# BlossomHill Therapeutics Presents First Preclinical Data from New Pan-KRAS Inhibitor Program Built on a Novel Chemical Scaffold During Mini Symposium at AACR 2026

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SAN DIEGO, April 22, 2026 (GLOBE NEWSWIRE) -- BlossomHill Therapeutics, Inc., a privately-held, clinical-stage biopharmaceutical company applying an intentional, chemistry-based approach to develop innovative small molecule medicines for the treatment of cancer, today announced new preclinical data presented at the American Association for Cancer Research (AACR) Annual Meeting, highlighting the discovery and characterization of its lead pan-KRAS inhibitor, BH-501284, along with additional updates across its pipeline.

“Despite recent advances, patients with KRAS-driven tumors continue to face limited and often short-lived treatment options. Our mini-symposium presentation at AACR introduces our differentiated pan-KRAS inhibitor, BH-501284, with pre-clinical data demonstrating the potential for this molecule to deliver broad and durable pathway inhibition across multiple KRAS-driven tumors,” said Jean Cui, Ph.D., Founder and Chief Executive Officer of BlossomHill Therapeutics. “We are particularly encouraged by the depth and consistency of activity observed across models, including sustained target engagement and tumor regression at low dose levels. Based on these results, we are advancing IND-enabling studies for BH-501284 and preparing for clinical development.”

Data presented in the AACR Mini Symposium provides pre-clinical data supporting BH-501284 as a novel, orally available, non-covalent, pseudo-irreversible pan-KRAS inhibitor designed to overcome limitations of current KRAS-targeted therapies.

**Key findings from the mini symposium titled “*Discovery and characterization of BH-501284: A non-covalent, pan-KRAS inhibitor for treatment of diverse KRAS-mutant tumors”* include:**

- **Median IC50 of 0.83 nM across 41 different G12/G13 KRAS mutant cell lines** (excluding G12R), but sparing HRAS and NRAS (>650 nM) to avoid the toxicity of pan-RAS inhibitors
- **Picomolar binding affinity and prolonged residence time (>54 hours)** mirroring the binding of covalent inhibitors but with a non-covalent, pseudo-irreversible chemistry leading to prolonged inhibition of KRAS signaling
- **Deep tumor regression** in multiple KRAS-mutant xenograft models achieved at lower doses (5-15 mg/kg BID) compared to what has been reported with other contemporary Switch-II pocket inhibitors

The company also presented a poster describing the preclinical characterization of additional molecules within the pan-KRAS program (BH-501242, BH-501352), in which BH-501242 demonstrated cellular potency exceeding that of comparator molecules, and both molecules demonstrated prolonged non-covalent binding, and favorable absorption, distribution, metabolism and excretion (ADME) properties.

**Additional AACR Posters from the BlossomHill Therapeutics Pipeline**

**BH-30643 (Mutant-selective EGFR inhibitor)**
*Poster: “BH-30643, a novel macrocyclic non-covalent, mutant-selective EGFR inhibitor, addresses the resistance and potency limitations of contemporary EGFR TKIs”*

- Potent and durable inhibition across diverse EGFR-mutant models, including C797S and T790M
- Maintained low nanomolar potency in presence of EGFR ligands
- Showed prolonged suppression of tumor cell growth compared to approved EGFR TKIs
- Induced deep tumor regression across multiple *in vivo* models, including intracranial models
- Sustained suppression of EGFR signaling in dose- and time-dependent manner
- Findings support potential of BH-30643 to overcome key limitations of current therapies

**BH-30236 (CLK inhibitor for hematologic malignancies)**
*Poster: “CLK inhibitor BH-30236 synergizes with venetoclax in anti-leukemia activity via splicing modulation in preclinical AML and CLL models”*

- Broad anti-proliferative activity through modulated mRNA splicing
- Reduced expression of pro-survival factors, leading to activation of apoptosis and DNA damage response pathways
- Consistent synergy with venetoclax across multiple blood cancer models
- Induced tumor regression in resistant acute myeloid leukemia (AML) xenografts, including complete responses
- Sustained tumor-free survival following treatment discontinuation, consistent with effects on leukemia stem cell populations
- Findings support continued clinical development of BH-30236 as both a monotherapy and in combination regimens

**About BlossomHill Therapeutics**
BlossomHill Therapeutics, Inc. is a privately held, clinical-stage biopharmaceutical company applying an intentional, chemistry-based approach to develop innovative small molecule medicines which address significant unmet medical needs in cancer treatment. Founded and led by industry veteran J. Jean Cui, Ph.D., with her proven track record in oncology drug design and development – including three FDA-approved drugs – BlossomHill applies cutting-edge science to address key oncogenic drivers and improve patient outcomes in difficult-to-treat cancers. The company's lead clinical programs include BH-30643, a first-in-class, macrocyclic, non-covalent, mutant-selective OMNI-EGFR™ inhibitor for the treatment of EGFR- mutated non-small cell lung cancer (NSCLC), and BH-30236, a macrocyclic CLK inhibitor for the treatment of relapsed or refractory acute myeloid leukemia (R/R AML) or higher-risk myelodysplastic syndrome (HR-MDS), representing a first-in-class opportunity. BlossomHill Therapeutics is headquartered in San Diego, California and has raised over $257 million since its inception from leading life sciences investors. For more information, visit bhtherapeutics.com and follow us on LinkedIn and X.

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