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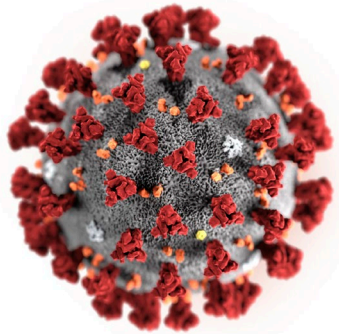
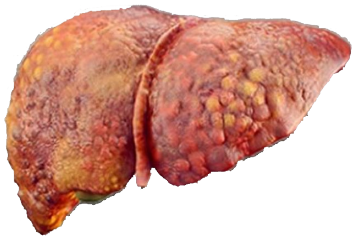
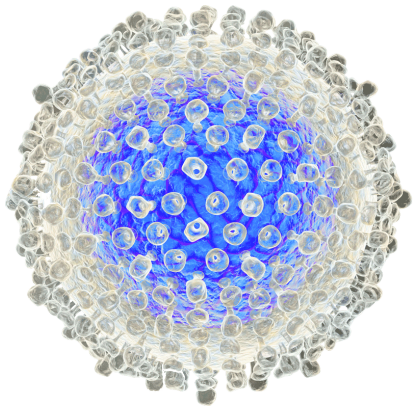






Lawrence M. Blatt, Ph.D.
Chairman, CEO & Co-Founder

Piper Sandler 35th Annual Healthcare Conference
November 30, 2023

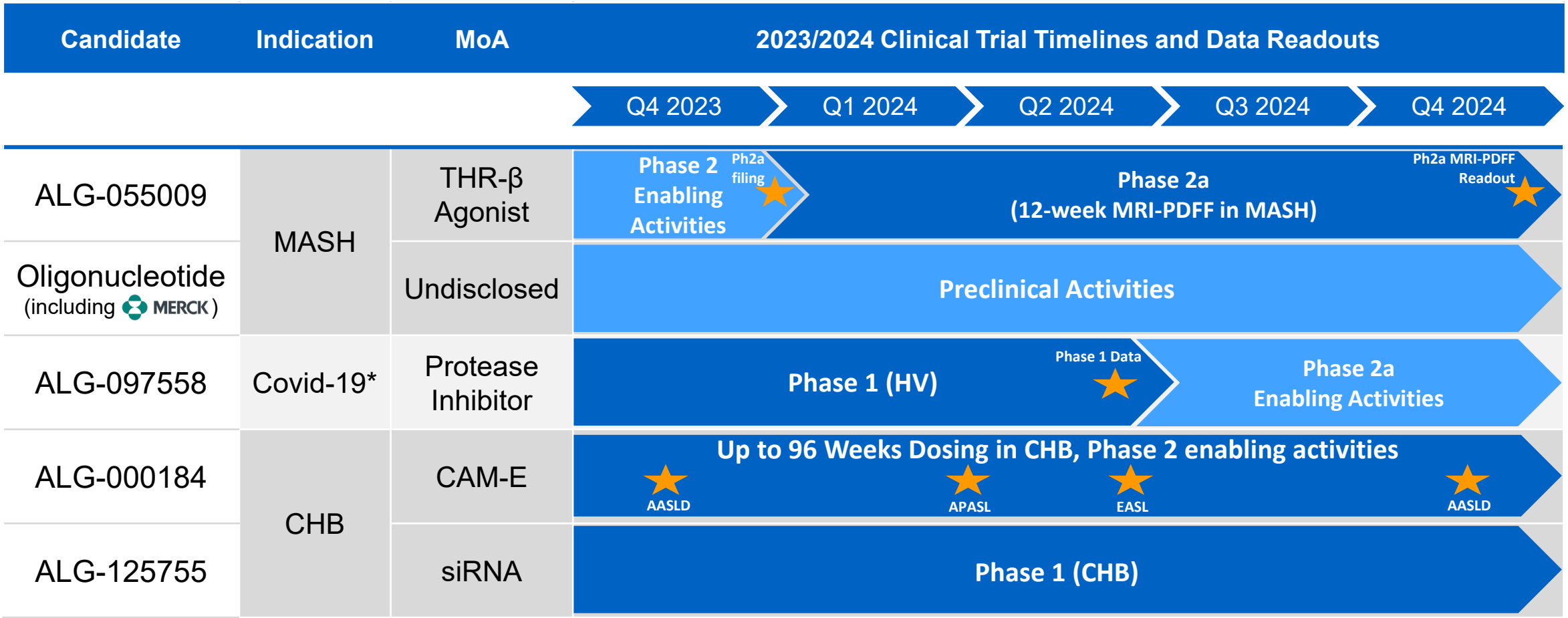
Disclosures

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' Quarterly Report on Form 10-Q filed with the Securities and Exchange Commission on November 2, 2023, 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
Multiple Milestones/Data Readouts Anticipated in 2023/2024



*Our Covid protease inhibitor efforts are partly funded (>$11M 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.

All timelines are approximate and subject to change based on enrollment and operational considerations. Presentations at conferences are subject to abstract approvals.

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; siRNA = small interfering ribonucleic acid (RNAi); THR-β = thyroid hormone receptor beta.

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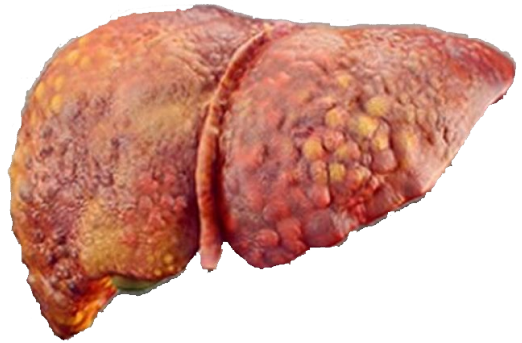
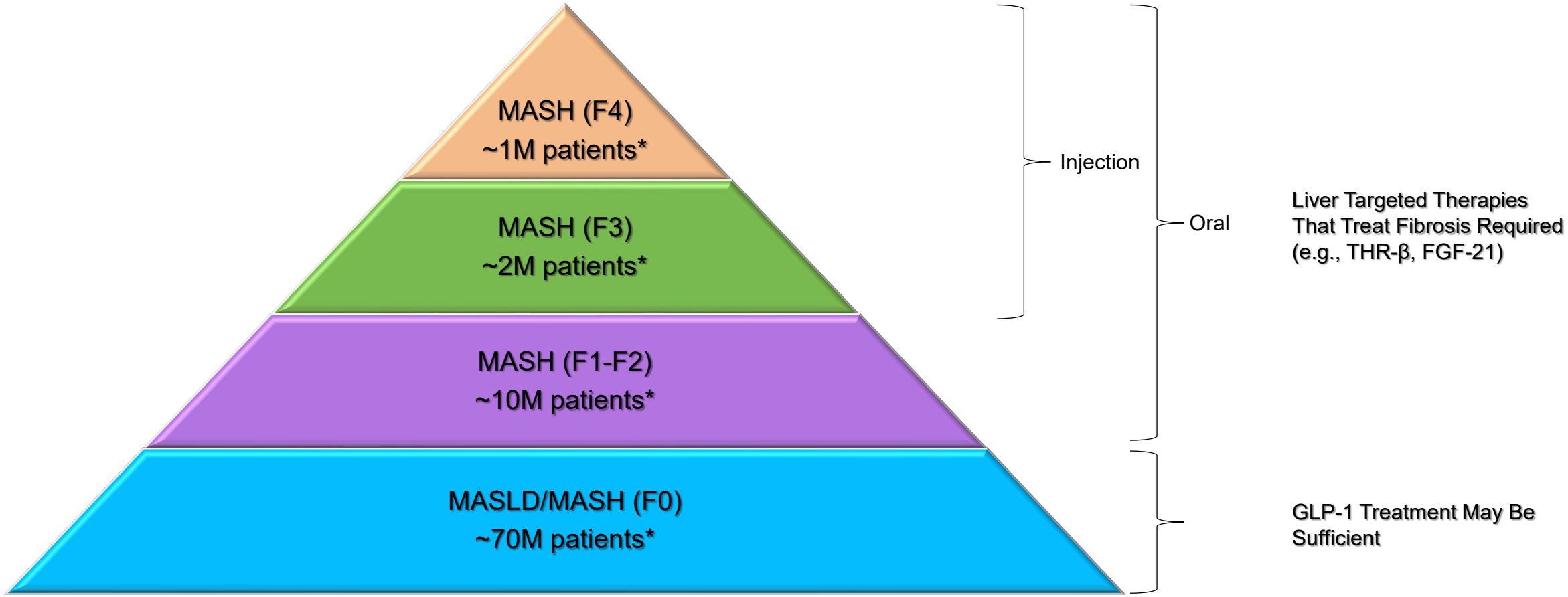
MASH

- ALG-055009, small molecule THR-β agonist



MASLD/MASH
Potential Future Treatment Paradigm



*Estimated US Incidence (~3% of Global Incidence)

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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Source of MASLD/MASH epidemiological data: https://www.ddw-online.com/evolving-models-for-nash-drug-discovery-tools-for-stemming-a-silent-epidemic-1791-201910/.

MASH Pathogenesis

Fatty acids = primary source of excess energy supply
(from de novo lipogenesis & lipolysis)

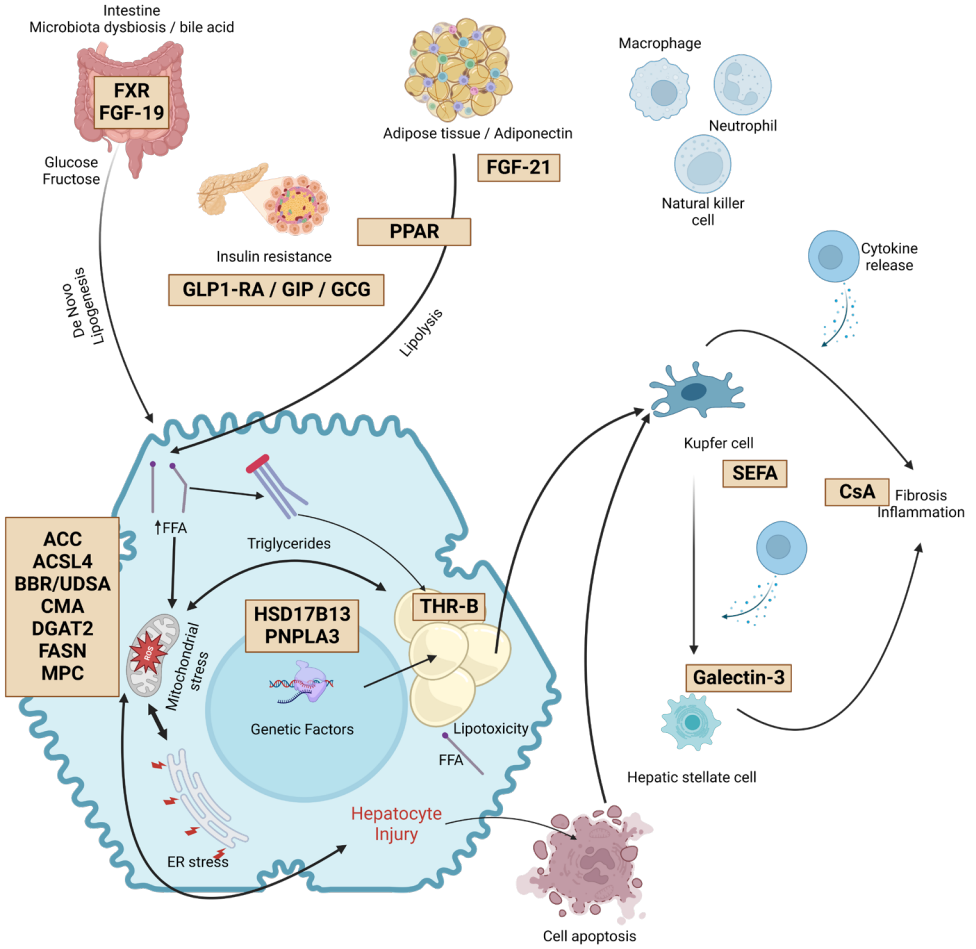
↓

Lipotoxic Species

↓

Liver Injury

Endoplasmic reticulum stress
Mitochondrial dysfunction
Pro-inflammatory cytokines release
Apoptosis



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

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Adapted from Stephen Harrison presentation, AASLD 2023.

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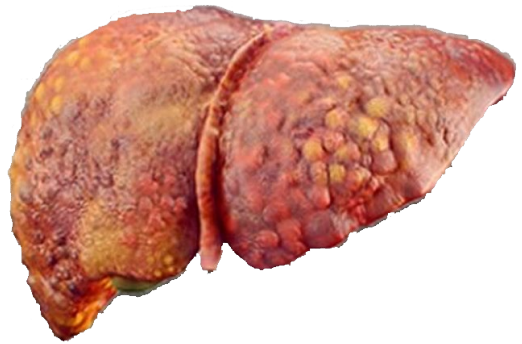
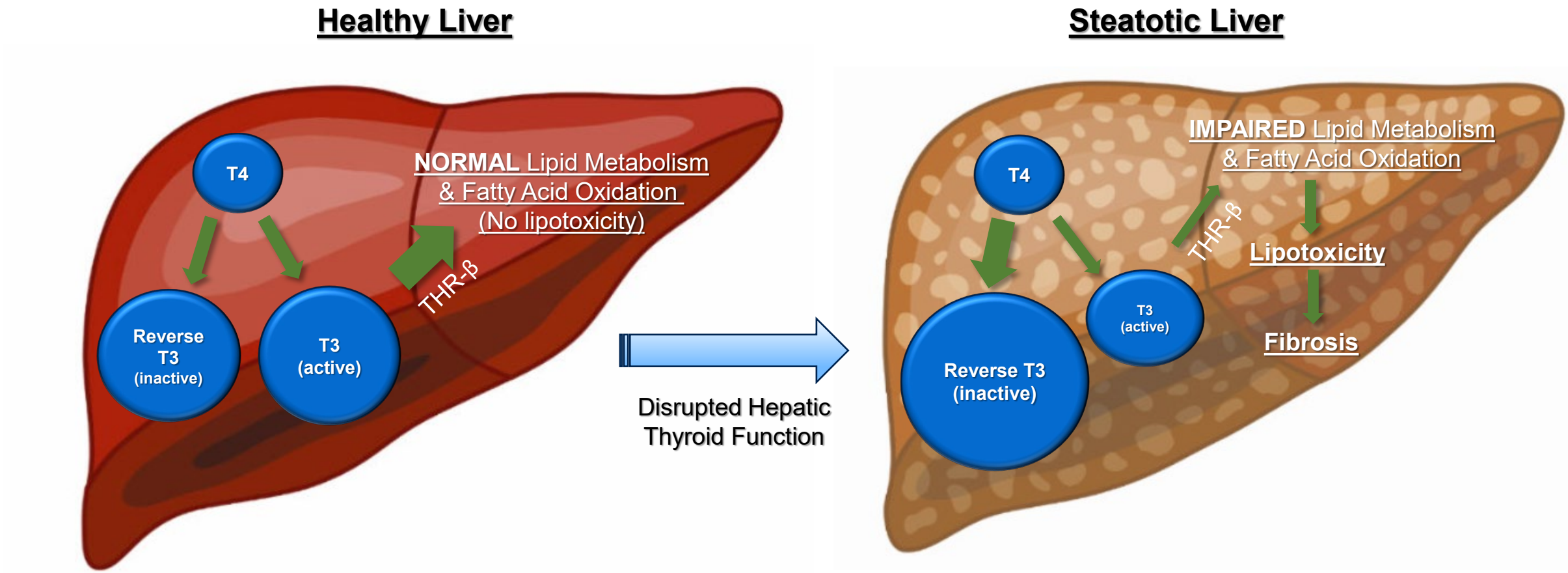
Thyroid Hormone Receptor β (THR-β)



Role of Hepatic Thyroid Dysfunction in NAFLD/MASH Pathogenesis



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

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

ALG-055009
Comparison vs. Other THR-β Drugs (Resmetirom, TERN-501, VK-2809)

Parameter	Resmetirom	TERN-501	VK-2809	ALG-055009
β/α Selectivity	2.5 ✓	2.3 ✓	1.4	3.8 ✓✓
THR-β Potency (EC_{50} HEK293T cell-based assay, nM)	2370	1750	267 ✓	50 ✓✓
PK - half life ($t_{1/2}$)	Short	Long ✓	Long ✓	Long ✓
PK - linearity	Nonlinear	Linear ✓	Linear ✓	Linear ✓
Potency (Ph2 daily doses)	Low (60-100 mg)	Higher (3 - 6 mg)	Higher (2.5-10* mg)	Highest (<1 mg) ✓
MRI-PDFF (12 weeks)				
Relative Fat Reduction (median)	36%	27-45%	48-55% ✓	TBD
% with ≥30% reduction	60%	39-64%	78-85% ✓	TBD

Enhanced PK and pharmacology vs. resmetirom results in enhanced 12-week MRI-PDFF
ALG-055009 profile may result in further enhanced efficacy

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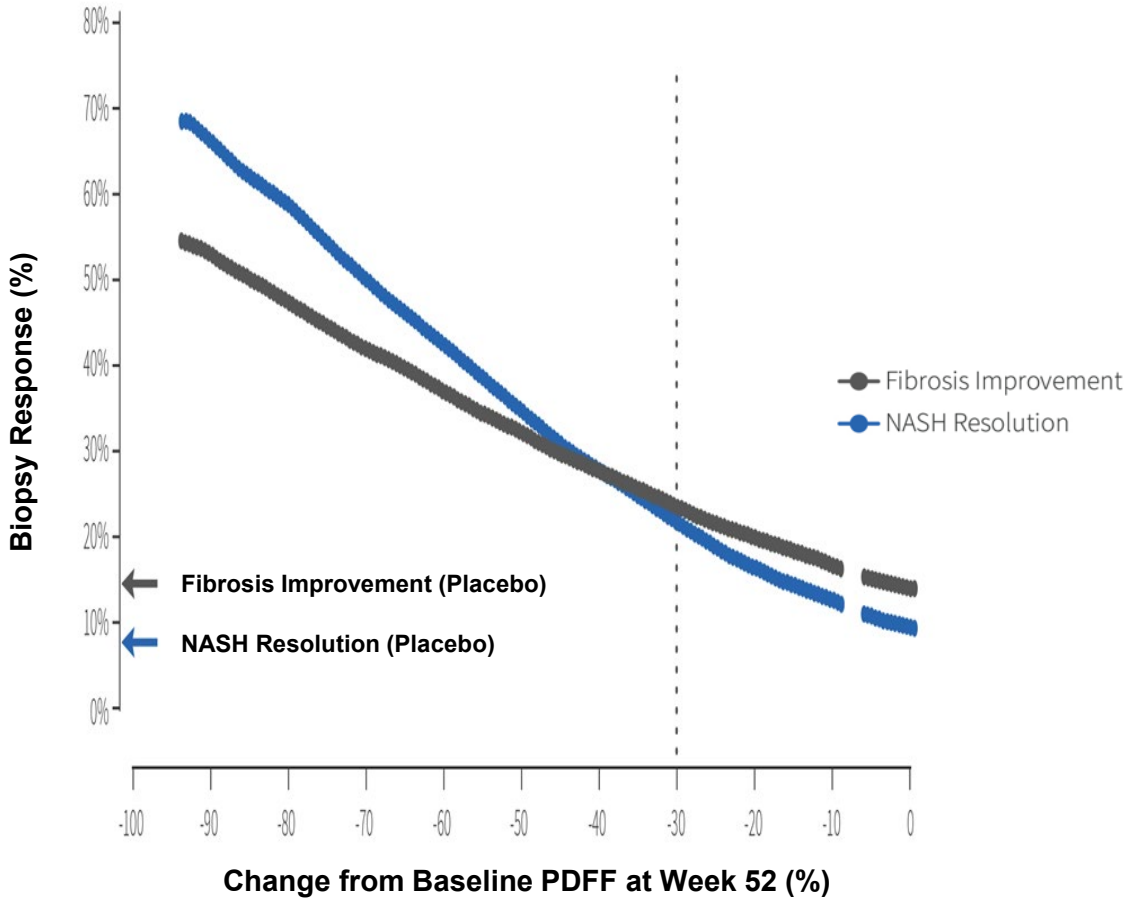
Viking data: Lian et al., ACC 2016 and May 16, 2023, press release. Madrigal data: Taub, Atherosclerosis, 2013; Harrison, EASL, 2023.
TERN-501 data: Quirk et al., TERNS Corporate presentation, August 8, 2023, Nelson et al., EASL 2022 and US 2020/0190064 A1.
*10 mg dose given QOD.

Resmetirom Phase 3 Data
MRI-PDFF and Liver Biopsy Correlation



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

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

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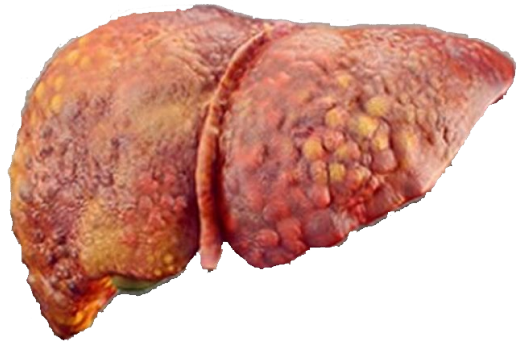
ALG-055009 Phase 1 Clinical Data



ALG-055009 Phase 1
Study Design

Part 1: Single Ascending Dose (SAD)

N = up to 64 Healthy Volunteers

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

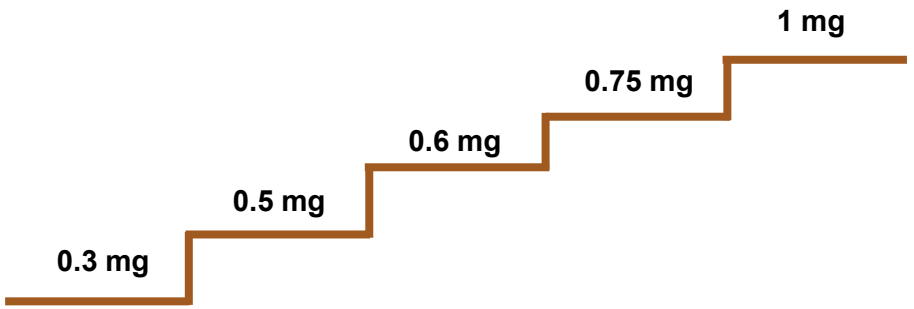
Part 2: Multiple Ascending Dose (MAD) – Dosing PO QD X 14 days

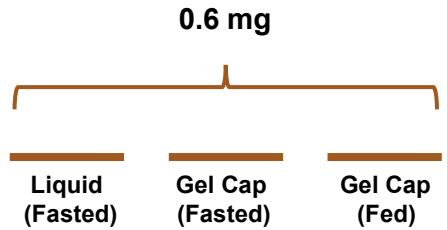
N = up to 80 Subjects with Hyperlipidemia

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

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

N = 10 Healthy Volunteers





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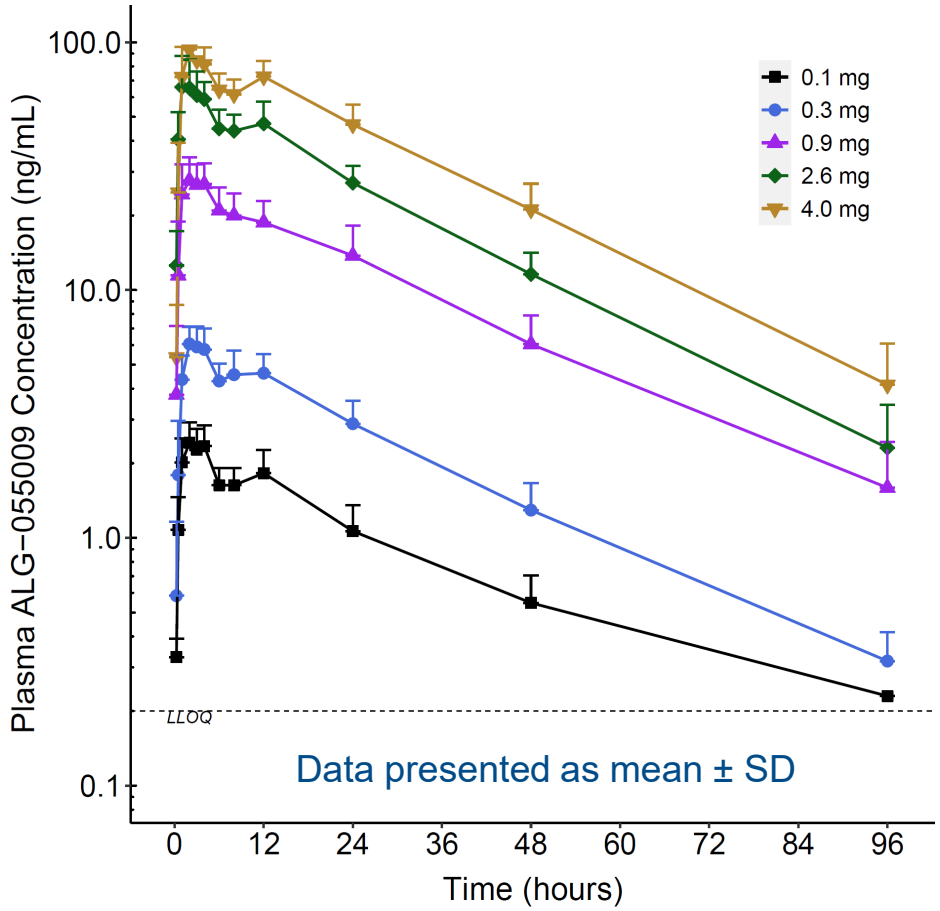
Study ALG-055009-301
Part 1: Single Ascending Dose - PK, Safety, Biomarkers

- 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



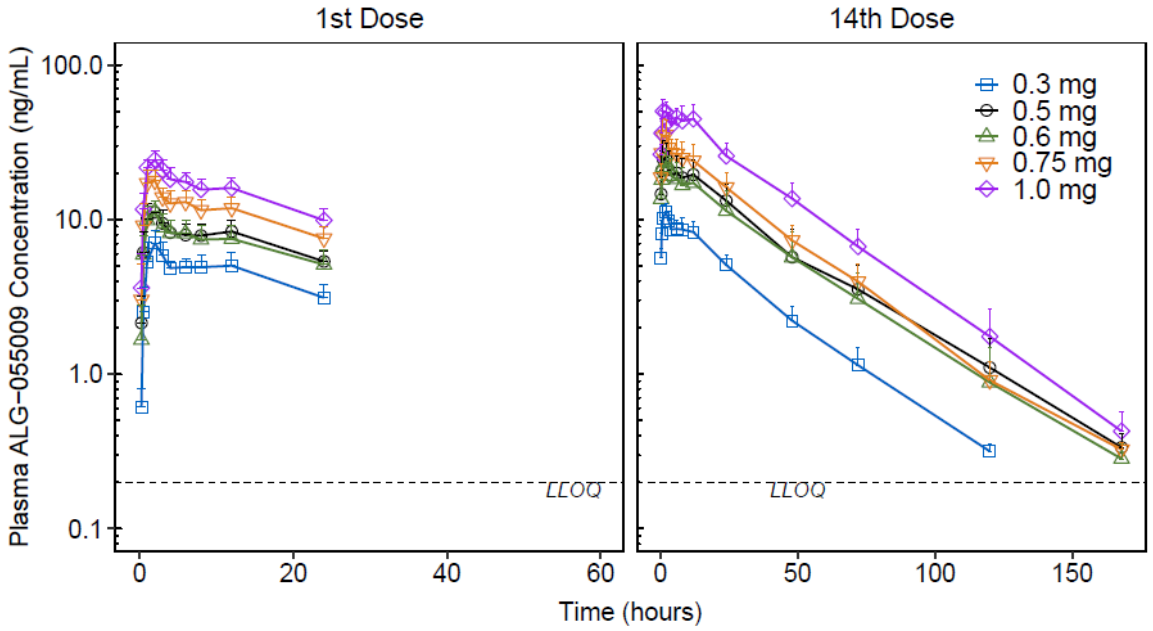
Single (≤4 mg) ALG-055009 doses well tolerated with favorable PK properties

ALIGOS THERAPEUTICS

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

Study ALG-055009-301
Part 2: Multiple Ascending Dose - PK, Safety

- Oral doses evaluated: 0.3, 0.5, 0.6, 0.75 and 1.0 mg QD x 14 days
- PK: favorable profile (dose-proportional, low variability (≤30%), 2x accumulation)
- Safety: well tolerated, no clinical evidence of hypo or hyperthyroidism
  - No SAEs, discontinuations
  - All TEAEs Grade ≤2
  - No concerning labs, ECGs, vital signs, physical examinations



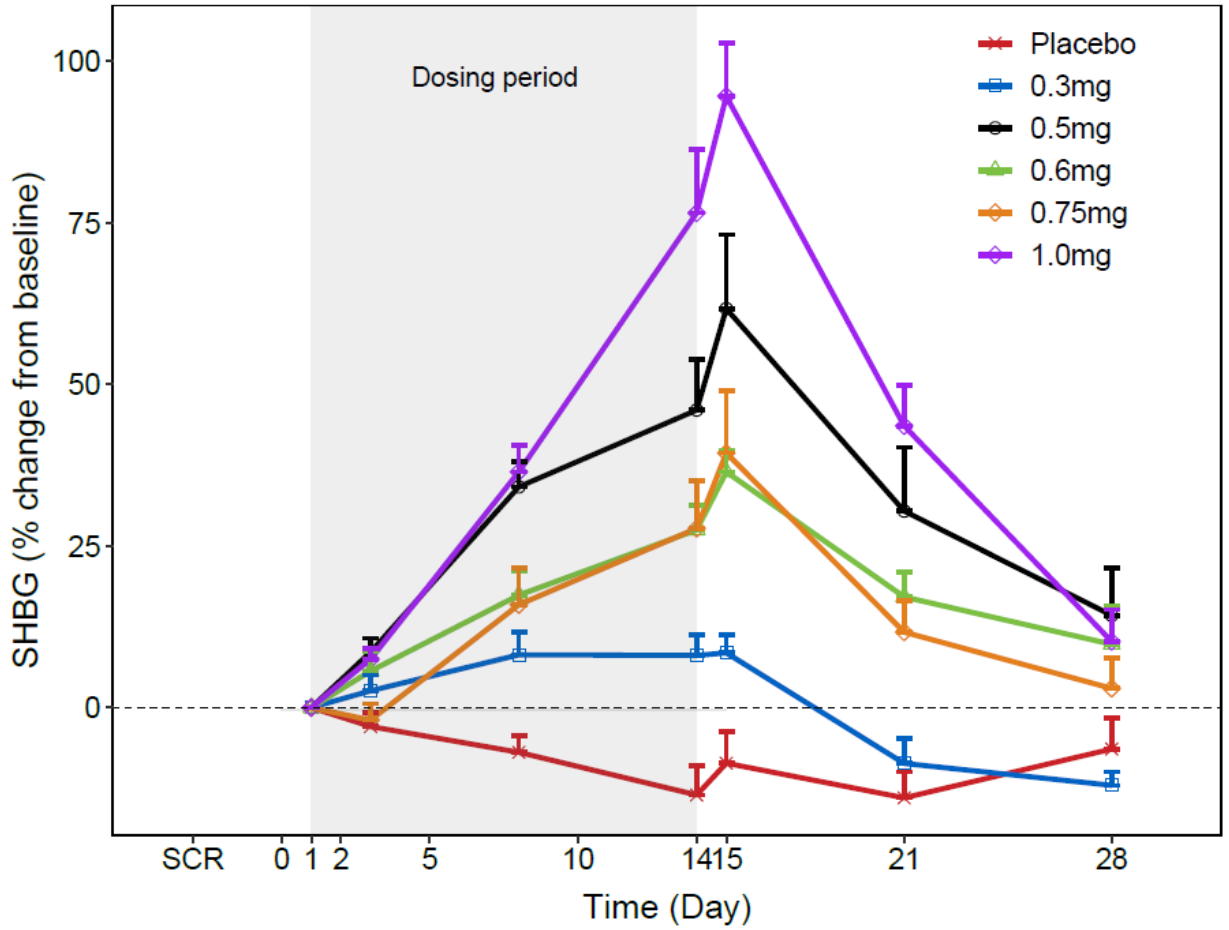
Data presented as mean ± SD

Multiple doses (≤1 mg) well tolerated with favorable PK

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

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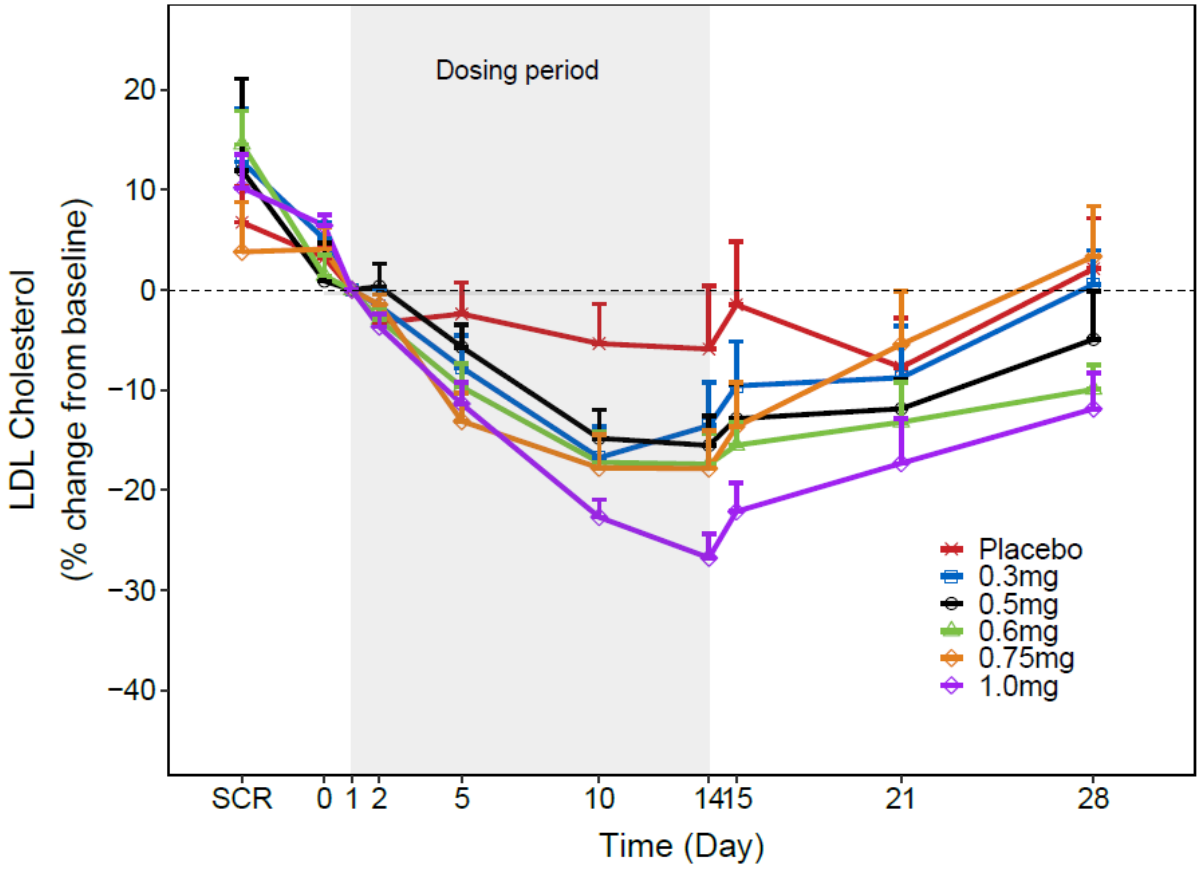
Charfi et al., EASL 2022.
clinicaltrials.gov; NCT05090111.

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



Dose responsive reductions in lipids (e.g., LDL, Apo-B, Triglycerides)

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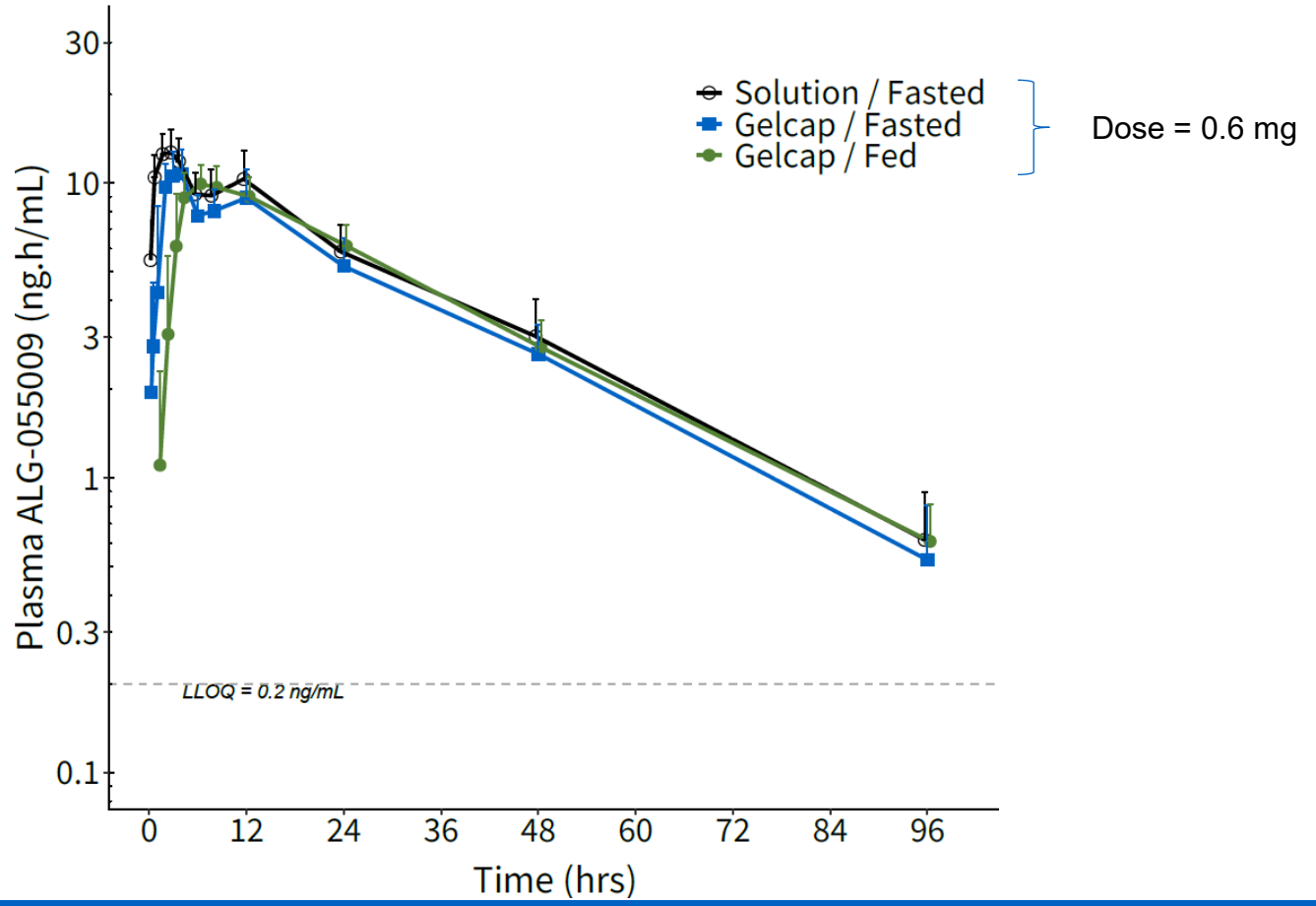
Charfi et al., EASL 2022.
clinicaltrials.gov; NCT05090111.

Formulation / Food Effect
Part 3: PK Data Comparison in Healthy Volunteers at 0.6 mg



Gel cap vs. liquid formulation: similar PK with low variability, no food effect
Phase 2 formulation (gel cap) confirmed

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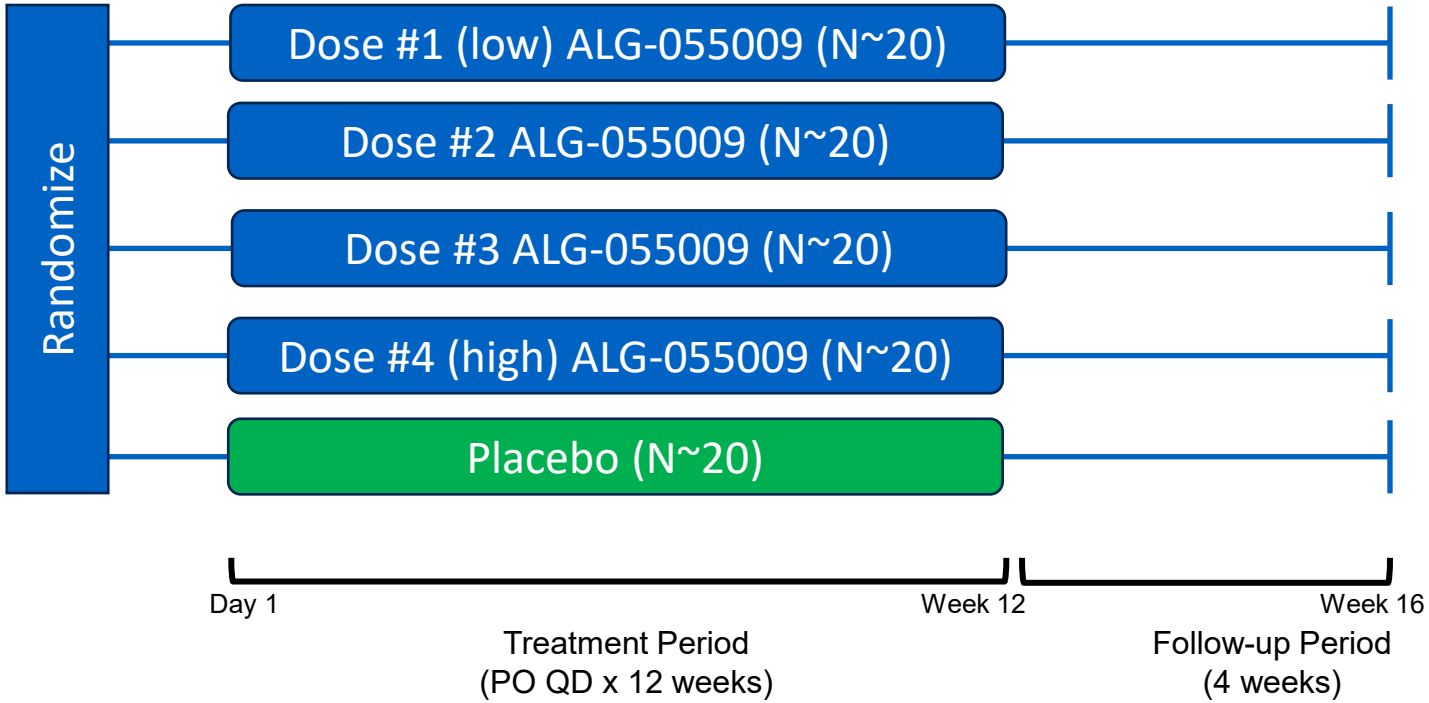
Charfi et al., AASLD 2023.
clinicaltrials.gov; NCT05090111.

ALG-055009
Preliminary Phase 2 Study Design



- Population: Adults subjects with MASH and liver fibrosis (F1-F3)
- Primary endpoint: Relative change in liver fat content by MRI-PDFF at Week 12
- Principal Investigator: Stephen Harrison, MD

Received IND clearance, Phase 2a filing Q4 2023, Topline data Q4 2024

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Proposed design subject to feedback from regulators and investigators.

ALG-055009
Summary

- Discovered by Aligos – issued US patent expires 2040
- THR-β agonist with high potency (EC_{50} = 50 nM) and β selectivity (3.8-fold)
- Phase 1
  - Safety – well tolerated without clinical safety signals
  - PK – favorable profile (linear, low variability) that is differentiated vs. resmetirom
  - Biomarkers – generally dose proportional increases in SHBG, decreases in lipids
  - Formulation – gel cap (Phase 2a) has similar PK to liquid formulation, no food effect
- US IND cleared
- Phase 2 dosing anticipated to start Q1 2024
- Phase 2 MRI-PDFF topline data anticipated Q4 2024

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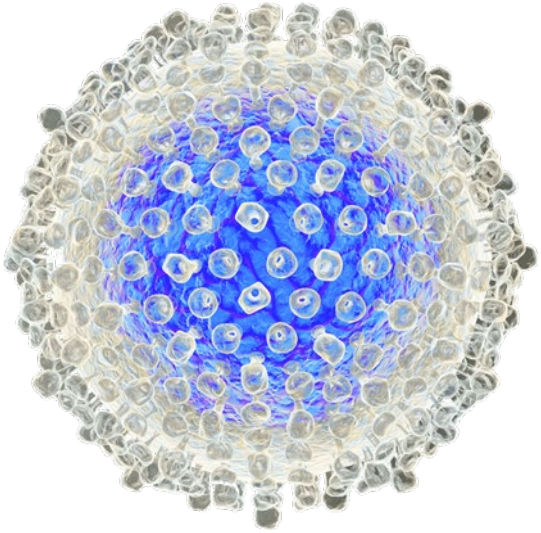
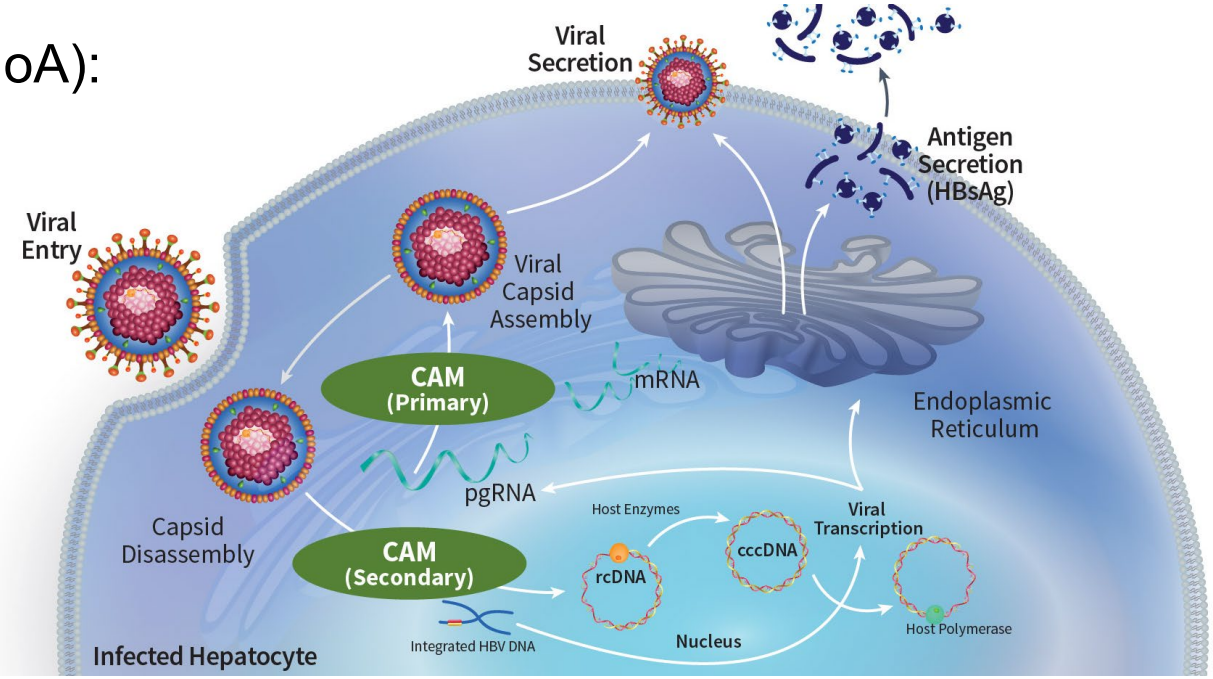
Chronic Hepatitis B



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

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



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

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

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ALG-000184
Superior Potency vs. Competitor CAM-Es

Compound	Current Status	HBV DNA reduction (EC_{50} nM)	Cell Type	
Aligos ALG-000184	**Phase 1**	**0.63**	**HepG2.117**	2nd Gen
		0.53	**HepG2.2.15**	
Assembly ABI-4334	Phase 1	1.2	AD38	
Assembly ABI-H3733	Partnered, BeiGene	5	AD38	
Enanta EDP-514	Phase 1	17	HepG2.115	1st Gen
Vebicorvir	Discontinued	172	AD38	
Janssen JNJ-6379	Discontinued	54	HepG2.117	
Arbutus AB-836	Discontinued	10	HepDE19	

ALG-000184 generally 10-300-fold more potent vs. other known CAM-Es
Exposures also enhanced via PK optimization strategies

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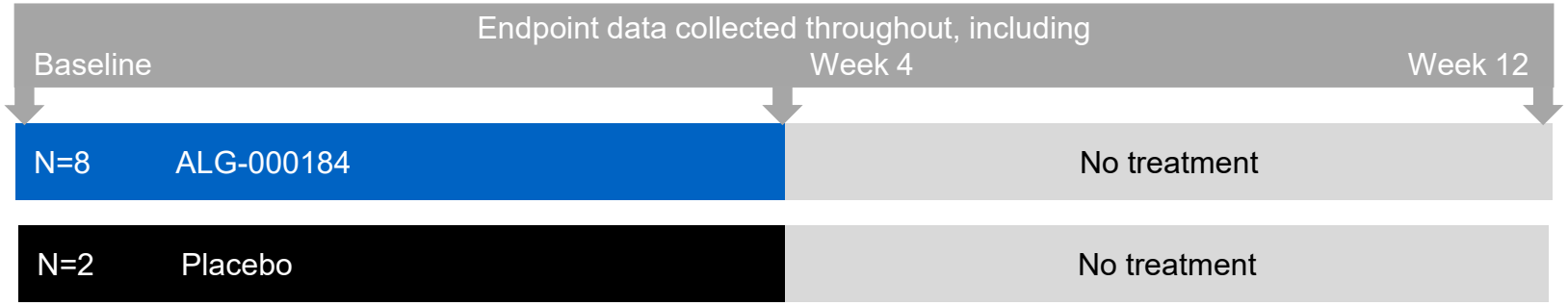
Data was sourced from publicly available literature, posters and presentations; ALG-000184 data was generated by Aligos on the parent compound ALG-001075.

ALG-000184-201
Phase 1 Study in HV (Parts 1& 2) and CHB Subjects (Parts 3-5)

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 (next slides)

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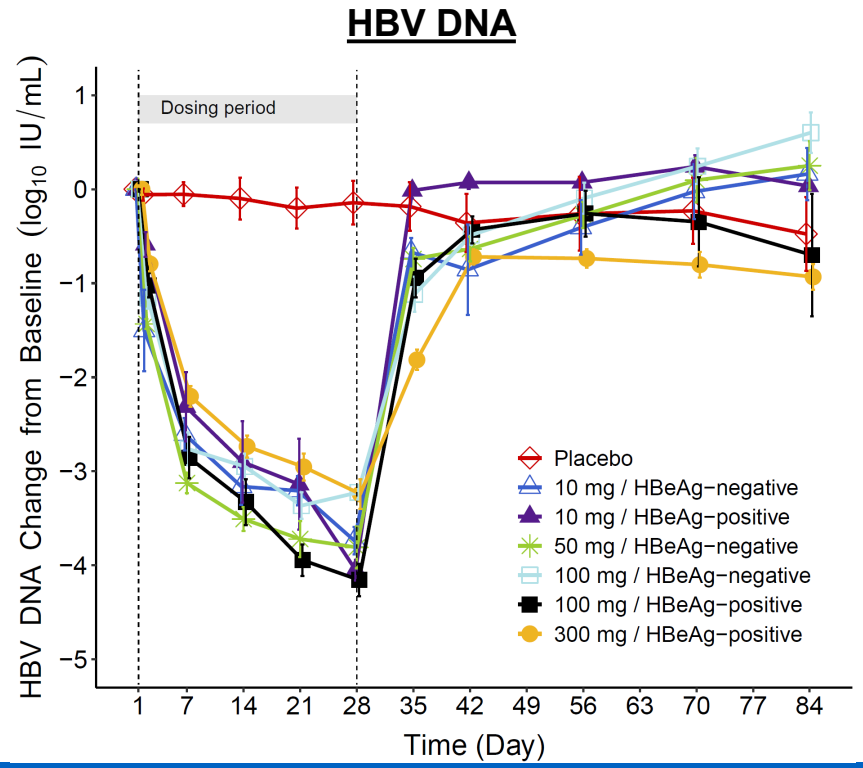
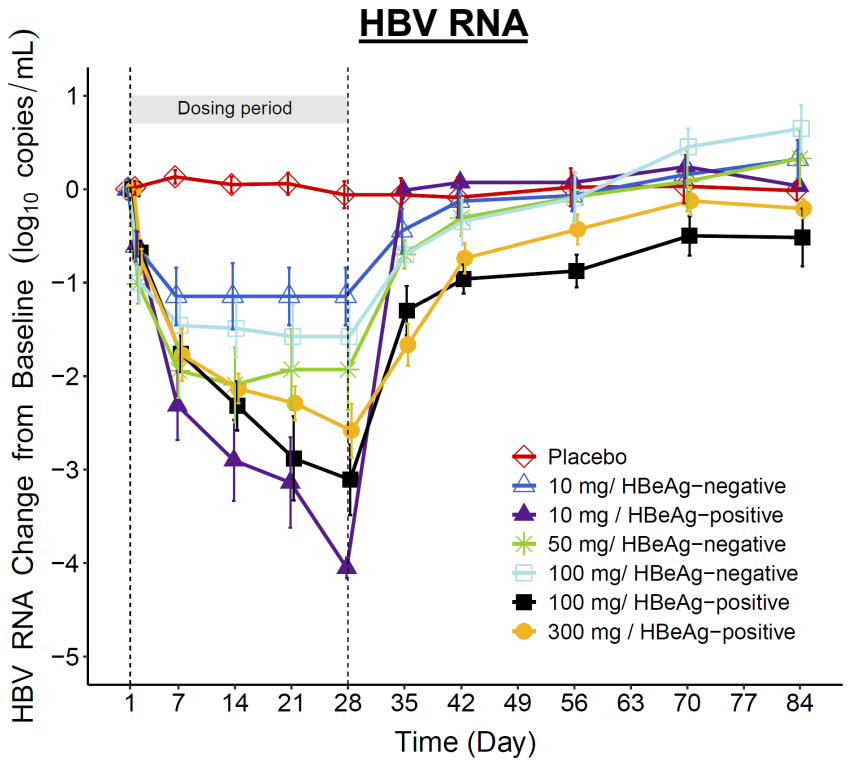
Gane Ed, APASL 2021; Gane Ed, HBV TAG 2021; Cohorts 1 & 2: Gane et al., AASLD 2021;
Cohort 4: Yuen MF. et al., APASL 2022; Cohort 3: Yuen MF. et al., EASL 2022; Clinicaltrials.gov: NCT04485663.

ALG-000184-201 - Part 3
Antiviral Activity Data in CHB Subjects

Mean (SEM) Serum HBV DNA* and HBV RNA Levels Change from Baseline Through the End of Study**





HBeAg- CHB subjects: Similar HBV DNA reduction for 10, 50 and 100 mg (~3-4 log_{10} IU/mL)
HBV DNA, HBV RNA <LLOQ in ≥75% and 100% of subjects, respectively

HBeAg+ CHB subjects: Potent HBV DNA, HBV RNA reductions observed (10 mg ≈ 100 mg ≈ 300 mg)

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*Roche Cobas® HBV DNA (LLOQ = 10 IU/mL).
**Roche Cobas® assay (LLOQ =10 copies/mL).
**China HBV RNA local assay (LLOQ=200 copies/mL).

ALG-000184 In Vivo
Superior Activity vs. Competitor CAMs (HBeAg Negative*)**

Drug Name	Most Advanced Status	Dose	HBV DNA	
			Mean Decline from BL to EOT (Log_{10} IU/mL)	% < LLOQ at Day 28
ALG-000184	**Phase 1**	**10 mg**	**3.7**	**100**
ABI-H0731[1,2]	Phase 2a	300 mg	2.5	25
JNJ-6379[3,4]	Phase 2	250 mg	2.7	56
EDP-514[5]	Phase 1b	200 mg	2.9	N/A
		800 mg	3.4	N/A
AB-836[6]	Phase 1	100 mg	3.1	N/A

10 mg ALG-000184 has more potent antiviral activity than competitor CAMs dosed at 100-800 mg

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

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

N/A – not available.
LLOQ (DNA) was ≤20 IU/mL for ABI and JNJ and 10 IU/mL for Aligos.
1. MF Yuen et al. Lancet Gastroenterol Hepatol 2019. 2. MF Yuen AASLD 2018.
3. Zoulim F., et al AASLD 2018. 4. Zoulim F., et al. Gastroenterology 2020.
5. MF Yuen, et al AASLD 2021 (Poster 823). 6. HepDart 2021 (preliminary data).

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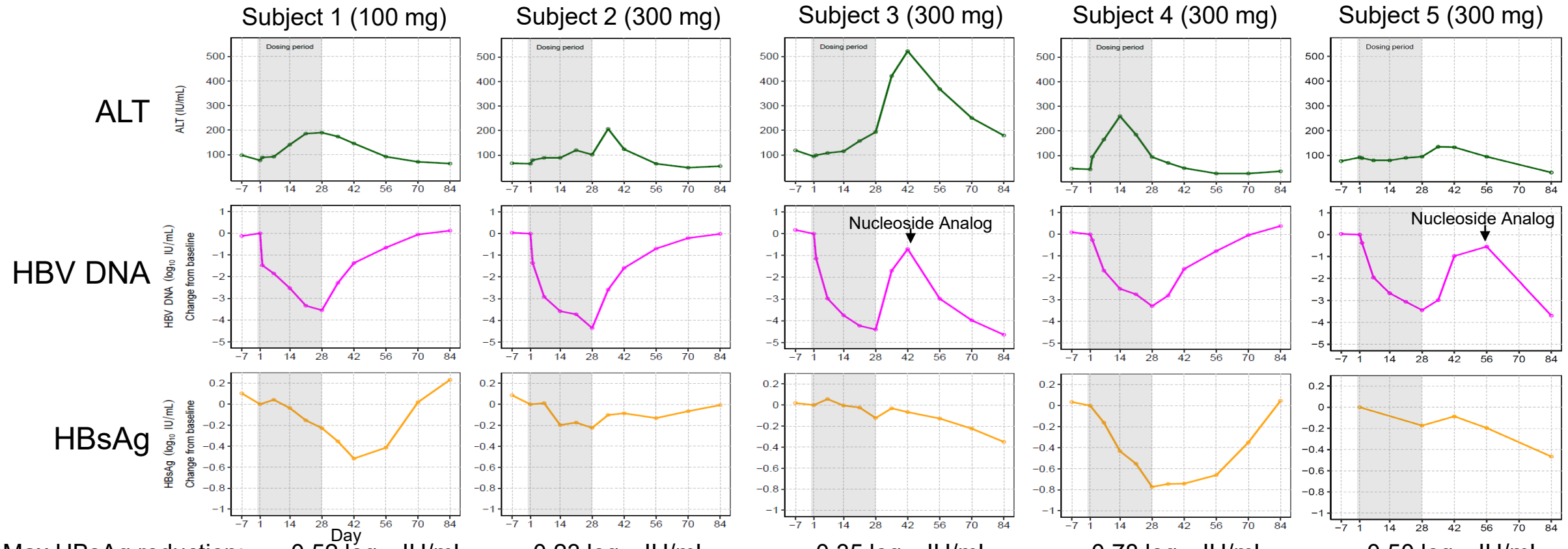
ALG-000184 - Part 3
HBsAg Reductions Observed in 28-Day Monotherapy (HBeAg+)



Max HBsAg reduction: 0.52 log_{10} IU/mL | 0.23 log_{10} IU/mL | 0.35 log_{10} IU/mL | 0.78 log_{10} IU/mL | 0.50 log_{10} IU/mL

300 mg ALG-000184 x 28 days results in 0.2-0.8 log_{10} IU/mL HBsAg decline in 50% of subjects
Best-in-class activity
Longer duration cohorts (± entecavir) ongoing (Parts 4-5)

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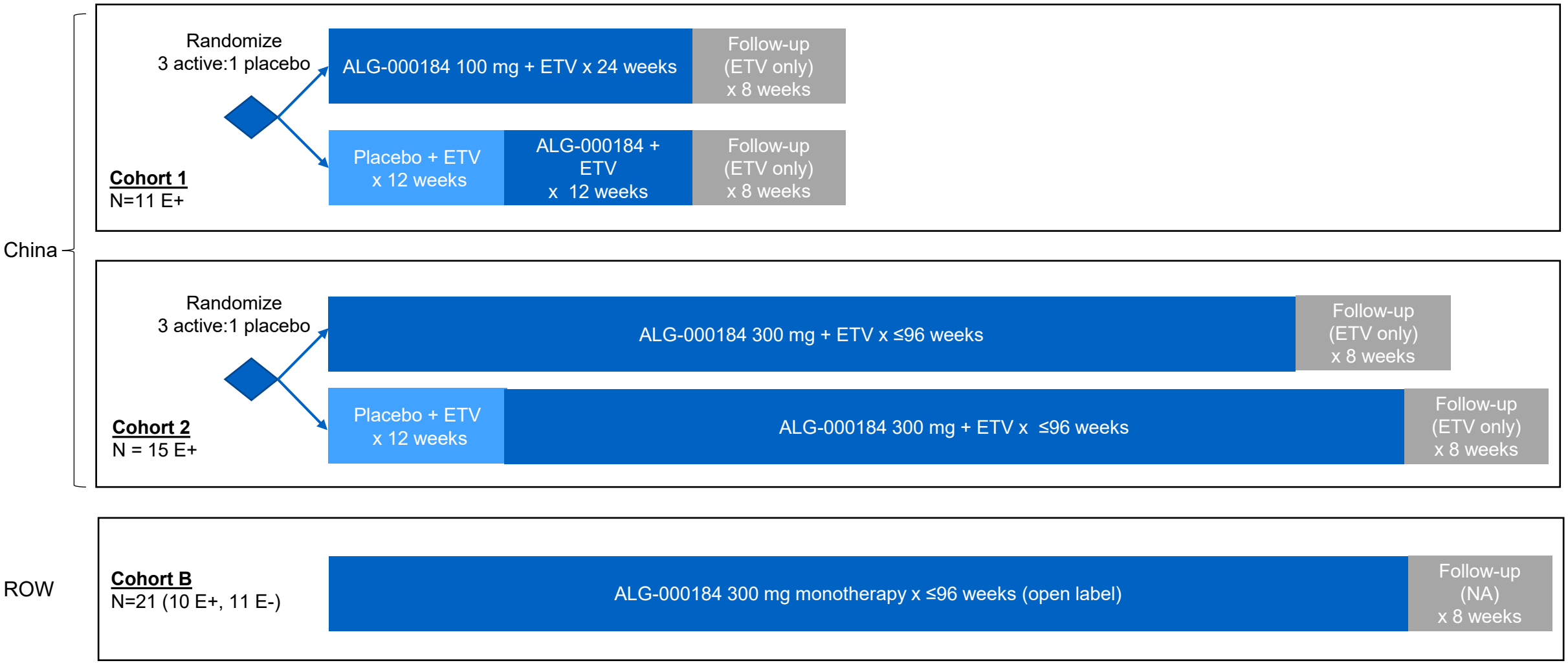
Hou et al., Poster #1329, AASLD 2022.
NCT04536337.

ALG-000184-201 - Part 4
Cohort Designs



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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.

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

Characteristic	P4C2 (n=15*)	P4CB (n=10)
Age, years, mean (SEM)	31.4 (3.3)	36.8 (2.9)
Male, n(%)	8 (53)	7 (70)
Asian, n(%)	3 (100)	9 (90)
BMI, kg/m^2, mean (SEM)	22.2 (0.8)	22.4 (0.8)
HBV Genotype, n(%)	B: 5 (33); C: 10 (67)	B: 5 (50); C: 4 (40); D: 1 (10)
HBV DNA, log_{10} IU/mL, mean (SEM)	8.1 (0.2)	8.0 (0.2)
HBV RNA, log_{10} copies/mL, mean (SEM)	6.7 (0.3)	5.3 (0.4)
HBsAg, log_{10} IU/mL, mean (SEM)	4.4 (0.2)	4.3 (0.1)
HBeAg, log_{10} PEI U/mL, mean (SEM)	2.4 (0.1)	2.6 (0.3)
HBcrAg, log_{10} U/mL, mean (SEM)	8.3 (0.3)	8.3 (0.2)
ALT, U/L, mean (SEM)	38.7 (5.2)	60.7 (11.7)

SEM: standard error of mean. *Three subjects prematurely discontinued due to non-safety related personal reasons (n=2) and non-compliance of dosing (n=1)

Baseline characteristics were comparable and typical for a HBeAg+ population
53% of subjects with normal baseline ALT levels in P4C2 compared to only 10% in P4CB

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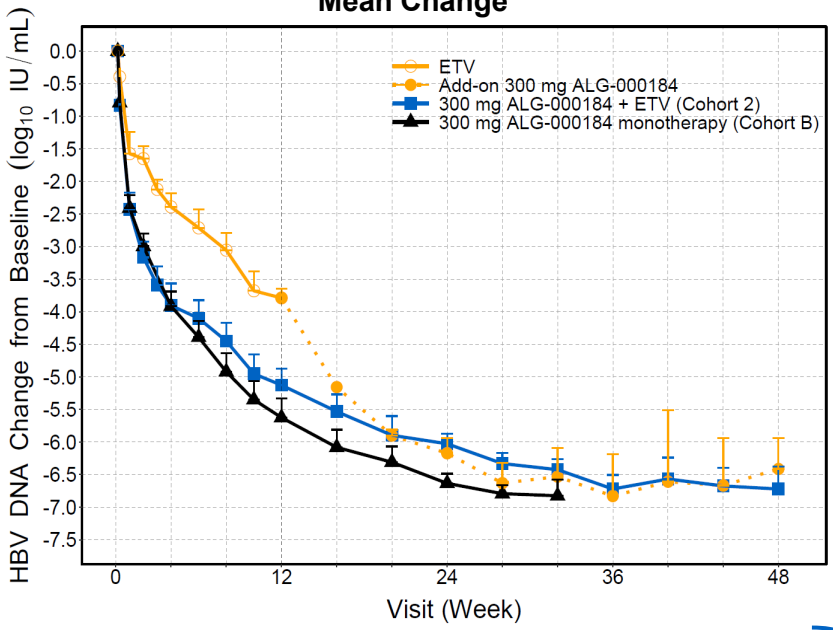
BMI = body mass index; SEM= standard error of the mean.
Yuen, M-F. et al., Late Breaker Poster #5028-C, AASLD (2023).

Antiviral Effect - Part 4 Cohort 2 & B
HBV DNA Change from Baseline

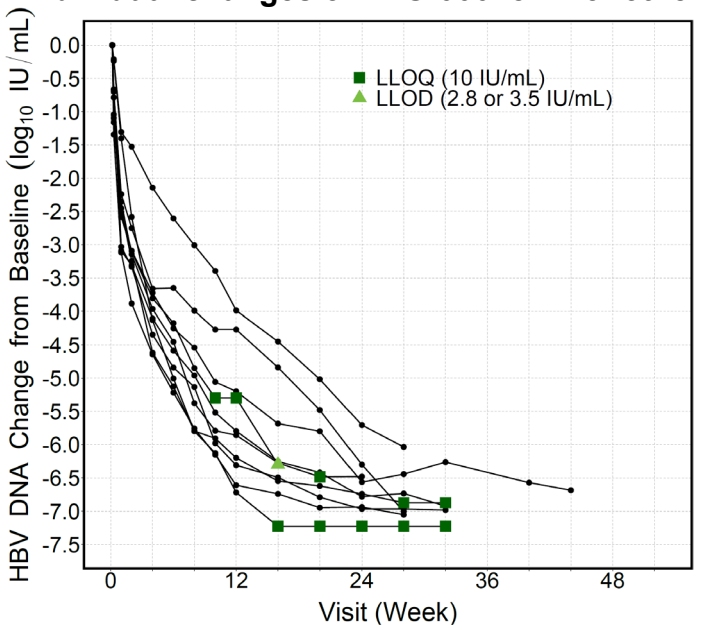
Mean Change



ETV	3	3			
Add-on 300 mg ALG-000184		3	3	3	2
300 mg ALG-000184 + ETV (Cohort 2)	11	9	9	8	5
300 mg ALG-000184 monotherapy (Cohort B)	10	10	9		

Number of subjects at timepoint

Individual changes of ALG-000184 monotherapy



<u>300 mg ALG-000184±ETV</u>

- Showed greater HBV DNA reduction than ETV monotherapy
- Achieved similar DNA reductions

No viral breakthrough was observed in ALG-000184 monotherapy x ≤44 weeks

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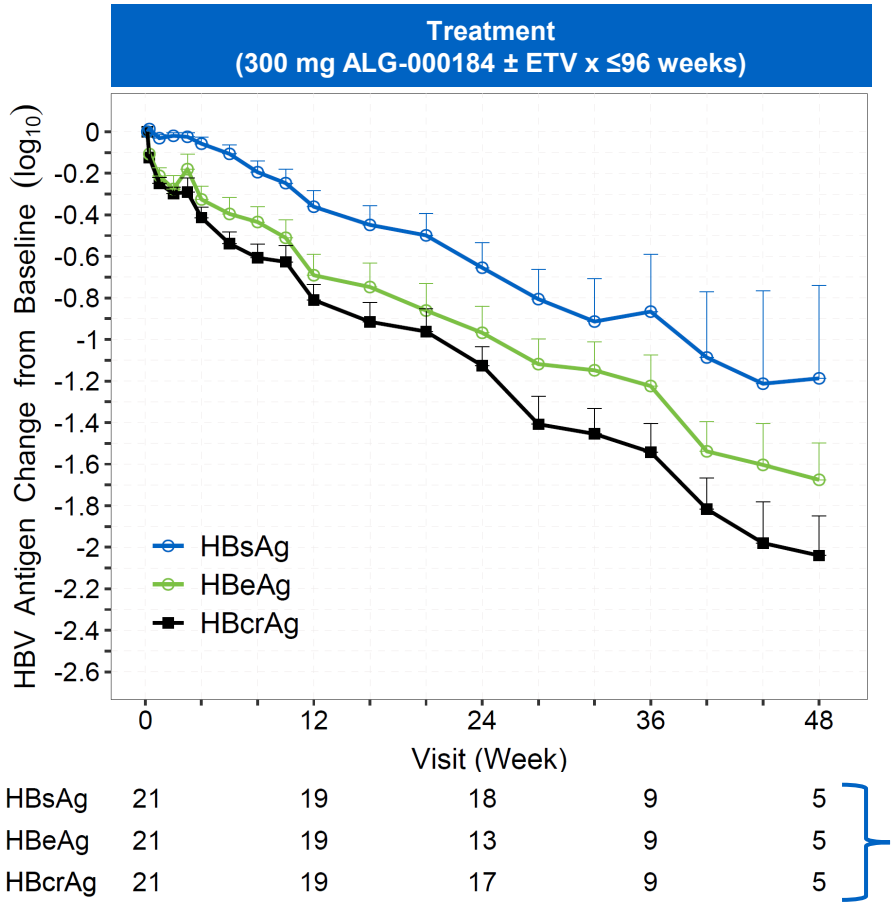
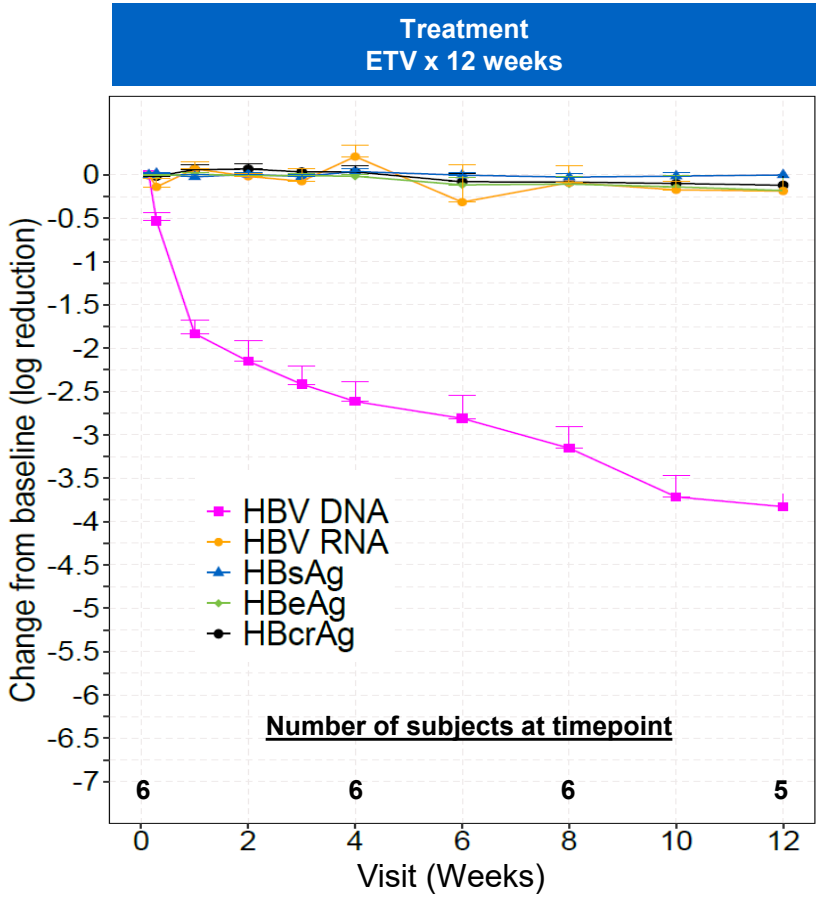
ALG-000184-201 - Antiviral Effect: Part 4 Cohort 2 & B
HBV Antigen Change from Baseline





Substantial HBsAg, HBeAg, and HBcrAg reductions seen with combo
Max declines: 2.0, 2.1 and 2.5 log_{10}, respectively
ETV alone only impacted HBV DNA; no impact on HBV RNA or antigens

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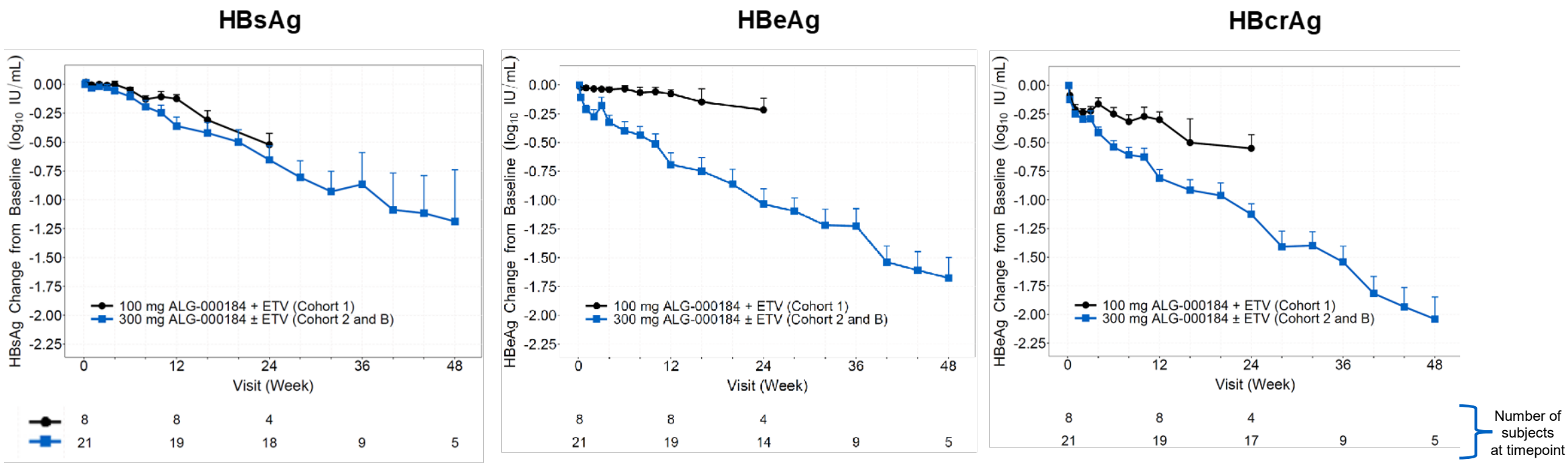
Graph plots subjects initially randomized to ALG-000184 ± ETV and were compliant (confirmed by PK)
Yuen, M-F. et al., Late Breaker Poster #5028-C, AASLD (2023).

ALG-000184-201 - Antiviral Effect: Part 4 Cohorts 1 vs. 2
HBV Antigen Change from Baseline



ALG-000184 treatment results in dose-dependent HBsAg, HBeAg and HBcrAg declines

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Yuen, M-F. et al., Late Breaker Poster #5028-C, AASLD (2023).

Safety Overview
Treatment Emergent Adverse Events

300 mg ALG-000184 +/- ETV	Part 4 Cohort 2 (n=15)	Part 4 Cohort B (n=10)
Serious Adverse Events (SAEs)	None	
Treatment emergent AEs (TEAEs) leading to study drug discontinuation	None	
Subjects with Grade ≥3 TEAEs	2 ALT/AST↑ (n=2) neutropenia↑ (n=1)*	3 ALT/AST↑
Concerning TEAE, laboratory, ECG, vital sign, or physical examination findings or trends	None	

- Neutropenia considered probably related to an acute upper respiratory infection and resolved following resolution of this infection in the setting of continued dosing with study drug
- Five Grade ≥3 TEAEs of liver transaminase elevations were transient, resolved on study drug, and were associated with decline in antigens. None were considered clinically concerning by the AFC.

A favorable safety profile was observed in untreated HBeAg+ CHB subjects with long term (≤ 48 weeks) treatment with 300 mg ALG-000184 ± ETV

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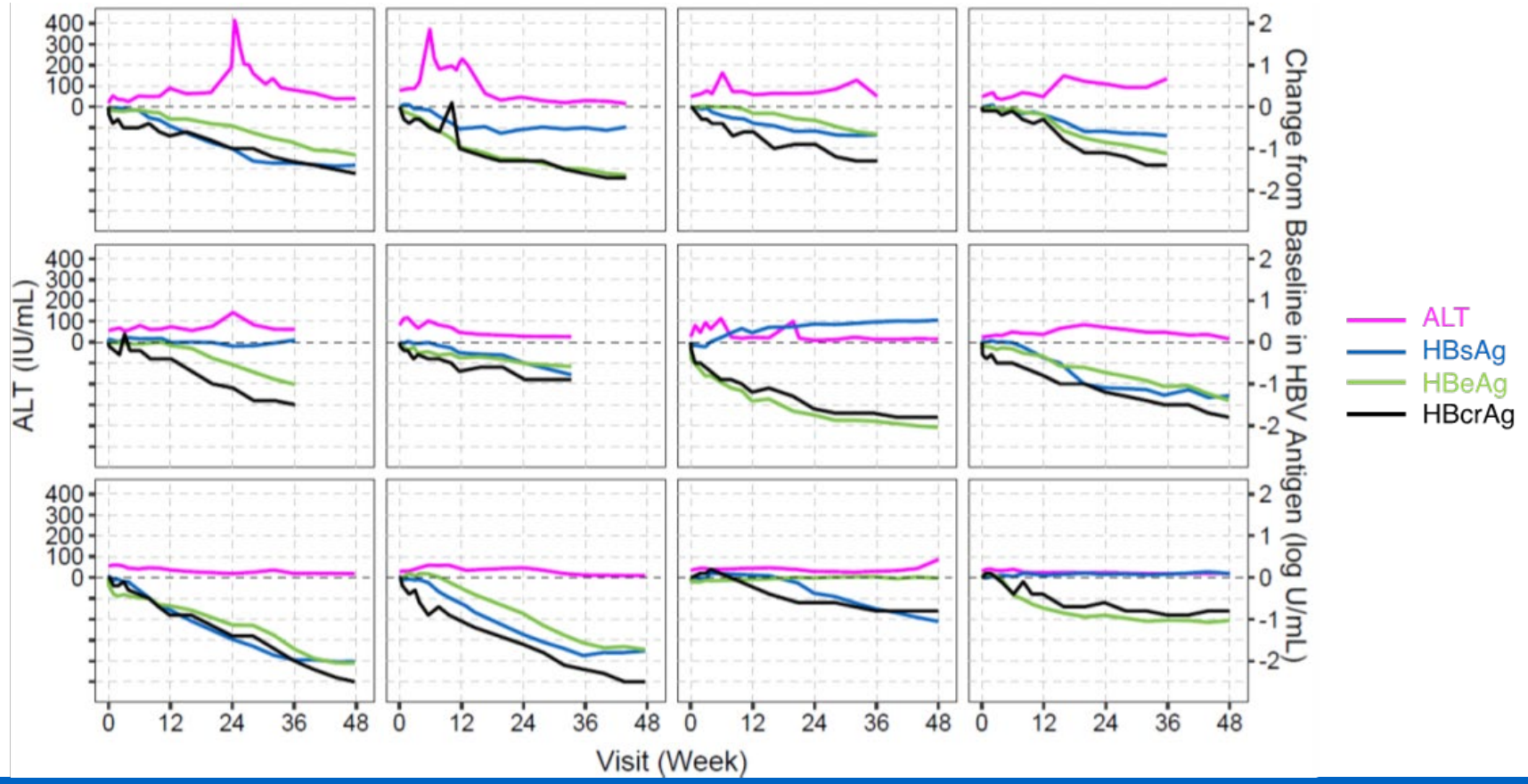
Yuen, M-F. et al., Late Breaker Poster #5028-C, AASLD (2023).

Antiviral Effect

ALT, HBsAg, HBeAg and HBcrAg Over Time by Subject – P4C2

Part 4 Cohort 2
(300 mg ALG-000184 + ETV x ≤96 wks)



All subjects (n=12) observed HBcrAg declines after receiving 300 mg ALG-000184 + ETV x ≥ 12 weeks
11/12 had HBeAg declines and 9/12 had HBsAg reductions
ALT flares seen in context of antiviral activity (antigen declines); resolve while continuing ALG-000184

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Yuen, M-F. et al., Late Breaker Poster #5028-C, AASLD (2023).

ALG-000184
Multiple Development Paths Available

Endpoint	Affected Viral Markers	Approvable Endpoint* In
Functional Cure	HBsAg, DNA	US, EMEA, China
Partial Cure	HBsAg, DNA	TBD
DNA Suppression	DNA	US, EMEA, China (Used for tenofovir alafenamide (TAF) approval)

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*Endpoints defined in regulatory guidance from: FDA (2022), EMEA (2006), and China FDA (2023).

Executive Summary
ALG-000184

- Oral dosing with 300 mg ALG-000184 ± ETV x ≤48 weeks in untreated HBeAg+ CHB subjects results in:
  - A favorable safety profile
  - Greater suppression of HBV DNA and RNA vs. ETV alone (1st MOA)
  - No viral breakthrough when ALG-000184 is given as monotherapy x ≤44 weeks
  - Multi-log reductions in HBsAg, HBeAg and HBcrAg, which appear to be mediated by ALG-000184 (2nd MOA)
- ALG-000184 appears to lower cccDNA levels
- ALG-000184 may play an important role in enhancing rates of chronic suppression (superior/non-inferior to NAs) and functional cure
- Dosing is ongoing in 96-week cohorts
- Phase 2 enabling activities are underway

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Executive Summary
Aligos Advancing Multiple Promising Drugs in Areas of Unmet Need

- MASH
  - ALG-055009 (THR-β agonist) - enhanced PK, pharmacology vs. competitor THR-β drugs; US IND cleared
  - Phase 2a study with ALG-055009 (12-week MRI-PDFF) - filing Q4 2023, topline data anticipated Q4 2024
  - Oligonucleotide efforts (including Merck collaboration) are progressing
- ALG-000184 (CAM-E) - best in class reductions in HBsAg, HBeAg, HBcrAg, HBV DNA and RNA
  - Up to 2 $\log_{10}$ IU/mL HBsAg reductions observed in HBeAg+ untreated CHB subjects
  - Additional dosing, cohorts ongoing
  - Phase 2 enabling activities underway
- Coronavirus Protease Inhibitor (ALG-097558)
  - Currently dosing; topline data expected H1 2024; seeking external funding to further advance
- As of September 30, 2023: cash, cash equivalents and investments was $70.4M*
- The Company believes our cash, cash equivalents and investments, including the expected net proceeds from the private placement, will provide sufficient funding of planned operations through the end of 2025

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*Reflects unrestricted cash, cash equivalents and investments, not including the expected net proceeds from the $92M private placement financing announced on October 23, 2023. This balance also does not include September 2023 NIAID Contract (~$8.5M USD) for various ALG-097558 Phase 2 enabling studies which began in Q4 2023 and will run over two years.


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