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Hepcludex® (bulevirtide-gmod) Pharmacokinetics

This document is in response to your request for information regarding pharmacokinetic (PK) data of Hepcludex® (bulevirtide-gmod [BLV]).

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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 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 PK properties of BLV were characterized after IV administration in healthy participants, and after SUBQ administration in healthy participants and participants with chronic HDV infection. The systemic exposure of BLV increased in a more than proportional manner with increasing doses. At steady state, AUC and C_{max} increased by approximately 2-fold compared to the AUC and C_{max} after the first dose.

The absolute bioavailability of BLV is 57% and the T_{max} is 3 hours (range, 1–4 hours). BLV is >99% bound to human plasma proteins and the mean terminal plasma $t_{1/2}$ is 3 hours (range, 2–6 hours). BLV is catabolized by peptidases to amino acids; meaning, it is expected to be degraded to smaller peptides and individual amino acids. No active metabolites are expected, and the major route of elimination is via excretion of the smaller peptides and amino acids.

The steady state PK parameters of BLV (based on population PK analysis of participants with chronic HDV infection in MYR301 study, N=100) are as follows:

- C_{max} GM (90% CI), 184 (160–211) ng/mL
- $AUC_{0–24h}$ GM (90% CI), 1935 (1680–2230) ng·h/mL

Age (18–65 years), sex, race (87.5% White, 1.9% Black, 10.3% Asian, 0.2% other), or body weight (39.7–110 kg) did not have a clinically relevant impact on the systemic exposure of BLV.

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.

BLV PK Data in Clinical Studies

The PK profile of BLV was investigated in seven studies: four phase 1 studies and three phase 2 studies. The phase 1 studies included two studies in healthy volunteers (MYR101 and MYR102) and one study each in participants with severe RI and moderate HI without HBV/HDV infection compared with healthy MCs. One phase 2 study included participants with HBV (MYR201), and two included participants with CHD (MYR202 and MYR203).[2-4]

- The T_{max} was estimated to be 1 to 3 hours over a SUBQ BLV dose range of 0.8 to 10 mg.[5]
- After C_{max} was reached, plasma levels declined, and the mean $t_{1/2}$ of BLV 2 mg SUBQ ranged from 4 to 7 hours.[2,6]
- In one phase 1 study that evaluated the PK of daily doses of BLV 10 mg SUBQ in participants with severe RI, no significant differences in PK exposure were observed on Days 1 and 6 when compared with MCs.[4]
- In another phase 1 study that evaluated the PK of daily doses of BLV 10 mg SUBQ in participants with moderate HI, GLSM ratios of C_{max}, $AUC_{0–24}$, and $AUC_{0-\infty}$ were reduced by approximately 22% to 46% on Days 1 and 6, respectively, compared with MCs; however, exposure was still >5-fold higher than with BLV 2 mg. Higher Cl/F and Vz/F among participants with moderate HI was also observed on Days 1 and 6 vs MCs.[3]
- Across both studies in severe RI and moderate HI, PK exposures in participants with severe RI, moderate HI and in their MCs showed roughly 2-fold R_{ac} from Days 1 to 6, with a corresponding reduction in Cl/F and Vz/F. The median $t_{1/2}$ values ranged between 3 and 4 hours in participants with severe RI and moderate HI and were similar to their MCs.[3,4]

An analysis of PK data from six studies (MYR101, MYR102, MYR202, MYR203, MYR204, and MYR301) was performed to determine the population PK parameters in patients with and without HDV infection and the impact of patient-specific covariates that could have affected BLV exposure.[7]

- The absolute bioavailability of SUBQ BLV on Day 1 relative to IV BLV was 53.4%. The most influential covariate on the PK of BLV was weight; however, investigators did not consider these changes to be clinically meaningful.

BLV PK Data in Real-World Studies

A real-world study evaluated BLV levels from a single time point in 72 consecutive patients who received BLV at a single center in Italy. Plasma BLV levels did not correlate with age; sex; BMI; the presence of cirrhosis; levels of HDV RNA, ALT, or bile acids; the achievement of virologic response; time between BLV dose and sampling (≤12 vs >12 hours); or duration of BLV treatment (≤48 vs 49–96 vs 97–144 vs >144 weeks). There was a non-significant inverse correlation between BLV use and HBsAg levels (r=0.23; *P*=0.06).[8]

- BLV levels were not significantly different between those with or without cirrhosis; levels were lower in those with CPT A5 than in those with A6, though the difference did not reach significance.

BLV PK Data in Clinical Studies

Phase 1 and 2 Studies

Study designs[2-4]

The PK profile of BLV was investigated in seven studies: four phase 1 studies and three phase 2 studies. The phase 1 studies included two studies in healthy volunteers (MYR101 and MYR102) and one study each in participants with severe RI and moderate HI without HBV/HDV infection compared with healthy MCs. One phase 2 study included participants with HBV (MYR201), and two included participants with CHD (MYR202 and MYR203).

Table 1. Phase 1 and 2 Study Designs With PK Parameter Assessments[2-4]

Study Name	Clinical Phase	Study Population	Treatment Groups[a]	n in PK Analysis
MYR101	1a	Healthy volunteers	BLV IV 300 ng to 20 mg	27
			BLV 800 mcg to 10 mg	9
MYR102	1	Healthy volunteers	BLV 10 mg + TDF 245 mg orally	12
Phase 1 study in severe RI[b]	1	Severe RI vs MCs	BLV 10 mg	20; 10 per arm
Phase 1 study in moderate HI[c]	1	Moderate HI vs MCs	BLV 10 mg	20; 10 per arm
MYR201 (HBV)	1b/2a	Participants with HBeAg- CHB	BLV 0.5 mg, 1 mg, 2 mg, 5 mg, and 10 mg	16
MYR202[d]	2	Participants with CHD	BLV (2 mg, 5 mg, and 10 mg) + TDF 245 mg orally	25

[a]All BLV doses were administered SUBQ unless noted otherwise.
[b]Severe RI was defined as eGFR of ≥15 to ≤29 mL/min/1.73 m^2 and participants who did not require dialysis within 90 days of study entry.
[c]Moderate HI was defined as Child-Pugh Class B
[d]In addition to this analysis, a main PK study was performed in which single plasma samples were collected from all study participants to investigate accumulation over time.

PK results

BLV demonstrated non-linear PK, and Cl/F and Vz/F decreased with increasing dose, while the AUC increased more than proportionally.[2,5,9]

Steady state is expected to be reached within the first few weeks, with roughly 2-fold R_{ac} for C_{max} and AUC. The AUC for a single dose of BLV 2 mg SUBQ was 46 ng/mL·h, with a median T_{max} of 0.5 hours in the phase 2 study in 4 participants with CHB. After reaching C_{max}, BLV plasma levels declined after a single dose of BLV 2 mg SUBQ, with a mean $t_{1/2}$ that ranged from 4 to 7 hours across several studies.[2] The T_{max} was 1 to 3 hours over a BLV dose range of 0.8 mg to 10 mg SUBQ in healthy volunteers.[5,9]

Overviews of the PK parameters with BLV 2 mg and 10 mg SUBQ in these studies are provided in Table 2 and Table 3, respectively.

Table 2. PK Parameters of BLV 2 mg SUBQ Dose Across Phase 2 Studies[2]

Study Details			PK Parameters					
Dose	**Name (N)**	**HBV or CHD and Study Drug(s)**	**C_{max},[a] ng/mL**	**T_{max},[b] h**	**AUC_{0-24},[a] ng·h/mL**	**$AUC_{0-\infty}$,[a] ng·h/mL**	**$t_{1/2}$,[c] h**	**Cl/F,[c] L/h**
Single dose (Day 1)	MYR201 HBV (N=4)	HBeAg- HBV BLV	11.55 (27.91)	0.5 (0–1)	45.84 (15.25)	NC	4.13 (1.45)	44.02 (7.06)
	MYR202 (N=9)	CHD BLV + TDF	73.3 (24)	1 (0.27–26.77)	NC	339.9 (56.6)	6.8 (2.1)	6.6 (3.1)
	MYR203 (N=10)	CHD BLV + PEG-IFNα	22.4 (74.4)	0.5 (0.5–2)	NC	117.3 (1.9)	6.6 (4.4)	20.27 (10.2)
Multiple dose (Day 14)	MYR201 HBV (N=4)	HBeAg- HBV BLV	17.76 (37.26)	1 (0.5–1)	88.25 (17.45)	NC	6.22 (4.9)	22.92 (3.99)
	MYR202 (N=9)	CHD BLV + TDF	139.5 (80.5)	0.5 (0.08–1.5)	574.1 (84.9)	NC	NC	4.2 (2.6)
	MYR203[d] (N=10)	CHD BLV + PEG-IFNα	30.1 (87.1)	1.2 (0.5–3.02)	NC	NC	NC	12.8 (8.9)

[a]Data are presented as GM (geometric CV%).
[b]Data are presented as median (range).
[c]Data are presented as mean (SD).
[d]Blood sampling was performed for up to 24 hours post dose after the first dose.

Table 3. PK Parameters of BLV 10 mg SUBQ Dose Across Phase 1 Studies[3,4]

Study Details			PK Parameters					
Dose	**Name**	**Participant Classification (N)**	**C_{max},[a] ng/mL**	**T_{max},[b] h**	**AUC_{0-24},[a] ng·h/mL**	**$AUC_{0-\infty}$,[a] ng·h/mL**	**$t_{1/2}$,[c] h**	**Cl/F,[a] L/h**
Single dose (Day 1)	Phase 1 study in severe RI	Severe RI (n=10)	121 (71.4)	4 (3–6)	843 (56.9)	NC	2.86 (2.45, 4.54)	16.8 (69.2)
		MCs (n=10)	146 (57.5)	4 (1.5–9)	876 (52.2)	NC	3.03 (2.67, 3.76)	19.1 (95.9)
	Phase 1 study in moderate HI	Moderate HI (n=10)	99.2 (95.1)	4 (1–9)	671.4 (64.7)	696.4 (60)	3.9 (2.6, 6.3)	22.8 (79.6)
		MCs (n=10)	126.8 (51.8)	5 (2–9)	853 (39.6)	859.4 (39.2)	2.8 (2.6, 3.2)	14.2 (57.7)
Multiple dose (Day 6)	Phase 1 study in severe RI	Severe RI (n=10)	311 (35.3)	3 (2–4.03)	1880 (34.1)	NC	2.79 (2.27, 2.99)	5.8 (29.1)
		MCs (n=10)	307 (50.8)	3 (3–6)	1810 (30.9)	NC	2.75 (2.46, 2.97)	6.1 (36.5)
	Phase 1 study in moderate HI	Moderate HI (n=10)	175 (64.7)	3 (3–6)	1266.4 (61.3)	1283.5 (60.1)	3.4 (2.6, 4.7)	9.6 (40.3)
		MCs (n=10)	260.6 (24.6)	3 (2–4)	1561.1 (22.2)	1568.8 (22)	2.8 (2.4, 3.1)	6.7 (24.2)

Abbreviations: NC=not calculated; Q=quartile.
[a]Data are presented as GM (geometric coefficient of variation%). [b]Data are presented as median (range).
[c]Data are presented as median (Q1, Q3).

In the study in participants with severe RI who received daily doses of BLV 10 mg SUBQ, no significant differences in PK exposure were observed between those with severe RI and their MCs on Days 1 and 6 based on the GLSM ratio evaluations of C_{max} and $AUC_{0–24}$. Cl/F and Vz/F also remained similar between groups.[4]

In the study in participants with moderate HI who received daily doses of BLV 10 mg SUBQ, GLSM ratios of C_{max}, $AUC_{0–24}$, and $AUC_{0-\infty}$ were reduced by approximately 22% to 46% on Days 1 and 6, respectively, compared with MCs; however, exposure was still >5-fold higher than with BLV 2 mg. Higher Cl/F and Vz/F was also observed on Days 1 and 6 compared with MCs.[3]

Across both studies, PK exposures in participants with severe RI, moderate HI and in their MCs showed roughly 2-fold R_{ac} from Days 1 to 6, with a corresponding reduction in Cl/F and Vz/F.[3,4]

In participants with severe RI and moderate HI, the median $t_{1/2}$ values ranged between 3 and 4 hours and were similar to those of their MCs.[3,4]

Additional details from phase 1 studies[3-5,9]

Two phase 1 studies in healthy volunteers aimed to gain the first information on the safety and tolerability of BLV, to identify a reasonable dose of BLV (MYR101), and to investigate a possible drug interaction with TDF (MYR102; Table 1). In MYR101, BLV was well tolerated, and no dose-limiting toxicities were observed up to the largest/highest administered doses (20 mg IV and 10 mg SUBQ). BLV displayed dose-dependent PK, and moderate inter-individual variability. In MYR102, single doses of BLV administered with TDF resulted in no significant changes in the PK of TDF.

Two separate phase 1 studies evaluated the PK of BLV 10 mg SUBQ on Days 1 and 6 in participants with severe RI or moderate HI compared with those in MCs (Table 1). Across both studies, TEAEs were generally mild, with no increases in severity and a similar frequency relative to MCs. No participants experienced Grade ≥3 TEAEs, serious AEs, or AEs resulting in discontinuation of BLV.

Additional details from phase 2 studies[2]

MYR201 HBV study

The systemic exposure of BLV 0.5, 1, 2, 5, and 10 mg SUBQ once daily for 2 weeks was studied in this phase 1b/2a study in participants with HBeAg- CHB (Table 1). PK parameters were estimated using non-compartmental analysis.

There was relatively high inter-individual variability. The exposure of BLV increased more than dose-proportionally with increasing doses. There was a trend towards a dose-dependent increase in elimination $t_{1/2}$ on Days 1 and 14. There was a corresponding decrease in total body clearance (with increasing doses). The systemic exposure of BLV increased after repeated dosing, with an R_{ac} of approximately 2-fold.

MYR202 study in participants with CHD

In a PK substudy, the systemic exposure of BLV (2, 5, and 10 mg SUBQ once daily) coadministered with TDF (245 mg orally once daily) was analyzed in participants with CHD (Table 1).

The systemic exposure of BLV increased more than dose-proportionally after a single dose and after repeated doses. Following BLV 2, 5, and 10 mg SUBQ once daily R_{ac} was determined to be 1.7-, 1.8-, and 1.4-fold, respectively.

Assessment of the Effect of Patient-Related Factors on PopPK[7]

Study design and demographics

An analysis of PK data from six phase 1 to 3 studies was performed to determine the popPK in patients with and those without HDV infection and the baseline characteristics that could have affected BLV exposure. This analysis included data from MYR101, MYR102, MYR202, MYR203, MYR204, and MYR301, and the patient-specific covariates that were evaluated included the following: baseline weight; BMI; age; sex; race; BLV dose and route of administration; health status (healthy or HDV-positive); cirrhosis status; HDV viral load; CrCl; presence of anti-drug antibodies; AST, ALT, and bilirubin levels; and concomitant oral antivirals and PEG-IFNα. The patient-specific covariates that had a significant effect on PK parameters were further evaluated and included in a final popPK model.

The dataset included 461 patients and a total of 5122 samples. Ninety-three percent of the study population had HDV; continuous and categorical covariates are shown in Table 4.

Table 4. Baseline Demographics and Disease Characteristics in PopPK Analysis[7]

Key Demographics and Characteristics	MYR101 (n=18)	MYR102 (n=12)	MYR202 (n=90)	MYR203 (n=75)	MYR204 (n=149)	MYR301 (n=99)	Total (N=443)
Age, median (range), years	25.5 (21–43)	32.5 (24–48)	39 (20–64)	37 (18–62)	40 (18–65)	41 (19–62)	39 (18–65)
Male sex at birth, n (%)	18 (100)	11 (92)	59 (66)	52 (69)	105 (70)	60 (61)	305 (69)
Race, White/ Black or AA/Asian/ other, n (%)	16 (89)/ 0/1 (6)/ 1 (6)	11 (92)/ 0/1 (8)/ 0	78 (87)/ 1 (1)/11 (12)/ 0	74 (99)/ 0/1 (1)/ 0	130 (87)/ 7 (5)/11 (7)/ 1 (1)	84 (85)/ 1 (1)/14 (14)/ 0	393 (89)/ 9 (2)/39 (9)/ 2 (<1)
Weight, median (range), kg	70 (64.7–88.1)	79.5 (63–99)	73.1 (51–110)	73.5 (49–107)	75 (39.7–106)	74.9 (48–105)	74.2 (39.7–110)
BMI, median (range), kg/m^2	22.3 (19.9–26)	24.3 (21.3–29)	24.8 (17.9–34.6)	24.2 (17–34.5)	24.8 (16.6–37)	24.7 (17.6–35.9)	24.7 (16.6–37)
CrCl, median (range), mL/min	136 (109–172)	145 (106–185)	117 (64.6–211)	120 (74.9–176)	119 (50.4–196)	112 (70.7–201)	119 (50.4–211)
HDV, n (%)	0	0	90 (100)	75 (100)	149 (100)	99 (100)	413 (93)
HDV RNA, median (range), IU/mL	N/A	N/A	315,000 (50–23,000,000)	1,740,000 (0–67,800,000)	372,000 (0–13,700,000)	155,000 (0–31,100,000)	369,000 (0–67,800,000)
Cirrhosis, n (%)	0	0	46 (51)	10 (13)	51 (34)	47 (47)	154 (35)
AST level, median (range), IU/mL	21 (12–32)	22.5 (13–28)	60.5 (26–321)	48 (24–766)	59 (25–377)	66 (24–216)	56 (12–766)
ALT level, median (range), IU/mL	19 (13–43)	22.5 (10–33)	98.5 (32–450)	78 (27–1810)	82 (33–741)	101 (25–335)	81 (10–1810)
Coadministration of PEG-IFNα, n (%)	0	0	2 (2)	45 (60)	99 (66)	0	146 (33)
Coadministration of TDF, n (%)	0	12 (100)	90 (100)	16 (21)	68 (46)	45 (45)	231 (52)

Abbreviation: AA=African American.

Results

A two-compartment model with sequential zero-first-order absorption and first-order elimination was used for the final PK model. The model adequately described data

according to diagnostic plots. The absolute bioavailability of SUBQ BLV on Day 1 relative to IV BLV was 53.4%. Increased body weight (vs median body weight) was associated with lower BLV exposure (AUC and C_{max}), whereas female sex (vs male) and presence of cirrhosis (vs no cirrhosis) were associated with slightly higher exposure values. The most influential covariate on the PK of BLV was weight (changes in C_{max} ranged from -17.6% to +30.6% in response to exposures to the fifth and 95th weight percentiles, respectively, relative to the median weight); however, investigators did not consider these changes to be clinically meaningful. The presence of cirrhosis and female gender had minimal effect (changes <20%) on exposure to BLV, and no other covariates significantly affected BLV exposure.

BLV PK Data in Real-World Studies

PK Sampling in a Single-Center, Real-World Study in Italy[8]

Study design and demographics

A real-world study evaluated BLV levels in 72 consecutive patients who received BLV at a single center in Italy. Plasma PK levels were sampled using a single time point. At the time of sampling, patients were a median (range) 56 (43–61) years of age, 53% were male, and 81% had cirrhosis; the median (range) bile acid and HBsAg levels were 20 (9–36) mcmol/L and 3.7 (3.1–3.9) log_{10} IU/mL, respectively. Patients had received BLV for a median (range) duration of 84 (72–138) weeks. Overall, 78% had achieved a virologic response, and 24% had target not detected; the median (range) HDV RNA was 2.3 (0.7–3.3) log_{10} IU/mL.

Results

Nearly all patients (97%; 70/72) had a detectable BLV level in plasma, with a median (range) level of 1.8 (0.6–7.8) ng/mL.

Plasma BLV levels did not correlate with age, sex, BMI, or the presence of cirrhosis (r-values not provided for each of these), nor did they correlate with the following biochemical or virologic features: HDV RNA (r=0.01); ALT (r=0.05); and bile acids (r=0.05). There was a non-significant inverse correlation between BLV and HBsAg levels (r=0.23; *P*=0.06).

Median (range) BLV levels were not significantly different between those with or without cirrhosis: 1.8 (0.6–8.4) ng/mL vs 1.9 (1.4–5.3) ng/mL, respectively (*P*=0.98). Among the 58 patients with cirrhosis, median (range) BLV levels were lower in those with CPT A5 than in those with A6, though the difference did not reach significance: 1.5 (0.6–7.2) ng/mL vs 4.5 (1.1–9.5) ng/mL, respectively (*P*=0.27).

Additionally, BLV levels did not correlate with the achievement of virologic response (*P*=0.67) or time between BLV dose and sampling (≤12 vs >12 hours; *P*=0.13). The duration of BLV treatment did not correlate with median (range) BLV levels: ≤48 weeks, 1.5 (0.8–2.9) ng/mL; 49 to 96 weeks, 2.9 (1.5–8.6) ng/mL; 97 to 144 weeks, 1.7 (0.6–9.3) ng/mL; and >144 weeks, 0.6 (0.4–2.5) ng/mL (*P*=0.13).

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Abbreviations

AE=adverse event
AUC=area under the concentration-time curve
$AUC_{0-\infty}$=AUC from Time 0 to infinity
AUC_{0-24}=AUC from Time 0 to 24 hours
BLV=bulevirtide-gmod
CHB=chronic HBV
CHD=chronic HDV
C_{max}=maximum drug concentration
Cl/F=apparent clearance
CPT=Child-Pugh-Turcotte
GLSM=geometric least-squares mean
GM=geometric mean
HBeAg=hepatitis B envelope antigen
HBsAg=hepatitis B surface antigen
HI=hepatic impairment
MC=matched control
PEG-IFNα=pegylated interferon α
PK=pharmacokinetic(s)
PopPK=population pharmacokinetics
R_{ac}=accumulation ratio
RI=renal impairment
SUBQ=subcutaneous(ly)
$t_{1/2}$=terminal half-life
TDF=tenofovir disoproxil fumarate
TEAE=treatment-emergent adverse event
T_{max}=time to maximal concentration
Vz/F=volume of distribution

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:

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