GILEAD | Medical Information

# Epclusa® (sofosbuvir/velpatasvir) Use in Patients With Decompensated Cirrhosis

This document is in response to your request for information regarding the use of Epclusa® (sofosbuvir/velpatasvir [SOF/VEL]) for the treatment of chronic HCV infection in patients with decompensated cirrhosis. This response was developed according to principles of evidence-based medicine and contains data from prospective studies (N≥100).

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

## Summary

Product Labeling[1]

SOF/VEL is indicated for the treatment of adults and pediatric patients 3 years of age and older with chronic HCV GT 1, 2, 3, 4, 5, or 6 infection without cirrhosis or with compensated cirrhosis, or with decompensated cirrhosis for use in combination with RBV.

The recommended treatment regimen and duration for TN and TE patients with decompensated cirrhosis (CP Class B or C) is SOF/VEL + RBV for 12 weeks.

No dosage adjustment of SOF/VEL is recommended for patients with mild, moderate, or severe hepatic impairment (CP Class A, B, or C).

Clinical and hepatic laboratory monitoring (including direct bilirubin), as clinically indicated, is recommended for patients with decompensated cirrhosis receiving treatment with SOF/VEL + RBV.

Clinical Studies: SOF/VEL Use in Decompensated Cirrhosis

The phase 3 ASTRAL-4 study evaluated 12 or 24 weeks of SOF/VEL vs 12 weeks of SOF/VEL + RBV in participants with HCV GT 1 to 6 and CPT Class B decompensated cirrhosis. SVR12 was 94% (77/90) in the SOF/VEL + RBV group. The most common AEs in the SOF/VEL + RBV group included fatigue, nausea, anemia, headache, and diarrhea.[2]

In a multicenter study of SOF/VEL treatment in Japanese participants with decompensated cirrhosis, the SVR rate was 91.3% (188/206). Improvement in liver function was observed, as indicated by an increased proportion of participants with CP Class A at Week 24 after EOT, followed by a gradual decline through Year 5. Liver transplant–free survival rates at Years 1, 3, and 5 were 94%, 82.9%, and 69%, respectively. Overall, 43 participants died, and 4 underwent liver transplantation; causes of death included liver failure, non–liver-related conditions, HCC, and variceal rupture.[3]

In a study that included Japanese participants with decompensated cirrhosis (n=134) who received 12 weeks of SOF/VEL treatment for HCV infection, the SVR24 rate was 96% and there were significant improvements in median modified ALBI scores from baseline through 2 years (*P*<0.001).[4]

In a phase 3 study of SOF/VEL vs SOF/VEL + RBV for 12 weeks in Japanese participants with decompensated cirrhosis, SVR12 rates were 92% (47/51) in each group. The most common AEs were nasopharyngitis in the SOF/VEL group and anemia and diarrhea in the SOF/VEL + RBV group.[5]

- Among the 94 participants with decompensated cirrhosis who were followed up to 5 years after EOT, the overall proportion of participants with CP Class A was significantly greater than at baseline (40% vs 12%, respectively; *P*<0.001).[6]
- In multivariate analyses, virologic response (*P*=0.002) and CP Class C 12 weeks after EOT (*P*=0.001) were significantly associated with liver transplant–free survival.[6]

# Clinical Studies: SOF/VEL Use in Decompensated Cirrhosis

## ASTRAL-4: SOF/VEL ± RBV for 12 or 24 Weeks in Participants With GT 1 to 6 and Decompensated Cirrhosis

### Study design and demographics[2]

ASTRAL-4 was a phase 3, open-label study that evaluated the safety and efficacy (SVR12) of 12 or 24 weeks of SOF/VEL vs 12 weeks of SOF/VEL + RBV in TN and TE participants with HCV GT 1 to 6 and CPT Class B decompensated cirrhosis.

**Table 1. ASTRAL-4: Baseline Demographics and Disease Characteristics[2]**

| Key Demographics and Characteristics | SOF/VEL × 12 Wks (n=90) | SOF/VEL × 24 Wks (n=90) | SOF/VEL + RBV × 12 Wks (n=87) |
|---|---|---|---|
| Race, White/Black, % | 88/7 | 90/7 | 91/6 |
| HCV GT, 1/2/3/4/5/6, % | 76/4/16/4/0/0 | 79/4/13/2/0/1 | 78/5/15/2/0/0 |
| MELD score, <10/10–15/≥16, % | 40/56/4 | 29/66/6 | 33/62/5 |
| TE, n (%) | 58 (64) | 42 (47) | 47 (54) |

### Efficacy

Overall, SVR12 was achieved by 83% (82/87) and 86% of participants (75/90) in the SOF/VEL 12- and 24-week groups, respectively, and by 94% of participants (77/90) in the SOF/VEL + RBV 12-week group. SVR12 rates by HCV GT are presented in Table 2.[2] SVR12 rates were sustained at the SVR24 checkpoint in all groups except the SOF/VEL 24-week group, which had an SVR24 rate of 88% (79/90).[7]

**Table 2. ASTRAL-4: SVR12 Rates by HCV GT[2]**

| HCV GT, n/N (%) | SOF/VEL × 12 Wks | SOF/VEL × 24 Wks | SOF/VEL + RBV × 12 Wks |
|---|---|---|---|
| 1a | 44/50 (88) | 51/55 (93) | 51/54 (94) |
| 1b | 16/18 (89) | 14/16 (88) | 14/14 (100) |
| 2 | 4/4 (100) | 3/4 (75) | 4/4 (100) |

| HCV GT, n/N (%) | SOF/VEL × 12 Wks | SOF/VEL × 24 Wks | SOF/VEL + RBV × 12 Wks |
|---|---|---|---|
| 3 | 7/14 (50) | 6/12 (50) | 11/13 (85) |
| 4 | 4/4 (100) | 2/2 (100) | 2/2 (100) |
| 6 | 0 | 1/1 (100) | 0 |

Twenty-two participants had virologic failure: 12% (11/90) in the SOF/VEL 12-week group, 9% (8/90) in the SOF/VEL 24-week group, and 3% (3/87) in the SOF/VEL + RBV group. Twenty participants relapsed, and 2 participants with GT 3 had virologic breakthrough.[2] Changes in CPT score from baseline to the 12- and 24-week follow-ups and SVR rates by baseline CPT class are presented in Table 3 and Table 4, respectively.[7]

**Table 3. ASTRAL-4: CPT Change From Baseline in Participants Who Achieved SVR[7]**

| SVR, n (%) | CPT Scores | | |
|---|---|---|---|
| | Improved CPT | No Change in CPT | Worsened CPT |
| SVR12 | 108 (47) | 99 (43) | 22 (10) |
| SVR24 | 115 (54) | 77 (36) | 21 (10) |

**Table 4. ASTRAL-4: SVR Results by Baseline CPT Class[7]**

| Baseline CPT Class, n/N (%) | SVR12 | SVR24 |
|---|---|---|
| A | 4/14 (29) | 6/13 (46) |
| B | 98/205 (48) | 102/191 (53) |
| C | 6/10 (60) | 7/9 (78) |

Of the participants who achieved SVR24, 39% of participants (84/213) had an improvement in albumin levels, 16% (35/213) had an improvement in bilirubin levels, 2% (5/213) had an improvement in INR, 15% (32/213) had an improvement in ascites, and 9% (20/213) had an improvement in encephalopathy.[7]

Improvements in MELD score were driven largely by improvements in total bilirubin, and improvements in MELD score at post-treatment Weeks 12 and 24 were more common in participants with higher MELD scores, lower BMI (<30 kg/m$^2$), or absence of encephalopathy at baseline.[7]

**Table 5. ASTRAL-4: Changes in MELD Score in Participants Who Achieved SVR24[7]**

| Baseline MELD Score, n (%) | MELD Score Results | | |
|---|---|---|---|
| | Improved MELD Score | No Change in MELD Score | Worsened MELD Score |
| <15 | 92 (49) | 47 (25) | 49 (26) |
| ≥15 | 18 (72) | 1 (4) | 6 (24) |

Of the 255 participants for whom pre-treatment NS5A sequencing data were available, 28% (72/255) had pre-treatment NS5A RAVs. Of these participants, 89% (64/72) achieved SVR, compared with 92% of participants (169/183) who did not have pre-treatment NS5A RAVs. Among participants with GT 1 who received SOF/VEL + RBV, the SVR rate in those with NS5A RAVs was 100%, and the rate in participants without such variants was 98%. Among participants with GT 1 in the SOF/VEL groups who had pre-treatment RAVs, the SVR rate was 80% for those who received 12 weeks of treatment and 90% for those who received 24 weeks of treatment. Among those who did not have RAVs, the SVR rates were 96% and 98%, respectively.[2]

## Safety[2]

A summary of key safety results is presented in Table 6.

**Table 6. ASTRAL-4: Summary of Safety Results[2]**

| Key Safety Parameters, n (%) | | SOF/VEL × 12 Wks (n=90) | SOF/VEL × 24 Wks (n=90) | SOF/VEL + RBV × 12 Wks (n=87) |
|---|---|---|---|---|
| AEs | | 73 (81) | 73 (81) | 79 (91) |
| AEs occurring in >15% of participants | Fatigue | 23 (26) | 21 (23) | 34 (39) |
| | Headache | 23 (26) | 17 (19) | 18 (21) |
| | Nausea | 22 (24) | 18 (20) | 22 (25) |
| | Diarrhea | 6 (7) | 7 (8) | 18 (21) |
| | Anemia | 4 (4) | 3 (3) | 27 (31) |
| SAEs | | 17 (19) | 16 (18) | 14 (16) |
| DCs due to AE | | 1 (1) | 4 (4) | 4 (5) |

Reductions in Hgb, lymphocytes, and platelets were common in all three groups. Decreases in Hgb to <10 g/dL and <8.5 g/dL occurred in 8% and 1% of participants, respectively, in the SOF/VEL 12-week group, in 9% and 1% in the SOF/VEL 24-week group, and in 23% and 7% in the SOF/VEL + RBV group. Overall, 3 participants in each of the three treatment groups died during the study, most due to complications of end-stage liver disease (ie, liver failure, sepsis, or multiorgan failure); none were considered to be treatment related. Two participants died after discontinuing study treatment, and 7 participants died >30 days after EOT.

# Long-Term Outcomes in SOF/VEL-Treated, Japanese Participants With Decompensated Cirrhosis[3]

## Study design and demographics

A multicenter study evaluated changes in liver function and liver transplant-free survival following treatment with SOF/VEL in Japanese participants with decompensated cirrhosis, defined as CP Class B or C at enrollment or CP Class A with prior decompensation. A total of 206 participants who received 12 weeks of SOF/VEL between February 2019 and December 2021 were enrolled. Clinical and laboratory data were collected at baseline, EOT, Weeks 12 and 24 after EOT, and every 6 months thereafter. Changes in liver function were evaluated based on the proportion of participants with CP Class A. For the survival analysis, the observation period began on the date of SOF/VEL initiation and ended at the earliest of death, liver transplantation, or last hospital visit.

**Table 7. Baseline Demographics and Disease Characteristics (Tahata et al)[3]**

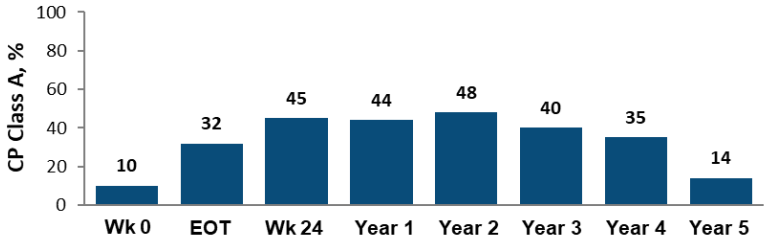
| Key Demographics and Characteristics | SOF/VEL (N=206) |
|---|---|
| GT, 1/2/3/4/1 + 2/unknown, n | 131/71/1/0/1/2 |
| HCV RNA, median (IQR), log IU/mL | 5.7 (5.1–6.1) |
| CP Class, A/B/C, n | 20/156/30 |
| MELD score, median (IQR) | 11 (9–12) |
| History of HCC, n (%) | 82 (40) |
| Esophageal gastric varix, absent/F1/F2 or more/history of varix rupture, n | 46/74/41/17 |
| Ascites, Grade 1/2/3, n | 72/110/24 |
| Encephalopathy, Grade 1/2/3, n | 173/33/0 |
| AFP, median (IQR), ng/mL | 8.6 (4.4–20.3) |

## Efficacy

Overall, 91.3% of participants (188/206) achieved SVR in the ITT analysis. Seven participants experienced virologic failure, 2 had missing HCV RNA data, 4 were

LTFU, and 5 died prior to SVR confirmation. The proportion of participants who changed to CP Class A increased at Week 24 after EOT, followed by a gradual decline through Year 5 after EOT (Figure 1).

**Figure 1. Changes in the Proportion of Participants With CP Class A Through Year 5 After EOT (Tahata et al)[3]**




Over the 43.7-month period following SOF/VEL initiation, 43 participants died, and 4 underwent liver transplantation. Causes of death included liver failure (n=19), non–liver-related conditions (n=13), HCC (n=10), and variceal rupture (n=1). The 1-year, 3-year, and 5-year liver transplant–free survival rates were 94%, 82.9%, and 69%, respectively. Liver transplant–free survival rates by virologic outcomes and CP class at 12 weeks after the EOT are presented in Table 8.

**Table 8. Liver Transplant-Free Survival Rates by Virologic Outcomes and CP Class at 12 Weeks After the EOT (Tahata et al)[3]**

| Virologic Outcomes and CP Class at 12 Weeks After EOT, n/N or % | Events[a] | Year 1 | Year 3 |
|---|---|---|---|
| Virologic failure | 3/7 | 100 | 50 |
| SVR | 38/188 | 96.2 | 86.3 |
| CP Class A[b] | 6/76 | 100 | 91.9 |
| CP Class B[c] | 25/97 | 95.8 | 86.4 |
| CP Class C | 10/18 | 81.6 | 46 |

[a]Died or underwent liver transplantation.
[b]*P*=0.012 vs CP Class B; *P*<0.001 vs CP Class C.
[c]*P*<0.001 vs CP Class C.

Factors associated with liver transplant–free survival were analyzed, excluding 11 participants with non-evaluable SVR data. In the multivariate analysis, virologic response and CP Class B and C at 12 weeks after the EOT were identified as significant factors (*P*=0.04, *P*=0.015, and *P*<0.001, respectively).

# Japanese Red Cross Liver Study Group[4]

## Study design and demographics

A prospective study conducted by the Japanese Red Cross Liver Study Group assessed outcomes of participants with HCV who received SOF/VEL between March 2019 and March 2025 (N=259). The primary outcome was SVR24, defined as undetectable HCV RNA at 24 weeks after EOT, and participants with decompensated cirrhosis who achieved SVR were followed to assess changes in hepatic function. Of the 134 participants with decompensated cirrhosis who had 24 weeks of follow-up after EOT, most (55%) were male; the median (range) age was 70 (39–93) years; 88% had a CP score >7; 37% had a history of HCC; and 69%, 28%, 0%, and 3% had GTs 1, 2, 3, and indeterminate, respectively.

## Efficacy

The SVR24 rate of participants with decompensated cirrhosis was 96%; in the ITT analysis, which included participants with decompensated cirrhosis who were LTFU, the SVR24 rate was 86% (128/149). In participants with decompensated cirrhosis who achieved SVR, the median modified ALBI score changed from -1.6 at baseline to -1.9 at EOT, -2 at SVR12/24, -2.1 at 1 year post-treatment, and -2.2 at 2 years post-treatment ($P$<0.001 at each time point compared with baseline). The proportion of participants with a modified ALBI grade of 1 or 2a increased from 5.2% at baseline to 23% at EOT, 31% at SVR12, 35% at SVR24, 37% at 1-year post-treatment, and 44% at 2 years post-treatment ($P$<0.001 compared with baseline).

Safety data for the subset of participants with decompensated cirrhosis were not presented. In the overall population, treatment was discontinued in 4 participants (due to deterioration of renal function, n=2; cerebral embolism, n=1; aspiration pneumonia, n=1) and temporarily interrupted in 2 participants (due to enteritis and heart failure, n=1 each).

# Phase 3, Open-Label, Prospective Study of SOF/VEL ± RBV in Japanese Participants With Decompensated Cirrhosis[5]

## Study design and demographics

A prospective, phase 3, multicenter, open-label study evaluated the efficacy (SVR12) and safety of SOF/VEL ± RBV in Japanese participants with chronic HCV infection and quantifiable HCV RNA at screening. Participants were randomly assigned to receive 12 weeks of SOF/VEL (n=51) or SOF/VEL + RBV (n=51) and were stratified by GT (GT 1 vs non-GT 1) and CPT class at screening (CPT Class B vs Class C). At baseline, 77% of participants were CPT Class B (score: 7–9), 20% were CPT Class C (score: 10–12), and 3% were CPT Class A (score: 6). Of the 44 who were TE, all except 1 participant had been treated with an IFN ± RBV, and the remaining participant was treated with simeprevir + pegylated IFNα 2a + RBV for 23 weeks. Of the 100 participants included in the resistance analysis, 41 had baseline NS5A RASs, and none had NS5B RASs.

**Table 9. Baseline Demographics and Disease Characteristics (Takehara et al)[5]**

| Key Demographics and Characteristics | SOF/VEL (n=51) | SOF/VEL + RBV (n=51) |
|---|---|---|
| GT 1/1a/1b, n (%) | 41 (80)/1 (2)/40 (78) | 39 (76)/0/39 (76) |
| GT 2/2a/2a-c/2b/no confirmed subtype, n (%) | 9 (18)/0/2 (4)/2 (4)/5 (10) | 11 (22)/2 (4)[a]/1 (2)/4 (8)/5 (10) |
| GT 3b, n (%) | 1 (2) | 0 |
| HCV RNA, mean (range), $\log_{10}$ IU/mL | 5.7 (3.7–7.1) | 5.8 (4.2–7) |
| TN, n (%) | 27 (53) | 31 (61) |
| *IL28B* CC GT, n (%) | 33 (65) | 37 (73) |
| MELD score ≤15, n (%) | 46 (90) | 48 (94) |
| Ascites, none/mild or moderate/severe, n (%) | 19 (37)/32 (63)/0 | 16 (31)/33 (65)/2 (4) |
| Encephalopathy, none/medication controlled, n (%) | 23 (45)/28 (55) | 22 (43)/29 (57) |

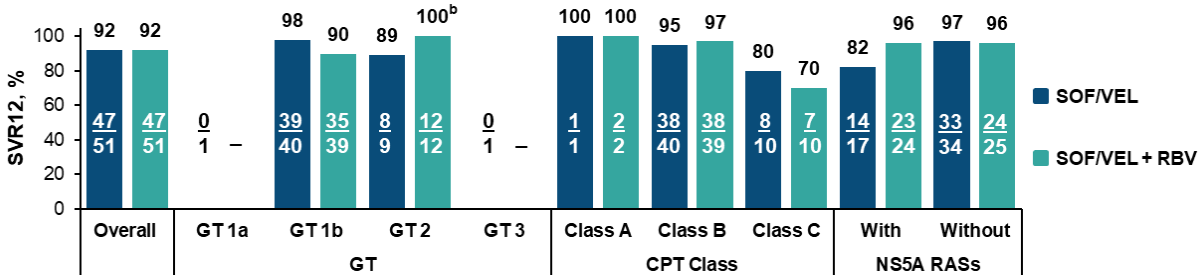
[a]One participant who initially had a missing GT was later determined to have GT 2a.

## Efficacy

Overall, 100% of participants had undetectable HCV RNA at EOT, and 92% of participants in each group achieved SVR12 (95% CI: 81–98%; Figure 2). Six participants had a virologic relapse, including 4 participants in the SOF/VEL group. Of the 4 participants in the

SOF/VEL + RBV group who did not achieve SVR12, 2 had a virologic relapse, and 2 discontinued treatment prematurely and subsequently died. Of the participants with GT 1 who were treated with SOF/VEL, 2 participants relapsed (1 with and 1 without baseline NS5A RASs). Similarly, of those with GT 1 who were treated with SOF/VEL + RBV, 2 participants relapsed (1 with and 1 without baseline NS5A RASs). Four of the 6 participants who experienced virologic relapse had treatment-emergent NS5A RASs; no treatment-emergent NS5B RASs were observed.

**Figure 2. SVR12 Rates Overall, by GT, by CPT Class, and by Baseline NS5A Status (Takehara et al)[5a]**




[a]SVR12 data were available for 100 participants who had NS5A RAS data at baseline.
[b]One participant who initially had a missing GT was later determined to have GT 2a.

From baseline to the SVR12 checkpoint among participants who achieved SVR12, 26% of participants (24/91) had an improvement in CPT class, and 2% of participants (2/91) had a worsening in CPT class (Table 10). The improvements in CPT scores were influenced by improvements in albumin levels, as 79% of participants who had increases in CPT scores also had improvements in albumin levels. MELD scores increased in 27% of participants (25/94) and worsened in 15% (14/94).

**Table 10. Changes in CPT Class From Baseline to Post-Treatment Week 12 (Takehara et al)[5]**

| Post-Treatment Week 12 CPT Class, n (%) | Baseline CPT Class (n=94) | | |
|---|---|---|---|
| | CPT Class A (n=3) | CPT Class B (n=76) | CPT Class C (n=15) |
| CPT Class A (5–6) | 3 (100) | 19 (25) | 0 |
| CPT Class B (7–9) | 0 | 55 (72) | 5 (33) |
| CPT Class C (10–15) | 0 | 2 (3) | 10 (67) |

## Safety

Most AEs were mild to moderate in severity (Table 11). No clinically significant trends in rates of AEs were observed by either age group or CPT class. After treatment completion, 3 participants developed HCC that was considered unrelated to study treatment; none of the participants with a history of HCC experienced a recurrence of HCC. The observed laboratory abnormalities were consistent with those generally observed in participants with decompensated liver disease.

**Table 11. AEs and Laboratory Abnormalities by Treatment Group (Takehara et al)[5]**

| Safety Outcomes, n (%) | SOF/VEL (n=51) | SOF/VEL + RBV (n=51) |
|---|---|---|
| Any AE | 35 (69) | 44 (86) |
| Nasopharyngitis | 7 (14) | 3 (6) |
| Anemia | 0 | 20 (39) |
| Diarrhea | 0 | 7 (14) |

| Safety Outcomes, n (%) | | SOF/VEL (n=51) | SOF/VEL + RBV (n=51) |
|---|---|---|---|
| Grade ≥3 AEs | | 2 (4) | 5 (10) |
| SAEs[a] | | 4 (8) | 7 (14) |
| AEs that led to DC of SOF/VEL | | 0 | 2 (4) |
| AEs that led to DC of RBV | | N/A | 9 (18) |
| AEs that led to alteration in RBV therapy | | N/A | 18 (35) |
| Deaths | | 0 | 3 (6)[b] |
| Grade ≥3 laboratory abnormalities | Total bilirubin >2.5 × ULN | 6 (12) | 12 (24) |
| | Hyperglycemia >250–500 mg/dL | 5 (10) | 9 (18) |
| | Hgb <10 g/dL | 2 (4) | 7 (14) |
| | Platelets 25,000–50,000/mm$^3$ | 1 (2) | 6 (12) |
| | Lymphocytes <500/mm$^3$ | 0 | 5 (10) |

Abbreviation: ULN=upper limit of normal.
[a]SAEs that occurred in >1 participant included hepatic encephalopathy (SOF/VEL, n=1; SOF/VEL + RBV, n=2) and femur fracture (SOF/VEL + RBV, n=2).
[b]All 3 participants had CPT Class C at baseline; all deaths occurred after the completion of treatment and were due to the progression of end-stage liver disease.
Note: Laboratory abnormalities had to increase from baseline by ≥1 toxicity grade. Safety data through 30 days after the last dose of study drug.

## Five-year follow-up[6]

Of the 102 participants enrolled in the original study, 94 participants with decompensated cirrhosis (CP Class B or C at screening) were prospectively followed up to 5 years after EOT to assess long-term changes in CP class, prognosis, and associated factors. At baseline, the median age was 67 years, 41% were male, 7% had a history of HCC, and 12%, 77%, and 10% had CP Class A, B, and C, respectively. The SVR rate was 93.6% (88/94); 2 of the 6 participants with virologic failure were retreated and achieved SVR.

At 4 years, the liver transplant–free survival rates were 91.5% in participants with SVR and 33.3% in participants with virologic failure ($P$<0.001). In participants with CP Class A, B, or C at 12 weeks after EOT, the liver transplant–free survival rates at 4 years were 96%, 86.4%, and 41.7%, respectively (CP Class A vs B, $P$=0.357; Class A vs C, $P$<0.001; Class B vs C, $P$=0.012). A univariate analysis found a significant association between the following factors at 12 weeks after EOT and liver transplant–free survival: ALT level, GGT level, total bilirubin level, albumin level, virologic response, and CP class (each, $P$≤0.008). In multivariate analyses, virologic response ($P$=0.002) and CP Class C 12 weeks after EOT ($P$=0.001) were significantly associated with liver transplant–free survival.

The proportion of patients with CP Class A increased significantly ($P$<0.001) from baseline to 5 years in both the overall population (12% vs 40%) and among those who achieved SVR (13% vs 41%). The proportion of participants with CP Class A did not increase among those with virologic failure.

From EOT through 4.8 years, 19 participants died due to liver failure (n=7), HCC (n=6), unknown causes (n=3), spontaneous bacterial peritonitis, necrotizing fasciitis, and cerebral hemorrhage (each, n=1), and 1 participant underwent liver transplantation. HCC recurrence was reported in 4 of the 7 participants with a history of HCC within a median of 1 year after EOT, with a cumulative HCC recurrence rate of 57.1% at 3 years. Among the 86 participants without a history of HCC, HCC occurred in 33 participants at a median of 3.2 years, with a cumulative incidence of 31.8% at 3 years. The cumulative 3-year incidence of HCC was 31.6% in participants who achieved SVR and 33.3% in those with virologic failure ($P$=0.682).

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3. Tahata Y, Hikita H, Mochida S, et al. Long-term prognosis and changes in liver function after direct-acting antiviral treatment in decompensated cirrhotic patients with hepatitis C virus [Poster THU-239]. Paper presented at: European Association for the Study of the Liver Congress; 7-10 May, 2025; Amsterdam, the Netherlads.
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6. Tahata Y, Hikita H, Takaki A, et al. Long-term changes in hepatic reserve and prognosis after direct-acting antiviral treatment in patients with hepatitis C virus-related decompensated cirrhosis: a five-year follow-up study of a Japanese phase 3 trial. *J Gastroenterol.* 2025.
7. O'Leary J, Brown RS, Reddy KR, et al. Baseline Clinical and Laboratory Parameters Associated With Clinical Benefits of Successful HCV Treatment with Sofosbuvir/Velpatasvir in Decompensated Cirrhotic Patients [Poster SAT-169]. Paper presented at: European Association for the Study of the Liver (EASL); 13-17 April, 2016; Barcelona, Spain.


# Abbreviations

AE=adverse event
AFP=α-fetoprotein
ALBI=albumin-bilirubin grading system
CP=Child-Pugh
CPT=Child-Pugh-Turcotte
DC=discontinuation
EOT=end of treatment
GT=genotype
HCC=hepatocellular carcinoma
IFN(α)=interferon (alpha)
LTFU=lost to follow-up
MELD=Model for End-Stage Liver Disease
RAS=resistance-associated substitution
RAV=resistance-associated variant
RBV=ribavirin
SAE=serious adverse event
SOF=sofosbuvir
SVR=sustained virologic response
SVR12/24=sustained virologic response 12/24 weeks after end of treatment
TE=treatment-experienced
TN=treatment-naive
VEL=velpatasvir

# Product Label

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

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