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): January 5, 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
(Address of principal executive offices)

018936
(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 at various industry and other conferences to provide updates and summaries of its business. On January 5, 2018, the Company updated its corporate presentation, which is available on the Investors & Media section of the Company's website at http://ir.wavelifesciences.com/. This presentation is attached as Exhibit 99.1 and is incorporated by reference herein.

The information in Item 7.01 of this Form 8-K, including Exhibit 99.1 attached hereto, is intended to be furnished and shall not be deemed "filed" for 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, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such a filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
99.1	Corporate presentation of Wave Life Sciences Ltd., dated as of January 5, 2018

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: January 5, 2018

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


WAVE
LIFE SCIENCES
Wave Life Sciences
Corporate Presentation
January 5, 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.

Genetic medicines company

Developing targeted therapies for patients impacted by rare diseases

- Rationally designed stereopure nucleic acid therapeutics
- Utilizing multiple modalities including antisense, exon skipping and RNAi
- 6 proprietary neurology development programs by the end of 2018
- Expertise and core focus in neurology
  - 2 Phase 1b/2a trials initiated in Huntington's disease
  - DMD Exon 51 trial initiated
  - Clinical data readouts anticipated in 2019 for first 3 programs
- Robust R&D platform, ability to partner additional therapeutic areas
- Cash position $169MM as of September 30 2017

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Paving the way to potentially safer, more effective medicines

1
first to design and bring stereopure and allele-specific medicines to clinic

6
neurology development programs by end of 2018

3
clinical studies initiated in 2017

10K+
Wave stereopure oligonucleotides created and analyzed to date

5
nucleic acid modalities being advanced with Wave stereopure chemistry

12+
discovery programs

5
therapeutic areas under active investigation

25M+
total potentially addressable patients amenable to Wave's partnered and proprietary programs

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Pipeline spanning multiple modalities, novel targets

	DISEASE	TARGET	BIOMARKER	ESTIMATED U.S. ADDRESSABLE PATIENTS *	MECHANISM	DISCOVERY	CANDIDATE	CLINICAL	NEXT ANTICIPATED MILESTONES
CNS	Huntington's disease	mHTT SNP1	mHTT	~10k / ~35k	A	●	●	Phase 1b/2a	Top line data 1H 2019
	Huntington's disease	mHTT SNP2	mHTT	~10k / ~35k	A	●	●	Phase 1b/2a	Top line data 1H 2019
	Amyotrophic lateral sclerosis	C9orf72	dipeptide	~1,800	A	●	●		Trial initiation Q4 2018
	Frontotemporal dementia	C9orf72	dipeptide	~7,000	A	●	●		Trial initiation Q4 2018
MUSCLE	Duchenne muscular dystrophy 51	exon 51	dystrophin	~2,000	E	●	●	Phase 1	Top line data Q3 2018
	Duchenne muscular dystrophy 53	exon 53	dystrophin	~1,250	E	●	○		Trial initiation Q1 2019
HEPATIC	Pfizer	APOC3			○	●	○		
	Pfizer	undisclosed			○	●	○		
	Pfizer	undisclosed			○	●	○		

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

Neurology leadership

Current programs

- **Huntington's disease (HTT SNP1)**
- **Huntington's disease (HTT SNP2)**
- **Duchenne muscular dystrophy (exon 51)**
- **Duchenne muscular dystrophy (exon 53)**
- **Amyotrophic lateral sclerosis (C9orf72)**
- **Frontotemporal dementia (C9orf72)**

Discovery engine

Neuromuscular diseases
- DMD (additional exons)
- Spinal muscular atrophy (SMN2)
- Charcot-Marie-Tooth type 1A (PMP22)

Neurodegenerative movement disorders
- Spinocerebellar ataxia (ATXN3)

Opportunities for expansion

Neurodegenerative movement disorders
- Parkinson's disease
- Progressive supranuclear palsy

Neurodegenerative dementias
- Alzheimer's disease

Developmental diseases
- Fragile X
- Batten disease

Neurophysiology/ neuropsychiatry/pain
- Epilepsy
- Schizophrenia

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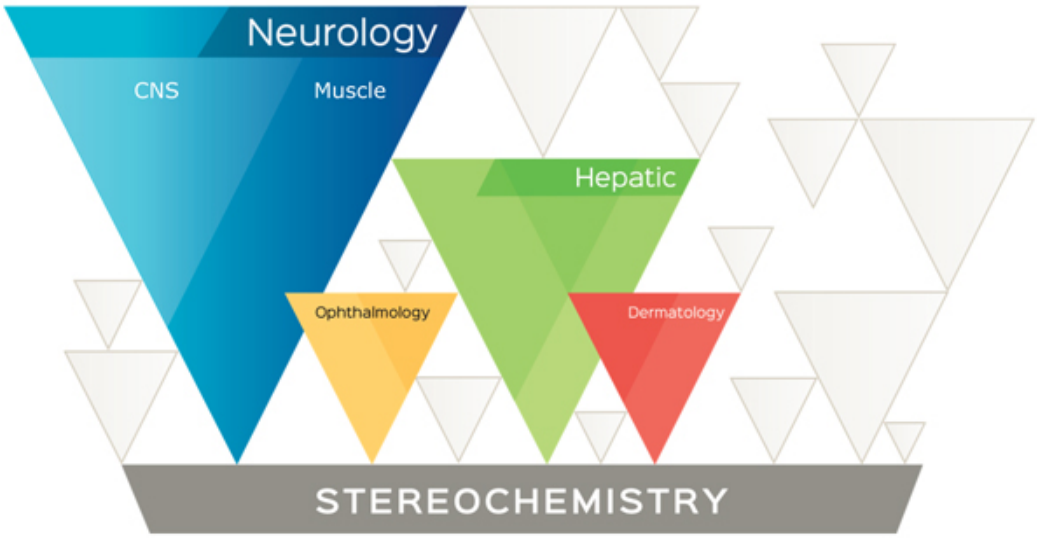
Broad platform relevance across therapeutic areas



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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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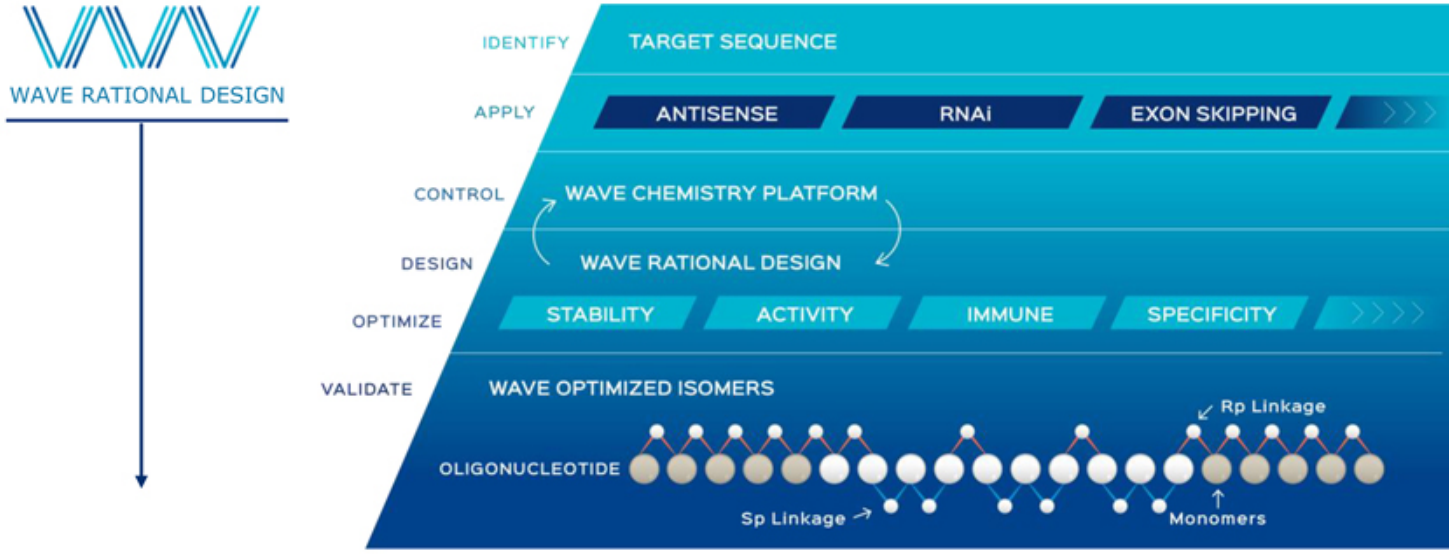
Creating a new class of oligonucleotides



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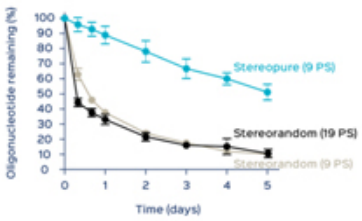
Source: Iwamoto N, et al. Control of phosphorothioate stereochemistry substantially increases the efficacy of antisense oligonucleotides. Nature Biotechnology. 2017.

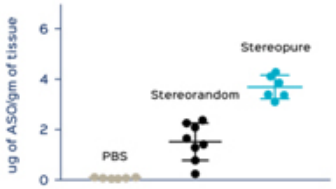
Chemistry may optimize medicines across multiple dimensions

Improved Stability

Stability of stereopure molecules with reduced PS content (liver homogenate)

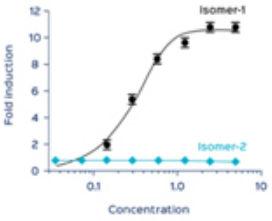


Oligonucleotide exposure (spinal cord)

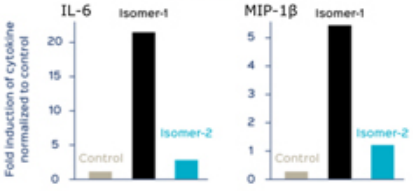


Controlled Immunogenicity

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

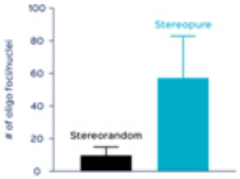


Cytokine induction in human PBMC assay

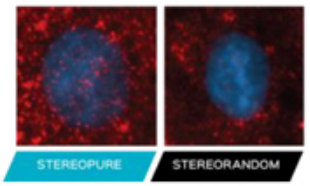


Enhanced Delivery

Stereochemistry enables enhanced delivery of oligonucleotides



Uptake without transfection agent between a stereopure and stereorandom oligonucleotide



Gymnotic uptake of ASOs: 18h differentiating myoblasts

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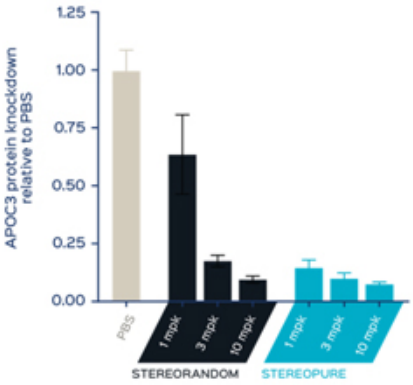
Data represented in this slide from in vitro studies. Experimental conditions: Human TLR9 assay – Source: Ohto U, et al. Structural basis of CpG and inhibitory DNA recognition by Toll-like receptor 9, Nature 520, 702-705, 2015. Intracellular trafficking assay – Cells were washed and fixed and oligos were detected by viewRNA assay and visualized on immunofluorescence microscope with deconvolution capabilities. Z-stacks were taken to eliminate artifacts.

Stereochemistry is applicable across modalities

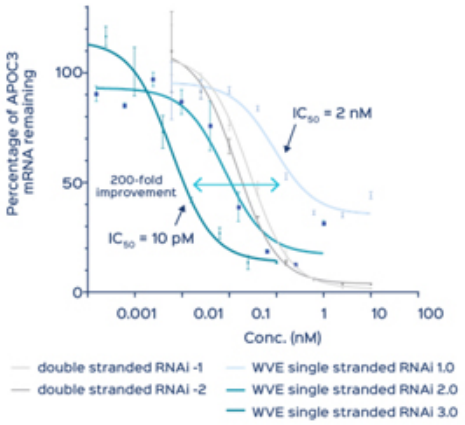
Antisense

In vivo potency and durability
(APOC3 transgenic mice, day 15)



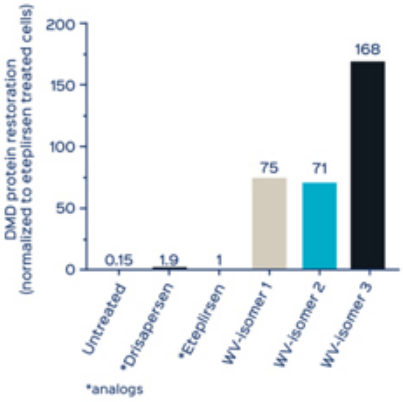
RNAi

200 Fold Higher Potency from
Original ssRNAi Designs



Exon skipping

71-168 Fold Increase in
Protein Restoration *
Compared to Eteplirsen



Stereochemistry allows for novel approaches to previously difficult diseases and inaccessible targets

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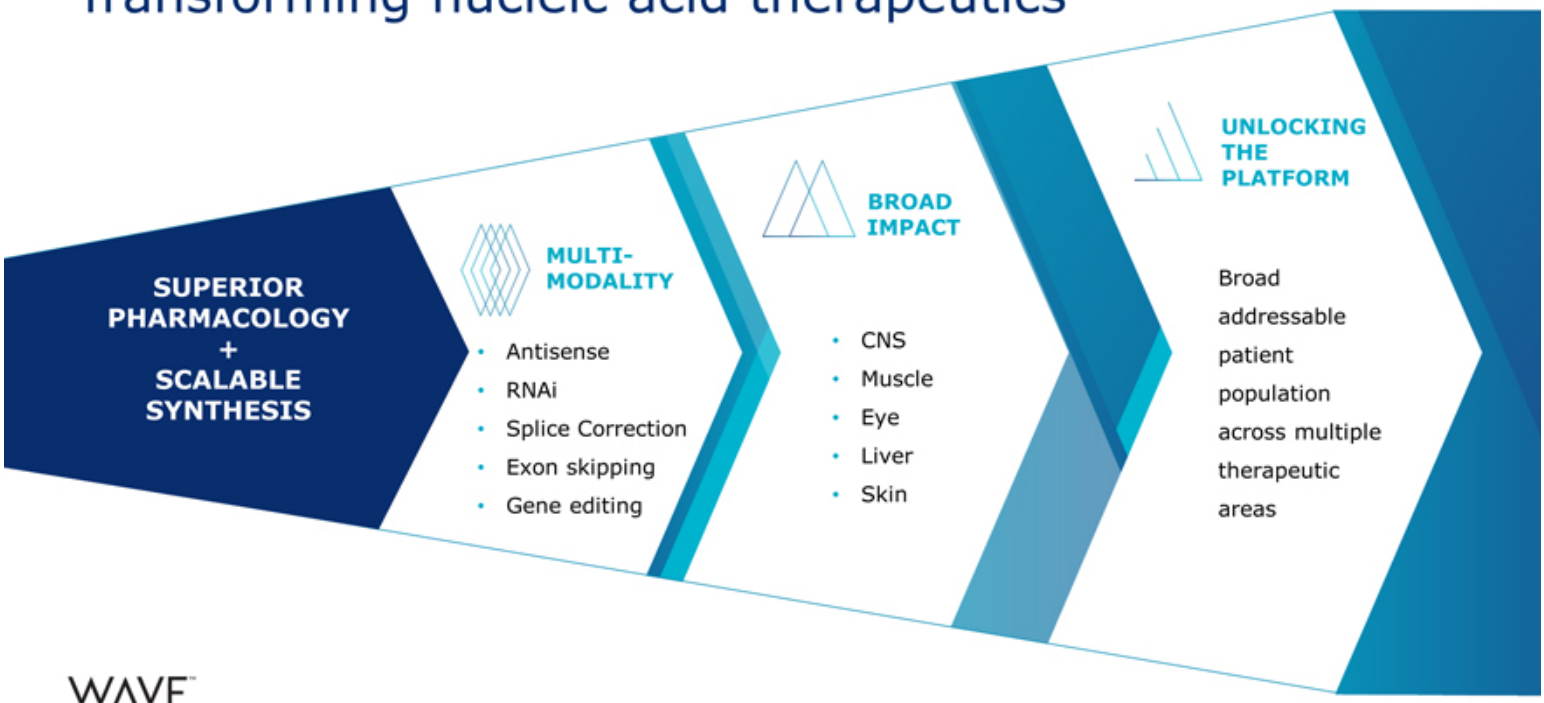
Transforming nucleic acid therapeutics



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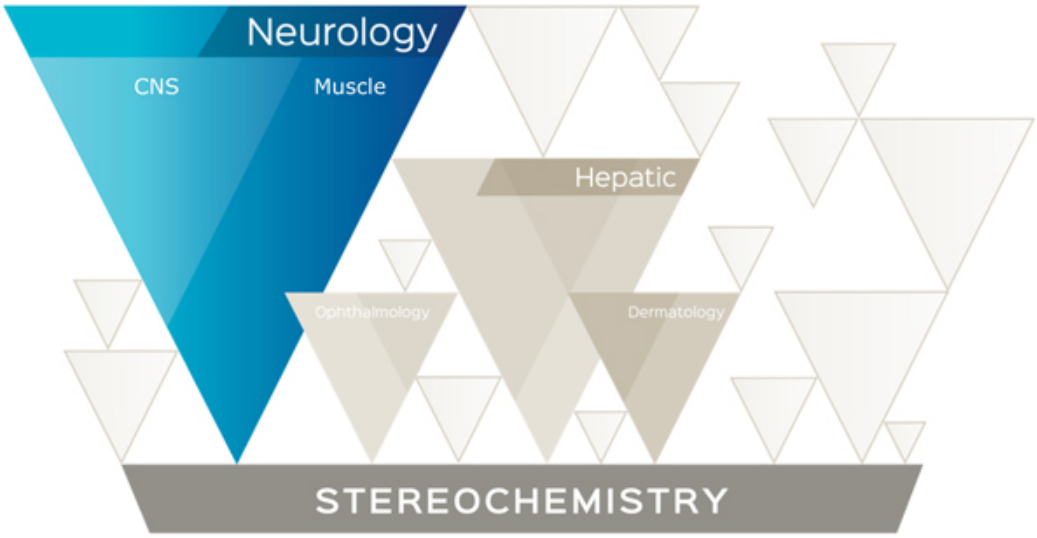
Neurology



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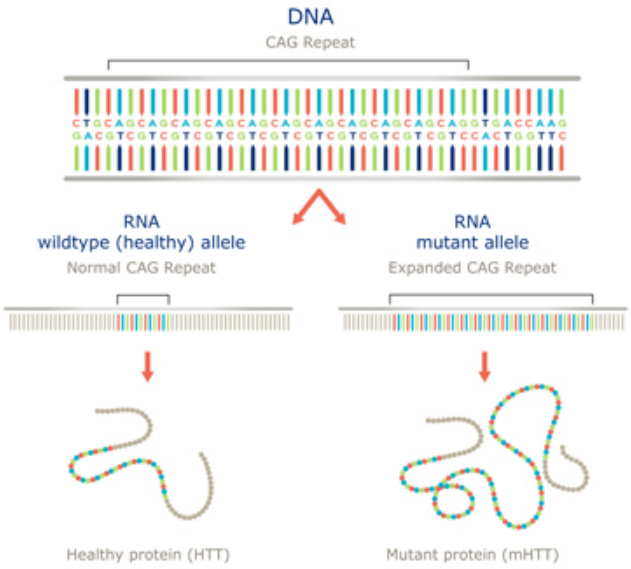
Huntington's Disease

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



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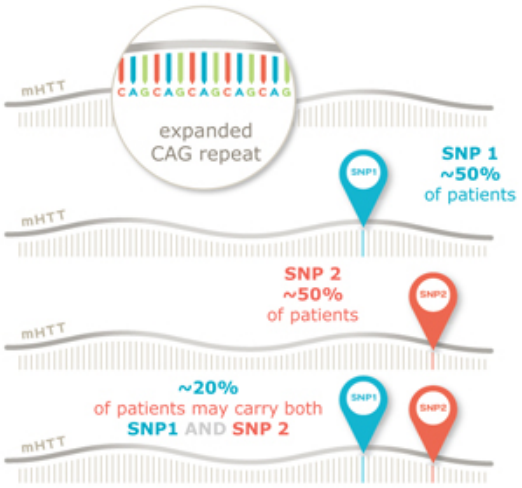
Sources: Auerbach W, et al. Hum Mol Genet. 2001;10:2515-2523. Dragatsis I, et al. Nat Genet. 2000;26:300-306. Leavitt BR, et al. J Neurochem. 2006;96:1121-1129. Nasir J, et al. Cell. 1995;81:811-823. Reiner A, et al. J Neurosci. 2001;21:7608-7619. White JK, et al. Nat Genet. 1997;17:404-410. Zeitlin 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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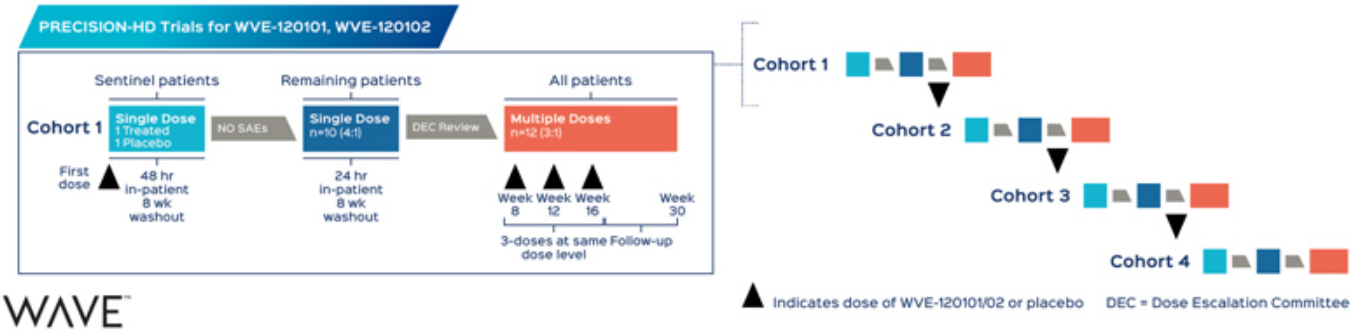
Source: Kaye, et al. Personalized gene silencing therapeutics for Huntington disease. Clin Genet 2014: 86: 29–36

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
- Blood test to determine presence of SNP 1 or SNP 2 done at pre-screening
- Approximately 50 patients per trial
- Key inclusion criteria: age ≥25 to ≤65, stage I or II HD
- Top line data anticipated 1H 2019



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Mutant huntingtin: a powerful, novel biomarker

- Novel immunoassay allows for quantification of mutant huntingtin, the cause of HD
- Level of mHTT detected is associated with time to onset, increased with disease progression, and predicts diminished cognitive and motor dysfunction
- Assay currently being utilized in clinical studies

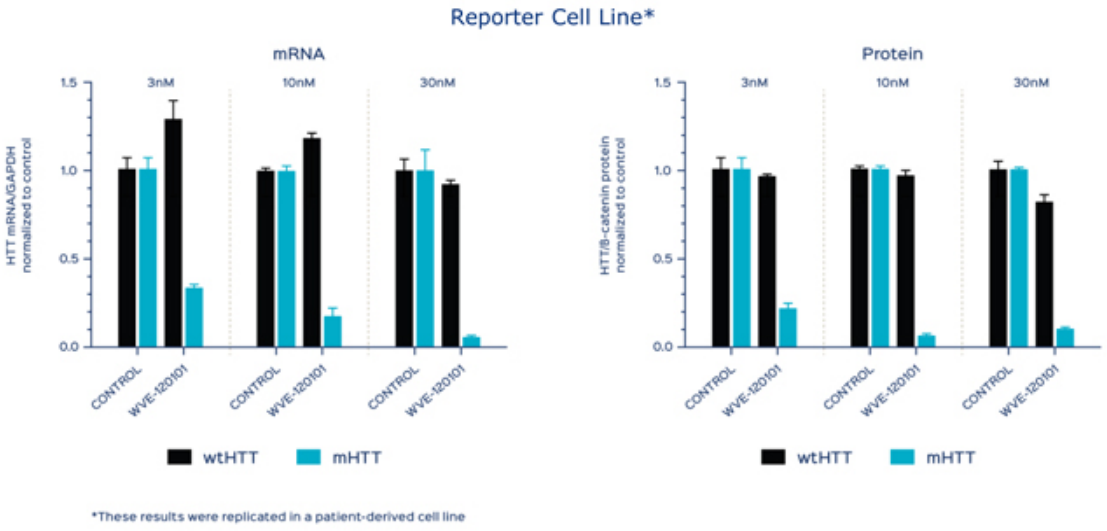
Novel approach enables precise measurement of target engagement and effect


Quantification of mutant huntingtin protein in cerebrospinal fluid from Huntington's disease patients

WAVE LIFE SCIENCES

Source: Wild E, et al. Quantification of mutant huntingtin protein in cerebrospinal fluid from Huntington's disease patients. J. Clin. Invest. 2015:125:1979–1986. Edward Wild, MA MB BChir PhD MRCP Principal Investigator at UCL Institute of Neurology and Consultant Neurologist at the National Hospital for Neurology and Neurosurgery, London

Selective reduction of mHTT mRNA & protein



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

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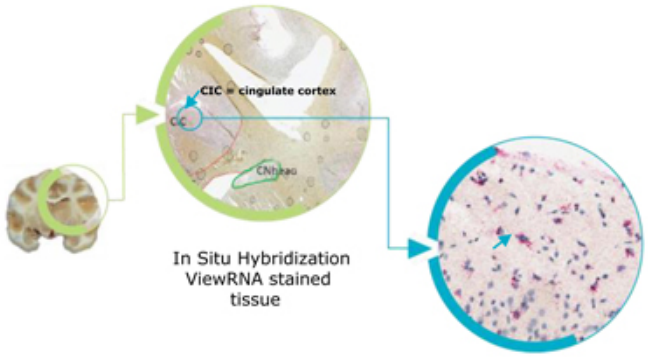
Demonstrated delivery to brain tissue

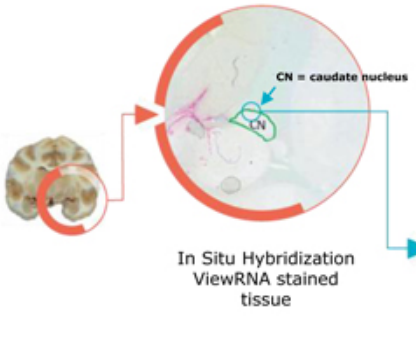
- WVE-120101 and WVE-120102 distribution in cynomolgus non-human primate brain following intrathecal bolus injection



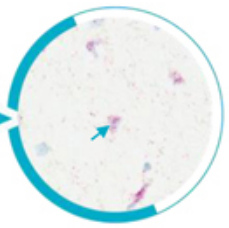
In Situ Hybridization ViewRNA stained tissue

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 3,500 newborn boys each year; 20,000 new cases annually worldwide

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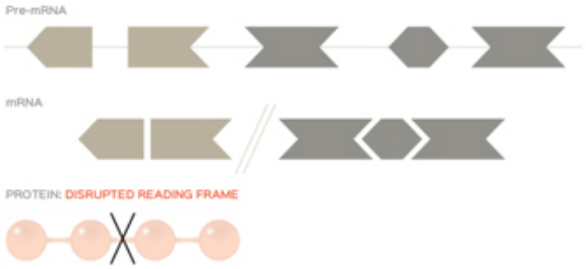
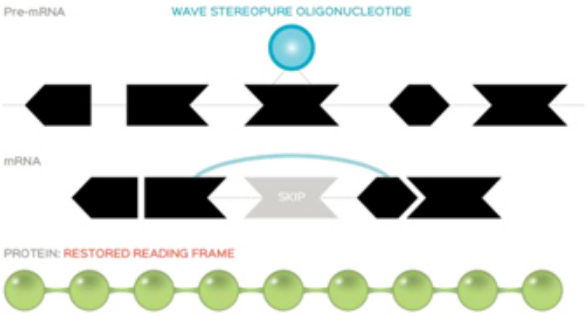
Wave approach:
meaningful restoration of dystrophin production through exon skipping

- 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 Q3 2018
  - Planned open-label extension (OLE) with muscle biopsy and ≥2-years of follow-up
- WVE-210201 planned efficacy study
  - 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
  - Efficacy readout anticipated 2H 2019
- Exploring intravenous and subcutaneous formulations for WVE-210201

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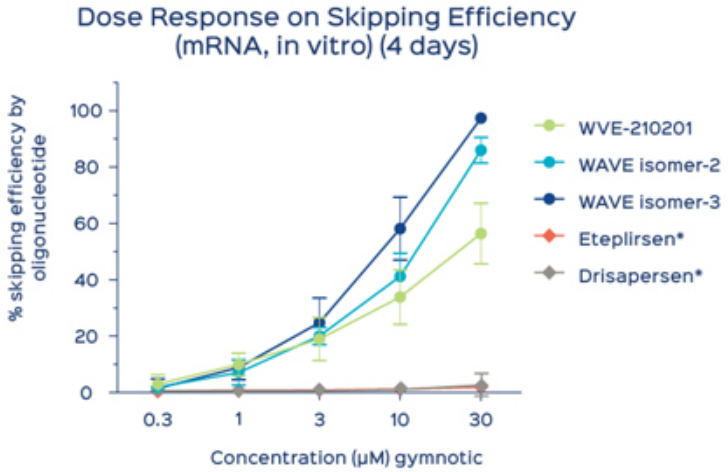
Exon 51: improved skipping efficiency

- 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



*analogs

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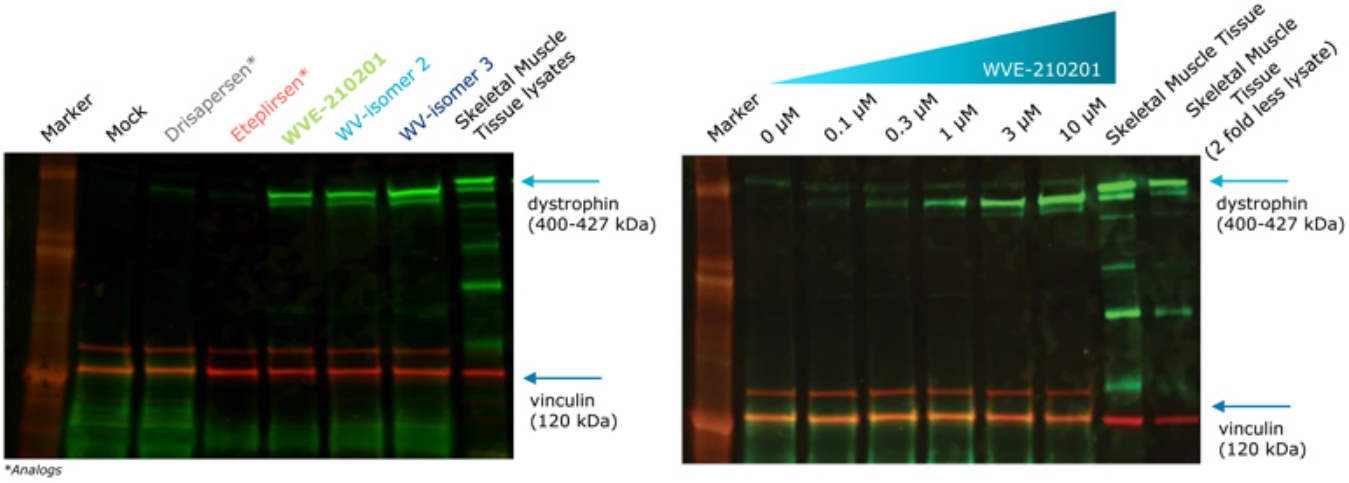
Exon 51: increased dystrophin restoration

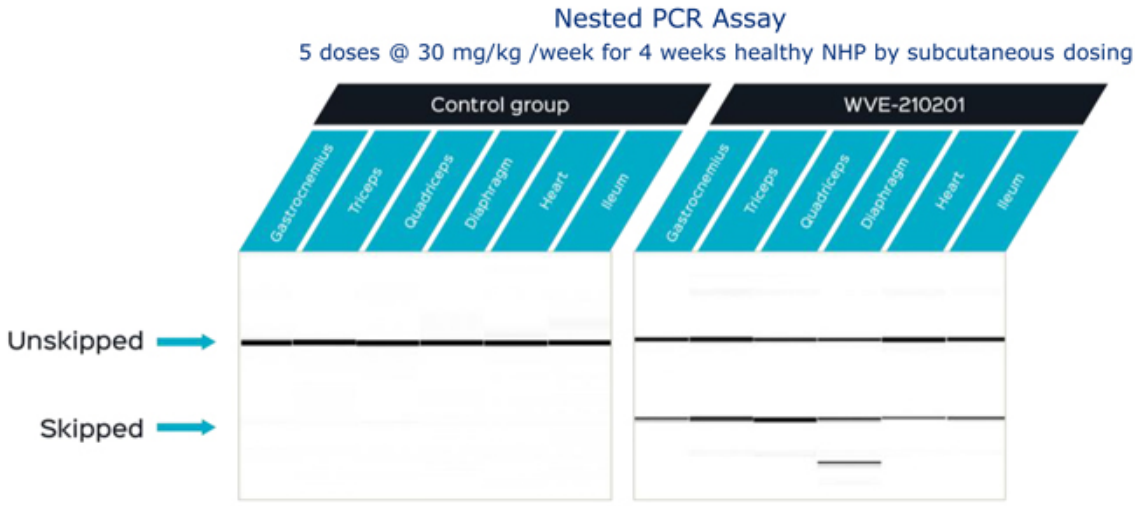


Dystrophin protein restoration in vitro *was quantified to be between* ***50-100% of normal*** *skeletal muscle tissue lysates, as compared to about 1% by drisapersen and eteplirsen analogs*

WAVE LIFE SCIENCES

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: target engagement in healthy non-human primate



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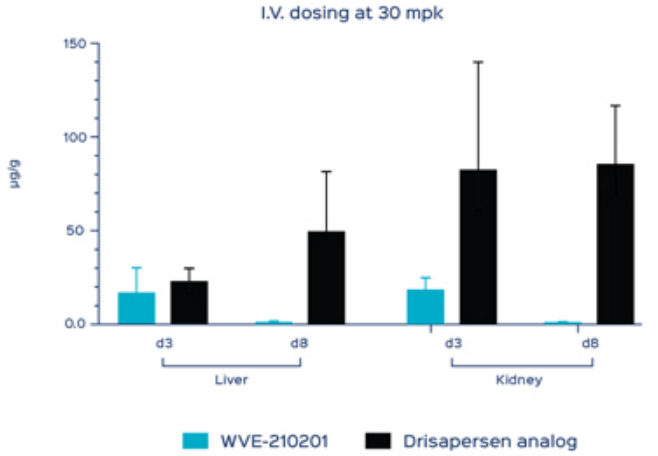
Experimental conditions: Muscle tissues were collected 2 days after the last dose and fresh frozen. Total RNAs were extracted with phenol/chloroform and converted to cDNA using high capacity kit. Nested PCR assay was performed and analyzed by fragment analyzer.

Exon 51: no apparent tissue accumulation observed

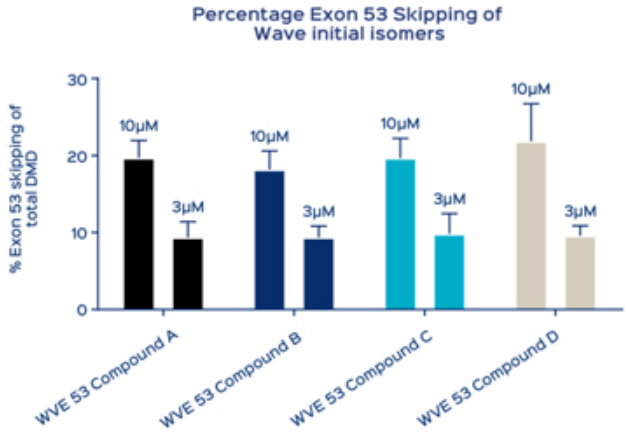
- Standard oligonucleotides tend to accumulate in liver and kidney
- Wave rationally designed oligonucleotides optimized to allow compound to clear more effectively
- WVE-210201 demonstrated wide tissue distribution in dose dependent fashion
- No apparent accumulation observed after multiple doses

Single in-vivo I.V. dose at 30 mpk in MDX 23 mice



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

Exon 53:
Stereopure lead molecules advancing toward candidate



- RNA skipping determined by quantitative RT-PCR
- Free uptake at 10uM and 3uM concentration of each compound with no transfection agent
- Current published clinical dystrophin levels achieved for Exon 53 are ~1%

Early Exon 53 data suggests initial skipping efficiency around 20% pre-optimization

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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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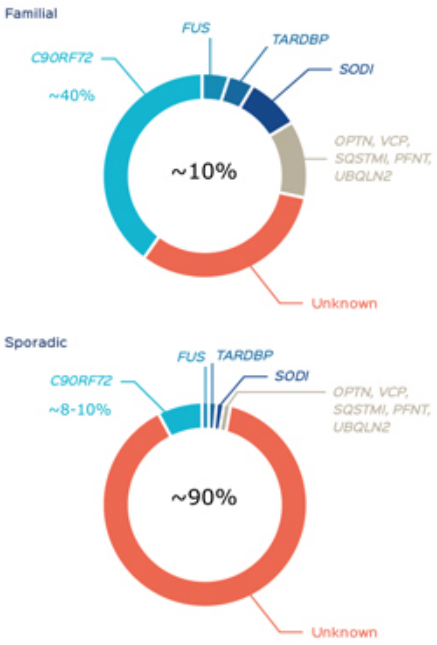
Amyotrophic lateral sclerosis

- 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 4Q '18



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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 4Q '18

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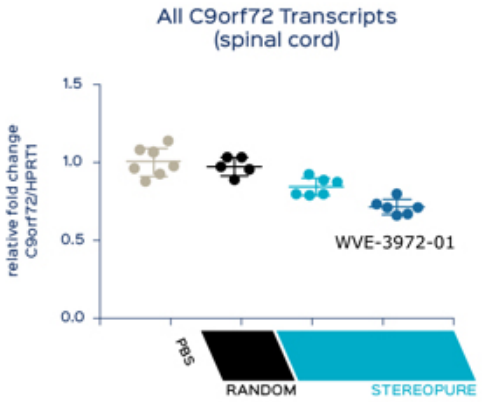
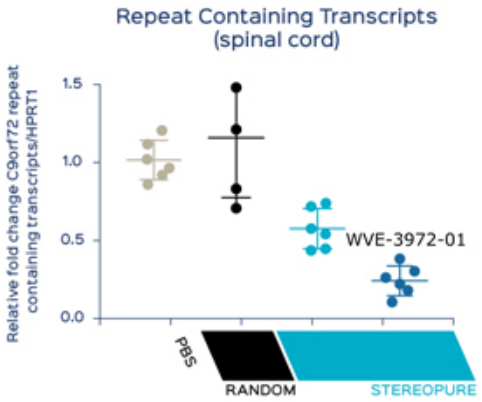
Sources: Familial aggregation in frontotemporal dementia, M. Stevens, MD; C.M. et al, Neurology 1998. Frequency of the C9orf72 hexanucleotide repeat expansion in patients with amyotrophic lateral sclerosis and frontotemporal dementia: a cross-sectional study. Elisa Majounie et al Lancet Neurology March 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





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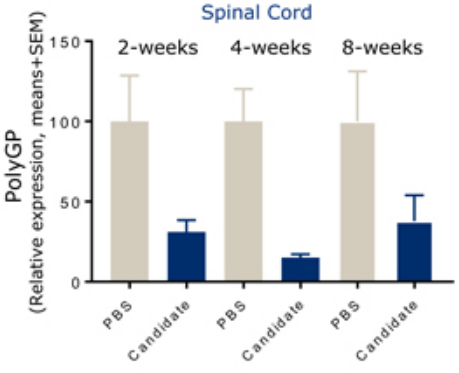
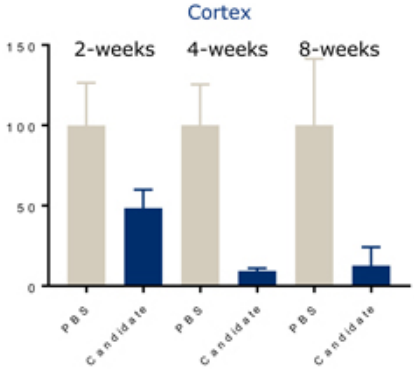
Experimental description: Samples were analyzed using quantitative PCR (Taqman assay)

Durable reduction of dipeptides and RNA foci in vivo

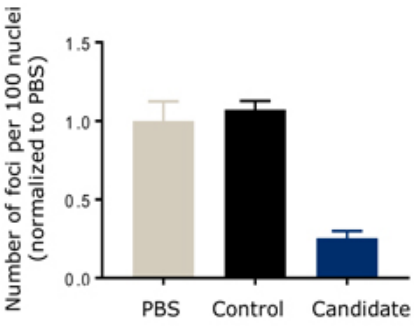
- Wave's candidate (WVE-3972-01) demonstrates durable reduction of dipeptides and reductions in RNA foci
- Data is consistent across blinded studies in independent laboratories (collaboration with Professor Bob Brown, U. Mass)

Durable reduction of dipeptide in vivo





Reductions in RNA foci in vivo (8 weeks)



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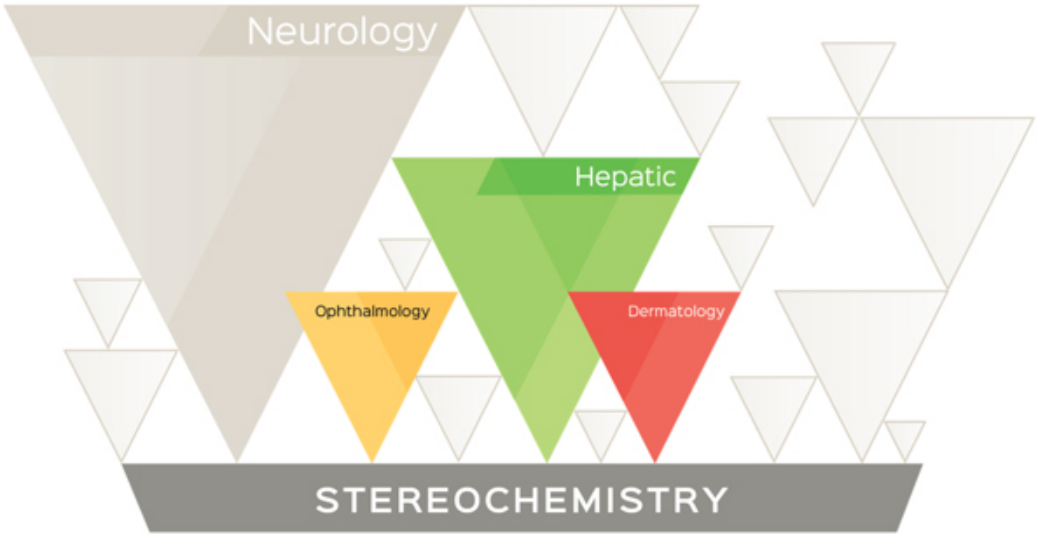
Emerging areas



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Pfizer hepatic collaboration

- Initiated May 2016
- Exploring targets across modalities, including ASO and ssRNAi
- Up to 5 hepatic-metabolic programs
  - 3 targets declared; APOC3, 2 undisclosed
  - Option to declare 2 additional targets
- Access to Pfizer's hepatic targeting technology
  - Potentially increasing potency beyond GalNAc
  - Freedom to leverage beyond collaboration targets

40

$M upfront payment

871

$M in potential milestone payments and royalties

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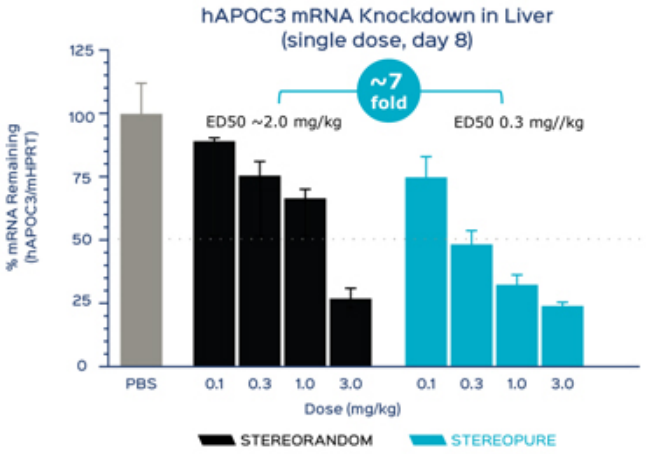
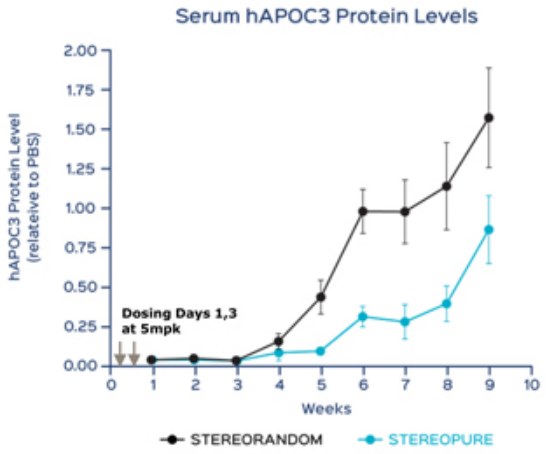
Stereopure ASOs: improved in vivo potency, extended duration

- Potency equivalent to state-of-the-art GalNAc conjugated double strand RNAi (ED50 0.3 mg/kg)
- Demonstrated increase in durability over GalNAc conjugated stereorandom





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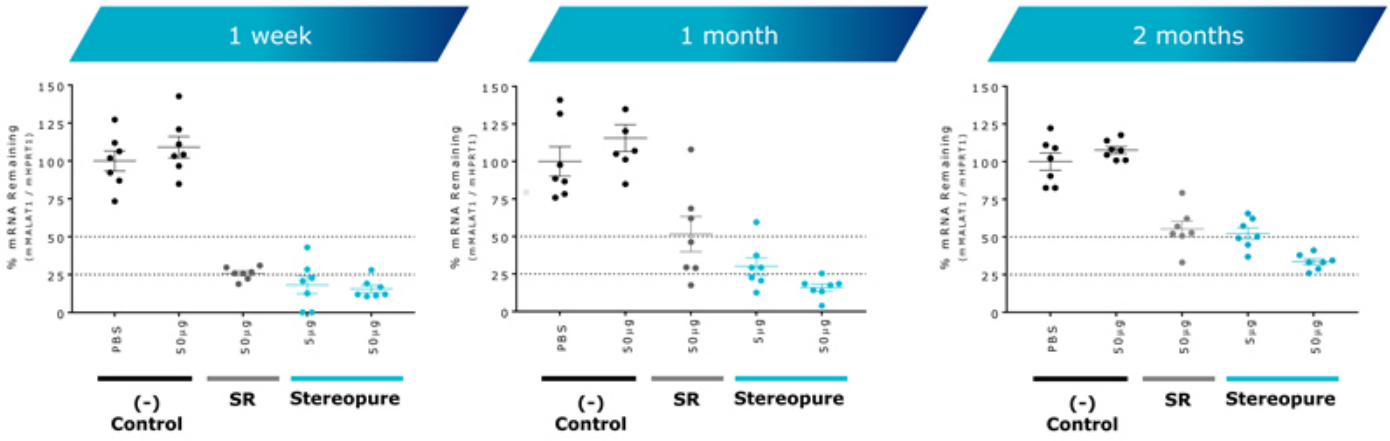
Experimental description: Male human APOC3 transgenic mice were dosed with APOC3 ASOs with indicated doses. APOC3 mRNA quantification in the liver was performed using Taqman assay specific for hAPOC3. For protein analysis, plasma samples were collected weekly and analyzed by ELISA assay specific to human APOC3 protein.

Stereopure ASOs: improved in vivo potency, extended duration

Back of the eye



10X lower dose of stereopure oligo is more potent than stereorandom oligo

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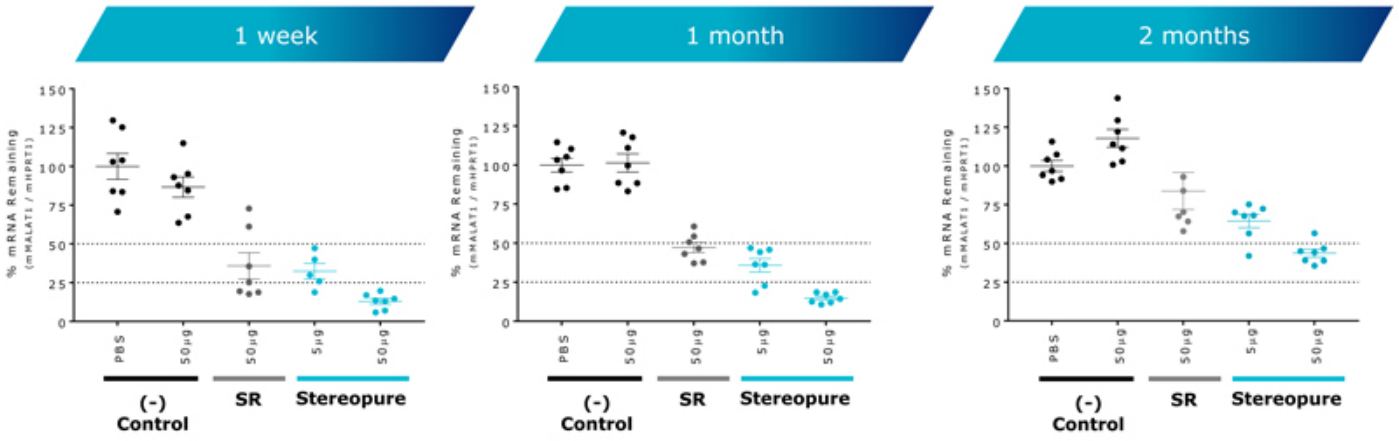
Experimental description: Single intravitreal injection to mouse eye on day 1.

Stereopure ASOs: improved in vivo potency, extended duration

Front of the eye



10X lower dose of stereopure oligo is more potent than stereorandom oligo

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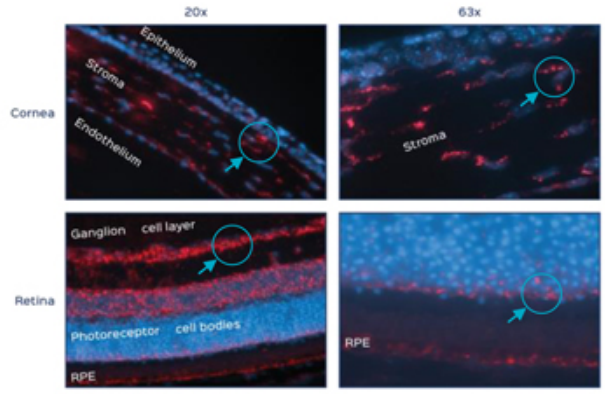
Experimental description: Single intravitreal injection to mouse eye on day 1.

Distribution and target engagement

Ophthalmology

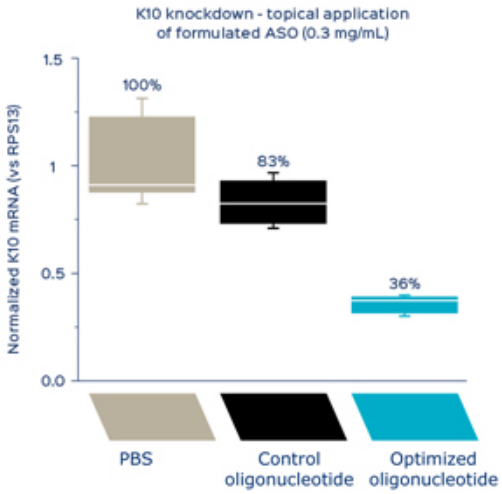
Distribution of oligonucleotide to key cellular Compartments following intravitreal injection in murine eye



Red dots = Oligonucleotides

Dermatology

Target engagement following topical administration on human skin explant model



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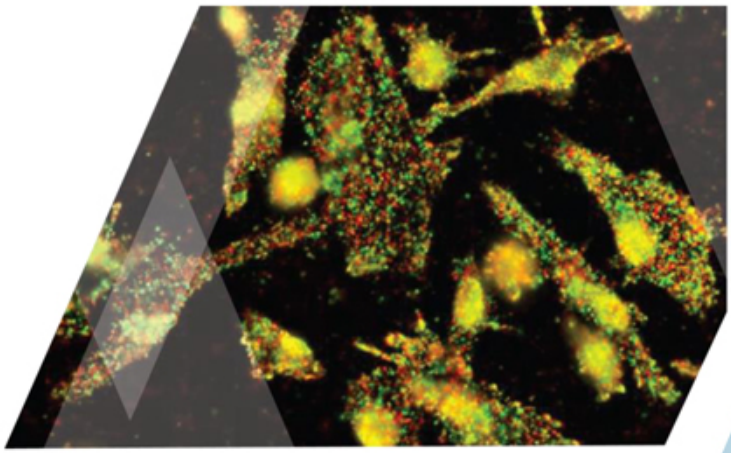
Enabling technologies: enhancing stereopure platform

READCOOR

- Collaboration leverages ReadCoor's proprietary FISSEQ (Florescent In-Situ Sequencing) platform designed to provide critical spatial data by combining next generation sequencing and three-dimensional imaging
- Developing a registry of brain cell network maps
- Advancing chemistry for targeted delivery to the brain



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Scalable nucleic acid synthesis

- Oligonucleotide synthesis capacity ranging from high throughput to large scale GMP production
- 90,000 square foot facility
- Ability to continue to meet synthesis demands of growing portfolio and increase control and visibility of product supply chain
- Comparable yield and cost-of-goods to standard stereorandom oligonucleotides
- Industry standard equipment with no biological processing required
- GMP manufacturing capacity potentially available to partners

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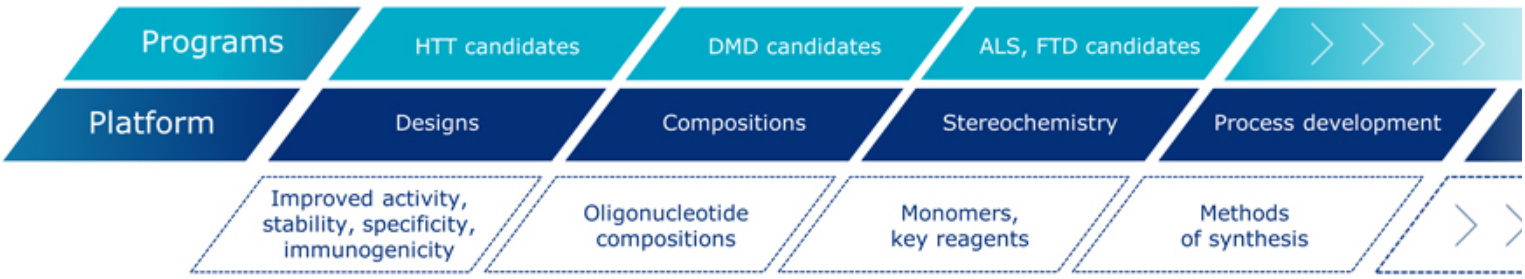
Secure patent and intellectual property position



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Wave catalysts

- **Q3 2018: data expected in DMD from Phase 1 trial for WVE-210201**
  - Initiated clinical trial in DMD (Exon 51) November 2017
  - WVE-210201 is the first stereopure oligonucleotide targeting Exon 51 with potential to be best-in-class
  - Interim dystrophin readout from planned efficacy and open label extension trials expected in 2H 2019

- **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 associated with ALS and FTD

- **1H 2019: data expected in HD from Phase 1b/2a trials for WVE-120101 and WVE-120102**
  - Initiated two clinical trials in HD July 2017
  - Potential to be first two allele-specific disease-modifying therapies selectively lowering mHTT
  - Received U.S. orphan drug designation for WVE-120101 and WVE-120102

- **Q1 2019: clinical trial expected to initiate for next DMD program (Exon 53)**

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Realizing the potential of nucleic acid therapeutics