Corporate Presentation
For investors

The switch  is the Key

(TSE : 4883)

Modalis therapeutics Corporation

December 2023

MODALIS

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Modalis at a glance

- The first and most advanced company developing CRISPR-based epigenome editing therapeutics
- Founded in 2016
- Publicly listed on Tokyo Stock Exchange (Ticker: 4883)
- Headquartered in Tokyo and R&D operated in Waltham MA
- ~40 FTE
- President and CEO: Haru Morita, formerly at Kirin, Booz-Allen and Hamilton, REGiMMUNE…

MODALIS' Value Highlights

- Established the first robust epigenetic platform for activation and inhibition of endogenous genes using CRISPR-GNDM® platform
- Demonstrated sustained modulation of gene expression in multiple species (mouse, cyno) resulting in functional efficacy without toxicities
- Pipeline of preclinical assets in muscular dystrophies, additional programs in CNS, cardiovascular and unlimited therapeutic potential in other areas
- Manufacturing established for challenging AAV capsids to enable tissue tropic delivery in lead programs
- Experienced team with deep knowledge of platform
- Strong IP portfolio and strategy that includes granted patents
- Established regulatory and clinical path based on recent FDA guidance

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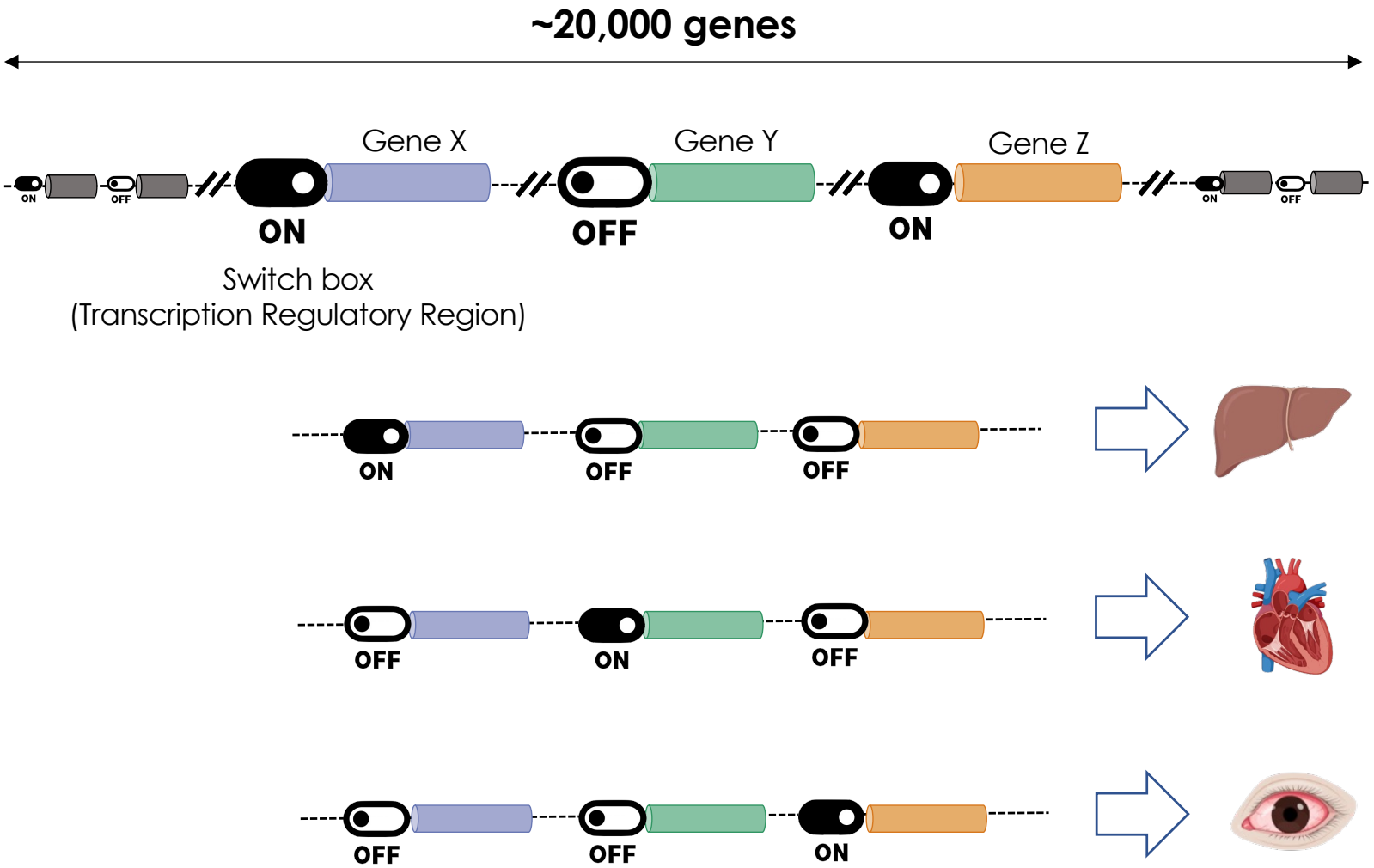
What is the epigenome?

Modifications that control where, when, and how much each gene is expressed by adapting regions of the genome, without changing the DNA sequence



Organisms are controlled by transcription machinery

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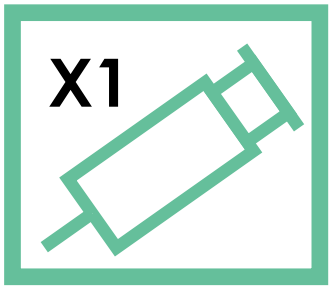
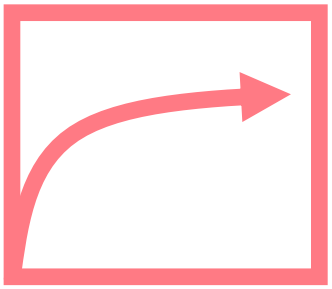
CRISPR-GNDM® is a promising new therapeutic modality that controls the epigenome

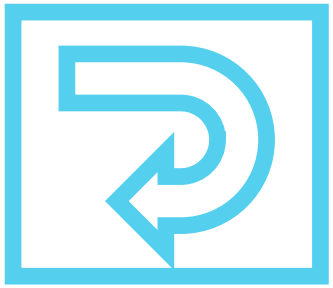
Potential benefits of CRISPR-GNDM® Technology



Single dose
Does not require
Repeated dosing



Long-lasting
Sustained effect
for years or decades



Disease Modifying
Not just reduction of
symptoms but
potential to cure

CRISPR-GNDM® does not alter DNA sequence

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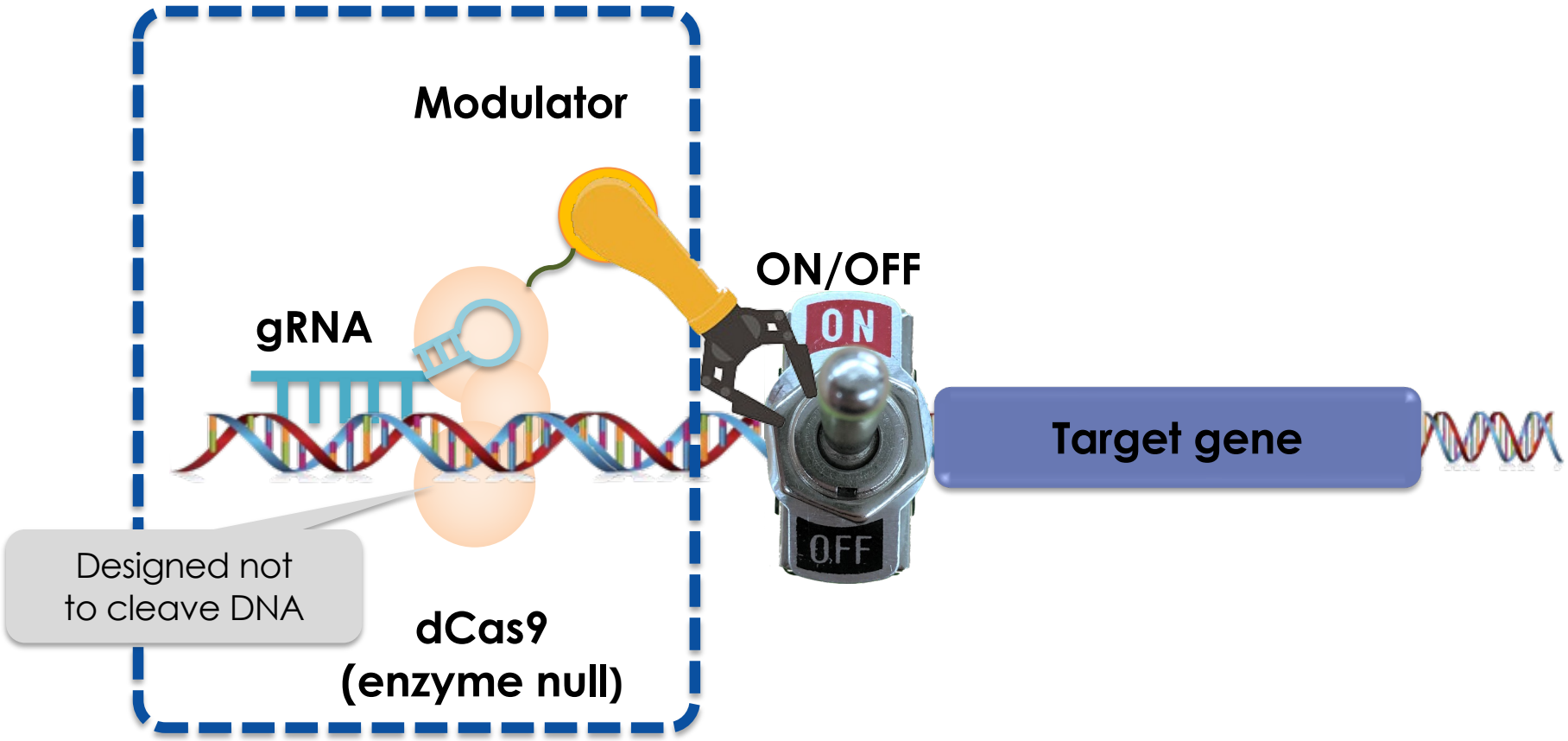
Non-cleaving CRISPR = CRISPR-GNDM®

Enables treatment of genetic disorders by controlling epigenetic ON/OFF switch

CRISPR-GNDM® (Guide Nucleotide-Directed Modulation) platform



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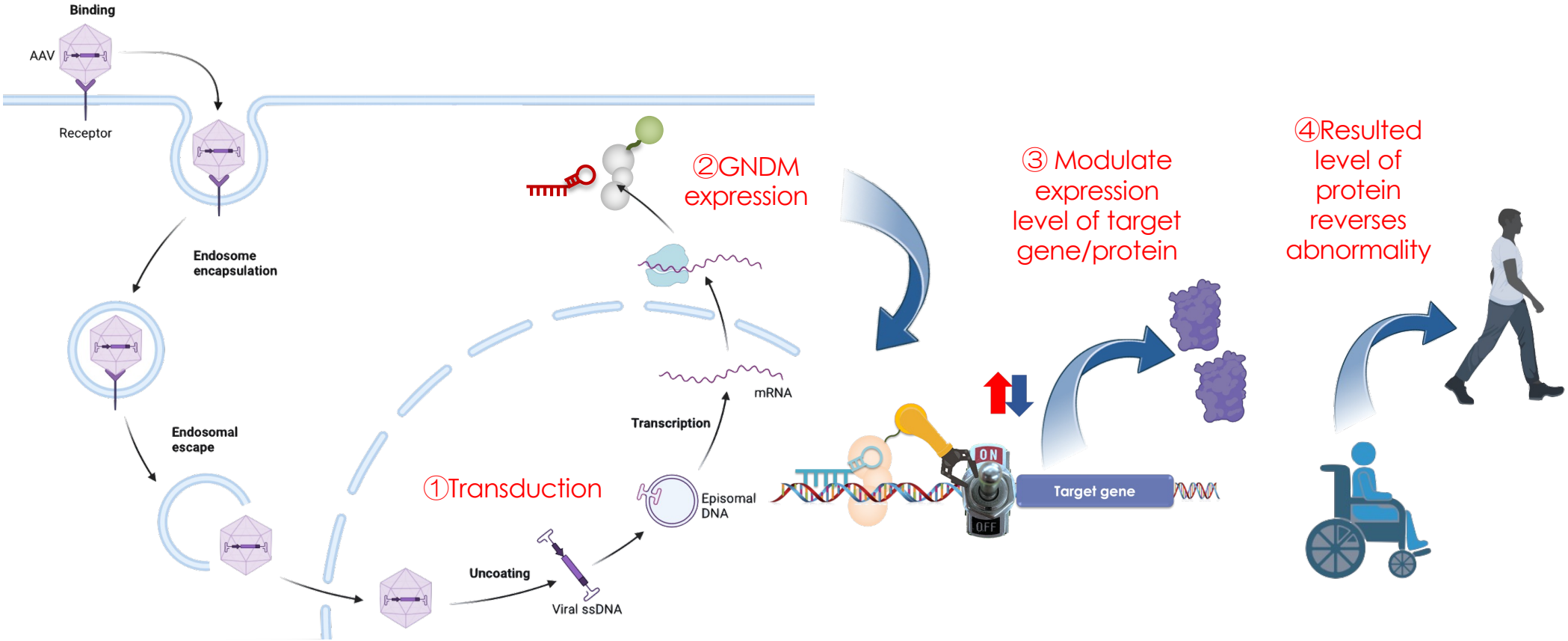
GNDM-MDL-101 has been validated - efficacy in mouse disease models and target engagement in monkey



①AAV Transduction	②GNDM expression	③Modulation of target	④Efficacy

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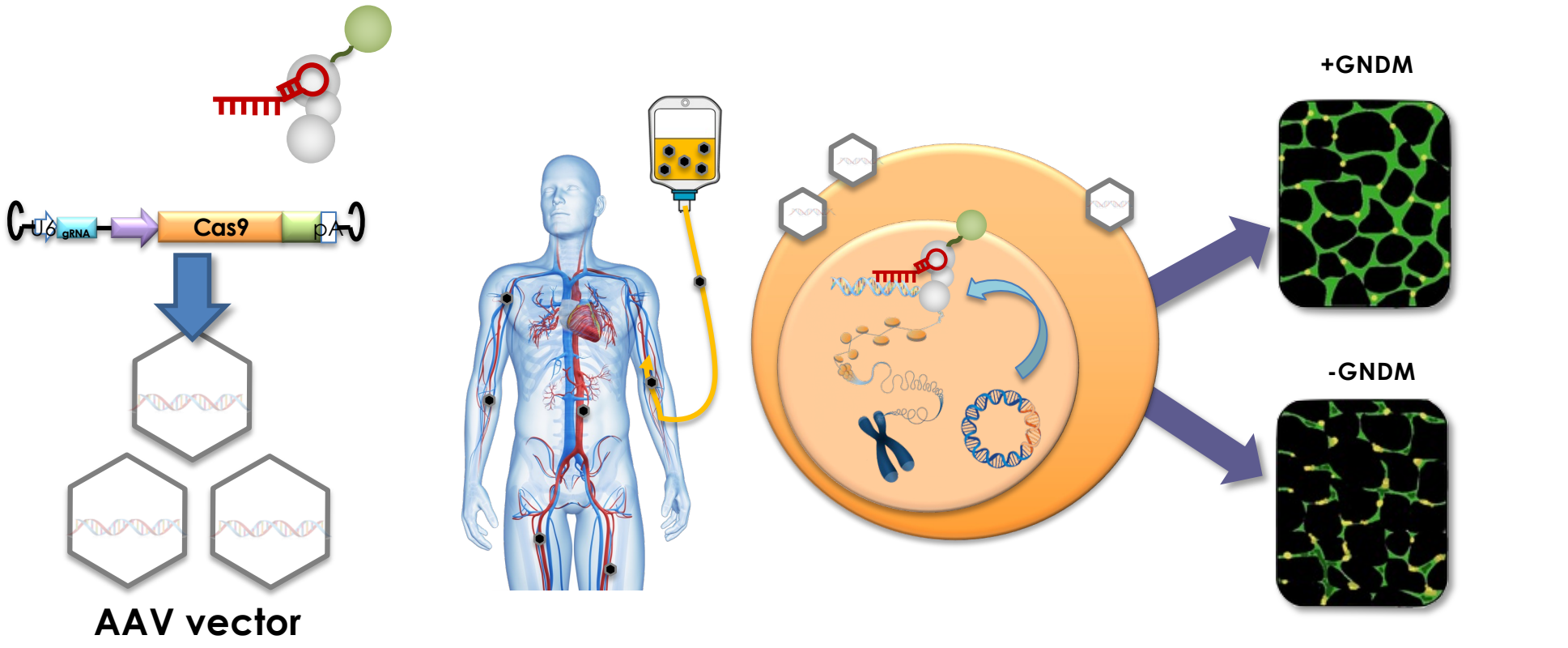
Delivery of CRISPR-GNDM® to target

AAV vector to deliver GNDM to target cell



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CRISPR-GNDM® targets genes that cannot be addressed by other modalities

	Conventional Gene therapy	Gene Editing	ASO siRNA	CRISPR-GNDM
Precise targeting	Yes	Yes	Delivered to off-target tissues	Yes
Durability	Years	Permanent	Require repeated injection	Years
Applications	LoF ONLY	Mostly GoF	GoF only	**LoF and GoF**
Target gene limitation	Limited to small size genes	Limited to a specific point of mutation	Causative tissue is limited (e.g. liver)	**Size agnostic**
effect on DNA	none	Causes double-stranded breaks	none	none

LOF=Loss of function, GOF=gain of function

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Epigenome editing competitive landscape
Modalis is in the lead

Company	Year Founded	Funding	Platform	Pipeline/Target indication	Stage of Development
Modalis Therapeutics	2016	Public	CRISPR-GNDM x AAV	• MDL-101/LAMA2-CMD • MDL-202/Myotonic Dystrophy Type 1(DM1)	IND enabling PreIND completed
Navega Therapeutics	2018	Government Grant	ZFN and dCas9 fused with transcription activator x AAV	Dravet syndrome	Animal study?
Tune Therapeutics	2020	Series A ($40M, Dec 2021)	DNMT-KRAB fusion dCas9 x LNP	PCSK9 for hypercholesterolemia? HBV	NHP study reported at ASGCT2023
Chroma Medicine	2021	Series B ($135M, Mar 2023)	DNMT-KRAB fusion dCas9 x LNP	PCSK9 for cardiovascular disease	Mice study reported at ASGCT2023
Epic Bio	2022	Series A ($55M Jul 2022)	Cas12f-fused with demethylation enzyme x AAVrh74	EPI-321/FSHD	Mice study reported at ASGCT2023

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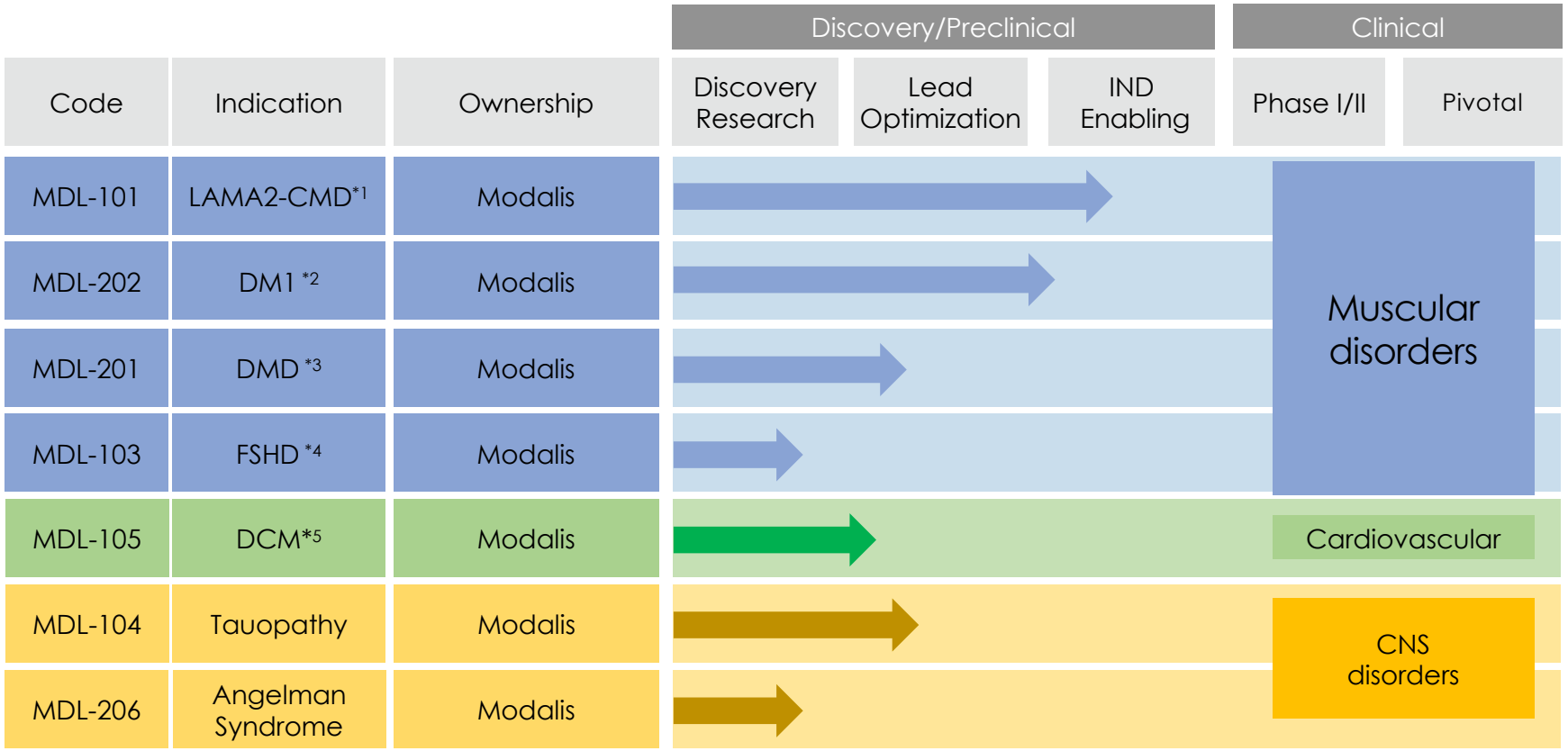
Modalis pipeline strategy focuses on musculoskeletal diseases



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

Note: The pipeline is being reorganized. For details, please refer to "4. Pipeline Status: Key Points of Pipeline Reorganization" on page 38.

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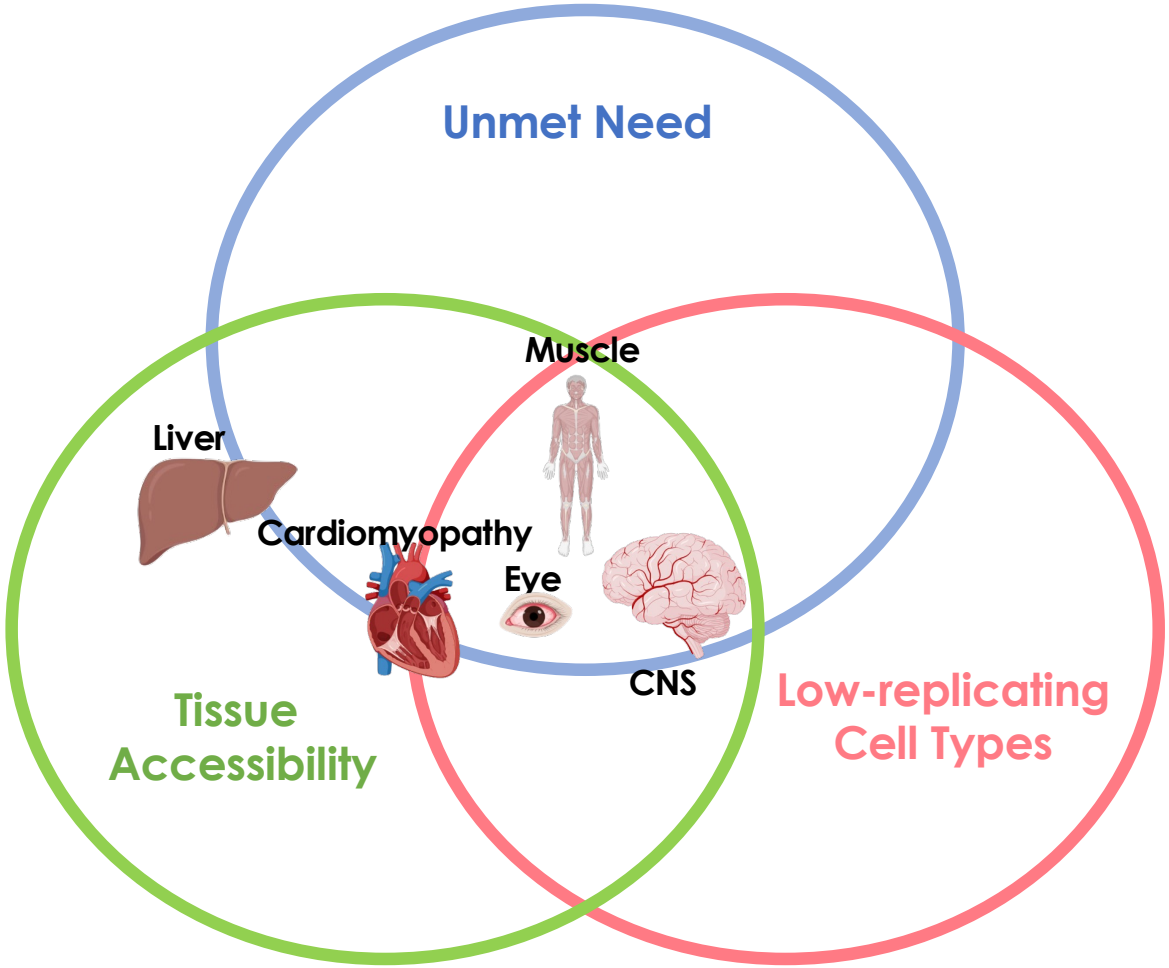
CNS and cardiomyopathy are additional active areas

Target selection criteria for Modalis' gene therapies



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With the initial success, gene therapy has begun to expand targets from local to systemic administration

Gene therapies approved by US FDA

Trade Name	Approval	cost	Indication	Manufacturer	Patient Population	WW market size* (mil USD)
Lxturna	2017	$850k	RPE65	Spark/Roche	2 per 100,000	$65M[♭]
Zolgensma	2018	$2.1M	SMA	Novartis (Avexis)	1 in 10,000 live births (Approx. 10,000 to 25,000 in US)	$1.3B[♭]
HEMGENIX	2022	$3.5M	Hemophilia B	uniQure CSL Behring	1 in 30,000 male	$88M[♭]
Vyjuvek	2023	$631k per patient per year	DEB[*2]	Krystal	3.5–20.4 in 1 million	~$200M[#2]
ELEVIDYS	2023	$3.2M	DMD	Sarepta	1 in 3,500 male birth	$4.1B[#]
Roctavian	2023	$2.9M	Hemophilia A	BioMarin	1 in 5,000 male	$262M[#]

♭ each company's website
#Grand view research, WW market size

Source: National Organization for Rare Disorder, Companies websites

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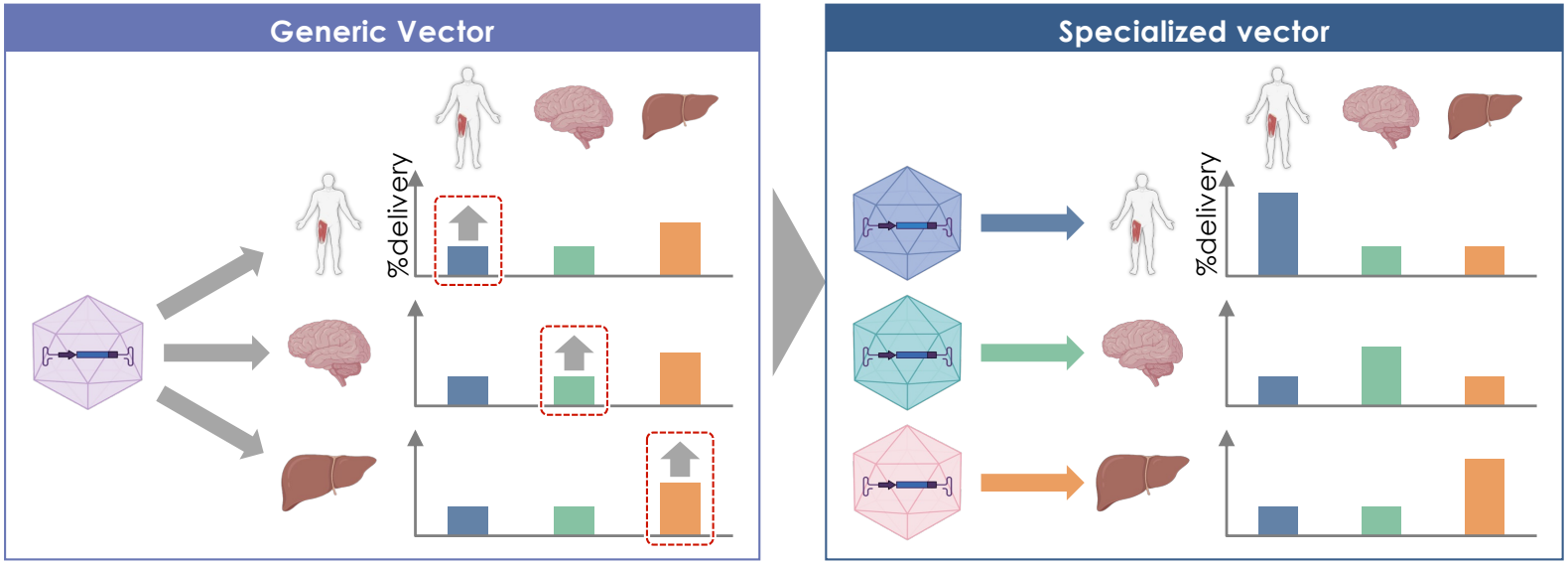
Current AAV capsids provide tissue tropic delivery

- Previous AAV 2, 6, 8,9 vectors were used across all tissues and diseases
- Systemically delivered capsids sequestered in the liver, causing hepato- and dose limiting toxicities
- Novel, engineered vectors have high target tissue tropism for muscle, CNS or liver with potential to:
  - ➢ **Increase delivery to target organ, eliminate off target organ toxicities**
  - ➢ **Lower dose required to achieve efficacy**
  - ❖ **Modalis approach is validated and can utilize multiple capsids**



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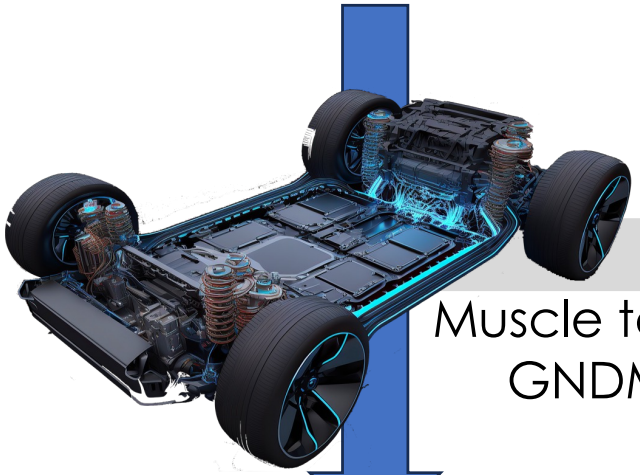
All muscle programs share the same platform as MDL-101



Muscle targeting AAV
GNDM platform

MD-101

MDL-202

MDL-201

MDL-103

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Pipeline

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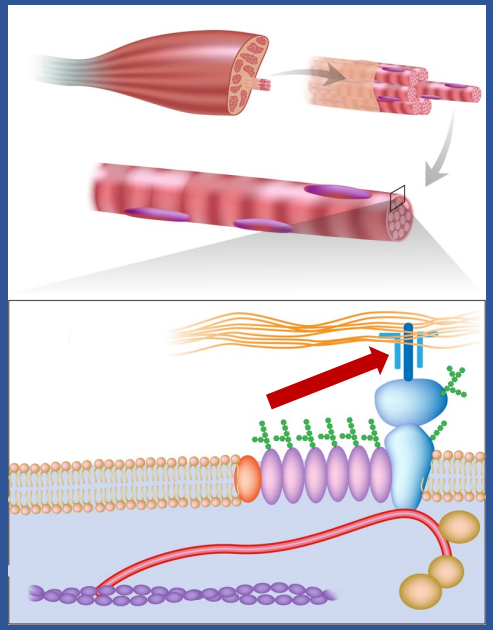
LAMA2-CMD (aka CMD1a)

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

MDL-101

Potential to be the first LAMA2-CMD treatment



Prevalence	**1 in 30,000*** 10,000 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: *Ophanet #*Modalis assumption based on prevalence and potential*

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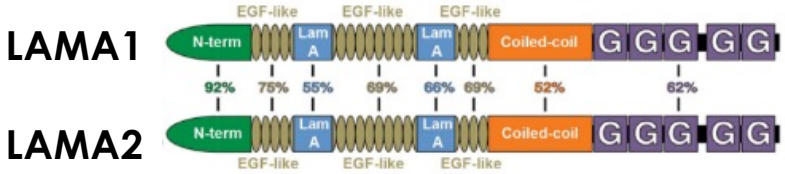
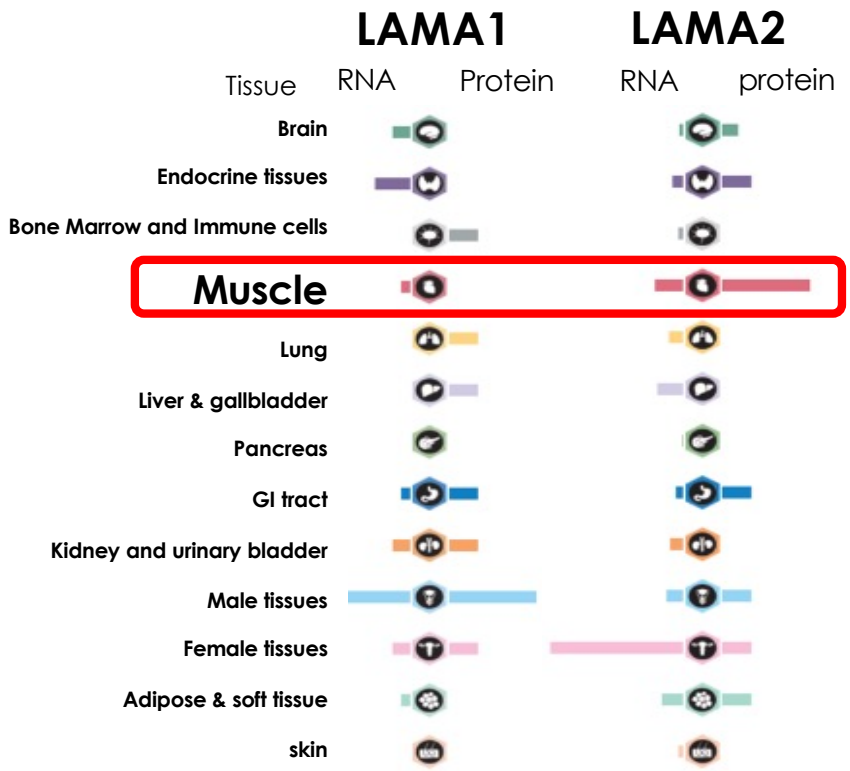
MDL-101 activates LAMA-1, compensates for the missing function of LAMA2, which is too large for classical gene therapy approach

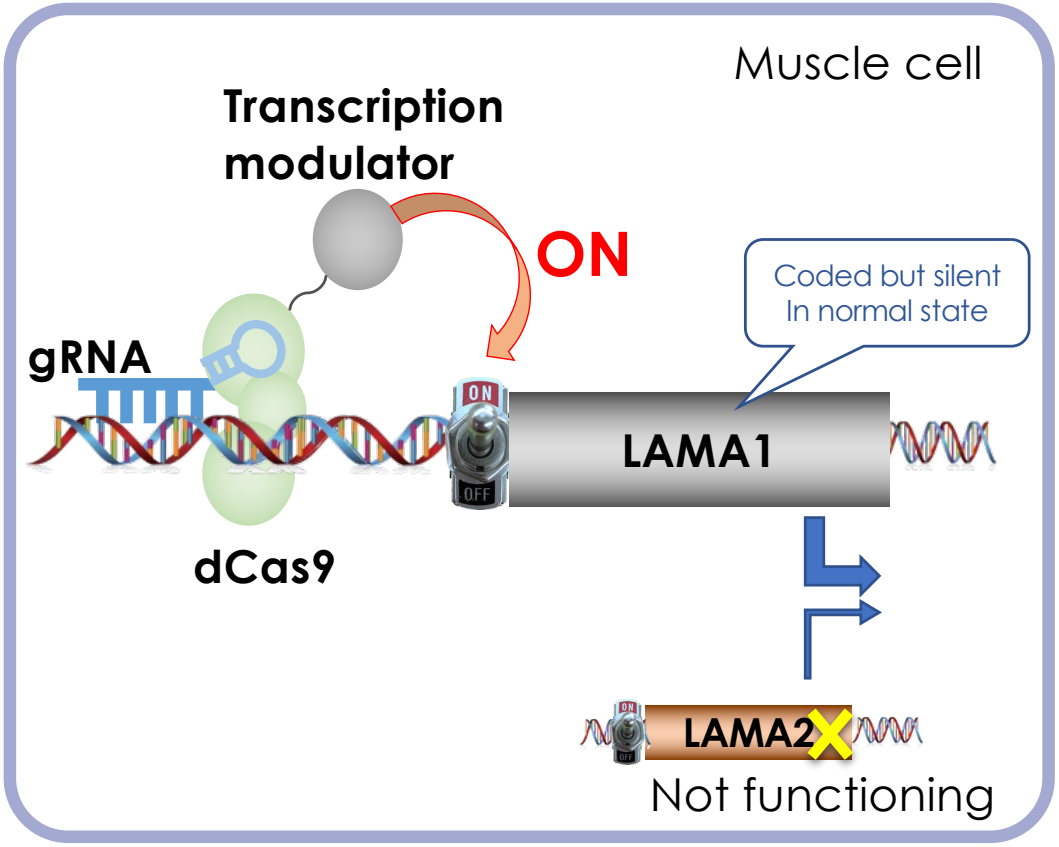
LAMA1/2 protein structure



Exspression pattern of LAMA1and LAMA2 by tissues



CRISPR-GNDM® targeting LAMA1



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MDL-101 Development

- Current status
  - Mouse efficacy in two disease strains (dy2j and dyW) and wild type
    - Upregulation of LAMA–1 gene and protein along with GNDM expression
    - Improvement in biochemical and physiological readouts and prolonged survival
    - Sustained expression of GNDM in WT mice for 2 years
  - Pilot NHP study ongoing to explore dose and assess immune response to GNDM
  - Muscle-specific capsid in use; new constructs have been evaluated in rodents and NHPs
    - Positive results including meaningful LAMA-1 expression
  - Positive feedback from pre-IND FDA meeting (June 2023)
- Next steps:
  - IND enabling GLP tox and PK/PD
  - Continue process development and pilot manufacturing for GMP campaign

KOL: Key Opinion Leader

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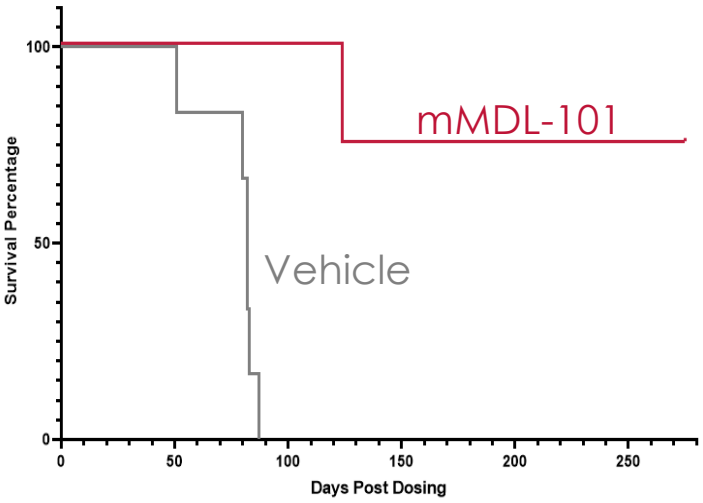
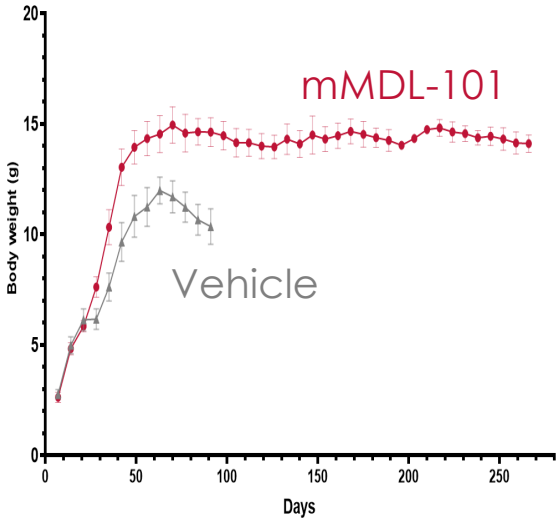
mMDL-101 is efficacious in severe MDC1a model
Increased lifespan, body weight and grip strength of dy^{W} mice

Extended lifespan
(% Survival)



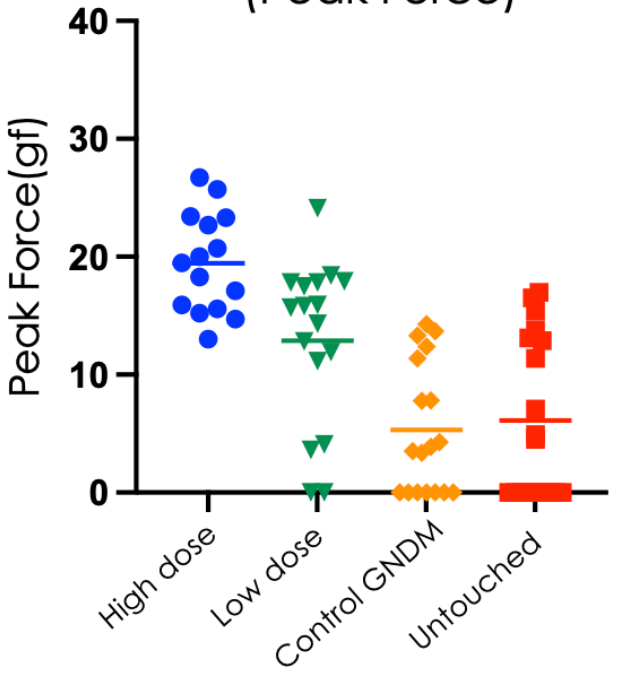
p<0.005, log rank test

Improved growth
(Weight)



p<0.0001, Mann-Whitney test

Forepaw Grip Strength
(Peak Force)



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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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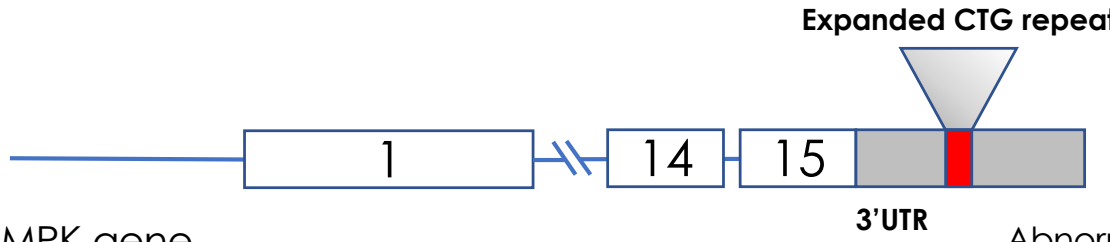
DM1 is caused by abnormal splicing rooted from CTG extension in 3'UTR of DMPK gene

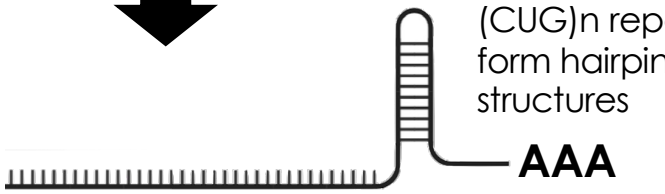
DM1disease causing mechanism



DMPK gene

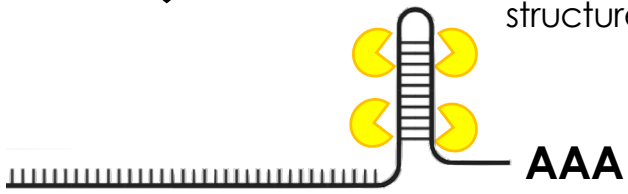
Abnormally expanded (CUG)n repeats form hairpin-like structures

DMPK mRNA With CUG repeat



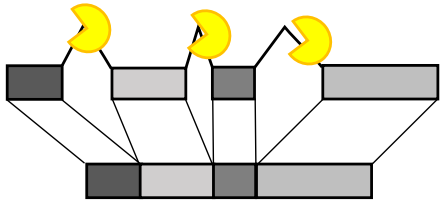
MNBL binds to hairpin-like structures

RNA to accumulate in nucleus by sequestering isoforms of the MBNL

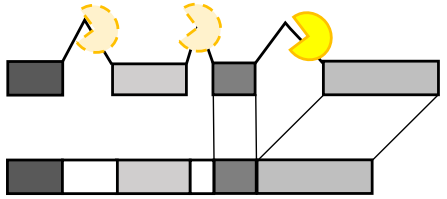


Splicing in healthy people

MBNL1



MBNL1 contributes normal splicing



MNBL goes back to functioning in normal splicing

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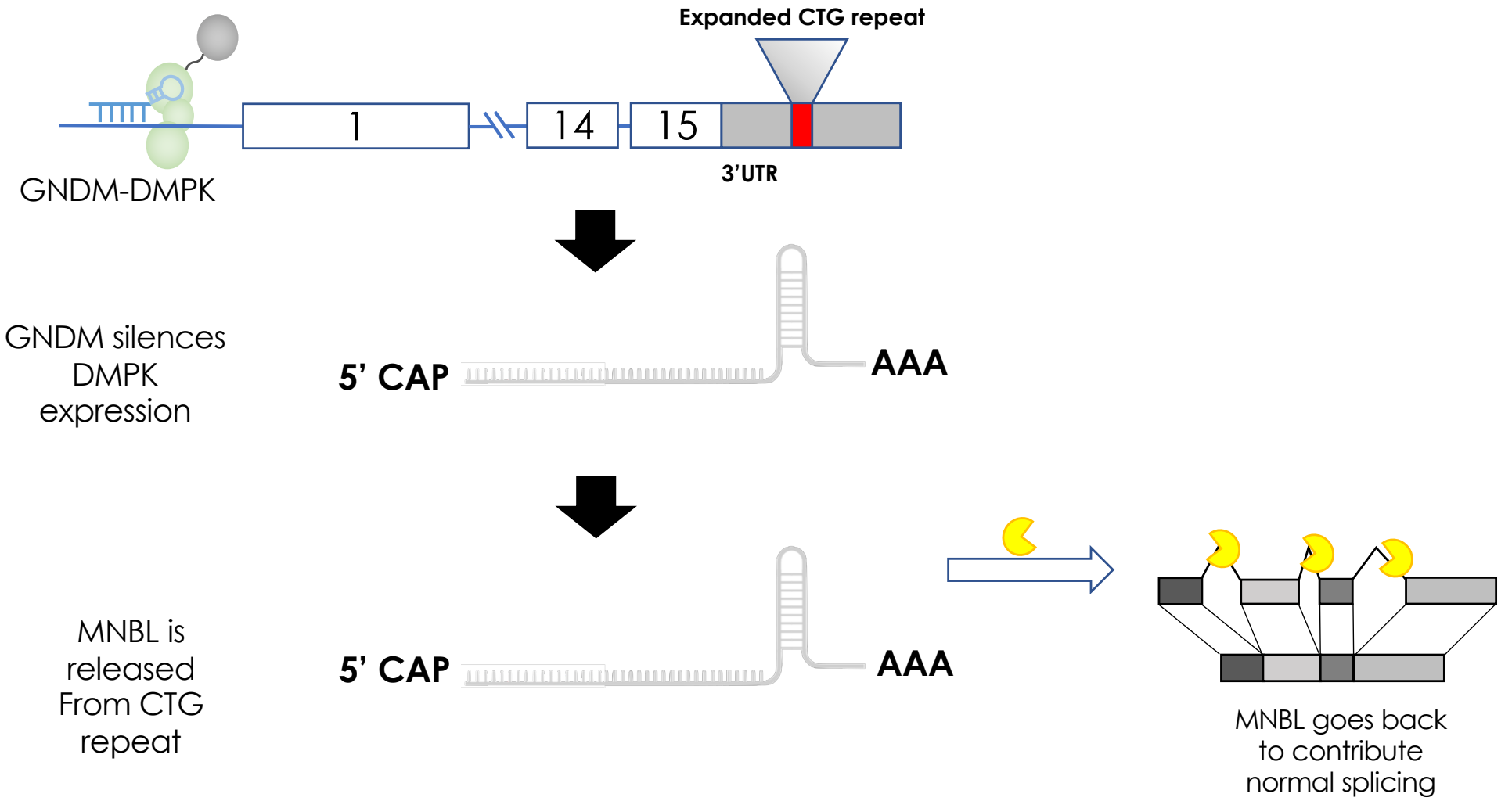
MDL-202 silences DMPK expression and releases splicing protein MNBL to function properly in muscle cells

MDK-202's mechanism of action



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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	$1.1BM 2022	Expected to grow at CAGR=42.5% with approval of new therapeutics

*Source: https://doi.org/10.1212/WNL.0000000000011425

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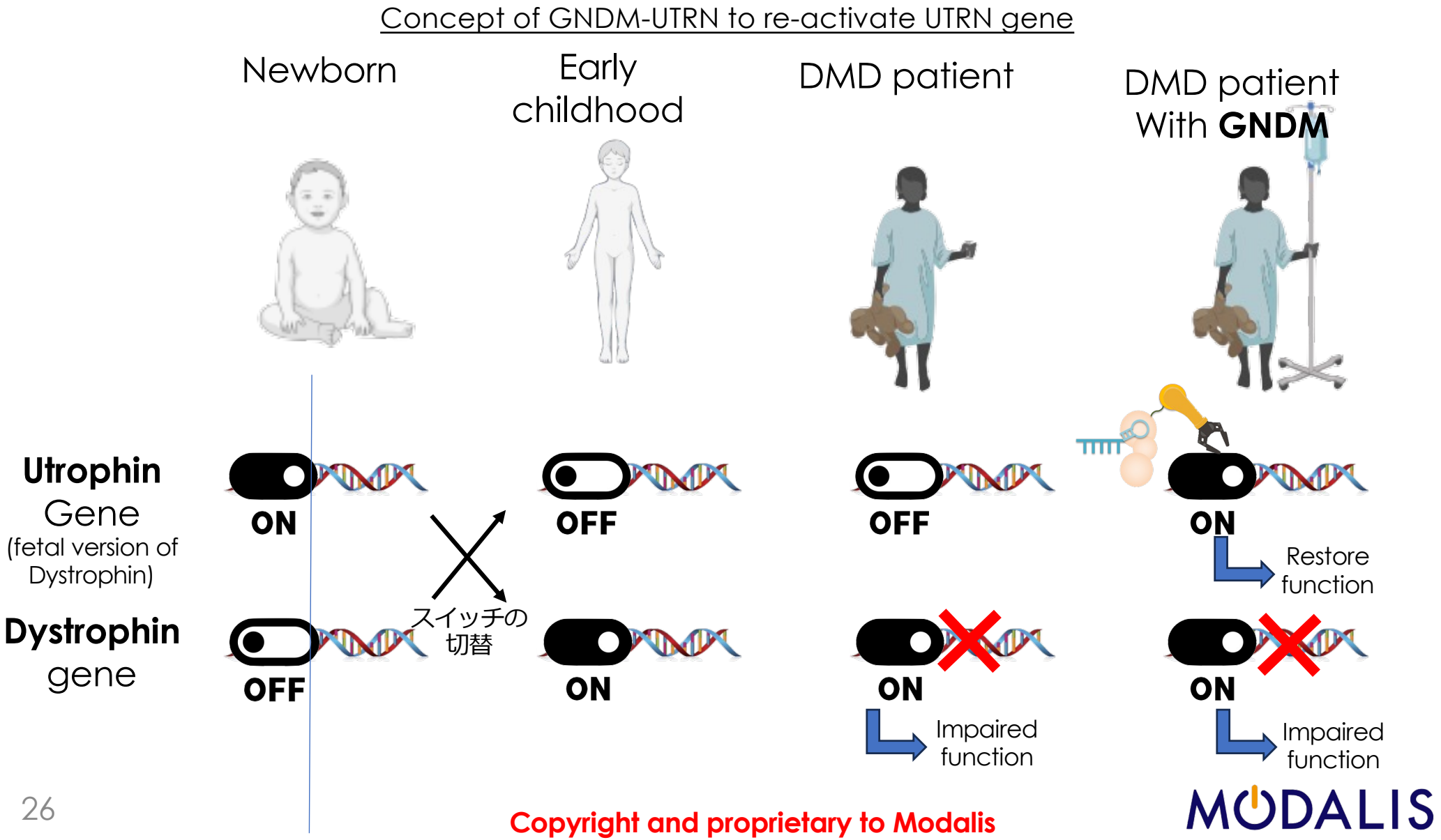
UTRN-GNDM reactivates Utrophin gene to compensate non-functional Dystrophin gene in DMD patients



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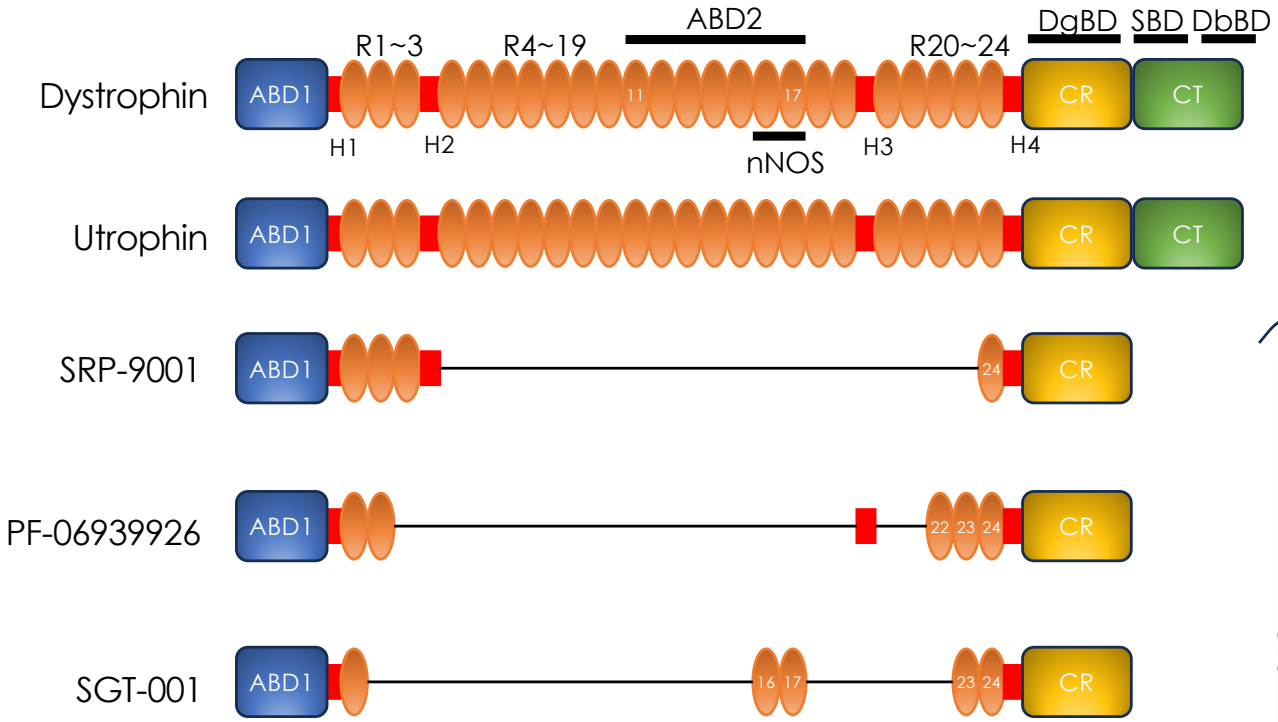
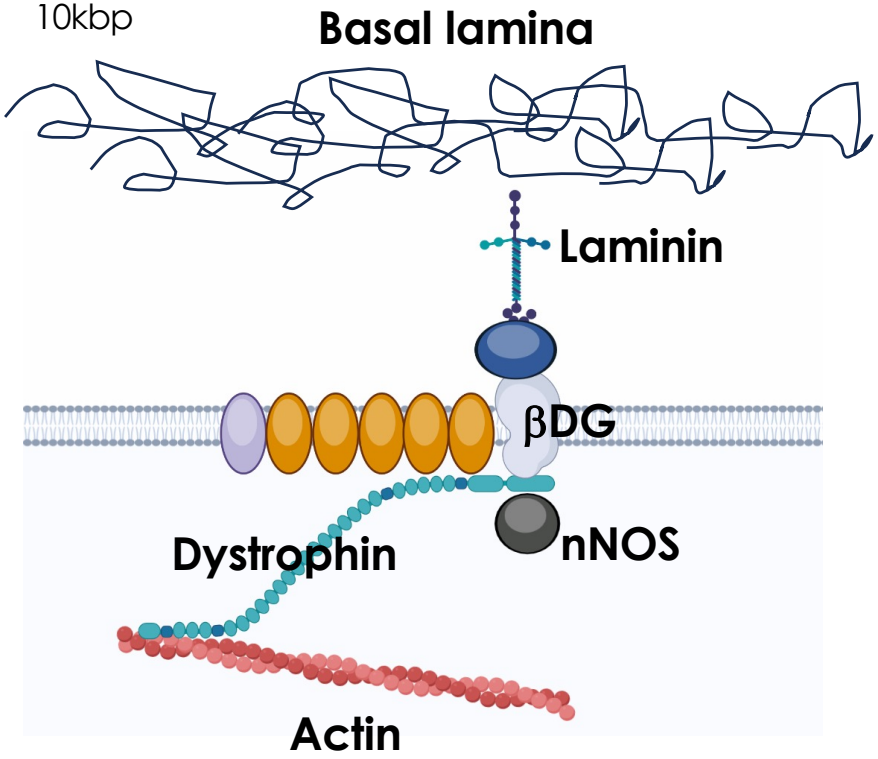
Competitors are developing mini-dystrophins, with known low functionality, as wt Dystrophin is too large to fit into the AAV

Dystrophin/Utrophin Structure and mini-Dystrophins





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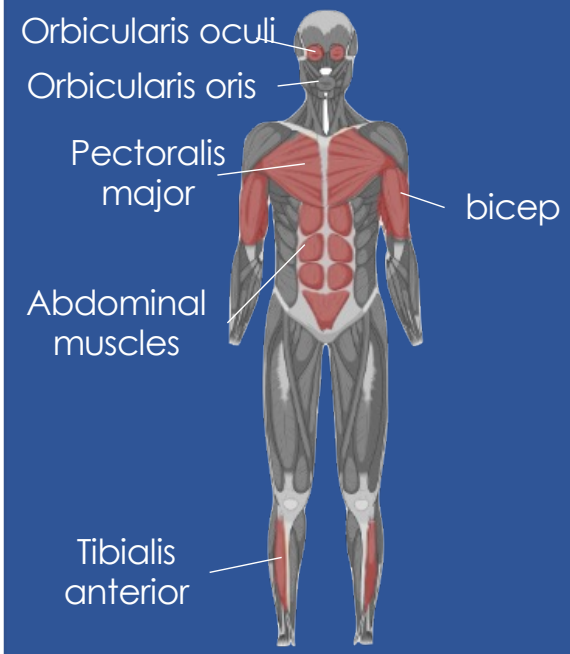
Facioscapulohumeral Muscular Dystrophy (FSHD)

A type of muscular dystrophy caused by impaired Dux4 gene expression

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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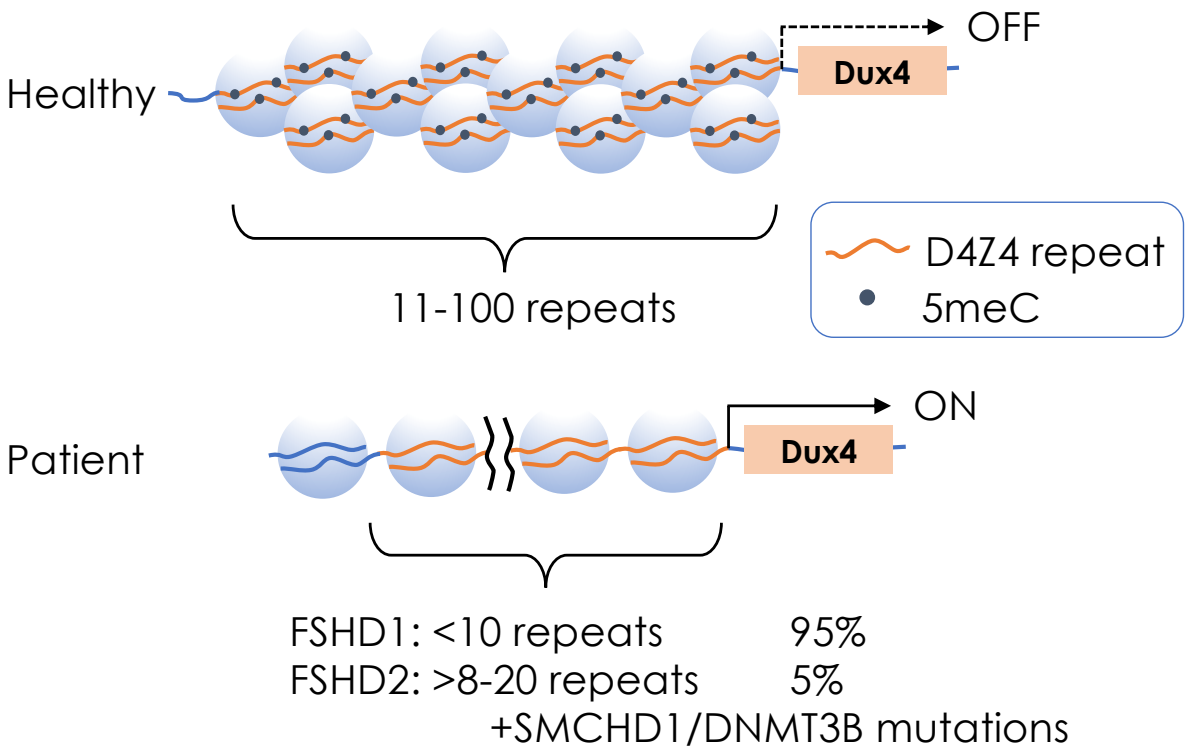
FSHD disease mechanism

Inappropriate over-expression of Dux4 in skeletal muscles



- The D4Z4 repeat region at location 4q35 on chr4
- Healthy individuals have numerous highly **methylated** D4Z4 repeats
- FSHD-1 and -2 affected individuals have **hypomethylated** D4Z4 repeats
- FSHD-1 non-manifesting, or unaffected, has few repeats, but these have **higher methylation**

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Tauopathy (incl. Alzheimer's Disease)

Neurodegenerative disorders caused by misfolding of the tau protein

MDL-104

Potentially best-in-class molecule by silencing Tau expression

Prevalence	**1 in 9 above 65*** 55 million in ww	60-80% of cognitive disorders
Disease Onset	Progressed in **6-8 yrs**	Slow and chronic progression, depending on the individual
Disease Burden	progressive disease beginning with mild memory loss	possibly leading to loss of the ability to carry on a conversation and respond to the environment.
Disease Causing Gene	Multiple causes have been proposed but not yet known	APO-E, PSEN1, PSEN2 and many other gene mutations reported All mutations are associated with Aβ hyperactivity
Commercial opportunity	**$4.2B** in 2022#	Estimated to grow to $15.6B by 2030#

Source: * Alz.org (for Alzheimer Disorder) #Grand View Research

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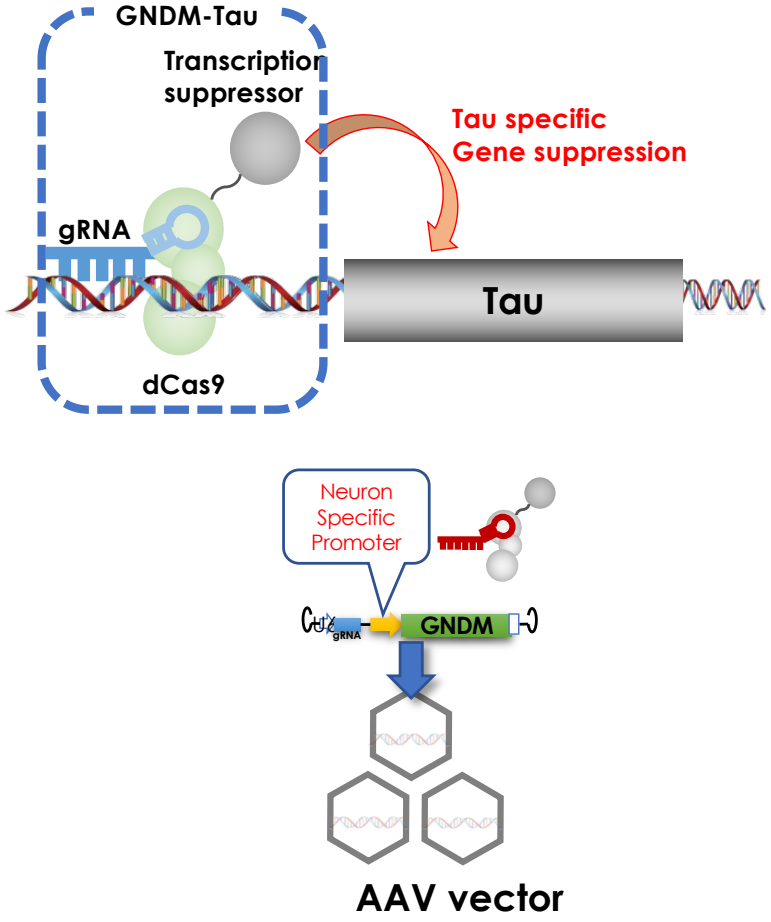
Product concept of Tau suppressor by CRISPR-GNDM®

- Reversing the pathogenic conditions of Tauopathy by partial or full suppression of Tau gene that leads to reduction of Tau protein in the brain
- GNDM-Tau, driven by neuron specific promoter delivered by AAV9 or alternative capsid
- ICM (intra-cisterna magna) injection to achieve efficient brain delivery and to avoid high-dose AAV related toxicities



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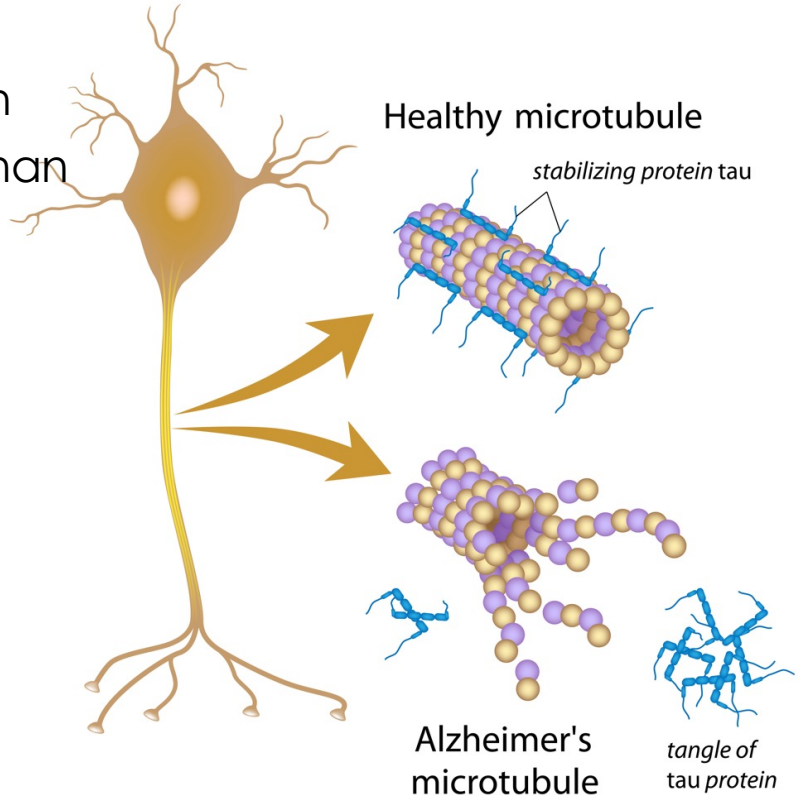
Tau is a center of attention in treating Alzheimer diseases

- Tau correlates with clinical symptoms and neuronal loss in Alzheimer's disease and other primary tauopathies.
  - Tau aggregates and tangles are thought to induce neuronal degeneration, synaptic loss and cell death
  - Tauopathies include a range of high value and orphan clinical diseases
    - AD (Alzheimer's Disease)
    - FTLD (Frontal Lobar Degeneration)
    - PSP (Progressive Supranuclear Palsy)
    - CBD (Corticobasal Degeneration)
    - Pick's disease
- Tau is likely to be a better target than Aβ because the tau burden correlates better with clinical impairments than does the Aβ burden
- Tau knockout has few adverse effects
- Therefore, reducing total Tau expression is a logical therapeutic strategy



Source: Congdon EE, Nature Review Neurology 2018 "Tau-targeting therapies for Alzheimer disease"

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GNDM mediated suppression of MAPT in humanized Tau mice

Humanized MAPT mice

ICV injection@P2

1.4e+11 vg/head

8wk

RNA-FISH

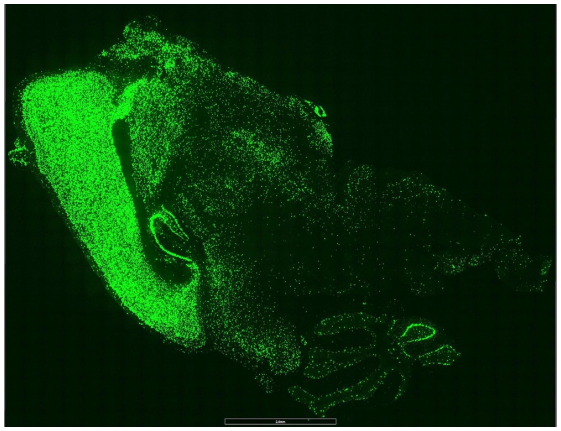
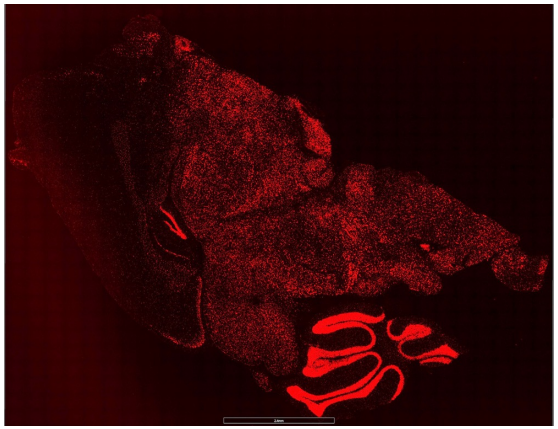
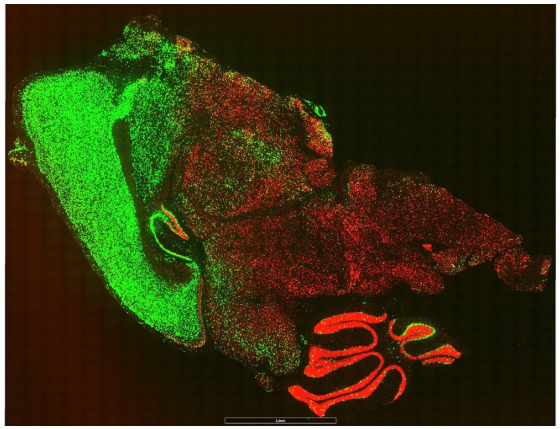
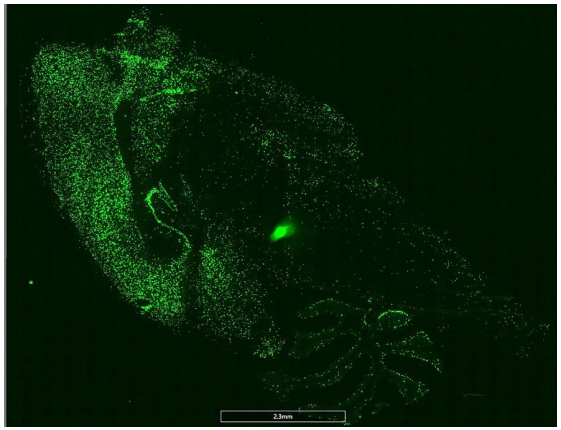
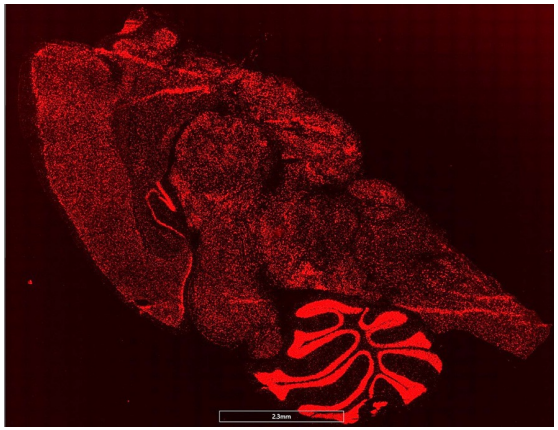
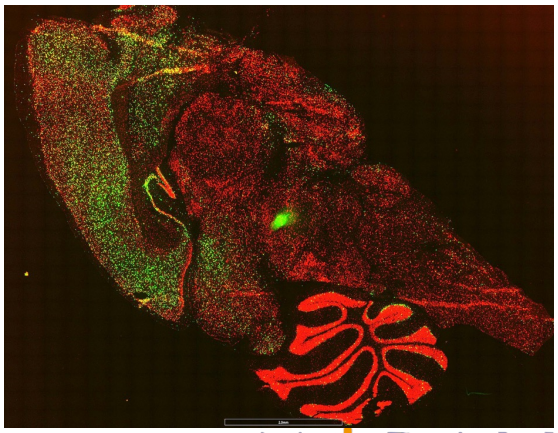
GNDM

Tau

GNDM+Tau

GNDM -Tau

GNDM -对照gRNA

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Angelman Syndrome

CNS disorder caused by impaired UBE3A gene expression

MDL-206

Potentially first-in-class treatment by unsilencing UBE3A gene expression

Prevalence	**1 in 10,000 to 20,000 live birth**	• 60,000 patients WW (2017) • 20,000 in US
Disease Onset	typically diagnosed at **6 to 12 months**	Seizures may begin at 2 to 3 years.
Disease Burden	Developmental delay autism	small head, specific facial appearance, intellectual and developmental disability, speech impairment, balance and movement problems, seizures, and sleep problems
Disease Causing Gene	Mutation in maternal allele of **UBE3A**	5-26% has unknown mechanism
Commercial opportunity	**$330M** (2017)	US, EU, and Japan

Source: Orpha.net

Angelman syndrome Market and Epidemiology forecast (delveinsight.com)

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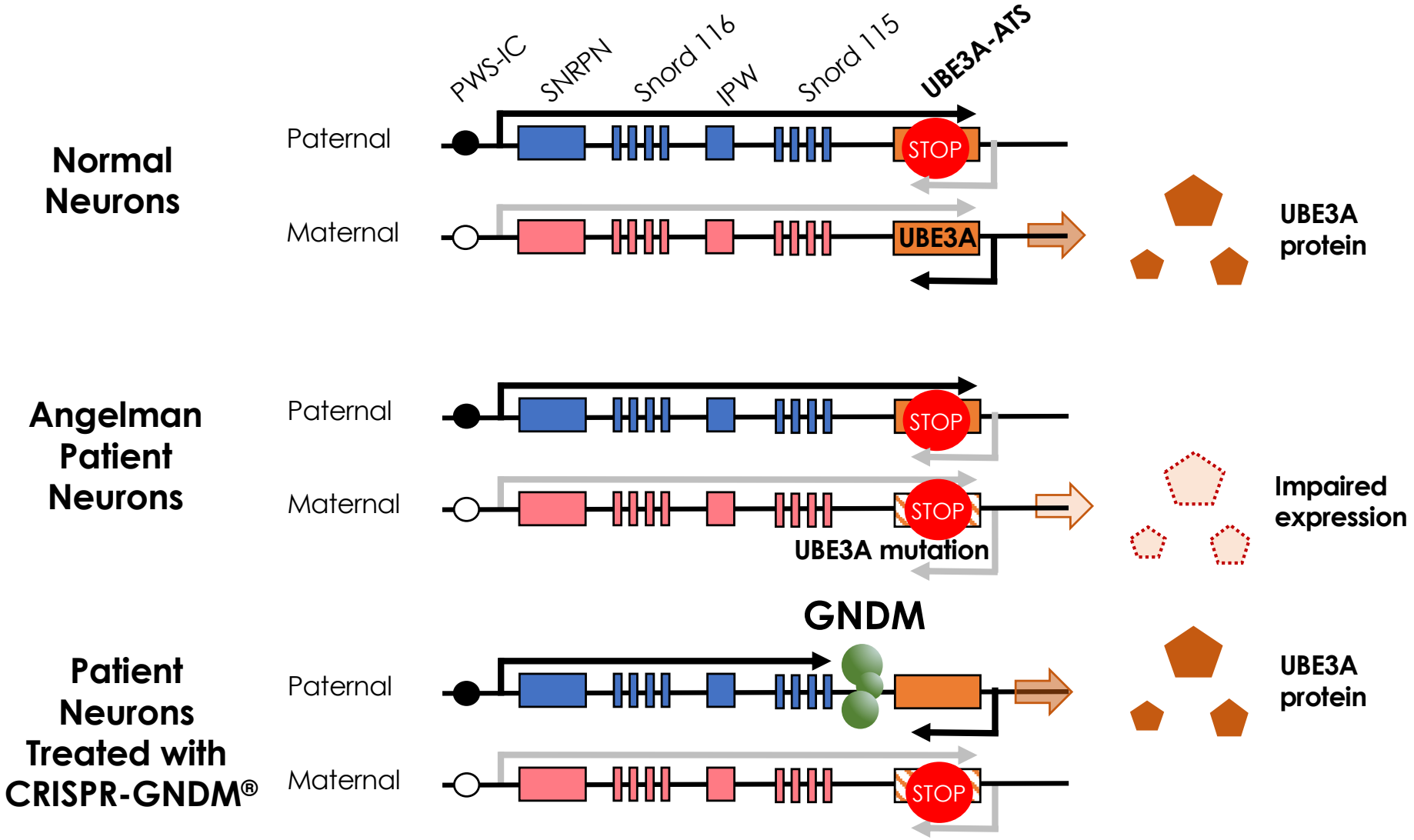
By blocking ATS transcript, GNDM un-silences UBE3A expression



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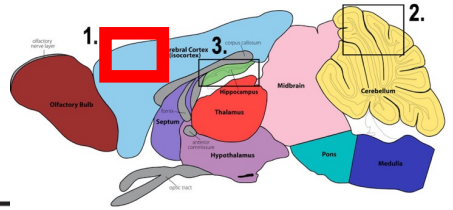
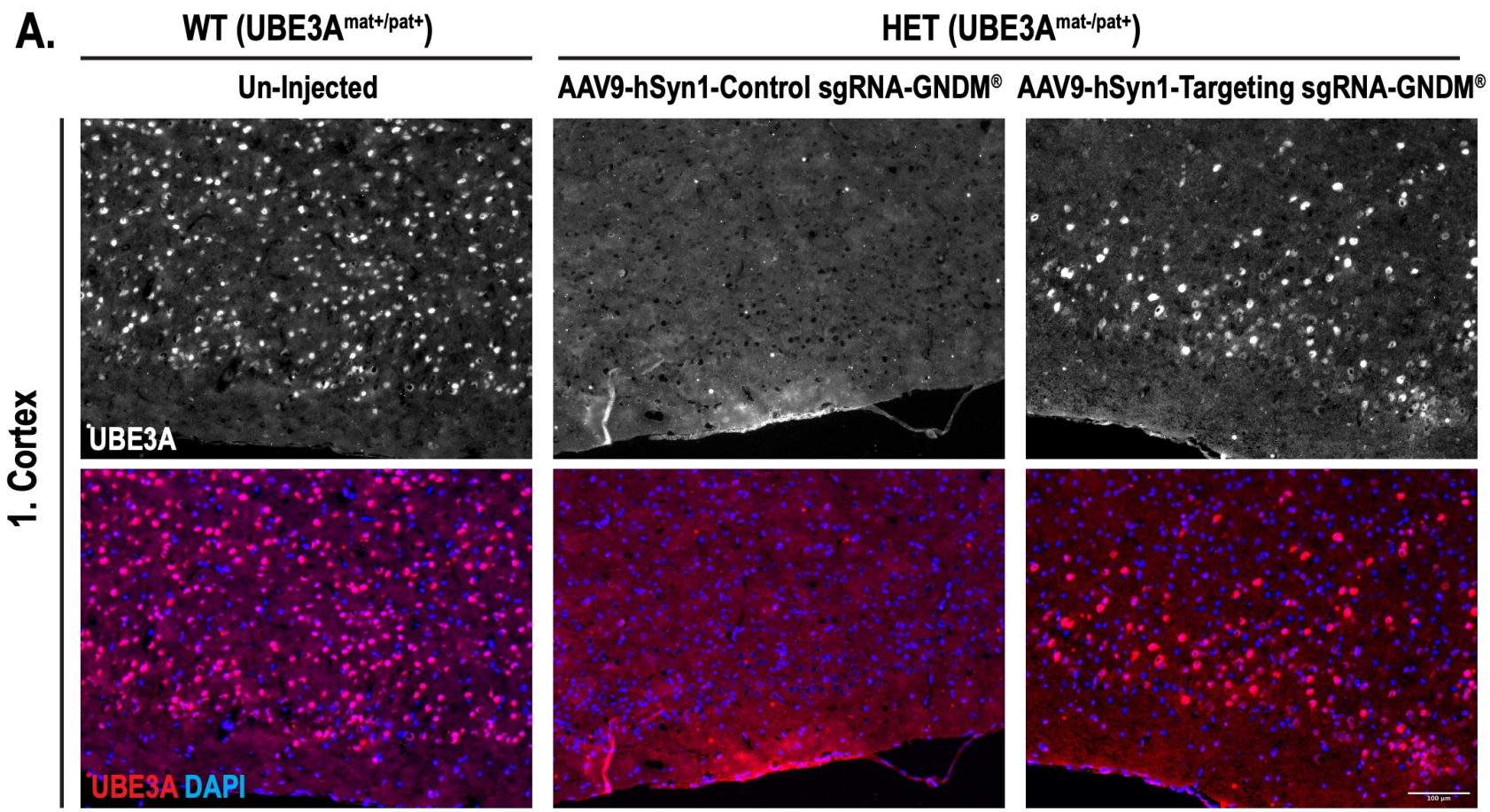
UBE3A is successfully unsilenced in Cortex





UBE3A staining in un-inject- ed WT mice brains (left) and HET (UBE3A$_{mat-/pat+}$) mice injected with AAV9-hSyn1-Control sgRNA-GNDM® (middle) or AAV9-hSyn1-Targeting sgRNA-GNDM® (right). UBE3A staining alone (black and white; top), UBE3A (red) and DAPI (blue) (merge; bottom).

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Business and Development Strategy

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Development stage of Modalis pipeline

	Current status	Next milestones
MDL-101 LAMA2-CMD	• Animal PoC in mouse disease models • Target engagement in NHP (ASGCT 2023 Late-breaking Abstracts #2) • PreIND meeting with FDA (June 2023)	• GLP-Tox • GMP manufacturing • IND (2H 2024)
MDL-202 DM1	• Animal PoC in disease mouse model • Regained rights from Astellas (Aug2023) • Transferred to new vector platform • Initiated mice disease model and NHP target engagement • Active partnering discussions	• NHP target engagement readout (1Q/2024) • Mice disease model data readout (2Q, 2024) • Partnering
Discovery Stage Programs	• MDL-201 (DMD) • MDL-103 (FSHD) • MDL-105 (DCM) • CNS programs: (Tauopathies) (Angelman's Syndrome)	• Conversion to muscle tropic capsid(s) • Animal PoC (FSHD, DCM) • NHP target engagement with new capsid • Partnering • Optimize gRNAS and animal PoC • Explore CNS tropic capsids and LNPs • Partnering

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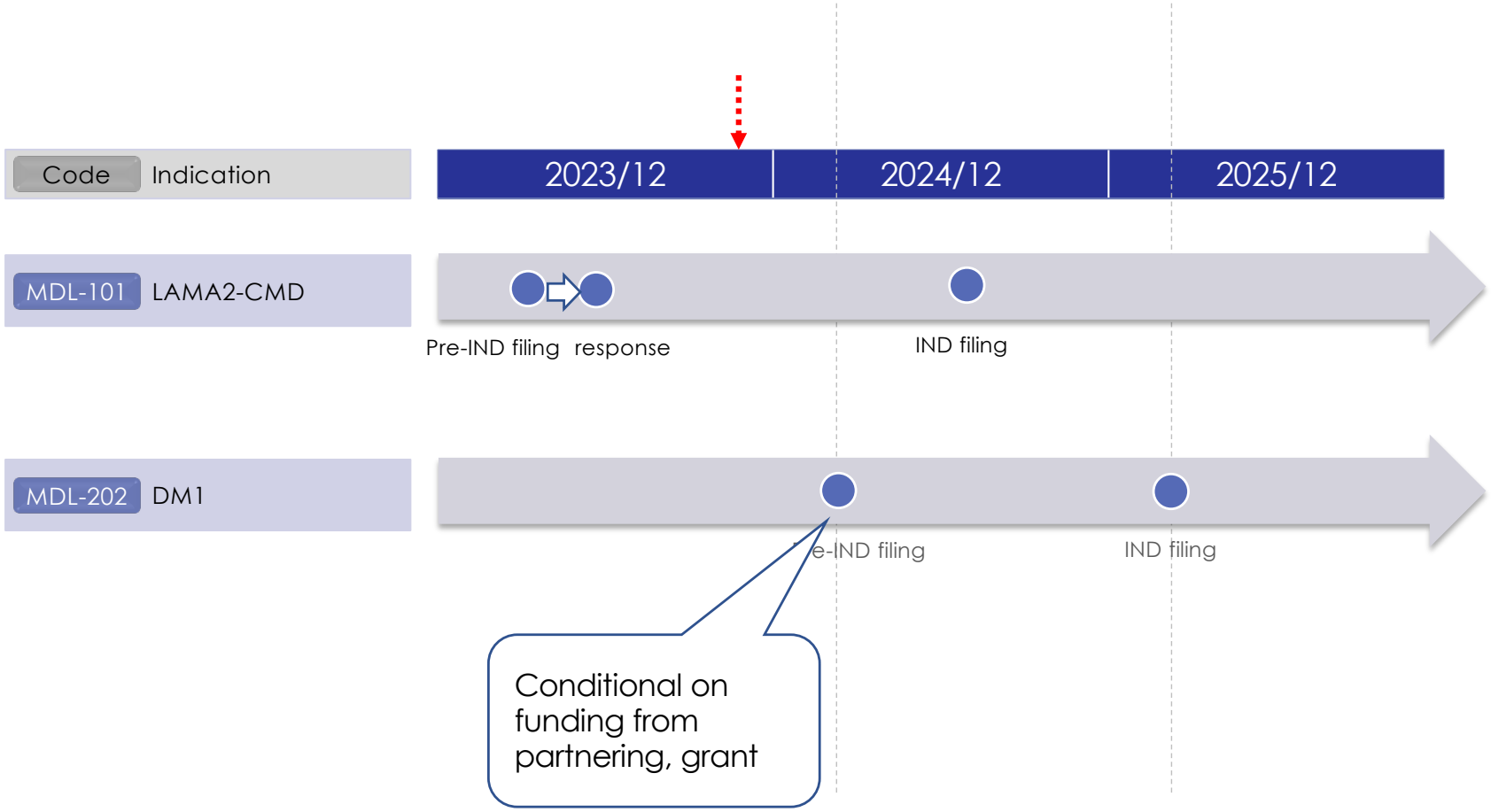
Status of the muscular dystrophy pipeline

MDL-201 and 202 prioritized upon regaining rights



* Milestone events are informational in the future and subject to change

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MODALIS GNDM platform provides a diversified pipeline

Pioneering gene modulation applied to high unmet need indication

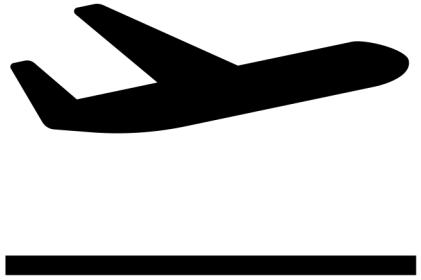
MDL-101

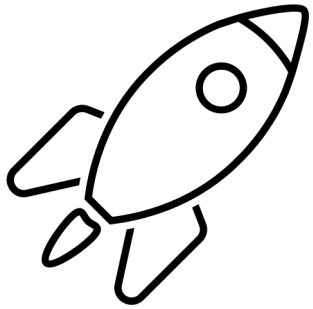
Apply technology to products with larger opportunities

MDL-202
MDL-201

Expand approach to challenging applications

CNS, CV and other programs





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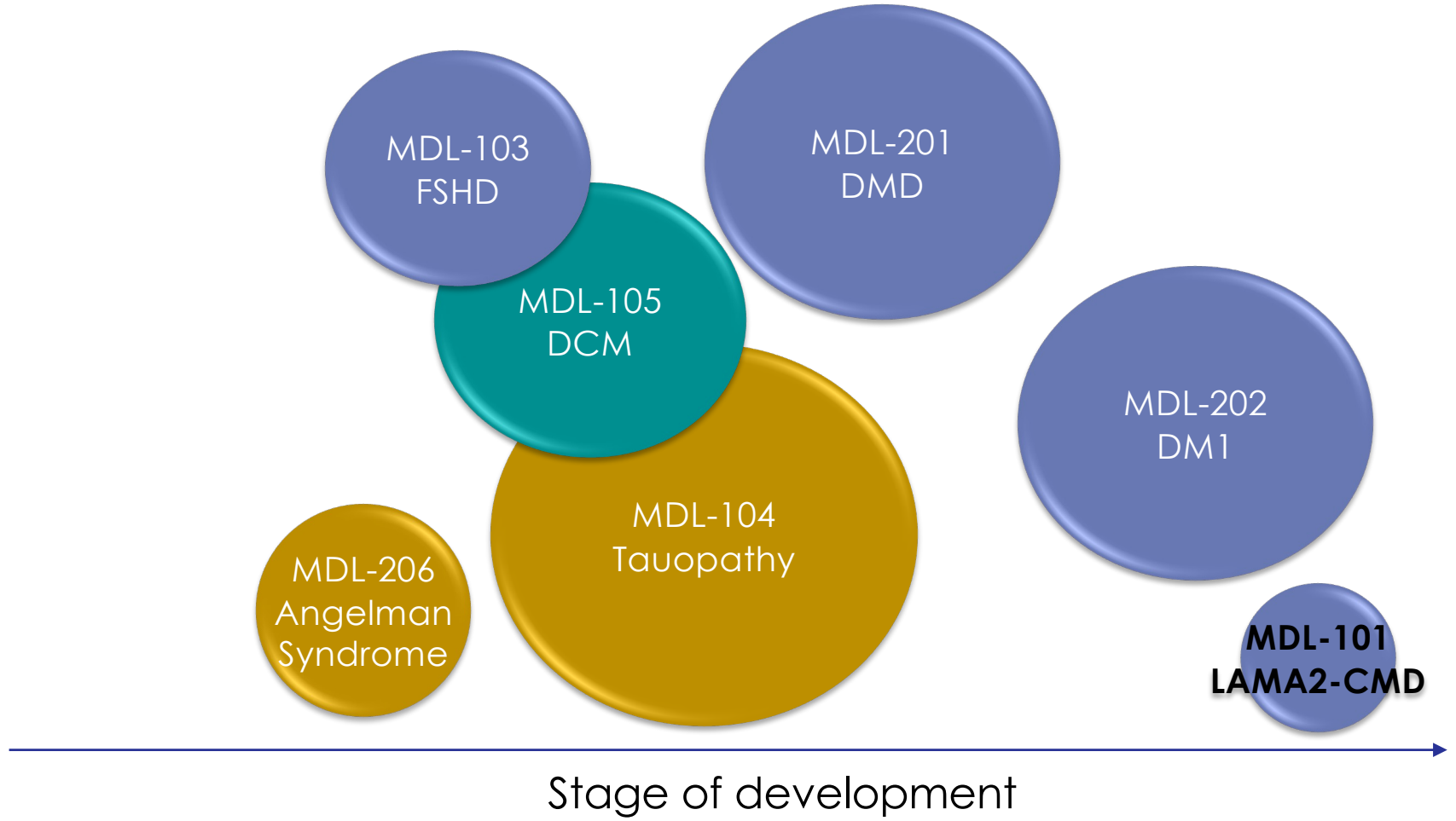
Modalis' pipelines and market size

MDL-101 paves the clinical path to larger indication programs

Modalis programs and market size



※ Size of circles represent market size or patient numbers for each indication

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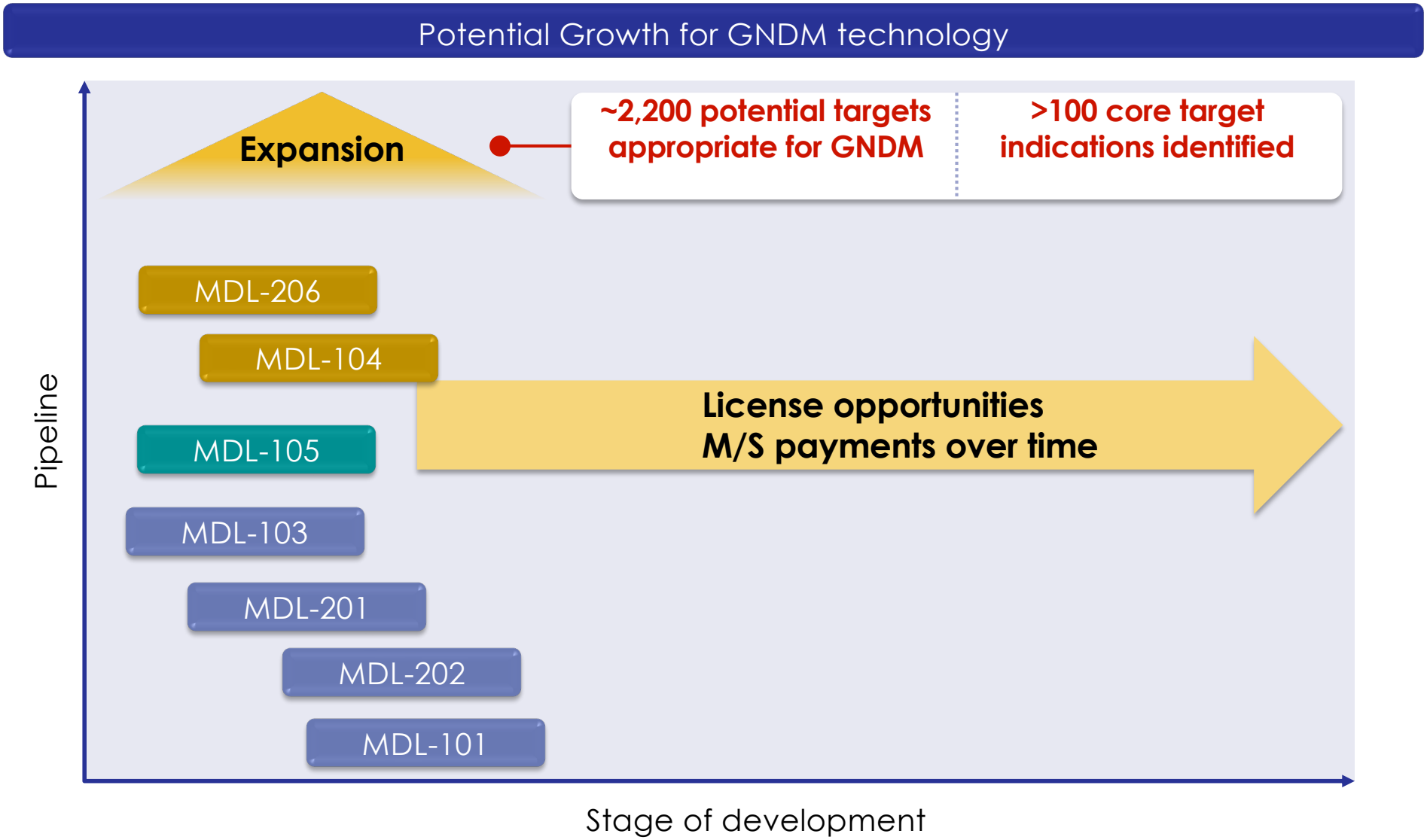
Growth Strategy for Modalis

Opportunities expand two dimensionally



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Partnering strategy

- CRISPR-GNDM® has demonstrated applicability to many high value diseases
- Partnering allows risk / profit sharing for programs developed beyond early PoC
- Partnering will be undertaken when conditions and timing are appropriate based on value and business characteristics of each asset
- Creative deal structures, including licenses, options, and co-development
- Timing and form of alliances to be negotiated to allow accumulation of development know-how, with a view to improving the efficiency of future development and maximizing profits

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Finance

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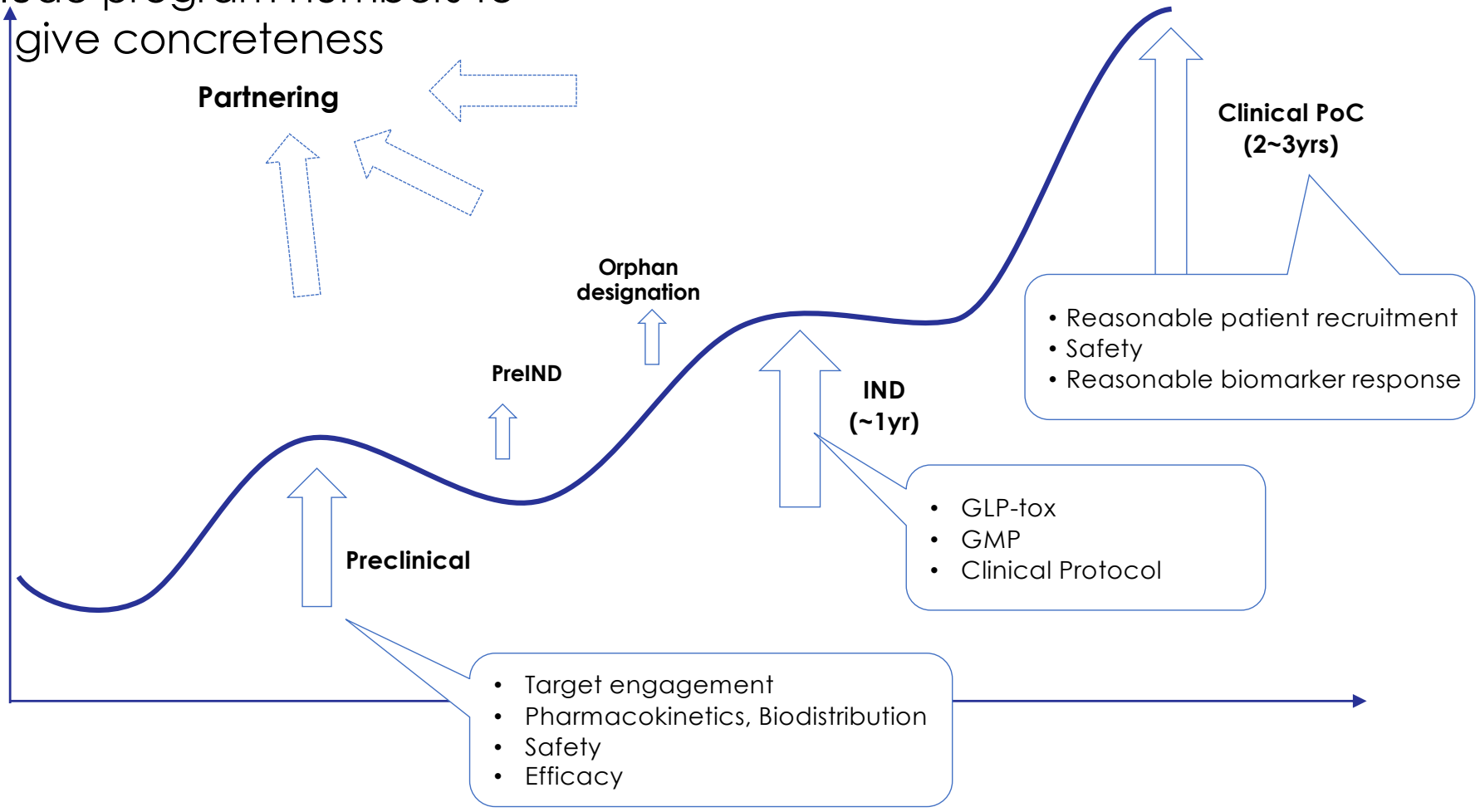
Value increase through future pre-clinical and clinical trials

Expected milestone events and impact on corporate value

To include program numbers to give concreteness



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Why MODALIS?

- **Innovator in the hot space=epigenetic editing**
  - High interest in this approach due to applicability to genetic diseases for which other platforms are not feasible
  - Wide applications in musculoskeletal, CNS, cardiovascular and in other areas
- **Validated technology**
  - Established animal PoC in disease models and target engagement in non-human primates for lead program (MDL-101)
  - Manufacturing, CMC, regulatory are being set up for coming clinical trial
- **Highly efficient platform**
  - rapidly identifies optimized leads and validates in vitro in multiple species
  - Scalable technology for multiple targets
- **Multi-layered IP protection**
  - covers product and technology portfolio
  - Access to CRISPR foundational IPs
- **A value inflection point can be expected in the near term**
  - IND filing within 12-15 months
  - Clinical PoC within 2-3 years

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Q&A

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Q1: The partnership is gone, but is the GNDM over?

- Rather, we believe that this is just the beginning.
- First of all, the field of epigenome editing is attracting a great deal of attention, as other companies backed by huge VC funds are entering the field one after another.
- Among these companies, we are ahead of the pack in development and have accumulated a great deal of know-how and intellectual property.
- The dissolution of an alliance is generally not only a technical issue, but also a strategic issue, including resource allocation on the other side, as it requires a lot of resources as the development stage progresses.
- Therefore, while we do not deny that partnerships are a barometer of evaluation, we do not believe that they are a reflection of everything.
- While we believe that our technology and products will continue to be a competitive advantage, we also believe that we can secure a stronger advantage by incorporating the technological advances of the past few years and improving our products.
- It is also possible that our development could be further accelerated by partnering with another partner.

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Q2: No partnering? Can't do it?

- Once successfully developed, partnering is almost always possible. On the other hand, "when" it can be done before the product is marketed is uncertain, depending on various factors.
- For those partnering, the development costs incurred in addition to the license fees are not negligible, so careful consideration is generally given to the impact on the overall portfolio and the company's overall strategy and budget.
- If you want to partner with a higher degree of certainty and on better terms, the right course of action is to increase the value of the pipeline by using more funds to advance development.
- The funds from this round of financing are sufficient to bring MDL-101 to the clinic, but partnering is essential if we want to further expand our preclinical pipeline.
- We are in ongoing discussions with several companies about partnering opportunities as we update our data, and we are confident that we will have the right size of partnership at the right time.

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Q3: Has MODALIS abandoned platform model?

- When we say "platform-based," the benefit or expectation is that it will scale, or reduce the risk, cost, and development time of a second or subsequent pipeline.
- With CRISPR-GNDM® technology, what Modalis is trying to achieve is to demonstrate with MDL-101 that the platform is a chicken that lays the golden egg, allowing other pipelines that share the platform to develop more efficiently and faster. On the one hand, the platform-type bottleneck is a bottleneck.
- On the other hand, the bottleneck of the platform type is to get people to widely understand that the platform is real, but as is the fate of advanced technology, many challenges can be seen, and it is undeniable that this has been the case in our company as well. However, we overcame these problems and are now approaching clinical trials with our first egg, MDL-101, which has been validated in two animal species. We believe we are proving that chickens are the real deal.
- Once these strategies and their benefits are widely understood (and we believe they are becoming widely understood), we believe there will be business progress, including partnerships, in a wide range of programs.
- In Japan, I feel that there is a high degree of allergy to the drug discovery pipeline (in-house development), but in the U.S., the pipeline is overwhelmingly more valuable than the drug discovery platform.

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Q4: Any implications to the coming milestones?

- The current PIPES fundraising will allow us to bridge the funding needed to start clinical trials.
- In the meantime, the Company expects that,
  - Partnering for MDL-101 will be in place to cover the **costs of the clinical trial** and/or
  - partnering and an **update of the corporate value** of the MDL-202 with the data from the ongoing monkey trial.
- As a result, the Company hopes that both MDL-101 and MDL-202 will enter clinical trials and that the value of the company will increase dramatically after the clinical PoC.

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Q5: What is the current status of non-muscle programs?

- In CNS diseases, AAV based delivery into the brain can be done by either by 1) I**CM** (intracranial) or **ICV** (intracerebroventricular) administration, in which AAVs are delivered into the spinal fluid and infiltrated from outside the brain, or 2) IV (intravenous) administration, in which they are delivered through the **BBB** (blood brain barrier) by some method and into the brain through the blood vessels. The advantage of the first method is that most of the administered genes are in the brain.
- The advantage of #1 is that most of the administered gene is delivered into the brain, which overwhelmingly reduces concerns about toxicity in the liver and other organs. On the other hand, the flip side of advantage #2 is that it is invasive and difficult to deliver genes deep into the brains of monkeys, humans, and other large animals.
- Although we have been considering the use of method #1 for CNS programs such as MDL-104 and 206, we believe that method #2 will become the mainstream method in the future due to technological innovation in the past few years, and we believe that responding to this will be necessary to maintain our competitive advantage in the future.
- Therefore, the Company is in the process of seeking alliances with pharmaceutical or biotech companies that possess such technologies.

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Q6: How will the funds from this fundraising be used?

- The Company plans to use the money to fund the research and development required for its own pipeline, including the re-acquired DM1 pipeline. Specifically, the Company plans to place the highest priority on the funds required for the **MDL-101 IND filing** and to give higher priority to **R&D of MDL-202** and other muscle disease areas. A total of 1,270 million yen is planned to be allocated for this purpose, of which 1,100 million yen will be used for MDL-101 IND and 170 million yen will be used for MDL-202 R&D.
- We believe that this will increase the certainty of the IND of MDL-101. On the other hand, if, in the course of drug development, which is subject to uncertainties, technological innovations or changes in the external environment necessitate costs that exceed reasonable expectations, we will explorer every option including partnering to share R&D costs, or controlling expenditures.

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