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Wave Life Sciences

Fourth Quarter and Full Year 2021 Earnings

March 3, 2022

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.

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Paul Bolno, MD, MBA

President and CEO

Today’s agenda

1. **Opening remarks -** Paul Bolno, MD, MBA, President and CEO
2. **Clinical pipeline -** Michael Panzara, MD, MPH, CMO, Head Therapeutics Disc. and Dev.
3. **AATD AIMer program update –** Paloma Giangrande, PhD, VP Biology and Platform Disc.
4. **4Q 2021 financial results –** Kyle Moran, CFO
5. **2022 outlook -** Paul Bolno, MD, MBA, President and CEO
6. **Q&A**

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Wave Life Sciences today

Building a leading genetic medicines company

PRISM

Innovative RNA therapeutics platform

Stereochemistry, PN chemistry | Silencing, splicing, or editing oligonucleotides | Strong and broad IP[1]

Clinical neurology pipeline: ALS/FTD, DMD, HD

- Genetic diseases with high unmet need
- Silencing CNS: ALS, FTD, HD
- Splicing Muscle: DMD

RNA editing with targeted GalNAc delivery: AATD

- Potential first-in-class AIMers for genetic hepatic diseases
- AATD is ideally-suited for an RNA base editing approach

Partnership opportunities to capitalize on assets

- Proprietary PN chemistry modifications improve potency duration, durability of effect of multiple therapeutic modalities
- Internal GMP oligonucleotide manufacturing facility

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ALS: Amyotrophic lateral sclerosis; FTD: Frontotemporal dementia; HD: Huntington's disease; DMD: Duchenne muscular dystrophy; AATD: Alpha-1 antitrypsin deficiency; [1]stereopure oligonucleotides and novel backbone chemistry modifications; AIMers: RNA editing oligonucleotides

Wave's PN chemistry highlighted in two Nucleic Acids Research (NAR) manuscripts in 2022 thus far

Nucleic Acids Research, 2022 1
https://doi.org/10.1093/nar/gkac018

Control of backbone chemistry and chirality boost oligonucleotide splice switching activity

Pachamuthu Kandasamy[1,†], Graham McClorey[2,†], Mamoru Shimizu[1], Rowshon Alam[1], Naoki Iwamoto[1], Jayakanthan Kumarasamy[1], Gopa Adam Bezigian[1], Onanong Chivatakarn[1], David C. D. Butler[1], Michae Katarzyna Chwalenia[2], Kay E. Davies[4], Jigar Desai[1], Julil Dilip She Ruth Ellerington[2], Ben Edwards[4], Jack Godfrey[1], Andrew Hoss[1], Far Kenneth Longo[1,3], Genliang Lu[1], Subramanian Marappan[1], Jacopo C Erin Purcell Estabrook[1], Chikdu Shivalila[1], Maeve Tischbein[1], Tomoi Carlo Rinaldi[2,3], Joana Rajão-Saraiva[4], Snehlata Tripathi[1], Hailin Ya Xiansi Zhao[1], Cong Zhou[1], Jason Zhang[1], Luciano Apponi[1], Matthew Chandra Vargeese[1,*]

[1]Wave Life Sciences, Cambridge, MA, USA, [2]Department of Paediatrics, University of C Oxford OX1 3QX, UK, [3]MDUK Oxford Neuromuscular Centre, University of Oxford, Oxfo [4]Department of Physiology, Anatomy and Genetics, University of Oxford, South Parks R

Received September 01, 2021; Revised December 18, 2021; Editorial Decision January 04, 2022; Accepted J

ABSTRACT

Although recent regulatory approval of splice-switching oligonucleotides (SSOs) for the treatment of neuromuscular disease such as Duchenne muscular dystrophy has been an advance for the splice-switching field, current SSO chemistries have shown limited clinical benefit due to poor pharmacology. To overcome limitations of existing technologies, we engineered chimeric stereopure oligonucleotides with phosphorothioate (PS) and phosphoryl guanidine-containing (PN) backbones. We demonstrate that these chimeric stereopure oligonucleotides have markedly improved pharmacology and efficacy compared with PS-modified oligonucleotides, preventing premature death and improving median survival from 49 days to at least 280 days in a dystrophic mouse model with an aggressive phenotype. These data demonstrate that chemical optimization alone can profoundly impact oligonucleotide pharmacology and highlight the potential for continued innovation around the oligonucleotide backbone. More specifically, we conclude that chimeric stereopure oligonucleotides are a promising splice-switching modality with potential for the treatment of neuro-

muscular and other gene
cult to reach tissues such
heart.

INTRODUCTION

Exon-skipping oligonucleoti
technology, are designed to
base pairing to pre-mRNA,
to bypass exon(s) to restore
and protein production fo
The recent FDA approval
pholino oligomers (PMOs)
muscular dystrophy has
for the exon-skipping field
PMOs, they have demon
modifying benefit. Becaus
dystrophin protein and have
//www.fda.gov/news-events/
grants-accelerated-approval
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dystrophy-mutation; ht
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continues to be substanti

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†The authors wish it to be known that, in their opinion, the first two authors should be regarded as Joint First Authors.

Nucleic Acids Research, 2022 1
https://doi.org/10.1093/nar/gkac037

NAR Breakthrough Article

Impact of guanidine-containing backbone linkages on stereopure antisense oligonucleotides in the CNS

Pachamuthu Kandasamy[†], Yuanjing Liu[†], Vincent Aduda, Sandheep Akare, Rowshon Alam, Amy Andreucci, David Boulay, Keith Bowman, Michael Byrne, Megan Cannon, Onanong Chivatakarn, Julil Dilip Sheike, Naoki Iwamoto, Tomomi Kawamoto, Jayakanthan Kumarasamy, Sarah Lamore, Muriel Lemaitre, Xuena Lin, Kenneth Longo, Richard Looby, Subramanian Marappan, Jake Metterville, Susovan Mohapatra, Bridget Newman, Ik-Hyeon Paik, Saurabh Patil, Erin Purcell-Estabrook, Mamoru Shimizu, Pochi Shum, Stephany Standley, Kris Taborn, Snehlata Tripathi, Hailin Yang, Yuan Yin, Xiansi Zhao, Elena Dale and Chandra Vargeese[*]

Wave Life Sciences, Cambridge, MA 02138, USA

Received June 30, 2021; Revised December 17, 2021; Editorial Decision January 10, 2022; Accepted January 13, 2022

ABSTRACT

Attaining sufficient tissue exposure at the site of action to achieve the desired pharmacodynamic effect on a target is an important determinant for any drug discovery program, and this can be particularly challenging for oligonucleotides in deep tissues of the CNS. Herein, we report the synthesis and impact of stereopure phosphoryl guanidine-containing backbone linkages (PN linkages) to oligonucleotides acting through an RNase H-mediated mechanism, using *Malat1* and *C9orf72* as benchmarks. We found that the incorporation of various types of PN linkages to a stereopure oligonucleotide backbone can increase potency of silencing in cultured neurons under free-uptake conditions 10-fold compared with similarly modified stereopure phosphorothioate (PS) and phosphodiester (PO)-based molecules. One of these backbone types, called PN-1, also yielded profound silencing benefits throughout the mouse brain and spinal cord at low doses, improving both the potency and durability of response, especially in difficult to reach brain tissues. Given these benefits in preclinical models, the incorporation of PN linkages into stereopure oligonucleotides with chimeric backbone modifications has the potential to render regions of the brain beyond the spinal cord more accessible to oligonucleotides and, consequently, may also expand the scope of neurological indications amenable to oligonucleotide therapeutics.

INTRODUCTION

Antisense oligonucleotides can be designed to promote degradation of a targeted transcript through an RNase H-dependent mechanism. RNase H binds a heteroduplex formed between a DNA-containing oligonucleotide and the targeted RNA and cleaves the RNA (1). Unmodified antisense oligonucleotides have poor pharmacokinetic (PK) properties, so chemical modifications are needed to enable their use in preclinical research and as therapeutics. Phosphorothioate (PS) modification of the phosphodiester (PO) backbone is one of the most common backbone modifications used in research and to improve the properties of oligonucleotide therapeutics (1,2). A consequence of PS modification is the creation of a chiral center, where the molecule can adopt either an *Rp* or *Sp* configuration (1,3). We have previously demonstrated that PS-modified stereopure oligonucleotides—with precisely controlled chirality of inter-nucleotide PS backbone linkages—can outperform stereorandom oligonucleotides—those racemic mixtures derived from traditional chemistries for which the chiral configuration of the backbone is not controlled (4–6). New backbone modifications and methods to control their stereochemistry continue to emerge (3,4,7–9).

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†The authors wish it to be known that, in their opinion, the first two authors should be regarded as Joint First Authors.

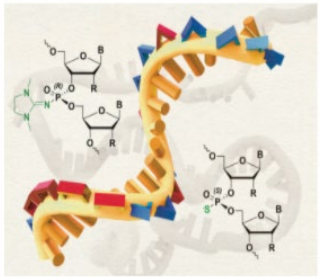
NAR press alert:

…"these data are the strongest evidence to date for the value of stereopure linkages in oligonucleotides designed to reduce gene expression…"

…Wave has "something unique, the data shows clear advantages of stereopure vs stereorandom P linkages. The duration of gene silencing achieved is impressive…"

…"work offers an encouraging suggestion that novel chemical approaches can push the frontiers of oligonucleotide drug development."

Breakthrough Articles



Impact of guanidine-containing backbone linkages on stereopure antisense oligonucleotides in the CNS

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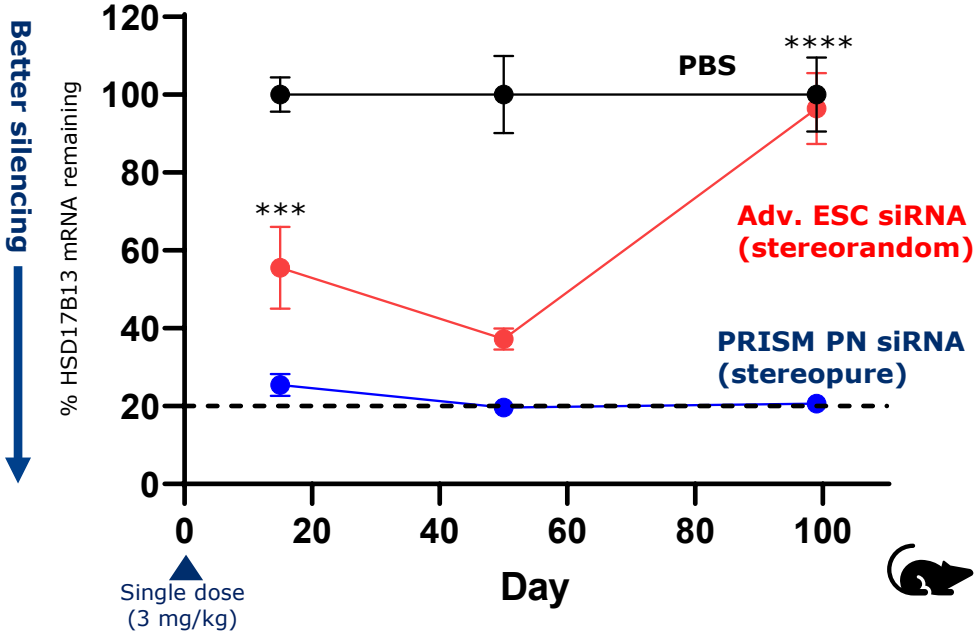
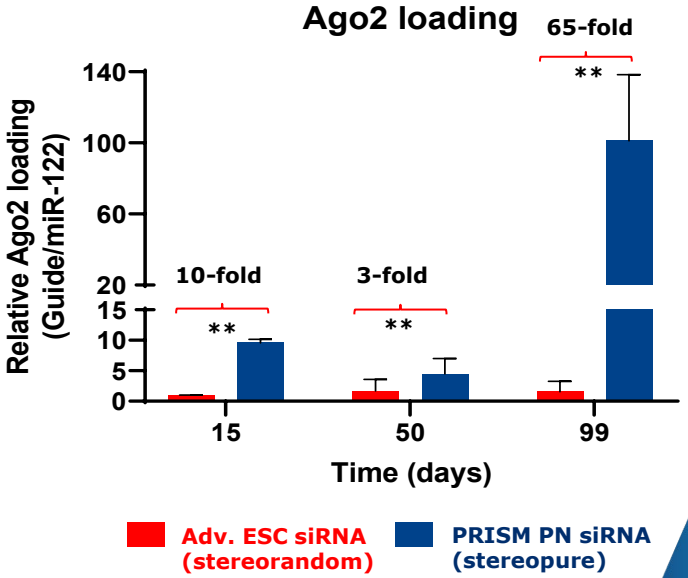
PRISM PN siRNA led to unprecedented silencing as compared to state-of-art >3 months after single dose

~80% silencing HSD17B13 mRNA *in vivo* with GalNAc-conjugated PRISM PN siRNA 14 weeks post single dose



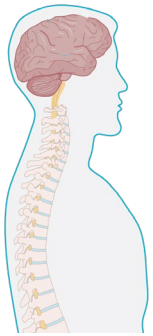
PRISM PN siRNA loaded in RISC is significantly greater than Adv. ESC siRNA



(Left) Proprietary human transgenic mouse model, Post hoc tests derived from Linear Mixed Effects Model with Random subject effects;
(Right) ** P<0.01, 2-way ANOVA

Diversified strategy to rapidly assess the potential of PN candidates across tissue types and modalities

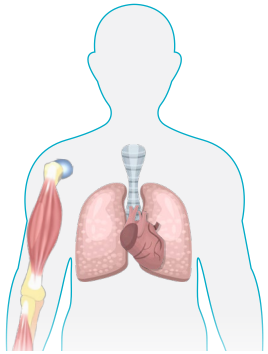
Silencing
CNS



WVE-004
C9 - ALS/FTD

WVE-003
SNP3 - HD

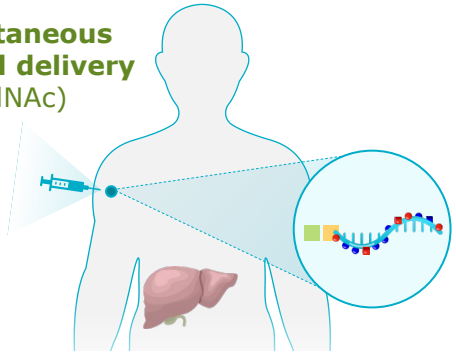
Splicing (exon-skipping)
Muscle



WVE-N531
Exon 53 - DMD

RNA editing (AIMers)
Liver

Subcutaneous targeted delivery
(GalNAc)



AIMer
SERPINA1 - AATD

AIMers are short and stable (fully chemically modified) to enable GalNAc-mediated delivery

Clinical data expected in 2022 for WVE-004, WVE-003, WVE-N531 to provide insight into PN chemistry and enable decision making for each program

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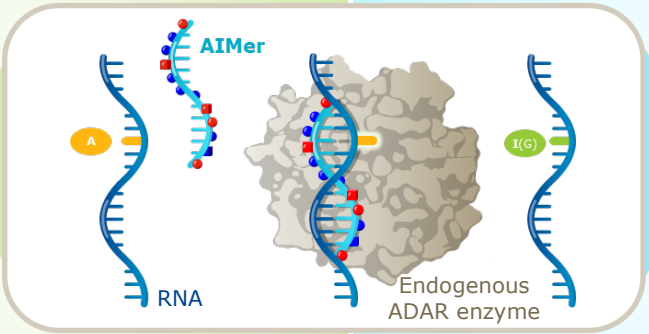
AIMer opportunity: novel RNA editing therapeutics, starting with targeted delivery to hepatocytes

Initial focus on correcting driver mutations of genetic hepatic diseases

Lead program:
Alpha-1 antitrypsin deficiency (AATD)

Restore or correct protein function



Modulate protein interactions with AIMers

- **Upregulate expression**
- **Modify function**
- **Modulate protein-protein interaction**
- **Post-translational modification**
- **Alter folding or processing**

- Cardiometabolic
- Oncology
- Immunology
- Neurological disorders

- Clinically proven GalNAc-mediated delivery
- Potential for first-in-class therapeutics
- **AATD:** ~200K people in US and EU with mutation in *SERPINA1* Z allele (PI*ZZ)

- Large patient populations
- Human Reference Interactome documents >50K protein-protein interactions involving >8K proteins[1]
- >90K Post-translational modifications across ~30K proteins mapped,[2] thousands associated with disease[3]

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A: Adenosine I: Inosine G: Guanosine
[1]Luck, K et al. *Nature* (2020) [2]Prasad, TSK et al. *Nucleic Acids Research* (2009) [3]Huang, K et al. *Nucleic Acids Research* (2016)

Established internal GMP manufacturing for multiple oligonucleotide modalities

Strong technical knowhow and operating expertise

- Experienced team led by Sridhar Vaddeboina, PhD (SVP Chemistry, Manufacturing, Controls)
- Experts in oligonucleotide synthesis (ASOs, DNAs, RNAs, siRNAs)
- Proven track record scaling complex chemistries; delivered clinical supply for six programs at Wave

Established infrastructure

- State of the art facilities (90,000 sq ft) and expansion space
- Process and analytical development labs
- GMP oligonucleotide (API) manufacturing
- Established Quality and GMP systems (QA, supply chain, logistics, QC testing)

Scalable to support Wave's GMP manufacturing needs, as well as potential new partners

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Mike Panzara, MD, MPH

Chief Medical Officer,

Head of Therapeutics

Discovery and Development

PN chemistry enhances potency, tissue distribution and duration of effect of splicing compounds in muscle

Benefits of PN chemistry for exon-skipping

- ✓ Increased exon-skipping activity *in vitro*
- ✓ More stable in cultured myoblasts than PS oligonucleotides
- ✓ Increased muscle exposure in mice and NHPs
- ✓ Increased exon-skipping activity in mice and NHPs
- ✓ Exon-skipping activity well correlated with restored dystrophin expression in mice

Exon 53

WVE-N531

Exon skipping candidate in DMD

Preclinical mouse models

Non-human primate studies

Clinical trial (ongoing)

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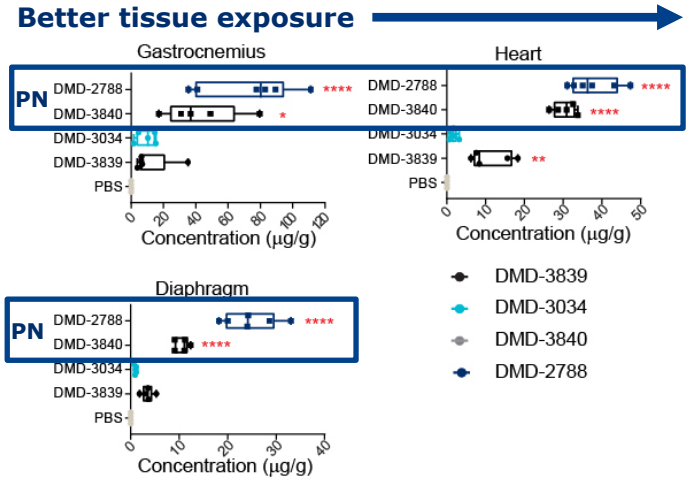
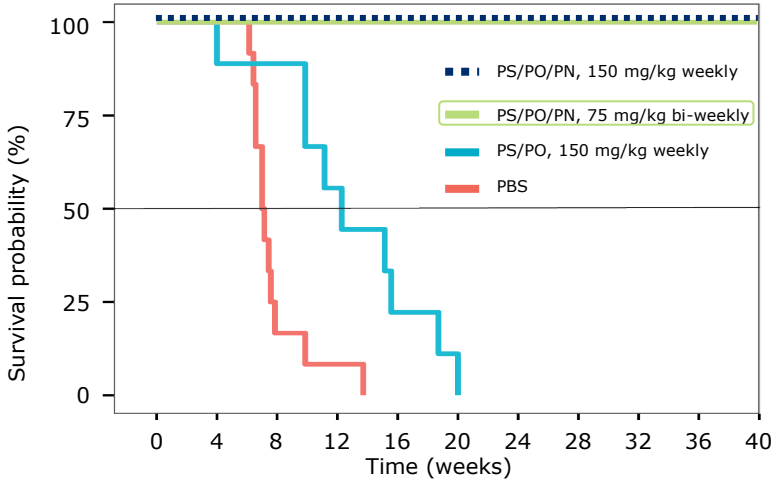
Kandasamy et al., 2022; doi: 10.1093/nar/gkac018,

PN chemistry improved muscle exposure and survival in preclinical mouse models

PN boosted muscle concentrations after single dose, which correlated with exon-skipping activity



Treatment with PN-modified molecules led to 100% survival of dKO mice at time of study termination



Note: Untreated, age-matched mdx mice had 100% survival at study termination [not shown]

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Kandasamy et al., 2022; doi: 10.1093/nar/gkac018

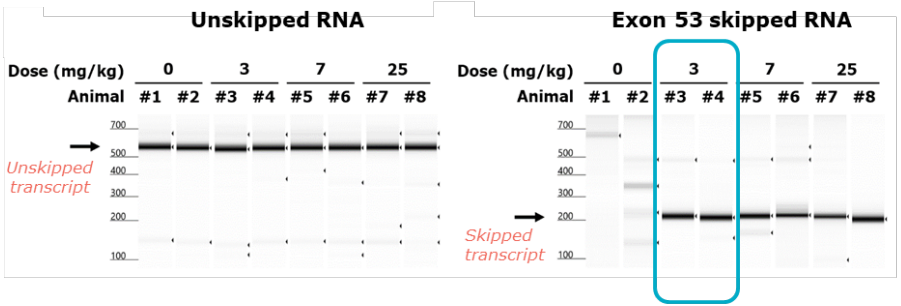
WVE-N531: PN chemistry enhances muscle distribution and exon-skipping in NHPs

Plasma and tissue concentrations of WVE-N531 (PS/PO/PN) significantly higher than suvodirsen (1st-gen PS/PO) in multiple NHP studies

- ✓ Substantially higher muscle concentrations (including heart and diaphragm) as compared to suvodirsen
- ✓ Higher plasma Cmax, AUC and Ctrough

WVE-N531 leads to exon-skipping in NHPs at doses significantly lower than suvodirsen

6 weekly doses of 3 mg/kg



Healthy NHPs have normal levels of dystrophin, but target engagement can be assessed by detection of skipped transcript

Non-human primates (NHPs) received 6 x weekly IV infusions of PBS or 3, 7, or 25 mg WVE-N531 (n=2 per dose); necropsied on day 38. Exon 53 skipping quantified by RT-PCR; W, week; HED: Human equivalent dose

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WVE-N531 plasma concentrations at starting dose significantly improved over suvodirsen

WVE-N531 Phase 1b/2a open-label clinical trial starting dose
Dose escalation is ongoing

	WVE-N531 (PN chemistry) fold increase over suvodirsen at the same dose level
Plasma:	
C_{max}	**~2.5x**
AUC	**~4x**
Muscle:	*Patient muscle biopsies expected in 2022*

Increase in plasma concentrations with single dose

WVE-N531 plasma half-life estimated to be >1 week
(vs. less than 24 hours for suvodirsen)

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WVE-N531 is designed with PN chemistry backbone modifications. Suvodirsen (first-generation Exon 51 candidate) did not include PN chemistry.
NHP: non-human primates; AUC: Area under curve; C_{max}: Maximum plasma concentration

Dose escalation ongoing in clinical trial of WVE-N531

- Open-label clinical trial of boys with DMD amenable to exon 53 skipping
- Dose level and dosing frequency guided by tolerability and plasma PK

Initial cohort

- Ascending intra-patient doses of WVE-N531
- Up to 4 dose levels (administered ≥4 weeks apart) evaluated to select dose level for multidose
- Up to 3 additional doses given every-other-week at selected dose level, followed by muscle biopsy

Cohort expansion to be guided by assessment of muscle biopsies: (drug distribution in muscle and biomarkers)

Possible cohort expansion (up to 15 boys)

- Additional patients enrolled and dosed every other week at selected dose level
- Up to 7 total doses to be given followed by a minimum 8-week safety monitoring period
- Powered to evaluate change in dystrophin expression

Clinical data, including muscle biopsies, expected in 2022

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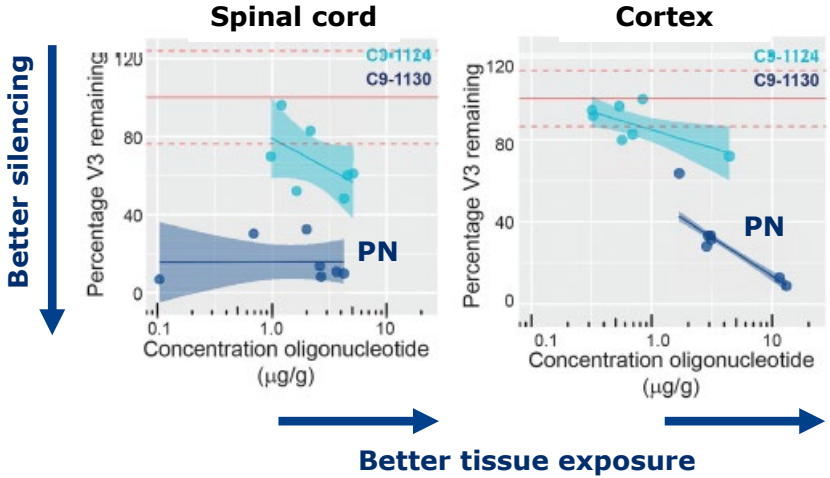
DMD: Duchenne muscular dystrophy

PN chemistry enhances tissue exposure, potency and duration of silencing in CNS of mice models

Benefits of PN chemistry in CNS

- ✓ Increased potency in neurons
- ✓ Well-tolerated in multiple *in vitro* & *in vivo* assays
- ✓ Increased potency & extended durability of silencing in mice
- ✓ Enhanced tissue exposure in mice

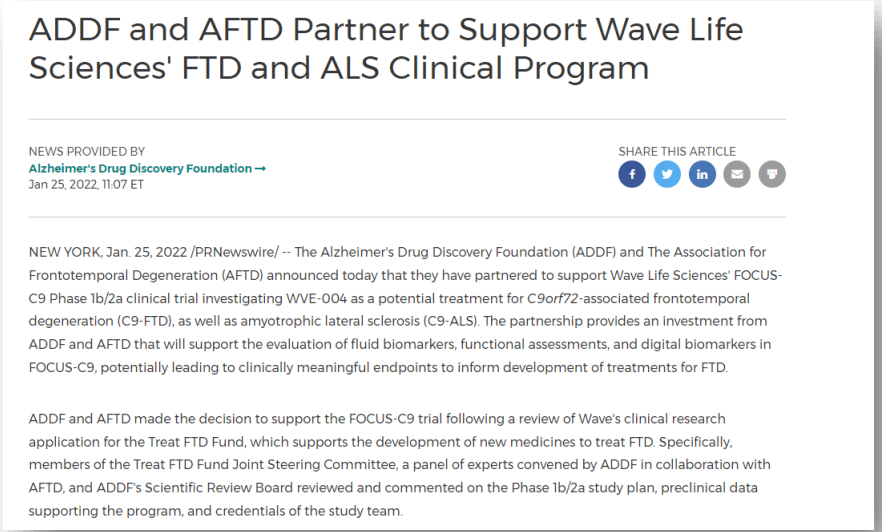


Kandasamy et al., NAR Breakthrough Article, 2022 doi: 10.1093/nar/gkac037

FOCUS-C9 trial recognized as innovative clinical study for patients with C9-FTD, C9-ALS or both

Alzheimer's Drug Discovery Foundation

AFTD The Association for Frontotemporal Degeneration
FIND HELP·SHARE HOPE



ADDF and AFTD Partner to Support Wave Life Sciences' FTD and ALS Clinical Program

NEWS PROVIDED BY
Alzheimer's Drug Discovery Foundation →
Jan 25, 2022, 11:07 ET

SHARE THIS ARTICLE

NEW YORK, Jan. 25, 2022 /PRNewswire/ -- The Alzheimer's Drug Discovery Foundation (ADDF) and The Association for Frontotemporal Degeneration (AFTD) announced today that they have partnered to support Wave Life Sciences' FOCUS-C9 Phase 1b/2a clinical trial investigating WVE-004 as a potential treatment for *C9orf72*-associated frontotemporal degeneration (C9-FTD), as well as amyotrophic lateral sclerosis (C9-ALS). The partnership provides an investment from ADDF and AFTD that will support the evaluation of fluid biomarkers, functional assessments, and digital biomarkers in FOCUS-C9, potentially leading to clinically meaningful endpoints to inform development of treatments for FTD.

ADDF and AFTD made the decision to support the FOCUS-C9 trial following a review of Wave's clinical research application for the Treat FTD Fund, which supports the development of new medicines to treat FTD. Specifically, members of the Treat FTD Fund Joint Steering Committee, a panel of experts convened by ADDF in collaboration with AFTD, and ADDF's Scientific Review Board reviewed and commented on the Phase 1b/2a study plan, preclinical data supporting the program, and credentials of the study team.

- Alzheimer's Drug Discovery Foundation (ADDF) and The Association for Frontotemporal Degeneration (AFTD) partnered to support FOCUS-C9 clinical trial
  - Evaluation of fluid biomarkers
  - Functional assessments
  - Digital biomarkers

FOCUS C9

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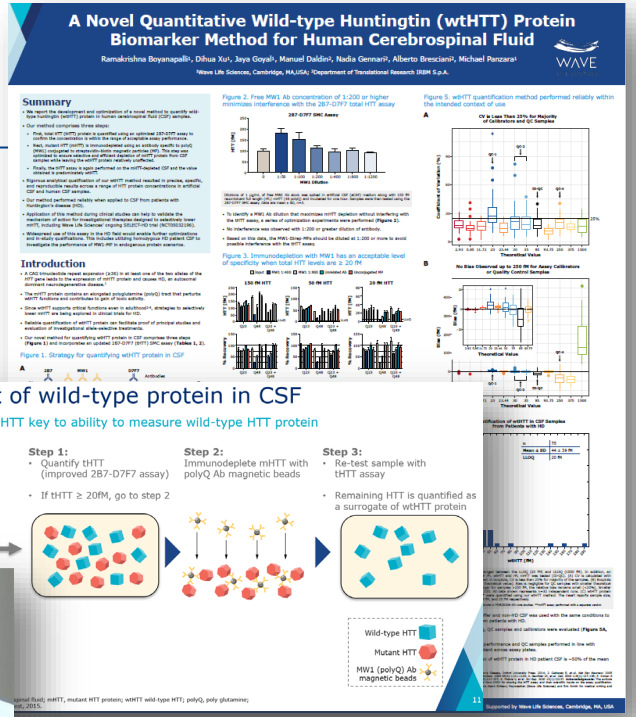
Wave's allele-selective approach to Huntington's disease highlighted at CHDI conference

- CHDI's 17th Annual HD Conference (Feb 28th – March 3)
- Interest on the importance of preserving wtHTT in the context of HTT reducing treatments remains high in the HD community
- Wave poster: "A novel quantitative wild-type huntingtin (wtHTT) protein biomarker method for human cerebrospinal fluid"
- Wave presentation: "Innovations that led to SELECT-HD, a Phase 1b/2a clinical trial of an allele-selective therapy for Huntington's Disease"



SELECT HD

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Paloma Giangrande, PhD

Vice President, Platform

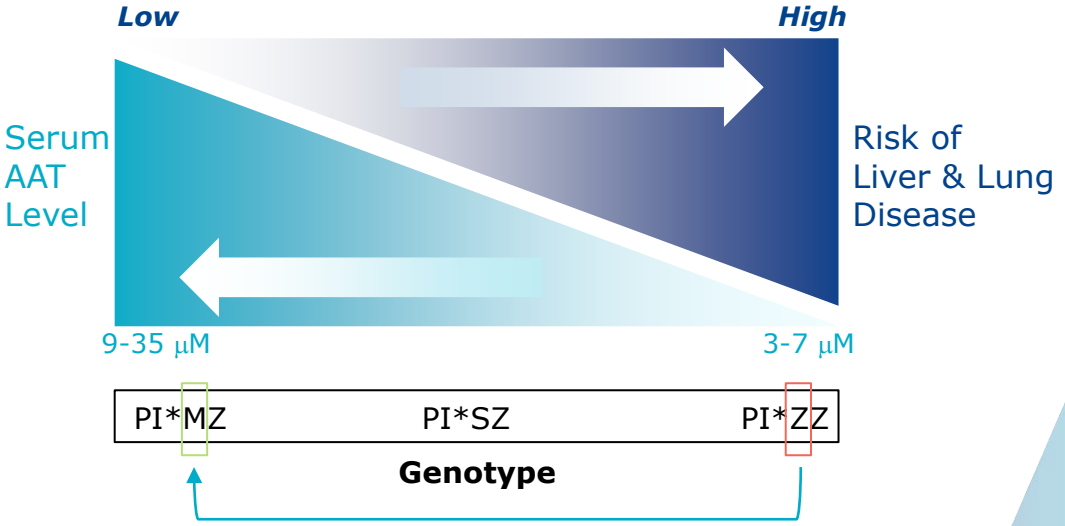
Discovery Sciences, Biology

ADAR editing approach to correct AATD with GalNAc-AIMers addresses lung and liver pathologies

Alpha-1 antitrypsin deficiency (AATD)

- AAT protein produced by hepatocytes
- Point mutation in *SERPINA1* gene (PiZ allele) leads to misfolding and aggregation of mutant Z-AAT protein in hepatocytes in form of globules
- Pathology:
  - Liver damage, fibrosis, cirrhosis, and hepatocellular carcinoma
  - Lung emphysema and bronchiectasis

Inverse relationship between circulating AAT levels and disease risk



Therapeutic goal: ~50% RNA editing with AIMers to reach MZ phenotype with low risk of lung / liver disease

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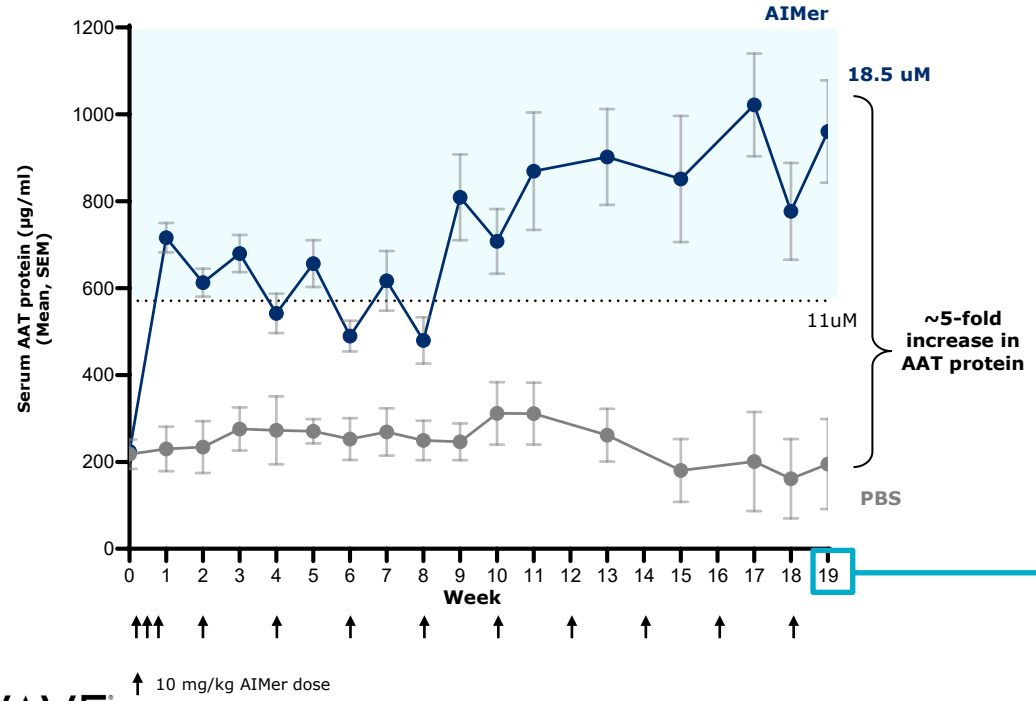
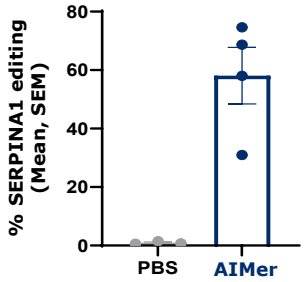
Sources: Strnad 2020; Blanco 2017; Remih 2021

Preclinical AIMer treatment results in circulating AAT protein levels well above anticipated therapeutic threshold

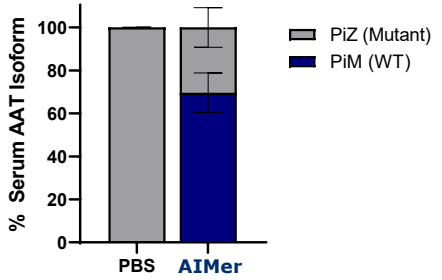
GalNAc-AIMer treatment bi-weekly results in serum AAT protein levels >11 uM at week 19 in transgenic mice



~60% RNA editing with GalNAc-AIMer treatment



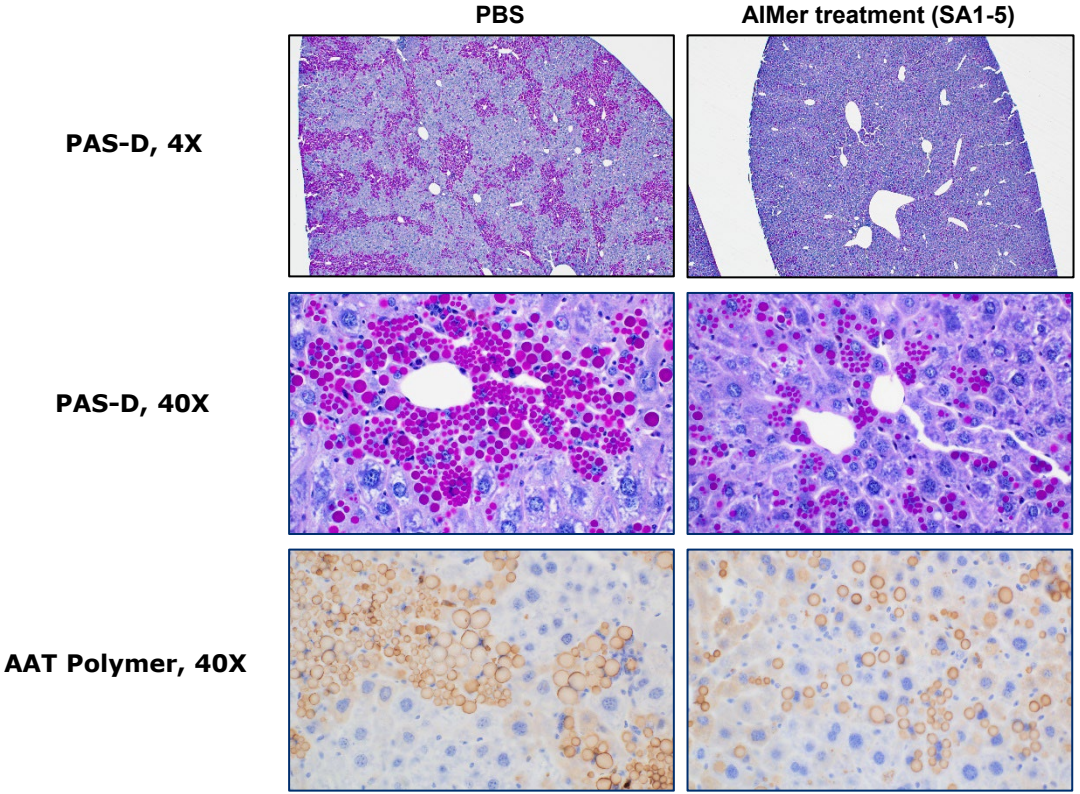
~70% wild-type, M-AAT protein in serum with GalNAc-AIMer treatment



AIMers (SA1-5) administered in hu*ADAR/SERPINA1* mice (8 – 10 weeks old) Left : Total AAT protein quantified by ELISA. Right: Liver biopsies collected at week 19 (one week after last dose) and SERPINA1 editing was quantified by Sanger sequencing

Histological analysis indicates reduction of liver aggregates at 19 weeks with AIMer treatment

Preliminary histological analysis of transgenic mouse liver aggregates (Week 19)



Representative images from liver biopsies stained with PAS-D (top, middle) or AAT-polymer specific antibody (bottom)

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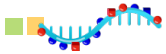
GalNAc-AIMers are uniquely suited to address the key treatment goals for AATD

- ✓ Recruit endogenous ADAR enzyme to edit SERPINA1 Z mRNA with high specificity
- ✓ Restore circulating, functional M-AAT protein above expected therapeutic threshold (11 μM)
- ✓ Reduce Z-AAT protein aggregation in liver



	AIMers	RNAi	AAT augmentation therapy
Restore circulating functional wild-type AAT	✓		✓
Reduce Z-AAT protein aggregation in liver	✓	✓	
Retain M-AAT physiological regulation	✓		

Expect to select an AATD AIMer development candidate and initiate IND-enabling toxicology studies in 3Q 2022

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https://www.labiotech.eu

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Kyle Moran

Chief Financial Officer

Fourth quarter 2021 financial results

	Three Months Ended December 31, 2021	Three Months Ended December 31, 2020
Figures are in thousands, except per share amounts		
Revenue	$1,765	$9,439
Operating Expenses:		
Research and Development	25,761	30,033
General and Administrative	12,114	9,719
Total Operating Expenses	37,875	39,752
Net Loss from Operations	(36,110)	(30,313)
Total Other Income, Net	1,121	683
Income Tax Benefit, net	204	841
Net Loss	($34,785)	($28,789)
Net Loss per Share	**($0.61)**	**($0.59)**

As of December 31, 2021 **Shares Outstanding:** 59.8 million **Cash Balance:** $150.6 million

Wave expects that its existing cash and cash equivalents will enable the company to fund its operating and capital expenditure requirements into 2Q 2023.

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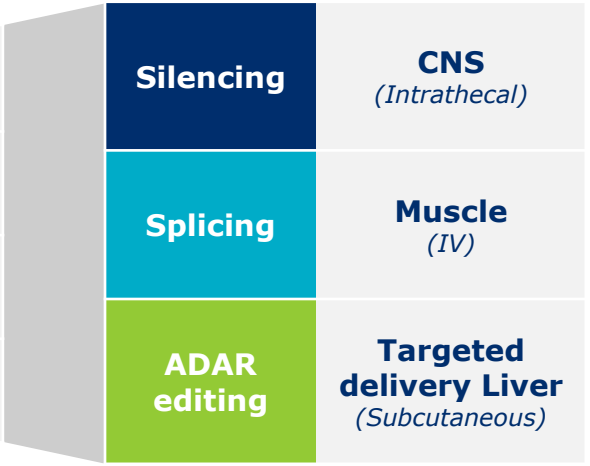
Paul Bolno, MD, MBA

President and CEO

Data generated in 2022 expected to inform future opportunities and unlock value

WVE-004 C9orf72 ALS & FTD	• Clinical data to enable decision making in 2022
WVE-003 HD SNP3	• Clinical data to enable decision making in 2022
WVE-N531 DMD Exon 53	• Clinical data to enable decision making in 2022
AIMer AATD SERPINA1	• Select an AATD AIMer development candidate and initiate IND-enabling toxicology studies in 3Q 2022



Success with any current program validates platform and unlocks modalities and tissues

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Realizing a brighter future for people affected by genetic diseases

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