

Novartis Enters into Agreement to Acquire Avidity

27 October 2025



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Disclaimer

Additional information and Where to Find It

In connection with the spin-off and the merger (the “Transactions”), Novartis, Avidity and SpinCo intend to file relevant documents with the Securities and Exchange Commission (the “SEC”), including a preliminary and definitive proxy statement to be filed by Avidity. The definitive proxy statement and proxy card will be delivered to the stockholders of Avidity in advance of the special meeting relating to the Transactions. This document is not a substitute for the proxy statement or any other document that may be filed by Avidity with the SEC. AVIDITY'S STOCKHOLDERS ARE URGED TO READ THE DEFINITIVE PROXY STATEMENT IN ITS ENTIRETY WHEN IT BECOMES AVAILABLE AND ANY OTHER DOCUMENTS FILED BY EACH OF NOVARTIS AND Avidity WITH THE SEC IN CONNECTION WITH THE TRANSACTIONS OR INCORPORATED BY REFERENCE THEREIN BECAUSE THEY WILL CONTAIN IMPORTANT INFORMATION ABOUT THE PROPOSED TRANSACTIONS AND THE PARTIES TO THE TRANSACTIONS. Investors and security holders will be able to obtain a free copy of the proxy statement and such other documents containing important information about Novartis and Avidity, once such documents are filed with the SEC, through the website maintained by the SEC at www.sec.gov. Novartis and Avidity make available free of charge at the Novartis website at www.novartis.com/investors/financial-data/sec-filings and Avidity's website at <https://investors.aviditybiosciences.com/sec-filings>, respectively, copies of documents they file with, or furnish to, the SEC.

Participants in the Solicitation

This presentation does not constitute a solicitation of a proxy. Novartis, Avidity and their respective directors, executive officers and certain employees may be deemed to be participants in the solicitation of proxies from the stockholders of Avidity in connection with the Transactions. Information regarding the special interests of these directors and executive officers in the Transactions will be included in the definitive proxy statement referred to above. Security holders may also obtain information regarding the names, affiliations and interests of the Novartis directors and executive officers in the Novartis Annual Report on Form 20-F for the fiscal year ended December 31, 2024, which was filed with the SEC on January 31, 2025. Security holders may obtain information regarding the names, affiliations and interests of Avidity's directors and executive officers in Avidity's definitive proxy statement on Schedule 14A, which was filed with the SEC on April 29, 2025. To the extent the holdings of Avidity's securities by Avidity's directors and executive officers have changed since the amounts set forth in Avidity's definitive proxy statement for its 2025 annual meeting of stockholders, such changes have been or will be reflected on Initial Statements of Beneficial Ownership on Form 3 or Statements of Change in Ownership on Form 4 filed with the SEC. These documents (when available) may be obtained free of charge from the SEC's website at www.sec.gov, the Novartis website at <https://www.novartis.com> and Avidity's website at <https://aviditybiosciences.com>. The contents of the websites referenced above are not deemed to be incorporated by reference into the proxy statement.

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Transaction summary

Overview of transaction terms

Novartis to acquire all outstanding shares of Avidity for USD 72.00 per share in cash, representing a 46% premium to its October 24 closing price

Avidity to separate its early-stage precision cardiology programs into a new SpinCo

Novartis to acquire neuromuscular franchise and follow-on compounds, and platform rights

Closing expected H1 2026, subject to completion of the separation of SpinCo from Avidity and other customary closing conditions

Acquired by Novartis

Del-desiran
(DM1)

Del-brax
(FSHD)

Del-zota
(DMD44)

Preclinical neuromuscular pipeline

AOC 1045 for DMD45 and other undisclosed programs

Platform for extra-hepatic delivery of xRNA

Exclusive rights to platform technologies outside of cardiology
and non-exclusive rights to cardiology indications

DM1 = Myotonic Dystrophy Type 1. FSHD = Facioscapulohumeral Muscular Dystrophy. DMD = Duchenne Muscular Dystrophy.

Agenda

Strategic rationale

Avidity core value drivers

Closing

Strong strategic fit for Novartis

Strengthens Neuroscience franchise

Adds three late-stage neuromuscular programs, building on Zolgensma experience in SMA

Advances xRNA strategy

Adds unique platform of antibody oligonucleotide conjugates, enabling RNA delivery to muscle



Adds first-in-disease pipeline

Del-desiran and del-brax on track to be first-ever disease-modifying therapies for DM1 and FSHD

Enhances mid-long-term growth profile

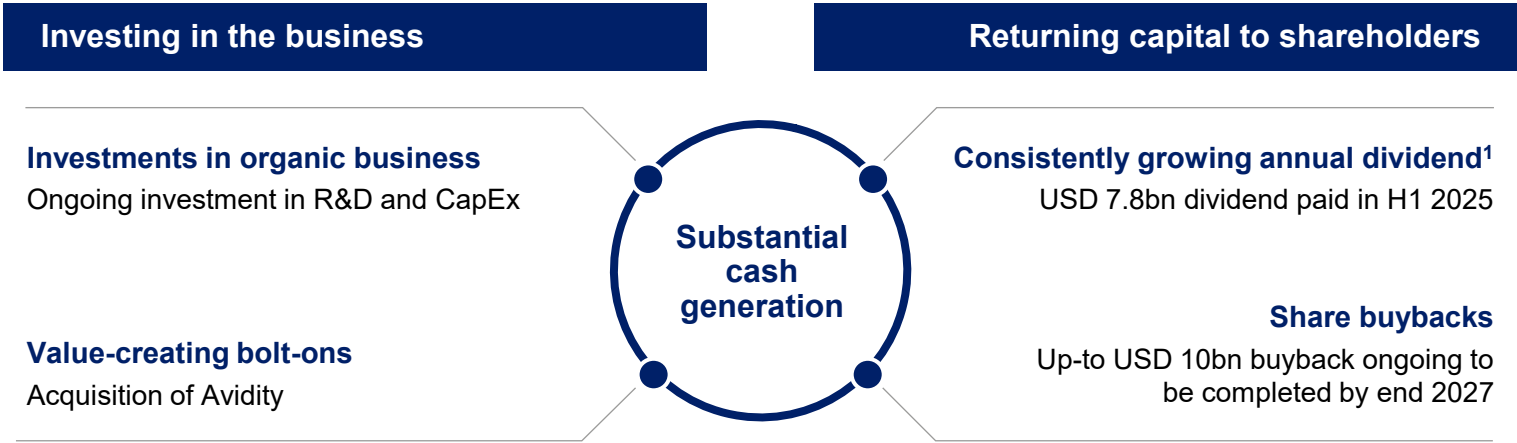
Expected to drive substantial sales and profit growth through 2040s; LOEs not before 2042 and IRA-exempt

Unlocks near-term, multi-billion-dollar opportunities

3 programs expected to launch before 2030, with multi-blockbuster potential in DM1 and FSHD

Transaction in line with capital allocation priorities and target M&A profile; builds on recent Neuroscience deals

Capital allocation priorities



Target M&A profile

- ✓ Strengthens key therapeutic area and advances xRNA strategy
- ✓ First- or best-in-class profile
- ✓ Attractive mid-long-term growth profile
- ✓ Attractive financial return profile

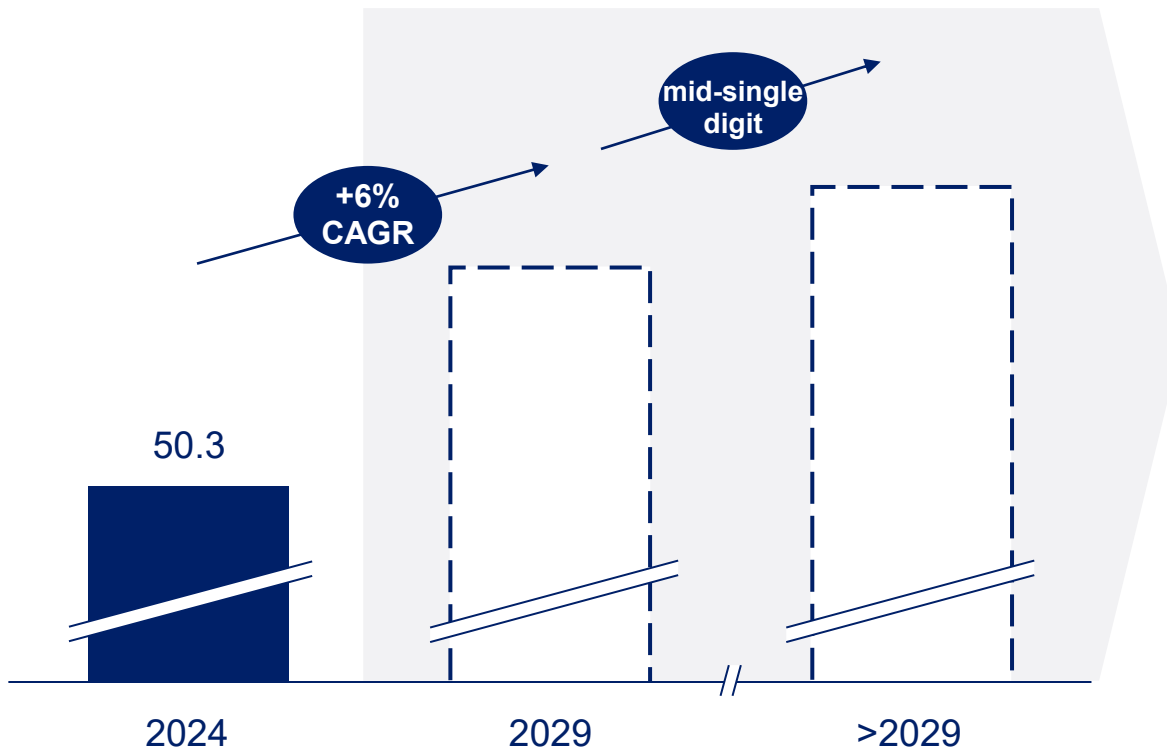
Select Neuroscience deals over the last ~2 years	PTC THERAPEUTICS	DT PHARMA	KATE THERAPEUTICS	arrowhead pharmaceuticals	voyager therapeutics	SIRONAX	BIOARCTIC
	Small molecule (Phase 2)	siRNA (Phase 1)	Gene therapy (preclinical)	siRNA (preclinical)	Gene therapy (preclinical)	Brain shuttle technology	Brain shuttle technology

1. Growing dividend policy in CHF/share

Avidity raises Novartis 2024-2029 sales CAGR from +5% to +6%, and bolsters mid-single digit long-term

Net sales

Illustrative, USD billion, % CAGR cc¹



- Expected near-term product launches with LOEs not before 2042 with no IRA impact
- Substantial sales growth expected by 2029, achieving multi-billion-dollar sales contribution by 2030
- Short-term 1-2%pts core margin dilution; expect to return to 40%+ core margin in 2029
- Strong sales and profit contributions post 2030 support robust top- and bottom-line growth over mid-long term

Deal expected to deliver substantial shareholder returns over time

1. Constant currencies (cc) is a non-IFRS measure. Details regarding non-IFRS measures can be found starting on page 40 of the Q2 2025 Interim Financial Report.

Agenda

Strategic rationale

Avidity core value drivers

Closing

Avidity brings pioneering AOC™ platform for RNA therapeutics, with industry-leading delivery of RNA to muscle

Combining the specificity of mAbs with the precision of oligonucleotide therapies



AOC platform advantages

Ability to **target new tissue** and cell types beyond the liver

Flexibility to **select and deploy the most potent oligonucleotides** (e.g. siRNAs, PMOs)

Maximizes therapeutic durability, enabling infrequent dosing

Readily **reproducible and scalable**

Avidity core value drivers

Select assets

Del-desiran in DM1	~80k patients in the US and Europe	On track to be 1st approved drug for DM1 (Phase 3 fully enrolled; readout expected 2026)	Designed to address underlying cause of myotonic dystrophy by liberating free MBNL
Del-brax in FSHD	~45-87k patients in the US and Europe	On track to be 1st approved drug for FSHD (Phase 3 underway; potential for accelerated approval)	Targets aberrant expression of DUX4 mRNA, the root cause of FSHD
Del-zota in DMD44	~900 patients in the US	Aligned on path for accelerated approval in the US (FDA submission expected 2026)	Designed to facilitate exon skipping to produce functional, near full-length dystrophin

DM1: Rare progressive neuromuscular disorder with a poor prognosis and no disease-modifying therapies

~80k

Patients with DM1 in the US and EU

Zero

Currently approved disease-modifying therapies

Underrecognized, progressive and often fatal neuromuscular disease that primarily affects skeletal, cardiac and smooth muscle

Increases in severity from generation to generation

Significant impact on quality of life due to muscle weakness and wasting, myotonia, and often cardiac and pulmonary comorbidities, which contribute to reduced lifespan

Current standard of care is limited to physical and pharmacological symptom management with **no disease-modifying therapy available**

***Del-desiran* is designed to address root cause of DM1** by degrading DMPK mRNA

Del-desiran has FDA Orphan Drug, Fast Track and Breakthrough Therapy designations, and EMA Orphan Drug designation

Del-desiran is designed to address the underlying cause of DM1

Scientific rationale

- DM1 caused by CTG trinucleotide repeat expansion in the DMPK gene
- CTG expansion changes mRNA structure such that the mRNA sequesters splicing factors, including MBNL, leading to loss of normal cell function and muscle wastage
- Del-desiran degrades DMPK mRNA in muscle cells and restores normal MBNL function and splicing

Ph1/2 MARINA study

- ✓ Delivery to muscle
- ✓ Target engagement
- ✓ Restoration of splicing

Ph1/2 MARINA-OLE

Efficacy measures

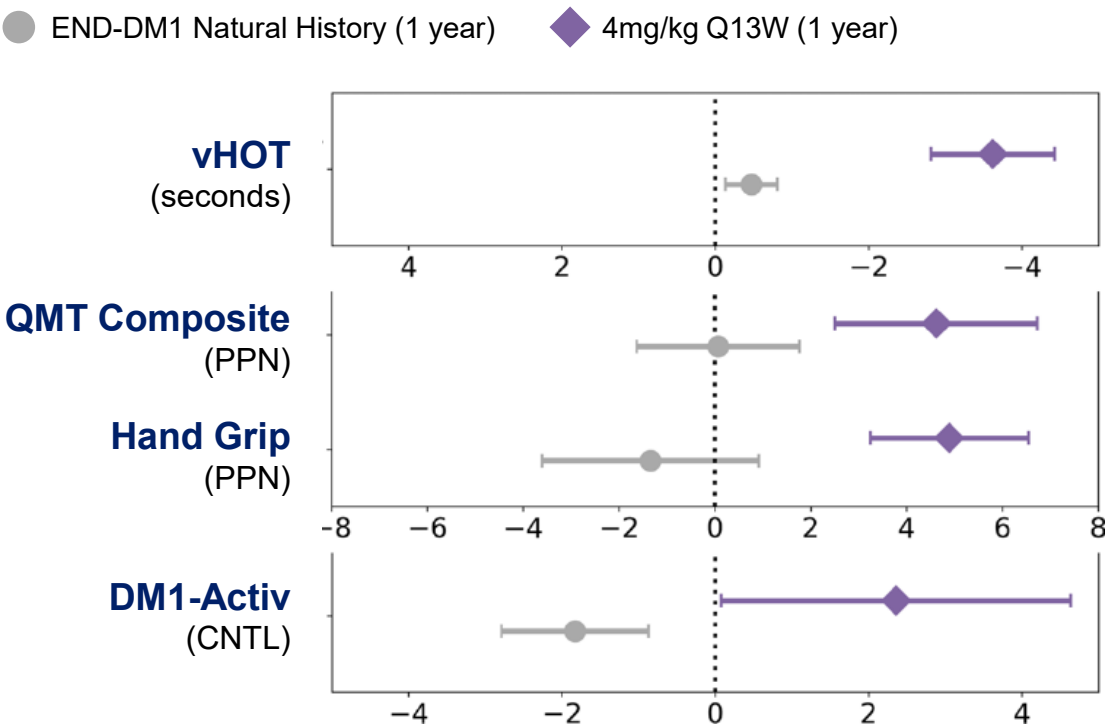
Myotonia	Video Hand Opening Time (vHOT)	
Strength	Hand Grip	Quantitative Muscle Testing (QMT)
Activities of daily living	DM1-Activ ¹	

Data shown as mean and standard error. Fold change is calculated per subject as post-treatment relative to baseline; *D<0.05 unpaired + test.
1. Rasch-built Myotonic Dystrophy Type 1 Activity and Participation Scale, designed to assess activities of daily living in individuals with DM1.

Wagner, SD, et al. PLOS Genet. 2016;12(9):e1006316.

Phase 1/2 MARINA study: *Del-desiran* demonstrated potential to be a transformational therapy for DM1 patients

MARINA-OLE efficacy data



Efficacy and safety overview

Met efficacy endpoints

Reversal of disease progression in MARINA and MARINA-OLE compared to END-DM1 natural history data

Consistent and durable improvement in multiple functional endpoint assessments

Improvements across the domains of **myotonia, strength and mobility**, driven by significant DMPK knockdown

Favorable safety and tolerability

All 37 patients enrolled remain on study, and all related AEs were **mild or moderate**

Most common related AE in >2 participants was nausea

No study drug-related treatment discontinuations or SAEs

vHOT = video hand opening time | QMT composite = quantitative muscle testing | PPN = percent predicted normal | DM1-Activ = Rasch-built myotonic dystrophy type 1 activity and participation scale.
Note: Data as of August 2024; AE data from MARINA-OLE and exposure data from MARINA and MARINA-OLE; SAEs considered unrelated to treatment included obstructive pancreatitis, rectal perforation, vomiting, basal cell carcinoma, invasive ductal breast carcinoma, atrial fibrillation, chest pain, and cholelithiasis; the unrelated SAEs are either consistent with DM1 or commonly seen in clinical trials.

Phase 3 HARBOR study of *del-desiran* in DM1 fully enrolled

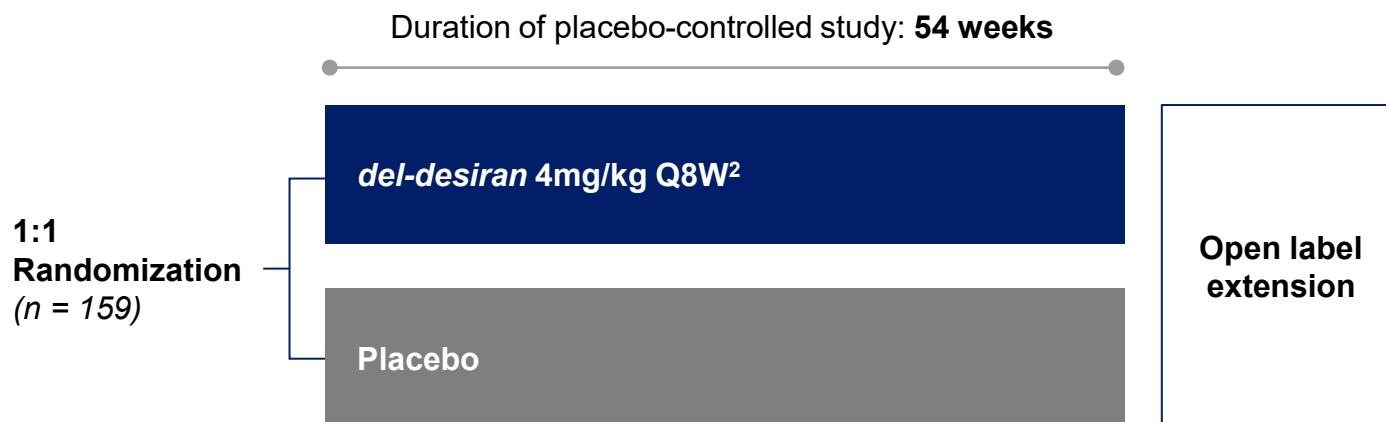
Global pivotal trial

- FDA, EMA and other regulatory authorities aligned on study design
- Completed enrollment in July 2025
- Participants eligible to roll over into OLE study
- ~40 global sites

Clinical endpoints

- **Primary:** Video Hand Opening Time (vHOT)
- **Key secondaries:** Hand Grip, Quantitative Muscle Testing, DM1-Activ¹

Study design



Population: Age ≥16, with clinical and genetic diagnosis of DM1 (≥100 repeats)

Next steps: 54-week readout expected in H2 2026; global regulatory submissions expected in 2027 (base case)

1. Rasch-built Myotonic Dystrophy Type 1 Activity and Participation Scale, designed to assess activities of daily living in individuals with DM1. 2. Initiating dose of 2 mg/kg of *del-desiran*.

FSHD: Rare hereditary disorder causing relentless loss of muscle function and progressive disability

~45-87k

Patients with FSHD in the US and EU

Zero

Currently approved therapies

One of the **most common forms of muscular dystrophy**, causing progressive muscle weakness, pain, fatigue and disability

Onset typically occurs in teenage or early adult years, with **steady loss of independence**; 20% of patients become **wheelchair dependent**

Caused by **aberrant expression of the DUX4 gene**, leading to cell death, immune response and oxidative stress

Autosomal dominant disorder, potentially **affecting multiple generations**; 20-30% of cases arise from **spontaneous mutations**

***Del-brax* is designed to address the root cause of FSHD**, and the only asset to demonstrate disease-modifying potential for FSHD in the clinic

Del-brax has FDA Orphan Drug and Fast Track designations, and EMA Orphan Drug designation

Phase 1/2 FORTITUDE study: *Del-brax* improved functional mobility, strength and upper limb function compared to patients treated with placebo

Data at 12 months with Q13W dosing¹



Efficacy and safety overview

Met efficacy endpoints

Consistent **improvement of functional mobility and muscle strength** as measured by 10MWRT, TUG and QMT vs. placebo

Consistent **improvement in QoL** as measured by patient reported outcomes vs. placebo

Rapid and significant reductions in levels of cDUX and creatine kinase (CK), a key marker of muscle damage

Favorable safety and tolerability

All participants enrolled **remain in study**

No discontinuations

Most AEs mild/moderate, and no related severe or serious AEs

10MWRT = 10-meter walk-run test | TUG = timed up-and-go; maximal effort | QMT = quantitative muscle testing | PPN = percent predicted normal | RWS = reachable workspace | RSA = relative surface area. ¹ Q13W dosing with 1 booster after first 6 weeks. Note: Most common related AEs occurring in >3 participants were fatigue and decreased hemoglobin; one unrelated severe, non-serious AE of herpes zoster occurred in one participant; three unrelated severe, serious AEs of radius fracture, pelvic fracture, and fractured sacrum occurred in one participant.

Compelling data in FSHD supports cDUX biomarker as a potential accelerated approval endpoint as upside case

Direct link to FSHD

cDUX, a direct target of DUX4, **elevated 6- to 9-fold in people living with FSHD** compared to healthy volunteers

Elevated cDUX levels **linked to worsening disease** and muscle weakness

Predictive of clinical activity

Significant and rapid reductions in cDUX and CK in FORTITUDE participants following del-brax treatment

Improved functional mobility and muscle strength in FORTITUDE consistent with changes seen for cDUX and CK

FORTITUDE biomarker cohort

Biomarker cohort in Ph1/2 FORTITUDE study ongoing

FDA confirmed potential for accelerated approval based on reduction in cDUX

Biomarker cohort readout expected Q2 2026; **base case remains filing with Phase 3 data**

Phase 3 FORTITUDE-3 study of *del-brax* in FSHD underway

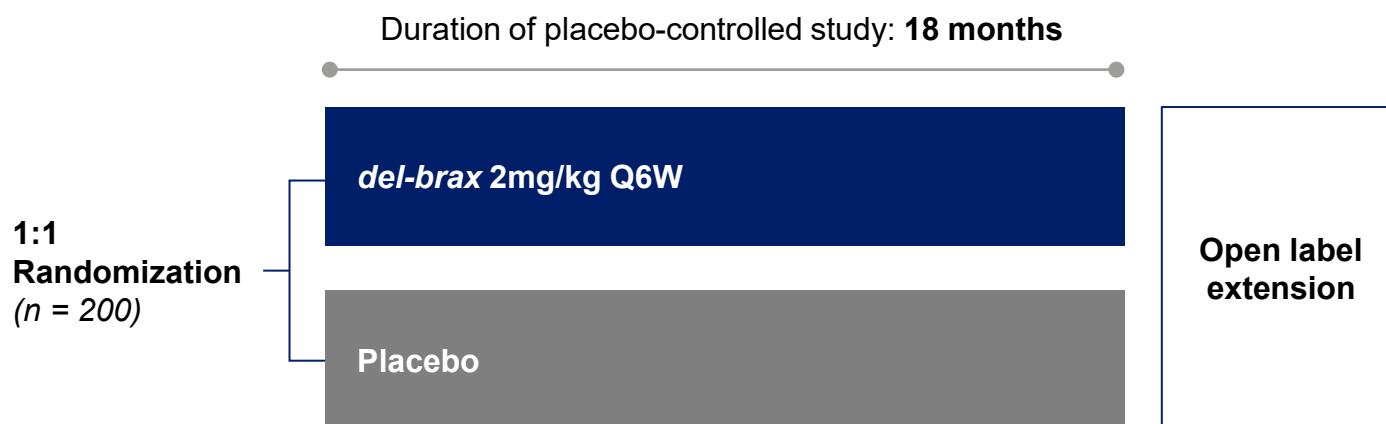
Global pivotal trial

- Intended to serve as confirmatory study for full approval
- Participants eligible to roll over into OLE study
- ~45 sites across North America, Europe, Japan

Clinical endpoints

- **Key registrational¹:** 10-Meter Walk-Run Test, Timed Up-and-Go, Quantitative Muscle Testing
- **Additional:** Signs and symptoms of FSHD, cDUX and CK biomarkers

Study design



Population: Age 16-70, with clinical and genetic diagnosis of FSHD

Next steps: Phase 3 readout and global regulatory submissions expected in 2028 (base case)

1. Final hierarchy will be selected prior to data base lock, based on evaluation of latest natural history data and efficacy data from FORTITUDE biomarker cohort; currently, QMT is assigned as the primary endpoint.

DMD: Severe, early-onset disease marked by progressive muscle damage and reduced life expectancy

~10-15k

Patients with DMD in the US;
similar prevalence in the EU

~900

Patients with DMD44
in the US

Monogenic, X-linked (primarily affecting males), recessive condition characterized by **progressive muscle damage and weakness**

Symptom onset and diagnosis by 4 years of age; leads to **loss of ambulation by teenage years** and **significantly reduced life expectancy**

Caused by **mutations in the DMD gene**, which encodes for the dystrophin protein

~6-7% of patients have mutations amenable to exon 44 skipping (DMD44)

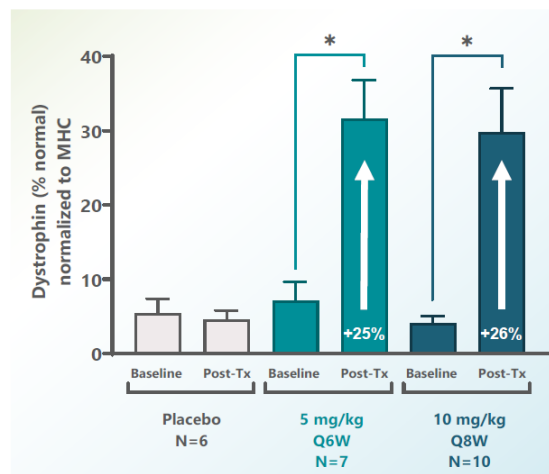
Del-zota is **designed to specifically skip exon 44** of dystrophin gene to produce functional, near full-length dystrophin

Del-zota has FDA Orphan Drug, Fast Track, Breakthrough Therapy and Rare Pediatric Disease designations, and EMA Orphan Drug designation

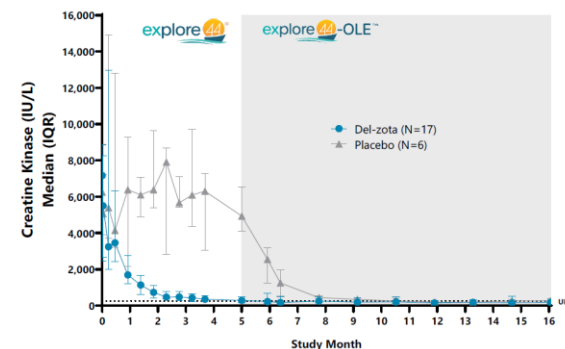
Phase 1/2 EXPLORE44 study: *Del-zota* showed improvements across key biomarkers and functional endpoints, with favorable safety and tolerability

EXPLORE44 efficacy data

Increase in dystrophin



Reduction in CK



Efficacy and safety overview

Met efficacy endpoints

~40% increase in exon skipping across dose cohorts

~25% increase in dystrophin production, with up to 58% normal dystrophin recovered

>80% reduction in CK levels, a key marker of muscle damage

Consistent, clinically meaningful improvements across functional endpoints at ~1 year

Favorable safety and tolerability

Most TEAEs were mild or moderate

3 participants discontinued due to hypersensitivity

Next steps: FDA submission for accelerated approval expected in 2026

Note: In EXPLORE44, most common TEAEs occurring in ≥ 3 participants in the del-zota arms include procedural pain and headache.

Novartis has the commercial capabilities and neuromuscular experience to maximize all three near-term launches

Rare disease capabilities

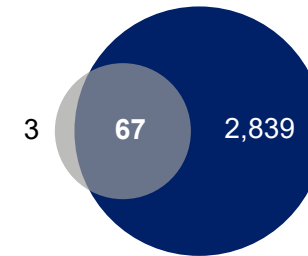
- **Deep understanding of patient journeys** in rare/ultra-rare diseases such as SMA, PNH and C3G
- **Leading patient identification and activation capabilities**, proven across multiple launches
- **Leading payer engagement capabilities** to enable fast and effective access across a broad, diverse portfolio
- **Field structure** ready to deploy across all neuromuscular indications
- **Best-in-class, scalable support programs** for patients and HCPs



Coverage of diagnosing Neurologists

DMD

~90% overlap

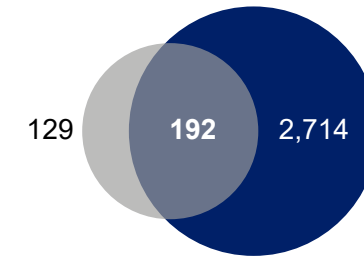


● DMD Diagnosing Neurologists¹ (n=70)

● SMA Prescribers² (n=2,906)

FSHD

~60% overlap

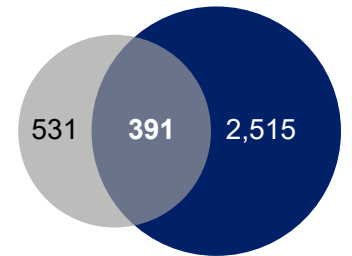


● FSHD Diagnosing Neurologists¹ (n=321)

● SMA Prescribers² (n=2,906)

DM1

~40% overlap



● DM1 Diagnosing Neurologists¹ (n=922)

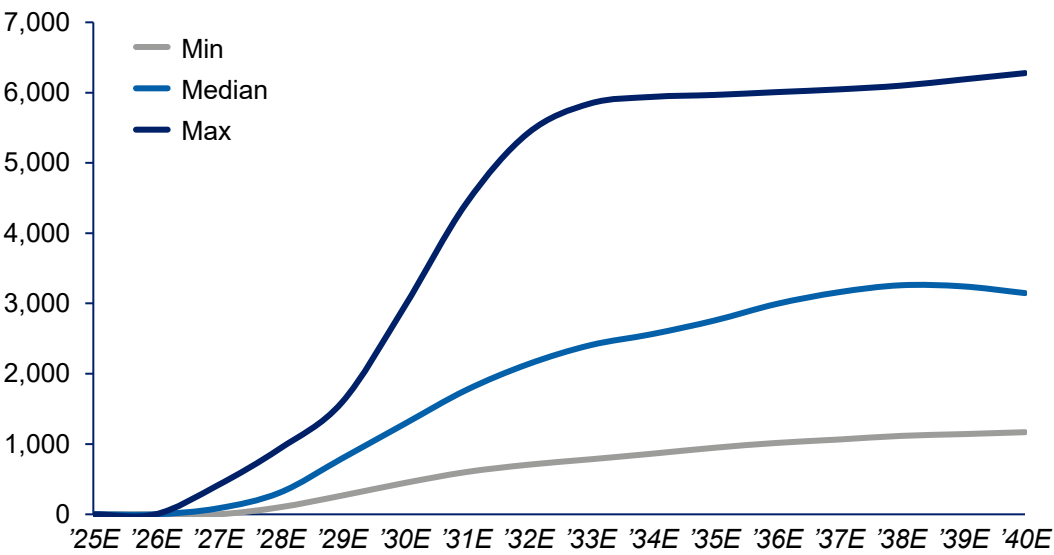
● SMA Prescribers² (n=2,906)

Avidity is highly synergistic with our commercial footprint in the rare neuromuscular space

1. Top 3 deciles, based on diagnosed patients. 2. SMA Prescribers: HCPs with at least one patient for Zolgensma and competitors (Spinraza, Evrysdi) since 2017. Source: Compile Claims, Compile Rx-Claims, Jan'17-Jun'25.

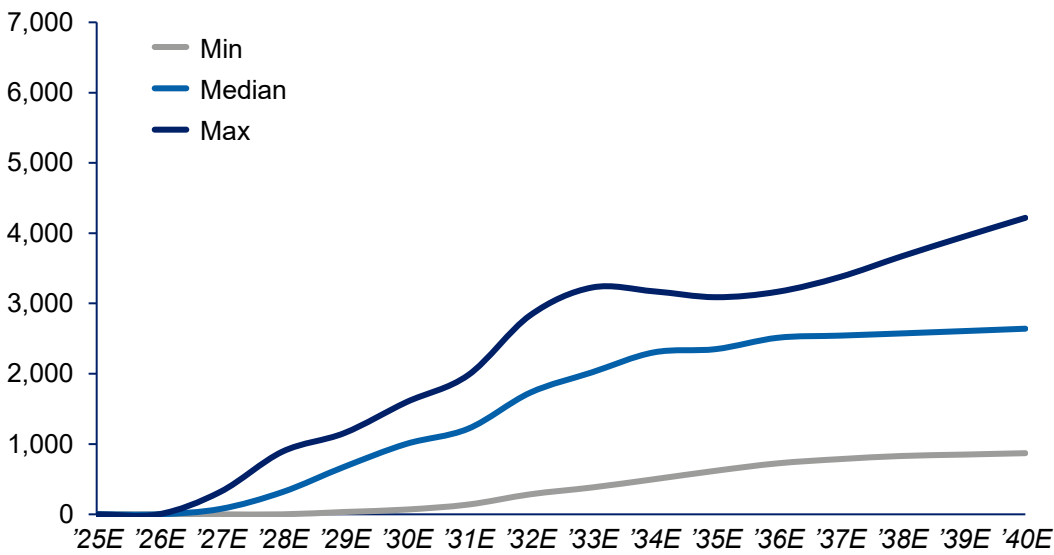
External forecasts indicate multi-billion-dollar peak sales potential in DM1 and FSHD

WW del-desiran unprobabilized revenues (USDm)



US LOE not before 2042, not subject to IRA
EU LOE not before 2044

WW del-brax unprobabilized revenues (USDm)



US LOE not before 2043, not subject to IRA
EU LOE not before 2044

Agenda

Strategic rationale

Avidity core value drivers

Closing

Financial highlights

Transaction summary

- Acquiring all outstanding shares of Avidity for **USD 72.00/share**
- Total transaction value of **USD 12bn** on a fully diluted basis, representing an enterprise value of ~USD 11bn at the expected closing date
- Expected to close in **H1 2026**, subject to the separation of SpinCo from Avidity and other customary closing conditions



Deal value

- **Multi-billion-dollar peak sales opportunities** in DM1 and FSHD, with LOEs not before 2042 and no IRA impact
- **Expected near-term launches** with path to commercialization for 3 late-stage programs before 2030
- **Exclusive rights to xRNA platform** outside of cardiology



Financial impact

- **Raises Novartis expected 2024-2029 sales CAGR from +5% to +6%**, bolsters mid-single digit growth long-term
- **Short-term 1-2%pts core margin dilution**, with return to 40%+ core margin in 2029
- Expected IRR well in excess of cost of capital with **significant value creation**



Capital allocation priorities

- Transaction **fits with capital allocation priorities** and target M&A profile
- **No change** to Novartis' capital allocation strategy post-close

