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): August 9, 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)	**(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 2.02 Results of Operations and Financial Condition.

On August 9, 2018, Wave Life Sciences Ltd. (the "Company") announced its financial results for the quarter ended June 30, 2018. The full text of the press release issued in connection with the announcement is furnished as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated by reference herein.

Item 7.01 Regulation FD Disclosure.

From time to time, the Company presents and/or distributes slides and presentations to the investment community to provide updates and summaries of its business. On August 9, 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 99.2 to this Current Report on Form 8-K.

The information in this Current Report on Form 8-K furnished pursuant to Items 2.02 and 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 Items 2.02 and 7.01 of this Current Report on Form 8-K.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

The following exhibits relating to Items 2.02 and 7.01 shall be deemed to be furnished and not filed:

Exhibit No.	Document
99.1	Press Release issued by Wave Life Sciences Ltd. dated August 9, 2018
99.2	Corporate Presentation of Wave Life Sciences Ltd. dated August 9, 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: August 9, 2018

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

Wave Life Sciences Reports Second Quarter 2018 Financial Results and Provides Business Update

CAMBRIDGE, Mass, August 9, 2018 – Wave Life Sciences Ltd. (NASDAQ: WVE), a biotechnology company focused on delivering transformational therapies for patients with serious, genetically-defined diseases, today announced financial results for the second quarter ended June 30, 2018 and provided a business update.

"We continue to advance our programs for the treatment of Duchenne muscular dystrophy with preparations underway for our global, pivotal trial of investigational WVE-210201 for boys amenable to exon 51 skipping. Recently presented preclinical data demonstrating that our stereopure oligonucleotides can restore up to 90% of natural dystrophin in an animal model underscore the significant potential of our platform to address Duchenne muscular dystrophy," said Paul Bolno, MD, MBA, President and Chief Executive Officer of Wave Life Sciences. "Through the rest of the year, we look forward to sharing Phase 1 safety data on WVE-210201, presenting advances in our Duchenne muscular dystrophy exon 53 skipping program, and maintaining strong momentum in the ongoing PRECISON-HD clinical program, the first clinical study conducted in the United States using an oligonucleotide approach for Huntington's disease."

Second Quarter Highlights and Business Update

- **WVE-210201 DMD exon 51 targeting program**

  The ongoing single ascending dose Phase 1 clinical trial of WVE-210201 in Duchenne muscular dystrophy (DMD) patients amenable to exon 51 skipping continues to progress through the planned dose escalation with no safety signals observed in the trial. Wave now expects to announce safety data from the Phase 1 trial by the end of the fourth quarter of 2018. Patient interest and enrollment in the trial continue to be strong. As patients complete the Phase 1 trial, they have the option to enroll in an ongoing open label extension study in which they receive continued treatment with WVE-210201.

  Wave remains on track to deliver an interim efficacy readout of dystrophin expression from muscle biopsies from ongoing and planned clinical trials in the second half of 2019.

  Wave has designed a global, pivotal, placebo-controlled Phase 2/3 efficacy and safety study of WVE-210201 in DMD patients amenable to exon 51 skipping, informed by ongoing discussions with global regulatory authorities and the DMD patient community. The study will be powered to assess clinical efficacy and will include dystrophin expression readouts as part of interim and final analyses.

  In addition, the positive opinion by the European Medicines Agency Committee for Orphan Medical Products recommending WVE-210201 for designation as an orphan medicinal product for the treatment of DMD was adopted by the European Commission.

- **DMD exon 53 targeting program**

  Wave is leveraging learnings from its ongoing DMD development and discovery efforts to advance its program to address DMD in boys amenable to exon 53 skipping, including recent data presented at the Project Parent Muscular Dystrophy (PPMD) Annual Conference. The company will present new data from its exon 53 skipping program at the 23rd International Annual Congress of the World Muscle Society in October 2018 and expects to deliver a clinical data readout for this program in 2020.

- **PRECSION-HD Phase 1b/2a clinical trials**

  The PRECISION-HD program, which consists of two global Phase 1b/2a clinical trials evaluating investigational therapies WVE-120101 and WVE-120102 for patients with Huntington's disease, continues to enroll patients at sites in the United States, Europe and Canada, and the company is on track to report topline data in the first half of 2019.

- **Promising *in vivo* data presented for ongoing and planned programs in DMD, ALS and FTD**

  At the PPMD Annual Conference on June 29, 2018, the company presented *in vivo* data demonstrating that its murine-specific stereopure oligonucleotide restored 70% to 90% of natural dystrophin in a dystrophin deficient mouse model for DMD (*mdx*23) with substantial protein expression in the heart and diaphragm. Wave also provided an update on its clinical and discovery programs in DMD at the 2018 New Directions in Biology and Disease of Skeletal Muscle Conference.

  In the last three months, Wave has presented *in vivo* preclinical study results for WVE-3972-01, the company's investigational stereopure antisense oligonucleotide designed to target the pathogenic allele of the *C9ORF72* gene for the treatment of amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD). Animal data for WVE-3972-01 demonstrating potent, sustained and preferential knockdown of toxic biomarkers associated with ALS and FTD were presented at the Alzheimer's Association International Conference, RNA Therapeutics Conference 2018: From Base Pairs to Bedside and the 5th International Conference on Molecular Neurodegeneration. The company intends to initiate clinical trials of WVE-3972-01 in ALS and FTD in the fourth quarter of 2018.

- **Continued strengthening of intellectual property position**

  Wave's advances with its core chemistry and stereochemistry platform continue to strengthen its intellectual property position relating to the design, synthesis and manufacture of stereopure nucleic acid therapeutic candidates, including the allowance of sequence-independent composition-of-matter claims in the United States.

Second Quarter 2018 Financial Results and Financial Guidance

Wave reported a net loss of $35.9 million in the second quarter of 2018 as compared to $24.6 million in the same period in 2017. The increase in net loss in the second quarter of 2018 was largely driven by increased research and development efforts and the continued growth of employee headcount to support Wave's programs.

Research and development expenses were $32.5 million in the second quarter of 2018 as compared to $19.1 million in the same period in 2017. The increase in research and development expenses in the second quarter of 2018 was largely driven by increases in research, preclinical and clinical investments, further expansion of our manufacturing capabilities and facility-related expenses, along with the continued growth of employee headcount to support Wave's programs.

General and administrative expenses were $8.9 million in the second quarter of 2018 as compared to $6.7 million in the same period in 2017. The increase in general and administrative expenses in the second quarter of 2018 was mainly driven by the increase in Wave's employee headcount, as well as increases in professional service expenses and other general operating expenses.

Wave ended the second quarter of 2018 with $241.4 million in cash and cash equivalents as compared to $142.5 million as of December 31, 2017. The increase in cash and cash equivalents was primarily the result of the $170.0 million of cash received from Takeda, which was partially offset by Wave's net loss of $71.1 million.

Wave expects that its existing cash and cash equivalents, together with expected and committed cash from existing collaborations, will enable it to fund its operating and capital expenditure requirements to the end of 2020.

About Wave Life Sciences

Wave Life Sciences is a biotechnology company focused on delivering transformational therapies for patients with serious, genetically-defined diseases. Its chemistry platform enables the creation of highly specific, well characterized oligonucleotides designed to deliver superior efficacy and safety across multiple therapeutic modalities. The company's pipeline is initially focused on neurological disorders and extends across several other therapeutic areas. For more information, please visit www.wavelifesciences.com.

Forward-Looking Statements

This press release contains forward-looking statements concerning our goals, beliefs, expectations, strategies, objectives and plans, and other statements that are not necessarily based on historical facts, including statements regarding the following, among others: the anticipated commencement, patient enrollment, data readouts and completion of our clinical trials; the protocol, design and endpoints of our ongoing and planned clinical trials; the future performance and results of our programs in clinical trials; the progress and potential benefits of our collaborations with partners; the potential of our *in vitro* and *in vivo* preclinical data to predict the behavior of our compounds in humans in clinical trials; our identification of future candidates and their therapeutic potential; the anticipated therapeutic benefits of our potential therapies compared to others; our advancing of therapies across multiple modalities and the anticipated benefits of that model; the anticipated benefits of our manufacturing process and our internal manufacturing facility; our future growth; the potential benefits of our stereopure compounds compared with stereorandom compounds, our drug discovery platform and nucleic acid therapeutics generally; the strength of our intellectual property; and the anticipated duration of our cash runway. Actual results may differ materially from those indicated by these forward-looking statements as a result of various important factors, including the following: our ability to finance our drug discovery and development efforts and to raise additional capital when needed; the ability of our preclinical programs to produce data sufficient to support our clinical trial applications and the timing thereof; our ability to continue to build and maintain the company infrastructure and personnel needed to achieve our goals; the clinical results of our programs, which may not support further development of product candidates; actions of regulatory agencies, which may affect the initiation, timing and progress of clinical trials; our effectiveness in managing future clinical trials and regulatory processes; the success of our platform in identifying viable candidates; the continued development and acceptance of nucleic acid therapeutics as a class of drugs; our ability to demonstrate the therapeutic benefits of our candidates in clinical trials, including our ability to develop candidates across multiple therapeutic modalities; our dependence on third parties, including our collaborators and partners; our ability to manufacture drug material to support our programs and growth; our ability to obtain, maintain and protect intellectual property; our ability to enforce our patents against infringers and defend our patent portfolio against challenges from third parties; and competition from others developing therapies for similar uses, as well as the information under the caption "Risk Factors" contained in our most recent Annual Report on Form 10-K filed with the Securities and Exchange Commission (SEC) and in other filings we make with the SEC from time to time. We undertake no obligation to update the information contained in this press release to reflect subsequently occurring events or circumstances.

WAVE LIFE SCIENCES LTD.
UNAUDITED CONSOLIDATED BALANCE SHEETS

(In thousands, except share amounts)

	June 30, 2018	December 31, 2017
Assets		
Current assets:		
Cash and cash equivalents	$ 241,431	$ 142,503
Current portion of accounts receivable	15,000	1,000
Prepaid expenses and other current assets	11,243	6,985
Total current assets	267,674	150,488
Long-term assets:		
Accounts receivable, net of current portion	50,000	—
Property and equipment, net	32,384	27,334
Restricted cash	3,616	3,610
Other assets	69	411
Total long-term assets	86,069	31,355
Total assets	$ 353,743	$ 181,843
Liabilities, Series A preferred shares and shareholders' equity		
Current liabilities:		
Accounts payable	$ 7,151	$ 7,598
Accrued expenses and other current liabilities	8,494	8,898
Current portion of capital lease obligation	—	16
Current portion of deferred rent	80	60
Current portion of deferred revenue	27,294	1,275
Current portion of lease incentive obligation	762	344
Total current liabilities	43,781	18,191
Long-term liabilities:		
Deferred rent, net of current portion	4,864	4,214
Deferred revenue, net of current portion	149,921	7,241
Lease incentive obligation, net of current portion	6,474	3,094
Other liabilities	1,533	1,619
Total long-term liabilities	162,792	16,168
Total liabilities	$ 206,573	$ 34,359
Series A preferred shares, no par value; 3,901,348 shares issued and outstanding at June 30, 2018 and December 31, 2017	$ 7,874	$ 7,874
Shareholders' equity:		
Ordinary shares, no par value; 29,293,350 and 27,829,079 shares issued and outstanding at June 30, 2018 and December 31, 2017, respectively	$ 373,151	$ 310,038
Additional paid-in capital	30,147	22,172
Accumulated other comprehensive income	201	116
Accumulated deficit	(264,203)	(192,716)
Total shareholders' equity	$ 139,296	$ 139,610
Total liabilities, Series A preferred shares and shareholders' equity	$ 353,743	$ 181,843

WAVE LIFE SCIENCES LTD.
UNAUDITED CONSOLIDATED STATEMENTS OF OPERATIONS AND COMPREHENSIVE LOSS

(In thousands, except share and per share amounts)

	Three Months Ended June 30,		Six Months Ended June 30,	
	2018	2017	2018	2017
Revenue	$ 4,879	$ 1,097	$ 6,301	$ 1,480
Operating expenses:				
Research and development	32,547	19,103	61,743	33,843
General and administrative	8,905	6,667	16,906	12,517
Total operating expenses	41,452	25,770	78,649	46,360
Loss from operations	(36,573)	(24,673)	(72,348)	(44,880)
Other income (expense), net:				
Dividend income	934	482	1,290	772
Interest income (expense), net	4	1	11	4
Other income (expense), net	(259)	(64)	84	(136)
Total other income (expense), net	679	419	1,385	640
Loss before income taxes	(35,894)	(24,254)	(70,963)	(44,240)
Income tax provision	—	(343)	(172)	(1,453)
Net loss	$ (35,894)	$ (24,597)	$ (71,135)	$ (45,693)
Net loss per share attributable to ordinary shareholders—basic and diluted	$ (1.23)	$ (0.91)	$ (2.49)	$ (1.81)
Weighted-average ordinary shares used in computing net loss per share attributable to ordinary shareholders—basic and diluted	29,144,466	26,899,058	28,535,149	25,224,725
Other comprehensive income (loss):				
Foreign currency translation	$ 36	$ 3	$ 85	$ 18
Comprehensive loss	$ (35,858)	$ (24,594)	$ (71,050)	$ (45,675)

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Exhibit 99.2

Wave Life Sciences

Corporate Presentation

August 9, 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.

Focused on delivering transformational therapies for patients with serious, genetically defined diseases

- Rationally designed stereopure nucleic acid therapeutics
- Platform company utilizing multiple modalities including antisense, exon skipping and RNAi
- 6 neurology development programs by the end of 2018
- Robust R&D platform, ability to partner additional therapeutic areas
- Expertise and core focus in neurology
  - Huntington's disease: Two Phase 1b/2a trials ongoing
  - Duchenne muscular dystrophy: Exon 51 Phase 1 trial ongoing
  - Amyotrophic lateral sclerosis and Frontotemporal dementia for C90rf72: Trials expect to initiate Q4 2018
  - Key data readouts anticipated in 2019 for first 3 programs

WAVE™
LIFE SCIENCES

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+
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

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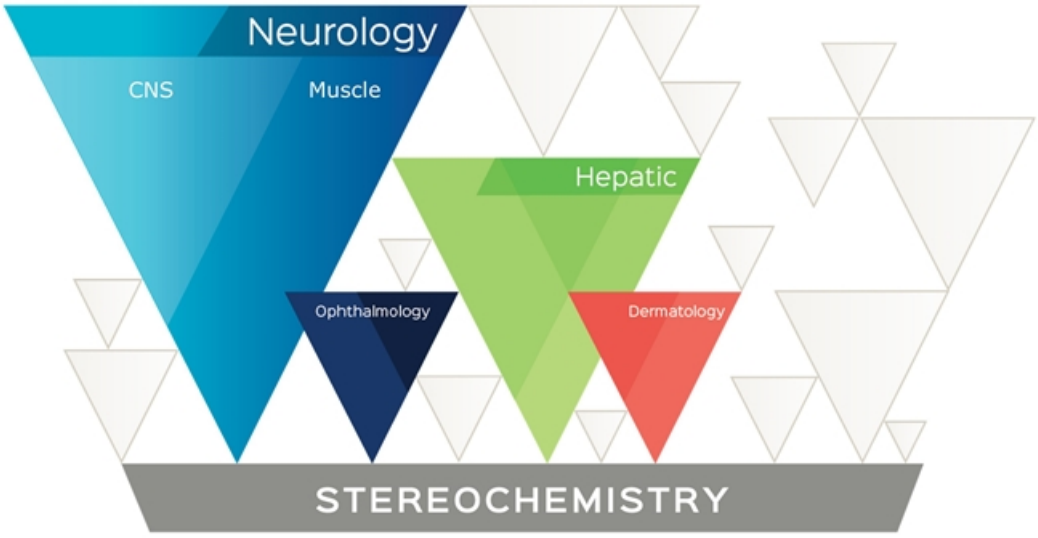
*Estimates of U.S. prevalence and addressable population by target based on publicly available data and are approximate; for Huntington's disease, numbers approximate manifest and pre-manifest populations, respectively.
† During a four-year term, Wave and Takeda may collaborate on up to six preclinical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

Broad platform relevance across therapeutic areas



WAVE LIFE SCIENCES

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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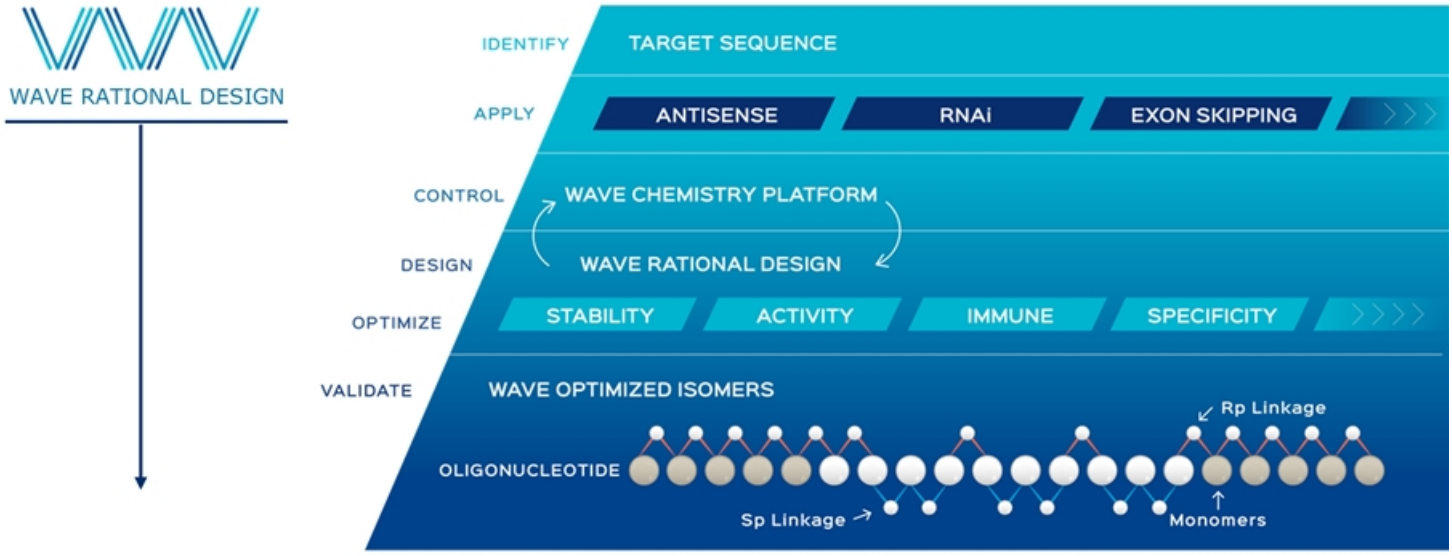
Creating a new class of oligonucleotides



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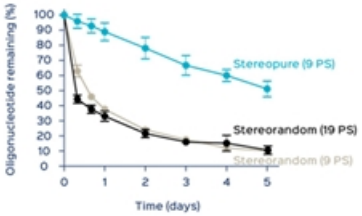
Source: Iwamoto N, et al. Control of phosphorothioate stereochemistry substantially increases the efficacy of antisense oligonucleotides. Nature Biotechnology. 2017.

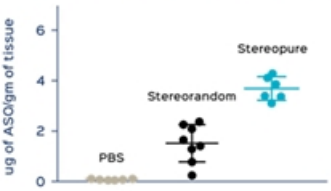
Chemistry may optimize medicines across multiple dimensions

Improved Stability

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

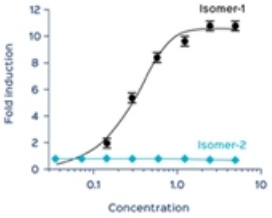


Oligonucleotide exposure (spinal cord)

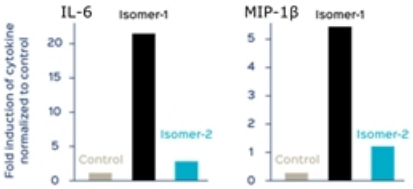


Controlled Immunogenicity

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

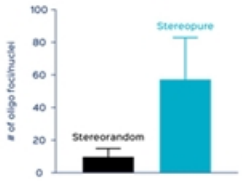


Cytokine induction in human PBMC assay

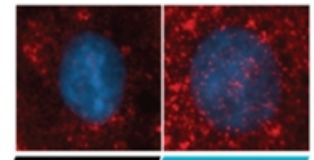


Enhanced Delivery

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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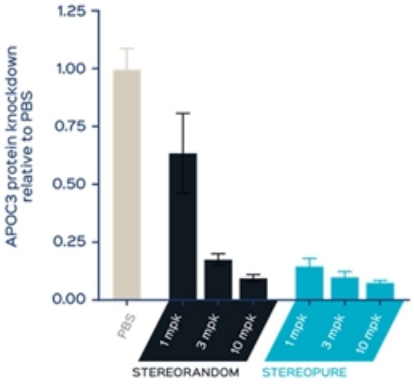
Data represented in this slide from in vitro studies. Experimental conditions: Human TLR9 assay – Source: Ohto U, et al. Structural basis of CpG and inhibitory DNA recognition by Toll-like receptor 9, Nature 520, 702-705, 2015. Intracellular trafficking assay – Cells were washed and fixed and oligos 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

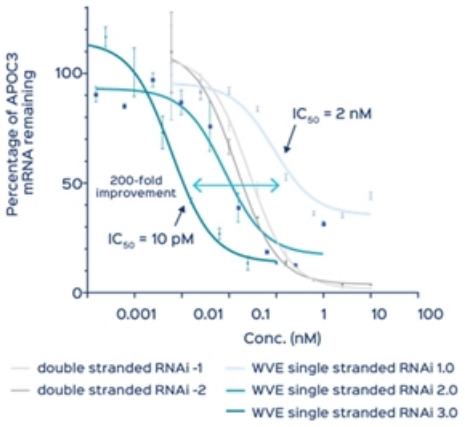
Antisense

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



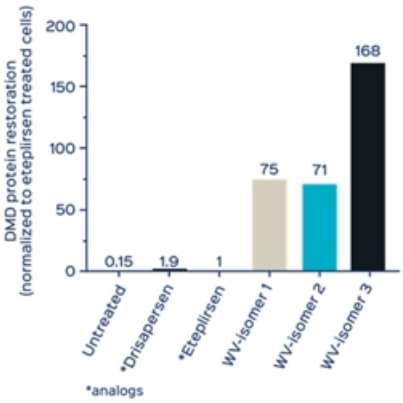
RNAi

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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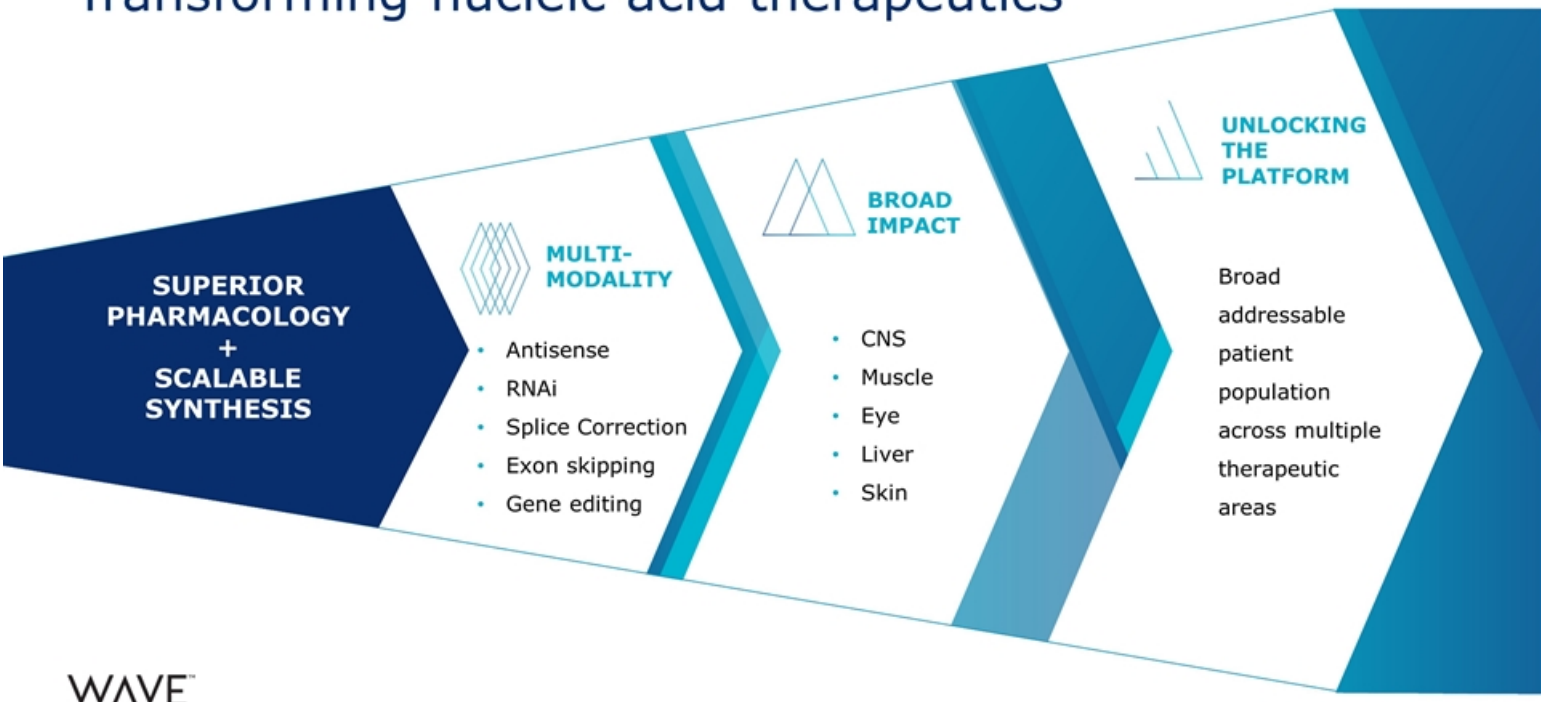
Transforming nucleic acid therapeutics



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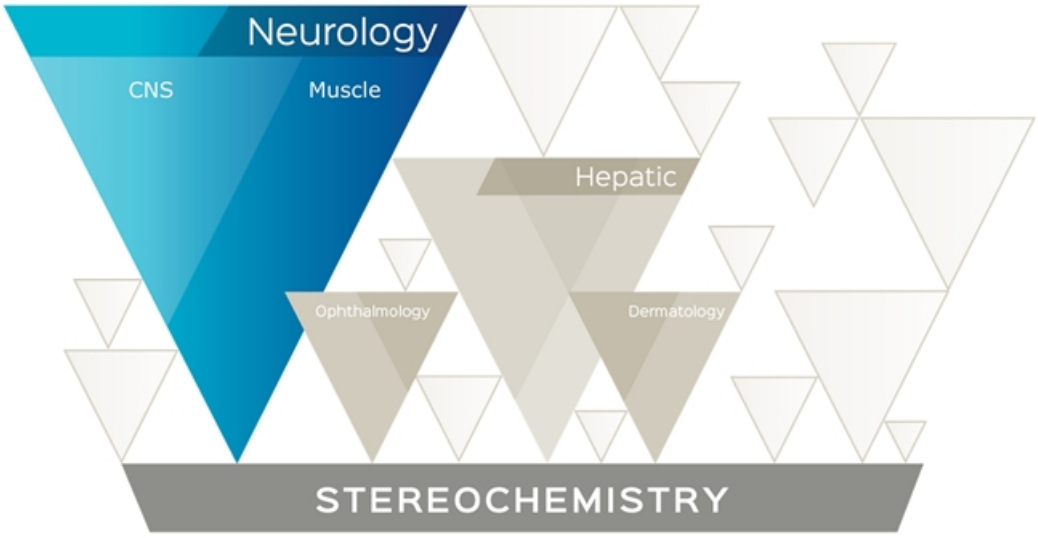
Neurology



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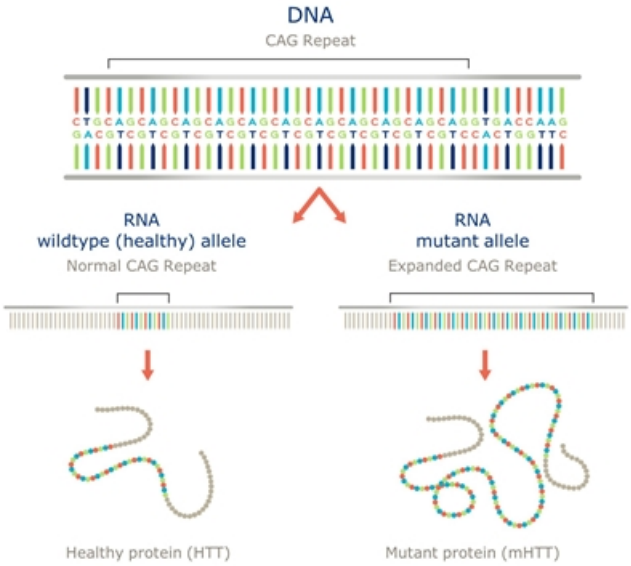
Huntington's Disease

Huntington's Disease: a hereditary, fatal disorder

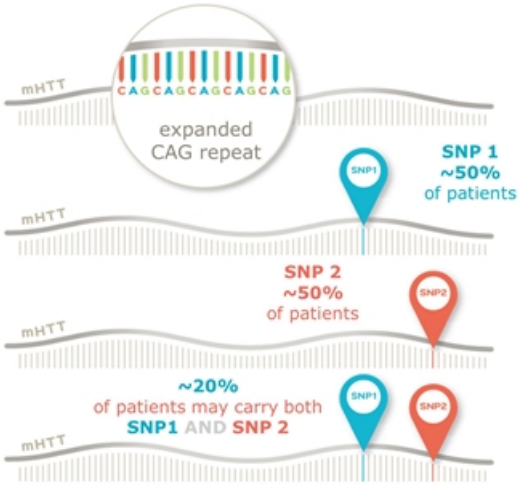
- Autosomal dominant disease, characterized by cognitive decline, psychiatric illness and chorea; fatal
- No approved disease-modifying therapies
- Expanded CAG triplet repeat in HTT gene results in production of mutant huntingtin protein (mHTT); accumulation of mHTT causes progressive loss of neurons 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



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)



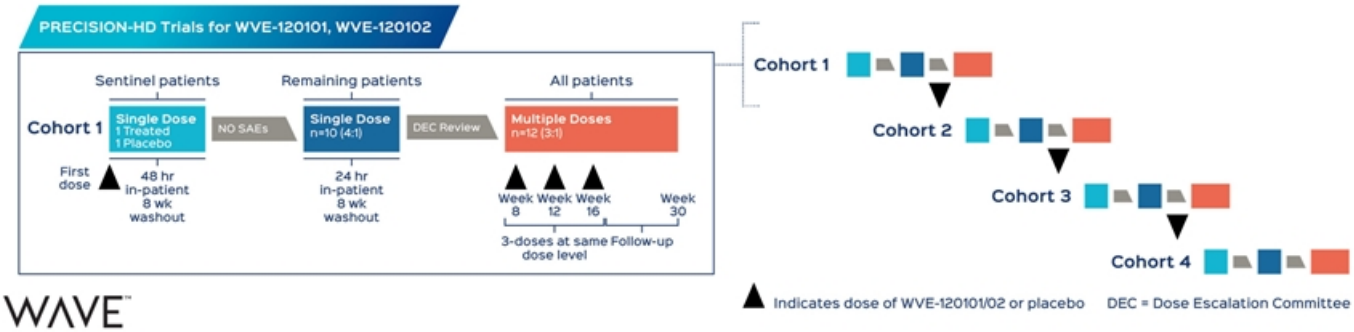
Total: Due to overlap, an estimated **~70%** of the total HD patient population carry SNP 1 and/or SNP 2

WAVE LIFE SCIENCES

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
- 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 H1 2019



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Mutant huntingtin: a powerful, novel biomarker

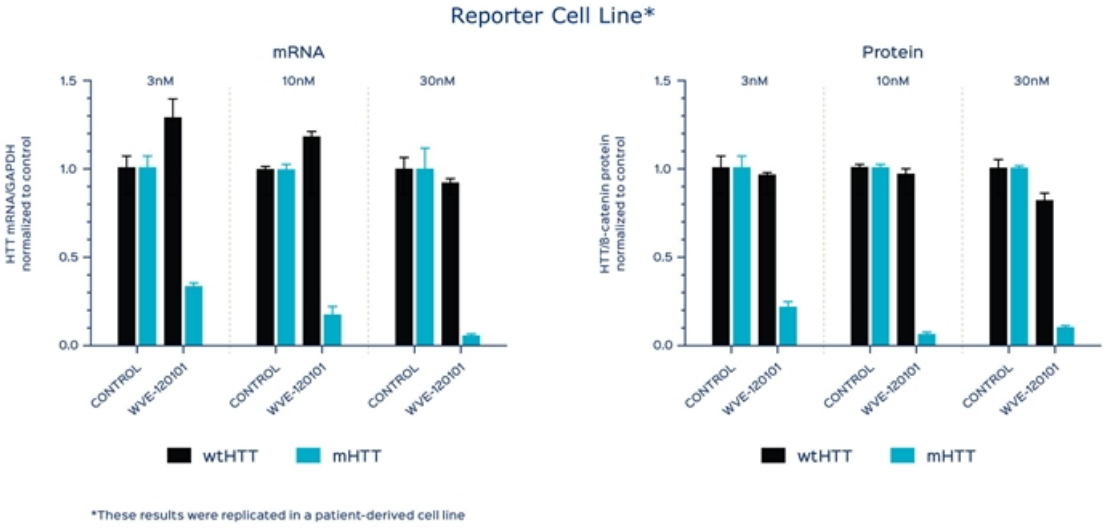
- Novel immunoassay allows for quantification of mutant huntingtin, the cause of HD
- Level of mHTT detected is associated with time to onset, increased with disease progression, and predicts diminished cognitive and motor dysfunction
- Assay currently being utilized in clinical studies

Novel approach enables precise measurement of target engagement and effect

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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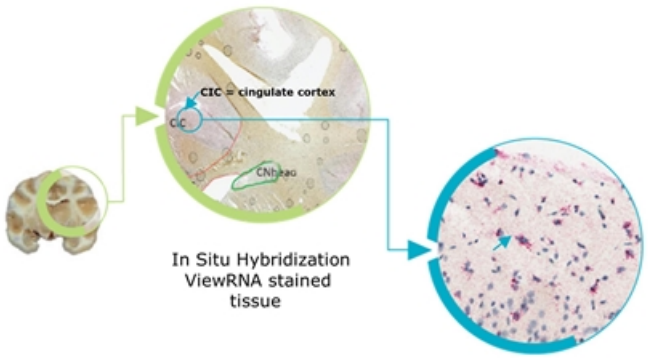
Demonstrated delivery to brain tissue

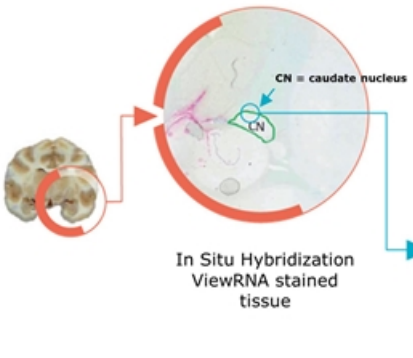
- WVE-120101 and WVE-120102 distribution in cynomolgus non-human primate brain following intrathecal bolus injection



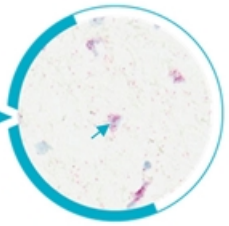
In Situ Hybridization ViewRNA stained tissue

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 5,000 newborn boys each year; 20,000 new cases annually worldwide

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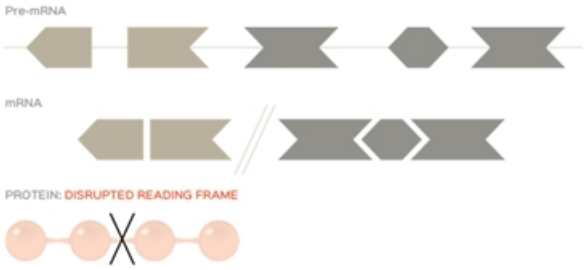
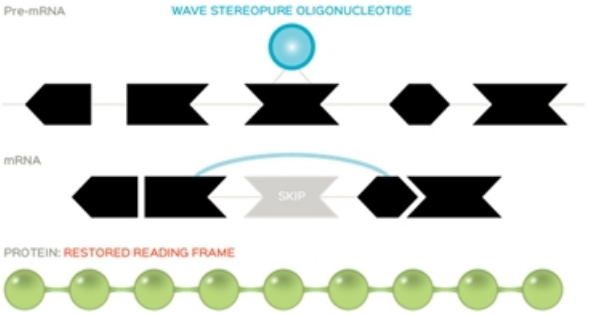
Wave approach:
meaningful restoration of dystrophin production through exon skipping

- 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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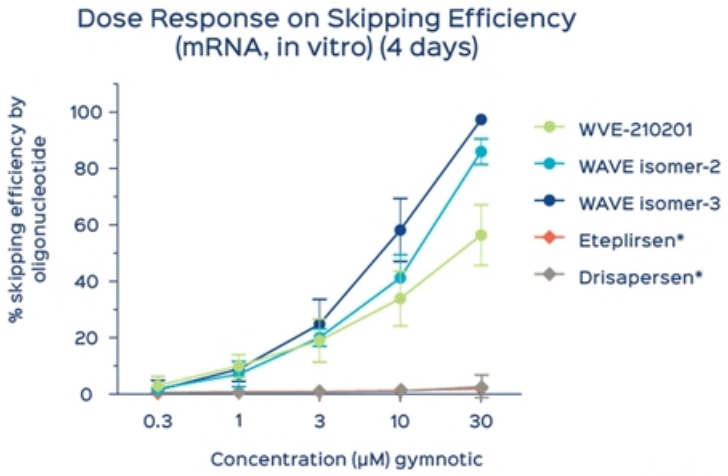
Exon 51: WVE-210201 clinical program

- 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



*analogs

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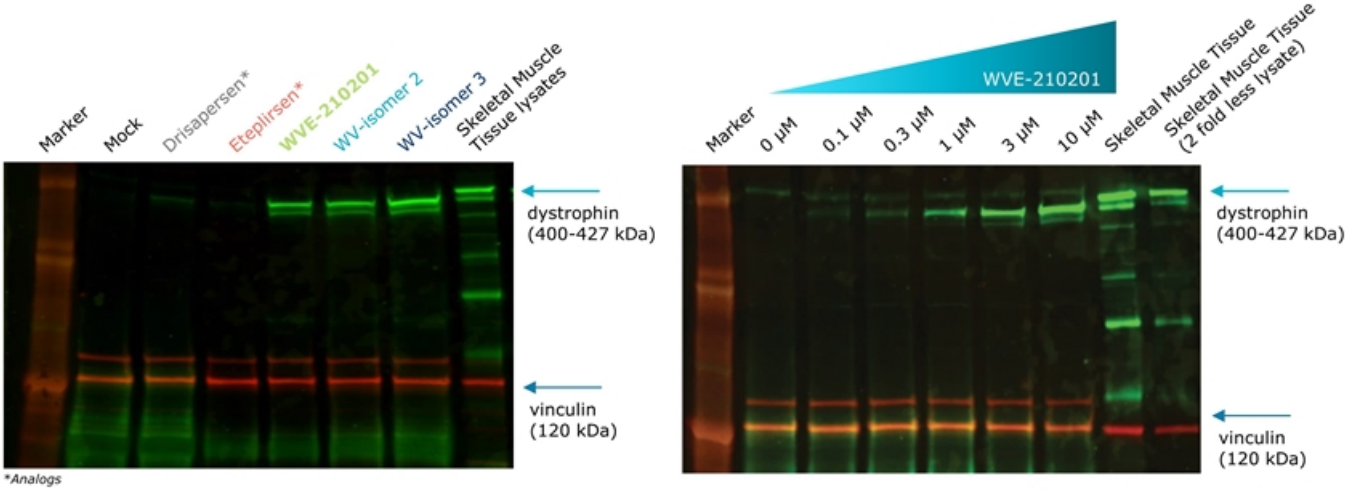
Exon 51: increased dystrophin restoration



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*

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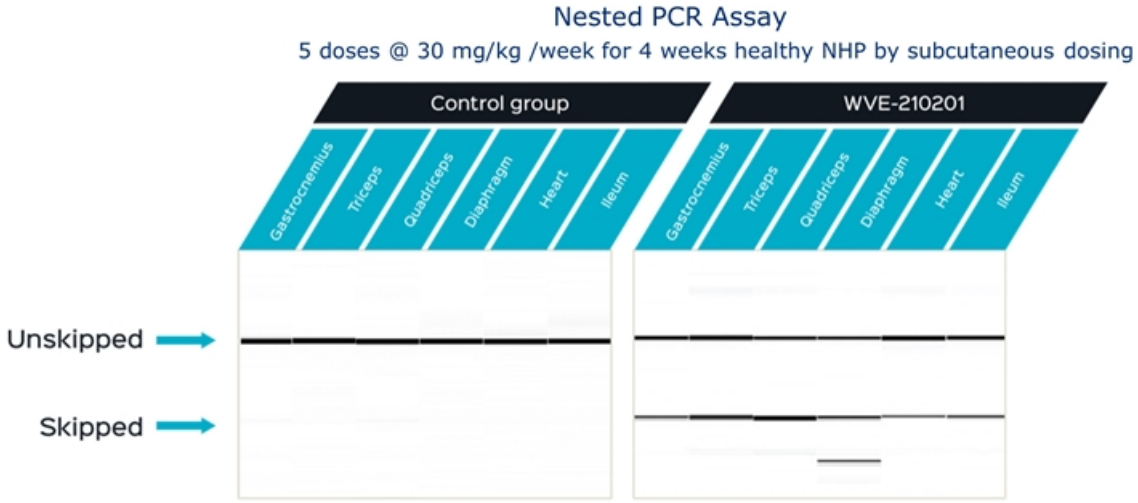
Experimental conditions: DMD protein restoration by Western Blot in patient-derived myotubes with clear dose effect. Free uptake at 10uM concentration of each compound with no transfection agent

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



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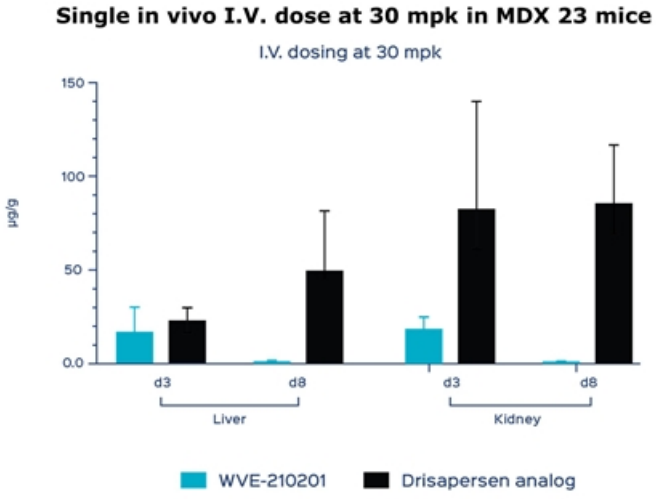
Experimental conditions: Muscle tissues were collected 2 days after the last dose and fresh frozen. Total RNAs were extracted with phenol/chloroform and converted to cDNA using high capacity kit. Nested PCR assay was performed and analyzed by fragment analyzer.

Exon 51: no apparent tissue accumulation observed

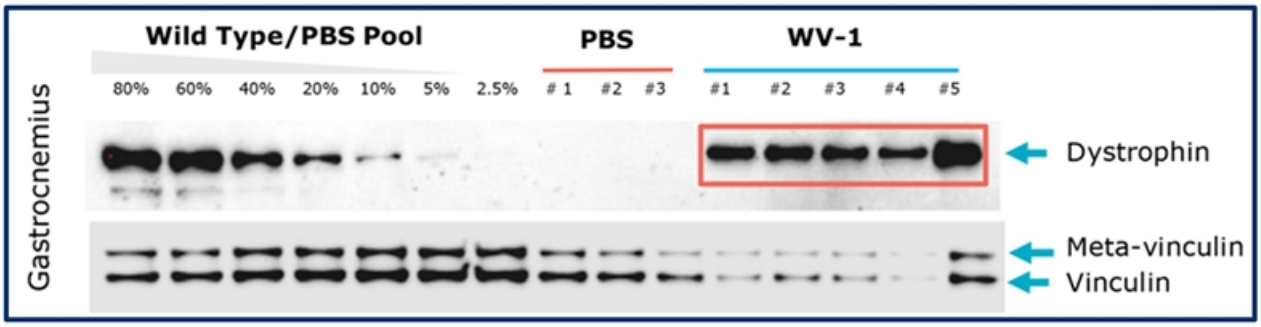
- Standard oligonucleotides tend to accumulate in liver and kidney
- Wave rationally designed oligonucleotides optimized to allow compound to clear more effectively
- WVE-210201 demonstrated wide tissue distribution in dose dependent fashion
- No apparent accumulation observed after multiple doses



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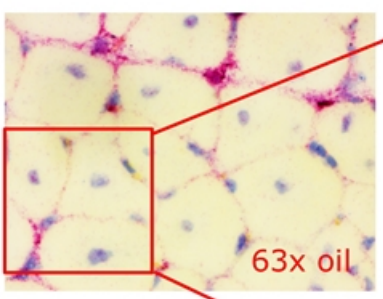
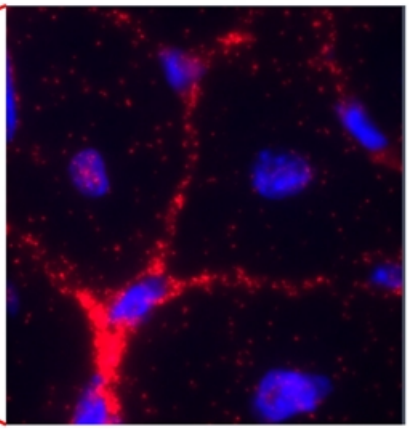
*Numbers indicate individual animals
Methods: *mdx* 23 mice received 4 weekly IV doses (150 mg/kg). Tissues collected 96 hours post final dose. Protein expression determined by western blot.

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



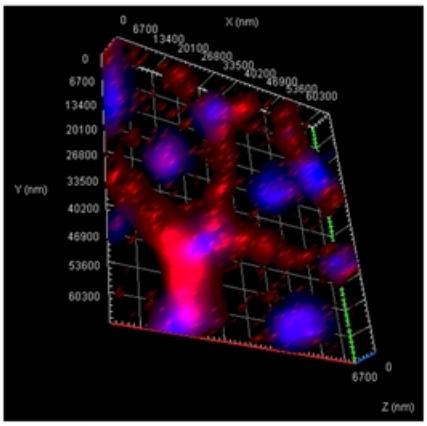
Bright field view

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



Fluorescence channel view

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



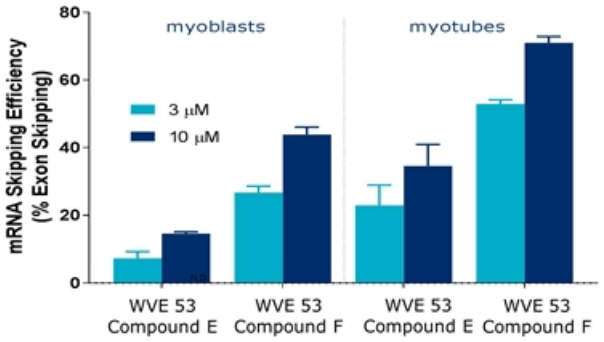
Z Stack view

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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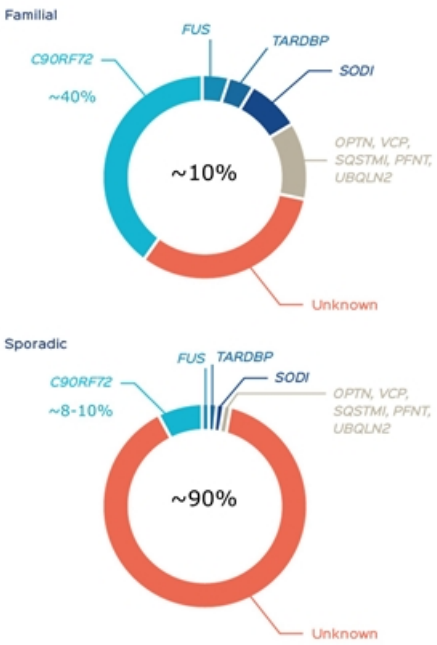
Amyotrophic lateral sclerosis

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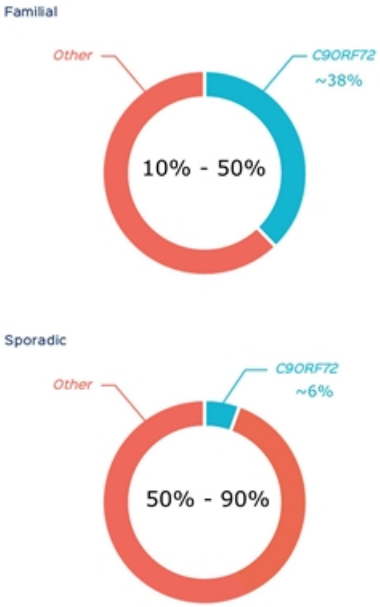
Source: State of play in amyotrophic lateral sclerosis genetics Alan E Renton, Adriano Chiò & Bryan J. Traynor Nature Neuroscience 17, 17–23 (2014) doi:10.1038/nn.3584

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

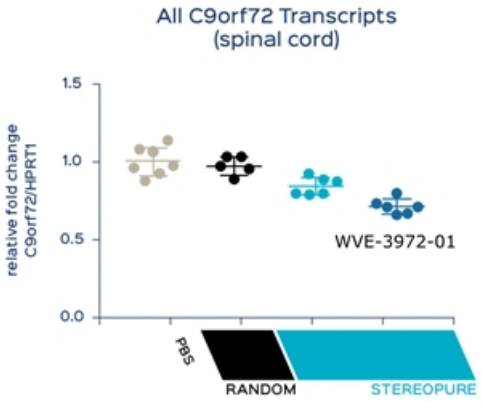


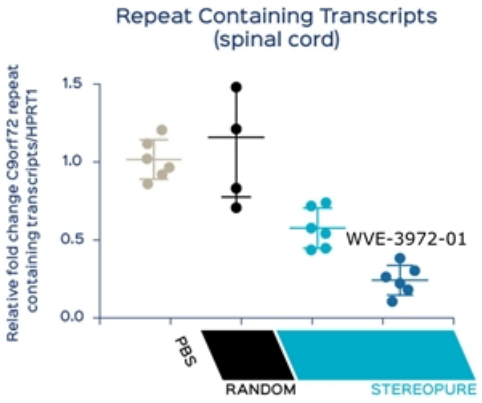
WAVE LIFE SCIENCES

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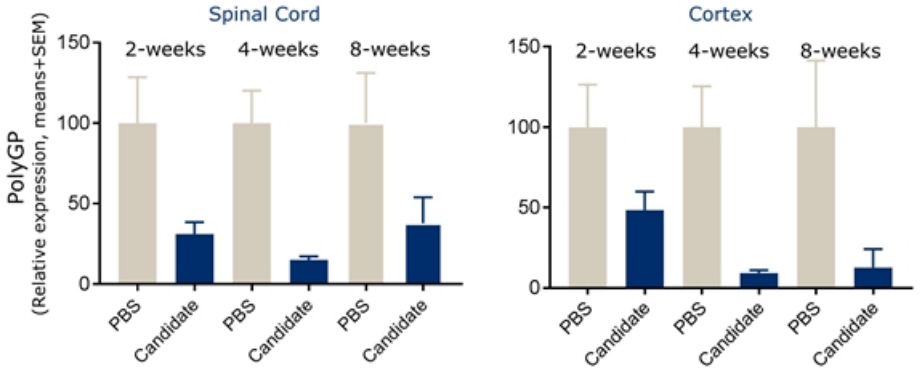
Experimental description: Samples were analyzed using quantitative PCR (Taqman assay)

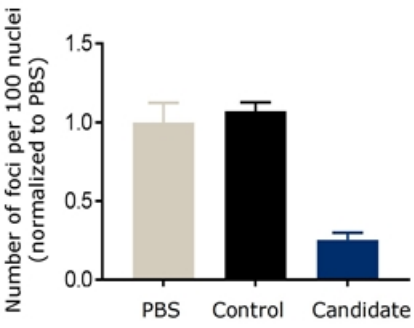
Durable reduction of dipeptides and RNA foci in vivo

- 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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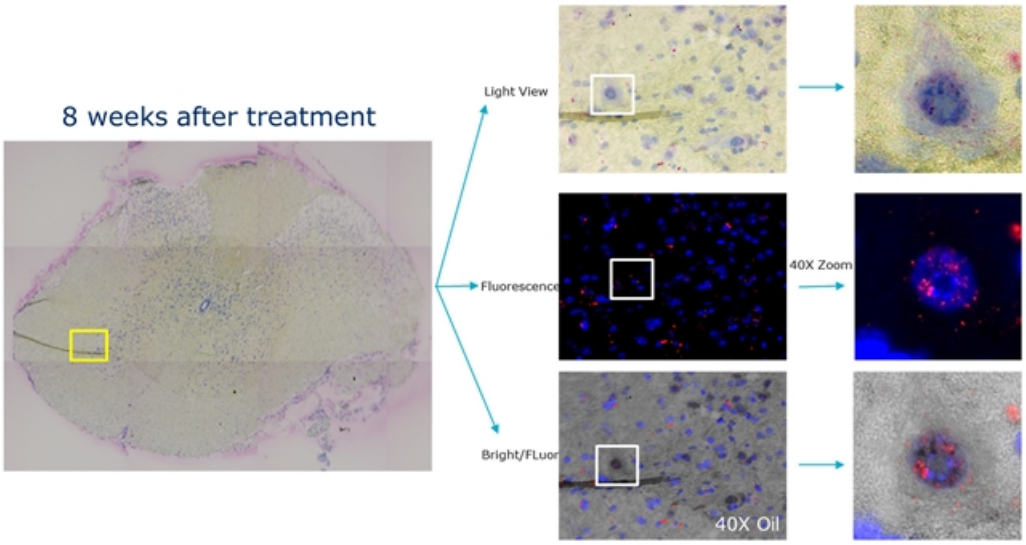
In vivo distribution of WVE-3972-01



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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Spinocerebellar ataxia type 3

Spinocerebellar ataxia type 3

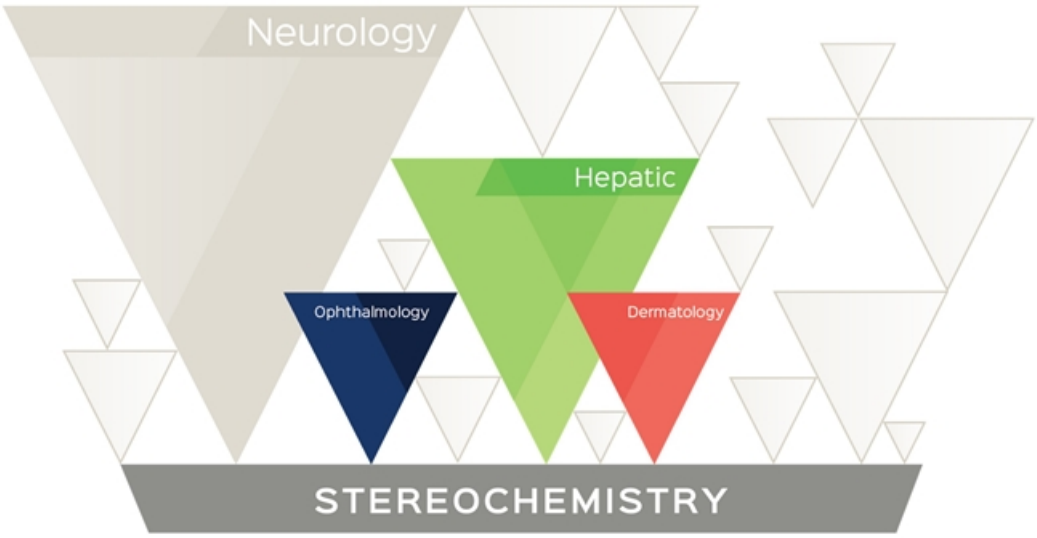
- Also known as Machado-Joseph disease
- Rare, hereditary, progressive neurodegenerative disorder that results in a lack of muscle control and coordination in upper and lower extremities; gradually leads to paralysis and loss of ability to speak or swallow
- Life expectancy is 10-20 years from symptom onset
- Prevalence: 1-2 in 100,000 people; most common dominantly inherited form of ataxia, representing 20% to 50% of all SCAs
- Expanded CAG repeat in *ATXN3* gene results in mutant ATXN3 protein that causes widespread neuronal loss in brain and spinal cord

Candidate targeting *ATXN3* expected to be named by YE 2018

WAVE LIFE SCIENCES

Source: Paulson H. Machado-Joseph disease/spinocerebellar ataxia type 3. Handb Clin Neurol 103, 437—449 (2012).
National Institute of Health. Spinocerebellar ataxia 3. Accessed at: https://ghr.nlm.nih.gov/condition/spinocerebellar-ataxia-type-3 on February 15, 2018

Emerging areas



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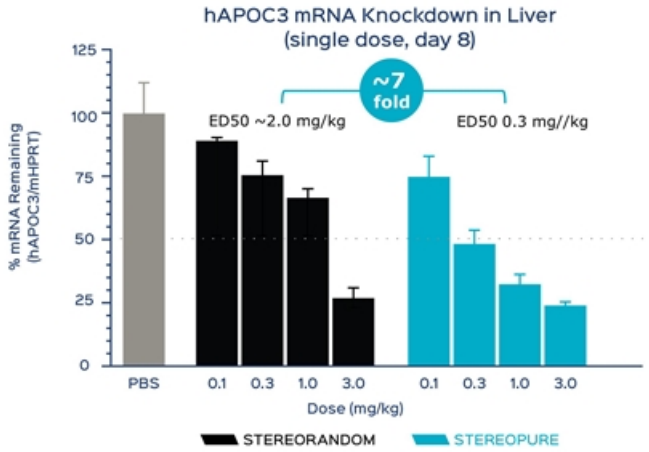
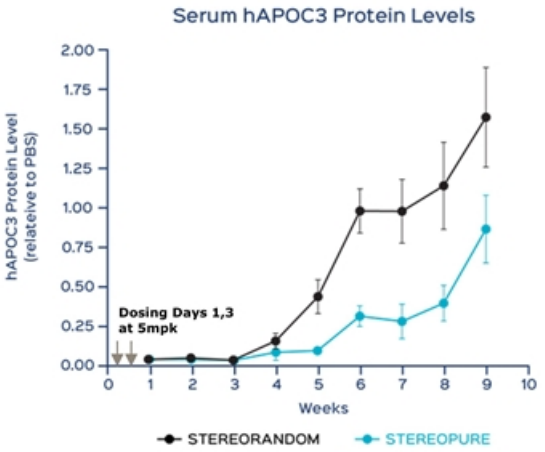
Stereopure oligonucleotides: improved in vivo potency, extended duration

- 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 oligonucleotide





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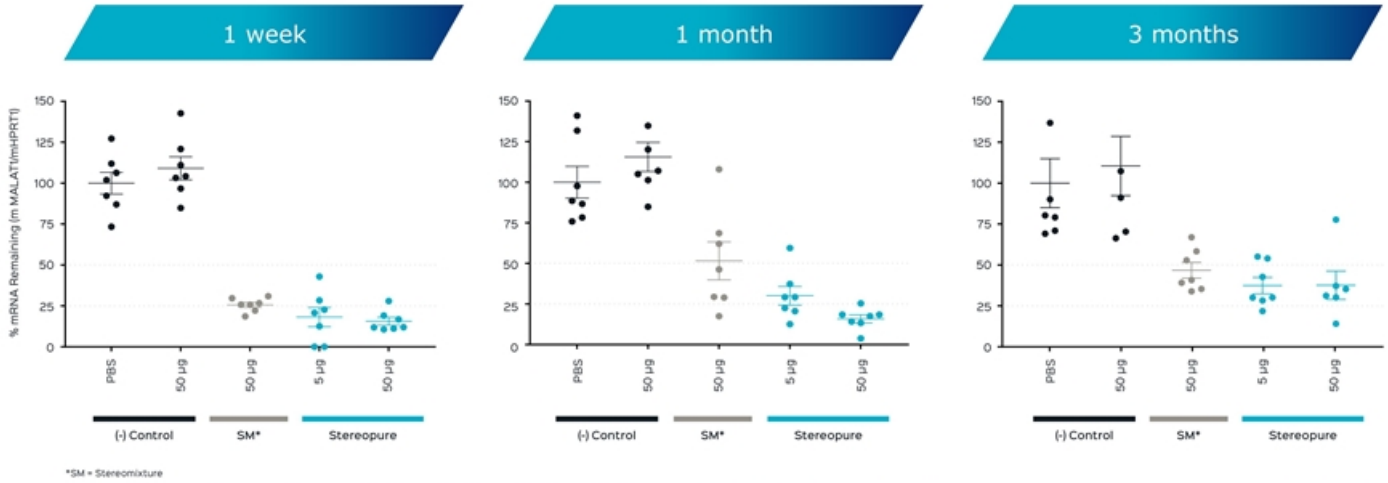
Experimental description: Male human APOC3 transgenic mice were dosed with APOC3 ASOs with indicated doses. APOC3 mRNA quantification in the liver was performed using Taqman assay specific for hAPOC3. For protein analysis, plasma samples were collected weekly and analyzed by ELISA assay specific to human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

Improved in vivo potency, extended duration

Back of the eye



*SM = Stereomixture

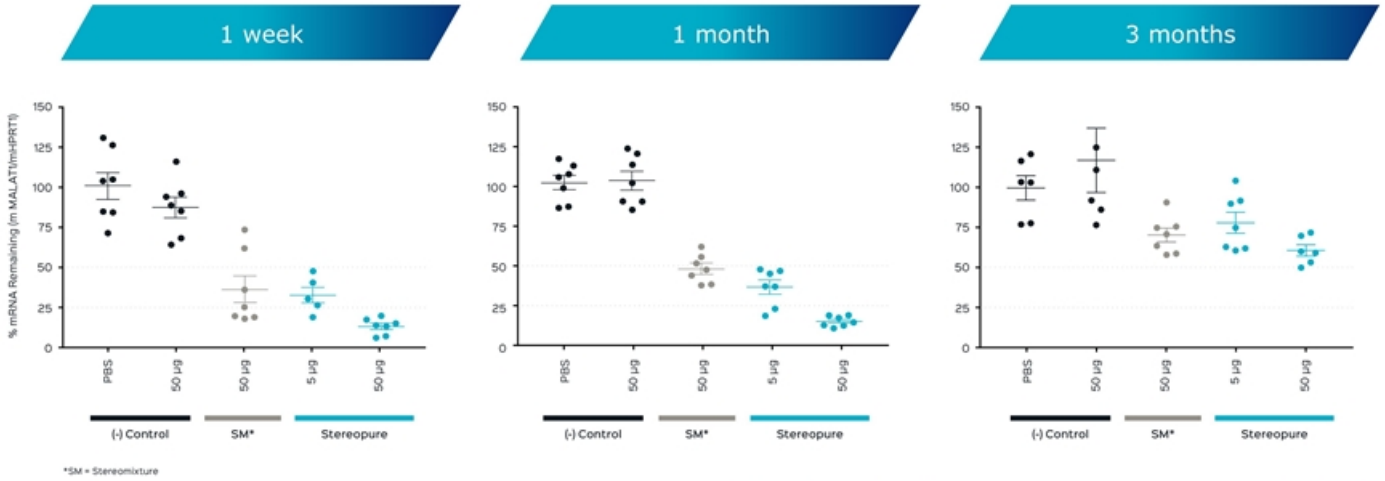
10X lower dose of stereopure oligonucleotide is more potent than stereorandom oligonucleotide

WAVE LIFE SCIENCES

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

Improved in vivo potency, extended duration

Front of the eye



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

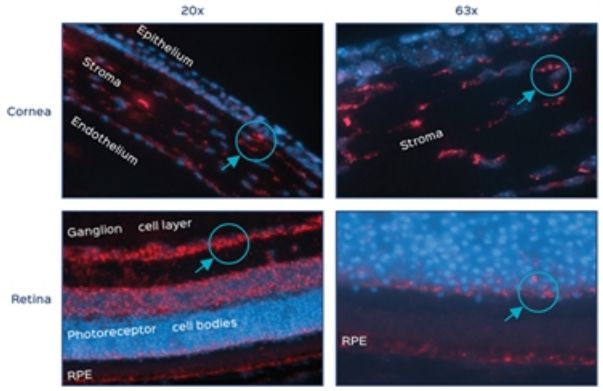
WAVE LIFE SCIENCES

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

Distribution and target engagement

Ophthalmology

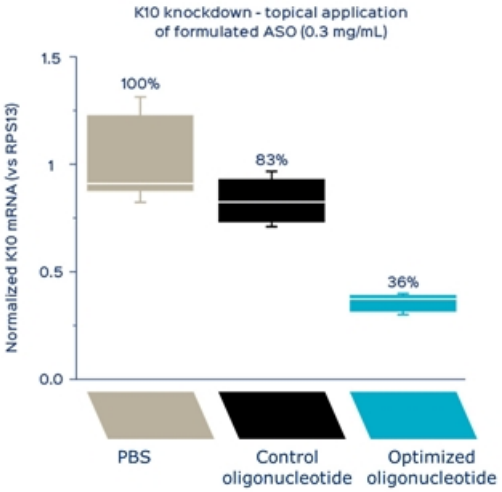
In vivo distribution of oligonucleotide to key cellular compartments following intravitreal injection in murine eye



Red dots = Oligonucleotides

Dermatology

Target engagement following topical administration on human skin explant model



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Partnerships

CNS collaboration with Takeda

Committed capital

$230+ million in committed cash; eligible for milestones and royalties in excess of $2 billion*

- Expected to fund Wave operations to end of 2020, through multiple data readouts

Significant value in 50:50 profit share

Takeda option on global 50:50 share of CNS programs in HD, ALS, FTD and SCA3

- After opt-in, Takeda to pay 50% of development costs
- Wave will lead manufacturing and joint clinical development; participate in joint co-commercialization in the US

Fully funded R&D activities in CNS

Takeda right to license additional preclinical CNS targets over four years

- Wave CNS R&D fully funded
- Includes potential milestones and royalties in large CNS disorders such as Alzheimer's and Parkinson's diseases

WAVE LIFE SCIENCES

* 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.

Hepatic collaboration with Pfizer

- Initiated May 2016
- Exploring targets across modalities, including ASO and ssRNAi
- Up to 5 hepatic-metabolic programs
  - 5 targets declared; APOC3 and 4 undisclosed
- 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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Enabling technologies: Applying artificial intelligence to discover novel therapies for genetic neuromuscular disorders

deep genomics

- Deep Genomics is a world leader in artificial intelligence with a platform that combines automation, advanced biomedical knowledge, high volume data acquisition and machine learning
- Wave is collaborating with Deep Genomics to predict the impact of genetic mutations and oligonucleotide approaches to splicing
- The goal is to identify new targets and optimal regions or sequences within those targets to be addressed by Wave's rationally designed oligonucleotides

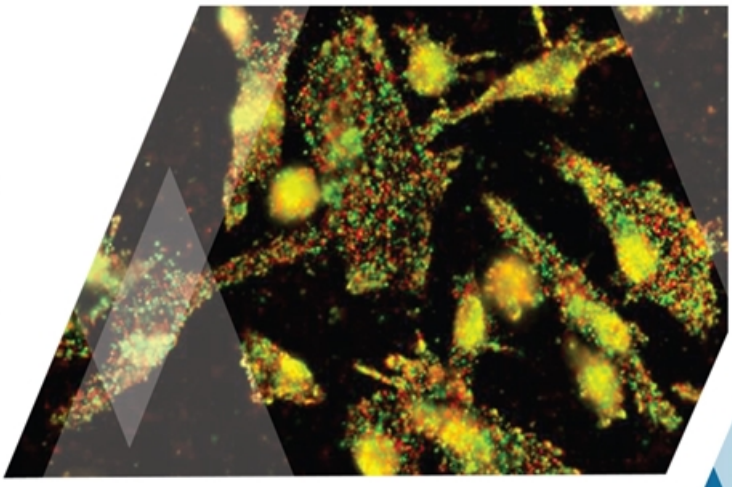
Understanding splicing biology to illuminate new approaches to increase size of addressable patient populations

WAVE LIFE SCIENCES

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
- Imaging allows for target engagement assessment in specific regions, cell types and subcellular compartments of the brain
- Provides meaningful insight into disease state, treatment effect of oligonucleotides and outcomes at the molecular and cellular level



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Manufacturing strength: 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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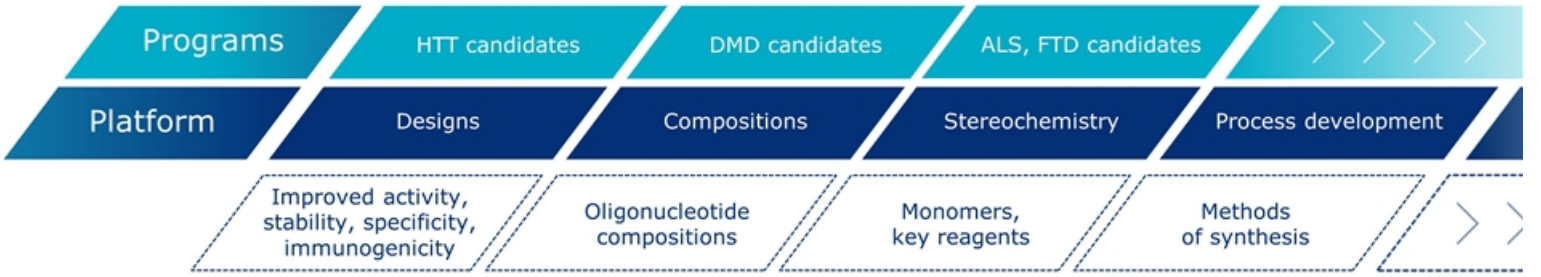
Intellectual property strength: breadth and depth of patent portfolio



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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 with potential to be best-in-class
  - Received EU orphan drug 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 U.S. orphan drug designation
- **H2 2019: Interim dystrophin readout from ongoing open label extension and planned efficacy trials expected for WVE-210201**
- **2020: DMD Exon 53 Program clinical data readout expected**

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Realizing the potential of nucleic acid therapeutics