaboration focused on cardiac disease

NEW TISSUES

Plan to add additional leading partners to accelerate and expand the utility of AOCs in tissues and cell types beyond skeletal muscle

Q1 2021 – Financial Results

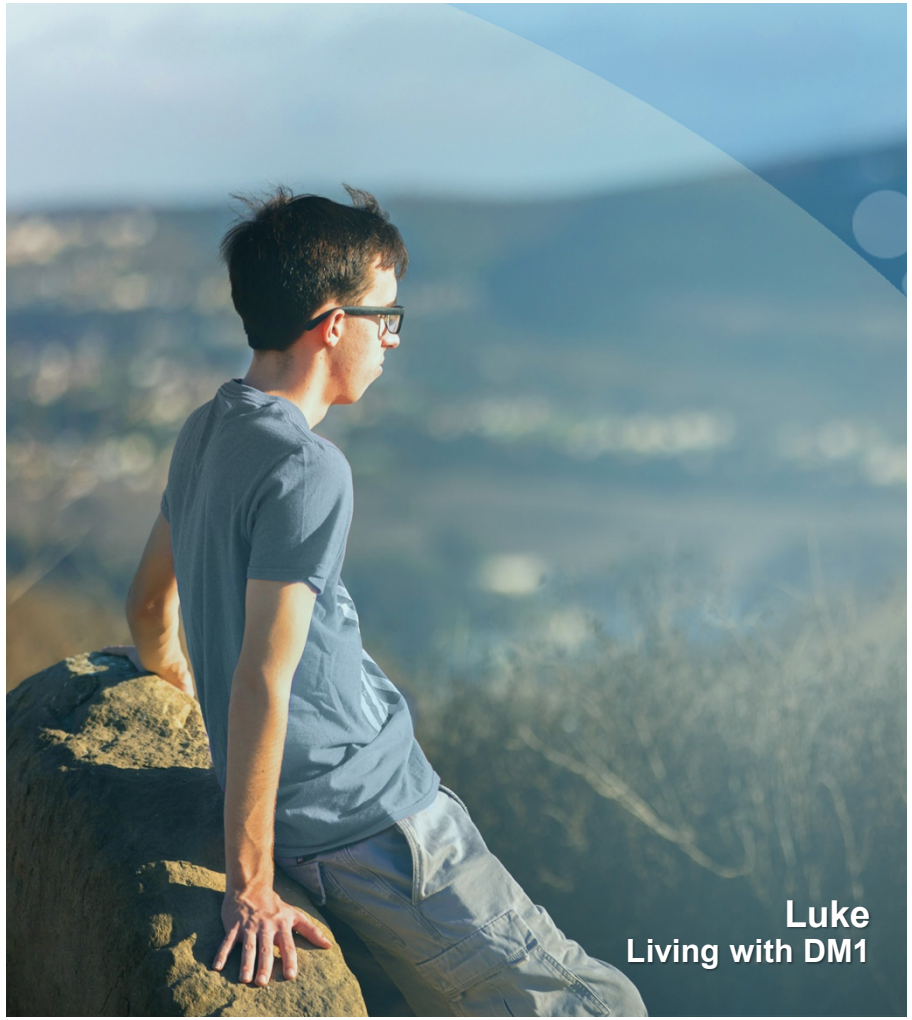
In millions (unaudited)	Q121	Q420	Q120	Q121 vs Q420	Q121 vs Q120
Collaboration Revenue	$2.7	$2.1	$1.3	$0.6	$1.4
R&D expenses	20.6	13.6	5.5	7.0	15.1
G&A expenses	5.9	4.8	2.0	1.1	3.9
Total operating expenses	26.5	18.4	7.5	8.1	19.0
Loss from operations	(23.8)	(16.3)	(6.2)	(7.5)	(17.6)
Other income (expense)	0.0	0.0	0.1	0.0	(0.1)
Net loss	($23.8)	($16.3)	($6.1)	($7.5)	($17.7)

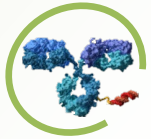
In millions (unaudited)	Q121	Q420
Cash, cash equivalents and marketable securities	$307.9	$328.1

Strong cash position as we move toward the clinic with AOC 1001

AVIDITY BIOSCIENCES

Delivering on the RNA Revolution





DELIVERING NEXT

AOC 1001 into the clinic in H2 2021

Progress FSHD & DMD programs

Preclinical proof of concept in additional skeletal muscle and other tissues