ALIGOS
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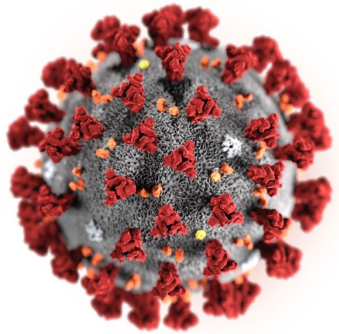

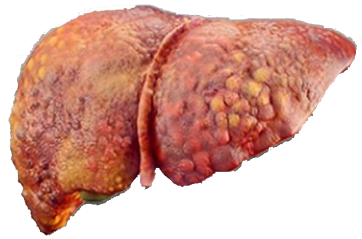

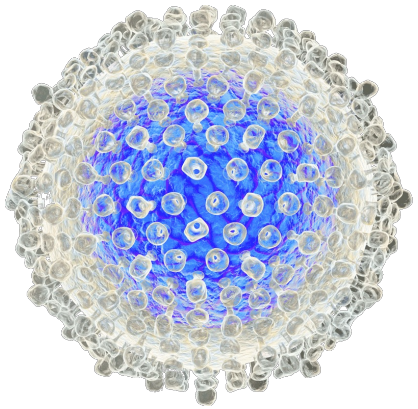

Corporate Presentation

May 2024

Disclosures

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. All statements other than statements of historical facts contained in this presentation, including statements regarding our future results of operations and financial position, business strategy, prospective drugs and drug candidates, the potential scope, progress, results and costs of developing our drug candidates or any other future drug candidates, the potential market size and size of the potential patient populations for our drug candidates, the timing and likelihood of success of obtaining drug approvals, ability to maintain existing, and establish new, strategic collaborations, licensing or other arrangements, plans and objectives of management for future operations, the scope of protection we are able to establish and maintain for intellectual property rights covering our drug candidates, future results of anticipated drugs and drug candidates, and the impact of global events and other macroeconomic conditions on the business are forward-looking statements. These statements involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. Because forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified and some of which are beyond our control, you should not rely on these forward-looking statements as predictions of future events. The events and circumstances reflected in our forward-looking statements may not be achieved or occur and actual results could differ materially from those projected in the forward-looking statements. Except as required by applicable law, we do 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. For a further description of the risks and uncertainties that could cause actual results to differ from those anticipated in these forward-looking statements, as well as risks relating to the business of Aligos Therapeutics in general, see Aligos' Annual Report on Form 10-Q filed with the Securities and Exchange Commission on May 7, 2024, and its future periodic reports to be filed with the Securities and Exchange Commission. Except as required by law, Aligos Therapeutics undertakes no obligation to update any forward-looking statements to reflect new information, events or circumstances, or to reflect the occurrence of unanticipated events.

Except where otherwise indicated, the information contained in this presentation speaks as of the date hereof or as of the date at which such information is expressed to be stated, as applicable, and such information may express preliminary estimated, unaudited results which shall be subject to audit or other year-end adjustments and such audited or adjusted results may materially differ from those contained in this presentation.

This presentation concerns drug candidates, some of which are undergoing nonclinical studies and others of which are under clinical investigation, and all of which have not yet been approved for marketing by the U.S. Food and Drug Administration. These drug candidates are currently limited by federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

Aligos
Investment Thesis

- **Aligos has decades of drug development experience in medicinal chemistry and liver/viral diseases**
- **ALG-055009 for Metabolic Dysfunction-Associated Steatohepatitis (MASH)**
  - Thyroid hormone receptor beta (THR-β) is a clinically validated mechanism (MDGL)
  - ALG-055009 has enhanced pharmacologic properties vs. competitor THR-β agonists
  - Phase 1 data: linear and non-variable PK, well-tolerated, with expected thyromimetic effects
  - Phase 2a enrollment complete with topline data expected in early Q4 2024
- **ALG-000184 for Chronic Hepatitis B (CHB)**
  - ALG-000184 (CAM-E) has enhanced pharmacologic properties and is a best/first-in-class molecule
  - Demonstrated greater DNA suppression compared to standard of care (NAs)
  - Dosing ongoing in 96-week Ph1b cohorts with interim readouts expected at APASL, EASL, AASLD
  - Phase 2 enabling activities ongoing
  - Regulatory discussions underway (superior chronic DNA suppression)

As of 3/31/24 - Cash, cash equivalents and investments were $112.7M. Projected runway through the end of 2025

Aligos Development Portfolio
Multiple Milestones Anticipated in 2024

Candidate	Indication	MOA	2024 Clinical Trial Timelines and Data Readouts
			Q1 2024 · Q2 2024 · Q3 2024 · Q4 2024
ALG-055009	MASH	THR-β Agonist	Phase 2a (12 week MRI-PDFF in MASH) — Topline data ★
Oligonucleotide (including MERCK)	MASH	Undisclosed	Preclinical Activities
ALG-000184	CHB	CAM-E	Phase 1b (Dosing x ≤ 96 Weeks), Phase 2 Enabling Activities — ★ APASL · ★ EASL · ★ AASLD
ALG-097558	Covid-19*	Protease Inhibitor	Phase 2 Enabling Activities (Clinical, Nonclinical) — FIH Topline Data ★

*Our Covid-19 protease inhibitor programs are partly funded (>$12M USD awarded) by the NIH and NIAID's AViDD Centers for Pathogens of Pandemic Concern program through the MAVDA consortium and our recently awarded NIAID Contract.

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AASLD = American Association for the Study of Liver Diseases; APASL = Asian Pacific Association for the Study of the Liver; CAM-E = capsid assembly modulator (empty); CHB = chronic hepatitis B; Covid-19 = coronavirus disease of 2019 (SARS-CoV-2); EASL = European Association for the Study of the Liver; HV = healthy volunteers; MoA = mechanism of action; MRI-PDFF = Magnetic Resonance Imaging Proton Density Fat Fraction; MASH = metabolic dysfunction associated steatohepatitis; ; THR-β = thyroid hormone receptor beta.
All timelines are approximate and subject to change based on enrollment and operational considerations. Presentations at conferences are subject to abstract approvals.

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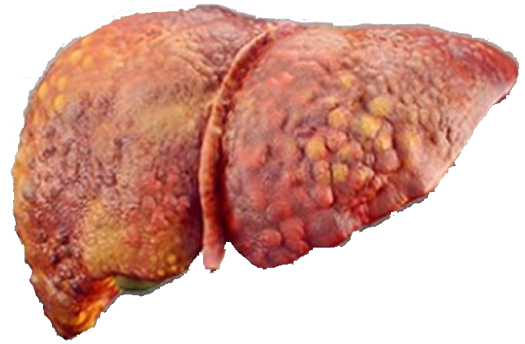
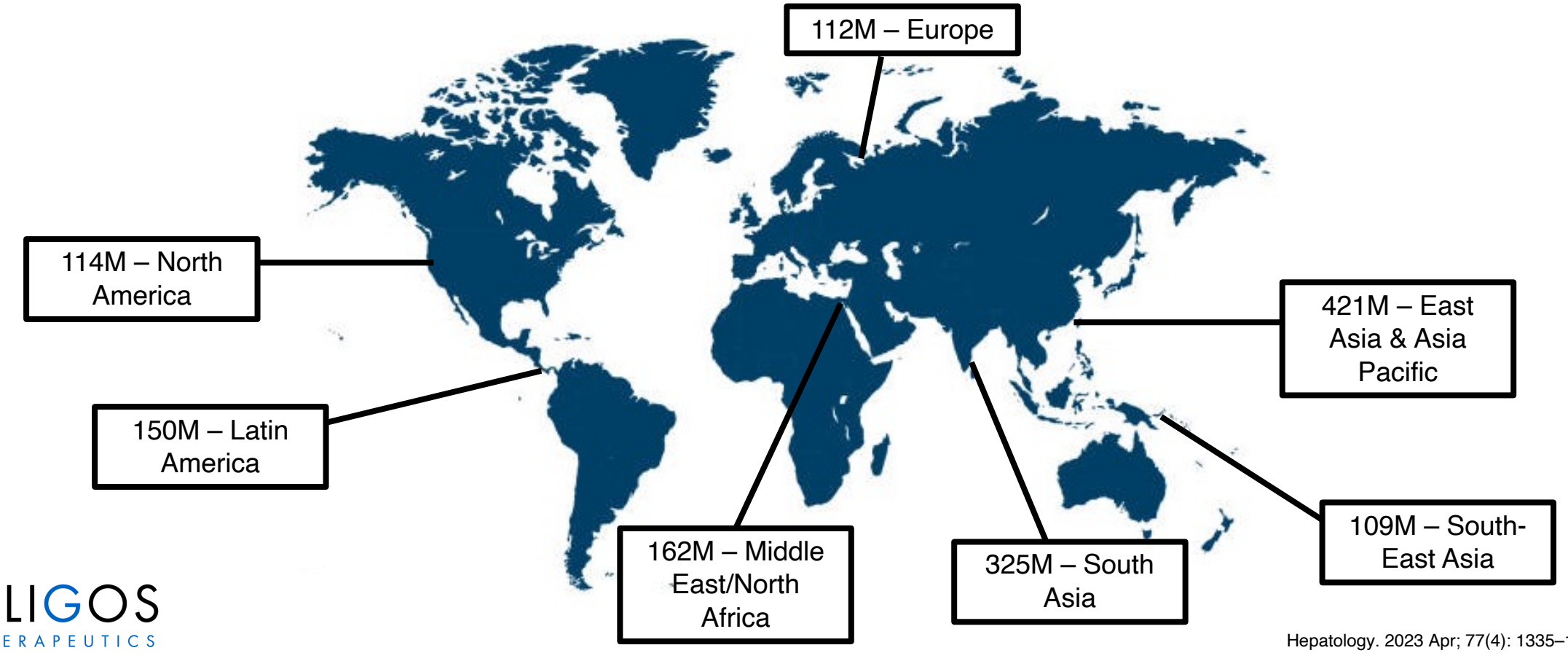
MASH

- Epidemiology, Pathogenesis
- Competitive Landscape



MASLD/ MASH
A Global Disease with Limited Treatment Options

- 1.66B people worldwide living with MASLD/MASH; the highest prevalence in East Asia/Asia Pacific and South Asia
- Global prevalence is 30% and a leading cause of liver-related morbidity including cirrhosis, hepatocellular carcinoma, liver transplant, and end-stage liver disease



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Hepatology. 2023 Apr; 77(4): 1335–1347.

MASH Pathogenesis

Fatty acids = primary source of excess energy supply

(from de novo lipogenesis & lipolysis)

↓

Lipotoxic Species

↓

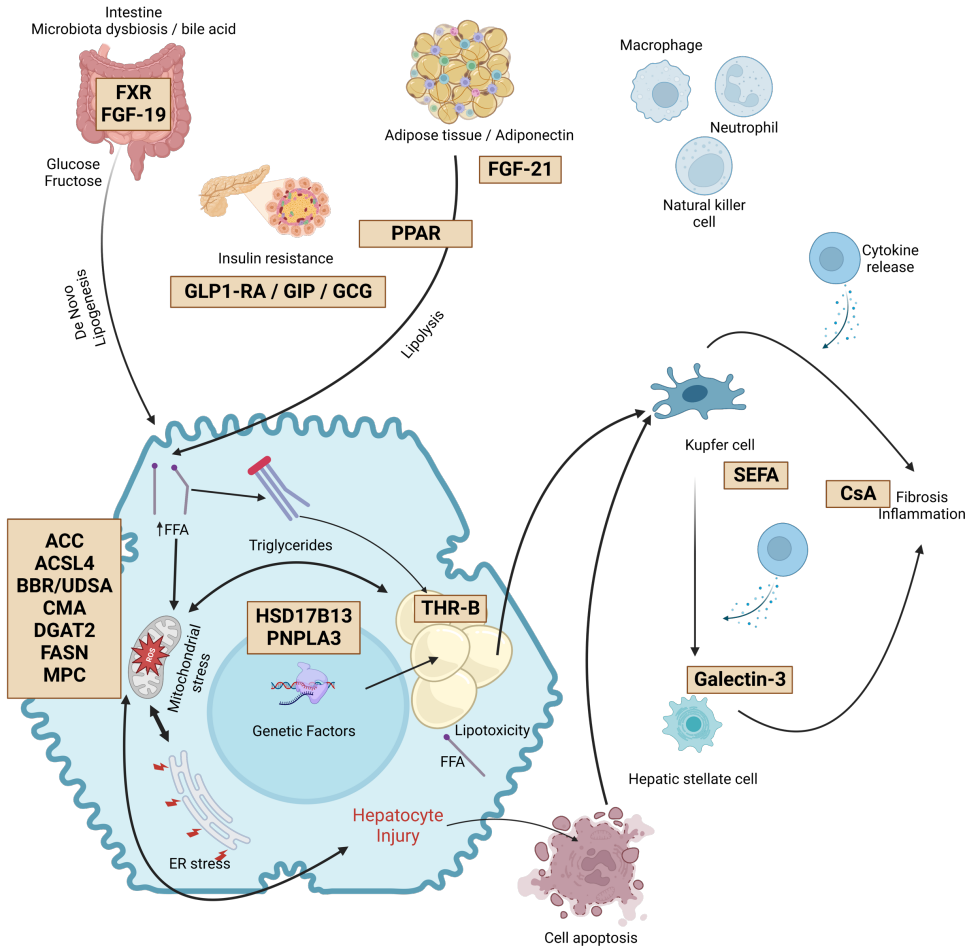
Liver Injury

Endoplasmic reticulum stress

Mitochondrial dysfunction

Pro-inflammatory cytokines release

Apoptosis



MASH biology is complex with multiple therapeutic approaches being evaluated; combination regimens may be required

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Adapted from Stephen Harrison presentation, AASLD 2023.

THR-β is a Differentiated Mechanism in Late-Stage Clinical Development

GLP-1

Pros
- Demonstrated clinical success in MASH resolution and weight loss in Ph2

Cons
- Injectable
- GI tolerability issues
- No clinical evidence of fibrosis improvement presented to date

FGF21

Pros
- Demonstrated clinical success in MASH resolution **and** fibrosis improvement in Ph2

Cons
- Injectable
- GI tolerability issues
- Bone mineral density issues

PPAR

Pros
- Demonstrated clinical success in MASH resolution **and** fibrosis improvement in Ph2
- **Oral**

Cons
- GI, weight gain, and other tolerability issues
- Liver toxicity

FASN

Pros
- Demonstrated clinical success in MASH resolution **and** fibrosis improvement in Ph2
- **Oral**

Cons
- Poor tolerability demonstrated to date: alopecia, dry skin, dry eyes, etc.

THR-β

Pros
- Demonstrated clinical success in MASH resolution **and** fibrosis improvement in Ph3
- **Oral**
- Generally well tolerated
- Cost of goods

Cons
- 1st gen compounds not reaching full efficacy potential

Oral

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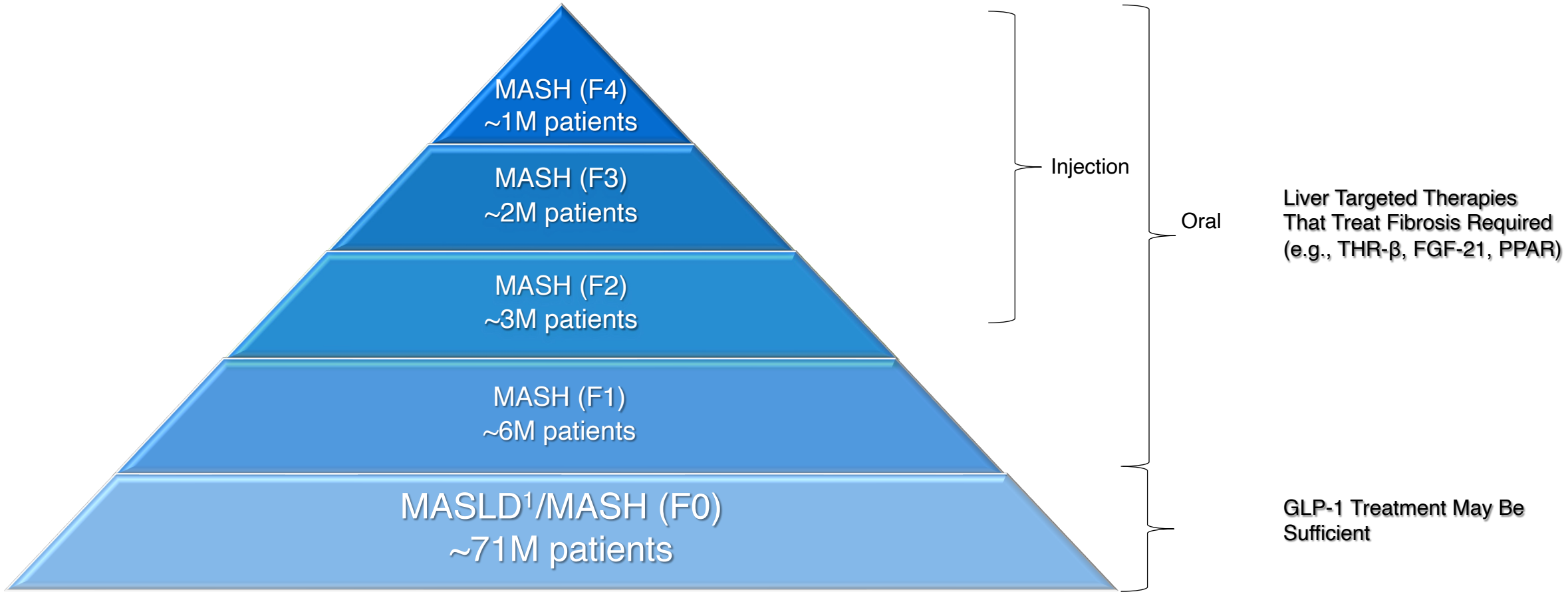
MASLD/MASH Potential Future Treatment Paradigm



Liver-targeted therapies, likely in combination, required for F1-F4 MASH patients for whom typical lifestyle modifications and/or comorbidities treatments are insufficient

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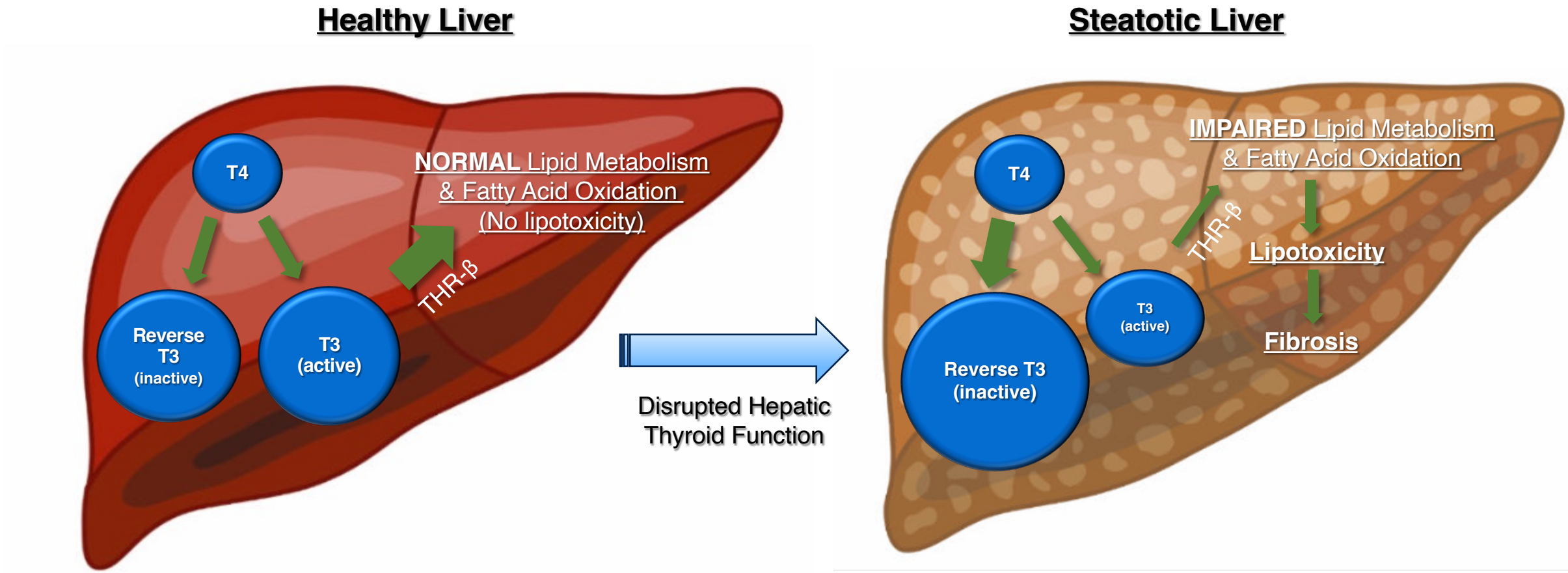
Estimated US Incidence (~3% of Global Incidence)
Source of MASLD/MASH epidemiological data: https://www.pharmaceuticalonline.com/doc/analysis-of-the-non-alcoholic-steatohepatitis-nash-drug-pipeline-market-sizing-up-the-first-wave-0001.

Role of Hepatic Thyroid Dysfunction in MASLD/MASH Pathogenesis



Hepatic hypothyroidism results in reduced active T3 production, allowing increased production of pro-inflammatory lipotoxic fat that causes hepatocellular injury/death, fibrosis, and cancer

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Liver figures adapted from https://www.gblhospital.com/centre-for-excellence/gi-liver-surgeries/advance-liver-institute/fatty-liver/.

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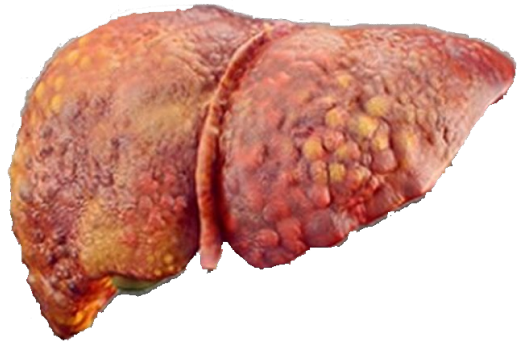
ALG-055009: Small molecule THR-β agonist



ALG-055009
A Potential Best-in-Class THR-β Agonist for MASH

- **Discovered by Aligos; issued US patent expires 2040[1]**
- **Purpose-built with enhanced pharmacologic properties**
  - 5-50x fold more potent
  - More β selective
  - Optimized for PK

  } vs. competitor THR-β agonists
- **Phase 1 highlights**
  - PK - dose proportional, low variability, $t_{1/2}$ ~20 hours (enhanced vs. resmetirom)
  - Safety - well tolerated without clinical safety signals
  - Pharmacodynamics - expected thyromimetic effects (e.g., dose proportional increases in SHBG, decreases in lipids)
- **Phase 2a HERALD study**
  - Evaluating 4 dose levels vs. placebo x 12 weeks - safety, PK, PD (MRI-PDFF)
  - Enrollment complete with topline data expected in early Q4 2024

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[1]Not including any patent term extension.

Enhanced Potency, PK Correlated with Efficacy

Best-in-class potency, PK of ALG-055009 may result in best-in-class efficacy

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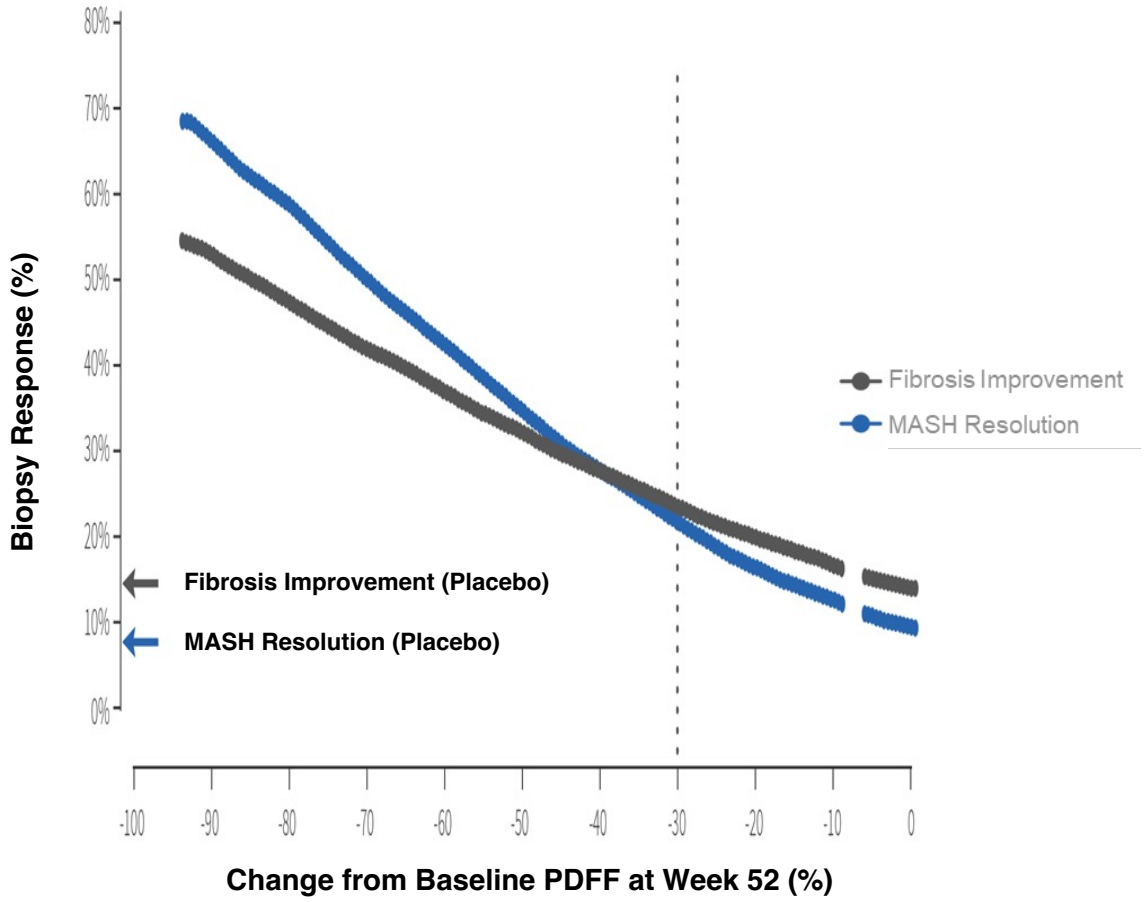
Viking data: Lian et al., ACC 2016 and May 16, 2023, press release. Madrigal data: Taub, Atherosclerosis, 2013; Harrison, EASL, 2023.
TERN-501 data: Quirk et al., TERNS Corporate presentation, August 8, 2023, Nelson et al., EASL 2022 and US 2020/0190064 A1.
Gannex / Ascletis ASC41 data; Ascletis Pharma Inc. press release, January 2, 2024.
[1]EC_{50} HEK293T cell-based assay, nM; ^Same structure as VK-2809 per patent literature/lawsuit.

Resmetirom Phase 3 Data
MRI-PDFF and Liver Biopsy Correlation



THR-β induced MRI-PDFF de-fatting strongly correlated with histologic improvement

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NR = Non-Responder.
Image adapted from Loomba et al, AASLD 2023.

ALG-055009
Phase 1 Study Design

Part 1: Single Ascending Dose (SAD)

N = up to 64 Healthy Volunteers

N = 8 per Cohort, N = 6 ALG-055009 and N = 2 Placebo

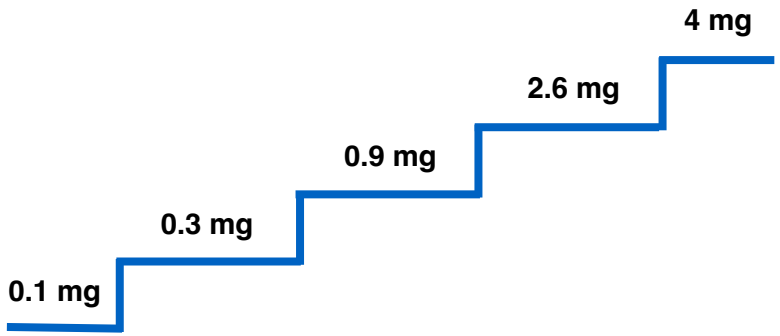
Part 2: Multiple Ascending Dose (MAD) – Dosing PO QD X 14 days

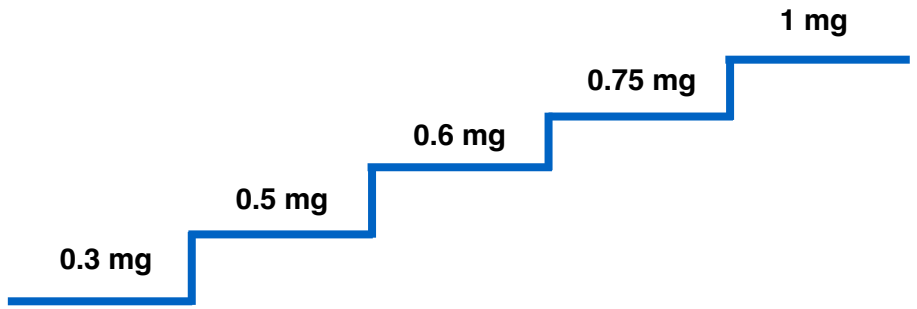
N = up to 80 Subjects with Hyperlipidemia

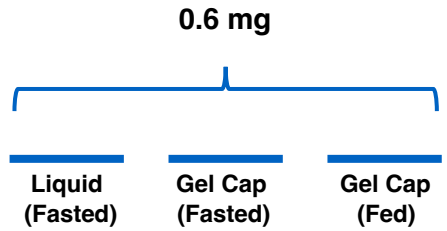
N = 10 per Cohort, N = 8 ALG-055009 and N = 2 Placebo

Part 3: Relative Bioavailability, Food Effect (Gel Cap)

N = 10 Healthy Volunteers







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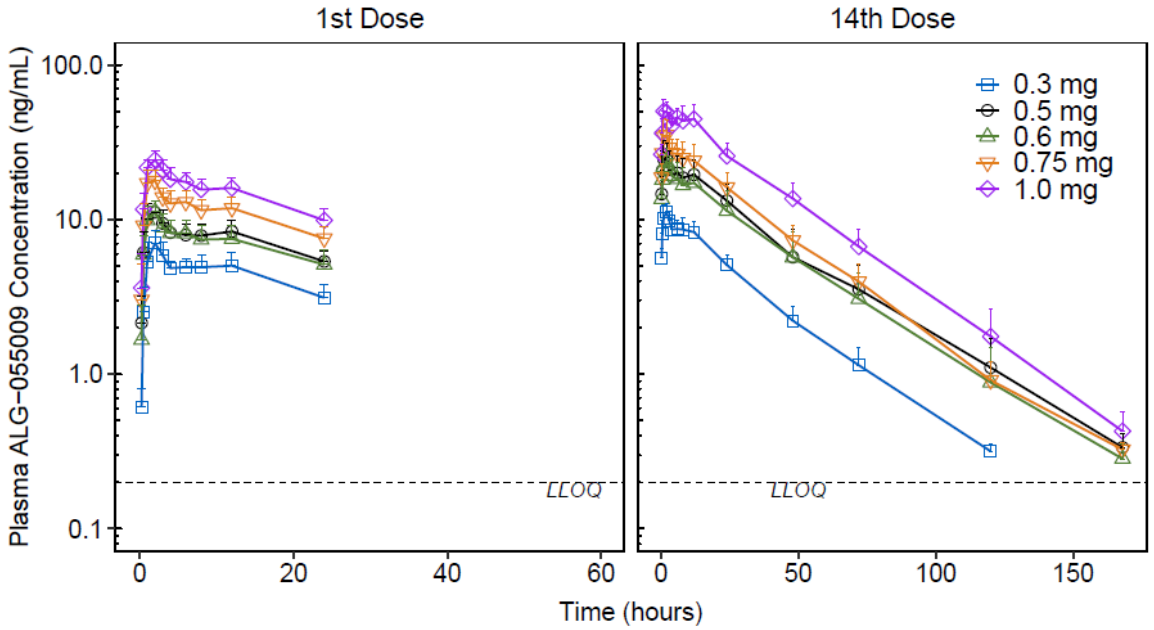
Study ALG-055009-301
Phase 1 Highlights: Doses Well Tolerated with Favorable PK

Single Ascending Dose - PK, Safety, Biomarkers

- **Oral doses evaluated:** 0.1, 0.3, 0.9, 2.6, 4.0 mg
- **PK:** dose proportional, with low variability
  - $t_{1/2}$ = 20-24 hours (supports once daily (QD) dosing)
- **Safety:** well tolerated
  - No serious adverse events (SAEs), Grade ≥3 treatment emergent adverse events (TEAEs), or clinical hyper/hypothyroidism
- **Biomarkers:** expected thyromimetic effects observed

Multiple Ascending Dose - PK, Safety

- **Oral doses evaluated:** 0.3, 0.5, 0.6, 0.75, 1.0 mg QD x 14 d
- **PK:** dose-proportional, low variability (≤30%), 2x accumulation
- **Safety:** well tolerated
  - No SAEs, discontinuations, or clinical hyper/hypothyroidism
  - All TEAEs Grade ≤2
  - No concerning labs, ECGs, vital signs, physical examinations



Data presented as mean ± SD

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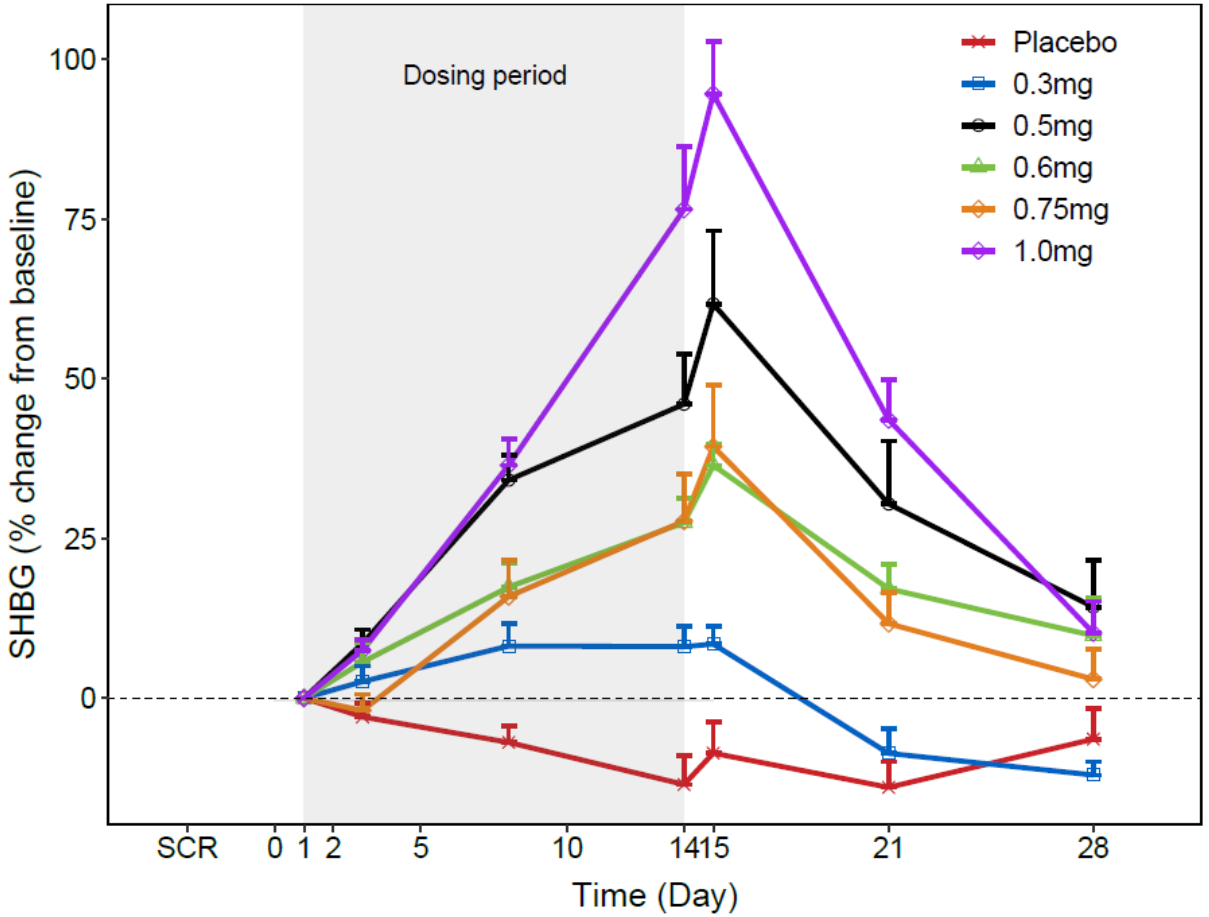
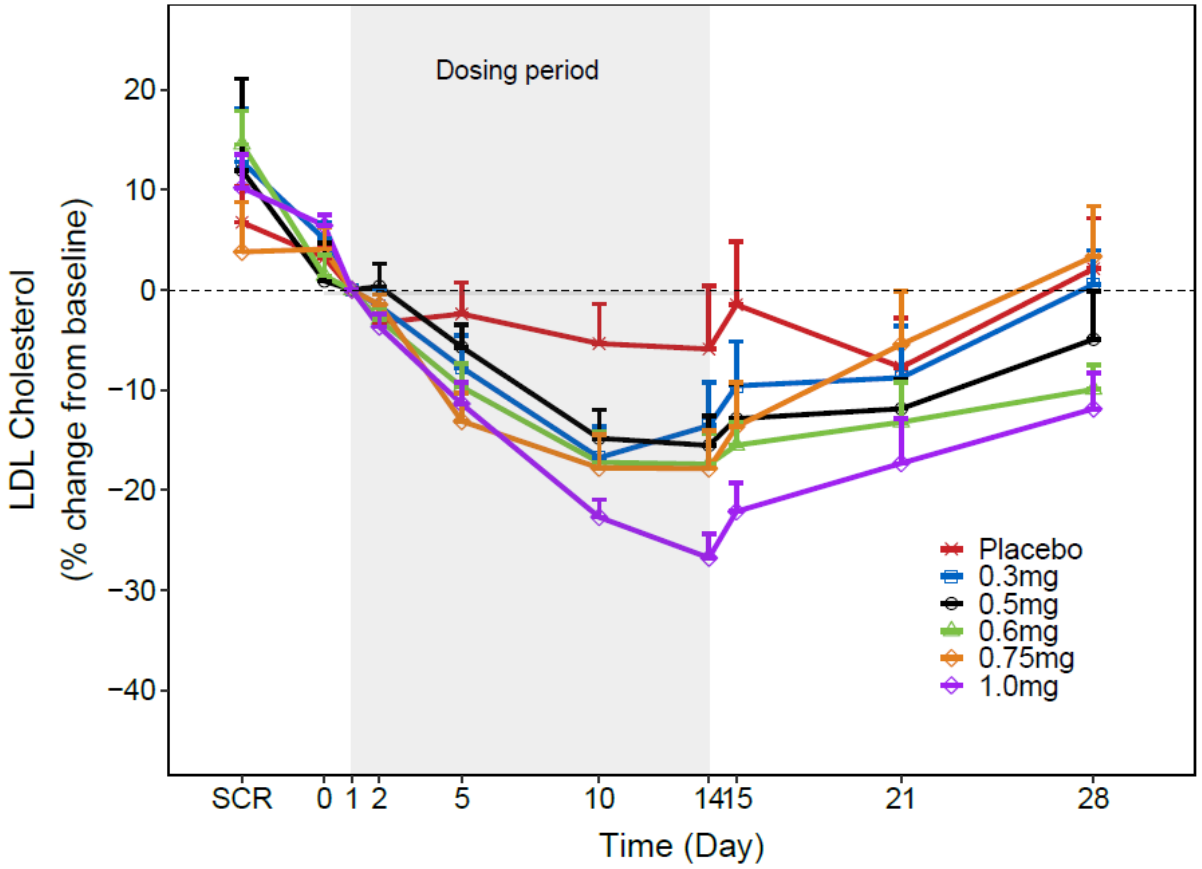
Charfi et al., EASL 2022.
clinicaltrials.gov; NCT05090111.

Multiple Ascending Dose - Biomarkers
Part 2: Expected Thyromimetic Effects Observed



Generally dose proportional increases in SHBG



Generally dose proportional decreases in lipids (e.g., LDL, Apo-B, Triglycerides)

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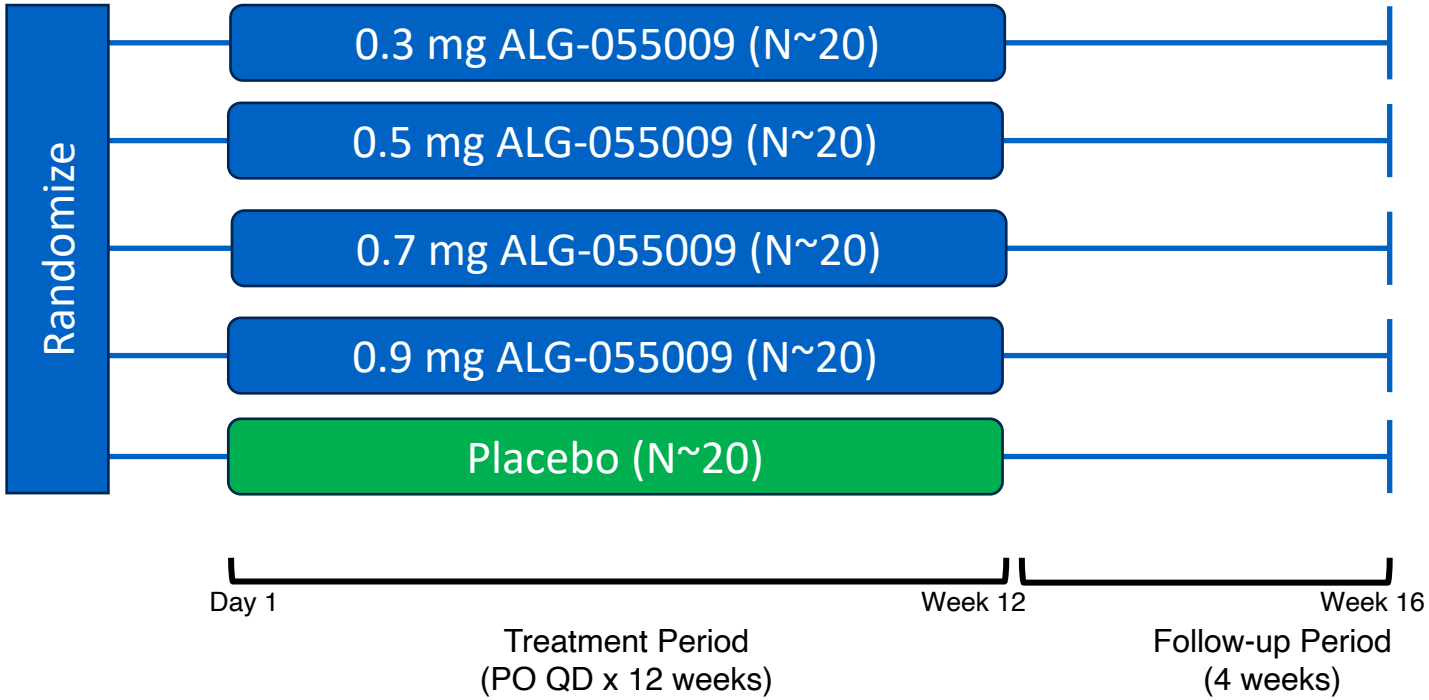
Charfi et al., EASL 2022.
clinicaltrials.gov; NCT05090111.

ALG-055009
Phase 2a HERALD Study Design



- Population: ~100 adult subjects with MASH and liver fibrosis (F1-F3)
- Primary endpoint: Relative change in liver fat content by MRI-PDFF at Week 12
- Principal Investigator: Dr. Rohit Loomba

Dosing ongoing; Topline data anticipated in early Q4 2024

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NCT06342947.

HERALD Study
Key Entry Criteria

- **Inclusion**
  - 18-75 years old, BMI ≥25 kg/m^2
  - F1 – F3 MASH diagnosis based on,
    - Liver biopsy (within 6 months) – NAS ≥4 with a score of ≥1 in each category
    - Having ≥2 metabolic syndrome criteria and Fibroscan between 7-20 kPa
  - CAP >300 dB/m
  - MRI-PDFF ≥10%

- **Exclusion**
  - Evidence of cirrhosis (clinical, laboratory)
  - Pituitary or thyroid disorder, use of thyroid replacement therapy (within 6 months) or TSH, free T4, or Total T3 >1.1 x ULN or <0.9 x LLN
  - Concerning cardiac history or abnormal ECG
  - Labs:
    - HbA1c ≥9.5%, Platelets ≤135,000/mm^3
    - ALT or AST >5 x ULN
    - INR > 1.3, Albumin <3.5 g/Dl, eGFR <45 mL/min/1.73 m^2

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ALG-055009
A Potential Best-in-Class THR-β Agonist for MASH

- **Discovered by Aligos; issued US patent expires 2040[1]**
- **Purpose-built with enhanced pharmacologic properties**
  - 5-50x fold more potent
  - More β selective
  - Optimized for PK

  } vs. competitor THR-β agonists
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[1]Not including any patent term extension.

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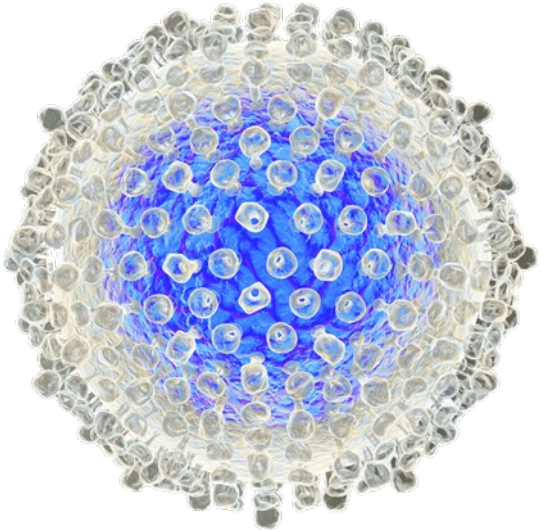
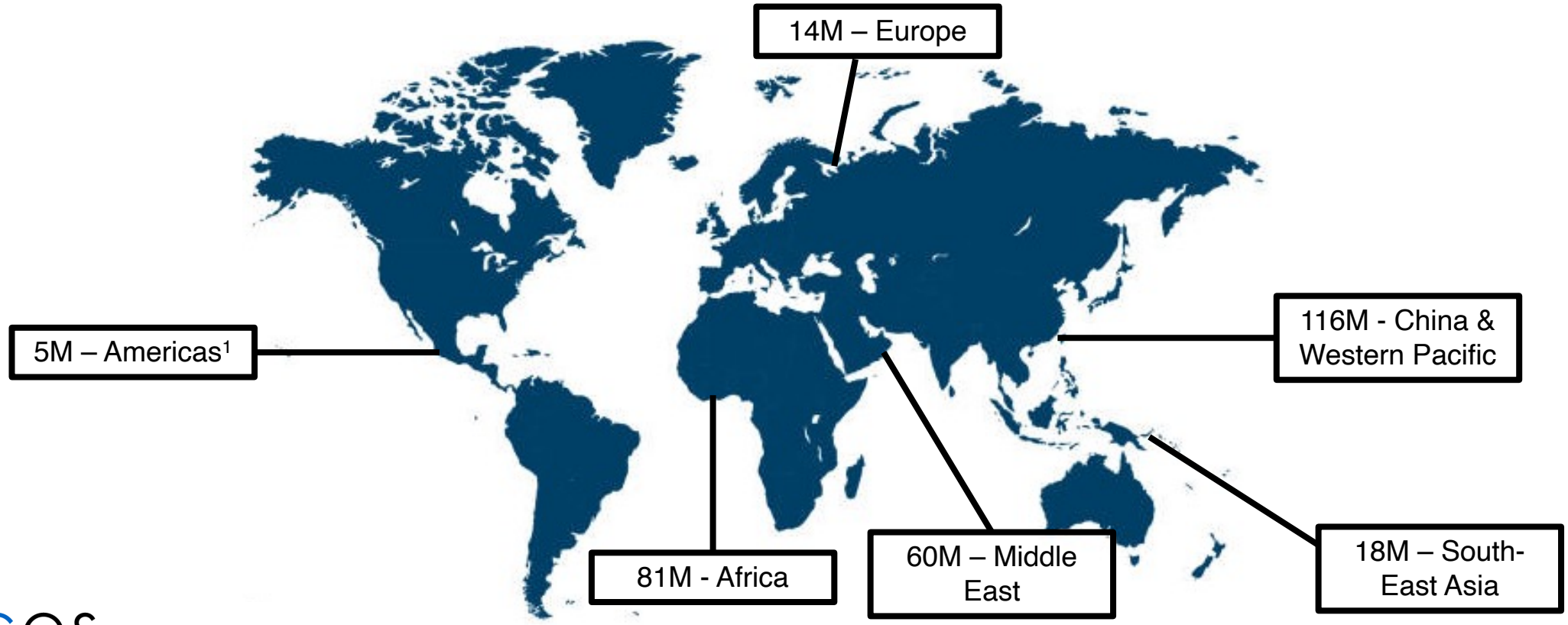
Chronic Hepatitis B

- ALG-000184: Small molecule CAM-E



CHB
High Unmet Medical Need

- 296M people worldwide living with CHB with 1.5M new infections each year
- 820k deaths per year, mostly from cirrhosis and hepatocellular carcinoma
- Primary cause of liver cancer worldwide
- Market opportunity estimated at $6.2B by 2031[2]; Gilead HBV sales of $1B in 2023[3]



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Data from the World Health Organization; 2019.
[1]Data largely unavailable for Latin America. 1-2M estimated for the US.
[2]Data from Allied Market Research.[3]Gilead 2023 10-K Filing, figure includes HDV sales.

Therapeutic Goals of HBV Antiviral Drugs

Current treatment options

Nucleoside/Nucleotide analogs (NAs):

- Oral
- For use in chronic DNA suppression
  - HBV DNA <LLOQ after 48 weeks on treatment
- Leads to improvement in inflammatory components of liver histology
- Suboptimal efficacy in some patient populations
- Rate of functional cure no greater than in untreated populations
- Widely used
- Well tolerated

Interferon alfa (IFNα)

- Injectable
- For use in functional cure
  - HBsAg < LLOQ ~6 months after a finite treatment regimen
- Frequent adverse effects and high number of contraindications
- Efficacy rates low, limited to subsets of HBV patients
- Not widely used

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Rethinking CHB Treatment: A New Era

The industry has learned from the issues of first-generation investigational mechanisms such as siRNA, ASO, NAPs, CAMs, immunomodulators, and therapeutic vaccines. Functional cure is a difficult pathway

The DNA suppression observed with ALG-000184 to date is greater vs. SOC, and is an approvable, de-risked regulatory pathway that can meaningfully help CHB patients (chronic suppression)

We have solved the potency issues previously seen with CAMs, leading to greater DNA suppression and clinical demonstration of the second mechanism

The importance of all relevant biomarkers has not been a key focus for the space. Treating CHB patients is more than reductions in HBV DNA and HBsAg; it is also HBV RNA, HBcrAg, and HBeAg

ALG-000184 is paving the way for the future of CHB treatments
First potential new mechanism advancing towards approval for chronic suppression in CHB in 25+ years

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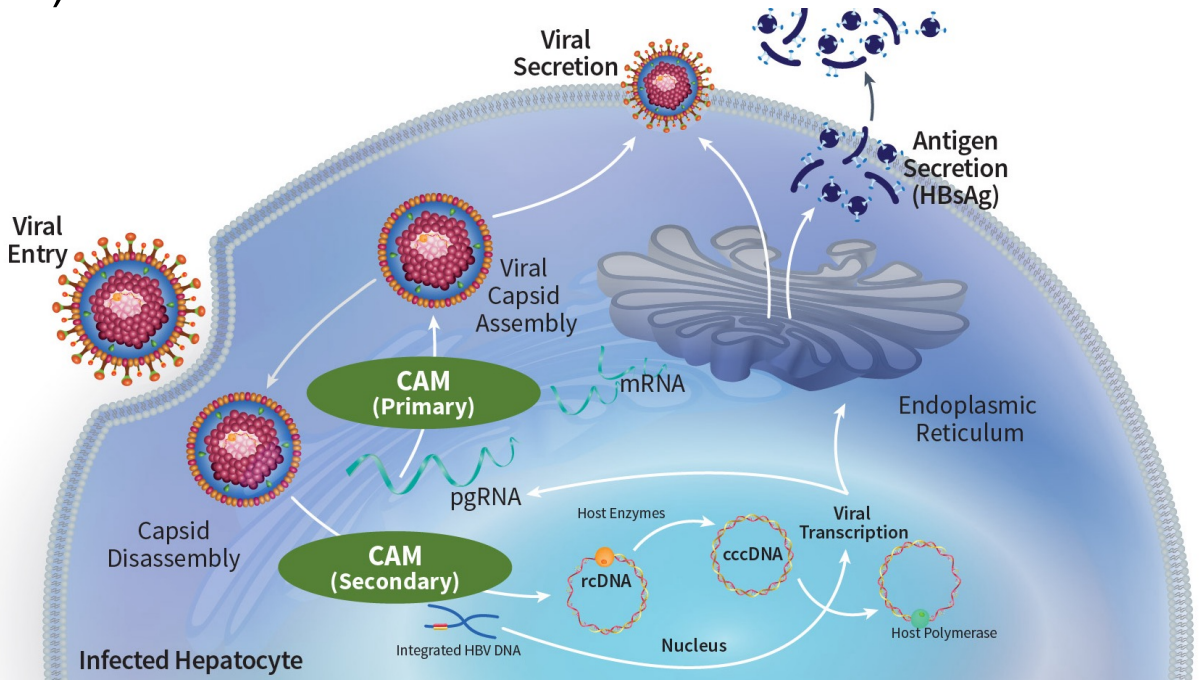
Hepatitis B Virus (HBV) Treatment
The Dual Role of Capsid Assembly Modulators (CAMs)

- In preclinical studies, 2 mechanisms of action (MoA):
  - Primary mechanism
    - › Promotes the premature assembly of core protein, leading to the formation of empty capsids
    - › Responsible for the deep reductions of HBV DNA and RNA observed clinically
  - Secondary mechanism
    - › Requires >10-fold higher drug concentrations
    - › Prevents the establishment / replenishment of cccDNA, which produces HBcrAg, HBeAg, and (some of) HBsAg



- 1st generation CAMs in development since 2014
  - Consistently demonstrated DNA, RNA reductions (1st MoA)
  - To date, no clear evidence of effects on 2nd MoA

Observing both mechanisms clinically likely requires potent compounds with excellent PK properties

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ALG-000184
A Potential Best-in-Class CAM-E for CHB

- **Initial IP licensed from the lab of Dr. Raymond Schinazi at Emory University; issued US patent expires in 2040[1]**
- **Enhanced pharmacology**
  - Picomolar potent
  - Enhanced absorption with high liver uptake
- **Phase 1 highlights** (≤300 mg ALG-000184 ± ETV x ≤64 weeks in untreated CHB)
  - PK: dose proportional, low-moderate variability
  - Safety: no safety signals observed
  - Antiviral activity: best-in-class reductions seen in HBsAg, HBeAg, HBcrAg, HBV DNA & RNA
  - Dosing x ≤96 weeks ongoing (through 2025)
- **Phase 2**
  - Enabling activities underway
  - Regulatory discussions in progress (superior chronic DNA suppression)

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[1]Not including any patent term extension.

ALG-000184
Superior Potency and Activity vs. Competitor CAM-Es

Parameter	JNJ-6379[3,4]	EDP-514[5]	AB-836[6]	ABI-4334[1,2]	ALG-000184
Potency (cell culture HBV DNA reduction, EC_{50} nM)	54	17	10	1.2	0.63
Dose	250mg	800mg	100mg	200mg	10mg
Mean HBV DNA decline in clinic (log_{10} IU/mL)	2.7	3.4	3.1	NA	3.7
% of subjects < LLOQ at Day 28	56	NA	NA	NA	100
Most advanced/current development status	Phase 2; Discontinued	Phase 1b; Seeking partner	Phase 1; Discontinued	Phase 1a	Phase 1b (Ph2 enabling)

ALG-000184 more potent vs. other CAM-Es, resulting in enhanced antiviral activity
Exposures also enhanced via PK optimization strategies

*89% of subjects were HBeAg negative in 250 mg JNJ-6379 arm. An unknown percentage of subjects dosed with 800 mg EDP-514 were HBeAg negative.

**The comparisons shown in the table above are not based on data resulting from head-to-head trials and are not direct comparisons. Different protocol designs, trial designs, patient selection and populations, number of patients, trial endpoints, trial objectives and other parameters that are not the same between the relevant trials may lead to bias in the results causing comparisons of results from different trials to be unreliable.

N/A – not available.
LLOQ (DNA) was ≤20 IU/mL for ABI and JNJ and 10 IU/mL for Aligos.
1. Xu, X. et al. AASLD 2021 LP4. 2. Gane, E. et al. EASL 2023 SAT-186.
3. Zoulim F., et al AASLD 2018. 4. Zoulim F., et al. Gastroenterology 2020.
5. MF Yuen, et al AASLD 2021 (Poster 823). 6. HepDart 2021 (preliminary data).
Data was sourced from publicly available literature, posters and presentations; ALG-000184 data was generated by Aligos on the parent compound ALG-001075.

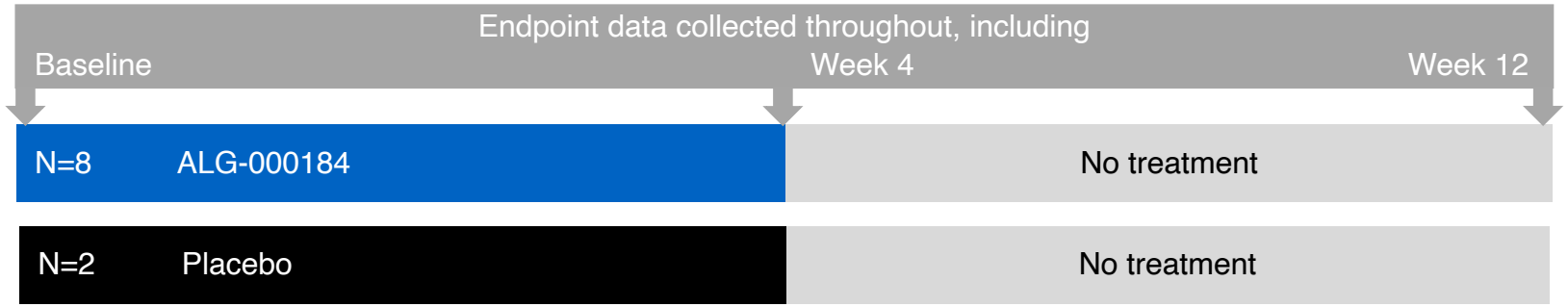
ALIGOS THERAPEUTICS

ALG-000184-201
Phase 1 Study in HV and CHB Subjects

Parts 1-3: Complete

- Part 1 (SAD): single oral doses (40, 100, 250 and 500 mg) in HV
- Part 2 (MAD): 7 QD oral doses (150 and 250 mg) in HV
- Part 3: QD oral doses (10-300 mg) x 28 days in treatment naïve (TN)/currently not treated (CNT) CHB

Multiple Doses in Currently Not Treated/Treatment Naïve CHB Subjects
HBV DNA > 2000 IU/mL, HBeAg Negative or Positive
Up to 6 cohorts (n=10 per Cohort, n=8 ALG-000184, n=2 Placebo)
28 daily oral doses
Endpoints: PK, safety, antiviral activity (e.g., DNA, RNA)



Part 1 & 2: Single oral dose ≤500 mg and multiple oral daily doses ≤250 mg x 7 days well tolerated with linear PK in HV
Part 3: multiple daily doses ≤300 mg well tolerated with linear PK and excellent antiviral activity (DNA, RNA, HBsAg)

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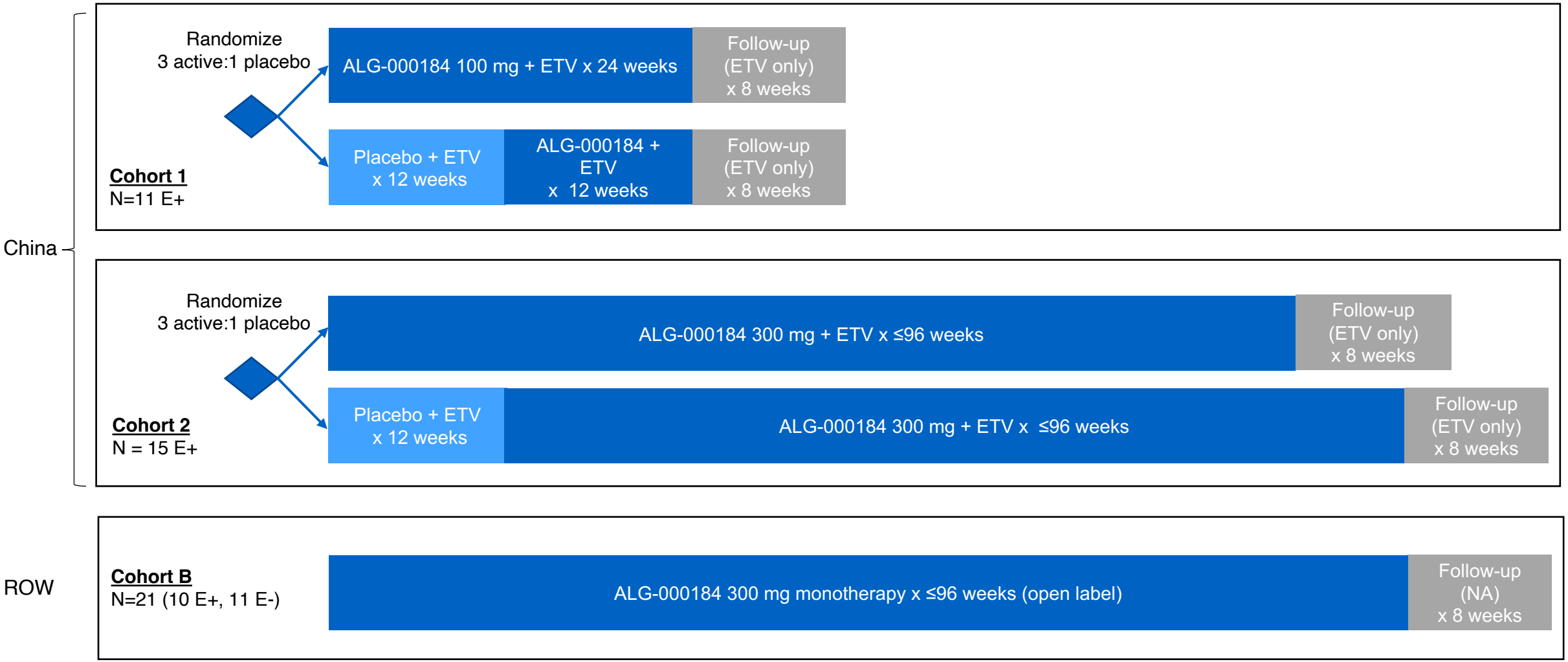
Gane Ed, APASL 2021; Gane Ed, HBV TAG 2021; Cohorts 1 & 2: Gane et al., AASLD 2021;
Cohort 4: Yuen MF. et al., APASL 2022; Cohort 3: Yuen MF. et al., EASL 2022; Clinicaltrials.gov: NCT04485663.

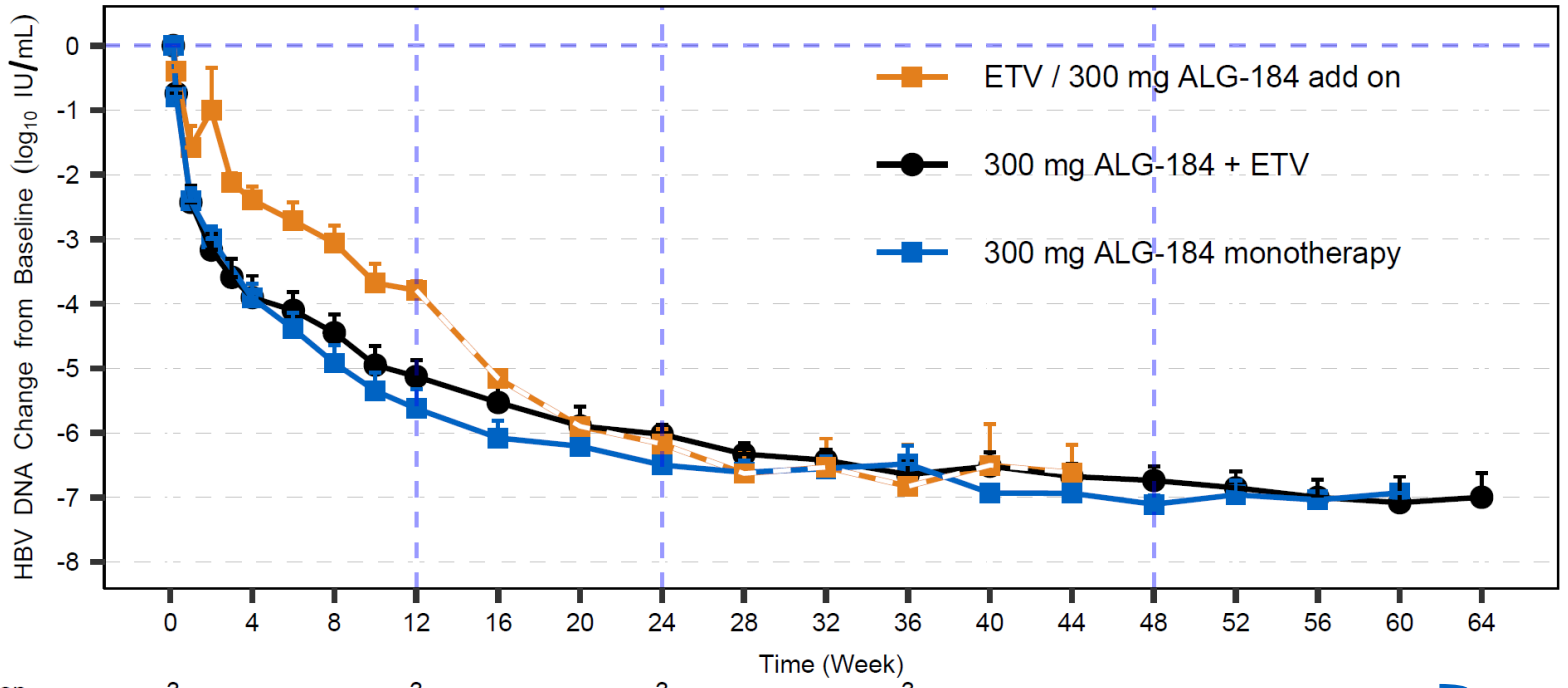
ALG-000184-201 – Long Term Dosing in CHB Subjects
Cohort Designs



ALIGOS THERAPEUTICS

Hou, JL. et al., Poster 1483-C, AASLD (2023).
Yuen, M-F. et al., Late Breaker Poster #5028-C, AASLD (2023).
All cohorts fully enrolled. NCT04536337.

Antiviral Effect in CHB Subjects
HBV DNA Change from Baseline



ETV / 300 mg ALG-184 add on	3	3	3	3		
300 mg ALG-184 + ETV	11	9	9	9	8	6
300 mg ALG-184 monotherapy	10	10	10	10	8	1

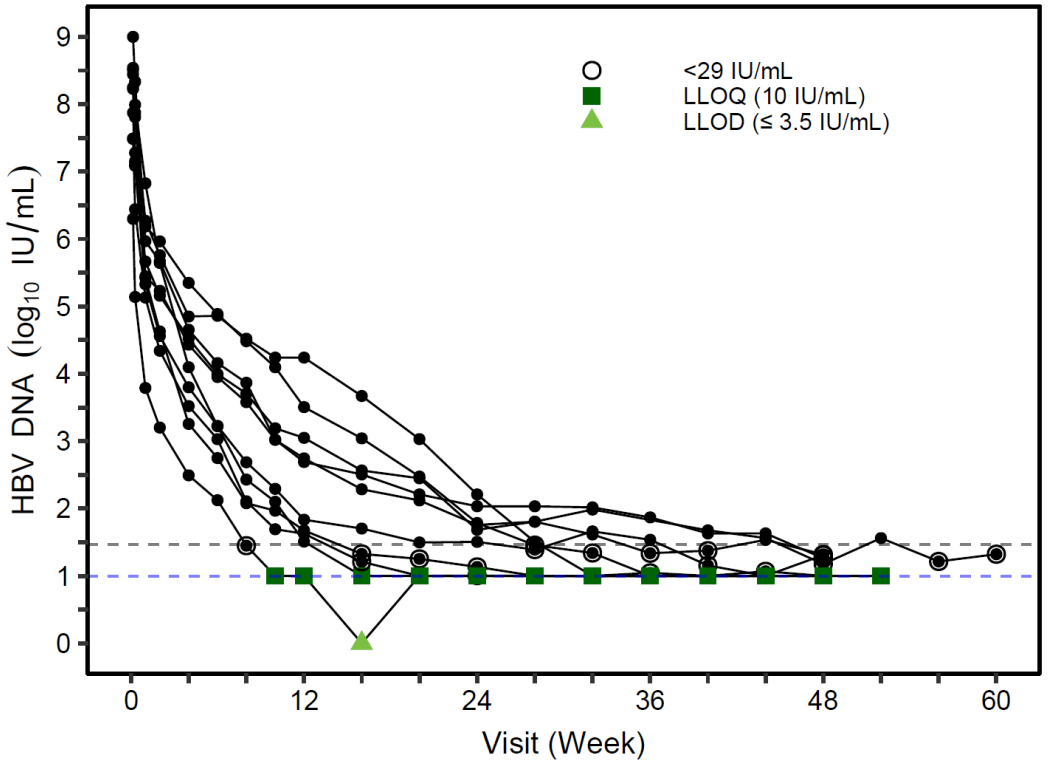
Number of subjects at timepoint

300 mg ALG-000184±ETV
Showed greater HBV DNA reduction than ETV monotherapy
Achieved similar DNA reductions

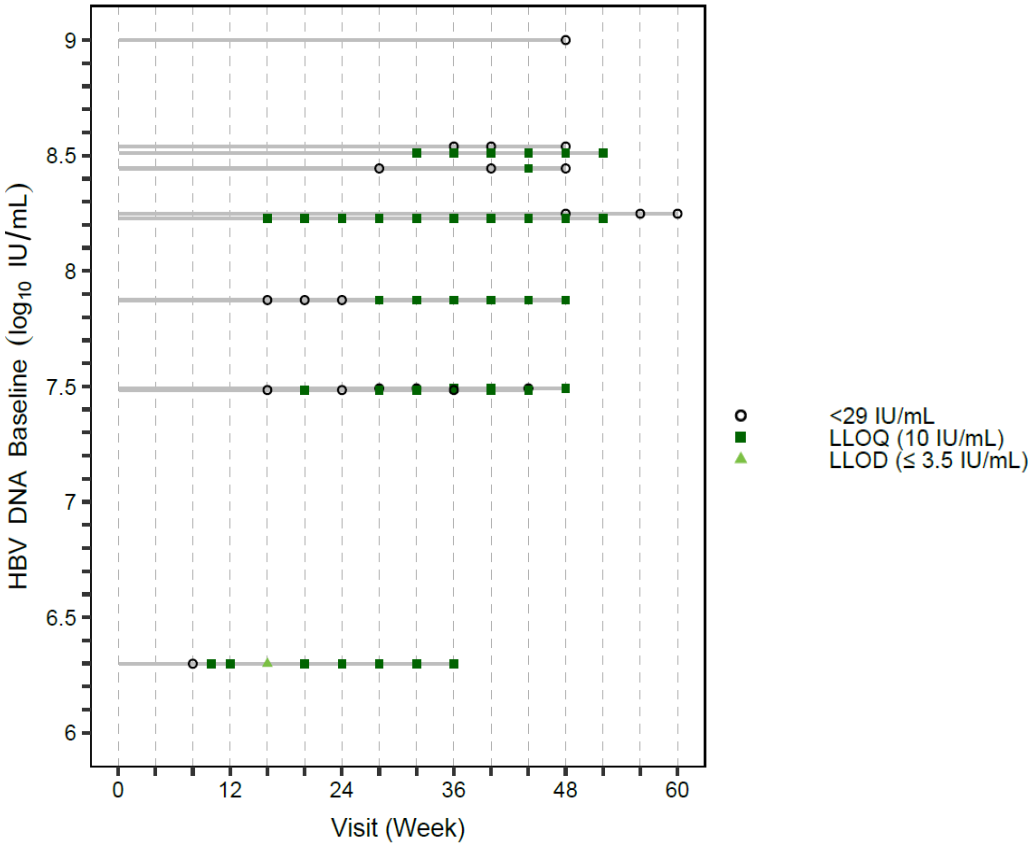
ALIGOS THERAPEUTICS

Yuen, M-F. et al., Oral Presentation 101807; APASL 2024.

300 mg ALG-000184 Monotherapy (HBeAg+)
Individual HBV DNA Over Time



Total n	10	10	10	10	10	10	10	10	10	9	9	9	8	3	1	1
Above 29 IU/mL	10	10	9	9	6	6	6	4	4	2	2	3	0	1	0	0
< 29 IU/mL	0	0	1	1	4	4	4	6	6	7	7	6	8	2	1	1
< LLOQ [10 IU/mL]	0	0	0	1	2	3	2	4	5	5	5	5	4	2	0	0
< LLOD [≤ 3.5 IU/mL]	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0



No viral breakthrough during ALG-000184 monotherapy x ≤60 weeks
100% (10/10) of subjects achieved HBV DNA < 29 IU/mL
60% (6/10) of subjects achieved sustained HBV <10 IU/mL by week 48

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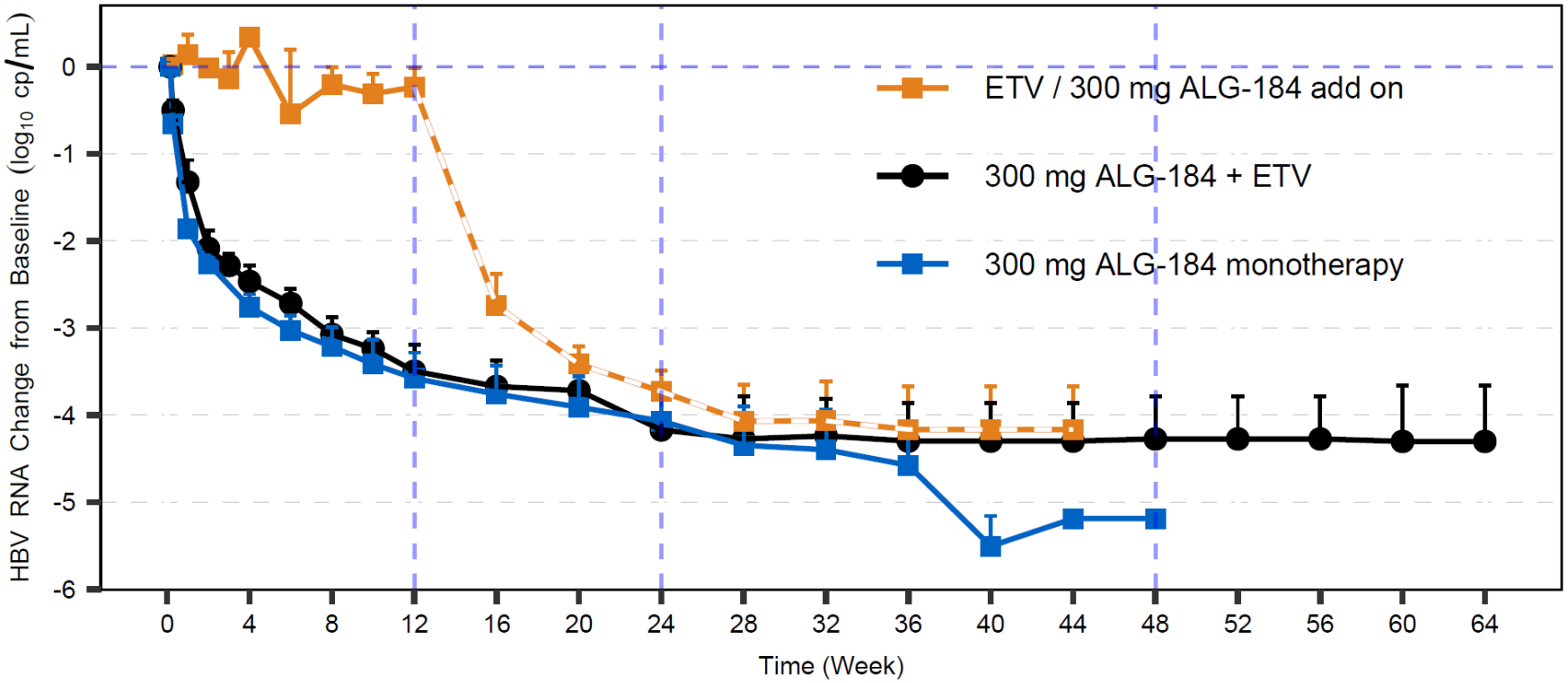
300 mg ALG-000184 ± ETV vs. ETV
Mean HBV RNA Over Time



ETV / 300 mg ALG-184 add on	3	3	3	3		
300 mg ALG-184 + ETV	11	9	9	9	8	6
300 mg ALG-184 monotherapy	10	10	9	8	1	

At Week 12, there was a >3 log_{10} copies/mL RNA decline with ALG-000184 ± ETV vs. no change with ETV
After adding ALG-000184 on top of ETV at Week 12, the RNA decline was similar to the combo regimen
Up to Week 64, 100% of subjects experienced HBV RNA < LLOQ
RNA levels correlated with HCC risk^

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Yuen, M-F. et al., Oral Presentation 101807; APASL 2024.
^Liu et al., J Infect Dis, 2022. Mak et al., J Gastroenterology, 2021. Ding et al. Hepatology, 2021.

ALG-000184
Chronic DNA Suppression versus Standard of Care

CHB HBeAg Status	Drug	% Patients < LLOQ at Week 48 (by HBV DNA Assay Sensitivity)		Regulatory Pathway
		% < LLOQ 29 IU/mL	% < LLOQ 10 IU/mL	
E+	TDF (n=292)[b]	67%	Presumably <21%	Superior Chronic Suppression vs. NAs
	TAF (n=581)[b]	64%		
	300 mg ALG-000184 (n=10)[c]	**10/10 (100%)**	**6/10 (60%)**	
E-	TDF (n=140)[a]	93%	17%	
	TAF (n=285)[a]	94%	21%	
	300 mg ALG-000184 (n=11)	**Data to be presented at EASL**		

Comparative HBV DNA data indicate 300 mg ALG-000184 may achieve superior chronic suppression vs. NAs

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TAF-tenofovir alafenamide, TDF-tenofovir; LLOQ-lower limit of quantification.
[a] Buti et. al., Lancet Gastro 2016; [b] Chan et. al., Lancet Gastro 2016. [c] Yuen, M-F. et al., Oral Presentation 101807; APASL 2024

Chronic Suppression
Well Defined, Validated Approval Pathway

Pathway endorsed by FDA, EMEA, and China FDA (CDE). Definition: HBV DNA <LLOQ at Week 48

a. Chronic suppressive therapy

Sponsors developing drugs for chronic suppressive therapy of CHB should consider the following trial design options:

- A randomized controlled noninferiority (NI) (or superiority) trial comparing the investigational drug with an approved active control arm—The primary efficacy endpoint should be undetectable HBV DNA[13] after 48 weeks on-treatment in HBeAg-positive subjects, HBeAg-negative subjects, or both. The active comparator should be an antiviral drug that is recommended for treatment of CHB and reflects current practice at the time of trial initiation. The patient population may be treatment-naïve or previously treated subjects with detectable HBV DNA. Potential concerns related to the development of resistance when evaluating an investigational drug with a low barrier to resistance as a monotherapy should be addressed. For an NI design, determination of the NI margin should be discussed with the FDA.

Pathway was used to get tenofovir alafenamide (TAF; Gilead) approved in all 3 regions

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HBV Guidance from: FDA 2022; EMEA 2006; China 2023.

ALG-000184 Phase 2 Chronic Suppression Study
Planned Efficacy Endpoints

- Primary endpoint (approvable endpoint*)
  - Proportion of subjects with HBV DNA < 10 IU/mL at W48 for both HBeAg+/- CHB
- Secondary endpoints (clinically meaningful and/or corroborative)
  - HBeAg seroconversion in HBeAg+ CHB subjects
  - Histologic improvement (liver biopsy)
    - Inflammation
    - Fibrosis
  - Reduction of cccDNA levels
  - Reduction of HBV integrants^
  - HBV RNA < LLOQ^

In addition to superior DNA reductions, multiple clinically meaningful secondary efficacy endpoints may be achieved

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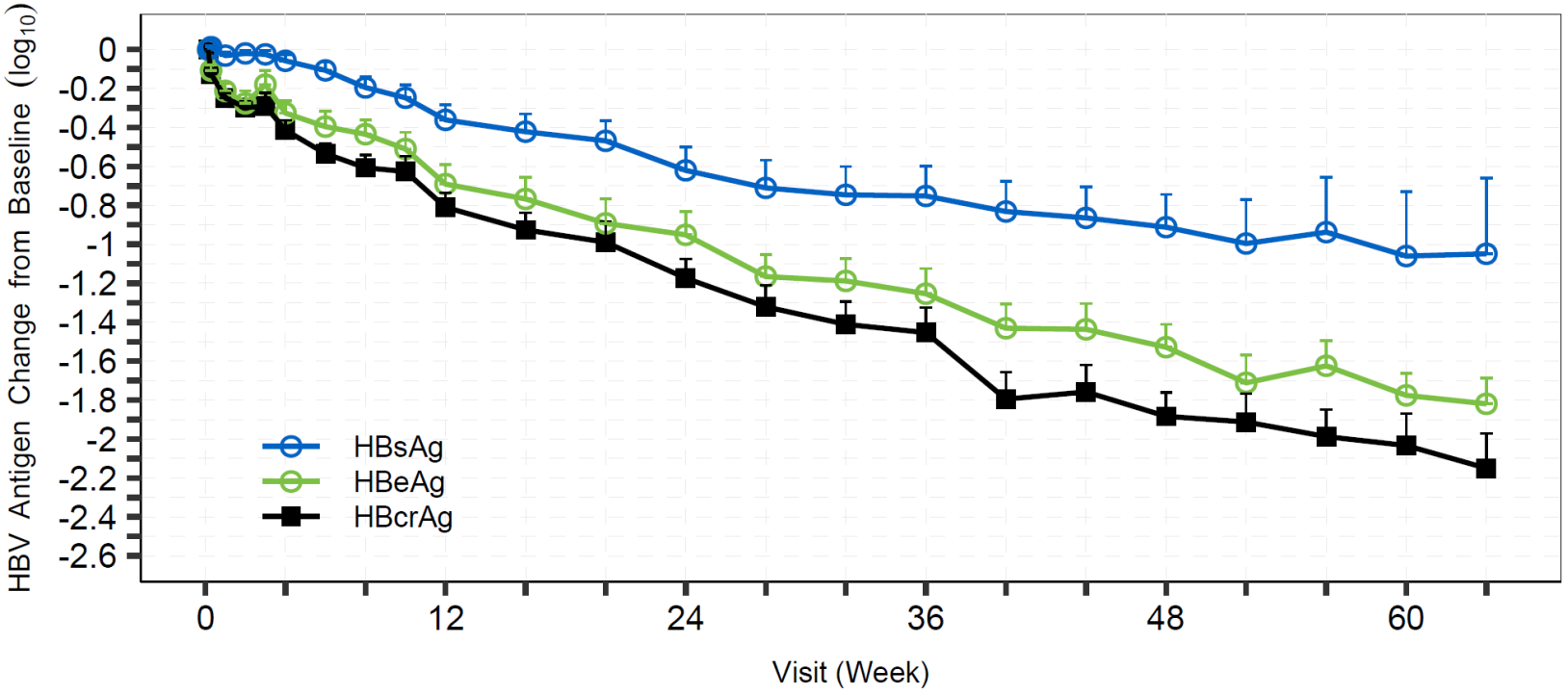
*FDA Guidance April 2022.
^Possible link to cancer risk (HCC)

ALG-000184-201 - Antiviral Effect in CHB Subjects
HBV Antigen Change from Baseline



HBsAg	21	19	19	19	16	7
HBeAg	21	19	19	18	16	7
HBcrAg	21	19	18	17	9	6

Substantial HBsAg, HBeAg, and HBcrAg reductions seen with combo
Max declines: 1.9, 2.2 and 2.6 log_{10}, respectively

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Yuen, M-F. et al.,Oral Presentation 101807; APASL 2024.

Safety Overview
Treatment Emergent Adverse Events

	300 mg ALG-000184 + ETV	300 mg ALG-000184
	N=15	N=10
Serious Adverse Event (SAE)	0	0
Treatment Emergent Adverse Event (TEAE) leading to study drug discontinuation	0	0
Grade ≥3 TEAE	4*,#	3#
Concerning laboratory, ECG, vital sign, or physical examination findings	0	0

* 3 subjects experienced Grade ≥ 3 TEAEs of asymptomatic laboratory abnormalities including eGRF decrease, uric acid increase and neutrophil count decrease. All had normalized or returned to baseline in setting of continued study dosing.

2 subjects in Part 4 Cohort 2 and 3 subjects in Part 4 Cohort B experienced Grade ≥ 3 ALT ↑ with or without associated AST ↑. All were asymptomatic and none were associated with hepatic synthetic dysfunction or considered concerning to the study's ALT Flare Monitoring Committee.

300 mg ALG-000184 ± ETV well tolerated x ≤64 weeks

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Antiviral Activity of HBV Drug Classes

Viral Marker	NAs (ETV, TDF, TAF)	siRNA*	ASO**	ALG-000184 (CAM-E)	Clinical Significance
DNA	++	+	+	+++	DNA <LLOQ on treatment (chronic suppression) associated with improved outcomes and is an approvable endpoint
RNA		+	+	+++	RNA levels correlate with HCC risk^
HBsAg		++	++	++	HBsAg loss (whether spontaneous or via medicines (functional cure)) associated with improved outcomes
HBeAg		+		++	HBeAg loss with seroconversion associated with improved outcomes and guidance for stopping HBV treatment
HBcrAg		+	+	++	Surrogate biomarker for HBV replication activity

ALG-000184 is the only oral HBV antiviral drug that broadly inhibits viral markers which are associated with improved outcomes in CHB

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*Yuen et al., AASLD 2020.
** Yuen et al., Nature Medicine 2021 *https://doi.org/10.1038/s41591-021-01513-4.*
^Liu et al., J Infect Dis, 2022. Mak et al., J Gastroenterology, 2021. Ding et al. Hepatology, 2021.

Our Portfolio of Best-in-Class Drug Candidates Will Drive Value

ALG-055009 for MASH

- ✓Phase 2a HERALD enrollment completed in May 2024
- Phase 2a HERALD topline safety and MRI-PDFF data expected in early Q4 2024

ALG-000184 for CHB

- ✓Greater DNA suppression observed vs. NAs
- Phase 1b study is ongoing with interim data readouts expected at APASL, EASL, AASLD
- Phase 2 enabling activities ongoing
- Regulatory discussions underway (superior chronic DNA suppression)

ALG-097558 for Pan-Coronavirus

- ✓Phase 1 FIH topline data presented April 2024
- Phase 2 enabling activities (externally funded) ongoing

Strong Cash Position

- As of 3/31/24: Cash, cash equivalents and investments were $112.7M
- The Company believes our cash, cash equivalents and investments will provide sufficient funding of planned operations through the end of 2025


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