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CORPORATE PRESENTATION

December 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 November 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 Development Portfolio

2025: An Execution Year

Candidate	Indication	MOA	Discovery	Nonclinical	Phase 1	Phase 2	Phase 3	Partner
ALG-000184	CHB Monotherapy	CAM-E						
ALG-000184	CHB Combination[1]	CAM-E + PEGBING® (mipeginterferon alfa-2b)						AMOYTOP BIOTECH
ALG-055009	MASH	THR-β Agonist						
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.

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

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CHRONIC HEPATITIS B INFECTION

OVERVIEW

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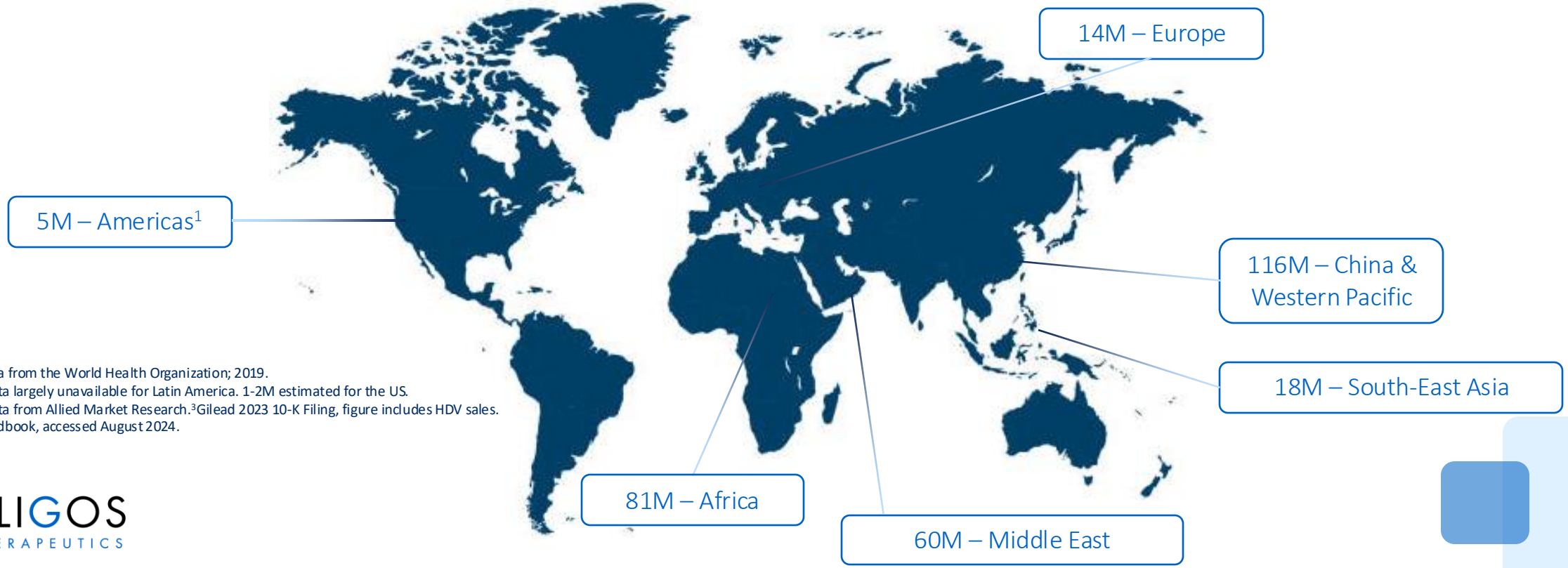
CHB

High Unmet Medical Need

- ~296M people worldwide living with CHB with 1.5M new infections
- 820k deaths per year, mostly from cirrhosis and hepatocellular carcinoma; Primary cause of liver cancer worldwide
- Opportunity estimated at $6.2B by 2031[2]; Gilead HBV sales of $1B in 2023[3] at ~$17k/year[4] for continuous therapy
- ALG-000184 has the potential for favorable pricing and payor coverage due to its potential to improve patient outcomes



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.
[4]Redbook, accessed August 2024.

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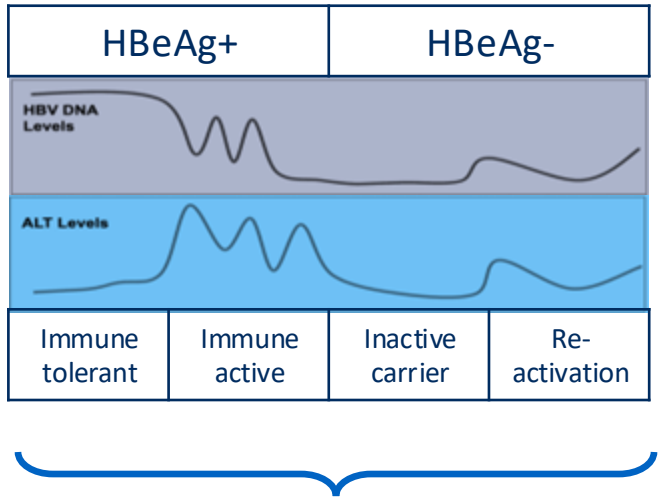
Therapeutic Goals of HBV Antiviral Drugs

- **Patient Journey:** The primary treatment goal is to reduce HBV DNA to undetectable levels, normalize liver enzymes, prevent liver damage, and reduce the risk of developing HCC
- WHO Guideline 2024 recommends treatment if significant fibrosis, or DNA>2,000 IU/ml + ALT>ULN, or presence of special risk factors

Nomenclature		HBeAg+ Infection	HBeAg+ Disease	HBeAg- Infection	HBeAg- Disease
Other terms		Immune tolerant	Immune (re)active	Inactive carrier state	Immune-active or HBeAg- disease
Serology	HBsAg	Positive	Positive	Positive	Positive
	Quantitative HBsAg (log_{10} IU/mL)	3.5 - 4.5	3.5 - 4.5	2.5 - 3.5	2 - 3
	HBeAg	Positive	Positive	Negative	Negative
	Anti-Hbe	Negative	Negative	Positive	Positive
	HBV DNA (IU/mL)	Typically > 10^7	Typically > 10^5 to 10^7	< 10^3	Typically > 10^3 to 10^5
Biochemistry	ALT	Around ULN	Raised	Around ULN	Raised



All patients at risk for progression to liver disease

Table adapted from WHO Guideline 2024, Figure adapted from Kim et.al, Hepatitis B online.

Current Treatment Options

Nucleoside/Nucleotide analogs (NAs) & Pegylated Interferon alfa (IFNα)

NAs

- Standard of care for 25+ years
  - Indicated for chronic suppression
    - HBV DNA <LLOQ at Week 48 following treatment
  - **Inadequate:** In a recent study with NAs dosed over 5 years, patients continued to progress[2]
    - 4% of patients developed HCC
    - 5% suffered from liver decompensation (variceal bleeding, hepatorenal syndrome, hepatic encephalopathy, ascites)
    - 1% underwent liver transplantation or died

PEG-IFNα

- Not widely used due to tolerability profile
  - Indicated for functional cure
    - HBsAg < LLOQ ~6 months after a finite treatment regimen
  - **Inadequate:** A 1-year course of PEG-IFNα results in an overall seroclearance rate of 2-3% at the end of treatment[1]

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[1]Wong et. al., J Hepatology 2021. [2] Kim et. al., AASLD 2024.

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 than SOC 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

First potential new mechanism advancing towards approval for chronic suppression in CHB in 25+ years

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ALG-000184

Small Molecule CAM-E

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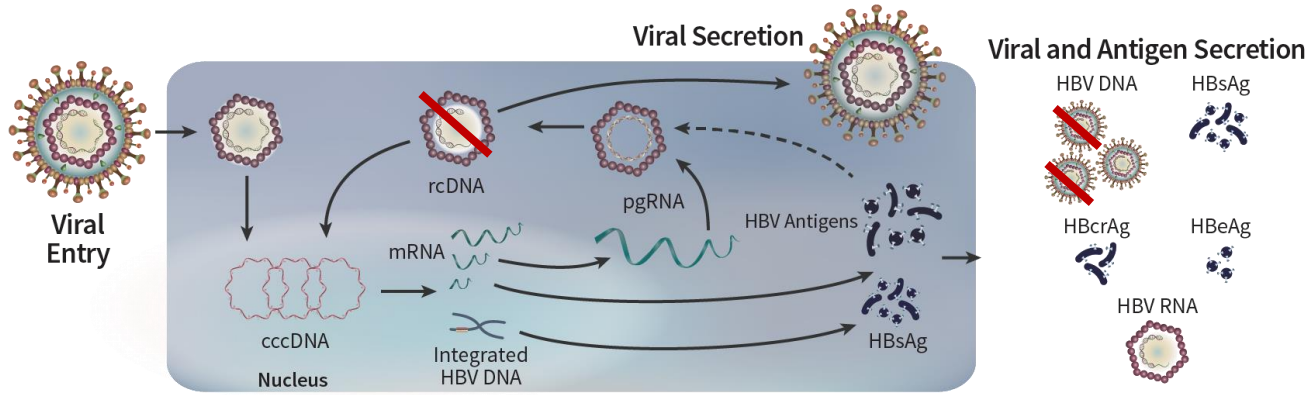
MOA: ALG-000184 (CAM-E) vs. Nucleos(t)ide Analogs Suppressing the Entire HBV Lifecycle

Key Attributes

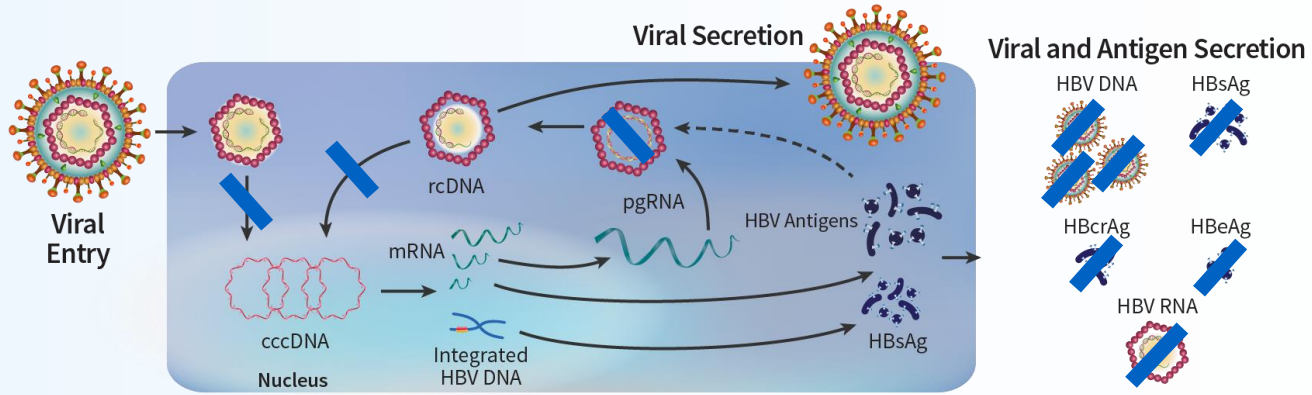
Nucleos(t)ide Analogs



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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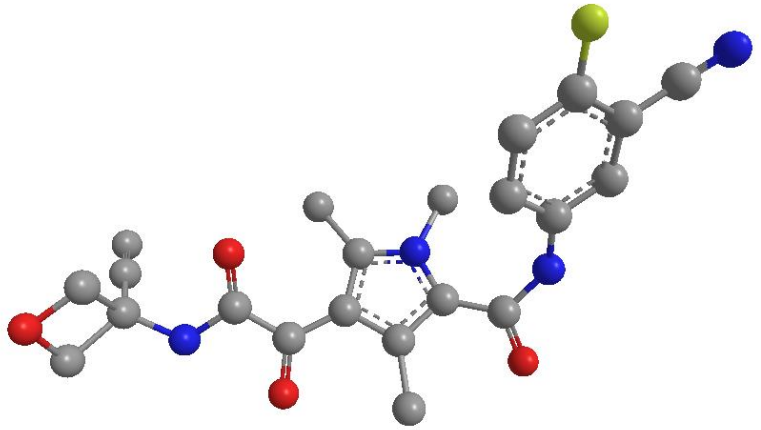
Our Vision

Paving the Way for the Future of CHB Treatment

ALG-000184
has the potential to…



- Become standard of care
- Act as the backbone of next-generation therapies
- Treat all HBV patients
- Affect the entire HBV lifecycle
- Lower HBsAg, allowing more patients to be eligible for curative therapies

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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 potency with enhanced absorption and high liver uptake
- **Preclinical profile**
  - ~2-300-fold improvement in in vitro potency vs. other known CAMS; superior DMPK properties
- **Phase 1 highlights** (SAD/MAD + QD oral doses (10-300 mg) x 28 days in treatment naïve/currently not treated subjects)
  - **PK:** Dose proportional, low-moderate variability
  - **Safety:** All doses well tolerated
  - **Efficacy**: The lowest dose (10mg) achieved maximum HBV DNA reductions; more potent antiviral activity than competitor CAMs
  - **MOA:** Evidence of second mechanism seen at 300mg monotherapy dose through reductions in HBsAg
    - The only CAM to date that has demonstrated HBsAg reduction at 28 days
- **Phase 1b:** Dosing for 96 weeks ongoing through 2025
- **Phase 1b exploratory combination study with mipeginterferon alfa-2b (Amoytop) expected to begin in 2025**
- **Phase 2 enabling activities underway:** Planned submission of Phase 2 protocol to the FDA and globally in Q1 2025

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[1]Not including any patent term extension.

ALG-000184

- Phase 1b Study Design
- Phase 1b Clinical Data

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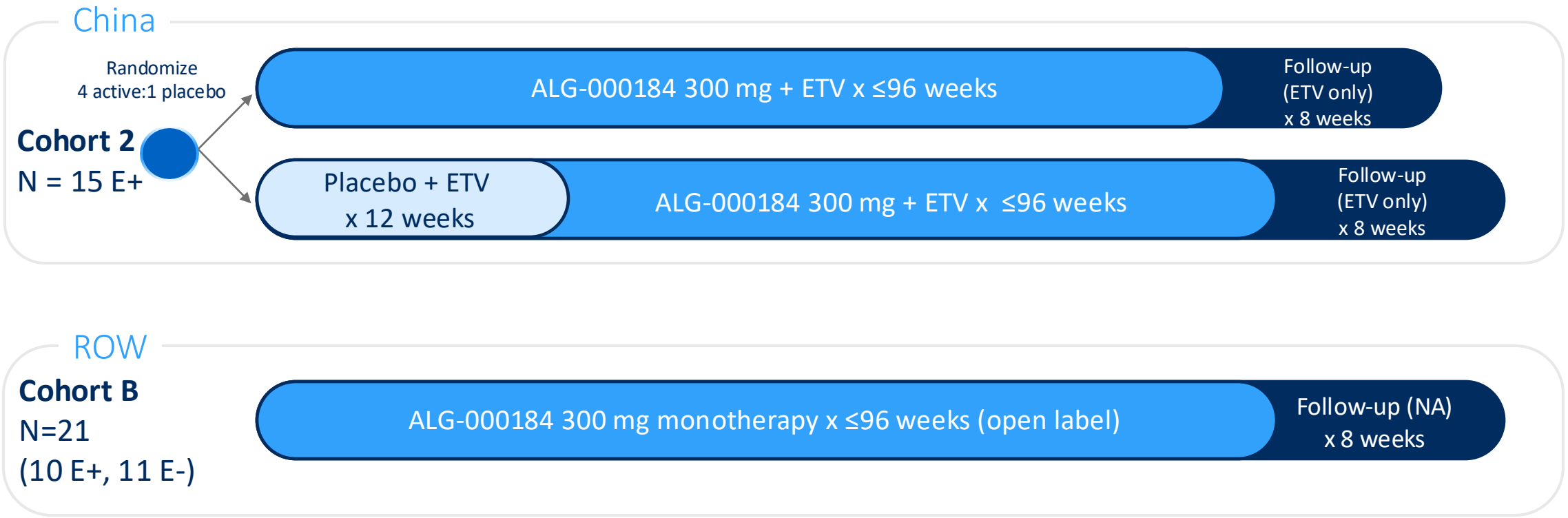
ALG-000184-201 – Long Term Dosing in CHB Subjects

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.

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ALG-000184-201 - Part 4 Cohort 2 & B

Baseline Characteristics

	Part 4 Cohort 2 (300 mg ALG-000184/Placebo + ETV)	Part 4 Cohort B (300mg ALG-000184 mono)	
N	HBeAg+ N=15	HBeAg + N=10	HBeAg – N=11
Age, years, mean (SEM)	31.4 (3.3)	34.8 (2.9)	48.5 (3.1)
Female, N (%)	7 (46.7)	3 (30.0)	5 (45.5)
Asian, N (%)	15(100)	9 (90.0)	3 (27.3)
BMI, kg/m^2, mean (SEM)	22.2 (0.8)	22.4 (0.8)	26.0 (1.1)
HBeAg positive, N	15	10	0
HBV Genotype B/C, N (%)	B: 5 (33), C: 10 (67)	B: 5 (50), C: 4(40), D: 1 (10)	B:2(18), C:1(9), D:7(64), A:1(9)
HBV DNA, log_{10} IU/mL, mean (SEM)*	8.1 (0.2)	8.0 (0.2)	4.3 (0.2)
HBV RNA, log_{10} copies/mL, mean (SEM)	6.7 (0.3)	5.3 (0.4)	2.0 (0.3)
HBsAg, log_{10} IU/mL, mean (SEM)	4.4 (0.2)	4.3 (0.1)	3.5 (0.2)
HBcrAg, log_{10} U/mL, mean (SEM)	8.3 (0.3)	8.3 (0.2)	3.2 (0.3)
HBeAg, log_{10} PEI U/mL, mean (SEM)	3.0 (0.1)	2.6 (0.3)	-
ALT, U/L, mean (SEM)	38.7 (5.2)	60.7 (11.7)	35.0 (4.4)

BMI= body mass index; SEM= standard error of the mean.

Yuen, M-F. et al; AASLD 2024. Hou, et. Al., AASLD 2023. BMI = body mass index; SEM= standard error of the mean.

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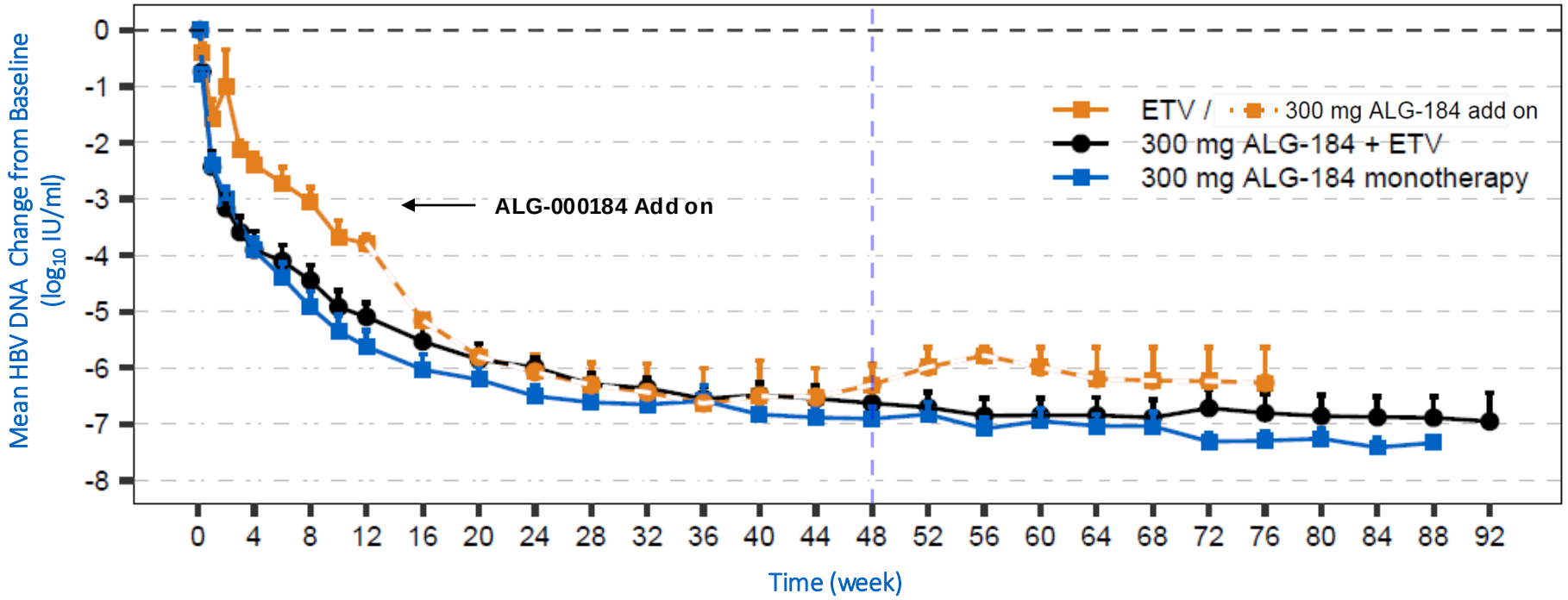
Antiviral Effect in CHB Subjects (HBeAg+)

Mean HBV DNA Change from Baseline



ETV / 300 mg ALG-184 add on	3	3	3	3	3	2	2		
300 mg ALG-184 + ETV	11	9	9	9	8	8	7	7	5
300 mg ALG-184 monotherapy	10	10	10	10	10	10	9	7	

Number of subjects at each timepoint

Aligos oral, The Science of HBV Cure Meeting 2024. Data on file.

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

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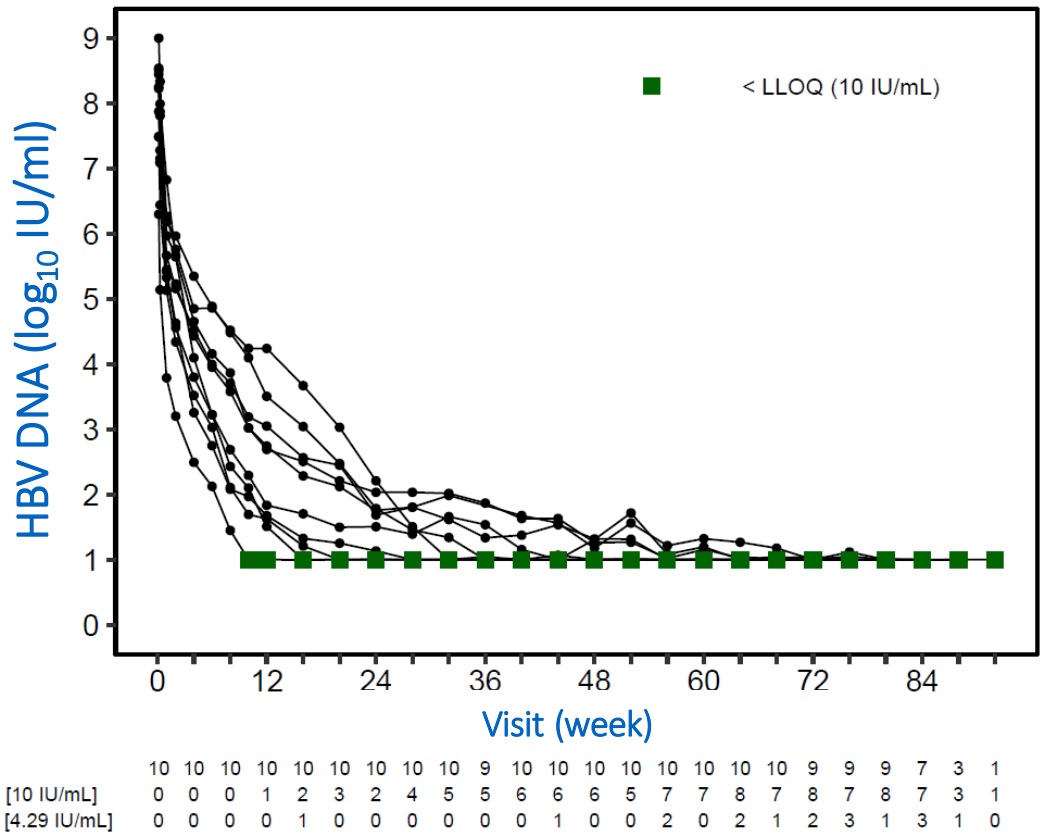
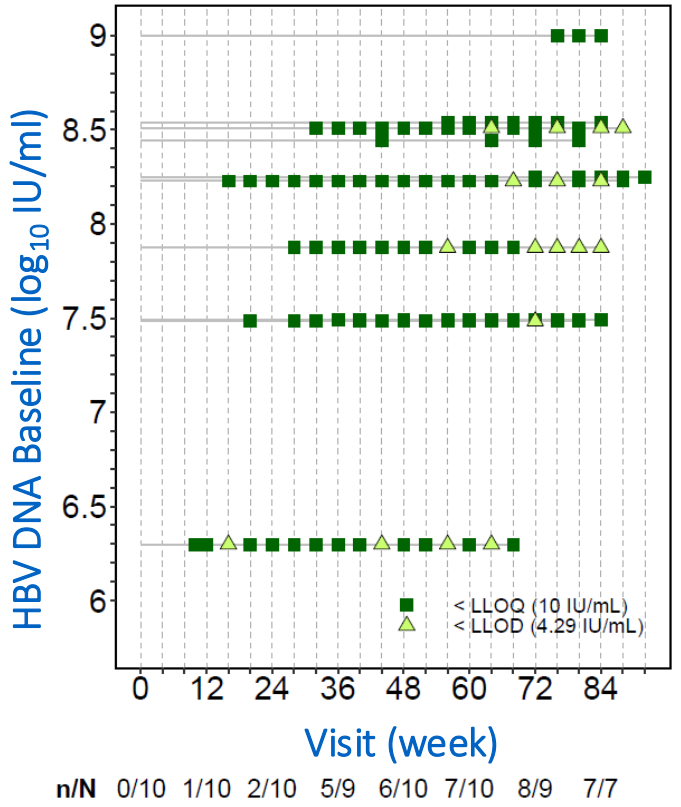
300mg ALG-000184 Monotherapy (HBeAg+)

Individual HBV DNA Level Over Time





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

No viral breakthrough during ALG-000184 monotherapy x ≤92 weeks
60% (6/10) of subjects achieved HBV DNA <10 IU/mL by week 48
and 100% (7/7) by week 84

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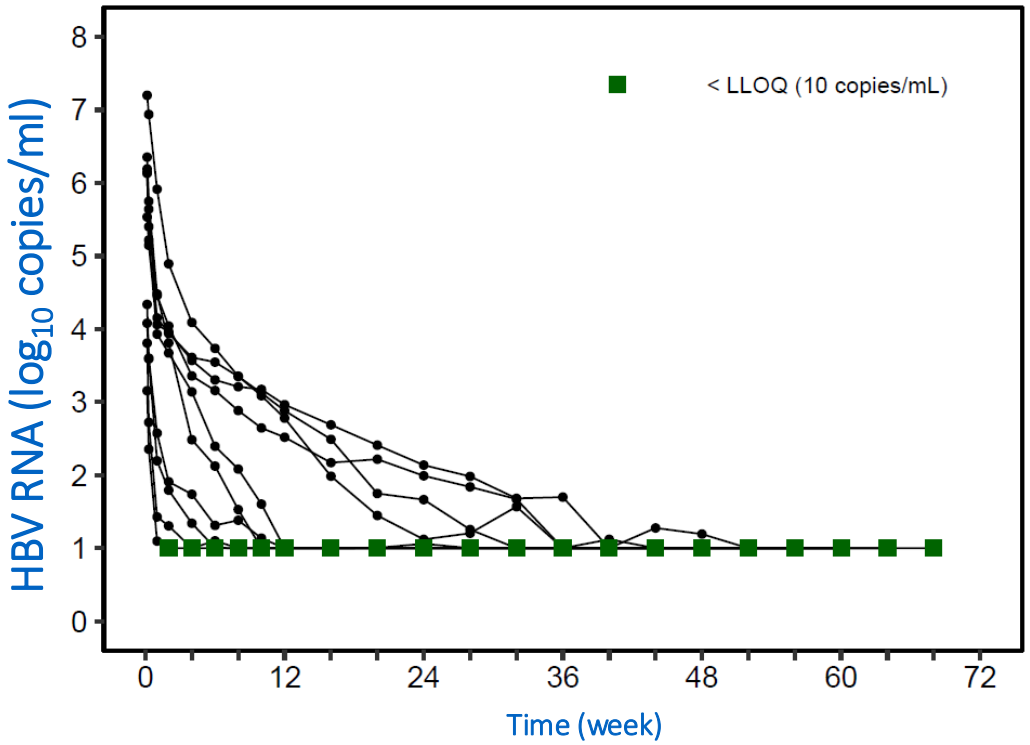
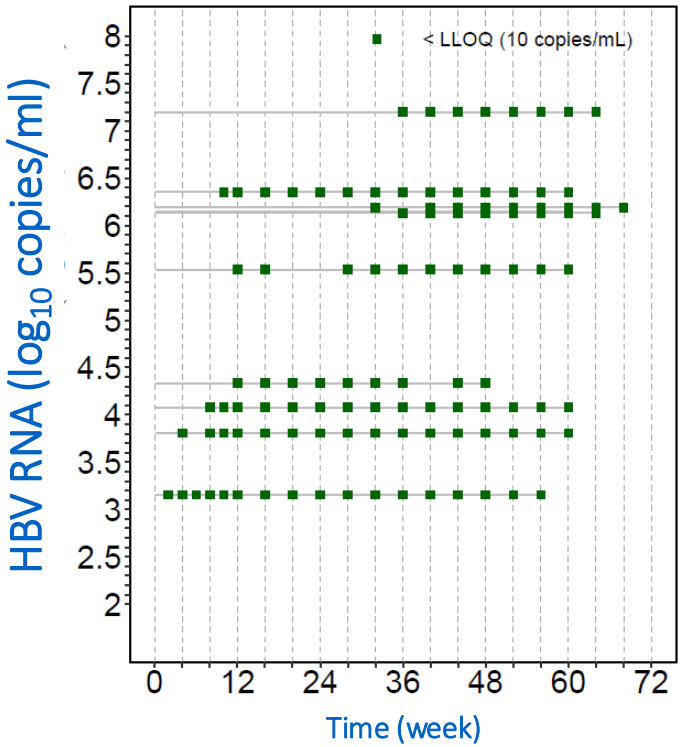
300mg ALG-000184 Monotherapy (HBeAg+)

Individual HBV RNA Level Over Time





Total n	10	10	10	10	10	10	10	10	10	9	10	10	10	9	9	8	3	1
< LLOQ [10 copies/mL]	0	2	3	6	6	5	5	6	7	8	9	9	9	9	9	8	3	1

n/N	0/10	6/10	5/10	8/9	9/10	8/8

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

100% (10/10) of subjects experienced HBV RNA <LLOQ by week 40
RNA levels correlated with HCC risk^

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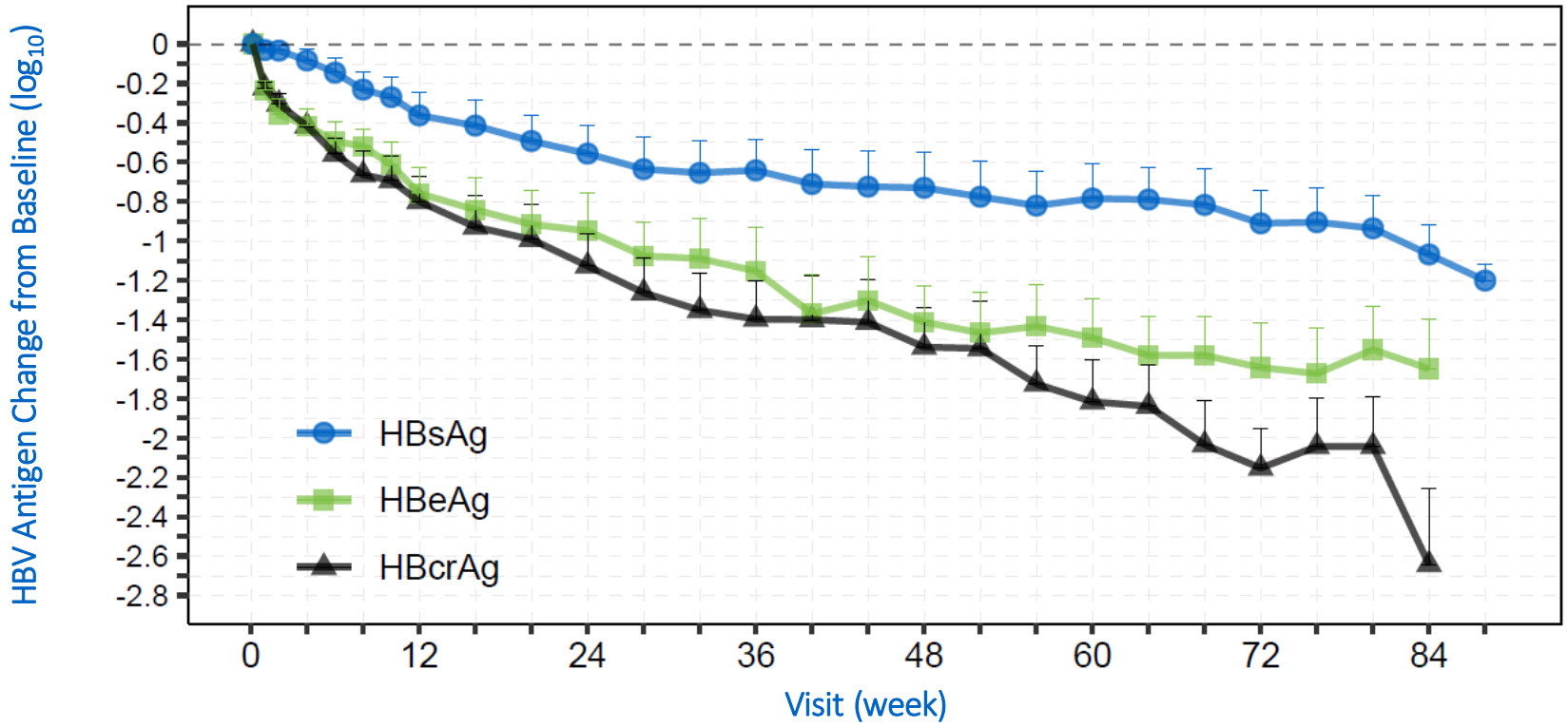
ALG-000184-201 - Antiviral Effect of 300 mg ALG-000184 Monotherapy (HBeAg+)

Mean HBV Antigen Change from Baseline



HBsAg	10	10	10	10	10	10	9	8	3
HBeAg	10	10	10	10	10	10	9	7	
HBcrAg	10	10	10	10	10	10	9	3	

Number of subjects at each timepoint

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

Continued substantial HBsAg, HBeAg, and HBcrAg reductions noted through week 84
Max individual declines: 1.5, 2.6 and 3.2 log10 IU/mL, respectively

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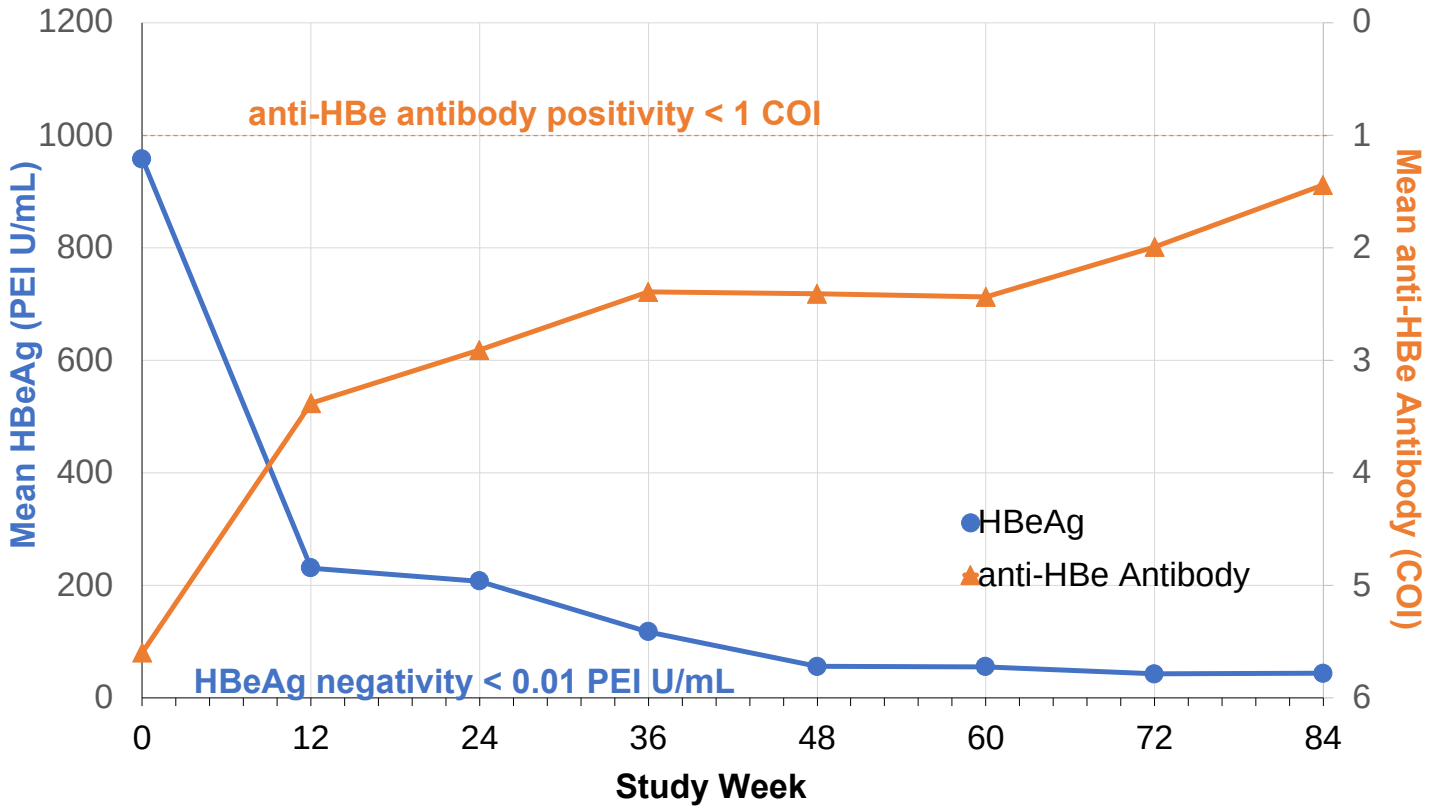
300 mg ALG-000184 Monotherapy (HBeAg+)

Mean HBeAg and Anti-HBe Antibody Level Over Time



HBeAg	10	10	10	10	10	10	9	7
HBeAb	10	10	10	10	10	10	9	2

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

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

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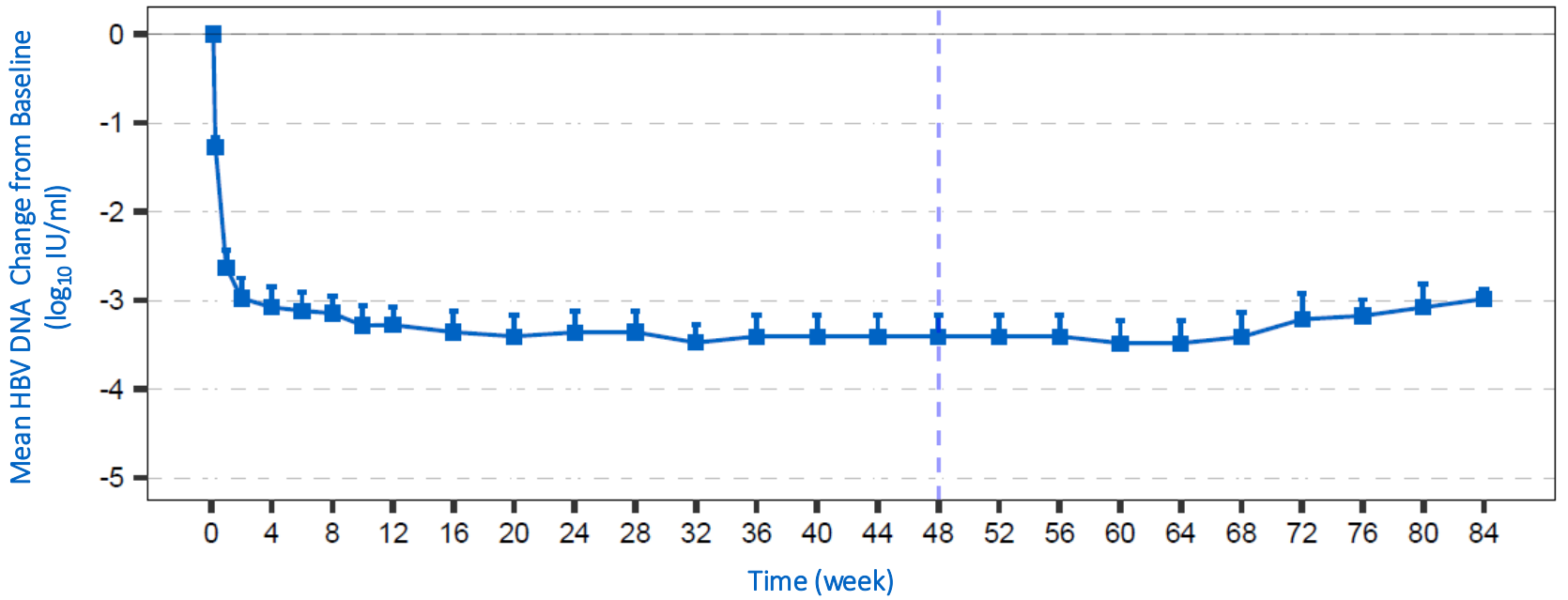
Antiviral Effect in CHB Subjects (HBeAg-)

Mean HBV DNA Change from Baseline



Week	0	4	8	12	16	20	24	28	32	36	40	44	48	52	56	60	64	68	72	76	80	84
Total n	11	11	11	11	10	11	11	10	11	11	11	11	11	11	11	10	10	9	8	6	4	2
≤ LLOQ [10 IU/mL]	0	10	10	10	10	11	11	10	11	11	11	11	11	11	11	10	10	9	7	6	4	2

Number of subjects at each timepoint

Data on file.

300 mg ALG-000184 showed rapid and sustained DNA reductions by week 24 with no viral breakthrough

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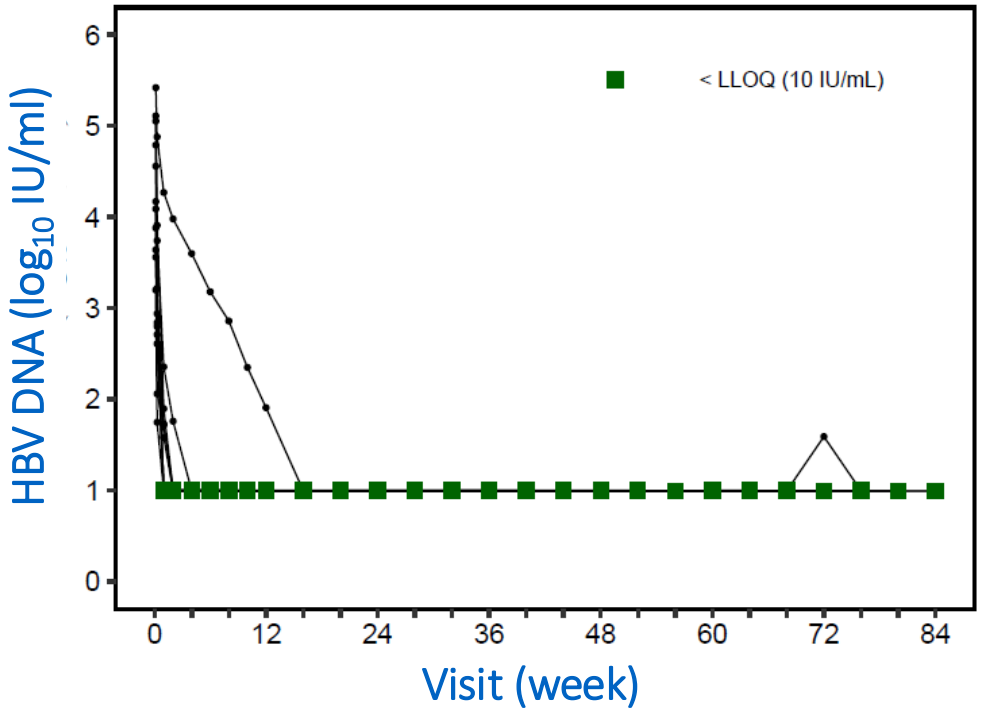
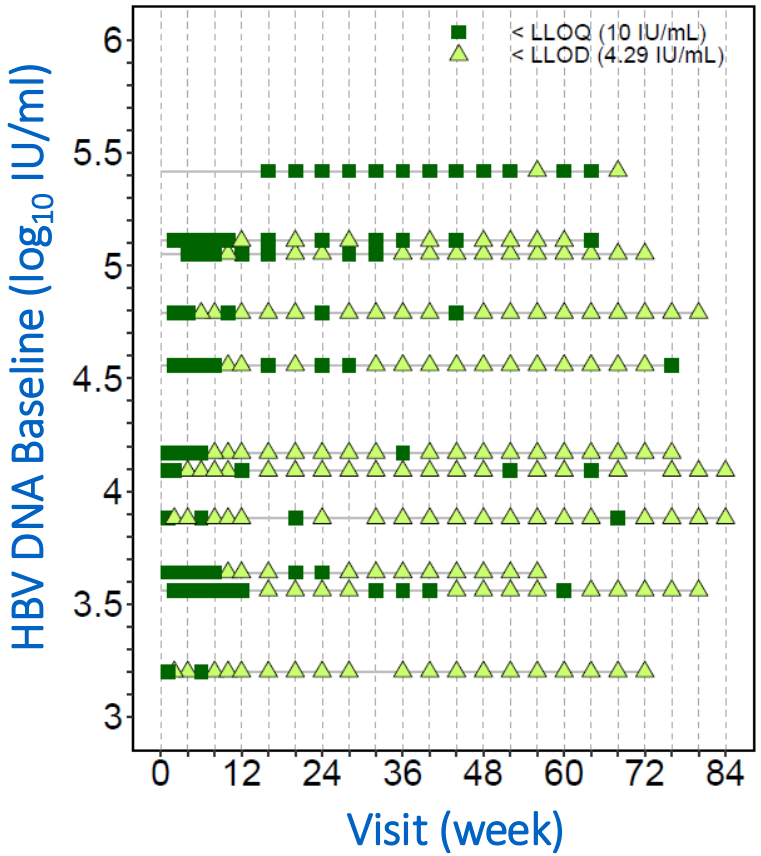
300 mg ALG-000184 Monotherapy (HBeAg-)

Individual HBV DNA Level Over Time



Total n	11	11	11	11	10	11	11	10	10	11	11	11	11	11	11	10	10	9	8	6	4	2
< LLOQ [10 IU/mL]	0	10	10	10	10	11	11	10	10	11	11	11	11	11	11	10	10	9	7	6	4	2
< LLOD [4.29 IU/mL]	0	3	5	7	6	8	6	7	6	7	9	8	10	9	11	8	7	8	7	5	4	2



n/N	0/11	10/11	11/11	11/11	11/11	10/10	7/8	2/2

Agarwal, K. et al. AASLD 2024. Data on file.

100% (11/11) of subjects achieved HBV DNA < LLOQ (10 IU/mL) by Week 24
91% (10/11) of subjects achieved HBV DNA < LLOD (4.29 IU/mL) by Week 48

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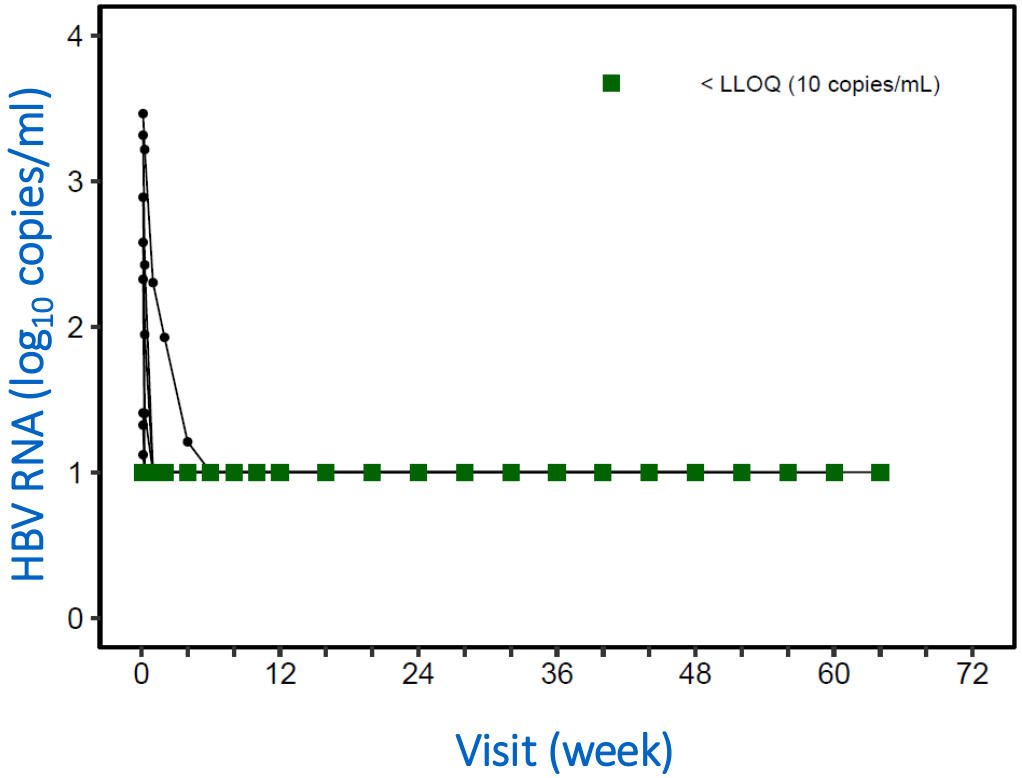
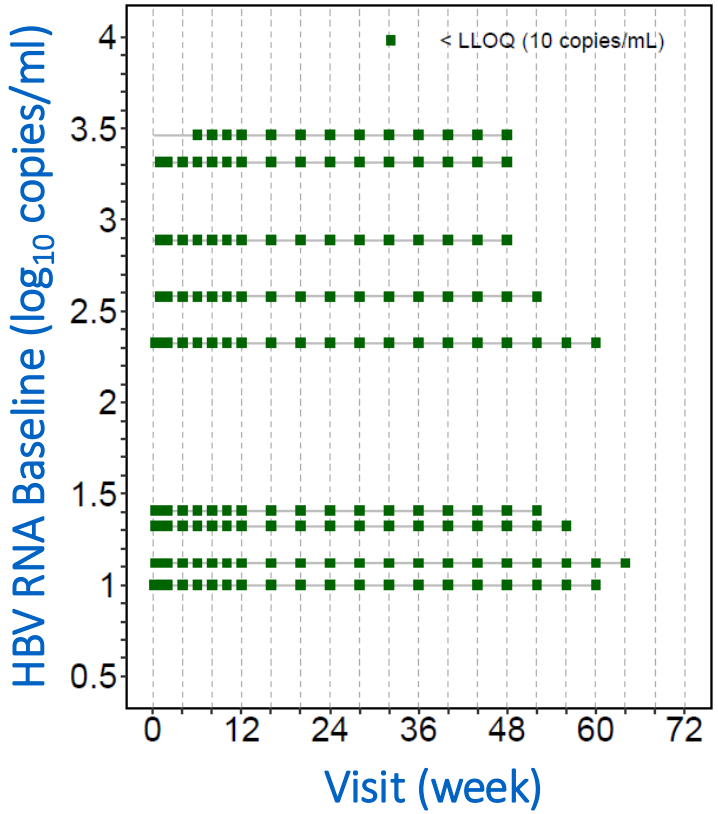
300 mg ALG-000184 Monotherapy (HBeAg-)

Individual HBV RNA Level Over Time



Total n	11	11	11	11	10	11	11	10	11	11	11	11	11	8	5	4	1
< LLOQ [10 copies/mL]	3	10	11	11	10	11	11	10	11	11	11	11	11	8	5	4	1



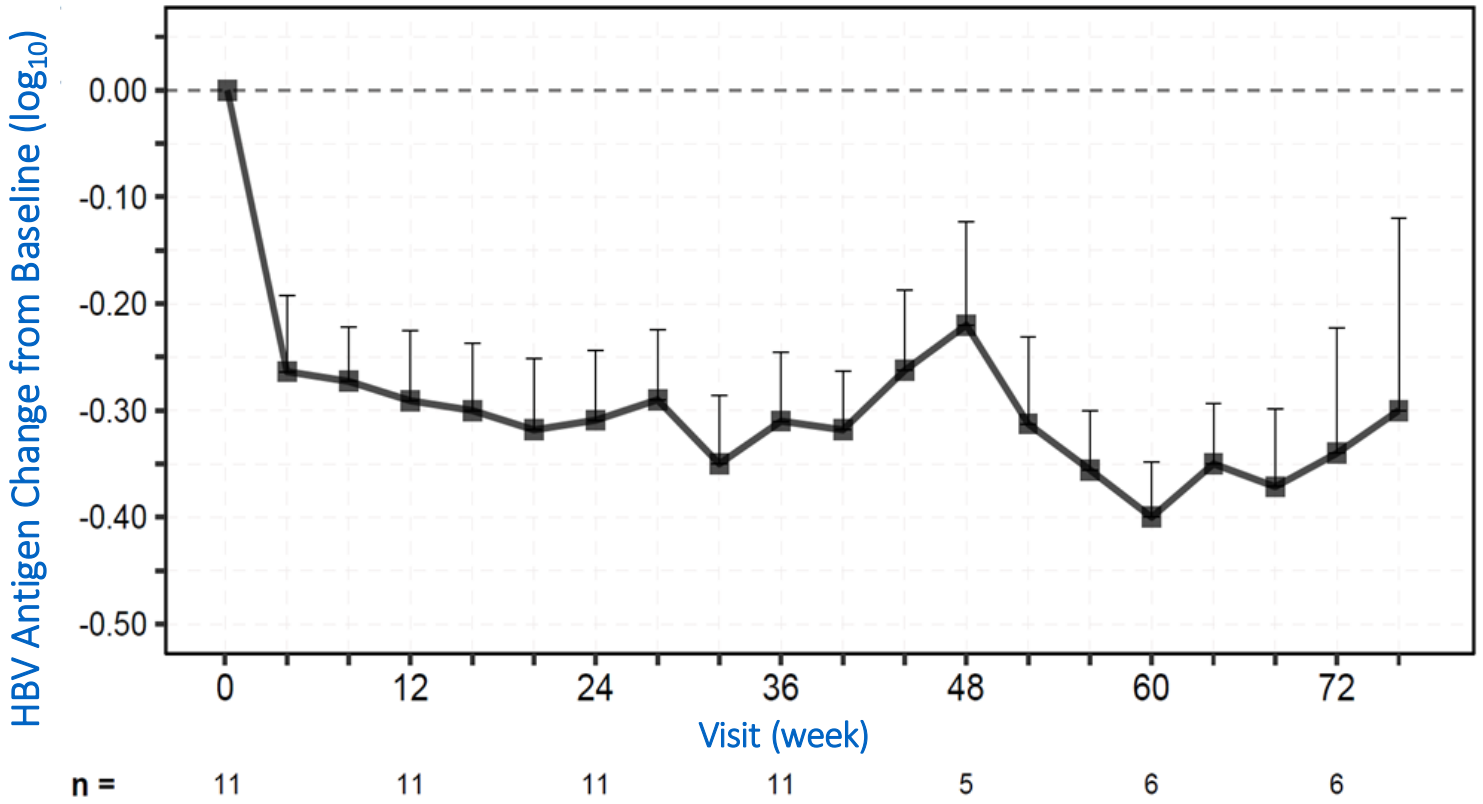
n/N	3/11	11/11	11/11	11/11	11/11	4/4

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

100% (11/11) of subjects achieved sustained HBV RNA < LLOQ (10 copies/mL) by week 8

300mg ALG-000184 Monotherapy (HBeAg-): Mean Change

Mean Decline in HBcrAg



Yuen, M-F. et al.; AASLD 2024. [1]Hosaka, et. al, AASLD, 2024.

Max mean decline of HBcrAg was 0.4 log_{10} U/mL in 300 mg ALG-000184 monotherapy ≤ 84 weeks
HBcrAg has been associated with the risk of developing HCC [1]

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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]	100%[4]	100%[4]	100% (60 weeks)[3]	100% (60 weeks)[3]
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]	100%[3]	60%[2]	100% (84 weeks)[1]	100% (84 weeks)[1]

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., AASLD 2024. [d] Agarwal, K. et al. EASL 2024. [e] Kosh A. et al.; Journal of Hepatology 2018 V68: 672-681.
[1] 7/7 subjects. [2] 6/10 subjects. [3] 10/10 subjects. [4] 11/11 subjects.

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

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300 mg ALG-000184 Monotherapy: Well-Tolerated for ≤ 92 Weeks in HBeAg+ and ≤84 Weeks in HBeAg- Subjects

Part 4 Cohort B 300 mg ALG-000184 Monotherapy	HBeAg + n=10	HBeAg- n=11
Total number of subjects with at least one TEAE	9	8
Subjects with Grade 3-4 TEAE		
Liver transaminase elevation	3*	1*
Other	0	1#
SAE	0	0
Total number of subjects withdrawn from study due to a TEAE	0	0
Concerning laboratory, ECG, vital sign, or physical examination findings	0	0

TEAE: treatment Emergent Adverse Event; SAE: serious adverse event; ECG: electrocardiogram

* 3 HBeAg+ subjects and 1 HBeAg- subject experienced Grade ≥ 3 ALT↑ with or without associated AST↑. All subjects were asymptomatic and none of the ALT increases were associated with hepatic synthetic dysfunction. The ALT Flare Monitoring Committee assessed the events were associated with potent antiviral effects; There was no concern of liver toxicity. All ALT↑ resolved in setting of continued ALG-000184 dosing.

1 HBeAg- subjects with abnormal lipid measurements at baseline experienced asymptomatic increase in cholesterol and LDL levels, that fluctuated between DAIDS Grade 2 and Grade 4 laboratory abnormalities (all deemed Grade 3 TEAEs by the investigator) during the dosing period.

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

ALG-000184

- Regulatory Pathway
- Development Plans

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Chronic Suppression

Well Defined, Validated Approval Pathway

- Regulatory pathway for chronic suppressive therapy endorsed by FDA, CHMP (EMA) , 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; EMA 2006; China 2023; LLOQ: lower limit of quantitation.

Aligos has received FDA, CHMP (EMA), and National Medical Products Administration in China feedback supporting subsequent studies utilizing this pathway

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ALG-000184 Phase 2 Chronic Suppression Study

Planned Efficacy Endpoints & Potential for Clinical Differentiation

Primary endpoint (approvable endpoint*)

- Proportion of subjects with HBV DNA < 10 IU/mL at W48 for both HBeAg+/- CHB subjects

Key secondary and exploratory endpoints (clinically meaningful and/or corroborative)

- Reduction of HBsAg, HBcrAg[1], HBeAg
- Reduction of cccDNA level and/or related serum biomarkers
  - Via paired biopsies
- Reduction of HBV integrants^
- HBV RNA < LLOQ^
- ALT normalization

} Clinical differentiation vs. SOC

*FDA Guidance April 2022.
^Possible link to cancer risk (HCC). [1] Chang, KC, et.al, Viruses, 2022.

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

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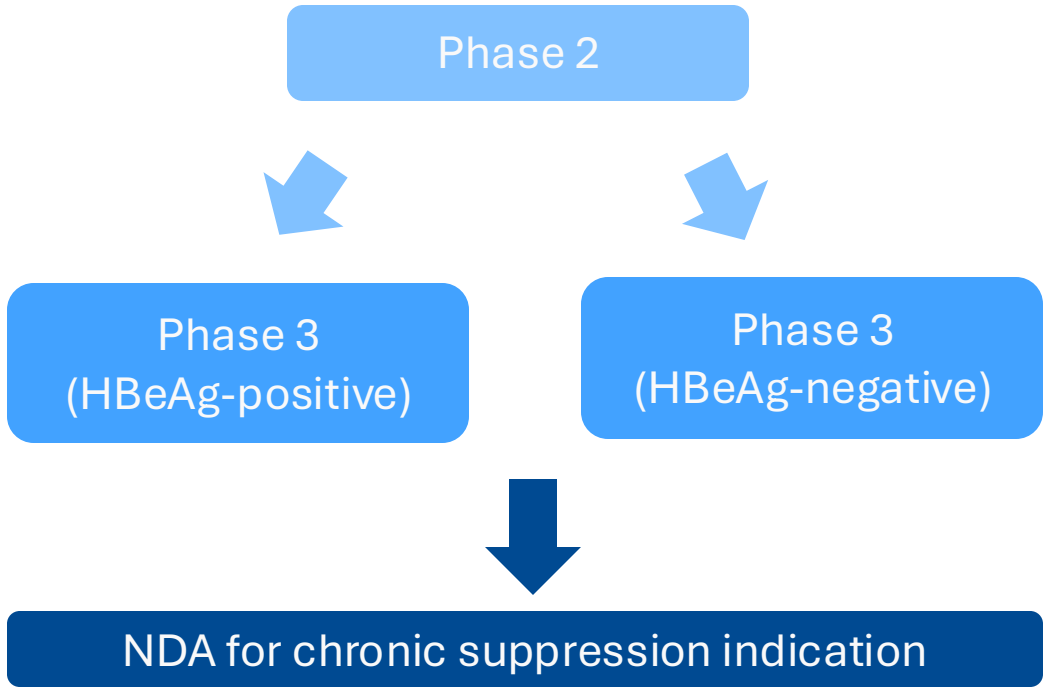
ALG-000184 Development Plans



Phase 2

- **Trial design:** Randomized, double-blind, active controlled study of ALG-000184 vs. tenofovir disoproxil fumarate (TDF) in HBeAg-positive and HBeAg-negative CHB subjects
- **Primary efficacy endpoint:** Proportion of subjects with HBV DNA levels below 10 IU/mL (<LLOQ) at Week 48

Phase 3

- **Trial designs:** Randomized, double-blind, active controlled registrational studies of ALG-000184 monotherapy versus tenofovir alafenamide (TAF) monotherapy in HBeAg-positive and HBeAg-negative CHB subjects
- **Approvable endpoint:** Superiority as determined by the proportion of subjects with HBV DNA levels below 10 IU/mL (i.e., <LLOQ, target detected) at Week 48

LLOQ: lower limit of quantitation using most sensitive Roche COBAS assay.

ALG-000184 is the only investigational drug with a path to approval utilizing this regulatory pathway

ALIGOS THERAPEUTICS

ALG-055009 for MASH

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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
  - 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

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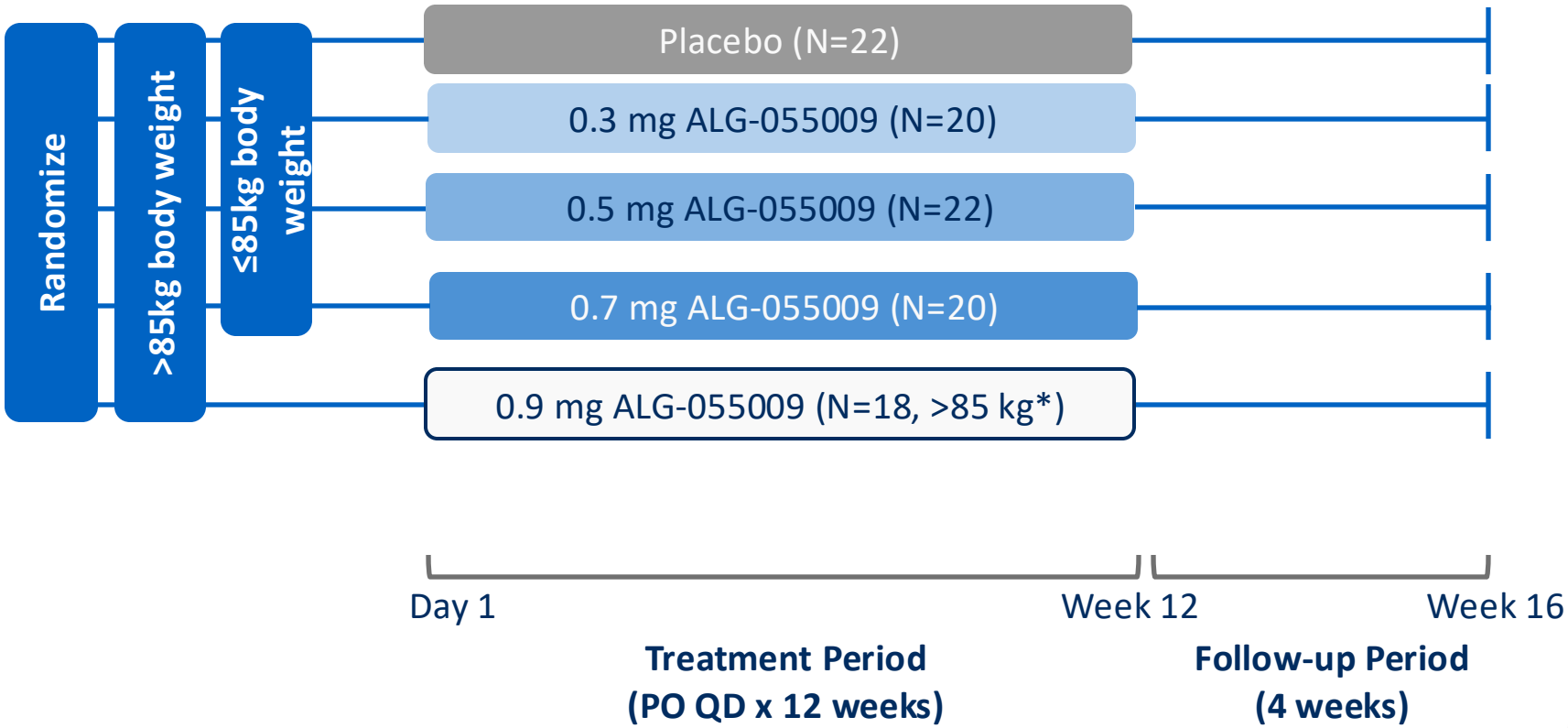
[1]Not including any patent term extension.

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

^stable use (67% subjects for >1 year)

*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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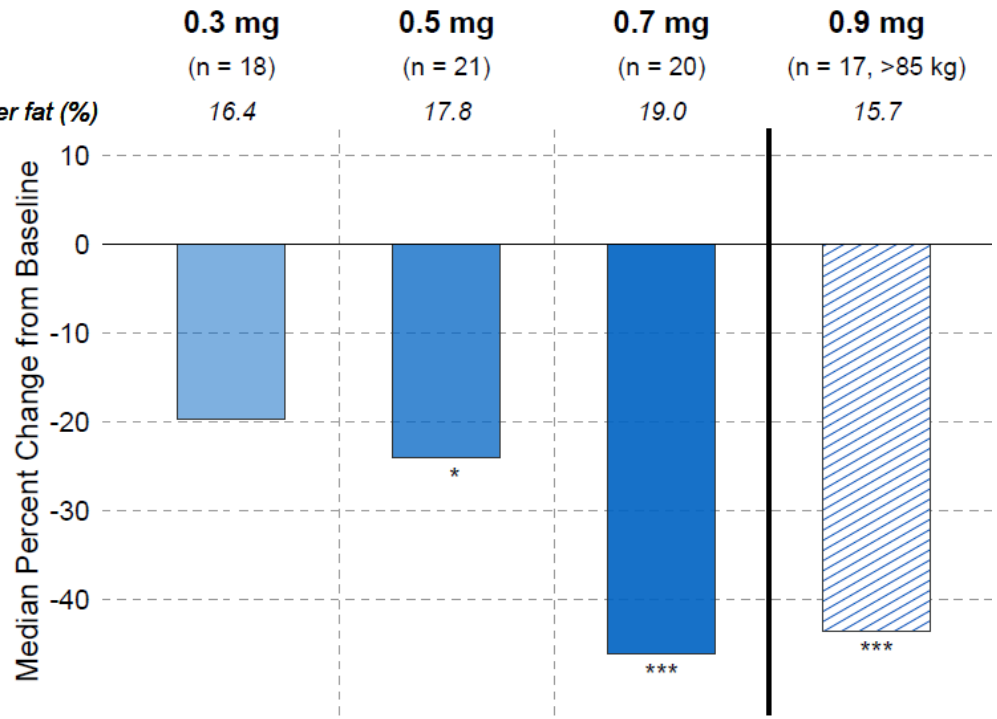
HERALD: Primary Endpoint Achieved

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

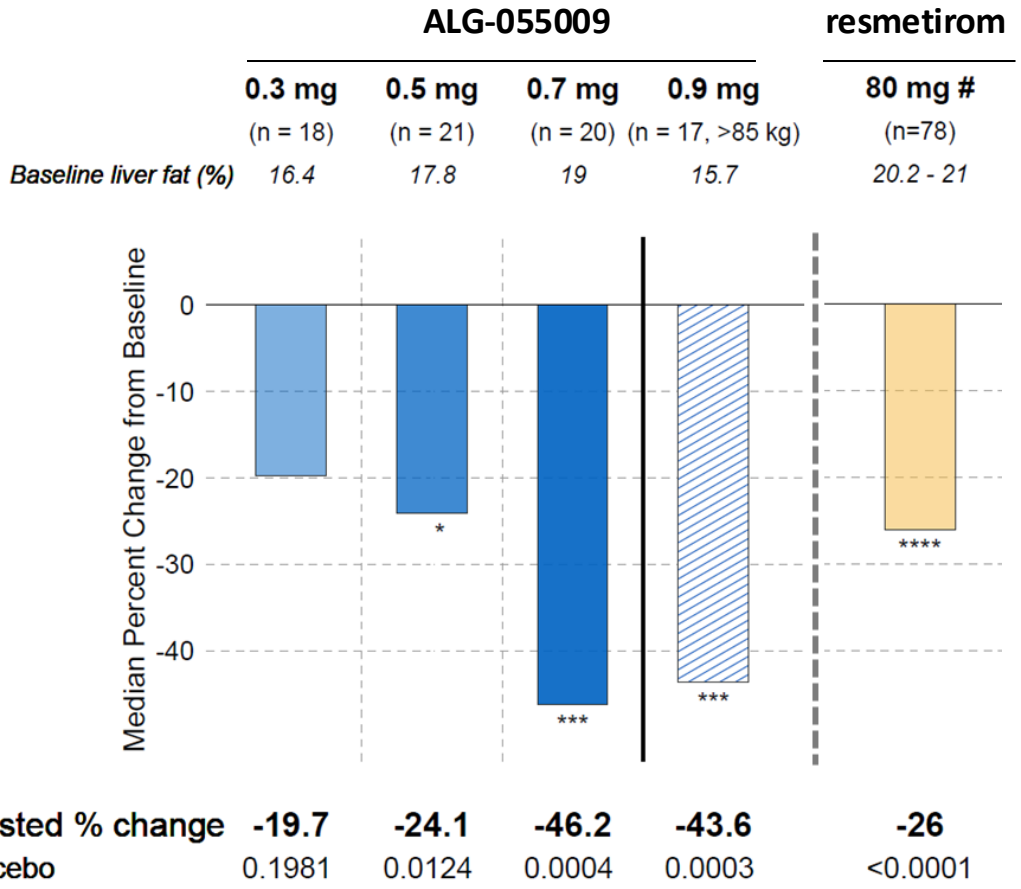
Note: Only subjects weighing >85 kg were enrolled in the 0.9 mg dose group, no body weight restrictions were implemented in other dose groups.
Data from MRI-PDFF analysis dataset, defined as all randomized subjects who have MRI-PDFF measurements available at both baseline and Week 12; median % change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

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

ALIGOS THERAPEUTICS

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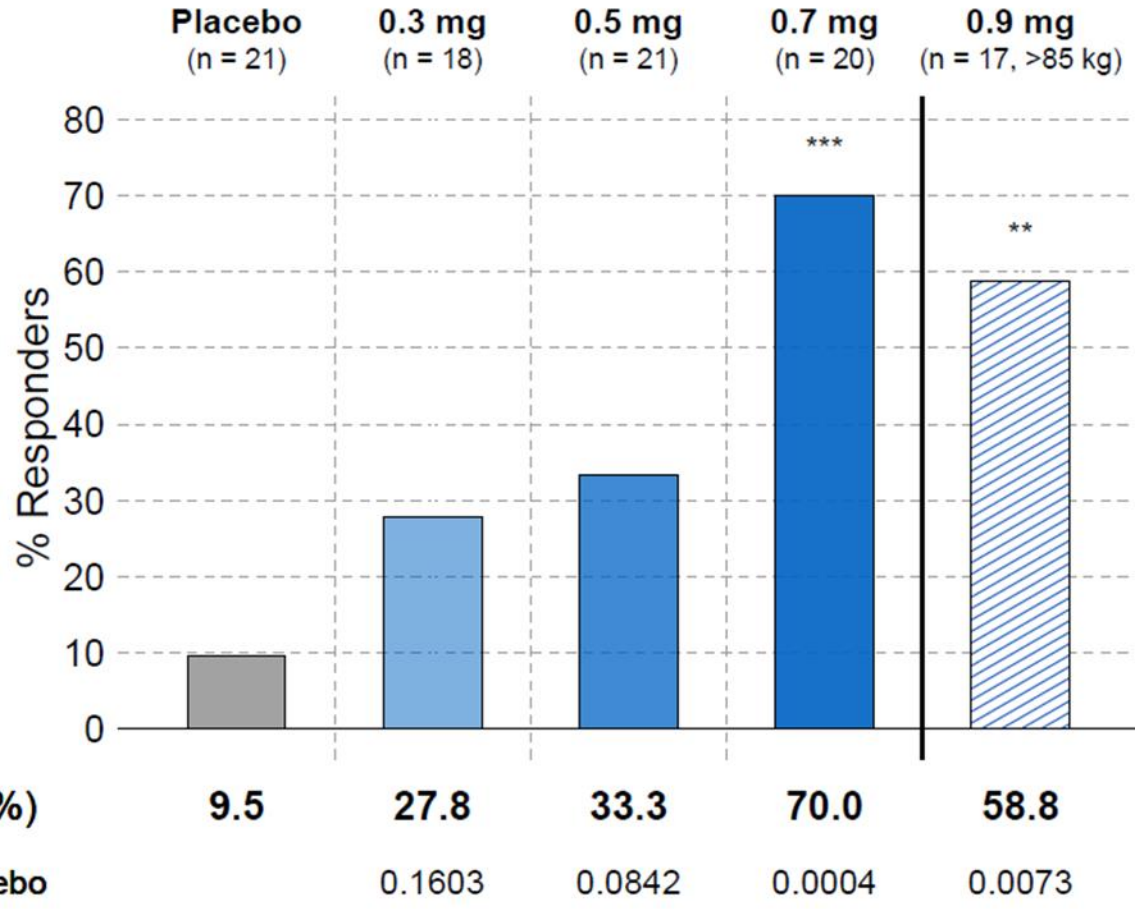
ALG-055009 demonstrated robust improvements in liver fat content compared to data from the study evaluating the only approved MASH treatment

HERALD: Significant MRI-PDFF Response Rates at Week 12

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



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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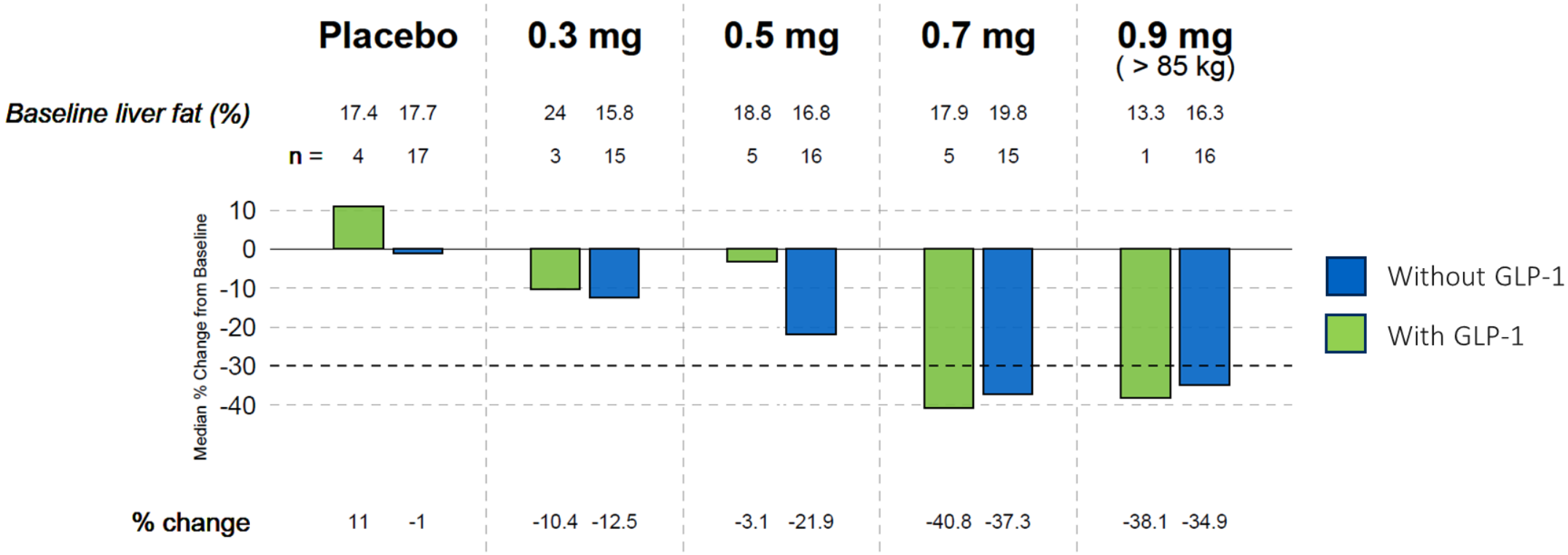
Additional Fat Reduction in Subjects with Stable Use of GLP-1 Agonists

Median Relative Percent Change in Liver Fat at Week 12



Note: Subjects on GLP-1 agonists (semaglutide (n=12), liraglutide (n=4) or dulaglutide(n=2)) at baseline were required to have stable use for ≥12 weeks prior to randomization; for derivation of duration of use, if a month and/or day for the start of GLP-1 agonist use was unknown, it was imputed as January and/or the 1st of the month, respectively. Bolded dashed line indicates 30% relative reduction in liver fat.

11/14 subjects on stable GLP-1 treated with ALG-055009 had liver fat decreases, whereas 4/4 subjects on stable GLP-1 treated with placebo had liver fat increases

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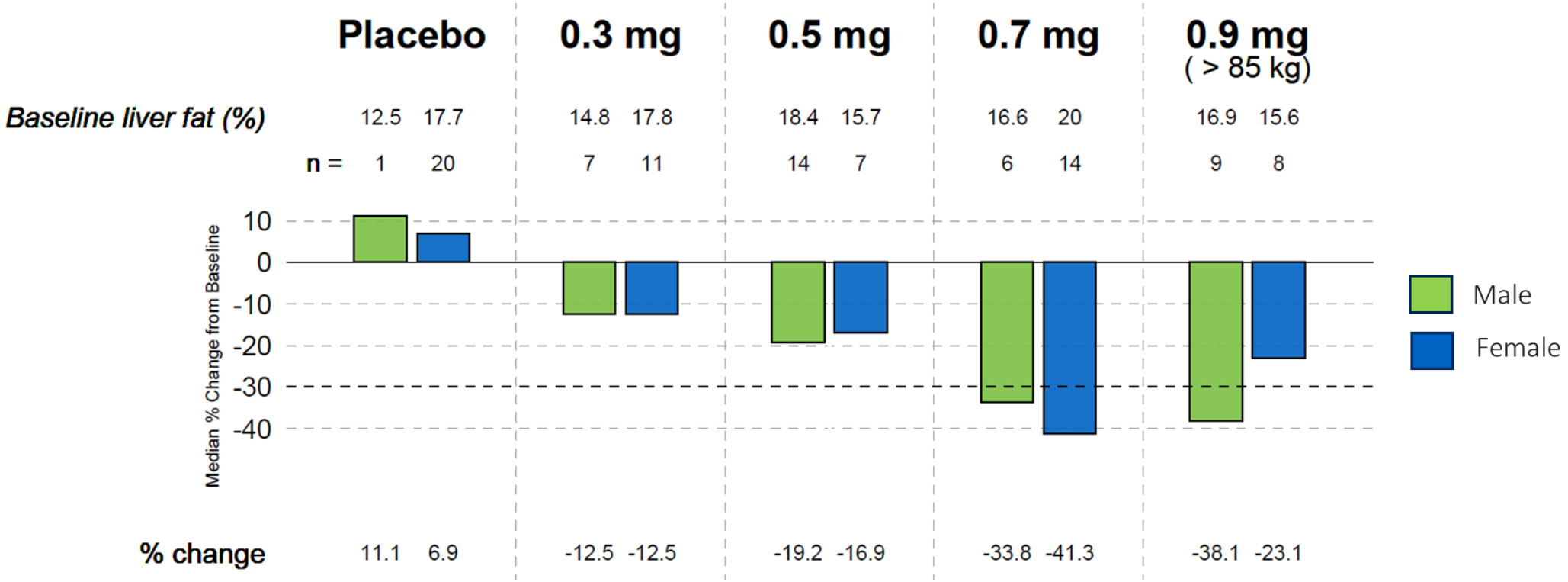
No Apparent Impact of Gender on Liver Fat Reduction with ALG-055009

Median Relative Percent Change in Liver Fat at Week 12



Note: Bolded dashed line indicates 30% relative reduction in liver fat.

Substantial, dose-dependent liver fat reductions observed among both males and females

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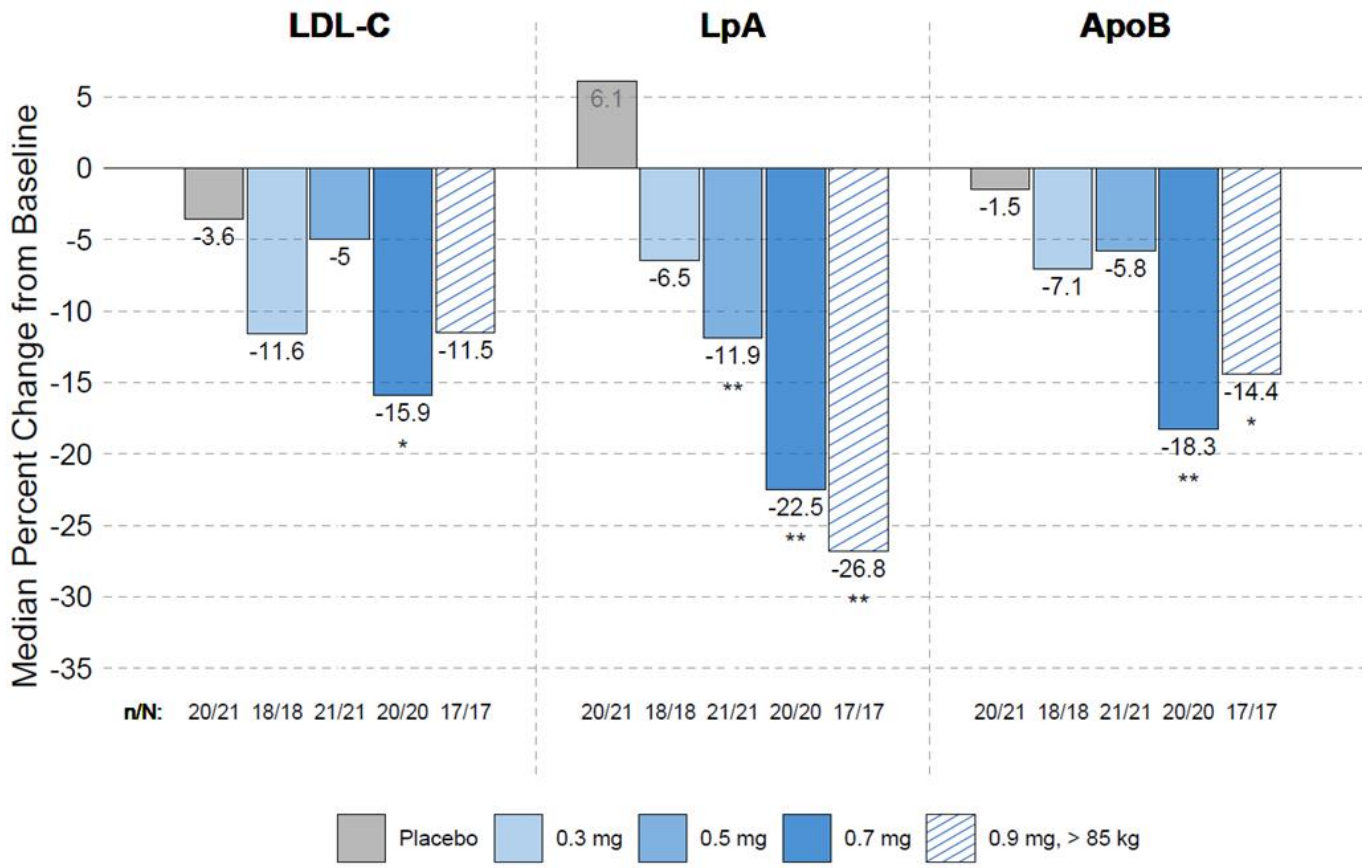
ALG-055009 Demonstrated Improvements in Lipid/Lipoproteins

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.

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

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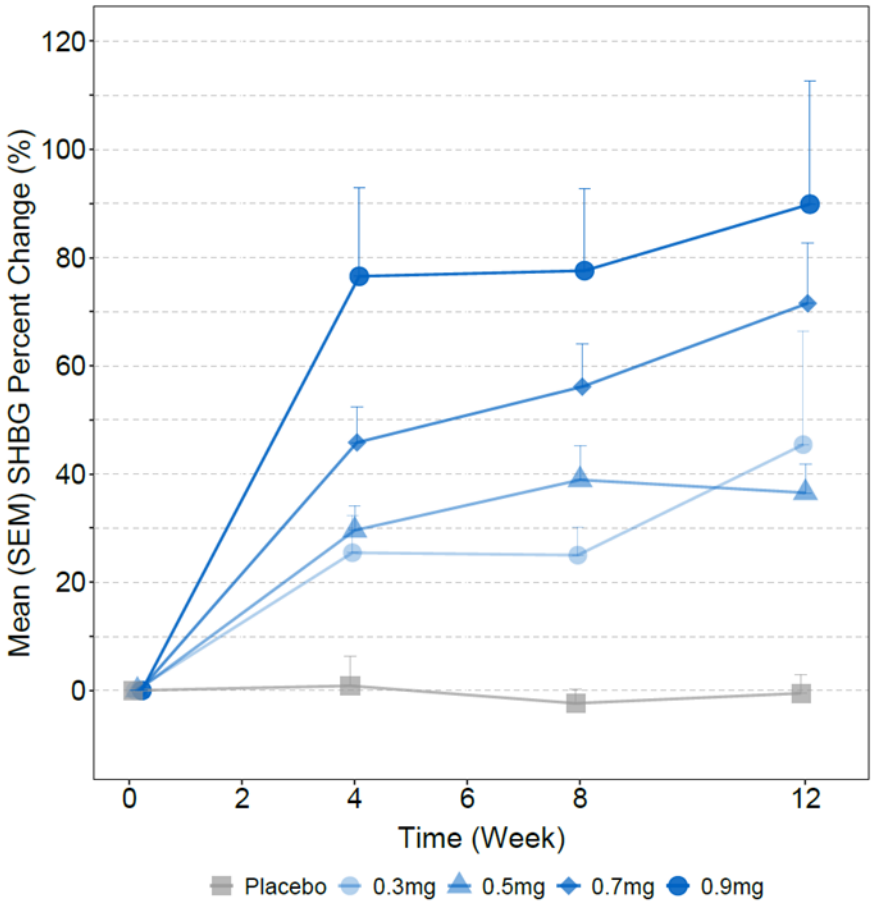
HERALD: Dose-dependent Increases in Sex Hormone Binding Globulin

Up to ~90% Increase from Baseline in Sex Hormone Binding Globulin (SHBG)



SHBG is a marker of THR-β target engagement in the liver
Significant ($p<0.0001$) increases compared to placebo observed

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HERALD: Favorable Safety and Tolerability Profile

Rates of GI-related TEAEs Similar to Placebo

- No SAEs in subjects receiving ALG-055009
  - One unrelated SAE (hemangioma of bone) in a subject receiving placebo
- One discontinuation due to a treatment emergent adverse event (TEAE) of worsening insomnia in a subject with pre-existing insomnia
- Majority of TEAEs (97%) mild or moderate with no clinical hypo/hyperthyroidism
- Similar rates of diarrhea noted for active dose groups compared to placebo, with no dose-response
- No clinically concerning laboratory, ECG, physical exam or vital sign trends/findings

HERALD: Favorable Safety and Tolerability Profile

Rates of GI-related TEAEs Similar to Placebo

n, (%)	Placebo (N=22)	ALG-055009			
		0.3mg (N=20)	0.5mg (N=22)	0.7mg (N=20)	0.9mg (N=18)
Any TEAE	17 (77.3)	14 (70.0)	11 (50.0)	14 (70.0)	11 (61.1)
TEAE Leading to Study Drug Discontinuation	0	0	1[a] (4.5)	0	0
Serious AE	1[b] (4.5)	0	0	0	0
Grade 3 or higher TEAE	1[b] (4.5)	1[c] (5.0)	0	0	0
Gastrointestinal TEAEs	5 (22.7)	4 (20.0)	2 (9.1)	7 (35.0)	5 (27.8)
Diarrhea	5 (22.7)	1 (5.0)	0 (0.0)	2 (10.0)	2 (11.1)
Constipation	0 (0.0)	2 (10.0)	0 (0.0)	3 (15.0)	0 (0.0)
Nausea	1 (4.5)	2 (10.0)	0 (0.0)	0 (0.0)	1 (5.6)
Vomiting	1 (4.5)	1 (5.0)	1 (4.5)	0 (0.0)	0 (0.0)

TEAE = treatment emergent adverse event

a. Grade 2 worsening insomnia in a subject with pre-existing insomnia; b. Grade 3 hemangioma of bone; c. Grade 3 anemia assessed by the Investigator as not related to study drug in a subject with heavy menstrual bleeding and a history of polycystic ovary syndrome and heavy menstrual periods.

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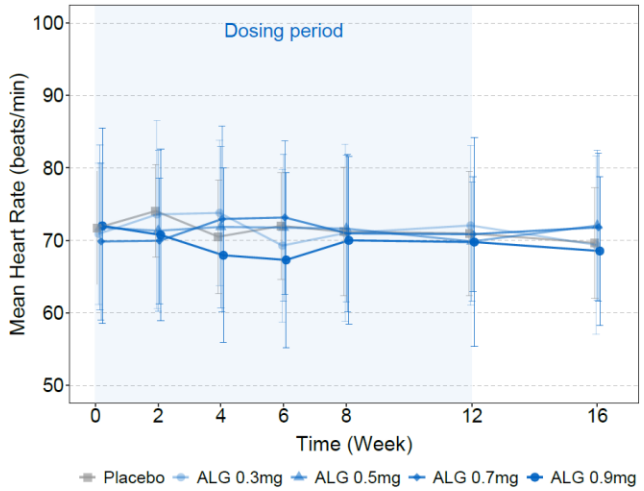
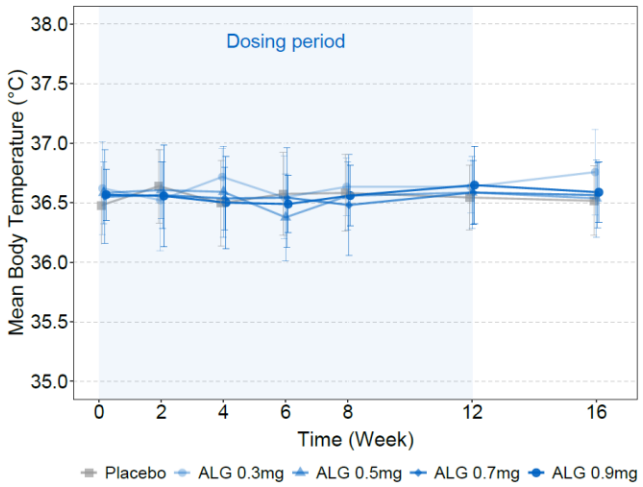
HERALD: No Treatment Emergent Changes in Vital Signs Observed

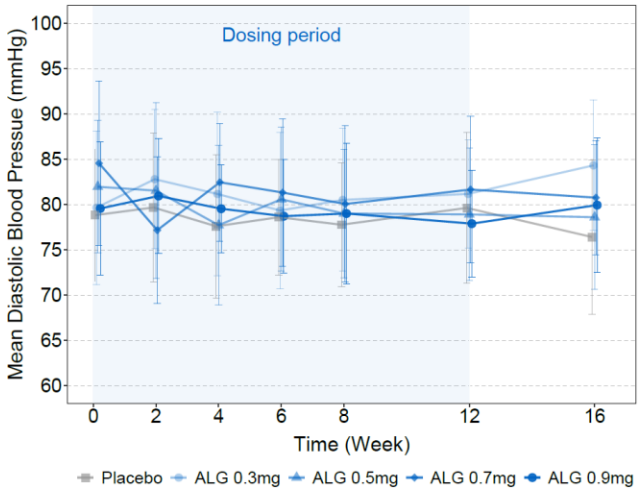
Heart Rate



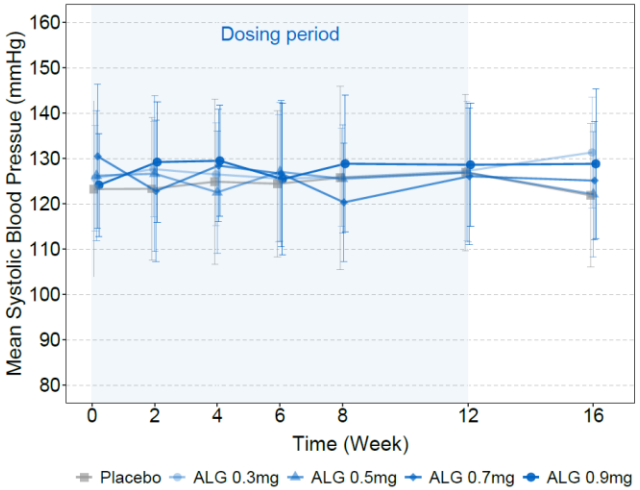
Body Temperature



Diastolic Blood Pressure



Systolic Blood Pressure



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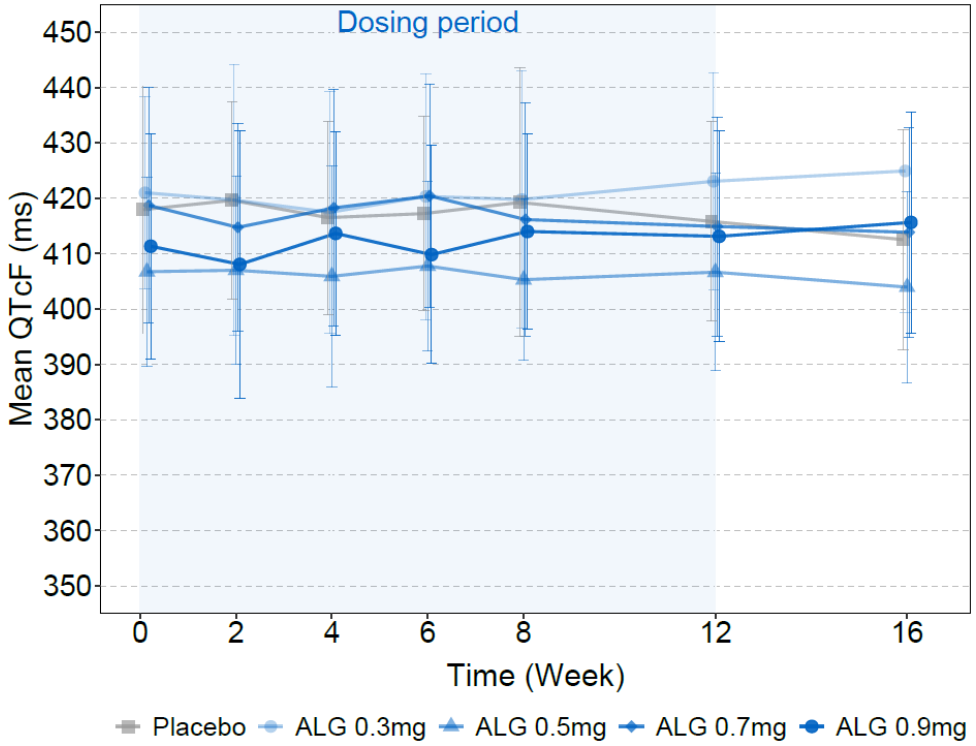
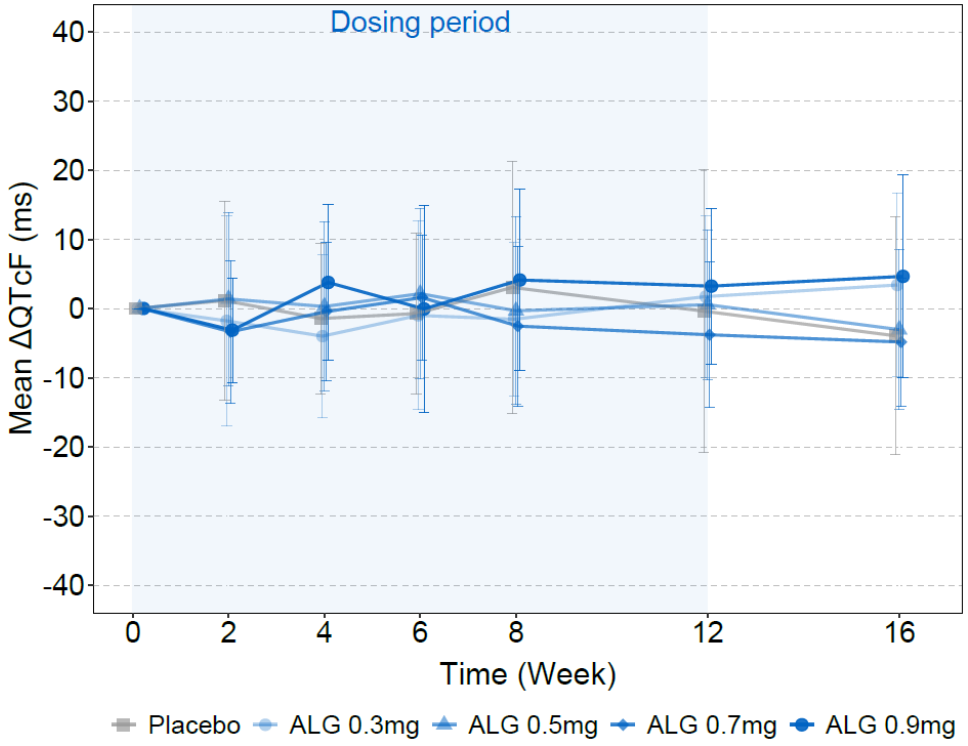
HERALD: ALG-055009 has no Apparent Effect on QTcF Intervals





No trends for QTcF prolongation across dose levels
No subject receiving ALG-055009 experienced a QTcF >500 ms and/or change from baseline in QTcF of >60 ms

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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

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

ALG-000184 for CHB

- ✓ Greater DNA suppression observed vs. NAs
- ✓ Phase 1b study is ongoing with interim data readouts at future scientific conferences
- ✓ Clear regulatory path forward for chronic suppressive therapy with superiority label with the FDA, CHMP (EMA), and National Medical Products Administration in China
- Phase 1b exploratory combination study with mipeginterferon alfa-2b (Amoytop) expected to begin in 2025
- Phase 2 enabling activities ongoing; planned Phase 2 IND filing in Q1 2025

ALG-055009 for MASH

- 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
- Discussions with partners underway; evaluating a variety of options to fund continued development

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)

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

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