Effects of the siRNA JNJ-3989 and/or the Capsid Assembly Modulator JNJ-6379 on Viral Markers of Chronic Hepatitis B: Results From the REEF-1 Study

Man-Fung Yuen,[1,*] Tarik Asselah,[2] Ira M. Jacobson,[3] Maurizia Brunetto,[4] Harry L.A. Janssen,[5] Tetsuo Takehara,[6] Jin Lin Hou,[7] Thomas N. Kakuda,[8] Tom Lambrecht,[9] Ronald Kalmeijer,[10] Carine Guinard-Azadian,[9] Cristiana Mayer,[10] John Jezorwski,[10] Thierry Verbinnen,[9] Oliver Lenz,[9] Umesh Shukla,[10] Michael Biermer[9]

[1]Department of Medicine & State Key Laboratory of Liver Research, Queen Mary Hospital, The University of Hong Kong, Hong Kong, China; [2]Université de Paris, INSERM UMR1149 and Hôpital Beaujon, APHP, Clichy, France; [3]New York University Grossman School of Medicine, New York, NY, USA; [4]University of Pisa, Pisa, Italy; [5]University of Toronto, Toronto, Canada; [6]Osaka University Graduate School of Medicine, Osaka, Japan; [7]Nanfang Hospital, Southern Medical University, Guangzhou, China; [8]Janssen Research & Development, LLC, South San Francisco, CA, USA; [9]Janssen Research & Development, Beerse, Belgium; [10]Janssen Research & Development, LLC, Titusville, NJ, USA

*Presenting author.

Introduction

- JNJ-73763989 (JNJ-3989) is a liver-targeted short-interfering RNA (siRNA) designed to target all hepatitis B virus (HBV) RNAs for degradation, thereby reducing all HBV viral proteins and pregenomic RNA[1]
- JNJ-56136379 (JNJ-6379) is a capsid assembly modulator that interferes with HBV replication by causing the formation of structually normal capsids that are devoid of HBV DNA and RNA (CAM-N)[2]
- The phase 2b REEF-1 study (ClinicalTrials.gov Identifier: NCT03982186) assessed the efficacy and safety of 48 weeks of JNJ-3989 and/or JNJ-6379 in combination with nucleos(t)ide analogues (NA) in patients with chronic hepatitis B (CHB)[3]
  - JNJ-3989 treatment resulted in a dose-dependent reduction in hepatitis B surface antigen (HBsAg) through follow-up Week 24; the greatest decline was observed with the subcutaneous (SC) 200 mg dose received every 4 weeks (Q4W)
  - There was no beneficial effect of coadministration with JNJ-6379 on HBsAg decline
  - JNJ-3989 and/or JNJ-6379 were safe and well tolerated

Objective

- To assess JNJ-3989- and/or JNJ-6379-induced changes to viral markers in CHB patients who were not currently treated (NCT) or virologically suppressed (VS) with NA treatment and who were hepatitis B e antigen (HBeAg)+ or HBeAg−

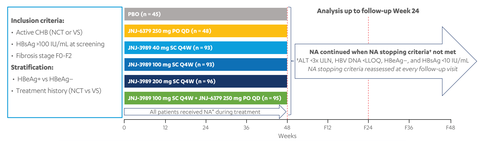
Methods

Study Design and Participants

- REEF-1 is a phase 2b, multicenter, double-blind, active-controlled, randomized study; results through follow-up Week 24 are reported here
- Eligible patients included those aged 18 to 65 years
- Patients were randomized to 6 treatment arms (**Figure 1**), all of which included NA, and received study treatment for 48 weeks

- Patients who met the criteria for stopping NA treatment at Week 44 (primary endpoint; **Figure 1**) terminated NA treatment at the Week 48 visit and began a 48-week NA-free follow-up phase
- Patients could stop NA treatment and enter a 48-week NA-free follow-up phase at any time if NA stopping criteria were met
- Patients having met NA stopping criteria and having stopped NA treatment were monitored for HBV DNA and alanine aminotransferase (ALT); NA treatment restarted based on predefined NA retreatment criteria

Figure 1. Study design.



Primary endpoint: Proportion of patients meeting NA stopping criteria (ALT <3x ULN, HBV DNA <LLOQ, HBeAg−, and HBsAg <10 IU/mL) at Week 48

ETV, entecavir; F, follow-up; LLOQ, lower limit of quantitation; PBO, placebo; PO, oral; QD, daily; TAF, tenofovir alafenamide; TDF, tenofovir disoproxil fumarate; ULN, upper limit of normal.
*NA = ETV/TDF/TAF.

Results

Participants

- A total of 470 patients with CHB were included, with a mean age of 43 years and a mean duration of CHB infection of 25.4 years; 66% were male and 40% were Asian (**Table 1**)
- 95% of patients enrolled completed the 48-week treatment phase, with no differences across treatment groups

Table 1. Baseline Demographics and Clinical Characteristics by Treatment History and HBeAg Status at Screening

	NCT		VS	
Characteristic*	HBeAg+ (n = 75)	HBeAg− (n = 97)	HBeAg+ (n = 67)	HBeAg− (n = 231)
Male, n (%)	45 (60.0)	61 (62.9)	49 (73.1)	155 (67.1)
Asian, n (%)	42 (56.0)	16 (16.5)	48 (71.6)	84 (36.5)
Age, years	37.2 (10.99)	40.6 (10.17)	41.7 (9.06)	46.2 (10.21)
HBsAg, $\log_{10}$ IU/mL	4.48 (0.79)	3.95 (0.50)	3.61 (0.58)	3.46 (0.61)
HBV DNA, $\log_{10}$ IU/mL	7.93 (1.11)	5.07 (1.37)	Not applicable†	Not applicable†
ALT, U/L	107.1 (103.16)	92.5 (94.08)	24.4 (11.60)	23.7 (12.21)
HBeAg, $\log_{10}$ IU/mL	2.31 (1.16)	–	0.27 (0.79)	–
HBcrAg, n (%) <LLOQ‡	0	19 (19.8)	0	110 (48.2)
HBV RNA, n (%) <LOD§	0	30 (31.9)	23 (34.8)	202 (89.0)
Liver stiffness,‖ kPa	6.30 (1.88)	5.90 (1.45)	4.84 (1.33)	4.92 (1.38)

HBcrAg, hepatitis B core related antigen; LOD, limit of detection; SD, standard deviation.
*Mean (SD) unless otherwise noted.
†98% of patients had HBV DNA <LLOQ (1.3 $\log_{10}$ IU/mL = 20 IU/mL).
‡LLOQ = 3.0 $\log_{10}$ IU/mL.
§LOD = 2.49 $\log_{10}$ copies/mL.
‖Measured with FibroScan® Paris, France.

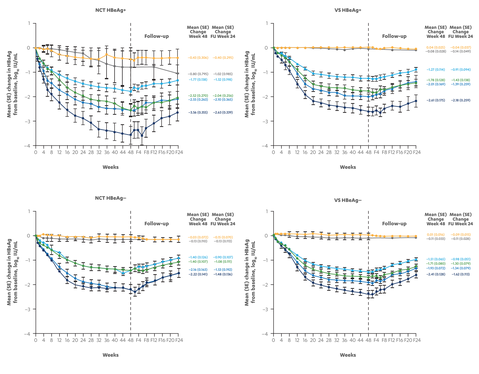
Primary Endpoint

- 19.1% (18/94) and 29.8% (28/94) of patients (all of whom were VS, except for 7) met NA stopping criteria with JNJ-3989 200 mg at Week 48 and until follow-up Week 24, respectively
  - For HBeAg− patients, the main reason for not meeting NA stopping criteria was not achieving HBsAg <10 IU/mL
  - For HBeAg+ patients, the main reasons for not meeting stopping criteria were not achieving HBeAg seroclearance and/or HBsAg <10 IU/mL and, in those who were also NCT, not achieving HBV DNA <LLOQ
- 2 of 63 (3.2%) patients who met NA stopping criteria and stopped NA treatment subsequently met NA restarting criteria through follow-up Week 24

Changes in HBsAg

- Consistent with the overall population,[3] JNJ-3989 reduced HBsAg in a dose-dependent manner in subgroups by treatment history and HBeAg status (**Figure 2**); the greatest mean declines were generally seen with the 200 mg dose
- The largest reductions of HBsAg were observed in NCT HBeAg+ patients (**Figure 2**)

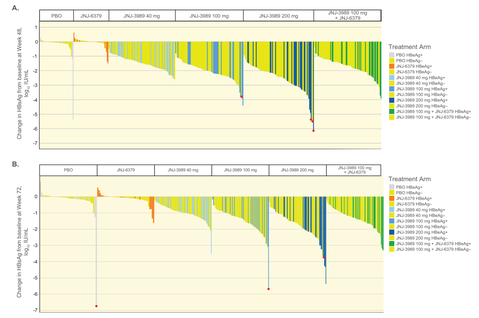
Figure 2. Mean change in HBsAg (±SE) from baseline by treatment history and HBeAg status.



FU, follow-up; SE, standard error.

- The JNJ-3989 200 mg arm had the highest proportion of patients who achieved HBsAg reduction ≥2 $\log_{10}$ and ≥3 $\log_{10}$ at Week 48 (73.6% and 27.5%, respectively; **Figure 3A**) and at the time of follow-up Week 24 (37.3% and 10.8%, respectively; **Figure 3B**)

Figure 3. Individual changes in HBsAg from baseline at (A) Week 48 and at (B) follow-up Week 24 in individual patients by HBeAg status.



Patients who achieved HBsAg loss are noted with red dots.

Changes in HBeAg

- Declines in HBeAg were observed across all treatment arms and sustained during follow-up, with reductions being JNJ-3989 dose dependent (**Figure 4**). Of note, the combination arm of JNJ-3989 100 mg + JNJ-6379 had the numerically greatest decline among NCT patients
- Reductions were most pronounced in NCT patients, potentially due to higher baseline HBeAg levels (**Figure 4** and **Table 2**)
- The number of patients in each treatment arm who achieved HBeAg <LLOQ was similar between NCT and VS patients, with the greatest proportion of patients reaching HBeAg <LLOQ in the JNJ-3989 100 mg (n = 3, 30.0%) and JNJ-3989 100 mg + JNJ-6379 (n = 4, 28.6%) treatment arms in VS patients (**Table 2**)

Figure 4. Mean (±SE) change in HBeAg over time in HBeAg+ patients (A) NCT or (B) VS.

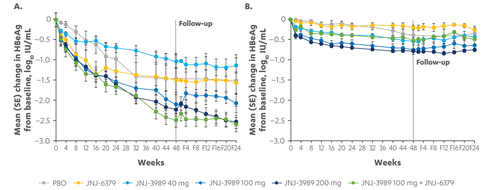


Table 2. Baseline and Change From Baseline HBeAg Values

	NCT and HBeAg+						VS and HBeAg+					
	N	BL, $\log_{10}$ IU/mL	Change from BL at W24, $\log_{10}$ IU/mL	<LLOQ at W24, n/N (%)	Change from BL at W48, $\log_{10}$ IU/mL	<LLOQ at W48, n/N (%)	Change from BL at FU W24, $\log_{10}$ IU/mL	<LLOQ at FU W24, n/N (%)	N	BL, $\log_{10}$ IU/mL	Change from BL at W24, $\log_{10}$ IU/mL	<LLOQ at W24, n/N (%)	Change from BL at W48, $\log_{10}$ IU/mL	<LLOQ at W48, n/N (%)	Change from BL at FU W24, $\log_{10}$ IU/mL	<LLOQ at FU W24, n/N (%)
PBO	7	1.92 (0.54)	−0.97 (0.35)	0/7	−1.45 (0.63)	1/7 (14.3)	−1.50 (0.64)	1/7 (14.3)	6	−0.02 (0.07)	−0.15 (0.07)	0/6	−0.39 (0.40)	1/6 (16.7)	−0.35 (0.36)	1/6 (16.7)
JNJ-6379	8	1.60 (0.42)	−1.27 (0.31)	1/7 (14.3)	−1.49 (0.31)	1/7 (14.3)	−1.56 (0.28)	1/8 (12.5)	7	−0.11 (0.18)	−0.18 (0.08)	0/6	−0.20 (0.16)	0/7	−0.26 (0.26)	0/7
JNJ-3989 40 mg	15	1.86 (0.38)	−0.69 (0.12)	1/15 (6.7)	−1.03 (0.17)	2/13 (15.4)	−1.13 (0.19)	2/15 (13.3)	15	0.67 (0.22)	−0.39 (0.05)	1/15 (6.7)	−0.54 (0.15)	1/14 (7.1)	−0.43 (0.36)	1/12 (8.3)
JNJ-3989 100 mg	14	2.77 (0.22)	−1.53 (0.13)	1/14 (7.1)	−2.09 (0.25)	2/14 (14.3)	−2.06 (0.24)	2/13 (15.4)	11	0.29 (0.30)	−0.60 (0.10)	3/11 (27.3)	−0.75 (0.39)	3/10 (30.0)	−0.64 (0.40)	3/10 (30.0)
JNJ-3989 200 mg	16	2.65 (0.25)	−1.60 (0.23)	0/15	−2.22 (0.30)	1/14 (7.1)	−2.52 (0.33)	2/14 (14.3)	14	0.40 (0.22)	−0.70 (0.12)	1/14 (7.1)	−0.78 (0.56)	1/14 (7.1)	−0.75 (0.53)	1/13 (7.7)
JNJ-3989 + JNJ-6379	13	2.52 (0.25)	−1.66 (0.20)	0/15	−2.48 (0.25)	1/12 (8.3)	−2.59 (0.26)	1/12 (8.3)	14	0.02 (0.69)	−0.39 (0.12)	2/14 (14.3)	−0.52 (0.48)	4/14 (28.6)	−0.49 (0.50)	1/13 (7.7)

BL, baseline; W, Week.
Values are mean (SE) unless otherwise noted; LLOQ = 0.11 IU/mL = −0.96 $\log_{10}$ IU/mL.

Changes in HBV DNA

- A numerically greater decline in HBV DNA was seen with JNJ-3989 100 and 200 mg and JNJ-6379-containing arms compared to control in NCT HBeAg+ patients (**Figure 5A**)
- Assessment of mean change from baseline in HBV DNA in NCT HBeAg− patients was limited by a high proportion of patients reaching HBV DNA <LLOQ in all treatment arms beginning at early time points (**Figure 5B** and **Table 3**)

Figure 5. Mean (±SE) change in HBV DNA over time in patients NCT and (A) HBeAg+ or (B) HBeAg−.

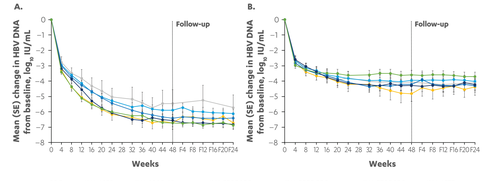


Table 3. Baseline and Change From Baseline HBV DNA Values

	NCT and HBeAg+						NCT and HBeAg−					
	N	BL, $\log_{10}$ IU/mL	Change from BL at W24, $\log_{10}$ IU/mL	<LLOQ at W24, n/N (%)	Change from BL at W48, $\log_{10}$ IU/mL	<LLOQ at W48, n/N (%)	Change from BL at FU W24, $\log_{10}$ IU/mL	<LLOQ at FU W24, n/N (%)	N	BL, $\log_{10}$ IU/mL	Change from BL at W24, $\log_{10}$ IU/mL	<LLOQ at W24, n/N (%)	Change from BL at W48, $\log_{10}$ IU/mL	<LLOQ at W48, n/N (%)	Change from BL at FU W24, $\log_{10}$ IU/mL	<LLOQ at FU W24, n/N (%)
PBO	7	7.92 (0.49)	−5.03 (0.56)	2/7 (28.6)	−5.46 (0.90)	4/7 (57.1)	−5.72 (0.82)	5/7 (71.4)	9	5.20 (0.47)	−3.81 (0.43)	5/9 (55.6)	−4.07 (0.48)	9/9 (100)	−4.11 (0.47)	8/9 (88.9)
JNJ-6379	8	8.03 (0.31)	−6.17 (0.38)	1/7 (14.3)	−6.72 (0.43)	5/7 (71.4)	−6.72 (0.42)	6/8 (75.0)	10	5.24 (1.14)	−4.22 (0.47)	7/9 (77.8)	−4.83 (0.50)	8/8 (100)	−4.39 (0.55)	9/9 (100)
JNJ-3989 40 mg	15	7.57 (0.38)	−5.28 (0.25)	2/15 (13.3)	−5.89 (0.29)	8/14 (57.1)	−6.12 (0.29)	8/15 (53.3)	18	4.87 (0.30)	−3.84 (0.29)	15/16 (93.8)	−3.96 (0.36)	15/16 (93.8)	−4.02 (0.33)	16/16 (100.0)
JNJ-3989 100 mg	14	7.77 (0.29)	−5.44 (0.28)	3/14 (21.4)	−6.41 (0.22)	8/14 (57.1)	−6.40 (0.25)	8/13 (61.5)	19	5.29 (0.33)	−4.08 (0.23)	15/18 (83.3)	−4.22 (0.36)	13/18 (72.2)	−4.31 (0.42)	13/16 (81.3)
JNJ-3989 200 mg	16	8.29 (0.21)	−6.07 (0.16)	2/15 (13.3)	−6.56 (0.20)	3/14 (21.4)	−6.82 (0.26)	7/14 (50.0)	19	5.26 (0.35)	−4.16 (0.32)	14/18 (77.8)	−4.28 (0.35)	16/18 (88.9)	−4.24 (0.38)	15/17 (88.2)
JNJ-3989 + JNJ-6379	13	8.04 (0.28)	−6.00 (0.19)	2/13 (15.4)	−6.72 (0.24)	6/12 (50.0)	−6.87 (0.27)	9/12 (75.0)	20	4.62 (0.24)	−3.63 (0.26)	18/19 (94.7)	−3.60 (0.29)	17/18 (94.4)	−3.71 (0.29)	18/18 (100)

Values are mean (SE) unless otherwise noted.

Changes in HBcrAg

- JNJ-3989 200 mg resulted in pronounced reduction of HBcrAg in NCT patients, with up to 2.56 $\log_{10}$ reduction at Week 48 in the HBeAg+ subgroup, while HBcrAg decline in VS patients was limited, potentially due to lower baseline levels (**Figure 6** and **Table 4**)
- The proportion of patients reaching HBcrAg <LLOQ was greatest in the NCT and HBeAg− patients who received JNJ-3989 200 mg (**Table 4**)

Figure 6. Mean (±SE) change in HBcrAg over time by treatment history and HBeAg status in patients with HBcrAg >LLOQ at baseline who received treatment with either PBO or JNJ-3989 200 mg.

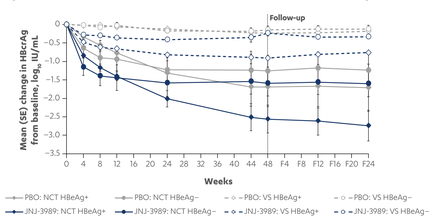


Table 4. Baseline and Change From Baseline Mean (SE) HBcrAg by Subgroup for NA and JNJ-3989 200 mg Treatment Arms in Patients With HBcrAg >LLOQ at Baseline

	N*	BL, $\log_{10}$ IU/mL	Change from BL at W24, $\log_{10}$ IU/mL	<LLOQ at W24, n/N (%)	Change from BL at W48, $\log_{10}$ IU/mL	<LLOQ at W48, n/N (%)	Change from BL at FU W24, $\log_{10}$ IU/mL	<LLOQ at FU W24, n/N (%)
NCT HBeAg+								
PBO	7	7.76 (0.47)	−1.31 (0.33)	0/7	−1.69 (0.53)	0/7	−1.71 (0.59)	0/7
JNJ-3989 200 mg	16	8.43 (0.28)	−2.01 (0.18)	0/15	−2.56 (0.25)	0/14	−2.74 (0.30)	0/14
NCT HBeAg−								
PBO	8	4.8 (0.55)	−1.23 (0.32)	3/8 (37.5)	−1.26 (0.43)	3/8 (37.5)	−1.24 (0.45)	3/8 (37.5)
JNJ-3989 200 mg	16	4.71 (0.37)	−1.58 (0.37)	6/16 (37.5)	−1.59 (0.35)	7/16 (43.8)	−1.60 (0.36)	6/15 (40.0)
VS HBeAg+								
PBO	6	5.42 (0.14)	−0.12 (0.04)	0/6	−0.23 (0.08)	0/6	−0.18 (0.04)	0/6
JNJ-3989 200 mg	14	5.80 (0.22)	−0.82 (0.14)	0/14	−0.91 (0.37)	0/14	−0.76 (0.15)	0/14
VS HBeAg−								
PBO	11	3.73 (0.39)	−0.17 (0.06)	3/11 (27.3)	−0.14 (0.10)	1/11 (9.1)	−0.12 (0.10)	1/11 (9.1)
JNJ-3989 200 mg	22	3.92 (0.12)	−0.41 (0.07)	4/22 (18.2)	−0.23 (0.13)	2/22 (9.1)	−0.33 (0.09)	4/18 (22.2)

*Patients with detectable levels of HBcrAg at baseline.
Values are mean (SE) unless otherwise noted; LLOQ = 3 $\log_{10}$ IU/mL.

Changes in HBV RNA

- JNJ-3989 200 mg resulted in pronounced reduction of HBV RNA across all patient populations, with up to 3.66 $\log_{10}$ reduction in NCT HBeAg+ patients (**Figure 7** and **Table 5**)
- Reductions in HBV RNA were sustained through 24 weeks of follow-up
- Assessment of RNA reduction in VS patients was hampered by a high proportion of patients achieving HBV RNA <LOD during treatment

Figure 7. Mean (±SE) change in HBV RNA over time by treatment history and HBeAg status in patients with HBV RNA >LOD at baseline who received treatment with either PBO or JNJ-3989 200 mg.

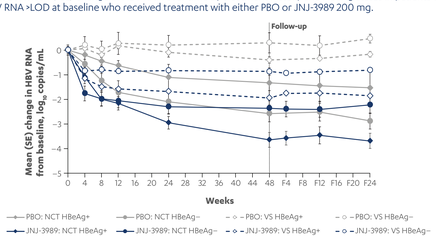


Table 5. Baseline and Change From Baseline Mean (SE) HBV RNA by Subgroup for PBO and JNJ-3989 200 mg Treatment Arms in Patients With HBV RNA >LOD at Baseline

	N*	BL, $\log_{10}$ copies/mL	Change from BL at W24, $\log_{10}$ copies/mL	<LOD at W24, n/N (%)	Change from BL at W48, $\log_{10}$ copies/mL	<LOD at W48, n/N (%)	Change from BL at FU W24, $\log_{10}$ copies/mL	<LOD at FU W24, n/N (%)
NCT HBeAg+								
PBO	6	6.90 (0.67)	−1.12 (0.53)	0/6	−1.34 (0.57)	0/6	−1.54 (0.62)	0/6
JNJ-3989 200 mg	16	7.33 (0.23)	−2.96 (0.26)	2/15 (13.3)	−3.66 (0.30)	4/14 (28.6)	−3.70 (0.31)	5/13 (38.5)
NCT HBeAg−								
PBO	5	5.02 (0.32)	−2.11 (0.47)	2/5 (40.0)	−2.59 (0.34)	3/5 (60.0)	−2.89 (0.33)	3/4 (75.0)
JNJ-3989 200 mg	13	4.64 (0.32)	−2.31 (0.30)	10/13 (76.9)	−2.37 (0.30)	11/13 (84.6)	−2.23 (0.36)	10/11 (90.9)
VS HBeAg+								
PBO	5	3.31 (0.47)	−0.10 (0.20)	1/5 (20.0)	−0.41 (0.14)	3/5 (60.0)	−0.18 (0.12)	1/5 (20.0)
JNJ-3989 200 mg	9	4.29 (0.44)	−1.69 (0.26)	5/9 (55.6)	−1.95 (0.35)	7/9 (77.8)	−1.87 (0.30)	6/8 (75.0)
VS HBeAg−								
PBO	3	3.25 (0.47)	0.20 (0.40)	1/3 (33.3)	0.29 (0.41)	1/3 (33.3)	0.46 (0.18)	0/2
JNJ-3989 200 mg	9	3.13 (0.14)	−0.84 (0.15)	8/9 (88.9)	−0.88 (0.17)	8/9 (88.9)	−0.75 (0.26)	5/7 (71.4)

*Patients with detectable levels of HBV RNA at baseline.
Values are mean (SE) unless otherwise noted; N = patients with detectable HBV RNA at baseline; LOD = 2.49 $\log_{10}$ copies/mL.

Key Findings

- Similar to HBsAg, a JNJ-3989 dose-response relationship was observed for HBeAg, HBcrAg, HBV DNA, and HBV RNA
- The greatest reductions in viral markers were observed during the 48-week treatment phase and in patients who were NCT and HBeAg+
- The reductions in HBeAg, HBcrAg, and HBV RNA generally remained stable or declined during the 24-week follow-up phase
- Safety results have been previously reported and showed that all regimens were generally safe and well tolerated[3]

Conclusions

- JNJ-3989, with or without JNJ-6379, reduced all viral markers, with the strongest effect observed for HBsAg
- This study supports the continued development of JNJ-3989 in combination therapies that may provide functional cure to patients with CHB

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Disclosures

M-FY serves as advisor/consultant for AbbVie, AlloVir International, Arbutus Biopharma, Bristol Myers Squibb, ClearB Therapeutics, Dicerna Pharmaceuticals, Gilead Sciences, GlaxoSmithKline, Janssen, Merck Sharp & Dohme, Roche, and Spring Bank Pharmaceuticals; and receives grant/research support from Assembly Biosciences, Arrowhead Pharmaceuticals, Bristol Myers Squibb, Fujirebio Diagnostics Inc., Gilead Sciences, Merck Sharp & Dohme, Roche, Spring Bank Pharmaceuticals, and Sysmex Corp. TA serves as advisor/consultant for AbbVie, Antios Therapeutics, Enyo Pharma, Gilead Sciences, GlaxoSmithKline, Janssen, and Roche. IMJ serves as a consultant for Aligos, Arbutus, Janssen, and Gilead; has received research funds from Janssen and Assembly Biosciences; and serves on a data monitoring committee for GlaxoSmithKline. MB serves as a consultant and speaker for AbbVie, Gilead Sciences, Janssen, Esai, MSD, and Roche. HLAJ received grants from AbbVie, Arbutus, Gilead Sciences, Janssen, and Roche; and is a consultant for Arbutus, Arena, Enyo, Gilead Sciences, GlaxoSmithKline, Janssen, Merck, Roche, Vir Biotechnology Inc., and Viroclinics. TT has received lecturer fees from Bristol Myers Squibb, Gilead Sciences, and GlaxoSmithKline; research funding from Janssen; and consulting fees from Janssen. JLH serves as advisor/consultant for Aligos, Assembly, Gilead Sciences, Johnson & Johnson, and Roche; and receives grant/research support from Bristol Myer Squibb. TNK, TL, RK, CG-A, CM, JJ, TV, OL, US, and MB are employees of Janssen Pharmaceuticals and may be Johnson & Johnson stockholders.

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