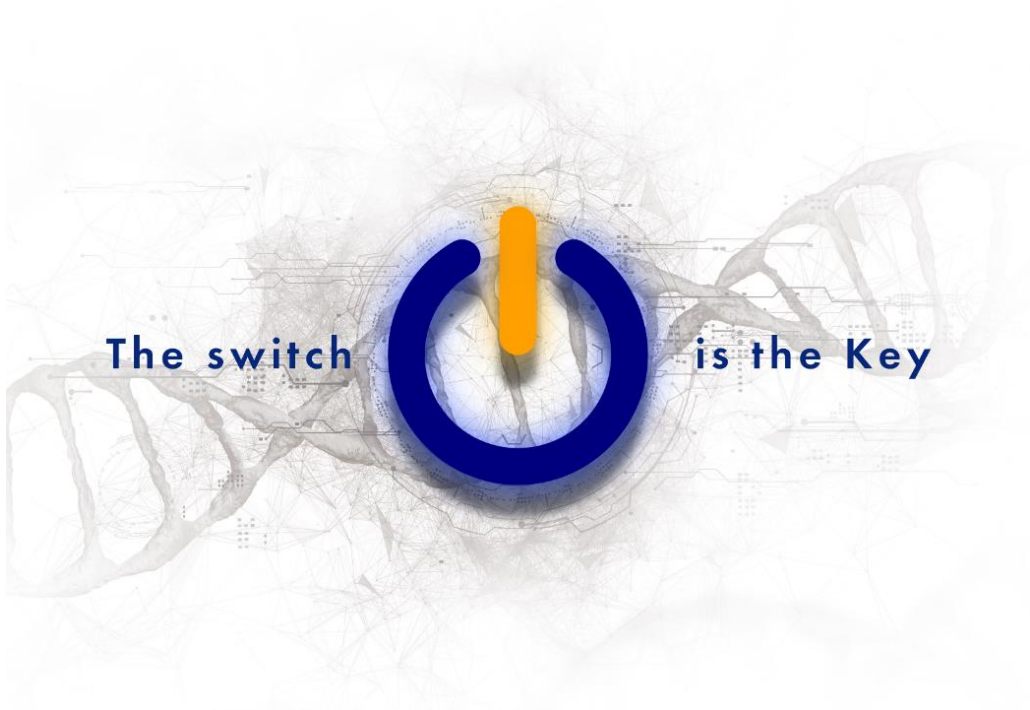
English translation for reference purposes only. In case of any discrepancy, the Japanese version shall prevail

Business Plans and Matters Related to Growth Potential

MODALIS THERAPEUTICS CORPORATION
TSE 4883



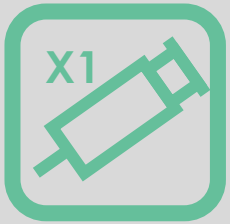
July 14th, 2026

Our vision: *Every Life Deserves Attention*

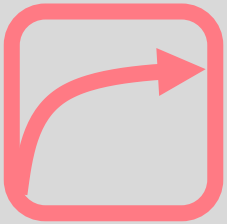
Leveraging CRISPR Epigenome Editing technology to provide fundamental solutions to rare diseases

Key Benefits of Our CRISPR-GNDM® Technology

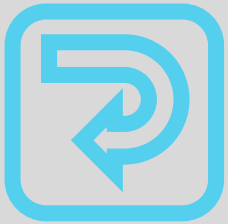
No DNA Cutting
Regulates gene expression without altering DNA



Single dose
Doesn't require Repeated dosing



Long-lasting
Sustained effect for years or decades



Disease Modifying
Aims to provide transformative benefit

INDEX

MODALIS Value Highlights

Building the Next Generation of Genetic Medicines

Platform: CRISPR-GNDM®

Programmable gene activation and repression without DNA cutting

1st company advancing CRISPR-based gene activation therapeutics toward the clinic

4 Programs across neuromuscular diseases

Multiple therapeutic targets validated in vivo

IND MDL-101 is Making Progress Toward

Competitive Advantages

Gene Regulation Without DNA Cleavage

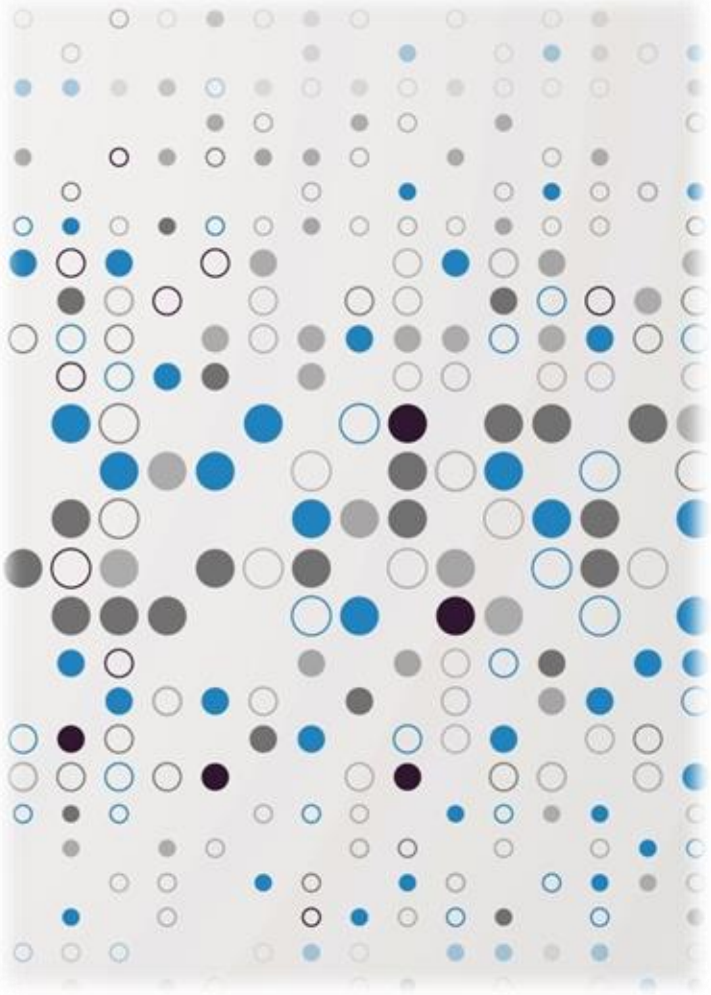
Demonstration of Long-Term Gene Expression Regulation

Applicable to neuromuscular to CNS disorders

Establishing tissue-selective AAV Delivery

A robust intellectual property portfolio

1. Corporate Overview



Corporate Information (As of June 30th, 2026)

MODALIS

Every Life Deserves Attention

Name	Modalis Therapeutics Corporation (Ticker symbol: 4883)
Foundation	Jan 2016
President CEO	Haru Morita
HQs	3-11-5 Nihonbashi-Honcho, Nihonbashi-Lifescience-Bldg.2 7F Chuo-ku, Tokyo 103-0023 Japan
US subsidiary	Modalis Therapeutics Inc. (43 Foundry Avenue, Waltham, Massachusetts)
Capital stock	885 million yen
Outstanding share	97,334,098 shares of common stock
Number of employee	21 (including 8 Ph.D.) (5 in Japan, 16 in US)

What is Modalis?

One Gene Regulation Platform
CRISPR-GNDM®
an epigenome editing technology
↓
Multiple Disease Applications

Turning beneficial genes **ON**
and harmful genes **OFF**

Providing a solution for the long tail of disease

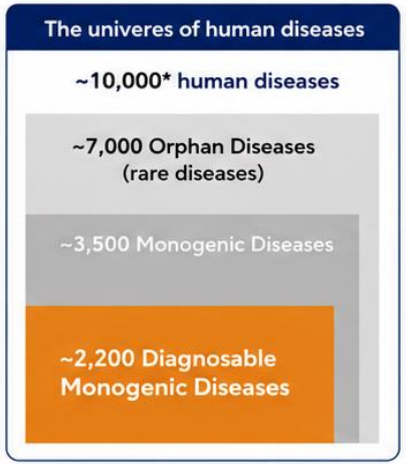
Using scalable technology and supportive policies to address low productivity in rare disease drug development

1 Rare diseases belong to the long tail of disease.

2 Scalable technologies and bespoke regulatory pathways enable us to reach the long tail.

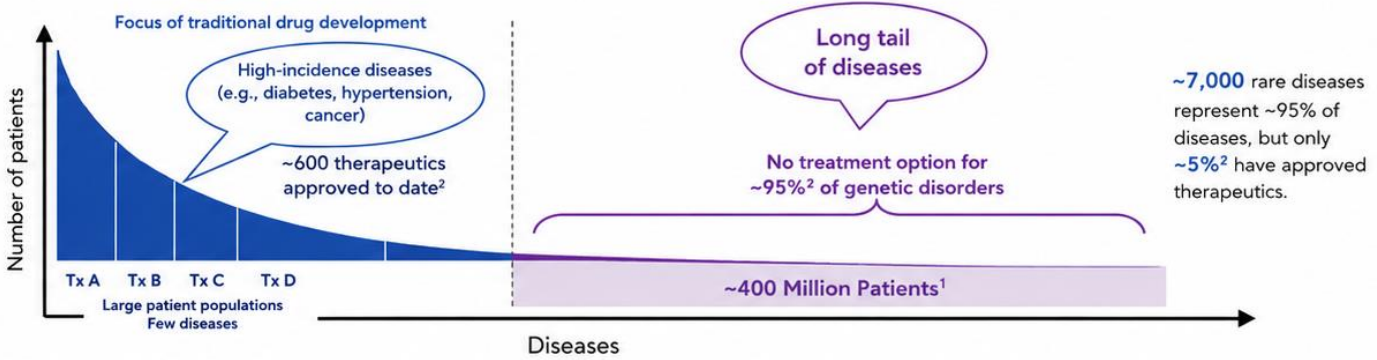
3 The long tail represents ~400 million patients worldwide—an enormous and underserved opportunity.

4 To date, therapeutics have focused on large diseases—only ~600 of ~7,000 (≈5%) have treatments.



~400 Million Patients[1] living with the long tail of diseases worldwide

Unlocks access to the long tail of diseases



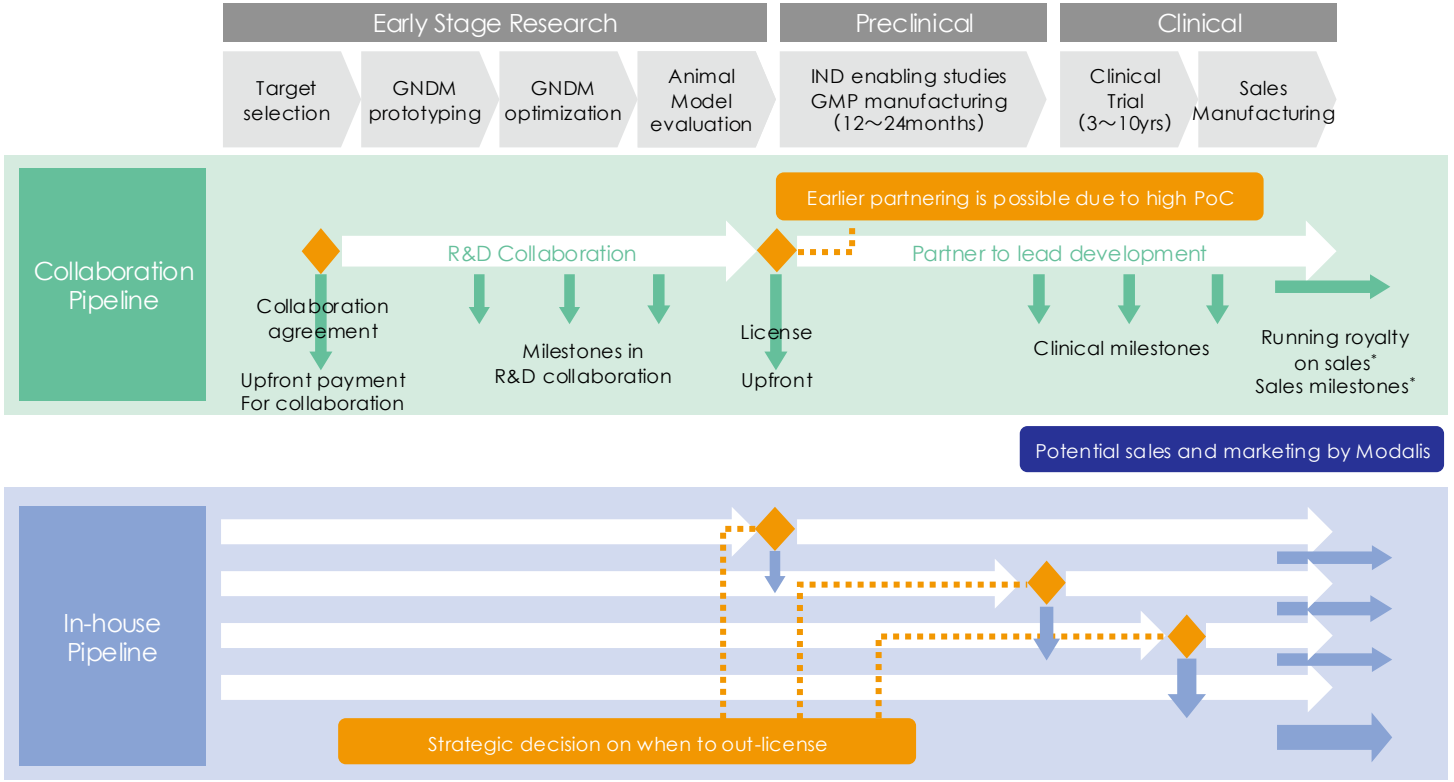
GNDM and scalable approaches, supported by adaptive regulatory pathways, enable us to tackle the long tail and deliver cures to patients who have been left behind

*Estimated total number of human diseases
Source: Evaluate Pharma and CitiResearch (Jan 2024)
2As of July 22, 2019. Source: KEGG, FDA (Active therapeutics of 491 NME, 106 BLA, 17 cell and gene therapy products)
References: 21st Century Cure Act, NIH GARD, Global Genes

MODALIS

Business Model

Hybrid of own pipeline and collaboration pipeline



* future plan

Board of directors

A management team composed of directors and outside directors with extensive experience

Haru Morita President,CEO

Haru began his career in biopharmaceutical R&D at Kirin Brewery (now Kyowa Kirin) and later worked in corporate strategy consulting at Booz & Company (now Strategy&). He subsequently held leadership roles in business development and U.S. operations at Y's Therapeutics. In 2006, he founded REGiMMUNE and, as CEO, advanced its lead program to a Phase II clinical trial in the United States. In 2016, he founded Modalis and has served as Representative Director and CEO since inception. He is a serial biotech entrepreneur and holds B.Eng. and M.Eng. degrees from the University of Tokyo.

Eisaku Nakamura Board member(Audit committee)

Eisaku began his career at a major trading company, where he was involved in businesses across the chemical, healthcare, and high-tech sectors. He later worked as a venture capitalist, investing in startup companies, and subsequently served as a director and auditor of several biotechnology companies. He currently serves on the boards of D. Western Therapeutics Institute and Solasia Pharma. He holds B.Eng. and M.Eng. degrees from the University of Tokyo.

Hideki Takeda Board Member

Mr. Takeda began his career in research and intellectual property at Fujisawa Pharmaceutical (now Astellas Pharma), where he specialized in patent strategy and IP management. He later served as a Science and Technology Coordinator for a national life sciences innovation program. In 2009, he founded Medical Patent Research Inc. and has since advised numerous biotech startups on intellectual property strategy. He also played a key role in establishing the Japan Retina Institute, the predecessor of Healios. He holds B.S. and M.S. degrees in Biology from Osaka University.

Teruhisa Tajima, CPA Board member(Audit committee)

At a major auditing firm and related organizations, he was engaged in consulting and support services on finance and accounting for biotech startups. He later established Tajima Certified Public Accountant Office, where he provides tax and consulting services mainly for biotech ventures.
He serves as an outside auditor and outside director for companies such as PRISM BioLab Co., Ltd., Obunsha Co., Ltd., and Matsuya Foods Holdings Co., Ltd.
He graduated from Kobe University and is a Certified Public Accountant.

Joseph S. McCracken, DVM Board member

Joe previously served as Vice President of Business Development at Genentech and held senior business development and leadership roles at Roche, Aventis Pharma, and Rhône-Poulenc Rorer (now Sanofi). As Global Head of Business Development and Licensing at Roche Pharma, he led worldwide partnering activities. He currently advises biopharmaceutical companies on corporate strategy and business development and serves on the board of Savara Pharmaceuticals. He holds B.S., M.S., and D.V.M. degrees from The Ohio State University.

Toshio Furuta, Attorney at Law Board member(Audit committee)

As the representative of Clair Law Firm, he has long supported the establishment and growth of startup companies. He also serves as a director (Audit and Supervisory Committee member) at listed companies, including CanBas Co., Ltd.
He is an attorney and a member of the Tokyo Bar Association.

MODALIS

Execution team

A management team with deep expertise in gene therapy and its business

Haru Morita

President,CEO

- Founder and CEO responsible for corporate strategy, business development, and financing
- Serial biotech entrepreneur with experience spanning R&D, business development, and management
- Founded REGiMMUNE and advanced its lead program to a U.S. Phase II clinical trial
- Founded Modalis in 2016 following roles at Kyowa Kirin, Strategy&, and Y's Therapeutics
- B.Eng. and M.Eng., University of Tokyo

Tetsuya Yamagata

CSO

- Oversees R&D strategy from discovery through clinical development
- Physician-scientist with expertise in immunology and molecular biology
- Conducted immune tolerance research at Harvard University; served as Research Fellow and Instructor
- Previously held positions at Dokkyo Medical University, Tempero Pharmaceuticals, and GSK
- M.D. and Ph.D., University of Tokyo

Nakashima Yosuke

VP, Operations and Business Development

- Responsible for business development and management of the U.S. subsidiary
- Engaged in agribio research and development and business planning in the life sciences field at Sumitomo Chemical
- Led the establishment and operation of the corporate venture function at its U.S. subsidiary
- Executed numerous partnering and investment projects in the life sciences field
- Graduated from the Faculty of Agriculture at Nagoya University and completed graduate studies there; holds an MBA from MIT Sloan

Yuanbo Qin

VP, Neuromuscular Diseases

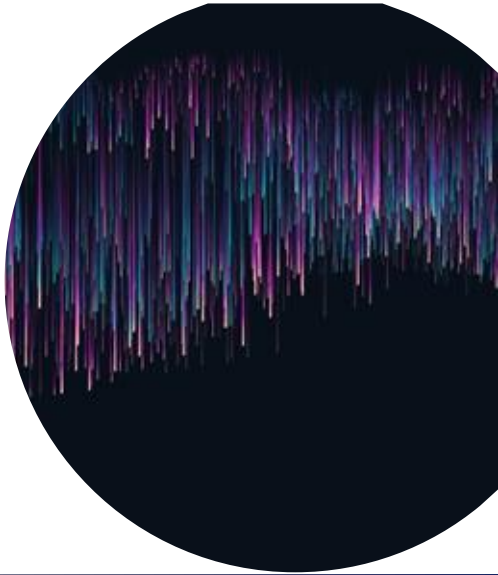
- Oversees R&D in the neuromuscular disease field
- Joined from the founding stage and led the establishment and development of the CRISPR-GNDM® technology; an inventor of several key patents forming the foundation of Modalis's IP portfolio
- Played a central role in launching and advancing the lead program MDL-101
- Holds a PhD from the Chinese Academy of Sciences; served as a Visiting Researcher at Yale University and a Research Fellow at Harvard Medical School

Seth Levy

VP, Manufacturing

- Oversees the CMC function, driving manufacturing strategy, process development, and analytical development
- An expert in manufacturing development with extensive CMC experience in gene therapy
- Supported the clinical advancement of numerous gene therapy programs at Thermo-Fisher Scientific / Brammer Bio
- Led analytical and process development as well as the establishment of manufacturing platforms
- Graduated from the University of Massachusetts Amherst; holds a PhD from the University of Alabama (Molecular and Cellular Pathology)

2. Gene Therapy to Epigenome Editing



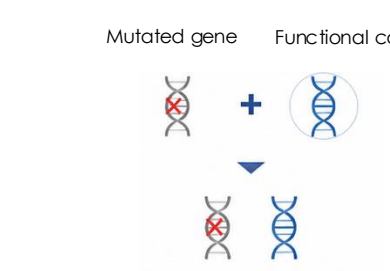
Three Approaches to Genetic Medicines

Different ways to address the root cause of genetic diseases

Addition

Gene Supplementation

Add a functional copy of the missing gene



A new healthy gene is added while the original mutation remains

- Delivers a new gene
- DNA sequence unchanged
- Long-term expression depends on vector

Luxturna® Zolgensma®

Repair

Genome/Base Editing

Correct the disease-causing mutation

Genome Editing

Modify DNA sequence At the target site

Permanent correction of the mutation

Base Editing

Correct specific DNA letters

AGATCA

AGCTCA

Precise single-base Correction without Double-strand break

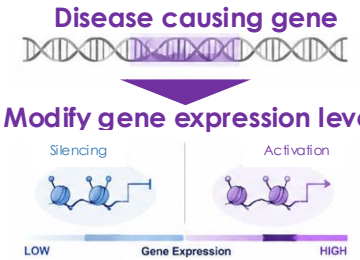
- Directly corrects the mutation
- Permanent change in DNA
- Potential for one-time treatment

Casgevy® Verve-101®

Regulate

Epigenome Editing (CRISPR-GNDM®)

Adjust gene expression without changing DNA seq



gene regulation at the transcriptional level without double-strand breaks

- Regulates gene expression
- No change to DNA sequence
- Reversible and tunable

MODALIS CRISPR-GNDM®

Level of Intervention

Protein
Add a functional protein/gene product

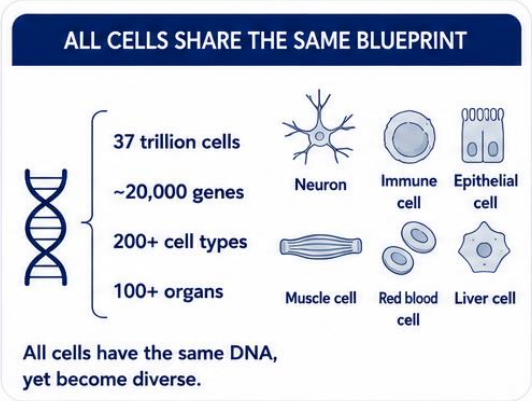
DNA sequence
Modify DNA sequence

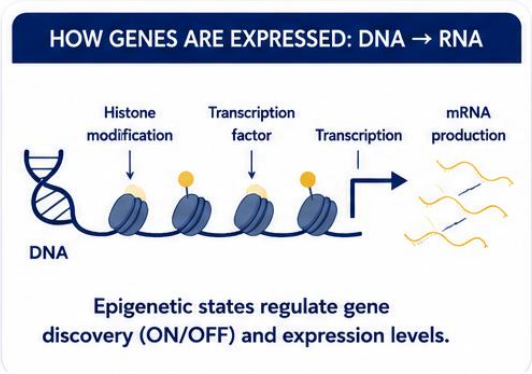
Gene Expression
Control when, where, and how much a gene to express

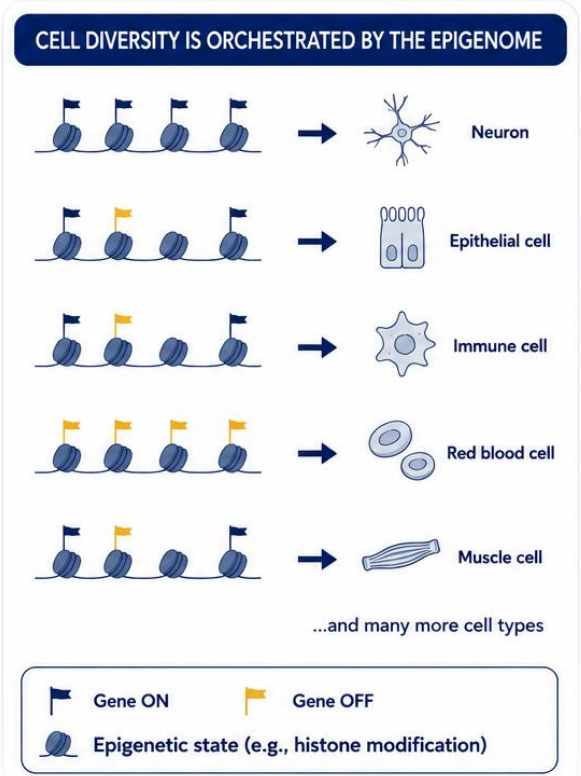
MODALIS

Epigenome

The mechanism by which genes determine when, where, and how much they are transcribed, thereby creating diversity in cells and their functions



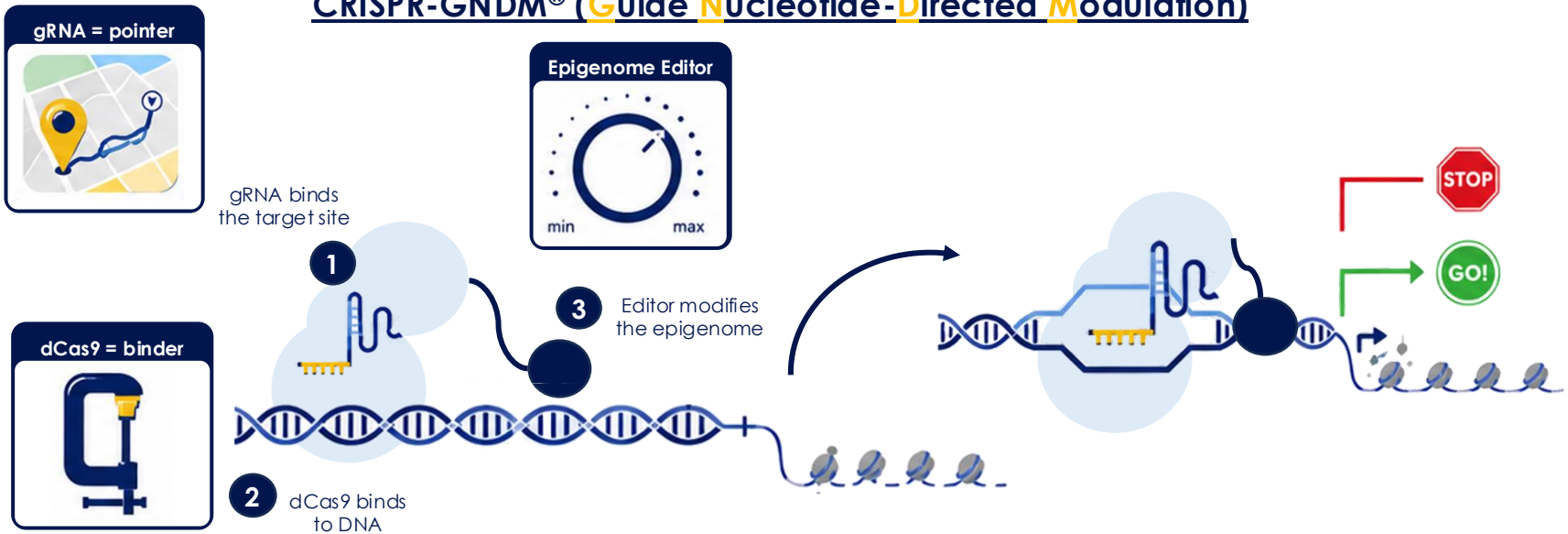




Cell diversity is controlled not only by the DNA sequence but by the epigenome

CRISPR-GNDM®'s mechanism of action

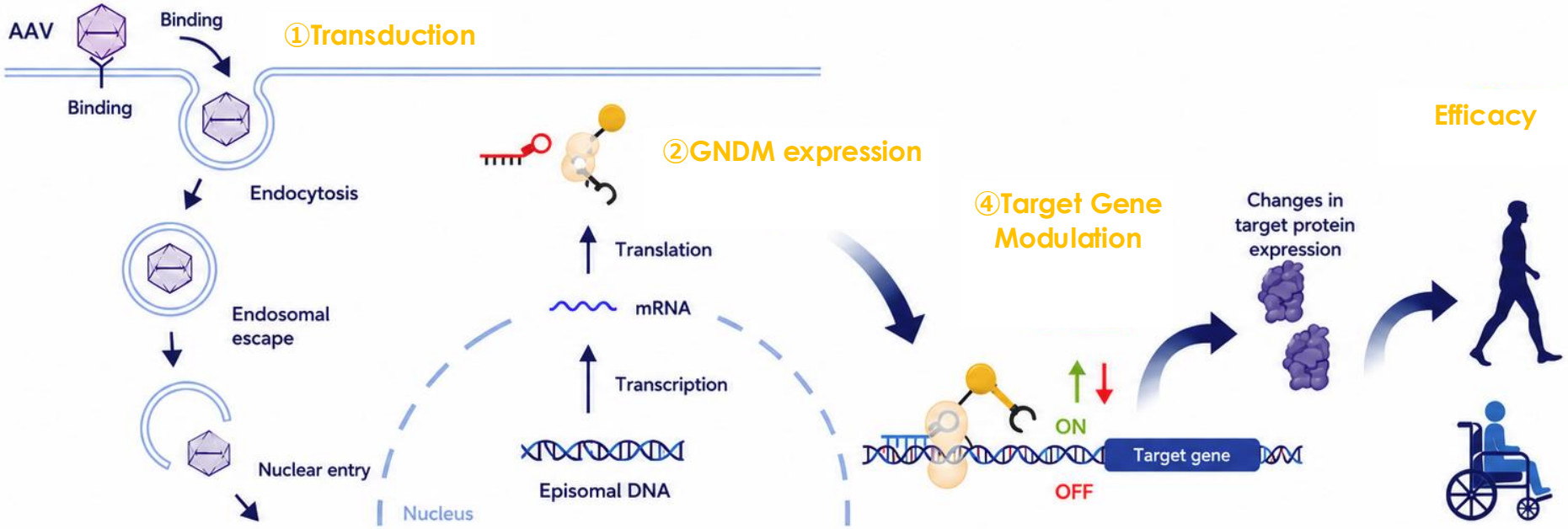
GNDM restores the controls target protein expression through epigenetic regulation



1	**Guide RNA (gRNA)** Directs the system to the target genomic site.	2	**dCas9** Binds to the DNA without cutting (no double-strand breaks)	3	**Epigenome Editor** Modifies the epigenome to precisely turn gene expression ON or OF

Three Steps from Dosing to Therapeutic Efficacy

AAV delivers GNDM to target cells, GNDM is expressed as a protein, and then modulates the target gene to generate therapeutic benefit



Our core competency = GNDM payload

intellectual property, target selection expertise, drug discovery engine, and the selection of capsids and our subsequent development capabilities

IP

A robust IP foundation for our proprietary GNDM technology

Selecting target

Extensive knowledge and experience in the identification and evaluation of optimal targets for gene therapy

Discovery Engine

A Drug Discovery Engine that Accelerates Design, Evaluation, and Optimization

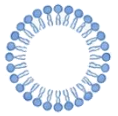
Capsid Evaluation

Expertise that drives the process from selecting the optimal capsid and development

Payload
=CRISPR-GNDM®

Gene Therapy

Vector



In collaboration with

JCR Pharmaceuticals Co.,Ltd.

BROAD INSTITUTE

GENIXCURE

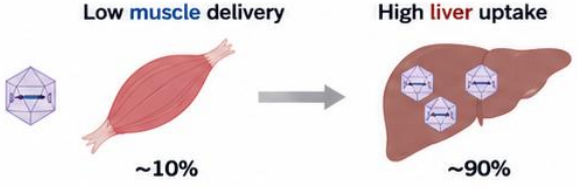
By integrating these strengths, we will accelerate the realization of gene therapy and deliver value to patients.

MODALIS

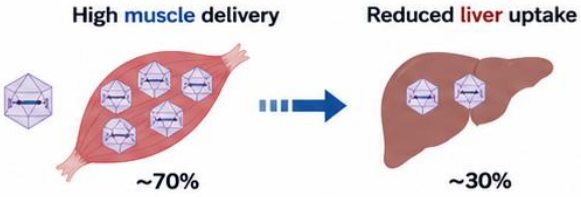
Innovation in Muscle-Selective Capsids Improves Efficacy, Safety and Economics

MyoAAV enables more delivery to muscle while reducing off-target exposure in the liver, resulting in better efficacy, improved safety and lower cost

Traditional AAV Capsids
(e.g., AAV2, 6, 8, 9)



Muscle-Selective Capsids (MyoAAV)



Engineered capsids dramatically improve tissue tropism, minimizing off-target exposure in the liver.

What This Means for Gene Therapy

Higher Efficacy
More vector reaches muscle, enabling stronger therapeutic effect.

Better Safety
Lower off-target exposure reduces risks such as hepatotoxicity and thrombosis.

Lower Manufacturing Cost
More efficient transduction allows lower dosing and reduces overall production cost.

Adopted Across Modalis Muscle Programs

MDL-101 (LAMA2-RD)	MyoAAV+dCas9-mini VPR High efficacy and safety with optimized dosing
MDL-201 (DMD)	Muscle selective systemic delivery aim for greater efficacy at low dose
MDL-202 (DM1)	Muscle selective delivery to maximize efficacy and safety
MDL-103 (FSHD)	Precise gene upregulation with muscle selective delivery capsid

MyoAAV technology enhances the potential of our muscle disease pipeline.

Advances in muscle-selective capsids enable higher efficacy, improved safety and lower cost, accelerating the development and value of Modalis' muscle disease pipeline

The ratios shown in the figure are for illustrative purposes only and do not represent actual measurements.

Another Paradigm Shift in AAV GTx

From Competing on "Performance" to Mastering the "Drive" (Immunology)

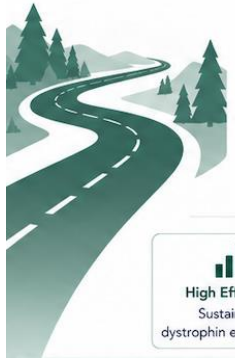
Recent reports from other companies have demonstrated that, with appropriate immunosuppressive measures, even existing AAV technologies can achieve high efficacy and safety at high doses.

The Old Paradigm: Competing on Vector Performance
Focus on building a better car

- **Accurate rotation** (Tissue trophism)
- **Bigger engine** (High dose)
- **Light Body** (smaller gene)
- **Smarter Design** (Promoter, codon optimization)

Key Challenges
- Insufficient transduction
- Cannot reach enough target cells

The New Paradigm: Mastering the "Drive" (Immunology)
Focus on navigating the course of immune responses



Immunosuppression Protocol (RGX-202)

Eculizumab (C5 inhibitor) + Sirolimus (mTOR inhibitor) + Prednisone (Steroid)

- ✓ AAV8 (established capsid)
- ✓ High dose: 2×10^{14} vg/kg
- ✓ DMD patients

Key Findings (Interim Phase 1/2)

High Efficacy	High VCN Achieved	Favorable Safety
Sustained dystrophin expression	Mean VCN >20 (some >30)	No major safety signals to date

Demonstrates that with the right immunology, even conventional technology can deliver both efficacy and safety

Redefining the Bottleneck
The limit is not just the capsid, but immunology.

Changes to the Game Rules
Multi-thlon-ization

Rising Importance of Immune Control
The Emergence of New Operational Variables

Expanding the Ecosystem
Immune control technologies and biomarker companies may gain strategic value.

The next era of AAV gene therapy success is defined not only by "what car we build," but also by "how well we drive."

Source: REGENBIO reports (ASGCT 2026), company press releases, public information Note: VCN = Vector Copy Number

Approval Status of GTx

Otarmeni, a Treatment for Hereditary Hearing Loss, Receives Approval

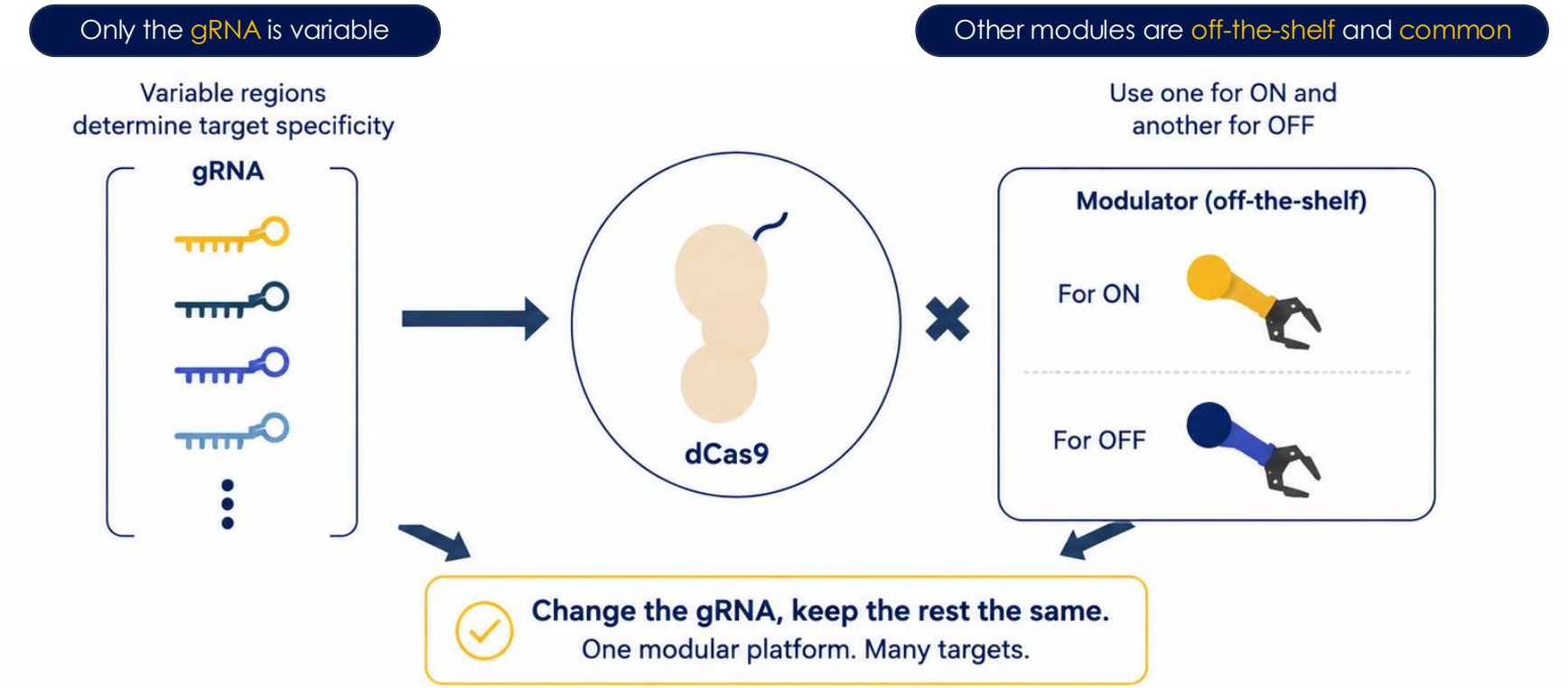
In vivo gene therapies approved by US FDA

Trade Name	Year of Approval	Price	Indication	Manufacturer	Patient Population	WW market size* (mil USD)
Lxturna	2017	$850k	RPE65	Roche(Spark)	2 per 100,000	$65M
Zolgensma	2018	$2.1M	SMA	Novartis(Avexis)	1 in 15,000 live births (Approx. 100s ~1000 patients in US)	$1.3B[#3]
HEMGENIX	2022	$3.5M	Hemophilia B	uniQure CSL Behring	1 in 30,000 male	$88M[#3]
Vyjuvek	2023	$631k per patient year	DEB[*2]	Krystal	3.5–20.4 in 1 million	~$200M[#2]
Roctavian		$2.9M	Hemophilia A	BioMarin	1 in 5,000 male	$262M[#4]
beqvez	2024	$3.5M	Hemophilia B	pfizer	1 in 30,000 male	$88M[#3]
ELEVIDYS		$3.2M	DMD	Sarepta	1 in 3,500 male birth	$4.1B[#4]
Itvisma	2025	$2.6M	SMA	Novartis	Over 2yrs SMA patients Approx. 1000s ~10k patients in US	~$10B[#2]
Otarmeni	2026	TBD	hearing loss	Regeneron	50 newborn in a year/US	$130 million

Source: National Organization for Rare Disorder、#2 Fierce Biotech #3 Corporate website # Mordor Intelligence, 2026 #5 Fortune Business Insight
*1: Spinal muscular atrophy *2: dystrophic epidermolysis bullosa *3: Duchenne muscular dystrophy *4: severe leukocyte adhesion deficiency type I

GNDM is scalable

gRNA is the only variable which is unique for each target. Other components are off-the-shelf to assemble.



Precision Medicine Modalities: Different Tools for Different Needs

Precision technologies are complementary, not competitive.
GNDM expands the treatable space by enabling regulation of large genes and complex biology

	Conventional Gene Therapy (Gene Replacement)	Gene Editing (Nuclease / Base / Prime)	RNA Therapeutics (ASO / siRNA / mRNA)	Gene Regulation (GNDM®) (Activation / Repression)
Best Fit Applications	• Missing or non-functional proteins • Monogenic diseases with small genes	• Correction of disease-causing mutations • Single nucleotide or small insertion/deletion	• Temporary knockdown or modulation • Diseases where repeat dosing is acceptable	• Activation of protective genes • Repression of disease genes • Large genes / complex biology requiring physiological regulation
Target Gene Size	Small (≤~4kb)	Small to medium (≤ ~8 kb for many approaches)	Not limited by gene size (Target transcript dependent)	Large (No practical limit)
Durability	Long-term (potentially lifelong)	Permanent (one-time treatment)	Temporary (months)	Long-term (potentially lifelong)
Dosing Paradigm	One-time administration (in most cases)	One-time administration (in most cases)	Repeat dosing (frequency depends on modality)	One-time administration (in most cases)
Key Considerations	• Packaging capacity limit • Immune response to AAV	• Off-target editing risk • DNA double-strand breaks (with some platforms)	• Requires repeat dosing • Delivery to target tissues	• Regulation maintains natural biology • Broad applicability (LoF & GoF) • Size agnostic
Approved Examples (US/EU/JP)	LUXTURNA® (RPE65-LCA) ZOLGENSMA® (SMA) ELEVIDYS® (DMD)	CASGEVY® (SCD/β-thalassemia) (Additional approvals expected in the future)	SPINRAZA® (SMA) AMVUTTRA® (hATTR-PN) QFITLIA® (HAE)	No approved products yet (First-in-class approach) Modalis programs: MDL-101, MDL-201, MDL-202, MDL-103 and more

Where GNDM® Creates Unique Value

- Addresses large genes that are difficult to deliver or edit
- Enables upregulation of beneficial genes and repression of disease drivers
- Provides long-term, durable control with a single administration
- Expands the treatable space across a wide range of genetic diseases

ASO: Antisense Oligonucleotide, siRNA: small interfering RNA,
LOF: Loss of function, GOF: gain of function

MODALIS

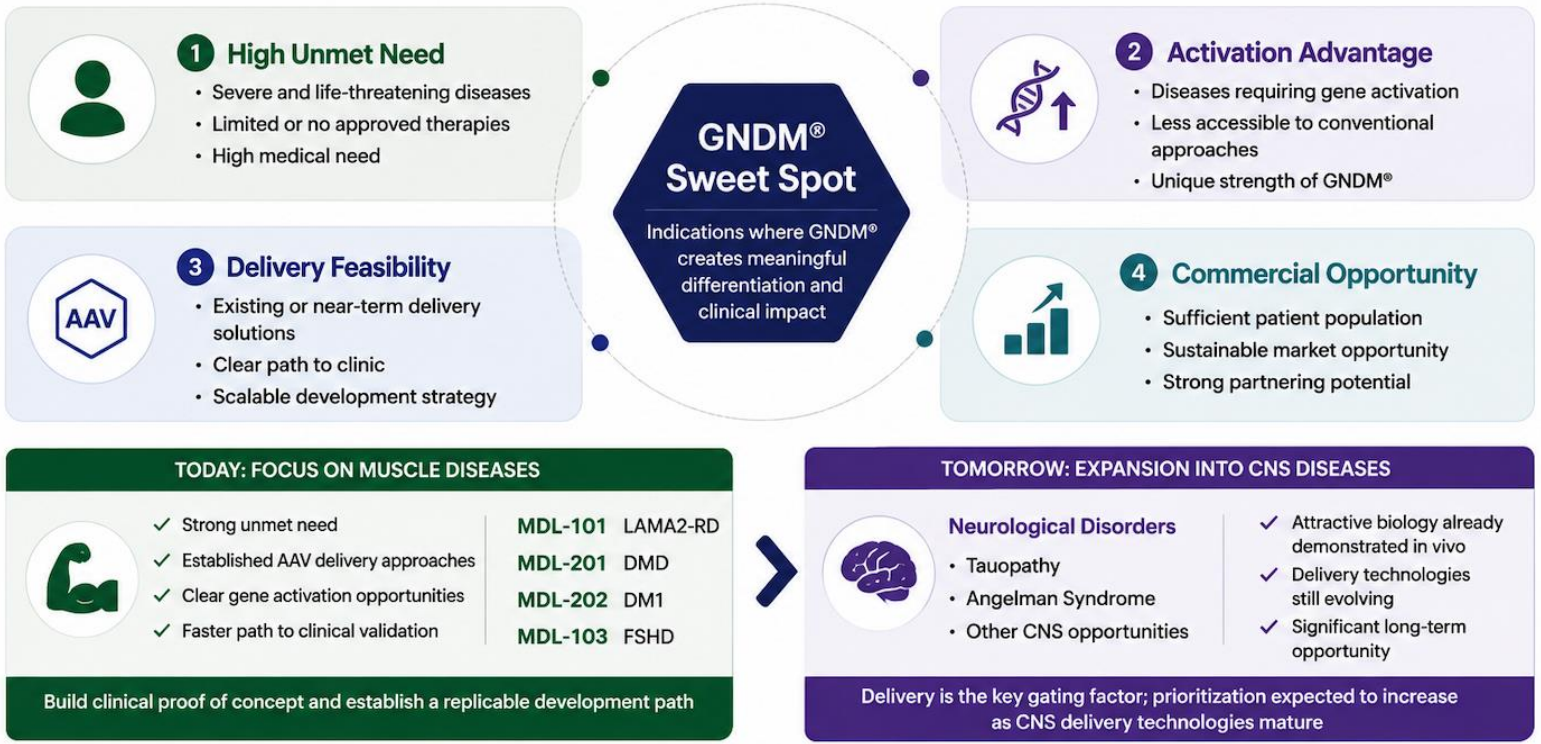
Epigenome editing competitive landscape

The field is gaining more traction and MODALIS is uniquely differentiated from the other epigenome companies

Company	Year Founded	Funding	Platform	Pipeline/Target indication	Stage of Development
MODALIS	2016	Public	CRISPR-GNDM x AAV	MDL-101 for LAMA2-RD gene **activation**	IND enabling
Tune	2020	Series B ($175M, 2025)	DNMT-KRAB fusion dCas9 x LNP	Une-401 for HBV Gene suppression	CTA approval from Hongkong, NZ and Moldova on HBV
nChroma	2021	Merged into nChroma (Dec 2024)	DNMT-KRAB fusion dCas9 x LNP	CRMA-1001 for PCSK9 Gene suppression	P1/2
Epicrispr	2022	Series B ($68M, 2025)	Cas12f-fused with demethylation enzyme x AAVrh74	EPI-321/FSHD Gene suppression	P1/2
Epigenic	2022	Series B ($60M, 2025)	dCas+editor x LNP	EPI-001 PCSK9 for hypercholesteremia Gene suppression	Chinese IIT
Scribe	2018	SerC? ($75M, 2025)	dCas+repressor x LNP	STX1150 PCSK9 for hypercholesteremia Gene suppression	IND enabling
Mammoth	2017	Corp.Minority ($2024, $95M)	miniCas x editor x LNP	MB-111 APOC3 for Triglycemia Gene suppression	IND enabling

Where GNDM® Creates the Greatest Value

We focus on indications where our gene activation technology can deliver the highest clinical impact with a clear path to patients and long-term value

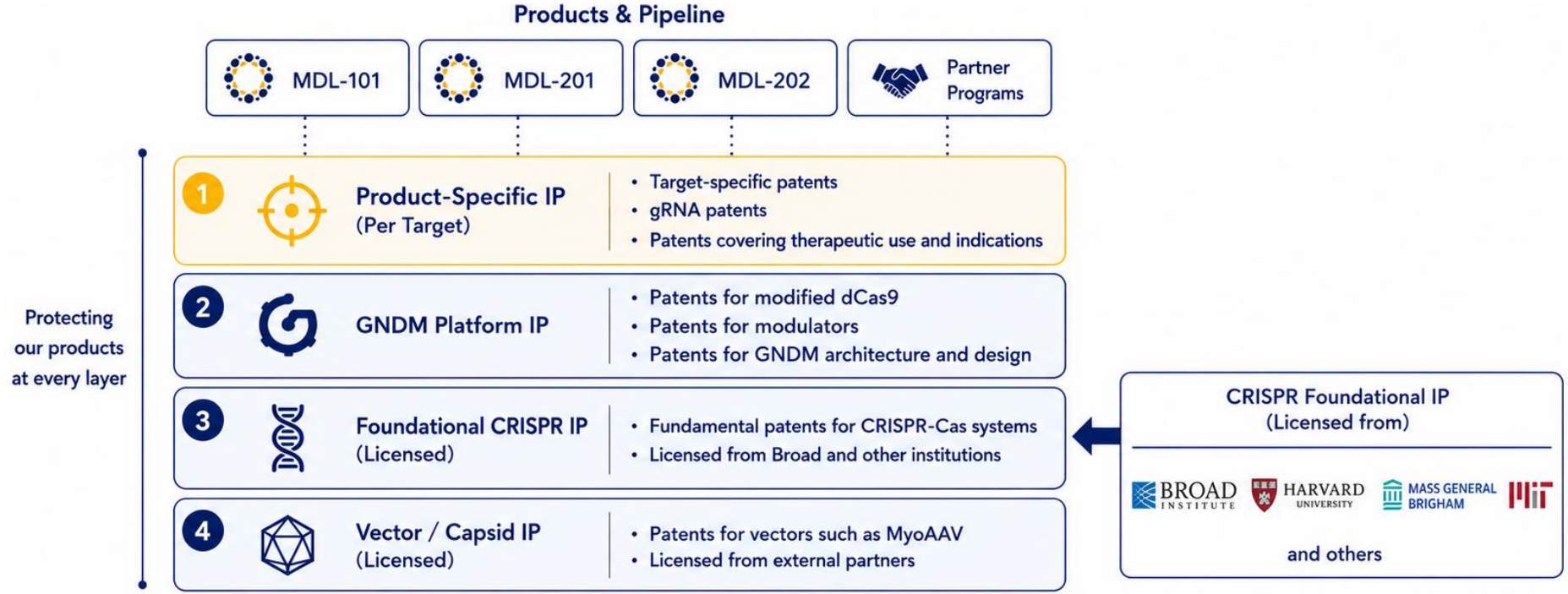


By focusing on the right biology and the right modalities, we maximize the impact of GNDM® today and create the foundation for durable growth and broad patient benefit tomorrow.

MODALIS

Multiple Layers of IP Protection

Build a multi-layered intellectual property portfolio to protect proprietary products based on core technologies

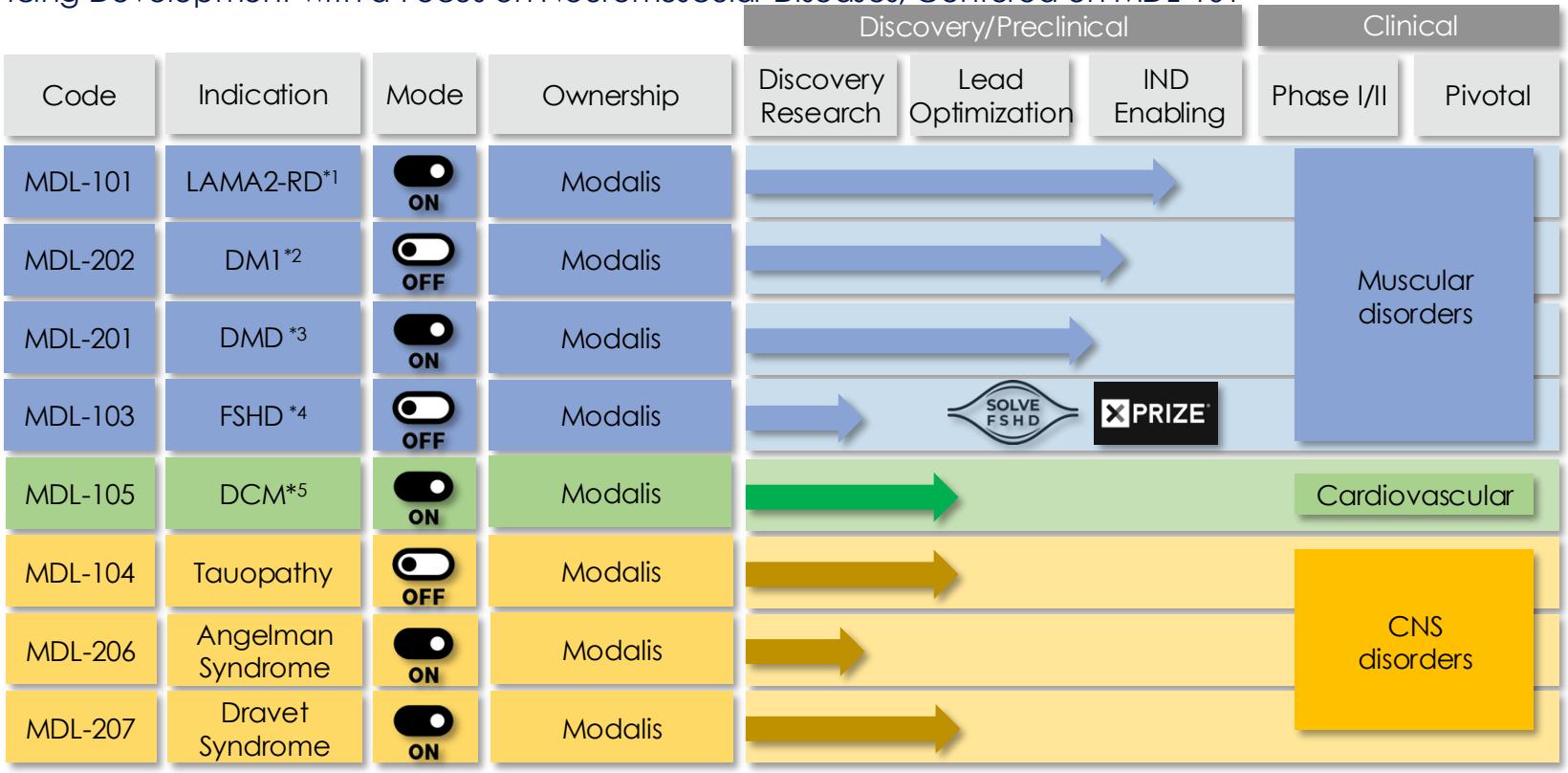


A layered IP portfolio that covers foundational technology and product for our competitive edge

3. Pipeline

Current Pipeline at Modalis Therapeutics

Advancing Development with a Focus on Neuromuscular Diseases, Centered on MDL-101



*1: LAMA2-related congenital muscular dystrophy
*2: Myotonic Dystrophy Type 1
*3: Duchene Muscular Dystrophy
*4: facioscapulohumeral muscular dystrophy
*5: Dilated Cardiomyopathy

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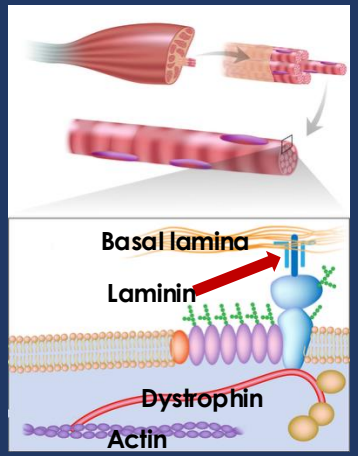
LAMA2-RD (aka CMD1a)

Severe muscular dystrophy caused by loss of function mutation in LAMA2 gene

MDL-101

Potential to be the first LAMA2-RD gene activation therapy



Prevalence	**8.3 in 1 million*** 2500 in US	
Disease Onset	Apparent at birth or within a few months after birth	
Disease Burden	Patients do not survive past adolescence	• Severe muscle weakness • Lack of muscle tone (hypotonia) • Little spontaneous movement • Joint deformities (contractures) • Heart problems and seizures
Disease Causing Gene	**LAMA2 mutation**	
Commercial opportunity	**$500M+**	

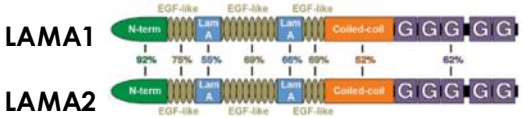
Source: *Estimating the Prevalence of LAMA2 Congenital Muscular Dystrophy using Population Genetic Databases (2023)

Mechanism of Action of CRISPR-GNDM®

Restores muscle function by activating the sister gene LAMA1 in response to LAMA2 mutations

Protein structure of LAMA1 and 2

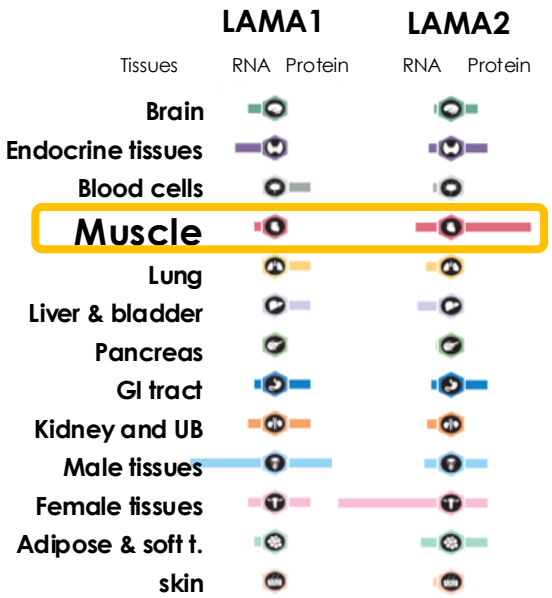
LAMA1 and LAMA2 are sister genes with high sequence homology (approximately 9 kb) that encode similar laminin a chains.



~9kb
(Too big to fit in single AAV)

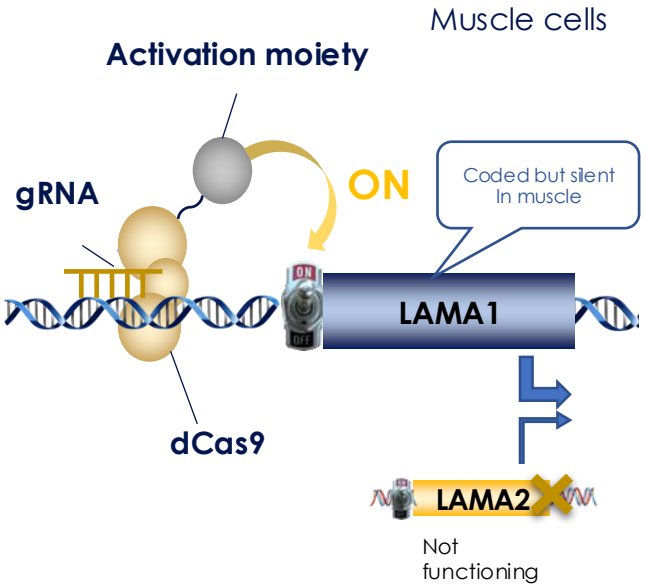
Due to their high homology, LAMA1 can compensate for the LAMA2

Expression pattern of LAMA1and 2 by tissues



LAMA1 is typically silent in muscle cells

Targeted LAMA1 activation using CRISPR-GNDM®



Enabling LAMA1 compensates for the loss of function caused by LAMA2 deficiency

★ CRISPR-GNDM® aims to restore muscle function in patients with LAMA2 deficiency by targeting the sister gene LAMA1

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Publication

Preclinical trial data for our MDL-101 published in *Human Gene Therapy*

Mar 2026

Efficient LAMA1 Gene Activation by Epigenome Editing as a Therapeutic Approach for LAMA2-CMD

Yuanbo Qin, Talha Akbulut, Rajakumar Mandraju, Keith Connolly, John Bechill, Farzaneh Assadian, Claudia Foster, Alison Shottek, Seth Levy, Jamie Benoit, and Tetsuya Yamagata

Modalis Therapeutics, Inc., Waltham, Massachusetts, USA.

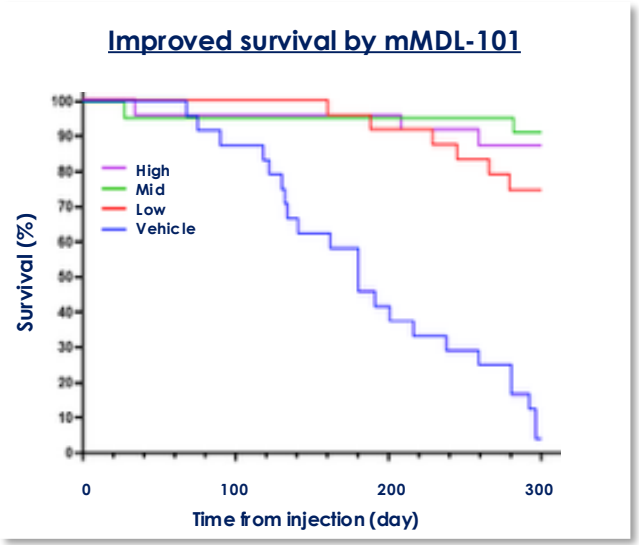
Epigenome editing technology holds great promise for treating diverse genetic disorders. In this study, we demonstrate epigenetic activation of the *LAMA1* gene for the treatment of *LAMA2*-CMD, a severe congenital muscle dystrophy (CMD) caused by biallelic mutations in the *LAMA2* gene. *LAMA1* is a sister homolog that is known to compensate for the function of *LAMA2*. However, supplementing *LAMA1* or *LAMA2* gene via viral platform is not feasible due to the large size of their coding sequences. Through a single administration of our adeno-associated virus (AAV) vector encoding all the necessary elements for epigenetic activation, we observed significant *LAMA1* gene upregulation and phenotype improvements in mouse disease models. The muscle-tropic AAV capsid exhibited desired vector biodistribution and promising pharmacodynamics with good safety profiles in 2-year-old juvenile nonhuman primates (NHPs). Moreover, administration to 8-month-old infant NHPs demonstrated superior pharmacodynamics compared with 2-year-old juveniles, even at half the dose. Our approach holds broad applicability for a range of loss-of-function genetic disorders and could offer a therapeutic breakthrough where active epigenome offers clinical benefit.

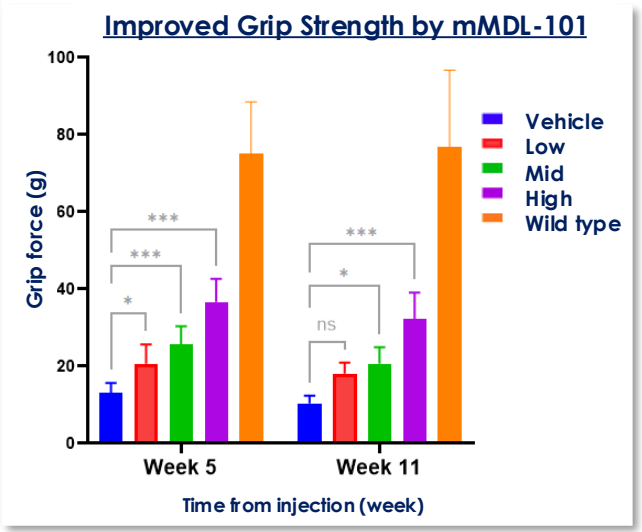
Keywords: gene therapy, epigenetic editing, CRISPR, LAMA1, LAMA2-CMD, MDC1A, AAV

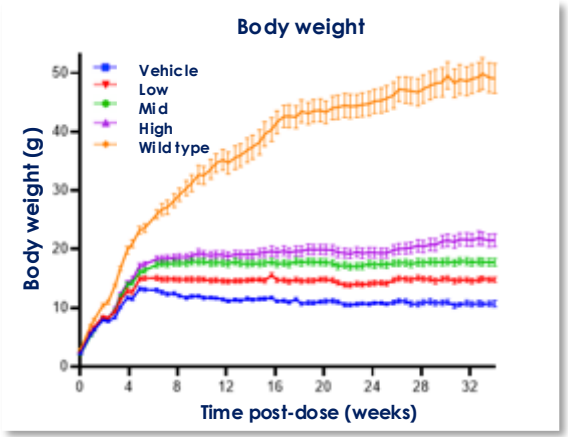
doi: 10.1177/10430342251401873

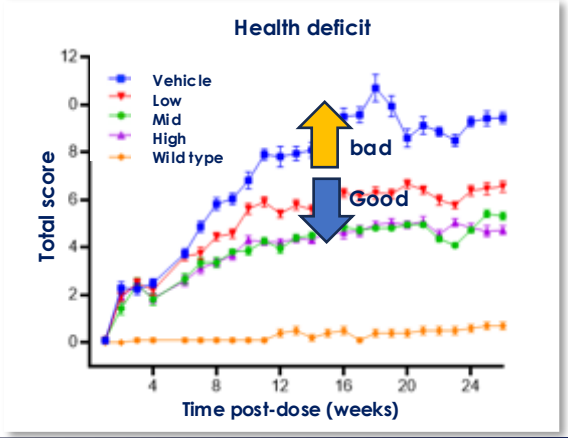
Improvements in body weight, grip strength, and survival in disease model mice

Administration of mMDL-101 significantly improved the clinical condition in patients with LAMA2-RD







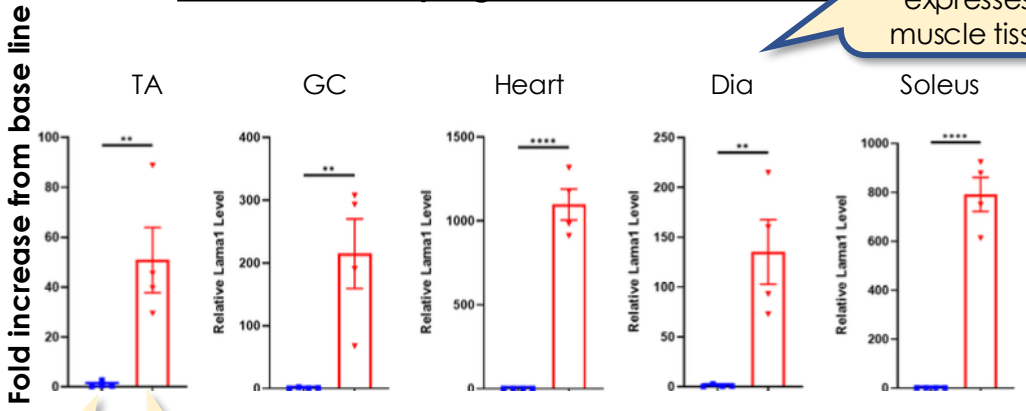


Significant and selective increase of LAMA1

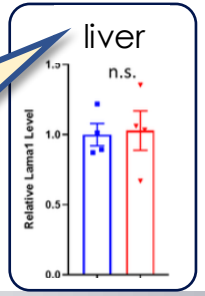
GNDM selectively increases LAMA1 levels in a wide range of muscle tissues

Point #1
LAMA1 is widely expresses in muscle tissues

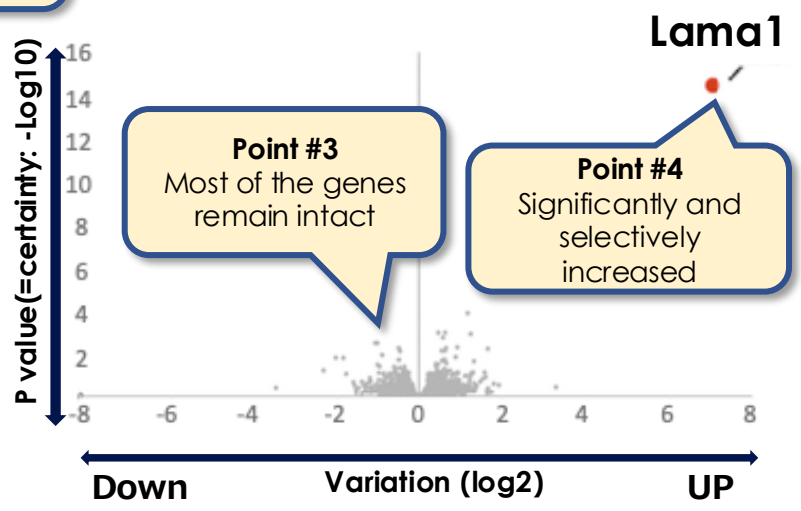
LAMA1 mRNA upregulation in muscle tissue



Point #2
No change in non-muscle tissues



gene perturbation by GNDM-LAMA1 in RNAseq

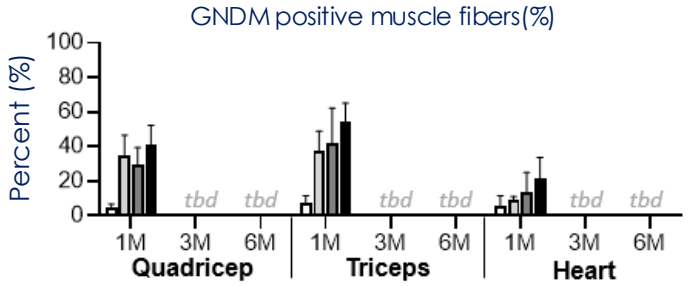


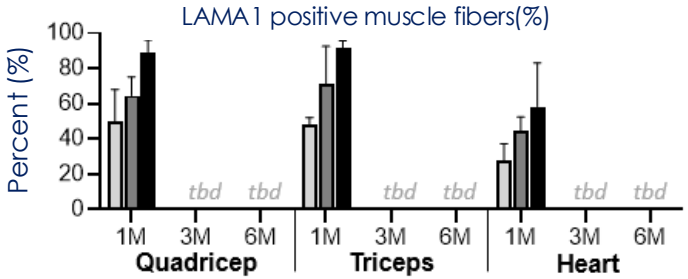
Point #3
Most of the genes remain intact

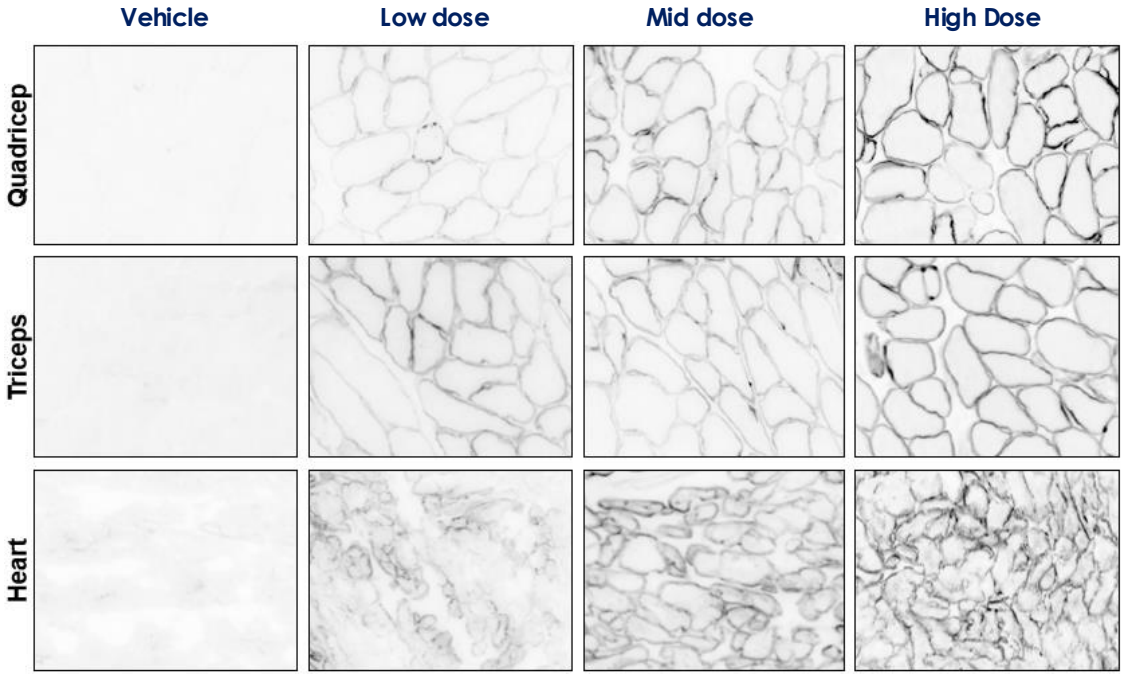
Point #4
Significantly and selectively increased

LAMA1 distribution by MDL-101

Robust, Dose-dependent LAMA1 protein induction in Quad, Triceps, and Heart via GNDM protein expression

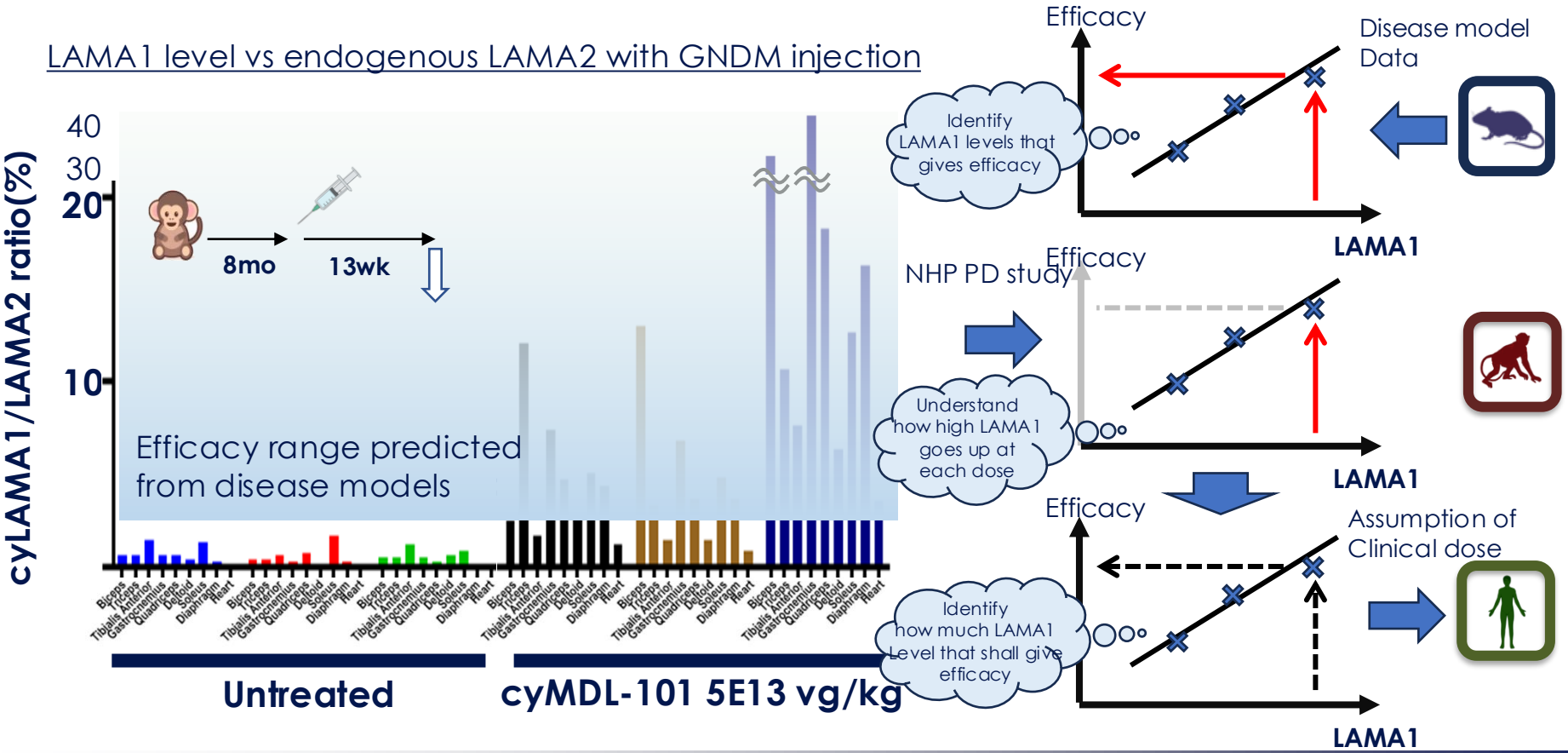






NHP data suggest efficacy in human

Clinical efficacy was estimated based on the therapeutic dose in mice and the level of LAMA1 elevation in monkeys



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Preparations for clinical development are proceeding

Through robust data and systematic execution, aim to ensure safety and efficacy and move into clinical trials asap

4 Key Factors for Initiating a Clinical Trial

1 rodent IND enabling study

- Study Design
- Development of Analytical Methods
- From Dosing to Data Readout and Data Analysis

Verification of biomarkers and efficacy (survival, function, etc.)

2 GLP Tox study

- Study Design
- Development of Analytical Methods
- From Dosing to Data Readout and Data Analysis

Safety and Pharmaco-kinetic/dynamics Evaluation

3 GMP manufacturing

- Process development
- Analytical method development
- GMP manufacturing
- Release tests

Establishing a system to reliably supply clinical vectors

4 Clinical site preparation

- Selection of Potential Clinical Trial Sites and PIs
- Interviews → Selection
- Protocol Coordination
- Logistics Confirmation

Recruitment and Alignment with Clinical Trial Sites

Development road map (provisional)

Achievements to Date (by 2Q 2026)

- ✓ Verification of efficacy and target engagement (Mice and Monkeys)
- ✓ Verification of Long-Term GNDM and LAMA1 expression in Mice
- ✓ Verification of Safety and Efficacy in juvenile NHP studies
- ✓ Efforts to Establish Manufacturing Processes and Improve Quality
- ✓ Preparation of Clinical Protocols
- ✓ Selection of Primary Clinical Trial Sites

GLP tox study (ongoing)

- ☐ Data readout
- ☐ analysis

Manufacturing (ongoing)

GMP campaign

- ☐ Plasmid release in 3Q
- ☐ AAV: release in 4Q

IND filing to FDA Late 2026 ~ early 2027

Preparation of Data Package → Submission of IND Appl. → Clearance

FIH clinical trial (1H 2027)

Start of the FIH Trial in US
Patient Recruitment
FPFD*

Ensuring Safety
Robust nonclinical data and thorough risk management

Systematic Execution
Steady Development Progress Based on Clear Milestones

Contributing to Patients
With the goal of delivering safe and effective treatments as soon as possible

Long-Term Value Creation
Driving Sustainable Growth in Corporate Value Through Innovative Treatments

★ Steadily achieving key milestones toward clinical trial and aim to enter clinical trials asap

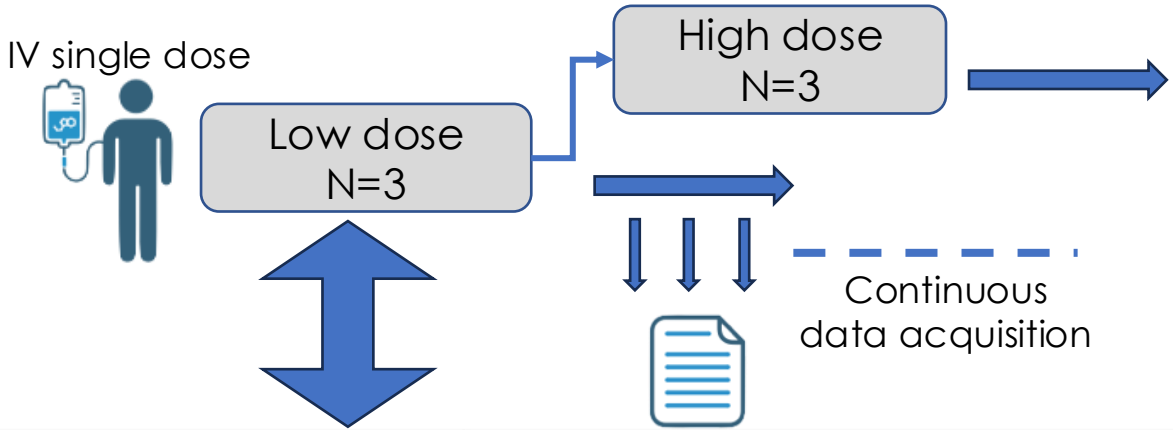
FPFD: First patient first dose(最初の患者さんへの最初の投与)

MDL-101-001 Trial design

Open-label trial with two doses. Efficacy evaluated in comparison with natural history observation trial.

- Patients ≤ 5 years.
- Genetically confirmed, biallelic pathogenic *LAMA2* mutations.
- Severe LAMA2-RD clinical phenotype. Difficulty with independent walking or sitting.
- AAV9 TAb negative.

Phase1/2 Open-label dose escalating trial



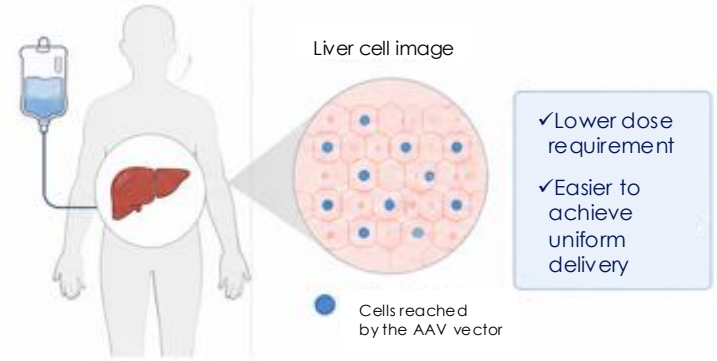
Retrospective	Natural History
Hinkley *et al.*, 2024 *(NCT04299321)* • 60 LAMA2-RD < 5 years old	**NCT06132750** (LAST STRONG) • 40 (est.) LAMA2-RD or SELENON-RM patients • Est. completion Sept 2026
Zambon et al., 2020 • 42 LAMA2-RD < 18 years old • 8 'complete deficient' < 5 years old	**NCT06354790** (Institut de Myologie, France) • 60 (est.) LAMA2-RD 2 to 15 years old • Est. completion Jan 2028
	NCT06503367 (READY CMD LAMA2) • 44 (est.) LAMA2-RD < 5 years old • Est. completion Sept 2028

Why does development take time?

In the field of muscular dystrophy, achieving systemic delivery, long-term expression and safety requires extensive technical validation and optimization

Delivery to a specific organ (e.g. liver)

Because the target is limited to single organ, the required vector dose is relatively small

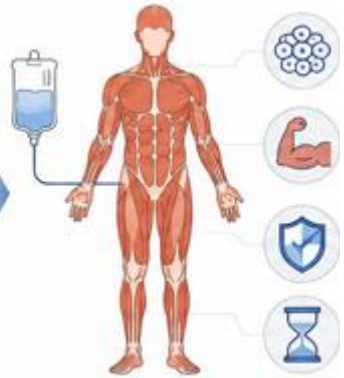


- ✓Lower dose requirement
- ✓Easier to achieve uniform delivery

VS

Systemic delivery to all skeletal muscles (challenge in muscular dystrophy)

Because the target is large muscle mass throughout the body, the required vector dose is extremely high.



Large amount of Muscle tissue
→require a very high vector dose to reach the entire body

Wide distribution of tissues
→Must reach the heart, diaphragm, limb muscles, and all skeletal muscles throughout the

Safety risks at high dose
→Management of inflammation, immune responses, and other safety aspects in critical

Need for long-term expression
→A single dose must provide long-lasting efficacy

- ✓ Vector dose requirement is extremely high
- ✓ Harder to achieve uniform delivery
- ✓ Ensuring safety while maintaining long-term efficacy

Therefore, development requires advanced technology, extensive validation, and optimization, which take time

Key technical requirements in muscular dystrophy gene therapy

- **Delivery efficiently to all muscles throughout the body** (High efficiency & broad distribution)
- **Ensure stable long-term expression with a single dose** (durable efficacy)
- **Design for safety at high vector doses** (immune & inflammation management)
- **Establish appropriate measurement and evaluation system** (biomarkers endpoints)

Our goal is not merely "early IND filing", but to maximize the probability of long-term success and the ultimate value of our therapeutic products

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However, once we get past the first hurdle, the highway will be built.

The MDL-101 is both a product development project and a validation of the shared platform.

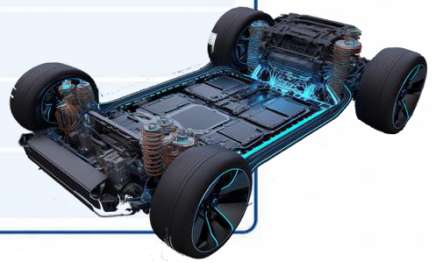
Platform Value

Success in MDL-101 builds confidence in the platform and accelerates the development of additional programs.

MDL-101 (LAMA2-RD)
First Clinical Program

Shared CRISPR-GNDM® Platform
Common technologies and capabilities

- MyoAAV Capsid
- Muscle-specific Promoter (CK8)
- CRISPR-GNDM® Payload
- Manufacturing Process
- Safety Package
- Regulatory Learnings



MDL-201 (DMD)
Duchenne Muscular Dystrophy

MDL-202 (DM1)
Myotonic Dystrophy Type 1

MDL-103 (FSHD)
Facioscapulohumeral Muscular Dystrophy

Additional neuromuscular programs leveraging the same platform

MDL-101 validates the shared platform and de-risks subsequent neuromuscular programs

MDL-101 is potentially the first upregulating epigenome-editing therapeutic program to enter the clinic

01

CRISPR-GNDM efficiently targets and upregulates LAMA1 gene

02

STRONG ANIMAL POC CONFIRMED IN MICE *IN VIVO* INCLUDING PROLONGED SURVIVAL AND FUNCTIONAL IMPROVEMENT

03

THE PROCESS TO MANUFACTURE THE MOLECULE WITH MUSCLE-SPECIFIC VECTOR HAS BEEN ESTABLISHED WITH FEASIBLE PRODUCTIVITY,

04

THE INJECTION OF THE PRODUCT DID NOT CAUSE DETRIMENTAL SAFETY ISSUES IN MICE AND NHPS

05

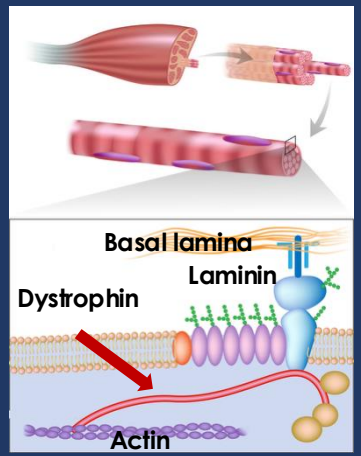
US FDA'S ODD AND RPDD DESIGNATED

Duchenne Muscular Dystrophy (DMD)

A type of muscular dystrophy caused by mutation in Dystrophin gene

MDL-201

Potentially best-in-class molecule by rebooting UTRN gene expression by GNDM



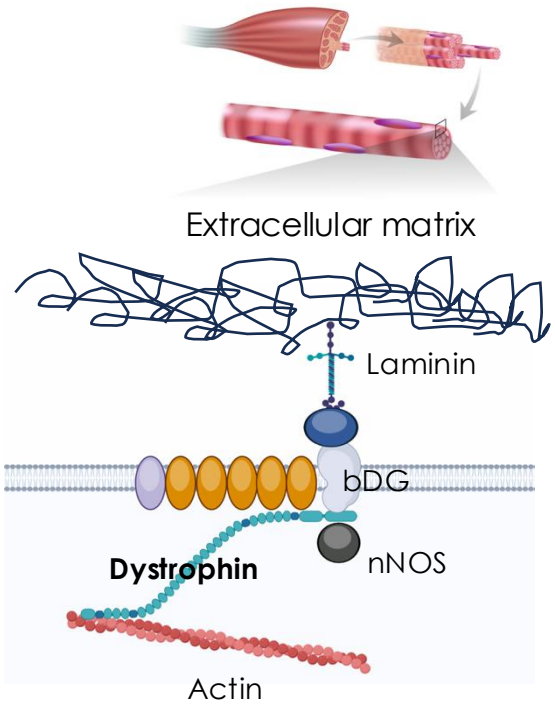
Prevalence	**1 in 3,500 to 5,000 male newborns**	Relatively high in genetic disorders
Disease onset	most commonly appears **between 3 and 6 years old**	
Disease Burden	Most severe clinical symptoms of all the muscular dystrophies including muscles weakness and atrophy	Motor development begins to slow in early childhood and muscle weakness progresses, followed by cardiomyopathy, scoliosis, and respiratory complications
Cause of disease	Disruption or mutation in Dystrophin gene	Loss of dystrophin and abnormal histological development of muscle necrosis and regeneration
Market size	**US$4.1B** in 2026 to **US$9.6B** by 2031 (CAGR 18.8%)#	Expected to grow at CAGR=42.5% with approval of new therapeutics

*Source: https://doi.org/10.1212/WNL.0000000000011425 #Mordor Intelligence, 2026

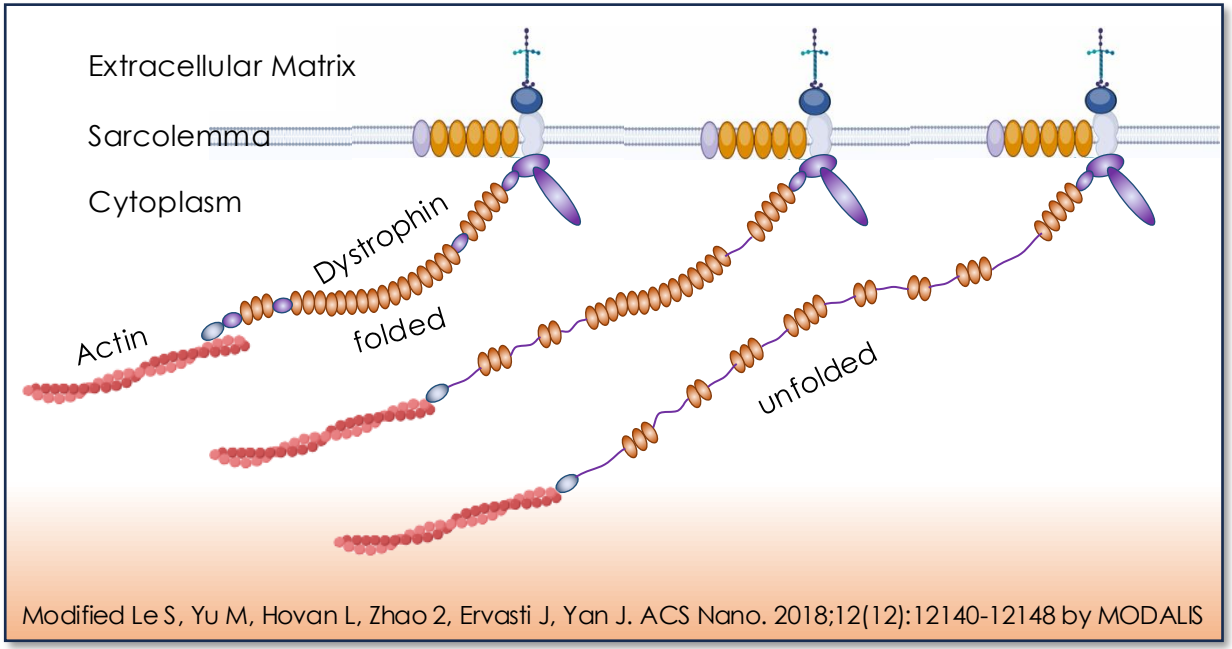
Dystrophin's function

Functions as a shock absorber and signal transmitting molecule in muscles

Dystrophin location



Dystrophin stretches and contracts to connect the cell membrane and actin



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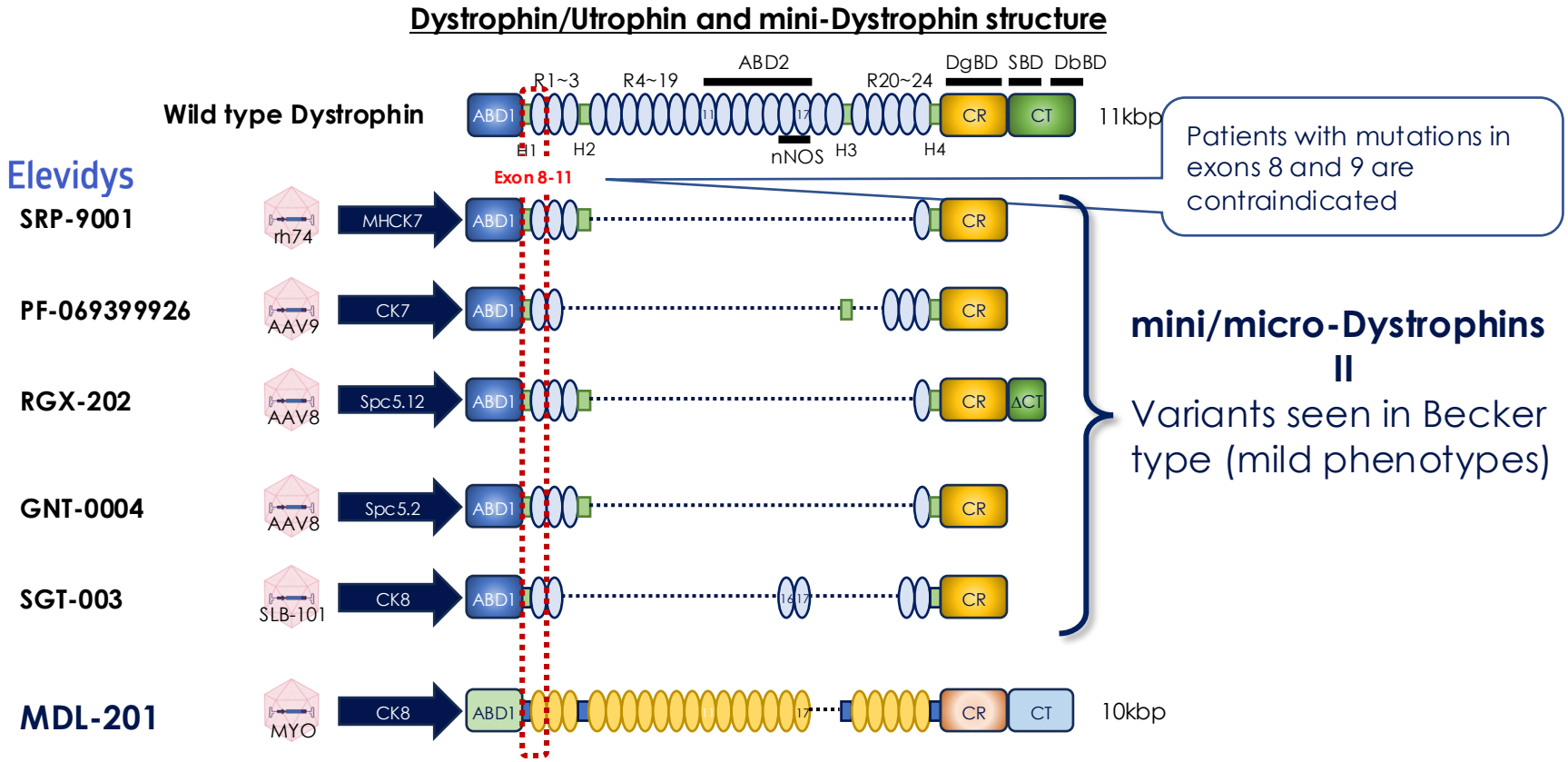
GNDM and micro-Dystrophins payloads comparison

Due to size constraints, small dystrophin derived from Becker patients is used for GTx



Modified from Crudele &Chamberlain, 2019

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Reactivating Utrophin to compensate for Dystrophin loss

Utrophin is naturally active during early development and can compensate for dystrophin deficiency when reactivated

1. Developmental Gene Switch

During normal development, a natural switch occurs from **Utrophin** to **Dystrophin**

Neonate (fetal/early life)

Utrophin ON

Dystrophin OFF

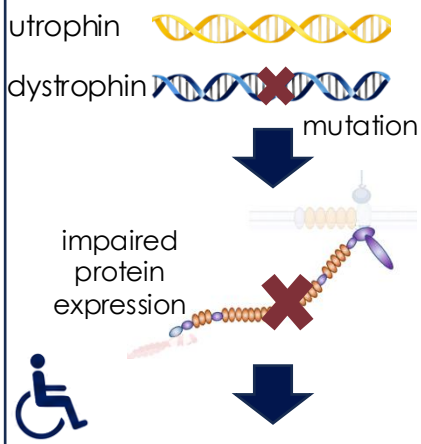
Childhood

Utrophin OFF

Dystrophin ON

2. Disease Mechanism in DMD

When a mutation exists in the **Dystrophin** gene, The natural switch leads to disease



Loss of muscle integrity
Progressive muscle degeneration

3. MDL-201 Treatment Concept

MDL-201 reactivate endogenous **Utrophin** to compensate for the loss of **Dystrophin**

In DMD patients

Utrophin OFF

Dystrophin ON

MDL-201 CRISPR-GNDM®

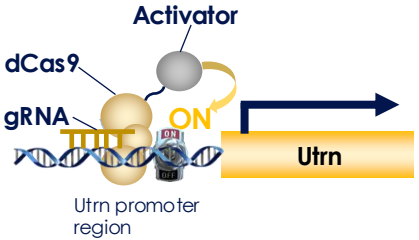
Utrophin ON

Dystrophin ON

Functional compensation
Restore of muscle function

4.. MDL-201 Mechanism of Action

MDL-201 upregulates **Utrophin** expression via epigenetic modulation



Utrophin ON
expression

MDL-201 restores muscle function by reactivating endogenous Utrophin, a natural functional substitute for dystrophin

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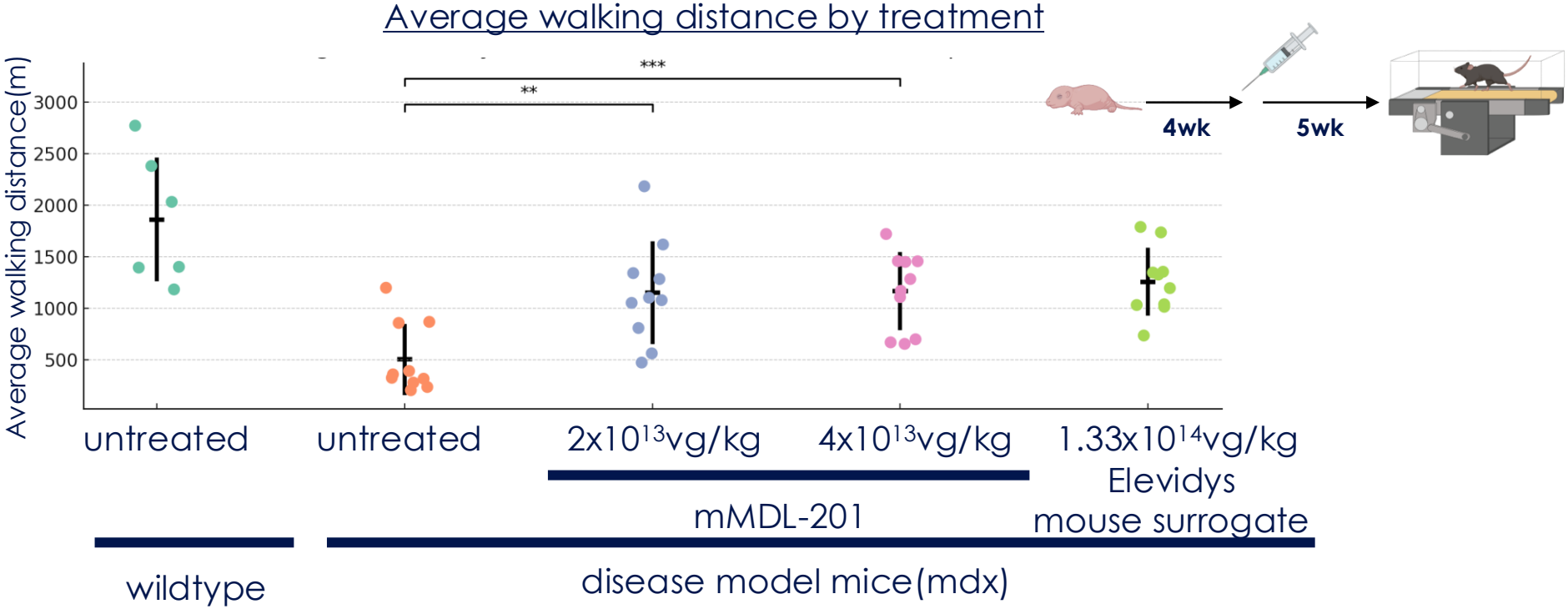
Functional improvement of DMD by MDL-201

achieves the same level of efficacy as the benchmark drug at a dose one order of magnitude lower



Data is presented as mean ± SEM. Normality was assessed using Shapiro-Wilk tests for all treatment groups. Post-normality test, unpaired t-tests were performed between the BL10 Vehicle and MDX Vehicle groups for both A and B (### p<0.001). Non-parametric ANOVAs (Kruskal-Wallis tests with Dunn's post-hoc test for multiple comparisons) were performed to compare all treatment groups against the MDX Vehicle (**p<0.01; ***p<0.001).

MDL-201 potentially is a best-in-class DMD GTx that surpasses micro/mini-dystrophin approaches

01

Utrophin retains functional domains that are absent in µ-dystrophin

02

MDL-201 shows higher functional improvement than µ-Dystrophin in mouse pathological models

03

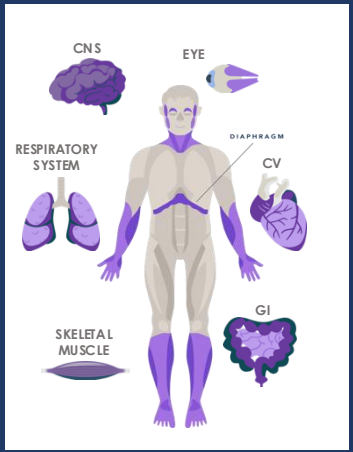
MDL-201 shares the same platform as MDL-101, on which much experience has been accumulated

Myotonic dystrophy type 1(DM1)

extension of CTG repeat in 3' UTR of DMPK gene

MDL-202

Potential to be the first-in-class and the first DM1 treatment



Prevalence	**1-4.8 in 10,000** (1 in 2,300*)	DM is the most common muscular dystrophy among adults of European ancestry
Disease onset	DM1 can occur from birth to old age	Age at onset is between 20 and 70 years (typically onset occurs after age 40)
Disease Burden	muscle weakness and wasting (atrophy), myotonia	DM causes weakness of the voluntary muscles, although the degree of weakness and the muscles most affected vary greatly according to the type of DM and the age of the person with the disorder
Cause of disease	Microsatellite expansion in 3' UTR of DMPK gene	Extended CTG repeat capture MBNL1 protein which is essential for normal splicing
Market size	**$2.2B #** By 2032	$80M market as of 2022 without any treatment but is expected grow

*Source: Myotonic Disease Foundation

DelveInsight (including both DM1 and DM2)

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Molecular Pathogenesis of DM1 and MDL-202 mechanism of action

An abnormally elongated CUG traps splicing proteins and impairs their function

What is DM1?

In DM1 patients, abnormal expansion of CUG repeats in the DMPK gene causes myotonic dystrophy type 1.

What causes it?

Expanded CUG repeats in the DMPK gene produce toxic RNA that binds and sequesters MBNL proteins.
This leads to widespread splicing defects in many genes, triggering various organ dysfunction including muscles.

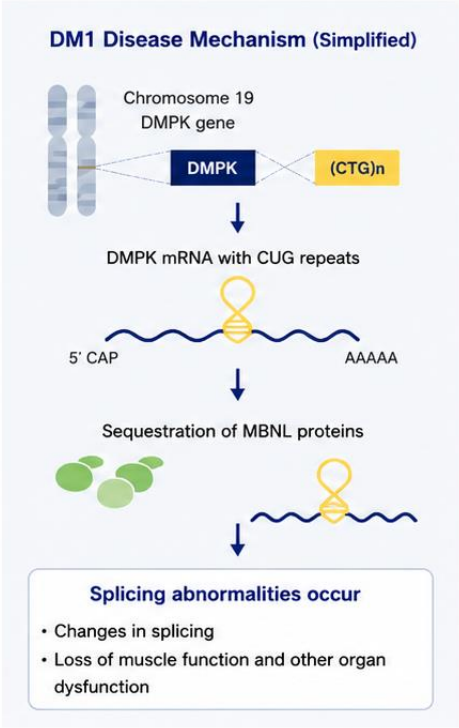
CUG Repeat Length and Disease Severity (Examples)

50–150	50–1,000	>800	>1,000
Associated with mild or non-disease (one-allele carriers)	Associated with classic or adult-onset DM1	Associated with juvenile-onset DM1	Associated with congenital-onset DM1

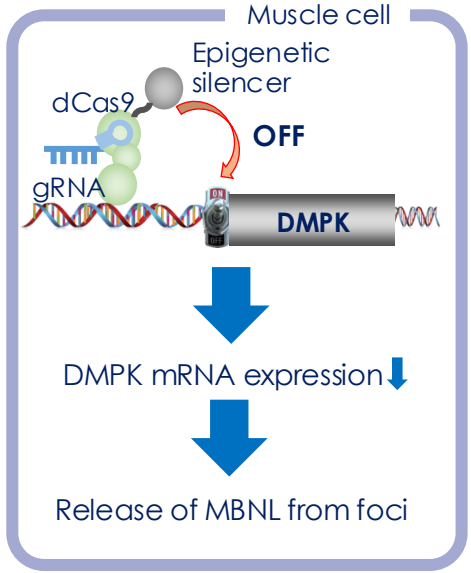
Longer repeats → Earlier onset and more severe disease

What happens as a result?

Toxic RNA binds to MBNL proteins and reduces their function.
This causes splicing abnormalities across a wide range of genes, leading to muscle dysfunction and other symptoms.



CRISPR-GNDM® targeting DMPK

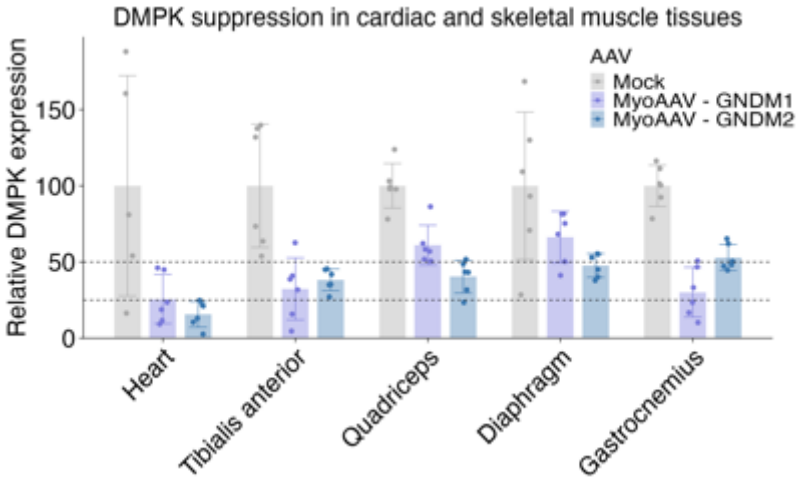


By targeting the root cause of DM1 and restoring normal splicing, we aim to improve muscle function and patients' quality of life.

Strong DMPK Suppression Across Multiple Muscle Tissues

GNDM exhibited a greater DMPK suppression compared to vectorized RNA in all muscle tissues

MyoAAV - GNDM suppression of DMPK



8 wks dosing at 1e14 vg/kg to DMSXL mouse model (n=6)
8 wks post-injection harvest

Why we believe these findings are important

- ✅ **Strong suppression in disease-relevant tissues**
  High DMPK suppression observed in heart and diaphragm, which are important tissues in DM1 pathology
- ✅ **Broad muscle distribution**
  Suppression observed across multiple skeletal muscle groups
- ✅ **Potential advantage over other one-time treatment approaches**
  Demonstrated stronger DMPK suppression compared with previously reported vectorized RNA-based approaches in multiple muscle tissues
- ✅ **Mutation-independent approach**
  Potential applicability across a broad range of DM1 patients regardless of repeat length
- ✅ **Synergy of capsid and GNDM**
  Results support the combination of muscle-targeted capsid delivery and CRISPR-GNDM® regulation

Estimated %DMPK suppression	Heart	Diaphragm	Quadriceps	Tibialis Anterior	Gastrocnemius
GNDM2-MyoAAV	87%	52%	60%	63%	49%
Sanofi '268	~76%	~32%	~25%	~40%	~35%

A benchmark of vectorized siRNA

Source: Molecular Therapy Vol. 33 No 12 Dec 2025; Dose = 9e13, N=10-12

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MDL-202 holds significant potential as a GTx for DM1, a disease affecting many patients

01

Directly acts on the DMPK gene at the DNA level, which is the mechanism of onset for DM1

02

Significant DMPK inhibition has been confirmed in the disease model

03

Access to a relatively large patient population even for muscle disorders

Facioscapulohumeral Muscular Dystrophy (FSHD)

A type of muscular dystrophy caused by impaired Dux4 gene expression

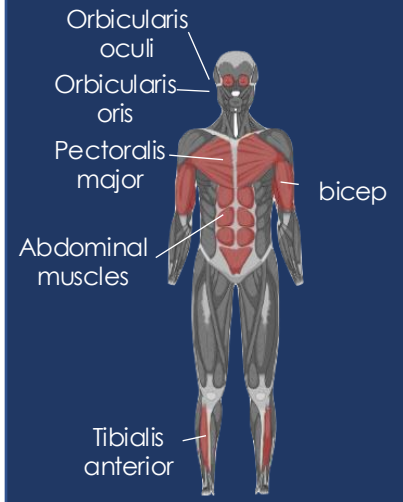
Funded by

SOLVE FSHD

XPRIZE

MDL-103

Potentially first-in-class treatment by silencing expression of toxic Dux4 gene product



Prevalence	**1 in 10,000-20,000**	Muscular dystrophy most frequent in adults
Disease Onset	Often not recognized until the 20s and tends to worsen during adolescence	Progression of disease to face, shoulders, and arms is generally slow
Disease Burden	weakness of the facial muscles, the stabilizers of the scapula, or the dorsiflexors of the foot	Symptoms of asymmetrical (unbalanced) muscle weakness Visual impairment, vascular abnormalities, hearing impairment, etc.
Disease Causing Gene	Over expression of Dux4 gene	DUX4 is originally expressed in germline cells but need to be suppressed in somatic cells
Commercial opportunity	**$500M+**	

Source: https://doi.org/10.1212/WNL.0000000000011425
Orphanet, Raymond A. Huml MD A concise guide

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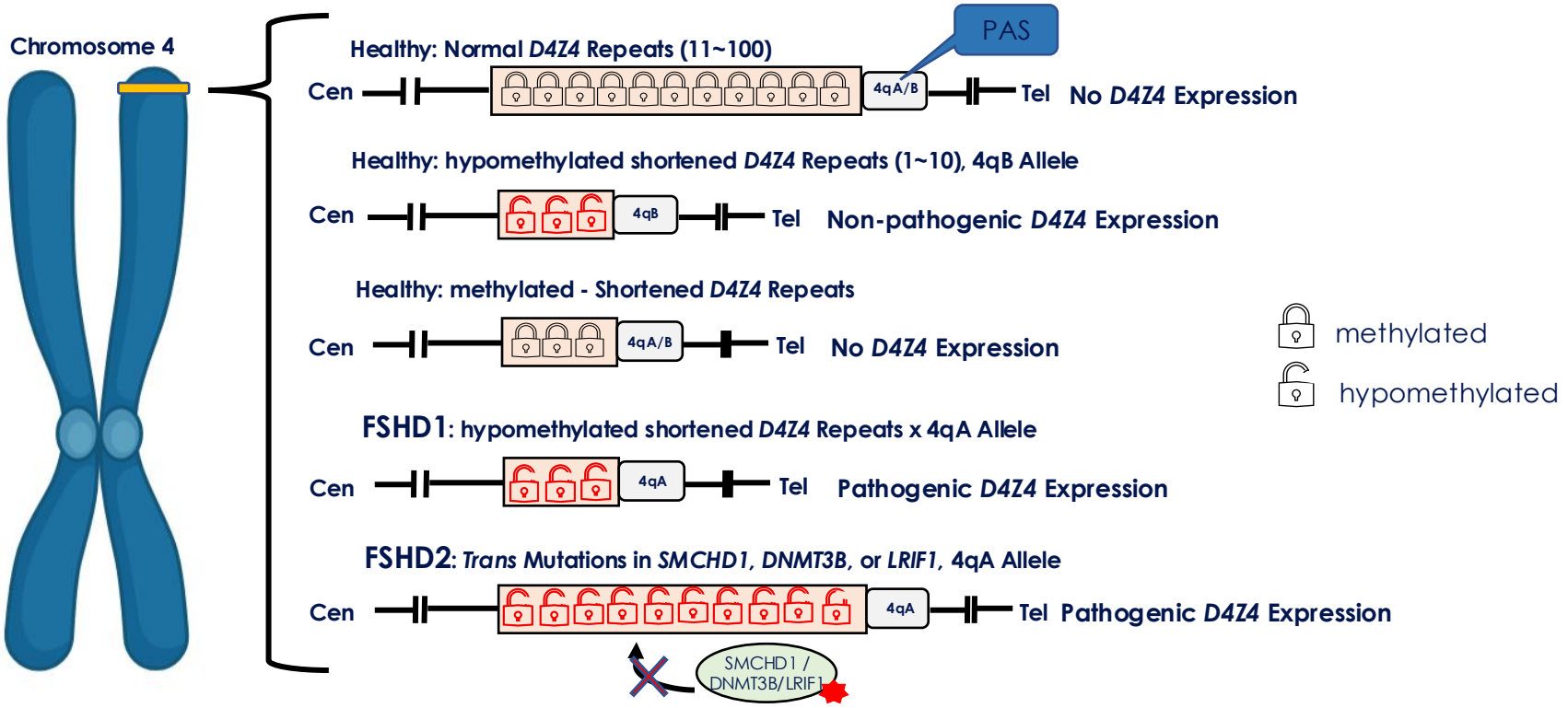
FSHD disease mechanism

Dux4 expression causing toxicity to skeletal muscle due to epigenetic changes leading to a state of disinhibition

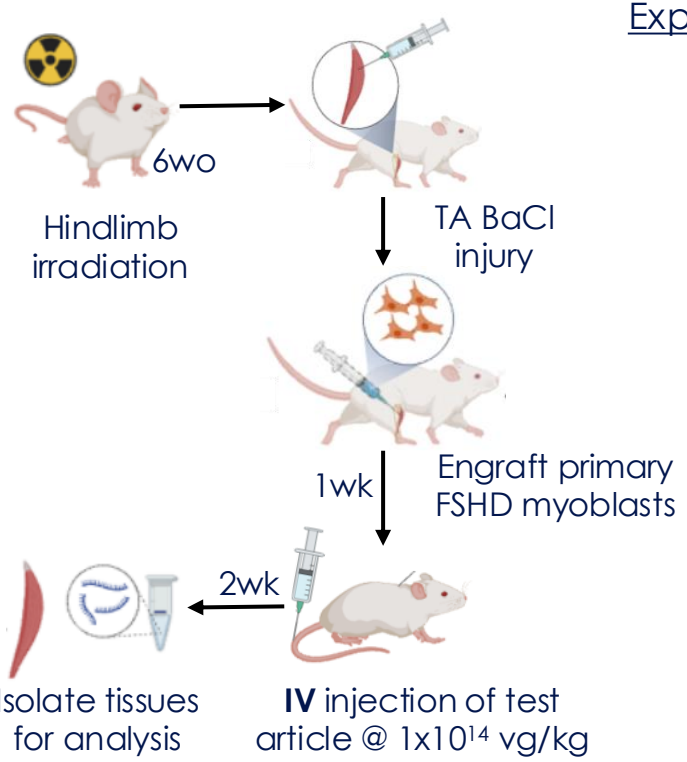


DeSimone et al. 2020, *Dis Model Mech*

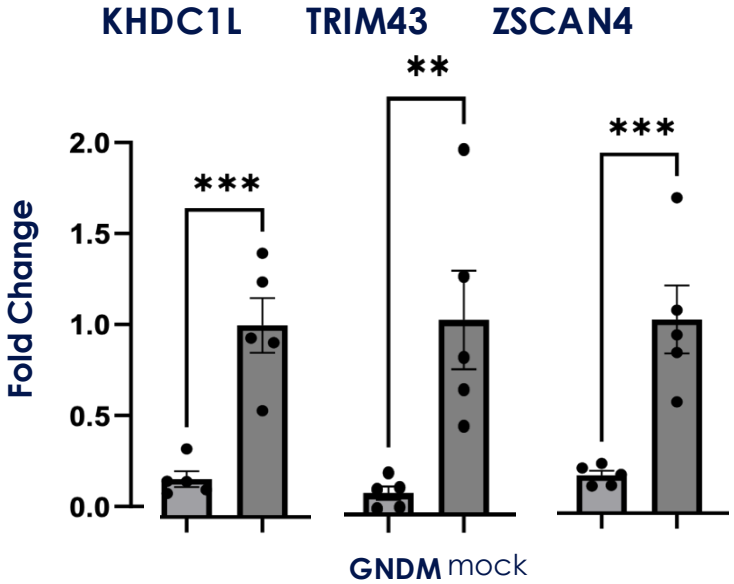
PAS: polyadenylation signal

Clear efficacy in a FSHD disease model

The DUX4 downstream gene was even strongly suppressed by systemic administration



Expression of DUX4-Target Genes In Xenografted human muscle



- iv injection
- N=5 Normalized to hRPL13A
- Statistical significance by 1-way ANOVA with Dunnett's test

MDL-103 offers a reasonable development potential as an FSHD gene therapy

01

A target with clearly demonstrated causal relationships between disease and gene

02

Powerful suppression of the DUX4 pathway has been clearly demonstrated in animal disease models

03

Development is supported by funding from multiple external grants

Future Expansion Opportunities Enabled by CRISPR-GNDM®

Future value creation opportunities with the potential for application to a wide range of diseases

Platform Demonstration Using the MDL-101 | Verification of Delivery Technology

MDL-105 Titin Activation

Dilated Cardiomyopathy (DCM)

Target
Activation of the TTN (Titin) gene

Scientific Rationale
TTN is critical for cardiac structural integrity, and its dysfunction is a major cause of DCM

Current Status
TTN activation demonstrated in muscle cells and animal models

Future Opportunity
Potential to become a novel therapy targeting large genes such as TTN

MDL-103 Tau Suppression

Alzheimer's Disease

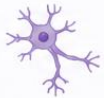
Target
Suppression of the MAPT (Tau) gene

Scientific Rationale
Tau accumulation is strongly linked to disease progression; CRISPR-GNDM® potently suppresses Tau

Current Status
Robust Tau suppression observed in animal models via ICV administration; BBB delivery remains a key challenge

Future Opportunity
Evaluation of BBB-penetrant AAV capsids and partnership-driven CNS delivery strategies

MDL-205 UBE3A un-silencing



Angelman Syndrome

Target
Activation of the UBE3A gene

Scientific Rationale
Loss of maternal UBE3A is the underlying cause; GNDM can reactivate the silent allele

Current Status
UBE3A activation confirmed in neuronal cells and animal models

Future Opportunity
Aiming to develop a disease-modifying therapy for a rare neurological disorder with high unmet need

MDL-207 SCN1A Activation

Dravet Syndrome

Target
Activation of the SCN1A gene

Scientific Rationale
Loss-of-function SCN1A mutations drive disease; activating SCN1A alleviate symptoms

Current Status
Activation of SCN1A from healthy allele leads to longer survival and less epilepsy in disease model mice

Future Opportunity
Potential to address a severe epilepsy with limited treatment options

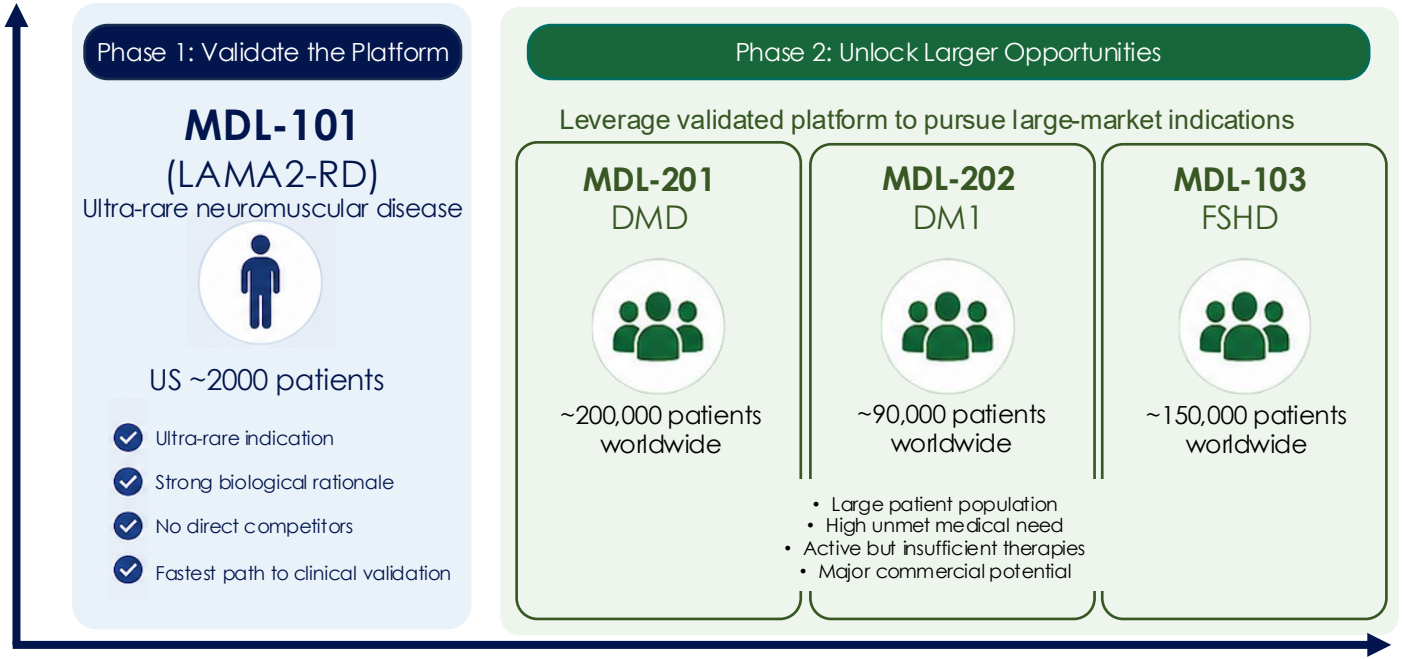
These programs aim to further expand the platform value of CRISPR-GNDM® and serve as a source of medium- to long-term growth and diverse value creation.

4. Where we are and the Growth Strategy

Growth Opportunity Expands Along Two Dimensions

Growth is driven by two levers: development progression of our lead program and expansion into larger indications

Pipeline Expansion
Expand into larger patient populations and new indications



Development Progression Advance clinical development and de-risk to unlock the next wave of programs

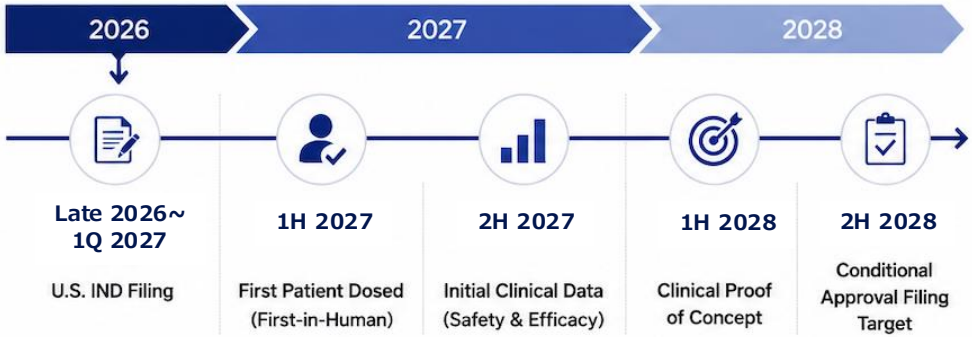
Clinical validation of MDL-101 is expected to reduce development risk, improve funding access, and accelerate expansion into larger indications.

Development Priorities and Key Milestones

Focus on advancing MDL-101 to deliver early clinical value and validate GNDM and will expand our pipeline through partnerships, funding and internal resource growth

Lead Program: MDL-101 (LAMA2-RD)

U.S. IND filing planned for late 2026 to 1Q 2027



Why MDL-101 Is Our Top Priority

- Early clinical validation of GNDM
- Establish development and regulatory pathway
- Enable expansion into larger opportunities

Pipeline Expansion Opportunities

Preclinical proof of concept established

- **MDL-201 (DMD)** — Duchenne Muscular Dystrophy
- **MDL-202 (DM1)** — Myotonic Dystrophy Type 1
- **MDL-103 (FSHD)** — Facioscapulohumeral Muscular Dystrophy
- **MDL-104 (Tauopathy)** — Tauopathies
- **MDL-206 (Angelman)** — Angelman Syndrome

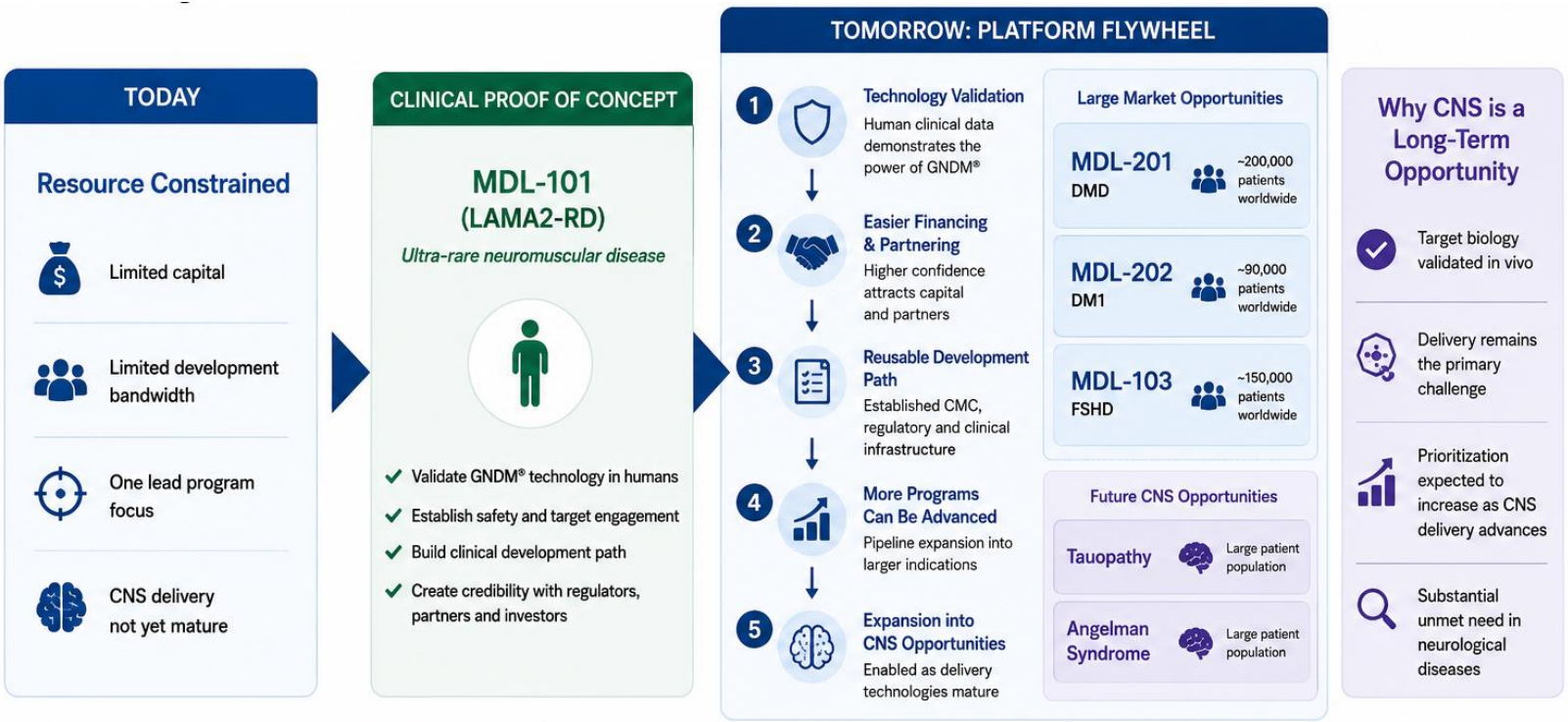
Future advancement expected through:

- Partnering
- Grant funding
- Internal resource expansion

Advancing MDL-101 is our best path to value creation. We will use partnerships and capital to accelerate the expansion of our muscle and CNS pipeline.

From MDL-101 Validation to Broad Platform Expansion

Clinical proof of concept of MDL-101 will unlock financing, partners, and bandwidth to advance a diversified pipeline and create long-term value



MDL-101 is the key to unlock our next phase of growth-from platform validation to leadership across multiple large-market and CNS indications

Opportunities for Value Creation in Line with the Development Progress of MDL-101

The impact of reselling RPVs after the commercialization approval in US will be significant

*The following is an example scenario based on certain assumptions and does not constitute a financial forecast.

NOW

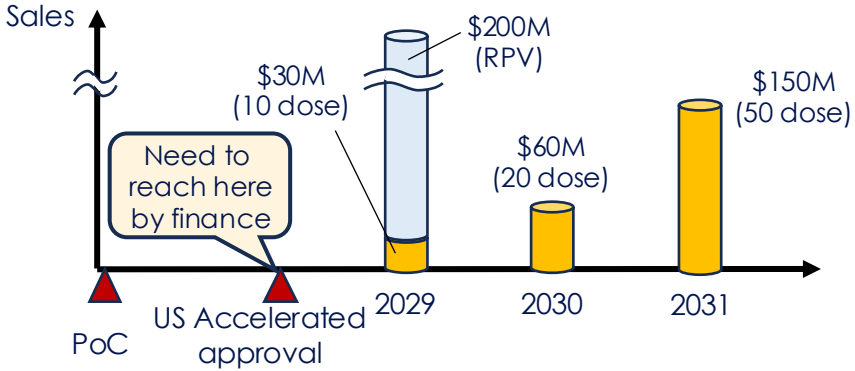
scenario

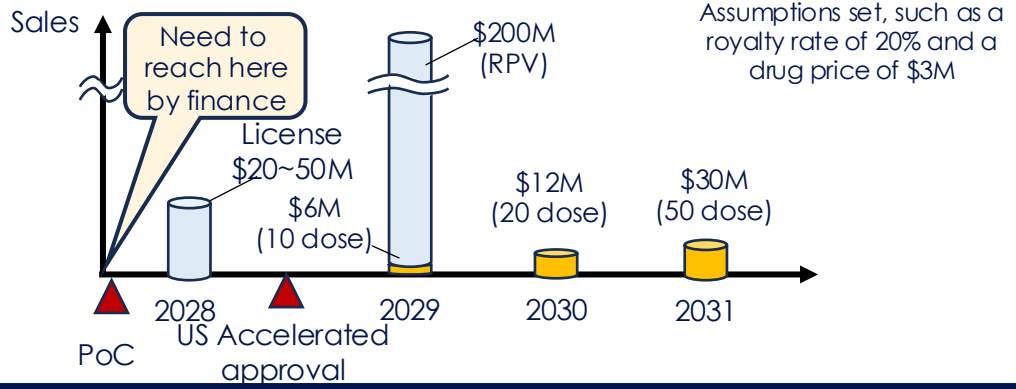
Direct Sales

- Maximize future sales
- Bear development and sales costs in-house
- Significant need for additional funding

Partnering

- Secure non-dilutive funding at an early stage
- Share development and sales risks
- Future revenue is limited to royalties, etc.





The funds raised will be used to advance the clinical development of MDL-101 toward demonstrating proof of concept (PoC) and to maximize shareholder value over the medium to long term by enhancing the value of the CRISPR-GNDM® platform as a whole.

RPV: Rare Pediatric Disease Priority Review Voucher

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Funding Strategy for Achieving Clinical Proof of Concept and Sustained Growth

Advancing MDL-101 into clinical trials to demonstrate its value and expand the platform's potential

1 Continued funding is essential for development

Ongoing funding is critical to maximize the value of our clinical development and late-stage pipeline

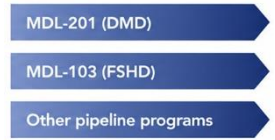
2 This financing is to achieve PoC for MDL-101

The proceeds from this financing will be used to advance MDL-101 into the clinic and generate PoC that demonstrates its safety and efficacy

3 Platform validation drives corporate value creation

Achieving PoC for MDL-101 will enhance the value of the asset and validate the CRISPR-GNDM® platform, leading to an increase in corporate value

4 Substantial growth opportunities in our pipeline



In addition to MDL-201 and MDL-103, we will strategically invest in our pipeline to capture large growth opportunities

The Value We Aim to Create Through This Financing Round

2nd series SB **300mil yen**

19th Series Stock Options
240,000 units
(24 million shares)
1.44–2.4 billion yen*

- **Clinical Proof of Concept (PoC) for MDL-101**
- **Validation of the CRISPR-GNDM® Technology Platform**

Investment in Follow-on Programs (Maximizing Growth Opportunities)

- Realizing a path to **conditional approval** in the U.S. and other countries → Potential for in-house sales
- **Increased likelihood of success** for subsequent large-scale programs

Driven by this round of funding, we aim to continuously enhance corporate value by achieving proof of concept (PoC) for MDL-101 and expanding our subsequent programs

* Assumed exercise price of 60–100 yen

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(for Reference) Value Creation and Cumulative Cost in a Biotech Company

Partnering is not a sale, but a near-term cash in exchange for a share of future economics

Value and Cumulative Cost in the Pipeline

Equity Financing (Capital Raising through New Share Issuance)

Raise capital by issuing new shares

Benefits	Costs (Trade-off)
✓ Large capital can be raised ✓ Funds can be used freely by the company ✓ All rights to the product are retained	⚠ Equity Dilution (Share Dilution) Existing shareholders' ownership percentage decreases

or

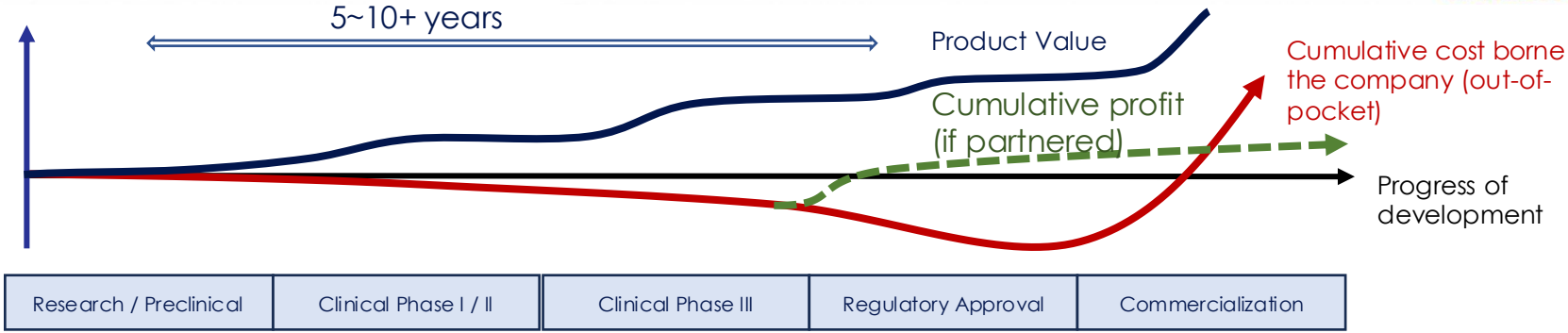
Partnering (Capital Raising through Collaboration)

Receive cash from partner

Benefits	Costs (Trade-off)
✓ Upfront payment ✓ Milestone payments ✓ Reduction of development cost ✓ Sharing of development risk	⚠ Share of future economics (Economic Dilution) A portion of future sales and profits will be shared with the partner

Exchange "today's cash" for "a share of future economics"

After commercialization, large returns (profits) are realized



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5. Risk information

Known Risks and Preventative Measures（1）

Topic	Main Risks	Probability	Level of Impact	Preventative Measure(s)
(1) Risks related to the research and development of gene therapies	The risk of unforeseeable problems developing due to working with cutting-edge experimental medical treatments	Low	High	Constantly monitoring cutting-edge scientific technologies and related businesses, making pertinent judgements, taking appropriate actions
	Due to gene editing technology being a field with steady progress and rapid advancement, there is a possibility of new technologies appearing and risk of competing with other modalities	Low	High	Ensuring we are using the most up-to-date version of the underlying technologies in our research and subsequently monitoring new trends in technologies, and adopting necessary technologies as needed R&D will be carried out with priority given to pipelines with competitive advantage, and portfolio reviews such as discontinuation decisions will be made as needed for diseases for which competitive advantage cannot be maintained
(2) Risks related to the pharmaceutical industry	The risk of failure or decision to suspend development caused by a certain product or technology used in pharmaceutical development	Moderate	High	Regularly reviewing risk mitigation measures and making appropriate modifications to our portfolio when collaborating with partner companies and adding pipelines to our portfolio
(3) Risks related to execution of business activities	Due to the fact that execution and related decisions are driven by our partners, there is a possibility development is suspended and the risk of contract cancellation even if there are no failures during development	Moderate	High	Aiming to stabilize for the future and maximize profits through the stratification of our pipelines and strategically adding to our portfolio
	The risk of delays in the timeline if appropriate business coordination measures are not made regarding production that is entrusted to external parties, preclinical experiments, etc.	Low	High	Performing appropriate project management, concurrently negotiating with various candidate service providers, and securing a slot to prevent delays in the timeline

Known Risks and Preventative Measures (2)

Topic	Main Risks	Probability	Level of Impact	Preventative Measure(s)
(4) Risks related to intellectual property rights	The risk that patents other than the originally introduced license will be required while the basic patent is in a disputed state.	Low	High	Striving to secure patents for each project (including the Company's own patents) and concurrently investigating the introduction of necessary patents
(5) Risks related to business performance, financial condition, etc.	The risk that recorded profits will not be stable, since profit is strongly influenced by the license agreements, milestones, etc.	Moderate	High	Using a hybrid model to stabilize for the future and maximize profits through the stratification of our pipelines and strategically adding to our portfolio
	The risk of significant events related to the going concern assumption.	Moderate	High	Using a hybrid model to stabilize for the future and maximize profits through the stratification of our pipelines and strategically adding to our portfolio
(6) Risks related to the company structure	The risk that we are unable to secure talented individuals who possess technical knowledge or skills for scientific research and development	Low	High	To attract talent, we are engaged in R&D that appeals to potential candidates and creating a favorable working environment in addition to adopting of a restricted stock unit system to recruit competitively
	The risk that negative gossip or rumors influence the public credibility of the Company group	Low	Moderate	Misinformation and rumors are taken seriously, and the Company maintains their position by the equitable, fair, and timely disclosure of information

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Every Life Deserves Attention

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