GILEAD | Medical Information

Hepcludex® (bulevirtide-gmod) Study MYR301: Efficacy and Safety

This document is in response to your request for information regarding the use of Hepcludex® (bulevirtide-gmod [BLV]) in the phase 3 MYR301 clinical study for the treatment of chronic HDV infection.

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The full indication, important safety information, and boxed warnings are available at: www.gilead.com/-/media/files/pdfs/medicines/hdv/hepcludex/hepcludex_pi.

Summary

Product Labeling[1]

BLV is indicated for the treatment of chronic HDV infection in adults without cirrhosis or with compensated cirrhosis.

This indication is approved under accelerated approval based on a decrease in HDV RNA and ALT normalization. An improvement in disease-related clinical outcomes has not been established. Continued approval for this indication may be contingent upon verification and description of clinical benefit in a confirmatory trial(s).

The recommended dosage in adults is BLV 8.5 mg once daily administered by SUBQ injection.

The efficacy of BLV once daily in the treatment of adults with chronic HDV infection without cirrhosis or with compensated cirrhosis is based on data through Week 144 from a multicenter, randomized, open-label, parallel-arm phase 3 trial, Trial MYR301 (NCT03852719), in which 100 participants received BLV 8.5 mg once daily. The MYR301 protocol specified the BLV dose as 10 mg; however, a dose recovery study later showed that the delivered dose was 8.5 mg.

Phase 3 MYR301 Clinical Study of BLV Use in Chronic HDV Infection

MYR301, a phase 3 study, evaluated the safety and efficacy of BLV 2 mg or 10 mg compared with delayed treatment (no-treatment arm) in adult participants with HDV (N=150).[2]

- At Week 48, rates of combined response (HDV RNA declines and ALT normalization; primary endpoint) were higher with BLV monotherapy (2 mg, 45%; 10 mg, 48%) than with no treatment (2%). Potential predictors of undetectable HDV RNA at Week 144 included lower HDV RNA and lower HBsAg levels at baseline (P<0.05).[3,4]
- Through Week 144, no BLV discontinuations, SAEs, or deaths were attributed to BLV, and no HBV reactivation was reported. ISRs occurred more frequently among participants in the BLV 10 mg arm than among those in the BLV 2 mg arm.

Dose-dependent elevations in total bile acids were asymptomatic and not associated with any clinical sequelae through Week 144.[3]

- In a subanalysis of participants who did not have a virologic response at Week 24, the proportion of participants in each treatment arm with a partial response or nonresponse decreased over time through Week 96.[5]
- Final results at Week 240 showed that response rates across all efficacy outcomes were sustained between 48 and 96 weeks after EOT. Across all arms, 65/149 participants (44%) had undetectable levels of HDV RNA at EOT; of those with follow-up data, 23/64 participants (36%) sustained undetectable levels through 96 weeks after EOT. Most relapses (93%) occurred within 24 weeks after EOT, and all relapses occurred within 48 weeks after EOT.[2]

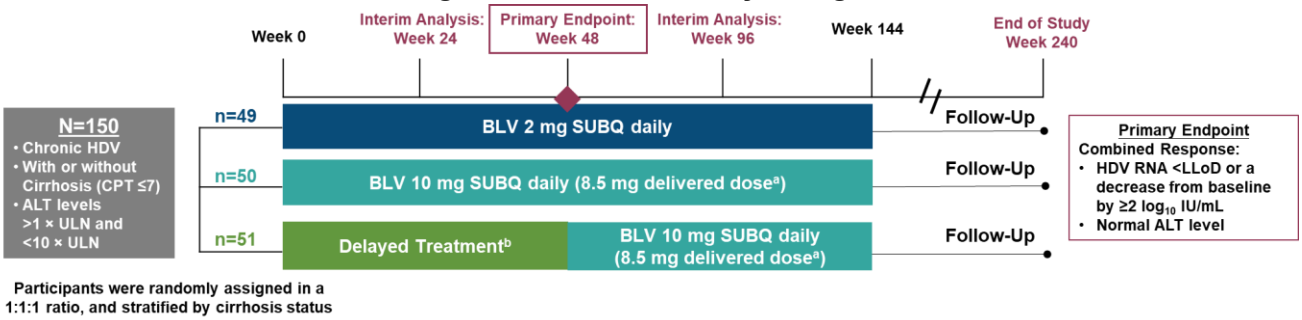
Phase 3 MYR301 Clinical Study of BLV Use in Chronic HDV Infection

Study Design and Demographics

MYR301 was an open-label, multicenter, randomized, phase 3 study that evaluated the safety and efficacy of immediate treatment with BLV 2 mg or 10 mg compared with delayed treatment with BLV 10 mg (no-treatment arm) in participants with chronic HDV (N=150).[2]

The primary endpoint was the combined response of undetectable (<LLoD) HDV RNA or a decrease of ≥2 $\log_{10}$ IU/mL from baseline and ALT level normalization at Week 48. Other endpoints included the following: rates of undetectable HDV RNA levels; rates of ALT normalization; and change from baseline in LSM, as measured by elastography.[4,6-8]

Figure 1. MYR301: Study Design[4]



[a]The MYR301 protocol specified the dose as 10 mg per vial, the delivered dose was 8.5 mg.
[b]Participants in the delayed treatment arm did not receive BLV treatment through Week 48.

Table 1. MYR301: Baseline Demographics and Disease Characteristics[4]

Key Demographics and Characteristics		Delayed Treatment BLV 10 mg (n=51)	BLV 2 mg (n=49)	BLV 10 mg (n=50)
Age, mean (SD), years		41 (7.5)	44 (9)	41 (8.5)
Male, n (%)		26 (51)	30 (61)	30 (60)
Race, n (%)	White	40 (78)	41 (84)	43 (86)
	Asian	11 (22)	8 (16)	6 (12)
	Black	0	0	1 (2)
Cirrhosis,[a] n (%)		24 (47)	23 (47)	24 (48)
LSM,[b] mean (SD), kPa		15 (9)	14 (8.2)	15 (9.3)

Key Demographics and Characteristics		Delayed Treatment BLV 10 mg (n=51)	BLV 2 mg (n=49)	BLV 10 mg (n=50)
HDV RNA, mean (SD), log_{10} IU/mL		5.1 (1.36)	5 (1.32)[c]	5.1 (1.4)
HDV GT, 1/5, n (%)		51 (100)/0	49 (100)/0	48 (96)[c]/1 (2)
HBV DNA, mean (SD), log_{10} IU/mL		0.9 (0.99)	1.3 (1.3)[c]	1.1 (1.26)[c]
HBeAg not detected, n (%)		47 (92)	45 (92)	43 (86)
HBsAg, mean (SD), log_{10} IU/mL		3.7 (0.47)	3.7 (0.52)	3.6 (0.59)
HBV GT, n (%)	A/D/E	4 (8)/39 (77)/0	2 (4)/44 (90)/0	3 (6)/43 (86)/1 (2)
	Could not be determined	8 (16)	3 (6)	3 (6)
ALT level, mean (SD), U/L		102 (61.9)	108 (62.5)	123 (80.6)
Previous IFN therapy, n (%)		29 (57)	26 (53)	29 (58)
Concomitant HBV nucleo(t)side analog treatment, n (%)		32 (63)	31 (63)	27 (54)

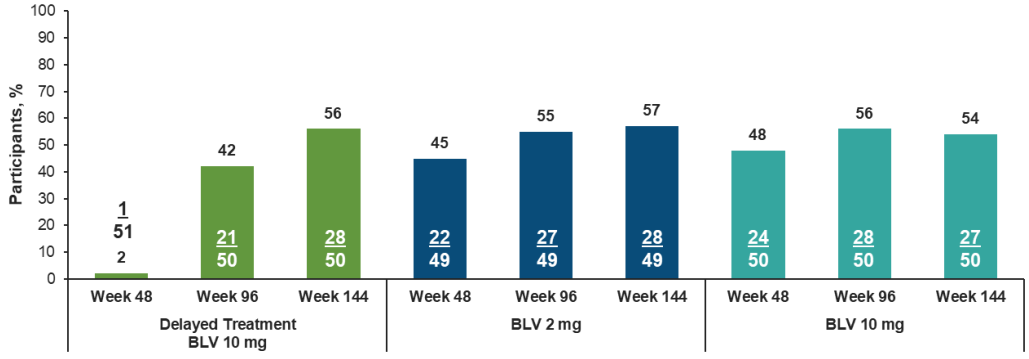
Abbreviations: HBeAg=hepatitis B envelope antigen; IFN=interferon.
[a]According to CPT, Class A cirrhosis (ie, scores of 5 or 6) correlates with mildly impaired liver function; Class B (ie, scores of 7 to 9) correlates with moderately impaired liver function; and Class C (ie, scores of 10 to 15) correlates with severely impaired liver function.
[b]Assessed using transient elastography (FibroScan, Echosens; range: 2.5–7.5 kPa; higher levels indicate more severe liver scarring).
[c]One participant had missing data.

Results Through Week 144[3]

Efficacy

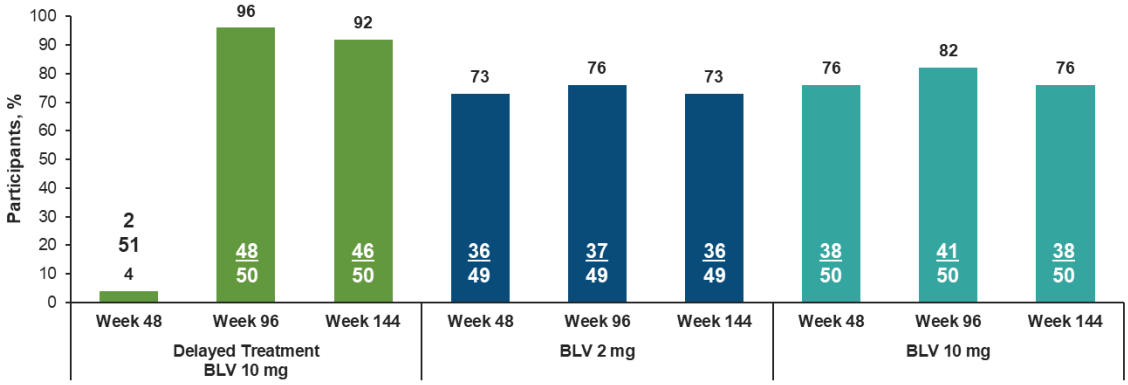
At the Week 48 primary endpoint, BLV monotherapy (2 mg and 10 mg) resulted in higher rates of combined response (ie, HDV RNA declines and ALT normalization) than no treatment. Combined response rates were numerically similar with BLV 2 mg and 10 mg monotherapy. Response rates between the BLV 2 mg and BLV 10 mg arms were also similar through Week 144, and increases in combined response (Figure 2), virologic response (Figure 3), and ALT normalization rates (Figure 4) observed at Week 96 were maintained through Week 144.

Figure 2. MYR301: Combined Response Rates (Virologic Response[a] and ALT Normalization[b]) Through Week 144[3,4]



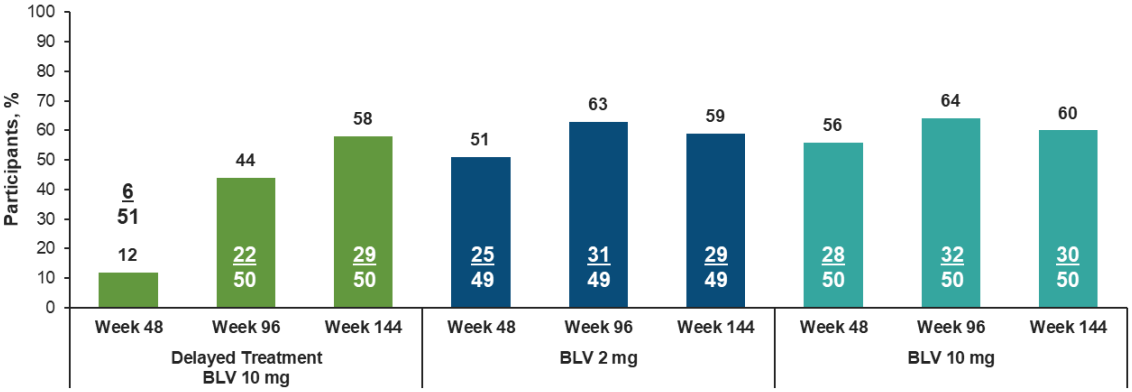
[a]Defined as an undetectable (<LLoD) HDV RNA or a decrease of ≥2 log_{10} IU/mL from baseline.
[b]Defined at Russian sites as ≤31 U/L for females and ≤41 U/L for males; at all other sites, it was defined as ≤34 U/L for females and ≤49 U/L for males.
Note: Week 48, 96, Week 144 data for the delayed treatment group reflect treatment durations of 0, 48, and 96 weeks of BLV 10 mg treatment, respectively.

Figure 3. MYR301: Virologic Response[a] Through Week 144[3]



[a]Defined as an undetectable (<LLoD) HDV RNA or a decrease of ≥2 $\log_{10}$ IU/mL from baseline.
Note: Week 48, 96, Week 144 data for the delayed treatment group reflect treatment durations of 0, 48, and 96 weeks of BLV 10 mg treatment, respectively.

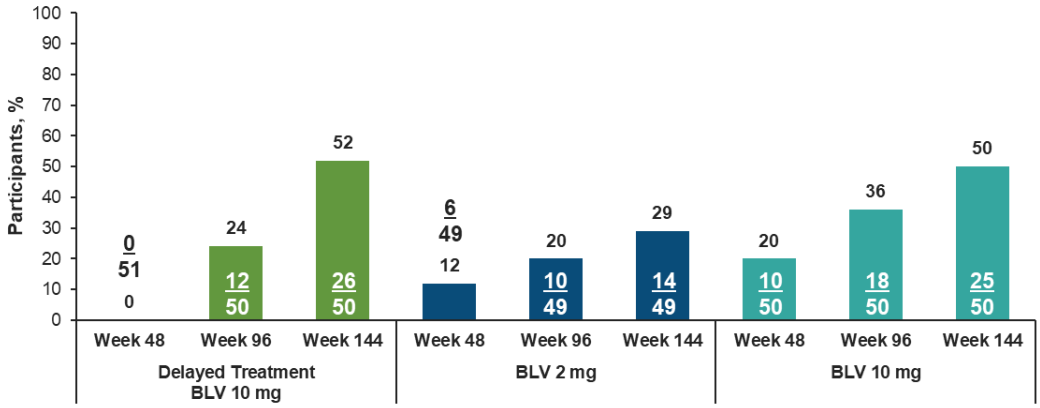
Figure 4. MYR301: ALT Normalization[a] Through Week 144[3]



[a]Defined at Russian sites as ≤31 U/L for females and ≤41 U/L for males; at all other sites, it was defined as ≤34 U/L for females and ≤49 U/L for males.
Note: Week 48, 96, Week 144 data for the delayed treatment group reflect treatment durations of 0, 48, and 96 weeks of BLV 10 mg treatment, respectively.

In both the BLV 2 mg and BLV 10 mg arms, rates of undetectable HDV RNA increased over 144 weeks (Figure 5); participants in the BLV 10 mg arm had numerically higher rates of undetectable HDV RNA and were the first to reach an undetectable level after a mean (SD) of 69 (41) weeks, compared with 77 (45) weeks in the BLV 2 mg arm. Potential predictors of undetectable HDV RNA at Week 144 included lower HDV RNA and lower HBsAg levels at baseline (P<0.05). In the delayed treatment arm, 1 participant experienced HBsAg loss without seroconversion.

Figure 5. MYR301: Undetectable HDV RNA[a] Through Week 144[3]



[a]Defined as <LLoD (50 IU/mL; target not detected).
Note: Week 48, 96, Week 144 data for the delayed treatment group reflect treatment durations of 0, 48, and 96 weeks of BLV 10 mg treatment, respectively.

Safety

Through Week 144, no BLV discontinuations, SAEs, or deaths were attributed to BLV monotherapy, and no HBV reactivation was reported. ISRs occurred more frequently among those in the BLV 10 mg arm than among those in the BLV 2 mg arm. Dose-dependent elevations in total bile acids were asymptomatic and not associated with any clinical sequelae through Week 144.

Table 2. MYR301: Overall Safety Summary Through Week 144[3]

Safety Outcomes		Delayed Treatment BLV 10 mg (n=50)		BLV 2 mg (n=49)		BLV 10 mg[a] (n=50)	
		Week 48–96	Week 48–144	Week 96	Week 144	Week 96	Week 144
Any AE, n (%)		42 (84)	46 (92)	47 (96)	48 (98)	48 (96)	48 (96)
Any Grade 3–4 AE, n (%)		3 (6)	5 (10)	9 (18)	12 (24)	8 (16)	10 (20)
Any SAE, n (%)		2 (4)	3 (6)	2 (4)	3 (6)	4 (8)	6 (12)
Any BLV-related AE, n (%)		22 (44)	23 (46)	25 (51)	27 (55)	36 (72)	37 (74)
Total bile salts, mean (SD), mcmol/L		63 (50)[b]	64 (59)[b]	24 (26)	28 (40)	52 (83)	59 (63)
AEs of special interest,[c] n (%)	Headache	7 (14)	7 (14)	9 (18)	10 (20)	12 (24)	12 (24)
	ISRs[d]	6 (12)	8 (16)	10 (20)	10 (20)	15 (30)	15 (30)
	Fatigue	2 (4)	3 (6)	7 (14)	7 (14)	9 (18)	9 (18)
	Nausea	1 (2)	1 (2)	3 (6)	3 (6)	6 (12)	6 (12)
	Dizziness	1 (2)	1 (2)	2 (4)	2 (4)	4 (8)	4 (8)
	Pruritis	0	0	6 (12)	6 (12)	9 (18)	8 (16)

[a]Please note, in the MYR301 study, participants were given two 5-mg injections.
[b]Values reflect total bile salt levels after 48 and 96 weeks of BLV 10 mg treatment.
[c]Refers to AEs that occurred at a greater frequency with BLV than with delayed treatment.
[d]ISRs combined the following injection site AEs: bruising, dermatitis, erythema, hematoma, induration, pain, pruritus, rash, reaction, or swelling.

Over 144 weeks of BLV monotherapy, platelet counts, liver chemistries, LSMs, and HBsAg values remained stable or improved, including among the subgroup of participants with cirrhosis (data not provided by source; Table 3). In the delayed treatment group, 1 participant with baseline cirrhosis developed a mild case of ascites that improved with continued BLV treatment.

Table 3. MYR301: Laboratory Values Through Week 144[3]

Parameter	BLV 2 mg (n=49)			BLV 10 mg (n=50)		
	Baseline	Week 96	Week 144	Baseline	Week 96	Week 144
Platelets, mean (SD), $\times 10^9$/L	153 (52.5)	169 (69.1)	162 (62.5)	160 (53.1)	174 (70.7)	163 (63.5)
<90 $\times 10^9$/L, %	14	13	14	12	11	14
Total bilirubin, median (Q1, Q3), mcmol/L	10.4 (6.7, 14.7)	12.7 (7.5, 15.9)	13.6 (8.6, 19)	10.5 (7.6, 13.6)	10.2 (7.5, 14.5)	11.8 (7.2, 15)
Albumin, mean (SD), g/L	44 (3.1)	45 (2.6)	47 (2.9)	44 (3.2)	45 (3.2)	46 (3.3)
<35 g/L, %	0	0	0	2	2	0
GGT, median (Q1, Q3), U/L	57 (27, 73)	27 (18, 37)	25 (18, 37)	42 (27, 73)	24 (17, 36)	23 (19, 39)
INR, mean (SD)	1.12 (0.111)	1.1 (0.116)	1.12 (0.145)	1.14 (0.112)	1.1 (0.129)	1.14 (0.222)
HBsAg, mean (SD), $\log_{10}$ IU/mL	3.67 (0.515)	3.47 (0.578)	3.29 (0.866)	3.61 (0.593)	3.47 (0.638)	3.41 (0.583)
LSM, mean (SD), kPa	13.99 (8.19)	10.2 (6.434)	9.43 (5.281)	14.81 (9.265)	10.04 (6.754)	11.28 (9.836)
>15 kPa, %	31	15	11	30	15	19

Abbreviations: GGT=γ-glutamyltransferase; Q=quartile.

Subanalysis: Participants Without a Virologic Response at Week 24[5]

A subanalysis was performed to evaluate whether continued BLV 2 mg or 10 mg treatment up to Week 96 would result in improved virologic response rates and biochemical levels among the participants who did not achieve a virologic response at Week 24. The proportion of participants in each treatment arm with a partial response or nonresponse decreased over time (Table 4). Of the 14 participants with a nonresponse and 22 participants with a partial response at Week 24, 43% (n=6) and 82% (n=18) achieved a virologic response at Week 96, respectively. A total of 35% of participants with a nonresponse (n=5/14) and 5% of those with a partial response (n=1/22) at Week 24 had a nonresponse at Week 96.

Table 4. MYR301 Subanalysis: Changes in HDV RNA Levels From Week 24 Through Week 96[5]

Response, n (%)		Virologic Response Groups at Week 24					
		BLV 2 mg (n=47)			BLV 10 mg (n=47)		
		No Response (n=10)	Partial Response (n=12)	Virologic Response (n=25)	No Response (n=4)	Partial Response (n=10)	Virologic Response (n=33)
Week 48	No response	8 (80)	1 (8)	0	3 (75)	0	0
	Partial response	1 (10)	0	2 (8)	0	5 (50)	1 (3)
	Virologic response	1 (10)	11 (92)	23 (92)	1 (25)	5 (50)	32 (97)
Week 96	No response	4 (40)	1 (8)	0	1 (25)	0	0
	Partial response	3 (30)	0	2 (8)	0	3 (30)	2 (6)
	Virologic response	3 (30)	11 (92)	23 (92)	3 (75)	7 (70)	31 (94)

Note: No response was defined as having an HDV RNA decline of <1 $\log_{10}$ IU/mL from baseline. Partial response was defined as having an HDV RNA decline of 1 to 2 $\log_{10}$ IU/mL from baseline.

By Week 96, participants with a nonresponse or partial response at Week 24 had decreases in HDV RNA and ALT levels; numerically higher decreases were seen among those who had a partial response than among those who had a nonresponse (Table 5). Among the 6 participants who had a nonresponse at Week 96, 5 participants had ≥50% decrease in ALT levels from baseline, including 1 participant who had ALT levels within normal limits.

Table 5. MYR301 Subanalysis: Changes in ALT and HDV RNA Levels Through Week 96 Among Participants With a Partial Response or Nonresponse at Week 24[5]

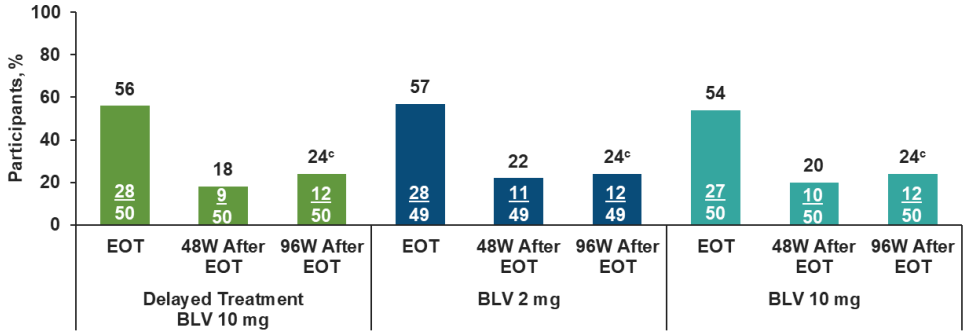
	Timepoint	Virologic Response Groups at Week 24	
		Nonresponse (n=14)	Partial Response (n=22)
ALT, mean (SD), U/L	Baseline	112 (59)	98 (64)
	Week 24	71 (37)	44 (26)
	Week 48	67 (42)	36 (14)
	Week 96	89 (123)	35 (24)
	Change from baseline at Week 96	-24 (119)	-64 (61)
HDV RNA, mean (SD), log_{10} IU/mL	Baseline	4.4 (2)	5.3 (1.4)
	Week 24	3.8 (1.8)	3.7 (1.4)
	Week 48	3.6 (2.1)	2.6 (1.5)
	Week 96	2.8 (2.1)	1.9 (1.3)
	Change from baseline at Week 96	-1.6 (1.7)	-3.4 (1.3)

Final Results at Week 240[2]

Efficacy

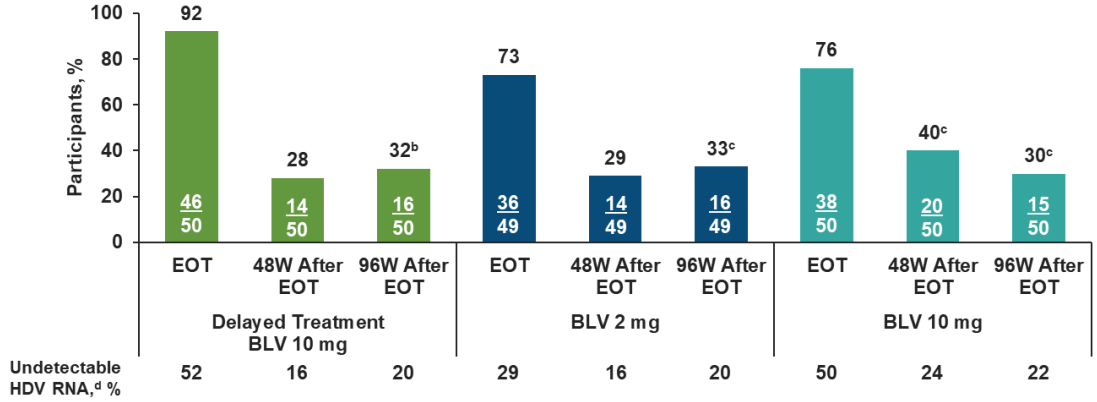
Results at Week 240 showed that response rates across all efficacy outcomes were sustained between 48 and 96 weeks after EOT (Figure 6, Figure 7, and Figure 8).

Figure 6. MYR301: Combined Response Rates (Virologic Response[a] and ALT Normalization[b]) at EOT and at 48 and 96 Weeks After EOT[2]



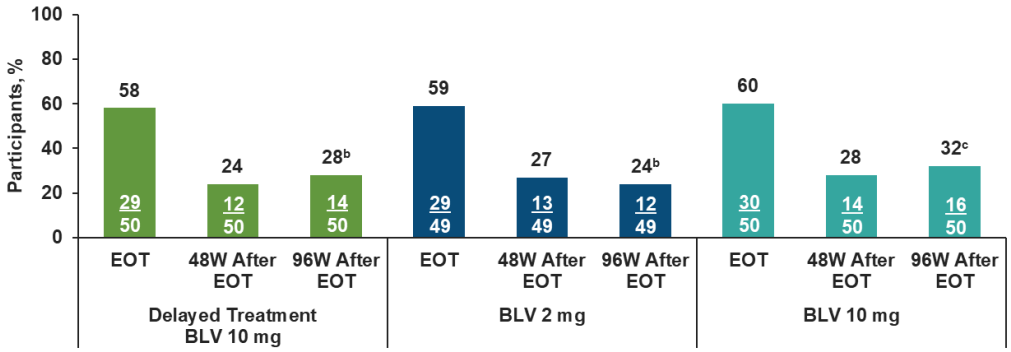
[a]Defined as an undetectable (<LLoD) HDV RNA or a decrease of ≥2 log_{10} IU/mL from baseline.
[b]Defined at Russian sites as ≤31 U/L for females and ≤41 U/L for males; at all other sites, it was defined as ≤34 U/L for females and ≤49 U/L for males.
[c]Included 1 participant who restarted BLV before the visit.

Figure 7. MYR301: Virologic Response[a] at EOT and at 48 and 96 Weeks After EOT[2]



[a]Defined as an undetectable (<LLoD) HDV RNA or a decrease of ≥2 $\log_{10}$ IU/mL from baseline.
[b]Included 2 participants who restarted BLV before the visit.
[c]Included 1 participant who restarted BLV before the visit.
[d]Quantified using RoboGene version 2.0, which has a LLoD of 6 IU/mL.

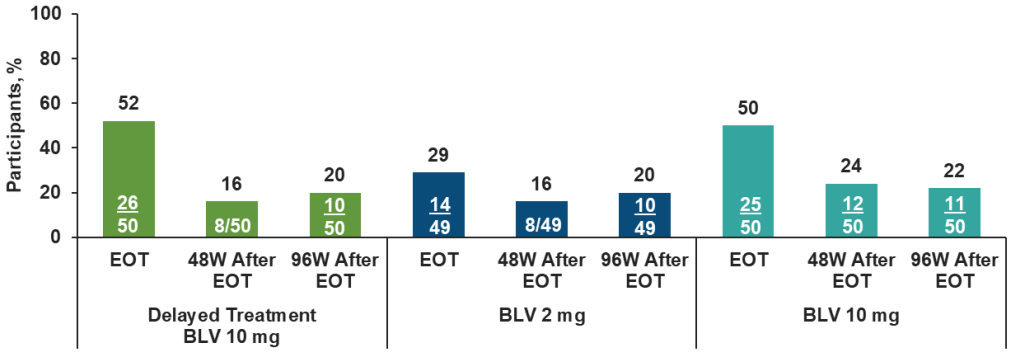
Figure 8. MYR301: ALT Normalization[a] at EOT and at 48 and 96 Weeks After EOT[2]



[a]Defined at Russian sites as ≤31 U/L for females and ≤41 U/L for males; at all other sites, it was defined as ≤34 U/L for females and ≤49 U/L for males.
[b]Included 1 participant who restarted BLV before the visit.
[c]Included 2 participants who restarted BLV before the visit.

At 96 weeks after EOT, 21% of participants overall had undetectable levels of HDV RNA (Figure 9). Across all arms, 65/149 participants (44%) had undetectable levels of HDV RNA at EOT; of those with follow-up data, 23/64 participants (36%) sustained undetectable levels through 96 weeks after EOT. Across all treatment arms, 90% of participants with ≥96 weeks of continuous undetectable levels of HDV RNA at EOT sustained undetectable levels after EOT. Most relapses (93%) occurred within 24 weeks after EOT, and all relapses occurred within 48 weeks after EOT.

Figure 9. MYR301: Undetectable HDV RNA[a] at EOT and at 48 and 96 Weeks After EOT[2]



[a]Quantified using RoboGene version 2.0, with an LLoD of 6 IU/mL.
Note: Some participants with undetectable levels of HDV RNA at 96 weeks after EOT had low-level viremia or transient virologic relapses during follow-up.

HBsAg loss occurred in 2 participants in the delayed treatment BLV 10 mg arm and in 1 participant each in the BLV 2 mg and BLV 10 mg arms.

Safety

During treatment, 1 participant in the delayed BLV 10 mg arm developed nonserious ascites. Between EOT and 96 weeks after EOT, 1 case of ascites was reported in the delayed BLV 10 mg arm, 1 case each of bleeding from varices and hepatocellular carcinoma was reported in the BLV 2 mg arm, and 1 case of hepatic encephalopathy was reported in the BLV 10 mg arm. Post-treatment ALT flares (ALT >10 × ULN) were reported in 6, 5, and 3 participants in the delayed BLV 10 mg, BLV 2 mg, and BLV 10 mg arms, respectively; most occurred early and resolved. Most participants (55–60%) remained in the study through 96 weeks after EOT, and withdrawal of consent was the most common reason for study discontinuation.

References

1. Enclosed. Gilead Sciences Inc. HEPCLUDEX® (bulevirtide) injection, for subcutaneous use. US Prescribing Information. Foster City, CA.
2. Wedemeyer H, Aleman S, Blank A, et al. Final Results of MYR301: A Randomised Phase 3 Study Evaluating the Efficacy and Safety of BLV Monotherapy for Chronic Hepatitis Delta. [Presentation #LBO-004]. Paper presented at: European Association for the Study of the Liver; May 7–10, 2025; Amsterdam, the Netherlands.
3. Lampertico P, Aleman S, Brunetto M, et al. Efficacy and Safety of 144 Weeks of Bulevirtide 2 mg or 10 mg Monotherapy From the Ongoing Phase 3 Study MYR301 [Poster LBP-029]. Paper presented at: European Association for the Study of the Liver (EASL); June 05-08, 2024; Milan, Italy.
4. Wedemeyer H, Aleman S, Brunetto MR, et al. A Phase 3, Randomized Trial of Bulevirtide in Chronic Hepatitis D. *N Engl J Med.* 2023;389(1):22-32.
5. Lampertico P, Wedemeyer H, Brunetto M, et al. Continued Treatment of Early Nonresponders or Partial Virologic Responders with Bulevirtide Monotherapy in Patients with Chronic Hepatitis Delta Through Week 96 Leads to Improvement in Virologic and Biochemical Responses. [Poster LBP-020]. Paper presented at: European Association for the Study of the Liver (EASL); June 21-24, 2023; Vienna, Austria.
6. Wedemeyer H, Aleman S, Brunetto M, et al. Efficacy and safety at 96 weeks of bulevirtide 2 mg or 10 mg monotherapy for chronic hepatitis D (CHD): results from an interim analysis of a phase 3 randomized study. [Abstract]. Paper presented at: European Association for the Study of the Liver (EASL); June 21-24, 2023; Vienna, Austria.

7. Wedemeyer H, Aleman S, Andreone P, et al. Bulevirtide Monotherapy at Low and High Doses in Patients With Chronic Hepatitis Delta: 24-Week Interim Data of the Phase 3 MYR301 Study [Poster 2730]. Paper presented at: European Association for the Study of the Liver (EASL): The Digital International Liver Congress; 23-26 June, 2021.
8. ClinicalTrials.gov. Study to Assess Efficacy and Safety of Bulevirtide in Participants With Chronic Hepatitis Delta (CHD). ClinicalTrials.gov Identifier: NCT03852719. Available at https://clinicaltrials.gov/ct2/show/study/NCT03852719. Accessed: April 2026. Last Updated: 22 August 2025.

Abbreviations

AE=adverse event
BLV=bulevirtide-gmod
CPT=Child-Pugh-Turcotte
EOT=end of treatment
GT=genotype
HBsAg=hepatitis B surface antigen
ISR=injection site reaction
LLoD=lower limit of detection
LSM=liver stiffness measurement
SAE=serious adverse event
SUBQ=subcutaneous(ly)
ULN=upper limit of normal

Product Label

For the full indication, important safety information, and boxed warning(s), please refer to the Hepcludex US Prescribing Information available at: www.gilead.com/-/media/files/pdfs/medicines/hdv/hepcludex/hepcludex_pi.

Follow-Up

For any additional questions, please contact Gilead Medical Information at:

☎1-866-MEDI-GSI (1-866-633-4474) or 🖱 www.askgileadmedical.com

Adverse Event Reporting

Please report all adverse events to:

Gilead Global Patient Safety ☎ 1-800-445-3235, option 3 or
🖱 www.gilead.com/utility/contact/report-an-adverse-event

FDA MedWatch Program by ☎ 1-800-FDA-1088 or ✉MedWatch, FDA, 5600 Fishers Ln, Rockville, MD 20852 or 🖱 www.accessdata.fda.gov/scripts/medwatch

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