REGENERON® ROUNDTABLE

MASH + RGC®

Regeneron Genetics Center

NOVEMBER 10, 2026

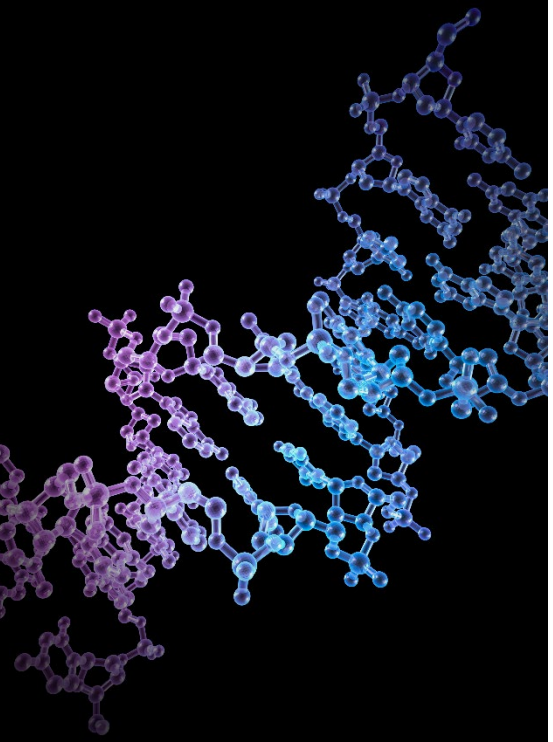
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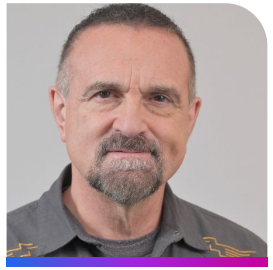
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Regeneron's Leadership RGC / MASH





George Yancopoulos, MD, PhD
Co-founder
Board co-Chair
President
Chief Scientific Officer

Regeneron's scientific differentiation starts with human genetics and proteomics

- Large-scale database links multi-omics with health data to accelerate drug discovery, as illustrated by the identification of protective CIDEB genetic variants and the MASH clinical pipeline



Aris Baras, MD
Senior Vice President,
Head of Regeneron Genetics Center, Co-Head of Regeneron Genetic Medicines

Regeneron Genetics Center (RGC) provides competitive advantage across discovery and development

- Human genetics helps identify promising targets and guide development decisions across diseases
- AI and multi-omics build on RGC's growing database to accelerate research and support new diagnostic opportunities



Gary Herman, MD
Senior Vice President,
Clinical Development Unit Head,
Genetic Medicines

Translating genetically validated targets into clinical candidates

- MASH siRNA pipeline built on RGC's genetic discoveries and validation
- CIDEB^{siRNA} is rapidly advancing into registration-enabling trials, supported by encouraging clinical results



Josh Adelson
Executive Director
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New Products

A large and growing MASH commercial opportunity

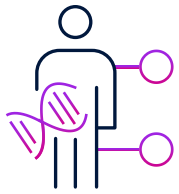
- MASH is a large, growing market with significant unmet need
- CIDEB^{siRNA} could offer a first-in-class treatment for a broad MASH population, pending clinical validation

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Regeneron Genetics Center: From Human Data to Human Health

Leveraging the world's largest multi-omics database: nearly 4 million* individuals with multi-omics (DNA sequences and proteomics) linked to health records



Leveraging world's largest multi-omics database

- To discover new drug targets: identifying novel genetic and proteomic pathways that drive specific diseases
- To revolutionize healthcare: exploiting genetic and proteomic approaches for detecting, following, and managing disease



Drug Discovery & Development

- Multi-omics helps link the right target to the right disease
  - Helped guide Dupixent to nine FDA-approved indications
- Multi-omics helps identify the promising drug targets, including CIDEB for liver disease:
  - CIDEB discovered in 2021
  - CIDEB clinical proof-of-concept achieved in 2026



Revolutionizing Healthcare

- Multi-omics demonstrating potential to better diagnose and predict disease progression
  - Proteomics replaces liver biopsy
- Single test for almost every disease
- Combining our multi-omics database and approaches can reimagine healthcare management
  - Greater efficiency
  - Lower costs
  - Better outcomes

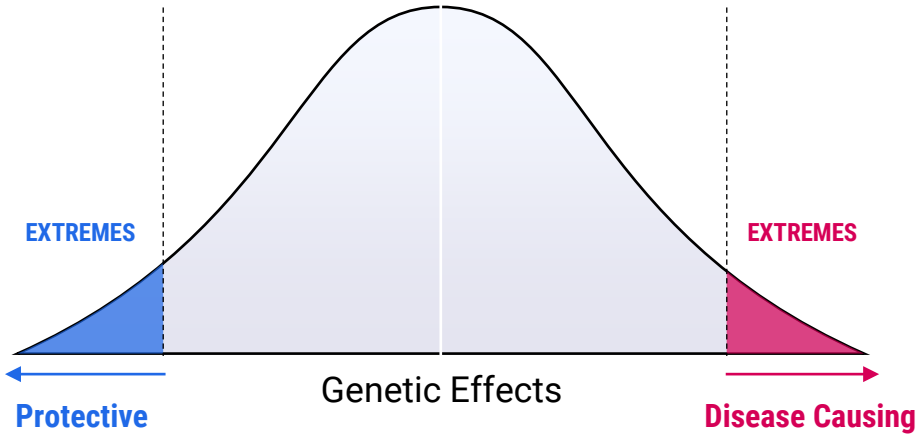
*Including ~3.3 million with data generated internally as well as ~700,000 from in-licensing

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RGC has focused on large-effect genetic discoveries leading to ~50 novel targets across all of Regeneron's therapeutic areas

Our proprietary, de-identified, longitudinal health record-linked genomic and proteomic data at massive scale uncovers hidden targets that can lead to breakthrough therapeutics

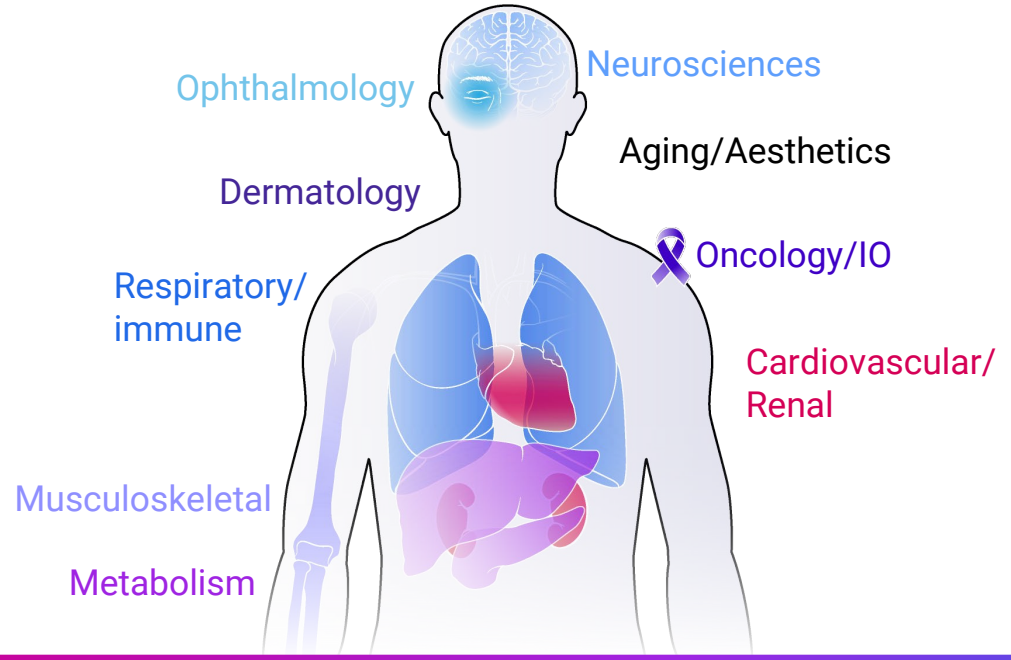
Identifying novel large-effect genetic factors



6x **more likely to be approved: targets backed by rare protective variants vs. targets with no genetic support***



Discovering drug targets across organ systems



Many programs in our pipeline are discovered (e.g. CIDEB) or supported (e.g. Dupixent) by human genetics

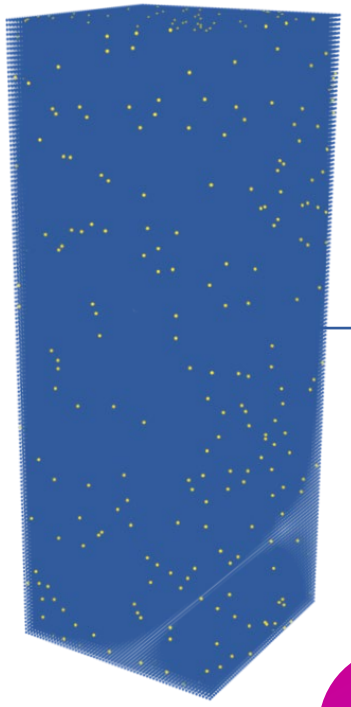
*Relative probability of success, Phase 1 to launch. Source: RGC analysis; see slide 8 for data

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RGC discovery that genetic loss of CIDEB led to protection of liver disease has translated to clinical proof-of-concept

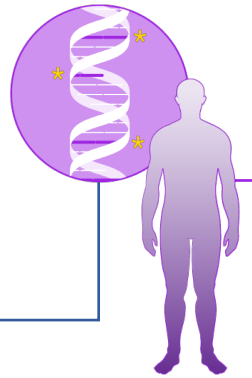
Proof-of-concept results with our CIDEB-silencing siRNA support the target as a potential medicine for the growing MASH patient population

Analysis of millions of individuals



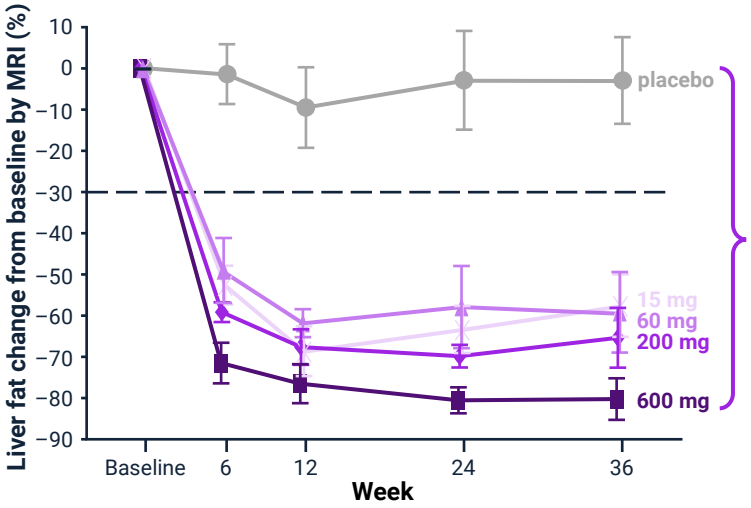
1 person out of ~500 carries the protective allele

CIDEB loss-of-function (LOF) mutations



Carriers of rare protective CIDEB LOF variant have less liver fat, lower serum ALT and a 43% lower risk of developing MASLD/MASH

Single dose of CIDEB^{siRNA} induced rapid, deep, and long-lasting normalization of liver fat in patients with MASLD*



up to 80%
reduction in liver fat from baseline

Clinical benefit with CIDEB^{siRNA} validates our genetics-driven target-discovery approach

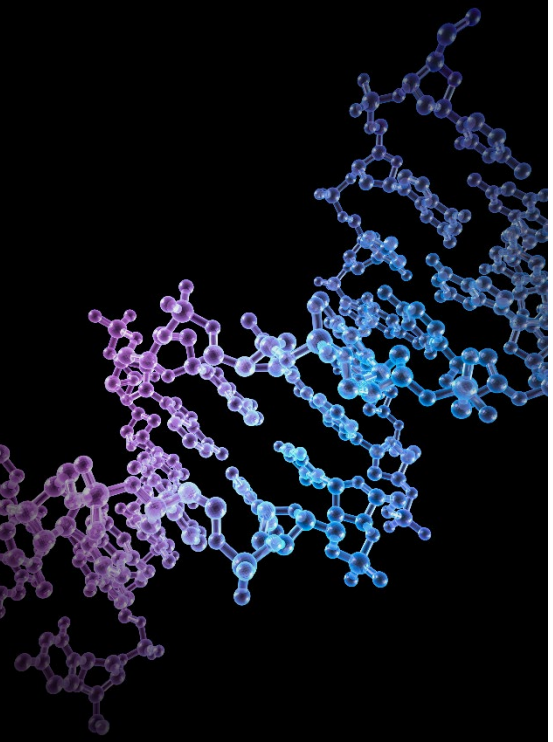
RGC has developed a new MASH proteomics assay that can replace invasive liver biopsy

References:
Verweij N et al. N Engl J Med. 2022
Norton T et al. N Engl J Med. 2026
ALT – Alanine transaminase; *Part A of ongoing FIH trial NCT06836609; N=40

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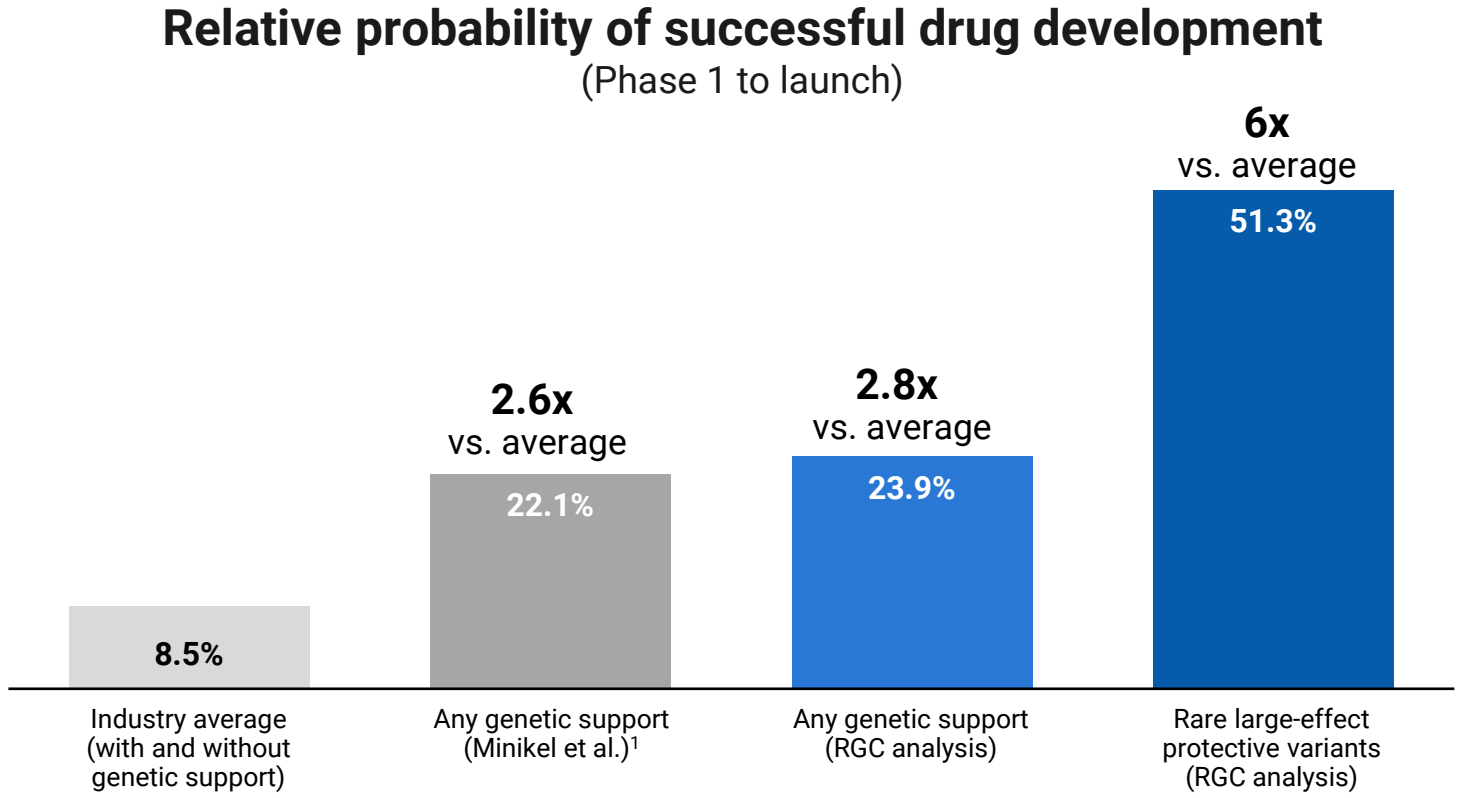
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Human genetics evidence greatly improves probability of success for drug discovery and development

RGC's emphasis on rare large-effect, protective genetics provides high level of validation and greatest probability of success

Genetic support increases the odds of success

- Less than 10% of clinical programs end in approval
- Targets with human genetic support are 2.6-2.8x more likely to succeed
- Rare protective variants provide strongest support, 6x more likely to be approved



Targets backed by rare protective variants are 6x more likely to yield an approved drug than targets unsupported by genetic evidence

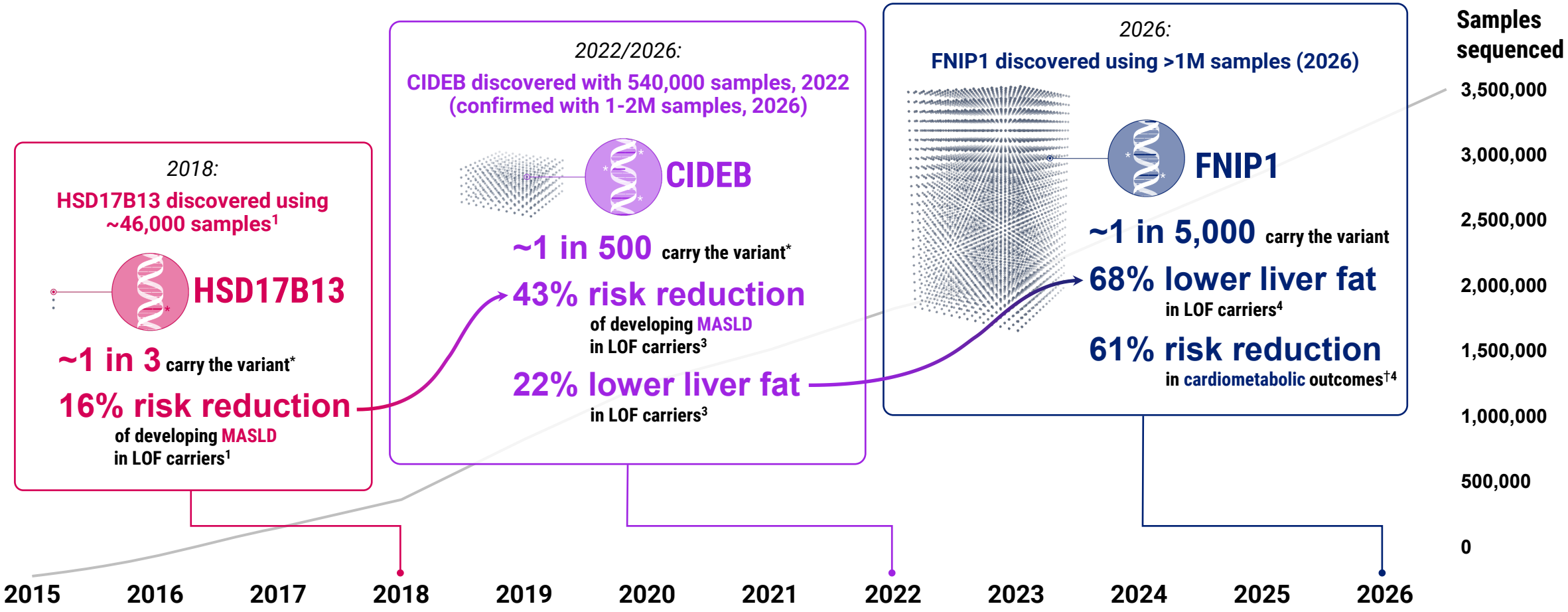
"Any genetic support" is based on analysis of common gene variants. Source: RGC analysis of Citeline Pharmaprojects data and RGC internal data covering more than 40,000 drug development programs mapped to nearly 3,000 drug targets; 1. Minikel et al., Nature 629, 624–629 (2024).

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Increasing the scale and diversity of our dataset is unlocking rarer variants with larger effects

Allows Regeneron to discover incredibly rare, therapeutically relevant gene variants, leading to better drug targets



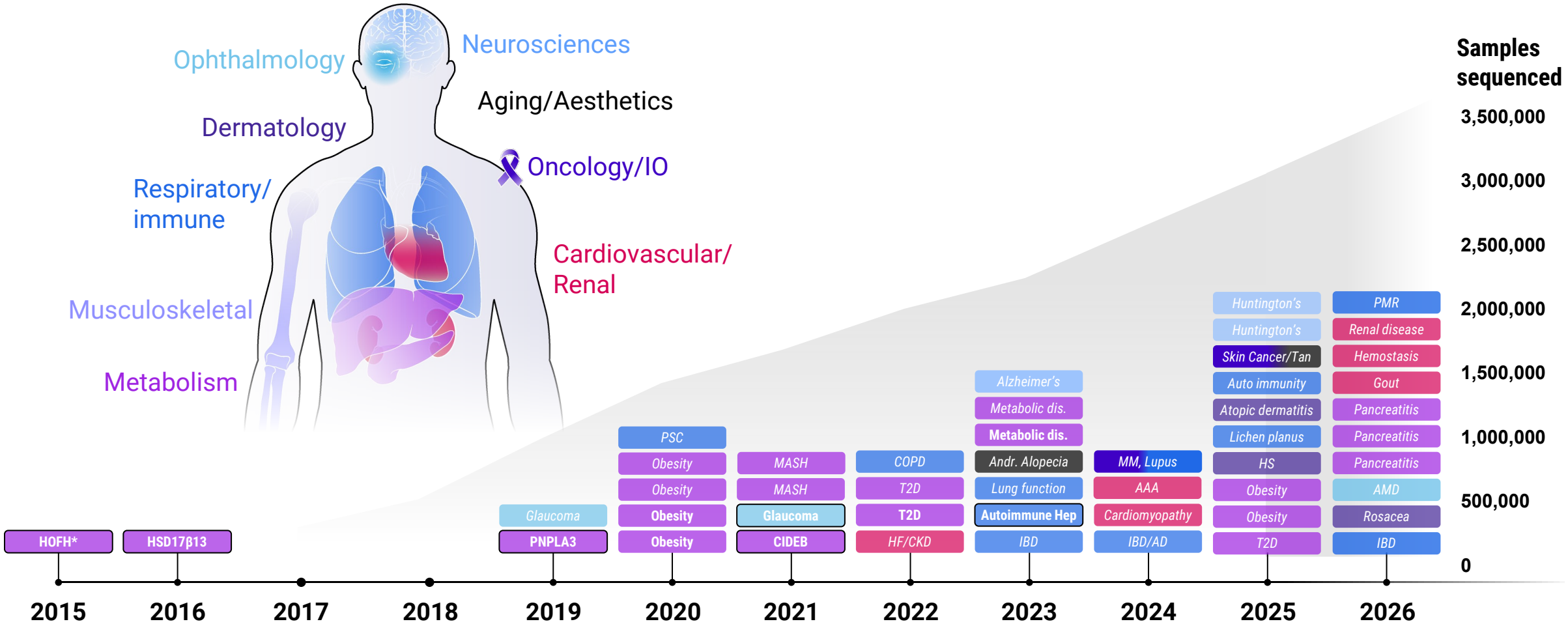
Timeline indicates year of publication, not target discovery

1. Abul-Husn et al. N Engl J Med. 2018; 2. Verweij N et al. N Engl J Med. 2022; 3. Norton T et al. N Engl J Med. 2026; 4. Hindy et al. Nature 2026; *Allele carrier frequencies are from the most recent publication
†Defined as the composite of myocardial infarction, type 2 diabetes, metabolic dysfunction associated liver disease and cirrhosis.

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RGC discovered and/or validated ~50 targets across multiple therapeutic areas

Programs for six of these targets are in the clinic; potential for many more to follow CIDEB's clinical validation



Timeline indicates year of target discovery/validation

Each tile represents one target, labeled with its lead indication; color designates therapeutic area

Outline – target in the clinic, * - FDA approved, **bold** – target disclosed, *italic* – target undisclosed

HOFH – Homozygous familial hypercholesterolemia; MASH -- Metabolic dysfunction-associated steatohepatitis; PSC –Primary sclerosing cholangitis; COPD – Chronic obstructive pulmonary disease; T2D – Type 2 diabetes; HF – Heart failure; CKD – Chronic kidney disease; IBD – Inflammatory Bowel Disease; AAA -- Abdominal Aortic Aneurysm; MM – Multiple Myeloma; AD – Atopic Dermatitis; Tan – Safe Tanning; HS – Hidradenitis suppurativa; PMR – Polymyalgia rheumatica; AMD – Age-related macular degeneration

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RGC enables competitive advantages in guiding development strategy: Dupixent indication expansion

RGC genetic insights validated indications for Dupixent and now support development strategy for most of our other development programs

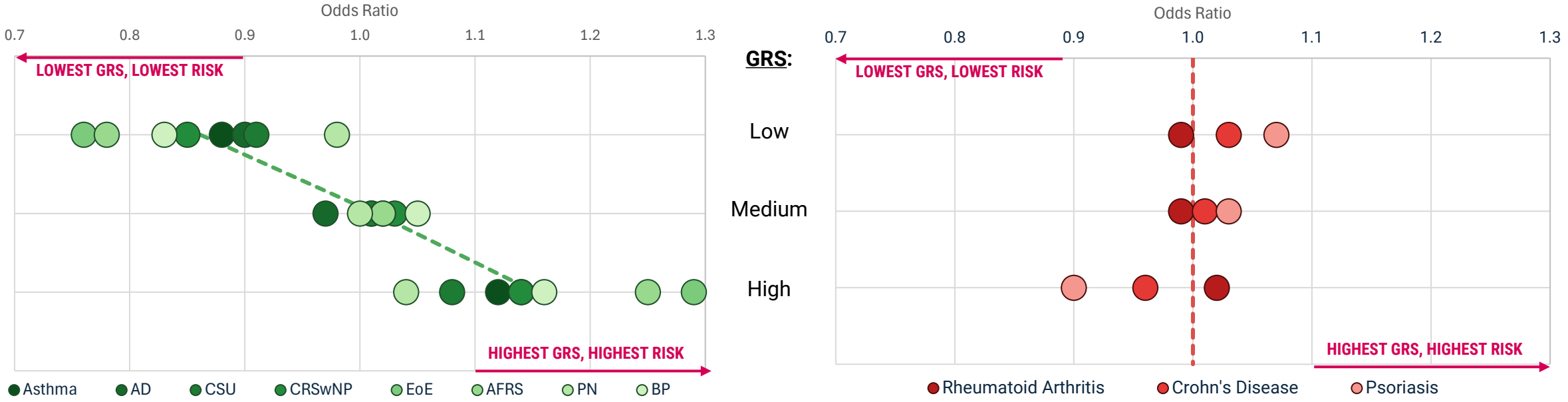
IL4/IL13 Genetic Risk Score (GRS) – initially validated for asthma, and later used to inform Dupixent development for other indications

- Large-scale genetics paired with health records linked the **IL4/IL13 Genetic Risk Score** to a higher risk of asthma
- The same approach identified or validated other diseases that might benefit from IL4/IL13 pathway blockade with Dupixent
- It also identified diseases with no observed association with the pathway, indicating limited genetic support for pursuing those indications

Correlation exists

Association of the IL4/IL13 pathway activity (represented by the Genetic Risk Score) with odds of developing disease

Correlation does not exist



Adapted from Hamilton, Jennifer D et al. Allergy, doi:10.1111/all.70380; RGC internal data

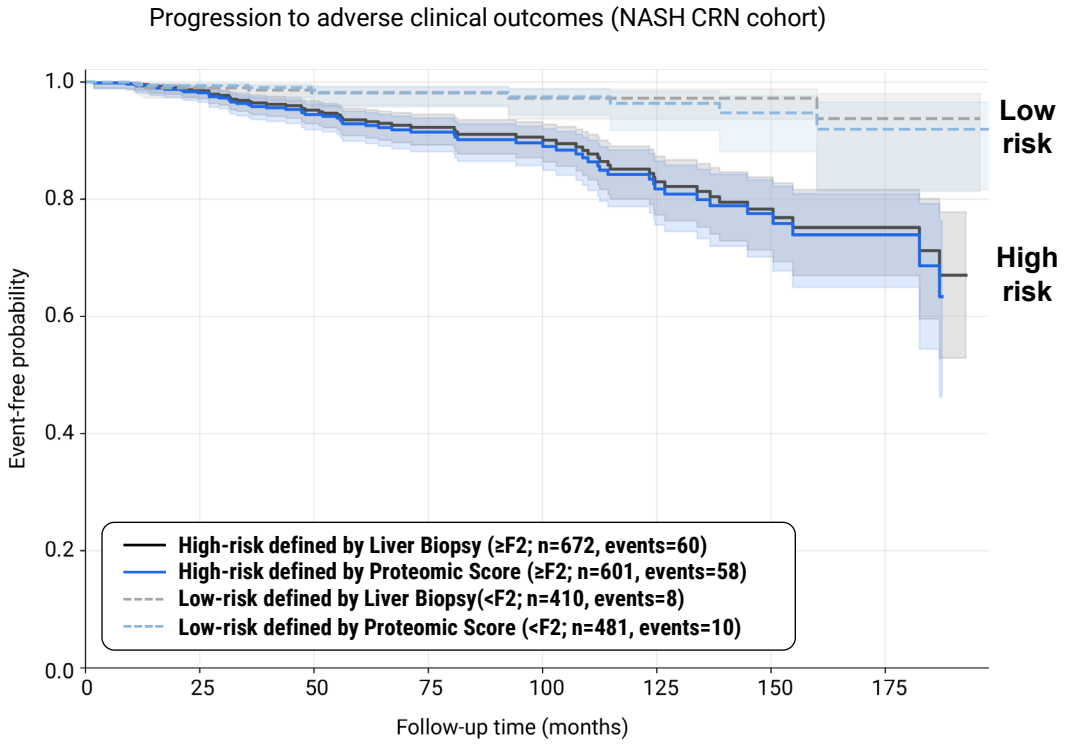
AD – Atopic dermatitis; CSU – Chronic spontaneous urticaria; CRSwNP – Chronic rhinosinusitis with nasal polyps; EoE – Eosinophilic esophagitis; AFRS – Allergic fungal rhinosinusitis; PN – Prurigo nodularis; BP – Bullous pemphigoid

RGC has leveraged genetics and now proteomics to develop advanced disease risk scores and diagnostic opportunities

RGC has developed a new MASH Proteomics Risk Score that can replace invasive liver biopsy

Proteomics

RGC's proteomics blood test performs as well as liver biopsies in predicting clinical outcomes in at-risk MASH patients



A potential proteomic blood test for at-risk MASH

Potential to replace invasive liver biopsy with a simple blood draw

- **Equivalent prognostic ability to biopsy**: in individuals with ≥ F2 fibrosis, 10-year adverse event rate estimate was 15.8% according to our proteomic 'liquid liver biopsy' vs. 14.9% per invasive liver biopsy
- **Outperforms other non-invasive tests:** identifies 90% of ≥ F2 fibrosis cases at a false positive rate of 11%; significantly higher performance than existing non-invasive tests for at-risk MASH

A deployable proteomic panel could become a **non-invasive diagnostic for MASH**, powering trial screening and enrollment as well as potential future commercial applications

AASLD Late-Breaker — Backman et al. Clinical outcomes include HCC, decompensation, transplant, ascites, varices, encephalopathy

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Over a decade of proprietary human genetics data, amplified by AI

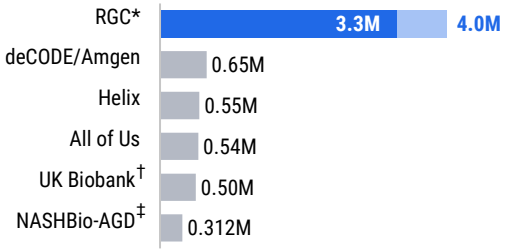
RGC capabilities provide competitive advantage, enabling us to sequence and analyze genomes, proteomes, and data at scale

RGC data

RGC Regeneron Genetics Center

~4M sequences, including 3.3M generated in-house → **10-50M** sequences in 5–10 years

RGC has largest cohorts of sequenced human samples



Consumer genetics
AncestryDNA (~30M) and 23andMe (~14M): genotype array-only, no exomes or genomes, limited health record data

400M+ health records, increasing via collabs, e.g. TriNetX, Truveta → **Large Opportunity** tokenization (pairing) biospecimens with health records at scale

AI and our unique analysis toolkit

- **RGC has developed custom LLMs to analyze DNA sequences, proteomics, medical images, and health data**
  - Our model for finding disease-causing protein variants outperformed competitors, including Google's AlphaMissense[1]
- **A decade of experience in iterating, assessing, and refining keeps improving our models and data**
  - Discoveries per sequenced person have doubled in recent years
  - AI is built into our workflows and data pipelines, and leverages AI-assisted coding
- **We can train LLMs on proprietary RGC data, enabling development of AI-driven healthcare products for consumers and companies**
  - Example: Developing new proteomics assays to replace traditional diagnostics

*RGC includes exomes and imputed genomes. Representative cohorts shown. Counts are from each organization's own public statements and are not strictly comparable (e.g., exomes vs. genomes); †The UK Biobank 500K exome and whole genome studies are included in RGC, and ~247K of them are also included in deCODE/Amgen count. ‡The NASHbio-AGD 312K whole genome project is included in the deCODE/Amgen participant counts. 1. Gillies CE et al. *Genet Epidemiol*. 2025;49(8):e70023. doi:10.1002/gepi.70023

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RGC drives Regeneron's R&D process, with future ambition to revolutionize healthcare management

Human genetics raises the odds of success

Targets backed by large-effect protective variants are 6x more likely to achieve technical and regulatory success

RGC has delivered ~50 actionable targets

Six programs are in the clinic; CIDEB is the latest to achieve clinical proof-of-concept

Multi-omics guides development

Genetics that validated Dupixent's indications, now joined by proteomics, informs development decisions across our pipeline

Large-scale, health-record-linked data combined with AI creates a major strategic advantage

~4M DNA sequences and proteomics data from ~1M patients, all linked to health records, represent the world's largest proprietary multi-omics database

Exploring ways to revolutionize healthcare management

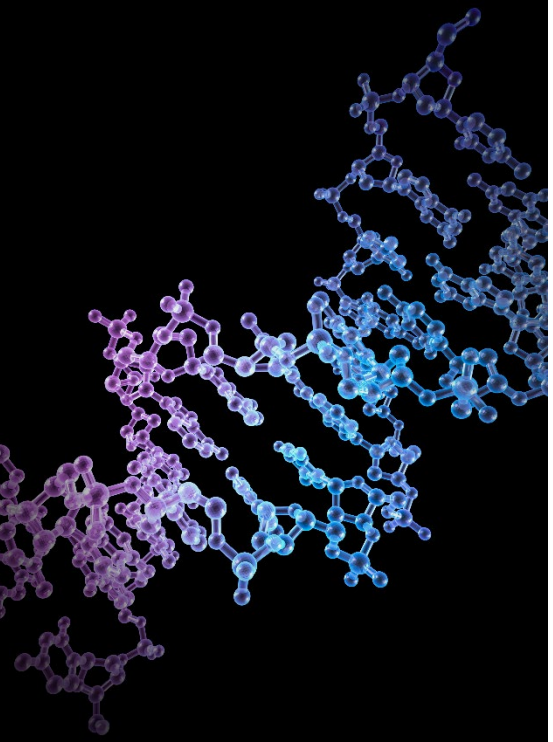
Genomic and proteomic data can pinpoint who needs intervention, shifting care from reactive to proactive to potentially create efficiencies, improve outcomes and lower healthcare system costs

CIDEB siRNA is one of many novel therapeutic candidates with first-in-class and/or best-in-disease potential

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Genetics to therapeutics: Regeneron's differentiated pipeline of liver-directed siRNAs for a large and growing MASH market

In proof-of-concept trials, Regeneron's siRNAs have demonstrated rapid and sustained reductions in liver fat after a single dose

What is MASH?

Metabolic Dysfunction-Associated Steatohepatitis (MASH) is an inflammatory and fibrotic liver disease driven by excess liver fat that affects **~13M U.S. adults**

Rapidly developing market

MASH is a rapidly growing market that remains under-penetrated with low diagnosis rates

- The addressable population is expected to grow rapidly as disease awareness increases, non-invasive testing expands, and more patients are referred to specialists

Market is projected to exceed **+$15B***

Reducing liver fat and/or fibrosis is critical to improve liver disease and decrease clinical liver events

Genetic validation

Regeneron Genetics Center data validate MASH targets affirming liver fat's causal role:

- **CIDEB** LoF variants: reduced liver fat, protection vs. MASH/ALD/cirrhosis
- **PNPLA3** Risk variant: 2x liver fat with higher risk of severe outcomes

Pipeline programs

We have generated PoC data for two siRNAs and are in the process of initiating registration-enabling studies:

- **CIDEB^{siRNA}:** single dose **rapidly reduces liver fat by up to ~80%** and normalizes liver fat, for use in an *all-comers* MASH population
- **PNPLA3^{siRNA} (pivicasiran)**: single dose **rapidly reduces liver fat by ~50%**, for use in *PNPLA3 homozygotes* with fibrotic MASH

(HSD17B13^{siRNA} development has been deprioritized, based on comparative genetic and clinical data)

Potential Differentiation of Regeneron's siRNAs

Efficacy:

- Regeneron siRNAs have demonstrated rapid and profound liver fat reduction

Tolerability & Safety:

- Regeneron's siRNA candidates have shown favorable tolerability, with **no nausea or vomiting AEs reported**
- ~40-50%† of patients discontinue weekly GLP-1 therapy within 12 months, driven in part by significant GI tolerability issues

Dosing Convenience:

- siRNAs can be dosed infrequently (~3-6 months)

CIDEB: cell death inducing DFFA like effector B; **PNPLA3**: patatin-like phospholipase domain-containing 3; **HSD17B13:** hydroxysteroid 17-beta dehydrogenase 13; ALD: alcohol-associated liver disease, LoF: Loss of Function; PoC: Proof of Concept

* Evaluate Pharma, Clarivate

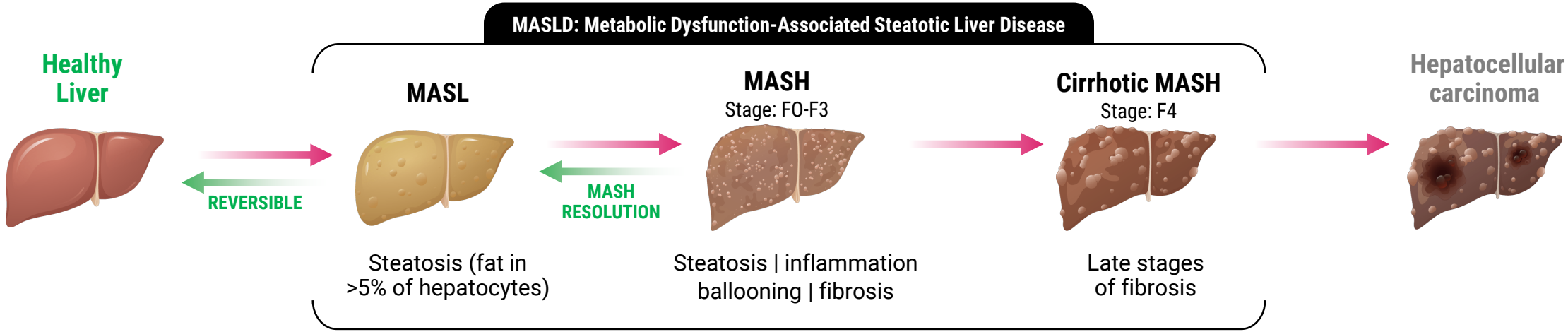
† Gleason, P, et al. Journal of Managed Care & Specialty Pharmacy 2024; Ho, D, et al. Journal of the American Medical Association 2024

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Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) disease progression

MASLD is one of the most common chronic liver diseases and is increasing in prevalence, driven by rising rates of obesity, type 2 diabetes and metabolic syndrome



Prevalence:

- ~13 million MASH patients in U.S.
- ~5.0-5.5M addressable MASH patients with moderate-to-advanced fibrosis (F2-F3)*
- Fewer than 10% of the addressable population are diagnosed, and 20-25% of those diagnosed receive advanced treatment*

Treatment Landscape:

- Lifestyle changes, including sustained weight loss, can improve steatosis, inflammation, and fibrosis in some patients, while use of GLP-1 based therapies continues to grow
- Disease management also includes optimization of metabolic comorbidities (obesity, T2D), dyslipidemia, and hypertension

FDA-approved Therapies (F2-F3):

- **Rezdiffra** (resmetirom, THR-β agonist, once-daily oral), is a liver-directed therapy and was the first approved FDA-medicine indicated for MASH with moderate-to-advanced fibrosis (F2-F3)
- **Wegovy** (semaglutide, GLP-1 receptor agonist, weekly injectable), addresses body's overall metabolism delivering secondary liver improvements

Investigational Classes of Agents:

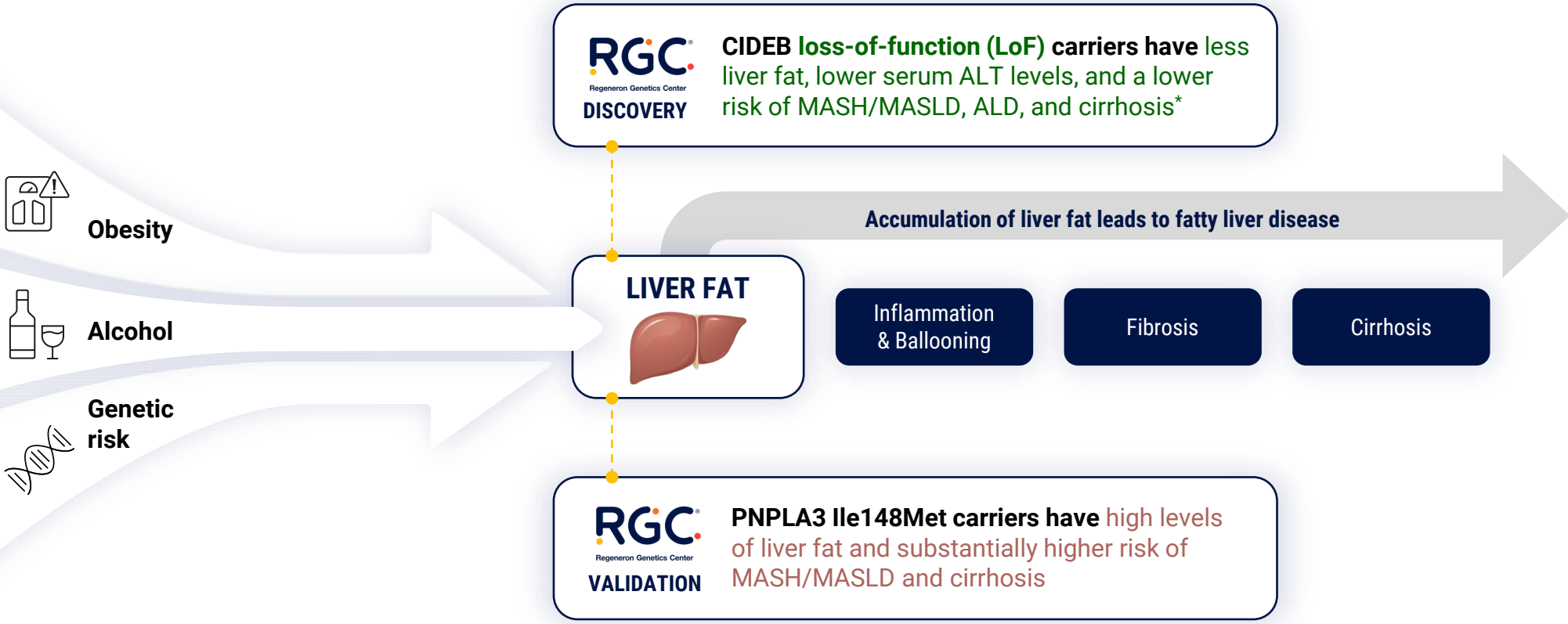
- **Genetics-based siRNAs** | Newer Rs | THR-β agonists | FGF21 analogs | PPAR agonists

GLP-1: glucagon-like peptide-1; **FGF21**: fibroblast growth factor 21; **THR-β:** Thyroid hormone receptor beta; **PPAR:** peroxisome proliferator-activated receptor
* Evaluate Pharma, Clarivate

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Human genetic evidence indicates that liver fat drives fibrosis and cirrhosis, guiding Regeneron's approach for steatotic liver disease



RGC's powerful engine has discovered and validated the most promising drug targets for MASH/MASLD, while also elucidating critical underlying biology

* Verweij, N. et al. (2022) 'Germline mutations in CIDEB and protection against liver disease', New England Journal of Medicine, 387:332–344, doi:10.1056/NEJMoa2117872

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MASH clinical programs are rapidly advancing with registration-enabling trials underway for CIDEB^{siRNA}

	Progress to Date		2027	2028	2029	2030	2031+
PNPLA3^{siRNA}	Phase 1b – MASLD POC	*Data presented in 2025*	Initiating additional Phase 2 study in MASH				
	Phase 2 Combination – PNPLA3-Related MASLD (PNPLA3 ± GLP-1R)	Trial initiated					
CIDEB^{siRNA}	Phase 1/2a POC: Single dose – MASLD Two doses – MASH	***New Data Presented***	Registration-enabling trials underway				
	Phase 2b – Fibrotic MASH Phase 3 – Safety Study (MASLD)	Trials initiated					

RGC has powered our MASH clinical programs that collectively have significant commercial potential

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PNPLA3^{siRNA} (Pivicasiran): Precision medicine approach for genetic MASH

RGC
Regeneron Genetics Center

- Homozygote carriers for the PNPLA3 Ile148Met variant have more liver fat and a higher risk of liver disease and liver-related death than non-carriers
- RGC studies of more than 900,000 individuals linked two copies of the variant to approximately twice the liver fat and up to four times the risk of cirrhosis compared with non-carriers
- The variant amplifies obesity-related liver disease risk, with an impact comparable to approximately 30 kg of additional body weight

Unmet Need | Opportunity

Current MASH Landscape	Unmet Need
• Limited therapies for fibrotic MASH (F2–F3) and no approved treatments for cirrhotic MASH (F4)	• More effective liver-directed therapies that address unmet needs across disease stages
• GI side effects and limited or plateauing benefit can restrict GLP-1Rs use and lead patients to discontinue treatment	• Well-tolerated therapies that offer infrequent dosing without GI side effects
• Patients may need additional treatment alongside or after GLP1Rs	• Therapies that can complement GLPR1s or provide options for patients who switch or discontinue treatment

Precision medicine therapeutic for the ~2M U.S. MASH homozygote carriers of the PNPLA3 Ile148Met risk variant

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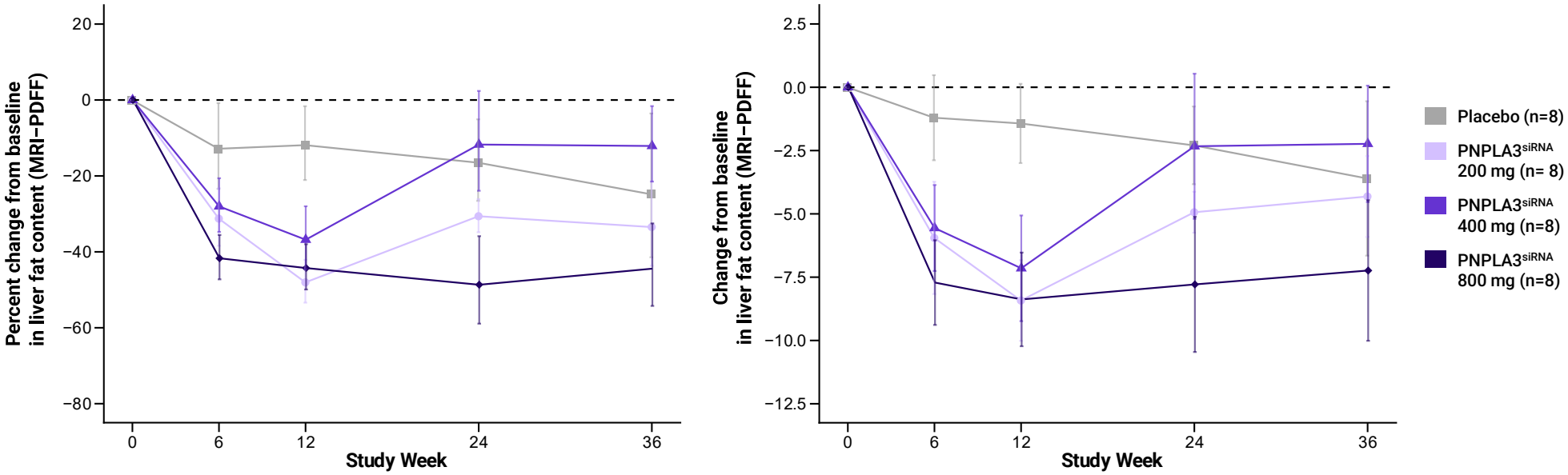
Pooled POC data showed that PNPLA3^{siRNA} induces large and sustained reductions in liver fat in patients with homozygous PNPLA3-related MASLD

Results provide evidence informing a new precision medicine approach for those with the PNPLA3 Ile148Met homozygous genotype

MASLD Patient Population

- Patients homozygous for the PNPLA3 risk allele
- Elevated body mass index ≥27 kg/m^2 (≥23 kg/m^2 for East Asian participants)
- Liver fat ≥8.5% by MRI-PDFF

PNPLA3^{siRNA} reduced liver fat by 48%, 37%, and 44% (relative to baseline at week 12) in the PNPLA3^{siRNA} 200mg, 400mg, and 800mg groups, respectively, compared to 12% in placebo



A single dose of PNPLA3^{siRNA} reduced liver fat by up to ~50% and was generally well tolerated
Clinical development program to be tailored for homozygous PNPLA3-related MASH population

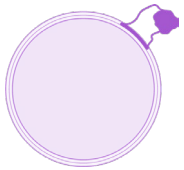
Studies: NCT05648214 and NCT06024408

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CIDEB^{siRNA}: First-in-class mechanism with all-comers applicability

Regeneron Genetics Center (RGC) identified rare CIDEB **loss-of-function (LoF)** variants associated with substantial protection against liver disease

- RGC's analyses*,† of 1-2 million individuals shows that carriers of one predicted CIDEB loss-of-function variant had 22% relative reduction in liver fat, measured by MRI-PDFF, and lower serum alanine aminotransferase (ALT) levels
- Analyses*,† also reinforced CIDEB's protective association: 43% lower odds of MASLD/MASH, and 43% lower odds of MASLD-related cirrhosis
- Based on protective effects in carriers (who have ~50% reduction in CIDEB), it is anticipated that an effective siRNA (which can lower CIDEB by ~80%-95%) could have even greater benefits



CIDEB is a lipid droplet–associated protein that contributes to lipid droplet fusion and growth, as well as lipoprotein lipidation, maturation, and transport

Unmet Need | Opportunity

Current MASH Landscape	Unmet Need
• Limited therapies for fibrotic MASH (F2–F3) and no approved treatments for cirrhotic MASH (F4)	• More effective liver-directed therapies that address unmet needs across disease stages
• GI side effects and limited or plateauing benefit can restrict GLP-1Rs use and lead patients to discontinue treatment	• Well-tolerated therapies that offer infrequent dosing without GI side effects
• Patients may need additional treatment alongside or after GLP-1Rs	• Therapies that can complement GLPR1s or provide options for patients who switch or discontinue treatment

CIDEB^{siRNA} is a first-in-class liver-targeted investigational therapy and currently the only one in clinical development

*Verweij N et al. N Engl J Med. 2022;387:332–344
† Norton T et al. N Engl J Med. 2026 [In Press]

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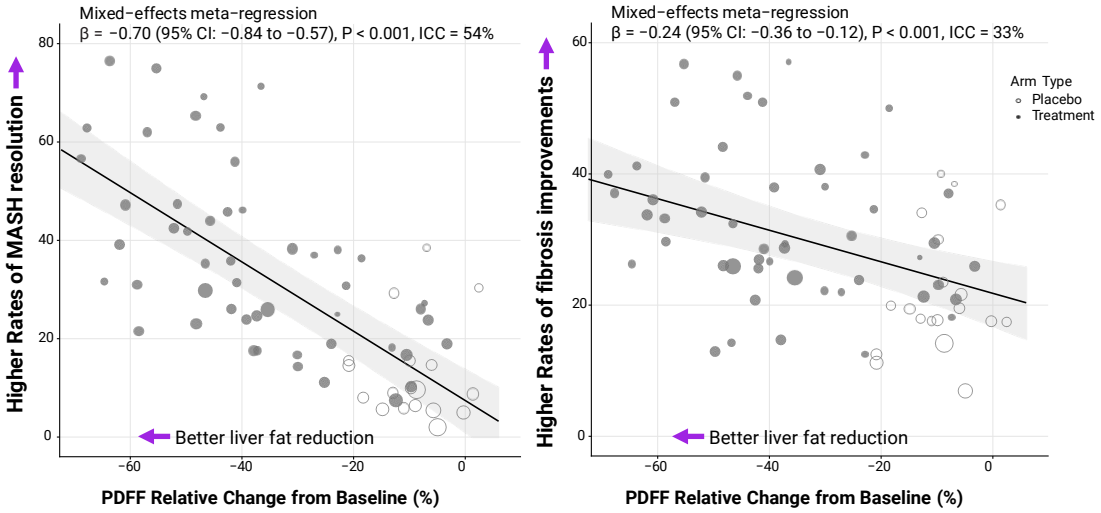
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Meta-analyses of investigational agents link liver fat reduction to histological improvement; CIDEB genetics show lower liver disease risk

A generally well-tolerated therapy that induces deep and sustained reductions in liver fat is anticipated to improve liver histology and reduce liver-related outcomes for people with MASLD/MASH

Hypothesis: A therapeutic that reduces hepatic CIDEB expression mimicking the protective allele may reduce liver fat and improve liver health, preventing the progression of steatotic liver diseases

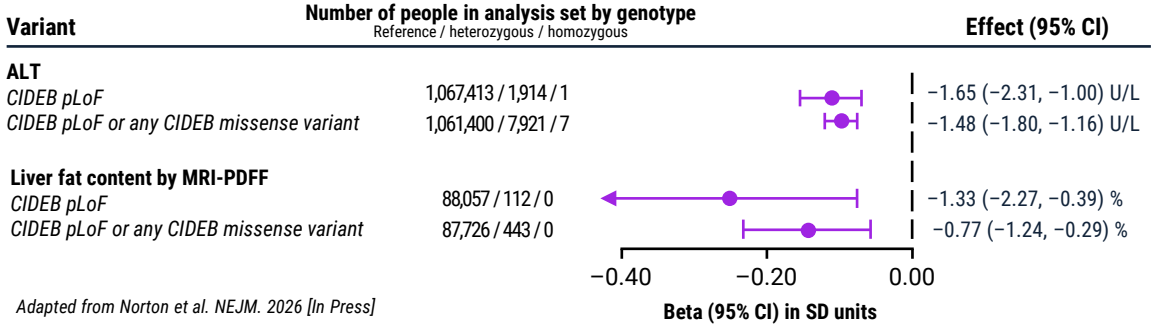
Meta-analyses of clinical trials of patients with MASH indicate that the depth of liver fat reduction correlates with liver biopsy response (e.g., MASH resolution and fibrosis improvement), supporting changes in liver fat as an informative early pharmacodynamic readout



Causal relationship between liver fat reduction and clinical outcomes in human genetic studies and randomized clinical trials of metabolic-dysfunction associated steatohepatitis
Verweij N, et al. MedRxiv 2026.09.27.26364101

RGC

Carriers of rare CIDEB LoF variants (protective allele in ~1/500 individuals) have less liver fat, lower ALT and lower risk of MASLD/MASH



Adapted from Norton et al. NEJM. 2026 [In Press]

Heterozygous carriers of rare LoF variants for CIDEB have 43% lower odds of MASLD/MASH, and 43% lower odds of MASLD-related cirrhosis

MRI-PDFF, magnetic resonance imaging-derived proton density fat fraction; PDFF, Proton Density Fat Fraction; ALT, alanine aminotransferase

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CIDEB^{siRNA}: Phase 1/2 First-in-Human PoC study

Two-part study to evaluate safety and tolerability, with liver fat and markers of liver injury evaluated as secondary endpoints

Part A – MASLD Patient Population

- BMI ≥30 kg/m^2
- Liver fat ≥8.5% by MRI-PDFF
- Prohibited GLP-1 agonists, weight-loss drugs, and approved MASH therapies

Part A: 36 Weeks

R

Single Dose

CIDEB^{siRNA} (single SC dose)
(5 single ascending dosing cohorts)

~3:1, n=73*

Placebo

MRI-PDFF: Baseline, W6, W12, W24, & W36

Part B – MASH Patient Population

- Histologically-confirmed MASH: F0-F3 (F1-F3 US); NAS≥3
- Liver fat ≥8.5% by MRI-PDFF
- Prohibited GLP-1 agonists, weight-loss drugs, and approved MASH therapies

Part B: 48 Weeks

R

2 Doses (12 weeks apart)

CIDEB^{siRNA} (two SC doses 12 weeks apart)
(2 cohorts)

~3:1, n=45

Placebo

MRI-PDFF: Baseline, W6, W12, W24, W36, & W48
Biopsy Timepoints: Different post-treatment biopsy timepoints (6 weeks and 24 weeks after 2nd dose) to understand pharmacodynamics of CIDEB mRNA reduction

Primary Endpoints (Safety):
Incidence and severity of TEAEs through the end of the study

Secondary Endpoints:
Change from baseline in liver fat content by MRI-PDFF, plasma drug concentration, change in liver enzymes

Part A results featured at the recent AASLD Liver Meeting; Part B ongoing

TEAEs, Treatment Emergent Adverse Events; MRI-PDFF, Magnetic Resonance Imaging-Proton Density Fat Fraction; ClinicalTrials.gov ID:NCT06836609
* Data from first 40 patients presented at AASLD Liver Meeting

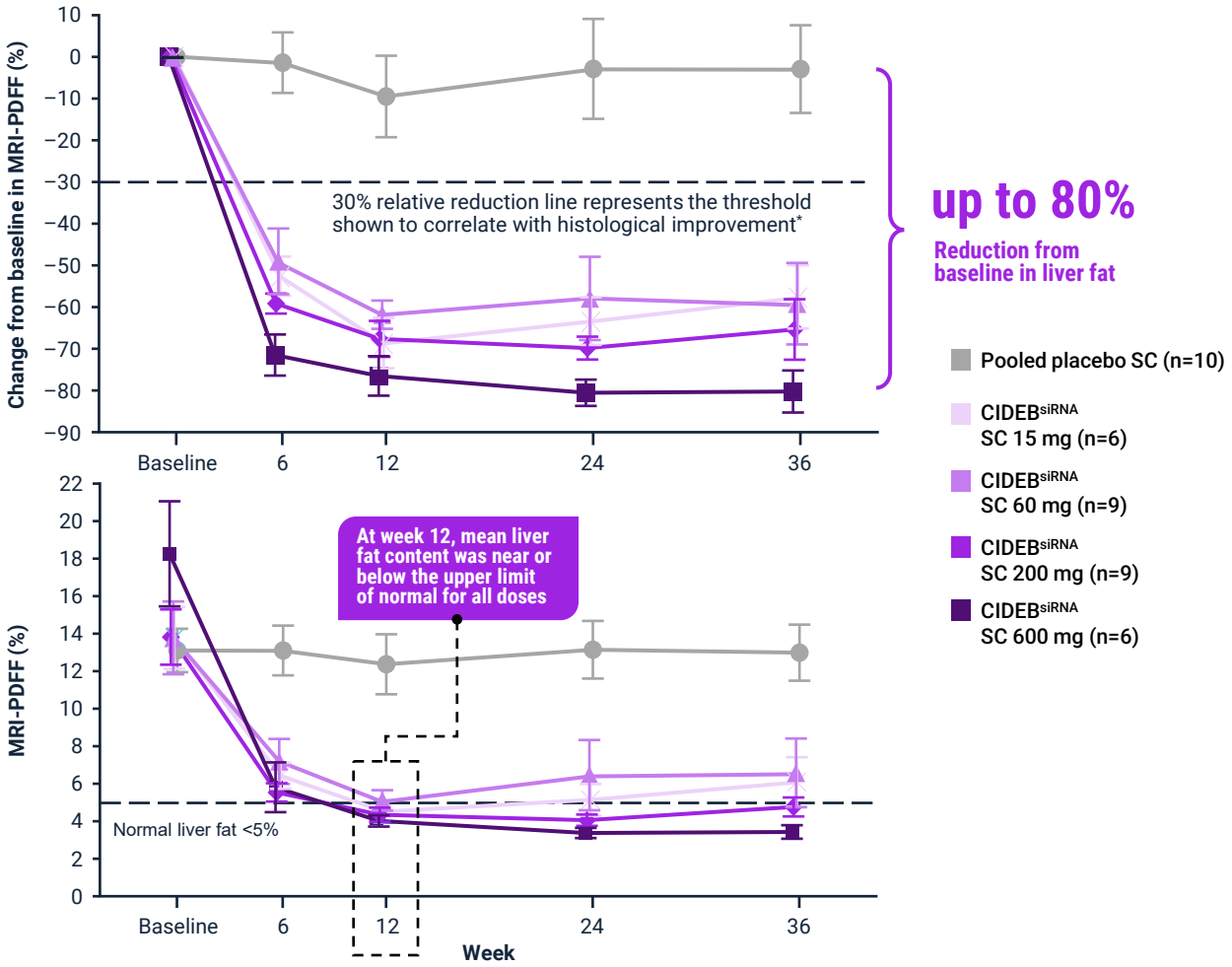
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Single doses of CIDEB^{siRNA} induced rapid, deep and durable reductions in liver fat in an all-comers MASLD population

AASLD The Liver Meeting® NOV. 5–9, 2026

One CIDEB^{siRNA} dose normalized liver fat in majority of participants; with nearly all exceeding the 30% reduction threshold previously linked to histologic improvement



Rapid, deep, and durable reduction in liver fat predicted to translate better histological efficacy than current standard of care

at **Week 12**

mean liver fat levels were near or below the upper limit of normal (5% by MRI-PDFF) for all CIDEB^{siRNA} dose levels

- 58-80% dose-dependent liver fat reductions at week 36, which had deepened from the rapid 49-72% reductions at week 6
- Liver fat normalized (fat content under 5%) in the majority (62%) of CIDEB^{siRNA} recipients at week 36
- 93% of participants had reductions of liver fat of ≥30%, a threshold linked to histological MASH resolution
- Liver fat reductions occurred without meaningful body weight changes in CIDEB^{siRNA} groups

Data are presented as mean ± SE; MRI-PDFF data were missing for 3 participants at certain timepoints; 1 participant in the 15 mg group at Week 12, 1 participant in the 60 mg group at Weeks 24 and 36, and 1 participant in the 600 mg group at Week 6. * Stine JG, et al. Clin Gastroenterol Hepatol. 2021;19:2274–2283.
MRI-PDFF, magnetic resonance imaging-proton density fat fraction; SC, subcutaneous; siRNA, small interfering RNA; ULN, upper limit of normal.

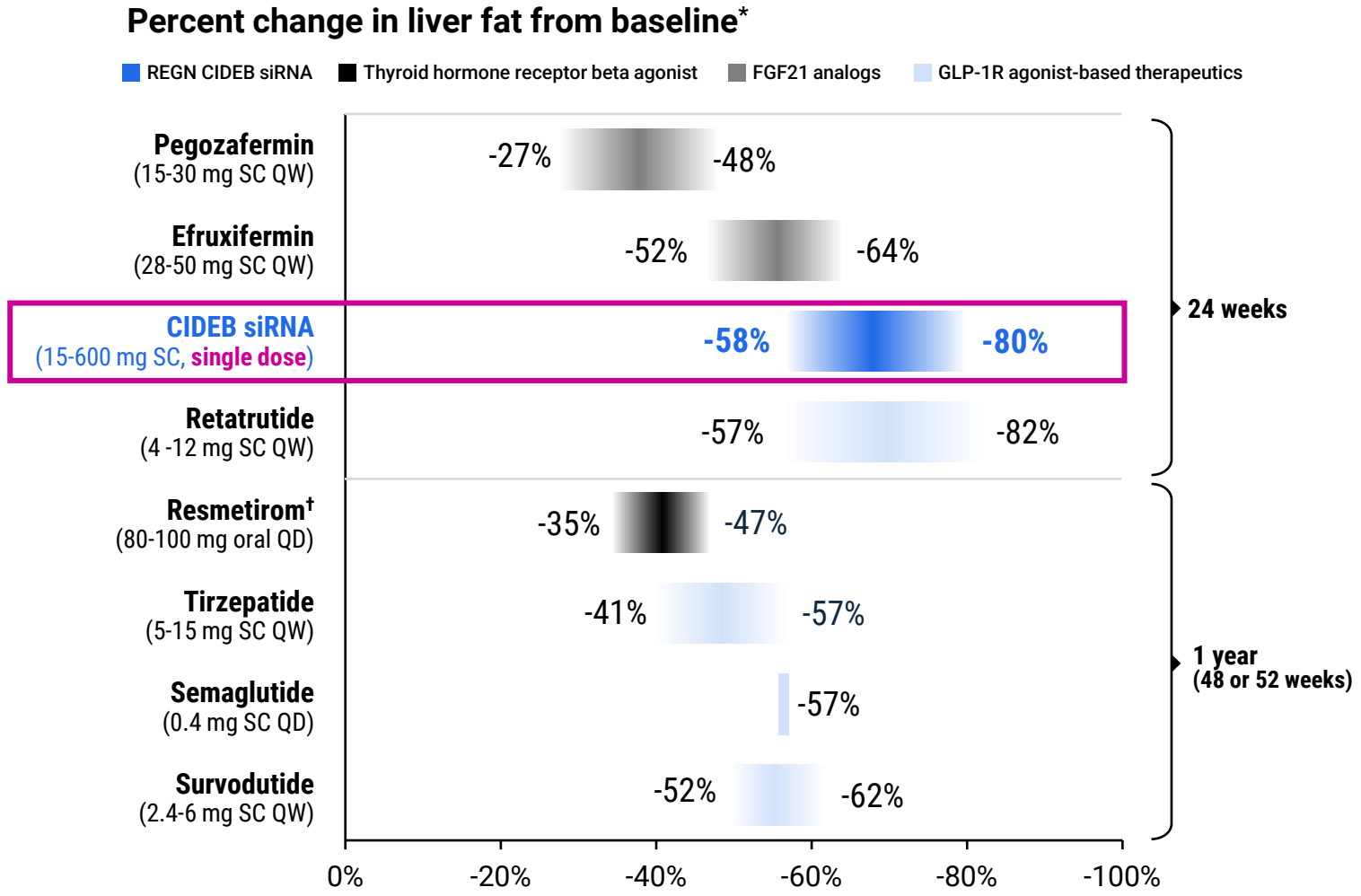
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Single dose of CIDEB^{siRNA} demonstrated 58-80% dose-dependent liver fat reductions at week 24, suggesting best-in-disease potential

CIDEB^{siRNA} liver fat reductions at week 24 appear comparable to or better than other approved or development-stage agents for MASH at similar or later timepoints

*There are no randomized, head-to-head clinical trials between these products. Available data is being provided for descriptive purposes only. Caution is advised when drawing conclusions based on cross-trial comparisons.



† Data from pivotal trial

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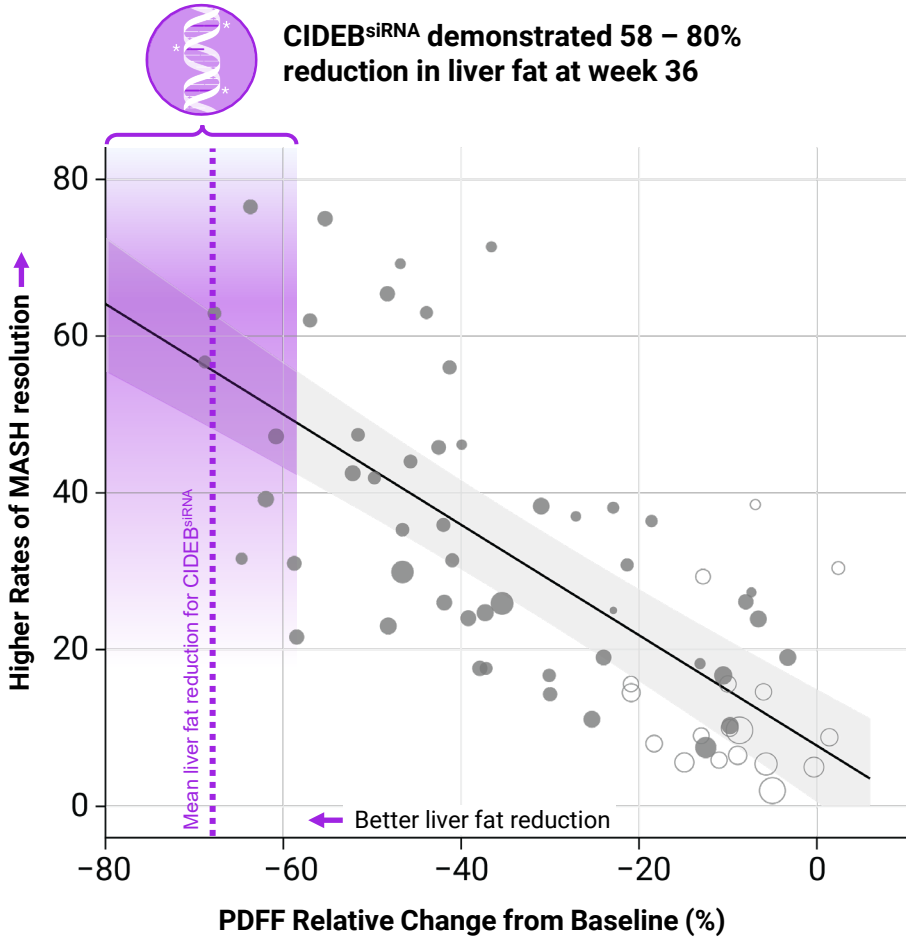
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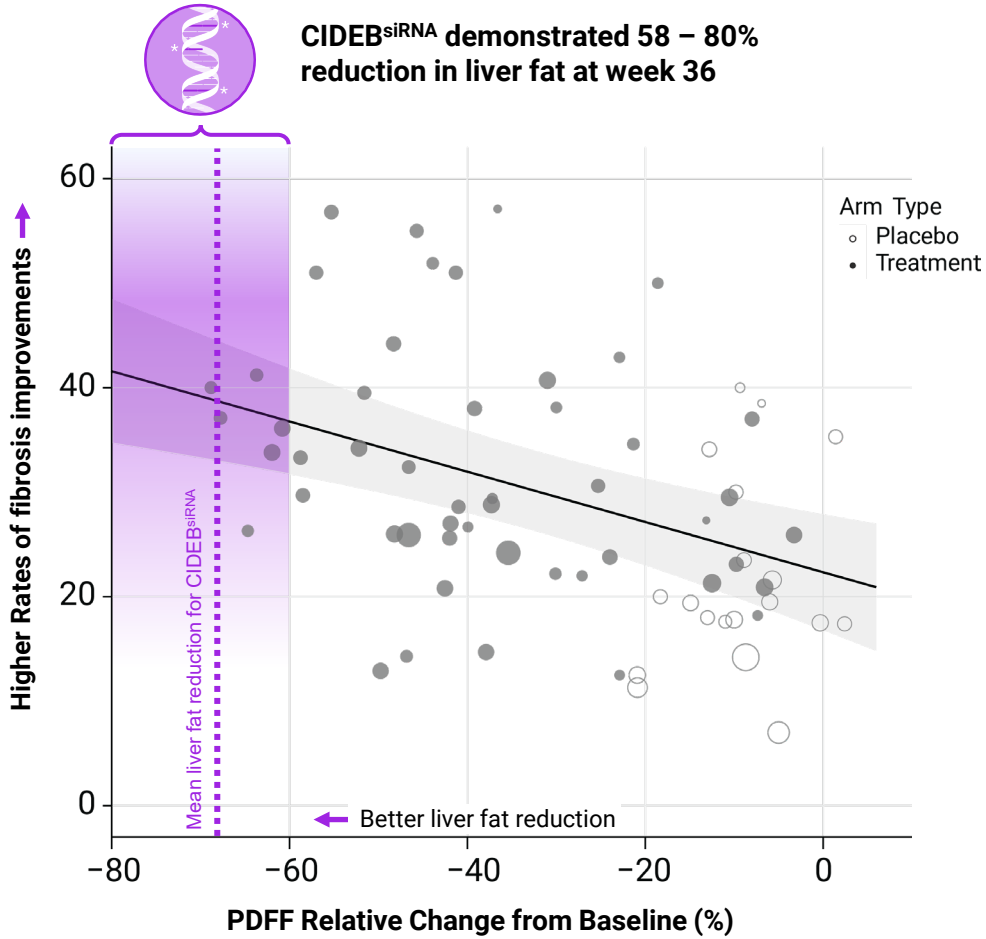
Deep, sustained liver fat reductions demonstrated with CIDEB^{siRNA} support potential to improve MASH resolution and fibrosis

Meta-analyses of clinical trials of patients with MASH indicate that the depth of liver fat reduction correlates with liver biopsy response (e.g., MASH resolution and fibrosis improvement), supporting changes in liver fat as an informative early pharmacodynamic readout

CIDEB^{siRNA} Efficacy After a SINGLE DOSE:

- 58-80% liver fat reductions at week 36
- 68% mean liver fat reduction across all dose levels
- Normalized liver fat in 62% of participants at week 36
- 93% of participants had reductions of liver fat of ≥30%



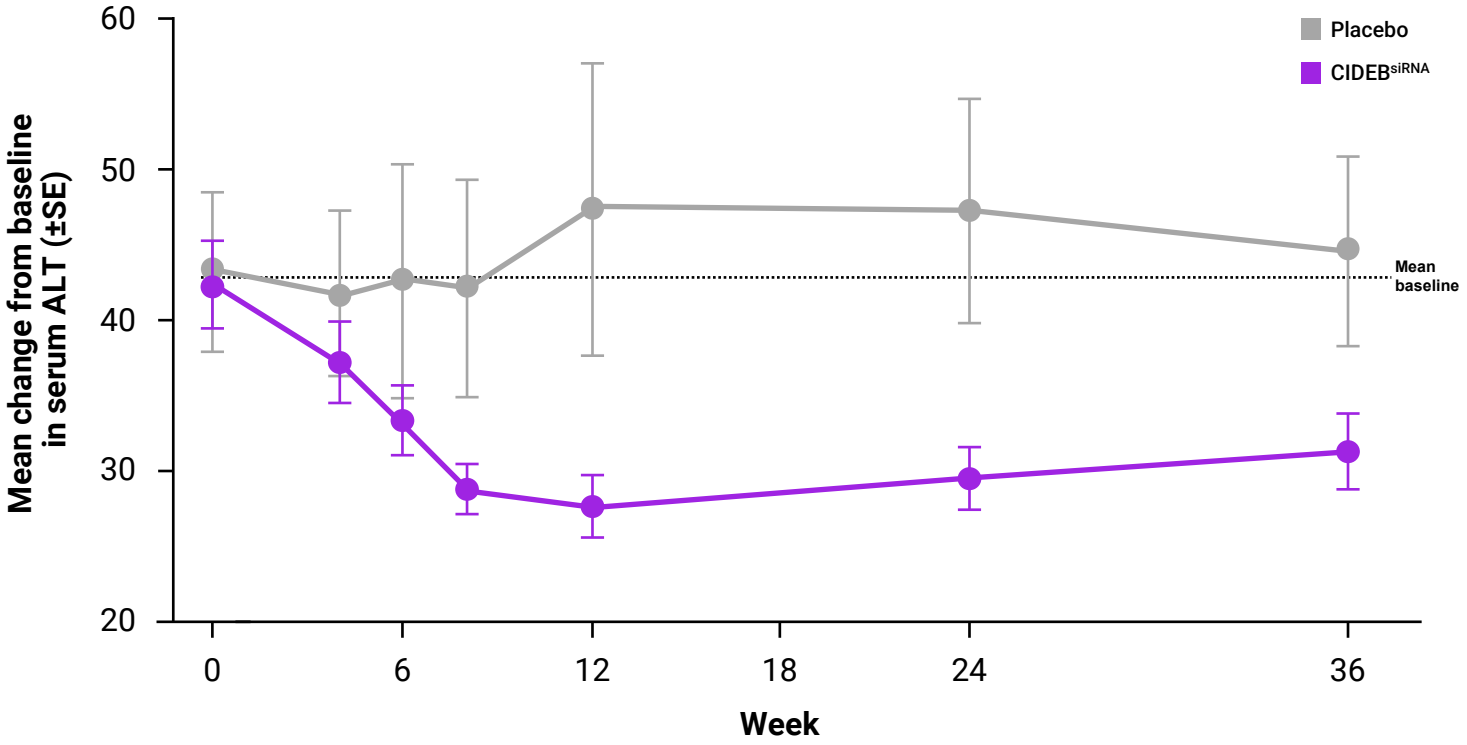


Adapted from Causal relationship between liver fat reduction and clinical outcomes in human genetic studies and randomized clinical trials of metabolic-dysfunction associated steatohepatitis
Verweij N, et al. MedRxiv 2026.09.27.26364101

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Rapid and sustained ALT reductions (secondary endpoint) signal improvement in liver injury at early time points and were sustained through week 36



- At Week 12, mean serum ALT levels for CIDEB^{siRNA} declined from baseline by 32%, compared to a 5% increase from baseline in the placebo group; the effects were sustained through Week 36
- Modest reductions in serum AST levels were also observed

Data are presented as mean ± SE; ALT, alanine aminotransferase; AST, aspartate aminotransferase

This slide contains investigational drug candidates that have not been approved by any regulatory authority.

CIDEB^{siRNA} was generally well-tolerated, with an acceptable safety profile through 36 weeks of follow up

Treatment Emergent Adverse Events (AE) n (%)	Placebo (N=10)	Total CIDEB^{siRNA} (N=30)
Any AEs	9 (90)	26 (87)
Serious AEs	0	1 (3)
Severe AEs	0	1 (3)
AE related to study drug	0	3 (10)
AE leading to study discontinuation or death	0	0
AE occurring in ≥2 participants in any dose level or placebo n (%)		
Upper respiratory tract infection	2 (20)	6 (20)
Toothache	2 (20)	0
Increased appetite	2 (20)	1 (3)
Headache	0	3 (10)
AST increased	1 (10)	2 (7)
Back pain	0	3 (10)
Type 2 diabetes (worsening of)	1 (10)	3 (10)

- Similar adverse event rates between placebo and CIDEB^{siRNA} groups
- No nausea or vomiting reported in the CIDEB^{siRNA} groups, a key tolerability limitation associated with other therapies
- All adverse events were mild or moderate in severity, except for a single severe, serious event of hyperglycemia in a CIDEB^{siRNA} treated participant and not attributed to study drug (participant diagnosed with adult-onset Type 1 diabetes)

AST, aspartate aminotransferase; siRNA, small interfering RNA; AE, adverse event.

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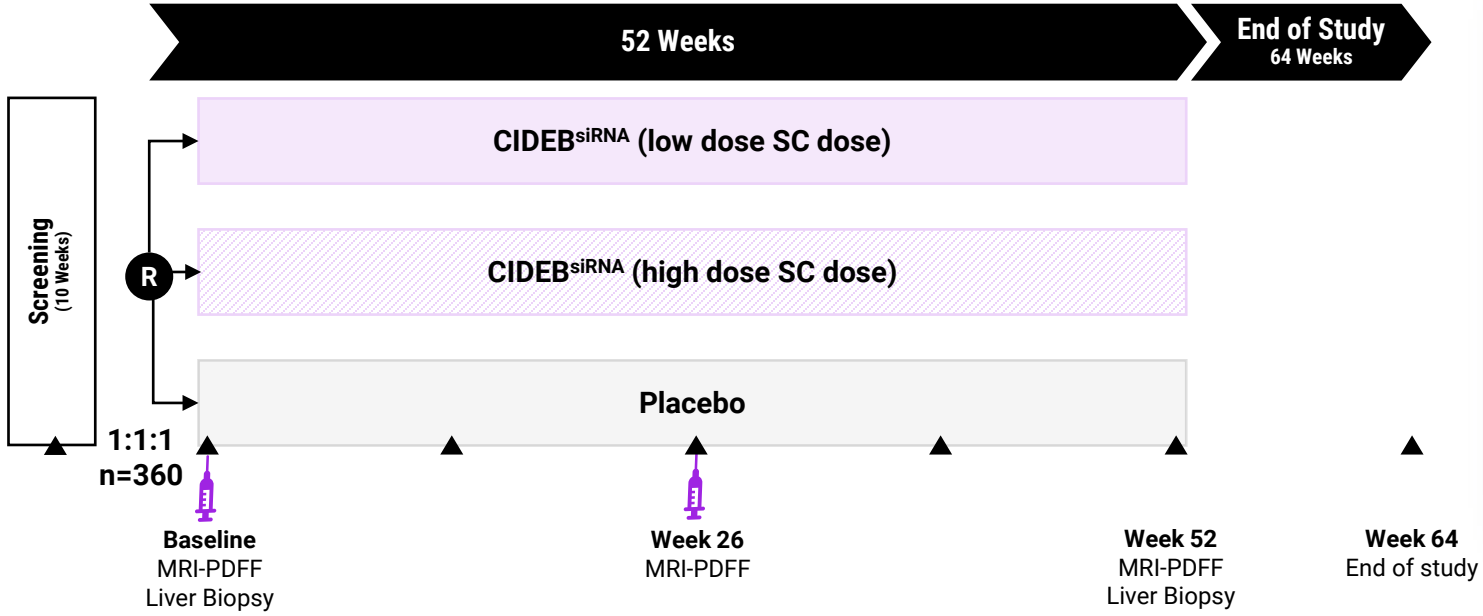
CIDEB^{siRNA} Phase 2b Fibrotic MASH (F2-F3) study initiated

Phase 2b study with registrational intent underway in liver biopsy-confirmed F2 or F3 MASH patients, with final data expected in 2029

MASH Patient Population

- Histologically-confirmed MASH: F2 or F3; NAS ≥4
- Liver fat ≥8.5% by MRI-PDFF
- Patients may be on a GLP-1 based therapy or approved MASH medication (generally stable dose and regimen and not anticipated to change during treatment period)

▲: FibroScan



Primary Endpoint:

- Percent change from baseline in liver fat by MRI-PDFF at week 52

Key Secondary Endpoints:

- Resolution of MASH with no worsening of fibrosis at week 52
- Fibrosis improvement with no worsening of MASH at week 52

1. **Registrational pathway for F2-F3 MASH subject to regulatory feedback**
2. **Phase 3 safety and non-invasive endpoint study in MASLD population initiated**

MRI-PDFF, Magnetic Resonance Imaging-Proton Density Fat Fraction; ClinicalTrials.gov ID:NCT07631637

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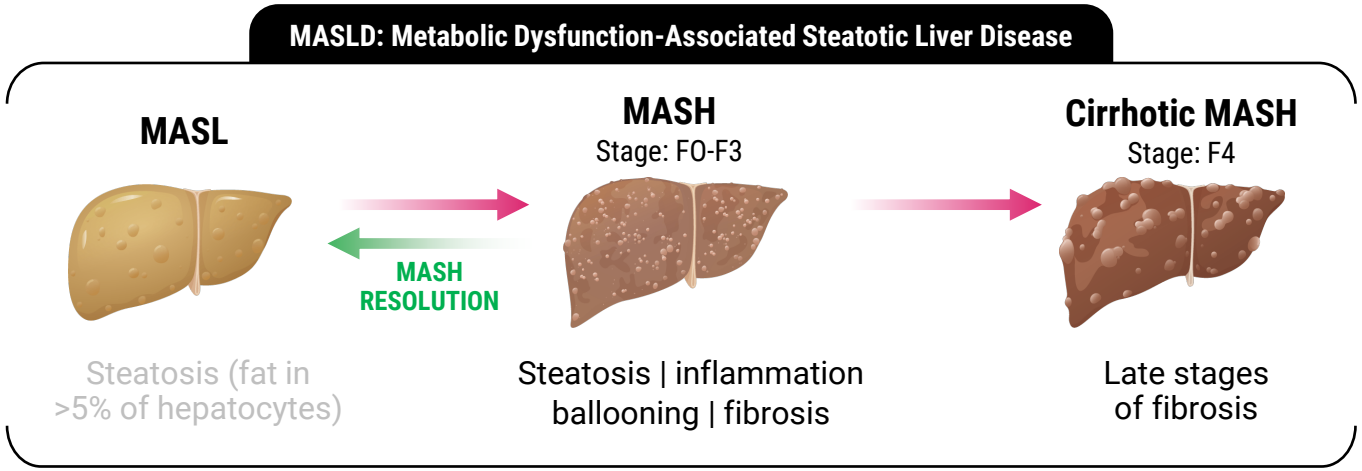
CIDEB^{siRNA} : A potential first-in-class therapy for an all-comers MASH population

CIDEB^{siRNA}'s Differentiated Clinical Profile

Efficacy: Rapid, deep, and durable reductions in liver fat predicted to translate better histological efficacy vs. current and investigational therapies

Safety: Generally well-tolerated with no nausea or vomiting AEs reported

Convenience: Every 6-month dosing supports better long-term adherence



CIDEB^{siRNA} Development Plan

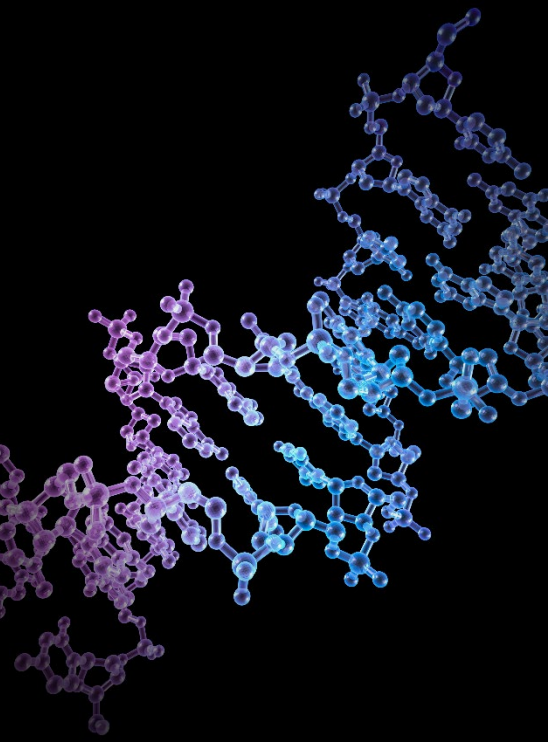
(Subject to regulatory feedback)

	Target Enrollment	Study	Phase 1	Phase 2	Phase 3	Anticipated final data Readout
Proof of Concept (MASLD/ MASH)	118	ALN-CIDEB-NASH-2486 NCT06836609	Phase 1/2			
Fibrotic MASH (F2-F3)	360	ALN-CIDEB-MASH-2603 (CIDEB^{siRNA} vs. Placebo) NCT0763163	Phase 2b – initiated			2029
MASLD (Safety – non-biopsy data)	1500	ALN-CIDEB-MASLD-2604 (CIDEB^{siRNA} vs. Placebo) NCT07854782	Phase 3 – initiated			2032
Cirrhotic MASH (F4 – Cirrhosis)	TBD	ALN-CIDEB-MASH (CIDEB^{siRNA} vs. Placebo)	Phase 3 – 2027 initiation			TBD
Alcohol-Associated Liver Disease (ALD)	120	ALN-CIDEB-ALD-25113 (CIDEB^{siRNA} vs. Placebo) NCT Pending	Phase 2 – 2026 initiation			2029

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Regeneron's Leadership RGC / MASH



George Yancopoulos, MD, PhD
Co-founder
Board co-Chair
President
Chief Scientific Officer

Regeneron's scientific differentiation starts with human genetics and proteomics

- Large-scale database links multi-omics with health data to accelerate drug discovery, as illustrated by the identification of protective CIDEB genetic variants and the MASH clinical pipeline

Aris Baras, MD
Senior Vice President,
Head of Regeneron Genetics Center, Co-Head of Regeneron Genetic Medicines

Regeneron Genetics Center (RGC) provides competitive advantage across discovery and development

- Human genetics helps identify promising targets and guide development decisions across diseases
- AI and multi-omics build on RGC's growing database to accelerate research and support new diagnostic opportunities

Gary Herman, MD
Senior Vice President,
Clinical Development Unit Head,
Genetic Medicines

Translating genetically validated targets into clinical candidates

- MASH siRNA pipeline built on RGC's genetic discoveries and validation
- CIDEB^{siRNA} is rapidly advancing into registration-enabling trials, supported by encouraging clinical results



Josh Adelson
Executive Director
Commercial
New Products

A large and growing MASH commercial opportunity

- MASH is a large, growing market with significant unmet need
- CIDEB^{siRNA} could offer a first-in-class treatment for a broad MASH population, pending clinical validation

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MASH is a large patient opportunity with significant unmet need

FDA-approved therapies currently limited to fibrotic MASH (F2-F3), investigational agents may shape the future treatment landscape

MASH: Metabolic Dysfunction-associated Steatohepatitis

	F1 (Mild Fibrosis) ⟷	F2 (Significant Fibrosis) ⟷	F3 (Significant Fibrosis) ⟷	F4 (Cirrhosis)
U.S. MASH Population*	**30 – 40%** of U.S. MASH pts. (4.0 – 5.5M)	**20 – 25%** of U.S. MASH pts. (2.5 – 3.5M)	**10 – 20%** of U.S. MASH pts. (1.5 – 2.5M)	**5 – 15%** of U.S. MASH pts. (0.75 – 2.0M)
Therapeutic Unmet Need	• Steatosis with mild fibrosis, no cirrhosis • Patients may be considered lower risk and followed-up infrequently	• Pronounced fibrosis with increased steatosis (30 – 60% liver fat), where reversal may be more challenging • Fibrosis progression likely assessed at least once per year for patients receiving approved therapies		• Liver cirrhosis with or without decompensating events (e.g., ascites, variceal hemorrhage, or encephalopathy)
Approved Therapies	Rezdiffra™ resmetirom tablets 60mg · 80mg · 100mg	wegovy® semaglutide injection		
Investigational Agents (MOA)	Efimosfermin α *(FGF-21)* Survodutide *(Dual GLP-1)*	Efruxifermin *(FGF-21)* Pegozafermin *(FGF-21)*	Regeneron's MASH siRNAs *CIDEB^{siRNA} & PNPLA3^{siRNA}*	ION-224 *(DGAT2 Inhibitor)* Lanifibranor *(Pan-PPAR)* Resmetirom *(THR-β)* GSK-4532990 *(RNAi)*

Key: ⟷ Reversible ⟷ Irreversible

~13 million U.S. adults are currently living with MASH* *(~5% prevalence*)*

- **Obesity is highly prevalent** among people with MASH, with **~80% of MASH patients** are living with obesity or overweight
- **We expect GLP-1 use among MASH patients to increase,** but real-world evidence suggests **~3 out of 4 (72%) patients discontinued GLP-1-based therapy by 2 years†**

* Estes et al J of Hepatology 2018, American Liver Foundation, Regeneron Internal Analysis
† Rodriguez, P, et al. Journal of the American Medical Association 2025

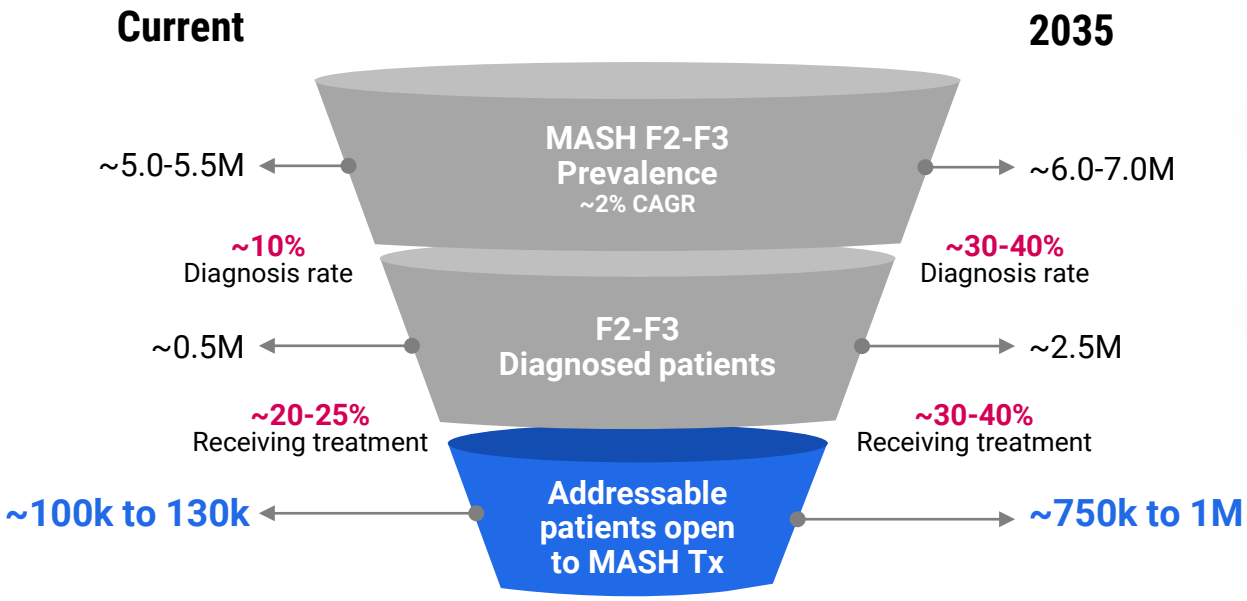
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U.S. MASH market expected to grow rapidly driven by advanced therapies and increased diagnosis/treatment rates

MASH patients on treatment

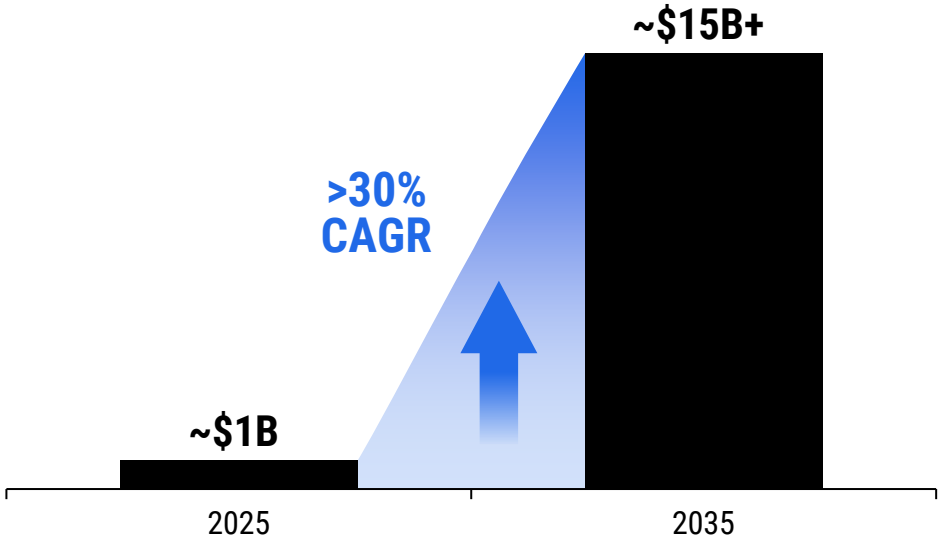
% of patients on MASH-specific treatment anticipated to increase



U.S. MASH net sales

Fibrotic MASH (F2-F3) and cirrhotic MASH (F4) markets

Commercial opportunity expected to significantly increase over the next decade



Increasing usage of approved agents and potential approval of investigational agents will shape the MASH treatment landscape and could materially expand the addressable market

*Sources: Evaluate Pharma, Clarivate

A large unmet need persists in MASH, with room to improve on efficacy, tolerability, and convenience

	Resmetirom THR-β, Ph 3	Semaglutide GLP-1, Ph 3
Market Entry	**2024**	**2025**
Fibrosis Improvement without MASH worsening*	**12%**	**14%**
MASH Resolution without fibrosis worsening*	**20%**	**29%**
Safety & Tolerability	Diarrhea, nausea, pruritus, abdominal pain, vomiting, dizziness, constipation	Boxed warning for thyroid c-cell tumors Nausea, diarrhea, fatigue, vomiting
Dosing & Admin	**Oral** **Daily** (365 doses per year)	**SC** **Weekly** (52 doses per year)

Key Unmet Need

Greater antifibrotic benefit with faster onset

- Current therapies provide modest fibrosis improvement in fibrotic MASH (F2–F3) MASH, with no approved treatments for cirrhotic MASH (F4)

*"I would say that the **current two therapies** that are approved are exciting, but **not groundbreaking**... there's a **lot of room for improvement**."*

– Community Hepatologist

Improved safety and GI tolerability

- GI side effects can lead patients to discontinue treatment or prevent them from reaching GLP-1 doses needed for MASH benefit

*"Some people don't even get to the therapeutic dose [of GLP-1s] because of **GI side effects**... they may not achieve the weight loss signals that we're looking for."*

– Hepatology KOL

Subcutaneous dosing twice per year

- Less frequent administration could reduce treatment burden and support long-term adherence

*"I think that would be **very, very attractive** to both patients and providers. You [patients] come in **twice a year** for your liver clinic appointment, and then you get your treatment as well."*

– Community Hepatologist

* Placebo-adjusted efficacy.
Source: Prescribing Information of approved agent

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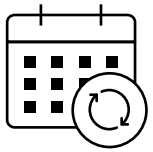
CIDEB^{siRNA} has the potential to be a novel, highly differentiated MASH therapy in a rapidly expanding market

Significant opportunity if CIDEB^{siRNA} delivers best-in-class efficacy and safety while offering all-comers eligibility and infrequent dosing



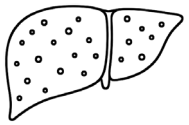
Efficacy & Safety Profile

- CIDEB^{siRNA} has first-in-class and best-in-disease potential for both fibrosis and MASH resolution with fast onset of action
- Favorable tolerability without GI side effects could be a key differentiator from approved and investigational therapies



Infrequent Dosing

- Twice-a-year dosing adds patient convenience and supports better adherence
- Timing of dosing administration is aligned with patients' routine office visit cadence



Liver-Targeted siRNA

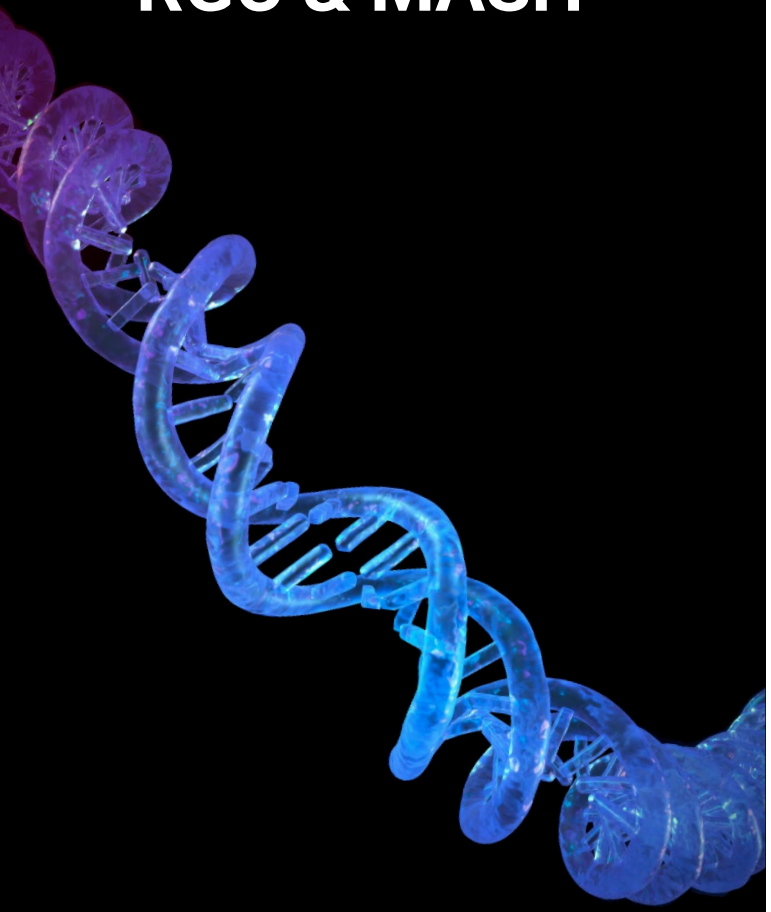
- Broad all-comers applicability across the fibrotic (F2-F3) and cirrhotic (F4) MASH treatment landscape
- Potential for CIDEB^{siRNA} to be used as a monotherapy or alongside GLP-1-based therapies

CIDEB^{siRNA} could bring a first-in-class, broadly applicable treatment to MASH, with the opportunity to set a new standard, pending clinical validation

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Key Roundtable Takeaways: RGC & MASH



RGC drives Regeneron's scientific process

Large-scale human multi-omics database provides significant competitive advantage in discovering important new medicines, validating existing research programs and optimizing clinical trials

Aspiring to revolutionize healthcare using big data

Powered by RGC's genomics and proteomics data, multi-omics has the potential to optimize healthcare management, leading to greater efficiency, lower costs, and better outcomes

MASH pipeline rapidly advancing to late-stage development

Regeneron's genetically-validated MASH pipeline is advancing, with CIDEB^{siRNA} development prioritized for its potential to address a broad patient population

CIDEB^{siRNA} could become a paradigm-changing, first-in-class MASH therapy with best-in-disease potential, pending clinical validation, in a market projected to reach $15B+ over the next decade

RGC's human genetics and proteomics database gives Regeneron a repeatable competitive advantage in drug discovery, with our advancing MASH pipeline validating a model we are applying across a wide range of diseases

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Question & Answer Session

Regeneron Roundtable – RGC & MASH Pipeline



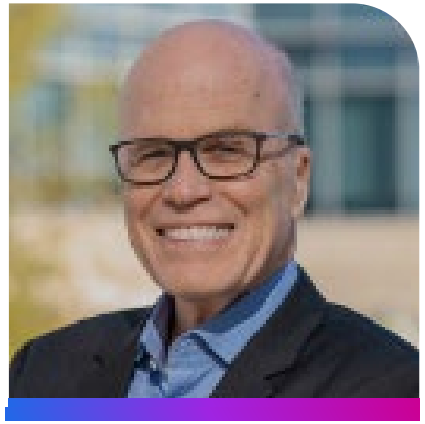
George Yancopoulos, MD, PhD
Co-Founder
Board co-Chair
President
Chief Scientific Officer

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Aris Baras, MD
Senior Vice President,
Head of Regeneron Genetics Center, Co-Head of Regeneron Genetic Medicines

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Gary Herman, MD
Senior Vice President,
Clinical Development Unit
Head, Genetic Medicines

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Josh Adelson
Executive Director
Commercial
New Products

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Royalty and milestone obligations for MASH portfolio

Regeneron will be leading development and commercialization of these programs (Alnylam opted-out)

PNPLA3^{siRNA} (Pivicasiran, ALN-PNP) & HSD^{siRNA} (Rapirosiran, ALN-HSD)		CIDEB^{siRNA} (ALN-CIDEB)	
Royalties Tiered by net sales in a calendar year	Tiered, double-digit royalties (up to 17.5%)	**Royalties** Tiered by net sales in a calendar year	Tiered, double-digit royalties (up to 20%)
Development Milestones	None	**Development Milestones**	$30 million remaining
Commercial Milestones	None	**Commercial Milestones**	Up to $90 million