Wave Life Sciences

J.P. Morgan Healthcare Conference

January 13, 2026

Forward-looking statements

This document contains forward-looking statements. All statements other than statements of historical facts contained in this document, including statements regarding possible or assumed future results of operations, preclinical and clinical studies, business strategies, research and development plans, collaborations and partnerships, regulatory activities and timing thereof, competitive position, potential growth opportunities, use of proceeds and the effects of competition are forward-looking statements. These statements involve known and unknown risks, uncertainties and other important factors that may cause the actual results, performance or achievements of Wave Life Sciences Ltd. (the "Company") to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. In some cases, you can identify forward-looking statements by terms such as "may," "will," "should," "expect," "plan," "aim," "anticipate," "could," "intend," "target," "project," "contemplate," "believe," "estimate," "predict," "potential" or "continue" or the negative of these terms or other similar expressions. The forward-looking statements in this presentation are only predictions. The Company has based these forward-looking statements largely on its current expectations and projections about future events and financial trends that it believes may affect the Company's business, financial condition and results of operations. These forward-looking statements speak only as of the date of this presentation and are subject to a number of risks, uncertainties and assumptions, including those listed under Risk Factors in the Company's Form 10-K and other filings with the SEC, some of which cannot be predicted or quantified and some of which are beyond the Company's control. The events and circumstances reflected in the Company's forward-looking statements may not be achieved or occur, and actual results could differ materially from those projected in the forward-looking statements. Moreover, the Company operates in a dynamic industry and economy. New risk factors and uncertainties may emerge from time to time, and it is not possible for management to predict all risk factors and uncertainties that the Company may face. Except as required by applicable law, the Company does not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise.

WAVE® LIFE SCIENCES

Our Mission

To unlock the broad potential of RNA medicines to transform human health

Building a leading RNA medicines company

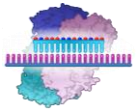
Differentiated RNA medicines platform and chemistry

PRISM

- **Proprietary chemistry**
- Leveraging deep insights in **human genetics**
- Strong and **broad IP**
- **In-house GMP** manufacturing

Translating genetic insights into potentially best-in-class medicines

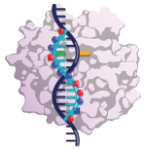
RNAi



WVE-007 (obesity)

- Differentiated mechanism focused on fat loss and muscle preservation

RNA editing



WVE-006 (AATD)
WVE-008 (liver disease)

- Restoration of functional protein production

Other modalities: **DMD** and **HD** clinical programs

Unlocking emerging pipeline

- **Extra-hepatic capabilities:** with RNAi and RNA editing
- **Novel bifunctional modality:** simultaneously edit and silence with single oligonucleotide construct

Well capitalized with ~$602 million[1] and expected cash runway into 3Q 2028

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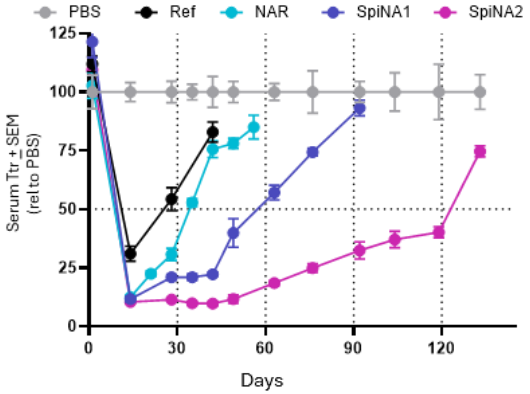
[1] Preliminary, unaudited cash and cash equivalents of ~$602 million are as of December 31, 2025. These preliminary, unaudited results are subject to adjustment. Wave expects to report its final and complete fourth-quarter and full-year 2025 financial results in late February 2026, and the actual results could be different from these preliminary, unaudited financial results

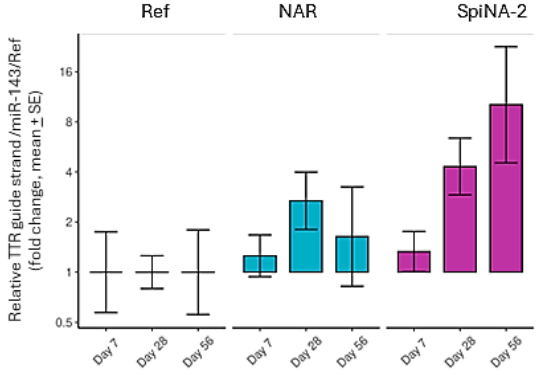
For over a decade Wave has been extending the frontiers of RNA therapies delivering breakthroughs in nucleic acid chemistry

RNAi — SpiNA

Protein reduction

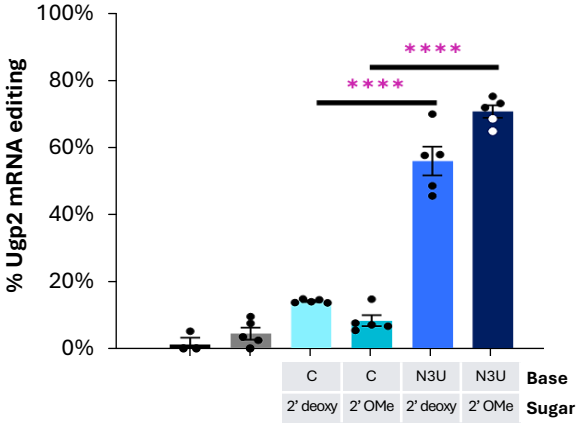


Ago2 loading



Substantial improvements in potency, duration of activity, and Ago2 loading with Wave's proprietary SpiNA design

RNA editing — AIMer



Increased RNA editing efficiency achieved with proprietary chemistry

Proprietary chemistry has dramatically increased potency and durability

Robust, diversified RNA medicines pipeline including first-in-class RNA editing and RNAi programs

Program	Discovery	IND / CTA Enabling Studies	Clinical	Rights	Patient population (US & Europe)
RNAi					
WVE-007 (GalNAc) INHBE (obesity)				100% global	175M (>1 billion globally)
GalNAc / extra-hepatic Multiple				100% global	--
RNA EDITING					
WVE-006 (GalNAc) SERPINA1 (AATD)				GSK exclusive global license	200K
WVE-008 (GalNAc) PNPLA3 (liver disease)				100% global	9M
GalNAc / extra-hepatic Multiple				100% global	--
SPLICING					
WVE-N531 Exon 53 (DMD)				100% global	2.3K
Other exons (DMD)				100% global	Up to 18K
ALLELE-SELECTIVE SILENCING					
WVE-003 mHTT (HD)				100% global	25K Symptomatic (SNP3) 60K Pre-Symptomatic (SNP3)

AATD: Alpha-1 antitrypsin deficiency; DMD: Duchenne muscular dystrophy; HD: Huntington's disease

Strategic focus for 2026: Accelerate development of WVE-007 (INHBE) and extend leadership in RNA editing

Accelerating development plan for WVE-007 (multiple treatment settings)

- Higher dose and longer follow-up data from INLIGHT Phase 1 single dose
- Initiate Phase 2a multidose portion of INLIGHT (higher BMI and comorbidities)
- Initiate new clinical trials: add-on to incretin and post-incretin maintenance

Rapidly advancing RNA editing portfolio (WVE-006 and WVE-008)

- Data from 400 mg and 600 mg cohorts of RestorAATion-2 clinical trial in AATD

Building on successful clinical translation of WVE-006

- File CTA for WVE-008 (PNPLA3) for ~nine million individuals living with liver disease

Obesity is a metabolic disease in need of a treatment paradigm shift

Individuals living with **obesity** have higher risk for many serious health conditions, including heart disease, type 2 diabetes, and some forms of cancer[1]

Current standard of care: GLP-1s

Impact of GLP-1s is often limited by:

- Loss of muscle mass[2]
- Frequent dosing[3]
- Poor tolerability[4]
- High discontinuation rates[5,6]

WVE-007

(INHBE GalNAc-siRNA)

- **Fat loss similar to GLP-1 at three months**
- **Preserves muscle**
- **Potential 1–2 per year dosing**
- **Generally safe and well-tolerated**

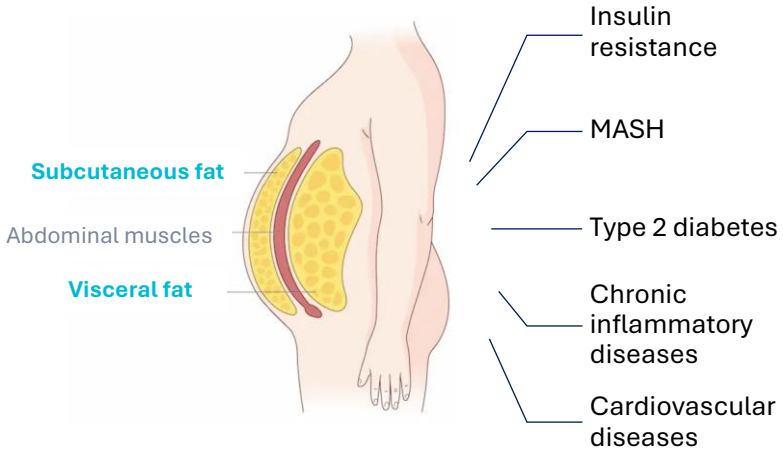
Improving body composition is the future for the > 1 billion people living with obesity globally

1. Koenen et al. 2021 Circ Res 128, 951; 2. Sargeant, et al. 2019 Endocrinol Metab (Seoul) 34, 247; 3. Wegovy PI; 4. Ghusn and Hurtado. 2024 Obesity Pillars 12, 100127; 5. Leach, et al. 2023 Prime Therapeutics Claims Analysis; 6. Gasoyan, et al. 2024 Obesity (Silver Spring) 32, 486.

Body composition improvements: Reducing fat, including visceral fat, while also preserving lean mass

Reduce fat, including visceral fat

Increased visceral adiposity is associated with many diseases including cardiometabolic disorders



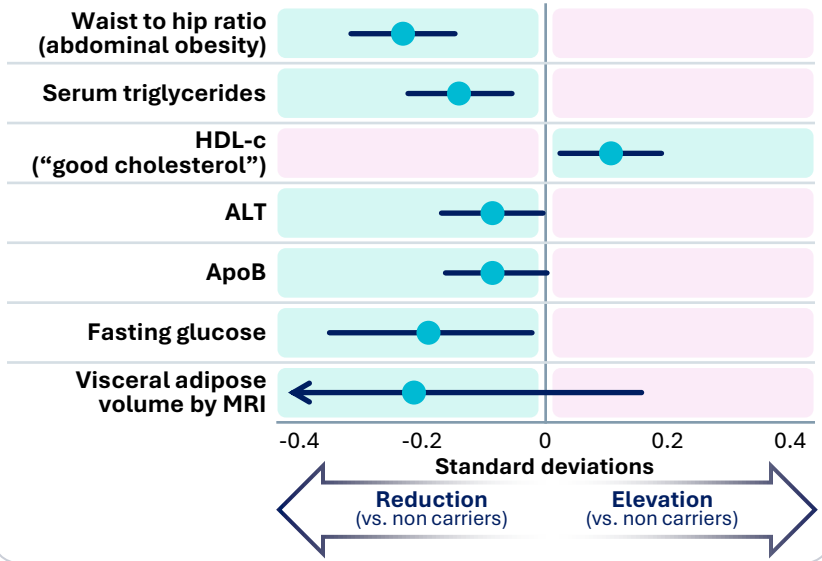
Preserve lean mass, including muscle

- Maintaining metabolic rate
- Improved insulin sensitivity
- Prevent weight regain
- Preserve muscle strength and function
- Reduce frailty

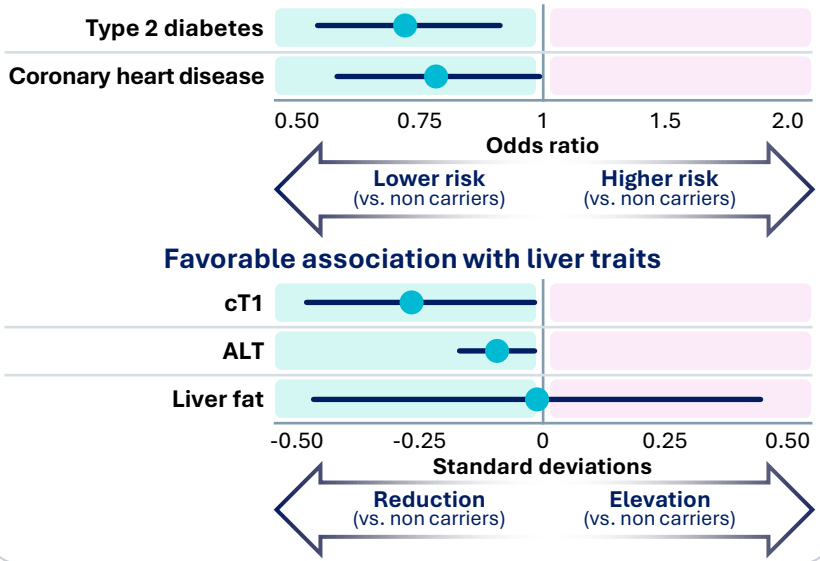
1. Liao, Y. *et al.* 2025 *Front. Nutr.* 11, 1517186; 2. Ibarra-Reynoso, L.d.R. *et al.* 2025 *J. Clin. Med.* 14, 8365; 3. Wu, Q-W., *et al.* 2025 *Front. Endocrinol.* 16, 1635282; 4. Galvan, B. *et al.* 2025 J. *Funct. Morphol. Kinesiol.* 10, 244. MASH: Metabolic Dysfunction Associated Steatohepatitis

Human genetic data demonstrate that heterozygous INHBE loss-of-function (LoF) carriers have a healthy metabolic profile

Heterozygous INHBE LoF carriers have favorable traits: lower abdominal obesity, lower triglycerides, higher HDL-c



Heterozygous INHBE LoF carriers have lower risk of Type 2 diabetes and CHD

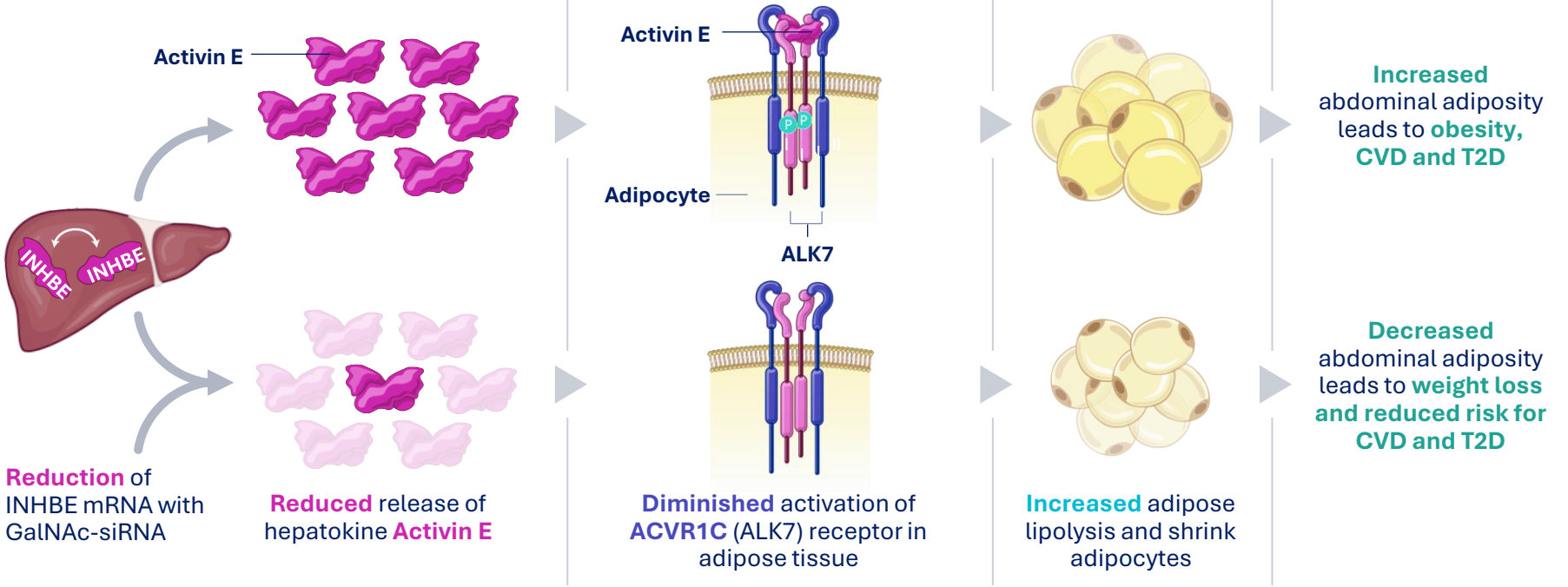


Silencing INHBE mRNA by ≥50% is expected to recapitulate the healthy metabolic profile of heterozygous INHBE LoF carriers

Akbari et al. *Nat Commun*. 2022 Aug 23;13(1):4844; Deaton et al. *Nat Commun*. 2022 Jul 27

Waist to hip ratio: waist to hip ratio adjusted for BMI; HDL-c: high-density lipoprotein cholesterol; ALT: alanine transaminase; ApoB: apolipoprotein B; CHD: coronary heart disease; cT1: corrected T1

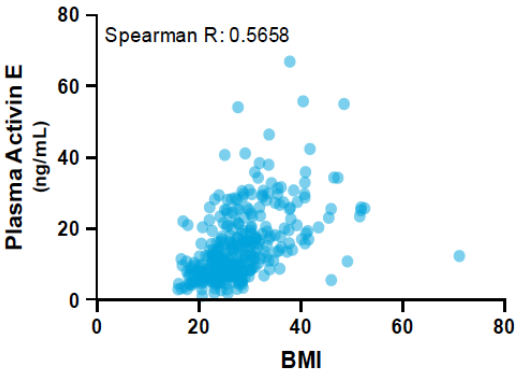
Silencing INHBE mRNA has potential to treat obesity and associated metabolic diseases



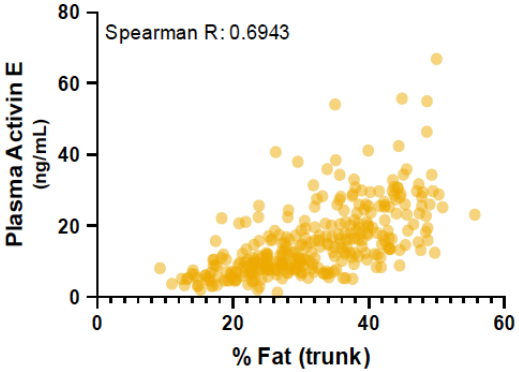
1. Cell Reports (2018) 25, 1193–1203; 2. Biochemical Journal (2024) 481 547–564; 3. PNAS 2023 Vol. 120 No. 32 e2309967120; 4. Nat Commun 2022. https://doi.org/10.1038/s41467-022-32398-7; 5. Nat Commun 2022. https://doi.org/10.1038/s41467-022-31757-8

Higher circulating Activin E levels are correlated with higher BMI, higher abdominal fat, and higher fasting insulin in non-diabetic adults

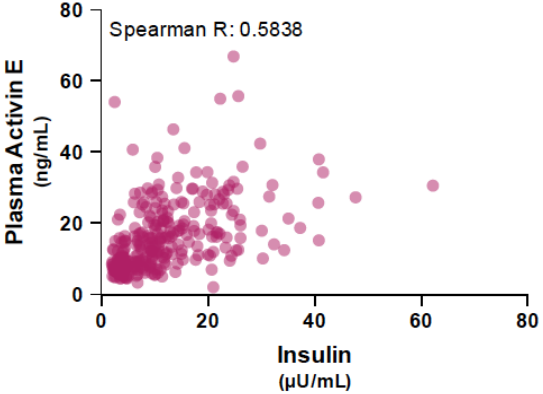
BMI



Abdominal Fat



Fasting Insulin



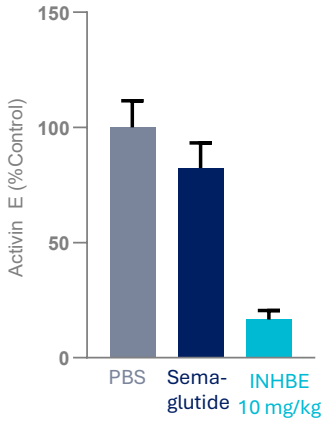
Further supports INHBE suppression as a weight loss approach for individuals living with obesity

Ref: Dubey et al. Activin E levels correlate with indicators of metabolic dysfunction in humans. ObesityWeek 2025.

Single dose of INHBE GalNAc-siRNA led to durable Activin E reductions, and sustained improvements in body composition in DIO mice

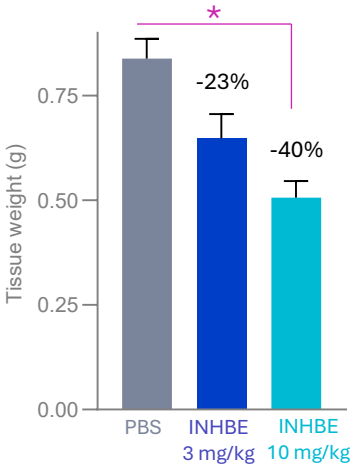
Durable Activin E reduction

Serum Activin E



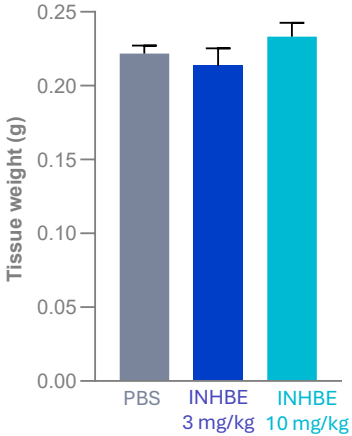
Reduction in fat

Epididymal fat weight



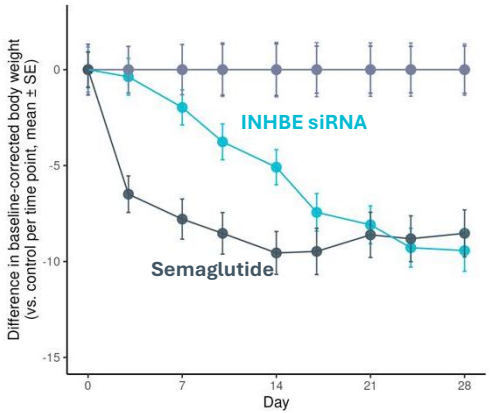
Muscle preservation

Quadricep weight



Reduction in body weight

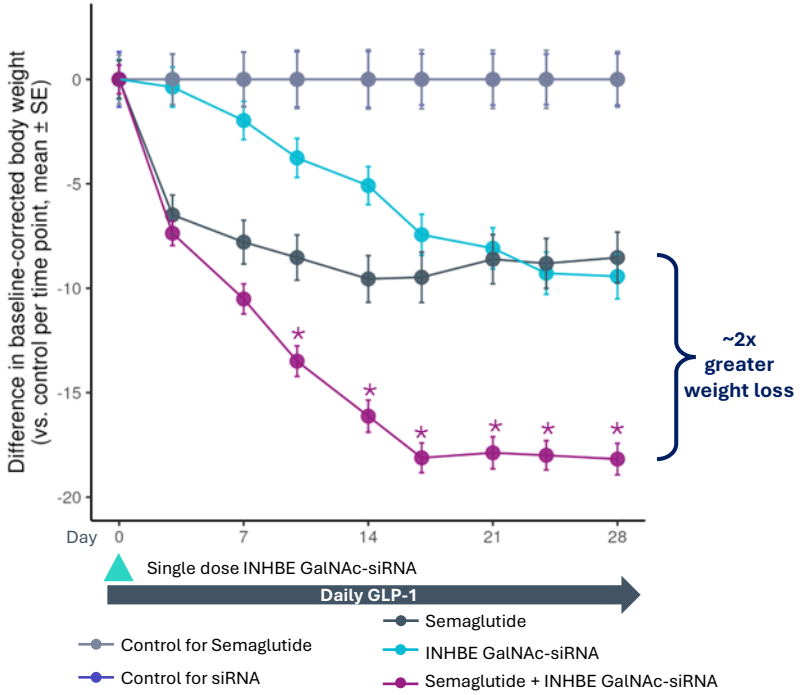
Single dose INHBE GalNAc-siRNA



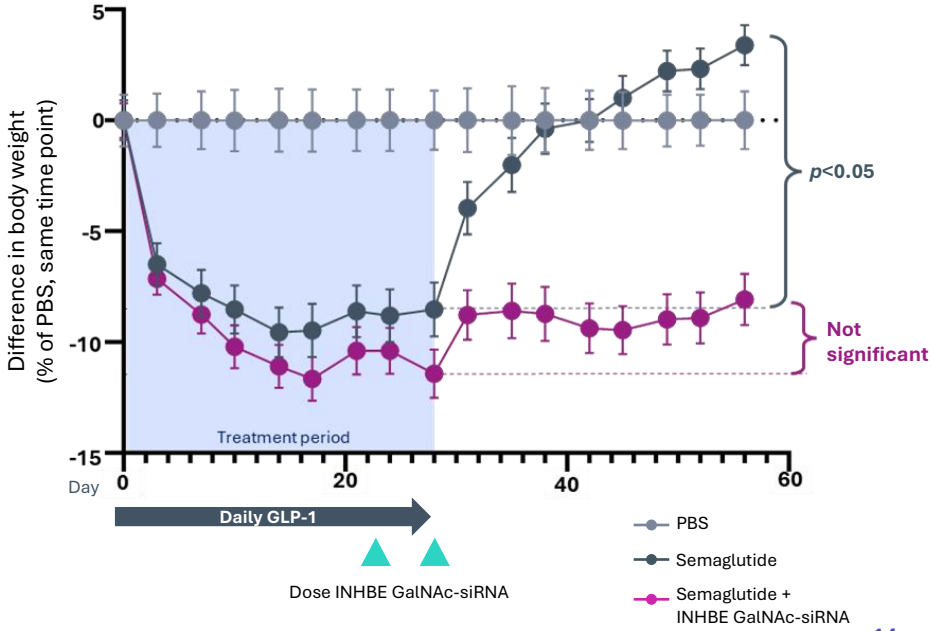
Left and right panels: Semaglutide 10 nmol/kg daily SC in mouse is equivalent to therapeutic dose of 2.4mg weekly SC in human; INHBE GalNAc-siRNA 10 mg/kg dose. All data from preclinical studies were conducted in mice fed with 60% high fat diet. Linear Mixed Effects ANOVA with post hoc comparisons of marginal treatment effects vs. PBS per tissue.* $p < 0.05$

WVE-007 has potential for use synergistically with GLP-1s or to curtail weight regain after the cessation of treatment with GLP-1, based on preclinical data

Combined with GLP-1: Greater weight loss



After cessation of GLP-1: Curtails weight regain



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Data from preclinical studies conducted in mice fed with 60% high fat diet; Left: semaglutide10 nmol/kg daily SC in mouse is equivalent to therapeutic dose of 2.4mg weekly SC in human. Left Stats: Linear Mixed Effects ANOVA with post hoc comparisons of marginal treatment effects of Semaglutide vs. Semaglutide + INHBE GalNAc-siRNA per time point * $p < 0.05$; Right Stats: Linear Mixed Effects ANOVA with post hoc comparison of Day 28 vs. Day 56 marginal effects per treatment

INLIGHT: Clinical trial of WVE-007 in otherwise healthy individuals living with overweight or obesity

Multiple clinical trial sites, including US

INLIGHT

Phase 1 SAD Portion

Key study criteria:

- HbA1c: <5.9
- BMI: 28 – 35 kg/m^2 (SAD)

No diet or exercise modifications are instituted

SAD Cohort 4
600 mg (n=32)

SAD Cohort 3
400 mg (n=32)

SAD Cohort 2
240 mg (n=32)

SAD Cohort 1
75 mg (n=8)
PK/PD and safety only (no DEXA)

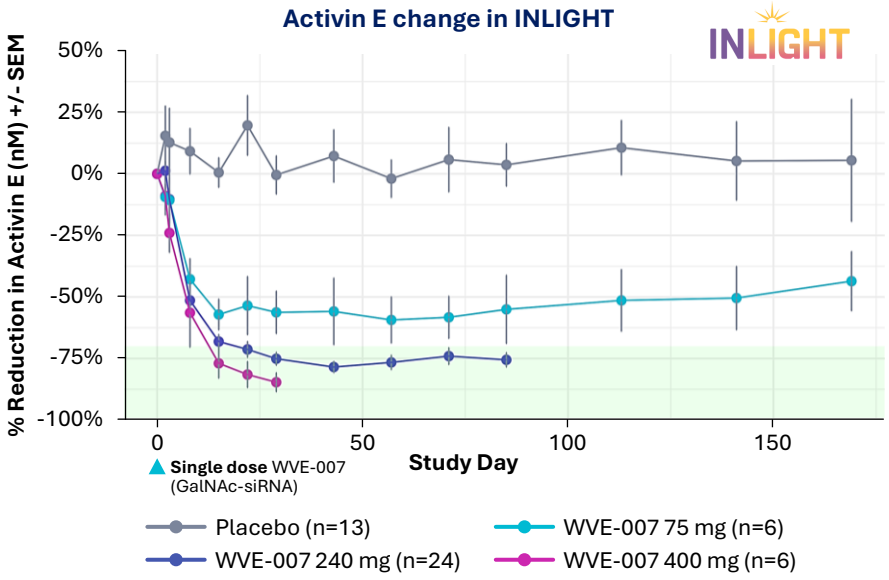
Key measurements include:

- Safety and tolerability
- PK
- Activin E
- Body composition
- Biomarkers
- Body weight

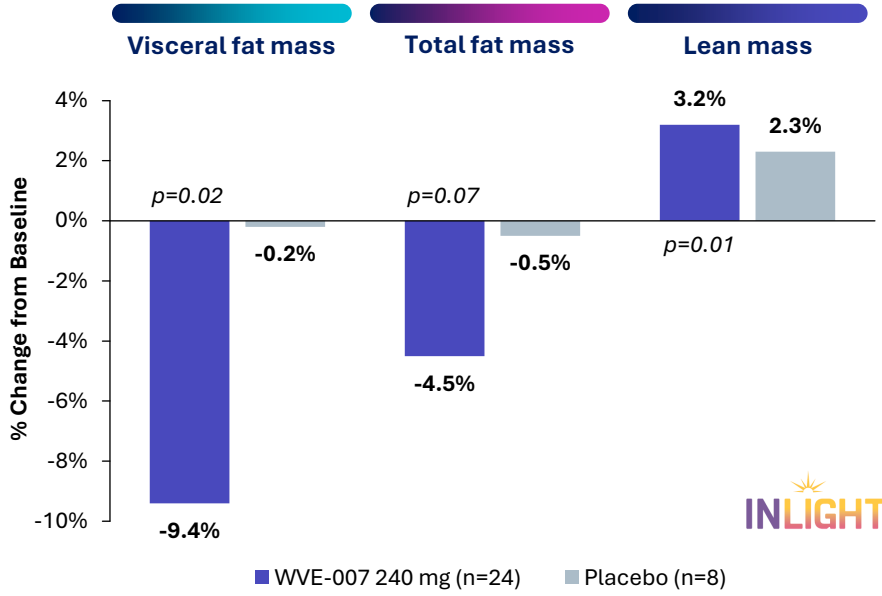
SAD: single-ascending dose; MAD: multi-ascending dose; PK: pharmacokinetics; PD: pharmacodynamics
Randomized, double-blind, placebo-controlled (3:1) study of ascending doses of WVE-007

Improvements in body composition observed at three months after a single WVE-007 dose (lowest therapeutic dose); durability supports 1-2x yearly dosing

Highly durable, dose dependent, serum Activin E reductions support dosing once or twice per year



Reductions in visceral and total fat mass with preservation of lean mass observed at three months



Left: Figure shows sample means and SEMs. All MMRM baseline and placebo comparisons from Day 8 onwards are p<0.003. Placebo includes one individual from 400 mg expansion. Green shading: >70% Activin E reductions in preclinical models led to fat loss; Right: All DEXA percentage changes and p-values are model-based, using the SAP pre-specified analysis; p-values are from tests of within-group change over time; no p values were statistically significant for placebo.

Single dose of WVE-007 led to improvements in body composition with fat loss similar to GLP-1 at three months without muscle loss

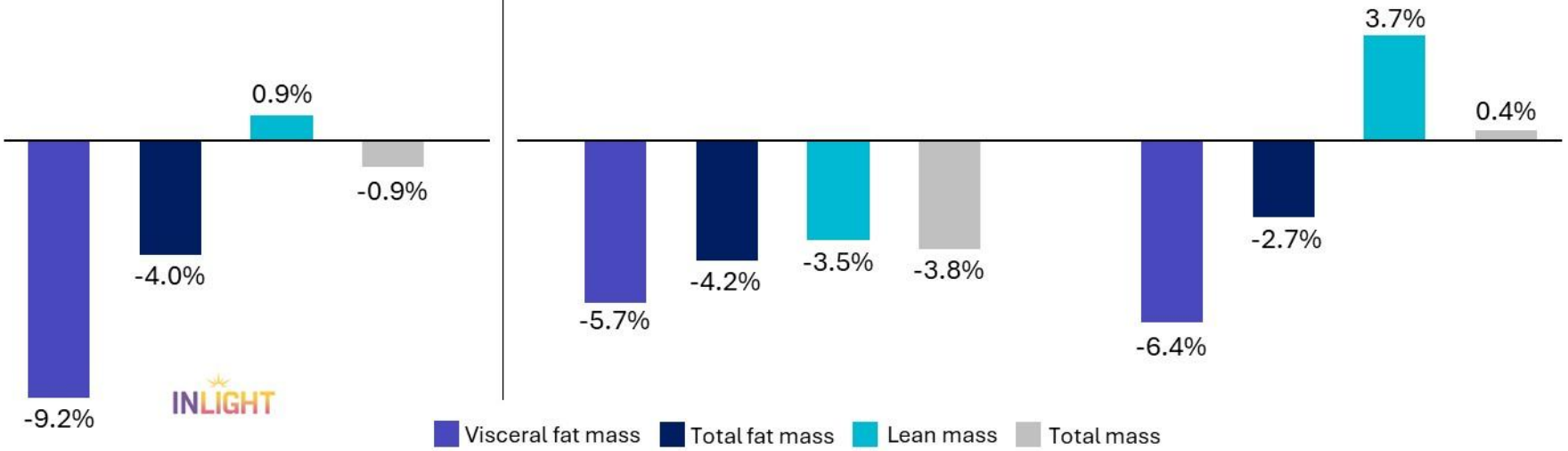
INLIGHT Phase 1 Trial
Placebo-adj. values at 12 weeks[1]

BELIEVE Phase 2 Trial
Placebo-adj. estimated values at 12 weeks[2]

WVE-007 (INHBE GalNAc-siRNA)
Single dose; 240 mg
Mean BMI: 32.1 kg/m^2
No diet/exercise modifications

Semaglutide (GLP-1 agonist)
Weekly[3]; 2.4 mg
Mean BMI: 36.6 kg/m^2
With diet/exercise modifications

Bimagrumab (myostatin inhibitor)
2 doses[4]; 10 mg/kg
Mean BMI: 36.5 kg/m^2
With diet/exercise modifications



1. For INLIGHT, all DEXA percentage changes and p-values are model-based, using the SAP pre-specified analysis. 2. For BELIEVE, all data points are approximate and based on placebo-adjusted estimates extracted from figures, body weight reported as total mass, from Heymsfield SB, et al. Symposium – "*Can we improve the quality of weight loss by augmenting fat mass loss while preserving lean mass? The BELIEVE study of bimagrumab + semaglutide*". Presented at: American Diabetes Association Scientific Sessions; June 20-23, 2025; Chicago. 3. Semaglutide in BELIEVE study was subcutaneously administered weekly and titrated to maintenance dose. N=57 in semaglutide 2.4 mg arm, N=56 in placebo arm. 4. Within the first 12 weeks of the BELIEVE study, bimagrumab was dosed IV at baseline and week 4. N=56 in bimagrumab 10 mg/kg arm, N=56 in placebo arm. **Note: The data presented above are derived from different clinical trials with differences in trial design and patient population, including with respect to BMI. As a result, cross-trial comparisons cannot be made and no head-to-head clinical trials have been conducted.**

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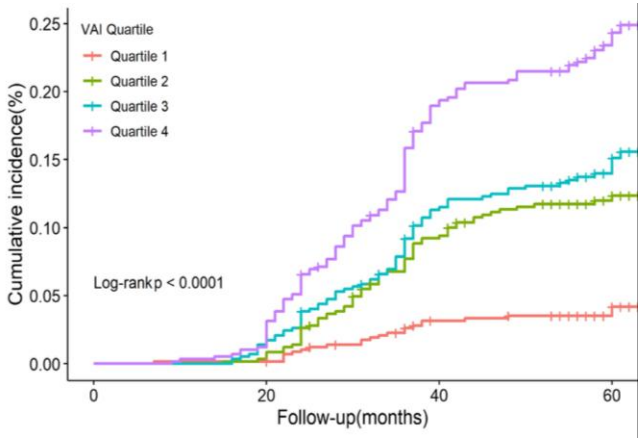
Visceral fat is associated with insulin sensitivity and incidence of MASH, type 2 diabetes and cardiovascular disease

Visceral fat reduction is associated with multiple health benefits

Health Outcome	Visceral Fat Reduction	Associated Benefits
Insulin Sensitivity[1]	≥ 5% decrease in visceral fat	Improved insulin sensitivity, lower HbA1c, better lipid profile
Cardiovascular Risk[2]	≥ 5–10% decrease in visceral fat	Reduced blood pressure, improved lipids, lower systemic inflammation
Liver Fat (Steatosis)[3]	≥ 10% decrease in visceral fat or ≥ 7–10% body weight loss	Significant reduction in hepatic triglycerides, improved liver enzymes
Hepatic Fibrosis[4]	≥ 10% decrease in visceral fat or ≥ 7–10% body weight loss	Resolution of steatohepatitis in up to 90%, fibrosis regression in many cases

Visceral fat increases risk of cardiovascular disease[5]



WVE-007 aims to shift body composition by reducing body fat while preserving muscle, to deliver a healthier cardiometabolic profile

1. Gabriely et al., Diabetes 2002; Campos et al., Diabetes & Vascular Disease Research 2019; Huang et al., Front Endocrinol 2023. 2. Cesaro et al., Front Cardiovasc Med 2023; Khawaja et al., Curr Cardiol Rep 2024; Hiuge-Shimizu et al., J Atheroscler Thromb 2011. 3. Liao et al., PLoS ONE 2023; Jung et al., Endocrinol Metab 2020; ; Krittayaphong et al., Scientific Reports 2024; Hanlon & Yuan, Clin Liver Dis 2021. 4. Liao et al., PLoS ONE 2023; Jung et al., Endocrinol Metab 2020; Vilar-Gomez et al Gastroenterology 2015.. 5. Qaio et al. Cardiovasc Diabetol 21, 225 (2022). VAI = Visceral Adiposity Index

INLIGHT: Clinical trial of WVE-007 in otherwise healthy individuals living with overweight or obesity

Multiple clinical trial sites, including US

INLIGHT

Phase 1 SAD Portion

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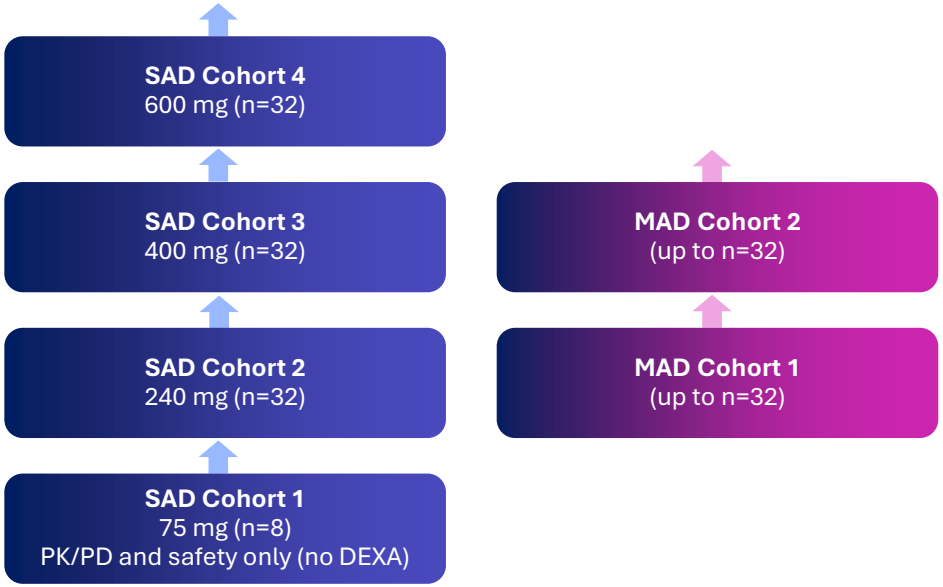


Phase 2a MAD Portion

In individuals with **higher BMI** and **comorbidities**

Plan to initiate in 1H 2026

Key measurements include: Safety and tolerability, PK, Activin E, body composition, biomarkers, body weight

SAD: single-ascending dose; MAD: multi-ascending dose; PK: pharmacokinetics; PD: pharmacodynamics
Randomized, double-blind, placebo-controlled (3:1) study of ascending doses of WVE-007

Potential to address more than one billion individuals with obesity globally

Monotherapy

Single agent in individuals living with obesity

- To induce fat loss with muscle preservation and favorable safety and tolerability

Add-on

Combination with incretin treatments

- To leverage an orthogonal mechanism to incretins for enhanced efficacy

Maintenance

An off-ramp post-incretin treatments

- To prevent weight rebound and maintain metabolic improvements upon incretin cessation

Potentially transformational profile for treating obesity across multiple treatment settings

Phelps, NH, et al. 2024 Lancet 403, 1027

Near term anticipated updates for WVE-007

INLIGHT

1Q 2026

- **6-month** follow-up data from the **240 mg** single-dose cohort
- **3-month** follow-up data from the **400 mg** single-dose cohort

2Q 2026

- **6-month** follow-up data from the **400 mg** single-dose cohort
- **3-month** follow-up data from the **600 mg** single-dose cohort

1H 2026

Initiate Phase 2a MAD Portion

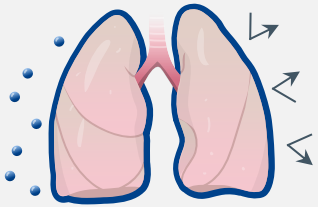
- In individuals with **higher BMI** and **comorbidities**

Expect to initiate new trials of WVE-007 as an add-on to incretin and as post-incretin maintenance in 2026

WVE-006: Potential first-in-class, convenient AIMer (RNA editing) for AATD that addresses both liver and lung manifestations of the disease

- AATD is a rare, inherited genetic disorder commonly caused by G-to-A point mutation
- Patients are predisposed to progressive lung damage, liver damage or both
- Aggregation of mutant Z-AAT protein in hepatocytes and a lack of functional, wild-type M-AAT drives liver and lung disease, respectively

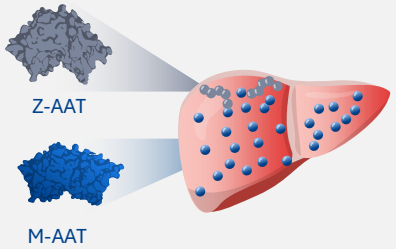
Restore circulating M-AAT and physiological AAT protein production



M-AAT reaches lungs to protect from proteases and **reduce risk of lung pathology**

Standard of care:
IV augmentation therapy

Reduce Z-AAT protein aggregation in liver



RNA correction replaces mutant Z-AAT protein with M-AAT protein to **reduce risk of liver pathology**

Standard of care:
No approved therapies

~200K people in the US and Europe are homozygous for the Z allele (Pi*ZZ genotype)

Strnad et al., 2020 *N Engl J Med* 382:1443-55; Stoller et al., 1993 Alpha-1 Antitrypsin Deficiency GeneReviews. M-AAT: Wild-type alpha-1 antitrypsin protein Z-AAT: mutant alpha-1 antitrypsin protein

RNA editing aims to increase M-AAT and restore physiological AAT production during acute phase response

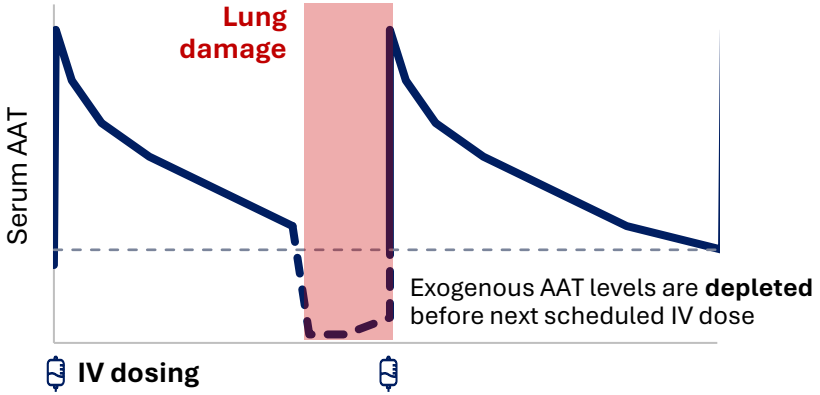
	Homozygous Pi*ZZ No healthy M-AAT	Heterozygous Pi*MZ AAT protein of >11uM, >50% M-AAT
AAT levels increase during acute phase response	✗ No	✓ Yes
Risk of lung pathology	↑ High	↓ Low
Risk of liver pathology	↑ High	↓ Low

Goal:
~50% editing to shift **Pi*ZZ** individuals to AAT function consistent with **Pi*MZ** genotype

WVE-006 enables endogenous AAT production during an acute phase response while augmentation therapy leaves patients at risk

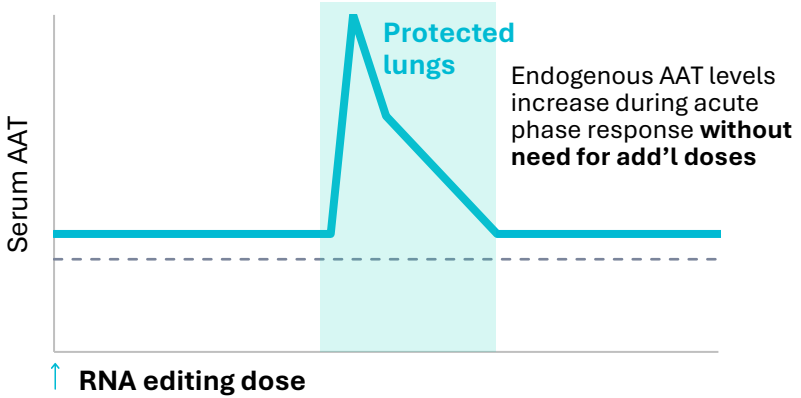
Illustrative model of impact of acute phase response

Augmentation therapy



- Augmentation therapy has **no impact** on liver disease

WVE-006 treatment approach



- WVE-006 also reduces levels of Z-AAT

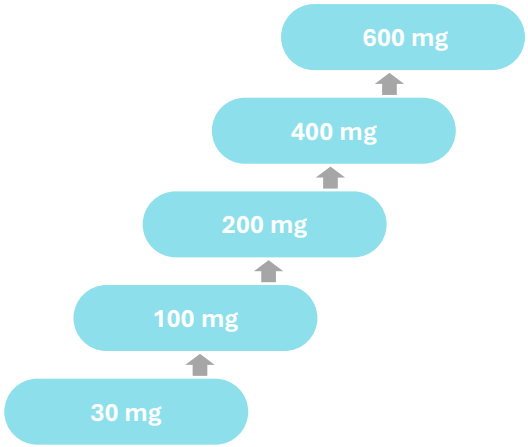
WVE-006 therapeutic goal is to restore dynamic AAT physiology; augmentation therapy goal is to maximize AAT levels as dynamic response is not enabled

RestorAATion-2 clinical trial ongoing

RESTORAATION

RestorAATion-1: Healthy Volunteers → **RestorAATion-2: AATD Patients**

SAD → MAD Multi-dosing complete



SAD Cohort 3
600 mg → **MAD Cohort 3**
600 mg; Q4W

SAD Cohort 2
400 mg → **MAD Cohort 2**
400 mg; Q4W

SAD Cohort 1
200 mg → **MAD Cohort 1**
200 mg Q2W

Study key objectives		
Safety and tolerability	Pharmacokinetics	Serum M-AAT levels

HV: healthy volunteer; SAD: single-ascending dose; MAD: multi-ascending dose

WVE-006 achieved key treatment goals of restoring MZ phenotype

Total AAT levels exceeded 11 µM, production of wild-type M-AAT of greater than 50%, restored physiological AAT production

Plasma AAT of ~13 µM

- Protein levels associated with lower risk of AATD liver and lung diseases

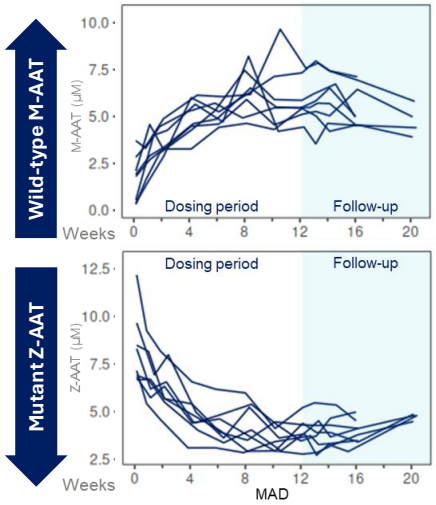
400 mg single dose

12.8 µM total AAT

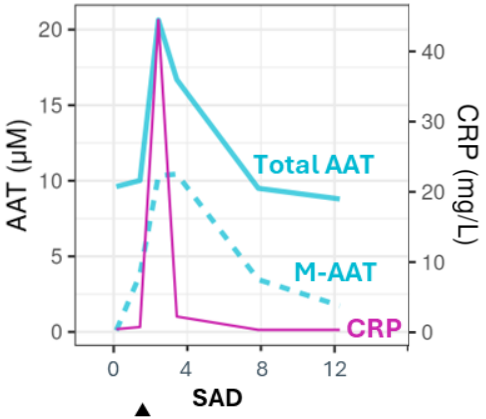
200 mg multidose

11.9 µM total AAT

Wild-type M-AAT protein of 64% of total, reduction in Z-AAT



AAT reached >20 µM during an acute phase response



Acute phase response due to a kidney stone

400 mg MAD data expected in 1Q 2026 and 600 mg SAD and MAD data expected in 2026

Circulating M-AAT, Z-AAT, and total (M + Z) AAT protein in the serum were measured by highly selective and sensitive LC-MS/MS assays (LLOQ: 0.096 µM (M), 0.029 µM (Z)) and reported as mean participant SAD and MAD maximums
Middle: from 200 mg MAD cohort; Right: from 200 mg SAD cohort

RNA editing program: WVE-008 (PNPLA3 AIMer) for liver disease

Clinically-validated RNA editing

- Efficient and consistent RNA editing
- Durable RNA editing supporting infrequent dosing
- Generally safe and well-tolerated

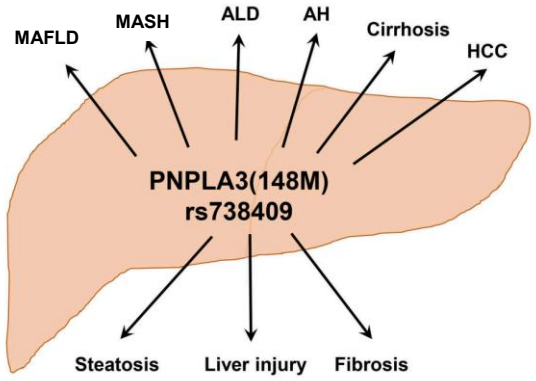
WVE-008 (PNPLA3)

- Strong foundation in human genetics
- Over nine million homozygous PNPLA3 I148M patients with liver disease in US and Europe
- GalNAc RNA editing approach uniquely aims to restore PNPLA3 function to fully address disease

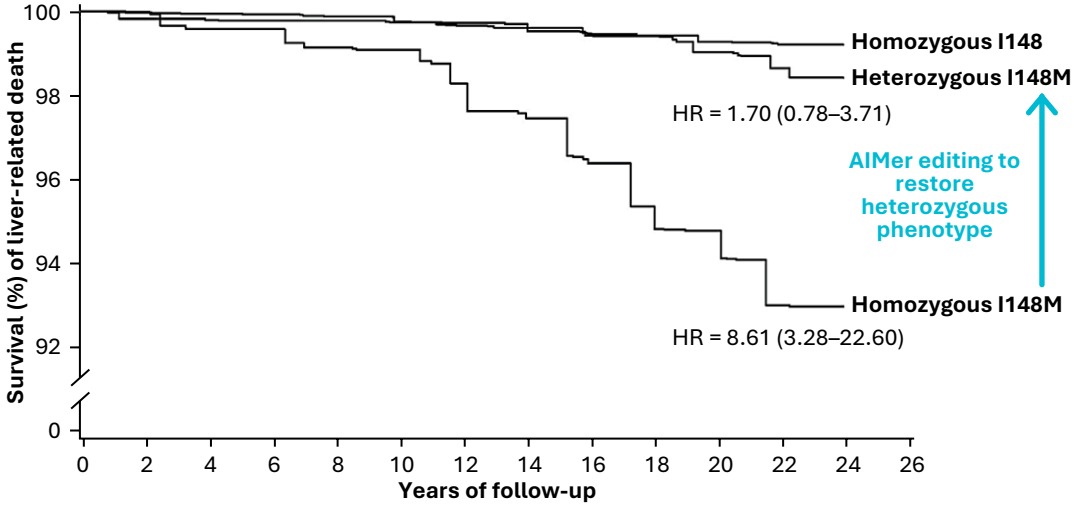
People homozygous for PNPLA3 I148M are at high risk for liver disease

Over nine million homozygous PNPLA3 I148M patients with liver disease in US and Europe

Homozygous PNPLA3 I148M carriers have significantly higher risk of multiple liver diseases



Heterozygous carriers have 80% lower risk of liver-related death as compared to homozygous carriers



>50% RNA editing would support restoration of heterozygous phenotype with lower risk of liver complications and death

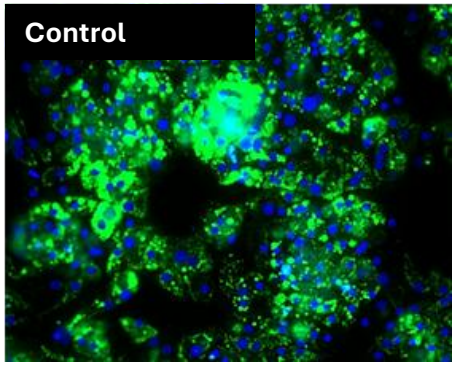
1. Carlsson, B., et al. 2020 *Aliment Pharmacol Ther.*; 2. Unalp-Arida and Ruhl 2020 *Hepatology*; 3. Dong, XC, 2019 *Front. Med*. 4. Liver International, 2025; 45:e16133

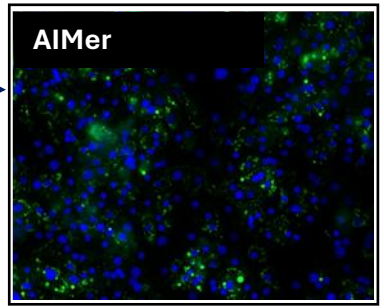
MAFLD, Metabolic dysfunction-associated fatty liver disease; MASH, Metabolic dysfunction-associated steatohepatitis; ALD, alcoholic liver disease; AH, Alcohol-associated hepatitis; HCC, hepatocellular carcinoma

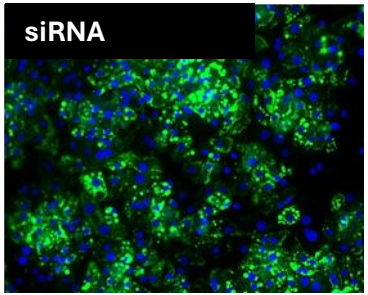
Wave's editing approach delivers greater decreases in liver fat compared to silencing in human liver models

Monolayer model

Decrease in liver fat



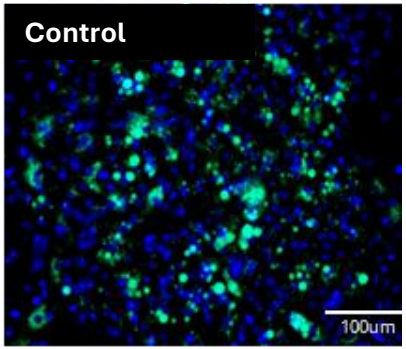


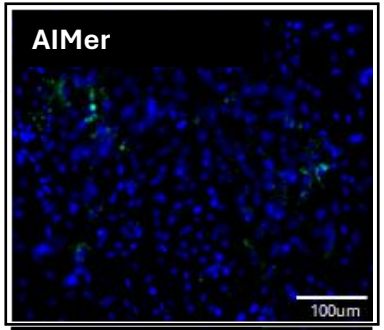


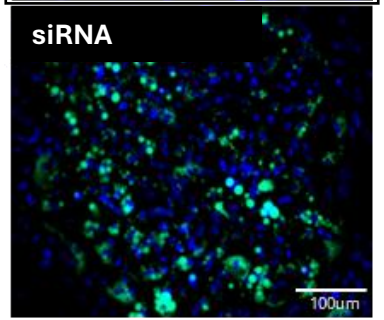
Liver fat

Human HEPATOPAC® model with homozygous I148M

Decrease in liver fat

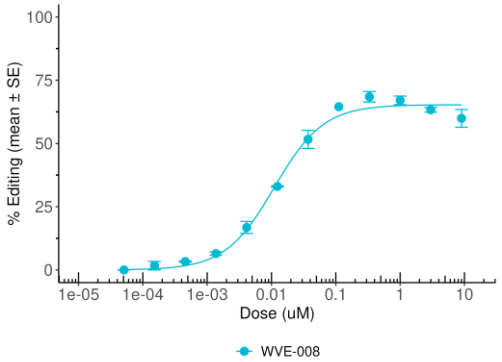




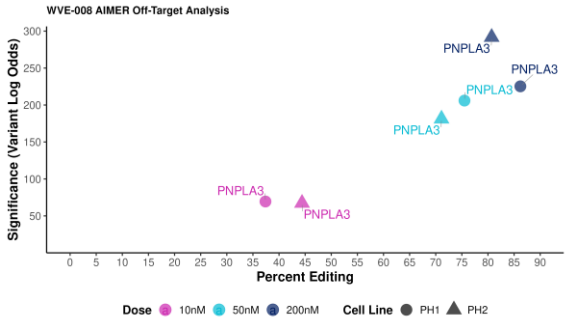


WVE-008: Potential first-in-class, disease modifying therapy, for treatment of PNPLA3 I148M-driven liver disease

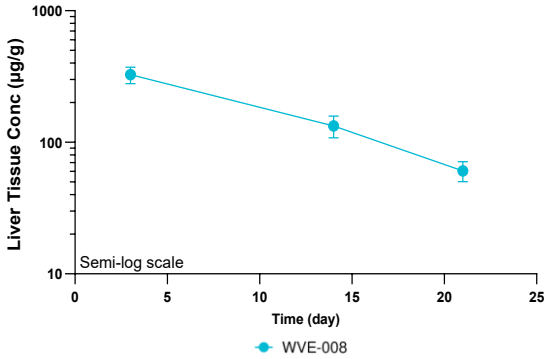
Potential editing with WVE-008



Highly specific editing with WVE-008



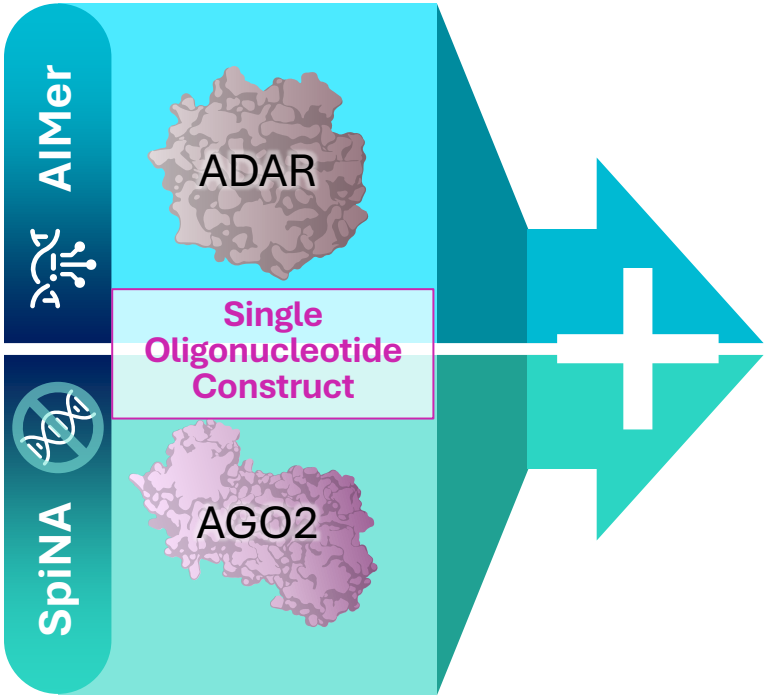
Tissue exposure supports excellent delivery



Expect to file Clinical Trial Application (CTA) for WVE-008 in 2026

Left: 4-parameter log-logistic dose response curve; Middle: Analysis utilized RNA-sequencing with two separate primary human hepatocyte cell lines (PH1/2). Variant calling utilized GATK best practices for RNA variant calling using Mutect2 and display A->G evidence found when filtering for variants found in both cell lines and all doses.

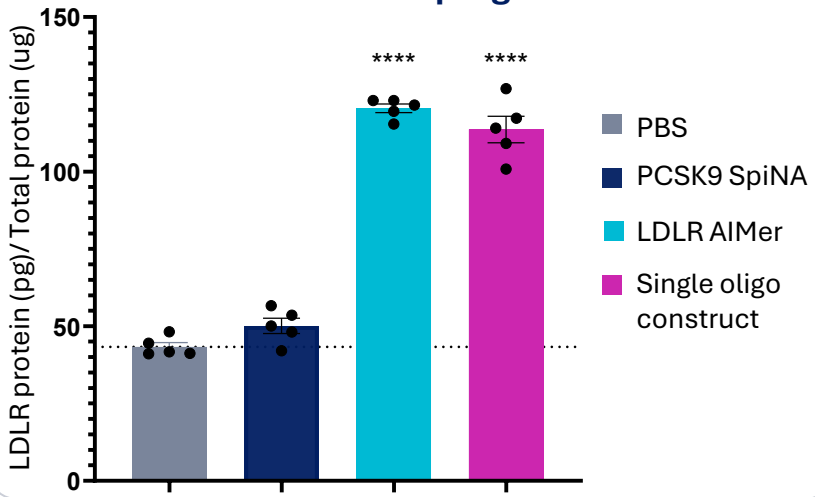
Reimagining RNA medicines: Bifunctional modality to simultaneously knockdown and edit RNA with single oligonucleotide construct



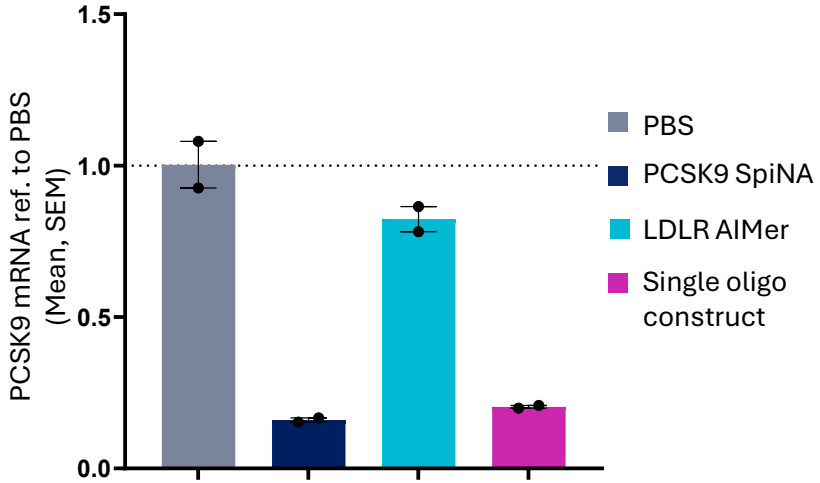
- ✓ Engage both endogenous Ago2 and ADAR enzymes
- ✓ Silence one target, while simultaneously editing or upregulating another unique target
- ✓ Unlock complex indications that require engaging multiple targets
- ✓ May continue to increase durability of editing

Single GalNAc-conjugated oligonucleotide construct can simultaneously upregulate and silence protein *in vitro*

LDLR Protein upregulation



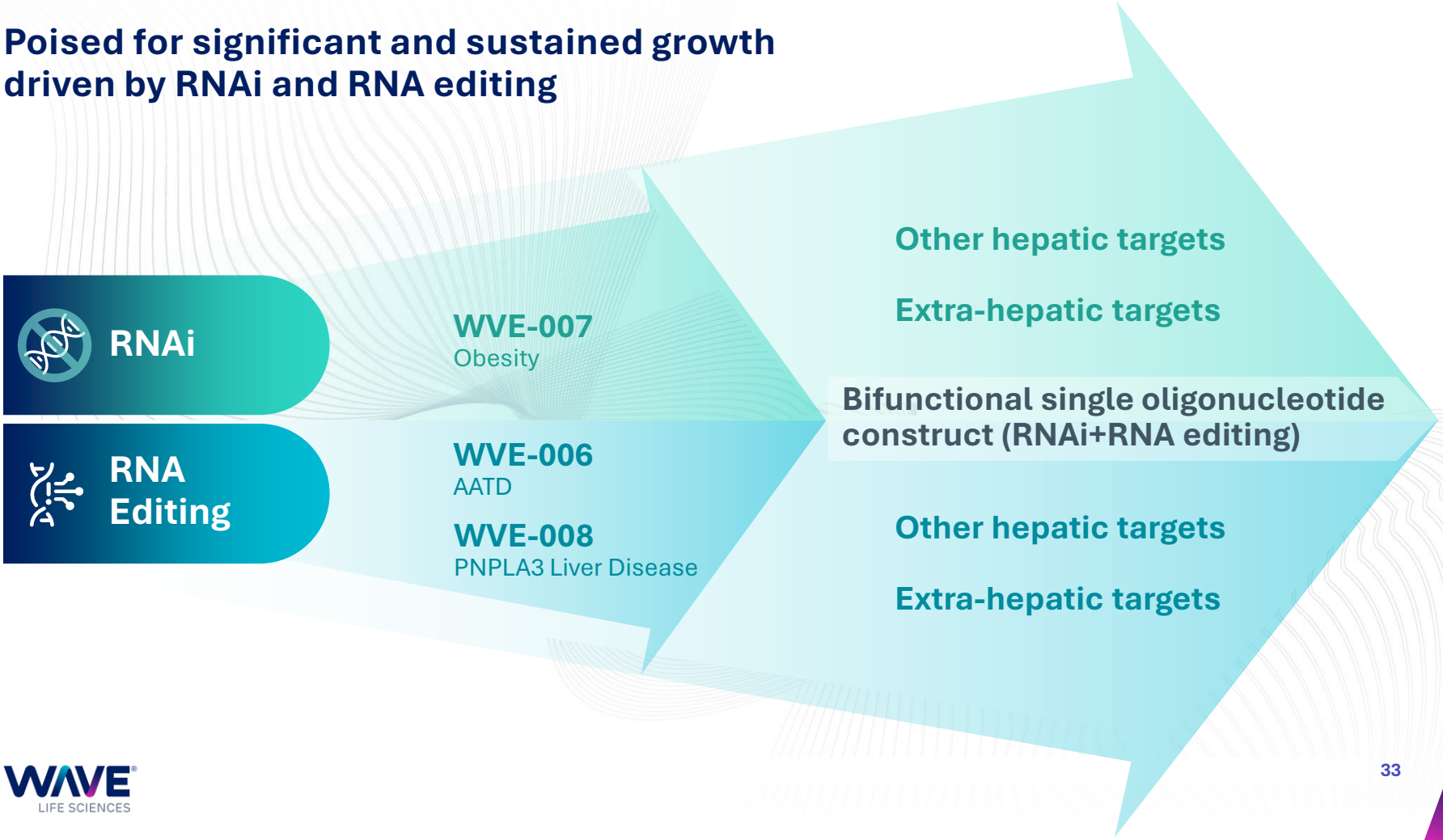
PCSK9 mRNA knockdown



Demonstrates potential to address complex indications that require engaging multiple targets

Primary human hepatocytes were treated in vitro with indicated compounds for 96 hours. Left: LDLR protein was measured using an ELISA; Stats: Welch's One-Way ANOVA with Dunnett's T3 post hoc-comparison. Right: PCKS9 transcript was measuring by qPCR using the ΔΔCt method

Poised for significant and sustained growth driven by RNAi and RNA editing



Anticipated upcoming milestones

RNAi	**WVE-007** *(INHBE)* *Obesity*	**1Q 2026:** Deliver 3-month 400 mg data and 6-month 240 mg data **2Q 2026:** Deliver 3-month 600 mg data and 6-month 400 mg data **1H 2026:** Initiate Phase 2a multidose portion of INLIGHT in individuals living with obesity with higher BMI and comorbidities **2026:** Initiate new trials evaluating WVE-007 as an add-on to incretin and as post-incretin maintenance
RNA editing	**WVE-006** *AATD*	**1Q 2026:** Deliver data from 400 mg multidose cohort **2026:** Deliver single and multidose data from 600 mg cohort
	WVE-008 *Liver disease*	**2026:** File CTA for WVE-008
Splicing	**WVE-N531** *(Exon 53)* *DMD*	**2026:** Submit NDA to support accelerated approval of WVE-N531 with monthly dosing
Antisense	**WVE-003** *(SNP3)* *HD*	Submit IND application for potentially registrational Phase 2/3 study of WVE-003 in conjunction with prospective strategic partner

CTA: Clinical Trial Application; NDA: New Drug Application; IND: Investigational New Drug

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For questions contact:
investorrelations@wavelifesci.com