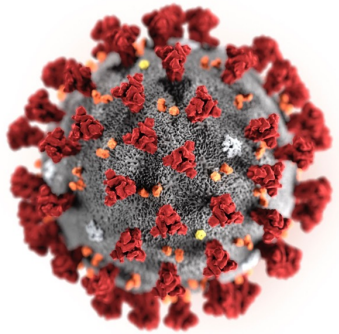
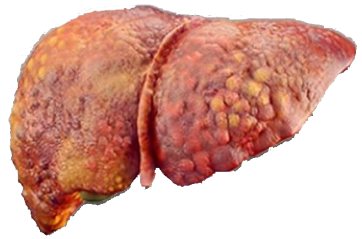
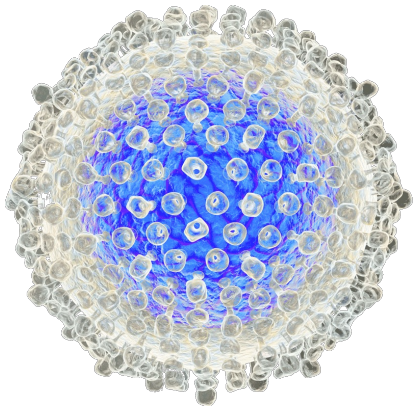
ALIGOS
THERAPEUTICS







Corporate Presentation

August 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 August 6, 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
  - Clear regulatory path forward for chronic suppressive therapy with superiority label
  - Phase 2 enabling activities ongoing; planned Phase 2 IND filing in Q1 2025

As of 6/30/24 - Cash, cash equivalents and investments were $94.5M. 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 Monotherapy	CAM-E	Phase 1b (Dosing x ≤ 96 Weeks), Phase 2 Enabling Activities — APASL ★ EASL ★ AASLD ★
ALG-000184 (including AMOYTOP BIOTECH)	CHB Combination[1]	CAM-E + PEGBING® (Mipeginterferon alfa-2b)	Phase 1b Enabling Activities
ALG-097558	Covid-19*	Protease Inhibitor	Phase 2 Enabling Activities (Clinical, Nonclinical) — FIH Topline Data ★

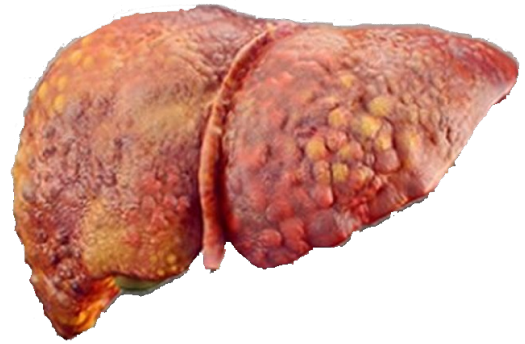
ALIGOS THERAPEUTICS

*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. 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. [1]Amoytop sponsoring combo study.

ALIGOS
THERAPEUTICS

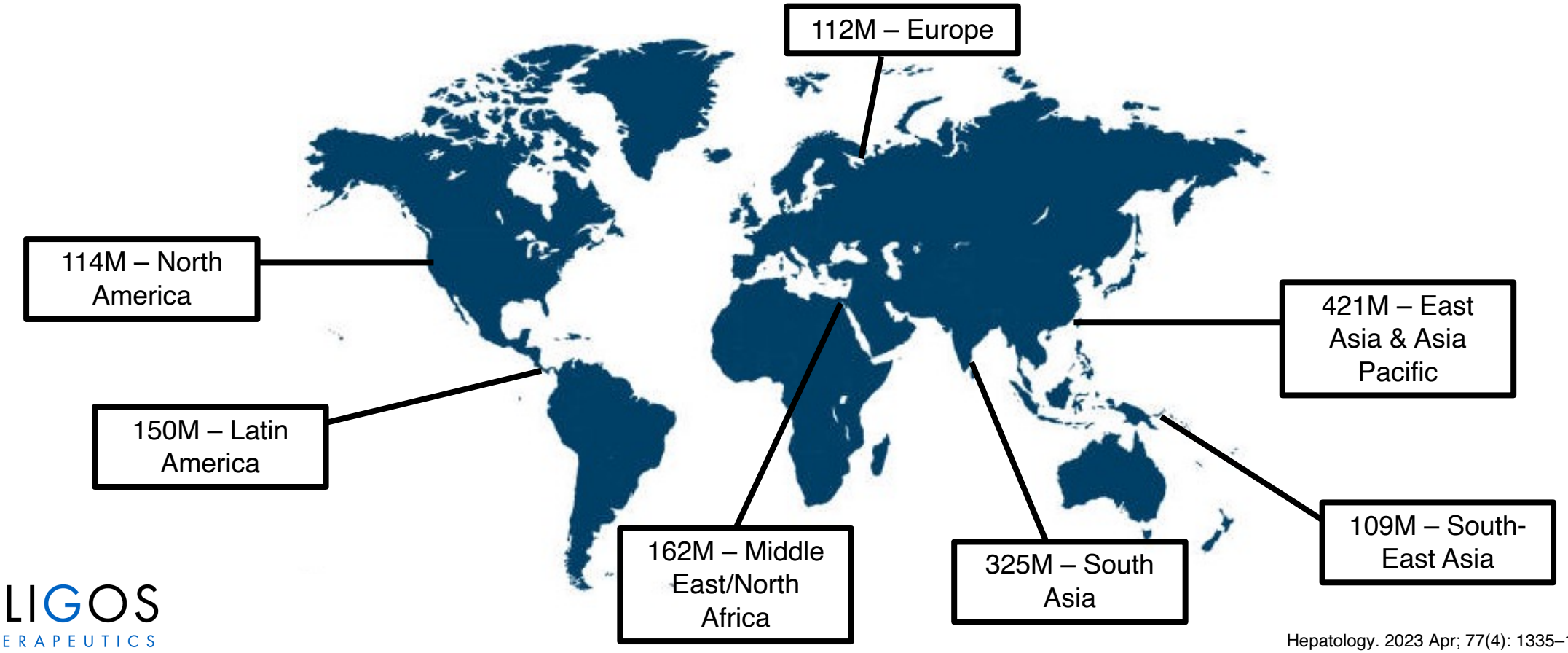
MASH

- 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



ALIGOS THERAPEUTICS

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

MASH Pathogenesis

Fatty acids = primary source of excess energy supply

(from de novo lipogenesis & lipolysis)

↓

Lipotoxic Species

↓

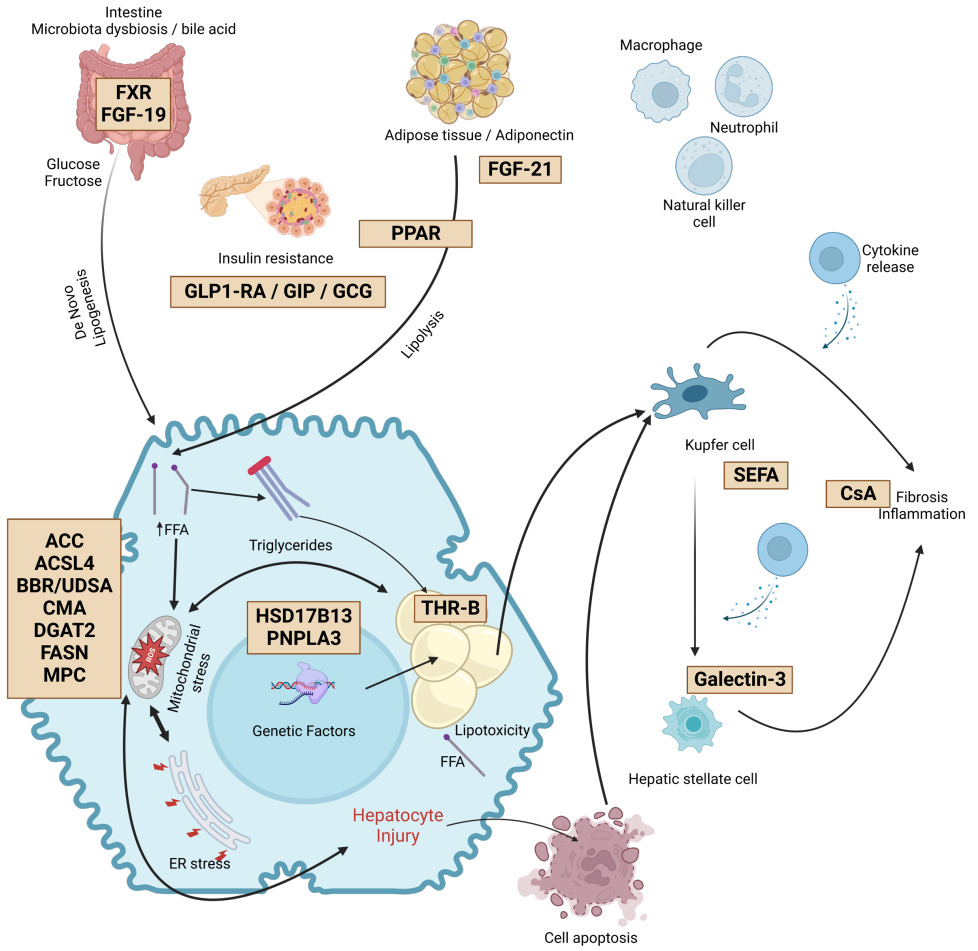
Liver Injury

Endoplasmic reticulum stress

Mitochondrial dysfunction

Pro-inflammatory cytokines release

Apoptosis

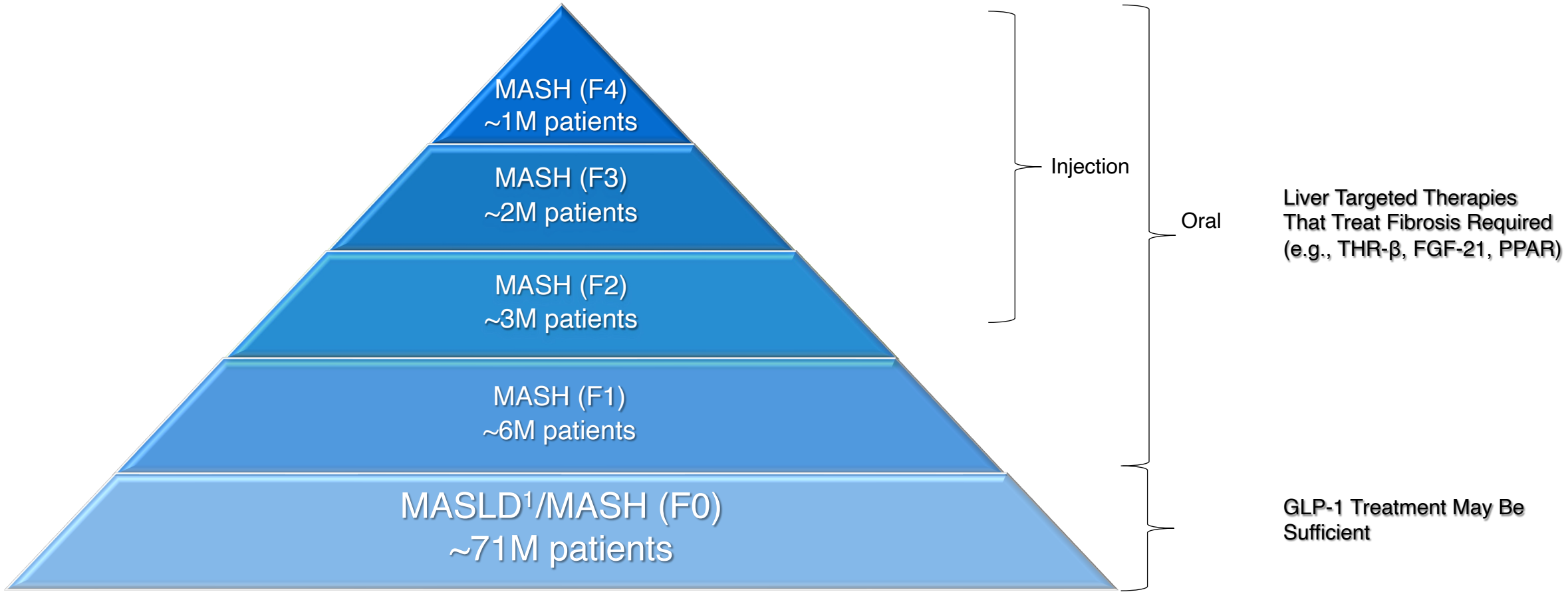


MASH biology is complex with multiple therapeutic approaches being evaluated; combination regimens may be required

ALIGOS THERAPEUTICS

Adapted from Stephen Harrison presentation, AASLD 2023.

MASLD/MASH Potential Future Treatment Paradigm

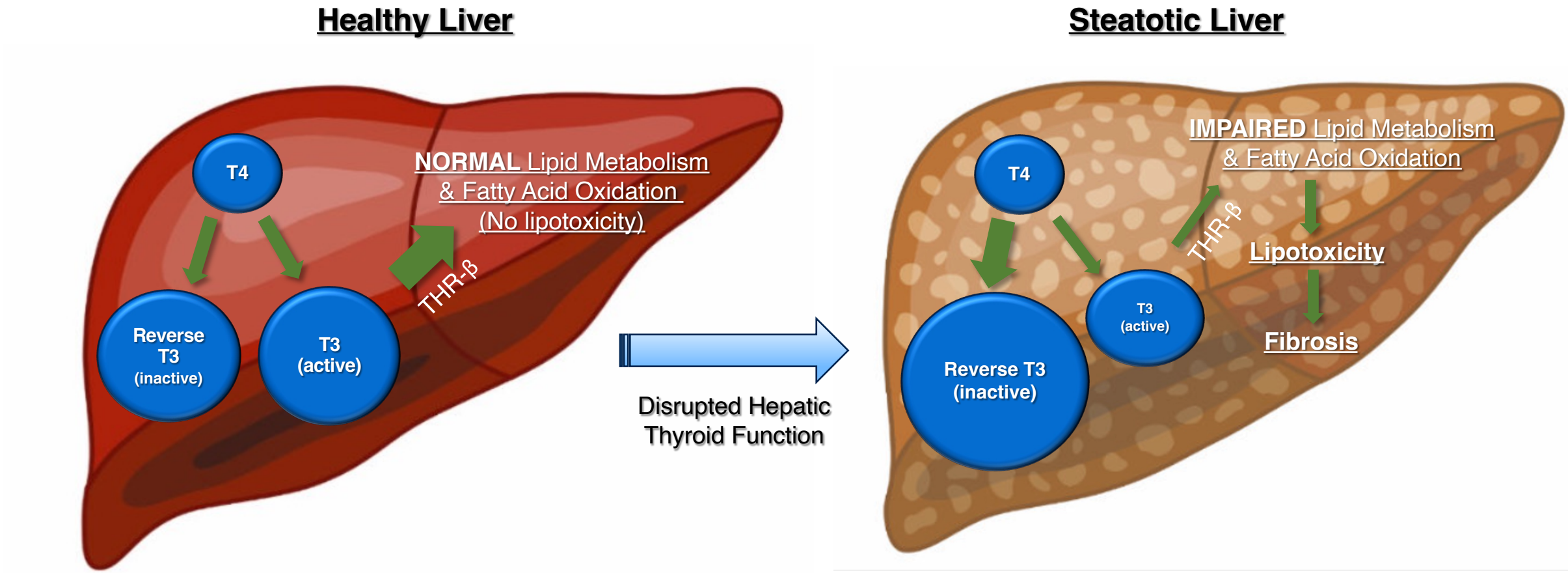


Liver-targeted therapies, likely in combination, required for F1-F4 MASH patients for whom typical lifestyle modifications and/or comorbidities treatments are insufficient

ALIGOS THERAPEUTICS

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



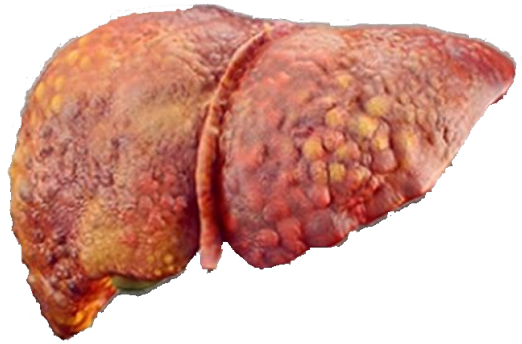
Hepatic hypothyroidism results in reduced active T3 production, allowing increased production of pro-inflammatory lipotoxic fat that causes hepatocellular injury/death, fibrosis, and cancer

ALIGOS THERAPEUTICS

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

ALIGOS
THERAPEUTICS

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

ALIGOS THERAPEUTICS

[1]Not including any patent term extension.

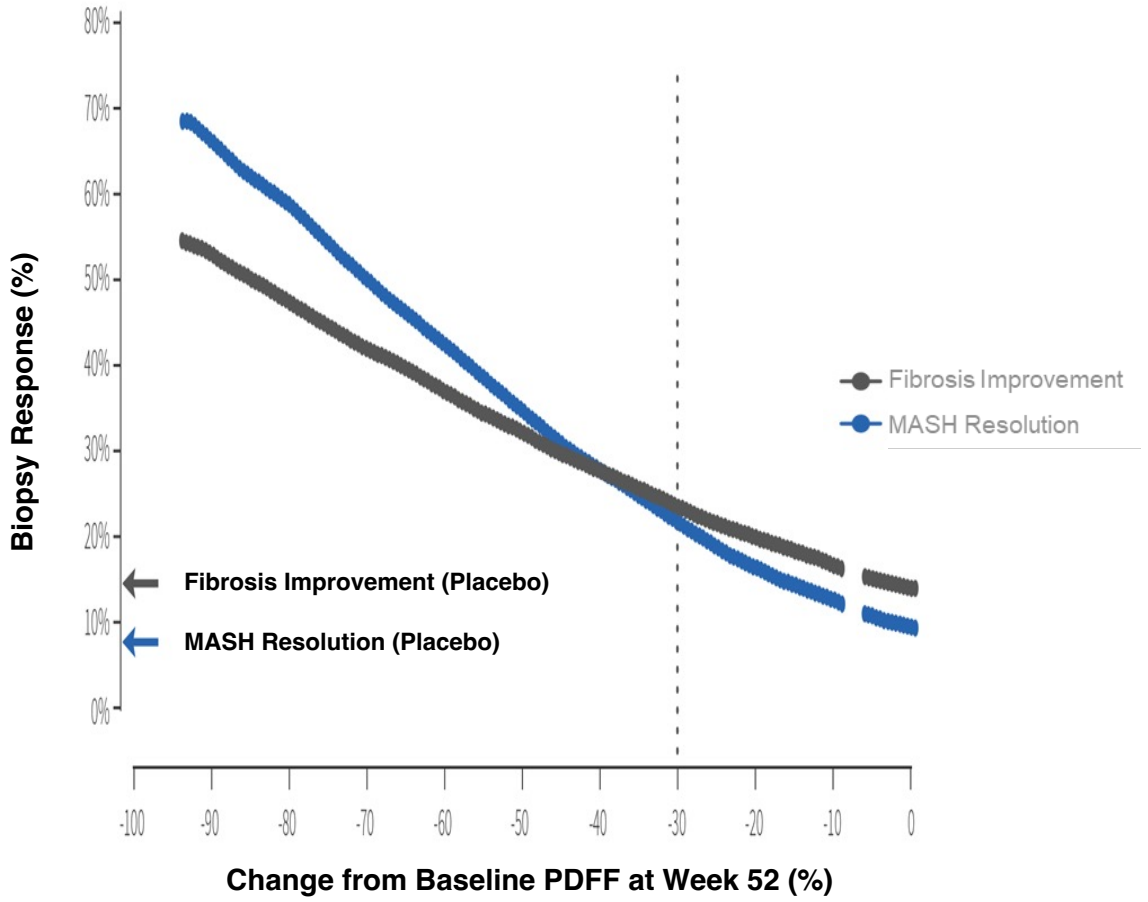
Enhanced Potency, PK Correlated with Efficacy

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

ALIGOS THERAPEUTICS

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

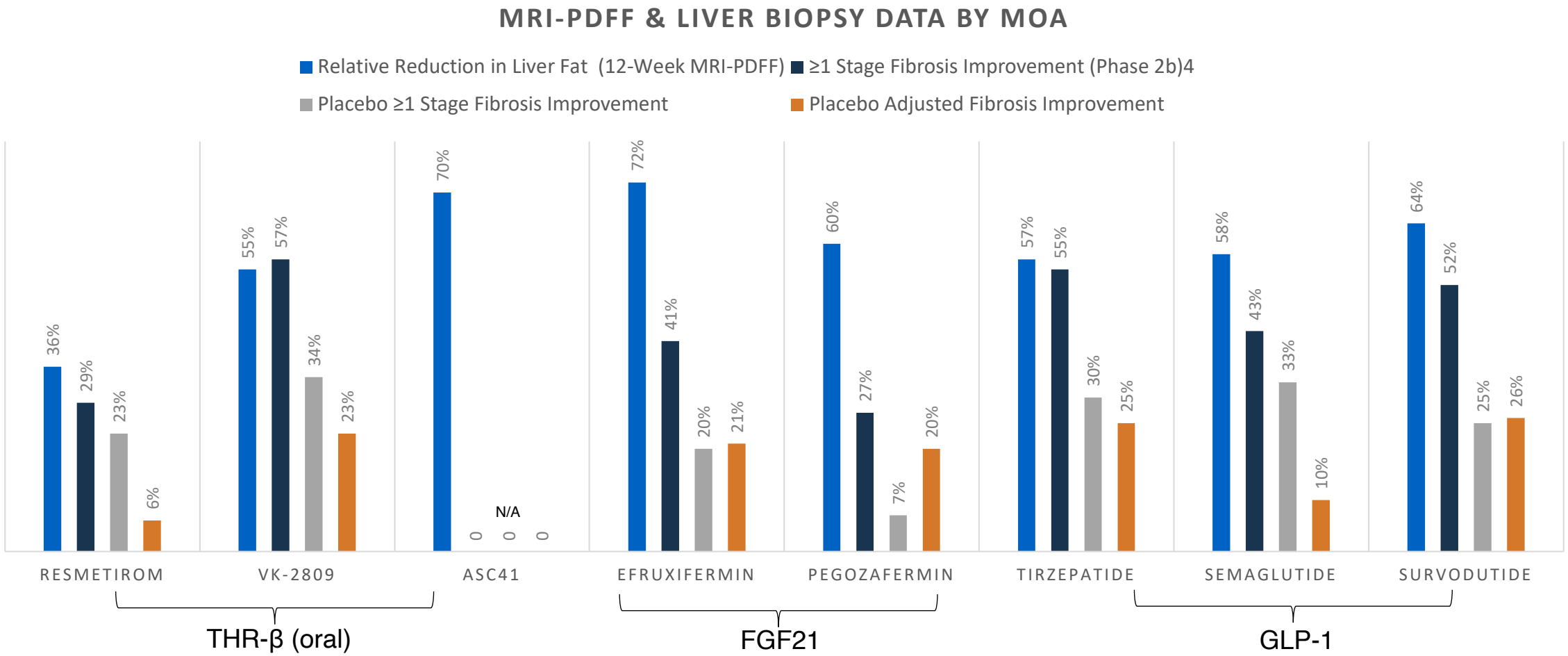


THR-β induced MRI-PDFF de-fatting strongly correlated with histologic improvement

ALIGOS THERAPEUTICS

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

The Continued Need in MASH
Translation of MRI-PDFF to Liver Biopsy



Data to date suggests placebo adjusted fibrosis improvement is similar across mechanisms

ALIGOS THERAPEUTICS

Data from Dr. Stephen Harrison DLC Presentation. Resmetirom: Taub, Atherosclerosis, 2013. Company press release 5/2018. Biopsy time 36 wks. VK-2809: Lian et al., ACC 2016 and May 16, 2023, press release, and company website. Biopsy time 52 wks. Efruxifermin: Company presentation 3/2024, press releases 9/2022, 3/2020. Biopsy time 24 wks. Pegozafermin: Company press releases 9/2020 and 3/2023. Biopsy time 24 wks. Tirzepatide: Gastaldelli et. Al., Lancet Diabetes Endocrinol, 2022. EASL 2024 abstract. MRI-PDFF data at 52 wks. Biopsy time 52 wks. Semaglutide: Flint, et. al. Alim Pharm Ther. 2021. Newsome, et.al. N Engl J Med. 2020. MRI-PDFF data at 48 wks. Biopsy time 72 wks. Survodutide: Sanyal, et al N Engl J Med 2024. Company press release 6/2024. Biopsy time 48 wks.[4]Without worsening of MASH.

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

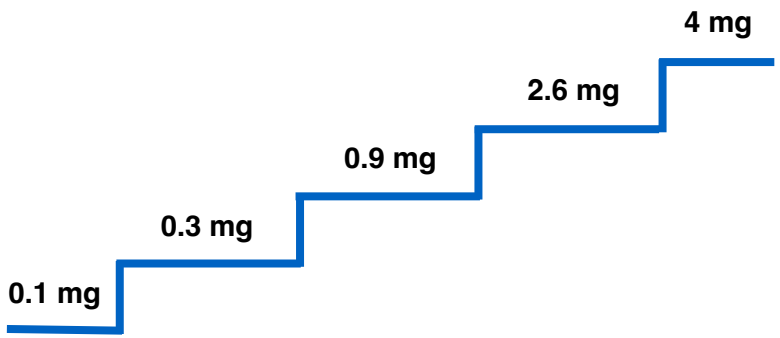
Part 2: Multiple Ascending Dose (MAD) – Dosing PO QD X 14 days

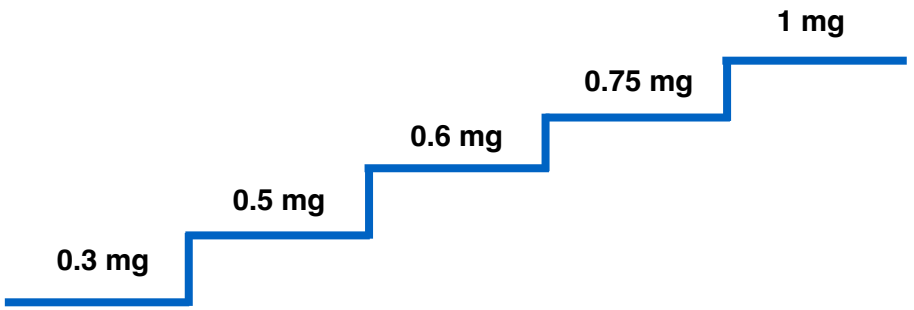
N = up to 80 Subjects with Hyperlipidemia

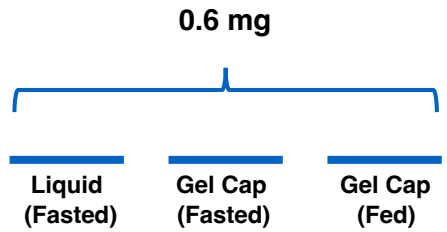
N = 10 per Cohort, N = 8 ALG-055009 and N = 2 Placebo

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

N = 10 Healthy Volunteers







ALIGOS THERAPEUTICS

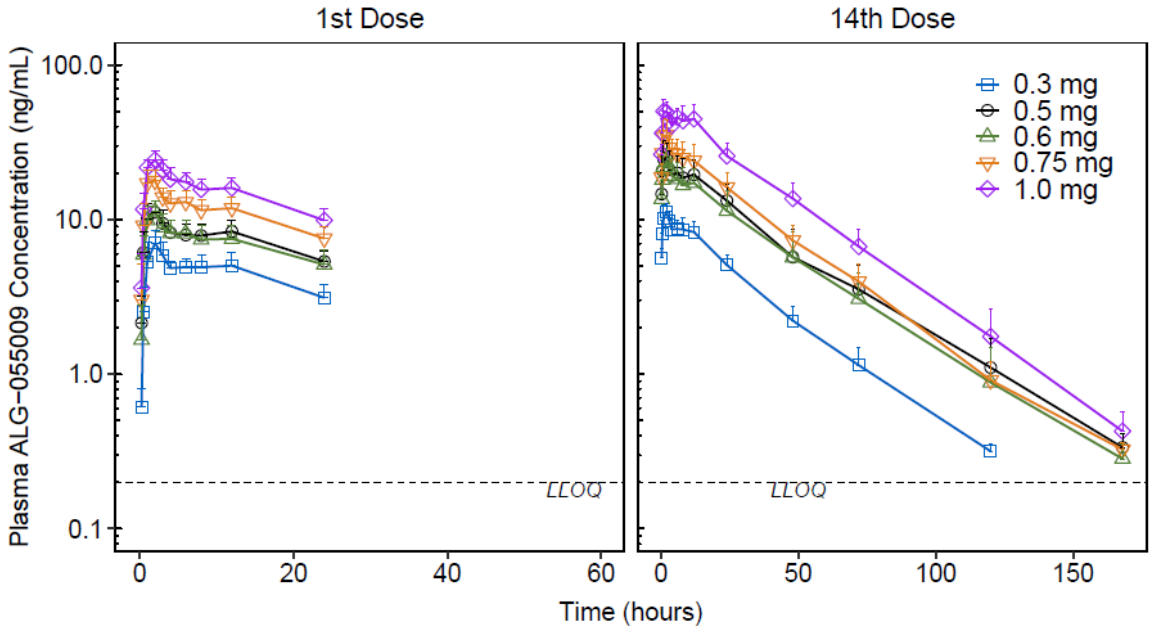
Study ALG-055009-301
Phase 1 Highlights: Doses Well Tolerated with Favorable PK

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

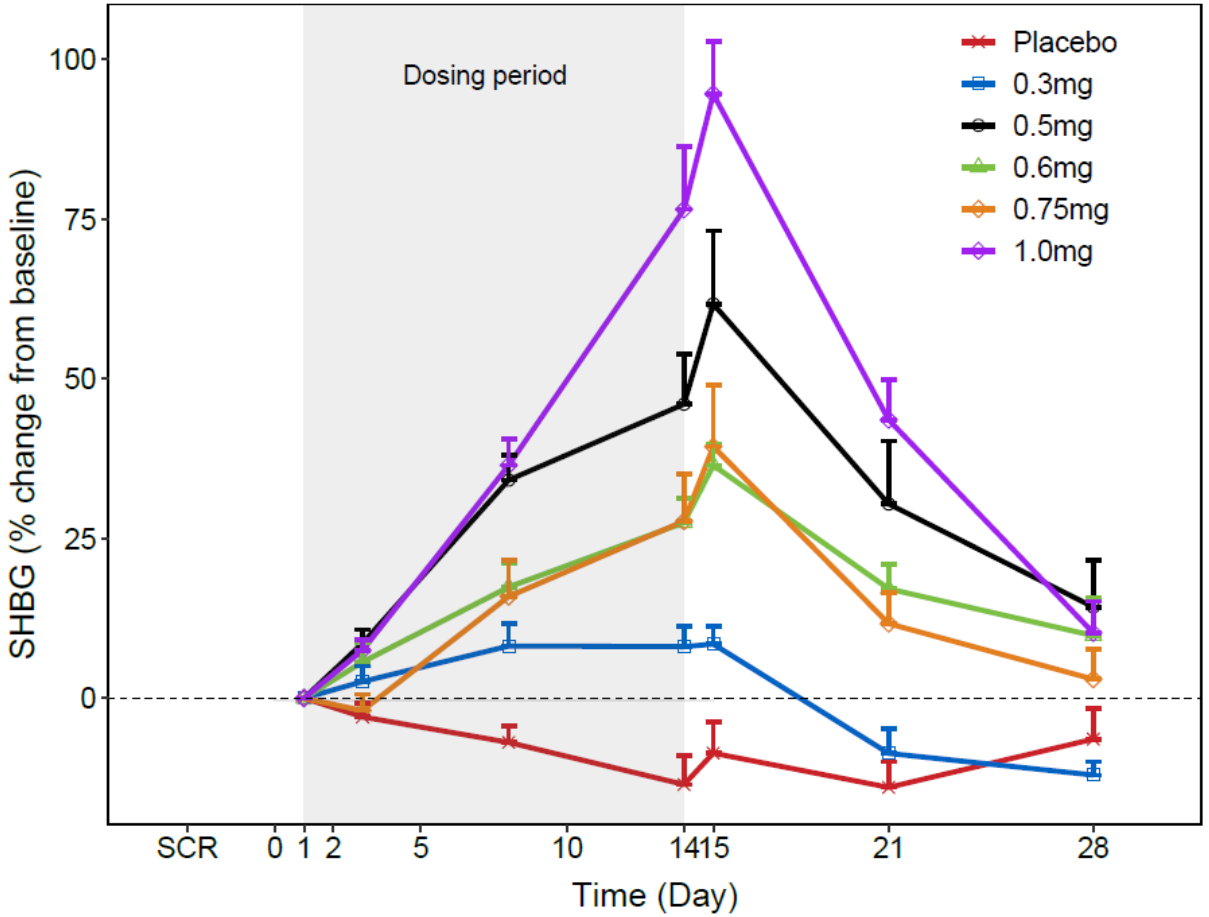


Data presented as mean ± SD

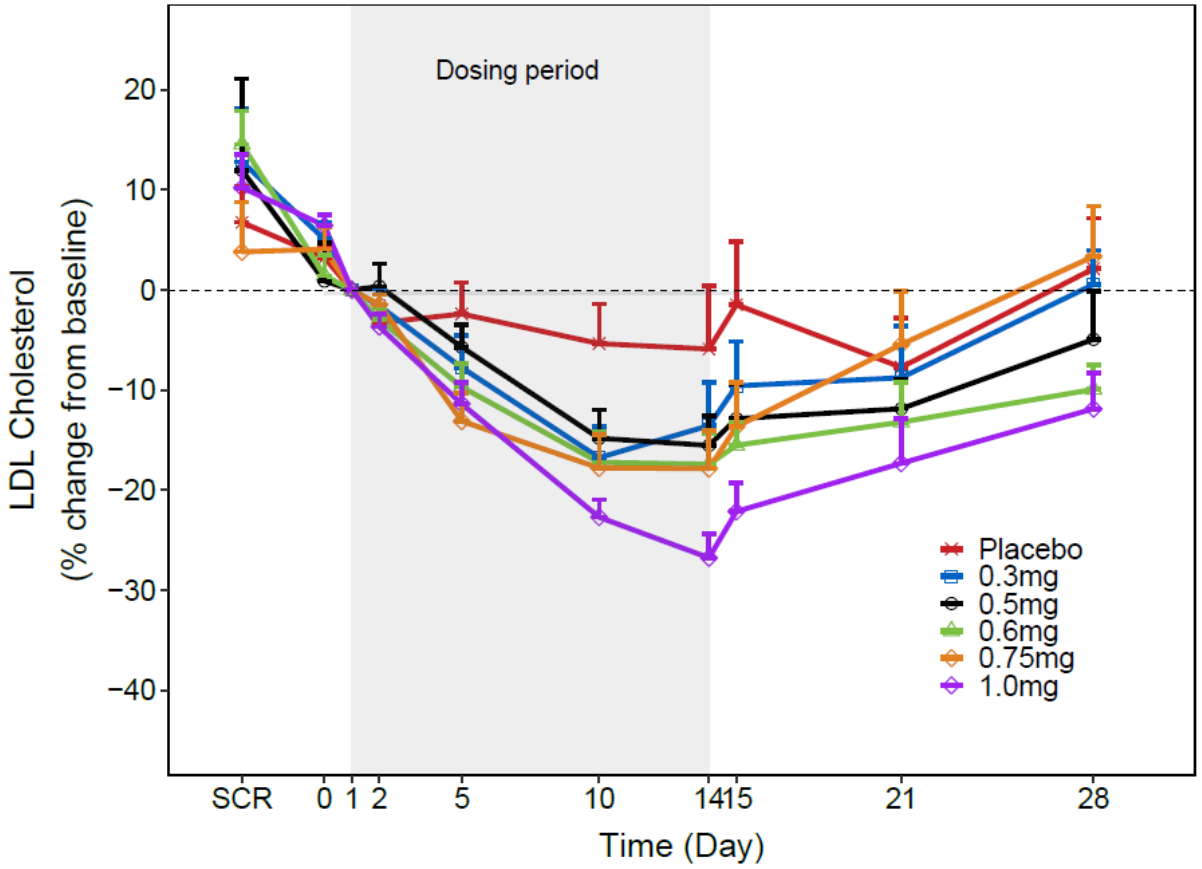
ALIGOS THERAPEUTICS

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

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



Generally dose proportional increases in SHBG

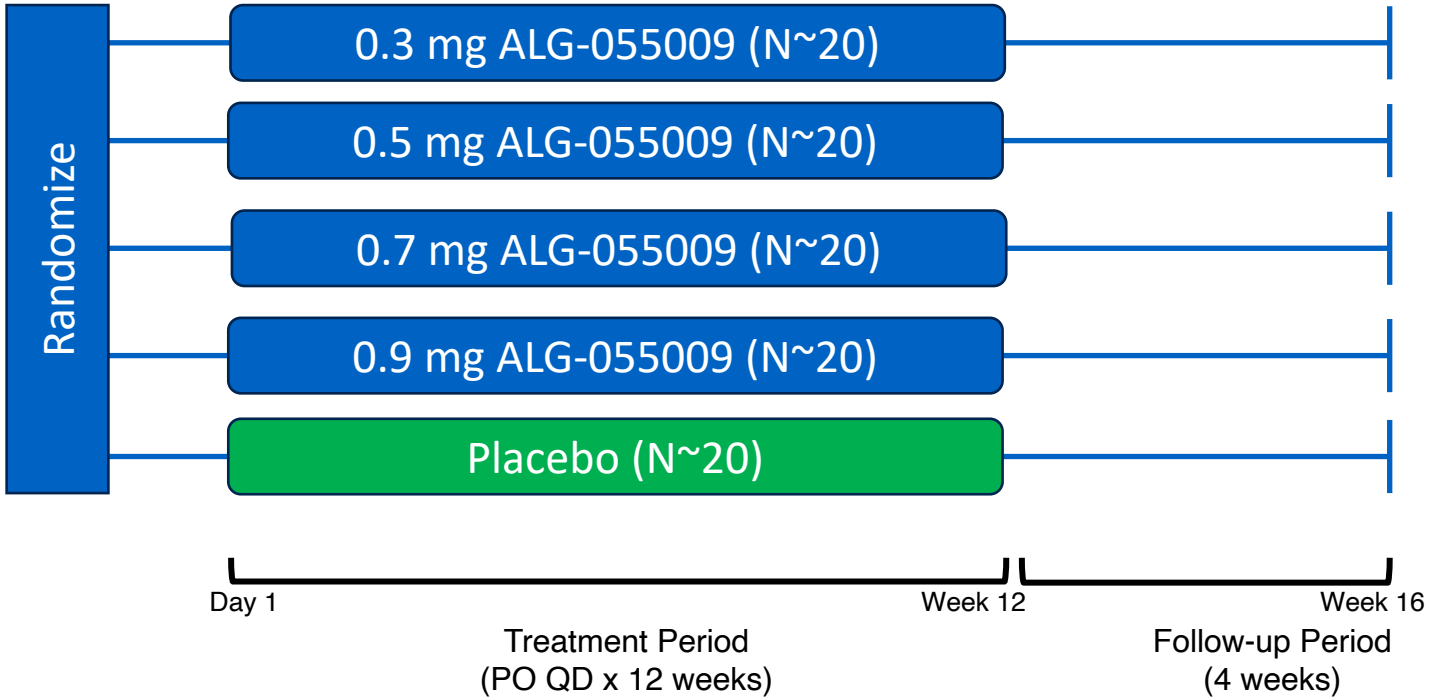


Generally dose proportional decreases in lipids (e.g., LDL, Apo-B, Triglycerides)

ALIGOS THERAPEUTICS

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

Enrollment complete; Topline data anticipated in early Q4 2024

ALIGOS THERAPEUTICS

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%

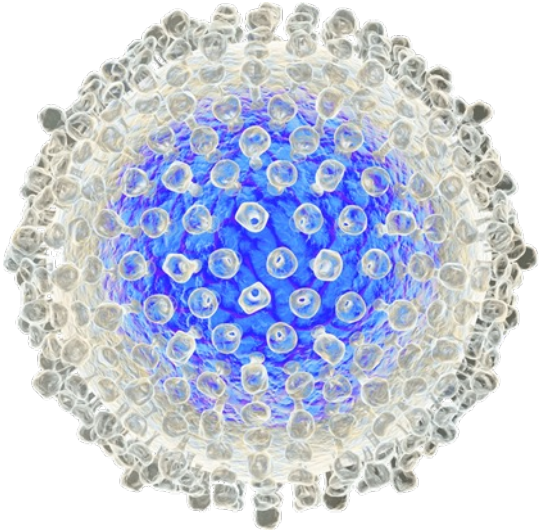
- **Exclusion**
  - Evidence of cirrhosis (clinical, laboratory)
  - Pituitary or thyroid disorder, use of thyroid replacement therapy (within 6 months) or TSH, free T4, or Total T3 >1.1 x ULN or <0.9 x LLN
  - Concerning cardiac history or abnormal ECG
  - Labs:
    - HbA1c ≥9.5%, Platelets ≤135,000/mm^3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

ALIGOS THERAPEUTICS

ALIGOS
THERAPEUTICS

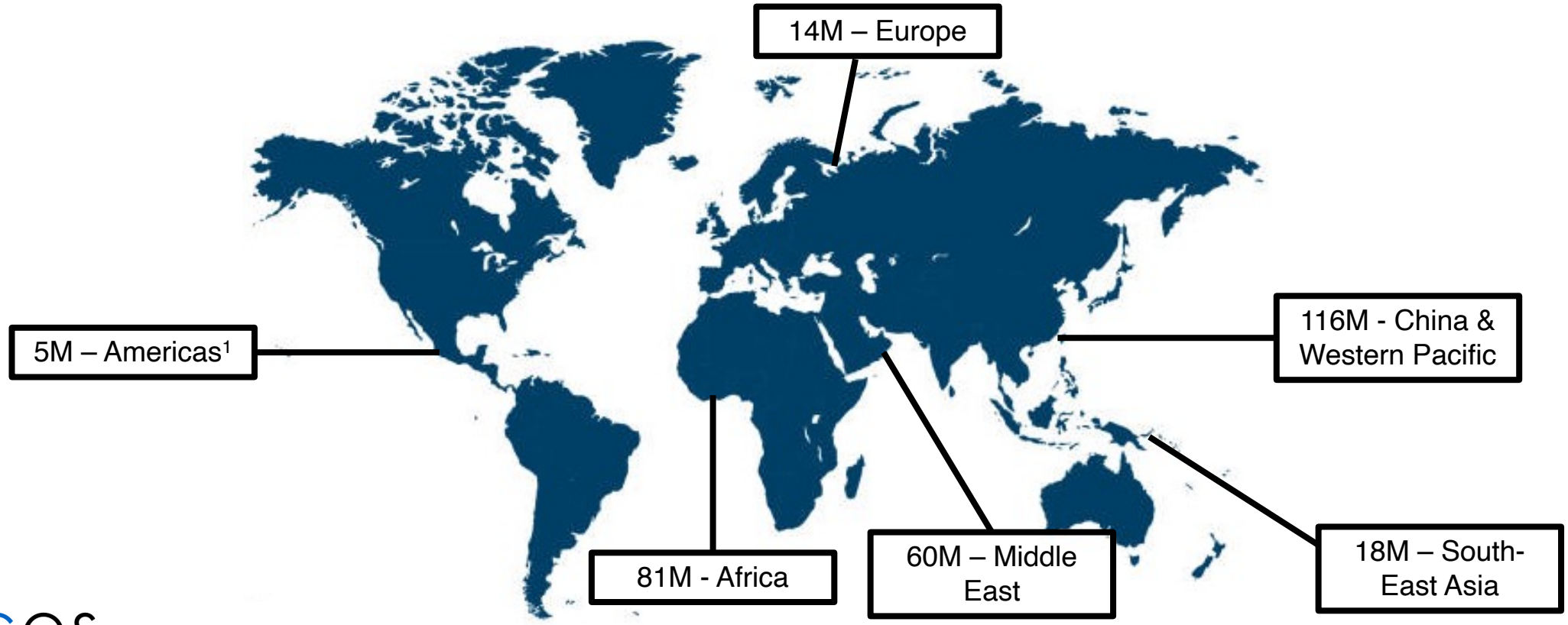
Chronic Hepatitis B

- 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]



ALIGOS THERAPEUTICS

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

ALIGOS THERAPEUTICS

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 suppressive therapy)

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

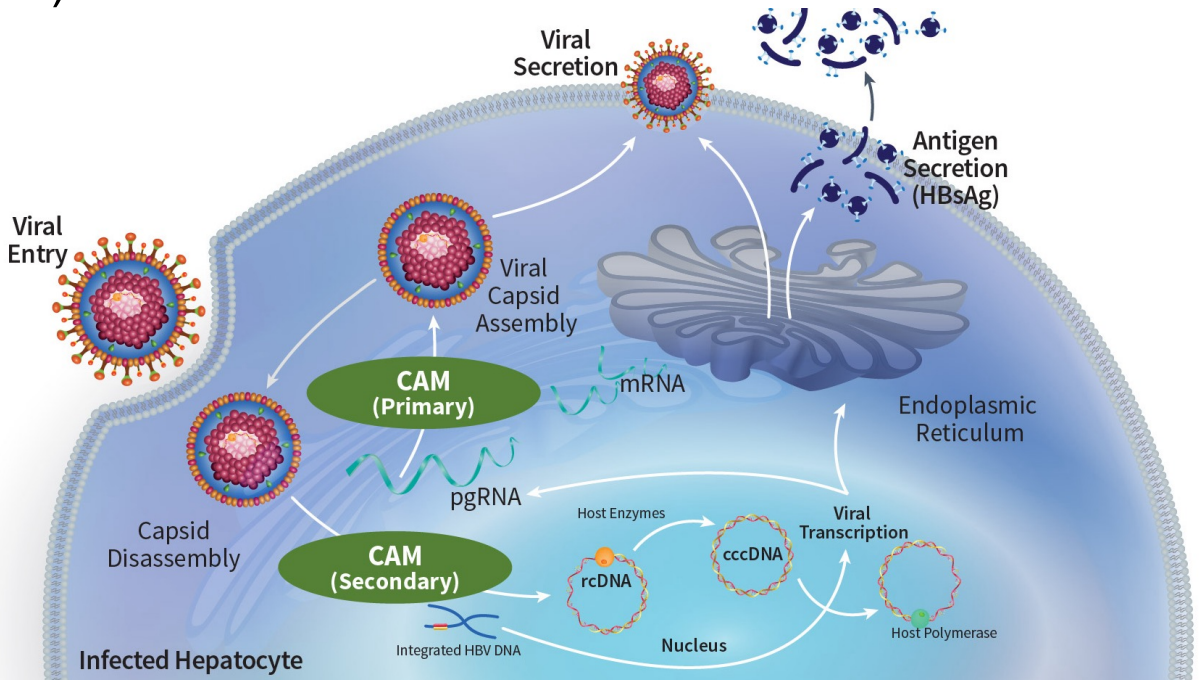
The importance of all relevant biomarkers has not been a key focus for the space. Treating CHB patients is more than reductions in HBV DNA and HBsAg; it is also HBV RNA, HBcrAg, and HBeAg

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

ALIGOS THERAPEUTICS

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

ALIGOS THERAPEUTICS

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 ≤72 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**
  - Clear regulatory path forward for chronic suppressive therapy with superiority label
  - Enabling activities underway; planned Phase 2 IND filing in Q1 2025

ALIGOS THERAPEUTICS

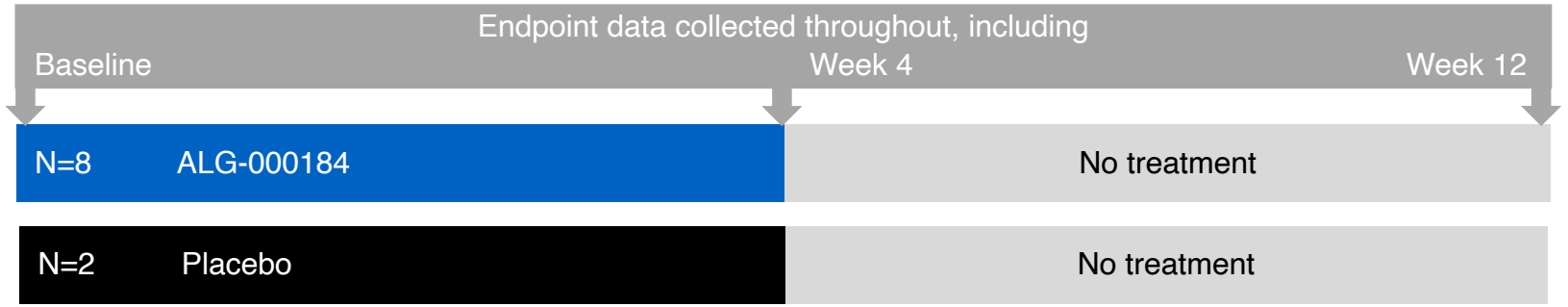
[1]Not including any patent term extension.

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)

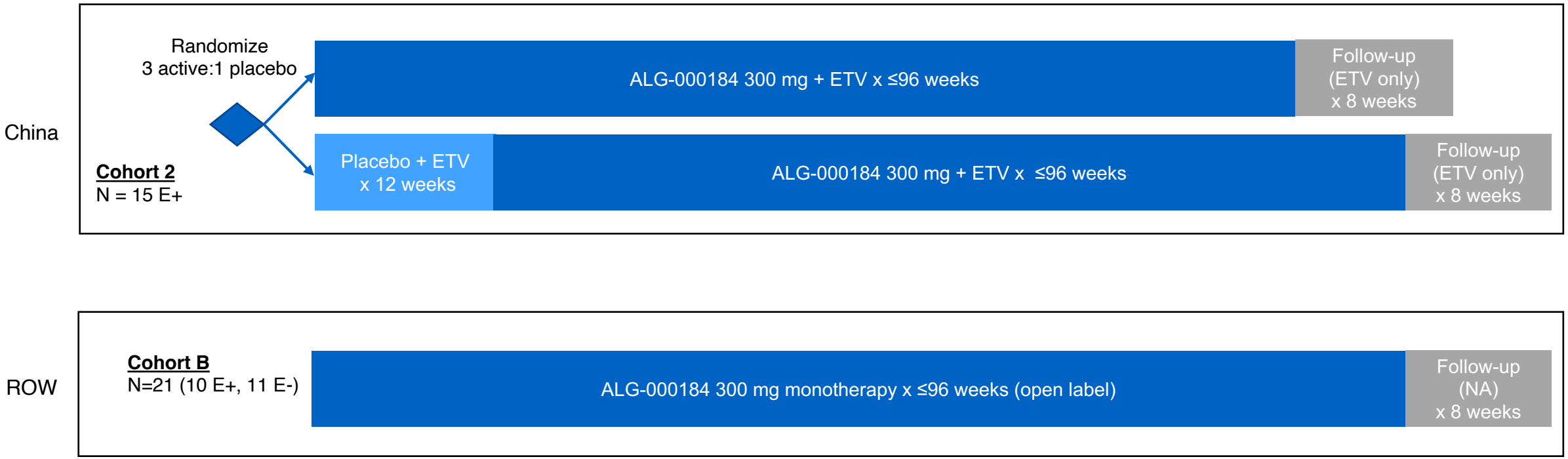


Part 1 & 2: Single oral dose ≤500 mg and multiple oral daily doses ≤250 mg x 7 days well tolerated with linear PK in HV
Part 3: multiple daily doses ≤300 mg well tolerated with linear PK and excellent antiviral activity (DNA, RNA, HBsAg)

ALIGOS THERAPEUTICS

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.

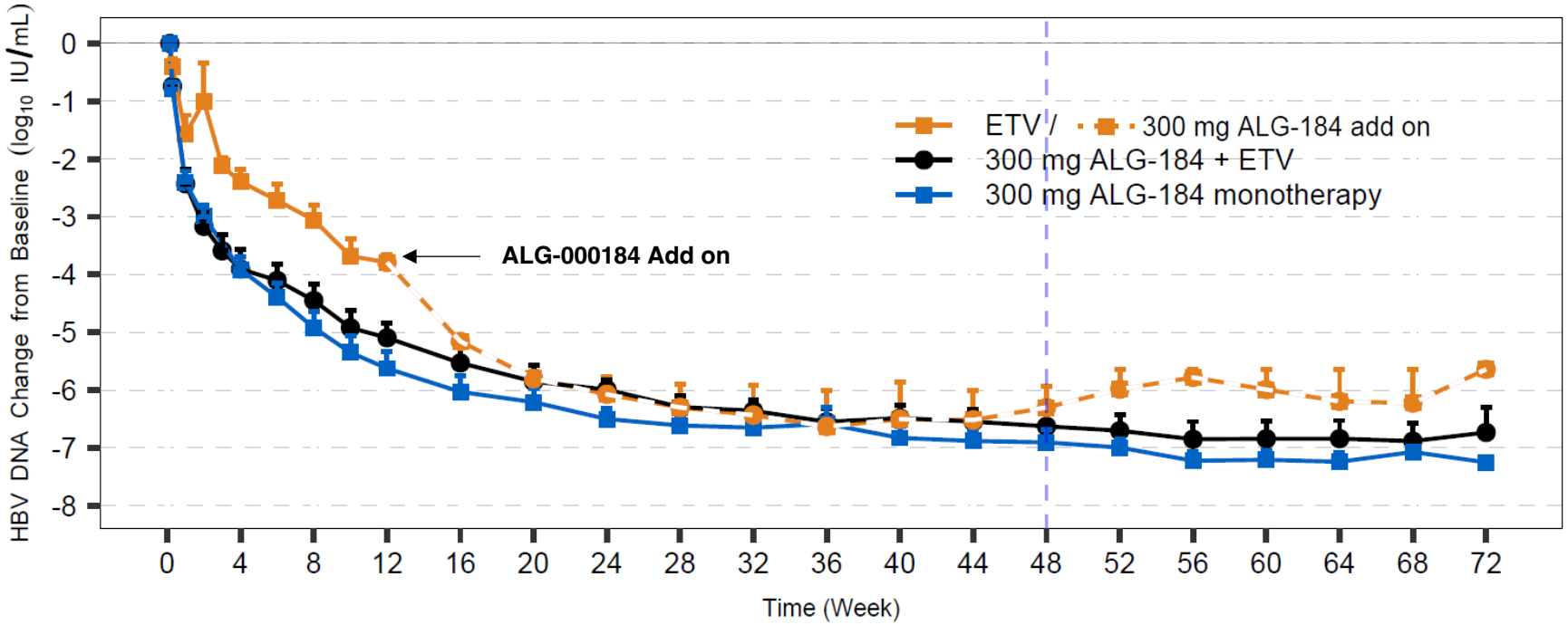
ALG-000184-201 – Long Term Dosing in CHB Subjects
Part 4 Cohort Designs



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; ROW:rest of the world.

Antiviral Effect in CHB Subjects (HBeAg+)
HBV DNA Change from Baseline



ETV / 300 mg ALG-184 add on	3	3	3	3	3	2	1
300 mg ALG-184 + ETV	11	9	9	9	8	8	5
300 mg ALG-184 monotherapy	10	10	10	10	10	8	1

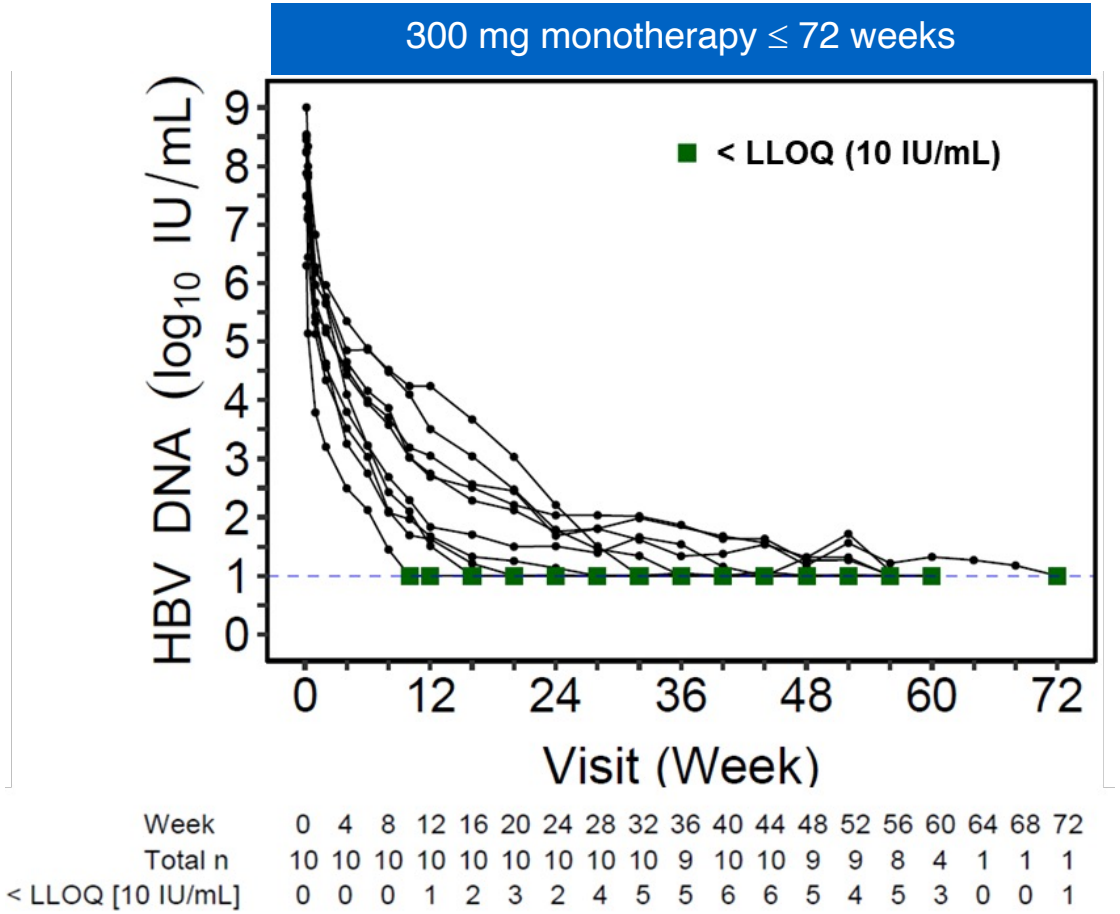
Number of subjects at each timepoint

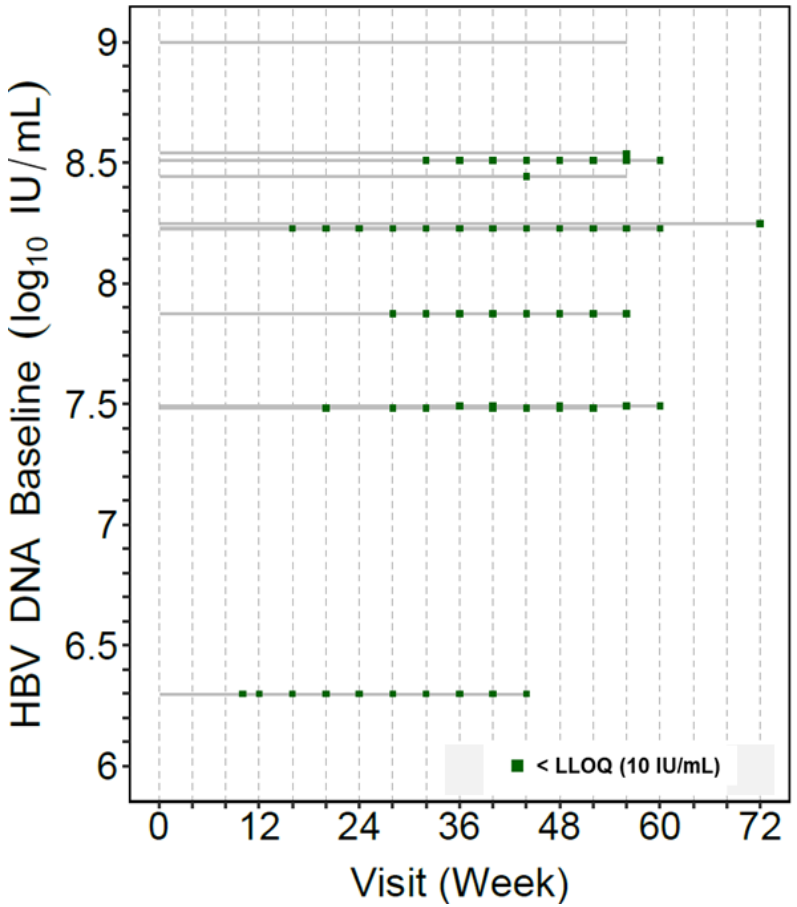
300 mg ALG-000184±ETV
Showed greater HBV DNA reduction than ETV monotherapy
Achieved similar DNA reductions +/-ETV

ALIGOS THERAPEUTICS

Yuen, M-F. et al.,; EASL 2024.

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



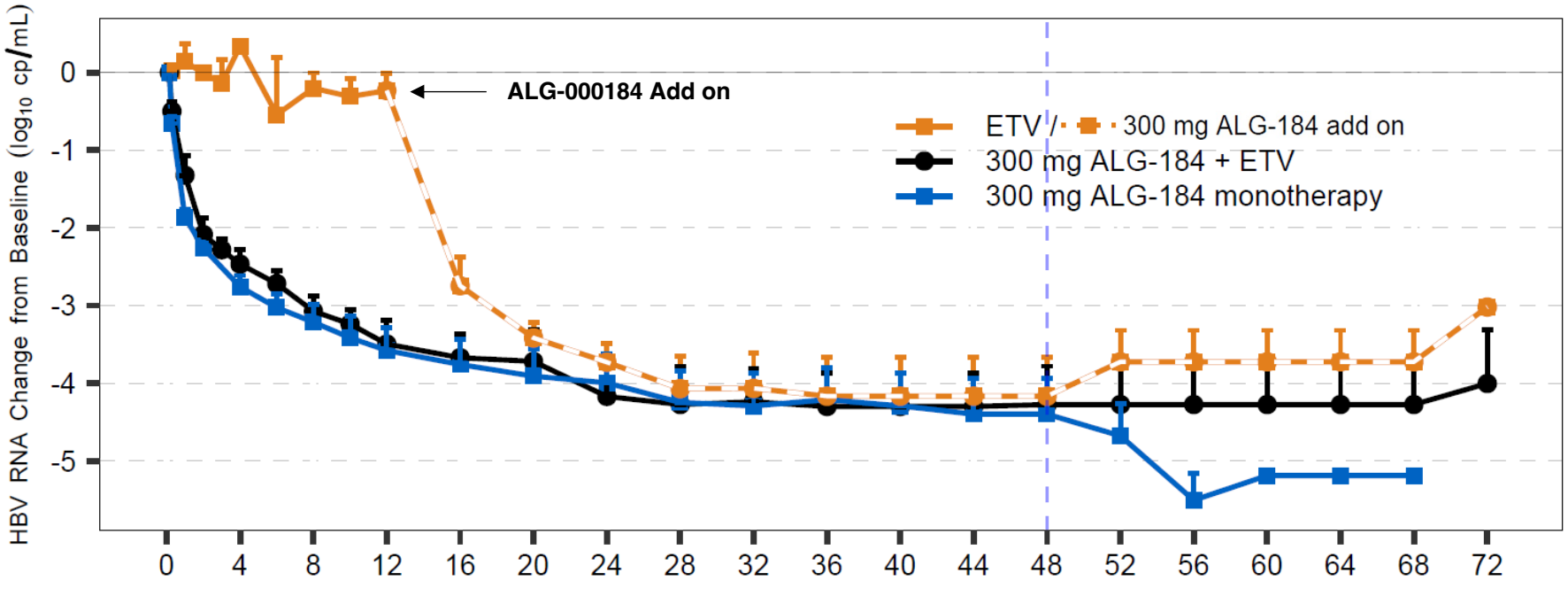


No viral breakthrough during ALG-000184 monotherapy x ≤72 weeks
60% (6/10) of subjects achieved sustained HBV DNA <10 IU/mL by week 48 and 90% (9/10) by week 72

ALIGOS THERAPEUTICS

Yuen, M-F. et al.,; EASL 2024.

300 mg ALG-000184 ± ETV vs. ETV (HBeAg+)
Mean HBV RNA Over Time



ETV / 300 mg ALG-184 add on	3	3	3	3	3	2	1
300 mg ALG-184 + ETV	11	9	9	9	8	8	5
300 mg ALG-184 monotherapy	10	10	10	10	9	1	

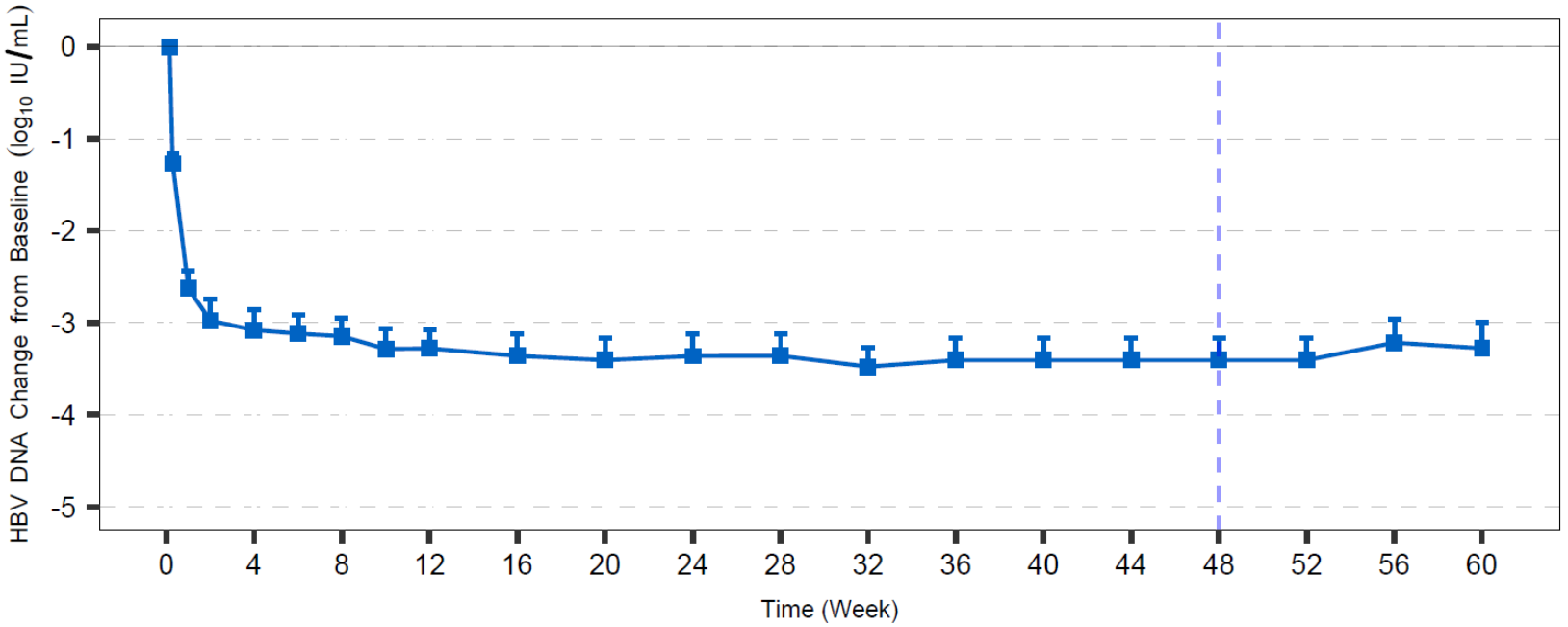
Number of subjects at each timepoint

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
100% (22/22) of subjects experienced HBV RNA < LLOQ by week 40
RNA levels correlated with HCC risk^

ALIGOS THERAPEUTICS

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

Antiviral Effect in CHB Subjects (HBeAg-)
HBV DNA Change from Baseline



Week	0	4	8	12	16	20	24	28	32	36	40	44	48	52	56	60
Total n	11	11	11	11	10	11	11	10	11	11	11	11	11	11	9	5
≤ LLOQ [10 IU/mL]	0	10	10	10	10	11	11	10	11	11	11	11	11	11	9	5

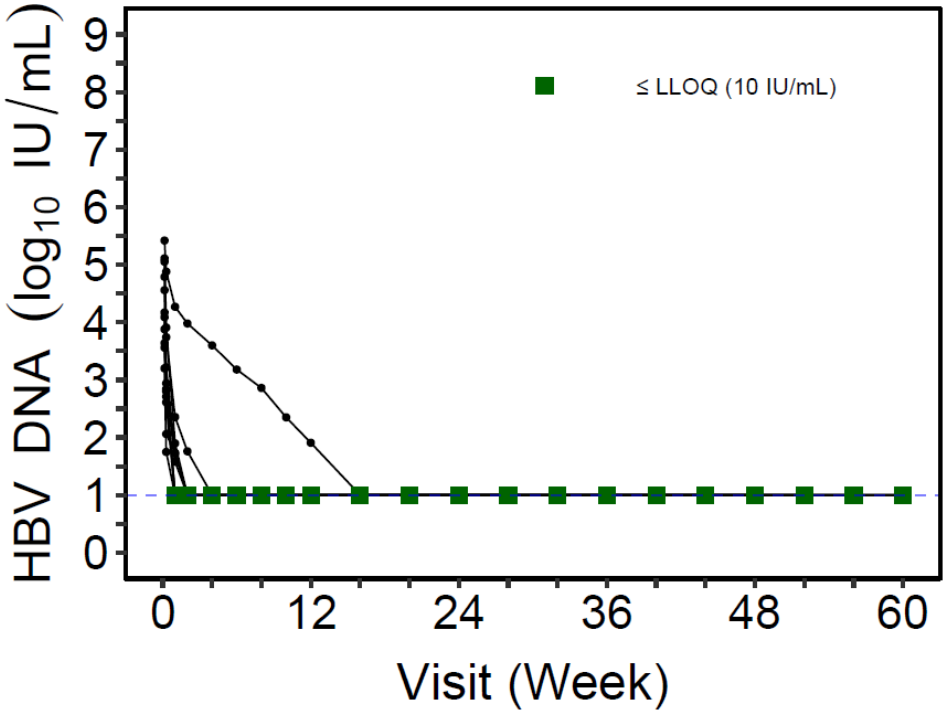
Number of subjects at each timepoint

300 mg ALG-000184 showed rapid and sustained DNA reductions with no viral breakthrough

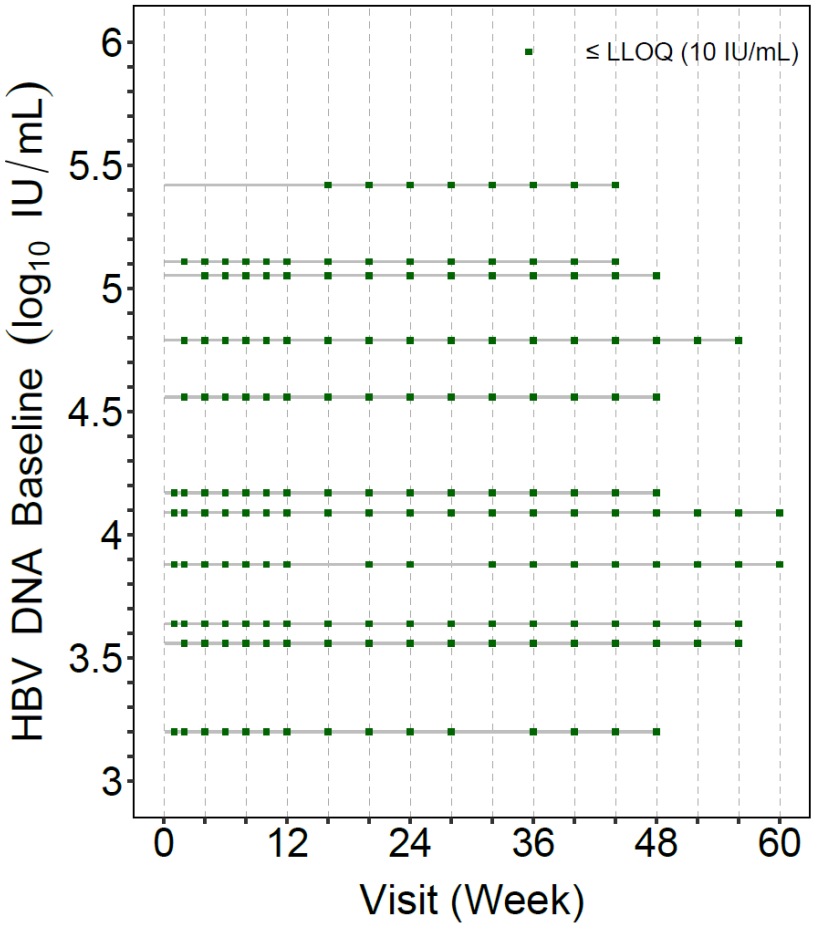
ALIGOS THERAPEUTICS

Agarwal, K. et al. EASL 2024.

300 mg ALG-000184 Monotherapy (HBeAg-)
Individual HBV DNA Decline



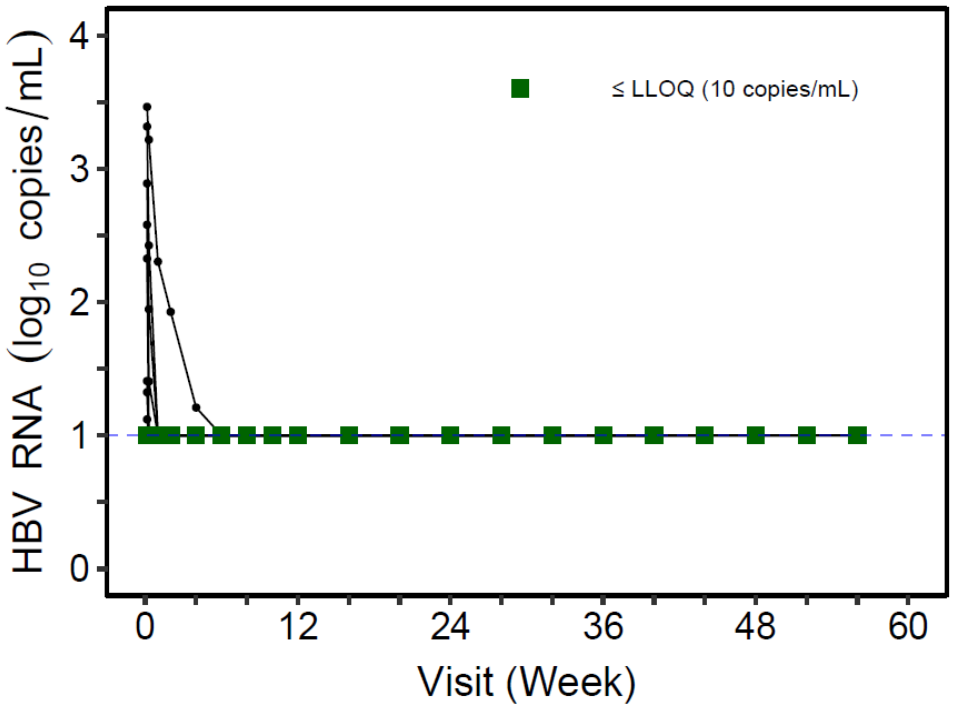
Week	0	4	8	12	16	20	24	28	32	36	40	44	48	52	56	60
Total n	11	11	11	11	10	11	11	10	11	11	11	11	10	7	5	4
≤ LLOQ [10 IU/mL]	0	10	10	10	10	11	11	10	11	11	11	11	10	7	5	4



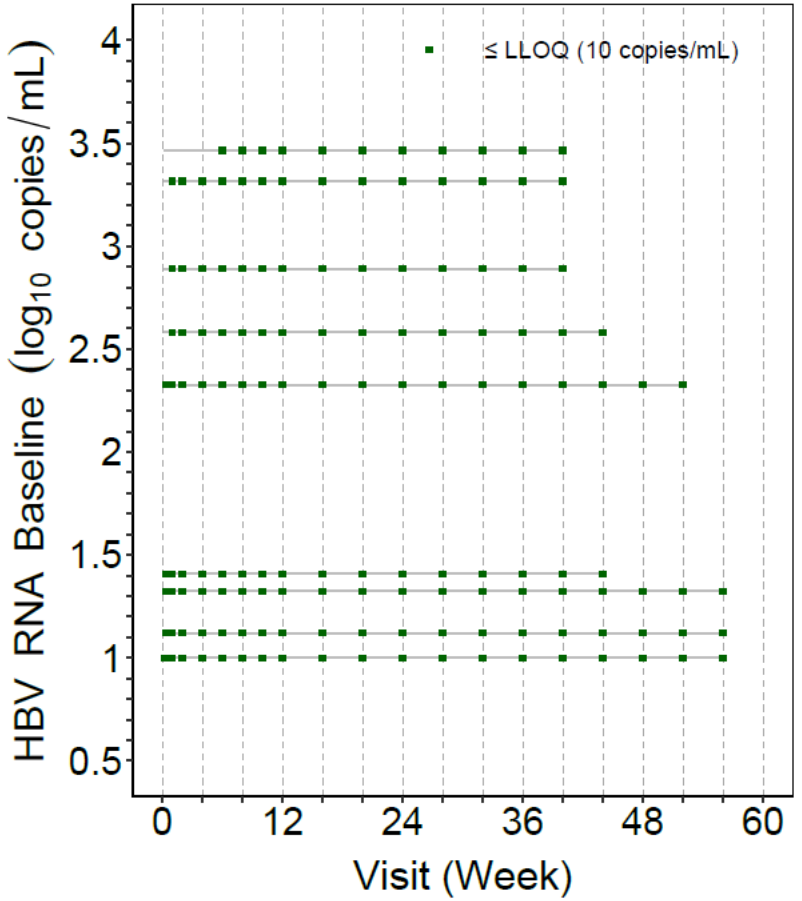
100% (11/11) of subjects achieved HBV DNA < LLOQ (10 IU/mL) by Week 20

91% (10/11) of subjects achieved HBV DNA < LLOD (< 4.92 IU/mL) by Week 48

300 mg ALG-000184 Monotherapy (HBeAg-)
Individual HBV RNA Decline



Week	0	4	8	12	16	20	24	28	32	36	40	44	48	52	56
Total n	11	11	11	11	10	11	11	10	11	11	11	8	5	5	3
≤ LLOQ [10 copies/mL]	3	10	11	11	10	11	11	10	11	11	11	8	5	5	3



100% (11/11) of subjects achieved HBV RNA < LLOQ (10 copies/mL) by week 6

ALIGOS THERAPEUTICS

Agarwal, K. et al. EASL 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)		% Patients < LLOQ at Week 96 (by HBV DNA Assay Sensitivity)	
		% < LLOQ 29 IU/mL	% < LLOQ 10 IU/mL	% < LLOQ 29 IU/mL	% < LLOQ 10 IU/mL
E-	TDF (n=140)[a]	93%	17%	91%	28%
	TAF (n=285)[a]	94%	21%	90%	30%
	300 mg ALG-000184 (n=11)[d]	**11/11 (100%)**	**11/11 (100%)**	**TBD**	**TBD**
E+	TDF (n=292)[b]	67%	N/A	75%	7%
	TAF (n=581)[b]	64%	N/A	73%	10%
	300 mg ALG-000184 (n=10)[c]	**10/10 (100%)**	**6/10 (60%)**	**TBD**	**TBD**

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

ALIGOS THERAPEUTICS

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., EASL 2024. [d] Agarwal, K. et al. EASL 2024.

Chronic Suppression
Well Defined, Validated Approval Pathway

Regulatory pathway for chronic suppressive therapy endorsed by FDA, EMEA, and China FDA (CDE)
Primary endpoint: Subjects with HBV DNA <LLOQ at Week 48 following treatment with investigational agent versus standard of care (nucleos(t)ide analogs)

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.

Aligos has received FDA feedback supporting subsequent studies utilizing this pathway

ALIGOS THERAPEUTICS

HBV Guidance from: FDA 2022; EMEA 2006; China 2023; LLOQ: lower limit of quantitation.

ALG-000184 Phase 2 Chronic Suppression Study
Planned Efficacy Endpoints

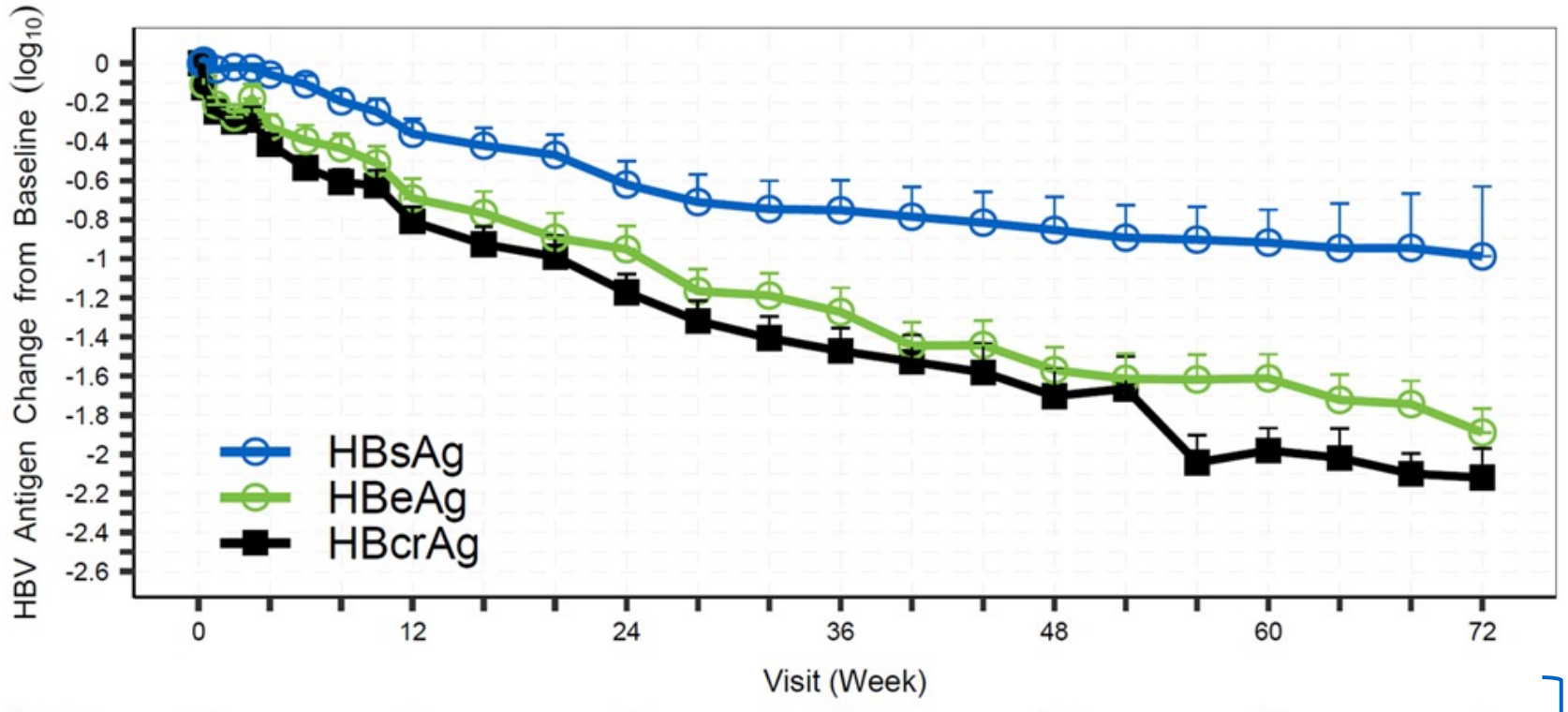
- Primary endpoint (approvable endpoint*)
  - Proportion of subjects with HBV DNA < 10 IU/mL at W48 for both HBeAg+/- CHB infected subjects
- Secondary endpoints (clinically meaningful and/or corroborative)
  - HBeAg seroconversion in HBeAg+ CHB infected subjects
  - Reduction of HBsAg, HBcrAg, HBeAg
  - Reduction of cccDNA level and/or related serum biomarkers
  - Reduction of HBV integrants^
  - HBV RNA < LLOQ^

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

ALIGOS THERAPEUTICS

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

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



HBsAg	21	19	19	19	17	16	6
HBeAg	21	19	19	19	17	16	6
HBcrAg	21	19	19	19	17	9	5

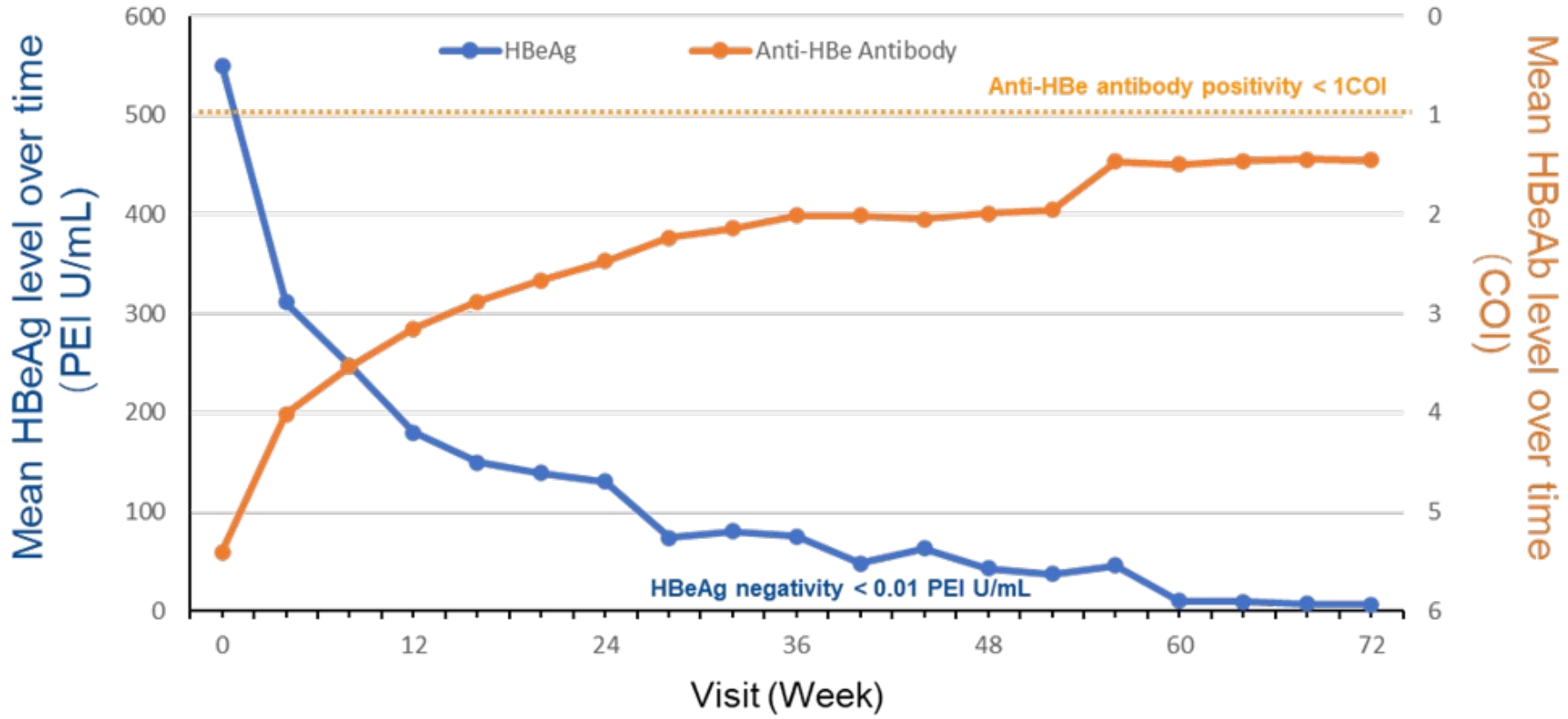
Number of subjects at each timepoint

Continued substantial HBsAg, HBeAg, and HBcrAg reductions noted with combo through week 72
Mean max declines: 2.1, 2.6 and 2.7 log_{10} IU/mL, respectively

ALIGOS THERAPEUTICS

Graph plots subjects initially randomized to ALG-000184 + ETV and were compliant (confirmed by PK).
Yuen, M-F. et al.,; EASL 2024.

300 mg ALG-000184 ± ETV
Mean HBeAg and Anti-HBe Antibody Level Over time



Anti-HBe antibody (HBeAb) level showed positive trend with decline of HBeAg

ALIGOS THERAPEUTICS

Yuen, M-F. et al.,; EASL 2024.

Safety Overview – 300 mg ALG-000184 ± ETV
Treatment Emergent Adverse Events

	HBeAg-Positive Population		HBeAg-Negative Population
ALG-000184 Regimen	300mg QD + ETV	300mg QD	300mg QD
N of subjects	N=15	N=10	N=11
Serious Adverse Events (SAEs)	None		
Treatment emergent AEs (TEAEs) leading to study drug discontinuation	None		
Subjects with Grade ≥ 3 TEAEs	4 ALT/AST↑ (n=3); neutropenia↑ (n=1) eGFR↓ (n=1); Uric acid ↑ (n=1)	3 ALT/AST↑ (n=3)	2 ALT/AST↑ Cholesterol/Triglycerides ↑
Concerning TEAE, laboratory, ECG, vital sign, or physical examination findings or trends	None		

- All Grade ≥3 TEAEs of liver transaminase elevations were transient, resolved (n=6) and improving (n=1) in setting of continued dosing with study drug, and were associated with a potent antiviral effect. None were considered clinically concerning by the AFC.
- Neutropenia considered probably related to an acute respiratory infection and resolved post-infection in the setting of continued dosing with study drug
- Grade 3 eGFR decrease was reported in one subject with Grade 2 baseline level; returned to baseline level within 2 weeks in setting of continued study dosing
- Uric acid increase and cholesterol/triglycerides increase were asymptomatic and fluctuated between Grade 1 and 3 in setting of continued dosing

A favorable safety profile was observed in untreated HBeAg+ and HBeAg- CHB subjects with long term (≤88 weeks) treatment with 300 mg QD ALG-000184 ± ETV

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 at APASL, EASL,
- ✓Clear regulatory path forward for chronic suppressive therapy with superiority label
- Additional interim data readouts expected at AASLD
- Phase 2 enabling activities ongoing; planned Phase 2 IND filing in Q1 2025

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 6/30/24: Cash, cash equivalents and investments were $94.5M
- The Company believes our cash, cash equivalents and investments will provide sufficient funding of planned operations through the end of 2025


ALIGOS
THERAPEUTICS