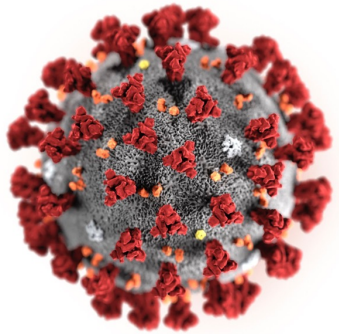
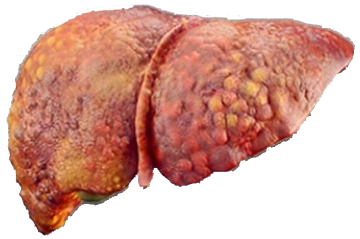
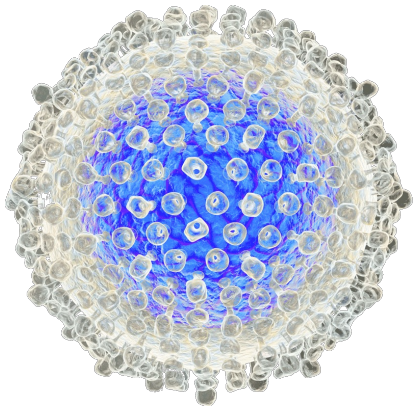
ALIGOS
THERAPEUTICS







CHB KOL Event with Mark Sulkowski, MD

Professor of Medicine at the Johns Hopkins University School of Medicine and the Director of the Division of Infectious Diseases at Johns Hopkins Bayview Medical Center

July 2024

Disclosures

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. All statements other than statements of historical facts contained in this presentation, including statements regarding our future results of operations and financial position, business strategy, prospective drugs and drug candidates, the potential scope, progress, results and costs of developing our drug candidates or any other future drug candidates, the potential market size and size of the potential patient populations for our drug candidates, the timing and likelihood of success of obtaining drug approvals, ability to maintain existing, and establish new, strategic collaborations, licensing or other arrangements, plans and objectives of management for future operations, the scope of protection we are able to establish and maintain for intellectual property rights covering our drug candidates, future results of anticipated drugs and drug candidates, and the impact of global events and other macroeconomic conditions on the business are forward-looking statements. These statements involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. Because forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified and some of which are beyond our control, you should not rely on these forward-looking statements as predictions of future events. The events and circumstances reflected in our forward-looking statements may not be achieved or occur and actual results could differ materially from those projected in the forward-looking statements. Except as required by applicable law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise. For a further description of the risks and uncertainties that could cause actual results to differ from those anticipated in these forward-looking statements, as well as risks relating to the business of Aligos Therapeutics in general, see Aligos' Annual Report on Form 10-Q filed with the Securities and Exchange Commission on May 7, 2024, and its future periodic reports to be filed with the Securities and Exchange Commission. Except as required by law, Aligos Therapeutics undertakes no obligation to update any forward-looking statements to reflect new information, events or circumstances, or to reflect the occurrence of unanticipated events.

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

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

Agenda

1	**Aligos overview**	**Lawrence M. Blatt, Ph.D., MBA** Chairman, President, & Chief Executive Officer Aligos Therapeutics
2	• **Chronic Hepatitis B (CHB) treatment landscape & unmet need** • **ALG-000184 profile for CHB** • **Regulatory pathway and development plans**	**Mark Sulkowski, M.D.** Professor of Medicine at the Johns Hopkins University School of Medicine and the Director of the Division of Infectious Diseases at Johns Hopkins Bayview Medical Center
3	**Moderated Q&A session**	**Corey Davis, Ph.D.** Managing Director, LifeSci Advisors, LLC
4	**Concluding remarks**	**Lawrence M. Blatt, Ph.D., MBA** Chairman, President, & Chief Executive Officer Aligos Therapeutics

Aligos
Investment Thesis

- **Aligos has decades of drug development experience in medicinal chemistry and liver/viral diseases**
- **ALG-055009 for Metabolic Dysfunction-Associated Steatohepatitis (MASH)**
  - Thyroid hormone receptor beta (THR-β) is a clinically validated mechanism (MDGL)
  - ALG-055009 has enhanced pharmacologic properties vs. competitor THR-β agonists
  - Phase 1 data: linear and non-variable PK, well-tolerated, with expected thyromimetic effects
  - Phase 2a enrollment complete with topline data expected in early Q4 2024
- **ALG-000184 for Chronic Hepatitis B (CHB)**
  - ALG-000184 (CAM-E) has enhanced pharmacologic properties and is a best/first-in-class molecule
  - Demonstrated greater DNA suppression compared to standard of care (NAs)
  - Dosing ongoing in 96-week Ph1b cohorts with interim readouts expected at APASL, EASL, AASLD
  - Clear regulatory path forward for chronic suppressive therapy with superiority label
  - Phase 2 enabling activities ongoing; planned Phase 2 IND filing in Q1 2025

As of 3/31/24 - Cash, cash equivalents and investments were $112.7M. Projected runway through the end of 2025

ALG-000184
A Potential Best-in-Class CAM-E for CHB

- **Initial IP licensed from the lab of Dr. Raymond Schinazi at Emory University; issued US patent expires in 2040[1]**
- **Enhanced pharmacology**
  - Picomolar potent
  - Enhanced absorption with high liver uptake
- **Phase 1 highlights** (≤300 mg ALG-000184 ± ETV x ≤72 weeks in untreated CHB)
  - PK: dose proportional, low-moderate variability
  - Safety: no safety signals observed
  - Antiviral activity: best-in-class reductions seen in HBsAg, HBeAg, HBcrAg, HBV DNA & RNA
  - Dosing x ≤96 weeks ongoing (through 2025)
- **Phase 2**
  - Clear regulatory path forward for chronic suppressive therapy with superiority label
  - Enabling activities underway; planned Phase 2 IND filing in Q1 2025

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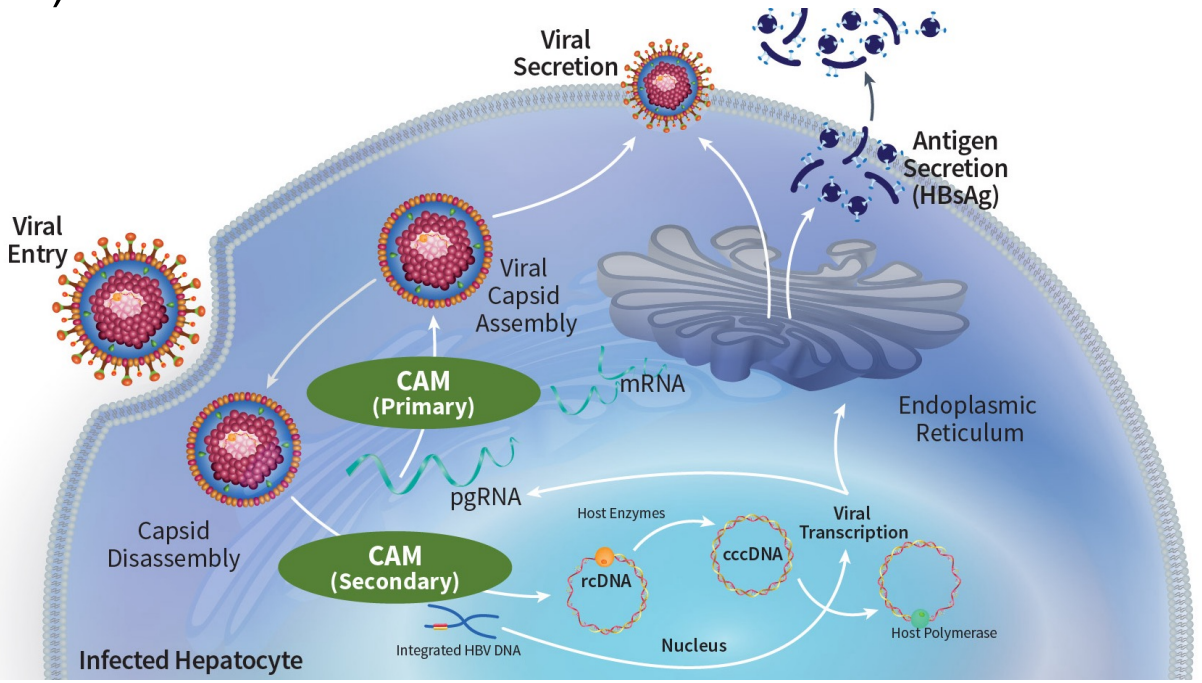
[1]Not including any patent term extension.

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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Rethinking CHB Treatment: A New Era

The industry has learned from the issues of first-generation investigational mechanisms such as siRNA, ASO, NAPs, CAMs, immunomodulators, and therapeutic vaccines. Functional cure is a difficult pathway

The DNA suppression observed with ALG-000184 to date is greater vs. SOC, and is an approvable, de-risked regulatory pathway that can meaningfully help CHB patients (chronic suppressive therapy)

We have solved the potency issues previously seen with CAMs, leading to greater DNA suppression and clinical demonstration of the secondary mechanism

The importance of all relevant biomarkers has not been a key focus for the space. Treating CHB patients is more than reductions in HBV DNA and HBsAg; it is also HBV RNA, HBcrAg, and HBeAg

ALG-000184 is paving the way for the future of CHB treatments
First potential new mechanism advancing towards approval for chronic suppression in CHB in 25+ years

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Mark Sulkowski, MD Biography

- Mark Sulkowski, MD, is a Professor of Medicine, the Senior Associate Dean for Clinical Trials, and the Founding Director of the Office of Clinical Trials at the Johns Hopkins University School of Medicine. He also serves as the Director of the Division of Infectious Diseases at the Johns Hopkins Bayview Medical Center, a Deputy Director for the Johns Hopkins Institute for Clinical and Translational Research, and the Medical Director of the Viral Hepatitis Center in the Divisions of Infectious Diseases and Gastroenterology/Hepatology in the Department of Medicine. He received his MD from Temple University School of Medicine, Philadelphia, PA, pursued training in Internal Medicine at Duke University School of Medicine, Durham, NC, and completed his Fellowship in Infectious Diseases at the Johns Hopkins University School of Medicine.

Disclosures

- The terms of these arrangements are being managed by the Johns Hopkins University in accordance with its conflict of interest policies
- Research (JHU): Abbvie, GSK, Janssen, Vir, Virion
- Scientific advisory board: Abbvie, Aligos, Arbutus, Galapagos, Gilead, GSK, Precision, Vir, and Virion
- DSMB: Gilead, Immunocore
- Editorial board: Journal of Viral Hepatitis (Wiley)

Hepatitis B was discovered in 1963

Clinical Science

A "New" Antigen in Leukemia Sera

The "Australia antigen" is found in the sera of some normal individuals from foreign populations. The total absence of the antigen from the sera of normal United States subjects and its relatively high frequency in acute leukemia suggests that the presence of the antigen may be of value in the diagnosis of early acute leukemia. Whether the antigen results from or precedes the leukemia process remains to be seen.

Baruch S. Blumberg, MD, Harvey J. Alter, MD, and Sam Visnich

Hepatitis B surface antigen

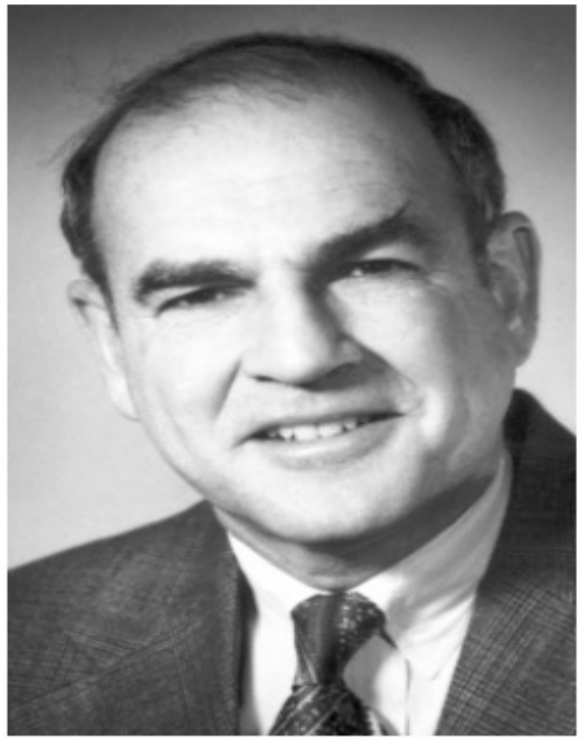


Photo from the Nobel Foundation archive.

Baruch S. Blumberg

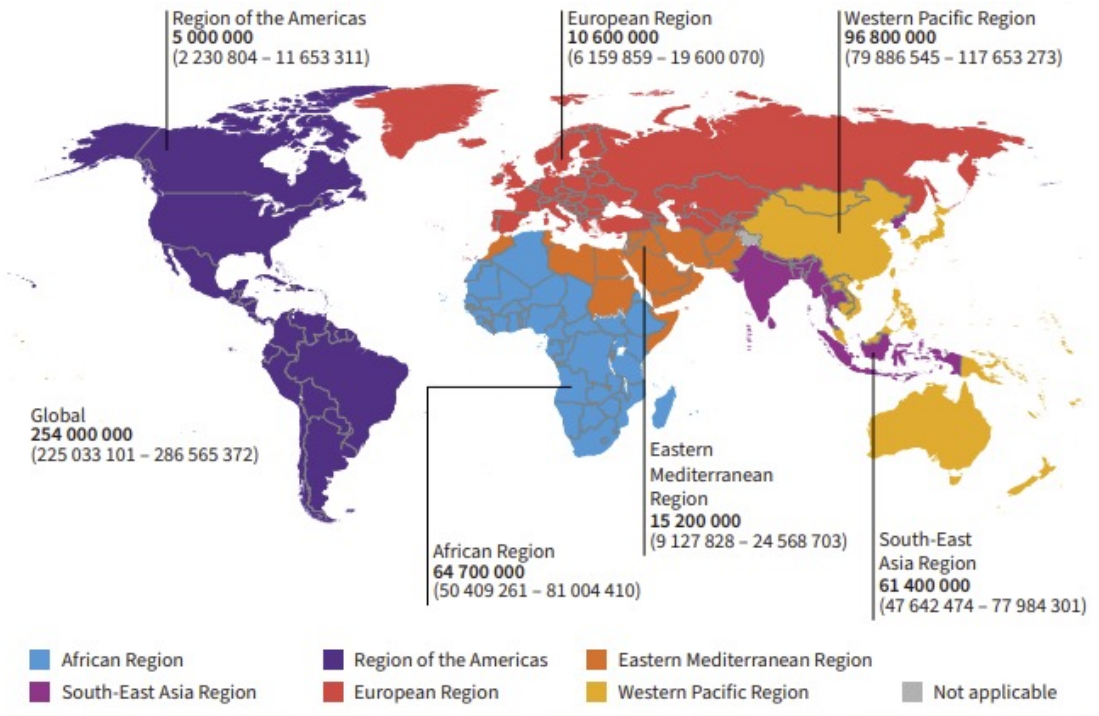
Prize share: 1/2

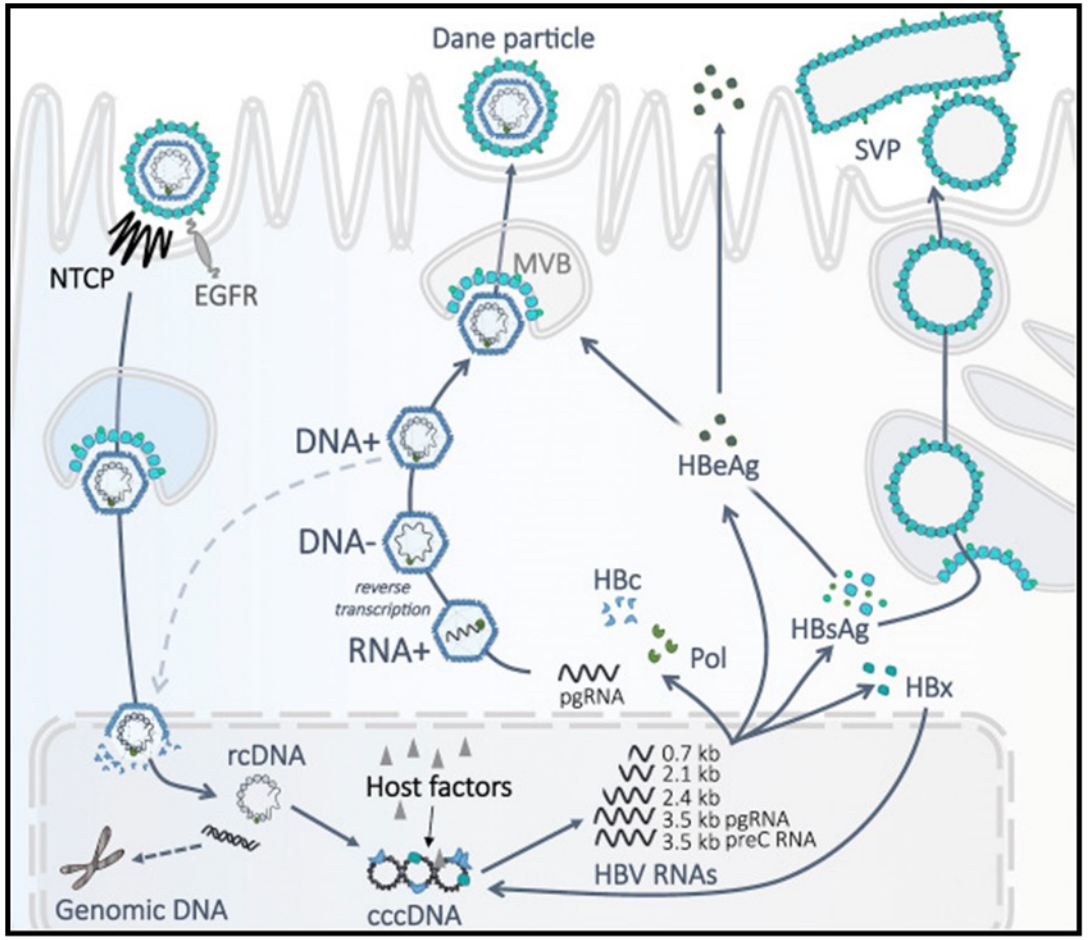
The Nobel Prize in Physiology or Medicine 1976

https://www.nobelprize.org/prizes/medicine/1976/blumberg/lecture/; B.S. Blumberg, H.J. Alter, S. Visnich. A "new" antigen in leukemia sera JAMA, 191 (1965), pp. 541-546

254 million people are living with Hepatitis B

Most are not diagnosed (13%) or treated (3%), and cure is difficult





- 75% of people live in the African, SE Asia, and Western Pacific regions
- HIV coinfection is common
- 1.1 million deaths annually

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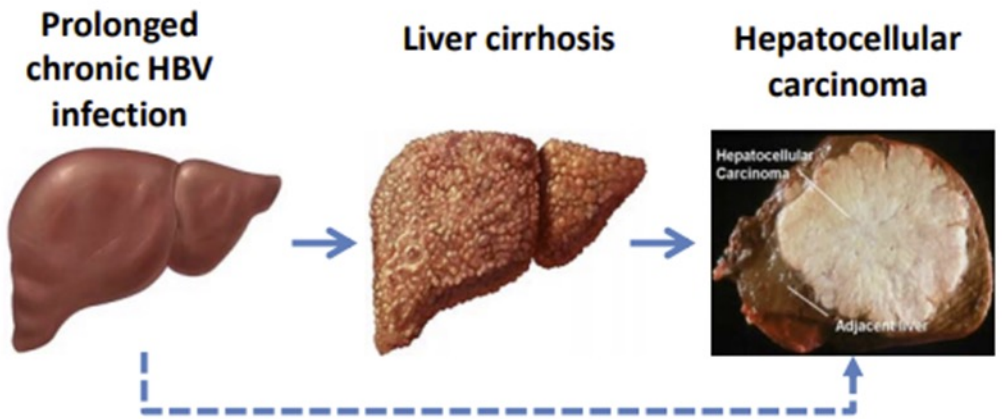
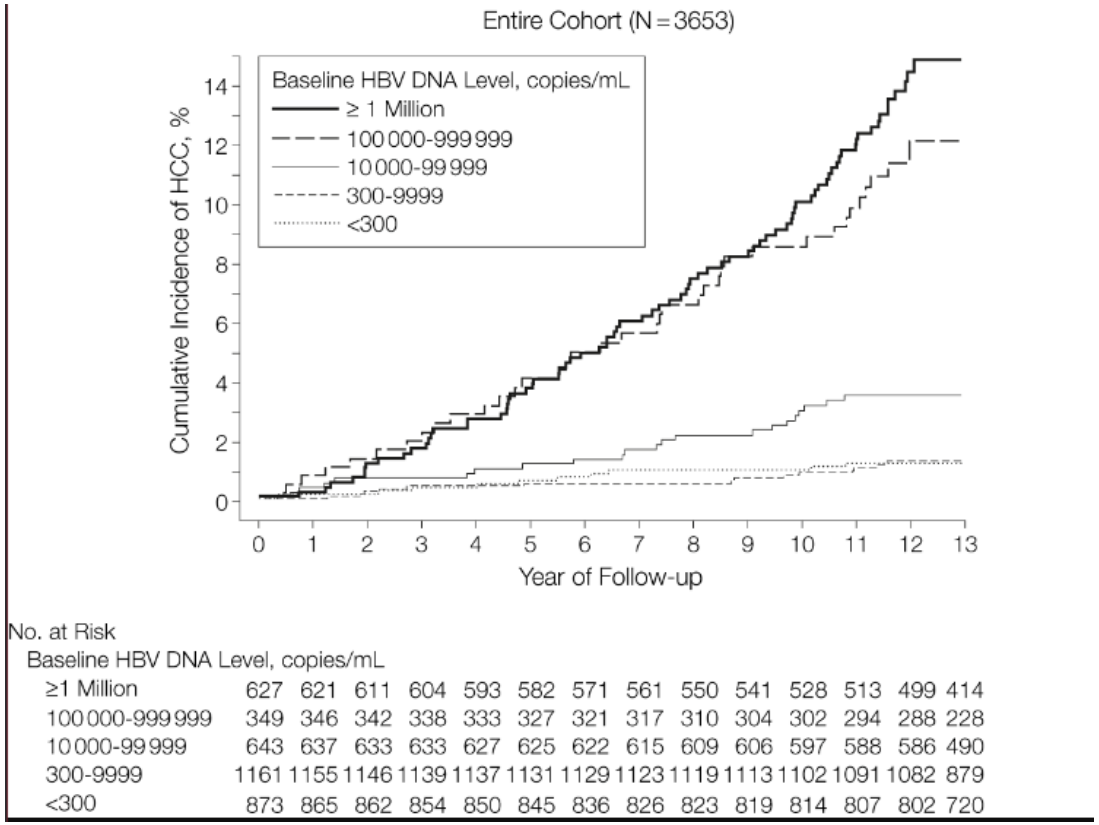
https://www.who.int/publications/i/item/9789240091672

Clinical Consequences of chronic hepatitis B



HCC Incidence by Baseline Serum HBV DNA Level



REVEAL-HBV Study

- 3653 adults with HBsAg+ recruited in Taiwan 1991
- HCC risk: Older age, alcohol, HBeAg+, cirrhosis (aHR 9.1) and HBV DNA at entry (not ALT)

Chen et al. JAMA 2006

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Incomplete HBV DNA suppression is associated with hepatocellular carcinoma in patients with HIV-HBV

- Longitudinal cohort study of 8,354 persons with HIV/HBV in NA-ACCORD
  - 1995 to 2016
  - Outcome = incident HCC
- Risk of HCC increased with HBV DNA level (n= 3,054; incident HCC 30)
  - > 200 IU/mL, aHR, 2.7
  - > 200,000, aHR 4.34

Kim HN et al. Hepatology March 2021

Risk of HCC Associated With Time-Updated HBV DNA Level and Time-Updated Detectable HIV and HBV Status (n= 5,316; 87 Incident HCC)

Viral Suppression	N	HCC	aHR (95% CI)
Both HIV and HBV undetectable	3494	42	Reference
HIV detectable, HBV undetectable	1881	2	0.27 (0.06-1.14)
HIV undetectable, HBV detectable	2835	27	1.77 (1.07-2.92)
Both HIV and HBV detectable	2480	16	2.21 (1.17-4.18)

Sustained HBV suppression with HBV-active ART for ≥1 year was associated with a 58% reduction in HCC risk

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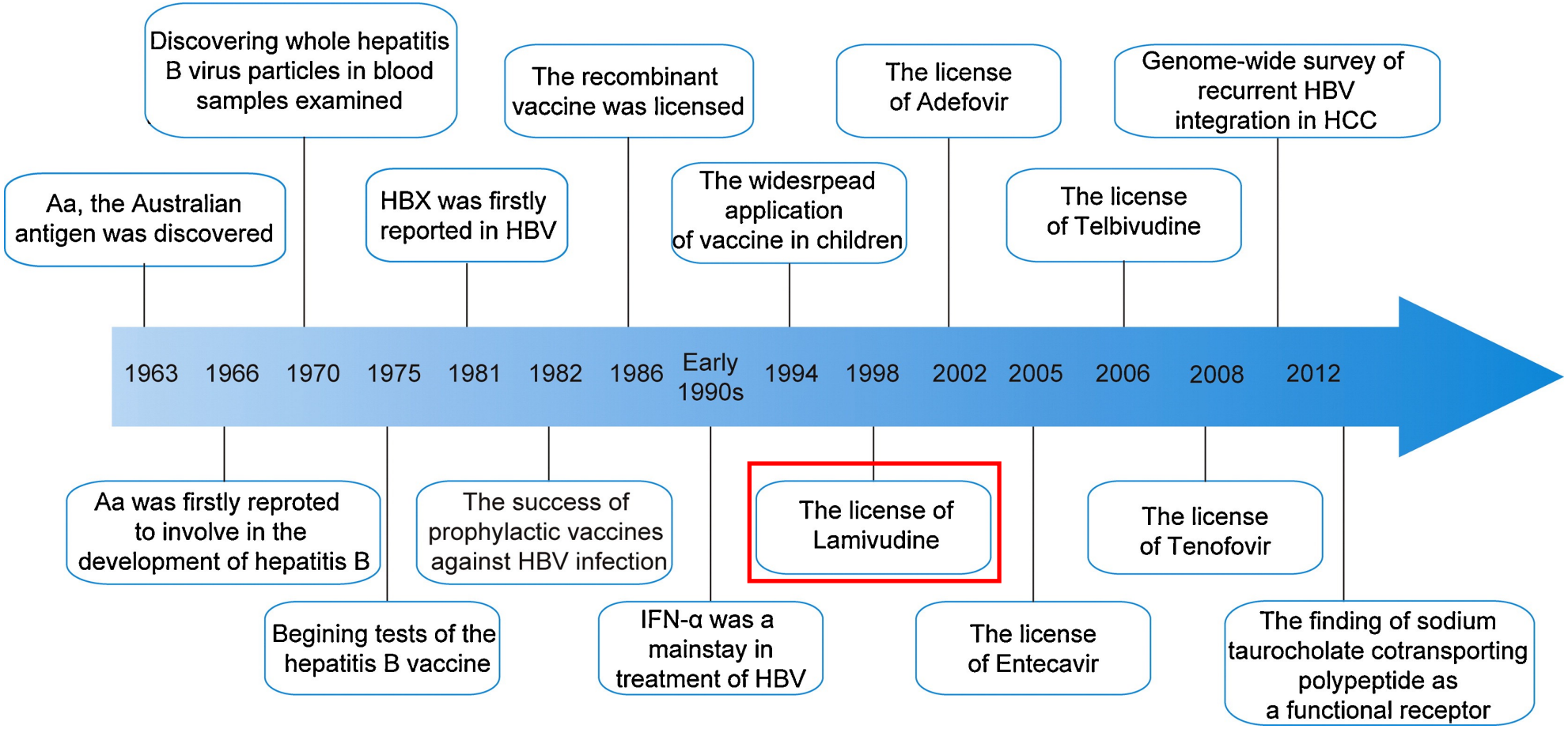
No novel HBV antivirals since lamivudine, 1998



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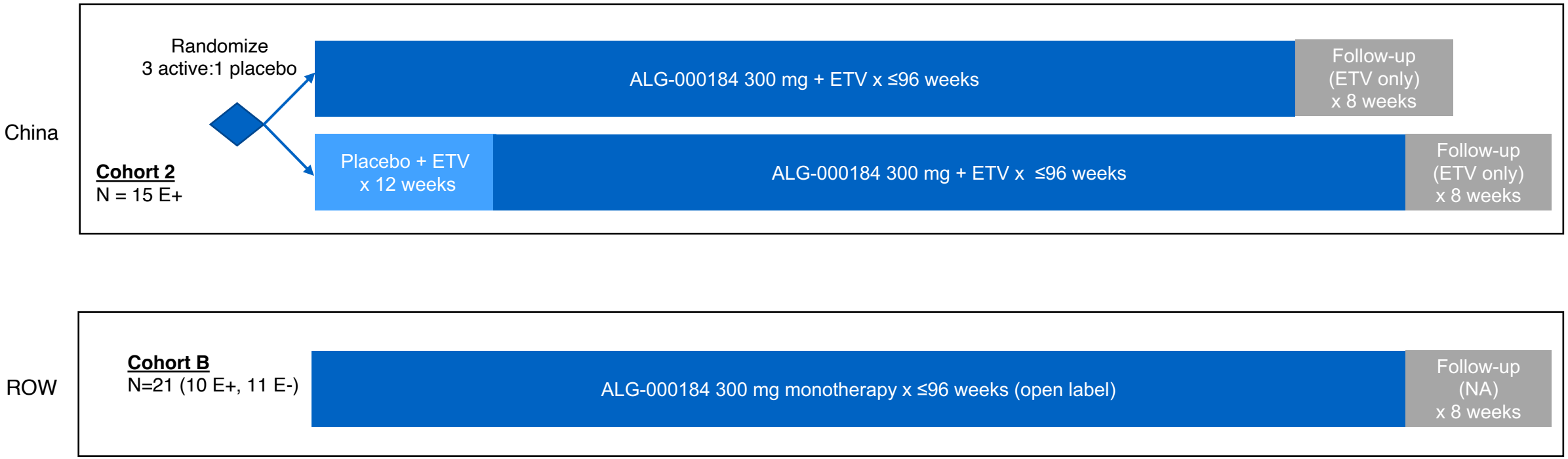
Liu et al; https://doi.org/10.1016/j.biocel.2013.06.017.

ALG-000184-201 – Long Term Dosing in CHB Subjects
Part 4 Cohort Designs



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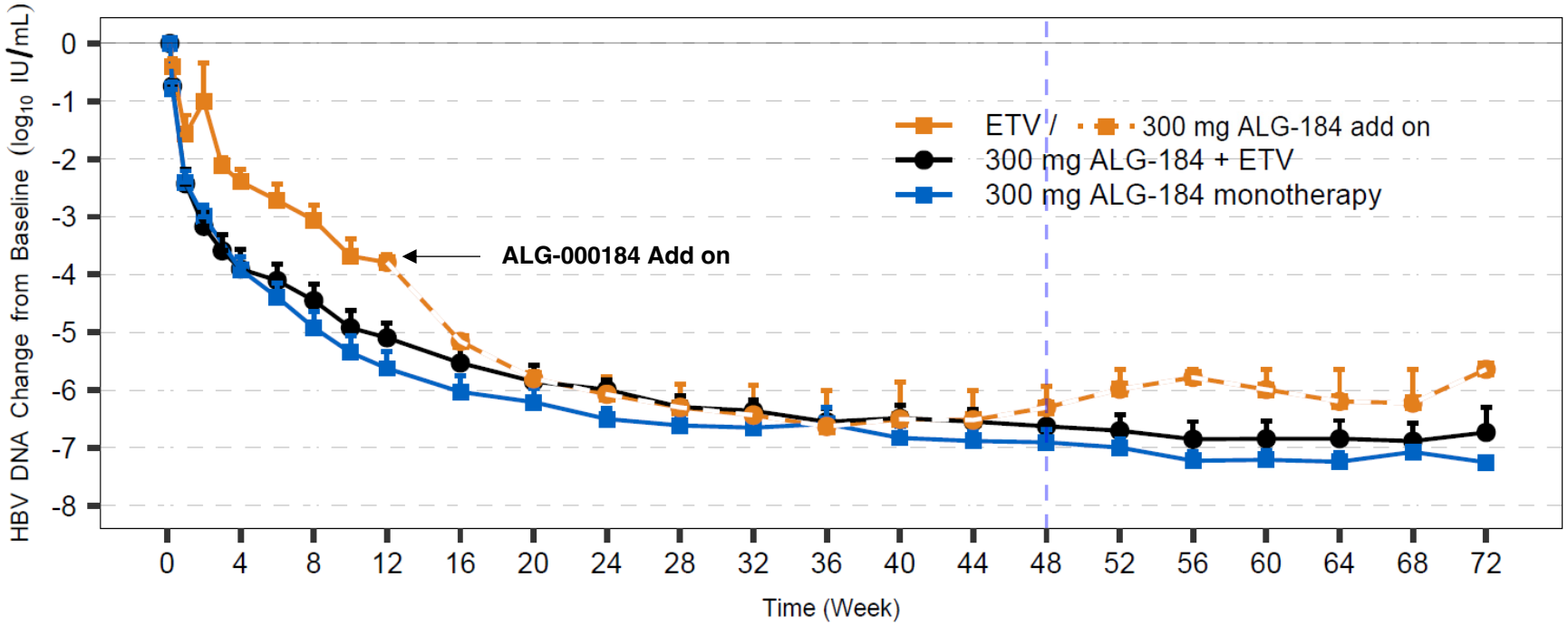
Hou, JL. et al., Poster 1483-C, AASLD (2023).
Yuen, M-F. et al., Late Breaker Poster #5028-C, AASLD (2023).
All cohorts fully enrolled. NCT04536337; ROW:rest of the world.

Antiviral Effect in CHB Subjects (HBeAg+)
HBV DNA Change from Baseline



ETV / 300 mg ALG-184 add on	3	3	3	3	3	2	1
300 mg ALG-184 + ETV	11	9	9	9	8	8	5
300 mg ALG-184 monotherapy	10	10	10	10	10	8	1

Number of subjects at each timepoint

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

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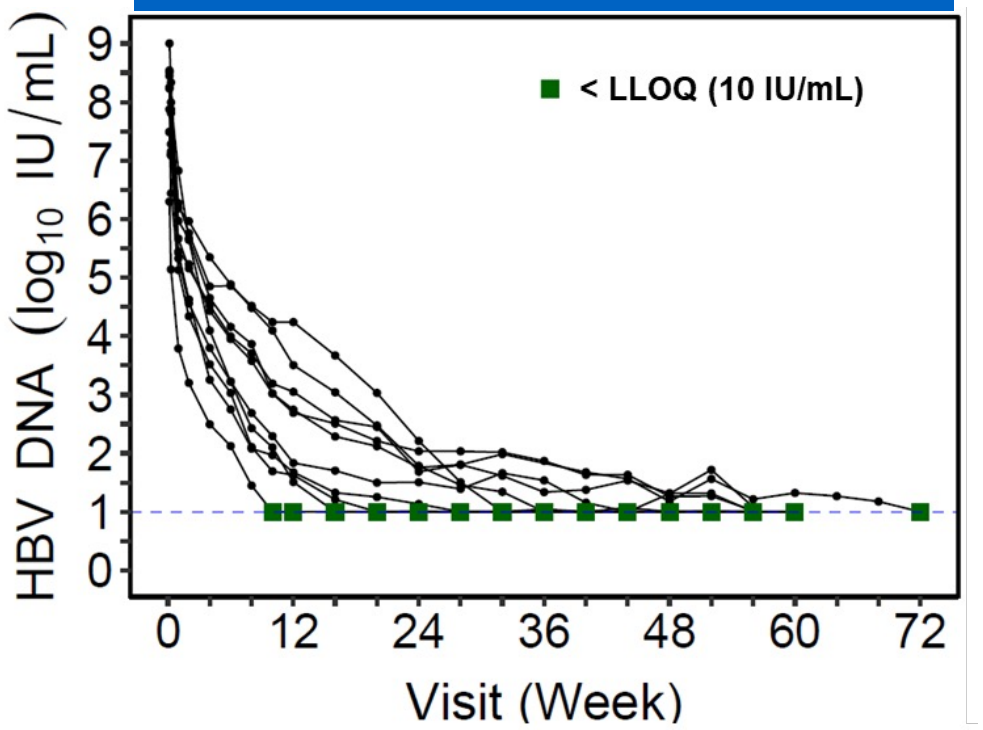
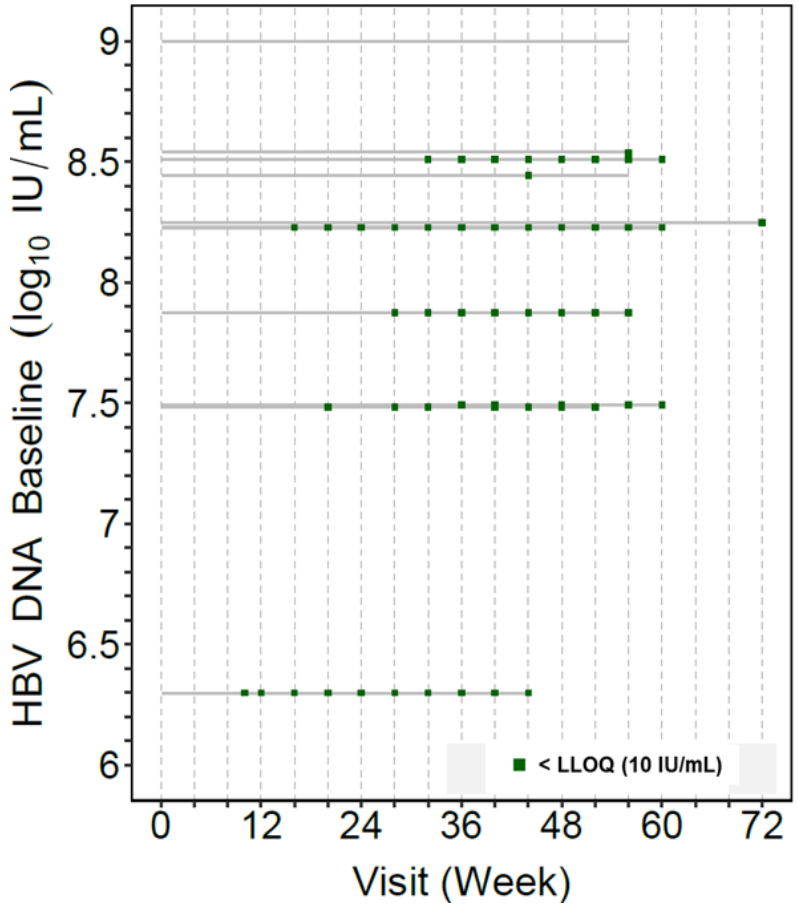
Yuen, M-F. et al.,; EASL 2024.

300 mg ALG-000184 Monotherapy (HBeAg+)
Individual HBV DNA Decline

300 mg monotherapy ≤ 72 weeks



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



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

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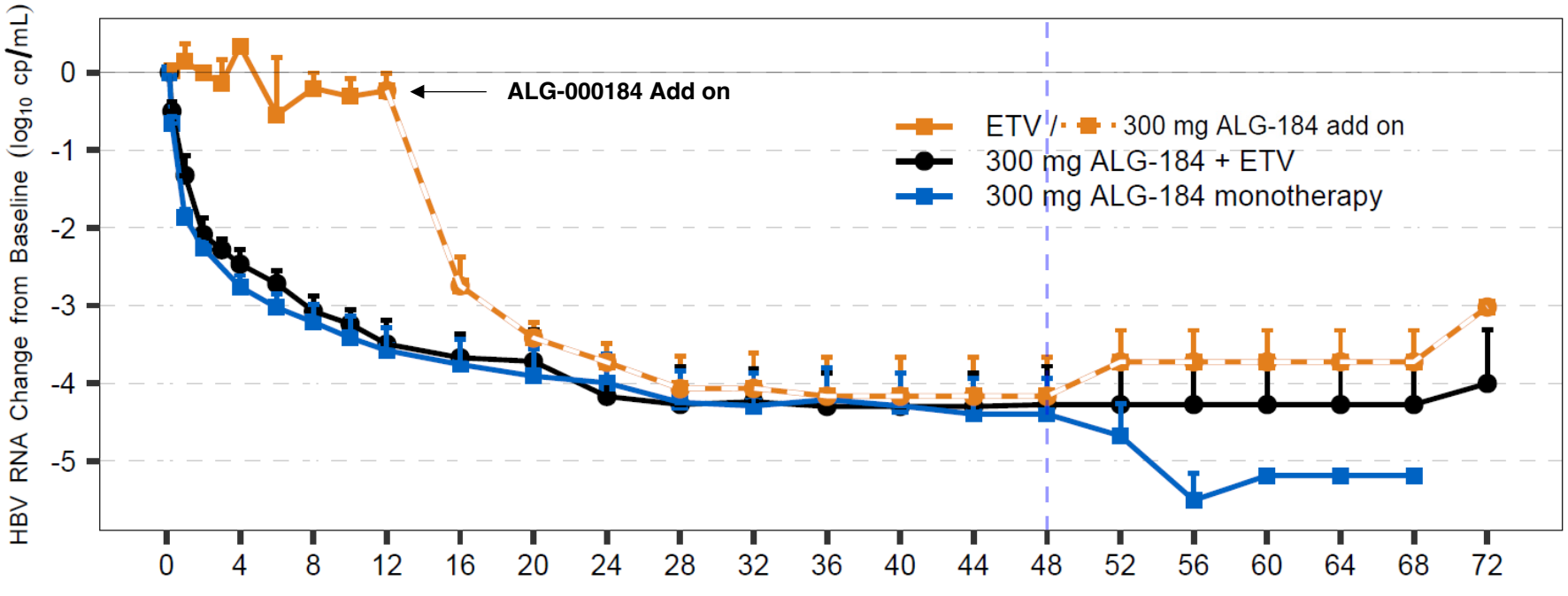
300 mg ALG-000184 ± ETV vs. ETV (HBeAg+)
Mean HBV RNA Over Time



ETV / 300 mg ALG-184 add on	3	3	3	3	3	2	1
300 mg ALG-184 + ETV	11	9	9	9	8	8	5
300 mg ALG-184 monotherapy	10	10	10	10	9	1	

Number of subjects at each timepoint

At Week 12, there was a >3 log_{10} copies/mL RNA decline with ALG-000184 ± ETV vs. no change with ETV
After adding ALG-000184 on top of ETV at Week 12, the RNA decline was similar to the combo regimen
100% (22/22) of subjects experienced HBV RNA < LLOQ by week 40
RNA levels correlated with HCC risk^

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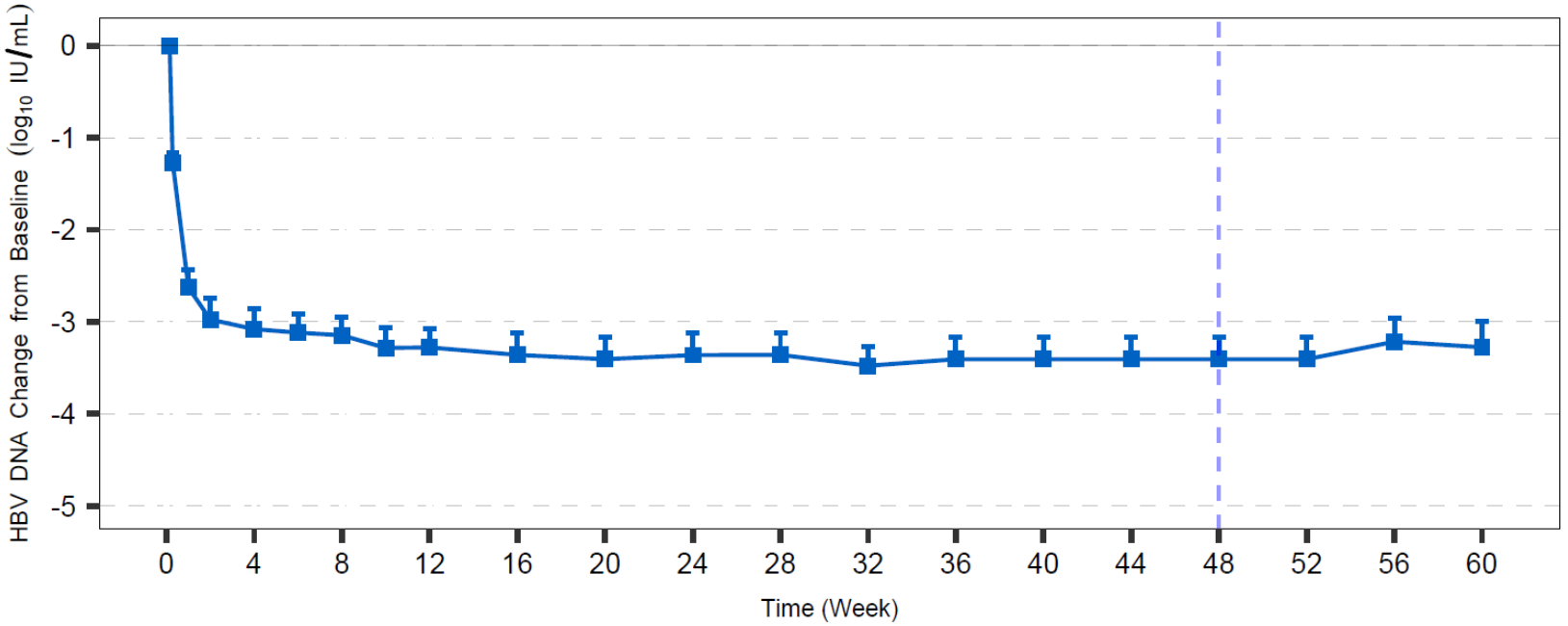
Yuen, M-F. et al.,; EASL 2024.
^Liu et al., J Infect Dis, 2022. Mak et al., J Gastroenterology, 2021. Ding et al. Hepatology, 2021.

Antiviral Effect in CHB Subjects (HBeAg-)
HBV DNA Change from Baseline



Week	0	4	8	12	16	20	24	28	32	36	40	44	48	52	56	60
Total n	11	11	11	11	10	11	11	10	11	11	11	11	11	11	9	5
≤ LLOQ [10 IU/mL]	0	10	10	10	10	11	11	10	11	11	11	11	11	11	9	5

Number of subjects at each timepoint

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

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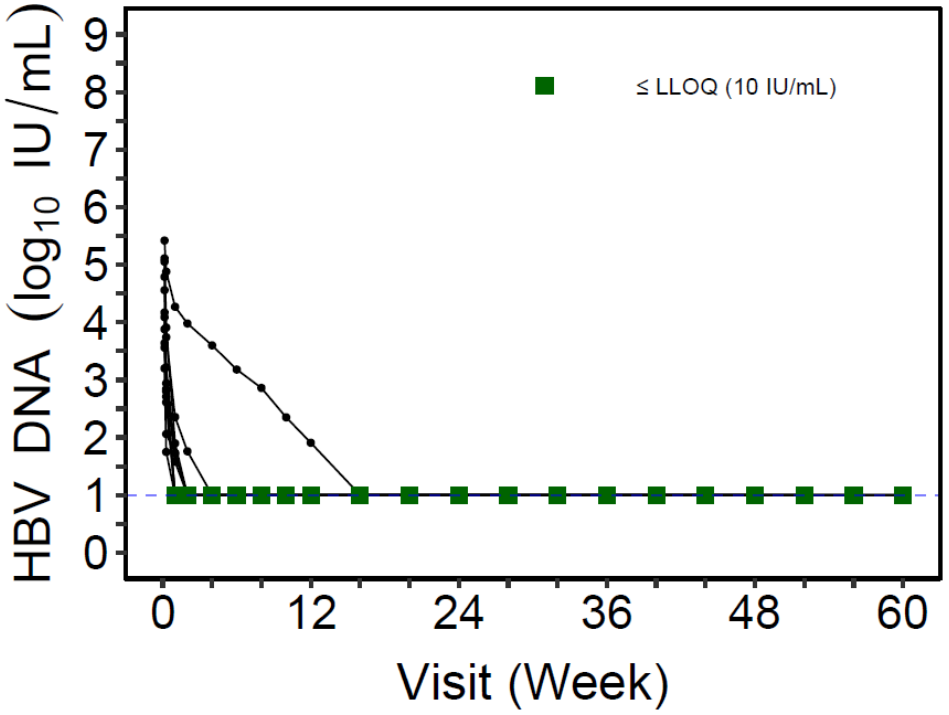
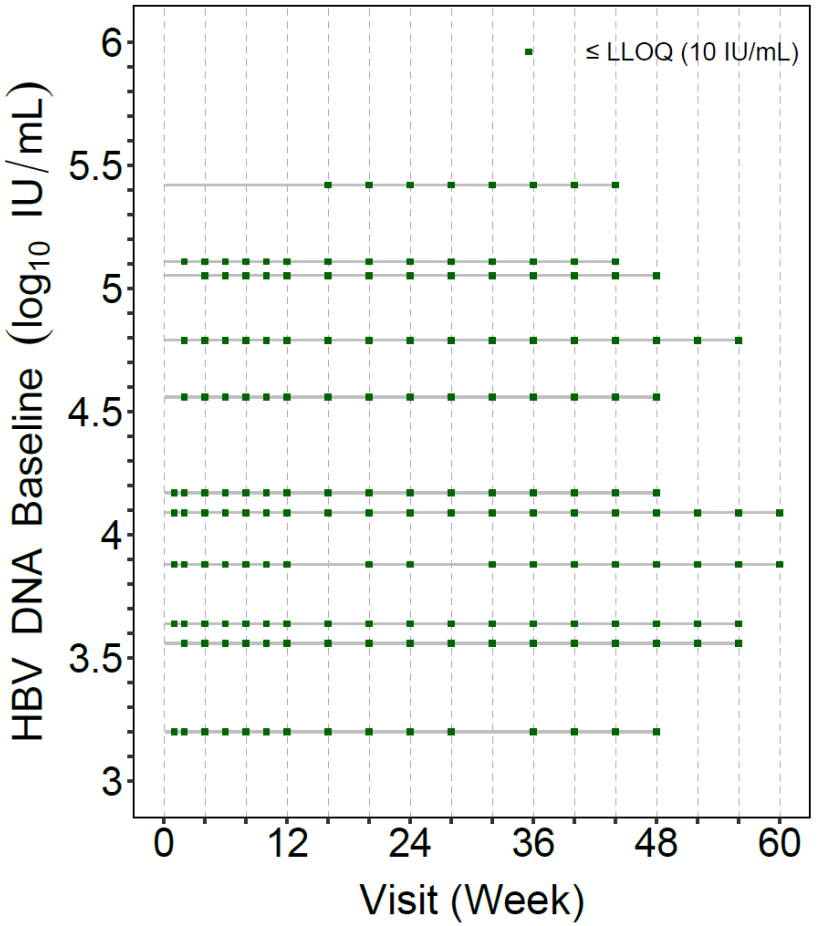
Agarwal, K. et al. EASL 2024.

300 mg ALG-000184 Monotherapy (HBeAg-)
Individual HBV DNA Decline



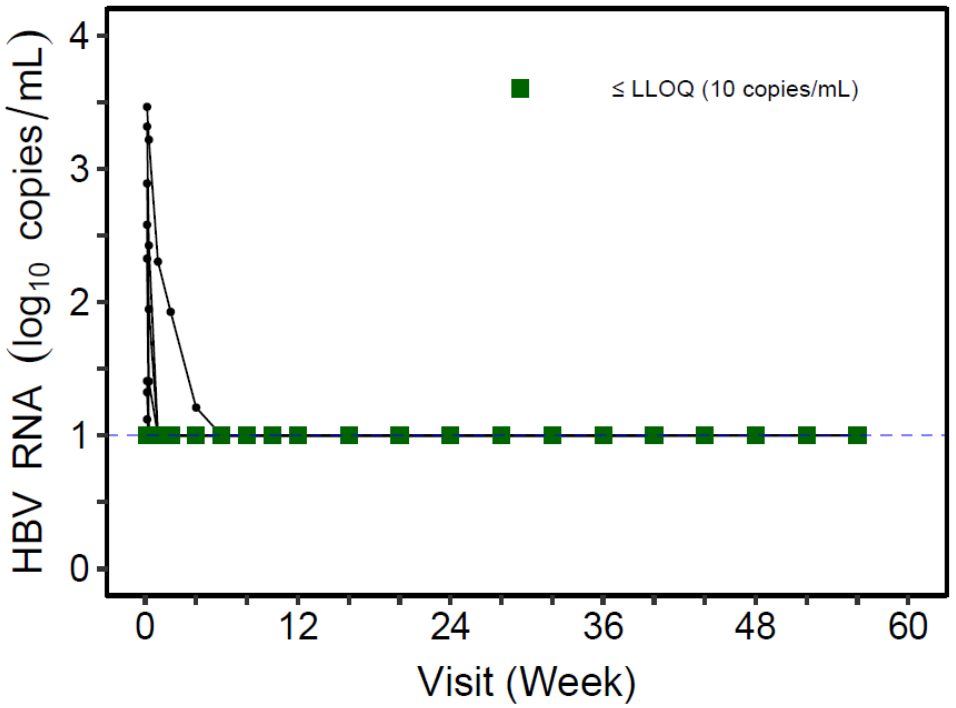
Week	0	4	8	12	16	20	24	28	32	36	40	44	48	52	56	60
Total n	11	11	11	11	10	11	11	10	11	11	11	11	10	7	5	4
≤ LLOQ [10 IU/mL]	0	10	10	10	10	11	11	10	11	11	11	11	10	7	5	4



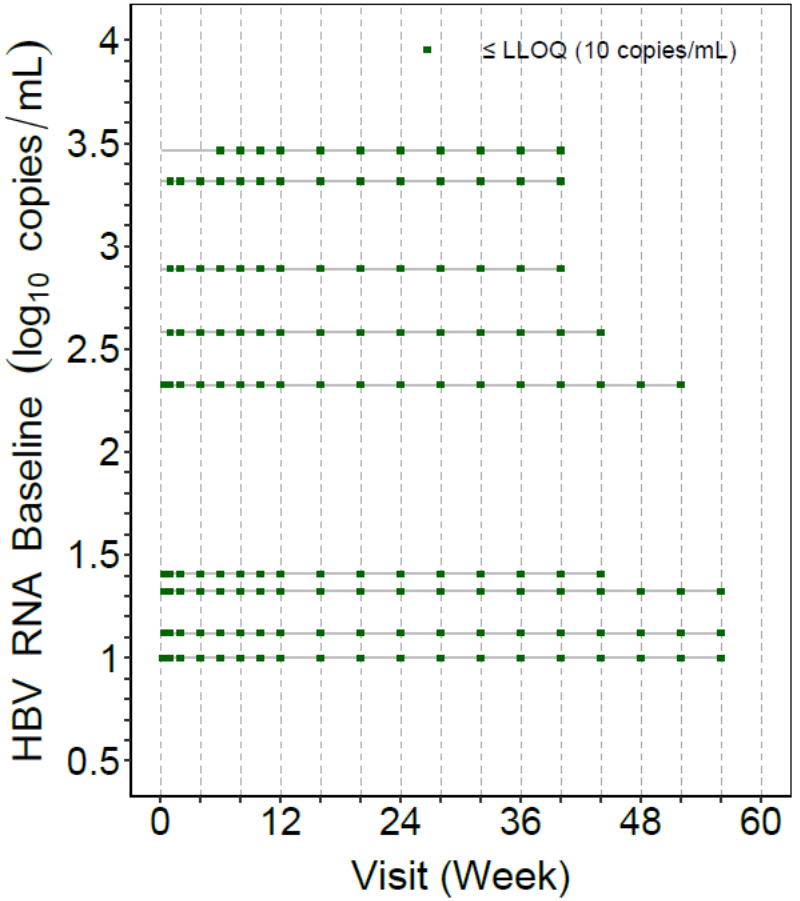
100% (11/11) of subjects achieved HBV DNA < LLOQ (10 IU/mL) by Week 20

91% (10/11) of subjects achieved HBV DNA < LLOD (< 4.92 IU/mL) by Week 48

300 mg ALG-000184 Monotherapy (HBeAg-)
Individual HBV RNA Decline



Week	0	4	8	12	16	20	24	28	32	36	40	44	48	52	56
Total n	11	11	11	11	10	11	11	10	11	11	11	8	5	5	3
≤ LLOQ [10 copies/mL]	3	10	11	11	10	11	11	10	11	11	11	8	5	5	3



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

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

ALG-000184
Chronic DNA Suppression versus Standard of Care

CHB HBeAg Status	Drug	% Patients < LLOQ at Week 48 (by HBV DNA Assay Sensitivity)		Regulatory Pathway
		% < LLOQ 29 IU/mL	% < LLOQ 10 IU/mL	
E-	TDF (n=140)[a]	93%	17%	Superior Chronic Suppression vs. NAs
	TAF (n=285)[a]	94%	21%	
	300 mg ALG-000184 (n=11)[d]	**11/11 (100%)**	**11/11 (100%)**	
E+	TDF (n=292)[b]	67%	Presumably <21%	
	TAF (n=581)[b]	64%		
	300 mg ALG-000184 (n=10)[c]	**10/10 (100%)**	**6/10 (60%)**	

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

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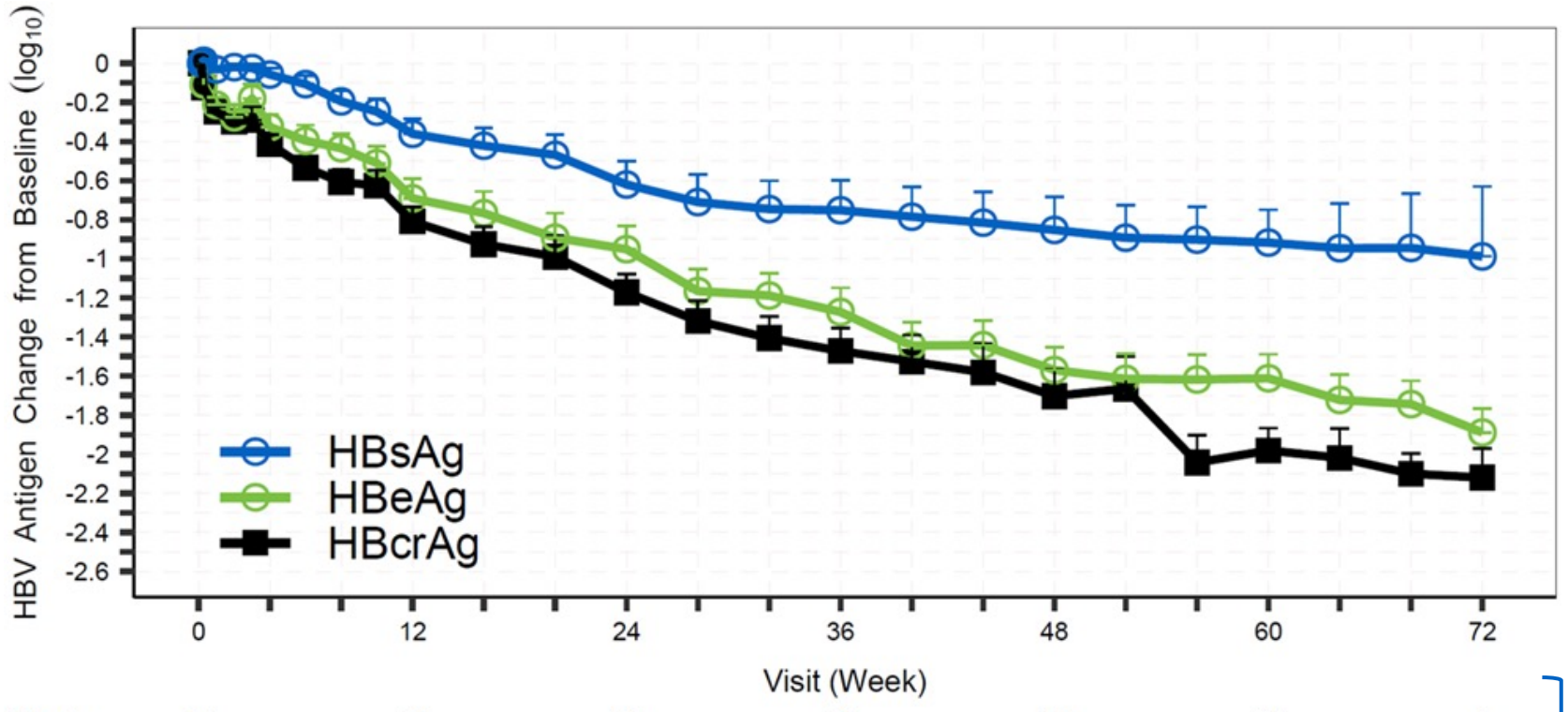
TAF-tenofovir alafenamide, TDF-tenofovir; LLOQ-lower limit of quantification.
[a] Buti et. al., Lancet Gastro 2016; [b] Chan et. al., Lancet Gastro 2016. [c] Yuen, M-F. et al., EASL 2024. [d] Agarwal, K. et al. EASL 2024.

ALG-000184-201 - Antiviral Effect in HBeAg+ CHB Subjects
HBV Antigen Change from Baseline



HBsAg	21	19	19	19	17	16	6
HBeAg	21	19	19	19	17	16	6
HBcrAg	21	19	19	19	17	9	5

Number of subjects at each timepoint

Continued substantial HBsAg, HBeAg, and HBcrAg reductions noted with combo through week 72
Mean max declines: 2.1, 2.6 and 2.7 log_{10} IU/mL, respectively

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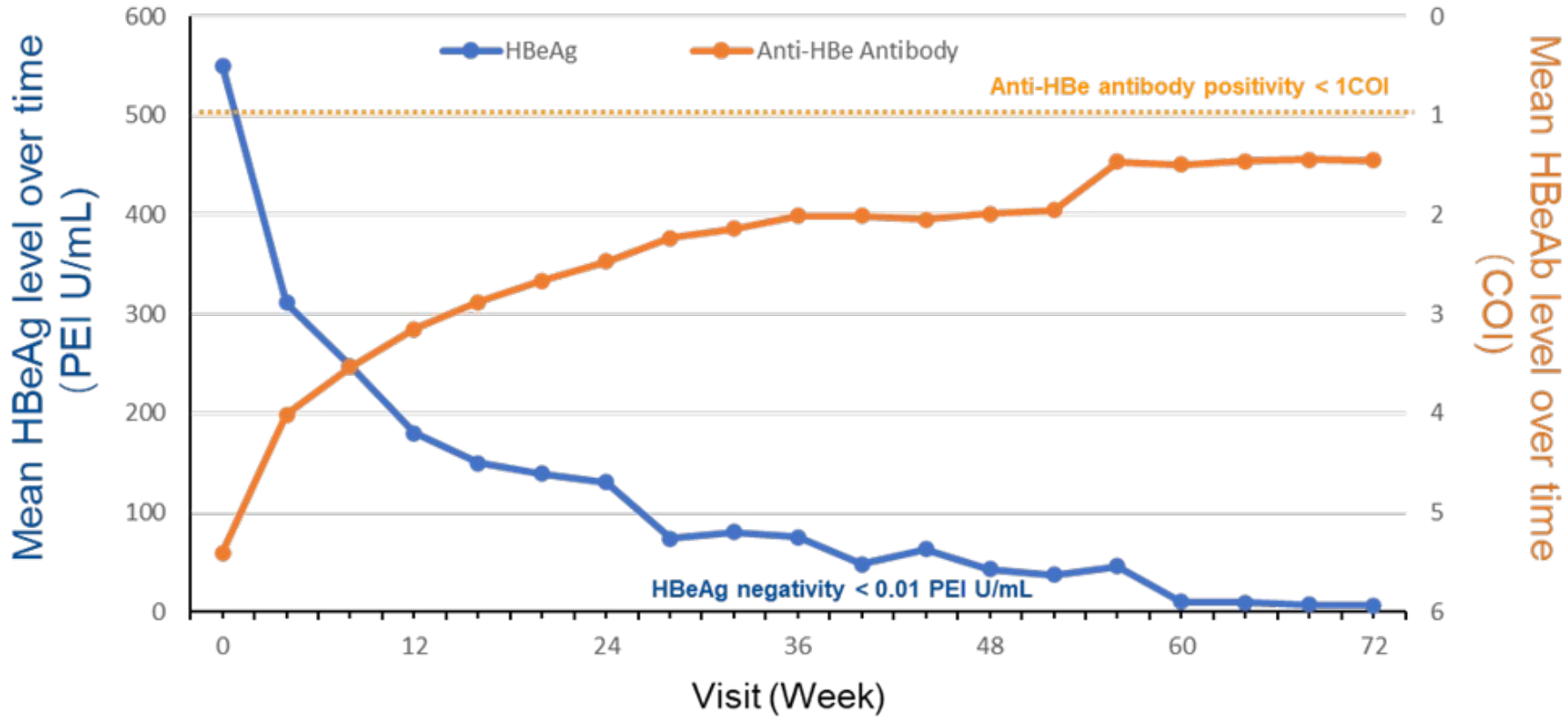
Graph plots subjects initially randomized to ALG-000184 + ETV and were compliant (confirmed by PK).
Yuen, M-F. et al.,; EASL 2024.

300 mg ALG-000184 ± ETV
Mean HBeAg and Anti-HBe Antibody Level Over time



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

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

Safety Overview – 300 mg ALG-000184 ± ETV
Treatment Emergent Adverse Events

	HBeAg-Positive Population		HBeAg-Negative Population
ALG-000184 Regimen	300mg QD + ETV	300mg QD	300mg QD
N of subjects	N=15	N=10	N=11
Serious Adverse Events (SAEs)	None		
Treatment emergent AEs (TEAEs) leading to study drug discontinuation	None		
Subjects with Grade ≥ 3 TEAEs	4 ALT/AST↑ (n=3); neutropenia↑ (n=1) eGFR↓ (n=1); Uric acid ↑ (n=1)	3 ALT/AST↑ (n=3)	2 ALT/AST↑ Cholesterol/Triglycerides ↑
Concerning TEAE, laboratory, ECG, vital sign, or physical examination findings or trends	None		

- All Grade ≥3 TEAEs of liver transaminase elevations were transient, resolved (n=6) and improving (n=1) in setting of continued dosing with study drug, and were associated with a potent antiviral effect. None were considered clinically concerning by the AFC.
- Neutropenia considered probably related to an acute respiratory infection and resolved post-infection in the setting of continued dosing with study drug
- Grade 3 eGFR decrease was reported in one subject with Grade 2 baseline level; returned to baseline level within 2 weeks in setting of continued study dosing
- Uric acid increase and cholesterol/triglycerides increase were asymptomatic and fluctuated between Grade 1 and 3 in setting of continued dosing

A favorable safety profile was observed in untreated HBeAg+ and HBeAg- CHB subjects with long term (≤88 weeks) treatment with 300 mg QD ALG-000184 ± ETV

Chronic Suppression
Well Defined, Validated Approval Pathway

Regulatory pathway for chronic suppressive therapy endorsed by FDA, EMEA, and China FDA (CDE)
Primary endpoint: Subjects with HBV DNA <LLOQ 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.

Aligos has received FDA feedback supporting subsequent studies utilizing this pathway

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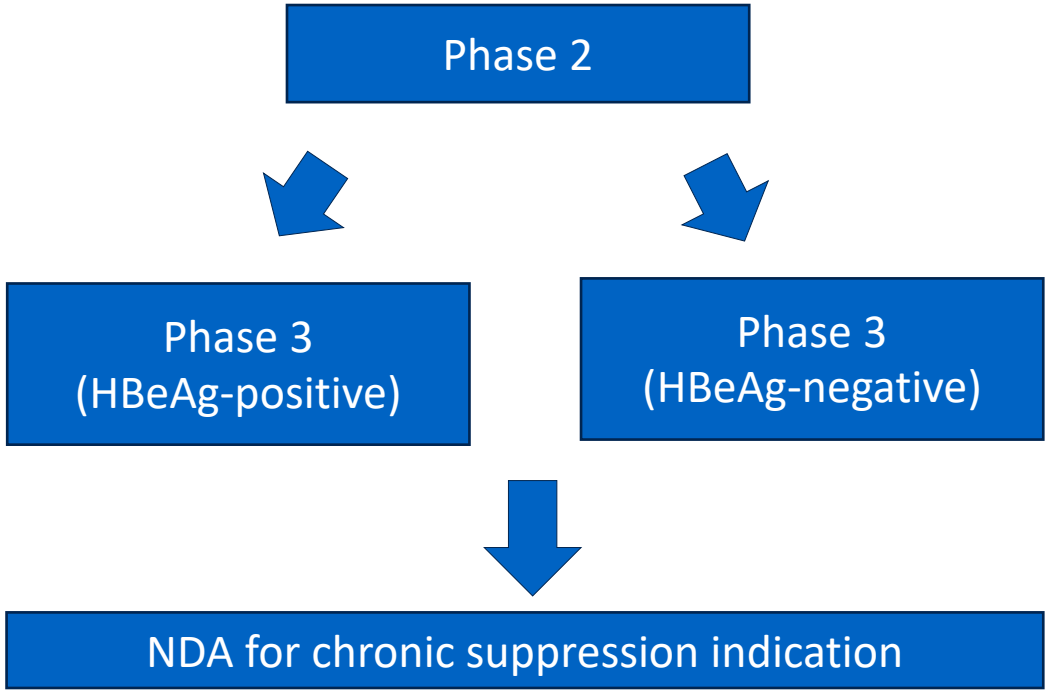
HBV Guidance from: FDA 2022; EMEA 2006; China 2023; LLOQ: lower limit of quantitation.

ALG-000184 Development Plans



Phase 2

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

Phase 3

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

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

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

ALG-000184 Phase 2 Chronic Suppression Study
Planned Efficacy Endpoints

- Primary endpoint (approvable endpoint*)
  - Proportion of subjects with HBV DNA < 10 IU/mL at W48 for both HBeAg+/- CHB infected subjects
- Secondary endpoints (clinically meaningful and/or corroborative)
  - HBeAg seroconversion in HBeAg+ CHB infected subjects
  - Reduction of HBsAg, HBcrAg, HBeAg
  - Reduction of cccDNA level and/or related serum biomarkers
  - Reduction of HBV integrants^
  - HBV RNA < LLOQ^

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

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

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

ALG-055009 for MASH

- ✓Phase 2a HERALD enrollment completed in May 2024
- Phase 2a HERALD topline safety and MRI-PDFF data expected in early Q4 2024

ALG-000184 for CHB

- ✓Greater DNA suppression observed vs. NAs
- ✓Phase 1b study is ongoing with interim data readouts at APASL, EASL,
- ✓Clear regulatory path forward for chronic suppressive therapy with superiority label
- Additional interim data readouts expected at AASLD
- Phase 2 enabling activities ongoing; planned Phase 2 IND filing in Q1 2025

ALG-097558 for Pan-Coronavirus

- ✓Phase 1 FIH topline data presented April 2024
- Phase 2 enabling activities (externally funded) ongoing

Strong Cash Position

- As of 3/31/24: Cash, cash equivalents and investments were $112.7M
- The Company believes our cash, cash equivalents and investments will provide sufficient funding of planned operations through the end of 2025


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