UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): February 8, 2024

Foghorn Therapeutics Inc.

(Exact name of registrant as specified in its charter)

Delaware	**001-39634**	**47-5271393**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

500 Technology Square, Ste 700
Cambridge, MA **02139**
(Address of principal executive offices) (Zip Code)

(Registrant's telephone number, including area code): (617) 586-3100

Not Applicable
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, $0.0001 par value per share	**FHTX**	**The Nasdaq Global Market**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

Foghorn Therapeutics Inc. (the "Company") is furnishing as Exhibit 99.1 to this Current Report on Form 8-K a presentation, dated February 2024, which the Company intends to use in meetings with or presentations to investors.

The information in this Item 7.01 (including Exhibit 99.1 attached hereto) is being furnished and shall not be deemed "filed" for purposes of Section 18 of the Exchange Act, or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference into any filing by the Company under the Securities Act or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 8.01 Other Events.

On February 8, 2024, the Company issued a press release announcing that Eli Lilly and Company ("Lilly") has selected FHD-909, a first-in-class oral BRM selective inhibitor of BRG1, for clinical development. Lilly plans to file an IND for FHD-909 in the second quarter of 2024. The primary target patient population is BRG1 mutated non-small cell lung cancer.

A copy of the Company's press release is attached hereto as Exhibit 99.2 and is incorporated herein by reference.

Forward-Looking Statements

This Current Report on Form 8-K contains "forward-looking statements." 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, 2022, as filed with the Securities and Exchange Commission. Any forward-looking statement made in this Current Report speaks only as of the date on which it is made.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Investor Presentation dated February 2024
99.2	Press release issued on February 8, 2024

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

FOGHORN THERAPEUTICS INC.

By: /s/ Michael LaCascia
Michael J. LaCascia
Chief Legal Officer

Date: February 8, 2024

Exhibit 99.1

FOGHORN® THERAPEUTICS

Unique biology

Precision therapeutics

Broad impact

February 2024

Forward Looking Statements

This presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation are forward-looking statements. In some cases, you can identify forward-looking statements by terms such as “could,” “may,” “might,” “will,” “likely,” “anticipates,” “intends,” “plans,” “seeks,” “believes,” “estimates,” “expects,” “continues,” “projects” or the negative of these terms or other similar expressions, although not all forward-looking statements contain these words. Forward-looking statements include, but are not limited to, statements concerning: the potential outcomes from our collaboration agreements with Lilly; the initiation, timing, progress and results of our research and development programs and pre-clinical studies and clinical trials, including with respect to our Phase 1 study of FHD-286 in combination with decitabine or cytarabine in relapsed and/or refractory AML patients and anticipated timing of release of clinical data, and the planned Phase 1 dose escalation study of FHD-909 with Loxo@Lilly; our ability to advance product candidates that we may develop and to successfully complete preclinical and clinical studies; our ability to leverage our initial programs to develop additional product candidates using our Gene Traffic Control Platform; the impact of exogeneous factors, including macroeconomic and geopolitical circumstances, on our and our collaborators’ business operations, including our research and development programs and pre-clinical studies; developments related to our competitors and our industry; our ability to expand the target populations of our programs and the availability of patients for clinical testing; our ability to obtain regulatory approval for FHD-286 and any future product candidates from the FDA and other regulatory authorities; our ability to identify and enter into future license agreements and collaborations; our ability to continue to rely on our CDMOs and CROs for our manufacturing and research needs; regulatory developments in the United States and foreign countries; our ability to attract and retain key scientific and management personnel; the scope of protection we are able to establish, maintain and enforce for intellectual property rights covering FHD-286, our future products and our Gene Traffic Control Platform; and our use of proceeds from capital-raising transactions, estimates of our expenses, capital requirements, and needs for additional financing. Any forward-looking statements represent the Company’s views only as of today and should not be relied upon as representing its views as of any subsequent date. The Company explicitly disclaims any obligation to update any forward-looking statements. The Company’s business is subject to substantial risks and uncertainties.

First-In-Class Precision Medicines Targeting Major Unmet Needs in Cancer

Leader in Unique Area of Cancer Biology

Foghorn is a **leader in targeting chromatin biology,** which has the potential to address underlying dependencies of many genetically defined cancers

Broad pipeline across a range of targets and modalities

Large Market Potential

Chromatin biology is implicated in up to **50% of tumors**, potentially impacting **~2.5 million patients**

Foghorn's current pipeline potentially addresses **more than 500,000** of these patients

Well-Funded

$259.9 million in cash and equivalents
(as of 9/30/2023)

Provides **runway into H1'26**

Value Drivers

Anticipate data from the Phase 1 study of FHD-286 in combination with decitabine in **H2'24**

BRM Selective Inhibitor (FHD-909), partnered with Loxo@Lilly, **anticipating IND filing in Q2'24**

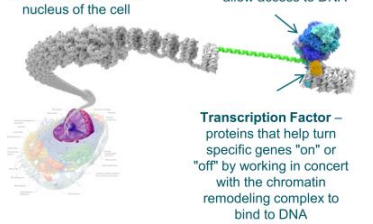
Advancement of preclinical assets (BRM Selective Degrader, CBP, EP300, ARID1B) towards INDs

Major Strategic Collaboration

Strategic collaboration with Loxo@Lilly; **$380 million upfront;** 50/50 U.S. economic split on two lead programs

Unique Insights into Chromatin Biology to Prosecute Untapped Area for Novel Targets and Therapeutics

Chromatin Regulatory System Critical for Gene Expression



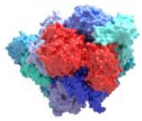
Chromatin – compacted form of DNA inside the nucleus of the cell

Chromatin Remodeling Complex – specialized multiprotein machineries that allow access to DNA

Transcription Factor – proteins that help turn specific genes "on" or "off" by working in concert with the chromatin remodeling complex to bind to DNA

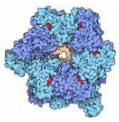
Novel Targets Guided by Genetic Dependencies

Chromatin Remodeling Complex Mutations / Overexpression



Transcription Factor Mutations / Overexpression

Helicases & Other Chromatin Binding Proteins involved in gene expression / function



Tailored Drugging Approaches

Enzymatic Inhibitors Highly selective and allosteric small molecule inhibitors

Targeted Protein Degradation Molecular glue and bi-functional protein degraders

Transcription Factor Disruptors Disrupt interactions between chromatin remodeling complexes and transcription factors

Foghorn's Validated Gene Traffic Control© Platform Enables an Integrated, Scalable, Efficient and Repeatable Paradigm

Targeting Disease

Deep mechanistic understanding of the chromatin regulatory system

What to Drug:

Identify disease dependencies with novel targets

Specialized Approach

Biochemistry, biophysics and assays of large complexes and proteins

Where to Drug:

Engineer selectivity via unique assays and protein capabilities

Selective Therapeutics

Biology first, small molecule modality agnostic

How to Drug:

Small molecules, degrader and delivery platform

Enzymatic Inhibitors	Targeted Protein Degraders	Transcription Factor Disruptors

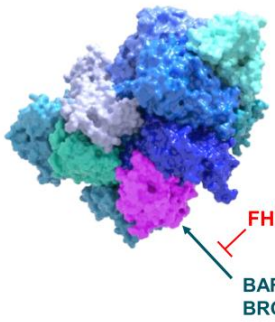
Broad and Deep Pipeline Across a Range of Targets and Modalities

Modality	Program	Disease	Commercial Rights
Enzyme Inhibitors	FHD-286 (BRG1/BRM)	Relapsed/Refractory AML	FOGHORN THERAPEUTICS
	FHD-909 (Selective BRM)	BRG1 mutant cancers (e.g., ~8-10% of NSCLC, bladder, endometrial, colorectal)	LOXO FOGHORN THERAPEUTICS
Protein Degraders	Selective BRM	BRG1 mutant cancers (e.g., ~8-10% of NSCLC, bladder, endometrial, colorectal)	LOXO FOGHORN THERAPEUTICS
	Selective EP300	EP300 dependent cancers (e.g., prostate, DLBCL), CBP mutant cancers (e.g., ~9-10% of NSCLC, bladder, melanoma)	FOGHORN THERAPEUTICS
	Selective CBP	EP300 mutant cancers (e.g., ~5-10% of bladder, gastric, breast, NSCLC, colorectal)	FOGHORN THERAPEUTICS
	Selective ARID1B	ARID1A mutant cancers (~5% of all solid tumors)	FOGHORN THERAPEUTICS
Transcription Factor Disruptors	Undisclosed	Undisclosed	FOGHORN THERAPEUTICS
Partnered Program	Undisclosed	Undisclosed	LOXO FOGHORN THERAPEUTICS
3 Discovery Programs	Undisclosed	Undisclosed	LOXO FOGHORN THERAPEUTICS

FHD-286: Dual BRM/BRG1 Inhibition

Targeting BAF Dependency in Cancer

Exploring BAF Dependency in Cancer with FHD-286 – Potent, Small Molecule Inhibitor Targeting BRM and BRG1



FHD-286:

- **Allosteric modulation inhibiting the activity of both BRM and BRG1**
- **Oral, daily, potent small molecule inhibitor**

Current and Potential Future Opportunity

Mutations	Pre-clinical data support ability to address BAF mutated cancers (e.g., BRG1 mutant)
Differentiation	Clinical and pre-clinical data demonstrate broad-based differentiation across AML and multiple solid tumors
Overcoming Drug Resistance	Pre-clinical data support ability to overcome drug resistance (i.e., EGFR NSCLC, enzalutamide-resistant CRPC, PD-1 refractory)
Immune Modulation	Clinical data demonstrate an increase of CD8+ T-cells and a reduction of T-regulatory cells

First-in-Class Broad-Based Differentiation Agent With Significant Combination Potential in AML

Significant Opportunity

- ~27,000 drug treated relapsed and/or refractory (R/R) AML patients*
- No broad differentiation agent approved in AML
- Significant combination potential

*U.S., EU5, Japan

Completed Phase I Monotherapy Safety and Efficacy Results

Efficacy

- Differentiation observed in heavily pre-treated patients, regardless of mutational status
- Multiple patients with bone marrow and peripheral blast improvements and associated ANC recovery

Safety

- Adverse event profile consistent with late-line AML population
  - Most frequent ≥ grade 3 TRAEs: increased blood bilirubin, hypocalcemia, differentiation syndrome (DS), stomatitis, increased ALT
- Adjudicated Differentiation Syndrome rate of 15%

Ongoing Phase I Combination Study

- Phase I dose escalation study evaluating oral daily dosing of FHD-286 with fixed dose decitabine or cytarabine
- Standard 3+3 dose escalation design
- Data anticipated in H2'2024

FHD-286 Demonstrated Differentiation Across a Broad Range of Genetic Backgrounds

Dose Level	Mutations	Cytogenetics Risk	Starting CD11b%	Max CD11b%	CD11b+ Fold Change	Starting CD34%	Min CD34%	CD34+ % Decrease
10mg	N/A	Adverse	7	62	9.2x	94	27	(71%)
7.5mg	CBFB (locus at 16q22)		2	94	59.4x	70	2	(97%)
7.5mg	KMT2A rearrangement	Adverse	3	58	21.4x	85	9	(90%)
7.5mg	RUNX1, KRAS, ASXL1, JAK2, TET2, EZH2, ETNK	Adverse	5	73	15x	95	18	(81%)
7.5mg	N/A	Adverse	8	52	6.3x	94	33	(65%)
7.5mg	ASXL1, TP53, U2AF1	Adverse	19	63	3.3x	92	51	(45%)
5mg	RUNX1, NRAS, KRAS, SF3B1, ASXL2, CSF3R, GATA2	Adverse	3	74	29x	94	19	(80%)
5mg	RUNX1, NRAS, ASLX1	Adverse	4	97	22.8x	98	7	(93%)
5mg	N/A	Adverse	6	79	13x	93	11	(88%)
5mg	TET2, WT1 GATA2 PLCG2 ARHGEF28, BRD4, CDK12, DDX41, KMT20, PARP1, ZRSR2		3	24	8.1x	86	62	(27%)
5mg	N/A	Adverse	4	28	6.5x	93	66	(29%)
5mg	DNMT3a, TET2		21	88	4.1x	30	4	(88%)
2.5mg	NRAS, WT1	Adverse	3	13	4.8x	93	89	(4%)

CD34 (leukemic stem cell marker) decreases

CD11b (marker of differentiation) increases

Clear Signs of Differentiation in Heavily Pre-Treated, Secondary AML Patient with Abnormal Karyotype

Patient Background:

- 47-year-old male, secondary AML
- Abnormal karyotype: Del (7Q), Inv (3), Der (7;12), -8, ADD(1)

Prior AML Treatment:

- Progressive disease: 4 lines prior treatment and 2 bone marrow transplants

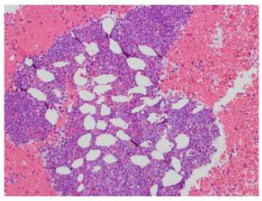
Prior non-AML treatment:

- MDS with inv(3) and der(7;12) and ASXL1 mut. Received AZA x 4.

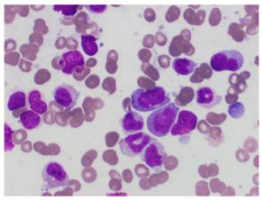
Initiation of FHD-286 at 10 MG Dose:

- Bone marrow blast from 40% to 6% with clear evidence of differentiation with persistence of cytogenetics abnormalities. ANC recovery.

Bone Marrow Blast Reduction from 40% to 6%



Bone Marrow Aspirate: Clear Evidence of Differentiation



Meaningful Clinical Benefit in Heavily Pre-Treated Patient

Patient Background:

- 25-year-old male, treatment-related AML
- KMT2A rearrangement

Prior AML Treatment:

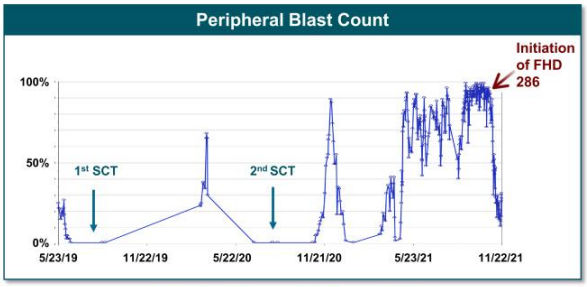
- Progressive disease with CNS Leukemia: 7 lines prior treatment and 2 bone marrow transplants

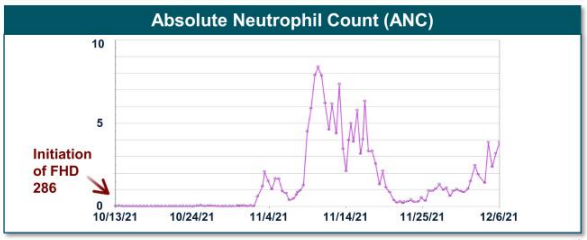
Prior non-AML treatment:

- Ewing's sarcoma: Treated with Chemo/RT/Surgery (VCR, doxo, cyclophos, ifos, etoposide)

Initiation of FHD-286 at 10 MG Dose:

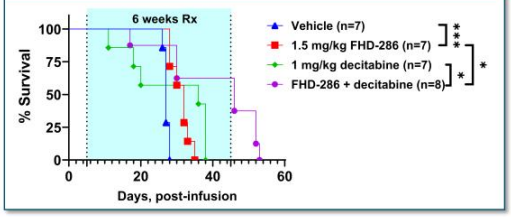
- Drop in peripheral blast, 97% to 5%
- Bone marrow reduction from 89% to 48%, with ANC recovery



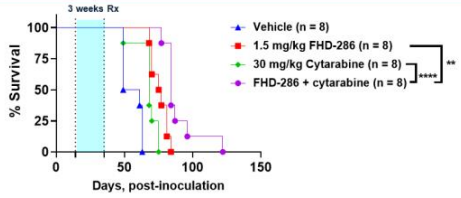


Pre-Clinical Data Demonstrate Significant Combination Potential with Multiple Agents in AML

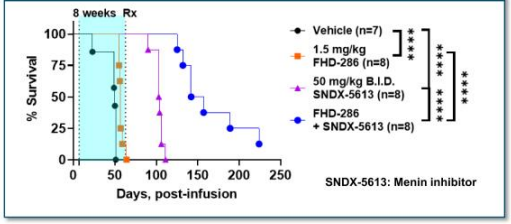
FHD-286 + decitabine
(MLL-AFP + FLT3 TKD Luc/GFP PDX)



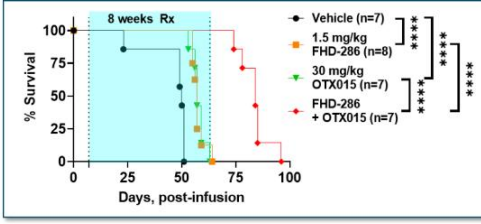
FHD-286 + cytarabine
(MV4, 11 FLT3 ITD CDX)



FHD-286 + menin inhibitor
(mtNPM1 + FLT3 ITD Luc/GFP AML PDX)



FHD-286 + BET inhibitor
(mtNPM1 + FLT3 ITD Luc/GFP AML PDX)



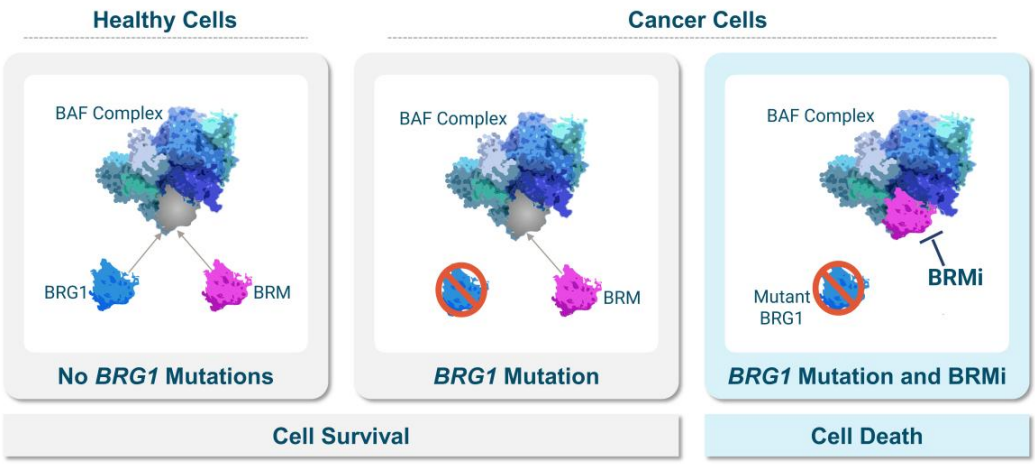
Selective BRM Modulators

For BRG1 Mutated Cancers

BRM Selective Inhibitor FHD-909 IND Targeted in Q2'24, BRM Selective Degrader Continues Late-Stage Pre-Clinical Development

	BRM Selective Inhibitor (FHD-909)	BRM Selective Degrader
Biology	Exploit the synthetic lethal relationship between BRM and mutated BRG1	
Stage	IND submission planned in Q2'24	Advancing in parallel through late pre-clinical development
Opportunity	BRG1 mutated cancer including ~10% of NSCLC and up to 5% of all solid tumors	
Loxo@Lilly Partnership	50/50 global R&D cost share | 50/50 U.S. economics | tiered ex-U.S. royalties starting in the low double-digit range and escalating into the twenties	

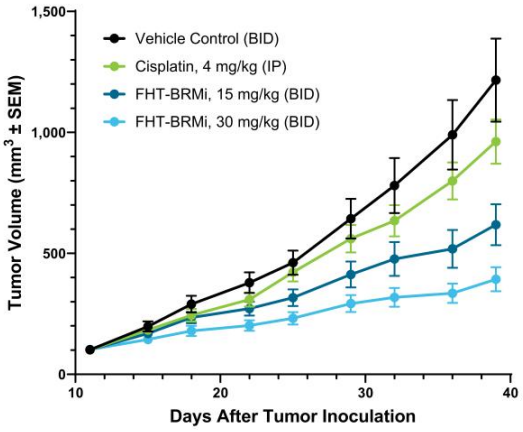
BRM Selective Inhibition and Degradation Exploit the Synthetic Lethal Relationship Between BRM and Mutated BRG1



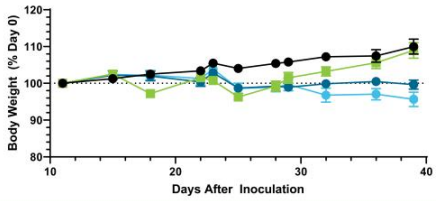
- *BRG1* mutant cancer cells are dependent on BRM ATPase for survival
- Selectively targeting BRM ATPase is a potentially effective therapeutic option for *BRG1* mutated cancers

BRM Selective Inhibitor Demonstrates PK/PD and *In Vivo* Efficacy in a BRG1 Mutant Lung CDX Model

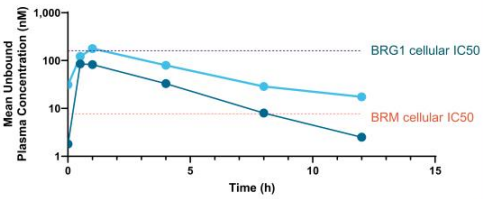
A549-BRG1 Mutant NSCLC Model



Body Weight Loss



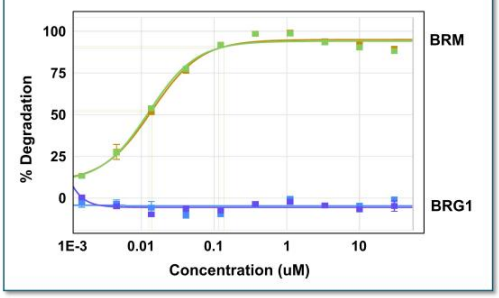
PK/PD



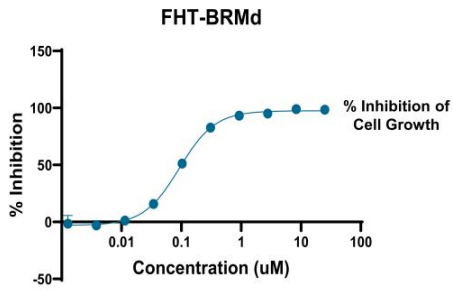
Data as of Q4 2021

BRM Selective Degrader Achieves Complete BRM Degradation and Cell Growth Inhibition

BRM/BRG1 HIBIT Data



A549 Ten-Day Proliferation Assay



Degraders Cause Time- and Dose-Dependent BRM Degradation Antiproliferative Effects in A549 Mutant NSCLC Model

Data as of Q4 2021

Selective EP300 Protein Degrader

For CBP Mutated and EP300 Dependent Cancers

Summary: Selective EP300 Protein Degrader for CBP Mutant & EP300 Dependent Cancers

Target / Approach	• E1A binding protein p300 (EP300) • Targeted protein degrader
Initial Indications	• AR+ Prostate • DLBCL • Bladder, melanoma, others
Mutation / Aberration	• EP300 dependent cancers • CBP mutant cancers
Stage	• Pre-clinical
New Patients Impacted / Year*	• Over 100,000

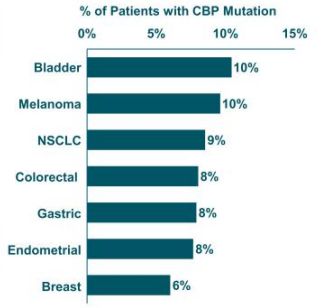
* Per year incidence in the U.S., EU5, Japan

Commercial Opportunity

EP300 Dependent Cancers

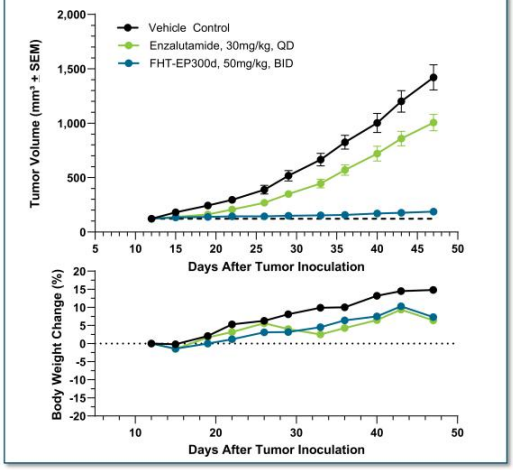
- **Solid Tumors**
  - AR+ mCRPC
  - HR+ breast
- **Hematologic malignancies**
  - DLBCL
  - Multiple Myeloma

CBP Mutant Cancers

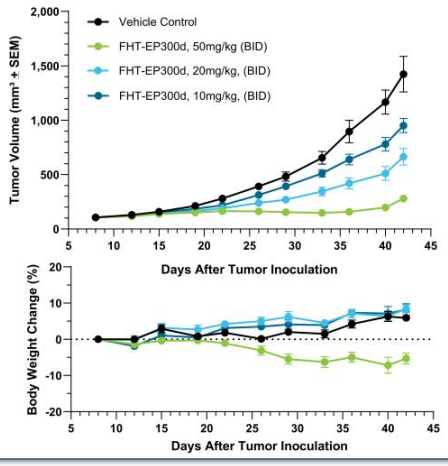


EP300 Degradation Results in Significant Tumor Growth Inhibition in AR+ VCAP Prostate and KARPAS422 DLBCL Models

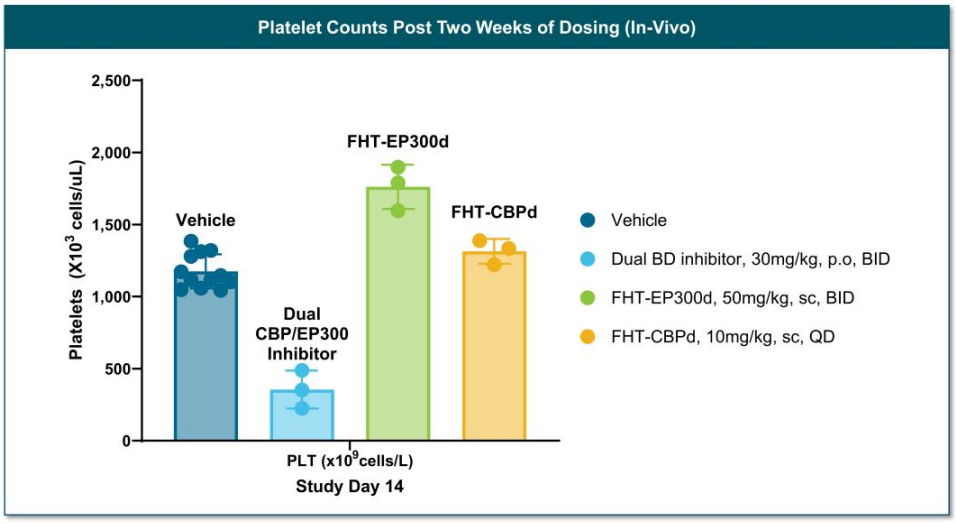
Prostate
AR+ VCAP Model



DLBCL
KARPAS422 (CBP Null) Model



Selective Degradation of EP300 and CBP Does Not Show Thrombocytopenia in Mice at Relevant Doses



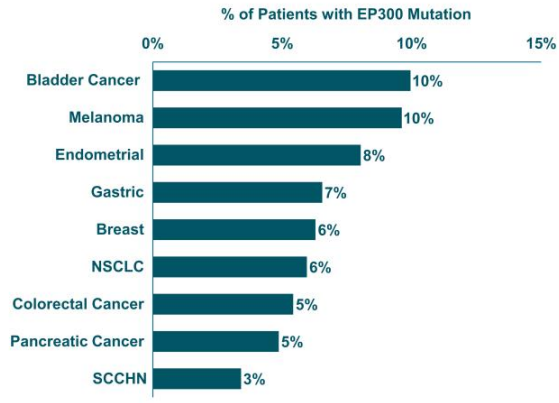
Selective CBP Protein Degrader

For EP300 Mutated Cancers

Summary: Selective CBP Protein Degrader for EP300 Mutated Cancers

Target / Approach	• CREB binding protein (CBP) • Targeted protein degrader
Initial Indication	• EP300 mutated cancers (e.g., subsets of bladder, colorectal, breast, gastric and lung cancers)
Mutation / Aberration	• EP300 mutated cancers
Stage	• Pre-clinical
New Patients Impacted / Year*	• Over 100,000

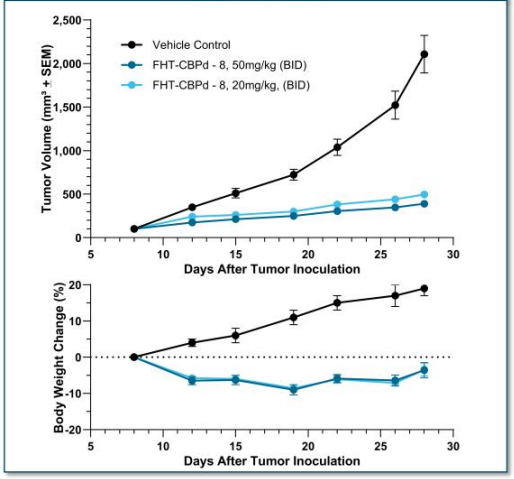
Commercial Opportunity



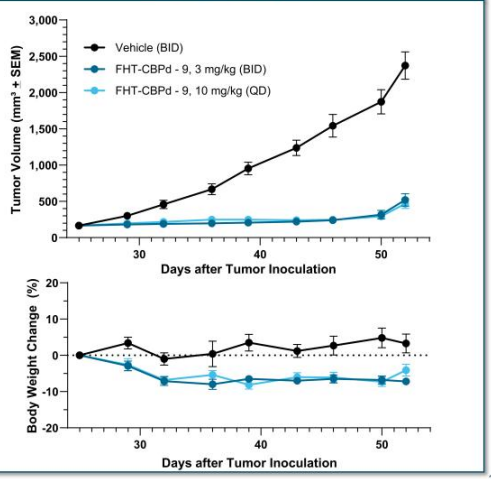
* Per year incidence in the U.S., EU5, Japan

Selective CBP Protein Degrader Result in Significant Tumor Growth Inhibition in Colorectal and Bladder EP300 Null Models

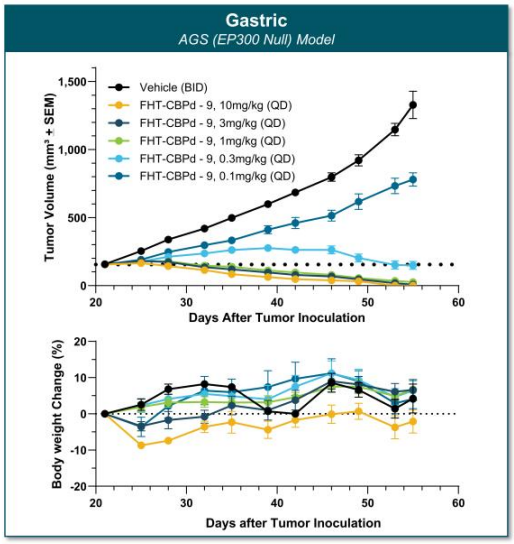
Colorectal
RKO (EP300 Null) Model



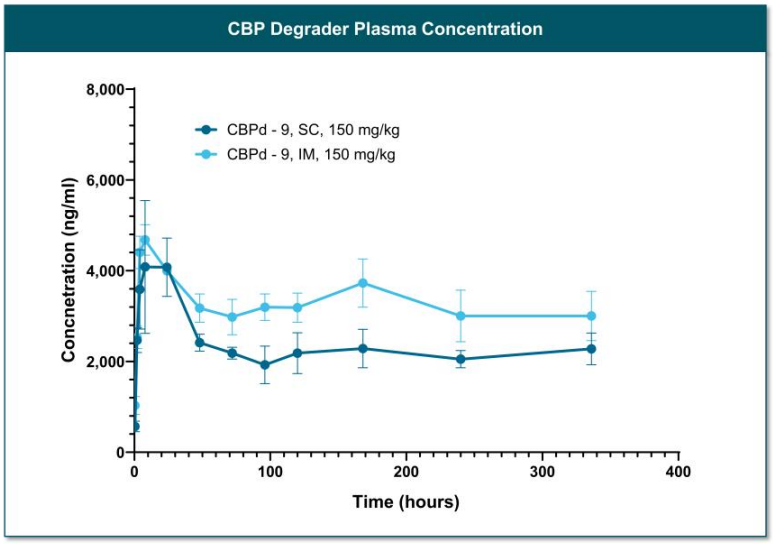
Bladder
639V (EP300 Null) Model



Selective CBP Protein Degrader Result in Tumor Regression in Gastric EP300 Null Models



Long-Acting Injectable Formulations of CBP Degrader Could Enable Once Every 2 Weeks (or Better) Dosing Frequency

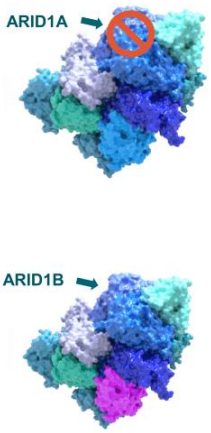


Selective ARID1B Protein Degrader

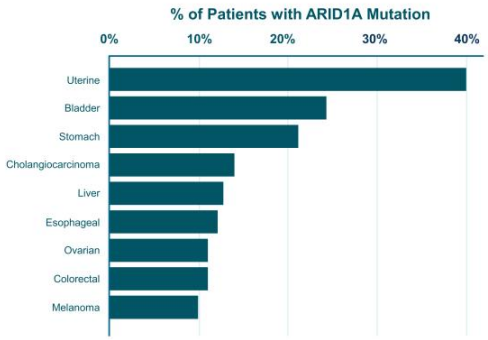
For ARID1A Mutated Cancers

ARID1B is a Major Synthetic Lethal Target Implicated in Up To 5% of All Solid Tumors

Target / Approach	• ARID1B • Targeted protein degrader
Initial Indication	• ARID1A mutated cancers
Mutation / Aberration	• ARID1A mutations (e.g., ovarian, endometrial, colorectal, bladder and other cancers)
Stage	• Pre-clinical
New Patients Impacted / Year*	• > 175,000



Commercial Opportunity



~5% of all solid tumors harbor ARID1A mutations

* Per year incidence in the U.S., EU5, Japan

Targeting ARID1B for ARID1A Mutated Cancers is Enabled by Foghorn's Unique Biology and Discovery Capabilities

Gene Traffic Control Platform

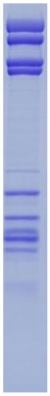
- Platform produces BAF complexes and subcomplexes containing either ARID1A or ARID1B at scale
- Enables proprietary screens against ARID1B

Protein Degrader Capabilities

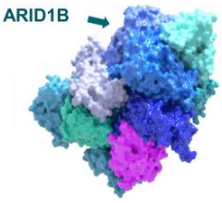
- Utilize protein degrader toolbox to create ARID1B hetero-bifunctional degraders

Program Status

- Validated selective chemical binders of ARID1B
- In process of expanding binders into novel selective protein degraders
- Assessing outcomes of ARID1B degradation and impact on BAF complex formation



Highly purified ARID1B / BAF complex



Transcription Factors

A Novel Approach

Foghorn's Novel Approach to Drugging Transcription Factors Enabled by Its Protein Production and Discovery Capabilities

Transcription Factors are Compelling Drug Targets…

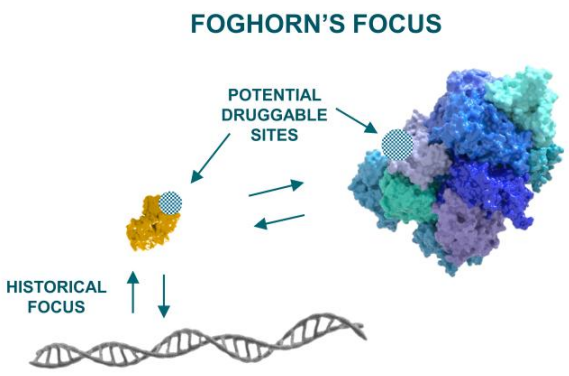
- Highly involved in gene expression
- Implicated in range of cancers and other diseases

…But Historically Difficult to Target…

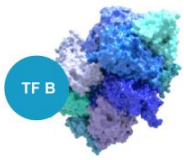
- Featureless surface: no druggable binding pocket
- Tight interactions with DNA: undruggable affinities

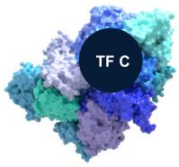
Foghorn Has a New Approach Focusing on Interaction with BAF

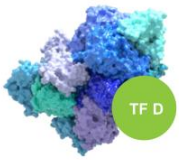
- Druggable binding pockets
- Druggable affinities



Transcription Factors Bind to BAF Directly with High Degree of Specificity; Unique Insights into Where and How Transcription Factors Bind

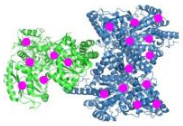




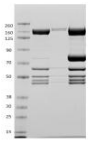


Mapping the TF-BAF Interaction

Mass spec. foot-printing



Pull-down assays

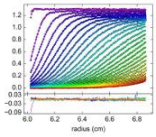


Foghorn's collection of BAF sub-complexes and domains

Validating the TF-BAF Interaction

Biophysical

AUC / SPR / ITC

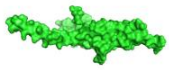


Biochemical

TR-FRET / FP

Structural

Crystal / NMR



Broad and Deep Pipeline Across a Range of Targets and Modalities

Modality	Program	Disease	Commercial Rights
Enzyme Inhibitors	FHD-286 (BRG1/BRM)	Relapsed/Refractory AML	FOGHORN THERAPEUTICS
	FHD-909 (Selective BRM)	BRG1 mutant cancers (e.g., ~8-10% of NSCLC, bladder, endometrial, colorectal)	LOXO FOGHORN THERAPEUTICS
Protein Degraders	Selective BRM	BRG1 mutant cancers (e.g., ~8-10% of NSCLC, bladder, endometrial, colorectal)	LOXO FOGHORN THERAPEUTICS
	Selective EP300	EP300 dependent cancers (e.g., prostate, DLBCL), CBP mutant cancers (e.g., ~9-10% of NSCLC, bladder, melanoma)	FOGHORN THERAPEUTICS
	Selective CBP	EP300 mutant cancers (e.g., ~5-10% of bladder, gastric, breast, NSCLC, colorectal)	FOGHORN THERAPEUTICS
	Selective ARID1B	ARID1A mutant cancers (~5% of all solid tumors)	FOGHORN THERAPEUTICS
Transcription Factor Disruptors	Undisclosed	Undisclosed	FOGHORN THERAPEUTICS
Partnered Program	Undisclosed	Undisclosed	LOXO FOGHORN THERAPEUTICS
3 Discovery Programs	Undisclosed	Undisclosed	LOXO FOGHORN THERAPEUTICS

First-In-Class Precision Medicines Targeting Major Unmet Needs in Cancer

Leader in Unique Area of Cancer Biology

Foghorn is a **leader in targeting chromatin biology,** which has the potential to address underlying dependencies of many genetically defined cancers

Broad pipeline across a range of targets and modalities

Large Market Potential

Chromatin biology is implicated in up to **50% of tumors**, potentially impacting **~2.5 million patients**

Foghorn's current pipeline potentially addresses **more than 500,000** of these patients

Well-Funded

$259.9 million in cash and equivalents
(as of 9/30/2023)

Provides **runway into H1'26**

Value Drivers

Anticipate data from the Phase 1 study of FHD-286 in combination with decitabine in **H2'24**

BRM Selective Inhibitor (FHD-909), partnered with Loxo@Lilly, **anticipating IND filing in Q2'24**

Advancement of preclinical assets (BRM Selective Degrader, CBP, EP300, ARID1B) towards INDs

Major Strategic Collaboration

Strategic collaboration with Loxo@Lilly; **$380 million upfront;** 50/50 U.S. economic split on two lead programs

Foghorn Provides Pipeline Update on FHD-909 BRM Selective Inhibitor

CAMBRIDGE, Mass. -- (GLOBE NEWSWIRE) – February 8, 2024 -- 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 that Lilly has selected FHD-909, a first-in-class oral BRM selective inhibitor, for clinical development. Lilly plans to file an IND for FHD-909 in Q2 2024. The primary target patient population is BRG1 mutated non-small cell lung cancer (NSCLC).

Selective BRM inhibition has been a sought-after objective in cancer research for many years. A variety of tumor types, including NSCLC, are known to have mutations in BRG1, which we believe make them dependent on BRM activity for their survival. Selective blocking of BRM activity is considered a promising strategy for causing tumor cell death while sparing healthy cells.

In December 2021, Foghorn announced a strategic collaboration with Lilly to create novel oncology medicines. The collaboration includes a US 50/50 co-development and co-commercialization agreement for Foghorn's Selective BRM oncology program and an additional undisclosed oncology target. In addition, the collaboration includes three discovery programs using Foghorn's proprietary Gene Traffic Control platform.

The Companies plan to present preclinical data at upcoming scientific conferences.

About FHD-909
FHD-909 is a highly potent, allosteric and orally available small molecule that selectively inhibits the ATPase activity of BRM over its closely related paralog BRG1, two proteins that are the catalytic engines across all forms of the BAF complex, one of the key regulators of the chromatin regulatory system. In preclinical studies, tumors with mutations in BRG1 rely on BRM for BAF function. FHD-909 has shown significant anti-tumor activity across multiple BRG1-mutant lung tumors.

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 (formerly Twitter) and LinkedIn.

Forward-Looking Statements

This press release contains "forward-looking statements." Forward-looking statements include statements relating to the planned Phase 1 dose escalation study of FHD-909, 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, 2022, 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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