ALIGOS

THERAPEUTICS

CORPORATE PRESENTATION

November 2024

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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.

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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 HERALD data: primary endpoint achieved; up to 46% placebo-adjusted median relative reductions in liver fat
  - Phase 2b enabling activities ongoing; estimated completion middle of 2025
- **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 AASLD
  - Phase 1b exploratory combination study with mipeginterferon alfa-2b in collaboration with Amoytop
  - Clear regulatory path forward with the FDA and China's National Medical Products Administration for chronic suppressive therapy with superiority label compared to standard of care
  - Phase 2 enabling activities ongoing; planned submission of Phase 2 protocol under the recently cleared IND in Q1 2025

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As of 9/30/24 - Cash, cash equivalents and investments were $74.9M.
Projected runway through the end of 2025

Aligos Development Portfolio

Multiple Milestones Anticipated in 2024

Candidate	Indication	MOA	Discovery	Nonclinical	Phase 1	Phase 2	Phase 3	Partner
ALG-055009	MASH	THR-β Agonist						
ALG-000184	CHB Monotherapy	CAM-E						
ALG-000184	CHB Combination[1]	CAM-E + PEGBING® + ETV (mipeginterferon alfa-2b)						AMOYTOP BIOTECH
ALG-097558	Covid-19*	Protease Inhibitor						NIH National Institute of Allergy and Infectious Diseases; agile Coronavirus Drug Testing Initiative; KU LEUVEN

*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. CAM-E = capsid assembly modulator (empty); CHB = chronic hepatitis B; Covid-19 = coronavirus disease of 2019 (SARS-CoV-2); 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. [1]Amoytop sponsoring combo study.

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MASH

- Epidemiology, Pathogenesis
- Competitive Landscape

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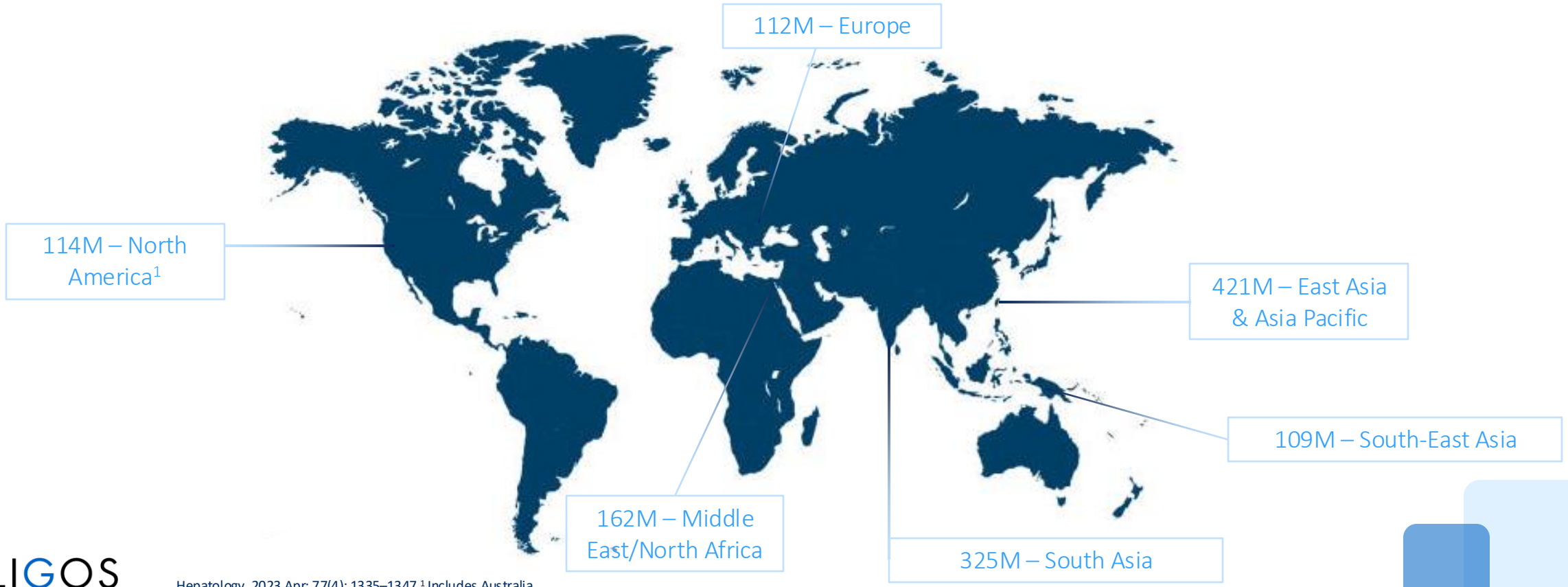
MASLD/MASH

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



Hepatology. 2023 Apr; 77(4): 1335–1347.[1] Includes Australia.

MASH Pathogenesis

Fatty acids = primary source of excess energy supply

(from de novo lipogenesis & lipolysis)

↓

Lipotoxic Species

↓

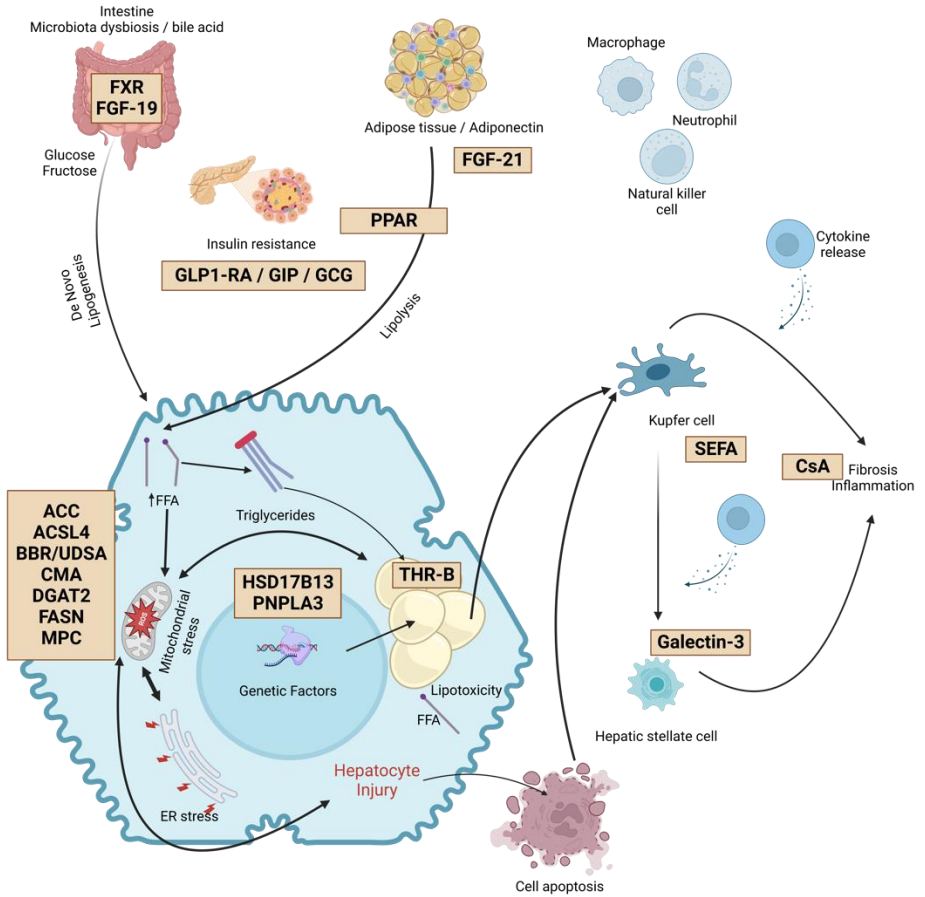
Liver Injury

Endoplasmic reticulum stress

Mitochondrial dysfunction

Pro-inflammatory cytokines release

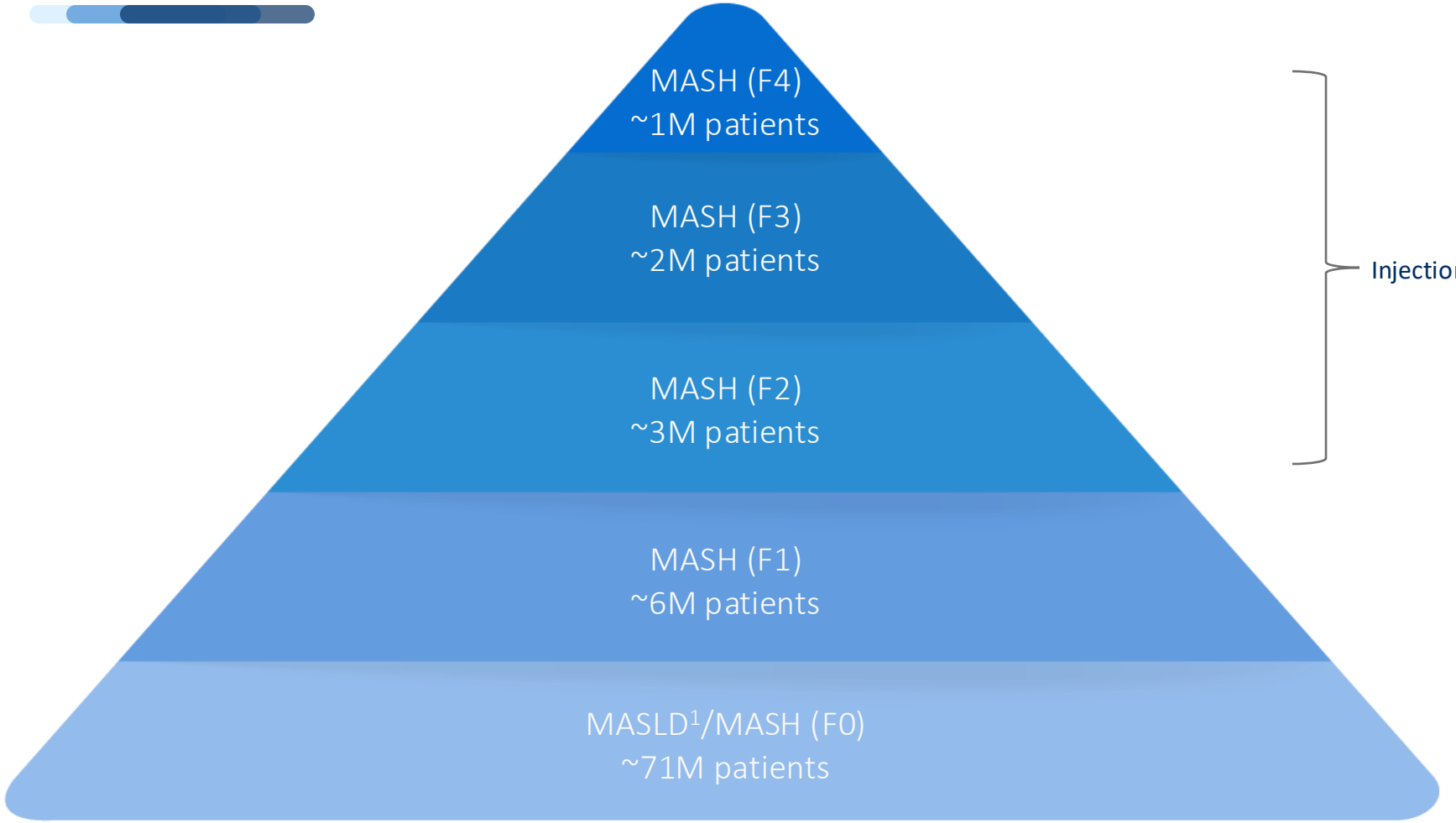
Apoptosis



Harrison, et al. Clin Gastro and Hep. 2023.

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

MASLD/MASH Potential Future Treatment Paradigm



[1]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.

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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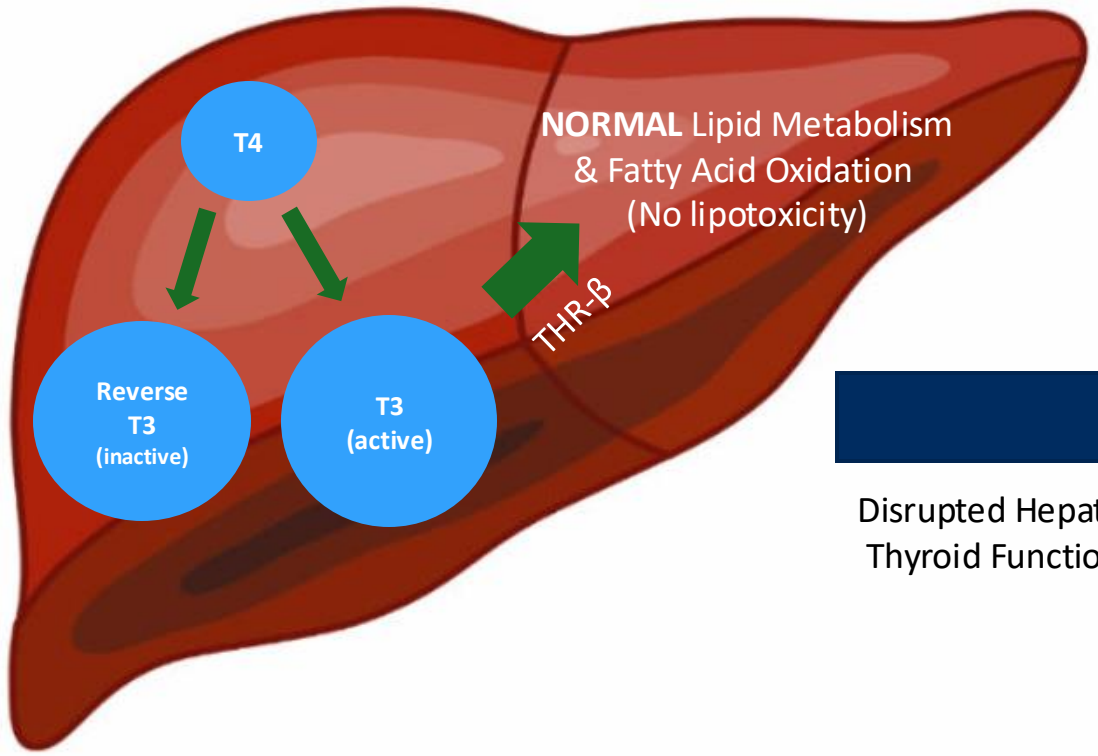
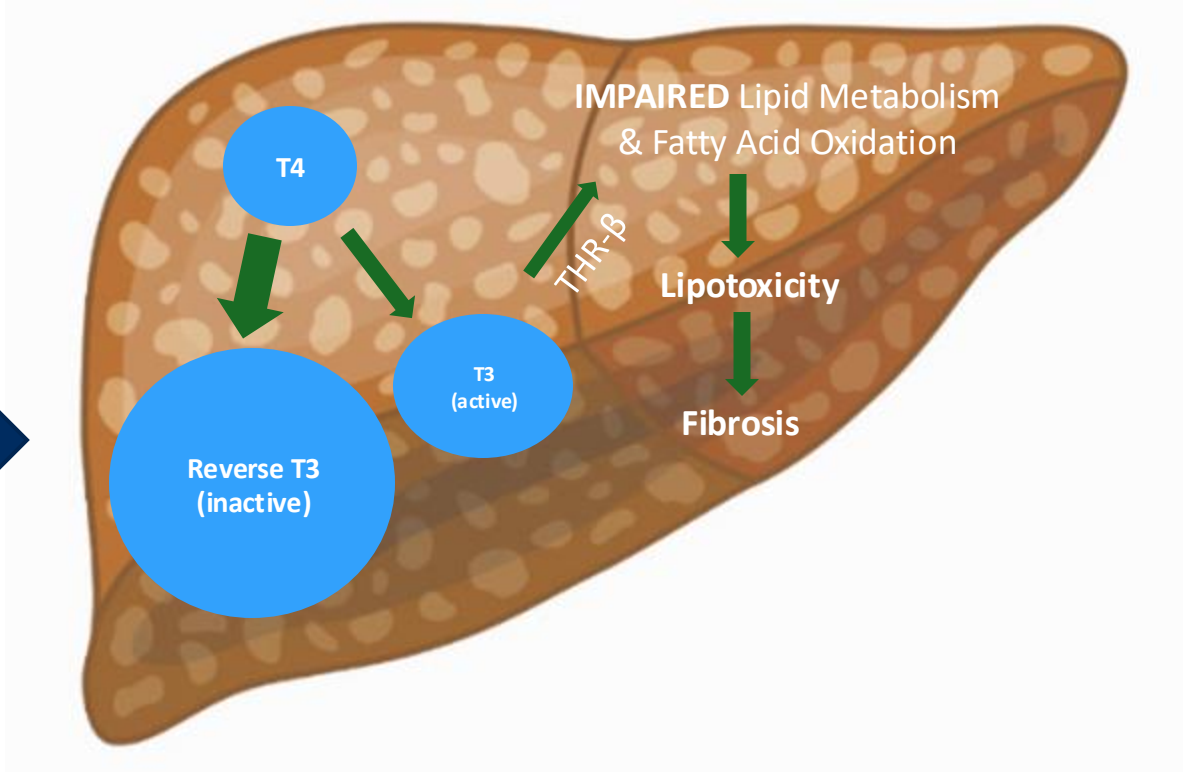
Role of Hepatic Thyroid Dysfunction in MASLD/MASH Pathogenesis

Healthy Liver



Disrupted Hepatic
Thyroid Function

Steatotic Liver



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

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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ALG-055009

Small molecule THR-β agonist

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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
- **Phase 1 highlights**
  - PK - dose proportional, low variability, $t_{1/2}$ ~20 hours (enhanced vs. resmetirom)
  - Well tolerated without clinical safety signals
  - Pharmacodynamics - dose proportional increases in SHBG, decreases in atherogenic lipids
- **Phase 2a HERALD study highlights**
  - Primary endpoint achieved with robust reductions in liver fat content at Week 12
    - Up to 46% placebo-adjusted median reductions
    - Up to 70% of patients with ≥30% decrease in liver fat
  - Significant reductions in atherogenic lipids, including LDL-C, lipoprotein (a) and apolipoprotein b
  - Dose-dependent increases in SHBG (marker of THR-β activation in liver)
  - Well-tolerated, with rates of GI-related AEs similar to placebo

[1]Not including any patent term extension.

Study ALG-055009-301: SAD, MAD, Relative BA

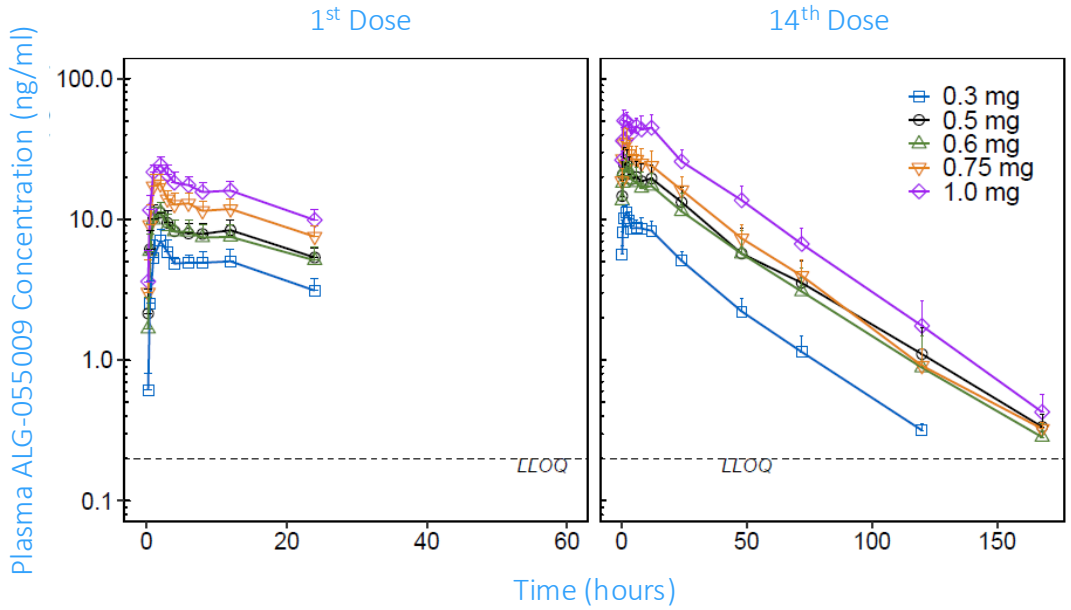
Phase 1 Highlights: Doses Well Tolerated with Favorable PK

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
  - $t_{1/2}$ = 20-23 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

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

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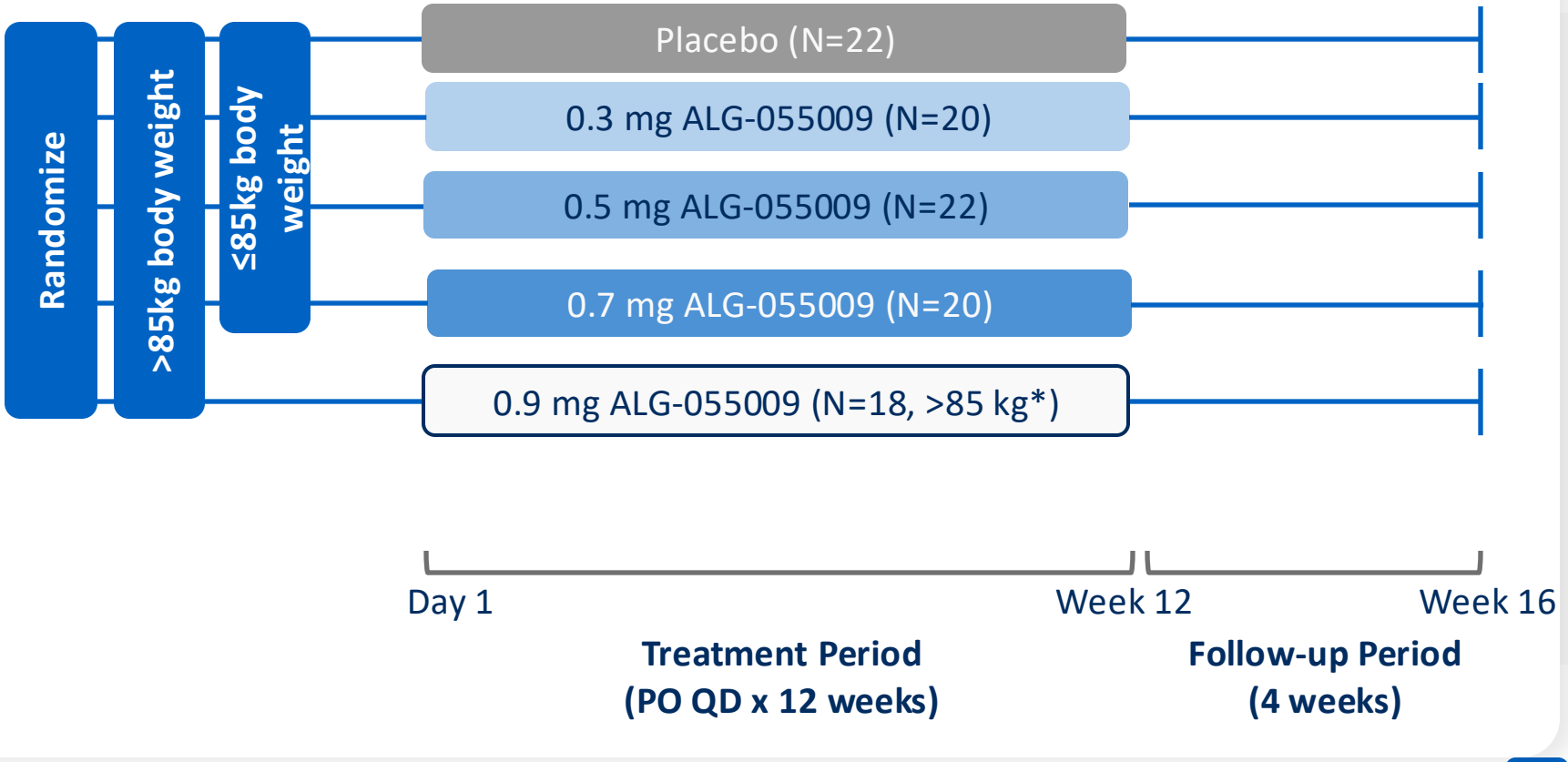
ALG-055009

Phase 2a HERALD Study Design

- **Population:** 102 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



NCT06342947.

*Only subjects weighing >85 kg were enrolled in the 0.9 mg dose group, no body weight restrictions were implemented in other dose groups

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HERALD: Baseline Characteristics Generally Balanced Across Arms

Consistent with Today's At-risk MASH Population

	Placebo (N=22)	ALG-055009			
		0.3 mg (N=20)	0.5 mg (N=22)	0.7 mg (N=20)	0.9 mg* (N=18, >85 kg)
Age, mean (years)	48.5	53.3	49.5	51.4	48.1
Female, n (%)	21 (95.5)	12 (60.0)	8 (36.4)	14 (70.0)	8 (44.4)
Hispanic, n (%)	13 (59.1)	9 (45.0)	8 (36.4)	8 (40.0)	9 (50.0)
BMI, mean (kg/m^2)	42.1	37.8	39.0	37.4	40.2
Weight, mean (kg)	109.1	107.0	115.2	106.4	116.5*
MRI-PDFF, mean (%)	18.6	18.2	17.9	19.4	19.0
Type 2 Diabetes, n (%)	11 (50.0)	9 (45.0)	10 (45.5)	10 (50.0)	7 (38.9)
GLP-1 Agonists, n (%)	4 (18.2)	3 (15.0)	6 (27.3)	5 (25.0)	1 (5.6)
Statins, n (%)	4 (18.2)	11 (55.0)	6 (27.3)	8 (40.0)	6 (33.3)
ALT, mean (U/L)	39.5	39.9	53.0	38.3	38.5

BMI = body mass index; ALT = alanine aminotransferase; GLP-1 = glucagon-like peptide-1; BW = body weight

*Only subjects weighing >85 kg were enrolled in the 0.9 mg dose group, no body weight restrictions were implemented in other dose groups

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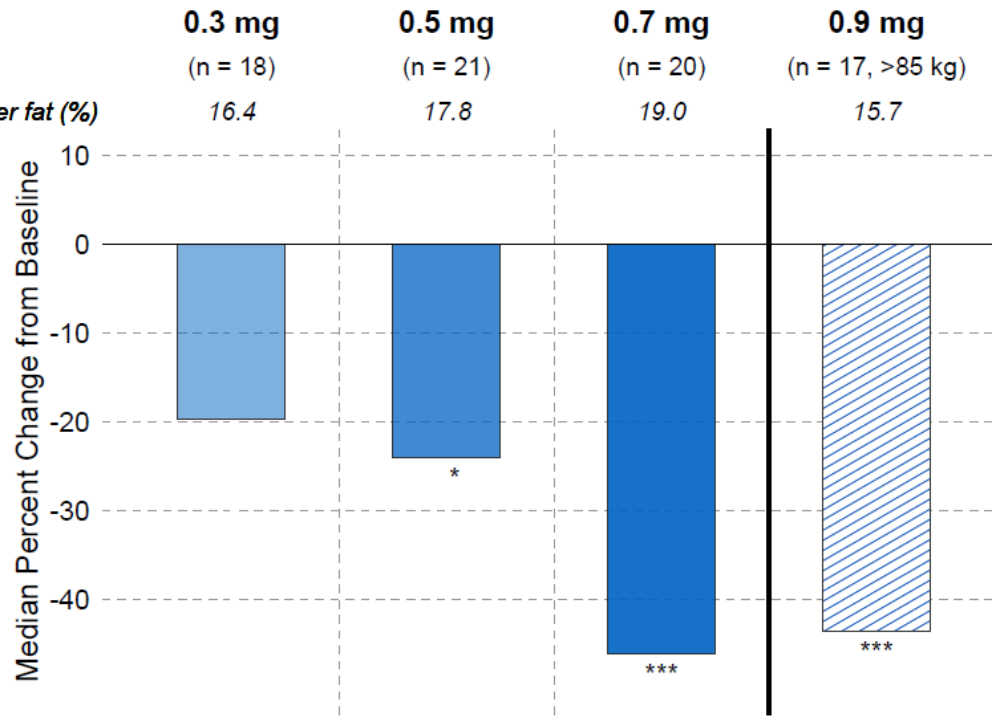
HERALD: Primary Endpoint Achieved

Placebo-Adjusted Median Relative Change in Liver Fat at Week 12



	0.3 mg	0.5 mg	0.7 mg	0.9 mg
Placebo-adjusted % change	**-19.7**	**-24.1**	**-46.2**	**-43.6**
p-value vs. Placebo	0.1981	0.0124	0.0004	0.0003

Note: Only subjects weighing >85 kg were enrolled in the 0.9 mg dose group, no body weight restrictions were implemented in other dose groups.
Data from MRI-PDFF analysis dataset, defined as all randomized subjects who have MRI-PDFF measurements available at both baseline and Week 12; median % change in placebo was +7.2%; *p<0.05 ***p<0.001.

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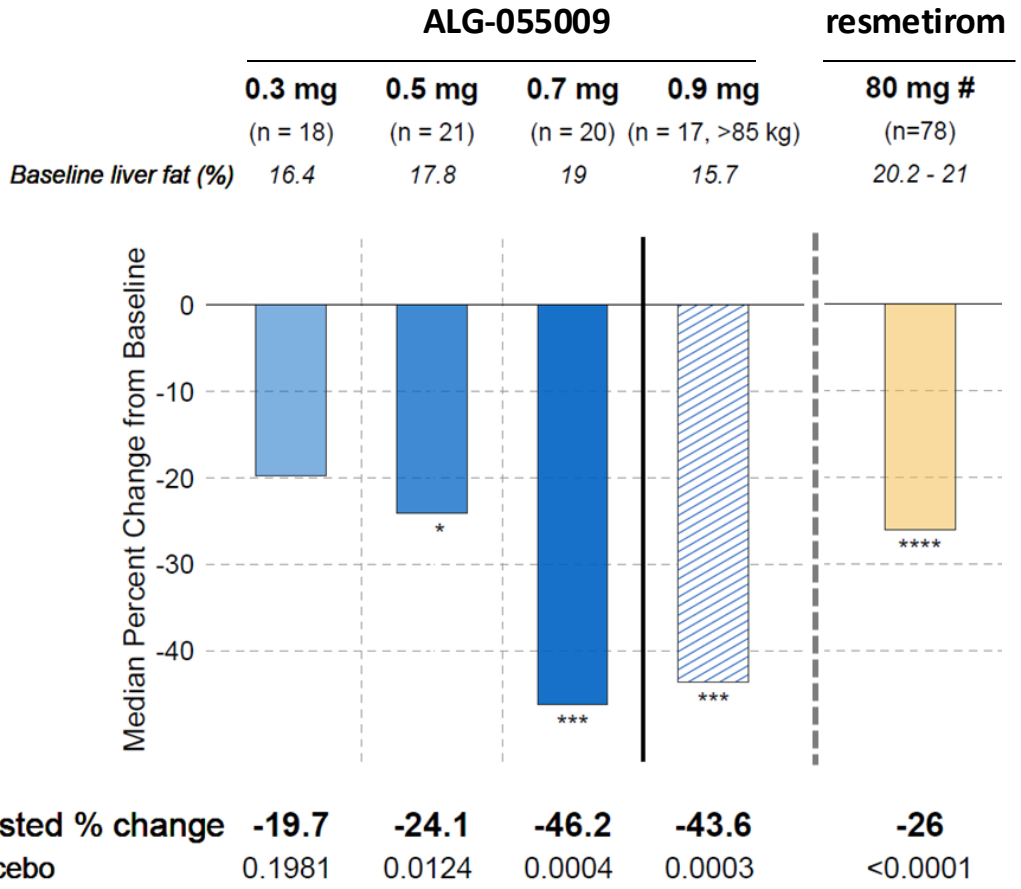
Significant decreases in liver fat, with up to 46% placebo-adjusted median reductions from baseline

ALG-055009 Demonstrated Significant Improvements in Liver Fat

Placebo-adjusted Median Relative Percent Change in Liver Fat at Week 12^



^Includes publicly reported data from the placebo-controlled Phase 2 trial of Resmetirom in a similar patient population conducted with different protocols at different sites and at different times from HERALD. No head-to-head clinical trials have been conducted. Resmetirom data: Figure 2 of Harrison et al. Lancet 2019: 2012-24. There are differences in study protocols, conditions, patient populations and reporting standards. Caution should be exercised when comparing data across trials. HERALD: only subjects weighing >85 kg were enrolled in the 0.9 mg dose group; no body weight restrictions were implemented in other dose groups. # ± 20 mg possible dose adjustment at Week 4. *p<0.05 ***p<0.001 ****p<0.0001.

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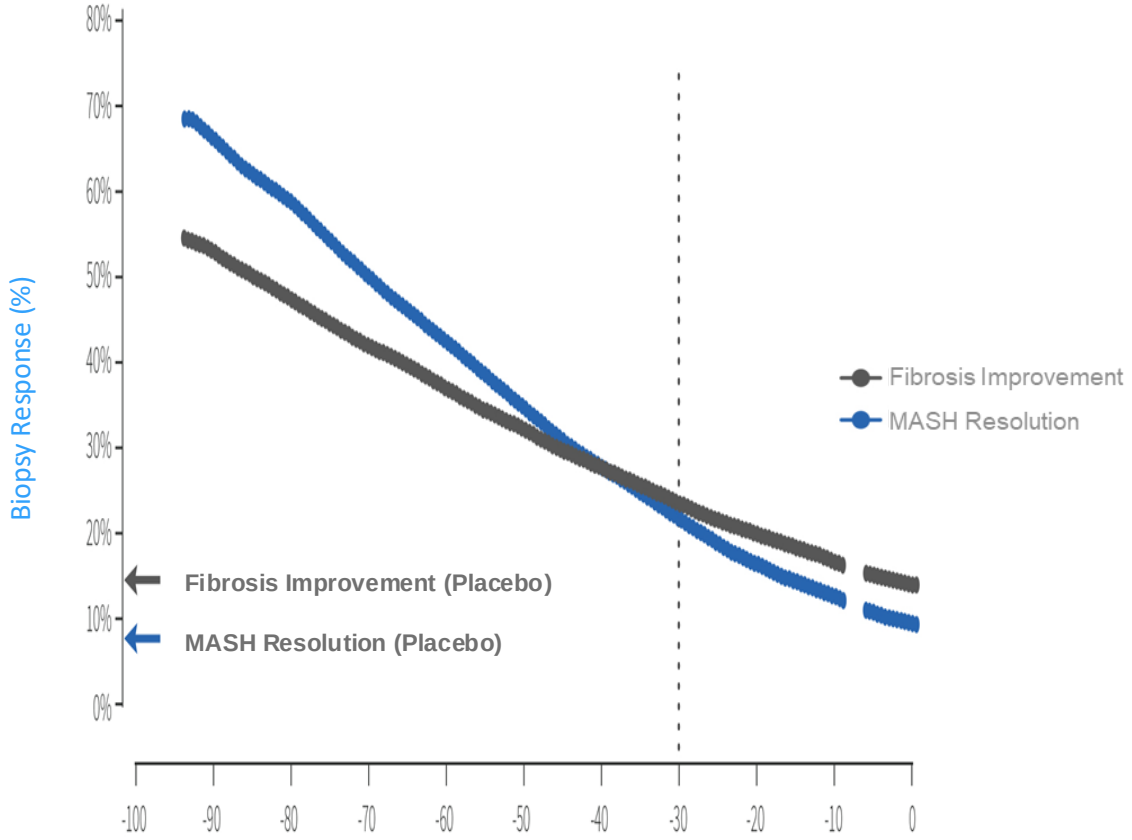
ALG-055009 demonstrated robust improvements in liver fat content compared to data from the study evaluating the only approved MASH treatment

Resmetirom Phase 3 Data

MRI-PDFF and Liver Biopsy Correlation

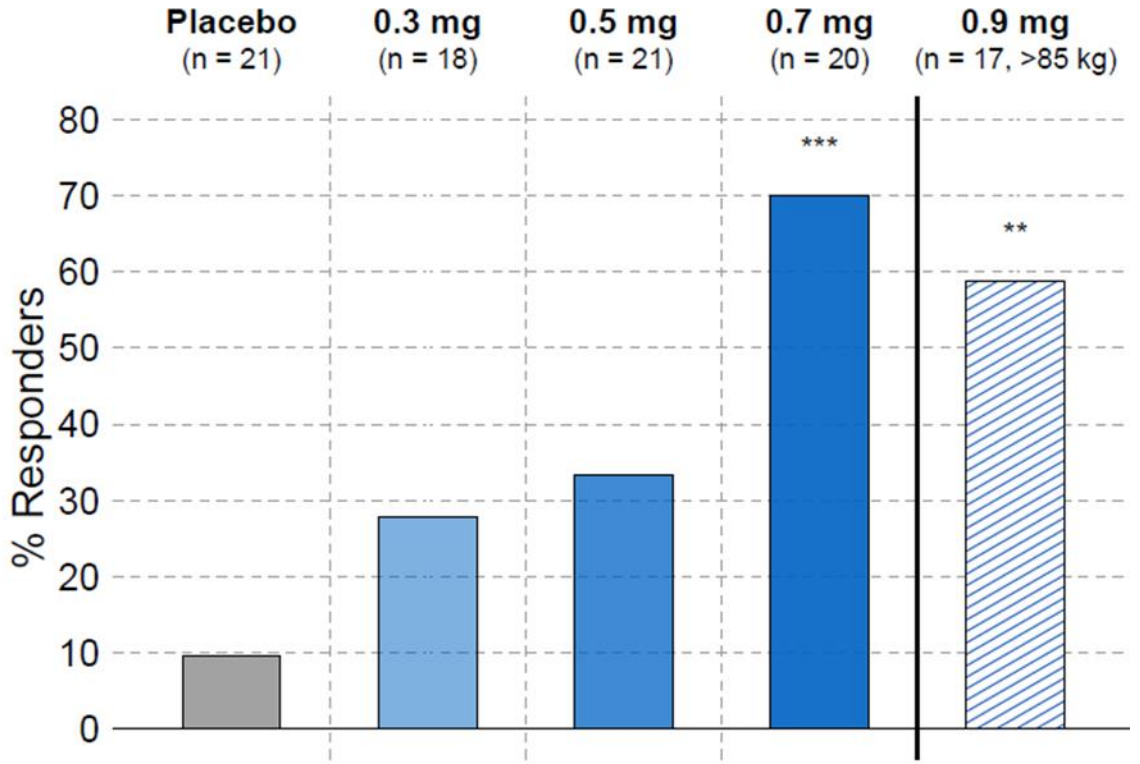


NR = Non-Responder.
Image adapted from Loomba et al, AASLD 2023.

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

HERALD: Significant MRI-PDFF Response Rates at Week 12

Up to 70% of Patients Achieved ≥30% Relative Reduction in Liver Fat



	Placebo (n = 21)	0.3 mg (n = 18)	0.5 mg (n = 21)	0.7 mg (n = 20)	0.9 mg (n = 17, >85 kg)
Responders (%)	9.5	27.8	33.3	70.0	58.8
p-value vs. Placebo		0.1603	0.0842	0.0004	0.0073

1. Loomba et al. Hepatology (2021). **p<0.01 ***p<0.001.
Only subjects weighing >85 kg were enrolled in the 0.9 mg dose group; no body weight restrictions were implemented in other dose groups.

MRI-PDFF response of ≥30% relative reduction in liver fat is predictive of histologic improvements[1]

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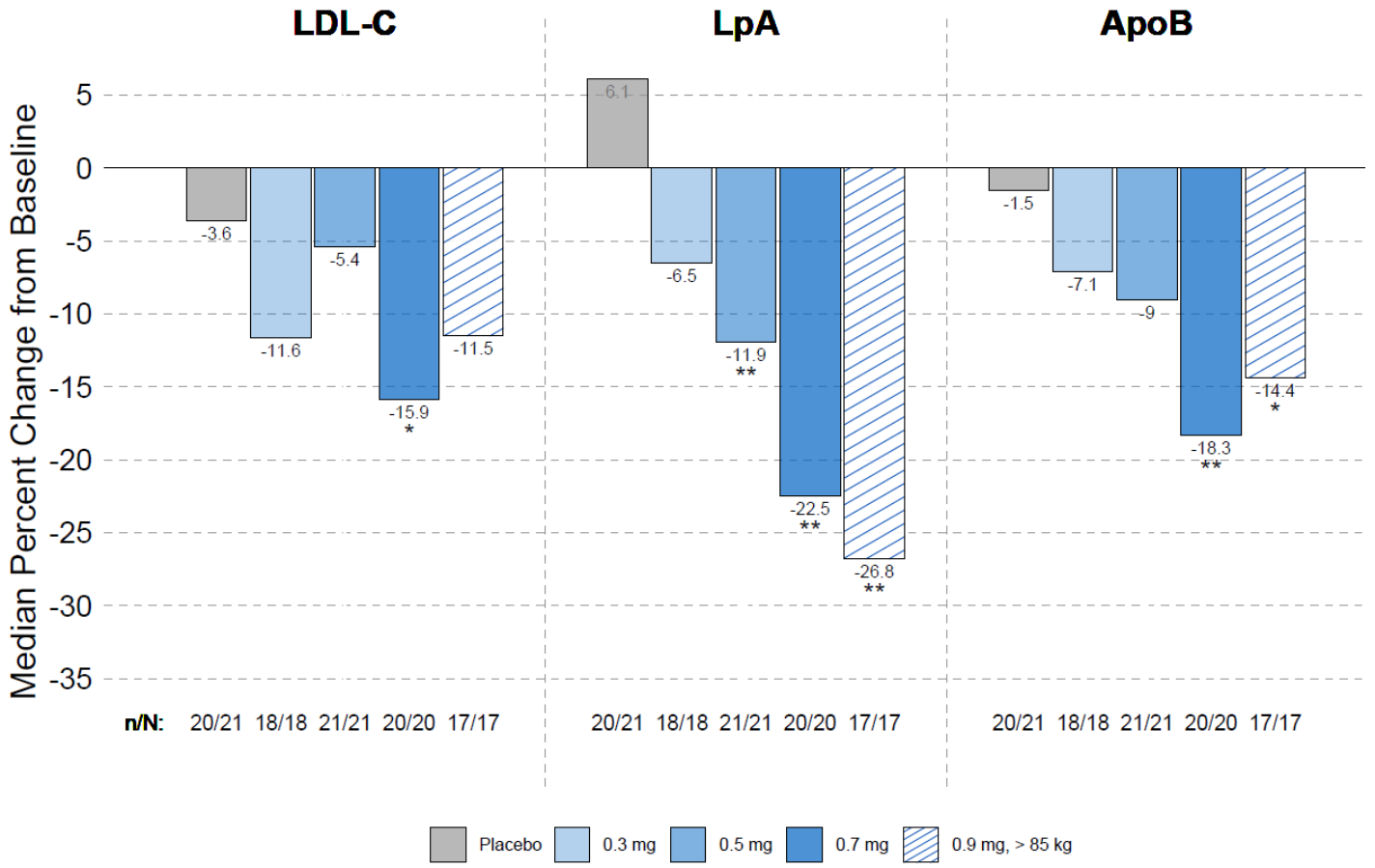
ALG-055009 Demonstrated Improvements in Lipid/Lipoproteins

Median Percent Change from Baseline at Week 12



LDL-C = low density lipoprotein cholesterol; LpA = lipoprotein (a); ApoB = apolipoprotein B; n: number of subjects with available data at week 12; N: number of subjects in MRI-PDFF analysis set; *p<0.05 **p<0.01. Only subjects weighing >85 kg were enrolled in the 0.9 mg dose group; no body weight restrictions were implemented in other dose groups.

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Significant reductions in LDL-C, Lipoprotein (a), and apolipoprotein B

HERALD Topline Data: Favorable Tolerability Profile

- Generally well tolerated, with no dose reductions
- No serious adverse events
- One discontinuation due to a treatment emergent adverse event (TEAE) of worsening insomnia in a subject with pre-existing insomnia
- Majority of the TEAEs (98%) were mild or moderate
- Less diarrhea noted for active dose groups compared to placebo, with no dose-response
- No evidence of clinical hypo/hyperthyroidism
- No clinically meaningful findings in laboratory tests, electrocardiograms, vital signs, or physical examinations were observed

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HERALD Phase 2a Study

ALG-055009 Continues to Demonstrate the Potential to be the Best-in-class THR-β Agonist

- **Primary endpoint achieved, with robust reductions in liver fat content at Week 12**
  - Up to 46% placebo-adjusted median relative reductions
  - Up to 70% of patients with ≥30% decrease in liver fat
- **Significant reductions in atherogenic lipids, including LDL-C, lipoprotein (a) and apolipoprotein B**
- **Dose-dependent increases in SHBG (marker of THR-β activation)**
- **Well-tolerated, with rates of GI-related AEs similar to placebo**
  - No serious AEs and 1 study drug discontinuation (1/102 or 1% of patients)
  - Majority of TEAEs (98%) mild or moderate
  - Less diarrhea noted for active dose groups compared to placebo, with no dose-response
- **ALG-055009 warrants further development**
  - Phase 2b enabling activities underway; expected completion middle of 2025
  - Assessing potential Phase 2b clinical trial study designs with KOLs, and plan to consult with the FDA
  - Early discussions with partners underway; evaluating a variety of options to fund continued development

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ALG-055009 demonstrated robust reductions in liver fat with a favorable tolerability profile, excellent PK and convenient once daily oral dosing

CHRONIC HEPATITIS B

ALG-000184: Small molecule CAM-E

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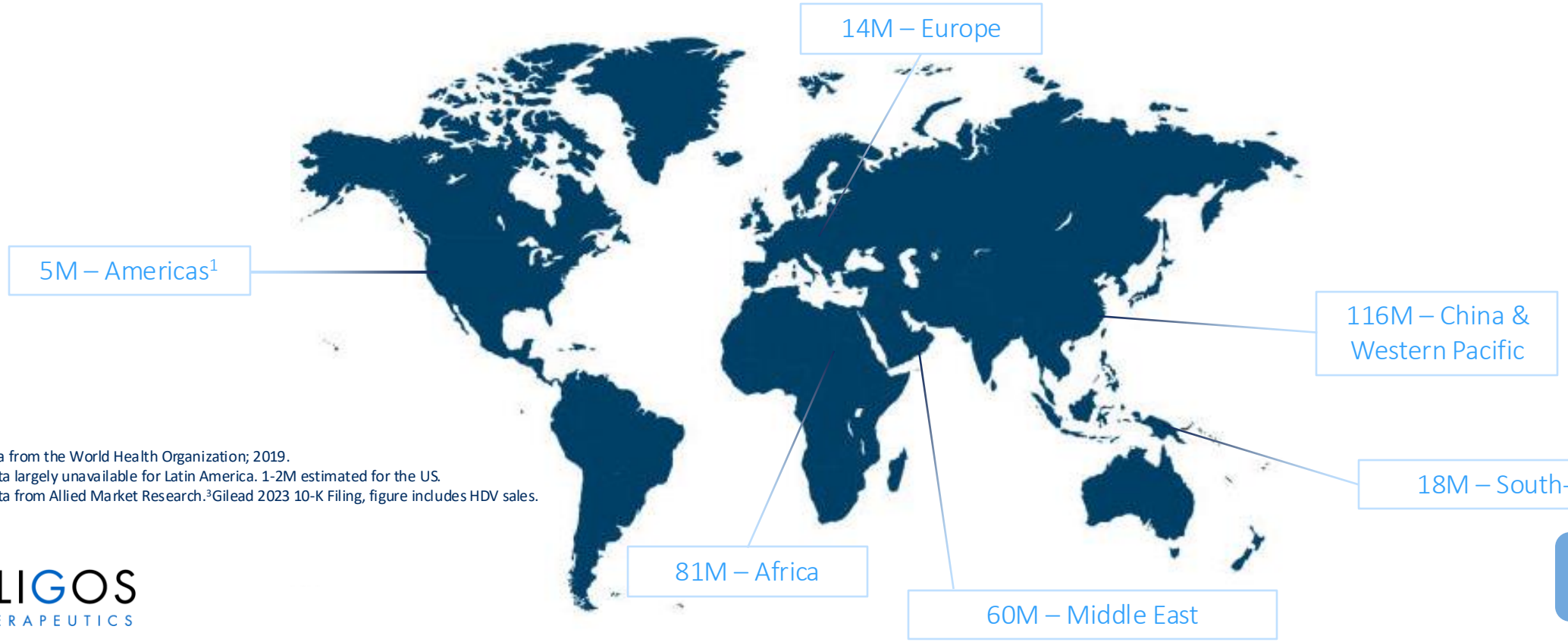
CHB

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]



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.

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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 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 a 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 associated with 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 patients with CHB (chronic suppressive therapy)

We have a high potency CAM that leads to greater DNA suppression and clinical demonstration of the secondary mechanism of action of CAMs

The importance of all relevant HBV 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 reductions in HBV RNA, HBcrAg, and HBeAg which result in complete suppression of viral replication

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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

ALG-000184

Small molecule CAM-E

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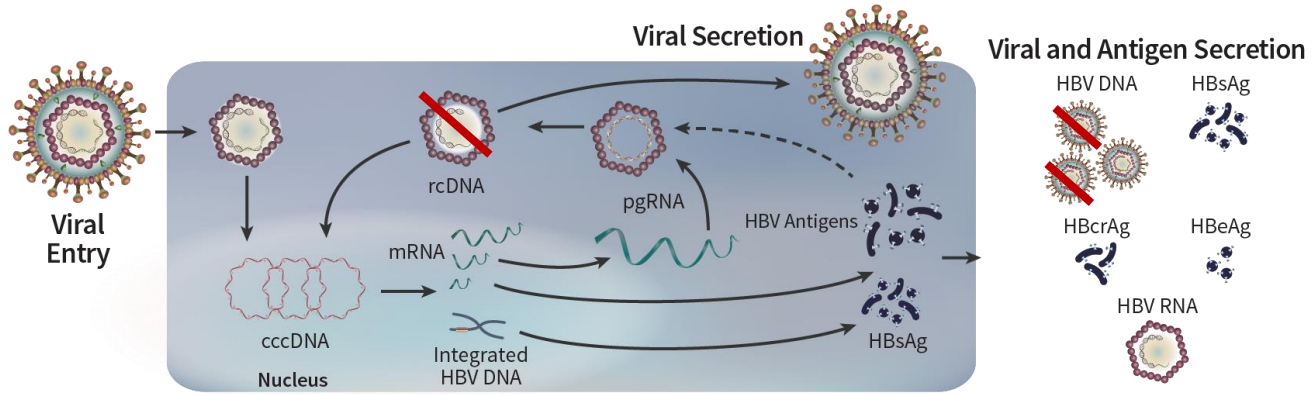
MOA: ALG-000184 (CAM-E) vs. Nucleos(t)ide Analogs

Suppressing the Entire HBV Lifecycle

Key Attributes

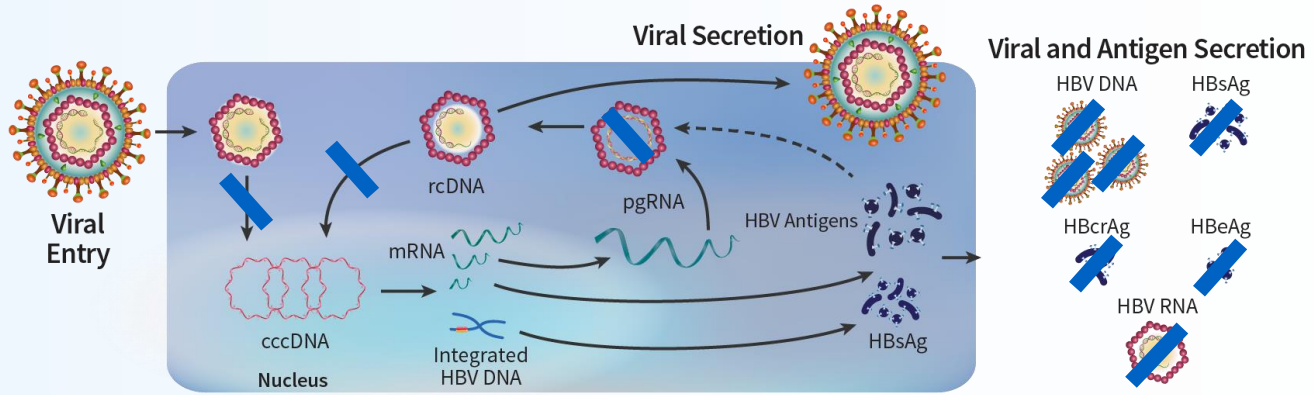
Nucleos(t)ide Analogs



Blocks the conversion of pgRNA to rcDNA

- Reduces viral secretion

ALG-000184



1st MOA

- Causes the formation of empty capsids
  - Reduces viral secretion (HBV DNA/RNA)

2nd MOA

- Prevents capsid disassembly
  - Prevents establishment/replenishment of cccDNA, which produces HBcrAg, HBeAg, and (some) HBsAg

Leads to significant reduction of viral and antigen secretion

ALG-000184 has the potential to improve clinical outcomes

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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 ≤72 weeks in untreated CHB)
  - PK: dose proportional, low-moderate variability
  - Safety: no safety signals observed
  - Antiviral activity: potential best-in-class reductions demonstrated in HBsAg, HBeAg, HBcrAg, HBV DNA & RNA
  - Dosing x ≤96 weeks ongoing (through 2025)
- **Phase 1b combination study**
  - Phase 1b exploratory combination study with Amoytop (mipeginterferon alfa-2b)
- **Phase 2**
  - Clear regulatory path forward with the FDA and China's National Medical Products Administration for chronic suppressive therapy with superiority label compared to standard of care
  - Enabling activities underway; planned submission of Phase 2 protocol under the recently cleared IND in Q1 2025

[1]Not including any patent term extension.

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We envision ALG-000184 as the backbone of next-generation HBV treatment

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 HVs
- Part 2 (MAD): 7 QD oral doses (150 and 250 mg) in HVs
- Part 3: QD oral doses (10-300 mg) x 28 days in treatment naïve (TN)/currently not treated (CNT) CHB subjects

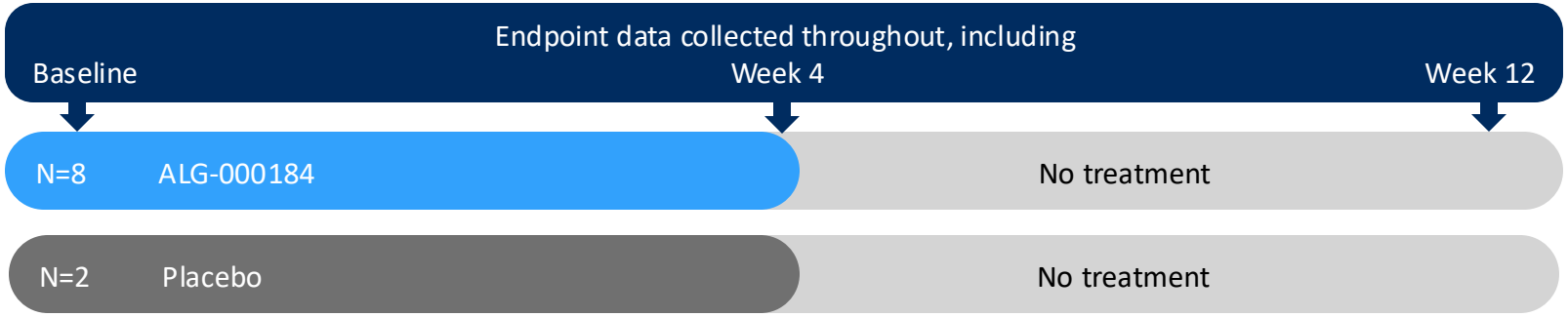
Multiple Doses in TN/CNTCHB Subjects

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)



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.

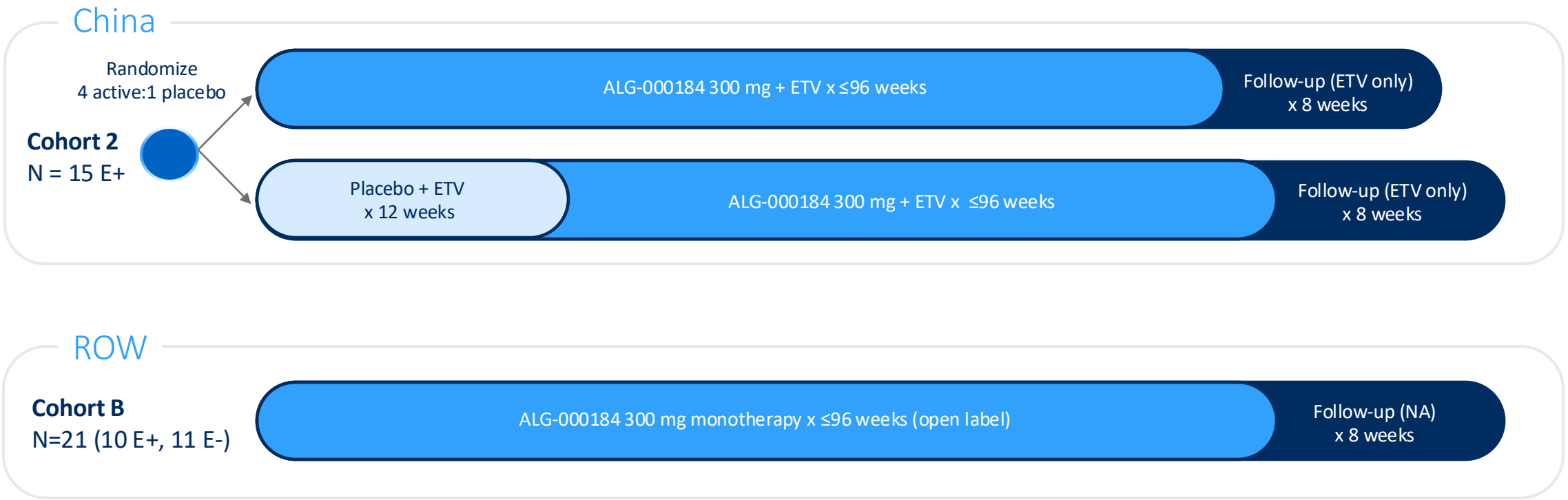
Part 1 & 2: Single oral dose ≤500 mg and multiple oral daily doses ≤250 mg x 7 days well tolerated with linear PK in HV

Part 3: multiple daily doses ≤300 mg well tolerated with linear PK and excellent antiviral activity (HBV DNA, RNA, HBsAg)

ALIGOS THERAPEUTICS

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

Part 4 Cohort Designs

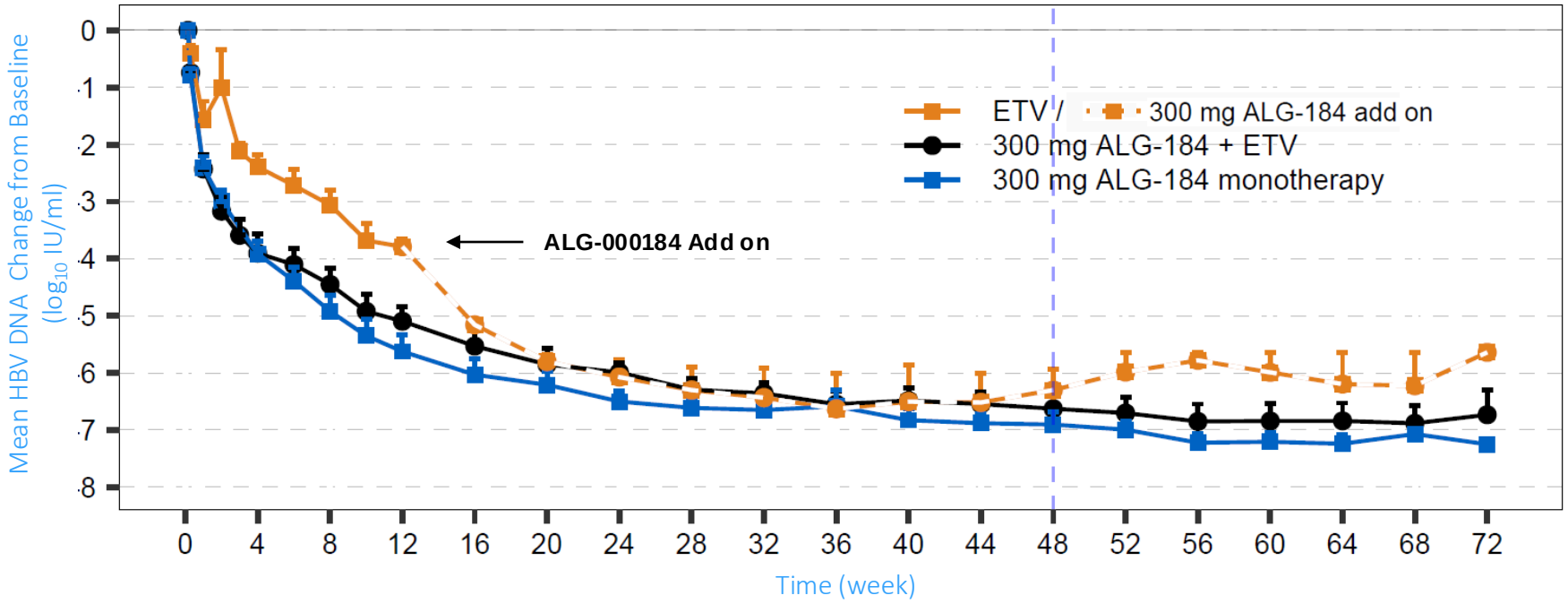


Hou, JL. et al., Poster 1483-C, AASLD (2023).
Yuen, M-F. et al., Late Breaker Poster #5028-C, AASLD (2023).
All cohorts fully enrolled. NCT04536337; ROW:rest of the world.

ALIGOS THERAPEUTICS

Antiviral Effect in CHB Subjects (HBeAg+)

Mean 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

Aligos oral, The Science of HBV Cure Meeting 2024.

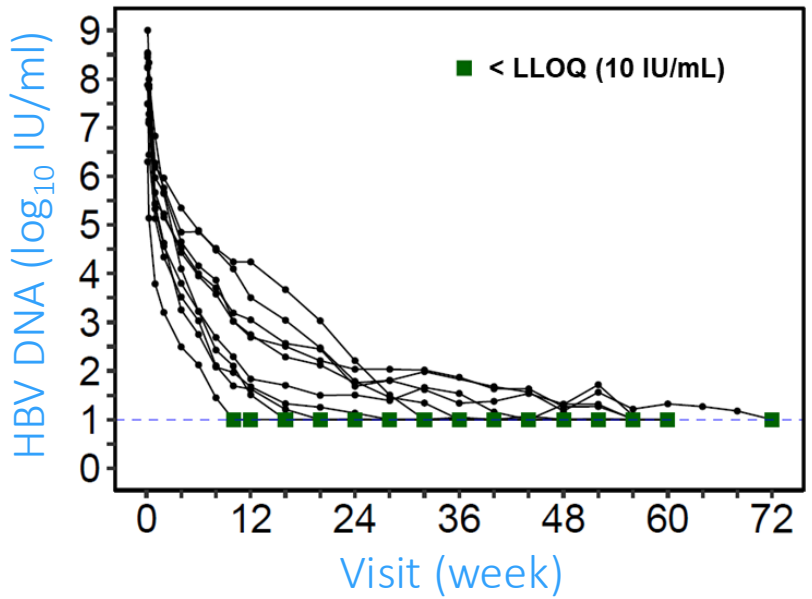
ALIGOS THERAPEUTICS

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

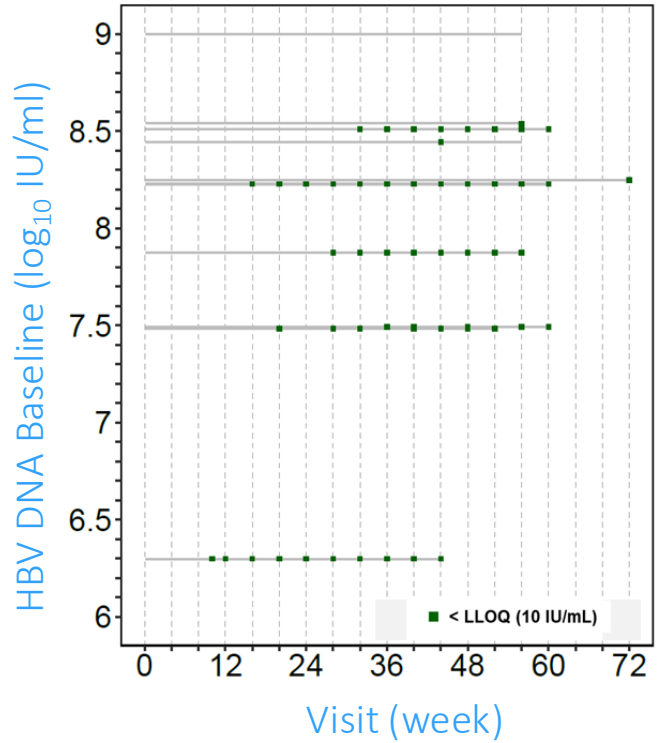
300mg ALG-000184 Monotherapy (HBeAg+)

Individual HBV DNA Decline

300 mg monotherapy ≤ 72 weeks



Week	0	4	8	12	16	20	24	28	32	36	40	44	48	52	56	60	64	68	72
Total n	10	10	10	10	10	10	10	10	10	9	10	10	9	9	8	4	1	1	1
< LLOQ [10 IU/mL]	0	0	0	1	2	3	2	4	5	5	6	6	5	4	5	3	0	0	1



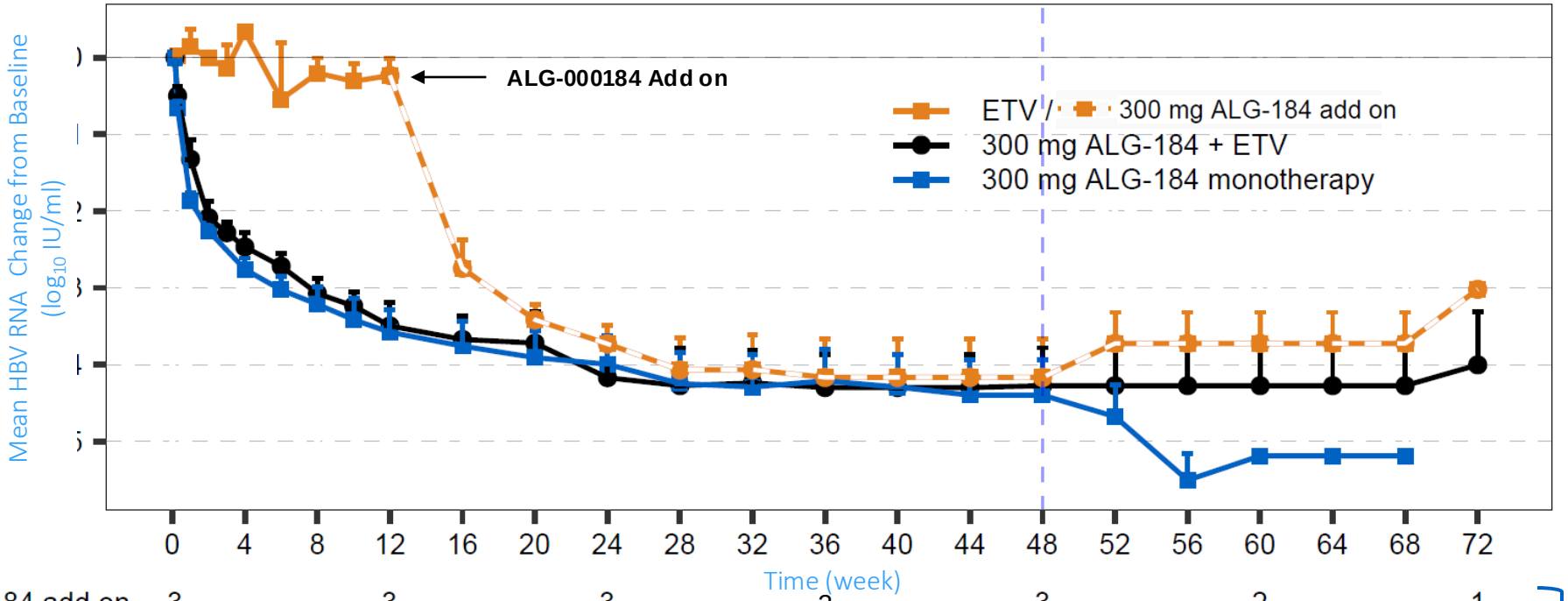
Yuen, M-F. et al; EASL 2024.

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

ALIGOS THERAPEUTICS

Antiviral Effect in CHB Subjects (HBeAg+)

Mean HBV RNA 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	9	1	

Number of subjects at each timepoint

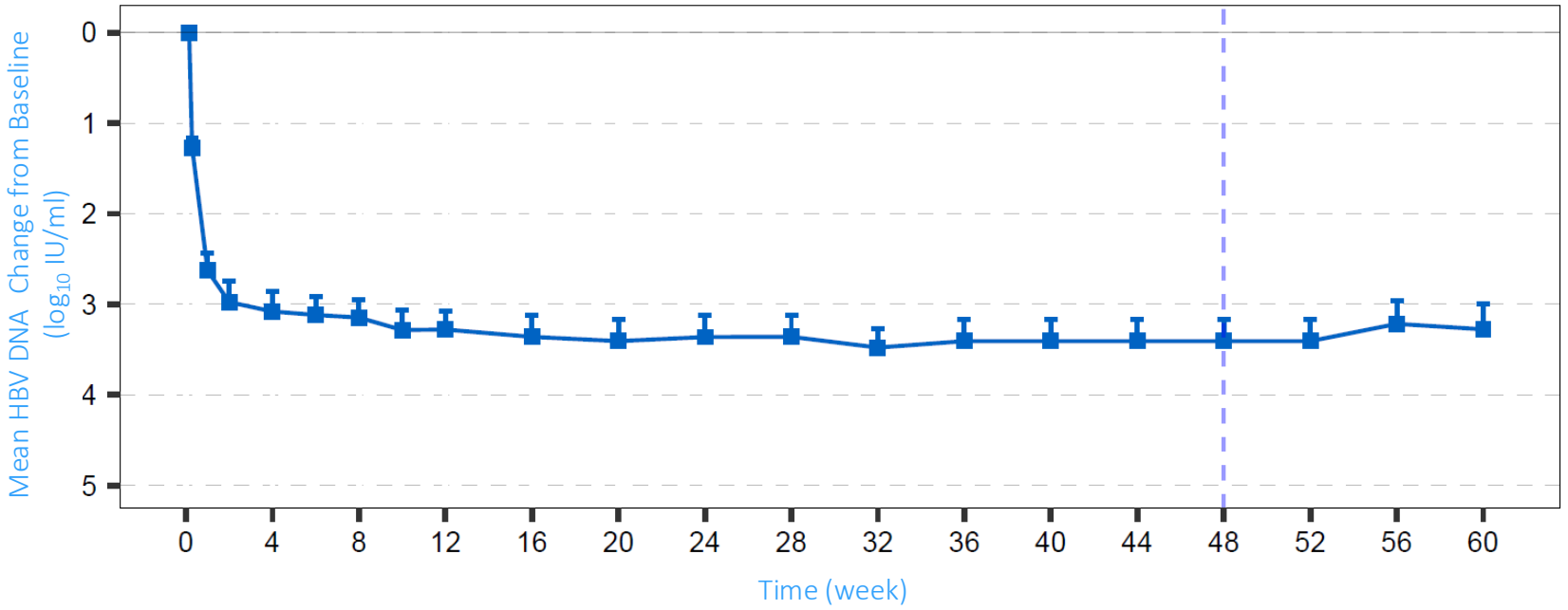
Aligos oral, The Science of HBV Cure Meeting 2024. ^Liu et al., J Infect Dis, 2022. Mak et al., J Gastroenterology, 2021. Ding et al. Hepatology, 2021.

At Week 12, there was a >3 log10 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

Antiviral Effect in CHB Subjects (HBeAg-)

Mean 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

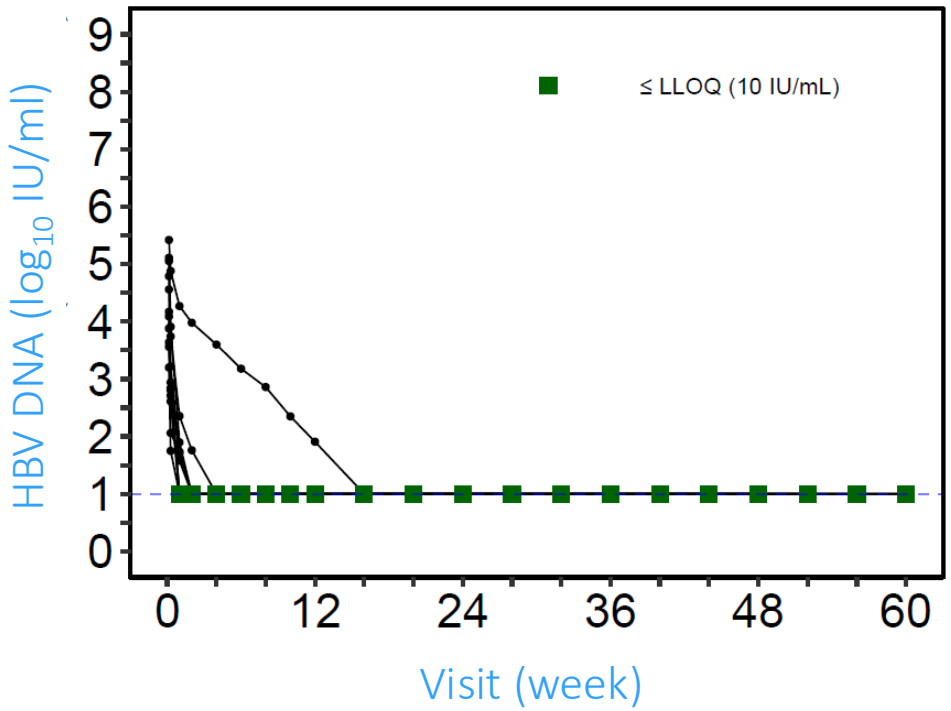
Data on file.

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

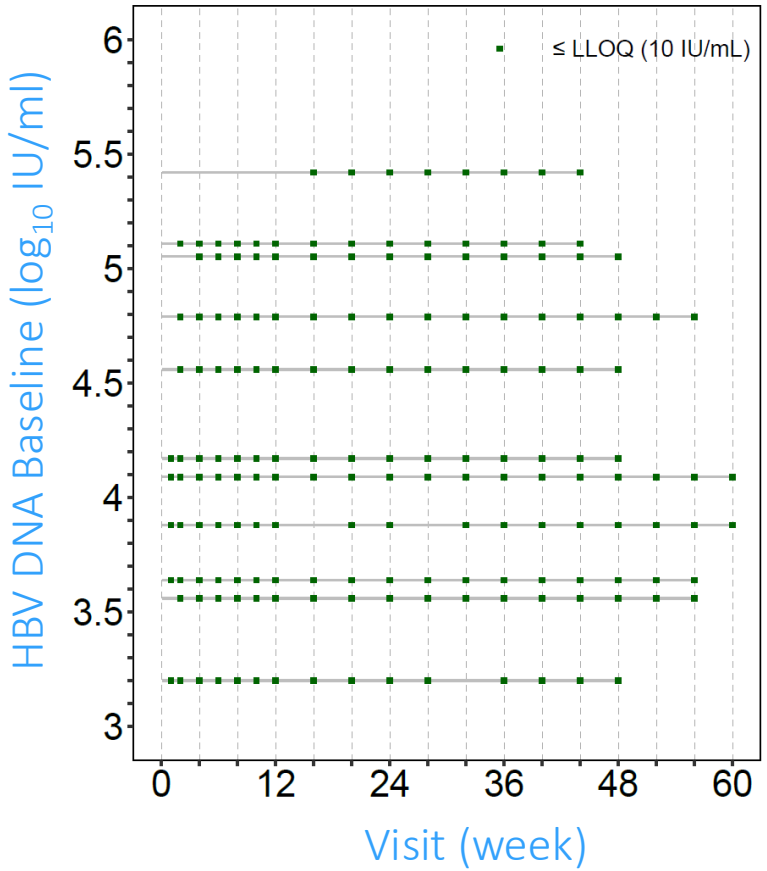
ALIGOS THERAPEUTICS

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



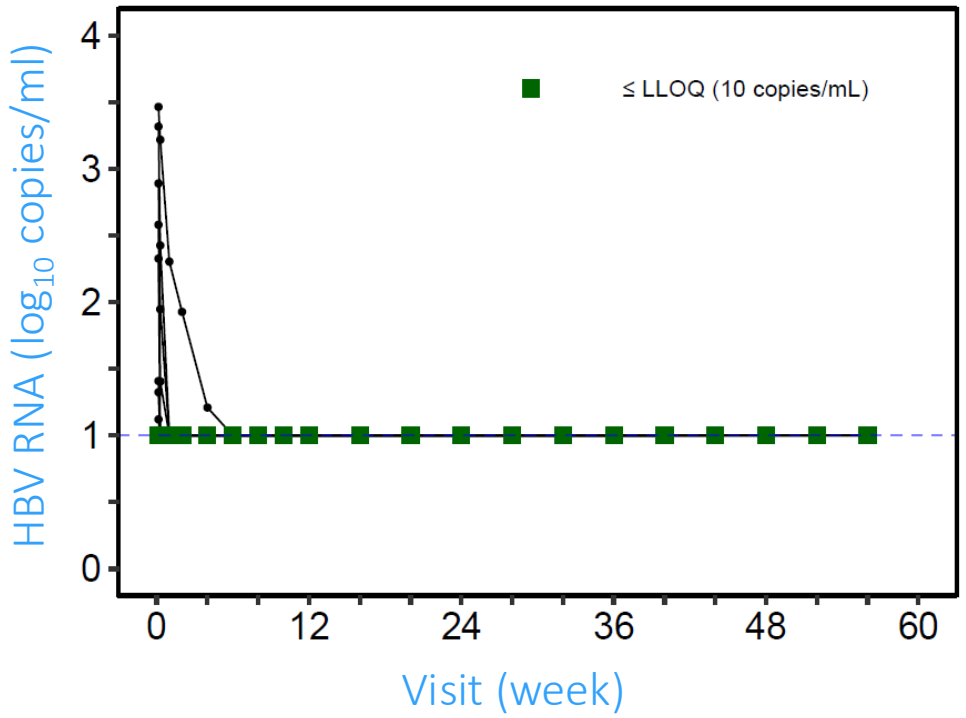
Agarwal, K. et al. EASL 2024.

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

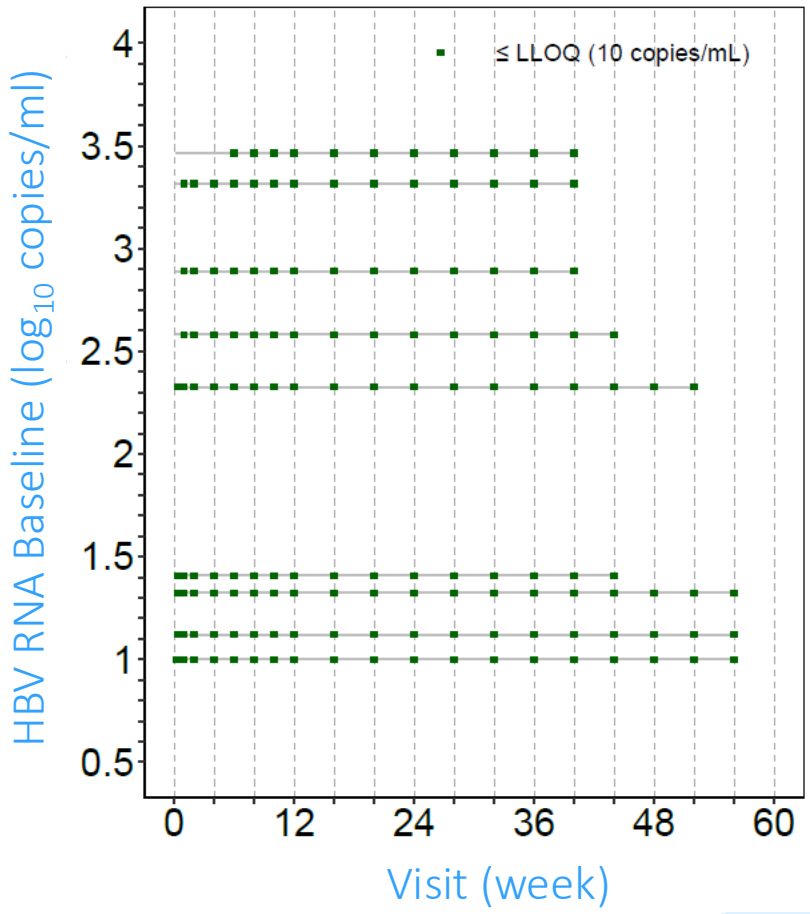
ALIGOS THERAPEUTICS

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



Agarwal, K. et al. EASL 2024.
^Liu et al., J Infect Dis, 2022. Mak et al., J Gastroenterology, 2021. Ding et al. Hepatology, 2021.

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

ALG-000184

Chronic DNA Suppression vs. 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%	31%
	TAF (n=285)[a]	94%	21%	90%	33%
	300 mg ALG-000184 (n=11)[d]	11/11 (100%)	11/11 (100%)	TBD	TBD
E+	TDF (n=292)[b]	67%	N/A	75%	9%
	TAF (n=581)[b]	64%	N/A	73%	14%[e]
	300 mg ALG-000184 (n=10)[c]	10/10 (100%)	6/10 (60%)	TBD	TBD

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. [e] Kosh A. et al.; Journal of Hepatology 2018 V68: 672-681.

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

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Chronic Suppression

Well Defined, Validated Approval Pathway

- Regulatory pathway for chronic suppressive therapy endorsed by FDA, EMEA, and National Medical Products Administration in China
- Primary endpoint: Subjects with HBV DNA <LLOQ (10 IU/mL) 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.

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

Aligos has received FDA and National Medical Products Administration in China feedback supporting subsequent studies utilizing this pathway

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 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^

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

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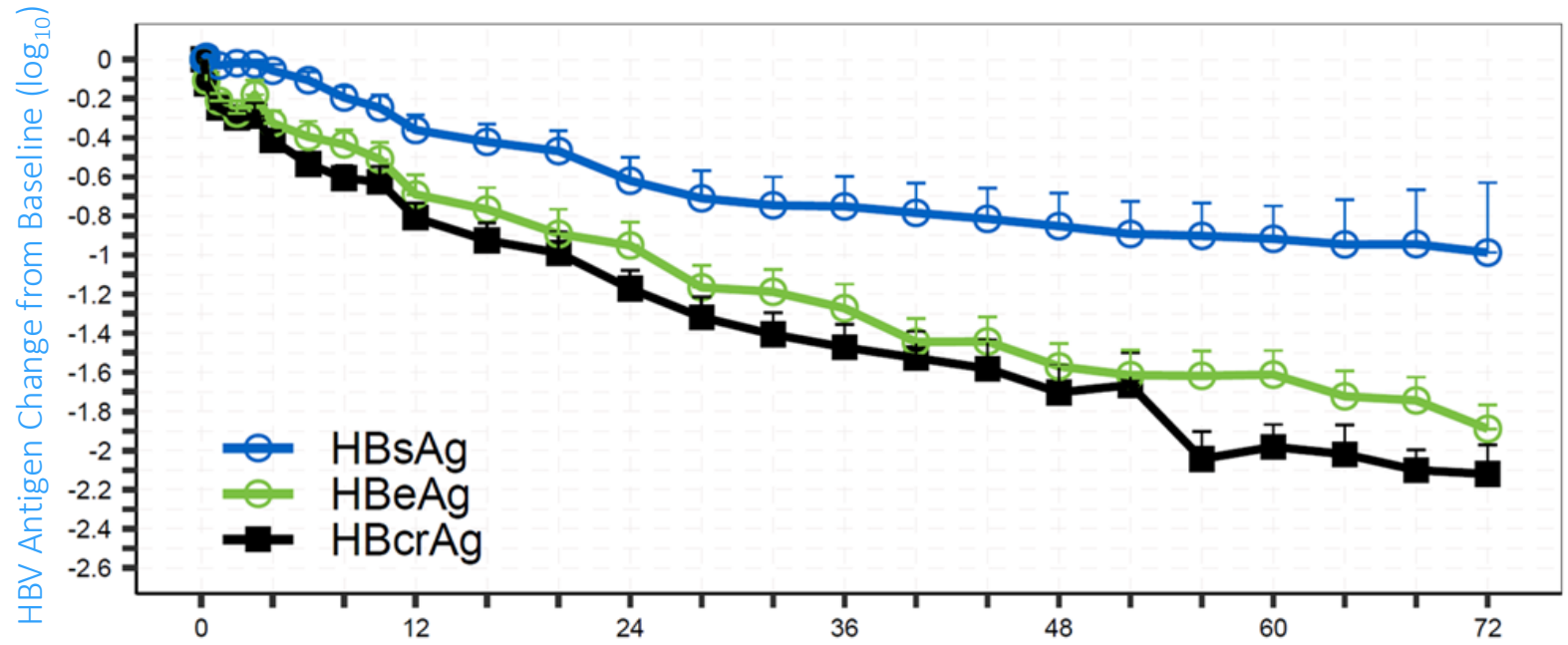
In addition to superior DNA reductions, multiple clinically meaningful secondary efficacy endpoints may be achieved

ALG-000184-201 - Antiviral Effect in HBeAg+ CHB Subjects

Mean 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

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

Continued substantial HBsAg, HBeAg, and HBcrAg reductions noted with combo through week 72
Mean max declines: 1.0, 1.9 and 2.1 log10 IU/mL, respectively

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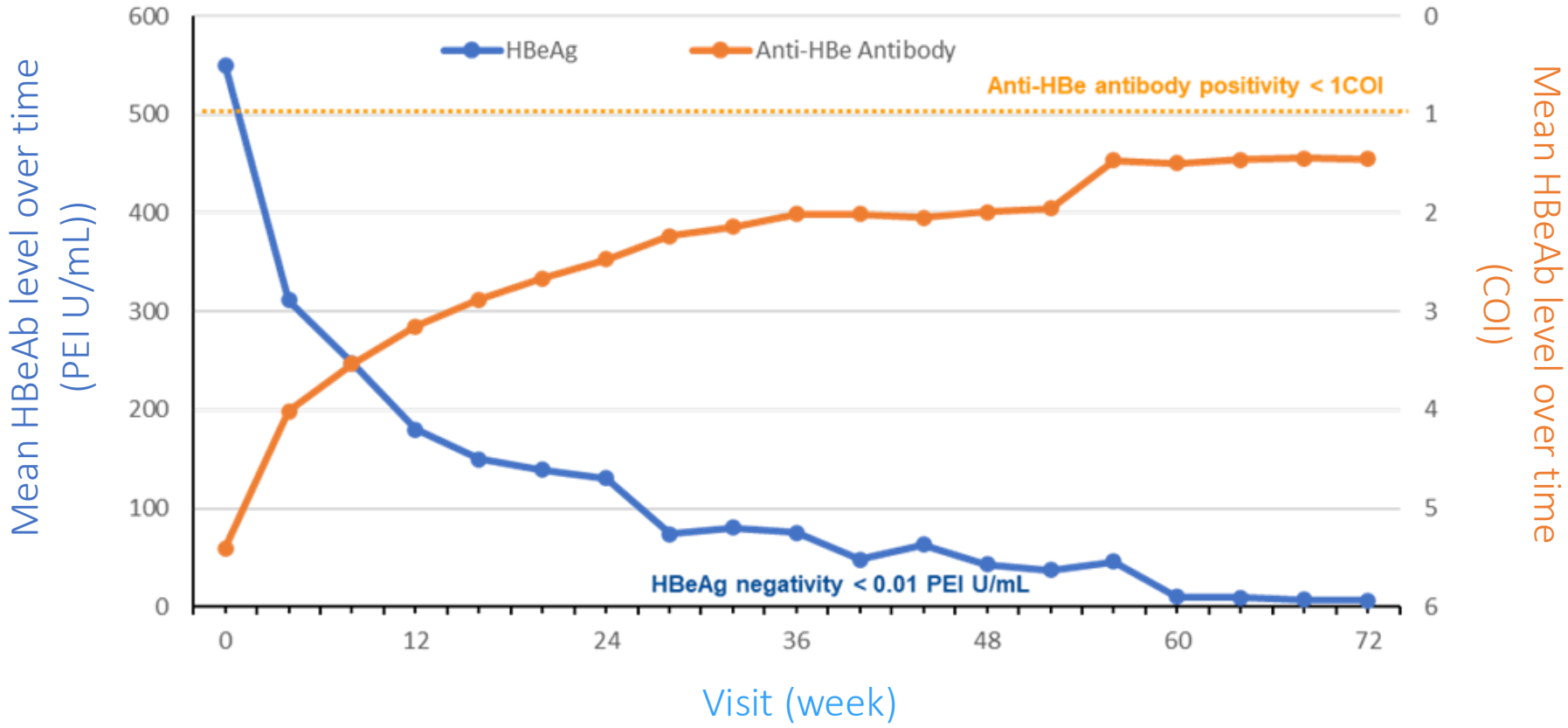
300 mg ALG-000184±ETV

Mean HBeAg and Anti-HBe Antibody Level Over time



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

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

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↑ and neutropenia↑ (n=1) ALT/AST↑ and Uric acid ↑ (n=1) ALT/AST↑ (n=1) eGFR↓ (n=1)	3 ALT/AST↑ (n=3)	2 ALT/AST↑ (n=1) Cholesterol/Triglycerides ↑ (n=1)
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

As of June 2024.

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

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

ALG-055009 for MASH

- ✓ Positive Phase 2a HERALD topline safety and MRI-PDFF data readout ahead of schedule in Q3 2024
- Phase 2b enabling activities underway; expected completion middle of 2025
- Assessing potential Phase 2b clinical trial study designs with KOLs, and plan to consult with the FDA
- Early discussions with partners underway; evaluating a variety of options to fund continued development

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 compared to standard of care with the FDA & National Medical Products Administration in China
- Additional interim data readouts expected at AASLD
- Expect to receive approval from the National Medical Products Administration in China to begin Phase 1b exploratory combination study with Amoytop (mipeginterferon alfa-2b)
- Phase 2 enabling activities ongoing; planned submission of Phase 2 protocol under the recently cleared IND in Q1 2025

ALG-097558 for Pan-Coronavirus

- ✓ Phase 1 FIH topline data presented
- ✓ Phase 2 enabling activities (externally funded) ongoing
- ALG-097558 is expected to begin three clinical trials in Q4 2024
  - AGILE University of Liverpool, a UK government supported platform trial (MRC and Wellcome Trust funding), will sponsor and perform a study in high-risk COVID patients evaluating ALG-097558 as monotherapy or in combination with remdesivir
  - The NIAID will sponsor clinical studies to evaluate pharmacokinetic (PK) differences in special populations (renal/hepatic impairment subjects)

Strong Cash Position: As of 9/30/24: Cash, cash equivalents and investments were $74.9M
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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