ALIGOS THERAPEUTICS

CORPORATE PRESENTATION

July 2026

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Except where otherwise indicated, the information contained in this presentation speaks as of the date hereof or as of the date at which such information is expressed to be stated, as applicable, and such information may express preliminary estimated, unaudited results which shall be subject to audit or other year-end adjustments, and such audited or adjusted results may materially differ from those contained in this presentation.

This presentation concerns drug candidates, some of which are undergoing nonclinical studies and others of which are under clinical investigation, and all of which have not yet been approved for marketing by the U.S. Food and Drug Administration. These drug candidates are currently limited by federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

Aligos Development Portfolio

Candidate	Indication	MOA	Discovery	Nonclinical	Phase 1	Phase 2	Phase 3	Partner
pevifoscorvir sodium (pevy)[1,2]	Chronic HBV Infection *Monotherapy*	CAM-E						AMOYTOP BIOTECH *Greater China*
ALG-170675[3]	Chronic HBV Infection	ASO						AMOYTOP BIOTECH *Greater China*
TBD	Hepatitis Delta Virus	ASO						
ALG-055009	MASH, Obesity	THR-β Agonist						*In partnering discussions*

HBV = hepatitis B virus; CAM-E = capsid assembly modulator (empty); 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. ALG-097558 is currently being funded by government organizations in a Phase 2 study for COVID-19 and is not being funded by the company at this time.

As of 3/31/26 - Cash, cash equivalents and investments were $54.9M. Projected runway through Q4 2026, inclusive of the $25M upfront received from the Amoytop Greater China License

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CHRONIC HEPATITIS B VIRUS INFECTION

OVERVIEW

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Chronic HBV Infection: A Leading Cause of End-Stage Liver Disease & Liver Cancer

Liver cancer is the 6th most diagnosed form of cancer worldwide, but results in the 3rd most deaths of all cancers

240M people worldwide

27% diagnosed

4.3% treated

1M new infections in 2024

1.1M HBV-related deaths in 2024

HBV-related deaths now exceed those from HIV

Data from WHO 2026, EASL Clinical Practice Guideline 2025, and WHO 2019.
Bray, et. al. CA J Clin. 2024.

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Current and Novel Treatment Options for Prevention of End-Stage Liver Disease & Liver Cancer

Current drug candidates on track for regulatory approval: bepirovirsen and pevifoscorvir sodium

Current Standard of Care for 25+ years: Nucleoside/Nucleotide analogs (NAs)

- Monotherapy indicated for chronic suppression (HBV DNA <LLOQ at Week 48 following treatment)
- 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
- **Inadequate: Patients still progress while on therapy to end stage liver disease and liver cancer**

Bepirovirsen, ASO for functional cure: PDUFA Date October 2026

- Combination with NAs for functional cure (HBsAg < LLOQ ~6 months after a finite treatment regimen)
- Patients are only eligible for therapy with HBsAg≤3,000 IU/mL, which is **~30-40%** of all HBV patients[4]
- **Inadequate: ~19% of eligible patients achieve a functional cure at 72 weeks, or only ~6-8% of all patients[3]**

Pevifoscorvir Sodium, CAM-E: Phase 2 B-SUPREME Topline Data Expected in 3Q2027

- Blocks the drivers of disease progression: replication, integration, reservoir

[1]Wong et. al., J Hepatology 2021. [2] Kim et. al., AASLD 2024. [3] Hou, et al. *N Engl J Med*. 2026. [4]Figures are estimated based on feedback from HBV KOLs.

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Pevifoscorvir sodium has the potential to provide better outcomes for patients through monotherapy and combination with other agents

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
- Pevifoscorvir sodium received **FDA Fast Track Designation** for chronic HBV infection
- 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:

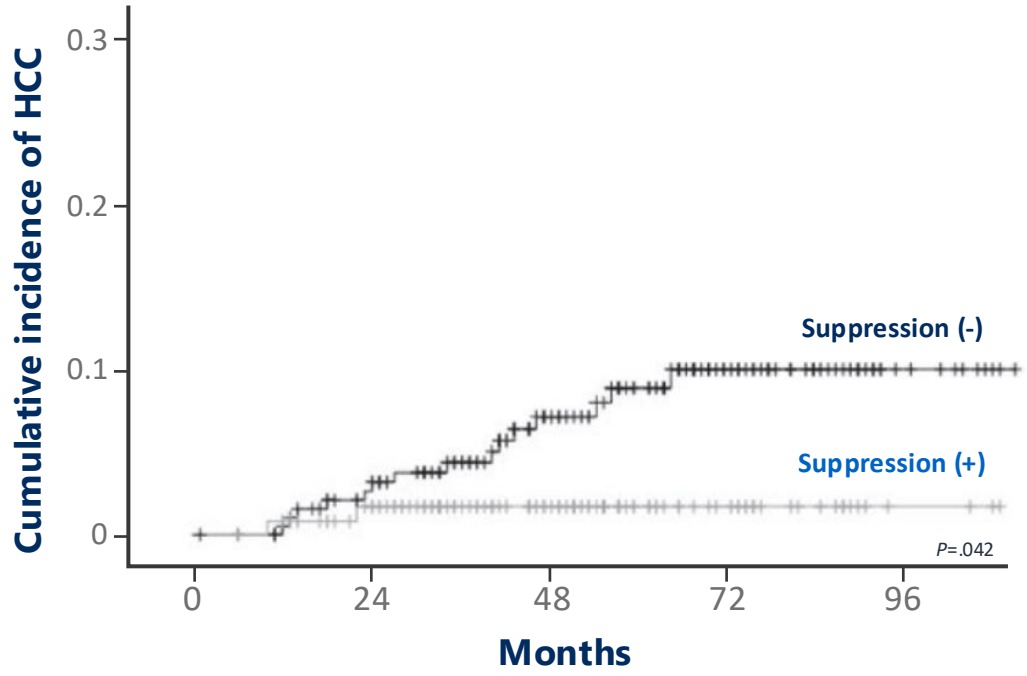
- A randomized controlled noninferiority (NI) (or superiority) trial comparing the investigational drug with an approved active control arm—The primary efficacy endpoint should be undetectable HBV DNA[13] after 48 weeks on-treatment in HBeAg-positive subjects, HBeAg-negative subjects, or both. The active 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

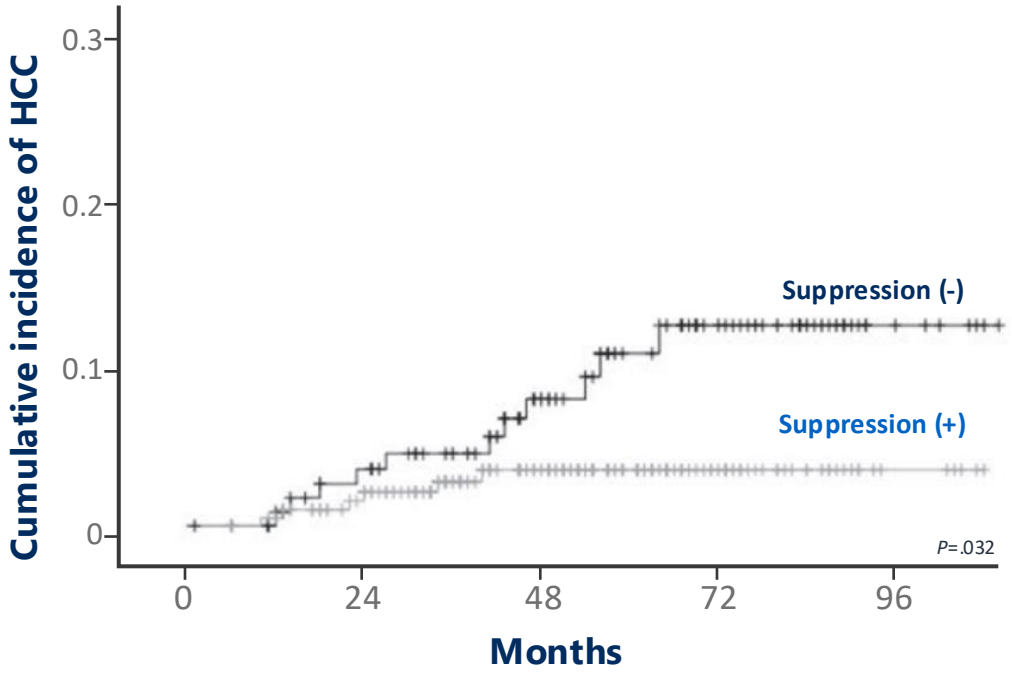
Of Those Diagnosed & Treated: Complete & Faster HBV DNA Suppression Improve Clinical Outcomes

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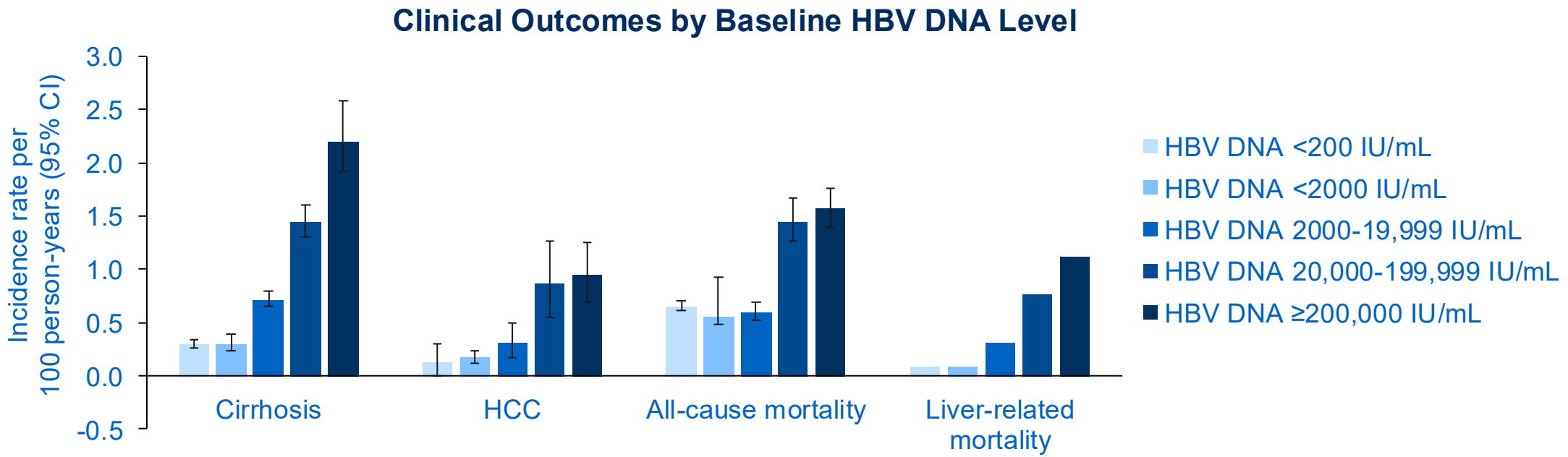
Cumulative HCC and cirrhosis incidence was significantly lower in patients who achieved complete viral suppression[a] within 1-2 years compared with patients who failed to achieve viral suppression within these periods[b]

Elevated Baseline DNA Predicts Poor Clinical Outcomes



1. Yucuma D, et al. *Lancet Gastroenterol Hepatol*. 2026;S2468-1253(25)00302-4.

Meta analysis of 71 longitudinal cohort studies and clinical studies among untreated, noncirrhotic patients with chronic HBV infection demonstrate the need for early intervention

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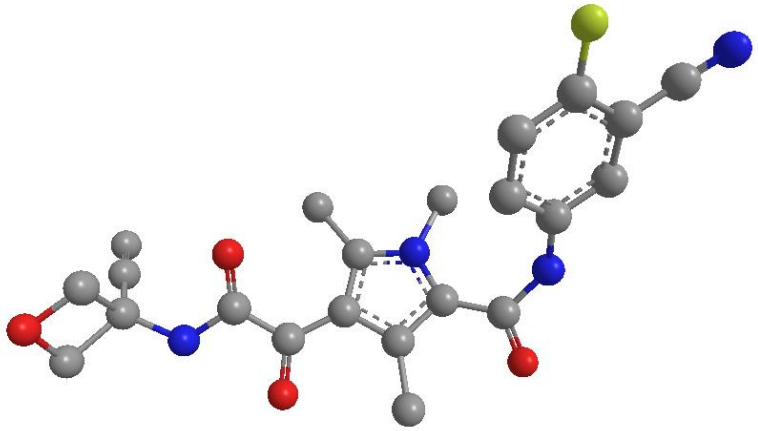
Our Vision to Prevent End-Stage Liver Disease & Liver Cancer

Paving the Way for the Future of Chronic HBV Infection Treatment

pevifoscorvir sodium
has the potential to…



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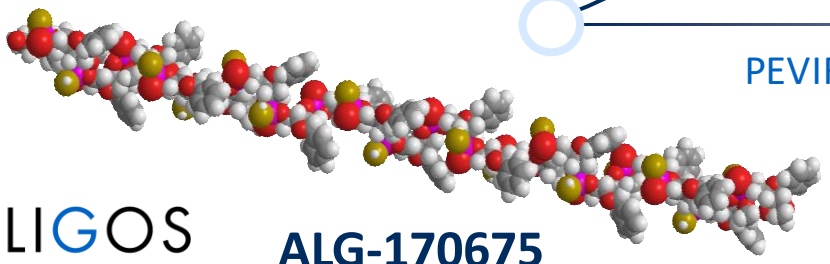

ALG-170675

PEVIFOSCORVIR SODIUM + ALG-170675

Become standard of care for chronic suppression

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

Near Term Goal

Act as the backbone of next-generation therapies, inclusive of in-house programs (ex. ASO)

Long Term Goal

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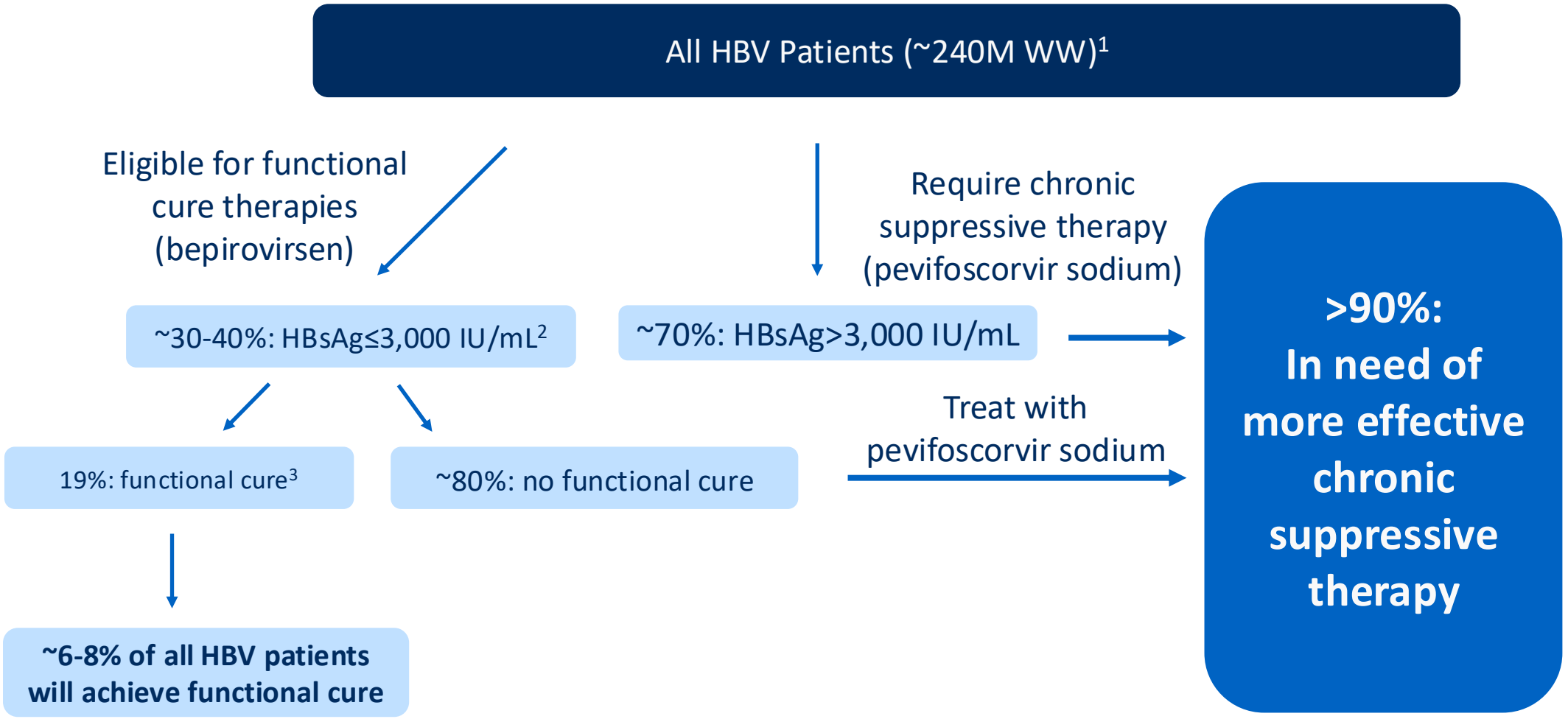
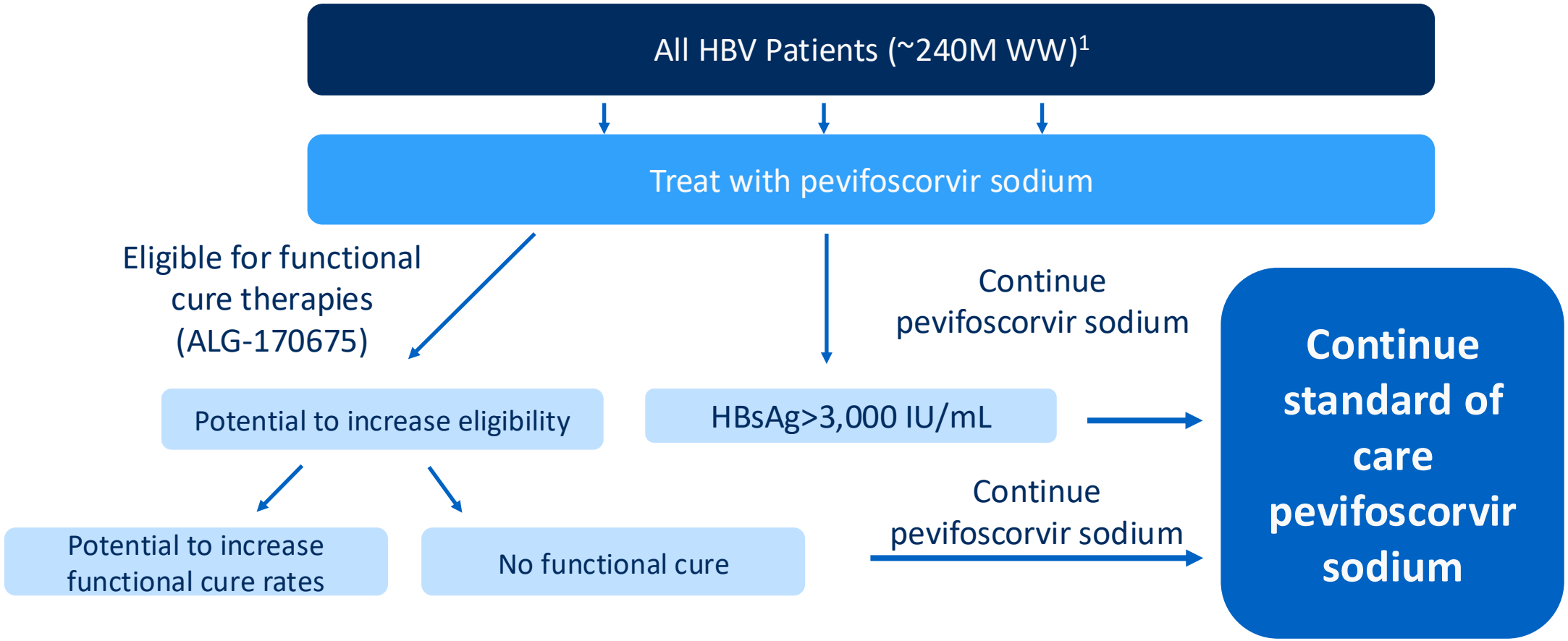
Our Vision for The Future Patient Journey for HBV Therapy



1. Data from the World Health Organization; 2026. 2. Figures are estimated based on feedback from HBV KOLs. 3. Hou, et al. *N Engl J Med*. 2026.

Our Vision for Pevifoscorvir Sodium as the Standard of Care



1. Data from the World Health Organization; 2026. 2. Yuen, et al. EASL 2026.

In clinical studies to date, pevifoscorvir sodium has reduced HBsAg to <3,000 IU/mL in 40% of HBeAg+ participants at week 48[2]

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Pevifoscorvir Sodium

Small Molecule CAM-E

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Treatment as Prevention: The Need to Treat as an Infectious Disease

With functional cure rates at ~6-8%[1] of all HBV patients, how do we reduce the risk of end-stage liver disease and liver cancer?

Deeper viral control

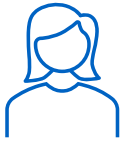
- Rapid and profound viral suppression
- Antigen reduction
- Reduction/elimination of cccDNA and integrated HBV DNA

Complementary mechanisms

- Combination therapy using agents with distinct, synergistic MOAs
- Address multiple virologic barriers simultaneously

Durable responses

- Enable functional cures that are not reactivated due to immuno-suppression



Patient-centered outcomes

- Match treatment strategy to individual risk
- Safety considerations with new therapies
- Improve quality of life

cccDNA, covalently closed circular DNA; HBV, hepatitis B virus; MOA, mechanism of action. 1. Hou, et al. *N Engl J Med*. 2026.

The goal of HBV treatment: Prevent end-stage liver disease and liver cancer

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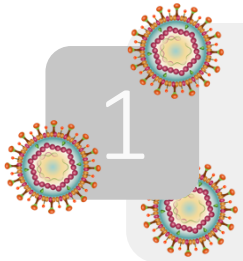
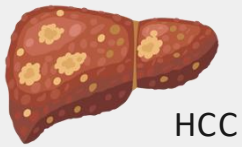
HBV Pathogenesis: The Three Drivers That Lead to End Stage Liver Disease/Cancer



1 REPLICATION

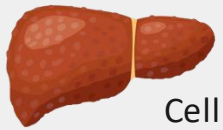
Inflammation

Fibrosis/cirrhosis

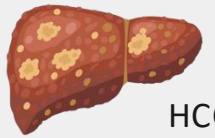


HCC

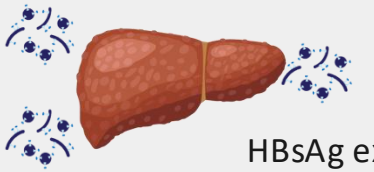
2 INTEGRATION



Cell death

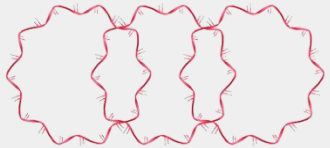


HCC

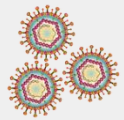


HBsAg expression → immune suppression

3 RESERVOIR



cccDNA



HBV DNA

HBV RNA

HBsAg

HBcrAg

HBeAg

Maintenance of viral lifecycle and infection

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

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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 enrollment completed in July 2026:** 131 participants enrolled in the HBeAg+ cohort; 114 participants enrolled in the HBeAg- cohort
- **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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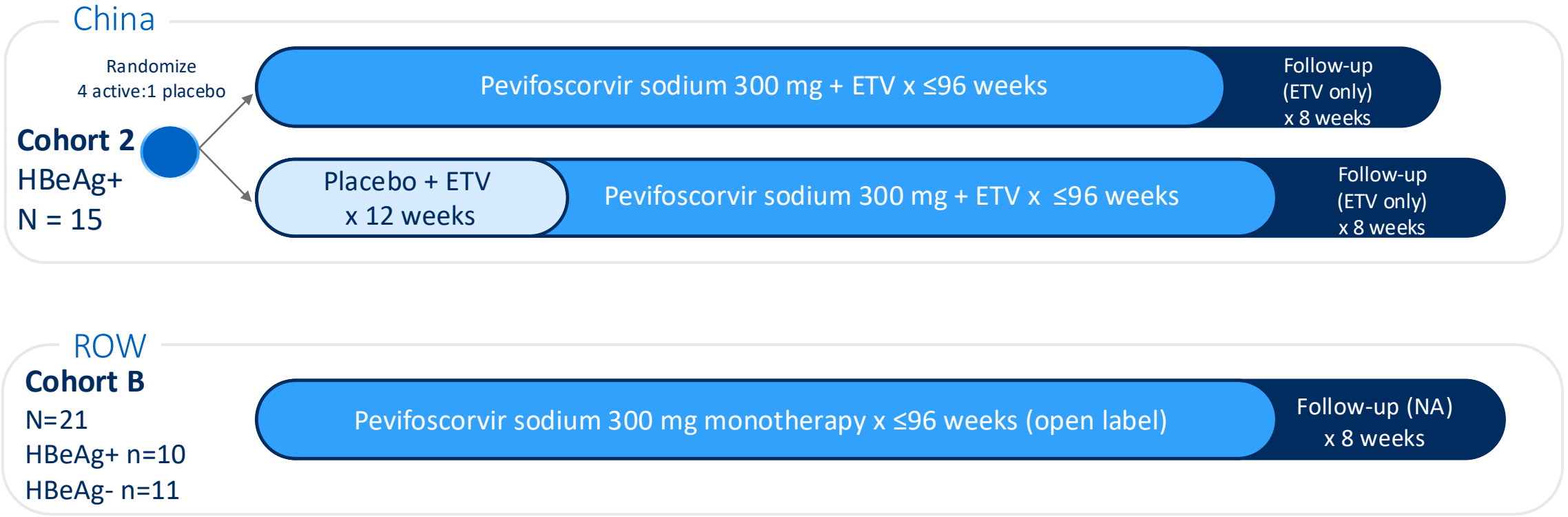
ALG-000184-201, A Multi-Part Phase 1 Study of Pevifoscorvir Sodium
Part 4 Cohort Design for Long Term Dosing in TN/CNT Subjects with Chronic HBV Infection

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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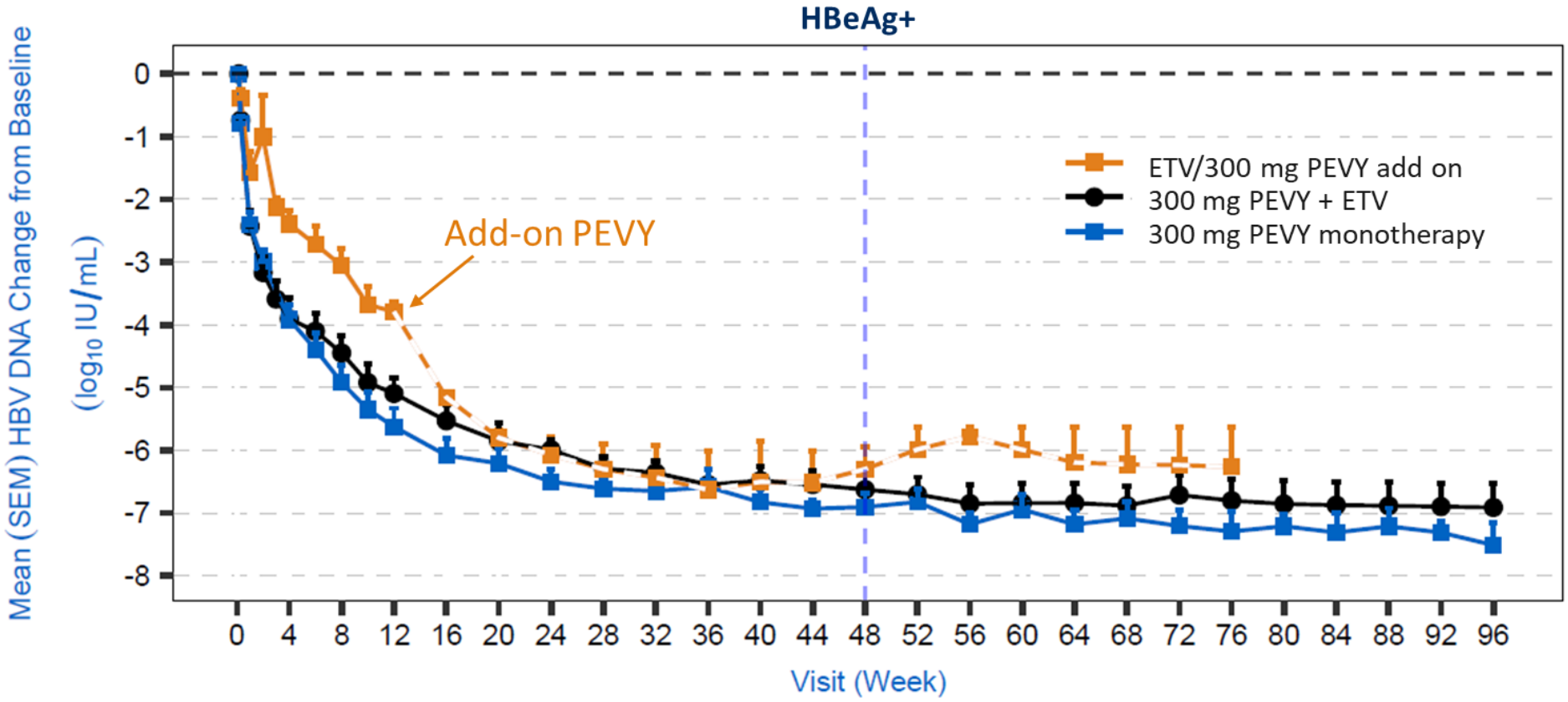
300 mg Pevifoscorvir Sodium ± ETV in HBeAg+ Subjects

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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ALG-001075 In Vitro Resistance Mutations 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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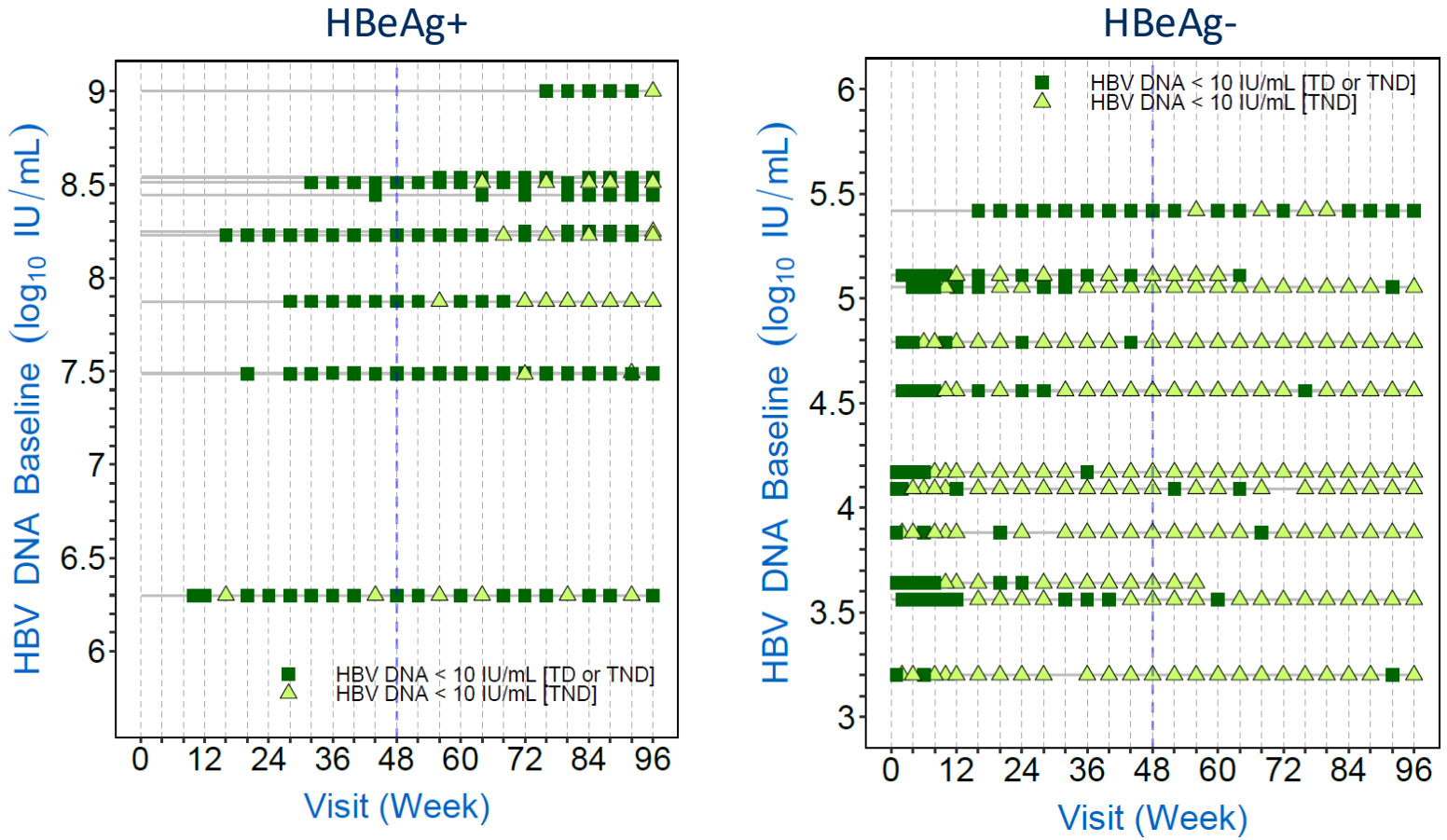
Antiviral Effect in TN/CNT Subjects with Chronic HBV Infection

Time to achieve suppression were dependent on the HBV DNA level at baseline



Yuen, M-F. et al; EASL 2025. Data on file. TD-target detected; TND-target not detected. LLOQ - lower limit of quantification. LLOD - lower limit of detection. LLOQ <10 IU/mL [TD or TND]. LLOD <10 IU/mL [TND].

60% of HBeAg+ participants reached LLOQ at week 48;
100% of HBeAg- participants reached LLOQ at week 20

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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.

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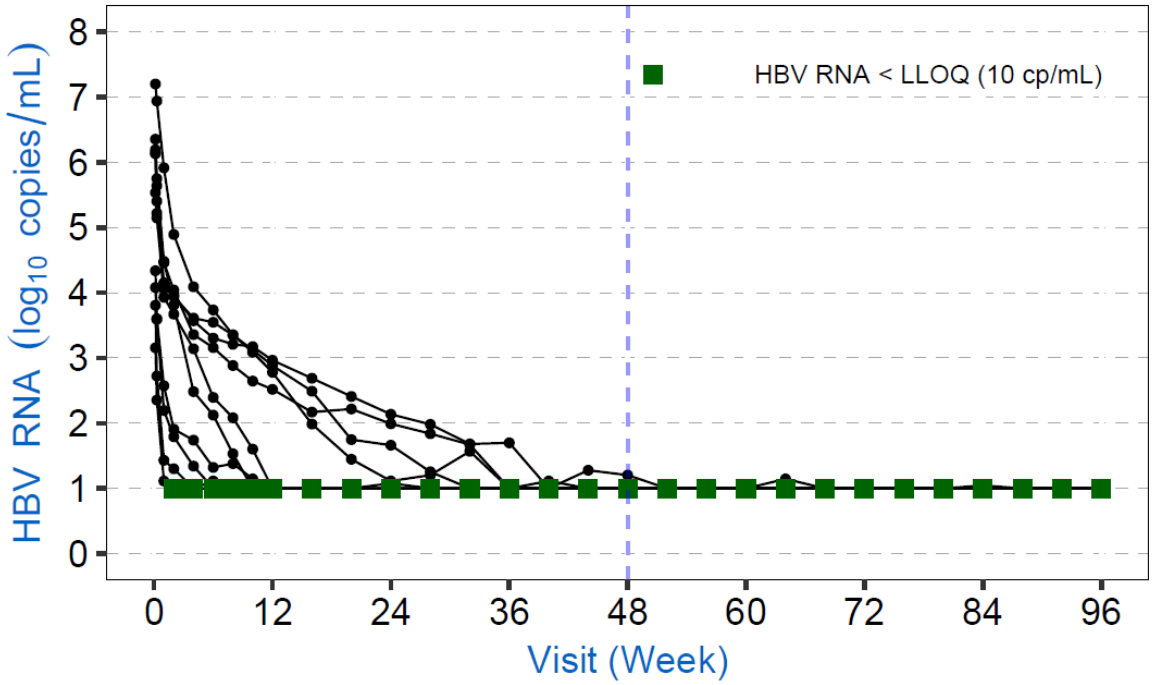
HBV DNA data suggests 300 mg pevifoscorvir sodium monotherapy may achieve superior chronic suppression, which is currently being evaluated in a Phase 2 study

300 mg Pevifoscorvir Sodium Monotherapy

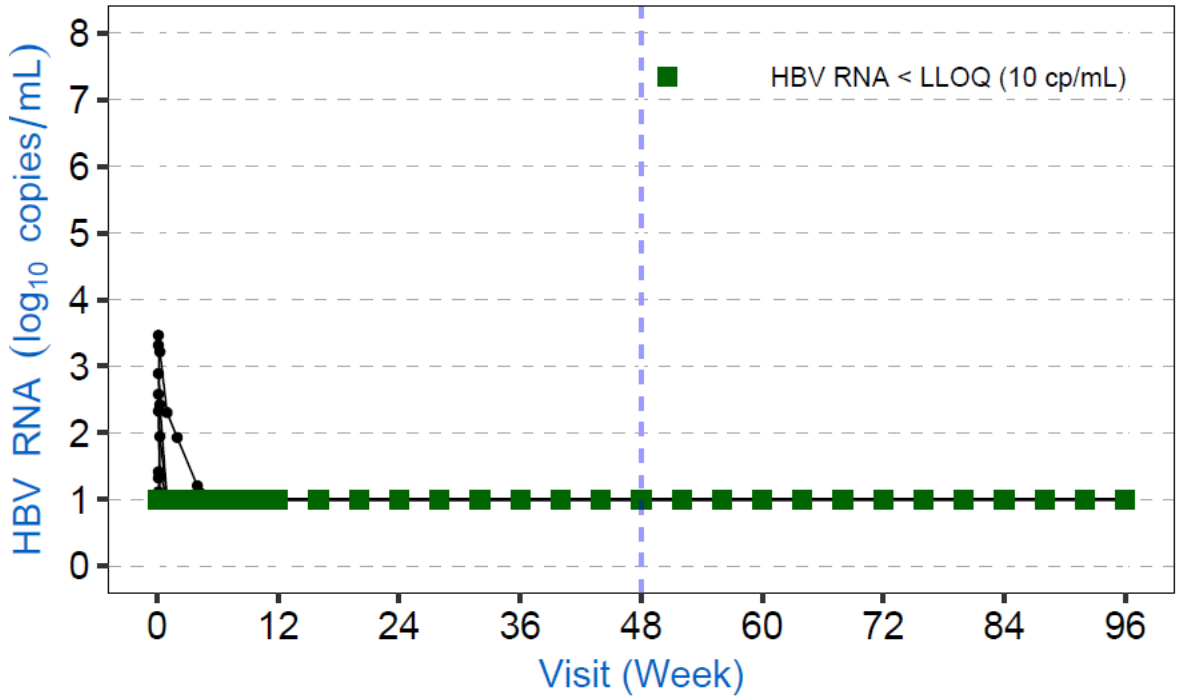
Reduction in Individual HBV RNA Level Over Time

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

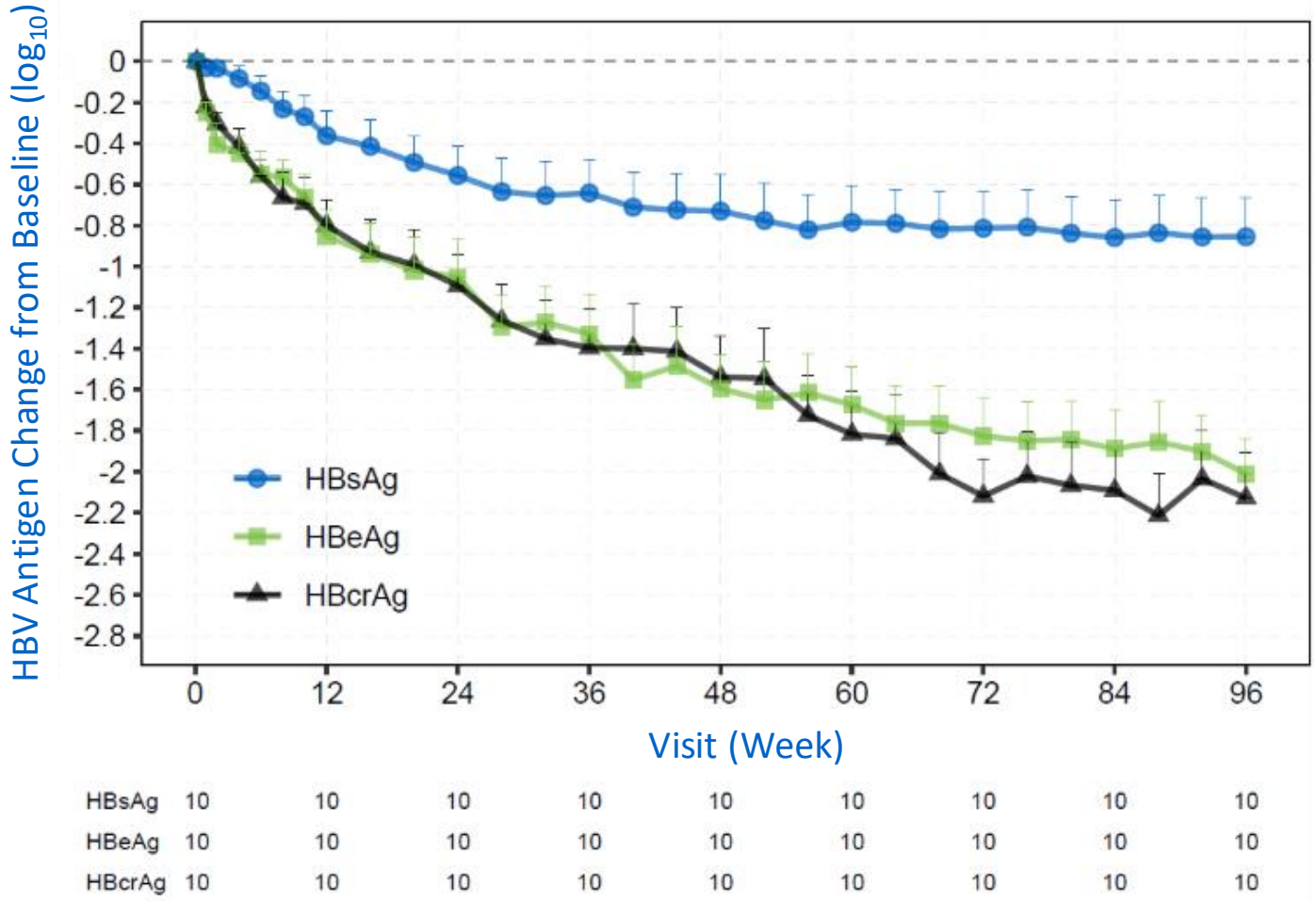
Yuen, M-F. et al; AASLD 2025. LLOQ – lower limit of quantitation. cp - copies.

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

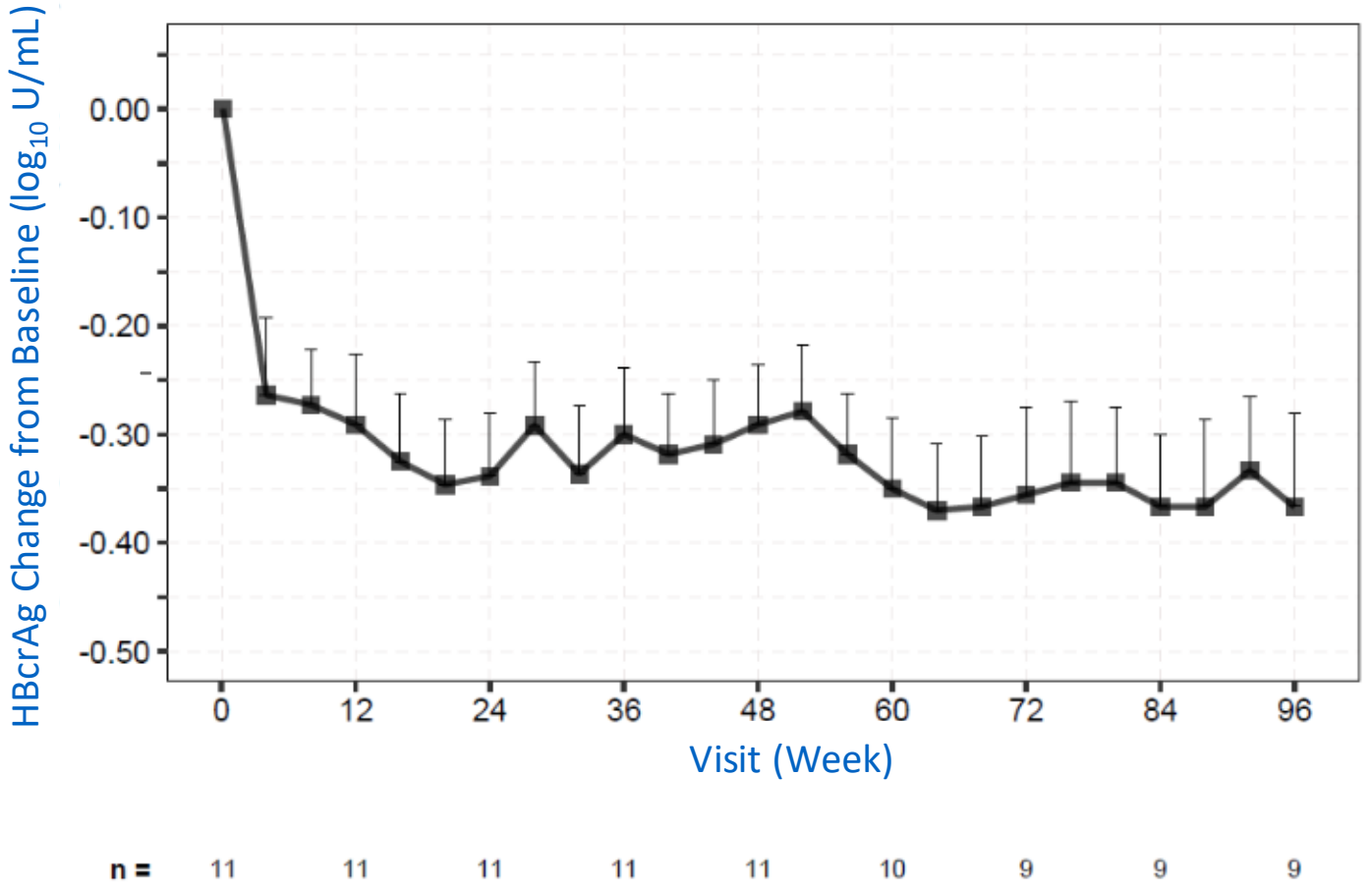


Yuen, M-F. et al; AASLD 2025. Note: Data represents Mean (SEM) at each visit.

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

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

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.

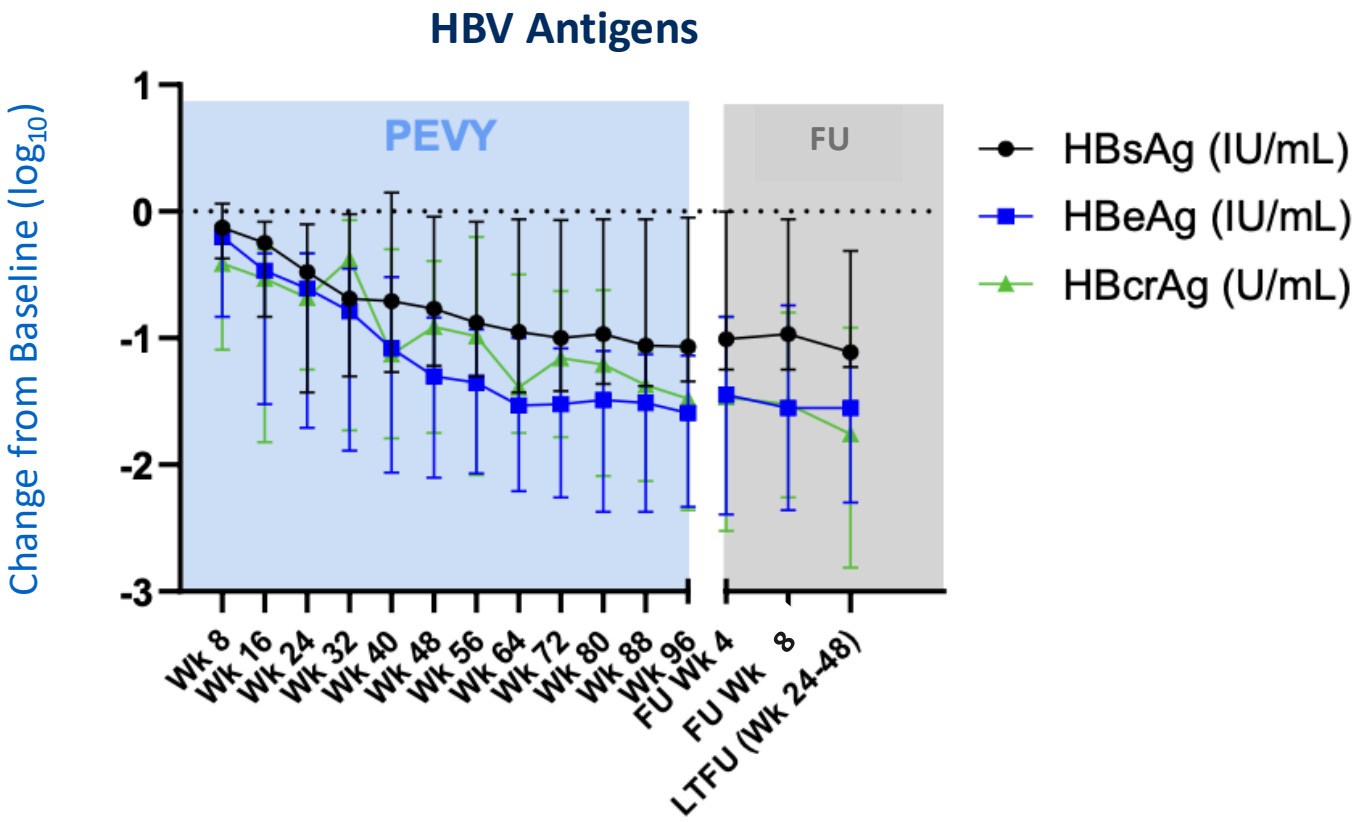
Yuen, M-F. et al; AASLD 2025.

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 and ≥24-Week NA Follow Up in HBeAg+ Participants



Median log reduction from baseline at different timepoints for HBsAg, HBeAg and HBcrAg

Mak, et al. EASL 2026. FU= Follow up. LTFU = long term follow up. Follow up period treatment with ETV only.

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

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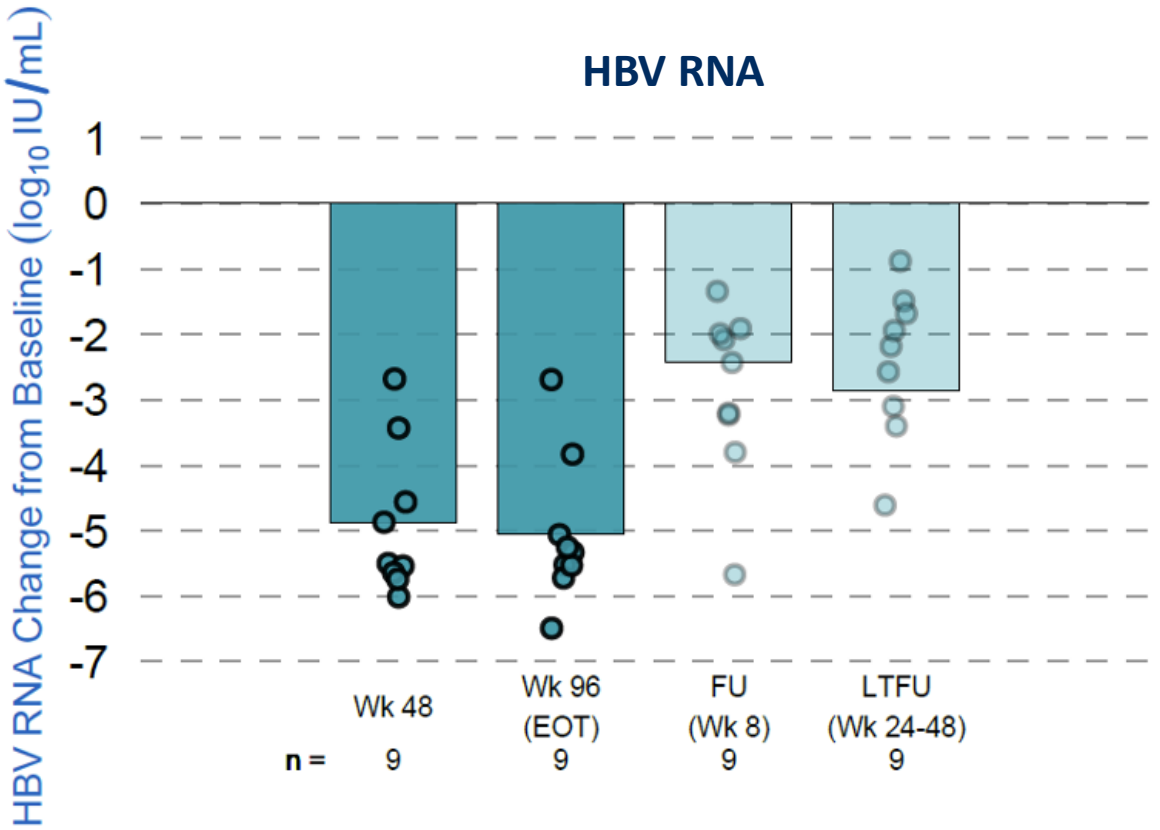
Sustained Reductions in HBV RNA After LTFU



EASL 2026 Lung Yi Loey Mak WED 588. LTFU = long term follow up. Follow up period treatment with ETV only.

Sustained HBV RNA observed during LTFU

Likely because of reduction of cccDNA reservoir

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SUPREME – Phase 2 Study Design

Primary Analysis at 48 Weeks; Extension Period Analysis at 96 Weeks

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.

Enrollment completed July 2026;

Topline Phase 2 data expected in late Q3 2027

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Pevifoscorvir Sodium

- Commercial Opportunity
- Upcoming Milestones

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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.

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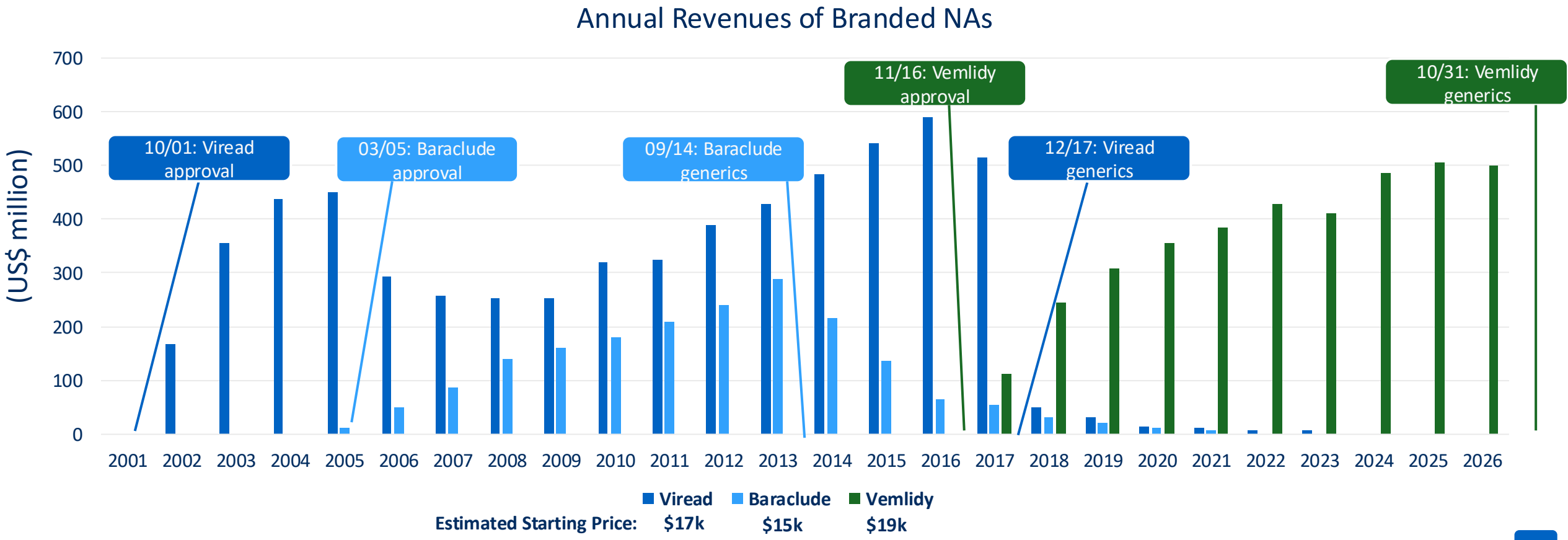
Estimated ~$1B US market opportunity annually with greater potential worldwide sales; not including potential market opportunity as part of a functional cure regimen

Consistent US Annual Revenues from Branded NAs Over Time

Despite Available Generics



Aligos data on file; GlobalData. Viread generic = TDF, Baraclude generic = ETV.

Global branded revenue also consistently performed well, generating >~1B in revenues ~30% of years since 2001

ALIGOS THERAPEUTICS

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

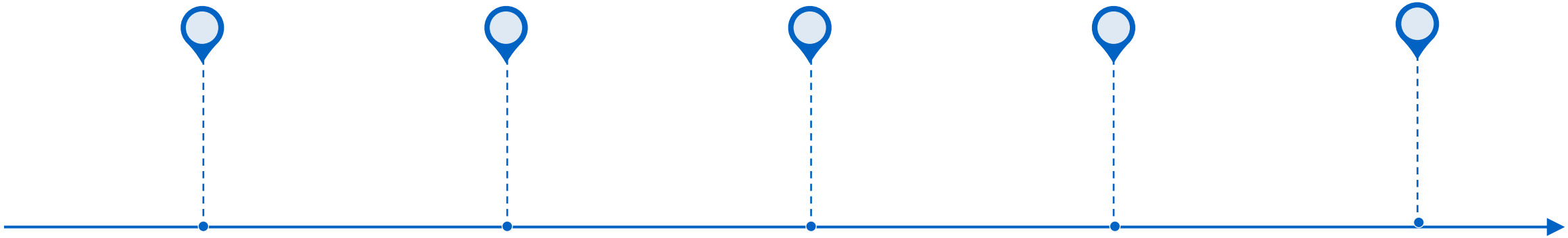
Aligos to receive an upfront payment of $25M USD and is entitled to up to $420M USD in clinical, regulatory, and sales milestones with tiered, high single-digit royalties

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 expected to enter the clinic in Q3 2026

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

✓ **July 2026:** Completion of enrollment

Late Q3 2027: Topline Phase 2 expected

ALIGOS THERAPEUTICS

ALG-170675

OVERVIEW

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ALG-170675

A Potential Best-in-Class ASO for Functional Cure of Chronic Hepatitis B Virus Infection

Discovered in house in a research collaboration with Amoytop, with rights in Greater China. Novel IP filed

Showed improved RNase H mediated in vivo activity over GSK-836 in mice. Similar hTLR8 agonist activity to GSK-836 was observed in vitro and in vivo

Preclinical Profile: Demonstrated advantages when compared with GSK-836 with better in vitro safety; enhanced liver exposure, liver to kidney ratio, and improved in vivo efficacy in mice

Aligos ASO nonclinical studies showed additive to synergistic effects when combined with CAM-E

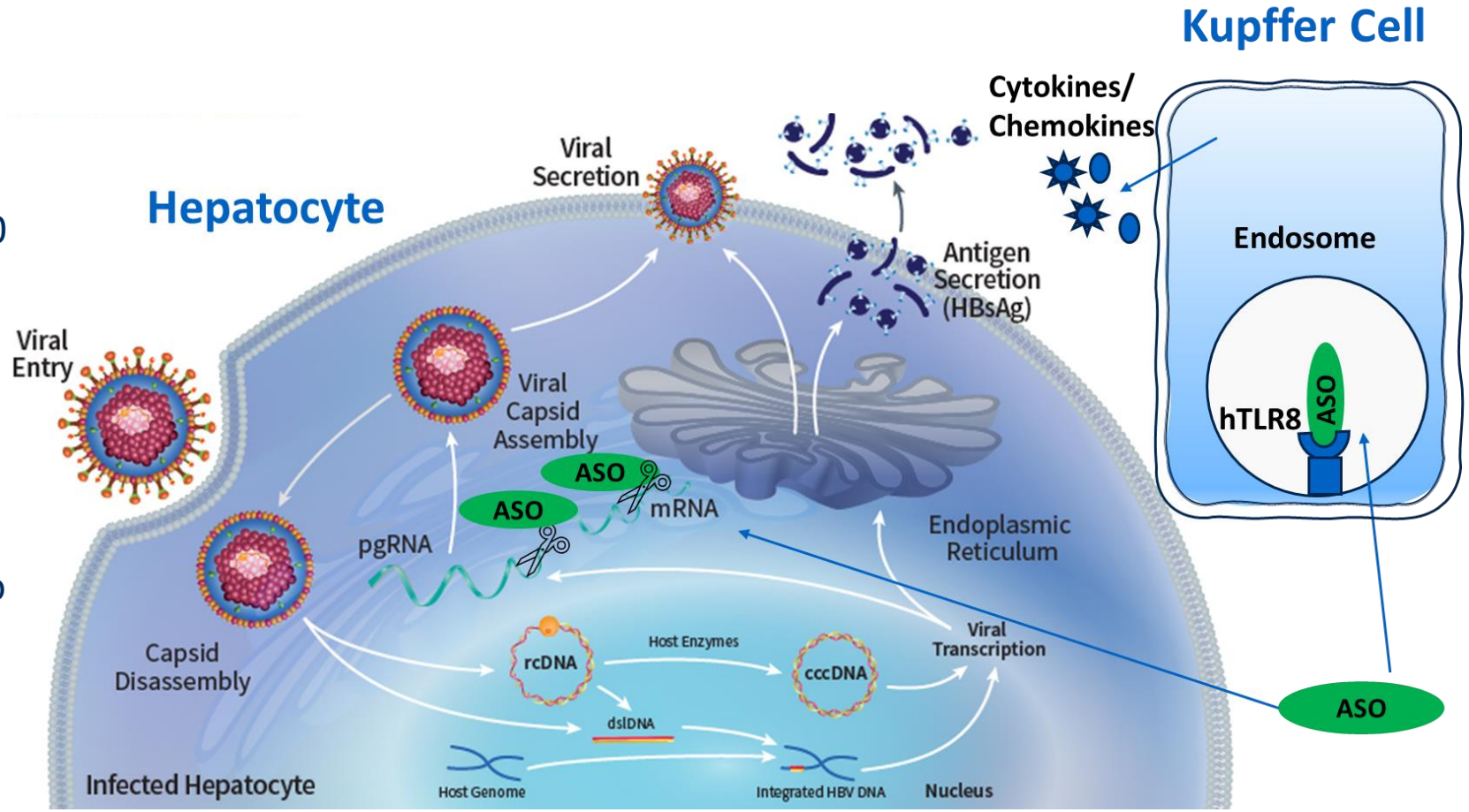
Upcoming Milestones: Amoytop plans to initiate a Phase 1 study in China in 3Q2026

ALIGOS THERAPEUTICS

ALG-170675 for Chronic HBV Infection

Proposed Antisense Oligonucleotide Mechanism of Action

- Data with other ASOs currently in clinical trials have demonstrated that those participants with HBsAg ≤ 3,000 IU/ml achieve a ~19% functional cure[1]
- ALG-170675 is an investigational ASO targeting HBV RNA to inhibit replication and antigen production[2]
  - Includes TLR8 activation to enhance immune-mediated viral control
  - Designed to reduce HBV DNA and HBsAg levels



1. Hou, et al. *N Engl J Med*. 2026. 2. Hong, et. Al. AASLD 2025.

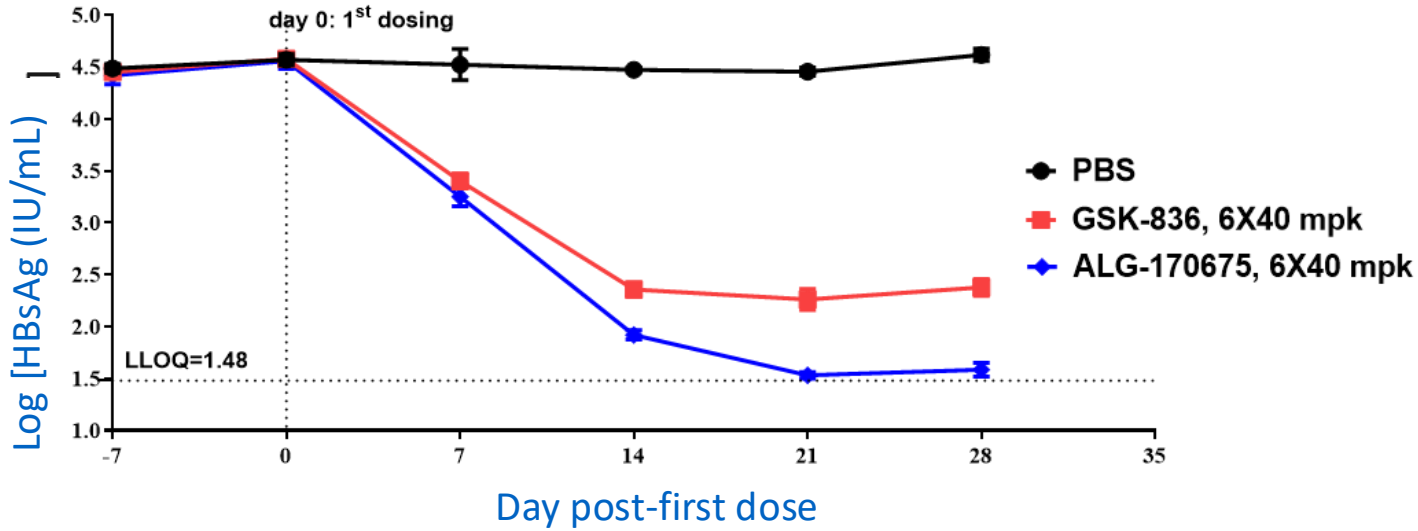
ALIGOS THERAPEUTICS

ALG-170675 Showed Improved RNase H Mediated In Vivo Activity with Improved Liver Exposure Compared to Bepirovirsen

AAV-HBV mice were dosed with PBS, ALG-170675 (40 mg/kg) and GSK-836 (40 mg/kg) on days 0, 3, 7, 10, 14, 21

ASO Mechanistic Data

HBsAg Level in Plasma



Hong, et. Al. AASLD 2025.

Demonstrated improved properties over GSK-836 (bepirovirsen)

ALG-170675 Demonstrated TLR8 Agonist Activity with Minor TLR9 Activation

HEK-Blue TLR Cell Lines

	hTLR7 Emax Fold vs. PBS	hTLR8 Emax Fold vs. PBS	hTLR9 Emax Fold vs. PBS	mTLR8 Emax Fold vs. PBS	Null Emax Fold vs. PBS
GSK-836	**1.07**	**1.89**	**1.29**	**1.06**	**1.12**
ALG-170675	**1.03**	**1.87**	**1.18**	**1.06**	**1.07**

Hong, et. Al. AASLD 2025.

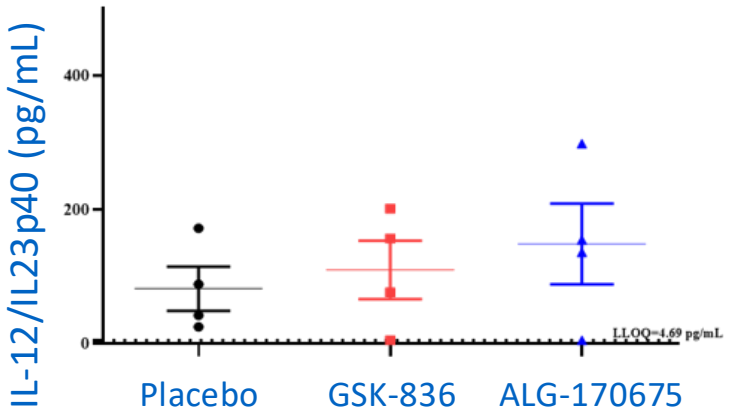
Similar TLR8 agonist activity to GSK-836 (bepirovirsen)

ALG-170675 Showed Similar Cytokine Induction Profiles to Bepirovirsen

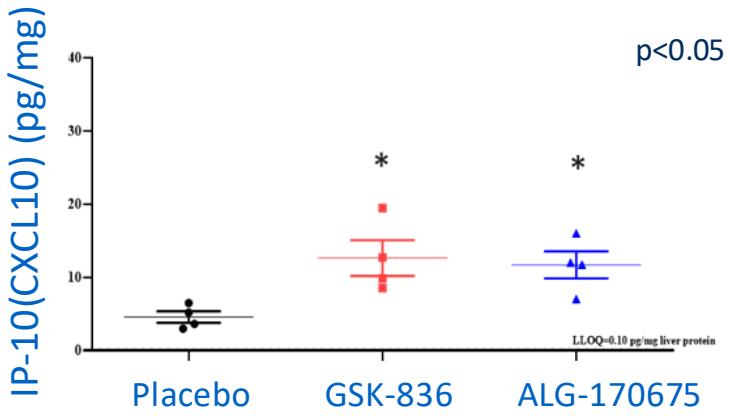
hTLR8 knock-in mice were dosed with single 40 mg/kg of each ASO subcutaneously at hour 0, cytokines and chemokines were assayed at 4 hours post dosing

Immuno-Activation Data

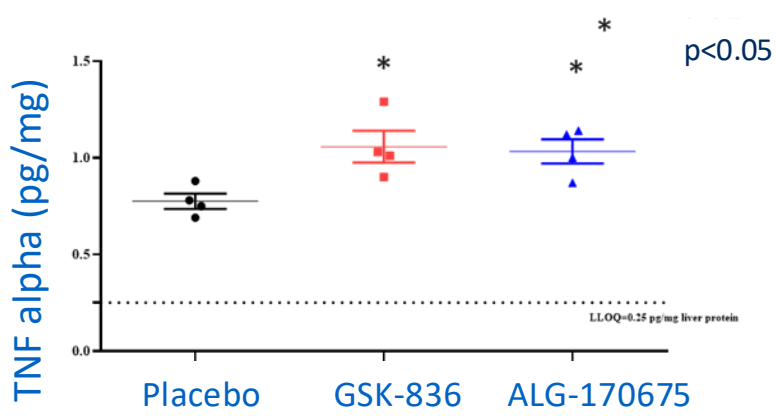
IL-12/IL23p40 Level in Mouse Plasma



IP-10(CXCL10) Level in Mouse Liver



TNF Alpha Level in Mouse Liver

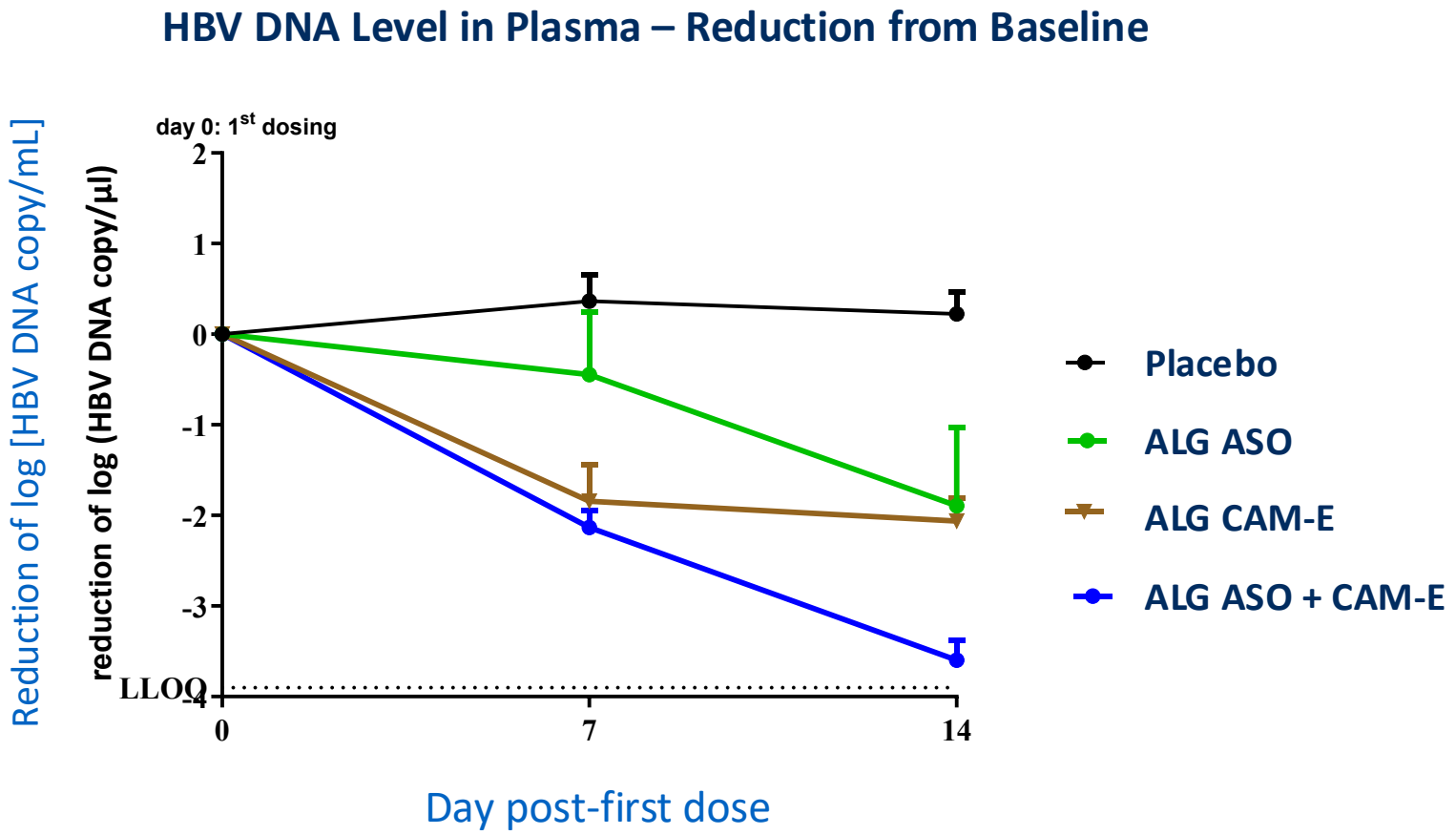


Hong, et. Al. AASLD 2025.

Consistent cytokine induction across key markers similar to GSK-836 (bepirovirsen)

Nonclinical Studies Showed Additive to Synergistic Effects When Combined with CAM-E Prodrug

AAV-HBV Mouse Model



Hong, et al. EASL 2026.

Potential to explore clinical combination studies for functional cure of chronic HBV infection

ALG-170675 Potential Upcoming Clinical Milestones

Data sharing/co-development allows Aligos to utilize the data from the Amoytop China studies

Concurrent MAD & study in HBV participants

3Q2026: Phase 1 SAD/MAD study in healthy participants (China)

AMOYTOP BIOTECH

2028: Phase 2 study

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28-day study to define dose for Phase 2 study

4Q2026: Phase 1 study in participants with HBV (China)

AMOYTOP BIOTECH

Phase 2 study (China)

AMOYTOP BIOTECH

Per the executed agreement, Amoytop and Aligos have agreed to share data on ALG-170675. Each party is responsible for their own development costs.

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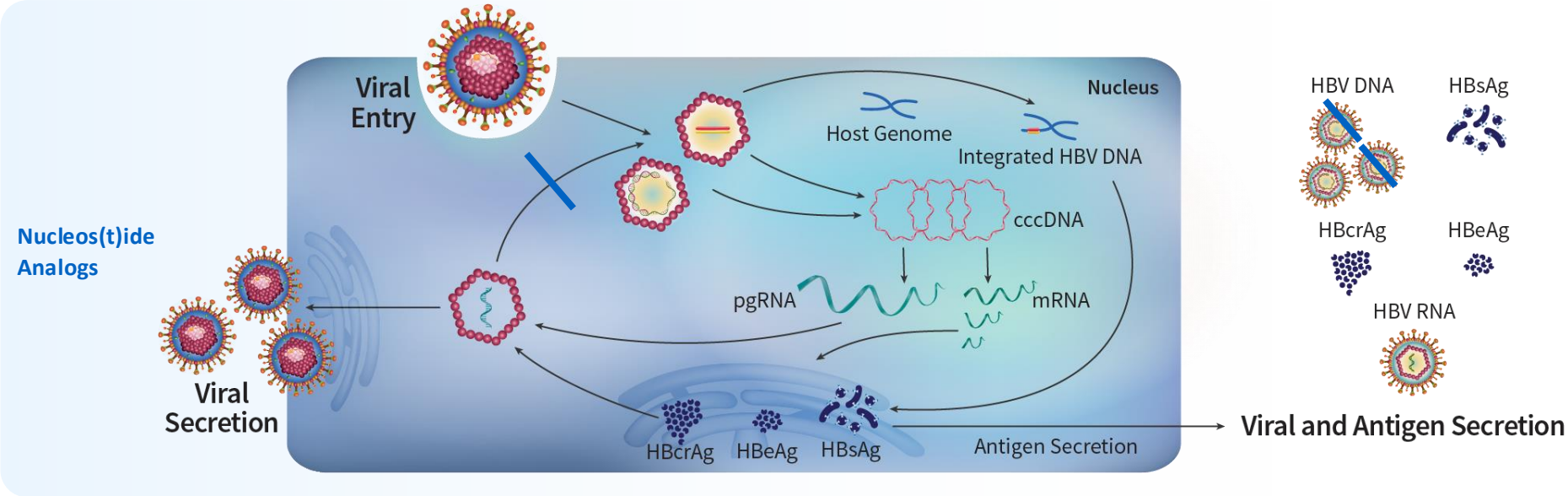
MOA: NAs



Key Attributes

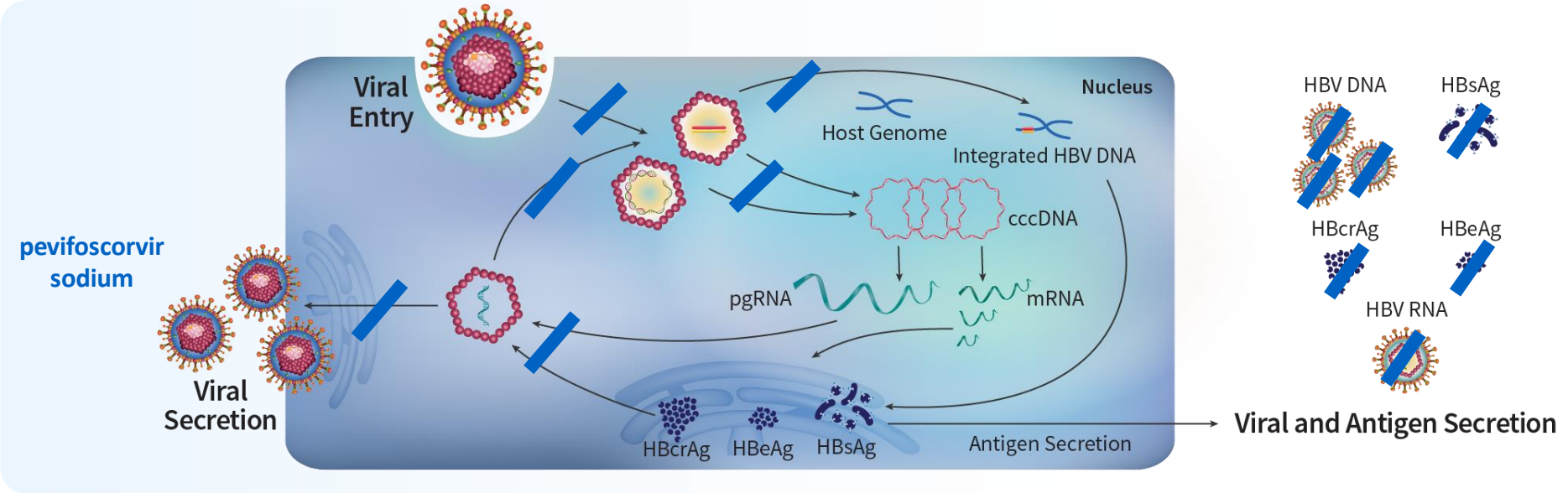
- NAs improve clinical outcomes by controlling HBV replication through targeting HBV polymerase activity, **though they do not**[1,2]**:**
  - Affect cccDNA[2,3]
  - Substantially lower HBsAg and HBcrAg[3]
  - Completely inhibit rcDNA synthesis from pgRNA[1]
  - Lead to HBeAg seroconversion above the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

ALIGOS THERAPEUTICS

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.

ALIGOS THERAPEUTICS

Pevifoscorvir Sodium: A Favorable Profile

In vitro: potency vs. first generation CAM-Es

Compound	Current Status	HBV DNA reduction (EC_{50} nM)	Cell Type	Knockdown of HBV Antigens in the Clinic as Monotherapy	
Aligos pevifoscorvir sodium[1]	**Phase 2**	**0.63**	**HepG2.117**	✓	2nd Generation
		0.53	**HepG2.2.15**	✓	
Assembly ABI-4334	Phase 1 – Seeking Collaboration, GILD Passed	1.2	AD38	X	
Assembly ABI-H0731 (vebicorvir)	Discontinued	172	AD38	X	
Janssen JNJ-56136379 (bersacapavir)	Discontinued	54	HepG2.117	X	1st Generation
Arbutus AB-836	Discontinued	10	HepDE19	X	
Canocapavir (ZM-H1505R)	Phase 3 (China Only)	10-12	HepG2.115	X	
Neracorvir (GST-HG141)	Phase 3 (China Only)	8.16	HepG2.115	X	

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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.