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

Form 8-K

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

Date of Report (Date of earliest event reported): September 28, 2021

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

Singapore	**001-37627**	**00-0000000**
(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. ☐

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading symbol	Name of each exchange on which registered
$0 Par Value Ordinary Shares	**WVE**	**The Nasdaq Global Market**

Item 7.01 Regulation FD Disclosure.

On September 28, 2021, Wave Life Sciences Ltd. (the "Company") hosted an Analyst and Investor Research Webcast and shared a slide presentation that is available on the "For Investors & Media" section of the Company's website at http://ir.wavelifesciences.com/. This presentation is also furnished as Exhibit 99.1 to this Current Report on Form 8-K.

In addition, on September 28, 2021, the Company issued a press release announcing the data presented during the Company's Analyst and Investor Research Webcast. A copy of the press release is furnished as Exhibit 99.2 to this Current Report on Form 8-K.

The information in this Item 7.01 is being 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 into any registration statement or other filing under the Securities Act of 1933, as amended, or the Exchange Act, except as shall be expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Analyst & Investor Research Webcast for Wave Life Sciences Ltd. dated September 28, 2021
99.2	Press Release issued by Wave Life Sciences Ltd. dated September 28, 2021
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURES

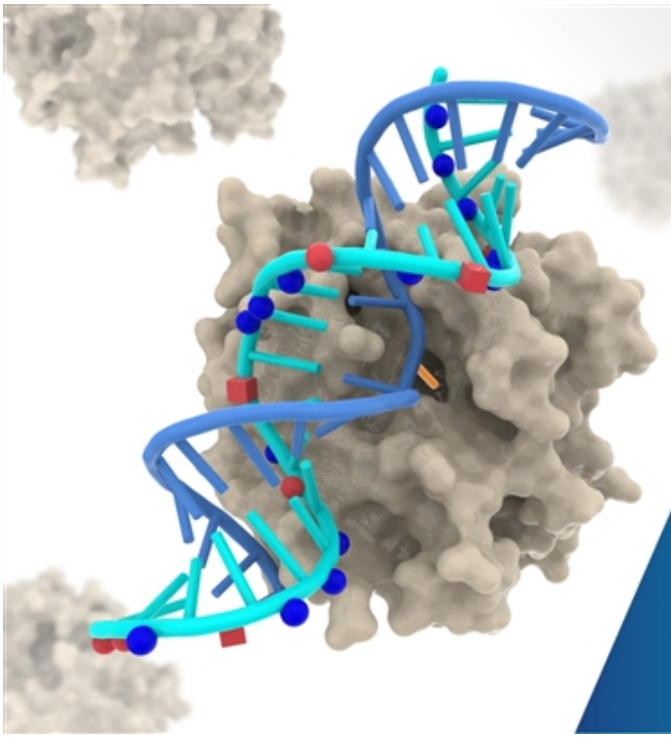
Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

WAVE LIFE SCIENCES LTD.

By: */s/ Paul B. Bolno, M.D.*
Paul B. Bolno, M.D.
President and Chief Executive Officer

Date: September 28, 2021

Exhibit 99.1



WAVE™
LIFE SCIENCES

Analyst & Investor Research Webcast

September 28, 2021

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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Today's agenda

PRESENTATION	SPEAKER
Opening Remarks	**Paul Bolno, MD, MBA** President and CEO
Applying PRISM Principles for Rational Oligonucleotide Design	**Chandra Vargeese, PhD** Chief Technology Officer
Building a Best-in-Class ADAR Editing Capability: Introducing AIMers	**Chandra Vargeese, PhD** Chief Technology Officer
Advancing ADAR Editing in the CNS	**Ken Rhodes, PhD** SVP, Therapeutics Discovery
Restoring Functional AAT Protein with ADAR Editing: Program Update	**Paloma Giangrande, PhD** VP, Platform & Discovery Sciences Biology
Q&A	
Closing Remarks	**Paul Bolno, MD, MBA** President and CEO

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Opening Remarks

Paul Bolno, MD, MBA

President and CEO

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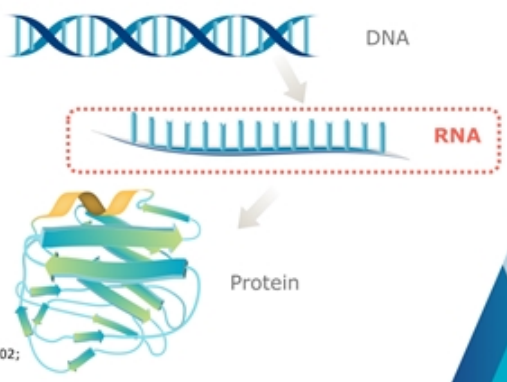
We are taking part in a genetic revolution

- **Greater understanding** of genetic drivers of disease and definition at molecular level
- **>6,000** genetically defined diseases
- Increase in **genetic testing** enabling identification of individuals likely to benefit from treatment

Many diseases beyond the reach of traditional treatments

- Wave is developing therapeutics to drug the **transcriptome** to turn on, switch off, or modulate expression of faulty genes



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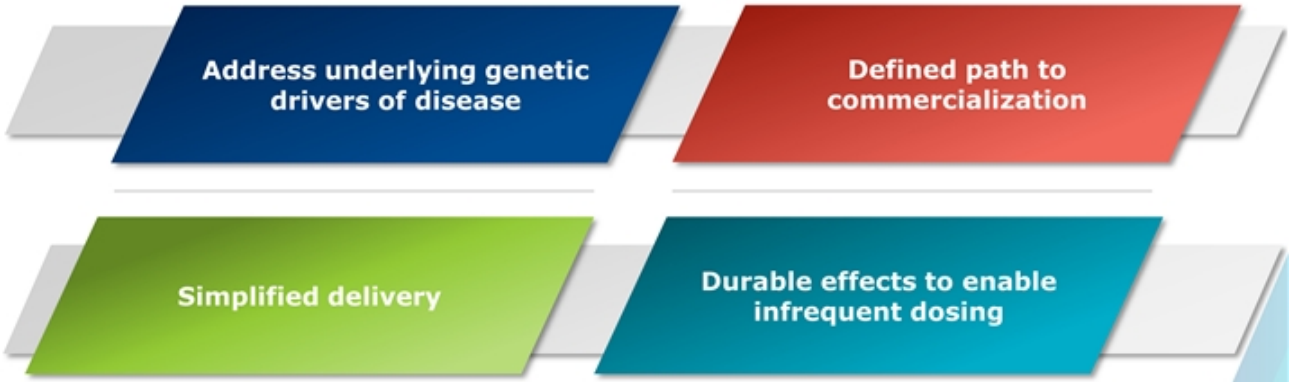
Sources: Shen et al, Genetics Research, 2015; Hopkins et al, Nat Rev Drug Discov, 2002; geneticdiseasefoundation.org

Strategic decision to intervene at RNA level

RNA-targeting therapeutics offer ideal balance of precision, durability, potency, and safety



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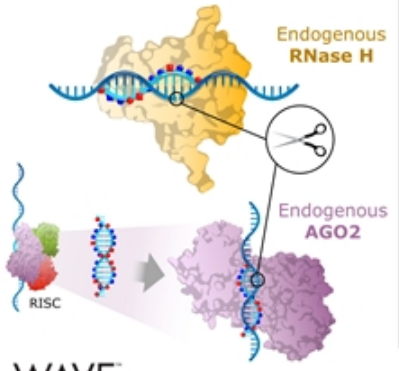
Biological machinery in our cells can be harnessed to treat genetic diseases

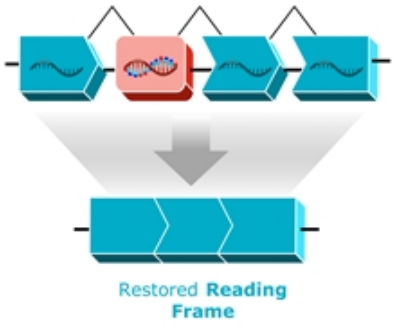
Silencing

- **Oligonucleotide-directed delivery** of RNA to regulate enzymes



Splicing

- Leverages **exon skipping machinery** to restore a working transcript



Editing

- Efficient editing of RNA bases using **endogenous ADAR**

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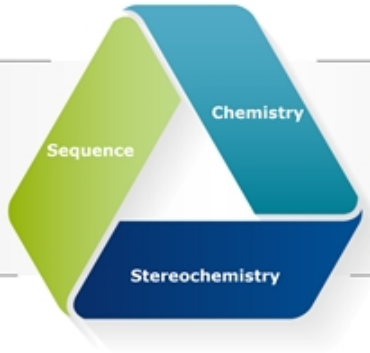
PRISM Unlocking the body's own ability to treat genetic disease

DESIGN

Unique ability to construct single isomers and control three structural features of oligonucleotides to efficiently engage biological machinery



OPTIMIZE

Provides the resolution to observe this structural interplay and understand how it impacts key pharmacological properties

Built-for-Purpose Candidates to Optimally Address Disease Biology

Silencing | Splicing | RNA Editing

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Wave is the leader in chirally-controlled rationally designed stereopure oligonucleotides

Stereochemistry is a reality of chemically-modified nucleic acid therapeutics

Current therapeutics with chiral backbone modifications:

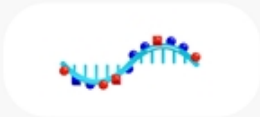
- **Antisense oligonucleotides**
- **siRNA**
- **Exon-skipping oligonucleotides**
- **mRNA therapeutics**
- **RNA guide strands**

Chirality matters: affects pharmacology of oligonucleotides *in vitro* and *in vivo*

Increasingly recognized by leaders in nucleic acid therapeutics:

PRISM controls stereochemistry throughout drug discovery and development process

Enables design and optimization of fully-characterized, **single-isomer** RNA therapeutics



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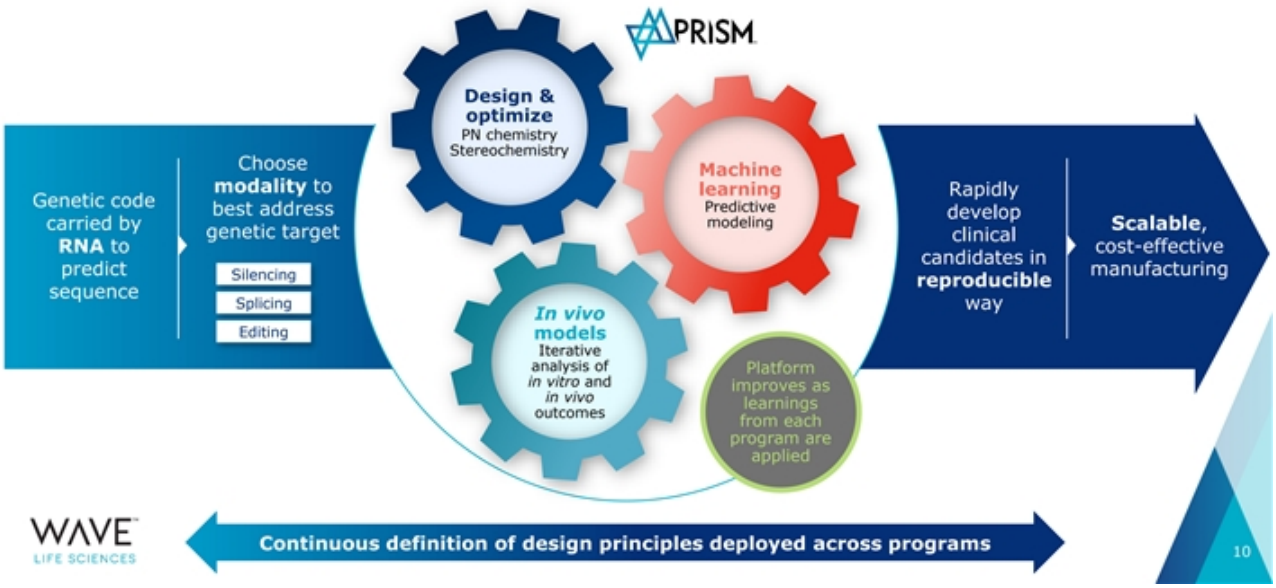
Dominant IP portfolio and unique ability to manufacture and screen stereopure oligonucleotides

Jahns et al., NAR, 2021; Hansen, et al. 2021; Funder, Albaek et al. 2020

PRISM platform is continuously improving



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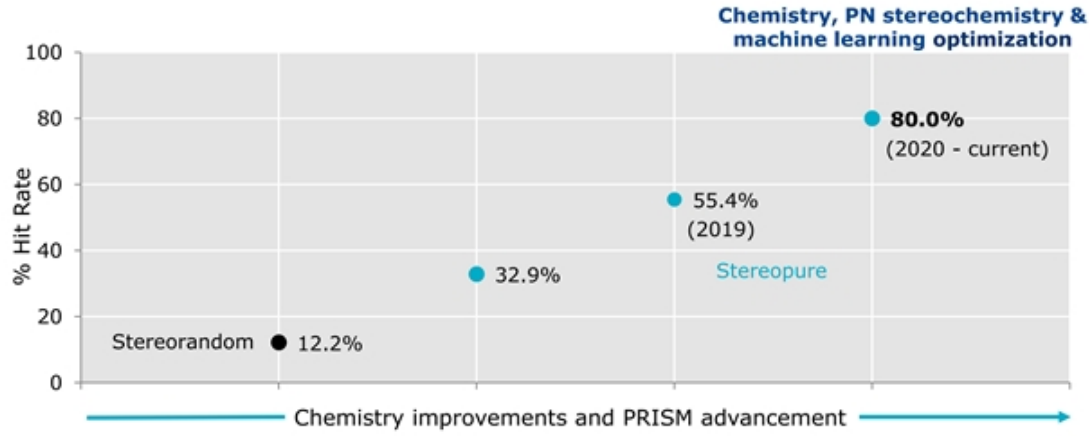
Continuous definition of design principles deployed across programs

Improvements in PRISM primary screen hit rates accelerate drug discovery

Primary screen hit rates with silencing far above industry standard hit rates



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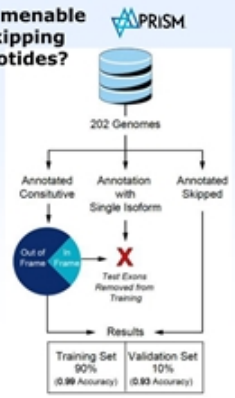
All screens used iPSC-derived neurons; Data pipeline for improved standardization. Hit rate = % of oligonucleotides with target knockdown greater than 50%. Each screen contains >100 oligonucleotides. ML: machine learning

PRISM

Data sciences enable prediction of new potential therapeutic exon-skipping targets

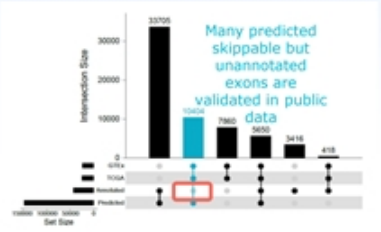
Model trained on millions of known protein sequences

Is an exon amenable to exon-skipping oligonucleotides?



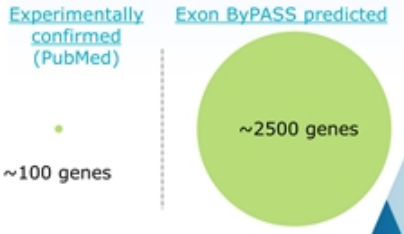
Predicts skippable exons that are currently undiscovered

Identified >10,000 exons that are predicted to be skippable but are currently unannotated



Identifies clinically relevant genes with skippable exons

Identified ~2,500 potential exon-skipping targets with oligonucleotide therapeutics as compared ~100 identified skippable in literature



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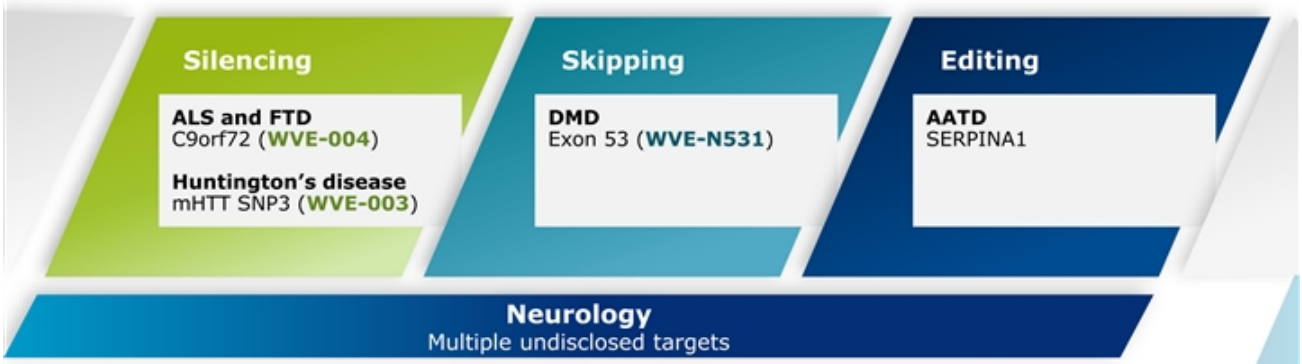
Exon ByPASS: predicting Exon-skipping Based on Protein Amino acid SequenceS; Data presented at OTS 2021. Manuscript submitted

Advancing programs using multiple modalities



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ALS: Amyotrophic lateral sclerosis; FTD: Frontotemporal dementia; DMD: Duchenne muscular dystrophy; AATD: Alpha-1 antitrypsin deficiency

Building a leading genetic medicines company

- Scientific approach focused on unlocking the body's own ability to treat genetic disease
- PRISM platform enables multiple modalities for built-for-purpose therapeutics
- Leading the way in rationally designed stereopure oligonucleotides with innovative backbone chemistry
- Robust portfolio of PN-modified, stereopure oligonucleotides, including three programs in clinic and multiple ADAR editing discovery programs

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Applying PRISM Principles for Rational Oligonucleotide Design

Chandra Vargeese, PhD

Chief Technology Officer

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PRISM platform enables rational drug design

Sequence

B: bases

A, T, C, mC, G, U,
other modified bases

Stereochemistry

Chiral control of
any stereocenter

5′ modifications,
backbone modifications

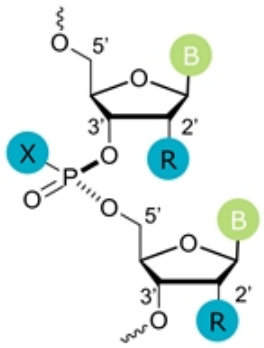
Chemistry

R: 2′ modifications

OMe, MOE, F,
other modifications

X: backbone chemistry

PO, PS, PN



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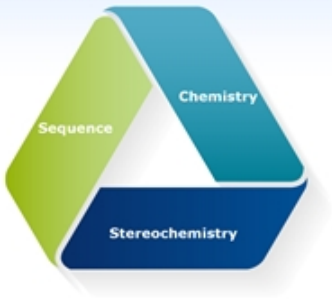
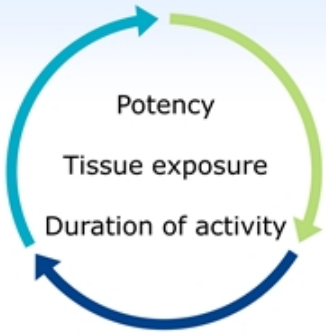
Optimization framework compatible across different modalities

PRISM

Interplay between key structural components of oligonucleotides…



..to modulate key aspects of activity…



… and apply to multiple therapeutic modalities

- Silencing
- Splicing (exon-skipping)
- ADAR editing

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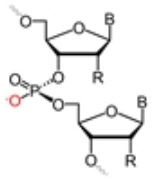
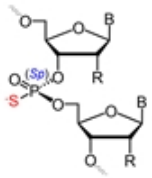
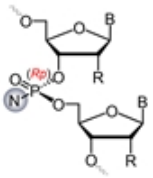
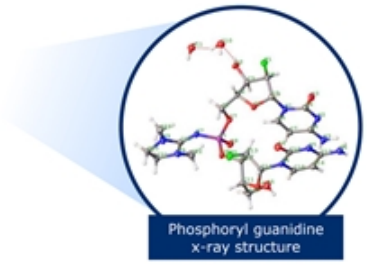
Innovating new backbone chemistry modifications

PRISM backbone linkages

PO	PS	PN
		
		
Chirality None	**Chirality** PS backbone *Rp* PS backbone *Sp*	**Chirality** PN backbone *Rp* PN backbone *Sp*
Negative charge	**Negative charge**	**Neutral charge**



Phosphoryl guanidine x-ray structure

example

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PO: phosphodiester PS: phosphorothioate

Rationally placed stereopure PN modifications enhance pharmacology across modalities

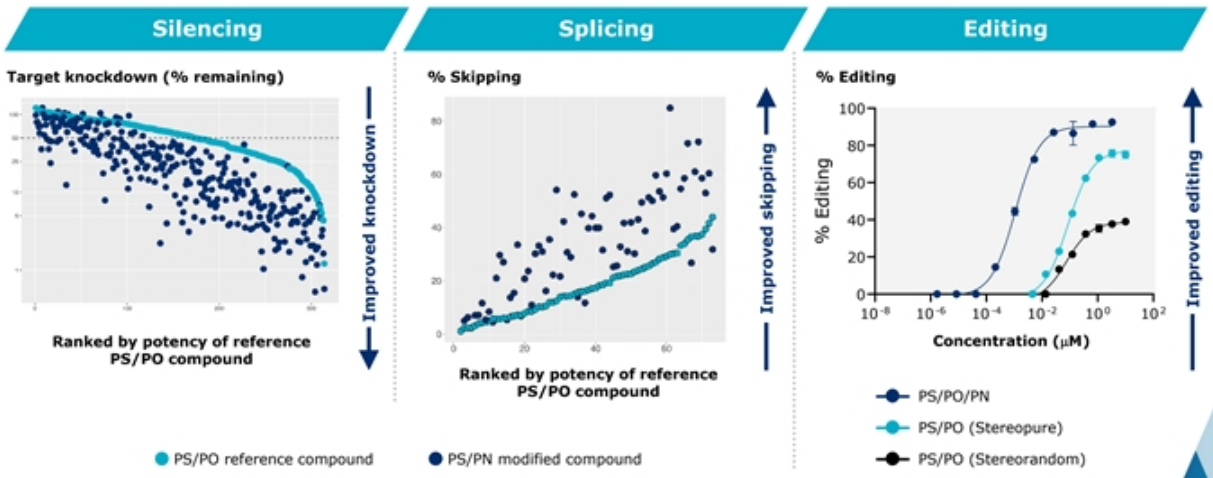
Adding PN linkages benefits all PRISM modalities…

Silencing	Efficient engagement of RNase H or Ago2
Splicing	Efficient uptake in the cell nucleus
Editing	Efficient engagement of ADAR

… and improves key pharmacological drivers of translation

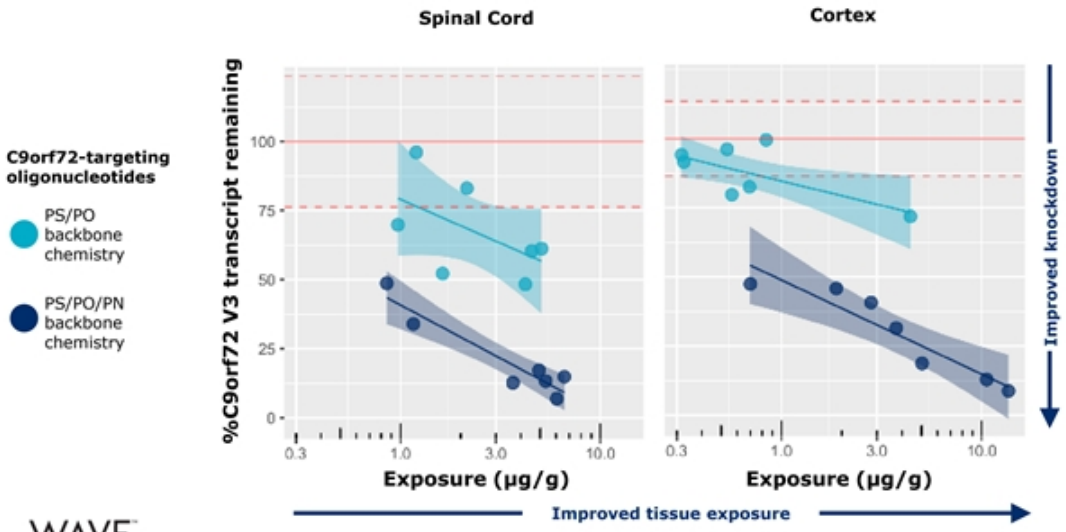
Potency	Target knockdown, splicing or editing
Exposure	In the right tissues, cells and cellular compartments
Durability	Enabling infrequent administration

Potency is enhanced with addition of PN modifications across modalities



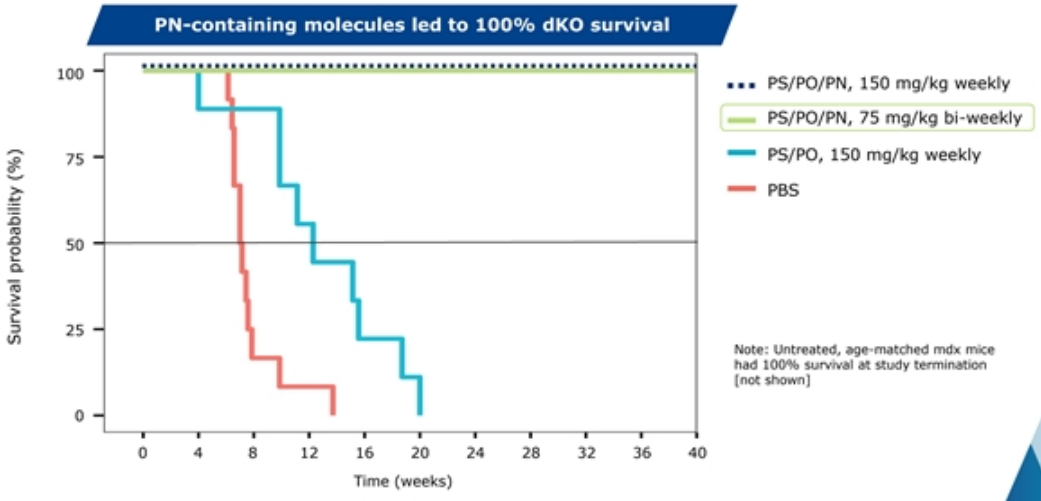
Left: Experiment was performed in iPSC-derived neurons *in vitro*; target mRNA levels were monitored using qPCR against a control gene (HPRT1) using a linear model equivalent of the ΔΔCt method; Middle: DMD patient-derived myoblasts treated with PS/PO or PS/PO/PN stereopure oligonucleotide under free-uptake conditions. Exon-skipping efficiency evaluated by qPCR. Right: Data from independent experiments

Adding PN chemistry modifications to C9orf72-targeting oligonucleotides improved potency *in vivo*



Target knockdown: Liu, TIDES poster 2021; Oligonucleotide concentrations quantified by hybridization ELISA. Graphs show robust best fit lines with 95% confidence intervals (shading) for PK-PD analysis. Manuscript submitted.

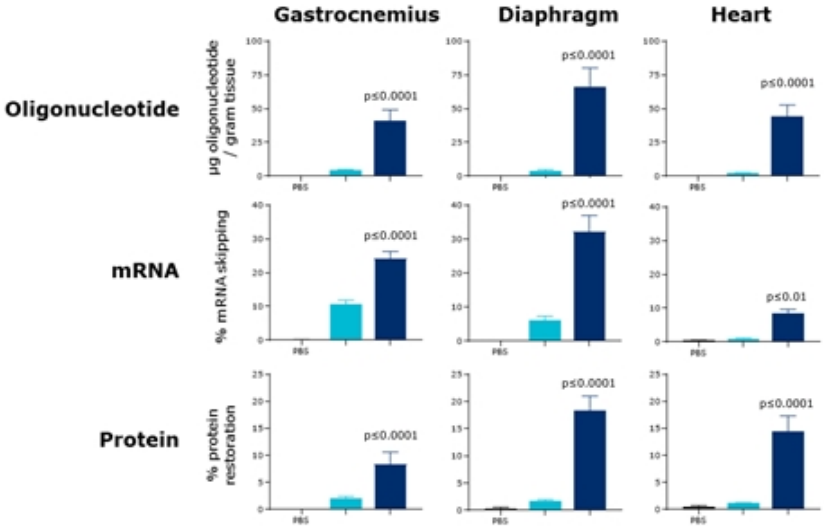
Adding PN chemistry modifications led to overall survival benefit in dKO model



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

dKO; double knockout mice lack dystrophin and utrophin protein. mdx mice lack dystrophin. Left: Mice with severe disease were euthanized. dKO: PS/PO/PN 150 mg/kg n= 8 (p=0.0018); PS/PO/PN 75 mg/kg n=9 (p=0.00005); PS/PO n=9 (p=0.0024), PBS n=12 Stats: Chi square analysis with pairwise comparisons to PBS using log-rank test. Manuscript submitted.

PN chemistry improves exposure and target engagement in key tissues

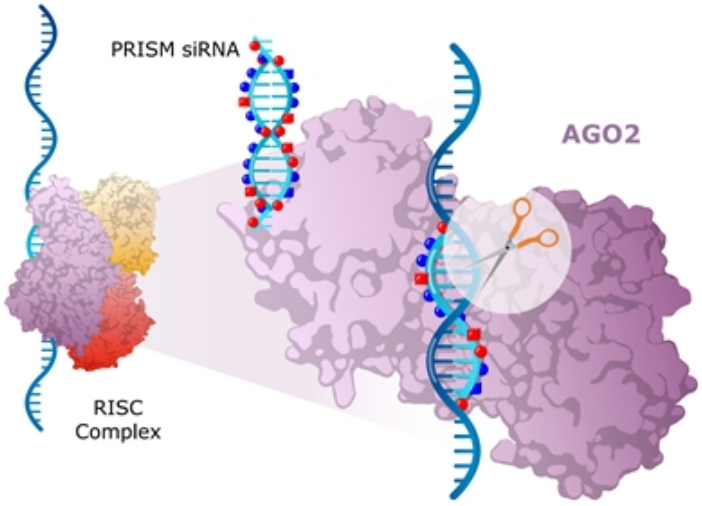


Exon-skipping oligonucleotides

- PS/PO backbone chemistry
- PS/PO/PN backbone chemistry

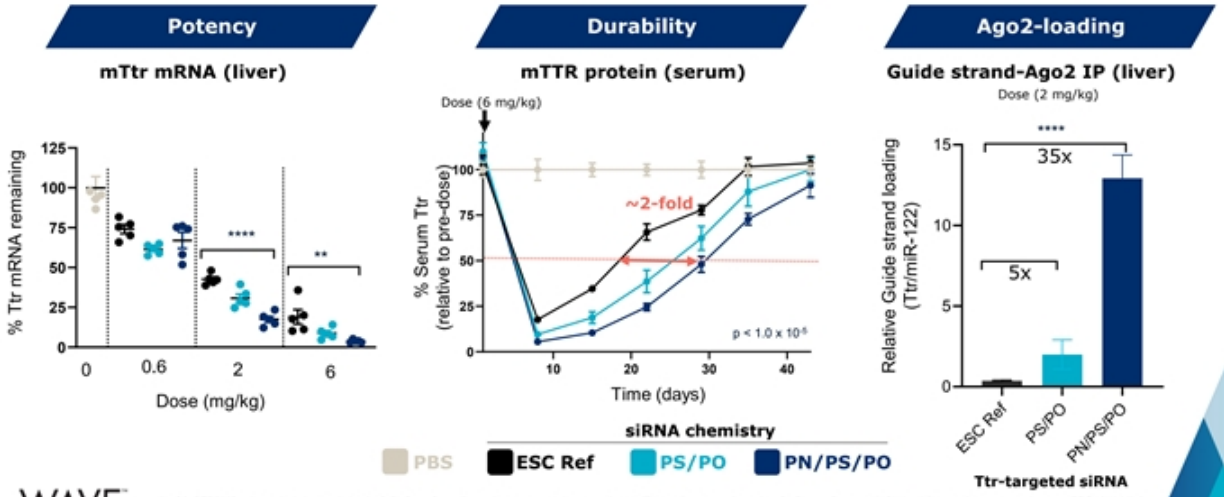
6x weekly 75 mg/kg subcutaneous doses; Sample collected 2 days after last dose. Manuscript submitted

PRISM principles applied to another class of silencers: siRNA



Application of PRISM principles to siRNA improves another class of silencers

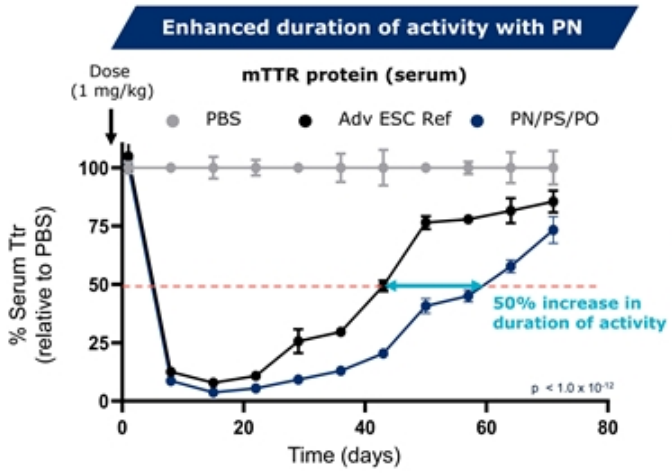
PN chemistry improves potency and durability of ESC format



(Left) C57Bl/6 mice administered single 0.2, 2 or 6 mg/kg subcutaneous dose on day 1. Tissue harvested on day 8. Stats: 2-way ANOVA with post-hoc comparison to ESC. (Middle) Mice received single 6 mg/kg subcutaneous dose on day 1. Serum collected weekly. Stats: 2-way mixed ANOVA with post hoc comparisons PN vs Reference. (Right) As described for left panel (2 mg/kg); Ago2 loading measured by qPCR after immunoprecipitation (IP) and normalized to miR-122; Stats: 1-way ANOVA followed by Tukey's honest significance test. ** P<0.01, *** P<0.001****, P<0.0001. All post-hoc P values Bonferroni-corrected for multiple hypotheses. Reference: Enhanced Stabilization Chemistry

Application of PN chemistry to siRNA: Improving on the state-of-the-art

PN chemistry extends duration of GalNAc-conjugated Advanced ESC format



- PN extends 50% knockdown period for GalNAc-conjugated Adv ESC siRNAs
- Further optimization studies are in progress

Mice received a single 1 mg/kg subcutaneous dose on day 1. Serum was collected weekly. Stats: 2-way mixed ANOVA with post hoc comparisons PN vs Reference. P value is Bonferroni corrected for multiple hypotheses.

PRISM provides visibility into effects of backbone stereochemistry within every sequence

- Backbone stereochemistry impacts pharmacologic properties
- PRISM enables stereochemical control to fully characterize and investigate structure activity relationship (SAR) of each therapeutic candidate
- Standard in small molecule and antibody development

Backbone stereochemistry can be a tool to modulate pharmacologic properties, including tolerability

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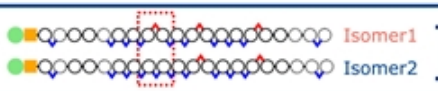
A single stereoisomeric change can dramatically alter the tolerability profile *in vivo*

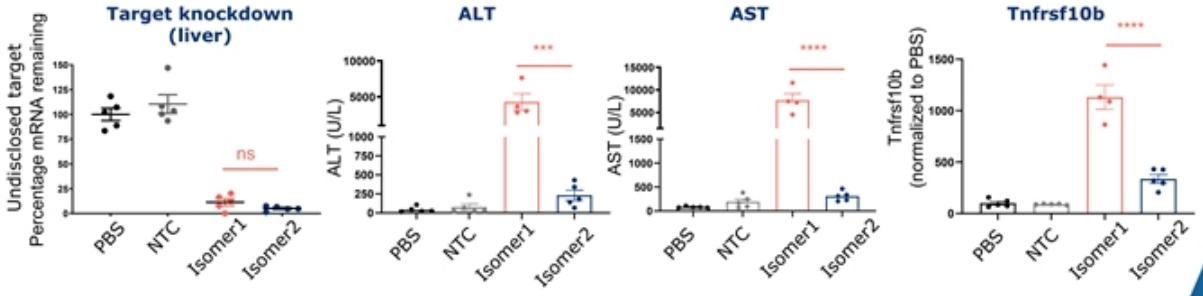
GalNAc conjugated oligonucleotide administered **subcutaneously**



Same sequence and chemical modifications, but different stereochemistry

Stereoisomers have **similar** pharmacodynamic effects

Changing backbone stereochemistry leads to **different** hepatotoxicity profiles *in vivo*



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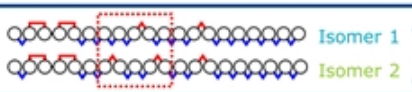
C57Bl/6 mice were administered 5 mg/kg oligonucleotide or PBS by subcutaneous injection on days 1, 3, 5 and 8. Liver tissue was collected on day 11. Target mRNA was normalized to Hprt1. Data are presented as mean ± sem (n=5). Stats: One-way ANOVA ns not significant, PBS phosphate buffered saline, NTC non-targeting control

Stereoisomeric changes can dramatically alter the tolerability profile in the CNS *in vivo*

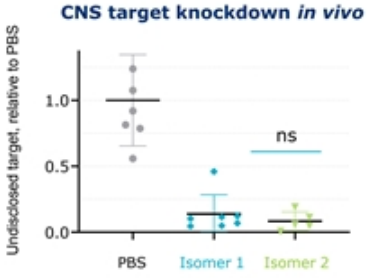
Unconjugated oligonucleotide administered **ICV**



Same sequence and chemical modifications, but different stereochemistry

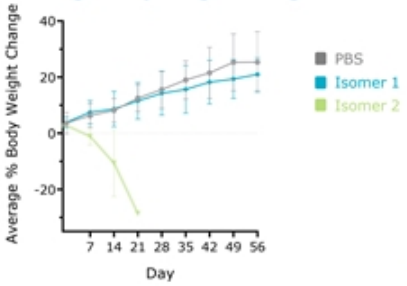
Stereoisomers have **similar** pharmacodynamic effects *in vivo*

CNS target knockdown *in vivo*



Changing backbone stereochemistry leads to **different** tolerability profiles *in vivo*

Percentage Body Weight Change



Left: In a target engagement study, 7 mice administered 2 x 50 ug oligonucleotide or PBS by ICV on days 0 and 7. Tissue collected on day 14. Target mRNA normalized to Tubb3 and plotted relative to PBS. Data presented as mean ± SD (n=7). Stats: One-way ANOVA ns not significant, PBS phosphate buffered saline. Right: wtmouse tolerability study, n=4 administered 100 ug oligonucleotide or PBS by ICV on day 0 and monitored for 8 weeks.

PRISM enables novel advances in oligonucleotide design for optimization of RNA therapeutics

- PRISM uses deep understanding of interplay between sequence, chemistry and stereochemistry
- Rationally placed PN backbone chemistry modifications improve potency, durability of effect and distribution *in vitro* and *in vivo* across silencing, including RNAi, splicing and editing modalities
- Backbone stereochemistry can be a tool to modulate pharmacologic properties, including tolerability

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Building a Best-in-Class RNA Editing Capability: Introduction of AIMers

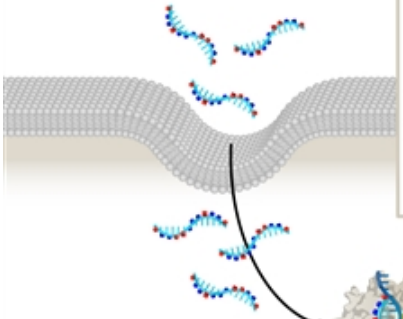
Chandra Vargeese, PhD

Chief Technology Officer

WAVE LIFE SCIENCES

Unlocking RNA editing with PRISM platform to develop AIMers: A-to-I editing oligonucleotides

Free-uptake of chemically modified oligonucleotides



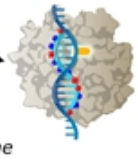
- First publication (1995) using oligonucleotide to edit RNA with endogenous ADAR[1]
- Wave goal: Expand toolkit to include editing by unlocking ADAR with PRISM oligonucleotides

- ✓ Learnings from biological concepts
- ✓ Applied to ASO structural concepts
- ✓ Applied PRISM chemistry

AIMer:
Wave's **A**-to-**I** editing oligonucleotides

Endogenous enzymes

ADAR
RNase H
AGO2
Spliceosome



ADAR enzymes

- Catalyze conversion of A-to-I (G) in double-stranded RNA substrates
- A-to-I (G) edits are one of the most common post-transcriptional modifications
- ADAR1 is ubiquitously expressed across tissues, including liver and CNS

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[1]Woolf et al., PNAS Vol. 92, pp. 8298-8302, 1995

Building best-in-class ADAR editing capability

Topics of discussion

1 Applications | **2 Design & Optimize** | **3 Translation *in vivo***

- Restore protein expression
- Modulate protein activity

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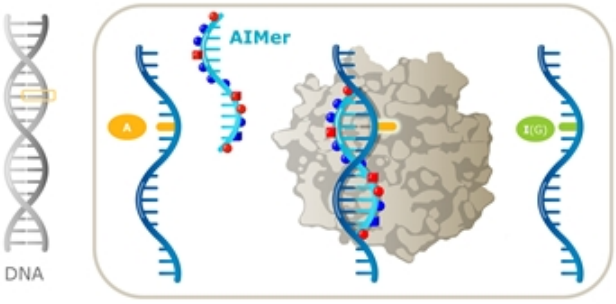
ADAR editing enables correction of single-point mutations to restore functional protein

Restore functional protein



Restore or correct expression

Example therapeutic areas

- AATD
- Rett syndrome
- Recessive or dominant genetically defined diseases

- >32,000 pathogenic human SNPs[2] – nearly half are ADAR amenable (G-to-A mutations)
- Tens of thousands of potential disease variants A-to-I(G) editing could target[1]
- ~12% of all reported disease-causing mutations are single point mutations that result in a premature stop codon[3]

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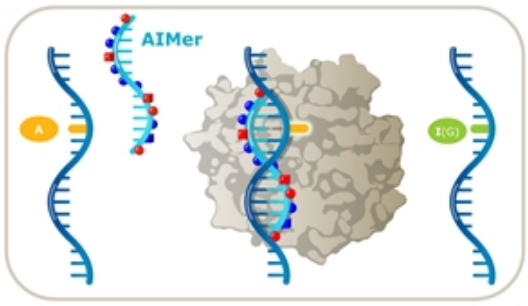
SNP: single nucleotide polymorphism A: Adenosine I: Inosine G: Guanosine
[1]ClinVar database [2]Gaudeli NM et al. *Nature* (2017) [3]Keeling KM et al., Madame Curie Bioscience Database 2000-2013

ADAR editing to modulate proteins at transcript level opens wide range of large therapeutic applications

Modulate downstream protein interactions with single RNA base edit



Upregulate expression

Modify function

Modulate protein-protein interaction

Post-translational modification

Alter folding (stability)

Alter processing

Example therapeutic areas

- Haploinsufficient diseases
- Loss of function
- Neuromuscular
- Dementias
- Familial epilepsies
- Neuropathic pain

- Opens wide range of therapeutic applications with large patient populations

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Building best-in-class ADAR editing capability

Topics of discussion

1 Applications

- Restore protein expression
- Modulate protein activity

2 Design & Optimize

- Applying unique chemistry capabilities to AIMers enhances editing
- Optimization of chemistry and SAR informs design principles for future rational design

3 Translation *in vivo*

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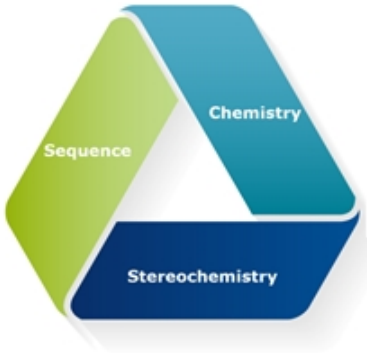
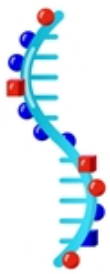
Unique chemistry platform enables rational design of AIMers to efficiently recruit ADAR enzymes



AIMers

- RNA base editing oligonucleotides
- Short, single-stranded
- Fully chemically modified
- Modified nucleobases
- Stereopure PS and PN backbone modifications
- Compatible with targeting ligands



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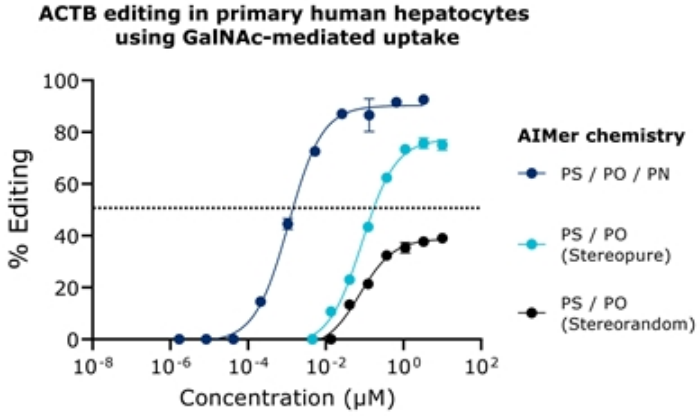
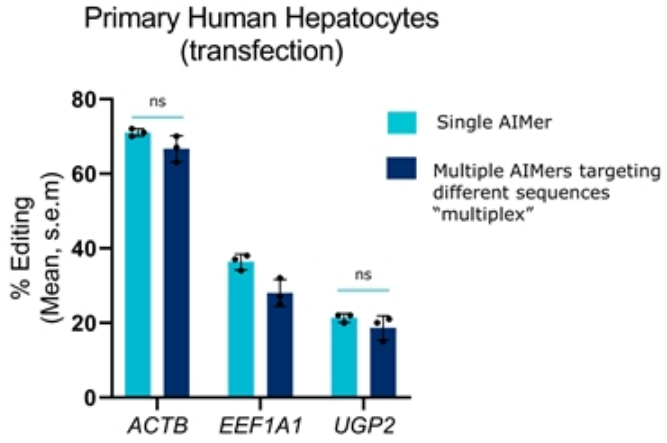
Stereochemistry and PN chemistry enhance potency and editing efficiency of AIMers



Data from independent experiments; Total RNA was harvested, reverse transcribed to generate cDNA, and the editing target site was amplified by PCR and quantified by Sanger sequencing

Levels of endogenous ADAR enzyme are not rate limiting for editing



- Endogenous ADAR enzyme supports editing on multiple independent targets
- Editing efficiency comparable even when additional AIMers targeting different sequences are added, suggesting there is a more than sufficient reservoir of ADAR enzyme

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Percentage A-to-I editing detected on the indicated transcripts in presence of 20 nM each of a single (Isolated) or multiple (Multiplex) AIMers after transfection of primary human hepatocytes (left), or in the presence of 1.1 mM each of a single (Isolated) or multiple (Multiplex) GalNAc conjugated AIMers (right). Data are presented as mean ± SEM, n=3. P values as determine by two-tailed Welch's t-test are indicated. NTC non-targeting control. Manuscript submitted.

Optimization of every dimension to inform future rational design of AIMers

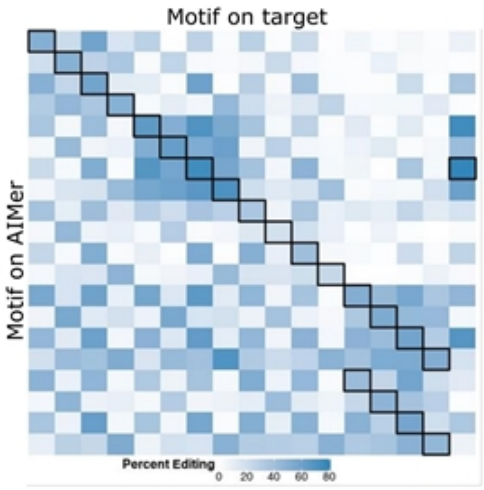
Sequence is one of multiple dimensions for optimization

Sequence space is defined

NNN — AIMer

XAX — mRNA target

- >300 unique AIMers tested containing different base pair combinations
- Identified base modification combinations with high editing efficiency to optimize sequence



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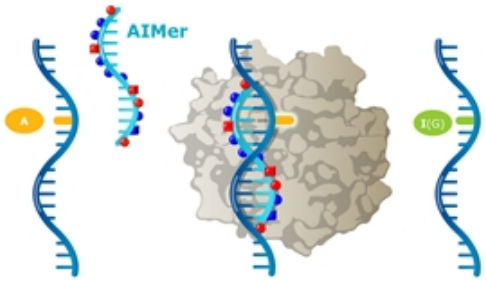
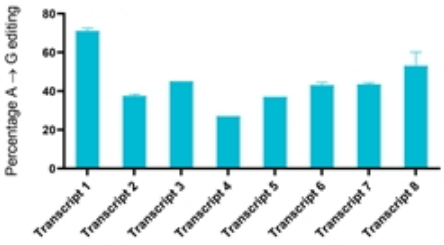
Learnings inform design principles deployed across future targets

ADAR interacts with double-stranded RNA duplex in a sequence independent way



- The intrinsic function of ADAR is to recognize dsRNA **independent of sequence**

RNA-editing design applicable across targets *in vitro* in primary human hepatocytes



- Editing achieved across several distinct RNA transcripts
- Supports potential for technology to be applied across variety of disease targets

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Data presented at 1st International Conference on Base Editing – Enzymes and Applications (Deaminet 2020). Manuscript submitted.

Building best-in-class ADAR editing capability

Topics of discussion

1 Applications

- Restore protein expression
- Modulate protein activity

2 Design & Optimize

- Applying unique chemistry capabilities to AIMers enhances editing
- Optimization of chemistry and SAR informs design principles for future rational design

3 Translation *in vivo*

- GalNAc-conjugated AIMers: liver
- Unconjugated AIMers: CNS, ophthalmology and beyond

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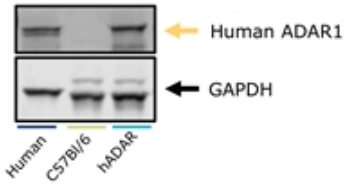
huADAR mouse enables optimization of AIMers to human ADAR

huADAR mouse

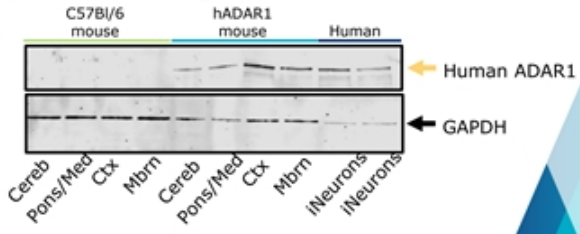
Genotype
✓ huADAR/mADAR
Human ADAR expressed in all tissues

- Transgenic mouse expressing **human ADAR1**
- Expression of ADAR in liver and neurons in mouse approximates expression in corresponding human tissues

Human ADAR expression in hepatocytes



Human ADAR expression in neurons



Western blots showing expression of ADAR1 and GAPDH proteins in the indicated tissue
Left: Protein extracts from human hepatocytes, C57Bl/6 or hADAR1 mouse liver; Right: Protein extracts from cerebellum (Cereb), pons/medulla (pons/med), cortex (Ctx), midbrain (Mbrn), or human iCell neurons (iNeurons)

GalNAc-conjugated AIMers demonstrate proof-of concept of RNA editing in liver

Rapidly advancing first therapeutic program

Delivery	Routes of administration	Tissue types	
GalNAc-conjugated 	**Subcutaneous**	**Liver**	**AATD program**
Unconjugated 	IVT Intrathecal (IT)	Ophthalmology Central nervous system (CNS)	

PN-modified AIMers direct potent and durable editing *in vivo*

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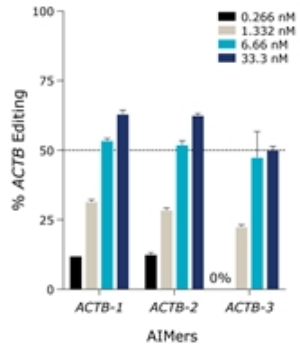
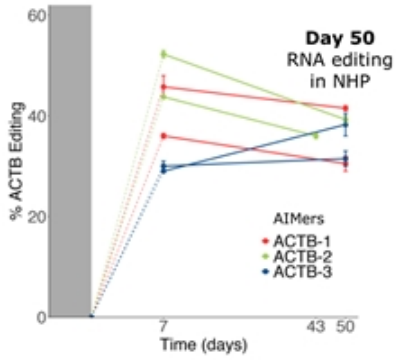
GalNAc-conjugated AIMers support efficient, durable and highly specific ADAR editing in NHPs

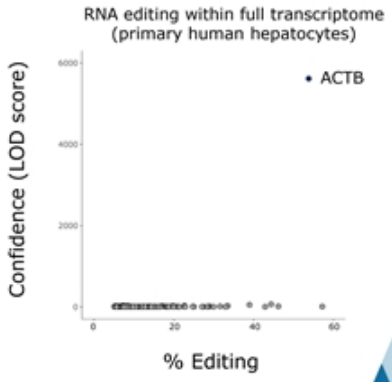
Dose-dependent editing in NHP hepatocytes *in vitro*



Substantial and durable editing in NHP liver *in vivo*



ADAR editing with ACTB AIMer is highly specific



RNA editing only detected at editing site in ACTB transcript

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Left: Total RNA harvested, reverse transcribed to generate cDNA, and editing target site amplified by PCR; % Editing quantified from Sanger sequencing using EditR program; Center: 5mg/kg SC: Day 1,2,3,4,5; Liver Biopsy for mRNA (ACTB Editing); Right: Dosed 1um AIMer, 48 hours later RNA collected, RNAseq conducted using strand-specific libraries to quantify on-target ACTB editing and off-target editing; plotted circles represent sites with LOD>3. Manuscript submitted. NHP: non-human primate; ACTB: Beta-actin

Unconjugated AIMers expand tissues amenable to ADAR editing

Opportunity for future pipeline programs

Delivery	Routes of administration	Tissue types	
GalNAc-conjugated 	Subcutaneous	Liver	
Unconjugated 	**IVT** **Intrathecal (IT)**	**Ophthalmology** **Central nervous system (CNS)**	**MECP2 and undisclosed exploratory programs**

PN-modified AIMers direct potent and durable editing *in vivo*

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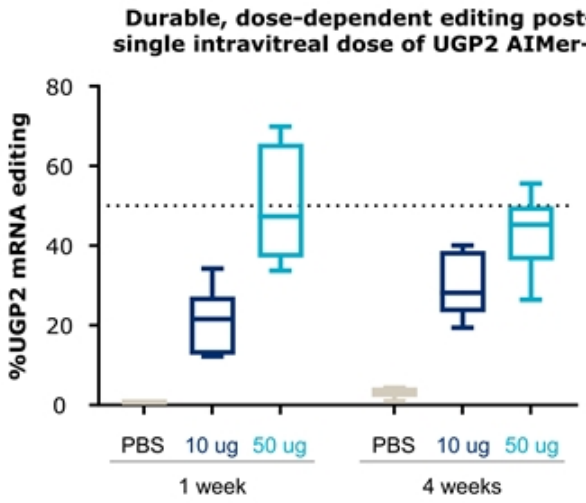
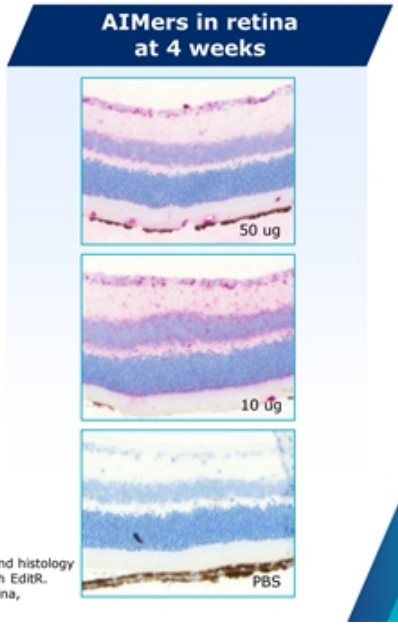
Up to 50% editing *in vivo* in the posterior of eye one month post-single IVT dose

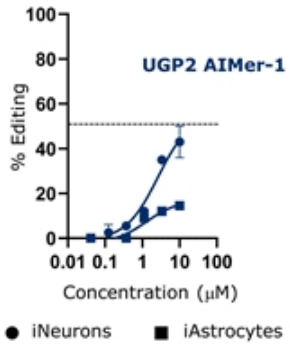




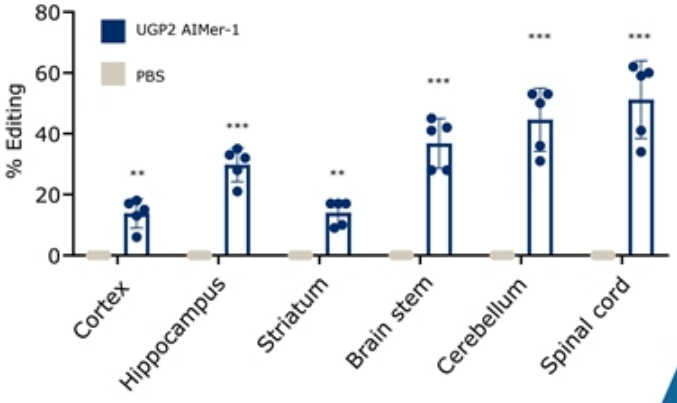
Mice received a single IVT injection (10 or 50 ug AIMer), and eyes were collected for RNA analysis and histology 1 or 4 weeks later. Left: editing evaluated by Sanger sequencing, and % RNA editing calculated with EditR. Right: FFPE and RNA scope assay specific for AIMer, red = oligo, blue = nuclei. Posterior region: retina, choroid, sclera.

AIMers direct editing *in vitro* in multiple CNS cell types and throughout CNS *in vivo*

***In vitro* dose-response**



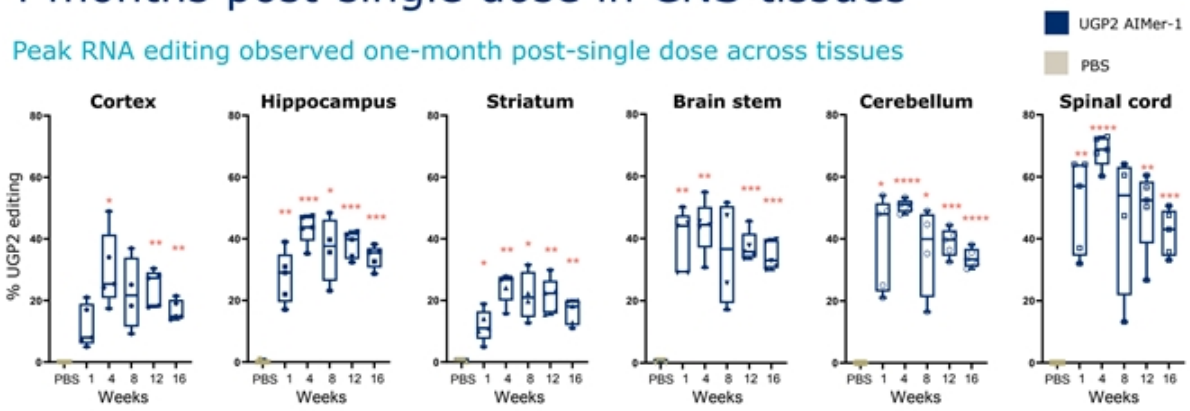
Editing in CNS of hADAR mouse (Single ICV injection, 100 μg)



hADAR: human ADAR; UGP2: Glucose Pyrophosphorylase 2; CNS: central nervous system; Editing observed across all tested tissues of huADAR-transgenic mice. N=5 PBS or single 100 μg ICV dose on day 0, necropsied on day 7. RNA harvested, editing measured by Sanger sequencing. ACTB β-actin; Stats: *p<0.05; **p<0.01; ***p<0.001; ****p<0.0001; all comparisons to PBS-treated group by t test (P values Bonferroni corrected)

Substantial *in vivo* RNA editing out to at least 4 months post-single dose in CNS tissues

Peak RNA editing observed one-month post-single dose across tissues



Peak editing	**30%**	**>40%**	**25%**	**>40%**	**50%**	**>65%**

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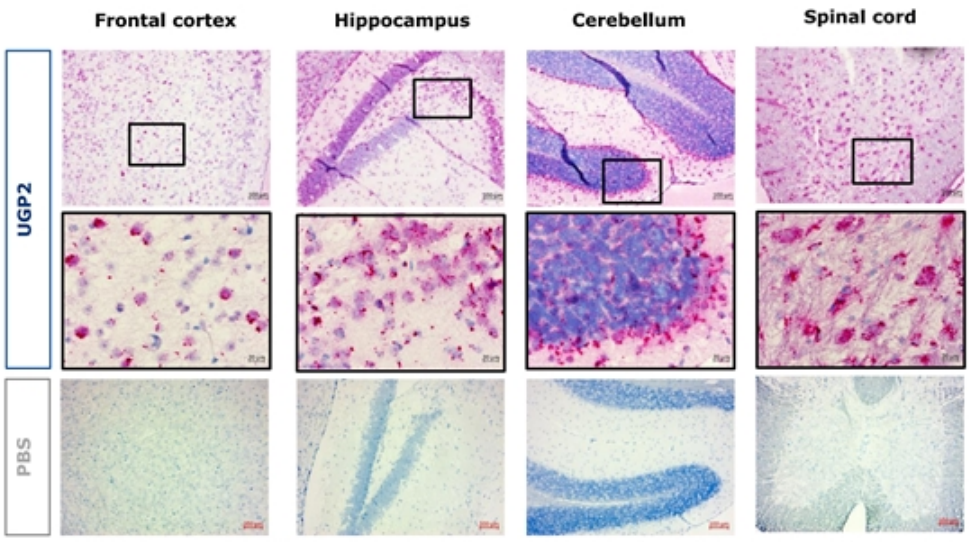
Transgenic huADAR mice administered 100 μg AIMer or PBS on day 0 and evaluated for UGP2 editing across CNS tissues at 1, 4, 8, 12, and 16-weeks post dose. Percentage UGP2 editing determined by Sanger sequencing. Stats: 2-way ANOVA compared to PBS (n=5 per time point per treatment) *P<0.05, **P<0.01, ***P<0.001, ****P<0.0001. ICV intracerebroventricular; PBS phosphate buffered saline

UGP2 AIMer-1 distributes throughout CNS

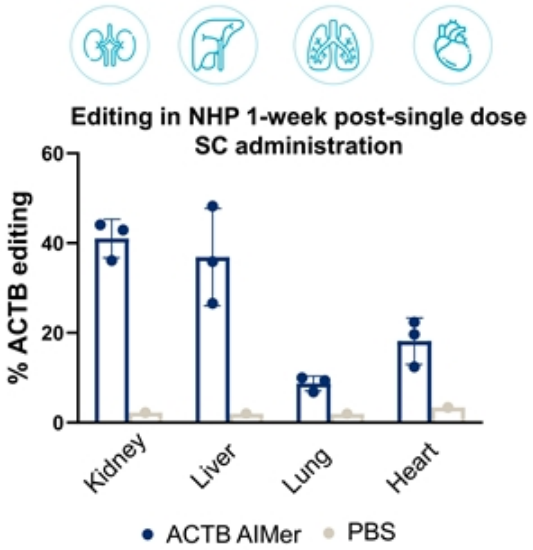


Sections from treated mice 12-weeks after a single 100 μg dose of UGP2-AIMer or PBS (bottom). ViewRNA (red, Fast red) was used to detect oligonucleotides; sections are counterstained with hematoxylin (blue nuclei). Magnification 10X (top & bottom), 40X (middle, oil), 10X

Achieving productive editing in multiple NHP tissues with unconjugated systemic AIMer delivery

- ✓ GalNAc-conjugated *(Targeted - subcutaneous)*
- ✓ Unconjugated *(Local – IVT, IT)*
- ✓ **Unconjugated *(Systemic)***

- NHP study demonstrated productive editing in kidney, liver, lung and heart with single subcutaneous dose



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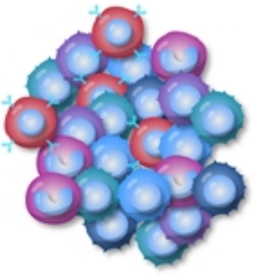
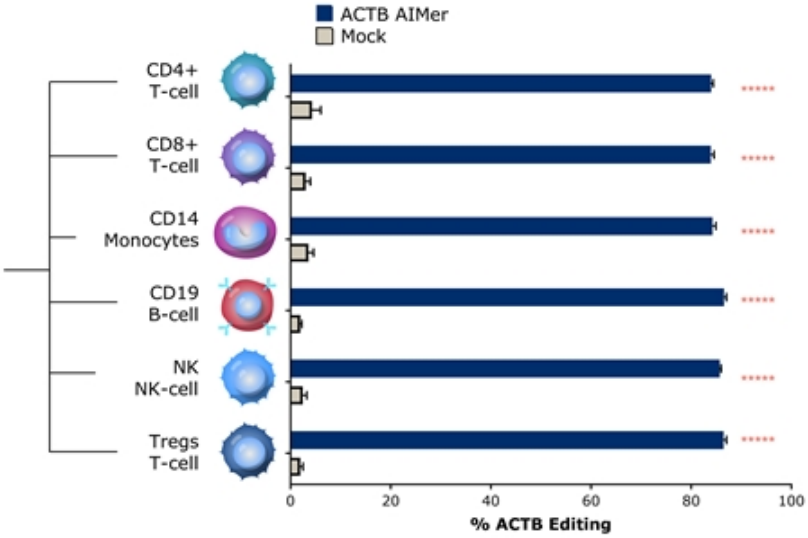
NHP: non-human primate; ACTB: Beta-actin
Dose: 50 mg/kg SC on Day 1 Necropsy for mRNA (ACTB Editing) Day 8

Achieving productive editing in multiple immune cell types with AIMers *in vitro*

Human peripheral blood mononuclear cell (PBMC)



Activate (PHA) → Dose → Sort



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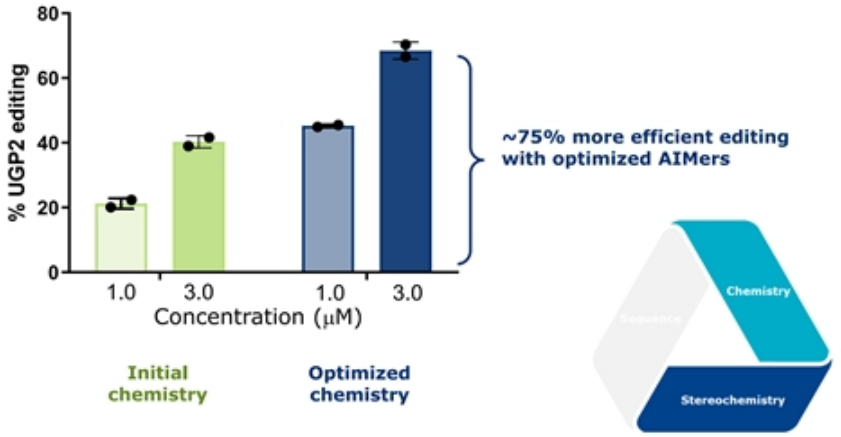
Human PBMCs dosed with 10 uM ACTB AIMers, under activating conditions (PHA). After 4 days, different cell types isolated, quantitated for editing %. ACTB: Beta-actin; Two-way ANOVA followed by post hoc comparison per cell line. P values were Bonferroni-corrected for multiple hypotheses

Ongoing chemistry optimization continues to drive potency gains

***In vitro* dose-response in iCell neurons**



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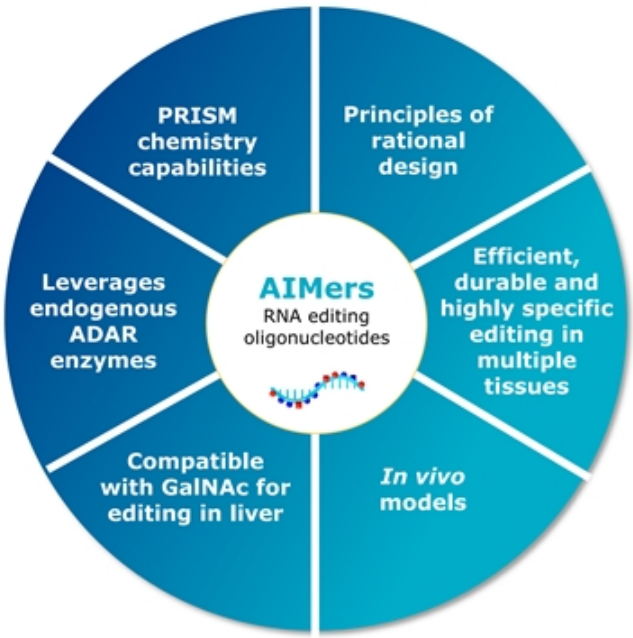
iCell neurons treated with 1 or 3 mM UGP2 AIMer with old (left, blue) or new (right, green) chemistry. Data are mean ± sd.

Rapidly advancing best-in-class ADAR editing capability



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Advancing ADAR Editing in the CNS

Ken Rhodes, PhD

SVP, Therapeutics Discovery

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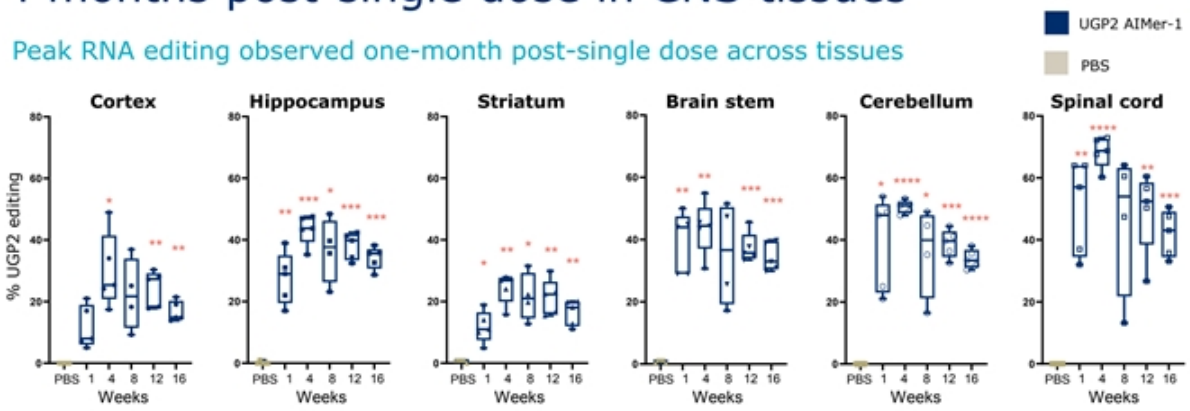
Substantial *in vivo* RNA editing out to at least 4 months post-single dose in CNS tissues

Peak RNA editing observed one-month post-single dose across tissues



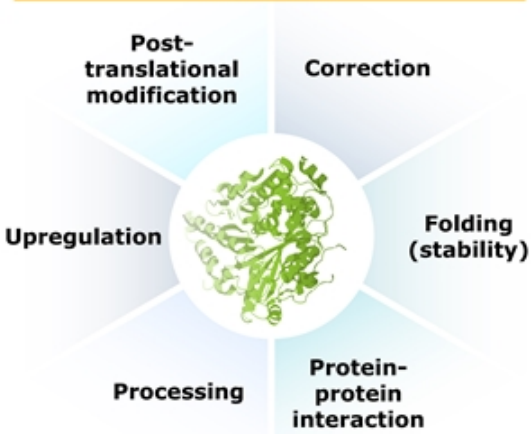
Peak editing	**30%**	**>40%**	**25%**	**>40%**	**50%**	**>65%**

Transgenic huADAR mice administered 100 μg AIMer or PBS on day 0 and evaluated for UGP2 editing across CNS tissues at 1, 4, 8, 12, and 16-weeks post dose. Percentage UGP2 editing determined by Sanger sequencing. Stats: 2-way ANOVA compared to PBS (n=5 per time point per treatment) *P<0.05, **P<0.01, ***P<0.001, ****P<0.0001. ICV intracerebroventricular; PBS phosphate buffered saline

Expanding addressable disease target space using ADAR editing to modulate proteins

ADAR editing of mRNA

Restore or modify protein function



Impact diseases

Examples:

- Familial epilepsies
- Neuropathic pain
- Neuromuscular disorders
- Dementias
- Haploinsufficient diseases
- Loss of function

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Correct a nonsense mutation using ADAR editing to restore protein expression and function

ADAR editing of mRNA

Downstream protein interactions

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RNA editing of nonsense mutation found in MECP2 (Rett Syndrome) restores functional protein

Normal: ... CGA... wild type protein
Rett Syndrome: ... TGA... premature stop codon
ADAR editing: ... TGG... restored protein

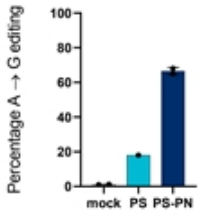
Variant base

ADAR editing site

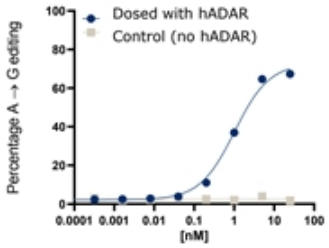
Nonsense mutations found in Rett Syndrome can occur in multiple locations on RNA transcript:

in vitro ADAR editing of over 60% targeting MECP2 disease transcript

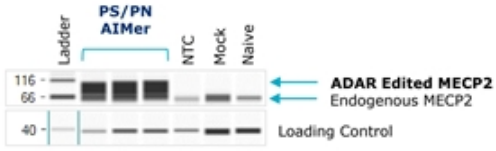
PN chemistry improved editing efficiency *in vitro*



Dose-dependent RNA editing of MECP2 mutation with PS/PN AIMer



Full length MECP2 protein is expressed following ADAR editing



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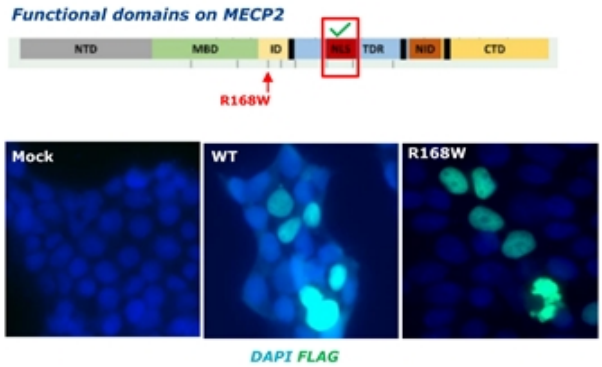
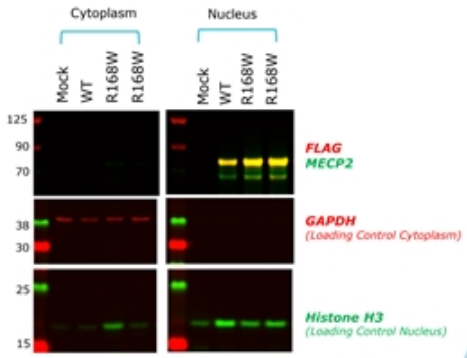
293T cells transfected with both nonsense mutation on MECP2 (GFP-fusion construct) and ADAR plasmids. AIMers transfected for 48h prior to RNA extraction and sequencing. Percentage editing determined by Sanger sequencing. Left: Single dose (25nM) treatment Middle: Full dose response curve (25nM, 5-fold dilution, 48h treatment) in presence or absence of hADAR Right: Western blot for MECP2 protein. Three biological replicates, NTC AIMer, mock and naive 293T cells probed for fusion protein.

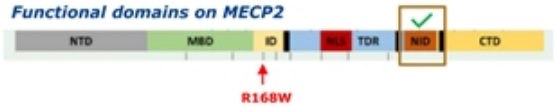
Restored MECP2 retains proper nuclear localization

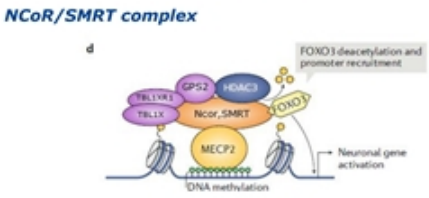


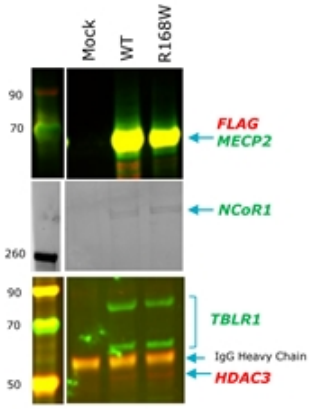


293T cells transfected with plasmids containing either wildtype or R168W MECP2 (FLAG-fusion construct) for 72hr. Left: Immunofluorescence staining with anti-FLAG monoclonal antibody (green), nuclei were counterstained with DAPI (blue). Right: Western blot analysis of cellular fractionation to isolate cytoplasm and nucleus. Mock, WT MECP2, and two biological replicates of MECP2 (R168W) transfected 293T cells probed for FLAG-tagged MECP2 protein.

Restored MECP2 binds to coregulatory proteins and recruits HDAC3, further suggesting functional restoration







NCoR1 - Transcriptional coregulatory proteins that facilitates the recruitment of HDAC3 to DNA promoter regions
TBLR1 - Scaffold protein facilitating assembly of multi-protein complexes
HDAC3 – Histone deacetylase that removes acetyl group from histones, allowing histones to wrap DNA more tightly and suppress target gene expression

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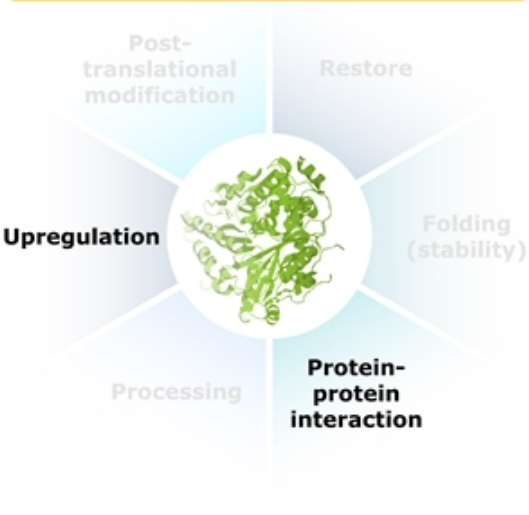
293T cells transfected with plasmids containing either wildtype or R168W MECP2 (FLAG-fusion construct). Transfected for 72h prior to nuclear extraction and immunoprecipitation with anti-FLAG tagged magnetic beads. Right: Western blot analysis of immunoprecipitation eluates probed for FLAG-tagged MECP2 protein and NCoR1/SMRT complex.

ADAR editing to modulate protein-protein interactions: upregulating gene expression

ADAR editing of mRNA

Downstream protein interactions



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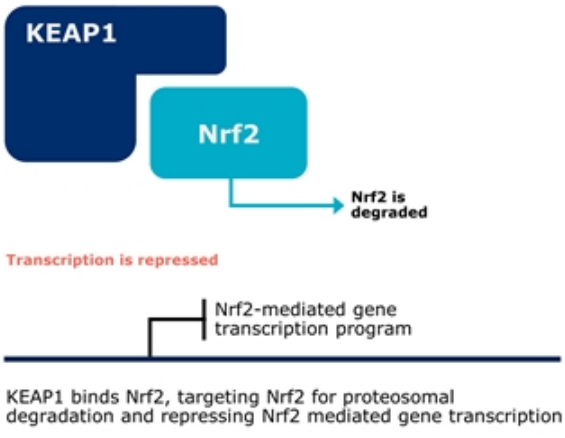
ADAR to modify protein-protein interactions

Basal conditions



KEAP1 binds Nrf2, targeting Nrf2 for proteosomal degradation and repressing Nrf2 mediated gene transcription

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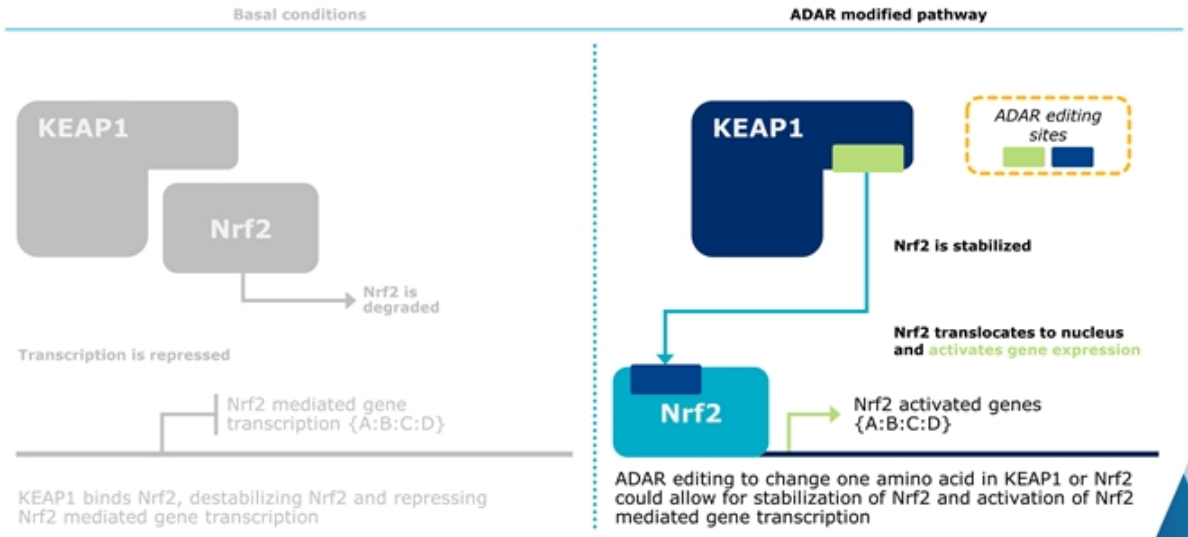
ADAR to modify protein-protein interactions



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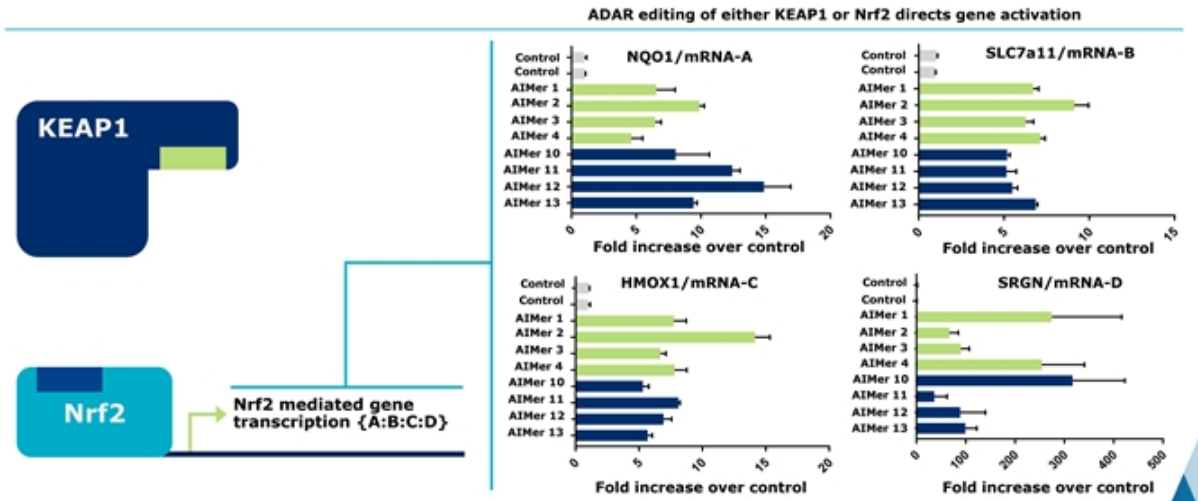
ADAR editing alters multiple amino acids on two different proteins *in vitro*

293T cells transfected with 20nM of AIMer, ADAR-p110 or ADAR-p150 plasmid. RNA collected 48h, editing quantified by PCR and Sanger (n=2).

ADAR editing activates multiple genes confirming disrupted protein-protein interaction *in vitro*



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Gene expression quantified by PCR (n=2)

ADAR editing expands target universe in CNS

- PN chemistry expands addressable CNS disease target space, enabling protein restoration and protein modulation by leveraging shared learnings across ADAR programs
  - Editing of UGP2 *in vivo* in CNS tissues is durable out to 4 months
  - Discovery-stage MECP2 program for Rett Syndrome demonstrates restoration of functional MECP2 protein with ADAR editing *in vitro* to correct nonsense mutation
  - Disrupting protein–protein interactions enables access to new mechanisms

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Restoring Functional AAT Protein with ADAR Editing: Program Update

Paloma Giangrande, PhD

VP, Platform Discovery Sciences

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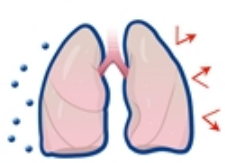
Leading RNA editing program provides optimal approach for treatment of AATD

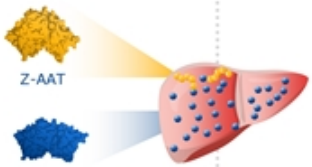
1) Restore circulating, functional wild-type M-AAT

2) Reduce Z-AAT protein aggregation in liver

3) Retain M-AAT physiological regulation

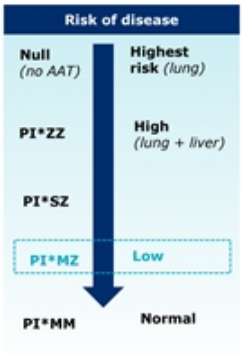


M-AAT reaches lungs to protect from proteases



Wild-type M-AAT protein replaces Z-AAT with RNA correction

M-AAT secretion into bloodstream



Wave ADAR editing approach addresses all goals of treatment

GalNAc-conjugated for subcutaneous delivery

~200K people in US and EU with mutation in *SERPINA1* Z allele (PI*ZZ)

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AAT: Alpha-1 antitrypsin; Sources: Strnad 2020; Blanco 2017

Today's update on AATD program

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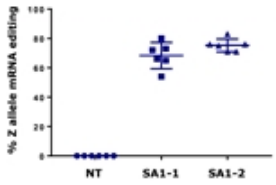
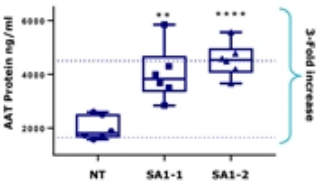
Focused on restoring wild-type M-AAT *in vivo*

In vitro proof of concept

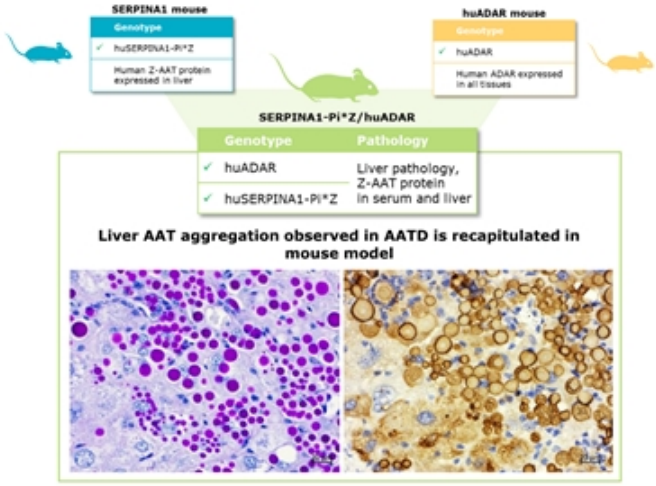
SERPINA1 Z allele mRNA editing



AAT protein concentration in media



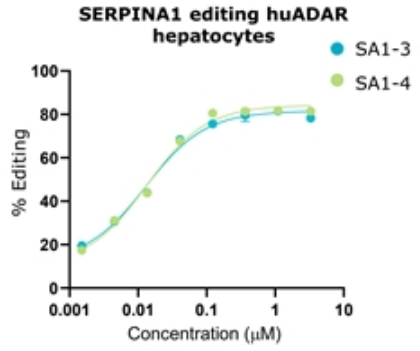
In vivo proof of concept

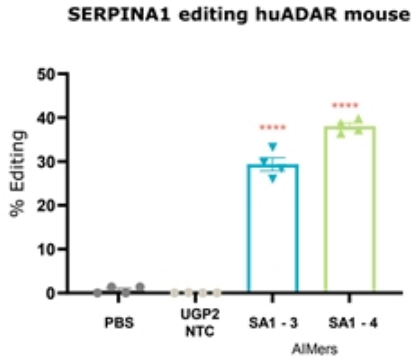


AATD: Alpha-1 antitrypsin deficiency, Z-AAT: mutated protein, M-AAT: wild-type human AAT protein
(Left) Hematoxylin and PAS stain and (Right) immunohistochemistry for AAT protein with hematoxylin counterstain in the huADAR/AATD mouse liver

Achieving 40% editing of Z allele mRNA at single time point

SERPINA1 Z allele mRNA editing levels nearing correction to heterozygote (MZ)





- GalNAc-conjugated compounds
- Up to 40% editing of Z allele mRNA in liver of transgenic human ADAR mice at day 7

Z allele mRNA editing *in vivo* | **AAT protein increase** | **Wild-type M-AAT functional**

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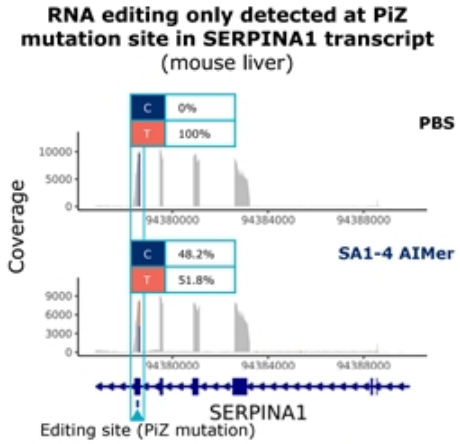
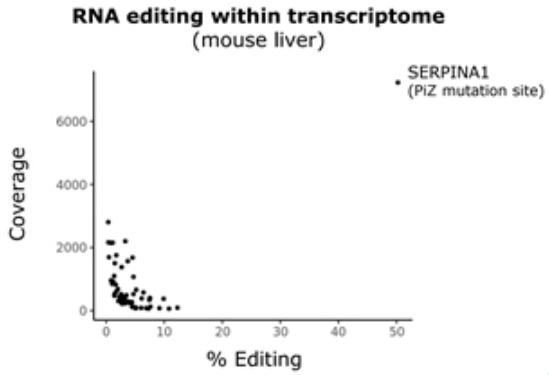
hu*ADAR/SERPINA1* mice administered PBS or 3 x 10 mg/kg AIMer (days 0, 2, and 4) SC. Samples collected day 7. Stats: One-way ANOVA; NTC: non-targeting control

ADAR editing is highly specific; no bystander editing observed on SERPINA1 transcript





Highly specific Z allele mRNA editing *in vivo*

AAT protein increase

Wild-type M-AAT functional

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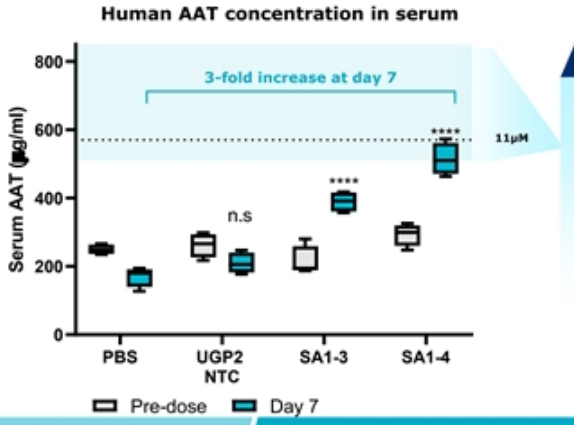
Mice were at dosed at 3 x 10mg/kg on days 0, 2, and 4 SC. Liver biopsies were collected on day 7. RNAseq was conducted using strand-specific libraries. To quantify on-target SERPINA1 editing reads were mapped to human SERPINA1 and to quantify off-target editing reads were mapped to entire mouse genome; plotted circles represent sites with LOD>3 (N=4)

PRISM

Achieving therapeutically meaningful increases in circulating human AAT protein

3-fold increase in circulating human AAT as compared to PBS at initial timepoint



AAT serum levels by genotype

PI*MZ	~3 to 5-fold increase
PI*SZ	~2-fold increase
PI*ZZ	~3 – 7 uM

✓ Z allele mRNA editing *in vivo* | ✓ AAT protein increase | Wild-type M-AAT functional

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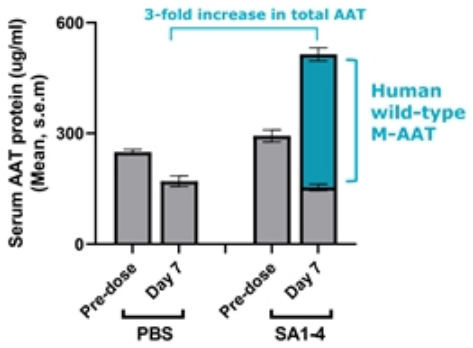
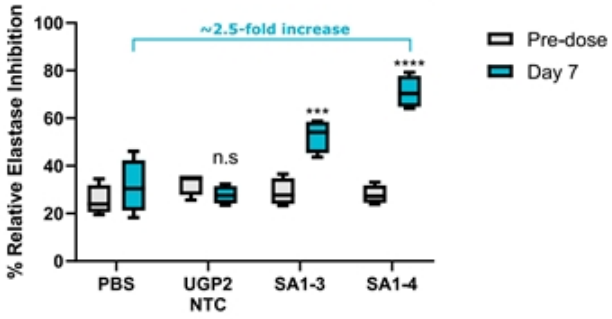
Statistics (ELISA): Matched 2-way ANOVA with correction for multiple comparisons (Bonferroni) was used to test for differences in AAT abundance in treated samples compared to PBS Statistics; de Serres et al., J Intern Med. 2014; NTC: non-targeting control

ADAR editing restores circulating, functional M-AAT

Wild-type M-AAT detected with ADAR editing



Significant increase in neutrophil elastase inhibition with ADAR editing



✓ Z allele mRNA editing *in vivo* | ✓ AAT protein increase | ✓ Wild-type M-AAT functional

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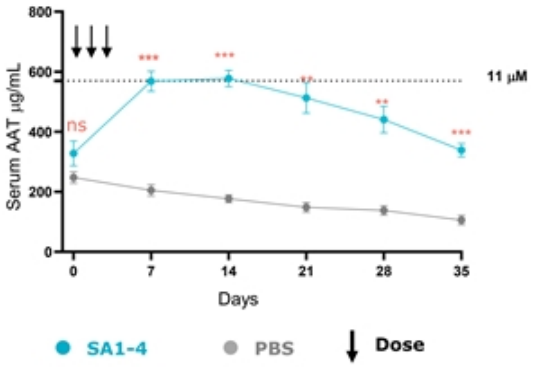
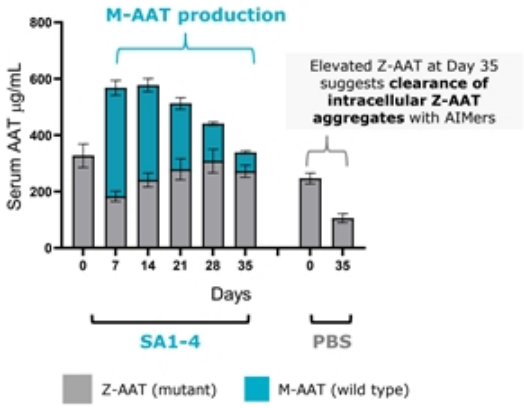
Left: Mass spectrometry and ELISA Right: (Elastase inhibition): Matched 2-way ANOVA with correction for multiple comparisons (Bonferroni) was used to test for differences in elastase inhibition activity in serum collected at day 7 vs pre-dose for each treatment group; NTC: non-targeting control

Increase in circulating human AAT is durable, with restored M-AAT detected one month post last dose

Human AAT serum concentration ≥3-fold higher over 30 days post-last dose



Restored wild-type M-AAT detected over 30 days post-last dose



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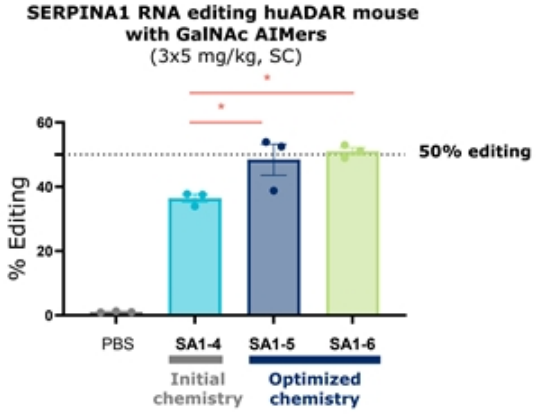
SA1-4: GalNAc AIMer (Left) hu*ADAR/SERPINA1* mice administered PBS or 3 x 10 mg/kg AIMer (days 0, 2, and 4) SC. AAT levels quantified by ELISA. Data presented as mean ± sem. Stats: Matched 2-way ANOVA ns nonsignificant, ** P<0.01, *** P<0.001. (Right) Proportion of AAT in serum, Z type (mutant) or M type (wild type), measured by mass spectrometry, total AAT levels quantified by ELISA

Optimization further improves potency

50% mean editing observed with half dose in mice at Day 7



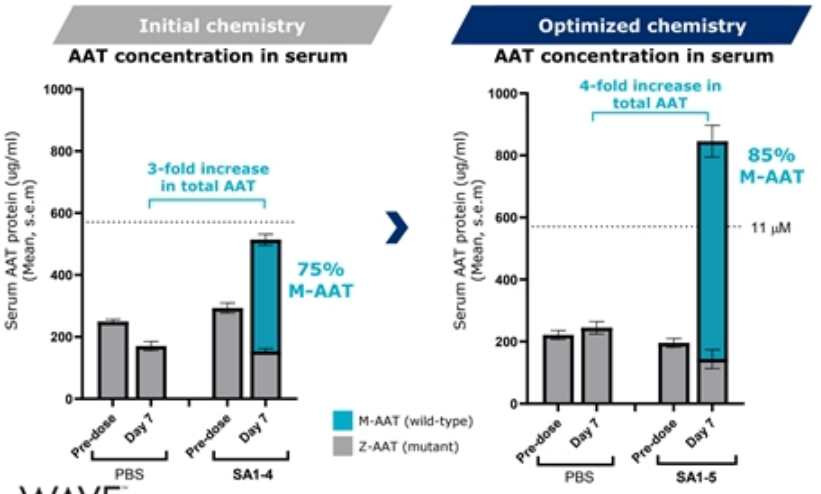
Chemistry optimization:

- ✓ Increases Z allele mRNA editing efficiency

AIMers administered hu*ADAR/SERPINA1* mice (3x5 mg/kg) on days 0, 2, and 4. Livers collected on day 7, and SERPINA1 editing was quantified by Sanger sequencing (shown as mean ±. sem) Stats: One-way ANOVA was used to test for differences in editing between SA1-4 and other oligos * P<0.05

Optimization further improves M-AAT restoration

4-fold increase in AAT protein (>15uM) relative to PBS at Day 7 with optimized AIMer



Chemistry optimization:

- ✓ Increases Z allele mRNA editing efficiency
- ✓ Higher fold-increase in circulating AAT protein relative to control
- ✓ Greater percentage of restored wild-type M-AAT protein relative to total AAT

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hu*ADAR/SERPINA1* mice administered PBS or 3 x 10 mg/kg AIMer (days 0, 2, and 4) SC. Proportion of AAT protein in serum, Z type or M type, measured by mass spectrometry, total AAT protein levels quantified by ELISA.

AATD development candidate expected in 2022

Demonstrate restoration of functional M-AAT

- ✓ GalNAc-conjugated AIMers restore therapeutically meaningful levels of functional, wild-type M-AAT

Assess specificity and duration of effect

- ✓ ADAR editing is highly specific
- ✓ Restored, circulating wild-type M-AAT in serum at 1-month post-last dose

Chemistry optimization to improve potency

- ✓ Chemistry optimization of AIMers further increases potency
- ✓ Optimized AIMers restore AAT in serum by 4-fold (>15uM) at Day 7
- ✓ Restored wild-type M-AAT at 85% of total AAT

Path to development candidate

- Ongoing and planned preclinical studies assessing durability, dose response and PK/PD
- Assessment of reduction in Z-AAT aggregates and changes in liver pathology

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Closing Remarks

Paul Bolno, MD, MBA

President and CEO

WAVE LIFE SCIENCES

Q&A

Dr. Paul Bolno
President and
Chief Executive Officer

Dr. Chandra Vargeese
Chief Technology Officer

Dr. Ken Rhodes
SVP, Therapeutics Discovery

Dr. Paloma Giangrande
VP, Platform Discovery
Sciences, Biology

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Realizing a brighter future for people affected by genetic diseases

For more information:
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Wave Life Sciences Announces New Data for Leading RNA Editing Capability Across Multiple Tissues and Provides Update on AATD Program During Analyst and Investor Research Webcast

Durable ADAR editing in vivo in preclinical models, including in CNS tissues with editing out to at least four months

Chemistry optimization yields a four-fold increase over PBS control in AAT protein restoration in vivo preclinically (or more than 15 micromolar)

CAMBRIDGE, Mass., September 28, 2021 – Wave Life Sciences Ltd. (Nasdaq: WVE), a clinical-stage genetic medicines company committed to delivering life-changing treatments for people battling devastating diseases, today presented new data for its ADAR-mediated RNA editing capability (ADAR editing), including new preclinical editing data across multiple tissues, as well as an update on its discovery-stage alpha-1 antitrypsin deficiency (AATD) program during the company's 2021 Analyst and Investor Research Webcast. The webcast also included updates on the company's PRISM™ platform and initial results from the application of ADAR editing to neurology targets.

"Our presentations today represent robust and meaningful contributions to the rapidly advancing field of RNA editing, where we are at the forefront defining both new levels of editing, as well as the tissues and cell types amenable to this approach," said Chandra Vargeese, PhD, Chief Technology Officer of Wave Life Sciences. "The application of PRISM to RNA editing means that there is the potential for therapeutic applications extending beyond the restoration of protein function, such as upregulation of protein expression, modification of protein function by altering post-translational modifications or protein-protein interactions, or alteration of protein stability. Additionally, with our AATD program, we have shown an ability in preclinical experiments to drive alpha-1 antitrypsin protein significantly above levels that are potentially therapeutically meaningful and increase the overall percentage of secreted wild-type M-AAT protein."

A summary of the RNA editing presentations is below. A replay of the Analyst and Investor Research Webcast is available on Wave's Investor Relations website.

Leading RNA Editing Capability Using Endogenous ADAR

- Wave's RNA editing capability leverages widely expressed endogenous ADAR enzymes to achieve highly specific A-to-I (G) RNA editing using stereopure oligonucleotides, called "AIMers," without the need for lipid nanoparticles (LNPs) or viral vectors, and without altering the genome.
- Wave is developing short, fully-chemically modified AIMers with and without GalNAc conjugation, with the objective of achieving productive editing in the liver, central nervous system (CNS), and other tissues.
- **CNS**: Wave presented new *in vivo* data that demonstrated potent editing (up to 65%) and durable editing of UGP2 mRNA out to at least four months in multiple regions of the CNS in a mouse model with human ADAR.
    - Wave is applying ADAR editing to multiple therapeutic targets in the CNS, including MECP2, seeking to correct a nonsense mutation and potentially restore functional protein in Rett Syndrome.
    - Additionally, *in vitro* data were presented demonstrating the potential to target protein-protein interactions and upregulate downstream gene expression with AIMers.

- **Ophthalmology**: Wave also presented preclinical data demonstrating up to 50% editing of UGP2 mRNA in the posterior of the eye of mice at one-month post-single intravitreal injection.
- **New tissue and cell types:** Wave shared ACTB RNA editing in non-human primates (NHPs) using systemic administration, including in the kidneys, liver, lungs and heart, as well as editing of ACTB in multiple immune cell types *in vitro*, including CD4+ T-cells, CD8+ T-cells, and others.

ADAR Editing Provides Promising Treatment Approach for Alpha-1 Antitrypsin Deficiency (AATD)

- Wave's AATD program, its first investigational ADAR editing program, uses AIMers to potentially correct the single base mutation in mRNA coded by the *SERPINA1* Z allele. ADAR editing may provide an ideal approach for addressing AATD by increasing circulating levels of functional alpha-1 antitrypsin (M-AAT) protein and reducing mutant protein aggregation in the liver, thus potentially addressing both the lung and liver manifestations of the disease.
- Today Wave shared new *in vivo* data demonstrating durable restoration of M-AAT protein in the liver of transgenic mice with human *SERPINA1* and human ADAR following initial doses of a GalNAc-conjugated SERPINA1 AIMer. Serum concentrations of human AAT protein remained at least three-fold higher over PBS control for 30 days post-last dose with the SERPINA1 AIMer.
- Wave also shared data demonstrating progress in enhancing editing activity and protein restoration following PRISM chemistry optimization.
    - These AIMers achieved mean editing of approximately 50% of SERPINA1 mRNA *in vivo*.
    - Also with chemistry optimization, Wave demonstrated *in vivo* a four-fold increase over PBS control in AAT protein restoration in serum (or more than 15 micromolar), representing an improvement over the three-fold increase achieved with Wave's initial AIMers. Approximately 85% of circulating AAT was confirmed to be M-AAT in treated transgenic mice with human *SERPINA1* and human ADAR.
- Wave's ADAR editing appears highly specific with nominal off-target edits observed following transcriptome analysis, nor were there bystander edits observed in the SERPINA1 transcript.
- Ongoing and planned preclinical studies are assessing durability, dose response, pharmacokinetics, and pharmacodynamics. Wave also plans to assess reduction of Z-AAT aggregates in the liver and changes in liver pathology in its transgenic mouse model.
- Wave expects to have an AATD development candidate in 2022.

About PRISM™

PRISM™ is Wave Life Sciences' proprietary discovery and drug development platform that enables genetically defined diseases to be targeted with stereopure oligonucleotides across multiple therapeutic modalities, including silencing, splicing and editing. PRISM combines the company's unique ability to construct stereopure oligonucleotides with a deep understanding of how the interplay among oligonucleotide sequence, chemistry and backbone stereochemistry impacts key pharmacological properties. By exploring these interactions through iterative analysis of *in vitro* and *in vivo* outcomes and machine learning-driven predictive modeling, the company continues to define design principles that are deployed across programs to rapidly develop and manufacture clinical candidates that meet pre-defined product profiles.

About Wave Life Sciences

Wave Life Sciences (Nasdaq: WVE) is a clinical-stage genetic medicines company committed to delivering life-changing treatments for people battling devastating diseases. Wave aspires to develop best-in-class medicines across multiple therapeutic modalities using PRISM™, the company's proprietary discovery and drug development platform that enables the precise design, optimization, and production of stereopure oligonucleotides. Driven by a resolute sense of urgency, the Wave team is targeting a broad range of genetically defined diseases so that patients and families may realize a brighter future. To find out more, please visit www.wavelifesciences.com and follow Wave on Twitter @WaveLifeSci.

Forward-Looking Statements

This press release contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, as amended, including, without limitation, our understanding of the application of PRISM to RNA editing and the anticipated therapeutic benefits of RNA editing capabilities using endogenous ADAR; our beliefs regarding the learnings gained from our first-generation clinical programs and our initial chemistry; our understanding of AIMers and their expected capabilities; the anticipated therapeutic benefits of our ADAR editing program for AATD; the anticipated timing for our AATD development candidate; and the potential benefits of PRISM, including our stereopure oligonucleotides. The words "may," "represent," "expect," "plan," "objective," "achieve," "demonstrate," "represent," "predict," "appear," "potential," "continue," "target" and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Any forward-looking statements in this press release are based on management's current expectations and beliefs and are subject to a number of risks, uncertainties and important factors that may cause actual events or results to differ materially from those expressed or implied by any forward-looking statements contained in this press release and actual results may differ materially from those indicated by these forward-looking statements as a result of these risks, uncertainties and important factors, including, without limitation, the risks and uncertainties described in the section entitled "Risk Factors" in Wave's most recent Annual Report on Form 10-K filed with the Securities and Exchange Commission (SEC), as amended, and in other filings Wave makes with the SEC from time to time. Wave undertakes no obligation to update the information contained in this press release to reflect subsequently occurring events or circumstances.

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