UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of report (Date of earliest event reported): September 11, 2018

WAVE LIFE SCIENCES LTD.
(Exact name of registrant as specified in its charter)

Singapore	**001-37627**	**Not Applicable**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

7 Straits View #12-00 Marina One East Tower Singapore 018936	**018936**
(Address of principal executive offices)	**(Zip Code)**

Registrant's telephone number, including area code: +65 6236 3388

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

- ☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
- ☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
- ☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
- ☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☒

Item 7.01 Regulation FD Disclosure.

From time to time, Wave Life Sciences Ltd. (the "Company") presents and/or distributes slides and presentations to the investment community to provide updates and summaries of its business. On September 11, 2018, the Company updated its corporate presentation, which is available on the "For Investors & Media" section of the Company's website at http://ir.wavelifesciences.com/. This presentation is also furnished as Exhibit 99.1 to this Current Report on Form 8-K.

The information in this report furnished pursuant to Item 7.01 shall not be deemed "filed" for the purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section. It may only be incorporated by reference in another filing under the Exchange Act or the Securities Act of 1933, as amended, if such subsequent filing specifically references the information furnished pursuant to Item 7.01 of this report.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

The following exhibit relating to Item 7.01 shall be deemed to be furnished, and not filed:

Exhibit No.	Document
99.1	Corporate Presentation of Wave Life Sciences Ltd.

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

WAVE LIFE SCIENCES LTD.

Date: September 11, 2018

/s/ Keith C. Regnante
Keith C. Regnante
Chief Financial Officer

Exhibit 99.1

Wave Life Sciences

Corporate Presentation

September 10, 2018

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.

WAVE™
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We are leading a new era of precision medicine in which rationally designed nucleic acid therapies are the key to delivering safer, more effective treatments for serious, genetically defined diseases

Architects of transformation

Wave Life Sciences is a clinical-stage, genetic medicines company unlocking the potential of a proprietary chemistry platform that enables the precise design, optimization and production of stereopure nucleic acid therapies.

Wave's chemistry platform is built on a foundation of two core capabilities:

PRECISION

Ability to design nucleic acid compounds that have **one defined and consistent profile**

SCALE

Platform potential across **multiple modalities and tissues**

Internal expertise and capacity for **large-scale GMP manufacturing**

Wave has reinvented the design, synthesis and manufacture of nucleic acid therapies to potentially optimize potency, durability and safety

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Building the optimal, stereopure medicine

STANDARD OLIGONUCLEOTIDE APPROACHES

Pharmacologic properties include >500,000 permutations in every dose

↓

Impact:
Unreliable therapeutic effects
Unintended off-target effects

WAVE RATIONAL DESIGN

Stereochemistry enables precise control, ability to optimize critical constructs into one defined and consistent profile

↓

Impact:
Potential for safer, more effective, targeted medicines that can address difficult-to-treat diseases

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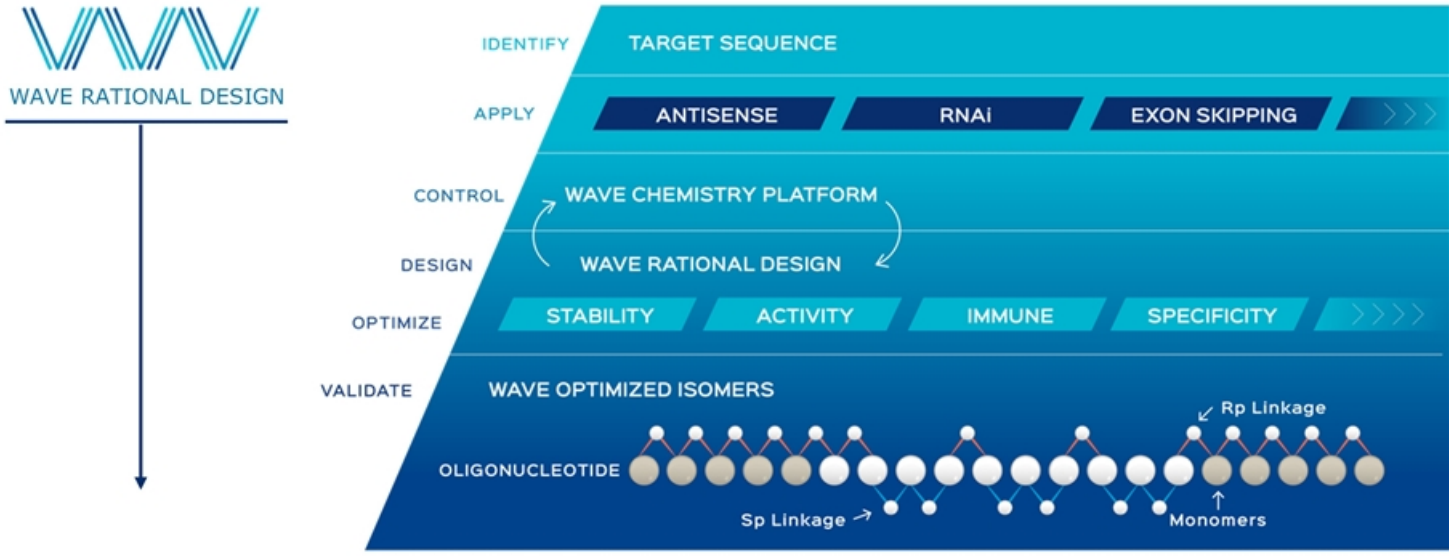
Creating a new class of oligonucleotides



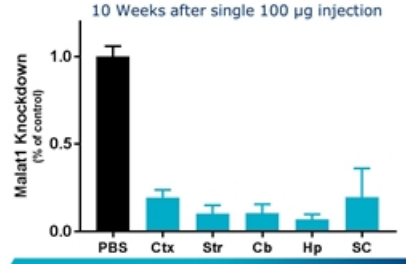
WAVE LIFE SCIENCES

Source: Iwamoto N, et al. Control of phosphorothioate stereochemistry substantially increases the efficacy of antisense oligonucleotides. Nature Biotechnology. 2017.

Optimizing potency and durability across multiple tissues

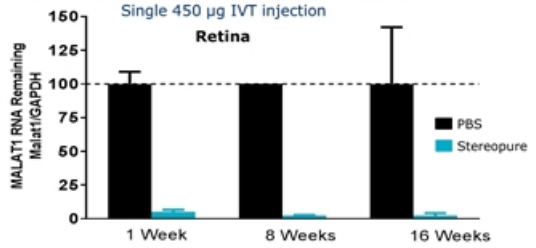
CNS

MALAT1 Knockdown in Mice
10 Weeks after single 100 µg injection



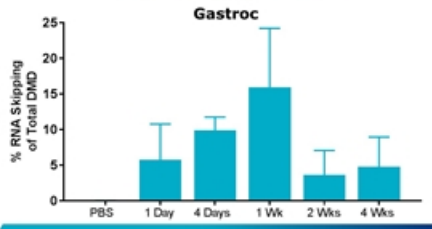
Eye

MALAT1 Knockdown in Non-Human Primates
Single 450 µg IVT injection



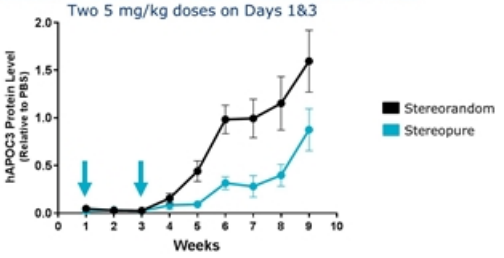
Muscle

DMD: Percent Skipped Transcript in *mdx*23 Mice
Single 150 mg/kg IV injection



Liver

Knockdown of Serum hAPOC3 Protein Levels in Mice
Two 5 mg/kg doses on Days 1&3

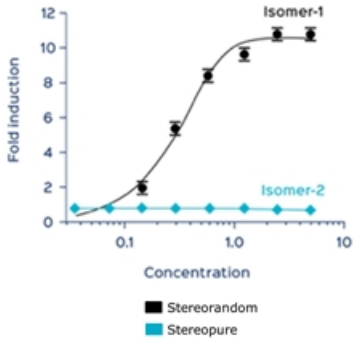


Data represented in this slide from *in vivo* studies. CNS: PBS = phosphate buffered saline; Ctx = cortex; Str = striatum; Cb = cerebellum; Hp = hippocampus; SC = spinal cord.

Chemistry affects immune activation

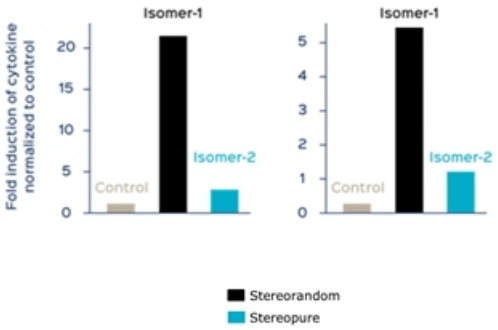
Human TLR9 Activation

Human TLR9 activation assay with 5mC modified CpG containing MOE gapmer



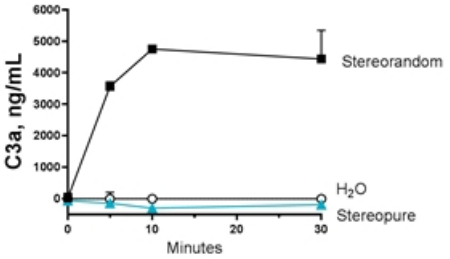
Cytokine Induction

Cytokine induction in human PBMC assay



Complement Activation

Complement activation in non-human primate serum assay



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Data represented in this slide from *in vitro* studies. MOE = 2′-O-methoxyethylribose; PBMC = peripheral blood mononuclear cell; TLR9 = toll-like receptor 9.

Pipeline spanning multiple modalities, novel targets

	TARGET	BIOMARKER	ESTIMATED U.S. PREVALENCE*	MECHANISM	DISCOVERY	CANDIDATE	CLINICAL	WAVE'S COMMERCIAL RIGHTS	PARTNER	NEXT ANTICIPATED MILESTONES
CNS										
Huntington's disease	mHTT SNP1	mHTT	~10k / ~35k	(A)	●	●	Phase 1b/2a	50% Global	Takeda	Top line data H1 2019
Huntington's disease	mHTT SNP2	mHTT	~10k / ~35k	(A)	●	●	Phase 1b/2a	50% Global	Takeda	Top line data H1 2019
Amyotrophic lateral sclerosis	C9orf72	Dipeptide	~1,800	(A)	●	●		50% Global	Takeda	Trial initiation Q4 2018
Frontotemporal dementia	C9orf72	Dipeptide	~7,000	(A)	●	●		50% Global	Takeda	Trial initiation Q4 2018
Spinocerebellar ataxia 3	ATXN3		~4,500	(S)	●	○		50% Global	Takeda	Candidate by YE 2018
CNS diseases	Multiple†			○	●	○		Milestones & Royalties	Takeda	
MUSCLE										
Duchenne muscular dystrophy	Exon 51	Dystrophin	~2,000	(E)	●	●	Phase 1	100% Global	—	Top line data Q4 2018
Duchenne muscular dystrophy	Exon 53	Dystrophin	~1,250	(E)	●	○		100% Global	—	
Neuromuscular diseases	Multiple			○	●	○		100% Global	—	
OPHTHALMOLOGY										
Retinal diseases	Multiple			○	●	○		100% Global	—	
HEPATIC										
Metabolic liver diseases	APOC3	Triglyceride		(S)	●	○		Milestones & Royalties	Pfizer	
Metabolic liver diseases	Multiple (4)‡			○	●	○		Milestones & Royalties	Pfizer	

(S) = silencing. (A) = allele-specific silencing. (E) = exon skipping.

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*Estimates of U.S. prevalence and addressable population by target based on publicly available data and are approximate; for Huntington's disease, numbers approximate manifest and pre-manifest populations, respectively.
† During a four-year term, Wave and Takeda may collaborate on up to six preclinical targets at any one time.
‡ Pfizer has nominated four undisclosed targets in addition to APOC3.

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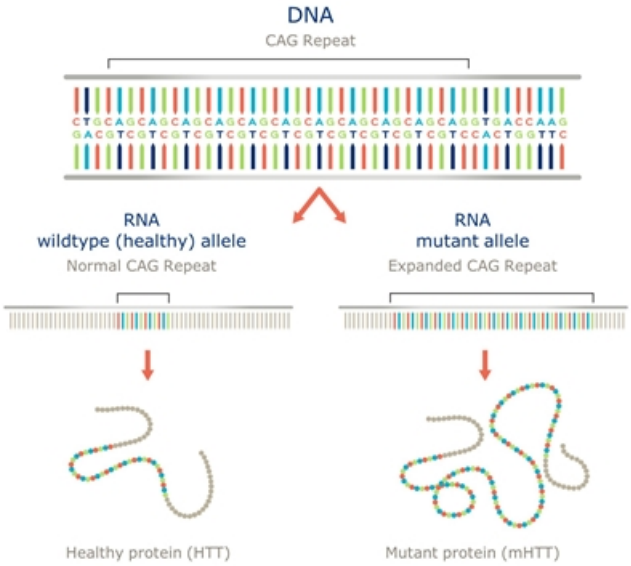
Huntington's Disease

Huntington's Disease: a hereditary, fatal disorder

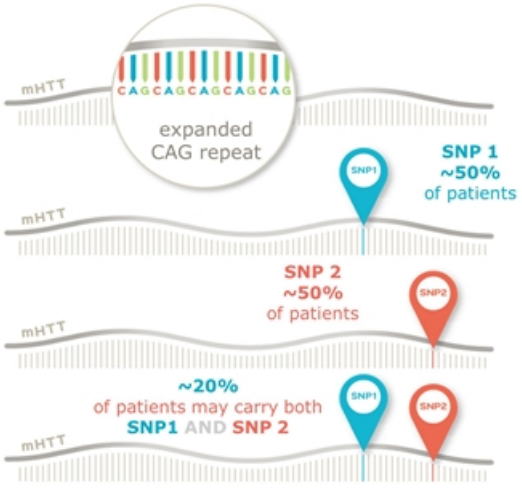
- Autosomal dominant disease, characterized by cognitive decline, psychiatric illness and chorea; fatal
- No approved disease-modifying therapies
- Expanded CAG triplet repeat in HTT gene results in production of mutant huntingtin protein (mHTT); accumulation of mHTT causes progressive loss of neurons in the brain
- Wildtype (healthy) HTT protein critical for neuronal function; suppression may have detrimental long-term consequences
- 30,000 people with Huntington's disease in the US; another 200,000 at risk of developing the condition



Sources: Auerbach W, et al. Hum Mol Genet. 2001;10:2515-2523. Dragatsis I, et al. Nat Genet. 2000;26:300-306. Leavitt BR, et al. J Neurochem. 2006;96:1121-1129. Nasir J, et al. Cell. 1995;81:811-823. Reiner A, et al. J Neurosci. 2001;21:7608-7619. White JK, et al. Nat Genet. 1997;17:404-410. Zeitlin S, et al. Nat Genet. 1995;11:155-163. Carroll JB, et al. Mol Ther. 2011;19:2178-2185.

Wave approach: novel, allele-specific silencing

- Utilize association between single nucleotide polymorphisms (SNPs) and genetic mutations to specifically target errors in genetic disorders, including HD.
- Allele-specificity possible by targeting SNPs associated with expanded long CAG repeat in mHTT gene
- Approach aims to lower mHTT transcript while leaving healthy HTT relatively intact
- Potential to provide treatment for up to 70% of HD population (either oligo alone could address approximately 50% of HD population)



Total: Due to overlap, an estimated **~70%** of the total HD patient population carry SNP 1 and/or SNP 2

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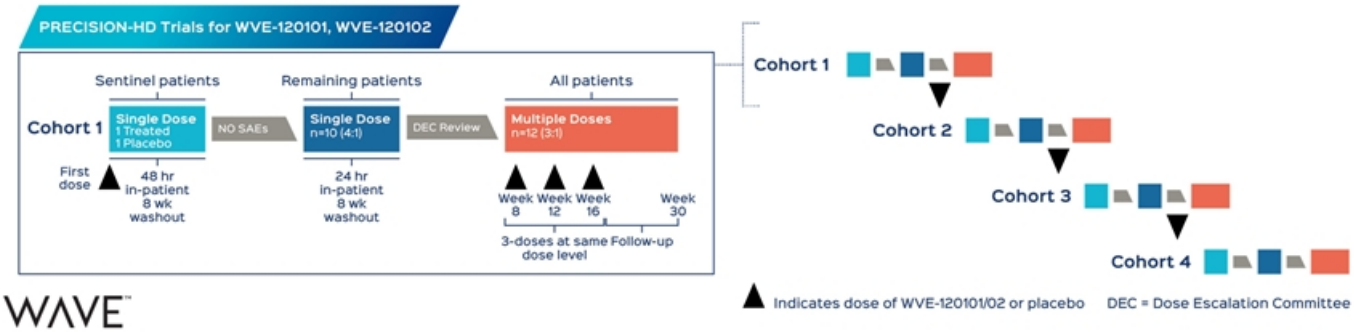
Source: Kay, et al. Personalized gene silencing therapeutics for Huntington disease. Clin Genet 2014: 86: 29–36

Two simultaneous Phase 1b/2a clinical trials

- Two parallel global placebo-controlled multi-ascending-dose trials for WVE-120101, WVE-120102
- Primary objective: assess safety and tolerability of intrathecal doses in early manifest HD patients
- Additional objectives: exploratory pharmacokinetic, pharmacodynamic, clinical and MRI endpoints
- Pre-screening blood test to determine presence of SNP 1 or SNP 2
- Approximately 50 patients per trial
- Key inclusion criteria:
  age ≥25 to ≤65, stage I or II HD
- Top line data anticipated H1 2019



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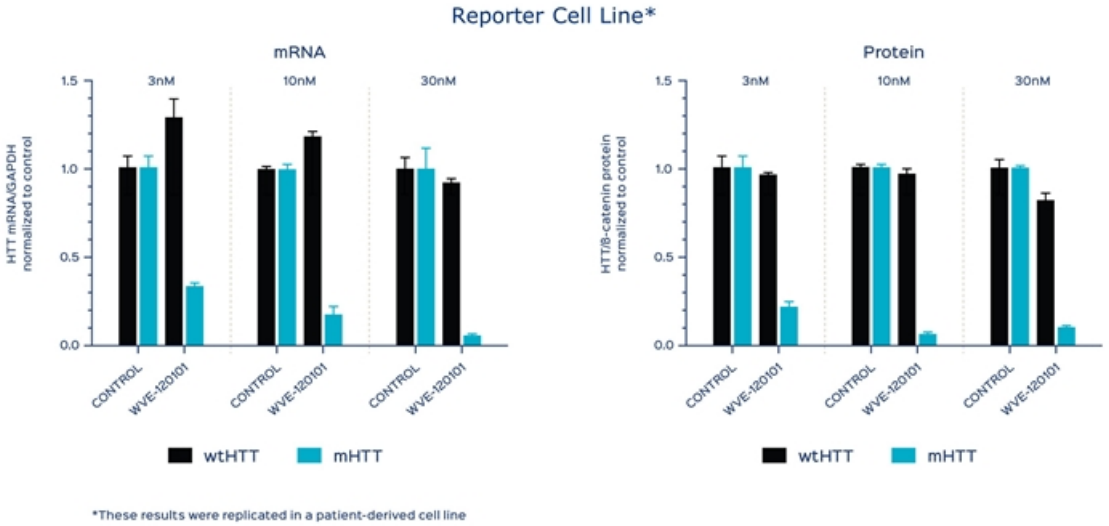
Selective reduction of mHTT mRNA & protein



*These results were replicated in a patient-derived cell line

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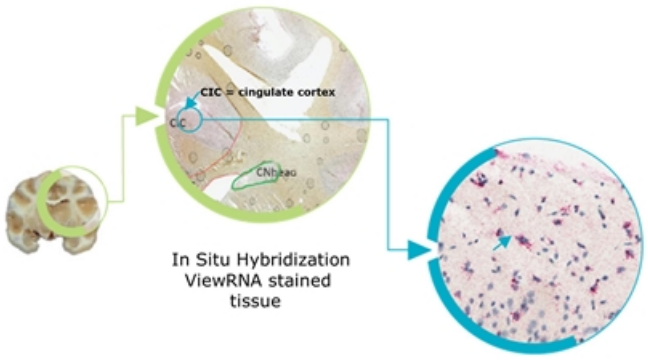
Demonstrated delivery to brain tissue

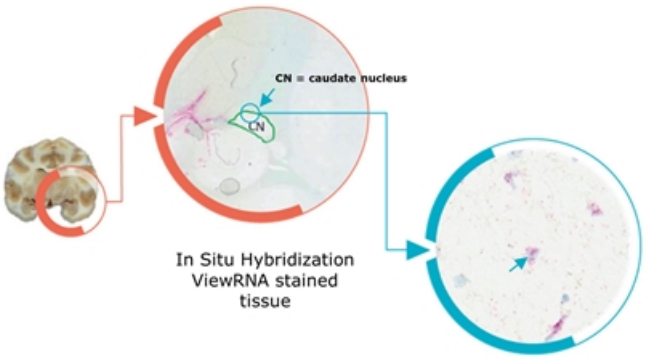
- WVE-120101 and WVE-120102 distribution in cynomolgus non-human primate brain following intrathecal bolus injection



In Situ Hybridization ViewRNA stained tissue

Red dots are WVE-120101 oligonucleotide.

Arrow points to nuclear and perinuclear distribution of WVE- 120101 in cingulate cortex



In Situ Hybridization ViewRNA stained tissue

Red dots are WVE-120102 oligonucleotide.

Arrow points to nuclear and perinuclear distribution of WVE-120102 in caudate nucleus

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CIC = cingulate cortex.
CN = caudate nucleus.

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Duchenne Muscular Dystrophy (DMD)

DMD: a progressive, fatal childhood disorder

- Fatal, X-linked genetic neuromuscular disorder characterized by progressive, irreversible loss of muscle function, including heart and lung
- Genetic mutation in dystrophin gene prevents the production of dystrophin protein, a critical component of healthy muscle function
- Symptom onset in early childhood; one of the most serious genetic diseases in children worldwide
- Current disease modifying treatments have demonstrated minimal dystrophin expression and clinical benefit has not been established
- Impacts 1 in every 5,000 newborn boys each year; 20,000 new cases annually worldwide

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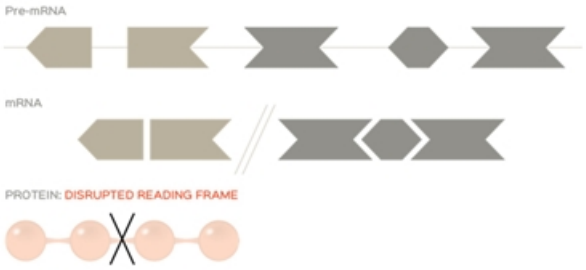
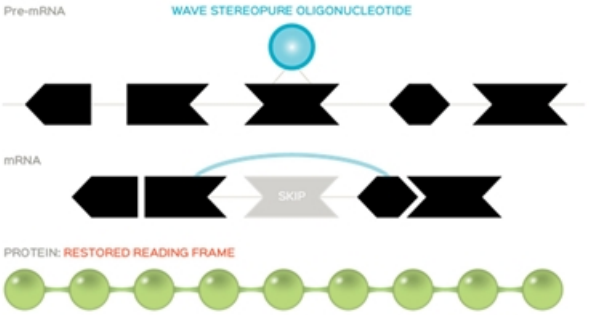
Wave approach:
meaningful restoration of dystrophin production through exon skipping

- Meaningful restoration of dystrophin production is expected to result in therapeutic benefit
- Exon-skipping antisense approaches may enable production of functional dystrophin protein
- Initial patient populations are those amenable to Exon 51 and Exon 53 skipping

Dysfunctional splicing (Disease)



Exon skipping (Potential Remedy)



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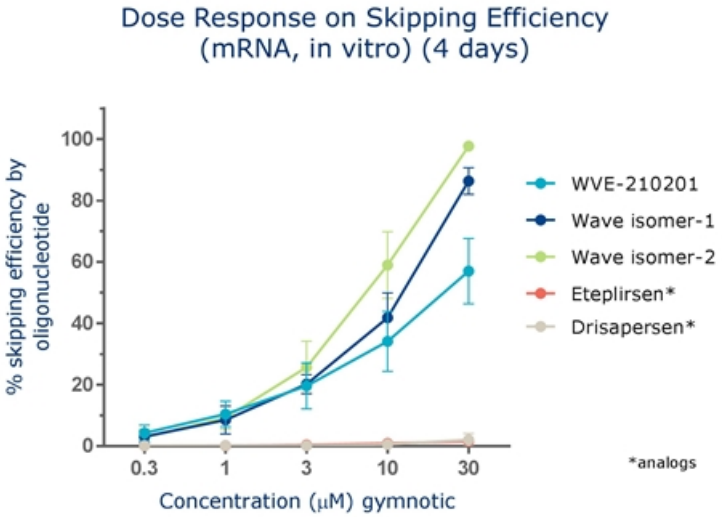
Exon 51: WVE-210201 clinical program

- WVE-210201 Phase 1 clinical trial initiated November 2017
  - Design: Multicenter, double-blind, placebo-controlled, single ascending dose study with I.V. administration
  - Primary endpoint: Safety and tolerability
  - Inclusion criteria: ages 5 to 18, amenable to exon 51 skipping
    - Ambulatory and non-ambulatory boys eligible, including those previously treated with eteplirsen (following appropriate washout period)
  - Readout expected Q4 2018
  - Open-label extension (OLE) with muscle biopsy and ≥2-years of follow-up
- WVE-210201 planned efficacy study
  - Efficacy readout anticipated H2 2019
  - Design: Double-blind, placebo-controlled, multi-dose study assessing dystrophin expression and clinical outcomes
  - Measurement of dystrophin via standardized Western Blot
  - Interim analysis of dystrophin expression in muscle biopsies
- Exploring intravenous and subcutaneous formulations for WVE-210201

Exon 51: improved skipping efficiency

- RNA skipping determined by quantitative RT-PCR
- Wave isomers demonstrated a dose-dependent increase in skipping efficiency
- Free uptake at 10uM concentration of each compound with no transfection agent
- Same foundational stereopure chemistry for Wave isomers; individually optimized to assess ideal profile



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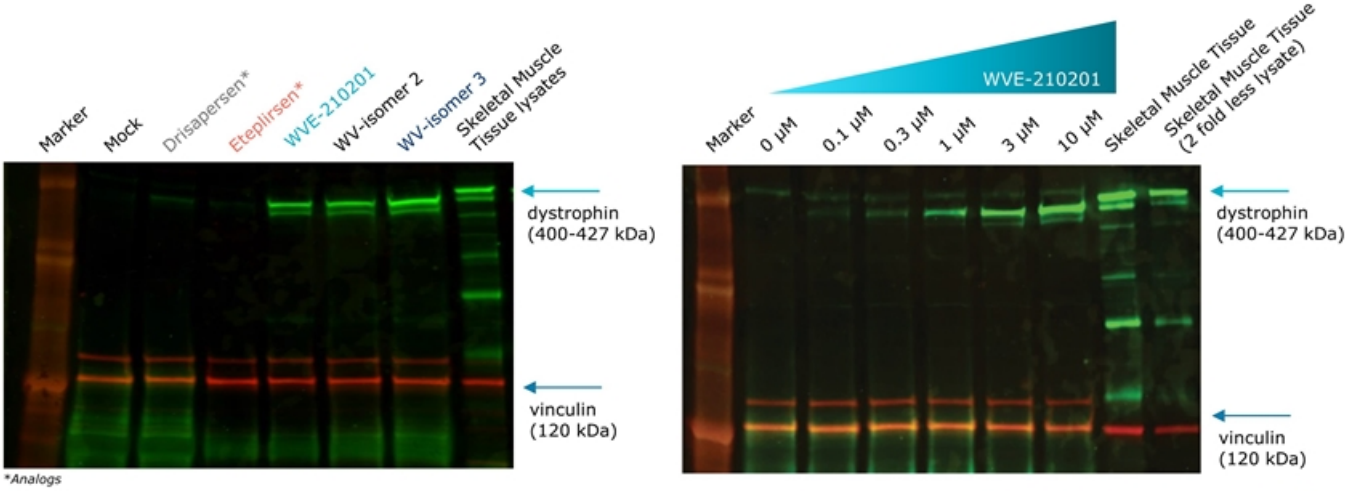
Exon 51: increased dystrophin restoration



Dystrophin protein restoration in vitro *was quantified to be between* ***50-100% of normal*** *skeletal muscle tissue lysates, as compared to about 1% by drisapersen and eteplirsen analogs*

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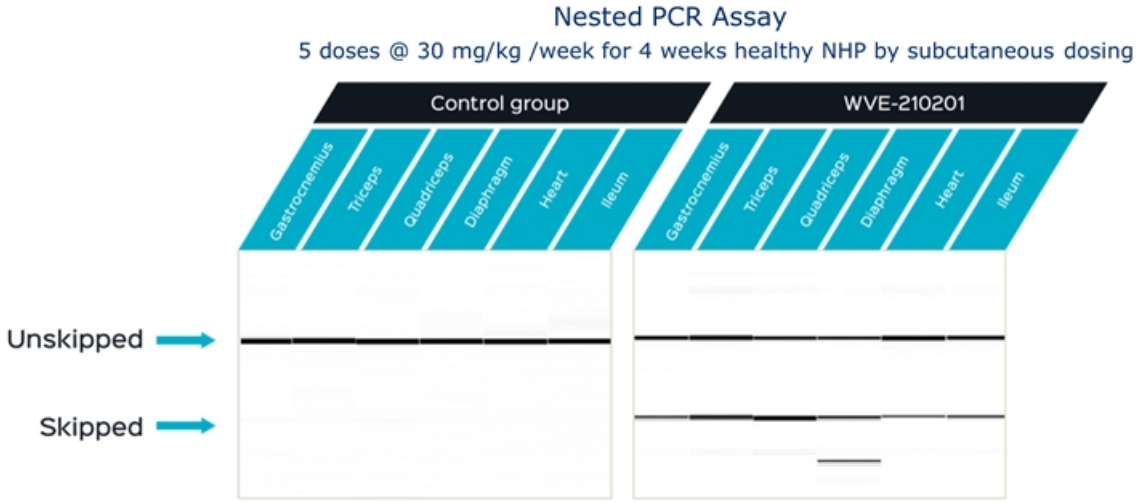
Experimental conditions: DMD protein restoration by Western Blot in patient-derived myotubes with clear dose effect. Free uptake at 10uM concentration of each compound with no transfection agent

Exon 51: in vivo target engagement of WVE-210201 in healthy non-human primate



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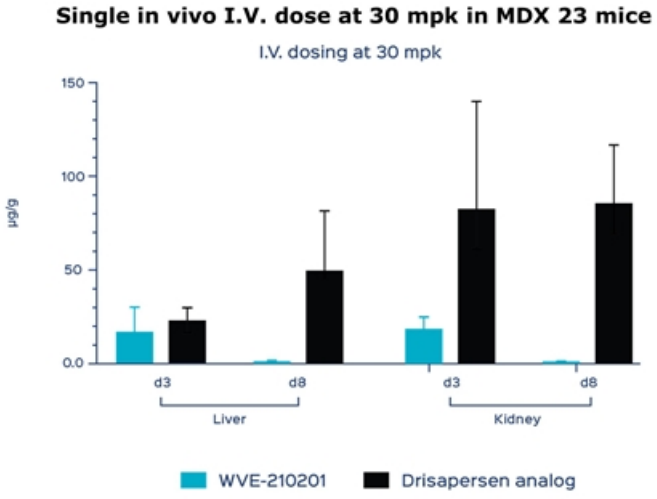
Experimental conditions: Muscle tissues were collected 2 days after the last dose and fresh frozen. Total RNAs were extracted with phenol/chloroform and converted to cDNA using high capacity kit. Nested PCR assay was performed and analyzed by fragment analyzer.

Exon 51: no apparent tissue accumulation observed

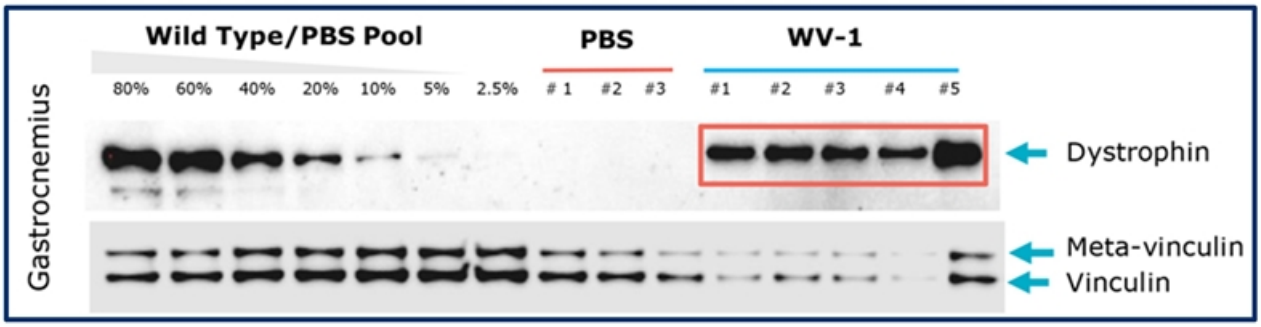
- Standard oligonucleotides tend to accumulate in liver and kidney
- Wave rationally designed oligonucleotides optimized to allow compound to clear more effectively
- WVE-210201 demonstrated wide tissue distribution in dose dependent fashion
- No apparent accumulation observed after multiple doses



Experimental description: Oligo quantifications in tissues were performed using hybridization ELISA assay

Wave stereopure surrogate yields substantial natural dystrophin protein restoration in *mdx 23* mice

70-90% of natural dystrophin production *in vivo*



- An exon 23 skipping molecule with a similar profile to WVE-210201
- Level of transcript production observed in vivo correlates strongly to what was observed in vitro at the same 10uM doses
- Protein production after 1 month of treatment (4 weekly doses)

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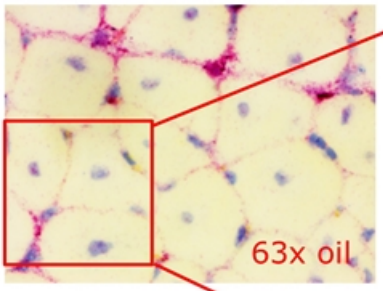
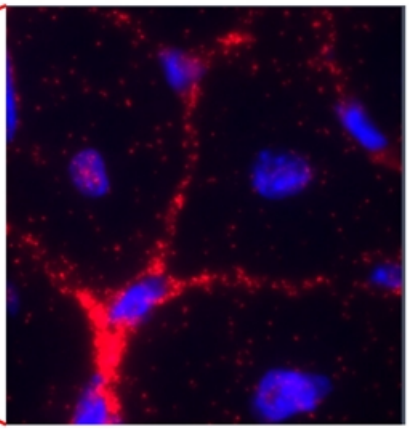
*Numbers indicate individual animals
Methods: *mdx* 23 mice received 4 weekly IV doses (150 mg/kg). Tissues collected 96 hours post final dose. Protein expression determined by western blot.

Exon 53: targeting oligonucleotide rapidly distributes to muscle within 24 hours after injection



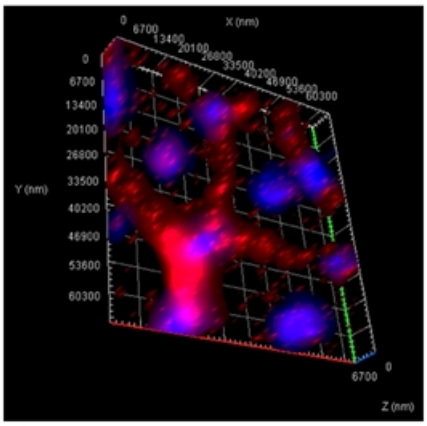
Bright field view

Nucleus: Hematoxylin; Light Blue
Wave oligo: ViewRNA, Fast Red



Fluorescence channel view

Nucleus: Hoechst33342; Blue
Wave oligo: Fast Red/Cy3; Pink Red



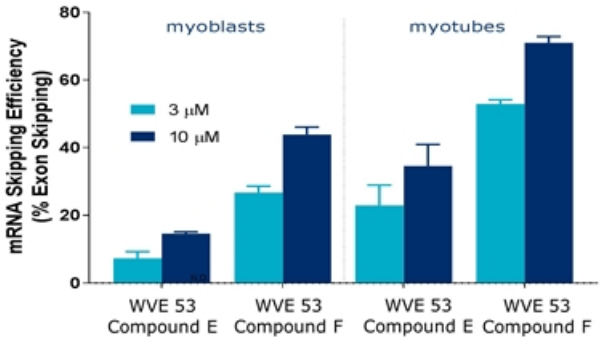
Z Stack view

Data derived from *in vivo* preclinical research.

Methods: A single dose of stereopure ASO 30 mg/kg IV was administered to *mdx* 23 mice. Tissues collected 24 hours post dose and ASO was detected in muscles using ViewRNA.

Exon 53 Program: improved skipping efficiency

Percentage Exon 53 Skipping of Preliminary Wave Isomers



- RNA skipping determined by quantitative RT-PCR
- Free uptake at 10uM and 3uM concentration of each compound with no transfection agent

Wave early Exon 53 data suggests skipping efficiency up to 70%

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C9orf72

Amyotrophic Lateral Sclerosis (ALS)

Frontotemporal Dementia (FTD)

C9orf72: a critical genetic risk factor

- C9orf72 gene provides instructions for making protein found in various tissues, with abundance in nerve cells in the cerebral cortex and motor neurons
- C9orf72 genetic mutations are the strongest genetic risk factor found to date for the more common, non-inherited (sporadic) forms of Amyotrophic Lateral Sclerosis (ALS) and Frontotemporal Dementia (FTD); GGGGCC repeat drives the formation and accumulation of dipeptide repeat proteins that accumulate in brain tissue
- First pathogenic mechanism identified to be a genetic link between familial (inherited) ALS and FTD
- Most common mutation identified associated with familial ALS and FTD
- Availability of dipeptide biomarker in CSF has potential to accelerate drug development

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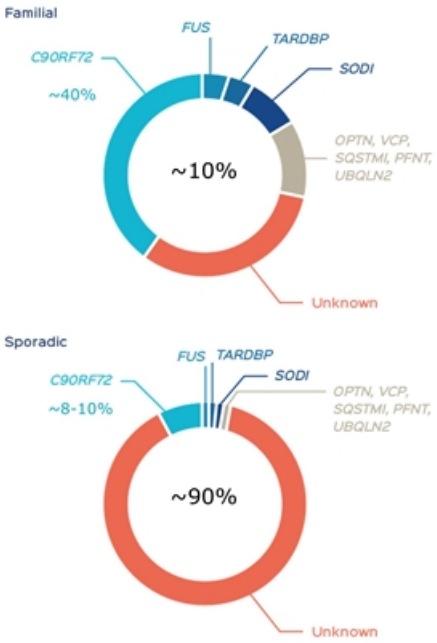
Amyotrophic lateral sclerosis

- Neurodegenerative disease characterized by the progressive degeneration of motor neurons in the brain and spinal cord
- Affects approximately 15,000-20,000 people in the US with a median survival of 3 years
- C9orf72 is present in approximately 40% of familial ALS and 8-10% of sporadic ALS; currently the most common demonstrated mutation related to ALS, far more so than SOD1 or TDP-43
- Pathogenic transcripts of the C9orf72 gene contain hundreds to thousands of hexanucleotide repeats compared to 2-23 in wild-type transcripts; dominant trait with high penetrance

Initiation of clinical study expected Q4 2018



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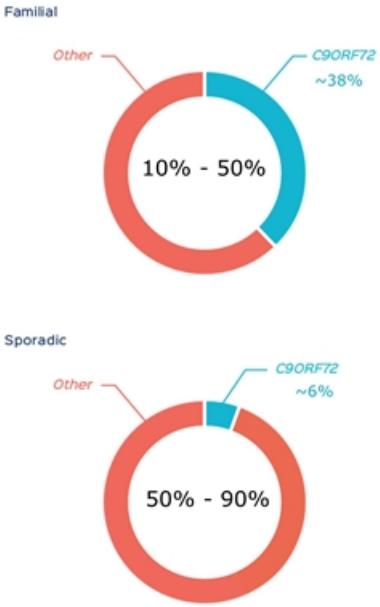
Source: State of play in amyotrophic lateral sclerosis genetics Alan E Renton, Adriano Chiò & Bryan J. Traynor Nature Neuroscience 17, 17–23 (2014) doi:10.1038/nn.3584

Frontotemporal dementia

- Progressive neuronal atrophy with loss in the frontal and temporal cortices characterized by personality and behavioral changes, as well as gradual impairment of language skills
- Affects approximately 55,000 people in the US
- Second most common form of early-onset dementia after Alzheimer's disease in people under the age of 65
- Up to 50% of FTD patients have a family history of dementia, many inheriting FTD as an autosomal dominant trait with high penetrance
- Pathogenic transcripts of the C9orf72 gene contain hundreds to thousands of hexanucleotide repeats compared to 2-23 in wild-type transcripts

Initiation of clinical study expected Q4 2018



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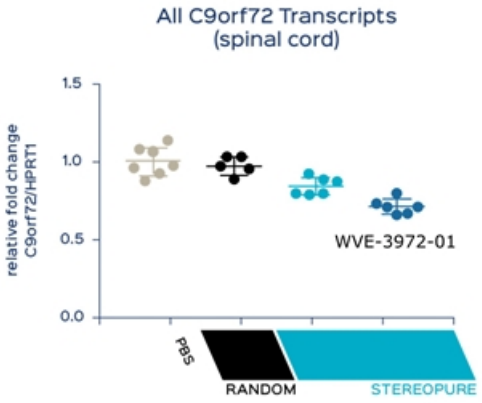
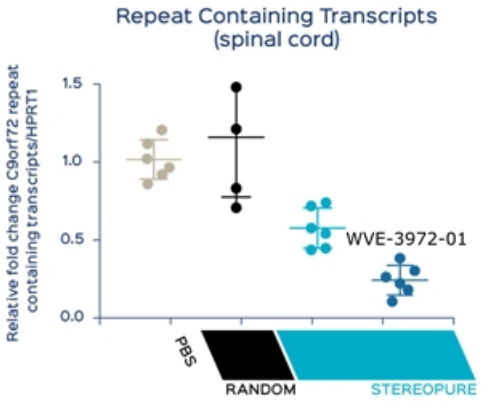
Sources: Familial aggregation in frontotemporal dementia, M. Stevens, MD; C.M. et al, Neurology 1998. Frequency of the C9orf72 hexanucleotide repeat expansion in patients with amyotrophic lateral sclerosis and frontotemporal dementia: a cross-sectional study. Elisa Majounie et al Lancet Neurology March 9, 2012 DOI:10.1016/S1474-4422(12)70043-1

Selective silencing in vivo of expanded C9orf72 repeat transcripts

- Wave has developed a series of highly optimized antisense compounds which selectively silence the repeat containing transcript in C9orf72 transgenic mice
- These compounds show target engagement across cell types and regions of the nervous system critically implicated in ALS and FTD





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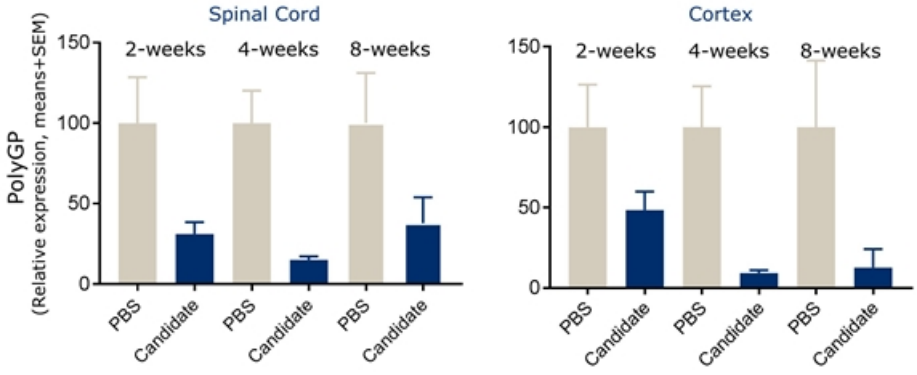
Experimental description: Samples were analyzed using quantitative PCR (Taqman assay)

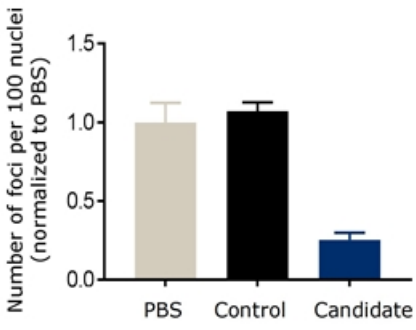
Durable reduction of dipeptides and RNA foci in vivo

- Wave's candidate (WVE-3972-01) demonstrates durable reduction of dipeptides and reductions in RNA foci
- Data is consistent across blinded studies in independent laboratories (collaboration with Professor Bob Brown, U. Mass)

Durable reduction of dipeptide in vivo



Reductions in RNA foci in vivo (8 weeks)



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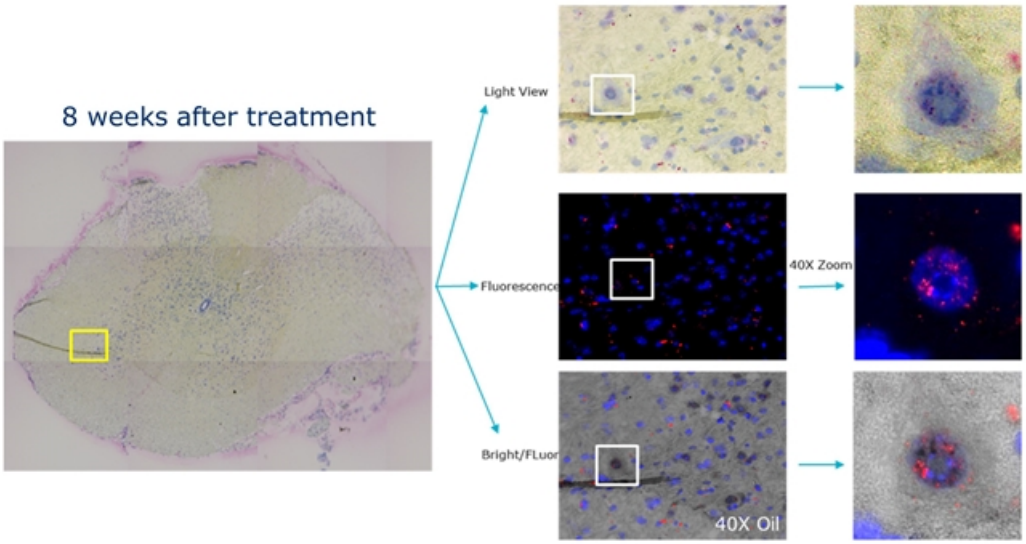
In vivo distribution of WVE-3972-01



Widespread and sustained distribution in nuclei of motor neurons in the spinal cord

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Experimental description: C9-BAC mice were administered 50μg of WVE-3972-01 ICV on day 1 and day 8; detection using ViewRNA.

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Partnerships

Collaborating to maximize portfolio and platform

Takeda

- **$230+ million** in committed cash; eligible for milestones and royalties in excess of $2 billion*
- Takeda option on **global 50:50 share** of CNS programs in HD, ALS, FTD and SCA3
- **Fully funded CNS R&D** with Takeda right to license additional preclinical CNS targets over 4 years

Pfizer

- **$40 million** upfront payment; **$871 million** in potential milestone payments and royalties
- **Advancing 5 targets**, including APOC3, for the treatment of metabolic liver diseases
- Leveraging **Wave proprietary chemistry platform** across modalities with GalNAc and Pfizer's hepatic targeting technology

Platform technologies

deep genomics

- Applying **artificial intelligence** to discover novel therapies for genetic neuromuscular disorders

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- Utilizing **3D imaging** to assess target engagement in specific regions, cell types and subcellular compartments of the brain

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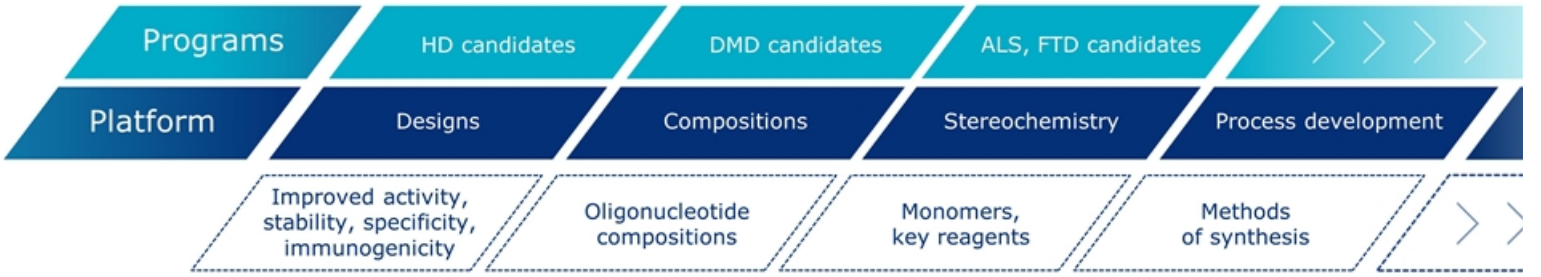
*Assuming Takeda advances six programs that achieve regulatory approval and commercial sales, Wave will be eligible to receive up to $2 billion in cash milestone payments, of which more than $1 billion would be in precommercial milestone payments.

Intellectual property strength: breadth and depth of patent portfolio



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Upcoming Wave catalysts

- **Q4 2018: safety data expected in DMD from Phase 1 trial for WVE-210201**
  - WVE-210201 is the first stereopure oligonucleotide targeting Exon 51
  - Received EU and US orphan drug designations and US rare pediatric disease designation
- **Q4 2018: clinical trials expected to initiate in ALS and FTD for WVE-3972-01**
  - WVE-3972-01 is designed to target the pathogenic allele of the C9orf72 gene
  - In vivo animal data demonstrate potent, sustained and preferential knockdown of toxic biomarkers
- **H1 2019: data expected in HD from Phase 1b/2a trials for WVE-120101 and WVE-120102**
  - Potential to be first two allele-specific disease-modifying therapies selectively lowering mHTT
  - Received US orphan drug designation
- **H2 2019: Interim dystrophin readout from ongoing open label extension and planned efficacy trials expected for WVE-210201**

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Realizing the potential of nucleic acid therapeutics

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