Foghorn Therapeutics Announces Updates for Selective ARID1B, Selective CBP and Selective EP300 Degrader Programs

Oct 30, 2025

- Data presented at the TPD and Induced Proximity Summit demonstrate that novel Selective ARID1B degrader selectively binds and degrades ARID1B; potentially relevant in up to 5% of all solid tumors

- Selective CBP degrader is on track for non-GLP toxicology studies in Q4 2025 with potential in EP300-mutant cancers and in ER+ breast cancer; IND-ready in 2026

- Selective EP300 degrader demonstrates efficacy and favorable tolerability in preclinical models in hematological malignancies with significant differentiation from dual CBP/ EP300 approaches

- Foghorn to host a virtual investor event today, October 30, 2025, at 12 p.m. ET

CAMBRIDGE, Mass., Oct. 30, 2025 (GLOBE NEWSWIRE) -- Foghorn® Therapeutics Inc. (Nasdaq: FHTX), a clinical-stage biotechnology company pioneering a new class of medicines that treat serious diseases by correcting abnormal gene expression, today announced updates for its Selective ARID1B, Selective CBP, and Selective EP300 degrader programs, which will be presented during a Foghorn-hosted virtual investor event.

"We have made significant progress across our degrader portfolio, further highlighting our ability to address challenging and prevalent targets," said Adrian Gottschalk, President and Chief Executive Officer of Foghorn. "Earlier this week, we presented new preclinical data at the TPD and Induced Proximity Summit demonstrating significant progress for our first-in-class Selective ARID1B degrader, with potential as a new therapy for endometrial, gastric, gastroesophageal junction, bladder and non-small cell lung cancer. Our Selective CBP degrader, with potential in EP300-mutant cancers and ER+ breast cancer, is advancing towards IND in 2026 and on track for non-GLP toxicology studies this quarter. Additionally, our Selective EP300 degrader shows encouraging anti-tumor efficacy with favorable tolerability in hematological malignancies in preclinical studies. This is particularly exciting in multiple myeloma where we believe we are significantly differentiated versus dual CBP/ EP300 programs. These advancements along with our continued innovation and disciplined execution are positioning Foghorn at the forefront in the field of targeted protein degradation."

Steven Bellon, Chief Scientific Officer of Foghorn, added, "ARID1B has long been difficult to selectively drug due to its high homology to ARID1A, lack of enzymatic activity, and its largely unstructured nature. Our demonstration of selective degradation of ARID1B represents a major scientific breakthrough that underscores the strength of our protein degrader capabilities to overcome challenges that have historically limited the field."

The live webcast for the investor presentation will be available under the Events & Presentations section of Foghorn's website, and a replay of the event and presentation will be available immediately following the event.

Selective ARID1B Degrader Program

ARID1A is the most mutated subunit in the BAF complex and amongst the most mutated proteins in cancer. These mutations lead to a dependency on ARID1B in up to 5% of all solid tumors including endometrial, gastric, gastroesophageal junction, bladder and non-small cell lung cancer (NSCLC). Attempts to selectively drug ARID1B have been challenging because of the high degree of similarity between ARID1A and ARID1B and the fact that ARID1B has no enzymatic activity to target.

Foghorn is developing a Selective ARID1B degrader that is advancing towards *in vivo* proof of concept in 2026. Key program updates include:

- Developed VHL and cereblon based bifunctional degraders with potential for oral delivery
- Selective degradation of ARID1B achieved
- Modulation of downstream target genes following ARID1B degradation

Data presented at the TPD and Induced Proximity Summit is available under the Science section of the Company's website.

Selective CBP Degrader Program

CBP is an acetyltransferase that selectively targets a synthetic relationship established in EP300-mutated cancers, which includes endometrial, cervical, ovarian, bladder and colorectal cancer. Attempts to selectively drug CBP have been challenging due to the

high level of similarity with EP300, and dose-limiting toxicities associated with dual inhibition of both CBP and EP300. CBP lineage dependencies are established in several cancers, including ER+ breast cancer.

Foghorn is advancing a Selective CBP degrader, on track to be Investigational New Drug (IND)-ready in 2026. Key updates include:

- Highly potent and selective lead candidate CBPd-171 advancing to dose range finding toxicology studies in Q4 2025
- Anti-tumor activity in EP300-mutant solid tumors and in CBP-dependent cancers, including promising potential in ER+ breast cancer
- No significant impact on platelet counts and megakaryocytes spared with CBPd-171 dosing
- Long Acting Injectable (LAI) formulation optimized for subcutaneous injection weekly or every other week for convenient administration

Selective EP300 Degrader Program

EP300 is an acetyltransferase that is implicated in hematological malignancies such as multiple myeloma (MM) and diffuse large b-cell lymphoma (DLBCL), and prostate cancer. Attempts to selectively drug EP300 have been challenging due to the high level of similarity with CBP, and dose-limiting toxicities associated with dual inhibition of both CBP and EP300.

Foghorn is advancing a Selective EP300 degrader program, with an initial focus in MM and DLBCL, on track for IND-enabling studies in 2026. Key updates include:

- Broad anti-tumor activity in over 70% of all heme sub-lineages tested
- VHL based selective degrader shows efficacy in MM without hematological toxicities, including thrombocytopenia
- EP300 degraders show efficacy in IMiD-resistant MM cell lines
- Tolerability profile with widespread potential for combinations

About Foghorn Therapeutics

Foghorn® Therapeutics is discovering and developing a novel class of medicines targeting genetically determined dependencies within the chromatin regulatory system. Through its proprietary scalable Gene Traffic Control® platform, Foghorn is systematically studying, identifying and validating potential drug targets within the chromatin regulatory system. The Company is developing multiple product candidates in oncology. Visit our website at www.foghorntx.com for more information on the Company, and follow us on X and LinkedIn.

Forward-Looking Statements

This press release contains “forward-looking statements” including statements relating to the Company’s proprietary drug discovery programs, the timing and outcome of any potential regulatory filings, including INDs, and the safety and efficacy of the Company’s drug candidates. Forward-looking statements include statements regarding the Company’s clinical trials, product candidates and research efforts and other statements identified by words such as “could,” “may,” “might,” “will,” “likely,” “anticipates,” “intends,” “plans,” “seeks,” “believes,” “estimates,” “expects,” “continues,” “projects” and similar references to future periods. Forward-looking statements are based on our current expectations and assumptions regarding capital market conditions, our business, the economy and other future conditions. Because forward-looking statements relate to the future, by their nature, they are subject to inherent uncertainties, risks and changes in circumstances that are difficult to predict. As a result, actual results may differ materially from those contemplated by the forward-looking statements. Important factors that could cause actual results to differ materially from those in the forward-looking statements include regional, national or global political, economic, business, competitive, market and regulatory conditions, including risks relating to our clinical trials and other factors set forth under the heading “Risk Factors” in the Company’s Annual Report on Form 10-K for the year ended December 31, 2024, as filed with the Securities and Exchange Commission. Any forward-looking statement made in this press release speaks only as of the date on which it is made.

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