Press release

For media and investors only

Issued: 5 November 2026, Philadelphia PA

GSK showcases progress in liver portfolio at AASLD 2026

- *Hibsago* (bepirovirsen) treatment for patients with chronic hepatitis B (CHB) reinforced by new long-term dataset
- Further *Hibsago* research on surface antigen (HBsAg) reduction, retreatment modeling and functional cure predictors to inform future treatment strategies
- Latest efimosfermin phase II data highlight the potential for fibrosis improvement in patients with advanced metabolic dysfunction-associated steatohepatitis (MASH) with compensated cirrhosis (F4)
- 21 abstracts reflect significant progress across CHB, MASH and alcohol-related liver disease (ALD) which account for the majority of liver-related deaths[1-4]

GSK plc (LSE/NYSE: GSK) will present new data from its advancing liver portfolio at the American Association for the Study of Liver Diseases (AASLD) The Liver Meeting® 2026, taking place in Denver, Colorado (5-9 November). These findings demonstrate significant progress across chronic hepatitis B (CHB), metabolic dysfunction-associated steatohepatitis (MASH) and alcohol-related liver disease (ALD), reinforcing GSK's pipeline to improve patient outcomes across the spectrum of chronic liver disease.

Advancing *Hibsago* (bepirovirsen) as a foundational treatment for CHB

Following the recent US Food and Drug Administration (FDA) approval of *Hibsago,*[5] a comprehensive long-term dataset at AASLD reinforces its benefit in CHB patients.

- B-Well phase III results will show functional cure rates 48 weeks after all chronic hepatitis B virus (HBV) treatment is stopped in virologically suppressed CHB patients (Oral presentation #0266).
- Data will be presented on surface antigen (HBsAg) reduction in participants who did not achieve functional cure a year after therapy was stopped, informing GSK's continued research into the clinical significance of HBsAg reductions in patient outcomes (Poster presentation #4151).
- Additional B-Well analysis will be shared on HBsAg levels at week 12 and treatment completion as predictors of likelihood of functional cure, supporting identification of patients for treatment response (Oral presentation #0260).
- Predictive modeling will be presented on bepirovirsen retreatment and likelihood of functional cure in patients who relapse after an initial treatment course (Poster presentation #4163).
- Retrospective cohort analysis will be shared on HBsAg loss and the reduction in long-term risk of severe health outcomes, including cirrhosis and hepatocellular carcinoma, in CHB patients in China (Poster presentation #4045).

Investigating efimosfermin for fibrosis improvement in MASH

Latest data further support the potential of efimosfermin in patients with MASH, particularly those with advanced disease or at high-risk of disease progression who have limited or no approved treatment options.

- Phase II results will be presented on non-invasive markers of liver fibrosis across MASH patients with moderate to advanced fibrosis or compensated cirrhosis, supporting continued evaluation in phase III trials (Poster presentation #2299).
- Phase II analysis will be shared on the association between liver fat reduction and fibrosis in F2-F3 patients, reinforcing efimosfermin's potential impact on key drivers of MASH progression (Poster presentation #5025

Kaivan Khavandi, SVP, R&D Head Respiratory, Immunology & Inflammation (RI&I), and Head of Translational & Development Sciences, GSK, said: **"**With liver diseases like CHB, MASH, and ALD causing around 2 million deaths globally each year, the urgent need for new treatments is clear. At AASLD, we're sharing

new data from our accelerating liver portfolio, demonstrating our ambition to drive new research and deliver transformative solutions for patients who currently face limited therapeutic options."

Despite existing therapies, liver diseases represent a significant global disease burden, accounting for 1 in 25 deaths worldwide,[6] with CHB, MASH and ALD comprising the majority of liver-related deaths.[1-4] Existing treatments for CHB can suppress the virus but many patients often require lifelong therapy.[7] Treatments currently approved under accelerated or conditional pathways for MASH are indicated for moderate to advanced liver fibrosis; there are no approved treatment options for more advanced forms of MASH.[8] Beyond medications that support alcohol abstinence, there are currently no approved medicines that directly address ALD.[9-10]

Full list of GSK presentations at AASLD 2026

Abstract name	Presenter	Presentation details
bepirovirsen		
Rates of functional cure in participants with chronic hepatitis B virus (HBV) infection treated with bepirovirsen: B-Well Phase 3 trials end of study results	Man-Fung Yuen	Oral presentation #0266
Bepirovirsen B-Well Phase 3 trials: Predicting functional cure rates based upon hepatitis B surface antigen at Week 12	Pietro Lampertico	Oral presentation #0260
Hepatitis B surface antigen dynamics in participants treated with bepirovirsen who did not achieve functional cure: B-Well Phase 3 trials	Norah Terrault	Poster presentation #4151
Baseline Predictors of Functional Cure in Chronic Hepatitis B Patients Treated with Bepirovirsen: B-Well Phase 3 Trials	Maria Buti	Poster presentation #4150
Analysis of hepatitis B virus (HBV) genotype association to bepirovirsen treatment response in patients with chronic HBV infection: Phase 3 B-Well 1 and 2 studies	Jerome Bouquet	Poster presentation #4160
Serum biomarkers associated with and mediating therapeutic alanine aminotransferase (ALT) increases during bepirovirsen treatment in B-Well 1 and B-Well 2	Jenn Singh	Poster presentation #4153
Frequency of baseline bepirovirsen binding site nucleotide polymorphisms and impact on treatment response to bepirovirsen (Phase 3 B-Well 1 and 2 studies)	Christopher Sampson	Poster presentation #4152
Predicting the effects of bepirovirsen dosing in patients who relapse after an initial course of treatment	Qianwen Wang	Poster presentation #4163
Up to 15 months durability of functional cure in response to bepirovirsen therapy in B-Well responders enrolled into B-Sure	Edward Gane	Poster presentation #4140
Quality of life improvements observed in B-Clear complete responders following bepirovirsen treatment: Off-treatment follow-up in the B-Sure study	Shannon Keith	Poster presentation #4162
Patient and physician perspectives on unmet needs in chronic hepatitis B virus (HBV) management in China	Lu Zhang	Poster presentation #4074
Low HBsAg Levels and HBsAg Loss Are Associated with Reduced Risk of Long-Term Adverse Clinical Outcomes in a Large Cohort of Chronic Hepatitis B Individuals in China	Myriam Drysdale	Poster presentation #4045
New Clinical Care Pathway in Era of HBV Functional Cure: A US Multidisciplinary Expert Consensus	Joseph Ahn	Poster presentation #4144
Predicting functional cure response with bepirovirsen in patients with baseline HBsAg levels <100 IU/mL	Dominic G. Whittaker	Poster presentation #5024
efimosfermin		

Once-monthly efimosfermin alfa improves non-invasive markers of fibrosis with an acceptable safety profile in participants with metabolic dysfunction-associated steatohepatitis with moderate to advanced fibrosis or compensated cirrhosis (F2–4): Results up to 52 weeks from an open-label, Phase 2 study	Mazen Noureddin	Poster presentation #2299
How Are Patients With Metabolic Dysfunction-Associated Steatohepatitis (MASH) Managed in the Real World? A Descriptive Analysis of Diagnostic Testing, Treatment, and Monitoring in US Administrative Claims	Christopher F. Bell	Poster presentation #2456
ZENITH-1 and ZENITH-2: Phase 3, randomized, double-blind, placebo-controlled trials in progress of efimosfermin alfa in participants with metabolic dysfunction-associated steatohepatitis (MASH) and F2/F3 fibrosis	Mazen Noureddin	Poster presentation #3576
NEBULA-1 and NEBULA-2: Phase 3, randomized, double-blind, placebo-controlled trials in progress of efimosfermin alfa in participants with metabolic dysfunction-associated steatohepatitis (MASH) and compensated cirrhosis	Mazen Noureddin	Poster presentation #3558
Liver fat reduction following once-monthly efimosfermin alfa treatment is associated with histological fibrosis improvement in participants with MASH and stage F2/F3 fibrosis (MRI PDFF)	Rohit Loomba	Poster presentation #5025
Above Asset		
Hepatic Adaptation to Experimental Drugs in Clinical Trials: Results of the IQ DILI Initiative Survey of Multi-Stakeholder Experts: Impact on Drug Development and Implications for DILI Detection and Evaluation	Melissa Palmer	Poster presentation #3334
How patients with alcohol-related liver disease feel and function: Toward a literature-informed patient-reported outcome	Yekaterina Poloz	Poster presentation #3137

About chronic hepatitis B
Hepatitis B is a viral infection that can cause both acute and chronic liver disease. Chronic hepatitis B (CHB) occurs when the immune system is unable to clear the virus, resulting in long-lasting infection that affects over 240 million people worldwide. CHB causes approximately 1.1 million deaths each year[11] and is the leading cause of liver cancer cases globally.[12] Currently, many patients often require lifelong antiviral therapy for viral suppression, making functional cure a critical goal in disease management.

About MASH and ALD
Metabolic-dysfunction associated steatohepatitis (MASH) and alcohol-related liver disease (ALD) are chronic, progressive liver diseases where fibrosis (scarring) and inflammation can lead to major adverse liver outcomes (MALOs) including cirrhosis, liver cancer and liver failure. In MASH, the accumulation of fat and associated fibrosis is generally in response to metabolic dysregulation while in ALD, it is primarily alcohol related. MASH and ALD are each estimated to affect up to 5% of the global adult population and are two of the leading causes of liver transplants.[13-17]

About Hibsago (bepirovirsen)
Bepirovirsen is an antisense oligonucleotide (ASO) designed to recognize and inhibit the production of the genetic components (i.e. RNA) of the hepatitis B virus that can lead to chronic disease, potentially allowing a person's immune system to regain control. Bepirovirsen reduces the production of RNA and viral proteins associated with HBV, suppresses the level of hepatitis B surface antigen (HBsAg) in the blood, and may stimulate the immune system to increase the chances of a durable and sustained response.

GSK licensed bepirovirsen from Ionis and collaborated with them on its development. Bepirovirsen has been recognized by global regulatory authorities for its innovation and potential to address significant unmet need in

hepatitis B, with Breakthrough Designation and priority review from the US FDA, Breakthrough Therapy and Priority Review designation in China and SENKU designation in Japan. Bepirovirsen is approved in Japan and the US.

INDICATION
HIBSAGO is indicated in combination with nucleos(t)ide analog reverse transcriptase inhibitor (NrtI) therapy to treat chronic hepatitis B virus (HBV) infection in adults without cirrhosis who have been on NrtI therapy for ≥6 months with suppressed HBV DNA and who have HBV surface antigen (HBsAg) levels ≤3,000 IU/mL as determined by an FDA-authorized test.

WARNING: ON-TREATMENT HEPATIC FLARES AND THROMBOCYTOPENIA
On-Treatment Hepatic Flares
HIBSAGO may cause on-treatment hepatic flares with increases in alanine aminotransferase (ALT) of >20-40x upper limit of normal (ULN) associated with decline in HBsAg. Some cases were associated with hyperbilirubinemia. Most occurred within the first 12 weeks and resolved when treatment was interrupted or discontinued. Do not initiate HIBSAGO in patients with evidence of acute hepatic impairment or cirrhosis. ALT and total bilirubin must be measured prior to treatment and frequently monitored during treatment. Treatment interruption, followed by dose reduction or discontinuation, may be necessary based on liver laboratory results.

Thrombocytopenia
HIBSAGO may cause reductions in platelet counts that may result in sudden and unpredictable thrombocytopenia, which can be life-threatening. Obtain platelet count prior to treatment; HIBSAGO is contraindicated in patients with platelet count <100 x 10^9/L. Monitor platelet counts weekly during treatment and more frequently if platelet count falls <75 x 10^9/L. Do not re-dose unless platelet count returns to ≥100 x 10^9/L. Permanently discontinue HIBSAGO if platelet count falls <50 x 10^9/L.

CONTRAINDICATIONS
HIBSAGO is contraindicated in patients:
• with acute hepatic impairment or cirrhosis who have mild, moderate, or severe hepatic impairment (Child-Pugh Class A, B, or C).
• with a platelet count <100 x 10^9/L prior to initiation of treatment.

WARNINGS AND PRECAUTIONS
On-Treatment Hepatic Flares
Increases in ALT ≥3x ULN to 69x ULN were observed during treatment with HIBSAGO, most within the first 12 weeks. Isolated cases of ALT increases associated with an increase in total bilirubin resolved when treatment was permanently discontinued.

Thrombocytopenia
In addition to sudden and unpredictable thrombocytopenia, a gradual, dose-dependent decline in platelet count was observed in most patients that continued to worsen while on HIBSAGO. If a patient develops signs or symptoms of thrombocytopenia, pause HIBSAGO and obtain a platelet count as soon as possible. Instruct patients to report any symptoms of thrombocytopenia to their healthcare provider immediately. Consider the risks and benefits of treatment with HIBSAGO when treating patients who may be particularly susceptible to platelet count decreases.

Worsening Renal Function
Mean estimated glomerular filtration rate (eGFR) declined within 4 weeks of initiation and continued to decline during treatment. These changes were generally reversible after treatment discontinuation. Obtain eGFR and urine albumin-creatinine ratio (uACR) in all patients prior to initiation of treatment. Monitor eGFR frequently during and after treatment; treatment interruption or discontinuation may be necessary. Although no cases of glomerulonephritis were observed with HIBSAGO, glomerulonephritis has been observed with other antisense oligonucleotides; if suspected or confirmed, permanently discontinue HIBSAGO.

Inflammatory and Immune Effects
Influenza-like symptoms, laboratory test changes (eg, decreases in complement factors C3 and C4), purpuric leg rashes, cutaneous leukocytoclastic vasculitis, dermatitis, pruritus/erythematous plaque, and polyarthralgia were observed. In addition, serious inflammatory and immune adverse reactions were reported, including immune

thrombocytopenia, autoimmune hemolytic anemia, systemic inflammatory response syndrome, and a cryoglobulinemic flare.

Consider the risks and benefits of HIBSAGO in patients who may be susceptible to inflammatory and immune effects, including solid organ transplant recipients, patients with autoimmune hepatitis, patients with a history of or conditions associated with vasculitis, or extrahepatic disorders related to HBV immune-mediated effects. If signs and symptoms suggestive of vasculitis/immune-associated events occur, and no alternative cause is identified, permanently discontinue treatment with HIBSAGO.

Serious Adverse Reaction with Improper Administration
Improper HIBSAGO administration, including not rotating injection sites, has been associated with the serious adverse reaction of omental infarction in one participant. Ensure injection sites are rotated from one week to the next and that the two injections making up each dose are separated by at least 2 inches (5 cm). Never inject into areas where the skin is tender, bruised, red, or hard.

Post-Treatment Exacerbations of HBV Infection After NrtI Discontinuation
Severe acute exacerbations of HBV infection may occur after discontinuation of anti-HBV therapy. If NrtI therapy is discontinued after completing HIBSAGO treatment, frequent monitoring is recommended for at least one year to detect acute exacerbations of HBV infection and the possible need for resumption of HBV treatment.

Risk of human immunodeficiency virus-1 (HIV-1) Virologic Failure in Patients with HBV/HIV-1 Coinfection Who Discontinue NrtI Therapy
Patients who are taking NrtI for HIV-1 and HBV coinfection must not stop taking their NrtI unless an HIV healthcare provider has directed them to do so.

Risk of Hepatitis Flare in Patients with HBV/Hepatitis D Virus (HDV) Coinfection
HIBSAGO is not recommended in patients with HDV coinfection due to the potential for an exacerbated risk of clinically significant ALT increases.

Unknown Effect on Preventing Transmission of HBV
There are no data on the effect of HIBSAGO in preventing transmission of HBV. Patients must be advised to use appropriate precautions to prevent transmission of HBV.

ADVERSE REACTIONS
The most common adverse reactions (≥10%), excluding laboratory abnormalities, reported with HIBSAGO included injection site reactions, pyrexia, fatigue, and headache.

Selected laboratory abnormalities worsened from baseline, including but not limited to, increases in ALT and total bilirubin, decreases in platelets and eGFR.

DRUG INTERACTIONS
- Monitor patients for signs of bleeding and thrombocytopenia if HIBSAGO is taken concomitantly with anticoagulants, antiplatelets, or drugs that decrease platelet count.
- Monitor patients for signs of worsening renal function during concomitant administration of HIBSAGO with drugs that may impair renal function.
- Concomitant administration of HIBSAGO and peginterferon alfa-2a is not recommended due to the potential for an exacerbated risk of increased ALT, decreased platelet count, and immune-mediated effects.

USE IN SPECIFIC POPULATIONS
Pregnancy and Lactation
There are insufficient data on HIBSAGO use in pregnancy or during breastfeeding. Consider the benefits of breastfeeding together with the mother's clinical need for HIBSAGO.

Pediatric Use
HIBSAGO's safety and effectiveness have not been established in patients under 18 years of age.

Renal Impairment
HIBSAGO can cause worsening renal function. No dosage adjustment is needed for mild renal impairment. Data are insufficient for moderate or severe renal impairment or end-stage renal disease (ESRD).

Please see full Prescribing Information, including Boxed Warning, for HIBSAGO

Please see accompanying US Prescribing Information here.

About efimosfermin
Efimosfermin is an investigational, once-monthly subcutaneous injection of a long-acting variant of FGF21 that is designed to regulate multiple metabolic pathways to decrease liver fat, ameliorate inflammation, and reverse liver fibrosis in MASH and ALD patients. Efimosfermin is advancing through clinical development across multiple potential indications, including in the ZENITH phase III trials for MASH with moderate to advanced fibrosis (F2-3), the NEBULA phase III trials for MASH with compensated cirrhosis (F4), and the ALLSTAR phase II trial for ALD.

Efimosfermin has received Breakthrough Therapy Designations from the US FDA and China's CDE, as well as Priority Medicines (PRIME) Designation from the European Medicines Agency (EMA), for the treatment of MASH.

About GSK in hepatology
GSK is extending its expertise in hepatology to develop a next wave of innovation for the millions of people affected by chronic and life-threatening fibro-inflammatory liver conditions. GSK has a growing hepatology pipeline, harnessed by the science of the immune system and advanced technologies, with programs focused on chronic hepatitis B (CHB), metabolic dysfunction-associated steatohepatitis (MASH) and alcohol-related liver disease (ALD).

About GSK
GSK is a global biopharma company with a purpose to unite science, technology, and talent to get ahead of disease together. Find out more at www.gsk.com.

GSK enquiries

Media:	Tim Foley	+44 (0) 20 8047 5502	(London)
	Jonny Reilly	+44 (0) 20 8047 5502	(London)
	Kathleen Quinn	+1 202 603 5003	(Washington DC)
	Alison Hunt	+1 540 742 3391	(Washington DC)
	Stephanie Caccomo	+1 202 893 2840	(Washington DC)
Investor Relations:	James Dodwell	+44 (0) 7881 269066	(London)
	Mick Readey	+44 (0) 7990 339653	(London)
	Joanna Tuplin	+44 (0) 7788 351650	(London)
	Sam Piper	+44 (0) 7824 525779	(London)
	Dan Smith	+44 (0) 7823 523885	(London)
	Jeff McLaughlin	+1 215 751 7002	(Philadelphia)
	Frannie DeFranco	+1 215 751 3126	(Philadelphia)

Cautionary statement regarding forward-looking statements
GSK cautions investors that any forward-looking statements or projections made by GSK, including those made in this announcement, are subject to risks and uncertainties that may cause actual results to differ materially from those projected. Such factors include, but are not limited to, those described in the "Risk Factors" section in GSK's Annual Report on Form 20-F for 2025, and GSK's Q3 Results for 2026.

Registered in England & Wales:
No. 3888792

Registered Office:
79 New Oxford Street
London
WC1A 1DG

References

1 Devarbhavi H, et al. Global Burden of Liver Disease: 2026 update. 2026. DOI: 10.1053/j.gastro.2026.09.014.
2 Xiao J, Wang F, Yuan Y, et al. Epidemiology of liver diseases: Global disease burden and forecasted research trends. Science China Life Sciences. 2024;68(2):541-557. DOI:10.1007/s11427-024-2722-2
3 Younossi ZM, Paik JM, Lazarus JV, et al. Projected Global Clinical, humanistic, and economic impact of metabolic dysfunction-associated steatohepatitis (MASH): The cost of inaction based on data from nine countries. Clinical Gastroenterology and Hepatology. 2026;24(4):1016-1034. DOI:10.1016/j.cgh.2025.09.002
4 Shen F, Li B, Lv H, et al. Global, regional, and national burden of chronic hepatitis B, 1990–2021, and projections to 2030: An urgent call for action to achieve hepatitis B elimination goals by 2030. Human Vaccines & Immunotherapeutics. 2026;22(1). DOI:10.1080/21645515.2026.2641857
5 GSK press release - Hibsago US approval. Available at: https://www.gsk.com/en-gb/media/press-releases/hibsago-bepirovirsen-approved-in-the-us-for-the-treatment-of-chronic-hepatitis-b-chb/.
6 Devarbhavi H, et al. J Hepatol. 2023;79(2):516–537
7 Gopalakrishna H, Ghany MG. Perspective on emerging therapies to achieve functional cure of chronic hepatitis B current Hepatology Reports. SpringerLink. February 10, 2024. Accessed October 2026.
8 Miller DM, McCauley KF, Dunham-Snary KJ. Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD): Mechanisms, Clinical Implications and Therapeutic Advances. Int J Mol Sci. 2024;25(10):5487. Available at: https://pmc.ncbi.nlm.nih.gov/articles/PMC12627968/. Last accessed: October 2026.
9 Zhang M, et al. Current Therapeutic Targets for Alcohol-Associated Liver Disease. American Journal of Pathology. 2026;196(1). DOI: 10.1016/j.ajpath.2025.06.009.
10 Queralt Herms, Jordi Gratacós-Ginès, Elisa Pose, Alcohol-related liver disease, Gastroenterology Report, Volume 13, 2025, goaf088, https://doi.org/10.1093/gastro/goaf088 .
11 WHO Global Hepatitis Report 2026. Available at: https://www.who.int/publications/i/item/9789240122383 (last accessed August 2026)
12 Rumgay H et al. Global burden of primary liver cancer in 2020 and predictions to 2040. J Hepatol. 2022;77:1598–1606. doi: 10.1016/j.jhep.2022.08.021
13 Åberg F, et al. Alcohol-associated liver disease-Global epidemiology. Hepatology. 2024;80(6):1307-1322. DOI: 10.1097/HEP.0000000000000899.
14 Niu X, et al. Global prevalence, incidence, and outcomes of alcohol related liver diseases: a systematic review and meta-analysis. BMC Public Health. 2023;23(1):859. DOI: 10.1186/s12889-023-15749-x.
15 Povsic M, et al. A Structured Literature Review of the Epidemiology and Disease Burden of Non-Alcoholic Steatohepatitis (NASH). Advances in Therapy. 2019;36(7):1574-94. DOI: 10.1007/s12325-019-00960-3.
16 Paklar N, Mijic M, Filipec-Kanizaj T. The Outcomes of Liver Transplantation in Severe Metabolic Dysfunction-Associated Steatotic Liver Disease Patients. Biomedicines. 2023 Nov 20;11(11):3096. DOI: 10.3390/biomedicines11113096.
17 Musto et al. Early Liver Transplant for Alcohol-associated Liver Disease Has Excellent Survival but Higher Rates of Harmful Alcohol Use. Clin Gastroenterol Hepatol. 2024 Aug;22(8):1646-1656.e2. DOI: 10.1016/j.cgh.2024.04.025.

...