ALIGOS THERAPEUTICS

CORPORATE PRESENTATION

April 2026

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, ability of our collaborators to successfully develop and commercialize our licensed drug candidates, 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-K filed with the Securities and Exchange Commission on March 5, 2026, 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

Candidate	Indication	MOA	Discovery	Nonclinical	Phase 1	Phase 2	Phase 3	Partner
pevifoscorvir sodium (pevy)[1,3]	Chronic HBV Infection *Monotherapy*	CAM-E						Amoytop Biotech *Greater China*
ALG-055009	MASH, Obesity	THR-β Agonist						
ALG-097558	Covid-19*	Protease Inhibitor						NIH National Institute of Allergy and Infectious Diseases; agile Coronavirus Drug Testing Initiative; KU LEUVEN
ALG-170675[2]	Chronic HBV Infection	ASO						Amoytop Biotech *Greater China*
TBD	Hepatitis Delta Virus	ASO						

*Our Covid-19 protease inhibitor programs are partly funded ($13.8M 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. HBV = hepatitis B virus; CAM-E = capsid assembly modulator (empty); Covid-19 = coronavirus disease of 2019 (SARS-CoV-2); MASH = metabolic dysfunction-associated steatohepatitis; THR-β = thyroid hormone receptor beta. ASO= antisense oligonucleotide. All timelines are approximate and subject to change based on enrollment and operational considerations. [1]Pevifoscorvir sodium formerly known as ALG-000184. [2]Amoytop has rights in China, Taiwan, Hong Kong, and Macau. [3]Amoytop has rights in China, Taiwan, Hong Kong, and Macau.

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As of 12/31/25 - Cash, cash equivalents and investments were $77.8M. Projected runway into Q4 2026, inclusive of the $25M upfront from the Amoytop Greater China License

CHRONIC HEPATITIS B VIRUS INFECTION

OVERVIEW

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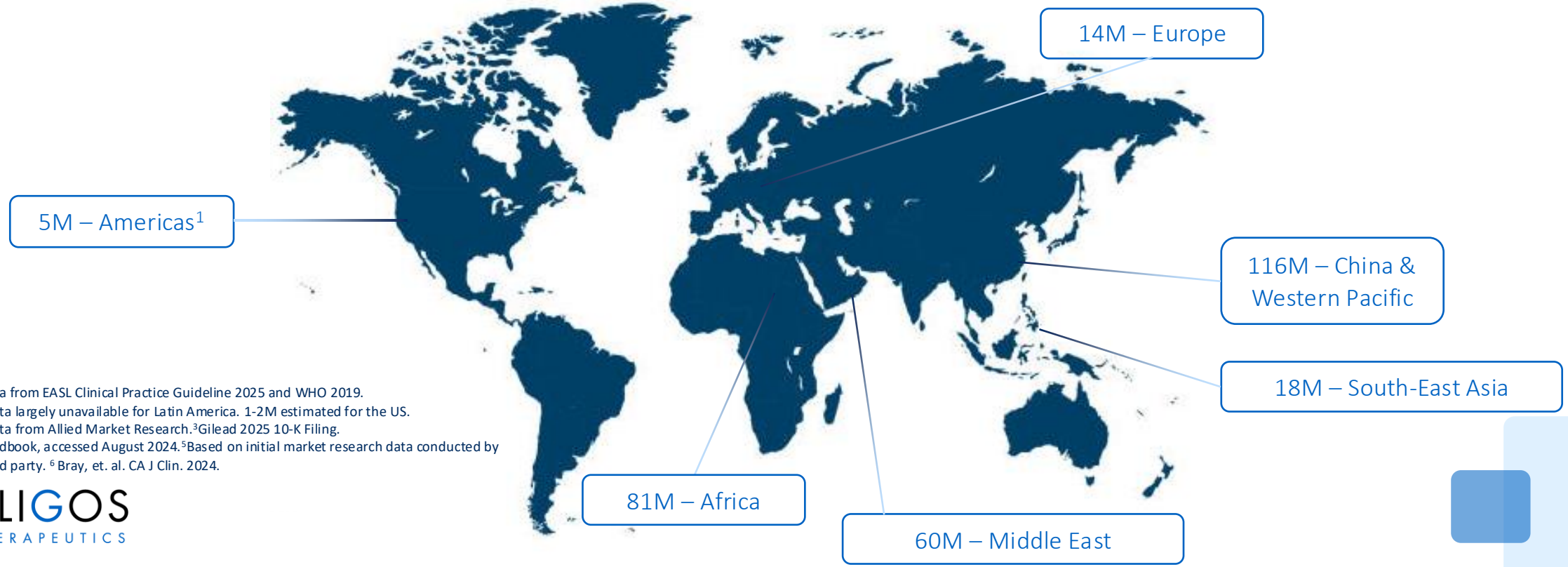
Chronic Hepatitis B Virus Infection

High Unmet Medical Need

- ~254M people worldwide living with HBV with 1.2M new infections annually in 2022
- 1.08M deaths per year, mostly from cirrhosis and hepatocellular carcinoma; Primary cause of liver cancer worldwide
  - Liver cancer is the 6th most diagnosed form of cancer worldwide, but results in the 3rd most deaths of all cancers[6]
- Opportunity estimated at $6.2B by 2031[2]; Gilead HBV sales of ~$1.1B in 2025[3] at ~$17k/year[4] for continuous therapy
- Pevifoscorvir sodium has the potential for favorable pricing and payor coverage due to its potential to improve patient outcomes[5]



Data from EASL Clinical Practice Guideline 2025 and WHO 2019.
[1]Data largely unavailable for Latin America. 1-2M estimated for the US.
[2]Data from Allied Market Research.[3]Gilead 2025 10-K Filing.
[4]Redbook, accessed August 2024.[5]Based on initial market research data conducted by third party. [6] Bray, et. al. CA J Clin. 2024.

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Current Treatment Options

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

NAs

- Standard of care for 25+ years
  - Indicated for chronic suppression
    - Monotherapy
    - 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
    - Combination
    - 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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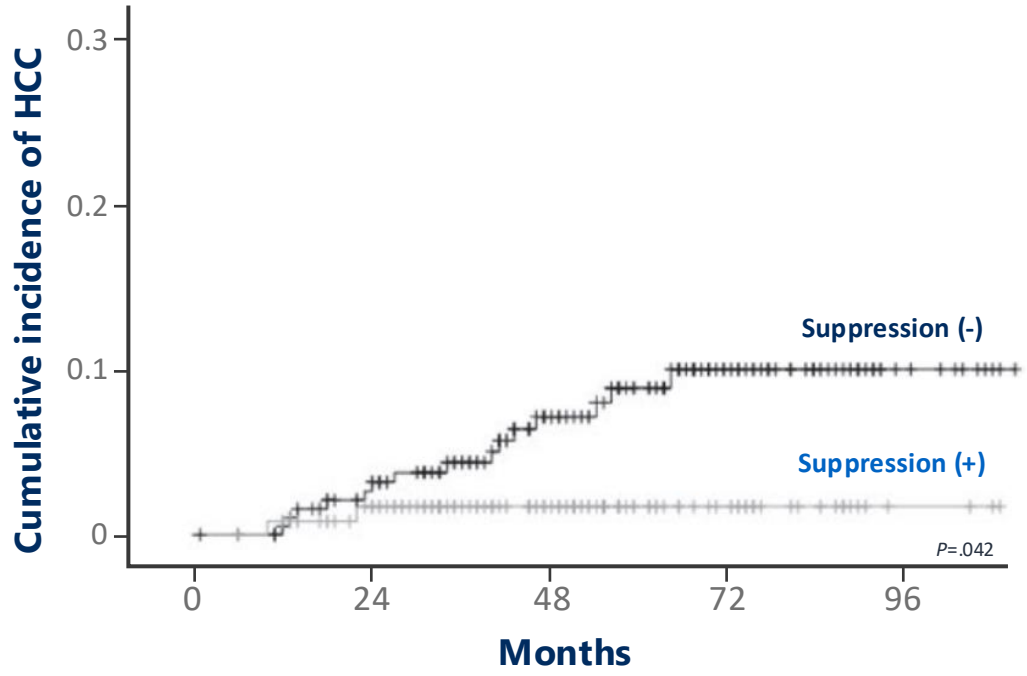
[1]Wong et. al., J Hepatology 2021. [2] Kim et. al., AASLD 2024.

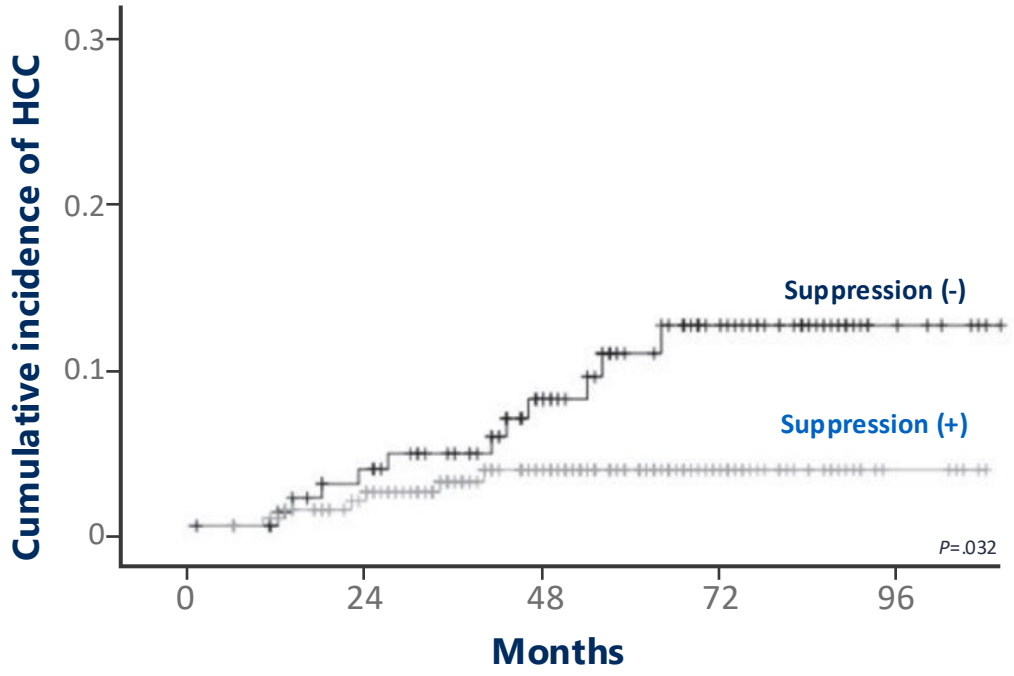
Complete & Faster HBV DNA Suppression Improve Clinical Outcomes

Viral suppression at 1 year



Suppression (-)	201	177	122	54	9
Suppression (+)	124	108	61	23	3

Viral suppression at 2 years



Suppression (-)	123	106	73	38	7
Suppression (+)	202	179	110	39	5

[a] Defined suppression level as 12 IU/mL.[b] Retrospective cohort study of 325 HBeAg-positive patients with chronic HBV infection on antiviral therapy between 2007-2013 in Seoul, Korea. HBeAg, hepatitis B e antigen; HBV DNA, hepatitis B virus DNA; HCC, hepatocellular carcinoma. Nam JY, et al. *J Viral Hepat*. 2018;25(5):552-560.

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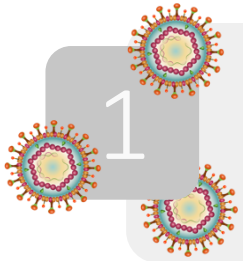
Cumulative HCC and cirrhosis incidence was significantly lower in patients who achieved complete viral suppression[a] within 1-2 years compared with patients who failed to achieve viral suppression within these periods[b]

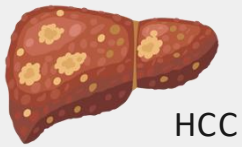
HBV Pathogenesis: The Three Drivers That Lead to End Stage Liver Disease/Cancer



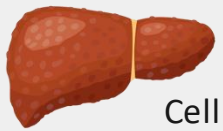
REPLICATION

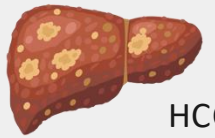

Inflammation

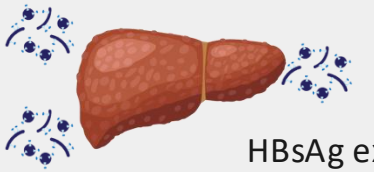

Fibrosis/cirrhosis


HCC

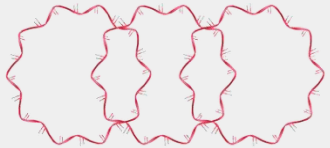
INTEGRATION

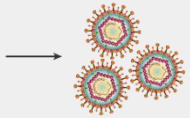

Cell death


HCC


HBsAg expression → immune suppression

RESERVOIR


cccDNA


HBV DNA


HBV RNA


HBsAg


HBcrAg


HBeAg

Maintenance of viral lifecycle and infection

Effective HBV therapies must address all three drivers: replication, integration, and reservoir

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Pevifoscorvir Sodium

Small Molecule CAM-E

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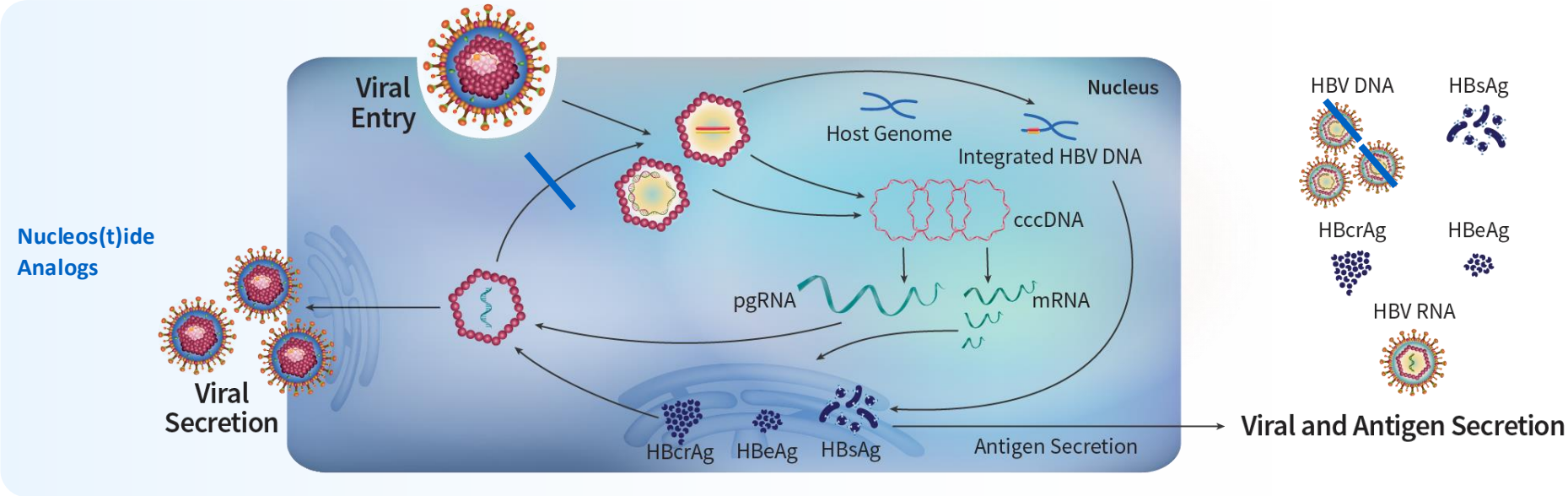
MOA: NAs



Key Attributes

- NAs improve clinical outcomes by controlling HBV replication through targeting HBV polymerase activity, **though they do not**[1,2]**:**
  - Affect cccDNA[2,3]
  - Substantially lower HBsAg and HBcrAg[3]
  - Completely inhibit rcDNA synthesis from pgRNA[1]
  - Lead to HBeAg seroconversion above the spontaneous rate[4]

1. Martinez MG, et al. *J Hepatol*. 2021;75(3):706-717. 2. Boyd A, et al. *J Hepatol*. 2016;65(4):683-691. 3. Broquetas T, Carrión JA. *Hepat Med*. 2022;14:87-100. 4. Xing T, et al. *PLoS One*. 2017;12(1):e0169444.

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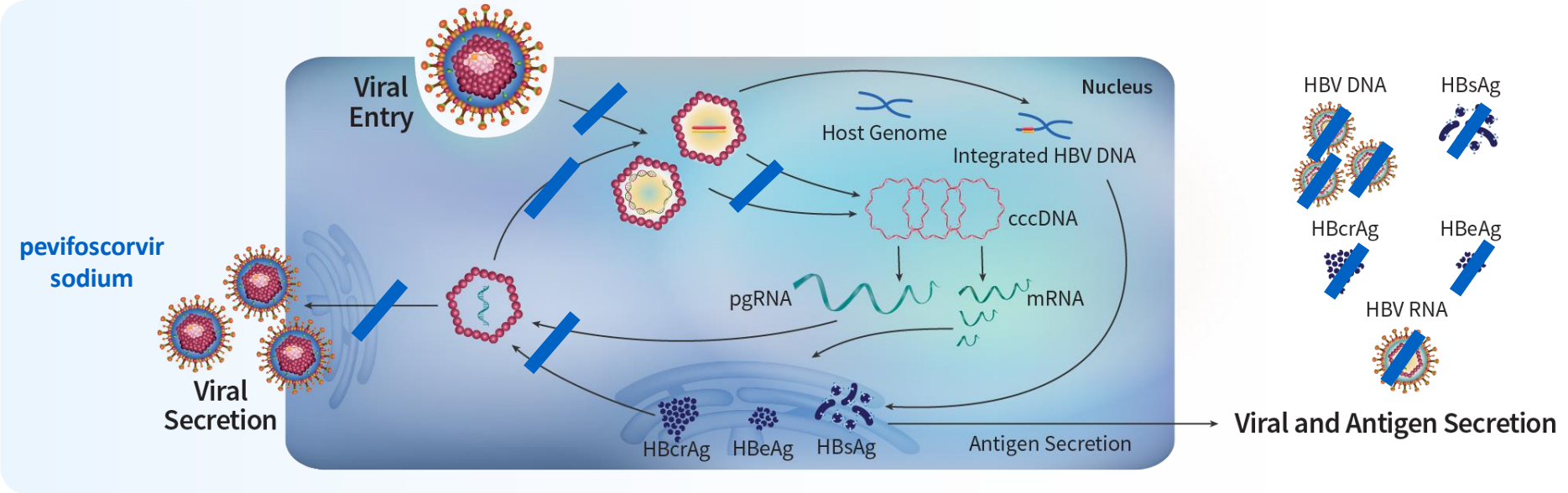
Pevifoscorvir Sodium (CAM-E): A Paradigm Shift in the Treatment of HBV

Suppressing the Three Drivers of Liver Disease/Cancer



The three drivers of chronic HBV infection that can lead to cirrhosis and/or HCC:

	1 REPLICATION	2 INTEGRATION	3 RESERVOIR
Pevifoscorvir sodium	✓	✓	✓
Nucleos(t)ide Analogs	✓	X	X

Yuen, et. al, AASLD 2025. Verheyen, et. al, AASLD 2025.

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Pevifoscorvir Sodium: A Favorable Profile

In vitro: potency vs. first generation CAM-Es

Compound	Current Status	HBV DNA reduction (EC_{50} nM)	Cell Type	
Aligos pevifoscorvir sodium[1]	**Phase 2**	**0.63**	**HepG2.117**	2nd Generation
		0.53	**HepG2.2.15**	
Assembly ABI-4334	Phase 1	1.2	AD38	
Assembly ABI-H3733	Partnered (BeOne) – Discontinued	5	AD38	
Enanta EDP-514	Phase 1 – Seeking Collaboration	17	HepG2.115	1st Generation
Assembly ABI-H0731 (vebicorvir)	Discontinued	172	AD38	
Janssen JNJ-56136379 (bersacapavir)	Discontinued	54	HepG2.117	
Arbutus AB-836	Discontinued	10	HepDE19	

Data was sourced from publicly available literature, posters and presentations; No head-to-head clinical trials have been conducted. Caution should be exercised when comparing data across experiments. [1]ALG-000184 data was generated by Aligos on the parent compound ALG-001075. Studies conducted in vitro.

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Pevifoscorvir Sodium (ALG-000184)

A Potential Best-in-Class CAM-E for Chronic Hepatitis B Virus Infection

- **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 (10 mg) achieved maximum HBV DNA reductions; more potent antiviral activity than competitor CAMs[2]
  - **MOA:** Evidence of second mechanism seen at 300 mg monotherapy dose through reductions in HBsAg
    - The only CAM to date that has demonstrated HBsAg reduction at 28 days
- **Phase 1:** 96-week study completed
- **Phase 2 study enrolling patients:** Enrollment ongoing in HBeAg+ with 103 participants enrolled as of April 2026; 74 participants enrolled in the HBeAg- cohort as of April 2026 with expected completion in 2H2026
- **Recently granted FDA Fast Track Designation**

[1] Not including any patent term extension. [2] Head-to-head data seen in preclinical context.

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SUPREME – Phase 2 Study Design

Primary Analysis at 48 Weeks; Extension Period Analysis at 96 Weeks; Interim Readouts Planned

Primary Endpoint: HBV DNA <LLOQ (10 IU/mL, **target detected *or* target not detected**) at Week 48

Primary Endpoint: HBV DNA <LLOQ (10 IU/mL, **target not detected**) at Week 48

Part 1b will be a liver biopsy sub-study inclusive of n≈12. Part 2b will be a liver biopsy sub-study inclusive of n≈12. pevy = pevifoscorvir sodium.

As of April 2026: 103 participants enrolled in the HBeAg+ Part 1a;

74 participants enrolled in the HBeAg- Part 2a

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B-SUPREME HBeAg- Interim Analysis at Week 12

Overview	Finding from the interim analysis
• The study design for the Phase 2 B-SUPREME study includes sample size re-estimations for both Parts 1a and 2a to ensure sufficient power to evaluate a statistically significant treatment effect at the primary endpoint. • The first pre-specified interim analysis of the Phase 2 B-SUPREME study was performed after approximately 60% HBeAg- participants (N=34, Part 2a) reached Week 12 or later. • In addition, safety data was reviewed for all participants enrolled in the study (N=174) at the time the analysis was performed.	– The DSMB recommended increasing the sample size of Part 2a from 74 currently enrolled to 100 participants. – A futility analysis was performed; the prespecified futility criteria was not met, per the statistical analysis plan. – The study drugs were well-tolerated with no clinically concerning laboratory, vital sign, or ECG abnormalities. – No viral break through related to study drugs has been observed in the study to date.

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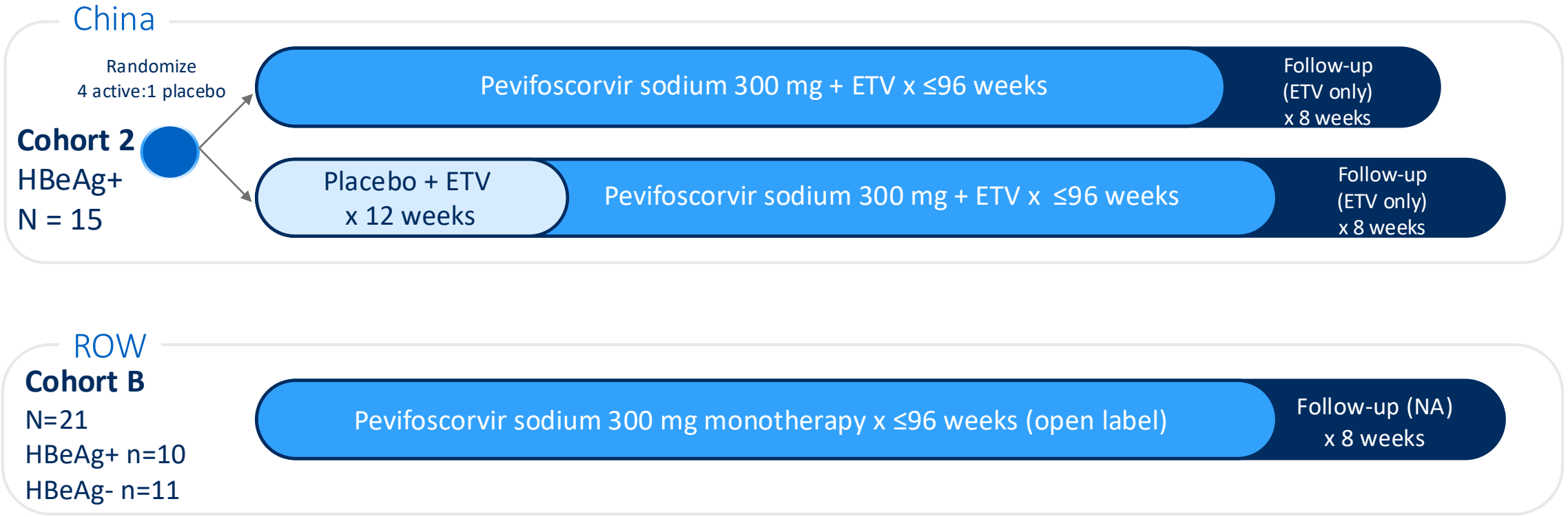
ALG-000184-201, A Multi-Part Phase 1 Study of Pevifoscorvir Sodium
Part 4 Cohort Design for Long Term Dosing in TN/CNT Subjects with Chronic HBV Infection

Part 4 Cohort Designs



Hou, JL. et al., EASL 2025. Yuen, M-F. et al., EASL 2025.
All cohorts fully enrolled. NCT04536337; ROW: rest of the world.
Note: TN-treatment naïve; CNT-currently not treated.
ETV-entecavir.

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ALG-000184-201

Part 4 Baseline Characteristics

	HBeAg+			HBeAg-
	Part 4 Cohort 2		Part 4 Cohort B	Part 4 Cohort B
	ETV ×12 Weeks followed by 300 mg pevifoscorvir sodium +ETV	300 mg pevifoscorvir sodium +ETV	300 mg pevifoscorvir sodium monotherapy	300 mg pevifoscorvir sodium monotherapy
N	3	12	10	11
Age, years, mean (SD)	28 (4.6)	32.3 (10.2)	34.8 (9.1)	48.5 (12.0)
Female, N (%)	2 (66.7)	6 (50)	3 (30.0)	5 (45.5)
Asian, N (%)	3 (100)	12 (100)	9 (90.0)	3 (27.3)
BMI, kg/m^2, mean (SD)	22.3 (2.8)	22.2 (3.1)	22.4 (2.4)	26.0 (3.5)
HBV Genotype, N (%)	B: 1 (33) C: 2 (67)	B: 4 (33) C: 8 (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 (SD)	7.8 (1.1)	8.1 (0.8)	8.0 (0.8)	4.3 (0.7)
HBV RNA, $\log_{10}$ copies/mL, mean (SD)	6.5 (0.9)	6.7 (1.1)	5.3 (1.3)	2.0 (1.0)
HBsAg, $\log_{10}$ IU/mL, mean (SD)	4.1 (0.4)	4.5 (0.7)	4.3 (0.5)	3.5 (0.5)
HBeAg, $\log_{10}$ PEI U/mL, mean (SD)	2.0 (0.3)	2.5 (0.3)	2.6 (0.8)	-
HBcrAg, $\log_{10}$ U/mL, mean (SD)	8.0 (1.2)	8.3 (0.5)	8.3 (0.6)	3.3 (0.6)
ALT, U/L, mean (SD)	38.7 (9.1)	41.5 (22.7)	60.7 (36.9)	35.0 (14.5)

Yuen, M-F. et al; AASLD 2025.

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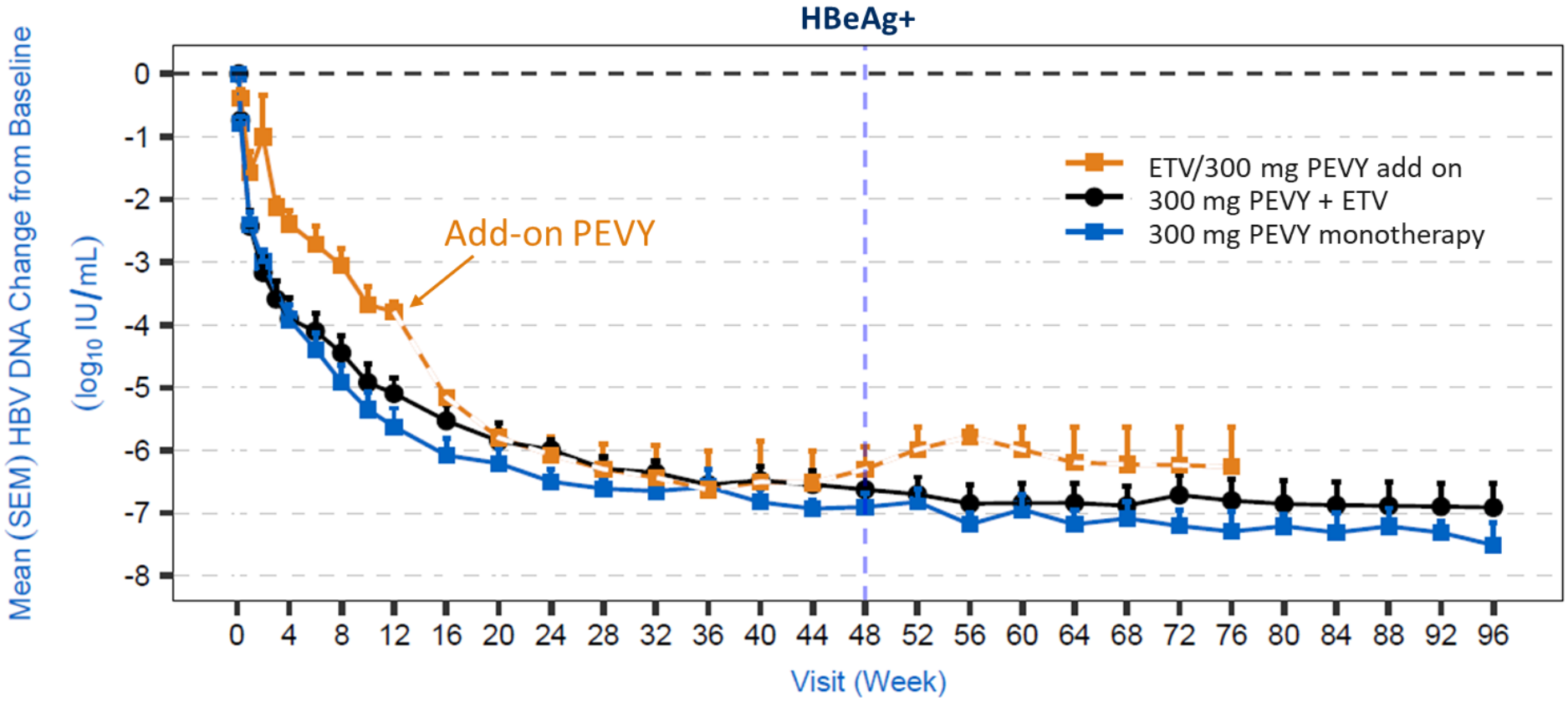
300 mg Pevifoscorvir Sodium ± ETV in HBeAg+ Subjects

Mean HBV DNA Change from Baseline



ETV/300 mg PEVY add on	3	3	3	3	3	2	2		
300 mg PEVY + ETV	11*	9	9	9	8	8	7	7	7
300 mg PEVY monotherapy	10	10	10	10	10	10	10	10	10

Number of subjects at each timepoint

Yuen, M-F. et al; AASLD 2025. *one subject excluded due to dosing non-compliance; PEVY – pevifoscorvir sodium.

300 mg pevifoscorvir sodium demonstrates greater HBV DNA reduction than ETV monotherapy

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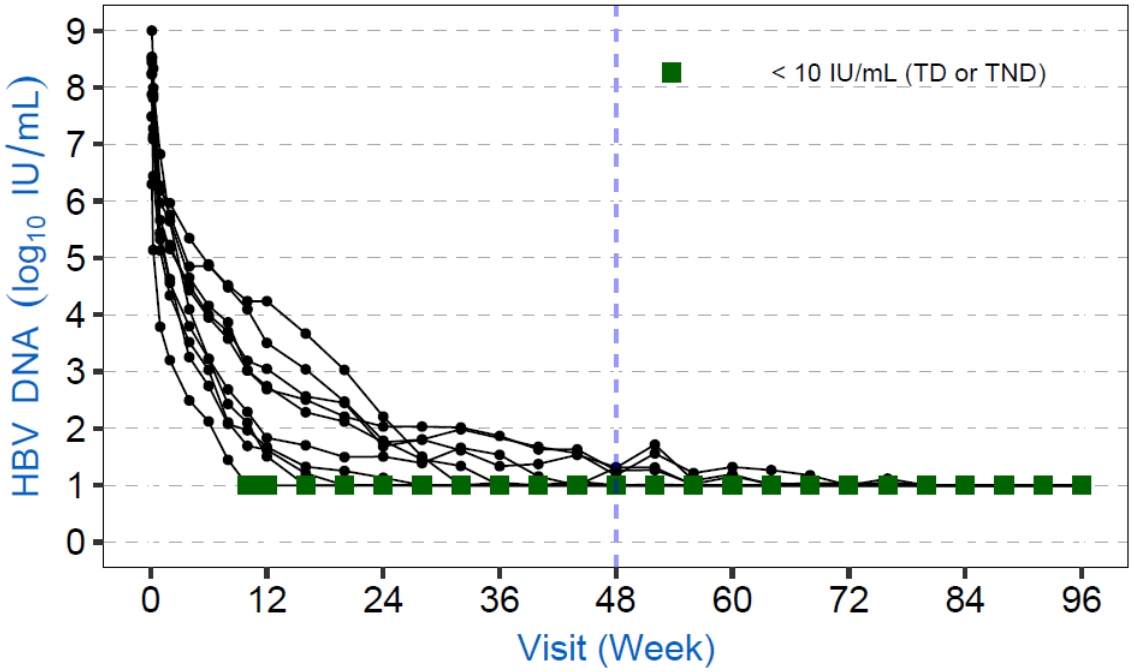
300 mg Pevifoscorvir Sodium Monotherapy

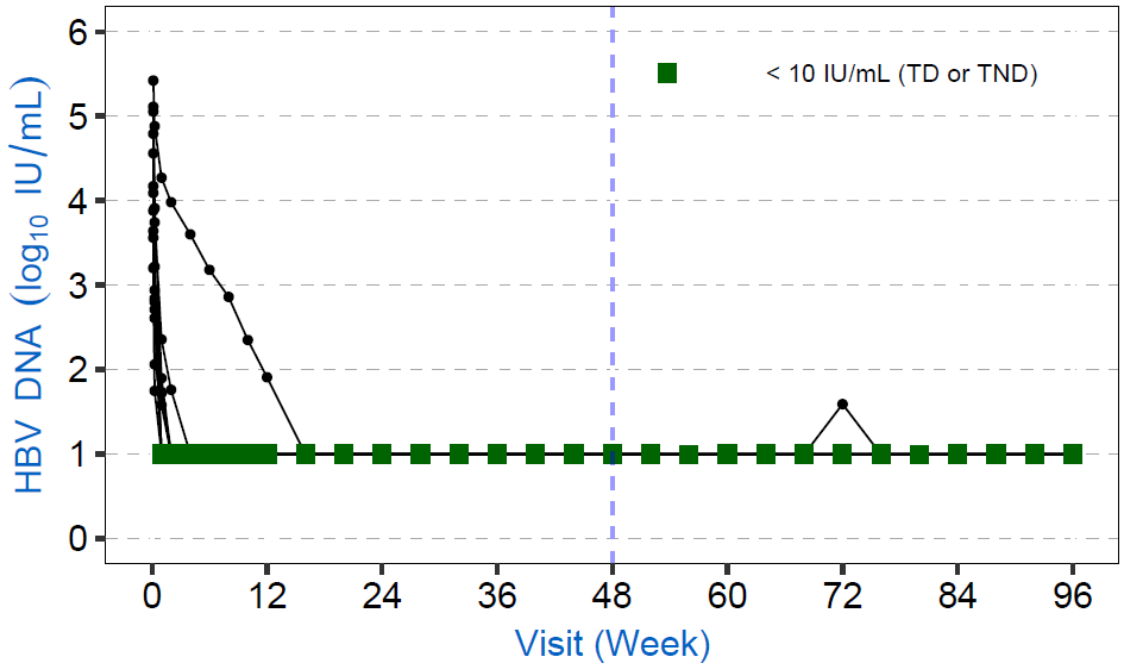
Reduction in Individual HBV DNA Levels Over Time

HBeAg+



Total n	10	10	10	9	10	10	10	10	10
< 10 IU/mL (TD or TND)	0	1	2	5	6	7	9	10	10
< 10 IU/mL (TND)	0	0	0	0	0	0	2	3	5

HBeAg-



Total n	11	11	11	11	11	10	9	9	9
< 10 IU/mL (TD or TND)	0	10	11	11	11	10	8	9	9
< 10 IU/mL (TND)	0	7	6	7	10	8	7	8	8

Note: one HBeAg- subject had transient HBV DNA increase from < LLOQ to 39 IU/mL at Week 72. At the subsequent visit (Week 76) HBV DNA was < LLOQ (10 IU/mL).

Yuen, M-F. et al; AASLD 2025. TD – target detected; TND – target not detected, LOD ≤ 4.29 IU/mL.

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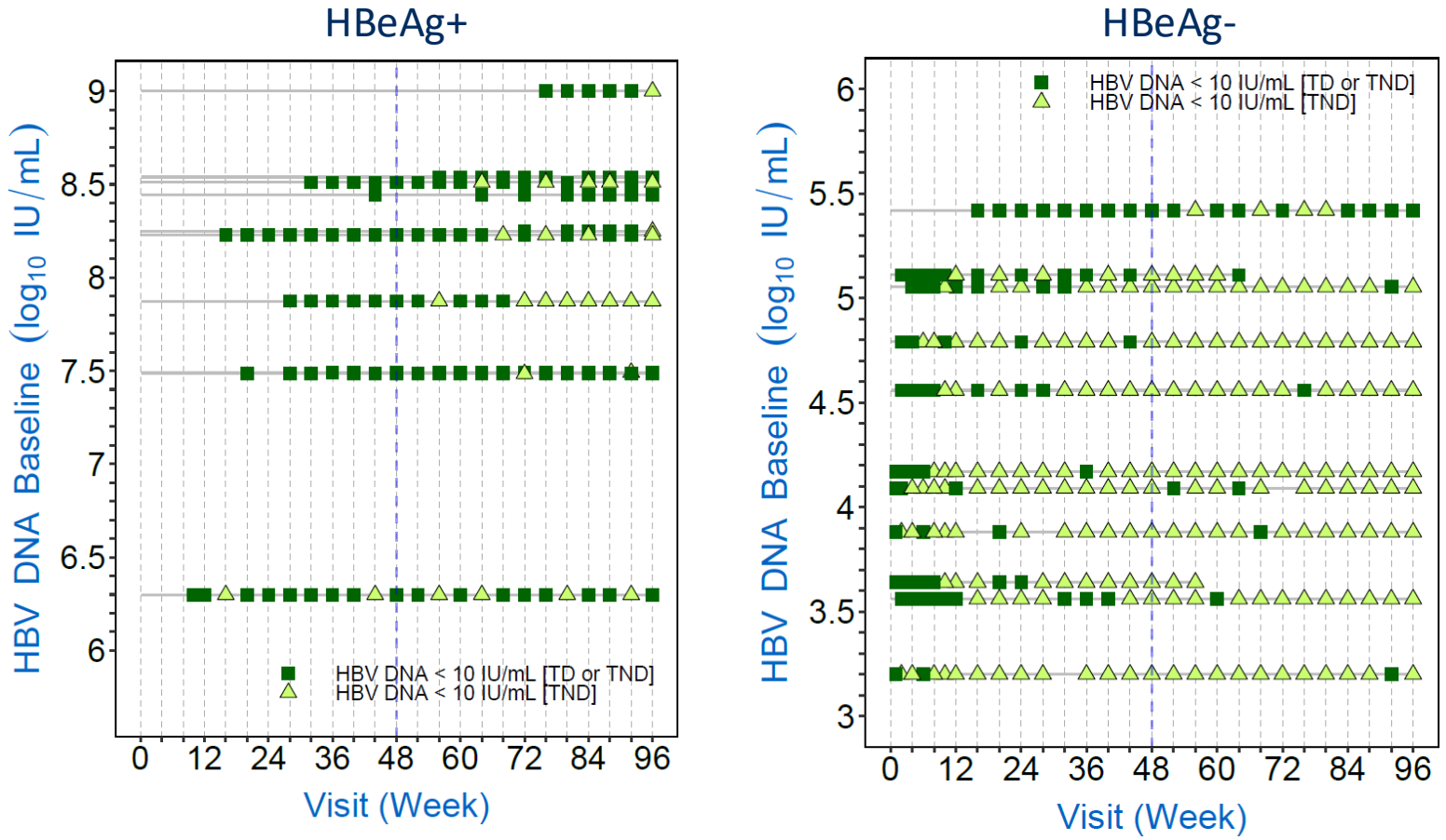
300 mg pevifoscorvir sodium monotherapy demonstrates profound HBV DNA reduction in TN/CNT subjects with chronic HBV infection; no viral breakthrough observed

Antiviral Effect in TN/CNT Subjects with Chronic HBV Infection

Time to Achieve HBV DNA Suppression with 300 mg Pevifoscorvir Sodium Monotherapy



Yuen, M-F. et al; EASL 2025. Data on file. Note: TD-target detected; TND-target not detected.

The time to achieve HBV DNA suppression (<10 IU/mL [TD or TND]) were dependent on the HBV DNA level at baseline

ALG-001075 In Vitro Resistance Mutation Were Not Detected after 96 W of 300 mg Pevifoscorvir Sodium Monotherapy

Resistance Mutations to other CAMs Detected were observed

Subject	HBeAg Status	Time Point	HBV DNA (IU/mL)	Core Mutation associated with CAM Resistance	Frequency [%]
A	**Negative**	Day 1	1.23E+04	**F23Y**	5.10
				I105V	18.33
				Y118F	8.60
B	**Negative**	Day 1	6.17E+04	I105L	1.98
				I105V	65.88
				T109M	99.82
C	**Negative**	Day 1	1.48E+04	I105L	11.16
				I105T	6.52
				I105V	30.01
D	**Negative**	Day 1	1.59E+03	**Y38F**	97.67
				I105T	99.97
E	**Positive**	Day 1	2.78E+8	none	n.a.
		Day 42	1.68E+3	**D29G**	1.06

- Resistance mutations to other CAMs were detected:
  - Bersacapavir: (F23Y, 5x; Y118F, 7x; D29G, 4x)
  - Vebicorvir: (Y118F, 14x; T109M, >68x; Y38F, 3x; D29G, 20x)

Jekle, et. al. EASL 2025. Data was sourced from publicly available literature, posters, and presentations.

No viral breakthrough or viral non-response based on HBV DNA levels and sequence analysis seen in pevifoscorvir sodium clinical studies to date

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300 mg Pevifoscorvir Sodium Monotherapy

Phase 1 Results vs. Standard of Care TDF/TAF

HBeAg Status	Drug[6]	% 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/D 10 IU/mL	% < LLOQ 29 IU/mL	% < LLOQ/D 10 IU/mL
E-	TDF (n=140)[a]	93%	17%[f]	91%	31%[f]
	TAF (n=285)[a]	94%	21%[f]	90%	33%[f]
	300 mg pevy (n=11)[c]	100%[4]	100%[4]	100%[5]	100%[1]
E+	TDF (n=292)[b]	67%	N/A	75%	9%[e]
	TAF (n=581)[b]	64%	N/A	73%	14%[e]
	300 mg pevy (n=10)[c]	100%[1]	60%[2]	100%[1]	100%[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 2025. [e] Kosh A. et al.; Journal of Hepatology 2018 V68: 672-681. [f] LLOQ <29 IU/mL (LLOD <10 IU/mL). [1] 10/10 subjects. [2] 6/10 subjects. [4] 11/11 subjects. [5] 9/9 subjects. [6] **No head-to-head clinical trials have been conducted. Caution should be exercised when comparing data across trials**. Pevy = pevifoscorvir sodium.

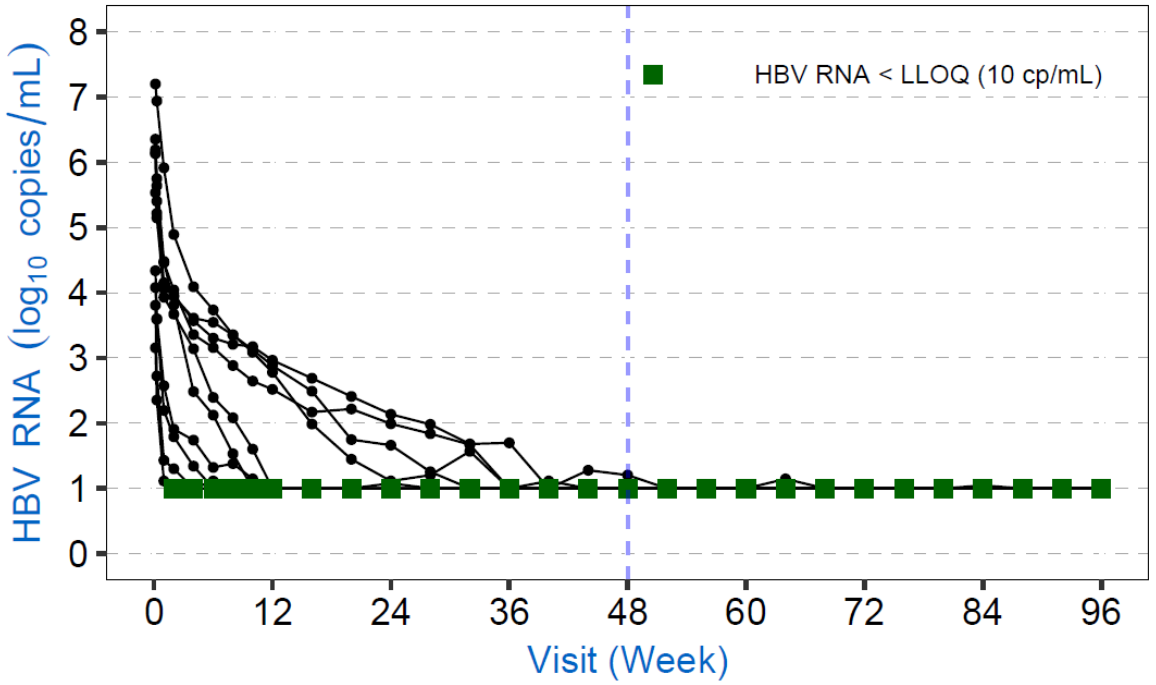
ALIGOS THERAPEUTICS

HBV DNA data suggests 300 mg pevifoscorvir sodium monotherapy may achieve superior chronic suppression, which is currently being evaluated in a Phase 2 study

300 mg Pevifoscorvir Sodium Monotherapy

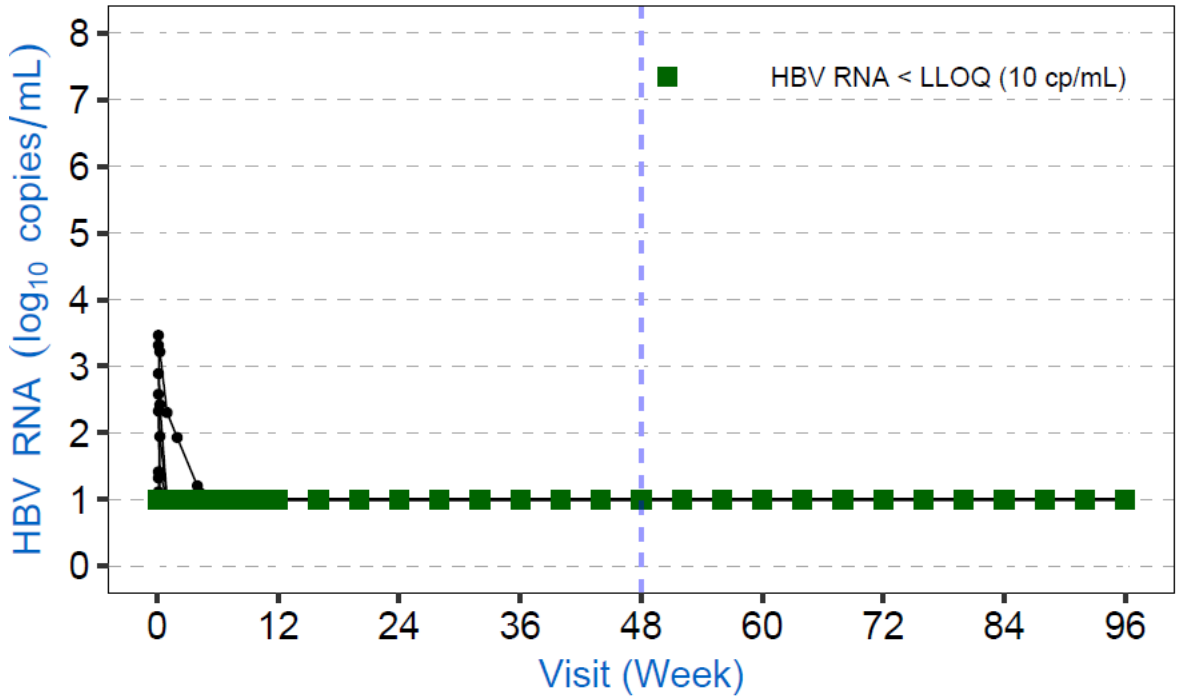
Reduction in Individual HBV RNA Level Over Time

HBeAg+



Total n	10	10	10	9	10	10	10	10	10
< 10 cp/mL	0	6	5	8	9	10	10	9	10

HBeAg-



	11	11	11	11	11	10	9	9	9
	3	11	11	11	11	10	9	9	9

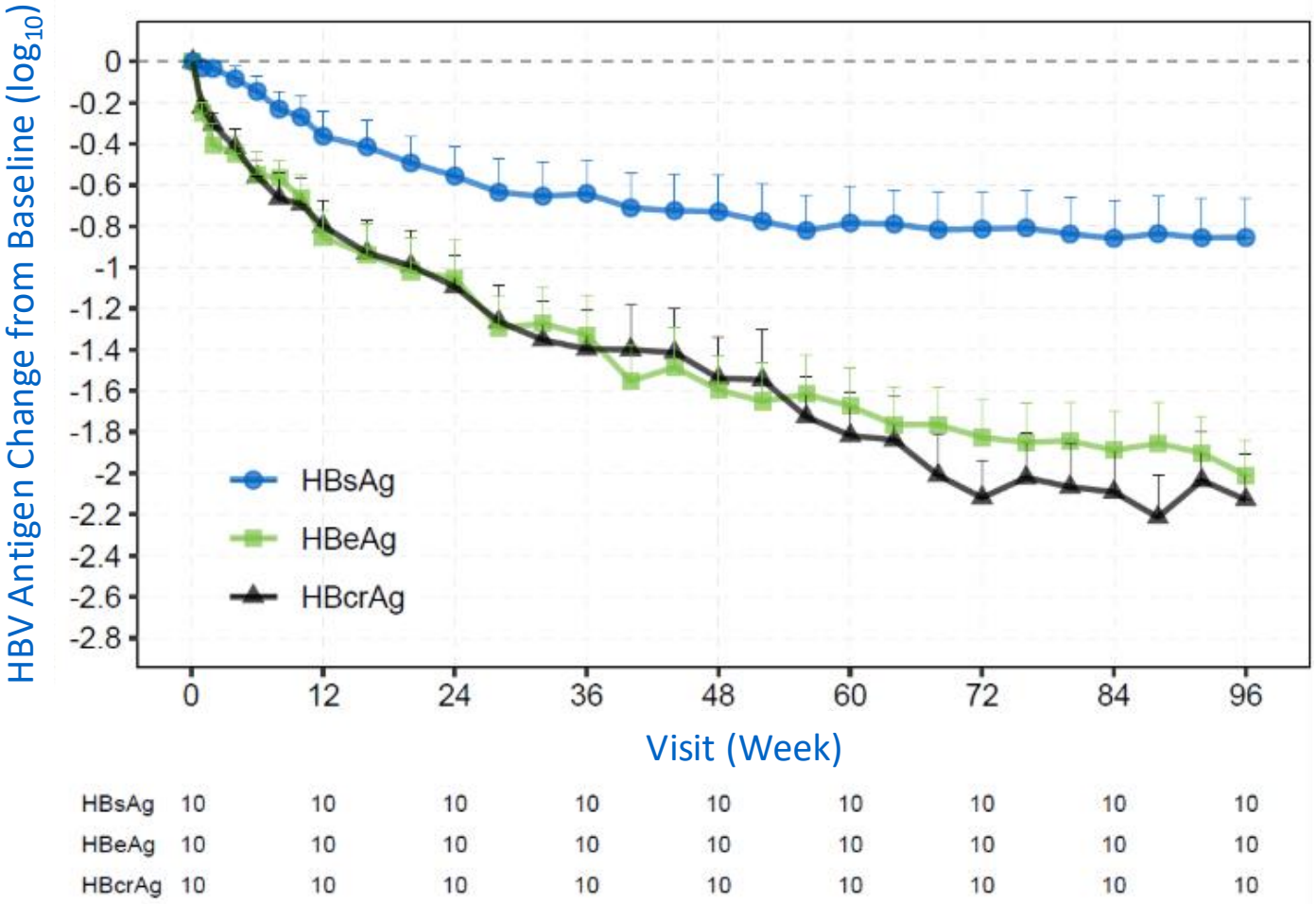
Yuen, M-F. et al; AASLD 2025. LLOQ – lower limit of quantitation. cp - copies.

ALIGOS THERAPEUTICS

300 mg pevifoscorvir sodium monotherapy demonstrated a rapid and profound reduction in HBV RNA in TN/CNT subjects with chronic HBV infection

300 mg Pevifoscorvir Sodium Monotherapy in HBeAg+ Subjects

Mean HBV Antigen Change From Baseline



Yuen, M-F. et al; AASLD 2025. Note: Data represents Mean (SEM) at each visit.

Substantial HBsAg, HBeAg, and HBcrAg reductions noted in HBeAg+ subjects

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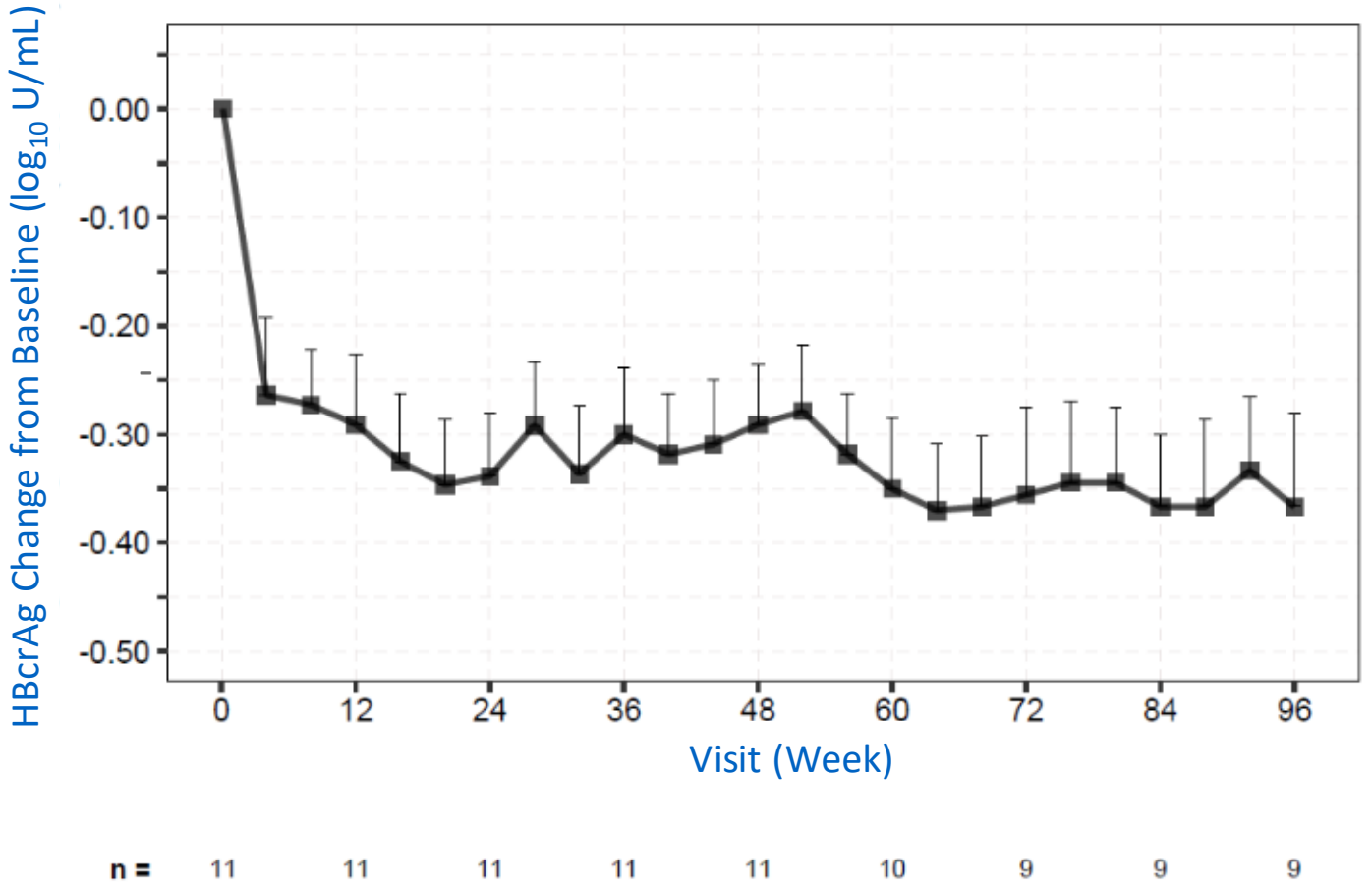
300 mg Pevifoscorvir Sodium Monotherapy in HBeAg- Subjects

Mean HBcrAg Change From Baseline



Yuen, M-F. et al; AASLD 2025. Data represents Mean (SEM) at each visit.

Meaningful HBcrAg reduction in HBeAg- subjects, no change in HBsAg

300 mg Pevifoscorvir Sodium Monotherapy

Safety Following 96 Weeks of Treatment

	HBeAg+	HBeAg-
Numbers of subjects with	N=10	N=11^
• **at least one TEAE, n (%)**	9 (90)	9 (81.8)
• **SAE**	0	0
• **TEAE leading to study drug discontinuation**	0	0
• **TEAE Grade ≥3**	3*	2*,#

^ Two HBeAg- subjects withdrew at Week 56 and 64, respectively, due to non-safety personal decisions.
* Grade ≥3 TEAEs of ALT/AST elevation were observed in 3 HBeAg+ and 1 HBeAg- subjects with preserved synthetic and excretory functions. All events resolved in the setting of continued pevifoscorvir sodium dosing and were not considered clinically concerning by the ALT Flare Committee.
# Grade 3 cholesterol/triglycerides increase in HBeAg- subject resolved in the setting of continued pevifoscorvir sodium dosing.

Yuen, M-F. et al; AASLD 2025.

ALIGOS THERAPEUTICS

Favorable safety profile observed in TN/CNT HBeAg+ and HBeAg- subjects with chronic HBV infection who received 300 mg pevifoscorvir sodium monotherapy for 96 weeks

96-Week 300 mg Pevifoscorvir Sodium Monotherapy Post Treatment Data

8-Week NA Follow Up

HBeAg Status	Viral Marker	Data
E-	HBV DNA	8/8 subjects had HBV DNA level < 10 IU/mL during NA only 8 Week follow-up
	HBV RNA	HBV RNA rebounds slightly after switching to NA but remains lower than baseline
	HBV Antigens	HBcrAg decline was maintained during the NA only 8 Week follow-up
E+	HBV DNA	6/8 subjects had HBV DNA level < 10 IU/mL during the NA only 8 Week follow-up
	HBV RNA	HBV RNA rebounds after switching to NA but remains lower than baseline
	HBV Antigens	No apparent HBV antigens increase observed during the NA only 8 Week follow-up

Yuen, et. al. AASLD 2025.

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These data suggest that pevifoscorvir sodium may reduce the cccDNA reservoir through engagement of its secondary mechanism of action

Pevifoscorvir Sodium

- Regulatory Pathway
- Commercial Opportunity

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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.

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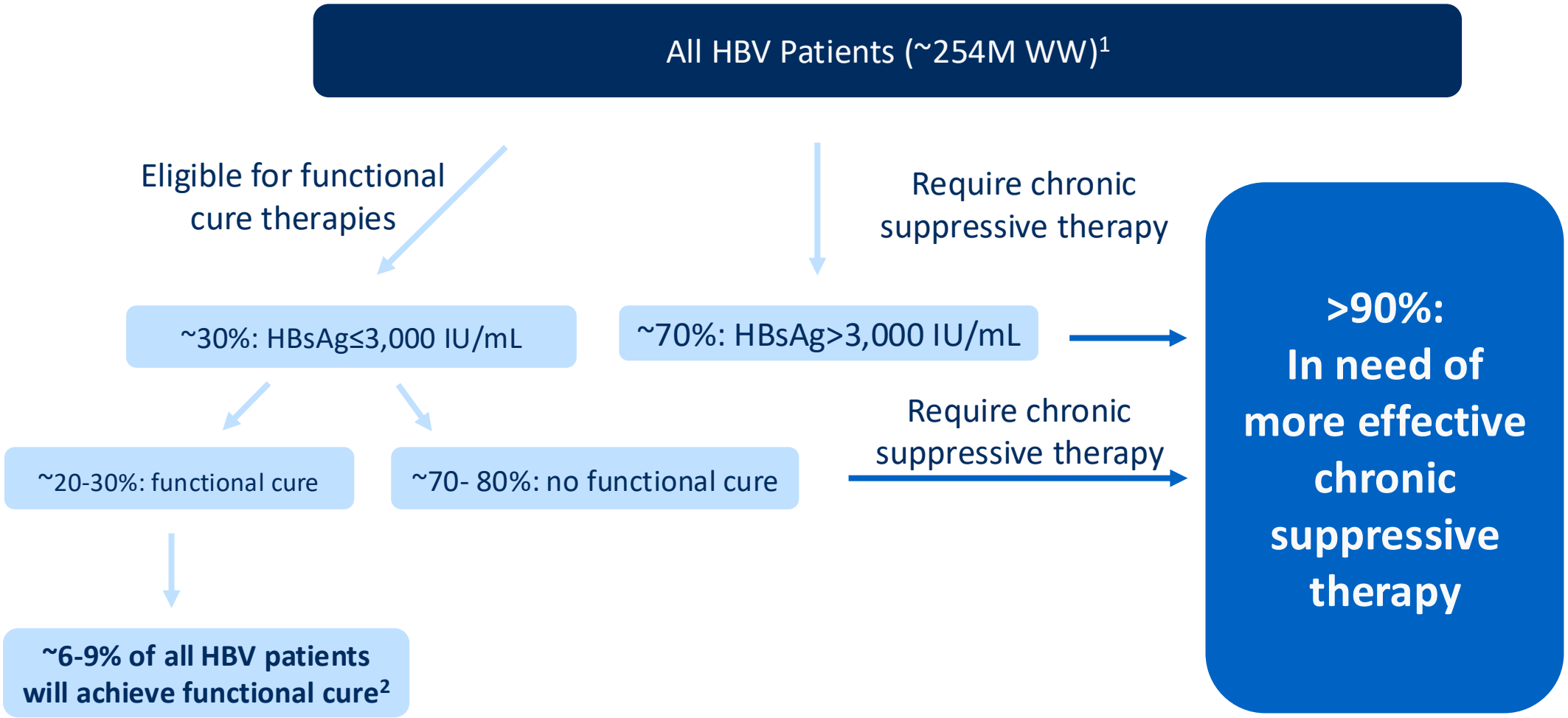
Aligos has received FDA, CHMP (EMA), and National Medical Products Administration in China feedback supporting subsequent studies utilizing this pathway

The Future Patient Journey for HBV Therapy

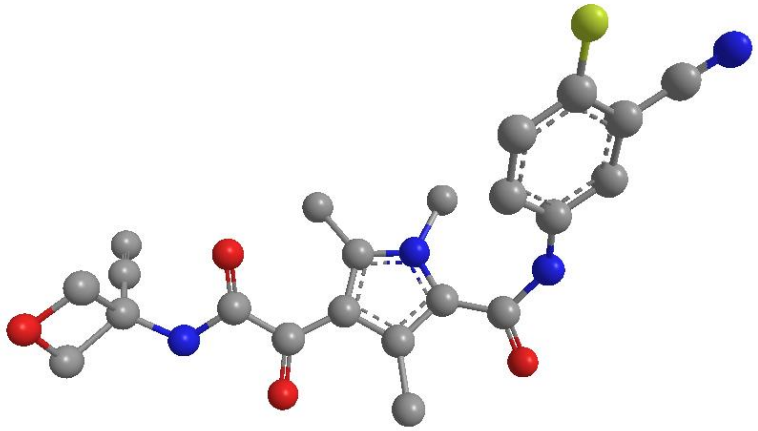


1. Data from the World Health Organization; 2019. Figures are estimated based on feedback from HBV KOLs. 2. Yuen M-F, et al. *N Engl J Med*. 2022;387(21):1957-1968.

Our Vision

Paving the Way for the Future of Chronic HBV Infection Treatment

pevifoscorvir sodium
has the potential to…



- Become standard of care
- Act as the backbone of next-generation therapies, inclusive of in-house programs (ex. ASO)
- Treat HBV patients that may not qualify for current therapies
- Affect the entire HBV lifecycle
- Lower HBsAg, allowing more patients to be eligible for curative therapies

ALIGOS THERAPEUTICS

Payor Perspectives: HBV Remains a Significant Unmet Medical Need

Chronic HBV Infection is the Leading Cause of Liver Transplant Worldwide

Initial market research indicates that payors are potentially willing to pay a premium for drugs that affect the entire HBV lifecycle to mitigate the expense associated with liver disease[4]

Liver disease and liver cancer are costly for payors

- Average cost for a liver transplant is **$1.2M**[1]
- Yearly healthcare costs can exceed **$184k/year**[2] for patients with decompensated cirrhosis
- Cumulative direct costs were estimated at **$45B**[3] in the US over the next 30 years

Faster and more profound reductions in all viral markers of disease have been shown as prognostic indicators for improved outcomes, which were cited by some payors as reasons to cover non-generic options

- Generics already exist, yet certain brand name drugs are still generating ~$1B in revenue per year

Current standard of care drugs are not indicated for all HBV patients, potentially leading to greater healthcare costs

1. Kaplan, et. al. Liver Transpl. 2023. 2. Nguyen, et. al. J. Hep. 2019. Data from 2004-2015. 3. Razavi-Shearer, et. al. JVH, 2023. 4. Based on initial market research data conducted by third party.

ALIGOS THERAPEUTICS

Estimated ~$1B US market opportunity annually with greater potential worldwide sales

China HBV Commercial Opportunity

Exclusive License Deal with Xiamen Amoytop Biotech for Pevifoscorvir Sodium in Greater China

90M+ Patients

Greater China is home to the largest HBV patient population

Robust Commercial Organization

Amoytop is the leading seller of pegylated interferon in Greater China

6 Approved Drugs in China

Amoytop is one of the leading hepatology companies in Greater China

Up to $445M in Upfront & Milestones

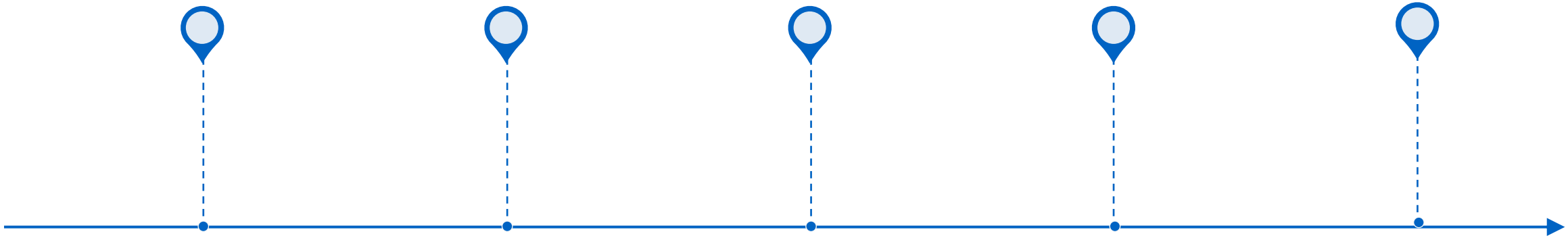
Aligos to receive an upfront payment of $25M USD and is entitled to up to $420M USD in clinical, regulatory, and sales milestones with tiered, high single-digit royalties

The future of pevifoscorvir sodium in Greater China

This agreement is expected to position pevifoscorvir sodium for greater development and regulatory success in Greater China

Amoytop has proven to be a valuable partner with our preclinical ASO program, ALG-170675, which is currently in IND-enabling studies

Pevifoscorvir sodium Upcoming Milestones



✓ **April 2026:** 12-week interim phase 2 analysis in 34 participants (HBeAg-)

✓ **April 2026:** Greater China outlicense deal signed for pevy with $25M USD upfront and up to $420 in milestones

2026: Phase 1 long-term NA only follow up data

2H2026: 24-week interim phase 2 analysis in approx. 50% of subjects (HBeAg+)

2027: Topline phase 2

To preserve study integrity and comply with FDA regulatory requirements, the company will remain blinded to subject-level data and will receive insights from the Data Safety Monitoring Board on protocol-specified assessments, including the potential for sample size re-estimation at each interim analysis

ALIGOS THERAPEUTICS

ALG-055009 for MASH, Obesity

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ALG-055009

A Potential Best-in-Class THR-β Agonist for MASH, Obesity

- **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[2,3]
- **MASH: Phase 1 highlights**
  - PK - dose proportional, low variability, $t_{1/2}$ ~20 hours (enhanced vs. resmetirom[2])
  - Well tolerated without clinical safety signals
  - Pharmacodynamics - dose proportional increases in SHBG, decreases in atherogenic lipids
- **MASH: Phase 2a HERALD study highlights**
  - Well-tolerated, with rates of GI-related AEs similar to placebo based on data to date
  - Primary endpoint achieved with robust reductions in liver fat content at Week 12 with a low oral dose
  - 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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[1] Not including any patent term extension. [2] No head-to-head clinical trials have been conducted. Caution should be exercised when comparing data across trials. [3] Data on file. EC_{50} HEK293T cell-based assay, nM.

THR-β Agonist (ALG-055009) + Incretin Receptor Agonist for Obesity

- **New preclinical and clinical findings from other companies suggest that THR-β agonists can significantly enhance weight loss when administered in combination with incretin receptor agonists (RAs) for obesity**
- **The concept of combining THR-β agonists and incretin RAs for obesity treatment has been clinically tested by others**
  - Ascletis combined their less potent, subcutaneously-dosed THR-β agonist (ASC47) with semaglutide in a Phase 1 study in the U.S. for obesity management
  - The combination resulted in over 50% greater weight reduction compared to semaglutide monotherapy
  - Furthermore, combination therapy mitigated rebound body weight gain after discontinuation of treatment
- **Aligos recently generated compelling preclinical data combining our proprietary THR-β agonist, ALG-055009, with incretin RAs in a diet-induced obese (DIO) mouse model**
  - ALG-055009 exhibited profound synergistic effects when used in combination with semaglutide or tirzepatide
  - The combination therapy also demonstrated enhanced antihyperlipidemic effects as compared to monotherapy
- **ALG-055009 warrants further development for MASH, obesity**
  - Phase 2b enabling activities underway for MASH; expected completion in 2025
  - Assessing potential Phase 2b clinical trial study designs with KOLs for MASH, and plan to consult with the FDA
  - **Discussions with partners underway; evaluating a variety of options to fund continued development**

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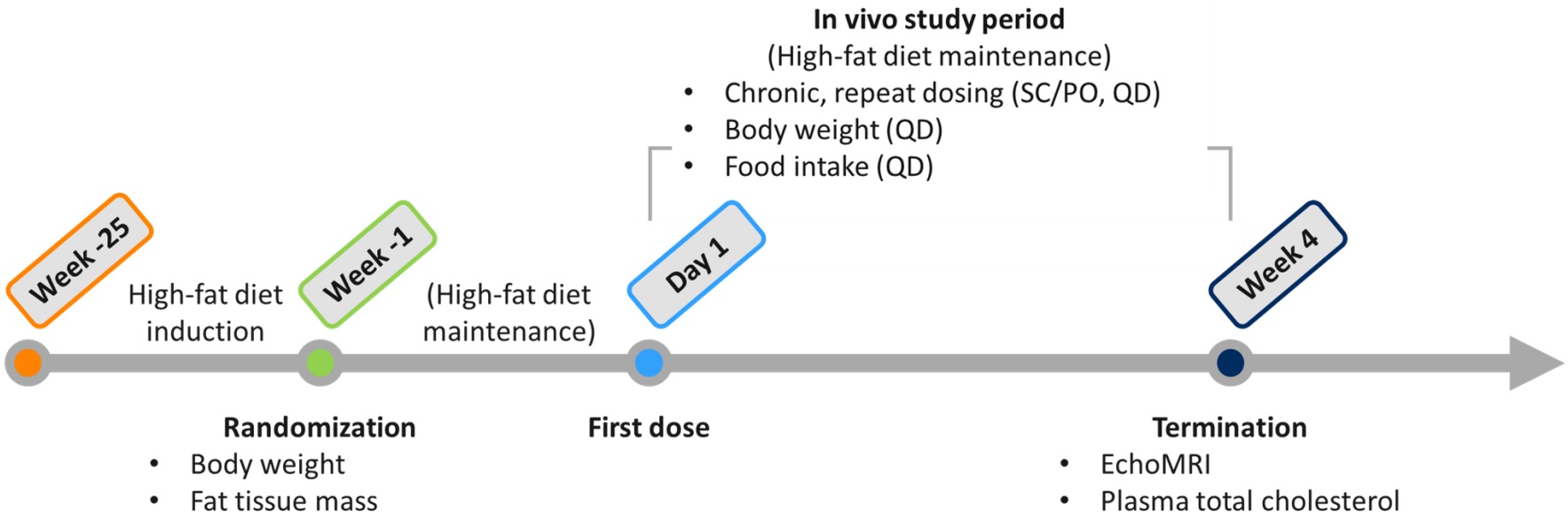
THR-β Agonist (ALG-055009) + Incretin Receptor Agonists

Study Design

- Hypothesis: addition of a THR-β agonist will enhance the magnitude and duration of weight loss effect by GLP-1 RA by attenuating the metabolic adaptation response via normalizing metabolic rate



Treatment groups

1. Vehicle
2. ALG-055009 (PO 1 mg/kg)
3. semaglutide (SEMA, SC 10 nmol/kg)
4. ALG-055009 + SEMA
5. tirzepatide (TIRZEP_lo, SC 3 nmol/kg)
6. ALG-055009 + TIRZEP_lo
7. tirzepatide (TIRZEP_hi, SC 10 nmol/kg)
8. ALG-055009 + TIRZEP_hi

PO =oral; QD =once daily; SC =subcutaneous; RA =receptor agonist.

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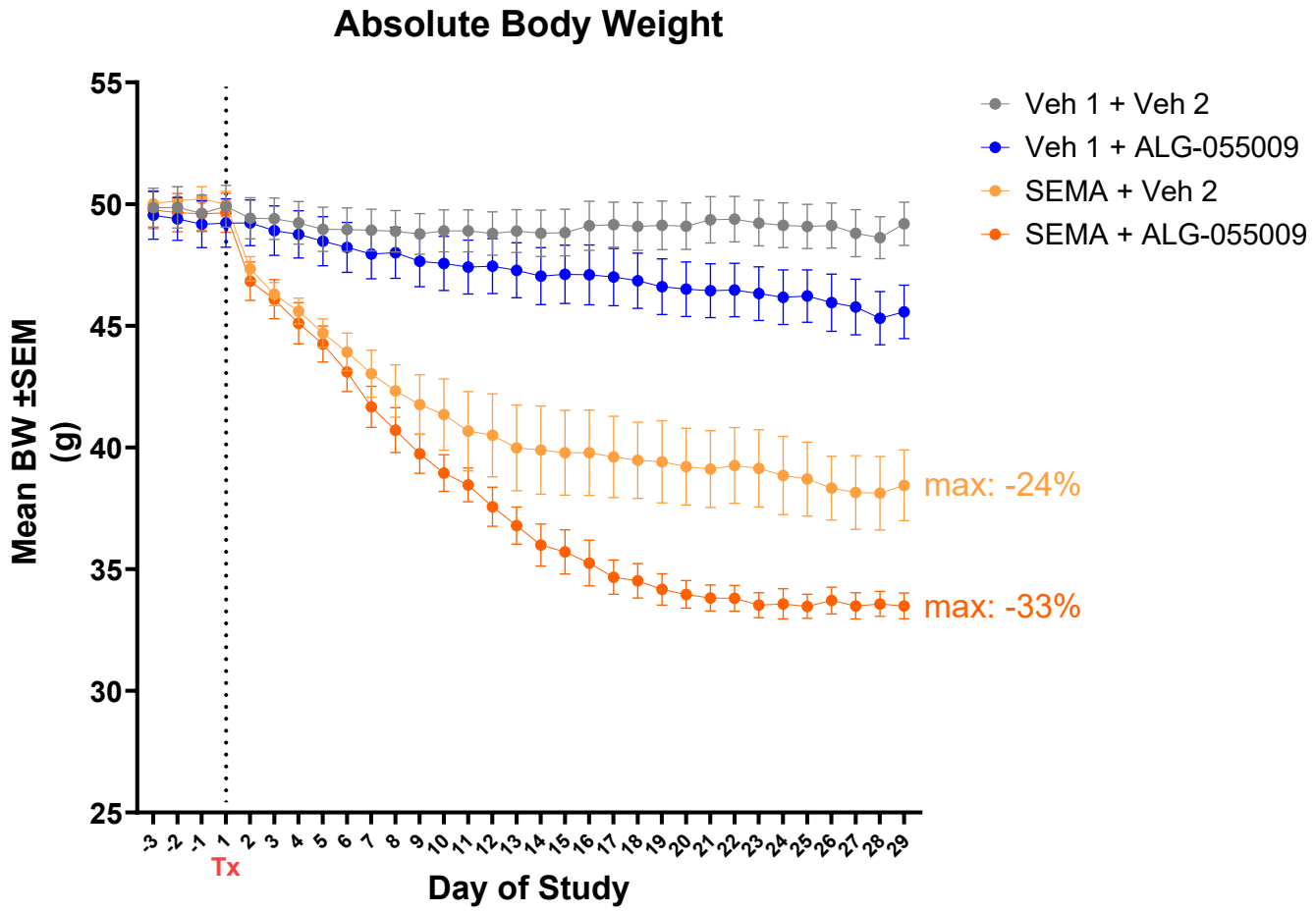
THR-β Agonist (ALG-055009) + Semaglutide

Combination Therapy Enhances Body Weight Loss in Diet-Induced Obese (DIO) Mice



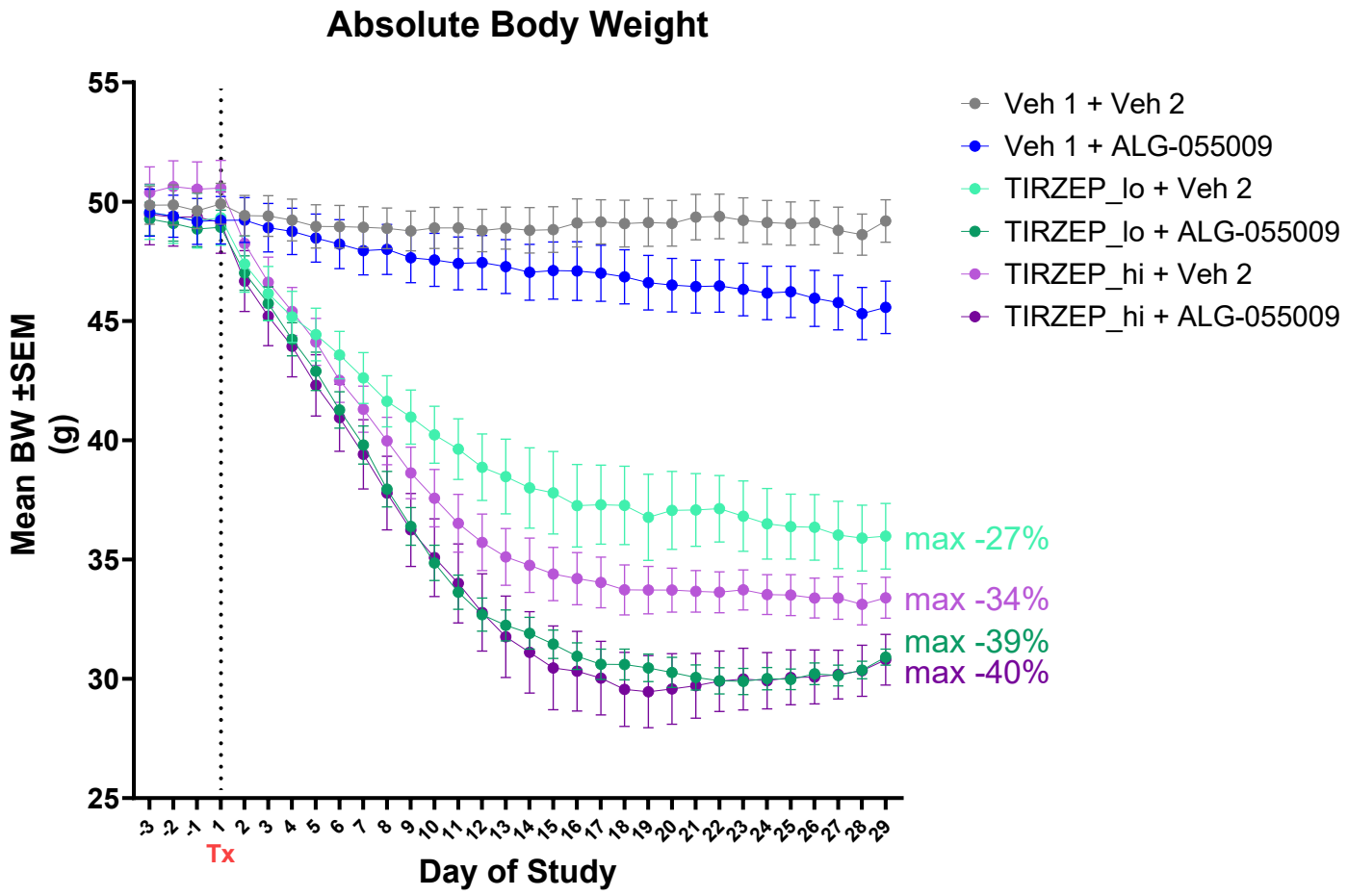
BW = body weight; Tx = start of therapeutic; percentages reported correspond to percent changes in weight as compared to baseline measurements (nadir).

ALG-055009 has profound synergistic effects on BW loss when combined with semaglutide, with as much as 33% decrease in BW within 28 days

ALIGOS THERAPEUTICS

THR-β Agonist (ALG-055009) + Tirzepatide

Combination Therapy Enhances Body Weight Loss in Diet-Induced Obese (DIO) Mice



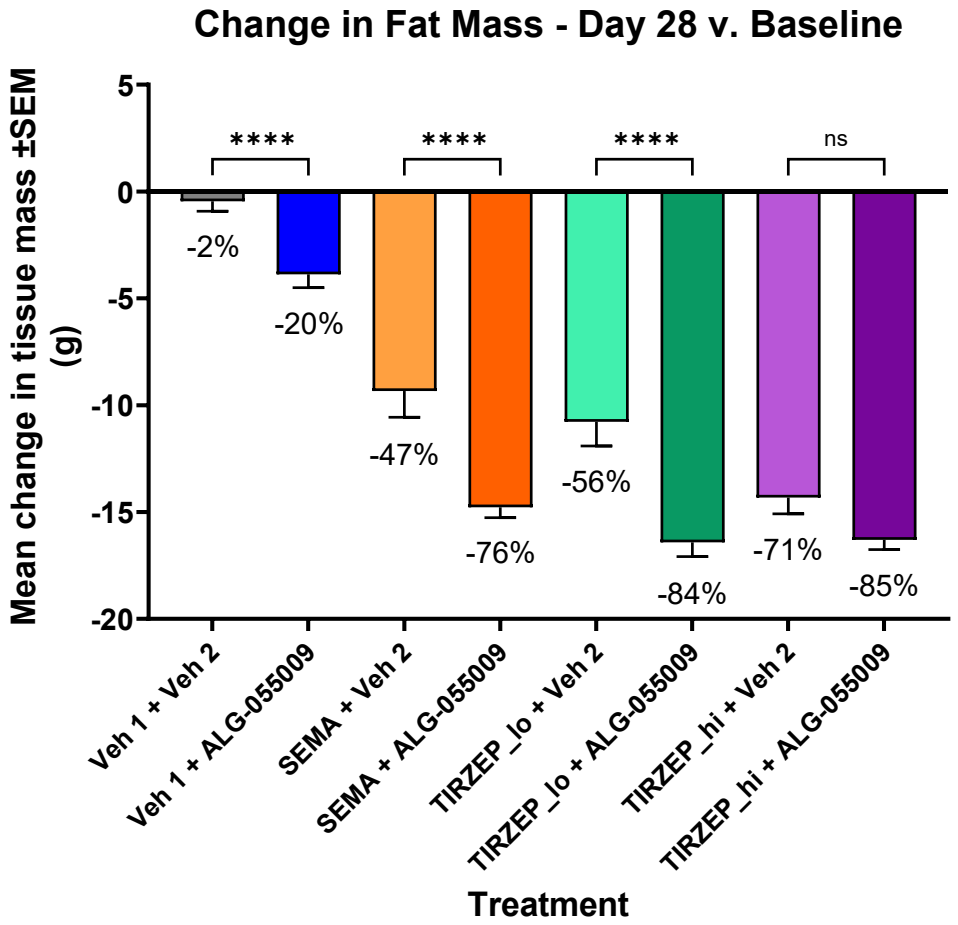
BW = body weight; Tx = start of therapeutic; percentages reported correspond to percent changes in weight as compared to base line measurements (nadir).

ALG-055009 has profound synergistic effects on BW loss when combined with both low- and high-dose tirzepatide, with as much as 40% decrease in BW within 28 days

ALIGOS THERAPEUTICS

THR-β Agonist (ALG-055009) + Incretin Receptor Agonists

Combination Therapy Enhances Loss of Fat Mass in Diet-Induced Obese (DIO) Mice



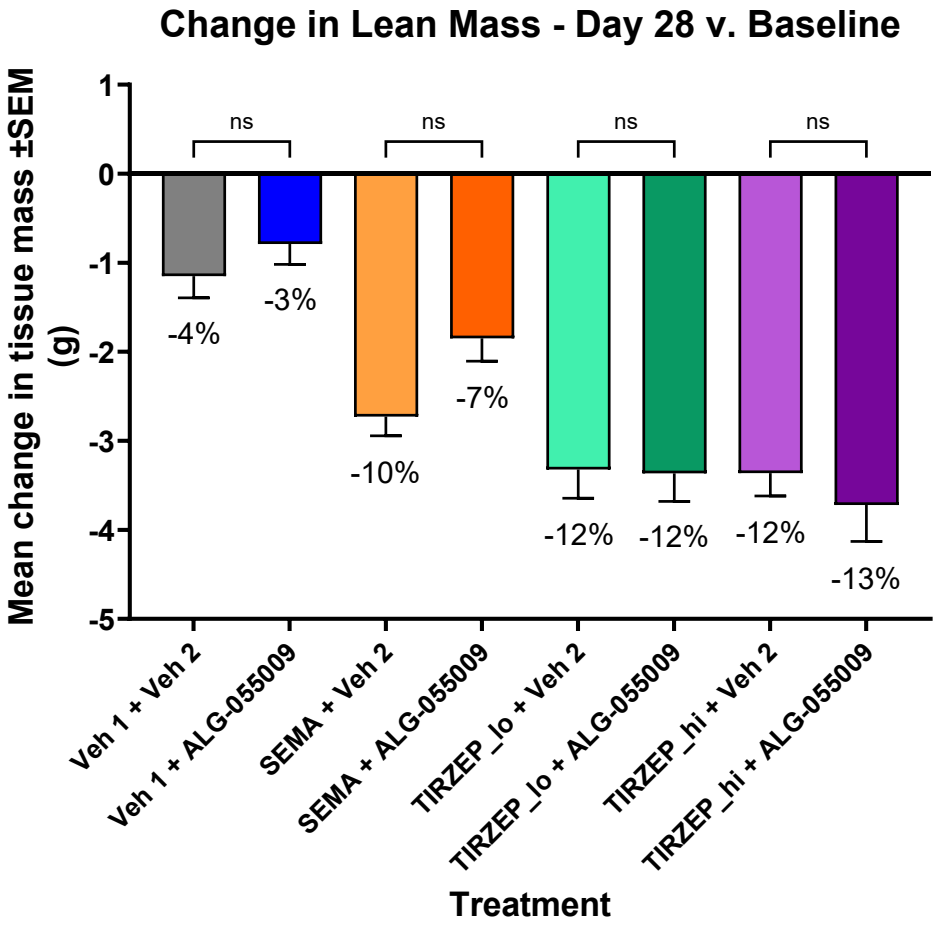
One-way ANOVA with Tukey's multiple comparisons test; percentages displayed below each bar correspond to percent changes in tissue mass as compared to baseline measurements; **** = p-value <0.0001; ns = not statistically significant.

The addition of ALG-055009 to incretin RA treatment results in greater decreases in fat mass as compared to incretin RA monotherapy

ALIGOS THERAPEUTICS

THR-β Agonist (ALG-055009) + Incretin Receptor Agonists

Combination Therapy Does Not Affect Changes in Lean Mass in Diet-Induced Obese (DIO) Mice



One-way ANOVA with Tukey's multiple comparisons test; percentages displayed below each bar correspond to percent changes in tissue mass as compared to baseline measurements; ns = not statistically significant.

The addition of ALG-055009 to incretin RA treatment had no significant effects on changes in lean mass as compared to incretin RA monotherapy

ALIGOS THERAPEUTICS

THR-β Agonist (ALG-055009) + Incretin Receptor Agonists

Summary

- DIO mice treated with ALG-055009 in combination with semaglutide or tirzepatide (low or high dose) experience greater weight loss for longer durations compared to animals treated only with incretin RA
  - The additional weight loss conferred by ALG-055009 is mainly due to decrease in fat tissue mass and not due to changes in lean tissue mass or food intake
  - ALG-055009 + SEMA reaches similar weight loss as TIRZEP_hi monotherapy
  - ALG-055009 + TIRZEP_lo has greater weight loss compared to TIRZEP_hi monotherapy indicating a possible dose-sparing regimen
- We provide evidence that the synergism is a result of ALG-055009's ability, as a THR-β agonist, to increase overall metabolism and overcome the metabolic adaptation that emerges after a period of weight loss
- Furthermore, combination therapy exhibited enhanced antihyperlipidemic effects as compared to monotherapy

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