Wave Life Sciences

Corporate Presentation

November 20, 2024

WAVE™
LIFE SCIENCES

Forward-looking statements

This document contains forward-looking statements. All statements other than statements of historical facts contained in this document, including statements regarding possible or assumed future results of operations, preclinical and clinical studies, business strategies, research and development plans, collaborations and partnerships, regulatory activities and timing thereof, competitive position, potential growth opportunities, use of proceeds and the effects of competition are forward-looking statements. These statements involve known and unknown risks, uncertainties and other important factors that may cause the actual results, performance or achievements of Wave Life Sciences Ltd. (the "Company") to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. In some cases, you can identify forward-looking statements by terms such as "may," "will," "should," "expect," "plan," "aim," "anticipate," "could," "intend," "target," "project," "contemplate," "believe," "estimate," "predict," "potential" or "continue" or the negative of these terms or other similar expressions. The forward-looking statements in this presentation are only predictions. The Company has based these forward-looking statements largely on its current expectations and projections about future events and financial trends that it believes may affect the Company's business, financial condition and results of operations. These forward-looking statements speak only as of the date of this presentation and are subject to a number of risks, uncertainties and assumptions, including those listed under Risk Factors in the Company's Form 10-K and other filings with the SEC, some of which cannot be predicted or quantified and some of which are beyond the Company's control. The events and circumstances reflected in the Company's forward-looking statements may not be achieved or occur, and actual results could differ materially from those projected in the forward-looking statements. Moreover, the Company operates in a dynamic industry and economy. New risk factors and uncertainties may emerge from time to time, and it is not possible for management to predict all risk factors and uncertainties that the Company may face. Except as required by applicable law, the Company does not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise.

Building a leading RNA medicines company

Novel RNA medicines platform (PRISM®)

- Multi-modal: RNA editing, RNAi, splicing, allele-selective silencing
- Best-in-class, clinically-validated oligonucleotide chemistry (PN, stereochemistry)

Differentiated RNA medicines pipeline

WVE-006 in AATD

WVE-007 in Obesity

WVE-N531 in DMD

WVE-003 in HD

Strong and broad IP | **In-house GMP manufacturing** | **Well-capitalized with cash runway into 2027***

AATD: Alpha-1 antitrypsin deficiency DMD: Duchenne muscular dystrophy HD: Huntington's disease

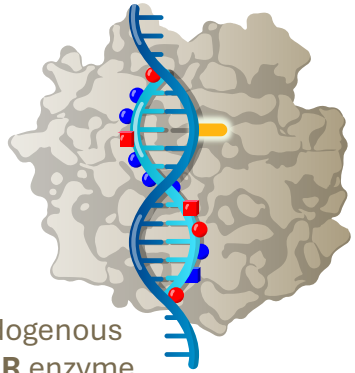
*Cash runway does not include potential future milestones or opt-in payments under GSK collaboration

Wave's best-in-class multi-modal platform

Clinically-validated oligonucleotide chemistry (including PN, stereochemistry)

Editing

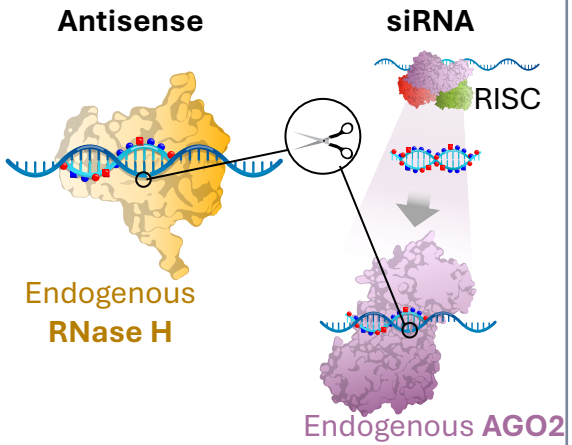
Efficient editing of RNA bases to **restore** or **modulate** protein production



WVE-006 (AATD)
PNPLA3 (Liver disease)**, LDLR, APOB** (HeFH)

Silencing

Degradation of RNA transcripts to **turn off** protein production

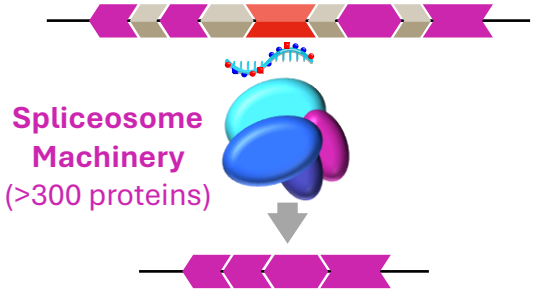


WVE-003 (HD)

WVE-007 (obesity)

Splicing

Restore RNA transcripts and **turn on** protein production



Restored **Reading Frame** for **Functional Protein**

WVE-N531 (DMD)

Wave has driven foundational advances in nucleic acid chemistry to expand platform technologies and develop next generation of RNA therapeutics

Further information can be found in recent platform publications

Silencing (RNase H and Ago2) | **Splicing** | **Editing**

Full list of Wave publications: https://ir.wavelifesciences.com/events-publications/publications

Three clinical updates in 2024 demonstrate continued platform translation

Proprietary PRISM platform

- Stereopure oligonucleotides
- Novel backbone modifications (including PN chemistry)
- Novel base and sugar chemistry modifications

Therapeutic modalities	Preclinical publication	Clinical translation	Clinical trial results
Splicing (WVE-N531 for DMD)	✓	✓ 53% exon skipping, 42 µg/g muscle tissue concentrations in 6 weeks	✓ *9.0% mean muscle-adjusted dystrophin; safe and tolerable (interim analysis)* FORWARD-53
Allele-selective silencing (WVE-003 for HD)	✓	✓ 35% allele-selective mHTT silencing with single dose	✓ 46% allele-selective mHTT silencing; correlation with slowing of caudate atrophy SELECT HD
GalNAc-RNA editing (WVE-006 for AATD)	✓	✓ First ever RNA editing achieved; 11 µM total AAT protein, >60% (6.9 µM) M-AAT with single dose	*Multidose data expected in 2025* RESTORAATION
GalNAc-RNAi (WVE-007 for obesity)	✓	*Clinical trial initiation expected 1Q 2025*	

Robust, diversified RNA medicines pipeline including first-in-class RNA editing programs

Program	Discovery	IND / CTA Enabling Studies	Clinical	Rights	Patient population (US & Europe)
RNA EDITING					
WVE-006 SERPINA1 (AATD)			RestorAATion Clinical Program	GSK exclusive global license	200K
GalNAc-AIMer PNPLA3 (liver disease)				100% global	9M
GalNAc-AIMer LDLR (HeFH)				100% global	900K (30M expansion)
GalNAc-AIMer APOB (HeFH)				100% global	70K
RNAi					
WVE-007 (GalNAc) INHBE (Obesity and other metabolic disorders)				100% global	47M
GalNAc-siRNA Undisclosed				100% global	--
SPLICING					
WVE-N531 Exon 53 (DMD)			FORWARD-53 Trial (Phase 2)	100% global	2.3K
Other exons (DMD)				100% global	Up to 18K
ALLELE-SELECTIVE SILENCING					
WVE-003 mHTT (HD)			SELECT-HD Trial (Phase 1b/2a) - *Trial Completed*	100% global	25K Symptomatic (SNP3) 60K Pre-Symptomatic (SNP3)

Editing for correction Editing for upregulation

WAVE™ LIFE SCIENCES

AATD: Alpha-1 antitrypsin deficiency; DMD: Duchenne muscular dystrophy; HD: Huntington's disease; HeFH: heterozygous familial hypercholesterolemia

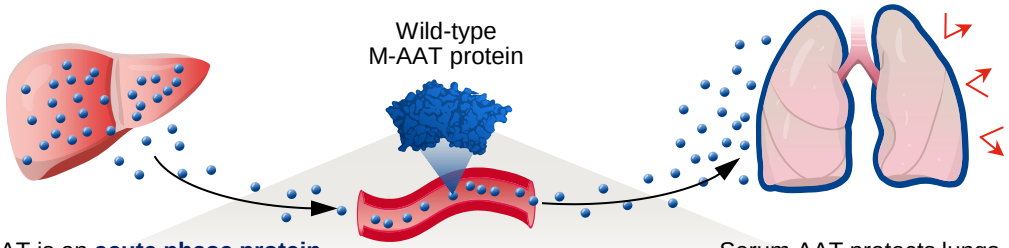
WVE-006
RNA editing (AIMers)

Alpha-1 antitrypsin deficiency (AATD)

WAVE™ LIFE SCIENCES

WVE-006: GalNAc-conjugated AIMer designed to correct mutant SERPINA1 transcript to address both liver and lung manifestations of AATD

Healthy



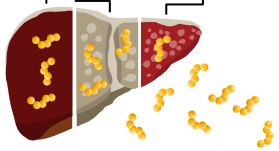
AAT is an **acute phase protein** predominantly produced by hepatocytes

Serum AAT protects lungs against neutrophil elastase

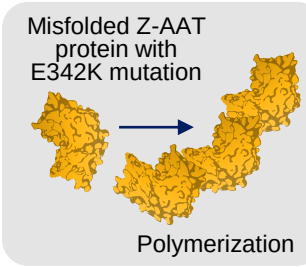
AATD

Gain of Function: Liver Disease

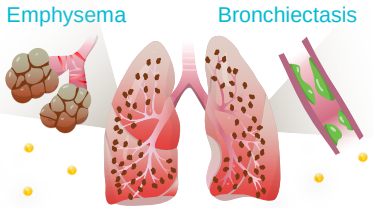
Fibrosis → Cirrhosis → Hepatocellular Carcinoma



Z protein causes AAT proteotoxic stress, leading to progressive liver disease



Loss of Function: Lung Disease



Low serum AAT leads to lung disease

WVE-006 for AATD

SERPINA1 Z allele mRNA encodes Z-AAT protein with E342K mutation

Edited *SERPINA1* mRNA enables wild-type M-AAT protein production

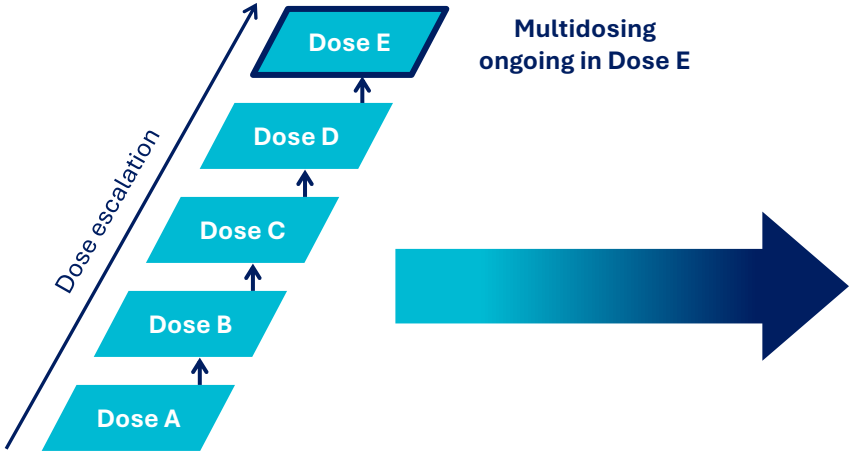
- ✓ Subcutaneous injection (GalNAc)
- ✓ Infrequent dosing
- ✓ Highly specific (No bystanders)

Strnad et al., 2020 *N Engl J Med* 382:1443-55; Stoller et al., 1993 Alpha-1 Antitrypsin Deficiency GeneReviews.

RestorAATion-1 and RestorAATion-2 ongoing

RestorAATion-1: Healthy Volunteers → **RestorAATion-2: AATD Patients**

Single ascending dose (SAD) → Multiple-ascending dose (MAD) cohorts



Multidosing ongoing in Dose E

Up to 7 doses

Cohort 3

Cohort 2

Cohort 1
200 mg

Study key objectives

Safety and tolerability
Pharmacokinetics
Serum M-AAT levels

HV: healthy volunteer; SAD: single-ascending dose; MAD: multi-ascending dose

Achieved proof-of-mechanism for Wave's RNA editing platform

Proof-of-mechanism after a single dose in RestorAATion-2

- **Circulating wild-type M-AAT protein in plasma:** Mean of 6.9 µM at day 15; more than 60% of total AAT
- **Increases in neutrophil elastase inhibition from baseline:** Consistent with production of functional M-AAT
- **Mean total AAT protein:** Increased from below level of quantification at baseline to 10.8 µM at day 15
  - Meets level that has been the basis for regulatory approval for AAT augmentation therapies
- **Increases in total AAT from baseline and M-AAT protein:** Observed as early as day 3 and through day 57
- WVE-006 well tolerated with a favorable safety profile; all AEs mild-to-moderate, no SAEs

Wave expects to share multidose data from RestorAATion-2 in 2025

October 16, 2024 Proof-of-mechanism disclosure on first two "ZZ" AATD patients in first dose cohort of RestorAATion-2 to reach day 57;

Strategic collaboration with GSK to develop transformative RNA medicines

Collaboration Highlights

- $170 million upfront[1]
- Additional research funding
- Potential for up to $3.3 billion in milestones[2]
- Leverage GSK's expertise in genetics and genomics

	Maximize global potential for WVE-006 for AATD	Advance up to eight GSK collaboration programs	Expand Wave's pipeline
	Up to $525 million in total milestones and **tiered royalties on net sales**	**Up to $2.8 billion** in total milestones and **tiered royalties on net sales**	Wave to advance up to **three wholly owned collaboration programs** (or more with GSK's consent)[3]
Recent Highlights	✓ **$20 million milestone** with first individual dosing RestorAATion-2 trial underway (AATD patients)	✓ **$12 million aggregate initiation payment** for GSK's selection of two programs to advance	✓ **INHBE is Wave's first wholly owned program** emerging from GSK collaboration

1. $120 million in cash and $50 million equity investment; 2. Initiation, development, launch, and commercialization milestones for WVE-006 and programs progressed during initial 4-year research term (8 GSK collaboration programs); 3. GSK eligible to receive tiered royalty payments and commercial milestones from Wave

Wholly owned GalNAc-AIMer programs

New targets meet key criteria, expected to improve probability of success:

- Strongly supported by human genetics
- Leverage unique platform capabilities; GalNAc-AIMers building on learnings of WVE-006
- Completely novel ways of treating diseases with high unmet need
- Readily accessible biomarkers and approaches to assess PD, defined regulatory paths

Correction of PNPLA3
Genetically defined liver disease

Upregulation of LDLR
Familial hypercholesterolemia

Correction of APOB
Familial hypercholesterolemia

PNPLA3, LDLR, APOB clinical candidates expected in 2025

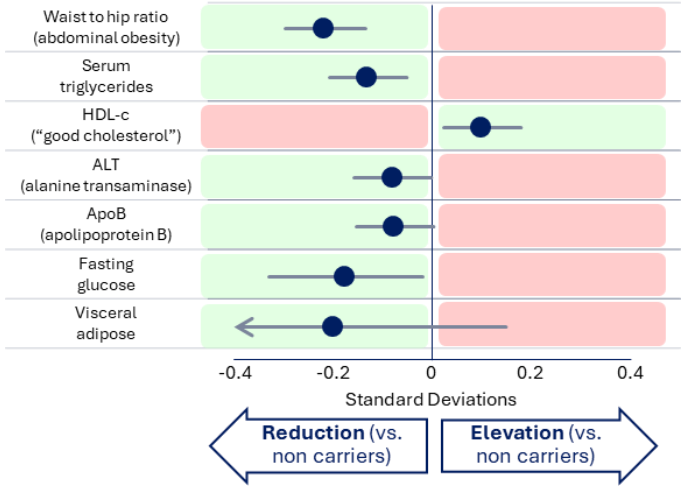
WVE-007 (INHBE program)
GalNAc-siRNA silencing

Obesity and other metabolic disorders

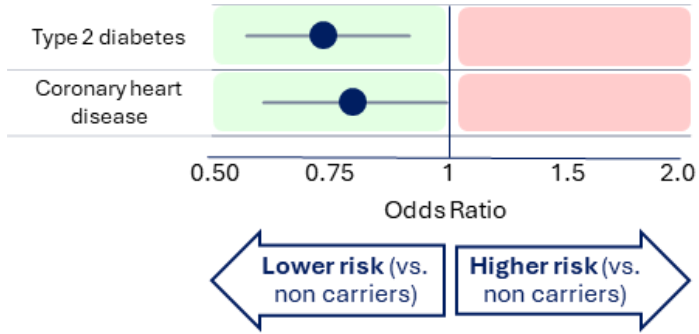
WAVE™ LIFE SCIENCES

Human genetic data demonstrate that INHBE heterozygous carriers have a healthy metabolic profile

Heterozygous INHBE LoF carriers have favorable traits: lower abdominal obesity, lower triglycerides, higher HDL-c



Heterozygous INHBE LoF carriers have lower risk of Type 2 diabetes and CHD



Silencing INHBE mRNA by ≥50% is expected to recapitulate the healthy metabolic profile of heterozygous INHBE loss of function (LoF) carriers

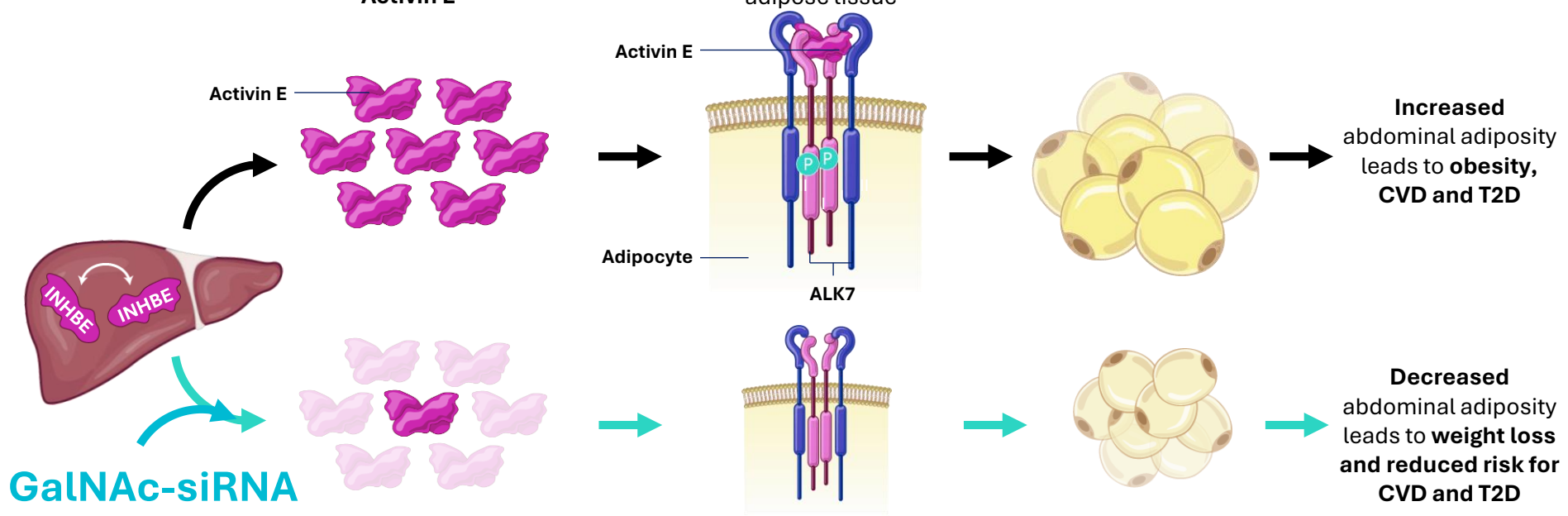
Akbari et al. Nat Commun. 2022 Aug 23;13(1):4844; Deaton et al. Nat Commun. 2022 Jul 27

INHBE GalNAc-RNA expected to address health issues associated with pathogenesis of obesity, associated metabolic disease

Release of **dimerized** INHBE subunits creates hepatokine **Activin E**

Binds to and **activates** **ACVR1C** (ALK7) receptor in adipose tissue

Block adipose lipolysis



Increased abdominal adiposity leads to **obesity, CVD and T2D**

GalNAc-siRNA

Reduced release of hepatokine **Activin E**

Diminished activation of **ACVR1C** (ALK7) receptor in adipose tissue

Increased adipose lipolysis and shrink adipocytes

Decreased abdominal adiposity leads to **weight loss and reduced risk for CVD and T2D**

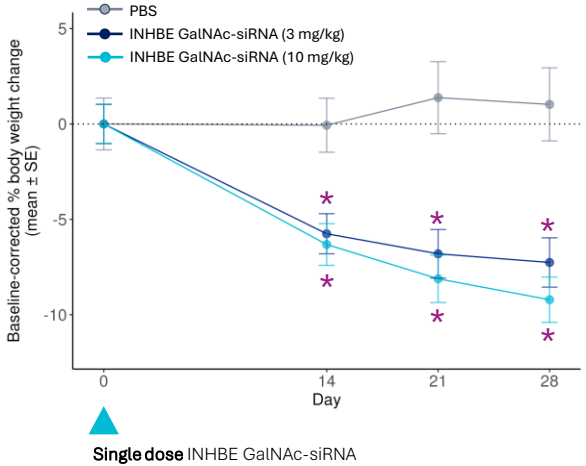
1. Cell Reports (2018) 25, 1193–1203; 2. Biochemical Journal (2024) 481 547–564; 3. PNAS 2023 Vol. 120 No. 32 e2309967120; 4. Nat Commun 2022. https://doi.org/10.1038/s41467-022-32398-7; 5. Nat Commun 2022. https://doi.org/10.1038/s41467-022-31757-8

Single doses of INHBE GalNAc-siRNA result in dose-dependent weight loss and reduction of visceral fat, without affecting muscle mass

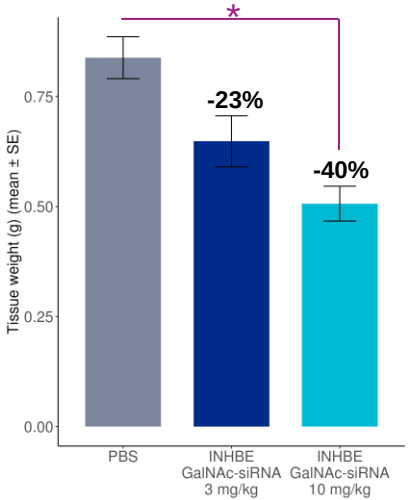
✓ Reduction in body weight

✓ Reduction in visceral fat

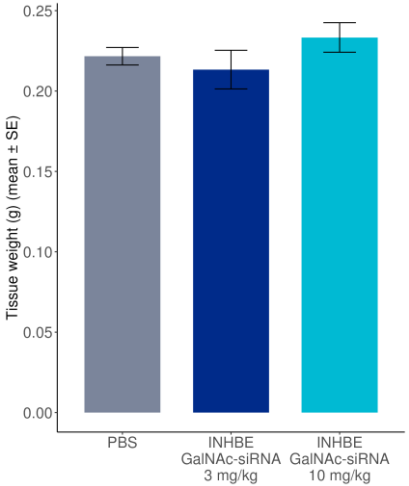
✓ No muscle loss



Epididymal fat weight (Day 28)



Quadricep weight (Day 28)

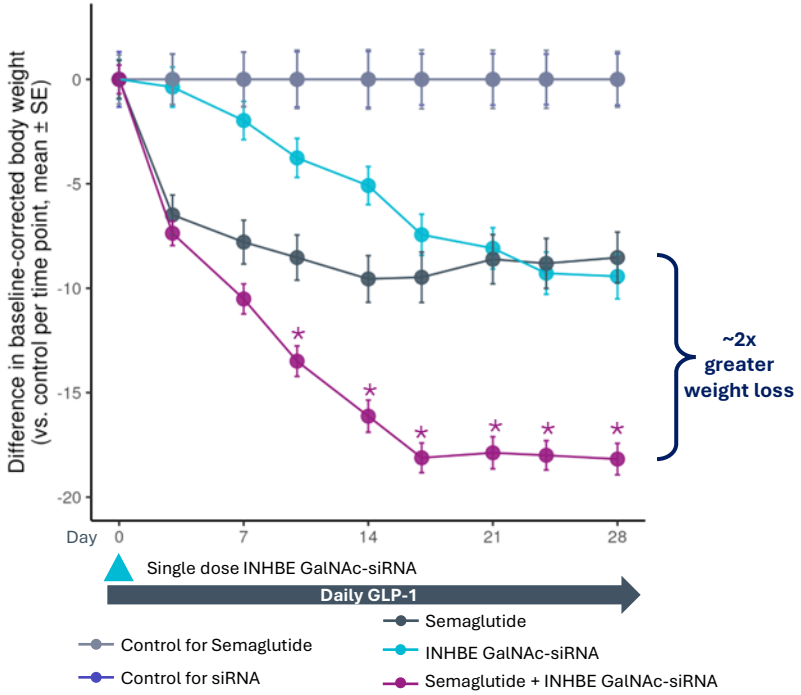


INHBE GalNAc-siRNA has potential as monotherapy weight loss therapeutic

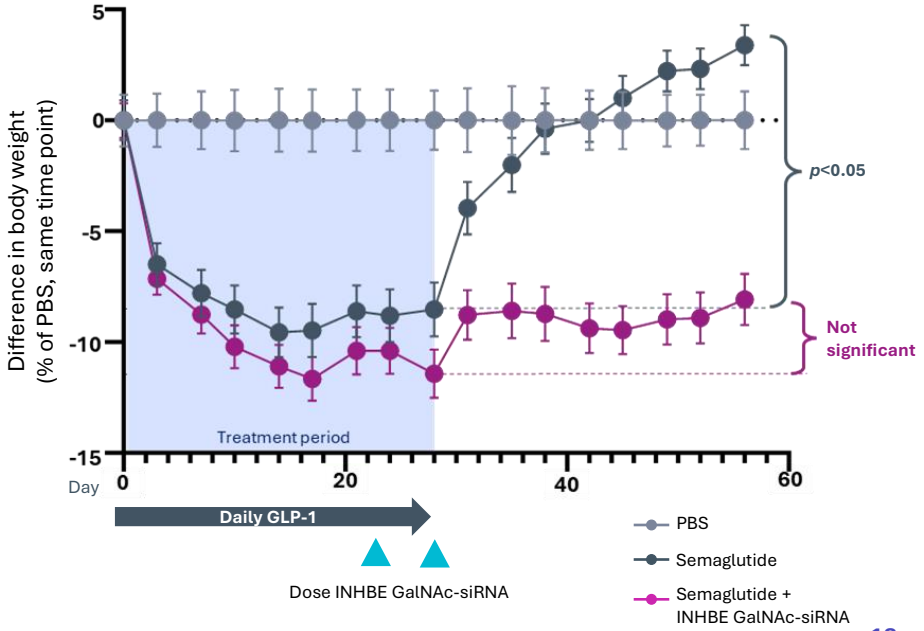
Stats: (left, middle, right) Linear Mixed Effects ANOVA with post hoc comparisons of marginal treatment effects vs. PBS per timepoint (left) or per tissue (middle, right) * $p < 0.05$

INHBE GalNAc-siRNA can be used synergistically with GLP-1s or to prevent weight regain after the cessation of treatment with GLP-1s

~2x greater overall weight loss when added to GLP-1s



Prevents weight regain after the cessation of GLP-1s



WAVE™ LIFE SCIENCES

Left: 10nmol/kg in mouse is equivalent to therapeutic dose of GLP-1s in human. Stats: Linear Mixed Effects ANOVA with post hoc comparisons of marginal treatment effects of Semaglutide vs. Semaglutide + INHBE GalNAc-siRNA per time point * $p < 0.05$; Right Stats: Linear Mixed Effects ANOVA with post hoc comparison of Day 28 vs. Day 56 marginal effects per treatment * $p < 0.05$

Preclinical data support best-in-class profile and potential to use WVE-007 across multiple treatment settings with 1-2x a year dosing

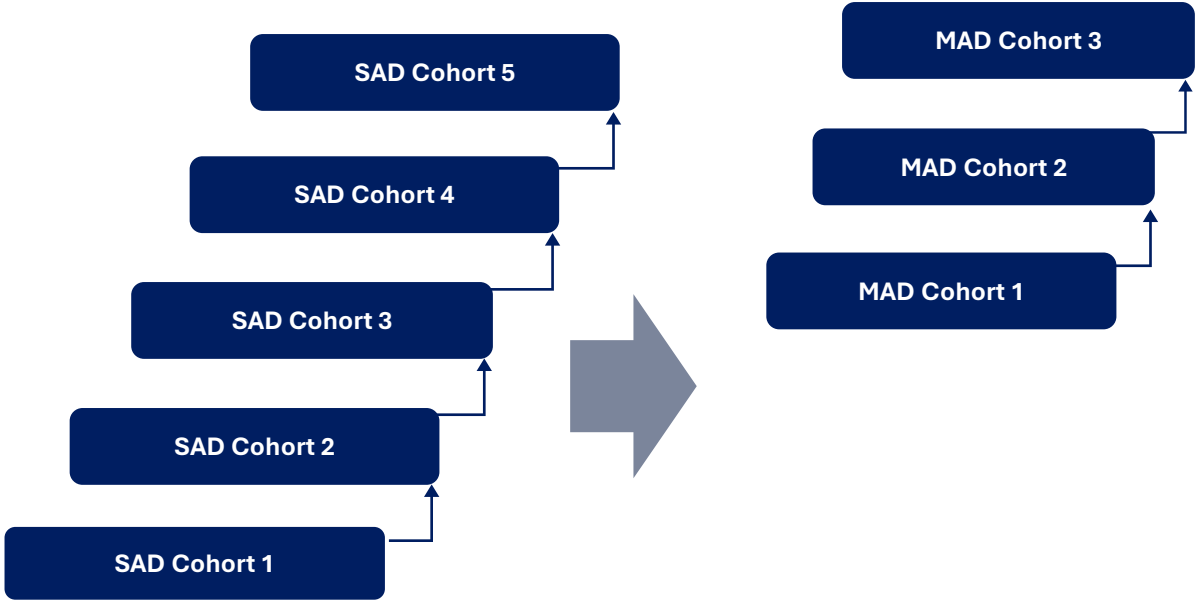
- ✓ **Monotherapy: as a single agent.** Weight loss similar to semaglutide with no loss of muscle mass and a reduction in fat mass with preferential effect to the visceral fat, and without suppressing food intake
- ✓ **Add-on to GLP-1s: WVE-007 in addition to GLP-1 therapy.** When administered as an add-on with semaglutide, a single dose of Wave's INHBE GalNAc-siRNA doubled the weight loss observed with semaglutide alone
- ✓ **Maintenance: for patients who stop treatment with GLP-1 therapy.** Curtailed rebound weight gain upon cessation of semaglutide and prevention of weight cycling, which worsens the outcomes of various metabolic diseases

CTA expected before year-end for Phase 1 trial of WVE-007 in adults living with overweight or obesity, otherwise healthy

Randomized, double-blind, placebo-controlled study of ascending doses of WVE-007

Trial Design

- **Objective**: Assess dose safety, tolerability, PK and PD
- **Key measurements**
  - **Primary:** Safety and Tolerability
  - **Secondary:** PK, Activin E
  - **Exploratory PD:**
    - Body Weight
    - Body compositions
    - Metabolic health
    - Biochemical markers



Expect to initiate clinical trial for WVE-007 in 1Q 2025

WVE-N531
Splicing

Duchenne muscular dystrophy

WAVE™ LIFE SCIENCES

Urgent need for improved therapeutic options for the treatment of DMD

Duchenne is a devastating and fatal disease

- Genetic mutation in dystrophin gene prevents the production of dystrophin protein, a critical component of healthy muscle function
- Impacts ~1 / 5,000 newborn boys annually; ~20,000 new cases annually worldwide
  - ~8–10% are amenable to exon 53 skipping
  - Potential for Wave to address up to 40% of DMD with additional exon skipping therapeutics

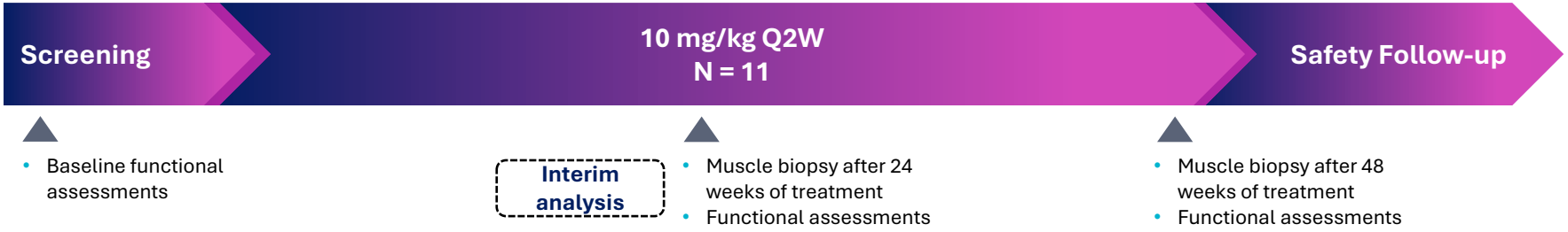
Multiple urgent unmet needs

- Need for therapies delivering **more consistent dystrophin expression,** as few patients today achieve dystrophin >5% of normal
- **Opportunity to extend dosing intervals** beyond weekly standard of care to alleviate burden for patients and caregivers
- **Need to reach stem cells and distribute broadly to muscle tissues** to potentially enable muscle regeneration and impact respiratory and cardiac function

Boy living with DMD

Duan, D. et al. 2021 Nat Rev Dis Primers 7, 13; Muscular Dystrophy Association; Aartsma-Rus, et al. 2009 Hum Mutat 30, 293.

FORWARD-53: An ongoing potentially registrational open-label Phase 2 clinical trial of WVE-N531 in boys with DMD amenable to exon 53 skipping



Key Assessments:

- Safety and tolerability
- Muscle biopsies after 24 and 48 weeks of treatment
  - PK: Drug tissue concentrations
  - PD: Exon-skipping, Dystrophin level (% of normal) as assessed by Western Blot
- Functional outcome measures
- 11 participants enrolled, including two from prior Part A clinical trial
  - Pre-specified analyses in ambulatory patients

IV: Intravenous; Q2W: Every 2 Weeks; PK: Pharmacokinetics; PD: Pharmacodynamics

Results of interim analysis: WVE-N531 has potential to be the best-in-class therapeutic for exon 53 DMD

Highly consistent dystrophin expression across patients

- 9.0% muscle-content adjusted dystrophin (5.5% unadjusted), quantified from two isoforms that are consistent with Becker patients who display milder disease
- 89% of patients over 5% of normal (muscle-content adjusted)

Muscle delivery and extended dosing intervals

- Skeletal muscle tissue concentrations of WVE-N531: ~41,000 ng/g
- WVE-N531 tissue half-life of 61 days supports monthly dosing
- Preclinical data suggests WVE-N531 is translating in heart and diaphragm

Evidence supporting improved muscle health

- Improvement in serum biomarkers for muscle health
- Localization of WVE-N531 in myogenic stem cells
- Improvement in myofiber regeneration

Safe and well tolerated

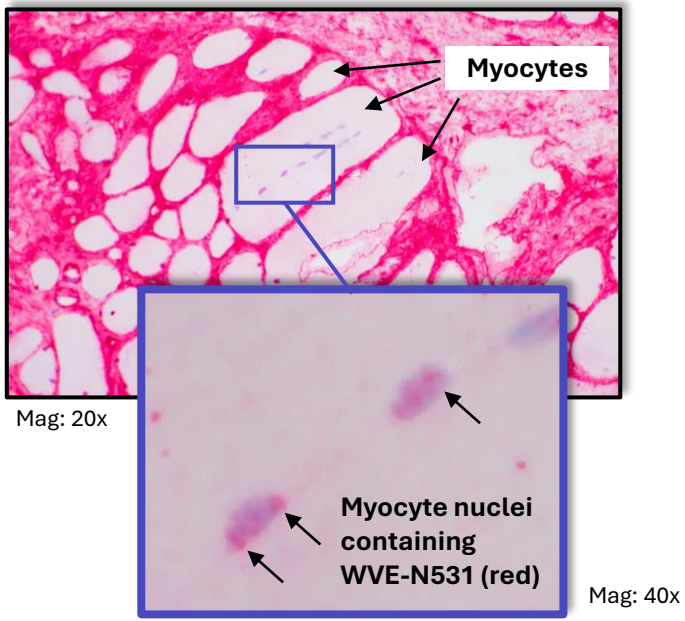
- No SAEs
- No discontinuations
- No oligonucleotide class effects

Expect to receive feedback from regulators on pathway to accelerated approval and deliver 48-week FORWARD-53 data in 1Q 2025

Dystrophin data from prespecified analysis of ambulatory boys; Muscle content adjustment was done using the formula: MHC-normalized dystrophin/(total myofiber area/total area of biopsy section). Interim analysis results announced September 24, 2024.

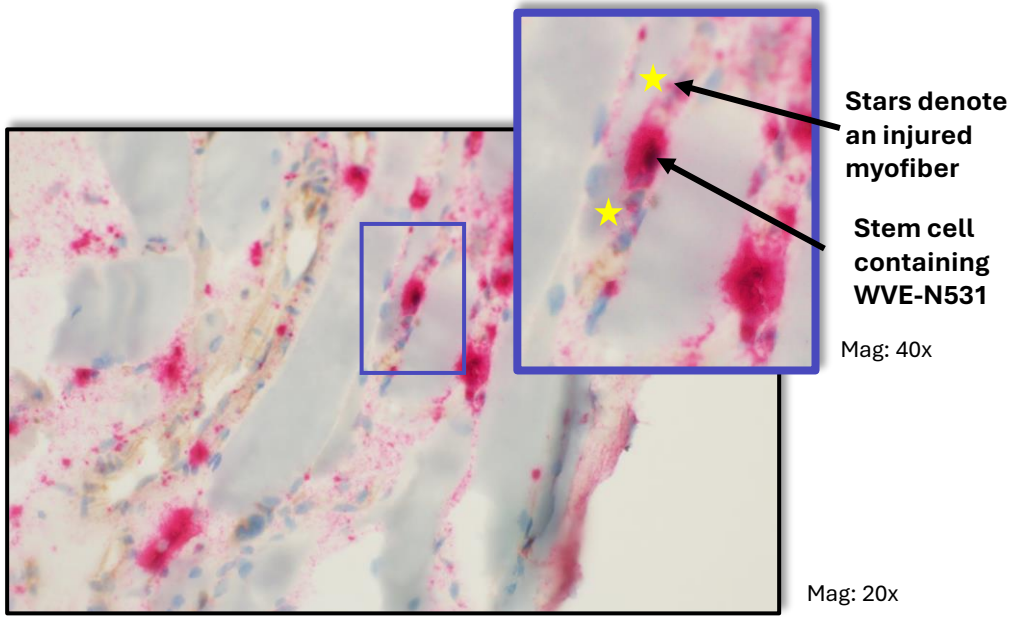
WVE-N531 was localized in myofiber nuclei and myogenic stem cells

WVE-N531 uptake in myofiber nuclei



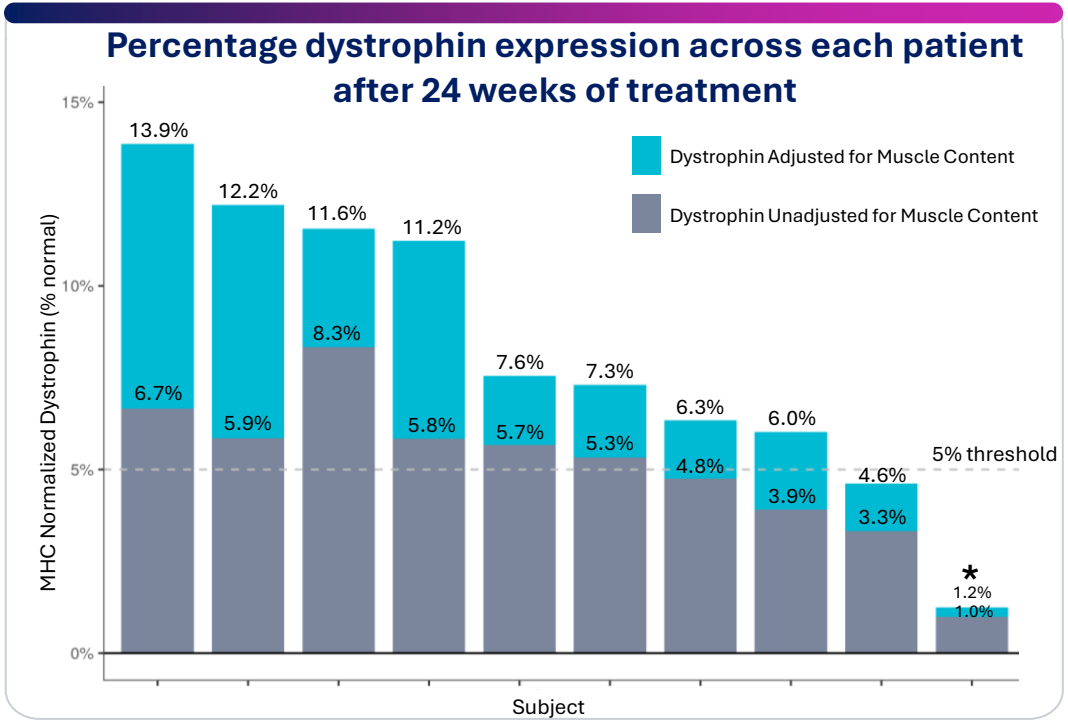
In-situ hybridization for WVE-N531

WVE-N531 uptake in myogenic stem cells



Dual staining utilizing in-situ hybridization for WVE-N531 and PAX7 immunohistochemistry for stem cells

Dystrophin expression of up to ~14% with high consistency across participants



- Mean 9.0% absolute muscle content adjusted dystrophin
- Mean 5.5% absolute unadjusted dystrophin
- Dystrophin expression was quantified from two isoforms consistent with those observed in Becker patients who display milder disease

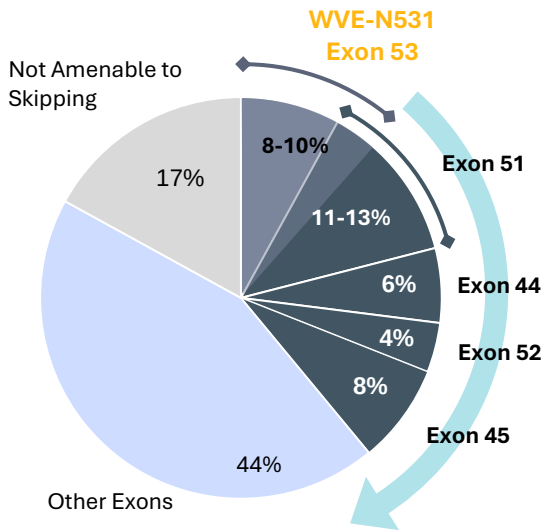
89% of ambulatory participants achieve muscle content-adjusted dystrophin levels of at least 5%

WAVE LIFE SCIENCES

*Excluded from prespecified mean analysis of ambulatory patients; Muscle content adjustment was done using the formula: MHC-normalized dystrophin/(total myofiber area/total area of biopsy section); Graph shows all patients (including non-ambulatory) with appropriate biopsy sample; dystrophin measured by Western Blot (AB15277); Data as of August 19, 2024

Unlocking Wave's best-in-class exon skipping portfolio

DMD Population



- Data for exons 51, 44, 52, 45 demonstrate potential for even greater dystrophin expression
- Opportunity to address up to 40% of population
- Expect to engage regulators on a platform trial design that incorporates multiple exons

Aartsma-Rus, et al. 2009 Hum Mut 30, 293

WVE-003
Allele-selective silencing

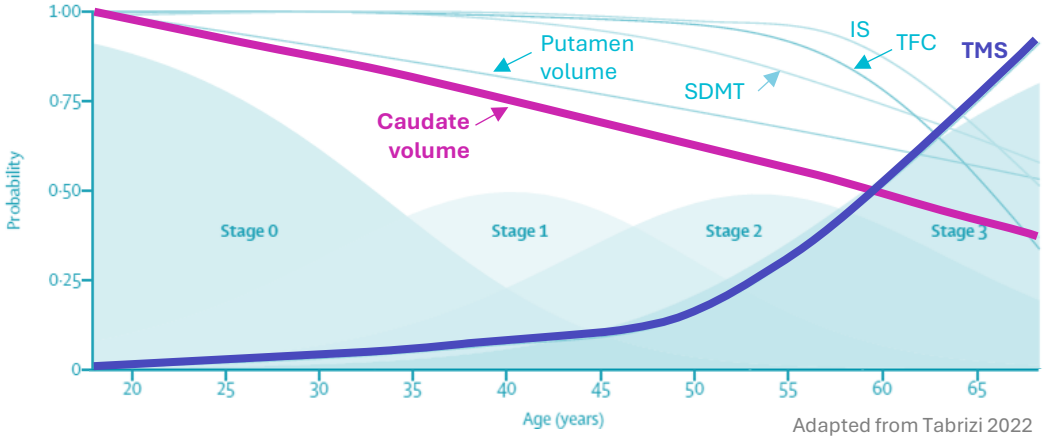
Huntington's Disease

WAVE™ LIFE SCIENCES

Huntington's disease is a devastating neurological disorder caused by a toxic gain of function and concurrent loss of function

- HD is a monogenic autosomal dominant genetic disease; fully penetrant and affects entire brain
- No current disease modifying therapies for HD
- Characterized by cognitive decline, psychiatric illness, and chorea; ultimately fatal
- Expanded CAG triplet repeat in *HTT* gene results in production of mutant huntingtin protein (mHTT) and loss of function in wild-type huntingtin protein (wtHTT)

HD-ISS stage probability and predicted clinical landmark changes as a function of age



Adapted from Tabrizi 2022

>200,000 patients with HD across all disease states

Pre-Symptomatic HD	Symptomatic HD
(~160K in US and Europe)	(~65K in US and Europe)

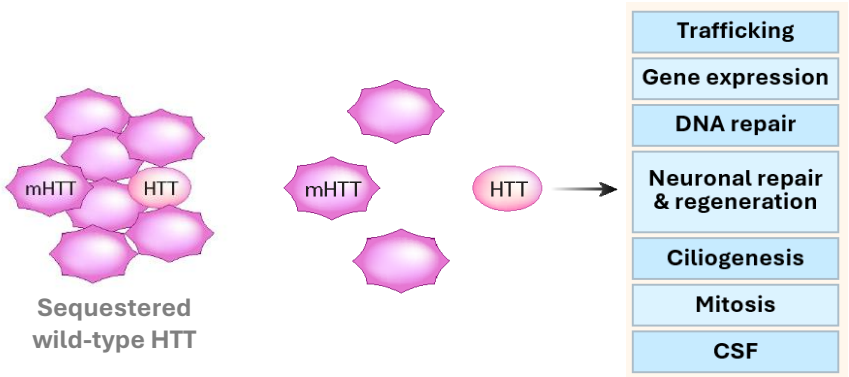
An allele-selective, wtHTT-sparing approach is uniquely suited to address HD across all stages of disease

Sources on wtHTT: 1. Leavitt 2006 2. Cattaneo 2005 3. Kumar 2016 4. Franco-Iborra 2020 5. Hamilton 2015 6. Ochaba 2014 7. Wong 2014 8. Rui 2015 9. Caviston 2007 10. Twelvetrees 2010 11. Strehlow 2007 12. Milnerwood 2010 13. Smith-Dijak 2019 14. Tousley 2019 15. Zhang 2018 16. McAdam 2020 17. Altar 1997 18. Zuccato 2001 19. Gauthier 2004 20. Ferrer 2000 21. Baquet 2004 22. Liu 2011 23. Karam 2015; IS, Independence Scale; SDMT, Symbol Digit Modalities Test; TFC, Total Functional Capacity; TMS, Total Motor Score

Wild-type HTT (wtHTT) is critical for normal neuronal function and loss of wtHTT contributes to cellular dysfunction

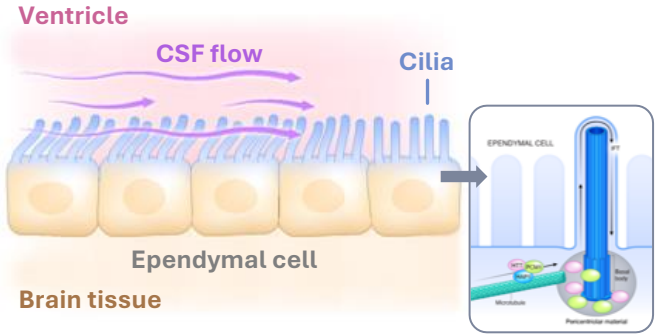
Mutant HTT has a detrimental effect on wild-type HTT function

- Lowering mHTT is expected to restore physiological control over HTT gene expression and relieve its detrimental effect on wtHTT function



Wild-type HTT is crucial for cilia health

- In the absence of wtHTT, ciliogenesis fails, disrupting CSF flow, causing hydrocephalus



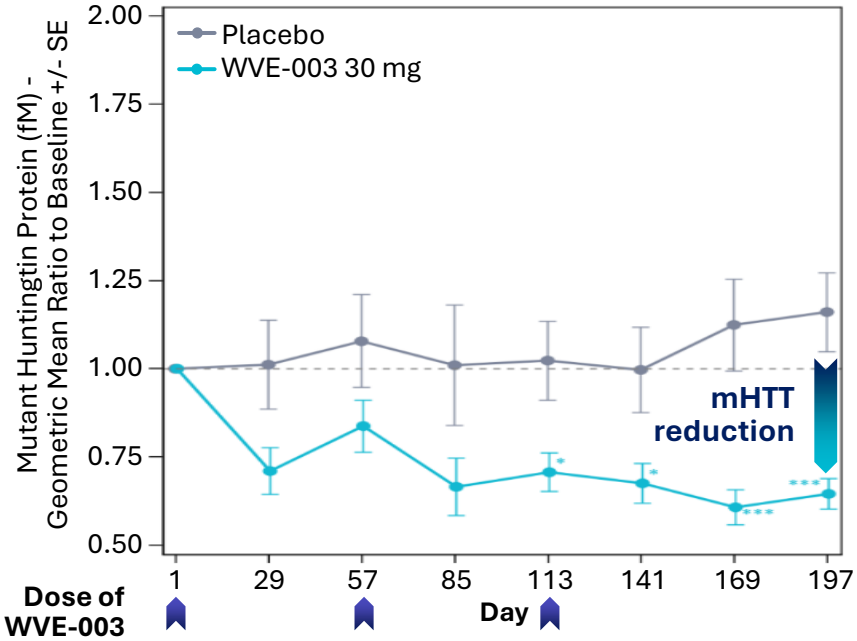
Only an allele-selective approach can ameliorate both loss-of-function and gain-of-function disruptions driven by mHTT

Saudou & Humbert 2016 Neuron; Cason et al., 2022 Nat Rev Cell Biol; Laundos et al., 2023 Front Cell Dev Biol; Kaliszewski et al., 2015 Cell Death Diff; Keryer et al., 2011 J Clin Invest
Khoshnan & Patterson, 2011. Neurobiol Dis; Pogoda et al., 2021 Curr Med Chem; Hsiao et al., 2013 Hum Mol Genet

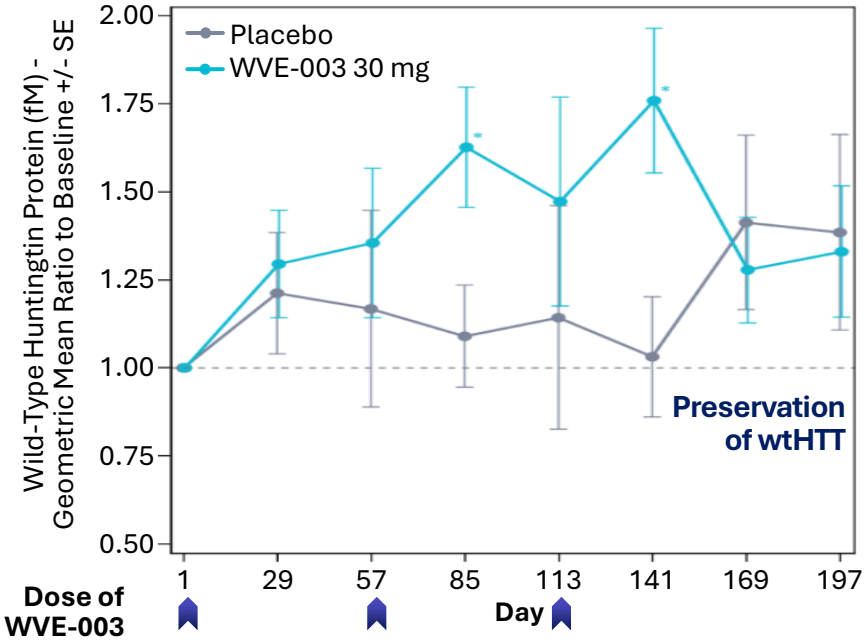
Allele-selective lowering of mutant HTT protein of up to 46% with three doses of WVE-003 and preservation of wild-type HTT

Durability of mHTT reductions supports potential for quarterly dosing intervals

Mutant HTT protein levels



Wild-type HTT protein levels



WAVE LIFE SCIENCES

* p<0.05, **p<0.01, ***p<0.001, ****p<0.0001
mHTT: mutant huntingtin protein; wtHTT: wild-type huntingtin protein
From June 25, 2024 SELECT-HD disclosure

WVE-003 leads to allele-selective mHTT reduction, correlating with slowing of caudate atrophy

Allele-selective mHTT silencing with wtHTT Preservation

- mHTT reduction of up to 46% vs. placebo
- wtHTT preserved/increased throughout study

Slowing of Caudate Atrophy

- WVE-003 trended towards less caudate atrophy vs. placebo (4.68% vs. 5.10%, not significant)

Functional Benefit

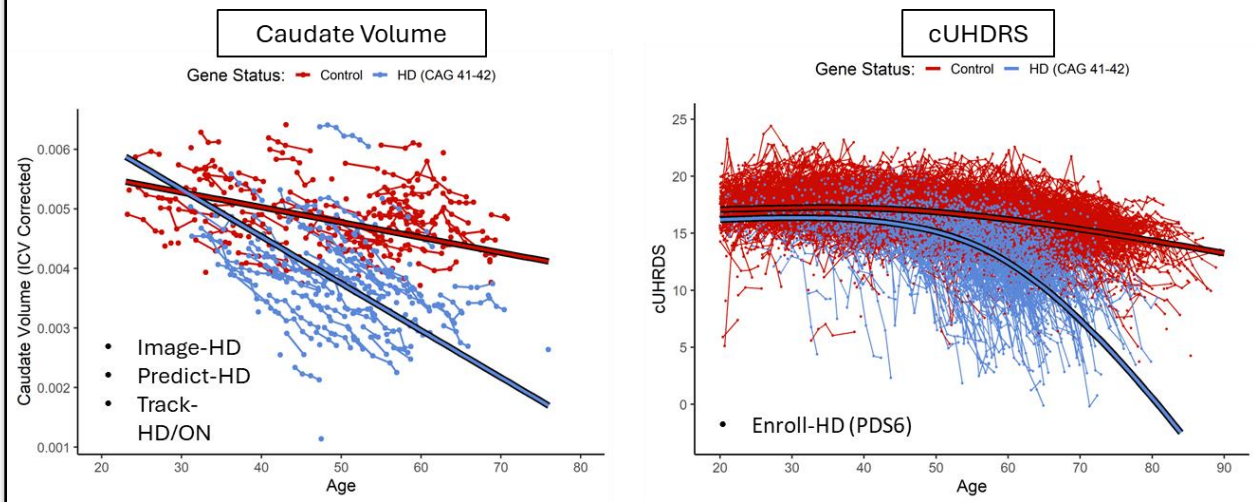
- Caudate atrophy is an imaging biomarker expected to predict clinical outcomes, including clinically meaningful worsening of Total Motor Score (TMS)

Greater allele-selective mHTT reduction correlated with the slowing of caudate atrophy at 24 weeks (R = -0.50, p=0.047)

Liu et al., 2023 Brain Comm

Regularity of caudate change makes it an ideal biomarker for more efficient clinical development in HD

- HD-ISS Stage 2



- Caudate volume has a more regular, linear change at individual level versus variability in individual trajectories with cUHDRS
- Wave analysis[1] of PREDICT and TRACK-HD reinforces the relationship between caudate volume loss and clinical outcomes
  - Difference of 1% reduction in rate of caudate atrophy results in ≥6-year delay in loss of function (i.e. reduction from TFC 13)
- Using caudate volume as a primary endpoint will enable smaller, faster and more efficient clinical trials

"Using the HD-ISS for Planning Clinical Trials", Long, J; presented at EHDN & Enroll-HD Conference, Strasbourg, France, Sept 2024
1 – internal analysis conducted by Wave

Preservation of caudate volume offers an efficient pathway for potential accelerated approval for HD

- Received supportive initial feedback from FDA:
  - Recognize the severity of HD
  - Receptive to and engaged with Wave regarding a potential pathway to accelerated approval
  - Open to Wave's plan to evaluate biomarkers, including vMRI for caudate atrophy, as an endpoint to assess HD progression with the potential to predict clinical outcome
- Planning is underway for a global, potentially registrational Phase 2/3 study in adults with SNP3 and HD, including finalization of key aspects of design

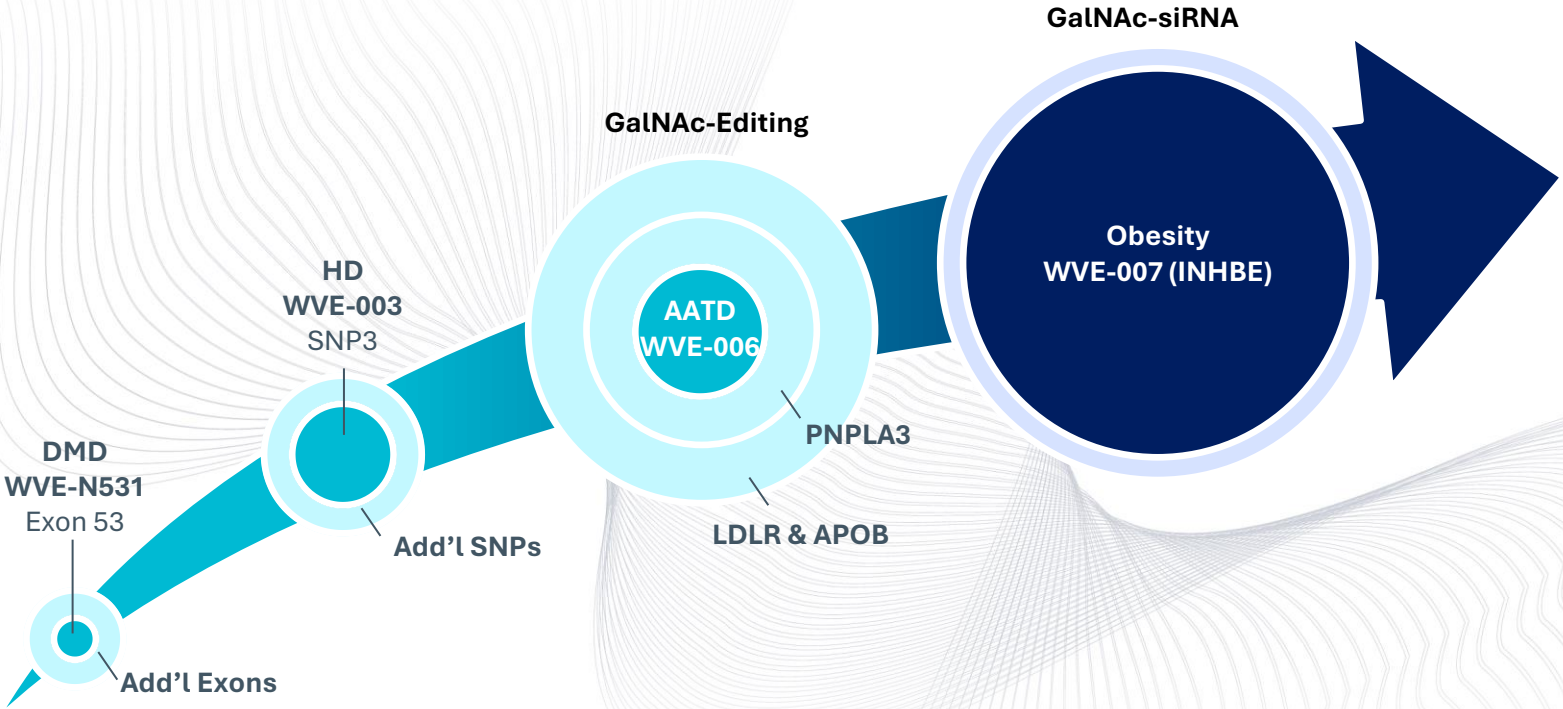
Wave expects to submit an IND for WVE-003 in 2H 2025

vMRI: volumetric MRI

Reimagining RNA medicines

WAVE™ LIFE SCIENCES

Poised for significant and sustained growth driven by editing and siRNA



Wave's platform is translating in the clinic and has potential to treat >90M patients in the US and Europe

Note: Bubble size illustrative of size of total addressable US market (assuming 100% share of addressable patients)

Anticipated upcoming milestones

GalNAc-RNA editing		*GalNAc-siRNA*	*Splicing*	*Allele-selective silencing*
WVE-006 **AATD**	**PNPLA3 - Liver disease** **LDLR, APOB - HeFH**	**WVE-007 (INHBE)** **Obesity**	**WVE-N531 (Exon 53)** **DMD**	**WVE-003 (SNP3)** **HD**
2025: Deliver multidose data from RestorAATion-2	**2025:** Select clinical candidates	**Year-end 2024:** Submit CTA **1Q 2025:** Initiate clinical trial	**1Q 2025:** Deliver 48-week FORWARD-53 data & receive feedback from regulators on pathway to accelerated approval	**2H 2025:** Submit an Investigational New Drug (“IND”) application

Well-capitalized with cash runway into 2027

AATD: Alpha-1 antitrypsin deficiency; HeFH: Heterozygous familial hypercholesterolemia; DMD: Duchenne muscular dystrophy; HD: Huntington’s disease; mHTT: Mutant huntingtin; wtHTT: Wild-type huntingtin; CTA: clinical trial application

WAVE™
LIFE SCIENCES

Reimagine possible.

For questions contact:
investorrelations@wavelifesci.com