UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d) of the
Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): November 11, 2019

ASSEMBLY BIOSCIENCES, INC.
(Exact name of registrant as specified in its charter)

Delaware	**001-35005**	**20-8729264**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

11711 N. Meridian St., Suite 310
Carmel, Indiana 46032
(Address of principal executive offices, including zip code)

(833) 509-4583
(Registrant's telephone number, including area code)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, par value $0.001	**ASMB**	**The Nasdaq Global Select Market**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 or Rule 12b-2 of the Securities Exchange Act of 1934.

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 8.01. Other Events.

On November 11, 2019, Assembly Biosciences, Inc. (the Company) issued a press release announcing the presentation of (1) final 24-week results and interim long-term data from the Company's Phase 2 studies of ABI-H0731 (731), the Company's lead hepatitis b virus (HBV) core inhibitor product candidate, and (2) interim data from the first dose cohort of the Company's ongoing Phase 1b study of ABI-H2158 (2158), the Company's second HBV core inhibitor product candidate. These data were presented on posters at the late-breaker poster session at the American Association for the Study of Liver Diseases Annual Meeting (The Liver Meeting®) in Boston, Massachusetts.

ABI-H0731

The Company presented final 24-week data from HBeAg-positive patients in its studies of ABI-H0731-201 (Study 201 in nucleos(t)ide (NrtI)-suppressed patients) and ABI-H0731-202 (Study 202 NrtI-naïve patients) in addition to interim data from an ongoing open-label extension study ABI-H0731-211 (Study 211), where all patients received a combination of 731 and NrtI therapy.

Of the 97 patients completing Study 201 or Study 202, 87 are currently receiving a combination of 731 and NrtI therapy and have been treated for at least 16 weeks in Study 211 (cumulative duration of treatment with 731 and NrtI therapy of 16 to more than 40 weeks). 731 was well-tolerated when administered in combination with NrtI therapy. Overall, 26 out of 58 patients whose results were reported in the final 24-week results reported no adverse events (AEs). The remaining patients reported AEs that were Grade 1 or 2 and no serious AEs have been reported to date.

Study 201 and Study 202

As previously reported in the literature, the vast majority of long-term NrtI therapy treated patients continue to harbor low level infectious virus, which was confirmed in Study 201 patients at the time of their enrollment. Final Week 24 results from the HBeAg-positive patients (n=47) demonstrated that, among those with detectable DNA at baseline, 22 out of 27 (81%) of 731 and NrtI therapy treated patients achieved target not detected (TND) by Week 24 compared to zero out of 12 (0%) NrtI therapy only treated patients ($p<0.001$), as measured with a highly sensitive PCR assay (lower limit of quantification (LLOQ) 5 = IU/mL). These results indicate that the addition of 731 reduced viral burden to levels not achieved by NrtI therapy alone.

Final Week 24 results from HBeAg-positive patients in Study 202 (n=25) demonstrated faster and deeper HBV DNA declines in patients receiving 731 and entecavir (ETV) than those receiving ETV alone. Statistically significant reductions of pgRNA were observed by Week 2 with 731 and ETV ($p<0.001$).

Study 211

Longer-term treatment with 731 and NrtI therapy resulted in deeper reductions in HBV DNA and pgRNA. The 21 out of 25 patients from Study 202 that are now in Study 211 demonstrated mean HBV DNA and pgRNA declines from baseline of 6.3 logs and 3.0 logs, respectively, at Week 48.

A significant finding based on interim data from Study 211 is the observed correlation between the degree of pgRNA reductions and viral antigen declines. Eleven out of 21 (52%) patients from Study 202 that are now on Study 211 who have been treated with 731 and NrtI therapy for 16 to 60 weeks have achieved decreases in pgRNA of greater than 3 logs. The results in the table below demonstrate that these larger declines in pgRNA were strongly associated with observed reductions in viral antigens. As cccDNA is the only known source of pgRNA, the deeper decline of pgRNA levels may therefore indicate a reduction in cccDNA pools.

Number	<40 U/L	Log_{10} Decrease	Mean Log Reductions at Last Time Point (range)			Patients Exhibiting ≥0.5 Log Decline (%)		
Patients	ALT	pgRNA	HBeAg	HBcrAg	HBsAg	HBeAg	HBcrAg	HBsAg
11	10	>3.0	1.03 (0.0-2.5)	1.42 (0.0-3.1)	0.86 (0.0-3.6)	9 (82)	10 (91)	6 (55)
8	8	2.0-3.0	0.34 (0.1-0.7)	0.45 (0.1-1.0)	0.14 (0.0-0.5)	2 (25)	6 (75)	1 (13)
2	2	<2.0	0.15 (0.9-1.8)	0.29 (0.3-0.3)	0.17 (0.0-0.3)	0 (0)	0 (0)	0 (0)

Of the 27 NrtI-suppressed HBeAg-positive patients receiving 731 and NrtI therapy for at least 40 weeks in Study 201 and who are now in Study 211, 18 (67%) have achieved HBV DNA TND + pgRNA less than 35 U/mL, along with significant declines in HBeAg and HBcrAg levels.

Safety Overview

731 was well-tolerated in both HBeAg-positive and -negative patients when administered with a NrtI therapy for 24 weeks with no serious AEs reported. Five patients receiving 731 and NrtI reported a rash (four Grade 1 and one Grade 2). No associated systemic signs or laboratory abnormalities were observed, and all patients continued treatment through Week 24. Overall, laboratory abnormalities observed were of Grade 1 or 2 severity and occurred in similar proportions of patients across the two treatment groups. With longer-term ongoing treatment in Study 211, interim data indicated that the nature, frequency and severity of AEs and laboratory abnormalities observed were similar to the initial 24 week treatment period.

ABI-H2158

The Company reported interim data from the first cohort of its Phase 1b study of 2158, which is currently enrolling HBeAg positive patients in sequential dose cohorts of nine patients, with each cohort randomized to receive oral 2158 or placebo (7:2) once daily for 14 days. The interim data from the initial cohort receiving the lowest dose of 2158 at 100 mg demonstrated potent antiviral activity at this initial dose level, reflected by mean declines from baseline to day 15 of 2.3 log10 [range 1.7 – 3.0] and 2.1 log10 [range 1.5 – 2.7] in HBV DNA and pgRNA respectively.

No serious AEs, dose limiting toxicities or premature discontinuations have been reported to date. All treatment emergent adverse events (TEAEs) were Grade 1. One patient assigned to placebo and three patients on 2158 reported TEAEs that resolved without intervention: dizziness, fatigue, rash, headache and upper abdominal pain. Observed steady-state exposures were in excess of the EC90's for *in vitro* antiviral and cccDNA assays. The Company believes that the safety and pharmacokinetic (PK) data and parameters from this interim analysis support once daily administration and the continued evaluation of 2158 across the planned dose cohorts in patients with chronic HBV infection. The Phase 1b study is expected to be completed in the first quarter of 2020.

A copy of the presentation is filed as Exhibit 99.1 to this Current Report on Form 8-K and incorporated by reference herein.

Forward-Looking Statements

The information in this report contains forward-looking statements regarding future events, including statements about the clinical and therapeutic potential of the Company's HBV core inhibitor product candidates, and the timing of the initiation of and the availability of data from the Company's ongoing and planned clinical trials. Certain forward-looking statements may be identified by reference to a future period or by use of forward-looking terminology such as "expected," "may" and "potential." The Company intends such forward-looking statements to be covered by the safe harbor provisions contained in Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. Actual results or developments may differ materially from those projected or implied in these forward-looking statements. These risks and uncertainties include, among others: the Company's expectations regarding sustained benefits and antiviral reductions of patients in its clinical trials; the scientific theory for the Company's therapeutics is unproven and novel; outcomes of clinical studies are uncertain; results observed in earlier preclinical and nonclinical studies and early clinical studies, including with respect to tolerability results, may not be predictive of future clinical studies results; the components, timing, cost and results of clinical trials and other development activities involving our product candidates; the duration and results of regulatory review of those candidates by the FDA and foreign regulatory authorities; whether our cash resources will be sufficient to fund continuing operations for the periods and/or trials; and the possible impairment of, or inability to obtain, intellectual property rights and the costs of obtaining such rights from third parties. Additional information about the risks and uncertainties faced by the Company are more fully detailed under the heading "Risk Factors" in the Company's Quarterly Report on Form 10-Q for the quarter ended September 30, 2019 filed with the Securities and Exchange Commission. Except as required by law, the Company assumes no obligation to update publicly any forward-looking statements, whether as a result of new information, future events or otherwise.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits:

Exhibit No.	Description
99.1	AASLD 2019 Presentation dated November 11, 2019.
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Date: November 12, 2019

Assembly Biosciences, Inc.

By: /s/ John G. McHutchison, A.O., M.D.
John G. McHutchison, A.O., M.D.
President and Chief Executive Officer

Exhibit 99.1

assemblybio

AASLD 2019 Review

November 11, 2019

Agenda and Speakers on Today's Call

Agenda

- Introductions and Safe Harbor
- Overview of ASMB vision and HBV core inhibitor portfolio
- Review of HBV data from AASLD

  ABI-H0731
  - Final 24 week data from Study 201 and 202
  - Interim long-term data from Study 211

  ABI-H2158
  - Interim data from initial dosing cohort of Phase 1b study (100 mg)
- Q&A

Speakers

- Lauren Glaser - SVP IR and Corporate Affairs
- John McHutchison, AO, MD – CEO and President
- Richard Colonno, PhD – EVP & CSO Virology Operations

Cautionary Note Regarding Forward-Looking Statements

The information in this presentation contains forward-looking statements regarding future events, including statements about the clinical and therapeutic potential of Assembly Biosciences' HBV-cure program, the therapeutic potential of core inhibitors, including ABI-H0731, ABI-H2158 and ABI-H3733, and the plans, strategies and intentions related to its HBV-cure program and proposed stages to cure. Certain forward-looking statements may be identified by reference to a future period or periods or by use of forward-looking terminology such as "may," "planned," "potential," and "will." Such forward-looking statements, which are intended to be covered by the safe harbor provisions contained in Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended, are just predictions and are subject to risks and uncertainties that could cause the actual events or results to differ materially. These risks and uncertainties include, among others: the scientific theory for our therapeutics is unproven and novel; outcomes of clinical studies are uncertain; results observed in earlier preclinical and nonclinical studies and early clinical studies may not be predictive of future clinical studies results; and the emergence of unforeseen safety issues. These and other potential risks and uncertainties that could cause actual results to differ from the results predicted are more fully detailed under the heading "Risk Factors" in our Quarterly Report on Form 10-Q for the quarter ended September 30, 2019, filed with the Securities and Exchange Commission (the "SEC") and any additional reports filed with the SEC following the date of this presentation. It is not possible for Assembly Biosciences management to predict all risks nor can it assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in any forward-looking statements. In light of these risks, uncertainties and assumptions, the forward-looking events and circumstances discussed in this presentation may not occur and actual results could differ materially and adversely from those anticipated. Any forward-looking statement speaks only as of the date on which it is made, and no obligation to update or revise any forward-looking statement is assumed, whether as a result of new information, future events or otherwise, except as required by law.

Our Vision for Achieving HBV Curative Finite Regimens

HBV can and will be cured

Over time, cure rates will increase and Tx duration shorten

Core inhibitors will be central to curative strategies

Block multiple steps in the HBV life cycle

Cure rates will increase as the field advances

Tx will be **finite** but longer than HCV to start

Combination regimens will be required for cure

Multiple agents with complementary MOA's

Not all patient populations will respond equally

Different Tx regimens for different patient subgroups

Simple all oral regimen that is safe and well-tolerated

Ultimate winner for the 250M HBV infected patients globally

Assembly believes it has the most potent, most advanced, and broad series of core inhibitors at multiple stages of development

Assembly's Planned Approach to Hepatitis B Clinical Development

First wave

1st generation CI Candidate

- Designed to achieve complete suppression of viral replication (HBV DNA and RNA)

Two potential paths to registration that are complementary:

- Chronic suppressive therapy
- Finite Duration
  - Consolidation period of Tx
  - Withdrawal to observe for cure

Second wave

Next generation CI Candidates

- More potent next generation CI candidates (2158 and 3733)
- Potential to accelerate the speed, efficiency and proportion of patients with complete suppression of viral replication

Potential for increased cure rates and/or shortened duration to cure

Third wave

Triple Drug CI Combinations

- Potential multi drug combinations with non-overlapping MOA's may drive response rates higher in a shorter duration of treatment

Focus on future POC studies to evaluate these approaches through carefully executed, scientific cross-company collaborations

Program Objectives - Targeted Steps Toward Cure

✓ AASLD 2018 — Well-tolerated, PK supporting once daily dosing, and inhibits HBV DNA with monotherapy (Phase 1)

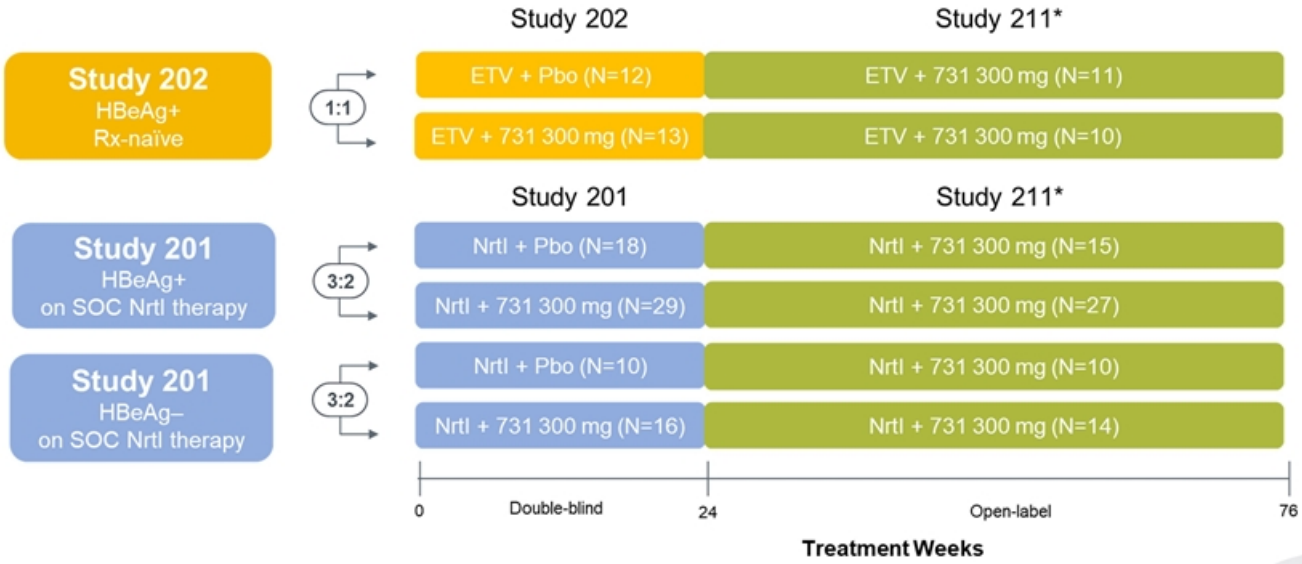
✓ EASL 2019 — Demonstrated elimination of residual viral replication not achievable on NrtI monotherapy (i.e DNA to "Target not Detected") (Phase 2)

✓ AASLD 2019 — Demonstrated decrease in cccDNA population as reflected by significant reductions in pgRNA levels and other surrogate markers in absence of ALT flares (Phase 2)

Goal: Demonstrate further decline of viral antigens during consolidation (Phase 2)

Goal: Following consolidation, demonstrate sustained viral DNA/RNA suppression off therapy (Phase 2)

Overview of ABI-H0731 Phase 2a Clinical Studies



*n values represent the 87 patients who transitioned to 211 and remain on treatment and included in this analysis
ETV, entecavir, Pbo, placebo, SOC, standard of care

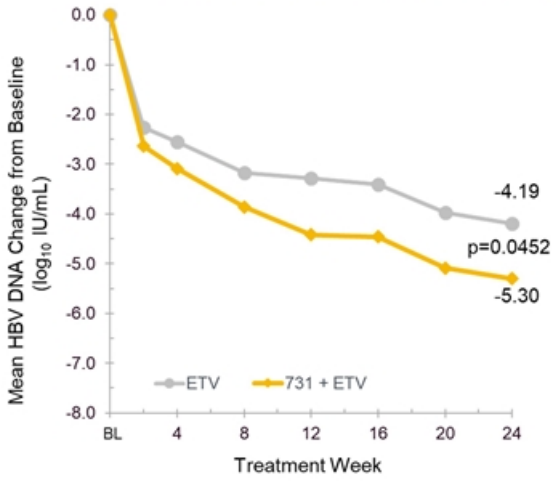
Demographics and Baseline Characteristics

	Study 202	Study 201	
	HBeAg+ (N=25)	HBeAg+ (N = 47)	HBeAg– (N = 26)
Demographics			
Age, years, mean (range)	35 (20–66)	44 (20–66)	48 (34–64)
Female, n (%)	17 (68)	16 (34)	10 (38)
Asian, n (%)	24 (96)	42 (89)	21 (81)
Genotype B, C[a] (%)	11, 11 (88)	12, 19 (66)	4, 2 (23)
Baseline characteristics, mean (range)			
ALT U/L	56.7 (13–295)	26.8 (13–97)	24.7 (9–67)
HBV DNA $\log_{10}$ IU/mL[b]	8.0 (5.5–9.1)	45 (96% <LLOQ)	26 (100% <LLOQ)
HBV pgRNA $\log_{10}$ U/mL	7.2 (4.6–8.6)	3.5 (1.5–6.3)[c]	1.6 (1.5–2.6)[c]
HBsAg $\log_{10}$ IU/mL	4.6 (3.3–5.2)	3.5 (2.9–4.5)	3.1 (2.2–4.2)
HBeAg $\log_{10}$ IU/mL[d]	2.5 (–0.7–3.1)	0.5 (–0.9–2.6)	25 (96% <LLOQ)
HBcrAg $\log_{10}$ kU/mL	5.4 (2.8–6.2)	3.0 (1.4–4.8)	0.4 (–1.0–1.9)

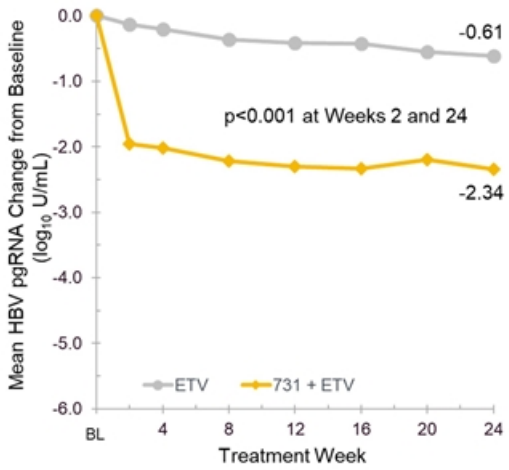
LLOQ, lower limit of quantification;
[a]Genotypes in Study 201 were determined by sequence alignment; genotypes in Study 202 were determined by InnoLipa HBV at a central lab;
[b]As measured by Roche Cobas qPCR, LLOQ = 20 IU/mL; Reported mean (range) for Study 202 and n (% <LLOQ) for Study 201;
[c]Nine of 47 HBeAg-positive patients with baseline pgRNA <35 U/mL; 22 of 26 HBeAg-negative patients with baseline pgRNA <35 U/mL. HBV pgRNA values <35 U/mL were imputed at 34 U/mL;
[d]Reported mean (range) for Study 202 and Study 201 HBeAg-positive patients, and n (% <LLOQ) for Study 201 HBeAg-negative patients

Study 202: Superior DNA/pgRNA Declines Observed with 731+ETV Combination

Faster HBV DNA Declines on Combination



Significant pgRNA Declines on Combination

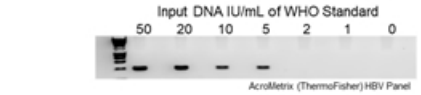


- Faster HBV DNA declines were observed with 731+ETV than with ETV alone, with statistically significant declines in HBV DNA observed in the combo arm at Week 24 (p=0.0452)
- Rapid 2-log reductions in HBV pgRNA levels by Week 2 were observed only in patients receiving combo (p<0.001)
- The initial rapid phase decline of pgRNA is thought to be mechanism-based inhibition (ie, pgRNA not packaged and secreted into plasma), while the second slower phase decline is believed to reflect reduction in cccDNA pools

Study 201: DNA/pgRNA Declines Observed in NrtI-Suppressed, HBeAg+ Patients

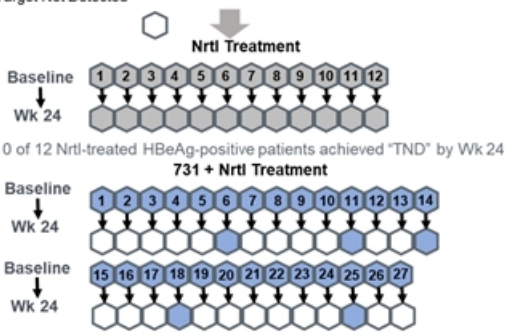
Deeper HBV DNA Declines on Combination

Highly sensitive semi-quantitative PCR assay developed to detect viral DNA levels as low as 5 IU/mL to monitor loss of residual virus



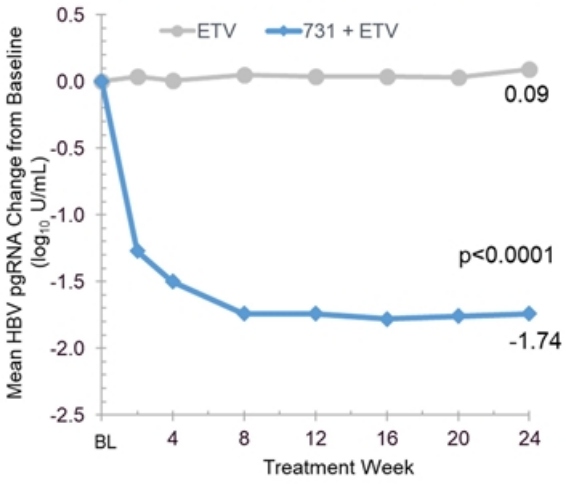
Gel Assay Standardization and Validation

Individual patient gel results; "Target Detected" or "Target Not Detected"



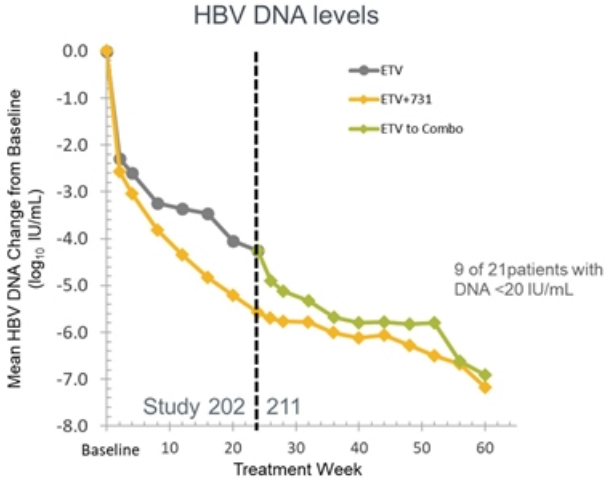
- 0 of 12 NrtI-treated HBeAg-positive patients achieved "TND" by Wk 24
- 22 of 27 (81%) 731+NrtI treated patients achieved TND by Week 24 (81% vs 0%, p<0.001)

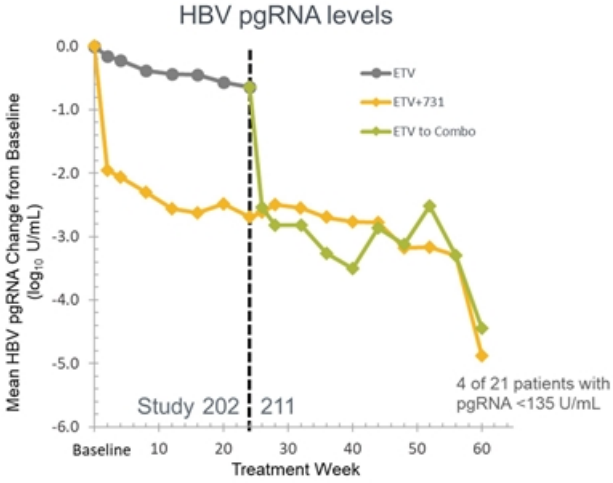
Significant pgRNA Declines on Combination



- Among HBeAg-positive patients, rapid reductions in HBV pgRNA levels by Week 8 were observed only in patients treated with 731+ETV

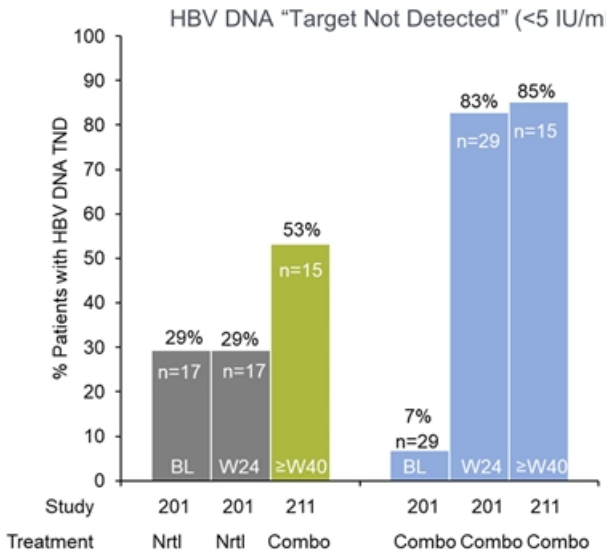
Study 202/211: Further DNA/pgRNA Declines Observed over Time

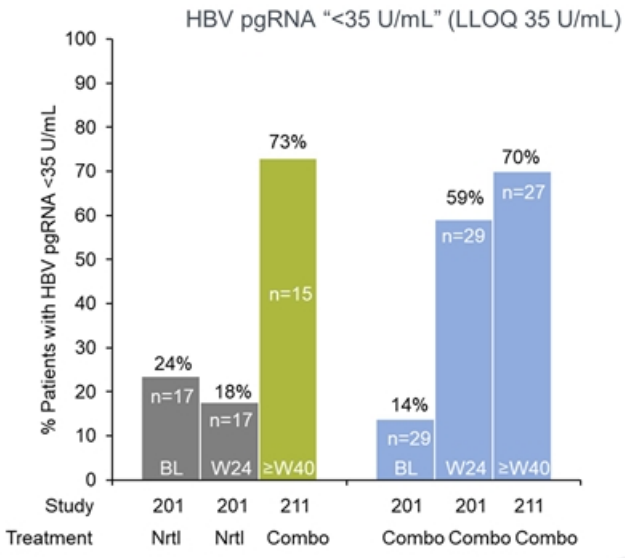




- Switch from ETV to 731+ETV resulted in immediate and enhanced declines in both HBV DNA and pgRNA levels
- The mean HBV DNA and pgRNA declines from baseline at Week 48 were 6.3 logs and 3.0 logs, respectively, for patients treated with 731+ETV
- The observed acceleration in second phase decline of HBV pgRNA levels likely reflects reductions of cccDNA pools

Study 201/211: DNA/pgRNA Declines to Highly Suppressed Levels Observed in Nrtl-Suppressed Patients



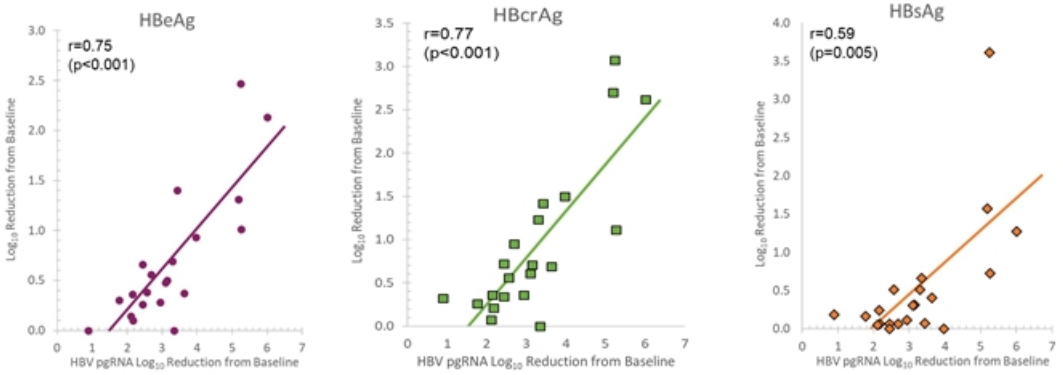


- Only patients receiving 731+ETV had reduced HBV DNA levels to TND and pgRNA levels to <35 U/mL

Study 202/211: Correlation Between HBV pgRNA Reductions and Viral Antigen Declines in the Absence of ALT Elevations

Patients Treated 16–60 Weeks with 731+ETV

Number	<40 U/L	Log_{10} Decrease	Mean Log Reductions at Last Timepoint (range)			Mean Max Log Reductions (range)			Patients Exhibiting ≥0.5 Log Decline (%)		
Patients	ALT	pgRNA	HBeAg	HBcrAg	HBsAg	HBeAg	HBcrAg	HBsAg	HBeAg	HBcrAg	HBsAg
11	10	>3.0	1.03 (0.0–2.5)	1.42 (0.0–3.1)	0.86 (0.0–3.6)	1.09 (0.4–2.3)	1.46 (0.6–3.1)	0.87 (0.0–3.6)	9 (82)	10 (91)	6 (55)
8	8	2.0–3.0	0.34 (0.1–0.7)	0.45 (0.1–1.0)	0.14 (0.0–0.5)	0.36 (0.1–0.8)	0.59 (0.0–1.0)	0.17 (0.0–0.7)	2 (25)	6 (75)	1 (13)
2	2	<2.0	0.15 (0.9–1.8)	0.29 (0.3–0.3)	0.17 (0.0–0.3)	0.15 (0.0–0.4)	0.40 (0.2–0.5)	0.21 (0.2–0.3)	0 (0)	0 (0)	0 (0)



r is Spearman's correlation between reduction in pgRNA and HBV antigen. The straight-line fit is calculated by choosing the line that minimizes the least square sum of the vertical distance d, of all the selected markers pictured by using the following equation: y = a + bx, where "a" is the intercept and "b" is the slope.

- Addition of 731 resulted in multi-log reductions in pgRNA; NrtI therapy failed to significantly reduce pgRNA
- Second phase decline of pgRNA appears to reflect decline in cccDNA pools: **>3 log reductions were associated with greatest level of declines in HBeAg and HBcrAg, surrogate markers of cccDNA**

Study 201/211: Progression of Viral Markers in HBV NrtI-Suppressed Patients

Parameter	Patients, n (%)
Combo Treatment ≥40 weeks	27 (100)
ALT ≤40 U/L	25 (93)
DNA TND (<5 IU/mL)	23 (85)
pgRNA <35 U/mL	19 (70)
DNA TND + pgRNA <35 U/mL	18 (67)
HBeAg <1 IU/mL and/or experienced a >0.5 log decline)	14 (52)
HBcrAg <100 kU/mL and/or experienced a >0.5 log drop	9 (33)
HBsAg experienced a >0.5 log drop	1 (4)
DNA TND + pgRNA <35 U/mL + HBeAg <1 IU/mL or ≥0.5 log decline	10 (37)

- Viral markers in these patients receiving long-term NrtI treatment were significantly lower than in Rx-naïve patients, with several approaching the LLOQ
- Results are supportive of mixed source (cccDNA and integrants) HBsAg in long-term HBeAg-negative and NrtI-suppressed patients that appears different than other viral antigens, similar to prior reports[1,2]

[1]Wooddell, C.I. et al. Sci Transl Med 2017 Sep 27;9(409). pii: eaan0241. doi: 10.1126/scitranslmed.aan0241.
[2]Podlaha, O. et al. The International Liver Congress. Vienna, Austria, April 10–14, 2019.

Study 201/202: Final Safety Data Summary

Preferred Term, n (%)	24-Week Controlled Period			
	Rx-Naïve Patients (202)		NrtI-Suppressed Patients (201)	
	ABI-H0731 + NrtI (N=13)	NrtI (N=12)	ABI-H0731 + NrtI (N=45)	NrtI (N=28)
Any Treatment-Emergent AE	7 (53.8)	5 (41.7)	25 (55.6)	9 (32.1)
Grade 1	6 (46)	4 (33)	17 (37.8)	6 (21.4)
Grade 2	1 (8)	1 (8)	8 (17.8)	2 (7.1)
Grade 3	0	0	0	1 (3.6)
Any Serious AE	0	0	0	0
Rash[a]	0	0	5 (11.1)	0
Upper Respiratory Tract Infection	1 (7.7)	1 (8.3)	5 (11.1)	1 (3.6)
Fatigue	0	0	1 (2.2)	1 (2.2)
Nausea	0	0	4 (8.9)	0
Pruritis	2 (15.4)	0	3 (6.7)	0
Headache	2 (15.4)	0	3 (6.7)	0

*5 patients receiving ABI-H0731 + NrtI reported a rash; 4 Grade 1 and 1 Grade 2; no systemic signs or laboratory abnormalities were observed and all patients continued treatment through Week 24

- 731 was well-tolerated when administered with a NrtI for 24 weeks
- Overall, 26/58 subjects reported no AEs.
- Of the 32 subjects reporting ≥1 AE, 23 had Grade 1, and 9 had Grade 2 events. No serious AEs were reported
- With longer-term treatment in Study 211, the safety and tolerability data are similar to the initial Week 24 placebo-controlled period

Study 201/202: Laboratory Abnormalities

Parameter, n (%)	24-Week Controlled Period					
	ABI-H0731 + NrtI (N=58)			NrtI (N=40)		
	Grade 1	Grade 2	Grade 3	Grade 1	Grade 2	Grade 3
ALT (SGPT)	3 (5.2)	3 (5.2)	0	5 (12.5)	4 (10.0)	0
AST (SGOT)	4 (6.9)	2 (3.4)	0	6 (15.0)	3 (7.5)	0
Creatinine	0	0	0	2 (5.0)	0	0
Serum amylase	8 (13.8)	3 (5.2)	0	2 (5.0)	4 (10.0)	0
Serum glucose	8 (13.8)	1 (1.7)	0	10 (25.0)	2 (5.0)	0
Serum glucose decreased	1 (1.7)	1 (1.7)	0	3 (7.5)	0	0
Serum sodium	2 (3.4)	0	0	3 (7.5)	0	0
Serum uric acid	3 (5.2)	0	0	4 (10.0)	0	0
Urine blood	1 (1.7)	4 (6.9)	0	2 (5.0)	2 (5.0)	0

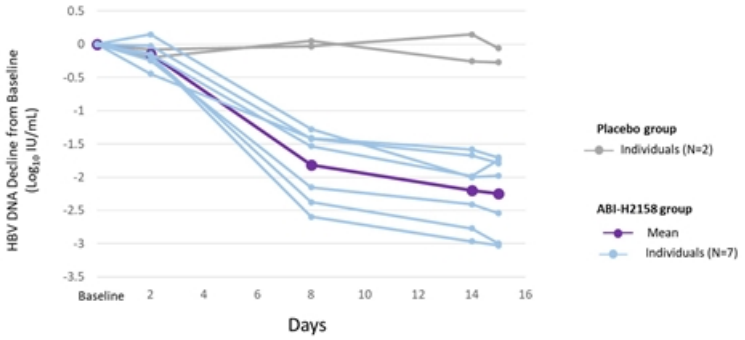
- Overall, laboratory abnormalities observed were of Grade 1 or 2 severity and occurred in similar proportions of patients across the two treatment groups
- With longer-term treatment in Study 211, the profile of laboratory abnormalities observed are similar to those at Week 24
- Grade 3 elevations in ALT and/or AST have been observed in 3 patients treated with 731+NrtI beyond Week 24
  - In 2 patients, elevations were transient and normalized within a 4–8-week period while continuing on treatment
  - In 1 patient ALT and AST fluctuated during treatment and were asymptomatic and Grade 3 (217 U/L) and Grade 2 (145 U/L), respectively at Week 52 of combination therapy
  - **All 3 patients remain on treatment**

ABI-H0731 Core Inhibitor Program Summary

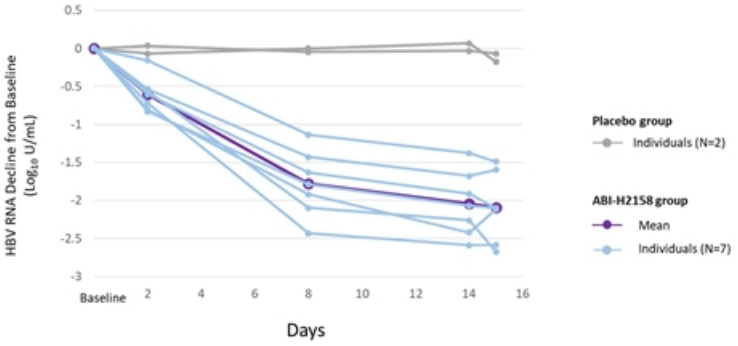
- **Summary of Interim Data for Phase 2a Studies with ABI-H0731**
  - Well tolerated
  - Combination of 731+NrtI demonstrated superior antiviral activity vs. NrtI monotherapy
    - Faster and deeper declines in HBV DNA observed
    - HBV DNA TND and pgRNA <35 U/mL thresholds only achieved in patients receiving the combination
    - Significant HBV pgRNA (surrogate marker of cccDNA) declines in both studies
    - Second phase declines in pgRNA >3 logs, which is a primary surrogate marker of cccDNA, were strongly associated with reductions in viral antigens, suggesting declining cccDNA pools

2158: Interim Phase 1b Antiviral Activity Data

HBV DNA Change from Baseline, Cohort 1 (100mg QD)

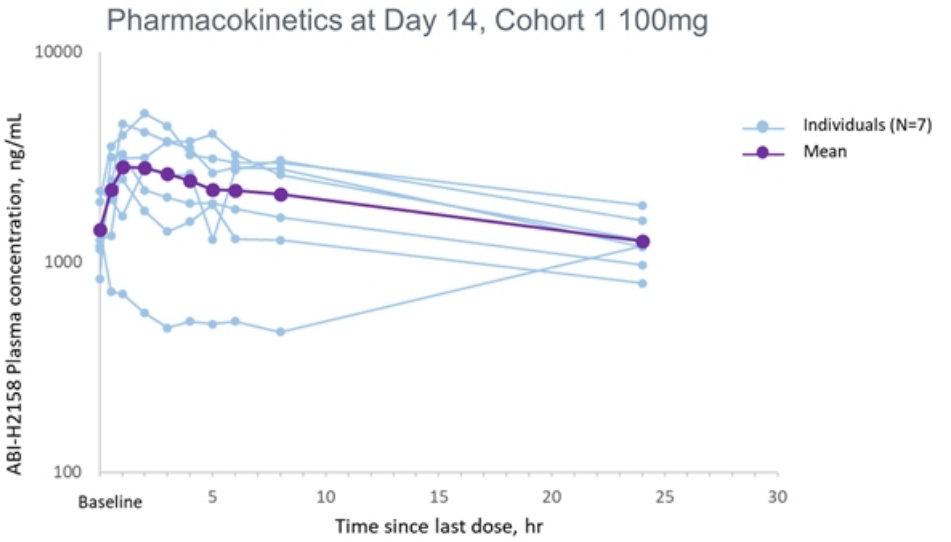


HBV pgRNA Change from Baseline, Cohort 1 (100mg QD)



- In patients receiving ABI-H2158, mean declines from Baseline to Day 15 in HBV DNA and pgRNA levels were 2.3 log10 IU/mL (range 1.7–3.0) and 2.1 log10 IU/mL (range 1.5–2.7), respectively

2158: Interim Phase 1b Pharmacokinetics Data



- Steady-state exposures observed at the lowest dose level of 100 mg QD are in excess of the EC_{90} values for *in vitro* antiviral and cccDNA assays

We believe that these data support once daily oral administration

Program Objectives - Targeted Steps Toward Cure

✓ AASLD 2018 — Well-tolerated, PK supporting once daily dosing, and inhibits HBV DNA with monotherapy (Phase 1)

✓ EASL 2019 — Demonstrated elimination of residual viral replication not achievable on NrtI monotherapy (i.e DNA to "Target not Detected") (Phase 2)

✓ AASLD 2019 — Demonstrated decrease in cccDNA population as reflected by significant reductions in pgRNA levels and other surrogate markers in absence of ALT flares (Phase 2)

Goal: Demonstrate further decline of viral antigens during consolidation (Phase 2)

Goal: Following consolidation, demonstrate sustained viral DNA/RNA suppression off therapy (Phase 2)

Thank you