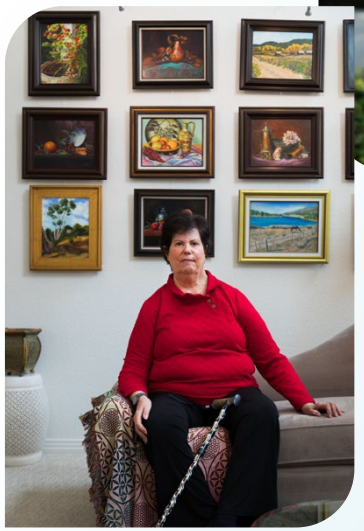
AVIDITY® BIOSCIENCES

Delivering on the RNA Revolution

March 2022

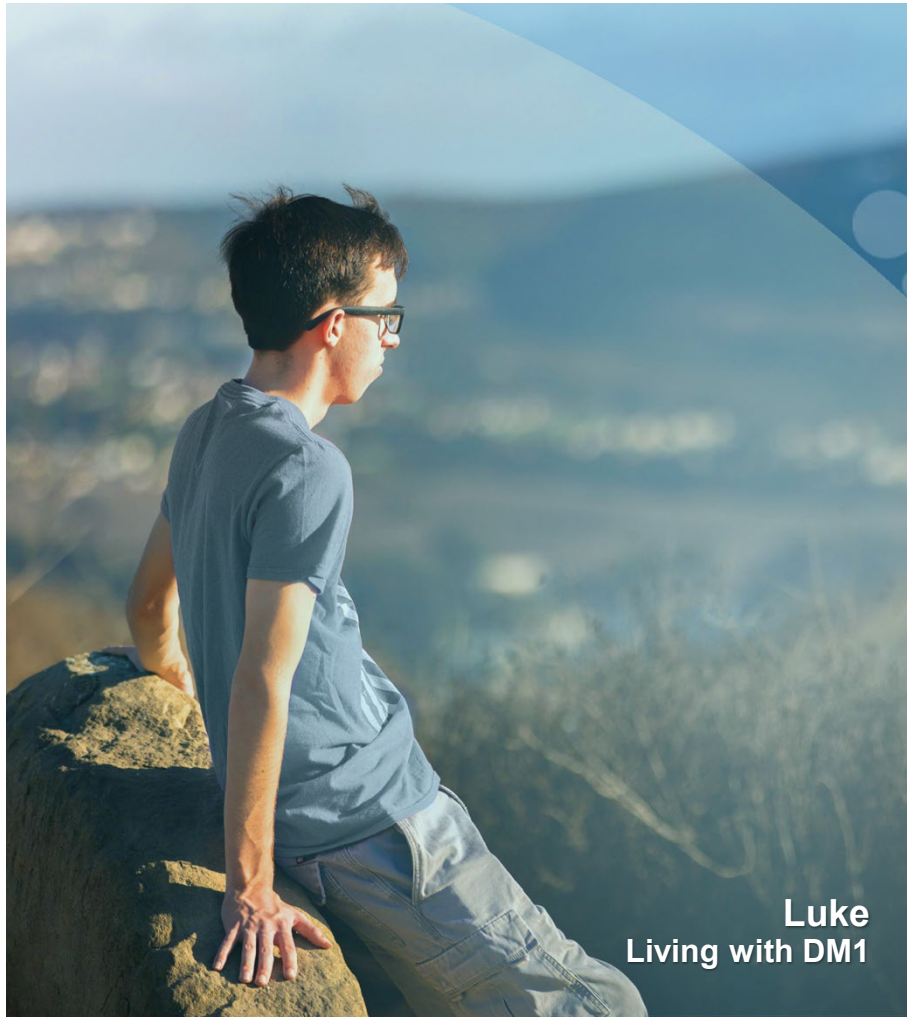


Forward Looking Statements

We caution the reader that this presentation contains forward-looking statements that involve substantial risks and uncertainties. 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 many 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 submissions 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 10-K for the year ending on December 31, 2021, filed with the SEC on March 1, 2022, 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.

Our Vision



To profoundly improve people's lives by revolutionizing the delivery of RNA therapeutics

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Delivering on Our Vision

DISRUPTIVE & BROAD
PLATFORM

- Committed to delivering a new class of RNA therapies
- Dosed first person with an AOC – a first for the platform
- Broadening to other tissues & cell types through partnerships & internal discovery

ADVANCING & EXPANDING
PIPELINE

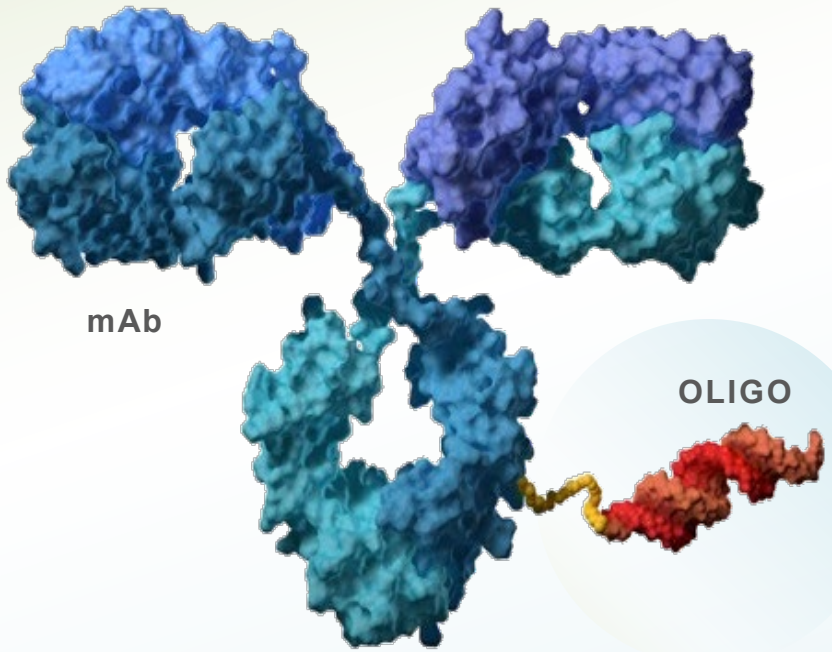
- Progressing robust pipeline in muscle
- Phase 1/2 MARINA™ trial of AOC 1001 ongoing
- Anticipated clinical trial initiations for both AOC 1044 for DMD and AOC 1020 for FSHD by the end of 2022

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

AOCs - A Powerful Potential New Class of Drugs

Utilizing decades of proven science in an effort to deliver the power of oligonucleotides



ANTIBODY OLIGONUCLEOTIDE CONJUGATE (AOC)

- Designed to combine the proven and safe technologies of approved monoclonal antibodies and oligonucleotides
  - ✓ Specificity of targeting with mAbs
  - ✓ Potency & precision of oligonucleotides
  - ✓ Targets tissues with durable agents
- Designed 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

Advancing our Muscle Disease Franchise of AOCs

PROGRAM / INDICATION	TARGET	DISCOVERY / LEAD OPTIMIZATION	IND ENABLING	PHASE 1/2
MUSCLE DISORDERS				
AOC 1001: Myotonic Dystrophy Type 1 (DM1)	DMPK			
AOC 1020: Facioscapulohumeral Muscular Dystrophy (FSHD)	DUX4		Clinical trial initiations planned for 2022	
AOC 1044: Duchenne Muscular Dystrophy (DMD)	Exon 44 Dystrophin		Clinical trial initiations planned for 2022	
Next AOC DMD Programs	Exon 51 Dystrophin			
	Exon 45 Dystrophin			
AOC Muscle Atrophy: Muscle Atrophy*	MuRF1			
AOC Pompe Disease: Pompe Disease	GYS1			

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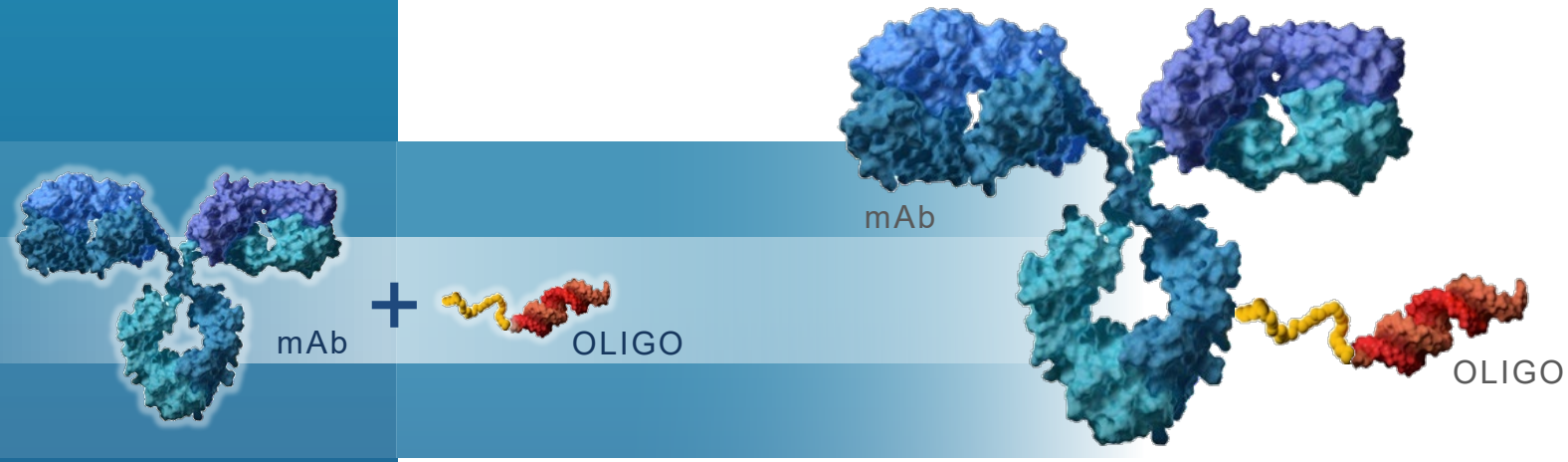
* Opportunity for a rare disease indication

Our AOC™ Platform

A Potential New Class of RNA Therapeutics

Engineering an AOC Therapeutic

Designed to combine the potency and precision of oligonucleotide therapies with the specificity of mAbs



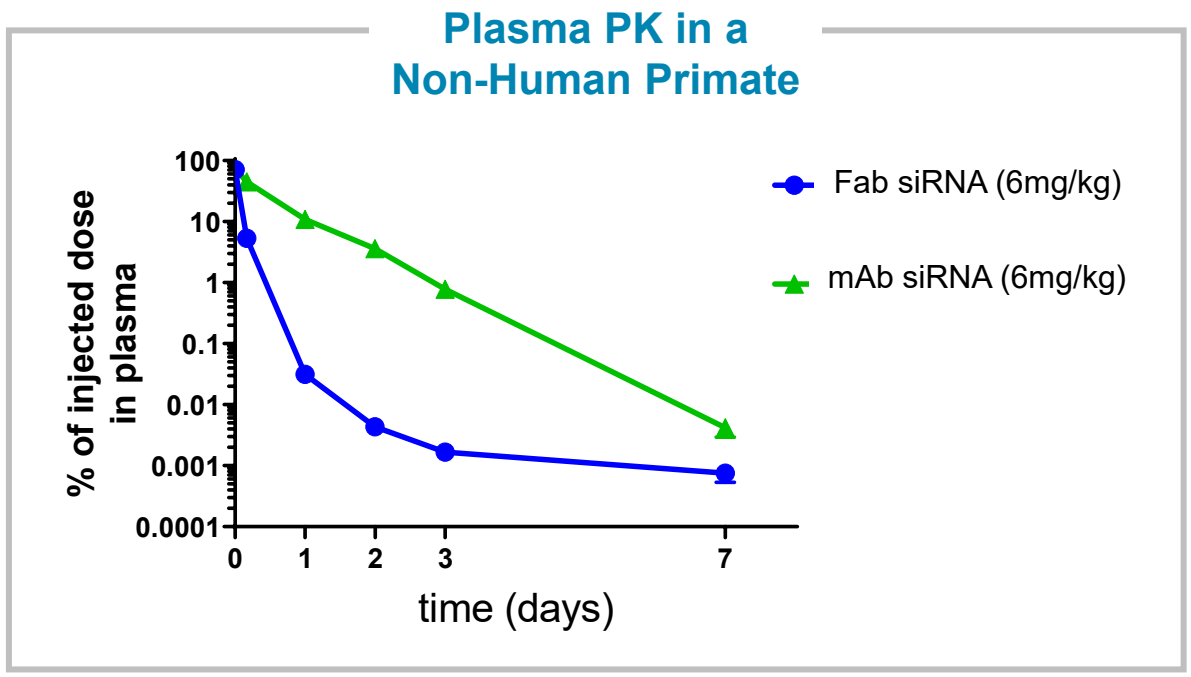
MONOCLONAL ANTIBODIES

OLIGONUCLEOTIDE THERAPIES

ANTIBODY OLIGONUCLEOTIDE CONJUGATE (AOC)

Following the Data: Choosing a mAb to Expand Delivery Beyond the Liver

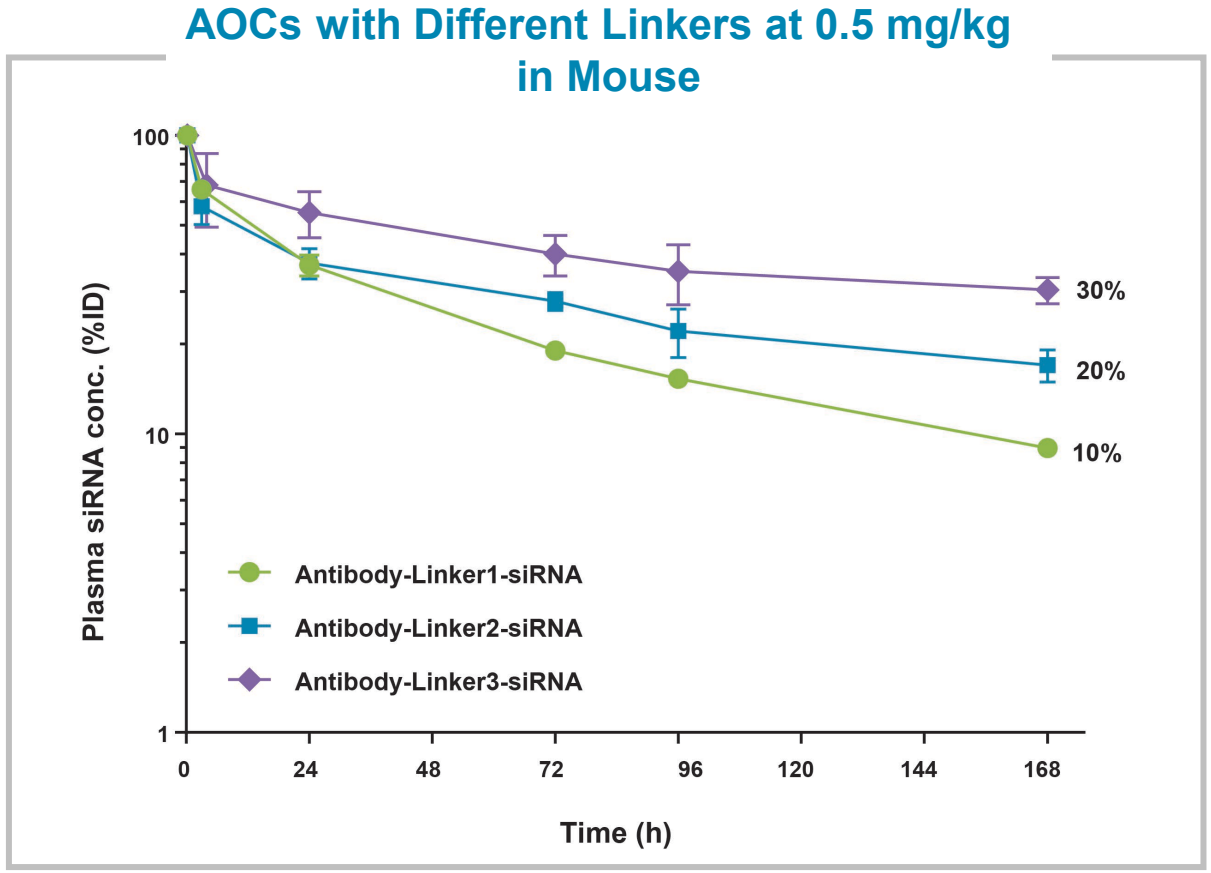
mAbs Offer Safe and Effective Targeting to Many Cells and Tissues



- To deliver oligonucleotide therapeutics, we followed the data to select the delivery moiety
  - Experiments showed that mAbs are superior
- Monoclonal antibodies (mAbs) are a proven technology that have been in use for 30 years
  - Chronic therapies with well-established safety profile
  - High specificity and affinity
  - Long half-life
- We optimized our mAbs through engineering to ensure:
  - Specific epitope binding to not compete with transferrin
  - Antibody is effector function null
  - Placement of the oligo itself on the antibody

Following the data: Engineering the Linker

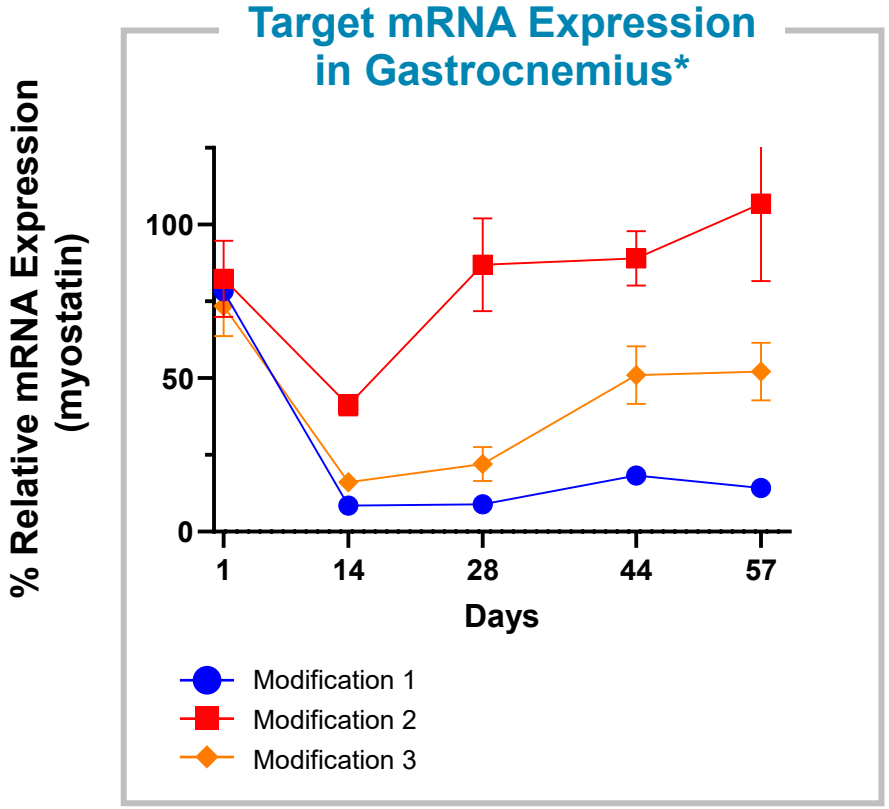
Our Linkers are Optimized for Stability and Durability



- In addition to engineering our linker, we optimize several other key aspects including:
  - sites of conjugation
  - the ratio of oligonucleotides to antibodies

Following the data: Choosing the Oligonucleotide

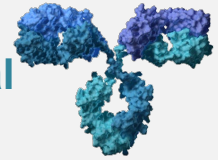
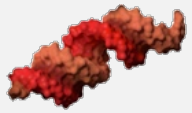
siRNA was a deliberate choice based on safety, potency and efficacy



**Target is myostatin, or MSTN in mouse*

- Selected siRNAs for activity and specificity and engineered them to withstand lysosomal enzymes
- siRNAs are well characterized
  - Attractive safety profile with no known thrombocytopenia, liver or renal toxicity
  - Potency in the nanomolar or picomolar range
  - Sustained activity in both the cytoplasm and the nucleus
  - Readily reproducible with many experienced manufacturers
- Leveraging this approach across the pipeline in different tissue and cell types

Following the Data: Each Component was Engineered in an Effort to Deliver the Optimal AOC for the Target

AOC COMPONENTS	DATA-DRIVEN COMPONENT CHARACTERISTICS	OUR ENGINEERING IMPACT
Monoclonal antibody 	Approved mAbs offer: • Well-established safety profiles • High specificity and affinity • Long half-lives	• Designed through engineering to be effector function null • Epitope selection designed for optimal activity
Linker 	• Known linker • Applicable to multiple oligo modalities	• Enhanced for durability • Engineered sites of conjugation • Optimized ratio of oligonucleotides to antibodies
siRNA 	Approved siRNA drugs have shown: • Attractive safety profiles - no known thrombocytopenia, liver or renal toxicity • Potency in the nanomolar range • Sustained activity in the cytoplasm and nucleus	• Engineered to withstand lysosomal enzymes • Selected and modified to diminish off-target effects

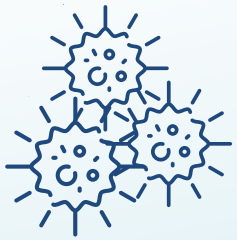
AOCs Engineered to Use Receptor Mediated Uptake for a Range of Tissue and Cell Types *In Vivo*

LIVER

SKELETAL MUSCLE

CARDIAC

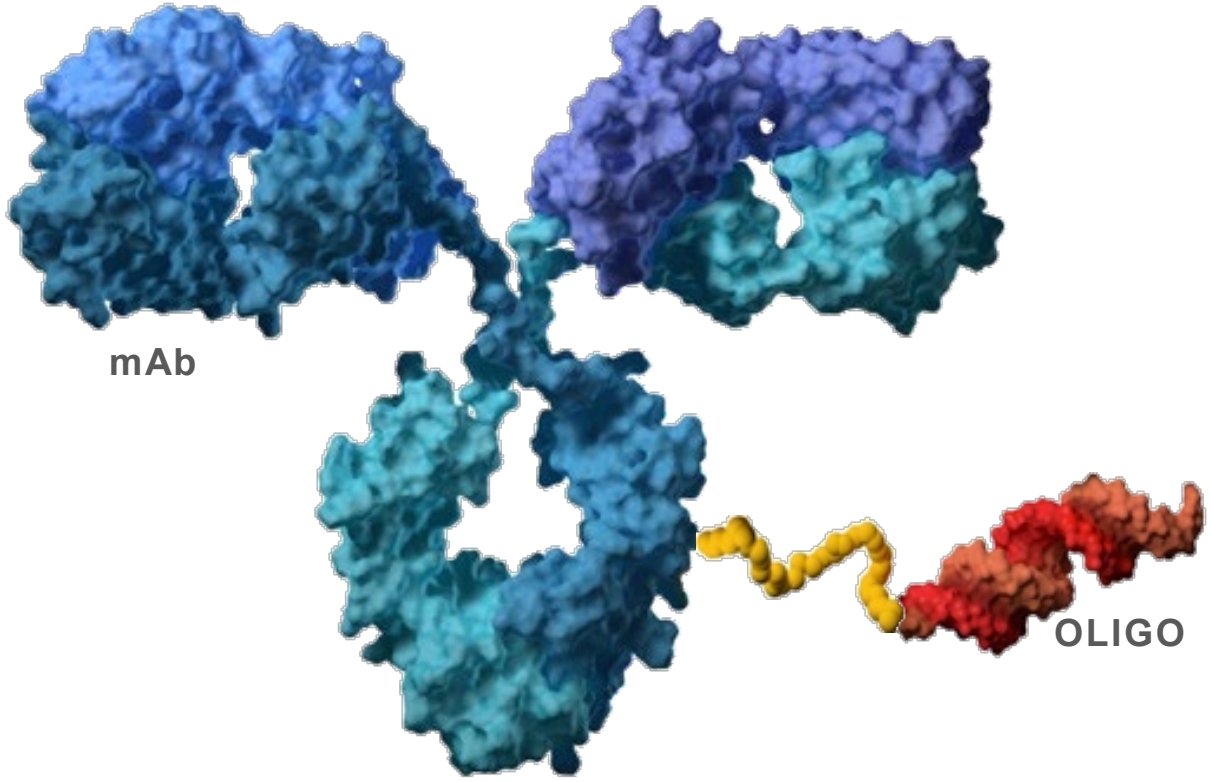
IMMUNOLOGY



IMMUNO-ONCOLOGY

Engineering AOCs:
Following the Data

- ✓ AOCs are designed to deliver RNA therapies with specificity, potency and precision
- ✓ AOCs are the result of years of in-house engineering
- ✓ AOCs are designed to exploit the well characterized safety and efficacy profiles of approved mAbs and siRNA drugs
- ✓ Potential for consistent delivery to muscle
- ✓ AOC 1001 – first AOC clinical program
- ✓ Advancing and expanding AOC pipeline – AOC 1044 (DMD Exon 44) and AOC 1020 (FSHD) in IND-Enabling Studies
- ✓ Focused today on rare disease with potential for much broader application



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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 (DM1): Disease Overview

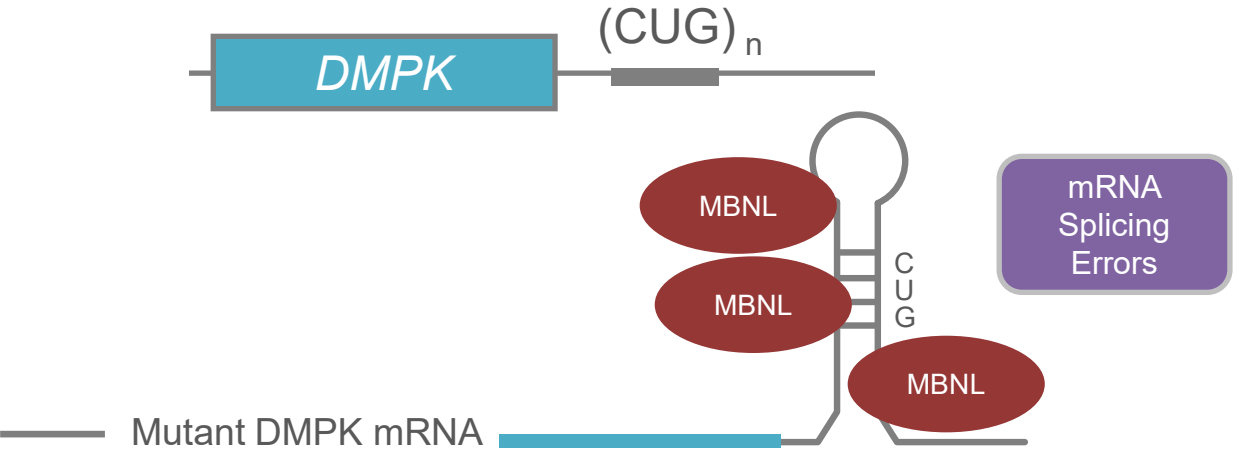
>40,000
PEOPLE WITH DM1 IN THE US

0
APPROVED THERAPIES

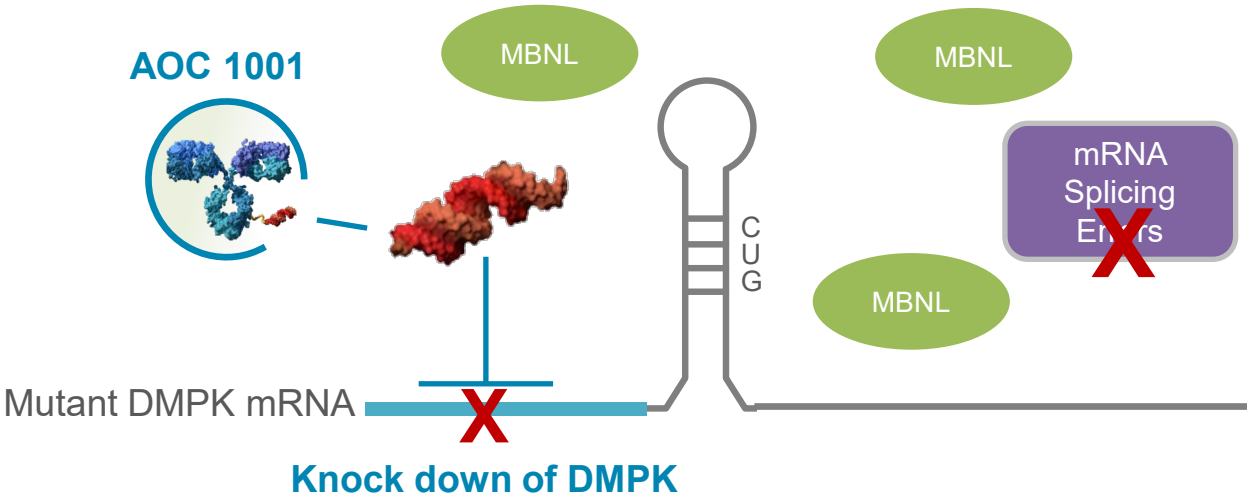
- DM1 is a complex disease with symptoms that present with high variability from patient to patient
- Monogenic, autosomal dominant, progressive disease that primarily affects muscle: skeletal, cardiac & smooth
- Increases in severity from generation to generation
- Significant impact on quality of life
- Shortened life-expectancy

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

MECHANISM OF DISEASE



THERAPEUTIC APPROACH

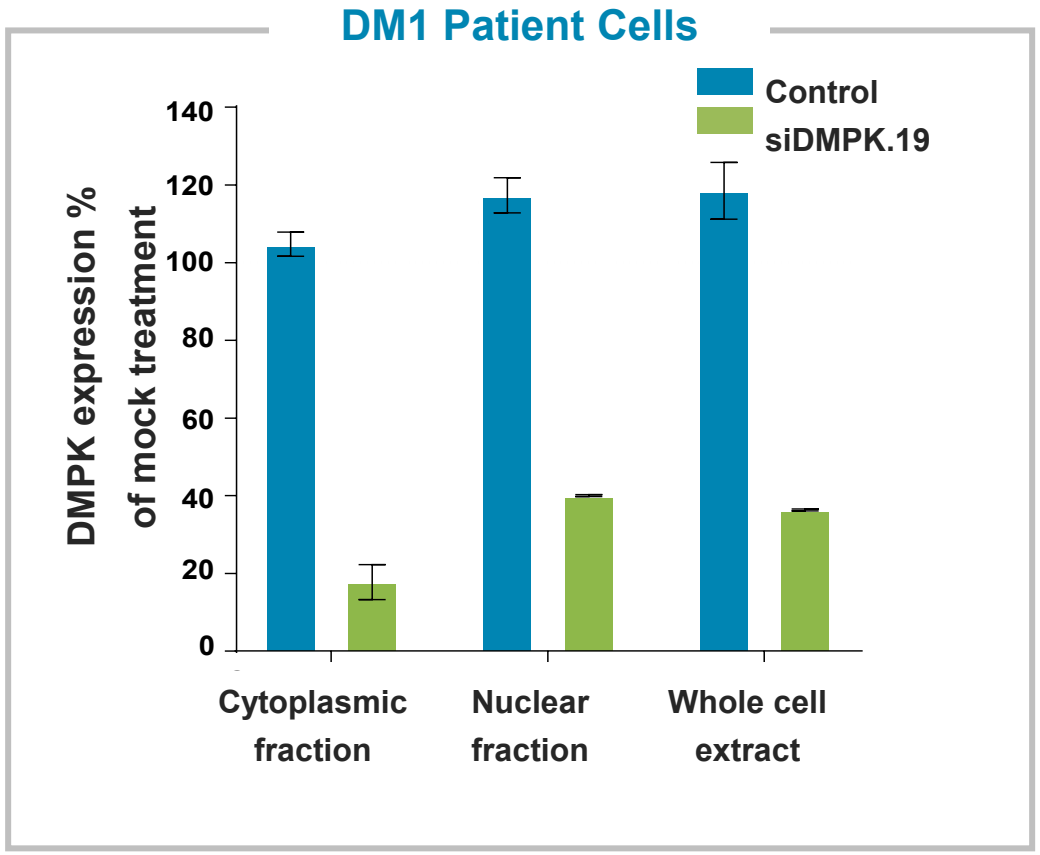


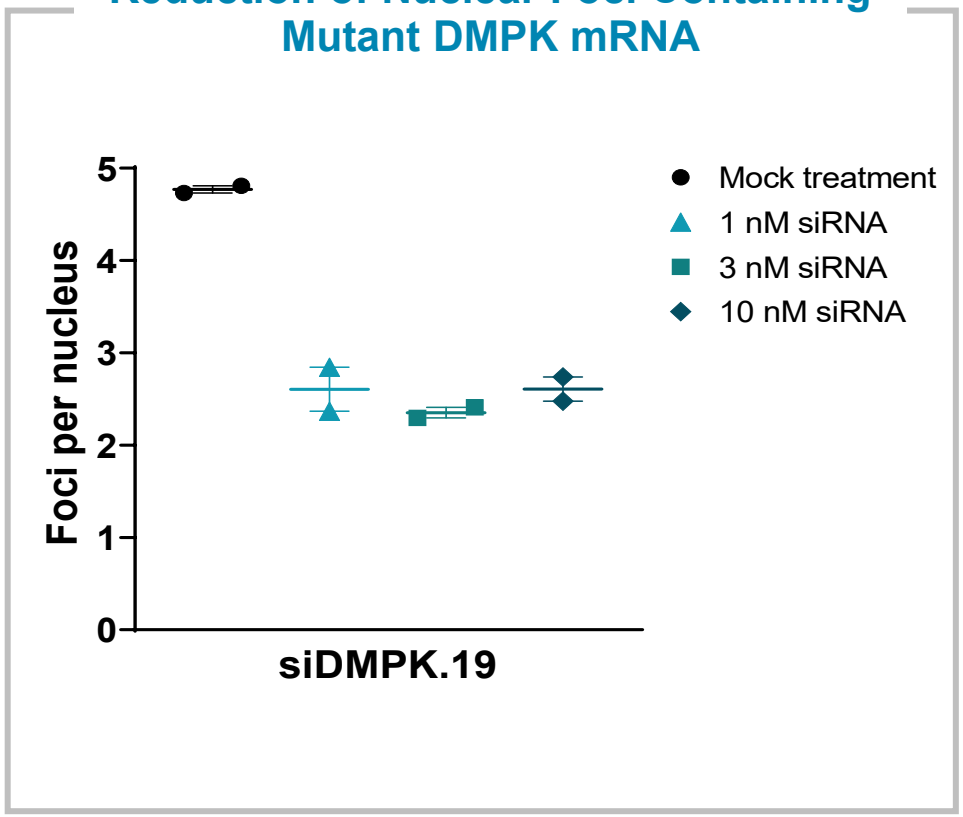
Knock down of DMPK

- Trinucleotide expansion in DMPK mRNA sequesters an RNA splicing protein MBNL (Muscleblind like) in nuclear foci.
- Sequestration of MBNL leads to RNA splicing errors in multiple muscle-related RNAs and induces DM1 disease manifestations.

- Allows MBNL to be released to perform its natural function to aid in splicing key mRNAs in muscle
- Improves the splice patterns and muscle function. Splice patterns can serve as biomarkers.

The AOC 1001 siRNA Showed Activity in the Nucleus and Cytoplasm and Reduced Nuclear Foci in Patient-Derived Muscle Cells





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

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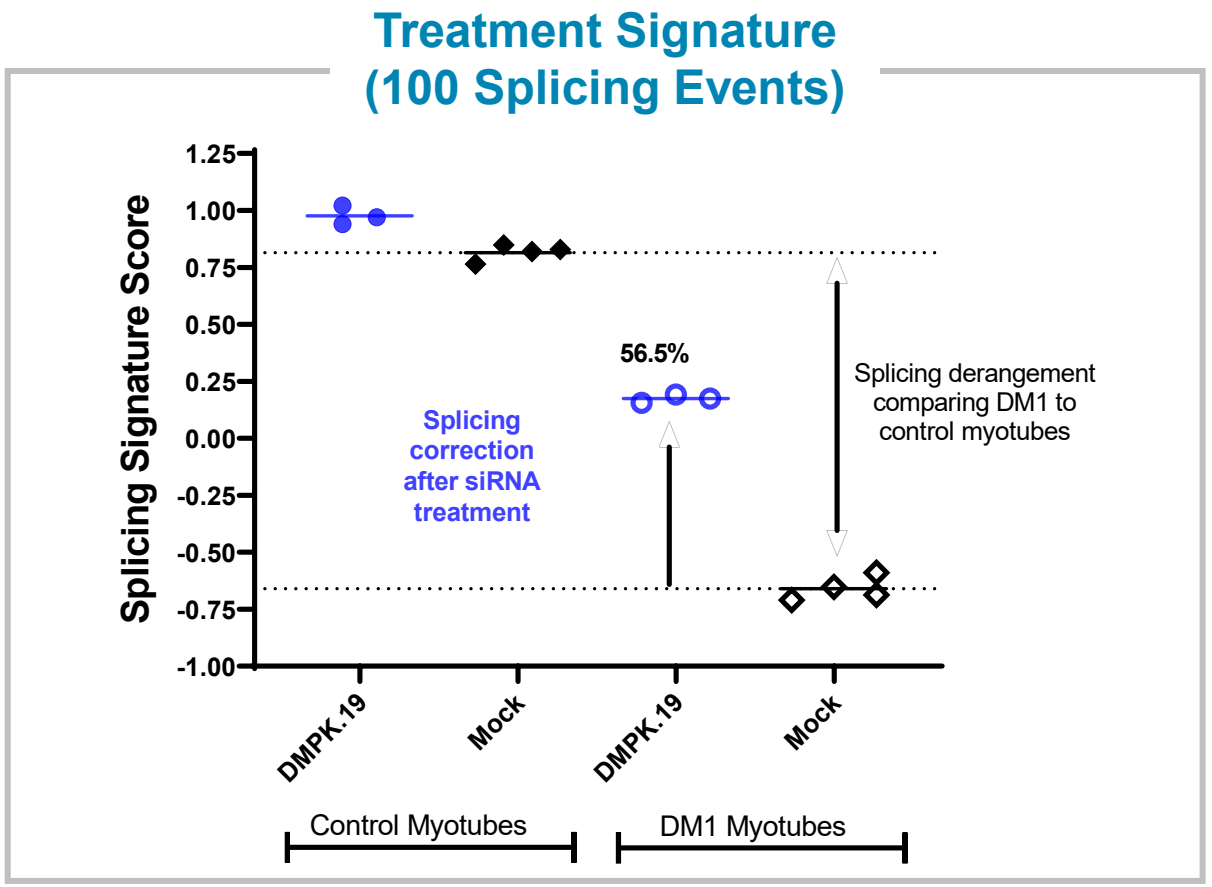
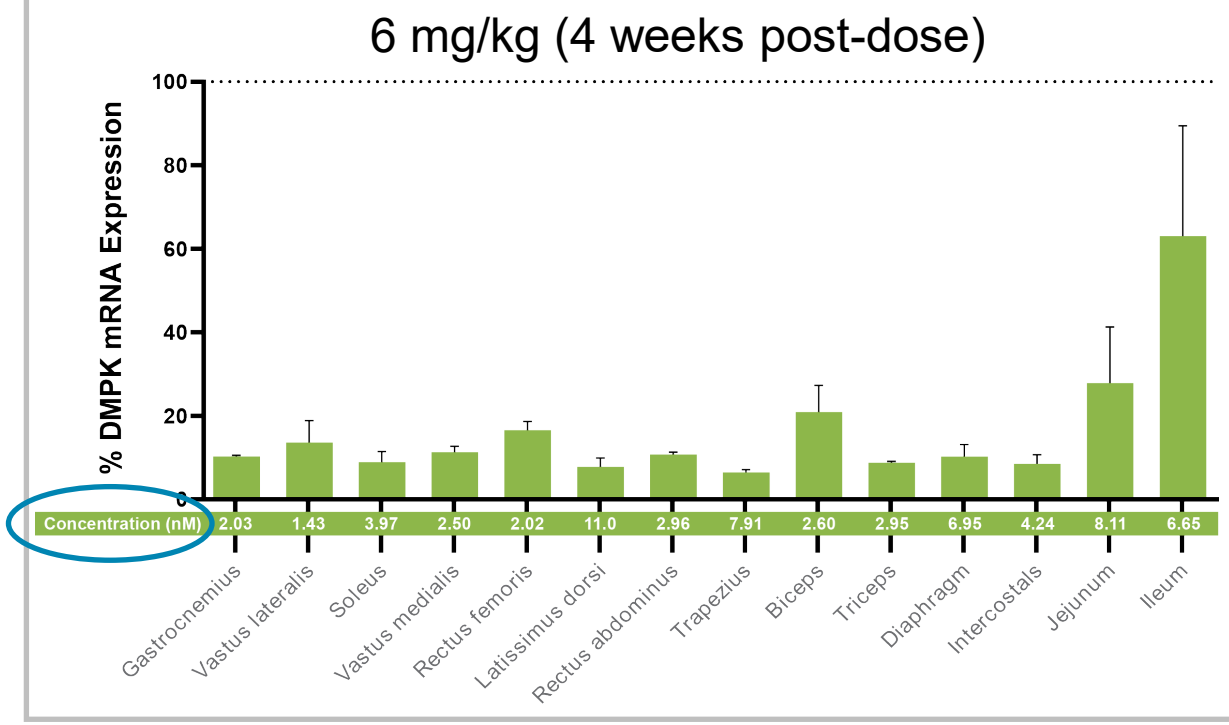
The AOC 1001 siRNA (siDMPK.19) Produced a 56% Improvement in Splicing in DM1 Myotubes

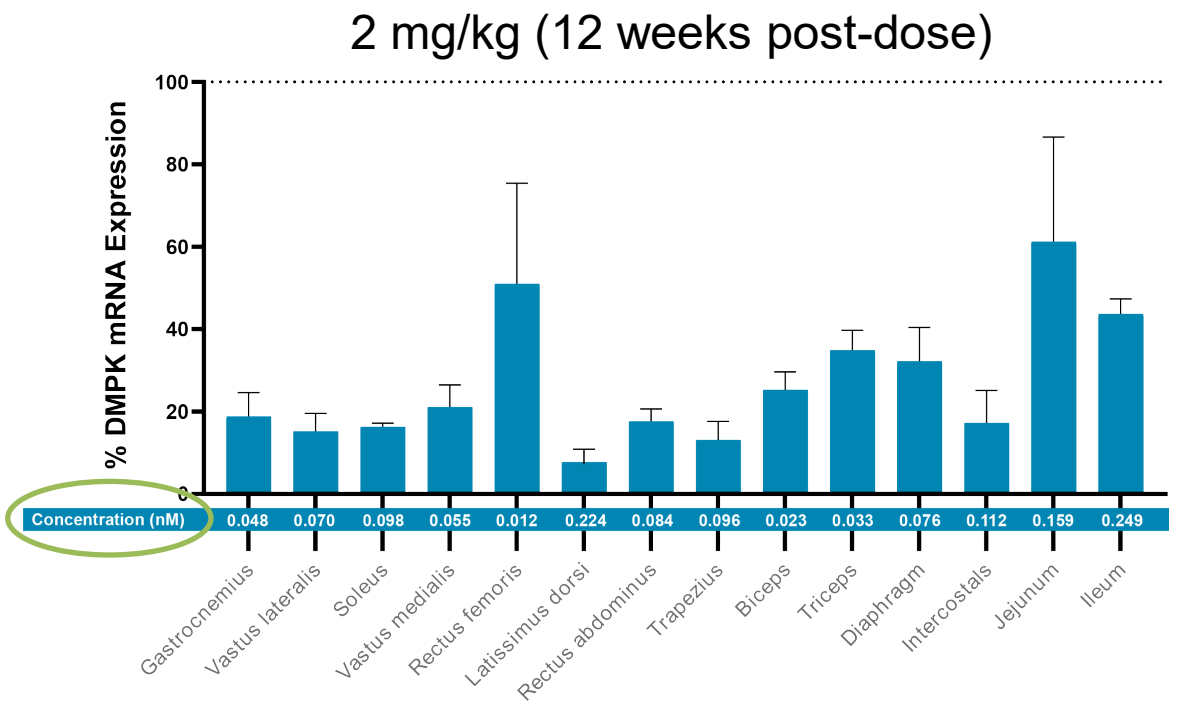


Durable ~75% Reduction of DMPK mRNA Observed in Monkey Skeletal Muscles After a Single Dose of 2mg/kg of siDMPK.19

The AOC 1001 siRNA Reduced DMPK mRNA in a Wide Range of Skeletal Muscle at Nanomolar Concentrations

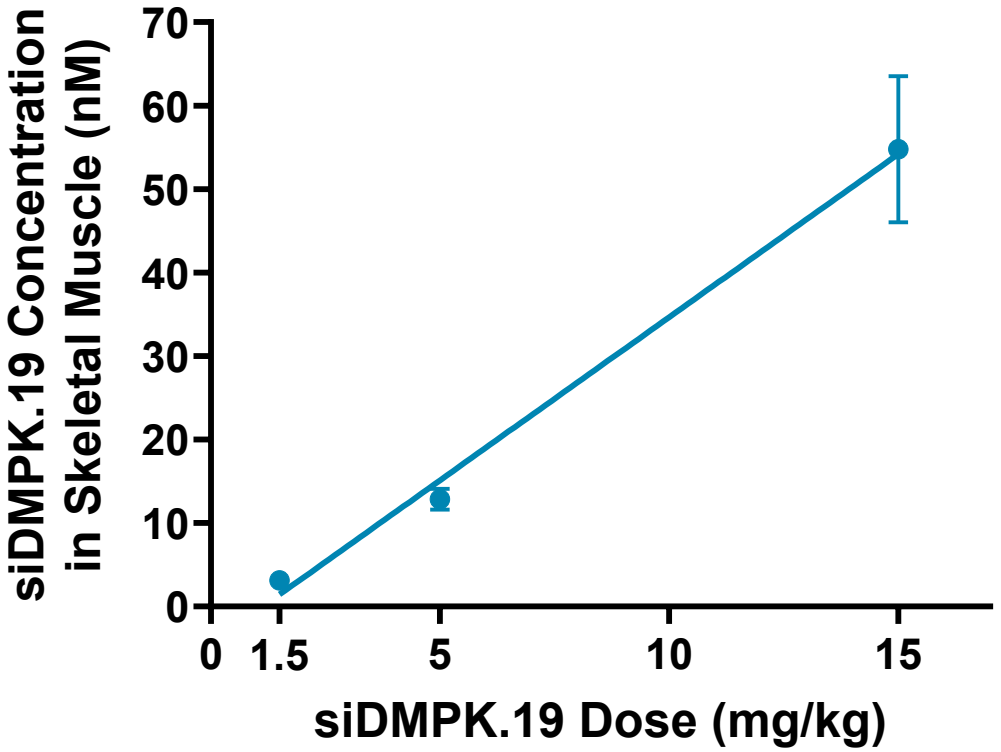
DMPK Expression in Skeletal Muscle and GI Tissue After a Single i.v. Dose in Monkey





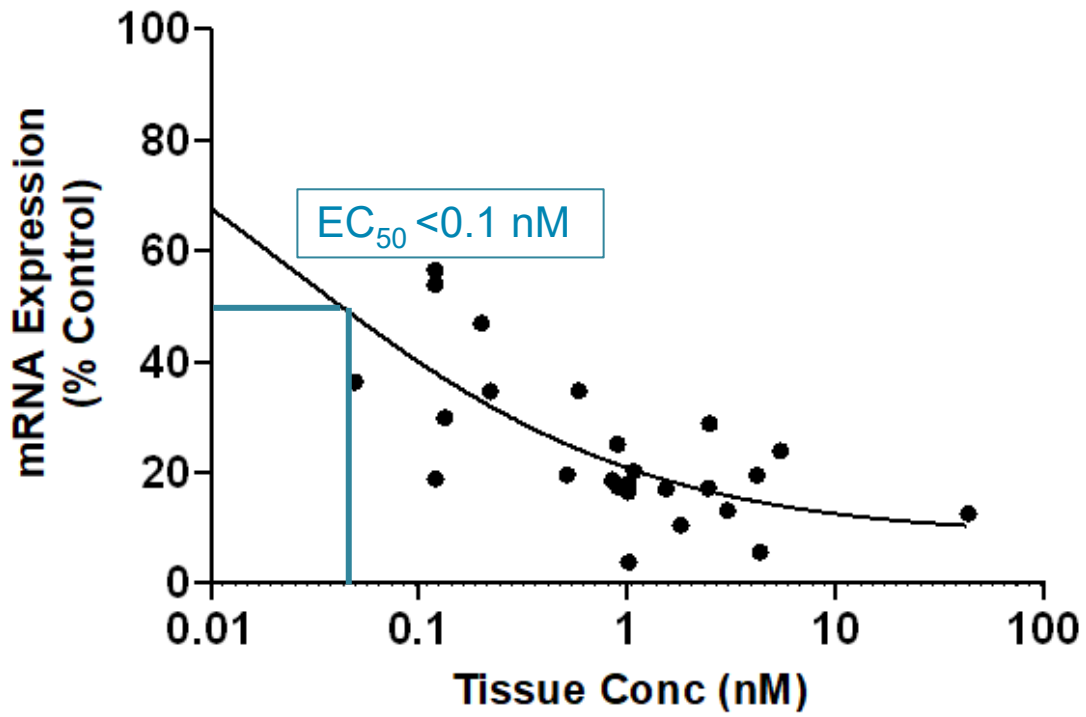
AOC 1001 Reduced Muscle DMPK mRNA in Concentration Responsive Manner in Non-human Primates

NHP Study 6



Necropsy was at 7 weeks,
following 3 doses at 0, 3, and 6 weeks

Day 21 or 28 Post Dosing



Gastrocnemius biopsies collected 21 or 28 days after single doses
ranging from 0.6 to 15 mg/kg siRNA

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AOC 1001 Toxicology Results Support Entry into the Clinic

Summary of IND-enabling 13-week toxicology studies:

- No dose limiting toxicity observed in monkey at the highest dose tested
- No observed platelet or renal toxicity
- No treatment-related histopathologic toxicity observed in monkey
- No changes observed in safety pharmacology parameters (cardiac, respiratory and neurological)
- NOAELs were identified at the highest doses tested in monkey in which pharmacology was essentially saturated
- Safety results were similar in mice

MARINA™: A Phase 1/2 Clinical Trial to Evaluate AOC 1001 in Adult Patients with DM1

Avidity is Collaborating on a Large Natural History Study (NHS) in DM1 called END-DM1

- END-DM1 is a non-interventional NHS that will advance the understanding of disease progression in DM1 patients
- END-DM1 is designed and run by the Myotonic Dystrophy Clinical Research Network (DMCRN)
- 700 patients with DM1 will be enrolled at multiple sites in the US and Europe

Avidity is one of several sponsoring organizations

Myotonic Dystrophy FOUNDATION

MDA For Strength, Independence & Life

FDA

MARINA™ Trial Objectives

Primary

- **Safety and tolerability** of single and multiple doses

Secondary

- **Pharmacokinetics**
- **Pharmacodynamics**
  (*DMPK* mRNA knockdown)
- **Spliceopathy**

Key Exploratory

- Measures of clinical activity:
  - **Mobility**
  - **Muscle strength**
  - **Muscle function**
- Patient-reported outcomes (**PRO**)
- **Quality of life**

MARINA™ Trial of AOC 1001 in Adults with DM1

Patient Population:

N = ~44; Ages 18-65

Dosing:

Single and multi-doses administered via an intravenous (IV) infusion;

Multidose quarterly with booster after 6 weeks;

Biopsies in all cohorts

3:1 RANDOMIZATION (AOC 1001:Placebo)

Part A
Single Dose (N=8)
(1 Cohort)

- 1mg/kg*

Part B
Multi-Ascending Dose (N=36)
(3 Cohorts)

- 2mg/kg (N=12)
- 4mg/kg (N = 12)
- 8mg/kg (N = 12)

- Placebo

OPEN LABEL EXTENSION STUDY

Treatment and observation duration = 6 months;
patients have the option to roll into the OLE study at the end of each cohort

*Dose listed for all cohorts is siRNA component of AOC 1001

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Delivering on DM1

DELIVERING NEXT

PHASE 1/2 MARINA TRIAL ONGOING

MARINA preliminary assessment planned for Q4 2022

FDA & EMA granted Orphan Designation

FDA granted Fast Track Designation

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Facioscapulohumeral Muscular Dystrophy (FSHD) Program

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

Amy, Living with FSHD

FSHD is Often Diagnosed in Young Adults with Few Treatment Options

There are no approved therapeutics for Facioscapulohumeral Muscular Dystrophy

AFFECTS

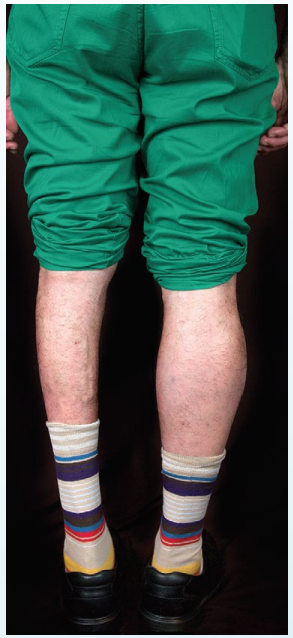
~16,000 - 38,000

PEOPLE IN THE US

0

APPROVED THERAPIES

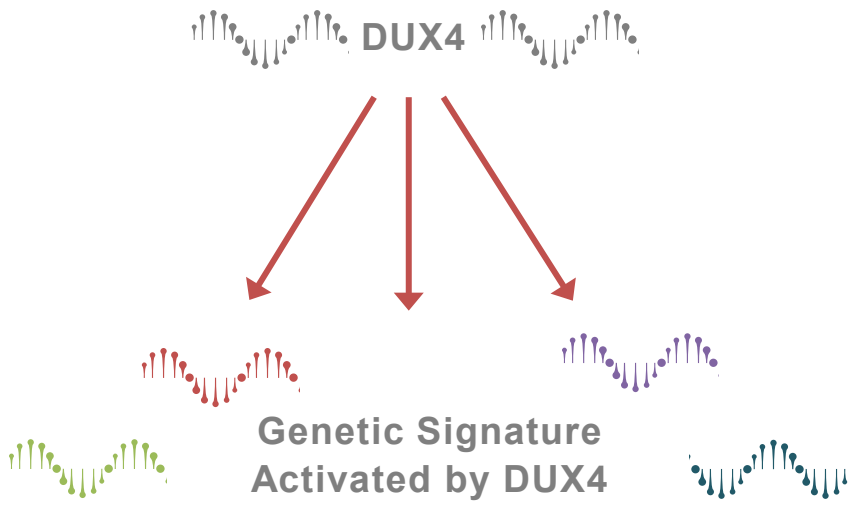
- One of the most common forms of muscular dystrophy
- Autosomal dominant disease caused by abnormal expression of double homeobox 4 (DUX4)
- Characterized by progressive, asymmetric skeletal muscle loss with onset often in teenage and adult years
- About 20% of patients will end up using a wheelchair



FSHD is Caused by Aberrant Expression of DUX4

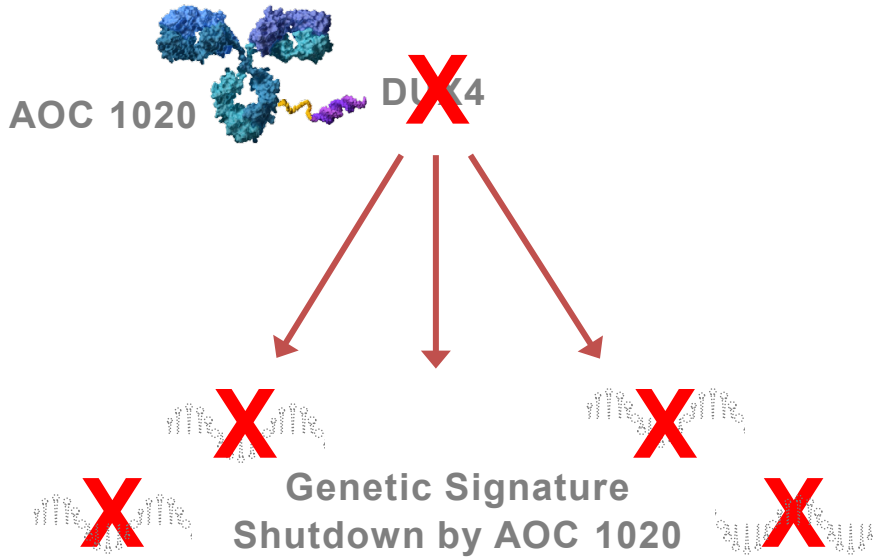
DUX4 activates genes that are toxic to muscle cells

MECHANISM OF DISEASE



Apoptotic, immune signaling, altered myogenesis activated

THERAPEUTIC APPROACH



Apoptotic, immune signaling, altered myogenesis inhibited

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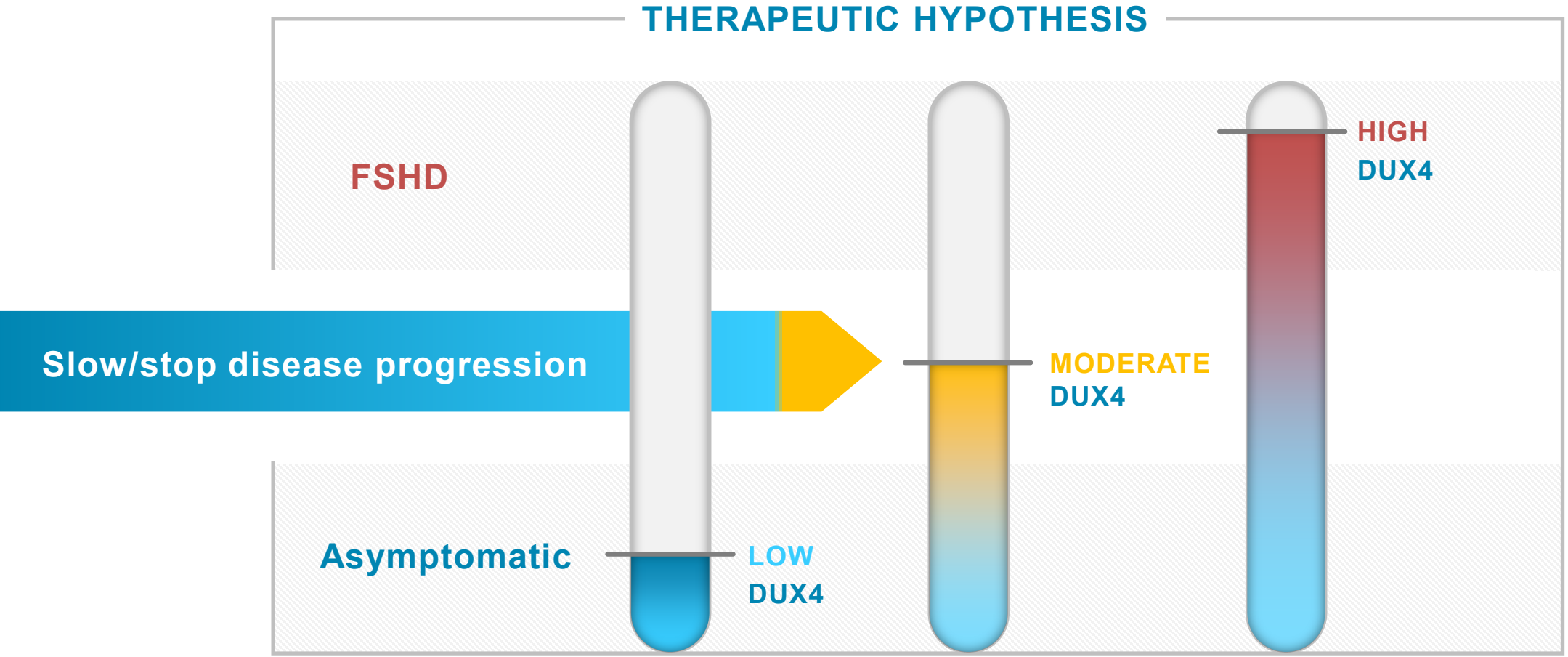
Small Reductions in DUX4 Expression may have Large Clinical Benefit

Avidity's AOC Targets DUX4 mRNA and is Designed to Address the Cause of FSHD



Jones, T.I., et al., Facioscapulohumeral muscular dystrophy family studies of DUX4 expression: evidence for disease modifiers and a quantitative model of pathogenesis. Human Mol Genet 21:4419 (2012)
Ricci,G., et al., Large genotype-phenotype study in carriers of D4Z4 borderline alleles provides guidance for facioscapulohumeral muscular dystrophy diagnosis. *Scientific Reports* **10**:21648 (2020)
Wong C.J. et al., Longitudinal measures of RNA expression and disease activity in FSHD muscle biopsies. *Hum Mol Genet* **29**:1030 (2020)

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AOC 1020: FSHD Development Candidate is Designed to be a Potent and Specific Inhibitor of DUX4 Expression

AOC 1020 Meets our Target Candidate Profile

Transferrin mAb

Screen DUX4 siRNAs in FSHD donor cells using gene signature

Off-target analysis by RNA seq

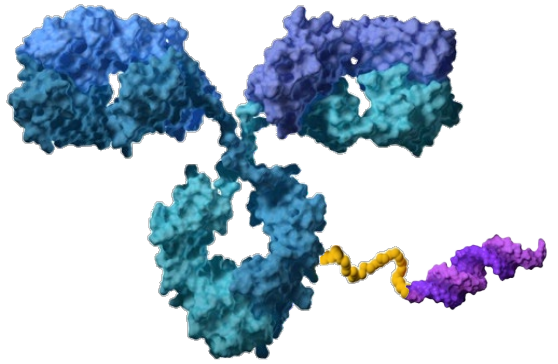
Select potent siRNAs with minimal off-target profile

Target Candidate Profile met

Evaluate in FSHD mouse model & GLP studies

COMPLETED

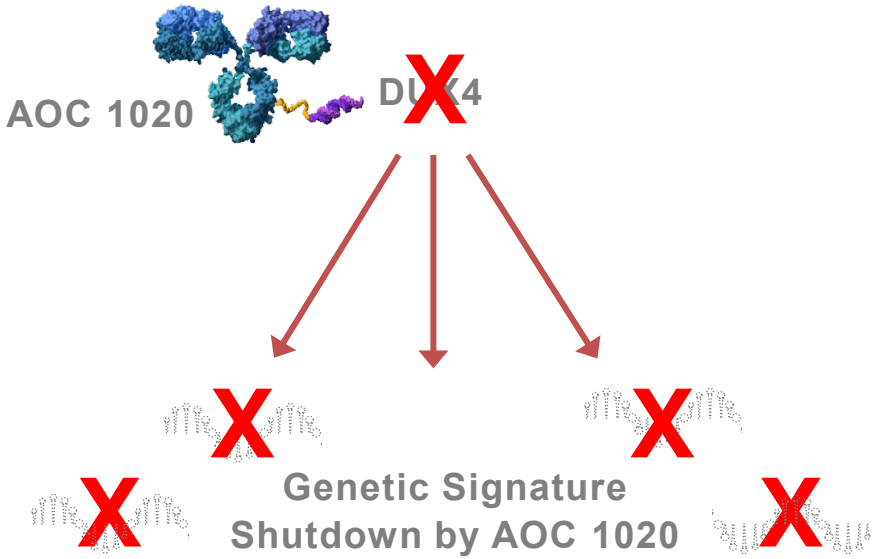
AOC 1020



- Antibody directed to human Transferrin receptor 1
- Non-cleavable linker designed to facilitate siRNA delivery to muscle
- Stabilized siRNA targeting DUX4 mRNA

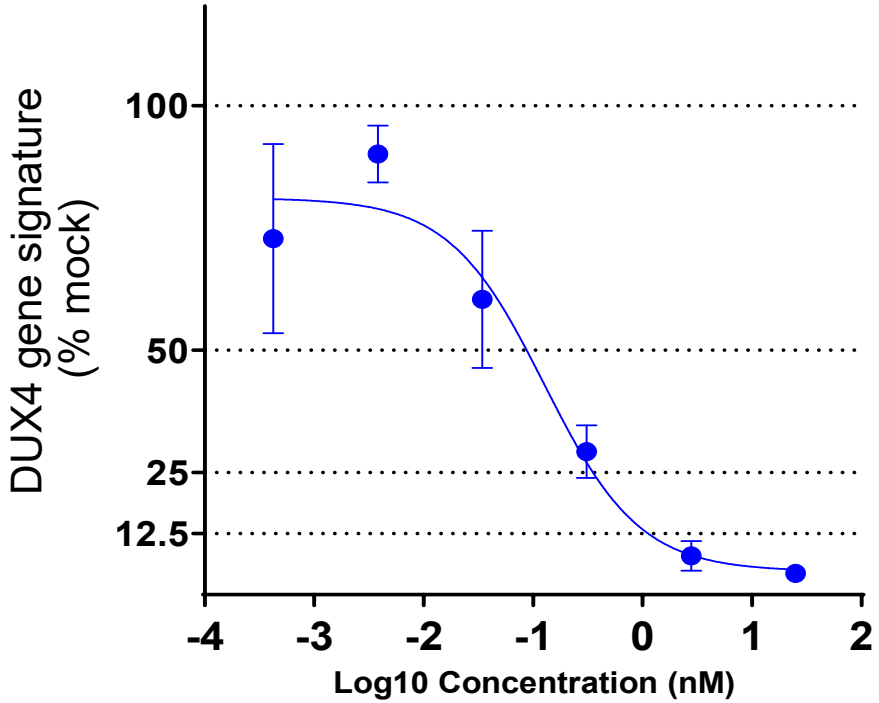
AOC 1020 siRNA Showed Potent Inhibition of DUX4-mediated Gene Signature in FSHD-patient Derived Myoblasts

THERAPEUTIC APPROACH



Apoptotic, immune signaling, altered myogenesis inhibited

FSHD Donor MB05

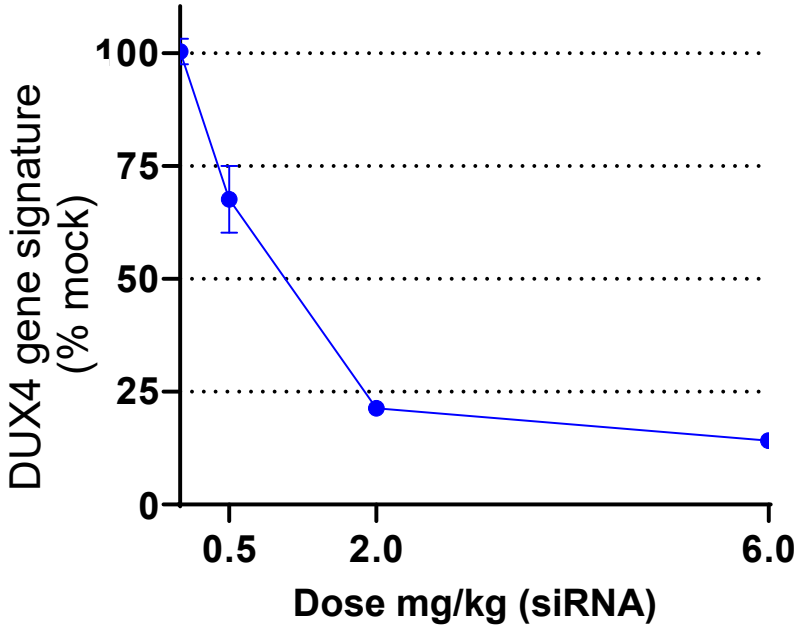


IC_{50} = 0.127 nM

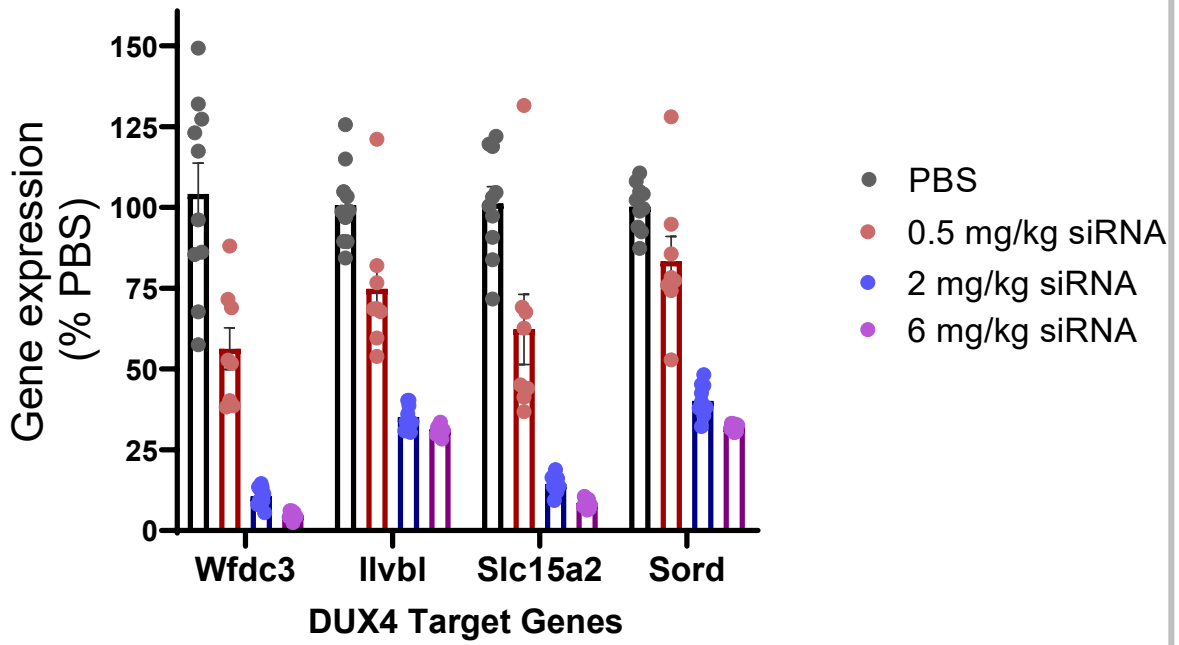
AOC 1020 siRNA Showed Potent Inhibition of DUX4 Gene Signature in Transgenic Mouse Model

Dose-dependent inhibition of DUX4-driven genes in skeletal muscles

Gastrocnemius



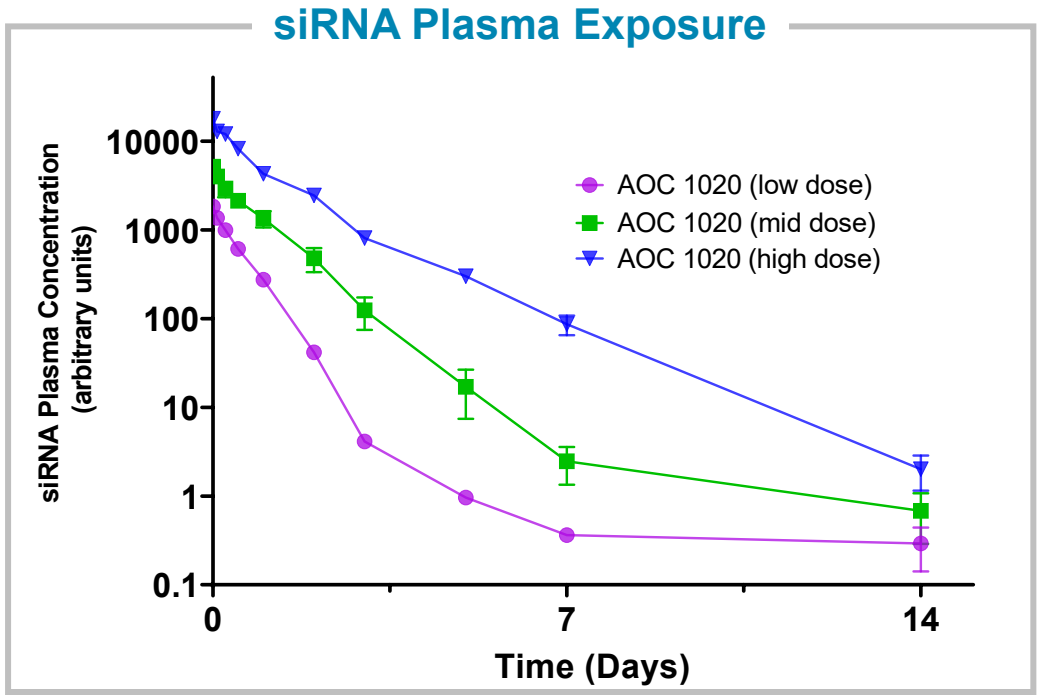
Gastrocnemius

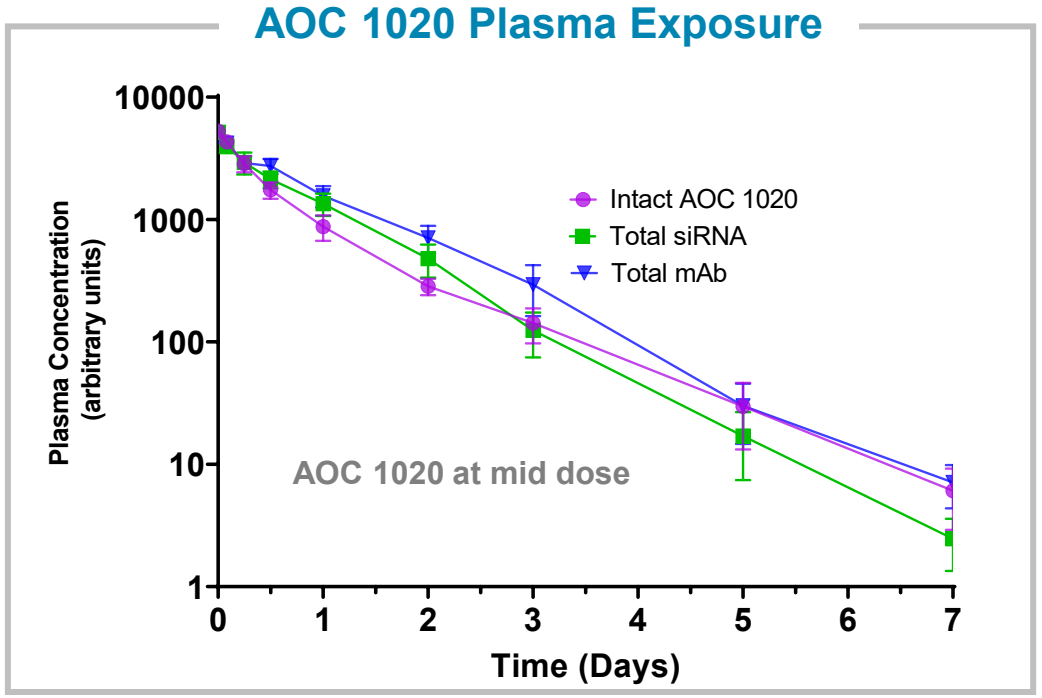


Next Steps: Functional studies are currently underway to support Phase 1 dose selection

AVIDITY BIOSCIENCES

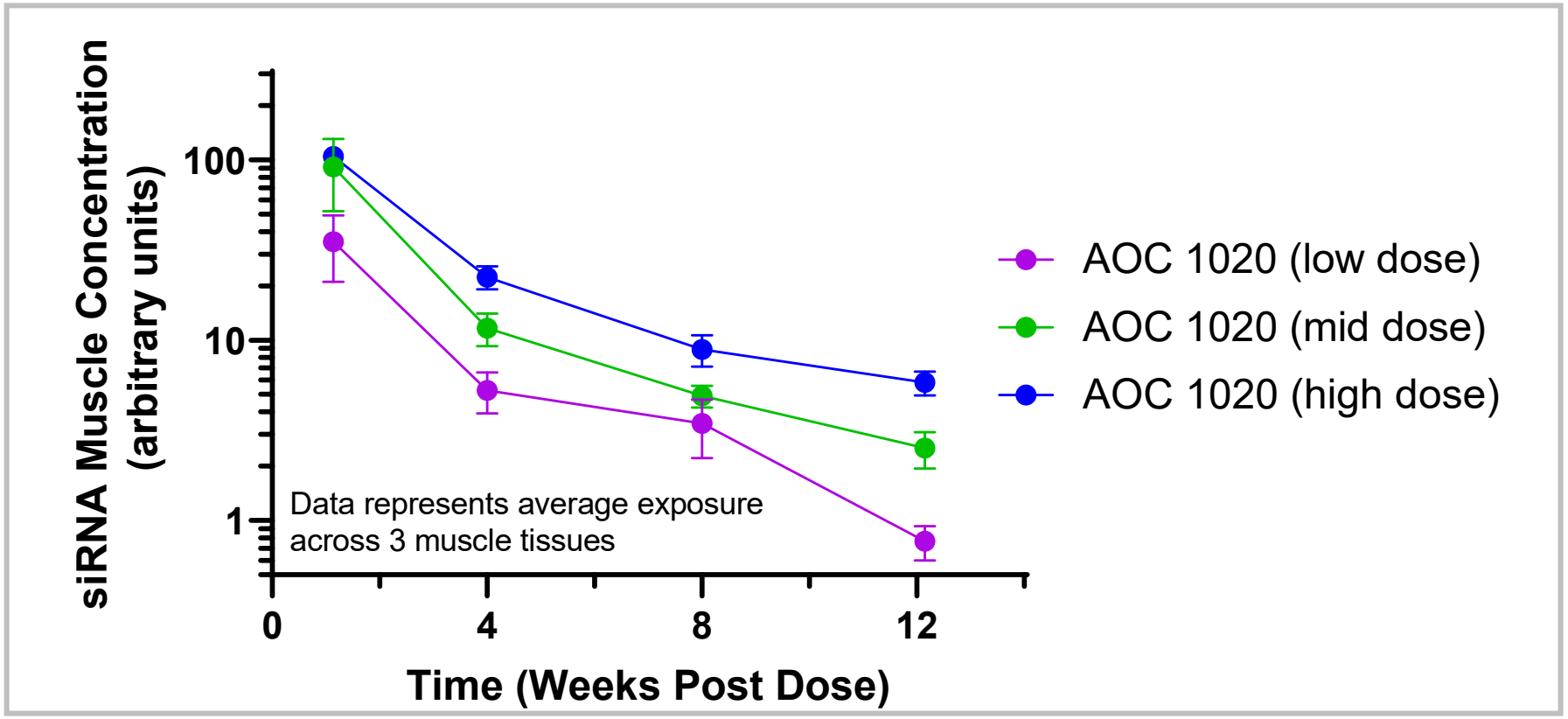
AOC 1020 Plasma PK Results in Monkey Demonstrated Stability of the Antibody-Oligonucleotide Conjugate





- AOC 1020 produced dose-dependent increase in siRNA plasma exposure following single doses
- AOC 1020 was observed to be stable in plasma with no evidence for degradation of the intact AOC

AOC 1020 PK Results in NHP Muscle Tissue Potentially Support an Infrequent Dosing Regimen



- AOC 1020 produced dose-dependent increase in siRNA tissue exposure following single doses in a broad panel of muscle tissue
- Based on our data, we anticipate this will allow for a similar dose schedule as AOC 1001

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AOC 1020 Nonclinical Activities Progressing Well to Support 2022 IND Filing

Summary of non-GLP NHP studies:

- AOC 1020 well tolerated in cynomolgus monkey PKPD study following single and repeat dosing
- No dose limiting toxicity observed in monkeys at the highest dose tested based on in-life safety assessments
- Supports study design for GLP toxicology studies
- AOC 1020 PK results consistent with anticipated exposure based on our AOC platform knowledge
- PK results in plasma and muscle tissue potentially support infrequent dosing

Delivering on FSHD

DELIVERING NEXT

APPROACHING THE CLINIC

Planned clinical trial initiation
by the end of 2022

Supporting ongoing MOVE+ natural history study by FSHD Clinical Trial Research Network

AVIDITY BIOSCIENCES

Duchenne Muscular Dystrophy (DMD) Program

Three programs advancing toward the clinic

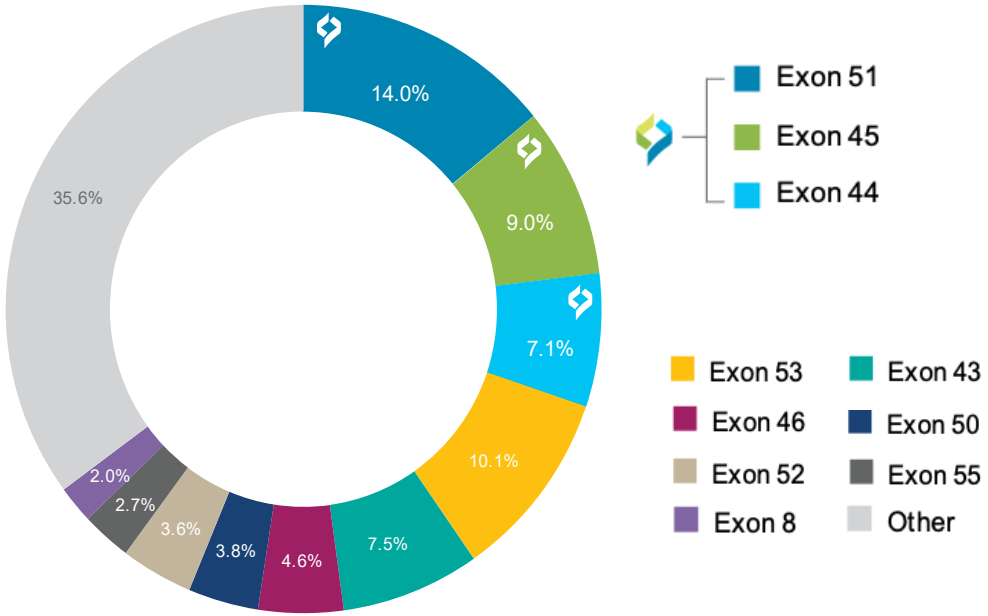
Duchenne Muscular Dystrophy: Disease Overview

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

SIMILAR PREVALENCE
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

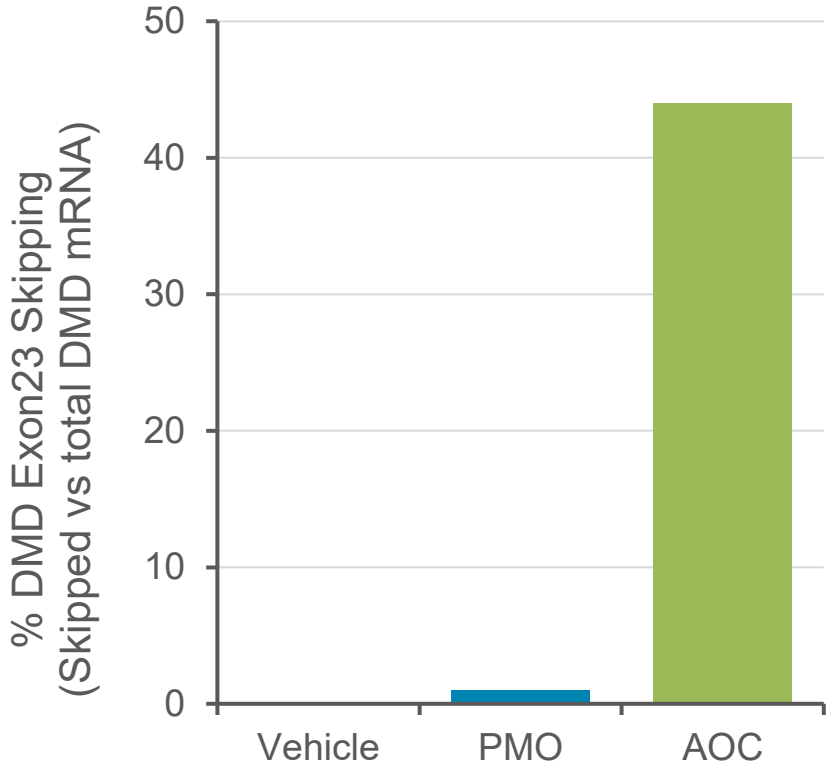


Exons 44, 51 and 45 represent ~30% of the total mutations observed amenable to skipping

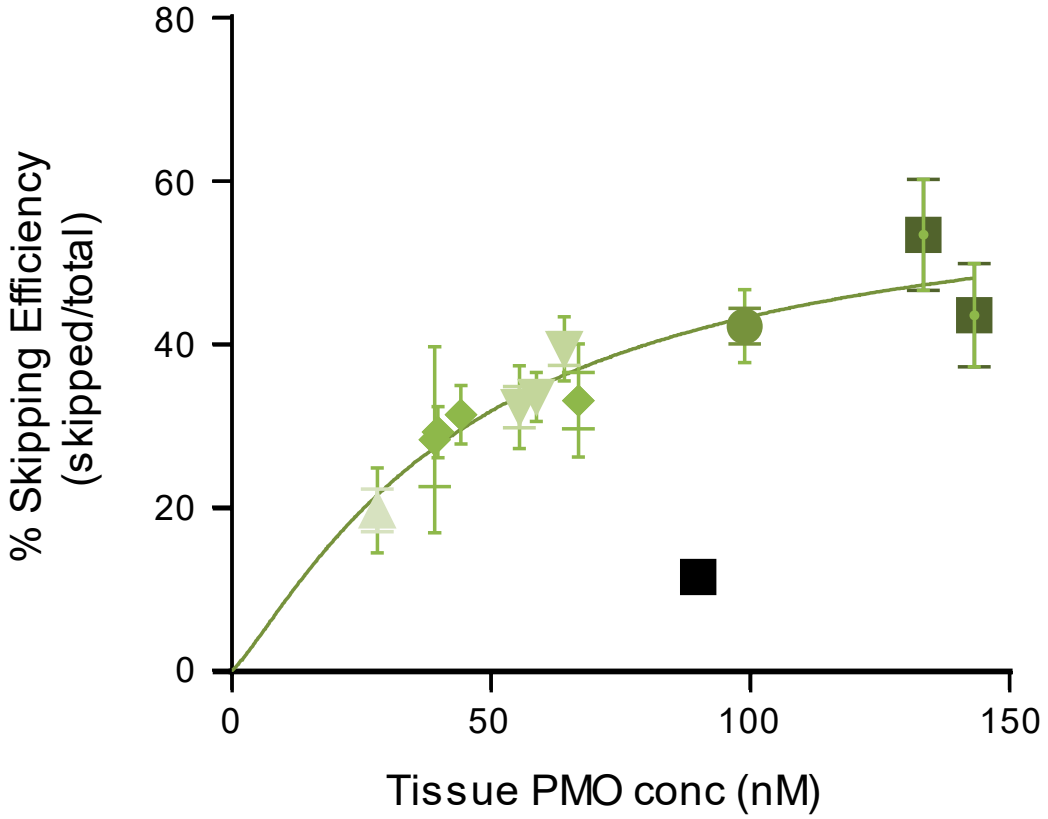
AVIDITY BIOSCIENCES

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



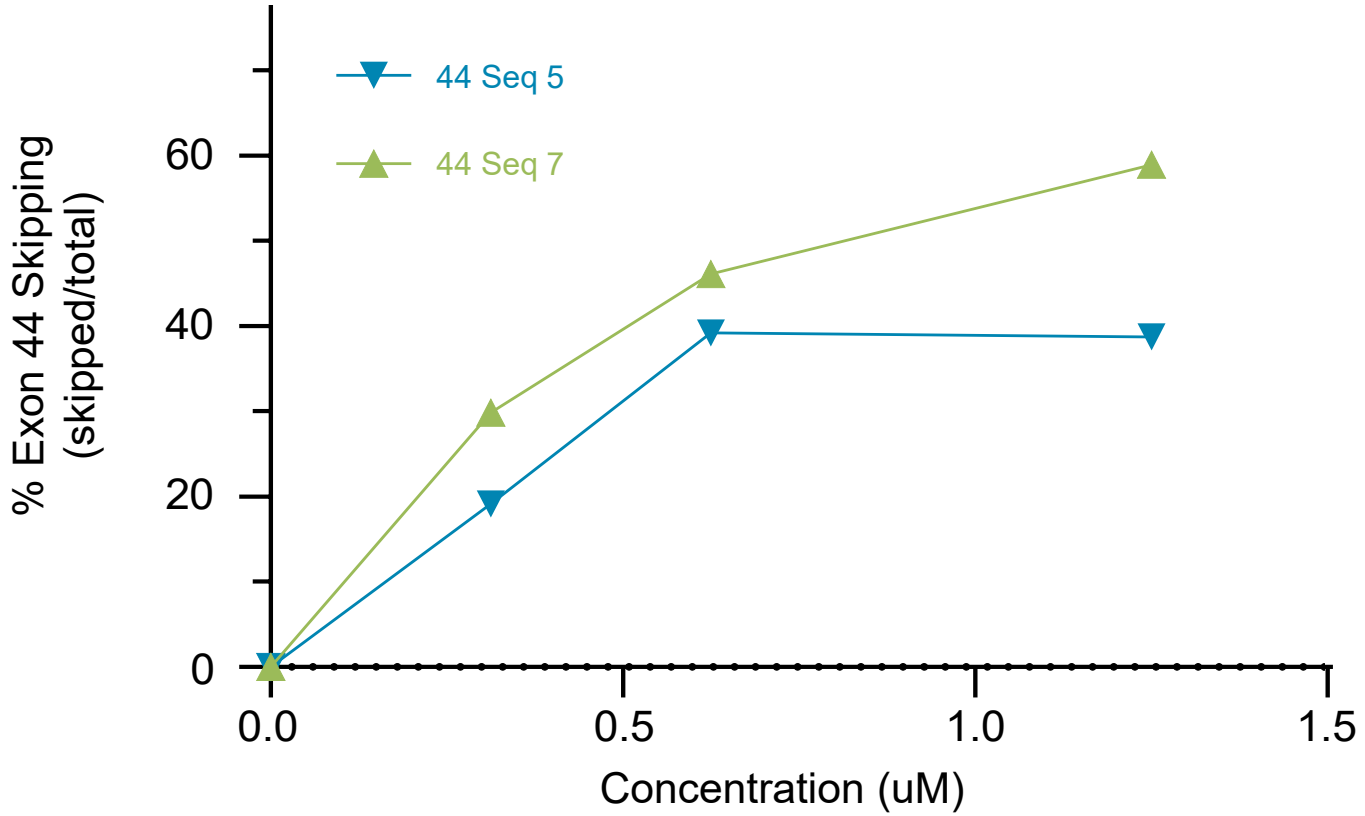
TfR1.mAb AOC 1.9 mg/kg
TfR1.mAb AOC 3.3 mg/kg
TfR1.mAb AOC 5.7 mg/kg
TfR1.mAb AOC 8.5 mg/kg
TfR1.mAb AOC 10 mg/kg
Control.mAb AOC 10 mg/kg

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 observed in human primary myocytes when incubated with two lead development candidates



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Delivering on DMD

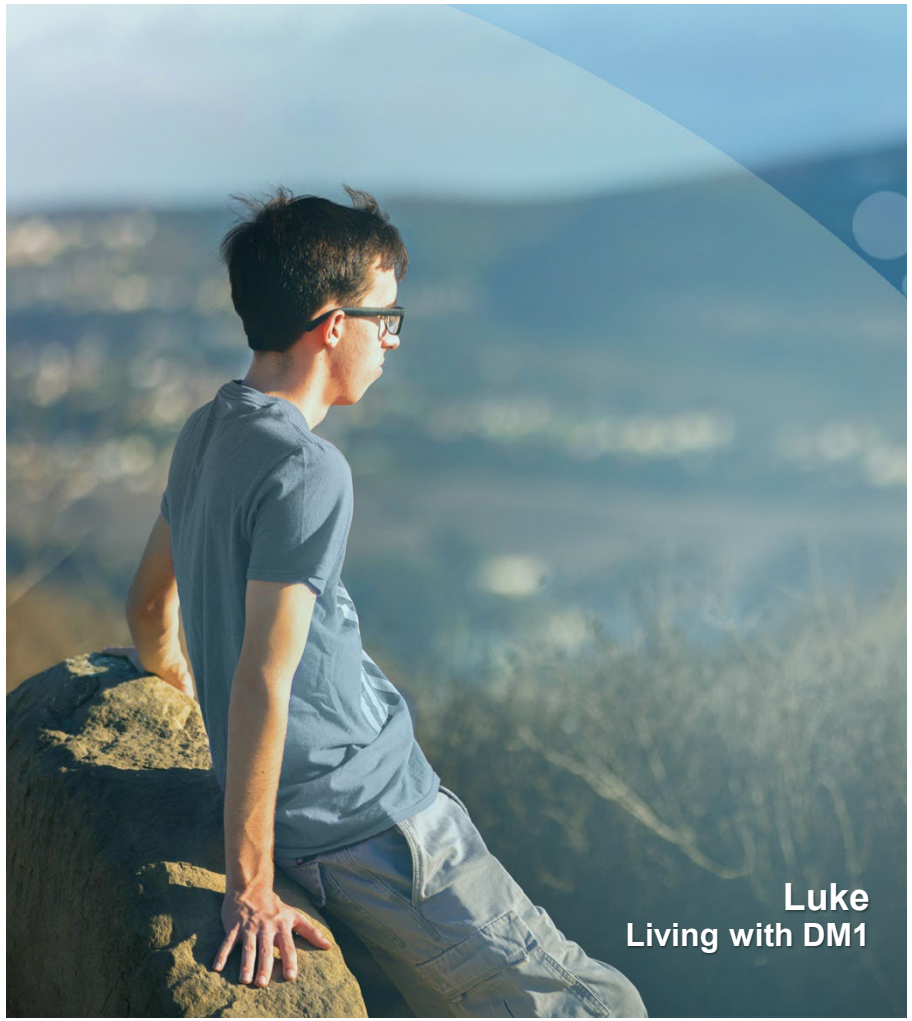
DELIVERING NEXT

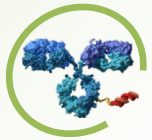
AOC 1044 APPROACHING THE CLINIC

Planned clinical trial initiation for AOC 1044 **by the end of 2022**

Two additional programs in preclinical development

Delivering on the RNA Revolution





DELIVERING NEXT

Phase 1/2 MARINA trial of AOC 1001 ongoing - preliminary assessment planned for **Q4 2022**

AOC 1020 for FSHD & AOC 1044 for DMD anticipated to enter the clinic by the **end of 2022**

Pursuing preclinical proof of concept in additional skeletal muscle and other tissues

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Building for the Future

Experienced Leadership with Significant RNA Therapeutic Expertise

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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 low double-digit tiered royalties

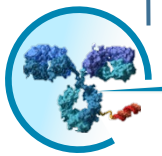
CARDIAC MUSCLE

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



Q4 2021 – Financial Results

In millions (unaudited)	Q421	Q321	Q420	Q421 vs Q321	Q421 vs Q420
Collaboration Revenue	$1.9	$2.2	$2.1	($0.3)	($0.2)
R&D expenses	33.0	24.9	13.6	8.1	19.4
G&A expenses	7.4	6.6	4.8	0.8	2.6
Total operating expenses	40.4	31.5	18.4	8.9	22.0
Loss from operations	(38.5)	(29.3)	(16.3)	(9.2)	(22.2)
Other income (expense)	0.0	0.0	0.0	0.0	0.0
Net loss	($38.5)	($29.3)	($16.3)	($9.2)	($22.2)

In millions (unaudited)	Q421	Q420
Cash, cash equivalents and marketable securities	$405.5	$328.1

Q421 cash balance includes **$155M** in net proceeds from the follow-on offering in August.

Strong cash position to fund the MARINA trial and progress our pipeline and platform