Vemlidy® (tenofovir alafenamide) Comparison With Entecavir

This document is in response to your request for information regarding Vemlidy® (tenofovir alafenamide [TAF]) compared with entecavir (ETV) in adult patients with chronic hepatitis B (CHB). This response was developed according to principles of evidence-based medicine and only contains data from prospective and retrospective studies (N≥1000).

Some data may be outside of the US FDA-approved prescribing information. In providing this data, Gilead Sciences, Inc. is not making any representation as to its clinical relevance or to the use of any Gilead product(s). For information about the approved conditions of use of any Gilead drug product, please consult the FDA-approved prescribing information.

The full indication, important safety information, and boxed warnings are available at: www.gilead.com/-/media/files/pdfs/medicines/liver-disease/vemlidy/vemlidy_pi.

Summary

Clinical Data on TAF Compared With ETV

In a meta-analysis that compared the efficacy and safety of TAF (n=2787) with those of ETV (n=3966) in patients with CHB, TAF demonstrated significantly higher 12-week (*P*=0.025) and 24-week (*P*=0.011) VR rates than ETV. Significant differences were also observed for 12-week (*P*=0.000) and 48-week (*P*=0.001) BR rates. No significant differences between groups in 48- or 96-week VR rates or 24- or 96-week BR rates were observed. No significant 96-week between-group differences were observed for the incidence of HCC, while TAF was associated with significantly fewer adverse events than ETV (*P*=0.004).[1]

Another meta-analysis that compared the efficacy and safety of TAF, ETV, and TDF as first-line therapy in patients with HBV-related liver failure found that TAF was associated with greater MELD score reduction at 12 weeks than ETV (SMD: -10.85), with similar effects between treatment groups observed at Weeks 4 and 24. Transplant-free survival was comparable across treatment groups, and no meaningful differences were observed in HBV DNA reduction, renal function, or safety outcomes.[2]

A retrospective cohort study examined the longitudinal changes in renal function in adult patients with CHB after they initiated TAF or ETV (PS-matched groups, n=578 per group). Over 4 years, the cumulative incidence of CKD progression by ≥1 stage was similar between treatment groups (PS-matched cohort, *P*=0.645; entire cohort, *P*=0.068).[3]

In a retrospective study that compared the efficacy of TAF (n=700) with that of ETV (n=2368) in TN patients with CHB, TAF treatment resulted in higher rates of VR and CR compared with ETV treatment (*P*<0.001 for each comparison). There were no between-group differences in BR (*P*=0.48).[4]

In a retrospective, PS-matched cohort study in Hong Kong that compared the rates of CKD progression in patients with CHB who received 12 months of treatment with either TAF (n=305) or ETV (n=1460), there was no significant difference between treatments in the cumulative 12-month incidence of CKD progression (TAF, 9.18%; ETV, 7.95%; *P*=0.492).[5]

In a retrospective real-world study that compared biochemical and virologic response rates between patients treated with TAF (n=270) and those treated with ETV (n=395), weighted BR rates at Week 96 were 70.6% with TAF and 79.4% with ETV, and weighted VR rates at Week 96 were 90.1% with TAF and 88% with ETV. Viral breakthrough resulted in drug discontinuation in 1.9% of patients (n=5) in the TAF group and 2.3% of patients (n=9) in the ETV group.[6,7]

Clinical Data on TAF Compared With ETV

Meta-Analysis in Patients With CHB: TAF vs ETV[1]

Study design

A meta-analysis compared the efficacy and safety of TAF (n=2787) with those of ETV (n=3966) in patients with CHB using data from 24 studies, which included three randomized controlled trials, seven prospective cohort studies and 14 retrospective cohort studies. The primary outcomes were 12-, 24-, 48- and 96-week VR (undetectable HBV DNA) and BR (ALT normalization) rates. Secondary outcomes were HBeAg seroconversion, HBeAg loss, HBsAg decline, HCC incidence, and AEs.

Results

Overall, patients treated with TAF achieved significantly higher 12-week (RR,1.64; 95% CI: 1.06–2.51; *P*=0.025) and 24-week (RR,1.37; 95% CI: 1.08–1.74; *P*=0.011) VR rates than patients treated with ETV, with no significant 48- or 96-week differences between treatment groups (RR,0.99; 95% CI: 0.92–1.07; *P*=0.798 and RR,1.03; 95% CI: 0.96–1.11; *P*=0.422, respectively).

TAF was also associated with significantly higher 12-week (RR,1.93; 95% CI: 1.35–2.74; *P*=0.000) and 48-week (RR, 1.07; 95% CI: 1.03–1.12; *P*=0.001) BR rates than ETV, with no significant 24- or 96-week differences between treatment groups (RR, 1.05; 95% CI: 0.77–1.43; *P*=0.776 and RR, 0.97; 95% CI: 0.83–1.13; *P*=0.683, respectively).

Significantly more patients treated with TAF achieved 24-week HBeAg loss (RR, 2.68; 95% CI: 1.21–5.94; *P*=0.015) than patients treated with ETV, with no significant 96-week differences between treatment groups. No significant 12-, 24-, and 48-week differences between treatment groups were observed for HBeAg seroconversion (RR, 1.62, 95% CI: 0.34–7.71; *P*=0.544). In addition, no significant 48-week differences between treatment groups were observed for HBsAg decline (SMD, -0.06; 95% CI: -0.29 to -0.18; *P*=0.635), and no HBsAg loss was observed in any patient treated with TAF or ETV.

No significant 96-week differences between TAF and ETV were observed for the incidence of HCC (RR, 0.40; 95% CI: 0.16–1.02; *P*=0.056). Overall, significantly fewer patients treated with TAF than with ETV experienced AEs (RR, 0.50; 95% CI: 0.31–0.80; *P*=0.004).

Meta-Analysis of TAF, ETV, and TDF in HBV-Related Liver Failure[2]

Study design

The efficacy and safety of TAF, ETV, and TDF treatments as first-line therapy for patients with HBV-related liver failure were compared in a meta-analysis using data from 10 studies. Outcomes included change in MELD score and HBV DNA levels, transplant-free survival, eGFR, and safety.

Results

A significantly higher proportion of TAF-treated patients than ETV-treated patients achieved reductions in MELD score at 12 weeks, while reductions were similar between treatment groups at Weeks 4 and 24 (Table 1). Transplant-free survival rates were comparable between groups at the timepoints evaluated, and no meaningful differences were observed in HBV DNA reduction at 2 weeks.

Table 1. Liver-Related Outcomes With TAF, ETV, and TDF (Li et al)[2]

Liver-Related Outcomes		ETV vs TAF	TAF vs TDF	ETV vs TDF
MELD score, SMD (95% CI)	Week 4	-1.81 (-5.35, 1.13)	0.06 (-3.89, 4.41)	-1.75 (-5.8, 2.09)
	Week 12	-10.85 (-21.47, -0.28)	6.15 (-4.24, 17.19)	-4.69 (-12.27, 3.95)
	Week 24	7.31 (-4.25, 18.4)	-7.69 (-19.52, 3.39)	-0.29 (-8.79, 7.13)
Reduction of HBV DNA, SMD (95% CI)	Week 2	0.8 (-0.26, 1.89)	-0.73 (-2.32, 0.87)	0.07 (-1.12, 1.29)
Transplant-free survival, OR (95% CI)	Week 4	0.74 (0.28, 2.24)	1.04 (0.29, 3.68)	0.77 (0.31, 2.12)
	Week 12	0.73 (0.27, 2.16)	0.76 (0.22, 2.49)	0.55 (0.23, 1.35)
	Week 24	1.56 (0.68, 3.28)	1.14 (0.47, 3.33)	1.77 (0.93, 3.93)
	Week 48	1.41 (0.46, 3.47)	0.85 (0.27, 2.99)	1.19 (0.47, 2.77)

Abbreviation: OR=odds ratio.

No significant differences between TAF, ETV or TDF were observed for the effects on eGFR or the incidence of hepatic encephalopathy, hepatorenal syndrome, gastrointestinal bleeding, ascites, and spontaneous peritonitis.

Retrospective Cohort Study: TAF vs ETV and Renal Dysfunction Risk[3]

Study design and demographics

A single-center, retrospective cohort study in South Korea was conducted to examine the longitudinal changes in renal function in adult patients with CHB who had not previously received treatment with NUCs before they started treatment with ETV or TAF. Eligible patients were HBsAg+ for >6 months with no history of HCC or non-HCC malignancy. Patients with <3 months of follow-up and patients with Stage 5 CKD, HCV, HDV, HIV, or missing eGFR data at baseline were excluded. The primary endpoint was the incidence of CKD progression by ≥1 stage. To minimize baseline characteristic differences, PS matching was conducted.

The entire cohort comprised 708 patients in the TAF group and 2635 patients in the ETV group. Compared with the ETV group, the TAF group had significantly fewer males

(61.8% vs 55.5%, respectively, *P*=0.03); fewer patients with diabetes (7.9% vs 4.4%, respectively, *P*<0.01) or liver cirrhosis (52.4% vs 38.1%, respectively, *P*<0.01); and significantly higher median HBV DNA levels (5.3 vs 5.8 $\log_{10}$ IU/mL, respectively, *P*<0.01), ALT levels (46 vs 61 IU/L, respectively, *P*<0.01), AST levels (48 vs 52 IU/L, respectively, *P*=0.01), and eGFR (81 vs 90 mL/min/1.73 m^2, respectively, *P*<0.01).

In the PS-matched cohort, baseline characteristics were similar between the TAF (n=578) and ETV (n=578) treatment groups. The median age was approximately 50 years in both groups; 55% and 55.6% were male, respectively; and the median HBV DNA levels were 5.5 and 5.6 $\log_{10}$ IU/mL. The following comorbidities were reported in the TAF and ETV groups: cirrhosis, 39.3% and 43.3%, respectively; diabetes, 4.7% each; and hypertension, 4.7% and 5.2%.

Results

Over 4 years, the cumulative incidence of CKD progression by ≥1 stage was similar between treatment groups in the entire cohort (*P*=0.068) and in the PS-matched cohort (aHR for TAF vs ETV, 0.86; 95% CI: 0.45–1.64; *P*=0.645).

Factors significantly associated with decreased renal function are presented in Table 2.

Table 2. Variables Associated With a Decline in Kidney Function (Lee et al)[3]

Variable	Multivariate Analysis
	aHR (95%CI); *P*-Value
Age, 1 year per increase	1.06 (1.04–1.08); <0.05
Male sex	0.21 (0.13–0.35); <0.05
Platelet count <100,000/mcL	1 (0.99–1); 0.054
Albumin	0.36 (0.29–0.46); <0.05
BMI	1.18 (1.03–1.31); 0.013
Hypertension	2.65 (1.79–3.94); <0.05
Diabetes mellitus	5.52 (3.96–7.69); <0.05

Safety data were not reported.

Retrospective Study in TN Patients: TAF vs ETV

Study design and demographics[4,8]

A retrospective study compared the efficacy of TAF (n=700) vs ETV (n=2368) in TN patients with CHB from sites in Argentina, South Korea, Japan, mainland China, Taiwan, and the US. A competing-risks regression analysis was performed to evaluate VR, BR, and CR (VR and BR). At baseline, patients in the ETV group had significantly higher HBV DNA and ALT levels than did those in the TAF group (Table 3).

Table 3. Baseline Demographics and Disease Characteristics (Li et al)[4]

Key Demographics and Characteristics		TAF (n=700)	ETV (n=2368)	*P*-Value	Standardized Difference[a]
Age, mean ± SD, years		50.2±13.5	49.9±13.1	0.68	0.017
Male, n (%)		396 (56.6)	1494 (63.1)	0.002	0.13
Study region, n (%)	Asia	407 (58.1)	2102 (88.8)	<0.001	0.74
	Outside Asia	293 (41.9)	266 (11.2)		
HBV DNA, mean ± SD, $\log_{10}$ IU/mL		5.5±2	5.8±2	0.001	0.14

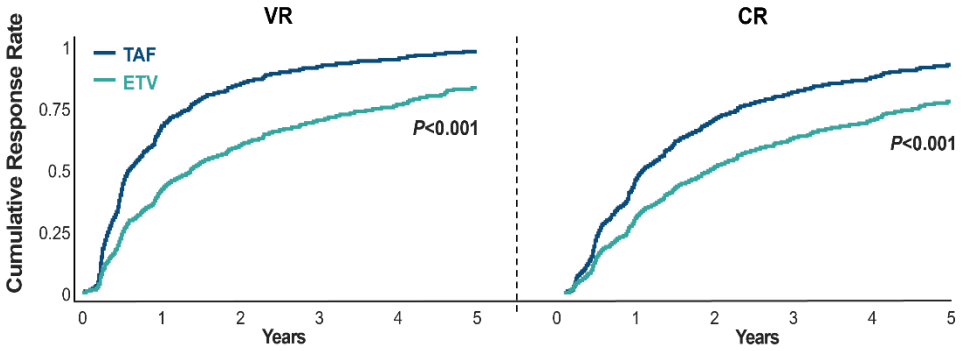
Key Demographics and Characteristics		TAF (n=700)	ETV (n=2368)	*P*-Value	Standardized Difference[a]
ALT level, median (range), IU/L		47 (29–98)	89 (39–193)	<0.001	0.31
ALT ≥2 × ULN, n (%)		274 (39.1)	1465 (61.9)	<0.001	0.47
HBeAg+, n (%)		187 (32.4)	888 (42.6)	<0.001	0.21
Fibrosis-4, n (%)	Low (<1.45)	271 (51.8)	714 (37.3)	<0.001	0.42
	Intermediate (1.45–3.25)	191 (36.5)	680 (35.5)		
	High (>3.25)	61 (11.7)	520 (27.2)		
Duration of follow-up, mean ± SD, years		3.1±1.7	5.6±4.2	<0.001	0.79

[a]Standardized difference values <0.1 indicate good balance in baseline characteristics and demographics, whereas values >0.1 indicate imbalance.
Note: Baseline demographics and characteristics were not available for all patients.

Results[4]

Overall, relative to the ETV group, the TAF group had significantly greater rates of VR and CR at Year 5 (Figure 1). The rate of BR did not differ between groups: TAF, 90.4%; ETV, 91.2% (*P*=0.48). After stratification by ALT levels (<2 × ULN vs ≥2 × ULN), each subgroup of patients who received TAF had higher rates of VR than those who received ETV: ALT <2 × ULN, 96.4% vs 77.2%, respectively; ALT ≥2 × ULN, 97.3% vs 86.8% (*P*<0.001 for each comparison). Similarly, rates of CR were higher in the TAF group than in the ETV group:ALT <2 × ULN, 90.3% vs 70.4%, respectively; ALT ≥2 × ULN, 93.2% vs 79.9% (*P*<0.001, for each comparison).

Figure 1. Competing Risk Regression Analyses: VR and CR (Li et al)[4]



After adjusting for confounders in a multivariate analysis, improved outcomes were observed with TAF vs ETV for VR (HR, 0.44; *P*<0.001) and CR (HR, 0.52; *P*<0.001). Treatment benefit with TAF vs ETV was also observed according to ALT stratification for VR (ALT <2 × ULN: HR, 0.39; ALT ≥2 × ULN: HR, 0.49; *P*<0.001 for each) and CR (ALT <2 × ULN: HR, 0.52; ALT ≥2 × ULN: HR, 0.48; *P*<0.001 for each).

Retrospective Study of CKD Progression With TAF vs ETV[5]

Study design and demographics

A retrospective, PS-matched cohort study in Hong Kong compared the rates of CKD progression, defined as a ≥1 stage increase in CKD stage for ≥3 consecutive months, in patients with CHB who received 12 months of treatment with either TAF (n=305) or ETV (n=1460). At baseline, the median (IQR) eGFR was 86.9 (73.3–100) mL/min/1.73 m^2 in the TAF cohort and 89 (74–99.7) mL/min/1.73 m^2 in the ETV cohort.

Results

At 12 months, the median (IQR) eGFR in the TAF and ETV cohorts was 84.8 (72.7–99.5) mL/min/1.73 m^2 and 88.2 (73.5–99.8) mL/min/1.73 m^2, respectively. CKD progression occurred in 28 patients who received TAF and in 116 patients who received ETV. There was no significant difference in the cumulative 12-month incidence of CKD progression between patients who received TAF (9.18%; 95% CI: 5.88–12.36%) and those who received ETV (7.95%; 95% CI: 6.55–9.32%; *P*=0.492).

Retrospective Study Comparing TAF, ETV, and TDF

Study design and demographics[6]

A multicenter, retrospective, real-world study in South Korea compared effectiveness outcomes among patients with CHB treated with TAF, ETV, or TDF. Included patients had received the treatment of interest for ≥3 months, were not co-infected with HCV or HIV, and did not have decompensated liver disease or HCC. Outcomes included the rate of BR, defined as the achievement of normal levels of AST or ALT (≤35 IU/L in males and ≤25 IU/L in females), and the rate of VR, as the achievement of undetectable HBV DNA levels <25 IU/mL. Analyses used IPTW to decrease bias and the impact of potential confounding clinical variables. After the IPTW, a time-dependent Cox proportional hazard model analyzed cumulative BR rates during the antiviral treatment, and the VR at each time point was utilized as a time-dependent variable.

A total of 1282 TN patients with CHB were included in the study (TAF, n=270; ETV, n=395; TDF, n=617). Overall, relative to those who received TAF or TDF, patients who were treated with ETV were older and more frequently had diabetes mellitus, hypertension, CKD, and negative HBeAg status (Table 4).

Table 4. Baseline Demographics and Disease Characteristics (Kim et al)[6]

Key Characteristics and Demographics		TAF (n=270)	ETV (n=395)	TDF (n=617)
Duration of follow-up,[a] mean ± SD, weeks		114.1±31.4	157.5±87.2	173.1±86.5
Male,[a] n (%)		135 (50)	223 (56.5)	375 (60.8)
Age,[a] mean ± SD, years		47.5±11.6	55.1±11.5	50.1±11.7
HBV DNA,[a] mean ± SD, log IU/mL		6±2.1	5.3±2.1	5.9±2
HBeAg+,[a] n (%)		135 (51.7)	144 (39.9)	305 (57.2)
Cirrhosis, n (%)		74 (27.4)	107 (27.1)	161 (26.1)
ALT,[a] mean ± SD, IU/L		120.9±173.7	201.4±400.2	199.4±375.5
AST,[a] mean ± SD, IU/L		85±115.6	147.5±278.9	147.1±342.9
eGFR, mean ± SD, mL/min		99.9±64	95.4±28.8	98.6±19.7
Comorbid conditions, n (%)	Radiologic fatty liver	74 (27.6)	83 (21.7)	164 (27)
	Dyslipidemia[a]	22 (8.1)	46 (11.6)	44 (7.1)
	Hypertension[a]	19 (7)	80 (20.3)	92 (14.9)
	Diabetes mellitus[a]	10 (3.7)	39 (9.9)	35 (5.7)
	Stage ≥3 CKD[a]	3 (1.1)	16 (4.1)	5 (0.8)
	Bone disorder, osteoporosis[a]	0	7 (1.8)	21 (3.4)

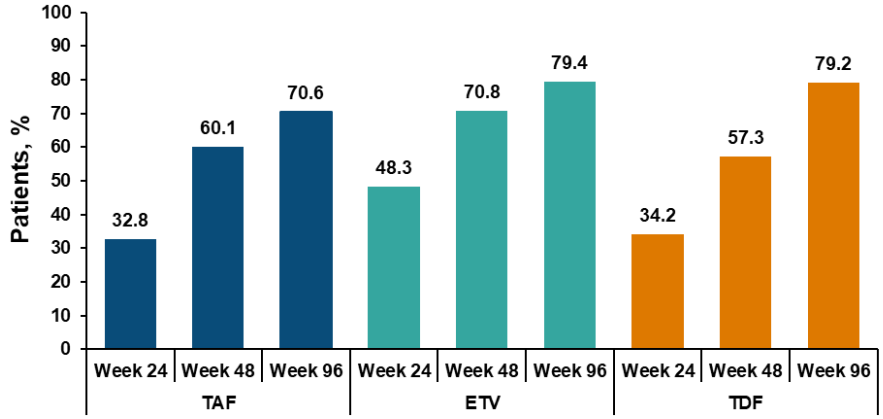
[a]*P*<0.05 for comparison of groups.

Results

In general, overall Week 96 BR rates after weighting were highest in the ETV cohort, and overall Week 96 VR rates after weighting were slightly higher in the TAF cohort than in the ETV and TDF cohorts (Figure 2 and Figure 3).[7] Rates in subgroups according to HBeAg

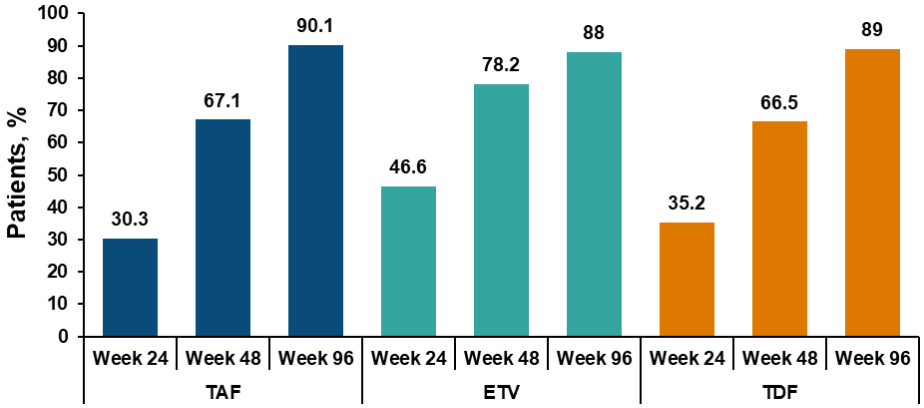
status are summarized in Table 5.[6,7] Univariable and multivariable Cox analyses were conducted after weighting to identify factors that affected BR rates. According to univariable analyses, several factors, including the presence of cirrhosis (HR, 0.64; 95% CI: 0.55–0.76; *P*<0.001), radiologic fatty liver (HR, 0.8; 95% CI: 0.67–0.95; *P*=0.009), obesity without diabetes mellitus (HR, 0.73; 95% CI: 0.61–0.88; *P*=0.001), and diabetes mellitus without obesity (HR, 0.61; 95% CI: 0.41–0.93; *P*=0.021) were associated with lower response rates. In multivariable time-dependent analyses, radiologic fatty liver (HR, 0.75; 95% CI: 0.61–0.94; *P*=0.01) and obesity without diabetes mellitus (HR, 0.85; 95% CI: 0.68–0.989; *P*=0.036) were independently associated with lower response rates, whereas treatment with ETV was associated with higher response rates than treatment with TAF and TDF (HR, 1.38; 95% CI: 1.13–1.68; *P*=0.002).[6]

Figure 2. Weighted BR Rates Through Week 96 (Kim et al)[7a]



[a]*P*=0.018 for comparison of groups.

Figure 3. Weighted VR Rates Through Week 96 (Kim et al)[7a]



[a]*P*=0.014 for comparison of groups.

Table 5. Weighted Response Rates Through Week 96 According to HBeAg Status (Kim et al)[6,7]

Response Rates, %		TAF (n=270)		ETV (n=395)		TDF (n=617)	
		HBeAg+	HBeAg-	HBeAg+	HBeAg-	HBeAg+	HBeAg-
BR[a]	Week 24	29.5	37.7	39.2	54.1	32.1	38
	Week 48	60.1	59.6	66.7	74.1	56.7	58.7
	Week 96	70.4	70.3	80.6	79.4	79.7	79.5
VR[b]	Week 24	19.4	45	25.4	58.2	24.5	48
	Week 48	53.2	83.2	49.4	91.6	53.4	80.4
	Week 96	83.8	97.2	69.4	96.5	81.7	98.7

[a]*P*=0.068 among HBeAg+ patients and *P*<0.001 among HBeAg- patients for comparison of groups.
[b]*P*=0.321 among HBeAg+ patients and *P*=0.05 among HBeAg- patients for comparison of groups.

The proportion of patients who discontinued drug therapy was lower in the TAF cohort (8.1%) than in the ETV (12.7%) and TDF cohorts (14.9%; *P*=0.021). Most discontinuations in all three cohorts were due to loss to follow-up; viral breakthrough resulted in drug discontinuation in 4.5% of patients (n=28) in the TDF cohort (primarily due to incomplete adherence [n=24]), 2.3% of patients (n=9) in the ETV cohort (reasons for viral breakthrough: incomplete adherence, n=5; drug-resistant mutation, n=4), and 1.9% of patients (n=5) in the TAF cohort (*P*=0.048).[6]

References

1. Luo JX, Chen G, Hu XY, Yu C. Tenofovir alafenamide versus entecavir in treating patients with chronic hepatitis B: A meta-analysis. Gastroenterol Hepatol. 2025;48(4):502276.
2. Li W, Wang Q, Wang Y, et al. Efficacy and safety of first-line nucleos (t)ide analogues for hepatitis B virus-related liver failure: a systematic review and network meta-analysis [Abstract PP0874]. Hepatol Int. 2025;19(Suppl 1):S850.
3. Lee Y, Cho M, Yang J, et al. Comparable Risk of Renal Dysfunction between Entecavir and Tenofovir Alafenamide in Patients with Chronic Hepatitis B [Poster 1407]. Paper presented at: American Association for the Study of Liver Diseases (AASLD); November 10-14, 2023; Boston, MA.
4. Li J, Chau A, Jun DW, et al. Antiviral Efficacy of Tenofovir Alafenamide Versus Entecavir in Treatment-Naïve Chronic Hepatitis B: A Multicenter Longitudinal Study. [Poster]. Paper presented at: AASLD The Liver Meeting; November 15-19 2024; San Diego, California.
5. Liang LY, Yip TC-F, Wong VW-S, Lai JC-T, Wong GL-H. Chronic Kidney Disease Progression in Patients With Chronic Hepatitis B on Tenofovir Alafenamide Versus Entecavir. [Poster #1265]. Paper presented at: AASLD The Liver Meeting; November 7-11, 2025; Washington, D.C.
6. Kim SH, Cho EJ, Jang B, et al. Comparison of biochemical response during antiviral treatment in patients with chronic hepatitis B infection. *Liver Int.* 2022;42(2):320-329.
7. Kim SH. Cho EJ. Jang B, Lee K, Choi JK, et al. Comparison of biochemical response during antiviral treatment in patients with chronic hepatitis B infection [supplemental tables]. *Liver International.*2022;(2):42:320-329.
8. Gilead Sciences Inc. Data on File.

Abbreviations

AE=adverse event
aHR=adjusted hazard ratio
BR=biochemical response
CHB=chronic hepatitis B
CKD=chronic kidney disease
CR=complete response
ETV=entecavir
HBeAg=hepatitis B envelope antigen
HBsAg=hepatitis B surface antigen
HCC=hepatocellular carcinoma
HR=hazard ratio
IPTW=inverse probability treatment weighting
MELD=Model for End-Stage Liver Disease
PS=propensity score
RR=risk ratio
SMD=standardized mean difference

TAF=tenofovir alafenamide
TDF=tenofovir disoproxil fumarate
TN=treatment-naive
ULN=upper limit of normal
VR=virologic response

Product Label

For the full indication, important safety information, and boxed warning(s), please refer to the Vemlidy US Prescribing Information available at: www.gilead.com/-/media/files/pdfs/medicines/liver-disease/vemlidy/vemlidy_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

Data Privacy

The Medical Information service at Gilead Sciences may collect, store, and use your personal information to provide a response to your medical request. We may share your information with other Gilead Sciences colleagues to ensure that your request is addressed appropriately. If you report an adverse event or concern about the quality of a Gilead or Kite product, we will need to use the information you have given us in order to meet our regulatory requirements in relation to the safety of our medicines.

It may be necessary for us to share your information with Gilead's affiliates, business partners, service providers, and regulatory authorities located in countries besides your own. Gilead Sciences has implemented measures to protect the personal information you provide. Please see the Gilead Privacy Statement (www.gilead.com/privacy-statements) for more information about how Gilead handles your personal information and your rights. If you have any further questions about the use of your personal information, please contact gilead.privacy@gilead.com.