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Biohaven's Novel FGFR3-Directed ADC with Proprietary Topoisomerase Payload, BHV-1530, Shows Clinical Activity and Confirmed Responses in FGFR3-Altered Urothelial and Head and Neck Cancers, Oral Presentation at ENA 2026

November 19, 2026

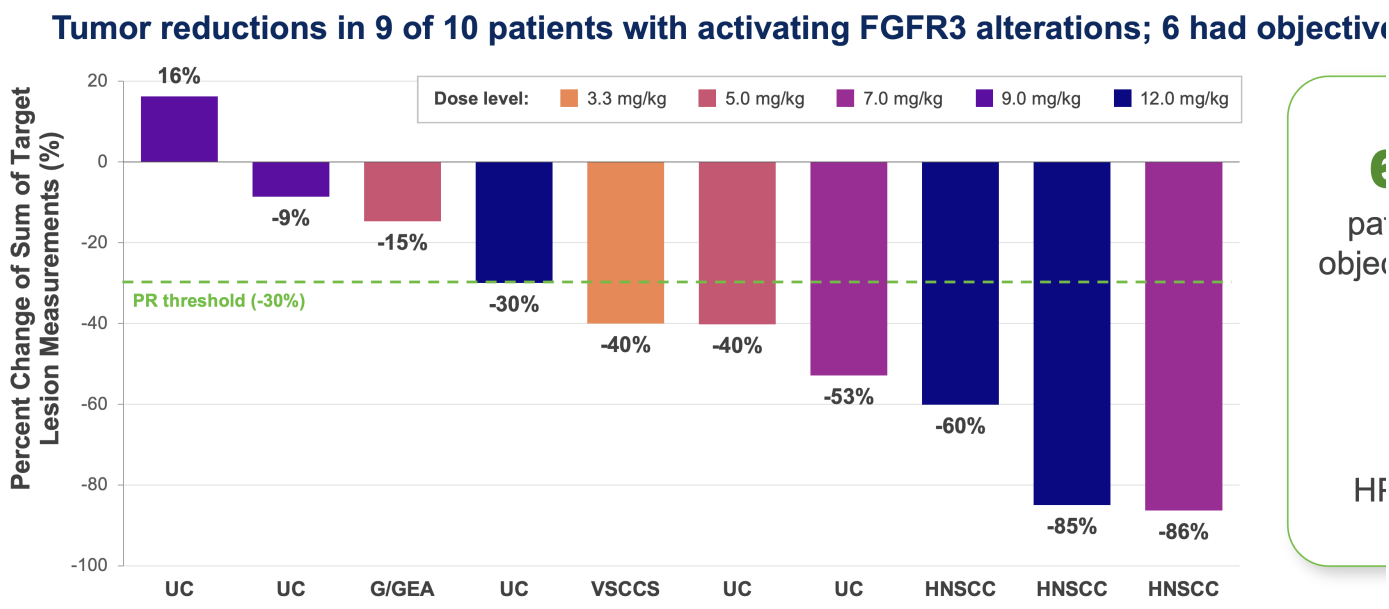
Phase 1 data presented in an oral plenary session at the 36th EORTC-NCI-AACR Symposium on Molecular Targets and Cancer Therapeutics (ENA 2026) in Barcelona, Spain, showed deep and durable responses in patients with FGFR3-altered urothelial cancer (UC) and head and neck squamous cell carcinoma (HNSCC). As of the data cutoff in the ongoing study, 6 of 10 evaluable patients with FGFR3-activating alterations treated at doses of ≥3.3 mg/kg had responded, including 3 of 9 with UC and 3 of 3 with HPV-positive HNSCC.

- *Responders to BHV-1530 in UC included patients who failed prior enfortumab vedotin plus pembrolizumab (EV+P) and oral FGFR tyrosine kinase inhibitors. All responders remained on treatment at data cutoff, some beyond 6 months.*
- *BHV-1530 was well tolerated with no FGFR class toxicities; no DLTs, treatment-related SAEs or deaths, and no ILD, no hyperphosphatemia, nail dystrophy or central serous retinopathy.*
- *Biohaven completed FDA Type C meeting in 4Q 2026 and plans to initiate a pivotal study evaluating BHV-1530 in FGFR3-altered tumors, starting with UC, expected to begin as early as 1H 2027 to support potential accelerated approval.*

NEW HAVEN, Conn., Nov. 20, 2026 /PRNewswire/ -- Biohaven Ltd. (NYSE: BHVN) ("Biohaven"), a global clinical-stage biopharmaceutical company focused on the discovery, development and commercialization of life-changing therapies to treat a broad range of rare and common diseases, today announced clinical data from the ongoing Phase 1 study of BHV-1530, its first in class potential FGFR3-directed antibody-drug conjugate (ADC) with a novel topoisomerase 1 (Topo1) payload. The data were presented today by Gerald S. Falchook, M.D., Director of South Carolina Research Institute at HCA HealthONE, in an oral presentation during Plenary Session 3 at the 36th EORTC-NCI-AACR Symposium on Molecular Targets and Cancer Therapeutics (ENA 2026) in Barcelona, Spain. As of the October 19, 2026 data cutoff, 30 patients had received BHV-1530 monotherapy every three weeks (Q3W) at doses from 1.0 to 12.0 mg/kg, and dose escalation is ongoing. Patients had received a median of two prior lines of therapy for advanced disease, and all patients with UC had progressed on EV+P.

Among 10 efficacy-evaluable patients with activating FGFR3 alterations, treated with BHV-1530 at doses of 3.3 mg/kg or higher, 9/10 had a tumor reduction and 6 achieved a partial response (PR, confirmed or unconfirmed, by investigator assessment per RECIST v1.1, Figure 1). Responses were observed in tumors with FGFR3 mutations and fusions, including in 3 of 9 patients with UC and all 3 patients with HPV-positive HNSCC. All responders remained on treatment as of the data cutoff, some for more than 6 months.

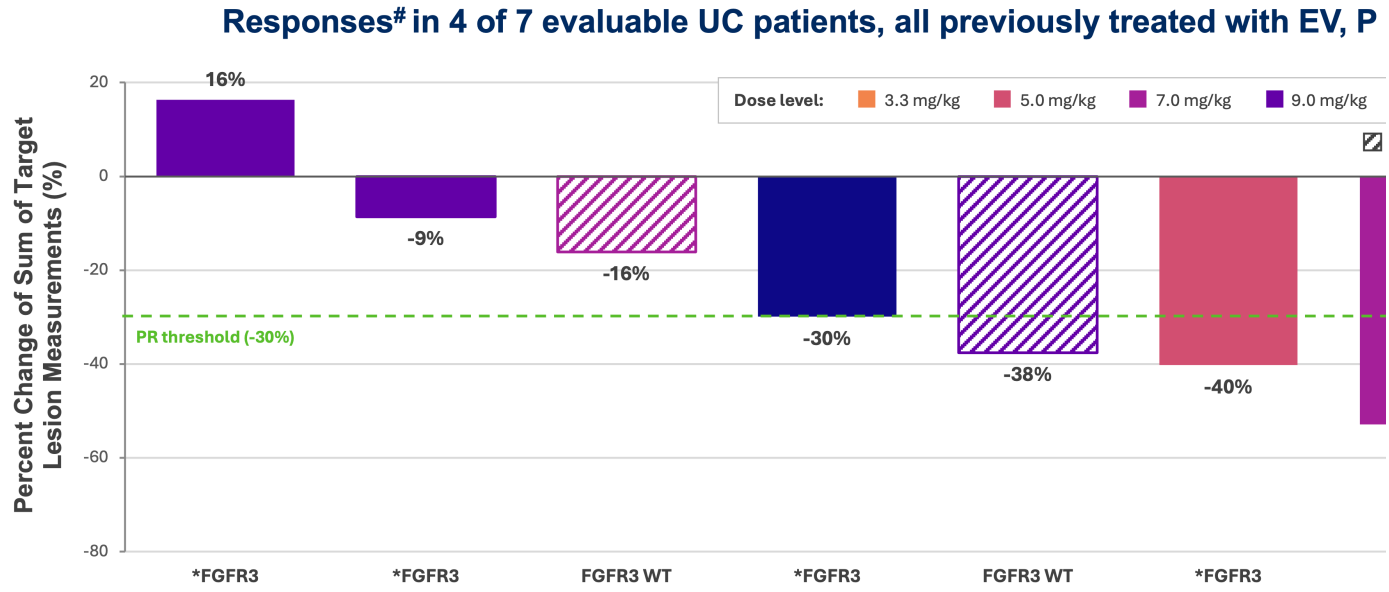
Figure 1. Efficacy in FGFR3-Altered Tumors



*Includes confirmed and unconfirmed partial responses. Efficacy-evaluable patients with an activating FGFR3 alteration treated at 3.3-12.0 mg/kg (n=10); tumor response assessed by inv
from ongoing study with live database; patients enrolled regardless of FGFR3 expression status.
HNSCC, head and neck squamous cell carcinoma; **G/GEA,** gastric/gastroesophageal adenocarcinoma; **UC,** urothelial carcinoma; **VSCCS,** verrucous squamous cell carcinoma of skin. **Cli**

Gerald S. Falchook, M.D. commented, "Many patients in this study had progressed on multiple prior therapies, including enfortumab vedotin plus pembrolizumab and FGFR inhibitors in urothelial cancer, so seeing durable responses is very encouraging." Dr. Falchook added, "We also saw deep responses in HPV-positive head and neck cancer, where options after immunotherapy are limited. Importantly, BHV-1530 has been well tolerated, with no dose-limiting toxicities and none of the hyperphosphatemia, nail or retinal toxicities that often restrict FGFR tyrosine kinase inhibitor therapy."
In UC, where all patients had progressed on prior EV+P, partial responses occurred in 3 of 9 patients with FGFR3-altered tumors, including a patient who had also received a prior FGFR inhibitor. Tumor reductions were also observed in the 2 patients with FGFR3 wild-type tumors, including one partial response pending confirmation (Figure 2).

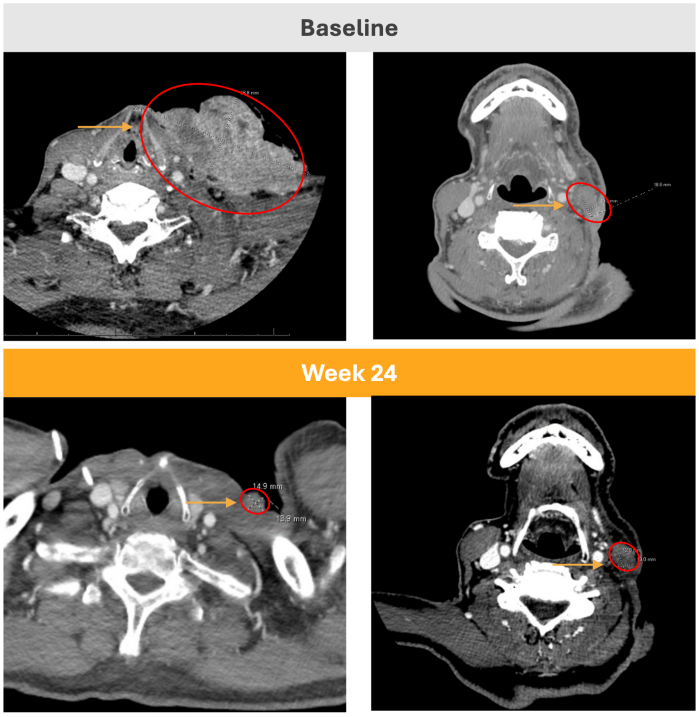
Figure 2. Efficacy in Urothelial Carcinoma



*Denotes an activating FGFR3 alteration, # Includes confirmed and unconfirmed partial responses; the wildtype response is pending confirmation. Efficacy-evaluable UC patients treated at 3.3-12.
enrolled overall, all with prior enfortumab vedotin and pembrolizumab (EV+P). Tumor response assessed by investigator per RECIST 1.1. Data from ongoing study with live database.
UC, urothelial carcinoma; **EV, P,** Enfortumab vedotin, pembrolizumab; **WT,** wild-type (no activating FGFR3 alteration). **Clinical cut-off:** 19-OCT-2026

Dr. Falchook presented the clinical case of a patient with HPV-positive metastatic HNSCC and an FGFR3 S249C mutation, who had received four prior lines of therapy including chemoradiation, a platinum doublet and immunotherapy. This patient achieved a confirmed partial response, with a 67% reduction in target lesions at Week 6 that deepened to 86% by Week 24, (Figure 3) and remains on therapy beyond 6 months with no treatment-related adverse events. This case illustrates the speed and depth of response in a highly refractory patient who failed prior standard of care.

Figure 3. Axial CT Images



BHV-1520 was well tolerated across all dose levels in 30 patients, with no dose-limiting toxicities, treatment-related serious adverse events or treatment-related deaths. The most common adverse events, nausea (43%) and fatigue (37%), were all Grade 1 or 2. Hematological toxicity was mostly low-grade and manageable. None of the toxicities that commonly limit FGFR inhibitor therapy (hyperphosphatemia, nail dyskrypty or central serous retinopathy) were observed, and no interstitial lung disease (ILD) was reported. Pharmacokinetic data showed that BHV-1520 is highly stable in circulation, with free payload exposure below 1% of ADC exposure, and a half-life of 7 to 10 days that supports QWW dosing.

Nushmia Khokhar, M.D., Chief Medical Officer of Oncology at BioHaven, stated, "Unlike FGFR tyrosine kinase inhibitors, which block kinase signaling inside the cell, BHV-1520 is an ADC designed to bind FGFR3 on the tumor cell surface, be internalized and release a potent topoisomerase I payload, independent of kinase-domain resistance mutations."

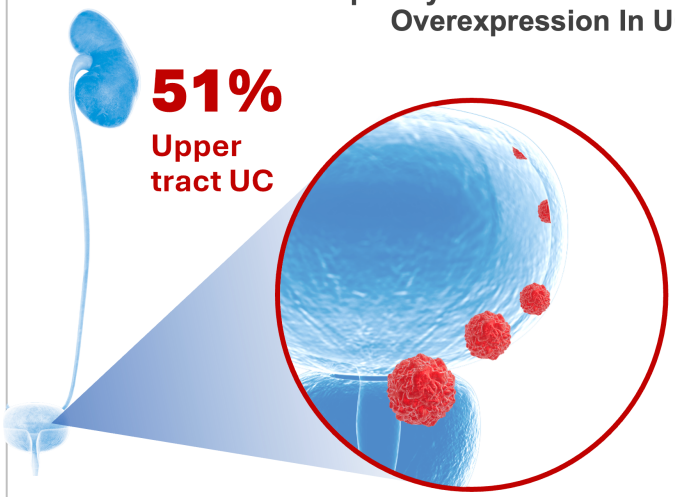
Dr. Khokhar added, "Following a recent Type C meeting with the FDA, we are excited to advance BHV-1520 into a pivotal study in FGFR3-altered tumors, beginning with urothelial cancer, as early as the first half of 2027, with the goal of supporting a potential accelerated approval."

FGFR3 is frequently altered or overexpressed in UC. Activating mutations or fusions, together with overexpression of the wild-type receptor, are found in about half of metastatic UC and in 70% to 80% of non-muscle-invasive bladder cancer, and are also seen in head and neck, lung and other solid tumors (Figure 4). Erdafitinib vedotin plus pembrolizumab is now the first-line standard of care for advanced UC, but many patients eventually progress and options after progression remain limited. The only FDA-approved FGFR-targeted therapy for advanced UC is an oral pan-FGFR tyrosine kinase inhibitor (TKI), indicated only for previously treated patients whose tumors carry a susceptible FGFR3 alteration, which leaves patients whose tumors overexpress wild-type FGFR3 without a targeted option. Pan-FGFR TKIs also inhibit FGFR1, FGFR2 and FGFR4, and their labeled class effects include hyperphosphatemia, nail disorders, stomatitis and retinopathy, which often require dose interruptions or reductions. Resistance can also emerge through mutations in the kinase domain where these drugs bind. BHV-1520 is designed to bind FGFR3 on the tumor cell surface rather than inhibit its kinase, and is being studied in patients with and without FGFR3 alterations.

Figure 4. Activating FGFR3 Alteration and Overexpression Prevalence*

FGFR3 alterations and overexpression are common in UC and other solid tum

Frequency of FGFR3 Alterations and Overexpression In UC:



51%
Upper tract UC

70–80%
Non-muscle-invasive BC

40–60%
Muscle-invasive BC

~50%
Metastatic UC

FGFR3 Alterati Overexpression Al



~5(
of HN



~4(
of NS



4%
altera other

* Activating FGFR3 Alteration includes mutations and fusions/rearrangements

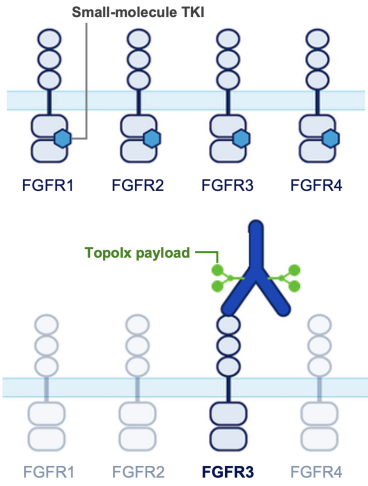
1. Müller DC, et al. Nat Commun. 2026;17:3263. **2.** Hammerman PS, et al. Cancer Discov. 2015;5(3):239-44.
3. Zhou Z, et al. Cancer Biol Med. 2021;18(2):490-501. **4.** Moes-Sosnowska et al. Front Oncol. 2022.

BHV-1520 takes a different approach to FGFR3 than approved pan-FGFR tyrosine kinase inhibitors, which block kinase signaling across the FGFR family (Figure 5). The investigational antibody-drug conjugate is designed to bind selectively to FGFR3 on the tumor cell surface and deliver its Topo1 payload inside the cell, with no detectable binding to FGFR1, FGFR2 or FGFR3 in preclinical studies.

Figure 5. BHV-1530: An FGFR3 Directed ADC Distinct From FGFR TKIs

A Different Way to Target FGFR3

BHV-1530 compared with approved pan-FGFR tyrosine kinase inhibitors (TKIs)



Approved pan-FGFR TKIs

Block FGFR kinases inside the cell; use in specific alterations only; kinase-domain mutations can limit activity

Labeled class toxicities: hyperphosphatemia, nail disorders, stomatitis and retinopathy

BHV-1530

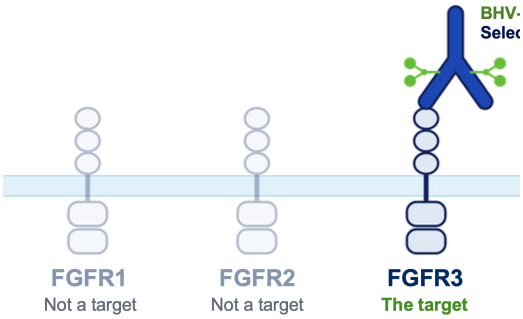
FGFR3-directed ADC

Binds surface FGFR3, blocks its signaling and delivers a Topo I inhibitor payload into the cell

Designed to avoid pan-FGFR class toxicities (FGFR3-selective*), limit free payload (stable linker) and bypass kinase-domain mutations

BHV-1530 ADC Binds FG

Selective by design, with no detectable binding to FGFR



Only FGFR3 ADC in the clinic

No other FGFR3 ADC is in clinical development.† Potential in FGFR3-altered and wild-type FGFR3-overexpressing tumors.

No oral TK

Hyperphosphate
and retinopathy

Schematic of mechanism and design; not head-to-head clinical data. Class toxicities per US prescribing information for approved pan-FGFR TKIs. BHV-1530 is investigational and has not been ap
authority; its safety and efficacy have not been established. *No detectable binding to FGFR1, FGFR2 or FGFR4 in preclinical studies. †Based on publicly available clinical trial registries as of Octo
2026 data cutoff; preliminary data from an ongoing study, not a head-to-head comparison. **ADC,** antibody-drug conjugate; **FGFR,** fibroblast growth factor receptor; **TKI,** tyrosine kinase inhibitor; **To**

About Study BHV1530-101

BHV1530-101 (NCT06874335) is a Phase 1, first-in-human, multicenter study of BHV-1530 as monotherapy and in combination with an anti-PD-1 antibody in adults with advanced solid tumors. Patients with UC, HNSCC or non-small cell lung cancer (NSCLC) were enrolled irrespective of FGFR3 alteration status, without a minimum FGFR3 expression threshold. Patients with other solid tumors were eligible if their tumors harbored an activating FGFR3 alteration.

About BHV-1530

BHV-1530 is an antibody-drug conjugate directed against FGFR3, a validated but underexploited target in urothelial cancer and other FGFR3-driven cancers and is a first-in-class potential FGFR3-directed ADC incorporating Biohaven's proprietary Topo payload. Unlike approved FGFR tyrosine kinase inhibitors, which are restricted to genomically selected patients and constrained by class-related toxicities, BHV-1530 is designed to target FGFR3 with the potential to address both FGFR3-altered and wild-type overexpressing tumors. BHV-1530 is being studied in an ongoing Phase 1 dose-escalation trial in unselected patients with advanced urothelial cancer, head and neck squamous cell carcinoma, and non-small cell lung cancer who have failed standard-of-care therapy, as well as other tumor types harboring FGFR3 genomic alterations. Biohaven has initiated combination cohorts evaluating BHV-1530 with anti-PD-1 therapy, including the combination with LIBTAYO® (cemiplimab) which is now enrolling.

About Biohaven

Biohaven Ltd. (NYSE: BHVN) is a biopharmaceutical company focused on the discovery, development, and commercialization of life-changing treatments in key therapeutic areas, including immunology, neuroscience, and oncology. Biohaven is advancing its innovative portfolio of therapeutics, leveraging its proven drug development experience and multiple proprietary drug development platforms. Biohaven's extensive clinical and preclinical programs include Kv7 ion channel modulation for epilepsy; MoDE™ and TRUE™ molecular protein degradation for immunological diseases; and myostatin inhibition for neuromuscular and metabolic diseases, including obesity. For more information, v

Forward-Looking Statements

This news release includes forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, including statements regarding the expected timing, conduct, and outcomes of Biohaven's ongoing and planned clinical trials of BHV-1530, including the combination with cemiplimab under the clinical supply agreement with Regeneron; the potential therapeutic benefits, efficacy, safety, and tolerability of BHV-1530; and the timing of planned regulatory interactions and filings. The use of certain words, including "continue," "plan," "will," "believe," "may," "expect," "anticipate," "on track," "potential," and similar expressions, is intended to identify forward-looking statements. Investors are cautioned that any forward-looking statements are not guarantees of future performance or results and involve substantial risks and uncertainties. Actual results, developments and events may differ materially from those in the forward-looking statements as a result of various factors, including the expected timing, commencement and outcomes of Biohaven's planned and ongoing clinical trials; the timing of planned interactions and filings with the FDA; the timing and outcome of expected regulatory filings; complying with applicable U.S. regulatory requirements; the potential commercialization of Biohaven's product candidates and the expected timing thereof; the potential for Biohaven's product candidates to be successful therapies; and the effectiveness and safety of Biohaven's product candidates. Additional important factors to be considered in connection with forward-looking statements are described in Biohaven's filings with the Securities and Exchange Commission, including within the sections titled "Risk Factors" and "Management's Discussion and Analysis of Financial Condition and Results of Operations." The forward-looking statements are made as of the date of this news release, and Biohaven does not undertake any obligation to update any forward-looking statements, whether as a result of new information, future events or otherwise, except as required by law.

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